A note on the compression of mortality

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Abstract. The rapid increase in human longevity has raised important questions about what implications this development may have for the variability of age at death. Earlier studies have reported evidence of a historical trend towards mortality compression. However, the period life table model, commonly used to address mortality compression, produces an artificially compressed picture of mortality as a built-in feature of the model. We base our study on an examination of the durations of exposure, in years of age, of birth cohorts and period life tables to selected levels of mortality observed at old age. We also address the problem in a more conventional fashion, by examining the distribution of ages at death above and below the mode. Overall, mortality has been *decompressing* since the 1960s. This finding contradicts most previously reported results. The decompression of old-age mortality may further indicate good prospects for ever-decreasing mortality. In the future, deaths may not be concentrated within a narrow age interval, but will instead become more dispersed, though at ever later ages on average.

Introduction

Human longevity continues to increase in developed countries. In countries that currently have low mortality, the average lifespan has increased from the pre-modern level of about 35 years, to about 75 years for the currently old cohorts. This increase is expected to continue: the United Nations (UN Population Division 2010) has forecast that period life expectancy at birth will increase, though at a gradually declining pace, to about 85 years by 2050 in low-mortality countries.

The rapid rise in human longevity has raised important questions about what implications this trend may have for the variability of age at death. Fries (1980), based on the assumption that there is a fixed upper limit to the human lifespan, argued that the decline in mortality may lead to a rectangularization of morbidity and survival curves; i.e., a compression of illness and death to the oldest age. This view is supported by the age pattern of mortality decline, which is characterized by a more rapid decline in mortality at young ages, and almost no change at the oldest-old ages (with only a few exceptions, of which Japan is the most notable).

Later studies, while questioning the strict link between mortality compression and the existence of an upper limit to the lifespan (Carnes et al. 1996; Olshansky et al. 2002), and even the very process of mortality decline (as Wilmoth and Horiuchi, 1999, put it: "the *average* length of life … is generally independent of its *variability*"), provided more evidence of a historical trend towards mortality compression, which may have slowed down or even stopped in recent decades in developed countries (Wilmoth and Horiuchi 1999; Kannisto 2000; Canudas-Romo 2008; Thatcher et al. 2010; Engelman et al. 2010). Some authors (e.g., Myers and Manton 1984) have

argued against the premise that mortality compression is occurring, but their evidence was based on a methodology with a built-in tendency towards decompression (see below).

The prospect of a compression of mortality and morbidity would have had obvious implications for public health policies. Mortality compression was argued to be relevant for health-related behavior (Wilmoth and Horiuchi 1999). Evidence of a compression, even of a stalled one, is important in helping us to better understand the future of aging and senescence. Even though the direct link between mortality compression and the existence of an upper limit to the lifespan was discarded in the literature, the compression of mortality may still be associated with increasing efforts to further extend life expectancy. The shifting mortality hypothesis, which is supported by the stalling of mortality compression in developed countries during recent decades (Canudas-Romo 2008), was associated with the inability to slow the pace of deterioration with age, at least so far (Vaupel 2010). Understanding the developments in mortality compression is also important for modeling mortality and formulating assumptions about future mortality dynamics (Tuljapurkar and Edwards 2011).

Despite numerous advances, however, the question of which methodology is most suitable for studying mortality compression remains unresolved. Existing methods are affected by the in-built biases of the period life table model and age censoring (see a critical review of methodological approaches in the next section). Building on an earlier analysis of the limitations of the conventional period life table model (Ediev 2011), we propose a new approach to studying mortality compression. Using extensive mortality data and a new, cohort-oriented methodology, we show that mortality compression at old ages was not a universal trend in the past in low-mortality countries: the whole period since the 1960s—i.e., the period of time when the greatest reductions in old-age mortality have occurred—has been characterized by mortality

decompression. Meanwhile, the period life tables in low-mortality countries have been characterized by mortality compression.

In the following two sections, we briefly review existing methodological approaches, reiterate some concerns about the limitations of the conventional period life table model in case of dynamic mortality, and show why the period life table model tends to artificially compress the mortality pattern when mortality is on the decline. This explains our reasons for introducing a new methodology based on a cohort approach, which we present in Section 3. The empirical evidence is provided in Section 4. We present more traditional indicators of compression of the distribution of ages at death in relation to the modal age at death in Section 5, and then offer some conclusions. The appendices contain the formal relations that support the main findings.

1. Literature review on the methodology of studying mortality compression

In this section, we briefly review key methodological approaches to studying mortality compression and account for the limitations of existing methods. The review is, for the most part, organized in chronological order.

Entropy, the Keyfitz *H* (Keyfitz 1977, Mitra 1978, Demetrius 1978, 1979, see also Goldman and Lord, 1986, and the following discussion with Mitra, 1986, in this journal), was an early index of variation in age at death. Along with variance, entropy is a common statistical measure of the uncertainty of a probability distribution; however, the two measures may differ in their judgments about the uncertainty and its change, and there may be no clear-cut basis for

preferring one measure over another (e.g., Ebragimi et al. 1999) [1]. Although it was used in some studies (e.g., Nagnur 1986, Nusselder and Mackenbach 1996, Wilmoth and Horiuchi 1999), its usage was limited not least by a clearer interpretation of the alternative: the variance in the age at death. A particular drawback of using entropy in our study is that it may not be computed for birth cohorts before the completion of their lifespan.

Fries (1980) – whose work played a key role in popularizing the topics of mortality and morbidity compression – as well as earlier researchers cited by him, based their research on a visual inspection of period survival curves, as well as on arguments in favor of the existence of *fixed* limits to the human lifespan. The latter arguments, while implying that a mortality compression must accompany the approach of lifespan limits, may be discarded in view of recent and more comprehensive data (e.g., Oeppen and Vaupel 2002, Vaupel 2010).

Myers and Manton (1984) challenged the very existence of mortality compression, examining a more complete life table for the U.S. population and computing standard deviations

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[1] Confusion regarding the statistical entropy of the probability distribution has contributed to some contradictory interpretations of entropy in demography. Strictly speaking, the entropy used in demography, $H = -\frac{1}{e_0}\int_0^\infty l(x)\ln l(x)dx$, $l(x)$ being the probability of surviving to age x and $e_0 = \int_0^\infty l(x)dx$ being the life expectancy at birth, is not entropy of the distribution of ages at death in the statistical sense. That would be $H^d = -\int_0^\infty d(x)\ln d(x)dx$, where $d(x) = -\frac{d}{dx}l(x)$ is the density function of the distribution of ages at death. The statistical entropy, at any given life expectancy, is maximal for the exponential distribution $d(x) = d(0)e^{-\mu \cdot x}$; this property has led some to assume that the demographic type of entropy may also be maximal (one) for the exponential distribution; this idea was, however, dispelled by Goldman and Lord (1986) in this journal. Demographic entropy is maximal for a bimodal distribution, where death occurs only at infancy and the lifespan limit. Demographic entropy, which may be interpreted as entropy of the distribution of the person-years lived $\frac{l(x)}{e_0}$, is, nonetheless, useful in some applications (e.g., Keyfitz 1977).

of ages of death at ages 60 and over. Those indicators were, however, criticized as being prone to a built-in decompression of mortality when life expectancy is growing and the distribution of deaths is shifted to older ages (Kannisto 2001). In a schematic model where deaths are uniformly distributed between some young age $a < 60$ and the maximum age $X > 60$, the standard deviation of ages at death above age 60, $S_{60} = \frac{X - 60}{2\sqrt{3}}$, would be proportional to the remaining life expectancy at age 60, $e_{60} = \frac{X - 60}{2}$. It would not show mortality compression even in the Fries' scenario, when the life expectancy *at birth* increases due to increasing a, but the limit to the lifespan X is fixed; S_{60} would show *decompression* when the whole distribution of deaths shifts to older ages without compressing or decompressing (when both a and X increase at the same speed). Results for more general mortality changes are less straightforward (see Appendix A), but also support the schematic model above: the standard deviation computed after left-censoring the distribution of deaths at a fixed age is usually biased towards showing decompression. In the context of our study, usage of the standard deviation of age at death is also limited due to an inability to calculate the index for birth cohorts with incomplete life histories.

Wilmoth and Horiuchi (1999) stressed the importance of objective indices of compression, and considered 10 such indices. They chose as their favorite the Interquartile Range of the distribution of ages at death (*IQR*) for its convenience and good correlation with other measures. They reported a historical compression of mortality attributable to declining risks of death at young ages, but also, to a lesser extent, at ages up to age 75. Finding Fries's prediction of a fixed limiting mortality pattern somewhat inconsistent, Wilmoth and Horiuchi also argued against "derectangularization" (Gavrilov and Gavrilova 1991), while arguing in favor of "a continuing pattern of stability in the variance of ages at death, as the entire distribution shifts upward."

However, the results based on period life table distributions of ages at death may be biased towards showing mortality compression, an issue we will discuss in more detail in the next section. When it comes to the cohort data, it would only be possible to compute the IQR for cohorts for whom the first three quartiles of the deaths have been observed.

Shkolnikov et al. (2003) used the Gini coefficient to measure the mortality compression (the coefficient was also listed, but not used, in Wilmoth and Horiuchi 1999). Shkolnikov et al. (2011) showed a close correlation between the Gini coefficient and the losses of expected lifetime, which, in turn, is equivalent to entropy (Mitra 1978, Vaupel 1986, Goldman and Lord 1986). As in other indices based on complete distributions of ages at death, the Gini coefficient may be computed for only a limited number of birth cohorts for whom such data exist.

Kannisto (2000, 2001) introduced two approaches to measuring mortality compression. His C-family of shortest age intervals covering C percent of the deaths is an extension of Wilmoth and Horiuchi's IQR. In his second approach, he followed Lexis' (1877) advice to distinguish between natural and premature deaths, with the latter deaths being responsible for most of the historical mortality compression. Since premature deaths are, conceptually, a phenomenon of transient effects that must cease once they are reduced to a possible minimum, Kannisto advocated measuring mortality compression by focusing on "natural" deaths. He also pointed, without providing any formal proof, to the presence of bias in compression indicators, like those used by Myers and Manton (1984) (and, more recently, by Engelman et al. 2010), which are based on studying age intervals left-censored at a fixed old age. Assuming the modal age at death, M, as the center of the distribution of "natural" deaths, Kannisto instead used the mode to left-censor the distribution of deaths in order to eliminate the effects of the premature deaths and the shifts in the distribution of deaths. Using the standard deviation from the mode of ages at

death above the mode, $SD(M+)$, he showed that mortality compression accompanied the continuing increase in life expectancy in 13 countries with good data. (He has also demonstrated empirically that $SD(M+)$, IQR, and C-family indicators have all changed in a similar direction.) The Lexis-Kannisto approach may be questioned in different ways: the concepts of premature and natural deaths may change over time; the distribution of deaths above the mode may deviate from the normal distribution; and the method may not be applied to birth cohorts who are not yet extinct, i.e., to any of the currently observed cohorts. Nonetheless, the approach has found many supporters and followers in the literature, and provides an interesting perspective on mortality dynamics.

