Genus

ORIGINAL ARTICLE

Open Access

The timing of the transition from mortality compression to mortality delay in Europe, Japan and the United States



Fanny Janssen^{1,2*} and Joop de Beer²

Netherlands

Abstract

Previous research found evidence for a transition from mortality compression (declining lifespan variability) to mortality delay (increasing ages at death) in low-mortality countries. We specifically assessed the year at which increases in life expectancy at birth transitioned from being predominantly due to mortality compression to being predominantly due to mortality delay in 26 European countries, Japan, and the United States of America (USA), 1950–2014.

To unsmoothed age- and sex-specific death rates from the Human Mortality Database, we applied the CoDe (compression and delay) mortality model.

Among women, the transition first occurred in the USA around 1950, then in North-Western Europe (1955–1970) and Southern Europe (1970–1975), and still later in Eastern Europe. Among men, the transition occurred about 10 years later and is still incomplete in Eastern Europe. We identified four stages: (1) predominance of compression mainly due to mortality declines at young ages, (2) declining importance of mortality compression due to the decreasing impact of mortality declines at young ages, (3) delay becomes predominant due to the increasing impact of mortality delay and the counterbalancing effects of mortality compression/expansion at different ages, and (4) strong predominance of delay accompanied by strong adult mortality declines and declining compression at old ages.

Our results suggest that life expectancy and maximum lifespan will increase further. With mortality delay, premature mortality and old-age mortality are shifting towards older ages.

Keywords: Mortality, Life expectancy, Transition, Modal age at death, Mortality compression, Mortality delay, Shifting mortality

Introduction

Within mortality research, a paradigm shift has occurred: rather than studying trends in the expected average age at death, or life expectancy at birth (e0), researchers are increasingly studying changes over time in the full age-at-death distribution. To describe the changes over time in the age-at-death distribution, two scenarios have been distinguished, which, empirically, can operate simultaneously (Kannisto 2001): (1) mortality compression (Fries 1980), which results from more people dying at the same ages and is indicative of declining lifespan variability or declining lifespan disparities (Bergeron-Boucher et al. 2015);



^{*} Correspondence: f.janssen@rug.nl 1 Population Research Centre, Faculty of Spatial Sciences, University of Groningen, P.O. Box 800, 9700, AV, Groningen, The Netherlands 2 Netherlands Interdisciplinary Demographic Institute, P.O.Box 11650, 2502, AR, The Hague, The

Janssen and de Beer Genus (2019) 75:10 Page 2 of 23

and (2) mortality delay, or the shifting of mortality, whereby the shape of the age-at-death distribution remains intact but shifts to the right, which results in higher ages at death (Kannisto 2001; Bongaarts 2005; Canudas-Romo 2008; Vaupel 2010). Whereas mortality delay results from a decline in mortality across all ages, differences in the rates of decline across ages will cause a change in the shape of the age pattern of mortality: either mortality compression or, sometimes, even mortality expansion (increasing lifespan variability). Mortality delay at older ages is described by an increase in the modal age at death (=the age at which most deaths are occurring)(Canudas-Romo 2008).

By means of decomposition techniques, changes in life expectancy can be decomposed into mortality compression and mortality delay (Rossi et al. 2013; Bergeron-Boucher et al. 2015; de Beer and Janssen 2016). Examining the relative roles of these two types of change and of the potential changes in these developments over time provides us with crucial information not only about the determinants of past mortality trends but also about future trends in both the average and the maximum human lifespan. As long as the delaying of mortality to older ages continues, and, consequently, the modal age at death continues to increase, a limit to life expectancy is unlikely to be reached in the near future, since the ongoing shift in the age pattern of mortality will result in further increases in life expectancy. Moreover, the decrease in death probabilities in old age is contributing to a strong increase in the number of centenarians (Robine and Caselli 2005). This development in turn increases the likelihood that some of these centenarians will survive to very old ages, and thus that the maximum individual lifespan will rise (de Beer et al. 2017).

Previous studies on the changes over time in the age-at-death distribution among low-mortality countries showed that the relative importance of compression (measured by declining variability in the age at death) and of delay (measured by an increase in the modal age at death) to changes in life expectancy are changing over time. Historically, compression of mortality has played the dominant role in low-mortality countries (e.g. Wilmoth and Horiuchi 1999; Kannisto 2000, 2001; Robine 2001; Canudas-Romo 2008, Cheung et al. 2009; Smits and Monden 2009; Engelman et al. 2010; Ouellette and Bourbeau 2011; Bergeron-Boucher et al. 2015). Since the 1950s, the pattern has been changing (Kannisto 1996; Wilmoth and Horiuchi 1999; Kannisto 2000, 2001; Robine 2001; Bongaarts 2005; Edwards and Tuljapurkar 2005; Canudas-Romo 2008; Cheung et al. 2008; Cheung et al. 2009; Thatcher et al. 2010; Ouellette and Bourbeau 2011). A number of scholars who have examined the long-term changes for low-mortality countries have found evidence that a transition from mortality compression to mortality delay has been taking place (Cheung et al. 2005; Canudas-Romo 2008; Ouellette and Bourbeau 2011; Bergeron-Boucher et al. 2015), with Japan leading the way in this transition (Cheung et al. 2009; Ouellette and Bourbeau 2011). Recent formal analysis for low-mortality countries has confirmed that delay is overtaking compression in the change in life expectancy (Janssen et al. 2015; Bergeron-Boucher et al. 2015; de Beer and Janssen 2016). Janssen et al. (2015) showed that the contribution of mortality compression to the overall gain in remaining life expectancy at age 50 between 1956 and 2009 was consistently less than 50% for the ten examined European low-mortality countries. Bergeron-Boucher et al. (2015) found that from 1965 onwards, more than 70% of the increase in e0 among Swedish women was caused by delay. De Beer and Janssen (2016) showed that two thirds of Janssen and de Beer Genus (2019) 75:10 Page 3 of 23

the increase in e0 between 1950 and 2010 in Japan, France, the USA, and Denmark was due to delay.

Until now, however, no studies have examined the existence and the timing of a transition from changes in e0 being predominantly due to mortality compression to changes in e0 being predominantly due to mortality delay simultaneously for a large number of countries, including Eastern European countries, or the role of compression at different ages in this potential transition.

Previous cross-national studies have demonstrated that there have been large differences between countries in the extent of delay and compression (e.g. Wilmoth and Horiuchi 1999; Edwards and Tuljapurkar 2005; Smits and Monden 2009; Canudas-Romo 2008; Cheung et al. 2008; Engelman et al. 2010; Thatcher et al. 2010; Shkolnikov et al. 2011; Ouellette and Bourbeau 2011; Gillespie et al. 2014; Bergeron-Boucher et al. 2015; Janssen et al. 2015; Muszyńska and Janssen 2016). For example, one study that examined mortality compression for an Eastern European country (Shkolnikov et al. 2003) observed that the variability in the age at death increased after 1950 in Russia, a trend that is indicative of mortality expansion rather than mortality compression. Other research has shown that there are large differences in variability among countries with similar levels of life expectancy (Smits and Monden 2009) and that the variability in the age at death increased in the USA in the 1980s and the early 1990s (Shkolnikov et al. 2011). Thus, it is likely that the timing of the transition from mortality compression to mortality delay differs between countries as well.

Recent studies have acknowledged the importance of distinguishing between compression at different ages (e.g. Zhang and Vaupel 2009; Bergeron-Boucher et al. 2015; de Beer and Janssen 2016). In their study of the "dynamic process of mortality compression", Zhang and Vaupel (2009) clearly demonstrated that overall mortality compression depends on the different processes that occur at younger versus older ages. Vaupel et al. (2011) also emphasised that whereas a reduction in premature deaths diminishes lifespan disparities, a reduction in old-age mortality increases lifespan disparities. Goldstein and Cassidy (2012) showed that slowing of senescence, i.e. more reduction of death rates at older ages than at middle age, results in expansion of mortality, while an equal reduction in death rates across all ages results in a shift of the age-at-death distribution.

