A Decline in the Health and Human Capital of Americans Born After 1947

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Abstract

Declines in American educational attainment and test scores in the 1970s, stagnating wages since that period, and recent increases in the mortality of white Americans have each sparked large literatures and public outcry. I propose a link between these notable episodes of decline and a few declines that had previously gone unnoticed. I present evidence of a precisely timed decline in the health and human capital of cohorts of Americans born after World War II, which predated labor market entry and played an important role in the above declines. There are discontinuous changes in the slope of "cohort effects" such that those born after 1947 or 1948 were less likely to work in white-collar occupations, earned less, gave birth to less healthy infants, and had a greater likelihood of dying prematurely, than they would have had the trend for earlier born cohorts continued. These declines are remarkably widespread among individuals born in the United States. The timing of the declines by cohort correspond closely to the schooling and test score declines but do not appear to be explained fully by them. I present tentative evidence that the root cause may have been a worsening of the respiratory health environment when these cohorts were infants.

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1. Introduction

In the mid-1960s, SAT scores, high school graduation rates, and college enrollment rates suddenly began to decline, prompting questions about the failure of the educational system (National Commission on Excellence in Education, 1983). In 1999, the mortality rate of white Americans at mid-life began to increase, driven particularly by suicides and drug overdoses (Case and Deaton, 2015). By many estimates, real earnings of the median American male have stagnated since the 1970s, with earnings for those without a college degree declining substantially (Gould, 2014; Acemoglu and Autor, 2011).

These declines have generally been analyzed in isolation, with a focus on the role of contemporaneous factors in each case. For example, educational declines have been attributed partially to cohort size (Card and Lemieux, 2001a), mortality increases to the opioid epidemic (Case and Deaton, 2015), and declining wages for non-college educated men to changes in relative supply and demand (Katz and Murphy, 1992; Acemoglu and Autor, 2011; Card and Lemieux, 2001a). Some papers have hinted at the possibility of a decline in health or human capital of particular cohorts in driving some of these patterns (Case and Deaton, 2017; Acemoglu et al., 2012; Bishop, 1989), but have stopped short of a systematic theory linking all of these patterns to declines for the same cohorts.

In this paper I present evidence of a precisely timed and broad decline in the health and human capital of cohorts of Americans born after World War II, which predated labor market entry and played an important role in the declines described above. I show evidence of discontinuous changes the slope of "cohort effects" such that those born after 1947 or 1948 were less likely to work in white-collar occupations, earned less, gave birth to less healthy infants, and had a greater likelihood of dying prematurely, than they would have had the trend for earlier born cohorts continued. This paper therefore proposes a link between a number of notable episodes of decline and a few that had previously gone unnoticed. And the results suggest that understanding what began to "go wrong" early in life for cohorts born after 1947 would contribute substantially to an understanding of the educational declines of the 1970s, wage stagnation since that period, and recent increases in the mid-life mortality of Whites. I also tentatively suggest a candidate root cause of the cohort declines: a decline in the respiratory health environment when these cohorts were infants, with lasting effects on their health and cognitive ability.

I first show that the age profiles of many outcomes exhibit irregularities across years which appear amenable to a cohort-based explanation. For example, Figure 1 shows that the age profiles of the share of men in white-collar occupations in 1980, 1990, and 2000 exhibit sharp changes in slope — at different ages in each year. However, when the same underlying data is recast by year of birth, or "birth cohort", it is evident that the changes in slope occur at precisely the same cohort, 1947, across years. These patterns appear consistent with a decline in skill or human capital for cohorts born after 1947. In contrast, an alternative explanation assuming no underlying differences across cohorts would require highly non-linear — and non-smooth — changes in age-specific factors over time. Using CPS and vital statistics microdata I show similarly sharp and suggestive patterns in the age profiles of men's median earnings, infant birth weight by mother's birth cohort, and white men's and women's log mortality.

I then discuss formally the key identification issue of the paper — how to separate the role of differences between cohorts from the impact of factors which vary by age and year. I conceptualize each outcome as depending on two additively separable components: cohort fixed effects and a separate function of age in each year. My first approach to identification of cohort effects is to assume further that age and year effects are additively separable. This assumption allows for the identification of cohort effects up to an unknown linear trend, and justifies the estimation of age-period-cohort models common in the literature (see eg. Deaton, 1997). Guided by the shape of cohort effects evident from estimation of these models, my second approach explicitly specifies the cohort effects to have a piecewise linear shape, and estimates the location of the change in cohort slope following the structural break methodology of Hansen (1999, 2000). This approach allows me to probe the robustness of the findings to controlling for smooth age-by-year interactions. My third approach builds on McKenzie (2006) and imposes assumptions on the changes in age effects between neighboring years which justify differencing age profiles between neighboring years to identify differenced cohort effects. The detection of trend breaks by cohort then amounts to the identification of "mean shifts" in these differenced outcomes — similar to those evident in a regression discontinuity design.

Using these strategies I document changes in the slope of cohort effects located at or near the 1947 cohort in men's earnings and occupational status, the health of infants by mother's birth cohort, and the mortality rate of men and women. These trend breaks are visually evident as "mean shifts" in the differenced age profiles, are detected by structural break estimation, and are statistically significant across many specifications. The estimated size of the changes in cohort slope are also large in magnitude. For example, results for men's earnings imply that the median man in the 1960 cohort has earnings roughly 23 percent lower than they would have had cohort effects followed the pre-1947 cohort trend. The magnitude of the intergenerational infant health effects are also large: implying for example that the 1960 cohort of mother's have given birth to nearly 2.8 percentage points more low birthweight infants than they would have had the trend for earlier cohorts continued. For both men's and women's mortality, my estimates imply that single-year mortality risk for the 1960 cohort was roughly 1.5 times what it would have been had the pre-late-1940s trend continued.

Having presented evidence of a decline in cohort health and human capital beginning for cohorts born after 1947, I then document that the decline is generally remarkably widespread, cutting across racial and geographical lines, but appears concentrated among those born in the United States. A notable exception worthy of further study is the cohort mortality pattern for black men, which shows evidence of improvements for post-1952 cohorts. Interestingly, black men exhibit cohort declines in all the other outcomes which are similar to, and for earnings larger than, those for whites and other groups. Importantly, the decline appears concentrated among those born in the United States and is not evident for those born outside the United States. This finding suggests that the cohort declines may have been caused by an early-life factor *specific* to the United States.

The timing of the cohort declines I document coincide closely with previously noted declines in educational attainment at all levels and in SAT scores. It therefore appears likely that they are linked. Under reasonable assumptions on the returns to schooling, the size of the trend break in educational attainment

could explain the decline in earning and in infant health in the next generation. However, for the educational attainment declines alone to explain the increased mortality would require an implausibly large causal effect of education on mortality. The timing of declines in maternal health and earnings for women also predate the educational trend break for women by a few cohorts. In sum, the evidence I present suggests that the schooling decline alone cannot explain the subsequent declines in adult outcomes. I therefore suggest that there was a broad decline in the latent health and human capital of these cohorts which predated adolescence, drove the initial education declines, and contributed to the subsequent declining adult outcomes.

Finally, I present more tentative evidence of a potential root cause of the cross-cohort decline in health and human capital: a post-war decline in the respiratory health environment in the United States. My hypothesis is that there was a broad decline in respiratory health "environment" after 1946 or 1947 — which increased respiratory mortality for infants and adults in the short run — and had a lasting effect on the health of cohorts who were in utero or infancy during that period. The key piece of evidence for a broad decline in respiratory health is that mortality from particular respiratory causes began to increase simultaneously for both infants *and* elderly adults near 1947. The hypothesized change in the respiratory health environment could conceivably have been caused by a decline in air quality, due for example to post-war industrial growth or increased driving; or the risk of respiratory infections may have increased for other reasons unrelated to air quality.

The remainder of the paper is organized as follows. Section 2 describes the data I use. Section 3 presents descriptive evidence of changes in age profiles across years which could be amenable to a cohort-based explanation. Section 4 outlines a model of cohort effects, my method to identify them, and the empirical methodology. Section 5 implements this methodology to demonstrate cohort declines in four outcomes. Section 6 documents the widespread nature of the cohort declines among the US-born. Section 7 considers the link between these patterns and educational declines in 1970s. Section 8 documents a post-war respiratory health decline, and posits the lasting effect of this decline for those in early life as a tentative explanation of the cohort declines. The final section concludes.

2. Data

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

A. Education, earnings, and occupation

To document cross-cohort declines in occupational status, earnings, and educational attainment I draw on two large, commonly-used data sources: decennial Census microdata and the Current Population Survey, Merged Outgoing Rotation Group (CPS-MORG). The Census data contain larger samples — six percent samples of households in each year — and record place-of-birth. The CPS-MORG has the advantage of

recording point-in-time hourly earnings, and its yearly frequency aids identification of cohort effects.

I combine six percent samples from the Integrated Public Use Microdata Samples from the Decennial Censuses of 1980, 1990, and 2000 [1]. My main analysis is restricted to individuals born in the United States between 1930 and 1970, aged 25 to 65. 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 use the CPS MORG data from 1979 to 1993. These files contain information on the hourly or usual weekly earnings of a third of the individuals in each monthly CPS. Combined with information on usual hours worked this allows for the construction of point-in-time hourly earnings. These hourly earnings measures have been used extensively in studies of wage inequality and trends, and have some notable advantages over other sources.[2] 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 Katz et al. (2005) suggest that the point-in-time nature of the earnings question reduces measurement error.[3]

I focus on prime-aged workers aged 25 to 54, and 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 Katz 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 sample restrictions lead to a sample of 970,479 men with non-missing earnings used in the main analysis. In some supplementary analysis I use a similar sample of 854,978 women.

I address a second concern of the CPS-MORG data, topcoding of earnings above a threshold which changes across years, by focusing on the median and other quantiles. I calculate the sample median and other quantiles separately for age-year-sex cells, using the provided survey weights. Due to their order-statistics properties, these estimated quantiles are unaffected by the topcoding — as long as the earnings value of the particular quantile is below the topcoding value. For regressions and other model-based analysis, I use a two step procedure: first estimating cell quantiles, and second estimating models on the cell quantiles. This approach follows Chamberlain (1994) and Chetverikov et al. (2016), and for sufficiently large cells has advantages over traditional LAD quantile regression.[4] 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.[5]

In both datasets, my main measure of occupational status 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

[1] 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.

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

[3] 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.

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

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

authors to obtain consistent occupational categories. I focus on employed workers, aged 25 to 64, and I use the years 1979-2000 of the CPS-MORG. I calculate the share of employed men in each age-year cell employed in white collar occupations. In supplementary analysis, I also consider the share in a set of "blue-collar" occupations, continuous measures of occupational status based on average earnings and average years of schooling, and task measures based on the US Department of Labor's Dictionary of Occupational Titles (DOT) (also drawn from Autor and Dorn (2013), based on Autor et al. (2003)).

B. 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.

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.

I calculate mortality by race manually. 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 — the ratio of mortality over mid-year population — within each cell. I then define birth cohort as $year - age$.

C. 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 assumption that maternal health enters the infant health production function, 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. 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.

As my main infant health outcome, I study infant 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.

In all of my analysis I use information on the year of birth and mother's age. As in the Census and CPS data, birth cohort — in this case mother's exact year of birth — is not recorded explicitly. I instead calculate the approximate birth year of each mother as the infant birth year minus the mother's age.

D. Historical mortality from respiratory causes

I use newly digitized data on historical mortality by *cause of death* to providence evidence of a post-war decline in respiratory health in the United States. The data has been digitized from tables in historical vital statistics books for 1933-1958. It consists of death counts for cause of death categories based on International Classification of Disease codes by race, sex, and 5-year age bins. I calculate analogous death counts from the Multiple Cause of Death File described above for 1959-1980. I combine these with population estimates from the Census Bureau to form cause-specific mortality rates by age, race, sex, and 5-year age bins for 1933-1980.[6]

3. Descriptive patterns amenable to cohort explanation

I show descriptive patterns in occupational status, earnings, mortality, and maternal health that appear amenable to an explanation based on a decline in human capital and health beginning with cohorts born after 1947. This descriptive analysis highlights the identification challenge posed by the collinearity of age, year, and cohort; and therefore motivates the formal identification and results that follow.

In particular, I show that the age profile of each of the outcomes examined exhibit sharp changes in slope which could be driven by a cross-cohort decline in health and human capital. Because age and cohort are collinear in a single year, theoretically these age profiles combine both the impact of age in the given year and the impact of latent, long-standing differences between cohorts born in different years. The slope changes which I document occur at different ages in each year, and therefore cannot be explained by impacts of age which are fixed across years. However, they could be explained by some particular non-smooth changes in age effects across years. Interestingly, the slope changes occur at nearly the same cohort — 1947 — across years. I therefore suggest that they could instead be explained by long-standing differences in health and human capital: in particular, a cross-cohort decline — relative to the prior trend — beginning with the 1947 cohort.

To formally distinguish between these two explanations, one based on complex age-by-year interactions and another based on cohort differences, requires additional assumptions. I take up this formal identification challenge in the next section. As motivation, this section provides some preliminary discussion and descriptive evidence.

[6]For now I just use white mortality rates. Plan to combine with more digitized data — including national mortality rates for blacks. And potentially also state-level data to examine how widespread the increases are.

A. Occupational status

Figure 1 shows these patterns for the share of men employed in white-collar occupations. The data come from the Census microdata described above and include working men ages 25 to 64. Panel A shows these shares by single year of age for 1980, 1990 and 2000.

A strange pattern is evident in these "age profiles" of occupational status. The age profiles in each year exhibit a sharp change in slope, and these slope changes occur at different ages in each year. Put another way, the graphs shown in Figure 1 exhibit "kinks" such that the slope — or first derivative with respect to age — changes discontinuously, and these kinks or "slope-changes" occur at different ages in each year shown. For example, in 1980 the white-collar share increases rapidly from near 32 percent at age 25 to nearly 42 percent by age 32, but then this trend reverses and the share decreases gradually for older ages. In 1990, however, the white-collar share increases nearly continuously from age 25 to age 42, before reversing trend at that age and declining subsequently. In 2000 a clear change in slope is evident at age 52.

One explanation for these patterns is that impact of age on occupational choices of individuals has changed over the years shown. For example, employers' demand for white-collar jobs may have changed such that they place a different premium on experience in 2000 than 1980. However, such an explanation would require that the way in which age-specific factors impacting occupational choice have changed over time has been highly non-linear — and in particular non-smooth.

An alternative explanation is that there are latent, long-standing differences between cohorts born in different years which drive the slope changes in each year. In particular, note that the age in each year which corresponds to the 1947 cohort is labelled in Panel A, and that the slope changes appear to occur at this cohort in each year. These patterns appear consistent with a decline in skill or human capital for cohorts born after 1947, such that in all years these cohorts have a lower propensity to work in white-collar occupations.

As further visual evidence consistent with a cohort-based explanation, consider Panel B of Figure 1. This panel shows the same underlying data as Panel A — the share of men of each age employed in white collar occupations in the 3 census years — but now plots the shares by birth cohort rather than age. It is possible to redefine the x-axis in this way because of the collinearity of age and birth cohort. This redefinition reveals clearly that the slope changes shown in Panel A each occur at the 1947 cohort.

B. Earnings

Figure 2 shows analogous patterns for the median hourly earnings of working men. The data come from the CPS-MORG described above and include working men ages 25 to 54. Panel A shows median hourly earnings by single year of age for the first year of this data, 1979, and for two subsequent years 5 and 10 year later, 1984 and 1989.

A broadly similar pattern to that in occupation status is evident in these age profiles of median log hourly earnings. The patterns are noisier than those in Figure 2, as the data come from a smaller sample and represent a variable likely more prone to measurement error. However, the age profiles in each year still

appear to exhibit changes in slope, occurring at different ages in each year. For example, in 1979 median earnings increase rapidly between age 25 and 32 — rising .2 log points from 2.9 to above 3.1 — and then are much flatter from age 32 on. In contrast, earnings in 1984 appear to increase steadily until age 36, and only began to flatten after that age. Earnings in 1989 are clearly increasing until an even later age, near age 41, before flattening.

Again, one explanation for these patterns is that the return to age, or experience, changed across the years shown. However, such an explanation would need to be more complex than a change in the return to a single-index experience factor. Such a single-index change would lead to a change in the slope of the age profile, and potentially a change in the curvature of the age profile, but would not be able to match the existence of kinks or slope changes occurring at different ages in each year.

As for the occupational patterns, an alternative explanation is that the slope changes in each year are driven by latent, long-standing differences between cohorts born in different years. Panel B shows the same underlying earnings data as Panel A, but now plots the median earnings by birth cohort rather than by age. It again reveals a clear pattern. For the two later years, 1984 and 1989, a sharp slope change clearly occurs at the 1947 cohort. For 1979, a slope change appears to occur one cohort later — at the 1948 cohort. These patterns are highly suggestive that an alternative explanation, based on a decline in the human capital of cohorts born after 1947, could match the documented patterns in the age profile of median earnings.

C. Maternal health

Figure 3 shows similar patterns in the mean birthweight of infants by the mother's age and year of birth. The data come from the Natality Detail Files described above and include births to mothers between the ages of 15 and 38. Under the assumption that maternal health is an important input in the infant health production function, these patterns may reveal information about the underlying health of cohorts of mothers.

Panel A shows mean birth weight by mother's age for 1968, 1972, and 1976 — the first year of data and two subsequent years. The birth weight of infants increases with mother's age. The age profiles are also convex, with the slope at which birth weight increases decreasing with maternal age. There is again some visual evidence of kinks, or changes in slope, at particular ages in each year. In particular there are clear slope changes at age 25 in 1972 and age 29 in 1976, such that while birth weight increases rapidly with age up to those ages, it is nearly flat for subsequent ages. In 1968, it appears that the slope of the age profile may change in the early-20s but the change is significantly less sharp.

Panel B again recasts the same data by birth cohort. The slope changes in 1972 and 1976 clearly both occur at the 1947 cohort. In these years the birth weight is nearly flat until the 1947 cohort, and in 1976 even increasing slightly, and then suddenly begins to decline for subsequent cohorts. The less sharp change in slope in 1968 also appears to correspond closely to that cohort. Though in this year birth weight is clearly also decreasing for earlier cohorts, that is for older age mothers.

Overall, the patterns in the birth weight of infants by mother's birth cohort again appear suggestive of a potential role for cohort factors. In particular, a decline in the health of cohorts, beginning with those born after 1947 could be causing these cohorts to be giving birth to lower birth weight infants — driving the slope

changes shown above. As for the other outcomes, an alternative explanation assuming no differences across cohorts would need to appeal to highly non-smooth changes in the impact of maternal age on infant health.

D. Mortality

In Reynolds (2018), I show that the age profile of log mortality of white men and women also exhibit sharp changes in slope, similar to those shown above for other outcomes. I briefly review a selection of those results here.

I show slightly different figures for log mortality than for the other outcomes. Log mortality increases very rapidly and remarkably linearly with age. This log-linear relationship between age and mortality is known as Gompertz law (Gompertz, 1825; Chetty et al., 2016). The rapidness of the increases with age make even highly statistically significant slope changes hard to detect visually. I therefore use an approach developed extensively in Reynolds (2018) based on fitting piecewise linear models to the log mortality age profiles. I leave the location at which the slope change occurs as a parameter to be estimated, and follow the estimation methodology of Hansen (1999) and Hansen (2000). More detail on this methodology is given below and in Reynolds (2018)

The top two panels of Appendix Figure A1 show these patterns for white men in 1995 and 2015. The red circles show the true observed log mortality rate for each single year of age. The solid blue line shows the estimated piecewise linear model. The vertical, labeled gray line shows the cohort at which the trend break is estimated to occur for that year. The dotted blue line extrapolates the estimated linear trend for cohorts born before the trend break to younger, post-break cohorts. In 1995 a change in slope is visually evident, and estimated to occur at age 48. In 2015, a smaller slope change is also apparent and is estimated to occur at age 68. Interestingly, the ages of these slope changes in the two years correspond to the same cohort, 1946.

