Declining Health and Human Capital of Americans Born after 1947

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Abstract

I present evidence of a decline in the health and human capital of Americans beginning with cohorts born after 1947 and continuing until at least mid-1960s cohorts. Age-adjusted educational attainment, earnings, maternal health (proxied by the birthweight of infants), and mortality all exhibit trend breaks near the 1947 cohort, such that each outcome worsens for subsequent cohorts relative to the prior trend. Evidence of these sharp breaks remain when I control for year and age effects or smooth age-by-year interactions, or use a novel nonparametric test. The same cohorts had lower scores on standardized tests as teenagers and their physical growth and development was delayed — suggesting a broad decline in the health and human capital of these cohorts, originating early-in-life and manifesting in poor outcomes throughout their lives.

The cohort decline is large enough to drive aggregate: i) educational declines in the 1960s, ii) increases in the low birthweight rate in the 1980s, iii) mortality increases since 1999, and to contribute substantially to iv) wage stagnation since the 1970s. My theory therefore partially unifies the disparate searches for the causes of each of these four declines into a single search for what went wrong early in life for Americans born after 1947. I begin this search: presenting evidence against a number of plausible hypotheses and showing that the decline is remarkably widespread across geography and race, for those born in the US.

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1. Introduction

Improvements in health and human capital were enormous across American cohorts born in the first half of the twentieth century. Robert Fogel and others have argued that the key driver of these improvements have been gains in health in early life.[1] In this paper, I present evidence that, in a number of dimensions, this progress in adult health and human capital hit a sudden stop and reversed trend for cohorts born after 1947 in the United States. Instead, this paper suggests that the underlying health and human capital of Americans born in each year between 1947 and the mid-1960s has been worse than that of those born a year earlier. I present evidence that this decline likely also originated early in life.

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

Figure 1 shows that the trend across cohorts in age-adjusted educational attainment, earnings, maternal health (proxied by the birthweight of infants), and mortality all exhibit trend breaks near the 1947 cohort, such that each outcome declines for subsequent cohorts relative to the prior trend. [2] These simultaneous trend breaks, while striking, could in principle reflect differences in external factors which these cohorts were exposed to, rather than underlying differences in health and human capital. That is, cohorts born after 1947 may have been otherwise similar to earlier cohorts, but were merely unlucky to have experienced bad conditions throughout their lifetime.

[1] For evidence of the health improvements and arguments that they are driven by early life health see, for example, Fogel (1986); Fogel and Costa (1997); Costa and Steckel (1997); Floud et al. (2011); Fogel (2012); Costa (2015).

[2] 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 birthsU sing 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); earnings of men, using CPS survey data; and the mortality rates of men and women, again using vital statistics data.

I therefore provide evidence, under increasingly weak assumptions about the nature of external age-by-year factors, that these patterns reflect a decline in the underlying health and human capital of cohorts born after 1947, relative to the prior trend. I define a generalized version of "cohort effects" as the counterfactual of what a given cohort's outcomes would have been had they been exposed to a different set of external age-by-year factors than those they experienced. First, I estimate traditional age-period-cohort models, which assume cohort, age, and year factors are each additively separable. Second, I estimate models with a trend break of unknown location in cohort effects while allowing for a separate polynomial in age in each year — adapting methods from the structural break literature (Hansen, 1999, 2000). Third, I develop a novel, nonparametric generalization of McKenzie (2006) which identifies second differences in "local average cohort effects" under the assumption that age-by-year shocks are "locally independent".[3]

All these methods reveal strong evidence of a trend break at the 1947 or 1948 cohort in underlying health and human capital evident in each of the above six outcomes. The remaining threat to validity would be nonsmooth changes in the 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 — which repeatedly "hit" the same cohorts. Alternative expladnations put forward in the related literatures do not have this feature. For example, the effects of shifts in supply or demand will be smooth as long as individuals who are close in age are substitutable (Card and Lemieux, 2001a).

Simulations suggest that the cohort-based decline contributed substantially to the year-over-year declines described above. The structural-break-based 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 of men and women 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, increases in the midlife mortality of whites since 1999, and to a lesser extent wage stagnation over the last 50 years, can be at least partially unified. To find the cause of all of these declines will require understanding what went deeply wrong early in life for cohorts of Americans born after 1947.

The above methods do not formally identify the long-run trend in cohort effects, and therefore cannot speak to whether cohort health and human capital declined in absolute terms or the trend just suddenly slowed. The absolute declines in educational attainment could be viewed as prima facie evidence of absolute declines in human capital. I also show that under reasonable assumptions on the long-run trend in period or age effects it appears that cohort effects in the other outcomes

[3]The writeup and results of this approach are in progress and will be added to future drafts. I define "Local average cohort effects" as the counterfactual average outcomes a cohort would have if they had experienced the external age-by-year factors of a cohort born one year earlier or later.

declined in absolute terms after 1947.[4]

I then present evidence suggesting that the health and human capital decline likely originated early in life. I show that cohorts born after 1947 did worse on standardized tests as early as age 17 and their physical growth and development was slower than for earlier born cohorts. I also show that the health decline is evident for individuals born in the United States but not for residents born outside the United States.

I provide a preliminary investigation of the underlying cause of the apparent early life health decline of these cohorts. First, I highlight that the health decline is widespread across demographic groups in the United States. I then provide evidence against a host of ex ante plausible causes. I am continuing the search for the underlying cause of this cohort decline. One promising candidate is lead pollution from motor vehicle exhaust. Motor vehicle use increased sharply after 1945 and the gasoline used in motor vehicles included large and increasing quantities of lead additives. Fetal and early life lead exposure has been linked to poor health and cognitive development, and high child blood lead content was ubiquitous across the US when these cohorts were children.[5]

The evidence above suggests that something went deeply wrong early in life for cohorts of Americans born after 1947, and that as these cohorts aged their poor outcomes have contributed substantially to aggregate declines over the last 60 years. This does not rule out an important role for period-based factors, such as labor demand or opioid supply. The analysis in this paper cannot identify potential interactions between the poor underlying human capital and such period-based factors. What this paper points to strongly is that these recent crises have deep roots and that the first link in the chain of "cumulative deprivation", in the words of Case and Deaton, came early in life for these cohorts. It seems likely that the initial poor start for these cohorts was exacerbated by the poor demand for "unskilled" workers when they entered the labor market, and again by massively expanded supply of prescription and illegal opioids as they hit midlife — and future research should examine these interactions.

The remainder of the paper is organized as follows. Section 2 reviews relevant literature. Section 3 describes the data and main outcomes. Section 4 presents a conceptual model to guide the analysis. Section 5 present the results from the age-period-cohort models typically used in the literature. Section 6 contains the results from the models which allow for smooth age-by-year interactions. Section 5 shows the sizeable impact of the cross-cohort declines on year-over-year changes in each outcome. Section 7 will present the nonparametric method and results. Section

[4] I use approaches similar to those in Lagakos et al. (2018) and Fosse and Winship (2017).

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

8 shows the role of the cohort decline in aggregate year-over-year declines. Section 9 presents evidence suggesting the decline originated in childhood. Section 10 present a preliminary investigation into the root cause of the cohort decline. The final section concludes.

2. Literature review

Some earlier work has discussed the possibility of cohort-based declines contributing to the aggregate patterns described above. Some papers also have presented evidence of cohort-specific declines in particular outcomes for Americans born after roughly the middle of the twentieth century.

My paper is unique in providing: a unified analysis which documents the shared timing of the cohort decline in multiple outcomes; a careful analysis showing that the break in trend is sharp, documenting its precise timing, and attempting to rule out external factors; evidence that the decline likely originated early in life; and an initial investigation into the cause of that early-life health decline.

Multiple outcomes

Very few papers have noted that multiple distinct outcomes have declined for these cohorts. A notable exception is Borella et al. (2020), which compares the outcomes for white Americans without a college degrees born in the 1940s to those born in the 1960s across a number of outcomes. They document declines in wages and projected life expectancy and increases in out-of-pocket medical expenses for this group.

There are a few important distinctions between their study and mine. First, I will study average outcomes for all Americans in each cohort born between 1930 and 1970, rather than focusing on a potentially selected subset of the population. Second, their focus is on the effects of the declines in the above outcomes labor supply, savings, and welfare, which they quantify using a life-cycle model. In contrast, I am interesting in understanding whether the declines in these (and other) outcomes are driven by "cohort effects" — long-standing differences in health and human capital — or external factors which have hit these cohorts at different points in their lifetime. Related to both these points, I have a particular focus on the sharp trend break in outcomes near the 1947 cohort which is not present in their study.

Educational attainment

The declines in educational attainment for cohorts born after the late-1940s is relatively well known but the cause is not well understood. 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. They also show that the share of each cohort completing a college degree suddenly began to decline around 1945 for men, and suddenly stagnated around 1950 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. [6] This decline in college completion rates is also a key driver of increases in the college-high school wage gap in the "canonical model" of Katz and Murphy (1992) and subsequent studies (Card and Lemieux, 2001a; Autor et al., 2008). Acemoglu and Autor (2012) also note this stagnation in educational attainment, and presaging the findings in this study, 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." To my knowledge, my finding below of trend breaks at the 1947 cohort in the share of men and women earning an advanced degree is novel.

Earnings

Initial research into wage stagnation beginning in the 1970s and the rapid decline in earnings for those without a BA in the 1980s considered but largely dismissed the role of a cross cohort decline in "earnings ability". See for example, the discussions in Bound and Johnson (1992); Katz et al. (1999). The results in this paper suggest it is perhaps time for a reconsideration. Interestingly, an earlier paper that seems to be largely forgotten, Bishop (1989), estimated that the decline in cognitive ability, as measured by test scores, for these cohorts had a large effect on wage rates and would continue to contribute substantially to a productivity decline over the coming decades.

More recently, Guvenen et al. (2022) use panel data on earnings from tax returns to study the life time income of successive cohorts. Among other findings, they document a decline in median lifetime earnings of American men of more than 10 percent between the 1942 and 1960 birth cohorts. They also note that these cohorts were already behind by the time they were 25, and therefore close the paper by stating that "the sources of the dramatic changes we have witnessed in the U.S. earnings distribution over the last 50 years may be found in the experiences of newer cohorts during their youth (and possibly earlier), and how those experiences differed from those of older cohorts." My paper builds on theirs by explicitly confronting the challenge of identifying whether these (and other) declines for these cohorts are driven by long-standing differences in health and human capital.

Maternal health

To my knowledge no prior papers have documented evidence of the cohort decline in maternal

[6] 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. Handy and Shester (2019) suggest that changes in the average parity or birth order by cohort can explain can explain more than one third of the decline in college completion for white men born between 1946 and 1960.

health shown in this paper. Both the idea of maternal endowments as contributing to birth outcomes of children (Rosenzweig and Schultz, 1983; Grossman and Joyce, 1990) and that of the life-course perspective on maternal health (Lu and Halfon, 2003) suggest the using birth outcomes of children can be useful to understand the underlying health of mother's. This strategy was previously used in Almond and Chay (2006) to study black-white differences in cohort health.[7]

Mortality

Case and Deaton (2015) documented a shocking increase in the all-cause mortality rate of non-Hispanic white Americans and noted a proximate cause was increases in so-called "deaths of despair" — drug and alcohol poisonings, suicide, and chronic liver diseases and cirrhosis. Subsequent research has suggested that cohort differences in health and disadvantage which predated the 1990s could be important as well (Case and Deaton, 2017; Lleras-Muney, 2017; Masters et al., 2017; Zang et al., 2018; Reynolds, 2023)

Case and Deaton (2017) develops a preliminary theory of "cumulative disadvantage" which emphasizes worsening opportunities at labor market entry — particularly for whites with low levels of education — as a key driver of the mid-life mortality increases. They posit that these worse opportunities at labor market entry trigger various negative outcomes which build on each other, and culminate in an increased likelihood of untimely death. While it seems very likely that worsening economic conditions interacted with and potentially exacerbated the poor health and human capital of post-1947 cohorts, the research in this paper and my earlier work (Reynolds, 2023) suggest that these cohorts had already fallen behind before labor market entry.

Height

Increases in adult height, often viewed as a proxy of childhood nutritional status (Floud et al., 2011; Tanner, 1990), also suddenly stopped for white Americans born between approximately 1955 and 1974 while the height of Europeans continued to grow rapidly (Komlos and Lauderdale, 2007a,b; Komlos, 2010). Though again, height does not appear to have declined in absolute terms.

I build on these findings by examining height and physical development of these cohorts as children. Preliminary this work shows that the males in cohorts born in the 1960s grew more slowly in adolescence than those born around a decade earlier.

Other outcomes

Since I began work on this project a series of papers by Hui Zheng and couthors have shown declines for the same, or approximately the same, cohorts in other outcomes. Zheng (2021) finds declines in cognitive functioning in old age for approximately the same cohorts for which I find

[7] Also potentially relevant is Tilstra and Masters (2020), which finds that recent declines in birth weight were due to increases in induced labor and cesarean delivery at earlier gestational ages. These findings could be complementary to mine: either these cohorts are more likely to have ceasareans or both explanations are important and could more than explain recent declines in birth weight.

declines. He groups cohorts into larger groupings so the precise timing of the decline is not clear. Zheng and Echave (2021) find that a summary measure of biomarkers of inflammation, metabolic functions, lung function, and renal function appears to have worsened continuously from the baby boom cohort onwards though Gen X and Gen Y cohorts.[8] Interpretation of these findings is complicated by the fact that all results come from a "mixed-model" in which age effects are modelled as fixed effects and cohort and period effects as random effects [9] Zheng et al. (2022) also find evidence of declines in similar measures and a chronic disease index, and attribute these declines to exposure to income inequality in childhood.

Early-life health and human capital development

This paper presents evidence of a broad decline across many dimensions of human capital and some additional evidence that the decline likely originated in childhood or adolescence.

The childhood roots of the health and human capital decline is at least plausible in light of the large literature, reviewed in Currie and Almond (2011), presenting evidence of large effects of infant and childhood health on educational attainment, health, and labor market outcomes. Most of these papers are focused on careful causal identification rather than the aggregate implications of changes in average childhood health for changes in average adult health and human capital by cohort. A notable exception is the earlier cited work by Robert Fogel and other economic historians. These studies focus primarily on earlier cohorts. Interestingly, they find evidence of a decline in height, as well as other proxies of childhood and cohort health, for cohorts born between the 1830 and 1890 cohorts (Steckel and Haurin, 1982; Komlos, 1987). This decline was then followed by rapid growth for the subsequent six decades (Costa and Steckel, 1997).

