Increased mortality of white Americans and a decline in the health of cohorts born after World War II

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Abstract

I show evidence that recent increases in the mortality of white Americans are rooted in a sharp decline in the health of cohorts born after World War II, relative to the trend for earlier-born cohorts. These cohort health differences are evident by the 1980s, suggesting recent mortality increases have deep roots which predate the opioid epidemic and recent economic distress. I identify the role of cohort health by imposing the impact of age on mortality to follow the log-linear, Gompertz form. This imposition yields the sharp, falsifiable prediction — strongly borne out in the data — that a decline in cohort health will result in changes in the slope of the age profile of log mortality at the same cohort in each year. That is, log mortality rates in *every* year between 1985 and 2019 exhibit slope changes centered at the 1946 cohort for white men and the 1949 cohort for white women, consistent with a health decline beginning precisely with those cohorts. The size of these slope changes imply that the average mortality rate of the 1960 cohort of white women has been 22 percent higher and that of men 37 percent higher, than they would have had health followed the trend for earlier-born cohorts. Any explanation of the root cause of recent increases in mortality must be able to explain this sharp cohort pattern. I close the paper by beginning the search for such a root cause.

Keywords: mortality | cohorts | white Americans

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1 Introduction

Case and Deaton (2015) document a disturbing trend of increasing mortality among 45 to 54 year old non-Hispanic white Americans since 1999. The initial discussion of a possible cause focused on factors that changed between 1999 and 2012, such as the increasing availability of prescription opioids and heroin and changing economic conditions for those without a college degree. Subsequent research has suggested instead that the mortality increase may be due to cohort differences in health and disadvantage which predated the 1990s (Case and Deaton, 2017; Lleras-Muney, 2017; Masters et al., 2017; Zang et al., 2018). That is, white Americans born between 1958 and 1968 — and therefore aged 45 to 54 in 2012 — may be less healthy on average than those born between 1945 and 1955 — who comprised the 45 to 54 year old age group in 1999.

I provide strong evidence and a detailed investigation of this cross cohort decline in health and its role in mortality increases of white Americans. I show that the age profile of log mortality departs from its usual linearity, known as Gompertz law or the Gompertz-Makeham law (Gompertz, 1825; Olshansky and Carnes, 1997; Chetty et al., 2016). The age profile is instead kinked, at a different age in each year. These kinks, or sharp slope changes, have a specific staggered structure by age across years, such that they are consistently located at (or very near) the 1946 cohort for men and the 1950 cohort for women. In each year between 1985 and 2019, log mortality rates are very close to linear in age between the 1930 and 1946/1950 cohorts. Then for each cohort born after 1946 (for men) or 1950 (for women) mortality is increasingly elevated relative to what the Gompertz law fitted to earlier cohorts would predict. These relative mortality increases are particularly steep until around the 1960 cohort when they began to taper off.

This staggered, kinked pattern is remarkably consistent with a model in which i.) the effect of age on log mortality is linear, and ii.) the trend in health across cohorts hit a sudden stop and health declined for subsequent cohorts relative to the prior trend. Under this assumption of a linear age effect, my evidence suggests that there was a break in the cross cohort health trend near the 1946 cohort for white men and near the 1950 cohort for white women. Health improvements across successive cohorts born between 1930 and the late-1940s suddenly stopped in the late-1940s. Instead, white Americans in each cohort born between the late-1940s and the mid-1960s appear to be *less* healthy than those born a year earlier, at least relative to the pre-break trend.

Any explanation of the root cause of recent mortality increases needs to be able to match these sharp cohort patterns. It also must take seriously the fact that the health decline was already evident in 1985, suggesting that these cohorts were already in worse health by their 30s. Recent expansions in opioid supply may have exacerbated the suffering of the cohorts born after 1946 but they cannot

be the root of these cohorts' decline. Refinements of Case and Deaton's preliminary theory of "cumulative disadvantage" would need to be able to explain why declining in the demand for unskilled labor which began in the 1970s so negatively affected men born after 1946 and women born after 1950, but had a much smaller effect on those born even a year or two earlier.

Consider an example of the model described above, and the staggered kinks in the age profile of log mortality it would produce. A decline in the health of cohorts born after 1946 would result in a slope change at age 39 in 1985, age 40 in 1986, age 41 in 1987, age 42 in 1988, etc. — with the age at which the slope change occurs increasing by one with each subsequent year — culminating in a slope change at age 73 in 2019. The existence of such particular non-smooth non-linearities by age across years would be remarkably hard to attribute to other differences in the health environment unrelated to cohort.

I show that these precise predictions, regarding staggered slope changes in the age profile of log mortality, are strongly borne out in all years between 1985 and 2019. For example, Figure 1 shows the log mortality rate of white men for 1985, 1995, 2005, and 2015, along with estimated piecewise-linear models. In each year the age-profiles exhibit kinks similar to the staggered structure described above: at age 40 in 1985, age 48 in 1995, age 60 in 2005, and age 68 in 2015. This staggered location of slope changes by age corresponds to a nearly identical location by cohort, with the estimated breaks at the 1944 cohort in 1985 and 2005, and at the 1946 cohort in 1995 and 2005. These example years are not unique. Estimation of piece-wise linear models of unknown location, using the structural break methodology of Hansen (1999, 2000) reveals strong statistical evidence that in *all years* between 1985 and 2019 a slope change exists and is located at or near the 1946 cohort. Similarly, for white women the change in slope is located at or near the 1949 cohort across all years. [1]

Motivated by these reduced form patterns consistent with a decline in cohort health, I estimate models of log mortality including linear age effects in each year and a trend break in cohort effects. As suggested by the graphical patterns and within-year regressions, I estimate a trend break in log mortality to occur at precisely the 1946 cohort for white men, and between the 1948 and 1950 cohort for white women. The trend break is also large: the smallest estimate across specifications suggests that the 1958 cohort of white women has had on average approximately 22 percent higher mortality rates than they would have absent the trend break. Similarly, it implies that the 1956 cohort of white men had on average approximately 28 percent higher mortality rates than what

[1] For women, in all 35 years the confidence interval for the location of the slope change includes the 1949 cohort and the point estimate is never more than three years away from 1949. For men, the fit is slightly less tight: for 30 of the 35 years the confidence interval for the location of the slope change includes the 1946 cohort — with a period between 2006 and 2012 where the slope change estimates are a few cohorts "too early."

would be predicted by the trend for preceding cohorts. These findings are also robust to the inclusion of higher-order polynomials in age — up to including a separate cubic-in-age in each year — validating the assumption of linearity against an alternative of smooth but non-linear age-effects.

The cohort-specific explanation can also explain the staggered timing of recent increases in mortality of different age groups. It explains the fact that white women's mortality rate at ages 35-44 began to increase in the early 90s, that of those age 45-54 in 1999, and that of those age 55-64 only in 2001 — unhealthy post-1949 cohorts drive mortality increases first at young ages and subsequently at older ages, as they age into different age bins. I perform a simple simulation exercise validating this intuition: the cohort-specific trend break model, without any other non-linear age-by-year interactions, can match this staggered timing for white women. The year-over-year pattern of the mortality rate of white men by age show less prima facie evidence of cohort effects, but the cohort-specific trend break model also fits these patterns well for men. While these simulations point to an important role for cohort factors in year-over-year patterns, my identification strategy does not allow for a strict decomposition of the role of cohort versus period factors. Future research should explore how interactions between the cohort decline in health and year-specific factors, such as the AIDS epidemic and the recent expansion of opioid supply, ultimately produced the observed mortality patterns.

Interestingly, there is some evidence of a similarly timed, but smaller, health decline for Americans of other racial groups — expanding the set of possible causes. The data is much noisier, precluding the full analysis I conduct for whites, but pooling all years in a single models yields evidence of a health decline beginning near the 1946 cohort for black women, and women and men of other races, but not for black men. In years after 1997, when its possible to distinguish, a similar decline is not evident for Hispanics — it is concentrated among non-Hispanic whites. The cross-cohort decline in health is also remarkably widespread geographically across the United States, with no obvious regional patterns in the size of breaks across states. I also show that rapid improvements in mortality rates under the age of 30 suddenly slowed for the same cohorts, at a minimum suggesting the adult mortality effects are not driven by mortality selection.

I conduct a preliminary investigation into the causes of the cross cohort health decline. There are three main findings. First, these cohorts have lower educational attainment. The timing of previously document declines in the share of each cohort with a high school or college college degree (Heckman and LaFontaine, 2010; Card and Lemieux, 2001b) are remarkably similar to the timing of the mortality increases. However, the educational declines appear unlikely to be large enough to directly explain the mortality increases. This could suggest that these cohorts health and "human capital" had already declined by age 17 — however other proxies of early life health

did not decline (though some suddenly stagnated). Second, the average birth order (or parity) of children also changed trend sharply in 1946, such that after that year an increasing share of children were the 3rd or later born child in their families. Simulations suggest this could explain around one-third to one-half of the mortality increase. Third, motor vehicle use increased sharply after 1945 and the gasoline used in motor vehicles included large and increasing quantities of lead additives. Fetal and early life lead exposure has been linked to poor health and cognitive development, and high child blood lead content was ubiquitous across the US when these cohorts were children [2] Observable characteristics of where these cohorts grew up and their family background, maternal smoking, cohort crowding, other forms of pollution, early life mortality selection, and early career labor market conditions all appear less likely to be important causes — though more research is needed.

The theory advanced in this paper also has the testable prediction that the age profile of log mortality should continue to exhibit the same staggered slope changes, evident as each new year of data is released, with slope changes continuing to occur at or near the 1946 cohort for white men and the 1949 cohort for white women. The initial draft of this paper only used data up to 2015 — so the theory has already "passed" four years of falsification tests in that the pattern still holds through 2019. The cohort "kink" may even be evident in the age profile of COVID-19 mortality, with cause-specific mortality rates from this virus of white men and women born after 1946 and 1949, respectively, being higher than expected given their age.

My paper contributes to the broad literature in economics and social science on the identification of age, period, and cohort effects.[3] This so-called age-period-cohort identification problem is usually solved by imposing additive separability of each factor plus an additional, often ad hoc, restriction. This usual approach can feel like a "black-box" — with no methods to assess credibility and little attention to model fit. The key advantage of my alternative approach is that it yields falsifiable predictions which allow for intuitive overidentification tests. Of course my approach also imposes different assumptions to achieve identification — the credibility of which must be assessed in any given setting. While, the usual approach is unrestrictive with respect to the shape of the age, period, and cohort effects respectively; it precludes any interactions across these effects

[2] See eg. McMichael et al. (1986); Needleman (2004); Hollingsworth et al. (2022); Aizer et al. (2018) on the health and cognitive effects of fetal and childhood lead exposure and Pirkle et al. (1994); Annest et al. (1983) for estimates of children's blood lead content. McFarland et al. (2022) estimate that the share of children with blood lead content above the 2015 threshold for "clinical concern" increased from 50 percent for the 1940-45 cohorts to 100 percent for the 1966-75 cohorts.

[3] See for example Hall (1968); Deaton (1997); Heckman and Robb (1985); Mason and Fienberg (2012) for methodological discussions; and Lagakos et al. (2018); Chay et al. (2014); Aguiar and Hurst (2013) for recent applications in economics.

— for example the impact of age is assumed to be fixed across years. My approach is more restrictive in one dimension by imposing a parametric form on the age effects, but less restrictive in allowing this parametric impact of age to be different in each year. My approach may be useful in other economic applications. For example, it could be used to test for cohort differences in unobserved "skill", in the context of Mincerian wage regressions in which the impact of experience is traditionally assumed to be quadratic (Mincer, 1974; Card and Lemieux, 2001a).

In addition to Case and Deaton (2015), my paper contributes to a larger literature in demography documenting patterns in the all-cause mortality rate of white Americans.[4] Yang (2008) and Masters et al. (2014) emphasize the importance of a cohort-perspective and estimate additively-separable age-period-cohort models of mortality whose results suggest that mortality declines since 1960 are largely driven by cohort factors. Masters et al. (2017) and Zang et al. (2018) apply these standard models to more recent data and find evidence for an important role for cohort factors in recent mortality increases. As noted above, my approach to identifying cohort effects builds on the standard approach used in these papers, with the advantage that it yields falsifiable predictions. My result therefore adds additional credibility to these claims of an important role for cohort factors in recent mortality increases. I also identify more precisely the cohort at which the health decline appears to have begun.

2 Data

I use vital statistics data derived from death certificates and population estimates from the Census Bureau to calculate mortality by single age and year.

I use the Multiple Cause of Death File from the Center for Disease Control and intercensal population estimates from the Census Bureau and the Surveillance, Epidemiology, and End Results (SEER) Program of the National Cancer Institute. Using these sources I calculate mortality and mid-year population by single-age, race, and sex. I then calculate crude death rates — the ratio of mortality over mid-year population — by single-age, race, and sex cells. I then define birth cohort as $year - age - 1$ [5]

My main analysis considers the aggregate mortality rate of white males and females of the

[4] A large literature also examines widening gaps in mortality by education level, see for example Meara et al. (2008), Olshansky et al. (2012), and Sasson (2016). My study differs from this literature, and follows Case and Deaton (2015), by focusing on an increase in the mortality rate of the aggregate population of white men and women of all education levels.

[5] This will introduce some measurement error into the birth cohort variable. If any thing this seems likely to lead to a slight understatement of the size of the trend break, because eg. the mortality rate of the 1946 cohort will be affected slightly by the mortality rate of the 1947 cohort.

United States by single year of age. Hispanic-origin was not reported on death certificates in all states until 1997, and is therefore not consistently recorded in the Multiple Cause of Death File until that year. I therefore focus primarily on mortality rates for all whites, including Hispanics and non-Hispanics.

In some supplementary analysis, I consider mortality rates of Hispanic and non-Hispanic whites separately for 1997-2019. I construct these series by single age and year using an analogous approach and the same data source as described above. For comparative analysis I also construct mortality rates for blacks and an "other race" group using the same approach and sources as for whites.

In addition to national mortality rates, I construct estimates of the mortality rates of white Americans separately for each of the 50 states. These estimates allocate deaths based on the state of *residence* reported on death certificates. I again use intercensal population estimates from SEER as the denominator.

I also digitize historical mortality data from annual vital statistics publications for the years before microdata became available in 1959, allowing me to study the mortality rates of the relevant cohorts early in life in Section 8. I digitized death counts by age-sex-race from the tables of historical vital statistics volumes from 1933 to 1958. I combine these with population estimates from the Census Bureau from 1933 to 1968[6]. Combined with the above described sources this allows me to calculate mortality rates for age-sex-race cells for 1933-2019.

I use a number of additional data sources, described in more detail below, in the preliminary investigation into the cause of the cohort decline in Section 9. This includes data from the Current Population Survey measuring educational attainment, data from the General Social Survey measuring parental characteristics and family background, estimates of the smoking prevalence of women of childbearing age from Holford et al. (2014), cause-specific mortality digitized from the above-described historical vital statistics volumes, and the distribution of births by birth order (or "parity") over time from vital statistics volumes and Heuser (1976).

3 Evidence of cohort-specific trend breaks in log mortality

I show that the mortality rate of white men and women has deviated, in a way that is systematically related to cohort, from its usual log-linear relationship with age, the Gompertz curve. In each year since 1985 those born after the 1946 cohort for men and 1949 cohort for women have higher log mortality rates than the Gompertz curve would predict.

[6]For each year I use the estimates titled "Resident Population–Estimates by Age, Sex, and Race: July 1."

3.1 Methodology

I use the structural break estimation and testing framework of Hansen (2000) to provide formal evidence of a break in the cross-cohort mortality trend.

Consider the following model of log-mortality:

$$ln(mort_{apc}) = \beta_a^p a + \beta_c^p c + \delta^p \cdot (\gamma^p - c) \cdot 1_{c \geq \gamma^p} + \mu^p + \epsilon_{apc} \tag{1}$$

where a denotes age, p denotes period (eg. year), c denote cohort; and $ln(mort_{apc})$ denotes the log-mortality rate of individuals age a, in period p, and from cohort c. The parameters β_a^p and β_c^p represent linear trends in age and cohort, respectively, in each year. I then allow in each year for a trend break by cohort — thereby letting the affect of cohort have a piecewise linear form. The size of the trend break is represented by δ^p. The precise cohort at which the trend break occurs is treated as unknown and a parameter to be estimated, γ^p. μ^p is a year-specific intercept, and ϵ_{apc} is an orthogonal error.

Because age and cohort are perfectly collinear in each year of data the linear trends in age and cohort, β_a^p and β_c^p, are not separately identified. However, the following transformed model is identified:

$$ln(mort_{apc}) = \tilde{\beta}^p a + \delta^p \cdot (\gamma^p - c) \cdot 1_{c \geq \gamma^p} + \mu^p + \epsilon_{apc} \tag{2}$$

where $\tilde{\beta}^p = \beta_a^p - \beta_c^p$. And the location, γ^p, and size δ^p, of the trend break by cohort are identified.

I estimate the model separately for each year, by weighted least squares, following the methodology in Hansen (2000) [7] Algorithmically, this amounts to looping through different assumed values of the trend break location γ^p, and selecting the location with the lowest sum of squared, weighted residuals.

Following Hansen (2000) I invert the following likelihood ratio statistic to form 99 percent confidence intervals for γ^p:

$$LR(\gamma^p) = n \frac{S(\gamma^p) - S(\hat{\gamma^p})}{S(\hat{\gamma^p})} \tag{3}$$

where n denotes the number of observations, $S(\gamma^p)$ is the weighted sum of squared residuals from estimating equation 3 with the trend break location fixed at a given γ^p, and $S(\hat{\gamma^p})$ is the sum

[7] As weights I use a consistent estimate of the inverse of the variance of observed log mortality in each cell: $\frac{population \cdot mortality}{(1-mortality)}$ (Schulhofer-Wohl and Yang, 2016).

of squared residuals with the *estimated* break location $\hat{\gamma^p}$. I constuct the 99 percent confidence interval as those values of γ^p such that $LR(\gamma^p) <= 10.35$, the critical value given in Hansen (2000). While I allow for heteroskedasticity in inference on other parameters, this test requires homoskedasticity.

Hansen (2000) also suggests that inference on δ^p is unaffected by treating γ^p as unknown. I therefore form confidence intervals for δ^p using the standard formula for weighted least squares.

Following standard practice, I employ an ad-hoc restriction to not allow the location of the cohort-break γ^p to be estimated to be one of the youngest of oldest cohorts in the sample: in each year I restrict the location of the break to not be one of the 3 youngest or oldest cohorts.

3.2 Gompertz law and age restrictions

As described in the introduction, and expanded in more detail below, my main analysis relies importantly on the log linearity of mortality by age, known as Gompertz law or Gompertz-Makeham law (Gompertz, 1825)[8]. Importantly, this log linear relationship does not generally hold at all ages. Initally, it appears Gompertz understood his "law" to apply to mortality rates for approximately ages 20 to 60 (Olshansky and Carnes, 1997).

In all of the main analysis I focus on log mortality rates between the ages of 30 and 75. I begin analysis at age 30 because the log linear relationship between age and mortality only begins near this age. Log mortality rates at ages under 30 are highly nonlinear in age in many years. Appendix Figure 1 shows evidence of this for select sample years before my study period, 1965 and 1975. Appendix Table 1 shows that the root mean squared error of simple Gompertz models of log mortality, with just a linear age term and a constant, is very low for ages 30-75. When these models are extrapolated to younger ages the fit substantially worsens and the root mean squared increases by around an order of magnitude. I impose the upper age restriction of age 75 for convenience, as the 1946 and 1949 cohorts for which I find the health decline to have begun are only 69 and 66 in the final year of my sample (the Gompertz curve is known to fit less well after age 90, so some upper age restriction would be required regardless).

3.3 Graphical examples for select years

In this section, I present graphical evidence of a break in the cross-cohort mortality trend for a set of select years between 1985 and 2015.

[8] See Chetty et al. (2016); Finkelstein et al. (Forthcoming) for recent applications in economics.

I provide graphical examples of this trend-break estimation method for select years, based on fitting a piecewise linear model to log mortality rates of white men and women. I show plots including the true log mortality rate, and the piecewise linear model and the location of the trend break — estimated based on the procedure described above. Additionally, I show an extrapolation of the linear trend estimated for pre-break cohorts to younger cohorts, and plot the deviation of the true log mortality rates from the estimated pre-break linear trend. I restrict the sample to log mortality rates for 30-75 year old white men and women, born between 1930 and 1965.

Figure 1 implements this graphical example of the trend-break estimation method for the log mortality of white men in 1985, 1995, 2005, and 2015. The red circles show the true observed log mortality rate for each single year of age. The solid blue line shows the estimated trend-break model based on equation 2. The vertical, labeled gray line shows the cohort at which the trend break is estimated to occur for that year. The dotted blue line extrapolates the estimated linear trend for cohorts born before the trend break to younger, post-break cohorts.

Across the four year shown, the trend break is estimated to occur at different ages but at nearly the same cohort — suggesting it is the result of health differences across cohorts. The breaks by age are staggered with approximately 10 year gaps — occurring at ages 40, 48, 60, 68 for the years 1985, 1995, 2005, and 2015 respectively. As a result, the estimated breaks by cohort are nearly identical — occurring at the 1944, 1946, 1944, and the 1946 cohort across the years shown.

Figure 2 plots the deviations of the true log mortality rates of white men from the estimated linear trend for pre-break cohorts. That is, it shows the difference between the pre-break trend — shown with a solid blue line for pre-break cohorts and a dotted blue line for post-break cohorts in Figure 1 — and the true log mortality rates — shown in red circles. A horizontal gray line is now plotted at the 1946 cohort.

In each year shown, the deviations are near zero for cohorts born before 1946, implying that the a linear trend fits the log mortality rates well for these cohorts. Then suddenly at or near the 1946 cohort there is a trend break and the deviations increase for each subsequent cohort. The trend break is particularly large in 1985 and 1995, with the 1955 cohort for example experiencing log mortality rates approximately 60 and 40 log points, in the respective years, above what the Gompertz curve for earlier cohorts would predict. The trend break is smaller but still clearly evident in 2005 and 2015 — with the 1955 cohort experiencing log mortality rates around 20 and 15 log points above the prior trend in these two years.

Figure 3 replicates for white women the trend break examples shown in Figure 1. The trend break in log mortality is less visible striking for women than for men — but still evident and detected by the Hansen trend break estimation to occur at a similar cohort across years. The breaks

by age are again staggered with approximately 10 year gaps — occurring at ages 37, 44, 56, 65 for the years 1985, 1995, 2005, and 2015 respectively. As a result, the estimated breaks by cohort are again close to identical — though later than for men — occurring at the 1947, 1950, 1948, and the 1949 cohort across the years shown.

Figure 4 plots the deviations of the true log mortality rates of white women from the estimated linear trend for pre-break cohorts. That is, it shows the difference between the pre-break trend — shown with a solid blue line for pre-break cohorts and a dotted blue line for post-break cohorts in Figure 1 — and the true log mortality rates — shown in red circles. A horizontal gray line is now plotted at the 1946 cohort.