The bottom two panels of Appendix Figure A1 plot the deviations of the true log mortality rates of white men from the estimated linear trend for pre-break cohorts. That is, it shows the difference between the pre-break trend, shown in the top-panels with a solid blue line for pre-break cohorts and a dotted blue line for post-break cohorts, and the true log mortality rates, which were shown in red circles. A horizontal gray line is now plotted at the 1946 cohort. In both 1995 and 2015, the deviations are near zero for cohorts born before 1946, implying that the a linear trend fits the log mortality rates well for these cohorts. Then suddenly at or near the 1946 cohort there is a change in slope and the deviations increase for each subsequent cohort. The change in slope is particularly large in 1995, with the 1960 cohort for example experiencing mortality rates approximately 50 log points higher than a linear age profile for earlier cohorts would predict. The change in slope is smaller but still clearly evident in 2015, with the 1960 cohort experiencing mortality rates around 20 log points above what would be predicted by the prior linear slope.

Appendix Figure A2 replicates these patterns for white women. The slope change in log mortality is less visibly striking for women than for men, but is still evident and detected by the Hansen estimation. The breaks in 1995 and 2015 occur at ages 37 and 56, respectively. As a result, the estimated breaks by cohort are again close to uniform, though later than for men, occurring at the 1947 and 1948 cohort across the years

shown.

Again, the bottom two panels of Appendix Figure A2 plot the deviations of the true log mortality rates — now for white women — from the estimated linear trend for cohorts born before the estimated change in slope. In both 1995 and 2015, the deviations are near zero for cohorts born before 1946, implying that the a linear trend fits the log mortality rates well for these cohorts. Then suddenly at the 1949 cohort there is a change in slope and the deviations increase for each subsequent cohort. The change in slope is large enough in both years that by the 1960 cohort the mortality rate is more than 20 log points higher than the linear trend for pre-1946 cohorts would predict.

4. Model and methodology to identify cohort effects

I use three approaches to address the well-known problem of separately identifying the role of "cohort effects" from year and age-specific factors. The collinearity of cohort, age, and year makes it impossible to separately identify the role of cohort factors without making assumptions about the nature of year and age-specific factors. Generalizing the usual assumption that age, year and cohort effects are additively separable, I conceptualize each outcome as depending on two additively separable components: cohort fixed effects and a separate function of age in each year. Based on this conceptual model I then outline three approaches to identification and estimation of cohort effects. I use these is subsequent sections to document discontinuous changes in cohort effects near the 1948 cohort suggestive of a decline in health and human capital after that year.

My first approach to identification of cohort effects is to assume that the age and year control-function consists of additively separable year fixed effects and age fixed effects. This assumption allows for the identification of cohort effects up to an unknown linear trend, and justifies the estimation of additively separable age-period-cohort models similar to those in Deaton (1997). Guided by the shape of cohort effects evident from estimation of these models, my second approach explicitly specifies the cohort effects to have a piecewise linear shape, and estimates the location of change in cohort slope following the structural break methodology of Hansen (1999, 2000). This approach therefore reduces dimensionality, and allows me to probe the robustness of the change in cohort slope to controlling for smooth age-by-year interactions, in particular separate polynomials in age in each year. My third approach builds on McKenzie (2006) and imposes assumptions on the changes in age effects between nearby years which justify differencing age profiles between nearby years to identify differenced cohort effects. It has the advantage of transparent visualization and relatively weak assumptions, but uses only a piece of the variation in the data and is therefore less efficient than the other approaches.

A. Conceptual Model

Differences in mortality, occupational status and other later-life outcomes could be caused by various factors that vary at the cohort, age, or year level. Theories of health and human capital accumulation and recent empirical evidence suggest that differences in early life investments have lasting effects on later life

outcomes (Grossman, 1972; Heckman and Cunha, 2007; Almond and Currie, 2011). If these early life investments differ across cohorts this will leave an imprint on cohort patterns in later life outcomes. However, theory also predicts clear impact of factors which vary by age and year. The natural aging process and skill accumulation will impact mortality and the occupations of individuals of different ages. Improvements in medical knowledge and technology are widely believed to have improved health and reduced mortality over time.

In some cases, we may even think that factors exist which vary by age and year, ie. age-by-year interactions. For example, the AIDS epidemic increased mortality particularly for young adults. Older and younger workers are often assumed to not be perfectly substitutable in production and therefore their return to working in particular occupations may change differentially over time.

As outlined informally above, the collinearity of age, year, and cohort makes it difficult to distinguish between explanations based on cohort differences and those based on complex age-by-year interactions. The problem of separately identifying the role of these factors — the so-called "age-period-cohort" problem is well known.[7] In the general case, in which age-by-year interactions are unrestricted, an observed set of data will be consistent with *any* set of cohort effects. To identify cohort effects, or even features of them such as first-differences, additional assumptions on the nature of age and year effects are required.

To fix ideas consider the following reduced-form model:

$$Y_{apc} = \gamma_c + f^p(a) + \varepsilon_{apc} \tag{1}$$

where Y_{apc} denotes an outcome — such as average years of education — 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. Cohort effects are represented by the sequence γ_c. The function $f^p(a)$ is a control function representing the impact of age in each year on the outcome. ε_{apc} is an orthogonal error. Cohort is generally not recorded explicitly, and is therefore defined as year minus age, creating an exact collinearity between the 3 measures: c = p - a.

Note that the identification of γ_c allows one to make counterfactual statements about the theoretical outcomes for cohorts in ages and years in which they are not observed. For example, one may want to ask: "What would the 1960 cohort have earned had they been 30 in 1980 in comparison to the 1950 cohort — who was 30 in 1980?" In the context of the model above the answer to this question is revealed by $\gamma_{1960} - \gamma_{1950}$. This suggests a conceptual link between the cohort identification problem and causal inference. One can only observe Y_{apc} in cases where $c = p - a$. However, to make statements about the role or "effect" of cohort differences one is interested in inference on potential outcomes which are not observed.

Just as additional structure is required in the problem of causal inference to infer features of potential outcomes which are not observed, to identify cohort effects one needs to impose additional structure on the impact of age and year, represented by $f^p(a)$. Below I outline three approaches to identification and estimation of γ_c.

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

B. Additively separable age-period-cohort models

My first approach to identification of cohort effects is to assume that the age-by-year control-function consists of additively separable year fixed effects and age fixed effects. This assumption allows for the identification of cohort effects up to an unknown linear trend, and justifies the estimation of additively separable age-period-cohort models common in the literature (see Deaton (1997) for a textbook treatment). These models allow for a flexible shape of the age effects and allow for secular year effects of any form, but do now allow for age-by-year interactions.

I assume that the age-by-year control function is additively separable in age and year. That is, that $f^p(a)$ consists of year fixed effects and age fixed effects. This assumption is 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.

Applying this assumption to equation 1 yields the following model:

$$Y_{apc} = \gamma_c + \phi_p + \theta_a + \varepsilon_{apc} \tag{2}$$

As is well known, this model is still unidentified. (see eg. Hall (1968), Deaton (1997)). Identification can be achieved by imposing one additional linear restriction, such as restricting two ages to have the same effect. Instead of imposing this or a similarly ad hoc restriction, I follow Fosse and Winship (2017) and Chauvel (2011) and focus on identifying reparametrized coefficients of the model in equation 2. In particular, I show that cohort effects can be identified up to an unknown linear trend, and estimate "detrended cohort effects" which normalize that trend to be 0.

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 age, year, and cohort effects as follows:

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

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" age, year, and cohort effects as:

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

The normalization of the first age, year, and 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, as revealed by inserting the definition of the trends into the above definition. They can be thought of as identifying effects up to both a level *and* a "trend"-shift. That is, these detrended effects normalize the first and last age, year, and cohort effects to 0, and each detrended effect represents the true effect minus the long-run linear "trend effect."

With these definitions one can rewrite equation 2 as:

$$Y_{apc} = \tilde{\gamma}_c + \tilde{\phi}_p + \tilde{\theta}_a + \underbrace{(\beta_a - \beta_c)}_{\equiv \tilde{\beta}_{ac}} \cdot a + \underbrace{(\beta_p + \beta_c)}_{\equiv \tilde{\beta}_{pc}} \cdot p + \varepsilon_{apc} \tag{3}$$

This reparameterized model is identified: the design matrix is full rank. Most importantly for my purposes, this implies that cohort effects are identified "up to trend." Below, I will estimate the sample analog by linear regression, and will focus on the detrended cohort effects $\tilde{\gamma}_c$.

Graphical inspection of the sequence of detrended cohort effects will allow for the identification of *slope changes* in cohort effects. For example, below I show that the estimated detrended cohort effects in multiple outcomes have a clear piecewise linear shape, increasing linearly until the 1947 cohort and then reversing trend and declining linearly for subsequent cohorts (see eg. Panel A of Figure 5). These results imply that there was a sharp *change* in slope at the 1947 cohort, but because the long-run trend in cohort effects β_c is not identified, cannot distinguish whether there were absolute declines starting with the 1947 cohort, or merely a sudden slowing of a previous trend of improvement.

C. Piecewise linear cohort effects

Guided by the shape of cohort effects evident from estimation of the age-period cohort models outlined above, I then estimate models which explicitly specify the cohort effects to have a piecewise linear shape. I leave the location of the kink or "knot" of these piecewise linear cohort effects as a parameter to be estimated, following the structural break methodology of Hansen (1999, 2000). This approach therefore reduces dimensionality, and yields summary measures of the particular cohort at which the change in slope of cohort effects occurs, the size of that slope change, and associated statistical tests and measures of uncertainty. Further, the imposed restriction on the shape of cohort effects allows me to control for smooth age-by-year interactions, in particular separate polynomials in age in each year. It therefore allows me to probe the robustness of the estimated change in cohort slope to increasingly flexible age-by-year interactions.

Assume cohort effects are piecewise linear with a single, *unknown* slope-change or knot. Then Equation 1 becomes:

$$Y_{apc} = \underbrace{\beta \cdot c}_{\text{pre-slope-change trend}} + \underbrace{\mathbb{1}_{c \geq \lambda} \cdot \delta \cdot (c - \lambda)}_{\text{change in slope}} + f^p(a) + \varepsilon_{apc} \tag{4}$$

where Y_{apc} denotes an outcome of the cell of age a, year p, and cohort c. As above, β represents a long-run trend in cohort effects. The second term on the left-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. I will include increasingly flexible specifications of the "control function" $f^p(a)$. Note that for similar reasons to those outlined in Section B, the linear trend in cohort β is generally not identified separately from aspects of the control function, but the location and size of trend break are identified.

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 slope change evident visually from that approach to be summarized in two parameters — the slope change size, δ and it's location, λ. Additionally, I follow the structural break methodology of Hansen (1999, 2000) to provide associated estimates of uncertainty for these parameters.

Interestingly, 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*. I therefore 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, and to a slightly lesser extent the magnitude, of the estimated change in cohort slope are remarkably robust to the inclusion of these higher order polynomials. This appears to suggest that the estimated change in cohort slope is not driven by *smooth* age-by-year interactions.

I estimate the model by least squares, following the methodology in Hansen (2000). Algorithmically, this amounts to looping through different assumed values of the trend break location λ, and selecting the location with the lowest sum of squared residuals.[8]

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

$$LR(\lambda) = n\frac{S(\lambda) - S(\hat{\lambda})}{S(\hat{\lambda})} \tag{5}$$

where n denotes the number of observations, $S(\lambda)$ is the sum of squared residuals from estimating Equation 4 with the trend break location fixed at a given λ, and $S(\hat{\lambda})$ is the sum of squared residuals with the *estimated* break location $\hat{\lambda}$. I constuct the 99 percent confidence interval as those values of λ such that $LR(\lambda) <= 10.35$, the critical value given in Hansen (2000). While I allow for heteroskedasticity in inference on other parameters, this test requires homoskedasticity.

Hansen (2000) also suggests that inference on δ is unaffected by treating λ as unknown. I therefore

[8]For other recent examples of use of similar piecewise-linear trend break models in economics, see Charles et al. (2018) and Chay and Munshi (2015).

form confidence intervals for δ using the standard formula for weighted least squares.

Following standard practice, I employ an ad-hoc restriction to prevent the location of the cohort break λ to be estimated to be one of the youngest of oldest cohorts in the sample. In particular, in each year I restrict the location of the break to not be one of the 3 youngest or oldest cohorts.

Finally, I follow the bootstrap procedure described in Hansen (2000) to test the null hypothesis that no trend break occurs, ie. that δ_c is equal to 0.

This approach provides a summary measure for each outcome of the location of the trend break that can be easily compared across outcomes, along with measures of uncertainty which complement the visual impression of a trend break from the graphical analysis describe above. It also provides an estimate of the magnitude of the trend break, which again can be compared across outcomes.

D. Differencing the age profile between years

Building on McKenzie (2006), my third approach involves differencing age profiles between nearby years, leaving differenced cohort effects and the change in the age function between the two years. Under assumptions on how the impact of age varies across years which I highlight, changes in the slope of cohort effects are then identified as "mean shifts" in these differenced outcomes — similar to those in regression discontinuity designs. This approach has the advantage of a transparent visualization and relatively weak assumptions, but uses only one piece of variation in the data and is therefore less efficient than the other 2 approaches.

To build intuition for the method, I first outline an example based on the mean birth weight of infants by mother's age/birth cohort. Panel A of Figure 4 shows the mean birth weight of infants by mother's age, for mothers aged 18 to 27 in 1968 and 1970. The two age profiles closely track each other up to age 21. Starting at age 22 the two series diverge, with the mean birth weight in 1970 increasing in relation to that in 1968, and remaining higher for all subsequent ages.

In the context of the model above, the difference between these two age profiles reflects a combination of i) differences in cohort effects, and ii) differences between the impact of age across the two years. The widening difference between the two profiles after age 21 could suggest that the difference in maternal health between the 1947 and 1949 cohort is *smaller* than the difference in health between the 1948 and 1946, ie. that a slope change in cohort health occurs near the 1947 cohort. Alternatively, it could suggest that changes in age-specific factors between the two years disproportionately improved the health of infants born to mother's over the age of 22, versus younger mothers.

Specifically, in the context of the model in Equation 1, the difference between the two age profiles can be expressed as:

$$Y_{a,1970,c} - Y_{a,1968,c} = \underbrace{\gamma_c - \gamma_{c-2}}_{\text{difference in cohort effects for cohorts 2 birth years apart}} + \underbrace{f^{1970}(a) - f^{1968}(a)}_{\text{difference in age effect between years}} + \Delta\varepsilon \quad (6)$$

These differences are plotted in Panel B and reveal a clear "mean-shift" pattern. The differences are indexed on the x-axis by the associated birth cohort in 1970. These differences are between 12 and 24 grams for the 1943 to 1948 cohorts, and then drop sharply at the 1949 cohort to near 1 gram. They increase gradually for subsequent cohorts but remain below 10 grams.

This sudden discrete change implies either that the difference in age effects between the two years is not "smooth", or that there is a large slope change in health occurring near the 1947 cohort. That is, one explanation for the pattern is that the difference in the impact of age in the two years, $f^{1970}(a) - f^{1968}(a)$ has discontinuous mean shift between age 21 and age 22 — and the cohort effects are linear. Alternatively, if the difference in age effects, $f^{1970}(a) - f^{1968}(a)$, are smooth then this implies that the sequence of differences in cohort effects, $\gamma_c - \gamma_{c-2}$, exhibits a discrete shift between the 1948 and 1949. In other words, that $\gamma_{1948} - \gamma_{1946}$ is much larger than $\gamma_{1949} - \gamma_{1947}$ — ie. that there is a large change in the slope of cohort effects near the 1948 or 1947 cohort — leading to declines for subsequent cohorts relative to the trend for earlier cohorts.

This logic underlies the identification approach in this section. In particular, I impose assumptions on the ways in which age effects change over time — such as imposing that they are smooth in a formal sense — which will allow identification of features of the slope of cohort effects.

General approach

As in the example above, my approach in this section will involve differencing age profiles across neighboring years. To improve efficiency however, I will focus on the average of these differenced age profiles across all years in the data. I outline identification arguments based on a theoretical population in which cohorts are observed for infinitely many ages and years — ie. for "large P and large A". These identification results will then justify sample-analog estimators which I will implement to provide evidence of a change in the slope of cohort effects for a number of outcomes.

The key object I consider is the difference between i) an outcome for cohort c, age a, and year p, and ii) the outcome for individuals of the the same age a, but L years earlier, ie. in year $p-L$. Because of the linear dependency of age, year, and cohort the individuals in the earlier year are also *born* L years earlier — that is they are a member of cohort $c-L$.

Formally define the *fixed-aged L-year-difference in outcomes* as:

$$\tilde{\Delta}_L Y_{apc} \equiv Y_{apc} - Y_{a,p-L,c-L} \tag{7}$$

The sequence of these fixed-aged L-year-differences across ages is the difference between the "age profile" in a given year and that in a year L years earlier. Because the object compares outcomes for individuals of the same age in nearby years it will "difference out" any age-specific factors which are constant across the two years. AAs it compares outcomes between cohorts who are born L years apart, it will also include the difference in cohort effects between a cohort and the cohort born L years earlier — the *L-birth-year-difference in cohort effects*.

In the context of the conceptual model in Equation 1 the *fixed-aged L-year-differences* can be written as follows:

$$\tilde{\Delta}_L Y_{apc} = \underbrace{\gamma_c - \gamma_{c-L}}_{\text{L-birth-year-difference in cohort effects}} + \underbrace{f^p(a) - f^{p-L}(a)}_{\text{difference in age effects between years}} + \varepsilon_{apc} - \varepsilon_{a,p-L,c-L} \tag{8}$$

As outlined in the example above, the fixed-aged L-year-difference reflects a combination of i) the the L-birth-year-difference in cohort effects, ii) differences between the impact of age across the two years, and iii) differences between structural errors.

Now consider the expectation of Equation 8 across all years and ages in which the cohort c is observed in the theoretical population — ie. ages and years such that $c = p - a$:

$$\mathrm{E}\big[\tilde{\Delta}_L Y_{apc} \,\big|\, c\big] = \gamma_c - \gamma_{c-L} + \mathrm{E}\big[f^p(a) - f^{p-L}(a) \,\big|\, c\big] + \mathrm{E}\big[\varepsilon_{apc} - \varepsilon_{a,p-L,c-L} \,\big|\, c\big] \tag{9}$$

Below I outline arguments which use this expression to identify features of the *L-birth-year-difference in cohort effects*. I first outline assumptions under which the sequence $\gamma_c - \gamma_{c-L}$ can be identified up to a constant — and therefore that the slope of cohort effects is identified up to trend (as above). Second, I outline weaker assumptions which allow for the identification of level- or mean-shifts in the sequence $\gamma_c - \gamma_{c-L}$ — and therefore of discrete slope-changes in cohort effects.

Point-wise identification of differenced cohort effects

I first outline assumptions under which differenced age profiles can provide point-wise identification of differenced cohort effects.

First assume that the expectation of the *difference* between the structural errors which affect a cohort across all years and ages in which they are observed, and those of a cohort born L years earlier, is equal to 0:

$$\mathrm{E}\big[\varepsilon_{apc} - \varepsilon_{a,p-L,c-L} \,\big|\, c\big] = 0 \qquad \text{for all } c$$

This assumption would be trivially satisfied if for all cohorts the mean of any idiosyncratic errors that affect their outcomes — across all the given ages and years in which they are observed — are equal to 0.

