Candidate Origins of the Recent Stagnation in Midlife Mortality in the United States

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This paper offers a new explanation for the alarming trend in midlife mortality observed in the United States since 2000, when death rates among working-age Americans stalled after decades of unprecedented progress. The explanation hinges on a striking parallel with a pattern that emerged just before 1950, in which death rates among children also stalled, ending a period of similarly historic gains. We will show that sustained reductions in both midlife and early-life mortality rates ceased abruptly with the same birth cohort. The implication, backed by empirical and theoretical literature connecting adulthood health to childhood circumstances, is that the recent stagnation may not be a new phenomenon, but the second manifestation of an older one. Supporting this hypothesis, we will find similar trends in midlife mortality in, and only in, peer countries where there was also a stagnation in early-life mortality decades prior, namely in other Anglo countries. We will also demonstrate that the slowdown in childhood has the capacity to be, not just a cause, but the primary cause of the slowdown in adulthood.

At the turn of the 21st Century, midlife mortality rates in the United States stopped improving after decades of steady decline (Case & Deaton, 2015). The number of deaths associated with this stagnation is now on the order of hundreds of thousands *annually*, and the losses continue to mount (Preston & Vierboom, 2021; Bor et al., 2023). The opioid epidemic has played a significant role in the ongoing crisis, but a large majority of the excess death of working-age Americans since 2000 is not well understood (Meara & Skinner, 2015).

This paper posits that the explanation could lie in a similar trend that unfolded several decades prior. In the middle of the 20th Century, after a period of rapid and sustained reductions, progress against early-life mortality slowed as well in the United States (Moriyama, 1960). Health officials at the time wrote extensively about death rates among infants and children leveling off around 1950, but no researcher since has drawn a critical parallel between that event and modern ails: They involve the same people.

We will show that celebrated progress in both midlife and early-life mortality ends suddenly with Americans born in 1947; precisely the cohorts ushering in surprisingly poor adult health outcomes now ushered in surprisingly poor health outcomes as children. We hypothesize accordingly that the current crisis is a reflection of some deterioration in childhood circumstances long ago that dampened the trajectory of childhood mortality and left survivors in worse health permanently.

Such an explanation for high midlife mortality rates in the United States would be new, but the underlying theory is familiar. A large body of research spanning many fields has established strong ties between early-life environment and later-life health (Elo & Preston, 1992; Gluckman & Hanson, 2004; Currie & Vogl, 2013). Within that literature, scholars have credited some of the remarkable progress in adult mortality at the end of the 20th Century to the remarkable progress in child health at the beginning (Cutler, Deaton &

Lleras-Muney, 2006). Our results may suggest, conversely, that adult mortality has stalled ever since because it lost that internal steam.

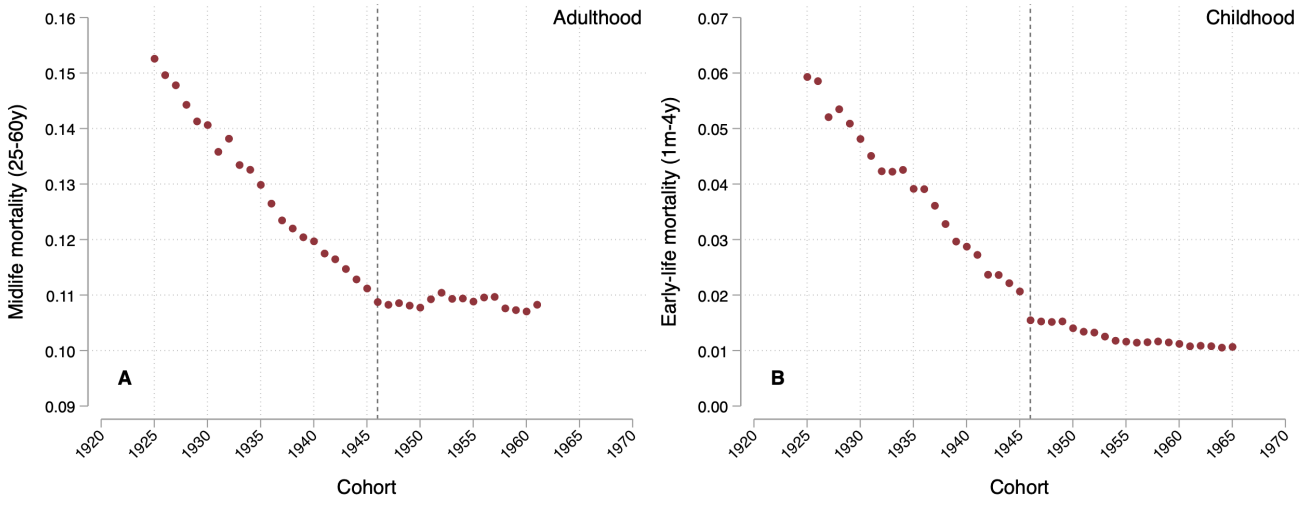
We will provide further evidence that a life-course relationship is present in a cross-country comparison. While the United States saw midlife mortality rates stall around 2000, most of its peer countries did not (Case & Deaton, 2015; Woolf & Schoomaker, 2019). In a pool of continental European countries, where adult mortality has not stalled, we see no antecedent stagnation in child mortality. In contrast, however, we find that the United States is not the only country to exhibit a break from cohort trend in working-age mortality around 1947. We observe one in other Anglo countries, and, in those countries, we observe a break from trend in child mortality for the same cohorts.