Cheung et al. (2005) proposed distinguishing between three dimensions of change in the shape of the survival curve: horizontalization of the left-hand part of the curve due to declining deaths at young ages, verticalization of the curve around the modal age at death, and longevity extension corresponding to how far the highest life durations may exceed the modal age at death. Of these dimensions, the second one is most relevant to the mortality compression. Cheung and co-authors used the prolate ndex, or PI , developed by Eakin and Witten (1995) to measure the degree of verticalization. PI is the angle of the line connecting the points of maximum acceleration and deceleration in attrition around the modal age at death. It was also included in the set of indictors developed by Wilmoth and Horiuchi (1999). Based on data from Hong Kong, Cheung and co-authors demonstrated continuous mortality compression in 1976-2001 for both males and females. In more recent works, Cheung and Robine (2007) and Thatcher et al. (2010) turned to Kannisto's indicator $SD(M+)$, and presented evidence for mortality compression above the mode in recent data in six countries.

Edwards and Tuljapurkar (2005), Tuljapurkar and Edwards (2011), and Engelman et al. (2010) used an approach similar to that of Myers and Manton, considering standard deviations S_x of ages at death above selected fixed ages *x*. (Edwards and Tuljapurkar used S_{10} and Engelman et al. considered a variety of left-censoring ages.) From their analysis, Edwards and Tuljapurkar drew important conclusions about socioeconomic inequality and racial differences in mortality compression, yet their conclusion about the stalling of mortality compression since 1960 was, in part, driven by the bias of S_{10} (we consider the bias in Appendix A). The same applies to the conclusions of Engleman et al., who reported a moderate compression of period mortality at ages below 50 and decompression at older ages. If the bias were eliminated, S_x would show a compression of period mortality at all ages. Another limitation of these indicators is that it is not possible to compute them for birth cohorts with incomplete life histories.

Lynch and Brown (2001) developed a new approach to mortality compression by considering the slope of the mortality curve at the inflection point, and suggested that mortality has been decompressing since the 1960s. Unfortunately, the inflection of the mortality curve (currently around age 95) takes place at such an old age that the shape of the curve in its vicinity is not very informative about the distribution of most of the deaths; even above the mode (currently around age 85), the overwhelming majority of deaths occur before the inflection point of the mortality curve.

Other indicators have also been mentioned in the literature, but have never actually been used for studying compression; Wilmoth and Horiuchi (1999) and Cheung et al. (2005) present extended lists of such indicators. Notably, the alternative indicators are well correlated with each other (e.g., Wilmoth and Horiuchi 1999), which makes it possible to choose any of them as a preferred

indicator. However, all of those indicators also share limitations stemming from two important aspects of contemporary approaches to studying mortality compression. A desire to overcome these limitations motivated us to undertake this study.

First, researchers have recently been focusing on the compression of old-age deaths, which is not affected by declining infant and child, and, in many studies, young adult mortality. However, both methods used so far to separate the old-age mortality, censoring at a fixed or the modal age, have their limitations. When censoring at a fixed old age, such as 60, the compression indicators are severely affected by the built-in tendency to show *decompression* when the distribution of deaths is shifting to older ages (Kannisto 2001; see also Appendix A). Censoring at the modal age at death has a disadvantage as well, as this ignores a significant part of the mortality experience; besides, the underlying assumptions of this method may be questioned unless strong substantive evidence is found that distinguishes natural and premature deaths fitting the Lexis-Kannisto framework.

Second, most articles on mortality compression dealt with period life tables and reported a compression of the *life table* distribution of deaths. Although some authors (e.g., Wilmoth and Horiuchi 1999, Engelman et al. 2010) did study the compression of mortality from the cohort perspective, that line of research was limited because the results for cohorts who have completed their mortality history are already outdated. Meanwhile, these might be the most recent developments in mortality compression, which are of the greatest interest. As we have indicated elsewhere (Ediev 2011; see also the next section), however, compression is a built-in feature of the period life table model in the context of declining mortality; it may not indicate an actual compression of mortality for birth cohorts. The need to shed light on and overcome this limitation of the period life table model is our main motivation for conducting the current study.

2. When the period life table model (artificially) compresses the life span

The period life table model describes the current mortality as a combination of currently observed age-specific mortality rates, each of which in fact describes the mortality of a different birth cohort. When there is no systematic temporal change in mortality, the period life table provides a relevant picture of the mortality pattern for each and every cohort observed. When mortality changes over time, however, the period life table induces age-specific compressions or decompressions of the mortality schedule.

Consider the typical case of adult mortality declining with time and increasing with age. In Figure 1, the inclined strip bounded by two contour lines of the force of mortality (the instantaneous death rate $\mu(x,t)$, x standing for age and t – for time) represents the area in the Lexis diagram where the force of mortality increases from level **A** to level **B**. The positive inclination of the strip indicates that mortality increases with age, but declines with time: as time passes, the same level of mortality is observed at more and more advanced ages. Mortality is higher above the strip and lower below it. The synthetic cohort of the conventional period life table (represented by the vertical line in the diagram) is 'exposed' to the selected range of the force of mortality during the period indicated by the age interval $\mathbf{x_1x_2}$ in the figure. But the birth cohorts (represented by the bisector) are exposed to the same range of the force of mortality over a longer age interval $\mathbf{x_1x_3}$. By its very design, a conventional period life table cuts off the part of the cohorts' experience indicated by age interval $\mathbf{x_2x_3}$, which leads to both an overestimation and a compression of mortality in the life table.

Another way to note the artificiality of the mortality compression imposed by the period life table is to consider the conventional forecast under the 'constant mortality' scenario, when the forecast scenario applies after some period of mortality decline. As the period combination of mortality rates (the period life table) is considered to provide a full account of current mortality, the conventional constant mortality scenario assumes time-constant, age-specific mortality rates.

An illustrative example is given in Figure 2, where the arrows correspond to parts of the lifespan of birth cohorts falling in the three periods of time depicted by the three panels: time-invariant mortality prior to the observation period, declining mortality in the observation period, and the future mortality assumed to remain constant at the most recently observed levels. Levels of the force of mortality are denoted by capital letters. Thus, in the past, the force of mortality changed from level **A** to level **B** in the first age group, from level **B** to level **C** in the second age group, and so on. Because mortality is decreasing during the current observation period, the youngest depicted cohort starts with mortality level **A** and ends up with mortality level $\mathbf{B}^{\mathbf{1}}$, which is lower than the mortality level at the beginning of the observation period for the second-youngest cohort (**B**). By a similar logic, every cohort followed in the observation period ends up with a mortality level lower than that of the older cohort at the same age at the beginning of the observation. In the future, the common constant mortality scenario assumes mortality at the same levels of $\mathbf{A}^{\mathbf{1}}$, $\mathbf{B}^{\mathbf{1}}$, etc., at the same respective ages, as at the end of the observation period.

In spite of the intuitive soundness of the conventional scenario, a closer evaluation reveals the artificial compression of mortality within it. Consider, for example, the second age group in Figure 2, where the constant mortality scenario assumes that the force of mortality increases from level $\mathbf{B}^{\mathbf{1}}$ to level $\mathbf{C}^{\mathbf{1}}$. Already in the current year, however, the second-youngest cohort in the illustration has moved from a higher mortality level **B** to the same ultimate mortality level

$\mathbf{C}^{\mathbf{1}}$, while remaining in the same age group. Hence, contrary to intuition, the vitality of cohorts will deteriorate faster in the future than the vitality of cohorts currently observed: although they start off with better health conditions (as indicated by the lower force of mortality), cohorts in the 'future' do not end up being healthier than the current population by the end of the age group. This paradoxical implication of the time-constant mortality scenario may also be illustrated using *the life table aging rate* (Horiuchi and Coale 1990, Horiuchi and Wilmoth 1997); i.e., the rate of proportional increase of the force of mortality at given age: during times of mortality decline, the period life table and the constant mortality scenario will show a higher aging rate at any age than the cohort observed at that same age in that same period.

The conventional period life table mistakes the difference between the age when a birth cohort experiences a force of mortality $\mathbf{B}^{\mathbf{1}}$ and the age when another birth cohort experiences mortality level $\mathbf{C}^{\mathbf{1}}$ for the duration of *time* over which a (hypothetical) cohort moves from level $\mathbf{B}^{\mathbf{1}}$ to level $\mathbf{C}^{\mathbf{1}}$. However, a difference between ages indicates the time interval only when taken within one birth cohort. Replacing the actual time intervals over which birth cohorts are exposed to different mortality conditions by cross-cohort differences in age compresses the age pattern of mortality and accelerates aging when mortality declines over time.

The formal relationships between the period and cohort aging rates and the exposures (in years of age) to similar ranges of the death rate are presented in Appendix B (see also Ediev 2008). When the contour line of the force of mortality has a tangent slope *r*, the period life table shows the aging rate accelerated by $1-r$ times and the exposure durations to elementary ranges of the force mortality compressed by $1-r$ times as compared to the cohort schedules.

At this point we should comment on the link between the compression of the age pattern of the force of mortality, which the paper is primarily about, and the compression of the distribution of ages at death commonly considered in the literature. To an extent, the link has been explored in the literature. Tuljapurkar and Edwards (2011) have shown analytically, for a variety of mortality models, how the variance in age at adult death is related to the steepness of the mortality curve. Earlier, Pollard (1991) had shown that the standard deviation of the time to death is approximately reciprocal of the steepness of the curve of the death rate in the Gompertz model. The steepness of the curve of the death rate is, on the other hand, reciprocal of the exposure durations, in years of age, to a given range of the death rate. Hence, Pollard's and Tuljapurkar's and Edwards' results suggest that the standard deviation of time to death is in direct relation to the exposure durations studied here. At the modal age, a general analytical relation (Eq. (2) further down) has been established between the steepness of the mortality curve and the death rate, which is, in turn, proportional to the "fastest decline" described by Wilmoth and Horiuchi (1999), or the "highest proportion of deaths" described by Cheung et al., (2005). In Appendix C, we add a few other formal relations between the change in the exposure durations to ranges of the death rate and the distribution of ages at death. We show that inter-percentile ranges (of which Wilmoth and Horiuchi's (1999) IQR and Kannisto's (2000) C-family are special cases) change, at ages above 40, approximately in the same proportion as the exposure durations.

