Three Regimes of Human Development in Twentieth-Century America: Biological Growth, Health, and Human Capital

Nicholas Reynolds

University of Essex *

March 26, 2026

Abstract

This paper shows that the rise, decline, and uneven recovery of health and human capital across twentieth-century U.S. cohorts were mirrored by parallel changes in biological growth and maturation. In the first half of the twentieth century, remarkable gains in longevity and human capital were mirrored by large increases in height and earlier maturation. For cohorts born near mid-century, progress in health and human capital came to a sudden stop and reversed, with many measures declining through 1960s cohorts. For these same cohorts, gains in childhood and adult height stalled, adolescent growth declined, and puberty was delayed. For cohorts born after the 1960s, some measures of human capital rebounded modestly but did not return to earlier trends, while the growth process changed yet again, in ways linked to rising childhood obesity. I provide micro-level evidence supporting the interpretation of biological growth as a proxy for early-life conditions. This suggests an essential role for differences in early-life conditions in driving twentieth-century trends in health and human capital.

*Nicholas.Reynolds@essex.ac.uk. Thanks to Tim Hatton, Eric Schneider, Hampton Gaddy, and participants at the LSE Historical Economic Demography Working Group for helpful comments.

1 Introduction

Health and human capital improved enormously across American cohorts born in the first half of the twentieth century (e.g., Goldin and Katz, 2007; Cutler et al., 2006). For cohorts born after mid-century, however, progress suddenly stalled. Educational attainment declined for cohorts born between 1947 and the mid-1960s (Card and Lemieux, 2001b; Heckman and LaFontaine, 2010). In Reynolds (2025), I argue this is just one symptom of a broad decline in health and human capital, which reduced income and elevated mortality.

This paper shows that these cohort trends are closely mirrored by parallel changes in biological growth and maturation. Height gains stalled and puberty was delayed for the same cohorts whose health and human capital deteriorated, pointing to substantial changes in early-life environments and investments (e.g. Tanner, 1990; Schneider, 2023). I support the interpretation of biological growth as a proxy for early-life conditions with micro-level, panel evidence. The findings suggest that the rise, fall, and subsequent stagnation in health and human capital over the twentieth century are rooted in differences in early-life capabilities which have persisted through the lifecycle.

I document three distinct regimes in physical growth and maturation across twentieth-century U.S. cohorts. Figure 1 summarizes these patterns, combining historical anthropometric evidence with modern survey data to trace changes in height and development from childhood through adulthood. The figure shows large and sustained gains in height and maturation for cohorts born in the first half of the century, an abrupt break for cohorts born near mid-century, and a further shift in the pattern of growth for cohorts born later.

For cohorts born in the early twentieth century, physical development followed the well-known secular trend: childhood and adult height increased steadily, and adolescent growth accelerated, consistent with earlier maturation. Anthropometric historians have long interpreted this pattern as evidence of major improvements in early-life conditions, including reductions in disease burden and improvements in nutrition (e.g. Fogel, 2012; Floud et al., 2011). These gains occurred for the same cohorts that experienced large improvements in longevity and educational attainment.

For cohorts born after the mid-twentieth century, this progress stopped abruptly. Gains in childhood and adult height stalled, but the changes were most pronounced in adolescence: boys in these cohorts were shorter during the adolescent years, and among girls age at menarche rose, consistent with a delay in puberty and a slowing of the tempo of maturation. Strikingly, these cohorts for whom growth and development stalled and on some dimensions reversed, are the same for whom later in life test scores, educational attainment, and mortality stalled or reversed.

For cohorts born in the 1970s and later, the growth process changed again. Puberty occurred earlier and childhood growth patterns shifted, a development widely linked to the rise in childhood obesity and the emergence of an obesogenic environment. This was not a return to the earlier

secular trend, however, as adult height did not resume sustained growth. Educational attainment rebounded somewhat for these cohorts, but the full health and longevity consequences of this new pattern of biological development will only become clear as these cohorts age.

These patterns suggest an essential role for differences in early-life conditions in driving twentieth-century trends in health and human capital. I develop a simple conceptual framework, in the spirit of Cunha and Heckman (Cunha and Heckman, 2007), in which physical growth and maturation evolve alongside the formation of health, skills, and capabilities over the life cycle, and in which growth serves as a proxy for the early-life environments and investments that shape biological development. In this framework, early-life differences in capabilities—proxied in part by differences in the growth process—alter the trajectory of health and human capital over the life cycle through a dynamic process of development and investment.

I provide micro-level evidence consistent with this interpretation. Using panel data from the National Health Examination Survey, following children in mid-twentieth-century cohorts and linking height and test scores at two ages, I show that socioeconomic status predicts physical growth and maturation, and that greater growth and earlier maturation are associated with larger gains in test scores.

I present a range of additional evidence indicating that the cohort patterns reflect real changes in biological development driven by changes in early-life environments, rather than changes in measurement, selection, or genetic composition. The trends in adult height, based in part on historical anthropometric data, are independently corroborated in modern survey data, which helps address concerns about selection into military samples (Bodenhorn et al., 2017). Independent evidence on the timing of menarche (the age when females have their first menstrual period) exhibits a similar cross-cohort pattern in maturation to that implied by male adolescent height. In contrast, polygenic scores associated with height and menarche evolve smoothly across cohorts and show no corresponding break, suggesting that the observed changes in growth and maturation are driven primarily by environmental rather than genetic changes.[1]

This paper integrates ideas from several literatures: the use of height as a proxy for early-life health and nutrition, work on the role of early-life health in later-life human capital, and studies of long-run trends in educational attainment and health. Robert Fogel and others in the anthropometric history tradition interpret adult height as a proxy for early-life health and conditions and use the long-run "secular trend" in height to argue for a central role of early-life conditions in later-life health improvements (Fogel, 2012; Floud et al., 2011; Costa, 2015).[2] A large empirical and theoretical literature studies the role of early-life health conditions in shaping later-life health and human capital (e.g. Almond et al., 2005; Cunha and Heckman, 2007; Currie and Almond, 2011;

[1] Results available from the author. To be added to future drafts.

[2] See also Fogel (1986); Costa and Steckel (1997).

Chay et al., 2009). There is also a literature linking physical development and adult labor-market outcomes.

This paper applies insights from these micro-level and historical literatures to better understand aggregate trends in health and human capital across twentieth-century American cohorts. In particular, I contribute to the question of why the rapid rise in educational attainment stopped for cohorts born after mid-century (Goldin and Katz, 2007; Card and Lemieux, 2001b; Heckman and LaFontaine, 2010; Acemoglu and Autor, 2011), and why health and human capital more broadly appears to have declined for cohorts born after 1947 (Reynolds, 2025), with implications for understanding the United States' mortality disadvantage (Case and Deaton, 2015, 2017; National Academies of Sciences, Engineering, and Medicine, 2021). I also show that examining the entire process of human growth and development provides information beyond adult height alone,[3] and provide a conceptual framework in the spirit of Cunha and Heckman (2007), formalizing the use of growth and development information as proxies for early-life conditions and investments.

2 Data

A Height

To document trends in height in childhood, adolescence, and adulthood I combine data from numerous rounds of the National Health and Nutrition Examination Survey (NHANES) and its predecessor the National Health and Examination Surveys (NHES). I supplement the NHES and NHANES surveys with earlier historical estimates of childhood height compiled in Meredith (1964) and adult height series from (Bleakley et al., 2014).

The NHES and NHANES surveys are cross-sectional and were designed to be representative of the US population at the time of the survey (or of select age groups).[4] I combine data from the first, second and third National Health Examination Surveys (NHES I, II, and III), and numerous rounds of the National Health and Nutrition Examination Survey (NHANES I to III, and the "continuous NHANES" from 1999 to 2023). However, the samples are relatively small, and the early surveys occurred irregularly and each survey focused on a different age group. However, (by lucky coincidence) it is possible to observe the heights of the cohorts born between 1951-1957 and 1961-1967 at multiple ages from 5 to adulthood, and therefore compare their growth patterns.

Panel A of Figure A1 shows the childhood component of this sampling in a Lexis diagram. The NHES II was carried out in 1963-1965 and only sampled 6-11 year olds, and NHES III was carried out in 1966-1970 and sampled 12-17 year olds. This allows me to observe the 1951-1957 cohorts once between the ages of 6 and 11, and again between the ages of 12 and 17. The NHANES

[3] See Tanner (1990); Schneider (2023).

[4] Some respondents were interviewed in both NHES II and III, but the panel created from this sampling yields a very small sample and few cohorts.

I and NHANES II sampled ages 1 (or zero) to 74 and were carried out in 1971-74 and 1976-80. This allows me to observe some members of the 1951-1957 cohort a third time as teenagers. More importantly, it allows me to observe the 1961-1967 cohort twice, at similar ages to the earlier-born cohort.

I then use the NHANES I to III, and the Continuous NHANES from 1999 to 2023 to observe adult heights for both the 1951-57 and 1961-67 cohorts. I follow Komlos (2010) and focus on adults aged 23 to 40, ages at which height is approximately constant. I use sampling weights in all analyses and focus on individuals born in the US. For the NHES surveys this is defined based on a linkage to birth certificates and for the later surveys is self-reported.

Even when pooling data in this way, the sample sizes are not enormous but prove large enough to detect differences in adolescent height between the two groups. For the 1951-1957 birth cohorts, there are approximately 3000 observations of males with non-missing height at ages 6 to 11, a similar number at ages 12-17, and approximately 1300 observations in adulthood. For the 1961-1967 birth cohorts, there are only around 1200 and 900 observations at age 6-11 and 12-17, respectively, and 1200 in adulthood. See Appendix Table A1.

In Section 3, I extend the analysis to later birth cohorts, again using the NHANES I to III, and the Continuous NHANES from 1999 to 2023. Panel B of Figure A1 shows the extended sampling scheme in a lexis diagram. Appendix Table A1 shows the sample sizes for these cohorts as well, which range from approximately 500 to 2500 per age-group-birth-cohort cell.

Year of birth is not directly recorded in some of the later surveys; therefore, I impute it with some error based on age and the (approximate) year of the survey. For NHANES III, I define year of birth as 1990 minus age for those surveyed in Phase 1 (October 18, 1988, through October 24, 1991) and 1993 minus age for those surveyed in Phase 2 (September 20, 1991, through October 15, 1994). The continuous NHANES is organized into two-year waves, and the exact year of the survey within the wave is not recorded. For these surveys, I define year of birth as the later of the two survey years minus age. Native-born is not reported in NHES I; therefore, I include all individuals.

To my knowledge, the NHES II was the first nationally representative study which measured childhood height. Meredith (1964) compiled results from a large number of historical studies of childhood height. Although they do not come from nationally representative samples, the studies did not explicitly target children with high or low socioeconomic status. He was also focused on trends for "North American boys" — so while his estimates are primarily based on American samples, they do include some Canadian studies. I use the series he constructs based on these studies of the height of white boys aged 10 and 15.[5] The childhood height series for these cohorts has

[5]Specifically, I use the mean heights reported in the left panels of Table XII and XIII of Meredith (1964). I calculate the midpoint of each time period, then subtract the relevant age to calculate the midpoint of the birth cohort group which is represented.

more potential to be biased; for example, if children in school were more likely to be sampled, the selection bias may worsen in adolescence. This age-varying selection bias could also conceivably change over time as school enrollment rates change (Schneider, 2020).

The adult series is based on various military samples and comes from Costa (2015) and Bleakley et al. (2014), which updates Costa and Steckel (1997) and Fogel (1986).

For comparability with the historical sources, I initially restrict the NHES/NHANES data to white non-Hispanic native males. In additional analysis, I show trends in the second half of the twentieth century for the entire native born population for both men and women, and separately for non-Hispanic whites and blacks.[6]

An important finding of the paper is a turning point or "regime change" near mid-century. It is therefore potentially concerning that the change in data sources also occurs near mid-century. For adult height at least I am able to cross-validate the historical series by presenting trends in adult height constructed entirely from the NHES and NHANES data, going back to the 1920s cohorts. Again, for this analysis I am able to examine height trends by race and sex as well.

B Menarche

The retrospective menarche series pools data on self-reported age at menarche from NHES II–III, NHANES I–III, and Continuous NHANES 1999–2023 for native-born women aged 20–75 at the time of the survey. I also construct contemporaneous menarche measures from observations of girls aged 10–17 in NHES and NHANES, using interval-censoring methods to account for the fact that not all girls have experienced menarche at the time of the survey.

C Educational attainment

Educational attainment outcomes are constructed from pooled Decennial Census and American Community Survey microdata obtained through IPUMS (Ruggles et al., 2015). I focus on native-born males and females aged 35–55 at the time of the survey and examine mean years of schooling, high school completion rates, and bachelor's degree attainment by birth cohort. I supplement these with administrative high school graduation rates from the NCES Digest of Education Statistics.

D Mortality

Mortality outcomes are derived from Human Mortality Database (HMD) cohort life tables for the United States. I compute the probability of dying between ages 25 and 60 for each birth cohort, using standard life-table methods. I exclude cohort-age cells affected by the Covid-19 pandemic.

[6] NHES-I unknown nativity is retained where applicable.

3 Long-Run Patterns of Physical Growth, Development, Human Capital, and Health

A Height and Growth

Figure 1 documents long-run trends in the height of Americans born in the twentieth century at three developmental stages: childhood, adolescence, and adulthood. It uses the archival data described above for the first half of the century and nationally representative survey data from the NHES and NHANES for the second half of the century. The figure focuses on white North American males born during the twentieth century in order to maintain comparability with the archival data. Examining height at three ages allows us to trace out the entire growth process and potentially identify changes in the timing of growth during adolescence. Figure 2 summarizes the patterns shown in Figure 1 by reporting the average change in height at each developmental stage across each of three regimes: Regime I, cohorts born 1900-1950; Regime II, cohorts born from the 1950s through 1960s; and Regime III, cohorts born after 1970.

Cohorts in Regime I, born roughly 1900 to 1950, experience steady increases in height at all observed ages. The pattern is consistent with the well-known secular trend in height observed across industrialized countries during the twentieth century. The increase in adolescent height exceeds the increases in both childhood and adult height. Figure 2 implies gains of about 0.62 inches per decade in childhood, 0.86 in adolescence, and 0.51 in adulthood. This is a hallmark of the twentieth-century secular growth trend, found across industrialized countries (e.g. Cole, 2003). This pattern suggests an acceleration in the timing or 'tempo' of development in addition to increase in final adult stature. Across cohorts, adolescents reach a larger share of their final adult height earlier in the life cycle, in addition to reaching a taller final adult height. In particular, this suggests these cohorts experienced a progressively earlier and/or faster adolescent growth spurt.

For cohorts born after roughly 1950, the upward trend in height slows abruptly. For those born from 1950 to the late 1960s, which I label Regime II, a new pattern of delayed growth occurs as well. While adult height had been growing at half an inch per decade for the prior 50 years, it slows to less than .1 inches between the 1950s and 1960s cohorts (and is statistically indistinguishable from zero). A similar stagnation occurs in childhood height, with point estimates suggesting childhood declined by roughly .05 inches per decade for these cohorts, compared to the growth of nearly .6 inches per decade preceding them. The most striking change, however, is in adolescent height which declines by roughly half an inch between the 1950s and 1960s cohorts. This pattern of adolescent height being more negative than both childhood and adult height, is in some sense a mirror of the pattern for Regime I. It suggests that the 1960s cohorts experienced a delay in the timing of growth, either through a delay in the timing of the adolescent growth spurt or a slowing of

the velocity of growth during the spurt. Therefore Regime II represents a cessation of the secular growth in childhood and adult height, and a reversal of the earlier trend towards earlier adolescent growth.

Some of these patterns had been noted previously. Komlos and Lauderdale (2007a,b) noted the stagnation in adult height, and highlight the lack of a similar trend in European countries. Surprisingly, the stagnation in childhood height was already noted in the 1970s. A National Center for Health Statistics report Hamill et al. (1977) described a "marked diminution and near cessation of the trend to constantly increasing size of successive generations of American children" and called it the "most dramatic and significant finding relating to human biology and human growth in general." However, the comparison across developmental stages for these cohorts, and therefore the realization that the growth spurt was likely delayed, has not to my knowledge been previously documented.

For cohorts born after roughly 1970, a new distinct regime emerges, which I label Regime III. For cohorts born in the 1970s and later, childhood and adolescent height increase substantially, by about 0.28 and 0.41 inches per decade, respectively. Adolescent height therefore more than rebounds from its temporary dip for 1960s cohorts. However, this is decidedly not a return to the secular trend of the first regime, as adult height is approximately flat (about -0.08 inches per decade and statistically indistinguishable from zero). Thus, individuals in these cohorts appear to be growing taller earlier in life, particularly in adolescence, but not ending up any taller as adults. This pattern implies an acceleration of growth tempo, with children and adolescents reaching higher shares of their final adult height earlier in the life cycle, but also a cessation of growth at an earlier age in young adulthood. Later sections discuss the possibility that these patterns may be tied to the rise in childhood obesity and the emergence of a "obesogenic environment" which has been linked to earlier puberty and accelerated growth tempo (e.g. Biro et al., 2006b; Kaplowitz, 2008; Kaplowitz et al., 2001).

Figure 3 provides additional evidence, and visual intuition, of the change in the timing of growth during Regime II. It plots smooth curves for individuals born between 1951–1957 and those born between 1961–1967. The two cohorts are nearly identical ages in childhood, but a difference in height emerges during adolescence, with the earlier cohort being about 0.45 inches taller on average at ages 12–17. The gap widens through the teenage years, suggesting a delay in the timing of the adolescent growth spurt for the later cohort. Unfortunately, these two cohorts are not sampled during their early 20s, and the sample sizes do not allow precise timing of exactly the ages where height diverges. However, by the time the cohorts reach adulthood, and likely final height (ages 23-40) they have converged to approximately the same height. Again, this pattern is consistent with a delay in the adolescent growth spurt followed by continued growth longer into early adulthood by the later cohort. In other words, individuals in the later cohort appear to experience their adolescent

growth spurt later but continue growing for longer.

I estimate regression models to assess the statistical significance of the height differences shown in Figure 3. For statistical power I pool ages into larger groupings, while flexibly controlling for more detailed age. In particular, I estimate models of the following form:

$$Y_{i,ac} = \gamma_{c=1961\text{-}67} + \mu + f(a) + \epsilon_{i,ac} \tag{1}$$

where $Y_{i,ac}$ denotes the height of individual i at age a in cohort c. I run the above regression separately for different age groups: 6–11, 12–17, and 23–40. In each regression $\hat{\gamma}_{c=1961\text{-}67}$ estimates the height differences between cohorts, and $f(a)$ controls for small differences in the distribution of ages when the cohorts are observed in the different surveys. For ages 6 to 11 and 12 to 17, I include dummies for age in years and a quadratic in age in months. I restrict to the native-born population and use sampling weights in all analyses.

Figure 4, panel (a) shows the estimated differences in height, $\hat{\gamma}_{c=1961\text{-}67}$, between native-born males in the 1951-1957 and the 1961-1967 cohorts, for each of the three age groups. The results suggest that the two cohort groups had nearly identical average heights as children at ages 6 to 11. The point estimate would imply that the average height of the 1960s cohort was less than a tenth of an inch below that of the earlier-born cohort at these ages. The difference is statistically indistinguishable from zero, and the 95 percent confidence intervals would rule out differences larger than a quarter inch in either direction.