Finally, we will provide evidence that a life-course relationship is not only present, but quantitatively important. Under strong but defensible assumptions, we will estimate the number of working-age deaths associated with the observed change in child mortality. We find that, when coupled with the opioids epidemic, the stagnation in early-life mortality in the middle of the 20th Century can explain almost all of the recent stagnation in midlife mortality in the United States.

Progress in both midlife and early-life mortality ends abruptly with the same birth cohort

We first show that the cohorts who initiated the recent stagnation in midlife mortality are also the cohorts who initiated the stagnation in early-life mortality decades earlier. Figure 1 illustrates this alignment by holding their mortality rates in adulthood and childhood side by side.

Fig. 1: Cohort alignment



***Note**: In Panel A, for the United States, we plot the probability of dying before or during age 60 conditional on surviving to age 25 by year of birth (source: Human Mortality Database). In Panel B, again for the United States, we plot the probability of dying before or during age 4 conditional on surviving the first month of life by year of birth (source: for child mortality, Social Security Administration cohort life tables for cohorts prior to 1932 and the Human Mortality Database for the cohort of 1932 onward; for post-neonatal mortality, "Vital Statistics Rates in the United States, 1900-1940"). In both panels, the dashed line is drawn through the cohort of 1946, the final cohort to experience significant improvements in either probability.*

In Panel A of Figure 1, we plot midlife mortality by year of birth. We define midlife mortality as the probability of death between the ages of 25 and 60 ($_{35}q_{25}$ in standard notation). We choose this age range in order to overlap with the definition of "working ages" (ages 25 through 64) while still allowing for a large number of cohorts born in the second half of the century to age through the window of observation. We obtain these midlife mortality rates, which we will sometimes call "adult" or "working-age" rates, from publicly available cohort life tables released by the Human Mortality Database (HMD, 2025).

Panel A shows a rapid improvement in midlife mortality that ends abruptly with the 1947 cohort. Between the 1925 and 1946 cohorts, midlife mortality fell by nearly 30 percent. It remains, however, at the level of the 1946 cohort through the 1961 cohort, who are now in their early sixties.

Panel A essentially recasts midlife mortality rates from a period perspective to a cohort perspective. The perspectives are equally valid; they contain the same information. However, the cohort perspective more clearly illustrates the concordance between midlife mortality and early-life morality revealed in Panel B.

