

Mortality Postponement, Mortality Compression and Healthy Aging

David McCarthy

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Abstract

The future of healthy aging and successful retirement depends on individuals having an accurate perception of their likely longevity. A key issue in how long individuals live is whether mortality at older ages is being compressed (i.e. whether more individuals are reaching older ages, but mortality at the oldest ages is broadly unchanged), or postponed (i.e. whether more individuals are reaching the oldest ages, but mortality at these ages is falling). We analyze historical period population mortality in industrialized countries and show that while there is significant evidence of postponement, compression has also been an extremely important factor in mortality improvements. One implication of this is that while the savings target needed to ensure a successful retirement at the median has increased dramatically, the increase at the 95th percentile has been much more modest: retirement savings targets have increased with increasing longevity, but the amount of risk in these targets has decreased both in absolute and in relative terms.

Keywords: Healthy aging, successful retirement, longevity perception, mortality compression, mortality postponement, historical population mortality

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David McCarthy

University of Georgia

101 Sanford Drive. Athens, GA 30602

david.mccarthy@uga.edu

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David McCarthy*

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Abstract

A key issue in planning a successful retirement is whether mortality at older ages is being compressed (i.e. whether more individuals are reaching older ages, but mortality at the oldest ages is broadly unchanged), or postponed (i.e. whether more individuals are reaching the oldest ages, but mortality at these ages is falling). We analyze historical period population mortality in industrialized countries between 1959 and 2019 and show that compression accounts for around two thirds (depending on sex and country) of the increase in the median age at death, while postponement becomes increasingly dominant at higher percentiles. One implication is that while the savings target needed to ensure a successful retirement at the median has increased dramatically, the increase at the 95th percentile has been much more modest: retirement savings targets have increased with increasing longevity, but the required margin for longevity risk in these targets has decreased both in absolute and in relative terms.

1 Introduction

Understanding the patterns of human mortality at older ages is crucial for predicting future life expectancy and planning for retirement needs. A central question both of these is whether mortality is primarily being compressed — with deaths increasingly clustered around a certain age — or postponed, shifting mortality risks to later in life. These two processes have different origins — compression may be the result of longevity reaching a biological limit (Dong et al, 2016, Manton et al, 1991), while postponement may reflect the dependence of longevity on underlying genetic

*McCart Family Distinguished Professor of Risk Management and Insurance, Terry College of Business, University of Georgia, Athens GA, e-mail: david.mccarthy@uga.edu.

or economic endowments (Bosworth et al, 2016; Chetty, 2016; Poterba, Venti and Wise, 2015), or widespread medical advances (Crimmins and Beltran-Sanchez, 2011; Crimmins and Finch, 2006).¹ This paper develops a new approach for measuring the relative importance of the two processes, uses it to analyze historical period mortality rates in industrialized countries, and then examines the implications of the results for individuals approaching retirement.

We begin by providing a broad overview of human mortality, with a particular focus on old-age mortality and the Gompertz law of mortality, which describes an exponential increase in mortality rates with age. We then use the Gompertz law to define mortality compression and mortality postponement more precisely.

Next, we examine two empirical tests that have been used in the literature to distinguish between compression and postponement. The first method, following Zuo et al (2018), analyzes changes in the percentiles of the distribution of age at death, while the second approach, following McCarthy and Wang (2023), relies on the Gompertz law to track shifts in mortality patterns. We propose a unified approach that combines insights from both perspectives and apply this framework to period mortality data from industrialized countries from 1959 to 2019, providing new insights into the relative importance of compression and postponement.

Finally, we explore the implications of our findings for successful retirement planning, highlighting how shifts in mortality patterns affect both the expected present value of consumption needs in retirement and the amount of risk in this quantity. We show that while the expected present value of future consumption in retirement has risen as longevity has increased, the amount of risk has decreased in both relative and absolute terms, largely because of the important role played by mortality compression in longevity improvements. The paper concludes by summarizing key findings, discussing how mortality may change in the future, and why this is important, and discussing potential future research.

¹See Cutler et al (2006) for a review of the determinants of mortality and Kirkwood and Austad (2000) for a discussion of the biological factors underlying aging. Vaupel (2010) discusses the biodemography of aging.

2 Mortality compression and mortality postponement

A key issue in studies of aging is whether mortality at older ages is being postponed or compressed.

In this section, we define the two alternatives and show why answering this question is key to understanding developments in individual life expectancy at older ages.

However, before turning to this issue, it is useful to understand the broad characteristics of human mortality. As an example, Figure 1 shows the one-year death probability of US males and females at each age in 2000, obtained from the Human Mortality Database. The vertical axis is on a logarithmic scale.

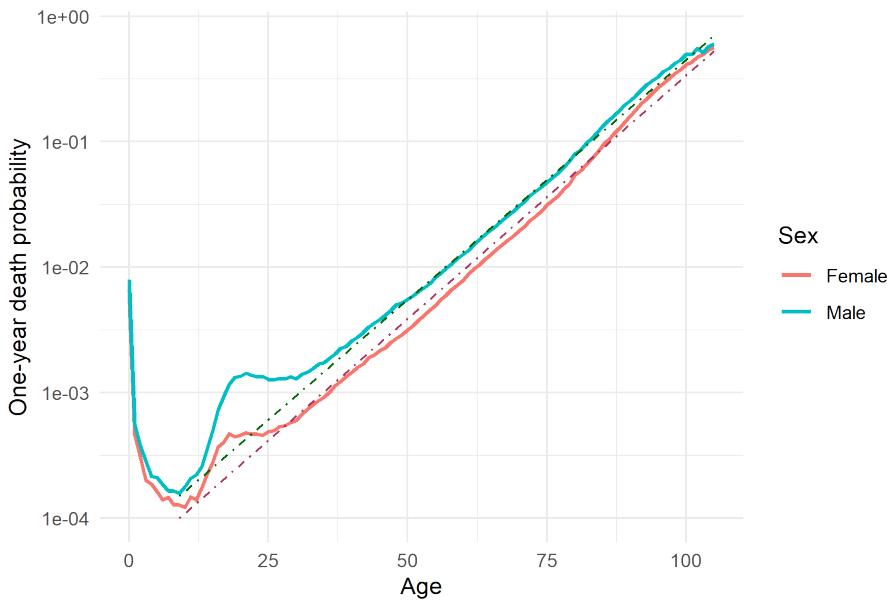


Figure 1: US population mortality rates by age and gender, 2000 (www.mortality.org). Lines are indicative.

Various features are notable. First, the death probabilities of newborns and infants are very high, being roughly the same as those of 50-year-olds for males and 60-year-olds for females. Mortality declines after birth, reaching a lifetime low point around 10 years of age for both boys and girls. From this point onwards, three effects are visible. First, the so-called accident hump, which affects the mortality of teenagers and young adults, and which is more pronounced for males than for

females. Second, there is a more gentle, broader hump in female mortality, largely representing risks associated with childbirth, but which has almost vanished in modern times due to advances in health care. Finally, there is an exponential increase in death probabilities with age, starting around age 40, called senescence. (On the graph, an exponential increase shows as linear because of the logarithmic scale). The first researcher to notice the near-exponential effect of aging on human mortality at older ages in humans was Benjamin Gompertz (1825), after whom the associated mortality ‘law’ is named; the Gompertz law states that mortality at older ages increases exponentially with age. The Gompertz law has been applied to the mortality of various species, as well as to machinery, and it may be an inherent feature of the processes governing the failure of all complex machines, as suggested by Gavrilov and Gavrilova (1981, 2015). In this paper, we will adopt the working assumption that the ‘natural’ pattern of human aging is provided by the Gompertz law. Some evidence in favor of this proposition is provided by age-mortality curves in the early industrial period (although data are incomplete and possibly incorrect). Further indicative evidence can be seen by extending the Gompertz line backwards before age 40, as has been done in the figures (lines are purely indicative). It is perhaps not a coincidence that the Gompertz line rejoins the mortality curves at the age at which mortality reaches a minimum; this suggests that senescence following the Gompertz law is present from as early as age 10, but obscured by the accident humps. While we stress that both of these sets of evidence are simply indicative, and that this assumption must therefore remain speculative, we will show in this paper that it is useful in interpreting broad patterns of mortality change at older ages over the last 70 years.

We now narrow our focus to consider only the mortality rates only of those aged 50+, shown in Figure 2. The solid lines represent the same mortality rates shown in Figure 1: those of the US population in 2000. The solid curves have a gradient of around 9.11% for males, and a slightly higher 9.72% for females. Female mortality at age 50 is lighter than for males of the same age, and so the curves gradually converge as individuals age.

We stress that these so-called period curves represent different underlying people (those dying aged 50 in 2000 were born in 1950; those dying aged 100 were born in 1900), and so do not represent the

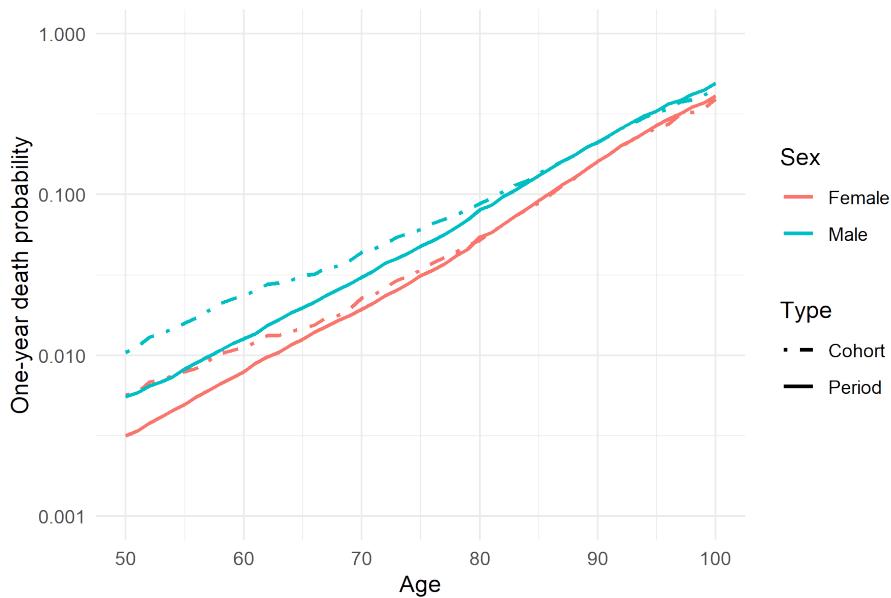


Figure 2: US cohort and period mortality rates by age and gender (period rates for 2000, cohort rates for generation of 1910, www.mortality.org)

lived experience of any individual or cohort, nor can they be used to examine biological features of the aging process. Instead, for these purposes, we must use so-called cohort mortality rates, which track the mortality of a given population of people as they age.

These cohort rates are shown as dashed lines in Figure 2, for the birth cohort of 1910. Surviving members of this cohort reached the age of 90 in 2000, so the two curves coincide at that point. At younger ages, the birth cohort of 1910 experienced heavier mortality than those of the same age who were alive in 2000, because mortality improved between 1960 (when that cohort reached age 50) and 2000; at ages older than 90, the birth cohort of 1910 experienced slightly lighter mortality than the population in the year 2000, because mortality improved between 2000 and 2010, when surviving members of the birth cohort of 1910 reached age 100. The slope of the cohort mortality curve is slightly shallower than for the period mortality curve, at around 8% per year of age for males and 8.5% per year of age for females; individual mortality for the cohort of 1910 therefore approximately doubled with each advancing decade of age. While noting that period mortality rates are simply an

analytical tool not applicable to any single individual, in this paper, we focus on period mortality rates, and return to a discussion of cohort mortality rates in the conclusion.

Although it cannot be seen in Figure 2, the Gompertz law appears to fail at older ages, in modern times usually after people have reached the age of 100 (or even older). At these very advanced ages, increases in mortality rates with age begin decline and mortality may even plateau at very advanced ages. Although data is very sparse, perhaps precluding any definitive empirical conclusion, there are strong theoretical reasons for believing that some degree of leveling at advanced ages is correct, related to differential frailty within each cohort (Vaupel et al, 1979). Imagine that each individual within a cohort has mortality that follows the Gompertz law with the same rate of increase by year of age, but some start off with higher annual mortality probabilities than others, for inherent, perhaps biological, reasons. As time progresses, frailler individuals will die at a faster rate, meaning that the cohort is increasingly comprised of those individuals who initially had lower mortality probabilities. The implication is that the observed mortality of the cohort will rise by less than the rate of increase in mortality rates of each individual member. At younger old ages, this survivor bias is very small because mortality probabilities are low. But as mortality probabilities reach high levels, it becomes significant and observed mortality rates increase by less than the rate of increase of the mortality of each individual person. If severe enough, mortality may eventually plateau, and there is some empirical evidence - admittedly disputed - that suggests that this happens when the annual mortality probability is around 0.5 (Barbi et al, 2018, Alvarez et al, 2021), or the mortality intensity is around $2/3$.² Following McCarthy and Wang (2023), we call the age at which the mortality intensity first reaches $2/3$ the Gompertzian Maximum Age (GMA), or Λ , and use Λ to divide mortality differences between two populations between compression and postponement.

Now, given this mortality pattern, imagine two possible medical interventions that affect mortality. Imagine first a disease that strikes independently of age once people reach age 50, but which is fatal, and to which, say, 50% of the population is vulnerable for genetic or other reasons. Assume that

²A mortality intensity or hazard rate, usually written as m or μ , refers to the instantaneous probability of death conditional on being alive (more precisely, the limit of the ratio of the probability of observing a death during a given time interval and the length of that interval, as this length approaches zero). Death probabilities, usually written as q , refer to the probability of dying over the next year

some medical advance allows this disease to be cured without any long-term deleterious effects on the mortality of survivors. The effect on overall mortality of this intervention will be much greater at age 50 than at age 100. The reason for this is that when people are aged 50, all 50% who are vulnerable could potentially get the disease, and so are saved, whereas by the time people have reached age 100, almost all of the people who could have got this disease have done so and are already dead. So the effect on mortality rates of this medical advance are as shown in the blue area in Figure 3 - a large reduction in mortality at age 50, which gradually decreases with age, leaving mortality at age Λ broadly unchanged. The effect is a fall in the intercept of the Gompertz line at age 50, and an increase in its slope, keeping the mortality at age Λ unchanged. This type of mortality improvement causes the distribution of age at death to shift to the right, but become more concentrated, so there is an increase in the mean age at death but a reduction in the variance of age at death. For this reason, this type of mortality improvement is called mortality compression, because the distribution of age at death becomes increasingly compressed (Frees, 1980; Cheung and Robine, 2007; Kannisto, 1994, 1996, 2000, 2001; Thatcher et al, 2010, Olshansky, 2016, Wilmoth and Horliuchi, 1999).

