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The Long Arm of childhood hypothesis and systematic low-grade inflammation: Evidence from parental education of older European adults

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ABSTRACT

Childhood SES has been extensively studied as a predictor for health outcomes in adulthood, though the direct mechanisms remain unclear. The Long Arm of Childhood Model hypothesizes that this process is a chain of events, moderated by numerous factors such as family economic status and environment, health behaviors, as well as biological processes. We expand on this model with objective measures of health in older age, namely C-reactive protein (CRP), as chronic low grade inflammation, which has been found to be connected to both childhood SES as well as a number of cardiovascular diseases in adulthood. Using life history data from SHARE, as well as a novel dried blood spot dataset, we explore the protective role of parent education on the blood level of C-reactive protein in adulthood. Estimating a stepwise linear regression model, we find evidence that years of parental education are negatively associated with CRP in adulthood, with a one-year increase in mother's (father's) years of education decreasing adult CRP by 1.8% (1.1%). Using a modified Sobel test, we measure both the direct and indirect effects, estimating the extent in which later-life mediators significantly alter the relationship between parental education and CRP. While father's education is completely mediated by individual factors such as respondent's education, employment, and health behavior – we observe a lasting association from mother's education, suggesting a direct link between mother's education and CRP in adulthood.

1. Introduction

There is clear evidence of a socio-economic gradient when it comes to the health outcomes of older adults, made more pervasive by the social and economic challenges accompanying demographic aging. More and more, social scientists are turning to life course models to explain these health disparities in aging populations, as there is evidence that the adverse experiences made in childhood are predictive of future negative health outcomes in adulthood. The link between childhood experience and adult health has been well documented in the literature, with researchers connecting childhood conditions such as health, disease and hospitalization rates (Blackwell et al., 2001; Burgner et al., 2015; Haas, 2007), parental income and education (Case et al., 2005; Dowd et al., 2009), stress (Dube et al., 2009), as well as biological factors like low birth weight (Case et al., 2005) to adult health outcomes. This phenomenon has been discussed as the Long Arm of Childhood Model, a theory that childhood experience cycles into adult health through extended, incremental, and often indirect pathways through education, saving and employment choices, health behaviors, as well as biological processes (Hayward & Gorman, 2004; Järvisalo et al., 2002; Pakpahan, Hoffmann, & Kröger, 2017).

While the connection between childhood SES and adult health has been well documented, the underlying biological mechanisms which track these disparities are unclear. One hypothesis is that childhood infections, adversity, or stress may induce the secretion of pro-inflammatory molecules, which in turn stimulate the production of acute phase proteins, such as C-reactive protein (CRP), into the bloodstream. This is the body's natural and protective response mechanism to an illness, damaged tissue, or a "fight or flight" situation, which persists for a limited amount of time until the threat has passed. However, with repeated activation, the body struggles to complete the inflammatory response, known as immune dysregulation, leading to the prolonged presence of inflammatory markers (Fagundes et al., 2013) and chronic low-grade inflammation. Over time, the presence of elevated CRP levels in the bloodstream has been found to be related to negative health concerns, such as cardiovascular disease, diabetes, stroke, hypertension, as well as mental health issues like depression and anxiety (Ridker et al., 1998; Lagrand et al., 1999; Bautista et al., 2001; Anand et al., 2004; Casas et al., 2008).

Others explore the long-term effects of childhood experience,

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independent of specific trauma or disease, on inflammatory markers, such as CRP. Some report an inverse relationship between SES and inflammatory markers, with higher parental education, income, and employment rates predicting lower CRP in both adolescence and adulthood (Muscatell et al., 2020; Cole et al., 2011; Miller et al., 2009). There is evidence that the relationship between SES and CRP starts early, with adolescents who experience adverse events in their childhood already reporting higher CRP levels than their peers (Chiang et al., 2015; Dowd et al., 2010; Schmeer & Yoon, 2016; Schreier et al., 2014). Moreover, a longer exposure to high inflammation in early life leads to an overall longer time spent with higher CRP levels once reaching adulthood (Gurven et al., 2008; Järvisalo et al., 2002).

A putative mechanism that links low SES and elevated CRP is that lower childhood SES is associated with early life stressors such as more negative social experiences, family disruptions, harsher parenting, as well as greater psychological issues in childhood (Lacey et al., 2013, O'Connor et al., 2020). As SES is tightly coupled with educational level, parental education can be used as a proxy for childhood experience. In line, researchers find that parental education is associated with lower CRP in young adults (Brummett et al., 2013), with one study finding that a one-unit increase in parental education is associated with an estimated 35% lower CRP in children, and 19% when including BMI (Schmeer & Yoon, 2016).

However, at the current state of the literature, researchers focus on certain time periods throughout the life cycle, finding mixed results to what extent childhood SES is associated with systemic low-grade inflammation. While some studies find evidence of body mass index (BMI) moderating the relationship between SES and CRP (Schmeer et al., 2016; Slopen et al., 2012; Lacey et al., 2013; Nazmi et al., 2010), to the best of our knowledge, very few studies have put that finding into a larger perspective of a life-cycle model in order to measure both the direct and indirect pathways in which parental education influences CRP in older adulthood (Carmeli et al., 2020). Given the long and interconnected path the Long Arm of Childhood Model hypothesizes, it is likely that a large portion of this effect is indirect. Similarly, very few studies use European data, for a lack of multinational data availability.

Using data from 20,113 individuals in 10 European countries and Israel from SHARE, as well as a large and novel dried blood spot dataset, we explore the direct and indirect pathways in which parental education is related to systematic low-grade inflammation in older adults (50+). We hypothesize that greater parental educational attainment will be inversely related to CRP, though much of the total effect may be working through indirect pathways. Through estimating a stepwise linear regression model, we test for a lasting influence of mother's and father's education with the addition of potential mediating variables: respondent education, economic status, employment, and health behaviors. Taking advantage of a mediation decomposition method, we additionally disentangle the direct and indirect effects of parental education on later-life CRP blood levels. Given the multi-faceted and persisting nature of chronic inflammation, understanding this inflammatory mechanism in which childhood experience cycles into adult health outcomes helps us to better understand the pervasive health inequalities, biological responses to stress, and health fluctuations over the life cycle.

2. Data and methodology

Our sample comes from the biomarker dataset available from the Survey of Health, Ageing, and Retirement in Europe (SHARE). SHARE is a research infrastructure for studying the effects of health, social, economic, and environmental policies over the life course of European citizens and beyond (Börsch-Supan, A. et al., 2013; Börsch-Supan, A. 2022). In its Wave 6 in 2015, SHARE collected dried blood spot (DBS) samples from ca. 27,000 respondents in 12 SHARE countries (Belgium, Switzerland, Germany, Denmark, Estonia, Spain, France, Greece, Israel, Italy, Sweden, and Slovenia) at their place of residence. The inclusion of DBS data aimed to provide further objective measures of health in older adults, measures of which are not yet available in Europe at this scale.