In each year shown, the deviations are near zero for cohorts born before 1949, implying that the a linear trend fits the log mortality rates well for these cohorts. Then suddenly at or near the 1949 cohort there is a trend break and the deviations increase for each subsequent cohort. Notably, the trend breaks occur approximately 3 birth years later than those for white men. They are also are more similar in size across years than those of men. Each of the years shown exhibit a trend break such that the 1955 cohort experiences nearly .15 log points higher mortality than they would have had the pre-break trend continued. As a result, the breaks in 1985 and 1995 are smaller for women than men, and the breaks in 2005 and 2015 are similar for the two genders.

3.4 Estimates for all years

I now implement the above described approach to estimate trend breaks in log mortality rates for all the years between 1985 and 2019. I estimate equation 2 for white women and men separately for each year between 1985 and 2019 — restricting the sample to ages 30-75, and cohorts 1930 to 1965.

Panels A and B of Figure 5 show the results across years for the log mortality rate of white women. The left panel shows for each year of data the estimated location of the cohort specific trend break, $\hat{\gamma^p}$, as well as the 99 percent confidence intervals. For all years from 1985 to 2019, the estimated trend break is between 1948 and 1952, and the confidence interval includes 1949. The right panel shows for each year the size of the estimated trend break, $\hat{\delta^p}$. The size of the trend break in log mortality is initially near .04 and then declines to nearer .02 after 1990, but has begun increasing again slightly since 2012.

I also use the asymptotically valid bootstrap procedure suggested in Hansen (1999) to test the null hypothesis of no break, eg. $H_0 : \delta^p = 0$. For all years, the value of the F-type statistic for the true data is larger than that calculated in all of the 1000 bootstrap repetitions that I run — implying a P-value of less than .001 for the null of no break.

Panels C and D of Figure 5 show analogous results for the log mortality rate of white men. The location of the trend break is slightly less stable for men than for women. From 1985 to 2004, the estimated trend break is precisely estimated and consistently located between 1945 and 1946, and the confidence interval includes 1946. After 2004, the location becomes less precisely estimated. Between 2004 and 2009 the point estimate drops to near 1940 and the confidence interval no longer includes 1946. For 2009 to 2014, the point estimate jumps to 1945 and the confidence interval again includes 1946. From 2015 to 2019 test point estimates are again all located at the 1946 cohort.

The size of the trend break for log mortality is initially larger for men than for women, it is above .06 for the first 7 years examined. In later years it falls to near .02, similar to that estimated for women. The size of the break for men is also growing in the last 4 years of data.

For men, in all years the implied P-value from the bootstrap procedure is less than .003 for the null of no break.

Overall, the results in this section demonstrate that the patterns shown for select years in Figures 1 through 4 are not anomalous. For women, in each year between 1985 and 2019 there exist precisely estimated slope changes in the age-profile of log mortality, which occur almost exactly at the 1949 cohort. For men, in in all years before 2004, the location of the slope change is precisely estimated and very consistently at or near the 1946 cohort, but precision falls somewhat between 2004 and 2015 and the breaks move earlier; however since 2015 the breaks have become more precisely estimated again and are again consistently located at 1946. Further, in all years statistical tests provide strong evidence in favor of rejecting the null hypothesis that no change in slope exists. As outlined in the introduction, these slope changes are consistent with a cross cohort decline in health, beginning for white men born after 1946 and white women born after 1949.

The initial draft version of this paper only included data through 2015 and was first submitted to a conference in September 2018 when the 2017 to 2019 mortality data had not even been released. Therefore the fact that the slope changes in log mortality are still located at the 1946 and 1949 cohorts, for men and women respectively, in the four most recent years provides an important out of sample validation of the cohort-based theory.

4 A single structural break model

The above analysis allowed the location of the trend break in log mortality to vary by year. The results appear to suggest that the trend break occurs in the same cohort across years. Therefore, in this section I estimate a single structural break model across years, which imposes that the cohort-

specific break occurs at the same cohort in all years. Guided by the above results, I allow the size of the break to vary across years. Estimation of this single model allows me to probe the robustness of the trend break results by including different specifications of a control function — which allows a separate polynomial in age for each year.

I again use the approach of Hansen (2000) to estimate the following model:

$$ln(mort_{apc}) = \delta^{p}_{1,c} \cdot c + \delta^{p}_{2,c} \cdot 1_{c \geq \gamma} \cdot (c - \gamma) + f(a, p) + \epsilon_{apc} \tag{4}$$

where $ln(mort_{apc})$ denotes the log mortality rate of the cell of age a, period p, and cohort c — for either white men or women. $\delta^{p}_{2,c}$ estimates the size of the break in each year p, and γ estimates the cohort at which a break occurs. I include increasingly flexible specifications of the "control function" $f(a, p)$. In most specifications the cohort trend $\delta_{1,c}$ is not separately identified from aspects of the control function, but main objects of interest, the size and location of the trend break are identified.

As above all models are estimated by weighted least squares, following the approach outlined in Hansen (2000). The sample includes the years 1985-2019, ages 30-75, and cohorts born from 1930-1970.

Table 1 reports the results of estimating equation 4, with the log mortality rate of white women or men for single age-by-year bins as the dependent variable. Each column contains the results from a separate regression, with progressively more flexible specifications of the control function $f(a, p)$ from left to right.

Column 1 includes a a full-set of year FEs, as well as a linear age term *interacted with year*, eg. it allows for a different Gompertz curve in each year. The remaining two columns gauge robustness, by adding progressively higher-order polynomials in age *interacted with year*. At the most extreme, column 3 allows a separate cubic in age for each year, so allows the impact of age on mortality to vary by year, albeit in a smooth parametric way.

The results for white women are shown in Panel A. For women the location of the trend break is consistently estimated to occur between the 1948 and 1950 cohort across all specifications. Additionally, the 99 percent confidence intervals, calculating by inverting the likelihood ratio statistic of Hansen (2000) are very tight, each including at most two cohorts.

For women the average size of the estimated cohort break — the average value of $\delta_{2,c}$ across all years—changes slightly depending on the specification of the control function. For the main specification in column 1, when I allow for separate linear-age-by-year controls, the break size is estimated to be .027, with a standard error of .002. Adding the higher order polynomials-in-age interacted with year yields estimates ranging from .025 to .035. Even, the smallest estimate of .025

implies that the white women born in 1960 have had on average a 28 percent higher mortality rate then if their mortality experience matched the trend for pre-1950 cohorts.

For each model I follow the bootstrap procedure described in Hansen (2000) to test the null hypothesis that no trend break occurs, ie. that $\delta^p_{2,c}$ is equal to 0 for all p. For all models, the value of the F-type statistic for the true data is larger than all of the 1000 bootstrap repetitions, suggesting a P-value of less than .001 for the null of no break.

Panel B reports analogous results with the log mortality rate of white men as the dependent variable. For white men, the estimated location of the trend break is even more consistent and precisely estimated. It is estimated to occur at the 1946 cohort across all specifications, and the 99 percent confidence intervals do not overlap any other cohorts.

As for women, the average size of the estimated cohort break for white men varies depending on the specification of the control function. The range of estimates are very similar to that for women: ranging from .034, with a standard error of .001, in the main specification with linear-age-by-year controls, to .026 when I allow for cubic-age-by-year controls.

Again even the smallest estimate of .026, suggests a non-trivial impact of the trend break on mortality rates. It implies that the white men born in 1956 have had on average a 29 percent higher mortality rate then if their mortality experience matched the trend for pre-1946 cohorts.

For men as well, in all models the value of the F-type statistic for the true data is larger than all of the 1000 bootstrap repetitions — implying a P-value of less than .001 for the null of no break.[9]

5 The cohort-specific trend break in log-mortality and year-over-year trends in mortality at different ages

I next use the previously estimated model to assess whether the cohort-specific pattern can explain the timing of recent increases, and stagnating improvements, in mortality by age. As described above, the timing by year of mortality trend breaks has not been uniform across age: while the age-adjusted mortality rate of non-Hispanics white women aged 45-54 began to increase in 1999 (Case and Deaton, 2015; Gelman and Auerbach, 2016), that of those aged 35-44 began to increase earlier in 1991, while that of 55-64 year olds continued to decline until a sudden break in 2010. Below, I use the shared cohort-specific trend break model estimated in the previous sec-

[9]For robustness, I also estimated models which include a full set of age fixed-effects and a full-set of year fixed-effects. This model is akin to traditional additively-separable age-period-cohort models, but specifies the cohort effects as piecewise linear. It therefore does not allow any age-by-year interactions. Interestingly, these models also yield precisely estimated break locations, at 1946 and 1949 for men and women respectively, and larger estimated break sizes, of .45 and .43 for men and women respectively.

tion to assess whether this cohort pattern can explain the staggered timing of mortality increases by age-bin. Intuitively, the question is whether "unhealthy" post-1949 cohorts have driven mortality increases first at young ages and subsequently at older ages, as they move through the age distribution.

To do so, I use the estimation results from the shared trend break model based on equation 4 and described in the previous section. I use the specification including a full set of year fixed-effects and a separate linear age effect for each year, reported in column 1 of Table 1. I then use the estimated model to simulate mortality rates by year and age group. Specifically, for each age-year cell I predict log mortality, and then calculate predicted mortality as the natural exponential of predicted log mortality, for each single age-by-year pair. Finally, I calculate the simulated age-adjusted mortality for age-bins as the simple average across single ages.

Figure 6 shows the true age-adjusted mortality rates and those simulated from the above model, for white women and men by year, for 35-44, 45-54, 55-64, year olds respectively. The simulated series is not shown when the age-bin includes cohorts born before 1930 or after 1970, as the model cannot produce counterfactual predictions for them. For women's mortality, the simulated series closely tracks the true mortality rates for all age bins. Notably, the simulated mortality rates match realized trend breaks in the mortality rate of 45-54 year olds in 1998 and of 55-64 year olds in 2010. The simulated series also nearly matches the increase in mortality of 35-44 year olds, predicting an increase starting after 1992 rather than the observed break in 1991.

The mortality experience of white men aged 35-44 shows the clear imprint of the AIDS epidemic — increasing between the early 1980s and 1995 and then declining sharply. Never the less, the simulated series from the simple cohort-specific trend break model again closely tracks the true mortality rates for all age bins. Again for men, the simulated mortality rates matches the observed trend break in the mortality rate of 45-54 year olds in 1998. It also matches the timing of the break for 55-64 year olds in 2010 — though the size of the true increase is larger than the simulated one.

There is a relatively tight mapping between the mortality rates predicted from the cohort-specific trend break model — without any other non-linear age-by-year interactions — and the true year-over-year patterns in mortality by age. This suggests that cohort specific differences in health plausibly played a key role in recent increases in mortality by age. As described in the introduction however, it seems likely that the cohort decline in health interacted with year-specific factors, such as the AIDS epidemic and the recent expansion of opioid supply, to ultimately produce the observed mortality patterns.

6 Comparison to other ethnic groups and geographic heterogeniety

I next show some suggestive evidence that a similar — though smaller in magnitude — cohort-specific trend break in log morality rates to that for white Americans is evident for black women, and women and men of other races, but not for black men. I further show that the trend break documented above is concentrated among non-Hispanic whites —in years when its possible to distinguish — and a similar break is not evident for Hispanics.

To do so, I estimate the shared cohort-specific trend-break model of Section 4 for other racial and ethnic groups. As in the previous section examining geographic heterogeneity, I estimate the trend break model based on equation 2 with a full set of year fixed-effects and a separate linear age effect for each year (as shown for all whites in column 2 of 1). I again follow Hansen (2000) and the procedure described above.

I first estimate the same model for blacks and Americans of all other racial groups combined. The sample again includes the years 1985-2019 and ages 30-75. I restrict the cohorts included in the sample to 1930 to 1960. I do so to avoid inclusion of cohorts born near the passage of the Civil Rights Act — for which previous research has documented large improvements health of blacks linked to increased hospital access following desegregation (Almond et al., 2006; Chay et al., 2009, 2014).

Results for women are shown in Panel A of Table 2. The cohort at which the trend break is estimated to occur is very similar across the 3 racial groups: 1949 for whites, 1946 for blacks, and 1948 for other races. The average size of the estimated trend breaks is positive for women of all racial groups, but the magnitude differs: from .0306 for white women, to .0199 for women of other races, and .0115 for black women. Results for men are shown in Panel B — and those for black men differ substantially from the pattern for other groups. The cohort at which the trend break is again very similar for whites and the other racial group category: 1946 for white men, and 1944 for men of other races (with a confidence interval which includes 1946). And again the average size of the estimated trend breaks are positive for both these groups: .0388 for white men and .0262 for men of other racial groups. However, for black men the location of the estimated trend break is the 1952 cohort and the average size is estimated to be slightly *negative* at -.0066. A thorough analysis of trends in mortality and health for other racial groups — and an understanding of why they appear to differ for black men — is worthy of further study, but outside the scope of this paper. [10]

[10]Two additional facts suggest that the differing pattern for black men may be linked in some way to the HIV

I then estimate the above model for the years after 1997 — when Hispanic-origin is reported on death certificates in all states — separately for i) all whites, regardless of Hispanic-origin; ii) non-Hispanic whites; and iii) Hispanic whites. Results shown in Appendix Table 2 show that restricting estimation to non-Hispanic whites alone does not change the estimated location of the trend break but increases slightly it's magnitude. In contrast, the estimated location of the trend break for Hispanic white women and men are 1939 and 1958 respectively. That for Hispanic women is positive and slightly smaller than that for non-Hispanic whites, and that for men is negative and small in magnitude. The apparently different cohort-specific pattern of health for Hispanics is outside the scope of this study — but could be potentially due to changing immigration patterns.

Appendix A shows that the cohort-specific trend break in log mortality rates of whites documented above is remarkably widespread across the United States, suggesting that the associated health decline in similarly widespread. For example, the precise cohort at which the trend break is estimated to have occurred and the estimated magnitude of the trend break vary only slightly across the four Census regions. Further, all 50 states have estimated trend breaks which are positive in magnitude and greater than .01 for women and men. Estimates for women in all states are between .005 and .045, and 30 out of 50 states have estimated break sizes between .015 and .025. For men estimates range from .01 to .055, and 32 out of 50 states have break sizes between .025 and .035 [11]

7 Early-life mortality

As described above, my main empirical strategy is only applicable to mortality above the age of 30, when mortality is generally log linear in age. Below I provide a descriptive analysis of mortality patterns of White Americans by cohort at ages below 30 — which shows evidence of trend breaks in infant and childhood mortality by birth cohort near the late 1940s such that rapid improvements between the 1930s and the late-1940s cohorts suddenly slowed — and at some ages stopped completely. More detail on the data and estimation, as well as additional results are given in Appendix B.

While the main analysis in earlier sections identifies cohort breaks as changes in the slope of log mortality within a given year, and therefore "controls" quite flexibly for external factors which vary by year, as well as factors which vary by age and year which are smooth in age; the analysis

epidemic. First, the mortality rate of HIV for black men — which was much higher than that for other racial groups — peaked for the 1952 cohort and declined for subsequent cohorts. Second, the cohort-specific decline in log mortality rates after the 1952 cohort, relative to the preceding trend, appeared suddenly in the late 90s. Results available from author by request.

[11] Given smaller population sizes at the state-level, I impose the location of the cohort-specific trend break in each state to match that at the national level and estimate the magnitude of the break.

in this section identifies cohort breaks by pooling across years but for a fixed age. These results are therefore more descriptive in nature.

Figure 7 plots estimates of the cumulative mortality rate from birth to age 30 by cohort. Panels A and B show that both white male and white female early life mortality declined rapidly from the 1933 to the mid-1940s cohorts. A trend break is estimated to have occurred at the 1948 cohort for women and the 1945 cohort for men, such that for cohorts born after these years cross cohort improvements in log mortality were much slower than they would have been had the prior trend continued. The deviations of log mortality at these ages from the pre-break trend are plotted in Panels C and D, for women and men respectively. Though the methodology differs from that in previous sections the pattern is very similar. The magnitude of the trend break and resulting deviations are large: resulting in a log mortality rate which is nearly 60 log points higher for women and more than 40 log points higher for men, than it would have been had the trend for earlier born cohorts continued.

The fact that improvements in early-life mortality slowed after the late-1940s cohorts points against a simple mortality selection mechanism driving the health decline of these cohorts as adults. In the context of a simple single-index selection framework (Bozzoli et al., 2009; Chay et al., 2009), if the mortality improvements across the twentieth century were due to improvements in the "threshold of survival", then the trend break in infant and childhood mortality would imply an effect of selection which would work in exactly the opposite direction of the adult health decline I've found above. The selection effect would reduce the average health of surviving adults across cohorts born between 1930 and around 1950, with this effect suddenly slowing for cohorts born from 1950 to the mid-1960s. Alternatively, the patterns could be consistent with a more complex model in which the threshold of survival is improving smoothly over time and the sharp break in the infant and childhood mortality trend is caused by a decline in the entire distribution of latent

childhood health. [12]

8 Preliminary investigation into the cause of the cohort decline

I have presented evidence of a large decline in health across cohorts of White Americans, relative to the trend for prior born cohorts, which began for white men born after 1946 and white women born after 1949 and has contributed substantially to mortality trends over the last few decades. This section provides a preliminary investigation into the cause of this cross cohort health decline. The evidence I present reveals no clear smoking gun and suggests that a number of ex ante plausible explanations are unlikely.

The large cohort health differences appear likely to be driven by large changes in the experiences of these cohorts during critical periods in their lives— such as at labor market entry, during adolescence, early life, or in utero. They also could be driven by some form of selection across cohorts.

8.1 Educational attainment

Previous authors have noted a sudden decline in the educational attainment for cohorts of Americans (of all races combined) born after the late 1940s. Heckman and LaFontaine (2010) estimate that the U.S. high school graduation rate peaked at around 80 percent in the late 1960s — roughly when the 1946 cohort was 18 — and has declined by 4-5 percentage points since then. Card and Lemieux (2001b) highlight a sudden 12 percent fall in college entrance rates for men from 1968 to 1978 — approximately the 1947 cohort to the 1957 cohort — and a stagnation in prior improvements for women. These authors are largely unable to find an answer to why the previous trend of improvement in educational attainment suddenly stagnated and even reversed. Card

[12] Appendix Figure 5 also shows that improvements in the infant mortality rate for whites in the United States did not occur in Canada or a set of European countries, and that the difference in infant mortality rate between the United States and these countries suddenly began to widen after 1946. If one assumes that medical technology is similar across countries and therefore that the "threshold of survival" is the same across countries, then one could interpret the deviations of the United States as representing shifts in the underlying distribution of health — suggesting the health of white infants in the United States who survive began to decline after 1946.

Of course if one moves beyond the single-index selection than the infant and childhood mortality patterns are not informative about the time-varying selection effects at all. For example, if medical technology changed after 1946 such the the correlation between infant and childhood mortality and underlying health increased, this could work to the reduce the average health of survivors. Additionally, it is also possible in principle that there were coincident trend breaks at 1946, leading to both a) an improvement in the threshold of survival and b) a worsening of the underlying health distribution, which both would then contribute to the worsened adult health of post-1946 cohorts. However, these explanations appear quite complex and there is not an obvious historical change in medical technology which would have these effects.

and Lemieux (2001b) present an extensive study of possible causes of the decline and conclude that for women it could be explained by low returns to education and cohort size, but that for men the decline represents a fundamental trend break with no observable explanation. Acemoglu et al. (2012) also note this stagnation in educational attainment, and suggest that the sharpness of the change in trend by cohorts suggests it is unlikely to be caused by a sudden change in the school system, and that "other factors are thus likely to be at play."[13]

Panel A of Table 3 summarizes these patterns using data from the Current Population Survey Merged Outgoing Roation Groups. I fit models which allow for two possible trend breaks by cohort to the average years of schooling by birth cohort.[14] The estimates suggest a precisely estimated, large trend break in years of schooling located at the 1947 cohort for white men and the 1949 cohort for white women. The models actually imply absolute declines in years of schooling between 1947/1949 and the early to mid-60s when a second trend break is estimated and a rebound began. Appendix Table 4 and Appendix Figure 6 show this is driven by trend breaks along the educational ladder: the share completing high school, a 4-year college degree, and an advanced degree all exhibit trend breaks near those for years of schooling such that prior improvement in educational attainment stops and *reverses*, before rebounding beginning with early-to-mid-1960s cohorts.[15]

However, the size of the cohort slope change in mortality appears likely too large to be explained by the decline in education alone. Quasiexperimental estimates of the causal effect of schooling on mortality, based on changes in compulsory schooling laws generally suggest that a year of schooling reduces mortality by 0 to 6 percent.[16] Applying even the 6 percent estimate to the trend breaks in table 3 would imply that the trend break in schooling would directly cause a trend break in log mortality of .0066 for men and .0052 for women — approximately a quarter and 19 percent respectively of my preferred estimate of trend break in log mortality above. In a notable outlier, Buckles et al. (2016) use a different research design based on Vietnam draft avoidance in

[13]Evocatively, they also write: "we do not believe that social science has so far produced an adequate account of what went wrong with the U.S. human capital machine."

[14]I pool data from 1990 to 2018, white individuals age 25 to 75, cohort is defined as age - year -1. I calculate approximate average years of schooling for each cohort based on the 16 schooling categories in the CPS. I then estimate the models allowing for two trend breaks in cohort using the sequential estimation approach suggested in Hansen (2000) for such models.

[15]The size of the trend break in high school completion may actually be an underestimate due to the sampling frame of the CPS and GED-recipients being counted as completing high school, see Heckman and LaFontaine (2010).

