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

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Abstract

In the 1970s, American educational attainment and test scores declined sharply, and steady growth in real wages halted. In the 1980s, the incidence of low birthweight births suddenly reversed trend and began increasing. In 1999, the mortality rate of white Americans at midlife also reversed trend and began to rise. I present evidence that *all* of these patterns are linked to a decline in health and human capital across American-born cohorts, that began suddenly with those born after 1947. This cross-cohort decline is evident from the estimation of standard age-period-cohort models of: earnings, maternal health as measured by the birth weight of infants, and the mortality rates of men and women. I also implement a novel methodology in which the decline is evident in each outcome as a sharp discontinuity and is confirmed by structural break testing. There is no decline for the foreign-born population, but it is otherwise remarkably widespread across race and geography in the United States. The decline in educational attainment for these cohorts appears too small to directly explain all of the other declines. I present suggestive evidence that the root cause may have been a worsening respiratory health environment when these cohorts were infants.

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1. Introduction

At distinct points over the last sixty years, sudden episodes of decline in the United States have sparked public concern and research literatures. In the late-1960s and 1970s, SAT scores, high school graduation rates, and college enrollment rates declined sharply, prompting questions about the failure of the educational system (National Commission on Excellence in Education, 1983). In the 1970s, growth in men's real earnings also suddenly slowed and has been stagnant since that period, with earnings for those without a college degree declining substantially (Gould, 2014; Acemoglu and Autor, 2011). In the 1980s, the share of infants born at low birthweight began to increase (Centers for Disease Control, 1994). In 1999, the mortality rate of white Americans at midlife began increasing, driven particularly by suicides and drug overdoses (Case and Deaton, 2015).

These episodes of decline have generally been analyzed in isolation, with an emphasis on contemporaneous factors as causes. For example, educational declines have been attributed partially to a combination of cohort size and fixed supply in the educational system (Card and Lemieux, 2001b), mortality increases to the opioid epidemic (Case and Deaton, 2015), and declining wages for non-college-educated men to changes in relative supply and demand (Katz and Murphy, 1992; Card and Lemieux, 2001a; Acemoglu and Autor, 2012). All these explanations are period-based: otherwise normal individuals are hit by bad conditions at a particular life juncture and their outcomes suddenly suffer. Case and Deaton (2017) suggest a fundamentally different, cohort-based explanation for the rise in mortality in particular. They suggest that cohorts born after World War II have been disadvantaged since they entered the labor market more than 50 years ago, and that the recent increase in mortality at midlife is merely a manifestation of this longer-standing disadvantage.

In this paper I go a step further by presenting evidence consistent with the theory that *all* of the above episodes of decline are linked to a single decline in health and human capital for the *same* set of cohorts, which predated labor market entry. The trend of health and human capital across American-born cohorts changed abruptly at the 1947 cohort. The health and human capital of each successive cohort born after 1947 was worse than that of the cohort born a year earlier, relative to trend. The depressed human capital led to declining educational outcomes in the 1960s and 1970s when these cohorts were in their teens and early-20s. The depressed labor market ability of men in these cohorts then led them to have lower earnings, contributing to earnings stagnation since the 1970s. Depressed health as mothers led the women in these cohorts to give birth to less healthy infants, driving the upturn in the low birthweight rate in the 1980s. Ultimately, the poor health of these cohorts has increased their likelihood of untimely death, contributing to recent mortality increases of whites at midlife.

My estimates imply that absent this cross-cohort health and human capital decline: the rise in low birthweight births in the 1980s would not have occurred; there would have been very modest real earnings growth between 1979 and 1993, rather than real declines; and midlife mortality would have continued to fall steadily at its pre-1999 rate. This suggests that previously independent searches for the causes of educational declines of the 1970s, increases in the low birthweight rate in the 1980s, and increases in the midlife mortal-

ity of whites since 1999 can be unified.[1] To find the cause of all of these declines will require understanding what went deeply wrong early in life — during a time of rapid economic and technological progress — for cohorts of Americans born after 1947. This early life decline has also contributed significantly to wage stagnation over the last 50 years. Additionally, elasticities and other estimates from previous studies that assume no underlying differences across cohorts are likely to be biased by the large differences I document.

As a first piece of suggestive evidence, consider Figure 1. It shows that the age profiles of the share of men in white-collar occupations in 1980, 1990, and 2000 exhibit sharp changes in slope — at different ages in each year. However, recasting the same underlying data by year of birth reveals that the slope changes occur at precisely the same *birth cohort*, 1947, across years — with occupational status falling sharply for those born after 1947. Appendix Figures A1 through A4 show similarly sharp and suggestive patterns in the age profiles of men's median earnings, infant birth weight (by mother's year of birth), and the log mortality rates of white men and women. These patterns appear consistent with a decline in health and human capital for cohorts born after 1947. In contrast, an alternative explanation assuming no underlying differences across cohorts would require highly non-linear, and nonsmooth, changes in age-specic factors over time.

My primary analysis documents evidence of this health and human capital decline in maternal health, labor market outcomes, and mortality. I examine maternal health by studying the birth weight of infants by the birth cohort *of their mother* using vital statistics microdata on more than 75 million births.[2] I examine earnings of men, using CPS survey data; and the mortality rates of men and women, again using vital statistics data.

I first take the traditional approach to separating the impact of cohort factors from those which vary by age and year. I estimate additively separable age-period-cohort models, common in economics and demography.[3] The pattern of estimated cohort effects for each of the above outcomes are remarkably similar. They each have a clear piecewise-linear shape with a large slope change at or near the 1947 cohort, and declines in health and human capital for cohorts born after that year. The patterns also closely match the timing of previously documented declines in educational attainment and standardized test scores for the same cohorts.[4]

These results, while striking, could be biased by external factors that disproportionately impact individuals of a particular age in certain years, which the additively separable model rules out. On the other hand, adding unrestricted age-by-year interactions would leave the cohort effects unidentified, due to the perfect collinearity of age, period and cohort. To make progress, I allow age-by-year interactions but assume that they take the form of a polynomial in age of a known order. Intuitively, I allow the impact of age to change over time but restrict it to do so smoothly. The remaining threat to validity is nonsmooth changes in the

[1] The cohort decline is generally evident for blacks and whites. The only exception is black men's mortality, while all other outcomes exhibit evidence of a similar decline for blacks and whites.

[2] Using birth weight to proxy for maternal health follows from the idea that a mother's health "endowment" enters the infant health production function (Rosenzweig and Schultz, 1983; Grossman and Joyce, 1990).

[3] For a textbook treatment see Deaton (1997). Recent applications in economics include Aguiar and Hurst (2013) and Lagakos et al. (2018), and in demography include Yang (2008), Masters et al. (2014), Masters et al. (2017), and Zang et al. (2018).

[4] See Koretz (1987), Card and Lemieux (2001b), Goldin and Katz (2007), Heckman and LaFontaine (2010), and Acemoglu and Autor (2012). I also document a sudden, and to my knowledge previously unnoticed, decline beginning with the 1947 cohort in the share of men earning an advanced degree; as well as a similarly-timed stagnation of prior improvements for women.

impact of age across years— such as a large shock that impacts the health of individuals aged 30-and-under, but not of 31-year-olds. None of the alternative explanations put forward in the related literatures have this feature. For example, the effects of shifts in supply or demand will be smooth as long as individuals who are close in age are substitutable.

I use two novel approaches to identify the cross-cohort decline in health and human capital, while controlling for age-by-year interactions of a polynomial form. My first approach, by differencing the age profile of outcomes across neighboring years, allows the abrupt cohort decline to be identified as a sharp discontinuity. It therefore extends the idea in McKenzie (2006) of "differencing out" age effects, to derive a sharp visual test of the existence of the cohort decline similar to that found in regression discontinuity designs. Second, I estimate a model in which cohort effects are specified to have a piecewise linear shape, while controlling for a separate polynomial-in-age in each year. I follow structural break methodology (Hansen, 1999, 2000) to test whether a cohort decline exists and to estimate precisely at which cohort it began.

Large discontinuities are visually and statistically evident in the differenced age profiles of all of the above outcomes, consistent with a broad health and human capital decline. Structural break estimation yields strong statistical evidence of large changes in the slope of cohort effects occurring at or near the 1947 cohort for all outcomes. The findings are generally robust to including increasingly flexible controls for the impact of age in each year, up to including a separate quartic-in-age in each year. The size of the maternal health decline *increases* as the higher order interaction terms are added. The estimates imply that the median earnings of men in the 1960 cohort are roughly 23 percent lower than they would have been in the absence of the post-1947 change in cohort trend; and that women born in the 1960 cohort gave birth to nearly 2.8 more low birthweight infants per 100 births than they would have if their health had followed the pre-1947 cohort trend.

I present a number of additional findings which suggest that the cross-cohort declines were caused by an early-life factor — widespread within, but unique to the United States — which worsened between 1947 and at least the mid-1960s. First, the cross-cohort educational declines cannot explain the mortality increases unless the effect of education on mortality is an order of magnitude larger than past instrumental variable estimates (Galama et al., 2018). There are also large cross-cohort declines in earnings and maternal health *conditional* on educational attainment. Second, there is no evidence of a cross-cohort decline for the foreign-born population, but it is otherwise remarkably widespread across race and geography in the United States. The declines are quite similar across the four Census Regions, and are generally evident in all racial groups and in urban and rural areas.[5] Third, the cross-cohort patterns in parental education, survival to adulthood, and cohort size make the hypothesis that the declines were caused by family background, selection, or "cohort crowding" appear unlikely.

I close the paper with suggestive evidence pointing towards one such potential early-life factor as the root cause of the cross-cohort decline: a postwar deterioration in the respiratory health environment in the

[5] As mentioned above, the one exception is the mortality of black men, which shows evidence of improvements for post-1956 cohorts. Each of the other outcomes exhibit similar patterns for blacks and whites, and the earnings declines for black men are actually *larger* than those for whites.

United States. I show that mortality from particular respiratory causes began to increase simultaneously for both infants *and* elderly adults near 1946. My hypothesis is that the same environmental factor that caused these short-run mortality increases also had a lasting impact on the health of cohorts who were in utero or infancy during that period. The change in the respiratory health environment may have been caused by a decline in air quality, due to postwar industrial growth or the rapid increase in automobile use. Previous research has found evidence that early-life exposure to poor air quality has large impacts on outcomes later in life (eg. Isen et al., 2017).

The remainder of the paper is organized as follows. Section 2 describes the data and main outcomes. Section 3 presents the results from the age-period-cohort models typically used in the literature. Section 4 contains the results from the more novel approaches which impose weaker restrictions on the age-period interaction effects. Section 5 shows the sizeable impact of the cross-cohort declines on year-over-year changes in each outcome. Section 6 considers the link between the documented cross-cohort declines and educational declines for the same cohorts. Section 7 demonstrates the widespread nature of the cross-cohort decline for those born in the U.S. Section 8 presents evidence of the post-war deterioration in the respiratory health environment and discusses its plausibility as an early-life factor that could have caused the cross-cohort declines in later-life outcomes. The final section concludes.

2. Data and main outcomes

To document the cohort declines in health and human capital I use microdata from the Current Population Survey, and vital statistics data recording nearly the universe of births and deaths. To provide evidence of a postwar change in the respiratory health environment I use newly digitized historical vital statistics data.

A. Maternal health

I use detailed microdata on 50 to 100 percent of the births in the United States in each year between 1968 and 1995 to study patterns in infant health by *mother's birth cohort*. The data are known as the "natality microdata files" and are produced by the National Center for Health Statistics. They provide detailed information derived from birth certificates. They include a 50 percent sample of all births in the United States in 1968, and include progressively larger samples until 1985, after which they include the universe of births.

I consider the health of infants both as a proxy for maternal health, and also to provide evidence of an intergenerational effect of the apparent decline in cohort health. Under the usual assumption that a mother's health "endowment" enters the infant health production function (Rosenzweig and Schultz, 1983; Grossman and Joyce, 1990), a decline in the health of infants by *mother's birth cohort* may be viewed as evidence of a decline in maternal health for these cohorts. This view is also consistent with the "life-course perspective" in maternal and child health research, which emphasizes the importance of underlying difference in maternal health for the health of infants (Lu and Halfon, 2003).[6]

[6] A similar use of this data to study cohort health can be found in Almond and Chay (2006), who focus on the black-white gap

Unfortunately, mothers' exact year of birth is not recorded explicitly. I therfore calculate the approximate birth year of each mother as the infant birth year minus the mother's age. I restrict my analysis to births to mothers born between 1930 and 1970. I use the years 1968 to 1995 and births to mothers ages 18 to 40. This results in an analysis sample of more than 75 million births.

As my main infant health outcome, I study birth weight. I study birth weight as a continuous measure, and also use the commonly defined measure of low birthweight, birth weight less than 2500 grams. For all analysis I calculate these measures in cells by year, single age, and birth year using sampling weights; and then conduct regressions and other analysis on these cell means. In analysis of heterogeneity I calculate means in cells defined based on age-year-cohort crossed with additional variables such as race, education, or Census region.

B. Earnings

To document cross-cohort declines in earnings I draw on a large, commonly-used data source: the Current Population Survey, Merged Outgoing Rotation Group (CPS-MORG). The CPS-MORG has the advantage of recording point-in-time hourly earnings, and its yearly frequency aids identification of cohort effects.

I use the CPS MORG data from 1979 to 1993. These files contain information on the hourly or usual weekly earnings of a third of the individuals in each monthly CPS. Combined with information on usual hours worked this allows for the construction of point-in-time hourly earnings. These hourly earnings measures have been used extensively in studies of wage inequality and trends, and have some notable advantages over other sources.[7] Pooling across all months in a year yields a sample three times the size of a monthly CPS — such as the March CPS. Further, Lemieux (2006) and Autor et al. (2005) suggest that the point-in-time nature of the earnings question reduces measurement error.[8]

I focus primarily on men to avoid the need to address large confounding changes in women's labor market participation over this period (Goldin, 2006). I focus on prime-age men age 25 to 54.

Exact year of birth is again not recorded explicitly. I therefore calculate birth cohort as the survey year minus reported age. That is, $c \equiv p - a$, where c denotes birth cohort, p denotes the year (or "period") of the survey, and a denotes age. I restrict my analysis to cohorts born between 1930 and 1965.

I end the analysis in 1993 for two reasons. First, there was a substantial redesign of the CPS earnings questions in 1994, and for the first 21 months after the redesign individuals with allocated earnings cannot be identified in the data (see Autor et al. (2005) for a further description). Second, this restriction ensures that each year includes a number of cohorts who were born before 1947, allowing for detection of the cohort trend break. These restrictions lead to a sample of 970,479 men with non-missing earnings used in the main analysis.

in health by cohort.

[7] See for example DiNardo et al. (1996); Lemieux (2006); Autor et al. (2008); Acemoglu and Autor (2011); and Gould (2014).

[8] I obtained the data from ceprdata.org and use their earnings definition, which is designed to follow NBER's recommendation and similar to past research.

I address a second concern of the CPS-MORG data, topcoding of earnings above a threshold which changes across years, by focusing on the median and other quantiles. I calculate the sample median and other quantiles separately for age-year-sex cells, using the provided survey weights. Due to their order-statistics properties, these estimated quantiles are unaffected by the topcoding — as long as the earnings value of the particular quantile is below the topcoding value. For regressions and other model-based analysis, I use a two step procedure: first estimating cell quantiles, and second estimating models on the cell quantiles. This approach follows Chamberlain (1994) and Chetverikov et al. (2016), and for sufficiently large cells has advantages over traditional LAD quantile regression.[9] In analysis of heterogeneity I use a similar procedure, calculating quantiles by cells defined based on age-sex-year crossed with additional variables such as race, education, or Census region.[10]

My main outcome variable is therefore median real hourly earnings by single age and year. Earnings are adjusted for inflation using the CPI-U-RS, and reported in 2014 dollars.

In some supplementary analysis I use decennial Census data to examine occupational status, focusing in particular on differences those born in versus outside of the United States. My measure of occupational standing is the share of each cohort employed in a broad category of white-collar occupations. This category corresponds to the "managers/professionals/technicians/finance/public safety" category in Autor and Dorn (2013), and I use the occupational crosswalk from these authors to obtain consistent occupational categories. I combine six percent samples from the Integrated Public Use Microdata Samples from the Decennial Censuses of 1970, 1980, 1990, and 2000 [11]. I calculate the share of employed men in each age-year cell employed in white-collar occupations.

C. Mortality

My main mortality analysis uses data from the Human Mortality Database (HMD) on number of deaths and population counts by year and cohort. These data are derived from official United States vital statistics and Census estimates, and are adjusted for errors using a uniform method. I use the cohort life tables which provide an estimate of mortality by year and cohort — rather than year and "age at last birthday." I then define age as year minus cohort. Therefore, the definition of cohort is slightly different for this data than for the other outcomes. I restrict my analysis to the years 1975-2015, ages 25 to 85, and cohorts born between 1930 and 1965.

To analyze mortality by Census Region I use data from the United States Mortality Database (USMDB). The USMDB uses similar procedures to the HMD to create subnational mortality estimates for the United States. These data are only available as period-based life-tables, and I therefore define cohort as the year minus age.

[9] For example it is unbiased in the presence of left hand side measurement error, unlike traditional quantile regression, see Hausman et al. (2019).

[10] There are actually two topcoding values in the CPS MORG. One is quite rare. I include all cells for which less than 1 percent of cell is topcoded.

[11] I obtain all data from IPUMS-USA (Ruggles et al., 2015). For 1980 and 1990, I combine the "5 percent state" sample with the "1 percent metro" sample. For 2000, I combine the 5 percent and 1 percent samples.

To calculate mortality separately by race 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 the number of deaths and the mid-year population by single age, sex, race cells. I then calculate crude death rates — number of deaths over mid-year population — within each cell. I define birth cohort as $year - age$.

D. Historical mortality from respiratory causes

I digitize data on historical mortality by *cause of death* to provide evidence of a postwar decline in respiratory health in the United States. The data was digitized from tables in historical vital statistics books for 1933-1958. It consists of death counts for cause of death categories based on International Classification of Disease codes by race, sex, and 5-year age bins. I calculate analogous death counts from the Multiple Cause of Death File described above for 1959-1980. I combine these with population estimates from the Census Bureau to form cause-specific mortality rates by age, race, sex, and 5-year age bins for 1933-1980.

3. Evidence of cohort decline from traditional age-period-cohort models

I first present evidence of cohort declines in health and human capital using the traditional approach to separate the impact of cohort factors from those which vary by age and year. I estimate additively separable age-period-cohort models, common in economics and demography.[12] The estimated cohort effects for each of the outcomes described above exhibit remarkably similar patterns. These patterns are consistent with a large decline in cohort health and human capital beginning with the 1947 cohort which has impacted the health of mothers and their infants, men's earnings, and the mortality rate of men and women.

I estimate models of the following form:

$$Y_{apc} = \gamma_c + \phi_p + \theta_a + \epsilon_{apc} \tag{1}$$

where Y_{apc} denotes an outcome — such as earnings — for individuals who are age a, in the year or "period" p, and who are members of the cohort c, ie. they were born in year c. The main object of interest are the cohort effects, represented by the sequence of fixed effects γ_c. I also control for separate additive fixed effects for each year, ϕ_p, and each age, θ_a.

Conceptually, the cohort effects reflect the impact of underlying, fixed differences between individuals born in different years. Individuals from different cohorts enter the labor market, give birth, and reach old age in different years. They are therefore exposed to different external year-specific factors which may impact their earnings and health outcomes. In the context of the model, the difference between the cohort effect for the 1947 cohort, γ_{1947}, and that of the 1960 cohort, γ_{1960}, reveals how the outcomes of individuals born in these two years would have differed — holding these external factors fixed. Large differences in

[12] For a textbook treatment see Deaton (1997). Recent applications in economics include Aguiar and Hurst (2013) and Lagakos et al. (2018), and in demography include Yang (2008), Masters et al. (2014), Masters et al. (2017), and Zang et al. (2018).

cohort effects therefore imply large differences in the latent health or human capital of different cohorts, not just that the cohorts were unlucky to experience poor labor market or health conditions.

To identify the cohort effects, the above model assumes that the impact of external factors can be decomposed as additively separable year and age components. It is therefore unrestrictive with respect to the dynamics of year-over-year changes impacting each outcome, and with respect to the *shape* of age effects. However, it does not allow age-by-year interactions of any kind. Year-specific factors are assumed to impact all ages equally. Visually, year effects can be thought of as shifting the entire "age profile" of outcomes evenly. I will relax this assumption in later sections, and allow for changing external factors which impact individuals of different ages differently.

Even this additively-separable model is not identified, due to the exact collinearity of age, period, and cohort (see eg. Hall, 1968; Deaton, 1997). Identification can be achieved by imposing one additional linear restriction, such as restricting two ages to have the same effect. Instead of imposing this or a similarly ad hoc restriction, I follow Fosse and Winship (2017) and Chauvel (2011) and focus on identifying reparametrized coefficients of the model in equation 2.

In particular, I normalize the first and last cohort effect to be 0. These reparameterized cohort effects then reflect the true cohort effect, minus some unknown long-run trend in cohort effects. That is, estimation will yield the following "detrended cohort effects":

$$\tilde{\gamma}_c = \begin{cases} 0 & \text{if } c = 1 \\ \gamma_c - \beta \cdot c & \text{if } c \in (2, C] \end{cases}$$

where 1 denotes the first cohort included in the model and C denotes the last; and $\beta \equiv \frac{\gamma_C - \gamma_1}{C}$ is the long-run trend in cohort effects. This trend is the average per-year-of-birth change in effects between the first and last cohort in the sample. For example, when analyzing the 1930 to 1965 cohorts β would represent the average improvement or decline in health or human capital between each cohort, from those born in 1930 to those born in 1965.

The normalization of the first cohort effect to 0 is standard in fixed-effect models. This type of normalization is often described as identifying effects up to a "level-shift". The detrended effects shown above further normalize the last effect to be zero. They can be thought of as identifying effects up to both a level *and* a "trend"-shift.

I estimate the reparameterized version of Equation 1 by linear regression, and will focus on the detrended cohort effects $\tilde{\gamma}_c$. Graphical inspection of the sequence of detrended cohort effects will allow for the identification of *slope changes* in cohort effects. For example, below I show that the estimated detrended cohort effects in multiple outcomes have a clear piecewise linear shape, increasing linearly until the 1947 cohort and then reversing trend and declining linearly for subsequent cohorts. These results imply that there was a sharp *change* in slope at the 1947 cohort. However, because the long-run trend in cohort effects β is not identified, the results cannot distinguish whether there were absolute declines starting with the 1947 cohort, or merely a sudden slowing of a previous trend of improvement.

Results

Figure 2 shows the results of estimating the detrended, additively separable age-period-cohort models of Equation 1 for the four main outcomes: men's median log earnings, the low birthweight rate by mother's birth cohor, and the log mortality of men and women. The estimated cohort effects for each of the different outcomes exhibit remarkably similar patterns. They each have an approximately piecewise linear shape with a large slope change located at or near the 1947 cohort, and declines for cohorts born after that year. These patterns are consistent with a large decline in cohort health and human capital, relative to trend. This decline began near the 1947 cohort and was broad enough to impact outcomes as disparate and far reaching as maternal health, earnings, and mortality.

Panel A shows results for the low birthweight rate of infants by their mother's birth cohort, using the natality vital statistics microdata. Recall that age and cohort in these models refer to the age and year of birth of the *mother*. Therefore the estimated cohort effects can be viewed as estimates of the maternal health of given cohorts using the health of their infants as a proxy, and also reflect an intergenerational effect of cohort health on infant health in the next generation.

The estimated cohort effects exhibit a piecewise linear shape: declining rapidly until the 1947 cohort, sharply changing slope after that cohort, and increasing nearly linearly until the 1965 cohort. The cohort effects decline from the normalized 0 in 1935 to a minimum of -.87 for the 1947 cohort, before reversing trend. Under the additive separability assumptions of this model, this figure suggests a large change in the slope of the cross-cohort maternal health trend at the 1947 cohort, such that each cohort born after this year has declining health relative to the trend for prior cohorts. The size of the change in slope suggests than the 1965 cohort would have had a low birthweight rate approximately 2.2 percentage points lower had the cohort health decline not occurred.

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

Panels C and D show similar results for the log mortality rate of men and women, using data from the Human Mortality Database (HMD). The shape of the estimated cohort effects are not as sharply piecewise linear as those for the labor market and maternal health outcomes. However, they exhibit clear changes in slope near the late 1940s cohorts, consistent with elevated mortality and declining health for subsequent cohorts.

For men's log mortality the cohort effects decline — not precisely linearly from 0 to below -.1 by the 1946 cohort, then suddenly reverse trend and increase rapidly until reaching above .05 by the late 1950s

cohorts.[13] They then flatten and decline slightly for subsequent cohorts. This pattern suggests that men born in 1960 had mortality near .25 log points higher than they would have had health improvements continued at the same rate as for the 1930 to 1946 cohorts.

For women's log mortality the cohort effects exhibit two smaller slope changes at the 1946 and 1950 cohort, but still show evidence of a decline in health after the late 1940s relative to the prior trend. The cohort effects decline from 0 to below -.11 by the 1946 cohort, they then change trend and are nearly flat until the 1950 cohort. They then change trend *again* after the 1950 cohort and increase nearly linearly to 0 by the 1965 cohort. Overall, the size of the two slope changes imply that the 1965 cohort has had nearly .25 log points higher than it would had the health improvements for the 1930 to 1946 cohorts continued at the same rate for later cohorts.

These results are striking. However, they could in principle be biased by external factors which disproportionately impact individuals of particular ages in particular years. That is, cohorts born after 1947 may have been otherwise similar to earlier cohorts, but were just unlucky to have experienced bad conditions throughout their lifetime. Because the same pattern holds for very different outcomes, measured at different points in individuals lifetimes this "bad luck" would reflect a surprising coincidence. Each cohort born after 1947 would have to have been coincidentally exposed to poor obstetric conditions at the particular ages which they gave birth — which did not impact the outcomes of mothers of other ages giving birth in the same year. Similarly, they would have to have been exposed to labor market conditions that were particularly bad for workers of their age — for example low demand for young workers when they were young. Finally, they would have to have been exposed to diseases and other mortality risk factors which specifically increased mortality for individuals of their age, while not impacting individuals of other ages.

While such a coincidence would be surprising, below I implement two novel methodological approaches which allow for such external age-by-year interactions, and can still identify a decline in cohort health and human capital. The methodologies relax the additive-separability assumptions of the traditional approach used in this section. They can identify a slope change in cohort effects while allowing for changing conditions which disproportionately impact individuals of particular ages — under the assumption that those changes take the form of a polynomial in age.

4. Evidence of cohort decline under weaker assumptions

I present evidence of a decline in cohort health and human capital using two novel methodologies, which allow me to identify a slope change in cohort effects while controlling for changes in external, age-specific factors across years. The key assumption is that these age-by-year interactions take the form of a polynomial-in-age in each year, of a known order.

[13] Note that cohort is defined directly in this data, rather than measured with error based on age and year. The one-cohort difference in the timing of the cohort slope change is therefore perhaps not surprising.

A. Conceptual model

My goal in this section will be to identify a model with piecewise linear cohort effects, against a null model in which cohort effects are linear. In other words, I want to test whether the patterns in each of the above outcomes are consistent with a change in the cross-cohort trend in health and human capital leading to declines for those born after 1947, relative to the prior trend. I will seek to rule out a null in which there was no change in the cross-cohort trend in health and human capital. This null includes the possibility of constant cohort effects, ie. that the health and human capital of all cohorts are identical and they have merely been subjected to different conditions.

