

Life Course Socioeconomic Position and health in older adulthood: A Formal Mediation Analysis in the 1958 British Birth Cohort

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Summary

Background Childhood socioeconomic position (SEP) is a key determinant of later-life health. Understanding the extent to which adult SEP mediates this association into early old age is important for explaining how health inequalities are propagated across generations and how they might be addressed in later life. To our knowledge, no prospective study has examined whether childhood SEP remains associated with health at the threshold of older age and the extent to which any such association is mediated by adult SEP.

Methods We used data from the 1958 British Birth Cohort, a prospective study that has followed participants since birth, drawing on earlier data collected at birth and ages 33 and 55 years and newly collected data from the age 62 sweep. Using interventional causal mediation analyses, we assessed whether adult occupational class, education, housing tenure, and income mediate associations between childhood social class (manual vs non-manual) and health at age 62 (self-rated health, C-reactive protein [CRP], cholesterol ratio, Glycated hemoglobin [HbA1c], and N-terminal pro-B-type natriuretic peptide [NT-proBNP]).

Findings Associations between childhood SEP and self-rated health, CRP, cholesterol ratio, and HbA1c persisted after accounting for adult SEP. Mediation was outcome-specific and differed by sex. Among men, occupational class mediated 39% of the association with self-rated health (indirect effect RR 0·90, 95% CI 0·86–0·95) and education mediated 27% (0·93, 0·90–0·96). Among women, education mediated 10% (0·95, 0·91–0·98) and housing tenure mediated 6% (0·97, 0·94–0·99). Indirect effects for CRP were smaller, and mediation was minimal for cholesterol ratio, HbA1c, and NT-proBNP.

Interpretation Population-level improvements in adult SEP could reduce, but are unlikely to eliminate, later-life health inequalities associated with childhood SEP. Reducing these inequalities will require policies that address disadvantage in early life and improve adult financial and employment conditions.

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Introduction

Population ageing presents a major challenge for health systems, pension and social policy. It is widely understood that health in older age is shaped by processes that begin early in life and unfold across the life course¹⁻², making it essential to understand the early-life determinants of healthy ageing. Evidence from British birth cohort shows a childhood SEP gradient in survival, with those from the disadvantaged childhood social class experiencing roughly 1.7-2.7 times higher premature mortality risk than those from more advantaged childhood social class³. Similar gradients have also been reported in other settings, such as the U.S. and Northern Europe⁴.

A central debate in life-course epidemiology concerns how much of this early-life imprint is amenable to intervention through adult socioeconomic circumstances, as one pathway among many. The critical period hypothesis posits that childhood SEP has independent effects on later health that are not fully modified by subsequent socioeconomic mobility⁵. In contrast, the accumulation and pathway hypotheses emphasize cumulative and sequential processes in which childhood SEP shapes adult SEP, which in turn influences later health⁵. In this framing, adult SEP represents one pathway linking childhood (dis)advantage to later health, with policy relevance for whether interventions should priorities early-life conditions, adult socioeconomic inequalities, or both?

Evidence suggests that childhood (dis)advantage influences later health through adult SEP and through effects that persist independent of adult SEP, suggesting that accumulation, pathway, and critical processes operate concurrently⁶⁻⁸. However, much of this evidence relies on traditional mediation approaches, inferring indirect effects either from attenuation of the exposure coefficients after adjustment (difference-in-coefficients approach)⁷, or from multiplying regression coefficients after adjustment (product-of-coefficients approach)⁸. These approaches require restrictive assumptions, including correctly specified linear models and no exposure-mediator interaction, and may yield biased estimates in complex life course settings characterized by non-linearity, interaction, and confounding⁹.

In the UK, much of the evidence to date on childhood SEP and health focuses on early or mid-adulthood, when socioeconomic trajectories and health risks are still unfolding¹⁰⁻¹³. Studies looking at health in older age have often relied on retrospectively reported childhood circumstances¹⁴, which do not prospectively trace socioeconomic trajectories across the full life course within the same individuals. To our knowledge, no prospective evidence addresses whether childhood SEP remains associated with health at the threshold of older age, and the extent to which any association is consistent with operating via adult SEP.

We aimed to fill these gaps by examining how the associations between childhood SEP and health at age 62 were mediated by adult SEP. Grounded in a counterfactual framework, we applied formal mediation methods to estimate interventional indirect effects via adult SEP and interventional direct effects representing the remaining association not operating through adult SEP as defined and measured in our study. Our estimates provide policy-relevant ‘what if’ contrasts by quantifying the potential reduction in later health inequalities under hypothetical shifts in adult SEP. Leveraging rich longitudinal data from the 1958 British Birth Cohort, this study provides rigorous empirical insight into the mechanisms linking childhood SEP to health at the onset of older age.

