Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

HHS Public Access

Author manuscript

Demography. Author manuscript; available in PMC 2021 August 01.

Published in final edited form as:

Demography. 2020 August ; 57(4): 1513–1541. doi:10.1007/s13524-020-00892-6.

The Effects of Education on Mortality: Evidence from Linked U.S. Census and Administrative Mortality Data

Andrew Halpern-Manners,
Department of Sociology, Indiana University

Jonas Helgertz,
Minnesota Population Center, University of Minnesota

Centre for Economic Demography and Department of Economic History, Lund University

John Robert Warren,
Department of Sociology, Minnesota Population Center, University of Minnesota

Evan Roberts
Department of Sociology, Minnesota Population Center, University of Minnesota

Abstract

Does education change people's lives in a way that delays mortality? Or is education primarily a proxy for unobserved endowments that promote longevity? Most scholars conclude that the former is true, but recent evidence based on Danish twin data calls this conclusion into question. Unfortunately, these potentially field-changing findings—that obtaining additional schooling has no independent effect on survival net of other hard-to-observe characteristics—have not yet been subject to replication outside Scandinavia. In this article, we produce the first U.S.-based estimates of the effects of education on mortality using a representative panel of male twin pairs drawn from linked complete-count Census and death records. For comparison purposes, and to shed additional light on the roles that neighborhood, family, and genetic factors play in confounding associations between education and mortality, we also produce parallel estimates of the education-mortality relationship using data on (1) unrelated males who lived in different neighborhoods during childhood; (2) unrelated males who shared the same neighborhood growing up; and (3) non-twin siblings who shared the same family environment but whose genetic endowments vary to a greater degree. We find robust associations between education and mortality across all four samples, although estimates are modestly attenuated among non-twin siblings and twins. These findings—coupled with several robustness checks and sensitivity analyses—support a causal interpretation of the association between education and mortality for cohorts of boys born in the U.S. in the first part of the 20th century.

The association between educational attainment and adult mortality in modern developed societies is well known and virtually universally observed (Elo and Preston 1996; Hummer and Hernandez 2013; Hummer and Lariscy 2011; Kitagawa and Hauser 1968, 1973; Lleras-

Please direct correspondence to Andrew Halpern-Manners, 1020 E. Kirkwood Avenue, Ballantine Hall 744, Indiana University, Bloomington, IN 47405, ahm@indiana.edu.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Muney 2005; Phelan et al. 2004; Preston and Taubman 1994). Sizable educational gradients in individuals' ages at and causes of death have been detected across birth cohorts, in different population groups, and in many social and institutional contexts (Hayward, Hummer and Sasson 2015). What is less clear is *why* these educational gradients exist. Are educational attainment and human survival etiologically linked, such that obtaining more schooling causes people to enjoy lower levels of mortality and longer lives? Or are the two variables related to one another because common endowments influence both, generating a spurious (or partially spurious) association?

Answering these questions is of profound scientific and policy significance. The magnitude of the association between education and mortality is large (Hummer and Lariscy 2011). If education causally affects mortality, investments in schooling could be an efficient and cost-effective means to reducing the "longevity penalty" that some groups face. On the other hand, if obtaining more schooling on its own does not cause people to live longer, then efforts to reduce mortality differentials by increasing education would be of little value (Hummer and Hernandez 2013). In the first scenario, education is a causal variable and could be the target of longevity-enhancing interventions. In the second, it is a (at least partial) proxy for the actual drivers of human survival.

In this article, we link records across different administrative data sources to create a set of nationally representative longitudinal samples of male-male twins, non-twin siblings, unrelated neighbors, and unrelated non-neighbors. We then use these samples to derive causal estimates of the relationship between education and longevity for the most recent cohort of American men to complete their lifespan. The unique data at our disposal, which we describe in more detail below, allow us to build up from a conventional covariate adjustment design to a more strenuous test of the causal relationship between education and mortality that accounts for hard-to-observe confounds at several theoretically relevant levels of analysis. By comparing estimates obtained using different estimation strategies and across different strategically selected subsamples of the adult population, we (1) assess the degree to which specific background and contextual characteristics confound the association between education and mortality and (2) evaluate variation in education effects across subgroups defined by their socioeconomic characteristics. We know of no prior work in the U.S. (or elsewhere) that has carried out such an exercise.

Background

Understanding the origins of educational gradients in health and mortality has long been a priority of America's research and public health agenda. Causal accounts have traditionally focused on the importance of resources, skills, and knowledge (Baker et al. 2011; Link and Phelan 1995; Mirowsky and Ross 2003; Rogers, Hummer and Everett 2013; Ross and Wu 1996), acquired through education, and then translated through multiple mechanisms into better health behaviors and outcomes (Denney et al. 2010; Hayward et al. 2015; Hummer and Hernandez 2013; Mirowsky and Ross 1998; Phelan, Link and Tehranifar 2010). The basic conceptual model that underlies this account—which flows directly from Link and Phelan's (1995) work on fundamental cause theory and which complements other foundational work in medical sociology (see, e.g., Cockerham 2005)—is summarized

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

graphically in Figure 1. In the figure, causal arrows connect education, E, to a series of mediating variables, R (economic and social resources), S (cognitive skills), K (knowledge and other flexible resources), and V (health behaviors), which in turn influence health (through a variety of more proximate channels not shown) and mortality, M.

Associations between education and mortality could also arise if the two variables share common causes, inducing a spurious (or partially spurious) relationship between E and M (Behrman et al. 2011). Potential confounds include people's social or economic background, their intelligence, their early-life health, and any other hard-to-observe endowments or contextual exposures that jointly predict educational attainment and survival (Hayward et al. 2014). Adding these variables—labeled B, I, H, and Z, respectively—to the causal diagram specified above, as in Figure 2, opens a series of backdoor (i.e., non-causal) paths that connect E to M ($E \leftarrow B \rightarrow M$, $E \leftarrow I \rightarrow M$, $E \leftarrow H \rightarrow M$, and $E \leftarrow Z \rightarrow M$), raising doubts about the causal nature of the association between the two variables. This concern has led some researchers to question whether education *causally* affects mortality (as medical sociological theory suggests), or whether the observed association is merely the end state of a more complicated sequence of selection processes (Behrman et al. 2011; Gottfredson and Deary 2004).

Empirically adjudicating between these perspectives is methodologically challenging. Most work has relied on covariate adjustments to rule out possible confounders and isolate (presumably causal) effects (Kitagawa and Hauser 1973). Findings from these analyses have shown that the association between education and mortality is robust to the inclusion of several covariates, including measures of intelligence (Link et al. 2008), race (Montez et al. 2011), childhood socioeconomic status (Montez et al. 2011), and early-life health endowments (Montez and Hayward 2011). If these statistical controls are enough to eliminate the threat posed by omitted variable bias (i.e., all of the backdoor paths running from E to M can be closed by conditioning on observed confounds), then the parameter of interest (the effect of education) is identified and the conditional association between education and mortality can be said to be causal.

Efforts to validate this assumption have taken several forms. One approach is to instrument education using historical information about compulsory school attendance and/or child labor laws, mimicking an experimental setup where exogenous factors sort individuals into different levels of the treatment (educational attainment). Although early proponents of this strategy observed significant (and substantively large) effects associated with education (Lleras-Muney 2005), replications attempts have frequently failed (see, e.g., Black, Hsu and Taylor 2015; Mazumder 2008). One reason could be the strength of the instrument: Compulsory schooling laws are, in many cases, only weakly related to variation in educational attainment (and are only relevant for the subsample of students who were induced to obtain additional schooling), making it difficult to estimate effects with sufficient precision (or to make claims about effects for students not on the margins) (Fletcher 2015). Studies that use birth registry data from outside the U.S. have sought to circumvent this problem by analyzing larger samples (Lager and Torssander 2012; Meghir and Palme 2005), but findings there have been mixed as well. In some cases, researchers have observed sizable declines in mortality for birth cohorts (or jurisdictions) that were compelled to attend school

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

for an additional year (Fischer, Karlsson and Nilsson 2013; van Kippersluis, O'Donnell and van Doorslaer 2011); in other cases, they have not (Braakmann 2011; Clark and Royer 2013; Lager and Torssander 2012; Meghir, Palme and Simeonova 2018). Scholars have speculated that these discrepancies could be due, in part, to heterogeneity in the effects of education across birth cohorts and/or social and political contexts (Hayward et al. 2015), but even this hypothesis has been difficult to confirm.

As an alternative to this approach, a small but growing number of studies have turned to within-twin pair comparisons as a way to "difference out" observed and unobserved factors (e.g., *B*, *I*, and *Z*) that could confound the association between education and mortality (see, e.g., Behrman et al. 2011; Lundborg, Lyttkens and Nystedt 2016; Madsen et al. 2010). Twins —even those who end up with different levels of schooling—experience very similar social, economic, family, school, neighborhood and other environmental exposures, and have identical (in the case of monozygotic or MZ twins) or similar (in the case of dizygotic or DZ twins) genes. If educational attainment is associated with mortality among pairs of twins that are concordant (or mostly concordant) with respect to these endowments, but discordant with respect to their educational attainment, the association is (conditional on several identifying assumptions) less likely to be spurious. All shared genetic and environmental exposures fall out of the model, providing arguably cleaner estimates of the effect of obtaining higher levels of education.

Applications of this strategy have produced intriguing, and sometimes surprising, results. Using a large population-based data set from Denmark that included just over 2,500 identical (MZ) twin pairs born between 1921 and 1950, Behrman et al. (Behrman et al. 2011) showed that the estimated causal effect of education on mortality in Denmark is reduced to zero when comparing the mortality outcomes of MZ twins who are discordant on education; broadly similar patterns were observed for DZ twins. That the same was not true for pairs of unrelated individuals suggests that shared early-life endowments and exposures —common within pairs of identical twins but not within pairs of unrelated adults—may explain, or partially explain, the existence of educational gradients in mortality. This inference is generally consistent with a *non*-causal interpretation of the diagram presented in Figure 2. Other researchers who have used the same Danish data have reached similar conclusions, observing null or attenuated effects when modeling within-twin pair differences in mortality or related health outcomes (Madsen et al. 2010; Osler, McGue and Christensen 2007).

