STRUCTURAL BREAKS IN ITALIAN AGE-SPECIFIC MORTALITY TRENDS: ASSESSING THE COHORT-EFFECT HYPOTHESIS[1]

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Abstract. Using Human Mortality Database cohort life-tables for Italy, we reconstructed calendar-year log-mortality rates for five-year age groups between 50–54 and 85–89 from 1980 to 2019. An unconstrained segmented-regression search located one or two structural breaks per series; almost all fell within 2008-2010. Treating 2008 as a common breakpoint, basic interrupted time-series models then estimated separate slopes before and after that year. The post-2008 slope is significantly flatter for essentially every age group, implying that annual improvements in age-specific mortality slowed by roughly 0.6 % to 2.2%. Men experience the sharper early-old-age slowdown, but the effect tapers after their mid-sixties; among women it grows with age and peaks near 80. The near-synchronous timing of the breaks across cohorts, their consistency across sexes, and their alignment with the onset of Italian health-care austerity are difficult to reconcile with a cohort-driven explanation. Instead, they point to a period effect that dampened mortality gains almost overnight.

1. Introduction

Life expectancy in high-income nations has followed an almost uninterrupted upward trajectory for well over a century, driven by remarkable improvements in infant mortality, nutrition, living standards, and the control of infectious diseases, as well as by subsequent improvements in chronic-disease prevention and treatment (Riley, 2001; Cutler *et al*., 2006).

Yet this secular rise has shown clear signs of deceleration since about 2011 across most OECD members (Raleigh, 2018). This is a major phenomenon since, with the exception of the recent COVID-19 pandemic, this secular rise in life expectancy had previously been temporarily interrupted only during the two World Wars and the 1918 flu pandemic.

Recent literature has focused on the causes of death most connected to this process. Life expectancy decompositions indicate that the slowdown is concentrated in the waning pace of mortality reductions from cardiovascular disease and several

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common cancers (OECD/The King's Fund, 2020; Steel *et al*. 2025). Parallel trends in upstream risks—most notably the sustained increase in population exposure to high body-mass index, sub-optimal diet, low physical activity and harmful alcohol use—together with the stalling or reversal of progress in lowering elevated LDL-cholesterol and systolic blood pressure, have offset the earlier gains achieved through declining smoking prevalence (Steel *et al*. 2025).

A complementary line of inquiry invokes wider social and macro-economic forces at play. Growing socioeconomic inequalities in mortality, health-system pressures, and the fiscal austerity that followed the 2008 financial crisis have all been linked to stalling longevity, particularly in disadvantaged groups (Karanikolos *et al*., 2013; Van Gool & Pearson, 2014).

An alternative hypothesis attributes the recent slowdown to cohort effects. Some scholars argue that successive birth cohorts may now be brushing up against an intrinsic ceiling of human longevity (Dong *et al*., 2016; Olshansky *et al*., 2024). More recently, Andrade *et al*. (2025) have extended the cohort perspective using a forecasting approach: applying multiple mortality projection methods, they document a marked reduction in the pace of cohort life expectancy improvements, suggesting that the slowdown may not be confined to period indicators alone.

These two latter explanatory avenues are in our opinion potentially at odds: if a cohort mechanism—especially one predicated on an impending biological ceiling to human lifespan—were truly dominant, the onset of deceleration should unfold asynchronously across nations with diverse demographic histories, rather than clustering in the same calendar years as the near-synchronous slowdown we currently observe. The deceleration we are observing in developed countries seems to be marked by a striking temporal synchrony, a feature that aligns more convincingly with a period effect—an exogenous shock acting on all populations simultaneously—than with a cohort-driven explanation.

But there is more. The experience of a handful of Nordic and Benelux countries—Norway foremost—demonstrates that continued life-expectancy gains are possible when cardiovascular and cancer mortality keep improving and risk-factor exposures are effectively contained. These same countries showed remarkable resilience during the 2019-21 COVID-19 shock (Steel *et al*. 2025). If the hypothesis of approaching a ceiling holds true, it remains to be explained why countries such as Norway, Sweden, Iceland, Belgium, and Denmark have not exhibited similar patterns. In other words, such heterogeneity suggests that the post-2011 slowdown is contingent, not inevitable.