However, the point estimate in adolescence implies that the average height of the 1960s cohort group was *nearly half an inch* shorter in adolescence (ages 12 to 17) than the 1950s cohort group. This difference is significantly different from zero, and the 95 percent confidence interval would rule out cross-cohort declines smaller in magnitude than .24 inches.

Strikingly, the estimates suggest that the later-born cohort then caught back up in height in adulthood. The point estimate actually implies that the 1960s cohort group was .15 inches *taller* than the 1950s cohort group as adults, although the confidence interval includes values consistent with anything from an approximately .15 inch cross-cohort decline in height to a .45 inch cross-cohort improvement in height. Importantly, the estimates imply with a high degree of confidence that the gap in adulthood was much smaller than it had been in adolescence.

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

$$Y_{i,ac} = \delta^{a=12-17}_{c=1961\text{-}67} + \gamma_{c=1961\text{-}67} + \mu + f(a) + \epsilon_{i,ac} \tag{2}$$

where $\delta^{a=12-17}_{c=1961\text{-}67}$ estimates how much larger the between-cohort height difference at ages 12-17 is than it had been at ages 6-11.

For the sample of 12-to-17- and 23-to-40-year-olds, I estimate:

$$Y_{i,ac} = \delta^{a=23-40}_{c=1961\text{-}67} + \gamma_{c=1961\text{-}67} + \mu + f(a) + \epsilon_{i,ac} \tag{3}$$

where $\delta^{a=23-40}_{c=1961\text{-}67}$ estimates how much larger the between-cohort height difference at ages 23-40 is than it had been at ages 12 to 17. I again include a flexible control for age, $f(a)$, — I include an adult dummy variable and for ages 6 to 11 and 12 to 17 I include dummies for age in years and a quadratic in age in months.

Figure 4, panel (b) shows the resulting estimated growth differences, $\delta^{a=12-17}_{c=1961\text{-}67}$ and $\delta^{a=23-40}_{c=1961\text{-}67}$. The point estimates imply that the later-born cohort grew .43 inches less between ages 6-11 and ages 12-17 than the earlier-born cohort. However, this reversed between ages 12-17 and adulthood, when the later-born cohort then grew .56 inches *more* than the earlier-born cohort. The differences in growth are statistically significant.

The estimates strongly suggest that the 1960s cohort grew more slowly in adolescence, either because of a later or a smaller adolescent growth spurt, and then was able to catch up in height by subsequently growing faster or for a longer period into early adulthood.[7]

Unfortunately, the relatively small sample sizes do not allow me to distinguish the precise age at which the gap in height first opened and when it began to close. This makes it unclear whether the height differences are due to differences in the timing of the adolescent growth spurt or differences in the growth velocity achieved during (or around) the adolescent growth spurt. Historically, in South Korea and Japan, these two processes, timing and peak height velocity, moved in tandem. That is, the enormous cross-cohort changes in growth in South Korea and Japan involved a move towards both an earlier growth spurt and a faster peak height velocity during the spurt (Cole and Mori, 2018). Therefore, it could be plausible that a similar change occurred between the 1950s and 1960s cohorts in the United States, but in reverse: that the 1960s cohort had both a later and smaller adolescent growth spurt on average than those born a decade earlier. However, note that the East Asian changes coincided with changes in final attained height, which are not seen for the American cohorts studied here.[8]

Without individual panel data, even larger sample sizes would not provide a complete picture of the cross-cohort changes in the growth pattern. As explained clearly by Tanner (1990), growth and velocity curves at the cohort level look very different from those at the individual level because of within-cohort variability in the timing of the growth spurt. For example, a reduction in the within-cohort variability of the age at peak height velocity can lead to an apparent increase in peak

[7]Note that this is a catch-up in absolute terms, not just catch-up relative to a growth reference, which can be more difficult to interpret Schneider (2023).

[8]Interestingly, studies of the individual level variation between timing and peak velocity in a single cohort have found the opposite association, with boys entering puberty later having a larger peak velocity (Cole et al., 2010).

height velocity in adolescence when measured at the population level — even if individual level peak height velocities remain unchanged.[9]

A number of extensions and robustness checks of these broad patterns are presented in the appendix. One potential concern is that the onset of height stagnation for cohorts born near mid-century corresponds to the timing of the change in measurement from historical to modern representative sources. Appendix Figure A9 cross-validates the finding of adult height stagnation using only NHES and NHANES data.[10] Panel (a) shows the adult-height series for all native-born males, retaining NHES I observations with unknown nativity; Panels (b) and (c) show the corresponding series for white non-Hispanic and Black non-Hispanic males. The all-native series makes clear that the stagnation in adult height is not specific to one racial subgroup. The NHES and NHANES data also allow me to examine trends in other groups beyond non-Hispanic whites. A similar qualitative pattern, of steady secular growth in height in the first-half of the twentieth followed by near stagnation in the second half of the twentieth century emerges for native-born men and women as a whole, and for native-born non-Hispanic whites and blacks of both genders (Appendix Figures A6, A7, and A8).

For the Regime II and III cohorts fully covered by NHES and NHANES, I also examine the childhood and adolescence height trend for these additional demographic groups. The decline in adolescent height appears to have occurred for both black and white males, though results for blacks are noisier. Both of these groups also exhibit the post-1970 increase in adolescent height, alongside stagnant adult height.

However, there is not strong evidence that females' adolescent height declined between the 1950s and 1960s cohorts. The point estimate for the difference in height at ages 12-17 is just $-.07$ inches (see Figure A2). The estimate is not significantly different from zero, though one also cannot rule out declines of about a quarter of an inch at conventional levels. The adolescent growth spurt for females occurs approximately 2 years earlier for girls than boys. Therefore, I reran the above analysis, shifting the child and adolescent age groupings down by two years. The results are very similar qualitatively with height differences small in magnitude and not significantly different from zero at conventional levels (see Figure A5)

Females do however exhibit the same qualitative Regime III pattern as men: rising childhood and particularly adolescence height with no concomitant increase in adult height (and some evidence of decline in adult height). Appendix Figures A8, A6, and A7 show the patterns for all native-born females, and non-Hispanic whites and blacks. It appears females born in the 1970s and subsequent decades also had an accelerated growth tempo, without gains in final height.

[9]For an extended discussion of related issues see also Gao and Schneider (2021).

[10]This replicates the NHES/NHANES adult-height evidence in Komlos and Lauderdale (2007a,b); Komlos (2010) and extends it with additional survey waves through the 2021–2023 cycle.

Why might the Regime II pattern for girls differ so significantly? Girls have slightly smaller adolescent growth spurts than boys. In the three years including peak height velocity, boys tend to grow about 9 inches in total, while girls tend to grow slightly less than 8 inches (Tanner, 1990). It is also of course possible that there were small changes in the growth pattern of girls that were not detectable in the NHES and NHANES samples. Puberty also differs substantially between biological males and females. It is a rapid period of sex differentiation, and the hormonal processes driving the adolescent growth spurt in males and females differ (Tanner, 1990). Below, I show evidence that a different measure of pubertal development, menarche, age at first menstrual period, did change dramatically between the 1950s and 1960s cohorts of females.

B Pubertal Timing: Age at Menarche

In this section, I show evidence that a different measure of pubertal development, age at menarche, the onset of menstruation in girls, changed dramatically across twentieth century cohorts. The changes point to a similar pattern in developmental 'tempo' to that found above for males height: an accelerating rate of development for cohorts born in the first half of the twentieth century (Regime I), a stagnation and reversal for 1960s cohorts (Regime II), and a partial rebound but no resumption of the prior secular trend for cohorts born in 1970s and after (Regime III).

The above section documented patterns in height which suggested profound changes in the tempo of growth and the timing of the adolescent growth spurt. The adolescent growth spurt is closely tied to the broader biological process of puberty (e.g., Tanner, 1990, 1986). Individuals and populations who experience earlier puberty also tend to experience earlier adolescent growth spurts. Many measures of pubertal development exist for males and females, though menarche is the most widely used and consistently measured. Menarche provides an additional lens through which to study developmental 'tempo' over the long run, in this case for females.

Figure 5 plots cohort trends in age at menarche using data from NHES and NHANES surveys described above. The data in this series are based on retrospective reports in which adult women report the age at which they had their first menstrual period. The figure plots binned cohort means with confidence regions shown in grey.

For Regime I cohorts born in the first half of the twentieth century, age at menarche declines steadily, from roughly 13.5 years for the early cohorts to approximately 12.5 years for mid-century cohorts. This pattern is part of the well-documented secular decline in age at menarche observed in many countries (Hauspie et al., 1997; Cole, 2003). Similar to the secular trend in height, historical research has linked this global decline to improvements in childhood nutrition, disease environments, and overall living conditions (Cole, 2000, 2003).

As for height, a striking stagnation and then reversal occurs for cohorts born between mid-century and the 1960s (Regime II cohorts). Interestingly, the binned scatter plots put the turning

point in the mid-1940s — remarkably similar to the 1947 turning point I found in health and human capital, measured in larger datasets allowing more precision, in Reynolds (2025). For cohorts born after this period progress stalls, and then beginning with early 1950s cohorts actually reverses. The estimates suggests females born in the 1960s experienced menarche approximately .25 years later than those born a decade earlier. In other words puberty was delayed for these cohorts. This mirrors the finding in the previous section for males height: which suggested a delay in the adolescent growth spurt for 1960s cohorts. The evidence here suggests female pubertal development was delayed for the same cohorts.

For cohorts born after the 1960s, Regime III cohorts, age at menarche rebounded to approximately mid-century levels. The secular trend towards earlier development did not resume, age at menarche appears to have remained at around 12.5 years for cohorts born between the late-1970s and 1990s. This rebound is again broadly similar to that seen in height of males and females for the same cohorts.

Unlike for adult height, this is some evidence that the secular decline in menarche slowed or stopped in other high income countries. However, there does not seem to be any evidence of an actual reversal in the secular trend and temporary increases in age at menarche, such as that documented here, in other countries (Hauspie et al., 1997; Cole, 2003).

I also show that the Regime II and III patterns are evident in contemporaneous reports of menarche onset, from NHES and NHANES observations of these cohorts as adolescents. This ameliorates concerns about complex recall bias driving the patterns evident from the retrospective reports for these cohorts.

Figure 5 uses contemporaneous reports to show that the striking Regime II reversal is evident as a shift in entire distribution of age at menarche. Figure 5 shows smoothed estimates of the share of American-born females reporting having had their first menstrual for the 1951-57 and 1961-67 cohorts of American-born females between ages 12 and 17. The lines come from separate kernel regressions of a menarche dummy on age in months, with an Epanechnikov kernel and a bandwidth of 12 months. At all ages from 12 to 17, the estimated share of girls in the 1960s cohort group who have had their period is approximately three percentage points lower than for those born a decade earlier. Regression estimates imply that at a given age between 12 and 17, females in the 1960s cohort were 2.9 percentage points less likely to have experienced menarche than those in the 1950s cohort (with the confidence interval ranging from 5.6 percentage points to .1 percentage points).

Appendix Figure A11 shows trends in age at menarche across cohorts born in the second half of the twentieth century. It uses censoring-adjusted weighted means from intercept-only interval-regression models to account for the fact that not all girls have experienced menarche at the time of the survey. For each cohort group, I fit a weighted interval regression of latent age at menarche on a constant, where the dependent variable is interval-censored: observed age at menarche for

girls who have already menstruated, and left-censored at the survey age for those who have not. The implied mean age at menarche for 1950s cohorts is 12.9 years, rising to 13.1 years for the 1960s cohorts — a shift of approximately 0.15 years, slightly smaller than that implied by the retrospective reports. Interestingly, the implied rebound for 1970s through 1990s cohorts is larger, with all these cohorts estimated to have a mean age at menarche of approximately 12.6 to 12.7 years. Qualitatively, these patterns validate the Regime II and III findings of reversal for 1960s cohorts, and rebound for subsequent cohorts.

C Educational Attainment

This section reviews previously documented trends in educational attainment across American cohorts born in the twentieth century. The educational patterns closely track those documented above for height and menarche for Regime I and Regime II cohorts: sustained growth in educational attainment for cohorts born in the first half of the century (Regime I), a sudden stop and temporary reversal for cohorts born from 1947 to the mid 1960s (Regime II). The educational attainment of Regime III cohorts, those born after the 1960s, partially rebounds, though does not return to the Regime I trend. The rebound is particularly shallow for men and larger for women.

Panel A of Figure 7a plots cohort trends in educational attainment for native-born males and females. It shows average years of schooling reported in pooled Decennial Census and ACS from IPUMS (Ruggles et al., 2015). [11] Appendix Figures A13, A14, and A17 show trends in educational attainment by high school degree, Bachelor's degree and other levels, as well as additional breakdowns by race.

Educational attainment increased steadily across cohorts born in the first half of the twentieth century, echoing the Regime I pattern shown above in height. Average years of schooling increased from 13 to more than 15 years of schooling for both men and women. These rapid gains underlie Goldin and Katz (2007) description of the twentieth century in America as the "human capital century." The most important driver of this increase was the enormous increase in the rate of high school graduation, from around 30 percent in the early 1900s to near 90 percent by 1947 (see Panel A of Figure A13). Goldin and Katz (2007) highlight the grassroots nature of the "high school movement" and argue the key driver was the large economic returns to a high school degree. Though they note that the returns to a high school degree were already high early in the twentieth century. The rate of at which cohorts attended college, earned bachelor's degrees, and even advanced degrees also increased substantially across these cohorts (see Figures A13 and A17). The gains were widespread (though not uniform) across the US and across racial groups, with Blacks experiencing more rapid growth and partial convergence towards white educational levels. If anything, these estimates from census data likely understate the growth in educational attainment,

[11] Education variables are normalized to years of completed schooling following Goldin and Katz (2007).

as older generations appear to have inflated their reported schooling in the 1940 and 1950 census Goldin (1998) [12]

The rapid increases in educational attainment hit a sudden stopped for cohorts born after mid-century. For men attainment declined by about half a year between the late-1940s and 1960 cohorts, and began only a slow, partial rebound in the 1960s. For women, the stagnation hits slightly later, for early-1950s cohorts, and rebounds after 1960 were more rapid — leading women's educational attainment to substantially outpace men's. This slowdown is noted in Goldin and Katz (2007) and elsewhere. As measured in the IPUMS data, the previously consistent growth in HS graduation rates of both males and females slow to a crawl after 1947, with that for men declining slightly. The IPUMS data do not allow one to distinguish between high school graduates who earned their diploma through the traditional route. Heckman and LaFontaine (2010) argue that GED recipients should be treated as HS dropouts, as their labor market outcomes are more similar to dropouts than HS graduates. As they show, if one follows this convention, the declines in high school completion after mid-century cohorts are substantial. This can be seen in Appendix Figure A14, which shows estimates of high school graduation rates based on the number of diplomas issues per year divided by cohort size at 17. By this measure HS graduation rates peak at approximately 90 percent for the 1947 cohort and subsequently decline.

Bachelor's degree attainment rates also decline sharply for cohorts born after mid-century, particularly for men. Card and Lemieux (2001b) provide a detailed study of the decline in both college enrollment and completion for the same groups. They show 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. Panel B of Figure A13 shows the particularly sharp decline in Bachelor's degree completion rates for men, from a peak of 33 percent for the 1947 cohort to a trough of 26 percent for the 1960 cohort. This decline in college completion rates is the key driver of increases in the college-high school wage gap in the "canonical model" of Katz and Murphy (1992) and subsequent studies (Card and Lemieux, 2001a; Autor et al., 2008).

While this stagnation, and on some dimensions decline, in educational attainment for Americans born after mid-century is well-known its causes are not. Acemoglu and Autor (2012) write: "We do not believe that social science has so far produced an adequate account of what went wrong with the U.S. human capital machine." They also argue that the sharpness of the change in trend by cohort suggests it is unlikely to be caused by a sudden change in the school system, and that "other factors are thus likely to be at play."[13]

[12] Appendix Figure A14 shows estimates of high school graduation rates coming from historical estimates of the number of degrees awarded in a given years divided by the population of 17-year-olds, plotted based on approximate birth year (NCES Digest of Education Statistics). The level of high school graduation rates is indeed lower around 1900, at around 19 percent, but is also lower in 1947, at around 75 percent, so the increase is quite similar to that derived from Census data.

[13] Relating to the decline in college completion in particular, Card and Lemieux (2001b) present an extensive study

Regime III cohorts, those born in the 1970s and later, experience a partial and mixed recovery in educational attainment. Women show rapid gains in average years of schooling, driven by enormous progress in college attendance and completion (see Goldin et al. (2006)). As Panel B of Figure A13 shows, women's rate of Bachelor's degree completion rises from around 27 percent for the 1960 cohort to 37 percent for the 1970 cohort, and continues climbing to 46 percent by the 1985 cohort. Men's progress in college completion is much more modest, rising from 26 percent for the 1960 cohort to 32 percent for the 1970 cohort and 37 percent by the 1985 cohort.

D Mortality

This section documents cross-cohort trends in adult mortality rates. For cohorts born from 1900 through the 1960s (Regime II and III), there is a remarkably close tracking between the mortality trends and those documented above for height, biological development, and educational attainment. Regime III cohorts are still too young to have experienced substantial mortality from internal causes, though their external mortality has been elevated and by many measures they are less healthy than earlier cohorts.

Panel (b) of Figure 7 shows mortality rates from age 25 to 60 for cohorts born from 1908 to 1963. For each cohort these reflect the share of 25-year-olds who died before their 60th birthday. They are calculated from data from the Human Mortality Database using standard lifetable methods. The shorter birth cohort range is the results of two restrictions. First the national vital statistics system, and therefore my data, began in 1933. Second, I restrict the cohort/age cells to not include year affected by the Covid-19 epidemic. Appendix Figure A10 shows results separately by gender.

The figure shows rapid and sustained declines in mortality across cohort born in the first half of the twentieth century. More than 19 percent of 25-year-olds in the 1908 cohort died before they reached 60. By the 1947 cohort this had fallen to 11 percent. This trend parallels the large gains in height, accelerated pubertal development, and educational attainment documented above.

This mortality progress suddenly stagnated for cohorts born after mid-century. The timing of the stagnation closely corresponds to the end of the secular trend growth in height, and the temporary reversal of the trend towards earlier puberty, representing Regime II of human development. Appendix Figure A10 suggests that while both men's and women's mortality stalled near mid-century, the timing and exact pattern differ slightly. For men the break occurs in 1947, initially mortality increases and it then fluctuates around a stable level. For women, a sharp break is clear at the 1950 cohort, and mortality increases linearly between that cohort and the 1963 cohort, rising from 7.5 to 8.0 percent (an increase of approximately 0.5 percentage points).

of possible causes, concluding that for women it could be explained by low returns to education and cohort size, but that for men the decline represents a fundamental trend break with no observable explanation. Handy and Shester (2019) suggest that changes in the average parity or birth order by cohort can explain around one third of the decline in Bachelor's completion for white men born between 1946 and 1960.