In Panel B of Figure 1, we plot early-life mortality for the same cohorts. We define early-life mortality as the probability of death in the first five years of life, conditional on survival through the first month of life. We exclude the neonatal period because the causes of death in the first month of life are markedly different from those in the remainder of childhood, a point to which we will soon return. We obtain these rates by augmenting the year-by-age mortality rates in the Human Mortality Database with post-neonatal mortality rates from a special report by the National Center of Health Statistics (Lindner & Grove, 1943).[1]

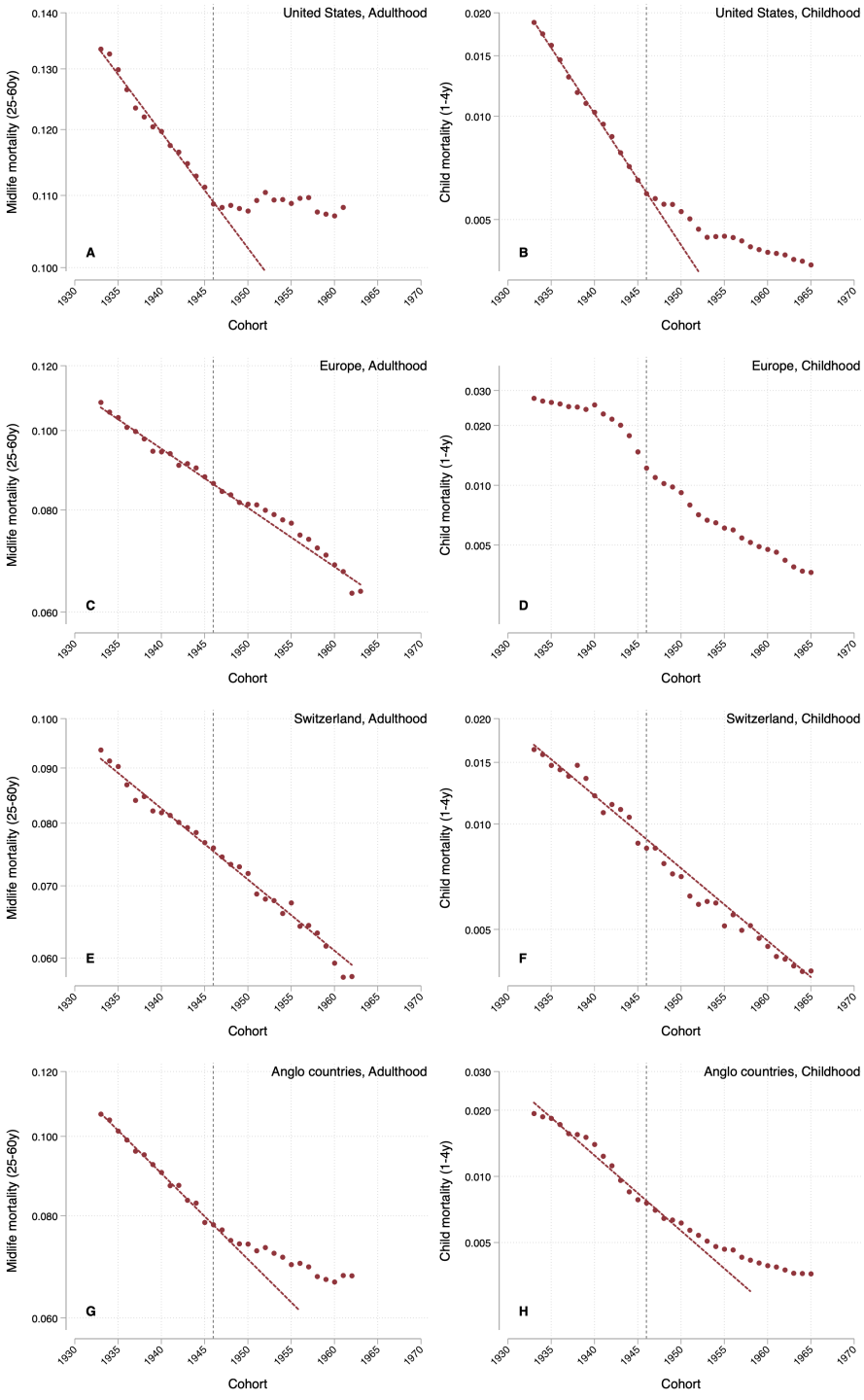
Panel B shows the dramatic decline in early-life mortality observed in the first part of the 20th Century, falling from six percent to less than two percent between the 1925 and 1946 cohorts. Like midlife mortality, however, early-life mortality levels off abruptly with the 1947 cohort.

