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Reasearch Article

Unequal before death: The effect of paternal education on children's old-age mortality in the United States

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A growing body of research documents the relevance of parental education as a marker of family socio-economic status for children's later-life health outcomes. A strand of this literature evaluates how the early-life environment shapes mortality outcomes during infancy and childhood. However, the evidence on mortality during the life course and old age is limited. This paper contributes to the literature by analysing the association between paternal education and children's old-age mortality. We use data from Social Security Administration death records over the years 1988–2005 linked to the United States 1940 Census. Applying a family(cousin)- fixed-effects model to account for shared environment, childhood exposures, and common endowments that may confound the long-term links, we find that having a father with a college or high-school education, compared with elementary/no education, is associated with a 4.6- or 2.6-month-higher age at death, respectively, for the child, conditional on them surviving to age 47.

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Introduction

Health disparities in old age partially reflect socio-economic conditions in early life (Hayward and Gorman 2004; Engelman et al. 2010; Elo et al. 2014; Lazuka 2019). Parental education, as a major component and determinant of socio-economic conditions, may play an important part in the later-life health and mortality outcomes of their children. Several studies suggest that parental education and family socio-economic status (SES) shape early childhood mortality (Hatt and Waters 2006; Gakidou et al. 2010; Gage et al. 2013; Balaj et al. 2021). However, the evidence for mortality through the life course is less established, despite the potential importance and policy relevance of a long-term association. In a recent study, Huebener (2019) investigates the effects of parental education on children's life expectancy in Germany. The results from survival analysis show that higher parental education is correlated with higher survival rates for children's mortality, conditional on survival up to age 65. Also, the effects of father's education are smaller than those of mother's education and are insignificant in most cases. Lee and Ryff (2019) use Midlife in the United States data and explore how a range of early-life adversities affect later-life mortality. They find evidence that early-life SES is associated with adult mortality risk. In a similar study, Montez and Hayward (2011) use the Health and Retirement Study to explore the effects of early-life conditions on old-age mortality. They find that non-Hispanic white people and those whose fathers

This article has been corrected with minor changes. These changes do not impact the academic content of the article.

are less than high-school educated display higher risks of mortality.

The current study extends this line of research by establishing an association between paternal education and children's longevity in the United States (US). We construct a longitudinal sample based on the full-count 1940 Census linked with Social Security Administration (SSA) death records for the years 1988–2005. The results of family-fixed-effects models, which compare the longevity of cousins within a family tree, suggest that higher paternal education significantly increases children's age at death and that the cross-sectional ordinary least squares (OLS) estimates do not generate a noticeable bias in the results. The preferred specification, which includes a wide array of family controls and fixed effects, in addition to father's family fixed effects, implies that fathers with a college education have children with 4.6 months of additional lifespan compared with low-educated fathers. Similarly, the ages at death of children whose fathers are high-school or middle-school educated are 2.6 or 1.8 months higher, respectively, than for those with a father with elementary/no education.

In addition, we find substantial heterogeneity in the effects. The effects are more pronounced among Black people, males, those in families with lower SES, and those residing in the South census region. Moreover, we find complementarity between father's and mother's schooling: the effects of father's education are larger when the mother is more educated. Further analyses show that father's education affects the material resources of a family. Highly educated fathers are more likely to be in the labour force, have higher income, have more children, own a house, and work in occupations with higher income scores. Also, families engage in assortative matching in the marriage market and more highly educated fathers are also more likely to be matched with more highly educated mothers. While the data offer these channels as the mediating paths between father's education and children's longevity, we also discuss alternative pathways suggested by the literature.

This study contributes to the growing literature on education and mortality in three ways. First, we assess endogeneity issues by implementing a family-fixed-effects strategy, thus extending the literature. While some studies take advantage of compulsory schooling as the shock to parental education in order to explore children's life-cycle outcomes, hardly any studies apply a family-fixed-effects model to explore this long-term link specifically in the US context (see e.g. Currie and Moretti 2003; Oreopoulos et al. 2006; Lindeboom et al. 2009; Chou et al. 2010; Chalfin and Deza 2018, 2019; Hamad et al. 2018; Cui et al. 2019; Noghanibehambari et al. 2022). Second, while previous studies exploit data with small sample sizes and low power to explore parental education on children's health and longevity, the constructed data set in the current study offers unprecedented and unparalleled sample sizes to explore this long-term link using US data (Montez and Hayward 2011; Huebener 2019, 2020, 2021; Lee and Ryff 2019). Third, this paper contributes to the growing literature on non-monetary returns to education and intergenerational effects of human capital (Currie and Moretti 2003; Silles 2011, 2017; Suhonen and Karhunen 2019). This aspect of the current study has important policy implications. It adds to understanding of the usually unobserved and long-term benefits of intervention policies that aim to improve educational outcomes.

The rest of the paper is organized as follows. We first provide a brief review of the literature and then discuss the process of constructing the final sample and the sample selection criteria. Next we introduce the econometric framework and underlying assumptions in the models. The next two sections go over the results and suggest several mechanism channels. We finish with some concluding remarks.

Literature review

There are several ways through which paternal education may affect children's old-age mortality. We categorize these mediating channels into three general pathways: (1) improvements in children's own education; (2) improvements in available material resources during childhood; (3) the association between father's and mother's education through assortative matching. In this section, we discuss each channel briefly.

First, individuals with higher education also tend to have more highly educated parents. Several studies establish a causal link between parental schooling and children's educational attainments (Behrman and Rosenzweig 2002; Daouli et al. 2010; Pronzato 2012; Zeng and Xie 2014; Dickson et al. 2016; Zhou and Dasgupta 2017; Lundborg et al. 2018; Agüero and Ramachandran 2020). Education, in turn, may affect health and mortality in several ways; for example by providing better health-related knowledge, change in social peers, improved resources through changes in lifetime earnings, and safer occupations with better health insurance (Acemoglu and Angrist 2000; Card 2001;

Grimard and Parent 2007; Conti et al. 2010; Goldin and Katz 2010; Cutler et al. 2015; Fletcher 2015; Fletcher et al. 2021; Lleras-Muney 2022; Lleras-Muney et al. 2022; Noghanibehambari 2022a). Higher lifetime earnings can translate into a healthier environment and better health-related resources to improve health status and increase life expectancy (Lindahl 2005; Snyder and Evans 2006; Gonzalez and Quast 2015; Fitzpatrick and Moore 2018; Lefèbvre et al. 2019).

In addition, some studies also establish a link between education and health-related behaviour, such as drinking, smoking, and obesity-related habits (Fletcher and Frisvold 2009; Tenn et al. 2010; Fletcher and Frisvold 2011; Cawley et al. 2013; Cohen et al. 2013; Maralani 2013; Koning et al. 2015; Kim 2016; Barcellos et al. 2018). Drinking, smoking, and obesity are also linked to mortality outcomes (Pampel 2005; Leon et al. 2007; Cecchini et al. 2010; Ahima and Lazar 2013; Carter et al. 2015). Several studies explore the causal effect of education on health, longevity, and mortality regardless of the pathway. While some studies find a protective effect of education against mortality (Lleras-Muney 2005; van Kippersluis et al. 2011; Fischer et al. 2013; Gathmann et al. 2015; Buckles et al. 2016; Davies et al. 2016; Galama et al. 2018; Halpern-Manners et al. 2020), others reach a null result and fail to detect a significant impact (Mazumder 2008; Lindeboom et al. 2009; Cutler and Lleras-Muney 2012; Clark and Royer 2013; Leuven et al. 2016; Meghir et al. 2018; Barcellos et al. 2019; Albarrán et al. 2020). For instance, Halpern-Manners et al. (2020) use linked US 1940 Census and SSA death records and implement a twin fixed-effects strategy. They show that education significantly increases longevity; however, their results suggest that accounting for common endowments through a twin fixed-effects strategy causes the coefficients to drop slightly. They find that an additional year of schooling is associated with a 4.2-month-higher age at death. Lleras-Muney (2005) explores the effect of own education on mortality using changes in compulsory schooling laws and child labour laws during the years 1915–39 as the instrument for education. She constructs synthetic cohorts using US decennial censuses to compute 10-year mortality rates. She finds that an additional year of education reduces the 10-year mortality rate by approximately 3.6 percentage points. However, other studies that implement changes in compulsory schooling as the shock to education find different results (Mazumder 2008; Fletcher 2015). For instance, Black et al. (2015) refine Lleras-Muney's (2005) estimations by providing more precise measures of mortality using vital statistics data and find that including cohort fixed effects absorbs all the effects on mortality. Fletcher (2015) implements the same strategy on a relatively large sample of individual data and finds significant evidence for the impacts of own education on a wide range of health conditions and self-reported health outcomes. However, the effect of education on mortality is not precisely estimated, although the magnitude of the effect is large.

Second, parental education can change the available material resources that matter for prenatal or postnatal development, leaving an initial health endowment by improving birth outcomes and nurturing child growth during early life (Almond and Currie 2011; Almond et al. 2018). Education-induced increases in parental income can affect child health through increases in consumption of health-related inputs (e.g. better prenatal care during pregnancy, better health insurance, and better healthcare utilization during childhood) and/or other non-health-related consumption that affects child development (e.g. better food, cleaner neighbourhood of residence, and better health environment). These improvements influence health at birth and at postnatal ages, which may change the trajectory of life-cycle outcomes (Almond et al. 2011; Hoynes et al. 2011; Hoynes et al. 2015; Hoynes et al. 2016; Rosales-Rueda 2018; Noghanibehambari and Salari 2020). For example, Almond et al. (2011) show that the introduction of the Food Stamp Program (an anti-poverty programme that provided resources for the poor in the US) was associated with improved birth outcomes. Hoynes et al. (2016) complement this analysis, showing that those individuals who benefited from the programme during their childhood displayed improved health outcomes during adulthood.