Drawing on these considerations, our study seeks to determine whether a detailed, country-level inquiry can resolve, at least locally, the apparent tension between a predominance of cohort effects (whatever they might be) and the near-synchronous onset of the slowdown.

Italy, with its distinctive timing and policy context, provides an instructive test case for adjudicating between these competing explanations.

Italian national time-series, in fact, reveal an earlier inflection, with the slope of life-expectancy improvement flattening around 2008, a full three years before the modal OECD pattern (Salinari *et al*., 2023; Carboni *et al*. 2024). That timing coincides with a nationwide flattening of public health-care expenditure and the subsequent intensification of austerity through region-specific deficit-recovery plans (*piani di rientro*), a suite of austerity measures that curtailed hospital capacity and, according to causal analyses, precipitated a measurable rise in avoidable mortality (Arcà *et al*., 2020).

To probe whether cohort forces are truly at play in the Italian case, we therefore asked whether the deceleration emerges at the same historical moment across all age groups. This approach builds on earlier work by Ouellette, Barbieri, and Wilmoth (2014), who showed that major turning points in adult mortality trends across high-income countries can be used to discriminate between cohort-driven and period-driven processes. In their framework, discontinuities that occur at roughly the same calendar time across a wide age range point to contemporaneous, period-based forces, whereas cohort mechanisms imply age-staggered turning points that shift forward in time as cohorts age. The underlying logic in our Italian case is quite similar. If the national slowdown begins in 2008 yet its onset varies markedly by age—appearing earlier for some cohorts and later for others—the pattern would point to age-linked, cohort-specific mechanisms. By contrast, if the break occurs synchronously across the age spectrum, and that synchrony aligns with the rollout of health-care austerity, the evidence would favour a period effect driven by a system-wide shock. In the latter scenario, a common external force—as opposed to the life-course histories of distinct cohorts—must be acting on the entire population, regardless of birth year (and then cohort), to curb further gains in longevity. Our contribution differs in both scope and empirical strategy from that of Andrade *et al*. (2025). Rather than forecasting cohort life expectancy, we focus on observed age-specific mortality trends at older adult ages and examine whether the timing of their deceleration is consistent with staggered cohort dynamics or instead points to a synchronous period shock.

2. Methodological strategy

We use five-year age-group log mortality rates derived from cohort life tables available in the Human Mortality Database (HMD). Cohort tables are preferable here because the underlying single-year death rates ($m_{x,x+4}$) are released without the smoothing that HMD applies to period data, letting us analyse trends free of model-

imposed structure (see HMD Methods Protocol, version 6, for details). After retrieving the cohort rates, we rearrange them into a calendar-year matrix, simply by adding the cohort year to the central age of each class. In doing so, each cell records mortality at age-class $(x, x+4)$ in year t.

From this matrix we retain the years 1980–2019, a span long enough to detect structural changes yet narrow enough to centre on the late-2000s slowdown while stopping short of the COVID-19 perturbation that begins in 2020.

The analysis is confined to the 50 to 89year bracket. This range captures the ages most clearly implicated in the international slowdown (those in which cardiovascular disease and cancers dominate the burden of death) and produces cleaner signals by avoiding the volatility that characterises age outside that range.

Equally important, it steers clear of younger ages whose mortality trends in Italy were strongly perturbed by the mid-1980s heroin crisis and the first HIV/AIDS wave, shocks that hit men in their twenties and thirties particularly hard (Conti *et al*., 1994; Conti *et al*. 1997) and would inject unrelated noise into our estimates. By concentrating on 50+ we therefore focus on the part of the life course where the deceleration appears to be most pronounced while minimising confounding from epidemic phenomena outside our research scope.

The analytical strategy unfolds in two stages.

In the first stage we apply segmented regression to each five-year age-group series, allowing the algorithm to search, without prior constraints, for structural breakpoints and to estimate their timing.