Also particularly related to my study are Almond and Chay (2006) and Chay et al. (2009, 2014) who find long-run effects of infant health gains for African Americans born in the South after the Civil Rights Act of 1966 led to gains in test scores, educational attainment, earnings, and maternal health (with an intergenerational effect on infants). They also suggest that these cohort improvements were large enough to drive changes in the aggregate black-white gaps in these outcomes, which had previously been attributed to period-based factors.

3. 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.

[8] Interestingly, they do not find similar patterns in a summary measure of clinical risk factors for cardiovascular diseases, stroke, kidney disease, and type 2 diabetes mellitus.

[9] See Bell and Jones (2013); Luo and Hodges (2020) for critiques of these models. More broadly it would be useful to begin with age-adjusted patterns by cohort before turning to identification.

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).[10]

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 in-

[10] 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 in health by cohort.

formation 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.[11] 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.[12]

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.

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.[13] This approach follows Chamberlain (1994) and Chetverikov et al. (2016), and for sufficiently large cells has advantages over traditional LAD quantile regression.[14] 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.

My main outcome variable is therefore median real hourly earnings by single age and year.

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

[12] 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.

[13] 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.

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

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 between those born in the United States and foreign-born. 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.[15]. 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 1975-2019, 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.

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 $year - age$.

4. Conceptual model

This section presents a conceptual model which generalizes the idea of "cohort effects" and suggests a link to counterfactuals and potential outcomes. This model will guide the identification

[15]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.

and estimation in the remainder of the paper which provides evidence of a decline in the underlying health and human capital of cohorts born after 1947.

I will assume that each of the main outcomes I consider are determined by the following model:

$$Y_{apc} = g(\theta_c, \epsilon_{a,p}) \tag{1}$$

where Y_{apc} denotes an outcome — such as earnings or the log mortality rate — 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 outcome is a function of: i) θ_c, underlying, fixed features of the cohort and ii) ϵ_{ap} external factors in the year p which affect individuals of the given age, a. θ_c and ϵ_{ap} are nonseparable and there is no restriction (yet) on the structural function $g(\cdot, \cdot)$ or on the dimensionality of θ_c or ϵ_{ap}.

Conceptually, θ_c represents the underlying, fixed differences between individuals born in different years which impact their outcomes. It reflects very broadly the health, human capital, cognitive ability and "skills" of the cohort. θ_c can be multidimensional to represent both the potentially multidimensional nature of health and "skill" (see eg. Heckman (2007)), and to represent differences in the distribution of skills within cohorts. I do not specify explicitly when or how these cohort differences develop, just that they originate before the age at which I begin measuring the outcome. There is a long history in social science as well as neuroscience and human biology of studying differences between cohorts and of the suggestion that they are likely to differ due to different experiences at "critical periods" in their life.[16] For example, the cohort differences could date to labor market entry, schooling age, infancy, or in utero.

In contrast, ϵ_{ap} represents the external factors such as technology or labor demand which will impact the outcome of a cohort who is age a in year p. The interaction between θ_c and ϵ_{ap} is completely unrestricted, so the model allows for example for a situation where the labor market for 30 year old workers would be a very good match for the skills of one cohort but a poor match for those of another cohort.

The model treats underlying cohort effects as fixed from the age at which the outcomes began to be measured. It therefore abstracts from investment in skills after that age and does not allow past shocks to have persistent effects on later outcomes.

In the context of this model, the concept of "cohort effects" can be generalized and linked to the idea of counterfactuals and potential outcomes[17]. For example, a natural way to summarize a decline in the health of cohorts born between 1947 and 1960 would be to ask: had both cohorts faced the set of external age-by-year factors actually experienced by the 1947 cohort how would

[16] See for example Ryder (1965); Easterlin (1987); **?**, and Cunha et al. (2006) and the citations therein.

[17] See eg. **?**

there mortality rates had differed? This is fundamentally a counterfactual question and involves the comparison of the observed outcomes for the 1947 cohort to a set of potential outcomes for the 1960 cohort which will never be observed. In particular, the outcomes Y_{apc} will only be observed for cohorts, ages, and periods such that $c = p - a$. The structural function $g(\cdot, \cdot)$ however is defined for all pairs of θ_c and ϵ_{ap} — defining "potential outcomes" which a cohort would have had if they had been exposed to different external factors. Identifying these generalized cohort effects therefore is akin to the "Fundamental Problem of Causal Inference" (Holland, 1986).

To identify evidence of a cohort decline in health and human capital from data on outcomes by age, year, and cohort will therefore require additional assumptions. Below, I pursue methods to identify evidence of this cohort decline, under increasingly weak assumptions about the nature of the external age-by-year factors.

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

I first present evidence of cohort declines in health and human capital under the assumption that the impact of age, year, and cohort are additively separable, such that:

$$Y_{apc} = \gamma_c + \phi_p + \alpha_a + \epsilon_{apc} \tag{2}$$

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

This is the traditional additively separable age-period-cohort models, common in economics and demography.[18] Conceptually, the cohort effects reflect the impact of underlying, fixed differences between individuals born in different years.

To identify the cohort effects, the above model assumes that the impact of external factors can be decomposed as additively separable year and age components. It is 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.

The additive separability in this model simplifies the counterfactual questions described above.

[18]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).

The difference between the cohort effect for the 1947 cohort, γ_{1947}, and that of the 1960 cohort, γ_{1960}, reveals how the outcomes of individuals born in these two years would have differed — holding all external factors fixed. Large differences in 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.

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 or two periods to have the same effect.

I will proceed in two directions. First, I will estimate "detrended cohort effects", which will reflect the true cohort effect, minus some unknown long-run trend in cohort effects [19] 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 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.

Second, I will consider what additional assumptions on either the age or period effects would imply about the long trend in cohort effects, and therefore about the entire sequence of cohort effects. Importantly, this will allow me to consider under what assumptions the data suggest there were *absolute* declines in health and human capital for cohorts born after 1947.

For both approaches a few additional definitions are helpful. Denote the first cohort included in the model as 1 and the last as C . Similarly, denote the first year as 1 and the last as P. And finally, the first age included in the model as 1 and the last as A.

Then, define linear "trends" in cohort, year, and age effects as follows:

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

For the year effects, this trend is the average annual change in effects between the first and last year in the sample. Analogously, for cohort effects it reflects 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 it would represent how much health or human capital had improved or declined on average between each cohort from those born in 1930 to those born in 1965. Similarly, for age effects it reflects the per-single-age difference between the effects for the youngest and oldest ages in the sample.

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

[19] This is similar to approaches in Deaton (1997); Chauvel (2011).

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

And finally define two composite linear "trends":

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

One can then rewrite the model in Equation 2 as:

$$Y_{apc} = \tilde{\gamma}_a + \tilde{\phi}_p + \tilde{\delta}_c + \tilde{\beta}_{ac} \cdot a + \tilde{\beta}_{pc} \cdot p + \epsilon_{apc} \tag{3}$$

This reparameterized model is identified — the design matrix is full rank. One can identify detrended age, period, and cohort effects. Additionally, one can identify two composite linear trends which represent i) the sum of the period and cohort trends, and ii) the age trend minus the cohort trend. By making additional assumptions on age or period trends one can identify or bound the cohort trend, and therefore the full set of cohort effects.

A. Detrended cohort effects

First, I estimate the reparameterized version of Equation 2 by linear regression, and will focus on the detrended cohort effects $\tilde{\gamma}_c$. This can be thought of as normalizing both the first and last cohort effect to be 0. 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. Each detrended cohort effect represents the true cohort effect minus the long-run linear "cohort trend."

Results

Figure 2 shows the results of estimating the detrended, additively separable age-period-cohort models of Equation 3 for the six main outcomes: men's median log earnings, the low birthweight rate by mother's birth cohort, the log mortality of men and women, and the average years of schooling of men and women. The estimated cohort effects for each of the different outcomes 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.[20] 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

[20]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.

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.

Panels C and D show similar results for the average years of schooling of men and women, respectively. Again, the estimated cohort effects appear approximately piecewise linear in shape, with a large slope change precisely at the 1947 cohort. For men, the cohort effects increase from a normalized 0 in 1930 to .83 by 1947, before suddenly changing slope and declining for subsequent cohorts. For women the pattern is simlar though the differences in cohort effects are smaller in magnitude; and the 1947 trend break is smaller in magnitude but followed by a second trend break in the early 1950s (similar to the pattern for women's log mortality).

B. Possible cohort effects under additional assumptions

The above approach does not identify the long-run trend in cohort effects, and therefore cannot speak to whether cohort health and human capital declined in absolute terms. The absolute declines in educational attainment could be viewed as prima facie evidence of absolute declines in human capital. In this section for the other outcomes I show what different additional assumptions about age or year effects would imply about the sequence of cohort effects. This allows me to assess whether health and human capital declined in absolute terms, under different assumptions. The approach is similar in spirit to that in Lagakos et al. (2018), and to the bounding ideas in Fosse and Winship (2017).

Restrictions on period trend

For low birth weight and log mortality I proceed by considering the implications for cohort effects of different restrictions on the period trend. Lagakos et al. (2018) call this the "Deaton-Hall" approach in reference to Hall (1968) and Deaton (1997). The basic idea is that by restricting the trend in period effects in different ways, one can pin down the trend in cohort effects based on the composite period/cohort trend defined above.

I will consider three benchmark cases: i) assuming that the period trend, β_p, is equal to 0, ii) assuming that the cohort trend, β_c, is equal to 0, and an intermediate case iii) assuming that β_p and β_c are equal.[21] Of course many other cases are possible, but examining what these assumptions imply about the sequence of cohort effects can help to make the mechanics of the model and the identification problem clear.

Additionally, it is arguably natural to think of case i) as an upper bound on the period trend, and therefore a lower bound on the cohort trend. For example, for low birth weight assuming

[21] Note that case ii) will yield the "detrended" cohort effects presented above.

$\beta_p \leq 0$ is assuming that average growth in period effects between 1968 and 1995 is non-positive, in other words that over this long period there was at a minimum not a worsening of external, non-cohort factors which impact the low birth weight rate. For mortality analogously, it would be assuming that between 1975 and 2019 the external environment impacting mortality rates did not worsen. Assuming $\beta_p \geq 0$, implies that $\beta_c \leq \tilde{\beta}_{pc}$ — placing a lower bound on the trend in cohort effects. This corresponds to the cohort trend in case i) above. Therefore we can view the sequence of cohort effects in case i) as a "best case" scenario for cohort effects, in the sense that they represent the most that cohort effects impacting low birthweight or mortality could have declined between the 1930 and 1965 cohort (or any two cohorts for that matter). Under this assumption the true sequence of cohort effects must lie above the case i) sequence, with the case ii) and iii) sequences representing two other possible sequences above the lower bound.[22]

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

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

No growth in wage age effects after 50

When examining median log wages, it is not as natural to think of zero growth in period effects as a plausible upper bound. I therefore proceed by making different assumptions on the age effects, and showing the implications of these assumptions for the sequence of cohort effects.

In particular, I adopt what Lagakos et al. (2018) call the Heckman-Lochner-Taber approach in

[22]Note that case ii) is not necessarily an upper bound on the cohort trend. It is possible that the long-run cohort trend in positive, ie. that cohort health has declined over the full sample.

reference to Heckman et al. (1998).[23] Economic theory predicts very little or no growth in wages in the last few years of a workers career. Interpreting the age effects as representing growth in wages due to human capital growth over a workers career, then the argument is that the incentive to invest in human capital growth is very low late in a workers career. Therefore, the age effects at these ages will be zero, or slightly negative if human capital depreciates. Lagakos et al. (2018) emphasize that the same prediction holds for the other two main mechanisms for life cycle wage growth emphasized in the literature: search and learning.

I implement this approach by first estimating a restricted version of the age-period-cohort model in Equation 2. First, I restrict the age effects from ages 50 to 54 to be equal to 0.[24]. This represents a first benchmark case i) of no growth in age effects between ages 50 and 54, with no depreciation of human capital. I then consider two additional cases in which human capital depreciation (or other factors) lead age effects between age 50 to 54 to i) decline by .005 per single-year-of-age, and ii) decline by .01 per single-year-of-age. Because the model is estimated on log wages these correspond to human capital depreciation rates of .5 $\%$ and 1 $\%$ respectively (no depreciation and 1 $\%$ are benchmark cases considered in Lagakos et al. (2018)).

The cohort effects implied by these three scenarios are shown in Panel A of Figure 3. All three scenarios show declines in cohort effects between the 1947 and 1965 cohorts — implying that median labor market ability declined across these cohorts. The magnitude of the decline differs substantially across the different assumptions. The bottom series shows cohort effects under the assumption that age effects are constant between ages 50 and 54, ie. that human capital does not depreciate. This series would imply that labor market skill declined between the 1947 and 1965 cohorts enough to reduce log wages by approximately .16. The middle series shows cohort effects under the assumption that age effects decline .005 per year for ages 50-54, ie. that human capital depreciates by .5 $\%$ per year. This assumption would imply instead that labor market skill declined between the 1947 and 1965 cohorts enough to reduce log wages by around .11. Finally, the top series shows cohort effects under the assumption that age effects decline .01 per year for ages 50-54, ie. that human capital depreciates by 1 $\%$ per year. This assumption of course does not change the size of the trend break in cohort effects, but because it implies a larger long run trend in period effects, it implies a much smaller absolute decline in cohort effects between the 1947 and 1965 cohorts, of around .02.

[23] They also reference the following papers as employing variants of the same idea: McKenzie (2006); Huggett et al. (2011); Bowlus and Robinson (2012); Schulhofer-Wohl (2018).

[24] Note, I do not omit the age 25 fixed effect in this specification. Also, note that this is a stronger assumption than is necessary to identify the sequence of cohort effects. It is chosen to correspond approximately to the benchmark case in **?** of no experience effects in the last 5 years of a workers career.

C. Summary

The above results from the age-period-cohort models 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. The first focuses on identifying 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.