Now imagine a second type of medical intervention that delays the underlying biological processes governing aging. There are many such drugs available for which these types of claims are being made - Rapamycin, NAD+ and Ozempic, to name a few, but such delays could conceivably also be due to environmental factors such as reduced stress, better nutrition, less exposure to extreme temperatures and other factors. Imagine that the population at age 50 is suddenly given access to this medication. From that point onwards, their rate of aging slows down, meaning that at each chronological age they now have mortality probabilities that untreated individuals would have attained at earlier ages. The effect of this type of intervention is that the slope of the age-mortality rate curve falls and the GMA rises, shown as the orange area in Figure 3. This type of mortality improvement is called mortality postponement, because the distribution of age at death shifts to the right reflecting the slowing of the aging process itself (in this example). (Wilmoth et al, 2000, Robine and Vaupel, 2001, Vaupel 1997, Oeppen and Vaupel, 2002, Rau et al 2008).

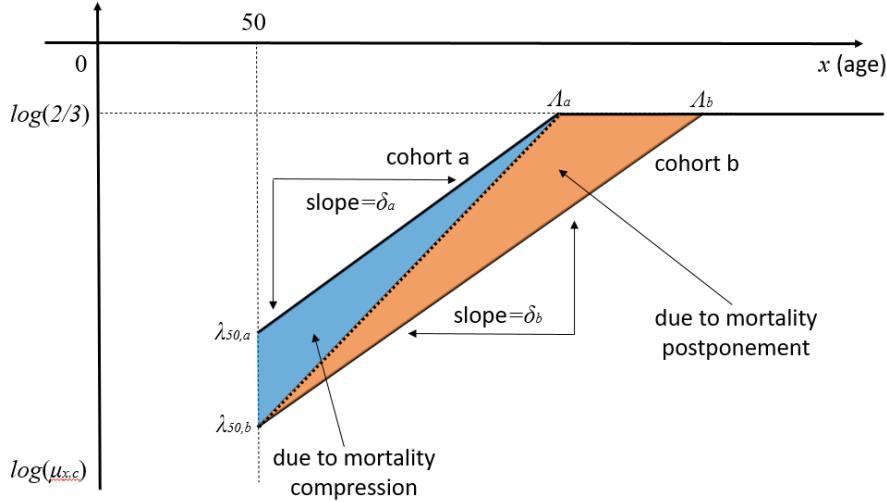


Figure 3: Decomposition of changes in slope and intercept of Gompertz mortality law into compression and postponement

Note that mortality compression changes both the slope and the intercept of the fitted Gompertz law, while mortality postponement changes only the slope; equivalently, mortality compression leaves the GMA unchanged but postponement causes it to increase. This division allows changes in mortality rates between two different populations to be apportioned to changes due to compression and changes due to postponement.³

3 Two tests

The analysis in the previous section suggests two tests of whether mortality is being postponed or compressed. The first uses the distribution of age at death, and tests whether it is becoming more

³Note that the disaggregation shown in Figure 3 ascribes all reduction in the intercept to compression, and all changes in the GMA to postponement. An alternative division could ascribe changes in the GMA keeping the slope constant to postponement, and then any changes in the slope to compression. Neither approach is entirely satisfactory. If, for instance, the slope remains the same but the intercept falls, the first approach might wrongly ascribe part of what might reasonably be called mortality postponement to compression, while if the intercept remains the same but the GMA increases, the second approach would suggest large amounts of postponement and negative compression. For ease of comparability, we retain the approach used by McCarthy and Wang (2023), while acknowledging its shortcomings.

compressed or not, by examining, say, the distance between the median and the 90th percentile. This approach was used in period data by Zuo et al (2018). They used period mortality data to calculate the difference between the 90th percentile and other percentiles of the age at death conditional on reaching a certain age (say 50; they used 65). Under mortality compression, this distance should fall. Under postponement, it should stay the same. They found that there was little evidence that the distribution of age at death over time was becoming tighter, and suggested that the dominant pattern was therefore mortality postponement, and that this was a period-based phenomenon. The difficulty with this approach is that the effects of changes in slope and intercept of the Gompertz law is ambiguous as far as expected death and variance are concerned. Changes in dispersion alone are therefore not indicative of whether mortality is being compressed or postponed in the sense illustrated in Figure 3.

A second approach approximates mortality rates with the Gompertz law by estimating the intercept and the slope, and examines the relationship between changes in the slope and changes in the intercept. The second approach was used by McCarthy and Wang (2023) on cohort, rather than period, data, using a Bayesian estimation technique to overcome difficulties associated with cohort censoring. In contrast to Zuo et al (2018), they found that the dominant pattern in historical cohort data was mortality compression, with occasional busts of postponement.

In this paper, we attempt to reconcile the two approaches by applying the method of McCarthy and Wang (2023), with some³ changes, to the same period data used by Zuo et al (2018). This entails the following steps:

1. Estimating Gompertzian parameters using period mortality data for each country, year and sex.
2. Calculating various measures of interest, such as the average various percentiles of the age at death using these parameters as well as the actual mortality data to quantify the error associated with using the Gompertz model.
3. Decomposing the changes in these quantities of interest between different years into the portion due to compression, the portion due to postponement and the portion due to changes in model error.

We now discuss each step in more detail. For ease of reading, mathematical details are given in the

mathematical appendix.

3.1 Estimating Gompertzian parameters

We used linear regression to estimate a set of Gompertzian parameters for period mortality data for each country, year and sex. We used data from the Human Mortality Database (www.mortality.org), the same data used by Zuo et al (2018). We set the intercept parameter equal to the observed mortality intensity at age 50 to ensure that each Gompertzian curve started at the correct point.⁴ When performing the estimation, we note that each estimated mortality hazard rate in the HMD does not have equal statistical credibility. In fact, the variance of the estimate depends on the number of deaths at that point: the fewer deaths that occur (either because the underlying population is small or because the mortality probability is small) the less statistically reliable the estimated rate from the HMD. In doing the regression, we therefore weighted the HMD hazard rate at each age with the number of deaths at that age (this approach was first used by ***). As a consequence, the decline in mortality at older ages does not bias the results very much because very few deaths occur at these extremely advanced ages. We include all data, suitable weighted as discussed, in our estimates. Details are shown in the mathematical appendix.

Numerical results are shown for the US in Table 1. Panel A refers to males and Panel B to females. We chose to use 2019 as the final year because it is the last year pre-Covid, so it prevents (the hopefully temporary) effects of Covid on mortality from biasing our conclusions, and examined mortality each decade starting in 1959 (roughly the same starting point used by Zuo et al (2018); they used 1960). The estimated intercept is shown in line A and the slope in line B in each panel. For males and females, the intercept falls fairly consistently over the period, indicating mortality improvement at age 50. From 1959 to 1999, the slope parameter increases for males, suggesting the presence of compression, and decreases between 1999 and 2019, suggesting the presence of postponement. For females, the slope parameter is more variable. But we cannot decisively choose between compression and postponement without examining the Gompertzian Maximum Age (as

⁴We could have fitted both the intercept and the slope using regression; the line of best fit would then under-predict mortality at the very youngest ages. The overall predictions would be similar but the interpretation would be somewhat clouded. Because mortality rates at 50 are reasonably precisely estimated, we retain this approach here.

a reminder, denoted the GMA, or Λ , this is the age at which the Gompertzian-implied mortality intensity first reaches $2/3$ (equivalent to an annual death probability of ~ 0.5). Because the slope parameter is estimated with error, our estimate of Λ has a statistical distribution, which we calculated using results shown in the mathematical appendix.

Results are reported in lines C and D of each panel. Strikingly, the GMA changed by less than 9 months for US males between 1959 and 1999, suggesting that mortality improvements for males were driven largely by compression. For US females over the same period, the GMA increased by nearly two years. Between 1999 and 2019, however, the GMA increased by nearly 5 years for males and 3 years for females, indicating significant and rapid mortality postponement. Over the whole period, the GMA increased by 5.63 years for males and 4.94 years for females. The expected age at death calculated using the Gompertzian model (shown in line E, see the mathematical appendix for details) increased consistently over the period for both males and females, over the whole period by 6.30 years for males and 5.22 years for females.

To aid interpretation of these results, Figures 4 and 5 show the true and fitted Gompertzian hazard rates for males and females from 1959 to 2019. The patterns of compression and postponement described in the previous section are evident in these graphs. But more insights can be gleaned: until 1999, the Gompertz model fits mortality hazard rates for males extremely well until at least age 90, and possibly even higher. At older ages, mortality rates flatten and even appear to decline. Part of this is due to differential frailty leading to selection at older ages, but part may also be due to misrecording of age at death (individuals dying aged ~ 100 in 1959 were born around 1859, and their true ages might have been difficult to determine; see the work of Saul Newman ***). But starting in 2009 in these data, the Gompertz model fits male mortality increasingly badly: mortality is too low relative to the Gompertzian estimate for US males in their 70's and early 80's, and too high relative to the Gompertzian estimate for those in their late 80's and 90's. Maintaining our working assumption is that the Gompertz model represents 'natural' mortality due to senescence, the conclusion would be that some intervention is shifting some deaths from the seventh and eighth decades of life to the ninth and tenth, and that this effect is getting stronger over time. Note also

that the slowing of the rate of mortality increase with age for those older than age 100 is still present; this evidence can be assumed to be more credible than similar results in earlier years due to better recording of birth dates in the first decades of the 20th century relative to fifty years earlier in the US.

For females, similar patterns hold. The Gompertz law fits again better in earlier years, and worsens in the same systematic way in more recent years: deaths are increasingly being shifted from those in their sixties and seventies to those in their eighties and nineties. But for females, this shifting of mortality relative to the Gompertz law is observable even in 1959.

These patterns are made more concrete when we examine the distribution of age at death rather than the hazard rates. Figure 6 for males and Figure 7 for females shows the distribution of age at death of a theoretical population aged 50 that experiences the mortality rates at each age that the US population experienced in each year of our investigation. These types of calculations are the basis of the life expectancy at birth measures often reported in the popular press, and produce measures that are useful to understand patterns in mortality tables, even if they do not apply to real individuals who will only reach older ages in future years when mortality patterns will have changed, and do not reflect the actual distribution of age-at-death in the population due to changes in cohort size. Dotted lines show the distribution of age at death implied by the fitted Gompertz model, while solid lines show the distribution of age at death if mortality rates at each age equaled those experienced by the US population in that year. In earlier years, the Gompertz model fits well, but in later years the shifting of deaths into the ninth and tenth decades of life is evident. Interestingly, however, the Gompertz model still fits well at younger ages (by design), and at the oldest ages. Again, making the assumption that the Gompertz model represents the 'natural' level of mortality, this suggests two operative effects: one is causing the Gompertz parameters themselves to shift, and the other is increasingly causing deviations from the Gompertz law in the middle of the age distribution. Note that the number of deaths of people in their nineties is higher than the Gompertz model would predict both because the surviving population at these ages is larger (because fewer individuals died earlier than the Gompertz model predicted) and because the actual mortality rates

are higher than the Gompertz model would predict for that age. This population effect magnifies the errors in the Gompertz model, which is why such small differences in the predicted hazard rates appear to have such large effects in the distribution of age at death.

A quantitative measure of the goodness-of-fit of the Gompertz model is provided by the Kolmogorov-Smirnov test statistic, defined as the largest absolute value of the difference between the empirical cumulative distribution function (not the density functions shown in the figures) of age at death and the theoretical distribution of the same quantity derived using the Gompertz model. Results are shown in line E of table 1. A higher number indicates a worse fit. For males the Gompertz model fits equally well until 1989, when it starts to worsen. For US females, the fit is worse than for males, even in 1949, and rises fairly consistently over the sample period.

3.2 Calculating measures of interest

Since the focus of this paper is on retirement preparedness, we focus on two questions that should concern any person planning their financial affairs. First, what is my expected lifespan, and second, how uncertain is it? While variance is a traditional measure of uncertainty, it is hard to interpret where the underlying distribution is not Guassian (as is the case here). So to answer the second question, we focus on percentiles of the distribution of age at death. Following Zuo et al (2018), we calculate the median, 75th and 90th percentiles, to which we add the 95th percentile using both the fitted Gompertz model and the true distribution of age at death. The GMA can be thought of as the 99.99th percentile - not quite the top of the distribution, but very close to it.

The expected age at death of a theoretical population aged 50 that suffers the mortality experienced by the US population at each age in 1959 was 73.1 years for males (shown in line G of Table 1), and rose steadily to reach 80.1 years by 2019. For females, the expected age at death was nearly 5 years higher at 77.1 years, rising to 83.7 years by 2019. Note that these values are higher than life expectancy at birth for those years because we are assuming that the population is initially aged 50; these are therefore life expectancy measures conditional on reaching age 50. They imply that a 50-year old male who expects to suffer mortality at each future age equal to the mortality

suffered by the US population in 2019 can expect to live for an additional 30.1 years, and a female an additional 33.7 years.

Given the apparently large errors in the distribution of age at death, it is perhaps surprising to note that the Gompertz model would predict very similar numbers (shown in line E): 72.9 years in 1959, rising to 79.2 years in 2019 for males, and 77.1 years in 1959 rising to 82.3 years in 2019 for females.

But any individual who planned their financial affairs only on this basis would run out of money with a probability of around 0.5, depending on whether the distribution is left- or right-skewed. Most people would probably regard this risk as unacceptable. A more reasonable approach would be choose a percentile of the age-at-death distribution, and plan around that. To accommodate various levels of risk aversion, we calculated the median (50th percentile) (line K), 75th percentile (line O), 90th (line S) and 95th (line W) percentiles; a 50-year old individual who planned their consumption and savings using the 95th percentile as a reference would face a 1/20 chance of running out of money and so on. In 2019, the median age at death for males aged 50 was 81 years, not too different from the mean. But the 75th percentile was 88.1 years, the 90th 92.9 years and the 95th 95.4 years. For females, the results are even more extreme: the median is 84.9 years (again, slightly higher than the mean), the 75th percentile is 91 years, the 90th 95 years and the 95th 97.8 years. These results imply that to be 95% certain of not outliving their assets, a 50-year old US male would need to plan on living another 45.4 years and a female 47.8 years, significantly higher than the mean value typically used for financial planning. This point has been made forcefully by Mitchell ***.

