# Dynamic Shifts in Mortality Patterns at Extreme Ages

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**For over a century, researchers have asked whether mortality at very old ages continues to rise without limit or slows and levels off into a plateau. Using cohort data from Denmark, France, Italy, and Sweden, we find that the ages of mortality deceleration and plateau onset shift steadily later for cohorts born between the mid-19th and early-20th centuries. In the most recent cohorts, deceleration begins only near age 100, with plateaus reached after 108 in some populations. These shifts help explain why past studies reach conflicting conclusions: they are observing different cohorts at different stages of this moving process. Our findings show that mortality at extreme ages has not been tied to a fixed age, but has been progressively postponed across successive cohorts.**

The number of centenarians worldwide is projected to increase from over half a million in 2024 to more than three million within three decades (*1*). As more people survive into such advanced ages, understanding how mortality behaves at the oldest ages is essential for anticipating the health and social challenges of an aging world.

For decades, studies on extreme age mortality have reached diverging conclusions on whether death rates at very old ages continue to rise, slow down, or level-off. Many find evidence for mortality deceleration at late ages (*2–5*), with some finding also that death rates eventually reach a plateau (*5–10*). Analyses of high-quality data from Italy (*9*) and from several European and North American populations (*11*) support this view. Others, however, reject the deceleration hypothesis (*12–14*), attributing apparent deceleration to errors in reporting or statistical artifacts (*15–17*). A recent study of French cohorts, for example, found no evidence of a plateau (*18*).

We hypothesize that the entire late-life mortality schedule—from the first deceleration of death rates to the onset of the plateau—has been progressively postponed across cohorts. These thresholds do not occur at fixed ages but instead shift upward as deaths are delayed and longevity rises (*19–22*). If this is the case, then the apparent contradictions in studies would naturally appear inconsistent: studies of earlier cohorts may have observed mortality still in its slowing phase, while studies of later cohorts captured mortality that had already leveled off into a plateau.

We test this hypothesis using high-quality cohort mortality data from Denmark, France, Italy, and Sweden, spanning from the mid-19th to the early-20th century. By applying a two-step Bayesian approach, we reconstruct mortality trajectories beyond age 100 and track three features of the late-life schedule: the age of mortality deceleration, the onset age of the mortality plateau and its level.

We find that the ages of both mortality deceleration and plateau onset have shifted to later ages. For some cohorts born after 1900, mortality deceleration begins near age 100, and the plateau is reached only after age 108.

# Data and Methods

We analyze raw cohort mortality data from Denmark, France, Italy, and Sweden, drawn from the Human Mortality Database (*23*). These countries provide high-quality population registers and long time series, offering a solid basis for studying mortality dynamics at advanced ages. The data include age-specific death counts and exposures after age 40 for both sexes, covering cohorts born from the mid-19th to the early-20th century.

To study mortality, we use the gamma–Gompertz–Makeham (ΓGM) model, a flexible framework that captures the exponential rise of mortality at adult ages as well as its deceleration and flattening at the oldest ages (*24*). We estimate the model in a Bayesian setting, with priors that aid identifying whether heterogeneity—and therefore deceleration and plateauing—is supported by the data (*25*). Posterior samples were drawn with Hamiltonian Monte Carlo, using the No-U-Turn Sampler implemented in RStan (*26*).

We then combine the ΓGM model with regression trees. The ΓGM provides smooth estimates of the overall mortality curve, while the regression tree serves as a non-parametric diagnostic tool to identify change points where the level of log mortality shifts significantly, without assuming in advance where those changes should occur. The final change in its level is taken as the onset of the plateau, and the predicted value beyond this point as its level.

To ensure robust estimates, we rely on ensemble methods, drawing repeatedly from the posterior distribution of the ΓGM model. This allows us to quantify uncertainty in all three outcomes—the age of deceleration, the age of the plateau, and its level. Full methodological details are provided in the Supplementary Materials (*27*).

# Results

Between the mid-19th and early-20th centuries, mortality slows down and levels off at increasingly higher ages across cohorts in Sweden, Denmark, France, and Italy. Females consistently reach these thresholds later than males. Our analysis highlights three key aspects of this shift: (i) the postponement of deceleration and plateau ages, (ii) the gap between these thresholds and the share of survivors reaching the slowing point, and (iii) the time trajectory of plateau level over cohorts.

### Postponement of Mortality Deceleration and Plateau

Figure 1 shows the ages at which mortality deceleration begins (left panels) and the mortality plateau is reached (right panels) for cohorts born between the mid-1800s and early-1900s in Sweden, Denmark, France, and Italy. In all countries and for both sexes, the onset of the mortality plateau shifts progressively to later across cohorts.