Also, the results of previous studies on compression seem to depend heavily on whether these analyses examined compression over all ages, or for a selection of ages only. For example, previous studies that focused on overall compression (e.g. Wilmoth and Horiuchi 1999; Robine 2001; Yashin et al. 2001; Bergeron-Boucher et al. 2015) generally found that compression of mortality had been stable at least since the 1970s; whereas, the studies that focused on compression above the modal age at death showed that compression continued through the 1970s and the 1980s (Thatcher et al. 2010; Kannisto 2001; Cheung et al. 2008), and that stagnating trends have only recently been observed in some countries (Cheung et al. 2009).

In this paper, we will assess the timing of the point at which increases in e0 transitioned from being predominantly due to mortality compression to being predominantly due to mortality delay in 26 European countries and in Japan and the USA over the

Janssen and de Beer Genus (2019) 75:10 Page 4 of 23

1950–2014 period. We will also examine the role mortality compression at young, adult, and old ages that plays in this transition.

Data and methods

For our analysis, we used unsmoothed age-specific death rates from the Human Mortality Database (2017) by single year of age (up to age 100), by year (1950–2014), and by sex for all European countries with data from at least 1959 onwards, as well as for Japan and the USA. After excluding Estonia, Iceland, and Luxembourg because of data issues, our sample consisted of 26 European countries, Japan, and the USA.

We divided the European countries into four main regions: Northern Europe (Denmark, Finland, Ireland, Norway, Sweden, United Kingdom (UK)), Western Europe (Austria, Belgium, Switzerland, West Germany, France, the Netherlands), Southern Europe (Italy, Portugal, Spain), and Eastern Europe. We based these geographic categories on those of the United Nations (2016), although we made some adjustments to enable that countries with similar e0 values and/or e0 trends were grouped together. That is, unlike the United Nations, we placed Latvia and Lithuania in Eastern Europe and distinguished between eastern Germany (Eastern Europe) and western Germany (Western Europe). We then subdivided the Eastern European countries into the following categories: former Soviet republics (Belarus, Russia, and Ukraine), Baltic countries (Lithuania and Latvia), and remaining Eastern European countries (Bulgaria, the Czech Republic, East Germany, Hungary, Poland, and Slovakia).

For our modelling exercise, we used the parametric CoDe mortality model (Compression and delay mortality model), which has been previously described, validated, and discussed (de Beer and Janssen 2016). We have chosen the CoDe model because in addition to providing a good model for fitting the full age pattern of mortality (de Beer and Janssen 2016), it enables us to distinguish between mortality delay and mortality compression, and-in doing so-between mortality compression at young, adult, and old ages. Previous models describing the full age pattern of mortality (the Thiele model, the Heligman-Pollard model, the Siler model, the Rogers & Little model, and the Kostaki adaptation of the Heligman-Pollard model) could not capture mortality delay and mortality compression (de Beer and Janssen 2016). Previous models that included mortality delay did not fit the full age pattern of mortality, but instead fitted only adult mortality (Bongaarts 2005; Horiuchi et al. 2013). The few previous studies that assessed the relative contributions of mortality delay and mortality compression did not distinguish between mortality compression at young, adult, and old ages (Rossi et al. 2013 using the method developed by Rousson and Paccaud 2010; Bergeron-Boucher et al. 2015 using the Siler model; Börger et al. 2018 in their classification system).

The CoDe mortality model uses five additive terms representing mortality at successive life stages (child mortality, teenage mortality, and mortality during young adult, middle, and old age) and includes the modal age at death as a parameter to account for the delay in mortality. The model describes child and teenage mortality with two simple functions and uses a mixed logistic model with different slopes for young adult, middle, and old ages. The CoDe mortality model is defined by:

Janssen and de Beer Genus (2019) 75:10 Page 5 of 23

$$\begin{split} q(x) &= \frac{A}{x+B} + \frac{ae^{(x-16)}}{1+e^{(x-16)}} + I(x \le M-h) \left[\frac{b_1 e^{b_1(x-M)}}{1+\frac{b_1}{g} e^{b_1(x-M)}} \right] \\ &+ I(M-h < x \le M) \left[\frac{b_2 e^{b_2(x-M)}}{1+\frac{b_2}{g} e^{b_2(x-M)}} + c_1 \right] + I(x > M) \left[\frac{b_3 e^{b_3(x-M)}}{1+\frac{b_3}{g} e^{b_3(x-M)}} + c_2 \right] \end{split} \tag{1}$$

where q(x) is the death probability at age x, A reflects infant mortality, B affects the decline in mortality with age at young ages, a reflects the level of teen mortality. To distinguish the different life stages after teen mortality, we used an indicator function I(.), with M the modal age at death and h assumed to be 30, and added constants c_1 and c_2 —which are calculated from the other parameters—to enable smooth patterns over age. b_1 and b_2 determine the increase in mortality with age at ages representing adult premature mortality and b_3 determines the increase in mortality with age above the modal age. We assume that g, the upper bound of q(x), equals 0.7 (de Beer and Janssen 2016).

Appendix 1 illustrates the effects of the main parameters of the CoDe model on the age-at-death distribution. An increase in the modal age M describes the delay in mortality, i.e. the shift in the mortality age pattern from younger to older ages while the age-at-death distribution retains its shape—that results in increasing life expectancy at birth (e0). The increase in the modal age also implies that the normal age at death increases. Without changes in old-age mortality compression, a delay in mortality also implies a higher maximum age at death. Declines in the level of infant mortality A and teen mortality a result in compression of mortality: as mortality declines at these ages, a higher concentration of deaths around the modal age at death occurs. This compression of mortality due to mortality declines at young ages (hereafter referred to as compression at young ages) results in increases in e0. An increase in the parameters b_1 , b_2 , and b_3 affects the slope of the age curve of death probabilities and results in an increase in the steepness of the age-at-death distribution at adult (b_1, b_2) and old ages (b_3) , relative to the modal age at death and, consequently, in declining lifespan variability (=mortality compression). Compression below the modal age as a result of an increase in b_1 and b₂ results in fewer deaths at ages far below the modal age and more deaths at ages just below the modal age, which in turn results in an increase in e0. This compression below the modal age at adult ages is hereafter referred to as compression at adult ages. Compression of deaths at young ages combined with compression of deaths at adult ages, and thus all compression below the modal age, is referred to as compression of premature mortality. Compression of deaths above the modal age due to an increase in b_3 results in fewer deaths at very old ages, but more deaths at ages slightly above the modal age. In this case, e0 declines. Compression of deaths at ages above the modal age also means that relatively fewer people are achieving exceptional longevity. This compression above the modal age is hereafter referred to as compression of old-age mortality.

Janssen and de Beer Genus (2019) 75:10 Page 6 of 23

The CoDe model takes into account that premature mortality and old-age mortality are relative concepts. The ages that are considered "premature" and "old" depend on the modal age at death. We, therefore, go beyond previous research that studied age-specific mortality trends only, or that distinguished between premature and late deaths using a fixed age of either 65 or 75. Unlike in Zhang and Vaupel (2009), our threshold age is directly linked to the modal age at death, which enables us to compare our results with the findings of previous studies that focused on compression above the modal age. On the other hand, as illustrated in the last paragraph of the "Country and sex differences in the timing of the transition from mortality compression to mortality delay" section, this also means that when there is a large decline in the modal age at death, the likelihood of observing mortality compression at adult ages and mortality expansion at older ages is larger. Similarly, when there is a large increase in the modal age at death, the likelihood of observing mortality compression at old ages is more likely than mortality expansion.

We estimated the model parameters in R, using non-linear minimization (nlm), and using as starting values for M the observed modal age at death, above age 30, and a range surrounding that age (-3, -2, -1.5, -1, -0.5, -0.25, +0.25, +0.5, +1, +1.5, +2, +3)and selected the outcome with the lowest weighted least squares (WLS), where we weighted the squared errors in death probabilities, the logarithm of death probabilities, and the density of the age-at-death distribution by their standard errors (de Beer and Janssen 2016). Based on a careful analysis of the WLS and visual inspection of the resulting trends in the parameters, we decided to omit the estimates for Bulgaria in 1950 and 1951, and for Latvia in 1959, and to use linear interpolation of the estimates in another 11 single cases. In all remaining cases, the model fit of the CoDe model turned out to be good. From the last column in Table 1, it can be observed, for example, that the absolute residual effect was in general low and on average 0.17 years for men and 0.10 years for women over an average change in e0 over 1980-2014 of 7.1 and 5.9 years, respectively. For 1950-2014, the average absolute residual effect was respectively 0.21 for men and 0.19 for women over an average change of 12.1 and 13.5 years, respectively.