We believe these findings are important and provocative, but we also see reasons for skepticism. First, it is unclear whether similar findings would hold if the same twin-differencing models were fit using data from outside of the Danish context (Hayward et al. 2015; Lundborg et al. 2016). It could be the case that Denmark's set of social and educational policies render the education-mortality relationship less important than in the U.S. or other Western countries (Lundborg et al. 2016), where the social safety net is less comprehensive. In fact, the role of the welfare state, levels of inequality, demographic differences, and differences in life expectancy could all contribute to cross-national differences in the education-mortality relationship, as well as variation at the sub-national level (see, e.g., Montez et al. 2019). Until now, it has been impossible to consider this

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

possibility (at least as it pertains to the U.S.) because there have been no large, nationally representative samples of U.S. twins with requisite information about education and mortality. Current U.S.-based twin data repositories (e.g., the NAS-NRC Twin Registry of WWII Military Veterans and the Minnesota Twin Registry) have been used for a variety of research on *health* gradients (Amin, Behrman and Kohler 2015a), but these studies pertain to particular sub-populations (e.g., WWII veterans) or to people in particular geographic regions (e.g., Minnesota or Southern California), and only the NAS-NSF data include mortality information for most respondents.

Second, it is not clear that effects estimated using twin-differencing models pertain equally within all population subgroups. Prior twin-based estimates of the effects of education on health and mortality can be thought of as estimates of the *average* treatment effect (or ATE). A recent study by Heyward et al. (2015), however, suggests that the association between education and mortality may be stronger for certain segments of the population; in particular, for men, for whites, and for younger adults. These findings are consistent with a growing number of other studies, which show that the association between education and health can vary significantly by social background characteristics (Andersson 2016; Bauldry 2014; Conti and Heckman 2010; Ross and Mirowsky 2006, 2011; Schafer, Wilkinson and Ferraro 2013).

What are the processes driving these results? Effect heterogeneity could stem from (1) resource substitution or (2) cumulative advantage processes (Ross and Mirowsky 2011). If the education-mortality relationship is characterized by resource substitution, we would expect to see larger effects among individuals from less advantaged backgrounds—because individuals from less advantaged backgrounds have fewer alternative resources to fall back on, making education more decisive. The cumulative advantage perspective, on the other hand, predicts the opposite: the effects of education should be greater for the most advantaged individuals, not the least, because individuals from more advantaged backgrounds are in a better position to leverage the health-enhancing potential of education. Although these perspectives lead to fundamentally different predictions about how the effects of education will be distributed, their methodological implications are the same. If the health returns to education vary in meaningful ways across subgroups, estimates of the ATE, which represent a weighted average of all group-specific estimates, would obscure this variation and potentially miss non-zero (or null) effects within certain segments of the population.

Finally, it is not known *why* twin-based studies have produced results that diverge from findings obtained using more conventional covariate adjustment designs and/or alternative identification strategies. In their conclusion, Behrman and colleagues (2011) write that education may serve as “a marker for parental family and individual-specific endowments that are uncontrolled in the usual estimates” (p. 1367), but they do not provide additional information about what those endowments might be. Because twins share the same (or most of the same) genetic, family, neighborhood, and school characteristics, finely grained analyses of individual confounds (i.e., the additional variables included in Figure 2) are generally not feasible. Although this does not diminish the overall contribution of their

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

research, it does lead to a fairly coarse “reduced form” assessment of the underlying causal model.

RESEARCH DESIGN

Data and Measures

We address the above listed issues using a unique and untapped data resource: the digitized complete-count U.S. Censuses for 1920 and 1940. With support from the U.S. National Science Foundation and the U.S. National Institutes of Health, and in collaboration with Ancestry.com, the Minnesota Population Center has now finished work on complete-count versions of the 1850–1940 Census files (Ruggles et al. 2019). These data are freely available at ipums.org.

From the 1920 U.S. Census, we extracted records for all male children born in the U.S. between 1910 and 1920 (n = 11,749,361).[1] Then, using techniques described in detail below, we linked those records to the 1940 U.S. Census, from which we obtained information about educational attainment. Of the 11,749,361 U.S.-born boys in 1920, we were able to confidently and uniquely link 34 percent (n = 4,153,206) to 1940 Census records. Finally, information about age at death was obtained by linking 1920–1940 Census records to death records in the (1) NUMIDENT and to (2) the Social Security Death Master File (SSDMF). Of the 4,153,206 males linked across 1920 and 1940, we were able to link 41 percent (n = 1,720,980) to mortality records. In all, then, we were able to fully and confidently link records for about 14 percent of the baseline population. Although simple side-by-side comparisons can be misleading (due to differences in the underlying quality of record linkages), we believe these results stand up favorably to other historical record linking efforts (see, e.g., Beach et al. 2016; Ferrie 1996). Below, we (1) describe our record linking procedures in more details, (2) compare the characteristics of successfully linked cases to the characteristics of the baseline population, and (3) discuss the strategies we used to make adjustments for non-random selection into our fully linked sample.

Linking 1920 to 1940 U.S. Census Records

Linking the 1920 baseline population of 11,749,361 boys to the 1940 Census requires that we first define the universe of potential matches. To make the task computationally tractable, we restrict the population of potential matches to records that display identical or similar characteristics on features that should be consistent over time (e.g., gender and place of birth).[2] As an example, when attempting to find Michael Corcoran, male, born in Massachusetts according to the 1920 Census, we limit the population of potential matches in 1940 to males who, in 1940, reported Massachusetts as their place of birth. Since age is reported in the 1920 and 1940 Censuses rather than date of birth, and because of reporting

[1] In ongoing work, we are exploring the feasibility of implementing similar machine-linking procedures for a subsample of female children (in female-female sibling and twin pairs and female-male sibling and twin pairs). Unfortunately, the technical challenges involved in obtaining reliable links for girls are much steeper, due to more frequent name changes at marriage. We discuss this issue in more detail in our conclusion.

[2] The place of birth assumption is probably not entirely accurate, but the implications thereof should not be important. Furthermore, the potential benefits of relaxing this assumption should be weighed against the obvious downside of increasing the population of potential matches and, thereby, also the risk of declaring false positives.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

inaccuracies, we allow for deviations in birth year across data sources. Specifically, we stipulate that birth years must be within +/− three years, implying that each unique individual who, according to the 1920 Census, was born in 1919 will be compared and possibly linked to individuals who in the 1940 Census, conditional on sex and place of birth being the same, were recorded as being born between 1916 and 1922.

Allowing for a broad range of potential matches has advantages as well as disadvantages. One key advantage is the ability to link individuals for whom year of birth in either Census was reported, enumerated, or digitized incorrectly by more than a few years. This cuts down on the chances of false negatives (i.e., rejecting a candidate match that is in fact correct). The main disadvantage is the increased risk of false positives (i.e., declaring a match when the match is incorrect). If our hypothetical Michael Corcoran, born in 1919 and observed in the 1920 Census, dies at the age of two, he will not be enumerated in the 1940 Census. If his parents have another male child, born in 1922, and decide to also name him Michael, this identically named but different individual would be one of the candidate matches. More generally, the wider the birth year window, the larger the pool of potential matches and, at the same time, the higher the probability of (1) finding the right individual *and* (2) making an incorrect link. This is an issue that we return to below.

Employing a probabilistic method of record linkage means that an algorithm is trained to recognize patterns in a dataset of potential matches that are consistent with a true match. We use a modification of Feigenbaum's (2016) probit regression approach, which—like other methods of supervised machine learning—requires input from training data. The training data represent a subsample of the population that one wishes to link, but where links have been declared by a trained human in order to ascertain that confirmed links are as accurate as possible. We not only use the training data to calibrate the linking algorithm, but also to evaluate how well it performs at declaring matches and avoiding false positives.

To start, we randomly selected 1,000 individuals from the 1920 sample who were linked to a similarly defined universe of possible 1940 matches. Here, the universe of potential matches was limited to cases where the name similarity scores on both the first and last name (using the Jaro-Winkler algorithm) were at least 0.8 (e.g., "Bertus Wilson" and "Burtis Watson"). We assessed all potential matches using the wealth of digitized historical information available from Ancestry.com. Historical information about parents, siblings and the focal individual's place of residence from the time between the 1920 and the 1940 Census allowed us to make confident assessments regarding the validity of potential matches, and death records allowed us to cut down on false positives. Using these procedures, we were able to manually declare 50.2 percent of the training data sample as uniquely matched across 1920 and 1940.

To calibrate our linking algorithm, we implemented a "train-test-split" procedure using our training data (in which true matches are known). In the first part of the procedure, we split our training data into two equally sized parts. To train the algorithm, we fit a probit regression model on one-half of the sample and then evaluated its out-of-sample performance on the other. The model specification we used is similar to the one proposed by Feigenbaum (2016), but we added additional individual- and household-level covariates to

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

reduce the risk of false positives. Results from the model inform the algorithm as to which, if any, of the "plausible set" of matches should be considered a valid link. The algorithm declared a unique link based on (1) the greatest similarity between any 1-to-1 match (technically the predicted probability based on the probit regression estimates); and (2) the relative difference between the best and second-best possible match. By looping multiple times over a range of realistic values on both parameters, we were able to choose values for (1) and (2) that optimized the overall performance of the linking algorithm. We judged overall performance by the algorithm's ability to minimize false positives (incorrectly linked cases), while maximizing true positives (correctly linked cases) and true negatives (correctly unlinked cases).

In selecting thresholds for declaring matches in our data, we used the Matthew's Correlation Coefficient (MCC), which is an especially useful measurement for two-class data where the classes are not well balanced (Chicco 2017). This is definitely the case in our situation, where the 1,000 individuals in the training data are, on average, linked to 23.2 potential matches in the 1940 Census and are thus represented by a data set of 23,200 observations. In the data set, 502 observations (about 2 percent of potential matches) were declared to be a true match. The MCC, in Eq. (1) below, compares the predictions of the algorithm to all possible outcomes (true/false positives/negatives) and provides a single metric (ranging from −1 to +1) to be used to select which thresholds to use. The formula is as follows, where TP represents true positive, TN represents true negative, FP represents false positive, and FN represents false negative:

$$\mathrm{MCC} = \frac{TP \times TN - FP \times FN}{\sqrt{(TP+FP)(TP+FN)(TN+FP)(TN+FN)}} \quad (1)$$

Linking Census Records to Mortality Records

Our strategy for linking the 1920–1940 Census sample to mortality records proceeded in a similar manner, with some unavoidable differences due to variable availability. The primary source for death records was a merged version of the Social Security Administration's NUMIDENT data files containing social security claims data ($N \approx 35{,}000{,}000$ for both men and women). NUMIDENT includes several pieces of information not provided by the Social Security Death Master File (SSDMF). In particular, it includes state of birth, the person's gender, as well as their mother's and father's first and last names. We used this information to (1) improve the precision of the training data file and (2) create linking features for the machine learning algorithm to use. To provide better coverage of deaths (the NUMIDENT data contains no deaths after 2007 and very few deaths prior to 1973), we supplemented the NUMIDENT records with a secondary source of mortality data: the publicly available Social Security Death Master File (SSDMF). The SSDMF—which records mortality information based on reports from funeral directors, family members, financial institutions, the post office, and various government agencies—includes deaths occurring as recently as May 2013 ($N \approx 93{,}000{,}000$ for both men and women).