One interpretation of the cohort alignment seen in Figure 1 is that the recent stagnation in midlife mortality reflects "scars" from early life. The notion of scarring is that changes in early-life environment, typically proxied by changes in early-life mortality rates, affect health outcomes at older ages. Such relationships have been observed repeatedly in a variety of settings (Elo & Preston, 1992; Fogel & Costa, 1997; Gluckman & Hanson, 2004; Gluckman et al., 2008; Gluckman et al., 2016; Currie & Vogl, 2013). In fact, much of the decline in adult mortality in the second half of the 20th Century has been attributed to the decline in early-life mortality in the first half of the century (Cutler, Deaton & Lleras-Muney, 2006). A corollary to this view is that, if improvements in early life cease, as they appear to have done around 1947 (Panel B), so too will the health benefits they confer at older ages (Panel A).

The exclusion of neonatal mortality is standard practice in the literature on scarring. Bozzoli, Deaton & Quintana-Domeque (2009), who develop a parsimonious model of scarring, disregard neonatal mortality because it is determined by technological innovations, such as intensive care units, rather than

[1] For cohorts prior to 1932, which is the first year available in the Human Mortality Database for the United States, we use cohort life table from the Social Security Administration (Bell & Miller, 2005).

Fig. 2: Geographic consistency



***Note**: In Panels A and B, for the United States, we plot midlife and child mortality rates, respectively, by year of birth (source: Human Mortality Database). The diagonal dashed line is fitted to the cohorts from 1933 thorough 1946. In Panels C and D, we show the same for a pool of equally weighted European countries that includes Belgium, Denmark, Finland, France, Iceland, Italy, the Netherlands, Norway, Spain, Sweden, and Switzerland; in Panels E and F, Switzerland alone; in Panels G and H, Australia, Canada, and the United Kingdom, equally weighted. In all panels, the vertical axis is in log scale; the vertical dashed line is drawn through the cohort of 1946.*

environmental factors, such as infectious diseases, which leave what they call "residue" on survivors.[2]

The burden of infectious disease has long been the focus of the literature on scarring, and it is salient in our context. Finch & Crimmins (2004), for instance, describes a connection between early-life burden of infectious disease and later-life incidence of cardiovascular disease. The principle driver of the stagnation in early-life mortality in the middle of the 20th Century was stalled progress against death from infectious disease (Moriyama, 1960). The principle driver of the recent stagnation in midlife mortality is now understood to be stalled progress against death from cardiovascular disease (Mehta et al., 2020).

Although scarring that operates through infectious disease appears to be a possible explanation, exactly what changed for cohorts beginning in 1947 is not something we will ascertain here. Our goal is to document that two of the most pronounced changes in American mortality in the last hundred years appear to be related, and we now turn to other countries for additional evidence of a connection.

Mortality trends in other countries appear consistent with the scarring hypothesis

The stagnation in midlife mortality witnessed in the United States is not widespread, as other developed countries have continued their improvements from the end of the 20th Century into the 21st (Woolf & Schoomaker, 2019). These peer countries, therefore, provide a test of the explanation we have posited for the stagnation in midlife mortality. If it is indeed rooted in early-life events, then the stagnation in early-life mortality decades prior should be confined to the United States as well. Figure 2 shows that it is, with one important exception.

In Panels A and B of Figure 2, for reference, we again show midlife and early-life mortality rates, respectively, in the United States by cohort. We make two adjustments. First, we show these rates on a log scale to more clearly illustrate their breaks from trend. Prior to their breaks, both rates are declining linearly in logs. Second, we show specifically child mortality, death in the first five years of life conditional on survival through the first year. Our previous measure of early-life mortality included post-neonatal mortality, which we obtained from a special report by the National Center for Health Statistics (Lindner & Grove, 1943). For the cohorts we study, such granularity with respect to age is not available in many countries. The choice of child mortality, which can be calculated from the Human Mortality Database alone, allows for the inclusion of many more countries in the results to follow.