We decomposed the change in fitted e0 from 1950 to 2014 into changes in the modal age and changes in mortality compression at young, adult, and old ages. In doing so, we distinguished between the sub-periods of 1950 to 1979 and 1980 to 2014, and separate 10-year sub-periods from 1950 and 1955 onwards. Our decision to divide our observation period into the 1950-1979 and 1980-2014 sub-periods was based on our finding that the trends in M before and after 1975-1980 were quite distinct (see the first paragraph of the "Results" section) and on our wish to divide the overall period into sub-periods that were as equal as possible. Our decomposition involved the cumulative adjustment of the subsequent parameters to the final level, while keeping the remaining parameters at the starting level. We used the following order: A + B, a, M, b_1 , b_2 , and b_3 , to enable that indeed compression is measured relative to the modal age at death. We eliminated the effect of c_2 in compression below the modal age (b_1, b_2) to ensure that a decline in the probability of dying before reaching the modal age does not affect the probability of dying after reaching the modal age. Similarly, the effect of c_1 was eliminated from compression at young adult ages (b_1) . Unweighted average contributions were calculated for the different regions.

Janssen and de Beer *Genus* (2019) 75:10 Page 7 of 23

In order to determine in a robust manner the timing of the transition from changes in e0 being predominantly due to mortality compression to changes in e0 being predominantly due to mortality delay, we assessed the 5-year period in which the contribution of delay to the change in e0 became larger than the contribution of compression. We did so by smoothing the yearly contributions of delay and compression to the change in e0 by means of 3-year moving averages (resulting in yearly contributions from 1951-1952 up until 2012–2013) and subsequently summing these yearly contributions over the 5-year periods (1951–1955, 1955–1960,..., 2005–2010, 2010–2013). To ensure that the change was long-lasting, we considered a minimum of two consecutive 5-year periods that had to last until the end of the observation period. We allowed the long-lasting change to be interrupted by a single 5-year period in which compression was still more important than delay, but only if before and after this 5-year period there were two subsequent periods in which the delay was more important than compression.

Results

Past trends in the modal age at death

Figure 1 shows the trends over time (1950–2014) in the estimated modal age at death (M) for the different countries. The figure clearly displays the different trends in M before and after 1975–1980, particularly for men. Among men, M hardly increased up to 1975–1980. However, from 1975–1980 onwards, M among men increased strongly in the non-Eastern European countries; whereas, in the Eastern European countries, M among men stagnated and underwent significant temporal declines. Only recently have increases in M again been observed among men in Eastern Europe. Among women, M increased quite linearly throughout the 1950–2014 period in the non-Eastern European countries but increased less strongly in the Eastern European countries up to 1975–1980. As a result, women in Eastern Europe clearly had lower levels of M than women in non-Eastern Europe from 1975–1980 onwards.

Relative importance of compression and delay in the trends in life expectancy

Figure 2 shows the relative effects of mortality compression and mortality delay on the trends in life expectancy at birth (e0) over the 1950-2014 period, divided into 1950-1979 and 1980-2014. In the 1950-1979 period, the changes in e0 were largest among Japanese women and smallest among Eastern European men. Whereas the increase in e0 tended to be greater among women in 1950-1979, it tended to be greater among men in 1980-2014. Mortality compression generally played a bigger role than mortality delay up to 1980, which suggests that the increases in e0 occurred primarily because of declining disparities in the ages at death that resulted from large declines in infant and child mortality. From 1980 onwards, however, mortality delay was more important than compression, which indicates that declines in mortality across all ages led to a shift in the distribution of deaths towards older ages. On the contrary, among Eastern European men, delay contributed less than compression to the change in e0 also over the 1980-2014 period. This can be related to the significant temporal declines in M from 1975 onwards among men in many Eastern European countries (Fig. 1). Among men in Bulgaria, Lithuania, Latvia, Belarus, and Ukraine, these declines resulted in substantial negative effects on e0 over the 1980-2014 period (Table 1).

Janssen and de Beer Genus (2019) 75:10 Page 8 of 23

Table 1 Contributions (in years) of changes in the modal age at dying (*M*) and of mortality expansion/compression at young, adult, and old ages in the change in life expectancy at birth (e0) between 1980 and 2014*, by country and sex

	Observed	Observed	Observed	Effect	Effect of	Effect compression/expansion		ansion	Effect
	e0 1980	e0 2014	Change e0	Change M		Before M		After M	Residual
			1980-2014		Total	Young	Adult	Old age	
Men									
Japan	73.38	80.52	7.14	6.70	0.47	1.09	- 0.47	- 0.15	- 0.03
USA	69.99	76.66	6.67	8.17	- 1.23	1.90	- 2.58	- 0.56	- 0.26
Denmark	71.17	78.55	7.38	7.75	- 0.22	1.98	- 1.48	- 0.72	- 0.15
Finland	69.23	78.12	8.89	9.48	- 0.43	1.07	- 0.79	- 0.71	- 0.17
Ireland	69.93	79.16	9.23	10.20	- 0.73	1.39	- 1.46	- 0.66	- 0.24
Norway	72.34	80.02	7.68	7.74	- 0.08	1.82	- 1.12	- 0.78	0.02
Sweden	72.78	80.35	7.57	6.60	1.05	0.84	0.63	- 0.43	- 0.08
UK	70.51	79.01	8.49	10.13	- 1.52	1.65	- 2.50	- 0.67	- 0.11
Austria	68.97	78.92	9.95	8.23	1.91	2.62	- 0.39	- 0.33	- 0.20
Belgium	69.88	78.57	8.70	8.97	- 0.11	2.16	- 1.71	- 0.56	- 0.17
The Netherlands	72.44	79.88	7.44	8.41	- 0.99	1.67	- 1.77	- 0.89	0.02
Germany, West	69.87	78.67	8.80	8.08	0.97	2.30	- 0.94	- 0.40	- 0.25
France	70.16	79.27	9.11	8.94	0.61	1.92	- 0.85	- 0.46	- 0.43
Switzerland	72.23	80.93	8.70	9.05	- 0.25	2.28	- 1.86	- 0.68	- 0.10
Italy	70.67	79.72	9.05	8.08	1.09	1.95	- 0.30	- 0.56	- 0.12
Portugal	68.11	77.93	9.82	7.36	2.91	4.09	- 0.82	- 0.37	- 0.44
Spain	72.39	80.10	7.71	6.64	1.34	1.90	- 0.27	- 0.28	- 0.28
Bulgaria	68.44	70.31	1.87	- 1.26	3.26	1.70	1.00	0.56	- 0.13
Czech Republic	66.81	75.72	8.91	6.94	2.03	1.89	0.18	- 0.04	- 0.07
Germany, East	68.71	77.42	8.71	8.20	0.92	2.43	- 1.29	- 0.22	- 0.41
Hungary	65.52	72.26	6.74	0.04	6.82	2.04	3.42	1.37	- 0.12
Poland	65.76	73.66	7.90	1.41	6.51	2.29	3.22	1.00	- 0.01
Slovakia	66.71	73.25	6.54	1.69	4.80	1.21	2.94	0.64	0.05
Lithuania	65.58	68.52	2.94	- 4.99	8.24	2.05	4.58	1.61	- 0.31
Latvia	63.74	69.25	5.52	- 1.78	7.55	3.51	2.77	1.26	- 0.25
Belarus	65.95	67.81	1.85	- 4.27	6.11	1.82	3.26	1.04	0.01
Russia	61.39	65.26	3.87	1.05	2.98	2.04	0.54	0.40	- 0.16
Ukraine	64.62	66.31	1.69	- 3.17	4.97	1.56	2.37	1.04	- 0.11