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

In this section I present evidence of a decline in cohort health and human capital using a novel methodology, which allows 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. This approach yields similarly strong evidence, for all outcomes, of large changes in the slope of cohort effects at or near the 1947 cohort.

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, which restricts the general model from Equation 1:

$$Y_{apc} = \underbrace{\beta \cdot c}_{\text{long-run trend in cohort effects}} + \underbrace{\mathbb{1}_{c \geq \lambda} \cdot \delta \cdot (c - \lambda)}_{\text{change in slope of cohort effects}} + \underbrace{f^p(a)}_{\text{year-specific impact of age}} + \epsilon_{apc} \tag{4}$$

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.[25]

I will directly estimates the model given in Equation 4 in which the cohort effects are specified to have a piecewise linear shape. I 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. When $f^p(a)$ is specified as additively-separable age and year effects, then the model in Equation 4 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, 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 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. 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.

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

[25] 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, 2023).

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.

Results

A graphical depiction of the estimation and inference of the location of the cohort slope change, λ, is shown in Figure 4. 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, $\hat{\lambda}$. 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 1 provides the full results of estimating the piecewise linear cohort effects based on Equation 4, fore five 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 Table A1 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. The results are generally quite robust.[26]

7. Non-parametric evidence of cohort decline

The writing of this section is in progress. In future drafts it will outline a novel, nonparametric generalization of McKenzie (2006) which identifies second differences in "local average cohort effects" under the assumption that age-by-year shocks are "locally independent". Where I define "Local average cohort effects" as the counterfactual average outcomes a cohort would have if they had experienced the external age-by-year factors of a cohort born one year earlier or later. It will then show the results of applying this methodology to test for evidence of the non-parametric equivalent of a trend break in cohort effects.

[26]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.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 (2023) 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.

8. 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 of men and women 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 4. 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 the other outcomes, I use estimates from the baseline specification reported in Table 1. 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 5 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.[27] This measure of earnings declines in real terms

[27] 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.

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), Figure A1 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.

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. Figure A1 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, 2023).

9. Evidence suggesting decline originated in childhood

This section presents three sets of results suggesting that the cohort decline likely originated in childhood or adolescence. First, growth and physical development stagnated, and in some dimensions appears to have declined, beginning with approximately the same cohorts. Second, scores on standardized tests taken as early as 16 declined for the same cohorts. Third, the decline is concentrated among the US-born population with no evidence of a similar cohort decline for the

foreign-born population, in outcomes where it is possible to distinguish.

A. Growth and physical development

Previous research has found that increases in adult height, often viewed as a proxy of childhood nutritional status (Floud et al., 2011; Tanner, 1990), suddenly stopped for Americans born between approximately 1955 and 1974 while the height of Europeans continued to grow rapidly (Komlos and Lauderdale, 2007a,b; Komlos, 2010).

I build on these findings for adult height and also examine height and physical development of these cohorts as children. I combine historical data compiled in Meredith (1964) with more recent survey data to show that for white males a sudden stagnation in heights is evident at age 6 and age 10, beginning with approximately the 1950s cohorts. This stagnation represents a sudden stop to relative constant growth between 1870 and around 1950. And there is little sign of renewed growth even with the most recent cohorts born after 2010. I also show preliminary evidence of an absolute decline in the size and/or tempo of the adolescent growth spurt: males in cohorts born in the 1960s appear to have grown more slowly in adolescence than those born around a decade earlier.

Stagnating adult height

Following Komlos and Lauderdale (2007b,a); Komlos (2010), I combine data from various waves of the nationally representative National Health and Nutrition Examination Survey (NHANES) and it's precursor conducted in various years since 1959. As in these past articles I focus on adults age 23 to 47, ages at which height is approximately constant, individuals born in the United States, and use survey weights adjusted for the combined surveys as suggested in Korn and Graubard (1999). I include 12 more years of data than Komlos (2010), bringing the data up to 2018.

Figure 6 panels A and B show binscatter plots of height by birth cohort for men and women (Cattaneo et al., 2019). The sample sizes are much smaller than those for the main outcomes examined above, leading to imprecision but some patterns emerge. For men, an increasing trend from the 1920s cohorts to approximately the 1950s cohorts is clearly evident — followed by a stagnation beginning at some point around the 1950 cohort and basically constant heights thereafter. The pattern for women is much noisier and fits less clearly the pattern of a trend break around the 1947 cohort — it appears that heights continued to increase until the mid-to-late-1950s cohorts before stagnating. Estimating simple trend break regressions and imposing a trend break in 1947 yields for men: an estimated pre-1947 trend of around .4 inches of growth per decade, a large trend break and a trend of only around .02 inches of growth per decade after the 1947 cohort (indistinguishable from zero). For women the same estimates suggest increase of nearly a quarter of an inch per

decade before the 1947 cohort, and then only .06 inches per decade thereafter.

Estimations of Hansen-style trend break models with an unknown break location, as above, are imprecise but confidence intervals for the break location include the 1947 cohort for both men and women.

Stagnating childhood height

Growth in childhood height also appears to have stagnated around the 1950 birth cohort, at least for white boys. Surprisingly this was already noted in the 1970s by reports of the National Center for Health Statistics (Hamill et al., 1977b,a). The report describing new child growth curves estimated based on data from NHES and NHANES surveys (Hamill et al., 1977a) reads: "In the analysis of these data, the marked diminution and near cessation of the trend to constantly increasing size of successive generations of American children is the most dramatic and significant finding relating to human biology and human growth in general." Trends in childhood height need not exhibit the same pattern as adult height. Height at various ages in childhood depends on so-called growth tempo, while adult height depends on both growth tempo and the age at which growth stops (Tanner, 1990).

Panels C and D show evidence of this stagnation in heights of white boys at age 6 and age 10. The pre-1950 data come from various historical studies which were compiled in Meredith (1964). They do not come from a nationally representative sample, but also did not explicitly target high or low socioeconomic status children, for example. Each point shows the mean reported in Meredith (1964) for an interval of birth cohorts, plotted at the middle cohort of the interval. The post-1950 data show sample means for white non-Hispanic males from the combined NHES and NHANES data for 5-year birth cohort intervals, again plotted at the middle cohort. At age 6 there is clear visual evidence of an increasing trend in heights from the 1870 to approximately 1950 cohort, from 43 to more than 47 inches. However, this growth suddenly stagnates and for the next 60 cohorts there is little or no growth in heights. The estimated trend in height for the pre-1950 cohorts is .45 inches per decade, but slows to an estimated trend of only .09 inches per decade after 1950.

Smaller adolescent growth spurt and later leg growth

The NHES and NHANES samples are all small compared to those used for other outcomes and the early surveys occur at irregular years and for varied and selected ages. However, (by lucky coincidence) it is possible to observe heights from age 6 though adulthood of the cohort born between 1951-1957 and compare them to heights at the same ages for those born 1960-1967, for relatively large samples.[28]

[28]The second NHES surveys was carried out in 1963-1965 and only sampled 6-11 year olds, and the third was carried out in 1966-1970 and only sampled 12-17 year olds. Therefore in these two surveys one can observe the 1952 to 1957 cohorts heights at various ages of childhood for relatively large samples. NHANES 1 and 2 sampled ages 1 (or zero) to 74 and were carried out in 1971-74, 1976-80 and allow one to observe the 1960-1967 cohort at similar ages and again with relatively large sample sizes. The remaining surveys allow one to observe adult heights.

I use regressions of the following form to estimate differences in height for different age groups:

$$Y_{i,ac} = \gamma_{c=1960-66} + \mu + f(a) + \epsilon_{i,ac} \tag{5}$$

where $Y_{i,ac}$ denotes height of individual i at age a in cohort c. I run the above regression separately for different age groups: 6 to 11, 12 to 17, and 23 to 40. In each regression $\hat{\gamma}_{c=1960-66}$ estimates height differences between cohorts, and $f(a)$ controls for small differences in the distribution of ages when the cohorts are observed in the different surveys. For children I include dummies for age in years and a quadratic in age in months. I restrict to the native-born population and use sampling weights in all analysis.

Panel E of Figure 6 shows the results. It reports the estimated difference in height between the 1951-1957 and the 1960-66 cohorts of males, $\hat{\gamma}_{c=1960-66}$ for different age groups. The estimated differences in height are small and statistically indistinguishable from zero for age 6 to 11 and in adulthood. However, I estimate a statistically significant difference in height of *nearly half an inch* at ages 12 to 17.

These results imply that the two cohort groups were nearly identical in height as children but the later born cohort had a much smaller or later growth spurt in adolescence — causing them to fall behind in height at ages 12 to 17. However, the later born cohort then caught back up in height in adulthood.

I estimated additional models to directly estimate and gauge the statistical significance of the differential growth in height implied by these patterns. For the sample of 6 to 17 year olds I estimate:

$$Y_{i,ac} = \gamma_{c=1960-66}^{a=12-17} + \mu + f(a) + \epsilon i, ac \tag{6}$$

And for the sample of 12 to 17 and 23 to 40 year olds I estimate:

$$Y_{i,ac} = \gamma_{c=1960-66}^{a=23-40} + \mu + f(a) + \epsilon i, ac \tag{7}$$

Panel F of Figure 6 shows the results. It reports the estimated difference in the implied growth in height between the 1951-1957 and the 1960-66 cohorts from ages 6-11 and 12-17, $\hat{\gamma}_{c=1960-66}^{a=12-17}$, and from ages 12-17 and adulthood, $\hat{\gamma}_{c=1960-66}^{a=23-40}$. The differences in growth are statistically significant. The point estimates imply that the later born cohort grew .35 inches less between ages 6-11 and 12-17, but then grew .43 inches more between ages 12-17 and adulthood.

Overall the patterns appear consistent with the males in these cohorts having a delayed and/or smaller adolescent growth spurt but then growing longer into early adulthood, allowing them to "catch-up." This could point towards a hormone or nutrient deficiency in adolescence, either due

to an exposure occurring during adolescence or an exposure which particularly impacts hormones and biological processes that impact the adolescent growth spurt. J.M. Tanner, an expert in human growth cited often by Robert Fogel and other economists using height as a proxy for childhood health, described human growth as a "target-seeking function" (Tanner, 1986, 1990). Each person has a target growth path and if they are knocked off that path by lack of nutrition or sudden hormone deficiency, then when the nutrition or hormone supply is restored they will exhibit "catch-up growth" — growing faster than they would have until they catch up to their "target" growth path. Additionally, it is common for individuals or populations who have a later or smaller growth spurt in adolescence to then continue growing for longer into their early twenties, at least partially catching up in adult height. This later growth also generally particularly occurs in the legs as opposed to the upper body — due to later ossification of the bones (Tanner, 1990). Preliminary results show that the height patterns shown in Panel B and C are nearly entirely driven by differences in leg length, rather than trunk length.

There is no evidence of a similarly delayed or slower adolescent growth spurt for females in these cohorts. In future drafts I plan to examine other measures of growth and physical development which are recorded in the NHES and NHANES surveys for males and females in these cohorts, such as: "bone age", a measure of physiological maturity derived from x-rays which could help to pinpoint the source of the slower adolescent growth spurt; and age at menarche and other measures of puberty and sexual maturity.

B. Test score decline

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. Figure A4 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 in Iowa and Minnesota (Harnischfeger and Wiley, 1975).

C. 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.

Figure 7 shows detrended cohort effects separately for foreign and native-born mothers, based on additively-separable age-period-cohort models.[29] As for the earlier figures, the models are based on Equation 2, 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.

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

[29] 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.

may not exist.

Panel A of Table 2 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.

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 two reasons. First, the Census data only records outcomes every 10 years — hampering the ability to distinguish 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, Table A2 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.[30] 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.[31] However,

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

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

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.

10. Preliminary investigation into root cause

A. Link between educational declines and other declines

In general, the educational declines for the post-1947 cohorts occurred before their declines in other outcomes. A natural question is therefore whether the direct causal effect of education on earnings and health can explain the other declines in health and earnings documented above. Appendix A describes results link between the educational declines and subsequent declines. The evidence is not definitive but it appears unlikely that the educational declines could be causing all of the subsequent declines. It therefore appears more likely that a broader decline in health and human capital caused both the educational declines and the subsequent declines.

Two main findings detailed in Appendix A point against such an "education-only" explanation. First, the increase in log mortality is too large to be explained by the decline in earnings, unless one assumes the causal effect of education on mortality is much larger than commonly found. Second, a large cohort decline is evident at or very near the 1947 cohort in conditional maternal health (as proxied by infant birth weight) across the maternal educational distribution, from mother's with less than high school to those with more than 5 years of college education. In principle this could be explained by complex changes in the selection of women by maternal health after the 1947 cohort — but a broad decline in health appears more likely.[32]

B. Decline widespread among native-born

With the exception of the foreign-born population (shown above), 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.

[32] Interestingly, the cohort decline in earnings appears to be concentrated among those without a Bachelor's degree, suggesting the cohort decline could contribute to the college high-school wage gap.

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 4, 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).[33]

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 de-

[33]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).

clines 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 4, separately for each Census Region.[34] 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.[35]

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.

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.

Table A5 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 4, 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.

By Commuting Zone

To be added in future drafts.

C. Evidence against some plausible causes

I briefly present evidence that appears inconsistent with a few plausible hypotheses. First, Figure A5 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

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

[35] 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.

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 A7 shows that rapid improvements in the infant mortality rate beginning in the 1930s suddenly *slowed* beginning in the late-1940s — interestingly there is also a sudden relative increase in comparison to other high come countries. Similarly, Panel C of Figure A7 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 Panels A and B of Figure A6, 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 (Easterlin, 1987; 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.

The existing evidence on trends in smoking by women of childbearing age also make maternal smoking appear an unlikely driver of the cohort health decline.[36] There does not appear to be survey evidence on *maternal* smoking specifically for this period, but the pattern of smoking rates by women of childbearing age do not show any evidence of a sudden increase after the late-1940s. Figure A9 shows estimates of smoking rates by women of childbearing age from **?**, which show a slowing of growth in smoking rates after 1945.

A number of common forms of pollution do not appear likely to be important causal factors. Figure A10 show trends in eight important air pollutants from the Community Emissions Data System (O'Rourke et al.). None of them show evidence of trend breaks leading to more rapid increases after the late-1940s.

