Lifespan variation is declining among cohorts

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Abstract

Lifespan variation is a fundamental dimension of inequality. However, little is known about lifespan variation for real cohorts – the majority of research has focused on the period perspective. We use the Human Mortality Database data to compare adult (ages 30+) lifespan variation for cohorts and periods. Under declining mortality, death rates at adult ages are lower in the cohort perspective. This compresses the lifespan distribution at young ages, decreasing the variation. However, increased old-age survival stretches the right tail of the distribution, increasing the variation. We contrast the trends in cohort and period lifespan variation and investigate the age patterns driving the trends. We extend the range of observations by forecasting the mortality of partially observed cohorts. We find that adult cohort lifespan variation is steadily declining, in contrast to the stagnation observed in period perspective.

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Introduction

Lifespan variation is a fundamental dimension of inequality – "the final inequality" as termed by Tuljapurkar (Tuljapurkar 2010). Uncertainty in this dimension can affect life course decisions such as investment in education and training, retirement planning, and the adoption of healthy behaviour. Individuals observe the mortality of their peer group, which develops over a cohort basis. Moreover, many of the determinants of mortality, such as smoking, are driven by cohort trends. Yet to date, most studies of lifespan variation have exclusively considered the period dimension. Understanding the uncertainty, or variation, in cohort lifespan, however, is important as only the cohort perspective can inform us about the uncertainty that real people face.

The relationship between cohort and period measures of lifespan variation is a priori not clear. With each calendar year survived, successive birth cohorts can expect lower death rates than those experienced by earlier cohorts at the same age. The farther ahead we look into time, the greater is the impact of declining mortality on the experienced mortality rates. For example, assuming a modest 1 percent annual mortality decline, a cohort born in 2000 will experience in year 2050 at age 50 a 40% lower mortality than the year 2000 period life table would suggest. By age 80 the mortality difference between the birth year period life table and the experienced mortality rate in 2080 would have grown to 65%. Because of this, we can expect the age-at-death distribution of the 2000 birth cohort to be stretched over the *x* axis compared to the 2000 period age-at-death distribution. This might lead to either greater or lower lifespan variation. On the one hand, lower death rates through early adulthood compress the left tail of the age-at-death distribution for birth cohorts, leading to a more 'normal' distribution and less variation. For example, prior studies suggest that it is mostly

differences in premature adult mortality that drive differences in lifespan variation between populations (van Raalte et al. 2011; Vaupel, Zhang and van Raalte 2011; Wilmoth and Horiuchi 1999). On the other hand, increased old-age survival stretches the right tail of the distribution, increasing the variation.

Several recent papers have analyzed lifespan variation in the period perspective (Edwards 2011; Edwards and Tuljapurkar 2005; Engelman, Canudas-Romo and Agree 2010; Shkolnikov, Andreev and Begun 2003; Shkolnikov et al. 2011a; Smits and Monden 2009; Vaupel et al. 2011; Wilmoth and Horiuchi 1999). Only three papers have also examined the cohort perspective: Wilmoth and Horiuchi (1999) found that the interquartile range of life table ages at death was slowly rising from the 1751-55 to the 1871-75 Swedish birth cohorts, before falling sharply up until 1901-05 cohorts, where their analysis ended. Hill (1993) found similar patterns over similar years for France, the USA and Canada using a wider variety of indices. Engelman et al. (2010) examined the lifespan variation of completed Swedish cohorts from 1850 to 1916. They found that the standard deviation at ages 50 and 70 was higher in each year for cohorts than it was for periods, while the standard deviation at age 10 was higher for birth cohorts 1850-1900 but lower for birth cohorts 1900-1916. In this study we use the Human Mortality Database data to compare adult lifespan variation for cohorts and periods. We extend the analysis of these earlier papers by examining the relationship between cohort and period lifespan variation for 18 countries in the Human Mortality Database with long time series. We forecast the mortality of partially completed cohorts in order to allow analysis up to late 20th century cohorts. We contrast the developments in cohort lifespan variation with that which has been observed on the period basis-and make further

investigations into the age patterns driving these trends. In doing so, we hope to gain a better understanding of how period and cohort mortality dynamics shape lifespan variation.

Background and hypothesis

Although in principle lifespan variation could be either higher or lower in the cohort perspective, we would expect cohorts to have higher lifespan variation when compared from the same starting age. This would be the case under a situation of declining mortality where the population experiences a Gompertz mortality hazard, i.e., $\mu_x = ae^{bx}$, where *x* is age and *a* and *b* are the two parameters typically known as the initial mortality level at age zero and the life table rate of aging.

