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The Broad Decline in Health and Human Capital of Americans Born after 1947

By Nicholas Reynolds*

I present evidence of a cross-cohort decline in the health and human capital of Americans, beginning with those born after 1947 and continuing until those born in the mid-1960s. Education, men's wages, women's maternal health (proxied by their infants' birthweight), and mortality all exhibit trend breaks near the 1947 cohort, such that each outcome worsens for subsequent cohorts relative to prior trend. The decline is large enough to drive: i) educational declines in the 1960s, ii) increases in low birthweight in the 1980s, iii) mortality increases since 1999, and to contribute substantially to iv) wage stagnation since the 1970s.

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Health and human capital improved enormously across American cohorts born in the first half of the twentieth century. Many argue that the key driver of these improvements were gains in early life health.¹ I present evidence that, in a number of dimensions, this progress in adult health and human capital hit a sudden stop and reversed trend for cohorts born after 1947. Instead, the underlying health and human capital of Americans born in each year between 1947 and the mid-1960s has been worse than that of those born a year earlier. I also argue this decline likely originated in adolescence or earlier.

My cohort-based theory provides a partially unified explanation of puzzling societal declines occurring at distinct points over the last 60 years. As post-1947 cohorts have aged their depressed health and human capital have manifested as lower test scores, educational attainment, wages, and maternal health, and increased mortality rates — contributing to well-known aggregate declines in these outcomes. In the 1960s and 1970s, these cohorts drove the sharp declines in achievement tests, high school graduation rates, and college enrollment rates (National Commission on Excellence in Education, 1983; Card and Lemieux, 2001b). The depressed labor market ability of men in these cohorts then led them to have lower wages, contributing to wage stagnation since the 1970s, particularly for those without a college degree (Gould, 2014; Katz and Murphy, 1992; Autor, Katz and Kearney, 2008). Depressed health as mothers led the women in these cohorts to give birth to less healthy infants, driving the upturn in the low birth weight rate in the 1980s (Centers for Disease Control, 1994; Currie and Gruber, 1996). Ultimately, the poor health of these cohorts has increased their likelihood of untimely death, contributing to recent mortality increases at midlife (Case and Deaton, 2015, 2017; National Academies of Sciences, Medicine et al., 2021).

Figure 1 shows that the trend across cohorts in age-adjusted educational attainment, wages, maternal health (proxied by the birth weight of infants), and mortality all exhibit trend breaks near the 1947 cohort, such that each outcome declines for subsequent cohorts relative to the prior trend. These simultaneous trend breaks, while striking, could in principle reflect differences in external factors which these cohorts were exposed to, rather than underlying differences in health and human capital. That is, cohorts born after 1947 may have been otherwise similar to earlier cohorts, but were merely unlucky to have experienced bad conditions throughout their lifetime.

I therefore provide evidence, under increasingly weak assumptions about the nature of external factors, that these patterns reflect a decline in the underlying health and human capital of cohorts born after 1947, relative to the prior trend. First, I estimate traditional age-period-cohort models, which assume cohort, age, and year factors are each additively separable. Second, I estimate models with a trend break of unknown location in cohort effects while allowing for a separate polynomial in age in each year, adapting methods from the structural break

 $^{^1 \}rm See$ for example Fogel (1986); Fogel and Costa (1997); Costa and Steckel (1997); Floud et al. (2011); Fogel (2012); Costa (2015).

literature. In companion work, I present evidence from a method which allows identification and estimation of a relative cohort decline in a general nonseparable framework (Reynolds, 2023b).

These methods reveal strong evidence of a trend break at the 1947 or 1948 cohort in underlying health and human capital evident in each of the above outcomes. The remaining threat to validity would be changes in the impact of age across years which cannot be approximated by smooth polynomials, and repeatedly "hit" the same cohorts. Alternative explanations in the literature do not have this feature. For example, the effects of shifts in supply or demand will be smooth as long as individuals who are close in age are substitutable (Card and Lemieux, 2001a).

The cohort-based decline is large enough to have contributed substantially to the year-over-year declines described above. This suggests that previously independent searches for the causes of educational declines of the 1970s, increases in the low birth weight rate in the 1980s, increases in midlife mortality since 1999, and to a lesser extent wage stagnation over the last 50 years, can be at least partially unified.

Why did things go so deeply wrong for Americans born after 1947? My investigation so far yields no smoking gun. The cohort decline appears to have originated in adolescence or earlier — predating labor market entry — though the effects of this initial poor start may plausibly have been exacerbated by later shocks to labor demand and opioid supply. The decline is remarkably widespread across racial groups and geography, among native-born Americans. Easterlin (1987) predicted that Baby Boomers would be negatively affected by their sheer number. The simplest version of such a "cohort crowding" theory cannot match the timing of the cohort decline, but the contribution of the large demographic changes associated with the Baby Boom should be studied further.

I. Literature review

The decline in educational attainment for cohorts born after the late-1940s is well known but it's cause is not. See Card and Lemieux (2001b); Heckman and LaFontaine (2010); Acemoglu and Autor (2012); Goldin and Katz (2007); Handy and Shester (2019).