D. Fetal lead exposure from motor vehicle gasoline

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

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

Automobile production and fuel use were restricted during World War II and began to rapidly increase soon after. Figure A11 shows time series of i) motor vehicle registrations and ii) fuel usage by motor vehicles, for 1930 to 1970 from Historical Statistics of the United States (US Census Bureau, 1975). Both series exhibit slow growth between 1930 and 1941, and declines during World War II. They then exhibit sharp trend breaks near 1945 and very rapid grwoth until 1970.

Exposure to pollution from lead added to gasoline also likely increased rapidly over this period. Lead concentrations of automotive gasoline decreased during WWII because the lead additive was needed for aviation gasoline for military planes (Oudijk, 2010). The lead content of gasoline generally increased after this period, and with the rapid increase in total fuel consumption the total tons of lead added to gasoline consumed in the US increased rapidly.[37] When lead additives were phased out of gasoline beginning in the 1970s blood lead levels of children fell rapidly.

Fetal and early life lead exposure has been linked to poor health and cognitive development. Early life lead exposure is thought to have broad and lasting negative health effects, impacting for example the development of multiple organ systems, cognitive ability and emotional regulation, and cardiovascular disease.[38]

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

The facts above could point towards fetal lead exposure in particular as a possible driver of the health decline. However, the timing appears off by one cohort: the timing of the trend break in automobiles — of 1945 — would lead to a trend break in fetal exposure to automobile-based lead pollution at approximately the 1946 cohort. There is evidence that lead is eventually stored in bones and remobilization of this lead during pregnancy and lactation is a potentially important source of toxins for fetuses and infants (Silbergeld, 1991; Gomaa et al., 2002). So perhaps the lag

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

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

is due to time for the lead to be stored in women's bones, though this is conjecture. The storage and remobilization of bone lead could provide an explanation for why there is not a sharp improvement in outcomes with the phaseout of lead. Note that if *childhood*, rather than fetal, lead exposure has particularly large effects it would generate a trend break earlier. For example, if all ages under 5 represented a critical period for lead exposure we would see health declines beginning with around the 1940 cohort, who would have been around 5-years old in 1945. Of course much more research is needed to establish or falsify the importance of fetal lead exposure for the documented health decline.

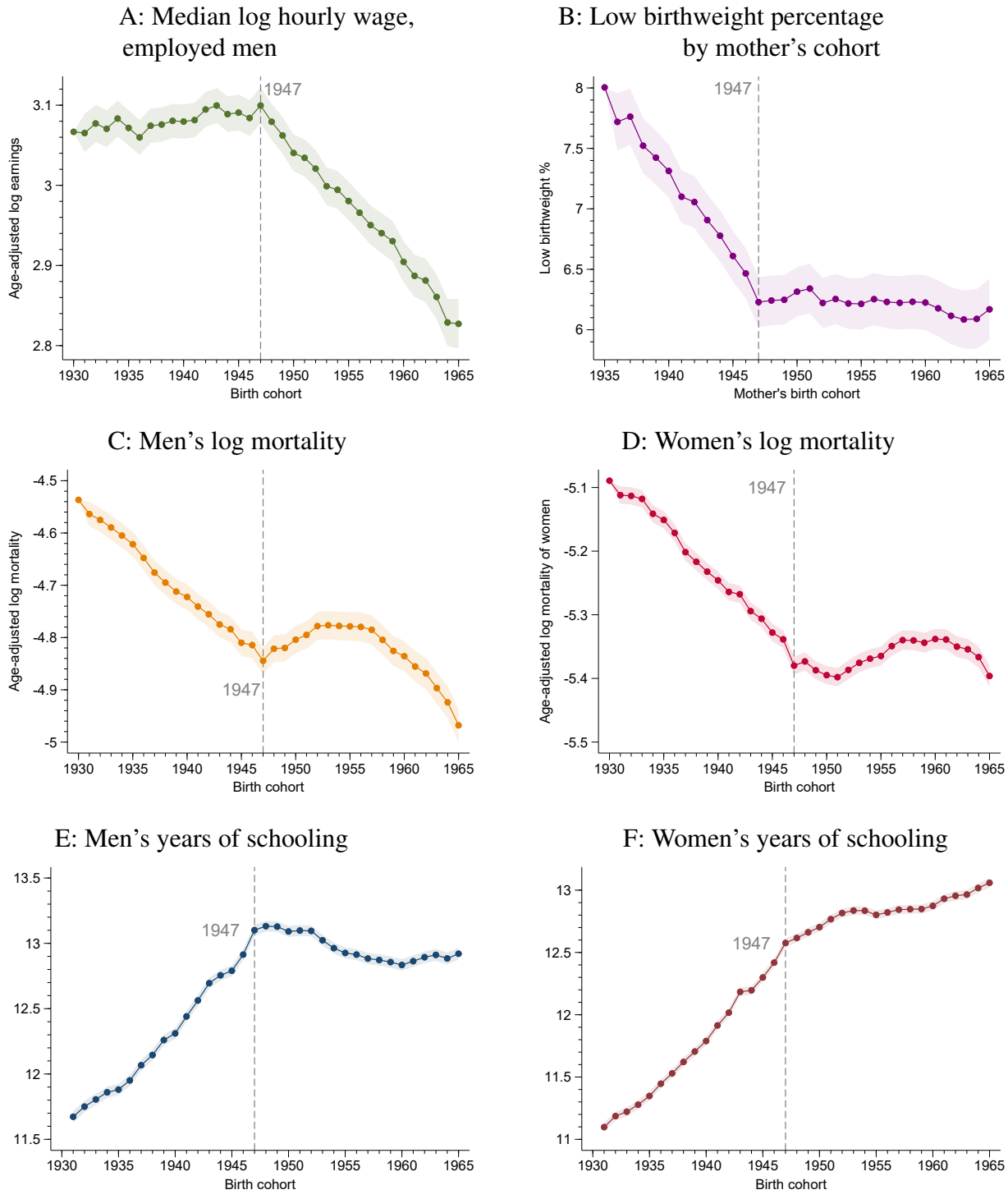
11. Conclusion

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 early in life 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. There appears to be surprisingly little variation across states or racial groups in the size of the decline. It will therefore be necessary to compile data which allows for examination across additional dimensions, such as county-of-birth, parental characteristics, birth order, or family size.

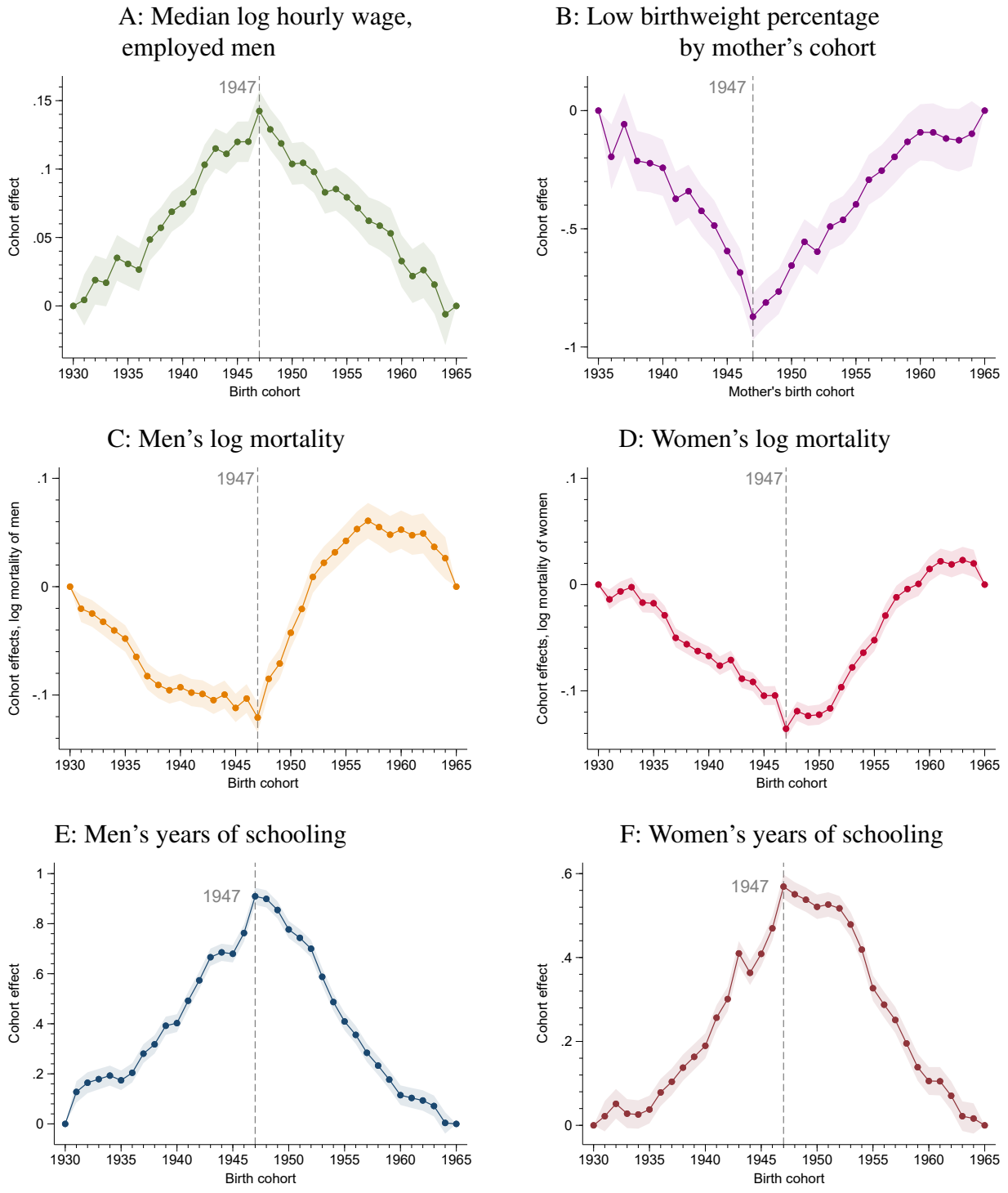
The documented health and human capital decline may also be evident in other outcomes not examined in this paper. Secular changes over time in outcomes such as labor force participation or medical spending may be the result, at least in part, of cohort differences in health and human capital rather than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



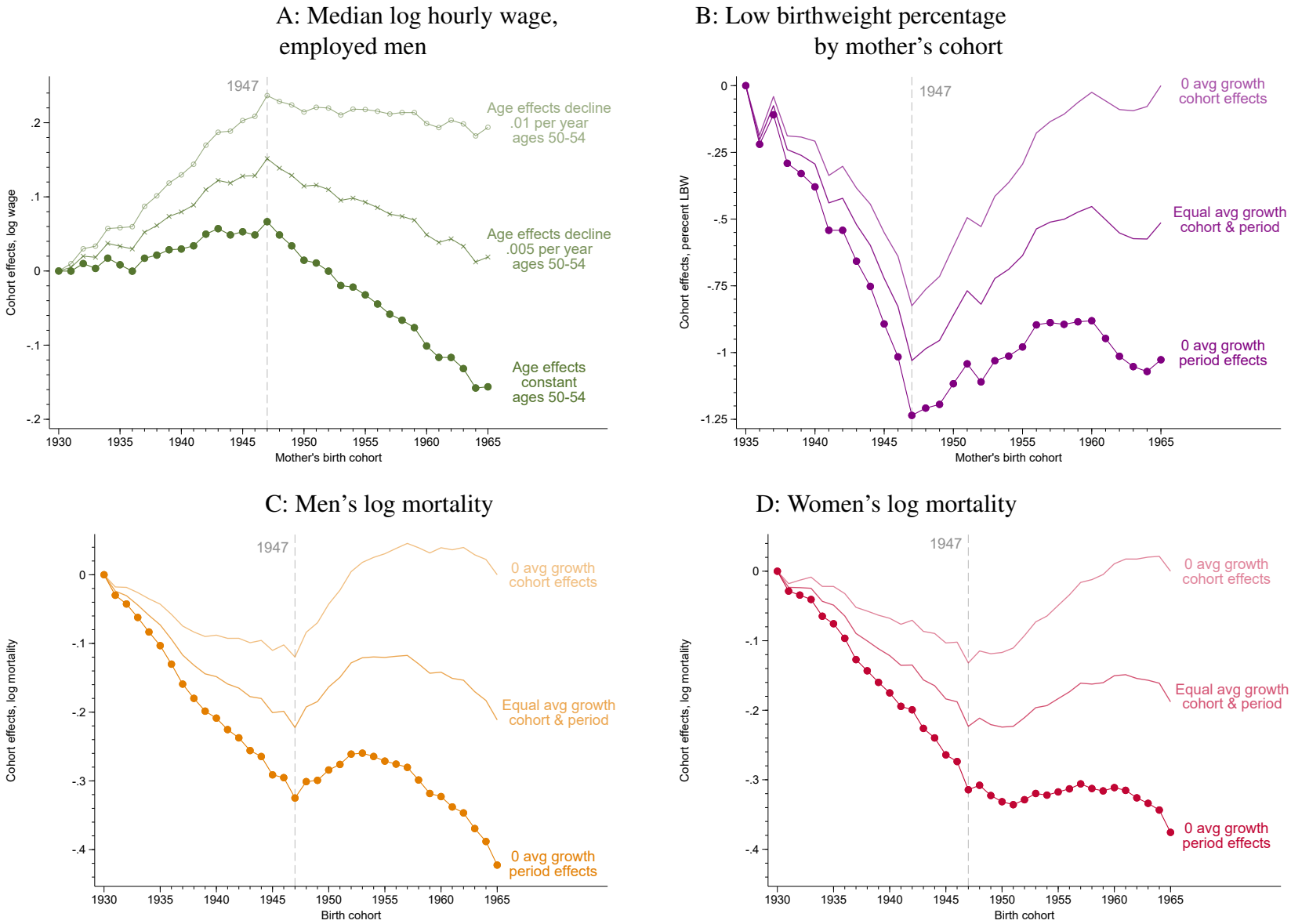
Each panel plots age-adjusted outcomes by birth cohort. Age adjustment is done by regressing the outcome on a full set of cohort fixed effects and age effects, and plotting predicted values. Panel A is based on CPS-MORG data, 1979-1993, and includes men age 25-54, who were born from 1930 to 1965. Panel B is based on vital statistics natality microdata, 1968-1990, mothers age 18-40, who were born from 1935-1965. Panel C and D are based on data from the Human Mortality Database, and include 1975-2019, ages 25-85, cohorts born from 1930 to 1965. Panels E and F are based on CPS-MORG data, 1979-2016, and includes men and women aged 25-75, who were born from 1930 to 1965.

