Three Regimes of Human Development in Twentieth-Century America

Biological Growth, Health, and Human Capital

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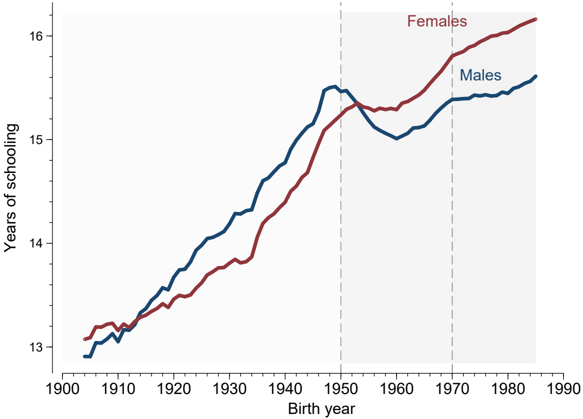
The stalling of the US human capital machine



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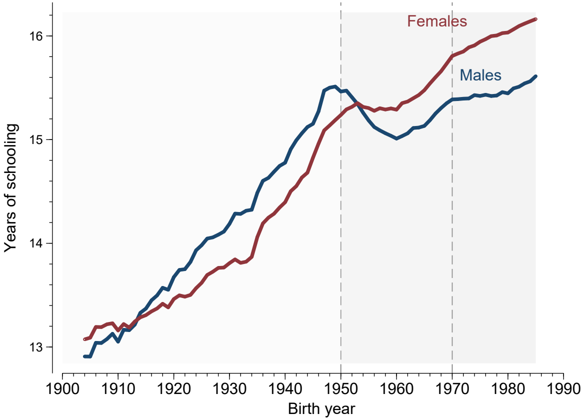

"the **human capital century**"

— Goldin & Katz (2007)

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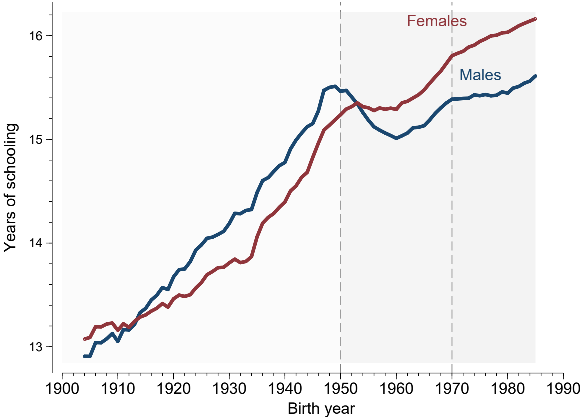

"the **human capital century**"

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"no **adequate account** of what went wrong with the U.S. human capital machine"

— Acemoglu & Autor (2012)

Prior work — part of a broader decline (Reynolds 2025)

Cohorts born after ∼1947:

1. poor educational outcomes in teens and early-20s → educational declines in 1960s and 1970s
2. lower earnings for men → earnings stagnation since the 1970s
3. give birth to less healthy infants in 20s and 30s → increases in LBW in 1980s
4. greater likelihood of dying prematurely → post-1999 mid-life mortality increases of Whites

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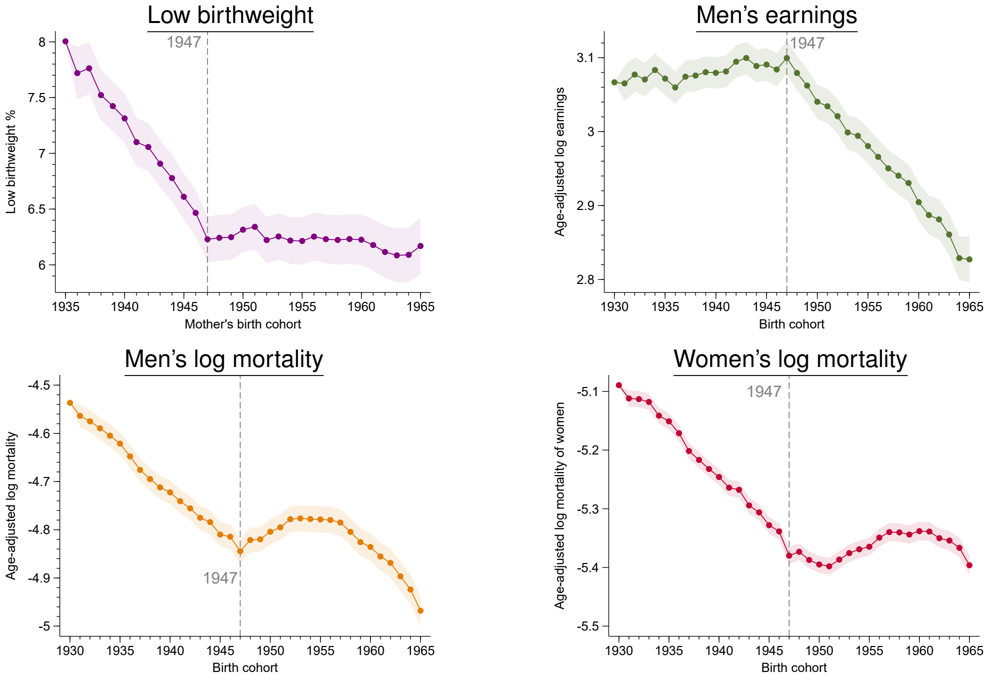
Unifies previously independent searches for cause of these 4 declines

→ What went wrong for cohorts born after ∼1947?



Low birthweight
Low birthweight %
1947
Mother's birth cohort
Men's earnings
Age-adjusted log earnings
1947
Birth cohort
Men's log mortality
Age-adjusted log mortality
1947
Birth cohort
Women's log mortality
Age-adjusted log mortality of women
1947
Birth cohort

This paper's origin

"Why are you not looking at height?"
–Tim Hatton

(at my job talk at Essex)

This paper takes Robert Fogel seriously.

- Fogel argued that early-life health was a key driver of the enormous gains in health and longevity across cohorts born ∼1890–1940s (Fogel 1986; Floud et al. 2011)

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- Key evidence: adult height rose for the same cohorts
  - height as a signal of the change in early-life environment
  - which *also* produced fundamental changes in the human body (Fogel & Costa 1997 "technophysio evolution")

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- This paper: apply that lens to the post-1947 stall in health and human capital, and the rest of the twentieth-century cohorts

Related: Steckel; Floud; Komlos; Harris; Case & Paxson (2008); Bozzoli, Deaton & Quintana-Domeque (2009)

This paper takes Robert Fogel (and J.M. Tanner) seriously.

"Growth is a window into society."

- Examine entire growth process — not just final height
- Childhood, adolescence, adulthood + timing of puberty $\Rightarrow$ *how* people grew, not just *how tall* they ended up
    - multidimensional signal of early-life environment
- Combined with insights from human biology and history can (partially) infer *what* changed about early-life conditions
    - beyond just taller $=$ better

Three Regimes of Human Development in 20th Century America

- Regime I ($\sim$1900–1950):
  rising height at all ages, earlier puberty

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- Regime III ($\sim$1970+):
  faster tempo, no gain in adult height; rising obesity

  portends long-run health risks

Roadmap

1. Data
2. Main evidence
   - Height
   - Menarche
   - Human capital
3. Interpretation
   - Conceptual framework
   - Additional evidence (micro + PGS)
   - Candidate explanations

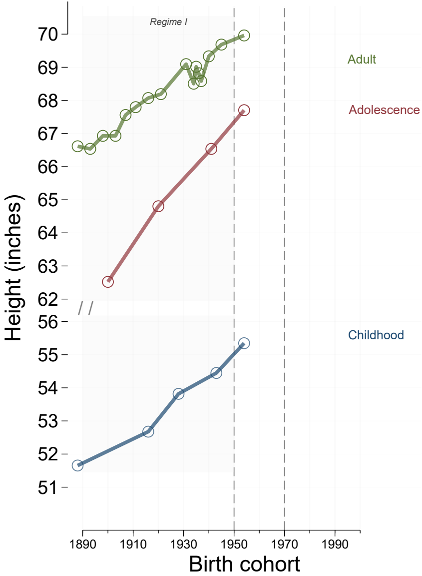
Data

Data

- Pre-1950 (archival sources):
  - Meredith (1964) compiled and averaged childhood height studies
  - Military samples — Costa (2015), Bleakley et al. (2014)
  - Non-Hispanic white males
- Post-1950 (national surveys with measured height):
  - NHES II/III + NHANES I–III + Continuous NHANES
  - Non-Hispanic native-born white males (main analysis)
- Age at menarche: NHANES; retrospective + contemporaneous reports
- Outcomes: IPUMS Census/ACS (education); HMD cohort life tables (mortality)
- Panel data on growth and human capital: NHES II→III linked panel (˜2,000 children)
- HRS (Polygenic scores)