Third, in an assortative matching marriage market, paternal education is also reflected in maternal education, and the latter, in turn, affects child health outcomes as a complement to the effects of paternal education (Currie and Moretti 2003; Chen and Li 2009; Ali and Elsayed 2018; Chang 2018; Keats 2018; Shen 2018; Noghanibehambari et al. 2022). Maternal education can also influence the initial health endowment of offspring through health behaviour channels such as smoking and drinking, both of which are documented to be associated with negative infant health outcomes (Yan 2014; Barreca and Page 2015). Several studies establish the long-term link between health at birth and in infancy and childhood on health and mortality at

older ages (Behrman and Rosenzweig 2004; Black et al. 2007; Royer 2009; Wherry et al. 2018; Maruyama and Heinesen 2020; Goodman-Bacon 2021).

Several studies examine the impacts of parental education on the life-cycle outcomes of their children. For instance, Carneiro et al. (2013) use matched data from female respondents in the National Longitudinal Survey of Youth (1979 cohort) to explore the effect of maternal education on children's outcomes. They find considerable returns to mother's education in terms of test scores and Behaviour Problems Index. Nevertheless, they do not find an effect on children's overweight measures, although the coefficients are negative. Chou et al. (2010) exploit the sharp changes in compulsory schooling laws in Taiwan accompanied by the construction of a series of new high schools to assess the effect of parental education on infant health and mortality. They find significant reductions in low-birthweight infants and infant mortality. The effects are quite similar for both maternal and paternal education. Of particular relevance to the current study, Huebener (2019) investigates the effects of parental education on children's life expectancy in Germany. The survival analysis results show that higher parental education is associated with lower mortality for children conditional on survival up to age 65. Also, the effects of father's education are smaller than those of mother's education and are insignificant in most cases. Lundborg et al. (2014) explore the education–health association using a compulsory schooling reform in Sweden and find that increasing mother's education has a positive effect on sons' height, health, physical capacity, and cognitive and non-cognitive abilities. The results of other similar studies are inconclusive (Caldwell and McDonald 1982; Thomas et al. 1990, 1991; Breierova and Duflo 2004; Alderman et al. 2006; Lindeboom et al. 2009; Gakidou et al. 2010; McCrary and Royer 2011). For instance, Lindeboom et al. (2009) show that the UK schooling reform, which raised the minimum school leaving age by one year in 1947, did not have a significant impact on the health of children of affected cohorts.

Data and sample construction

The primary source of data is the 'Numident' (Numerical Identification System) files of the SSA death records (1988–2005) linked to the full-count 1940 Census in the US. The Numident data are provided by the CenSoc project (Goldstein et al. 2021). The linkage technique employs the 'ABE fully automated approach', which is based on first name, last name, and age, to identify individuals (Abramitzky et al. 2012, 2014, 2019). We should note that CenSoc uses exact name to match and later evaluates the robustness of this method compared with three other methods (using raw names, New York State Identification and Intelligence System (NYSIIS) standardization, and the Jaro–Winkler distance method) to link individuals across data sets (Ferrie 1996; Abramitzky et al. 2012, 2019; Abramitzky et al. 2021; Breen and Osborne 2022).

Data from the 1940 Census and other historical full-count censuses between 1900 and 1930 are extracted from Ruggles et al. (2020). Since women often change their last name after marriage, we are unable to carry out the merging for mothers when we explore historical censuses to search for the family tree. Therefore, our primary variable of interest is father's education, and we impose sample restrictions based partly on father's characteristics.

We use the information on dates of birth and death to construct our measure of longevity: age at death. We restrict the sample to cohorts born 1923–40, as children usually leave the household by age 18 and would thus not be recorded in the parental household in the 1940 Census. We also limit the sample to those whose fathers are aged 25–55 in 1940. The Numident-census-linked data contain a wide array of parental information, including parental education, which can be used to estimate long-run effects. However, we are concerned that even after controlling for a full battery of characteristics and fixed effects, such correlations may fail to capture the full effects of family environment, neighbourhood influences, and local health environment, as well as children's initial abilities and genetic endowments. For instance, the local availability of schools and colleges may influence paternal education and also children's health. Moreover, these institutes are also located in places with different characteristics compared with places with fewer schooling options; these local factors may result in better health accumulation during childhood and also affect old-age longevity. In addition, more able fathers may acquire more schooling and also have more able children, who live longer due to factors that operate through genetic mechanisms.

To control for a set of these potential confounders, we rely on variation in educational attainment between fathers who are siblings in assessing the outcomes of their children (who are cousins). We argue that siblings are more likely to experience similar exposures to internal and external shocks during childhood than two unrelated individuals in pooled

samples. They are plausibly similarly exposed to local economic shocks, school availability, nurturing environments, parental culture regarding education, and genetic endowment compared with two unrelated individuals with similar characteristics. We should note that implementing family-fixed-effects models and exploiting within-sibling variation does not account for the full set of shared environments and genetic endowments. Moreover, unlike studies that implement a twin-fixed-effects strategy, this so-called cousin-fixed-effects method does not account for non-shared genetic traits. We also note that we cannot use twin- or sibling-fixed-effects models to analyse children's outcomes, as there is no variation in paternal education. A remaining strategy would be to use fathers who are identical twins but are discordant for educational attainment; in this case, the children (cousins) would share the same amount of genetic similarity as traditional biological siblings from the same father but would be exposed to larger unshared non-genetic variation than would biological siblings raised in the same household.

During the first decades of the twentieth century, there were many early-life adversities that could have affected accumulation of human capital and health endowment with long-lasting legacies for subsequent generations: these include the Spanish flu, the Great Depression, two world wars, and many social and political movements (Almond 2006; Myrskylä et al. 2013; Evans et al. 2016; Kose et al. 2021; Noghanibehambari and Engelman 2022; Schmitz and Duque 2022; Noghanibehambari and Fletcher 2023). The potential differential exposure to these adversities may confound cross-sibling comparisons. However, there are two benefits of such a method (see Boardman and Fletcher 2015). First, comparing two siblings still accounts for a portion of childhood environment and shared genetic traits and hence provides more reliable estimates than comparing two arbitrary individuals with similar characteristics. Second, in the Main results subsection, we show the surprising stability in coefficients across OLS and family-fixed-effects models. The fact that taking into account a portion of shared childhood exposures and common innate endowments does not change the effects suggests that these factors do not confound the estimates. Hence, it is likely that other unobservables do not confound the results either. Therefore, we expect within-sibling variation in education to provide a good framework for reducing the impact of environmental factors that may influence education and also have an impact on the health of the family's subsequent generations.

To detect cousins within our Numident-census-linked sample, we search for the father's family tree in the decennial full-count censuses between 1900 and 1930 and then identify cousins in the 1940 Census. To identify grandparents and build a family tree, we start with historical identification of the father and use historical crosswalk data files provided by the Census Linking Project (Abramitzky et al. 2020) to search for their family tree in historical full-count censuses between 1900 and 1930. In Appendix A (supplementary material), we replicate the results using IPUMS Multigenerational Longitudinal Project (MLP) data, which provide similar crosswalks across historical censuses. We find very similar effects when we use MLP as the linking source.

As Abramitzky et al. (2019) explain, various automated linking techniques give a less than 5 per cent false positive rate. To assure that our results are not driven by false positive rates (negative match wrongly categorized as a positive match), we consider a match between two historical identification values if for all techniques we have a positive match. We consider two persons to be siblings if they share at least one parent when they appear in historical censuses as children. As an example, assume that in 1940 there are two persons named A and B with their fathers' unique identifiers being FA and FB. From the 1930 Census, we deduce that FA's and FB's birth years are 1887 and 1891, respectively. The reason for using the 1930 Census is that the 1940 Census does not report birth year, whereas previous censuses do. Also, using father's age to deduce birth year is problematic due to potential measurement errors (e.g. age heaping). We start with the 1900 Census (the earliest year that FA and FB could appear in the census data) and search for their fathers' and mothers' identifiers. If both identifiers point to the same father or mother, we assume that FA and FB are siblings and hence A and B are cousins. In the Additional analyses subsection, we show that the main results are robust and quite similar if we focus on those with the same father *and* the same mother. Such selection reduces our current sample by only 4.65 per cent.