Studies of wage declines in the 1980s for lower educated men, considered but largely dismissed the role of a cross cohort decline in "earnings ability" (eg. Bound and Johnson, 1992; Katz et al., 1999). An earlier paper, Bishop (1989), estimated that the decline in cognitive ability, as measured by test scores, had a large effect on wage rates for these cohorts and would continue to contribute substantially to productivity decline. Guvenen et al. (2022) document a decline in men's median lifetime earnings of more than 10 percent between the 1942 and 1960 birth cohorts.

Case and Deaton (2015) documented a shocking increase in the all-cause mortality rate of non-Hispanic white Americans and noted a proximate cause was increases in so-called "deaths of despair" — drug and alcohol poisonings, suicide, and chronic liver diseases and cirrhosis. Subsequent research has examined mortality across racial groups and suggested that cohort differences in health and disadvantage which predated the 1990s are likely important as well (Case and Deaton, 2017; Lleras-Muney, 2017; Masters, Tilstra and Simon, 2017; Zang et al., 2018; Acosta et al., 2020; Reynolds, 2023*b*; National Academies of Sciences, Medicine et al., 2021). With the exception of my earlier paper focused on white mortality (Reynolds, 2023*b*), these papers do not focus on the precise timing of the cohort decline.

Borella, De Nardi and Yang (2020) highlight that both earnings have been lower and mortality has been higher for cohorts born in the 1960s compared to those born in the 1940s — though their analysis is restricted to white Americans without a college degree.

My paper is unique in documenting the shared timing of the cohort decline across multiple outcomes, for the overall US population. Also unique is my explicit focus on identifying whether the declines for these cohorts are driven by long-standing differences in health and human capital, as opposed to external factors or "bad luck." I also emphasize the sharp nature of the cross-cohort trend break and its precise location at the 1947 cohort; which should aid the search for it's cause. To my knowledge no prior papers have documented evidence of the cohort decline in maternal health shown in this paper.²

Since I began work on this project a series of papers by Hui Zheng and coauthors have shown declines in cognitive ability and various health outcomes for approximately the same cohorts (Zheng, 2021; Zheng and Echave, 2021; Zheng et al., 2022). Interpretation is complicated by the fact that all results come from a "mixed-model" in which age effects are modelled as fixed effects and cohort and period effects as random effects; and there is no focus on the precise timing of the decline. Adult height also suddenly stopped increasing for white Americans born between approximately 1955 and 1974, while the height of Europeans continued to grow rapidly (Komlos and Lauderdale, 2007a, b).

II. Data

I study four outcomes which are thought to reflect or be affected by health and human capital, in which puzzling aggregate declines have been noted: educational attainment, wages, birth weight of infants by their mother's age, and mortality rates. I use survey or vital statistics data to measure each outcome by singleyear-of-age for many years. This creates unbalanced pseudo-panels which follow cohorts as they age.

 $^{^{2}}$ Tilstra and Masters (2020) present evidence that declines in birth weight were due to increases in induced labor at earlier gestational ages. These findings could be complementary to mine: either these cohorts are more likely to be induced or the two explanations together could more than explain birth weight declines.

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A. Wages and educational attainment

I use the Current Population Survey, Merged Outgoing Rotation Group (CPS-MORG), from 1979 to 1993, which has been used extensively in studies of wage inequality and trends, and has some notable advantages over other sources.³

I focus on wages of men age 25 to 54. This sidesteps difficulties with large changes in selection for employed women in this period (Goldin, 2006). I calculate the approximate birth year as the survey year minus the respondent's age. I restrict my analysis to cohorts born between 1930 and 1965. I end the analysis in 1993 to avoid issues related to the substantial redesign of the CPS earnings questions in 1994 (Autor, Katz and Kearney, 2005). These restrictions lead to a sample of 970,479 men with non-missing earnings.

I address the problem of topcoding of earnings, by focusing on the median. I calculate the sample median separately for age-year-sex cells, using the survey weights. I follow Chamberlain (1994) and Chetverikov, Larsen and Palmer (2016) and use a two step procedure: first estimating cell medians, and second estimating models on the cell medians. I adjust earnings using the CPI-U-RS.

To study educational attainment I also use the CPS-MORG data, 1979-2016, and include men and women aged 25-75, who were born from 1930 to 1965. I calculate approximate average years of schooling for each cohort based on the 16 schooling categories in the CPS, and calculate the share of each cohort who have achieved different levels of educational attainment.

B. Maternal health

I use the 1968 to 1995 Birth Data Files (National Center for Health Statistics, n.d.). They provide detailed information derived from birth certificates, from a 50 percent sample of all births in 1968, and progressively larger samples until 1985, after which they include the universe of births.

I consider the health of infants as a proxy for maternal health and also to provide evidence of an intergenerational effect of the apparent decline in cohort health. Under the common assumption that a mother's health "endowment" enters the infant health production function (Rosenzweig and Schultz, 1983; Grossman and Joyce, 1990), a decline in the health of infants by *mother's birth cohort* may be viewed as evidence of a decline in maternal health for these cohorts.⁴ I calculate the approximate birth year of each mother as the infant birth year minus the mother's age. I restrict my analysis to births occuring in years years 1968 to 1995, to mothers who were born between 1930 and 1970 and are ages 18 to 40. This results in a sample of more than 75 million births.