In prior papers I present an array of evidence suggesting that this break in trend reflects to a large degree a true change in underlying health, a trend break in "cohort effects", rather than merely bad luck for these cohorts — being exposed to bad external age-by-year shocks. In Reynolds (2025), I show sharp trend breaks in cohort effects are evident in both additively separately age-period-cohort (APC) models and more flexible models allowing for quadratic-age-by-year shocks. Reynolds (2024) focuses on white Americans in particular, and shows evidence of a cohort-specific kink in the log-linear relationship between mortality and age, the Gompertz curve, evident already by the mid-1980s.

The Regime III cohorts are still young enough to have low mortality rates, shaped significantly by external causes of death. These cohorts have had remarkably high mortality rates in early and young adulthood compared to Regime I and even Regime II cohorts (Case and Deaton, 2015, 2017). However, this elevated mortality is driven primarily by deaths from drug overdoses, which appear to be driven largely by a period-specific shock to the supply of prescription and illicit opioids (Case and Deaton, 2015). Mortality rates from this epidemic have recently begun falling. It is not obvious that these cohorts elevated mortality in midlife reflected poor physiological health which will persist into older ages.

However, it is worth noting that Regime III cohorts have elevated rates of metabolic syndrome and other signals of poor underlying health which may presaged elevated mortality as they age. Below, I discuss the rise of childhood obesity and a "obesogenic" environment for these cohorts, as both a potential cause of the increased growth tempo, and as a driver of poor health throughout the lifecycle, which may become increasingly apparent in mortality — particularly from cardiovascular disease — as they age.

In sum, the cross-cohort patterns in adult mortality align very closely with the Regime I and Regime II pattern shown in developmental outcomes above. Regime III cohorts have had elevated mortality in young adulthood and mid-life from external causes, and appear to have poor metabolic health, but the mortality implications of the third development regime will only become clearer as these cohorts age.

4 Interpretation, micro evidence, and implications

The above section showed that cross-cohort trends in height and age-at-menarche reveal three distinct regimes in human development. It also showed that the first two regimes closely mirrored the rise and stagnation (and on some dimensions fall) of educational attainment and adult mortality. This section combines ideas from the economics of human development (e.g., Cunha and Heckman, 2007; Case and Paxson, 2008) and from human biology to interpret these patterns and their implications for our understanding of human development and human capital in the twentieth century United States.

A Biological development as a signal of childhood conditions

A large body of evidence suggests that both biological development (height, growth tempo, menarche), human capital development, and health are shaped by the environment experienced early-in-life. Nutrition, pollution, and other intentional investments and external shocks in the first few years of life are thought to have lasting effects on the development of the human body, cognitive development, and therefore economic outcomes (e.g., Almond et al., 2005; Currie and Almond, 2011).

This has led to a large history of economists and economic historians using adult height as a proxy for early-life environment. (e.g., Fogel, 2012; Floud et al., 2011; Costa, 2015). Case and Paxson (2008) present a simple economic framework justifying this approach and highlighting some caveats. One important point is that to that extent that differences in early-life environments in one domain (eg. nutrition) are correlated with differences in another domain (eg. parenting style) then correlations between height and adult outcomes cannot distinguish *which* features of early-life drive later life outcomes. [14] Case and Paxson (2008) also review and document correlations between height, cognitive ability and health in both adulthood and childhood. While menarche and the timing of growth have been studied less by economists, human biologists believe that they are driven by environmental conditions. As Tanner (1990) famously wrote, "growth is a mirror of the conditions of society."

At a minimum, the existence of three distinct regimes suggests environmental conditions changed across cohorts. If these environmental conditions also effect childhood health and human capital, this suggest that early-life health and human capital move as well: that it improved across Regime I cohorts, stopped improving and declined slightly for Regime II cohorts.

Below I present some suggestive calibrations of the magnitude of the role of early-life health in driving cross-cohort trends in human capital and health. It is however worth pausing to discuss conceptually what we mean by early-life health conditions "driving" or "causing" a particular percent of the changes in health and human capital across cohorts. I the context of a model of skill-development of the form of Cunha and Heckman, changes in the vector of skills early-in-life can affect skills/health later in life both directly through persistence and dynamic complementarities, and but als through endogenous changes in investments by parents induced by the early-life changes. At the cohort level additional endogenous mechanisms may operate to reinforce initial differences in early-life health and human capital. For example, Goldin and Katz (2007) argue that gain in schooling in the twentieth century were the result of local grassroots campaigns. For example, people recognized there was a high return to secondary schooling and organized locally to fund

[14] Standard omitted variable bias logic also of course suggests that if subsequent shocks later in life are themselves correlated with child environments then a correlation between height and adult outcomes cannot isolate early-life as the cause of the adult outcome differences.

high schools. If these returns or the non-pecuniary costs of schooling are shaped by early-life health and human capital — then changes in early life health and human capital could induce the grassroots organizations to invest in public education.

To the extent that there are such endogenous investment responses to changes in early-life health and human capital, a sensible reduced-form causal effect of the changes in early-life health would include both the direct effects on skill and the endogenous response of future investments. In principle, the existence of such compensating investments at the local level could be tested, though I leave this for future work.

B Micro evidence consistent with developmental mechanisms:

This section provides novel evidence consistent with the interpretation of biological development as a proxy or "signal" for early-life conditions. To do so I use the small panel dataset that results from a linkage between NHES II and NHES III surveys. This extends prior evidence, such as that in Case and Paxson (2008) by focusing on growth in height and age at menarche, in addition to the level of height. It also is of direct applicability to the Regime I to Regime II transition, as the sample comprises children and adolescents in mid-century birth cohorts.

The NHES II and III surveys examined many of the same children at two points in time (roughly ages 6–11 and 12–17), creating a small panel of approximately 2,000 native-born children with height measurements at both waves. Growth rate is defined as the height gain in inches divided by the years between waves, winsorized at the 99th percentile. The outcome variable is the Achievement Index score at wave 2, an IQ-scaled composite with mean approximately 100 and standard deviation approximately 15.

I first show that there is a "gradient" between socioeconomic status of parents and physical growth and development of children. To the extent that higher socioeconomic status parents invest more in the health and human capital development of their children, this pattern is consistent with biological development as a proxy for early-life conditions conducive to health and human capital development. Figure 8 panel (B) shows a binned scatter plot of the relationship between household income rank and childhood-to-adolescent height growth. The figure shows a clear positive gradient: children from higher-income households grow faster during adolescence.

Second, I show that growth is correlated with one measure of childhood human capital, test scores. Figure 8 panel (A) visualizes this relationship in a binscatter of test score gains versus childhood-to-adolescent height growth. The figure shows a clear positive relationship between test scores and growth. In both panels, the plotted bins partial out sex-specific quadratics in age at both waves and wave-1 height percentile, matching the baseline regression specification.

Table 2 shows regression results from similar specifications. Both columns control for age quadratics at both waves and wave-1 height percentile. In the baseline pooled specification (col-

umn 1), an additional inch per year of growth is associated with a 1.7-point increase in the Achievement Index ($p < 0.01$). Adding wave-1 household income percentile (column 2) attenuates the coefficient to 1.3 ($p < 0.05$), while income itself is strongly predictive (0.19 points per percentile, $p < 0.01$). Appendix Table A2 shows the corresponding by-sex estimates and suggests that the relationship is stronger for girls than boys, consistent with menarche-related growth variation being an important component of the signal for girls.

I have conducted preliminary analysis using CNLSY data which corroborate and extend these results. A similar relationship between growth and test scores is found, and the relationship holds in sibling fixed effects models. These result will be added to future drafts.

C Possible causes of the regime shifts:

The findings of this paper provide additional evidence that the declines in adult outcomes for Americans born after midcentury have their roots in adolescence, or earlier. The relatively delayed puberty of cohorts born soon after midcentury strongly suggests that something had already gone wrong for these cohorts by adolescence. Puberty generally occurs later for individuals of lower socioeconomic status, and there has been a secular trend towards earlier puberty over the last 200 years in many high-income countries.[15] For example, Lleras-Muney et al. (2022) find that the timing of the adolescent growth spurt and age at menarche are both negatively associated with socioeconomic status in a large sample of low-income countries. While the exact biological mechanisms determining the age of puberty and the adolescent growth spurt are not fully understood, the trend over time and differences by income are thought to be driven by differences in nutritional status and disease exposure (e.g. Cole, 2003). Recent findings suggest that caloric deprivations in childhood activate particular receptors in the brain which delay the onset of puberty and the adolescent growth spurt, but also lead to a period of leg growth longer into early adulthood (Lam et al., 2021). Broadly, it seems likely that these cohorts had some kind of hormone or nutrient deficiency in adolescence — either due to a shock occurring during adolescence or an earlier shock that particularly affected biological processes related to puberty.

One intriguing way to view the findings is through an adaptive framework. This framework is based on work by human biologists (Gluckman et al., 2005; Gluckman and Hanson, 2006a,b), and outlined and applied to findings from the anthropometric history literature in Schneider (2017). Broadly, the idea is that many of the effects of responses to environmental shocks on human growth are adaptive, in the sense that they increase the chances of survival to reproductive age and of producing viable offspring. For example, the delayed adolescent growth spurt and subsequent catch-up growth leading to a similar final height in adulthood, observed for the 1960s cohorts, is

[15] See for example Lleras-Muney et al. (2022); Wyshak and Frisch (1982); Hauspie et al. (1997); Cole (2003) and the citations therein. Long run changes in height-for-age are also generally largest in the teenage years for boys, consistent with a long-run trend towards an earlier growth spurt Meredith (1964); Cole (2003).

the predicted adaptive response to a postnatal shock in this framework.

Interestingly, the pattern of growth for 1970s to 1990s and cohorts is approximately consistent with the predicted adaptive response to a prenatal shock in the adaptive framework. The logic here is that a negative shock in the prenatal period suggests that conditions are likely to be bad in the future. Therefore, an adaptive response is to speed up maturation to increase the probability of reaching reproductive age, but to reach a shorter adult height that will require fewer calories to maintain. This described pattern is similar to that found in the 1970s to 1990s cohorts: growth tempo sped up and age at menarche decreased, but adult height was unchanged — and may have declined for the cohort born in the 1990s. In addition to these adaptive responses to a prenatal shock, there is (debated) evidence that exposure to prenatal shocks also increase the risk of cardiovascular disease and other conditions (Barker and Osmond, 1986; Almond and Currie, 2011).

While much more research is needed, this interpretation paints a coherent and troubling story of health trends across American cohorts born since mid-century. It would suggest that a postnatal shock negatively affected cohorts born in the first approximately two decades after midcentury. These cohorts would then start to give birth themselves, and I have shown previously that they gave birth to lower birthweight infants (Reynolds, 2025); therefore, their depressed health could plausibly have led to a worsening of the prenatal health environment for cohorts born between the 1970s and the 1990s.

Regime I to Regime II shift What could have been the underlying postnatal shock that negatively affected cohorts born in the first two decades after midcentury? In previous work, I presented evidence against a number of ex ante plausible causes of the broader cohort health decline but was unable to find a smoking gun (Reynolds, 2023, 2024, 2025). I suggested that two remaining broad candidate hypotheses were the most plausible. The first broad hypothesis is that the complex demographic process of the baby boom led to the cohort decline — for example, through "cohort crowding" (eg. Freeman, 1979; Easterlin, 1987; Bound and Turner, 2007; Macunovich, 2002) and birth order effects. The second candidate hypothesis is prenatal exposure to lead pollution from motor vehicle exhaust, discussed in detail in Reynolds (2024).

Do the new findings in this paper, that the adolescent growth spurt and puberty were particularly affected, shift the weight of evidence for or against either of these hypotheses?

In my view the new findings provide additional evidence against traditional cohort crowding mechanisms. The key cohort crowding mechanisms discussed in the above literature — such as decreased educational investment due to crowding and worsened labor market outcomes due to "oversupply" — appear unlikely to affect adolescent height.

However, baby-boom-related causes more focused on family size and birth order come out looking stronger. For example, the idea that increased birth rates and larger family sizes led to a

worse infectious disease environment appears worth exploring. The average birth order, or "parity," across cohorts changed sharply with the baby boom, and, as I have discussed in previous work, could be a plausible contributor to the cohort health decline. There is a large body of literature documenting the effects of birth order on adult height. A common finding is that later-born children are shorter than their earlier-born siblings, with the comparison made within-family to control for other family specific factors correlated with family size (e.g. Hermanussen et al., 1988; Hatton and Martin, 2010; Myrskylä et al., 2013). There is a smaller literature on the effect of birth order on the adolescent growth spurt. I found two studies which find that first-born children are taller in adolescence and further along in pubertal development than later-born children, but they do not contain sibling samples and therefore cannot control for unobserved family specific factors (Wells et al., 2011; Kwok et al., 2016). Two prominent mechanisms have been suggested as driving birth-order effects on other outcomes, respiratory disease spillovers within the family (Daysal et al., 2021) and decreased parental health investment (Pruckner et al., 2021), seem to quite plausibly have the potential to affect adolescent growth and development as well.

This paper's new findings on adolescent height and delayed puberty also seem to strengthen the argument for lead pollution as a potential cause of the cohort health decline. Motor vehicle use began to increase rapidly after 1945, and lead additives were ubiquitous in gasoline. By the 1960s children across the US had blood lead levels well above those now considered safe. For example, McFarland et al. (2022) estimate that the share of children with blood lead levels above the 2015 threshold for "clinical concern" was 50 percent for the 1940-45 cohorts and reached 100 percent by the 1966-75 cohorts — meaning every single child's blood lead content was above this threshold. Prenatal and early life lead exposure is thought to have broad and lasting negative health effects, impacting for example the development of multiple organ systems, cognitive ability and emotional regulation, and cardiovascular disease.[16]

Importantly, lead exposure has also been linked to delayed puberty and slower adolescent growth. Lead is thought to be an endocrine disruptor that can affect the hormones that control puberty and the adolescent growth spurt. therefore affect the . A large number of studies have shown that adolescenct boys and girls with higher blood lead levels, measured either contemporaneously or in early childhood, are shorter and have delayed pubertal development.[17]

Additionally, the lead phaseout appears to coincide with gains in childhood and adolescence. Lead additives were phased out of gasoline beginning in the 1970s, and blood lead levels of children fell rapidly. The results of this paper show that childhood growth and adolescent growth and development also improved for these cohorts. However, as noted above, completed adult

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

[17]See for example Selevan et al. (2003); Wu et al. (2003); Hauser et al. (2008); Naicker et al. (2010); Sergeyev et al. (2017); Liu et al. (2019).

height remained stagnant, and many other measures of health and human capital were stagnant or falling. As described above in the context of the adaptive framework, perhaps the mechanism driving outcomes for the 1960s cohorts was different than those which drove subsequent cohorts: lead exposure drove delayed puberty and poor outcomes in adulthood for the 1960s cohort; 1970s to 1990s cohorts were less exposed to lead, allowing their pubertal development to rebound, but the underlying prenatal shock of being *born to* lead-exposed cohorts led to stagnant final height and other poor outcomes in adulthood.

Regime III shift Cohorts born after the late 1960s exhibit a distinct pattern of biological development that differs sharply from both earlier regimes. Childhood and adolescent height increase, and measures of pubertal development indicate a return toward earlier maturation relative to the 1960s cohorts. However, this is not a return to the earlier secular trend. Adult height remains flat, and the broader patterns in human capital and health outcomes for these cohorts are mixed. Educational attainment shows a partial recovery—particularly for women—while the long-run mortality implications remain unclear, as these cohorts have not yet reached ages where chronic disease mortality is fully realized. Instead, Regime III is characterized by a shift in the timing of development, with individuals reaching higher shares of their final height earlier in the life cycle, but without corresponding gains in final adult stature.

This is not a simple return to the earlier secular trend. In Regime I, earlier maturation coincided with increases in final adult height. In contrast, Regime III combines faster developmental tempo with no gains in final stature. What does this pattern suggest about the childhood environments experienced by these cohorts, and about the contribution of early-life conditions to health and human capital in adulthood?

As described above, one possible interpretation, consistent with the adaptive framework, is that this pattern reflects responses to early-life conditions operating in utero. In this framework, adverse prenatal environments can induce developmental responses that favor earlier maturation and reduced investment in final body size. The combination of faster developmental tempo and flat adult height observed for these cohorts is qualitatively consistent with such a response.

Another possible explanation is the emergence of an "obesogenic" environment in the United States beginning in the 1970s. A large medical and epidemiological literature documents substantial increases in childhood adiposity over this period, alongside changes in diet, physical activity, and broader environmental conditions. This literature also finds that higher levels of body fat in childhood are associated with earlier pubertal development and accelerated growth tempo, particularly for girls (eg Biro et al., 2006a; Kaplowitz, 2008). Proposed mechanisms include the role of adipose tissue in hormonal regulation, which can trigger earlier onset of puberty. Its worthing noting thought that estimates vary across populations, effects differ by sex, and causality is

difficult to establish due to confounding by other early-life conditions. Nevertheless, the timing and biological plausibility of these links suggest that rising childhood adiposity may be an important component of the environmental changes underlying Regime III patterns of development.

These interpretations need not be mutually exclusive. One possibility is that the rise in childhood adiposity itself reflects changes in early-life conditions, including prenatal environments. For example, a mismatch between prenatal signals and postnatal environments, such as a nutritionally constrained prenatal environment followed by caloric abundance in childhood, could lead to both increased adiposity and accelerated developmental timing.

Under either interpretation, the patterns observed for Regime III cohorts do not point to clear improvements in underlying physiological health. The accelerated maturation and increased adiposity of these cohorts raise concerns for metabolic health. A large literature documents rising prevalence of obesity and metabolic risk factors in recent decades. While these cohorts have not yet reached ages at which chronic disease mortality is fully realized, these patterns suggest the importance of carefully monitoring future trends in cardiovascular and all-cause mortality.

Future drafts will examine these implications more directly by linking cohort patterns in early-life development to adult health outcomes. Using NHANES, I will relate cohort variation in childhood adiposity and developmental tempo to adult metabolic health. Complementary analyses using longitudinal datasets, CNLSY and ADD Health, will examine individual-level relationships between early-life growth and later-life health and economic outcomes.

5 Conclusion

This paper documents three distinct regimes of biological development across Americans born in the twentieth century, and shows that the timing of these regime changes mirror the rise, fall, and stagnation of health and human capital. I argue that the changes in biological development are the likely result of profound changes in childhood environment, which could have played a key role in driving the distinct health and human capital trajectories of each cohort. In particular, the sudden stagnation in childhood and adult height after mid-century, and the decline in adolescent height and age at menarche point to deep early life roots of the broad decline in health and human capital of Americans born after mid-century which I've documented previously (Reynolds, 2025).

References

Daron Acemoglu and David Autor. Skills, tasks and technologies: Implications for employment and earnings. *Handbook of Labor Economics*, 4:1043–1171, 2011.

Daron Acemoglu and David Autor. What does human capital do? A review of goldin and katz's "The race between education and technology". *Journal of Economic Literature*, 50(2):426–63, 2012.

Anna Aizer, Janet Currie, Peter Simon, and Patrick Vivier. Do low levels of blood lead reduce children's future test scores? *American Economic Journal: Applied Economics*, 10(1):307–41, 2018.