In Panels A and B, as before, both midlife and child mortality break sharply from a linear (in logs) trend with the 1947 cohort. Progress in child mortality does not cease as it does in adult mortality, but the break in child mortality is just as pronounced. Whereas adult mortality rates fall by about 1.5 percent per year before halting in 1947, child mortality rates fall by almost 8.0 percent per year before slowing to about 2.5 percent.

In contrast, in Panels C and D of Figure 2, we show midlife and child mortality for a pool of European

[2] For completeness, in Appendix Figure 1, we nonetheless show neonatal mortality rates by cohort. Unlike midlife and other early-life morality rates, they do not break trend with the 1947 cohort. They do break trend later, in about 1951, but that departure is consistent with a broader trend in mortality that affected all ages (Kitagawa & Hauser, 1973).

countries. The pool consists of the eleven countries for which we have data on the needed cohorts from the Human Mortality Database.[3] A notable exception is the United Kingdom, which we will discuss separately. In Panel C, as expected, Europe shows little indication of a trend break in adult mortality in the cohorts we study.[4] In Panel D, the pattern in child mortality is somewhat difficult to discern because World War II drives mortality up for several cohorts prior to 1947, but there is no clear evidence of a sustained break from trend.

In Panels E and F of Figure 2, we isolate Switzerland from the pool of European countries. We highlight Switzerland because, although no country was immune to World War II, it was relatively unscathed. There, we see a clearer picture in which both adult and child mortality stay their course.

In Panels G and H of Figure 2, we examine the three Anglo countries for which data are available, Australia, Canada, and the United Kingdom. We select these countries because, unlike the European countries above, they show evidence of a trend break in midlife mortality in the middle of the century. The United States is often thought to be an outlier in terms of working-age mortality, but, when viewed from a cohort perspective, the United States does not appear to be alone in its flagging progress.[5] It is joined by its Anglo peers, and in those countries, crucially, we also see a break from trend in child mortality. The break in midlife mortality in the Anglo countries appears to be delayed relative to the United States, emerging about two years later. Their break in child mortality occurs with the same delay. The break in midlife mortality in the Anglo countries also appears less sharp than it is in the United States. Their break in child mortality is muted as well.

The geographic variation presented in Figure 2 is consistent with scarring. In places where there is no stagnation in adult mortality, we see no evidence of an earlier stagnation in child mortality. In places where adult mortality has stalled, child mortality among the same cohorts stalled as well. Although the two patterns share timing and geography, whether the stagnation in early-life mortality decades ago can explain a small share or a large share of the recent stagnation in midlife mortality is an outstanding question, which we address in the final part of our analysis.

Capacity for childhood circumstances to explain adulthood stagnation is large

If the stagnation in midlife mortality in the United States is indeed the echo of the stagnation in early-life mortality fifty years prior, the question remains: How much of the midlife stagnation can we attribute to the early-life stagnation?

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[3] These European countries are Belgium, Denmark, Finland, France, Iceland, Italy, the Netherlands, Norway, Spain, Sweden, and Switzerland.

[4] There is a modest but apparent rise in adult mortality in European countries among cohorts from the 1950s, which is consistent with the pandemic of HIV/AIDS. Although the United States has its sharpest trend break in the 1947 cohort, its adult mortality rates are elevated in the 1950s as well.