Figure 2: Detrended cohort effects from additively separable age-period-cohort models



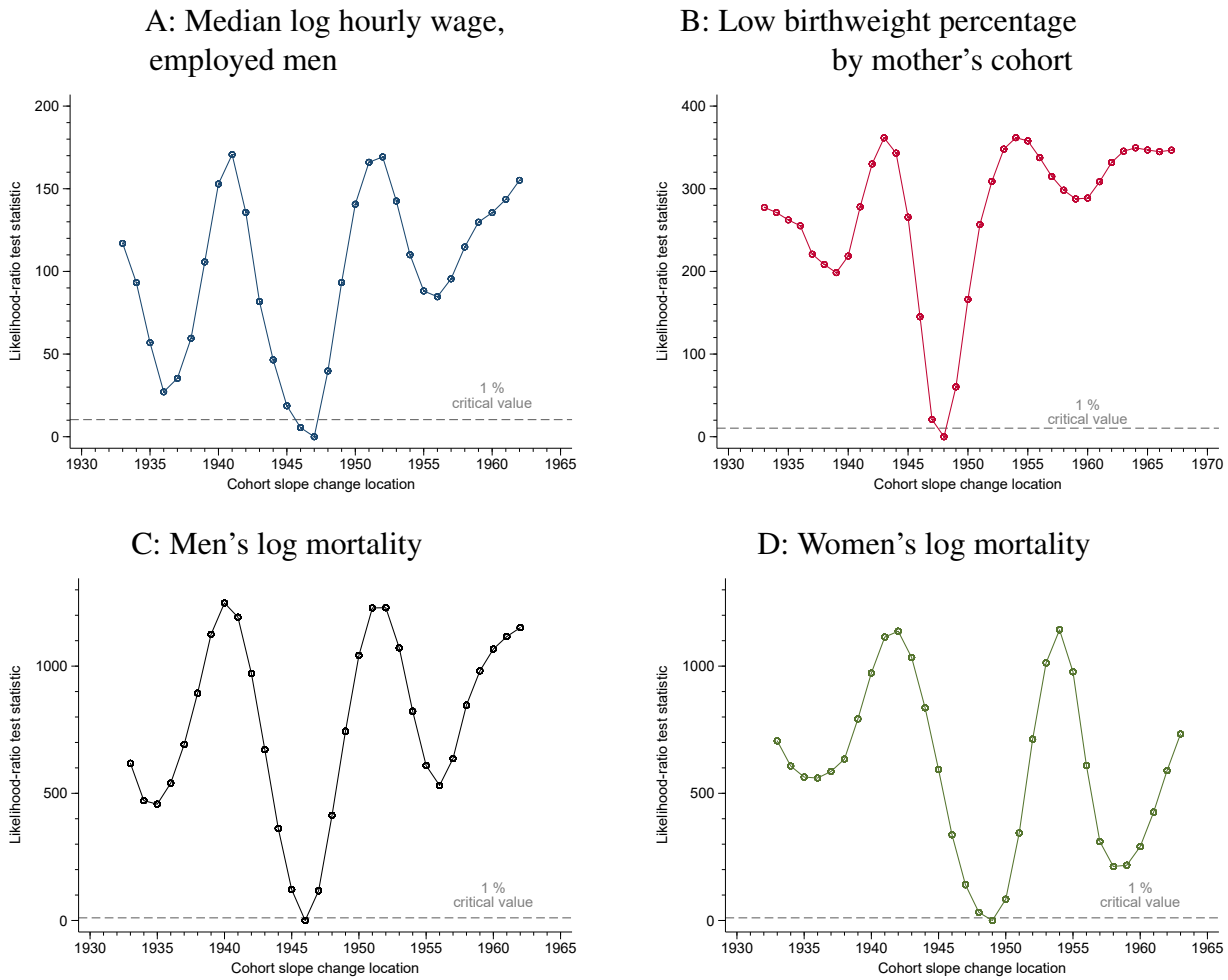
Each panel plots detrended cohort effects from estimation of age-period-cohort models based on Equation 1. Panel A is based on CPS-MORG data, 1979-1993, and includes men age 25-54, who were born from 1930 to 1965. Panel B is based on vital statistics natality microdata, 1968-1990, mothers age 18-40, who were born from 1935-1965. Panel C and D are based on data from the Human Mortality Database, and include 1975-2019, ages 25-85, cohorts born from 1930 to 1965. Panels E and F are based on CPS-MORG data, 1979-2016, and includes men and women aged 25-75, who were born from 1930 to 1965.

Figure 3: Cohort effects under different assumptions, additively separable 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 CPS-MORG data, 1979-1993, and includes men age 25-54, who were born from 1930 to 1965. Panel B is based on vital statistics natality microdata, 1968-1990, mothers age 18-40, who were born from 1935-1965. Panel C and D are based on data from the Human Mortality Database, and include 1975-2019, ages 25-85, cohorts born from 1930 to 1965. Panels E and F are based on CPS-MORG data, 1979-2016, and includes men and women aged 25-75, who were born from 1930 to 1965.

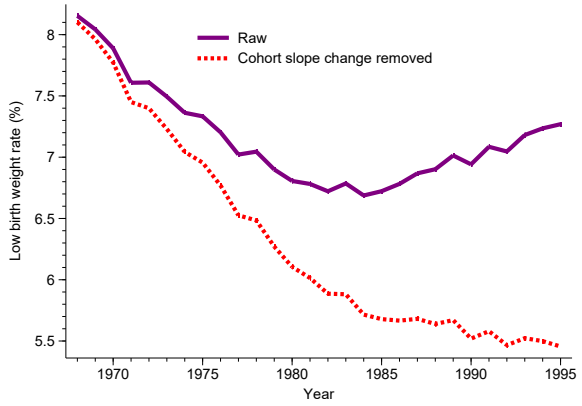
Figure 4: Hansen-type estimation of cohort at which decline begins controlling for separate quadratic-in-age in each year



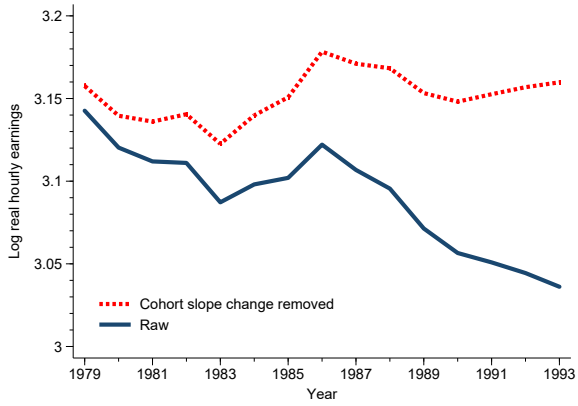
This figure shows estimation and inference of the location at which a cohort decline begins based on the model in Equation 4, with age fixed effects, year fixed effects, and separate quadratic-in-age in each year included as controls. Each plot shows the likelihood-ratio test statistic suggested in Hansen (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 2.

Figure 5: Impact of cohort decline on year-over-year declines

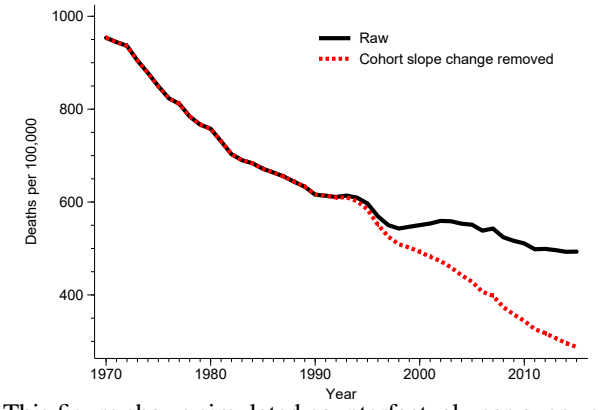
A: Low birthweight (%)



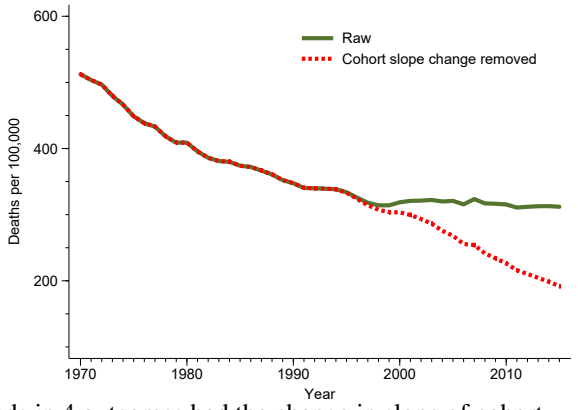
B: Median log hourly wage, employed men



C: Men's mortality, age 45-54

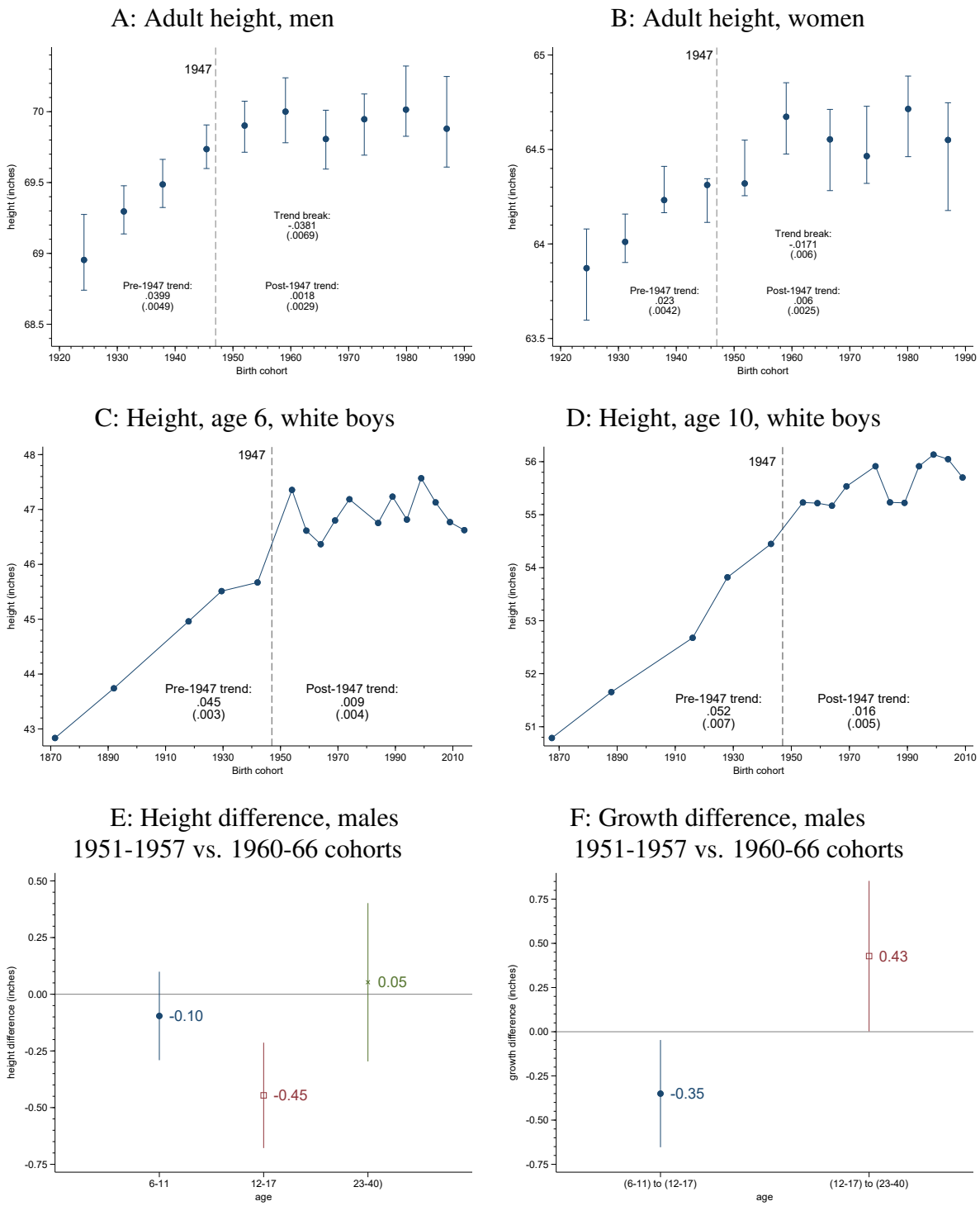


D: Women's mortality, age 45-54



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 4 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 and 3.

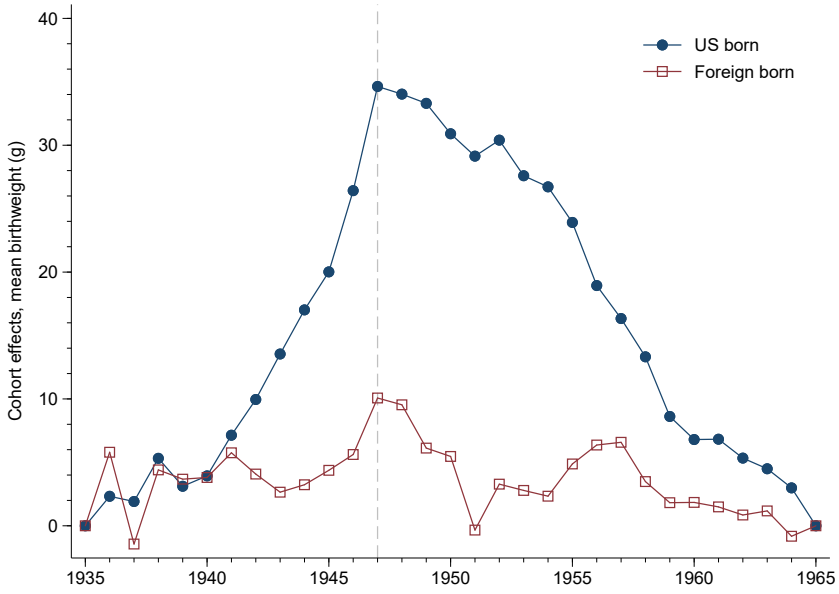
Figure 6: Growth and physical development stagnated (and may have worsened) for approximately same cohorts



All panels use data from multiple rounds of NHES and NHANES surveys. Panels A and B show binscatter plots following Cattaneo et al. (2019) of heights of men and women ages 23 to 47. Panels C and D plot the height of white boys age 6 and 10 respectively. Pre-1950 data comes from non-representative historical studies compiled by Meredith (1964), with each point representing the mean for an interval of birth cohorts given in Meredith (1964), plotted at the midpoint. Post-1950 data comes from the NHES and NHANES surveys and shows the mean for 5-year birth cohorts. Panels E and F show estimates of the height difference of the listed cohorts at different ages and the difference in implied in implied growth between different ages, estimated from the regressions described in the text.