The second stage tackles the same problem from the opposite direction. Guided by the stage-one results, we posit a single breakpoint common to all series. That breakpoint corresponds to the year most often selected in the initial search. In this way, we fit a basic interrupted time-series model to every age group. The model estimates separate linear trends before and after this year. If the slope difference proves statistically significant, the break is deemed to capture a genuine shift in the underlying mortality trajectory.

2.1. Stage one: Segmented regression

Segmented (or piece-wise) regression is a flexible extension of ordinary least-squares regression that allows the slope of a continuous time trend to change at one or more unknown points, termed breakpoints. Rather than pre-specifying the dates at which the relationship might shift, the method iteratively searches for the location(s) that best reconcile the data with two or more linear segments. In practical terms, it asks: *Is there a calendar year after which the rate of mortality improvement slows (or speeds up) in a statistically detectable way?*

To answer that question, segmented regression treats each portion of the series as linear. This assumption fits log-mortality rates time-series well, because, barring major shocks, their evolution tends to follow an almost straight-line trend over time, a regularity first documented by Lee and Carter (1992) and later confirmed for both mid-life and old-age mortality (Kannisto *et al*., 1994; Wilmoth, 1995; Thatcher *et al*., 1998).

For each fixed age group we then model the annual log mortality rate Y_t (with $t = 1980, \ldots, 2019$) as

$$Y_t = \beta_0 + \beta_1 t + \beta_2 (t - \tau) I_{\{t>\tau\}} + \varepsilon_t, \quad (1)$$

where $I_{\{t>\tau\}}$ is an indicator function that equals 1 after the breakpoint τ and 0 otherwise; β_0 is the intercept, β_1 is the pre-break slope, β_2 captures the change in slope after τ, and ε_t is an independent error term.

Since age-specific mortality rates have been trending downward for decades, a more negative slope ($\beta_2 < 0$) translates into a faster decline in death rates and larger annual gains in life expectancy (good news). Conversely, a positive post-break change in slope ($\beta_2 > 0$) makes the slope less negative: mortality is still falling, but more slowly, so improvements in life expectancy decelerate (bad news). The same interpretation applies whether the series are expressed in levels or in logs; the log transformation merely stabilises variances without altering the direction of change.

Breakpoints and coefficients of model (1) are estimated with the iterative procedure proposed by Muggeo (2008)'s *segmented* R package, which also allows the computation of confidence intervals for τ.

Because the 1980–2019 series could plausibly contain more than one historical shift, we followed the practice adopted in recent segmented/joinpoint-regression studies of life-expectancy trends (Minton *et al*., 2023; Zazueta-Borboa *et al*., 2024) and allowed the algorithm to search for no more than two breakpoints in each age-group series. In practice, some age-groups show only one; when a second appears, we report it but centre our discussion on the later break, as doing so aligns the analysis with the contemporaneous nature of the phenomenon we aim to analyse. Our approach differs only in the response variable: whereas those studies analyse life expectancy, we model age-group log-mortality rates, a choice justified by the aforementioned evidence that such rates also evolve approximately linearly over time.

2.2. Stage two: Basic interrupted time series

The second phase asks a simple question: *Can a single calendar year account for the trend change in every age-group series?* If such a common breakpoint exists, the pattern would be hard to square with a cohort explanation (cohorts age at different calendar times) but fully consistent with a population-wide period shock. Stage two fixes the year most frequently identified as breakpoint in Stage one for all series and asks whether the slope before that year differs significantly from the slope after.

Concretely, we run the same basic interrupted time-series model for each five-year age group, always imposing a single predetermined year as the breakpoint. Formally:

$$Y_t = \beta_0 + \beta_1 t + \beta_2 I_{\{t>\bar{t}\}} + \beta_3 t I_{\{t>\bar{t}\}} + \varepsilon_t. \quad (2)$$

Here β_1 captures the pre-break yearly change in log mortality, β_2 measures any shift in the level of the series associated with the post-break period and, finally, β_3 quantifies how much the post-break slope differs from the pre-break slope.

The form of model (2) is deliberately more flexible than the segmented-regression model (1). Now we fix a single, ex-ante breakpoint for all age groups. Allowing both a level shift (β_2) and an independent slope change (β_3) ensures that any departure from the pre-$\bar{t}$ trend can be detected.