Janssen and de Beer Genus (2019) 75:10 Page 9 of 23

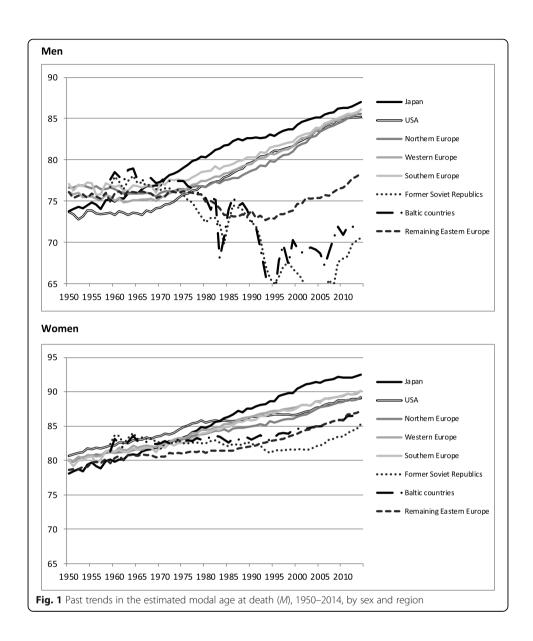
Table 1 Contributions (in years) of changes in the modal age at dying (*M*) and of mortality expansion/compression at young, adult, and old ages in the change in life expectancy at birth (e0) between 1980 and 2014*, by country and sex (*Continued*)

	Observed	Observed	Observed	Effect	Effect of	compress	sion/exp	ansion	Effect
	e0 1980	e0 2014	Change e0	Change M		Before M		After M	Residual
			1980-2014		Total	Young	Adult	Old age	
Women									
Japan	78.75	86.89	8.13	7.70	0.28	0.69	- 0.28	- 0.13	0.14
USA	77.48	81.48	4.00	3.56	0.47	0.73	- 0.06	- 0.20	- 0.03
Denmark	77.18	82.67	5.50	3.41	1.99	0.43	1.63	- 0.08	0.10
Finland	77.86	83.84	5.99	6.00	0.04	0.66	- 0.30	- 0.32	- 0.05
Ireland	75.38	83.24	7.87	7.63	0.03	0.86	- 0.43	- 0.41	0.21
Norway	79.18	84.09	4.91	4.57	0.51	0.78	- 0.03	- 0.24	- 0.16
Sweden	78.85	84.06	5.20	4.32	1.03	0.46	0.82	- 0.24	- 0.15
UK	76.57	82.78	6.21	5.54	0.57	0.82	0.10	- 0.35	0.10
Austria	76.06	83.75	7.69	6.76	0.96	1.33	- 0.08	- 0.29	- 0.02
Belgium	76.66	83.53	6.87	6.18	0.80	1.22	- 0.16	- 0.26	- 0.11
the Netherlands	79.13	83.30	4.16	4.01	0.22	0.79	- 0.29	- 0.28	- 0.06
Germany, West	76.62	83.35	6.72	5.83	1.04	1.29	- 0.01	- 0.24	- 0.15
France	78.41	85.45	7.05	6.16	0.95	1.20	- 0.07	- 0.18	- 0.06
Switzerland	78.85	85.12	6.27	5.31	0.86	1.06	0.06	- 0.26	0.10
Italy	77.42	84.47	7.04	5.76	1.31	1.25	0.30	- 0.25	- 0.03
Portugal	75.21	84.15	8.94	6.67	2.25	2.68	-0.10	- 0.32	0.01
Spain	78.55	85.65	7.10	5.96	1.06	1.16	0.17	- 0.27	0.08
Bulgaria	73.90	77.26	3.35	3.61	- 0.32	1.37	- 1.43	- 0.27	0.07
Czech Republic	73.93	81.73	7.80	7.04	0.93	1.19	- 0.03	- 0.23	- 0.17
Germany, East	74.64	83.42	8.78	7.65	1.21	1.25	0.19	- 0.23	- 0.09
Hungary	72.76	79.25	6.50	5.04	1.82	1.55	0.34	- 0.06	- 0.36
Poland	74.22	81.43	7.21	5.82	1.52	1.76	- 0.08	- 0.16	- 0.13
Slovakia	74.26	80.32	6.06	4.90	1.17	1.13	0.10	- 0.06	- 0.02
Lithuania	75.64	79.34	3.70	3.67	0.17	1.43	- 0.82	- 0.43	- 0.15
Latvia	74.11	78.73	4.63	3.66	1.21	1.77	- 0.36	- 0.19	- 0.25
Belarus	75.61	78.43	2.83	1.90	0.88	1.39	- 0.12	- 0.38	0.04
Russia	72.97	76.48	3.51	2.96	0.58	1.20	- 0.46	-0.17	- 0.02
Ukraine	74.06	76.21	2.15	1.88	0.22	1.06	- 0.67	- 0.18	0.04

^{*2010} for Bulgaria; 2012 for Italy; 2013 for UK, Lithuania, Latvia, and Ukraine

Considerable differences in the relative importance of compression and delay in the change in e0 from 1980 to 2014 showed (1) between the regions, with Southern Europe experiencing more compression than Northern and Western Europe; (2) between the sexes, with more delay among men especially in Northern and Western Europe and the USA; and (3) between the individual countries (Fig. 2, Table 1).

Janssen and de Beer *Genus* (2019) 75:10 Page 10 of 23



Timing of the transition from a predominance of compression to a predominance of delay

The transition from a predominance of compression to a predominance of delay in the increases in e0 started earlier among women than among men and occurred earlier in Northern and Western Europe than in Southern and Eastern Europe (Table 2).

Among women in the USA, the effect of delay was larger than the effect of compression throughout the period. The onset of the transition occurred between 1955 and 1970 among women in Japan and in most other Northern and Western European countries, between 1970 and 1980 in the three Southern European countries and between 1970 and 1995 in all of the Eastern European countries, except Belarus (2000) and Latvia (2005). Among men, the transition from a predominance of compression to a predominance of delay occurred later, from 1965 onwards. Like the transition among women, the transition among men started relatively early in the USA, Northern and Western Europe, and in Japan, albeit surprisingly late in Denmark, Norway, and the Netherlands. Among men in Southern Europe, the

Janssen and de Beer Genus (2019) 75:10 Page 11 of 23

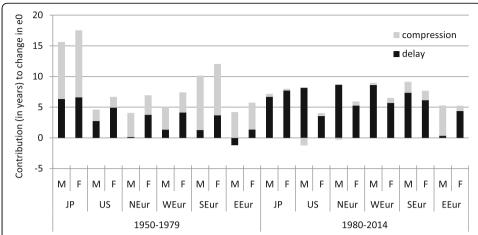


Fig. 2 Contributions (in years) of mortality delay (or declining *M* when negative) and mortality compression (or expansion when negative) to changes in life expectancy at birth (e0), 1950*-1979 and 1980–2014*, by sex. *1952 for Belarus, 1956 for East and West Germany; 1958 for Poland; 1959 for Belarus, Lithuania, Russia and Ukraine; 1960 for Latvia. *2010 for Bulgaria; 2012 for Italy; 2013 for UK, Lithuania, Latvia, and Ukraine. JP Japan, US United States, NEur Northern Europe, WEur Western Europe, SEur Southern Europe, EEur Eastern Europe

transition started between 1975 and 1985, and even later in Eastern Europe. Among men in Lithuania, Latvia, and Belarus, there is no evidence that the transition from compression to delay has occurred.

The importance of compression at young, adult, and old ages in the transition

In the 1950s in non-Eastern Europe, the predominance of mortality compression was caused by the large contribution of mortality decline at young ages to the increase in e0 (Fig. 3). In addition, among men, the modal age was either only marginally increasing or even declining (Fig. 1). For women in non-Eastern Europe, delay was already contributing on average 44% of the increase in e0.

Between the 1950s (1950–1959) and the 1960s (1960–1969), the decline in the relative role of compression in the increase in e0 was mainly due to a decrease in the importance of mortality declines at young ages. This trend occurred across Europe among both men and women.

Between the 1960s and the 1970s (1970–1979), the role of mortality delay increased considerably in non-Eastern Europe (Fig. 3). Mortality delay first became predominant from 1970 onwards among women in North-Western European countries and later became predominant among women in Southern Europe and among men (see as well Appendix 2). In this period in which delay was becoming predominant, the positive contribution of compression of mortality at young ages to the change in e0 was counterbalanced by the negative contributions of expansion at adult ages and compression at old ages, especially among men (Fig. 3).