Consider the following model:

$$Y_{apc} = \underbrace{\beta \cdot c}_{\text{long-run trend in cohort effects}} + \underbrace{\mathbb{1}_{c \geq \lambda} \cdot \delta \cdot (c - \lambda)}_{\text{change in slope of cohort effects}} + \underbrace{f^p(a)}_{\text{year-specific impact of age}} + \epsilon_{apc} \tag{2}$$

where as above Y_{apc} denotes an outcome — such as earnings — for individuals who are age a, in the year or "period" p, and who are members of the cohort c.

The first two terms on the right-hand-side specify the cohort effects as piecewise linear with a single, *unknown* slope-change or knot. As above, β represents a long-run trend in cohort effects. The second term on the right-hand side now introduces a change in the slope of cohort effects at some unknown location λ. δ represents the size of this change in cohort slope, and λ estimates the cohort at which it occurs. This specification of the shape of cohort effects is similar to that seen visually in the detrended cohort effects estimated above and shown in Figure 2.

The goal in this section is to test whether that visual evidence of piecewise linear cohort effects, and therefore of a cross-cohort decline in health and human capital, are robust to controlling more flexibly for external factors which impact individuals of different ages in different years. In particular, I assume that these changing external age-by-year factors, $f^p(a)$, take the form of a polynomial of known order in each year. I assume that after controlling for these polynomials the only remaining factor is an orthogonal error, ϵ_{apc}. I then test whether the location of change in cohort slope λ is consistently estimated to occur near the 1947 cohort. I also test whether a slope change in cohort effects occurs by testing the null that $\delta = 0$. I am able to identify the above model because the discontinuous change in the slope of cohort effects is orthogonal to the smooth, polynomial age-by-year interactions.

Intuitively, I allow external factors which impact individuals of different ages to change but restrict them to do so smoothly. Most alternative explanations I want to rule out would take such a smooth form. For example, changes in the supply or demand for workers of different experience levels will have a smooth impact across ages, as long as individuals of nearby ages are sufficiently close substitutes. Labor economists traditionally control for quadratic experience terms in Mincerian wage regressions (Mincer, 1974). Similarly, if the biological aging process is smooth then changes in the disease environment — such as the HIV epidemic — may disproportionately increase the mortality of young adults; but they will not discontinuously increase mortality for those under age 30 and have no impact on 31-year-olds. Log mortality is generally

found to be remarkably *linear* in age (Gompertz, 1825; Chetty et al., 2016), suggesting that allowing for higher-order polynomials is quite unrestrictive.[14]

nonsmooth changes in the impact of age across years are the remaining threat to validity. An example of such a confounder would be: a large shock in a given year to the health of those age 30 and under, which did not impact those age 31 and over. Social policies with age-based eligibility cutoffs could take such a form. However, to erroneously generate a slope change in cohort effects such policies would have to have disproportionately and discontinuously impacted particular cohorts in comparison to neighboring ones. As described above, it is important to note that I find similar evidence of a cohort decline in multiple outcomes measured at different times in an individuals lifetime, from different data sources. Any source of bias would therefore have to have "hit" the same cohorts repeatedly at different times in their life, in such a way to impact the health of their infants, their earnings, *and* their likelihood of early death.

B. Differencing age profiles

Guided by the above model, my first novel approach involves taking the difference between outcomes in neighboring years, of individuals who are the same age. I refer to this as differencing the "age profile" of outcomes in neighboring years. A discontinuous slope change in cohort effects will be evident as a discontinuity or "mean shift" in these differenced outcomes, similar to those in regression discontinuity designs. If no such slope change exists — if the cross-cohort human capital trend is linear — there will be no discontinuity.

Hypothetical example

I first motivate the age-profile-differencing approach with a hypothetical example. In the example cohort effects are piecewise linear and the impact of age takes a different cubic form in the two years shown. Taking the difference between the age profiles in the two years then reveals a discontinuity or "mean shift" at the cohort at which the slope change in cohort effects is located.

Consider hypothetical age profiles of earnings in 1981 and 1980. Assume that there is a slope change in cohort effects of size $\delta < 0$ which occurs at the 1947 cohort. There are also external factors which impact earnings of workers of different ages differently in the two years. Assume that the impact of these external factors can be summarized by a *different* cubic function of age in each of the two years.

Then the age profiles can be written as:

$$Y_{a,1981,c} = \underbrace{\beta \cdot c}_{\text{cohort trend}} + \underbrace{\delta \cdot \mathbb{1}[c >= 1947] \cdot (c - 1947)}_{\text{change in slope of cohort effects}} + \underbrace{f^{1981}(a)}_{\text{age effects}}$$

$$Y_{a,1980,c-1} = \underbrace{\beta \cdot (c-1)}_{\text{cohort trend}} + \underbrace{\delta \cdot \mathbb{1}[c-1 >= 1947] \cdot (c-1-1947)}_{\text{change in slope of cohort effects}} + \underbrace{f^{1980}(a)}_{\text{different age effects}}$$

Age profiles of this form are shown in Panel A of Figure 3. The dashed lines show what the age profiles

[14] In an earlier paper I use this remarkable log linearity, known as Gompertz law, to provide graphical and statistical evidence for the importance of a cohort health decline in the recent increases in white mortality, in particular (Reynolds, 2019).

would be absent the cohort slope change. In this example, there is a decrease in the return to experience between 1980 and 1981: external factors are pushing wages of young workers up significantly in comparison to older workers. The solid lines show the age profiles of earnings including the change in slope of cohort effects. The slope change in cohort effects induces slope changes in the age profiles of size δ at age 33 in 1980 and at age 34 in 1981.

Comparing workers of the same age in the two years combines two effects. First, it reflects the changing impact of external factors which impact workers of different ages differently. The difference in age profiles will include a term $f_{1981}(a) - f_{1980}(a)$. Second, it will compare workers who were born one year apart. For example, workers who are 40 in 1981 were born in 1941, while those who are 40 in 1980 were born a year earlier in 1940.[15] Therefore, the difference in age profiles will also include a term reflecting the *slope* of cohort effects. Under the specified piecewise linear shape of cohort effects, the slope of cohort effects changes discontinuously by δ from the 1947 to the 1948 cohort. This discontinuous change in slope will therefore be reflected in a discontinuous level or "mean shift" in the differenced age profiles.

That is the difference in age profiles in this example can be written as:

$$Y_{a,1981,c} - Y_{a,1980,c-1} = \beta + \underbrace{\delta \cdot \mathbb{1}[c >= 1948]}_{\text{cohort slope change as "mean shift"}} + \underbrace{f^{1981}(a) - f^{1980}(a)}_{\text{change in age-effects across years}}$$

This mean shift shape of the differenced age profiles is shown in Panel B of Figure 3. The changing impact of external factors across years induces a smooth polynomial shape to the differences. The dashed line shows that the differenced age profiles would therefore take a smooth polynomial shape absent a cohort slope change. In contrast, the black dots show that with the existence of the cohort slope change, the differenced age profiles exhibit a mean shift from the age 33 to age 34, and otherwise inherit the global polynomial form of the change in age-effects across years. The size of the mean shift is equal to the size of the slope change in cohort effects δ.

Panel C simply recasts the differenced age profiles with birth cohort in 1981 on the x-axis. When the differences are indexed in this way the mean shift occurs from the 1947 to 1948 cohort, falling sharply by δ.

Graphical test

Building on the intuition in the above example, I implement a graphical test of whether there is a cohort slope change. For each of the outcomes I will plot the average of the differenced age profiles by birth cohort. If a change in the slope of cohort effects occurs at the 1947 cohort, a discontinuity or "mean shift" will be visually evident in these differenced outcomes. Absent such a slope change, the differenced outcomes will take a smooth polynomial form.

If the model takes the form of Equation 2, a general expression of the differenced age profiles is:

[15] This discussion takes as given that $c = p - a$. In practice, I will not observe cohort directly and will *define* it in this way, inducing some error.

$$Y_{apc} - Y_{a,p-1,c-1} = \beta + \underbrace{\delta \cdot \mathbb{1}[c >= \lambda + 1]}_{\text{mean shift at } \lambda+1} + \underbrace{f^p(a) - f^{p-1}(a)}_{\text{difference in age effects between years}} + \epsilon_{a,p,c} - \epsilon_{a,p-1,c-1} \quad (3)$$

Then taking the average of these differenced outcomes across all years *by birth cohort* yields:

$$\mathrm{E}\big[\, Y_{apc} - Y_{a,p-1,c-1} \;\big|\; c \,\big] = \beta + \underbrace{\delta \cdot \mathbb{1}[c >= \lambda + 1]}_{\text{mean shift at } \lambda+1} + \underbrace{\mathrm{E}\big[\, f^p(a) - f^{p-1}(a) \;\big|\; c \,\big]}_{\text{polynomial in cohort}} + E[\epsilon_{a,p,c} - \epsilon_{a,p-1,c-1} | c] \quad (4)$$

The average of the differenced age profiles by cohort will combine three effects. First, the slope change in cohort effects will induce a mean shift, located one cohort after the location of the change in slope. Second, there will be a term reflecting the changing impact of age across years, averaged by birth cohort. Under the assumption that changes in the impact of age, $f^p(a) - f^{p-1}(a)$, take the form of a polynomial of known order D, this second term will also take the form of a polynomial in cohort of the same order D. Third, there will be a final term reflecting the difference in orthogonal errors. I assume that this term is orthogonal and equal to 0. That is, that after "controlling" for a polynomial in age in each year, all remaining external shocks are mean 0 by cohort.

Under these assumptions, plotting the sample average of these differenced outcomes across all years allows for a visual test of the existence of a change in the slope of cohort effects. A slope change in cohort effects at cohort λ of size δ will induce a sudden level or "mean shift" from λ to $\lambda + 1$ of size δ. Under the null of no cohort slope change, δ is equal to 0, the mean shift term will drop out, and the plot will instead take the smooth polynomial form inherited from the changing impact of age. No sharp mean shifts will be evident.

I first implement this graphical test for the mean infant birthweight of infants by mother's birth cohort. This outcome appears to be the most precisely estimated, because the vital statistics natality data cover nearly the universe of births and it measures the mean rather than a tail outcome. I plot the average of the differenced age profiles in this outcome for the years 1968 to 1995, by mother's birth cohort.

Figure 4 shows the results. The differenced outcomes exhibit a large, visually-evident shift in mean from the 1947 to the 1948 cohort. While the differences are centered near 5 to 10 grams for the 1930 to 1947 cohorts of mothers, they suddenly drop to near 0 for the 1948 cohort. They then remain near, and primarily below, 0 for the remaining cohorts born until 1970. In the context of the above model, this large shift in mean from the 1947 to 1948 cohorts in the differenced age profiles is consistent with a large change in the slope of cohort effects occurring precisely at the 1947 cohort. The visually evident size of the shift appears to be nearly a 7 gram decline, implying a change in slope of cohort effects, δ, of that size.

Figure 5 then implements this graphical test for the four primary outcomes. Because of the lower precision of these outcomes, I plot the average of the age profile differences in 2-cohort bins. Three of the outcomes, share low birthweight, earnings, and men's mortality, show clear visual evidence of a mean shift

from the 1947 to the 1948 cohort, consistent with change in slope of cohort effects and declining health and human capital after that year. The visual evidence of a mean shift is less sharp for women's log mortality. For this outcome there appear to be two smaller discontinuities, consistent with two smaller changes in the slope of cohort effects.

Panel A shows results for the percentage of infants born at low birthweight, by mother's birth cohort. The differenced outcomes are centered near -.15 for the 1930 to 1947 cohort. There is then a discrete increase from the 1947 to the 1948 cohort and the differenced outcomes are centered near .025 for all subsequent cohorts, up to mothers born in 1970. The visually apparent shift in mean of approximately .15 is consistent with slope change in cohort effects of that size. In other words, each cohort of mothers born after 1947 was .15 percentage points more likely to give birth to a low birthweight infant, relative to trend.

Panel B shows analagous results for the median log hourly wage, measured in the CPS-MORG. The differenced outcomes are near 0 for the 1930 to 1947 cohort. There is then a discrete decrease from the 1947 to the 1948 cohort. The differenced outcomes are then centered near -.015 for all subsequent cohorts, up to men born in the late-1960s. The visually apparent shift in mean of approximately -15 log points is consistent with a change in the slope cohort effects of that size. This would imply a decline in earnings ability, such that each subsequent cohort born after 1947 had 15 log points, or near 1.5 percent, lower earnings than that born a year earlier, relative to trend.

Panel C shows similar results for the log mortality rate of men. The differenced log mortality rates from the 1930 to 1947 cohort increase gradually from -.02 to -.01. They then exhibit a discrete mean shift and suddenly increase to .01 for the 1948 cohort. They then decline linearly, and relatively rapidly, until the 1965 cohort. In the context of above model, the discrete change in mean from the 1947 to 1948 cohort is consistent with a large slope change in cohort effects at the 1947 cohort — leading to increased likelihood of premature death for subsequent born cohorts, relative to trend. The size of the mean shift of near -2 log points, implies a slope change in cohort effects of that size.

Panel D shows results for women's log mortality, and reveals the least sharp visual evidence of a mean shift. The differenced age profiles appear to exhibit two smaller shifts in mean: one increase from the 1947 to 1948 cohort, and one a few cohorts later at the 1951 cohort. Interestingly, this pattern is consistent with the shape of the detrended cohort effects estimated above for this outcome and shown in Figure 2, which exhibited two smaller slope changes in cohort effects at the 1947 cohort and the 1951 cohort. However, the visual evidence is much less striking than for other outcomes.

Formal test

I also implement a formal test for the existence of a cohort slope change by testing for a mean shift in the differenced age profiles, while controlling for separate polynomials in age in each year.

I estimate the following model with a mean shift of *unknown location*:

$$Y_{apc} - Y_{a,p-1,c-1} = \beta + \underbrace{\delta \cdot \mathbb{1}[c >= \tilde{\lambda}]}_{\text{discontinuous change in mean at } \tilde{\lambda}} + \underbrace{\tilde{f}^p(a)}_{\text{separate poly in age in each year}} + \Delta\epsilon_{apc} \quad (5)$$

$\tilde{\lambda}$ denotes the unknown location of the mean shift, which is a parameter to be estimated. The mean shift will occur one cohort later than the change in cohort slope, ie. $\tilde{\lambda} = \lambda + 1$. The parameter δ denotes the size of the mean shift, and therefore provides an estimate of the size of the slope change in cohort effects. I then control for the possible changing impact of external factors impacting individuals of different ages, with a separate polynomial-in-age in each year, $\tilde{f}^p(a)$.

I estimate the model by least squares, following the structural break methodology in Hansen (1999, 2000). Algorithmically, this amounts to looping through different assumed values of the mean shift location $\tilde{\lambda}$, and selecting the location with the lowest sum of squared residuals.

I invert the likelihood ratio statistic in Hansen (2000) to form 99 percent confidence intervals for $\tilde{\lambda}$. Hansen (2000) also suggests that inference on δ is unaffected by treating $\tilde{\lambda}$ as unknown. I therefore form confidence intervals for δ using the standard formula for least squares. Following standard practice, I employ an ad-hoc restriction to prevent the location of the cohort break $\tilde{\lambda}$ to be estimated to be one of the youngest or oldest cohorts in the sample. In particular, in each year I restrict the location of the break to not be one of the 5 youngest or oldest cohorts.

To test for the existence of a slope change in cohort effects, I test the null that $\delta = 0$. A standard t-test would be invalid in this setting, because the location of the mean shift $\tilde{\lambda}$ is not identified under the null. I therefore follow the bootstrap procedure described in Hansen (1996, 2000) to test the null hypothesis that no trend break occurs, ie. that δ is equal to 0.

Table 1 shows results of fitting mean shift models based on Equation 5 for the main outcomes, while controlling for a separate quadratic-in-age in each year. The results confirm the existence of a discontinuous change in slope in all of the outcomes at or near the 1947 cohort.

The location of the estimated shift in mean — paralleling the visual impression from the plots in Figures 4 and 5 — are again located at or near 1948. For *all* of the first three outcomes — mean birth weight, percentage low birthweight, and the median log wage — the estimated location of the mean shift is precisely estimated to be the 1948 cohort — consistent with a change in the slope of cohort effects located at the 1947 cohort. Further, the 99 percent confidence interval for all of these estimates include only the 1948 cohort. The estimated locations of the mean shift for log mortality are 1947 and 1951 respectively, consistent with slope changes located at the 1946 and 1950 cohort. Again, the 99 percent confidence intervals include only a single cohort.

The estimated mean shifts — and therefore the implied change in slope of cohort effects — are all large in magnitude. The estimated mean shift for median log wage is -.020, implying a large decline in the cohort slope of labor market ability. The estimated mean shifts for intergenerational infant health are -7.49 grams for mean birth weight and .196 for the percentage low birthweight. The mean shift estimates for log mortality are .030 and .027 for men and women respectively.

Applying the bootstrap test of Hansen (1996) to test whether a mean shift exists, I fail to reject the null of no mean shift at a very low significance level. For median log wage the p-value is .001. For all other 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 mean shift. Following the logic outlined above

this therefore suggests that the data are consistent with a discontinuous change in the slope of cohort effects — located at or near the 1947 cohort — for all outcomes.

Appendix Table A1 examines the robustness of these results to varying the age-by-year control function. I estimate models including just age fixed effects and year fixed effects, as well as those also including polynomials of increasing order — up to a separate quartic-in-age in each year. The intergenerational infant health and median log wage results are remarkably robust. For all of the models the mean shift is estimated to occur at the 1948 cohort, and p-values for the null of no change in mean consistently fall at or below .005. The estimated size of the slope change δ actually *increases* in magnitude as more flexible age-by-year controls are added.

The mortality results are less robust. The confidence intervals for the location of the mean shift always include 1947 — but for models with cubic or higher polynomials the point estimate at times moves to 1953. Additionally, some of the estimates of the size of the slope change turn negative. The results for log mortality below using the second, and likely more efficient, approach are more robust.

C. Piecewise linear cohort models

My second novel approach directly estimates the model given in Equation 2 in which the cohort effects are specified to have a piecewise linear shape. I again follow the structural break methodology of Hansen (1999, 2000), leaving the location of the kink or "knot" of these piecewise linear cohort effects as a parameter to be estimated. This approach yields similarly strong evidence, for all outcomes, of large changes in the slope of cohort effects at or near the 1947 cohort.

When $f^p(a)$ is specified as additively-separable age and year effects, then the model in Equation 2 is nested in the age-period-cohort models described above, and restricts the shape of the cohort effects to be piecewise linear. It therefore allows for the visually evident slope change from that approach to be summarized in two parameters — the slope change size, δ and its location, λ. Following the structural break methodology of Hansen (1999, 2000) I also provide associated estimates of the uncertainty of these parameters.

Restricting the shape of cohort effects in this way also allows for the introduction of additional age-by-year interactions which would have made the general age-period-cohort model unidentified. In particular, the location and size of the trend break are still identified with the introduction of separate polynomials in age *in each year*. This approach, as in the age-profile-differencing approach above, allows me to probe the robustness of the estimated change in cohort slope to increasingly flexible "smooth" age-by-year interactions. I experiment with including higher order polynomials in age in each year, up to including a separate quartic in age in each year. In general, the location and sign of the estimated change in cohort slope are robust to the inclusion of these higher order polynomials. For maternal health the magnitude actually *increases* when higher order polynomials are included.

I estimate the model in Equation 2 for the main outcomes described above by least squares, again following the methodology in Hansen (1999, 2000). My baseline specification includes the following as controls: age fixed effects, year fixed effects, and a separate quadratic-in-age in each year. They therefore

allow for smooth age-by-year interactions of a quadratic form.

Results

A graphical depiction of the estimation and inference of the location of the cohort slope change, λ, is shown in Figure 6. As above, I invert the likelihood ratio statistic given in Hansen (2000) to form 99 percent confidence intervals. Each panel plots this likelihood-ratio test statistic for different assumed locations of the change in the slope of cohort effects. The cohort with the minimum value of the likelihood-ratio test statistics yields the point estimate of the location, λ. The 99 percent confidence region is those cohorts falling below the 1 percent critical value shown with a dashed grey line. The four panels each plot the results from models with a different outcome, and all reveal precisely estimated locations of the cohort slope change.

Table 2 provides the full results of estimating the piecewise linear cohort effects based on Equation 2, for the five main outcomes. The estimated location of the change in cohort slope — and therefore the implied cohort after which the health and human capital decline begins — are centered at the 1947 and 1948 cohorts. For both of the maternal health outcomes, infant mean birth weight and share low birthweight, the slope change is estimated to occur at the 1948 cohort. For both of these outcomes, the 99 percent confidence interval includes *only* a single cohort. For the median log wage it is estimated to occur at the 1947 cohort, with a confidence region including only 2 cohorts — 1946 and 1947. Cohort effects in models of the log mortality of men and women are estimated to have a slope change at the 1946 and 1949 cohorts respectively. Again these locations are precisely estimated — with the 99 percent confidence interval including only a single cohort.

The estimated size of the changes in cohort slope are all large in magnitude. The estimated size of the change in slope, δ for the median log wage is -.016. This implies that the median man in the 1960 cohort has earnings roughly 23 percent lower than they would have had the cohort effects followed the pre-1947 cohort trend.

The magnitude of the intergenerational infant health effects are also large. For mean birth weight the size estimate of -6.35 grams implies that the 1960 cohort has given birth on average to 76.2 grams lighter infants, than they would have if the pre-1948 cohort trend had continued. More strikingly, the share low birthweight estimated slope change size of .23 implies that the 1960 cohort had a low birthweight rate near 2.8 percentage points higher than they would had the earlier cohort trend continued. The low birthweight rate nationally in 1975 was only 7.4 perecentage points. Therefore this effect of the health decline by the 1960 cohort is on the order of 40 percent.

The size of the slope change in cohort effects for log mortality are similarly striking in their large magnitude. For men the estimated size of the slope change is .029, implying the single-year mortality risk for the 1960 cohort was roughly *1.5 times* what it would have been had the pre-1946 trend continued. For women the estimated size is quite similar at .031, implying a similar counterfactual difference in mortality risk.

For all six outcomes, I fail to reject null hypothesis of no change in cohort slope at a very low significance level. As outlined above, I follow the bootstrap procedure described in Hansen (2000) to test the null hypothesis that no change in cohort slope occurs, ie. that δ is equal to 0 and that cohort effects are linear. 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 linear cohort effects.

Appendix Tables A2 show the robustness of these estimates to different specifications of the age-by-year control function. I examine the robustness to including different age-by-year interactions, from allowing none, to including separate quadratic, cubic, or quartic polynomials in age in each year.

For the maternal health and median log wage models, the finding of a change in the slope of cohort effects located at or near the 1947 or 1948 cohort are quite robust. Results across *all* specification for both the mean birth weight and low birthweight share reveal estimated break locations between 1947 and 1949. The sign of the slope change estimates all imply a decline in maternal health. The magnitude of the implied decline *increases* once a quadratic-in-age in each year is added, and is stable with the addition of higher-order polynomials.

For the median log wage, 3 out of the 4 specifications yield an estimated break at the 1947 cohort; only the specification including cubic age-polynomials yields a different estimate — of 1953 — and the confidence region in this model also includes 1946 to 1947 as alternatives break locations. For the cubic specification, the implied p-value for the likelihood ratio test of whether the break point occurs at 1947 is just .01. If one imposes the break location location to be 1947, the estimate of the size of the break is again negative and of nearly identical magnitude to the other specifications, at -.016.

For men's log mortality, including no age-by-year controls yields an estimated health decline beginning a few cohorts earlier, but with the addition of quadratic or higher polynomials the estimated location stabilizes at the 1946 or 1947 cohort. The magnitudes also stabilize near .029. For women's log mortality a cohort health decline, beginning between 1947 and 1950, is detected in models up to including a cubic-in-age in each year. When a quartic is included the estimated slope change moves to 1942 and turns negative.[16]

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

The estimated cross-cohort health and human capital decline is large in magnitude, and has therefore been an important driver of the year-over-year trends in the low birthweight rate, earnings, and mortality. In this section, I present the results of a simple estimation procedure which demonstrates this contribution. My estimates suggest that absent the cohort health decline: the increase in low birthweight births in the 1980s would not have occurred, there would have been modest real earnings growth since the late-70s rather than real declines, and midlife mortality would have continued to decline steadily at it's pre-1999 rate.

I perform a simple simulation to examine the counterfactual in which there was no cohort health decline. For each outcome, I use the estimated location, $\hat{\lambda}$, and size, $\hat{\delta}$, of the change in cohort slope from the models estimated above based on equation 2. To be conservative, for the low birthweight percentage I use estimates from models with only age and year fixed effects, for which the change in slope is of smaller magnitude. For

[16]The relative instability of the results for the log mortality for women may be the result of the cohort effects being misspecified as piecewise linear. Also note that findings for white mortality in Reynolds (2019) using a different methodology reveal highly robust evidence of a decline in cohort health located near 1946 for white men and 1949 for white women.

the other outcomes, I use estimates from the baseline specification reported in Table 2. For each outcome I "remove" the cohort slope change, by subtracting it from each observation. That is, I create a transformed dataset, where each observation takes the following form:

$$\tilde{Y}_{apc} \equiv Y_{apc} - \mathbb{1}_{c \geq \hat{\lambda}} \cdot \hat{\delta} \cdot (c - \hat{\lambda})$$

I then calculate summary measures of each outcome by year using both the raw data, Y_{apc}, and the transformed data, $\tilde{Y}_{apc}$, in which the estimated cohort slope change has been removed. Comparing the trends in these summary measures then reveals the contribution of the change in cohort slope to the year-over-year trend.

Figure 7 shows the results. Panel A shows trends in the percent of infants born at low birthweight. The raw series shows that while the low birthweight rate declined from above 8 percent in 1968 to 6.7 percent by 1985, it then reversed trend and increased to near 7.3 percent by 1995 (Centers for Disease Control, 1994). In contrast, the transformed series in which the cohort slope change has been removed exhibits no similar increase. The series implies that absent the cohort health decline, the low birth weight rate would have declined much more rapidly until 1985 — falling to 5.7 percent by that year. These improvements would have slowed after the mid-1980s — but the low birthweight rate would have continued to decline slowly until 1995 to just below 5.5 percent.

Panel B shows similar results for men's wages. In particular it shows the average across men age 25 to 54 of median wages by single age-bins.[17] This measure of earnings declines in real terms from 1979 to 1993, from 3.14 to below 3.04. By this measure real wages declined from 23 to below 21 dollars an hour, in constant 2014 dollars. In contrast the transformed log wage series, in which the cohort slope change is removed, increases very slightly from 3.16 in 1979 to 3.17 in 1993.

Panel C and D show analogous results for the mortality rates of men and women age 45 to 54, age-adjusted assuming a uniform population distribution by age. I focus on this age group to ease comparison with the focus of Case and Deaton (2015) on midlife mortality. The raw data show declines in men's and women's midlife mortality between 1970 and 1990. Both series then show a slowing of declines during the AIDS epidemic, followed by a rebound in the mid-1990s after the development of antiretroviral treatment — with a larger impact for men. Both series then exhibit a clear change in trend near 1999 and slowing improvements in the mortality rate after that year. For men the mortality rate even increases between 1999 and the mid-2000s. This change in trend near 1999 in men's and women's mortality is completely absent in the transformed series in which the cohort slope change in log mortality is removed. That is, the decline in cohort health can completely explain the stagnation — and for men slight increase — in mortality after 1999.