Figure 7: Intergenerational infant health decline only evident for native-born

Detrended cohort effects — by mother's place of birth



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. The figure plots detrended cohort effects from estimation of age-period-cohort models based on Equation 1 — separately for the two groups of mothers.

Table 1: 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 4, 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 1975-2019, ages 25-85, cohorts born from 1930 to 1965.

Table 2: 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 4, 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 3: 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 4, 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 1975-2019, 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 4: 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 4, 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 1975-2019, ages 25-85, cohorts born from 1930 to 1965.

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Appendix A: Connection between educational declines and other declines

A. 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.[39]

To calculate the "reduced-form" I simply apply Equation 4 to each outcome, and estimate the location 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 4 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." [40]

Table A6 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

[39]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.

[40]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).

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[41]. 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 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).

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

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.

B. 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 A7 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 4. 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. [42]

Panel B of Table A7 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

[42] 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).

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.

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 estimate models on 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.[43]

Table A7 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 A8 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 4 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 A4 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.

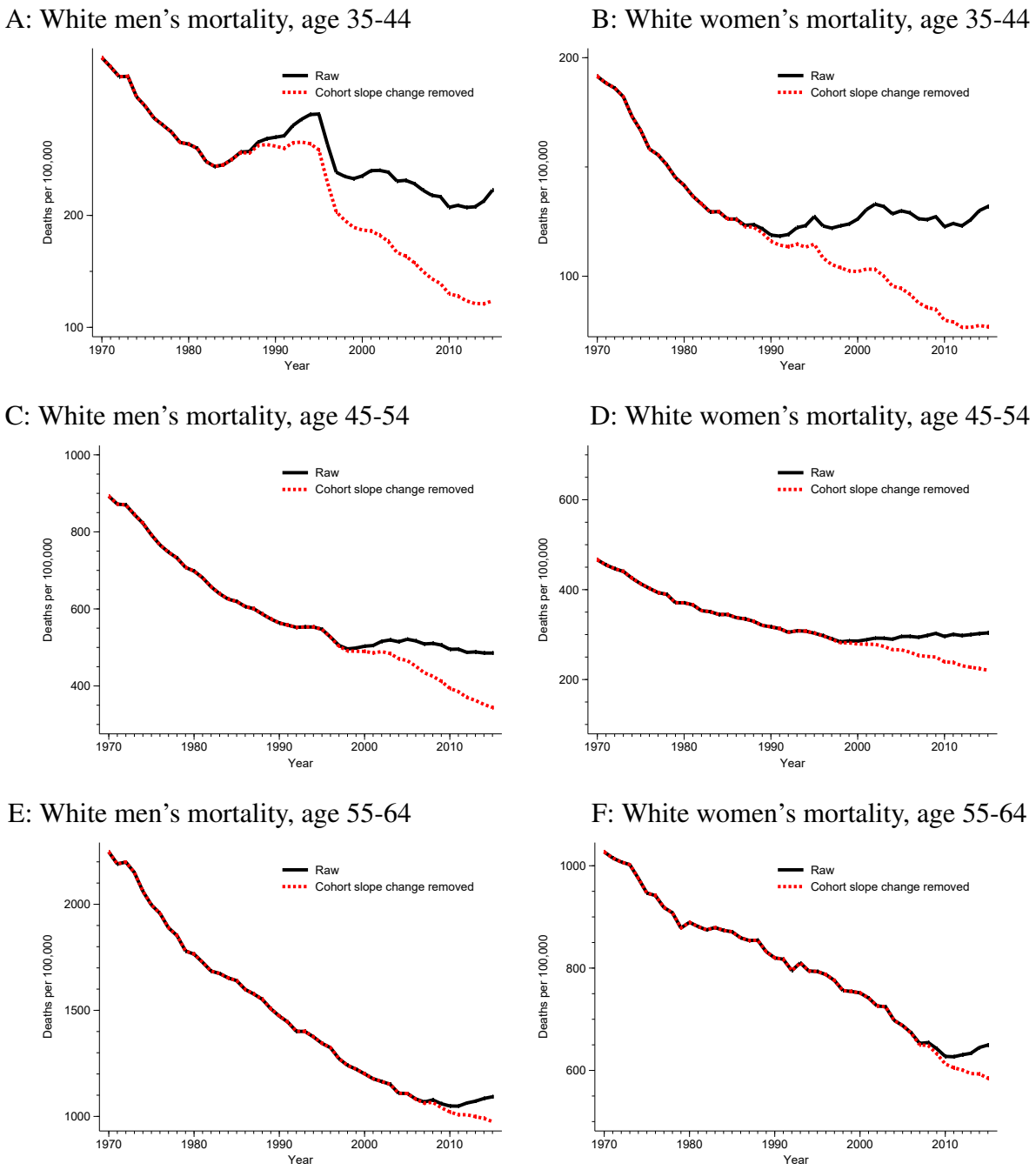
The above results show strong evidence of cohort declines in maternal health *conditional* on mother's education across the education distribution. These results would rule out an explanation

[43]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.

in which a change in the supply of education alone has driven maternal health declines, if that change in supply affected 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.

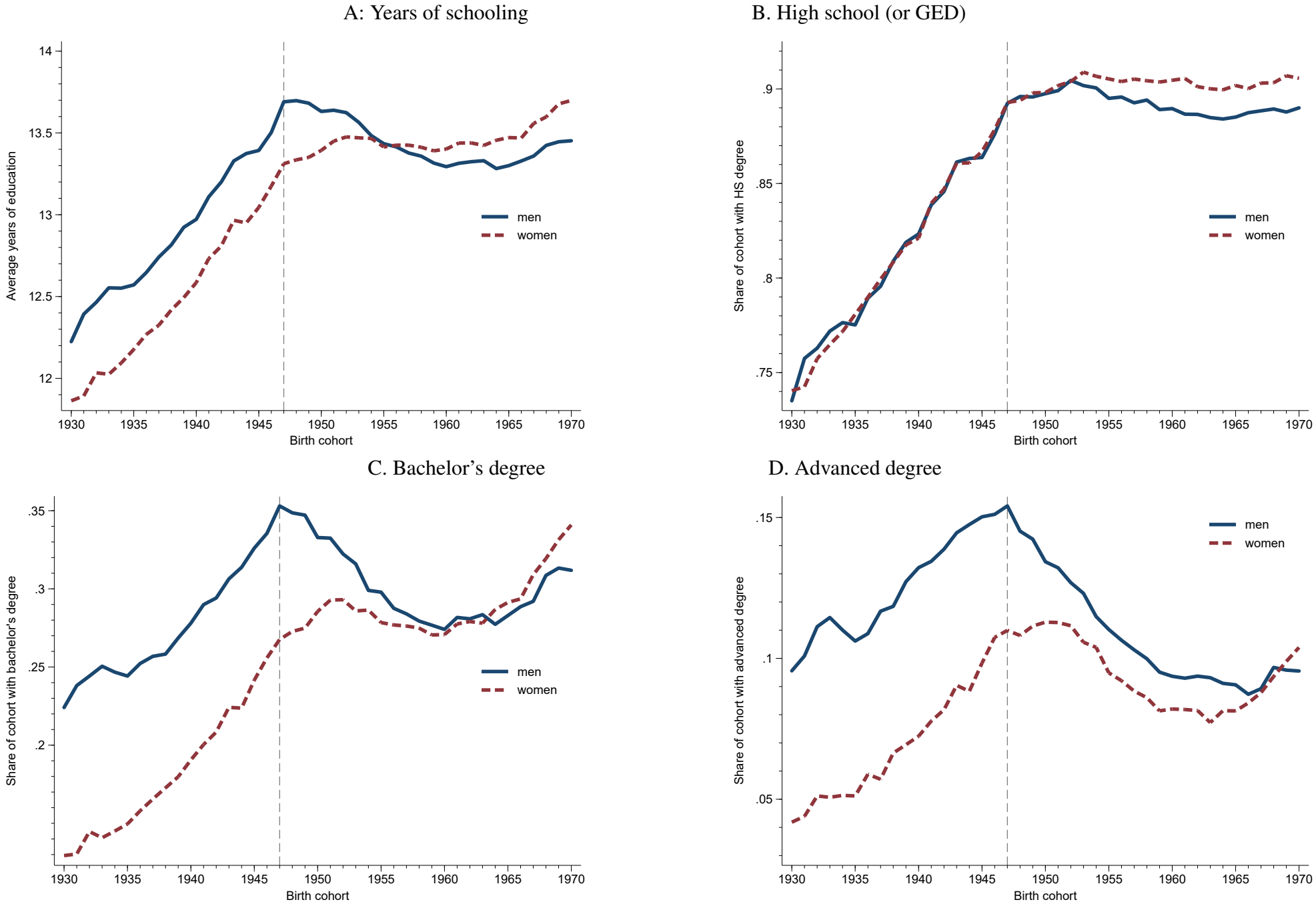
One final piece of evidence against an "education-only" explanation is the different timing by cohort of the declines in women's college graduation rate and of the conditional maternal health declines. Recall that Panel C of Appendix Figure A6 clearly shows that the share of women with a bachelor's degree 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.

Figure A1: 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 4 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.

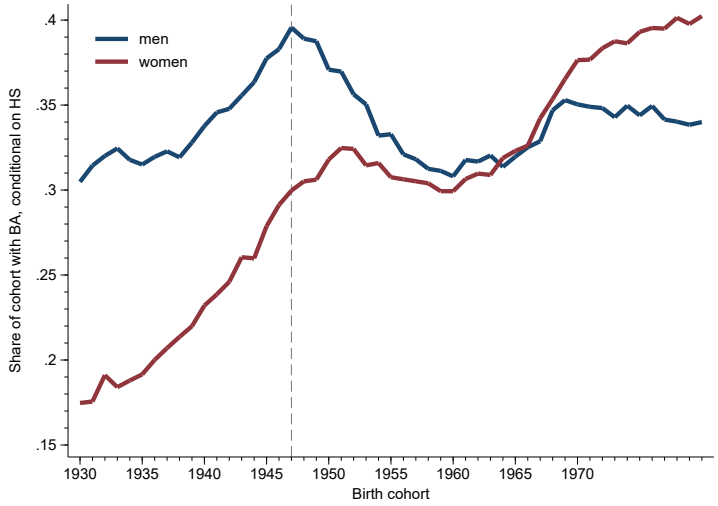
Figure A2: Declines in educational attainment



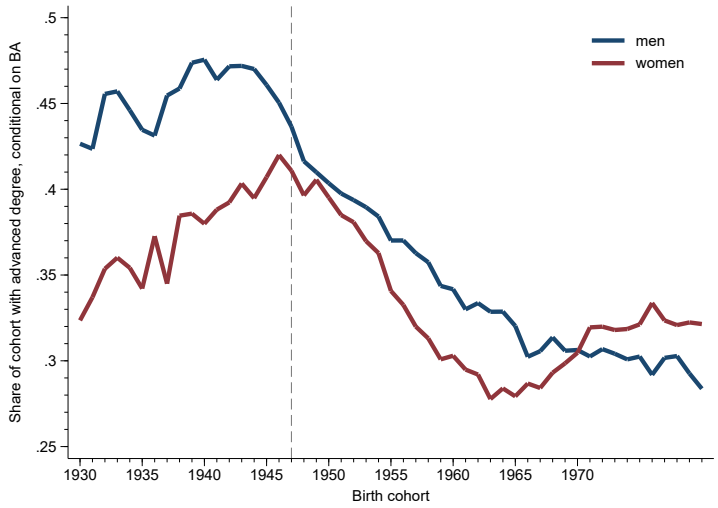
Data is from CPS Merged Outgoing Rotation Group and includes men and women age 25-75 in years 1990-2018. Panel A plots the average years of schooling by birth cohort — approximated based on 16 educational categories. Panels B-D plot respectively the share of each birth cohort with a high school or GED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

Figure A3: Declines in conditional educational attainment along educational ladder