3.3 Understanding changes in these measures

We now use the approach in Figure 3 to divide changes in these quantities over each decade into three pieces. For the first two, the change due to mortality postponement and the change due to mortality compression, we use the fitted Gompertz model and formulae explained in the mathematical appendix. To this we add a third component: changes in the extent to which the Gompertz model is a good fit, representing model error. Because the Gompertz distribution appears to fit well at the very oldest ages, this third set of errors can be regarded as compression of a different type: some

intervention which is increasingly shifting the the middle of the age-at-death distribution to the right with little effect at the very oldest ages, in addition to the Gompertzian compression and postponement identified following Figure 3. Of course, future advances in medical technology may change this as medical attention shifts to the process of aging itself.

This disaggregation is shown in rows H, I and J of Table 1 for the expected age at death, rows L, M and N for the median, rows P Q and R for the 75th percentile etc. In each column, these figures represent changes over the subsequent decade, with total changes over the period due to each factor in the right-most column. For instance, in 1959, the expected age at death was 73.09 years for males. Over the subsequent decade, postponement reduced this by 0.11 years, compression raised it by 0.13 years, and model error reduced it by 0.1 years. The sum of these three components is a reduction of 0.08 years, and the expected age at death in 1959 was $73.09 - 0.08 = 73.01$ years, as shown in the next column of the table (figures may not add due to rounding). For US males, the figures show that most mortality improvements between 1959 and 1999 were due to Gompertzian compression, with Gompertzian postponement and model error playing a very small role. Over the subsequent two decades, between 1999 and 2019, there was significant postponement. For females, on the other hand, postponement was more evenly spread throughout the period. Over the entire 60 years, the majority of changes in the mean, median, 75th and 90th percentiles were due to compression for both males and females, with postponement playing a much smaller role. Only for changes in the 95th percentile did postponement comprise more than half of the change. Note also that the higher the percentile, the lower the total change (compare, for example, rows K and W in the last column for both males and females). As expected from the figures, Gompertzian error is most significant at the median, given the shifting in the distribution of age at death that the error is capturing. But even at the median, the model error only accounts for around one sixth ($1.53/8.48$) of the total change for males and slightly less for females. At the 90th percentile, the error is of the order of one twentieth, before rising slightly in the 95th percentile.

3.4 Other countries

Much ink has been spilled - rightly - lamenting poor US life expectancy (see, for example, Case and Deaton, 2015). What is not often realized, however, is that US life expectancy at birth is so poor relative to peer countries primarily - but not entirely - because of higher mortality at younger ages. To explore this issue we now turn to a comparison of the US and other countries, focusing initially on France, Japan and the UK. These countries were chosen for size of population (larger is better), length of data availability (longer is better) and reliability of mortality statistics (Spain and Italy have large populations and available data, but show some evidence of inaccurate mortality and birth records in earlier years; German data is not available before reunification in 1990). Similar results to those shown for the US can be found in Tables 2 to 4 and Figures 8 to 19.

In 1959, US males and females had GMA's that were much higher, and life expectancy at 50 slightly higher than France, Japan and the UK. Over the subsequent 60 years, mortality in those countries improved much faster than the US. By 2019, all three countries had life expectancy at 50 between 2 and 3 years higher than the US for males, and between 1 and 5 years higher for females, although the GMA for the US was still higher than any country except Japan for males and females. The slope for the US was lower, but the intercept higher, meaning that the gap in mortality hazard rates between the US and other countries narrows with advancing age for both males and females. Older than around age 85 for instance, in 2019, US males had very similar death probabilities than males of the same age in the other three countries, but US females had lower mortality rates than UK females and higher mortality rates than either French or Japanese females of the same age. At ages younger than these, the US is a conspicuous outlier among this group.

The disaggregation into postponement, compression and model error shows that other countries typically had higher postponement and compression, but lower model error than the US. Improvements were much more consistent across time in the other countries than in the US, especially for males, where the US pattern of postponement, in particular, appears to be highly anomalous.

Table 5 shows the total change in the various measures over the period 1959 to 2019 for a larger sample of countries, and its disaggregation into postponement, compression and model error. Very

similar patterns are evident: most of the change is due to compression, with postponement taking a back seat. The greater the percentile, the more significant postponement becomes. Model errors are largest at the median and smallest at the 90th percentile, before increasing slightly at the 95th percentile, suggesting that deviations from the Gompertz law are primarily consistent with mortality compression rather than postponement in all of these countries.

4 Implications for successful retirement

We now turn to examining the implications of our results for successful retirement. Successful retirement depends on many factors; here we focus only on the effect of uncertainty in lifespan, and make the assumptions needed to reduce the complexity of the underlying problem to the point where it can be analyzed efficiently. There seem to be two main tests financial advisors use to assess retirement adequacy. Fidelity (2025) and T Rowe Price (2025) use an asset test and recommend that individuals have between 8 and 13 times their wages in assets in order to retire. It is unclear whether income should be net or gross of taxes and housing costs and whether assets include the value of owner-occupied housing or whether the value of assets held in tax-preferred accounts such as non-Roth 401(k) plans or IRA's should be netted down for taxes. Vanguard (2024), on the other hand, uses an income test based on the '4%' rule, and maintains that 4% of retirement assets plus social security should be sufficient to meet around 80% of pre-retirement wages. The income and asset tests are broadly interchangeable given an individual's social security (SS) replacement rate. For instance, if an individual's SS replacement rate is 30%, then Vanguard's approach would suggest that retirement assets would need to be $(0.8-0.3)/0.04 = 12.5$ times pre-retirement income.

For the purposes of this paper, we need a measure of retirement adequacy that focuses only on the effect of uncertainty in lifespan. While recognizing that both the asset- and income-based approaches have their uses, particularly where most individuals take on substantial amounts of investment risk in retirement, we therefore depart from both and use a consumption-based measure of adequacy instead. We ask a simple question: what multiple of total consumption do individuals need in assets at retirement in order to *guarantee* that they have sufficient assets to reach a particular percentile

of the age-at-death distribution (measured at age 50), if their consumption is to remain constant in real terms over the remainder of their lives. We exclude allowance for investment risk, and so discount future consumption at long-term real US Treasury rates (currently around 2.5% p.a.). By taking investment risk, individuals may earn higher expected returns, but these come at the expense of greater variability of outcomes, and the market price of this risk is reflected in asset prices and hence future returns. Of course, newly-retired people are likely to differ from the ‘average’ investor in systematic ways. First, they are likely to be more, rather than less, risk averse, if only because of their lack of human capital, and second, they probably have a long time horizon because they are investing to finance future consumption, which may be as much as three or even four decades distant. If equity market returns are mean-reverting, recent retirees may well do better by taking on some investment risk, and many financial advisors indeed recommend that they do, but precise examination of this point is beyond our scope. We focus on asset needs at the point of retirement because this allows easy comparability with the commonly-used tests described above, even though we are examining the distribution of age-at-death conditional on reaching 50 rather than on reaching retirement age.⁵ We also ignore the possibility that individuals can hedge some of their longevity risk, either by purchasing an annuity (to my knowledge, CPI-linked annuities are not currently available for purchase in the US, and few people purchase immediate annuities of any type in any case), or by engaging in mortality risk transfer with their families (by trading the possibility of a bequest in the case of early death with care provided or financed by family members if longevity is higher than expected; this approach is probably much more common, although most transactions of this type are probably implicit rather than explicit). Table 6 shows this multiple for retirement at age 62; table 7 for retirement at age 67 and table 8 for retirement at 70. In each table, Panel A shows figures for males and Panel B figures for females. Each year is shown in a different row, and each column shows the percentile of the age-at-death distribution (conditional on reaching age 50). The final column shows the GMA (remaining life expectancy at the GMA is 1.5 years under the assumption that mortality intensities remain constant thereafter).

Table 6 shows that a male retiring at 62 in 1959 would have needed assets worth 6.4 times

⁵We may alter our tables to reflect this point in future versions

consumption in order to be 50% sure that he would not outlive his assets, and assets worth 13.8 times consumption in order to be 95% sure. Note that these figures assume that social security replaces 30% of consumption and that assets are held in after-tax accounts. If assets are held in pre-tax accounts such as IRA's or 401(k) accounts, these values would need to be grossed up by dividing by $1 - \tau$ where τ is the individual's average tax rate in retirement. For instance, an individual whose social security income covered 30% of their consumption, but who paid an average tax rate in retirement of 20% would need assets in pre-tax accounts of $6.41/0.8 = 8.01$ times consumption in order to cover future consumption with 50% probability. Different SS replacement rates (or DB pension income or annuity income that is CPI-indexed) can likewise be allowed for by multiplying these figures by $(1 - RR_{SS,DB})/0.7$. So 1959 US male for whom SS covered only 20% of consumption would need post-tax assets of $6.41 \times 0.8/0.7 = 7.33$ times consumption.

Females have much higher asset needs than males at lower percentiles, reflecting their lower mortality, but as the degree of certainty required increases, asset needs rise and those of males approach those of females. As the degree of certainty required rises, asset need unsurprisingly rise. To be 95% sure of outliving his assets, a male retiring in 1959 at age 62 needed $13.8 / 6.41 = 2.15$ as many assets as someone who was content to be 50% sure of not outliving his assets. But by 2019, this multiple had shrunk for both males and females (to $15.71 / 10.49 = 1.50$ for males and from 1.62 in 1959 to 1.36 for females). This pattern holds in both relative and in absolute terms.

Table 7 show equivalent figures for people retiring at age 67, and Table 8 for those retiring at 70. As the retirement age gets older, the amount of assets required falls. Note that the tables likely underestimate the effect of working longer because they assume a constant 30% of consumption needs are met by SS; as retirement is delayed, SS payments increase due to the adjustment factors applied to SS pension amounts reflecting early or delayed retirement. As retirement age increases, the higher asset requirements caused by uncertainty in lifespan rises: males retiring at 62 in 2019 needed 50% more assets to move from the median to the 95th percentile, but males retiring at 67 needed $14.09/8.19 - 1 = 72\%$ more assets and males retiring at 70 needed $17.04 - 6.67 - 1 = 95\%$ more assets. Because SS affects both the assets needed to reach the median age-at-death and the assets

needed to reach any other percentile by an equal proportion, these ratios are unchanged regardless of social security.

We therefore have two stylized facts that need explanation: first, the effect of longevity uncertainty on savings targets at retirement has fallen dramatically over the last sixty years, and second that the effect of longevity uncertainty rises as retirement age rises. The first is explained by the pattern of mortality improvement over the last six decades: as we have shown, for US males and females, compression, rather than postponement, has been the dominant pattern in mortality improvement. This means that the lower percentiles of the age-at-death distribution have increased faster than the higher percentiles. For instance, the median age-at-death of US males conditional on reaching age 50 increased by 8.48 years (compression explains around 80% of this change), but the 95th percentile only increased by 5.85 years (postponement explains around 65% of this) (6.71 and 4.89 years for females). The second reason is that improvements in higher percentiles have a smaller effect on savings targets than improvements in lower percentiles because of the effect of compound interest: the median age at death is reached sometime like 14 years before the 95th percentile for US males and females, meaning that assets held have a longer time to accrue interest and a lower present value.

The second stylized fact (that the effect of uncertainty rises with the retirement age) is primarily a mechanical effect of working longer: the amount of assets needed to reach the median age at death falls by more than the amount of assets needed to reach the 95th percentile, implying that the amount of risk must increase.

5 Conclusion

This study has examined whether improvements in old-age mortality are primarily driven by mortality compression or mortality postponement, using period mortality data from industrialized countries. Our findings suggest that the dominant pattern has varied over time and between countries. In the US, for example, mortality improvement among US males were largely explained by mortality compression in the second half of the twentieth century, but postponement became highly significant

in the first two decades of the twenty-first, while for US females, postponement and compression were more evenly spread across the last sixty years. In other countries we have examined, postponement was relatively more important than in the US, suggesting that yet greater improvements in US old-age mortality are possible.

Our analysis also shows that the Gompertz model has become an increasingly poor model of old-age period mortality. In most countries, the Gompertz model fit mortality after age 50 reasonably well in 1959, especially for males. Over time, some deaths in the eighth and ninth decade of life have shifted (or been shifted) to the ninth and tenth decades, even once changes in the Gompertz parameters over time have been allowed for. We tentatively suggest that these changes may be due to the increased ability of medical technology to treat diseases afflicting the aged that in prior times would have been fatal. This 'Gompertzian error', as we have termed it, primarily affects the age-at-death distribution around its median, but appears to have little effect at extreme old ages.

These changes have had two significant effects on the savings target needed for a successful retirement. First, the savings target needed to sustain consumption until the average or median age-at-death has increased dramatically. In 2019, a US male retiring at 67 for whom social security supported 30% of his consumption needed after-tax savings of 8.19 times annual consumption to support himself until the median age-at-death, more than double the 3.58 times consumption he would have needed at the same age in 1959, representing a 129% increase. At the same time, however, the amount needed to reach the higher percentiles has not increased by as much. For instance, the amount of savings needed to be 95% sure of matching post-retirement consumption increased by only 18% between 1959 and 2019 for the same individual. Part of this is due to simple compound interest: payments late in life, discounted to retirement age, are less significant in present value terms than payments received earlier. But part of the difference also reflects the fact that a significant fraction of longevity improvement is due to compression rather than postponement.

One important qualification of our results is that they are based on period mortality rates. Individuals age with time, meaning that savings targets strictly depend on cohort, rather than period mortality. One interesting possibility is that the increasingly-large deviations from the Gompertz law that we

have identified in period mortality rates affecting primarily the middle of the old-age distribution are a product of cohort-related changes in Gompertzian parameters rather than changes in the applicability of the Gompertz law itself. As medical interventions increasingly target aging itself, rather than just its symptoms, and are successful in doing so, we may expect such a pattern to emerge in the data in the coming years. Investigation of this point requires the type of analysis we have presented here to be repeated for birth cohorts, rather than calendar years, an investigation we intend to undertake.