For mortality deceleration, postponement is observed in every population, but the pace differs by country. In Italy, the onset increases by 0.25 years per cohort for females and 0.23 for males. In France, the increases are 0.19 and 0.16. In Denmark, the rates are 0.08 for females and 0.05 for

males, while in Sweden they are smaller, at 0.02 for females and 0.07 for males. These small slopes for the Nordic countries suggest that the onset of mortality deceleration has already stabilized by the 1880 birth cohorts. Full estimates with standard errors, confidence intervals, and p-values are provided in table S1.

The onset of the mortality plateau shows a similar pattern. France and Italy consistently display later plateau ages than Sweden and Denmark. For the Nordic countries, stabilization occurs around ages 107 for females and 105 for males in cohorts born after the mid-1890s. In France and Italy, stabilization occurred at after ages 107 for males and 108 for females.

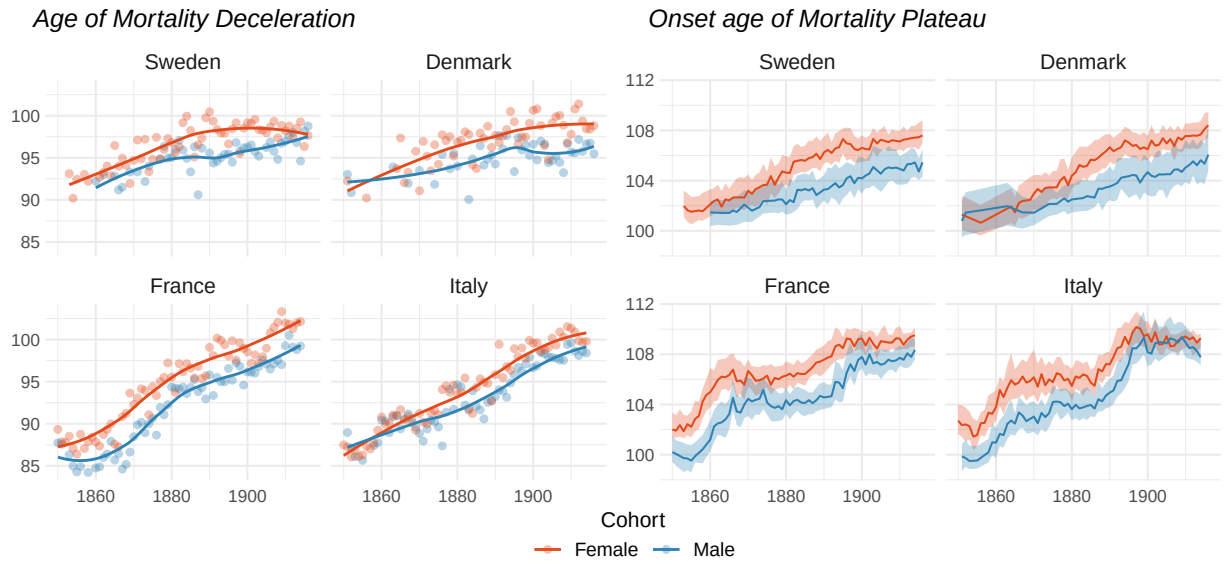


**Figure 1**: **Postponement of mortality deceleration and onset age of the plateau across cohorts.** The onset age of mortality deceleration (left panels) and the onset age of the mortality plateau (right panels) for cohorts born between the mid-1800s and early 1900s in Sweden, Denmark, France, and Italy. Female trajectories are shown in red and male trajectories in blue. Points indicate the estimates for individual birth cohorts, while solid lines represent the smoothed trends using Local Polynomial Regression. Shaded areas indicate 95% credible intervals.

## Gap between Deceleration and Plateau

The left panel of Figure 2 shows the interval between the onset of mortality deceleration and the start of the plateau. This interval can be understood as the duration of the late-life selection process. Frailer individuals are more likely to die earlier, so the group of people who survive to very old age is, on average, more robust and more similar in health (*4, 24*). This gap in years measures the time it takes for this filtering process to result in a highly selected group of survivors with a near-constant risk of dying after certain age.

In Sweden and Denmark, this interval was stable over cohorts. For both sexes, it remains close to 8 years, with only minor fluctuations. This stability mean that the two thresholds—the slowing down of mortality and the onset of the plateau—shifts upward together.

In France and Italy, by contrast, the interval shortens over cohorts. In Italy, older cohorts showed spans of around 15 years, however by the early 20th century, this has fallen to less than 10. France shows a similar pattern, most clearly for cohorts whose adult lives are exposed to the First and Second World Wars.

### Survival to the Age of Mortality Deceleration

The right panel of Figure 2 shows the proportion of individuals who survives to the age when mortality began to decelerate. In all four countries, this share declines over cohorts born up to about 1890, and then levels off. The stabilization suggests that by the late 19th century, the proportion reaching this age has become broadly constant.