In both data files, the first recorded deaths are from the early 1900s, but coverage during the first half of the century, into the 1960s and 1970s, was less complete. A comparison of our

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

death data to published life tables from the Social Security Administration (Bell and Miller 2005) suggest that our count of the cumulative percent dead by 1960 is about 6 percentage points too low; that our count of the cumulative percent dead by 1970 is about 11 percentage points too low; and that about 2.5 percent of our sample should have survived beyond 2013. If we make the reasonable assumption that early deaths were more (less) likely to occur among those with lower (higher) levels of education, then our estimates of the effects of education on mortality should be attenuated toward zero. In supplementary analyses, we evaluated the extent of this bias using a re-weighting procedure that aligned our observed distribution of deaths to the distribution inferred from published tabulations (see Appendix A). Results from these analyses suggest that our within-pair estimates may be downwardly biased by as much as 8 percent. This makes our estimates of education effects necessarily conservative.

Study Sample

To be included in our analyses, both members of pairs of twins, non-twin siblings, neighbors, or unrelated individuals had to be fully linked across the 1920 and 1940 Censuses and mortality records. We also drop a small subset of pairs where ages at death are implausible or where at least one member of the pair is missing information on education. For the twins, displayed in Table 1, this restriction results in the sample size of $n = 5{,}216$ unique individuals in 2,608 complete twin pairs.[3] For our subsamples of random individuals, non-relative neighbors, and non-twin siblings the final sample sizes are $n = 1{,}658{,}836$, 1,604,936, and 328,352, respectively.

One concern is that sample selection—occurring either through incomplete record linkage or missing data—results in an analytic sample that differs from the target population in nontrivial ways. Table 1 indeed shows differences by race and geographic region in the likelihood or remaining “in sample” after our various selection filters are in place (parallel tables for the non-twin subsamples can be found in appendix Tables A1 through A3). Non-white individuals and individuals born in the south are less likely to be in the linked sample than in the original 1920 sample, with the opposite applying to whites and individuals from the northeastern part of the country. Apart from this, there does not seem to be any selection into the linked sample on the basis of observed characteristics.[4] As described in Appendix A, in our analyses we re-weight the data to adjust for discrepancies between our analytic sample and the population we are trying to describe. Weights were generated by calculating the inverse of the probability of successful linkage, where the probability of linkage was determined using a simple logit model. Predictors in the model included race, region, family size, and householder’s occupation category.[5]

[3]We are unable to distinguish MZ from DZ twins in our analyses, but a publication from the period in question—which estimates that, among same-sex twin pairs born between 1922 and 1930, 50% were MZ (Hamlett 1935)—provides a rough guide. It is possible that the actual percentage we end up with in our analytic sample is somewhat lower (because pairs have to be discordant on education to contribute to our preferred within-pair estimates and rates of discordancy are likely to be lower among MZ twins), but we do not expect the difference to be especially large. Supplementary analyses of data from the Virginia Twin Registry show that, of male MZ twins born between 1910 and 1920 (and still alive in 1987), the rate of discordancy was 36%. The same figure for male-male DZ twins born during the same time period was 45%. If we take these percentages at face value, they imply that around 45% of discordant pairs [(0.36/(0.36+0.45) x 100% = 45%] in our twin sample are likely to be MZ.

[4]The primary determinant of successful linkage was name commonality (i.e., the number of people living in the same state with the same first and last name). In supplementary analyses, described in the Appendix, we show that name commonality is orthogonal to educational attainment net of basic sociodemographic and geographic controls.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Empirical Strategy

To estimate the effects of educational attainment on mortality, we use a standard fixed effects specification for within-pair estimation (we refer to twins here for convenience, but the same models will be fit for non-twin pairs as well, as we note below):

$$M_{ij} = \alpha + \beta S_{ij} + C_j + G_j + \varepsilon_{ij}, \quad (2)$$

where M_{ij} is a continuous measure of age at death for individual i ($i = 1, 2$) in twinship j ($j = 1, 2, \ldots, N$); S_{ij} measures years of schooling; C_j is a measure of unobserved contextual characteristics (e.g., family, peer group, or neighborhood attributes); G_j is a measure of unobserved genetic endowments; and ε_{ij} is a random individual-level error term that is assumed to be uncorrelated with the other explanatory variables in the model. In a within-twin-pair model, the unobserved components in Eq. (2) are "controlled away" by modeling differences within pairs of twins:

$$M_{1j} - M_{2j} = \Delta M_j = \beta \Delta S_j + \Delta \varepsilon_j, \quad (3)$$

where the Δ's represent differences between variables for the jth twin pair (i.e., $S_{1j} - S_{2j}$). This approach eliminates the effects of unobserved contextual characteristics (C_j)—since the vast majority of twins experience the same family, school, and neighborhood environments while growing up. It also (at least partially) eliminates the effects of unobserved genetic endowments (G_j)—since MZ twins share 100% of their genes at birth and DZ twins, like non-twin siblings, share 50% of their genes on average.

The modeling strategy described above can be modified in three respects to assess the degree (and nature) of omitted variable bias in prior U.S.-based work that uses data on unrelated individuals and more standard estimation procedures. First, we can estimate "unpaired" OLS models of age at death for our subsample of unrelated males who live in different neighborhoods, and parallel models for our subsamples of neighbors, siblings, and twins.[6] In these analyses, we include covariates for family socioeconomic origins, race, family structure and composition, nativity status, and geography; we expect the results to reproduce findings from prior research. Second, we can estimate within-neighborhood fixed effects models for our sample of unrelated pairs of males who lived in the same neighborhood. These models allow us to consider the degree to which the association between education and mortality is confounded by geographic and neighborhood factors that might be unobserved using a more conventional covariate adjustment design. Third, we can estimate within-family fixed effects models for our sample of pairs of male-male non-twin siblings. These models assess the degree to which the association between education and mortality is confounded by shared environmental, neighborhood, geographic, family, and genetic conditions, but it is a less stringent test than the within twin pair analyses because non-twin

[5]There is room for debate about whether such weighting adjustments are necessary in the first place (Amin et al. 2015b; Boardman and Fletcher 2015). Our within-pair models provide protection against differential selection during the linkage stage (and other related concerns about external validity) by adjusting for all characteristics (observed or otherwise) that are shared within pairs. We suspect that this is why weighted and unweighted estimates closely agree with one another.

[6]Fixed effects models for pairs of unrelated individuals will produce point estimates (but not variance estimates) that are equivalent to an unpaired model with identical controls. We opted to use pairs for this subsample to ensure consistency with our treatment of the other subsamples.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

siblings generally share fewer genetic endowments. Together with the estimates obtained using our sample of twins, these analyses will provide useful information about the magnitude of education effects on mortality and the role played by different sets of theoretically relevant (but typically hard to observe directly) confounds.

RESULTS

In Table 2, we present key descriptive statistics for education and mortality variables for each of the four analytic subsamples: Unrelated non-neighbors, unrelated neighbors, non-twin siblings, and twins. The mean years of education is between 10.13 and 10.40 across analytic samples. Within pairs, the rate of educational discordance—the percentage of pairs in which the two differed in their years of schooling completed—was high among unrelated non-neighbors (84%) and unrelated neighbors (80%), lower among non-twin siblings (63%), and lowest among twins (40%). Likewise, the mean absolute difference in years of schooling within pairs was highest among unrelated non-neighbors (3.2 years) and unrelated neighbors (2.7 years), lower among non-twin siblings (1.6 years), and lowest among twins (0.9 years). The mean age at death was between 74.8 and 76.0 across the four groups.[7]

In Table 3, we present estimates of the effect of education—expressed as years of schooling completed—on age at death. All models, as we noted earlier, are weighted to account for differential probabilities of selection into the final linked sample.[8] In the four leftmost columns of results, we present unpaired (OLS) models for unrelated non-neighbors, unrelated neighbors, non-twin siblings, and twins. In these models, we treat members of each pair as individuals and ignore pair structures (standard errors are clustered at the pair level to account for non-independence). All unpaired models adjust for the demographic variables listed in Table 2, plus state of residence in 1920. As expected, there is a positive and significant association between education and age at death. For each additional year of schooling an individual completes, they live about four-tenths of a year (or 4.8 months) longer, on average. The fact that this result is so consistent across sub-samples suggests that, when unobserved similarities are ignored, twins and non-twin siblings are unremarkable relative to each other and relative to subsamples composed of unrelated individuals. We take this as a sign of external validity.

In the right four columns of results in Table 3 we present paired models—corresponding to Eq. 3 above—for pairs of unrelated non-neighbors, unrelated neighbors, non-twin siblings, and twins. The model for unrelated non-neighbors adjusts for the same set of covariates as the unpaired model above. The model for unrelated neighbors makes the same adjustments, while also differencing out all aspects of the neighborhood environment that neighbor pairs have in common. The model for non-twin siblings and twins difference out all aspects of the shared neighborhood environment, all aspects of the shared family environment, and any other endowment both members of the pair possess. For unrelated non-neighbor pairs and

[7]The intra-pair correlations presented in Table 2 for twins and non-twin siblings can be used to back out a rough estimate of broad sense heritability, or H^2. If we assume the twin sample is approximately 50% MZ and 50% DZ—and if we invoke the usual assumptions regarding equal environments, minimal gene-environment interactions, and comparable shared environments within pairs—then Falconer's (1960) formula suggests the broad sense heritability of age at death for members of this cohort was approximately $1.5 \times (r_{\text{twins}} - r_{\text{siblings}}) = 1.5 \times (0.21–0.12) = 0.14$. We reiterate that this is a rough estimate.