[16]See Galama et al. (2018); Mazumder (2012) for reviews. For example, Clark and Royer (2013) estimate a precise zero effect of schooling on adult mortality in a particularly credible study based on UK schooling reforms. Gathmann et al. (2015) pool data from 19 European countries and estimate that a year of schooling reduces the mortality rate of men by 2.8 percent, but find no statistically significant effects for women; the largest individual country estimate for men from that paper is only 5.6 percent The credibility of the approximately 6 percentage point estimate of Lleras-Muney (2005) for the US is debated by Mazumder (2010, 2008) and Black et al. (2015).

the US and find IV estimates of the impact of a year of college schooling on men's mortality that are nearly identical to the cross-sectional association. Generously treating estimates of the cross-sectional mortality gap between those with 12 years and 16+ years of schooling from Jemal et al. (2008) as the causal effect of 4 years of schooling would imply that the trend break in schooling would directly cause a trend break in log mortality of .028 for men and .017 for women — 82 and 63 respectively of my preferred estimate of trend break in log mortality above. [17]

The above results could potentially suggest that there was a broader decline in health and human capital for these cohorts originating at age 17 or earlier. However, other proxies of childhood health generally either stagnated or continued to improve, but did not decline in absolute terms. As described above, improvements in infant and childhood mortality suddenly slowed around 1950 but did not stop. Increases in adult height, often viewed as a proxy of childhood nutritional status (Floud et al., 2011; Tanner, 1990), also suddenly stopped for white Americans born between approximately 1955 and 1974 while the height of Europeans continued to grow rapidly (Komlos and Lauderdale, 2007a,b; Komlos, 2010). Though again, height does not appear to have declined in absolute terms. Although the educational attainment declines were echoed in standardized test scores such as the SAT (Koretz, 1987; Bishop, 1989) — measures of famously continued increasing across these cohorts (Flynn, 1984). There was also no increase in the rate of low birthweight births between 1951, when it was first recorded in Vital Statistics reports, and 1970 (Chase and Byrnes 1970).[18]

8.2 Birth order

Changes in the average birth order, or "parity", across cohorts associated with the baby boom could be a plausible contributor to the cohort health decline. A large portion of the baby boom was driven by changes in the age at first birth across cohorts of mothers, rather than changes in family size (Ryder et al., 1980; Van Bavel and Reher, 2013). The first births of many cohorts of women were "stacked up" near 1946 and 1947. Therefore the 1947 cohort consists of a particularly large share of people who are first-born in their family. There is then a sharp trend break at the 1947 cohort in birth order, such that the share of people in each cohort who are later born children in their families increases between the 1947 cohort and mid-1960s cohorts. A large and growing body

[17] It is hard to directly compare Buckles et al. (2016) causal effect estimate to my result as their dependent variable is cumulative mortality over a 26 year period. As their IV estimates are indistinguishable from OLS, I treat their causal effect estimate as near the cross-sectional association listed above. It is also hard to know more generally how to treat their estimate, as it is such an outlier in the literature and their design differs considerably in using quadratic functions of state and national draft risk as instruments for both veteran status and educational attainment.

[18] Though see Almond et al. (2005, 2002) for evidence that birth weight is not a "sufficient index" of infant health.

of literature finds that later born children have worse outcomes as adults, even when outcomes are examined within-family, by controlling for family fixed-effects (Black et al., 2005, 2011; Barclay and Kolk, 2015; Breining et al., 2020).[19]

To summarize the potential effects of birth order on log mortality, I simulate the effect of observed birth order changes across cohorts. I use within-family estimates of the effect of birth order on mortality from Barclay and Kolk (2015) and observed birth order shares for white Americans from Vital Statistics to simulate the impact of birth order changes on log mortality rates by cohort.[20] The simulations aim to answer the question: how would mortality rates by cohort have differed if cohorts only differences was their birth order shares?

As I did for education, I then fit trend break models allowing for two trend breaks of unknown location, to the simulated mortality effects. The results, shown in Panel B of Table 3, suggest that the birth order changes could generate trend breaks in log mortality at the 1947 cohort of .0073 for men and .0107 for women — 21 and 40 percent respectively of my preferred estimates of the actual log mortality trend breaks. However, I also estimate a second trend break located at the 1964 cohort which is of opposite sign and larger in magnitude than the first break. This suggests that the birth order effects alone would have lead to a sharp rebound in health after the 1964 cohort which does not appear to have occurred. [21] Appendix Table 5 and Appendix Figure 7 show additional birth order results.

8.3 Fetal lead exposure from motor vehicle gasoline

A number of facts point towards fetal lead exposure from motor vehicle gasoline as a possible important driver of the cohort health decline.

Automobile production and fuel use were restricted during World War II and began to rapidly increase soon after. Panel C of Table 3 show estimates of trend break models allowing for two

[19]The mechanism for the birth order effect is not fully understood. There is evidence that later born children are actually healthier at birth than earlier born children, but by adolescence this pattern has already reversed with later born children having more hospitalizations and higher mortality (Björkegren and Svaleryd, 2017). A leading candidate mechanism is differences in parental 'investments' by parity, see eg. Price (2008) for direct evidence that parents spend less 'quality time' with their later born children. Daysal et al. (2021) suggest the transmission of respiratory diseases from older to younger siblings plays a role.

[20]Birth order shares for 1931-1939 are digitized from Vital Statistics reports, and for 1940-1970 are calculated from U.S. Cohort and Period Fertility Tables, 1917-1980 compiled by Robert D. Hauser and available from the Office of Population Research at Princeton. The simulation begins with observed mortality rates at age 40 of the 1949 and 1946 cohorts of white women and men respectively. I then use observed birth order shares and odds ratio estimates of the impact of birth order on mortality from Barclay and Kolk (2015) to calculate simulated mortality rates at age 40 for all other birth cohorts from 1931 to 1970. Trend break results for ages other than 40 are nearly identical.

[21]Handy and Shester (2019) conduct a similar exercise for educational attainment and find that birth order changes can explain more than one third of the decline in college completion for white men born between 1946 and 1960.

trend breaks of unknown location fit to annual time series of i) motor vehicle registrations and ii) fuel usage by motor vehicles, for 1930 to 1970 from Historical Statistics of the United States US Census Bureau (1975). Both series are estimated to have large trend breaks, in 1945 and 1944 respectively (with registrations increasing even more rapidly after another trend break in 1963). The time series are also plotted in Appendix Figure 8.

Exposure to pollution from lead added to gasoline also likely increased rapidly over this period. Lead concentrations of automotive gasoline decreased during WWII because the lead additive was needed for aviation gasoline for military planes (Oudijk, 2010). The lead content of gasoline generally increased after this period, and with the rapid increase in total fuel consumption the total tons of lead added to gasoline consumed in the US increased rapidly [22]. When lead additives were phased out of gasoline beginning in the 1970s blood lead levels of children fell rapidly (though lead would remain stored in bones and may be remobilized during pregnancy and lactation with potential toxic effects on pregnant women and fetuses (Silbergeld, 1991; Gomaa et al., 2002)).

Fetal and early life lead exposure has been linked to poor health and cognitive development. Early life lead exposure is thought to have broad and lasting negative health effects, impacting for example the development of multiple organ systems, cognitive ability and emotional regulation, and cardiovascular disease.[23]

High blood lead content was ubiquitous across the US when the cohorts whose health declined were children or in utero. McFarland et al. (2022) estimate that the share of children with blood lead levels above the 2015 threshold for "clinical concern" increased from 50 percent for the 1940-45 cohorts to 100 percent for the 1966-75 cohorts. The first nationally representative estimates of blood lead in 1976-1980 show average levels for 1-5 year olds more than 3 times this threshold for all of the following demographic groups: whites and blacks, children from high and low income families, residents of large and small MSAs, and in all 4 Census Regions (Egan et al., 2021).

The facts above could point towards fetal lead exposure in particular as a possible driver of the health decline. The timing of the trend break in automobiles — of 1945 — would lead to a trend break in fetal exposure to automobile-based lead pollution at approximately the 1946 cohort. In contrast, childhood exposure would have increased for earlier cohorts — for example if age 5 was

[22] See Shelton et al. (1982) for estimates of the lead content of regular and premium gasoline. These fluctuated considerably but generally increased over the period. The often cited series on total lead consumed in gasoline lead additives, United States Bureau of Mines (1941-1970), (see eg. Reyes (2007); Curci and Masera (2018)) actually measures the volume of lead consumed in domestic *production* of these additives — so will include production for export and does not include imports. A series shown in Mielke et al. (2010) based on Senate testimony by the leading manufacturer of lead additives also shows rapid increases from around 25,000 to more than 200,000 metric tons between 1945 and 1970.

[23] See for example McMichael et al. (1986); Needleman (2004); McFarland et al. (2022); Hollingsworth et al. (2022); Aizer et al. (2018) on the health and cognitive effects of fetal and childhood lead exposure.

a critical period for lead exposure we would see health declines beginning with around the 1940 cohort, who would have been around 5-years old in 1945. If fetal lead exposure is a main driver it appears that the effects have manifested slowly as these cohorts have aged, potentially showing up first in educational declines to lead exposures effect on cognitive and noncognitive skills. Of course much more research is needed to establish or falsify the importance of fetal lead exposure for the documented health decline.

8.4 Some evidence against other possible causes

8.4.1 Cohort crowding

The baby boom is often defined as beginning with the 1946 cohort — when there were sharp increases in the number of births — making the casual observation that it "caused" the health and human capital decline initially appealing. However, the cross cohort pattern in cohort size shown, in Appendix Figure 9, is quite different than the piecewise linear pattern in mortality. Also see Appendix Table 6 which fits trend break models, allowing for two breaks, to cohort size. Therefore, a simple "cohort-crowding" theory (Easterlin, 1987; Bound and Turner, 2007) cannot generate the cohort health decline. For cohort size to drive the patterns in cohort health a theory would have to posit a complex lagged effect of cohort size on health and human capital.

8.4.2 Maternal smoking

The existing evidence on trends in smoking by women of childbearing age also make maternal smoking appear an unlikely driver of the cohort health decline.[24] There does not appear to be survey evidence on *maternal* smoking specifically for this period, but the pattern of smoking rates by women of childbearing age do not show any evidence of a sudden increase after the late-1940s. Appendix Table 7 shows estimates of trend break models fit to smoking rates by women of childbearing age from Holford et al. (2014) which suggest a very different pattern. Also see Appendix Figure 10.

[24]Ex ante, if maternal smoking increased after 1946 it could be a plausible driver, as it has been associated with low birth weight (Kramer, 1987; Almond et al., 2005) which itself has been linked to adult health and other outcomes (Black et al., 2007), and there is some direct evidence of childhood cigarette smoking exposure and health (Simon, 2016).

8.4.3 Observable family background and childhood circumstances

Observable differences in the characteristics of the parents of these cohorts or where they grew up do not appear likely to be driving their decline in health. Appendix Figures 11, 12, and 13 and Appendix Table 8 show patterns and results of estimating trend break models by birth cohort for a range of such characteristics from the General Social Survey. With two possible exceptions they do not show evidence of trend breaks near the late-1940s in directions likely to negatively impact health. First, the share of individuals who lived with both their mother and father at 16 appears to decline rapidly after the mid-1950s cohorts. This share is estimated to have a trend break at 1954, which is evident visually in Appendix Figure 12. The 99 percent confidence interval for the trend break location just includes 1949. This could therefore be an important causal factor if that is the true trend break location and sampling variation lead to the later estimated break location. Second, there is an imprecisely estimated trend break near the 1942 cohort (and including the late-1940s cohorts) in the share of individuals with both parents born in the US, such that the share with both native-born parents decreases subsequently. I collected additional data from Vital Statistics volumes — based on the universe of births — which record the share of births in each year to mother's born outside the US. Fitting this data to trend break models, yields a precisely estimated trend break at 1944 (with that also the latest cohort in the 99 percent confidence interval) and a second break in 1956. This data therefore suggest parents nativity is not likely to be driving the health decline. [25]

8.4.4 Conditions at labor market entry

The preliminary theory of "cumulative disadvantage" in Case and Deaton (2017) emphasizes worsening opportunities at labor market entry — particularly for whites with low levels of education. They posit that these worse opportunities at labor market entry trigger various negative outcomes which build on each other, and culminate in an increased likelihood of untimely death. While it seems very likely that worsening economic conditions interacted with and potentially exacerbated the poor health of post-1946 cohorts, the time series pattern of real wages make it seem unlikely that labor market entry was the critical period where these cohorts first fell behind.

Appendix Table 9 shows the results of estimating trend break models to annual series for real mean log wages, the 10th percentile of real log wages, and mean real log wages for workers with

[25]The magnitude of the trend break is also very small so would require an enormous difference in health between children of native and foreign-born parents to drive the health decline. There is also a very small slowing in the rate of increase in the share of fathers and mothers with a high school diploma in the 1950s, though both continue to increase. Additionally, the small effect of this slowing seems likely to be offset by continued or accelerating increase in the share of mothers and fathers with a bachelor degree.

a high school degree, separately for men and women.[26] The series are also plotted in Appendix Figure 14. These results show rapid wage growth for men from 1962 until approximately 1972 — 1970 at the earliest —- and stagnation or decline thereafter. For women there is rapid growth, followed by a slowing which is less severe and begins later than that for men.

By these measures the quality of the labor market at entry (eg. age 19) was improving for men born between approximately 1942 and 1952. It was only after 1972, or 1970 at the earliest, that the real wages of men with less education and at the bottom of the wage distribution began to fall. If these measures are reasonable proxies for labor demand conditions then scarring effects would need to be *very* non-linear in age to contribute substantially to the cohort health decline. For example, in 1973, the first year of clear wage decline, the 1945 cohort was approximately 25, the 1946 cohort was approximately 24, and the 1947 cohort was approximately 23. For exposure to this poor labor market to contribute to the the observed health decline, there would need to be a large scarring effect on 23-year-olds, but little or no lasting effect on workers 24 and older. Similar arguments apply to other years. The wage patterns for women appear even less consistent with conditions at labor market entry an important role in the cohort health decline. Alternatively, it would need to be that the rapid wage growth between 1964 and 1972 masks some other feature of the labor market which was uniquely bad for young white men and women. [27]

8.4.5 Other pollution

A number of other forms of pollution do not appear likely to be important causal factors. Appendix Figure 15 and Appendix Table 10 show trends in eight important air pollutants from the Community Emissions Data System (O'Rourke et al.). None of them show evidence of trend breaks leading to more rapid increases after the late-1940s. Use of the pesticide DDT grew rapidly after it was made publicly available in 1945, however its use then began to decline rapidly after 1959 (US Environmental Protection Agency, 1975). Also, the evidence on the human health effects of DDT exposure is still mixed, in contrast to that for lead exposure (Turusov et al., 2002; Beard et al., 2006; Eskenazi et al., 2009). Atmospheric nuclear testing also began in 1945 and continued until 1963. However, while fallout from these tests covered the entire US, exposure was especially severe in certain geographic areas, including particularly a few southwestern states downwind of

[26] All series are calculated for full-time, full-year workers from the March Annual Social and Economic Supplement of the Current Population Survey (CPS).

[27] Another explanation, building on Greenwood and Yorukoglu (1997), could be that there was a level shift in some latent factor in 1974 which worked against demand for young unskilled workers. If each year of exposure early in ones career (under the age of 27) matters for later health this level shift in the demand factor in 1974 could generate a sharp trend break in cohort health around the 1946 cohort. However, to generate a sharp cohort trend break, workers above the age of 27 would have to be very protected from this scarring.

the Nevada Test Site (Meyers, 2019). The health decline is not particularly large in these states.

9 Conclusion

In this paper I document a sharp pattern in white mortality rates which suggests that the health of white women born since 1949 and white men born since 1946 suddenly declined relative to the trend for preceding cohorts. The uncovering of this apparent cross-cohort health decline could motivate further research in two directions: one trying to uncover the root cause of the decline; and two, exploring its implications beyond mortality.

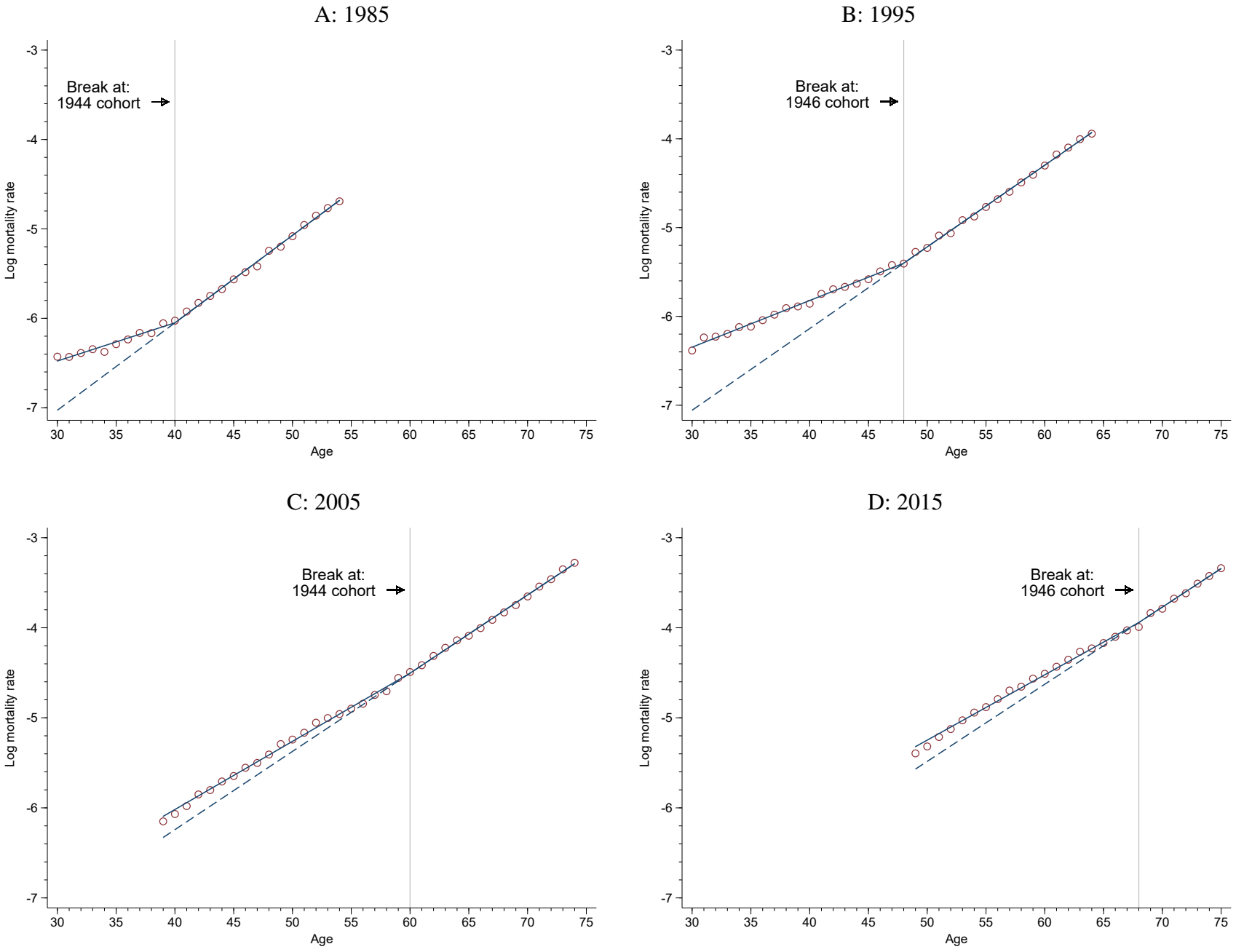
I provide a preliminary investigation into the root causes of the apparent health decline. I focused on national trends partly due to data availability but also because the health decline appears to be remarkably widespread across the US. The lack of an obvious geographic pattern in the health decline may make the search for a cause more difficult — this is not merely a Rust Belt problem with a Rust-Belt-specific cause for example. Future research will therefore need to compile large and detailed data to precisely summarize any variation in the health decline which does exist, and to allow for sharp tests of particular theories of it's cause. For example, data linking individuals' death records with their siblings could allow for a more complete test of the role of birth order and family background — by allowing a researcher to include family fixed-effects and birth order effects. A large sample linking adult mortality to individuals' detailed place of birth could be used to examine the role of environmental exposures and other negative childhood shocks.

Further, the health decline across cohorts of white Americans that I document seems likely to have broad implications beyond the recent mortality increase. These cohorts are beginning to enter old age and their depressed health could increase health care spending, depress labor force participation, and impact the solvency of specific programs such as Medicare and Social Security. In ongoing work I am studying the apparent impacts of this health decline on outcomes besides mortality (Reynolds, 2023).

The relative cohort health decline is already having a large impacting on period life expectancy. A simple model-based counterfactual suggests that in 2019 life expectancy of white men would have been 1 year higher, 77.3 rather than 76.3, if the relative cohort health decline had not occurred. For white women life expectancy would have been .4 years higher, 81.7 rather than 81.3.[28] Without a rebound of some kind, the impact on cohort life expectancy could ultimately be much greater.

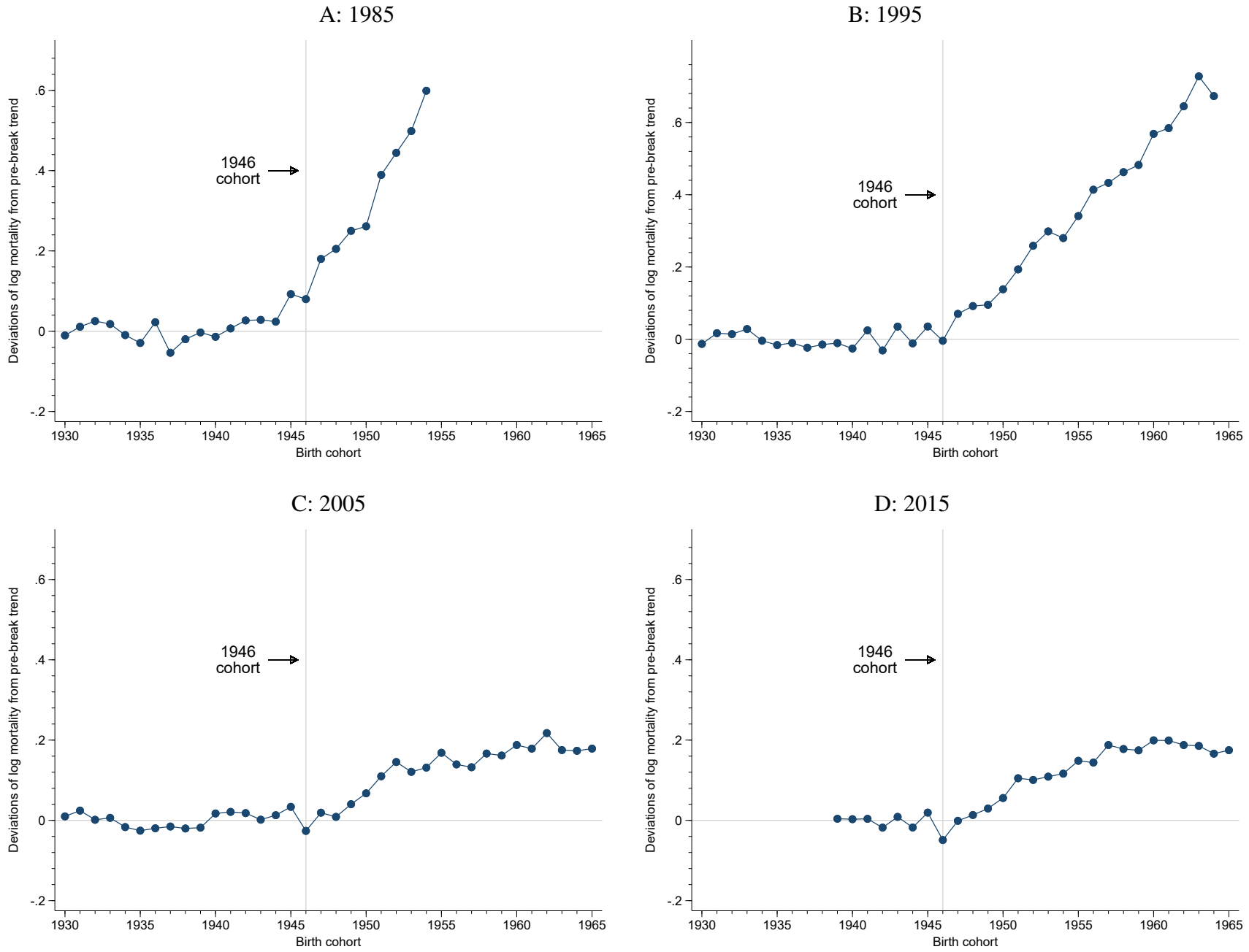
[28] See Appendix C for the details of this calculation.