6 Mathematical appendix

6.1 Regression analysis

The HMD produces estimates of the mortality hazard rate $m_{i,j,k}$ at age i in country j for sex k . Suppressing subscripts j and k , we have the following model:

$$\log(m_i) = \lambda + \delta(i - 50) + \epsilon_i \quad (1)$$

Each estimate of m_i is obtained by dividing the observed number of deaths at age i by the known total amount of time people in the data are observed at age i , called the exposure-to-risk at age i or E_i , so $m_i = D_i/E_i$ and $\log(m_i) = \log(D_i) - \log(E_i)$. Although D_i is binomial with mean $E_i q_i$, and variance $E_i q_i (1 - q_i)$ where q_i is the true but unknown probability of death at age i , we approximate it with the lognormal distribution with parameters μ and σ^2 , to obtain $\sigma^2 = \log(1 + \frac{1-q_i}{E_i q_i}) \approx \frac{1-q_i}{E_i q_i} \approx \frac{1}{E_i q_i}$. Following the general principle of weighted least squares, which is to set the observation weight equal to the inverse of the variance of the error, we therefore set the weight of each point equal to the number of observed deaths at each age, which is our best estimate of $E_i q_i$.

In the regression, we also set our estimate of λ equal to the observed mortality rate at age 50, and so estimate the slope δ using weighted least squares.

6.2 Gompertzian maximum age

We use our estimates of λ and δ obtained from (1) to calculate the GMA as:

$$\hat{\Lambda} = \frac{\log(2/3) - \hat{\lambda}}{\hat{\delta}} + 50$$

From the properties of regression estimators,

$$(\lambda, \delta) \sim BVN(M, \Sigma) \text{ so } (\log(2/3) - \lambda, \delta) \sim BVN(M^*, \Sigma^*)$$

where

$$M^* = \begin{bmatrix} \log(2/3) - \mu_\lambda \\ \mu_\delta \end{bmatrix}$$

and

$$\Sigma^* = \begin{bmatrix} \sigma_\lambda^2 & -\sigma_{\lambda\delta} \\ -\sigma_{\lambda\delta} & \sigma_\delta^2 \end{bmatrix}.$$

Hence,

$$E[\Lambda] \approx \frac{\log(2/3) - \mu_\lambda}{\mu_\delta} \left(1 + \frac{\sigma_\delta^2}{\mu_\delta^2} + \frac{\sigma_{\lambda\delta}}{(\log(2/3) - \mu_\lambda)\mu_\delta} \right) + 50.$$

and

$$\text{Var}(\Lambda) \approx \frac{1}{\mu_\delta^2} \left(\sigma_\lambda^2 + 2\sigma_{\lambda\delta} \frac{(\log(2/3) - \mu_\lambda)}{\mu_\delta} + \frac{(\log(2/3) - \mu_\lambda)^2}{\mu_\delta^2} \sigma_\delta^2 \right).$$

6.3 Life expectancy and percentiles of Gompertz distribution

If a population has mortality at each age exactly equal to that implied by the Gompertz law with parameters λ and δ , and mortality hazard rate plateaus at the GMA at $2/3$, then remaining period life expectancy at 50 is given by:

$$BLE(\lambda, \delta) = \frac{1}{\delta} \exp(\exp(\lambda)/\delta) (E_1(\exp(\lambda)/\delta) - E_1(2/3/\delta)) + 1.5 \exp\left(\frac{\exp(\lambda) - 2/3}{\delta}\right) \quad (2)$$

where $E_1(x)$ is the exponential integral defined as $E_1(x) = \int_x^\infty \frac{e^{-y}}{y} dy$.

Percentiles can be obtained from the distribution function of the Gompertz distribution.

6.4 Dividing changes between postponement and compression

The change in base life expectancy between two populations a and b with $b > a$ can then be calculated as:

$$BLE(\lambda_b, \delta_b) - BLE(\lambda_a, \delta_a) = BLE(\lambda_b, \delta_b) - BLE(\gamma_0, \gamma_1) + BLE(\gamma_0, \gamma_1) - BLE(\lambda_a, \delta_a), \quad (3)$$

where $\gamma_0 = \lambda$ and $\gamma_1 = \frac{\log(2/3) - \lambda_b}{\log(2/3) - \lambda_a}$

The first two terms represent the change due to postponement and the second two the change due to compression.

A similar approach is followed for percentiles.

Model error is estimated by taking the difference between actual life expectancy (or percentiles) and the life expectancy (or percentiles) measured off the fitted Gompertz distribution for that year.

7 References

Alvarez J, Villavicencio F, Strozza C and Camarda CG, (2021), Regularities in human mortality after age 105, *PLoS One*, <https://doi.org/10.1371/journal.pone.0253940>

Barbi, E., Caselli, G. & Vallin, J. (2003) Trajectories of Extreme Survival in Heterogeneous Populations. *Population* 58, 43065 <https://doi.org/10.3917/popu.301.0045>

Barbi, E., Lagona, F., Marsili, M., Vaupel, J. & Wachter, K. (2018) The plateau of human mortality: Demography of longevity pioneers. *Science* 360: 1459-1461 <https://www.science.org/doi/10.1126/science.aat3119>

Bongaarts J (2005) Long-range trends in adult mortality: Models and projection methods. *Demography* 42:23-49.

Bongaarts, J. (2009) Trends in senescent life expectancy. *Popul. Stud.* 63, 203-213 <https://doi.org/10.1080/00324720903165456> .

Bosworth B, Burtless G, Zhang K (2016) Later retirement, inequality in old age, and the growing gap in longevity between rich and poor. *Economic Studies at Brookings*(Brookings Institution, Washington DC).

Canudas-Romo, V. (2008) The modal age at death and the shifting mortality hypothesis. *Demogr. Res.* 19, 1179-1204 <https://dx.doi.org/10.4054/DemRes.2008.19.30>

Case A, Deaton A (2015) Rising morbidity and mortality in midlife among white non-Hispanic Americans in the 21st century. *Proc Natl Acad Sci USA* 112:15078–15083.

Chetty R, et al. (2016) The association between income and life expectancy in the United States, 2001-2014. *JAMA* 315:1750–1766.

Cheung, S. L. K. Robine, J.-M. (2007) Increase in common longevity and the compression of mortality: The case of Japan. *Popul. Stud.* 61, 85-97 <https://doi.org/10.1080/00324720601103833> .

Crimmins E, Beltran-Sanchez H (2011) Mortality and morbidity trends: Is there compression of morbidity? *J Gerontol Ser B Psychol Sci Soc Sci* 66:75–86.

Crimmins E, Finch C (2006) Infection, inflammation, height, and longevity. *Proc Natl Acad Sci USA* 103:498–503.

Cutler D, Deaton A, Lleras-Muney A (2006) The determinants of mortality. *J Econ Perspect* 20:97–120.

Dong X, Mulholland B, Vijg J (2016) Evidence for a limit to human lifespan. *Nature* 538:257–259.

Edwards R, Tuljapurkar S (2005) Inequality in life spans and a new perspective on mortality convergence across industrialized countries. *Popul Dev Rev* 31:645–674.

Engelman M, Canudas-Romo V, Agree EM (2010) The implications of increased survivorship for mortality variation in aging populations. *Popul Dev Rev* 36:511–539.

Fay, R. and Herriot, R., (1979) Estimates of Income for Small Places: An Application of James-Stein Procedures to Census Data *J. Am. Stat. Ass.*, 74, 269–277 <https://doi.org/10.1080/01621459.1979.10482505>

.

Fidelity, (2025) "How much do I need to retire?", <https://www.fidelity.com/viewpoints/retirement/how-much-do-i-need-to-retire>, accessed April 29th 2025.

Fries, J F. (1980) Aging, natural death, and the compression of morbidity. *N. Engl. J. Med.* 303, 130-135 <https://www.nejm.org/doi/full/10.1056/NEJM198007173030304>

Gavrilov, L.A. & Gavrilova, N.S. (2011) Mortality Measurement at Advanced Ages. *N. Am. Actuar. J.* 15, 432-447 <https://doi.org/10.1080/10920277.2011.10597629>

Gavrilova, N.S. & Gavrilov, L.S. (2015) Biodemography of Old-Age Mortality in Humans and Rodents. *J. Gerontol. Ser. A* 70, 1-9 <https://doi.org/10.1093/gerona/glu009>

Goldman D, et al. (2009) The benefits of risk factor prevention in Americans aged 51 years and older. *Am J Public Health* 99:2096-2101

Gompertz, B. XXIV. (1825) On the nature of the function expressive of the law of human mortality, and on a new mode of determining the value of life contingencies. In a letter to Francis Baily, Esq. F. R. S. &c. *Phil. Trans. R. Soc. Lond.* 115, 513-583 <https://doi.org/10.1098/rstl.1825.0026>

Horiuchi, S. & Wilmoth, J.R. (1998) Deceleration in the age pattern of mortality at older ages. *Demography* 35, 391-412 <https://doi.org/10.2307/3004009>

Human Mortality Database www.mortality.org (accessed 2025)

Kannisto V (2000) Measuring the compression of mortality. *Demographic Res* 3:6.

Kannisto, V. (1994) Development of Oldest-Old Mortality, 1950-1990: Evidence from 28 Developed Countries .

Kannisto, V. (2001) Mode and dispersion of the length of life. *Population* 13, 159-171 <https://doi.org/10.2307/3030264>.

Kannisto, V. (1996) The Advancing Frontier of Survival: Life Tables for Old Age. Odense Univ. Press .

Kannisto, V., Lauritsen, J., Thatcher, A. R. & Vaupel, J. W. (1994) Reductions in mortality at advanced ages: several decades of evidence from 27 countries in *Studies in the Economics of Aging* (ed. Wise, D. A.) 79-104. Univ. Chicago Press. <https://doi.org/10.2307/2137662>

Kaplanis J, et al. (2018) Quantitative analysis of population-scale family trees with millions of relatives. *Science* 360:171-175.

Kirkwood TB, Austad SN (2000) Why do we age? *Nature* 408:233-238.

Lenart A, Aburto JM, Stockmarr A, Vaupel JW. (2018) The human longevity record may hold for decades. In: Jeune B, Vaupel JW, Maier H, editors. *Supercentenarians*. Springer <https://arxiv.org/abs/1809.03732> .

Lenart, A. and Missov T., (2010) Max Planck Institute for Demographic Research Working Paper 2010-024. <https://www.demogr.mpg.de/papers/working/wp-2010-024.pdf>.

Manton, K.G., Stallard, E. and Tolley, H.D., (1991) Limits to Human Life Expectancy: Evidence, Prospects, and Implications. *Popul. Dev. Rev.* 17: 603-637 <https://doi.org/10.2307/1973599>

Oeppen, J. & Vaupel, J. (2002) Broken Limits to Life Expectancy. *Science* 296 (5570):1029-31 <https://www.science.org/doi/10.1126/science.1069675>

Olshansky, S. Measuring our narrow strip of life. *Nature* 538, 175–176 (2016). <https://doi.org/10.1038/nature19475>

Poterba J, Venti S, Wise DA (2015) What determines end-of-life assets? A retrospective view (National Bureau of Economic Research, Cambridge MA), Technical Report WP 21682.

Rau, R., Soroko, E., Jaslionis, D. & Vaupel, J. W. (2008) Continued Reductions in Mortality at Advanced Ages. *Popul. Dev. Rev.* 34, 747-768 <http://dx.doi.org/10.1111/j.1728-4457.2008.00249.x> .

Robine, J & Vaupel, J.W. (2001) Supercentenarians: slower ageing individuals or senile elderly? *Exp. Gerontol.* 36, 915-930 [https://doi.org/10.1016/S0531-5565\(00\)00250-3](https://doi.org/10.1016/S0531-5565(00)00250-3)

Rootzen, H and Zholud, D, (2017) Extremes, 20, 713-728 <https://doi.org/10.1007/s10687-017-0305-5>

T. Rowe Price (2025), "You're age 35, 50, or 60: How much should you have saved for retirement by now?", <https://www.troweprice.com/personal-investing/resources/insights/youre-age-35-50-or-60-how-much-should-you-have-by-now.html>, accessed 29th April 2025

Thatcher AR, Cheung SLK, Horiuchi S, Robine JM (2010) The compression of deaths above the mode. *Demographic Res* 22:505–538.

Thatcher, A.R., Kannisto, V. & Vaupel, J.W. (1998) The force of mortality at ages 80 to 120 (Odense Univ. Press).

Tuljapurkar S, Li N, Boe C (2000) A universal pattern of mortality decline in the G7countries. *Nature* 405:789–792.

Vanguard, (2024), "How much do I need to retire?", <https://investor.vanguard.com/investor-resources-education/retirement/how-much-do-i-need-to-retire>, accessed 29th April 2025.

Vaupel, J W, Manton, K G & Stallard, E. (1979) The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography* 16(3):439-54

Vaupel, J. (2010) Biodemography of human ageing. *Nature* 464, 536–542 <https://doi.org/10.1038/nature08984>

Vaupel, J. W. & Lundström, H. (1994) Studies in the Economics of Aging.