Second, consider the expectation for a given cohort across all years and ages in which they are observed: the difference between the year-specific age-effect impacting that cohort and the year-specific age-effect for the *same age* L years earlier. Assume that this expectation is equal to some constant H. That is:

$$\mathrm{E}\big[f^p(a) - f^{p-L}(a) \,\big|\, c\big] = H \qquad \text{for all } c$$

Under these two assumptions the expression in Equation 9 becomes:

$$\mathrm{E}\big[\tilde{\Delta}_L Y_{apc} \,\big|\, c\big] = \gamma_c - \gamma_{c-L} + H \tag{10}$$

That is, under the above assumptions the differences in age profiles across nearby years identify differences between cohort effects, up to a constant. Specifically, the expected value of *fixed-aged L-year-*

differences identify *L-birth-year-difference in cohort effects* up to a constant. For example with $L = 1$, one can identify 1-birth-year differences in cohort effects, that is the discrete slope of cohort effects between cohort $c-L$ and cohort c. Differences between years further apart, for larger values of L, identify the difference in cohort effects with a larger lags.

How reasonable are the above assumptions? The first assumption on the idiosyncratic errors is quite similar to standard assumptions for other fixed-effect models.

The second assumption regarding changes in the age effects across years would be trivially satisfied under the assumption of no change in age effects across years. For example, with the additive separability assumption from the first identification approach the changes in age effects across years is uniform — it is just a year fixed-effect. In this case, H is just L times the linear trend in year effects across the entire population. This assumption would also be satisfied if age-by-year "shocks" are uncorrelated with cohort — ie. particular cohorts are not systematically lucky or unlucky in the long-run.[9]

The above population-based identification argument suggests a simple sample-analog estimator. Equation 10 implies that the expected value of fixed-aged L-year-differences identify L-birth-year-differences in cohort effects — up to a constant. I therefore use the sample-analog of this as an estimator. That is, I take the average of the fixed-aged L-year-differences across all ages and years in which a cohort is observed in the sample as an estimator of the L-birth-year-difference in cohort effects. Equivalently, I estimate the following simple fixed effects regression:

$$\tilde{\Delta}_L Y_{apc} = \Gamma_c^L + \mu + \eta_{apc} \tag{11}$$

and use the fixed effects $\hat{\Gamma}_c^L$ as estimators of the L-birth-year-differences in cohort effects, $\gamma_c - \gamma_{c-L}$.

Identification of deviations from known form

I now outline a method to identify deviations of cohort effects from linearity, under weaker assumptions than those outlined above. In particular, I allow for differences in age effects across years which take the form of a polynomial of a known order. I then show how, under these assumptions, one can identify a model with piecewise-linear cohort affects against a null model in which cohort effects are linear. The discontinuous slope change of the piecewise-linear cohort model will be evident as a discontinuity, or "mean-shift", in the differenced age profiles. I implement a formal test distinguishing between the two-models based on a model with a mean-shift and the structural change methodology in Hansen (2000).

Again assume outcomes depend on cohort effects and age effects that vary by year. Assume that the differences in age effects across years take the form of a polynomial of a known order D, eg. a cubic-in-age in each year. If the difference in age effects across years take this polynomial form, then the expected value of the differenced age effects taken across years — the second term in 9 — can be written as a polynomial in *cohort*.[10] Therefore Equation 9 can be rewritten as:

[9] NOTE: Need to write this out in more detail.

[10] NOTE: need to write this out for appendix

$$\mathrm{E}\big[\tilde{\Delta}_L Y_{apc}\big] = \gamma_c - \gamma_{c-L} + g(c) \tag{12}$$

where $g(c)$ is polynomial in cohort of order D. That is, the expected value of the differenced age profiles is equal to differenced cohort effects plus a nuisance function, which is a polynomial in cohort. Therefore, intutitively aspects of the cohort differences which are orthogonal to this polynomial can be identified. In particular, I test a null model in which cohort effects are linear against an alternative model in which they have a piecewise-linear structure — ie. exhibit a discontinuous slope change.

Consider a null model with linear cohort effects. That is, $\gamma_c = \beta \cdot c$. Then Equation 9 becomes:

$$\mathrm{E}\big[\tilde{\Delta}_L Y_{apc}\big] = \beta \cdot L + g(c) \tag{13}$$

That is the expected value of the differenced age profiles will take the form of a polynomial of order D.

Alternatively, consider a model in which the cohort effects have a piecewise-linear shape as outlined above in section IIB. That is, $\gamma_c = \beta \cdot c + \mathbb{1}_{c \geq \lambda} \cdot \delta \cdot (c - \lambda)$. Then 9 instead becomes:

$$\mathrm{E}\big[\tilde{\Delta}_L Y_{apc}\big] = \beta \cdot L + \mathbb{1}_{\lambda \leq c < \lambda + L} \cdot \delta \cdot (c - \lambda) + \mathbb{1}_{c \geq \lambda + L} \cdot \delta \cdot L + g(c) \tag{14}$$

Notably for $L = 1$, this becomes:

$$\mathrm{E}\big[\tilde{\Delta}_L Y_{apc}\big] = \beta \cdot L + \mathbb{1}_{c \geq \lambda + 1} \cdot \delta \cdot L + g(c) \tag{15}$$

That is is the expected value of the differenced age profiles will take the form of a polynomial of order D with a "mean-shift", or discontinuity, occurring one cohort after the location of the kink in the cohort effects, ie. $\lambda + 1$.

I will first provide an intuitive visual test of the alternative model, by plotting the sample average of the differenced age profiles specified above in Equation 11. Under the null model they should take a smooth polynomial form. Under the alternative model they should instead exhibit a discontinuity similar to that in regression discontinuity plots, in which the mean shifts discontinuously at a particular cohort.

I will then formally test between the two models, again using the framework of Hansen (2000). In particular, I will estimate the following model:

$$\tilde{\Delta}_L Y_{apc} = \beta + \mathbb{1}_{c \geq \lambda} \cdot \alpha_c + g(c) + \tilde{\varepsilon}_{apc} \tag{16}$$

by least squares.

Note that testing between this alternative model and the null model in equation 13 amounts to testing whether $\delta = 0$. Because the threshold location λ is unidentified under the null classical tests have non-standard distributions (the so-called Davies' Problem). I therefore implement the test suggested in Hansen (1999) based on a fixed-regressor bootstrap.

The model in Equation 16 also identifies the location and size of the cohort slope change. Under the assumption that cohort effects are otherwise linear, these estimates therefore provide an alternative estimate of change in cohort slope to that based on the trend break models shown above.

5. Main results: cohort declines in six outcomes

Using the data and methodology outlined above I present evidence of changes in the slope of "cohort effects" such that those born after 1947 or 1948 were less likely to work in white-collar occupations, earned less, gave birth to less healthy infants, and had a greater likelihood of dying prematurely, thann they would have had the trend for prior cohorts continued.

A. Graphical results

I begin by estimating the detrended, additively separable age-period-cohort models of Equation 3 for labor market outcomes, using the CPS-MORG data described above. Panels A and B of Figure 5 show the estimated cohort effects from this model for the share of employed men in white collar occupations and the median hourly wage of employed men respectively. For both outcomes there is a clear piecewise linear shape in which the cohort effects are increasing until the 1947 cohort, at which point there is sudden change in slope and the estimated cohort effects decline for subsequent cohorts.

Under the additive separability assumptions of this model, these figures suggest a large change in the slope of the 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. Recall that the cohort effects in this model are only identified up to a long-run trend, and I normalize the first and last cohort effects to be the same. Therefore, these results could reflect an absolute decline in ability starting with the 1947 cohort, or a sudden slowing of previously rapid improvements. In any event the estimated changes in slope are large. The results imply that men in the 1965 cohort were 6 percentage points less likely to work in white collar occupations and had a median wage nearly .15 log points lower than they would have had the trend for 1930-1947 cohorts continued.

Panels C and D of Figure 5 show results of applying the second strategy to identify cohort effect slope changes to the same labor market outcomes. They show the average of the difference between the age profile in a given year and the age profile two years earlier. In other words applying Equation 11 with L equal to 2. As outlined above this will be a consistent estimator — up to a constant — of the difference in cohort effects with 2-year gap — $\gamma_c - \gamma_{c-2}$, if age-by-year effects are orthogonal to cohort effects. Alternatively, if age-by-year effects take a smooth polynomial form, the pattern of these differences will reflect both a bias from the age-by-year interactions — which will inherit the smooth polynomial form — plus the difference in cohort effects. Therefore large "mean shifts" or "discontinuities" in these differences are evidence of a large, discontinuous change in the slope of cohort effects.

Both figures exhibit clear visual evidence of such discontinuities. In Panel C for the share of employed men in white collar occupations, the differenced age profiles are near .01 up to the 1948 cohort before discontinuously shifting to near -.015, and increasingly gradually for subsequent cohorts. These visual

results suggest that $\gamma_{1948} - \gamma_{1946}$ is much larger than $\gamma_{1947} - \gamma_{1945}$. In other words, there is a large change in the slope of cohort effects near the 1947 cohort of around -1.2 percentage points.

The results in Panel D for the median log hourly wage have an even more stark mean-shift pattern. For all cohorts born before 1946, except the 1936 cohort, the estimated differences are centered near 0. Then a discontinuous change is evident, and for all cohorts born in 1949 and after the differences are suddenly near -.03. In the context of the above model and identification results, this would again suggest that $\gamma_{1948} - \gamma_{1946}$ is much larger than $\gamma_{1947} - \gamma_{1945}$. And that there is a large change in the slope of cohort effects for wages also near the 1947 cohort of around -.015 log points.

Figure 6 shows similar results for infant health outcomes by mother's birth cohort, using the natality vital statistics microdata. Panels A and B show cohort effects from additively-separable age-period-cohort models based on Equation 3 for the mean birth weight of infants and the share of infants who are low birthweight. Recall that age and cohort in these models refer to the age and year of birth of the *mother*. Therefore 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.

Estimated cohort effects for both outcomes again exhibit a piecewise linear shape which suggests a large decline in maternal health starting with those born after 1947, relative to the prior cohort trend. The detrended cohort effects for mean birth weight are shown in Panel A. They increase from the normalized 0 for the 1935 cohort, to 30 grams by the 1947 cohort, before suddenly reversing trend and declining consistently to (the again normalized) 0 by the 1965 cohort. The pattern suggests that the impact of maternal health on infant's average birth weight changed trend after the 1947 cohort, such that the 1965 cohort would have given birth to infants 30 grams heavier on average had the change in the trend of cohort health not occurred. The estimated cohort effects for the share low birthweight have the reverse piecewise linear shape. Declining initially and reaching a minimum of -.008 for the 1947 cohort, before reversing trend and increasing for subsequent cohorts. The size of the change in slope suggests than the 1965 cohort would have had a low birthweight rate approximately .8 percentage points lower had the change in the maternal health cohort trend not occurred.

Panels C and D apply the age profile differencing methodology to these infant health outcomes, and reflect a similarly large change in the slope of cohort health. As above, these figures show the average of the difference between the maternal-age profile in given year and the age profile two years earlier.

These average differences for both outcomes, mean birth weight in Panel C and share low birthweight in panel D, exhibit remarkably sharp "mean shift" patterns, with a large discontinuous change in mean between the 1948 and 1949 cohort. For mean birth weight the differences are near 15 grams for the 1936 to 1948 cohort, then suddenly shift to near 2 grams at the 1949 cohort, and remain between 5 and -5 grams until the 1965 cohort. For share low birthweight the differences are between -.003 and -.0045 for the 1936 to 1948 cohort, and then shift suddenly to near 0 for the 1949 cohort, and remain at or slightly above that level until the 1965 cohort.

In the context of the model and identification results above, these mean-shift patterns are also consistent

with a large change in the slope of cohort effects near the 1947 cohort. The size of the discontinuities suggest that the slope of cohort effects changed discontinuously near the 1947 cohort — by around -7 grams and .15 percentage points respectively.

Figure 7 shows similar results for the log mortality rate of men and women, using data from the Human Mortality Database (HMD). Panels A and B show cohort effects from additively separable models based on Equation 3. The shape of the estimated cohort effects exhibit less of a piecewise linear shape than 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 the cohort effects decline — not precisely linearly from 0 to -.1 by the 1946 cohort, then suddenly reverse trend and increase rapidly until reaching above .03 by the late 1950s cohorts. They then flatten and decline slightly for subsequent cohorts. This pattern therefore suggests that men born between the late 1950s and early 1960s had mortality near .15 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 -.11 by the 1946 cohort, they then change trend and are nearly flat until the 1950 cohort. They then change trend *again* after the 1950 cohort and increase nearly linearly to 0 by the 1965 cohort. Overall, the size of the two slope changes imply that the 1965 cohort has had -.12 higher log morality 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 of Figure 7 apply the age profile differencing methodology to men's and women's log mortality. Again, these graphs show the average of the difference between age profile in given year and the age profile two years earlier. For men, the average differences are centered near -.035 for the 1930 to 1947 cohort and then exhibit a large discontinuous increase to near .02 for the 1948 cohort. They then decline smoothly, but rapidly back to -.035 by the late 1950s cohorts and further for subsequent cohorts. These results appear consistent with large age-by-year interactions, which take a smooth polynomial form, and a large discontinuous change in the slope of cohort effects near the 1947 cohort.

For women, as prefaced by the age-period cohort results above, evidence of a discontinuity is much less clear. There is some visual evidence of 2 discontinuous increases one from the 1947 and 1948 cohort, and another from the 1951 to 1952 cohort. This would be consistent with two changes in slope: one near the 1947 cohort and another near the 1951 cohort. However, the visual evidence is much less striking than for other outcomes.

B. Piecewise-linear cohort effect models

Guided by the shape of cohort effects evident from estimation of the age-period cohort models shown above, I now estimate models which explicitly specify the cohort effects to have a piecewise linear shape. These models also reveal evidence across outcomes of a change in the slope of cohort effects occurring near the 1947 or 1948 cohort. Using the structural change approach in Hansen (2000), I find that the

changes in slope are precisely estimated, statistically significant, large in magnitude, and in general robust to increasingly flexible age-by-year interactions, up to a separate quartic in age in each year.

Table 1 shows estimates of models with piecewise linear cohort effects based on Equation 4, for the six main outcomes. The models shown in this table include 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. In the appendix I also examine the robustness of the results to different control functions, including a model with no age-by-period interactions, and those with higher-order age-by-year polynomials.

The estimated location of the change in cohort slope are centered at the 1947 and 1948 cohorts. For both of the labor market outcomes — the share of men in white collar occupations and the median log wage — the change in slope of cohort effects are estimated to occur at the 1947 cohort. For both outcomes the 99 percent confidence intervals are small: including only 1947 for share white collar, and 1946 and 1947 for the median log wage model. For both of the maternal health outcomes, infant mean birth weight and share low birthweight, the slope change is estimated to occur just one cohort later at 1948. For both these outcomes the 99 percent confidence interval again includes *only* a single cohort, in this case the 1948 cohort. Finally, 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. And 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 white collar occupational share is -.017. This implies that the 1960 cohort has a roughly 22 percentage points lower share of workers in white collar occupations than they would have had the cohort effects continued to followed the trend of pre-1947 cohorts. For median log wage, the size of the cohort slope change of -.016 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 -6.9 grams implies that the 1960 cohort has given birth to infants 82.7 grams lighter on average than they would have if the pre-1948 cohort trend had continued. More strikingly, the share low birthweight estimated slope change size of .0023 implies that the 1960 cohort had a low birthweight rate near 2.8 percentage points lower higher than they would had the earlier cohort trend continued. Given the ow birthweight rate in nationally in 1975 of only 7.4, this effect is quite large.

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

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

linear. For all models, the value of the F-type statistic for the true data is larger than all of the 1000 bootstrap repetitions — suggesting a P-value of less than .001 for the null of no linear cohort effects.

Appendix Tables A1 through A3 show the robustness of these estimates to different specifications of the age-by-year control function. I examine the robustness to including different age-by-year interactions, from allowing none, to including separate quadratic, cubic, or quartic polynomials in age in each year. For both labor market and maternal health outcomes, the finding of a change in the slope of cohort effects located at or near the 1947 or 1948 cohort are quite robust. For the share white collar all specifications detect a change in the slope of cohort effects, of negative sign, located precisely at the 1947 cohort and. 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 1946-1947 as alternatives break locations. Results across *all* specification for both the mean birth weight and low birthweight share reveal estimated break locations between 1947 and 1949. These estimated slope changes are all of negative sign, though somewhat varying in magnitude.

The mortality results are somewhat less robust.[11] However, findings for white mortality in Reynolds (2019) using a different methodology reveal highly sugestive and robust evidence of a decline located near 1946 for white men and 1949 for white women.

C. Mean-shift models of differenced age profiles

Following the methodology outlined above, and again based on Hansen (2000), I fit "mean-shift" models to the differenced age profiles similar to those shown in Figures 5 to 7. In particular, I estimate models based on Equation 16, with L equal to 1. Under the assumption that age-by-year interactions are either non-existent or orthogonal to cohort effects, these models provide an alternative estimate of the change in cohort slope estimated above. I also estimate models which include a polynomial in birth cohort, which will provide identification of a discrete change in cohort slope under weaker assumptions. I report baseline estimates including a quadratic in cohort, and show robustness in appendix tables.

Table 2 shows results of fitting these mean-shift models to the differenced age profiles with a 1-year lag, for each of the six main outcomes. The results confirm the existence of a discontinuous change in slope in all of the outcomes at or near the 1948 cohort. The size of the estimated mean-shifts are also similar in magnitude to those of the cohort slope changes estimated above, with those for labor market outcomes slightly larger than earlier estimates.

The location of the estimated shift in mean — paralleling the visual impression from the earlier graphs and the estimation of piecewise linear cohort-effect models — are again located at or near 1948. For *all* of the first four outcomes — share white collar, median log wage, mean birth weight, and share low birthweight — the estimated location of the mean-shift is precisely estimated to be the 1948 cohort. The 99 percent confidence interval for all of these estimates include only the 1948 cohort. The estimated locations of the mean shift for log mortality are 1947 and 1951 respectively, and again the 99 percent confidence intervals include only a single cohort.

[11] Need to describe in more detail. Think about why these findings may be less robust.

The estimated mean-shift for the two labor market outcomes — and therefore implied change in slope of cohort effects — are slightly larger than the change in slope estimated above from piecewise-linear models. That for share white collar is -0.023 and that for median log wage is -0.021, implying an even larger decline in the cohort slope of labor market ability. The estimated mean shifts for intergenerational infant health, of -7.5 grams for mean birth weight and .0019 for share low birthweight, imply a decline in maternal health similar to earlier estimates. The mean-shift estimates for log mortality are remarkably similar in size to the earlier estimates of the change in cohort slope — at .029 and .028 for men and women respectively.

Applying the bootstrap test of Hansen (1999) to test whether a mean-shift exists, I fail to reject null of no mean-shift at a very low significance level. 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 mean-shift. Following the logic outlined above this therefore suggests that the data are consistent with a discontinuous change in the slope of cohort effects — located at or near the 1948 cohort — for all outcomes.[12]

D. Summary

Using multiple methods, and across different outcomes, datasets, and years, the above results paint a consistent picture of a cross-cohort decline in health and human capital beginning with those born in 1947 or 1948.

I find evidence of a similar change in the slope of cohort effects for very different outcomes, suggesting the decline in health and human capital is quite broad. I find cohort declines in: occupational standing and earnings of prime-age men; the health of mothers as proxied by the birth weight of their infants; and in health more broadly as measured by the probability of death at different ages.