[8]The unweighted estimates (not shown) were substantively identical.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

unrelated neighbor pairs, the within-pair estimates of the effect of years of schooling on age at death ($\hat{\beta}$ = 0.40 in both cases) are about the same as the unpaired versions ($\hat{\beta}$ = 0.39 in both cases). For siblings and twins, the estimates are modestly attenuated ($\hat{\beta}$ = 0.34 and 0.35), but still non-zero and significant.[9] These estimates suggest that a conventional covariate adjustment design may modestly overstate the magnitude of the education-mortality relationship, insofar as it omits important but hard-to-observe family or individual endowments, but that the causal path from education to survival remains intact.

In Table 4, we repeat the analyses in Table 3 using a categorical parameterization of education to allow for possible non-linearities (Montez, Hummer and Hayward 2012). Here, we classify people as having completed fewer than 12 years schooling, or 12 years of schooling or more, which corresponds to the margin between high school completion or not.[10] The results are virtually identical to those presented previously. In the unpaired models, there are sizable and significant differences in life expectancy by level of education. Compared to those who did not complete secondary school, those who did lived between 2.2 years (the unrelated non-neighbor, unrelated neighbor, and non-twin sibling sub-samples) and 2.5 years (the twin subsample) longer, on average. We see the same thing in the paired fixed effect estimates, but with attenuated coefficients for non-twin siblings and twins. Once we difference out everything twins and siblings have in common, the coefficient for completing at least 12 years of schooling is reduced to approximately 1.6. All coefficients retain their significance at the $p < 0.10$ level or better.

Limitations and Robustness Checks

The results presented above provide evidence of a relationship between education and survival, but justifying a stronger causal interpretation of our estimates requires certain assumptions. Questions about measurement error, residual within-pair variation, and outliers have all been raised in response to prior studies, especially those that involve twins (Boardman and Fletcher 2015; Bound and Solon 1999; Gilman and Loucks 2014; Kaufman and Glymour 2011). To assess the sensitivity of our estimates to these concerns, we carried out a series of additional robustness checks. We describe these checks in more detail below.

Random measurement error.—It is well known that attenuation bias is more pronounced in fixed-effects models due to the weaker signal-to-noise ratio (Ashenfelter and Krueger 1994; Griliches 1979). For our purposes, this leads to (1) an increased chance of making a Type II error as we move from an unpaired (OLS) estimator to paired fixed effects models; and (2) possible *under*-estimates (but *not* over-estimates) of the true effect of education on length of life—particularly in our within-pair twin models, where the signal we wish to detect is at its weakest. In principle, we would be more concerned about this issue if

[9] In supplementary analyses, we pooled the non-twin sibling and twin subsamples and fit a model interacting an indicator of subsample membership and years of schooling. The results suggest that the sibling and twin estimates are not significantly different from one another ($p = 0.82$). The same is not true for a comparison of the sibling and neighbor estimates, which produced significant differences at the $p < .01$ level.

[10] We also experimented with a three-category measure of education, where education was coded as less than 12 years, 12 years, and more than 12 years of schooling. The three-category version produced a very similar (and statistically significant) educational gradient in age at death. We present results from the two-category version because cell sizes for some of the comparisons in the three-category version (e.g., more than 12 years versus less than 12 years of education) get small in the twin subsample.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

we saw evidence of substantial attenuation across subsamples (e.g., an estimate centered over zero for twins and a positively signed non-zero estimate for siblings), but this is not the case. When we formally compare the fixed effects estimates obtained for twins (where attenuation due to measurement error should be more pronounced) and non-twin siblings (where attenuation should be less pronounced), we are unable to reject the null that they are equal ($p = 0.82$). The same is true when we compare estimates for neighbors to estimates for unrelated pairs ($p = 0.81$). We interpret this to mean that measurement error is likely to be minimal.

Residual variation.—A second and more important concern relates to unobserved differences within pairs of twins. The model specified in Eq. (3) differences out all characteristics that are shared by both members of a twin (or non-twin) pair, but it does not account for characteristics that vary between members within pairs. This could bias our estimates if there are unobserved individual-specific factors, Z_{ij}, that are correlated with both amount of education completed and longevity. Differences in childhood health, intelligence, personality characteristics, and/or genetic endowments (due to the presence of DZ twins in our twin sample) are all possibilities.[11] The direction of the bias depends on the nature of the relationship: If Z_{ij} correlates with schooling and longevity in the same way (e.g., $r_{Z,X} > 0$ and $r_{Z,Y} > 0$), our estimate of β will be an overestimate of the true education effect. If the three variables correlate in opposite ways (e.g., $r_{Z,X} > 0$ and $r_{Z,Y} < 0$), it will be an underestimate (Kohler, Behrman and Schnittker 2011). To evaluate the risk that such bias poses for our analyses, we carried out a simple Monte Carlo-style simulation study. In the simulation, we randomly generated an unobserved variable, Z, whose correlation to years of schooling and age at death followed a pre-specified structure (we allowed the pairwise correlations to run from −0.30 to 0.30 in increments of 0.10). We then added Z to our within-twin pair specification, collected the resulting point estimate for years of schooling, and then averaged across 1,000 replications to stabilize the results.[12]

Findings from this exercise, which we have arranged into a simple matrix, are provided in Table 5. In scenarios where the unobserved confound is unrelated to age at death ($r_{Z,Y} = 0$), education ($r_{Z,X} = 0$), or both ($r_{Z,Y} = 0$ and $r_{Z,X} = 0$), we see little to no movement in our point estimate relative to the estimate presented in Table 3. This makes good intuitive sense, as Z is not a confound under these conditions. In scenarios where Z and years of schooling are positively (negatively) related, but Z and age at death are negatively (positively) related, we see evidence of a suppression effect that ranges in magnitude according to the strength of the

[11]The estimates presented in Tables 3 and 4 give at least some reason to think that unobserved differences in genetic endowments within twin pairs may be less consequential for our analyses. The within-twin pair estimates that we provide represent a weighted average of estimates for MZ and DZ twins (Conley, Strully and Bennett 2006). Prior research, as noted above, suggests that male-male twin pairs born during this period were approximately 50% MZ and 50% DZ (Hamlett 1935). If we set the DZ estimates equal to the age-adjusted estimates we obtain for non-twin siblings (who, like DZ twins, share 50% of their genes), we can calculate the MZ contribution to our within-twin pair results using Weinberg's (1901) method. For the within-pair model that uses a linear parameterization of education, we get a coefficient of $[0.347-0.338 \times (1-0.5)]/\ 0.5=0.356$. The fact that we do not see much of a difference between siblings and twins (and between the sibling estimates and our inferred estimates for MZ twins, who are genetically identical) does not imply that genes are somehow irrelevant to a person's educational attainment or longevity. It simply suggests that the additional endowments we are differencing out as we move from a within-sibling to within-twin pair model are not predictive of educational outcomes *and* survival. Prior work in other contexts has reached similar conclusions (Lundborg et al. 2016).

[12]This setup is conceptually similar to the type of bounding analysis performed in Rosenbaum and Rubin (1983) and Rosenbaum (1995), except we are deploying it within the context of a within-twin pair fixed effects model.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

correlations. We are less concerned about this possibility as it is difficult to think of an unobserved variable that exhibits this correlation structure. What we are more concerned about is the final scenario where Z correlates with education and age at death in the same way ($r_{Z,X}$ and $r_{Z,Y} > 0$ or $r_{Z,X}$ and $r_{Z,Y} < 0$). Results from the simulation suggest that, under this scenario, our within-twin pair estimate will be too large, but that the size of the over-estimate is likely to be modest in absolute terms. Only under fairly extreme conditions ($r_{Z,X} = -0.30$ and $r_{Z,Y} = -0.30$ or $r_{Z,X} = 0.30$ and $r_{Z,Y} = 0.30$) do we obtain coefficients that approach zero. Although we cannot definitively rule out the existence of an unobserved confound that fits this description, we can say with certainty that no *observed* variable in our data set comes close. Most observed measures that produce the required correlation with years of schooling completed (e.g., householder's socioeconomic status) are only weakly related to age at death (e.g., the correlation between householder's socioeconomic status and age at death is 0.01 in our sample of twins), and vice versa. We think these results provide reassurance against the threat of residual variation.

Outliers.—A third concern raised—particularly about twin studies that use a within-pair estimator like ours—is that non-null results could be driven by the presence of extreme values on key explanatory variables (Amin 2011; Lundborg et al. 2016). To consider this possibility, we pooled our twin and non-twin sibling subsamples (to maximize power) and then dropped all pairs where the within-pair difference in education was greater than or equal to 4 years of schooling (eliminating about 15% of all observations). Imposing this constraint did not diminish our point estimate for education ($\hat{\beta} = .39$, $p < .01$), as one would expect if extreme values were driving the results.[13] Instead, in the pooled sample—and also in supplementary analyses where we disaggregated by subsample—our estimate for years of schooling remained the same, or even increased marginally in size.

Effect heterogeneity

The findings to this point suggest that the effects of education on mortality are positive, on average, and that methodological complications are unlikely explanations for the observed relationship. Whether the same pattern holds across population subgroups, as defined by their socioeconomic status, is an open and important question. Prior theoretical work—mostly focusing on educational gradients in physical health—has developed competing theories for who stands to gain the most from completing additional schooling. One possibility is that the biggest returns go to individuals with the *fewest* advantages, because their socioeconomic success depends more critically on their educational attainment. This argument can be traced to Ross and Mirowsky's (1989) work on resource substitution theory. Another possibility is that the biggest returns go to individuals who are the *most* advantaged, because they are in a better position to leverage and consolidate the multiple social, economic, and health-related resources that education is thought to provide. This argument—which implies the presence of a cumulative advantage process—is typically referred to as the resource multiplication hypothesis (Andersson and Vaughan 2017; Ross and Mirowsky 2011; Schafer et al. 2013).

[13]These results are available upon request.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

In order to test these propositions, we fit an augmented version of our within-pair model that included an interaction between the respondent's completed education (expressed using a linear measure of years of schooling completed) and a measure of their parents' occupational standing in 1920 (derived from a constructed variable that assigns occupational income scores to each occupation based on the median income within that occupation).[14] Although the main effect of occupational standing cannot be estimated in our models—because it is perfectly correlated with the within-family fixed effects—the coefficient on its interaction with education *is* estimable and provides information about effect heterogeneity across the distribution of socioeconomic status. For the purposes of these analyses, we pooled our sibling and twin subsamples (again, to maximize power) and used the same set of inverse propensity of linkage weights as above. All other aspects of our model specification remained unchanged.