In Eastern Europe (see Appendix 2), the period from 1975 to 1995 is characterised by expansion of adult mortality among women, and, among men, large negative contributions from a decline in the modal age at death only partly counterbalanced by compression at adult ages and expansion at old ages. From 1975 onwards for women and 1995 onwards for men, a clear increase in the contribution of delay can be observed, coupled with compression of old-age mortality,

Janssen and de Beer Genus (2019) 75:10 Page 12 of 23

Table 2 Five-year period in which the contribution of mortality delay to the increase in life expectancy at birth became persistently larger than the contribution of mortality compression, by sex and country, 1951–2013

Country	Men	Women
Japan	1965–1970	1965–1970
USA	1965–1970	1951–1955*
Denmark	1980–1985	1995–2000 [†]
Finland	1965–1970	1965–1970
Ireland	1985–1990	1975–1980
Norway	1980–1985	1965–1970
Sweden	1965–1970	1955–1960*
UK	1965–1970	1960–1965*
Austria	1970–1975*	1970–1975
Belgium	1970–1975	1970–1975
The Netherlands [#]	1975–1980	1955–1960
Germany, West	1970–1975	1970–1975
France	1965–1970	1960–1965
Switzerland	1965–1970	1965–1970
Italy	1985–1990	1975–1980
Portugal	1985–1990	1975–1980
Spain	1975–1980	1970–1975
Bulgaria [#]	2000–2005	1975–1980
Czech Republic [#]	1990–1995*	1985–1990
Germany, East [#]	1990–1995 [†]	1975–1980
Hungary [#]	1995–2000	1980–1985
Poland [#]	1990–1995*	1970–1975*
Slovakia [#]	1990–1995	1985–1990
Lithuania [#]	N/A	1970–1975*
Latvia [#]	N/A	2005-2010
Belarus [#]	N/A	2000–2005
Russia [#]	2005-2010 [†]	1995–2000
Ukraine [#]	2005–2010	1995–2000

N/A = not applicable

resulting in a predominance of delay from 1990 onwards among women, and from 1995 onwards among men.

In more recent years, in non-Eastern Europe, delay remained highly predominant, and at about the same relative level, despite some fluctuations in its contribution because of fluctuations in the contribution of young age mortality, in particular teenage mortality (Fig. 3; Appendix 2). Whereas, the negative contributions of expansion of adult mortality

[#]At least two 5-year periods in which declines in e0 occurred, mostly only among men, but for Belarus, Latvia, Russia, and Ukraine for both men and women

^{*}One 5-year period in between in which the contribution of compression was larger than delay

[†]Also at an earlier point in time, an extended period occurred in which absolute delay was more important than absolute compression: DKF 1951–1980, RFM 1980–1990, DEM 1970–1980

Janssen and de Beer Genus (2019) 75:10 Page 13 of 23

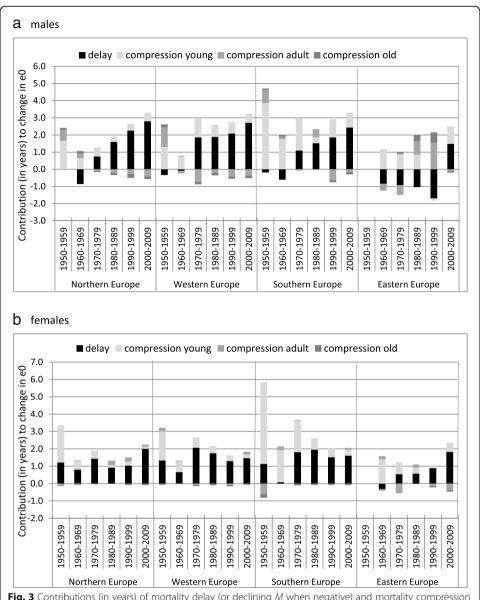


Fig. 3 Contributions (in years) of mortality delay (or declining *M* when negative) and mortality compression (or expansion when negative) at young, adult, and old ages to the changes in life expectancy at birth (e0), for the separate decades from 1950* to 2009 [results for 1955 to 2014 in Appendix 2]. **a** Males. **b** Females.*For West Germany from 1956 onwards. For Eastern Europe from 1960 onwards

have been declining among men, the positive contributions of compression of adult mortality have recently emerged among women, indicating that declines in adult mortality are particularly strong. Despite further increases in the modal age at death (Fig. 1), the small negative contribution of compression at old-age mortality stayed either stable or declined slightly, reflecting the absence of a further recent increase in the related parameter, with even signs of expansion recently for some countries (see Appendix 3).

Discussion of observed results

In almost all studied 26 European countries, Japan, and the USA, a transition occurred from increases in life expectancy at birth (e0) mainly due to compression of mortality to increases

Janssen and de Beer *Genus* (2019) 75:10 Page 14 of 23

in e0 predominantly due to mortality delay. Delay of mortality results from a shift in the age distribution of mortality towards older ages and implies an increase in the modal age at death. Compression of mortality implies that the share of deaths around the modal age increases and that, consequently, lifespan variability is declining. Compression can be caused by a strong decrease in deaths at young or advanced ages, which has a positive effect on e0, or by an increase in the share of deaths around the modal age (as a result of a decline in the share of deaths in advanced age), which results in a negative effect on e0.

Country and sex differences in the timing of the transition from mortality compression to mortality delay

Important differences between men and women and across countries occurred in the timing of the point at which increases in e0 transitioned from being predominantly due to compression of mortality to being predominantly due to mortality delay. Specifically, we found that, for women, the transition started at or before 1950 in the USA, between 1955 and 1970 among women in Northern and Western Europe, between 1970 and 1975 among women in Southern Europe, and still later among women in Eastern Europe. Generally, the transition occurred among men about 10 years later than among women, although it is important to note that delay has not yet overtaken compression among men in Lithuania, Latvia, and Belarus. Our own additional analysis using data for American women from 1933 onwards showed that the onset of the transition began in the 1951–1955 period and not earlier.

Generally, the transition from compression to delay appears to have occurred around 10 years later among men than among women. This observation is related to the differences between men and women in the increase in the modal age at death (Cheung et al. 2009), which were more modest among men than among women from 1950 up to 1975 (see Fig. 1). At least among men and women in non-Eastern European countries, these sex differences in the increase in the modal age can be partly attributed to the differential effects of the smoking epidemic for men and women. Men-and particularly men in the Anglo-Saxon countries and North-Western Europe—were the first to take up smoking in large numbers (Lopez et al. 1994). Women did not start smoking until some decades after men (Lopez et al. 1994; Janssen and van Poppel 2015). By that time, the negative effects of smoking on health were widespread and well-known. Consequently, the smoking prevalence levels of women never reached the enormously high levels of men (Lopez et al. 1994; Van Poppel and Janssen 2016). In line with this hypothesis regarding the impact of the smoking epidemic on sex differences in the increase in the modal age, Janssen and de Beer (2016) observed for the Netherlands that the trends in the modal age at death for non-smoking-related mortality—i.e. with the effects of smoking excluded—were roughly the same among men and women aged 40 and older over the period 1950-2012. Similarly, Janssen et al. (2015) observed that in a number of European low-mortality countries, from 1950 to 2009, patterns in longevity extension among men and women aged 50 and older were more similar for non-smoking-related mortality than for all-cause mortality. For Eastern European countries, sex differences in alcohol consumption might be important as well. Because men tend to consume more alcohol than women (Leon et al. 2009; Mäkelä et al. 2006), they also have higher levels of alcohol-attributable mortality (e.g. Trias-Llimós et al. 2017). This gender gap is especially striking in Eastern Europe (e.g. Trias-Llimós and Janssen 2018).