The different datasets were collected in different ways, cover different years and individuals at different ages, therefore it appears unlikely that the findings are driven by a data artifact. For example, the median log wage data comes from survey data, focuses on employed men between the ages of 25 and 54, and covers the years 1979-1993; while the intergenerational infant health data come from administrative records reported on birth certificates, focus on women who gave birth between the ages of 18 and 40, and comes from births occurring between 1968 and 1985. Data problems would therefore have to coincide in a very peculiar way to generate the same bias across these two very different datasets.

The cohort declines are also evident using 3 different methods to address the age-period-cohort problem, and are robust to various assumptions on the nature of age-by-period interactions. There is a clear visual impression of a piecewise-linear shape of cohort effects from estimates of standard additively-separable cohort models. Imposing this piecewise-linear shape to cohort effects I detect changes in slope at or near the 1948 cohort, which are robust to different specifications of age-by-year effects, up to including a separate quartic-in-age in each year. Finally, I implement a novel identification strategy based on differencing age profiles across nearby years. This method shows evidence of a similar change in the slope of cohort effects, evident both visually and detected using structural break estimation and testing.

[12] Need to write up robustness to other control functions. And maybe to using L equal to 2.

6. Heterogeneity: cohort declines widespread among native-born

Having presented evidence of a decline in cohort health and human capital beginning for cohorts born after 1947, I now consider heterogeneity in this decline across demographic groups. I find that the decline is generally remarkably widespread, cutting across racial lines and geography. Importantly, however the decline appears concentrated among those born in the United States and is not evident for those born outside the United States. This finding suggests that the cohort declines may have been caused by an early-life factor *specific* to the United States.

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

The cohort-specific decline in earnings ability appears remarkably similar across racial groups. The location of the estimated cohort-slope change in models of the median log wage are remarkably similar: at 1946 for whites, 1948 for blacks, and 1949 for Hispanics and other races. Further, the confidence intervals for all of these estimates include the 1947 cohort (though the estimate for the other race group is very imprecise). The size of the change in cohort slope are very similar for whites, Hispanics, and the other race group, at near -.013 log points. The change is notably larger for blacks, nearly double that of the other groups at -0.025 log points. I also again find for all models p-values less that .001 for the null that no change in slope exist.

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: .0010 for whites, .0008 for blacks, and .0007 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.

B. Cohort declines by Census Region

Table 4 examines analogous heterogeneity by place of residence, and shows that similar declines in cohort health and human capital are estimated across each of the four Census Regions. I again estimate the piecewise-linear cohort effect models based on Equation 4, while controlling for age fixed effects and year fixed effects. I return to the unrestricted sample including cohorts born up to 1965. 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. One view of this analysis is therefore as an additional robustness test. 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. Below, I will also directly examine heterogeneity by place of birth for those outcomes for which it is recorded in my data.

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 .0013. That for the Northeast, Midwest, and West are slightly smaller at .0012 and .0011 respectively. The standard errors of .0001 suggest the difference between the South and West is likely only marginally significant.

The results for the log mortality of both men and women also suggest a widespread decline in health. For these outcomes I use a control function which includes a separate quadratic-in-age in each year — in addtion to the age and year fixed effects included above. As described above I use mortality estimates from the United States Mortality Database.[13]

The results are shown in the bottom half of Table 4 and reveal remarkably similar estimates across the

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

4 Census Regions. For men's log mortality the location of the change in cohort slope is estimated to be precisely the 1946 cohort for *all 4 regions*, with each 99 percent confidence interval including only that cohort. For women, the location estimates range from only the 1948 to the 1950 cohort across regions. The estimate size of the change in slope is also quite similar across regions, as well as across the 2 sexes. The size estimates for men range from .0284 in the Northeast to .0341 in the South. Those for women range from 0.0269 in the West to .0331 in the South. [14]

C. Cohort declines for native vs. foreign 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 inside 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 mother's 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.

The fact that the natality records include such a large sample, and in many years the universe of births, allows for relative precise estimation of models focusing on the foreign-born population. 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.

Figure 8 shows cohort effects separately for foreign and native-born mothers, based on additively-separable age-period-cohort models. As for the earlier figures, the models are based on Equation 3, and assume no age-by-period interactions. The shape of cohort effects for native-born closely follows that shown in panel A of Figure 8 for the full sample. 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 mother's 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 may not exist.

Panel A of Table 5 shows these results for mothers born in the United States. Across all specifications of the age-by-period control function the location of the estimated change in cohort slope varies from only

[14] Note that these regional results are not fully robust to different control functions. However, using the quadratic control function the findings of a widespread decline at or near the late 1940s cohorts hold at even smaller geographic scale. Each of 9 divisions have quite similar estimates. I want to check this in more detail.

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 mothers. These results should likely be treated with more caution than the maternal health results for 2 reasons. First, the Census data only records outcomes every 10 years — hampering the ability to ditinguish cohort effects from age-by-year interactions. Second, they comprise a smaller sample of the population — and therefore include particularly small samples of the foreign-born.

With this caveats in mind, Appendix Table A5 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 similar magnitude to the estimate from the CPS data for the full population of men. 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.[15] However, adding polynomial age-by-year interactions yields estimates of a *positive* change in slope — ie. improvements in occupational status relative to the prior trend — either at the 1932 cohort or the 1956 cohort.

D. Lack of variation by state of birth/county of residence within the United States

Could add additional estimates of heterogeneity:

- state/region of birth
- charactersitics of counties — eg. rural/urban etc.

[15] EDIT: (could be explained by changes in immigration policy in 1960s leading to less-skilled imigrant entrants).

7. Connection to education and test score declines

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

A. Review of educational and test score declines

Previous authors have noted a sudden decline in the educational attainment for cohorts of Americans born after the late 1940s. Heckman and LaFontaine (2010) estimate that the U.S. high school graduation rate peaked at around 80 percent in the late 1960s — roughly when the 1946 cohort was 18 — and has declined by 4-5 percentage points since then. Card and Lemieux (2001b) highlight a sudden 12 percent fall in college entrance rates for men from 1968 to 1978 — approximately the 1947 cohort to the 1957 cohort — and a stagnation in prior improvements for women. These authors are largely unable to find an answer to why the previous trend of improvement in educational attainment suddenly stagnated and even reversed. Card and Lemieux (2001b) present an extensive study of possible causes of the decline and conclude that for women it could be explained by low returns to education and cohort size, but that for men the decline represents a fundamental trend break with no observable explanation. Acemoglu et al. (2012) also note this stagnation in educational attainment, and suggest that the sharpness of the change in trend by cohorts suggests it is unlikely to be caused by a sudden change in the school system, and that "other factors are thus likely to be at play."

Figure A3 summarizes these patterns using the CPS MORG data. For simplicity, I pool the 1990 to 2018 years of data and individuals age 25 to 75, and calculate the share of each cohort who have achieved different levels of educational attainment. I also calculate approximate average years of schooling for each cohort based on the 16 schooling categories in the CPS. The existence of a similar change in cohort slope is also robust to controlling for age effects, or age-and-year effects. While the stagnating high school graduation rates and declining college completion rates have been previously documented, to my knowledge the fact that the share of each cohort attaining an advanced degree also declined between the 1946 and 1965 cohort is novel.

For men the cross-cohort trends in years of schooling, high school graduation (or GED), bachelor's degree attainment, all exhibit clear breaks — or slope changes — at the 1947 cohort such that prior improvement in educational attainment stops and *reverses* before declining through the mid-1960s cohorts. For women there is clear evidence of a slowing in improvement located near the late-1940s cohorts though it is less sharp and consistently located across cohorts. Further, years of schooling and high school at-

tainment for women stagnate but do not decline in absolute terms. Notably, women's college attainment continues to increase until the 1950 cohort, before declining slightly.

There was also a widely noted decline in standardized test scores beginning in the late 1960s, which appears to closely match the timing by cohort of declines in other measures of human capital shown above. Most widely noted was a decline in Scholastic Aptitude Test (SAT) scores beginning in the early 1960s and lasting until the mid-1980s. Appendix Figure 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. 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 E. Wiley, 1975).

With regards to the cause, there were suggestions of a role for loosening of academic standards and social unrest of the 1960s ((Price and Carpenter, 1978). But these suggestions were largely based on casual empiricism and some of the timing appears inconsistent. Zajonc (1976) suggested that the test score declines were the result of changes across cohorts in average birth order — the share of individuals in each cohort who were the second or later born in their family increased after the late 1940s. Subsequent research with microdata however found very small effects of birth order on test scores — casting doubt that the differences across cohorts were large enough to explain the magnitude of the decline (CITE).

The shared timing of these declines by cohort in educational attainment and test scores to those documented above in other measures of health and human capital suggests they could plausibly be linked. A natural question is whether the direct causal effect of education on earnings and health can explain the other declines in health and earnings which I document. The complex and multi-directional causal connections between education, health, earnings, and other human capital investments makes answering this question challenging. Below I make an initial attempt and present some pieces of evidence which appear inconsistent with the "education-only" explanation. For example, the implied effect of education on mortality — under the assumption that other unobservables remain unchanged — appears implausibly large. Additionally, a

number of declines in other outcomes hold *conditional* on education within narrowly defined bins, in ways that would require very strong and unusual changes in selection effects, eg. the relationship between unobservables and years of schooling. Even if all the declines I've documented above were caused by the earlier declines in education and test scores — it would remain a notable finding that recent trends in health, mortality, and labor market outcomes are still being driven by changes in education supply nearly 50 years ago.

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

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

I now consider 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. 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. I focus on the low birthweight rate for easier comparison with prior estimates in the literature. 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.

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." [16]

Table 6 reports the results. Interestingly, the implied return for earnings to a year of schooling is .138 which is only slightly larger than OLS estimates and closely matches many of the IV estimates summarized in Card (2001). However, a separate IV estimate restricted to only Blacks 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 the 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 -.0092 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 -0.0096 to -.0099.

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.[17]

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. 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 to be just 37 percent of what it would have been with just a high school degree). 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*.[18]

These estimates are much larger than cross-sectional differences. For example the ratio of white mortality rates at ages 25 to 64 between those with 16+ years of education and 12 years of education where .48 and .55 for men and women respectively, and decreased by 2001 (Ma et al., 2017). Further, instrumental

[16]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).

[17]NOTE: I wonder if I can quantify this somehow?

[18]NOTE: Need to construct standard errors for these. Use bootstrap of TS2sls formula in Inoue and Solon

variable estimates of the causal effect of education on mortality using changes in compulsory schooling laws generally find *much* smaller causal effects than even 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 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 therefore appears *much* too large to be explained by the decline in education alone, and therefore strongly suggests that there was a broader decline in health and human capital for these cohorts.

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

I now show evidence of cohort declines in earnings and maternal health within some narrowly defined educational bins — health and earnings ability appears to have declined for post-1947 cohorts even *conditional* on education. First, I show robust evidence of a change in the slope of cohort effects of median earnings of those without a bachelor's degree. Then, I show evidence of similar change in slope of cohort effects for maternal health, as measured by infant birth weight, at many levels of maternal education, including exactly 12 years of education and exactly 16 years of education. These findings suggest either that the change in cohort slope was driven by a decline in latent health and human capital *broader* than the educational declines alone; or that the change in educational attainment for late-1940s cohorts also involved large and unusual changes in selection effects, eg. the relationship between unobservables and years of schooling.

Earnings by education

Table 7 shows estimates of the change in slope of cohort effects in median log earnings of employed men separately for those with and without a bachelor's degree. I estimate the models with piecewise linear cohort effects based on Equation 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.

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. This will provide as estimate of the implied return to schooling, under the assumption that unobserved ability across cohorts remained unchanged in this group. The last row of Table 6 shows the results. The implied causal effect of year of schooling on log earnings necessary for schooling alone to explain the decline is .187. This estimate is much larger than the cross-sectional relationship between earnings and schooling found in most

datasets, and larger than most — though not all — of the IV estimates reviewed in Card (2001). Therefore, the decline in earnings for this group could be explained by the direct effect of schooling if one thinks the causal effect of schooling for this group is much larger the the cross-sectional relationship and larger than most IV estimates. Of course, as described above a complex selection explanation — in which a trend break in schooling "supply" also changed the correlation between ability and obtaining a bachelor's degree could also contribute to this earnings decline.

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

The existence of a cohort decline for those *without* a bachelor's degree and no similar decline for those *with* a bachelor's degree implies a cohort-specific increase in the college-high-school wage gap — likely the same phenomenon documented in Card and Lemieux (2001a). Card and Lemieux (2001a) use census data and pool cohorts and ages into larger 5-age/cohort groupings. They therefore do not detect the sharpness by cohort of the decline in earnings of those without a college degree (and of the wage-gap). The discontinuous nature of the slope change appears hard to reconcile with their explanation based on falling relative supply of the college educated and imperfect substitutability of workers of different ages. For example, it would require workers only a year or two apart in age to be very poor substitutes.[19]

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. Within the context of a broad decline in health and human capital, it is not obvious why those with a bachelor's degree fail to have a decline in earnings. One explanation would be that the factor which declined across cohorts is a *substitute* in the labor market with a college education, in other words that a bachelor's degree has a "protective effect" in the labor market against the broader decline in human capital.

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

[19]Further, absolute declines — relative to the prior trend — for those without a bachelor's degree are inconsistent with their simple model, but may be consistent with a more complex models such as Acemoglu and Autor task framework.

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.

The natality detail files record mothers' years of schooling as reported on birth certificates starting in 1969. Given the smaller number of births in each cell and the lower incidence of low birthweight births among highly educated mothers, to improve precision I focus on the mean birth weight of infants. While mother's education is missing for a non-trivial fraction of mother's, as shown above the cohort decline in infant health is of similar size for the sample of births to mother's with non-missing education information. I further restrict the sample to include mother's over the age of 22, the years 1969 to 1990, and the maternal cohorts 1938 to 1960.

I first apply the age profile differencing methodology to infant birth weight for the two maternal education levels which comprise the largest share of the population: exactly 12 years and 16 years of schooling. Figure 9 shows for these two groups of births the average of the difference between the age profile in a given year and the age profile two years earlier. That is, the result of applying Equation 9 with L equat to 2 years.

Panels A and B each reveal a clear mean-shift pattern, implying a discontinuous change in the cohort slope of maternal health *conditional* on education for those with exactly 12 years of education and exactly 16 years of education. Panel A shows the results for mother's with exactly 12 years of education. The average lagged differences are centered near 10 grams between the 1938 and 1948 cohorts — with some variability between around 4 and 14 grams. The differences then appear to exhibit a discrete shift beginning with the 1949 cohort — falling suddenly to -2 grams — and remaining below or nearly below 0 until the 1960 cohort. Panel B shows a larger and even starker mean-shift for mother's with exactly 16 years of education. For this group, the lagged differences for all but one of the 1938 to 1948 cohorts are between 14 and 25 grams. They then discretely drop to near 5 grams, and remain between 8 and 0 grams until the 1960 cohort. This visual mean shift of more than 10 grams suggests a discrete change in the slope of maternal health for mother's with 16 years of education with a "size" of 5 grams.

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

Table 8 applies the piecewise linear cohort effect method to 5 educational categories, and shows evidence of a similar change in the cohort slope of maternal health across the maternal educational distribution. I apply the model based on Equation 4 with age and year fixed effects separately for the following 5 cate-

gories of maternal education: less than high school, high school, some college, 4 years college, and 5+ years of college. For each of the education levels the models detect a change in cohort slope between the 1946 and 1948 cohorts. The size of the change in slope are all negative — implying a relative decline in cohort health — and vary from -.99 grams for the high school group to -3.31 for the some college group. Based on the bootstrap-based test of existence, all the changes in slope are significant. Appendix Table A7 shows that controlling for a separate quadratic-in-age in each year suggests declines in cohort slope at a similar location and at least twice the magnitude, for all groups except those with 5+ years of college.

The above results show strong evidence of cohort declines in maternal health *conditional* on mother's education across the education distribution. These results would rule out an explanation in which a change in the supply of education alone has driven maternal health declines, if that change in supply affected 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 A3 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.

8. Candidate explanation: post-war decline in respiratory health environment

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

My hypothesis is that there was a broad decline in the respiratory health "environment" after 1946 or 1947 — which increased respiratory mortality for infants and adults in the short run — and had a lasting effect on the health of cohorts who were in utero or infancy during that period. The key piece of evidence for

a broad decline in respiratory health is that mortality from particularly respiratory causes began to increase simultaneously for both infants *and* elderly adults near 1947. The hypothesized change in the respiratory health environment could conceivably have been caused by a decline in air quality, for example due to post-war industrial growth or increased driving. Alternatively, the risk of respiratory infections may have increased for reasons unrelated to air quality.

A number of factors suggest a change in the respiratory health environment in infancy is a plausible cause of the cohort declines documented above. I showed that the change in cohort slope in maternal health was concentrated among those born in the United States, and not evident for foreign born mothers. The decline in test scores at age 17 suggests that the cause of the declines predated labor market entry. Therefore, a plausible candidate cause of the declines would be some early life factor unique to the United States which changed trend suddenly in the late 1940s. Further, past evidence suggests that a decline the respiratory health of infants, or of air quality specifically, would have long-run effects on exposed infants health and human capital as adults. A large literature reviewed in Currie and Almond (2011) presents evidence of effects of infant health on educational attainment, health, and labor market outcomes. A number of papers also find evidence of long-run effects of infant exposure to respiratory diseases and air pollution specifically. [20]

Rapid improvements in the infant mortality rate and the overall age-adjusted death rate began to noticeably stall near-1950, prompting a number of reports and articles. In the 1960s, a series of 12 reports from the National Center for Health Statistics examined the contributions of different causes of death, documented variation within the United States, compared the United States experience to that of other countries, and considered the possible root cause.[21] The slowdown was considered surprising in the context of a booming post-war economy and rapid improvements in medical technology. But it appears to have been largely forgotten as large declines in mortality rates began again in the late 1960s and early 1970s (Crimmins, 1981).

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

Figure 10 shows evidence of a post-war increase in respiratory mortality for infants and elderly adults. As described above all data is transcribed from printed Vital Statistics volumes. Panel A shows a clear

[20] Bhalotra and Venkataramani (2015) find evidence of long-run gains in adult outcomes driven by reductions in pneumonia exposure in infancy in the United States after 1937. Isen et al. (2017) find that reductions in particulate matter in utero following the Clean Air Act led to gains in earnings when these infants were adults. Almond (2006) found long run effects of in-utero exposure to the 1918 Influenza Pandemic. Almond and Chay (2006), Chay et al. (2009), and Chay et al. (2014) find long-run effects of infant health gains for African Americans in the 1960s South — which particularly included reductions in pneumonia mortality — led to gains in test scores, educational attainment, earnings, and maternal health — with an intergenerational effect on infants.

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

trend break in infant morality from all respiratory diseases. I include deaths from pneumonia and influenza, as well as a category called "Other Respiratory Diseases." This category includes deaths for IRDS. The mortality rate from these respiratory diseases feel rapidly from more than 10 deaths per 1,000 births in 1937 to nearly 4 deaths per 1,000 in 1946. The pace of decline then noticeably slows and by 1958 the respiratory infant mortality rate is nearly identical to that in 1946. Unfortunately, deaths from IRDS were reclassified into a broader category between 1959 and 1966, creating a break in the series. However, by 1967 when IRDS, was reclassified total respiratory infant mortality was slightly *higher* than it had been in the 1940s.