Methods

Data

The study used the National Child Development Study (NCDS, also known as the 1958 British birth cohort)¹⁵⁻¹⁶, a nationally representative longitudinal study of 17,415 individuals born in Britain during one week in 1958. To date, eleven sweeps of data are available, when cohort members were aged 0, 7, 11, 16, 23, 33, 42, 46, 50, 55, and 62 years. Information collected from age 0, 33, 55, 62 was used in this study. In 2020, 8,405 cohort members participated in the age 62 sweep, of whom 6,309 provided blood samples. The age 62 data collection took place over an extended period in 2020-2024, when study members were 62-66 years old.

Outcome

We examined biomarkers reflecting systemic inflammation (C-reactive protein [CRP]) and cardiometabolic risk (cholesterol ratio, glycated haemoglobin [HbA1c], and N-terminal pro-B-type natriuretic peptide [NT-proBNP; a marker of heart failure]) in midlife, given their strong social patterning and established links to later morbidity and mortality¹⁷⁻¹⁸.

CRP (mg/L) values <3 mg/L were classified as no inflammation (0) and 3-10 mg/L as inflammation (1); values >10 mg/L were excluded to reduce acute infection effects. The cholesterol ratio was calculated as total cholesterol divided by high-density lipoprotein cholesterol (mmol/L). HbA1c was converted from mmol/mol to percentage units (%). NT-proBNP (pg/mL) was log-transformed and analysed as observed, as no physiologically justified correction exists, and medication effects are heterogeneous. We applied add-back corrections for medication use to approximate untreated cholesterol ratio and HbA1c levels among medicated participants (appendix section 4). For all biomarkers, higher values indicate poorer health. We also examined self-rated health, dichotomised as good (excellent/very good/good = 0) versus fair/poor (=1).

Life course SEP

Childhood SEP was operationalised using parental occupational class at birth (Registrar General's Social Class scheme, RGSC) and dichotomised as non-manual (I, II, and IIIN) versus manual (IIIM, IV, and V). Adult SEP at age 55 was operationalised using occupational class (manual/non-manual), housing tenure (owner/renter), educational attainment (degree or higher/no degree), and income to assess which distinct measures exert the strongest mediating effects.

Directed acyclic graph and confounders

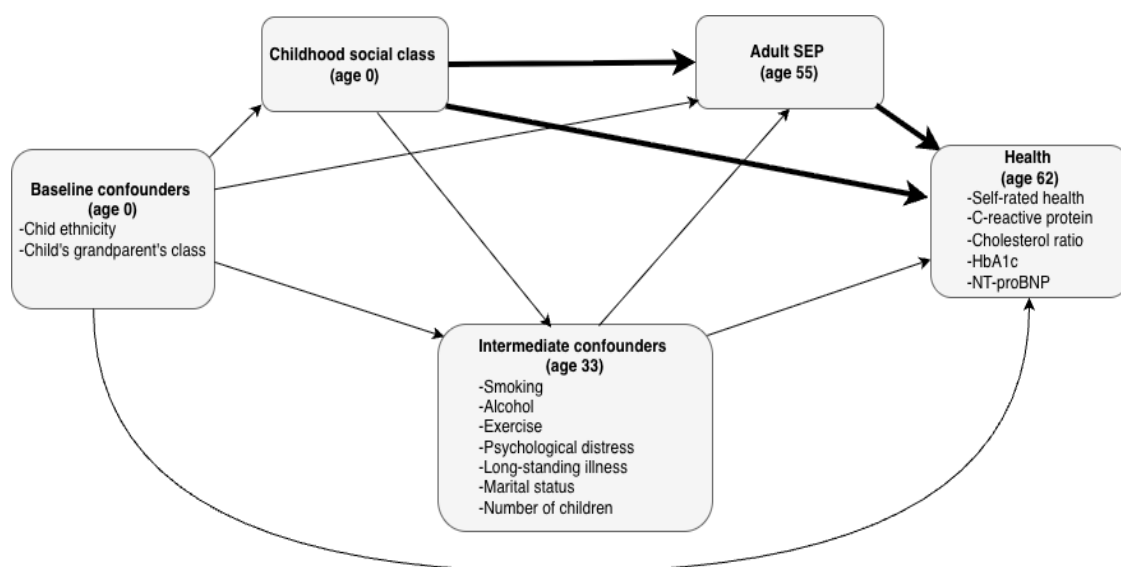
Figure 1 presents the directed acyclic graph (DAG) outlining the assumed causal relationships among confounders, childhood social class, adult SEP, and health outcomes. Childhood social class was the exposure, hypothesised to influence later-life health both directly and indirect via adult SEP. Separate analyses were conducted for each of the four adult SEP indicators specified as mediators.