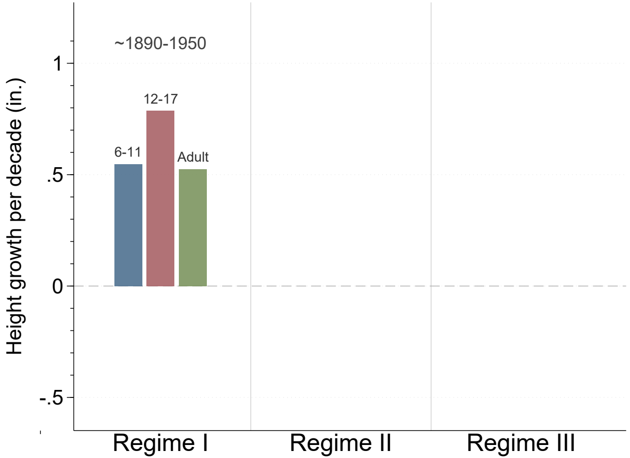
Three regimes of biological development

Regime I: secular growth (cohorts ∼1900–1950)



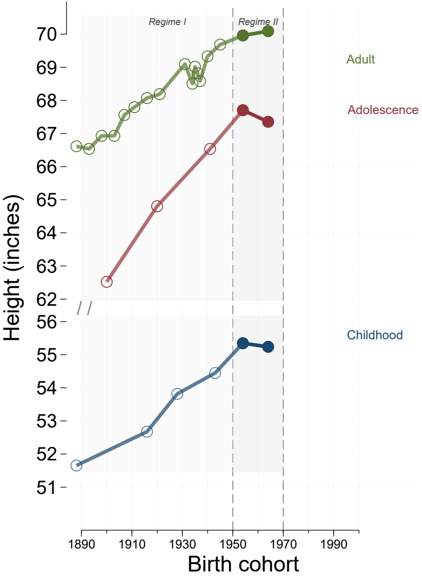
- Steady height gains at all ages

Regime I: magnitudes



- Largest gains in adolescence
- Suggests earlier puberty alongside taller adults

Regime II: comparing cohorts born 1951–57 and 1961–67



- Growth in childhood and adult height stall
- Adolescent height declines

Sampling structure / Survey design

White males Black males

Females

Childhood height stagnation already noted in 1970s!

NCHS Growth Curves for Children

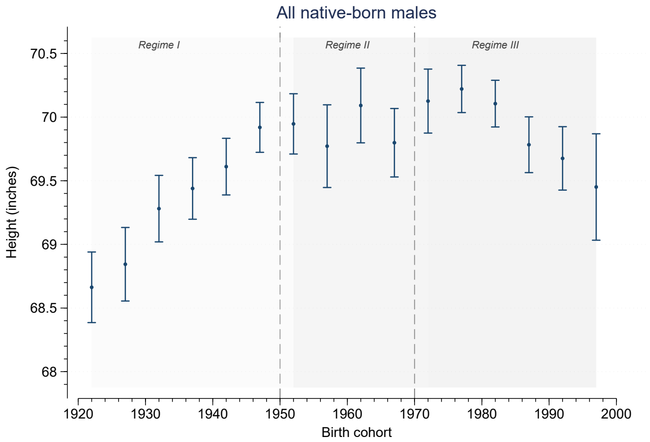
Birth-18 Years

United States

Secular Trend

In the analysis of these data, the marked diminution and near cessation of the trend to constantly increasing size of successive generations of American children is the most dramatic and significant finding relating to human biology and human growth in general. This secular trend to ever-increasing size and earlier maturation (a universal finding among the countries of the western world for the past century that has become a good biologic index of the degree of technological and socioeconomic advance of the developing countries) has been extensively discussed many times.[9,10,26-42]

Regime II: adult height stagnation



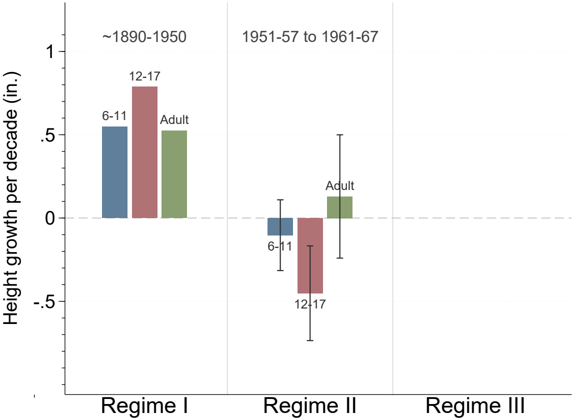
Sample: native-born males only. NHES I respondents (nativity unrecorded) included.

Already noted by Komlos and Lauderdale (2007)!

- U.S. adult height stalls for mid-century cohorts while Europe keeps growing

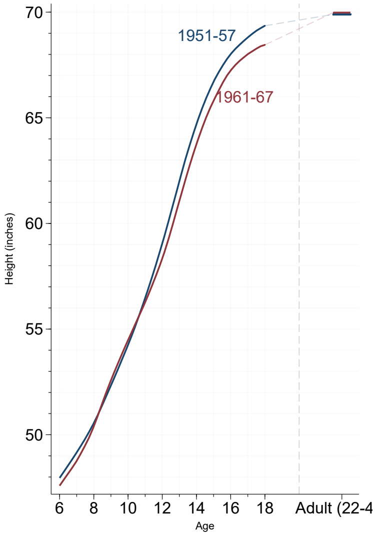
By race

Regime II summary



- Adolescent height declines by ∼0.5 inches while child and adult height stagnate

Regime II summary

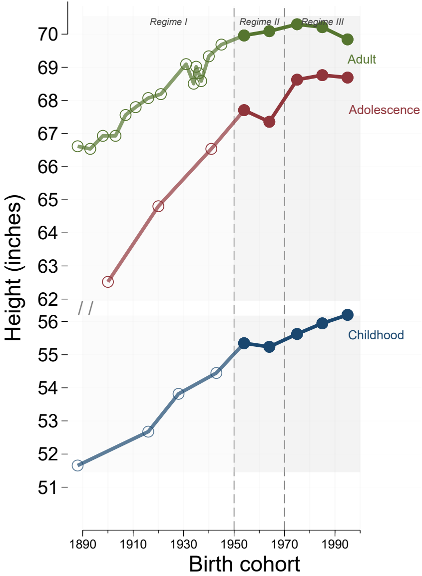


- Slower childhood-to-adolescent growth: -0.43 inches
- Faster adolescent-to-adult catch-up: +0.5 inches
- Consistent with delayed or smaller adolescent growth spurt

Zoom: falling behind

Zoom: catch-up

Regime III: faster tempo, flat adult height (∼1970+ cohorts)



- Childhood and adolescent height rise again
- But adult height stays flat
- → faster tempo of growth without gains in final attained height

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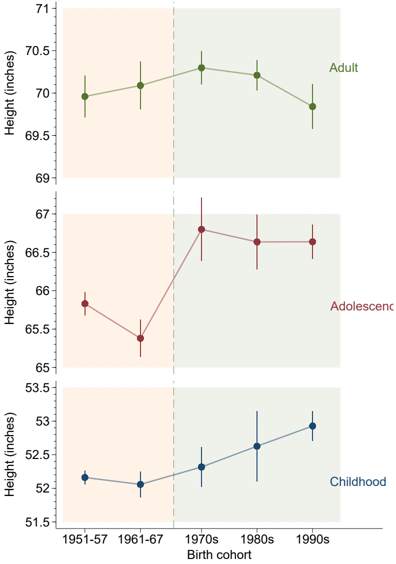

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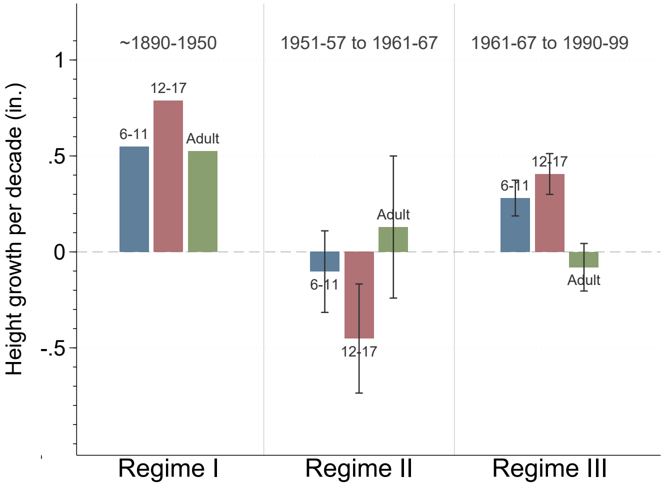
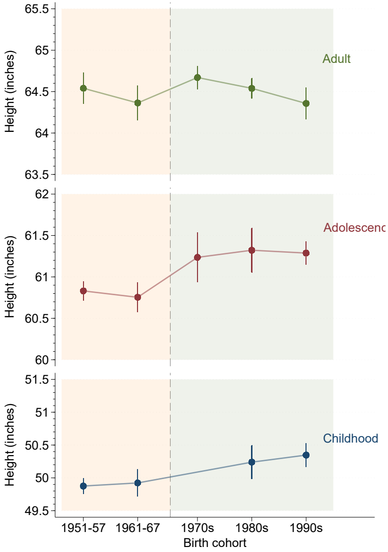
White males   Black males

