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Mortality compression in period life tables hides decompression in birth cohorts in low-mortality countries

1. INTRODUCTION

Human longevity continues to increase in developed countries. In countries that currently have low mortality, the average lifespan has more than doubled from the pre-modern level of about 35 years (Wilmoth, 2007). This increase is expected to continue: the United Nations (UN Population Division, 2013) has forecast that period life expectancy at birth will increase in low-mortality countries, though at a gradually declining pace, to about 87 years by 2050 and to 92 years by the end of the century.

The rapid rise in human longevity has raised 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 most extreme ages (Wilmoth, 1995).

The prospect of a compression of mortality and morbidity would have had numerous implications for public health policies. It would imply that gains in life expectancy mostly impact the duration of healthy life, while unhealthy years are limited to ever-shrinking terminal period in later life. Mortality compression was also 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 refuted by subsequent research, 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 until now (Vaupel, 2010).

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Understanding the developments in mortality compression is also important for modeling mortality and formulating assumptions about future mortality dynamics (e.g., Yashin *et al*., 2000; Yashin *et al*., 2001; Tuljapurkar and Edwards, 2011; Ediev, 2013a).

Most studies of period mortality provided evidence of a historical trend towards mortality compression, which may have slowed down or even stopped in recent decades in developed countries (Keyfitz and Golini, 1975; Fries, 1980, 1984, 1989; Nagnur, 1986; Nusselder and Mackenbach, 1996; Wilmoth and Horiuchi, 1999; Kannisto, 2000, 2001; Robine, 2001; Cheung *et al*., 2005; Cheung and Robine, 2007; Canudas-Romo, 2008; Cheung *et al*., 2008; Cheung *et al*., 2009; Thatcher *et al*., 2010; Ouellette and Bourbeau, 2011; Tuljapurkar and Edwards, 2011; Shkolnikov *et al*., 2011; Ediev, 2011a, 2013; Brown *et al*., 2012). Some other authors (Myers and Manton, 1984; Manton and Soldo, 1985; Rothenberg *et al*., 1991; Engelman *et al*., 2010) have argued, on the contrary, that period mortality is expanding at old age. However, their evidence was based on studying the variance of the distribution of ages at death left-censored at some fixed old age, a methodology with a built-in tendency towards decompression in the case of mortality decline (Fries, 1984; Kannisto, 2001; Robine, 2001; Ediev, 2013a). When adjusted for this bias, the variance in ages at death also shows a compression of period mortality in old age (Ediev, 2013a). Lynch and Brown (2001) also found patterns of decompression in period data; but their results apply to the vicinity of the mortality curve's inflection point that characterizes only very old age (currently, around age 95).

As convincing as it may be, the evidence on change of period mortality may not be conclusive, however, in addressing some problems related to compression, such as the existence of an upper limit to the lifespan or the change in the speed of biological aging, without being supplemented by an analysis of the change in cohort mortality. Compression is a built-in feature of the period life table model in the context of declining mortality; it may coexist with decompression, or expansion, of cohort mortality (Ediev, 2011a; see also the next section). Many authors (e.g., Manton *et al*., 1986; Wilmoth and Horiuchi, 1999; Cheung *et al*., 2008; Engelman *et al*., 2010) did study the compression of mortality from the cohort perspective, but that line of research has been limited because the traditional compression indicators may only be computed for (rather old) cohorts with complete or almost complete mortality histories and, therefore, may not be used to study recent developments in compression. Apart from concerns due to incompleteness of data for recent birth cohorts, a pure cohort approach may also be called into question because it mixes up mortality developments formed by conditions of calendar periods far apart from each other. Change of cohort mortality patterns in 20th century, for example, would be subject to a mixture of effects of ups and downs in period mortality conditions formed by major wars, epidemics, and acceleration of mortality decline.

The need to shed light on and overcome the mentioned bias of the period life table model, on the one hand, and to develop a cohort-oriented approach

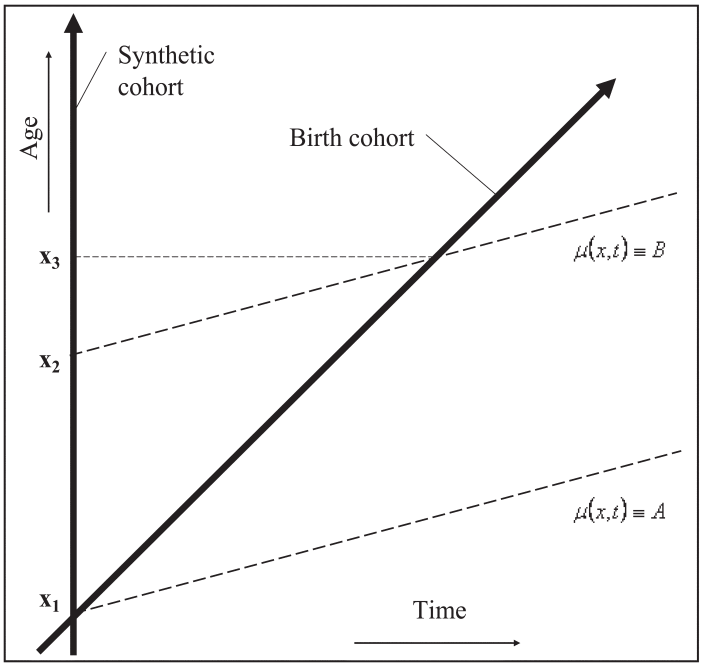
free from the limitations of the convectional cohort analysis, on the other hand, is our main motivation for conducting the current study. Building on an earlier analysis of limitations of the conventional period life table model (Ediev, 2011a), we propose a new approach that operates with durations of exposure, in years of age, of birth cohorts as well as of life table populations to selected ranges of the death rate. In the following section, we briefly reiterate 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 compress, when compared to cohort experiences, the mortality pattern when mortality is on the decline. In Section 3 we introduce the new methodology based on studying the durations of exposure. We present the empirical evidence in Section 4, and then offer some conclusions. The appendices contain the formal relations that support the main findings.

2. WHEN THE PERIOD LIFE TABLE MODEL (ARTIFICIALLY) COMPRESSES THE LIFE SPAN

This section follows Ediev (2011a) in revealing conditions and the source of discrepancies between period and cohort indicators of mortality compression. Indicators of period mortality are usually derived from the period life table model. That 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 $[x_1, x_2]$ in the figure. But the birth cohorts (represented by the 45-degree line) are exposed to the same range of the force of mortality over a longer age interval $[x_1, x_3]$. By its very design, a conventional period life table cuts off the part of the cohorts' experience indicated by age interval $[x_2, x_3]$, which leads to both an overestimation and a compression of mortality in the life table.