Below I show evidence that a change in slope of cohort effects is also evident for white mortality analyzed separately. To provide an even closer comparison with Case and Deaton (2015, 2017), Appendix Figure A5 shows the results of this simulation exercise for midlife white mortality rates. The results suggest the decline in cohort health can completely explain the increase in midlife white mortality after 1999.

[17] If log wages are assumed to take a symmetric distribution, such as if wages are assumed to be log normal, then this provides an estimate of age-adjusted mean log wages. Where the age-adjustment is made with a uniform population distribution.

My cohort-based theory also provides an explanation for the previously surprising timing of the midlife mortality increases — beginning in 1999, during an economic boom. Midlife mortality changed trend in 1999 not because of factors specific to that year, but because "unhealthy" cohorts born after the late-1940s began to age into the 45 to 54 age group after that year. Appendix Figure A5 shows that the differential timing of white mortality increases in different age groups can also be explained by the cohort-based theory. This argument is developed in more detail in my earlier paper which focused exclusively on the contribution of a cohort health decline to recent increases in the mortality rate of white Americans (Reynolds, 2019).

6. Connection to educational declines

The timing by cohort of previously documented declines in educational attainment and standardized test scores closely match that of the declines in health and human capital I show above — suggesting they are likely linked. One hypothesis is that the educational declines were driven by some external, "supply" factor, and the direct causal effect of education on earnings and health can explain the subsequent declines in other outcomes. I present preliminary evidence against this simple "education-only" explanation, and instead suggest that a broad decline in observable and unobservable health and human capital drove both the above declines and the educational and test score declines. However, a full accounting of the link between education, earnings, and health in this cohort decline will require further research.

I also document a sudden, and to my knowledge previously unnoticed, decline beginning with the 1947 cohort, in the share of men earning an advanced degree; as well as a similarly timed stagnation of prior improvements for women.

A. Review of educational and test score declines

Previous authors have noted a sudden decline in the educational attainment for cohorts of Americans 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 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 and Autor (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."

Figure A6 summarizes these patterns using the CPS MORG data.[18] For men the cross-cohort trends

[18] I pool data from 1990 to 2018, individuals age 25 to 75, and calculate the share of each cohort who have achieved different

in years of schooling, high school graduation (or GED), and Bachelor's degree attainment, all exhibit clear breaks — or slope changes — at the 1947 cohort such that prior improvement in educational attainment stops and *reverses* before declining through the mid-1960s cohorts. For women there is clear evidence of a slowing in improvement located near the late-1940s cohorts, though it is less sharp and consistently located across cohorts. Further, years of schooling and high school attainment for women stagnate but do not decline in absolute terms. Women's college attainment continues to increase until the 1950 cohort, before declining slightly.

Panel D reveals a sharp decline beginning with the 1947 cohort in the share of men earning an advanced degree. This share, after increasing from .1 for the 1930 cohort to more than .15 for the 1947 cohort, suddenly began to decline for later born cohorts. It reached a nadir of less that .09 by the mid-1960s cohorts. That is, the share of men earning an advanced degree was *lower* for the 1965 cohort than for the 1930 cohort. For women this share suddenly plateaus beginning in 1947, before declining in absolute terms after 1952. To my knowledge this decline had previously gone unnoticed. Cross-cohort declines are evident even considering the share earning an advanced degree *conditional* on earning a bachelor's degree. This decline, as well as a similar decline in bachelor's attainment conditional on finishing high school are shown in Figure A7.

There was also a widely noted decline in standardized test scores beginning in the late 1960s, which appears to closely match the timing by cohort of declines in other measures of human capital shown above. Most widely noted was a decline in Scholastic Aptitude Test (SAT) scores beginning in the early 1960s and lasting until the mid-1980s. Appendix Figure A8 shows SAT scores by approximate birth cohort, measured as the school year that the test was taken minus 17. By this measure, the average score on the Verbal portion of the SAT was nearly flat at around 475 points between the 1940 and mid-1940s cohorts. Scores then began to drop precipitously for cohorts born after the late-1940s, declining from a peak for the 1946 cohort of 478 points to 426 points by the 1965 cohort. Similarly, scores on the Math portion of the test were flat near 500 points for the 1940 to mid-1940s cohorts, and then also began to fall rapidly: from 502 for the 1946 cohort to 468 by the 1965 cohort.

A large literature studying this decline concluded that it could not be explained by changes in the composition of test-takers or changes in the difficulty of the test — but failed to find a conclusive cause (see eg. Koretz (1987)). A review commissioned by the College Board suggested that the declines in the 1960s could be explained to some extent by a changing composition of test takers — but that there was little scope for such observable selection effects in explaining continued declines after 1970 (Price and Carpenter, 1978). The share of 17-year-olds taking the test also remained nearly flat over the 1970s — ruling out a simple single-index selection story in which lower ability individuals were pulled into the test-taking population. The declines were also evident at the top of the score distribution, with the share of test-takers achieving perfect scores and scoring above 700 points both also falling in the 1970s. Finally, similar declines in test scores are evident in other achievement tests besides the SAT, including nearly universal tests given

levels of educational attainment. I calculate approximate average years of schooling for each cohort based on the 16 schooling categories in the CPS. The existence of a similar change in cohort slope is also robust to controlling for age effects, or age-and-year effects; and is detected by structural break estimation.

in Iowa and Minnesota (Harnischfeger and Wiley, 1975).

The shared timing of these declines by cohort in educational attainment and test scores to those documented above in other measures of health and human capital suggests they could plausibly be linked. A natural question is whether the direct causal effect of education on earnings and health can explain the other declines in health and earnings which I document. The complex and multi-directional causal connections between education, health, earnings, and other human capital investments makes answering this question challenging. Below I make an initial attempt and present some pieces of evidence which appear inconsistent with the "education-only" explanation. However, even if all the declines I've documented above were caused by the earlier declines in education and test scores — it would remain a notable finding that recent trends in health, mortality, and labor market outcomes are still being driven by changes in education supply nearly 50 years ago.

It is also interesting to note that many of the measures of educational attainment exhibit *absolute* declines — not just a slowing of prior rates of improvement. Recall, that the age-period-cohort methods models estimated above could not identify the long-run trend in cohort effects, and therefore could not determine whether cohort health and human capital declined in absolute terms, or prior improvements merely stagnated. If the educational declines are a symptom of a broader decline in health and human capital, it could suggest that underlying human capital also declined in absolute terms. Additionally, the evidence of declines in academic ability — as measured by test scores — at age 17 suggests that the decline in human capital predated labor market entry.

B. Implied causal effects of education — assuming no change in unobservables

I present a first test of the "education-only" hypothesis, by considering the implied causal effects of education on the other outcomes, under the assumption that unobservables remained unchanged across cohorts. Under the assumption that education is the only factor that changed across cohorts, one can use the post-late-1940s cohort change in slope as an instrument for education. For example, the estimated change in cohort slope in log earnings over the change in the cohort slope in years of schooling can be used as a two-stage least squares estimator of the return to a year of schooling. Analogous ratios for infant health and mortality can generate implied causal effects of mother's schooling on infant health and of education on mortality. By comparing these estimates to prior results in the literature I can assess the plausibility that the declines in other outcomes were caused by education alone.

I perform such a two-stage least squares exercise for log earnings, the low birthweight rate, and the log mortality of men and women. I calculate a separate "first-stage" estimate of the cohort slope change in years of schooling for each of the outcomes to address the slightly different selection in each sample.[19]

To calculate the "reduced-form" I simply apply Equation 2 to each outcome, and estimate the location

[19]For earnings I calculate years of schooling using the CPS MORG sample of employed men with non-missing earnings. For infant health I calculate mother's years of schooling directly from the natality files. Because mother's education is only available after 1969 and is missing for a non-trivial fraction of mothers, I also re-estimate the cohort decline in a restricted sample of births with non-missing maternal education, and find a similar cohort slope change to that in the full sample. For the mortality "first-stage" I use Decennial Census microdata from 1970-2000 to estimate education levels for the full population of the United States.

of the change in cohort slope as above following Hansen (1999, 2000). For the first-stage I then impose the location of the cohort slope change to occur at the same location as in the corresponding reduced-form. I then estimate by least squares a model similar to Equation 2 with years of schooling on the left-hand side, and with the location of the slope change, λ, treated as known. Finally, the two-stage least squares estimate is simply the ratio of change in cohort slope, delta, from the "reduced-form" over that in the "first-stage." [20]

Table 6 reports the results. Interestingly, the implied return for earnings to a year of schooling is .138 which is only slightly larger than OLS estimates and closely matches many of the IV estimates summarized in Card (2001). However, a separate IV estimate restricted to only black men would imply a much larger return to schooling of .25 — because the slope change in schooling for blacks is the same as for whites but that of earnings in nearly twice as large.

The implied causal effect of a year of maternal education on the low birthweight rate is twice the cross-sectional relationship, but is also remarkably close to the IV estimate of Currie and Moretti (2003). My two-stage-least-squares estimate of -.92 implies that a year of maternal education reduces the incidence of low birthweight by .9 percentage points. This estimate is nearly twice the cross-sectional correlation between low birthweight and years of schooling calculated from the same data However, it is remarkably similar to estimates from Currie and Moretti, who use the founding of colleges in a mother's county at age 17 as an instrument, and range from -0.96 to -.99.

The fact that the IV estimates are *larger* than the OLS estimate could be the result of measurement error in maternal years of schooling. Or as Currie and Moretti emphasize, it could be, following the logic regarding earnings of Card (2001), because those "marginal women" induced to change schooling levels by the instrument have a larger causal effect of education on maternal health than the average in the population. However, if one assumes that the direction of more traditional "ability bias" is negative, then this "selection on gains" would have to be very large to explain the IV estimates I find. In particular, the returns to schooling of those induced to change schooling levels by changes in education "supply" across cohorts would need to be double that of the average in the population. Given the large changes in the educational distribution across cohorts, this would imply that the causal effect of education in the population is *extremely* variable.

In contrast to the above results for earnings and maternal health, the implied log mortality effect of education appears implausibly large — in comparison to both the cross-sectional relationship and past estimates of the causal effect of schooling on mortality. My two-stage-least-squares estimates, using the cohort-slope change as an instrument for years of schooling and log mortality as the dependent variable, are -.251 and -.434 for men and women respectively[21]. This would imply that earning a 4-year college degree causes a male's mortality risk in a given year to be reduced by *nearly two-thirds* (specifically a reduction of .63 percent). The implied effect of a 4-year college degree for women is even larger — suggesting it would decrease the mortality rate by *more than 80 percent*.

These estimates are larger than cross-sectional differences. For example, in the 1980s the mortality rate

[20] The interpretation of the two-stage-least squares estimates for log earnings are somewhat complicated by the fact that I use median rather than mean earnings. One simple justification for this procedure would be if the distribution of latent earnings in each bin is symmetric. Under this assumption the sample median is a consistent estimator of the mean (one which address topcoding).

[21] Standard errors are constructed using the two-sample, two-staged-least-squares formula in Inoue and Solon (2010).

at ages 25 to 64 of men with 16+ years of education was 49 percent lower than for men with 12 years of education. Mortality of women with 16+ years of education was just 31 percent lower than for women with 12 years of education (Elo and Preston, 1996).

Further, instrumental variable estimates of the causal effect of education on mortality using changes in compulsory schooling laws generally find *much* smaller causal effects than the cross-sectional relationship (Galama et al., 2018). For example, in a particular credible study based on UK schooling reforms Clark and Royer (2013) estimate a precise zero effect of schooling on adult mortality. Gathmann et al. (2015) pool data from 19 European countries and exploit schooling reforms throughout the 20th century. They estimate that a year of schooling reduces the mortality rate of men by 2.8. percent, but find no statistically significant effects for women. Further, the largest individual country estimate for men from that paper is only 5.6 percent.

The size of the cohort slope change in mortality appears *much* too large to be explained by the decline in education alone, and therefore strongly suggests that there was a broader decline in health and human capital for these cohorts.

C. Cohort declines in earnings ability and health conditional on education

I now show evidence of cohort declines in earnings and maternal health within some narrowly defined educational bins — health and earnings ability appears to have declined for post-1947 cohorts even *conditional* on education. First, I show robust evidence of a change in the slope of cohort effects of median earnings of those without a bachelor's degree. Then, I show evidence of similar change in slope of cohort effects for maternal health, as measured by infant birth weight, at many levels of maternal education, including exactly 12 years of education and exactly 16 years of education.

These findings suggest either that the change in cohort slope was driven by a decline in latent health and human capital *broader* than the educational declines alone; or that the change in educational attainment for late-1940s cohorts also involved large and unusual changes in selection effects, eg. the relationship between unobservables and years of schooling.

Earnings by education

Table 7 shows estimates of the change in slope of cohort effects in median log earnings of employed men separately for those with and without a bachelor's degree. I estimate the models with piecewise linear cohort effects based on Equation 2. All models include age and year fixed effects, and from left to right each column includes higher order polynomials in age separately for each year — up to a quartic polynomial.

Panel A shows remarkably robust results for the earnings of men without a bachelors degree: a cohort decline beginning with the 1947 cohort of similar size to that estimated for unconditional earnings above. For all specifications of the control function the estimated location of the cohort slope change is 1947 with only that cohort included in the 99 percent confidence interval, and the F-type bootstrap tests all imply a p-value of less than .001 for the null of no break. The size of the estimated change in slope varies from -.0124 to -.0207 — quite similar to the estimate of -.016 found above for unconditional earnings for the

entire sample of employed men. [22]

Panel B of Table 7 shows that there is much less evidence of a change in the cohort slope of earnings ability for college educated men. In contrast to the non-college educated results in Panel A, the results for those with at least a bachelor's degree vary widely across different specifications. The point estimate for the location of the change in slope vary from 1941 to 1951, and a number of the confidence intervals are quite large. Further the estimated size of the change in cohort slope varies widely and even reverses sign. The model with just age and year fixed effects shown in column 1 implies a small decline in ability beginning with the 1941 cohort — with a slope change of -.0057. In contrast, with the addition of quadratic age polynomials in each year the estimated location of the cohort slope change moves to 1951, and the sign reverses with a size estimate of .0237.

The existence of a cohort decline for those *without* a bachelor's degree and no similar decline for those *with* a bachelor's degree implies a cohort-specific increase in the college-high-school wage gap — likely the same phenomenon documented in Card and Lemieux (2001a). Card and Lemieux (2001a) use census data and pool cohorts and ages into larger 5-age/cohort groupings. They therefore do not detect the sharpness by cohort of the decline in earnings of those without a college degree (and of the wage-gap). The discontinuous nature of the slope change appears hard to reconcile with their explanation based on falling relative supply of the college educated and imperfect substitutability of workers of different ages. For example, it would require workers only a year or two apart in age to be very poor substitutes.

The evidence presented in this paper of a broad decline in health and human capital may imply that a reassessment is needed regarding the cause of cohort patterns in earnings by education — and therefore of changes in the college high-school wage gap more generally. If there was a broad decline in health and human capital, it is not obvious why those with a bachelor's degree would not also have a decline in earnings. One explanation would be that the factor which declined across cohorts is a *substitute* in the labor market with a college education, in other words that a bachelor's degree has a "protective effect" in the labor market against the broader decline in human capital.

As noted above, the decline in earnings for those without a bachelor's degree could conceivably be explained by a large change in the selection of those who earn a bachelor's degree — even if the unconditional ability distribution remained unchanged across cohorts. However, these selection effects would need to be highly unusual. First, note that the share of each cohort who comprise the non-college-educated group *grew* substantially after the late 1940s cohorts, from near 65 percent for the 1946 cohort to near 73 percent by the 1960 cohort. For changing selection to explain the earnings decline for this group, the marginal college-goers induced to not get a college-degree would need to be lower ability than the median "never-taker" — those who would not get a college-degree whether they were born in the late 1940s or the 1960s. That is nearly a quarter of the college-educated men in the 1946 cohort would need to be lower ability than the median man *without* a college degree.

[22] Because average years of schooling *within* the bin of men without a bachelor's degree also exhibits a change in slope I apply the two-stage-least-squares procedure from the previous section. The implied return necessary for schooling alone to explain the decline is .187; much larger than the cross-sectional relationship, and larger than most of the IV estimates reviewed in Card (2001).

Intergenerational infant health by mother's education

I now show evidence of similar change in slope of cohort effects for maternal health, as measured by infant birth weight, at many levels of maternal education, including exactly 12 years of education and exactly 16 years of education.

I first apply the age profile differencing methodology to infant birth weight for the two maternal education levels which comprise the largest share of the population: exactly 12 and 16 years of schooling. I use reported mother's years of schooling in the natality detail files, and focus on mean birthweight to improve precision.[23] Figure 8 shows for these two groups of births the average of the difference between the age profile in a given year and the age profile one year earlier.

Panels A and B each reveal a clear mean shift pattern, implying a discontinuous change in the cohort slope of maternal health conditional on education for those with exactly 12 years of education and exactly 16 years of education. The size of the mean shift for both groups appears to be approximately 7 grams — similar to the size shown in Figure 4 for the full sample of mothers. The decline in maternal health *conditional* on education therefore appears to be similar in size to the *unconditional* decline, by this measure and for these groups.

Appendix Table A6 shows that the piecewise linear cohort effect method similarly detects robust evidence of a change in the cohort slope of maternal health for mothers with exactly 12 and 16 years of education. Across specifications of the age-by-year control function, the location of the estimated slope change is either the 1946 or 1947 cohort for both groups. The estimated slope changes across specification are all negative for both groups, and all tests for the existence of a slope change have implied p-values lower than .001.

Table 8 applies the piecewise linear cohort effect method to 5 educational categories, and shows evidence of a similar change in the cohort slope of maternal health across the maternal educational distribution. I apply the model based on Equation 2 with age and year fixed effects separately for the following 5 categories of maternal education: less than high school, high school, some college, 4 years college, and 5+ years of college. For each of the education levels the models detect a change in cohort slope between the 1946 and 1948 cohorts. The size of the change in slope are all negative — implying a relative decline in cohort health — and vary from -.99 grams for the high school group to -3.31 for the some college group. Based on the bootstrap-based test of existence, all the changes in slope are significant. Appendix Table A7 shows that controlling for a separate quadratic-in-age in each year suggests declines in cohort slope at a similar location and at least twice the magnitude, for all groups except those with 5+ years of college.

The above results show strong evidence of cohort declines in maternal health *conditional* on mother's education across the education distribution. These results would rule out an explanation in which a change in the supply of education alone has driven maternal health declines, if that change in supply affected

[23] In addition to the reduced cell-size, note that the incidence of low birthweight births is much lower for highly educated mothers. Mother's education is missing for a non-trivial fraction of mothers, however the cohort decline in infant health is of similar size in the restricted sample of births to mothers with non-missing education information. Mother's education is only recorded beginning in 1969. I further restrict the sample to include mothers over the age of 22, the years 1969 to 1990, and the maternal cohorts 1938 to 1960.

individuals with differing latent health equally. Further, a selection based explanation would require that those who were induced to reduce their education level in the later born cohorts were less healthy than the average mother in the *lower* educational category to which they fell. As described above the changes in cohort educational shares were quite large, so this would imply a large portion of mothers of higher education had lower latent health than the average mother of a lower educational category.

One final piece of evidence against an "education-only" explanation is the different timing by cohort of the declines in women's college graduation rate and of the conditional maternal health declines. Recall that Panel C of Appendix Figure A6 clearly shows that the share of women with a bachelor's degree continued to increase until the 1951 cohort. This later change in the cohort slope for this outcome is confirmed with estimation of piecewise linear cohort effect models, in both the CPS and natality detail files. Recall that the declines in maternal health for those with exactly a bachelor's degree, some college, and exactly HS all declined precisely and sharply beginning with the 1947 or 1948. Reconciling this differential timing would require a complex selection story in which there was a sudden change in trend near the 1947 cohort in the relationship between latent health and bachelor's degree attainment, but no corresponding change in the trend in the total share of population who earn a bachelor's degree, until four cohorts later. This would require that the post-1947 change in selection was "non-monotonic" in the sense of Imbens and Angrist (1994) — that some individuals were induced to get a college degree after 1947, while others who would have previously were induced to *not* earn a college degree.

7. Decline widespread among native-born

Having presented evidence of a decline in cohort health and human capital beginning for cohorts born after 1947, I now consider heterogeneity in this decline across demographic groups. Importantly, the decline appears concentrated among those born in the United States and is not evident for foreign-born U.S. residents. The decline is otherwise remarkably widespread, cutting across racial lines and geography. These findings suggests that the cohort declines may have been caused by an early-life factor which was *specific* to the United States, which had a widespread impact across the country.

A. Only evident for native-born

Given the possibility that the cohort differences documented above may have originated early in life, a natural question is whether they are evident for individuals born outside the United States to the same extent as those born in the United States. In this section, I address this question for the case of maternal health, because the detailed natality data include mother's place of birth. I find that the cohort decline is not evident for mothers who were born outside of the United States, while it is consistently estimated across specifications for those born in the United States. This suggests that a plausible cause of the cohort decline would be a change in an early life factor, specific to the United States. It also — by showing a null cohort result — suggests that the detection of cohort effects is not a mechanical result of model misspecification.

Panel A of Figure 9 shows cohort effects separately for foreign and native-born mothers, based on

additively-separable age-period-cohort models.[24] As for the earlier figures, the models are based on Equation 1, and assume no age-by-period interactions. The shape of cohort effects for native-born have a clear piecewise linear pattern, with a large change in slope at the 1947 cohort — consistent with a decline in maternal health after that year, relative to trend. In sharp contrast, the cohort effects for foreign-born are nearly linear and exhibit no change in slope. This suggests that there was no decline in maternal health for mothers born outside the United States.

Panels B and C reveals similar evidence from applying the age-profile-differencing methodology to the two groups of mothers. Panel C shows the average of the differenced age profiles for U.S. born mothers, and reveals clear visual evidence of a mean shift from the 1947 to the 1948 cohort, consistent with a large decline in maternal health. Panel D on the other hand shows no evidence of a mean shift for the foreign-born mothers. The differenced outcomes for this group are smooth through the 1947 cohort. The lack of a mean shift for the foreign-born does not appear to be due to a lack of precision: the differenced outcomes are slightly noisier for this group than for the U.S. born, but not significantly so.

Next, I estimate models with piecewise linear cohort effects separately for foreign and native-born mothers. For both groups, I examine the robustness to including different age-by-year interactions, from allowing none, to including separate quadratic, cubic, or quartic polynomials in age in each year. While a change in the slope of cohort effects occurring near the 1947 or 1948 cohort is consistently and precisely estimated for the native-born; results for the foreign-born vary across specifications, change sign, and in some specifications the bootstrap test suggests a trend break may not exist.

Panel A of Table 5 shows these results for mothers born in the United States. Across all specifications of the age-by-period control function the location of the estimated change in cohort slope varies from only the 1947 to the 1949 cohort. The estimated size of the change in slope also varies little, ranging from -5.2 grams with no controls to -7.3 when I include quadratic age-by-year controls. When I include a separate cubic or even a quartic in age in each year, the estimated size of the slope change fall between these two estimates. Further, the bootstrap-based test for the null of no change in slope implies a p-value of less than .001 for all specifications.

Panel B shows analogous results for mothers born outside of the United States, and paints a much different picture. In contrast to the native-born results in Panel A, these results vary widely across different specifications, the estimated sizes of the change in slope are generally smaller in magnitude and even change sign. The point estimate for the location of the change in slope vary only slightly more than for the native-born, ranging from 1943 to 1951, but a number of the confidence intervals are quite large. Further the estimated size of the change in cohort slope varies from 6.6 to -3.2 grams. That is, the sign of the change in cohort slope is unstable — some models suggest improvement in health while others suggest a decline in health. Further, for three of the four models the implied p-values for the test of existence of a trend break are greater than .04. For the model with a quadratic in age it is .139. This suggests that there is not strong evidence in the data that a change in cohort slope actually exists for the foreign-born.

[24] I drop the first two years of data, 1968 and 1969, because mother's place of birth is not recorded in those years. Using the remaining years I estimate models separately for mothers born within one of the 50 states, and for those born outside of them.

I also use decennial Census data to examine cohort declines in occupational status for the foreign and native-born separately. I use the 1970, 1980, 1990 and 2000 censuses and use the share of employed men in white-collar occupations as the dependent variable. I again estimate the piecewise linear cohort effect models separately for foreign and native-born workers. These results should likely be treated with more caution than the maternal health results for 2 reasons. First, the Census data only records outcomes every 10 years — hampering the ability to ditinguish cohort effects from age-by-year interactions. Second, they comprise a smaller sample of the population — and therefore include particularly small samples of the foreign-born.

With this caveats in mind, Appendix Table A3 reveals a quite similar pattern in white-collar status to that shown above for maternal health. For native-born men there is a robust evidence across specifications of a change in the slope of cohort effects at the 1946 cohort, of negative sign and large in magnitude — between -.07 and -.017.[25] In contrast, estimates for foreign-born men are highly variable across specification. The baseline estimate with no age-by-year interactions suggests a similar sized decline to that for the native-born but starting with the 1941 cohort.[26] However, adding polynomial age-by-year interactions yields estimates of a *positive* change in slope — ie. improvements in occupational status relative to the prior trend — either at the 1932 cohort or the 1956 cohort.

B. Otherwise widespread

The decline is otherwise remarkably widespread across demographic groups born in the United States. For example, the declines are quite similar across the four Census Regions, and are generally evident in all racial groups and in urban and rural areas. A notable exception is the mortality of black men, which shows evidence of improvements for post-1952 cohorts. However, black women exhibit evidence of cohort declines in mortality similar to those for whites, and black men and women exhibit cohort declines in all the other outcomes. The earnings declines for black men are significantly *larger* than those for whites. These findings suggest that the cohort declines were likely caused by an early-life factor, widespread within but specific to the United States.

By race

Table 3 examines potential heterogeneity by race for four of the main outcomes. I estimate the piecewise-linear cohort effect models based on Equation 2, separately for different racial groups. Given the smaller sample sizes I report estimates from models including just age fixed effects and year fixed effects as controls. I also restrict the sample to exclude cohorts born after 1960, to avoid focusing on the known improvements for blacks born after the Civil Rights Act documented in Almond and Chay (2006), Chay, Guryan and Mazumder (2009, 2014).[27]

[25] This magnitude is similar to estimates from models using the CPS-MORG for the full population.

[26] This decline could potentially be explained by changes in immigration policy in the 1960s leading to lower-skilled imigrant entrants after that year.

[27] The CPS data includes individuals of Hispanic-origin as a separate category, while the vital statistics data does not consistently include Hispanic-origin. For the log wage results I therefore estimate models for 4 racial groups: non-Hispanic whites, non-Hispanic blacks, Hispanics, and all other races pooled. For health outcomes, I estimate models for only 3 categories: whites, blacks, and other races (which each include Hispanics).

The cohort-specific decline in earnings ability appears remarkably similar across racial groups. The location of the estimated cohort slope change in models of the median log wage are remarkably similar: at 1946 for whites, 1948 for blacks, and 1949 for Hispanics and other races. Further, the confidence intervals for all of these estimates include the 1947 cohort (though the estimate for the other race group is very imprecise). The size of the change in cohort slope are very similar for whites, Hispanics, and the other race group, at near -.013 log points. The change is notably larger for blacks, nearly double that of the other groups at -.025 log points.