The implementation and the DBS collection in SHARE Wave 6 is described in detail in Börsch-Supan, M. et al., 2020, pp. 47–2020. In brief: Thoroughly trained interviewers collected the blood spots from a simple prick of the finger on special filter cards during the SHARE interview. In each of the affiliated countries, harmonized collection protocols and DBS collection kits were used. Similarly, interviewer and sample monitoring was implemented throughout the fieldwork stage in order to reduce measurement error. After collection, all DBS samples were sent via regular mail to the SHARE biobank in Odense, Denmark, where they were visually inspected for number, size, and quality of the blood spots before storage in freezers at −23 °C until analyzed.

CRP was eluted from a single punch together with the markers total cholesterol, triglycerides, and cystatin C. Laboratory assays were performed according to published techniques (hs-CRP, high-sensitive immunoassay, McDade et al., 2004; Brindle et al., 2010). Assay conditions were adapted at the Department of Laboratory Medicine, University of Washington, Seattle, USA for the SHARE-specific analyses of multiple markers from one punch.

In 2018, a post-field collection validation experiment with non-SHARE donor samples was conducted to validate the DBS results for identified fieldwork conditions in the laboratory (e.g., high temperature, shipment time, humidity protection, and small spot size). Structured conversion formulae were established and applied to the survey-collected DBS to correct the assay results of each marker for the impact of field conditions. (Börsch-Supan, A. et al., 2021).

Our study includes data of 20,113 participants from 11 countries. Due to missing life history information, Greece is dropped from the sample. Despite the mildly invasive procedure, overall participation in blood spot collection was around 72%. We employ non-response adjustment weights to limit the possibility of sample bias. Moreover, we did not observe significant country differences in participation. Participants in DBS collection were required to give informed consent, which could be revoked at any time, including after collection. As is common for ethical survey practice, samples were pseudonymized to protect the identity of the participants.

2.1. CRP

Long-term elevated CRP levels pose a particular risk for cardiovascular disease (Ridker, 2003). Shown in Fig. 1 below, we present the risk categories associated with CRP, with each additional category posing a higher threat to developing CVD (Ridker et al., 1998). While normal CRP levels fall below 1 mg/L, moderate risk falls between 1 and 3 mg/L, and high risk between 3 and 10 mg/L (Ridker, 2003; Pearson et al., 2003; Yeh & Willerson, 2003). As we are primarily focused on low-grade inflammation, we restrict SHARE CRP assay results to <10 mg/L, eliminating the very high values being rather the result of acute infections or underlying health conditions. CRP values are right-skewed, as most respondents fall within the normal to mild-risk (1–3 mg/L) category, and are therefore log-transformed (see Fig. 2).

2.2. Sociodemographics

We include respondent demographic characteristics from the SHARE Wave 6 interview data (Börsch-Supan, A. 2020; Malter and Börsch-Supan (eds.) 2017; Börsch-Supan, A. et al., 2013): Age at the

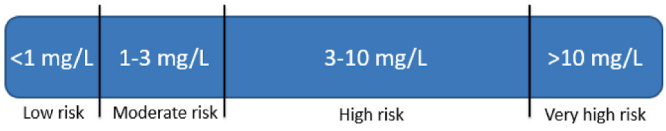

Fig. 1. Description of CRP categories. Clinical interpretation of CRP for CVD risk prediction adapted from Yeh & Willerson, 2003 and Ridker, 2003.

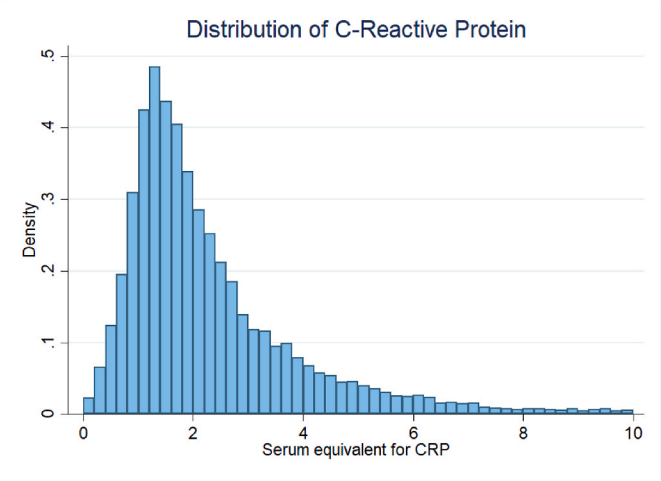

Fig. 2. Distribution of CRP
Note: Restricted to CRP <10 mg/L.

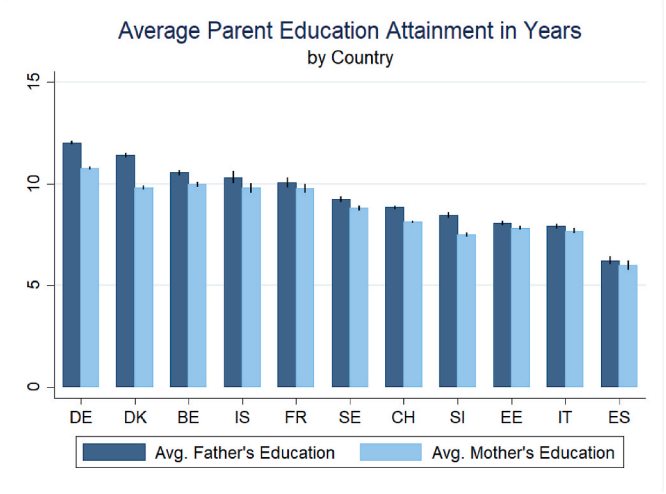

Fig. 3. Average Parent Education Attainment in Years by County
Note: Adjusted by individual cross-sectional weights, confidence intervals shown in brackets.

interview day measured in years; gender as a dummy for female sex; country where the interview was conducted. The current ability to make ends meet is measured by the following: 1 = with great difficulty, 2 = with some difficulty, 3 = fairly easily, and 4 = easily. Employment is categorical variable with 1 = not in active employment, 2 = currently employed, 3 = civil servant, 4 = self-employed. Alongside these demographic characteristics are our main variables of interest, namely educational attainment of both parents as a proxy for childhood SES.

In SHARE Wave 6, information on years of education was only available for the main respondents, coming from an imputed variable where respondents were asked "How many years have you been in full-time education?" In order to construct a "years of education'' variable for each parent from parental ISCED 1997 categories, we first take the average number of respondent years of education by ISCED 1997 classification, gender, and country, as countries may differ in the time required to achieve certain academic achievements. We then apply that average number of education years to each parent, based on their corresponding ISCED 1997 classification. However, for ease of interpretation of the mediation analysis, we choose to use the continuous variable, years of education.