Vaupel, J. W. (1997) The remarkable improvements in survival at older ages. *Phil. Trans. R. Soc. Lond. B.* 352, 1799-1804 <https://doi.org/10.1098/rstb.1997.0164>

Vaupel, J. W., et al. (1998) Biodemographic Trajectories of Longevity. *Science* 280, 855-860 <https://www.science.org/doi/10.1126/science.280.5365.855>

Wilmoth JR, Horiuchi S (1999) Rectangularization revisited: Variability of age at deathwithin human populations. *Demography* 36:475–495

Wilmoth, J. R., Deegan, L. J., Lundström, H. & Horiuchi, S. (2000) Increase of maximum life-span in Sweden, 1861-1999 *Science* 289, 2366-2368 <https://www.science.org/doi/10.1126/science.289.5488.2366>

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8 Tables

Panel A: US Males aged 50	1959	1969	1979	1989	1999	2009	2019	Total Δ
Intercept (A)	-4.5963	-4.6171	-4.8345	-5.0566	-5.2421	-5.2342	-5.3302	
Slope (B)	0.0801	0.0811	0.0826	0.0878	0.0911	0.0843	0.0849	
GMA (C)	102.35	101.92	103.59	102.99	103.09	107.3	107.98	5.63
Var(GMA) (D)	0.1743	0.1918	0.1415	0.1083	0.1135	0.4266	0.4173	
Exp. age at death (Gomp) (E)	72.9	72.92	74.75	75.94	77.06	78.39	79.2	6.30
K-S statistic (F)	0.01	0.01	0.01	0.01	0.03	0.06	0.05	
Expected age at death (G)	73.09	73.01	74.98	76.18	77.6	79.48	80.09	7
Δ due to: postponement (H)	-0.11	0.47	-0.18	0.03	1.37	0.22		1.8
compression (I)	0.13	1.36	1.38	1.09	-0.05	0.6		4.5
model error (J)	-0.1	0.14	0	0.3	0.57	-0.21		0.7
Median age at death (K)	72.53	72.38	74.56	76.08	77.92	80.31	81.02	8.48
Δ due to: postponement (L)	-0.11	0.47	-0.19	0.03	1.39	0.23		1.83
compression (M)	0.16	1.57	1.57	1.22	-0.05	0.67		5.13
model error (N)	-0.2	0.15	0.13	0.59	1.05	-0.19		1.53
75th perc. of age at death (O)	80.47	80.24	82.43	83.54	84.87	87.18	88.08	7.60
Δ due to: postponement (P)	-0.16	0.69	-0.26	0.05	1.92	0.31		2.54
compression (Q)	0.14	1.39	1.36	1.04	-0.04	0.57		4.46
model error (R)	-0.21	0.12	0.01	0.24	0.43	0.02		0.6
90th perc. of age at death (S)	86.37	86.32	88.39	89.19	90	92.04	92.87	6.5
Δ due to: postponement (T)	-0.21	0.86	-0.32	0.06	2.34	0.38		3.1
compression (U)	0.12	1.19	1.15	0.87	-0.04	0.47		3.77
model error (V)	0.03	0.03	-0.03	-0.11	-0.26	-0.03		-0.37
95th perc. of age at death (W)	89.49	89.55	91.59	92.21	92.73	94.58	95.35	5.85
Δ due to: postponement (X)	-0.23	0.95	-0.35	0.06	2.55	0.42		3.39
compression (Y)	0.11	1.07	1.03	0.77	-0.03	0.42		3.37
model error (Z)	0.19	0.01	-0.06	-0.31	-0.68	-0.07		-0.91

(table continues below)

Panel B: US Females aged 50	1959	1969	1979	1989	1999	2009	2019	Total Δ
Intercept (A)	-5.2074	-5.2505	-5.4632	-5.6282	-5.7897	-5.7217	-5.8165	
Slope (B)	0.0893	0.088	0.0876	0.0919	0.0972	0.0912	0.0923	
GMA (C)	103.78	105.03	107.72	106.85	105.41	108.32	108.62	4.84
Var(GMA) (D)	0.2391	0.2801	0.2711	0.1752	0.1657	0.4036	0.4424	
Exp. age at death (E)	77.09	77.75	79.93	80.59	80.93	81.66	82.31	5.22
K-S statistic (F)	0.04	0.05	0.05	0.04	0.04	0.07	0.07	
Expected age at death (G)	77.85	78.64	80.76	81.25	81.65	83.12	83.69	5.85
Δ due to: postponement (H)	0.41	0.94	-0.32	-0.56	1.1	0.11		1.69
compression (I)	0.25	1.24	0.97	0.9	-0.36	0.54		3.54
model error (J)	0.12	-0.05	-0.17	0.06	0.75	-0.07		0.63
Median age at death (K)	78.14	79.06	81.46	81.96	82.39	84.07	84.85	6.71
Δ due to: postponement (L)	0.42	0.97	-0.33	-0.58	1.14	0.12		1.73
compression (M)	0.28	1.37	1.06	0.97	-0.39	0.58		3.88
model error (N)	0.22	0.05	-0.24	0.04	0.93	0.09		1.1
75th perc. of age at death (O)	84.9	85.84	88.26	88.78	88.89	90.37	90.98	6.07
Δ due to: postponement (P)	0.58	1.3	-0.43	-0.76	1.49	0.15		2.34
compression (Q)	0.24	1.16	0.89	0.8	-0.32	0.48		3.24
model error (R)	0.11	-0.03	0.06	0.07	0.3	-0.02		0.5
90th perc. of age at death (S)	90.03	90.91	93.4	93.76	93.61	94.92	95.41	5.38
Δ due to: postponement (T)	0.7	1.56	-0.52	-0.89	1.76	0.18		2.8
compression (U)	0.2	0.96	0.74	0.66	-0.26	0.39		2.69
model error (V)	-0.02	-0.03	0.14	0.08	-0.19	-0.08		-0.1
95th perc. of age at death (W)	92.89	93.7	96.13	96.38	96.09	97.39	97.78	4.89
Δ due to: postponement (X)	0.77	1.69	-0.56	-0.96	1.9	0.19		3.04
compression (Y)	0.18	0.85	0.65	0.58	-0.23	0.35		2.38
model error (Z)	-0.14	-0.11	0.16	0.08	-0.37	-0.14		-0.53

Table 1: Gompertzian fitted parameters (first sub-panel) and various metrics (following sub-panels) of the distribution of age at death conditional on reaching age 50, and disaggregation of changes in these metrics due to postponement, compression and model error, US. Males in Panel A and Females in Panel B. Changes refer to changes over the subsequent decade.

Panel A: French Males aged 50	1959	1969	1979	1989	1999	2009	2019	Total Δ
Intercept (A)	-4.6865	-4.6676	-4.728	-4.9964	-5.1162	-5.3104	-5.6048	
Slope (B)	0.0862	0.086	0.0819	0.0856	0.0854	0.085	0.0906	
GMA (C)	99.67	99.54	102.78	103.61	105.17	107.73	107.42	
Var(GMA) (D)	0.1295	0.1005	0.2339	0.2544	0.41	0.5576	0.4949	
Exp. age at death (E)	72.76	72.61	73.85	75.77	76.98	79	80.66	
K-S statistic (F)	0.03	0.01	0.04	0.04	0.06	0.08	0.08	
Expected age at death (G)	73.23	72.96	74.66	76.5	78.19	80.29	81.89	8.66
Δ due to: postponement (H)	-0.04	0.89	0.25	0.49	0.85	-0.11		2.33
compression (I)	-0.11	0.36	1.66	0.72	1.17	1.77		5.58
model error (J)	-0.11	0.45	-0.07	0.48	0.08	-0.06		0.76
Median age at death (K)	73.07	72.43	74.66	76.92	78.79	81.41	83.28	10.22
Δ due to: postponement (L)	-0.04	0.87	0.25	0.49	0.86	-0.12		2.33
compression (M)	-0.13	0.42	1.91	0.82	1.31	1.95		6.28
model error (N)	-0.47	0.94	0.1	0.56	0.44	0.04		1.61
75th perc. of age at death (O)	80.22	79.86	81.82	83.97	85.62	87.84	89.47	9.25
Δ due to: postponement (P)	-0.05	1.29	0.36	0.7	1.19	-0.16		3.32
compression (Q)	-0.12	0.37	1.67	0.71	1.12	1.64		5.39
model error (R)	-0.19	0.29	0.12	0.26	-0.08	0.14		0.54
90th perc. of age at death (S)	85.53	85.48	87.25	89.04	90.46	92.46	93.76	8.23
Δ due to: postponement (T)	-0.07	1.63	0.44	0.85	1.43	-0.18		4.11
compression (U)	-0.1	0.31	1.42	0.59	0.94	1.37		4.53
model error (V)	0.12	-0.18	-0.06	-0.03	-0.37	0.12		-0.4
95th perc. of age at death (W)	88.4	88.47	90.11	91.79	92.98	94.86	96.03	7.63
Δ due to: postponement (X)	-0.08	1.82	0.49	0.93	1.57	-0.2		4.53
compression (Y)	-0.09	0.28	1.27	0.53	0.83	1.21		4.04
model error (Z)	0.23	-0.45	-0.08	-0.27	-0.52	0.16		-0.94

(table continues below)

Panel B: French Females aged 50	1959	1969	1979	1989	1999	2009	2019	Total Δ
Intercept (A)	-5.353	-5.38	-5.6527	-5.8882	-5.9615	-6.0521	-6.2343	
Slope (B)	0.0972	0.0955	0.0978	0.1	0.0986	0.0959	0.0992	
GMA (C)	100.93	102.11	103.65	104.82	106.35	108.89	108.77	
Var(GMA) (D)	0.2718	0.3024	0.3873	0.4223	0.5313	0.6568	0.5931	
Exp. age at death (E)	76.94	77.5	79.53	81.21	82.2	83.7	84.6	
K-S statistic (F)	0.05	0.06	0.08	0.09	0.11	0.12	0.13	
Expected age at death (G)	77.99	78.74	81	82.93	84.33	85.93	86.86	8.86
Δ due to: postponement (H)	0.42	0.59	0.47	0.62	1.05	-0.05		3.1
compression (I)	0.14	1.44	1.21	0.37	0.46	0.95		4.58
model error (J)	0.18	0.23	0.25	0.42	0.11	0.03		1.22
Median age at death (K)	78.34	79.09	81.6	83.69	85.2	87.01	88.09	9.75
Δ due to: postponement (L)	0.43	0.61	0.49	0.65	1.09	-0.05		3.22
compression (M)	0.16	1.57	1.29	0.39	0.49	0.99		4.9
model error (N)	0.16	0.33	0.3	0.47	0.23	0.14		1.63
75th perc. of age at death (O)	84.44	85.26	87.34	89.19	90.56	92.25	93.2	8.76
Δ due to: postponement (P)	0.58	0.8	0.63	0.83	1.39	-0.07		4.16
compression (Q)	0.13	1.3	1.06	0.32	0.4	0.81		4.01
model error (R)	0.11	-0.02	0.16	0.23	-0.1	0.21		0.59
90th perc. of age at death (S)	89.17	89.94	91.82	93.4	94.67	96.2	97.06	7.90
Δ due to: postponement (T)	0.69	0.95	0.74	0.96	1.61	-0.08		4.87
compression (U)	0.11	1.07	0.86	0.26	0.32	0.66		3.28
model error (V)	-0.03	-0.13	-0.01	0.04	-0.4	0.29		-0.25
95th perc. of age at death (W)	91.74	92.55	94.34	95.74	96.92	98.42	99.23	7.50
Δ due to: postponement (X)	0.75	1.02	0.79	1.03	1.73	-0.08		5.24
compression (Y)	0.1	0.94	0.76	0.23	0.28	0.58		2.88
model error (Z)	-0.04	-0.17	-0.15	-0.08	-0.52	0.32		-0.63

Table 2: Gompertzian fitted parameters (first sub-panel) and various metrics (following sub-panels) of the distribution of age at death conditional on reaching age 50, and disaggregation of changes in these metrics due to postponement, compression and model error, France. Males in Panel A and Females in Panel B. Changes refer to changes over the subsequent decade.

Panel A: Japanese Males aged 50	1959	1969	1979	1989	1999	2009	2019	Total Δ
Intercept (A)	-4.7744	-5.0284	-5.218	-5.4851	-5.4686	-5.7063	-6.0113	
Slope (B)	0.0944	0.1003	0.0957	0.0985	0.0939	0.0954	0.1015	
GMA (C)	96.29	96.11	100.27	101.56	103.9	105.56	105.22	
Var(GMA) (D)	0.1233	0.1403	0.2083	0.174	0.236	0.284	0.2173	
Exp. age at death (E)	72.3	73.59	75.98	77.87	78.62	80.54	81.99	
K-S statistic (F)	0.01	0.01	0.04	0.03	0.04	0.05	0.05	
Expected age at death (G)	72.62	73.91	76.76	78.45	79.44	81.42	82.84	10.22
Δ due to: postponement (H)	-0.06	1.44	0.48	0.84	0.63	-0.14		3.19
compression (I)	1.35	0.96	1.41	-0.09	1.28	1.59		6.51
model error (J)	0	0.45	-0.19	0.24	0.06	-0.04		0.52
Median age at death (K)	72.26	73.68	76.83	78.89	80.01	82.11	83.72	11.45
Δ due to: postponement (L)	-0.06	1.47	0.49	0.87	0.66	-0.14		3.27
compression (M)	1.54	1.07	1.55	-0.09	1.39	1.7		7.15
model error (N)	-0.06	0.62	0.02	0.34	0.06	0.05		1.03
75th perc. of age at death (O)	78.99	80.19	83.27	85.19	86.41	88.37	89.69	10.69
Δ due to: postponement (P)	-0.09	2	0.65	1.16	0.86	-0.18		4.4
compression (Q)	1.31	0.89	1.29	-0.08	1.15	1.39		5.96
model error (R)	-0.04	0.18	-0.02	0.14	-0.06	0.12		0.33
90th perc. of age at death (S)	84.25	85.32	88.28	90.06	91.22	93.04	94.07	9.82
Δ due to: postponement (T)	-0.1	2.42	0.78	1.38	1.01	-0.21		5.27
compression (U)	1.1	0.74	1.06	-0.06	0.95	1.13		4.91
model error (V)	0.08	-0.18	-0.06	-0.16	-0.14	0.11		-0.36
95th perc. of age at death (W)	87.16	88.07	90.97	92.75	93.83	95.6	96.45	9.29
Δ due to: postponement (X)	-0.11	2.64	0.84	1.5	1.09	-0.23		5.72
compression (Y)	0.97	0.65	0.94	-0.06	0.83	1		4.33
model error (Z)	0.06	-0.38	-0.01	-0.36	-0.16	0.08		-0.76

(table continues below)

Panel B: Japanese Females aged 50	1959	1969	1979	1989	1999	2009	2019	Total Δ
Intercept (A)	-5.1501	-5.4822	-5.8625	-6.095	-6.2076	-6.4529	-6.562	
Slope (B)	0.0957	0.1024	0.1052	0.1041	0.1009	0.1019	0.1039	
GMA (C)	99.56	99.59	101.89	104.67	107.51	109.37	109.27	
Var(GMA) (D)	0.2117	0.2239	0.3236	0.4334	0.4868	0.5254	0.5439	
Exp. age at death (E)	75.38	77.12	79.89	82.17	83.93	85.93	86.41	
K-S statistic (F)	0.04	0.04	0.05	0.07	0.08	0.09	0.11	
Expected age at death (G)	76.14	77.94	80.92	83.43	85.35	87.57	88.47	12.33
Δ due to: postponement (H)	0.01	0.95	1.19	1.23	0.84	-0.05		4.18
compression (I)	1.74	1.82	1.09	0.54	1.18	0.52		6.89
model error (J)	0.05	0.21	0.23	0.17	0.24	0.43		1.33
Median age at death (K)	76.24	78.2	81.32	83.98	86.1	88.46	89.43	13.19
Δ due to: postponement (L)	0.01	0.99	1.25	1.28	0.88	-0.05		4.36
compression (M)	1.92	1.96	1.14	0.56	1.22	0.53		7.33
model error (N)	0.03	0.17	0.27	0.28	0.25	0.48		1.49
75th perc. of age at death (O)	82.61	84.2	86.94	89.43	91.56	93.7	94.41	11.80
Δ due to: postponement (P)	0.01	1.26	1.57	1.61	1.09	-0.06		5.48
compression (Q)	1.6	1.59	0.92	0.45	0.98	0.43		5.97
model error (R)	-0.02	-0.12	0	0.07	0.07	0.35		0.34
90th perc. of age at death (S)	87.6	88.81	91.4	93.68	95.76	97.75	98.2	10.60
Δ due to: postponement (T)	0.01	1.47	1.81	1.85	1.24	-0.07		6.32
compression (U)	1.32	1.29	0.74	0.36	0.79	0.35		4.86
model error (V)	-0.12	-0.17	-0.28	-0.13	-0.05	0.18		-0.58
95th perc. of age at death (W)	90.35	91.36	93.84	96.03	98.1	99.94	100.29	9.94
Δ due to: postponement (X)	0.02	1.58	1.94	1.98	1.32	-0.08		6.76
compression (Y)	1.16	1.13	0.65	0.32	0.69	0.3		4.25
model error (Z)	-0.16	-0.24	-0.39	-0.22	-0.18	0.12		-1.07

Table 3: Gompertzian fitted parameters (first sub-panel) and various metrics (following sub-panels) of the distribution of age at death conditional on reaching age 50, and disaggregation of changes in these metrics due to postponement, compression and model error, Japan. Males in Panel A and Females in Panel B. Changes refer to changes over the subsequent decade.