A: Bachelor's degree conditional on finishing high school

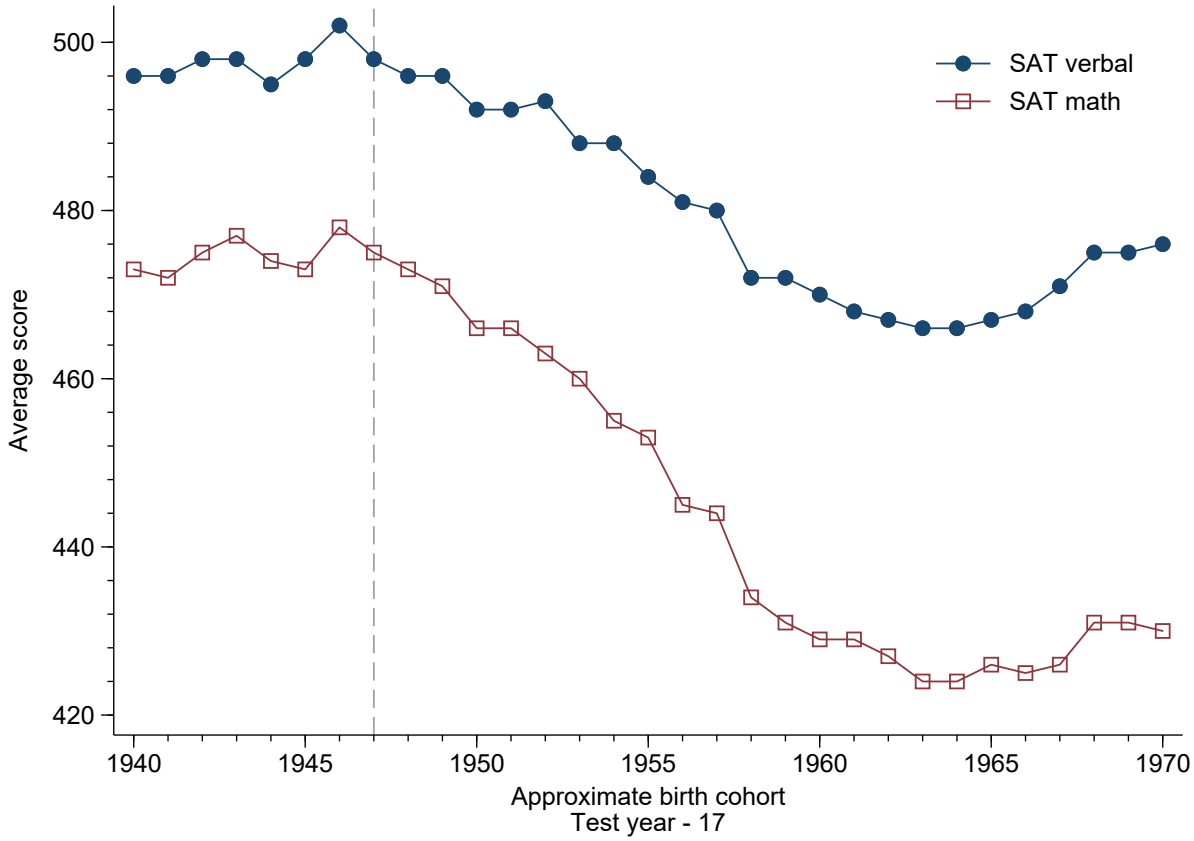


B: Advanced degree conditional on Bachelor's degree



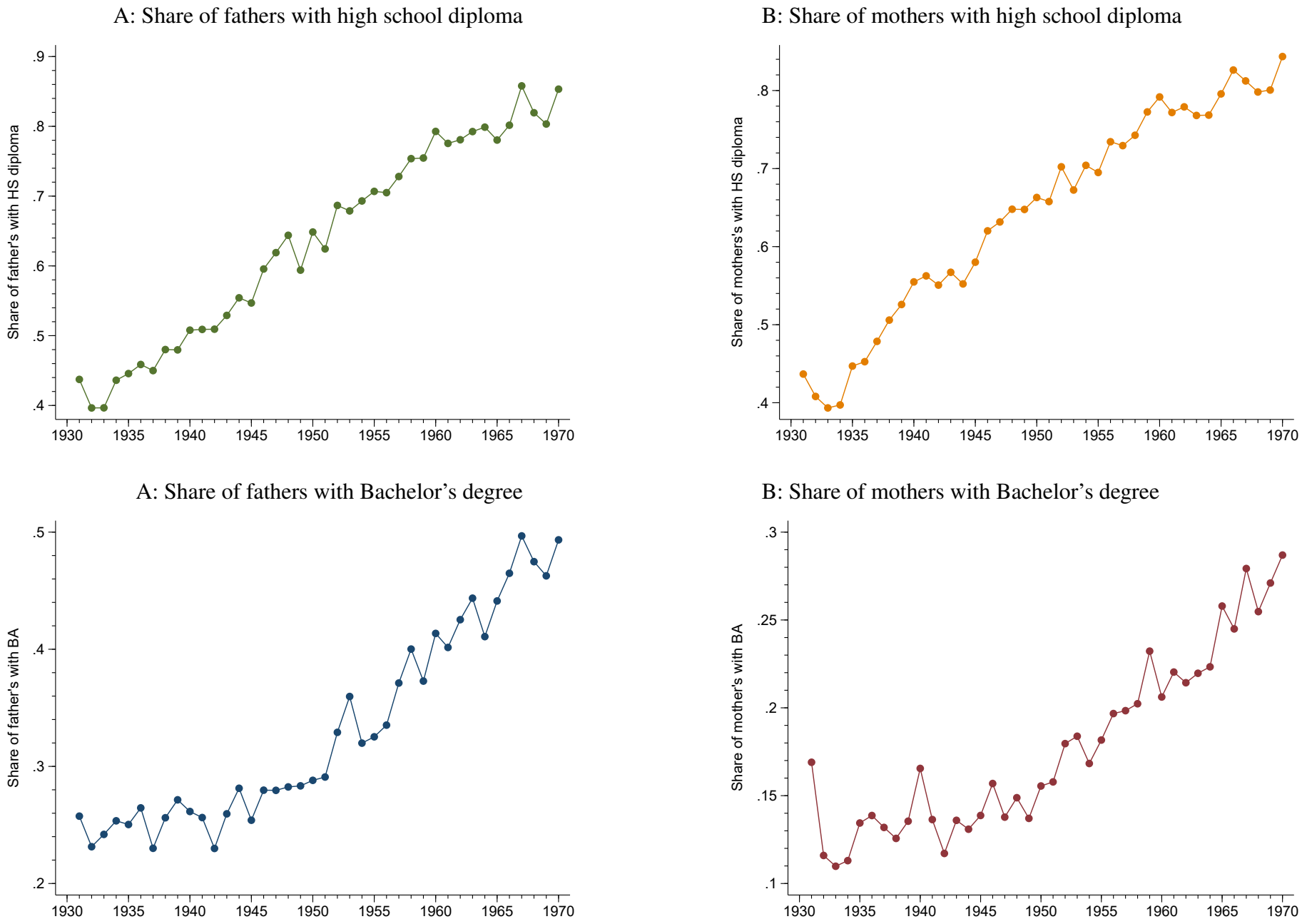
Data is from CPS Merged Outgoing Rotation Group and includes men and women age 25-75 in years 1990-2018. Panel A plots the the share of each birth cohort with a Bachelor's degree conditional on finishing high school — measured as the ratio of the share with a Bachelor's or more over the share with a 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 A4: Declines in Scholastic Aptitude Test (SAT) Scores



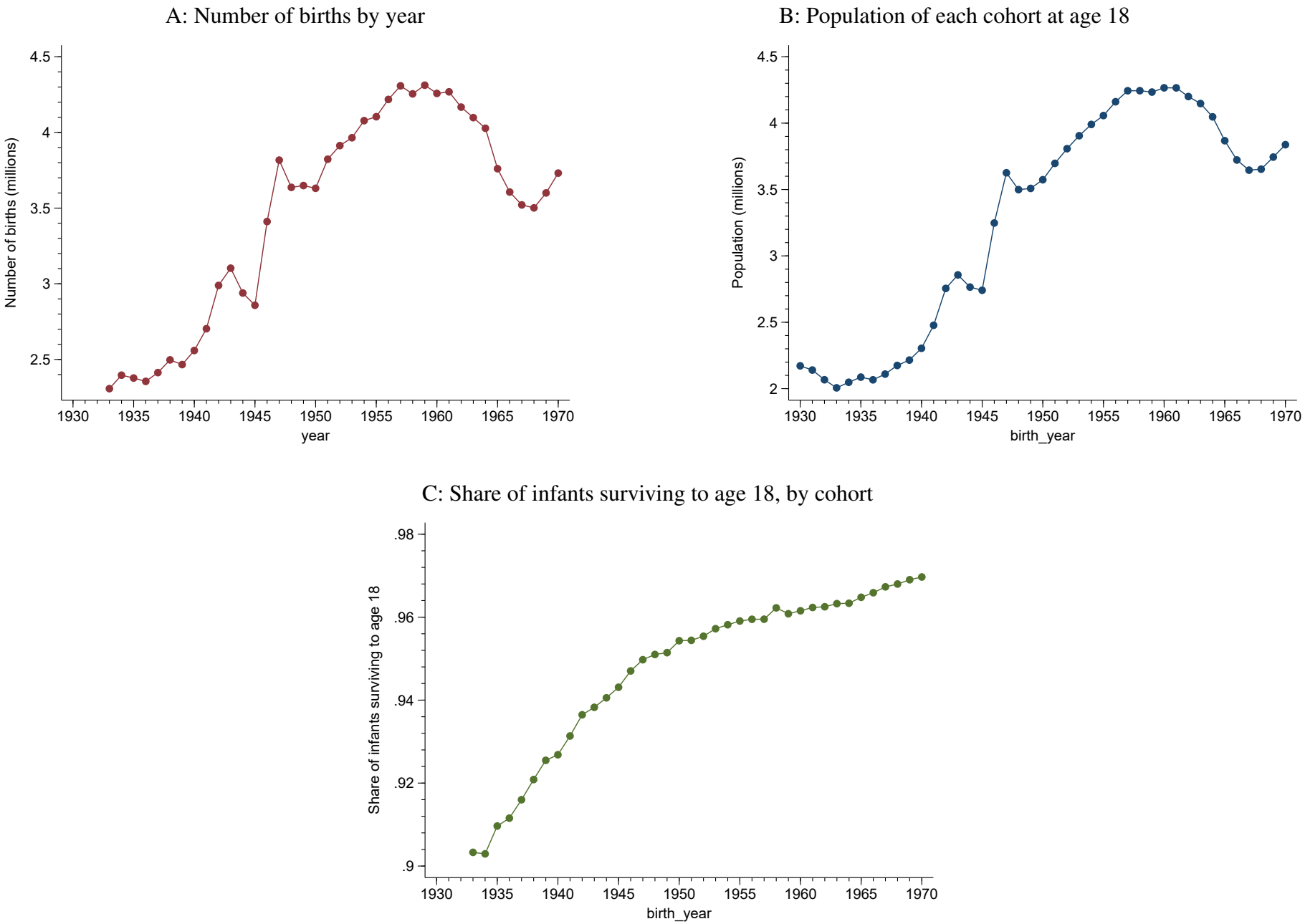
This figure shows verbal and mathematics scores on the Scholastic Aptitude Test. Approximate birth cohort is defined as the year the test was given minus 17. Data is from Harnischfeger and Wiley (1975).

Figure A5: 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.

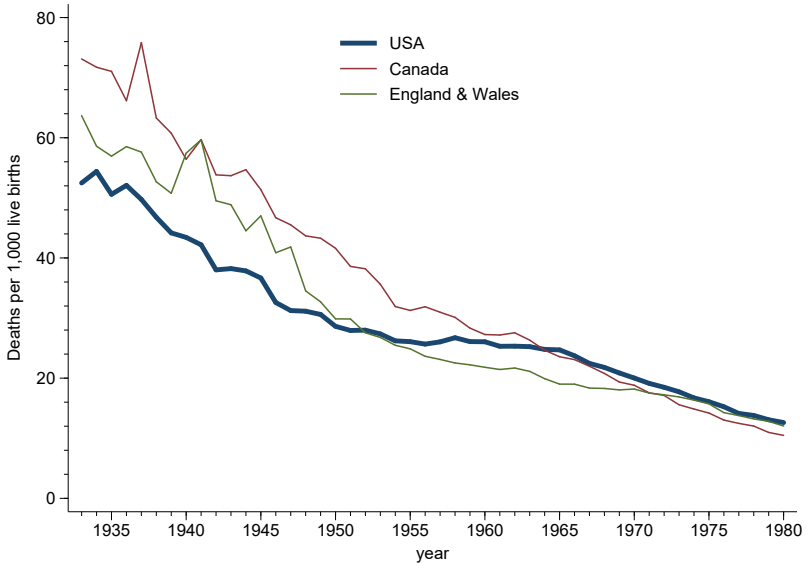
Figure A6: Cohort size and survival



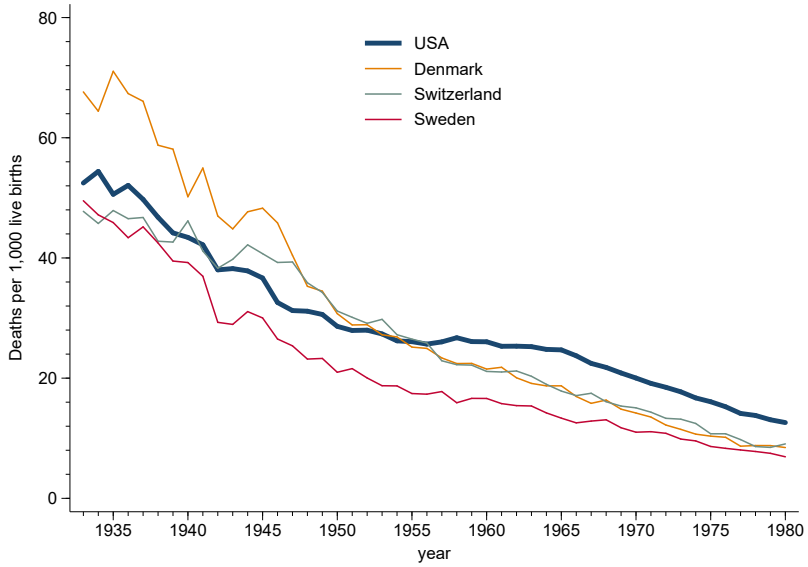
All data from the Human Mortality Database. Panel A shows the number of births in the United States by year. Panel B shows the population of each cohort when they were age 18. Panel C shows the share of infants surviving to age 18 for each cohort — calculated by decrementing the cohort life tables.