Females

Three distinct regimes



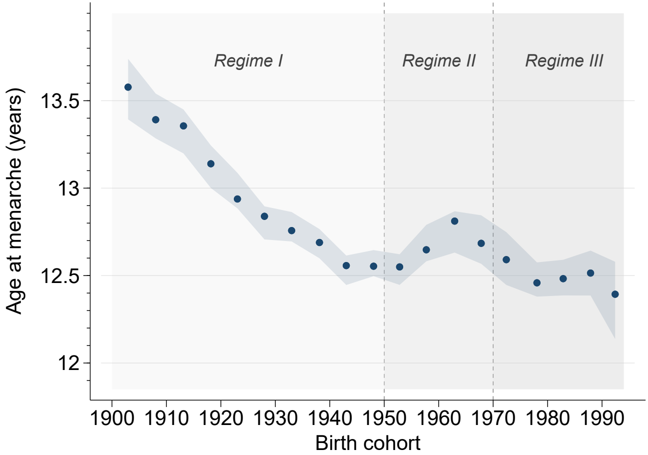
Females



- Regime II: stagnant height in childhood and adulthood; no evidence of adolescent decline
- Regime III: matches males — faster tempo, flat adult height

Slightly earlier age bins than males (6–9, 10–15, 23–40 vs 6–11, 12–17, 23–40) to reflect earlier female maturation.

Menarche: same three regimes



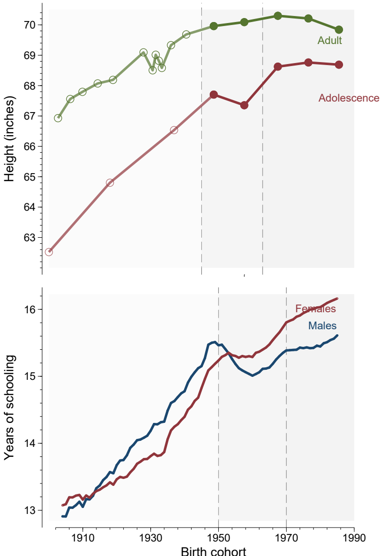
Independent evidence of changes in the timing of puberty

- Regime I: secular decline
- Regime II: reversal — ∼0.25 year delay for 1950s–60s cohorts
- Regime III: partial rebound, no return to prior trend

Confirmed by contemporaneous reports.

Contemporary reports

Tracking between height and education

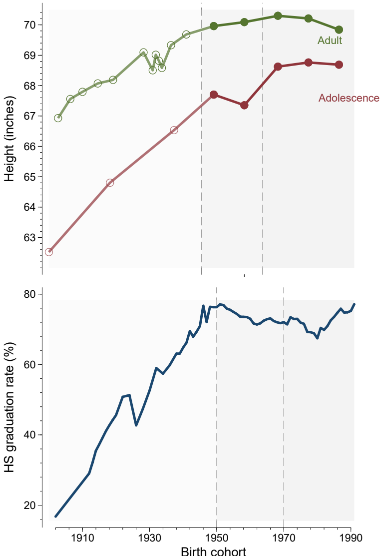


Height

Educational attainment

- Rise and fall/stall track, especially in Regimes I and II

Tracking between height and education

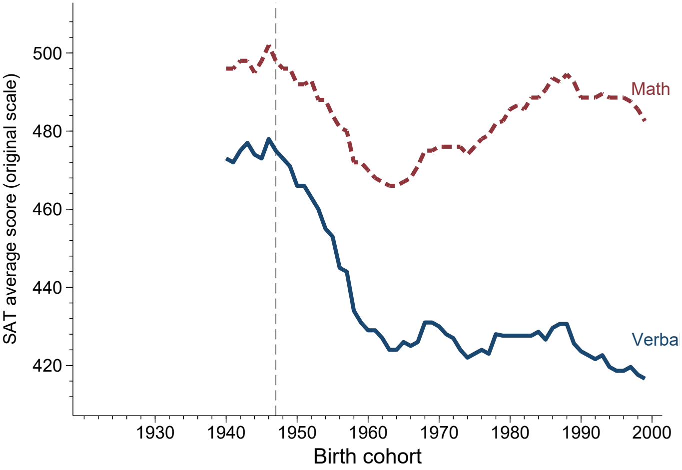


Height

HS graduation rate

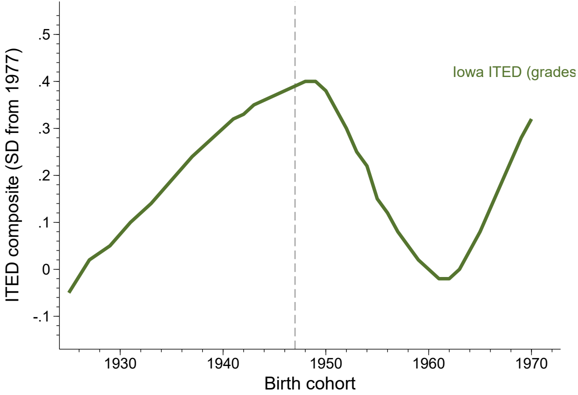
- Rise and fall/stall track, especially in Regimes I and II
- GED masks part of the HS decline

Contemporaneous decline in SAT scores



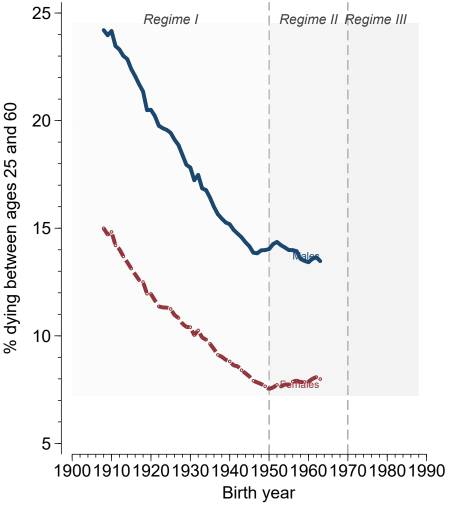
Test-taking population

And in near-universal test in Iowa



- Peak-to-trough decline ≈ 1.25 grade-level equivalents (Bishop, 1989)
- Much bigger rebound than SAT (?)

Working-age mortality



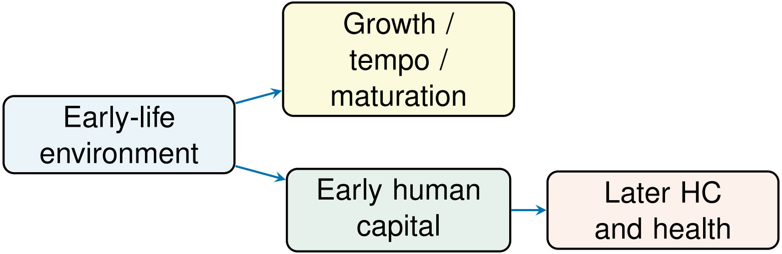
- Mortality decline and stall track Regimes I and II

- Regime III still young; some signs of poor metabolic health

Age detail Through age 54

Interpretation

Growth and tempo as a window into early-life environment



- Growth is an observable signal of the underlying developmental environment
- The object of interest is the shared environment, not height itself
- Different shocks leave different biological signatures
- Later investments can compensate — the mapping is not mechanical

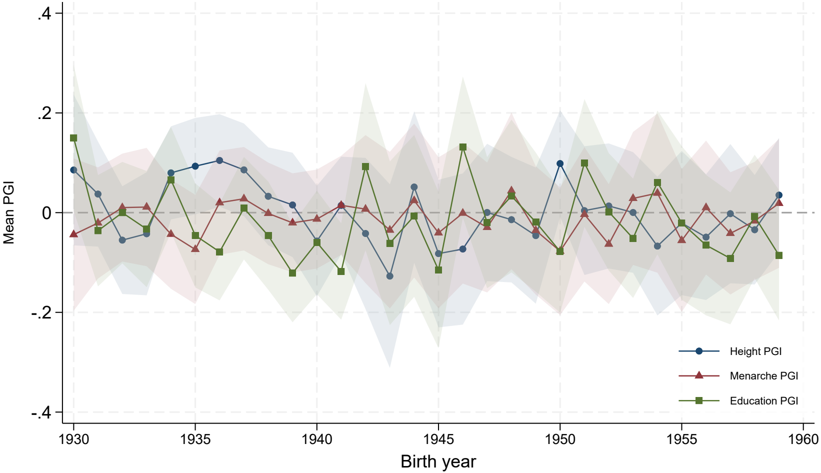
Height and growth predict test scores

	(1)	(2)	(3)	(4)
Childhood height (inches)	0.064***	0.068***	0.048***	0.051***
	(0.008)	(0.008)	(0.008)	(0.008)
Height growth (in/year)		0.146***		0.107***
		(0.042)		(0.039)
Family income (pctile, 0–1)			1.245***	1.233***
			(0.078)	(0.078)
Wave-1 height + age×sex poly. (both waves)	Yes	Yes	Yes	Yes
N	2,065	2,065	2,065	2,065
R^2	0.045	0.051	0.167	0.171

NHES II→III panel: children ages 6–11 at wave 1 (1963–65), re-examined ages 12–17 at wave 2 (1966–70). Birth cohorts ∼1953–1959. $N = 2{,}065$ native-born children. Outcome: Achievement Index (z-scored). Growth winsorized at p99. $^{*}\ p < 0.10$, $^{**}\ p < 0.05$, $^{***}\ p < 0.01$.