In Panel B, I attempt to provide the best picture of increases in infant mortality from IRDS based on the cause of death groupings which are reported in the printed volumes. The results in Panel B are consistent with IRDS barely featuring as a cause of infant death in the 1930s and early 1940s, and beginning a rapid increase around 1946 which continued until the early 1970s. By the 1960s, IRDS was one of the leading causes of infant death. As noted above, this cause was classified into a different cause of death grouping in the 1950s — from "Other respiratory diseases" to "Diseases peculiar to early infancy" — and then was reclassified back to "Other respiratory diseases" after 1968. Infant mortality from "Other respiratory diseases" was less than 1 death per 1,000 births in 1933 and declined slightly until 1946. Starting after that year it began to increase until 1956 — when IRDS was removed from that classification. During the subsequent period between 1957 and 1968 when IRDS was grouped in the category "Diseases peculiar to early infancy", deathins in this category increased rapidly . Finally in 1966 when IRDS is returned to "Other respiratory disease" the level of mortality in that grouping is nearly 6 times higher than it was in 1946 — at nearly 3 deaths per 1,000 births. Surfactant therapy, a treatment for IRDS, was developed in the late 1960s, potentially causing the rapid reductions in mortality from "Other Respiratory Diseases" after 1970.

Panel C and D show that beginning near 1946 there was also an increase in *adult* mortality from respiratory causes, including Chronic Obstructive Pulmonary Disease (COPD). Panel C shows cause-specific mortality from for white men age 65 to 69 for 3 cause of death groupings.[22] The series in red squares show mortality from "Other respiratory diseases" which primarily includes causes of death which would now be called Chronic Obstructive Pulmonary Disease (COPD), such as Bronchitis and Emphysema. Mortality from this cause grouping is remarkably flat at less than 40 deaths per 1,000 between 1933 and the mid-1940s. It then begins to increase gradually after the late 1940s. These increases continue unabated until the 1960s — during which time mortality from this cause has more than quadrupled from less than 40 *to 200* deaths per 1,000.

The series denoted by green crosses shows mortality from pneumonia and influenza, while blue circles show mortality from all respiratory diseases — combining the other two series. Pneumonia and influenza mortality exhibit rapid declines after 1937, which are generally credited to the development and use of Sulfa drugs (Jayachandran et al., 2010; Bhalotra and Venkataramani, 2015). The declines in mortality from these causes however suddenly stop in the late 1940s. Interestingly, the combination of the trends in these two groupings results in a U-shaped pattern in *total* respiratory mortality. Mortality from all respiratory diseases

[22] I plan to change these figures to show mortality from all races combined.

declined rapidly from the late 1930s to the late 1940s, from nearly 400 deaths per 1,000 to less than 150, but then reversed trend and began to increase — climbing back to more than 300 deaths per 1,000 by the mid-1960s. The declines in the first period were driven by rapid declines in pneumonia and influenza, while the change in trend was the result of a combination of these declines stalling *and* a rapid increase in previously rare mortality from chronic respiratory diseases.

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

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

Additionally, the trend break in IRDS mortality coincides approximately with a stagnation in infant mortality from *all causes*. Further, the infant mortality rate in the United States diverged suddenly in 1946 from that of other countries, which had previously had a similar infant mortality rate. Appendix Figure A5 shows these patterns. This sudden divergence of the infant mortality rate in the United States from that in other countries would also be consistent with a decline in infant health in the United States under some assumptions. In particular, assume as in Almond and Chay (2006) and Bozzoli et al. (2009) that infant death depends on latent health and a threshold of viability, and allow the distribution of latent health and the threshold to both vary across years. Then the above patterns would be consistent with a trend break and subsequent decline in latent infant health in the United States if: i) the year-specific threshold of viability is the same in the United States and the other countries, and ii) mean latent health continues to follow a linear trend in the other countries.

Finally, Appendix Figure A6 shows that the sex ratio at birth — though quite variable across nearby years — appears to begin a general declining trend after the late 1940s. The sex ratio at birth, the ratio of male to female live births, is often viewed as a proxy for fetal health because male fetuses are generally less robust than female fetuses. Therefore this pattern is also consistent with a decline in infant health beginning

in the late-1940s.

In summary, the above section shows evidence of post-war increases in respiratory mortality of both infants and adults which would be consistent with a broad decline in the respiratory health environment in that period. Previous evidence suggests that the respiratory environment in utero and early in life can have a lasting effect on adult health, cognitive ability, and human capital broadly defined. Further the broad decline in adult outcomes, documented in the other sections of this paper, begins with cohorts born after 1947. Therefore the cohorts who exhibit declining adult outcomes are also the cohorts who would have been exposed in utero or infancy to a declining respiratory health environment. I therefore suggest that the broad cohort declines in health and human capital may be the result of the lasting impact of early-life exposure to a post-war decline in the respiratory health environment.[23]

9. Conclusion

In this paper I presented evidence of a precisely timed and broad decline in the health and human capital of cohorts of Americans beginning with those born in 1947 or 1948, relative to the prior trend. This decline appears to have predated labor market entry and to have played a role in test score and education declines in the 1960s and 1970s, as well as recent mortality increases of white Americans. Further, it has left a distinct imprint on earnings, occupational status, and mortality risk. It is also evident in maternal health and therefore appears to have had an intergenerational effect on infants in the next generation.

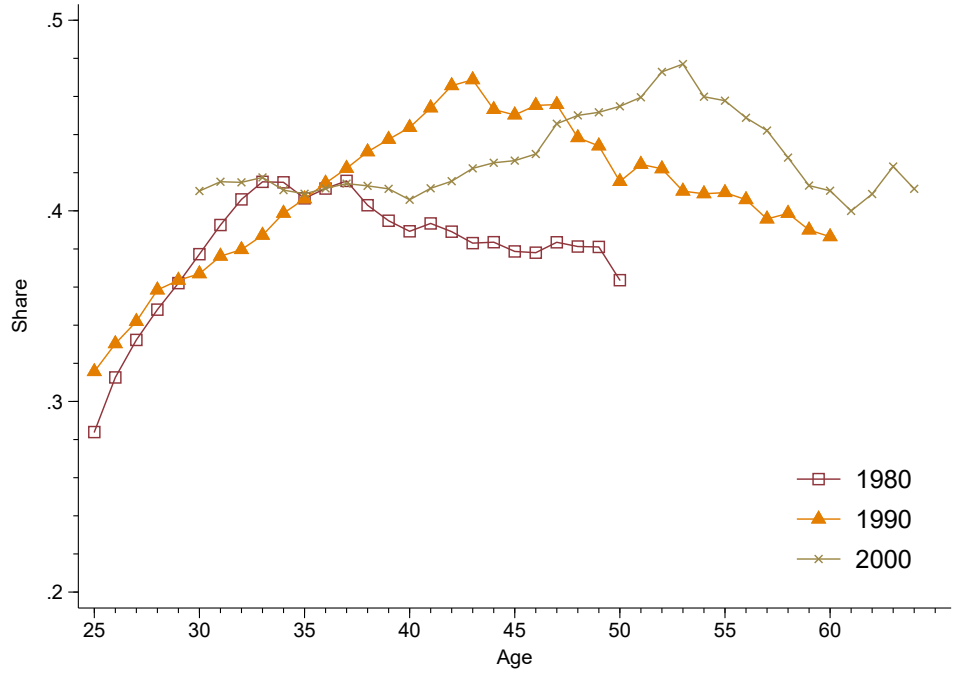
Something appears to have gone deeply wrong — by at least age 17 — for cohorts of Americans born after the late-1940s. I tentatively suggested the decline may have been caused by a post-war decline in the respiratory health environment. Future research should continue the search for the decline's underlying 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 likely interacted in interesting 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.

[23] I would like to also use state-level mortality data to document examine whether respiratory health decline is widespread.

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

A: By age



B: By birth cohort

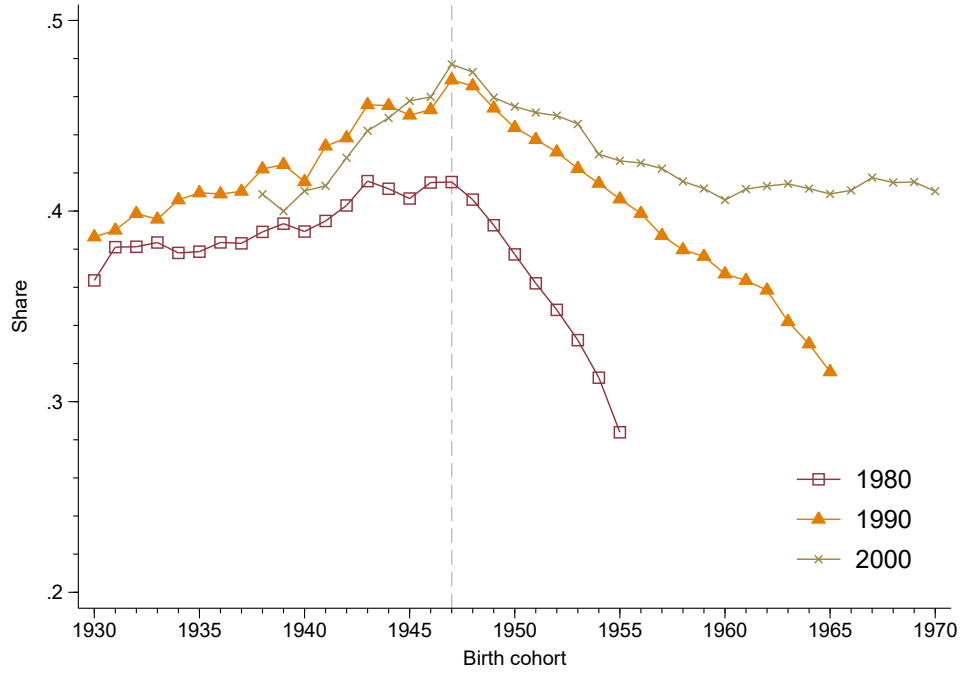
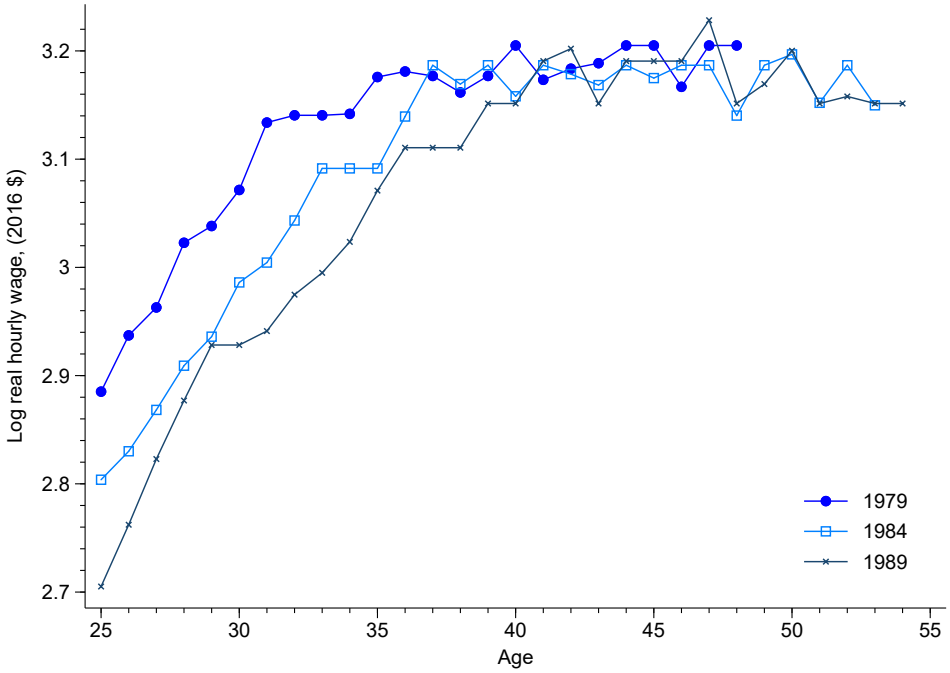


Figure 2: Median hourly wage of men

A: By age



B: By birth cohort

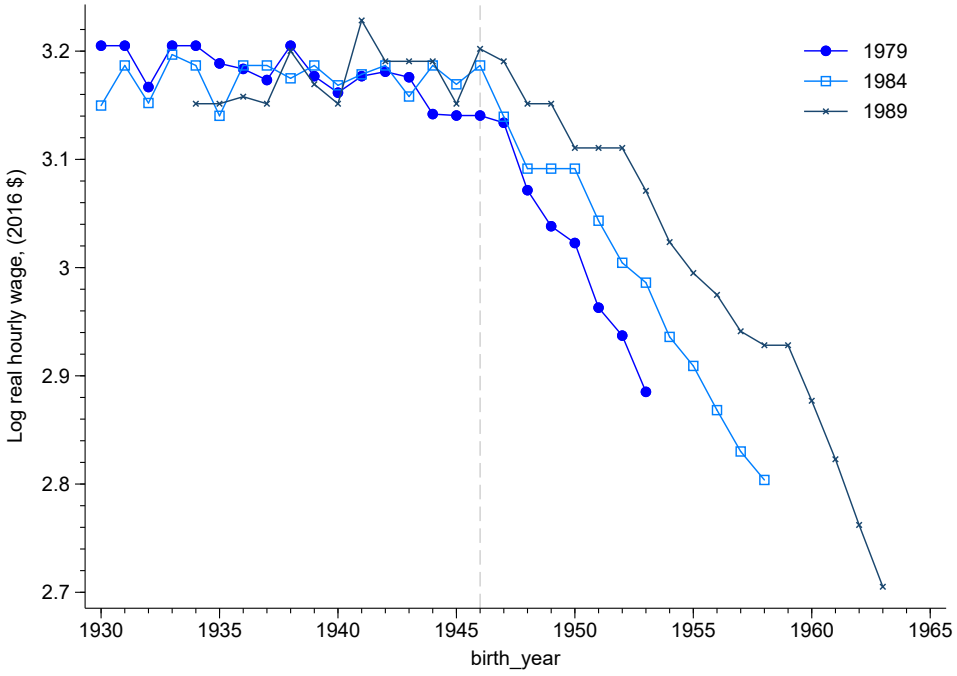
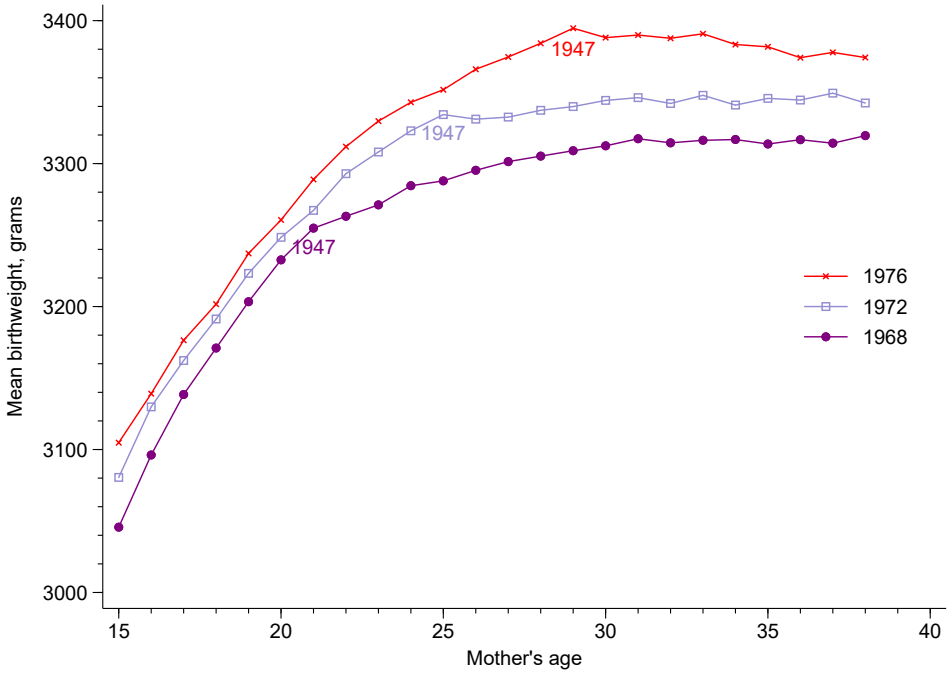


Figure 3: Mean birth weight of infants

A: By mother's age



B: By mother's birth cohort

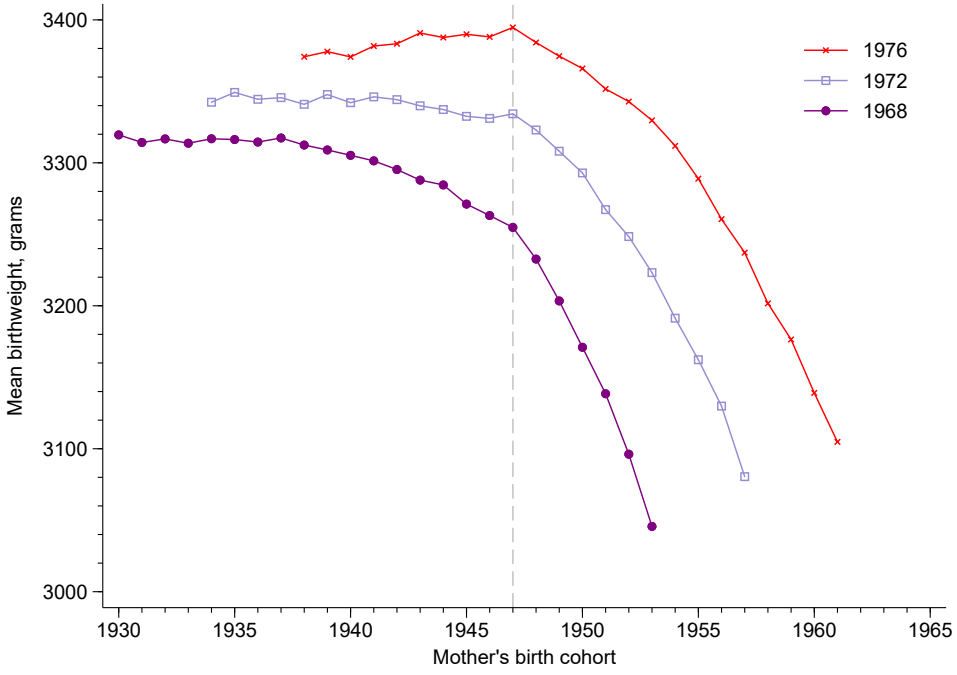
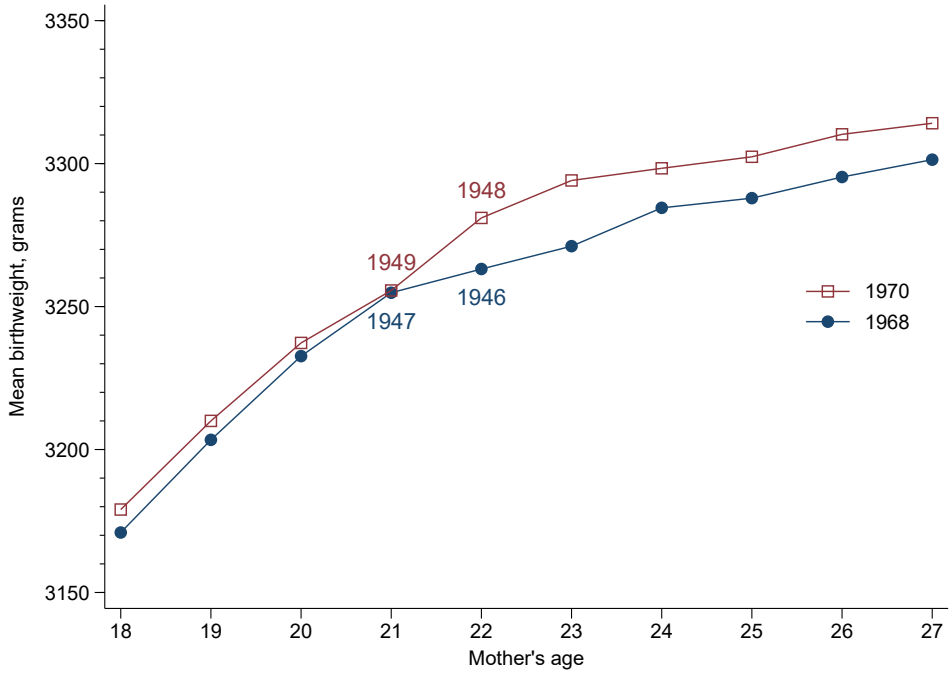
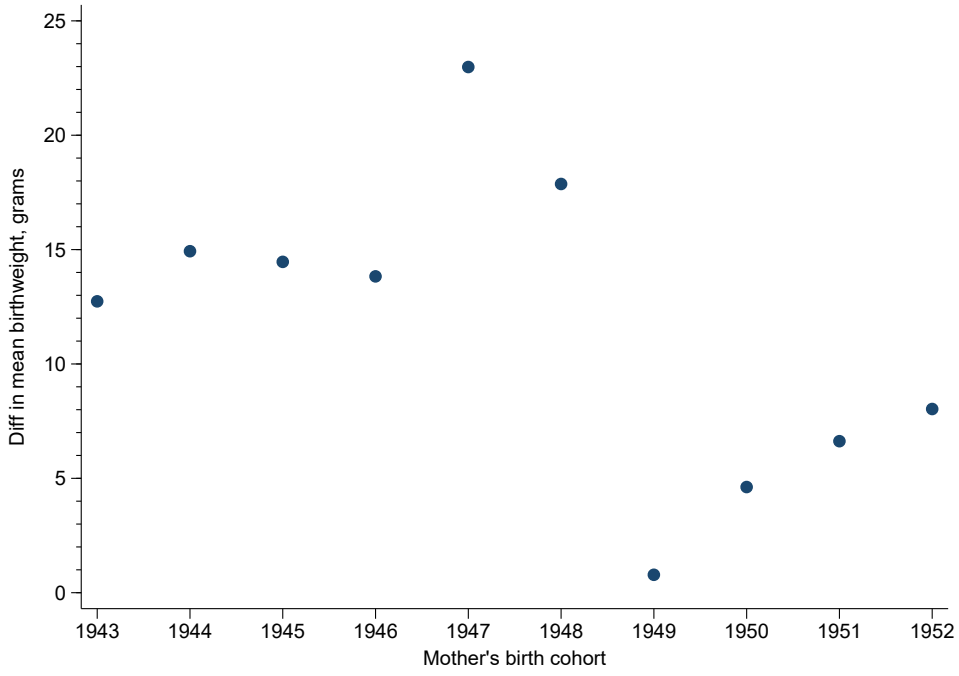