3. A new methodology for examining compression. Description of the data

In view of the limitations of the period life table model, our study seeks to contrast the compression in cohort and period age schedules of mortality. We recognize that the complete

picture of cohort mortality is usually available only after the data become significantly outdated, which presents challenges when studying contemporary developments in mortality. We therefore do not focus on the distributional characteristics of ages at death, as is usually done in the literature on mortality compression. Instead, our main method is based on examining the durations of exposure (in years of age) of birth cohorts (also as compared to period mortality schedules) to certain ranges of the death rate observed at old age. However, we will also consider, when possible in detail, the distributional characteristics of ages at death beyond and below the modal age at death, an issue which has attracted special attention in the literature (see Section 4).

To examine the compression of durations of exposure to various mortality levels, we have chosen seven ranges of the annual death rate prevailing at old age: death rates 0.01 to 0.011, 0.02 to 0.021, 0.05 to 0.051, 0.1 to 0.101, 0.15 to 0.151, 0.2 to 0.201, and 0.3 to 0.301. Such death rates are currently observed at ages of around 61 to 94 for males and of 68 to 96 for females in low-mortality countries, and go beyond the modal ages. While corresponding to different age ranges at different periods of time, the range of the death rate 0.01 to 0.301 used to cover 75%-85% of all female deaths at age 10 and older, and 80%-90% of male deaths at age 10 and older (currently about 85% for both sexes).

Because the duration of exposure, in years of age, to a given range of the death rate $m(x)$ might be a fraction of the year, while the data are provided in a discrete form, we need a procedure for estimating the exact duration of the exposure. To this end, we use a linear approximation of the death rate's logarithm in the vicinity of the age when the range is observed:

$$Expos\,[A;B] = \frac{\ln(A/B)}{b}, \quad (1)$$

where $Expos\,[A;B]$ is the estimated duration of exposure to the death rate ranging from A to B; b is the slope parameter of the regression line $\ln(m(x)) = a + bx$ fit in the age interval of at least nine years within which the given death rates were observed [2]. To reduce volatility due to small population size and lower data quality at oldest-old ages, we do not estimate the durations of exposure when the interval used to fit the regression extends beyond age 100; we also exclude a few cases with R^2 below 0.5.

We use data from the Human Mortality Database (2011), HMD, excluding the countries of the former Eastern Bloc, where the bias of the period life tables may have been the opposite of that of the rest of the countries represented in the database;[3] and also Iceland and Liechtenstein because of their small population size. Death rates from period life tables and from (partially incomplete) cohort mortality schedules of the HMD form the basis for our work. We study the dynamics of durations of exposure to the selected mortality levels over the long time period since 1900. For different years there are different numbers of countries with mortality data available in HMD, but this does not significantly affect our findings. The earliest and the latest years with death rates available for each of the countries studied are shown in Table 1; cohort-wise, (partially incomplete) sets of age-specific death rates are available starting three years later and ending one year earlier than the periods shown in Table 1 (exceptions are Belgium, Denmark,

[2] The regression resembles the Gompertz model of adult mortality. But we do not assume, as in the Gompertz model, that the slope parameter *b* is similar at all ages. Its equivalent, the *life table aging rate*, was shown to vary with age (Horiuchi and Coale 1990, Horiuchi and Wilmoth 1997).

[3] These countries showed declining life expectancy before and, in some cases, after the 1990s. In such situations, as follows from the results of the previous section, period life tables may be biased towards showing mortality decompression (indeed, Tesárková et al. 2010 report decompression of period mortality in Russia). Although the empirical part of our paper is focused on the case of increasing life expectancy, mortality (de)compression in the context of declining life expectancy may also be an important topic of study.

Finland, France, Italy, the Netherlands, Norway, Sweden, and Switzerland; for these countries cohort data are available starting from 1900).

In Table 1, we also present durations of exposure, in years of age, in selected period life tables to the death rate's range 0.01-0.301; this range comprises all seven smaller ranges of the death rate, which will be considered in more detail below. As indicated by the table, period mortality compression has been observed in all of the countries studied. It has slowed in recent years, but it does not seem to have stopped altogether, except in Chile, Finland, Ireland, Taiwan, and the U.S., where the durations of exposure in the male life tables have expanded slightly in recent periods. Our purpose here is to study the general tendencies in cohort and period mortality compression, and not the considerable cross-country variation. Therefore, in the following, we study mortality compression averaging durations of exposure and other indicators of compression for all of the selected 23 low-mortality countries.

In Figure 3, we present contour lines of the death rate corresponding to the lower ends of the selected ranges of the rate. As mortality was characterized by a declining trend in the past century in the low-mortality countries, the ages at which the selected mortality levels were observed used to move to older ages with time (this process accelerated in the 1970s). In such a situation, our above discussion indicates the bias of the mortality compression picture revealed by the period life tables. The ages at which the selected mortality levels are observed have shifted by about 0.13 years per year on average in recent decades. The formal theory (Appendix B, Ediev 2008) indicates that the period life table will compress the actual exposure durations to the selected ranges of the death rate by about 1/(1-0.13)=1.15, or 15%. This would be an average contemporary bias of the period life table estimates for the mortality compression indicators. In the next section, we will provide a more explicit and comprehensive picture of these differential

dynamics by considering cohort- and period-specific durations of exposure to selected mortality levels.

4. Empirical evidence on mortality (de)compression

The development over time of durations of exposure to the selected ranges of the death rate is presented in Figure 4 and Table 2. The overall trend after the 1960s was a decompression of cohort mortality. For males, at death rates 0.05 or higher—i.e., at around ages 70 and older—mortality has been decompressing cohort-wise since 1900. For females, a decompression of cohort mortality is observed at death rates below 0.05 (currently at ages below 80). At higher death rates, the durations of exposure, in years of age, of female cohorts may be characterized as moderately compressing or stable. Males are exposed over longer (and still expanding) durations of time to the selected mortality levels. However, they experience those levels at younger ages than females, which is consistent with male lower life expectancy.

Period life tables, on the other hand, indicate a continuous mortality compression during the whole period after 1900, except for the very recent developments in male mortality at lower mortality levels. In the most recent decade, the estimates of the exposures, in years if age, to the selected ranges of the death rate were, on average, about 18% shorter when obtained from period life tables than the estimates based on cohort data. This discrepancy is well in line with the rough estimate above (15%) derived from formal relations.

Over the last century, the age at which the selected mortality levels were experienced has shifted considerably. The death rate experienced by males at age 40 a century ago is now experienced at age 60 in low-mortality countries. At age 65, females are exposed to the mortality level they

would have been exposed to at age 45 a century ago. Since this process took place at different speeds in different populations, it may be informative to examine the exposure durations arranged as functions of the age when the respective ranges of the death rate were first experienced (Figure 5 and Tables 3 and 4). The exposure durations used to be longer when the selected ranges of the death rate were observed to older ages for males, and they were only moderately shorter for females. Period life tables would, nonetheless, show a pronounced mortality compression.

5. Mortality compression in relation to the mode

In this section, given our special interest in the literature on compression in relation to the modal age at death, we supplement our study with an analysis of the cohort and period distributions of ages at death above and below the modal age.

Although conceptually straightforward, finding the modal age at death may in practice be complicated by irregularities of the life table distribution of ages at death and by the need for computing the mode in fractions of a year while using a discrete life table. We estimate the mode using the formal relation equating the log-derivative of the force of mortality to the force of mortality itself at the mode:

$$\frac{d}{dx}\ln\mu(x)-\mu(x)=0 \text{ at } x=M \text{ . (2)}$$

(The relation was derived, in the context of the Gompertz law, by Pollard, 1991; it was rediscovered later as a general relation by Canudas-Romo, 2008, Thatcher et al., 2010, and Tuljapurkar and Edwards, 2011). We approximate the derivative in (2) by discrete increments.

Empirical estimates of the expression (2) are rather volatile around the mode (especially in cases of multiple modes). Hence, we smooth the expression by quadratic parabola applied to the age interval 60 to 95 years, and discard data whenever the fit of the parabola is not good enough (R^2 less than 0.5).

a. Mortality compression above the mode

Following the established practice, we measure the compression above the mode using the upward standard deviation from the mode of age at death above the mode:

$$SD(M+) = \sqrt{\frac{\int_{M}^{\infty} d(x)(x-M)^2 dx}{\int_{M}^{\infty} d(x)dx}} \text{ (years), (3)}$$

where $d(x)$ is the density function of the life table distribution of deaths. In practice, we use discrete approximation by using life table numbers of deaths d_x at ages x to x+1 and assuming deaths are uniformly distributed within the age groups; we compute the SD(M+) only for cohorts with death rates available up to at least age 101.

The dynamics of the standard deviation from the mode of age at death above the mode are presented in Figure 6. For males, the cohorts with data complete enough for obtaining the SD(M+) show mortality *decompression* above the mode since the 1900s. Meanwhile, decompression of period mortality above the mode in the 1950s and1960s was more than compensated for by continuous compression later on. For females, the cohorts do not show a

noticeable trend toward compression or decompression since 1900, while the period life table model indicates continuous compression above the mode since 1900.

b. Mortality compression below the mode

Deaths above the mode constitute only about one-third of deaths above age 30 and may not be representative of all adult deaths. That is why we supplement the conventional analysis of the compression above the mode by an analysis of the compression below the mode. To eliminate effects due to younger-age mortality (presumably, the premature deaths in the Lexis-Kannisto approach) and widening the observation window (in the case of left-censoring at a fixed age), we consider the distributions of ages at death in an age interval of a fixed width (30 years) below the mode:

$$SD({}_{30}M) = \sqrt{\frac{\int_{M-30}^{M} d(x)(x-M)^2\,dx}{\int_{M-30}^{M} d(x)dx}} \text{ (years). (4)}$$

We have set the width (30 years) of the age frame to about three SD(M+) so that it would be possible to catch in the frame most of the 'normal' deaths below the mode. At the same time, the frame is short enough that it does not cover mortality at younger adult ages, as senescence may be less important and cohort data may be less available for people in these age groups (with a modal age of 75 or higher, the left end of the frame is set at age 45 or older).

The dynamics of $SD({}_{30}M)$ are also presented in Figure 6. As in the cases considered above, the mortality compression below the mode is read differently from period and cohort records. The

period life tables suggest continuous compression below the mode for both males and females (it may have recently leveled off for males). This differs from the experience of birth cohorts: mortality below the mode has been decompressing for males and largely stagnating for females since the 1970s.

6. Conclusions

Mortality compression cannot be studied through the dynamics of period life tables, which by their very design tend to provide a compressed picture of the human life span when mortality is on the decline. The conventional reliance on period data, as well as the limitations of age-censoring methods, have contributed to contradictory findings in the literature. This reliance cannot be changed easily, as almost none of the compression indicators used in the literature can be computed for birth cohorts with incomplete data.