The estimated slopes of the log-survival series are consistent with the described results. In Denmark, the share surviving to the age of deceleration increases on average by 3.57% per cohort for females (s.e. 2.37) and 1.97% for males (s.e. 1.76). In other words, each successive female cohort has, on average, 3.57% more survivors reaching this threshold. For France, the estimates are –0.89% (s.e. 1.88) for females and 0.21% (s.e. 1.51) for males. In Italy, they are 1.03% (s.e. 1.26) for females and –0.48% (s.e. 1.20) for males. In Sweden, the slopes are 4.11% (s.e. 1.28) for females and –0.25% (s.e. 1.32) for males. Despite differences in sign and size, most slopes are small relative to their standard errors, suggesting no statistical evidence of any cross-cohort trend after the 1890s. Full estimates with standard errors, confidence intervals, and p-values are provided in table S2.

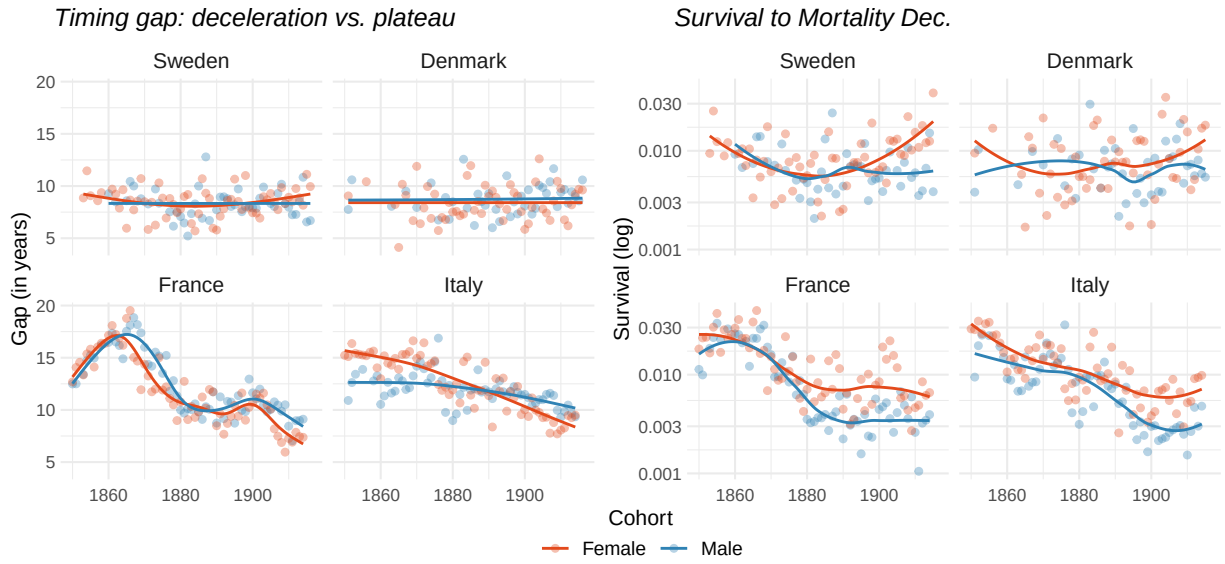


**Figure 2**: **Gap between deceleration and plateau, and survivorship to the age of mortality deceleration.** The left panels show the gap (in years) between the age of mortality deceleration and the onset of the mortality plateau for cohorts born between the mid-1800s and early 1900s in Sweden, Denmark, France, and Italy. The right panels show the proportion surviving to the age of mortality deceleration on a logarithmic scale. Female trajectories are shown in red and male trajectories in blue. Points indicate the estimates for individual birth cohorts, while solid lines represent the smoothed trends using Local Polynomial Regression.

### Cohort Variation in the Plateau Level

Figure 3 shows how the level of the mortality plateau varies across cohorts. In Sweden and Denmark, plateau levels remains largely constant across most cohorts, with only a slight upward trend observed for the most recent ones—particularly for females in Denmark and for both sexes in Sweden.

In contrast, France and Italy show higher cohort-to-cohort fluctuation. These countries display a wave-like pattern: plateau levels fall for cohorts born in the mid-1850s, rise for those born from

the mid-1860s to a peak around 1885, declines again for cohorts born in the late 1880s, and then begins to ries steadily from the 1890s onward. Once again, these patterns correspond to cohorts exposed to the First and Second World Wars, as well as the Spanish flu. In more recent cohorts, female plateau levels tend to be higher than those for males.