Figure 1: Trend break in log mortality rates, white men — select years



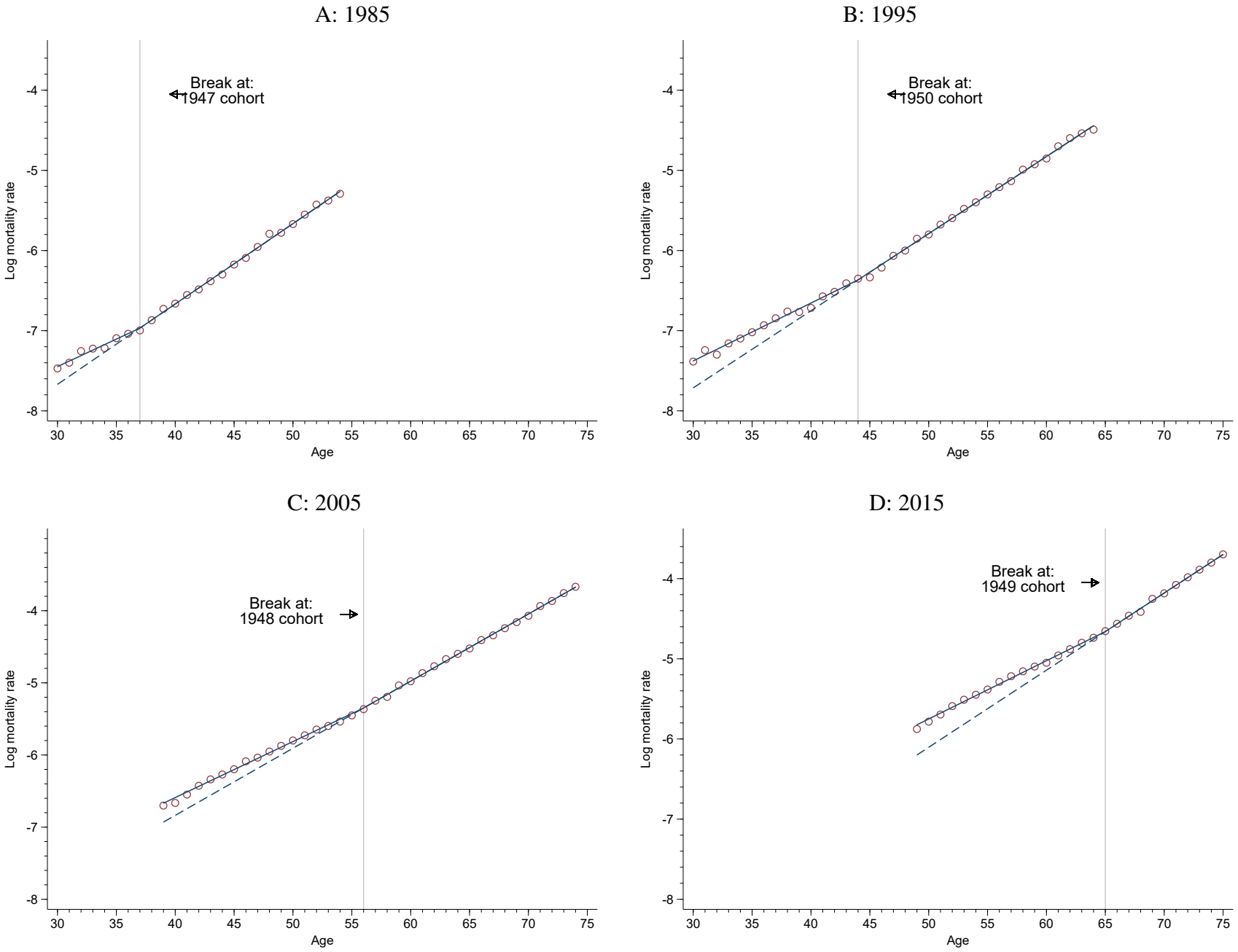
Each plot shows the log mortality rate of white men by age for the year listed, for 1930 to 1965 cohorts. Red circles show the observed log mortality rate by single year of age. The solid blue line shows plots the piecewise-linear, trend-break model estimated by weighted-least squares based on equation 2. The vertical gray line shows the age/cohort of the estimated break in trend. The dotted blue line extrapolates the linear trend for cohorts born before the break to post-break cohorts.

Figure 2: Deviations of log mortality from trend for pre-break cohorts, white men — select years



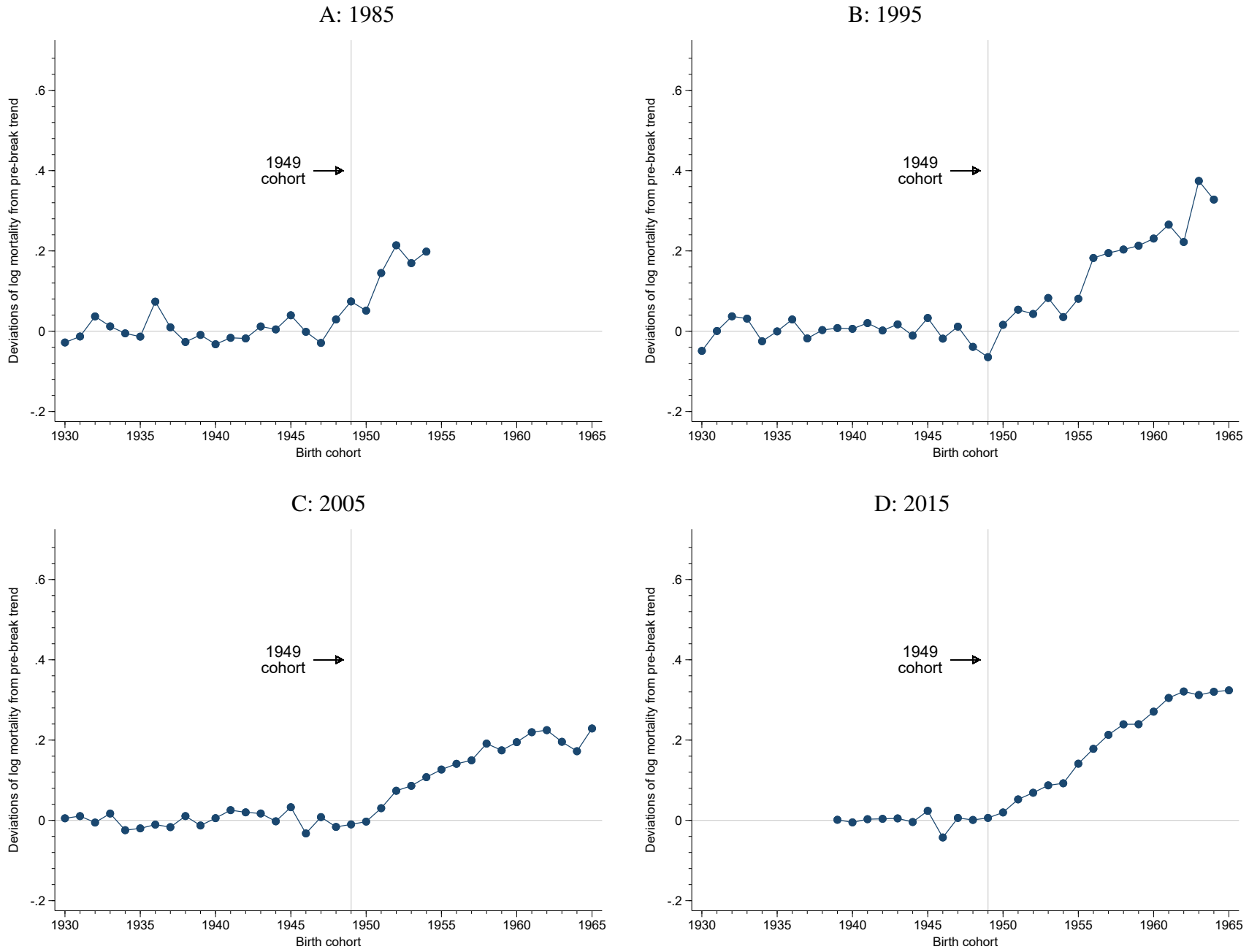
Each plot shows the deviations of the true log mortality rates of white men from the estimated linear trend for cohorts born before an estimated trend break for the year listed. The initial piecewise-linear, trend-break model is estimated by weighted-least squares based on equation 2, following the approach outlined in Hansen (2000). A horizontal gray line is plotted at the 1946 cohort. Sample is restricted to men born between 1930 and 1965.

Figure 3: Trend break in log mortality rates, white women — select years



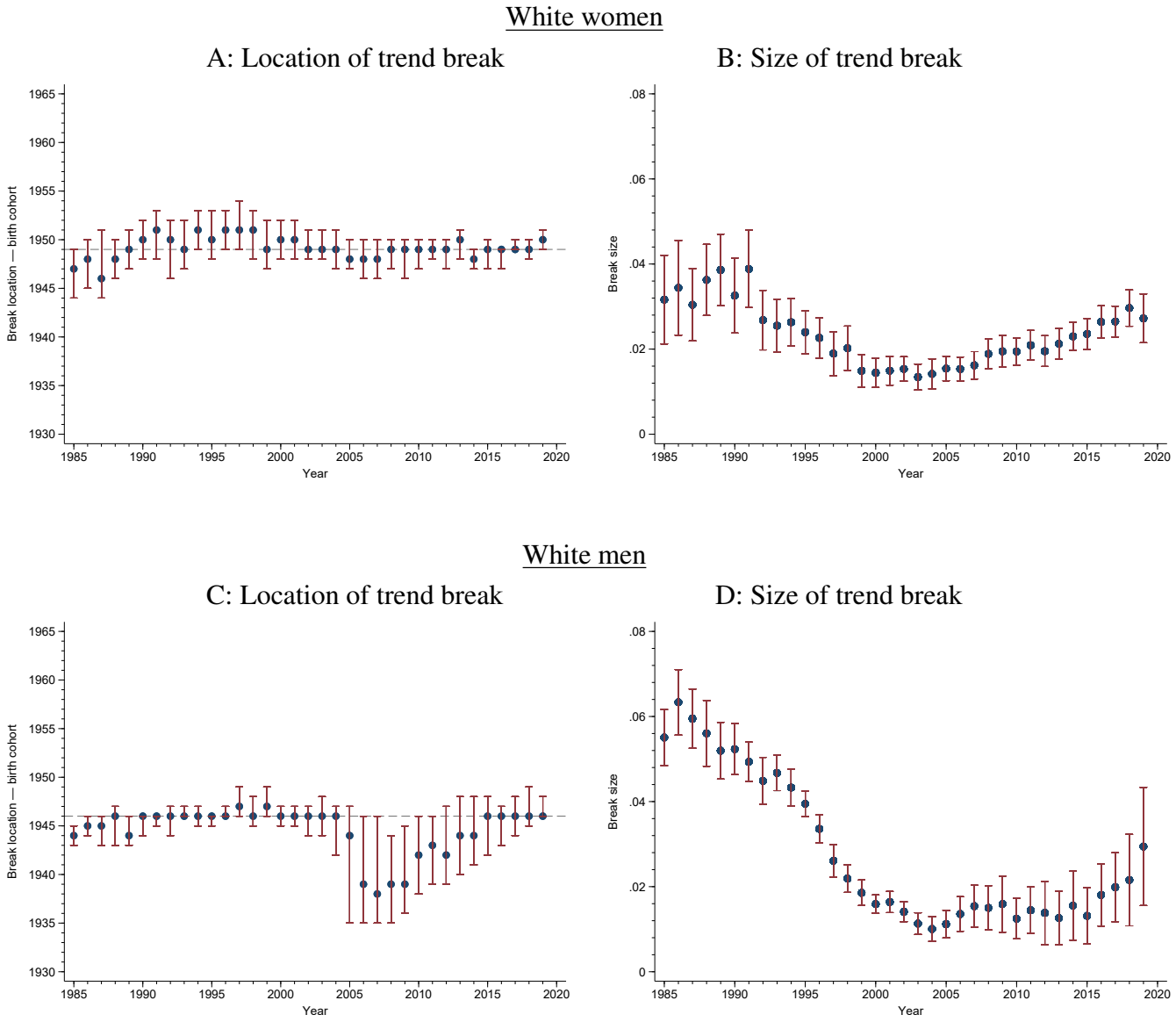
Each plot shows the log mortality rate of white women by age for the year listed, for 1930 to 1965 cohorts. Red circles show the observed log mortality rate by single year of age. The solid blue line shows plots the piecewise-linear, trend-break model estimated by weighted-least squares based on equation 2. The vertical gray line shows the age/cohort of the estimated break in trend. The dotted blue line extrapolates the linear trend for cohorts born before the break to post-break cohorts.

Figure 4: Deviations of log mortality from trend for pre-break cohorts, white women — select years



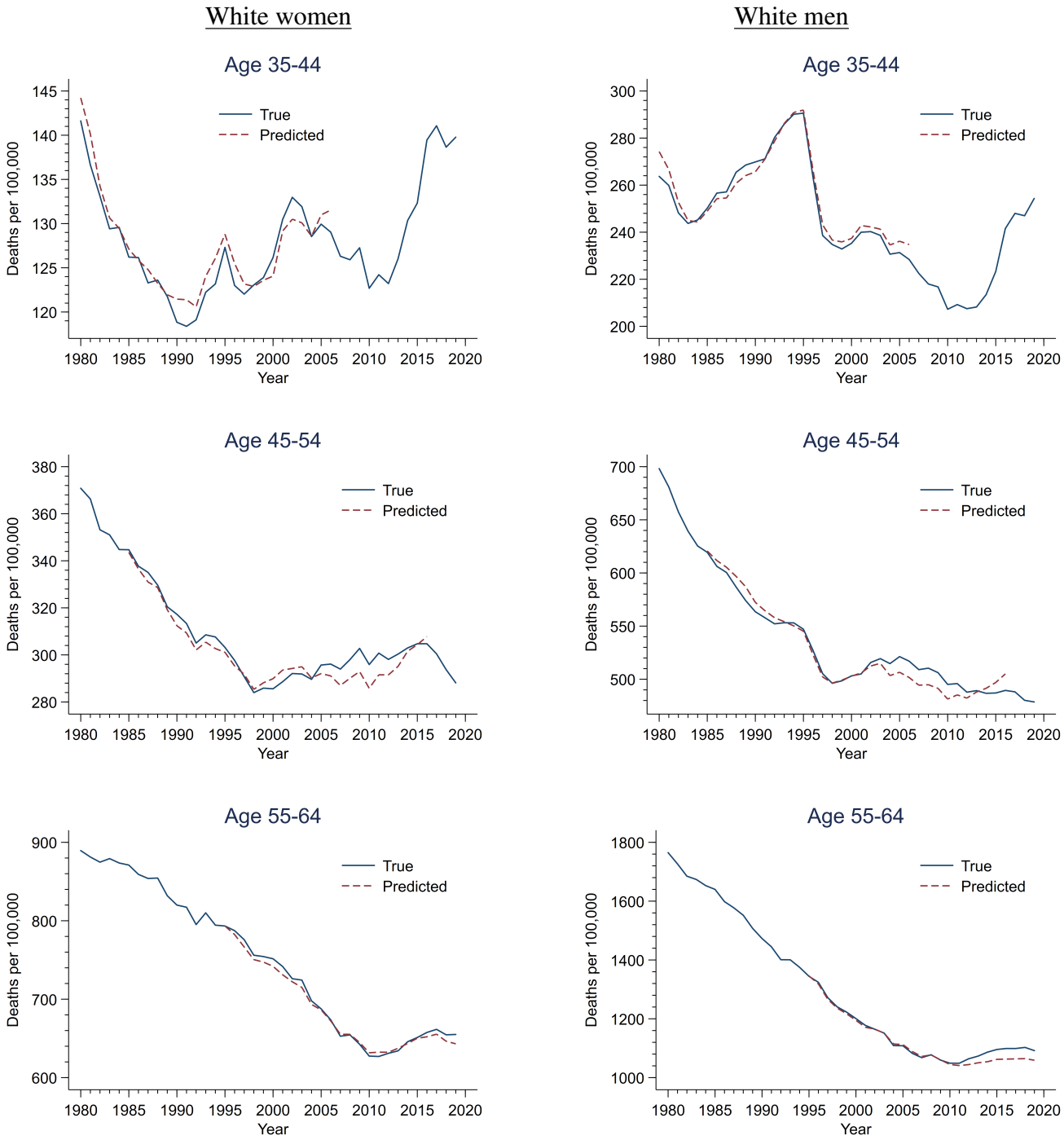
Each plot shows the deviations of the true log mortality rates of white women from the estimated linear trend for cohorts born before an estimated trend break for the year listed. The initial piecewise-linear, trend-break model is estimated by weighted-least squares based on equation 2. A horizontal gray line is plotted at the 1946 cohort. Sample is restricted to men born between 1930 and 1965.

Figure 5: Log mortality trend break and size estimates, separately by year



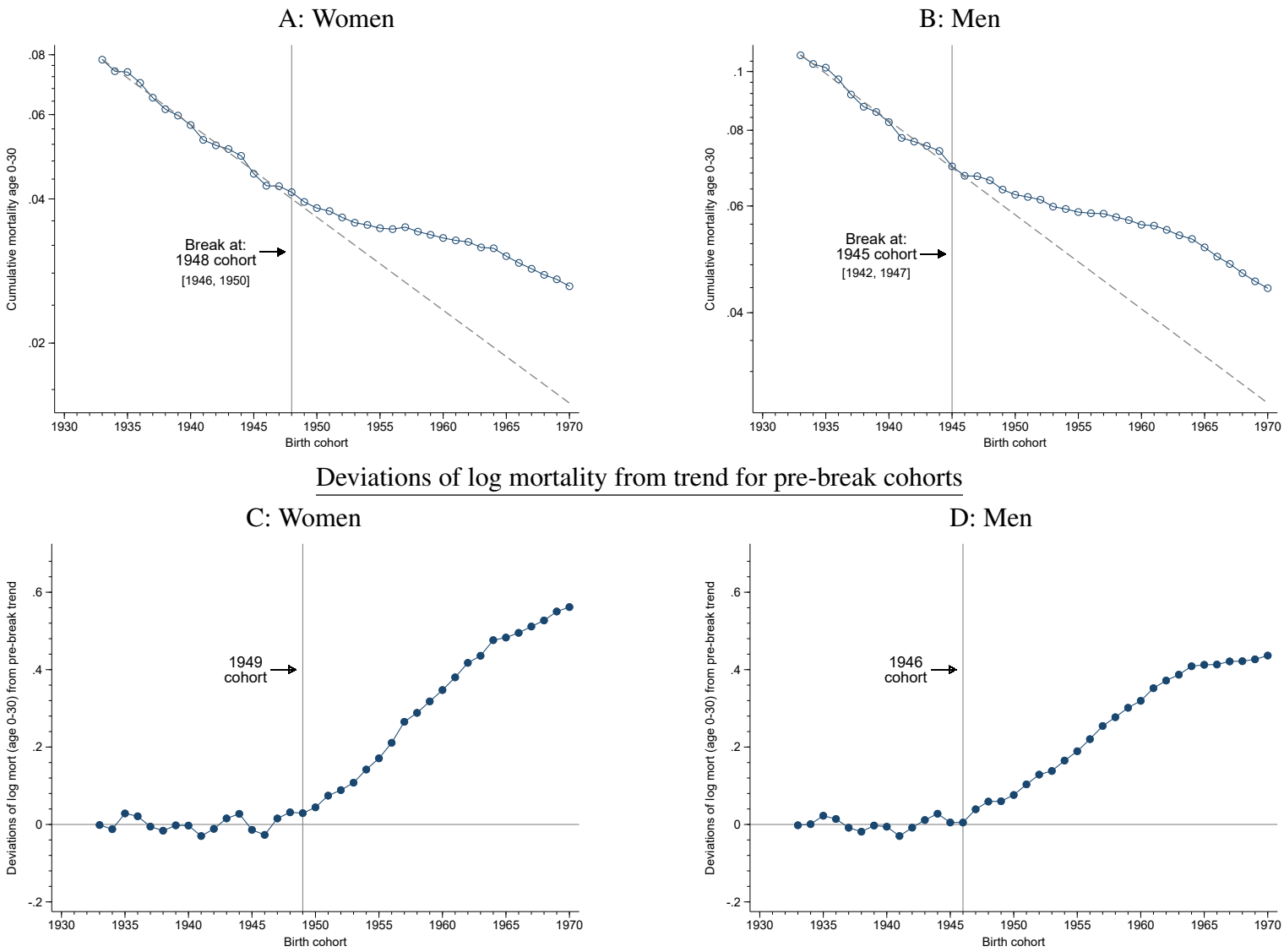
These figures show the results of estimation of the trend break model in equation 6, with the log mortality rate of white women or men by age, year and cohort as the dependent variable. A separate model is estimated for each year by weighted least squares, following the approach outlined in Hansen (2000). The sample includes ages 30-75, and cohorts 1930 to 1965. The left panel shows for each year of data the estimated location of the cohort specific trend break, $\hat{\gamma}^p$, as well as the 99 percent confidence interval calculated by inverting a likelihood ratio test statistic. The right panel shows for each year the size of the estimated trend break, $\hat{\delta}^p$.

Figure 6: True mortality rates and those predicted by a model with cohort-specific trend break



Each plot shows the true age-adjusted mortality rates and those predicted from a model including a cohort-specific trend break, for white women or men by year, for 35-44, 45-54, 55-64, year olds respectively. The true age-adjusted mortality rates are the simple average across single ages — ie. age adjusted assuming a uniform population distribution by age. To form the predicted series, I use the estimation results from a shared trend break model based on equation 5. I use the specification including a full set of year fixed-effects and a separate linear age effect for each year, reported in column 2 of Table 1. For each age-year cell I predict log mortality using this model, and then calculate predicted mortality as the natural exponential of predicted log mortality, for each single age-by-year pair. Finally, I calculate the simulated age-adjusted mortality for age-bins as the simple average across single ages.