³See for example DiNardo, Fortin and Lemieux (1996); Lemieux (2006); Autor, Katz and Kearney (2008); Acemoglu and Autor (2011); and Gould (2014). The underlying data come from the NBER (Bureau Of The Census, n.d.b,n) and cleaning scripts were used from CEPR (Center for Economic and Policy Research, 2020).

⁴Almond and Chay (2006) use this data similarly to study black-white gaps in health.

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I study birth weight as a continuous measure, and the low birth weight percentage, the percentage of infants weighing less than 2500 grams. For all analysis I calculate these measures in cells by year, single age, and birth year using sampling weights.

C. Mortality

I use data from the Human Mortality Database on number of deaths and population-at-risk by year and age. These data are derived from official vital statistics and census estimates. I again define cohort as year minus age. I restrict my analysis to the years 1975-2019, ages 25 to 85, and cohorts born between 1930 and 1965.

III. Evidence of cohort decline from age-period-cohort models

I first present evidence of cohort declines in health and human capital from additively separable age-period-cohort models, common in economics and demography⁵:

(1)
$$Y_{apc} = \gamma_c + \phi_p + \alpha_a + \epsilon_{apc}$$

The objects of interest are the sequence of cohort effects, γ_c . ϕ_p and α_a are full sets of fixed effects in year and age, respectively. ϵ_{apc} is an orthogonal error.

Conceptually, the cohort effects reflect the impact of underlying, fixed differences between individuals born in different years. To identify the cohort effects, the above model assumes that the impact of external factors can be decomposed as additively separable year and age components. It is unrestricted with respect to the dynamics of year-over-year changes impacting each outcome, and with respect to the *shape* of age effects. However, it does not allow age-by-year interactions.

Under these assumptions, the cohort effects answer meaningful counterfactual questions. The difference between the cohort effect for the 1947 cohort, γ_{1947} , and that of the 1960 cohort, γ_{1960} , reveals how the outcomes of individuals born in these two years would have differed — holding all external factors fixed.

This model is not identified due to the exact collinearity of age, period, and cohort (?Deaton, 1997). Identification can be achieved by imposing one additional linear restriction. I take two approaches. First, I estimate "detrended cohort effects", which will reflect the true cohort effect, minus some unknown long-run trend in cohort effects. Second, I will consider the implications for the sequence of cohort effects of imposing additional assumptions on either the age or period effects.

 $^{^5{\}rm Eg.}$ Aguiar and Hurst (2013); Lagakos et al. (2018); Yang (2008); Masters et al. (2014); Masters, Tilstra and Simon (2017); Zang et al. (2018).

Denote the first cohort included in the model as 1 and the last as C, the first year as 1 and the last as P, and the first age included in the model as 1 and the last as A. Define linear "trends" in each as follows:

$$\beta_c \equiv \frac{\gamma_C - \gamma_1}{C}$$
; $\beta_p \equiv \frac{\phi_P - \phi_1}{P}$; $\beta_a \equiv \frac{\theta_A - \theta_1}{A}$

Then define "detrended" cohort, age, and year effects as:

$$\begin{split} \tilde{\gamma}_c &= \begin{cases} 0 & \text{if } c = 1\\ \gamma_c - \beta_c \cdot c & \text{if } c \in (2, C] \end{cases} \\ \tilde{\phi}_p &= \begin{cases} 0 & \text{if } a = 1\\ \theta_a - \beta_a \cdot a & \text{if } a \in (2, A] \end{cases} \\ \phi_p - \beta_p \cdot p & \text{if } p \in (2, P] \end{cases} \end{split}$$

And finally define two composite linear trends:

$$\tilde{\beta}_{pc} = \beta_p + \beta_c ; \qquad \qquad \tilde{\beta}_{ac} = \beta_a - \beta_c$$

One can then rewrite the model in Equation 1 as:

(2)
$$Y_{apc} = \tilde{\gamma}_a + \tilde{\phi}_p + \tilde{\delta}_c + \tilde{\beta}_{ac} \cdot a + \tilde{\beta}_{pc} \cdot p + \epsilon_{apc}$$

This reparameterized model is identified. One can identify detrended age, period, and cohort effects, as well as two composite linear trends which represent i) the sum of the period and cohort trends, and ii) the age trend minus the cohort trend. Under additional assumptions on age or period trends one can identify or bound the cohort trend, and therefore the full set of cohort effects.

A. Detrended cohort effects

Figure 2 shows regression estimates of the detrended cohort effects for six outcomes: men's median log wage, the low birth weight percentage by mother's birth cohort, the log mortality of men and women, and the average years of schooling of men and women. The estimated cohort effects for each of the different outcomes each have an approximately piecewise linear shape with a large trend break located at or near the 1947 cohort, and declines for cohorts born after that year. These patterns are consistent with a large decline in cohort health and human capital, relative to trend, which was broad enough to impact outcomes as disparate as maternal health, wages, and mortality.