At this stage, then, our primary focus is on the coefficient β_3. A statistically significant positive β_3 confirms that the trend after $\bar{t}$ diverges from that before $\bar{t}$. In this second stage, we also separated the analysis by gender.

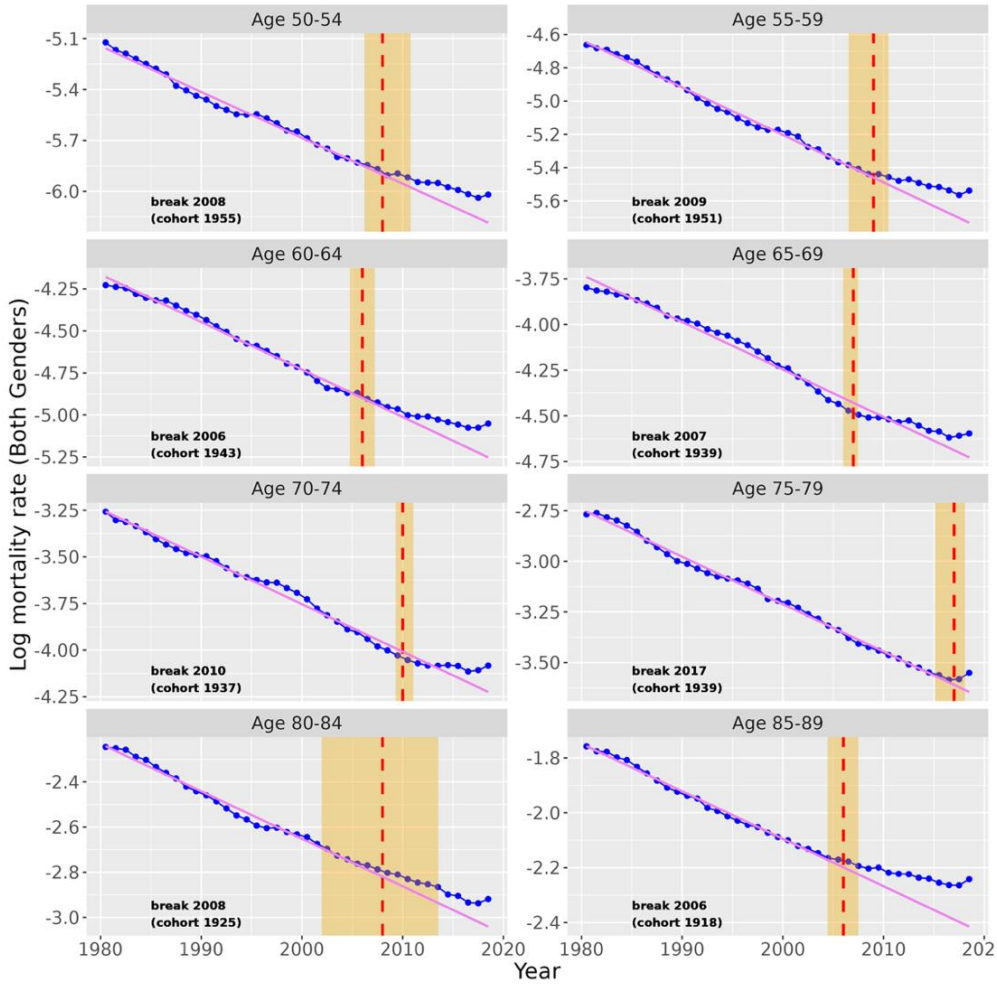
3. Results

Figure 1 plots the calendar-year (period) log-mortality rates for eight age classes (50–54 through 85–89) in the combined male-and-female population. Blue dots and line trace the observed series from 1980 to 2019; the red dashed line marks the breakpoint returned by the segmented-regression model, while the orange band gives its 95 % confidence interval. The purple line extends the pre-break slope forward, illustrating how mortality would have evolved had the earlier trajectory persisted. The caption in each panel records the point estimate of the break year and the birth cohort experiencing it (calculated as break year minus the midpoint age of the class).