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*



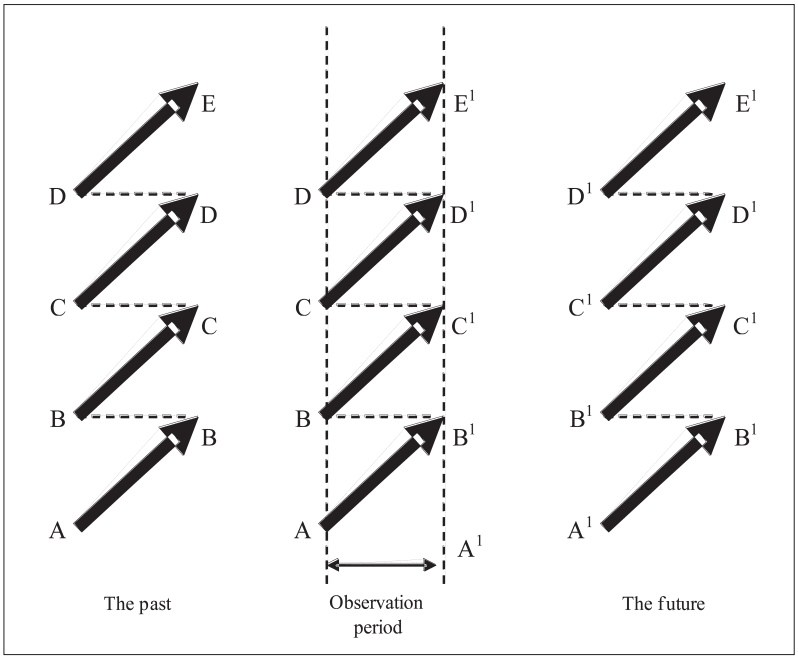
Notes: The two inclined dashed lines are contour lines of the force of mortality and bound the area where the force of mortality increases from level 'A' to level 'B'.

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 B^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 A^1, B^1, etc., at the same respective ages, as at the end of the observation period.

Figure 2 – *Illustration of the mortality compression implicitly assumed in the conventional 'constant mortality' scenario*



Notes: 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.

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 B^1 to level C^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 C^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 B^1 and the age when another birth cohort experiences mortality level C^1 for the duration of *time* over which a (hypothetical) cohort moves from level B^1 to level C^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 life table aging rates and the exposures (in years of age) to similar ranges of the death rate are presented in Appendix A1 (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 of mortality compressed by $1-r$ times as compared to the cohort schedules.

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 for studying contemporary developments in mortality. Besides, cohort mortality, when followed over the

entire or long part of a cohort's lifespan, may mix up mortality conditions of calendar periods extending up to 100 years apart, making it difficult to interpret traditional cohort indicators of mortality compression. We therefore do not focus on the distributional characteristics of ages at death, as is usually done in the literature on mortality compression. Instead of studying distribution of deaths over the entire life of the cohort, we examine processes of compression/decompression in small subsets of the cohort lifespan. To this end, we calculate the durations of exposure (in years of age) of birth cohorts (also as compared to period mortality schedules) to certain narrow ranges of the death rate observed at old age. Using sufficiently narrow ranges of the death rate, it will be possible to study cohort mortality experiences in selected calendar periods and not over the entire lifespan of birth cohorts. Shortening exposure times indicates compression processes going on in all cohorts involved. Cohort exposure times increasing in a given period indicate mortality decompression across the affected cohorts in that period. In this way, we do not need data or forecasts on cohorts' full lifespans; instead, we study the data referring to a given calendar period. We also do not mix together conditions of remote calendar periods.

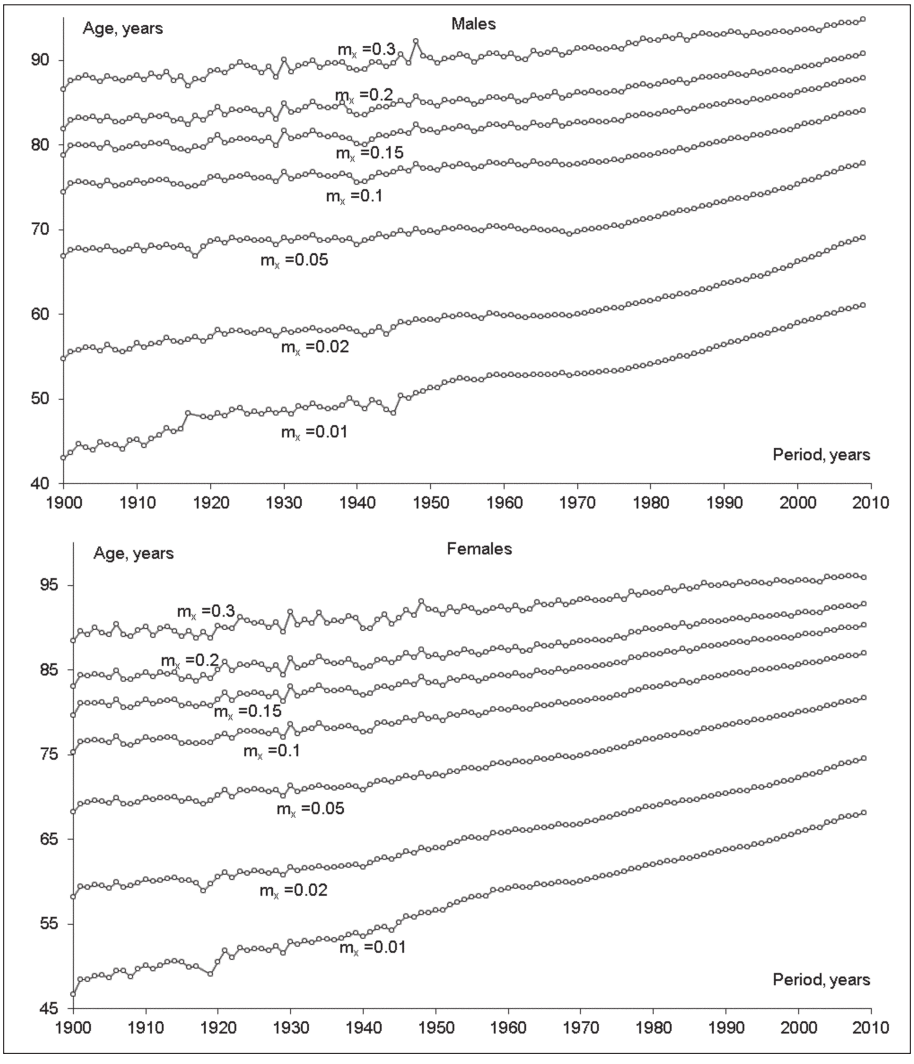
Our method is exploiting an idea similar to that in Lynch and Brown (2001), where the decompression was studied by estimating the slope of the mortality curve. The exposure times to a given narrow range of the death rate and the slope of the mortality curve in the respective age range are, obviously, inversely proportional to each other. Unlike in Lynch and Brown (2001), however, we do not focus only on compression of the curve at one single point. Instead, we study the compression at a number of points of the mortality curve representative of the whole pattern of old-age mortality. For several reasons, we prefer using the durations of exposure instead of the slope of the mortality curve or the slope-related life table aging rate (Horiuchi and Coale, 1990). First, durations of exposure, measured in years of age, are directly relevant to the concept of mortality compression. Second, the durations of exposure to several sub-ranges of the death rate may conveniently be added up to show exposure time to a wider range of the death rate. Thirdly, the durations of exposure considered here fit naturally into a more general family of exposure durations to different life conditions of interest. Morbidity compression, for example, may be studied by examining the duration of time from the onset of a disease and until death.