Gompertz curves are typically fitted starting at age 30. With the same a values, when the cohort b is lower than period b, the cohort curve sits below the period curve (panel A: Figure 1) and vice versa—the closer the b values, the closer the mortality level at age 30 and the smaller the divergence with age. In contrast the same b values for periods and cohorts produces parallel lines; the cohort line sits below the period line when it has a lower a value (panel B: Figure 1). When comparing periods and cohorts from the same initial mortality rate at age 30, with continuous mortality progress over time, we would expect the period and cohort mortality to diverge with increasing age. A combination of a higher a and lower bvalue for cohorts than for periods is needed to produce the expected diverging scenario (panel C: Figure 1).

Under Gompertz mortality, Vaupel and Canudas Romo (1999) showed that e^{\dagger} , a measure of lifespan variation (see methods section), could be expressed as (eq.49),

$$e^{\dagger} = \frac{1}{b} \left(1 - a e_0 \right). \tag{1}$$

In low mortality populations, *a* values are very low and equation (1) can be approximated by $e^{\dagger} \approx \frac{1}{b}$. This approximation gets better over time with mortality improvement because *a* typically declines faster than e_0 rises. Thus with decreasing *b*, lifespan variation should increase. As a result, we would expect the lower *b* value in the cohort perspective to also translate into higher lifespan variation.

Data

We used data from the Human Mortality Database (HMD; Human Mortality Database 2011). The HMD has data on mortality by single-year age, calendar year, and birth year for 37 countries. In order to forecast cohort data, we excluded countries which did not have time series dating back to 1940 or earlier. We also excluded Iceland because of its small population size. This left us with 18 countries for which the time series were long and data quality arguably good (Table 1).

For all countries we used data starting from the first year provided by the HMD. We excluded all ages below 30 from the analysis because of three reasons: for the early years, particularly before 1860s, the quality of infant and child mortality data is weak (Shkolnikov et al. 2011a and the references therein). Second, our focus is on period versus cohort differences in the variation in *adult* lifespan, not in total lifespan which is heavily influenced by infant and child mortality. Third, age 30 is the typical starting age for fitting Gompertz mortality,

and our hypothesis of higher lifespan variation for cohorts is motivated by predictions of mortality decline under Gompertz hazards.

Methods

Completing cohort mortality

For most countries, the last available year was 2009 (see Table 1). In order to calculate cohort measures of lifespan variation, we forecasted mortality for the years 2010-2100. We used the Lee-Carter method (Lee and Carter 1992)¹ in which mortality at age x and time $t(m_{xt})$ is expressed as $\ln m_{xt} = a_x + b_x k_t + \varepsilon_{xt}$, where a_x is the average age effect; k_t is the time effect describing the level of mortality and modeled here as a random walk with drift; b_x describes how much mortality at a given age x changes when the indicator k_t for the overall mortality level changes; and ε_{xt} is the residual. Following Shkolnikov et al. (2011b) we chose the period 1960-2009 as the basis of forecasting since this is the period identified by Vallin and Meslé (2009) as the last segment of period longevity progress. Since we excluded ages below 30 from the analysis, the estimation of the Lee-Carter model was also based on ages 30 and above. Because of instability in observed mortality rates at the oldest ages we collapsed ages 100 and above into the open-ended age group. For each country we then used the data observed over the period 1960-2009 to estimate the country-specific parameters of the Lee-Carter model and use the estimated parameters (fixed a_x and b_x but time-varying k_t) to forecast mortality rates for the years 2010-2100.

¹ To implement the Lee-Carter forecasts we used the function *lca.forecast* from R package *demography* by Rob J. Hyndman, available at http://robjhyndman.com/software/ demography.

After completing the mortality forecasts for ages 30-100 for the years 2010-2100 we had three pieces of data for each country: observed mortality rates by age and period up to the year 2009; observed mortality rates by age and year of birth up to the year 2009; and forecasted mortality rates for the years 2010-2100.² To calculate period measures of lifespan variation we used only observed data. For cohort measures we used both observed and forecasted data. For cohorts born in 1909 or earlier we have observed data up to age 100. We define these cohorts as cohorts for whom lifespan variation is *observed* since only a small proportion of the remaining deaths, those occurring at ages above 100, are based on forecasts. We define cohorts born from 1910 to 1929 as cohorts for whom lifespan variation is *forecasted*. For these cohorts mortality at ages above 80 is based on forecasts. For cohorts born in 1930 or later our estimates of lifespan variation are *speculative*.