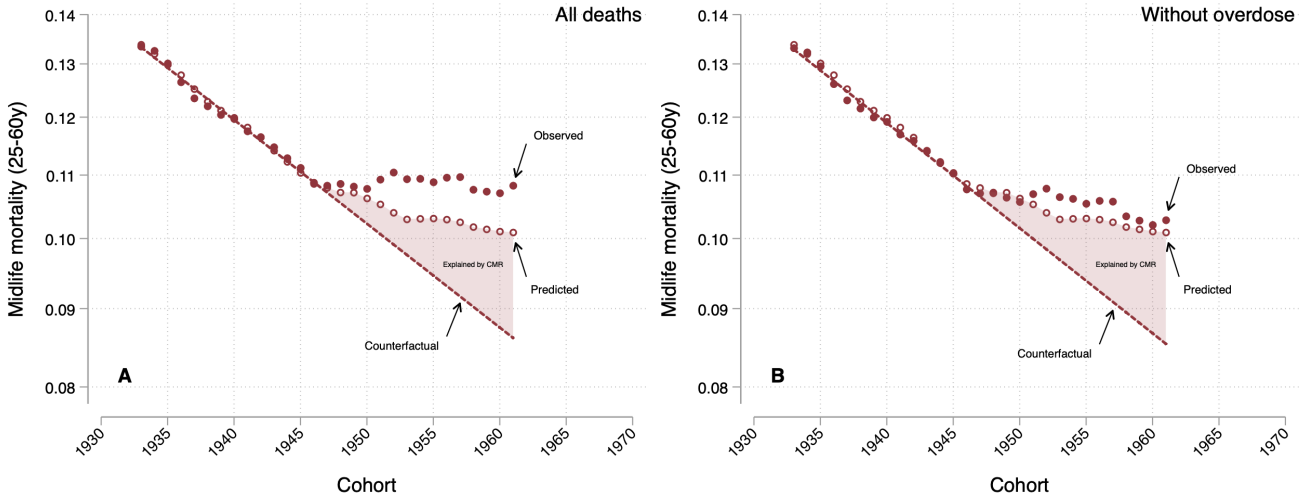
[5] Others have noted, from a period perspective, that progress in midlife mortality in the United Kingdom especially has slowed in recent years, although not to the same degree as in the United States (Ho & Hendi, 2018; Murphy, 2021).

Answering this difficult question first requires an assumption about how midlife mortality would have evolved for the 1947 and subsequent cohorts had they not experienced hardship in childhood. We will assume that working-age mortality would have continued on the steady downward log-linear trend set by previous cohorts. Other countries provide support for this counterfactual. In most European countries, it continued to fall on trend. It did so despite starting at lower levels in many of those countries, defying the possibility that midlife mortality reached some irreducible minimum in the United States.

The answer also requires an assumption about the relationship between midlife and child mortality. Specifically, we need a parameterization of scarring. We choose the linear relationship between midlife and child mortality observed in American cohorts prior to 1947, which we fit in logs in a regression with ordinary least squares. These estimates give a prediction of midlife mortality that is based solely on the child mortality rate of a given cohort. Therefore, as scarring would imply, when child mortality breaks from trend, so will the predictions of adult mortality. The question is how closely the predicted midlife mortality rates align with the observed midlife mortality rates.

Figure 3 shows the results of our "accounting exercise." Focusing on Panel A, the solid dots are exactly the data we have plotted before, the observed adult mortality rates in logs by cohort. The dashed line is their counterfactual path, which is a linear extrapolation of the cohort trend set prior to 1947. The hollow dots are the predictions of adult mortality based on child mortality rates. The shaded region consequently is the share of the stagnation in midlife mortality that can be explained by the stagnation in child mortality. For instance, for the 1961 cohort, the break in child mortality explains about two-thirds of the departure in observed mortality from its counterfactual trend. The share is similar for other cohorts.

Fig. 3: Accounting exercise



***Note**: In Panel A, we plot midlife mortality by cohort three ways. The solid dots are observed values (source: Human Mortality Database). The dashed line is a linear extrapolation of the observed values between 1933 and 1946, inclusive. The hollows dots are the predictions of midlife mortality based on child mortality. In Panel B, we do the same, but we exclude deaths from overdose, which are tabulated by cohort from the National Vital Statistics System.*

While scarring explains a substantial share, two thirds, of the stagnation in midlife mortality, it does not explain all. There is no reason, however, to believe that it should. The opioid crisis, as mentioned at the outset, plays a critical role in poor working-age mortality rates in the United States. Notably, researchers have estimated that the opioids crisis can explain about one third of the excess deaths since 2000 relative to the period trend established in the 1990s, approximately the amount left unexplained in our accounting exercise (Schmid, 2016).