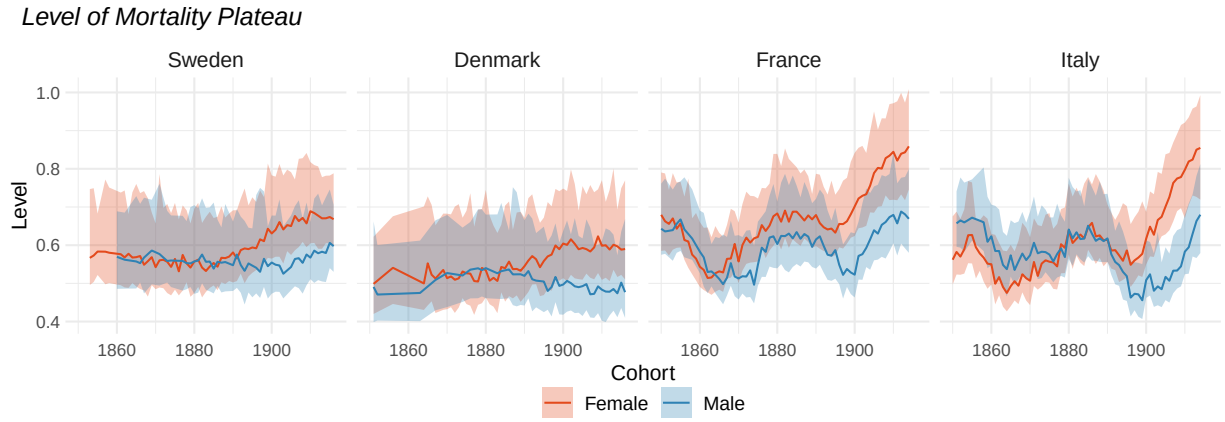


**Figure 3**: **Cohort variation in the level of the mortality plateau.** The figure shows the level of the mortality plateau for cohorts born between the mid-1800s and early 1900s in Denmark, France, Italy, and Sweden. Female trajectories are shown in red and male trajectories in blue, with shaded areas indicating 95% credible intervals. solid lines indicate the estimates for individual birth cohorts.

# Discussion

Our results support the hypothesis that mortality deceleration and the plateau have been progressively postponed across cohorts. Both thresholds shifts upward between the mid-19th and early-20th centuries. This aids understanding why earlier studies often reached different conclusions: rather than contradicting one another, they observe most likely cohorts at different stages of the same shifting process.

Other studies have shown that beyond ages 108–110, mortality risk tends to flatten into a plateau (*8, 10*). Our findings are consistent with this evidence but add another dimension: the onset of the plateau has itself moved upward across successive cohorts. For cohorts born after 1900 in France and Italy, we estimate plateau ages to be around 109 years, in line with these 108–110 thresholds (*8, 10*). In doing so, we provide direct support for the hypothesis that the mortality plateau is reached later for later cohorts (*28*). Apparent inconsistencies in earlier studies can thus be understood as a matter of timing: some cohorts were still in the phase of mortality deceleration, while others had already reached the plateau.

What earlier studies sometimes identify as inconsistencies may reflect analyses conducted at different ages along this shifting schedule.

### Frailty, Heterogeneity, and Stability

These patterns align with the theory of frailty. In any cohort, frailer individuals tend to die first, leaving behind survivors who are more robust (*24*). This process explains why at the population level mortality decelerates at very old ages and why, for the most selected survivors, it levels off.

The interval between deceleration and the plateau can be interpreted as the length of this final compression process (*4*).

In Sweden and Denmark, this interval has been remarkably stable—around a decade—with plateau levels also steady. Once large period shocks are set aside, late-life mortality in these populations seems to follow a consistent patten: mortality decelerates, selection plays out over about ten years, and a plateau is reached at a predictable level. In these settings, both the pace of aging and the degree of heterogeneity among survivors appear steady, even as the ages at which these processes begin shifted upward across cohorts.

### Historical Shocks and the Dynamics of Late Life

Cohorts born in the late 19th and early 20th centuries in France and Italy were exposed to the First World War, the Spanish flu, and later the Second World War. The shifts we observe in their late-life mortality are consistent with two competing mechanisms.

The first is *selective survival*: crises disproportionately remove frailer individuals at earlier ages, leaving behind a more robust and homogeneous group to reach very old age (*29, 30*). This shortens the gap between deceleration and plateau and lower the plateau level. The second is *long-term damage*: infection, malnutrition, stress, and health-system collapse during wars and pandemics leave lasting scars, raising frailty that persisted into later life (*31*). This would elevate plateau level and also shorten the gap.

Our results suggest that both forces were at play. In France and Italy, the gap narrowed for cohorts most exposed to crises, consistent with stronger early selection. Plateau levels, however, fluctuated—sometimes lower, sometimes higher—producing a wave-like pattern. This interplay of selection and scarring plausibly explains why late-life mortality in these countries shifted in irregular ways, while the Nordic countries, spared major disruption, remained steady.