Baseline confounders included ethnicity (Black and minority ethnic [BME]/non-BME), and grandparental social class (RGSC), reflecting demographic, and intergenerational social advantage that precede and may confound SEP trajectories and later health. Intermediate confounders, measured at age 33 and therefore prior to adult SEP at age 55, included smoking (yes/no), regular alcohol use (yes/no), low exercise (yes/no), psychological distress (standardized malaise score), long-standing illness (yes/no),

marital status (married/not), and number of children. These variables may be affected by childhood SEP and can influence both adult SEP and later health. Sample characteristics are provided in the Appendix Table A1-A2.

Our primary estimands were the interventional indirect effect and direct effect. The interventional indirect effect captures the component operating via adult SEP. The interventional direct effect represents the remaining childhood SEP and health association not captured by adult SEP as measured here (and may reflect pathways operating through other mechanisms). Causal interpretation additionally assumes no unmeasured confounding conditional on variables included in Figure 1, as well as positivity, consistency, and no interference¹⁹.

Figure. 1 Directed acyclic graph (DAG) depicting the assumed causal structure, bold arrows indicate the estimands of interest



Statistical analysis

We first summarized age 62 health outcomes overall and by childhood social class. We then used regression models to examine the association between childhood social class and health outcomes, adjusting for baseline confounders. Additional models then assessed how childhood social class related to adult SEP and how adult SEP related to health outcomes, helping clarify the pathways linking early life social class to later health. We then conducted mediation analyses to estimate interventional direct and indirect effect of childhood social class on health, adjusting for all confounders.

Interventional effects were estimated using a g-formula approach with direct counterfactual imputation. It allows identification of indirect effects despite exposure-induced intermediate confounding (i.e. midlife behavioural, health, and social factors in Figure 1), and without requiring the cross-world independence assumptions of natural effects²⁰⁻²³. It also yields estimates under well-defined hypothetical interventions, thereby clarifying the alternative scenarios being compared and enhancing the policy

relevance of the analysis (Table 1).

Mediation analyses were performed using the CMAverse R package²⁴, with additional g-formula options provided by the CMAversePlus add-on²⁵. Confidence intervals for all estimands were obtained via bootstrapping. All analyses were conducted separately for men and women using Stata 18.5 and R. For the regression-based analyses, missing data were imputed using multiple imputation by chained equations (MICE), generating 50 imputed datasets²⁶⁻²⁸. The mediation procedure implemented MICE internally (appendix section 2).

All analyses were restricted to participants with non-missing outcome data, and analytic sample sizes therefore varied across outcomes (appendix tables A1–A2). Regression and mediation analyses included 4183 men and 4180 women for self-rated health, 1996 men and 2018 women for C-reactive protein, 2479 men and 2505 women for cholesterol ratio, 2461 men and 2481 women for HbA1c, and 2413 men and 2473 women for NT-proBNP. Inverse probability weighting was applied to account for attrition. Models including adult occupational class as the mediator excluded participants who were out of the labour force at age 55 years, reducing the corresponding samples to 3715 men and 3347 women, 1787 men and 1649 women, 2220 men and 2034 women, 2204 men and 2015 women, and 2155 men and 2014 women, respectively.

Table 1 Interpreting patterns of interventional effects

Pattern of effects	Interpretation	Implications
Small direct effect; large indirect effect	Most of the association operates via the mediator.	Improving adult SEP may substantially offset childhood disadvantage.
Large direct effect; small indirect effect	The association is unexplained by the mediator.	Intervening on adult SEP alone will be insufficient; early-life interventions are critical.
Both direct and indirect effect are moderate	Multiple pathways contribute meaningfully.	A combined early-life and adult-life strategy may be required.
Both direct and indirect effect are null	No clear mediated or direct pathway.	Interventions on either SEP unlikely to change the outcome.

Role of the funding source

The funders of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results

Table 2 presents descriptive statistics based on participants with non-missing outcome data and observed childhood social class. Participants from manual childhood social class had a higher prevalence of fair or poor self-rated health than those from non-manual childhood social class. They also had a higher prevalence of inflammation, a less favorable mean cholesterol ratio, and higher mean HbA1c. Little difference was observed for log-transformed NT-proBNP. Similar patterns were seen in sex-stratified

descriptive analyses (appendix table A3).