Binscatter

Polygenic indices are smooth across cohorts



HRS target respondents of European ancestry, births 1930–1960. Polygenic indices residualized on ancestry PCs.

Environmental component of height predicts human capital

	Education (yrs)		Cognition (27-pt)
	(1)	(2)	(3)
Height (z-score)	0.455***	0.547***	0.498***
	(0.046)	(0.055)	(0.066)
Height PGI		−0.120***	−0.000
		(0.035)	(0.043)
Controls	Yes	Yes	Yes
N	6,591	6,591	6,558

HRS, European-ancestry genotyped subsample, birth cohorts 1930–1965. Controls include sex, cohort fixed effects, race/ethnicity, and ancestry controls.

From micro evidence to macro interpretation

- Within-cohort: height predicts human capital (NHES, HRS, literature)
- But the micro coefficient does not mechanically translate to cohort trends
  - Different shocks load differently on growth vs. cognition vs. health
  - Within-cohort variation $\neq$ between-cohort variation
- Approach: ask which candidate shocks best match the joint biological and human-capital signature
  - Inference to the best explanation, in the spirit of Heckman and Singer (2017)

Appendix: latent-factor formalization

Regime I: classical early-life improvements

- Canonical story: better nutrition, lower disease burden, sanitation, public health (Fogel, Floud et al.)
- Use the **INCAP atole supplement** (Guatemala) as an external benchmark for early-life-driven gains in height, schooling, and test scores
- Compare the implied per-decade gains to U.S. Regime I gains
- Order-of-magnitude plausible that nutrition-driven growth accounts for much of the rise
    - Endogenous responses (e.g. expanding schooling) likely amplify

Regime I calibration: per-decade gains

Outcome (per decade)	Within-cohort (NHES / HRS)	INCAP external	Actual Regime I
Adult height (cm)	—	—	1.3
Schooling (yrs)	~ 0.08	~ 1.0	0.5–1.0
Test score (SD)	~ 0.07	~ 0.23	~ 0.20

Within-cohort gradients explain only ~10–35%; the INCAP exchange rate gets the order of magnitude right.

INCAP: Hoddinott et al. 2008; Maluccio et al. 2009; Hoddinott et al. 2013. Test: ITED (Bishop). Schooling: women-only INCAP rate.

Regime II: joint pattern to match

Dimension	Direction	Magnitude
Adolescent height (males)	$\downarrow$	-0.45 in/decade
Age at menarche (females)	$\uparrow$ (delayed)	$+0.16$ yrs/decade
Adult height	$\rightarrow$ (flat)	~ 0
Educational attainment	$\downarrow$	~ -0.3 yrs/decade (men)
Test scores (SAT, Iowa)	$\downarrow$	~ 0.4 SD (Iowa composite)
Mortality (ages 25–60)	$\rightarrow\uparrow$	improvement stalls

Regime II: preliminary evaluation

Delayed adolescent growth
Delayed puberty
Flat adult height
Lower test scores / education
Worse mortality
Timing (1950s–60s)

Regime II: preliminary evaluation

	Schooling / cultural
Delayed adolescent growth	✗
Delayed puberty	✗
Flat adult height	✗
Lower test scores / education	✓
Worse mortality	✗
Timing (1950s–60s)	$\sim$

Regime II: preliminary evaluation

	Schooling / cultural	**Easterlin / crowding**	**Birth order**	**Lead / envtl. toxin**
Delayed adolescent growth	✗			
Delayed puberty	✗			
Flat adult height	✗			
Lower test scores / education	✓			
Worse mortality	✗			
Timing (1950s–60s)	∼			

Regime II candidate: baby boom and family size

- Standard crowding and congestion stories may fit schooling and labor markets
- But hard to see how school crowding alone generates delayed puberty and slower adolescent growth
- Hospital crowding at birth is more biologically plausible, but timing of cohort-size peak is earlier than the biological break
- **More promising**: birth order and changing selection over the baby boom — can operate through disease exposure and resource dilution
- In my data, birth order matters more for childhood height than adolescent height (tentative)

Appendix: birth-order simulation

Appendix: birth-order figure

Regime II: preliminary evaluation

	Schooling / cultural	**Easterlin / crowding**	**Birth order**	**Lead / envtl. toxin**
Delayed adolescent growth	✗	✗	(✗)	
Delayed puberty	✗	✗	(✗)	
Flat adult height	✗	✗	✓	
Lower test scores / education	✓	✓ partial	✓	
Worse mortality	✗	✗	✓	
Timing (1950s–60s)	$\sim$	peaks earlier	✓	

Regime II: preliminary evaluation

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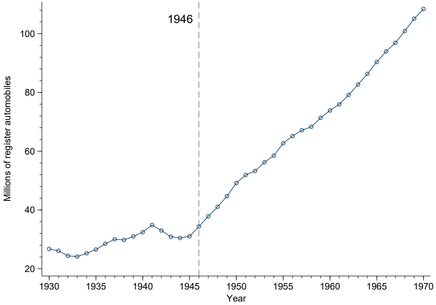
Lower test scores / education	✓	✓ partial	✓	✓
Worse mortality	✗	✗	✓	✓
Timing (1950s–60s)	$\sim$	peaks earlier	✓	✓

Regime II candidate: leaded gasoline

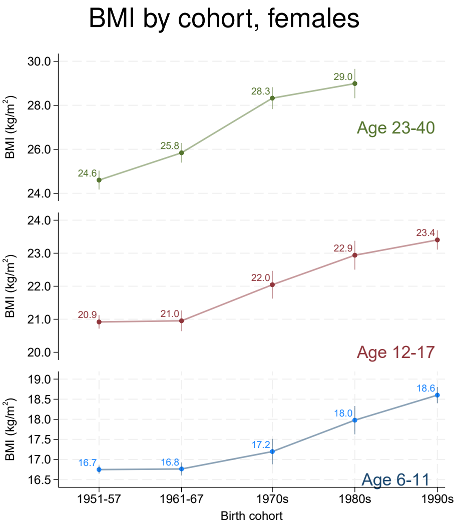
- Postwar auto diffusion $\rightarrow$ cohort-level timing fits
- **Systemic toxin** — multiple organ systems
- Known effects on **cognition** and non-cognitive traits
- **Endocrine disruptor**: puberty timing, adolescent growth spurt
- Plausible effects in particular on pregnancy and birth outcomes, found in Reynolds (2025)

U.S. automobile registrations



Lead additive trend   Pervasiveness

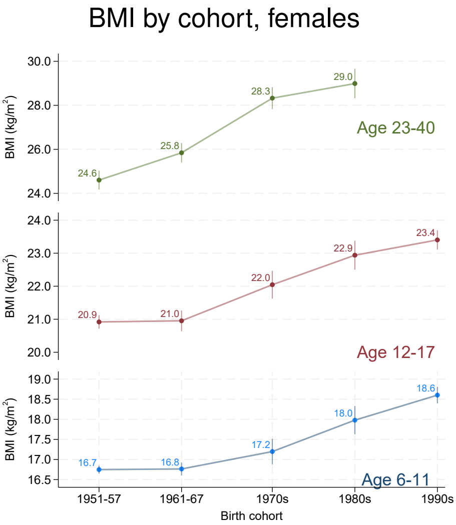
Regime III: the obesogenic environment



Males

- BMI rising at all ages — childhood, adolescence, and adulthood — for post-1970 cohorts

Regime III: the obesogenic environment



- BMI rising at all ages — childhood, adolescence, and adulthood — for post-1970 cohorts
- Higher adiposity/lipids thought to accelerate growth tempo without increasing adult height