### Who Reaches Late Life?

Across the countries we study, increases in longevity are accompanied mainly by a delay in the onset of very old age, rather than by a rise in the fraction of people who reached it. For cohorts born after 1890, the share surviving to the age of mortality deceleration remains broadly constant across countries, with no statistical evidence of a cross-cohort trend. This supports the view that mortality improvements shift the schedule of death to later ages without altering its overall shape (*20*). In this sense, survival gains are reflected more in when late-life processes occur than in how many individuals experience them.

Swedish women are a notable exception. After an initial decease, their survival to the age of deceleration began to increase in later cohorts. In Sweden—where neutrality in wartime and the sustained growth in public health and social protection provides unusually favorable conditions (*32*)—more women both reached late-life thresholds and do so at later ages. This pattern illustrates that, in certain contexts, improvements in health and living conditions can affect not only the timing of late-life mortality but also the share of the population that enters it.

### An Explanation for Conflicting Findings

A recent study of French cohorts born 1883–1901 concludes that while mortality decelerates at very old ages, it does not level off after age 105 (*18*). Our analysis suggests that for these cohorts, the plateau is not fixed but shifting to later ages, moving from about 106 to 109. By setting the threshold at 105, Dang et al. are effectively tracing part of the ongoing deceleration process rather than the plateau itself. This explains their estimated Gompertz slope of 0.062—about half the 0.11 typical at earlier adult ages (*33*).

In this light, their results are not evidence against a plateau but a reflection of its moving onset. Our findings are in line with other analyses of French data that identify a constant force of mortality after age 108 (*28*). This suggests that the plateau only becomes visible at ages above 108, while analyses starting at lower ages are likely to capture a mix of the slowing-down phase and the plateau itself (*10*).

A similar issue of threshold selection helps to interpret the findings for Italian cohorts by Barbi et al., who report a mortality plateau beginning at age 105 alongside a significant downward trend in its level across cohorts (*9*). The reason a plateau appears at this early age is that their model averages the mortality patterns of cohorts with different dynamics. A more detailed analysis of the same data provides evidences that for cohorts born before 1905, mortality is indeed constant from age 105; however, for later cohorts born after 1905, mortality was significantly lower in the 105–107 age range, suggesting that their plateau begin later (*28*). Therefore, what is modeled by Barbi et al. as a cohort trend in the level of the plateau is most likely the statistical signal of the cross-cohort postponement of the plateau's onset, which captures the ongoing deceleration of the younger cohorts.

### Implications and Next Steps

The evidence suggests that late-life mortality is not fixed. In Sweden and Denmark, it follows a stable course, with regular gaps and steady plateau levels. In France and Italy, wars and pandemics leave clear marks, shortening the interval between deceleration and plateau and producing fluctuations in plateau levels.

Our approach provides a way to detect these shifts, even when the data available at extreme ages is sparse. To strengthen the results, future work should combine such analyses with high-quality individual-level data on centenarians, such as those in the International Database on Longevity. It will also be important to separate more explicitly the roles of age, period, and cohort effects, to better distinguish long-term cohort trends from the lasting scars of historical crises.

## Conclusion

Our analysis shows that late-life mortality is not fixed at specific ages but has shifted upward across cohorts. Both the age when mortality begins to decelerate and the age when it reaches a plateau have been postponed, in some cases by nearly a decade. This aids explain why past studies reached contrasting conclusions: they were analyzing different cohorts at different stages of the late-life mortality schedule—some before the plateau had been reached, others after it had already begun. The debate over whether a plateau exists can thus be reframed, not as a question of presence or absence, but as recognition that its onset has moved across generations.

The overall pattern is one of stability in shape but delay in timing. In Sweden and Denmark, late-life mortality settles to a steady rhythm by the late 19th century. In France and Italy, by contrast, wars and pandemics leave lasting marks, shortening the interval between deceleration and plateau and producing fluctuating plateau levels.

As the number of centenarians worldwide is projected to increase sixfold within the next three decades (*1*), our results suggest that survival to such ages is no longer exceptional because people are more robust, but because the threshold of exceptionality has shifted. Extreme old age has not been redefined, only rescheduled.

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**Author contributions:** Conceptualization, Methodology, Visualization: S.C.P.; Project administration, Supervision: T.I.M.; Writing – review & editing, Writing – original draft: T.I.M. and S.C.P.

**Competing interests:** There are no competing interests to declare.

**Data and materials availability:** All mortality and exposure data used in this study are publicly available through the Human Mortality Database (*23*). Additional materials, including code and analysis scripts, are available from the corresponding author upon request.