Figure 7: Cumulative mortality from age 0-30 by birth cohort, white men and women



Panels A and B show estimates in hollow circles of the cumulative mortality rate from age 0 to 30 by year of birth for White Women and White Men respectively. The vertical gray line shows the cohort of the estimated break in trend from estimation of a piecewise-linear trend-break model of log mortality estimated by least squares. The estimated cohort break location is also written with the confidence interval in brackets. The dashed line shows the estimated linear trend for cohorts born before the break, and extrapolates it to post-break cohorts. Panels C and D show the deviations of the true log mortality rates from the estimated linear trend for cohorts born before the estimated break in trend, ie. the difference between the hollow circles and dashed lines in Panels A and B. Cumulative mortality rates for each cohort are calculated by decrementing each cohort using observed infant mortality rates and crude death rates at each from 1 to 30. These underlying rates are calculated based on birth and death counts from Vital Statistics volumes 1933-1959, the Multiple Cause of Death File 1959-2000, and population estimates from SEER and Census. Single-age mortality rates are used age 1 to 4, and five-age mortality rates from ages 5 to 30 (eg. 5-9, 10-14) are used assuming a constant death rate within each bin.

Table 1: Shared cohort-specific trend break, log mortality of white Americans 1980-2019

	(1)	(2)	(3)
Panel A: White women			
Average size of break	0.027	0.035	0.025
	(0.002)	(0.003)	(0.003)
Location of break	1948	1949	1950
	$[1948, 1949]$	$[1949, 1949]$	$[1950, 1950]$
P-value for existence of break	$< .001$	$< .001$	$< .001$
Panel B: White men			
Average size of break	0.034	0.033	0.026
	(0.001)	(0.001)	(0.002)
Location of break	1946	1946	1946
	$[1946, 1946]$	$[1946, 1946]$	$[1946, 1946]$
P-value for existence of break	$< .001$	$< .001$	$< .001$
Year FEs	Yes	Yes	Yes
Linear-age-by-year	Yes	Yes	Yes
Quadratic-age-by-year	No	Yes	Yes
Cubic-age-by-year	No	No	Yes

Each column shows the results of estimation of a model based on equation 4, with the log mortality rate of white men or women for single age-by-year bins as the dependent variable. All models are estimated by weighted least squares, following the approach outlined in Hansen (2000). The sample includes the years 1980-2015, ages 30-75, and cohorts born from 1930-1970. The row titled "Average size of break" reports the average value of $\delta_{2,c}$ across all years, with the standard error in parentheses calculated by the delta method. The row titled "Location of break" reports the estimated cohort at which a trend break occurs, with a 99 $\%$ confidence interval in brackets calculated by inverting the likelihood ratio statistic. The row titled "P-value for existence of break" reports p-value from an F-type test for the null hypothesis that no trend break occurs, based on 1000 bootstrap samples.

Table 2: Shared cohort-specific trend break, log mortality
By Race, 1980-2019
Cohorts born 1930 to 1960

	(1) Whites	(2) Blacks	(3) Other races
Panel A: Women			
Average size of break	0.0306	0.0118	0.0200
	(0.0020)	(0.0012)	(0.0060)
Location of break	1949	1946	1948
	[1949, 1950]	[1944, 1946]	[1947, 1949]
P-value for existence of break	$< .001$	$< .001$	$< .001$
Panel B: Men			
Average size of break	0.0378	-0.0084	0.0285
	(0.0007)	(0.0026)	(0.0019)
Location of break	1946	1952	1944
	[1946, 1946]	[1951, 1952]	[1944, 1945]
P-value for existence of break	$< .001$	$< .001$	$< .001$
Linear age	Yes	Yes	Yes
Year FEs	No	No	No
Linear-age-by-year	Yes	Yes	Yes
Quadratic-age-by-year	No	No	No
Cubic-age-by-year	No	No	No

Each column shows the results of estimation of a model based on equation 4, with the log mortality rate of men or women, of the listed racial group, for single age-by-year bins as the dependent variable. All models are estimated by weighted least squares, following the approach outlined in Hansen (2000). The sample includes the years 1980-2019, ages 30-75, and cohorts born from 1930-1960. The row titled "Average size of break" reports the average value of $\delta_{2,c}$ across all years, with the standard error in parentheses calculated by the delta method. The row titled "Location of break" reports the estimated cohort at which a trend break occurs, with a 99 $\%$ confidence interval in brackets calculated by inverting the likelihood ratio statistic. The row titled "P-value for existence of break" reports p-value from an F-type test for the null hypothesis that no trend break occurs, based on 1000 bootstrap samples.

Table 3: Trend breaks in potentially important causal factors

		First break		Second break	
	Pre-trend	Location	Size	Location	Size
Panel A: Years of schooling (by cohort)					
white men	0.0797 (.0019)	1947 [1947, 1948]	-0.1110 (.0036)	1961 [1958, 1963]	0.0743 (.0059)
white women	0.0816 (.0011)	1949 [1948, 1949]	-0.0860 (.0023)	1963 [1961, 1965]	0.0703 (.0049)
Panel B: Simulated effect of birth order on log mortality (by cohort)					
white men	-0.0044 (.0002)	1947 [1946, 1948]	0.0073 (.0004)	1964 [1962, 1965]	-0.0129 (.0009)
white women	-0.0061 (.0003)	1947 [1946, 1948]	0.0107 (.0006)	1964 [1962, 1965]	-0.0201 (.0013)
Panel C: Cars (by year)					
Registered motor vehicles (millions)	0.583 (.063)	1945 [1944, 1946]	2.155 (.099)	1963 [1959, 1965]	0.915 (.195)
Motor vehicle fuel use (millions of gallons)	598 (114)	1944 [1943, 1946]	1,830 (153)		

Each row shows the estimation results of a separate trend break model which allow for two possible trend breaks of unknown location, with the listed dependent variable. All models are estimated using the sequential estimation approach suggested in Hansen (2000) for such models. The two columns titled "Location" reported the estimated location of the first and second trend breaks, respectively, with 99 % confidence intervals in brackets calculated by inverting the likelihood ratio statistic. The two columns titled "Size" report the magnitude of first and second trend breaks respectively, with standard errors in parentheses. The column titled "Pre-trend" reports the estimated trend prior to the first break. I also calculate a bootstrap-based F-test suggested in Hansen (2000), for the null of a model with one break versus the alternative of a model with two breaks. For all models, except that for fuel usage, the p-value of this test is .001 or smaller. Therefore, for fuel usage I report results from a model with just a single break.

For years of schooling, I pool data from the CPS MORG data 1990 to 2018, white individuals age 25 to 75, cohort is defined as age - year -1. I then calculate approximate average years of schooling for each cohort based on the 16 schooling categories, and estimate the trend break models for average years of schooling by birth cohort. The simulated effect of birth order on log mortality is derived from within-family estimates of the effect of birth order on mortality from Barclay and Kolk (2015) and observed birth order shares for white Americans from Vital Statistics. I then estimate the described trend break models by birth cohort on the simulated data. The data on registered motor vehicles and fuel use by year comes from Historical Statistics of the United States US Census Bureau (1975). The trend break models for these outcomes are estimated by *year*.

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A Lack of geographic variation

I show that the cohort-specific trend break in log mortality rates documented above is remarkably widespread across the United States, suggesting that the associated health decline in similarly widespread. To do so, I estimate the shared cohort-specific trend-break model of Section 4 separately for different states and regions of the United States.

First, I examine the location and size of the trend break by Census region. For each of the four regions, I estimate the trend break model based on equation 2 with a full set of year fixed-effects and a separate linear age effect for each year (similar national results are in column 2 of Table 1). I again follow Hansen (2000) and the procedure described above.

Panel A of Appendix Table 3 shows the results for white women. The precise cohort at which the trend break is estimated to have occurred varies only slightly across the four Census regions — from 1946 in the West to 1950 in the Midwest, with the estimates in the South and Northeast in between at 1948 and 1949 respectively. The average size of the estimated cohort break — the average value of $\delta_{2,c}$ across all years — ranges from a low of .018 in the West to a high of .024 in the Northeast. For all regions, the bootstrap procedure to test the null hypothesis that no trend break occurs suggests a P-value of less than .001.

Panel B shows analogous results for white men. The cohort at which the trend break is estimated to have occurred again varies only slightly across the four Census regions — falling at 1942 in the West, 1946 in the Midwest and South, and 1944 in the Northeast. The average size of the estimated cohort break for men are *remarkably* similar across the four regions. This size is estimated to be identical up to 3 digits — at .026 — for the Northeast, South, and West. While the estimate for the Midwest is not far off at .029. For all regions, the bootstrap procedure to test the null hypothesis that no trend break occurs suggests a P-value of less than .001

To further explore potential geographic variation in the cohort-specific trend break I next examine it separately for each of the 50 states in the U.S. Given the smaller sample size at the state-level, I impose the *location* of the cohort-specific trend break in each state to match that at the national level. That is, I estimate separately for each state the trend break model based on equation 2 but fix γ_c to be 1949 for women and 1946 for men. I again use a specification with a full set of year fixed-effects and a separate linear age effect for each year, and estimate by weighted least squares — using the implied variance of log mortality as weights. For each state I then calculate the average size of the estimated cohort break — the average value of $\delta_{2,c}$.

Appendix Figure 2 shows maps and histograms of the distribution of these estimated break sizes for the 50 states — and demonstrates a surprising lack of variation in the size of the estimated

breaks across states. No obvious regional patterns are apparent in the maps for either sex — the trend break is widespread across the United States. Further, *all 50 states* have estimated trend breaks which are positive in magnitude and greater than .01 for women and men. Estimates for women in all states are between .005 and .045, and 30 out of 50 states have estimated break sizes between .015 and .025. For men estimates range from .01 to .055, and 32 out of 50 states have break sizes between .025 and .035.

Appendix Figure 3 shows a scatter plot between the break sizes of men and women, and reveals a positive association. States with large breaks for men tend to also have large breaks for women. Alaska and Vermont stand out as states with large breaks for both men and women. On the other extreme, California and Florida have particularly small estimated breaks for both sexes. This positive association suggests that a single factor varying at the state-level may be driving health differences for both men and women. A regression of mens break sizes on womens break sizes confirms the positive relationship shown in the scatter plot. Using the estimated variance of the female break sizes from the first-step as weights I perform a second-step, state-level regression. The estimated coefficient on male break size is .659, with a standard error of .079 and a corresponding t-statistic of 8.29.

B Early-life mortality: Additional details and results

This appendix provides more details and additional results based on the mortality rates of White Americans under age 30.

As described in the data section, I combine death counts by age-sex-race from historical vital statistics volumes from 1933 to 1958 and counts from multiple cause of death data from 1959 with population estimates from the Census Bureau and SEER.[29] The historical data reports mortality by exact year of age up to age 4, and then only reports mortality in 5-year age bins for older ages. For consistency I aggregate post-1959 data into the same age bins.

I again use the framework of Hansen (2000) to test for the existence of and estimate the location of trend breaks by cohort. I estimate models of the following form, separately for particular ages or age groups a:

$$ln(mort_{apc}) = \beta_c^a c + \delta^a \cdot (\gamma^a - c) \cdot 1_{c \geq \gamma^a} + \mu^a + \epsilon_{apc} \tag{5}$$

where a denotes age, p denotes period (eg. year), c denote cohort; and $ln(mort_{apc})$ denotes the log-mortality rate of individuals age a, in period p, and from cohort c.[30] The parameters β_c^a represents

[29] I use Census Bureau population estimates for 1933-1968 and SEER population estimates from 1969 on.

[30] For the five-year age bins the cohort is defined based on the youngest age in the bin. Eg. for the 5 to 9 year old

a linear trend in cohort for log mortality at age a. I then allow a trend break by cohort — thereby letting the affect of cohort have a piecewise linear form. The size of the trend break is represented by δ^a. The precise cohort at which the trend break occurs is treated as unknown and a parameter to be estimated, γ^a.

I calculate the cumulative mortality rate by first assuming that mortality is uniformly distributed by age within the five-year age bins and then decrementing each cohorts mortality using the single age mortality rates. This will introduce some measurement error in mortality for ages over 5, potentially smoothing and understating the size of the break because cohorts will inherit some of the mortality of nearby cohorts in the same age bin. For example, when the 1946 cohort is 9 it will share the 5-9 age bin with the 1947-1950 cohorts and will be assigned an average mortality which includes these cohorts potentially elevated mortality.

Appendix Figure 4 disaggregates these results by age, and shows broadly that this cohort specific trend break of mortality is evident across ages from infancy through childhood. It reports the results of estimating cohort trend break models separately for log mortality rates by single years of ages below age 4, and for 5-year age bins up to age 19. For all ages between 0 and 19 a trend break is estimated to occur somewhere between the 1940 and 1951 cohort, and the trend breaks are all estimated to have positive sign: implying a slowing of improvements in mortality after that cohort. The confidence intervals for the trend breaks for the single ages 1 to 4, as well as those for 5-9, for females all include 1949 and those for men all include 1946 — consistent with the estimates in Figure 7 and for adult mortality earlier in the paper. Those for infant mortality and for ages 10-14 and 15-19 differ slightly: with infant mortality for both sexes estimated to break a few cohorts later, and that for preteens and teens estimate to occur a few cohorts earlier (but with large confidence intervals).

The patterns by birth cohort of log mortality of white Americans in their twenties are different than that described above for earlier ages. However, the mortality rate at these ages is mostly be driven by external deaths (see eg. Figure A1 in Schwandt and Von Wachter (2020)) which seem likely to be less informative about the underlying "health" of these cohorts. Mortality rates at ages 20 to 29 do not exhibit any evidence of a health decline for the post-late-1940s cohorts, like that documented at older ages in earlier sections and potentially suggested at younger ages in this section. There are some fluctuations in mortality for these cohorts but they appear to be driven by period-specific phenomenon — for example there is a sharp increase in mortality from homicide, suicide, and drug poisonings beginning in the late 1960s, and mortality from HIV/AIDS sharply

age group, cohort c is defined as $p - 5 - 1$. Therefore, if the mortality rate has a trend break in the year when some cohort c' first enters a bin, the break will be estimated to occur at cohort c'.

increasing after 1980.[31]

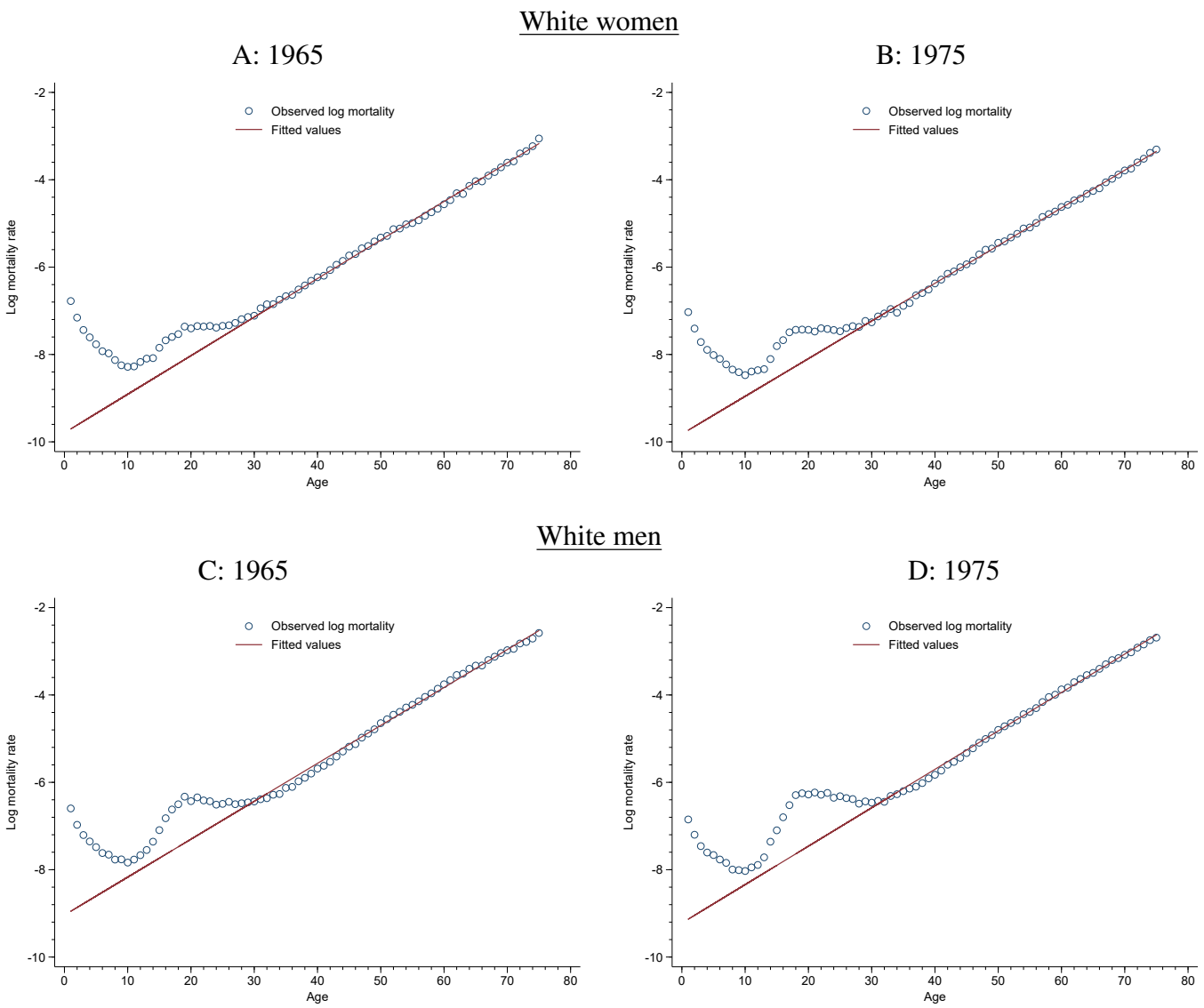
C Additional details on period life expectancy calculation

I estimate Equation 2 for log mortality rates in 2019, using the sample and methodology underlying Figure 5. I then form adjusted log mortality rates which remove the relative cohort health decline by subtracting $\hat{\delta} \cdot (\hat{\gamma} - c) \cdot 1_{c \geq \hat{\gamma}}$ from the observed log mortality rates in the sample. I then exponentiate these adjusted mortality rates and take the ratio with observed mortality, to form a mortality adjustment ratio r_a

I then use CDC life tables for non-Hispanic men and women in 2019 from Arias and Xu (2022) as a baseline. I form adjusted q_x values by multiplying q_x for each age in the above estimation sample by the mortality ratio r_a described above. I then use standard life table methods to calculate life expectancy using the adjusted q_x values.

[31] See Shahpar and Li (1999) for a discussion of the period-specific increases in the 1960s and 1970s in homicide mortality. My own informal, descriptive analysis suggests that there were sharp, period-specific (ie. occurring at the same year for all single-ages of the twenties) increases in the 1960s in homicide, suicide, and drug poisonings which were large enough to drive all-cause mortality increases. Results available by request.

Appendix Figure 1: Examples of fit of Gompertz curve for select pre-sample years

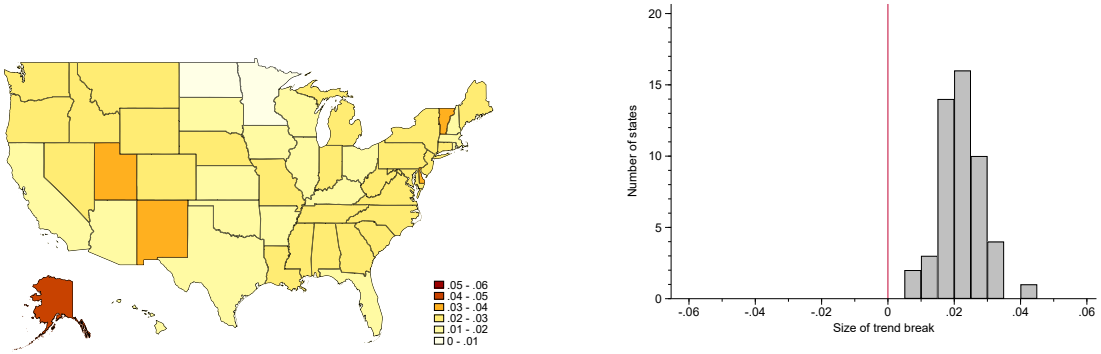


Each plot shows the log mortality rate of white men by age for the year listed, for 1930 to 1965 cohorts. Red circles show the observed log mortality rate by single year of age. The solid blue line shows plots the piecewise-linear, trend-break model estimated by weighted-least squares based on equation 2. The vertical gray line shows the age/cohort of the estimated break in trend. The dotted blue line extrapolates the linear trend for cohorts born before the break to post-break cohorts.