Our method, based on the estimation of the durations of exposure, in years of age, to selected ranges of the death rate, shows mortality decompression—not compression—have characterized mortality dynamics in 23 low-mortality countries since the 1960s, i.e., in the period during which the greatest reductions in old-age mortality have been achieved.

Even though the selected mortality levels were observed at increasingly old ages, this did not result in a significant compression of the durations of exposure to them; for males, the mortality curve is decompressing while shifting to an older age.

Patterns of decompression or stability, cohort-wise, of the exposure durations may indicate a future mortality decline at oldest-old ages that has not been observed so far only because the cohorts that will be involved in such a decline are still young.

Our findings support the view (Ediev 2011) that the durations of exposure, in years of age, of cohorts to currently observed mortality levels form a genuine part of current mortality conditions, and should be taken into account when formulating future mortality scenarios.

Although deaths above the modal age at death form only a relatively small fraction of the distribution of deaths, they may tell important stories about mortality at ages approaching the end of the human lifespan. Compression above the mode is indicative of an important aspect of mortality compression which attracted special attention in the literature—the rectangularization of the survival curve. However, the cohort data available so far do not support a compression of the distribution of ages at death, even above the mode. For males, there was a decompression above the mode since 1900. For females, deviation from the mode of the ages at death above the mode has been stagnating since 1900.

Below the mode, mortality was compressing until the 1970s. Since then, cohort-wise, the distribution of ages at death below the mode has been decompressing for males and has been stable for females. By contrast, the period life table model, due to built-in biases, suggests an overall compression of the distribution of ages at death above and below the mode.

Unlike most of the previous works on mortality compression, we do not come to the conclusion that a concentration of mortality in narrow age intervals will occur at some old age in the future. Rather, deaths seem to stay dispersed over ages, while shifting towards ever more advanced ages.

Appendix A. Biasness of compression indicators derived from the distribution of ages at death left-censored at a fixed age

Consider a reference model in which the age distribution of adult deaths $d(x,t)$ is shifted along the age scale at the speed r years per year. A good indicator of compression of the distribution of ages at death should indicate neither compression nor decompression in the reference model. A shift of the distribution of adult deaths is accompanied by a shift of life table functions, as long as those functions do not depend on infant and child mortality; in particular, this applies to the remaining life expectancy $e(x,t)$ and the standard deviation of age at death $S(x,t)$ above age x. The shifting process may be formalized, for $S(x,t)$, by the relation:

$$S(x(t),t) \equiv const \text{ at } x(t) = x(0) + rt \tag{A1.1}$$

or, differentiating by time and rearranging,

$$\frac{\partial}{\partial t} S(x,t) = -r \frac{\partial}{\partial x} S(x,t). \tag{A1.2}$$

It follows immediately that the standard deviation of the age at death above fixed age x will not show (de)compression in the case of shifting mortality only when the standard deviation does not depend on age. However, Engleman et al. (2010) showed that the standard deviation $S(x,t)$ is a decreasing function of age. Combined with (A1.2), this implies that the standard deviation will increase when mortality shifts to older age; it will be biased towards showing mortality decompression. The time derivative (A1.2) may be used as a quantitative measure of the bias of $S(x,t)$ as a mortality compression indicator. To study the bias analytically, we obtain the derivative by age in (A1.2) from the common expression for the variance:

$$S^2(x,t)=\frac{\int_x^{\omega}(y-[x+e(x,t)])^2 d(y,t)dy}{\int_x^{\omega} d(y,t)dy}. \qquad \text{(A1.3)}$$

Taking derivative of (A1.3),

$$\frac{\partial}{\partial x}S^2(x,t)=\frac{\frac{\partial}{\partial x}\int_x^{\omega}(y-[x+e(x,t)])^2 d(y,t)dy}{\int_x^{\omega} d(y,t)dy}-\frac{\int_x^{\omega}(y-[x+e(x,t)])^2 d(y,t)dy}{\left[\int_x^{\omega} d(y,t)dy\right]^2}\frac{\partial}{\partial x}\int_x^{\omega} d(y,t)dy=$$

$$=\frac{-e^2(x,t)d(x,t)+\frac{\partial}{\partial x}\int_x^{\omega}(y-[x+e(x,t)])^2 d(y,t)dy}{\int_x^{\omega} d(y,t)dy}-\frac{\int_x^{\omega}(y-[x+e(x,t)])^2 d(y,t)dy}{\left[\int_x^{\omega} d(y,t)dy\right]^2}(-d(x,t))=$$

/note: $\mu(x,t)=\frac{d(x,t)}{\int_x^{\omega} d(y,t)dy}$ is the force of mortality and we use (A1.3)/

$$=\frac{2\frac{\partial}{\partial x}[x+e(x,t)]\int_x^{\omega}2(y-[x+e(x,t)])d(y,t)dy}{\int_x^{\omega} d(y,t)dy}-\mu(x,t)\left(e^2(x,t)-S^2(x,t)\right)=$$

/note: $\frac{\int_x^{\omega}(y-[x+e(x,t)])d(y,t)dy}{\int_x^{\omega} d(y,t)dy}=\frac{\int_x^{\omega} yd(y,t)dy}{\int_x^{\omega} d(y,t)dy}-[x+e(x,t)]=x+e(x,t)-[x+e(x,t)]=0$ /

$$=-\mu(x,t)\left(e^2(x,t)-S^2(x,t)\right).$$

Hence, $2S(x,t)\frac{\partial}{\partial x}S(x,t)=\frac{\partial}{\partial x}S^2(x,t)=-\mu(x,t)\left(e^2(x,t)-S^2(x,t)\right)$. It follows, then, that

$$\frac{\partial}{\partial x} S(x,t) = -\mu(x,t) \frac{e^2(x,t) - S^2(x,t)}{2S(x,t)}, \tag{A1.4}$$

where $\mu(x,t) = \frac{d(x,t)}{\int_x^{\omega} d(y,t)dy}$ is the force of mortality (the derivation may be requested from the author). The time derivative (A1.2), or the bias of the standard deviation as a measure of mortality compression, then equals

$$\frac{\partial}{\partial t} S(x,t) = r\mu(x,t) \frac{e^2(x,t) - S^2(x,t)}{2S(x,t)}. \tag{A1.5}$$

Our general analytical results are as follows:

The change of the standard deviation $S(x,t)$ of age at death, left-censored at fixed age x in the shift scenario, is a function of the difference between the remaining life expectancy at age x and the standard deviation itself: when the difference is positive, the standard deviation will increase (indicating mortality decompression); when the difference is negative, the standard deviation will decline (indicating mortality compression); only if the hazard rate at the censoring age equals zero will the standard deviation not change over time irrespective of the difference.

In the exponential distribution of time to death, where the hazard rate is age-independent, the remaining life expectancy and the standard deviation are equal. In such a case, $S(x,t)$ would not change over time, and would show neither a compression nor a decompression of the distribution of deaths in the shifting model; it would behave as a 'good' indicator of mortality compression. The exponential distribution is, however, a very special one. It has the maximum entropy of the distribution of ages at death (Kagan et. al 1973, Demetrius 1978, 1979); i.e., it is,

in a sense, the least compressed of all the distributions. For realistic, more concentrated distributions, $S(x,t)$ will usually indicate 'decompression' in the shifting scenario.

To make the relations above more practical, we should approximate the shift parameter r. Assuming the shift model holds true, one option is to apply it to the remaining life expectancy at age x: $r\frac{\partial}{\partial x}e(x,t)+\frac{\partial}{\partial t}e(x,t)\equiv 0$. That would produce $r=-\frac{\frac{\partial}{\partial t}e(x,t)}{\frac{\partial}{\partial x}e(x,t)}$ and, combined with (A1.2), provide the approximation to the bias:

$$\frac{\partial}{\partial t}S(x,t)=\frac{\frac{\partial}{\partial t}e(x,t)}{\frac{\partial}{\partial x}e(x,t)}\frac{\partial}{\partial x}S(x,t). \tag{A1.6}$$

The standard deviation's change rate can then be adjusted for the bias:

$$\left[\frac{\partial}{\partial t}S(x,t)\right]^{*}=\frac{\partial}{\partial t}S(x,t)-\frac{\frac{\partial}{\partial t}e(x,t)}{\frac{\partial}{\partial x}e(x,t)}\frac{\partial}{\partial x}S(x,t). \tag{A1.7}$$

If the assumption about the mortality shift holds true, the adjusted derivative (A1.7) equals zero. If the shift is, however, combined with the additional transformation of the mortality curve, the approximated shift parameter *r* may be distorted and the adjusted change (A1.7) may remain biased. Another limitation of the adjustment is the inability to obtain an unbiased indicator $S^{*}(x,t)$ by integrating (A1.7) over long time intervals: adjusted derivatives at different moments in time might be interpreted as being derivatives of different incomparable measures of variance (each of them corresponding to different censoring ages). Nonetheless, the approximation above may still be useful in showing the bias of the mortality compression indicator $S(x,t)$ under the 'null hypothesis' of mortality shift.

In Figure A1, we present empirical estimates of the bias (A1.7) using period female life tables for low-mortality countries from the HMD. The bias is towards decompression at any given left-censoring age *x*. (The bias would be even higher for males.) Unlike the studies that

used such biased mortality compression indicators, the adjusted indicator shows mortality compression above any age, except for the oldest-old ages. In particular, the compression of period mortality is strong at ages above 50, where Meyers and Manton and Engelman et al. report decompression.

Appendix B. Relation between period and cohort aging rates and exposures (in years of age) to similar ranges of the death rate

Let $\mu(x,t)$ be the force of mortality as a function of age *x* and time *t* and $\mu_C(x,c)=\mu(x,c+x)$ be the force of mortality as a function of age *x* and cohort *c*. The life table aging rate (Horiuchi and Coale 1990, Horiuchi and Wilmoth 1997) calculated from the mortality schedule of the cohort aged *x* at time *t* is (we use the conventional notations for full and partial derivatives; the full derivative over *x* is the sum of both partial derivatives multiplied by the derivatives over *x* of the expressions replacing each of the variables):

$$k_C(x,t-x)\overset{def}{=}\tfrac{\partial}{\partial x}\ln\mu_C(x,t-x)=\tfrac{d}{dx}\ln\mu_C(x,t-x)+\tfrac{\partial}{\partial t}\ln\mu_C(x,t-x)=$$

$$=\tfrac{\partial}{\partial x}\ln\mu(x,t)+\tfrac{\partial}{\partial t}\ln\mu(x,t)=k(x,t)\left[1+\frac{\frac{\partial}{\partial t}\mu(x,t)}{\frac{\partial}{\partial x}\mu(x,t)}\right], \qquad \text{(A2.1)}$$

where $k(x,t)=\tfrac{\partial}{\partial x}\ln\mu(x,t)$ is the aging rate of the period life table at time *t*. When mortality is increasing by age ($\tfrac{\partial}{\partial x}\mu(x,t)>0$) and declining by time ($\tfrac{\partial}{\partial t}\mu(x,t)<0$), the expression in parenthesis in (A2.1) is less than one; i.e., the cohort aging rate is lower than the period life table aging rate. The expression in the parenthesis may be linked to the slope of the contour line $x=y(t)$ of the force of mortality, where:

$$\mu(y(t),t)\equiv const\,. \qquad \text{(A2.2)}$$

Differentiating by *t*,

$$\tfrac{d}{dt}y(t)\tfrac{\partial}{\partial x}\mu(y(t),t)+\tfrac{\partial}{\partial t}\mu(y(t),t)=0\,. \qquad \text{(A2.3)}$$

Hence, the tangent slope of the contour line $r=\tfrac{d}{dt}y(t)$ equals

$$r = -\frac{\frac{\partial}{\partial t}\mu(y(t),t)}{\frac{\partial}{\partial x}\mu(y(t),t)}. \tag{A2.4}$$

Substituting this into (A2.1),

$$k_C(x,t-x) = k(x,t)[1-r(x,t)], \tag{A2.5}$$

where $r(x,t)$ is the tangent slope of the contour line passing at age *x* at time *t*.