## Supplementary materials

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Supplementary Table
Tables S1 to S2
References *(34-45)*

# Supplementary Materials for Dynamic Shifts in Mortality Patterns at Extreme Ages

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## Materials and Methods

Our goal is to estimate the onset age and level of the mortality plateau, as well as the age at which mortality deceleration begins. To accomplish this, we integrate Bayesian estimation with machine learning techniques, specifically regression trees, and use resampling methods to capture uncertainty and variability in our estimates.

Let $D_x$ represent the number of deaths within the age range $[x, x+1)$ for $x = 0, \ldots, m$, and let $E_x$ denote the number of person-years at age $x$ exposed to the risk of dying (*34*). We define the vectors of deaths and exposures as $\boldsymbol{D} = (D_0, D_1, \ldots, D_m)^\top$ and $\boldsymbol{E} = (E_0, E_1, \ldots, E_m)^\top$, respectively. The parameter vector is given by $\boldsymbol{\theta} = (a, b, c, \gamma)^\top$, which defines the force of mortality at age $x$, denoted by $\mu(x; \boldsymbol{\theta})$. We assume that both the number of deaths and the number of person-years exposed are observed.

We model the force of mortality using the gamma-Gompertz-Makeham (ΓGM) model, defined as

$$\mu(x; \boldsymbol{\theta}) = \frac{a e^{bx}}{1 + \gamma \frac{a}{b} \left(e^{bx} - 1\right)} + c,$$

where $a > 0$, $b > 0$, $c \geq 0$, and $\gamma \geq 0$. This model captures different mortality patterns: when $\gamma = 0$, it reduces to the Gompertz-Makeham model, where mortality increases exponentially with age (*35, 36*); when $\gamma > 0$, it allows for mortality deceleration and eventual leveling-off, following a logistic trajectory (***?**, 24, 37*).

To estimate the parameters $\boldsymbol{\theta}$, we adopt a Bayesian framework, simulating $n$ lifetimes from the ΓGM distribution for each sampled $\boldsymbol{\theta}^{(i)}$, generating synthetic data: death counts $\boldsymbol{D}^{(i)}$, exposures $\boldsymbol{E}^{(i)}$, and ages $\boldsymbol{x}^{(i)} = (0, 1, \ldots, m^{(i)})^\top$.

We compute the logarithm of the mortality rate for each age $x^{(i)}$, $\log \mu_{x^{(i)}} = \log D_x^{(i)} - \log E_x^{(i)}$, and perform $M$ bootstrap iterations to capture uncertainty. In each iteration $j$, we bootstrap a sample from the simulated data, train a regression tree model $f^{(j)}(x)$ to predict $\log \mu_x$ based on age $x$ (*38*), and identify the terminal split node corresponding to the onset age of the mortality plateau, $x_{\text{onset}}^{(j)}$. The predicted mortality rate after $x_{\text{onset}}^{(j)}$ is taken as the level of the mortality plateau, $\mu_*^{(j)}$. After $M$ iterations, we compute the mode of the distributions of onset ages $\{x_{\text{onset}}^{(j)}\}$ and plateau levels $\{\mu_*^{(j)}\}$ to obtain the point estimates.

To estimate the age of mortality deceleration (AMD), corresponding to the inflection point in the mortality curve, we perform $K$ iterations: in each iteration, we randomly select a subset of the trained regression trees, aggregate their predictions to compute an averaged mortality function, and numerically estimate its inflection point $x_{\text{AMD}}^{(k)}$. We then compute the mode of the distribution of estimated AMDs $\{x_{\text{AMD}}^{(k)}\}$ to obtain the final estimate. Algorithm 1 summarizes the estimation procedure.

### Mitigating Data Sparsity and Biases in Mortality Estimation

Estimating mortality dynamics at extreme ages presents significant challenges due to data sparsity and potential biases, particularly when dealing with smaller cohorts. The inherently low number of observed deaths and exposures at advanced ages leads to high variability and less stable estimates (*10, 28*). This scarcity can result in overestimation or underestimation of key parameters, such as the onset ages of mortality deceleration and plateau.

**Algorithm 1:** Estimation of Onset Age and Level of Mortality Plateau, and Age of Mortality Deceleration

**Data:** Death counts $\boldsymbol{D}$, exposures $\boldsymbol{E}$, ages $\boldsymbol{x} = (0, 1, \ldots, m)^\top$
**Input:** $N$ (posterior samples), $n$ (simulated lifetimes), $M$ (bootstrap iterations), $K$ (bagging iterations)
**Output:** Estimates of onset age $x_{\text{onset}}$, plateau level $\mu_*$, and age of mortality deceleration $x_{\text{AMD}}$