Measuring lifespan variation

We measure lifespan variation using two widely used indices: life disparity (e_{30}^{\dagger}) and the interquartile range (IQR₃₀), both conditional upon survival to age 30. Life disparity is used because it is both intuitive (the average years of life lost due to death in the population), and is not overly sensitive to mortality in the early part of the age-at-death distribution (Shkolnikov et al. 2011a; van Raalte and Caswell 2012). The interquartile range has the advantage that it is

 $^{^{2}}$ Our interest is in measuring lifespan variation by year of birth. Thus it makes sense to ask whether the forecasts should be indexed by age and period, or age and year of birth. We compared two alternative methods for forecasting mortality, one in which the forecasts are based (as is conventional) on information ordered by age and calendar year, and one in which the forecasts are based on data where the values m(x,t) refer to age x for cohort t-x. The forecasted mortality rates differed only marginally. These preliminary results are based on the latter option.

not affected by our inability to forecast the precise ages at death above age 100, since deaths beyond this age lie above the 3^{rd} quartile.

Working with a life table radix of 1, life disparity is calculated as $e_{30}^{\dagger} = \sum_{i=30}^{\omega} d_x \overline{e}_x$, where

 d_x is the number of deaths in the age interval x to x+1, and \overline{e}_x is the average remaining life expectancy over the interval, i.e. $\overline{e}_x = 0.5(e_x + e_{x+1})$. The interquartile range is calculated by $IQR_{30} = \hat{x}_3 - \hat{x}_1$, where \hat{x}_1 and \hat{x}_3 are the interpolated first and third age quartiles, at which 25 and 75 percent of the total life table deaths have occurred. Both measures are calculated from starting age 30 to final age $\omega = 100+$.

Decomposing lifespan variation

A step-wise decomposition algorithm was used to decompose both indices of lifespan variation (Andreev, Shkolnikov and Begun 2002) by modifying a VBA program developed by Shkolnikov and Andreev (2010).

Since we started our examinations at age 30, there was some question about whether it was more appropriate to compare period and cohort populations in the same year (i.e. comparing the 1900 period and 1900 cohorts), as was done by Engelman et al. (2010), or to compare the populations when they had the same mortality at age 30 (i.e. comparing the 1900 period with the 1870 birth cohort). We opted for the latter because we were interested in comparing the two perspectives from the age at which mortality diverged.

Results

Trends in life expectancy and lifespan variation

For the 18 countries, cohort e_{30} levels, shifted by 30 years so as to be compared with the period having matching mortality at age 30, were always higher than period levels, with differences widening over time alongside increases in longevity (Figure 2).

Period and shifted cohort lifespan variation levels were similar and falling up until 1940 (Figure 3). Trends began to deviate in the two dimensions over the middle observation window, which compares the 1940-1970 period lifespan variation to the forecasted partially observed 1910-1940 birth cohorts. In most countries, period lifespan variation initially experienced a sharp drop, followed by stagnating levels from 1950 onwards. In the cohort dimension, however, lifespan variation declined more smoothly, although a hump was observed in some countries around 1950, especially among men. The period stagnation in lifespan variation continued through most of the late twentieth century, although by the end of the observation window (2000) some countries started to mark declines. Continued declines in cohort lifespan variation were forecasted up until the 2000 birth cohorts. Measuring lifespan variation by the IQR₃₀ (Figure 3, top panels) and e_{30}^{\dagger} (Figure 3, bottom panels) produced similar overall results, although the lifespan variation hump occurring around 1950 in the cohort dimension (referring to birth cohorts from the early 1920s) was more evident for the IQR₃₀.

Within this cloud of country data points, we examined trends in four countries from different parts of Europe and North America: Sweden, France, USA and the Netherlands. The

dominant period pattern among women was high and slowly declining lifespan variation until the 1930s, rapid declines between 1930 and 1950, and stagnation since 1950 (Figure 4). French women experienced less stagnation than the other countries, while data was not available for American women before 1937 to confirm the early trends found in other countries. In all countries, lifespan variation declined steadily for cohorts, apart from the 1950 hump in the Netherlands. This resulted in higher lifespan variation in the lagged cohort perspective over the 1940-1970 period, with a cross-over forecasted to occur in the 1970s (1960s in France). The male pattern was similar to the female pattern (Figure 5). However, the drop in period lifespan variation from an IQR_{30} of around 23 years to around 15 years was less steep than it was for females, whose level dropped to around 13 years. The cohort hump in lifespan variation around 1950 that was observed among Dutch women was also found in Sweden, France and the Netherlands among men, while the observation window was not long enough in the USA to determine whether it also existed there. French trends showed a greater impact from the world wars and the 1918 flu epidemic than other countries, in both the period and cohort perspectives. Overall, the patterns were similar when measured by e_{30}^{\dagger} and are shown in the appendix.