Janssen and de Beer Genus (2019) 75:10 Page 15 of 23

Differences between countries in the timing of the onset of the predominance of delay may have also been affected by the smoking epidemic. There are clear differences between countries in the impact and the timing of the smoking epidemic, as described by Lopez et al. 1994 in their smoking epidemic model. Men in Anglo-Saxon and North-Western European countries were the first to take up smoking in large numbers at the beginning of the twentieth century resulting in very high smoking-attributable mortality among these men some 30 to 40 years later. On average, men in Southern and Eastern Europe took up smoking 35 years later, during a period when the adverse health effects of smoking became increasingly known. Thus, among these men, smoking prevalence and smoking-attributable mortality was lower, although still substantial. The women in the abovementioned countries followed a similar pattern of tobacco use but with a delay of several decades (e.g. Lopez et al. 1994) and experienced lower smoking prevalence and smoking-attributable mortality than men.

It is likely, however, that our general observation that the transition occurred earlier in Northern and Western Europe, the USA, and Japan, and later in Southern and Eastern Europe, is mainly attributable to historic differences in socioeconomic developments and improvements in medical care, with greater improvements leading to larger historic declines in under-five mortality in particular and to higher current life expectancy values (e.g. Omran 1998; Mackenbach 2013). Higher life expectancy is known to be associated with smaller lifespan disparities (Vaupel et al. 2011; Smits and Monden 2009), and thus with improvements resulting primarily from a shift in the distribution of deaths towards older ages.

However, the transition from increases in e0 being predominantly due to compression of mortality to increases in e0 being predominantly due to mortality delay has not yet occurred in all European countries. Among men in Lithuania, Latvia, and Belarus, delay has not yet overtaken compression. In these and other Eastern European male populations, large (temporal) declines in e0 occurred from 1975 onwards. These declines are largely attributable to the health crisis in Eastern Europe that resulted from the policies of communist regimes (McKee and Shkolnikov 2001; Vallin and Meslé 2004; Leon 2011). This health crisis affected men and women of all ages, but particularly men and people of adult ages (McKee and Shkolnikov 2001). In line with our expectations, we found that the health crisis caused adult mortality to expand substantially among women in 1975-1984 and 1985-1994. However, the effects of the health crisis were larger among men, and because the crisis affected the broader population, it resulted among men predominantly in a decline in the normal (=modal) age at death. As the normal age at death decreased, lifespan disparities below the modal age at death also declined (=compression of adult mortality) simply because the distance of the onset of adult mortality to the normal age at death became smaller, and the increase in mortality with age steeper. As well as illustrating the relative concept of compression below the modal age at death, this pattern shows that a health crisis can result in a decline in the modal age at death, as a result of which a decline in lifespan variability below the modal age at death does not necessarily result in a positive effect on life expectancy. Similarly, the expansion of old-age mortality that was observed in this period while the modal age at death was declining took place Janssen and de Beer Genus (2019) 75:10 Page 16 of 23

because the older ages represented more ages than they did before the decline in the modal age at death occurred, which inevitably led to a decline in the steepness of mortality with age (=expansion of old-age mortality).

Phases in the transition from compression to delay

Distinguishing between compression/expansion at different ages allowed us to provide a more detailed description of the overall transition from changes in e0 being predominantly due to mortality compression to changes in e0 being predominantly due to mortality delay. More specifically, we could distinguish the following phases from 1950 onwards: (1) compression played a large and predominant role mainly because of mortality compression at young ages due to large mortality declines at young ages, while delay had very little effect; (2) mortality compression became less important as the effects of mortality compression and mortality declines at young ages tapered off; (3) mortality delay became more important than mortality compression due to large increases in the modal age at death, as well as to the counterbalancing effects of mortality compression at young ages on the one hand, and of mortality expansion at adult ages and old-age compression on the other; and (4) strong predominance of delay combined with larger mortality declines at adult ages and declining mortality compression at old ages.

Our description of the different stages from 1950 onwards could supplement the description of the current stage of the epidemiological transition theory in low-mortality countries (Omran 1998). Based on his study of the dispersion of lifespans in France, Robine (2001) proposed an alternative third stage of the epidemiological transition theory, i.e. instead of "the age of degenerative and man-made diseases", he suggested "the age of the conquest of the extent of life", a stage that is characterised by increasing life expectancies combined with no or very small reductions in lifespan dispersion. In our analysis, we could clearly discern different subphases in the transition from mortality compression to mortality delay that could be used to supplement the description of the third and later stages of the epidemiological transition theory.

As mortality delay has been the main contributor to the increase in e0 in recent years, the definition of premature mortality and old-age mortality has been changing as well. As first presented by Lexis (1878) and later revived by Kannisto (2001), the modal age at death can be seen as representing "normal" or "typical" longevity (Cheung et al. 2005). Thus, when the modal age increases, an older age at death becomes normal. Since premature mortality refers to mortality at an age below the point in the lifespan at which dying is considered normal, premature mortality is extended to higher ages. Similarly, since old-age or late mortality refers to mortality at an age above the point in the lifespan at which dying is considered normal, old-age mortality is shifted to higher ages. In the 28 countries studied here, the normal age at death among women was on average 81.4 years in 1960 (80.2 in the earliest available year) and 87.7 years in 2010 (88.4 in the latest available year), while among men, this was 76.2 years in 1960 and 80.4 years in 2010. If Eastern European countries are excluded, the equivalent values for men are 75.8 and 83.2. Thus, whereas age 83 was considered old in 1960, dying at this age can now be considered normal among men and premature among Janssen and de Beer *Genus* (2019) 75:10 Page 17 of 23

women in the majority of European countries. This is important information for policy-makers who are trying to further reduce premature mortality in the context of mortality delay. It is clear that policies should be adjusted to target an older group of people than in the past.

Although mortality delay is increasing in importance, mortality compression has also played a role in recent mortality changes. Tracking mortality compression and mortality expansion provides us with information about declines or increases in lifespan disparities between individuals, and this information can be indicative of underlying inequalities, such as socioeconomic differences that could be associated with differences in smoking habits (van Raalte et al. 2011). Whereas lifespan disparities at younger ages predominated in the past, today lifespan disparities at adult and older ages are becoming more important.

Trends in old-age compression/expansion also have important implications for the debate on a potential limit to life expectancy and the maximum life span. Our observation of a strong trend towards mortality delay implies that there is no limit to life expectancy in the near future. If we were approaching a limit to lifespan, we would expect to see that either mortality delay (i.e. the increase in the modal age) had slowed down or the effect of mortality compression in old age had exceeded the effect of mortality delay. A limit to lifespan implies that the upper bound of the age-at-death distribution is fixed. As long as mortality delay continues without compression in old age, the upper bound of the age-at-death distribution will continue to move to older ages. If compression in old age does occur, the upper bound of the age distribution will increase less strongly than the modal age. Thus, the finding that from 1980 onwards deaths above the modal age have become increasingly concentrated within a narrow age interval does not imply that there is a limit to life expectancy, provided mortality delay continues to play a more important role than mortality compression at old ages. This result does, however, indicate that the maximum lifespan has not been increasing as fast as the modal age at dying. Compression in old age implies that the difference between the modal age and the upper bound has been decreasing. Our observation that compression of old-age mortality has recently been stable or even slightly declining among women in non-Eastern Europe-and that there have even been signs of mortality expansion in certain countries—indicates that the maximum lifespan could increase further as the modal age at death increases. But if the maximum lifespan continues to increase more slowly than the modal age, we may expect to find that there is a limit to the increase in the modal age in the long run, as compression in old age cannot continue infinitely (Cheung et al. 2005).

Conclusion and implications

The transition from a predominance of mortality compression to a predominance of mortality delay in determining changes in e0 could be dated among women at around 1950 in the USA, between 1955 and 1970 in Northern and Western Europe, around 1970–1975 in Southern Europe, and still later in Eastern Europe, with the transition among men in these European regions generally occurring about 10 years later. Among men in Lithuania, Latvia, and Belarus, delay has not yet overtaken compression because of the health crises they have experienced. It

Janssen and de Beer Genus (2019) 75:10 Page 18 of 23

is, however, likely that for them the transition will occur soon. Differences in the timing of the transition could be linked to the past health crisis in Eastern Europe, past differences in the pace of socioeconomic change and associated improvements in medical care, but also to differences in the timing and impact of the smoking epidemic.