Table 2 Health outcomes by childhood social class

	Overall	Non-manual	Manual	SEP difference (CI)
Outcome				
Poor/fair self-rated health, n (%)	1933 (25.6)	463 (19.3)	1470 (28.6)	9.3 (7.3, 11.3)
Presence of inflammation, n (%)	803 (22.1)	193 (16.1)	610 (25.1)	9.0 (6.3, 11.7)
Cholesterol ratio, mean (SD)	4.03 (1.29)	3.88 (1.22)	4.11 (1.32)	0.23 (0.15, 0.31)
Glycated hemoglobin (HbA1c) (%), mean (SD)	6.05 (1.05)	5.93 (0.82)	6.11 (1.15)	0.17 (0.11, 0.23)
NT-proBNP (pg/mL), mean (SD)	4.15 (0.89)	4.16 (0.88)	4.15 (0.90)	-0.01 (-0.07, 0.04)

Notes: Data are n (%) or mean (SD). SEP difference is manual minus non-manual and is expressed as an absolute percentage-point difference for binary outcomes and a mean difference for continuous outcomes. Denominators vary across outcomes because of outcome-specific missingness. NT-proBNP was log-transformed prior to analysis.

Table 3 presents regression estimates of the total association between childhood social class and health at age 62, adjusted for baseline confounders. Among men, a non-manual (vs manual) childhood social class was associated with 34% lower odds of poor self-rated health (OR = 0.66, 95% CI: 0.48, 0.91) and 46% lower odds of elevated C-reactive protein (OR = 0.54, 95% CI: 0.35, 0.82). A non-manual childhood social class was also associated with more favorable cardiometabolic profiles, reflected in lower cholesterol ratio, glycated hemoglobin, and NT-proBNP levels. Among women, associations were in the same direction but generally stronger. Appendix Tables A6 and A7 show strong intergenerational socioeconomic persistence and graded associations between adult SEP and health at age 62, most clearly for self-rated health.

Table 3 Regression-based estimates of associations between childhood social class and health outcomes at age 62

Panel A. Men					
	Self-rated health	C-reactive protein	Cholesterol ratio	Glycated haemoglobin	NT-proBNP
	OR (CI)	OR (CI)	β (CI)	β (CI)	β (CI)
	(n = 4,183)	(n = 1,996)	(n = 2,479)	(n = 2,461)	(n = 2,413)
Childhood social class (ref: manual)	0.66 (0.48, 0.91)	0.54 (0.35, 0.82)	-0.11 (-0.33, 0.11)	-0.16 (-0.37, 0.05)	0.95 (0.81, 1.13)
Grandparental class (ref: manual)	0.74 (0.52, 1.06)	0.93 (0.58, 1.49)	0.04 (-0.20, 0.28)	-0.08 (-0.29, 0.14)	0.85 (0.71, 1.01)
BME (ref: not BME)	1.37 (0.66, 2.84)	0.32 (0.09, 1.08)	0.44 (-0.70, 1.58)	0.96 (-0.79, 2.72)	0.76 (0.50, 1.17)
Intercept	0.56 (0.48, 0.65)	0.32 (0.26, 0.40)	4.33 (4.23, 4.44)	6.24 (6.13, 6.34)	4.06 (3.97, 4.15)
Panel B. Women					

	Self-rated health OR (CI) (n = 4,180)	C-reactive protein OR (CI) (n = 2,018)	Cholesterol ratio β (CI) (n = 2,505)	Glycated haemoglobin β (CI) (n = 2,481)	NT-proBNP β (CI) (n = 2,473)
Childhood social class (ref: manual)	0.49 (0.37, 0.65)	0.51 (0.37, 0.70)	-0.35 (-0.48, -0.21)	-0.09 (-0.19, -0.00)	1.01 (0.91, 1.13)
Grandparental class (ref: manual)	0.74 (0.55, 1.01)	0.96 (0.67, 1.37)	-0.09 (-0.25, 0.07)	-0.22 (-0.31, -0.13)	0.94 (0.84, 1.06)
BME (ref: not BME)	0.97 (0.44, 2.16)	0.88 (0.31, 2.45)	-0.14 (-0.50, 0.22)	0.03 (-0.16, 0.22)	0.76 (0.61, 0.95)
Intercept	0.63 (0.55, 0.73)	0.42 (0.36, 0.50)	3.91 (3.83, 3.99)	6.05 (5.98, 6.13)	4.37 (4.31, 4.43)

Notes: BME = Black and minority ethnic. CI = 95% confidence interval. NT-proBNP was log-transformed prior to analysis; exponentiated coefficients represent ratios of geometric means. Models are adjusted for baseline confounders.