The cohort specific decline in maternal health appears similarly widespread across racial groups. Estimates of the piecewise linear slope change model for the share low birthweight, reveal a change in cohort slope of very similar location and magnitude for whites, blacks, and other races. The estimated location of this change in slope is 1949 for whites and 1947 for blacks, with the two confidence intervals overlapping. The estimated location for other races is 1950, with a very large confidence interval. The sizes of the decline are also very similar across the 3 racial groups: .10 for whites, .08 for blacks, and .07 for the other race group.

The change in cohort slope for log mortality are also mostly similar across racial groups, with black men standing out as a notable exception. For white men and women I estimate changes in slope located at the 1944 and 1950 cohorts respectively, with magnitudes of .023 and .022. For black men however, I estimate a change in cohort slope occurring at the 1956 cohort and a negative change in slope of -.0294, suggesting that health *improved* significantly starting with cohorts born after 1956. The estimated changes in slope for the remaining groups — black women, and men and women of other races — are similar in location and sign to that of whites though smaller in magnitude: at .012, .013, .010 respectively. The exceptional experience of black male mortality by cohort is therefore inconsistent with the otherwise broad pattern, across outcomes and races, and appears worthy of further study.

By Region

Table 4 examines analogous heterogeneity by place of residence, and shows that similar declines in cohort health and human capital are estimated across each of the four Census Regions. I again estimate the piecewise-linear cohort effect models based on Equation 2, separately for each Census Region.[28] Each Census Region has been exposed to different labor market demand shocks over the period, and has likely been exposed to different factors — with different timing and magnitude — more broadly. If the above cohort decline estimated at the national level reflect misspecified age-by-year interactions, one may expect these to vary by region and therefore lead to different cohort patterns in each region.[29]

Results for models of the median log wage suggest little variation across regions in the decline in earnings ability across cohorts. The estimated location of the change in cohort slope varies from only 1946 to 1948 across the four regions, and all of the confidence intervals include 1947. The size of the cohort decline varies from only -.0194 in the Midwest to -.0139 in the South — with the Northeast and West in between at -.017 and -.014 respectively.

[28] I age control for age fixed effects and year fixed effects. I return to the unrestricted sample including cohorts born up to 1965.

[29] Also, given that migration is costly and therefore place of residence is somewhat sticky, if there are large differences in the cohort declines by *region of birth* this analysis by region of residence should reveal attenuated estimates of these differences.

The results for share low birthweight suggest the decline in maternal health was similarly widespread. Again the estimated cohort at which the slope change occurs varies little across the four regions: with point estimates ranging from 1947 to 1949, and all confidence intervals including either 1947 or 1948. For this outcome the South now exhibit the largest declines with a change in slope of .13. That for the Northeast, Midwest, and West are slightly smaller at .12, .11, and .09 respectively. The standard errors of .01 suggest the difference between the South and West is likely only marginally significant.

The results for the log mortality of both men and women also suggest a widespread decline in health. For these outcomes I use a control function which includes a separate quadratic-in-age in each year — in addition to the age and year fixed effects included above. As described above I use mortality estimates from the United States Mortality Database.[30] The results are shown in the bottom half of Table 4 and reveal remarkably similar estimates across the 4 Census Regions. For men's log mortality the location of the change in cohort slope is estimated to be precisely the 1946 cohort for *all 4 regions*, with each 99 percent confidence interval including only that cohort. For women, the location estimates range from only the 1948 to the 1950 cohort across regions. The estimated size of the change in slope is also quite similar across regions, as well as across the 2 sexes. The size estimates for men range from .0284 in the Northeast to .0341 in the South. Those for women range from 0.0269 in the West to .0331 in the South. [31]

Appendix Table A6 shows evidence that the cohort decline in wages is also remarkably similar for urban, suburban, and rural workers. I estimate the piecewise-linear cohort effect models based on Equation 2, separately for the median wage of men residing in rural, suburban, and urban areas. For each group, the estimated location of the change in cohort slope is 1947. The size of the decline is also remarkably, similar with the slope change size estimates varying only from -.014 for rural workers to -.016 for urban workers.

8. Candidate root cause: post-WWII respiratory decline

The above results document a widespread decline in the health and human capital of Americans born after 1947 or 1948. I now turn to suggestive evidence of a potential root cause of this decline: a postwar decline in the respiratory health environment in the United States.

My hypothesis is that there was a broad decline in the respiratory health "environment" after 1946 or 1947 — which increased respiratory mortality for infants and adults in the short run — and had a lasting effect on the health of cohorts who were in utero or infancy during that period. The key piece of evidence for a broad decline in respiratory health is that mortality from particularly respiratory causes began to increase simultaneously for both infants *and* elderly adults near 1947. The hypothesized change in the respiratory health environment could conceivably have been caused by a decline in air quality, for example due to postwar industrial growth or increased driving. Alternatively, the risk of respiratory infections may have

[30] These data are only available as period-based life-tables, and I therefore define cohort as the year minus age. Note therefore that this cohort definition is slightly different than that for the other mortality results.

[31] Note that these regional results for log mortality are not fully robust to different control functions. However, using the quadratic control function the findings of a widespread decline at or near the late 1940s cohorts hold at even smaller geographic scale. For example, each of 9 Census Divisions have quite similar estimates.

increased for reasons unrelated to air quality.

Before presenting evidence supporting this hypothesis, I briefly present evidence that appears inconsistent with a few competing hypotheses. First, Appendix Figure A8 shows that mothers and fathers educational levels were increasing rapidly for the 1947 to 1970 cohorts. If anything cross-cohort increases in the share of each cohort whose mothers and fathers had a Bachelor's degree *accelerated* after the late 1940s cohorts. Therefore, selection on observable family background, as measured by parental education, cannot explain the decline in health and human capital. Second, Appendix Figure A10 shows that rapid improvements in the infant mortality rate beginning in the 1930s suddenly *slowed* beginning in the late-1940s. Similarly, Appendix Figure A9 shows that increases in the the share of each cohort surviving to age 18 began to decelerate after the late-1940s. These patterns rule out a single-index selection-based explanation — in which technology developed after 1947 suddenly allowed more unhealthy infants to survive to adulthood.

The baby boom began with a sharp increase in the sizes of the 1946 and 1947 cohort — making the casual observation that the baby boom "caused" the health and human capital decline initially appealing. However, the trend in cohort size shown, in Appendix Figure A9, is quite different than the piecewise linear pattern in health and human capital. Cohort size increased sharply in 1946 and 1947. It then stagnated for a few cohorts before increasing gradually until the mid-1950s cohorts. Cohort size then declined rapidly after the 1961 cohort. A simple "cohort-crowding" theory therefore cannot generate the pattern in health and human capital I've documented (Bound and Turner, 2007). To match the observed patterns a theory would have to posit a complex lagged effect of cohort size on health and human capital.

A number of factors suggest a change in the respiratory health environment in infancy is a plausible cause of the cohort declines documented above. I showed that the change in cohort slope in maternal health was concentrated among those born in the United States, and not evident for foreign born mothers. The decline in test scores at age 17 suggests that the cause of the declines predated labor market entry. Therefore, a plausible candidate cause of the declines would be some early life factor unique to the United States which changed trend suddenly in the late 1940s. Further, past evidence suggests that a decline the respiratory health of infants, or of air quality specifically, would have long-run effects on exposed infants health and human capital as adults. A large literature reviewed in Currie and Almond (2011) presents evidence of effects of infant health on educational attainment, health, and labor market outcomes. A number of papers also find evidence of long-run effects of infant exposure to respiratory diseases and air pollution specifically. For example, Isen et al. (2017) find that reductions in particulate matter in utero following the Clean Air Act led to gains in earnings when exposed infants were adults. [32]

Rapid improvements in the infant mortality rate and the overall age-adjusted death rate began to noticeably stall near-1950, prompting a number of reports and articles. In the 1960s, a series of 12 reports from the National Center for Health Statistics examined the contributions of different causes of death, documented variation within the United States, compared the United States experience to that of other countries, and

[32]Bhalotra and Venkataramani (2015) find evidence of long-run gains in adult outcomes driven by reductions in pneumonia exposure in infancy in the United States after 1937. Almond (2006) found long run effects of in-utero exposure to the 1918 Influenza Pandemic. Almond and Chay (2006), Chay et al. (2009), and Chay et al. (2014) find long-run effects of infant health gains for African Americans in the 1960s South — which particularly included reductions in pneumonia mortality — led to gains in test scores, educational attainment, earnings, and maternal health — with an intergenerational effect on infants.

considered the possible root cause.[33] The slowdown was considered surprising in the context of a booming postwar economy and rapid improvements in medical technology. But it appears to have been largely forgotten as large declines in mortality rates began again in the late 1960s and early 1970s (Crimmins, 1981).

Notable in the reports are sharp trend breaks in respiratory mortality of both infants and adults, leading to increased respiratory mortality after the late 1940s. Based on a detailed examination of death certificates, the reports also note a particular increase in deaths from a syndrome known alternatively as "Infant Respiratory Distress Syndrome", "Hyaline Membrane Disease", or "Surfactant Deficiency Disorder." It is a syndrome common particularly among premature infants, in which the lungs do not produce enough of a film known as surfactant. In healthy infants, surfactant covers the air sacs in the lungs, helping to keep them open. Therefore, infants suffering from Infant Respiratory Distress Syndrome (IRDS) have trouble breathing — which can lead to infant death in some cases. Below, I replicate and extend evidence of these increases in respiratory mortality, with a particular focus on IRDS.

Figure 10 shows evidence of a postwar increase in respiratory mortality for infants and elderly adults. As described above all data is transcribed from printed Vital Statistics volumes. Panel A shows a clear trend break in infant morality from all respiratory diseases. I include deaths from pneumonia and influenza, as well as a category called "Other Respiratory Diseases." This category includes deaths for IRDS. The mortality rate from these respiratory diseases fell rapidly from more than 10 deaths per 1,000 births in 1937 to nearly 4 deaths per 1,000 in 1946. The pace of decline then noticeably slows and by 1958 the respiratory infant mortality rate is nearly identical to that in 1946. Unfortunately, deaths from IRDS were reclassified into a broader category between 1959 and 1966, creating a break in the series. However, by 1967 when IRDS, was reclassified total respiratory infant mortality was slightly *higher* than it had been in the 1940s.

In Panel B, I attempt to provide the best picture of increases in infant mortality from IRDS based on the cause of death groupings which are reported in the printed volumes. The results in Panel B are consistent with IRDS barely featuring as a cause of infant death in the 1930s and early 1940s, and beginning a rapid increase around 1946 which continued until the early 1970s. By the 1960s, IRDS was one of the leading causes of infant death. As noted above, this cause was classified into a different cause of death grouping in the 1950s — from "Other respiratory diseases" to "Diseases peculiar to early infancy" — and then was reclassified back to "Other respiratory diseases" after 1968.

Infant mortality from "Other respiratory diseases" was less than 1 death per 1,000 births in 1933 and declined slightly until 1946. Starting after that year it began to increase until 1956 — when IRDS was removed from that classification. During the subsequent period between 1957 and 1968 when IRDS was grouped in the category "Diseases peculiar to early infancy", deathins in this category increased rapidly . Finally in 1966 when IRDS is returned to "Other respiratory disease" the level of mortality in that grouping is nearly 6 times higher than it was in 1946 — at nearly 3 deaths per 1,000 births.

Panel C and D show that beginning near 1946 there was also an increase in *adult* mortality from respiratory causes, including Chronic Obstructive Pulmonary Disease (COPD). Panel C shows cause-specific

[33] See Moriyama (1964); Shapiro et al. (1965); Shapiro and Moriyama (1963); Moriyama (1961, 1966, 1960); Klebba (1971); Chase (1967)

mortality for white men age 65 to 69 for 3 cause of death groupings.The series in red squares show mortality from "Other respiratory diseases" which primarily includes causes of death such as Bronchitis and Emphysema, which would now be grouped as Chronic Obstructive Pulmonary Disease (COPD). Mortality from this cause grouping is remarkably flat at less than 40 deaths per 1,000 between 1933 and the mid-1940s. It then begins to increase gradually after the late 1940s. These increases continue unabated until the 1960s — during which time mortality from this cause has more than quadrupled from less than 40 *to 200* deaths per 1,000.

The series denoted by green crosses shows mortality from pneumonia and influenza, while blue circles show mortality from all respiratory diseases — combining the other two series. Pneumonia and influenza mortality exhibit rapid declines after 1937, which are generally credited to the development and use of Sulfa drugs (Jayachandran et al., 2010; Bhalotra and Venkataramani, 2015). The declines in mortality from these causes however suddenly stop in the late 1940s. Interestingly, the combination of the trends in these two groupings results in a U-shaped pattern in *total* respiratory mortality. Mortality from all respiratory diseases declined rapidly from the late 1930s to the late 1940s, from nearly 400 deaths per 1,000 to less than 150, but then reversed trend and began to increase — climbing back to more than 300 deaths per 1,000 by the mid-1960s. The declines in the first period were driven by rapid declines in pneumonia and influenza, while the change in trend was the result of a combination of these declines stalling *and* a rapid increase in previously rare mortality from chronic respiratory diseases.

Panel D shows that the mortality increase from chronic respiratory diseases began concurrently for a number of older age groups, which suggests it was driven by a period-based cause, rather than cohort factors such as smoking habits. It shows the log mortality rate from respiratory diseases excluding pneumonia and influenza for white men in 3 age groups: 60-64, 65-69, and 70-74. The respiratory log mortality series for each of these ages is nearly flat from 1933 to the mid-1940s. Each of the series then exhibits a trend break near 1946 and increases approximately linearly through the 1960s. The coincident turning point of these mortality series at different ages suggest that they were caused by a change in the respiratory health "environment" near 1946. Alternatively, a change in cohort smoking patterns would leave a staggered imprint, with the 60-64 series increasing first, and the other series beginning to increase 5 and 10 years later respectively.

One concern with the evidence above is that the increase in mortality from the listed respiratory cases of death may reflect a change in coding, rather than a change in *actual* respiratory mortality and therefore health. There were large revisions in the International Classification of Diseases coding system used to classify deaths in 1939, 1949, 1958, and 1968 (Hetzel, 1997). It is consistent with an actual decline in respiratory health therefore that mortality gradually increases in periods between these changes, where official coding guidelines remained unchanged, and that the series do not appear to exhibit discrete changes at these dates. Further, it seems unlikely that a trend break in the coding of infant and adult respiratory deaths related to two very different conditions — COPD and IRDS — occurred coincidentally.

Additionally, the trend break in IRDS mortality coincides approximately with a stagnation in infant mortality from *all causes*. Further, the infant mortality rate in the United States diverged suddenly in 1946

from that of other countries, which had previously had a similar infant mortality rate. Appendix Figure A10 shows these patterns. This sudden divergence of the infant mortality rate in the United States from that in other countries is also consistent, under some assumptions, with a decline in infant health in the United States. In particular, assume as in Almond and Chay (2006) and Bozzoli et al. (2009) that infant death depends on latent health and a threshold of viability, and allow the distribution of latent health and the threshold to both vary across years. Then the above patterns would be consistent with a trend break and subsequent decline in latent infant health in the United States if: i) the year-specific threshold of viability is the same in the United States and the other countries, and ii) mean latent health continues to follow a linear trend in the other countries.

Finally, Appendix Figure A11 shows that the sex ratio at birth — though quite variable across nearby years — begins a general declining trend after the late 1940s. The sex ratio at birth, the ratio of male to female live births, is often viewed as a proxy for fetal health because male fetuses are generally less robust than female fetuses. Therefore this pattern is also consistent with a decline in infant health beginning in the late-1940s.

In summary, the above section shows evidence of postwar increases in respiratory mortality of both infants and adults which would be consistent with a broad decline in the respiratory health environment in that period. Previous evidence suggests that the respiratory environment in utero and early in life can have a lasting effect on adult health, cognitive ability, and human capital broadly defined. Further the broad decline in adult outcomes, documented in the other sections of this paper, begins with cohorts born after 1947. Therefore the cohorts who exhibit declining adult outcomes are also the cohorts who would have been exposed in utero or infancy to a declining respiratory health environment. I therefore suggest that the broad cohort declines in health and human capital may be the result of the lasting impact of early-life exposure to a postwar decline in the respiratory health environment.

9. Conclusion

In this paper I present evidence of a precisely timed and broad decline in the health and human capital of cohorts of Americans beginning with those born in 1947, relative to the prior trend. This decline appears to have predated labor market entry and to have played a key role in: test score and education declines in the 1960s and 1970s, increases in the low birthweight rate beginning in the mid-1980s, wage stagnation since the 1970s, and recent mortality increases of white Americans. The cohort decline has even had an intergenerational effect, though its effect on the health of mothers and their infants.

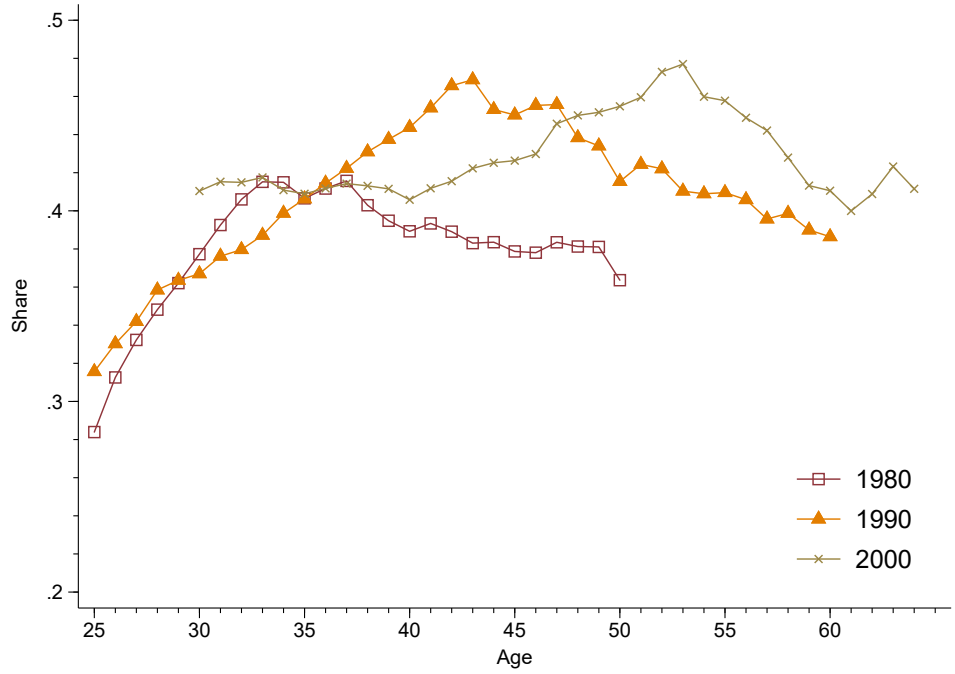
Something appears to have gone deeply wrong — by at least age 17 — for cohorts of Americans born after 1947. I suggest the decline may have been caused by a postwar decline in the respiratory health environment. Future research should continue the search for the decline's underlying cause.

The documented health and human capital decline may also be evident in other outcomes not examined in this paper. Secular changes over time in outcomes such as labor force participation or medical spending may be the result, at least in part, of cohort differences in health and human capital rather than year-specific

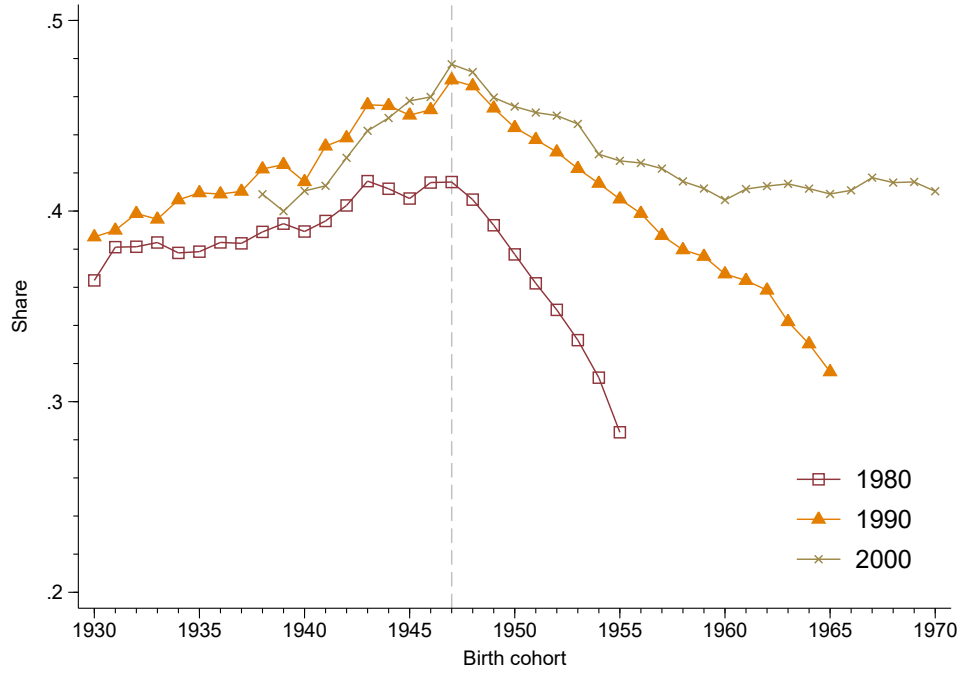
factors. Further, the decline in health and human capital may have interacted in important ways with year-specific shocks — such as the early 1980s recessions. A full accounting of the broad and lasting impact of the decline which I've documented will require additional research.

Figure 1: Share of employed men working in white-collar occupation

A: By age

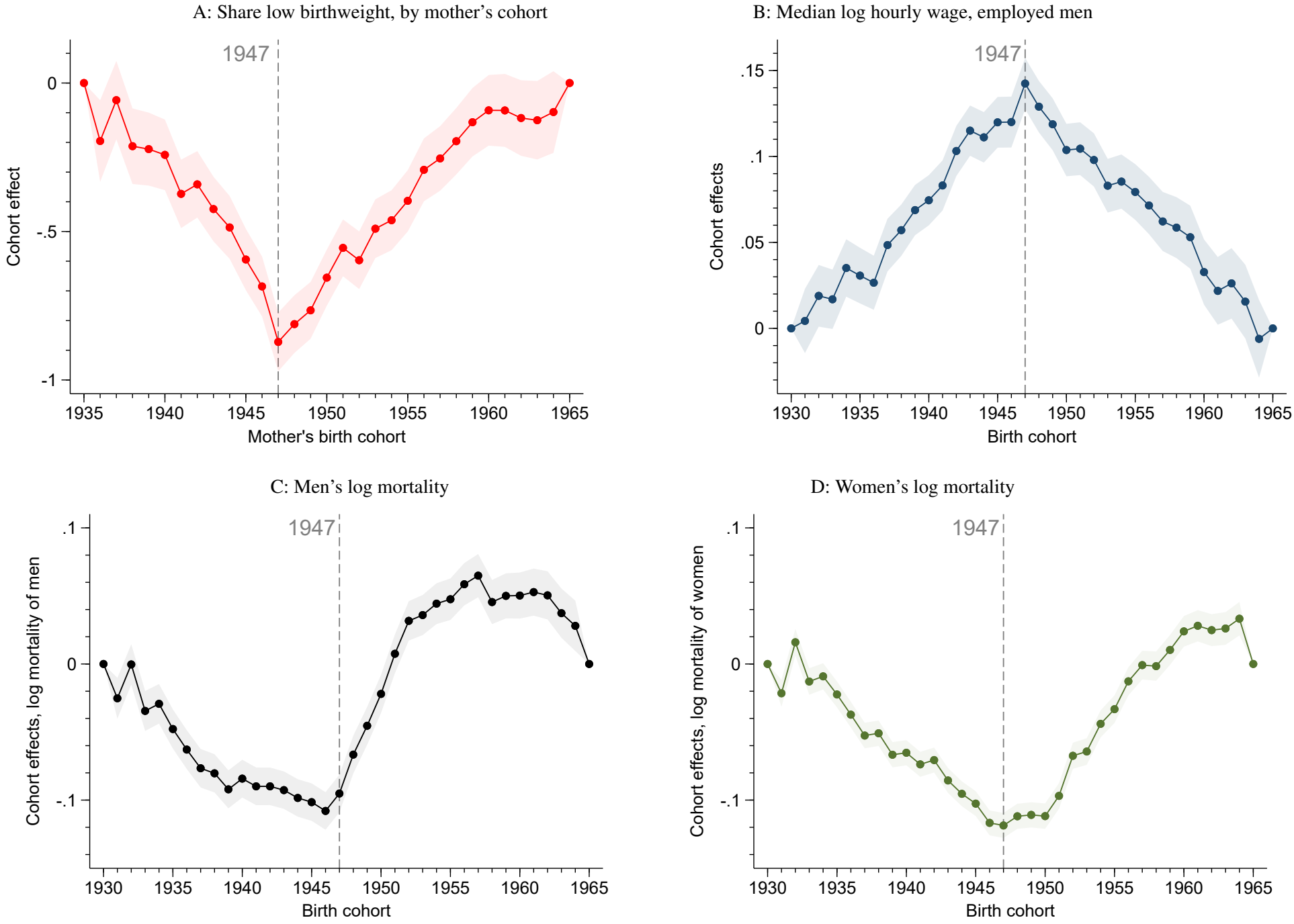


B: By birth cohort



The first panel shows the share of employed men working in a white-collar occupation by singe year of age, measured in the decennial Census IPUMS microdata for 1980, 1990 and 2000. The second panel recasts the same series by year of birth. white-collar occupations are those in the "managers/professionals/technicians/finance/public safety" category in Autor and Dorn (2013).

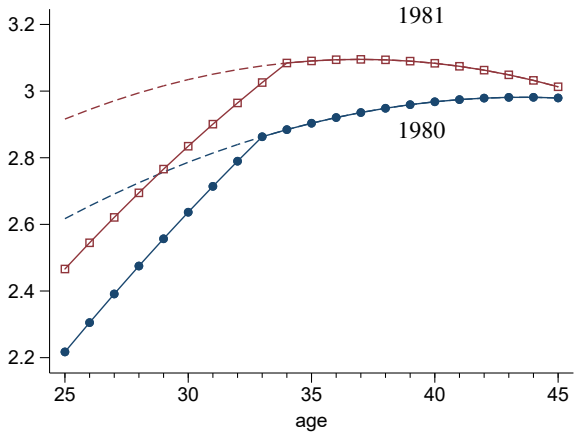
Figure 2: Evidence of cohort decline in 4 outcomes — detrended cohort effects from age-period-cohort models



Each panel plots detrended cohort effects from estimation of age-period-cohort models based on Equation 1. Panel A is based on vital statistics natality microdata, 1968-1990, mothers age 18-40, who were born from 1935-1965. Panel B is based on CPS-MORG data, 1979-1993, and includes men age 25-54, who were born from 1930 to 1965. Panel C and D are based on data from the Human Mortality Database, and include 1975-2015, ages 25-85, cohorts born from 1930 to 1965.