Can the opioid crisis complete the account? It can if it is distinct from the scarring mechanism we observe. If instead the early-life harm manifests in ways that lead to overdose, for instance through chronic pain, then there is overlap for which we must account. Evidence suggests, however, that they are distinct. Cutler & Glaeser (2021) show that the opioids crisis is not about rising pain, but about a new treatment for pain that hit markets in the late 1990s.[6] In this respect, the opioids crisis is a period phenomenon that happened to strike the cohorts we study. Supporting this view, in Appendix Figure 2, we plot, by cohort, the share of midlife deaths that is attributable to overdose, most of which involve opioids.[7] The share is accelerating such that cohorts born just a few years after 1947 fare much worse than those born just a few years prior, but the share appears to trend smoothly around 1947. Because adult mortality breaks sharply from trend (Panel A of Figure 1), if scarring had a large impact on overdose, we would expect similar sharpness, not the observed smoothness.

Accordingly, in Panel B of Figure 3, we repeat the exercise in Panel A, but we exclude deaths from overdose in the observed adult mortality rates. As a result, the predictions based solely on child mortality rates are nearly the same as the observed midlife mortality rates. Indeed, when we combine the cohort harm imparted early in life with a period opioids crisis that happened to strike the same cohorts, we can almost fully account for the recent stagnation in midlife mortality in the United States.

Discussion

Exceptionally poor rates of midlife mortality among developed nations is now widely regarded as the most urgent public health concern facing the United States, but the reasons for the country's deteriorating standing since the turn of the century remain unclear (Cutler, 2017; Meara & Skinner, 2015). This paper posits that the answers may lie in events that transpired about 75 years ago, when rates of early-life mortality stalled in an eerily similar fashion. Critically, the two departures from promising trends begin with same birth cohort, raising the possibility of scarring, a robust literature showing that child health can affect adult health many years later (Elo & Preston, 1992; Gluckman & Hanson, 2004; Currie & Vogl, 2013).

A cohort mechanism, such as this, has profound implications not only for the past and the present of population health in the United States, but for the future. It suggests that the poor mortality outcomes since 2000 are concentrated in midlife only because most of the affected cohorts remained in midlife when they were studied. These cohorts will carry their ills forward, and evidence is surfacing that mortality rates

[6] Currie & Schwandt (2021) reach a similar conclusion.

[7] We consider all overdose deaths partially because most of them involve opioids, but also because of the strong substitution patterns between opioids and other drugs that frequently lead to overdose, such as heroin (Alpert et al., 2018).

at older ages are beginning to stall (NASEM, 2021). A cohort mechanism also suggests that poor midlife mortality is not temporary. Because the slowdown in childhood mortality was persistent, younger cohorts will also experience an elevated risk of mortality throughout adulthood, and signs of that danger are emerging as well (NASEM, 2021).[8]

This paper is not the first to suggest a cohort explanation for the recent stagnation in midlife mortality in the United States. Case & Deaton (2015), the researchers who brought the urgent public health issue to light, ultimately adopt a cohort view of the stagnation.[9] The explanations that they and others since have offered, however, are focused on disadvantages that accumulate slowly over the life course, such as diminished job opportunities and a flawed healthcare system (Case & Deaton, 2020; Montez et al., 2020). We find disadvantages grounded much earlier in life.

Our results shift attention toward events that scarred infants and children born just after the Second World War. In doing so, we bring into focus another unresolved literature. Researchers have recorded several peculiar outcomes for these post-war cohorts, including poor anthropometric measures, low educational attainment, and high rates of unemployment (Hamill, 1977; Card & Lemieux, 2001; Reynolds, forthcoming). These findings are consistent with our hypothesis of truly lifelong problems, as early-life conditions have been linked causally to all of these outcomes, not just to mortality (Currie & Almond, 2011; Almond et al., 2018). Our results may even provide a cohesive explanation for the once disparate observations.[10]

Our results also suggest that the proximate causes of the cohort stagnation should extend to other Anglo countries, where we observe similar patterns to those in the United States. Further narrowing the set of possibilities, we find no evidence of these post-war trends in a large pool of European countries. Explanations, for example, involving the Baby Boom or rapid industrialization, which were pervasive, are unlikely to fit this mold. However, in just the last few years, some of those European countries have started to show signs of turning a grim corner (Dowd et al., 2024a; Dowd et al., 2024b). Whether more countries will follow and whether these turns were preceded by analogous changes in childhood mortality decades prior are open questions. Potentially, our results suggest that the United States is less an outlier among developed nations and more a harbinger of what's to come.