We are able to identify 132,810 fathers for whom parental information is available in previous censuses and whose siblings also have children in the 1940 pooled sample. Hereafter, we refer to these observations as the 'sibling-fathers sample'. This sample covers individuals in cohorts born between 1923 and 1940 and who die in the years 1988–2005. Therefore, age at death in our sample varies between 47 and 82. Figure 1 shows the geographic distribution of places in which fathers' families are

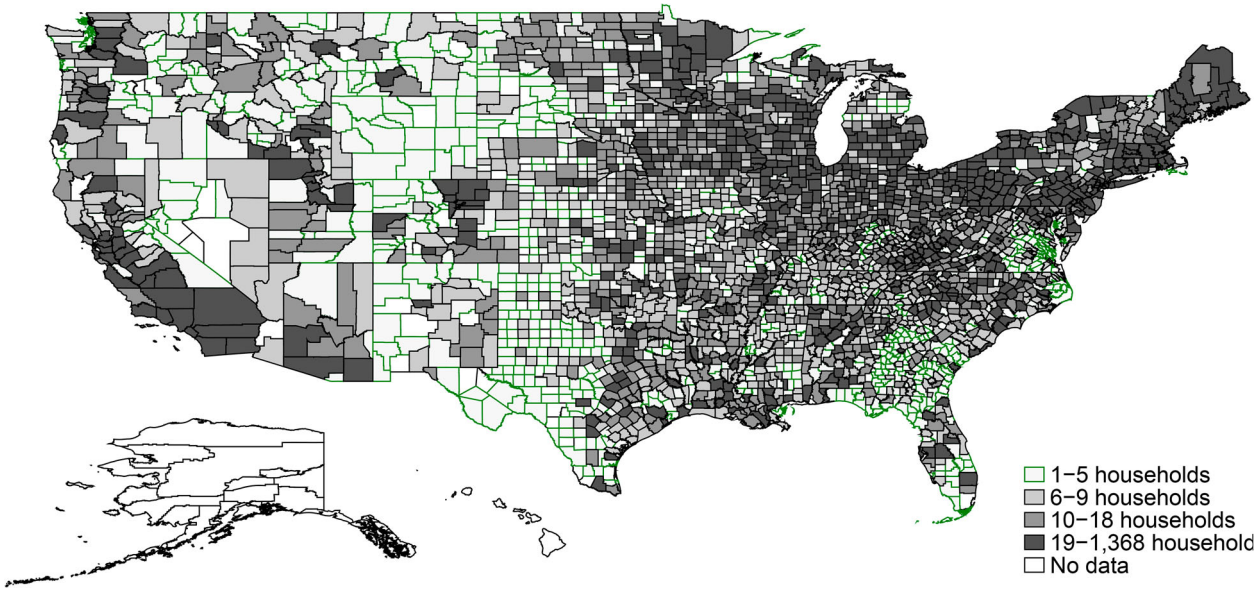

Figure 1 Distribution of identified households in the final sample (from historical censuses (1900–30) linked to the 1940 Census and Numident (1988–2005))
Source: Authors' analysis of Numident files linked to 1940 Census data.

identified. The Northeast region shows higher concentrations of identified family trees than the other regions (South, West, and Midwest).

Figure 2 provides a visual depiction of the process of final sample construction. The population of individuals born in 1923–40, who we observe in the 1940 Census and who satisfy our sample selection criteria, is roughly 32 million. About 7 per cent of these individuals die in 1988–2005 and are present in the Numident data. Further, we need to drop from the sample those fathers whose brothers cannot be traced in the historical censuses. We are able to locate about 36 per cent of fathers' households in historical censuses from 1900 to 1930. Finally, we need to drop those sibling fathers whose children are not present in Numident death records. These restrictions allow roughly 18 per cent of observations that remain from previous steps to be retained in the final sample. Overall, the final sample covers 4.2 per cent of the observations of the original sample.

Table 1 reports summary statistics for the original population, the Numident-census-linked sample, and the sibling-fathers sample, respectively. Our measure of mortality, age at death, is similar in the pooled and sibling-fathers samples. Compared with the original population, father's schooling is 0.15 years longer in the pooled sample and 0.07 years shorter in the sibling-fathers sample. For the sibling-fathers sample, we also report the percentage discordant (percentage of within-family fathers with different values) for the primary variable of interest (father's years of schooling) and the outcome (age at death). Well over 99 per cent of children in the sibling-fathers sample die at different ages. Among fathers within a family, roughly 75 per cent have different years of schooling (measured in whole years).

The final samples display slightly different demographic characteristics from the original population. To account for these differences, we treat the data as a longitudinal panel with consecutive attrition issues and reweight the data to adjust for these discrepancies. We follow Halpern-Manners et al. (2020) and weight the regressions by the inverse of the probability of linkage between the 1940 Census, Numident, and historical censuses using probit models conditioning on individual and family covariates. In Appendix B (supplementary material), we discuss the construction of weights.

Econometric method

We start our analysis with a standard OLS estimation that attempts to account for unobserved factors, applying a series of fixed effects and family covariates as follows:

$$D_{ibsj} = \alpha_0 + \alpha_1 S_{ij} + \alpha_2 X_i + \alpha_3 Z_j + \zeta_{bs} + \varepsilon_{ibsj}, \quad (1)$$

where the outcome is age at death for individual i who is from birth cohort b and birthplace (state of birth) s and whose family is indexed by j. The parameter S represents a series of dummies for

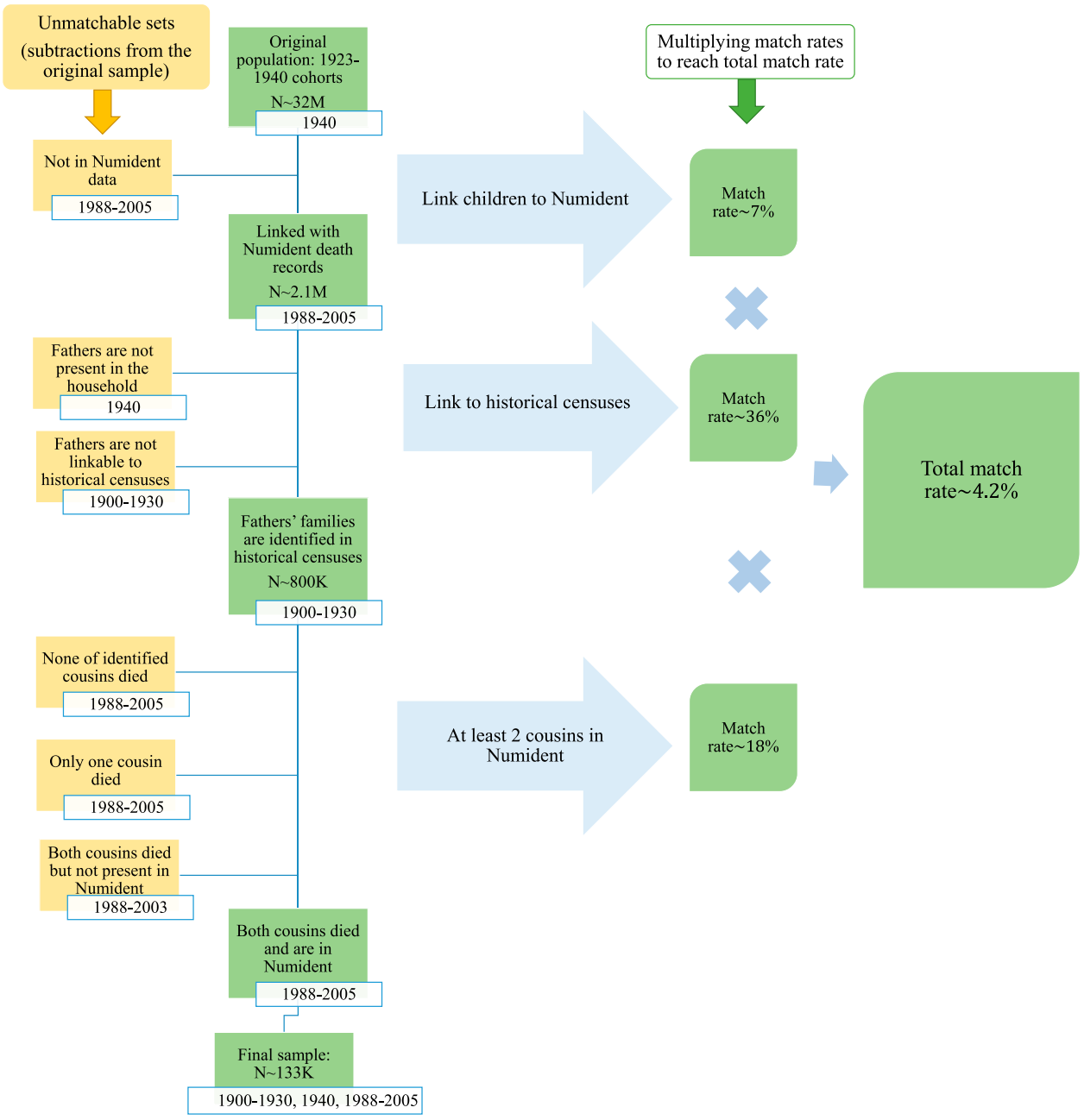

Figure 2 Steps in selection of the final sample from the original population
Source: As for Figure 1.

father's schooling. Specifically, we include three binary indicators to capture college education, high-school education, and middle-school education; the reference group is elementary/no education. College education is a dummy that equals one if the father has any college education and zero otherwise; high-school education is a dummy that equals one if the maximum years of father's schooling is 9–12 and zero otherwise; and middle-school education is a dummy that equals one if the maximum years of father's schooling is 6–8 and zero otherwise.

Birth-state-by-birth-year fixed effects are included in ζ. We use birth-state fixed effects as studies show that place of birth is more important for lifetime outcomes than place of residence (Xu, Engelman et al. 2020; Xu, Wu et al. 2020; Xu et al. 2021). However, in the robustness checks, we include a battery of fixed effects for current state and county of residence in 1940, and we find very similar coefficients.