Figure 3: Identification of slope change in cohort effects by differencing age profiles — hypothetical example with slope change at 1947 cohort

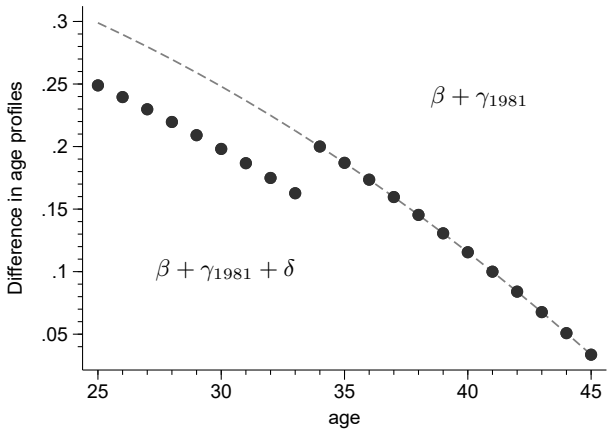
A: Age profiles in 1980 and 1981



Slope changes of size δ at 1947 cohort:

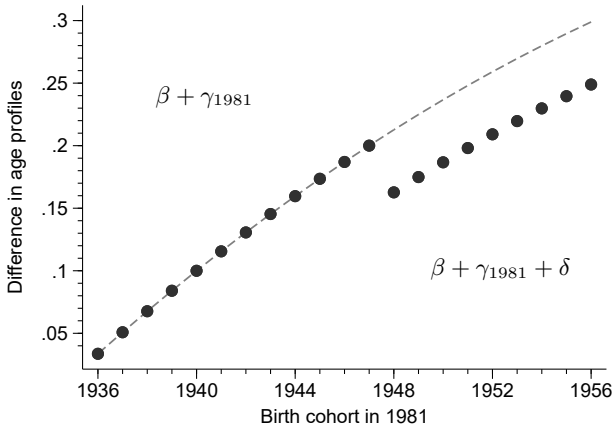
- *age 34 in 1981*
- *age 33 in 1980*

B: Difference in age profiles between 1980 and 1981



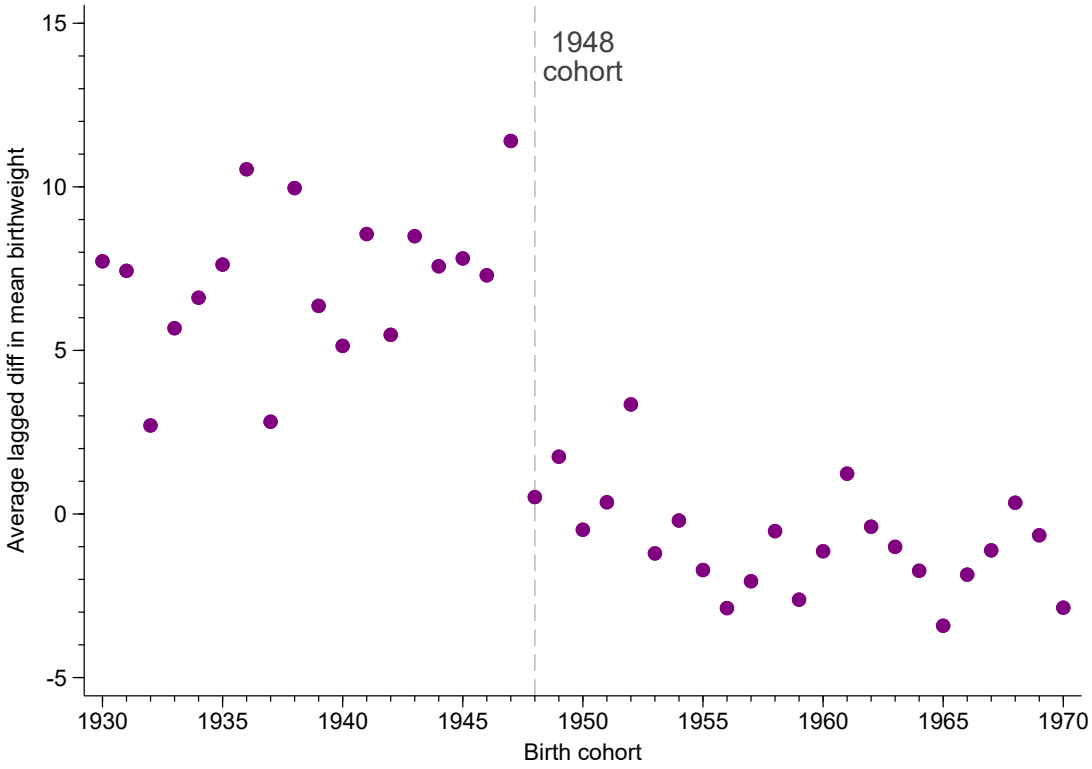
Mean shift of size δ at age 34

C. Difference in age profiles between 1980 and 1981
— indexed by birth cohort in 1981



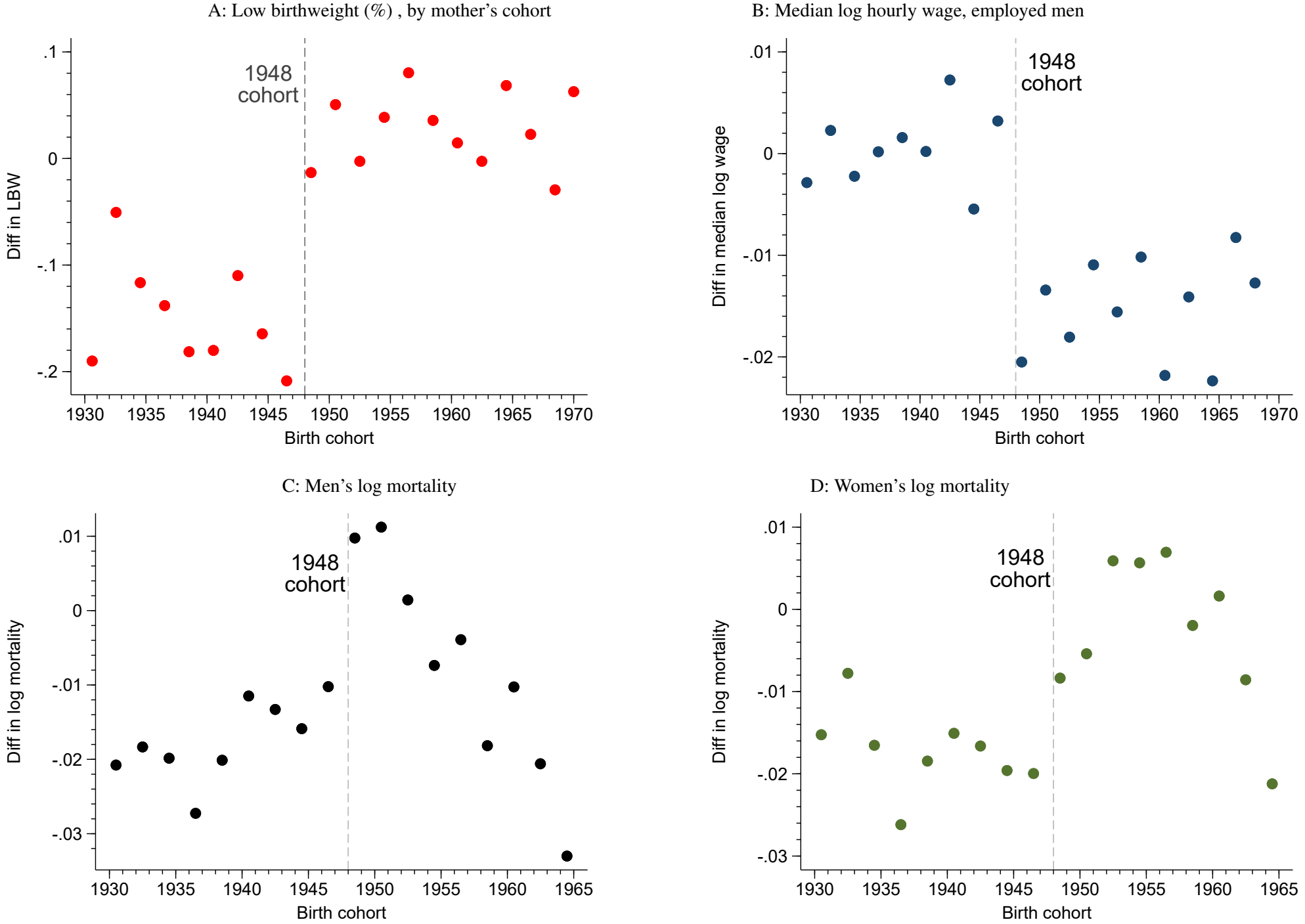
Mean shift of size δ at 1948 cohort

Figure 4: Evidence of cohort decline in intergenerational infant birthweight — average of differenced age profiles across years



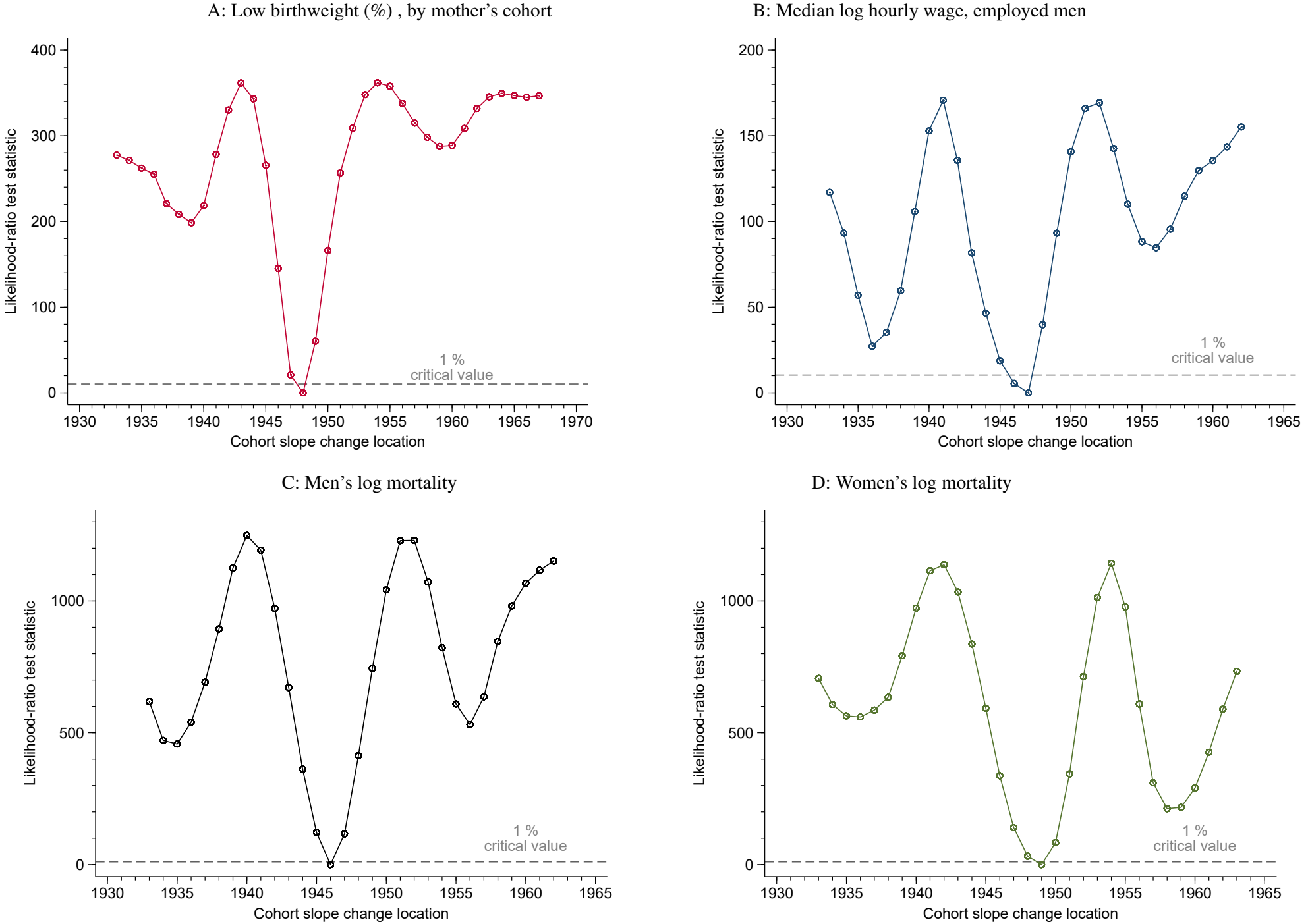
This figure provides a visual test of whether there is a slope change in the cohort effects of maternal health — as proxied by the mean birthweight of a mother's infant. The underlying data is vital statistics natality microdata, 1968-1995, mothers age 18-40. Each point shows the average across all years for a given cohort, c of mothers of: the difference between i) the mean birthweight of infants born to mothers in cohort c, at age a, and in year p, and ii) the mean birthweight of infants born to mothers in cohort $c-1$, at age a, and in year $p-1$. The large "mean shift' from the 1947 to the 1948 cohort is consistent with a large change in the *slope* of cohort effects at the 1947 cohort, in the context of the model outlined above.

Figure 5: Evidence of cohort decline in 4 outcomes — average of differenced age profiles across years



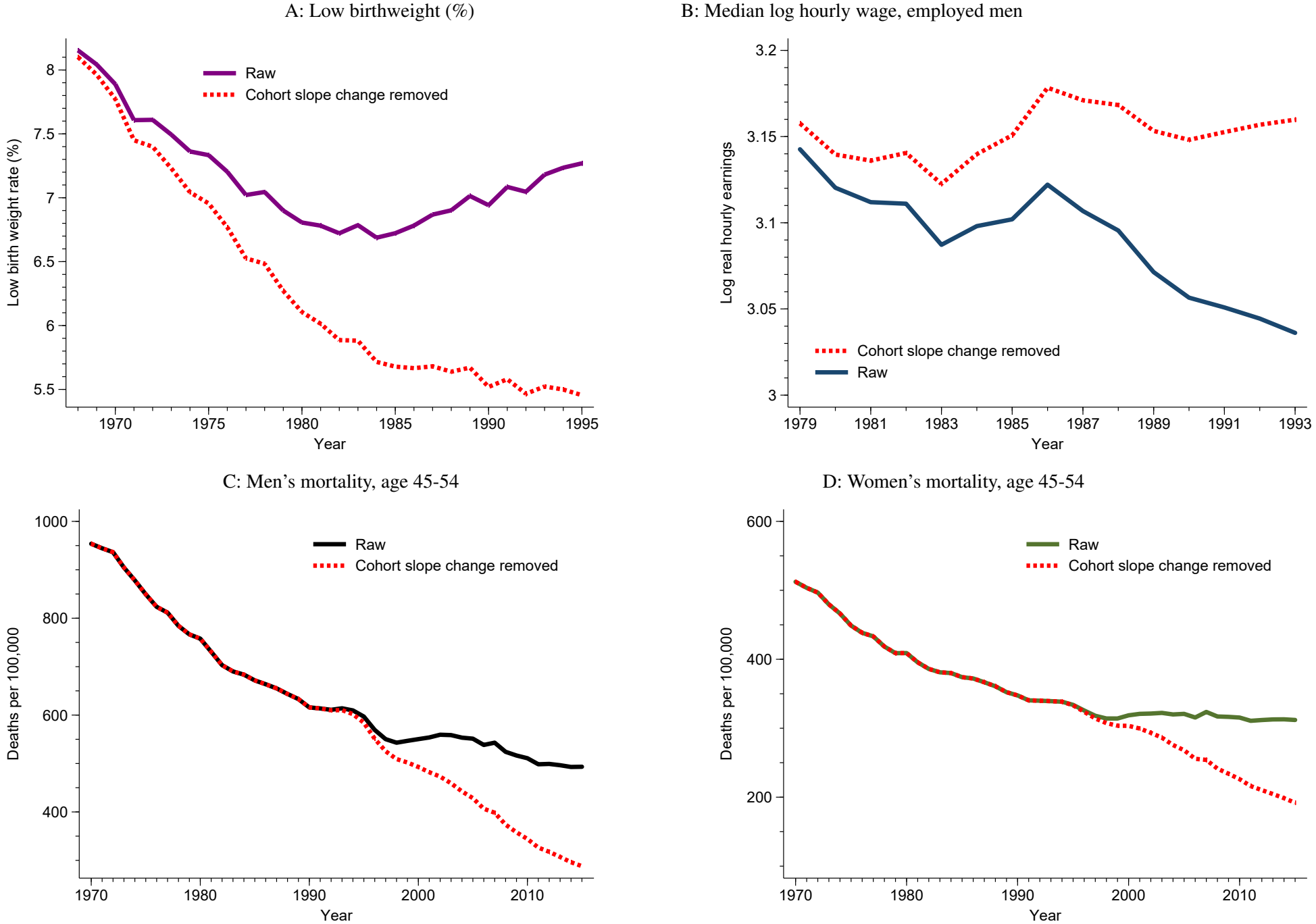
This figure provides a visual test of whether there is a slope change in cohort effects. Panel A is based on vital statistics natality microdata, 1968-1995, mothers age 18-40. Panel B is based on CPS-MORG data, 1979-1993, and includes men age 25-54, who were born from 1930 to 1965. Panel C and D are based on data from the Human Mortality Database, and include 1975-2015, ages 25-85, cohorts born from 1930 to 1965. Each point shows the average across all years for a given 2-cohort-bin of: the difference between i) the outcome of cohort c, at age a, and in year p, and ii) the outcome of cohort $c-1$, at age a, and in year $p-1$. The large "mean shift' from the 1947 to the 1948 cohort is consistent with a large change in the *slope* of cohort effects at the 1947 cohort, in the context of the model outlined above.

Figure 6: Hansen-type estimation of cohort at which decline begins



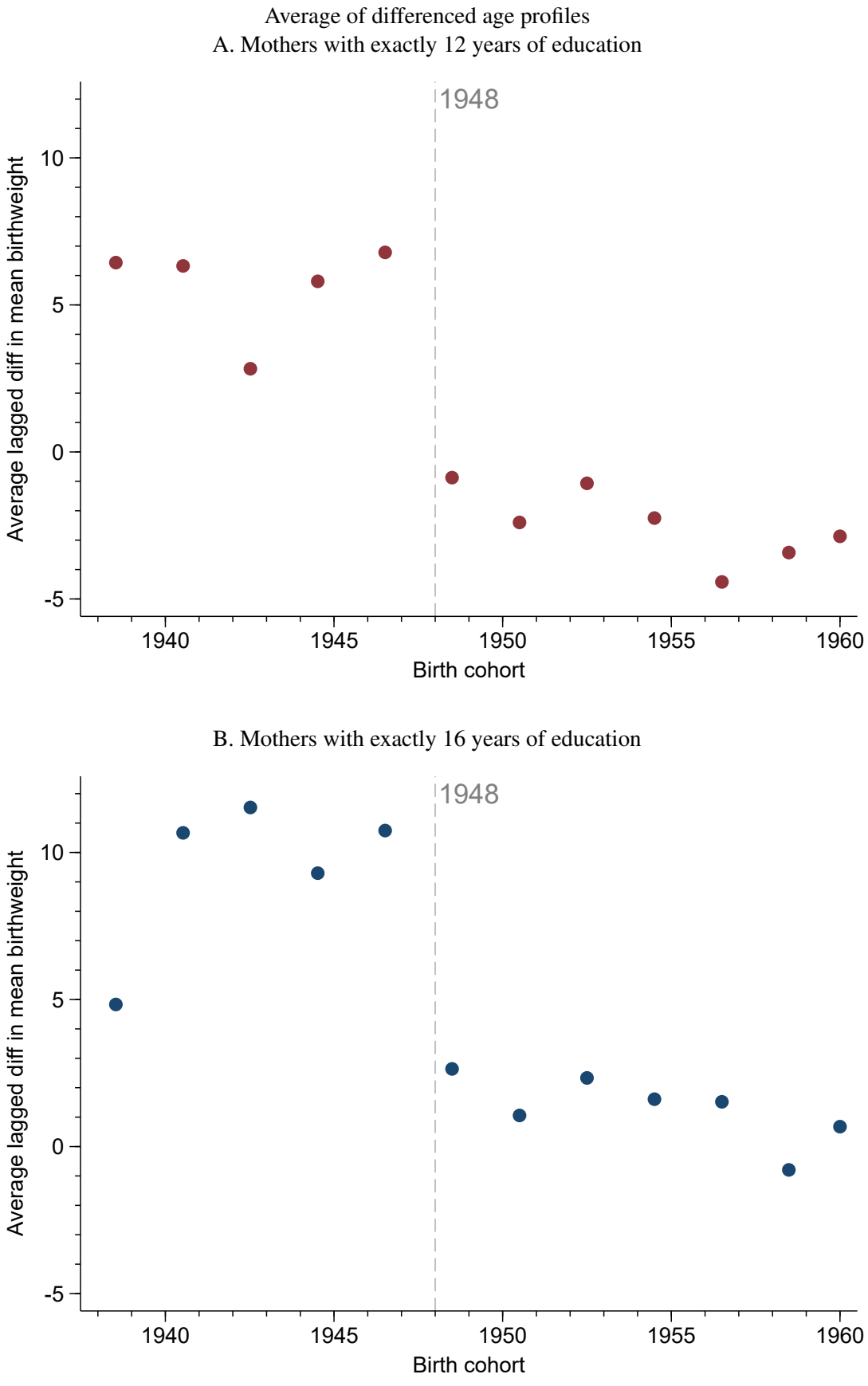
This figure shows estimation and inference of the location at which a cohort decline begins based on the model in Equation 2, with age fixed effects, year fixed effects, and separate quadratic-in-age in each year included as controls. Each plot shows the likelihood-ratio test statistic suggested in Hansen (1996), for a model with the listed outcomes as the dependent variable. The point estimate of this location is the cohort with the minimum value of the LR-test statistic. The 99 % confidence region is those cohorts with a LR-test statistic below the critical value shown with a dashed grey line. Data is the same as for Figure 5.

Figure 7: Impact of cohort decline on year-over-year declines



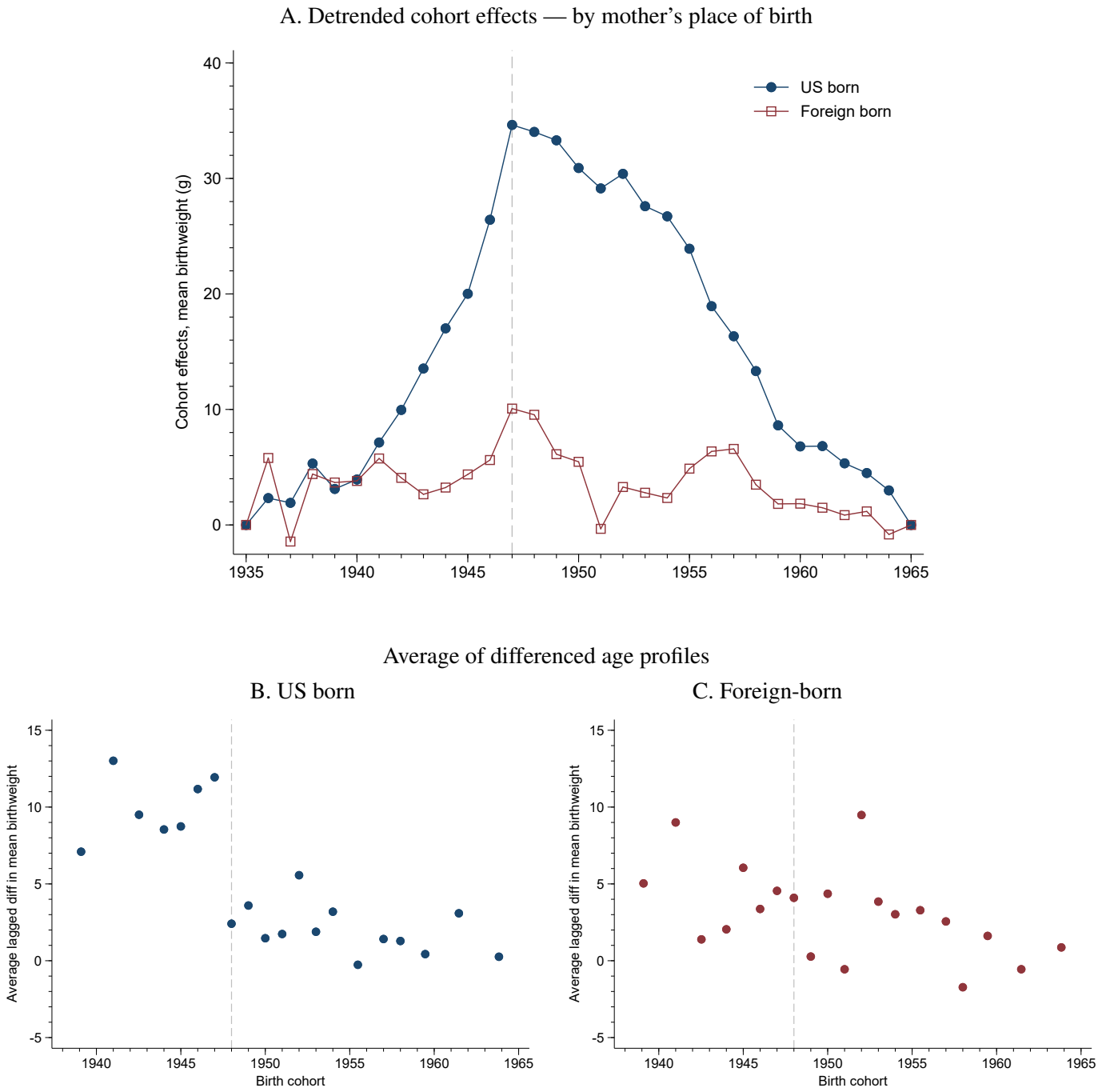
This figure shows simulated counterfactual year-over-year trends in 4 outcomes had the change in slope of cohort effects not occured, ie. if the pre-slope-change cohort trend had continued. The slope change estimated based on the model in Equation 2 is subtracted from each observation. These transformed data are then used to calculate each of the listed outcomes, which are plotted as the dashed red line. The same outcome based on the untransformed, raw data is plotted as a solid line. Panel A shows the percent of infants born at low birthweight (<2500 g). Panel B shows the average across men age 25-54 of median wages for single age-bins. Panel C and D show mortality rates of men and women age 45-54, age-adjusted assuming a uniform population distribution by age. Data sources for each panel are the same as in Figures 2, 5, and 6.

Figure 8: Intergenerational infant health decline — conditional on maternal education



This figure provides a visual test of whether there is a slope change in the cohort effects of the listed outcomes. Both panels are based on vital statistics natality microdata, 1969-1995, mothers age 22-38. Panel A is restricted to mothers with exactly 12 years of schooling. Panel B is restricted to mothers with exactly 16 years of schooling. Each point shows the average across all years for a given 2-cohort-bin of: the difference between i) the outcome of cohort c, at age a, and in year p, and ii) the outcome of cohort $c-1$, at age a, and in year $p-1$. The large "mean shift' from the 1947 to the 1948 cohort is consistent with a large change in the *slope* of cohort effects at the 1947 cohort, in the context of the model outlined above.