Douglas Almond and Janet Currie. Killing me softly: The fetal origins hypothesis. *Journal of economic perspectives*, 25(3):153–172, 2011.

Douglas Almond, Kenneth Y Chay, and David S Lee. The costs of low birth weight. *The Quarterly Journal of Economics*, 120(3):1031–1083, 2005.

David Autor, Lawrence F. Katz, and Melissa S. Kearney. Trends in US wage inequality: Revising the revisionists. *The Review of economics and statistics*, 90(2):300–323, 2008.

David JP Barker and Clive Osmond. Infant mortality, childhood nutrition, and ischaemic heart disease in england and wales. *The Lancet*, 327(8489):1077–1081, 1986.

Frank M. Biro, Philip Khoury, and John A. Morrison. Puberty in girls of the 21st century. *Journal of Pediatric and Adolescent Gynecology*, 19(3):161–165, 2006a.

Frank M Biro, Philip Khoury, and John A Morrison. Puberty in girls of the 21st century. *Journal of Pediatric and Adolescent Gynecology*, 19(3):161–165, 2006b.

Hoyt Bleakley, Dora Costa, and Adriana Lleras-Muney. Health, education, and income in the united states, 1820–2000. *NBER Chapters*, pages 121–159, 2014.

Howard Bodenhorn, Timothy W Guinnane, and Thomas A Mroz. Sample-selection biases and the industrialization puzzle. *The Journal of Economic History*, 77(1):171–207, 2017.

John Bound and Sarah Turner. Cohort crowding: How resources affect collegiate attainment. *Journal of public Economics*, 91(5-6):877–899, 2007.

David Card and Thomas Lemieux. Can falling supply explain the rising return to college for younger men? A cohort-based analysis. *The Quarterly Journal of Economics*, 116(2):705–746, 2001a.

David Card and Thomas Lemieux. Dropout and enrollment trends in the postwar period: What went wrong in the 1970s? In *Risky behavior among youths: An economic analysis*, pages 439–482. University of Chicago Press, 2001b.

Anne Case and Angus Deaton. Rising morbidity and mortality in midlife among white non-

Hispanic Americans in the 21st century. *Proceedings of the National Academy of Sciences*, 112 (49):15078–15083, 2015.

Anne Case and Angus Deaton. Mortality and morbidity in the 21st century. *Brookings Papers on Economic Activity*, 2017:397, 2017.

Anne Case and Christina Paxson. Stature and status: Height, ability, and labor market outcomes. *Journal of Political Economy*, 116(3):499–532, 2008.

Kenneth Chay, Jonathan Guryan, and Bhashkar Mazumder. Birth cohort and the black-white achievement gap: The roles of access and health soon after birth. *National Bureau of Economic Research, Working Paper*, 2009.

Tim J Cole and Hiroshi Mori. Fifty years of child height and weight in japan and south korea: Contrasting secular trend patterns analyzed by sitar. *American Journal of Human Biology*, 30 (1):e23054, 2018.

Tim J Cole, Malcolm DC Donaldson, and Yoav Ben-Shlomo. Sitar—a useful instrument for growth curve analysis. *International journal of epidemiology*, 39(6):1558–1566, 2010.

Timothy J Cole. Secular trends in growth. *Proceedings of the Nutrition Society*, 59(2):317–324, 2000.

T.J. Cole. The secular trend in human physical growth: a biological view. *Economics & Human Biology*, 1(2):161–168, 2003.

Dora Costa and Richard H Steckel. Long-term trends in health, welfare, and economic growth in the United States. In *Health and welfare during industrialization*, pages 47–90. University of Chicago Press, 1997.

Dora L Costa. Health and the economy in the United States from 1750 to the present. *Journal of Economic Literature*, 53(3):503, 2015.

Flavio Cunha and James J. Heckman. The technology of skill formation. *American Economic Review*, 97(2):31–47, 2007. doi: 10.1257/aer.97.2.31.

Janet Currie and Douglas Almond. Human capital development before age five. In *Handbook of Labor Economics*, volume 4, pages 1315–1486. Elsevier, 2011.

David Cutler, Angus Deaton, and Adriana Lleras-Muney. The determinants of mortality. *Journal of economic perspectives*, 20(3):97–120, 2006.

N Meltem Daysal, Hui Ding, Maya Rossin-Slater, and Hannes Schwandt. Germs in the family: The short-and long-term consequences of intra-household disease spread. Technical report, National Bureau of Economic Research, 2021.

Richard A Easterlin. *Birth and fortune: The impact of numbers on personal welfare*. University of Chicago Press, 1987.

Roderick Floud, Robert W Fogel, Bernard Harris, and Sok Chul Hong. *The changing body: health,*

nutrition, and human development in the western world since 1700. Cambridge University Press, 2011.

Robert W Fogel. Nutrition and the decline in mortality since 1700: some preliminary findings. In *Long-term factors in American economic growth*, pages 439–556. University of Chicago Press, 1986.

Robert W Fogel. *Explaining long-term trends in health and longevity*. Cambridge University Press, 2012.

Richard B Freeman. The effect of demographic factors on age-earnings profiles. *Journal of Human Resources*, 14(3):289–318, 1979.

Pei Gao and Eric B Schneider. The growth pattern of british children, 1850–1975. *The Economic History Review*, 74(2):341–371, 2021.

Peter D Gluckman and Mark A Hanson. The consequences of being born small–an adaptive perspective. *Hormone research*, 65(Suppl. 3):5–14, 2006a.

Peter D Gluckman and Mark A Hanson. Evolution, development and timing of puberty. *Trends in Endocrinology & Metabolism*, 17(1):7–12, 2006b.

Peter D Gluckman, Mark A Hanson, Hamish G Spencer, and Patrick Bateson. Environmental influences during development and their later consequences for health and disease: implications for the interpretation of empirical studies. *Proceedings of the Royal Society B: Biological Sciences*, 272(1564):671–677, 2005.

Claudia Goldin. America's graduation from high school: The evolution and spread of secondary schooling in the twentieth century. *The Journal of Economic History*, 58(2):345–374, 1998.

Claudia Goldin and Lawrence F. Katz. *The race between education and technology: The evolution of US educational wage differentials, 1890 to 2005*. National Bureau of Economic Research, 2007.

Claudia Goldin, Lawrence F. Katz, and Ilyana Kuziemko. The homecoming of American college women: The reversal of the college gender gap. *Journal of Economic Perspectives*, 20(4): 133–156, 2006.

Peter V Hamill, Terence A Drizd, Clifford L Johnson, Robert B Reed, and Alex F Roche. NCHS growth curves for children birth-18 years. Technical report, Department of Health Education and Welfare Washington DC, 1977.

Christopher Handy and Katharine L Shester. The baby boom and educational attainment. 2019.

Timothy J Hatton and Richard M Martin. The effects on stature of poverty, family size, and birth order: British children in the 1930s. *Oxford Economic Papers*, 62(1):157–184, 2010.

Russ Hauser, Oleg Sergeyev, Susan Korrick, Mary M Lee, Boris Revich, Elena Gitin, Jane S Burns,

and Paige L Williams. Association of blood lead levels with onset of puberty in russian boys. *Environmental health perspectives*, 116(7):976–980, 2008.

RC Hauspie, Martine Vercauteren, and Charles Susanne. Secular changes in growth and maturation: an update. *Acta Paediatrica*, 86(S423):20–27, 1997.

James J. Heckman and Paul A. LaFontaine. The American high school graduation rate: Trends and levels. *The Review of Economics and Statistics*, 92(2):244–262, 2010.

Michael Hermanussen, Beate Hermanussen, and Jens Burmeister. The association between birth order and adult stature. *Annals of human biology*, 15(2):161–165, 1988.

Alex Hollingsworth, Jiafang Mike Huang, Ivan Rudik, and Nicholas J Sanders. A thousand cuts: Cumulative lead exposure reduces academic achievement. *Journal of Human Resources*, pages 0222–12169R2, 2022.

Paul B. Kaplowitz. Link between body fat and the timing of puberty. *Pediatrics*, 121(Supplement 3):S208–S217, 2008.

Paul B Kaplowitz, Eric J Slora, Richard C Wasserman, Steven E Pedlow, and Marcia E Herman-Giddens. Earlier onset of puberty in girls: relation to increased body mass index and race. *Pediatrics*, 108(2):347–353, 2001.

Lawrence F. Katz and Kevin M. Murphy. Changes in relative wages, 1963–1987: Supply and demand factors. *The Quarterly Journal of Economics*, 107(1):35–78, 1992.

John Komlos. The recent decline in the height of African-American women. *Economics & Human Biology*, 8(1):58–66, 2010.

John Komlos and Benjamin E Lauderdale. The mysterious trend in American heights in the 20th century. *Annals of human biology*, 34(2):206–215, 2007a.

John Komlos and Benjamin E Lauderdale. Underperformance in affluence: the remarkable relative decline in US heights in the second half of the 20th century. *Social Science Quarterly*, 88(2): 283–305, 2007b.

Man Ki Kwok, Gabriel M Leung, and C Mary Schooling. Associations of birth order with early adolescent growth, pubertal onset, blood pressure and size: evidence from hong kong's "children of 1997" birth cohort. *PLoS One*, 11(4):e0153787, 2016.

BYH Lam, A Williamson, S Finer, FR Day, JA Tadross, A Gonçalves Soares, K Wade, P Sweeney, MN Bedenbaugh, DT Porter, et al. Mc3r links nutritional state to childhood growth and the timing of puberty. *Nature*, 599(7885):436–441, 2021.

Yun Liu, Martha M Téllez-Rojo, Brisa N Sánchez, Zhenzhen Zhang, Myriam C Afeiche, Adrianna Mercado-García, Howard Hu, John D Meeker, and Karen E Peterson. Early lead exposure and pubertal development in a mexico city population. *Environment international*, 125:445–451, 2019.

Adriana Lleras-Muney, Alessandro Tarozzi, Elisabetta Aurino, and Brendan Tinoco. The rise and fall of SES gradients in heights around the world. *Available at SSRN 4127021*, 2022.

Diane J Macunovich. *Birth quake: The baby boom and its aftershocks*. University of Chicago Press, 2002.

Michael J McFarland, Matt E Hauer, and Aaron Reuben. Half of US population exposed to adverse lead levels in early childhood. *Proceedings of the National Academy of Sciences*, 119(11): e2118631119, 2022.

Anthony J McMichael, Graham V Vimpani, Evelyn F Robertson, Peter A Baghurst, and Peter D Clark. The Port Pirie cohort study: maternal blood lead and pregnancy outcome. *Journal of Epidemiology & Community Health*, 40(1):18–25, 1986.

Howard V Meredith. Change in the stature and body weight of North American boys during the last 80 years. In *Advances in child development and behavior*, volume 1, pages 69–114. Elsevier, 1964.

Mikko Myrskylä, Karri Silventoinen, Aline Jelenkovic, Per Tynelius, and Finn Rasmussen. The association between height and birth order: evidence from 652 518 swedish men. *J Epidemiol Community Health*, 67(7):571–577, 2013.

Nisha Naicker, Shane A Norris, Angela Mathee, Piet Becker, and Linda Richter. Lead exposure is associated with a delay in the onset of puberty in south african adolescent females: findings from the birth to twenty cohort. *Science of the total environment*, 408(21):4949–4954, 2010.

National Academies of Sciences, Engineering, and Medicine. *High and Rising Mortality Rates Among Working-Age Adults*. The National Academies Press, Washington, DC, 2021.

Herbert Needleman. Lead poisoning. *Annual Review of Medicine*, 55(1):209–222, 2004.

Gerald J Pruckner, Nicole Schneeweis, Thomas Schober, and Martina Zweimüller. Birth order, parental health investment, and health in childhood. *Journal of Health Economics*, 76:102426, 2021.

Nicholas Reynolds. The broad decline of health and human capital of Americans born after 1947. *Working paper*, 2023.

Nicholas Reynolds. Increased mortality of white americans and a decline in the health of cohorts born after world war ii. *Journal of Human Resources*, 2024.

Nicholas Reynolds. The broad decline in health and human capital of Americans born after 1947. *American Economic Review: Insights*, 7(2):141–159, 2025.

Steven Ruggles, Katie Genadek, Ronald Goeken, Josiah Grover, and Matthew Sobek. Integrated public use microdata series: Version 6.0 [dataset]. 2015.

Eric B Schneider. Children's growth in an adaptive framework: explaining the growth patterns of

american slaves and other historical populations. *The Economic History Review*, 70(1):3–29, 2017.

Eric B Schneider. Sample-selection biases and the historical growth pattern of children. *Social Science History*, 44(3):417–444, 2020.

Eric B Schneider. The determinants of child stunting and shifts in the growth pattern of children: A long-run, global review. *Journal of Economic Surveys*, 2023.

Sherry G Selevan, Deborah C Rice, Karen A Hogan, Susan Y Euling, Andrea Pfahles-Hutchens, and James Bethel. Blood lead concentration and delayed puberty in girls. *New England Journal of Medicine*, 348(16):1527–1536, 2003.

Oleg Sergeyev, Jane S Burns, Paige L Williams, Susan A Korrick, Mary M Lee, Boris Revich, and Russ Hauser. The association of peripubertal serum concentrations of organochlorine chemicals and blood lead with growth and pubertal development in a longitudinal cohort of boys: a review of published results from the russian children's study. *Reviews on environmental health*, 32(1-2): 83–92, 2017.

James M Tanner. Growth as a target-seeking function: catch-up and catch-down growth in man. *Human Growth: A Comprehensive Treatise Volume 1 Developmental Biology Prenatal Growth*, pages 167–179, 1986.

James Mourilyan Tanner. *Foetus into man: Physical growth from conception to maturity*. Harvard University Press, 1990.

Jonathan CK Wells, Pedro C Hallal, Felipe F Reichert, Samuel C Dumith, Ana M Menezes, and Cesar G Victora. Associations of birth order with early growth and adolescent height, body composition, and blood pressure: prospective birth cohort from brazil. *American journal of epidemiology*, 174(9):1028–1035, 2011.

Tiejian Wu, Germaine M Buck, and Pauline Mendola. Blood lead levels and sexual maturation in US girls: the third national health and nutrition examination survey, 1988-1994. *Environmental health perspectives*, 111(5):737–741, 2003.

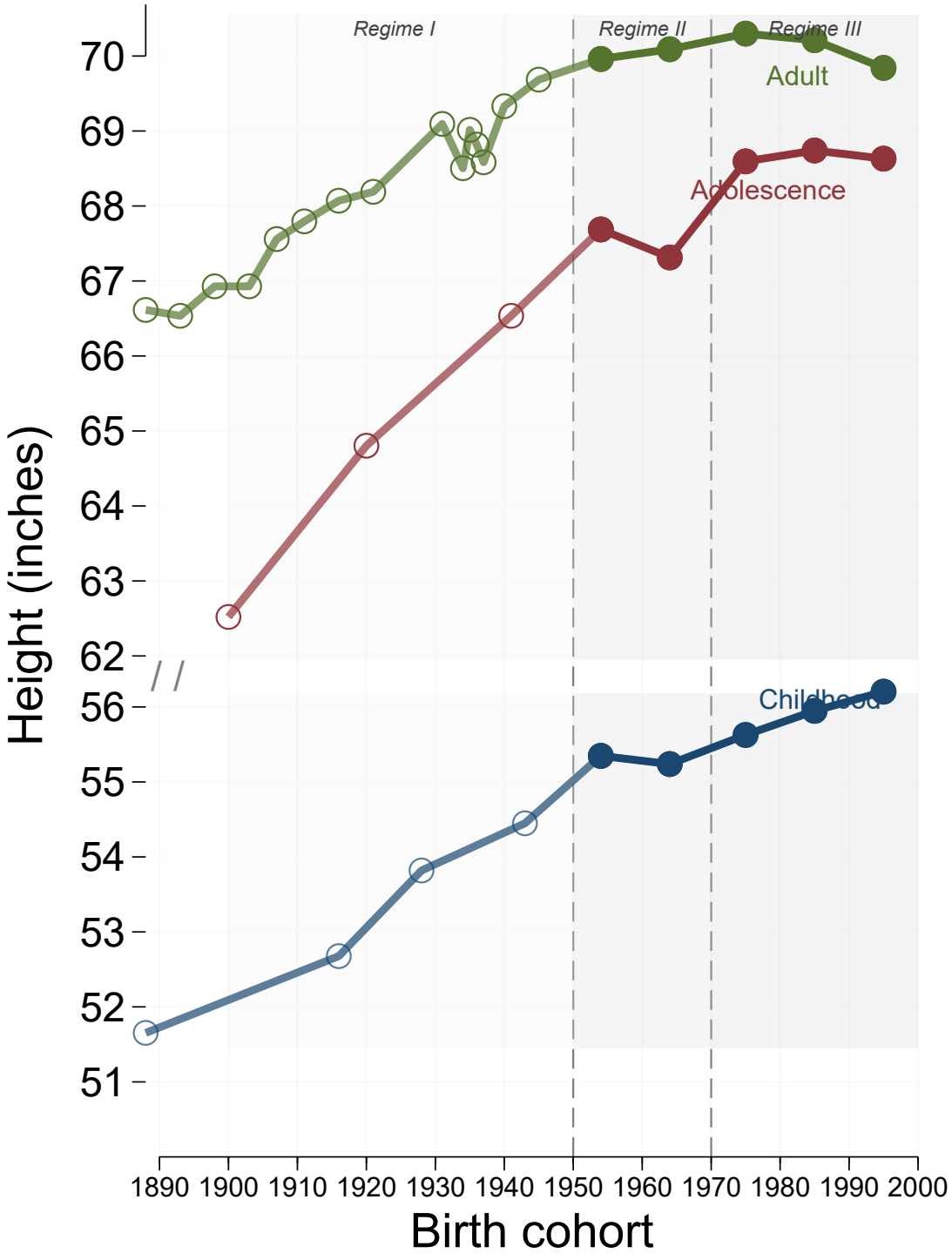
Grace Wyshak and Rose E Frisch. Evidence for a secular trend in age of menarche. *New England journal of medicine*, 306(17):1033–1035, 1982.