Panel A shows results for the median hourly wage of employed men. The estimated cohort effects exhibit a clear piecewise linear shape, with a large trend break precisely at the 1947 cohort. The cohort effects increase from a normalized 0 in 1930 to .14 by 1947, before suddenly changing slope and declining for subsequent cohorts. This pattern suggests a large break in the cross-cohort trend in labor market ability at the 1947 cohort, such that each cohort born after this year has declining ability relative to the trend for prior cohorts. The results imply that the 1965 cohort had a median wage nearly 29 log points lower, ie. 33 percent lower, than they would have had the trend in labor market ability for the 1930 to 1947 cohorts continued.

Panel B shows results for the low birth weight rate of infants by their mother's birth cohort. Recall that age and cohort in these models refer to the age and year of birth of the *mother*. The estimated cohort effects again exhibit a piecewise linear shape: declining rapidly until the 1947 cohort, sharply changing slope after that cohort, and increasing nearly linearly until the 1965 cohort. The cohort effects decline from the normalized 0 in 1935 to a minimum of -.87 for the 1947 cohort, before reversing trend. The results imply a large break in the cross-cohort maternal health trend at the 1947 cohort, such that each cohort born after this year has declining health relative to the trend for prior cohorts. The size of the trend break suggests than the 1965 cohort would have had a low birth weight rate approximately 2.2 percentage points lower had the cohort health decline not occurred.

Panels C and D show similar results for the log mortality rate of men and women. The shape of the estimated cohort effects are not as near piecewise linear as those for the labor market and maternal health outcomes. However, they exhibit clear changes in trend near the late 1940s cohorts, consistent with elevated mortality and declining health for subsequent cohorts. For men the cohort effects decline — not precisely linearly – from 0 to below -.1 by the 1947 cohort, then suddenly reverse trend and increase rapidly until reaching above .05 by the late 1950s cohorts. They then flatten and decline slightly for subsequent cohorts. This pattern suggests that men born in 1960 had mortality near .25 log points higher than they would have had health improvements continued at the same rate as for the 1930 to 1947 cohorts. For women the cohort effects exhibit two smaller trend breaks at the 1947 and 1951 cohorts, but still show evidence of a decline in health after the late 1940s relative to the prior trend. The cohort effects decline from 0 to below -.11 by the 1947 cohort, they then change trend and are nearly flat until the 1951 cohort. They then change trend again after the 1951 cohort and increase nearly linearly to around 0 by the 1960 cohort. The size of the two trend breaks imply that the 1965 cohort has had nearly .25 log points higher than it would had the health improvements for the 1930 to 1947 cohorts continued at the same rate for later cohorts.

Panels E and F shows results for the average years of schooling of men and women. Again, the estimated cohort effects appear approximately piecewise linear in shape, with a large trend break precisely at the 1947 cohort. For men, the cohort effects increase from a normalized 0 in 1930 to .83 by 1947, before suddenly changing trend and declining for subsequent cohorts. For women the pattern is similar though the differences in cohort effects are smaller in magnitude; and the 1947 trend break is smaller in magnitude but followed by a second trend break in the early 1950s (similar to the pattern for women's log mortality). Appendix Figure 2 shows results separately for the share of each cohort with a high school degree, bachelor's degree, and advanced degree. Consistent with the prior literature reviewed above, they all show evidence of a clear, large trend break at or near the 1947 cohort.

B. Cohort effects under additional assumptions

RESTRICTIONS ON PERIOD TREND

For low birth weight and log mortality I consider the implications for cohort effects of different restrictions on the period trend. Lagakos et al. (2018) call this the "Deaton-Hall" approach in reference to ? and Deaton (1997). By restricting the trend in period effects in different ways, one can pin down the trend in cohort effects based on the composite period/cohort trend defined above.⁶

I consider three benchmark cases: i) assuming that the period trend, β_p , is equal to 0, ii) assuming that the cohort trend, β_c , is equal to 0, and an intermediate case iii) assuming that β_p and β_c are equal.⁷ Many other cases are possible, but examining what these assumptions imply about the sequence of cohort effects can help to make the mechanics of the model and its underidentification clear.

It is arguably natural to think of case i) as an upper bound on the period trend, and therefore a lower bound on the cohort trend. For example, for low birth weight assuming $\beta_p \leq 0$ is assuming that average growth in period effects between 1968 and 1995 is non-positive, in other words that over this period there was at a minimum not a worsening of external factors which impact the low birth weight rate. For mortality analogously, it would be assuming that between 1975 and 2019 the external environment impacting mortality rates did not worsen. Therefore we can view the sequence of cohort effects in case i) as a "best case" scenario for cohort effects, in the sense that they represent the most that cohort effects impacting low birth weight or mortality could have declined between the 1930 and 1965 cohort (or any two cohorts). Under this assumption the true sequence of cohort effects must lie above the case i) sequence, with the case ii) and iii) sequences representing two other possible sequences above the lower bound. Though note case ii) is likely not a natural upper bound.

Figure 3, Panel A shows cohort effects under the different scenarios for the low birth weight percentage. The bottom series shows cohort effects under case i), assuming that β_p , is equal to 0. Even under this assumption, which could be viewed as the "best case" for the long run trend in cohort effects, the cohort effects increase between the 1947 and the 1960 cohort by more than .25. This would imply that maternal health declined across these cohorts enough to increase the low birth weight percentage for these mother's by more than .25 percentile points. The series based on scenarios ii) and iii) imply much *worse* declines in maternal health between the 1947 and 1965 cohorts.