In our sample, 6.3% (8.8%) of mother's (father's) ISCED 1997 education was missing. In order to allow for the single parent case, we set missings to the average educational attainment (ISCED category = 2) and employ missing dummies for mother's and father's education. These dummies are never significant in any model.

In Fig. 3, we present the average years of mother's and father's education by country from the constructed variable used above.

2.3. Health behavior

There is ample evidence that some health behaviors are related to elevated inflammation (Brummett et al., 2013; Tracy et al., 1997; Fröhlich et al., 2003; Kohut et al., 2006; Visser et al., 1999; Park et al., 2005). We included five self-reported measures of health behavior: (1) alcohol consumption, (2) smoking, (3) fruits and vegetables consumption, (4) physical inactivity, and (5) BMI. Alcohol is a dichotomous variable taking the value 1 if the respondent reported having at least one alcoholic beverage in the last 7 days. Smoking is also a dichotomous variable taking the value 1 if a respondent answered yes to "Have you ever smoked daily?" and 0 otherwise. We measure consumption of fruits and vegetables as such: 1 = Every day, 2 = 3–6 times a week, 3 = Twice a week, 4 = Once a week, 5 = Less than once a week. Physical inactivity is a dichotomous variable taking the value of 1 if the respondent reported that they "hardly ever, or never" participated in sports or activities that are vigorous. BMI is calculated as the standard weight (kg)/height (m^2), and we choose to keep BMI continuous.

2.4. Statistical analysis

$$Y_{Log\ CRP} = \gamma_1 + \tau_1 Mother\ Educ + \tau_2 Father\ Educ + X_i + \varepsilon_1 \quad (1)$$

$$M_{Resp\ Educ} = \gamma_2 + \alpha_1 Mother\ Educ + \alpha_2 Father\ Educ + X_i + \varepsilon_2 \quad (2)$$

$$Y_{Log\ CRP} = \gamma_3 + \tau_1' Mother\ Educ + \tau_2' Father\ Educ + \beta_1 M_{Resp\ Educ} + X_i + \varepsilon_3 \quad (3)$$

$$M_{Resp\ Health} = \gamma_4 + \alpha_3 Mother\ Educ + \alpha_4 Father\ Educ + \alpha_5 M_{Resp\ Educ} + X_i + \varepsilon_4 \quad (4)$$

$$M_{Resp\ SES} = \gamma_5 + \alpha_6 Mother\ Educ + \alpha_7 Father\ Educ + \alpha_8 M_{Resp\ Educ} + X_i + \varepsilon_5 \quad (5)$$

$$Y_{Log\ CRP} = \gamma_6 + \tau_1'' Mother\ Educ + \tau_2'' Father\ Educ + \beta_2 M_{Resp\ Educ} + \beta_3 M_{Resp\ Health} + \beta_4 M_{Resp\ SES} + X_i + \varepsilon_6 \quad (6)$$

We conduct a nested multiple mediation analysis to test the direct and indirect pathways in which parental education influences respondent CRP in adulthood. Equations (1)–(6) present our estimation strategy, where $Y_{Log\ CRP}$ represents respondent CRP, M represents the three mediation pathways (via respondent education, health, and SES), and X_i represents the vector of control variables (age, gender, and country). The full model, Equation (6), is shown graphically in Fig. 4.

Equation (1) shows the baseline model, or the total effect (τ) of parent education on CRP. In the theoretical framework proposed, parental education works through mediators sequentially, first through respondent education in Equation (3), and later through all proposed mediators the full model, Equation (6). Therefore, the indirect effect of parental education working through respondent education is estimated by ($\tau - \tau'$), and the indirect effect of parent education through respondent education, health, and SES by ($\tau - \tau''$).

While we may expect a large portion of the total effect to be driven by the mediators, the direct effect (τ'') represents the residual "unexplained" effect stemming from parental education alone, capturing the extent to which early childhood experience remains present in adult CRP.

We estimate whether the change in the magnitude of mother's and

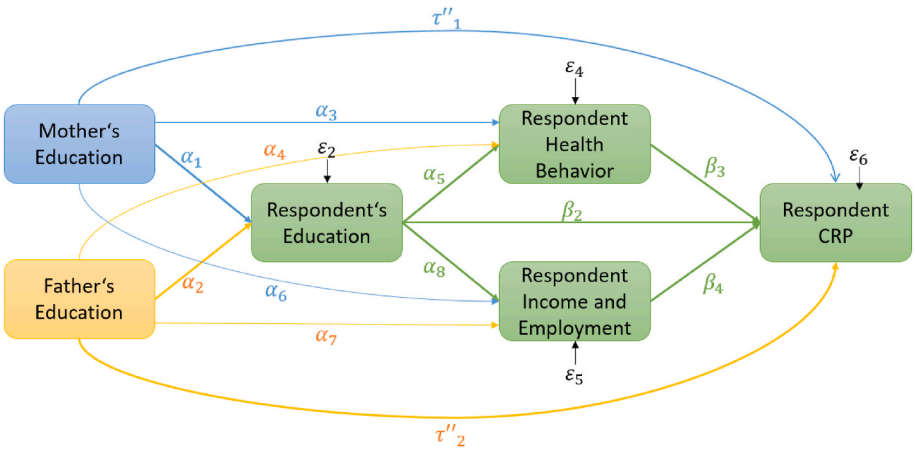

Fig. 4. Life-cycle model linking parent education and CRP

father's education once including potential mediator variables is significant using a modified Sobel test (Sobel 1986), allowing us to examine multiple mediators simultaneously. Analysis was conducted in Stata using the khb estimation command (Kohler et al., 2011, Karlson et al., 2012). As CRP is log-transformed, the main coefficients of interest are exponentiated and are therefore to be interpreted as the percent change in CRP.

Our analysis also uses calibrated cross-sectional individual weights available from SHARE Wave 6 to reduce bias due to sampling issues (Malter & Börsch-Supan 2017; De Luca & Rossetti, 2018). However, a non-response adjustment factor was also constructed to supplement this weight, as those who decide to participate in blood collection are a non-random sample, and therefore require further adjustment to reduce systematic bias. Using an inverse probability weighting method, we first estimated a logistic regression to predict the probability of consenting to DBS collection, given DBS eligibility. As such, those who were willing to participate in collection, yet produced no useable blood data, were still considered as participants. This model included demographic characteristics: age, gender, education level, and country dummies. We inversed the predicted probability produced by this model, creating a non-response adjustment factor, and multiplied these values to the calibrated cross-sectional individual weights from SHARE Wave 6. Weights are applied as probability weights in each model.

Some of the health behavior variables (BMI, smoking, and physical inactivity), respondent education, and the variable for financial distress are pulled from generated dataset that imputed missing values (De Luca & Rossetti, 2018).