The matrix X includes individual controls, such as sex, race, and origin. Family-level controls, represented in Z, include dummies for mother's education (with an indicator for missing values),

Table 1 Summary statistics for the original population, the Numident-census-linked sample, and the sibling-fathers sample

	Original population (1940 Census)		Pooled sample (linked with Numident death records)		Sibling-fathers sample (linked with grandparents and Numident)	
	Mean	*SD*	Mean	*SD*	Mean	*SD*
Age at death (months)	–	–	829.421	74.037	829.701	73.249
Within-family percentage discordant	–	–	–	–	0.998	0.046
Father's years of schooling	7.914	3.610	8.067	3.383	7.840	3.022
Within-family percentage discordant	–	–	–	–	0.750	0.433
Father's education						
None/elementary	0.168	0.374	0.137	0.344	0.124	0.329
Middle school	0.499	0.500	0.531	0.499	0.600	0.490
High school	0.250	0.433	0.254	0.435	0.221	0.415
College	0.082	0.274	0.078	0.267	0.055	0.229
Female	0.490	0.500	0.416	0.495	0.410	0.492
Race						
White	0.907	0.291	0.933	0.250	0.954	0.210
Black	0.089	0.284	0.064	0.245	0.044	0.205
Other	0.005	0.068	0.003	0.055	0.002	0.049
Origin						
Hispanic	0.022	0.145	0.013	0.111	0.008	0.089
Father's labour force status	0.965	0.184	0.966	0.181	0.968	0.176
Father's occupation						
Blue-collar	0.043	0.202	0.040	0.197	0.030	0.172
Farm	0.193	0.394	0.181	0.385	0.227	0.419
Father is married	0.983	0.130	0.983	0.129	0.985	0.123
Number of children in the household	3.569	2.069	3.658	2.062	3.914	2.098
Father homeowner	0.397	0.489	0.421	0.494	0.431	0.495
Mother's education						
Zero	0.110	0.313	0.086	0.280	0.071	0.257
Less than High School	0.469	0.499	0.493	0.500	0.538	0.499
High School	0.324	0.468	0.330	0.470	0.315	0.464
Some college	0.068	0.251	0.065	0.246	0.052	0.223
Missing	0.028	0.167	0.026	0.161	0.023	0.152
Observations	32,145,395		2,126,236		132,810	

Note: SD is the standard deviation.
Source: Authors' analysis of Numident files linked to 1940 Census data.

father's number of children, father's marital status, father's labour force status, father's homeownership status, and dummies for father's occupation (blue-collar or farming sector, with white-collar as reference group). We cluster standard errors at the family level. Later we show that the level of clustering does not change the standard errors, and clustering at other dimensions reveals the same set of results (Robustness checks subsection).

A primary concern with these regressions is their failure to account for unobservables either at the local-area level or at the father's family level. For instance, the availability of schools and colleges may influence father's education. Moreover, these institutes are also located in places with different characteristics compared with places with fewer schooling options; these local factors may result in better health accumulation during childhood and affect old-age longevity. In addition, more able fathers may acquire more schooling and also have more able children who live longer through channels that operate through inherent endowments. To account for these confounders, we refine equation (1) by including (father's) family fixed effects:.

$$D_{ibsj} = \alpha_0 + \alpha_1 S_{ij} + \alpha_2 X_i + \gamma_j + \zeta_{bs} + \varepsilon_{ibsj}, \qquad (2)$$

where γ represents (father's) family fixed effects for the sibling-fathers sample. All other parameters are similar to those in equation (1). We cluster the standard errors at the family level.

This strategy compares the mortality outcomes of cousins whose fathers share the same family and childhood environment, and, on average, 50 per cent of their genetic endowments (Hoekstra et al. 2008). However, in addition to the genetic

differences, two potential factors could still confound the estimations from equation (2), and we should be aware of these when interpreting the results. First, some studies in sociology, psychology, and psychopathology document differences among siblings in social and behavioural outcomes that can be partly attributable to non-shared environments and non-shared exposures (Dunn and Plomin 1991; Anderson et al. 1994; Conley et al. 2007; Jensen and McHale 2015). Second, the shared environment is not equally experienced by all children, and parents may engage in differential treatment of their children. This differential treatment could be related to child characteristics (e.g. sex, health) or parents' characteristics (e.g. education) and could reveal compensatory or reinforcing behaviour (Almond and Mazumder 2013; Frijters et al. 2013; Grätz and Torche 2016; Restrepo 2016; Fletcher et al. 2020). To the extent that non-shared experiences and differential treatments influence paternal education and may also appear in investments in their children's health, this generates a bias in equation (2). In the Selection on unobservables subsection, we show that these confounders would need to have a large degree of influence to show considerable effects on the coefficients.

Results

Main results

We start our analysis by discussing the results for specifications that exclude family fixed effects. These raw OLS results are reported in panel A1, Table 2. We add more covariates across columns, and the effects are quite robust across models. In the full specification in column (4), which adds a wide array of father's controls, maternal education, and fixed effects, we can see that children with college-educated and high-school-educated fathers live 4.4 and 2.8 months longer, respectively (compared with those whose fathers have elementary/no education). In panel A2, we add father's family fixed effects to all columns. The marginal effects are surprisingly stable and comparable to the OLS effects in panel A1. Another interesting aspect of both sets of results is the relatively large and very stable R-squared values. In Appendix C (supplementary material), we examine this feature further. We find that the highest contributor to the R-squared is family fixed effects and the second is birth-year fixed effects. The contributions of all other covariates and fixed effects are less than 1 per cent.

The evidence suggests that after controlling for fixed characteristics of families in which fathers are raised and taking into account (although only partially) genetic endowments across siblings, children whose fathers have a college, high-school, or middle-school education live 4.6, 2.6, or 1.8 months longer, respectively, than those with low-educated fathers. These effects are equivalent to 74, 42, and 29 per cent of the gap between females and males in the outcome after controlling for other factors (the coefficient for the 'female' dummy in the full specification of column (4) is 6.17).

In panel B, we replace the measure of father's education with a continuous measure of father's years of schooling. We observe a similarly robust and consistent pattern across specifications and when comparing OLS models with those that add family fixed effects (panels B1 and B2). For instance, a one-standard-deviation change in father's years of schooling (roughly three years) is associated with 1.1-month-higher longevity in both models (column (4)).

These results are in line with the findings of Montez and Hayward (2011), who examine the association between measures of childhood family SES and later-life mortality. They find that children whose fathers have low levels of education (less than eight years of schooling) are roughly 13–20 per cent more likely to die at each given age conditional on survival up to age 50. Huebener (2019) uses data from Germany to examine the association between parental education and children's life expectancy. He finds that, conditional on survival to age 65, those with more highly educated mothers live roughly two years longer than those with low-educated mothers. However, he does not find significant effects of father's education on children's longevity. Furthermore, we can also compare our estimated effects with studies that examine the association between own education and mortality. For instance, Halpern-Manners et al. (2020) implement a twin strategy to explore the effect of education on mortality among white males born between 1910 and 1920 and find that an additional year of schooling raises age at death by 4.2 months. One aspect of their findings is of most interest and is in line with our results: they show the effects across unpaired and paired samples of twins, siblings, and people living in the same neighbourhood. The marginal effects are very similar across different samples, either paired or unpaired, and whether including twin fixed effects or not; this is consistent with limited endogeneity issues in the long-term links between education and mortality.

Table 2 Effects of father's education on children's old-age mortality

Outcome: Age at death (months)	(1)	(2)	(3)	(4)
		Panel A. Education dummies		
		Panel A1. OLS models		
Father's education				
Middle school	1.83***	1.95***	1.92***	1.95***
	(0.57)	(0.57)	(0.57)	(0.58)
High school	2.61***	2.78***	2.76***	2.84***
	(0.64)	(0.64)	(0.65)	(0.68)
College	4.73***	4.77***	4.43***	4.35***
	(0.88)	(0.88)	(0.95)	(1.03)
Observations	132,810	132,810	132,810	132,810
R-squared	0.48	0.48	0.48	0.48
		Panel A2. Family-fixed-effects models		
Father's education				
Middle school	1.84**	1.87**	1.79**	1.82**
	(0.86)	(0.86)	(0.86)	(0.86)
High school	2.58**	2.64***	2.51**	2.58**
	(1.02)	(1.02)	(1.03)	(1.04)
College	4.54***	4.75***	4.46***	4.59***
	(1.43)	(1.43)	(1.50)	(1.55)
Observations	132,810	132,810	132,810	132,810
R-squared	0.71	0.71	0.71	0.71
		Panel B. Years of schooling		
		Panel B1. OLS models		
Father's years of schooling	0.35***	0.36***	0.35***	0.36***
	(0.06)	(0.06)	(0.07)	(0.08)
Observations	132,810	132,810	132,810	132,810
R-squared	0.48	0.48	0.48	0.48
		Panel B2. Family-fixed-effects models		
Father's years of schooling	0.37***	0.37***	0.35***	0.37***
	(0.10)	(0.10)	(0.11)	(0.11)
Observations	132,810	132,810	132,810	132,810
R-squared	0.71	0.71	0.71	0.71
Individual controls	✓	✓	✓	✓
Birth-state FE	✓	✓	✓	✓
Birth-year FE	✓	✓	✓	✓
Birth-state-by-birth-year FE		✓	✓	✓
Father's controls			✓	✓
Mother's controls				✓

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

Notes: Standard errors, clustered at the family level, are in parentheses. The regressions are weighted using the inverse of the probability of linkage between the 1940 Census and Numident using probit models conditioning on covariates. Individual controls include a dummy for sex. Father's controls include father's occupation dummies, father's marital status dummies, father's number of children in the household in 1940, and father's age. Mother's controls include dummies for educational attainment. FE refers to fixed effects.

Source: As for Table 1.

Our estimated effects are also comparable to those in the study by Fletcher and Noghanibehambari (2021), who explore the impact of county-level college opening during adolescent years on old-age longevity. They find that a four-year college (offering bachelor's degrees and above) opening in the county of residence when individuals are 17 years old is associated with 0.13-months-higher longevity. Their treatment-on-treated calculation suggests that having any college education raises age at death by about 12 months. The estimated effects in Table 2 suggest that having a father with any college education raises the child's age at death by 4.6 months, about one-third of the effect of own college education as reported by Fletcher and Noghanibehambari (2021).