At this point we should also comment on the link between the compression of the age pattern of the force of mortality, which the paper is 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 mor-

tality 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 to 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 to 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 at death, a general analytical relation has been established between the steepness of the mortality curve and the death rate (Pollard, 1991; Canudas-Romo, 2008; Thatcher *et al*., 2010; Tuljapurkar and Edwards, 2011), 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). Hence, the exposure durations to mortality levels around the modal age are in direct relation to other indicators of mortality compression in relation to the mode. In Appendix A2, 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 Rothenberg's (1991) and 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.

To examine the compression of durations of exposure at various mortality levels currently representative of old-age mortality, 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. For each range, we examine development of exposure durations, in years of age, of birth cohorts and of period life tables. 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 (but they have covered some younger age ranges in the past, see Fig. 3). While corresponding to different age ranges at different periods of time, the range of the death rate 0.01 to 0.301 covered, after 1900, 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). Given a special interest in the literature to the compression in relation to the modal age at death (e.g.: Kannisto, 2001; Cheung and Robine, 2007; Canudas-Romo, 2008; Thatcher *et al*., 2010), it is also worthwhile noting that the age at the death rate 0.1 at that time (after 1900) was close to the modal age at death. So, three of our selected ranges of the death rate cover ages below the modal age; three ranges cover ages above the modal age; and the middle range corresponds to the modal age.

Figure 3 – *Ages at which the selected death rates were observed in different years in 1900-2009 (the ages are averaged over up to 23 countries with available data in each year)*



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 exposure time. 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:

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

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[1]. To reduce volatility due to small population size, lower data quality, and leveling off of the mortality curve at most extreme 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 (2013) (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; and also Iceland and Luxembourg because of their small population size. This leaves 23 countries in HMD available for our study. 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 ranges of the death rate over the long time period since 1900. We do not show detailed results for the years before 1900 due to small number of countries with available data but also because the most important advances in old-age mortality has happened in the 20th century. We will, however, present summary indicators referring to the whole range of the death rate 0.01 to 0.301 for the period after 1850. For different years there are different numbers of countries with mortality data available in HMD, but this does not substantially affect our findings. The earliest and the latest years with death rates available in HMD for each of the countries studied are shown in Table 1; cohort-wise, (partially incomplete) sets of age-specific death rates are available in HMD starting three years later and ending one year earlier than the periods shown in Table 1.

[1] 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, Li *et al*., 2013).

[2] 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 another study.

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 1900[a]	Exposure in 1990	Exposure in 2000	Exposure in T_2
			Females			
Australia	1921	2009	44.9	31.6	28.4	26.6
Austria	1947	2010	32.7	28.2	26.5	25.0
Belgium [b]	1841	2009	38.8	29.1	27.2	26.9
Canada	1921	2009	42.2	32.3	30.4	29.8
Chile	1992	2005	34.5	34.5[c]	32.4	32.9
Denmark	1835	2011	38.1	35.1	34.6	30.0
Finland	1878	2009	39.5	29.2	26.3	25.6
France	1816	2010	42.7	27.2	26.5	25.4
Ireland	1950	2009	37.8	32.2	30.0	29.4
Israel	1983	2009	34.3	34.5	31.5	29.2
Italy	1872	2009	38.0	28.5	27.4	25.7
Japan	1947	2009	38.8	26.7	27.5	26.5
Netherlands	1850	2009	38.8	28.9	28.4	27.0
New Zealand	1948	2008	37.6	33.7	31.2	28.6
Norway	1846	2009	42.3	30.6	27.6	26.8
Portugal	1940	2009	36.5	29.1	27.9	24.9
Spain	1908	2009	48.0	27.2	26.2	24.7
Sweden	1751	2011	37.5	28.4	27.5	26.3
Switzerland	1876	2011	39.4	27.1	25.6	24.6
Taiwan	1970	2010	36.4	33.3	32.3	30.6
The United States	1933	2010	45.2	35.4	33.3	32.0
United Kingdom	1922	2009	38.5	33.9	31.3	29.9
West Germany	1956	2010	30.5	28.8	28.0	25.6
Average for females				30.7	29.0	27.6
			Males			
Australia	1921	2009	46.6	36.3	32.1	31.1
Austria	1947	2010	38.6	36.2	34.6	33.9
Belgium [b]	1841	2009	45.6	35.3	33.6	33.3
Canada	1921	2009	42.1	37.0	34.1	33.5
Chile	1992	2005	39.8	39.8 [c]	37.5	40.0
Denmark	1835	2011	42.2	37.8	35.3	32.6

...Cont'd...

Table 1 – *Cont'd*

Population	First year of data (T_1)	Latest year of data (T_2)	Exposure in 1900[a]	Exposure in 1990	Exposure in 2000	Exposure in T_2
			Males			
Finland	1878	2009	44.7	37.6	34.0	34.7
France	1816	2010	50.1	37.7	35.1	34.5
Ireland	1950	2009	38.2	35.4	33.0	33.1
Israel	1983	2009	39.4	38.8	35.8	35.1
Italy	1872	2009	48.4	36.2	33.3	31.1
Japan	1947	2009	42.3	33.3	33.8	33.1
Netherlands	1850	2009	42.0	35.3	32.7	31.0
New Zealand	1948	2008	40.8	37.1	33.8	33.2
Norway	1846	2009	44.5	36.5	31.5	30.2
Portugal	1940	2009	42.1	35.9	34.2	32.4
Spain	1908	2009	46.6	35.3	34.4	33.6
Sweden	1751	2011	43.6	33.7	31.1	29.4
Switzerland	1876	2011	48.3	33.7	32.0	29.7
Taiwan	1970	2010	41.0	37.6	37.9	39.3
The United States	1933	2010	48.4	39.7	36.4	36.3
United Kingdom	1922	2009	40.0	36.4	34.3	33.8
West Germany	1956	2010	36.6	35.5	34.3	33.4
Average for males				36.4	34.1	33.4

Notes: [a] In T_1, whenever T_1 >1900; [b] With a gap in 1914-1918; [c] Replaced by value for year 1992.

4. EMPIRICAL EVIDENCE

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. 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 for all of the selected 23 low-mortality countries.