Appendix Figure 2: Little variation across states in size of cohort-specific trend break in log mortality

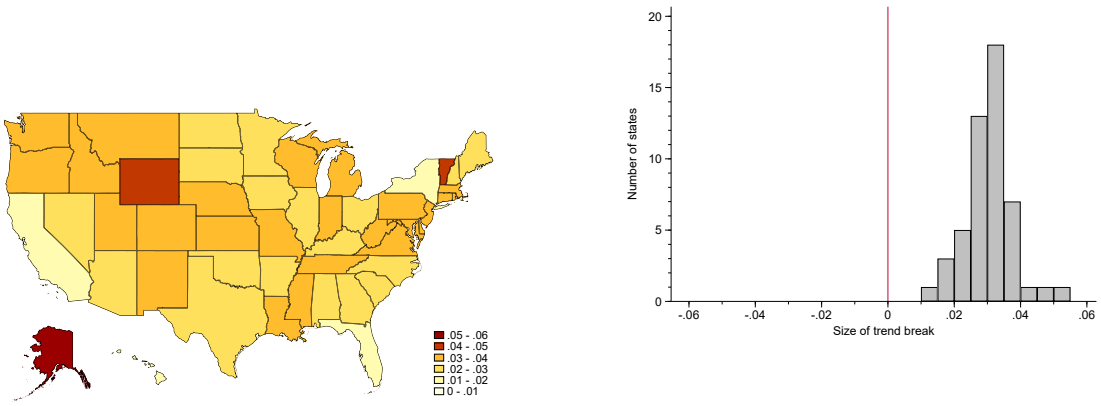
A: White women

break at 1949 cohort



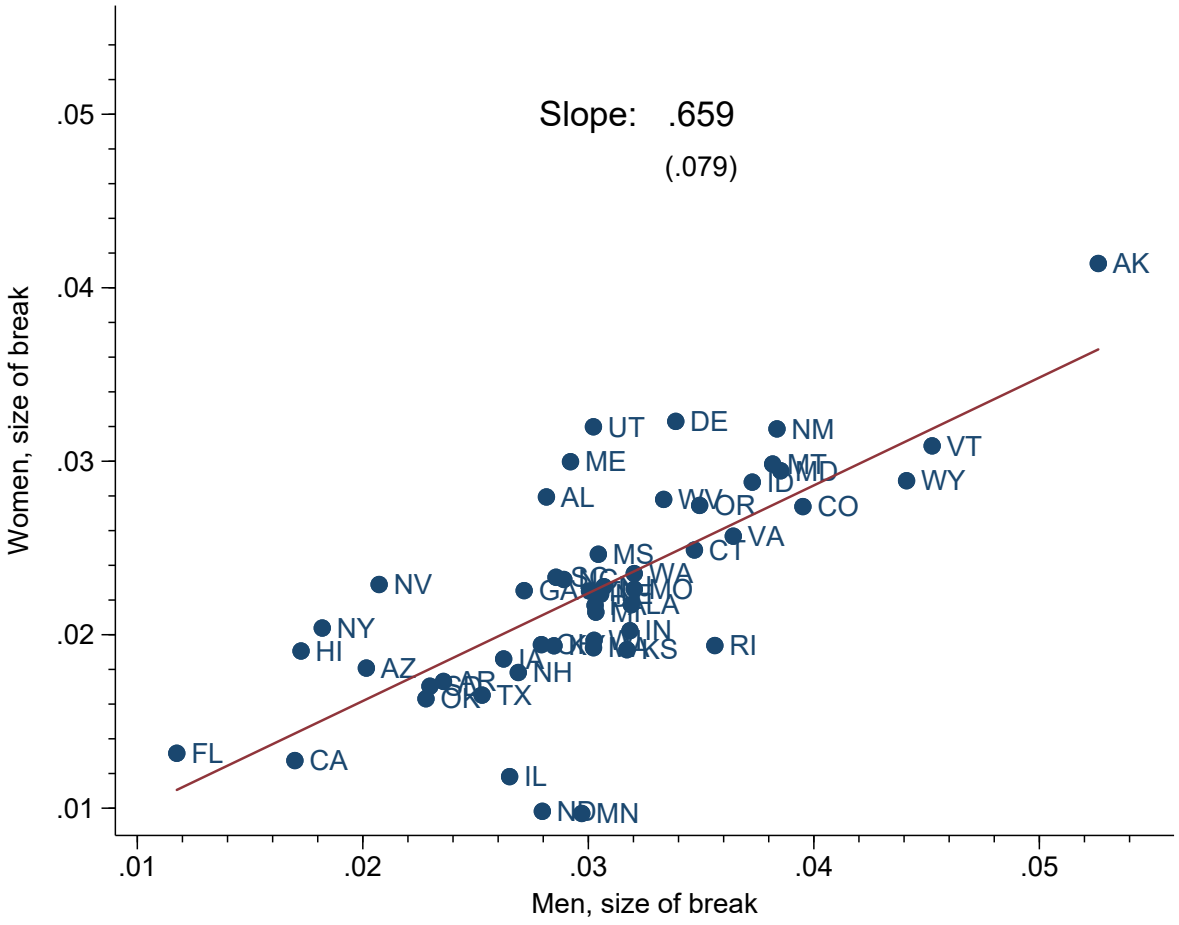
B: White men

break at 1946 cohort



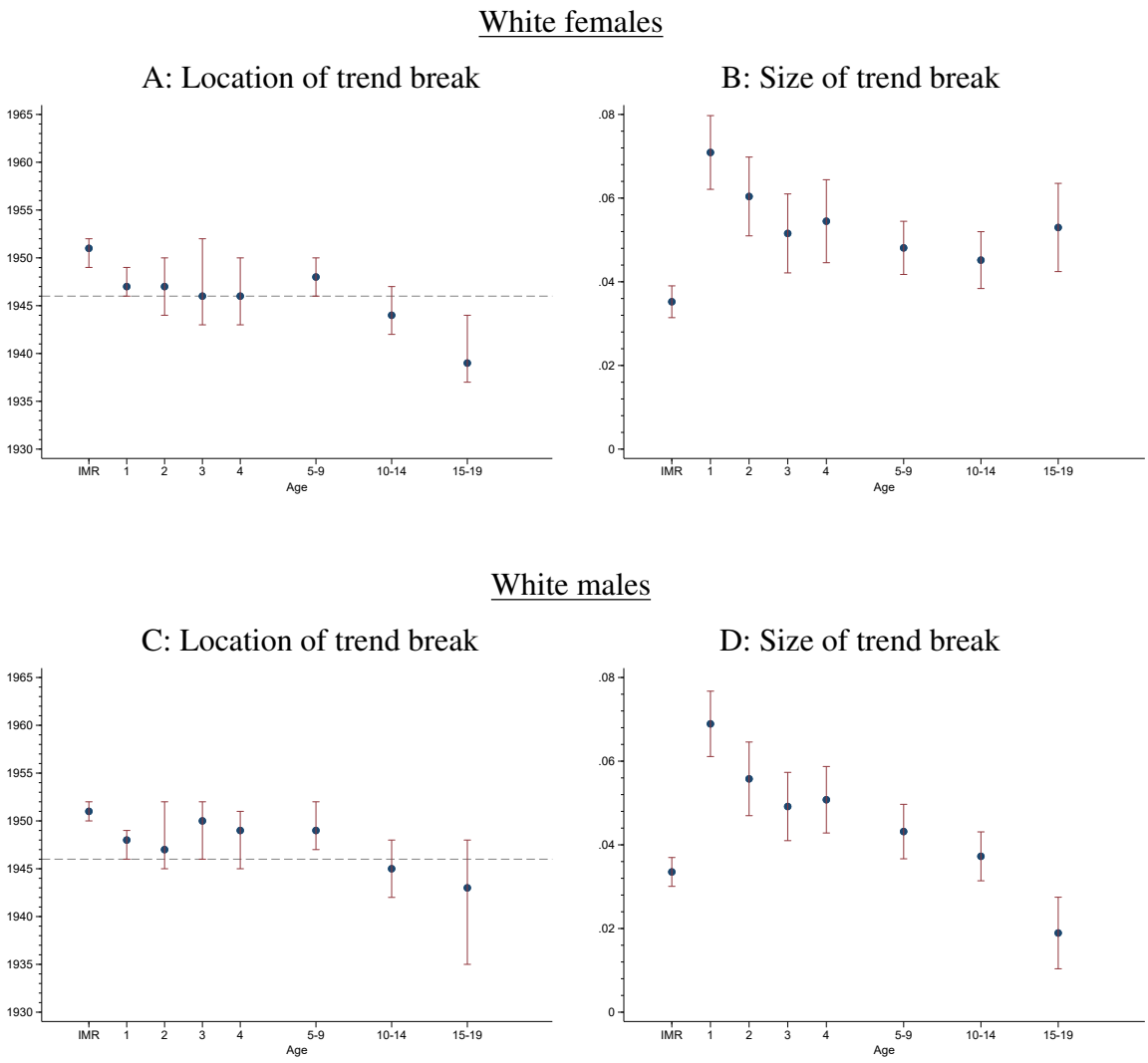
This figure shows the variation across states in the size of cohort-specific trend breaks in the log mortality of white women and men. All figures are based on separate estimation by state of cohort-specific trend break models of the log mortality of white women or men. Each model is based on equation 4 including a full set of year fixed-effects and a separate linear age effect for each year. The location of the trend break γ is treated as known — 1946 for men and 1949 for women — and estimation is done by weighted least squares. The sample includes the years 1985-2015, ages 30-75, and cohorts born from 1930-1970. For each state I calculate the average value of the trend break $\delta_{2,c}$ across all years. The maps display the values of these average trend break sizes for each state. The histograms show the distribution of these average trend break sizes across the 50 states.

Appendix Figure 3: Relationship between state-level size of cohort-specific trend break in white log mortality for women and men



This figure shows the relationship across the 50 states between the size of cohort-specific trend breaks in log mortality for white women and those for white men. The first step is separate estimation by state of cohort-specific trend break models of the log mortality of white women or men. Each model is based on equation 4 including a full set of year fixed-effects and a separate linear age effect for each year. The location of the trend break γ is treated as known — 1946 for men and 1949 for women — and estimation is done by weighted least squares. The sample includes the years 1985-2014, ages 30-75, and cohorts born from 1930-1970. For each state I calculate the average value of the trend break $\delta_{2,c}$ across all years. The above figure plots this average value for each state for men versus the average value for women in the same state. The second step is a regression with the estimated break sizes of women as the dependent variable and that of men as the independent variable. The variance of women's estimated break size from the first step are used as weights in the second step.

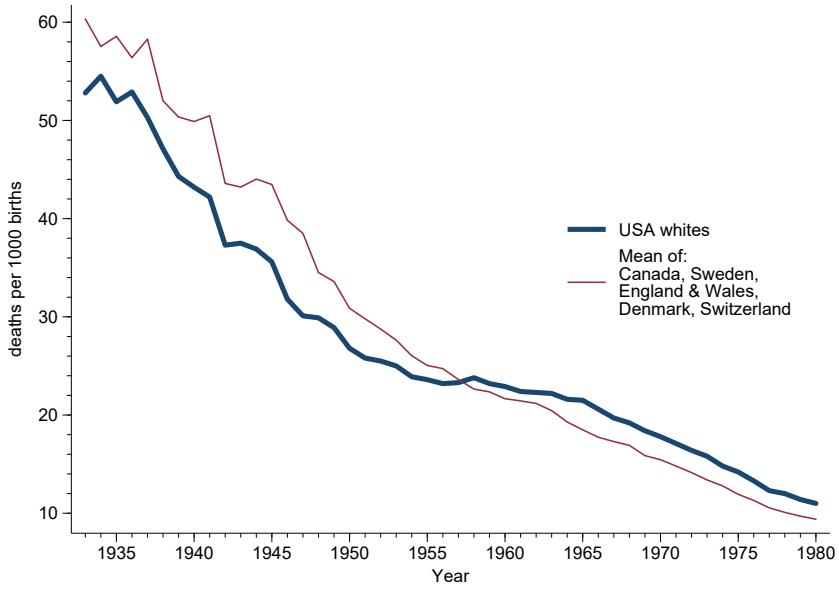
Appendix Figure 4: White childhood log mortality trend break estimates by age



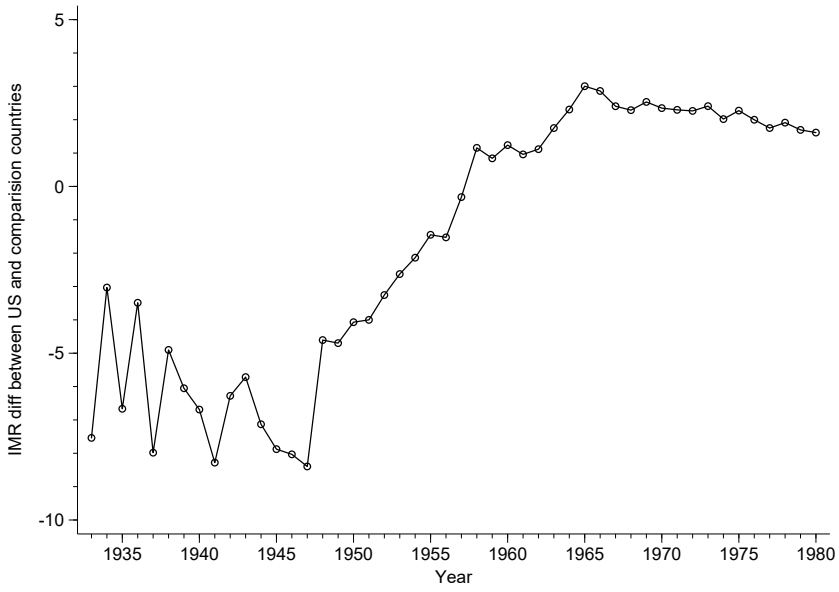
These figures show the results of estimation of the trend break model in equation 5, with the log mortality rate of white females or males by cohort and age as the dependent variable. A separate model is estimated for each age/age-group shown by least squares, following the approach outlined in Hansen (2000). IMR refers to the log of the infant mortality rate, ages 1 to 4 refer to the log mortality rate for single ages between 1 and 1, while the remaining points refer to the log mortality rate for the listed age bin based on decrementing the crude death rates for single years of age, and then taking the natural log (more detail in text). For the five-age bins cohort is defined based on the youngest age in the bin. Panels A and C report estimates of the cohort at which the break is located, $\hat{gamma}^a$ 99 percent confidence intervals. Panels B and D report estimates and 95 percent confidence intervals of the size of the break, δ^a of the The underlying rates are calculated based on birth and death counts from Vital Statistics volumes 1933-1959, the Multiple Cause of Death File 1959-2000, and population estimates from SEER and Census.

Appendix Figure 5: Infant mortality rate of White Americans in comparison to other English-speaking and European countries

A: IMR in US and comparison countries

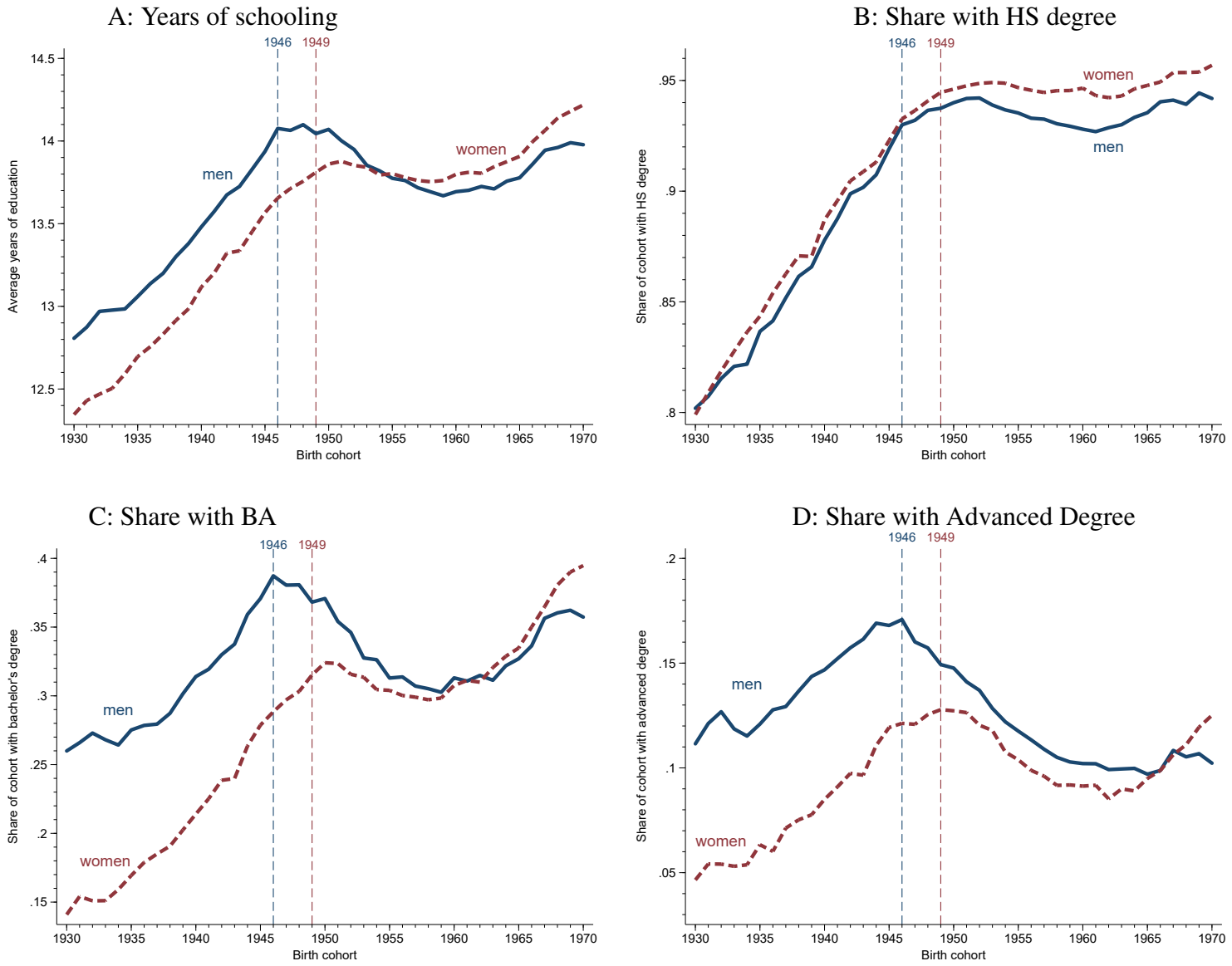


A: Difference in IMR between US and comparison countries



Panel A plots the infant mortality rate of white Americans based on vital statistics volumes and microdata, and the mean infant mortality rate across Canada, Sweden, England and Wales, Denmark, and Switzerland from the Human Mortality Database. Panel B plots the difference between these two series.

Appendix Figure 6: Cohort decline in educational attainment for white men and women

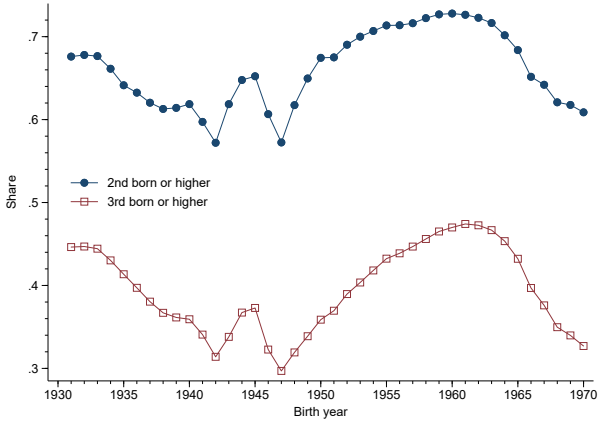


Data is from CPS Merged Outgoing Rotation Group and includes white men and women age 25-75 in years 1990-2018. Panel A plots the average years of schooling by birth cohort — approximated based on 16 educational categories. Panels B-D plot respectively the share of each birth cohort with a high school or GED degree, a bachelor's degree, and an advanced degree.

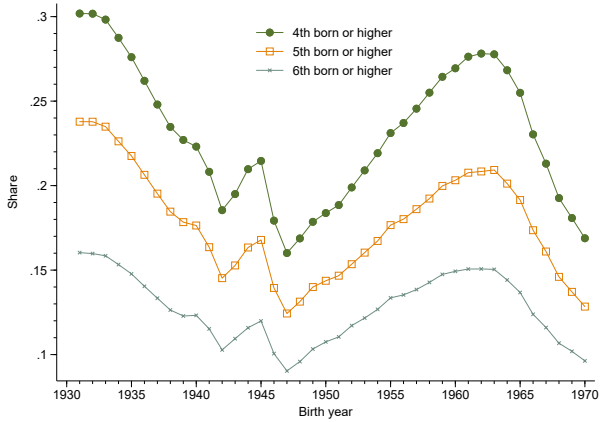
Appendix Figure 7: Birth order changes across white cohorts and simulated effect on log mortality

Birth order shares by cohort, white births

A: Second and third born or higher

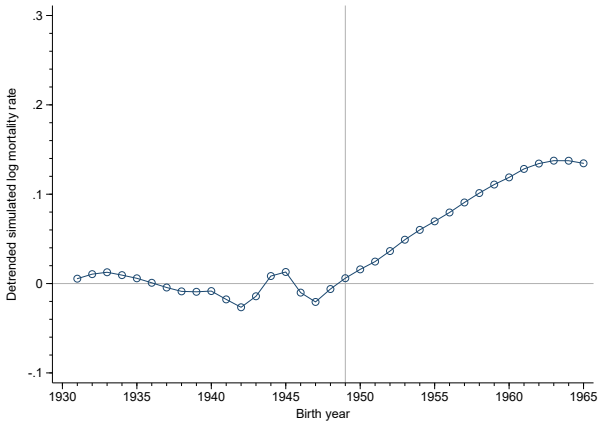


B: Higher birth orders

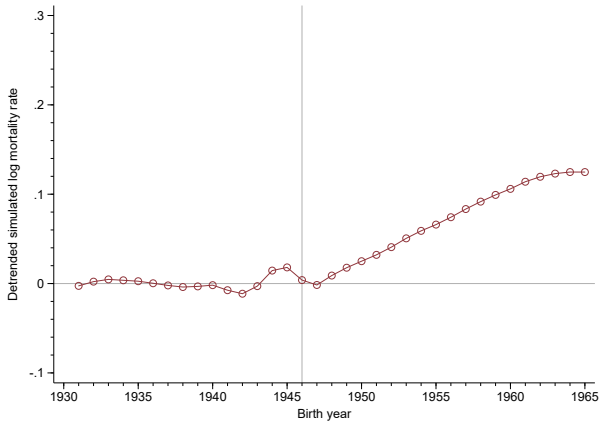


Simulated detrended log mortality by cohort — based on observed birth order changes

C: White women



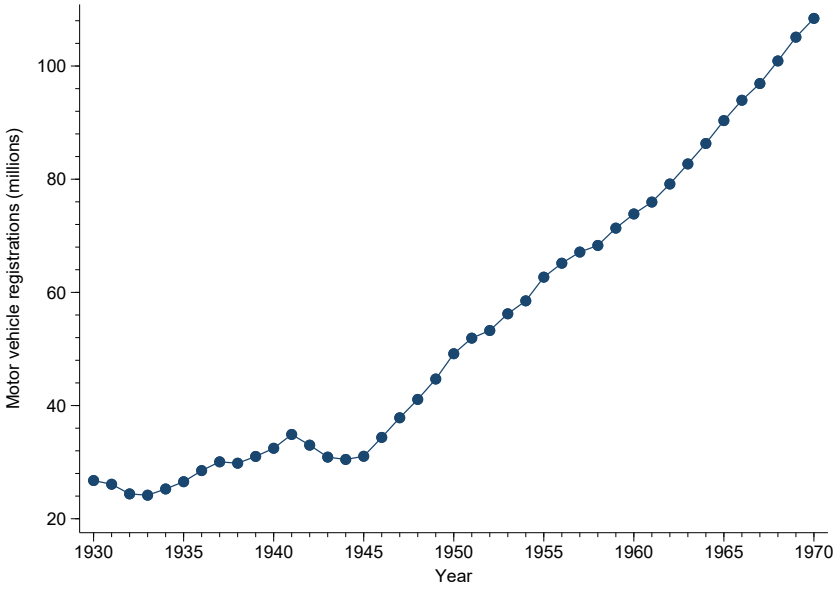
D: White men



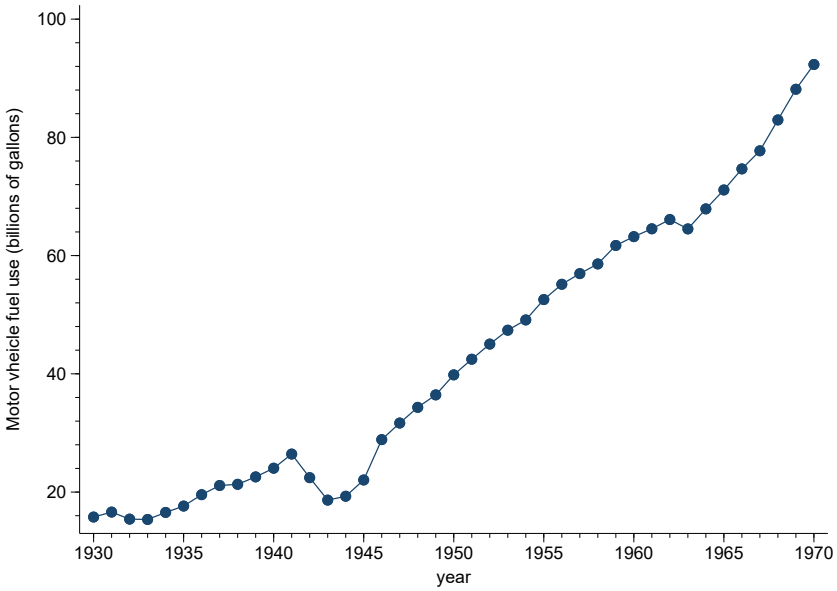
Panels A and B show the share of white births by cohort which are of the listed birth order (parity) or higher. Data for 1930-1939 are digitized from Vital Statistics reports, and for 1940-1970 are calculated from "U.S. Cohort and Period Fertility Tables, 1917-1980" compiled by Robert D. Hauser and available from the Office of Population Research at Princeton. Panels C and D report detrended simulated log mortality rates to show the impact of these birth order trends on log mortality rates by cohort. The simulation uses observed birth order shares, odds ratio estimates of the impact of birth order on mortality from Barclay and Kolk (2015), and observed mortality rates at age 40 of the 1949 and 1946 cohorts of white women and men, respectively. More details in text.