Panel A: UK Males aged 50	1959	1969	1979	1989	1999	2009	2019	Total Δ
Intercept (A)	-4.8999	-4.9516	-4.9788	-5.3142	-5.4691	-5.692	-5.6793	
Slope (B)	0.098	0.0991	0.0972	0.1032	0.1018	0.0993	0.0951	
GMA (C)	95.88	95.88	97.06	97.59	99.74	103.26	105.44	
Var(GMA) (D)	0.3018	0.4096	0.2327	0.231	0.0983	0.1788	0.3654	
Exp. age at death (E)	72.84	73.12	73.64	75.55	77.11	79.58	80.35	
K-S statistic (F)	0.02	0.04	0.01	0.02	0.01	0.04	0.06	
Expected age at death (G)	72.62	72.74	73.63	75.5	77.49	80.36	81.61	9.00
Δ due to: postponement (H)	0	0.38	0.19	0.81	1.37	0.83		3.58
compression (I)	0.27	0.14	1.72	0.76	1.1	-0.07		3.93
model error (J)	-0.15	0.36	-0.04	0.42	0.4	0.49		1.49
Median age at death (K)	72.1	71.99	73.26	75.26	77.52	80.96	82.43	10.33
Δ due to: postponement (L)	0	0.38	0.19	0.83	1.42	0.86		3.69
compression (M)	0.31	0.16	1.92	0.83	1.19	-0.07		4.33
model error (N)	-0.42	0.73	-0.12	0.6	0.83	0.68		2.3
75th perc. of age at death (O)	79.08	79.13	80.1	82.06	84.1	87.22	88.57	9.48
Δ due to: postponement (P)	0	0.54	0.26	1.1	1.85	1.13		4.89
compression (Q)	0.26	0.14	1.61	0.69	0.97	-0.06		3.61
model error (R)	-0.22	0.3	0.08	0.25	0.3	0.28		0.99
90th perc. of age at death (S)	84.63	84.92	85.61	87.31	89.12	91.87	92.98	8.35
Δ due to: postponement (T)	0	0.66	0.31	1.31	2.18	1.33		5.79
compression (U)	0.22	0.11	1.33	0.56	0.79	-0.05		2.96
model error (V)	0.07	-0.08	0.05	-0.05	-0.22	-0.18		-0.41
95th perc. of age at death (W)	87.51	88.04	88.63	90.16	91.82	94.32	95.3	7.80
Δ due to: postponement (X)	0	0.72	0.34	1.42	2.35	1.44		6.27
compression (Y)	0.19	0.1	1.17	0.49	0.69	-0.04		2.6
model error (Z)	0.34	-0.24	0.02	-0.25	-0.55	-0.41		-1.08

(table continues below)

Panel B: UK females aged 50	1959	1969	1979	1989	1999	2009	2019	Total Δ
Intercept (A)	-5.3621	-5.4337	-5.4487	-5.7402	-5.8455	-6.0365	-6.1344	
Slope (B)	0.0994	0.0981	0.0959	0.1011	0.1015	0.1009	0.1016	
GMA (C)	99.88	101.25	102.59	102.76	103.59	105.82	106.39	
Var(GMA) (D)	0.1613	0.1124	0.1751	0.0781	0.148	0.2912	0.3637	
Exp. age at death (E)	76.61	77.48	78.04	79.64	80.5	82.37	83.1	
K-S statistic (F)	0.03	0.02	0.04	0.02	0.04	0.07	0.08	
Expected age at death (G)	77.36	78.03	78.83	80.11	81.48	83.7	84.61	7.24
Δ due to: postponement (H)	0.5	0.48	0.07	0.33	0.94	0.24		2.56
compression (I)	0.37	0.08	1.53	0.53	0.94	0.49		3.93
model error (J)	-0.2	0.23	-0.31	0.51	0.35	0.18		0.76
Median age at death (K)	77.5	78.19	79.24	80.55	81.96	84.46	85.51	8.01
Δ due to: postponement (L)	0.51	0.5	0.07	0.35	0.98	0.25		2.65
compression (M)	0.41	0.09	1.65	0.56	0.99	0.51		4.21
model error (N)	-0.23	0.47	-0.42	0.5	0.53	0.29		1.15
75th perc. of age at death (O)	83.8	84.77	85.64	87.06	88.2	90.35	91.13	7.33
Δ due to: postponement (P)	0.69	0.66	0.09	0.45	1.24	0.32		3.44
compression (Q)	0.34	0.07	1.36	0.46	0.8	0.41		3.44
model error (R)	-0.06	0.14	-0.03	0.24	0.1	0.05		0.44
90th perc. of age at death (S)	88.67	89.79	90.58	91.86	92.77	94.67	95.32	6.65
Δ due to: postponement (T)	0.82	0.79	0.11	0.52	1.43	0.37		4.04
compression (U)	0.28	0.06	1.12	0.37	0.65	0.33		2.81
model error (V)	0.03	-0.06	0.05	0.01	-0.18	-0.06		-0.2
95th perc. of age at death (W)	91.36	92.57	93.19	94.42	95.23	97.02	97.56	6.21
Δ due to: postponement (X)	0.89	0.86	0.12	0.56	1.54	0.39		4.35
compression (Y)	0.24	0.05	0.98	0.33	0.57	0.29		2.47
model error (Z)	0.09	-0.29	0.13	-0.08	-0.31	-0.14		-0.61

Table 4: Gompertzian fitted parameters (first sub-panel) and various metrics (following sub-panels) of the distribution of age at death conditional on reaching age 50, and disaggregation of changes in these metrics due to postponement, compression and model error, UK. Males in Panel A and Females in Panel B. Changes refer to changes over the subsequent decade.

Country	Sweden	Sweden	Netherlands	Netherlands	Australia	Australia	Canada	Canada	Italy	Italy	Spain
Sex	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male
Total Δ : Mean age at death	6.88	7.56	6.25	6.25	10.35	8.80	8.34	7.80	8.08	8.53	8.02
Comprised of: Postponement	1.64	2.12	1.71	2.22	3.69	2.44	1.77	2.37	1.60	2.58	2.21
Compression	5.01	5.43	3.87	3.61	5.14	5.43	6.05	4.80	5.86	4.95	5.56
Model error	0.24	0.00	0.67	0.42	1.53	0.94	0.52	0.66	0.62	1.01	0.21
Total Δ : Median	7.61	8.16	6.91	7.07	11.86	9.59	9.70	8.71	8.92	9.15	9.14
Comprised of: Postponement	1.68	2.21	1.78	2.30	3.81	2.53	1.82	2.45	1.66	2.68	2.20
Compression	5.36	5.68	4.16	3.80	5.65	5.77	6.69	5.12	6.38	5.22	6.11
Model error	0.57	0.27	0.97	0.98	2.41	1.29	1.19	1.14	0.88	1.25	0.70
Total Δ : 75th percentile	6.75	7.47	5.75	6.45	10.67	8.23	8.31	7.71	7.59	8.09	8.55
Comprised of: Postponement	2.25	2.80	2.29	3.02	5.01	3.25	2.42	3.21	2.15	3.40	3.08
Compression:	4.37	4.56	3.40	3.08	4.70	4.70	5.62	4.20	5.29	4.22	5.11
Model error:	0.13	0.10	0.06	0.35	0.97	0.29	0.26	0.30	0.16	0.46	0.33
Total Δ : 90th percentile	5.96	6.77	5.00	5.82	8.91	7.20	7.10	6.84	6.63	7.18	7.71
Comprised of: Postponement	2.67	3.25	2.67	3.57	5.92	3.79	2.89	3.78	2.53	3.94	3.64
Compression:	3.56	3.68	2.76	2.50	3.86	3.82	4.67	3.44	4.34	3.41	4.21
Model error:	-0.28	-0.16	-0.43	-0.24	-0.87	-0.41	-0.46	-0.38	-0.23	-0.18	-0.1
Total Δ : 95th percentile:	5.60	6.33	4.73	5.30	8.14	6.62	6.37	6.29	6.11	6.70	7.31
Comprised of: Postponement	2.90	3.48	2.87	3.86	6.40	4.08	3.13	4.09	2.72	4.23	3.90
Compression:	3.11	3.20	2.42	2.18	3.40	3.35	4.13	3.02	3.81	2.98	3.75
Model error:	-0.41	-0.35	-0.56	-0.75	-1.66	-0.81	-0.89	-0.82	-0.42	-0.51	-0.3

Table 5: Disaggregation due to compression, postponement and model error (other) of changes in various metrics of the distribution of the age at death conditional on reaching age 50, between 1959 and 2019, males and females in various countries

Panel A: Males	Percentile of age-at-death distribution										
	50	55	60	65	70	75	80	85	90	95	GMA
1959	6.41	7.24	8.02	8.80	9.53	10.26	11.00	11.78	12.66	13.80	17.66
1969	6.33	7.13	7.90	8.67	9.42	10.15	10.93	11.73	12.64	13.82	17.55
1979	7.47	8.23	8.99	9.73	10.41	11.09	11.80	12.55	13.41	14.51	17.97
1989	8.22	8.95	9.64	10.29	10.92	11.55	12.20	12.90	13.69	14.72	17.82
1999	9.10	9.77	10.38	10.96	11.52	12.08	12.66	13.27	13.97	14.89	17.85
2009	10.18	10.80	11.37	11.92	12.44	12.96	13.49	14.05	14.66	15.47	18.85
2019	10.49	11.13	11.72	12.27	12.79	13.29	13.80	14.34	14.93	15.71	19.00

Panel B: Females	Percentile of age-at-death distribution										
	50	55	60	65	70	75	80	85	90	95	GMA
1959	9.20	9.81	10.42	10.99	11.54	12.09	12.67	13.29	13.99	14.94	18.02
1969	9.63	10.25	10.84	11.39	11.92	12.46	13.01	13.60	14.29	15.20	18.32
1979	10.68	11.26	11.81	12.33	12.84	13.36	13.89	14.46	15.10	15.95	18.95
1989	10.89	11.47	12.02	12.55	13.05	13.55	14.06	14.60	15.22	16.02	18.75
1999	11.08	11.63	12.15	12.64	13.11	13.59	14.07	14.58	15.17	15.93	18.41
2009	11.76	12.28	12.76	13.22	13.66	14.10	14.55	15.03	15.58	16.31	19.08
2019	12.07	12.57	13.03	13.47	13.89	14.31	14.74	15.20	15.73	16.43	19.14

Table 6: Savings target at retirement age (expressed as a proportion of annual consumption) needed to ensure that with SS replacing 30% of consumption, and retirement at age 62, the individual will have enough resources to maintain that real level of consumption until each percentile of length of life, conditional on reaching age 50. Real interest rates are 2.5% p.a.. US population mortality in each year. Figures in the table can be adjusted to account for different SS replacement rates or defined benefit pension or annuity income by multiplying by $(1 - RR_{SS,DB})/0.7$. If savings are held primarily in pre-tax accounts, these targets should be adjusted by a factor of $1/(1 - \tau)$, where τ is the individual's average tax rate in retirement.

Panel A: Males			Percentile of age-at-death distribution								
	50	55	60	65	70	75	80	85	90	95	GMA
1959	3.58	4.51	5.39	6.28	7.10	7.92	8.77	9.65	10.65	11.93	16.30
1969	3.48	4.39	5.26	6.13	6.98	7.81	8.69	9.59	10.62	11.96	16.18
1979	4.77	5.64	6.49	7.32	8.09	8.87	9.67	10.52	11.49	12.74	16.66
1989	5.62	6.45	7.23	7.96	8.67	9.39	10.13	10.92	11.81	12.98	16.49
1999	6.62	7.37	8.06	8.72	9.36	9.99	10.65	11.34	12.13	13.17	16.52
2009	7.84	8.54	9.19	9.80	10.40	10.99	11.58	12.21	12.91	13.83	17.65
2019	8.19	8.91	9.58	10.20	10.79	11.36	11.94	12.54	13.22	14.09	17.82

Panel B: Females			Percentile of age-at-death distribution								
	50	55	60	65	70	75	80	85	90	95	GMA
1959	6.73	7.42	8.11	8.76	9.38	10.00	10.65	11.35	12.14	13.23	16.71
1969	7.21	7.91	8.58	9.20	9.81	10.41	11.04	11.71	12.49	13.52	17.05
1979	8.41	9.07	9.68	10.27	10.84	11.44	12.03	12.68	13.41	14.36	17.76
1989	8.65	9.30	9.92	10.52	11.09	11.65	12.22	12.84	13.54	14.45	17.53
1999	8.85	9.48	10.07	10.62	11.16	11.69	12.24	12.82	13.49	14.35	17.15
2009	9.63	10.21	10.75	11.27	11.77	12.28	12.79	13.33	13.95	14.78	17.91
2019	9.98	10.55	11.07	11.56	12.03	12.51	13.00	13.52	14.12	14.91	17.98

Table 7: Savings target at retirement age (expressed as a proportion of annual consumption) needed to ensure that with SS replacing 30% of consumption, and retirement at age 67, the individual will have enough resources to maintain that real level of consumption until each percentile of length of life, conditional on reaching age 50. Real interest rates are 2.5% p.a.. US population mortality in each year. Figures in the table can be adjusted to account for different SS replacement rates or defined benefit pension or annuity income by multiplying by $(1 - RR_{SS,DB})/0.7$. If savings are held primarily in pre-tax accounts, these targets should be adjusted by a factor of $1/(1 - \tau)$, where τ is the individual's average tax rate in retirement.