⁶See also Fosse and Winship (2019).

⁷Case ii) will yield the "detrended" cohort effects presented above.

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Panels B and C show results for men's and women's log mortality. The bottom series shows cohort effects under case i), assuming that β_p , is equal to 0. The "best case" for the long run trend in cohort effects implies a small set of cohorts across which cohort effects were worsening in absolute terms. For men they would imply that the impact of cohort health on log mortality was worsening between the 1947 and 1952 cohort, before beginning to improve again. For women, they would imply that the impact of cohort health on log mortality was worsening in absolute terms only between the 1951 and 1957 cohorts. The series based on cases ii) and iii) imply much larger increases in cohort effects after the 1947 cohort, and a larger range of cohorts across which cohort health was worsening.

No growth in wage age effects after 50

It seems hard to argue that the trend in period effects for wages is definitely non-positive. I therefore adopt what Lagakos et al. (2018) call the Heckman-Lochner-Taber approach (Heckman, Lochner and Taber, 1998), and impose the theory-guided assumption of no growth in age effects in the last few years of a workers career (or a slight decline due to eg. human capital depreciation).

I first estimate a version of the age-period-cohort model but restrict the age effects from ages 50 to 54 to be equal to 0. This represents a first benchmark case i) of no growth in age effects between ages 50 and 54. I then consider two additional cases in which human capital depreciation (or other factors) lead age effects between age 50 to 54 to i) decline by .005 per single-year-of-age, and ii) decline by .01 per single-year-of-age; corresponding to human capital depreciation rates of .5 % and 1 % respectively.

The cohort effects implied by these three scenarios are shown in Panel D of Figure 3. All series imply an *absolute* decline in labor market skill between the 1947 and 1965. Though the implied magnitude of the decline differ: ranging from being enough to reduce log wages by .16 to by just .02.

IV. Evidence of cohort decline allowing for smooth age-by-year interactions

The above results from the age-period-cohort models are striking, but could be biased by external factors which disproportionately impact individuals of particular ages in particular years.

Therefore, I present evidence of a decline in cohort health and human capital using a novel methodology, which allows me to identify a trend break in cohort effects while allowing for smooth changes in external, age-specific factors across years.

Consider the following model:

(3)

$$Y_{apc} = \underbrace{\beta \cdot c}_{\text{long-run trend in cohort effects}} + \underbrace{1_{c \ge \lambda} \cdot \delta \cdot (c - \lambda)}_{\text{trend break in cohort effects}} + \underbrace{f^p(a)}_{\text{year-specific impact of age}} + \epsilon_{apc}$$

The first two terms on the right-hand-side specify the cohort effects as piecewise linear with a single, *unknown* trend break. β represents a long-run trend in cohort effects. The second term introduces a trend break in cohort effects. δ represents the size of this trend break, and λ represents the unknown cohort at which the break occurs. This specification of the shape of cohort effects is similar to that seen visually in the detrended cohort effects estimated above. I assume that the changing external age-by-year factors, $f^p(a)$, take the form of a polynomial of known order in each year. ϵ_{apc} is an orthogonal error.

The location and size of the trend break are still identified with the introduction of separate polynomials in age *in each year*. The sharp trend break in cohort effects is orthogonal to the smooth, polynomial age-by-year interactions.

I estimate the model by least squares, following the structural break methodology in Hansen (1999, 2000). The location of the trend break of these piecewise linear cohort effects is treated as an unknown parameter to be estimated. My baseline specification includes age fixed effects, year fixed effects, and a separate quadratic-in-age in each year as controls. In robustness checks, I include higher order polynomials in age in each year.⁸

Intuitively, I allow external factors which impact individuals of different ages to change but restrict them to do so smoothly. Most alternative explanations I want to rule out would take such a smooth form. For example, changes in the supply or demand for workers of different experience levels will have a smooth impact across ages, as long as individuals of nearby ages are sufficiently close substitutes. If the biological aging process is smooth then changes in the disease environment may disproportionately increase the mortality of young adults, but they will do so smoothly.

Table 1 shows the results. The estimated location of the cohort break are centered at the 1947 and 1948 cohorts. For both of the maternal health outcomes, infant mean birth weight and share low birth weight, the trend break is estimated to occur at the 1948 cohort. For both of these outcomes, the 99 percent confidence interval includes *only* a single cohort. For the men's median log wage it is estimated to occur at the 1947 cohort, with a confidence region including only 2 cohorts — 1946 and 1947. Cohort effects in models of the log mortality of men and women are estimated to have a trend break at the 1947 and 1949 cohorts respectively. The estimated cohort break location for years of schooling are 1948 and 1950 respectively. The estimated break locations for mortality and education are very precisely estimated — with the 99 percent confidence interval including only a single cohort.