Figure 3 visualizes the main result. Occupational income scores are plotted along the *x*-axis and estimated effects, measuring the expected change in life expectancy associated with an additional year of schooling, are given by the *y*-axis. The shaded regions, going from light to dark, provide the 95%, 75%, and 50% confidence intervals around the estimated effect at each level of occupational income. That the estimates presented in the graph slope upward suggests that the longevity returns to additional school are not uniform with respect to social background, but instead grow (nearly doubling in size) as one moves from the very low- to very high-ends of the occupational income distribution.[15] This pattern is broadly consistent with the idea of resource multiplication. Completing additional schooling seems to have had beneficial effects regardless of a person's social background, but the benefits appear to be most pronounced for those who were raised in more advantaged circumstances.

DISCUSSION

Educational gradients in mortality are strong and well documented (Hummer and Hernandez 2013), but recent work has raised questions about their etiology. One possibility is that the link between education and mortality is causal: completing additional schooling promotes the acquisition of skills, resources, and knowledge that, as a package, increase a person's chances of survival (Phelan et al. 2004). Another possibility is that the two variables share common causes (Fuchs 1982), confounding effect estimates and inducing a spurious (or partially spurious) relationship. In this project, we sought to adjudicate between these possibilities using an approach that allows for credible estimates of causal effects. In a series of increasingly stringent model specifications, we were able to difference out all features of the neighborhood, family, and genetic endowment that strategically paired members of our sample had in common. What we were left with was a slightly attenuated *but still strong and significant* relationship between education and survival, with unobserved aspects of family environment acting as the most important confound. This result held across alternative parametrizations, persisted except under fairly extreme empirical conditions, and does not

[14]We used the occupational income score of the householder (Hauser and Warren 1997), which in most cases meant the focal individual's father as opposed to mother.
[15]The *p*-value on the interaction term was 0.03.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

appear to be an artifact of errors in our data, censoring, and/or other methodological considerations.

These findings help to extend the already well-developed literature on education and mortality. Determining whether educational attainment is a cause or simply a correlate of survival requires specific data and a strong research design. Scholars working in the U.S. context have made considerable headway using a combination of observational and quasi-experimental approaches (Link et al. 2008; Lleras-Muney 2005; Montez and Hayward 2014), but concerns regarding identification have lingered. Although the twin-differencing strategy we employed in our analysis has been used in prior studies to address this issue (Behrman et al. 2011; Ericsson et al. 2019; Lundborg et al. 2016; Madsen et al. 2010; Søndergaard et al. 2012; van den Berg, Janys and Christensen 2015), applications in the U.S. have not been possible due to a lack of appropriate data. Using a supervised machine-learning algorithm, we were able to link large samples of U.S.-based twins, non-twin siblings, unrelated neighbors, and unrelated people living in different neighborhoods across censuses and to administrative records containing information on the timing of their deaths. This new data resource—which includes nearly 2 million fully linked records all told—allowed for a careful consideration of confounding across several levels of analysis and new estimates of effect heterogeneity.

Although we consider these to be valuable contributions, we also recognize the need for caution. Prior research on education and mortality suggests that the strength of the relationship—and the extent to which it derives from a true causal process—may vary substantially across time, space, and populations (Cutler, Huang and Lleras-Muney 2015; Galama, Lleras-Muney and van Kippersluis 2018; Gathmann, Jürges and Reinhold 2015; Hayward et al. 2015; Kunst and Mackenbach 1994; Smith et al. 2015). In our analyses, we considered variation in educational effects across the distribution of social background (operationalized in terms of father's occupational status) using one of the first birth cohorts to experience increased access to education (Goldin 1998), but similar comparisons across birth cohorts and/or by race/ethnicity or gender were not possible given the nature of our data.[16] It may very well be that the patterns we observed for mostly white boys living in the U.S. during the first part of the 20th century do not hold for girls or minorities from the same birth cohort, for earlier or subsequent cohorts of Americans, or for individuals living in other countries.[17] The good news is that some of these questions may be answerable in the near future. In ongoing work, we are (1) using parallel machine-learning procedures to link boys who were enumerated as a part of the 1900 and 1910 censuses (born between 1890 and 1910) and (2) developing specialized routines (that capitalize on parental surname information included as a part of NUMIDENT) to link large subsamples of girls across censuses and to mortality records. Our hope is that these efforts will facilitate new analyses

[16]If it is the case that education (and, in particular, higher levels of education) has become an increasingly important vehicle for obtaining valuable health-enhancing resources—as work by Hayward et al. (2015), Masters et al. (2012), Sasson (2016), and others clearly suggests—then we would expect to see larger and potentially more discontinuous education effects for later cohorts of adults (e.g., baby boomers).

[17]Rates of smoking could also contribute to cross-cohort differences. The 1910–1920 cohort shared with its predecessors and immediate successors high rates of smoking initiation and continuation (Preston and Wang 2006), with little variation by education (Escobedo and Peddicord 1996). If anything, this should suppress education effects relative to later cohorts, where educational gradients in smoking were more pronounced (Ho and Fenelon 2015).

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

of the causal relationship between education and mortality, and the way it is conditioned by specific historical, social, demographic, and epidemiological factors.[18]

Caution is also warranted with respect to internal validity. Within-family designs, including within-twin pair and within-sibling designs, have a number of well-known methodological issues (McGue, Osler and Christensen 2010). The most important one for us has to do with identification. The within-pair estimator we used allowed us to difference out the influence of unobserved factors operating at the family and neighborhood levels, but there is no guarantee that residual within-pair differences (in specific environmental exposures, in early-life health conditions and illnesses, in personality characteristics, and/or in genetic endowments) did not remain. In our analyses, we did what we could to assess the severity of this threat via targeted simulations. Results from this exercise suggest that the amount of residual variation would have to be extensive, and of a certain type, in order to invalidate our inferences regarding the effects of education on mortality. Although this does not *confirm* that education is uncorrelated with the individual-level error term in our main estimating equation (and thus unconfounded by residual differences that exist within pairs of twins and non-twin siblings), it does help to provide a plausible lower bound on the effects we are estimating. We think this is about the best one can do using observational data.

Just over 45 years ago Kitagawa and Hauser (1973) published results from a large-scale record linking project that matched a sample of death records to microdata from the 1960 census long form. In their analyses, they found that education and life expectancy were positively correlated and that this association existed, to varying degrees, within different subgroups of the population. In the years since, there has been a push to extend Kitagawa and Hauser's findings in ways that allow for stronger statements regarding causality (Montez and Friedman 2015). We believe this line of inquiry is crucially important. If education and mortality are causally related to one another, then intervening in a way that promotes schooling could have tangible benefits for survival and other health-related outcomes (Hummer and Hernandez 2013). Our own analyses—which were also based on a large-scale record linking project—provide at least some reason for optimism in this regard. Men in the U.S. who were born between 1910 and 1920 tended to live longer if they completed additional schooling, and this pattern was not readily explained by differences in environmental exposures during childhood or variation in other hard to observe endowments. Although we prefer to be as circumspect as possible when making causal inferences, we think these results are at the very least consistent with the notion of a true education effect.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

[18]Another possible extension would be to link to the National Death Index (NDI), which provides information on cause of death. Based on the conceptual model presented in Figure 1, we would expect to see a more robust relationship between education and deaths that were caused by chronic diseases linked to unhealthy lifestyles (Masters, Link and Phelan 2015; Phelan et al. 2004), as opposed to deaths from less preventable causes where education (and the various personal and social resources it affords) should be of less benefit.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Acknowledgments

* This research was supported by a grant (1R21AG054824-01A1) from the Eunice Kennedy Shriver National Institute for Child Health and Human Development (NICHD). Research support was also provided by the Minnesota Population Center, which receives core funding (P2CHD041023) from NICHD. Thanks are due to participants at several conferences and seminars for their constructive feedback and comments. All errors and omissions, however, are the responsibility of the authors.

REFERENCES

Amin V 2011 “Returns to Education: Evidence from UK Twins: Comment.” American Economic Review 101(4):1629–1635.

Amin V, Behrman JR, & Kohler H-P 2015a “Schooling has smaller or insignificant effects on adult health in the US than suggested by cross-sectional associations: New estimates using relatively large samples of identical twins.” Social Science & Medicine 127:181–189. [PubMed: 25110343]

Amin V, Behrman JR, Kohler H-P, Xiong Y, & Zhang J 2015b “Causal Inferences: Identical Twins Help and Clarity About Necessary Assumptions is Critical.” Social Science & Medicine 127:201–202. [PubMed: 25533143]

Andersson MA 2016 “Health returns to education by family socioeconomic origins, 1980–2008: Testing the importance of gender, cohort, and age.” SSM - Population Health 2:549–560. [PubMed: 29349171]

Andersson MA, & Vaughan K 2017 “Adult health returns to education by key childhood social and economic indicators: Results from representative European data.” SSM - Population Health 3:411–418. [PubMed: 29349234]

Ashenfelter O, & Krueger A 1994 “Estiamtes of the Economic Return to Schooling from a New Sample of Twins.” The American Economic Review 84(5):1157–1173.

Baker DP, Leon J, Smith Greenaway EG, Collins J, & Movit M 2011 “The Education Effect on Population Health: A Reassessment.” Population Development Review 37(2):307–332. [PubMed: 21984851]

Bauldry S 2014 “Conditional health-related benefits of higher education: An assessment of compensatory versus accumulative mechanisms.” Social Science & Medicine 111:94–100. [PubMed: 24768780]

Beach B, Ferrie J, Saavedra M, & Troesken W 2016 “Typhoid Fever, Water Quality, and Human Capital Formation.” The Journal of Economic History 76(1):41–75.

Behrman JR, Kohler H-P, Jensen VM, Pedersen D, Petersen I, Bingley P, & Christensen K 2011 “Does More Schooling Reduce Hospitalization and Delay Mortality? New Evidence Based on Danish Twins.” Demography 48(4):1347–1375. [PubMed: 21842327]

Bell FC, & Miller ML 2005 Life tables for the United States social security area, 1900–2100. Washington, D.C.: Social Security Administration, Office of the Chief Actuary.

Black DA, Hsu Y-C, & Taylor LJ 2015 “The effect of early-life education on later-life mortality.” Journal of Health Economics 44:1–9. [PubMed: 26340596]

Boardman JD, & Fletcher JM 2015 “To cause or not to cause? That is the question, but identical twins might not have all of the answers.” Social Science & Medicine 127:198–200. [PubMed: 25455476]

Bound J, & Solon G 1999 “Double trouble: on the value of twins-based estimation of the return to schooling.” Economics of Education Review 18(2):169–182.