Table 1: Regime summary across growth, maturation, schooling, and survival

	Regime I (1890–1950)	Regime II (1950–1970)	Regime III (1970–1995)
A. Height (inches per decade)			
Childhood (ages 6–11)	0.62†	-0.10	0.28
		(0.11)	(0.05)
Adolescence (ages 12–17)	0.86†	-0.45	0.41
		(0.14)	(0.05)
Adult (ages 23–40)	0.51†	0.13	-0.08
		(0.19)	(0.06)
B. Menarche (years per decade)			
Females	-0.21	0.16	-0.22
	(0.01)	(0.03)	(0.04)
C. Schooling (years per decade)			
Males	0.56	-0.04	0.15
Females	0.47	0.28	0.24
D. Survival 25–54 (p.p. per decade)			
Both sexes	1.44	0.46	—

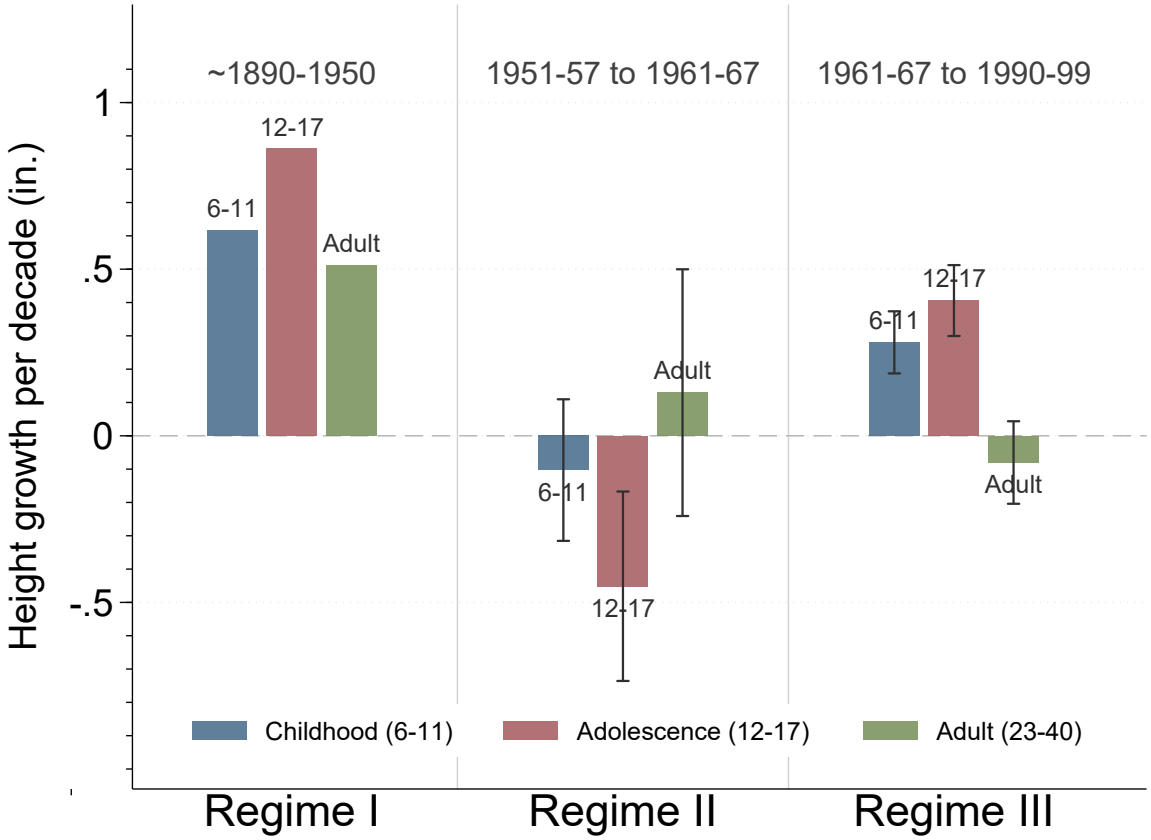
Note: Entries report change per decade within each regime. Regime boundaries are shared approximately across outcomes (1950 and 1970); when an outcome does not span a full regime, the change is computed over the available years only.
Panel A covers white non-Hispanic native-born males. Regime 1 uses Meredith (1964) and Costa (2015) and has no standard errors (†); Regimes 2–3 use NHES/NHANES survey-weighted means, with delta-method standard errors in parentheses.
Panel B reports slopes from a two-knot piecewise regression in retrospective menarche data. Panel C uses Census/ACS IPUMS means for native-born adults ages 35–55, shown separately for males and females. Panel D uses Human Mortality Database cohort life tables for survival from ages 25 to 54; Regime III may be blank where cohort coverage is incomplete.
Estimator note: Height rates (Panel A) are computed as cohort-group differences divided by regime duration. Education and mortality rates (Panels C–D) are linear regression slopes over the full regime window. These estimators use different approaches and are not directly comparable as per-decade rates.

Figure 1: Three regimes of height: long-run trends for white non-Hispanic males, ~1890–1990s birth cohorts



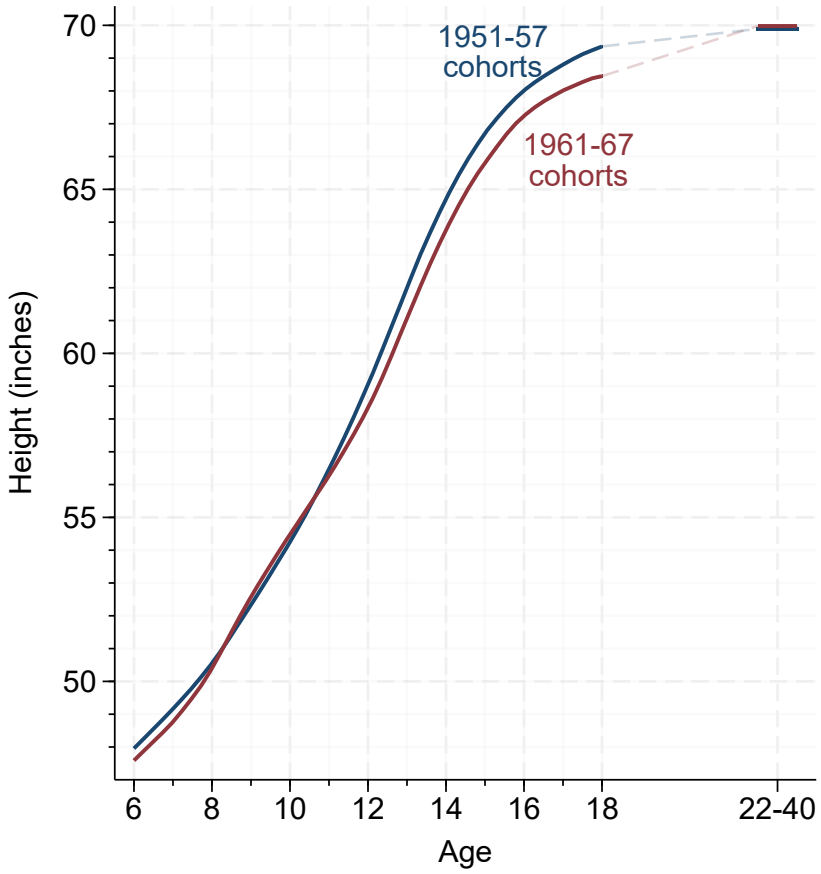
Bottom panel: height at age 10. Top panel: height at age 15 and adult height. Background shading marks the three regimes; dashed vertical boundaries at ~1950 and ~1970. The two panels share a common physical scale (one inch on the y-axis is the same physical length in both panels). Historical data from Meredith (1964) and Costa (2015); modern data from NHES/NHANES survey-weighted predicted heights.

Figure 2: Adolescent growth stalls in Regime II, then shifts again in Regime III



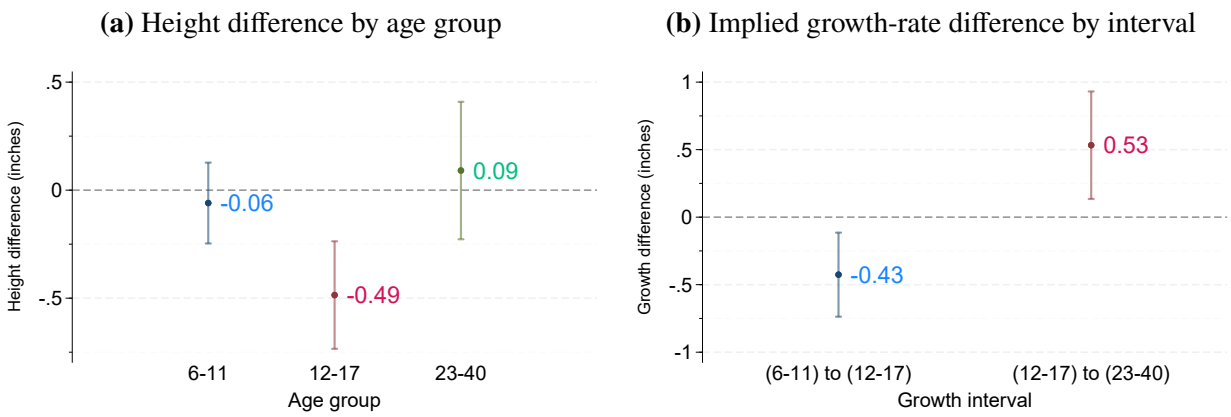
White non-Hispanic native-born males. Height scaling windows: R1 ~1890–1950 (6.0 decades), R2 compares 1951–57 to 1961–67 (1.0 decade), and R3 compares 1961–67 to 1990–99 (3.1 decades). Regime 1 estimates from Meredith (1964) and Costa (2015); no standard errors available (†). Regimes 2–3: NHES/NHANES survey-weighted means divided by the empirical comparison window; error bars are 95% CIs.

Figure 3: Late Regime II cohorts fall behind in adolescence but largely catch up by adulthood



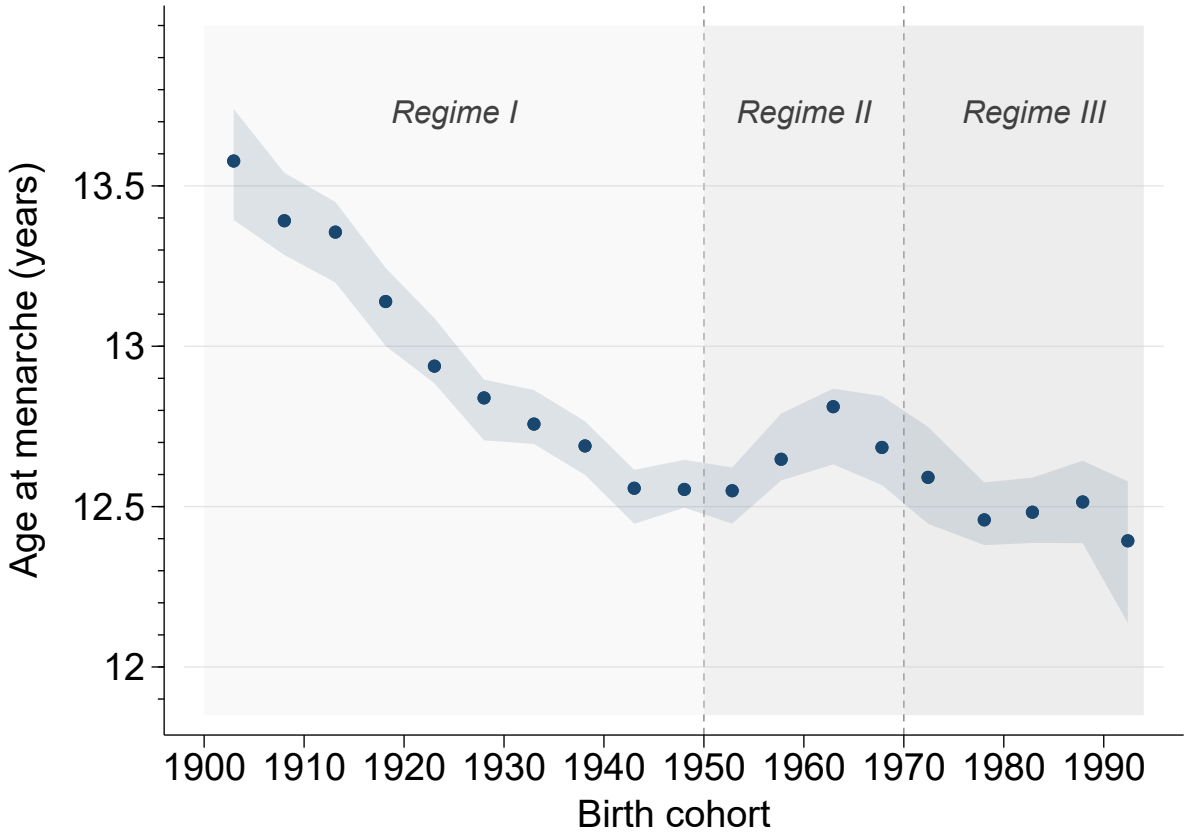
Smoothed height-by-age profiles for early-Regime II (1951–57) and late-Regime II (1961–67) cohorts of American-born males, estimated by kernel smoother (Epanechnikov kernel, 12-month bandwidth). Lines at age 22–40 report the pooled mean. Data from NHES II–III and NHANES I–II.

Figure 4: Late Regime II cohorts are shorter in adolescence and then exhibit catch-up growth



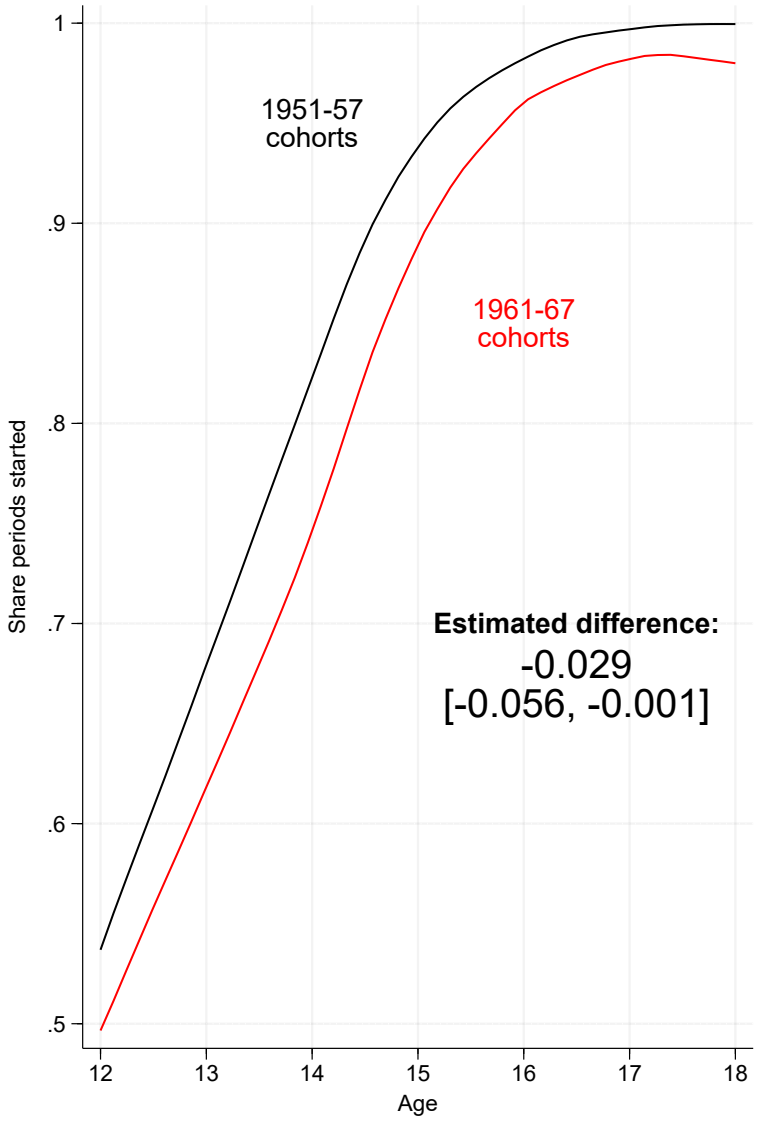
Regression estimates comparing early-Regime II (1951–57) and late-Regime II (1961–67) cohorts of American-born males. Panel (a): height difference by age. Panel (b): implied growth rate differences by age. Controls for ages 6–17 include single-year-of-age fixed effects and a quadratic in age-in-months. Data from NHES II–III and NHANES I–II.

Figure 5: Menarche timing follows the same three-regime cohort pattern, ~1905–1992



Retrospective self-reports of age at menarche for native-born women aged 20–75. Cohort means are summarized with 9 bins aligned to the regime boundaries and 95% confidence bands. Background shading marks the three regimes. Data pooled across NHES II–III, NHANES I–III, and Continuous NHANES 1999–2023.

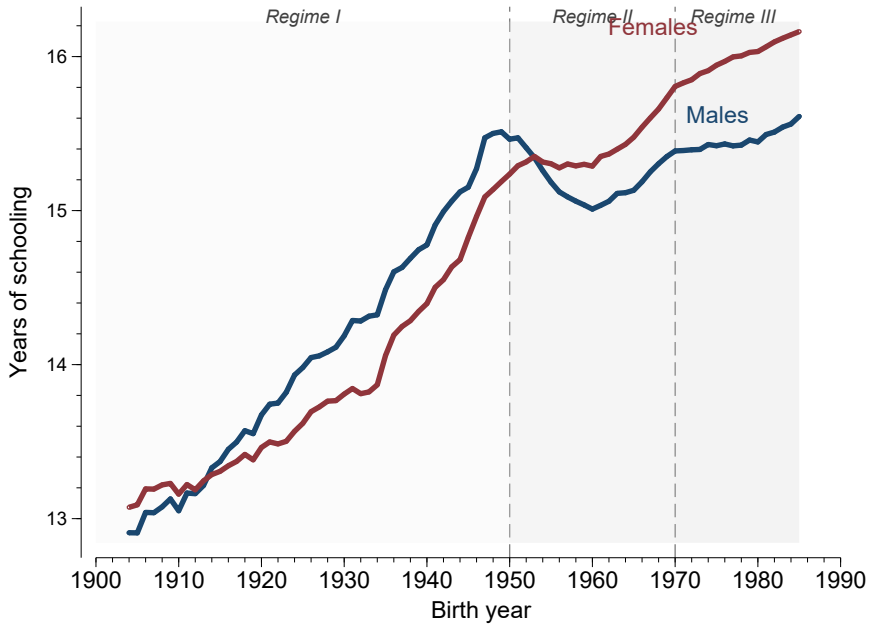
Figure 6: Regime II: share of American-born girls reporting menarche by age, contemporaneous reports (1951–57 vs. 1961–67 cohorts)



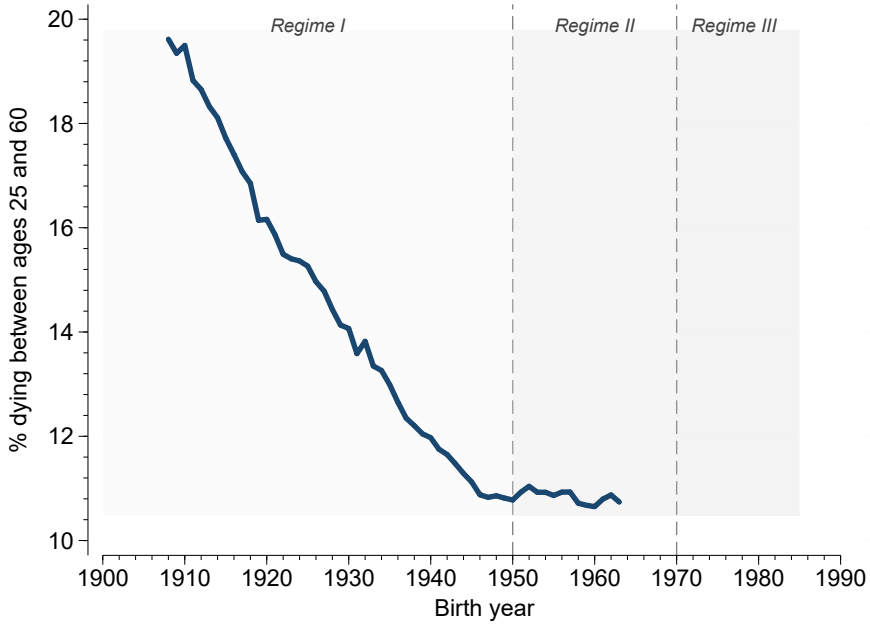
Kernel smoother (Epanechnikov kernel, 12-month bandwidth) applied separately to the Regime I (1951–57) and Regime II (1961–67) cohorts of American-born females. Data from NHES II–III and NHANES I–II.