Several features stand out. First, despite the independent estimation for each age group, the breakpoints crowd tightly around 2008–2010. Even allowing for the confidence interval, the overlap is substantial, indicating that disparate cohorts

encountered a similar inflection at roughly the same historical moment. Only the 75–79 group deviate slightly, breaking a few years later, yet still falls within a decade-wide window centred on the late-2000s.

Figure 1 – *(Stage one) Breakpoints in age–specific log-mortality trends, Italy 1980 – 2019.*

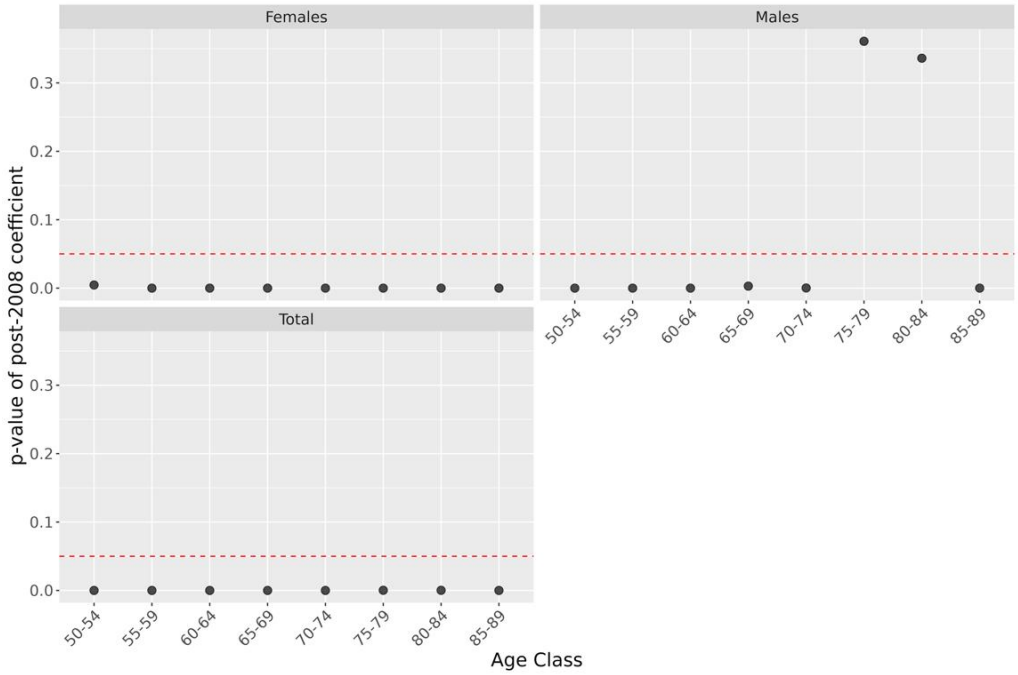


Blue lines show annual log-mortality rates for eight five-year age groups (50–54 to 85–89) in Italy, 1980-2019. The red dashed line marks the breakpoint estimated by segmented regression; the orange rectangle is its 95 % confidence interval. The purple line projects the pre-break trend beyond the breakpoint. Each panel reports the break year and the birth cohort to which it applies.
Source: Our elaboration on Human Mortality Database data.

Second, the direction of change is uniform: in every panel the post-break slope is less steep than the extrapolated purple line, signaling a slowdown in the pace at which mortality was falling. Put differently, improvements in survival diminished simultaneously across virtually the whole adult age spectrum.

Taken together, these patterns suggest a system-wide shock affecting all cohorts simultaneously rather than age-staggered dynamics consistent with a cohort mechanism. The second stage strengthens this evidence by formally testing whether the slope changes highlighted here remain statistically robust in basic interrupted-time-series models.

Figure 2 – *(Stage two) p-values for the post-2008 slope-change coefficient (β_3), by age class and sex, Italy 1980–2019.*



Each dot reports the p-value for the post-2008 slope-change coefficient (β_3) from the basic interrupted time-series model with a common breakpoint fixed at 2008, shown separately by five-year age class and sex. The red dashed line indicates the 0.05 significance threshold.
Source: Our elaboration on Human Mortality Database data.