Braakmann N 2011 “The causal relationship between education, health and health related behaviour: Evidence from a natural experiment in England.” Journal of Health Economics 30(4):753–763. [PubMed: 21715033]

Chicco D 2017 “Ten quick tips for machine learning in computational biology.” BioData Mining 10(1):35. [PubMed: 29234465]

Clark D, & Royer H 2013 “The Effect of Education on Adult Mortality and Health: Evidence from Britain “ American Economic Review 103(6):2087–2120.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Cockerham WC 2005 “Health Lifestyle Theory and the Convergence of Agency and Structure.” Journal of Health and Social Behavior 46(1):51–67. [PubMed: 15869120]

Conley D, Strully KW, & Bennett NG 2006 “Twin differences in birth weight: The effects of genotype and prenatal environment on neonatal and post-neonatal mortality.” Economics & Human Biology 4(2):151–183. [PubMed: 16439189]

Conti G, & Heckman JJ 2010 “Understanding the Early Origins of the Education–Health Gradient:A Framework That Can Also Be Applied to Analyze Gene–Environment Interactions.” 5(5):585–605.

Cutler DM, Huang W, & Lleras-Muney A 2015 “When does education matter? The protective effect of education for cohorts graduating in bad times.” Social Science & Medicine 127:63–73. [PubMed: 25113567]

Denney JT, Rogers RG, Hummer RA, & Pampel FC 2010 “Education inequality in mortality: The age and gender specific mediating effects of cigarette smoking.” Social Science Research 39(4):662–673. [PubMed: 20563305]

Elo IT, & Preston SH 1996 “Educational differentials in mortality: United States, 1979–1985.” Social Science & Medicine 42(1):47–57. [PubMed: 8745107]

Ericsson M, Pedersen NL, Johansson ALV, Fors S, & Dahl Aslan AK 2019 “Life-course socioeconomic differences and social mobility in preventable and non-preventable mortality: a study of Swedish twins.” Int J Epidemiol 48(5):1701–1709. [PubMed: 30929008]

Escobedo LG, & Peddicord JP 1996 “Smoking prevalence in US birth cohorts: the influence of gender and education.” American Journal of Public Health 86(2):231–236. [PubMed: 8633741]

Falconer DS 1960 Introduction to Quantitative Genetics. New York: Ronald Press Co.

Feigenbaum JJ 2016 “Automated Census Record Linking: A Machine Learning Approach” Working paper, Boston University.

Ferrie JP 1996 “A New Sample of Males Linked from the Public Use Microdata Sample of the 1850 U.S. Federal Census of Population to the 1860 U.S. Federal Census Manuscript Schedules.” Historical Methods 29(4):141–156.

Fischer M, Karlsson M, & Nilsson T 2013 “Effects of Compulsory Schooling on Mortality: Evidence from Sweden.” International Journal of Environmental Research and Public Health 10(8):3596–3618. [PubMed: 23945539]

Fletcher JM 2015 “New evidence of the effects of education on health in the US: Compulsory schooling laws revisited.” Social Science & Medicine 127:101–107. [PubMed: 25311783]

Fuchs VR 1982 “Time Preference and Health: An Exploratory Study” Pp. 93–120 in Economic Aspects of Health, edited by Fuchs VR. Chicago: University of Chicago Press.

Galama TJ, Lleras-Muney A, & van Kippersluis H 2018 “The Effect of Education on Health and Mortality: A Review of Experimental and Quasi-Experimental Evidence.” National Bureau of Economic Research Working Paper Series No. 24225.

Gathmann C, Jürges H, & Reinhold S 2015 “Compulsory schooling reforms, education and mortality in twentieth century Europe.” Social Science & Medicine 127:74–82. [PubMed: 24560098]

Gilman SE, & Loucks EB 2014 “Another casualty of sibling fixed-effects analysis of education and health: An informative null, or null information?” Social Science & Medicine 118:191–193. [PubMed: 25138687]

Goldin C 1998 “America’s Graduation from High School: The Evolution and Spread of Secondary Schooling in the Twentieth Century.” The Journal of Economic History 58(2):345–374.

Gottfredson LS, & Deary IJ 2004 “Intelligence Predicts Health and Longevity, but Why?” Current Directions in Psychological Science 13(1):1–4.

Griliches Z 1979 “Sibling Models and Data in Economics: Beginnings of a Survey.” Journal of Political Economy 87(5):S37–S64.

Hamlett GW 1935 “Human Twinning in the United States: Racial Frequencies, Sex Ratios, and Geographical Variations.” Genetics 20(3):250–258. [PubMed: 17246757]

Hauser RM, & Warren JR 1997 “Socioeconomic Indexes for Occupations: A Review, Update, and Critique.” Sociological Methodology 27(1):177–298.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Hayward MD, Hummer RA, & Sasson I 2015 “Trends and group differences in the association between educational attainment and U.S. adult mortality: Implications for understanding education’s causal influence.” Social Science & Medicine 127:8–18. [PubMed: 25440841]

Ho JY, & Fenelon A 2015 “The Contribution of Smoking to Educational Gradients in U.S. Life Expectancy.” 56(3):307–322.

Hummer RA, & Hernandez EM 2013 “The Effect of Educational Attainment on Adult Mortality in the United States.” Population Bulletin 68(1):1–16. [PubMed: 25995521]

Hummer RA, & Lariscy JT 2011 “Educational Attainment and Adult Mortality” Pp. 241–261 in International Handbook of Adult Mortality, edited by Rogers RG and Crimmins EM: Springer Netherlands.

Kaufman JS, & Glymour MM 2011 “Splitting the Differences: Problems in Using Twin Controls to Study the Effects of BMI on Mortality.” Epidemiology 22(1):104–106. [PubMed: 21150356]

Kitagawa EM, & Hauser PM 1968 “Education Differentials in Mortality by Cause of Death: United States, 1960.” Demography 5(1):318–353.

—. 1973 Differential Mortality in the United STates: A Study in Socioeconomic Epidemiology. Cambridge: Harvard University Press.

Kohler H-P, Behrman JR, & Schnittker J 2011 “Social Science Methods for Twins Data: Integrating Causality, Endowments, and Heritability.” Biodemography and Social Biology 57(1):88–141. [PubMed: 21845929]

Kunst AE, & Mackenbach JP 1994 “The size of mortality differences associated with educational level in nine industrialized countries.” American Journal of Public Health 84(6):932–937. [PubMed: 8203689]

Lager ACJ, & Torssander J 2012 “Causal effect of education on mortality in a quasi-experiment on 1.2 million Swedes.” Proceedings of the National Academy of Sciences 109(22):8461.

Link BG, & Phelan J 1995 “Social Conditions As Fundamental Causes of Disease.” Journal of Health and Social Behavior:80–94. [PubMed: 7560851]

Link BG, Phelan JC, Miech R, & Westin EL 2008 “The Resources That Matter: Fundamental Social Causes of Health Disparities and the Challenge of Intelligence.” Journal of Health and Social Behavior 49(1):72–91. [PubMed: 18418986]

Lleras-Muney A 2005 “The Relationship Between Education and Adult Mortality in the United States.” The Review of Economic Studies 72(1):189–221.

Lundborg P, Lyttkens CH, & Nystedt P 2016 “The Effect of Schooling on Mortality: New Evidence From 50,000 Swedish Twins.” Demography 53(4):1135–1168. [PubMed: 27393233]

Madsen M, Andersen A-MN, Christensen K, Andersen PK, & Osler M 2010 “Does Educational Status Impact Adult Mortality in Denmark? A Twin Approach.” American Journal of Epidemiology 172(2):225–234. [PubMed: 20530466]

Masters RK, Hummer RA, & Powers DA 2012 “Educational Differences in U.S. Adult Mortality: A Cohort Perspective.” American sociological review 77(4):548–572. [PubMed: 25346542]

Masters RK, Link BG, & Phelan JC 2015 “Trends in education gradients of ‘preventable’ mortality: A test of fundamental cause theory.” Social Science & Medicine 127:19–28. [PubMed: 25556675]

Mazumder B 2008 “Does Education Improve Health? A Reexamination of the Evidence from Compulsory Schooling Laws.” Economic Perspectives 32(2):2–16.

McGue M, Osler M, & Christensen K 2010 “Causal Inference and Observational Research:The Utility of Twins.” Perspectives on Psychological Science 5(5):546–556. [PubMed: 21593989]

Meghir C, & Palme M 2005 “Educational Reform, Ability, and Family Background.” American Economic Review 95(1):414–424.

Meghir C, Palme M, & Simeonova E 2018 “Education and Mortality: Evidence from a Social Experiment “ American Economic Journal: Applied Economics 10(2):234–256.

Mirowsky J, & Ross C 2003 Education, Social Status, and Health. New York: Aldine de Gruyter.

Mirowsky J, & Ross CE 1998 “Education, Personal Control, Lifestyle and Health: A Human Capital Hypothesis.” Research on Aging 20(4):415–449.

Montez JK, & Friedman EM 2015 “Educational attainment and adult health: Under what conditions is the association causal?” Social Science & Medicine 127:1–7. [PubMed: 25557617]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Montez JK, & Hayward MD 2011 “Early Life Conditions and Later Life Mortality” Pp. 187–206 in International Handbook of Adult Mortality, edited by Rogers RG and Crimmins EM: Springer Netherlands.

Montez JK, & Hayward MD 2014 “Cumulative Childhood Adversity, Educational Attainment, and Active Life Expectancy Among U.S. Adults.” Demography 51(2):413–435. [PubMed: 24281740]

Montez JK, Hummer RA, & Hayward MD 2012 “Educational Attainment and Adult Mortality in the United States: A Systematic Analysis of Functional Form.” Demography 49(1):315–336. [PubMed: 22246797]

Montez JK, Hummer RA, Hayward MD, Woo H, & Rogers RG 2011 “Trends in the Educational Gradient of U.S. Adult Mortality From 1986 Through 2006 by Race, Gender, and Age Group.” Research on Aging 33(2):145–171. [PubMed: 21897495]

Montez JK, Zajacova A, Hayward MD, Woolf SH, Chapman D, & Beckfield J 2019 “Educational Disparities in Adult Mortality Across U.S. States: How Do They Differ, and Have They Changed Since the Mid-1980s?” Demography 56(2):621–644. [PubMed: 30607779]

Osler M, McGue M, & Christensen K 2007 “Socioeconomic position and twins’ health: a life-course analysis of 1266 pairs of middle-aged Danish twins.” Int J Epidemiol 36(1):77–83. [PubMed: 17251245]

Phelan JC, Link BG, Diez-Roux A, Kawachi I, & Levin B 2004 ““Fundamental Causes” of Social Inequalities in Mortality: A Test of the Theory.” Journal of Health and Social Behavior 45(3):265–285. [PubMed: 15595507]

Phelan JC, Link BG, & Tehranifar P 2010 “Social Conditions as Fundamental Causes of Health Inequalities: Theory, Evidence, and Policy Implications.” Journal of Health and Social Behavior 51(1_suppl):S28–S40. [PubMed: 20943581]

Preston SH, & Taubman P 1994 “Socioeconomic Differences in Adult Mortality and Health Status” Pp. 279–318 in Demography of Aging, edited by Martin LG and Preston S.a.H.. Washington, D.C.: National Academy Press.