Figure 4: Example of identification by differencing — mean birth weight by mother's cohort

A. Mean birth weight by mother's age in 1968 and 1970



B. Difference between mean birth weight in 1970 and that of same mother's age in 1968



reveals cohort differences by 2-year gaps $\gamma_c - \gamma_{c-2}$, plus any age-by-year bias

Figure 5: Labor market outcomes

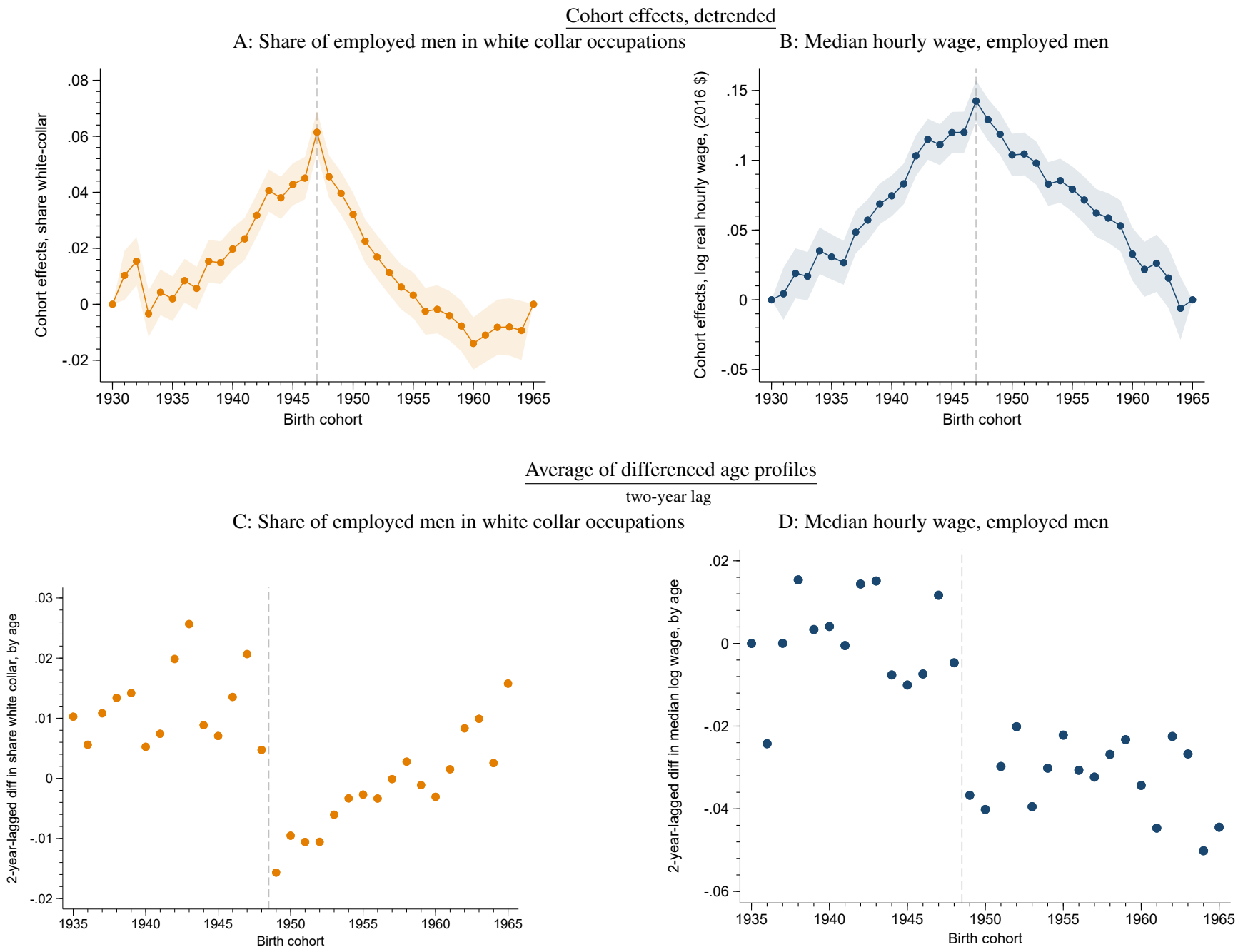


Figure 6: Maternal/integenerational health outcomes

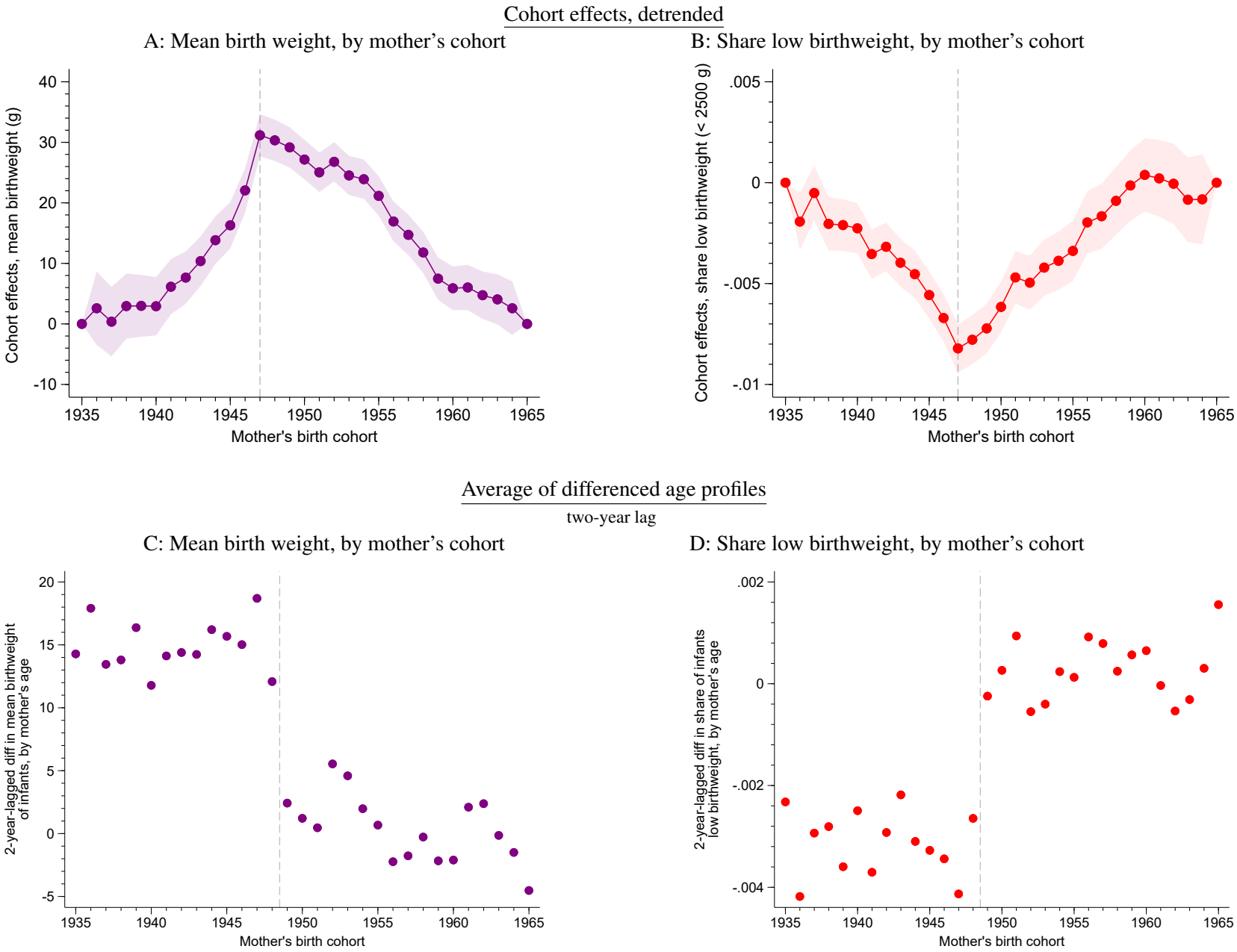


Figure 7: Log mortality

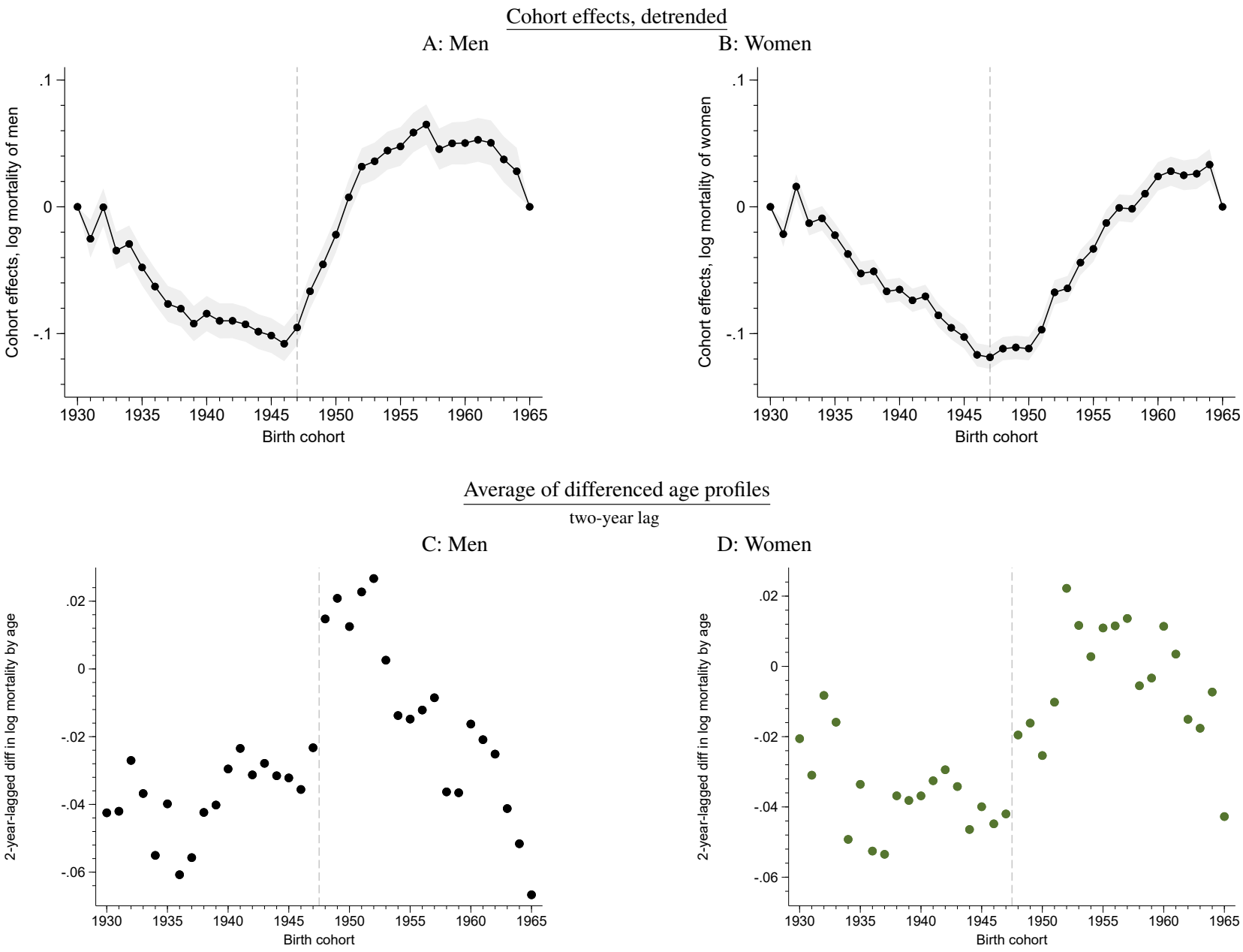


Figure 8: Estimated cohort effects — mean birth weight of infant by mother's cohort // mothers born in United States versus foreign-born mothers

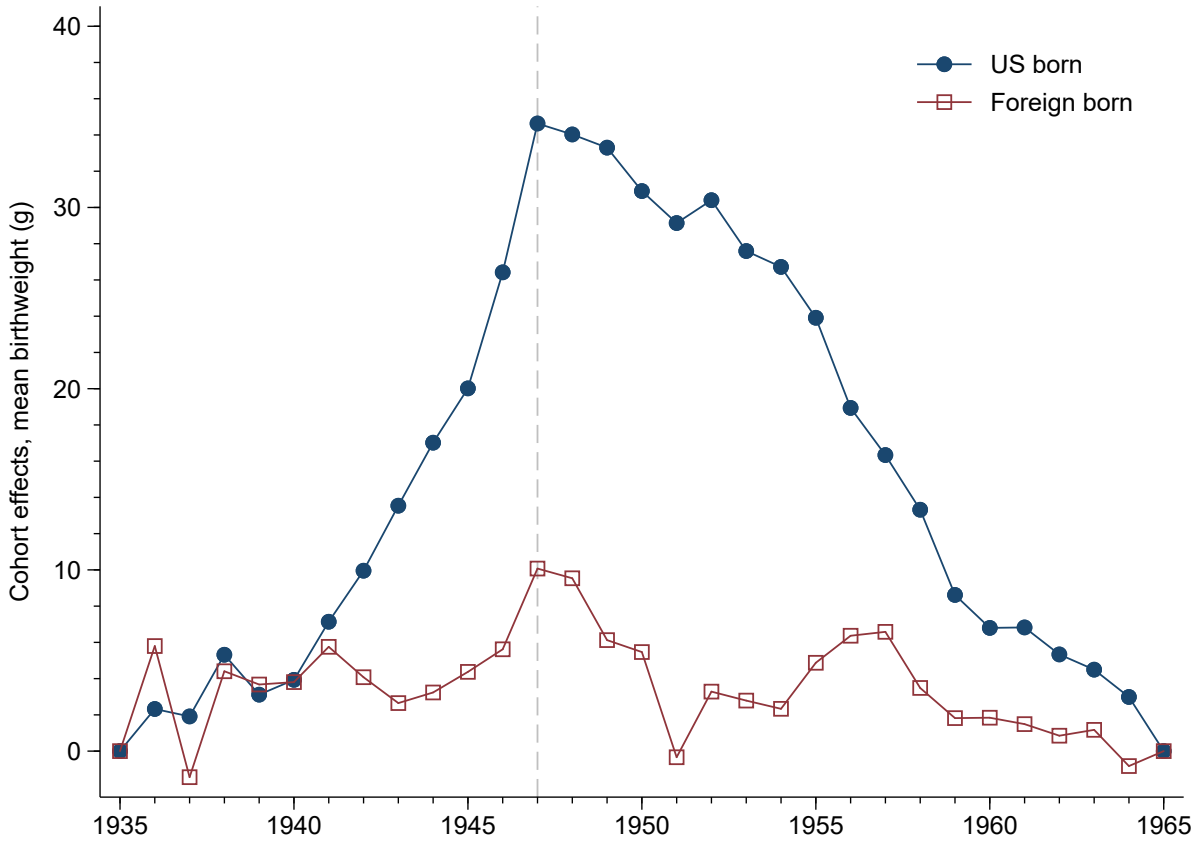


Figure 9: Mean birth weight of infant by mother's cohort // by mothers years of education