Key characteristics of the sample are shown in Table 1 below, including the means and proportions of our main variables of interest, potential mediators, and demographic characteristics.

3. Results

Results from the stepwise linear regression in Table 2 indicate a significant and inverse relationship between parent education and inflammation. Across each model iteration, we observe that mother's education remains significant and negatively associated with log CRP. Even with the inclusion of all mediator variables in Model (3), a one-year increase in mother's education (ß = −0.0109*, 95% CI -0.020 to −0.001) still shows a 1.1% decrease in CRP in older adulthood. Father's years of education, however, is only significant in the baseline model (ß = −0.011**, 95% CI -0.019 to −0.002). In sum, mother's education seems to have a larger direct effect on CRP in adulthood in comparison to father's education.

With the inclusion of respondent education in Model (2), we observe that a one-year increase in respondent education years is associated with a 1.3% decrease in adult CRP levels. In the full Model (3), we observe that respondent education remains a significant and negative predictor (ß = −0.005**, 95% CI -0.008 to −0.000) of CRP. The ability to make ends meet is also significant and negatively associated with CRP, with those who can "Easily" make ends meet having 8.8% lower CRP on average than their "With great difficulty" counterparts. Similarly, being in paid employment is associated with a 7.1% decrease in CRP, a result that fits the idea that those out of employment are generally older and sicker. Lastly, the inclusion of the health behavior variables in Model (3) decrease the magnitude of previous coefficients, as they are (for the most part) highly significant and fall in the predicted directions. The largest predictors of increased CRP in adulthood are smoking and physical inactivity, which are associated with a 6% and 7.2% increase in CRP, respectively. BMI is also highly significant, with a one-unit increase in BMI increasing CRP in adulthood by 4%. Since the inclusion of covariates in Models (2) and (3) reduce the magnitude and significance of our main coefficients of interest, we next disentangle their statistical contribution as mediators.

In the first column of Table 3, we observe a highly significant reduction in both mother's and father's education between Model (2) and Model (1), shown by the indirect effect of mother's education (ß = −0.005**, 95% CI -0.007 to −0.003) and father's education (ß = −0.006***, 95% CI -0.008 to −0.003) working through the mediator, respondent years of education. We additionally estimate how much of the total effect is captured through the indirect effect of parental education working through respondent education to ultimately influence CRP. We observe that a larger proportion of father's education (55.3%) is working through this indirect pathway vs. mother's education (27.2%) (see Table 3).

When we compare the full Model (3) to the baseline Model (1), a similar pattern emerges. We observe a highly significant decrease in our main coefficients of interest with the inclusion of health, employment, and financial distress variables. The indirect effect of mother's education (ß = −0.007***, 95% CI -0.011 to −0.004) working through these mediator variables accounts for 38.2% of the total effect, meaning there is a larger direct (i.e. unexplained) influence of mother's education years on adult CRP outcomes. The role of father's education on CRP, however, is entirely explained by the predicted indirect pathway. We observe that indirect effect of father's education (ß = −0.013***, 95% CI -0.017 to −0.009) accounts for 119.9% of the total effect. While not shown in Table 3, we further decompose the role of each mediator variable in Table 6, shown in the appendix. While the indirect effect of mother's education is somewhat evenly split between SES mediators (ß = −0.003**, 95% CI -0.005 to −0.001) and health mediators (ß = −0.004**, 95% CI -0.007 to −0.001), we observe a large and highly significant indirect effect of father's education working through health mediators (ß = −0.009**, 95% CI -0.012 to −0.006) specifically. BMI (ß = −0.008***, 95% CI -0.011 to −0.006) is especially responsible for the

Table 1
Descriptive statistics.

Study Sample (n = 20113)			
Variable	Mean (SD)	N (%)	Min - Max
CRP	2.35 (1.53)		.003–9.99
<1 mg/L		3316 (16.49)	
1–3 mg/L		11,858 (58.96)	
>3 mg/L		4939 (24.56)	
Log CRP	.63 (.70)		−5.69–2.30
Mother Years of Education	8.79 (2.56)		5–21
Father Years of Education	9.52 (2.99)		5–22
Age in 2015	68.20 (9.31)		51–102
Gender	.56 (.50)		0–1
Female		11,302 (56.19)	
Male		8811 (43.81)	
Respondent Years Education	11.48 (4.30)		0–25
Financial Distress	3.05 (.98)		0–4
With great difficulty		1631 (8.11)	
With some difficulty		4447 (22.11)	
Fairly easily		5306 (26.38)	
Easily		8726 (43.40)	
Employed	.60 (.91)		0–3
Not in active employment		12,777 (63.53)	
Employee		3744 (18.61)	
Civil Servant		2430 (12.08)	
Self-employed		1162 (5.78)	
BMI	26.90 (4.55)		13.79–53.33
Overweight (25–30):		8298 (41.26)	
Obese (>30):		4352 (21.64)	
Ever smoke cigarettes daily?	.47 (.50)		0–1
Yes		9501 (47.24)	
No		10,612 (52.76)	
Have you had alcohol in the last 7 days?	.59 (.49)		0–1
Yes		11,845 (58.89)	
No		8268 (41.11)	
Physical Inactivity	.08 (.28)		0–1
Fruit and Vegetable Consumption	1.27 (.67)		0–5
Every Day		16,378 (81.43)	
3–6 times a week		2656 (13.21)	
Twice a week		633 (3.15)	
Once a week		261 (1.30)	
Less than once a week		185 (.92)	
Country			
Germany		2526 (12.56)	
Sweden		2244 (11.16)	
Spain		1068 (5.31)	
Italy		1489 (7.4)	
France		350 (1.74)	
Denmark		2397 (11.92)	
Switzerland		1707 (8.49)	
Belgium		2704 (13.44)	
Israel		687 (3.42)	
Slovenia		1841 (9.15)	
Estonia		3100 (15.41)	

Note: SD = standard deviation.

Table 2
Results from OLS estimation.

