Mortality postponement and compression at older-ages in human cohorts

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1. Abstract

A key but unresolved issue in the study of human mortality at older ages is whether mortality is being compressed (which implies that we may be approaching a maximum limit to the length of life) or postponed (which would imply that we are not). We analyze historical and current population mortality data between ages 50 and 100 by birth cohort in 19 industrialized countries between ages 50 and 100, using a Bayesian technique to surmount cohort censoring caused by survival, to show that while the dominant historical pattern has been one of mortality compression, there have been occasional episodes of mortality postponement. The pattern of postponement and compression across different birth cohorts explain why longevity records have been slow to increase in recent years: we find that cohorts born between around 1900 and 1950 are experiencing historically unprecedented mortality postponement, but are still too young to break longevity records. As these cohorts attain advanced ages in coming decades, longevity records may therefore increase significantly. Our results confirm prior work suggesting that if there is a maximum limit to the human lifespan, we are not yet approaching it.

2. Introduction

Whether or not there is a limit to the human lifespan has been a subject of debate for millennia. Measured over very long time periods, there have been substantial increases in estimates of the maximum possible lifespan. The Hebrews of the late Bronze Age famously regarded 80 years as the maximum length of a human life. (Psalm 90:10; <500BC); around 1,000 years later, the ancient Romans set their official estimate of the maximum, the so-called *saeculum naturale*, at 100 or 110 years (Horace, 17BC, Censorinus, 248). Modern longevity records are higher still: the current human longevity record is 122), but has remained unchanged since 1997.

Dong et al (2016) use the slow change in longevity records in recent years to argue that the human lifespan has reached an absolute limit, a finding supported by Olshansky (2016). Kirkwood and Austad (2000) use biological hypotheses to reach the same conclusion. Oeppen and Vaupel (2002), Vaupel (2010) and Rootzen and Zholud (2017), on the other hand, interpret recent improvements in old-age mortality and the pattern of deaths at older ages to assert the opposite.

An important recent study in this area is Zuo et al (2018). They use period data to show that the gap between the percentiles of the distribution of age at death of those older than 65 remained roughly constant between 1960 and 2010 in 20 countries. They conclude that period effects therefore appear to be driving the improvement in longevity at older ages and that lifespan does not appear to be approaching an upper limit.

In this paper, like Zuo et al (2018), we analyze mortality of older individuals in richer countries using the Human Mortality Database (HMD, 2021). However, we analyze mortality by birth cohort, rather than by period. Using cohort data follows a fixed set of individuals over time, and is therefore most suited to clarifying the biological mechanisms underlying mortality. In particular, the use of cohort data may avoid the conflation in period data of changes in mortality rates over time and/or age with changes across cohorts. Cohort analysis is made difficult by cohort censoring due to survival, which we surmount using a Bayesian estimation approach (McCarthy, 2020; McCarthy and Wang, 2019, 2021) that improves the precision of estimates for non-extinct cohorts.

We fit the Gompertz law, which posits that mortality rates in humans increase roughly exponentially with age after around age 50 (Gompertz, 1825), to each individual cohort between the ages of 50 and 100. As we will demonstrate, the Gompertz law fits cohort mortality data extremely well in this age range, especially in recently extinct cohorts. A key parameter in the Gompertz law is the exponential rate of increase in the observed mortality rates with age, called the Rate of Demographic Aging (RDA). Because the RDA determines the distribution of age at death, changes in the RDA across cohorts are a key measure of whether mortality is being postponed or compressed. If lifespans are approaching an absolute limit, improvements in mortality at younger old ages will be associated with increases in the RDA and the distribution of age at death will be increasingly compressed at advanced ages below the limit. On the other hand if the RDA is constant, improvements in mortality at younger ages will simply shift the distribution of ages at death to the right, suggesting that old-age mortality is being postponed and that a limit to the human lifespan, if it exists, is still far away.