Relation (A2.5) is also informative about period-cohort differences in exposure durations (in years of age) to similar elementary ranges of the force of mortality. The exposure duration is reciprocal to the age derivative of the force of mortality; i.e., also to the aging rate. Hence:

$$\varepsilon_C(\mu(x,t),t-x) = \frac{\varepsilon(\mu(x,t),t)}{1-r(x,t)}, \tag{A2.6}$$

where $\varepsilon(\cdot)$ is the exposure, in years of age, to an elementary unit range of the force of mortality around the value $\mu(x,t)$ observed at age *x* at time *t*; the subscript "C" is applied to the cohort indicator

Appendix C. Compression of the age pattern of the mortality curve and the distribution of ages at death

Let $\varepsilon(u)$ and $\varepsilon_0(u)$ be exposures, in years of age, to the elementary unit range of the force of mortality around the value u in two mortality curves $\mu(x)$ and $\mu_0(x)$, which we label as the 'transformed' and the 'reference' curves, respectively. (The curves may represent, for example, period and cohort schedules or schedules at two periods of time/birth cohorts.) Our purpose in this note is to explore how differences in the exposures lead to differences in the distributions of ages at death between the reference and transformed schedules.

We study the scenario of *uniform* compression/decompression, when the exposures in the transformed schedule are related to the reference exposures by a constant decompression coefficient:

$$\varepsilon(u) = \alpha\varepsilon_0(u). \tag{A3.1}$$

If $\alpha > 1$, the transformed mortality curve is decompressed; it is compressed when $\alpha < 1$; the curves are similar when $\alpha = 1$.

Without a loss of the generality of the results for the compression of adult mortality, we also introduce a simplifying assumption that the mortality curves span over ages zero to infinity increase monotonically with age, and start at $\mu_0(0) = \mu(0) = u_0$. This may be interpreted as a shift of the origin of the age axis to the adult age at which the death rate reaches the level u_0; it follows, then, that our results will be concerned with mortality compression above the mentioned adult age. Alternatively, one may think of $\mu(x)$ and $\mu_0(x)$ as being free of infant and child mortality components, so that $\mu_0(0) = \mu(0) = 0$.

Given the simplifying assumption above, the ages at which the force of mortality reaches a given level *u* may be obtained as

$$X_0(u) = \int_0^u \varepsilon_0(w)dw,$$

$$X(u) = \int_0^u \varepsilon(w)dw = \int_0^u \alpha\varepsilon_0(w)dw = \alpha X_0(u), \tag{A3.2}$$

Hence, the decompression coefficient, in line with intuition, determines the stretch (compression when $\alpha < 1$) of the age scale of the transformed mortality curve as compared to the reference curve. It follows immediately that

$$\mu(x) = \mu_0\left(\frac{x}{\alpha}\right). \tag{A3.3}$$

Now we can derive the relation between the reference and transformed survival functions:

$$l(x) = \exp\left(-\int_0^x \mu(y)dy\right) = \exp\left(-\int_0^x \mu_0\left(\frac{y}{\alpha}\right)dy\right) = \exp\left(-\alpha\int_0^{x/\alpha} \mu_0(t)dt\right) = \left[l_0\left(\frac{x}{\alpha}\right)\right]^\alpha. \tag{A3.4}$$

This relation reveals two effects of the decompression of the exposure durations on proportions surviving to a given age. First, as indicated by the argument on the right-hand side, the stretch/compression of the age scale of the mortality curve has a similar effect on the age scale of the survival function. Second, however, the power on the right-hand side indicates that the stretching/compression effect is counterbalanced by a reduction/increase in survival due to longer/shorter exposure durations at all levels of the death rate. To assess the combined effect of these two processes on the distribution of ages at death, let us consider the ages $Y(\lambda)$, $Y_0(\lambda)$ at which the survival functions reach a given level λ (these would be $100(1-\lambda)$th percentiles of the distributions of ages at death). Using (A3.4),

$$\lambda = l(Y(\lambda)) = \left[l_0\left(\frac{Y(\lambda)}{\alpha} \right) \right]^{\alpha}. \tag{A3.5}$$

Rearranging the expression, $l_0\left(\frac{Y(\lambda)}{\alpha} \right) = \lambda^{1/\alpha}$, $\frac{Y(\lambda)}{\alpha} = Y_0\left(\lambda^{1/\alpha} \right)$, and

$$Y(\lambda) = \alpha Y_0\left(\lambda^{1/\alpha} \right). \tag{A3.6}$$

Expanding by the Taylor series, with respect to α at $\alpha = 1$, the first-order approximation of the transformed percentiles may be derived as

$$Y(\lambda) \approx Y_0(\lambda) + \left. \frac{d\left(\alpha Y_0\left(\lambda^{1/\alpha} \right) \right)}{d\alpha} \right|_{\alpha=1} \cdot (\alpha - 1) = Y_0(\lambda) + (\alpha - 1) \cdot \left[Y_0(\lambda) - \frac{dY_0(\lambda)}{d\lambda} \lambda \ln \lambda \right]. \tag{A3.7}$$

$Y_0(\lambda)$ is function inverse to $l_0(x)$, i.e., $\frac{dY_0(\lambda)}{d\lambda} = \left[\frac{dl_0}{dx}(Y_0(\lambda)) \right]^{-1}$. Substituting this in (A3.7) and noting that $\lambda^{-1} \frac{dl_0}{dx}(Y_0(\lambda)) = (l_0(Y_0(\lambda)))^{-1} \frac{dl_0}{dx}(Y_0(\lambda)) = -\mu_0(Y_0(\lambda))$ and that $\ln \lambda = \ln(l_0(Y_0(\lambda))) = -\int_0^{Y_0(\lambda)} \mu_0(x) dx$,

$$Y(\lambda) \approx \alpha Y_0(\lambda) - (\alpha - 1) \cdot \int_0^{Y_0(\lambda)} \frac{\mu_0(x)}{\mu_0(Y_0(\lambda))} dx. \tag{A3.8}$$

In the Gompertz model, $\mu_0(x) = u_0 \exp(kx)$, assuming that $\mu_0(Y_0(\lambda))$ is much higher than u_0, the last integral,

$$\int_0^{Y_0(\lambda)} \frac{\mu_0(x)}{\mu_0(Y_0(\lambda))} dx = \frac{1 - \exp(k(-Y_0(\lambda)))}{k} = \frac{1 - \frac{u_0}{\mu_0(Y_0(\lambda))}}{k} \approx \frac{1}{k}, \tag{A3.9}$$

is approximately constant. Although not perfect, the Gompertz model describes well the overall development of the death rate at ages 30-90 (e.g., Wetterstrand 1981). In our HMD data, the

expression (A3.9) deviated by less than 25% from the Gompertz estimate at all ages $Y_0(\lambda)$ above 40, for both females and males. Therefore, the multiplier in the second summand in (A3.8) may be taken as approximately constant G at adult ages (empirically, $G \approx 10$):

$$Y(\lambda) \approx \alpha Y_0(\lambda) - (\alpha - 1) \cdot G. \qquad \text{(A3.10)}$$

Hence, the stretch of the age scale of the survival function will, in the first-order approximation, be partly compensated for at ages of about 40 and older by an age-independent shift in the opposite direction (the compensation will be smaller at younger ages). The age-independent shift will, however, have no consequences on indicators of the compression of the distribution of ages at death, such as IQR or S_x. Hence, the compression/decompression of the exposure durations, in years of age, to ranges of the death rate will, in the first-order approximation, produce a similar compression/decompression of the distribution of adult ages at death.

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Table 1. Data availability and durations of exposure, in years of age, of period life tables to the range 0.01-0.301 of the death rate at selected periods of time

Population	First year of data (T_1)	Latest year of data (T_2)	Exposure in T_1	Exposure in 1990	Exposure in 2000	Exposure in T_2
			Females			
Australia	**1921**	**2007**	44.9	31.6	28.4	27.1
Austria	**1947**	**2008**	32.7	28.2	26.5	25.3
Belgium	**1900**	**2009**	38.8	29.1	27.2	26.9
Canada	**1921**	**2007**	42.2	32.3	30.5	30.2
Chile	**1992**	**2005**	34.5	34.5	33.5	32.9
Denmark	**1900**	**2008**	38.1	35.1	34.6	31.1
Finland	**1900**	**2009**	39.5	29.2	26.3	25.6
France	**1900**	**2007**	42.7	27.2	26.6	25.9
Ireland	**1950**	**2009**	37.8	32.2	30.0	29.4
Israel	**1983**	**2008**	34.3	34.5	31.6	30.3
Italy	**1900**	**2007**	38.0	28.5	27.4	26.0
Japan	**1947**	**2009**	38.8	26.7	27.5	26.5
Netherlands	**1900**	**2008**	38.8	28.9	28.4	27.2
New Zealand	**1948**	**2008**	37.6	33.7	31.0	28.6
Norway	**1900**	**2009**	42.3	30.6	27.6	26.8
Portugal	**1940**	**2009**	36.5	29.1	27.9	24.9
Spain	**1908**	**2006**	48.0	27.3	26.3	25.7