VARIABLES	Model (1)	Model (2)	Model (3)
	Log CRP	Log CRP	Log CRP
Mother Years of Education	**−0.0183*** **	**−0.0137** **	**−0.0109***
	(0.00506)	(0.00508)	(0.00486)
Father Years of Education	**−0.0108***	−0.00525	0.00207
	(0.00446)	(0.00458)	(0.00445)
Age in 2015	0.00520***	0.00394***	0.00298**
	(0.00100)	(0.00101)	(0.00114)
Female	0.0146	0.00524	0.0411*
	(0.0181)	(0.0180)	(0.0180)
Respondent Years Education		−0.0136***	−0.00551*
Make Ends Meet:		(0.00247)	(0.00237)
2. With Some Difficulty			−0.0635
			(0.0381)
3. Fairly Easily			−0.0843*
			(0.0365)
4. Easily			−0.0927*
Employment status:			(0.0368)
1. Employee			−0.0733**
			(0.0283)
2. Civil Servant			−0.115
			(0.0334)
3. Self Employed			−0.0408
			(0.0398)
BMI			0.0404***
			(0.00197)
Ever Smoke			0.0600***
			(0.0178)
Alcohol			−0.0441*
			(0.0180)
Physical Inactivity			0.0752*
Fruit and Vegetable Consumption:			(0.0324)
2. 3–6 Times a week			0.0225
			(0.0243)
3. Twice a week			0.0509
			(0.0533)
4. Once a week			0.0816
			(0.0625)
5. Less than once a week			−0.0425
			(0.101)
Constant	0.625***	0.767***	−0.397***
	(0.0942)	(0.0962)	(0.132)
Country Dummies	X	X	X
Missing Parent Educ Dum	X	X	X
Observations	20,113	20,113	20,113
Adj R-Squared	.0346	.0405	.1303

Note: All models control for the country, whether parent education was missing, and individual-level weights. Model (1) presents the baseline results, whereas Model (2) includes respondent's own education. In Model (3), own employment, income, and health behaviors are included. Robust standard errors in parentheses. (*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.)

reduction in the father's years of education coefficient, capturing around 72% of the overall mediating effect. Therefore, consistent with related research (Schmeer & Yoon, 2016; Brummett et al., 2013), we also find evidence for partial moderation via BMI, though this result is especially true for father's years of education.

4. Discussion and conclusion

With evidence linking elevated inflammation to both early life stress and CVD in older age, we may expect CRP to be a crucial component of the underlying biological mechanisms connecting childhood SES and health outcomes in adulthood. The present study uses DBS data from SHARE to examine the associations between parent years of education and inflammation, based on CRP-blood level, in older adults. While mother's education shows a significant direct association with CRP in adulthood, through mediation analysis we observe a significant indirect effect of both mother's and father's years of education through respondent education, financial status, and health behavior. We therefore observe a protective influence of parental education, with each additional year of mother's (father's) education decreasing CRP in adulthood by 1.8% (1.1%). While these percentages may appear smaller at first glance, if we consider the average difference between pre-primary education (ISCED 1997 category = 0) and first-stage tertiary education (ISCED 1997 category = 5) of approximately 9 years, we would expect a 16.2% decrease in CRP for respondents with highly

Table 3
Results from linear decomposition.

	Respondent Education Model (2) vs (1)		Respondent Health and SES Model (3) vs. (1)	
	Mother's Education	Father's Education	Mother's Education	Father's Education
Total Effect	−0.018 (−.028 -.008) ***	−0.011 (−.019 to −.002)**	−0.018 (−.028 -.008) ***	−0.011 (−.019 to −.002)**
Direct Effect	−0.014 (−.024 to −.004)**	0.005 (−.014 – .004)	−0.011 (−.020 to −.001) *	.002 (−.007 – .011)
Indirect Effect	−0.005 (−.007 to −.003)***	−0.006 (−.008 to −.003)***	−0.007 (−.011 to −.004)***	−.013 (−.017 to −.009)***
Mediating Effects (%)	27.2%	55.3%	38.2%	119.9%

Note: 95% Confidence intervals in parentheses. *p < 0.05; **p < 0.01; ***p < 0.001. Values over 100% in father's education represent complete mediation, and result from the sign change of a non-significant direct effect.

educated mothers and a 9.9% decrease for fathers. Given the linear model estimated, these effects could be considered additive in the case that both parents achieved tertiary education. In this case of two highly educated parents, we would expect around a 26.1% decrease in respondent CRP vs. their lower educated parental counterparts. Given that partners tend to match closely on education (Mare, 1991), we may believe that the health inequalities driven by childhood SES differences to be stark.

Further, this decomposition strategy of studying the direct and indirect pathways in which parental education influences CRP in older adults lends credence to the theory of the Long Arm of Childhood, a model in which early childhood experiences incrementally and collectively influence health outcomes over the life cycle. We find evidence that 38.1% of the total effect of mother's education on CRP is mediated through respondent characteristics indirectly, whereas the effect for father's education is completely mediated. We find evidence that BMI is an especially strong mediator for father's education, capturing around 72% of the overall mediating effect. While we observe a strong indirect effect for both mother's and father's education, the lasting significant direct effect for mother's education shows a residual unexplained effect on CRP driven by childhood experience alone.

Based on previous estimations of the relationship between parent education and CRP in adolescents (Schmeer & Yoon, 2016), we do not find evidence that this relationship is heightened over the lifecycle. However, we do find evidence of a lasting influence of parent education on CRP well into late adulthood that is of a similar magnitude to what is already found in childhood. With previous findings discussing the role of time spent in an elevated inflammatory state (Gurven et al., 2008), we may expect elevated CRP over a long period to further increase the risk of developing CVD. However, direct comparisons to younger populations should be done with caution, as there are likely unexplained cohort effects.

While we do not assume causal ordering when it comes to the present-day mediators, in perhaps a more realistic setting, we may expect these variables to occur incrementally. For example, though it is likely that respondent education predated current respondent employment and health behavior, it is up for discussion when respondent employment and respondent health should enter the model (shown in Fig. 4), as the timing is likely non-uniform across participants. Though the ability to make ends meet likely influences one's ability to participate in healthy behaviors (such as nutrition and physical activity), it could also be such that health behaviors (especially smoking or drinking alcohol) are related to the ability to maintain employment. However, as these mediators are causally downstream from parental education, there is less of a concern for a bias in the total effect. As such, our preference is to include these mediator variables simultaneously.

Findings from (Breen et al., 2013) as well as (Imai et al., 2010) suggest potential for causal mediation analysis under the sequential ignorability assumption, which relies on two assumptions. First: The predictor variable is conditionally independent of unobservables, given background covariates. Second: The mediator variable is conditionally independent of unobservables, given background covariates and predictor variable.

In the case of the first assumption, in order for our model to claim causality we would have to assume that parental education is randomly assigned. While individuals cannot self-select into the family in which they are born, it may be the case that parental education influences fertility and therefore likelihood of being in the sample. Similarly, we do not observe the pretreatment confounders, likely grandparent education. However, as we are primarily concerned with childhood experience through SES, missing pretreatment information, such as grandparent education, likely influences parent education but does not moderate the total effect between childhood experience and adult CRP. Another potential pretreatment confounder is parent migration status, as having a migration background may be related to parent education and childhood socioeconomic standing in the country birth. However, Table 9 shows that the inclusion of the interaction of mother and father migration status does not significantly alter the main coefficients of interest.

The second is a much stronger assumption, and assumes that the mediator is statistically independent of potential confounders on the relationship to the outcome. Even after controlling for the treatment and observed covariates, there is always a possibility of unobserved variables. In the next paragraphs, we will discuss and test these potential confounders to the best of our ability.