Cohort selection and truncation

Due to left and right truncation of the data, average age at death differs across cohorts. For instance, the average age at death of cohorts born in 1923–30

varies between 57 and 82 years and that of the 1931–40 cohorts varies between 47 and 74 years. Therefore, a portion of the variation in the final sample comes from comparing individuals in earlier cohorts (who die at relatively older ages) with those in later cohorts (who die at younger ages due to truncation). Since earlier cohorts contain more individuals with lower paternal education, part of the results could reflect this selection due to the truncated nature of the final sample. To test for this concern empirically, we attempt to compare cohorts that die at similar ages. In so doing, we extract two subsamples from the final sample: a subsample that includes those in the 1923–30 cohorts who die at ages 65–75 (death ages that overlap between 1923–30 and 1931–40 cohorts due to truncation) and a subsample of those in the 1931–40 cohorts who die at ages 57–65 (thus excluding the common death ages across earlier and later cohorts). We replicate the main results of family-fixed-effects models for these two groups in Table 3. The full models in column (4) across panels suggest very similar effects. For instance, the effects of father's years of schooling are 0.38 and 0.34 for earlier and later cohorts, respectively (panels B1 and B2). Moreover, these estimates are fairly similar to our main results, which further supports the conclusion that truncation and selection issues are not the driver of the main results.

Robustness checks

In Table 4, we evaluate the robustness of the results of the family-fixed-effects models to alternative specifications, sample selections, and functional forms. Column (1) reports the fully parametrized family-fixed-effects results of column (4) of panel A2 of Table 2 as the benchmark marginal effects. Column (2) shows the results for an unweighted regression. The magnitudes and standard errors remain quite similar to the benchmark estimates.

One concern is the dominance of outliers in influencing the results. In column (3) we use the sample from column (1) and drop those families in which the standard deviation of schooling across sibling fathers is more than 4.0 (dropping 7 per cent of observations). The resulting coefficients are slightly larger than the benchmark estimate for the college education dummy and smaller than the benchmark effects for high-school and middle-school education dummies. Further, we use the sample from column (1) and drop observations that are greater than or less than two standard deviations from the mean of father's schooling and age at death. The results, reported in columns (4) and (5), are comparable to the benchmark results.

The main analysis sample is based on individuals aged 18 at most as the threshold for being observed within a household so as to locate their father's characteristics. In column (6) we observe a slightly smaller coefficient for middle-school and high-school education dummies when we restrict the sample to those aged 12 at most. However, the effect of college education is larger and remains statistically significant.

Next, we examine the sensitivity of standard errors to alternative clustering levels. In columns (7) and (8), we replicate the regression in column (1) but cluster standard errors at the birth-state and the 1940-county-of-residence levels. In column (9), we use two-way clustering at the county-of-residence and birth-state levels. The statistical significance of the results does not appear to be sensitive to a specific clustering level.

We continue by exploring the robustness of the results to additional controls and alternative specifications. The marginal effects are practically unchanged when we add father's occupational income score as an additional covariate as well as fixed effects for father's birth state and father's birth year (column (10)). Furthermore, the effects are comparable to the benchmark estimate when we add a county-of-residence fixed effect (column (11)), a county-of-residence-by-birth-cohort fixed effect (column (12)), and birth-state-by-race and birth-year-by-race fixed effects (column (13)).

As a final check, we explore the functional form sensitivity of the results. We replace the outcome variable with the logarithm of age at death (column (14)). Although it is not intuitive to interpret the semi-elasticity coefficients, they are statistically significant, suggesting that the linear measurement of variables is not a problem in our analyses.

In Table 5, we replicate these results without family fixed effects. The effects across columns are very similar to the benchmark estimates in column (1) as well as to the results of Table 4.

Subsamples

The results reveal substantial heterogeneity across subpopulations. We use father's years of schooling as the main independent variable in this section to ease the two-by-two comparisons. In Figure 3 we

Table 3 Effects of father's education on children's old-age mortality: restricting the sample to specific cohorts and ages at death

Outcome: Age at death (months)	Fixed-effects models			
	(1)	(2)	(3)	(4)
		Panel A. Education dummies		
		Panel A1. 1923–30 cohorts, age at death 65–75		
Father's education				
Middle school	1.76	1.29	1.27	1.33
	(1.31)	(1.31)	(1.31)	(1.31)
High school	2.25	1.78	1.77	2.08
	(1.53)	(1.53)	(1.54)	(1.57)
College	6.56***	5.74***	4.68**	5.05**
	(2.15)	(2.16)	(2.24)	(2.32)
Observations	30,926	30,916	30,916	30,916
R-squared	0.48	0.49	0.49	0.49
Mean DV	837.8	837.8	837.8	837.8
		Panel A2. 1931–40 cohorts, age at death 57–65		
Father's education:				
Middle school	1.56	1.24	1.36	1.38
	(1.80)	(1.81)	(1.81)	(1.81)
High school	1.66	1.21	1.51	1.57
	(2.11)	(2.12)	(2.14)	(2.17)
College	3.90	4.04	4.13	4.15
	(3.11)	(3.15)	(3.30)	(3.41)
Observations	30,128	30,124	30,124	30,124
R-squared	0.62	0.63	0.63	0.63
Mean DV	961.5	961.5	961.5	961.5
		Panel B. Years of schooling		
		Panel B1. 1923–30 cohorts, age at death 65–75		
Father's years of schooling	0.46***	0.40**	0.34**	0.38**
	(0.16)	(0.16)	(0.16)	(0.17)
Observations	30,926	30,916	30,916	30,916
R-squared	0.48	0.49	0.49	0.49
Mean DV	837.8	837.8	837.8	837.8
		Panel B2. 1931–40 cohorts, age at death 57–65		
Father's years of schooling	0.34	0.30	0.33	0.34
	(0.22)	(0.22)	(0.23)	(0.24)
Observations	30,128	30,124	30,124	30,124
R-squared	0.62	0.63	0.63	0.63
Mean DV	961.5	961.5	961.5	961.5
Family FE	✓	✓	✓	✓
Individual controls	✓	✓	✓	✓
Birth-state FE	✓	✓	✓	✓
Birth-year FE	✓	✓	✓	✓
Birth-state-by-birth-year FE		✓	✓	✓
Father's controls			✓	✓
Mother's controls				✓

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

Notes: Standard errors, clustered at the family level, are in parentheses. The regressions are weighted using the inverse of the probability of linkage between the 1940 Census and Numident using probit models conditioning on covariates. Individual controls include a dummy for sex. Father's controls include father's occupation dummies, father's marital status dummies, father's number of children in the household in 1940, and father's age. Mother's controls include dummies for educational attainment. FE refers to fixed effects.

Source: As for Table 1.

show the effects across a variety of subsamples, from implementing family-fixed-effects and OLS strategies, respectively. The figure shows the estimated coefficients and their 90 per cent confidence intervals (horizontal axis) for each subsample (vertical axis) and for each model (dashed line for OLS and solid line for family-fixed-effects). In these regressions, we include the full specification as per

Table 4 Effects of father's education on children's old-age mortality: robustness checks of family-fixed-effects strategy

	Column (4), panel A2, Table 2 (1)	Unweighted regression (2)	Drop if within-family SD in schooling>4 (3)	Drop if schooling > Mean + 2*SD* | < Mean − 2*SD* (4)	Drop if death age > Mean + 2*SD* | < Mean − 2*SD* (5)	Drop if age in 1940 >12 (6)	Clustering SE at birth state (7)
Father's education							
Middle	1.82**	1.97**	1.21	1.41*	1.46	1.08	1.82
school	(0.86)	(0.82)	(0.96)	(0.81)	(0.94)	(1.21)	(1.09)
High	2.58**	2.78***	1.86	2.18**	2.23**	2.22	2.58*
school	(1.04)	(1.00)	(1.19)	(0.99)	(1.12)	(1.46)	(1.34)
College	4.59***	4.62***	5.88***	4.88***	5.66**	5.11**	4.59**
	(1.55)	(1.49)	(2.00)	(1.46)	(2.67)	(2.21)	(1.77)
Observations	132,810	132,810	123,676	125,387	119,608	67,729	132,810
R-squared	0.71	0.66	0.71	0.67	0.71	0.67	0.71

	Clustering at county of residence (8)	Two-way clustering at county of residence and birth state (9)	Adding father's birth-year FE, birth-state FE, and occupational income score (10)	Adding county-of-residence FE (11)	Adding county-of-residence-by-cohort FE (12)	Adding birth-state-by-race FE and birth-year-by-race FE (13)	Log of outcome (14)
Father's education							
Middle	1.82*	1.82**	1.86**	1.61*	1.80	1.58*	0.00**
school	(0.97)	(0.77)	(0.89)	(0.87)	(1.16)	(0.87)	(0.00)
High	2.58**	2.58***	2.68**	2.19**	3.02**	2.28**	0.00**
school	(1.16)	(0.96)	(1.08)	(1.06)	(1.38)	(1.05)	(0.00)
College	4.59***	4.59***	4.44***	4.23***	6.24***	4.24***	0.01***
	(1.75)	(1.48)	(1.59)	(1.56)	(1.98)	(1.55)	(0.00)
Observations	132,810	132,810	128,257	132,723	110,265	132,779	132,810
R-squared	0.71	0.71	0.71	0.72	0.80	0.71	0.71

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

Note: Standard errors (SE), clustered at the father's family level (except for columns (7)–(9)), are in parentheses. The regressions are weighted using the inverse of the probability of linkage between the 1940 Census, historical censuses, and Numident using probit models conditioning on individuals' sex, race, and origin, father's occupation dummies, mother's education dummies, father's marital status dummies, father's number of children in the household at 1940, father's age, and individual birth-state-by-birth-year fixed effects (FE). All regressions include individual race, sex, and origin dummies, fixed effects for birth state by birth year, and controls for family covariates including mother's education, father's labour force status, father's marital status, father's occupation type (blue-collar, farmer), father's total number of children, and a dummy for father owning the dwelling. SD refers to the standard deviation.