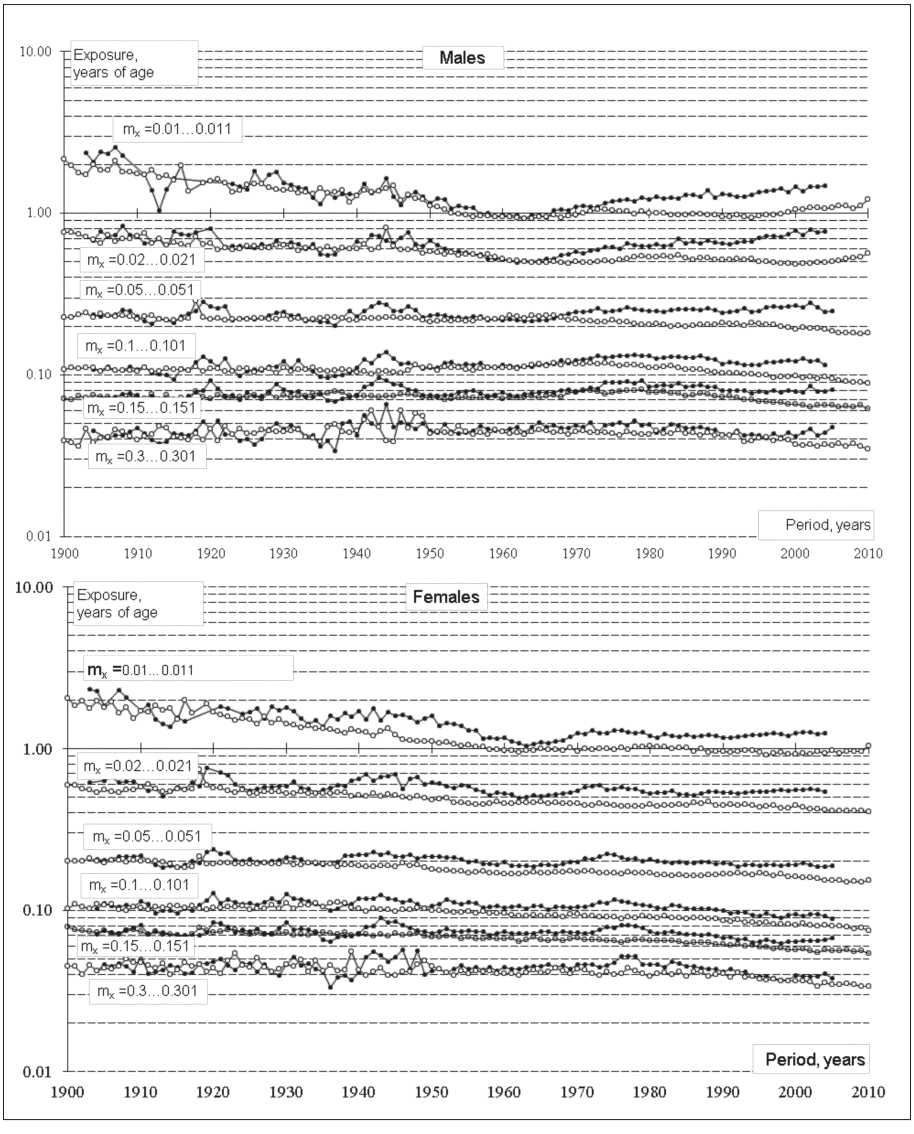
In Figure 3, we present contour lines of the death rate corresponding to

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 a gap between the mortality compression picture revealed by the period life tables and the one characterizing cohort experiences in the same period. Since 1990, the ages at which the selected mortality levels are observed have shifted by about 0.17 years per year (averaging estimates for males and females and for all seven mortality levels chosen). The formal theory (Appendix A1; Ediev, 2008) indicates that the period life table will compress the actual (cohort) exposure durations to the selected ranges of the death rate by about 1/(1-0.17) = 1.20, or 20%. This would be an average contemporary bias of the period life table estimates for the mortality compression indicators. Since the mortality decline has accelerated in 20th century, the bias of the period life tables towards showing a compressed mortality picture should have become stronger. This may have created a dynamic effect: while period life tables have shown ever more compressed mortality in the 20th century, cohorts' experiences may have been subject to less compression or may have not been subject to mortality compression at all. Below, we provide a more explicit and comprehensive picture of these differential dynamics by considering cohort- and period-specific durations of exposure to the chosen ranges of the death rate.

The development over time of durations of exposure to the selected ranges of the death rate is presented in Figure 4 and Table 2. In line with the theory (Section 2; Ediev, 2008, 2011a), cohort and period durations of exposure were similar in the periods of stagnation of mortality (most noticeable is the case of male mortality at ages 50-75, i.e., death rates of 0.01 to 0.1, in the 1960s). Periods of declining mortality (increasing contour lines in Figure 3) are marked, in Figure 4, by cohort exposure durations exceeding the period exposure durations. In 1996-2005, the estimates of the exposures, in years of age, to the selected ranges of the death rate were about 18% shorter when obtained from period life tables than the estimates based on cohort data (Table 2, averaging estimates for males and females and for all seven mortality levels chosen). This discrepancy is well in line with the rough estimate above (20%) derived from formal relations. After the 1960s, the discrepancy between the period and cohort compression indicators increased at all (except for the highest) selected levels of the death rate, which is in line with the accelerated mortality decline over that period. Males are exposed over longer durations of time to the selected ranges of the death rate. They are still expanding at least at the two lower mortality

levels. However, they experience those levels at younger ages than females, which is consistent with lower male life expectancy.

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 (values are averaged over countries and hidden when less than five country-cases are available)*