The estimated trend breaks in cohort effects for all outcomes are large in magnitude and precisely estimated. For a sense of magnitude, they would imply for example that had the cohort decline not occurred: the median man in the 1960

 $^{^{8}}$ For educational attainment I only include age fixed effects as external year factors and age-by-year factors do not seem likely to drive educational attainment after age 25. I restrict the location of the break to not be one of the 5 youngest or oldest cohorts.

cohort would have a wage 23 percent higher, infants born to women in the 1960 cohort would've been 2.8 percentage points less likely to be low birthweight, and the single-year mortality risk for men and women in the 1960 cohort would have been roughly 1.5 times lower.

For all six outcomes, I fail to reject null hypothesis of no break in the cohort trend at a very low significance level, using the bootstrap based test described in Hansen (1996, 2000). Appendix Table 1 shows that these estimates are generally quite robust to different specifications of the age-by-year control function.

V. Role of cohort decline in year-over-year declines

I use the cohort trend break estimates from the previous section to quantify the role of the cohort decline in driving year-over-year declines. The results are dependent on the model, and therefore do no allow for (potentially plausible) interactions between cohort human capital and period-specific factors.

Using the parameter estimates from the cohort trend break models above, I create a counterfactual dataset, where each observation takes the following form: $\tilde{Y}_{apc} \equiv Y_{apc} - 1_{c \geq \hat{\lambda}} \cdot \hat{\delta} \cdot (c - \hat{\lambda})$. I then compare summary measures of each outcome by year in the counterfactual dataset, in which the estimated cohort trend break has been removed, to the raw data.

Figure 4, Panel A shows that while the low birth weight rate declined from above 8 percent in 1968 to 6.7 percent by 1985, it then reversed trend and increased to near 7.3 percent by 1995. In contrast, the transformed series in which the cohort trend break has been removed exhibits no similar increase. The series implies that absent the cohort health decline, the low birth weight rate would have declined much more rapidly until 1985 — falling to 5.7 percent by that year. These improvements would have slowed after the mid-1980s — but the low birth weight rate would have continued to decline slowly until 1995 to just below 5.5 percent.

Panel B shows similar results for men's "age-adjusted median wages," the average across median wages of single ages 25 to 54. This measure of wages declined in real terms from 1979 to 1993, from 3.14 to below 3.04 — from 23 to below 21 dollars an hour in 2014 dollars. In contrast the counterfactual log wage series, in which the cohort trend break is removed, increases slightly from 3.16 in 1979 to 3.17 in 1993.

Panel C and D show analogous results for the age-adjusted mortality rates of men and women age 45 to 54, the ages focused on in Case and Deaton (2015). The raw data show: declines in men's and women's midlife mortality between 1975 and 1990; a "bump" during the AIDS epidemic, larger for men; then a clear slowing of mortality improvements after 1999. For men the mortality rate even increases between 1999 and the mid-2000s. The change in trend is completely absent in the transformed series in which the cohort trend break is removed. That is, the decline in cohort health can completely explain the stagnation, and for men slight increase, in mortality after 1999. Panels E and F show that the mortality rates of men and women, aged 55 to 64 both declined between 1975 and around 2010, before changing trend and increasing. Again the change in trend is absent in the counterfactual series in which the cohort trend break is removed — suggesting the mortality increases for this group are also plausibly driven by the cohort health decline.

VI. Conclusion

In this paper I present evidence of a precisely timed and broad decline in health and human capital across cohorts of Americans beginning with those born in 1947 and continuing until at least those born in the mid-1960s. This decline appears to have played a key role in: education declines in the 1960s and 1970s, increases in the low birth weight rate beginning in the mid-1980s, wage stagnation since the 1970s, and recent mortality increases. The cohort decline had an intergenerational effect through its effect on the health of mothers and their infants.

The large magnitude of the cohort decline and the sharpness of the break in trend cry out for a similarly large and sharp break in some causal factor. The cohort decline was also widespread across demographic groups born in the US, suggesting any causal explanation should have a broad reach. Appendix Tables 4 and 5 show for most outcomes it is evident across racial groups and across Census Regions (the one exception being black men's mortality). Reynolds (2023 a) shows further evidence of the decline's wide geographic reach.

A number of results suggest the cohort decline likely originated in adolescence or earlier. High school completion declined for these cohorts (Appendix Figures 1 and 2). Standardized test scores taken at 16 and 17 declined for the same cohorts — part of a widely noted decline beginning in the late 1960s (eg. National Commission on Excellence in Education, 1983; Koretz, 1987). Appendix Figure 3 shows that SAT scores decline beginning with approximately the 1947 cohort. Similar declines occurred in other achievement tests, including nearly universal tests in select states (Harnischfeger and Wiley, 1975). Appendix Figure 4 and Appendix Tables 2 and 3 show evidence that the cohort decline in health and human capital is concentrated among the native born and not evident for foreignborn US residents. In Reynolds (2023*a*) I show that the onset of puberty appears to have been delayed for approximately the same cohorts.

Two broad candidate hypotheses appear most plausible to me, and could generate an aggregate trend break with the correct timing. Reynolds (2023a) and Reynolds (2023b) present aggregate evidence against a number of other ex ante plausible hypotheses.