Males

The adaptive framework

- **Adaptive framework** (Gluckman & Hanson; Schneider):
  - Prenatal shocks $\rightarrow$ adaptive response $\rightarrow$ faster development / earlier maturity, shorter or same final height
  - Postnatal deprivation $\rightarrow$ slower growth, shorter in adulthood
  - **Regime I** $\approx$ improving postnatal environment $\rightarrow$ faster growth and taller adults
  - **Regime II** $\approx$ negative postnatal shock $\rightarrow$ slower adolescent growth, flat adult height
  - **Regime III** $\approx$ more consistent with prenatal influences $\rightarrow$ faster tempo without gains in adult height
- *Speculative*: prenatal shock to R3 cohorts is being **children of R2 mothers**
- Could be complementary explanation to obesogenic environment post-natally
  - In any event — portends poor health in adulthood

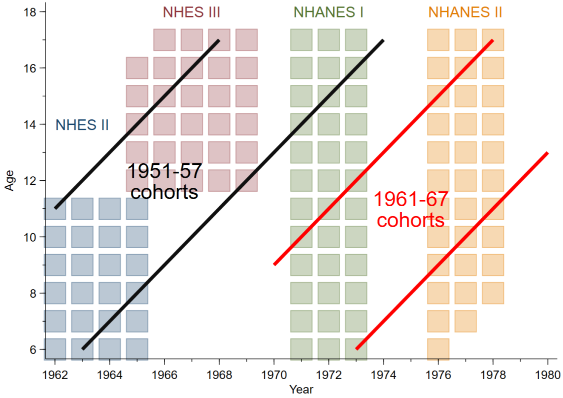
Conclusion

- Three regimes of biological development which track the rise and fall/stall of health and human capital
- Points strongly to **early-life shock** that can impact biology
- Biological evidence can restrict the class of plausible causes
  - **Lead** matches the Regime II pattern unusually well; pure demand-side or crowding stories do not

Related and ongoing work

- Using RDC data to document the precise signature of the decline; search for causes
- International comparisons
- Cohort distributional national accounts
- Real consumption and welfare

Appendix: NHES/NHANES sampling design



- NHES II/III catch 1951–57 cohort ages 6–17
- NHANES I/II catch 1961–67 at the same ages
- Later surveys $\rightarrow$ adult heights for both
- $N \approx$ 7,300 (early) / 3,300 (late)

Back

Appendix: Latent-factor formalization

$$H = \lambda_H E^S + \lambda_H^H E^H + \gamma_H G + \varepsilon_H$$
$$C = \lambda_C E^S + \lambda_C^C E^C + \gamma_C G + \varepsilon_C$$

Micro regression

$$\beta_{C,H}^{\text{micro}} = \frac{\text{Cov}(H, C)}{\text{Var}(H)}$$

Uses all cross-person covariance. Informative about whether a shared developmental channel exists; does not tell us which part survives stricter designs.

Cohort differences

$$\frac{\Delta \bar{C}}{\Delta \bar{H}} \approx \frac{\lambda_C \Delta \bar{E}^S + \lambda_C^C \Delta \bar{E}^C}{\lambda_H \Delta \bar{E}^S + \lambda_H^H \Delta \bar{E}^H}$$

The aggregate shock may load on the shared component, the height-only component, or the cognition-only component. A micro coefficient identifies a channel, not the composition of aggregate change.

Identification of channel is not identification of scale.

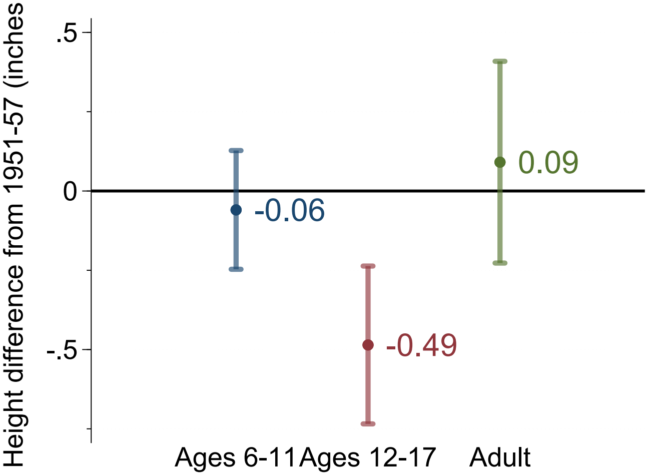
Back

Appendix: Specification

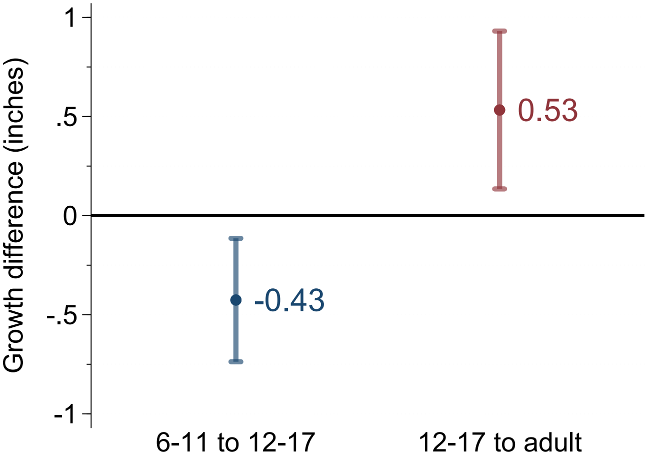
$$Y_{i,a,c} = \gamma_{c=\text{1961-67}} + \mu + f(a) + \epsilon_{i,a,c}$$

- Run separately for ages 6–11, 12–17, 23–40
- $f(a)$: single-year-of-age FE + quadratic in age in months (children/adolescents)
- Sampling weights; native-born only

Appendix: Regime II height differences

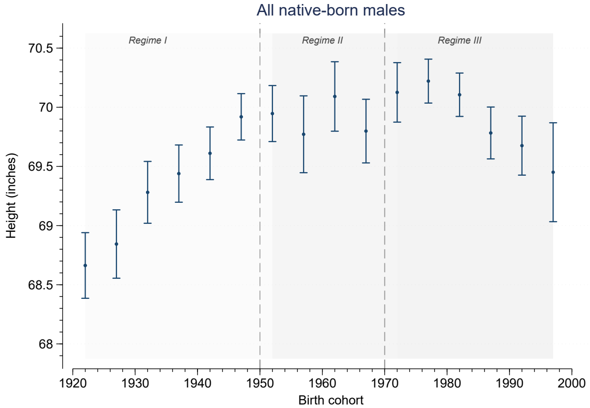


Appendix: Regime II growth differences



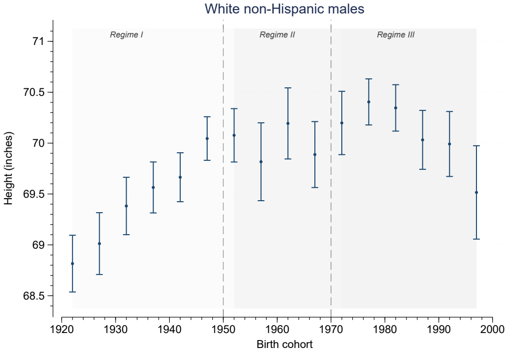
Back

Appendix: Adult height robustness (NHES/NHANES only)

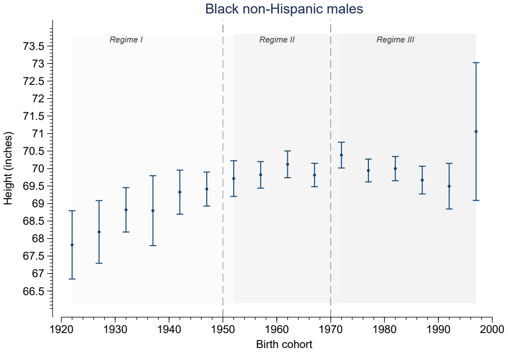


Sample: native-born males only. NHES I respondents (nativity unrecorded) included.

Appendix: Adult height by race (males)



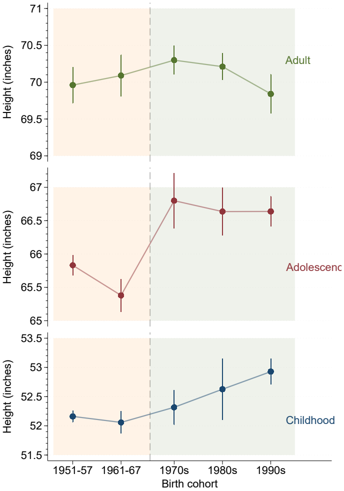
Sample: native-born males only. NHES I respondents (nativity unrecorded) included.



Sample: native-born males only. NHES I respondents (nativity unrecorded) included.