Based on our analysis of the role of compression at different ages, we distinguished four phases in the transition from mortality compression to mortality delay: (1) predominance of compression due to strong mortality declines at young ages, (2) declining importance of mortality compression due to the decreasing impact of mortality declines at young ages, (3) mortality delay becomes predominant due to strong increases in the modal age at death and the counterbalancing effects of mortality compression/expansion at different ages, and (4) strong predominance of delay combined with stronger mortality declines at adult ages and declining mortality compression at old age.

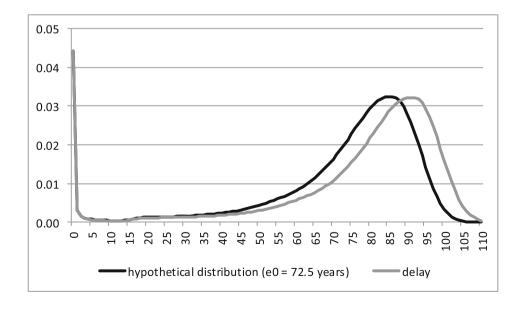
Our results indicate that we are not approaching a limit to life expectancy or a maximum lifespan. With mortality delay, premature mortality and old-age mortality are also shifting towards older ages.

Appendix 1

Effects of delay parameter M and compression parameters b_1 , b_2 , and b_3 of the CoDe model based on a hypothetical age-at-death distribution that resembles a life expectancy at birth (e0) of 72.5 years

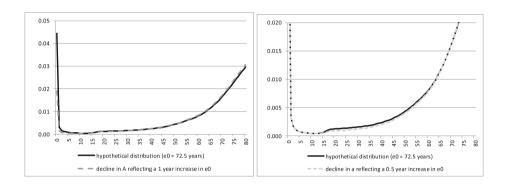
- Mortality delay

An increase in the modal age at death (=delay) from 85.2 to 90.86 years corresponded with a 5-year increase in e0 from 72.5 to 77.5 years.



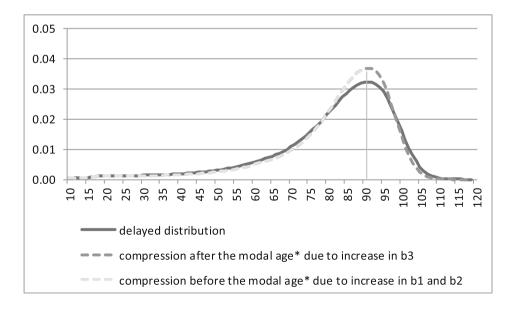
Janssen and de Beer Genus (2019) 75:10 Page 19 of 23

A decline in A (infant mortality) from 0.0035 to 0.0027 resulted in less lifetable deaths up until age 15, with more lifetable deaths around the modal age at death (not visible), which resulted in an increase in e0 of 1.0 years. A decline in a (teen mortality) from 0.0009 to 0.00063 resulted in less lifetable deaths from age 10 up until age 45, and slightly more lifetable deaths around the modal age at death (not visible), which resulted in an increase in e0 of 0.5 years.



An increase in b_1 from 0.088 to 0.0983 combined with an increase in b_2 from 0.113 to 0.13 resulted in less lifetable deaths up until age 79 and more lifetable deaths from age 79 up until the modal age at death (91) leading to a 1-year increase in e0.

An increase in b_3 from 0.125 to 0.145 resulted in more lifetable deaths between the modal age at death (91) and age 97 and less thereafter, which together led to a decline in e0 of 0.2 years.



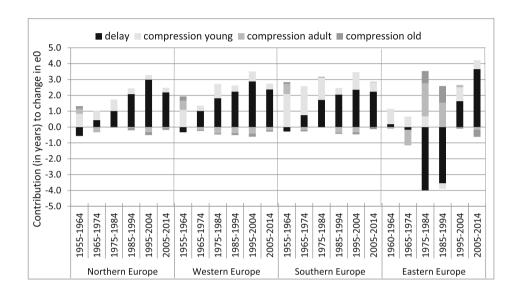
^{*} denoted by the vertical line

Janssen and de Beer *Genus* (2019) 75:10 Page 20 of 23

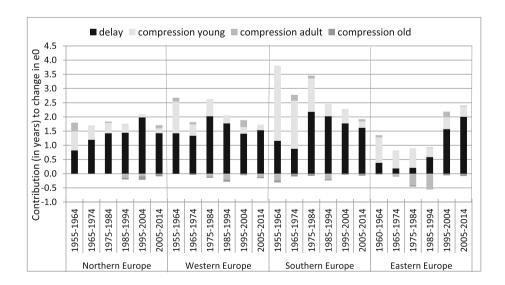
Appendix 2

Contributions (in years) of mortality delay (or declining *M* when negative) and mortality compression (or expansion when negative) at young, adult, and old ages to the changes in life expectancy at birth (e0), for the separate decades from 1955* to 2014*. * For West Germany from 1956 onwards. For Eastern Europe from 1960 onwards. *2010 for Bulgaria; 2012 for Italy; 2013 for UK, Lithuania, Latvia, and Ukraine

a) Males



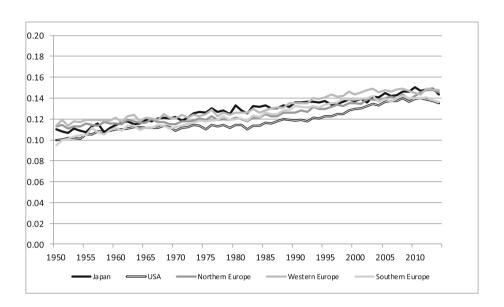
b) Females



Janssen and de Beer Genus (2019) 75:10 Page 21 of 23

Appendix 3

Past trends in compression above the modal age at death (b_3) , 1950–2014, women, by region (without Eastern Europe)



Abbreviations

CoDe mortality model: Compression and delay mortality model; e0: Life expectancy at birth; M: Modal age at death; nlm: Non-linear minimization; UK: United Kingdom; USA: United States of America; WLS: Weighted least squares

Acknowledgements

We thank Shady El Gewily (econometrics, University of Groningen) for his help in programming in R.

Funding

This work was supported by the Netherlands Organisation for Scientific Research (NWO) in relation to the research programme "Smoking, alcohol, and obesity, ingredients for improved and robust mortality projections", under grant no. 452–13-001. See www.futuremortality.com.

Availability of data and materials

For the analysis, data from the Human Mortality Database are used, which are available online at www.mortality.org. All calculations are done in R.

Authors' contributions

FJ designed the study, did the analyses, and wrote the manuscript. JdB aided in interpreting the results and reviewed the manuscript. Both authors read and approved the final manuscript.

Competing interests

The authors declare that they have no competing interests.

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Received: 4 May 2018 Accepted: 4 February 2019 Published online: 07 March 2019

References

Bergeron-Boucher, M.-P., Ebeling, M., & Canudas-Romo, V. (2015). Decomposing changes in life expectancy: compression versus shifting mortality. *Demogr Res*, 33(14), 391–424.

Bongaarts, J. (2005). Long range trends in adult mortality: models and projection methods. *Demography*, 42(1), 23–49. Börger, M., Genz, M., & Ruß, J. (2018). Extension, compression, and beyond: a unique classification system for mortality evolution patterns. *Demography*, 55(4), 1343–1361.