The first broad hypotheses is that the complex demographic process of the baby boom led to the cohort decline. Many have hypothesized that the large baby boom cohorts would be disadvantaged by a "cohort-crowding" mechanism: receiving less and lower quality schooling and having their wages driven down by the large labor supply shock of their entry (eg. Freeman, 1979; Easterlin, 1987; Bound and Turner, 2007). The simplest version of such a theory cannot match the

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timing of the cohort decline: cohort size increased dramatically in 1946 and 1947, but these cohorts' health and human capital was still *improving*. Macunovich (2002) hypothesized a "bottleneck" mechanism in which later baby boomers are worst off because they must compete for initial jobs with earlier-born boomers who are still trying to establish their careers, which could more closely match the timing of the cohort decline (though would need an additional expectations-based channel to explain pre-labor-market-entry declines). Observable characteristics of parents, such as education and occupational status, continued improving steadily for post-1947 cohorts. However, the cross-cohort trend in average birth order has a break in 1946 and the share of children who are later born in their families increased until the mid-1960s cohorts.⁹

The second candidate hypotheses is lead pollution from motor vehicle exhaust, discussed in detail in Reynolds (2023*b*). Motor vehicle use began to increase rapidly after 1945 and lead additives were ubiquitous in gasoline. By the 1960s children across the US had blood lead levels well above those now considered safe.

Sharp tests of these and other hypotheses will require large samples which link granular information on individuals' early life circumstances to their outcomes as adults.

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 9 Other work suggests that these changes explain around one third of the decline in Bachelor's completion for white men (Handy and Shester, 2019) and of the increase in white mortality (Reynolds, 2023*a*).

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TABLE 1—EVIDENCE OF RELATIVE COHORT DECLINE — COHORT TREND BREAK MODELS WITH UNKNOWN BREAK LOCATION

	<u>Trend break in cohort effects</u>		
	Size	Location	Existence
	δ	λ	p-value
Intergenerational infant health	0.044		0.01
Low birth weight $(\%)$	0.241 (0.014)	$\frac{1948}{[1948, 1948]}$	< .001
Mean birth weight (g)	-6.35 (0.35)	$1948 \\ [1948, 1948]$	< .001
Labor market			
Median log wage, men	-0.016 (0.001)	$\begin{array}{c} 1947 \\ [1946, 1947] \end{array}$	< .001
Log mortality			
Men	0.028 (.001)	$\begin{array}{c} 1947 \\ [1947, 1947] \end{array}$	< .001
Women	0.029 (.001)	$1949 \\ [1949, 1949]$	< .001
Years of schooling only controlling for year FEs, age FEs			
Men	-0.106 (.001)	$\frac{1948}{[1948,1948]}$	< .001
Women	-0.074 (.001)	1950 $[1950, 1950]$	< .001

controlling for year FEs, age FEs, and separate quadratic-in-age in each year

Each row shows the results of estimation of a model based on Equation 3, with the listed outcome as a dependent variable. All models are estimated by least squares, following the approach outlined in Hansen (1999, 2000). All models except those for years of schooling control for year fixed effects, age fixed effects, and a separate quadratic-in-age in each year. The years of schooling models only control for age FEs. The column titled "Size" reports the estimated size of the cohort break δ , with the standard error in parentheses. The column titled "Location" reports the cohort at which the trend break is estimated to occur, with a 99 % confidence interval in brackets calculated by inverting a likelihood ratio statistic. The column titled "Existence" reports a p-value from an F-type test, based on 1000 bootstrap samples, for the null hypothesis that no trend break occurs, ie. that cohort effects are linear. Intergenerational infant health results are based on Birth data files, National Center for Health Statistics (n.d.), 1968-1995, mothers age 18-40 who were born between 1930 to 1970. Labor market results are based on CPS-MORG data, 1979-1993, (Bureau Of The Census, n.d.b,n) and includes men age 25-54, who were born from 1930 to 1965. Log mortality results are based on data from the Human Mortality Database (Max Planck Institute for Demographic Research, University of California, Berkeley and French Institute for Demographic Studies, n.d.), and include the years 1975-2019, ages 25-85, cohorts born from 1930 to 1965. Years of schooling results are based on CPS-MORG data (Bureau Of The Census, n.d.b,n), 1979-2016, and includes men and women aged 25-75, who were born from 1930 to 1965.