The effect of childhood social class on health outcomes remained strong after mediation analysis was applied. Among men (Table 4), for self-rated health, the interventional direct effects ($RR \approx 0.78$ – 0.87 across mediators) indicated that holding men’s adult class to the distribution it would take under disadvantaged childhood class, shifting childhood class from disadvantaged to advantaged reduced fair/poor self-rated health by 13–22%. The interventional indirect effects implied that if everyone were born into disadvantaged class, shifting adult class to the distribution observed among those from advantaged childhood class would reduce the risk of fair/poor self-rated health by approximately 10% ($RR = 0.90$, 95% CI: 0.86, 0.95; 39% mediated). Smaller indirect effects were observed via education ($RR = 0.93$, 95% CI: 0.90, 0.96; 27% mediated), housing ($RR = 0.97$, 95% CI: 0.94, 0.99; 11% mediated), and income ($RR = 0.99$, 95% CI: 0.97, 1.00; 4% mediated).

A similar pattern was observed for C-reactive protein. Direct effects remained substantial ($RR \approx 0.63$ – 0.68 across mediators), indicating a strong remaining childhood social class gradient in elevated CRP after accounting for adult SEP among men. Indirect effects were evident via occupational class and education ($RR \approx 0.91$; 14–16% mediated), implying that shifting these adult SEP from the disadvantaged to the advantaged childhood distributions would reduce inflammation risk by around 9%. For cholesterol ratio, glycated hemoglobin (HbA1c), and NT-proBNP, interventional indirect effects were close to the null, suggesting that these childhood social class gradients were largely not captured by adult SEP measure here.

Among women (Table 5), direct effects for self-rated health were also sizable ($RR \approx 0.66$ – 0.69 across mediators), indicating that holding women’s adult class to the distribution it would take under disadvantaged childhood class, shifting childhood class from disadvantaged to advantaged reduced fair/poor self-rated health by 31–34%. Indirect effects were smaller than among men and were most evident via education ($RR = 0.95$, 95% CI: 0.91, 0.98; 10% mediated), with limited mediation via housing (6% mediated) and income (3% mediated), and little evidence via occupational class. For C-reactive protein, direct effects again remained sizeable ($RR \approx 0.62$ – 0.69), with limited mediation, mostly evidently via housing ($RR = 0.97$; 6% mediated). Indirect effects were small for cholesterol and HbA1c.

Table 4 Interventional direct and indirect effects of childhood social class on health outcomes at age 62, men

Mediator	Direct effect	Indirect effect	Overall effect	rPM
Self-rated health RR (CI)				
Class	0.87 (0.73, 1.00)	0.90 (0.86, 0.95)	0.79 (0.67, 0.91)	0.39 (0.15, 1.10)
Housing	0.79 (0.69, 0.90)	0.97 (0.94, 0.99)	0.77 (0.67, 0.87)	0.11 (0.03, 0.29)
Education	0.83 (0.73, 0.93)	0.93 (0.90, 0.96)	0.77 (0.67, 0.87)	0.27 (0.10, 0.52)
Income	0.78 (0.68, 0.88)	0.99 (0.97, 1.00)	0.77 (0.67, 0.88)	0.04 (0.00, 0.11)
C-reactive protein RR (CI)				
Class	0.65 (0.50, 0.86)	0.91 (0.83, 0.99)	0.59 (0.46, 0.76)	0.14 (0.01, 0.50)
Housing	0.63 (0.49, 0.85)	1.00 (0.96, 1.03)	0.62 (0.49, 0.84)	—
Education	0.68 (0.54, 0.93)	0.91 (0.86, 0.96)	0.62 (0.49, 0.84)	0.16 (0.07, 0.43)
Income	0.62 (0.49, 0.85)	1.00 (0.99, 1.01)	0.62 (0.49, 0.84)	—
Cholesterol ratio β (CI)				
Class	-0.15 (-0.30, 0.02)	0.00 (-0.05, 0.05)	-0.15 (-0.27, 0.01)	—
Housing	-0.12 (-0.27, 0.02)	-0.01 (-0.04, 0.00)	-0.14 (-0.28, 0.01)	—
Education	-0.10 (-0.24, 0.05)	-0.04 (-0.07, -0.01)	-0.14 (-0.28, 0.01)	—
Income	-0.14 (-0.28, 0.01)	0.00 (-0.01, 0.01)	-0.14 (-0.28, 0.01)	—
Glycated hemoglobin β (CI)				
Class	-0.10 (-0.22, 0.03)	-0.02 (-0.06, 0.01)	-0.12 (-0.22, -0.01)	—
Housing	-0.14 (-0.24, -0.02)	-0.01 (-0.03, 0.01)	-0.14 (-0.24, -0.03)	—
Education	-0.14 (-0.24, -0.02)	-0.01 (-0.03, 0.02)	-0.14 (-0.24, -0.03)	—
Income	-0.14 (-0.24, -0.04)	0.00 (-0.01, 0.01)	-0.14 (-0.24, -0.03)	—
NT-proBNP estimates β (CI)				
Class	0.03 (-0.07, 0.12)	-0.02 (-0.05, 0.01)	0.01 (-0.09, 0.10)	—
Housing	0.04 (-0.03, 0.14)	0.00 (-0.01, 0.02)	0.04 (-0.03, 0.14)	—
Education	0.04 (-0.04, 0.14)	0.00 (-0.02, 0.03)	0.04 (-0.03, 0.14)	—
Income	0.04 (-0.03, 0.14)	0.00 (-0.01, 0.00)	0.04 (-0.03, 0.14)	—