Preston SH, & Wang HJD 2006 “Sex mortality differences in The United States: The role of cohort smoking patterns.” 43(4):631–646.

Rogers RG, Hummer RA, & Everett BG 2013 “Educational differentials in US adult mortality: An examination of mediating factors.” Social Science Research 42(2):465–481. [PubMed: 23347488]

Rosenbaum PR 1995 Observational Studies. New York: Springer-Verlag.

Rosenbaum PR, & Rubin DB 1983 “Assessing Sensitivity to an Unobserved Binary Covariate in an Observational Study with Binary Outcome.” Journal of the Royal Statistical Society: Series B 45(2):212–218.

Ross CE, & Mirowsky J 1989 “Explaining the Social Patterns of Depression: Control and Problem Solving--or Support and Talking?” Journal of Health and Social Behavior 30(2):206–219. [PubMed: 2738367]

—. 2006 “Sex differences in the effect of education on depression: Resource multiplication or resource substitution?” Social Science & Medicine 63(5):1400–1413. [PubMed: 16644077]

—. 2011 “The interaction of personal and parental education on health.” Social Science & Medicine 72(4):591–599. [PubMed: 21227556]

Ross CE, & Wu C-L 1996 “Education, Age, and the Cumulative Advantage in Health.” Journal of Health and Social Behavior 37(1):104–120. [PubMed: 8820314]

Ruggles S, Flood S, Goeken R, Grover J, Meyer E, Pacas J, & Sobek M 2019 “IPUMS: USA: Version 9.0 [dataset].” Minneapolis, MN: IPUMS 10.18128/D010.V9.0.

Sasson I 2016 “Diverging Trends in Cause-Specific Mortality and Life Years Lost by Educational Attainment: Evidence from United States Vital Statistics Data, 1990–2010.” PLOS ONE 11(10):e0163412. [PubMed: 27701419]

Schafer MH, Wilkinson LR, & Ferraro KF 2013 “Childhood (Mis)fortune, Educational Attainment, and Adult Health: Contingent Benefits of a College Degree?” Social Forces 91(3):1007–1034.

Smith WC, Anderson E, Salinas D, Horvatek R, & Baker DP 2015 “A meta-analysis of education effects on chronic disease: The causal dynamics of the Population Education Transition Curve.” Social Science & Medicine 127:29–40. [PubMed: 25459208]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Søndergaard G, Mortensen LH, Nybo Andersen A-M, Andersen PK, Dalton SO, Madsen M, & Osler M 2012 "Does Shared Family Background Influence the Impact of Educational Differences on Early Mortality?" American Journal of Epidemiology 176(8):675–683. [PubMed: 23024135]

van den Berg G, Janys L, & Christensen K 2015 "The Effect of Education on Mortality." IZA Working Paper.

van Kippersluis H, O'Donnell O, & van Doorslaer E 2011 "Long-Run Returns to Education: Does Schooling Lead to an Extended Old Age?" The Journal of Human Resources 46(4):695–721.

Weinberg W 1901 "Beiträge zur Physiologie und Pathologie der Mehrlingsgeburten beim Menschen." Archiv für die gesamte Physiologie des Menschen und der Tiere 88(6):346–430.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

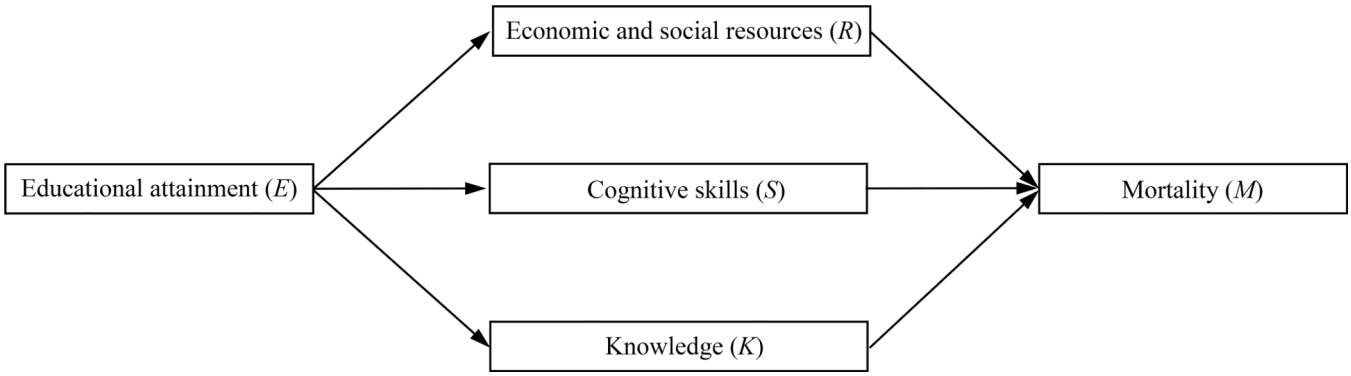

Figure 1.
Possible causal pathways between educational attainment and mortality

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

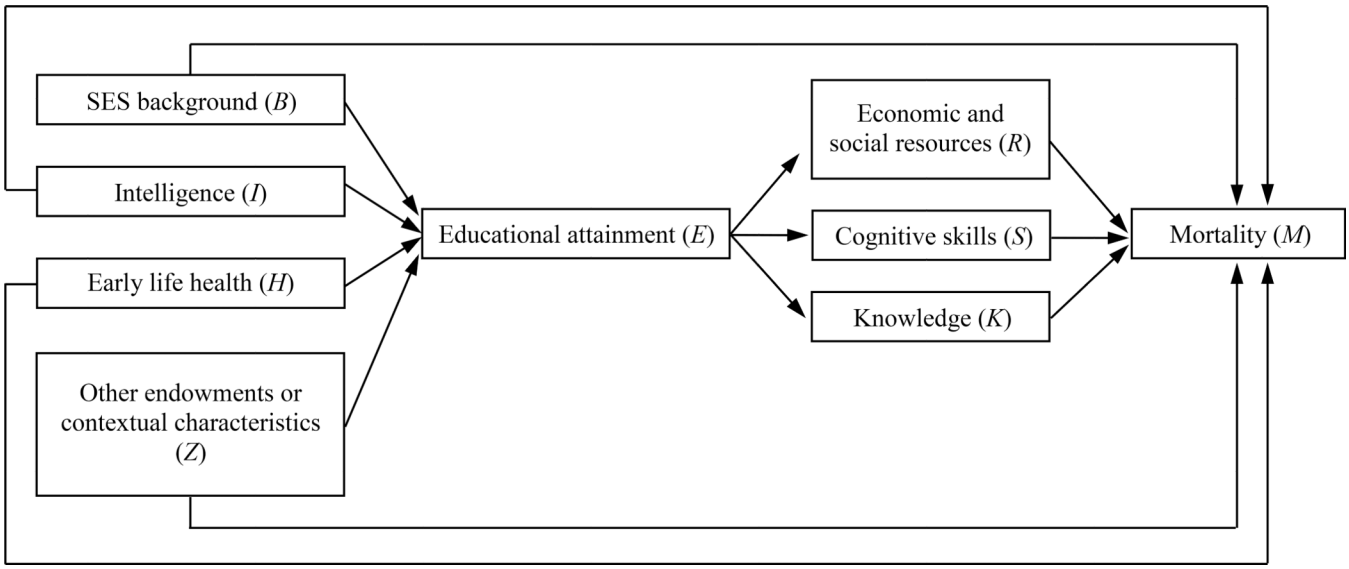

Figure 2.
Possible pathways between educational attainment and mortality, confounded by background characteristics

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

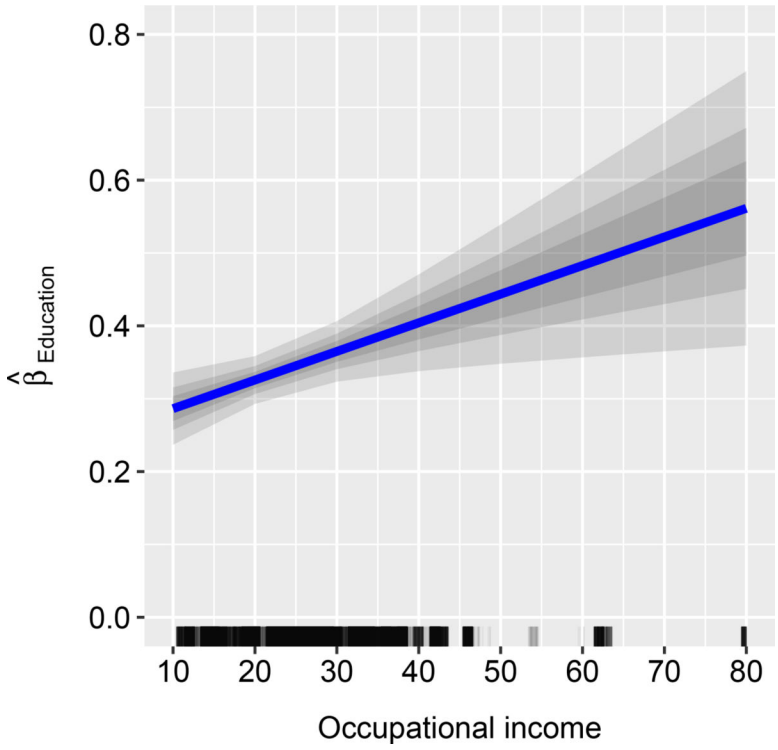

Figure 3.

Occupational income-by-education interaction. The solid blue line shows the estimated effect of an additional year of schooling on longevity, by SES during childhood. Higher values for occupational income indicate higher levels of advantage, and vice versa. Shading around the line, going from light to dark, provides the 95%, 75%, and 50% confidence intervals. The rug plot at the bottom of the graph gives the marginal distribution of occupational income, with each line corresponding to one family in our pooled non-twin sibling and twin data set. The lines in the rug plot have been perturbed slightly to reduce overlap. See text for more details.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Table 1.