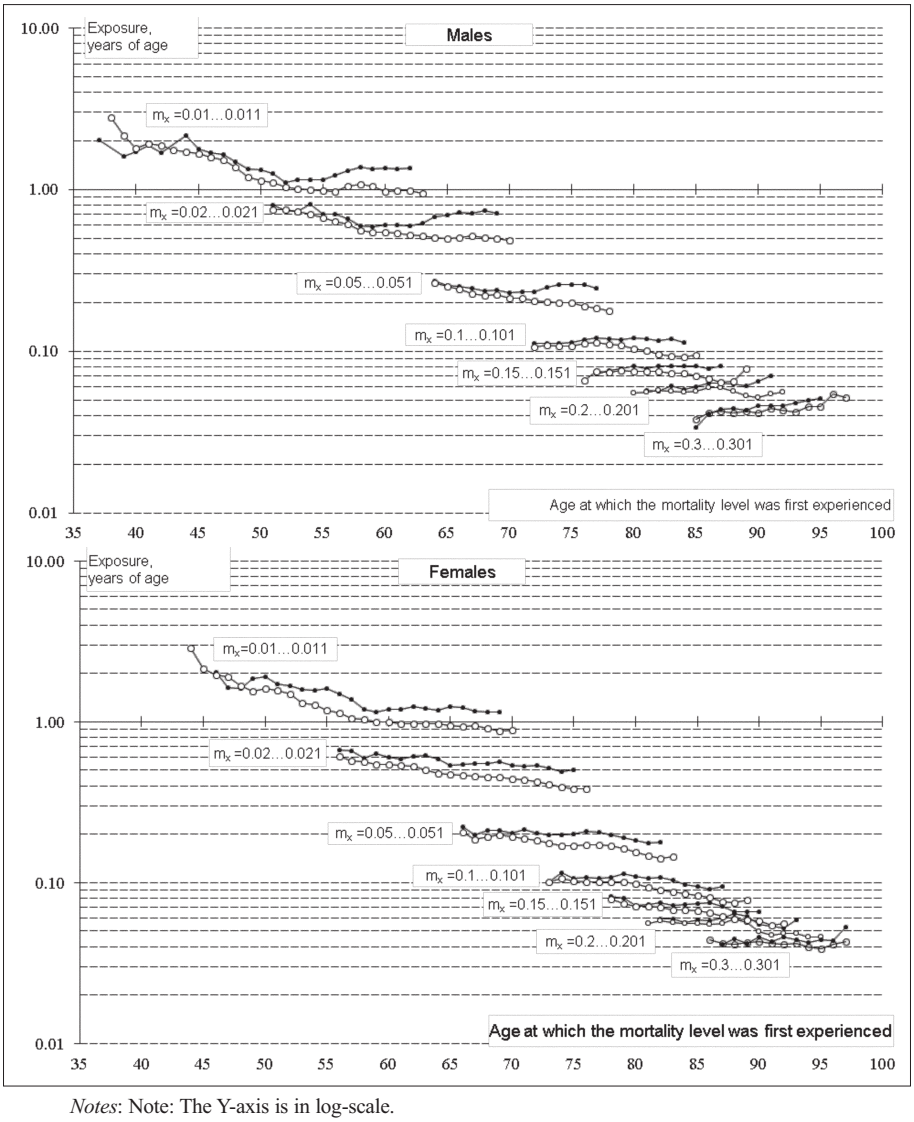
Notes: Note: The Y-axis is in log-scale.

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*

	Birth cohorts' actual exposure to the selected ranges of the death rate in periods:			Exposure estimates from period life tables		
Range of the death rate	1900-1909	1960-1969	1996-2005	1900-1909	1960-1969	1996-2005
			Males			
0.01-0.011	2.25	0.99	1.42	1.90	0.95	1.04
0.02-0.021	0.74	0.52	0.75	0.71	0.50	0.49
0.05-0.051	0.24	0.22	0.26	0.23	0.23	0.20
0.10-0.101	0.11	0.12	0.12	0.11	0.11	0.10
0.15-0.151	0.073	0.077	0.080	0.072	0.073	0.066
0.20-0.201	0.056	0.060	0.061	0.055	0.057	0.051
0.30-0.301	0.043	0.047	0.043	0.041	0.044	0.038
			Females			
0.01-0.011	2.18	1.10	1.24	1.83	0.98	0.94
0.02-0.021	0.62	0.51	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	0.064	0.074	0.066	0.057
0.20-0.201	0.059	0.061	0.051	0.059	0.056	0.046
0.30-0.301	0.045	0.045	0.039	0.045	0.041	0.036

Mortality change was not uniform across time, age and gender; nor was the gap between cohort and period exposures. Before the 1960s, exposure durations were getting compressed or remained stable in both period life tables and birth cohorts; with short-term fluctuations and few exceptions (the male exposures slightly grew in 1900-1960 at death rates above 0.05). Exposure times of period life tables remained stable or compressed after the 1970s, except at the two lowest selected mortality levels for males. During the 1960s and early 1970s, all cohort exposures went up and the gap has widened between cohort and period exposures (the latter remained stable or continued to be compressed for females, following more complex patterns for males). In and after the late 1970s, male cohort mortality continued to be decompressed at younger ages (death rates below 0.05) and was more stable (in terms of exposure durations, which implies that the mortality curve was shifting along the age axis) at older ages. That was accompanied by a further widening of the cohort-period gap in male exposure times, except at the highest mortality level. In the same period, female cohort mortality showed no clear trend in exposure durations at younger age (death rates below 0.05) and got compressed at older age.

Figure 5 – *Durations of exposure 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 (values are averaged over countries and hidden when less than five country-cases are available)*



Notes: Note: The Y-axis is in log-scale.

The above presentation was focused on *period* effects (Hobcraft *et al*., 1982) upon exposure durations. Visible similarity of change over time of exposure durations at different mortality levels, for both genders, for cohorts and

period life tables may indeed indicate the dominant role of period conditions in driving mortality compression or decompression. Most previous literature, however, has been conceptualizing the compression as a phenomenon that is driven by *age* effects (of which approaching an upper limit to the lifespan and biological aging are the most obvious ones). Following this tradition and also taking into account the sheer magnitude of age shifts in mortality (Figure 3), we 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; at each age, the exposures are estimated for each country irrespective of the period, after 1900, when the death rate was observed at agiven age and then averaged across all countries).

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
Males, cohorts											
0.01-0.011	1.84	1.54	1.17	1.25	1.35						
0.02-0.021		0.62 [a]	0.75	0.62	0.61	0.71	0.74 [a]				
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	0.080	
0.20-0.201							0.050 [a]	0.051 [a]	0.058	0.062	0.066
0.30-0.301										0.043	0.047
Males, period life tables											
0.01-0.011	1.82	1.42	1.05	1.02	0.98						
0.02-0.021		2.12 a	0.73	0.58	0.53	0.51	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	0.068	
0.20-0.201								0.067 a	0.056	0.057	0.055
0.30-0.301									0.040 a	0.042	0.043

Notes: [a] Values might be affected by a small number of cases (five or fewer country cases).

Period life tables show a pronounced compression or stability of the exposure durations as functions of age at most mortality levels for both genders (with the exception of decompressing male period mortality at the highest selected mortality level and a more complex pattern at the levels 0.1, 0.15, and 0.2 of the death rate). The cohort exposure durations used to be longer when the two highest selected ranges of the death rate were observed at older ages for males. For the middle range (0.1 to 0.101), that roughly corresponds to the modal age at death and for the fourth range (0.15-0.151), a moderate decompression at younger ages was followed by a pattern of stability at higher ages, while exposures at lower mortality levels show compression at younger ages followed by decompression and then stability at older ages. Female cohort

mortality was characterized overall by compression and stability although the pattern is very much specific to age- and mortality-level. An exceptional case was the highest of the selected mortality ranges, where female cohort exposures showed a moderate age trend towards decompression.