Source: As for Table 1.

Table 5 Effects of father's education on children's old-age mortality: robustness checks of OLS regressions

	Column (4), panel A1, Table 2 (1)	Unweighted regression (2)	Drop if within-family SD in schooling>4 (3)	Drop if schooling > Mean + 2 × *SD* | < Mean − 2 × *SD* (4)	Drop if death age > Mean + 2 × *SD* | < Mean − 2 × *SD* (5)	Drop if age in 1940>12 (6)	Clustering SE at birth state (7)
Father's education							
Middle	1.95***	2.23***	2.12***	1.86***	1.84***	1.94***	1.95***
school	(0.58)	(0.51)	(0.61)	(0.51)	(0.62)	(0.70)	(0.54)
High	2.84***	3.30***	2.81***	2.90***	2.75***	2.65***	2.84***
school	(0.68)	(0.61)	(0.72)	(0.60)	(0.72)	(0.82)	(0.63)
College	4.35***	4.92***	5.27***	4.53***	4.30**	4.47***	4.35***
	(1.03)	(0.93)	(1.15)	(0.91)	(1.79)	(1.24)	(0.90)
Observations	132,810	132,810	123,678	127,970	123,906	85,687	132,810
R-squared	0.48	0.43	0.48	0.44	0.48	0.39	0.48

	Clustering at county of residence (8)	Two-way clustering at county-of-residence and birth-state (9)	Adding father's birth-year FE, birth-state FE, and occupational income score (10)	Adding county-of-residence FE (11)	Adding county-of-residence-by-cohort FE (12)	Adding birth-state-by-race FE and birth-year-by-race FE (13)	Log of outcome (14)
Father's education							
Middle	1.95***	1.95***	2.17***	1.73***	2.32***	1.91***	0.00***
school	(0.58)	(0.46)	(0.59)	(0.58)	(0.68)	(0.58)	(0.00)
High	2.84***	2.84***	3.22***	2.67***	3.20***	2.84***	0.00***
school	(0.69)	(0.58)	(0.70)	(0.68)	(0.80)	(0.68)	(0.00)
College	4.35***	4.35***	4.73***	4.30***	4.56***	4.29***	0.01***
	(1.05)	(0.91)	(1.05)	(1.04)	(1.20)	(1.02)	(0.00)
Observations	132,810	132,810	129,900	132,762	119,607	132,793	132,810
R-squared	0.48	0.48	0.49	0.50	0.58	0.49	0.48

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

Notes: Standard errors (SE), clustered at the father's family level (except for columns (7)–(9)), are in parentheses. The regressions are weighted using the inverse of the probability of linkage between the 1940 Census, historical censuses, and Numident using probit models conditioning on individuals' sex, race, and origin, father's occupation dummies, mother's education dummies, father's marital status dummies, father's number of children in the household at 1940, father's age, and individual birth-state-by-birth-year fixed effects (FE). All regressions include individual race, sex, and origin dummies, fixed effects for birth state by birth year, and controls for family covariates including mother's education, father's labour force status, father's marital status, father's occupation type (blue-collar, farmer), father's total number of children, and a dummy for father owning the dwelling. SD refers to the standard deviation.

Source: As for Table 1.

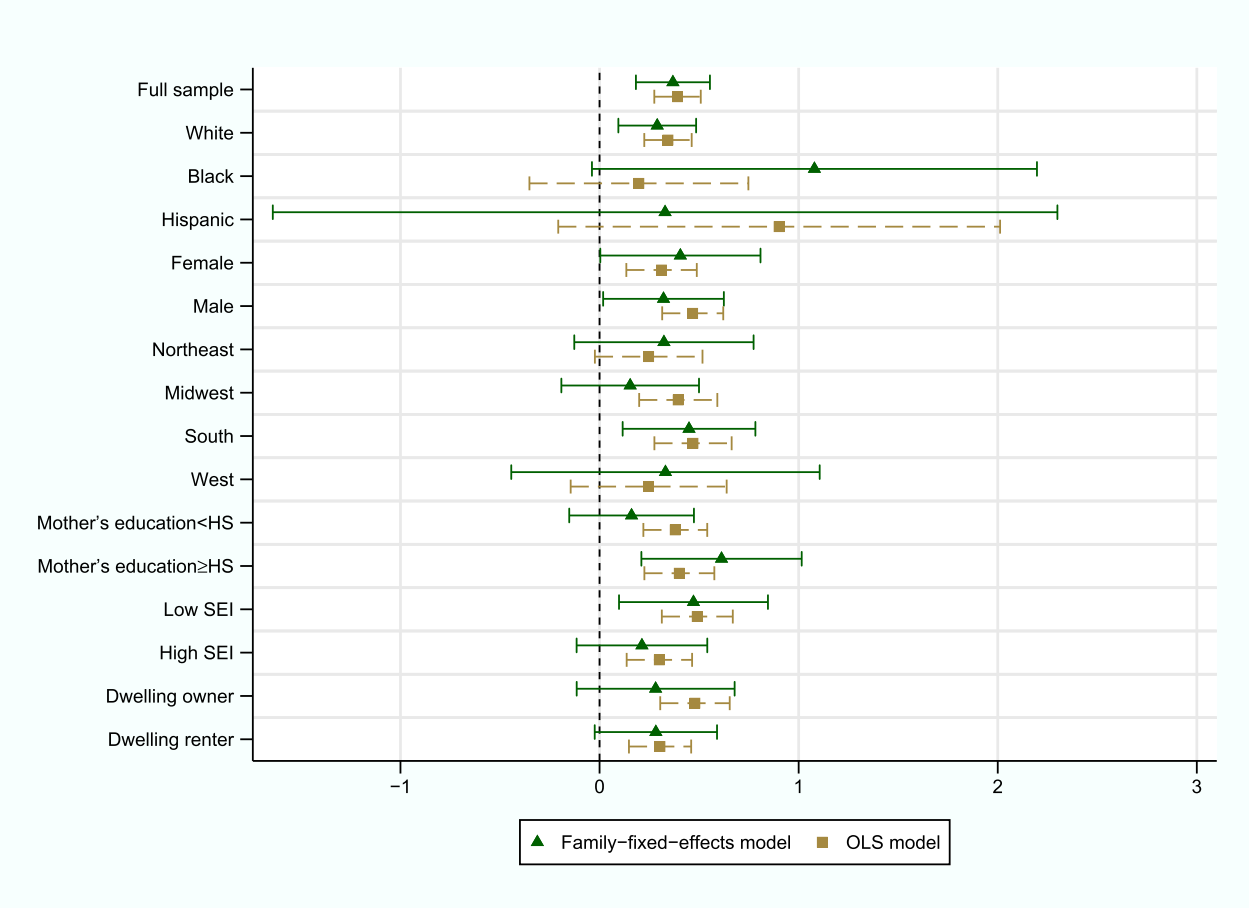

Figure 3 Heterogeneity of the main results across subsamples implementing father's family-fixed-effects strategy and OLS strategy

Notes: The 90 per cent confidence intervals are based on standard errors that are clustered at the father's family level. The regressions are weighted using the inverse of the probability of linkage between the 1940 Census, historical censuses, and Numident using probit models conditioning on covariates. All regressions include individual race, sex, and origin dummies, fixed effects for birth state by birth year, and controls for family covariates including mother's education, father's labour force status, father's marital status, father's occupation type (blue-collar, farmer), father's total number of children, and a dummy for father's being ownership of the dwelling.

Source: As for Figure 1.

column (4) of Table 2, including birth-state-by-birth-year fixed effects and a full set of family controls. For the family-fixed-effects models, we also add family fixed effects in addition to all other fixed effects and covariates included in the OLS regressions.

To provide a benchmark for comparison, we show the marginal effect (and its 90 per cent confidence interval) of paternal education on age at death for the full sample in the first line. The following lines report the results for subsamples based on race (white, Black), origin (Hispanic), sex (males, females), census division region of residence (Northeast, Midwest, South, and West), mother's education (less than high school, at least high school), socioeconomic index (SEI: low, high), and ownership of dwelling (owner, renter). While we show the OLS results for comparison purposes, our primary reference analysis is the family-fixed-effects model.