Population	First year of data (T_1)	Latest year of data (T_2)	Exposure in T_1	Exposure in 1990	Exposure in 2000	Exposure in T_2
Sweden	**1900**	**2008**	37.5	28.4	27.5	26.5
Switzerland	**1900**	**2007**	39.4	27.1	25.8	25.5
Taiwan	**1970**	**2009**	36.4	33.3	32.3	31.3
The United States	**1933**	**2007**	45.2	35.4	33.5	33.2
United Kingdom	**1922**	**2009**	38.5	33.9	31.3	29.9
West Germany	**1956**	**2008**	30.5	28.8	27.9	25.4
Average for females				**30.7**	**29.0**	**27.6**
			Males			
Australia	**1921**	**2007**	46.6	36.3	32.1	31.3
Austria	**1947**	**2008**	38.6	36.2	34.6	33.1
Belgium	**1900**	**2009**	45.6	35.3	33.6	33.3
Canada	**1921**	**2007**	42.1	37.0	34.2	33.6
Chile	**1992**	**2005**	39.8	39.8	39.5	40.0
Denmark	**1900**	**2008**	42.2	37.8	35.3	33.5
Finland	**1900**	**2009**	44.7	37.6	34.0	34.7
France	**1900**	**2007**	50.1	37.7	35.1	34.3
Ireland	**1950**	**2009**	38.2	35.4	33.0	33.1
Israel	**1983**	**2008**	39.4	39.0	35.9	35.1
Italy	**1900**	**2007**	48.4	36.2	33.4	31.5

Population	First year of data (T_1)	Latest year of data (T_2)	Exposure in T_1	Exposure in 1990	Exposure in 2000	Exposure in T_2
Japan	**1947**	**2009**	42.3	33.3	33.8	33.1
Netherlands	**1900**	**2008**	42.0	35.3	32.7	30.4
New Zealand	**1948**	**2008**	38.9	37.1	33.8	32.0
Norway	**1900**	**2009**	44.5	36.5	31.5	30.2
Portugal	**1940**	**2009**	42.1	35.9	34.2	32.4
Spain	**1908**	**2006**	46.6	35.3	34.5	34.0
Sweden	**1900**	**2008**	43.6	33.7	31.1	30.1
Switzerland	**1900**	**2007**	48.3	33.7	32.2	31.5
Taiwan	**1970**	**2009**	41.0	37.6	38.0	40.3
The United States	**1933**	**2007**	48.4	39.7	36.6	36.9
United Kingdom	**1922**	**2009**	40.0	36.4	34.3	33.8
West Germany	**1956**	**2008**	36.6	35.5	34.3	33.3
Average for males				**36.4**	**34.2**	**33.4**

Table 2. Durations of exposure, in years of age, of birth cohorts (left panel) and period life tables (right panel) to selected ranges of the death rate; the durations of exposure are averaged over 23 currently low-mortality countries and selected periods of time.

Range of the death rate	**Birth cohorts' actual exposure to the selected ranges of the death rate in periods:**			**Exposure estimates from period life tables**		
	1900-1909	**1960-1969**	**1997-2006**	**1900-1909**	**1960-1969**	**1997-2006**
	Males					
0.01-0.011	2.25	0.99	1.41	1.90	0.95	1.05
0.02-0.021	0.74	0.52	0.74	0.71	0.50	0.49
0.05-0.051	0.24	0.22	0.26	0.23	0.23	0.19
0.10-0.101	0.11	0.12	0.12	0.11	0.11	0.10
0.15-0.151	0.073	0.078	0.081	0.072	0.073	0.066
0.20-0.201	0.056	0.060	0.062	0.055	0.057	0.051
0.30-0.301	0.043	0.047	0.044	0.041	0.044	0.038
	Females					
0.01-0.011	2.18	1.11	1.24	1.83	0.98	0.94
0.02-0.021	0.62	0.52	0.55	0.56	0.46	0.43
0.05-0.051	0.21	0.19	0.19	0.20	0.17	0.16
0.10-0.101	0.11	0.10	0.09	0.10	0.09	0.08
0.15-0.151	0.074	0.073	0.065	0.074	0.066	0.057
0.20-0.201	0.059	0.061	0.052	0.059	0.056	0.046
0.30-0.301	0.045	0.045	0.039	0.045	0.041	0.036

Table 3. Durations of exposure, in years of age, of *male* birth cohorts and period life tables to selected ranges of the death rate; the durations of exposure are averaged over 23 currently low-mortality countries and selected ranges of age when the range of the death rate was first experienced (the data cover the entire period since 1900).

Range of the death rate	Age: 40-45	45-50	50-55	55-60	60-65	65-70	70-75	75-80	80-85	85-90	90-95	95-100
	Males, cohorts											
0.01-0.011	1.84	1.54	1.17	1.24	1.33							
0.02-0.021		0.62 [a]	0.75	0.62	0.61	0.70						
0.05-0.051					0.26	0.24	0.24	0.25				
0.10-0.101							0.11	0.12	0.12			
0.15-0.151							0.061 [a]	0.077	0.080	0.081		
0.20-0.201							0.050 [a]	0.051 [a]	0.058	0.063	0.070	
0.30-0.301										0.043	0.047	0.053
	Males, period life tables											
0.01-0.011	1.82	1.42	1.05	1.01	0.97							
0.02-0.021		2.12 [a]	0.73	0.58	0.53	0.50	0.49					
0.05-0.051				0.61 [a]	0.26	0.23	0.21	0.19				
0.10-0.101							0.11	0.11	0.10	0.10 [a]		
0.15-0.151								0.075	0.074	0.069		
0.20-0.201								0.067 [a]	0.056	0.057	0.055	
0.30-0.301									0.040 [a]	0.042	0.043	0.049

[a] Values might be affected by a small number of cases (three or fewer country cases)

Table 4. Durations of exposure, in years of age, of *female* birth cohorts and period life tables to selected ranges of the death rate; the durations of exposure are averaged over 23 currently low-mortality countries and selected ranges of age when the range of the death rate was first experienced (the data cover the entire period since 1900).

Range of the death rate	Age: 40-45	45-50	50-55	55-60	60-65	65-70	70-75	75-80	80-85	85-90	90-95	95-100
	Females, cohorts											
0.01-0.011	3.62 [a]	1.87	1.67	1.29	1.20	1.21	1.18 [a]					
0.02-0.021				0.63	0.60	0.55	0.53	0.51				
0.05-0.051						0.21	0.20	0.20	0.18			
0.10-0.101							0.11	0.11	0.11	0.094		
0.15-0.151								0.081	0.073	0.071	0.068	
0.20-0.201									0.058	0.061	0.054	0.056 [a]
0.30-0.301										0.043	0.044	0.045
	Females, period life tables											
0.01-0.011	2.83	1.75	1.45	1.06	0.98	0.93	0.86					
0.02-0.021			0.53 [a]	0.56	0.52	0.46	0.42	0.39				
0.05-0.051					0.19 [a]	0.20	0.18	0.17	0.15			
0.10-0.101							0.10	0.10	0.091	0.082		
0.15-0.151								0.076	0.069	0.062	0.058	
0.20-0.201									0.057	0.056	0.049	0.046
0.30-0.301										0.042	0.041	0.040

[a] Values might be affected by small number of cases (three or fewer country cases)

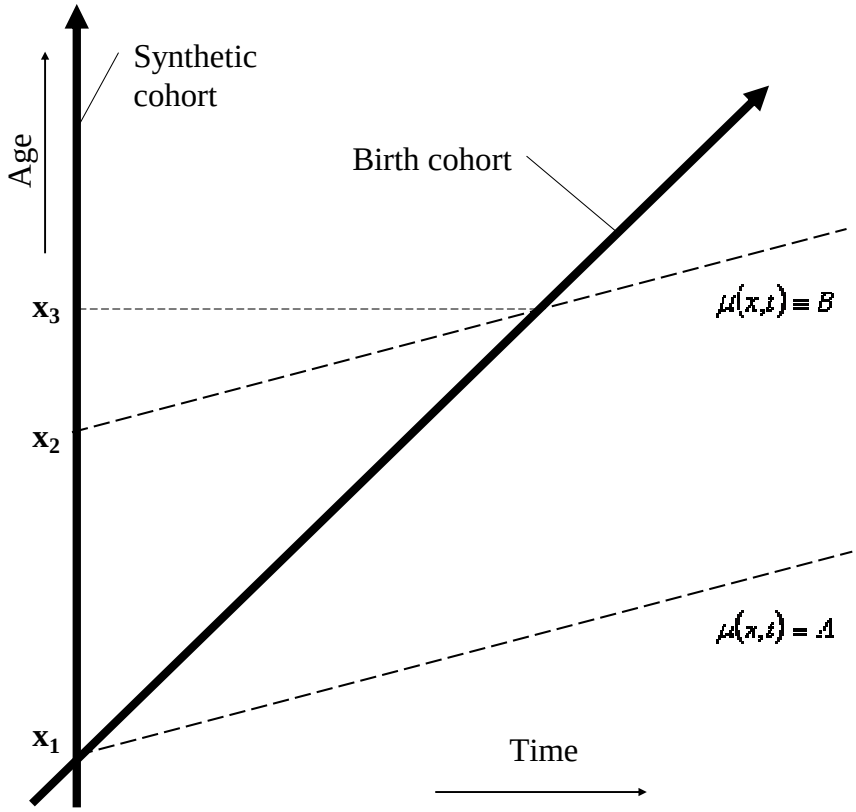


Figure 1. Illustration of different exposures (in years of age) of the birth cohort and of the synthetic cohort of the period life table to a given range of the force of mortality (the instantaneous death rate) when mortality is declining with time but increasing with age. The two inclined dashed lines are contour lines of the force of mortality and bound the area where the force of mortality increases from level 'A' to level 'B'.