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
	Females, cohorts										
0.01-0.011	3.62 a	1.87	1.67	1.28	1.20	1.21	1.18 a				
0.02-0.021				0.63	0.60	0.55	0.53	0.50			
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
0.30-0.301										0.043	0.044
	Females, period life tables										
0.01-0.011	2.83	1.75	1.45	1.06	0.98	0.93	0.87				
0.02-0.021			0.53 a	0.56	0.52	0.46	0.43	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	0.092	0.082	
0.15-0.151								0.076	0.069	0.062	0.058
0.20-0.201									0.057	0.056	0.049
0.30-0.301										0.042	0.042

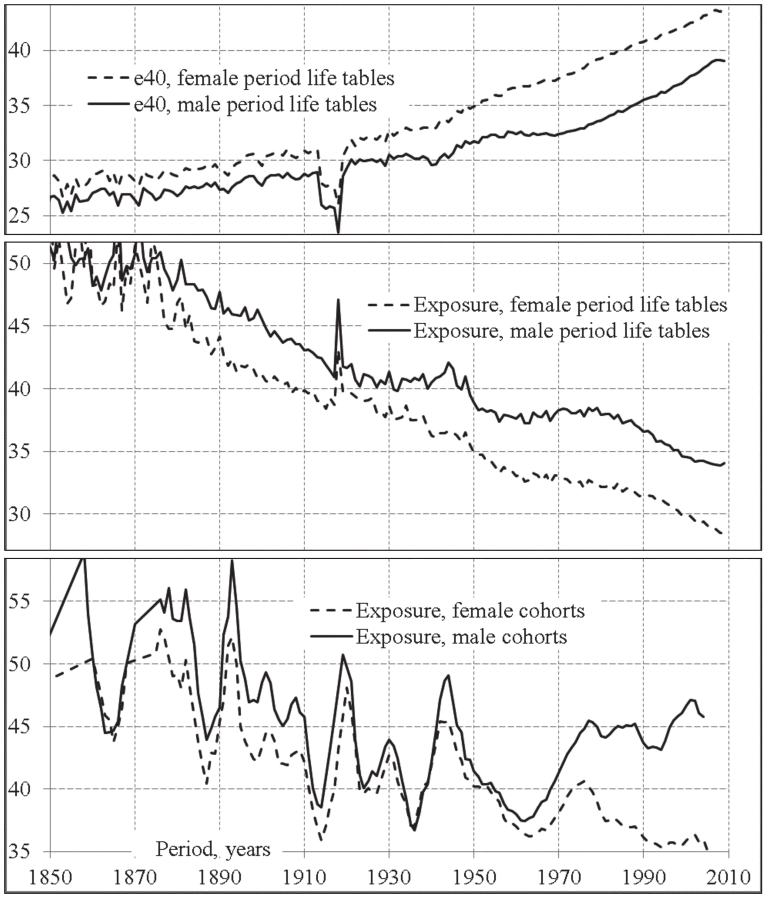
Notes: [a] Values might be affected by a small number of cases (five or fewer country cases).

In many aspects, age patterns in Figure 5 may be interpreted as an indirect reflection of the period effects shown in Figure 4. This is evidenced by the fact that the age patterns of the exposure durations at different mortality levels become more similar after shifting the curves in order to adjust for lags between the ages at which various mortality levels were observed (Figure 3). Another indication of the leading role of period effects may be generally smoother patterns (with shallower or even absent troughs corresponding to the minimal exposure times in the 1960s) in Figure 5 as compared to Figure 4; presumably, this is due to averaging the exposure times corresponding to different calendar periods. At the same time, some elements in Figure 5 may not be attributed to pure period effects. At a number of the selected mortality levels (e.g., the two lower levels for males, both cohort- and period-wise; the three highest mortality levels for male period mortality; the two lowest and the highest mortality levels for female cohort mortality; and the highest and the lowest levels for female period mortality), the most recent trends seen in Figures 4 and 5 are not of the same direction. These distinct age and time patterns might be explained by differential age or country effects that deserve further research.

All that said, Figures 4 and 5 suggest that period effects are the main driver of the cohort exposure durations. This justifies a simpler account of the gen-

eral evolution of mortality compression/decompression, using a single summary indicator. To this end, we calculate the exposure duration for a whole range of death rates covering all mortality levels studied (in our case, spanning death rates between 0.01 and 0.301), see Figure 6. To facilitate discussing the gender differentials, the graphs for males and females are put together. The figure covers a longer period, dating back until 1850, although peculiarities of the earlier periods should be interpreted with caution due to fewer countries with data available in HMD (for 1850, HMD contains period and cohort data only for Belgium, Denmark, France, Norway, and Sweden; see Table 1 for more details).

Figure 6 – *Period life expectancy at age 40 (the upper panel) and period (the middle panel) and cohort (the lower panel) durations of exposure to the range 0.01-0.301 of the death rate*



Notes: values are averaged over countries; cohort exposures are cross-sectional totals of exposures of birth cohorts in a given period to small sub-ranges of the death rate, see the text for details.

In the first panel of Figure 6, we present period life expectancies at age 40 averaged over the countries studied. The middle panel shows the total of exposure durations, in years of age, to ranges of the death rate 0.01-0.011, 0.011-0.012, …, 0.299-0.3, and 0.3-0.301 in period life tables. The third panel of the figure shows cohort totals of exposures to the same ranges. As noted above, we do not use indicators based on the complete birth cohorts both due to data limitations but also because such indicators mix up conditions of remote periods of time and may be problematic in interpretation. We therefore also avoid using cohort exposures to the wide range 0.01-0.301 of the death rate. Instead we compute yet another cross-sectional index by summing up exposures of birth cohorts observed in a given calendar year to small subranges of the death rate 0.01-0.011, 0.011-0.012, …, 0.299-0.3, and 0.3-0.301.

Both period life expectancy at age 40 and period exposure duration to death rates 0.01-0.301 show clear long-term trends, even if interrupted by temporary reversals or stalling episodes. Over the past century and a half, life expectancy at age 40 improved at an increasing rate, with an ever-widening gender gap that has only recently started to narrow. In the meantime, period exposure durations declined at a more linear pace, although with substantial deviations from the trend. The gender gap in period exposure times has also expanded, especially before 1980, but narrowed somewhat after that year. The gap in e_{40} reached its historical maximum of 5.6 years in 1979; in the same year, the gap in period summary exposure to death rates 0.01-0.301 reached the maximum of 6.0 years; it declined until 1993 when it started to increase again. All in all, the change in period life tables was a combination of shifts (indicated by increasing life expectancy in Figure 6 as well as shifting ages at selected mortality levels in Figure 3) and compressions (indicated by falling exposure durations).

The pattern of change of the summary index of cohort exposure times is different in principle. First, it does not show a clear time trend. Indeed, the indicator seems to have contracted by the early 20th century. While such a compression seems to be a logical outcome of reducing the death tolls from less age-discriminate external causes of death, it may be not significant and consistent with the following trend due to the small number of country cases available. Apart from the possible compression in the late 19th-early 20th century, cohort exposures show large volatility but not a strong time trend. The cohort trend, if there is one at all, is toward moderate decompression for males and moderate compression for females. After the 1960s, in the period of fatsest increases in life expectancy, male cohort mortality has substantially decompressed. A similar development of female mortality has reversed in the mid-1970s. In the very recent periods (after 1980 for males and 1990 for females), cohort exposures become more stable. Another notable difference is the gender gap that, for the cohort exposures, has continued expanding, with oscillations, since 1952. The gap continued to widen even after the 1980s, when gender gaps in the two period life table indicators have been narrowing.