Janssen and de Beer Genus (2019) 75:10 Page 22 of 23

- Canudas-Romo, V. (2008). The modal age at death and the shifting mortality hypothesis. Demogr Res, 19(30), 1179-1204.
- Cheung, S. L. K., Robine, J.-M., & Caselli, G. (2008). The use of cohort and period data to explore changes in adult longevity in low mortality countries. *Genus*, *LXIV*(1–2), 101–129.
- Cheung, S. L. K., Robine, J.-M., Paccaud, F., & Marazzi, A. (2009). Dissecting the compression of mortality in Switzerland, 1876-2005. Demogr Res, 21(19), 569–598.
- Cheung, S. L. K., Robine, J.-M., Tu, E. J. C., & Caselli, G. (2005). Three dimensions of the survival curve: horizontalization, verticalization, and longevity extension. *Demography*, 42(2), 243–258.
- De Beer, J., Bardoutsos, A., & Janssen, F. (2017). Maximum human lifespan may increase to 125 years. *Nature, 546*(7660), 16–17. De Beer, J., & Janssen, F. (2016). A new parametric model to assess delay and compression of mortality. *Popul Health Metrics,* 14(46), 1–21.
- Edwards, R. D., & Tuljapurkar, S. (2005). Inequality in life spans and a new perspective on mortality convergence across industrialised countries. *Popul Dev Rev, 31*(4), 645–674.
- Engelman, M., Canudas-Romo, V., & Agree, E. M. (2010). The implications of increased survivorship for mortality variation in aging populations. *Popul Dev Rev, 36*(3), 511–539.
- Fries, J. F. (1980). Aging, natural death, and the compression of morbidity. N Engl J Med, 303(3), 130–135.
- Gillespie, D. O., Trotter, M. V., & Tuljapurkar, S. D. (2014). Divergence in age patterns of mortality change drives international divergence in lifespan inequality. *Demography*, 51(3), 1003–1017.
- Goldstein, J. R., & Cassidy, T. (2012). How slowing senescence translates into longer life expectancy. *Popul Stud*, 66(1), 29–37. Horiuchi, S., Ouellette, N., Cheung, S. L. K., & Robine, J.-M. (2013). Modal age at death: lifespan indicator in the era of longevity extension. *Vienna Yearb Popul Res*, 11, 37–69.
- Human Mortality Database (2017). University of California, Berkeley, and Max Planck Institute for Demographic Research. Rostock. Available at http://www.mortality.org (data downloaded on 12 June 2017).
- Janssen, F., & de Beer, J. (2016). Projecting future mortality in the Netherlands taking into account mortality delay and smoking. Joint Eurostat/UNECE work session on demographic projections, working paper 18.
- Janssen, F., Rousson, V., & Paccaud, F. (2015). The role of smoking in changes in the survival curve: an empirical study in 10 European countries. *Ann Epidemiol, 25*(4), 243–249.
- Janssen, F., & van Poppel, F. (2015). The adoption of smoking and its effect on the mortality gender gap in the Netherlands, a historical perspective. Biomed Res Int, 370274, 1-12.
- Kannisto, V. (1996). The advancing frontier of survival life tables for old age. Odense: Odense University Press.
- Kannisto, V. (2000). Measuring the compression of mortality. Demogr Res, 3(6), 1-24.
- Kannisto, V. (2001). Mode and dispersion of the length of life. *Popul, 13*(1), 159–171.
- Leon, D. A. (2011). Trends in European life expectancy: a salutary view. Int J Epidemiol, 40(2), 271-277.
- Leon, D. A., Shkolnikov, V. M., & McKee, M. (2009). Alcohol and Russian mortality: a continuing crisis. *Addiction, 104*(10), 1630–1636. Lexis, W. (1878). Sur la durée normale de la vie humaine et sur la théorie de la stabilité des rapports statistiques [on the normal human lifespan and on the theory of the stability of the statistical ratios]. *Ann Démographie Internationale, 2*(5), 447–460.
- Lopez, A. D., Collishaw, N. E., & Piha, T. (1994). A descriptive model of the cigarette epidemic in developed countries. *Tob Control*, 3(3), 242–247.
- Mackenbach, J. P. (2013). Convergence and divergence of life expectancy in Europe: a centennial view. Eur J Epidemiol, 28(3), 229–240.
- Mäkelä, P., Gmel, G., Grittner, U., Kuendig, H., Kuntsche, S., Bloomfield, K., & Room, R. (2006). Drinking patterns and their gender differences in Europe. *Alcohol, 41*(1), 8–18.
- McKee, M., & Shkolnikov, V. (2001). Understanding the toll of premature death among men in eastern Europe. *BMJ*, 323(7320), 1051–1055.
- Muszyńska, M., & Janssen, F. (2016). The concept of the Equivalent Length of Life for quantifying differences in age-at-death distributions across countries. *Genus*, 72(6), 1–14.
- Omran, A. R. (1998). The epidemiologic transition theory revisited thirty years later. World Health Stat Q, 52, 99–119.
- Ouellette, N., & Bourbeau, R. (2011). Changes in the age-at-death distribution in four low mortality countries: a nonparametric approach. *Demogr Res*, 25(19), 595–628.
- Robine, J.-M. (2001). Redefining the stages of the epidemiological transition by a study of the dispersion of life spans: the case of France. *Popul*, 13(1), 173–193.
- Robine, J.-M., & Caselli, G. (2005). An unprecedented increase in the number of centenarians. *Genus*, 61(1), 57–82.
- Rossi, I. A., Rousson, V., & Paccaud, F. (2013). The contribution of rectangularization to the secular increase of life expectancy: an empirical study. *Int J Epidemiol*, 42(1), 250–258.
- Rousson, V., & Paccaud, F. (2010). A set of indicators for decomposing the secular increase of life expectancy. *Popul Health Metrics*, 8(18), 1-9.
- Shkolnikov, V. M., Andreev, E. M., & Begun, A. Z. (2003). Gini coefficient as a life table function: computation from discrete data, decomposition of differences and empirical examples. *Demogr Res*, 8(11), 305–358.
- Shkolnikov, V. M., Andreev, E. M., Zhang, Z., Oeppen, J., & Vaupel, J. W. (2011). Losses of expected lifetime in the United States and other developed countries: methods and empirical analysis. *Demography*, 48(1), 211–239.
- Smits, J., & Monden, C. (2009). Length of life inequality around the globe. Soc Sci Med, 68(6), 1114–1123.
- Thatcher, R., Cheung, S. L. K., Horiuchi, S., & Robine, J.-M. (2010). The compression of deaths above the mode. *Demogr Res*, 22(17), 505–538.
- Trias-Llimós, S., Bijlsma, M., & Janssen, F. (2017). The role of birth cohorts in long-term trends in liver cirrhosis mortality across eight European countries. *Addiction*, 112(2), 250–258.
- Trias-Llimós, S., & Janssen, F. (2018). Alcohol and gender gaps in life expectancy in eight Central and Eastern European countries. Eur J Pub Health, 28(4), 687–692.
- United Nations (2016). Composition of macro geographical (continental) regions, geographical sub-regions, and selected economic and other groupings. http://unstats.un.org/unsd/methods/m49/m49regin.htm#europe
- Vallin, J., & Meslé, F. (2004). Convergences and divergences in mortality. A new approach to health transition. Demogr Res, S2(2), 11–44.

Janssen and de Beer Genus (2019) 75:10 Page 23 of 23

Van Poppel, F., & Janssen, F. (2016). The mortality gender gap in the Netherlands 1850–2000. In M. Dinges & A. Weigl (Eds.), Gender-specific life expectancy in Europe 1950–2010 (pp. 111–130). Medizin, Gesellschaft und Geschichte-Beihefte.

- Van Raalte, A. A., Kunst, A. E., Deboosere, P., Leinsalu, M., Lundberg, O., Martikainen, P., Strand, B. H., Artnik, B., Wojtyniak, B., & Mackenbach, J. P. (2011). More variation in lifespan in lower educated groups: evidence from 10 European countries. *Int J Epidemiol*, 40(6), 1703–1714.
- Vaupel, J. W. (2010). Biodemography of human ageing. Nature, 464(7288), 536-542.
- Vaupel, J. W., Zhang, Z., & van Raalte, A. A. (2011). Life expectancy and disparity: an international comparison of life table data. *BMJ Open, 1*, e000128.
- Wilmoth, J. R., & Horiuchi, S. (1999). Rectangularization revisited: variability of age at death within human populations. *Demography*, 36(4), 475–495.
- Yashin, A. I., Begun, A. S., Boiko, S. I., Ukraintseva, S. V., & Oeppen, J. (2001). The new trends in survival improvement require a revision of traditional gerontological concepts. *Exp Gerontol*, *37*(1), 157–167.
- Zhang, Z., & Vaupel, J. W. (2009). The age separating early deaths from late deaths. *Demogr Res, 20*(29), 721–730.

Submit your manuscript to a SpringerOpen journal and benefit from:

- ► Convenient online submission
- ► Rigorous peer review
- ► Open access: articles freely available online
- ► High visibility within the field
- ► Retaining the copyright to your article

Submit your next manuscript at ▶ springeropen.com