We observe larger effects for Black and Hispanic people compared with white people. However, in both cases, the effects are statistically insignificant, which limits additional comments. This is due mainly to the small numbers of observations in these groups reducing statistical power. For instance, there are only 808 Hispanic and 4,419 Black people in our sample. The main reason for these small sample sizes is the difficulty in linking these subpopulations across several data sources. For instance, the probability of merging between Numident and the 1940 Census is lower for Black individuals (see Table 1). Nonetheless, there is evidence that linked observations of Black individuals

are representative of the Black population nationally (Breen and Osborne 2022). However, the difference between the share of Black (and Hispanic) individuals in our final sample and the original population is much larger than the difference between the share of Black (and Hispanic) individuals in Numident-census-linked data and the original population. Therefore, we should exercise caution in interpreting the heterogeneity analyses by race, as there could be selection based on unobservables that make the final Black and Hispanic samples less representative of their corresponding populations. One concern that may arise is that the estimates are biased due to incorrect links and that the incorrect links might be more prevalent in harder-to-link subpopulations such as Black and Hispanic groups. We should note that the Numident–census linking is based primarily on name commonalities, although some harder-to-link subpopulations, such as Black and immigrant groups, are more likely to be enumerated with error, hence lower linking rates and at the same time differential education–longevity associations. We discuss the endogenous merging concern in Appendix D (supplementary material). Specifically, we explore whether more educated fathers are more or less likely to be linked from the original 1940 Census population to the Numident data. We find very small education-linking coefficients that, even in the worst scenario, will induce an ignorable bias into our estimations. Moreover, we observe a very similar coefficient when we look at whites, a subpopulation with more accurate links, suggesting that the effects are unlikely to be confounded by the linking rules of harder-to-link subpopulations. Furthermore, incorrect links will lead to unrelated people being assigned into a family unit. Therefore, we expect to observe underestimated coefficients and attenuated effects, as the effects of education on longevity will be smaller in comparisons of non–family members.

Studies suggest that the health–education gradient, more specifically the mortality–education gradient, is a function of childhood and adulthood local-area characteristics, and hence the gradients vary by geographic regions (Sheehan et al. 2018; Kemp and Montez 2020). Therefore, we also expect to observe heterogeneous effects of paternal education on child mortality across different regions. In the subsample analyses based on different census regions, we observe larger impacts among people residing in the South compared with other regions (Figure 3).

The evidence also suggests complementarity between mother's and father's educational attainment. The effect of father's education is considerably smaller (and statistically insignificant) when mother's education is low (0.16 vs 0.37) and slightly larger (and statistically significant) when the mother is educated at least to high-school level (0.61 vs 0.37).

Finally, there is evidence that the education–health gradient depends on the socio-demographic status of families (Barrow and Rouse 2005; Kimbro et al. 2008; Seeman et al. 2008). To examine this source of heterogeneity, we replicate the results across families who are above and below median SEI. The results in Figure 3 suggest larger effects among families who are below the median socioeconomic score. The association is statistically insignificant for the above-median group. We also examine heterogeneity by ownership of dwelling, but we do not find differences in the effects across owners vs renters.

Additional analyses

For the main results, we consider two fathers to be siblings if, in historical censuses, they share the same family and at least one of their parents is the same. We now impose a stricter condition for two persons to be siblings: both parents should be alive (and living within the same family) in historical censuses to be identified. Therefore, we assume two fathers are siblings if they have the same parents and both of their parents are present in the household. The resulting sample size is only 4.65 per cent smaller than the final sample for the main results. We replicate the main results for this analytic sample and report them in Table 6. The OLS results are very similar to the main results. However, the family-fixed-effects estimates are slightly larger than those reported in Table 2.

Selection on unobservables

Equation (2) differences out all shared genetic characteristics, shared family features, and shared childhood environment exposures across siblings in the father's family. However, it does not account for non-shared environment, non-shared genetic characteristics, or any discriminatory behaviour of parents. The fact that the OLS estimates are very close to the family-fixed-effects estimates suggests that fathers' childhood experiences are not confounding the estimates. However, we may be concerned that while unobservable characteristics of

Table 6 Effects of father's education and children's old-age mortality: fathers with *both* parents present in historical censuses

Outcome: Age at death (months)	OLS			Family-fixed-effects strategy		
	(1)	(2)	(3)	(4)	(5)	(6)
Father's education						
Middle school	2.01***	1.96***	1.99***	2.32***	2.24**	2.26**
	(0.59)	(0.59)	(0.59)	(0.89)	(0.89)	(0.89)
High school	2.97***	2.94***	3.00***	3.18***	3.06***	3.15***
	(0.66)	(0.67)	(0.70)	(1.05)	(1.06)	(1.07)
College	4.74***	4.44***	4.34***	5.17***	5.12***	5.22***
	(0.90)	(0.98)	(1.06)	(1.47)	(1.53)	(1.58)
Observations	126,632	126,632	126,632	126,632	126,632	126,632
R-squared	0.49	0.49	0.49	0.71	0.71	0.71
Father's family FE				✓	✓	✓
Birth-state and birth-year FE	✓	✓	✓	✓	✓	✓
Individual controls	✓	✓	✓	✓	✓	✓
Birth-state-by-birth-year FE		✓	✓		✓	✓
Father's characteristics			✓			✓
Mother's education control			✓			✓

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

Notes: Standard errors, clustered at the family level, are in parentheses. The regressions are weighted using the inverse of the probability of linkage between the 1940 Census, historical censuses, and Numident using probit models conditioning on individual sex, race, and origin. Father's controls include father's labour force status, father's marital status, father's occupation type (blue-collar, farmer), father's total number of children, and a dummy for father owning the dwelling. Individual controls include race, sex, and origin dummies. FE refers to fixed effects. Columns (1)–(3) include the same set of covariates as columns (1), (2) and (4) in Table 2.

Source: As for Table 1.

families are not introducing bias in our estimates, their differential impacts on members of a family could be confounding them. To gauge the degree to which these unobservables could be biasing the estimates, we apply a series of artificial unobservable shocks with a pre-specified relationship with the outcome and explanatory variables and calculate the marginal effects in each scenario.

To begin, we regress the outcome (age at death) on a series of observables, namely mother's schooling, father's labour force status, father's income, family's SEI, father's occupational income score, father's marital status, father's number of children, and father's ownership of dwelling. We use the predicted value of this regression as a combination of observables. The correlation of this variable with father's education is 0.03 and with the outcome is 0.10. We posit that the differential effect of unobservable characteristics could be as strong as the effect of a combination of all observables, although only in extreme cases. Therefore, we generate an artificial random variable (z) that is correlated with a value between −0.1 and 0.1 with both the outcome and explanatory variables. We then implement a full specification of the family-fixed-effects model (column (4), panel A2, Table 2). To reach more precise correlations, we implement a semi–Monte Carlo simulation, running these regressions 1,000 times for each combination of correlations and computing the average of estimated coefficients. Here, in order to ease interpretation of the findings and construct a correlation matrix, we use years of schooling instead of the three education dummies. The calculated point estimates are reported in Figure 4.

Each point estimate in this figure is associated with a regression that adds an artificial confounder (z) with a pre-defined correlation with father's years of schooling (corr(z,x) as shown on the x-axis) and a pre-defined correlation with the outcome (corr(z,y)). For illustration purposes, we group and connect all point estimates with a specific corr(z,y) and reveal their specified correlation in the figure's legend. As we would expect, when the correlation of artificial variable (z) with father's education (x) is zero, there is no bias in the coefficients and they converge to the benchmark effect of 0.37. Also, when z is not correlated with the outcome (y), it should not change the marginal effects, as shown by the green-triangle dotted line in Figure 4. The artificial bias generated by z changes the marginal effects, but they vary between 0.22 and 0.48 (still far from zero) and are statistically significant at conventional levels in all cases. There are two extreme cases where the coefficients drop below 0.3. The first is where $\text{corr}(z, y) = 0.1$ and