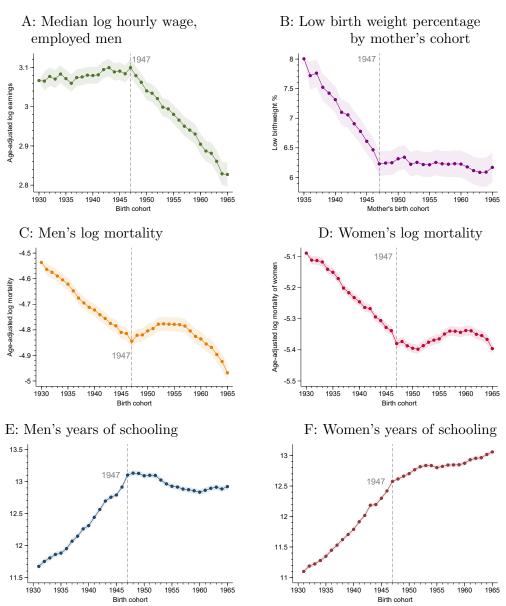


FIGURE 1. AGE-ADJUSTED OUTCOMES BY BIRTH COHORT

Each panel plots age-adjusted outcomes by birth cohort. Age adjustment is done by regressing the outcome on a full set of cohort fixed effects and age effects, and plotting predicted values. Panel A is based on CPS-MORG data (Bureau Of The Census, n.d.b,n), 1979-1993, and includes men age 25-54, who were born from 1930 to 1965. Panel B is based on Birth Data Files, National Center for Health Statistics (n.d.), 1968-1990, mothers age 18-40, who were born from 1935-1965. Panel C and D are based on data from the Human Mortality Database (Max Planck Institute for Demographic Research, University of California, Berkeley and French Institute for Demographic Studies, n.d.), and include 1975-2019, ages 25-85, cohorts born from 1930 to 1965. Panels E and F are based on CPS-MORG data (Bureau Of The Census, n.d.b,n), 1979-2016, and includes men and women aged 25-75, who were born from 1930 to 1965.

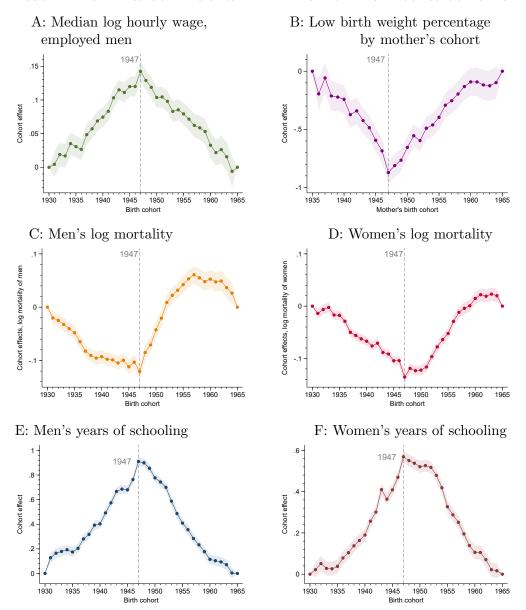
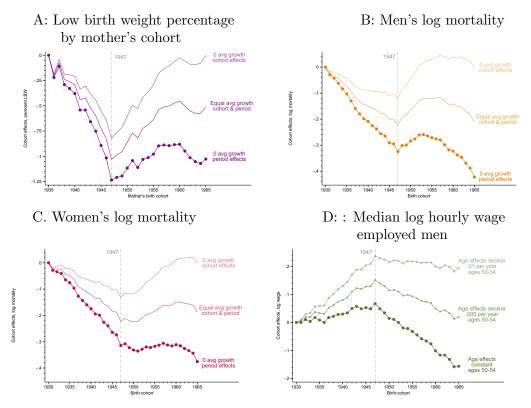


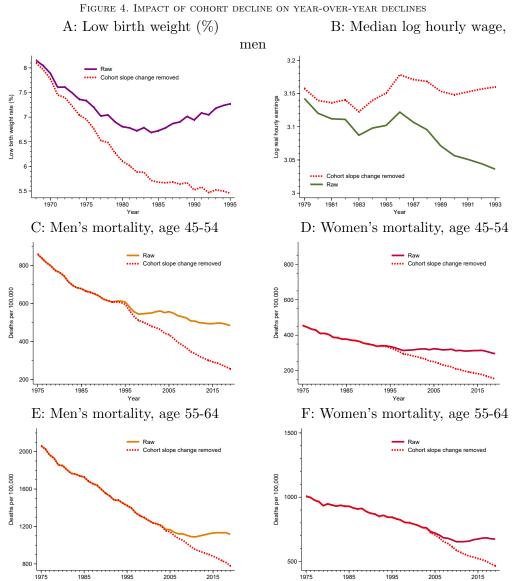
FIGURE 2. DETRENDED COHORT EFFECTS FROM ADDITIVELY SEPARABLE AGE-PERIOD-COHORT MODELS

Each panel plots detrended cohort effects from estimation of age-period-cohort models based on Equation 2. Data sources and sample restrictions for each outcome are the same as in Figure 1.

FIGURE 3. COHORT EFFECTS UNDER DIFFERENT ASSUMPTIONS, ADDITIVELY SEPARABLE AGE-PERIOD-COHORT MODELS



Each panel plots cohort effects from estimation of age-period-cohort models based on Equation 2. Each series shows cohort effects under different assumptions on the age effects, or the average growth in period/cohort effects. See Section 5b. Data sources and sample restrictions for each outcome are the same as in Figure 1.



This figure shows counterfactual year-over-year trends in 4 outcomes had the treffd break in cohort effects not occurred, ie. if the pre-break cohort trend had continued. The trend break estimated based on the model in Equation 3 is subtracted from each observation. These transformed data are then used to calculate each of the listed outcomes, which are plotted as the dashed red line. The same outcome based on the untransformed, raw data is plotted as a solid line. Panel A shows the percent of infants born at low birth weight. Panel B shows the average across men age 25-54 of median wages for single age-bins. Panels C through F show mortality rates of men and women, age-adjusted assuming a uniform population distribution by single year of age. Data sources for each outcome are the same as for Figure 1.