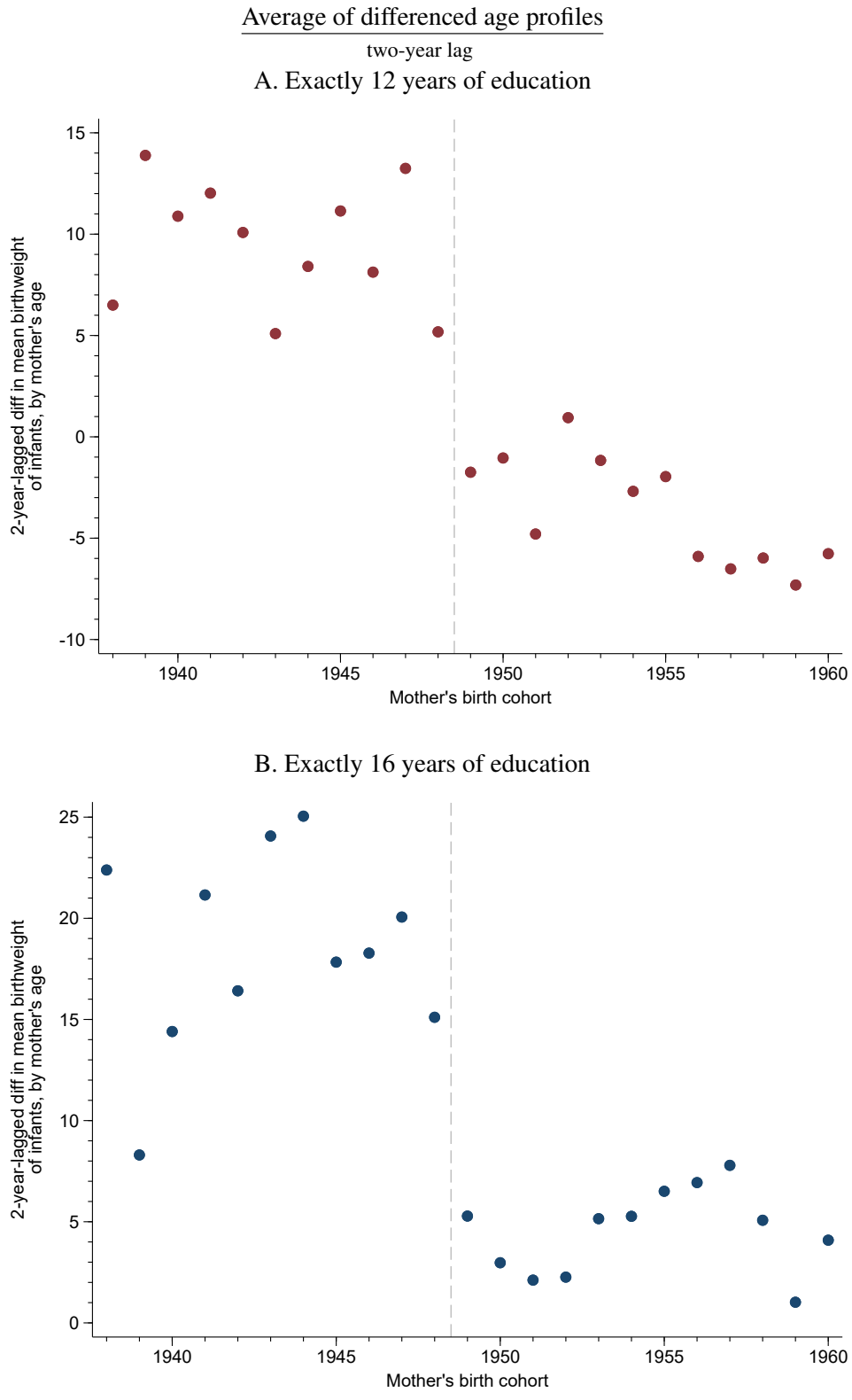
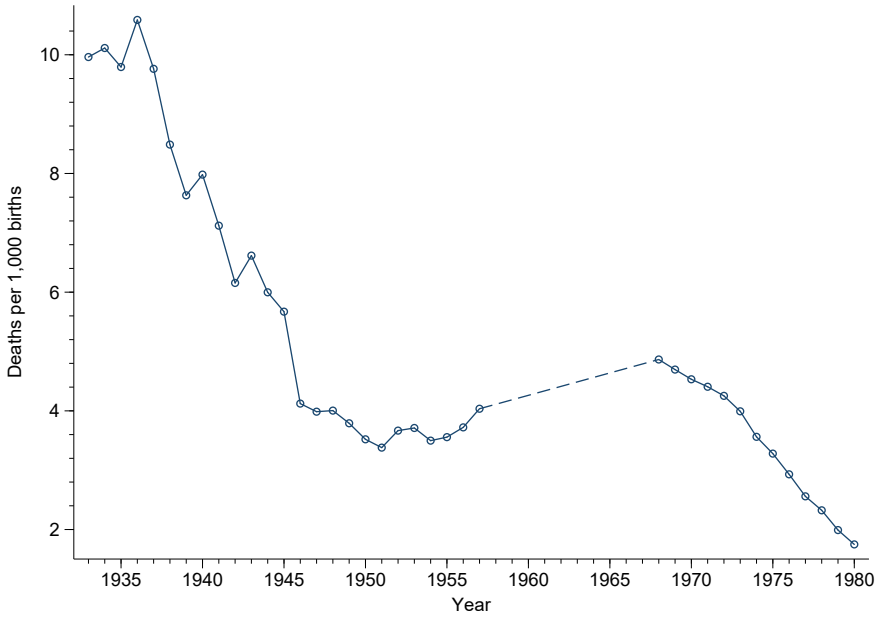


Figure 10: Post-war increase in respiratory mortality,
Infants

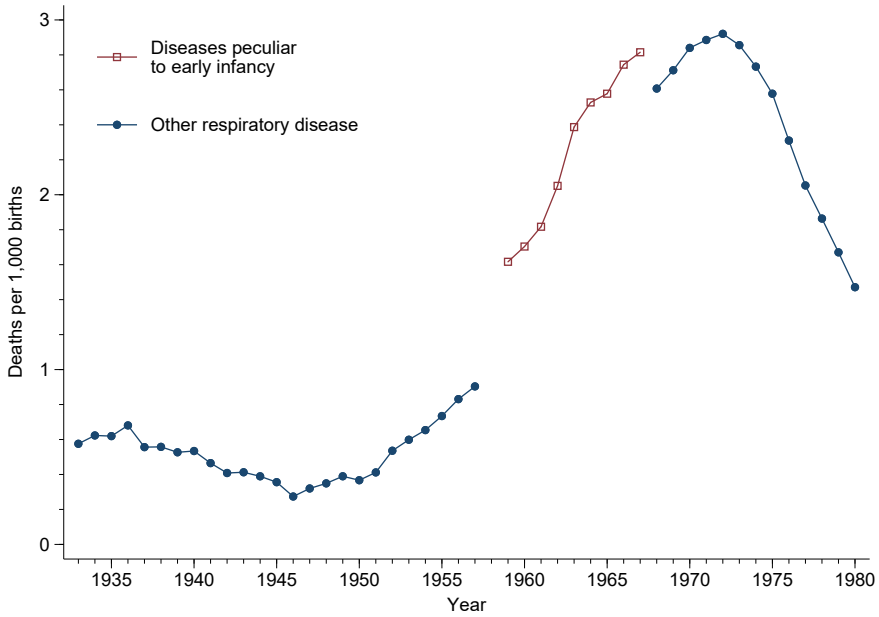
A: Infant mortality rate, all respiratory diseases

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



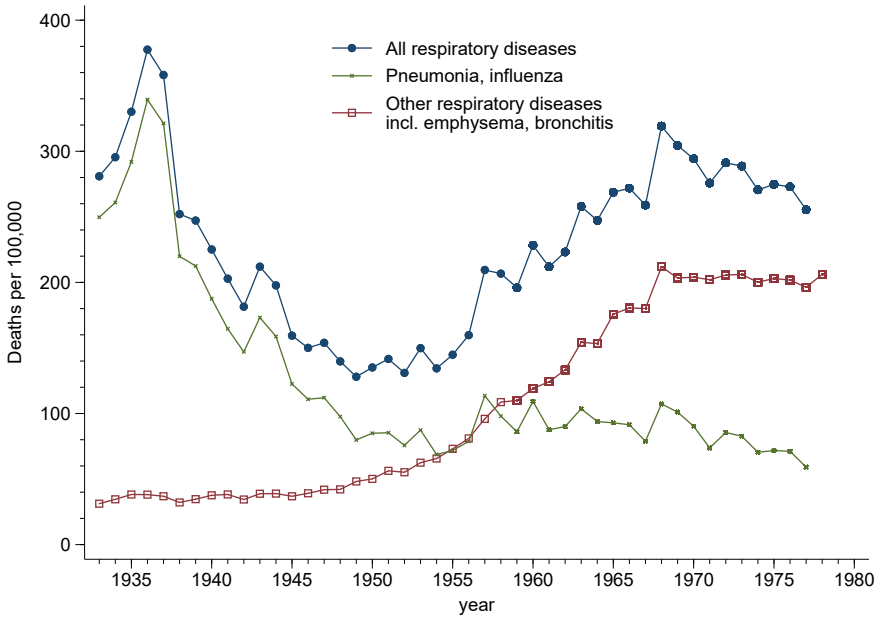
B: Infant mortality rate

cause of death categories which include IRDS



Adult white men

C: Respiratory mortality, age 65-69



D: Log mortality — respiratory diseases excluding pneumonia and influenza

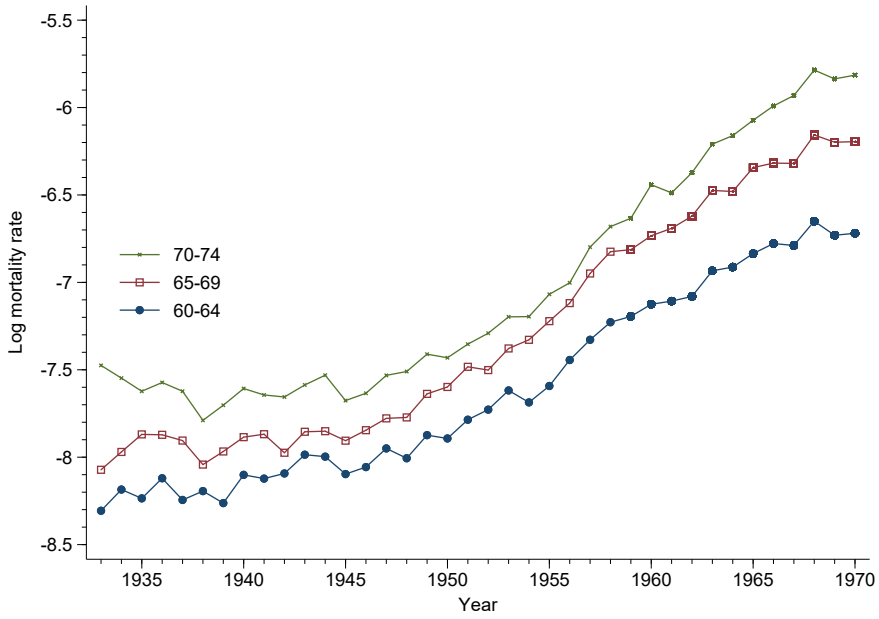


Table 1: Change in slope of cohort effects for 6 main outcomes

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

	Change in cohort slope		
	Size	Location	Existence
	δ	λ	*p-value*
Labor market			
Share white collar	-0.017	1947	$< .001$
	(.001)	[1947, 1947]	
Median log wage	-0.016	1947	$< .001$
	(0.001)	[1946, 1947]	
Intergenerational infant health			
Mean birth weight (g)	-6.892	1948	$< .001$
	(0.356)	[1948, 1948]	
Share low birthweight	0.0023	1948	$< .001$
	(0.0001)	[1948, 1948]	
Log mortality			
Men	0.029	1946	$< .001$
	(.001)	[1946, 1946]	
Women	0.031	1949	$< .001$
	(.001)	[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). 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 2: Mean shift in differenced age profiles by birth cohort, 1 year lag

controlling for quadratic in birth cohort

	Mean shift		
	Size	Location	Existence
	δ	λ	*p-value*
Labor market			
Share white collar	-0.023	1948	$< .001$
	(.003)	[1948, 1948]	
Median log wage	-0.021	1948	$< .001$
	(0.005)	[1948, 1948]	
Intergenerational infant health			
Mean birth weight (g)	-7.499	1948	$< .001$
	(1.187)	$[1948, 1948]$	
Share low birthweight	0.0019	1948	$< .001$
	(0.0004)	[1948, 1948]	
Log mortality			
Men	0.029	1947	$< .001$
	(.003)	[1947, 1947]	
Women	0.028	1951	$< .001$
	(.003)	[1951, 1951]	

Each column shows the results of estimation of a model based on Equation 16, with the listed outcome as a dependent variable. 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.

Table 3: Change in cohort slope — by race

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]
Share low birthweight	*Size*	0.0010	0.0008		0.0007
		(0.00005)	(0.0001)		(0.0003)
	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). 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: Change in cohort slope — by Census Region

		(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]
Share low birthweight	*Size*	0.0012	0.0011	0.0013	0.0009
		(.0001)	(.0001)	(.0001)	(.0001)
	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). EDIT: Wages and birth weight include controls for age fixed effects and year fixed effects — mortality also includes quadratic in Age. 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 5: Change in slope of cohort effects for intergenerational infant birth weight — native-born versus foreign-born mothers

robustness to varying age-by-year control function

	(1)	(2)	(3)	(4)
Panel A: Mother's 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: Mother's 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). SAMPLES 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 6: Implied causal effect of schooling on earnings and maternal health

assuming no change in unobservables across cohorts

		Change in cohort slope	
	Outcome	Years of schooling	Implied causal effect
	(reduced-form)	*(first stage)*	*(2sls)*
Unconditional			
Median log wage	-0.016	-0.115	0.138
	(0.0004)	(0.003)	(0.004)
Share low birthweight	0.0016	-0.173	-0.0092
	(0.0002)	(.027)	(.0009)
Male log mortality	.031	-.104	-.251
	(0.001)	(.002)	
Female log mortality	.026	-.072	-.434
	(0.001)	(.001)	
Conditional on no Bachelor's degree			
Median log wage	-0.0709	-0.013	0.187
	(0.0005)	(0.002)	(0.008)

EDIT: 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). 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 7: 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). SAMPLES 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 8: Change in slope of cohort effects for intergenerational infant birth weight
By Maternal Education

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 column shows the results of estimation of a model based on equation4, with the listed outcome as a dependent variable. 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.

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Figure A1: Log mortality rate of white men