Despite the good fit of the Gompertz law between ages 50 and 100, there are strong theoretical reasons to expect the RDA to fall at extreme old ages (Vaupel et al, 1979) and such a fall has been observed empirically in the mortality of super-centenarians. In fact, as more high-quality data becomes available, the evidence in support of a levelling-off of the risk of dying has increased. Rootzen and Zholud (2017) found that at extreme ages, mortality probabilities appear to be independent of age and sex, and are around ½; Barbi et al (2018) suggest that the mortality hazard rate among super-centenarians in Italy appears to level off at around 2/3 (and so annual mortality probabilities are around ½), a finding confirmed in other countries by Alvarez et al (2021). We therefore hypothesize the existence of a Gompertzian Maximum Age (GMA) – the age at which the Gompertz law would predict the mortality hazard rate to be 2/3 – and estimate confidence intervals for this GMA and the age at death of the longest-lived person in each cohort. This approach allows us to disaggregate changes in life expectancy at 50 in historical and current cohorts between postponement and compression explicitly using an analytical method. We confirm the finding of Zuo at al (2018) that longevity does not appear to be approaching an upper limit. In contrast, however, we show that old-age mortality patterns can be well explained by cohort effects, rather than period effects. These cohort patterns show why longevity records have not changed in recent decades despite the well-documented improvements in mortality at older ages across much of the industrialized world in recent years.

3. Materials and Methods

Although more complex approaches are possible, we define the base mortality of individuals aged x and born in year c to be $\mu_{x,c}$ and use the Gompertz law to write:

$$\log(\mu_{x,c}) = \lambda_{50,c} + \delta_c (x - 50), \qquad 50 \le x \le 100 \tag{1}$$

where δ_c is the RDA of cohort c and $\lambda_{50,c} \equiv \log(\mu_{50,c})$. This approach makes the assumption that each cohort is endowed with δ_c and $\lambda_{50,c}$ at age 50, and that these remain fixed thereafter. These fixed cohort parameters can be regarded as proxies for causes of death (such as heart disease, stroke, cancer and neurological disorders) that are the consequence of lifestyle and other factors operating over very

long time scales. Other than Covid-19, these have become the predominant cause of death among the elderly.

To account for shorter-latency causes of death, we allow a cohort's observed mortality to fluctuate around base mortality due to calendar year effects (e.g. epidemics such as Covid-19 and the 1918 Spanish flu, climatic fluctuations, wars and famines). The error term also captures model misfit (e.g. the gradual fall in the RDA at extreme old age), data errors, and random variation. We therefore write the observed central rate of death at age x of an individual born in year c, defined as $m_{x,c}$, as:

$$\log(m_{x,c}) = \log(\mu_{x,c}) + \varepsilon_{x,c} \,. \qquad (2)$$

We estimated equation (2) using population mortality data, organized by birth year, for males and females between the ages of 50 and 100 for a set of 19 rich industrialized countries from the Human Mortality Database, described in Table 1. (Human Mortality Database, 2021)

TABLE 1: Description of data

	Data period		Extinct cohorts†		Cohorts used to estimate prior distribution			
					Prior A (base case)		Prior B	
Country	Start year	End year	First year of birth	Final year of birth	First year of birth	Final year of birth	First year of birth	Final year of birth
Australia	1921	2017	1871	1917	1871	1927	1871	1927
Austria	1947	2018	1897	1918	1897	1928	1897	1928
Belgium*	1841	2017	1791	1917	1791	1927	1869	1927
Canada	1921	2017	1871	1917	1871	1927	1871	1927
Denmark	1835	2019	1785	1919	1785	1929	1869	1929
Finland	1878	2019	1828	1919	1828	1929	1869	1929
France	1816	2017	1766	1917	1766	1927	1869	1927
Ireland	1950	2016	1900	1916	1900	1926	1900	1926
Italy	1872	2017	1822	1917	1822	1927	1869	1927
Japan	1947	2018	1897	1918	1897	1928	1897	1928
Netherlands	1850	2018	1800	1918	1800	1928	1869	1928
New Zealand	1948	2012	1898	1912	1898	1922	1898	1922
Norway	1846	2019	1796	1919	1796	1929	1869	1929
Portugal	1940	2019	1890	1919	1890	1929	1890	1929
Spain	1908	2017	1858	1917	1858	1927	1869	1927
Sweden	1751	2018	1709	1918	1709	1928	1869	1928
Switzerland	1876	2017	1826	1917	1826	1927	1869	1927
UK	1922	2017	1872	1917	1872	1927	1872	1927
USA	1933	2018	1883	1918	1883	1928	1883	1928