5. DISCUSSION

Our empirical results are in good agreement with the theoretical prediction that the period life tables by their design provide a compressed picture of the underlying cohort experiences when mortality is on the decline. Given that the speed of mortality decline varies with time, the period life tables may be subject to varying levels of compression. Unfortunately, however, the conventional reliance on period data cannot be changed easily, as the traditional compression indicators cannot be computed for many birth cohorts with incomplete data. Even assuming that the problem of incomplete cohort data is resolved, traditional cohort compression indicators may be misleading because they mix up diverse conditions of remote calendar periods to which birth cohorts may have been exposed during their long lives. Our empirical results showing strong period effects upon cohort exposure times indicate that the mentioned problem of the conventional cohort approach may be a serious obstacle in studying mortality change. Our method, which is based on examining the exposure durations in years of age to selected short ranges of the death rates, is free from these limitations and allows us to study both period and cohort mortality compression across time and age.

Differential patterns of exposure times of cohorts and period life tables call for discussion and interpretation. Before continuing with the substantive discussion, however, it is necessary to remind ourselves of possible caveats to our results. First, despite pooling together mortality data for many developed countries from the best available international database, we may not have been able to secure equally reliable and comparable results for the long period of time covered by the study. The number of countries with available data was lower for earlier years than for more recent years (see Table 1). In a nutshell, our sample of countries with available cohort and period data was close to complete only from 1960 onwards (20 or more countries with period life tables, 17-20 countries with cohort data available and stable enough to meet our method's requirements at all the mortality levels selected for the study). It increased between 1920 and1960, and was small (about 9 countries) in 1890-1920, and even smaller in the earlier period (5 countries in 1850). Data quality may also be a problem for earlier periods due to missing data, different territorial coverage, absence of migration data and, hence, reconstructions, extrapolations and other indirect techniques of HMD (see the database's original website). Our estimates of cohort exposure times were based on regressions over at least nine years covering the period of interest; therefore, epidemics and major wars, and also fast epidemiological changes, may have contributed to a higher volatility of our estimates despite precautions made to avoid cases with poor fit of the regression line. Although the high correlation between cohort exposure times for males and females and for different mortality levels indicates that the effect of lower data quality may have been limited, our results for earlier periods should be interpreted with caution and may be revised using better data or an improved methodology.

Period life tables show compression or stability as a near-universal feature across time, age and gender. Although one may notice some phases of moderate decompression at selected levels of the death rate, including the most recent period for males at lower mortality, the summary exposure time of period life tables to death rates 0.01-0.301 presents no example of decompression, beyond short-term fluctuations. A short-lived decompression in the 1940s may have been caused by a very fast decline of death rates which was either due to the introduction of modern antibiotics or a selection effect of those who survived the harsh wartime conditions (it might, however, also be due to limitations of original death records or HMD interpolation techniques for the wartime period). Hence, our results for period life tables support the literature that suggests that period mortality is compressing or stable, not expanding, both historically and more recently. This aspect of mortality change is important to take into account when modeling and projecting period mortality (we will return to the implications for projections further down, after discussing our findings for the birth cohorts).

The pattern of change of exposure times of *birth cohorts* to selected ranges of the death rate was different from that of period life tables. Cohort exposure times show no clear time trend consistent across age and gender, except maybe for the period of compression in the late 19th-early 20th century. Cohort exposures were, however, subject to large-scale swings, a better understanding of which may be important in order to appreciate the long-term time trends possibly masked by the swings. These swings seem to be linked to periods of sudden change in mortality conditions (the two world wars, pandemics, and the spread of modern antibiotics (1940s) and cardiovascular disease prevention programs (1970s) in developed countries). The link may be explained by compensatory changes following the original shock in mortality conditions. Malnutrition, deteriorating sanitation and lack of access to medical and other care during wartime might have, for example, accelerated aging processes. This may happen both at the individual level but also at the population level if the healthiest are selectively enlisted in the military and other services. A conceivable outcome would be a steepening of the cohort mortality curves, i.e., compression. Once the hardship time and its 'tearing' effects are over, however, the cohort exposure durations may return to normal or even temporarily extend beyond the normal levels if a disproportionately high death toll of the least healthy during the period of high mortality modifies the composition of birth cohorts. Fast introduction of new screening or treatment technologies that reduce the fatality of major diseases may lead to a decompression of cohort mortality schedules. A (partial) compensation may, however, follow if reduced fatality contributes to increasing proportions of people with chronic conditions.

Period life tables were successfully hiding substantial, and seemingly informative, transient changes in cohort experiences. This is well explained by our theory, because the extension/compression of cohort exposure times will typically follow improving/deteriorating mortality when the theory shows the period life tables will tend to compress/decompress the underlying cohort experiences.

An explanation of cohort compression/decompression swings by abrupt changes in period conditions, if confirmed in future research, may suggest that the 'normal' cohort mortality should have been more stable in the 20th century, perhaps with a tendency towards compression for females and decompression for males. Male cohort mortality has been on a decompression trend since 1960, that is, in the period during which the greatest declines in old-age mortality have been achieved in developed countries. This contradicts Fries' original idea that declining old-age mortality should be accompanied by its compression.

The result of the outlined changes in cohort mortality was a historically unprecedented widening of the gender gap in cohort compression indicators after the 1950s, male cohort mortality becoming increasingly less compressed as compared to female mortality. Although this process started at the same time as the most recent period of the widening gap between male and female period life expectancy, it did not stop after the late 1970s when the gender gaps in period life expectancy and period life table exposure times started to narrow. These diverse patterns call for a revisiting of the theories explaining the evolution of the gender gap in mortality. Although those theories (e.g., Rogers *et al*., 2010) were built on the premise of a recently narrowing gap, our cohort indicators show that, at least in some aspects, mortality has been evolving with an increasing gender gap ever since the early 1950s. Future research may show whether the increasing gender gap can be explained by differential smoking behavior (Waldron, 1995; Lopez, 1995; Valkonen and van Poppel, 1997; Pampel, 2002; Preston and Wang, 2006), socio-economic differences (Vallin, 1995; Brown *et al*., 2012; Ross *et al*., 2012), faster senescence and a higher heriditary (difficult to eradicate) component of variation in mortality at older ages among females (Graves *et al*., 2006), gender differences in the accumulation of chronic conditions, or something else.

Although the durations of exposure demonstrate regular patterns when arranged as functions of age (more of compression or stability for period life tables and for female cohorts; more of decompression or stability for male cohorts; with the exceptions noted in the previous section), those patterns seem to be driven more by period than age effects, although a detailed age-period-country study may be needed to clarify the role of age effects. So far, we find no evidence of such effects working in the direction consistent with Fries' hypothesis of compression in male cohorts. For females, the compressing effect of age, if any, is considerably weaker in cohorts than in period life tables.