We hypothesized that lifespan variation would be higher for cohorts than for periods, based on expectations from Gompertz mortality trajectories. This result held true for most years and countries (76 % of the cases), but not all. In reality many mortality curves from our dataset are not well fitted by a Gompertz curve, especially those related to completed cohorts and historical period data, as seen in Figure 6, which fits a Gompertz curve to the male Swedish 1900 cohort and 1930 period. The difference between period and cohort mortality rates first widen, as we had predicted, but then narrow over middle adult ages before widening again at later ages. Overall this complicated pattern led to a narrowing rather than widening in the gap between fitted log mortality lines. This was an instance where lifespan variation was higher in the period, rather than the cohort dimension.

In some countries, period mortality around the turn of the 20th century led to higher lifespan variation than that experienced by cohorts (lagged by 30 years). Meanwhile apart from the USA (females), Denmark (females), Scotland (females) and Portugal (males), all countries had higher period lifespan variation in 2000 than was forecasted for the 1970 birth cohorts. Interestingly, the female exceptions all came from populations that are currently experiencing higher than average losses of life attributable to smoking (Preston, Glei and Wilmoth 2010). Future work should investigate whether smoking is linked with high forecasted cohort lifespan variation.

Decomposition of differences in lifespan variation

To further understand the changing period and cohort dynamics, we took a closer look at an earlier instance when the 1920 birth cohort had similar, but slightly higher lifespan variation than the 1950 period, and a later instance when the 1970 birth cohort had lower lifespan variation than the 2000 period. The age at death distributions are contrasted in Figure 5: in the earlier instance (panel 7a), the cohort distribution is similarly shaped to the period distribution but shifted to later years. In the later instance (panel 7b), the left tail is clearly more compressed for the cohort distribution and a greater number of deaths are occurring at the modal age at death. Decomposing these differences revealed that in the earlier instance,

lifespan variation was similar between period and cohort because mortality was lower at all ages for the 1920 cohort than the 1950 period (Figures 7c and 7d for IQR₃₀, Figures 7e and 7f for e_{30}^{\dagger}). This had the effect of compressing the lifespan distribution over younger ages and expanding this distribution over older ages. The two phenomena mostly balanced each other out, but the old age expansion was slightly larger than the younger age compression. By 2000 (1970 cohort), however, differences in period vs. cohort early adult mortality are forecasted to contribute larger differences lifespan variation levels than differences in period vs. cohort old age mortality. This would lead to lower lifespan variation forecasted for birth cohorts. Small differences were found between the two lifespan variation measures due to the different age sensitivity profiles of the measures, but broadly speaking they told the same story. Decompositions for other countries and neighboring years were similar (results not shown).

Comparing periods and cohorts at similar life expectancies

The findings from the previous sections document that cohorts have been experiencing declining lifespan variation, in contrast to the stagnation observed in the period dimension since 1950. The exception to the cohort pattern were the few years around 1950, corresponding to the birth cohorts of around 1920. Since this small stagnation in the cohort perspective occurred for the most part just prior to the onset of stagnation in the period perspective, we next investigated whether these two phenomena might be related. In Figure 8 we compared levels of IQR₃₀ at similar e_{30} levels for periods and cohorts. In this perspective the pattern of decline in lifespan variation was similar for periods and cohorts: declining IQR₃₀ from e_{30} levels of 30-45 years, followed by stagnation. It then appeared that IQR₃₀

began declining again in the cohort dimension from e_{30} levels of 50 (women) and 45 (men), a period mortality level which has not yet been reached in most countries. Although the patterns were similar, at shared e_{30} levels, the IQR₃₀ was lower in the period dimension, particularly for e_{30} levels above 40 years. Decomposition analysis showed that this was because at shared e_{30} levels, although early adult mortality was lower in the period perspective and older adult mortality higher, on balance the compression in early adult mortality more than offset the expansion in older adult mortality to result in an overall compression of mortality (results not shown).