Sample selection for twin sub-sample

	Step of sample selection				
	Original sample	**[1]**	**[2]**	**[3]**	**[4]**
N	139,195	136,598	21,962	5,376	5,216
White	88.09	88.15	95.90	97.81	97.78
Region					
New England	5.81	5.83	8.30	11.12	11.08
Mid-Atlantic	17.73	17.80	19.12	15.63	15.87
East North Central	18.35	18.37	23.18	26.86	27.30
West North Central	12.35	12.40	16.41	18.01	17.25
South Atlantic	15.80	15.75	10.75	8.41	8.44
East South Central	10.49	10.47	6.56	4.69	4.72
West South Central	12.45	12.40	7.89	6.81	6.83
Mountain	3.20	3.16	2.20	2.42	2.42
Pacific	3.81	3.82	5.59	6.06	6.10
Householder's occupation					
Blue collar	40.55	40.67	41.47	40.29	40.41
White collar	6.58	6.59	8.67	8.48	8.40
Farm (owner/tenant/manager	36.88	36.75	32.62	33.22	33.13
Not classified	15.99	15.99	17.24	18.01	18.06
Family size (mean)	7.21	7.18	6.91	6.92	6.92
US born father	75.82	75.81	75.61	74.98	74.92
US born mother	77.85	77.83	78.65	79.06	78.93
Age in 1920 (mean)	4.68	4.68	4.19	3.96	3.94

Note: The steps of sample selection were [1] drop cases in higher-order births; [2] drop cases where both members of the pair were not observed in 1940; [3] drop cases where both members in pair were not also observed in the SSDMF; and [4] drop pairs where one or more member is missing information on educational attainment, as observed in 1940. In this table, and throughout, *N* refers to the total number of cases, not the total number of pairs.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table 2.

Descriptive statistics, by subsample

	Random	Neighbors	Siblings	Twins
Education				
Mean	10.40	10.40	10.13	10.36
SD	2.91	2.91	2.75	2.78
Percent discordant	84.23	79.87	62.62	40.38
Correlation within pair	0.00	0.23	0.64	0.78
Absolute mean difference within pair	3.20	2.72	1.55	0.94
SD of absolute difference	2.59	2.37	1.77	1.57
Age at death				
Mean	74.77	75.63	75.73	75.96
SD	10.96	10.96	11.02	10.56
Percent discordant	99.99	99.99	99.98	99.73
Correlation within pair	0.00	0.01	0.12	0.21
Absolute mean difference within pair	12.29	12.22	11.49	10.25
SD of absolute difference	9.43	9.41	9.03	8.39
N	1,658,836	1,604,936	328,352	5,216

Note: The subsamples are restricted to pairs where there is full information on education and age at death, as obtained from the 1940 census and SSDMF, respectively.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Table 3.

Weighted models predicting age at death, by subsample

	Unpaired				Paired			
	Random	**Neighbors**	**Siblings**	**Twins**	**Random**	**Neighbors**	**Siblings**	**Twins**
Years of schooling	0.387 *** (0.004)	0.389 *** (0.004)	0.393 *** (0.009)	0.456 *** (0.067)	0.401 *** (0.005)	0.400 *** (0.005)	0.338 *** (0.017)	0.347 ** (0.159)
White	0.142 *** (0.051)	0.125 ** (0.052)	−0.119 (0.144)	0.486 (1.080)	0.014 (0.079)	−0.224 ** (0.090)		
Householder's occupation								
White collar	0.364 *** (0.035)	0.357 *** (0.036)	0.513 *** (0.088)	0.950 (0.648)	0.356 *** (0.049)	0.295 *** (0.052)		
Farmers (owner/tenant/ manager)	1.375 *** (0.025)	1.372 *** (0.025)	1.606 *** (0.057)	2.178 *** (0.452)	1.411 *** (0.033)	0.987 *** (0.049)		
Not classified	0.080 *** (0.026)	0.096 *** (0.027)	0.071 (0.064)	−0.271 (0.476)	0.099 *** (0.036)	0.056 (0.039)		
Family size	0.037 *** (0.005)	0.039 *** (0.005)	−0.008 (0.011)	0.087 (0.081)	0.034 *** (0.006)	0.039 *** (0.007)		
US born father	−0.441 *** (0.030)	−0.448 *** (0.030)	−0.360 *** (0.067)	−1.932 *** (0.502)	−0.403 *** (0.043)	−0.412 *** (0.044)		
US born mother	−0.347 *** (0.031)	−0.342 *** (0.032)	−0.374 *** (0.071)	0.574 (0.532)	−0.336 *** (0.045)	−0.414 *** (0.046)		
Age in 1920	0.170 *** (0.003)	0.168 *** (0.003)	0.188 *** (0.007)	0.268 *** (0.057)	0.161 *** (0.004)	0.165 *** (0.004)	0.191 *** (0.010)	
Constant	70.166 *** (0.106)	70.188 *** (0.109)	70.557 *** (0.277)	70.040 *** (2.329)	70.070 *** (0.144)	70.962 *** (0.113)	71.346 *** (0.179)	72.240 *** (1.620)

Note: Inverse probability of successful linkage weights were generated in order to adjust for non-random losses to the sample during the data linkage stage. To generate the weights, we modeled the likelihood of a successful outcome (linkage between 1920, 1940, and the SSDMF) using race, householder's occupational category in 1920, region of residence in 1920, and family size in 1920 as predictors. We then calculated predicted probabilities and took the inverse to generate weights. All unpaired models cluster standard errors at the pair level. All unpaired models also included state dummies, as does our model for random pairs. The reference category for white is non-white. The reference category for householder's occupation is blue collar. Sample sizes are as follows: random (n = 1,658,836), unrelated neighbors (n = 1,604,936), non-twin siblings (n = 328,352), twins (n = 5,216).

*** $p < .01$

** $p < .05$

* $p < .10$

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Table 4.

Weighted models predicting age at death using a categorical measure of education, by subsample

	Unpaired				Paired			
	Random	**Neighbors**	**Siblings**	**Twins**	**Random**	**Neighbors**	**Siblings**	**Twins**
12+ years of schooling	2.251 *** (0.020)	2.262 *** (0.020)	2.212 *** (0.045)	2.506 *** (0.353)	2.220 *** (0.027)	2.205 *** (0.029)	1.569 *** (0.081)	1.568 * (0.802)
White	0.539 *** (0.050)	0.525 *** (0.052)	0.238 * (0.143)	0.832 (1.101)	0.446 *** (0.079)	0.257 *** (0.090)		
Householder's occupation								
White collar	0.509 *** (0.035)	0.501 *** (0.035)	0.643 *** (0.087)	1.232 * (0.645)	0.536 *** (0.049)	0.443 *** (0.051)		
Farmers (owner/tenant/ manager)	1.267 *** (0.025)	1.264 *** (0.025)	1.503 *** (0.056)	2.071 *** (0.452)	1.307 *** (0.033)	0.995 *** (0.049)		
Not classified	0.121 *** (0.026)	0.137 *** (0.027)	0.113 * (0.064)	−0.193 (0.476)	0.152 *** (0.036)	0.090 ** (0.039)		
Family size	0.026 *** (0.005)	0.028 *** (0.005)	−0.016 (0.011)	0.081 (0.081)	0.021 *** (0.006)	0.028 *** (0.007)		
US born father	−0.453 *** (0.030)	−0.459 *** (0.030)	−0.372 *** (0.067)	−1.963 *** (0.503)	−0.415 *** (0.043)	−0.410 *** (0.044)		
US born mother	−0.353 *** (0.031)	−0.348 *** (0.032)	−0.380 *** (0.071)	0.547 (0.532)	−0.339 *** (0.045)	−0.404 *** (0.046)		
Age in 1920	0.180 *** (0.003)	0.178 *** (0.003)	0.196 *** (0.007)	0.285 *** (0.057)	0.172 *** (0.004)	0.175 *** (0.004)	0.195 *** (0.010)	
Constant	72.629 *** (0.101)	72.660 *** (0.104)	73.126 *** (0.265)	73.311 *** (2.312)	72.662 *** (0.138)	73.622 *** (0.103)	74.071 *** (0.060)	75.072 *** (0.383)

Note: Inverse probability of successful linkage weights were generated in order to adjust for non-random losses to the sample during the data linkage stage. To generate the weights, we modeled the likelihood of a successful outcome (linkage between 1920, 1940, and the SSDMF) using race, householder's occupational category in 1920, region of residence in 1920, and family size in 1920 as predictors. We then calculated predicted probabilities and took the inverse to generate weights. All unpaired models cluster standard errors at the pair level. All unpaired models also included state dummies, as does our model for random pairs. The reference categories are less than 12 years of schooling (educational attainment), non-white (race), blue collar (householder's occupation). Sample sizes are as follows: random (n = 1,658,836), unrelated neighbors (n = 1,604,936), non-twin siblings (n = 328,352), twins (n = 5,216).

*** $p < .01$

** $p < .05$

* $p < .10$

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table 5.

Sensitivity of weighted within-twin pair estimates to the presence of residual variation

	$r_{z,y}$						
$r_{z,x}$	**−0.3**	**−0.2**	**−0.1**	**0.0**	**0.1**	**0.2**	**0.3**
−0.3	0.01	0.15	0.27	0.35	0.50	0.63	0.78
−0.2	0.14	0.22	0.29	0.35	0.43	0.51	0.59
−0.1	0.25	0.28	0.32	0.35	0.38	0.42	0.46
0.0	0.32	0.33	0.34	0.35	0.34	0.34	0.32
0.1	0.46	0.42	0.38	0.35	0.32	0.29	0.25
0.2	0.60	0.51	0.43	0.35	0.29	0.22	0.14
0.3	0.77	0.62	0.50	0.35	0.27	0.14	0.01

Note: Alternative weighted within-twin pair estimates for years of schooling were obtained via simulation. In the simulation, we randomly generated an unobserved variable, *z*, with a pre-specified correlation to years of schooling, *x*, and age at death, *y*. We then included this variable in our within-pair model, collected the resulting point estimate for years of schooling, and then averaged across 1,000 replications to obtain stable results. For the sake of reference, the correlation between the householder's socioeconomic status (measured in terms of SEI) and children's years of schooling is 0.3, and its correlation with age at death is 0.01.