Appendix Figure 8: Motor vehicle registrations and fuel use

A: motor vehicle registrations



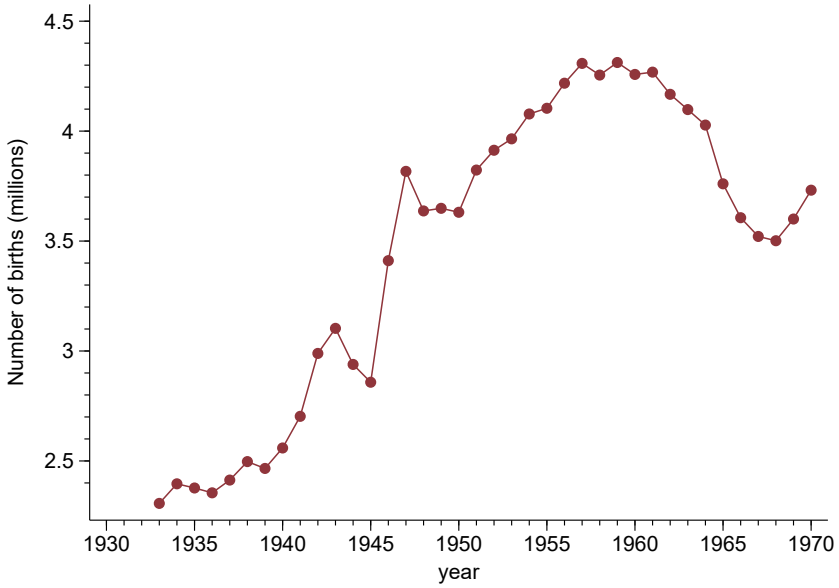
B: Fuel usage by motor vehicles



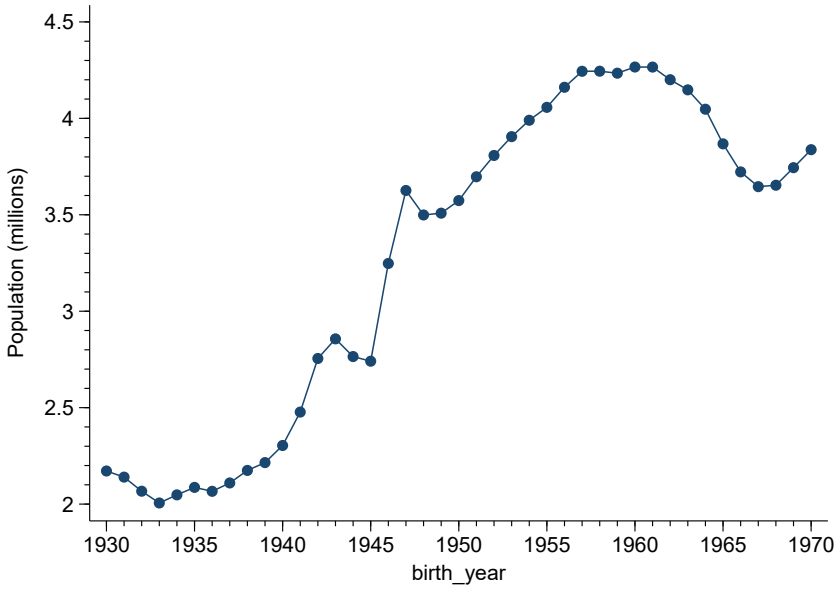
This figure shows annual time series of a) motor vehicle registrations and b) fuel usage by motor vehicles, for 1930 to 1970 in the United States. Both series come from Historical Statistics of the United States US Census Bureau (1975).

Appendix Figure 9: Cohort size

A: Number of births by year

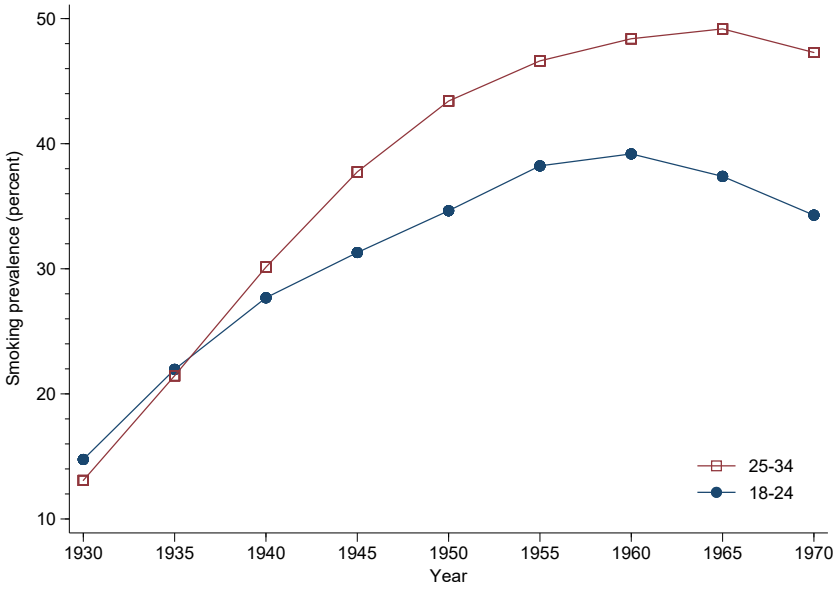


B: Population of each cohort at age 18



Panel A shows the number of births in the United States by year. Panel B shows the population of each cohort when they were age 18. Data comes from the Human Mortality Database, derived from vital statistics.

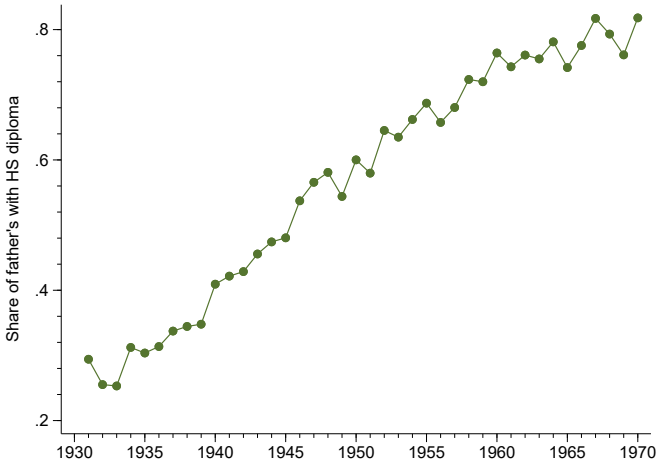
Appendix Figure 10: Estimates of smoking prevalence of American women of childbearing age



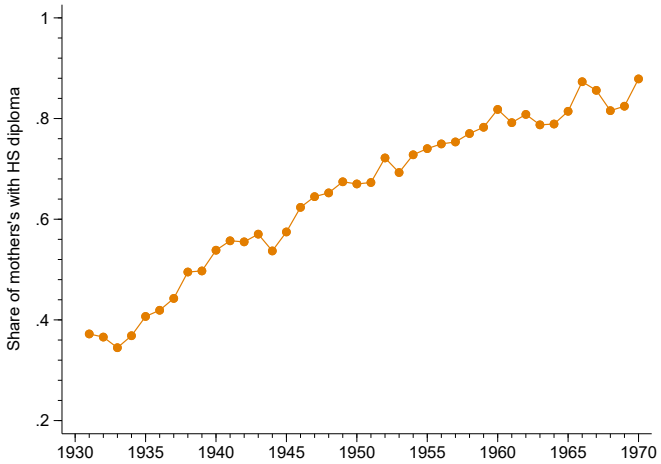
Based on estimates of smoking prevalence by age and year (every 5 years) from Holford et al. (2014), derived from survey data on retrospective smoking history. The figure plots age-adjusted smoking prevalence for women age 18-24 and 25-34 separately, assuming a uniform distribution of ages within age bins (ie. the unweighted average in each age bin across smoking rates by single year of age).

Appendix Figure 11: Parental education of White Americans by birth cohort

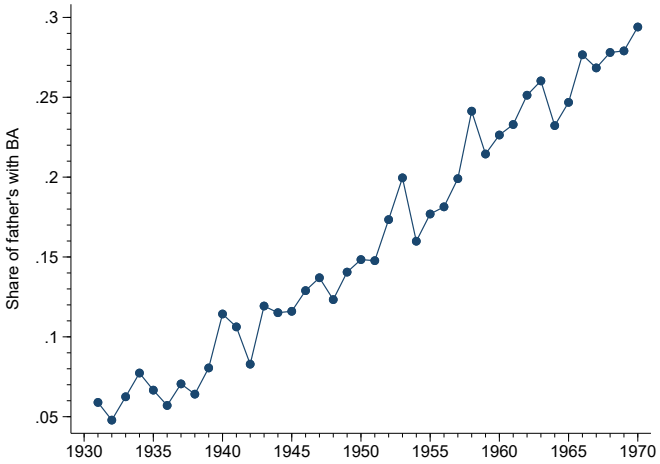
A: Share of fathers with high school diploma



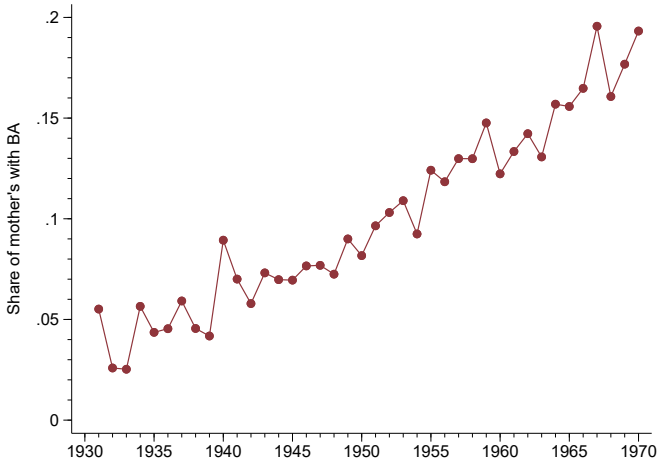
B: Share of mothers with high school diploma



A: Share of fathers with Bachelor's degree

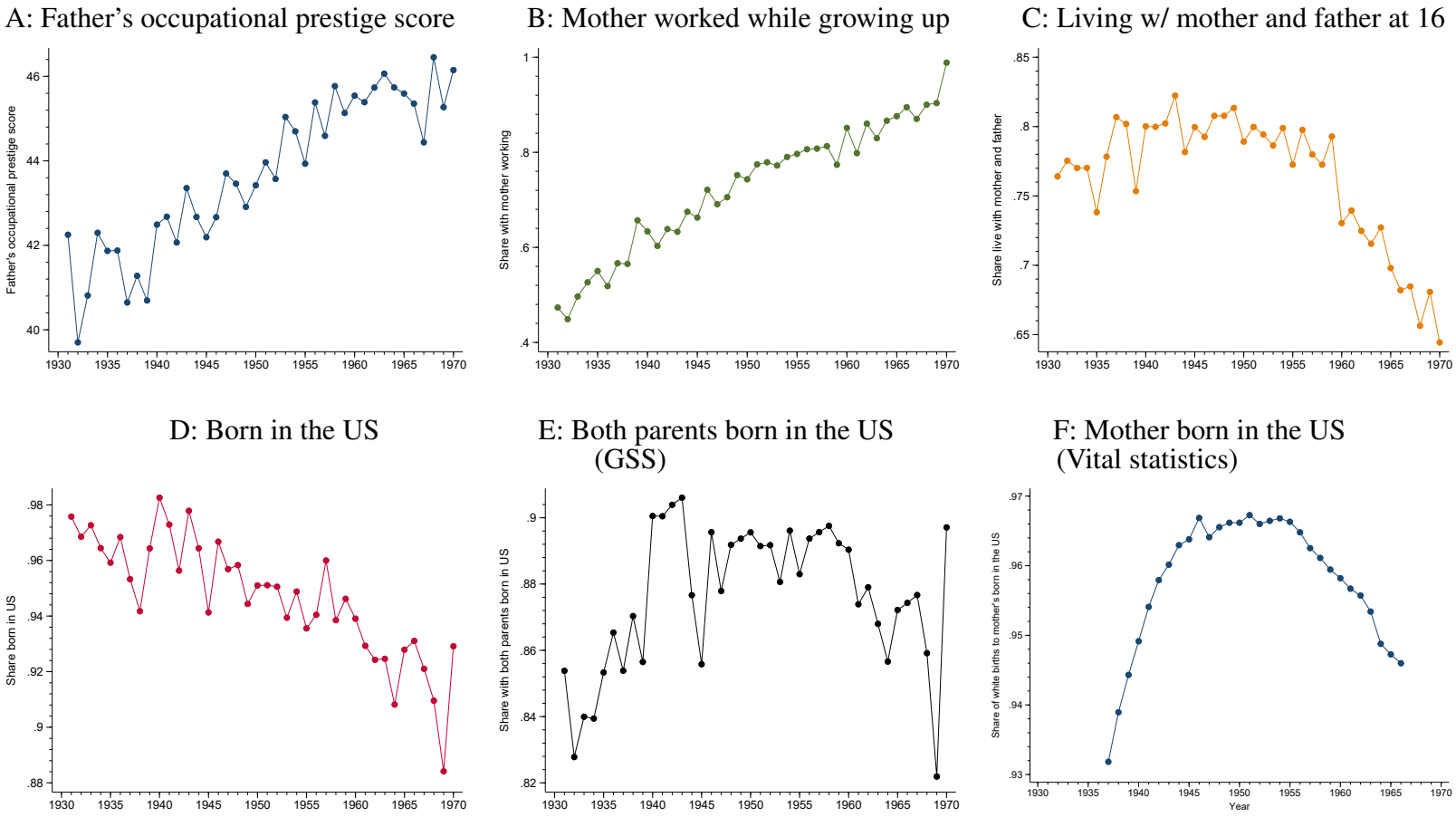


B: Share of mothers with Bachelor's degree



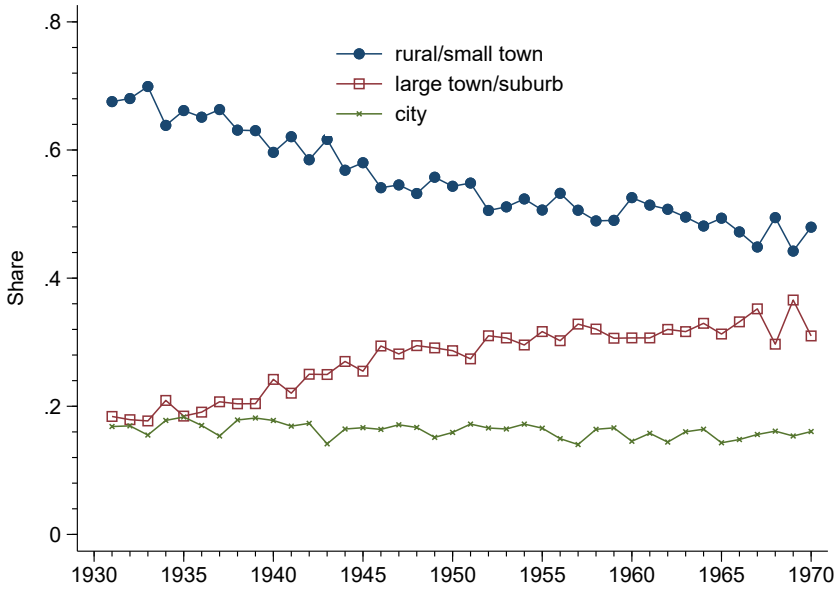
Each graph shows an estimate of parental educational attainment by individuals' birth cohort, estimated from the 1972-2016 waves of the General Social Survey. Each outcome is age-adjusted, by running a regression with cohort fixed effects and a quartic-in-age. The plots then show the estimated cohort effects, plus the estimated age effect for age 35. All regressions use sampling weights.

Appendix Figure 12: Family background and childhood circumstances of White Americans by birth cohort



Panels A-E shows an estimate of the average value of the listed variable for white Americans by year of birth, estimated from the General Social Survey. Father's occupational prestige score is based on 1980 occupational classifications and is only available in 1988-2010, so Panel A is based on those years. The question on whether an individual's mother was working is only available 1994-2016, so panel B is based on only those years. Panel C is based on 1972-2016. Panel D and Panel E are based on 1977-2016. Each outcome is age-adjusted, by running a regression with cohort fixed effects and a quartic-in-age. The plots then show the estimated cohort effects, plus the estimated age effect for age 35. All regressions use sampling weights. Panel F reports estimates directly from vital statistics volumes which report parent's nativity.

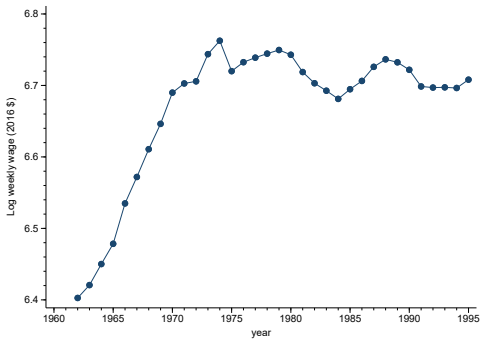
Appendix Figure 13: Where white Americans lived at age 16, by birth cohort



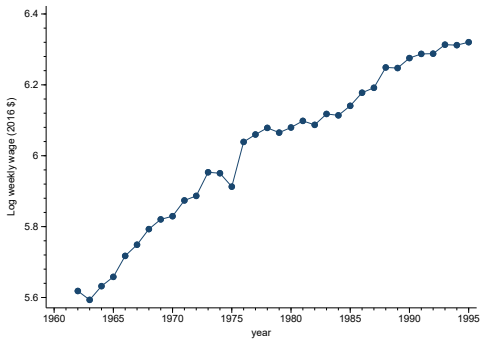
The figure shows estimates from the General Social Survey of the share of white Americans who lived in the listed type of place at age 16 by year of birth. ”City” refers to large cities over 250,000 people. ”Suburb” refers to a suburb near a large city. “Large town” refers to a city/town of 50,000 to 250,000. “rural/small town” includes smaller towns and rural areas. Each outcome is age-adjusted, by running a regression with cohort fixed effects and a quartic-in-age. The plots then show the estimated cohort effects, plus the estimated age effect for age 35. All regressions use sampling weights.

Appendix Figure 14: Wage trends, United States

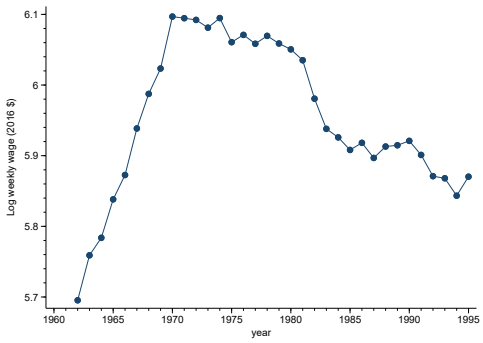
Mean ln weekly wage, FTFY men



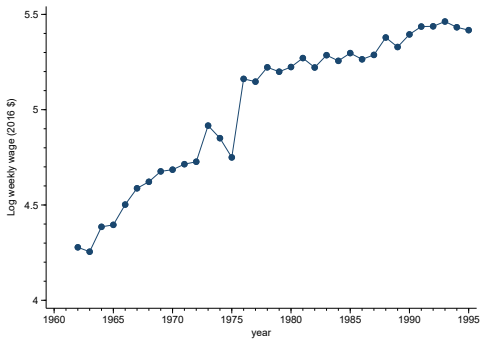
Mean ln weekly wage, FTFY women



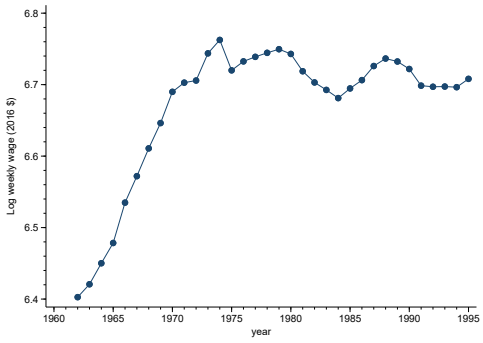
10th percentile ln weekly wage, FTFY men



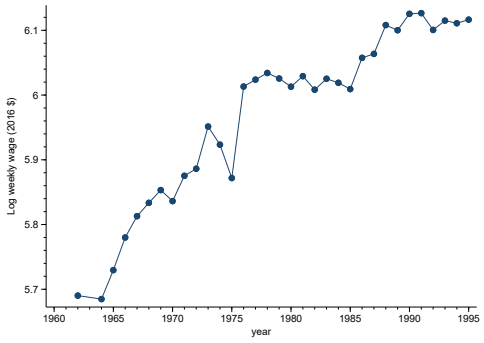
10th percentile ln weekly wage, FTFY women



High school, mean ln weekly wage, FTFY men

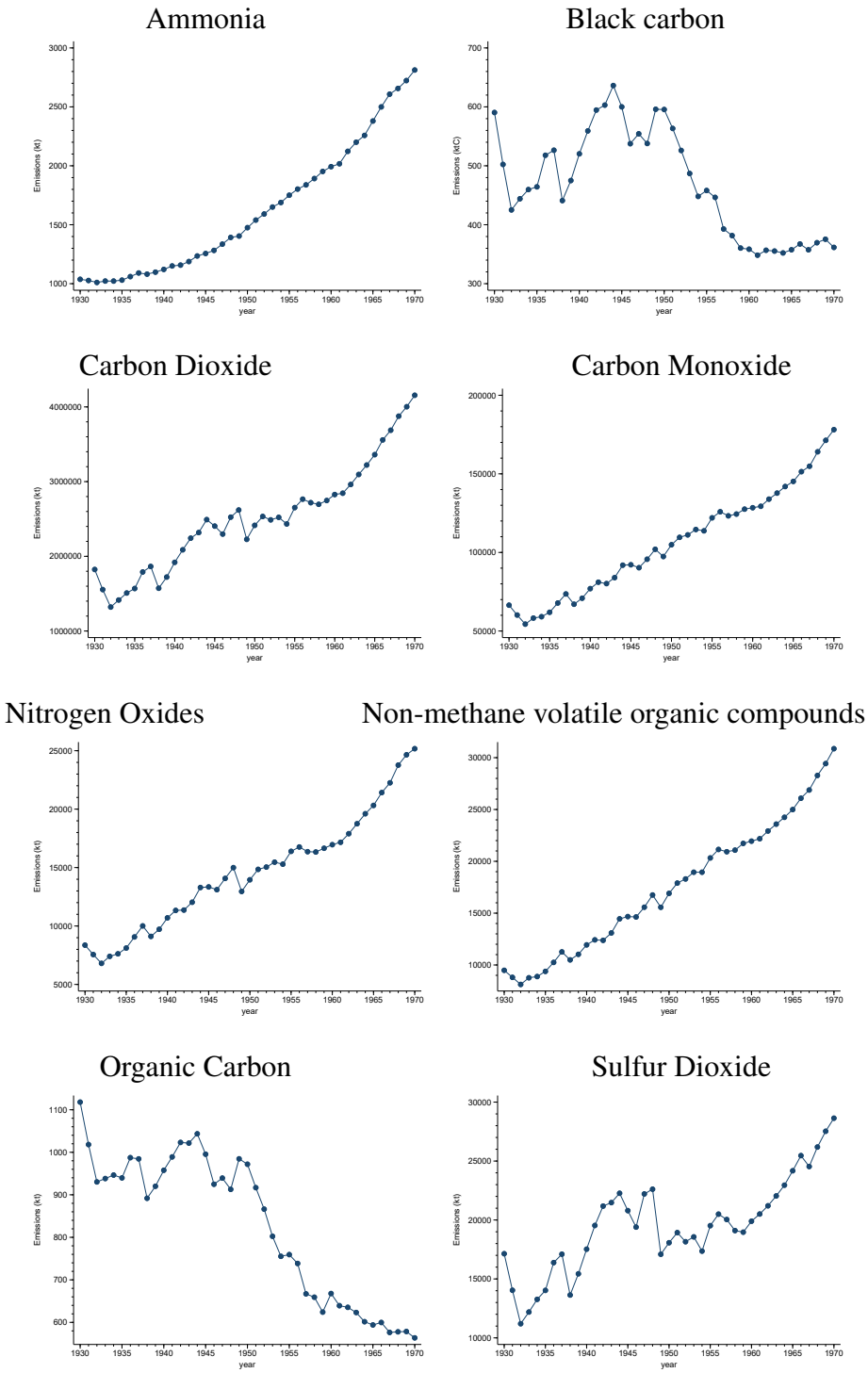


High school, mean ln weekly wage, FTFY women



Each panel shows the listed wage series estimated from the March Annual Social and Economic Supplement of the Current Population Survey (CPS).