Figure 9: Intergenerational infant health decline only evident for native-born



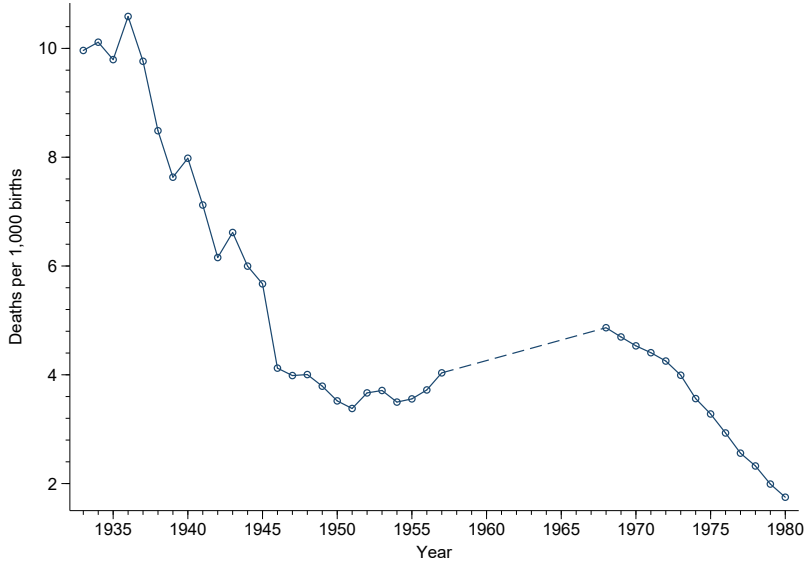
This figure analyzes outcomes separately for mothers born in one of the 50 United States or D.C., and those born outside of the United States. All panels are based on vital statistics natality microdata, 1970-1990, including mothers age 18-40, who were born from 1935-1965. Panel A plots detrended cohort effects from estimation of age-period-cohort models based on Equation 1 — separately for the two groups of mothers. Panel B and C show a visual test of whether there is a slope change in the cohort effects of the listed outcomes, for US born and Foreign-born mothers respectively. Each point shows the average across all years for a given 2-cohort-bin of: the difference between i) the outcome of cohort c, at age a, and in year p, and ii) the outcome of cohort $c-1$, at age a, and in year $p-1$. The large "mean shift' in Panel B from the 1947 to the 1948 cohort is consistent with a large change in the *slope* of cohort effects at the 1947 cohort, for US born mothers. The absence of such a mean shift in Panel C suggests no such slope change exists for foreign-born mothers.

Figure 10: Post-WWII increase in respiratory mortality,

Infants

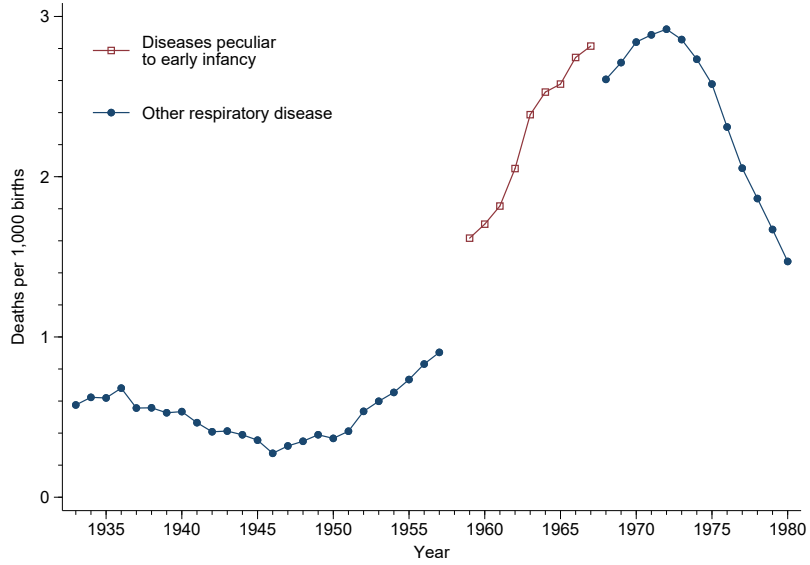
A: Infant mortality rate, all respiratory diseases

Includes Pneumonia, Influenza, and "Other Respiratory Diseases" — which includes IRDS



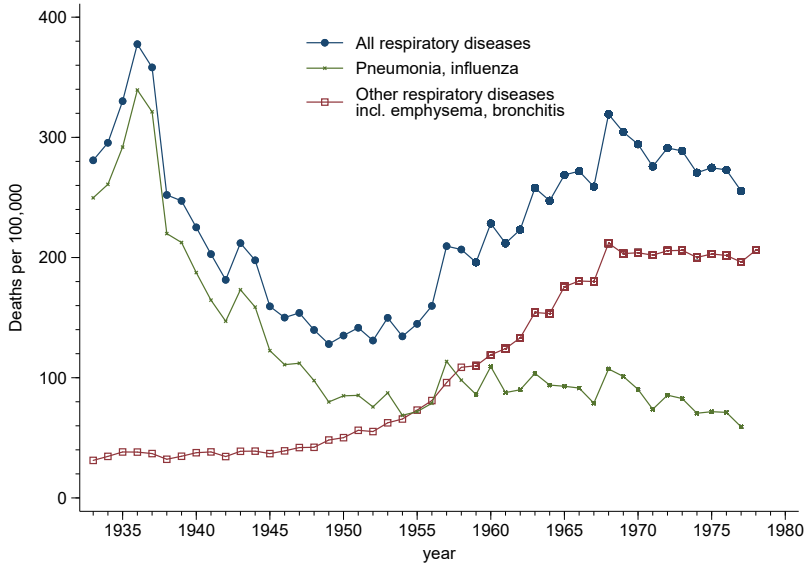
B: Infant mortality rate

cause of death categories which include IRDS

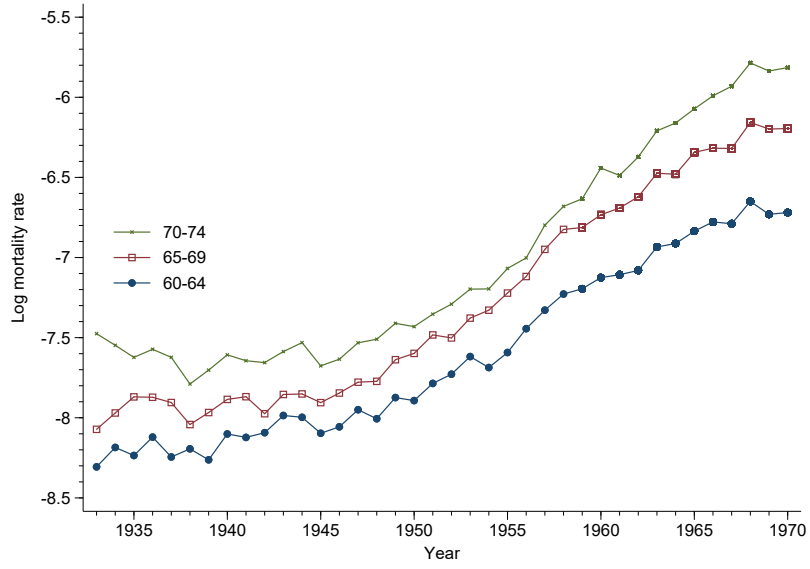


Adult white men

C: Respiratory mortality, age 65-69



D: Log mortality, respiratory causes excluding pneumonia and influenza



Death counts from 1933 to 1958 were digitized from tables in historical vital statistics volumes. Those from 1959-1980 are calculated from the Multiple Cause of Death File. I combine these with population estimates from the Census Bureau, and counts of births from the vital statistics volumes, to form the listed mortality rates.

Table 1: Evidence of cohort decline in 5 outcomes — mean shift in differenced age profiles by birth cohort

controlling for separate quadratic-in-age in each year

	Mean shift		
	Size	Location	Existence
	δ	λ	*p-value*
Intergenerational infant health			
Mean birth weight (g)	-7.49	1948	$< .001$
	(0.99)	$[1948, 1948]$	
Low birthweight (%)	0.196	1948	$< .001$
	(0.043)	[1948, 1948]	
Labor market			
Median log wage	-0.020	1948	.001
	(0.005)	[1948, 1948]	
Log mortality			
Men	0.030	1947	$< .001$
	(.002)	[1947, 1947]	
Women	0.027	1951	$< .001$
	(.003)	[1951, 1951]	

Each row shows the results of estimation of a model based on Equation 5, with the "differenced age profiles" of the listed outcome as the dependent variable. All models are estimated by least squares, following the approach outlined in Hansen (1999, 2000). The column titled "Size" reports the estimated size of mean shift in the differenced outcomes, δ, with the standard error in parentheses. The column titled "Location " reports the cohort at which the mean shift is estimated to occur, with a 99 $\%$ confidence interval in brackets calculated by inverting a likelihood ratio statistic. The column titled "Existence" reports a p-value from an F-type test, based on 1000 bootstrap samples, for the null hypothesis that no mean shift in the differened outcomes occurs, ie. that cohort effects are linear. Intergenerational infant health results are based on vital statistics natality microdata, 1968-1995, mothers age 18-40 who were born between 1930 to 1970. Labor market results are based on CPS-MORG data, 1979-1993, and includes men age 25-54, who were born from 1930 to 1965. Log mortality results are based on data from the Human Mortality Database, and include the years 1975-2015, ages 25-85, cohorts born from 1930 to 1965.

Table 2: Evidence of cohort decline in 5 outcomes — piecewise linear cohort effect models

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

	Change in cohort slope		
	Size	Location	Existence
	δ	λ	*p-value*
Intergenerational infant health			
Mean birth weight (g)	-6.35	1948	$< .001$
	(0.35)	$[1948, 1948]$	
Low birthweight (%)	0.241	1948	$< .001$
	(0.014)	[1948, 1948]	
Labor market			
Median log wage	-0.016	1947	$< .001$
	(0.001)	[1946, 1947]	
Log mortality			
Men	0.029	1946	$< .001$
	(.001)	[1946, 1946]	
Women	0.031	1949	$< .001$
	(.003)	[1949, 1949]	

Each row shows the results of estimation of a model based on Equation 2, with the listed outcome as a dependent variable. All models are estimated by least squares, following the approach outlined in Hansen (1999, 2000). The column titled "Size" reports the estimated size of the change in cohort slope δ, with the standard error in parentheses. The column titled "Location " reports the cohort at which the slope change is estimated to occur, with a 99 % confidence interval in brackets calculated by inverting a likelihood ratio statistic. The column titled "Existence" reports a p-value from an F-type test, based on 1000 bootstrap samples, for the null hypothesis that no change in slope occurs, ie. that cohort effects are linear. Intergenerational infant health results are based on vital statistics natality microdata, 1968-1995, mothers age 18-40 who were born between 1930 to 1970. Labor market results are based on CPS-MORG data, 1979-1993, and includes men age 25-54, who were born from 1930 to 1965. Log mortality results are based on data from the Human Mortality Database, and include the years 1975-2015, ages 25-85, cohorts born from 1930 to 1965.

Table 3: Implied causal effect of schooling on earnings, maternal health, and mortality assuming no change in unobservables across cohorts

		Change in cohort slope	
	Outcome	Years of schooling	Implied causal effect
	(reduced-form)	*(first stage)*	*(2sls)*
Unconditional			
Median log wage	-0.016	-0.115	0.138
	(0.0004)	(0.003)	(0.004)
Share low birthweight	0.16	-0.173	-0.92
	(0.02)	(.027)	(.09)
Male log mortality	.026	-.10	-.251
	(0.001)	(.002)	(.009)
Female log mortality	.031	-.072	-.434
	(0.001)	(.001)	(.014)
Conditional on no Bachelor's degree			
Median log wage	-0.0709	-0.013	0.187
	(0.0005)	(0.002)	(0.008)

This table reports the results of two-stage-least-squares estimation of the causal effect of a year of schooling on earnings and health, under the assumption that unobservables are unchanged across cohorts. Each row shows the results for the listed outcome. The column titled "Outcome (reduced-form)" reports the estimated size of a slope change in cohort effects, δ, from a model based on Equation 2 for the listed outcome. The column titled "Years of schooling (first stage)" reports the estimated size of a slope change in cohort effects from estimation of a similar model with years of schooling on the left-hand side, and with the location of the slope change, λ, set equal to the estimated location in the reduced-form and treated as known. The column titled "Implied causal effect (2sls)" uses these estimates to form a two-stage-least-squares estimate of the causal effect of years of schooling on the outcome. Median log wage results are based on CPS-MORG data, 1979-1993, and includes men age 25-54, who were born from 1930 to 1965. Share low birthweight results are based on vital statistics natality microdata, 1968-1995, mothers age 18-40 who were born between 1930 to 1970. Log mortality results are based on data from the Human Mortality Database, and include the years 1975-2015, ages 25-85, cohorts born from 1930 to 1965.

Table 4: Change in slope of cohort effects for median log earnings of employed men — separately for those with and without a Bachelor's degree

robustness to varying age-by-year control function

	(1)	(2)	(3)	(4)
Panel A: Without Bachelor's Degree				
Size	-0.0132	-0.0124	-0.0183	-0.0207
	(0.0005)	(0.0015)	(0.0029)	(0.0031)
Location	1947	1947	1947	1947
	[1947, 1947]	[1947, 1947]	[1947, 1947]	[1947, 1947]
P-value for existence	$< .001$	$< .001$	$< .001$	$< .001$
Panel B: With Bachelor's degree				
Size	-0.0057	0.0237	-0.0265	-0.0235
	(0.0010)	(0.0028)	(0.0034)	(0.0056)
Location	1941	1951	1943	1943
	[1938, 1943]	[1950, 1952]	[1942, 1951]	[1936, 1962]
P-value for existence	$< .001$	$< .001$	$< .001$	.002
Year FEs	Yes	Yes	Yes	Yes
Age FEs	Yes	Yes	Yes	Yes
Quadratic-age-by-year	No	Yes	No	No
Cubic-age-by-year	No	No	Yes	No
Quartic-age-by-year	No	No	No	Yes

Each column shows the results of estimation of a model based on Equation 2, with the listed outcome in single age-by-year bins as the dependent variable. All models are estimated by least squares, following the approach outlined in Hansen (2000). Results are based on CPS-MORG data, 1979-1993, and includes men age 25-54, who were born from 1930 to 1965. The row titled "Size" reports the size of the change in slope of cohort effects, δ, with the standard error in parentheses. The row titled "Location" reports the estimated cohort at which a trend break occurs, with a 99 $\%$ confidence region in brackets calculated by inverting the likelihood ratio statistic. The row titled "P-value for existence" reports a p-value from an F-type test, based on 1000 bootstrap samples, for the null hypothesis that no change in slope occurs, ie. that cohort effects are linear.

Table 5: Evidence of cohort decline conditional on educational attainment — piecewise linear cohort effect models of intergenerational infant birth weight

controlling for year FEs and age FEs

	Change in cohort slope		
	Size	Location	Existence
	δ	λ	*p-value*
Maternal education level			
Less than HS	-1.43	1948	0.022
	(0.35)	[1945, 1950], [1958, 1962]	
High school	-0.99	1947	$< .001$
	(0.22)	[1946, 1949]	
Some college	-3.31	1948	$< .001$
	(0.28)	$[1947, 1948]$	
4 years college	-2.15	1946	$< .001$
	(0.43)	[1944, 1948]	
5+ years college	-2.42	1946	$< .001$
	(0.63)	[1941, 1948]	

Each row shows the results of estimation of a model based on Equation 2, with birthweight by mother's birth cohort as the dependent variable and the sample restricted to mothers with the listed level of education. All models are estimated by least squares, following the approach outlined in Hansen (2000). Results are based on vital statistics natality microdata, 1969-1985, mothers age 22-38, cohorts born from 1938 to 1965. The column titled "Size" reports the estimated size of the change in cohort slope δ, with the standard error in parentheses. The column titled "Location " reports the cohort at which the slope change is estimated to occur, with a 99 % confidence interval in brackets calculated by inverting a likelihood ratio statistic. The row titled "Existence" reports a p-value from an F-type test, based on 1000 bootstrap samples, for the null hypothesis that no change in slope occurs, ie. that cohort effects are linear.

Table 6: Cohort decline concentrated among those born in the United States — piecewise linear cohort effect models of intergenerational infant birth weight

robustness to varying age-by-year control function

	(1)	(2)	(3)	(4)
Panel A: Mothers born in US				
Size	-5.209	-7.292	-5.879	-7.059
	(0.167)	(0.438)	(0.650)	(0.839)
Location	1949	1948	1947	1947
	[1949, 1949]	[1947, 1948]	[1947, 1947]	[1947, 1947]
P-value for existence	$< .001$	$< .001$	$< .001$	$< .001$
Panel B: Mothers born outside US				
Size	1.888	-1.669	-3.213	6.565
	(0.351)	(0.815)	(1.194)	(1.591)
Location	1943	1947	1947	1951
	[1941, 1947]	[1938, 1962]	[1938, 1962]	[1951, 1951]
P-value for existence	$< .001$	.139	.096	.041
Year FEs	Yes	Yes	Yes	Yes
Age FEs	Yes	Yes	Yes	Yes
Quadratic-age-by-year	No	Yes	No	No
Cubic-age-by-year	No	No	Yes	No
Quartic-age-by-year	No	No	No	Yes

Each column shows the results of estimation of a model based on Equation 2, with the listed outcome in single age-by-year bins as the dependent variable. All models are estimated by least squares, following the approach outlined in Hansen (2000). The row titled "Size" reports the size of the change in slope of cohort effects, δ, with the standard error in parentheses. The row titled "Location" reports the estimated cohort at which a trend break occurs, with a 99 $\%$ confidence region in brackets calculated by inverting the likelihood ratio statistic. The row titled "P-value for existence" reports a p-value from an F-type test, based on 1000 bootstrap samples, for the null hypothesis that no change in slope occurs, ie. that cohort effects are linear. Results are based on vital statistics natality microdata, 1970-1995, mothers age 18-40 who were born between 1930 to 1970.

Table 7: Evidence of cohort decline across racial groups — piecewise linear cohort effect models

controlling for age FEs and year FEs

		(1) White	(2) Black	(3) Hispanic	(4) Other races
Median log wage	*Size*	-0.0130	-0.0251	-0.0133	-0.0143
		(0.0005)	(0.0018)	(0.0023)	(0.0036)
	Location	1946	1948	1949	1949
		[1945, 1947]	[1947, 1949]	[1946, 1953]	[1936, 1953]
Low birthweight (%)	*Size*	0.10	0.08		0.07
		(0.005)	(0.01)		(0.03)
	Location	1949	1947		1950
		[1948, 1949]	[1945, 1949]		[1938, 1957]
Log mortality					
Men	*Size*	0.0235	-0.0294		.0103
		(0.0003)	(0.0023)		(0.0012)
	Location	1944	1956		1945
		[1944, 1945]	[1954, 1956]		[1943, 1947]
Women	*Size*	0.0218	0.0125		0.0131
		(0.0005)	(0.0007)		(0.0024)
	Location	1950	1945		1953
		[1950, 1950]	[1944, 1947]		[1948, 1956]

Each column shows the results of estimation of a model based on Equation 2, with the listed outcome as a dependent variable. All models are estimated by least squares, following the approach outlined in Hansen (2000). Intergenerational infant health results are based on vital statistics natality microdata, 1968-1995, mothers age 18-40 who were born between 1930 to 1960. Labor market results are based on CPS-MORG data, 1979-1993, and includes men age 25-54, who were born from 1930 to 1960. Log mortality results are based on data from the Human Mortality Database, and include the years 1975-2015, ages 25-85, cohorts born from 1930 to 1960. The column titled "Size" reports the estimated size of the change in cohort slope δ, with the standard error in parentheses. The column titled "Location " reports the cohort at which the slope change is estimated to occur, with a 99 % confidence interval in brackets calculated by inverting a likelihood ratio statistic. The row titled "Existence" reports a p-value from an F-type test, based on 1000 bootstrap samples, for the null hypothesis that no change in slope occurs, ie. that cohort effects are linear.

Table 8: Evidence of cohort decline across Census Regions — piecewise linear cohort effect models

		(1) Northeast	(2) Midwest	(3) South	(4) West
Median log wage	*Size*	-0.0170	-0.0194	-0.0139	-0.0149
		(0.0009)	(0.0013)	(0.0010)	(0.0012)
	Location	1947	1948	1946	1947
		[1946, 1947]	[1947, 1949]	[1945, 1947]	[1946, 1948]
Low birthweight (%)	*Size*	0.12	0.11	0.13	0.09
		(.01)	(.01)	(.01)	(.01)
	Location	1947	1949	1948	1948
		[1946, 1947]	[1948, 1950]	[1947, 1948]	[1946, 1949]
Log mortality					
Men	*Size*	0.0284	0.0287	0.0341	0.0286
		(0.0016)	(0.0014)	(0.0012)	(0.0015)
	Location	1946	1946	1946	1946
		[1946, 1946]	[1946, 1946]	[1946, 1946]	[1946, 1946]
Women	*Size*	0.0320	0.0288	0.0331	0.0269
		(0.0019)	(0.0019)	(.0016)	(0.0028)
	Location	1948	1950	1950	1949
		[1948, 1949]	[1950, 1950]	[1949, 1950]	[1949, 1949]

Each column shows the results of estimation of a model based on Equation 2, with the listed outcome as a dependent variable. All models are estimated by least squares, following the approach outlined in Hansen (2000). Wages and birth weight include controls for age fixed effects and year fixed effects — mortality also includes a separate quadratic-in-age in each year. The column titled "Size" reports the estimated size of the change in cohort slope δ, with the standard error in parentheses. The column titled "Location " reports the cohort at which the slope change is estimated to occur, with a 99 % confidence interval in brackets calculated by inverting a likelihood ratio statistic. The row titled "Existence" reports a p-value from an F-type test, based on 1000 bootstrap samples, for the null hypothesis that no change in slope occurs, ie. that cohort effects are linear. Intergenerational infant health results are based on vital statistics natality microdata, 1968-1995, mothers age 18-40 who were born between 1930 to 1970. Labor market results are based on CPS-MORG data, 1979-1993, and includes men age 25-54, who were born from 1930 to 1965. Log mortality results are based on data from the Human Mortality Database, and include the years 1975-2015, ages 25-85, cohorts born from 1930 to 1965.

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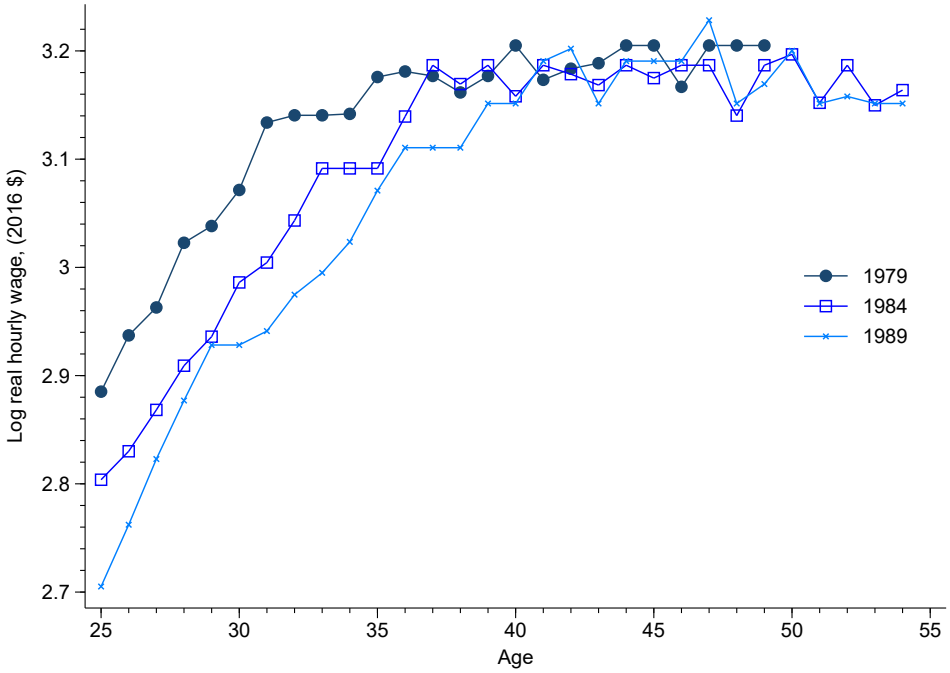
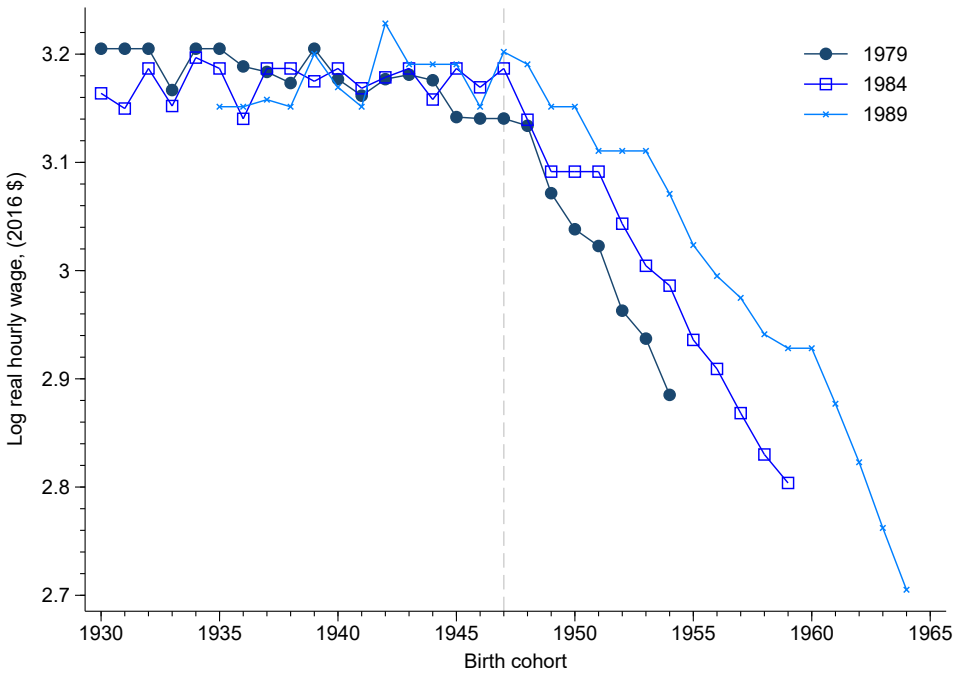
Figure A1: Median hourly wage of men

A: By age



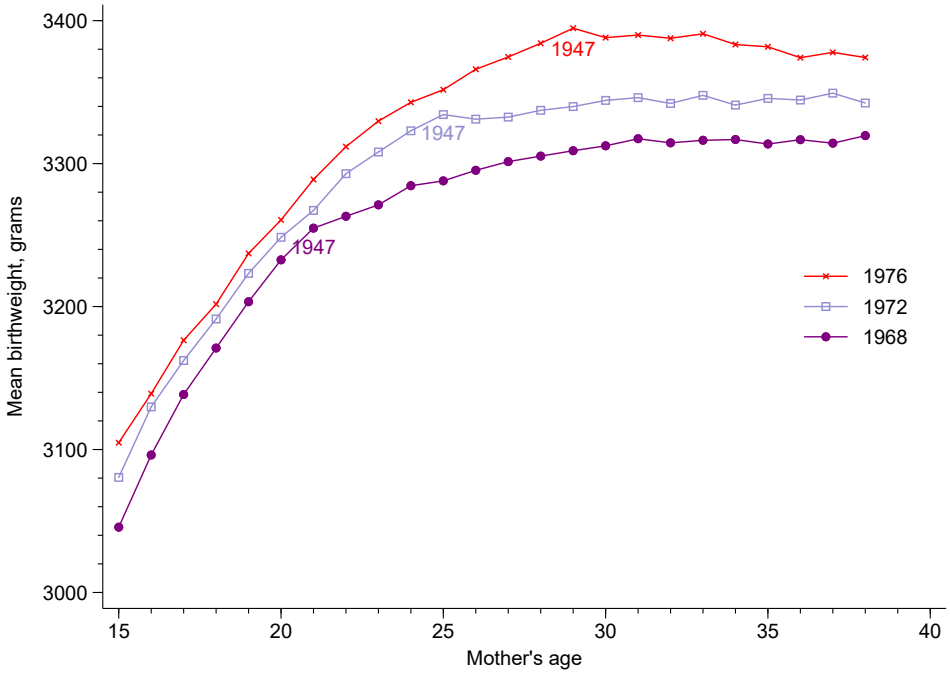
B: By birth cohort



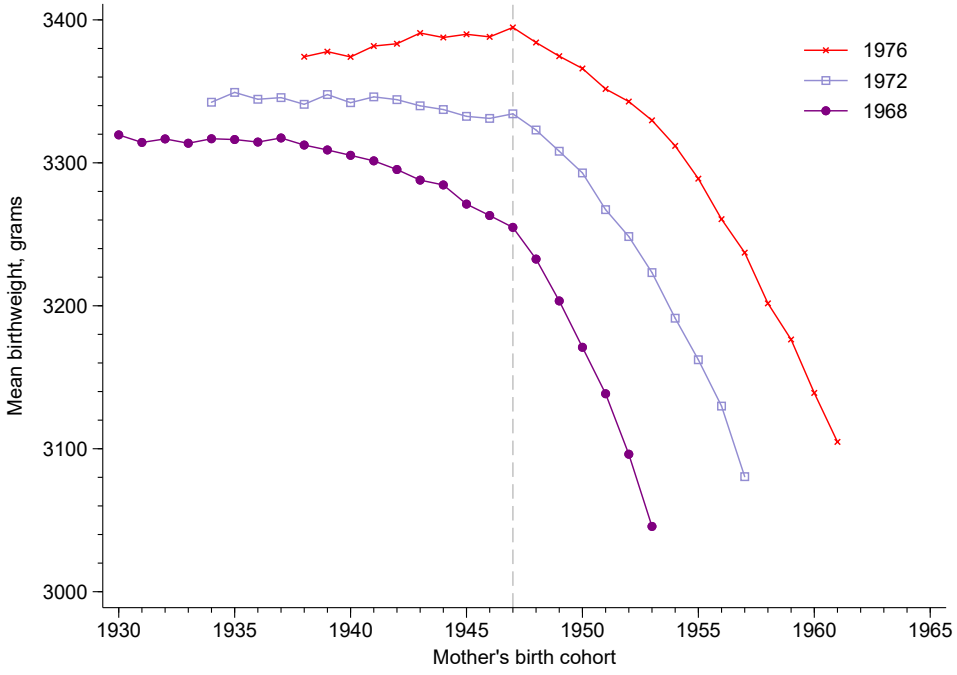
The first panel shows the median real log earnings of employed men by age, measured in the CPS MORG. The second panel recasts the same series by year of birth.