Figure 7: Educational attainment and adult mortality move in parallel with the growth regimes

(a) Educational attainment by birth cohort

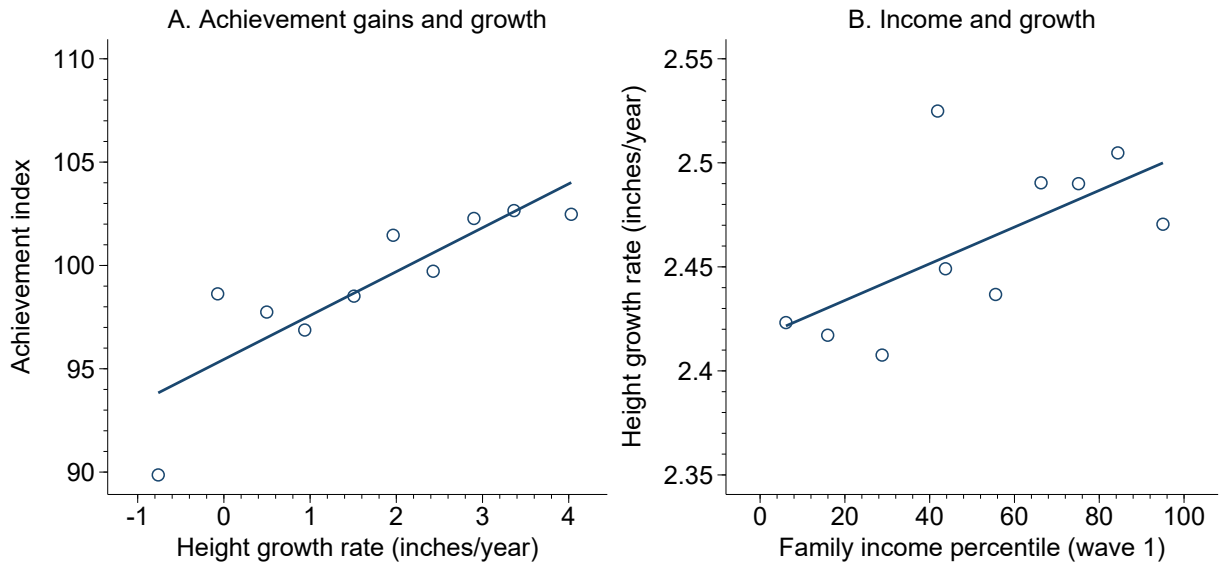


(b) Adult mortality by birth cohort



Panel (a): mean years of schooling by birth cohort from Census/ACS microdata (IPUMS), all native-born males aged 35–55 at time of survey. Panel (b): probability of dying between ages 25 and 60 by birth cohort, both sexes combined; Human Mortality Database (HMD), USA cohort 1×1 life tables. Both panels use the same birth-cohort axis; background shading marks the three regimes and dashed vertical boundaries indicate 1950 and 1970.

Figure 8: Faster childhood-to-adolescent growth is associated with larger test score gains and higher family income



Linked NHES II–III panel ($N \approx 2{,}065$), both sexes pooled. Panel (A) relates achievement gains to childhood-to-adolescent height growth; Panel (B) relates family income percentile at wave 1 to childhood-to-adolescent height growth. In both panels the binned points partial out sex-specific quadratics in age at both waves and wave-1 height percentile. Raw Achievement Index scores have mean ≈ 100 and SD ≈ 15. Growth rate is measured in inches per year and winsorized at the 99th percentile.

Table 2: NHES Panel: Growth Rate and Test Score Achievement

	(1)	(2)
Growth rate (in/year)	1.721***	1.255**
	(0.657)	(0.605)
Height percentile, wave 1	0.082***	0.060***
	(0.012)	(0.011)
Income percentile, wave 1		0.186***
		(0.012)
Observations	2,018	2,018
R^2	0.045	0.168

Note: Growth rate = height gain (inches) / years between NHES waves, winsorized at 99th pctile (4.19 in/year). Outcome: Achievement Index at wave 2 (IQ-scaled composite, mean ≈ 100, SD ≈ 15). All columns include both-wave age quadratics and wave-1 height percentile. Col 1: pooled baseline specification. Col 2: adds wave-1 income percentile. By-sex regressions are reported in Appendix Table A2. No lagged test score control in any column. Weights: NHES panel weights. Native-born children only.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Appendix

Table A1: Sample sizes by birth cohort and age group, males with recorded height

	Age Group		
Birth Cohort	**6-11**	**12-17**	**22-40**
1951-1957	3045	3337	1339
1961-1967	1186	863	1235
1970-1979	565	807	2282
1980-1989	1338	2066	1979
1990-1999	2182	2628	530

Reports the number of male observations with a recorded height, by age group and birth cohort group from the combined sample based on multiple rounds of NHES and NHANES surveys.

Table A2: NHES Panel: Growth Rate and Test Score Achievement, by Sex

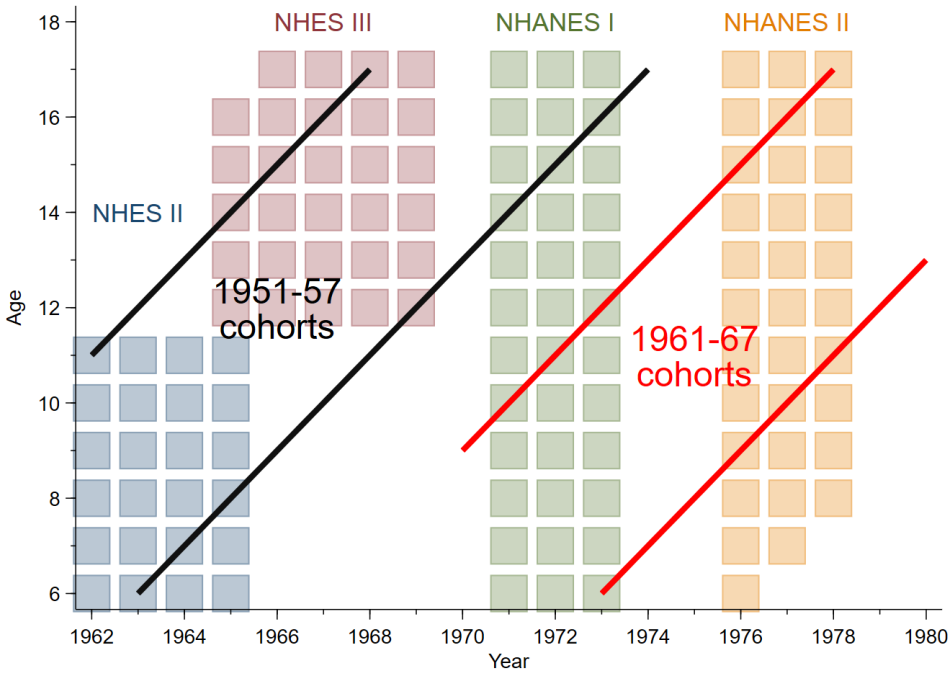
	Boys	Girls
Growth rate (in/year)	1.011	2.698***
	(0.918)	(0.988)
Height percentile, wave 1	0.082***	0.090***
	(0.016)	(0.018)
Income percentile, wave 1		
Observations	1,059	959
R^2	0.038	0.054

Note: Same outcome, sample restrictions, weighting, and controls as Table 2, estimated separately for boys and girls. Both columns include both-wave age quadratics and wave-1 height percentile. No lagged test score control. Growth rate winsorized at the 99th percentile (4.19 in/year). Native-born children only.

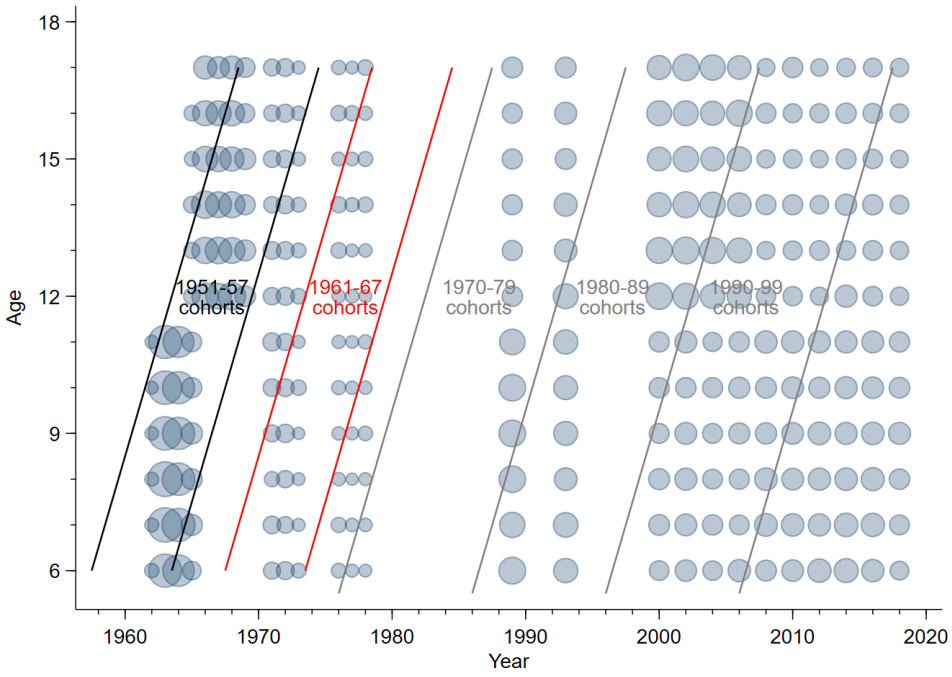
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Figure A1: Lexis diagram of NHES and NHANES coverage of children by age, year, birth cohort

(a) NHES II-III, NHANES I-II cover 1951-57 and 1961-67 cohorts



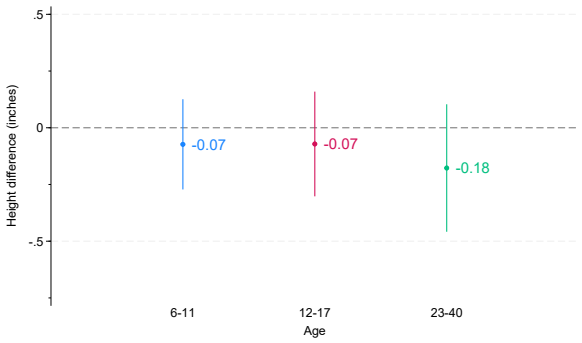
(b) Later NHANES cover additional cohorts



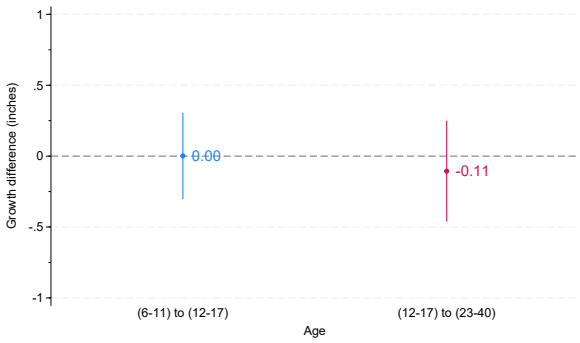
Shows in which years and at which ages, between 6 and 17, were sampled in various NHES and NHANES surveys. In panel A, each square shows an age-year cell with a sample size of at least 50 in the listed survey. In panel B circles are included if the relevant age-year cell has sample size of at least 50, and the size of the circles are proportional to the sample size.

Figure A2: Regression estimates of height and growth differences, American-born females 1951-1957 vs. 1961-67 cohorts

(a) Height difference

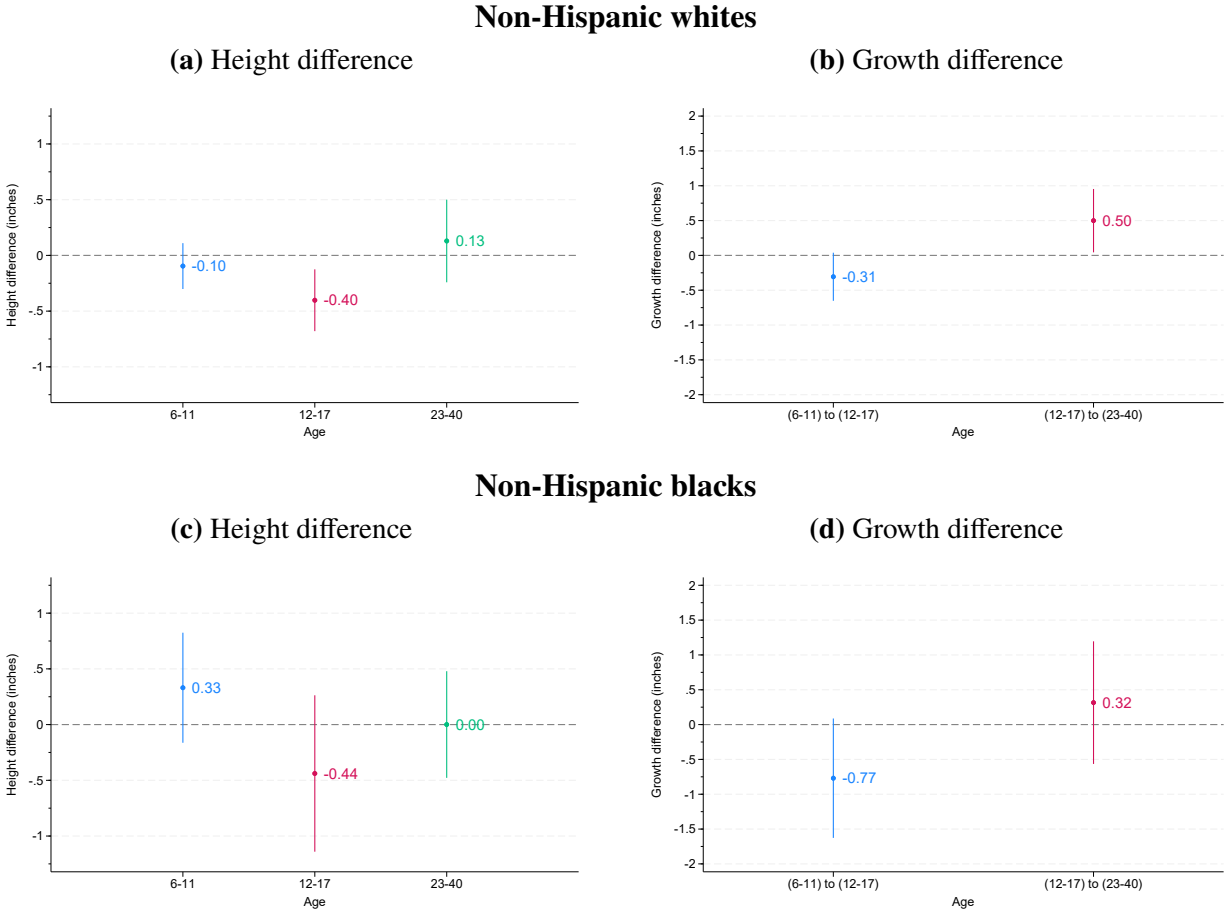


(b) Growth difference



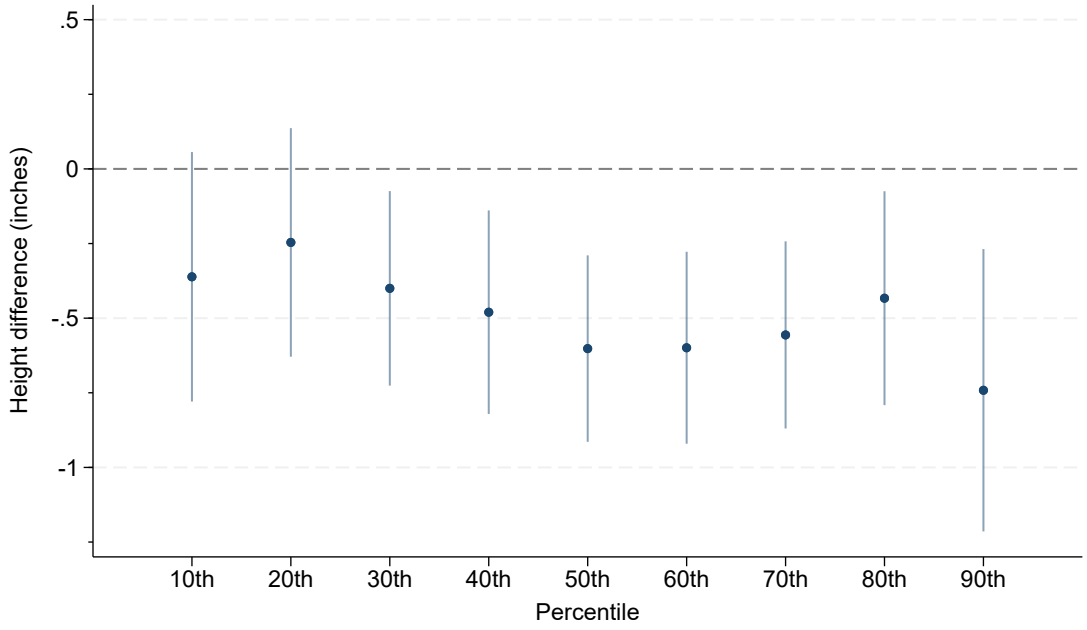
All panels use data from multiple rounds of NHES and NHANES surveys. Panel A shows estimates of the height differences between the listed cohorts at different ages. Panel B shows the estimates of the difference between the listed cohorts in implied growth at different ages. All estimates comes from regression described in Section 2 which, for thoses ages 6 to 17, control for fixed-effects for single-year-of-age and a quadratic in age-in-months.