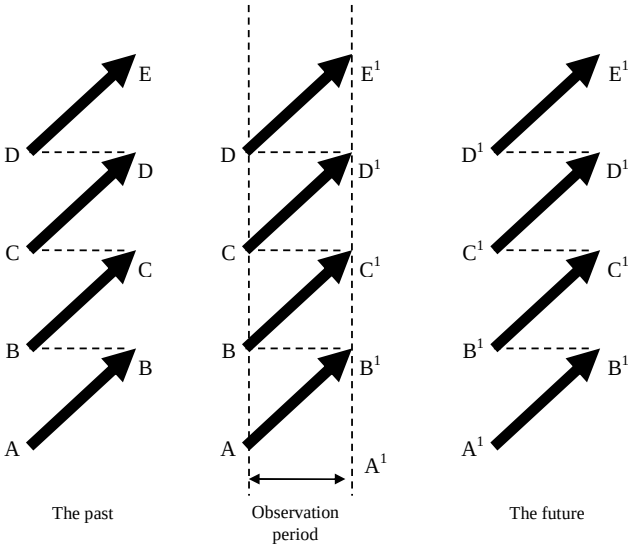
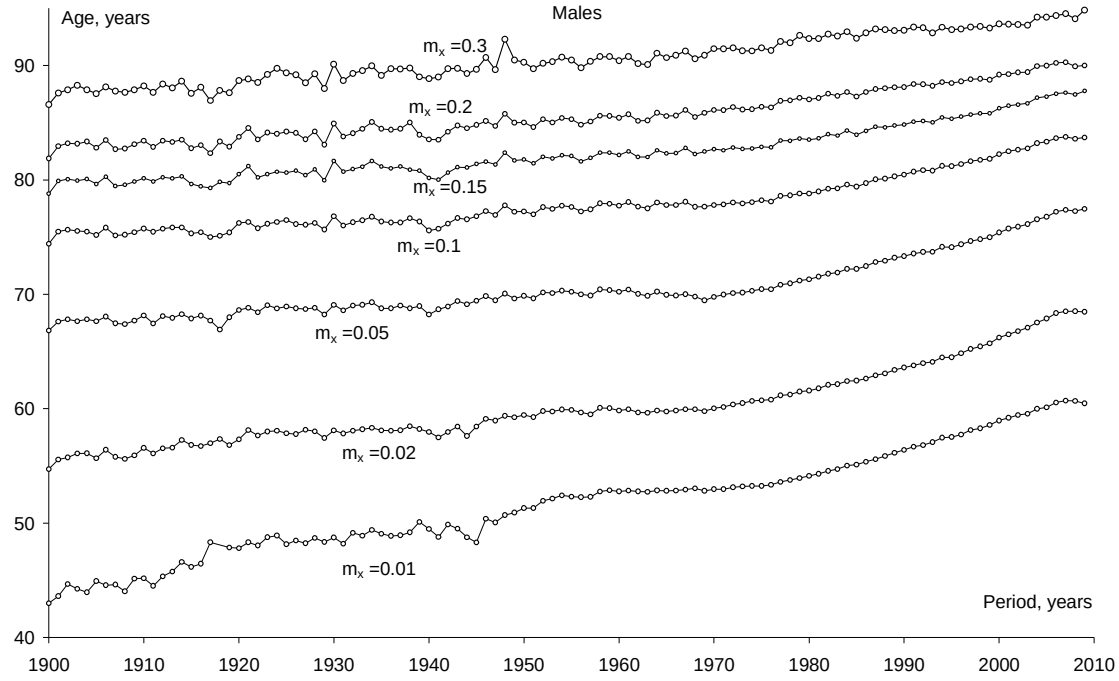


Figure 2. Illustration of the mortality compression implicitly assumed in the conventional ‘constant mortality’ scenario

Note: The arrows depict parts of the lifespan of birth cohorts in the Lexis diagram (age goes along the vertical axis). The capital letters denote different levels of mortality. The panel to the left depicts the stagnant mortality prior to the observation period; the panel in the middle depicts the declining mortality during the observation period; and the third panel depicts the conventional ‘constant mortality’ scenario of the future.



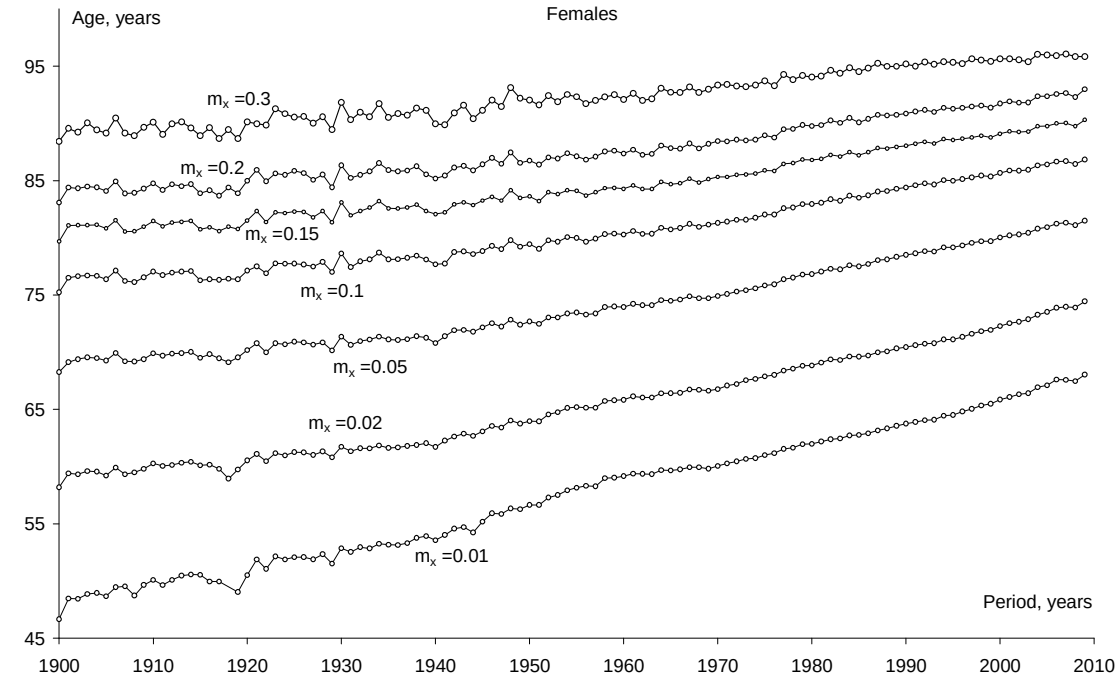
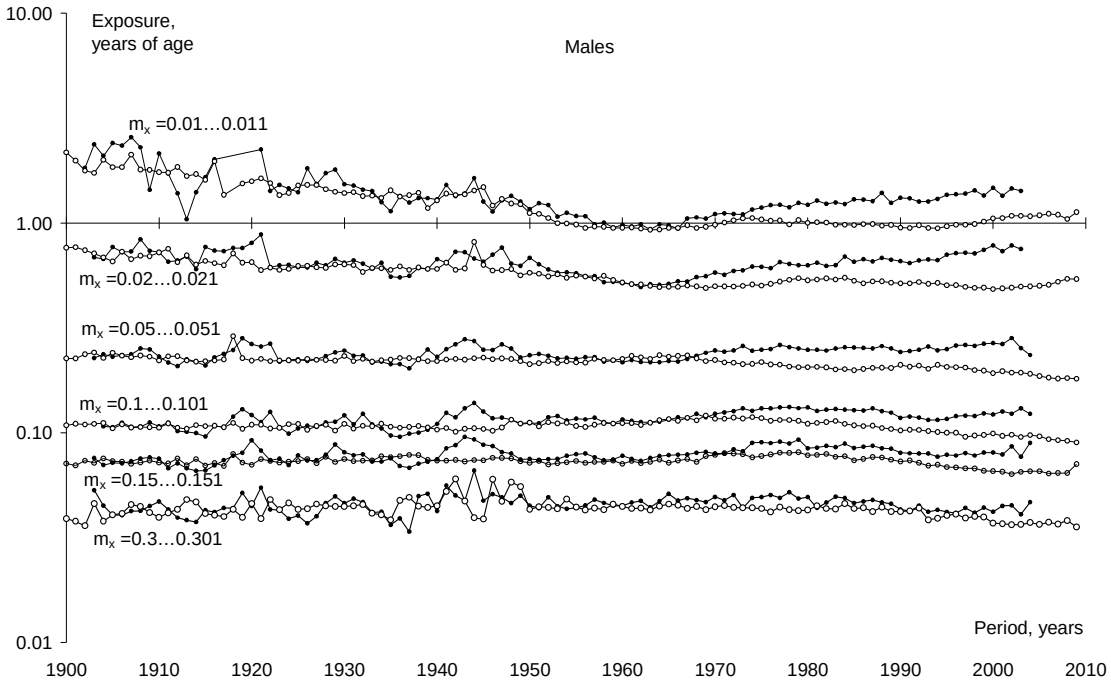


Figure 3. Ages at which the selected death rates were observed in different periods (the ages are averaged over 23 countries)



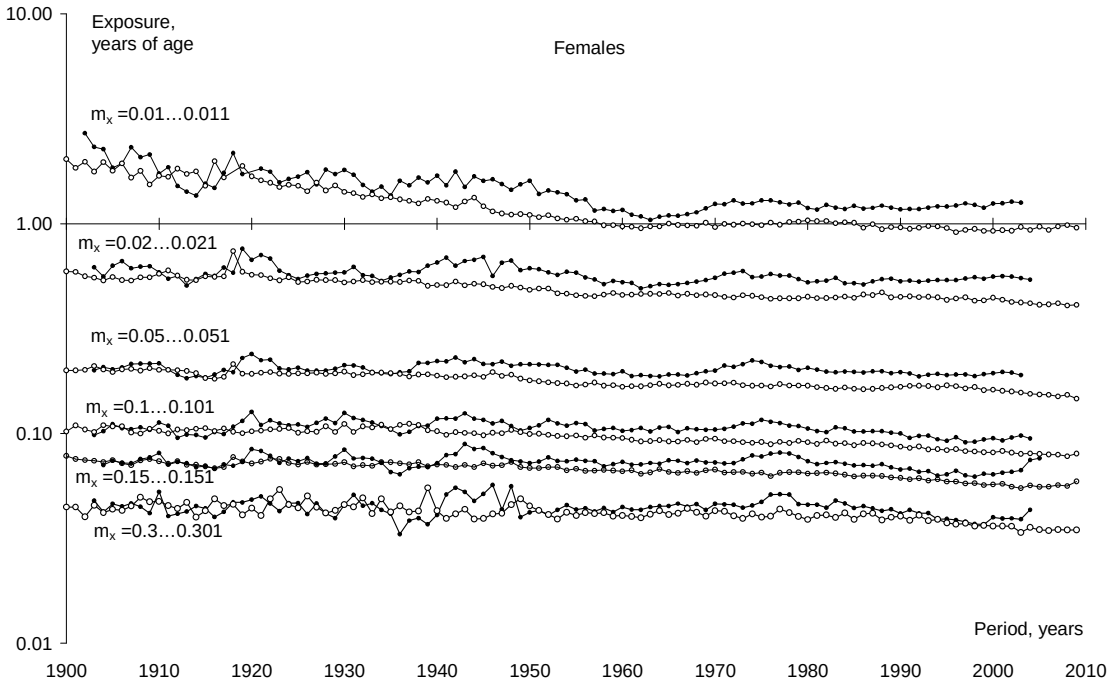


Figure 4. Dynamics over time of durations of exposure, in years of age, of birth cohorts (dots) and period life tables (circles) to selected mortality levels (the exposures are averaged over 23 countries)