Notes: RR = risk ratio. CI = 95% confidence interval. rPM = randomised analogue of proportion mediated. Overall effect and rPM are reported for completeness. rPM is reported only when the overall effect is meaningfully different from zero and the interventional direct and indirect effects operate in the same direction with a non-negligible indirect effect.

Table 5 Interventional direct and indirect effects of childhood social class on health outcomes at age 62, women

Mediator	Direct effect	Indirect effect	Overall effect	rPM
Self-rated health RR (CI)				
Class	0.68 (0.59, 0.80)	0.97 (0.93, 1.01)	0.66 (0.57, 0.78)	0.05 (-0.01, 0.16)
Housing	0.67 (0.59, 0.77)	0.97 (0.94, 0.99)	0.65 (0.56, 0.75)	0.06 (0.02, 0.12)
Education	0.69 (0.59, 0.79)	0.95 (0.91, 0.98)	0.65 (0.56, 0.75)	0.10 (0.03, 0.20)
Income	0.66 (0.57, 0.76)	0.98 (0.96, 1.00)	0.65 (0.56, 0.75)	0.03 (0.01, 0.08)
C-reactive protein RR (CI)				
Class	0.62 (0.47, 0.77)	1.04 (1.00, 1.09)	0.65 (0.50, 0.80)	—
Housing	0.68 (0.56, 0.81)	0.97 (0.92, 1.00)	0.66 (0.55, 0.79)	0.06 (0.00, 0.18)
Education	0.69 (0.56, 0.82)	0.96 (0.92, 1.01)	0.66 (0.55, 0.79)	0.07 (-0.03, 0.21)
Income	0.66 (0.54, 0.78)	1.02 (0.99, 1.05)	0.67 (0.56, 0.79)	—
Cholesterol ratio β (CI)				
Class	-0.28 (-0.38, -0.14)	0.00 (-0.03, 0.01)	-0.28 (-0.37, -0.16)	—
Housing	-0.28 (-0.38, -0.15)	-0.01 (-0.03, 0.00)	-0.29 (-0.39, -0.16)	—
Education	-0.29 (-0.39, -0.17)	0.00 (-0.02, 0.02)	-0.29 (-0.39, -0.17)	—
Income	-0.29 (-0.39, -0.17)	0.00 (-0.01, 0.01)	-0.29 (-0.39, -0.16)	—
Glycated hemoglobin β (CI)				
Class	-0.15 (-0.22, -0.06)	0.00 (-0.02, 0.01)	-0.16 (-0.23, -0.07)	—
Housing	-0.13 (-0.21, -0.06)	-0.01 (-0.02, 0.00)	-0.13 (-0.21, -0.07)	—
Education	-0.13 (-0.22, -0.06)	0.00 (-0.02, 0.01)	-0.13 (-0.22, -0.07)	—
Income	-0.13 (-0.21, -0.07)	0.00 (-0.01, 0.00)	-0.13 (-0.21, -0.07)	—
NT-proBNP β (CI)				
Class	0.04 (-0.05, 0.13)	-0.01 (-0.03, 0.00)	0.03 (-0.06, 0.11)	—
Housing	0.02 (-0.06, 0.08)	0.00 (-0.01, 0.01)	0.02 (-0.05, 0.08)	—
Education	0.03 (-0.05, 0.09)	-0.01 (-0.02, 0.01)	0.02 (-0.05, 0.08)	—
Income	0.02 (-0.06, 0.09)	0.00 (-0.01, 0.01)	0.02 (-0.05, 0.08)	—

Notes: RR = risk ratio. CI = 95% confidence interval. rPM = randomised analogue of proportion mediated. Overall effect and rPM are reported for completeness. rPM is reported only when the overall effect is meaningfully different from zero and the interventional direct and indirect effects operate in the same direction with a non-negligible indirect effect.