Figure A3: Regression estimates of height and growth differences, American-born males by race, 1951–1957 vs. 1961–67 cohorts



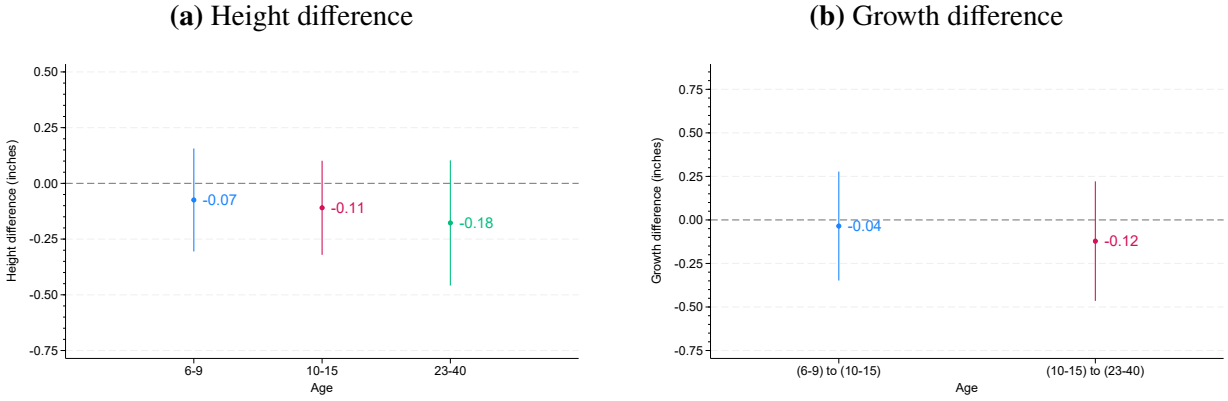
All panels use data from multiple rounds of NHES and NHANES surveys. Panels A and C show estimates of the height difference of the listed cohorts at different ages. Panel B and D show estimates of the difference between the listed cohorts in implied growth between different ages. All estimates comes from regression described in Section 2 which, for those ages 6 to 17, control for fixed-effects for single-year-of-age and a quadratic in age-in-months.

Figure A4: Quantile regression estimates of adolescent height differences, American-born males, 1951–1957 vs. 1961–67 cohorts



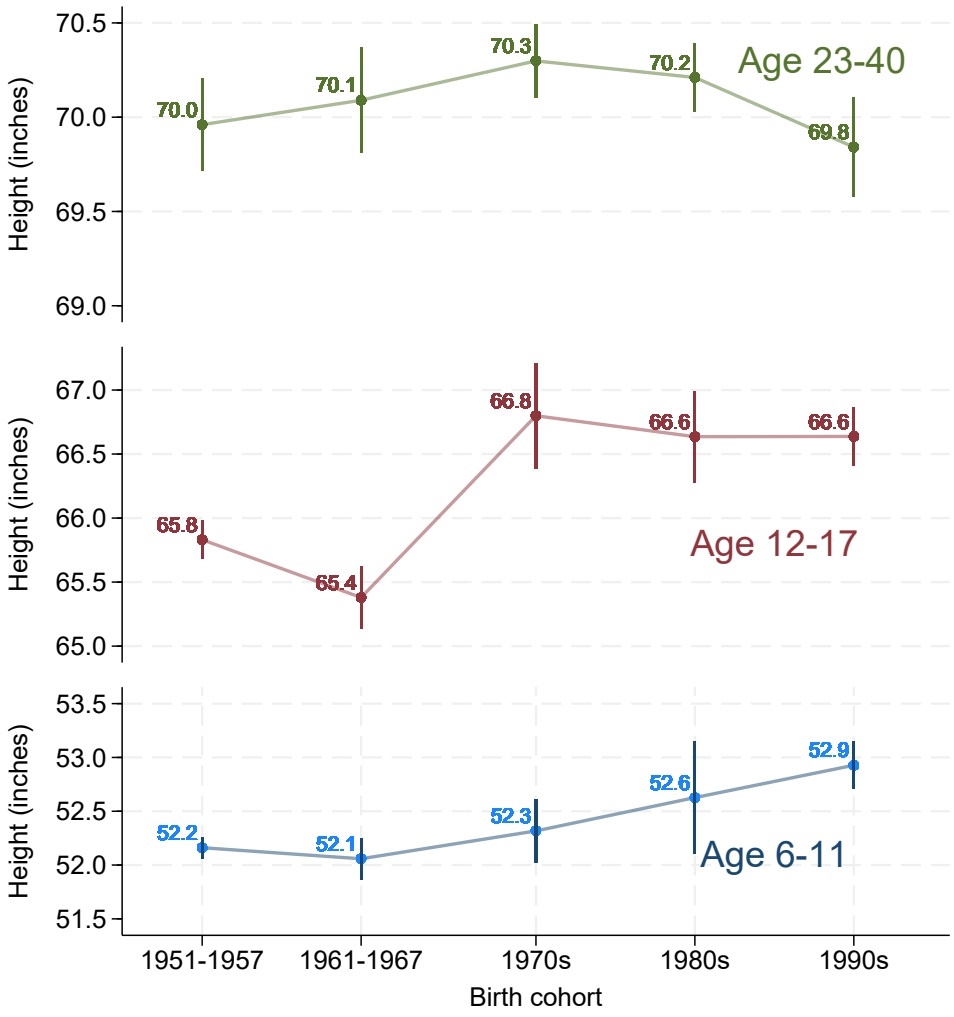
All panels use data from multiple rounds of NHES and NHANES surveys. Each point show estimates of the height difference of the listed cohorts at ages 12 to 17, at the listed percentile. Each estimate comes from a separate quantile regression, following the specification described in Section 2 which controls for fixed-effects for single-year-of-age and a quadratic in age-in-months.

Figure A5: Alternate age categories, regression estimates of height and growth differences, American-born females, 1951–1957 vs. 1961–67 cohorts



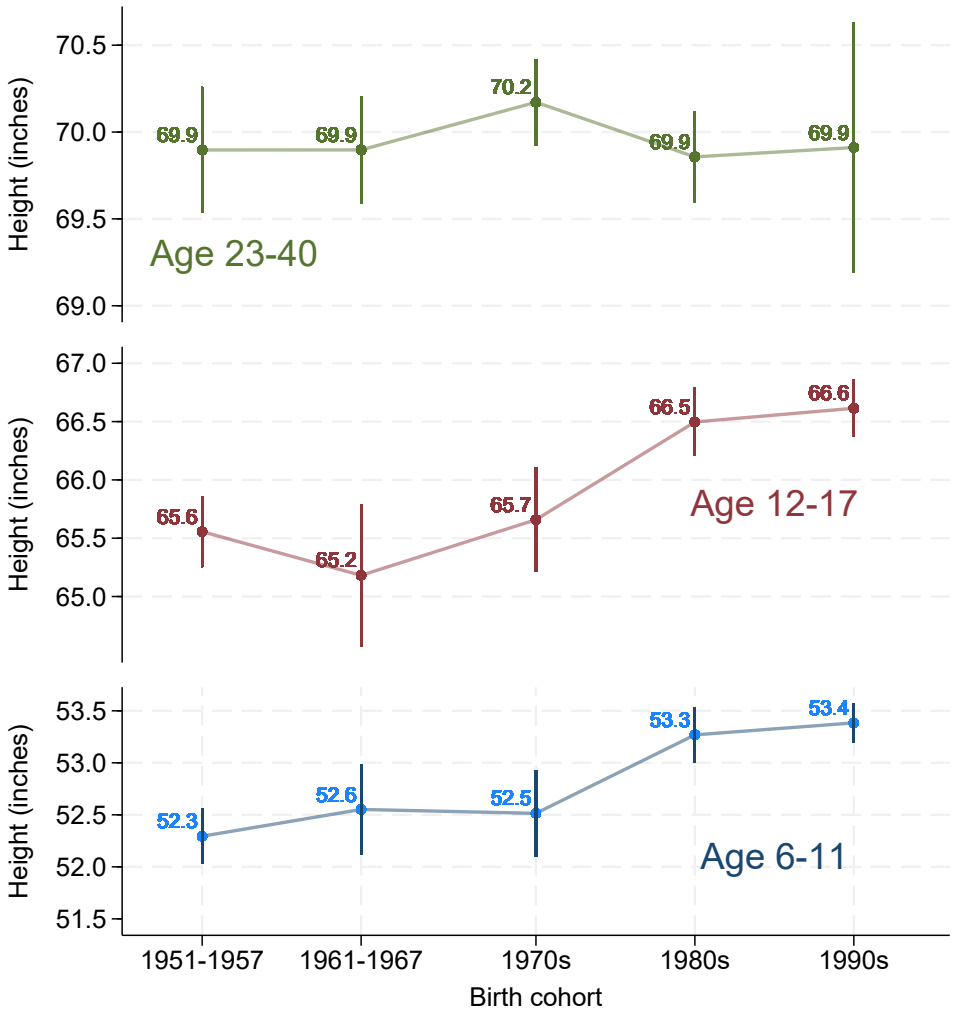
All panels use data from multiple rounds of NHES and NHANES surveys. Panel A show estimates of the height difference of the listed cohorts at different ages. Panel B shows estimates of the difference between the listed cohorts in implied growth between different ages. All estimates comes from regression described in Section 2 which, for those ages 6 to 17, control for fixed-effects for single-year-of-age and a quadratic in age-in-months.

Figure A6: Cross-cohort trends in height of non-Hispanic white males born in the US after 1950, by age



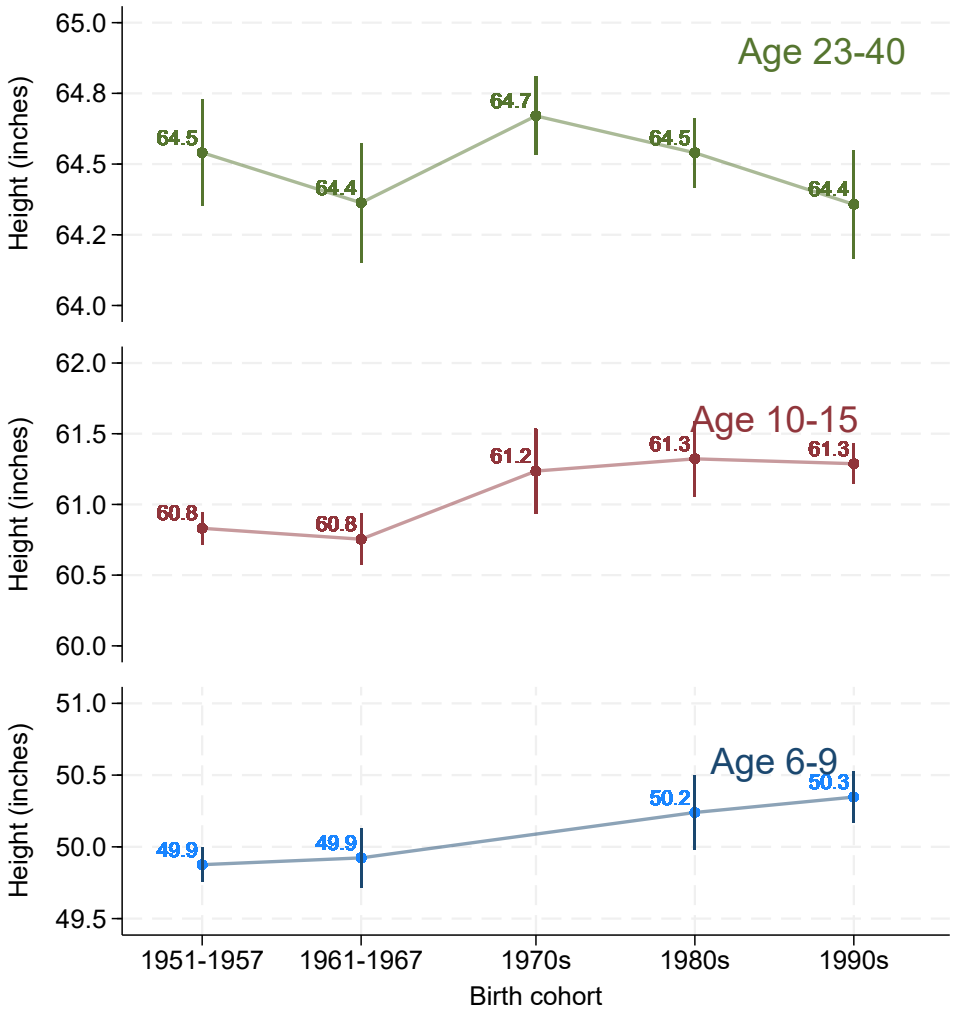
Uses data from multiple rounds of NHES and NHANES surveys. For each listed cohort group I first calculate means by single-year-of-age. For each listed age-group, I then take the simple average of these means across single-year ages. These age-group means are plotted for each listed cohort group. 95 percent confidence intervals for the age-group means are also shown. They were calculated by the delta method and take into account the sampling weights and survey design.

Figure A7: Cross-cohort trends in height of non-Hispanic black males born in the US after 1950, by age



Uses data from multiple rounds of NHES and NHANES surveys. For each listed cohort group I first calculate means by single-year-of-age. For each listed age-group, I then take the simple average of these means across single-year ages. These age-group means are plotted for each listed cohort group. 95 percent confidence intervals for the age-group means are also shown. They were calculated by the delta method and take into account the sampling weights and survey design.

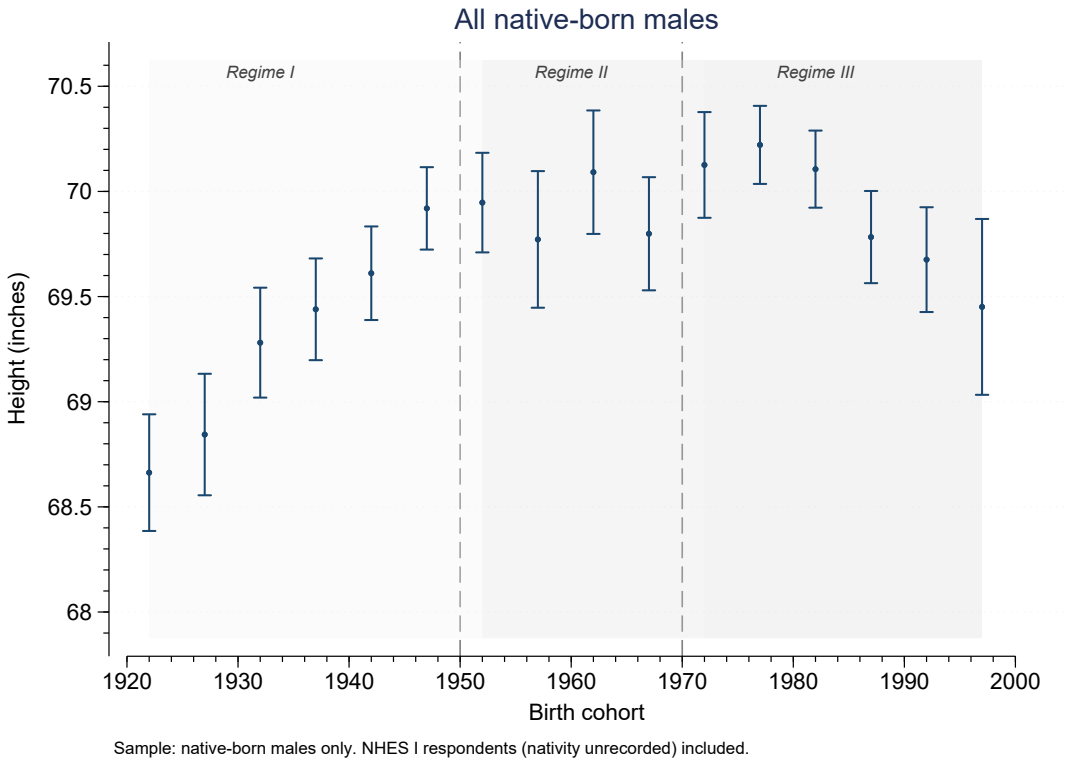
Figure A8: Cross-cohort trends in height of females born in the US after 1950, by age



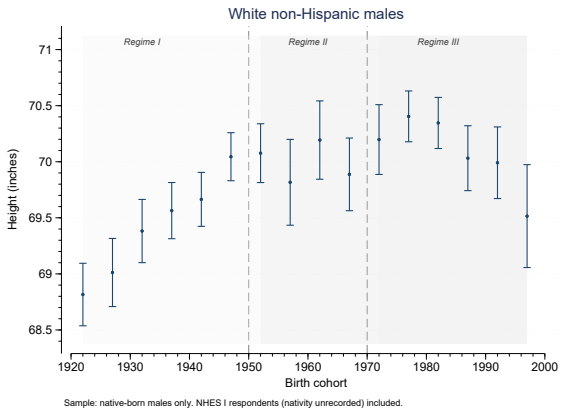
Uses data from multiple rounds of NHES and NHANES surveys. For each listed cohort group I first calculate means by single-year-of-age. For each listed age-group, I then take the simple average of these means across single-year ages. These age-group means are plotted for each listed cohort group. 95 percent confidence intervals for the age-group means are also shown. They were calculated by the delta method and take into account the sampling weights and survey design.