Appendix Figure 15: Air pollution trends, United States



Each panel shows estimates from the Community Emissions Data System (O'Rourke et al.) of the trend in emissions of the listed air pollutant in the United States.

Appendix Table 1: Root mean squared error of year-specific Gompertz models of log mortality,
white men and women 1959-2014

	Within sample	Out of sample	
	ages 30-75	ages 20-29	ages 1-19
White men	.092	.703	.914
White women	.057	0.432	1.164

This table reports the root mean squared error of simple Gompertz regression models of log mortality for white men and women betweeen 1959 and 2019. Weighted least squares regressions of log mortality with a linear age term and a constant are estimated separately for each year, on observed log moratlity rates ages 30 to 75. The within sample column reports the within sample root mean squared residual of these models pooled across all years (separately for white men and white women). The out of sample columns report the root mean squared error when these models are extrapolated out of sample to younger ages, ages 20-29 and ages 1-19 separately.

Appendix Table 2: Shared cohort-specific trend break, log mortality of white Americans By Hispanic origin, 1997-2019

	(1) All whites	(2) Non-Hispanic whites	(3) Hispanic whites
Panel A: White women			
Average size of break	0.0165	0.0185	0.0149
	(0.0004)	(0.0004)	(0.0021)
Location of break	1948	1949	1939
	[1948, 1949]	[1948, 1949]	[1938, 1945]
P-value for existence of break	$< .001$	$< .001$	$< .001$
Panel B: White men			
Average size of break	0.0112	0.0131	-0.0043
	(0.0006)	(0.0006)	(0.0008)
Location of break	1946	1946	1958
	[1944, 1946]	[1946, 1946]	[1957, 1960]
P-value for existence of break	$< .001$	$< .001$	$< .001$
Linear age	Yes	Yes	Yes
Year FEs	Yes	Yes	Yes
Linear-age-by-year	Yes	Yes	Yes
Quadratic-age-by-year	No	No	No
Cubic-age-by-year	No	No	No

Each column shows the results of estimation of a model based on equation 4, with the log mortality rate for single age-by-year bins as the dependent variable. The columns respectively show results for the mortality rate of i) all whites, regardless of Hispanic origin; ii) non-Hispanic whites; and iii) Hispanic whites. All models are estimated by weighted least squares, following the approach outlined in Hansen (2000). The sample includes the years 1997-2019, ages 30-75, and cohorts born from 1930-1970. The row titled "Average size of break" reports the average value of $\delta_{2,c}$ across all years, with the standard error in parentheses calculated by the delta method. The row titled "Location of break" reports the estimated cohort at which a trend break occurs, with a 99 % confidence interval in brackets calculated by inverting the likelihood ratio statistic. The row titled "P-value for existence of break" reports p-value from an F-type test for the null hypothesis that no trend break occurs, based on 1000 bootstrap samples.

Appendix Table 3: Shared cohort-specific trend break, log mortality of white Americans By Census Region, 1980-2014

	(1) Northeast	(2) Midwest	(3) South	(4) West
Panel A: White women				
Average size of break	0.024	0.021	0.020	0.018
	(0.001)	(0.001)	(0.001)	(0.001)
Location of break	1949	1950	1948	1946
	[1949, 1949]	[1950, 1950]	[1947, 1948]	[1946, 1946]
P-value for existence of break	$< .001$	$< .001$	$< .001$	$< .001$
Panel B: White men				
Average size of break	0.026	0.029	0.026	0.026
	(0.001)	(0.001)	(0.001)	(0.001)
Location of break	1944	1946	1946	1942
	[1944, 1944]	[1946, 1946]	[1946, 1947]	[1942, 1943]
P-value for existence of break	$< .001$	$< .001$	$< .001$	$< .001$
Linear age	Yes	Yes	Yes	Yes
Year FEs	Yes	Yes	Yes	Yes
Linear-age-by-year	Yes	Yes	Yes	Yes
Quadratic-age-by-year	No	No	No	No
Cubic-age-by-year	No	No	No	No

Each column shows the results of estimation of a model based on equation 4, with the log mortality rate of white men or women — in the listed Census Region — for single age-by-year bins as the dependent variable. All models are estimated by weighted least squares, following the approach outlined in Hansen (2000). The sample includes the years 1980-2014, ages 30-75, and cohorts born from 1930-1970. The row titled "Average size of break" reports the average value of $\delta_{2,c}$ across all years, with the standard error in parentheses calculated by the delta method. The row titled "Location of break" reports the estimated cohort at which a trend break occurs, with a 99 $\%$ confidence interval in brackets calculated by inverting the likelihood ratio statistic. The row titled "P-value for existence of break" reports p-value from an F-type test for the null hypothesis that no trend break occurs, based on 1000 bootstrap samples.

Appendix Table 4: Trend break estimates, Educational attainment of white Americans by birth cohort

		First break		Second break		
	Pre-trend	Location	Size	Location	Size	P-value
Years of schooling, men	0.0797	1947	-0.1110	1961	0.0743	$< .001$
	(.0019)	[1947, 1948]	(.0036)	[1958, 1963]	(.0059)	
Years of schooling, women	0.0816	1949	-0.0860	1963	0.0703	$< .001$
	(.0011)	[1948, 1949]	(.0023)	[1961, 1965]	(.0049)	
High school degree (or GED), men	0.0080	1948	-0.0090	1962	0.0031	$< .001$
	(.0001)	[1948, 1948]	(.0002)	[1959, 1965]	(.0004)	
High school degree (or GED), women	0.0080	1948	-0.0082	1964	0.0023	$< .001$
	(.0001)	[1947, 1948]	(.0002)	[1935, 1965]	(.0004)	
Some college, men	0.0122	1949	-0.0224	1960	0.0199	$< .001$
	(.0004)	[1948, 1949]	(.001)	[1958, 1963]	(.0015)	
Some college, women	0.0152	1949	-0.0147	1963	0.0121	$< .001$
	(.0003)	[1948, 1950]	(.0006)	[1959, 1965]	(.0013)	
Bachelor's degree, men	0.0079	1947	-0.0141	1960	0.0133	$< .001$
	(.0004)	[1946, 1948]	(.0008)	[1957, 1964]	(.0012)	
Bachelor's degree, women	0.0095	1950	-0.0108	1962	0.0137	$< .001$
	(.0002)	[1949, 1951]	(.0006)	[1959, 1965]	(.0011)	
Advanced degree, men	0.0041	1945	-0.0088	1960	0.0056	$< .001$
	(.0002)	[1945, 1946]	(.0003)	[1957, 1963]	(.0004)	
Advanced degree, women	0.0048	1948	-0.0076	1964	0.0101	$< .001$
	(.0002)	[1947, 1949]	(.0003)	[1961, 1965]	(.0008)	

Each row shows the estimation results of a separate trend break model which allow for two possible trend breaks of unknown location, with the listed dependent variable. All models are estimated using the sequential estimation approach suggested in Hansen (2000) for such models. The two columns titled "Location" reported the estimated location of the first and second trend breaks, respectively, with 99 % confidence intervals in brackets calculated by inverting the likelihood ratio statistic. The two columns titled "Size" report the magnitude of first and second trend breaks respectively, with standard errors in parentheses. The column titled "Pre-trend" reports the estimated trend prior to the first break. The column titled "P-value" reports the value of a bootstrap-based F-test suggested in Hansen (2000), for the null of a model with one break versus the alternative of a model with two breaks. I also conduct a similar test for the null of no break vs. the null of one break, which yields P-values $< .05$ for all variables.

I pool data from the CPS MORG data 1990 to 2018, white individuals age 25 to 75, cohort is defined as age - year -1. I then calculate approximate average years of schooling for each cohort based on the 16 schooling categories, and estimate the trend break models for average years of schooling by birth cohort.

Appendix Table 5: Trend break estimates, Birth order of white Americans by birth cohort

		First break		Second break		
	Pre-trend	Location	Size	Location	Size	P-value
Share 2nd born or later	-0.0095	1941	0.0176	1960	-0.0219	$< .001$
	(.0013)	[1937, 1943]	(.0017)	[1958, 1962]	(.0017)	
Share 3rd born or later	-0.0083	1946	0.0185	1963	-0.0368	$< .001$
	(.0008)	[1942, 1947]	(.0013)	[1961, 1964]	(.0025)	
Share 4th born or later	-0.0086	1947	0.0156	1964	-0.0288	$< .001$
	(.0004)	[1946, 1948]	(.0007)	[1962, 1965]	(.0017)	
Share 5th born or later	-0.0068	1947	0.0118	1964	-0.0210	$< .001$
	(.0003)	[1946, 1948]	(.0006)	[1962, 1965]	(.0013)	
Simulated effect on ln(mort), men	-0.0044	1947	0.0073	1964	-0.0129	$< .001$
	(.0002)	[1946, 1948]	(.0004)	[1962, 1965]	(.0009)	
Simulated effect on ln(mort), men	-0.0061	1947	0.0107	1964	-0.0201	$< .001$
	(.0003)	[1946, 1948]	(.0006)	[1962, 1965]	(.0013)	

Each row shows the estimation results of a separate trend break model which allow for two possible trend breaks of unknown location, with the listed dependent variable. All models are estimated using the sequential estimation approach suggested in Hansen (2000) for such models. The two columns titled "Location" reported the estimated location of the first and second trend breaks, respectively, with 99 % confidence intervals in brackets calculated by inverting the likelihood ratio statistic. The two columns titled "Size" report the magnitude of first and second trend breaks respectively, with standard errors in parentheses. The column titled "Pre-trend" reports the estimated trend prior to the first break. The column titled "P-value" reports the value of a bootstrap-based F-test suggested in Hansen (2000), for the null of a model with one break versus the alternative of a model with two breaks. I also conduct a similar test for the null of no break vs. the null of one break, which yields P-values $< .05$ for all variables.

Observed birth order shares for white Americans comes from Vital Statistics volumes and Heuser (1976). The simulated effect of birth order on log mortality is derived from within-family estimates of the effect of birth order on mortality from Barclay and Kolk (2015) and observed birth order shares for white Americans. I then estimate the described trend break models by birth cohort on the simulated data.

Appendix Table 6: Trend break estimates, Cohort size

		First break		Second break		
	Pre-trend	Location	Size	Location	Size	P-value
Cohort size at age 18 (mil.)	-0.026	1936	0.139	1957	-0.167	$< .001$
	(.016)	[1935, 1939]	(.018)	[1956, 1959]	(.009)	
Cohort size at birth (mil.)	0.091	1958	-0.169			$< .001$
	(.004)	[1956, 1960]	(.011)			

Each row shows the estimation results of a separate trend break model which allow for two possible trend breaks of unknown location, with the listed dependent variable. All models are estimated using the sequential estimation approach suggested in Hansen (2000) for such models. The two columns titled "Location" reported the estimated location of the first and second trend breaks, respectively, with 99 $\%$ confidence intervals in brackets calculated by inverting the likelihood ratio statistic. The two columns titled "Size" report the magnitude of first and second trend breaks respectively, with standard errors in parentheses. The column titled "Pre-trend" reports the estimated trend prior to the first break. I conduct bootstrap-based F-tests suggested in Hansen (2000), for i) the null of a model with one break versus the alternative of a model with two breaks, and ii) for the null of no break vs. the null of one break. When the p-value for i) is $< .05$ then I report results from the model with two breaks, and the column titled "P-value" reports the p-value from i). When the p-value for i) is $\geq .05$, I report results from the model with one break, and the p-value is that from test ii).

All data is from the Human Mortality Database.

Appendix Table 7: Trend break estimates, Smoking of American women of childbearing age, by year

	Pre-trend	Location	Size	P-value
Smoking prev., women 18-35	1.216	1955	-1.401	$< .001$
(percent)	(.059)	[1945, 1955]	(.145)	

This table shows the estimation results of a trend break model which allow for two possible trend breaks of unknown location, with the listed dependent variable. It is estimated using the sequential estimation approach suggested in Hansen (2000) for such models. I conduct bootstrap-based F-tests suggested in Hansen (2000), for i) the null of a model with one break versus the alternative of a model with two breaks, and ii) for the null of no break vs. the null of one break. The p-value for i) is $\geq .05$, so I report results from the model with one break, and the value in the column labelled "P-value" is the p-value from test ii). The column titled "Location" reported the estimated location of the trend break, with 99 $\%$ confidence intervals in brackets calculated by inverting the likelihood ratio statistic. The column titled "Size" report the magnitude of second trend breaks respectively, with standard errors in parentheses. The column titled "Pre-trend" reports the estimated trend prior to the first break.

Based on estimates of smoking prevalence by age and year (every 5 years) from Holford et al. (2014), derived from survey data on retrospective smoking history. I fit the trend break models to prevalence estimates for women ages 18-35.

Appendix Table 8: Trend break estimates, Family background and childhood circumstances of white Americans, by cohort

		First break		Second break		
	Pre-trend	Location	Size	Location	Size	P-value
Share whose father has BA	-0.002	1936	0.008			$< .001$
	(.002)	[1935, 1951]	(.002)			
Share whose mother has BA	0.003	1948	0.002			0.001
	(.0003)	[1935, 1965]	(.0006)			
Share whose father has HS diploma	0.005	1935	0.014	1955	-0.011	0.029
	(.004)	[1935, 1965]	(.004)	[1948, 1961]	(.001)	
Share whose mother has HS diploma	0.017	1952	-0.009			$< .001$
	(.0006)	[1945, 1959]	(.001)			
Father's occupational prestige	0.154	1962	-0.151			0.103
	(.012)	[1935, 1965]	(.068)			
Mother worked while child growing up	0.010	1965	-0.011			0.073
	(.0004)	[1935, 1965]	(.004)			
Living w/ mother and father at 16	0.001	1954	-0.011			$< .001$
	(.0004)	[1949, 1958]	(.0009)			
Born in the US	-0.001	1957	-0.002			0.022
	(.0002)	[1935, 1965]	(.0007)			
Both parents born in US	0.007	1942	-0.008			$< .001$
	(.0008)	[1938, 1951]	(.001)			
Mother born in the US	0.004	1944	-0.004	1956	-0.002	$< .001$
(Vital statistics)	(0.0002)	[1943, 1944]	(0.0002)	[1953, 1961]	(0.0002)	
Lived in rural/small town when 16	-0.008	1948	0.005			$< .001$
	(.0007)	[1942, 1959]	(.001)			
Lived in big town/suburb when 16	0.007	1949	-0.005			$< .001$
	(.0005)	[1944, 1958]	(.0009)			
Lived in city when 16	-0.002	1965	0.002			0.681
	(.0002)	[1935, 1965]	(.002)			

Each row shows the estimation results of a separate trend break model which allow for two possible trend breaks of unknown location, with the listed dependent variable. All models are estimated using the sequential estimation approach suggested in Hansen (2000) for such models. The two columns titled "Location" reported the estimated location of the first and second trend breaks, respectively, with 99 % confidence intervals in brackets calculated by inverting the likelihood ratio statistic. The two columns titled "Size" report the magnitude of first and second trend breaks respectively, with standard errors in parentheses. The column titled "Pre-trend" reports the estimated trend prior to the first break. I conduct bootstrap-based F-tests suggested in Hansen (2000), for i) the null of a model with one break versus the alternative of a model with two breaks, and ii) for the null of no break vs. the null of one break. When the p-value for i) is $< .05$ then I report results from the model with two breaks, and the column titled "P-value" reports the p-value from i). When the p-value for i) is $\geq .05$, I report results from the model with one break, and the p-value is that from test ii).

With one exception, all data come from various waves of the General Social Survey. shows an estimate of the average value of the listed variable for white Americans by year of birth, estimated from the General Social Survey. I first age-adjusted each outcome, by running a regression with cohort fixed effects and a quartic-in-age. I then run the trend break models on these age-adjusted series by cohort (which are the estimated cohort effects, plus the estimated age effect for age 35). See the notes to the corresponding Appendix Figures for more detail on exact GSS waves for each variable. The data for "Mother born in the US" come from vital statistics volumes which report the number of births in a year by parent's nativity.

Appendix Table 9: Trend break estimates, Wages in the United States, by year

	Pre-trend	Location	Size	P-value
Mean ln wage, men	0.035	1972	-0.037	$< .001$
	(.001)	[1971, 1973]	(.002)	
10th pct ln wage, men	0.047	1971	-0.058	$< .001$
	(.002)	[1970, 1971]	(.0024)	
Mean ln wage, women	0.030	1976	-0.013	$< .001$
	(.001)	[1971, 1979]	(.002)	
10th pct ln wage, women	0.056	1979	-0.040	$< .001$
	(.002)	[1977, 1982]	(.004)	
Mean ln wage, HS-only men	0.035	1972	-0.037	$< .001$
	(.001)	[1971, 1973]	(.002)	
Mean ln wage, HS-only women	0.021	1977	-0.014	$< .001$
	(.001)	[1972, 1980]	(.002)	

Each row shows the estimation results of a separate trend break model which allow for two possible trend breaks of unknown location, with the listed dependent variable. All models are estimated using the sequential estimation approach suggested in Hansen (2000) for such models. I conduct bootstrap-based F-tests suggested in Hansen (2000), for i) the null of a model with one break versus the alternative of a model with two breaks, and ii) for the null of no break vs. the null of one break. For all dependent variables, the p-value for i) is $\geq .05$, so I report results from the model with one break, and the value in the column labelled "P-value" is the p-value from test ii). The column titled "Location" report the estimated location of the trend break, with 99 $\%$ confidence intervals in brackets calculated by inverting the likelihood ratio statistic. The column titled "Size" report the magnitude of second trend breaks respectively, with standard errors in parentheses. The column titled "Pre-trend" reports the estimated trend prior to the first break.

I calculate each wage series from the March Annual Social and Economic Supplement of the Current Population Survey (CPS). I then fit the trend break models to the annual time series.

Appendix Table 10: Trend break estimates, Emissions in the United States by year

		First break		Second break		
	Pre-trend	Location	Size	Location	Size	P-value
Black carbon (kt)	6.21	1949	-27.4	1961	26.018	0.001
	(1.28)	[1944, 1951]	(2.87)	[1935, 1965]	(4.76)	
Carbon monoxide (kt)	593	1935	2,130	1965	3,815	0.005
	(503)	[1935, 1940]	(532)	[1963, 1965]	(532)	
Carbon dioxide (kt)	64,555	1945	-34,990	1961	119,7063	0.030
	(6089)	[1936, 1965]	(10024)	[1957, 1964]	(15223)	
Ammonia (kt)	14.0	1944	36.8	1962	41.4	$< .001$
	(1.01)	[1942, 1945]	(1.53)	[1960, 1964]	(2.52)	
Non-methane volatile	114	1935	403	1965	655	$< .001$
organic compounds (kt)	(73.5)	[1935, 1940]	(77.8)	[1963, 1965]	(77.8)	
Nitrogen oxides (kt)	417	1962	702	1947	-154	0.005
	(21.3)	[1960, 1964]	(71.0)	[1938, 1958]	(39.1)	
Organic carbon (kt)	-28.3	1943	-31.6	1935	40.9	0.025
	(8.99)	[1941, 1947]	(4.48)	[1935, 1965]	(11.8)	
Sulfur dioxide (kt)	628	1959	949	1943	-720	$< .001$
	(87.1)	[1952, 1963]	(152)	[1939, 1948]	(133)	

Each row shows the estimation results of a separate trend break model which allow for two possible trend breaks of unknown location, with the listed dependent variable. All models are estimated using the sequential estimation approach suggested in Hansen (2000) for such models. The two columns titled "Location" reported the estimated location of the first and second trend breaks, respectively, with 99 $\%$ confidence intervals in brackets calculated by inverting the likelihood ratio statistic. The two columns titled "Size" report the magnitude of first and second trend breaks respectively, with standard errors in parentheses. The column titled "P-value" reports the value of a bootstrap-based F-test suggested in Hansen (2000), for the null of a model with one break versus the alternative of a model with two breaks. I also conduct a similar test for the null of no break vs. the null of one break, which yields P-values $< .05$ for all variables.

All data is an estimate of the time series in emissions of the listed air pollutant in the United States, from the Community Emissions Data System (O'Rourke et al.).