Figure A2: Mean birth weight of infants

A: By mother's age

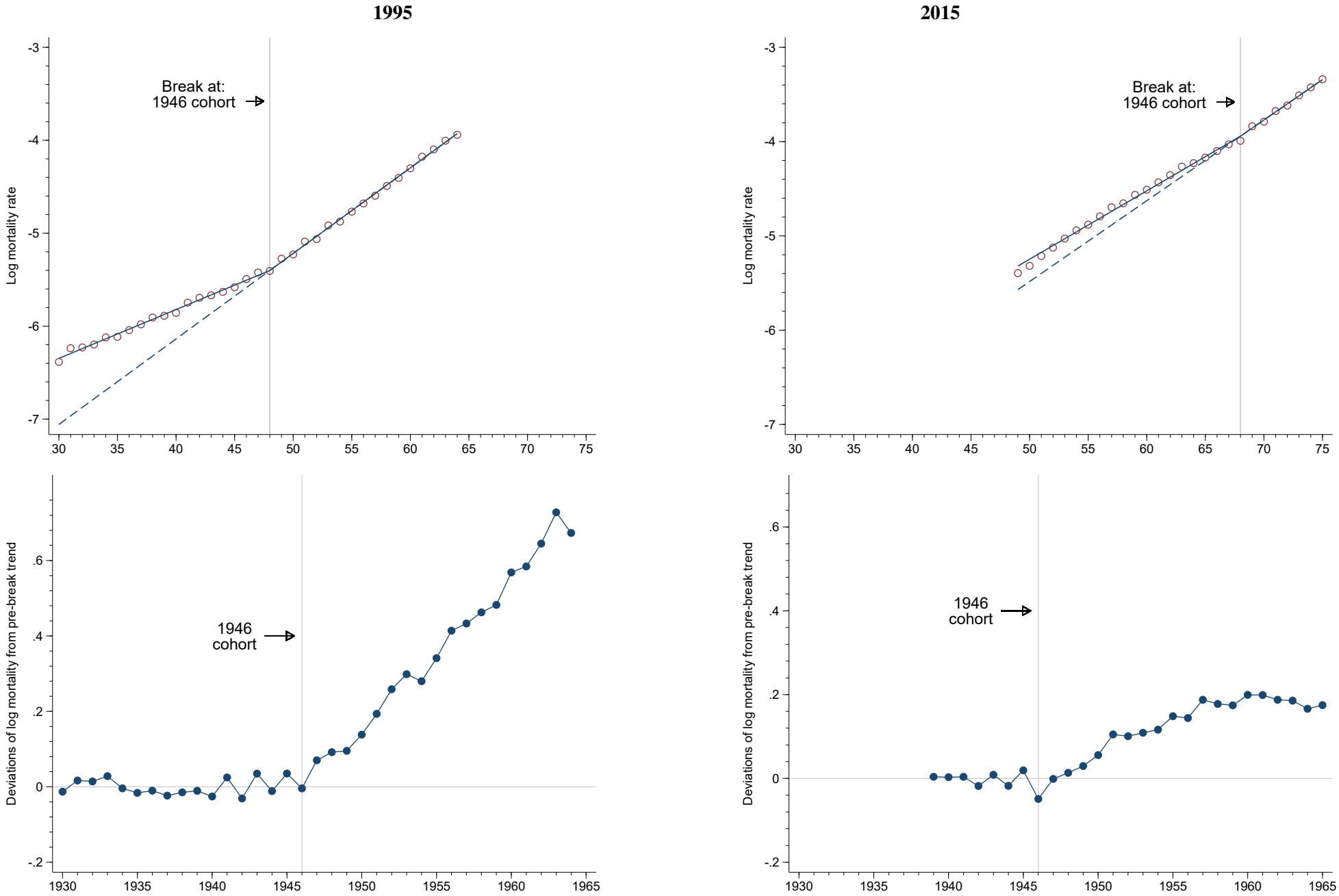


B: By mother's birth cohort



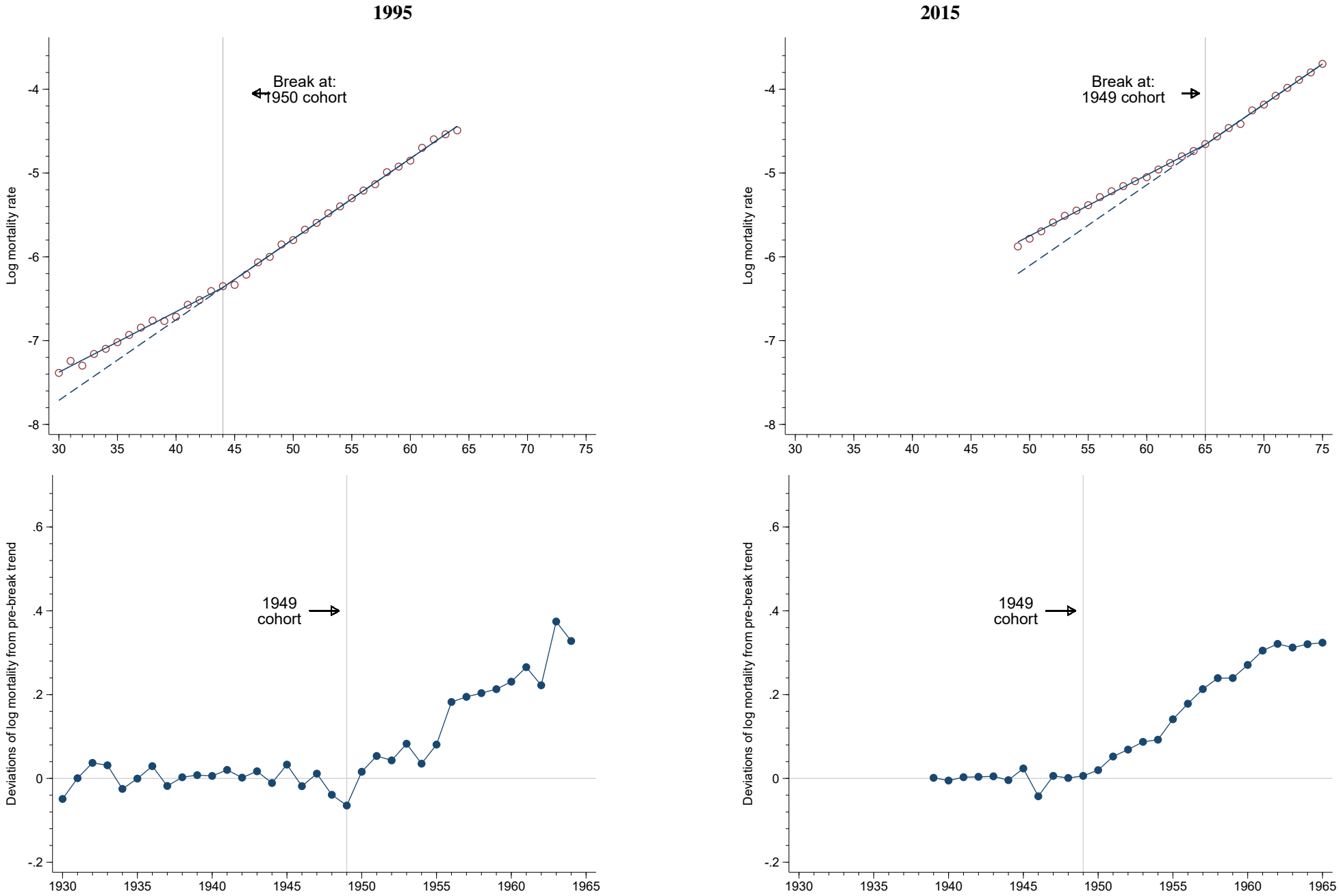
The first panel shows the mean birth weight of infants in grams by their mother's age, for the listed years. Data is from the vital statistics natality detail files. The second panel recasts the same series by mother's year of birth.

Figure A3: Log mortality rate of white men



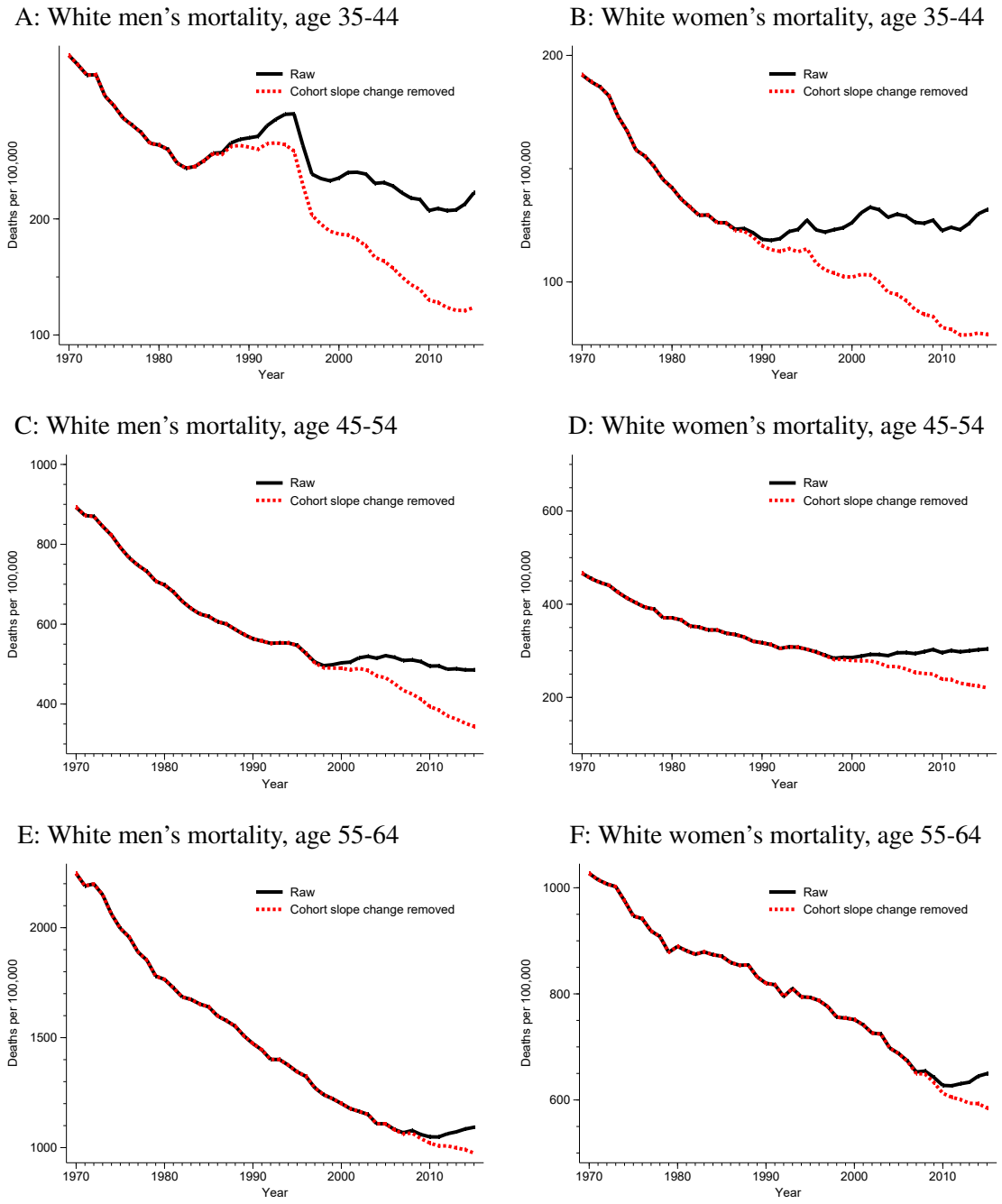
The top two panels shows the log mortality rate of white men by age for the year listed, for 1930 to 1965 cohorts. Red circles show the observed log mortality rate by single year of age. The solid blue line shows plots a piecewise-linear, trend-break model estimated by weighted-least squares. The vertical 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. The two panels show the deviations of the true log mortality rates of white women from the estimated linear trend for cohorts born before an estimated trend break. A horizontal gray line is plotted at the 1946 cohort.

Figure A4: Log mortality rate of white women



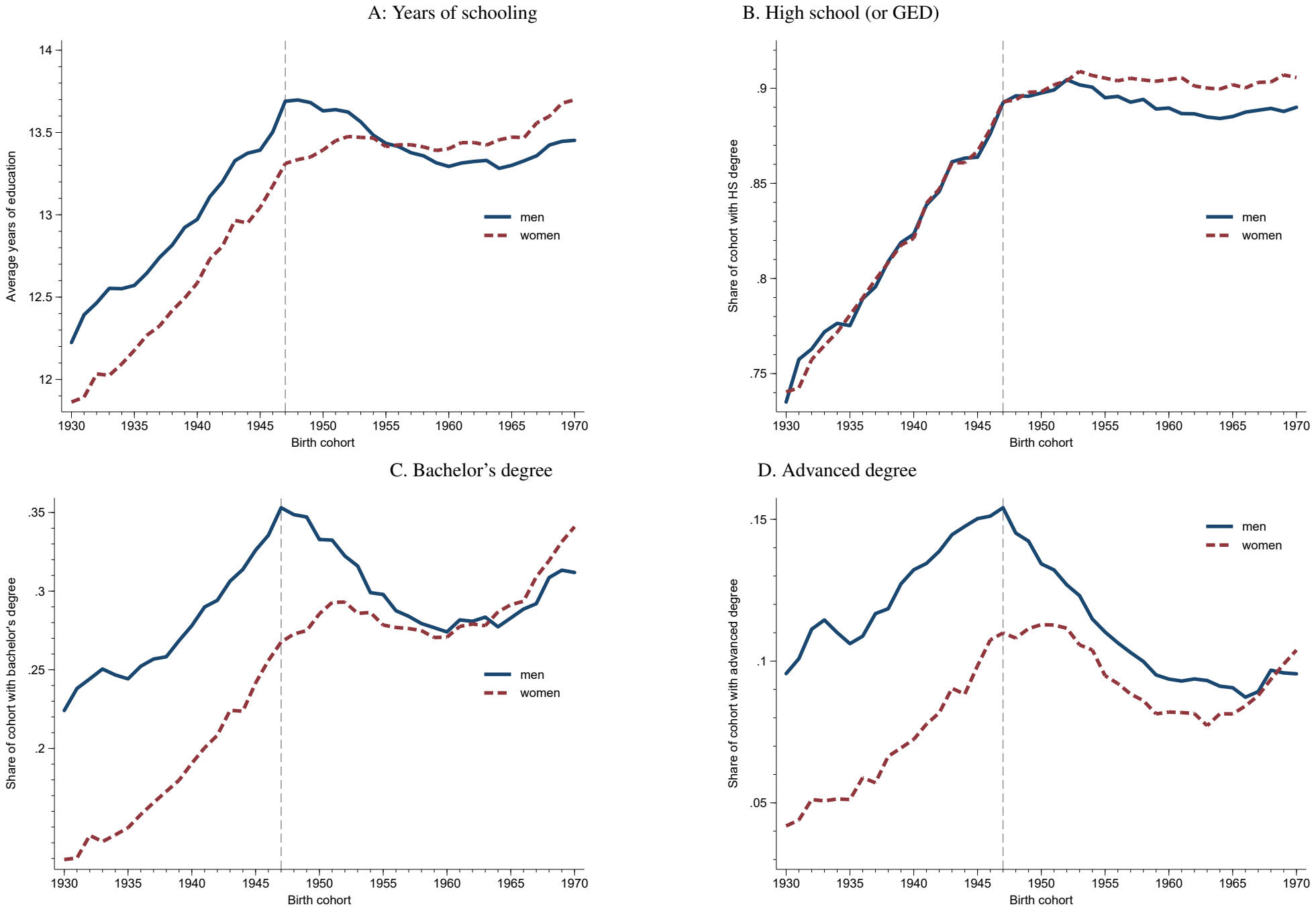
The top two panels shows the log mortality rate of white women by age for the year listed, for 1930 to 1965 cohorts. Red circles show the observed log mortality rate by single year of age. The solid blue line shows plots a piecewise-linear, trend-break model estimated by weighted-least squares. The vertical 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. The bottom two panels show the deviations of the true log mortality rates of white women from the estimated linear trend for cohorts born before an estimated trend break. A horizontal gray line is plotted at the 1946 cohort.

Figure A5: Impact of cohort decline on year-over-year trends in white mortality



This figure shows simulated counterfactual year-over-year trends in the white mortality rate had the change in slope of cohort effects not occured, ie. if the pre-slope-change cohort trend had continued. The slope change estimated based on the model in Equation 2 is subtracted from each log mortality observation. These transformed data are then used to calculate each of the listed outcomes, and is plotted as the dashed red line. The same outcome based on the untransformed, raw data is plotted as a solid line. Each panel shows white for the listed age group, age-adjusted assuming a uniform population distribution by age. Underlying data is the Multiple Cause of Death file and SEER population estimates.

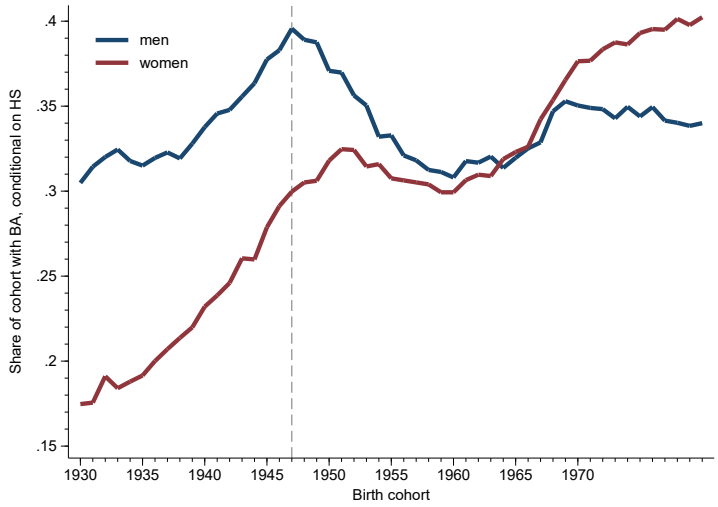
Figure A6: Declines in educational attainment



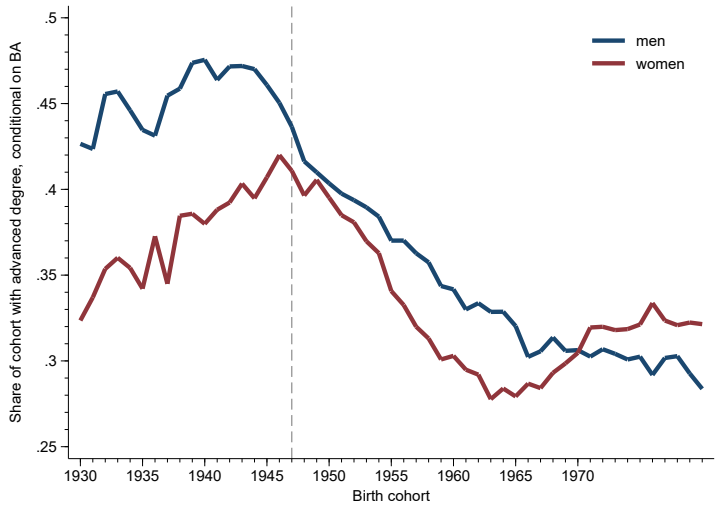
Data is from CPS Merged Outgoing Rotation Group and includes men and women age 25-75 in years 1990-2018. Panel A plots the average years of schooling by birth cohort — approximated based on 16 educational categories. Panels B-D plot respectively the share of each birth cohort with a high school or GED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

Figure A7: Declines in conditional educational attainment along educational ladder

A: Bachelor's degree conditional on finishing high school

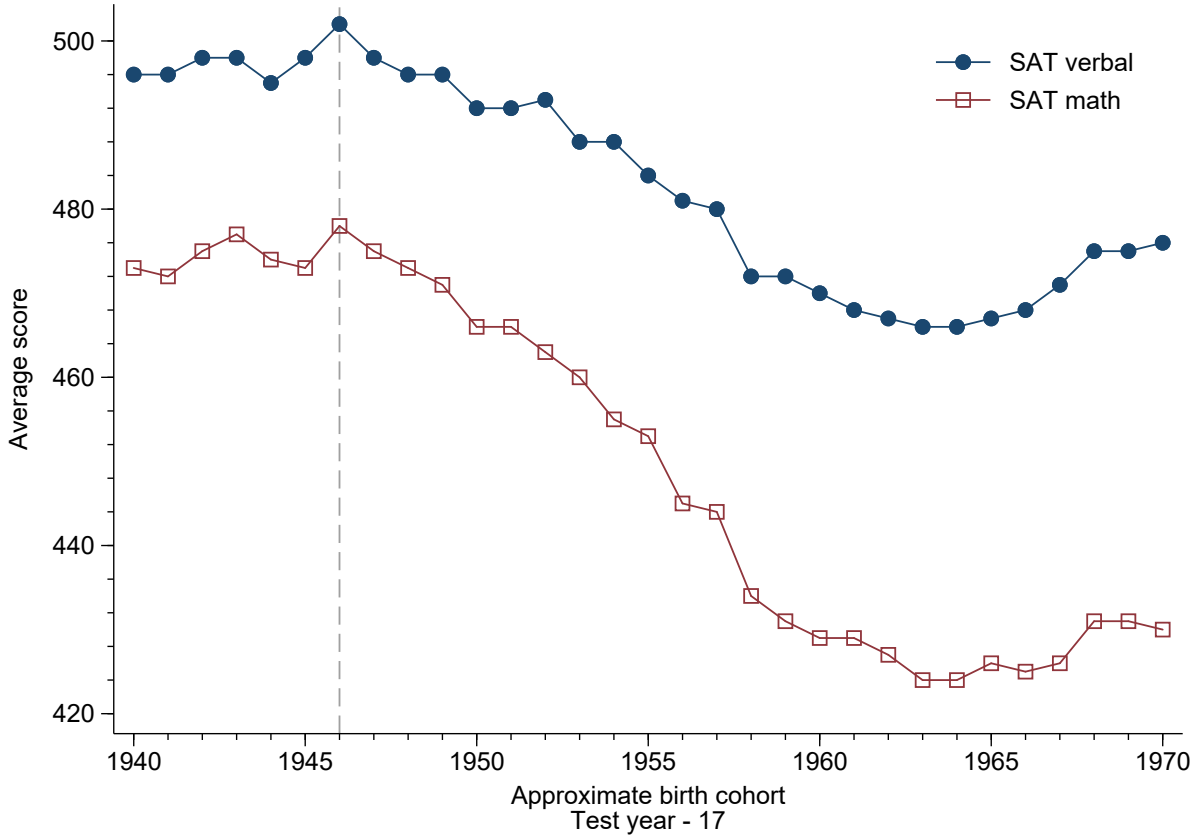


B: Advanced degree conditional on Bachelor's degree



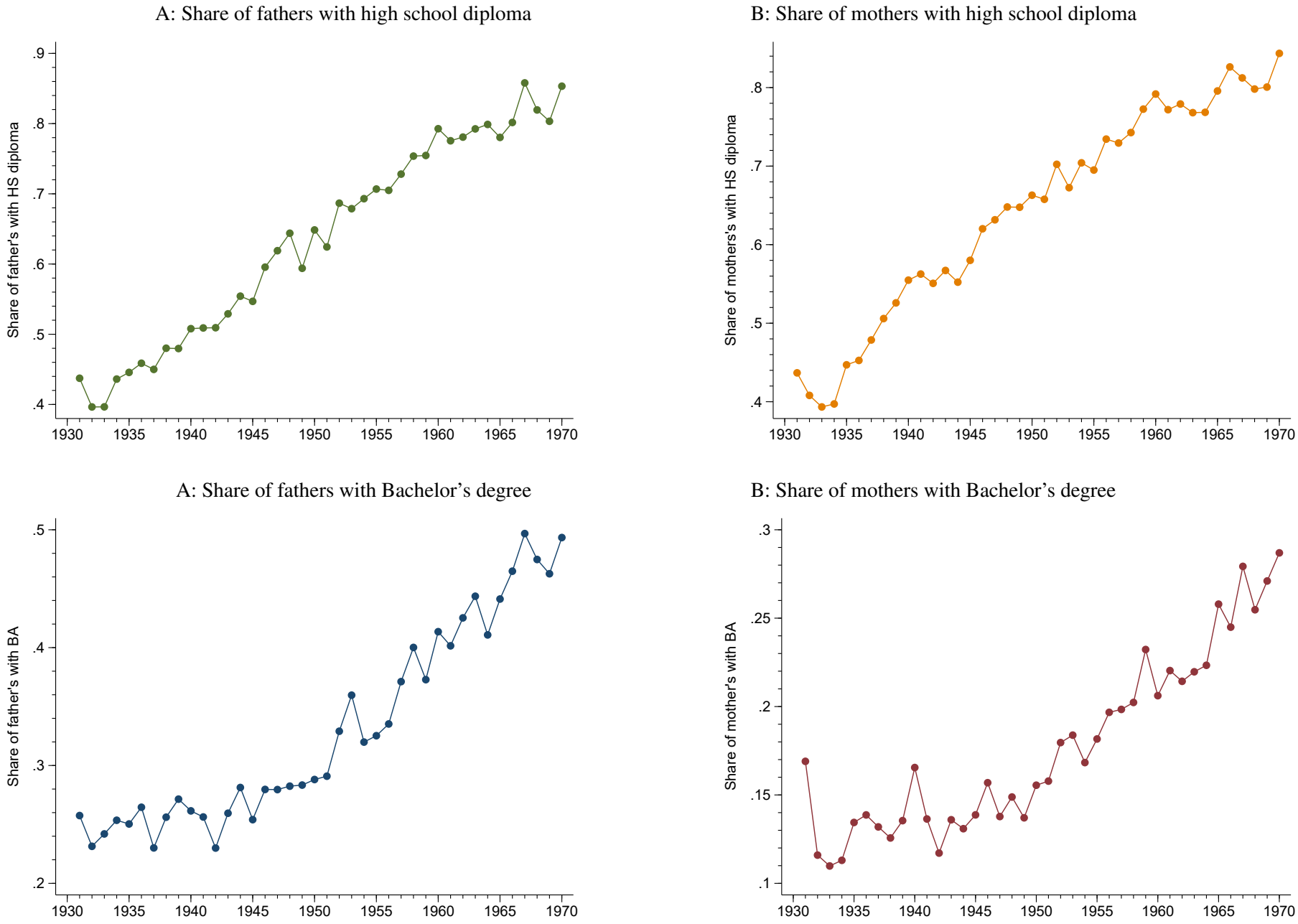
Data is from CPS Merged Outgoing Rotation Group and includes men and women age 25-75 in years 1990-2018. Panel A plots the the share of each birth cohort with a Bachelor's degree conditional on finishing high school — measured as the ratio of the share with a Bachelor's or more over the share with a high school diploma or more. Panel B plots the share of each birth cohort with an advanced degree conditional on having a Bachelor's degree — measured as the ratio of the share with an advanced degree or more over the share with a Bachelor's degree or more.