NOTES: Population data from the Human Mortality Database was used for each country. Separate estimates were prepared for males and females in each country. † We regards cohorts as extinct when they reach the age of 100. * Belgian data is incomplete over the war years. Each country's data is subject to inaccuracies and approximations, especially in earlier years[45]. Prior C uses the same data as Prior B but estimates a single model for all countries (a separate model is used for males and females).

3.1 Estimation technique

Details are provided in appendix M1. Combining (1) and (2), the central rate of death at age $50 \le x \le 100$ of an individual who was born in year *c*, written $m_{x,c}$, as:

$$\log(m_{x,c}) = \lambda_{50,c} + \delta_c (x-50) + \varepsilon_{x,c} \,. \qquad (3)$$

We use changes in the estimates of $\lambda_{50,c}$ and δ_c measured off extinct or nearly-extinct cohorts (where estimation errors are small) to formulate a Bayesian prior for how these parameters will change over cohorts that are currently censored by survival, and use Bayes' Theorem to calculate a joint posterior distribution of the parameters for both extinct and censored cohorts conditional on the data and the chosen prior. We then use the Metropolis-Hastings (MH) algorithm to draw a sample from this posterior distribution, denoted $\{\tilde{\boldsymbol{\lambda}}_{\mathbf{50}}, \tilde{\boldsymbol{\delta}}\}$, with mode $\hat{\boldsymbol{\lambda}}_{\mathbf{50}}$ and $\hat{\boldsymbol{\delta}}$.

We then define the Gompertzian Maximum Age of cohort c (GMA, denoted Λ_c) as that age at which the base mortality hazard in equation (1) first reaches 2/3 (so mortality hazards have likely plateaued and annual death probabilities are around ½):

$$\hat{\Lambda}_c = \frac{\log(2/3) - \hat{\lambda}_{50,c}}{\hat{\delta}_c} + 50 \qquad (4)$$

3.2 Division between compression and postponement

Details are provided in appendix M2. We used our MH sample $\{\tilde{\boldsymbol{\lambda}}_{\mathbf{50}}, \tilde{\boldsymbol{\delta}}\}$ to quantify the changes in remaining cohort base life expectancy at age 50 between cohorts born ten years apart due to compression and postponement, as well as 95% confidence intervals. As shown in Figure 1, the full change in $\lambda_{50,c}$ and any consequent change in δ_c needed to keep Λ_c fixed were ascribed to compression, and any further changes in δ_c to postponement.

FIGURE 1: Dividing changes in mortality rates between compression and postponement

3.3 95% confidence intervals for the GMA and the maximum length of life

Details are provided in appendix M3. We then calculated 95% confidence intervals for Λ_c directly using our sample $\{\tilde{\boldsymbol{\lambda}}_{\mathbf{50}}, \tilde{\boldsymbol{\delta}}\}$ and (4), and confidence intervals for M_c, the age at death of the longest-lived person in each cohort conditional on at least one person in each cohort reaching age Λ_c, under the assumption that the mortality hazard at ages older than Λ_c (so after the plateau) are constant and equal to two thirds. M_c will increase as cohort size increases, Λ_c only changes with underlying mortality rates.