Figure A9: Long-run trends in adult height using only NHES/NHANES data

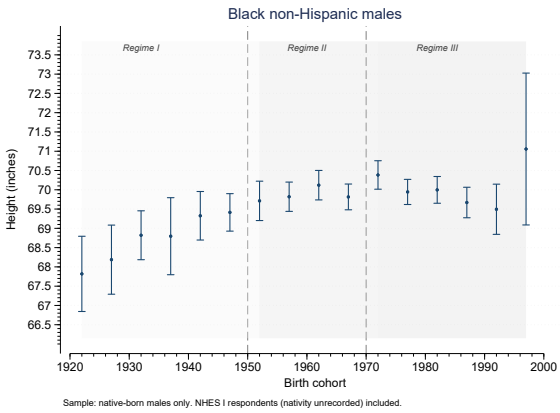
(a) All native-born males



(b) White non-Hispanic males

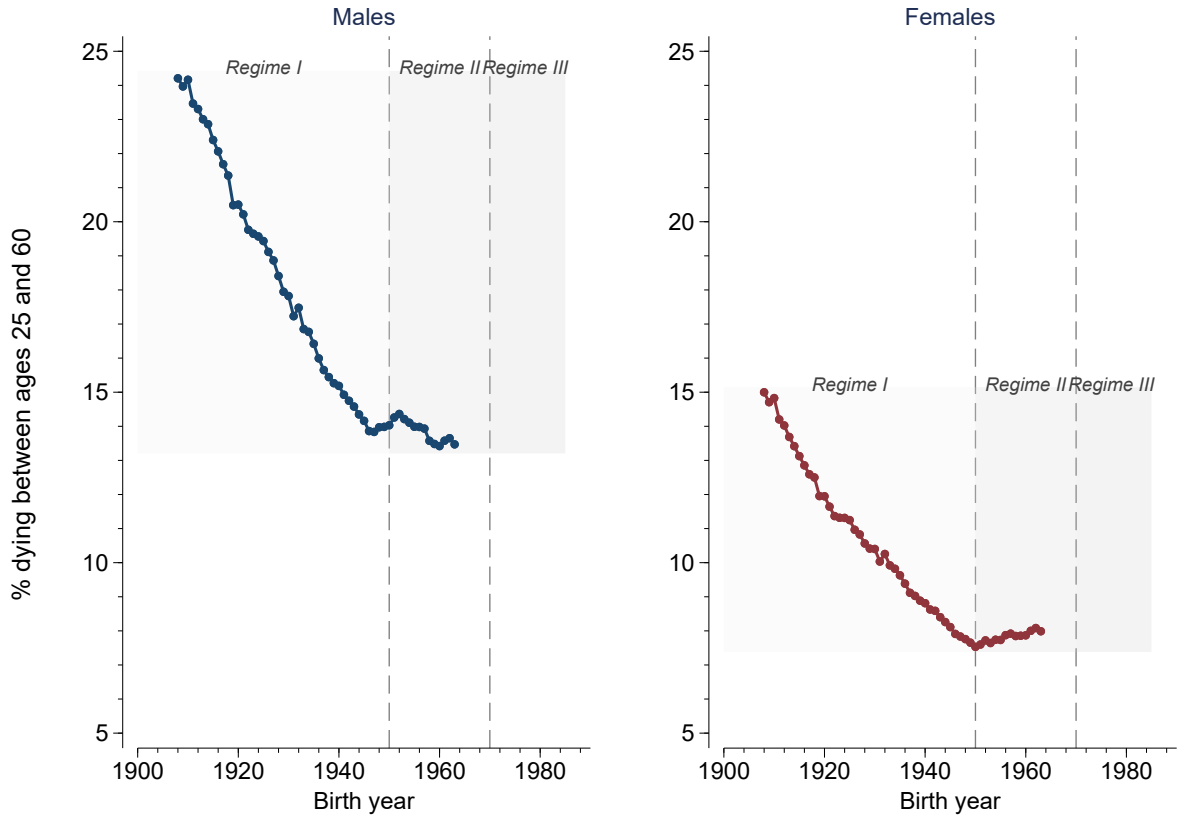


(c) Black non-Hispanic males



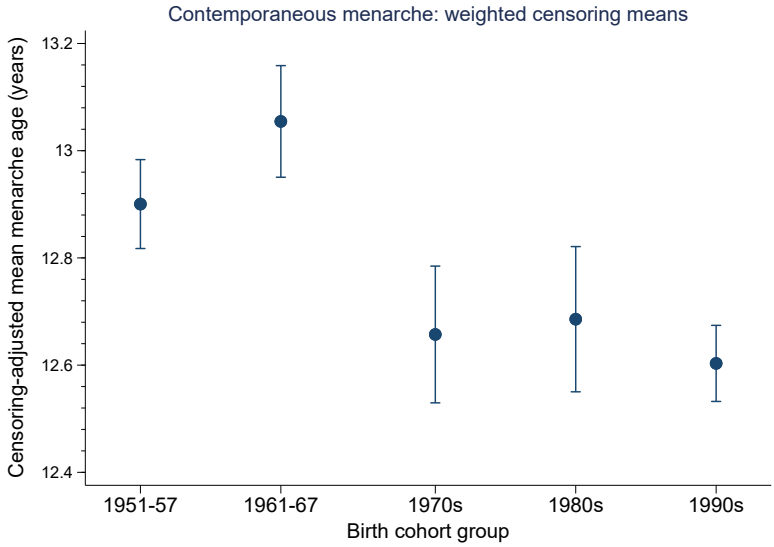
Points show survey-weighted mean adult height (ages 23–40) in 5-year birth-cohort bins, with 95% confidence intervals, using NHES I–III, NHANES I–III, and Continuous NHANES 1999–2023. Panel (a) uses all native-born males and retains NHES I observations with unknown nativity. Panels (b) and (c) restrict to white non-Hispanic and Black non-Hispanic males and likewise retain NHES I observations with unknown nativity. Dashed vertical lines mark 1950 and 1970.

Figure A10: Adult mortality by birth cohort, by sex



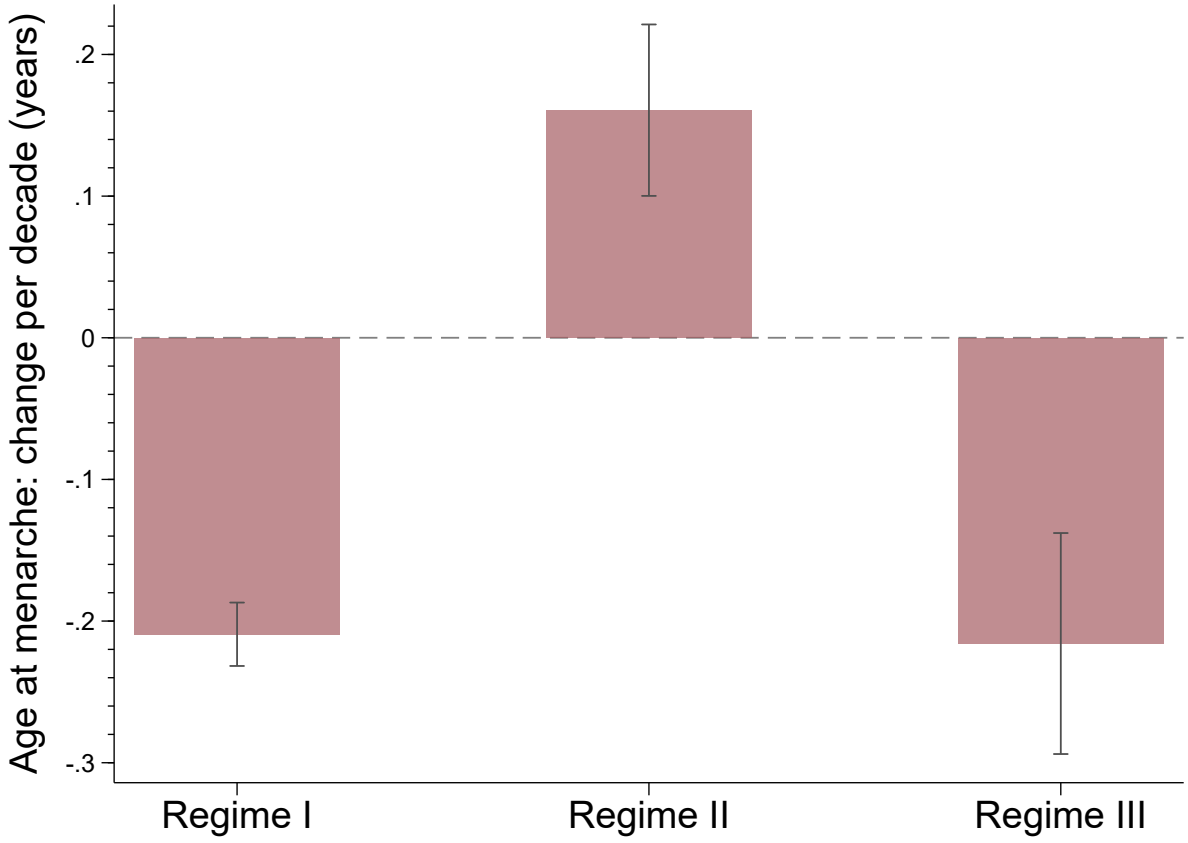
Probability of dying between ages 25 and 60 (conditional on surviving to age 25) by birth cohort, separately for males (left) and females (right). Human Mortality Database (HMD), USA cohort 1×1 life tables. q_x computed from central death rates under the uniform distribution of deaths assumption ($a_x = 0.5$). Background shading marks the three regimes; dashed vertical boundaries at 1950 and 1970.

Figure A11: Contemporaneous menarche timing by cohort group: censoring-adjusted weighted means



Girls ages 10–17, native-born (including NHES I unknown nativity code), pooled across NHES/NHANES. Each point is a cohort-specific censoring-adjusted mean age at menarche from an intercept-only interval-regression model with probability weights, where non-starters are right-censored at interview age, starters with reported timing are interval-censored on reported year, and starters without reported timing are left-censored at interview age.

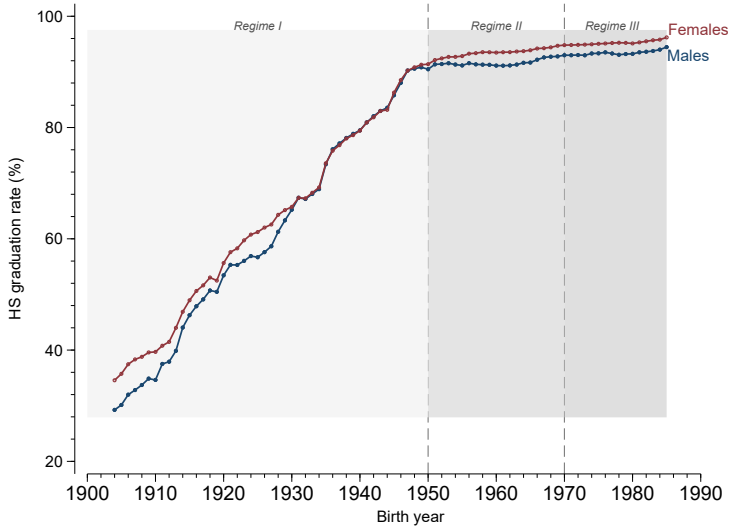
Figure A12: Age at menarche change per decade within each regime



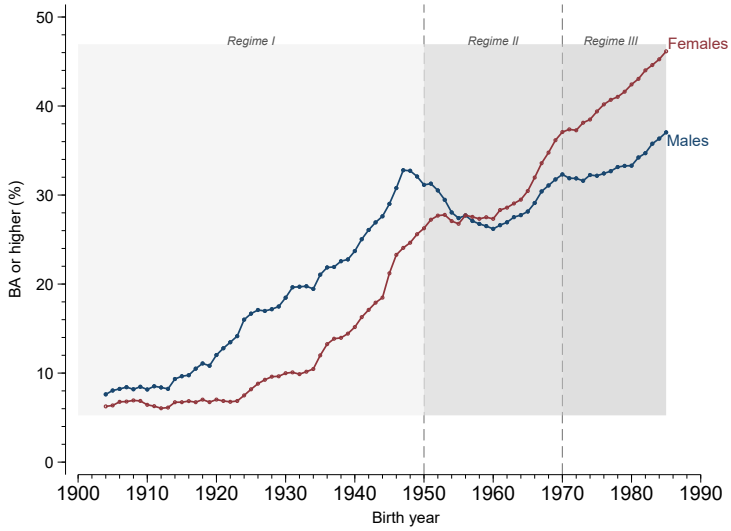
Change in age at menarche per decade by regime. R2 (1951–1970, 2.0 decades) and R3 (1970–1985, 1.5 decades) estimates from piecewise linear regression with breaks at 1950 and 1970; retrospective menarche reports from native-born women, pooled NHES II–III, NHANES I–III, and Continuous NHANES 1999–2023. Positive values indicate later menarche; negative values indicate earlier menarche.

Figure A13: Schooling outcomes by birth cohort: IPUMS series

(a) IPUMS high school

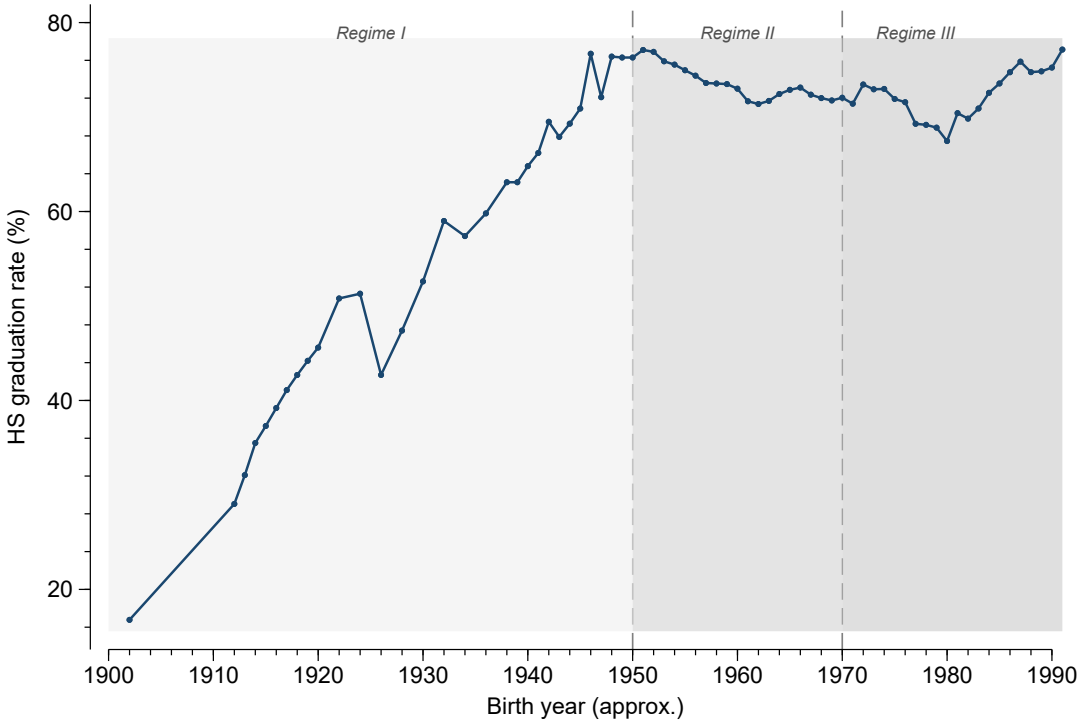


(b) Bachelor's degree



Panel (a): Share of all native-born males completing high school (grade 12, diploma, or GED), by birth cohort. Census/ACS microdata (IPUMS), men aged 35–55 at time of survey. *Panel (b):* Share of all native-born males completing a bachelor's degree or higher, by birth cohort, from Census/ACS IPUMS. Background shading marks the three regimes; dashed vertical boundaries at 1950 and 1970.

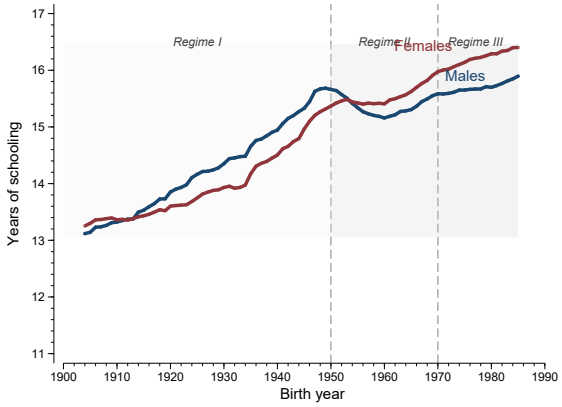
Figure A14: High school graduation by birth cohort: NCES administrative series



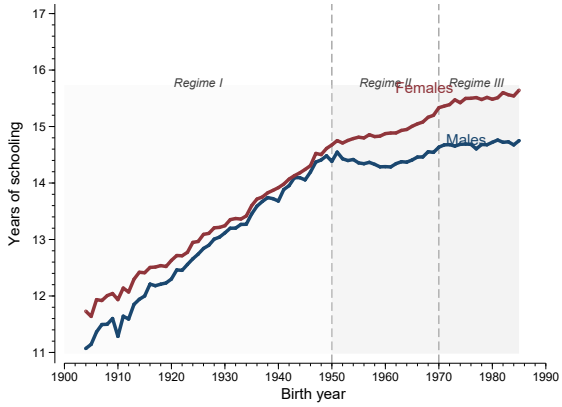
NCES administrative graduation rate (graduates as share of 17-year-olds), plotted by approximate birth cohort (graduation year − 18). Source: NCES Digest of Education Statistics, Table 103. Background shading marks the three regimes; dashed vertical boundaries at 1950 and 1970.

Figure A15: Years of schooling by birth cohort, by race

(a) White non-Hispanic



(b) Black non-Hispanic

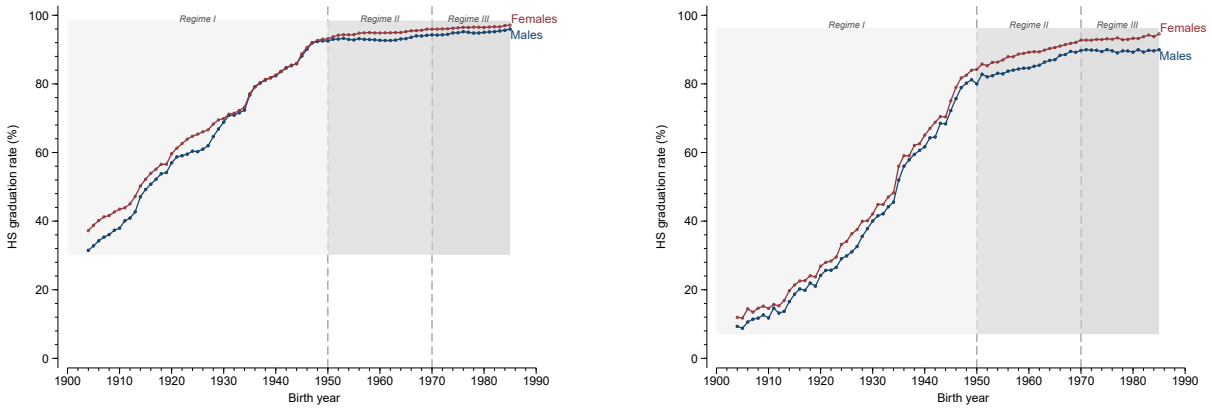


Mean years of schooling by birth cohort, separately for white non-Hispanic and Black non-Hispanic native-born males, aged 35–55 at time of survey. Census/ACS microdata (IPUMS). Background shading marks the three regimes; dashed vertical boundaries at 1950 and 1970.

Figure A16: High school graduation rate by birth cohort, by race

(a) White non-Hispanic

(b) Black non-Hispanic

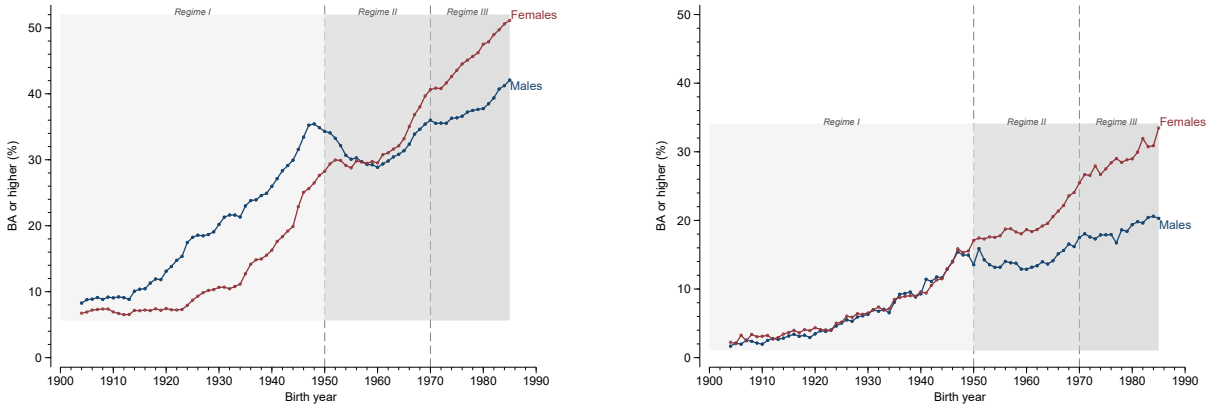


Share completing high school (grade 12, diploma, or GED) by birth cohort, separately for white non-Hispanic and Black non-Hispanic native-born males, aged 35–55 at time of survey. Census/ACS microdata (IPUMS). Background shading marks the three regimes; dashed vertical boundaries at 1950 and 1970.

Figure A17: Bachelor's degree or higher by birth cohort, by race

(a) White non-Hispanic

(b) Black non-Hispanic



Share completing a bachelor's degree or higher by birth cohort, separately for white non-Hispanic and Black non-Hispanic native-born males, aged 35–55 at time of survey. Census/ACS microdata (IPUMS). Background shading marks the three regimes; dashed vertical boundaries at 1950 and 1970.