Discussion

Our main findings from the analysis of long-term mortality patterns in 18 HMD countries were that: (a) Adult lifespan variation was not always higher in the cohort perspective, as we had predicted, and is in fact forecasted to be lower than comparable period lifespan variation levels by the late twentieth century birth cohorts for most countries; (b) cohort adult lifespan variation is declining in an almost linear manner, unlike period lifespan variation, which has been stagnating since the 1960s; and (c) these differences might be related to the mortality level, with periods still undergoing a transformation phase in their age-at-death distributions, and not having yet reached the high life expectancies projected for cohorts.

In recent years much research has documented the empirically observed stagnation in adult period lifespan variation since the 1960s in industrialized countries (Edwards and Tuljapurkar 2005; Engelman et al. 2010; Robine 2001; Shkolnikov et al. 2011a; Wilmoth and Horiuchi 1999). This break in trends was seen as so sudden and widespread among industrialized countries as to vindicate adding the "Age of the Conquest of the Extent of Life" as proposed by Olshanksy and Ault (1986) to Omran's (1971) classic epidemiologic transition theory (Robine 2001).

Not only are trends in lifespan variation important for understanding changing age and disease profiles, but also factor more generally into theories of aging. Much of this work was motivated by Fries (1980) who suggested that given a looming limit to the human lifespan, further mortality decline would reduce lifespan variation rather than increasing the ages of death of the oldest individuals, a process which would continue until only deaths from natural causes remained. Although a strict limit to lifespan has been largely discredited (Oeppen and Vaupel 2002), recent research continues to examine changes in lifespan variation above the modal age at death (Canudas-Romo 2008; Cheung, Robine and Caselli 2008; Kannisto 2000; Kannisto 2001; Ouellette and Bourbeau 2011; Thatcher et al. 2010). This perspective is largely used to test whether mortality is being further compressed at these ages, which might signal a looming limit to the human lifespan in the absence of corresponding increases in the modal age at death, or alternatively, to establish whether the entire older age distribution of death is shifting to the right, known as the 'shifting mortality' hypothesis (Bongaarts 2005; Canudas-Romo 2008; Kannisto 1996). A third possibility is that the survival of frail individuals in early life is leading to a postponement of selection in later life, which might lead to an expansion of old age mortality (Bonneux, Barendregt and Van der Maas 1998; Engelman et al. 2010).

Yet most of these studies, whether of the entire age at death distribution, the adult age at death distribution, or the distribution of ages at death above the mode, have been conducted on the basis of period mortality. This paper has uncovered an up until now overlooked phenomenon–cohort lifespan variation has been declining steadily since the 1900 birth cohorts, in contrast to the recent stagnation observed in the period perspective. The idea that the age-at-death distribution is approaching a fixed shape that is shifting along the age axis is not supported in the cohort perspective, and might simply be an artifact of using period data.

Goldstein and Wachter (2006) identified a 50-year lag between period and cohort life expectancies at birth in Sweden in 2000, a lag which Shkolnikov et al. (2011b) have forecasted to grow. Comparing period and cohort lifespan variation at similar underlying e_{30} levels led us to theorize that the different conclusions reached in the period and cohort perspective may be related to the underlying mortality level. Although in the time perspective, the decline in cohort lifespan variation appears more or less linear, we showed here that if examined at different levels of life expectancy, a transition phase around an e_{30} of 45-50 where lifespan variation did not decrease appeared, as early adult mortality compression balanced the old age expansion. To date only a few countries have exceeded this e_{30} range in the period perspective. It could be that period mortality is also currently undergoing a temporary transition phase where early adult mortality compression and old adult mortality are in balance, and in future reductions in lifespan variation will resume.

Limitations

The largest source of uncertainty in this analysis is the heavy use of forecasted mortality in completing the cohort age at death schedule. In particular, Lee-Carter forecasts may underestimate mortality declines. For example, in the 20th century the annual increases in life expectancy were markedly faster than 0.14 years per annum Lee-Carter forecast for early 21st century (Lee and Miller 2001; Oeppen and Vaupel 2002). This could in part explain the lower

cohort lifespan variation forecasted for later years, if the differences between forecasted and actual mortality levels were greater for older ages than over early ages. Nevertheless for the most recent observed cohorts, the decline in lifespan variation is clear. In the future, we will investigate whether the usage of a less conservative forecasting method might lead to smaller differences between period and cohort lifespan variation in later years.