Panel A: Males			Percentile of age-at-death distribution								
	50	55	60	65	70	75	80	85	90	95	GMA
1959	1.70	2.70	3.65	4.61	5.50	6.38	7.29	8.24	9.31	10.70	15.40
1969	1.60	2.57	3.51	4.45	5.36	6.26	7.20	8.17	9.29	10.72	15.27
1979	2.98	3.92	4.84	5.73	6.56	7.40	8.26	9.17	10.22	11.57	15.78
1989	3.90	4.80	5.63	6.42	7.19	7.96	8.76	9.61	10.57	11.82	15.60
1999	4.98	5.79	6.53	7.24	7.92	8.60	9.31	10.06	10.91	12.03	15.63
2009	6.29	7.04	7.74	8.40	9.04	9.68	10.32	11.00	11.75	12.74	16.85
2019	6.67	7.45	8.16	8.83	9.47	10.08	10.70	11.35	12.08	13.03	17.04

Panel B: Females			Percentile of age-at-death distribution								
	50	55	60	65	70	75	80	85	90	95	GMA
1959	5.10	5.84	6.58	7.28	7.95	8.62	9.32	10.07	10.92	12.09	15.84
1969	5.61	6.37	7.09	7.76	8.41	9.06	9.74	10.46	11.29	12.40	16.21
1979	6.90	7.61	8.27	8.91	9.53	10.16	10.80	11.50	12.29	13.31	16.97
1989	7.16	7.86	8.53	9.17	9.79	10.39	11.01	11.67	12.43	13.40	16.73
1999	7.38	8.05	8.69	9.28	9.86	10.44	11.03	11.65	12.37	13.30	16.32
2009	8.22	8.84	9.43	9.99	10.52	11.07	11.62	12.20	12.87	13.76	17.13
2019.00	8.60	9.21	9.76	10.29	10.81	11.32	11.85	12.41	13.05	13.90	17.21

Table 8: Savings target at retirement age (expressed as a proportion of annual consumption) needed to ensure that with SS replacing 30% of consumption, and retirement at age 70, the individual will have enough resources to maintain that real level of consumption until each percentile of length of life, conditional on reaching age 50. Real interest rates are 2.5% p.a.. US population mortality in each year. Figures in the table can be adjusted to account for different SS replacement rates or defined benefit pension or annuity income by multiplying by $(1 - RR_{SS,DB})/0.7$. If savings are held primarily in pre-tax accounts, these targets should be adjusted by a factor of $1/(1 - \tau)$, where τ is the individual's average tax rate in retirement.

9 Figures

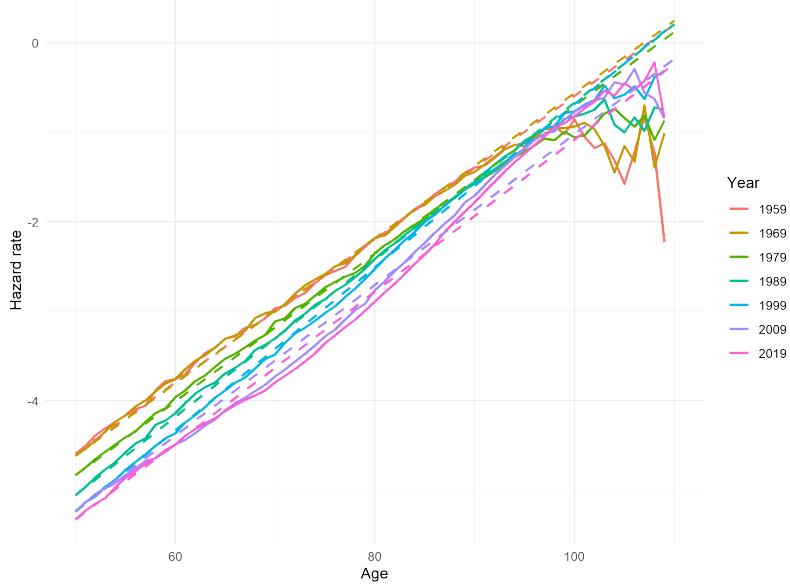


Figure 4: Empirical hazard rates and fitted Gompertz model, US males 1959-2019

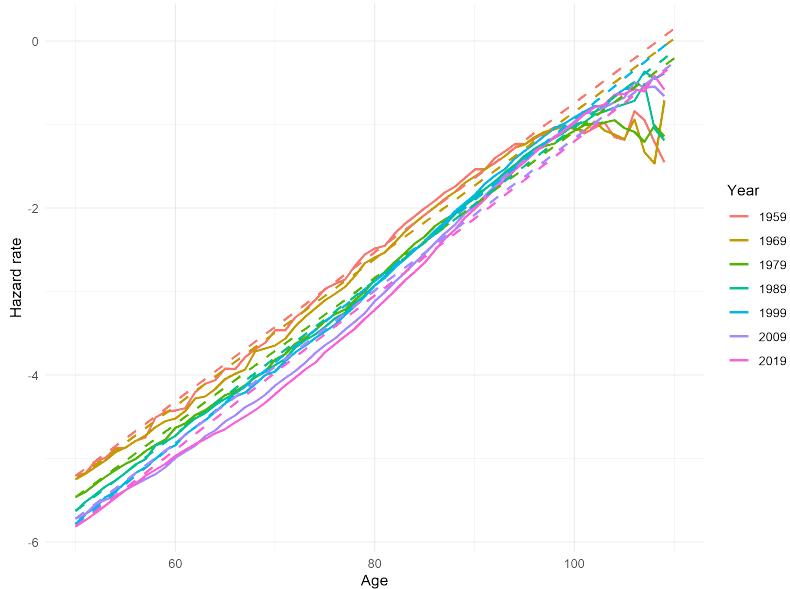


Figure 5: Empirical hazard rates and fitted Gompertz model, US females 1959-2019

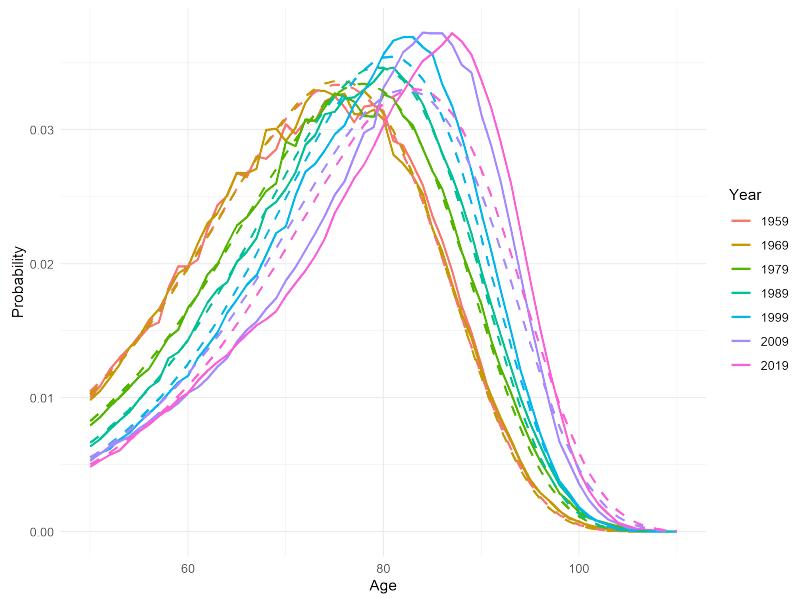


Figure 6: Probability distribution of age at death and fitted Gompertz model, US males 1959-2019

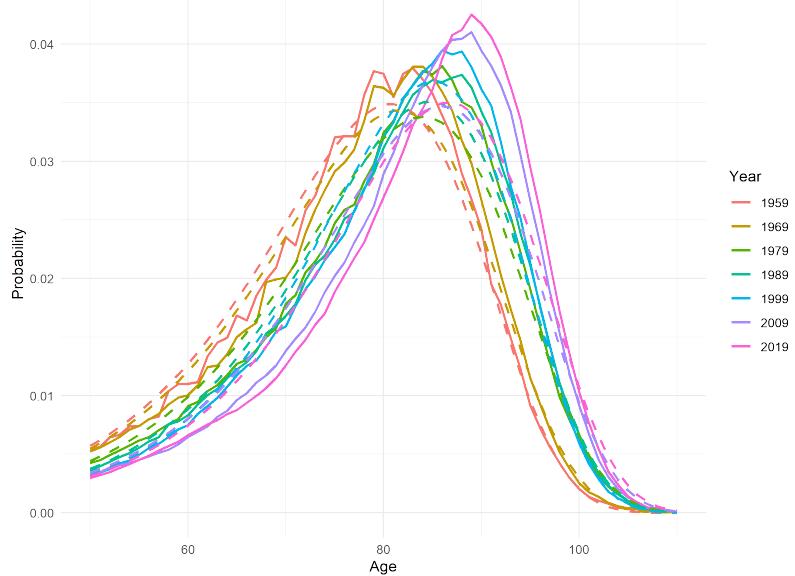


Figure 7: Probability distribution of age at death and fitted Gompertz model, US females 1959-2019

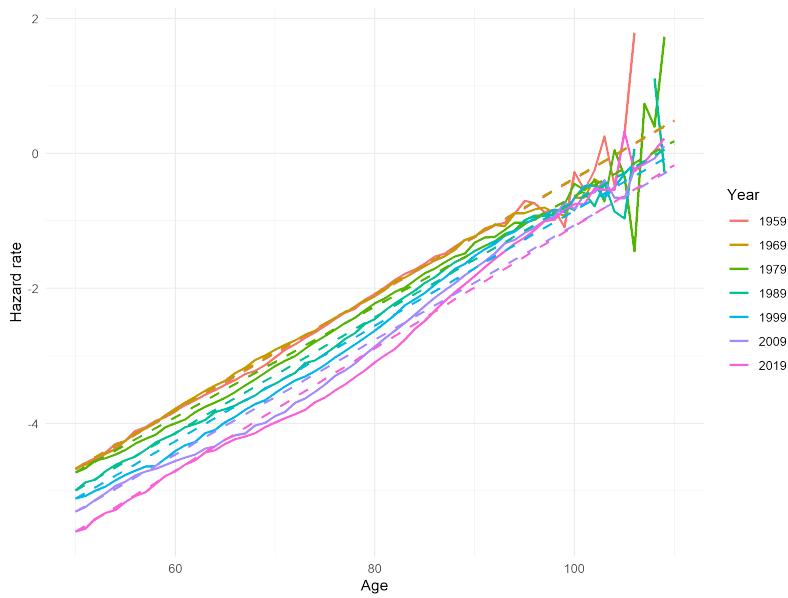


Figure 8: Empirical hazard rates and fitted Gompertz model, French males 1959-2019

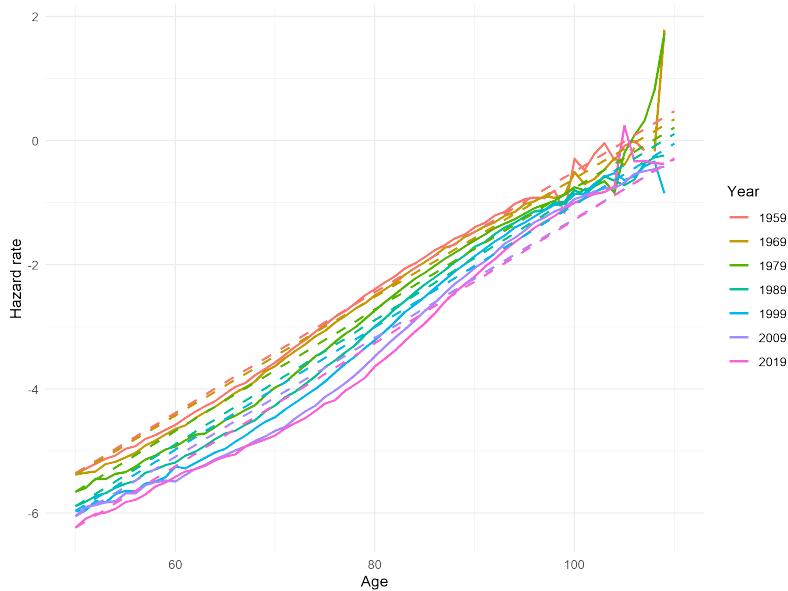


Figure 9: Empirical hazard rates and fitted Gompertz model, French females 1959-2019

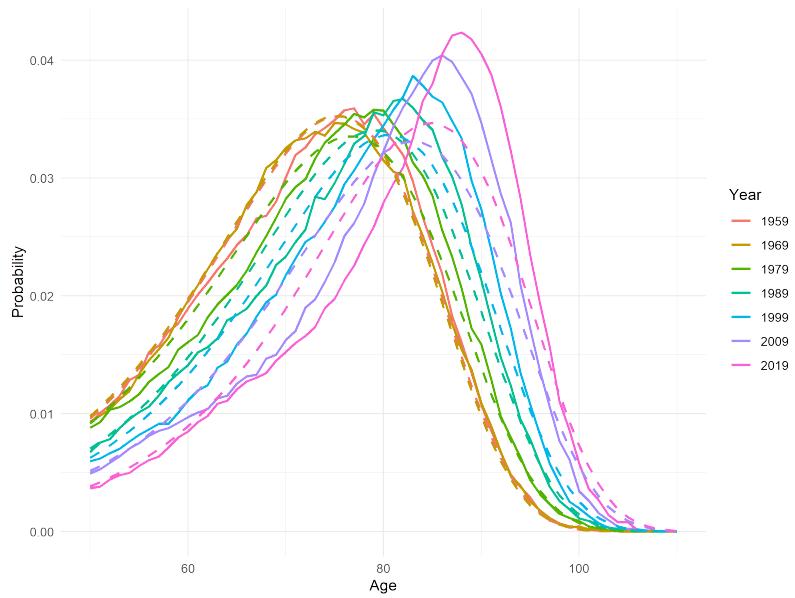


Figure 10: Probability distribution of age at death and fitted Gompertz model, French males 1959-2019