Back

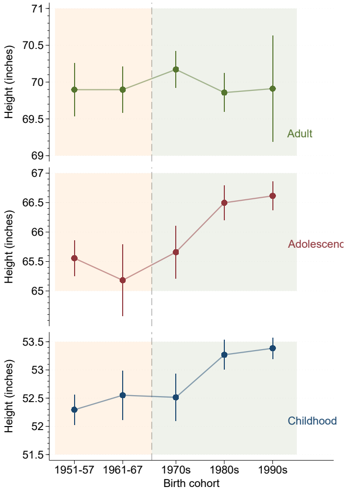
Appendix: White males — cohort height series (1950s–1990s)



Non-Hispanic native-born white males. Childhood (top) and adolescence/adult (bottom) by birth cohort.

Back

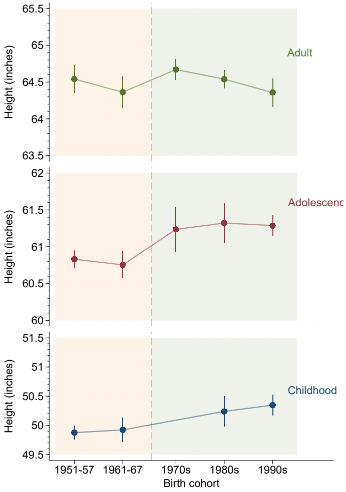
Appendix: Black males — cohort height series (1950s–1990s)



Non-Hispanic native-born Black males. Same age groups and cohort range.

Back

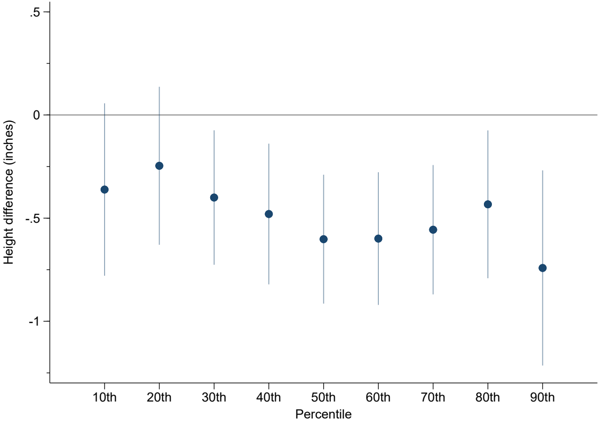
Appendix: Females — cohort height series (1950s–1990s)



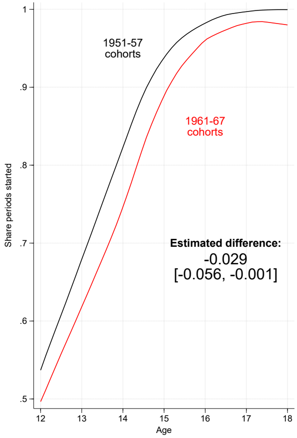
Non-Hispanic native-born females (all races). Shifted age definitions for female pubertal timing.

Back

Appendix: 1961–67 vs. 1951–57 adolescent height by quantile

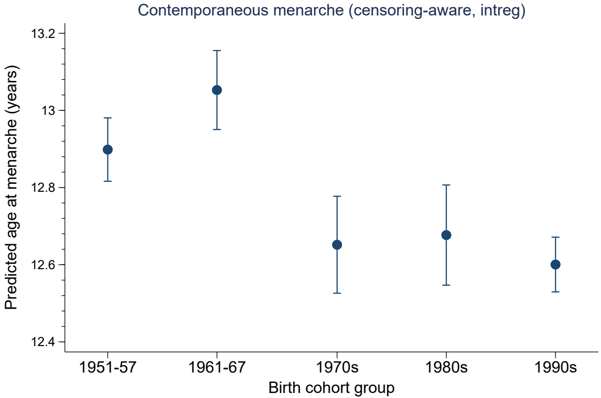


Appendix: Contemporaneous menarche by cohort (1951–57 vs 1961–67)



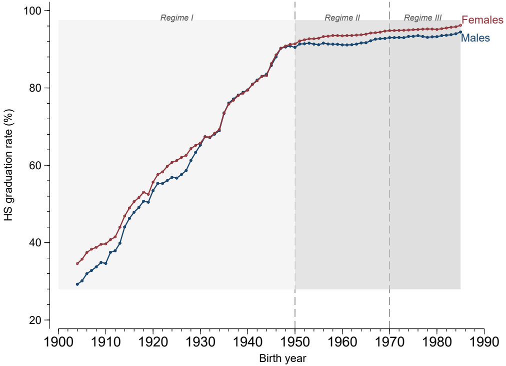
Back

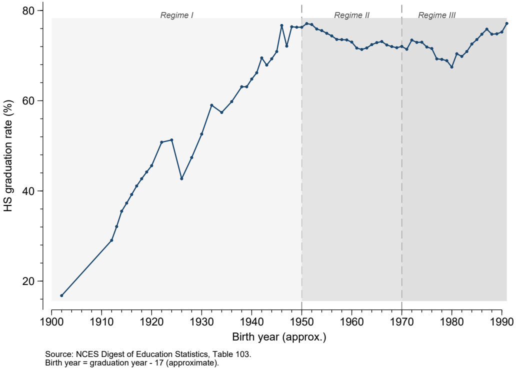
Appendix: Censoring-adjusted contemporaneous menarche



predict(xb) = conditional mean of the latent (uncensored) age at menarche.
Sample: native-born females (includes NHES I respondents, nativity unrecorded).

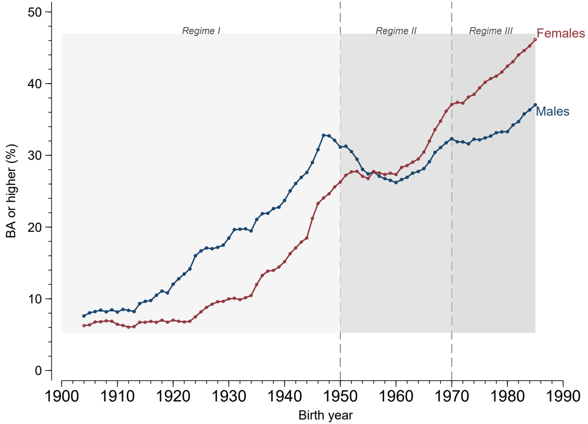
Appendix: HS attainment, three regimes



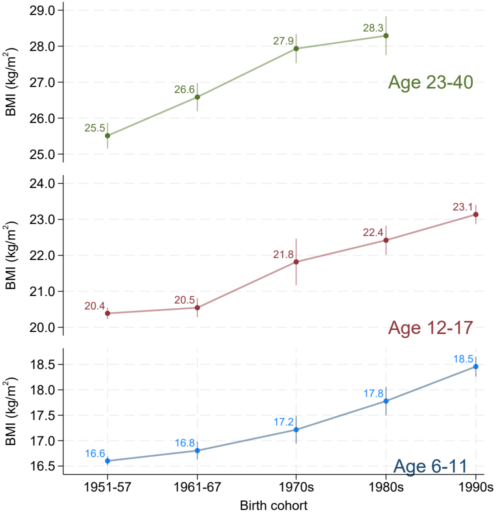


Left: Census/ACS self-reported HS completion. Right: NCES administrative records (graduation counts / 17-year-old population).

Appendix: BA attainment, three regimes

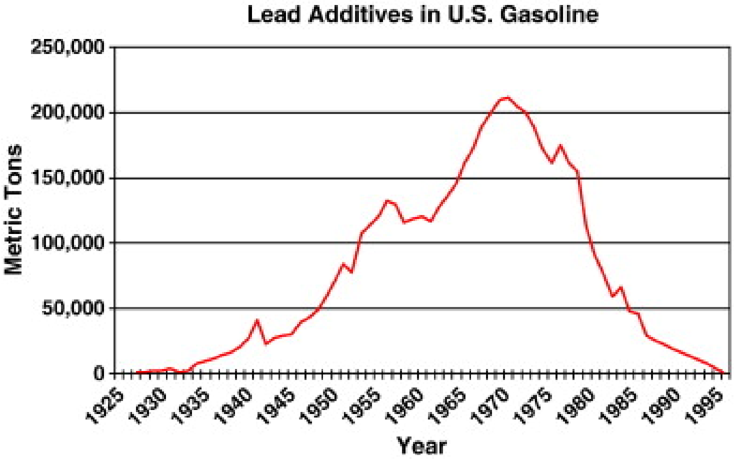


Appendix: BMI by cohort across life stages — males



Back

Appendix: lead additive trend



Back

Appendix: Blood lead was pervasive, not just urban

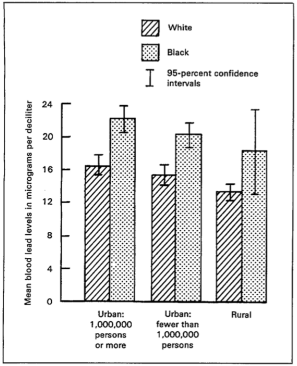


Figure 4. Blood lead levels of children 6 months–5 years by race and degree of urbanization: United States, 1976–80