Our empirical findings are relevant to mortality projections in several ways. A consistent long-run trend of compression in period mortality is not supportive to shifting models of period mortality (e.g.: Kannisto, 1996; Wilmoth and Horiuchi, 1999; Bongaarts, 2005; Canudas-Romo, 2008). A projection method more consistent with our results should be capable of combining both the shift and compression in period mortality (see Ediev, 2013a for an explicit assessment of the contribution of these two processes to change in period mortality). On the other hand, our findings for cohort schedules do not support common

extrapolations of age-specific death rates (e.g.: Pollard, 1987; Lee and Carter, 1992; Benjamin and Soliman, 1993) that tend to project mortality compression in both period and cohort life tables. The lack of a clear trend in cohort exposure durations suggests that fixing these exposure durations at their most recent level or assuming their convergence to an imputed level may be a good basis for a projection (a scenario of *mortality inertia* suggested in Ediev, 2011a and used, in comparison to common extrapolation, in Ediev, 2013b). Distinctive features of that scenario – features not shared by common extrapolations – are: continuation for at least several decades of a linear increase in life expectancy, transmission of accelerations in mortality improvement from younger to older ages, and consequently increasing rates of mortality decline at extremely old ages. While the first two features are well documented based on past observations (White, 2002 and Oeppen and Vaupel, 2002 for the linear trend; Wilmoth, 1997, Willets *et al*., 2004, Andreev and Vaupel, 2005 for 'aging of mortality improvement'), the accelerated mortality decline at extreme old age may not have been observed so far (but see some early signs of it in Rau *et al*., 2008) only because the cohorts that will be involved in such a decline are still young.

Our study may be extended in many directions. Time and age patterns presented here suggest the importance of further, more detailed, research of age, period and country effects in mortality compression. Our approach is well-suited for such a study. Another prominent line of research would be to apply our method to morbidity compression and to mortality compression by causes of death, as well as to more uniform subpopulations defined according the socioeconomic status, behavioral patterns, education, etc. Important insights may be gained by studying the causal mechanisms behind the changes in indicators of cohort mortality compression.

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Appendix A1. Relation between period and cohort life table 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 and relations between full and partial derivatives):

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

$$=\frac{\partial}{\partial x}\ln\mu(x,t)+\frac{\partial}{\partial t}\ln\mu(x,t)=k(x,t)\left[1+\frac{\frac{\partial}{\partial t}\ln\mu(x,t)}{\frac{\partial}{\partial x}\ln\mu(x,t)}\right]=k(x,t)\left[1+\frac{\frac{\partial}{\partial t}\mu(x,t)}{\frac{\partial}{\partial x}\mu(x,t)}\right] \quad [A1.1]$$

where $k(x,t)=\frac{\partial}{\partial x}\ln\mu(x,t)$ is the aging rate of the period life table at time t. When mortality is increasing by age ($\frac{\partial}{\partial x}\mu(x,t)>0$) and declining by time ($\frac{\partial}{\partial t}\mu(x,t)<0$), the expression in parenthesis in [A1.1] is less than one; i.e., the cohort life table 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:

$$\mu(y(t),t)\equiv const\,. \quad [A1.2]$$

Differentiating by t,

$$\frac{d}{dt}y(t)\frac{\partial}{\partial x}\mu(y(t),t)+\frac{\partial}{\partial t}\mu(y(t),t)=0\,. \quad [A1.3]$$

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

$$r=-\frac{\frac{\partial}{\partial t}\mu(y(t),t)}{\frac{\partial}{\partial x}\mu(y(t),t)}\,. \quad [A1.4]$$

Substituting this into [A1.1],

$$k_C(x,t-x)=k(x,t)[1-r(x,t)], \quad [A1.5]$$

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

Relation [A1.5] 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 life table aging rate. Hence:

$$\varepsilon_C\left(\mu(x,t),t-x\right)=\frac{\varepsilon\left(\mu(x,t),t\right)}{1-r(x,t)}, \qquad \text{[A1.6]}$$

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 A2. 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:

$$\varepsilon(u)=\alpha\varepsilon_0(u). \qquad \text{[A2.1]}$$

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

$$X_0(u)=\int_{u_0}^{u}\varepsilon_0(w)dw,$$

$$X(u) = \int_{u_0}^{u} \varepsilon(w)dw = \int_{u_0}^{u} \alpha\varepsilon_0(w)dw = \alpha X_0(u), \qquad \text{[A2.2]}$$

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

$$\mu(x) = \mu_0\left(\frac{x}{\alpha}\right). \qquad \text{[A2.3]}$$

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

$$l(x) = \exp\left(-\int_0^x \mu(y)dy\right) = \exp\left(-\int_0^x \mu_0\left(\frac{y}{\alpha}\right)dy\right) =$$

$$= \exp\left(-\alpha\int_0^{x/\alpha} \mu_0(t)dt\right) = \left[l_0\left(\frac{x}{\alpha}\right)\right]^{\alpha}. \qquad \text{[A2.4]}$$

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 (A2.4),

$$\lambda = l(Y(\lambda)) = \left[l_0\left(\frac{Y(\lambda)}{\alpha}\right)\right]^{\alpha}. \qquad \text{[A2.5]}$$

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

$$Y(\lambda) = \alpha Y_0\left(\lambda^{1/\alpha}\right). \qquad \text{[A2.6]}$$

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

$$Y(\lambda) \approx Y_0(\lambda) + \left.\frac{d\left(\alpha Y_0\left(\lambda^{1/\alpha}\right)\right)}{d\alpha}\right|_{\alpha=1} \cdot(\alpha - 1) =$$

$$= Y_0(\lambda) + (\alpha - 1) \cdot \left[Y_0(\lambda) - \frac{dY_0(\lambda)}{d\lambda} \lambda \ln \lambda \right]. \qquad \text{[A2.7]}$$

$Y_0(\lambda)$ 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 [A2.7] 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

$\ln \lambda = \ln(l_0(Y_0(\lambda))) = -\int_0^{Y_0(\lambda)} \mu_0(x) dx$,

$$Y(\lambda) \approx \alpha Y_0(\lambda) - (\alpha - 1) \cdot \int_0^{Y_0(\lambda)} \frac{\mu_0(x)}{\mu_0(Y_0(\lambda))} dx. \qquad \text{[A2.8]}$$

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,

$$\int_0^{Y_0(\lambda)} \frac{\mu_0(x)}{\mu_0(Y_0(\lambda))} dx = \frac{1 - \exp(k(-Y_0(\lambda)))}{k} = \frac{1 - \frac{u_0}{\mu_0(Y_0(\lambda))}}{k} \approx \frac{1}{k}, \qquad \text{[A2.9]}$$

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 [A2.9] 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 [A2.8] may be taken as approximately constant *G* at adult ages (empirically, $G \approx 10$):

$$Y(\lambda) \approx \alpha Y_0(\lambda) - (\alpha - 1) \cdot G. \qquad \text{[A2.10]}$$

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.