First, CRP is altered by various medications and health conditions, which were not included in the main regression either due to lack of information or endogeneity issues. Many respondents in our age group, especially older men, are at risk of CVD and are prescribed statins for lowering LDL-cholesterol. Statins are also able to reduce CRP levels (Asher & Houston, 2007). In our sample, we observe an educational gradient when it comes to statins, with lower educated individuals being more likely to take statins. If lower educated individuals artificially lower their CRP levels with the use of statins, it may be that our coefficients on education are underestimated. As a further check, we include medications for high blood cholesterol (statins) and nonsteroidal anti-inflammatories (such as ibuprofen or aspirin), shown in Table 5. The inclusion of these medications do not significantly alter the main coefficients of interest, nor do they explain a large portion of the variance in CRP (R squared: 0.002). Moreover, including these medications in the main model is a complex topic, as their influence on CRP can depend on the original level of circulating CRP, the specific drug and its dosage, and the health condition that drug is intended to target (Tarp et al., 2012). Their inclusion may also present a reverse causality issue if the mediation is targeting a health condition from Wave 6 which is a risk factor related to elevated inflammation. Upon investigation of statins and other nonsteroidal anti-inflammatory drugs, we do not observe a change to our results, and therefore choose not to include them in the main estimation table.

In a similar vein, our sample contains respondents who already have diagnosed cardiovascular and various inflammation-related conditions. In our sample of older adults, there are cases of heart attack (11.1%), stroke (3.2%), high blood cholesterol (24.6%), hypertension (41.2%), and diabetes (13.4%). While elevated CRP is a risk factor for CVD, it is unclear if having a cardiovascular condition would exacerbate immune dysregulation further, or similarly, if a cardiovascular condition is indicative of poor health behaviors which are responsible for elevated CRP levels. For this reason, including respondent health conditions in the model would warrant a reverse causality issue, if we were to believe in a vicious cycle between systematic low-grade inflammation and CVD. As a further check of our model's validity, we tackled this issue in two ways. First, we restricted the sample to those who have not been

diagnosed with any of the inflammation-related conditions mentioned above (8629 respondents), and found similar results in significance and magnitude for mother's education, but no significance for father's education. Secondly, we took advantage of the SHARE panel, and added whether a respondent already had a diagnosed inflammation-related condition in 2013 (SHARE Wave 5) into the model, two years before CRP information was collected during Wave 6. While we cannot observe CRP in this time period, a nonsignificant change in the main coefficients of interest would lend credence that our results are not biased due to the presence of unobserved CVD in the sample. Shown in Tables 7 and 8, we find no significant difference between Model (2) and Model (3), showing that the inclusion of Wave 5 cardiovascular conditions and associated drugs does not significantly change the coefficients on mother's and father's years of education. With these findings, as well as the findings from the split-sample analysis, we are assured that our results are not driven by the presence of cardiovascular and inflammatory-related conditions in Wave 6.

It is therefore our claim, should the true model be as Fig. 4 describes, that our analysis passes the conditions for causal mediation analysis, as further sensitivity analysis showed no signs that potential confounders disrupt the model. Moreover, we include reasonable controls, which capture remaining variance in CRP.

However, the potential for measurement error remains a topic of discussion. In an ideal setting, we would have a years-of-education measure for each parent, not one which is constructed based on respondent ISCED 1997 classification, gender, and country averages. However, the dummy variables which captured missing parental information were never significant in any model. Second, a replication of Table 2 using parent ISCED 1997 categories produced similar results (shown in Table 4), and provided evidence that our main findings may be in fact slightly underestimated.

Given that the data collected for education variables is retrospective, we may worry about recall bias of respondents. While recall error is an important consideration, in an application of SHARE life history data (Garrouste & Paccagnella, 2011), find that SHARE respondents are quite consistent reporters (with less than 10% recall errors over all events). Despite this, we acknowledge that childhood SES is a multidimensional indicator that cannot be fully captured by parent education. In a further robustness check, we include the number of books in childhood as well as the rooms in one's childhood home into the baseline model. Shown in Tables 10 and 11, while childhood books and rooms show a significant association with CRP, we also find evidence that these childhood SES indicators are significantly altered by parent education. As such, books and rooms act similarly as mediators, and therefore their omission does not change the overall total effect of parent education on CRP.

In an ideal setting, we would be able to observe CRP on multiple occasions throughout the life cycle to better understand the extent of elevated low-grade inflammation. However, CRP is a relatively recent biomarker of interest, being discovered in the 1930s, further studied in the 1970s, with population data only becoming available in the late 1990s, and high-sensitivity measurement only available since ca. 2000 (Ridker, 2009). Therefore, a longitudinal study following respondent CRP values from childhood to older adulthood is not currently possible. Despite the drawbacks of one wave of data, studies show that while CRP is generally increasing over the life cycle, it is relatively stable and reliable, which is why CRP is often used in this context (Myers et al., 2004).

Lastly, there is always a possibility that our sample is biased due to attrition, as we may have lost respondents who were especially sick as a result of or connected to our main variables of interest. However, the use of weights limits this possibility.

Despite these limitations, the analysis has a valuable addition in the literature as the first large multinational European study using biomarkers to explore the role of parental education on the CRP levels of older adults. Through a life-cycle approach, we showed a lasting and significant connection between parental education and the CRP levels of older adults, in both direct and indirect pathways. On one hand, the protective nature of a secure childhood experience seems to promote healthy behavior and outcomes in the long run. On the other hand, a less secure childhood environment may act as a potential health trap. In the context of the Long Arm of Childhood Model, this childhood insecurity may form the negative feedback loops, which gradually and systematically affect chronic low-grade inflammation over the life cycle. If individuals are able to overcome their childhood adversities through education, there is evidence of a positive trend in health outcomes. While the benefit of higher childhood SES is evident, educational and health interventions may be possible later in the life cycle to safeguard against developing cardiovascular diseases. In order to combat rising cardiovascular disease in Europe, policy makers should therefore not only focus on educational and health interventions in adulthood, but specifically target childhood poverty by uplifting parents, especially mothers. Future researchers may expand on this work by studying other objective health measures available in SHARE, such as further measures for CVD, all cause morality, and cognitive decline.

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Ethical statement

Ethical approval for dried blood spot collection was conducted at the country level due to varying legal, ethical, and administrative requirements. Each of the included countries met their country-level requirements from their ethical committees (for full ethical consideration summary, see: Schmidutz 2016; Schmidutz et al., 2013; Schmidutz & Weiss 2017).

Declaration of competing interest

None.

Data availability

The authors do not have permission to share data.

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Appendix

Table 4
OLS Results using ISCED 1997 Education Categories.