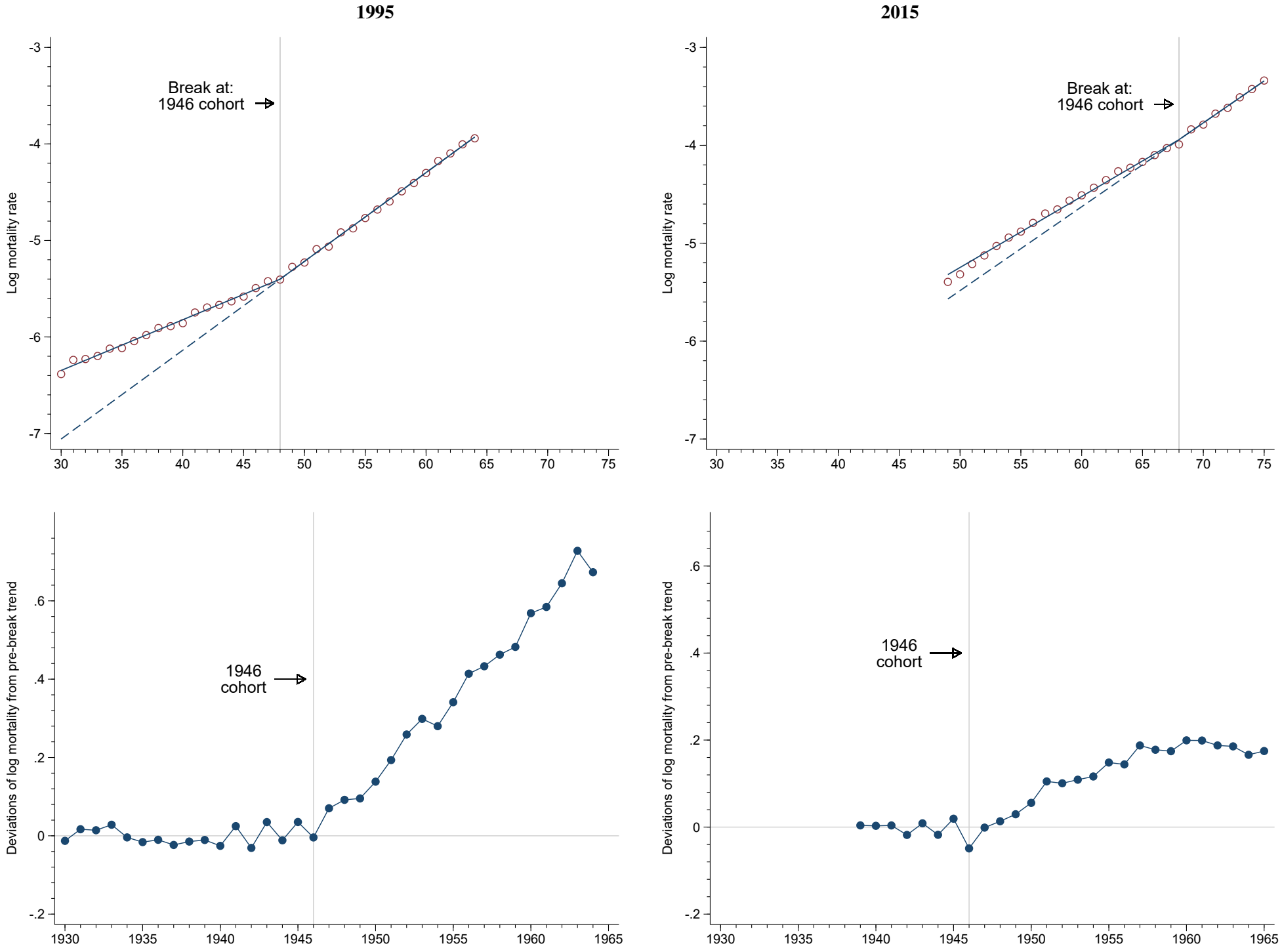


Figure A2: Log mortality rate of white women

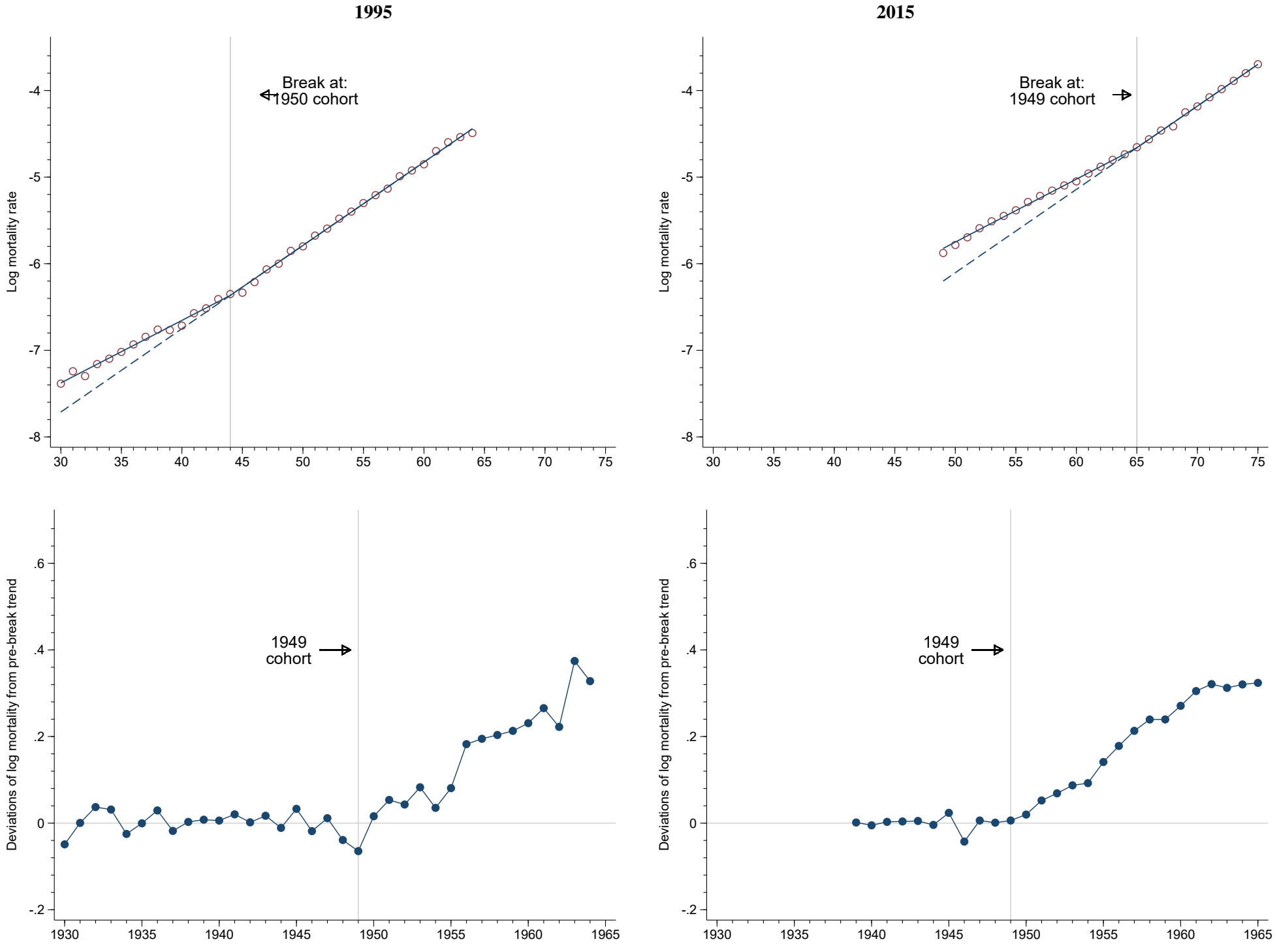
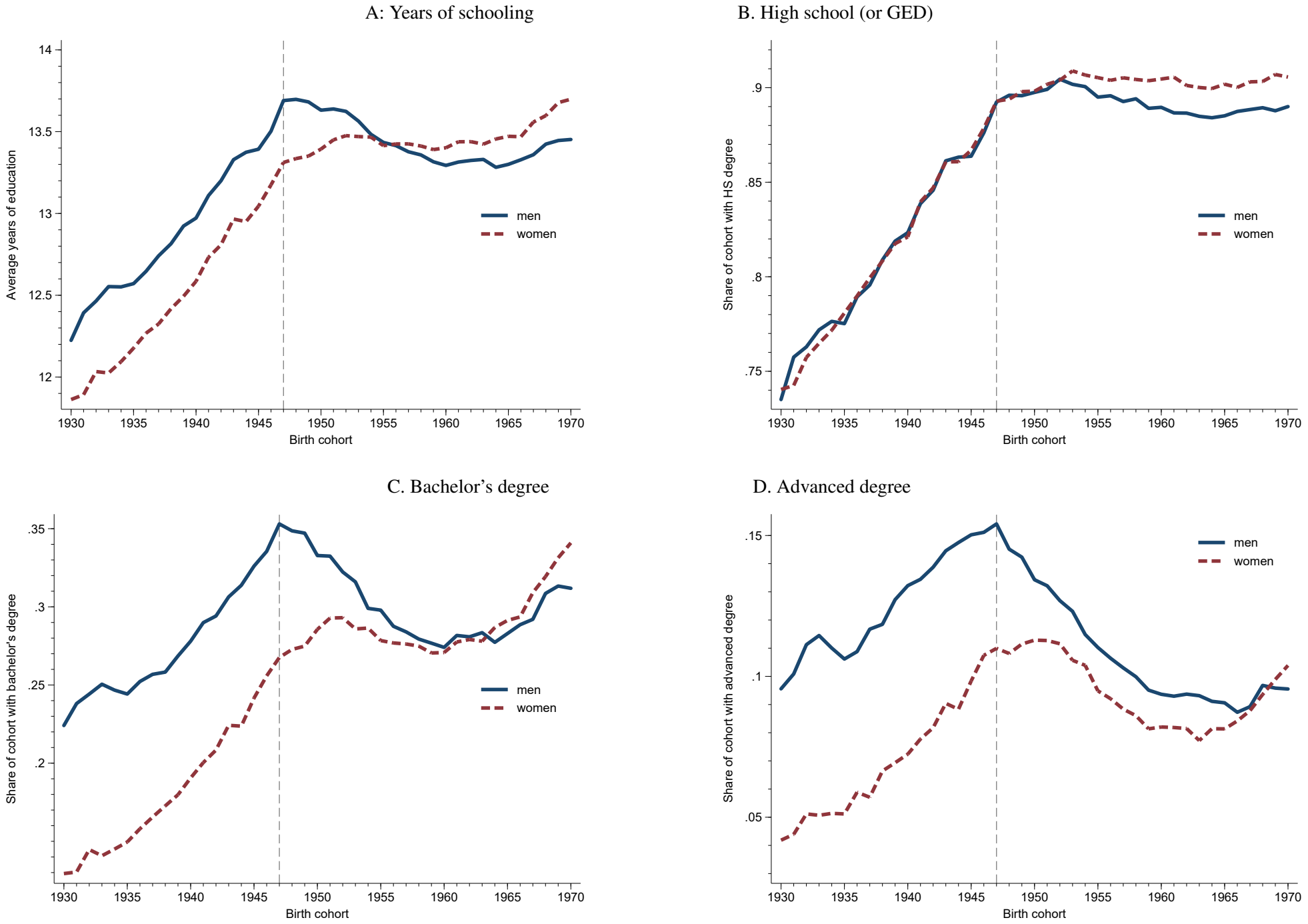
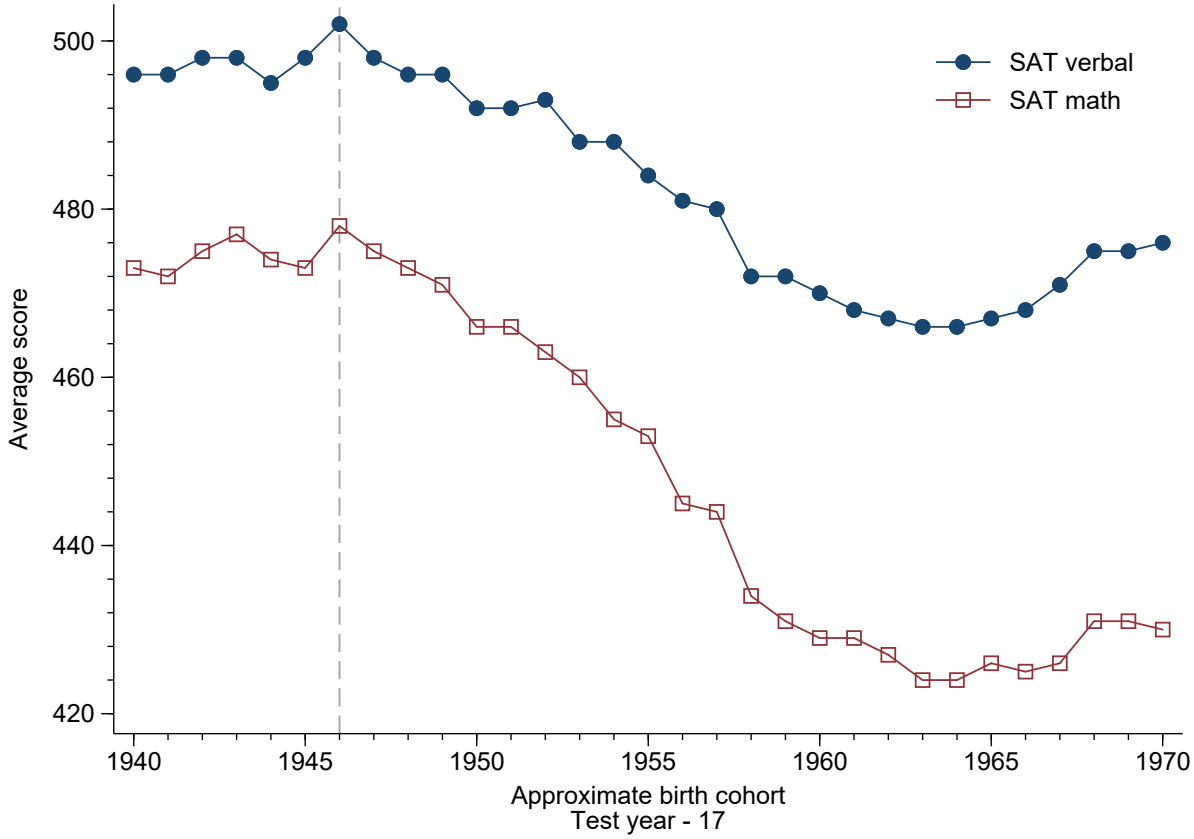


Figure A3: Declines in educational attainment



Data is from CPS Merged Outgoing Rotation Group and includes men and women aged 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.

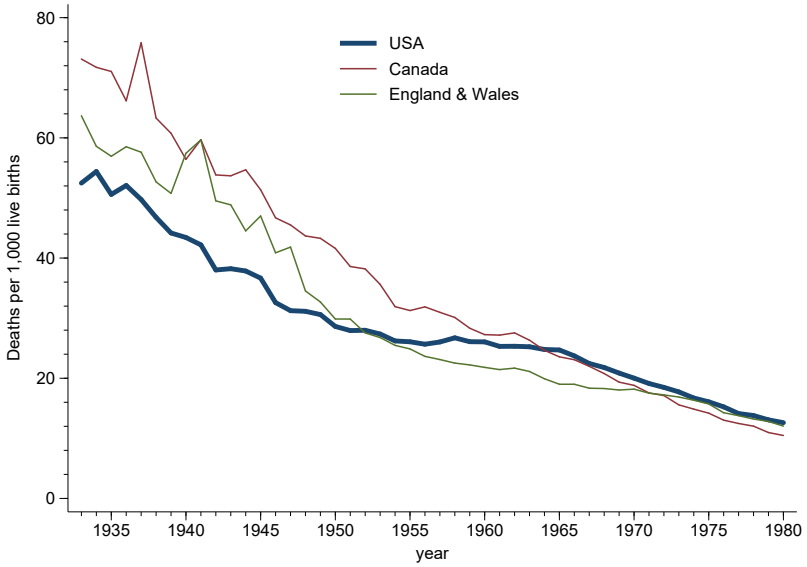
Figure A4: Declines in Scholastic Aptitude Test (SAT) Scores



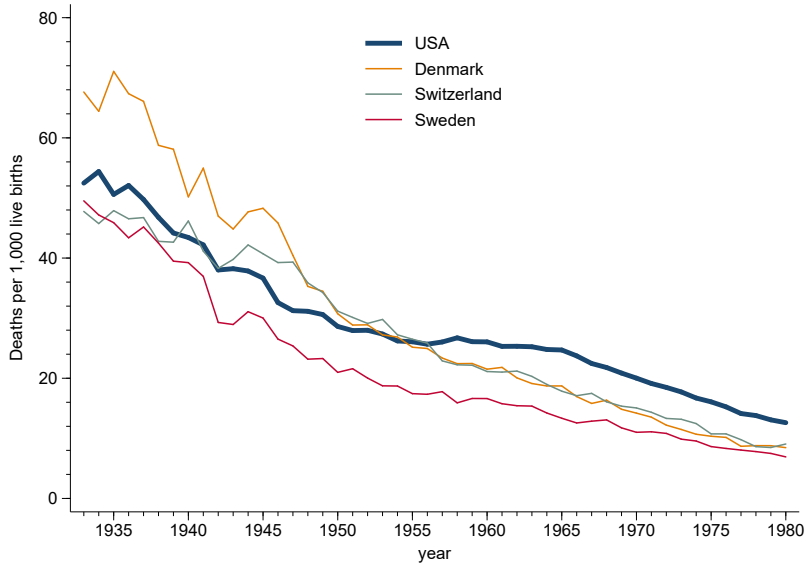
This figure shows verbal and mathematics scores on the Scholastic Aptitude Test. Approximate birth cohort is defined as the year the test was given minus 17. Data is from Harnischfeger and Wiley (1975).

Figure A5: 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

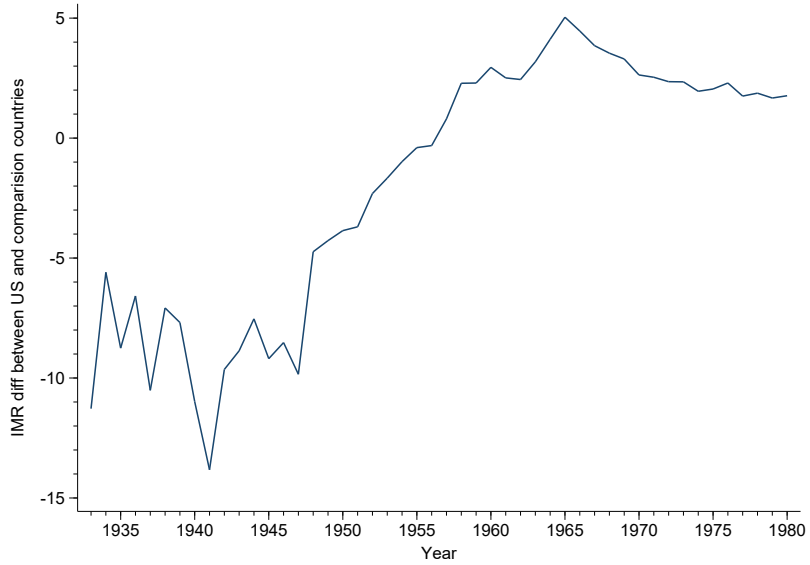
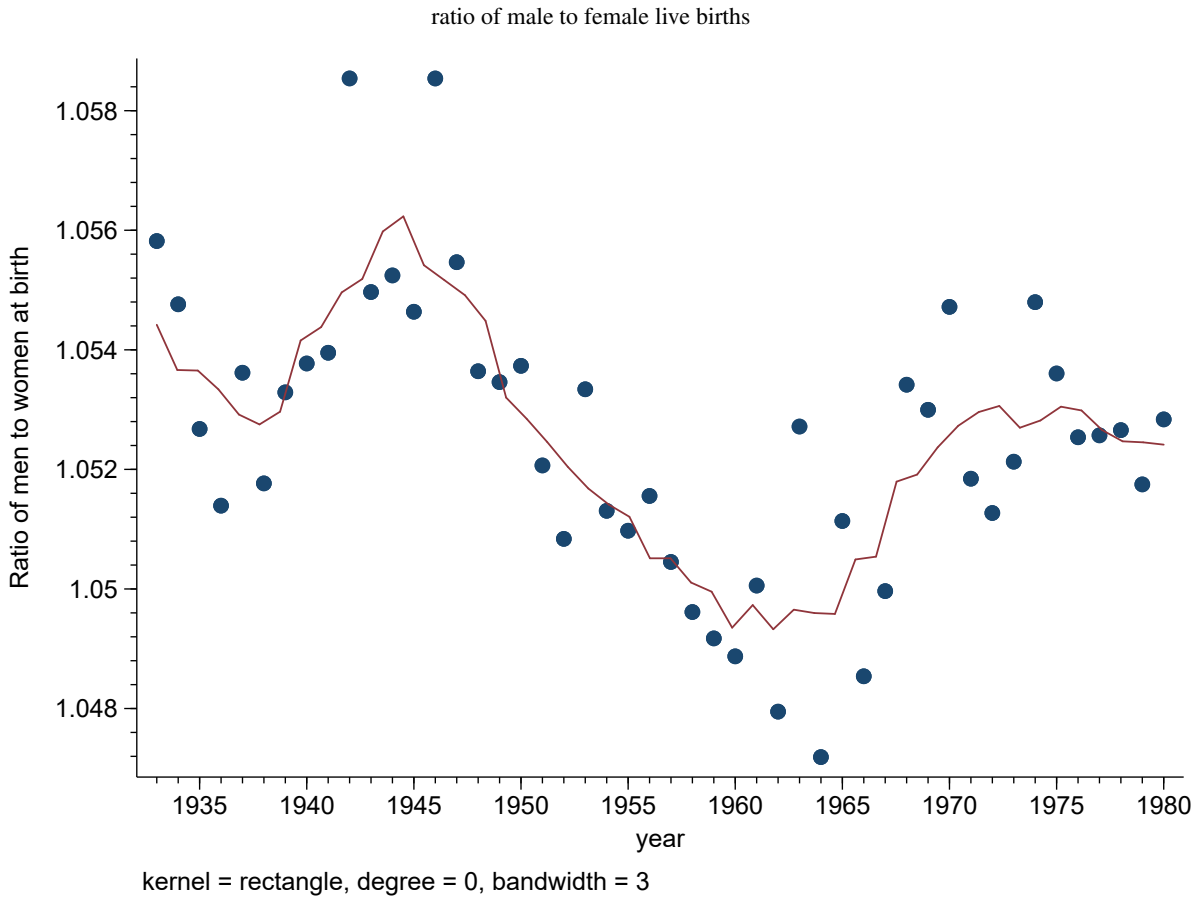


Figure A6: Sex ratio at birth in the United States



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

Table A1: Change in slope of cohort effects for men's labor market outcomes

robustness to varying age-by-year control function

	(1)	(2)	(3)	(4)
Panel A: Share white collar				
Size	-0.007	-0.017	-0.015	-0.015
	(0.000)	(0.001)	(0.002)	(0.002)
Location	1947	1947	1947	1947
	[1946, 1947]	[1947, 1947]	[1947, 1947]	[1947, 1947]
P-value for existence	$< .001$	$< .001$	$< .001$	$< .001$
Panel B: Median log wage				
Size	-0.016	-0.016	0.015	-0.017
	(0.000)	(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	$< .001$	$< .001$	$< .001$	$< .001$
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). SAMPLES 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 intergenerational infant health outcomes

robustness to varying age-by-year control function

	(1)	(2)	(3)	(4)
Panel A: Mean birth weight (g)				
Size	-4.212	-6.892	-5.597	-6.629
	(0.139)	(0.356)	(0.524)	(0.646)
Location	1949	1948	1947	1947
	[1949, 1950]	[1948, 1948]	[1947, 1947]	[1947, 1947]
P-value for existence	$< .001$	$< .001$	$< .001$	$< .001$
Panel B: Share low birthweight				
Size	0.0012	0.0023	0.0020	0.0019
	0.00004	0.0001	0.0002	0.0003
Location	1948	1948	1947	1947
	[1947, 1948]	[1948, 1948]	[1947, 1948]	[1947, 1947]
P-value for existence	$< .001$	$< .001$	$< .001$	$< .001$
Year FEs	Yes	Yes	Yes	Yes
Age FEs	Yes	Yes	Yes	Yes
Quadratic-age-by-year	No	Yes	No	No
Cubic-age-by-year	No	No	Yes	No
Quartic-age-by-year	No	No	No	Yes

Each column shows the results of estimation of a model based on equation 4, with the listed outcome in single mother's-age-by-year bins as the dependent variable. All models are estimated by least squares, following the approach outlined in Hansen (2000). SAMPLES 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 log mortality

robustness to varying age-by-year control function

	(1)	(2)	(3)	(4)
Panel A: Men				
Size	.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 B: Women				
Size	0.017	0.031	0.018	-0.023
	0.00004	0.0001	0.0002	0.0003
Location	1947	1949	1950	1942
	[1947, 1947]	[1949, 1949]	CHECK [1942, 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 mother's-age-by-year bins as the dependent variable. All models are estimated by least squares, following the approach outlined in Hansen (2000). SAMPLES The row titled "Size" reports the size of the change in slope of cohort effects, δ, with the standard error in parentheses. The row titled "Location" reports the estimated cohort at which a trend break occurs, with a 99 % confidence region in brackets calculated by inverting the likelihood ratio statistic. The row titled "P-value for existence" reports a p-value from an F-type test, based on 1000 bootstrap samples, for the null hypothesis that no change in slope occurs, ie. that cohort effects are linear.

Table A4: Change in mean of differenced age profiles, 2 year lag

controlling for quadratic in birth cohort

	Mean shift		
	Size	Location	Existence
	δ	λ	*p-value*
Labor market			
Share white collar	-0.037	1948	$< .001$
	(.004)	[1948, 1949]	
Median log wage	-0.033	1949	$< .001$
	(0.006)	[1948, 1949]	
Intergenerational infant health			
Mean birth weight (g)	-12.173	1949	$< .001$
	(1.648)	$[1949, 1949]$	
Share low birthweight	0.0032	1949	$< .001$
	(0.0005)	[1949, 1949]	
Log mortality			
Men	0.054	1948	$< .001$
	(.005)	[1948, 1948]	
Women	0.048	1951	$< .001$
	(.004)	[1951, 1951]	

Each column shows the results of estimation of a model based on Equation 16, with the listed outcome as a dependent variable. 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.

Table A5: 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

NOTE: USES 25 to 54 YEAR OLDS 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). SAMPLES 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 A6: Change in slope of cohort effects for mean birth weight of infants by mother's birth cohort — separately for mother's 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). SAMPLES 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 A7: 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 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). 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.