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[8] Progress in early-life mortality picked back up in the late 1960s, but it did so at slower pace and for different reasons, reasons that may not confer the same benefits as previous reductions.

[9] Presciently, they say of working-age Americans (Case & Deaton, 2020, p. 35), "There is something about these people that makes them susceptible and that they carry with them through life."

[10] Another possibility is that exactly this kind of intervening adversity amplifies latent harms from childhood into high rates of mortality in adulthood.

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Appendix

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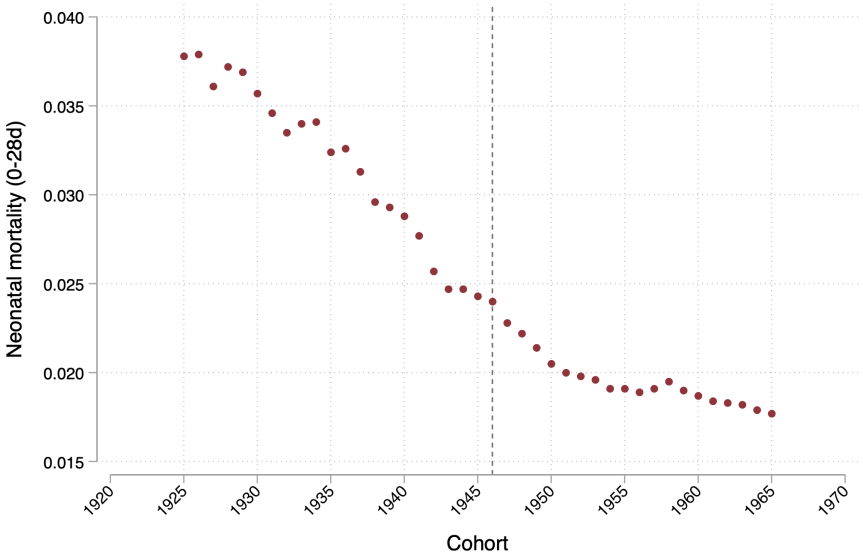
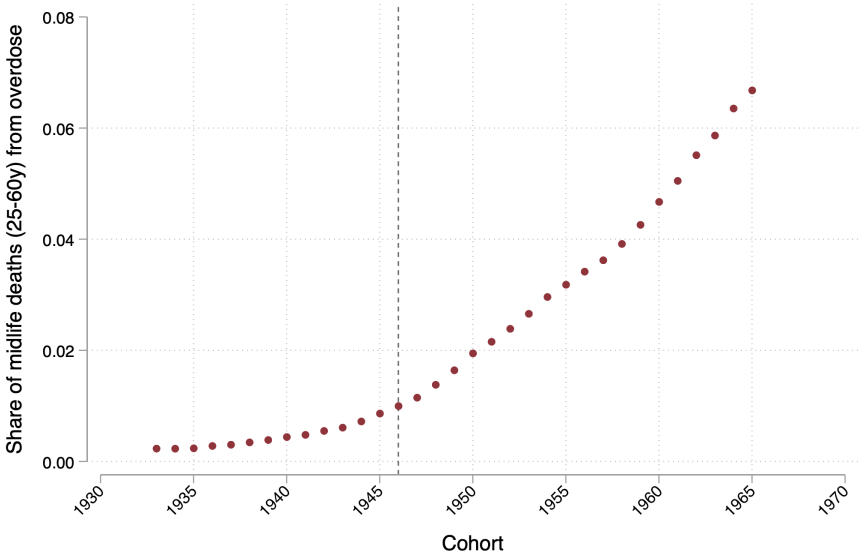
App. Fig. 1: Neonatal mortality



***Note**: We plot neonatal mortality rates, the probability of death in the first 28 days of life, by cohort (source: "Vital Statistics Rates in the United States, 1900-1940"). The dashed line is drawn through the cohort of 1946.*

App. Fig. 2: Overdose shares



***Note**: We plot the fraction of midlife deaths in the United States with underlying causes that fall under overdose, ICD codes ??? (source: National Vital Statistics System). The dashed line is drawn through the cohort of 1946.*