**Bayesian Estimation:**
Define likelihood $L(\boldsymbol{\theta}; \boldsymbol{D}, \boldsymbol{E})$ and priors for $\boldsymbol{\theta} = (a, b, c, \gamma)^\top$.;
Draw $N$ samples $\{\boldsymbol{\theta}^{(i)}\}_{i=1}^{N}$ from posterior $p(\boldsymbol{\theta}, |, \boldsymbol{D}, \boldsymbol{E})$.;
**for** $i \leftarrow 1$ **to** $N$ **do**
    Simulate $n$ lifetimes from ΓGM model with $\boldsymbol{\theta}^{(i)}$.; Compute $\boldsymbol{D}^{(i)}$, $\boldsymbol{E}^{(i)}$, and $\boldsymbol{x}^{(i)}$.; Calculate $\log \mu_{x^{(i)}} = \log D_x^{(i)} - \log E_x^{(i)}$.;
    **for** $j \leftarrow 1$ **to** $M$ **do**
        Bootstrap sample from $\boldsymbol{x}^{(i)}$ and $\log \mu_{x^{(i)}}$.;
        Train regression tree $f^{(j)}(x)$.;
        Record terminal split age $x_{\text{onset}}^{(j)}$.;
        Record plateau level $\mu_*^{(j)}$.;
    **end**
    Set $x_{\text{onset}}^{(i)}$ as mode of $\{x_{\text{onset}}^{(j)}\}$.;
    Set $\mu_*^{(i)}$ as mode of $\{\mu_*^{(j)}\}$.;
    **for** $k \leftarrow 1$ **to** $K$ **do**
        Select subset of models.;
        Compute aggregated mortality function $g^{(k)}(x)$.;
        Estimate inflection point $x_{\text{AMD}}^{(k)}$.;
    **end**
    Set $x_{\text{AMD}}^{(i)}$ as mode of $\{x_{\text{AMD}}^{(k)}\}$.;
**end**
Compute distributions of $x_{\text{onset}}^{(i)}$, $\mu_*^{(i)}$, and $x_{\text{AMD}}^{(i)}$.;
Set $x_{\text{onset}}$ as mode of $\{x_{\text{onset}}^{(i)}\}$.;
Set $\mu_*$ as mode of $\{\mu_*^{(i)}\}$.;
Set $x_{\text{AMD}}$ as mode of $\{x_{\text{AMD}}^{(i)}\}$.;
**return** $x_{onset}$, $\mu_*$, $x_{AMD}$

To overcome these challenges, we employ a Bayesian estimation framework that incorporates prior information about mortality patterns, drawing on historical data and biological plausibility (*39*). By specifying informative priors, we stabilize parameter estimates in the presence of limited data, enhancing the robustness of our findings. Additionally, we use resampling techniques, including bootstrapping and bagging, to generate multiple pseudo-samples from the observed data. This approach allows us to assess variability, quantify uncertainty, and reduce the influence of anomalies that may disproportionately affect smaller cohorts (*40*).

Similar methodologies have been successfully applied in contexts with sparse data. For example, (*41*) used Bayesian smoothing to estimate age-specific mortality rates in small populations, while (*42*) employed Bayesian hierarchical models to pool information across similar populations and smooth estimates over time, creating more reliable mortality estimates in contexts with sparse data. These studies underscore the effectiveness of combining Bayesian inference with resampling to mitigate the challenges posed by data sparsity.

Despite these methodological safeguards, we acknowledge that estimates from smaller cohorts may still exhibit increased statistical uncertainty. The inherent variability in small sample sizes necessitates cautious interpretation of the estimated parameters. Future research with larger cohort data or the development of methods specifically tailored to handle small sample sizes could further enhance the reliability of mortality estimates at extreme ages.

By integrating Bayesian estimation with resampling techniques, our methodological framework provides a robust approach to estimating mortality dynamics under conditions of data sparsity. This ensures that our findings on mortality deceleration and plateaus are grounded in sound statistical practices, contributing valuable insights even in the face of limited data.

### Modeling Nonlinear Mortality Patterns

In our study, we employ regression trees to detect significant changes in the logarithm of mortality rates as a function of age. The tree-based method recursively splits the data at ages where there is a substantial change in mortality patterns, effectively identifying the onset ages of mortality deceleration and plateau without imposing linearity or specific parametric relationships (*43*). This approach is particularly advantageous for handling the nonlinearity and heterogeneity inherent in mortality data at extreme ages, avoiding reliance on high-order derivatives for estimation, which can be unstable and highly sensitive to variations in parameter estimates (*44, 45*).

By capturing interactions and nonlinear effects, regression trees can model sudden shifts or plateaus in mortality rates that traditional parametric models might overlook or misrepresent. This flexibility allows for a more accurate representation of the underlying mortality dynamics and accommodates variations across different populations and historical periods. Additionally, regression trees are robust to outliers and can handle sparse data effectively, which is essential when analysing mortality at advanced ages where data points are limited.