Figure A7: Infant mortality rate in United States and comparision countries

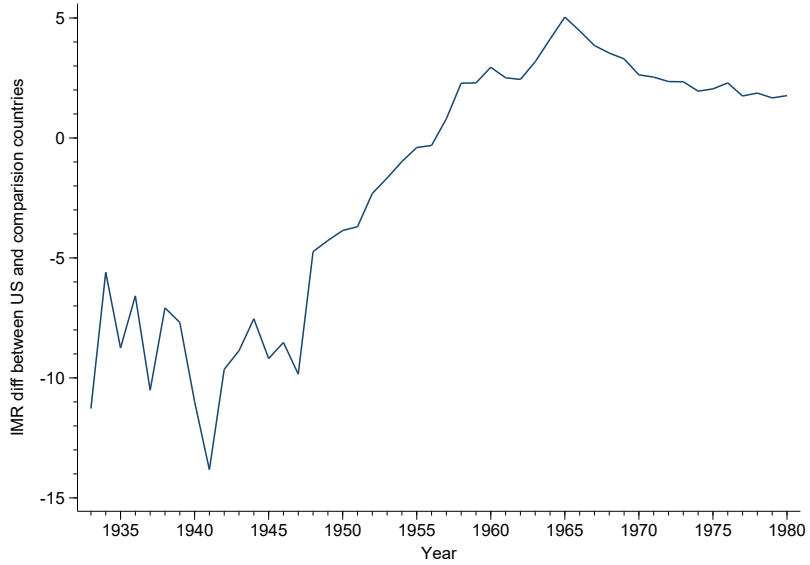
A: English-speaking countries



B: Scandinavian countries

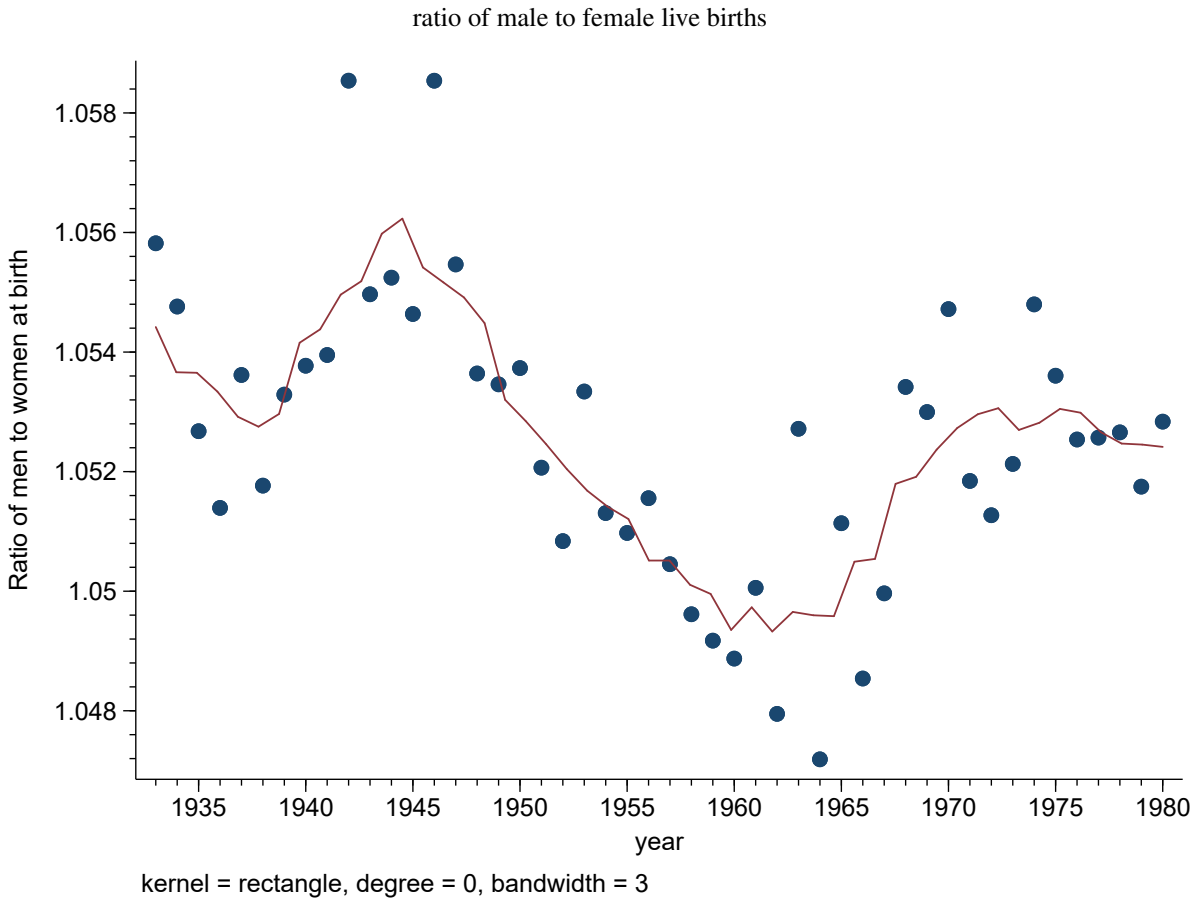


C: Difference between United States and mean across Canada, England and Wales, Denmark, Switzerland, and Sweden



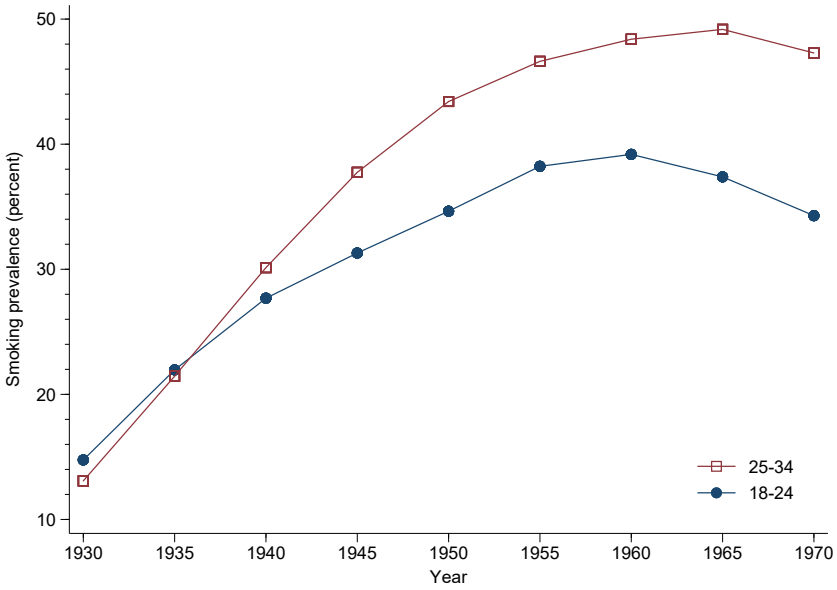
Panels A and B show the infant mortality rates in the listed countries. Panel C shows the difference between the infant mortality rate in the United States and the mean across Canada, England and Wales, Denmark, Switzerland, and Sweden. The mean in the above figure is weighted by the number of births.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

Figure A8: Sex ratio at birth in the United States



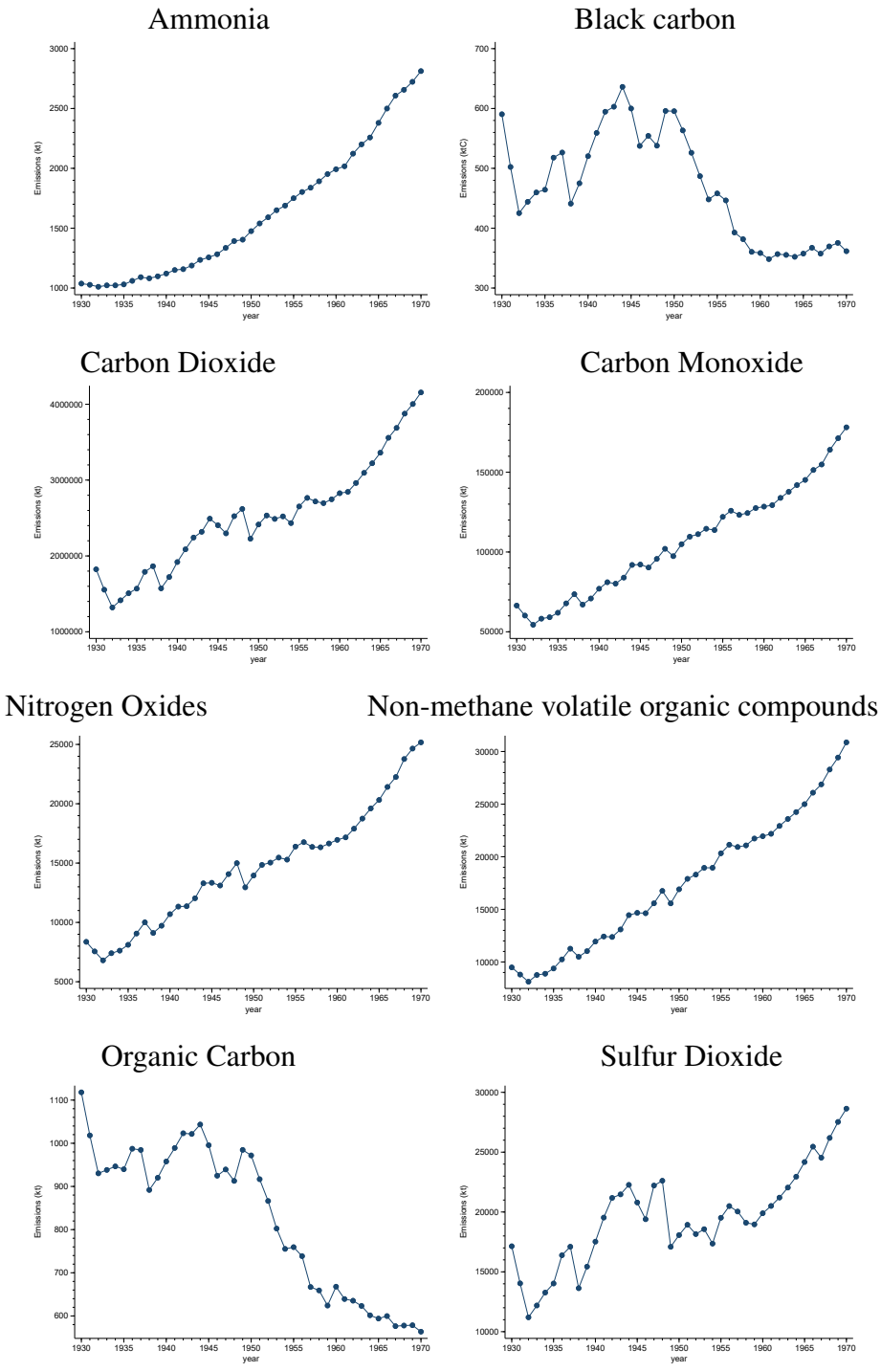
This figure shows the sex ratio at birth over time in the United States, as well as a running-mean smoother. The sex ratio at birth is defined as the ratio of male live births to female live births. Data is from the Human Mortality Database.

Figure A9: Estimates of smoking prevalence of American women of childbearing age



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

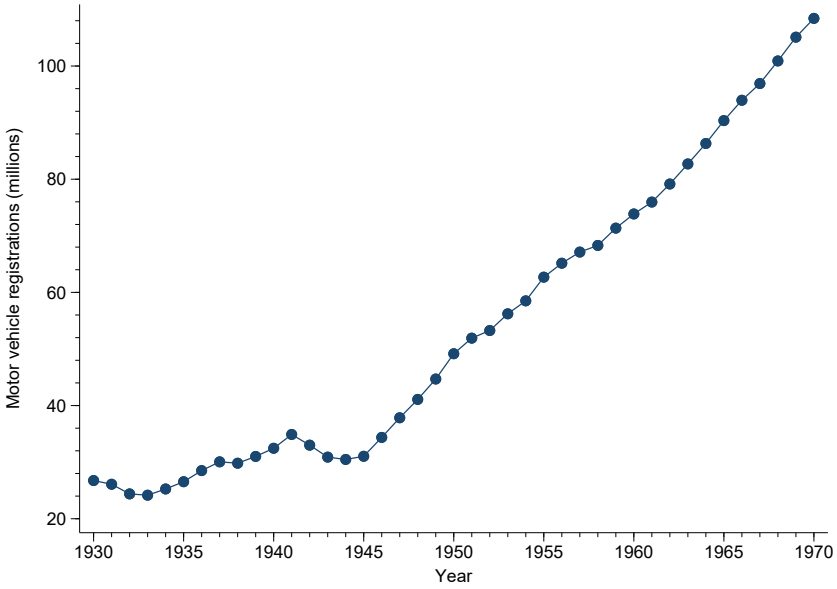
Figure A10: Air pollution trends, United States



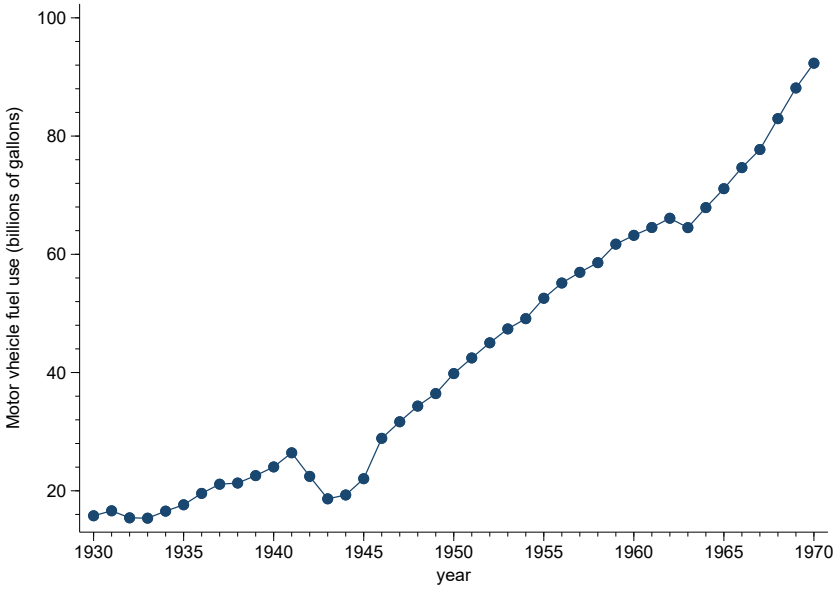
Each panel shows estimates from the Community Emissions Data System (O'Rourke et al.) of the trend in emissions of the listed air pollutant in the United States.

Figure A11: Motor vehicle registrations and fuel use

A: motor vehicle registrations



B: Fuel usage by motor vehicles



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

Table A1: 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 4, 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: 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 4, 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 A3: 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 4, 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 A4: 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 4. 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 A5: 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 4, 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.

Table A6: Implied causal effect of schooling on earnings, maternal health, and mortality assuming no change in unobservables across cohorts

	Change in cohort slope		
	Outcome *(reduced-form)*	Years of schooling *(first stage)*	Implied causal effect *(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 4 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 1975-2019, ages 25-85, cohorts born from 1930 to 1965.

Table A7: 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 4, 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 A8: 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 4, 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.