VARIABLES	Model (1)	Model (2)	Model (3)
	Log CRP	Log CRP	Log CRP
Mother Education			
ISCED class. 1	−0.0930***	−0.0793**	−0.0793**
	(0.0337)	(0.0335)	(0.0328)
ISCED class. 2	−0.0963**	−0.0766*	−0.0527
	(0.0426)	(0.0422)	(0.0412)
ISCED class. 3	−0.128***	−0.0987**	−0.0785*
	(0.0428)	(0.0428)	(0.0414)
ISCED class. 4	−0.182**	−0.137	−0.132
	(0.0831)	(0.0831)	(0.0844)
ISCED class. 5	**−0.221****	**−0.184****	**−0.147****
	(0.0515)	(0.0514)	(0.0492)
ISCED class. 6	−0.135	−0.0854	−0.0793**
	(0.296)	(0.309)	(0.0328)
Father Education			
ISCED class. 1	−0.135	−0.0854	−0.103
	(0.296)	(0.309)	(0.278)
ISCED class. 2	−0.0153	0.00137	0.0360
	(0.0360)	(0.0359)	(0.0352)
ISCED class. 3	−0.0578	−0.0259	−0.00549
	(0.0432)	(0.0433)	(0.0441)
ISCED class. 4	−0.0265	0.00874	0.0348
	(0.0408)	(0.0415)	(0.0416)
ISCED class. 5	−0.00567	0.0278	0.0661
	(0.103)	(0.0998)	(0.0886)
ISCED class. 6	**−0.108****	−0.0530	0.0188
	(0.0452)	(0.0465)	(0.0461)
Constant	0.450***	0.639***	−0.528***
	(0.0826)	(0.0876)	(0.118)
Country Dummies	X	X	X
Missing Parent Educ Dum	X	X	X
Respondent Education		X	X
Respondent Employment			X
Respondent Health			X
Observations	20,113	20,113	20,113
R-squared	0.037	0.042	0.131

Note: Replication of Table 2 using ISCED 1997 categories of education. Robust standard errors in parentheses, *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

Table 5
Model (3) Including statins and nonsteroidal anti-inflammatory drugs.

VARIABLES	Model (3)
	Log CRP
Mother Years Education	**−0.0112***
	(0.00486)
Father Years Education	0.00223
	(0.00445)
Age in 2015	0.00339**
	(0.00114)
Female	0.0373*
	(0.0180)
Respondent Education	−0.00556*
	(0.00236)
Statins	−0.0824***
	(0.0193)
NSAIDs	0.0297
	(0.0246)

(*continued on next page*)

Table 5 (*continued*)

VARIABLES	Model (3)
	Log CRP
Constant	−0.424***
	(0.132)
Country Dummies	X
Missing Parent Educ Dum	X
Respondent Employment	X
Respondent Health	X
Observations	20,107
R-squared	0.133

Note: Replication of Table 2 Model (3) including prescription drug usage. NSAID = nonsteroidal anti-inflammatory drugs. Robust standard errors in parentheses, *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

Table 6
Results from full linear decomposition.

	Respondent Education Model (2) vs (1)		Respondent Health Model (3) vs. (1)	
	Mother's Education	Father's Education	Mother's Education	Father's Education
Total Effect	−0.018 (−.028 -.008)***	−0.011 (−.019 to −.002)**	−0.018 (−.028 -.008)***	−0.011 (−.019 to −.002)**
Direct Effect	−0.014 (−.024 to −.004)**	0.005 (−.014 – .004)	−0.011 (−.020 to −.001) *	.002 (−.007 – .011)
Indirect Effect	−0.005 (−.007 to −.003)***	−0.006 (−.008 to −.003)***	−0.007 (−.011 to −.004)***	−.013 (−.017 to −.009)***
Mediating Effects (%)	27.2%	55.3%	38.2%	119.9%
Estimated Value (Components of Indirect Effects)				
All SES Mediators:			**−0.003 (-0.005 - -0.001)****	**−0.004 (-0.006 - -0.002)*****
Respondent Education			−0.002 (−0.004 to −0.000)*	−0.002 (−0.004 to −0.000)*
Work			−0.000 (−0.001 – 0.001)	−0.000 (−0.001 – 0.000)
Financial Distress			−0.001 (−0.002 to −0.000)*	−0.001 (−0.002 to −0.000)*
All Health Mediators:			**−0.004 (-0.007 – -0.001)****	**−0.009 (-0.012 - -0.006)*****
BMI			−0.003 (−0.006 to −0.000)*	−0.008 (−0.011 to −0.006)***
Smoking			−0.000 (−0.001 – 0.000)	0.000 (−0.000 – 0.001)
Alcohol			−0.000 (−0.001–−0.000)	−0.001 (−0.001 to −0.000)*
Physical Inactivity			−0.000 (−0.001 – 0.000)	−0.000 (−0.001 – 0.000)
Fruit and Vegetable Consumption			−0.000 (−0.000 – 0.000)	−0.000 (−0.000 – 0.000)

Note: Full decomposition of Table 3. 95% Confidence intervals in parentheses. *p < 0.05; **p < 0.01; ***p < 0.001.

Table 7
OLS results with lagged CVD.

VARIABLES	Model (1)	Model (2)	Model (3)	Model (4)
	Log CRP	Log CRP	Log CRP	Log CRP
Mother Years Education	**−0.0178*****	**−0.0128***	**−0.0130***	**−0.0108***
	(0.00539)	(0.00541)	(0.00521)	(0.00511)
Father Years Education	**−0.0111***	−0.00552	0.00173	0.00294
	(0.00460)	(0.00472)	(0.00460)	(0.00454)
Age in 2015	0.00514***	0.00391***	0.00448***	0.00303**
	(0.00103)	(0.00104)	(0.00106)	(0.00120)
Female	0.0186	0.00875	0.0468*	0.0371*
	(0.0186)	(0.0185)	(0.0190)	(0.0188)
Resp. Years Education		−0.0139***	−0.00825***	−0.00560*
CVD:		(0.00255)	(0.00247)	(0.00243)
Heart attack since w5			0.0704*	0.0595*
			(0.0293)	(0.0292)
Hypertension since w5			0.0549**	0.0372
			(0.0197)	(0.0191)
High blood chol. W5			0.0146	0.0180
			(0.0287)	(0.0278)
Stroke since w5			−0.0110	−0.0256
			(0.0470)	(0.0475)
Diabetes since w5			0.00356	−0.0208
CVD Drugs:			(0.0264)	(0.0250)
Statins since w5			−0.118***	−0.117***
			(0.0301)	(0.0288)
NSAIDs since w5			0.0404	0.0194
			(0.0251)	(0.0243)
Constant	0.626***	0.765***	0.770***	−0.410***
	(0.0956)	(0.0975)	(0.0965)	(0.134)

(*continued on next page*)

Table 7 (*continued*)

VARIABLES	Model (1)	Model (2)	Model (3)	Model (4)
	Log CRP	Log CRP	Log CRP	Log CRP
Country Dum	X	X	X	X
Missing Parent Educ Dum	X	X	X	X
Resp. Health W5			X	X
Resp. Health W6				X
Resp. Empl W6				X
Observations	18,774	18,774	18,774	18,774
Adj R-Squared	.035	.041	.115	.137

Note: All models control for the country, whether parent education was missing, and individual-level weights. Model (1) presents the baseline results. Model (2) includes respondent's own education. In Model (3), Wave 5 (w5) CVD conditions and health behaviors are included. Model (4) includes Wave 6 (w6) mediators. Robust standard errors in parentheses. **p < 0.05; **p < 0.01; ***p < 0.001.*

Table 8
Linear decomposition results – with CVD.