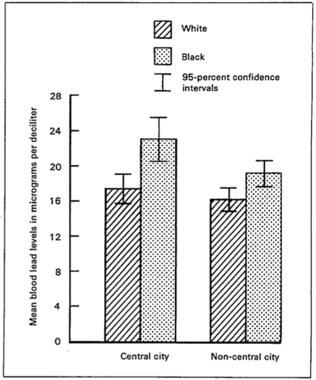
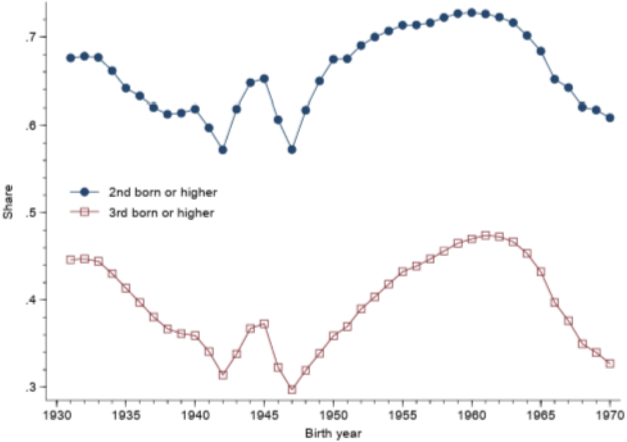


Figure 5. Blood lead levels of children 6 months–5 years in large urban areas by race and location: United States, 1976–80

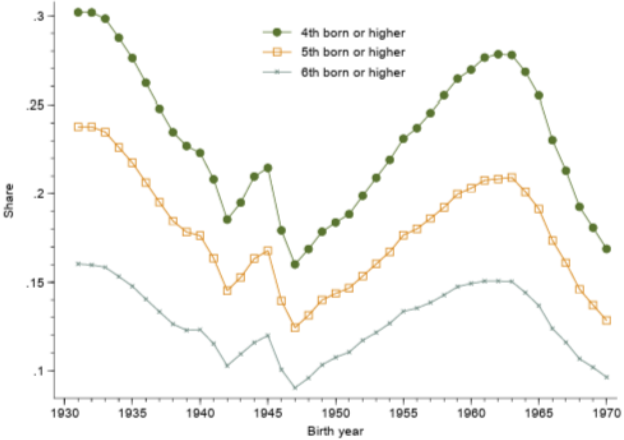
Source: Annest and Mahaffey (1984), "Blood Lead Levels for Persons Ages 6 Months–74 Years," *Vital and Health Statistics*, Series 11, No. 233. NCHS/NHANES II, 1976–80.

Back

Appendix: Birth order 1–2 share among white births

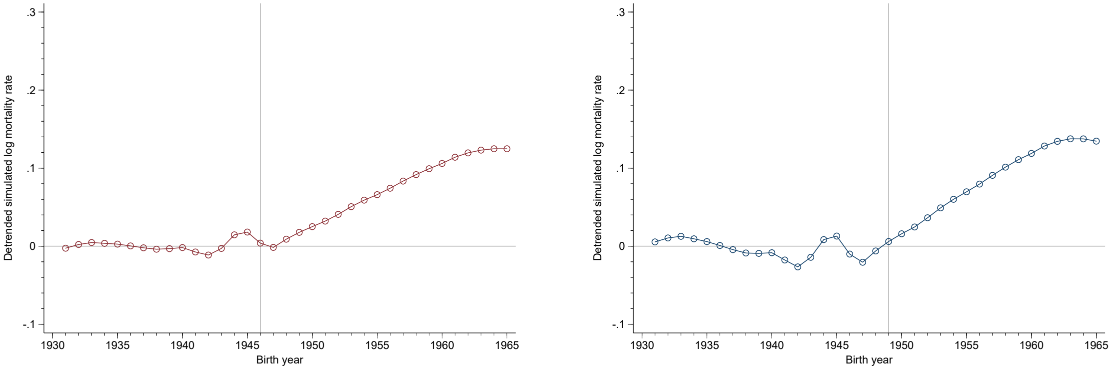


Appendix: birth order 4+ share over the baby boom



Share of white births that are birth order 4+, highlighting the baby-boom family-size shift.

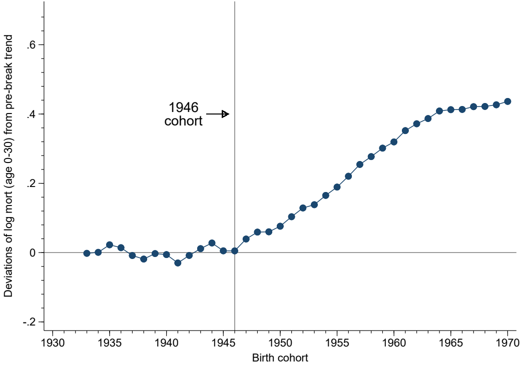
Appendix: birth-order simulations explain part, not all, of the shift

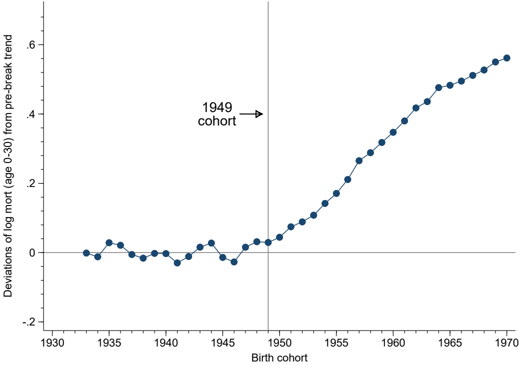


Mortality-paper simulations suggest changing birth order/family size could account for part of the later decline, but not the full broad pattern.

Back

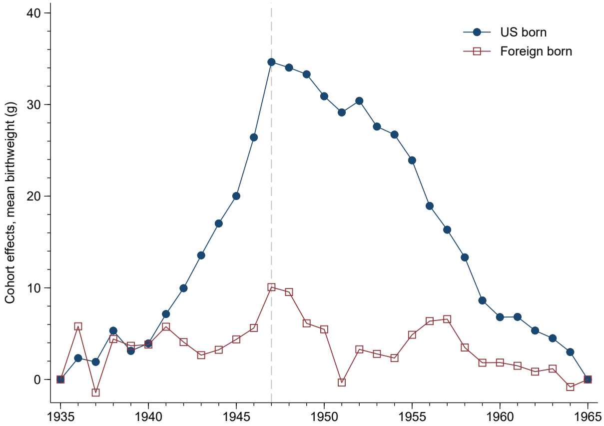
Appendix: no strong early-life mortality selection story





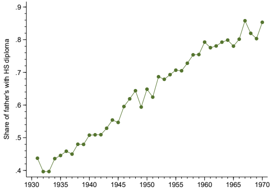
Residual cumulative mortality to age 30 does not show a pattern that makes selective survival a compelling explanation for the cohort break.

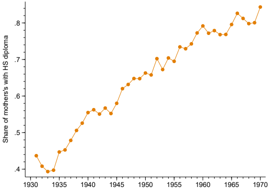
Appendix: the broad decline is concentrated among U.S.-born cohorts

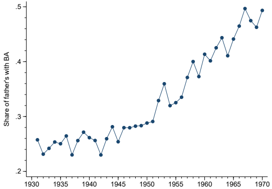


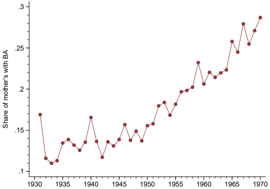
In the broad-decline paper, maternal-health deterioration is evident for U.S.-born mothers but not for mothers born abroad.

Appendix: observable parental background kept improving



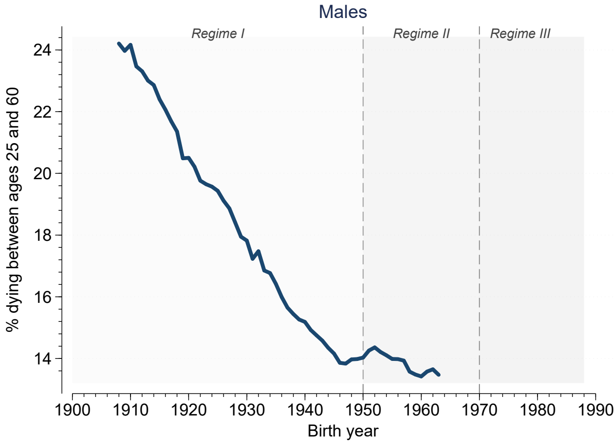




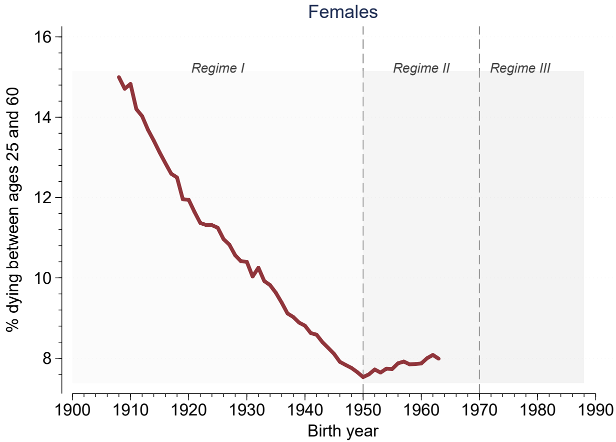


Broad-decline background checks: parental schooling trends do not line up with a deteriorating family-SES story around the late-1940s break.