4. Results

Despite its simplicity, the Gompertz law accounts for the vast majority of the variation in observed mortality rates between ages 50 and 100 across the extinct cohorts in our data (here we regard cohorts as extinct when they have reached the age of 100). Figure 2 shows the extinct cohorts with the highest and lowest R-squared values. Using $\hat{\boldsymbol{\lambda}}_{\mathbf{50}}$ and $\hat{\boldsymbol{\delta}}$ as point estimates, the modal value of R-squared of (2) for extinct cohorts is around 0.9925 (0.9975 for the most recent extinct cohorts, born in the 1910's). The residual mean squared error (RMSE) of (2), which represents the average percentage difference between actual mortality rates and estimated base mortality rates due to all causes declines from

around 20% of mortality rates for the very earliest extinct cohorts to around 7% of mortality rates for the latest ones, born in the 1910's. Figure 3 shows the decline in R-squared over extinct cohorts; while Figure 4 shows the decline in RMSE.

FIGURE 2: Extinct cohorts with highest (left) and lowest (right) R-squared in our dataset

FIGURE 3: Box-and-whisker plot of the distribution R-squared across extinct cohorts, by decade of birth

FIGURE 4: Box-and-whisker plot of the distribution of the RMSE across extinct cohorts, by decade of birth

The main source of differences driving the results in Figures 3 and 4 are calendar year effects representing short-latency causes of death. Examples are the 1918 influenza epidemic and the COVID-19 epidemic, which caused the mortality of affected cohorts to rise at the age they were when the epidemics occurred. Other examples might be climatic fluctuations, wars or famines. In general, calendar year effects are more significant for earlier cohorts of our sample, but have fallen in magnitude following World War II, as a result of greater prosperity, technological advancements in living standards and healthcare, and increased political and economic stability in the countries we examine (Carson et al, 2006; Astrom et al, 2013), although Covid-19 represents a significant exception. A small increase in the RMSE for cohorts born around 1850 is likely attributable to the Spanish Flu epidemic of 1918 and the First World War, and an increase for cohorts born around 1890 to the Second World War.

A second source of error is model misfit, as the Gompertz law may not fully capture the underlying dynamics of mortality. As discussed, the RDA may fall at extreme ages, senescent aging in females may be delayed due to childbirth, and calendar-year effects may have significant autocorrelation (a factor we ignore in our estimation procedure).

Random variation is especially important at older ages where relatively few cohort members are still alive, which we account for by ascribing less credibility to ages at which fewer deaths occur (Fay and Heriot, 1979).

A final source of error is data errors. With some caveats, data for recent years are likely to be accurate and substantially complete in all the countries we examine. However, in earlier years (before World War II in particular), data may be smoothed or imputed from periodic censuses or grouped data, or may simply be inaccurate. Readers can consult the data appendices for each country for more information about data errors when interpreting our results (see HMD, 2021).

Because Sweden has the longest data series in our sample, and exhibits patterns that are widely similar to other countries, we focus on results from Sweden before turning to other countries. Figure 5 shows log observed mortality hazard rates by age for Swedish males for cohorts born ten years apart from 1880 to 1950 (dots), along with our estimates of log base mortality (lines, calculated using $\hat{\lambda}_{50,c}$ and $\hat{\delta}_c$). As shown in the left panel, $\hat{\lambda}_{50,c}$ fell slightly for cohorts born between 1880 and 1910, but because $\hat{\delta}_c$ rose to compensate, there was little change in mortality rates at very old ages, consistent with mortality compression. Cohorts born after 1910, shown in the right panel, exhibit a strikingly different pattern: declines in $\hat{\lambda}_{50,c}$ were larger, but there appears to be little evidence of an increase in $\hat{\delta}_c$, a pattern

strongly consistent with mortality postponement. Unless there is a dramatic increase in their RDA's only at older ages, a pattern for which there appears to be little or no historical precedent, we can expect that the GMA and the maximum age at death of individuals in these cohorts will rise.

FIGURE 5: Observed mortality hazard rates (dots) and estimated base mortality hazard rates (lines) by age for Swedish males born 1880-1950

NOTE: Estimated base mortality rates are calculated using modal estimates $\hat{\lambda}_{50,c}$ and $\hat{\delta}_c$ for each cohort.