To examine the robustness of our main findings, we completed several post-hoc analyses. First, we tested alternative outcome specifications: C-reactive protein as a continuous outcome (appendix table A10), cholesterol ratio as a binary outcome (table A11), and HbA1c as a binary outcome (table A12). Second, we examined alternative medication assumptions and restrictions, including alternative statin adjustments for total cholesterol (table A13) and exclusion of participants taking heart-failure-related medications in NT-proBNP analyses (table A14). Third, we imposed sample restrictions, including restriction to a consistent 2022–23 biomarker collection window (tables A15–A16) and restriction of self-rated health analyses to blood-sample providers (table A17). Fourth, we assessed whether income mediation findings were sensitive to alternative operationalization of income, including binary income

measures based on different thresholds (tables A18–A19). Fifth, we examined the sensitivity of income mediation findings to possible measurement error by using income measured at a different timepoint (tables A20–A21). Main results were substantively unchanged in these analyses.

Discussion

Three central findings emerged from our analyses. First, childhood SEP showed strong independent influence on self-rated health, inflammatory marker (CRP), and cardiometabolic biomarkers (cholesterol ratio and HbA1c) more than six decades later. Second, mediation through adult SEP was modest and outcome-specific: adult SEP accounted for some share of the association for self-rated health and inflammatory marker, but little of the association for cardiometabolic biomarkers. Third, mediation differed by adult SEP indicators and sex: among men, occupational class and education explained a sizable share of the association, whereas among women the overall association was stronger but less explained by the adult SEP measures included.

The strong associations between childhood SEP and health were reflected in sizable interventional direct effects. Even under a hypothetical equalisation of adult SEP, a substantial childhood SEP on health gradient remained. This pattern was evident across subjective (self-rated health) and objective (CRP, cholesterol ratio, and HbA1c) health measures, strengthening the interpretation that early-life (dis)advantage has strong independent biological and health consequences that are independent of mid-life adult SEP. These findings align with developmental origins and life-course epidemiological frameworks, which emphasize early-life material and psychosocial conditions as foundational determinants of later-life health¹⁻².

Mediation effect was outcome specific. For self-rated health, indirect effects were evident, particularly through occupational class and education among men and through education and housing tenure among women. There was also some evidence of mediation for C-reactive protein, similarly through education among men and housing among women. For cholesterol ratio, HbA1c, and NT-proBNP, indirect effects were close to null. We think that these results should not be interpreted as evidence that adult SEP has no effect on later-life cardiometabolic health. Rather, they suggest that, under the specific hypothetical shifts in adult SEP considered here, the childhood SEP gradient in these biomarkers would change only modestly, pointing to mechanisms not captured by adult SEP alone.

Our findings are consistent with a broader life-course literature documenting that both childhood and adult SEP matter for health in mid-life⁶⁻⁸. Earlier NCDS analyses at age 44/45 have reported independent and comparably sized associations of childhood and adult social class with midlife cardiometabolic and inflammatory biomarkers in mutually adjusted models^{10,13}. Extending follow up to older age, we found that adult SEP explained only a small proportion of the association between childhood SEP and later-life cardiometabolic outcomes. This suggests that adult SEP may play a more limited mediating role at the threshold of older age than midlife evidence might imply. The same conclusion emerged in simpler mutually adjusted models, in which adjustment for adult SEP only modestly attenuated the associations of childhood SEP (results available upon request), indicating that the limited mediation is unlikely to be an artefact of the interventional approach and instead reflects the underlying pattern in the data at this later life-course stage.

Two potential explanations may account for the weaker role of adult SEP in our study. First, by age 62 cardiometabolic risk may increasingly reflect the long-term biological embedding of childhood disadvantage. Early-life adversity may contribute to cumulative physiological wear and tear that becomes more apparent with age and is not easily offset by later socioeconomic circumstances. Thus, even if adult SEP remains relevant, it may account for a smaller share of the childhood gradient in early older age than has been observed in mid-life. This interpretation should also be considered alongside differences across studies in measures and analytical approach, which may further limit direct comparability with earlier findings. Second, the weaker mediating role of adult SEP may reflect the later life-course stage captured in our analysis. Although socioeconomic differences in survival may become more consequential at older ages, they are unlikely to have heavily influenced our results at age 62, when such processes are probably only beginning to emerge. Even so, they may modestly narrow socioeconomic contrasts and thereby reduce the apparent mediating role of adult SEP.