We will also check whether our results are robust to different starting ages. Generally we are interested in the complete range of adult mortality, but we recognize that the choice of starting age in the 10-40 age range is subjective (Engelman et al. 2010; Robine 2001). Given the small number of deaths over these ages, we do not expect our measures of lifespan variation to be particularly sensitive to different starting ages within this range.

Finally, there could be some question about our choice of index of lifespan variation. Indices may reach different conclusions depending on the age sensitivity profile of the index, but generally, the most often used indices correlate well with one another (Shkolnikov et al. 2003; van Raalte and Caswell 2012; Wilmoth and Horiuchi 1999). Our results were similar using either the IQR₃₀ or e_{30}^{\dagger} . Both of these indices tend to be sensitive to mortality at older ages of the age-at-death distribution than some other indices such as the Theil's index or the variance in age at death. While the usage of one of these other indices might influence the timing of crossovers between period and cohort lifespan variation levels, we would expect our overall conclusions to remain robust. Moreover, disagreements between indices tend to largest when the direction of mortality change is not uniform across age, for instance when mortality is decreasing at young ages but increasing across adult ages, as was the case for many countries of the former Soviet Union during the late 20th Century (Shkolnikov et al. 2003; van Raalte and Caswell 2012). This was not the case in any countries used in our analysis.

Conclusion

We have shown that adult lifespan variation was not always higher in the cohort perspective, and is forecasted to be lower than period lifespan variation for most of the 18 countries of the Human Mortality Database with long data series. In contrast to period lifespan variation, which has been stagnant since the mid-twentieth century, cohort lifespan variation is declining smoothly over time and is forecasted to do so until at least the 2000 birth cohorts. Thus while 'shifting mortality' or the 'Age of the Conquest of the Extent of Life' might characterize current changes to the period mortality schedule, they are not reflective of what is occurring to actual cohorts. Uncertainty in the timing of death is declining.

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Tables and Figures

Countries	Years available
Australia	1921-2000
Belgium	1841-2000
Canada	1921-2000
Switzerland	1876-2000
Denmark	1835-2000
Spain	1908-2000
Finland	1878-2000
France (TNP)	1816-2000
Northern Ireland	1922-2000
Scotland	1855-2000
England & Wales (TNP)	1841-2000
Italy	1872-2000
Netherlands	1850-2000
Norway	1846-2000
New Zealand Non-Maori	1901-2000
Portugal	1940-2000
Sweden	1751-2000
USA	1933-2000

Table 1: Countries from the Human Mortality Database used in our analysis. "TNP" refers to total national population as opposed to the civilian population. The years available corresponded to the period data, and cohorts were compared lagged by 30 years.



Figure 1: Gompertz mortality curves under different assumptions about *a* and *b*.



Figure 2: Differences in period and cohort remaining life expectancy at age 30. In this and in subsequent figures, the birth cohort is shifted by 30 years to match mortality at age 30 (i.e. the birth cohort of 1900 is plotted at the year 1930).



Figure 3: Lifespan variation at age 30 contrasted in the period and cohort dimensions, measured by the interquartile range (top panels) and life disparity (bottom panels). The cohorts have been shifted by 30 years, i.e. the data point for the year 2000 in the cohort dimension refers to the 1970 birth cohort that became age 30 in 2000. The fully observed cohort points lie to the left of the dotted line, the forecasted points between the dotted and dashed lines, and the speculative cohort data points, to the right of the dashed lines.



Figure 4: A comparison of period and cohort lifespan variation levels for various countries, females



Figure 5: A comparison of period and cohort lifespan variation levels for various countries, males

Gompertz mortality fitted over males, ages 30:90



Figure 6: Data points from Swedish males fitted by a Gompertz mortality trajectory using maximum likelihood methods. Data was fitted over ages 30-90.



Figure 7: Decomposition of differences between period and cohort lifespan variation. Panels A-B depict the age-at-death distributions of the periods and cohorts examined, while panels C-F present the decomposition results. Ages with negative contributions are ages that contribute to lower cohort lifespan variation, while ages with positive contributions contribute to higher cohort lifespan variation.



Figure 8: Cohort and period lifespan variation, at different levels of life expectancy – all country points in dataset (Table 1).

Appendix figures



Appendix figure 1: Trends in lifespan variation measured by the e_{30}^{\dagger} index, females



Appendix figure 2: Trends in lifespan variation measured by the e_{30}^{\dagger} index, males