Figure 6 shows full results for Swedish females (left column) and males (right column). Panel A shows $\hat{\lambda}_{50,c}$ plotted against $\hat{\delta}_c$ for males and females by birth cohort. Females appear to have lower values of $\hat{\lambda}_{50,c}$ but higher values of $\hat{\delta}_c$ than males of the same cohort. $\hat{\delta}_c$ varies between 0.07 and 0.10 per year of age, and is not obviously constant across cohorts. In fact, statistical tests based on the Bayesian Information Criterion (BIC) and the Akaike Information Criterion (AIC), shown for all countries in Table 2, decisively reject the hypothesis that δ_c is constant across cohorts in Sweden for both males and females. Although changes in $\hat{\lambda}_{50,c}$ and $\hat{\delta}_c$ are small and appear random for the earliest cohorts (which may be related to data issues (see the data appendix for Sweden at HMD, 2021)), starting with cohorts born around 1775 the dominant pattern is that falls in $\hat{\lambda}_{50,c}$ are associated with rises in $\hat{\delta}_c$ for both males and females, a pattern associated with mortality compression (similar to Panel A of Figure 5). There are, however, two sets of cohorts for which both $\hat{\delta}_c$ and $\hat{\lambda}_{50,c}$ fell, a pattern associated with mortality postponement. The first occurred for cohorts born roughly between 1855 and 1875 for females (later and shorter for males), but was of much shorter duration and lower significance than the second such period, illustrated in Panel B of Figure 5, which is projected to occur largely for cohorts that are still censored by survival: those born between around 1920 and 1945 for females (1902 and 1930 for males). The historically dominant pattern of increases in $\hat{\delta}_c$ but falls in $\hat{\lambda}_{50,c}$ is expected to resume for cohorts born after about 1945 (although these estimates are imprecise and depend heavily on the chosen prior).

FIGURE 6: Bayesian estimates of slope and intercept parameters of male and female cohorts for Sweden (panel A); implied changes in remaining cohort base life expectancy at age 50 over ten year birth cohorts, divided into the portion due to postponement and portion due to compression (panel B), and 95% confidence intervals for the age at which cohort mortality hazard first reaches 2/3 (Λ_c) and the maximum observed lifespan (M_c) in each birth cohort (panel C).

NOTE: Confidence intervals are not shown in Panel A for ease of interpretation. In the other panels, bands or bars show 95% confidence intervals for the true base value. In panel B, bar heights show median estimates. Black dots show the actual change in life expectancy for extinct cohorts. Note that under the assumption that the model is correct, this would equal the true base value plus random error due to calendar-year effects and random variation, plus the change in bias due to these. In panel C, black dots show the longest-lived person in each birth cohort from ILD[39] and GRG[40] data (green if they are still alive in 2021). The mean of M_c increases in line with the logarithm of cohort size at age 50 even if there are no changes in mortality rates. The shaded area to the right of the diagonal line in panel C indicates censoring in 2021.