Figure 2 displays the p-values for the post-2008 slope-change coefficient (β_3) obtained from the basic interrupted time-series models (model (2) with $\bar{t}$ = 2008), plotted by age class and sex. The red dashed line marks the 0.05 threshold. We fix 2008 because it marks the onset of nationwide health-care austerity measures,

allowing us to test whether that single policy-inflection year can account for a shift in mortality trends across all age groups and both sexes.

Across the pooled-sex specification (right-hand panel) the evidence is unequivocal: every age group registers a p-value well below 0.05, confirming that 2008 corresponds to a statistically significant shift in the mortality trend. When the analysis is split by sex, the picture remains essentially unchanged: all female and male age classes return significant results except two cases: males 75-79 and 80-84. Their higher p-values are not straightforward to explain. One possibility is cohort selection: the men aged 75–84 in 2008 were born roughly between 1926 and 1931, placing their young adulthood squarely in the Second World War. Such cohort-specific factors may dampen the detectable change in slope, without altering the overall period-effect pattern. It is important to note that, although Figure 2 visualises only p-values, the underlying β_3 estimates are positive in every case that reaches statistical significance (p-value < 0.05). A positive β_3 means the post-2008 trend is less steeply negative, i.e. the decline in mortality slowed. In terms of size, β_3 estimates cluster between ≈ 0.006 and ≈ 0.022 log-points per year, implying that the annual pace of mortality decline slowed by roughly 0.6% to 2.2%. In early-old ages (50–54 and 55–59) the slowdown is clearly steeper for men than for women. The differential all but vanishes at 60–64, and from 65–69 onward the sign flips: the deceleration becomes progressively larger among women, reaching its widest margin around 80–84. In the final class, 85–89, the gap narrows again—yet the female slowdown still edges out the male one.

Taken together, these results reinforce the picture drawn in Stage one. Setting 2008 as a common breakpoint explains a statistically meaningful change in the slope of log-mortality for virtually the entire adult population, in both sexes. Moreover, all significant β_3 coefficients are positive, indicating that the decline in death rates slowed after 2008, regardless of cohort. A system-wide deceleration in mortality improvements began in 2008, consistent with a period effect rather than staggered, cohort-specific dynamics.

4. Conclusion and discussion

This study set out to weigh two contrasting interpretations of Italy's recent longevity slowdown: the possibility that it reflects a biological ceiling manifesting through cohort ageing versus a population-wide period shock rooted in the post-2008 wave of health-care austerity. To do so we combined an exploratory segmented-regression search for structural breaks with a confirmatory interrupted time-series test that fixed the candidate break year at 2008.

The two empirical stages converged on remarkably similar results. Across every age class from 50–54 to 85–89, the segmented model located an inflection in the late 2000s, with confidence intervals that overlap almost perfectly. When we then imposed 2008 as the breakpoint, the basic interrupted time-series models showed that the post-2008 slope became less steep in virtually every case. The result of this analysis appears clear: different cohorts, in their respective stages of the life course, encountered the same turning point at the same historical phase, and for every subpopulation that turning point signalled a slackening of mortality improvement.

Such synchrony is at odds with a dominant cohort mechanism, which would be expected to play out in a staggered fashion as successive birth cohorts age into later life. It is far more consistent with a period effect—an exogenous force acting simultaneously on the whole population.

The temporal match with Italy's fiscal retrenchment is striking. Public health-care spending stopped climbing in 2008 (Ciocci & Spagnolo, 2020), and the region-specific *piani di rientro* (a targered boost of health austerity) soon followed, trimming hospital capacity and tightening budgets precisely when our data suggest that mortality gains began to slow (Aimone Gigio *et al*., 2018).

Comparative work in other high-income countries indicates that mortality can react quickly to fiscal retrenchment (e.g. McCartney *et al*., 2022), so a short lag in Italy would not be unusual.

Whether the channel is delayed diagnostics, slower diffusion of new therapies, or strain on community care, the key empirical fact remains: improvements slowed almost simultaneously across the whole adult age spectrum.

Any convincing explanation of Italy's post-2008 deceleration must therefore begin by accounting for this temporal synchrony.

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