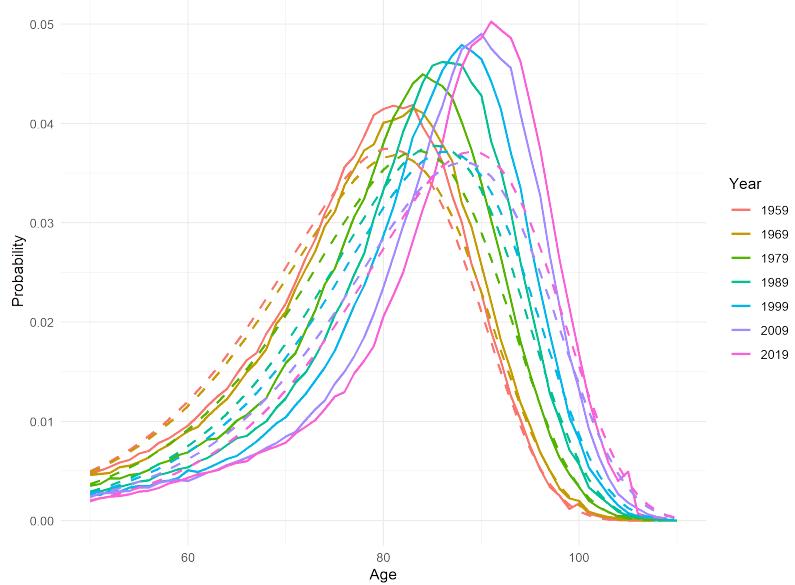


Figure 11: Probability distribution of age at death and fitted Gompertz model, French females 1959-2019

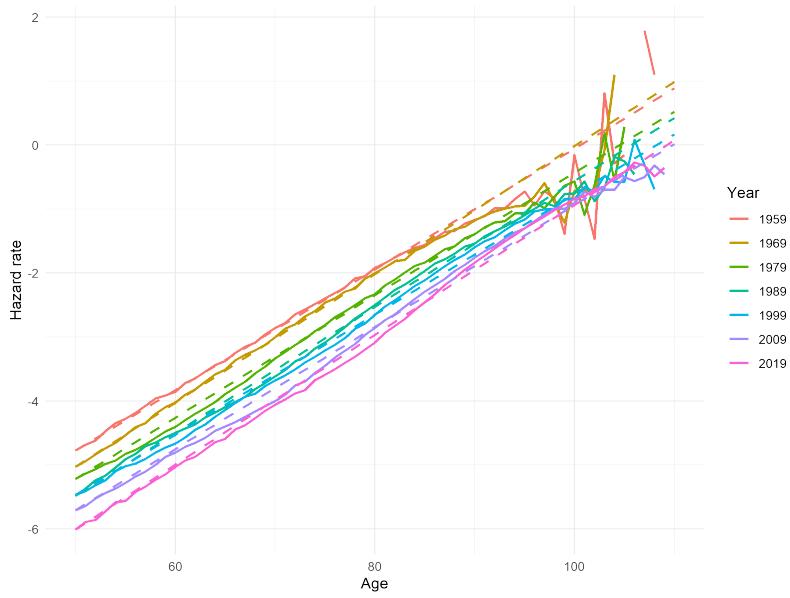


Figure 12: Empirical hazard rates and fitted Gompertz model, Japanese males 1959-2019

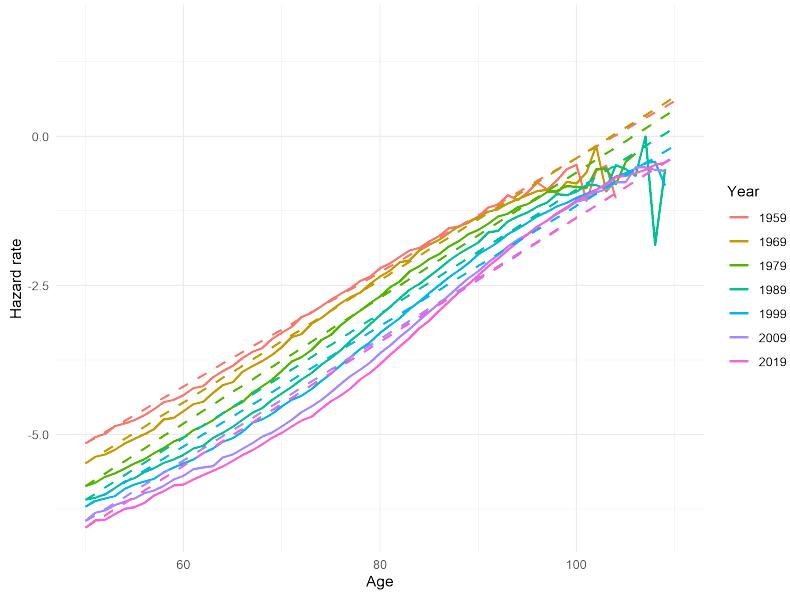


Figure 13: Empirical hazard rates and fitted Gompertz model, Japanese females 1959-2019

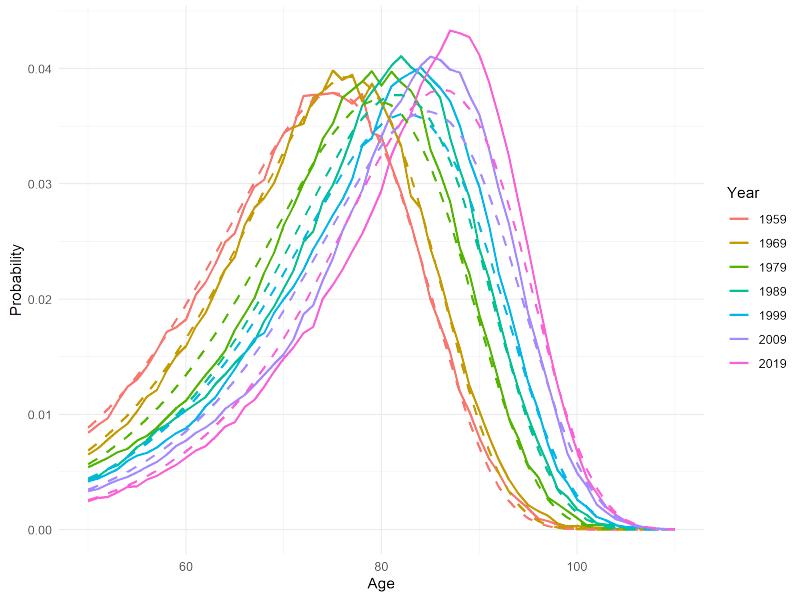


Figure 14: Probability distribution of age at death and fitted Gompertz model, Japanese males 1959-2019

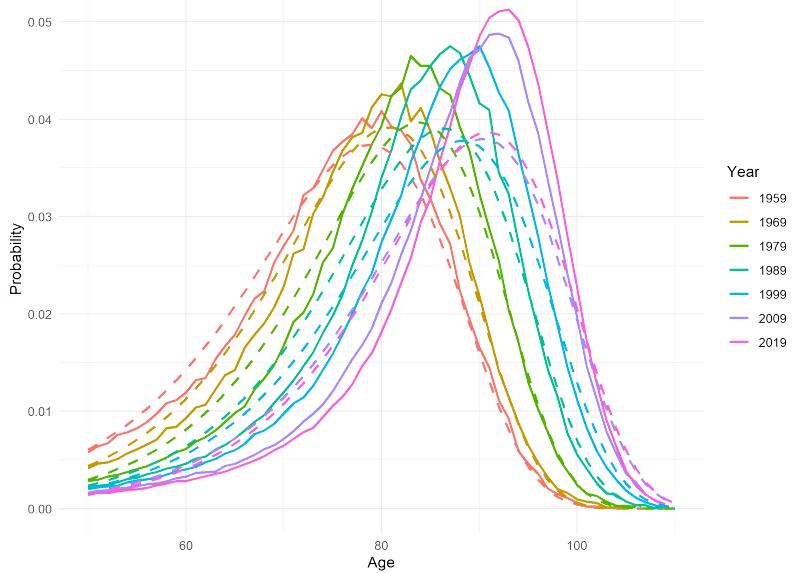


Figure 15: Probability distribution of age at death and fitted Gompertz model, Japanese females 1959-2019

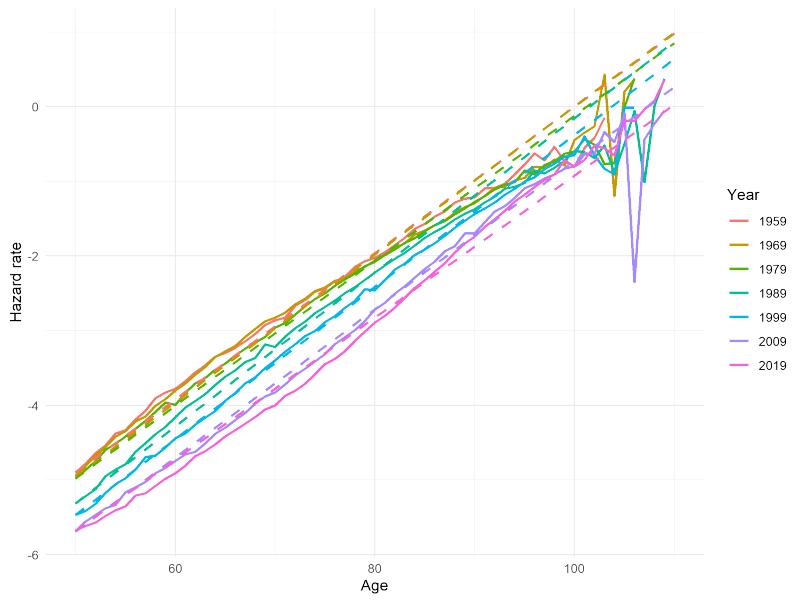


Figure 16: Empirical hazard rates and fitted Gompertz model, UK males 1959-2019

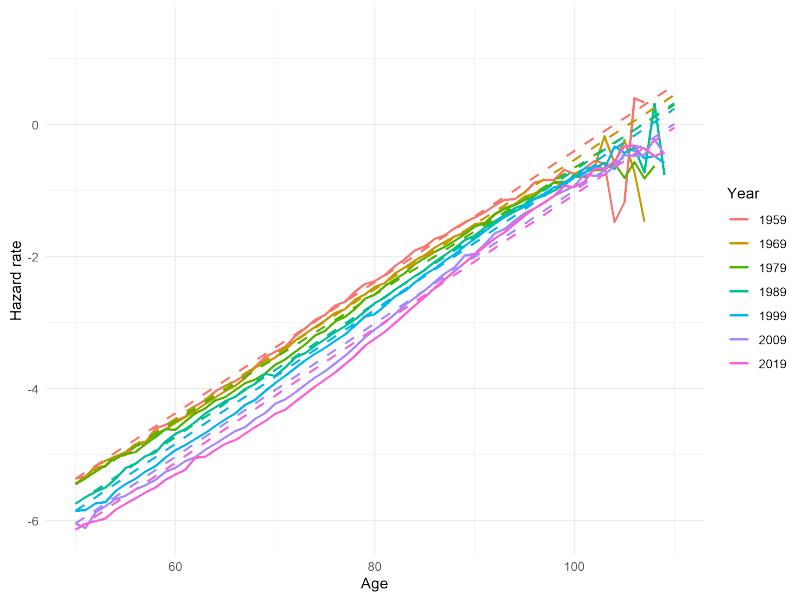


Figure 17: Empirical hazard rates and fitted Gompertz model, UK females 1959-2019

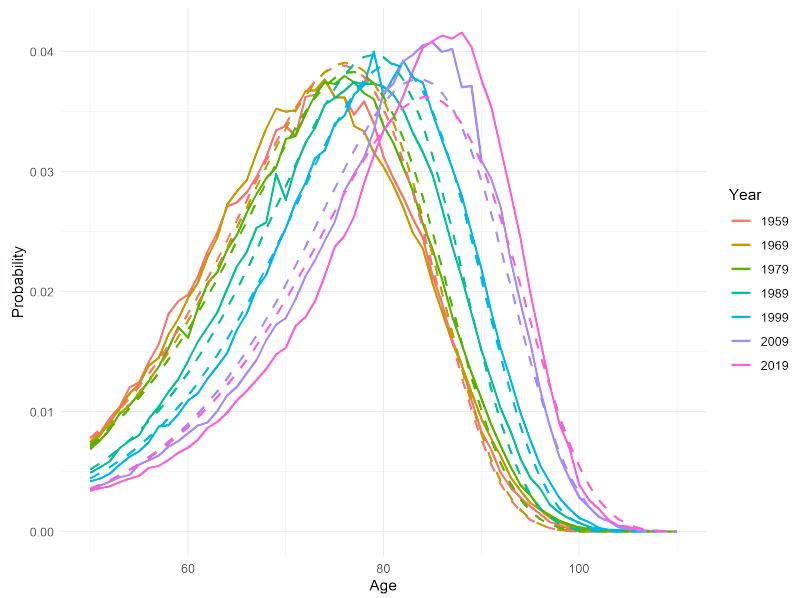


Figure 18: Probability distribution of age at death and fitted Gompertz model, UK males 1959-2019

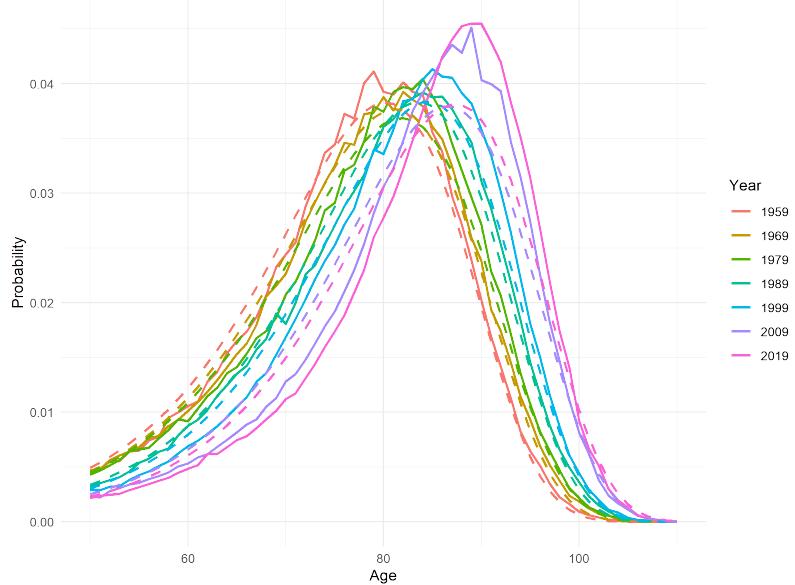


Figure 19: Probability distribution of age at death and fitted Gompertz model, UK females 1959-2019