Overall, the use of regression trees enhances our ability to uncover complex mortality patterns and provides a powerful tool for detecting nonlinearity in mortality dynamics that traditional methods may fail to capture.

### Model tuning

The results presented in this paper were obtained using methodological parameters chosen to balance computational efficiency and accuracy. We set the number of posterior samples to $N = 200$, each with $n = 20,000$ simulated lifetimes, and used $M = 250$ bootstrap iterations and $K = 150$ bagging iterations, with a burn-in of 10,000 for the Bayesian sampler, to capture uncertainty and ensure stability.

## Supplementary Tables

**Table S1**: **Linear regression estimates of yearly change in the onset age of mortality deceleration.** Estimates are shown by country and sex. Reported values are intercepts and slopes with standard errors, 95% confidence intervals, and p-values.

| Country | Sex | Term | Estimate | Std. error | 95% CI | p-value |
|---|---|---|---|---|---|---|
| Denmark | Female | Intercept | 96.73 | 0.5155 | [95.69, 97.78] | <0.0001 |
| | | Slope | 0.0766 | 0.0246 | [0.0266, 0.1266] | 0.0037 |
| | Male | Intercept | 94.54 | 0.5623 | [93.39, 95.68] | <0.0001 |
| | | Slope | 0.0475 | 0.0256 | [-0.0046, 0.0997] | 0.0724 |
| France | Female | Intercept | 95.44 | 0.4473 | [94.53, 96.35] | <0.0001 |
| | | Slope | 0.1930 | 0.0226 | [0.1469, 0.2390] | <0.0001 |
| | Male | Intercept | 93.29 | 0.3067 | [92.67, 93.92] | <0.0001 |
| | | Slope | 0.1613 | 0.0155 | [0.1297, 0.1929] | <0.0001 |
| Italy | Female | Intercept | 93.35 | 0.4594 | [92.41, 94.28] | <0.0001 |
| | | Slope | 0.2482 | 0.0232 | [0.2009, 0.2955] | <0.0001 |
| | Male | Intercept | 91.89 | 0.4083 | [91.06, 92.72] | <0.0001 |
| | | Slope | 0.2270 | 0.0207 | [0.1849, 0.2690] | <0.0001 |
| Sweden | Female | Intercept | 97.73 | 0.4181 | [96.88, 98.58] | <0.0001 |
| | | Slope | 0.0211 | 0.0200 | [-0.0195, 0.0617] | 0.2984 |
| | Male | Intercept | 94.61 | 0.4274 | [93.74, 95.48] | <0.0001 |
| | | Slope | 0.0673 | 0.0203 | [0.0260, 0.1086] | 0.0022 |

**Table S2**: **Linear regression estimates of yearly change in survivorship to the age of mortality deceleration.** Estimates are shown by country and sex. Reported values are intercepts and slopes (log scale) with standard errors, 95% confidence intervals, and p-values.

| Country | Sex | Term | Estimate | Std. error | 95% CI | p-value |
|---|---|---|---|---|---|---|
| Denmark | Female | Intercept | -5.179 | 0.3045 | [-5.812, -4.545] | <0.0001 |
| | | Slope (log) | 0.0357 | 0.0237 | [-0.0136, 0.0850] | 0.1473 |
| | Male | Intercept | -5.262 | 0.2310 | [-5.744, -4.780] | <0.0001 |
| | | Slope (log) | 0.0197 | 0.0176 | [-0.0170, 0.0564] | 0.2760 |
| France | Female | Intercept | -4.829 | 0.2310 | [-5.310, -4.347] | <0.0001 |
| | | Slope (log) | -0.0089 | 0.0188 | [-0.0482, 0.0304] | 0.6422 |
| | Male | Intercept | -5.682 | 0.1843 | [-6.067, -5.298] | <0.0001 |
| | | Slope (log) | 0.0022 | 0.0150 | [-0.0292, 0.0335] | 0.8874 |
| Italy | Female | Intercept | -5.234 | 0.1546 | [-5.556, -4.911] | <0.0001 |
| | | Slope (log) | 0.0103 | 0.0126 | [-0.0160, 0.0366] | 0.4241 |
| | Male | Intercept | -5.803 | 0.1472 | [-6.110, -5.496] | <0.0001 |
| | | Slope (log) | -0.0048 | 0.0120 | [-0.0299, 0.0202] | 0.6911 |
| Sweden | Female | Intercept | -4.980 | 0.1644 | [-5.322, -4.638] | <0.0001 |
| | | Slope (log) | 0.0410 | 0.0128 | [0.0144, 0.0676] | 0.0043 |
| | Male | Intercept | -5.078 | 0.1701 | [-5.432, -4.723] | <0.0001 |
| | | Slope (log) | -0.0025 | 0.0132 | [-0.0300, 0.0249] | 0.8501 |