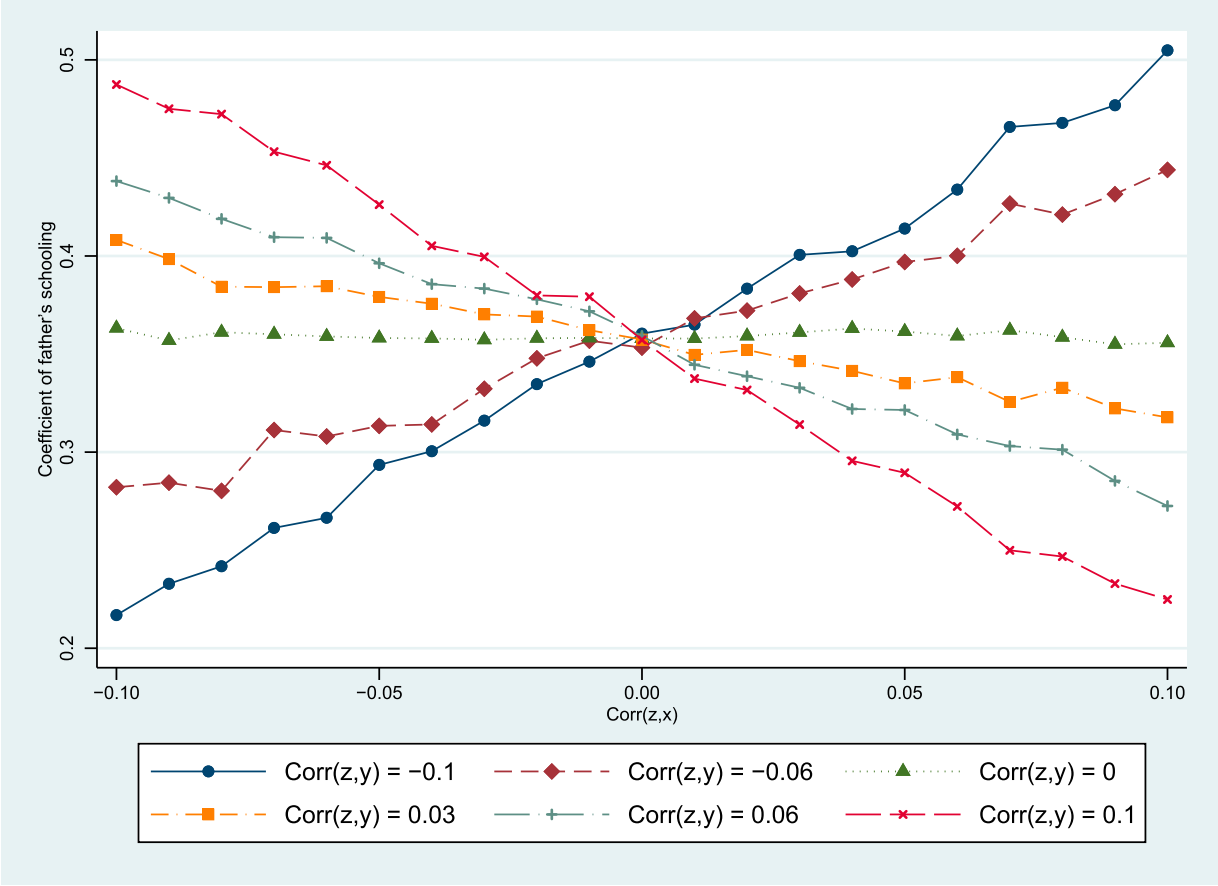

Figure 4 Monte Carlo simulation results from adding an additional regressor (z) with a pre-specified correlation with father's years of schooling (x) and child's age at death (y) in a family-fixed-effects strategy
Notes: The regressions are weighted using the inverse of the probability of linkage between the 1940 Census, historical censuses, and Numident using probit models conditioning on individuals' sex, race, and origin, father's occupation dummies, mother's education dummies, father's marital status dummies, father's number of children in the household at 1940, father's age, and individual's birth-state-by-birth-year fixed effects. All regressions include sex, race, and origin dummies, fixed effects for birth year by birth state, and a series of family controls including mother's years of schooling, father's labour force status, father's marital status, father's occupation type (blue-collar, farmer), father's total number of children, and a dummy for father's ownership of the dwelling.
Source: As for Figure 1.

corr$(z, x) = -0.1$, which is an unobservable that inhibits father's education but appears positive and strong for children's old-age health, a hard-to-find unobservable. The second is where both y and x show a relatively strong and positive correlation (0.1) with the artificial unobservable z. For instance, if there is a personality trait that leads to higher education in a father and not his brother, that personality trait is as important for his child's longevity as all other socio-economic and demographic characteristics of his family, including the child's mother's education, a genetic trait, or differential treatment by parents that outweighs all other observables. In this case, we expect the real marginal effects to decrease to 0.2, and in more extreme cases to zero.

Potential mechanisms

In this section, we explore potential mechanism channels between father's education and old-age longevity. An important channel is children's education, through the intergenerational transmission process. Since we do not have data on the completed education of children (they are still too young to have completed their education in 1940), we turn to alternative measures based on variables available in the 1940 Census, where the data report the highest grade attained. We compute the median grade level for each age. To avoid measurement errors in reported age, we calculate age based on exact date of birth available in

Table 7 Effects of father's education on families' socio-economic characteristics: OLS and family-fixed-effects strategies

	Child's old-for-grade status (1)	Child's school attendance status (2)	Mother's years of schooling (3)	Employed more than 48 weeks last year (4)	Father in the labour force (5)	Log father's income (6)	Father's Socio-Economic Index (7)	Father's occupational income score (8)	Father's number of children (9)	Father owns house (10)
Panel A. OLS models										
Father's education										
Middle	−0.13***	0.07***	2.22***	0.08***	0.02***	0.26***	4.02***	2.05***	−0.61***	0.05***
school	(0.00)	(0.00)	(0.03)	(0.01)	(0.00)	(0.04)	(0.17)	(0.09)	(0.03)	(0.01)
High	−0.18***	0.10***	4.05***	0.18***	0.03***	0.50***	14.75***	5.85***	−1.37***	0.07***
school	(0.00)	(0.00)	(0.04)	(0.01)	(0.00)	(0.04)	(0.23)	(0.11)	(0.03)	(0.01)
College	−0.21***	0.11***	6.46***	0.27***	0.03***	1.00***	37.96***	13.61***	−1.72***	0.16***
	(0.00)	(0.01)	(0.06)	(0.01)	(0.00)	(0.04)	(0.42)	(0.24)	(0.04)	(0.01)
Observations	110,063	110,063	129,629	132,810	132,810	97,523	128,038	129,903	132,810	132,810
R-squared	0.22	0.30	0.40	0.06	0.01	0.05	0.25	0.20	0.11	0.06
Mean DV	0.12	0.86	8.06	0.62	0.96	7.18	23.99	23.06	3.94	0.41
Panel B. Family-fixed-effects models										
Father's education										
Middle	−0.09***	0.04***	1.50***	0.05***	0.01***	0.16***	3.02***	1.55***	−0.32***	0.03***
school	(0.01)	(0.01)	(0.04)	(0.01)	(0.00)	(0.05)	(0.25)	(0.13)	(0.04)	(0.01)
High	−0.12***	0.06***	2.74***	0.11***	0.01***	0.32***	10.04***	3.92***	−0.77***	0.05***
school	(0.01)	(0.01)	(0.05)	(0.01)	(0.00)	(0.05)	(0.33)	(0.16)	(0.04)	(0.01)
College	−0.14***	0.07***	4.73***	0.16***	0.02***	0.66***	28.77***	10.15***	−1.06***	0.09***
	(0.01)	(0.01)	(0.08)	(0.01)	(0.01)	(0.07)	(0.57)	(0.30)	(0.05)	(0.01)
Observations	100,171	100,171	127,649	132,810	132,810	84,562	125,307	128,263	132,810	132,810
R-squared	0.57	0.64	0.78	0.63	0.60	0.69	0.72	0.71	0.68	0.64
Mean DV	0.12	0.87	8.06	0.62	0.96	7.17	23.96	23.06	3.94	0.41

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

Notes: Standard errors, clustered at the family level, are in parentheses. The regressions are weighted using the inverse of the probability of linkage between the 1940 Census, historical censuses, and Numident using probit models conditioning on individual sex, race, and origin, father's occupation dummies, mother's education dummies, father's marital status dummies, father's number of children in the household at 1940, father's age, and individual birth-state-by-birth-year fixed effects. All regressions include individual race, sex, and origin dummies, fixed effects for birth state by birth year. The outcome in column (1), child's old-for-grade status, is a dummy indicating the age of child being at least two years less than the median age of the grade they have attained. DV stands for dependent variable.

Source: As for Table 1.

the SSA Numident files and designate an old-for-grade status to those who do attend school but whose grade level lies at least two years below the age-specific median grade level in the data. Silles (2017) uses a similar method to determine the grade retention status of children. However, to obtain a precise grade retention status, we need information on exact state-level enrolment-age policies as well as the degree to which individuals comply with those policies. We call this variable 'old for grade', as some individuals start school later than others and not all people who are old for grade experience a grade retention.

We also use data on school attendance to generate a dummy indicating whether or not a person is currently attending school. We exclude individuals under six years old. We explore the effect of father's schooling on these outcomes using the sibling-fathers sample. The results are reported in Table 7, columns (1) and (2), for the OLS (panel A) and family-fixed-effects (panel B) strategies. All regressions include the fixed effects and individual covariates used in the main results in Table 2. The family-fixed-effects models suggest that having a college-educated or high-school-educated father is associated with a 13.5- or 12.1-percentage-point lower likelihood of old-for-grade status and a 6.6- or 6.2-percentage-point higher likelihood of attending school, respectively.

We continue to explore possible mechanisms by exploiting the available data on paternal socioeconomic measures. These results are reported in columns (3)–(10) of Table 7. There are strong associations between father's education and mother's years of schooling and between father's education and a range of other paternal characteristics, including longer duration of employment, being in the labour force, having a higher income or SEI, having fewer children, and owning the dwelling of residence. All these effects can be translated into improvements in well-being, better access to material resources, better healthcare access, better access to high-quality insurance, and a generally healthier environment during childhood, all of which in turn add to children's health capital over their childhood and can be detected in their old-age longevity and mortality improvements (Van Den Berg et al. 2006, 2009, 2011, 2015; Strand and Kunst 2006a, 2006b; Fletcher et al. 2010; Black et al. 2015; Scholte et al. 2015; Wherry and Meyer 2016; Sohn 2017; Fletcher 2018; Wherry et al. 2018; Goodman-Bacon 2021; Noghanibehambari 2022b, 2022c).

Conclusion

In an early book, Mangold (1920) suggests that parental SES, as well as parental education and knowledge, can directly or through other channels affect child health outcomes, such as disability and mortality. Several case studies also suggest that the parental non-genetic package (education, income, housing, wealth, health knowledge, etc.) influences child development and health outcomes and that this correlation is not mere coincidence (Levy 1919; Wile and Davis 1939; Gough 1946). Recent research establishes this *beyond-coincidence* relationship and documents a direct effect of the parental package and specifically parental education on children's health outcomes (Currie 2009; Chou et al. 2010; Ross and Mirowsky 2011; Reinhold and Jürges 2012; Currie and Goodman 2020). The current study adds to this long-standing literature by evaluating the effect of father's education on an important and precise measure of their children's long-run health: old-age mortality.

To account for shared characteristics of the environment in which fathers grow up and to control for the shared family experiences and childhood exposures that affect the father's educational decisions and also confound the children's old-age mortality equation, we implement a family-fixed-effects model. This strategy compares the within-family old-age mortality outcomes of cousins whose fathers experience and share the same family but obtain different years of schooling. Comparing the family-fixed-effects estimates with the OLS results suggests that common exposures and shared characteristics of siblings generate very little bias in the coefficients. The findings imply very robust and consistent evidence that father's education is positively associated with children's longevity. On average, children of college-educated fathers live 4.6 months more than low-educated fathers, conditional on survival up to age 47. This is roughly 74 per cent of the gap between females and males in the outcome (age at death).

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