Figure A8: Declines in Scholastic Aptitude Test (SAT) Scores



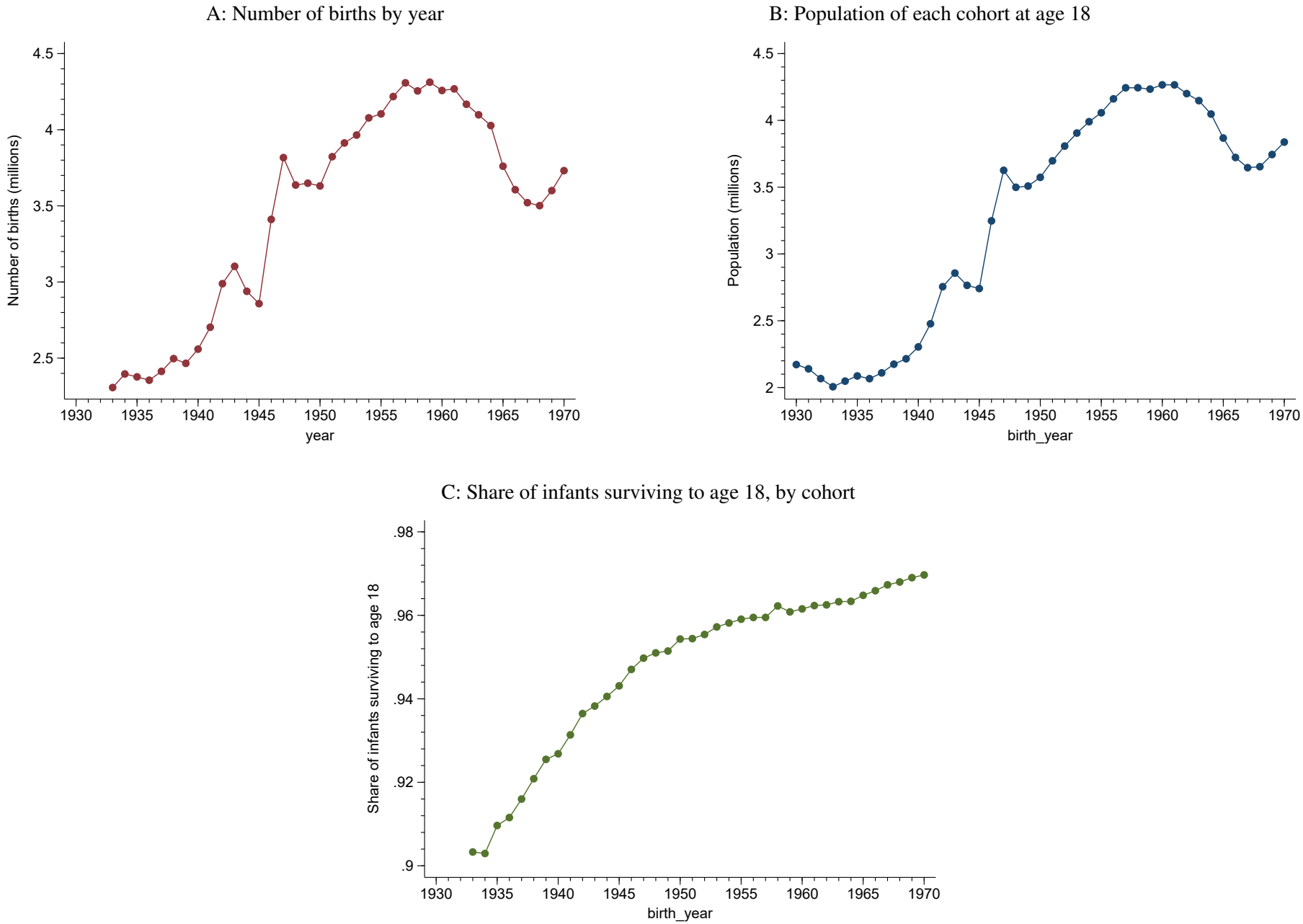
This figure shows verbal and mathematics scores on the Scholastic Aptitude Test. Approximate birth cohort is defined as the year the test was given minus 17. Data is from Harnischfeger and Wiley (1975).

Figure A9: Parental education by birth cohort



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.

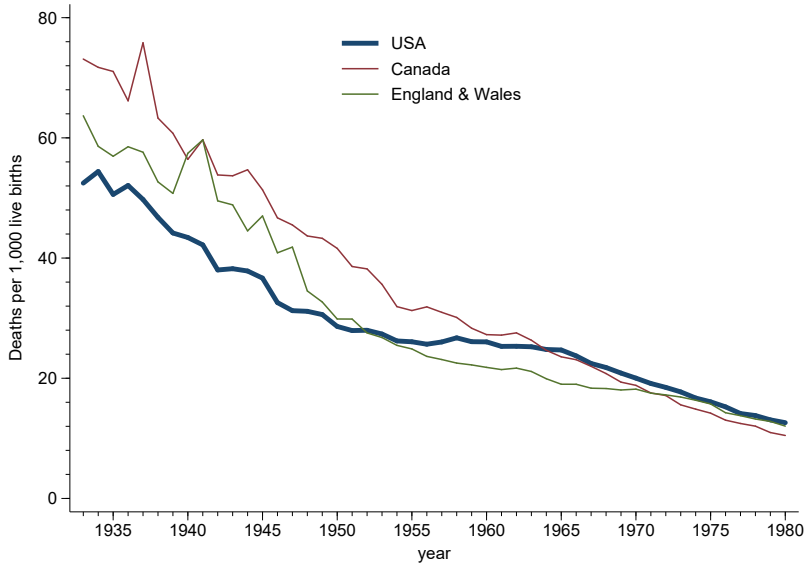
Figure A10: Cohort size and survival



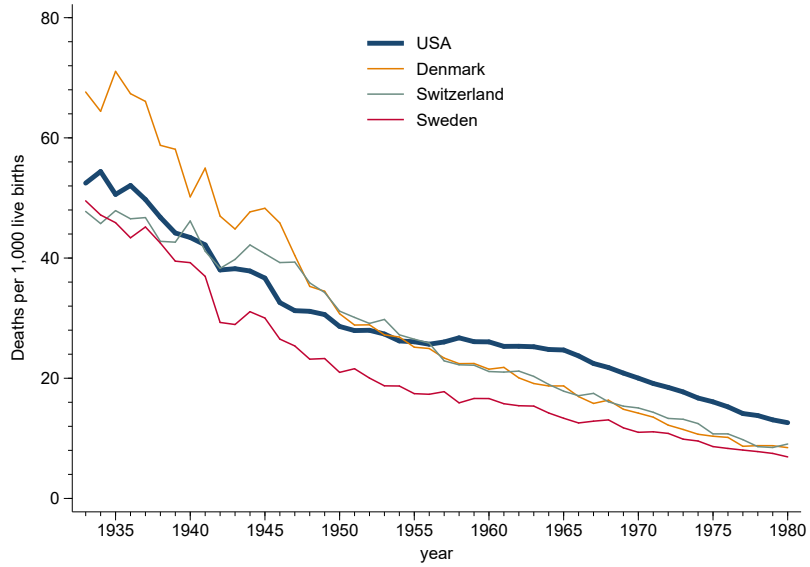
All data from the Human Mortality Database. Panel A shows the number of births in the United States by year. Panel B shows the population of each cohort when they were age 18. Panel C shows the share of infants surviving to age 18 for each cohort — calculated by decrementing the cohort life tables.

Figure A11: Infant mortality rate in United States and comparision countries

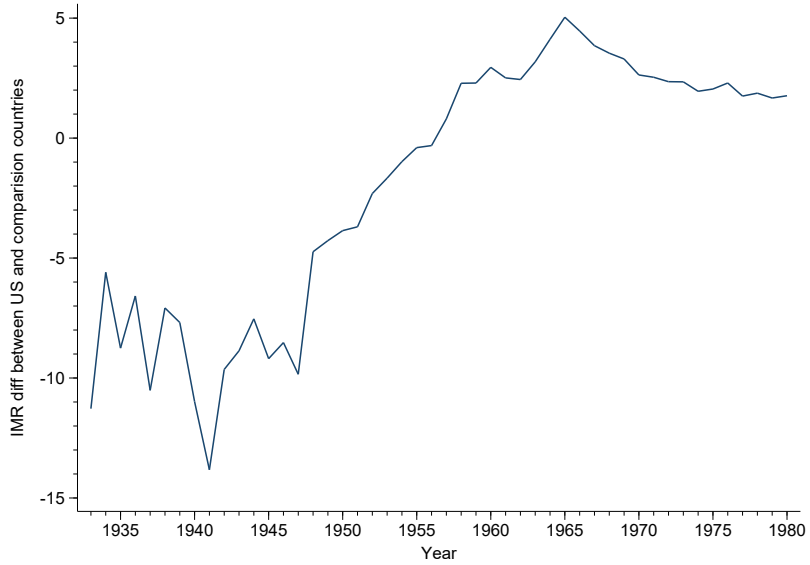
A: English-speaking countries



B: Scandinavian countries

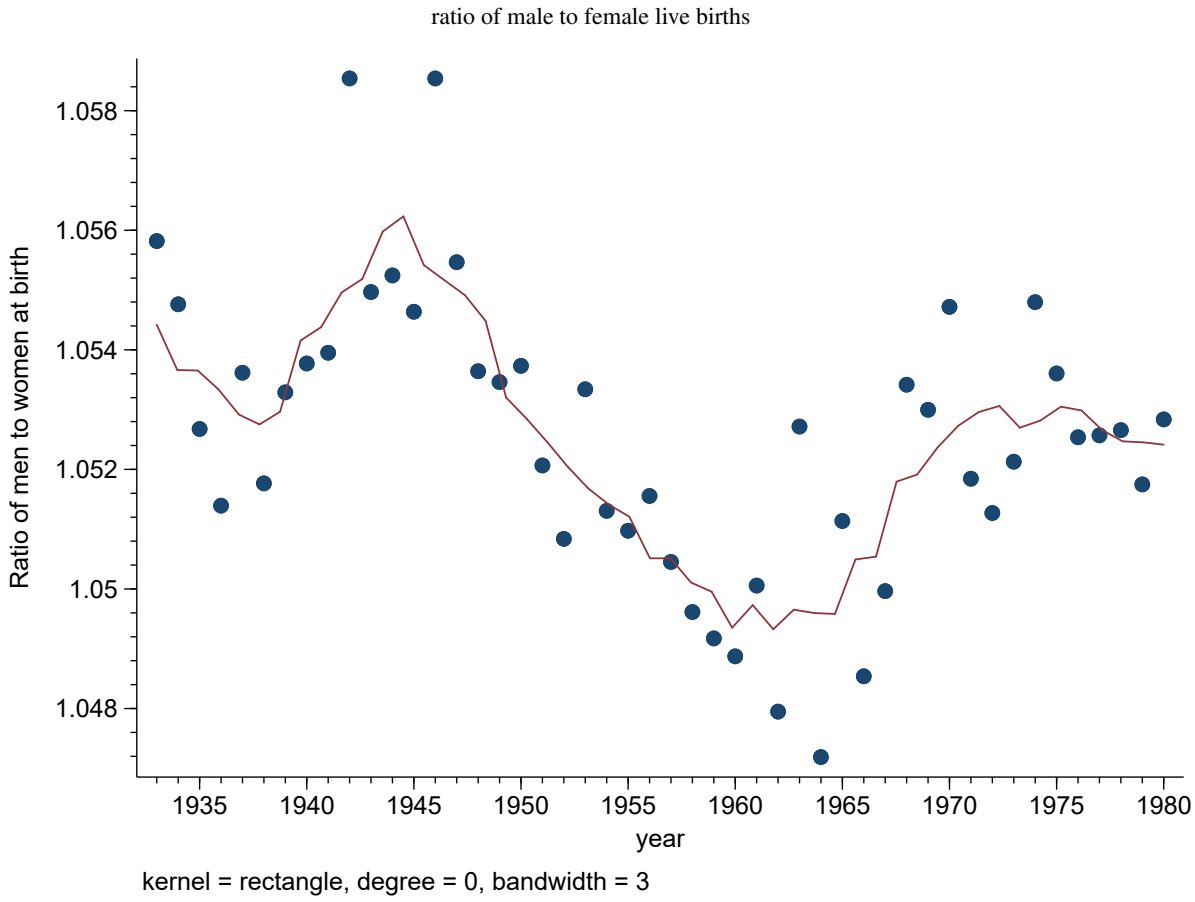


C: Difference between United States and mean across Canada, England and Wales, Denmark, Switzerland, and Sweden



Panels A and B show the infant mortality rates in the listed countries. Panel C shows the difference between the infant mortality rate in the United States and the mean across Canada, England and Wales, Denmark, Switzerland, and Sweden. The mean in the above figure is weighted by the number of births. Results are similar using the unweighted average. All data is from the Human Mortality Database.

Figure A12: Sex ratio at birth in the United States



This figure shows the sex ratio at birth over time in the United States, as well as a running-mean smoother. The sex ratio at birth is defined as the ratio of male live births to female live births. Data is from the Human Mortality Database.

Table A1: Mean-shift in differenced age profiles

robustness to varying age-by-year control function

	(1)	(2)	(3)	(4)
Panel A: Mean birth weight				
Size	-4.11	-7.49	-8.97	-10.14
	(0.55)	(0.99)	(1.08)	(1.14)
Location	1948	1948	1948	1948
	[1948, 1948]	[1948, 1948]	[1948, 1948]	[1948, 1948]
P-value for existence	$< .001$	$< .001$	$< .001$	$< .001$
Panel B: Low birthweight percentage				
Size	0.121	0.196	0.223	0.227
	(0.023)	(0.043)	(0.047)	(0.051)
Location	1948	1948	1948	1948
	[1948, 1949]	[1948, 1949]	[1937], [1948]	[1948, 1948]
P-value for existence	$< .001$	$< .001$	$< .001$	$< .001$
Panel C: Median log wage				
Size	-0.017	-0.020	-0.020	-0.023
	(0.002)	(0.005)	(0.006)	(0.007)
Location	1948	1948	1948	1948
	[1948, 1948]	[1948, 1948]	[1948, 1948]	[1948, 1948]
P-value for existence	$< .001$	.001	.005	.004
Panel D: Log mortality, men				
Size	0.013	0.030	-0.025	.023
	(0.001)	(0.002)	(0.003)	(0.003)
Location	1947	1947	1953	1947
	[1947, 1947]	[1946, 1947]	[1946, 1947] , [1952, 1953]	[1947, 1947]
P-value for existence	$< .001$	$< .001$	$< .001$	$< .001$
Panel E: Log mortality, women				
Size	0.013	0.027	-0.016	-0.017
	(0.001)	(0.003)	(0.003)	(0.004)
Location	1947	1947	1953	1947
	[1947, 1947]	[1947, 1947]	[1947], [1953]	[1947, 1947]
P-value for existence	$< .001$	$< .001$	$< .001$	$< .001$
Year FEs	Yes	Yes	Yes	Yes
Age FEs	Yes	Yes	Yes	Yes
Quadratic-age-by-year	No	Yes	No	No
Cubic-age-by-year	No	No	Yes	No
Quartic-age-by-year	No	No	No	Yes

Each column shows the results of estimation of a model based on Equation 2, with the listed outcome in single age-by-year bins as the dependent variable. All models are estimated by least squares, following the approach outlined in Hansen (2000). The row titled "Size" reports the size of the change in slope of cohort effects, δ, with the standard error in parentheses. The row titled "Location" reports the estimated cohort at which a trend break occurs, with a 99 % confidence region in brackets calculated by inverting the likelihood ratio statistic. The row titled "P-value for existence" reports a p-value from an F-type test, based on 1000 bootstrap samples, for the null hypothesis that no change in slope occurs, ie. that cohort effects are linear.

Table A2: Piecewise linear cohort effect models

robustness to varying age-by-year control function

	(1)	(2)	(3)	(4)
Panel A: Mean birth weight				
Size	-1.83	-6.35	-5.52	-6.91
	(0.13)	(0.35)	(0.54)	(0.70)
Location	1948	1948	1947	1947
	[1947, 1949]	[1948, 1948]	[1947, 1947]	[1947, 1947]
P-value for existence	$< .001$	$< .001$	$< .001$	$< .001$
Panel B: Low birthweight percentage				
Size	0.085	0.241	0.195	0.200
	(0.003)	(0.014)	(0.022)	(0.029)
Location	1947	1948	1947	1947
	[1947, 1948]	[1948, 1948]	[1937], [1948]	[1948, 1948]
P-value for existence	$< .001$	$< .001$	$< .001$	$< .001$
Panel C: Median log wage				
Size	-0.016	-0.016	0.015	-0.017
	(0.0005)	(0.001)	(0.002)	(0.003)
Location	1947	1947	1953	1947
	[1947, 1947]	[1946, 1947]	[1946, 1947], [1952, 1953]	[1947, 1947]
P-value for existence	$< .001$	$< .001$	$< .001$	$< .001$
Panel D: Log mortality, men				
Size	0.017	0.029	0.030	0.027
	(0.001)	(0.001)	(0.001)	(0.002)
Location	1941	1946	1946	1947
	[1940, 1942]	[1946, 1946]	[1946, 1946]	[1947, 1947]
P-value for existence	$< .001$	$< .001$	$< .001$	$< .001$
Panel E: Log mortality, women				
Size	0.017	0.031	0.018	-0.023
	(0.001)	(0.003)	(0.003)	(0.004)
Location	1947	1949	1950	1942
	[1947, 1947]	[1949, 1949]	[1942], [1951, 1950]	[1942, 1942]
P-value for existence	$< .001$	$< .001$	$< .001$	$< .001$
Year FEs	Yes	Yes	Yes	Yes
Age FEs	Yes	Yes	Yes	Yes
Quadratic-age-by-year	No	Yes	No	No
Cubic-age-by-year	No	No	Yes	No
Quartic-age-by-year	No	No	No	Yes

Each column shows the results of estimation of a model based on Equation 2, with the listed outcome in single age-by-year bins as the dependent variable. All models are estimated by least squares, following the approach outlined in Hansen (2000). The row titled "Size" reports the size of the change in slope of cohort effects, δ, with the standard error in parentheses. The row titled "Location" reports the estimated cohort at which a trend break occurs, with a 99 % confidence region in brackets calculated by inverting the likelihood ratio statistic. The row titled "P-value for existence" reports a p-value from an F-type test, based on 1000 bootstrap samples, for the null hypothesis that no change in slope occurs, ie. that cohort effects are linear.

Table A3: Change in slope of cohort effects for share of employed men working in white-collar occupations — native-born versus foreign-born

robustness to varying age-by-year control function

	(1)	(2)	(3)	(4)
Panel A: Born in US				
Size	-0.007	-0.017	-0.017	-0.014
	(0.0004)	(0.0009)	(0.002)	(0.002)
Location	1946	1946	1946	1946
	[1944, 1946]	[1946, 1946]	[1946, 1946]	[1946, 1947]
P-value for existence	$< .001$	$< .001$	$< .001$	$< .001$
Born outside US				
Size	-0.008	0.027	0.031	0.018
	(0.001)	(0.005)	(0.008)	(0.006)
Location	1941	1932	1932	1956
	[1940, 1943]	[1932, 1933], [1943, 1944]	[1932, 1933]	[1932], [1936, 1937], [1956, 1957]
P-value for existence	$< .001$	$< .001$	$< .001$	0.007
Year FEs	Yes	Yes	Yes	Yes
Age FEs	Yes	Yes	Yes	Yes
Quadratic-age-by-year	No	Yes	No	No
Cubic-age-by-year	No	No	Yes	No
Quartic-age-by-year	No	No	No	Yes

Each column shows the results of estimation of a model based on equation 2, with the the share of employed men in white-collar occupations as the dependent variable in single age-by-year bins as the dependent variable. The data are the IPUMS samples of the 1970, 1980, 1990 and 2000 censuses. All models are estimated by least squares, following the approach outlined in Hansen (2000). The row titled "Size" reports the size of the change in slope of cohort effects, δ, with the standard error in parentheses. The row titled "Location" reports the estimated cohort at which a trend break occurs, with a 99 % confidence region in brackets calculated by inverting the likelihood ratio statistic. The row titled "P-value for existence" reports a p-value from an F-type test, based on 1000 bootstrap samples, for the null hypothesis that no change in slope occurs, ie. that cohort effects are linear.

Table A4: Change in slope of cohort effects for mean birth weight of infants by mother's birth cohort — separately for mothers with exactly 12 and 16 years of schooling

robustness to varying age-by-year control function

	(1)	(2)	(3)	(4)
Panel A: 12 years of schooling				
Size	-0.99	-5.22	-7.32	-10.44
	(0.22)	(0.79)	(1.02)	(1.37)
Location	1947	1947	1947	1947
	[1946, 1949]	[1947, 1947]	[1947, 1947]	[1947, 1947]
P-value for existence	$< .001$	$< .001$	$< .001$	$< .001$
Panel B: 16 years of schooling				
Size	-2.15	-6.57	-6.61	-4.07
	(0.43)	(1.37)	(1.72)	(2.40)
Location	1946	1947	1947	1947
	[1944, 1948]	[1946, 1953]	[1947, 1947]	[1938, 1963]
P-value for existence	$< .001$	$< .001$	$< .001$	.002
Year FEs	Yes	Yes	Yes	Yes
Age FEs	Yes	Yes	Yes	Yes
Quadratic-age-by-year	No	Yes	No	No
Cubic-age-by-year	No	No	Yes	No
Quartic-age-by-year	No	No	No	Yes

Each column shows the results of estimation of a model based on Equation 2, with the listed outcome in single age-by-year bins as the dependent variable. All models are estimated by least squares, following the approach outlined in Hansen (2000). The row titled "Size" reports the size of the change in slope of cohort effects, δ, with the standard error in parentheses. The row titled "Location" reports the estimated cohort at which a trend break occurs, with a 99 $\%$ confidence region in brackets calculated by inverting the likelihood ratio statistic. The row titled "P-value for existence" reports a p-value from an F-type test, based on 1000 bootstrap samples, for the null hypothesis that no change in slope occurs, ie. that cohort effects are linear. Results are based on vital statistics natality microdata, 1969-1985, mothers age 22-40, cohorts born 1938-1965.

Table A5: Change in slope of cohort effects for intergenerational infant birth weight
By Maternal education level

controlling for year FEs, age FEs, and quadratic age-by-year

	Change in cohort slope		
	Size	Location	Existence
	δ	λ	*p-value*
Maternal education level			
Less than HS	-6.90	1948	0.014
	(1.69)	[1942, 1944], [1947, 1949]	
High school	-5.22	1947	0.023
	(0.79)	[1947, 1947]	
Some college	-7.19	1947	$< .001$
	(1.28)	$[1947, 1948]$	
4 years college	-6.57	1947	0.067
	(1.37)	[1946, 1953]	
5+ years college	9.52	1951	0.305
	(2.05)	[1951, 1952]	

Each row shows the results of estimation of a model based on equation 2. All models are estimated by least squares, following the approach outlined in Hansen (2000). The column titled "Size" reports the estimated size of the change in cohort slope δ, with the standard error in parentheses. The column titled "Location " reports the cohort at which the slope change is estimated to occur, with a 99 $\%$ confidence interval in brackets calculated by inverting a likelihood ratio statistic. The row titled "Existence" reports a p-value from an F-type test, based on 1000 bootstrap samples, for the null hypothesis that no change in slope occurs, ie. that cohort effects are linear. Results are based on vital statistics natality microdata, 1969-1985, mothers age 22-40, cohorts born 1938-1965.

Table A6: Evidence of cohort decline in wages for urban, suburban, and rural workers— piecewise linear cohort effect models controlling for age FEs and year FEs

	Change in cohort slope		
	Size	Location	Existence
	δ	λ	*p-value*
Rural	-0.014	1947	$< .001$
	(0.0009)	[1947, 1948]	
Suburban	-0.015	1947	$< .001$
	(0.0007)	[1946, 1947]	
Urban	-0.016	1947	$< .001$
	(0.0007)	$[1947, 1947]$	

Each row shows the results of estimation of a model based on Equation 2, with median wage as the dependent variable. The rows report results for samples restricted to workers residing in rural, suburban, and urban areas, respectively. All models are estimated by least squares, following the approach outlined in Hansen (2000). The column titled "Size" reports the estimated size of the change in cohort slope δ, with the standard error in parentheses. The column titled "Location " reports the cohort at which the slope change is estimated to occur, with a 99 $\%$ confidence interval in brackets calculated by inverting a likelihood ratio statistic. The row titled "Existence" reports a p-value from an F-type test, based on 1000 bootstrap samples, for the null hypothesis that no change in slope occurs, ie. that cohort effects are linear. All results are based on CPS-MORG data, 1979-1993, and includes men age 25-54, who were born from 1930 to 1965.