The contrasting pattern observed across outcomes is informative. A larger share of the childhood SEP gradient in self-rated health was captured by adult SEP. Among objective biomarkers, mediation patterns varied: we observed evidence of mediation for CRP, whereas mediation was limited for cardiometabolic markers. One plausible explanation is that self-rated health is a broad evaluative construct encompassing psychological and mental wellbeing alongside physical functioning¹⁷ and that the dimensions people draw on when rating their health may vary across social groups²⁹. This may make self-rated health more responsive to adult socioeconomic position measured close to outcome assessment. In contrast, cardiometabolic markers reflect longer-term physiological embedding of early life conditions³⁰. Consistent with this interpretation, prior work on mental health has similarly suggested mediation via adult SEP³¹, including evidence from the same cohort used here³². Future research should test whether similar patterns emerge across other health outcomes and physiological systems.

The mediation patterns also differed by adult SEP indicators and sex. Among men, occupational class and education explained a sizable share of the association between childhood SEP and self-rated health and C-reactive protein, suggesting that labor-market position and educational attainment were important pathways linking early-life SEP to later health. Among women, mediation was weaker overall. Occupational class explained little of the association, while education and housing tenure accounted for only a modest share, leaving most of the association between childhood SEP and health not captured by the adult SEP measures considered. The somewhat greater contribution of housing tenure than women's occupational class suggests that material living conditions and household-level advantage may have been more relevant pathways than individual labor-market position for women in this cohort.

In the 1958 cohort, occupational class among men often reflected continuous full-time employment and thus serve as a reasonable proxy for cumulative adult socioeconomic exposure. For women, however, occupational trajectories were often characterized by part-time work, labor-force interruptions, and weaker attachment to the labor market³³. Women's occupational class may therefore have captured their adult material and social circumstances less completely. By contrast, housing tenure, as a household-level indicator, may have better reflected the economic context in which many women in this generation lived, particularly where resources were pooled within couples or mediated through partners.

Given the limited mediation observed for several outcomes, it is likely that pathways beyond the adult SEP measures considered here contribute to the childhood SEP gradient in later-life health. Fundamental cause theory posits that socioeconomic advantage operates through access to flexible

resources, such as knowledge, power, material capacity, and social connections, that enable individuals to deploy varied strategies to protect health as risks and opportunities evolve over time³⁴. From this perspective, the substantial direct effects in our analyses are best interpreted as the remaining childhood SEP and health association not captured by the adult SEP indicator examined, rather than implying an absence of underlying mechanisms. More broadly, early-life socioeconomic conditions pattern cumulative environmental exposures, socially structured stress processes, access to supportive social networks, and interactions with institutions, which may contribute to the remaining association observed here.

Several limitations should be acknowledged. Adult SEP was measured at a single midlife time point, which may understate cumulative socioeconomic exposure across adulthood. Additionally, although the interventional mediation framework improves causal interpretability relative to conventional approaches, it relies on the assumption that all relevant confounders of the exposure–outcome, exposure–mediator, and mediator–outcome relationships have been adequately captured. Given the long temporal span between childhood SEP, adult SEP, and later-life health, some residual confounding from unmeasured early-life or long-term psychosocial and environmental processes can be expected.

Notwithstanding these limitations, this study makes methodological and substantive contributions to life-course epidemiology and ageing research. Drawing on over six decades of prospective data from the 1958 British Birth Cohort, we examined the long-term direct and indirect effects of childhood SEP on health at age 62 operating through adult SEP, capturing inequalities across an unusually long temporal horizon. Through a counterfactual interventional mediation framework, we further moved beyond conventional approaches that rely on strong and often implausible assumptions regarding exposure–mediator independence. This approach allows us to estimate policy-relevant decompositions of the childhood SEP and health association while accommodating exposure-induced mediator–outcome confounding³⁵.

Taken together, our findings suggest that, if policies were able to shift adult SEP at the population level in the manner represented by our hypothetical contrasts, some reduction in later-life health inequalities would be expected, although such shifts are unlikely to fully eliminate the health gradient associated with childhood SEP. In practice, the most realistic levers for shifting adult SEP at scale are population-level policies affecting financial and employment conditions, alongside early-life interventions to reduce disadvantage. The implication of these ‘what if’ contrasts depend on the extent to which comparable shifts in adult SEP are feasible within the prevailing social and policy context³⁶.

Contributors

All authors defined the research question and designed the study. Y.G. performed the statistical analyses and wrote the first draft. All authors critically contributed to the interpretation of the results, revised the manuscript, and approved the final version for publication.

Data sharing

All original datasets from the 1958 NCDS are freely available to download at the UK Data Archive.

Declaration of interests

We declare no competing interests.

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