	Respondent CVD Health Model (3) vs. (2)	
	Mother's Education	Father's Education
Total Effect	**−0.0128** (−.023 -.002)*	−.005 (−.014 – .003)
Direct Effect	**−0.0130** (−.023 to −.003) *	.002 (−.007 – .011)
Indirect Effect	0.000 (−.003–.003)	−.007 (−.010 to −.004)***
Mediating Effects (%)	0%	131.1%
Estimated Value (Components of Indirect Effects)		
All W5 CVD conditions:	**0.000 (-0.000–0.001)**	**−0.001 (-0.001–0.000)**
All W5 drugs (Statins and NSAIDS):	**0.000 (-0.001–0.001)**	**−0.001 (-0.001–0.000)**
All W5 health conditions:	**0.000 (-0.003–0.003)**	**−0.006 (-0.008 - -0.003)*****

Note: Decomposition Results of Table 7. 95% Confidence intervals in parentheses. *p < 0.05; **p < 0.01; ***p < 0.001.

Table 9
OLS results with parent migration status.

VARIABLES	Model (1)	Model (2)	Model (3)
	Log CRP	Log CRP	Log CRP
Mother Years of Education	**−0.0179*****	**−0.0131****	**−0.0104***
	(0.00512)	(0.00514)	(0.00489)
Father Years of Education	**−0.0109***	−0.00520	0.00250
	(0.00451)	(0.00464)	(0.00449)
1.Migrant Mother	**−0.104***	**−0.0980***	**−0.127****
	(0.0449)	(0.0452)	(0.0445)
1.Migrant Father	−0.0634	−0.0630	−0.0968
	(0.0738)	(0.0744)	(0.0641)
1.Migrant Mother X Migrant Father	0.173	0.164	**0.223****
	(0.0921)	(0.0929)	(0.0835)
Age in 2015	0.00507***	0.00385***	0.00297**
	(0.00101)	(0.00102)	(0.00116)
Female	0.0194	0.00956	0.0431*
	(0.0186)	(0.0185)	(0.0183)
Respondent Years of Education		−0.0138***	−0.00571*
		(0.00255)	(0.00242)
Constant	0.633***	0.773***	−0.470***
	(0.0943)	(0.0963)	(0.127)
Country Dummies	X	X	X
Missing Parent Educ Dum	X	X	X
Health and Income Mediators			X
Observations	18,785	18,785	18,785
Adj R-Squared	.036	.042	.133

Robust standard errors in parentheses.
*p < 0.05; **p < 0.01; ***p < 0.001.

Table 10
OLS results with childhood books and rooms.

VARIABLES	Model (1)	Model (2)	Model (3)	Model (4)
	Log CRP	Log CRP	Log CRP	Log CRP
Mother Years Education	**−0.0151****	**−0.0112***	−0.00849	−0.00749
	(0.00544)	(0.00549)	(0.00551)	(0.00522)

(*continued on next page*)

Table 10 (*continued*)

VARIABLES	Model (1)	Model (2)	Model (3)	Model (4)
	Log CRP	Log CRP	Log CRP	Log CRP
Father Years Education	**−0.0122***	−0.00582	−0.00259	0.00271
	(0.00476)	(0.00499)	(0.00510)	(0.00485)
Age in 2015	0.00523***	0.00410***	0.00333**	0.00314**
	(0.00111)	(0.00113)	(0.00113)	(0.00125)
Female	0.0276	0.0293	0.0208	0.0528***
	(0.0194)	(0.0192)	(0.0191)	(0.0189)
Books in Childhood:				
2. Enough to fill one shelf (11–25 books)		−0.116***	−0.0983***	−0.0829***
		(0.0278)	(0.0278)	(0.0259)
3.Enough to fill one bookcase (26–100 books)		−0.106***	−0.0811**	−0.0466
		(0.0287)	(0.0286)	(0.0270)
4. Enough to fill two bookcases (101–200 books)		−0.0992*	−0.0660	−0.0337
		(0.0389)	(0.0393)	(0.0374)
5. Enough to fill two or more bookcases (200+ books)		−0.144***	−0.108*	−0.0695
		(0.0428)	(0.0430)	(0.0409)
# Rooms in Childhood		−0.0124*	−0.0117*	−0.00707
		(0.00528)	(0.00518)	(0.00490)
Resp. Years Education			−0.0109***	−0.00423
			(0.00259)	(0.00248)
				(0.0646)
Constant	0.591***	0.674***	0.780***	−0.421***
	(0.102)	(0.102)	(0.104)	(0.141)
Country Dummies	X	X	X	X
Missing Parent Educ Dum	X	X	X	X
Resp. Health Variables				X
Resp. SES Variables				X
Observations	16,931	16,931	16,931	16,931
Adj R-Squared	.036	.043	.046	.134

Robust standard errors in parentheses.
*p < 0.05; **p < 0.01; ***p < 0.001.

Table 11
Linear decomposition results – childhood books and rooms.

	Childhood SES Indicators Model (2) vs (1)		Respondent Education Model (3) vs. (1)		Respondent Adult SES and Health Model (4) vs. (1)	
	Mother's Education	Father's Education	Mother's Education	Father's Education	Mother's Education	Father's Education
Total Effect	−0.015 (−.026 -.004)**	−0.012 (−.021 to −.003)*	−0.015 (−.026 -.004)**	−0.012 (−.021 to −.003)*	−0.015 (−.026 -.004)**	−0.012 (−.021 to −.003)*
Direct Effect	−0.011 (−.022 to −.000)*	−0.006 (−.015 – .004)	−0.008 (−.019–−.002)	−.002 (−.012 – .007)	−.007 (−.017 - .002)	.003 (−.008 - .012)
Indirect Effect	**−0.004 (-.006 – -.002)*****	**−0.006 (-.009 – -.003)*****	−0.007 (−.010 to −.004)***	−.010 (−.013 to −.006)***	−0.008 (−.012 to −.003)***	−.015 (−.019 to −.010)***
Mediating Effects (%)	25.9%	52.1%	43.7%	78.7%	50.3%	122.2%

Note: 95% Confidence intervals in parentheses. *p < 0.05; **p < 0.01; ***p < 0.001.

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