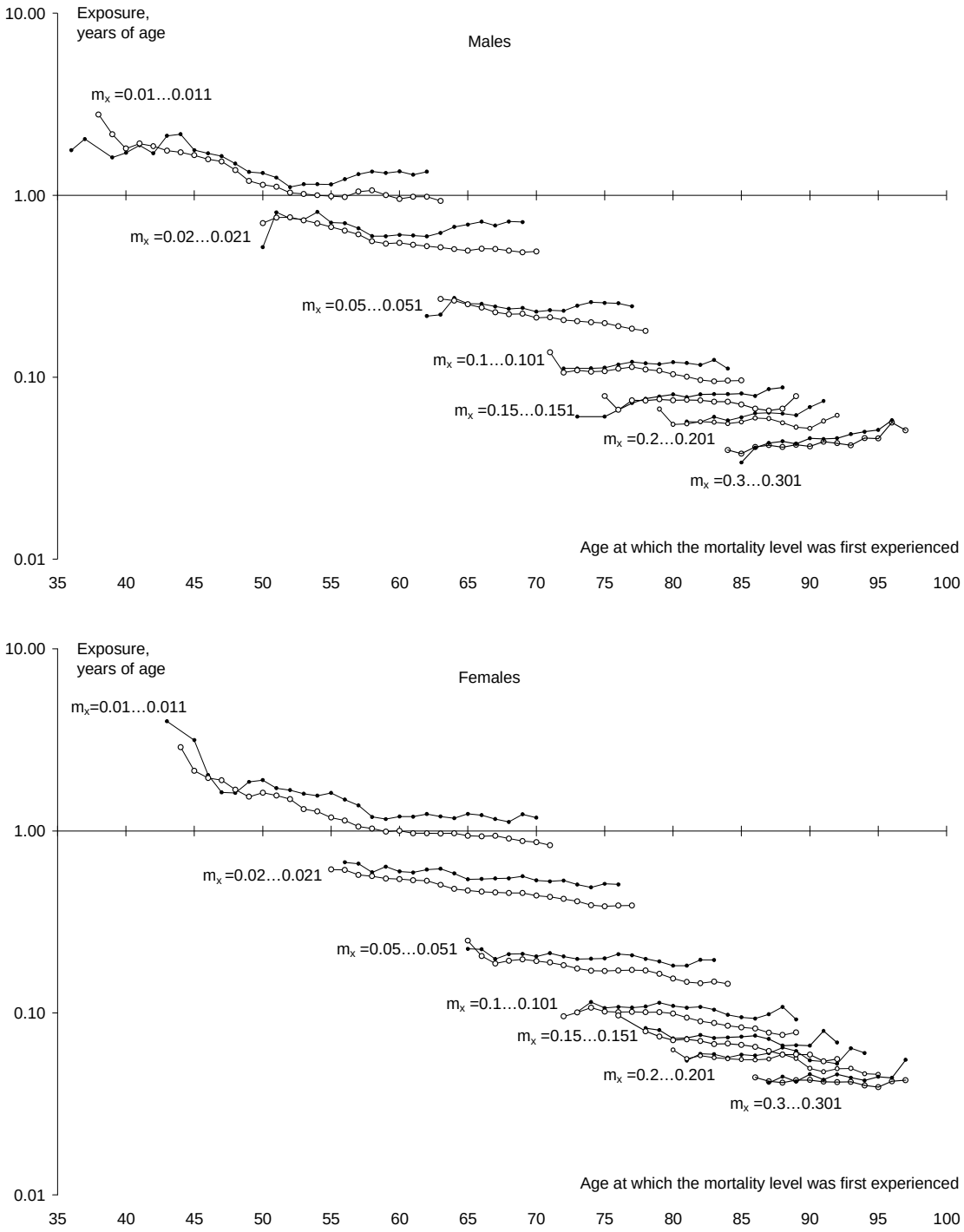


Figure 5. Durations of exposure, in years of age, of birth cohorts (dots) and period life tables (circles) to selected ranges of the death rate as functions of the youngest age at which the death rates were observed (the exposures are averaged over 23 countries)

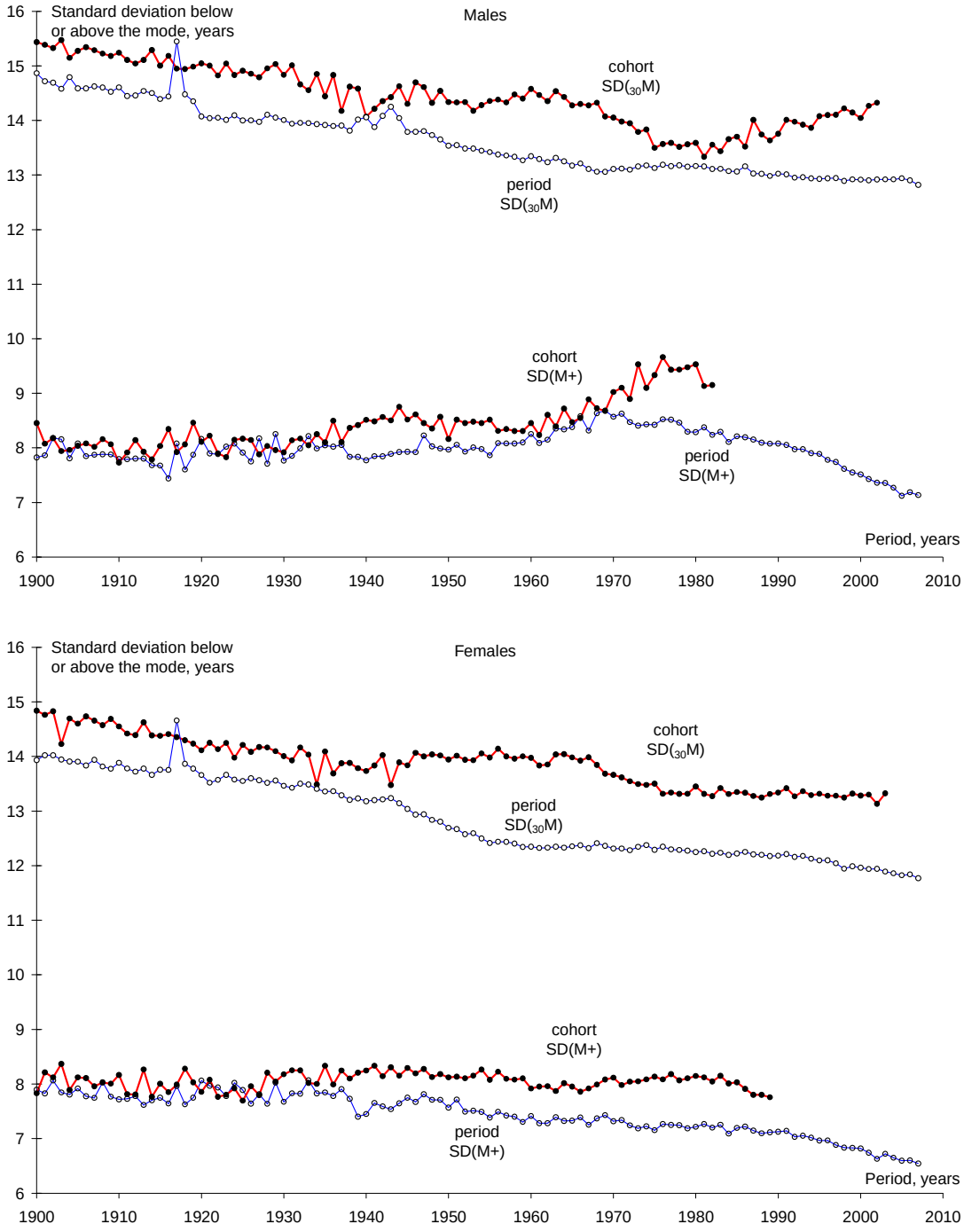


Figure 6. Cohort and period root mean square deviations form the modal age at death of ages at death above the mode (SD(M+)) and within a 30-year-long age interval below the mode ($SD(_{30}M)$), averaged over 23 countries as a function of the period when the mode was observed.

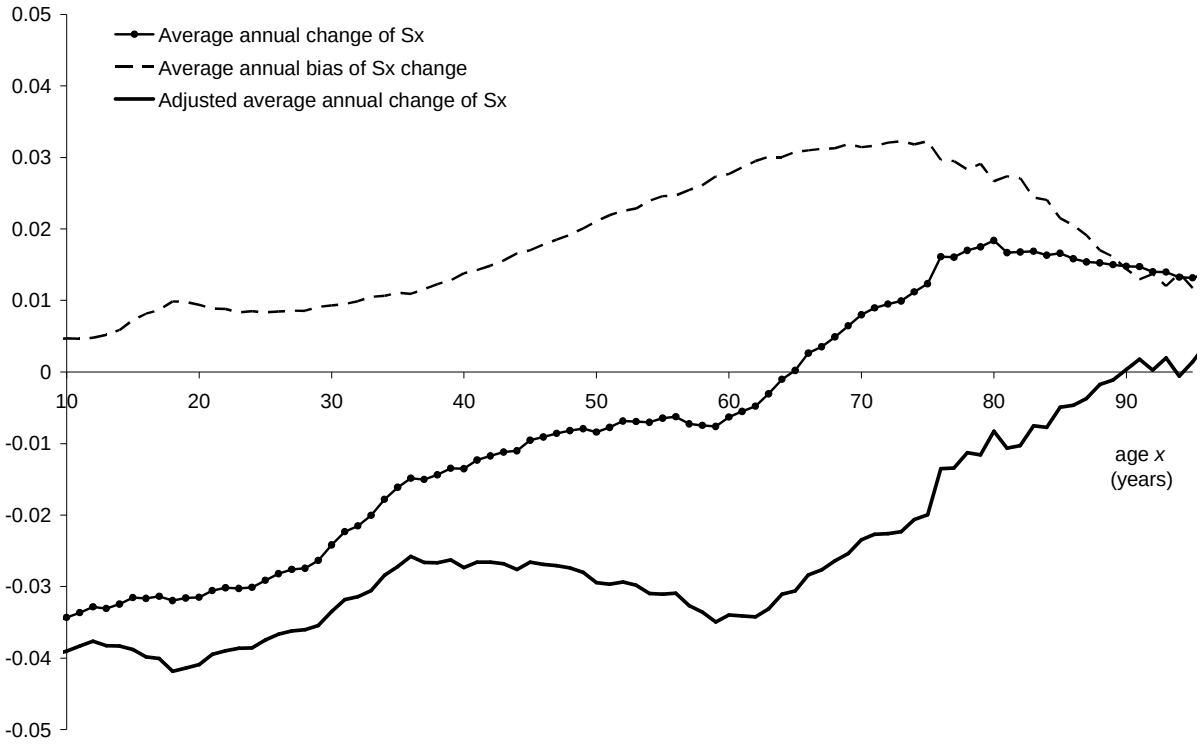


Figure A1. The average annual change in the standard deviation $S(x,t)$ of ages at death above age *x*, the average annual bias of the change of $S(x,t)$ as an indicator of mortality compression, and the average annual change adjusted for the bias in female period life tables in 1995-2000 (for each year, mortality rates are averaged over 23 female populations)