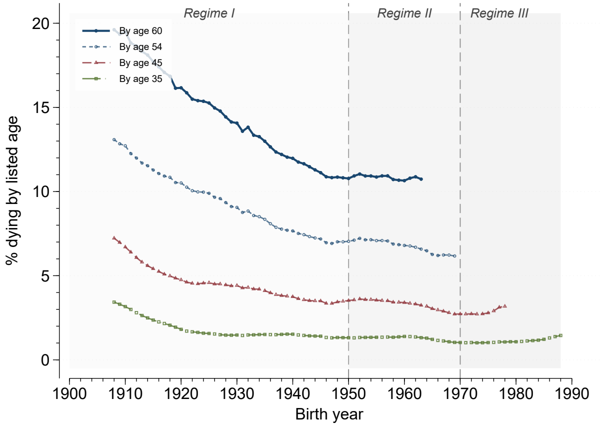
Appendix: Adult mortality — men



Appendix: Adult mortality — women

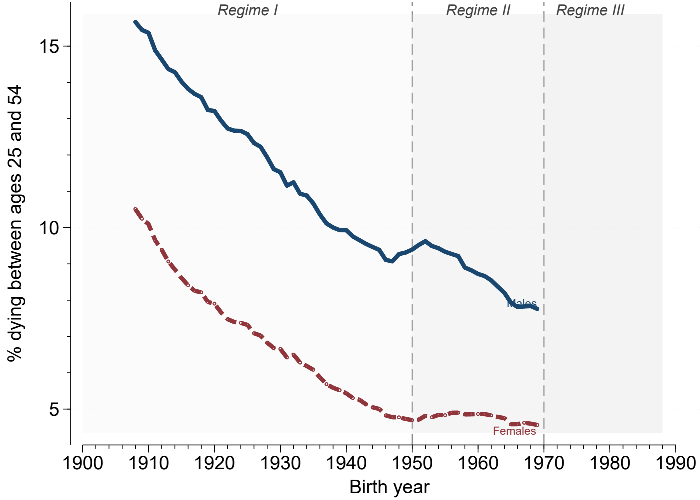


Appendix: Adult mortality by age



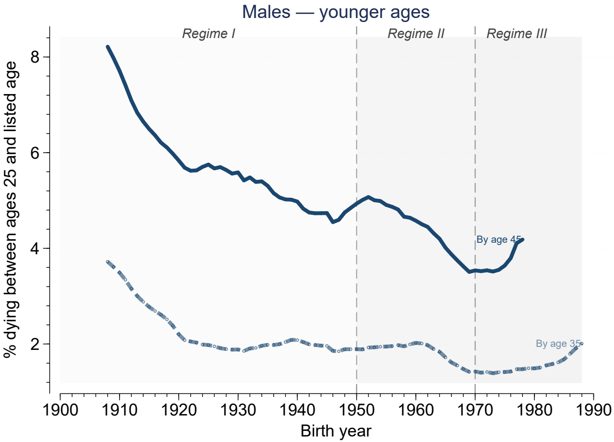
Back

Appendix: Working-age mortality through age 54

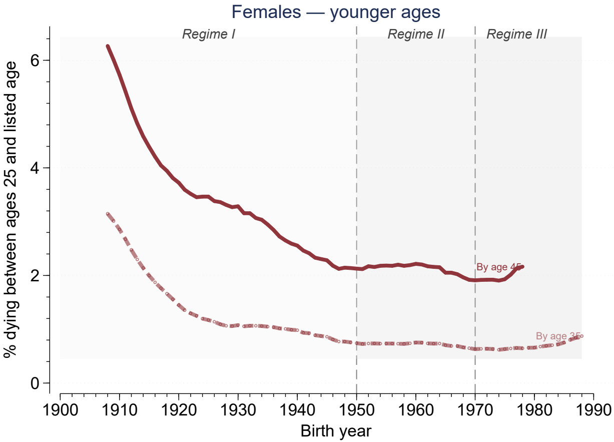


Back

Appendix: Younger-age mortality — men

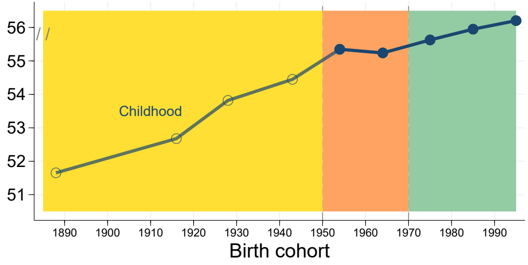


Appendix: Younger-age mortality — women

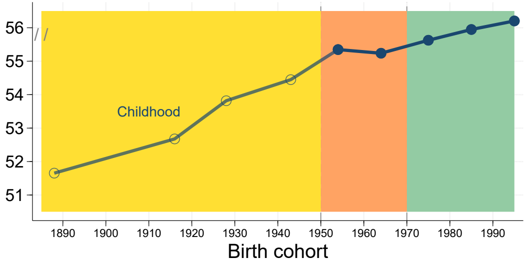


Appendix: Panel evidence by sex

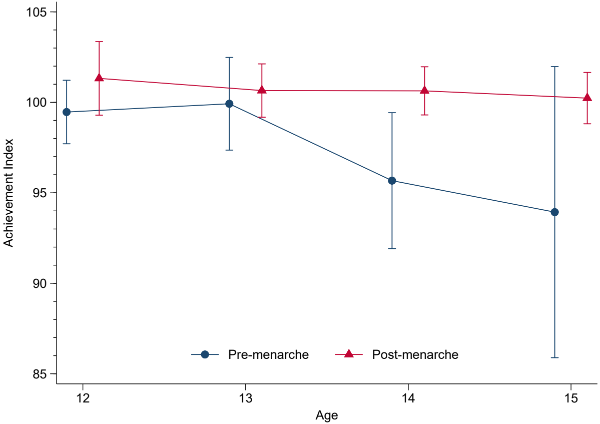
Boys: growth tempo and achievement



Girls: growth tempo and achievement



Appendix: Panel — girls and menarche

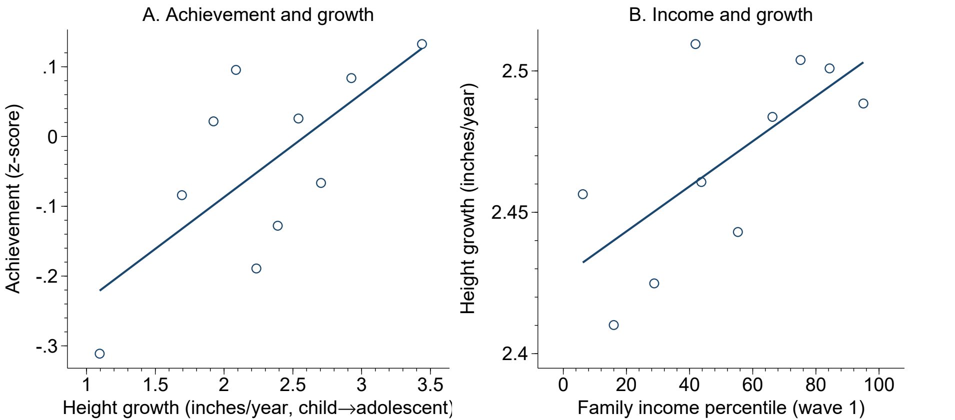


Appendix: SAT test-taking population

- SAT test-taking expanded rapidly during the decline period:
  - $\sim$80,000 takers in 1951 (cohort $\sim$1933)
  - $\sim$800,000 in 1961 (cohort $\sim$1943) — 10$\times$ increase in a decade
  - $\sim$1,000,000 in 1973 (cohort $\sim$1955) — $\sim$33% of HS seniors
  - $\sim$1,000,000 in 1986 (cohort $\sim$1968) — $\sim$38% of HS seniors
- Wirtz Commission (1977): $\sim$75% of the 1963–70 SAT decline was purely compositional (more lower-SES students taking the test)
- Only $\sim$25% of the 1970–77 decline was compositional; the rest reflected genuine achievement changes
- Iowa ITED eliminates this concern: near-universal coverage ($\sim$99% of schools)

Back

Appendix: NHES binscatter — family resources, tempo, and scores



Height in inches, growth in inches/year. Achievement z-scored. Controls: wave-1 height, age×sex polynomials (both waves). NHES II→III panel, $N = 2{,}065$.

Back