TABLE 2: Statistical tests for $\delta_c = \delta \ \forall \ c$

Country	Males				Females			
	AIC		BIC		AIC		BIC	
	Constant δ	Variable δ_c	Constant δ	Variable δ_c	Constant δ	Variable δ_c	Constant δ	Variable δ_c
Australia	-14,448	-14,796	-13,827	-13,579	-14,424	-14,520	-13,803	-13,303
Austria	-9,168	-9,928	-8,734	-9,083	-9,520	-9,618	-9,086	-8,774
Belgium	-29,044	-29,784	-27,799	-27,323	-26,869	-30,204	-25,624	-27,742
Canada	-17,222	-18,000	-16,601	-16,784	-14,789	-17,497	-14,168	-16,280
Denmark	-26,109	-29,494	-24,792	-26,888	-28,076	-32,015	-26,758	-29,408
Finland	-19,356	-20,235	-18,387	-18,324	-19,335	-20,685	-18,365	-18,771
France	-32,791	-35,427	-31,334	-32,541	-30,019	-33,769	-28,561	-30,883
Ireland	-7,602	-8,818	-7,205	-8,047	-8,353	-8,450	-7,956	-7,679
Italy	-22,652	-23,630	-21,650	-21,652	-19,733	-22,175	-18,731	-20,197
Japan	-10,456	-12,811	-10,023	-11,967	-9,517	-11,105	-9,083	-10,261
Netherlands	-23,370	-26,309	-22,192	-23,979	-21,722	-27,637	-20,543	-25,306
New Zealand	-7,080	-7,729	-6,698	-6,987	-7,305	-7,541	-6,923	-6,799
Norway	-25,500	-27,785	-24,272	-25,358	-23,539	-29,961	-22,312	-27,533
Portugal	-10,733	-11,549	-10,240	-10,587	-9,959	-10,765	-9,466	-9,802
Spain	-11,915	-14,302	-11,194	-12,887	-9,609	-12,806	-8,889	-11,391
Sweden	-33,316	-40,311	-31,306	-36,320	-32,313	-40,588	-30,301	-36,593
Switzerland	-22,705	-24,075	-21,734	-22,161	-17,909	-21,350	-16,939	-19,435
UK	-16,230	-17,525	-15,617	-16,323	-16,236	-18,319	-15,623	-17,118
USA	-15,463	-16,048	-14,925	-14,997	-13,035	-15,896	-12,497	-14,844

NOTE: The AIC and BIC prefer the model with the lowest criterion. In each case, the preferred model is highlighted in grey.

Panel B of Figure 6 illustrates our median estimates of the change in remaining base life expectancy at age 50 for cohorts born ten years apart, split into the portions due to compression and postponement, with 95% confidence intervals for each component. The pattern of postponement and compression described previously, and strikingly evident in Figure 5, is clear. We project that Swedish females born in 1950 can expect to live 7.5 years (males: 9.5 years) longer after the age of 50 than their counterparts born 50 years earlier. 60% (males: 70%) of this increase will be due to postponement. Postponement, but not compression, is projected to end for cohorts born after 1950 (although again these estimates are highly imprecise). Black dots in panel B show the actual change in cohort life expectancy over ten-year periods for extinct cohorts. These are close to our median estimates, but sometimes lie outside our confidence intervals due to random variation around base mortality, caused largely by calendar year effects.

Panel C shows confidence intervals (in red) for Λ_c, the Gompertzian maximum age at which the mortality hazard rate first reaches two thirds. Remarkably, Λ_c barely changed for males born over the two centuries before 1900 (or for females born between 1700 and 1860, after which the first period of mortality postponement identified in Panel A increased Λ_c by around 5 years), confirming that mortality postponement was the dominant pattern for these cohorts. Equally remarkably, despite different values of $\hat{\delta}_c$ and $\hat{\lambda}_{50,c}$ for males and females, Λ_c was very similar for Swedish males and females born between 1700 and 1860.

Panel C also shows 95% confidence intervals (in grey) for M_c, the age at death of the longest-lived person in each cohort, conditional on at least one person in each cohort reaching age Λ_c. Where data is available in the International Longevity Database (ILD, 2021) and a database maintained by the Gerontology Research Group (GRG, 2021), we plotted the actual age at death of the longest-surviving male and female in each birth cohort. The shading indicates the age/year-of-birth combinations that are

censored by survival, with the darker shade to the right representing censoring. For cohorts born before 1900, the mean of M_c increases due to increases in population, mortality compression and small amounts of postponement. But for cohorts born after 1900, the dramatic period of mortality postponement identified in Panel B may raise the observed maximum lifespan significantly in addition to the increases caused by population change. For instance, our estimates indicate that there is a 95% chance that the last Swedish female born in 1950 will only die aged between 117.1 and 125.5 years (so sometime between 2067 and 2075).

FIGURE 7: Changes in remaining base life expectancy at age 50 across 10-year birth cohorts separated into the portion due to postponement and the portion due to compression for males and females.

NOTE: These show the median changes in remaining cohort life expectancy at age 50 due to compression and postponement estimated from our posterior sample $\{\tilde{\lambda}_{50}, \tilde{\delta}\}$ for each country. Confidence bands for earlier cohorts are small, but widen for the most recent two decades shown. Detailed results for each country, along with confidence intervals, are available in the supplementary materials.

Figure 7 summarizes our median estimates for all 19 countries of the changes in remaining cohort life expectancy at age 50 between cohorts born 10 years apart, split between compression and postponement. Compression is the dominant pattern for cohorts born before 1900, replaced by postponement for those born after. These results show clearly why the distribution of the maximum lifespan has changed so little: cohorts that could have reached the maximum lifespan of 122 before 2021 were born before 1899, and so experienced improvements in life expectancy that were driven largely by compression.

FIGURE 8: 95% confidence intervals for the length of maximum life (M_c , grey) and the Gompertzian maximum age (Λ_c , red)

NOTE: Dots in the figures show the age at death (black dots) and current age (green triangles) of the longest-lived person in each birth cohort, from the ILD[39] and the GRG[40]. ILD data contains people over the age of 105 or 110 (depending on country); GRG data only show people over the age of 110. The mean length of maximum life increases due to compression and population increase (because more people are expected to reach age Λ_c) as well as postponement. Confidence intervals for M_c are conditional on at least one person reaching age Λ_c . Unconditional upper confidence intervals for M_c are always the same as the conditional ones, but the unconditional lower confidence interval may be smaller than shown in countries with small populations, especially for males. See methods section M3 for details.

Observed mortality patterns of censored cohorts suggest that this is likely to change as the cohorts that are benefiting from mortality postponement reach extreme old age. Figure 8 shows our estimated 95% confidence intervals for the GMA and the maximum observed lifespan of males and females in each country for cohorts born before 1970. (Figures similar to Figure 8, but showing Panels A and B of Figure 5 are shown for all countries in appendix S1.) Our confidence intervals for M_c fit ILD and GRG data very well in almost all countries. But our results show clearly that significant potential exists for longevity records to be broken in country after country and globally as cohorts born in the 1930's and 1940's reach extreme old age. Japanese females – who are already close to reaching longevity records – are simply at the forefront of this phenomenon.

As in all Bayesian approaches, our choice of prior is subjective. We therefore repeated our results for the three choices of prior shown in Table 1. Results are shown in the appendix. We found that our estimates of postponement are robust to the choice of prior for cohorts born before around 1950,

indicating a very strong signal from the data for these cohorts that mortality postponement is indeed occurring. We therefore emphasize results only for cohorts born before 1950. We also back-tested the model for Swedish data, and found reasonable, but not perfect, out-of-sample predictive power. Results are shown in appendix S1.

5. Discussion

Using historical data from 19 industrialized countries, we have shown that the dominant pattern of mortality improvement has been mortality compression, with little increase in either the GMA or in maximum attained ages over much of our historical data. We demonstrate, however, that there have indeed been occasional episodes of mortality postponement, which raise the GMA. The first of these occurred for cohorts born around 1880, raised the GMA by around 5 years, and appears more pronounced among females than among males. Other historical changes in observed maximum lives are attributable to increases in population rather than mortality postponement. We show that cohorts born between 1900 and 1950 are currently experiencing the most significant episode of mortality postponement yet recorded in our data. In combination with the effect of increases in cohort size, this indicates substantial potential for longevity records to rise by 10-15 years by the year 2060 in most of the countries we examine.

We emphasise that cohorts born before 1950 will only break existing longevity records if policy choices continue to support the health and welfare of the elderly, and the political, environmental and economic environment remains stable. The emergence of Covid-19 and its outsize effect on the mortality of the elderly provides a salutary warning that none of these is certain. If, however, the GMA does increase as the current mortality experience of incomplete cohorts suggests is likely, the implications for human societies, national economies and individual lives will be profound.

6. References

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