Does education improve health? A reexamination of the evidence from compulsory schooling laws

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Introduction and summary

Improving the long-term health of the population is clearly an important goal for policymakers. It is also likely to become even more so in the coming years with the aging of the baby boomers and the anticipated health-related costs that will accompany this demographic change. Therefore, understanding which policy levers might improve health is of interest. In a provocatively titled front page article, "A surprising secret to a long life: Stay in school," the *New York Times* recently suggested that many researchers now believe that education is the key factor in promoting health.[1] While social scientists have long known that there is a strong positive *correlation* between education and longevity, many researchers have speculated that this association was not truly *causal*, meaning one didn't necessarily lead to the other. Rather, the link was thought to reflect either the fact that for a variety of other reasons (for example, parental income and personal attitudes), people who tend to acquire more schooling also tend to be in better health, or that healthier children stayed in school longer. Of course, in the absence of evidence of a causal link, there is no reason to expect that policies aimed at increasing educational attainment will result in improvements in health.

The *New York Times* article was based upon the results of a recent study by economist Adriana Lleras-Muney (2005) that provides perhaps the strongest evidence to date that education has a causal effect on health. By implementing an instrumental variables (IV) strategy, this research analyzes changes in compulsory schooling and child labor laws across different states early in the twentieth century and uses this information to infer the effects of education on mortality. The idea behind this strategy is that if differences in these laws induced people born in different states in different years to obtain different levels of schooling for reasons that are unrelated to any other

determinants of health, then one can estimate a true causal effect that is not confounded by the other factors. Lleras-Muney finds that increased schooling due to these laws led to dramatic reductions in mortality rates during the 1960s and 1970s. In fact, the results imply that one more year of schooling would lower the mortality rate over a ten-year period by nearly 60 percent—a result that is perhaps implausibly large.

If it is true that more education leads to improved health, such a finding also raises a second important question—namely, *how*, exactly, does education affect health? Economists have proposed a variety of theories including: that more education leads to better jobs and more financial resources; that education improves knowledge and decision-making ability, which improves health; and that education influences other kinds of behavioral responses that, in turn, lead to better health outcomes. So far, however, there is little convincing empirical evidence on how to evaluate the importance of these factors.

In this article, I reexamine the use of these compulsory schooling laws as a way of identifying the causal effects of education on health through the IV approach. Given the fundamental importance of the question of whether more education is causally linked to better health, it is worth investigating the robustness of the relationship. I estimate the same types of models used in the earlier research, using a much larger sample and improved measures of compulsory schooling laws. I also present alternative specifications of the statistical model that may better account for other

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reforms that were going on during the same period. For example, during the early period of the twentieth century, there were fairly dramatic improvements in public health measures that led to large declines in concurrent mortality (Cutler and Miller, 2005). For school-age children specifically, new nutrition and vaccination programs may have resulted in improved long-term health, independent of any effects of increased education.

In addition, if compulsory schooling laws can be used to identify a causal relationship, then they also ought to be useful in identifying *how* education improves health. This can be analyzed by using data on very specific health conditions for which existing theories might favor one explanation versus another. For example, if processing information and decision-making ability are the critical channels by which education affects health, then we might expect lower incidences of chronic diseases, such as arthritis, cancer, diabetes, lung disease, and heart disease. These are conditions that might respond better to more sophisticated management plans or behavioral changes. If the key factor is increased access to high-quality health care due to greater financial resources, then we might expect that a broad range of health outcomes would be improved. Therefore, it makes sense to apply the same methodology to other outcomes besides mortality.

A careful analysis of how education affects health using the IV approach also serves as a credibility check on the methodology. If, for example, all of the health effects appeared to be related to the long-term effects of poor nutrition, then a plausible alternative hypothesis would be that changes in compulsory schooling laws are really just picking up the long-term health effects of improved nutrition in schools. In that case, the assumption that these laws represent exogenous sources of schooling differences would be invalid, and the estimates would not represent a causal relationship between education and health.

In order to address these issues, I first reexamine the effects of education on mortality from Lleras-Muney (2005, 2006) by replicating the results and extending them by adding significantly more data and employing a variety of robustness checks. I find that the effect of education on mortality is not robust to the inclusion of state-specific time trends, casting doubt on whether there is a true causal effect. At a minimum, my results show that the point estimates are much smaller than those previously found in the literature. Moreover, the results appear to be driven by the earliest cohorts (born in 1901–12) during the 1960–70 period.

Second, I use individual-level data on health outcomes from the U.S. Census Bureau's *Survey of Income and Program Participation* (SIPP) to further investigate the causal pathways between compulsory schooling and health. In contrast to the U.S. Census data, which requires the use of a cohort grouping strategy to infer mortality, the SIPP provides data on the health status of each individual so that we can be sure that those who were affected by the compulsory schooling laws are indeed the same individuals registering the change in health. Using the SIPP with the same IV strategy, I find large and statistically significant effects of education on general health status that are robust to the inclusion of state-specific time trends. This suggests that the SIPP micro data are able to overcome the limitations of the U.S. Census data.

However, when I turn to the results that identify which specific health conditions were affected by education improvements induced by compulsory schooling laws, the results do not point to a coherent story of how education affects health. For example, only a small fraction of health conditions are affected by education, and several of those affected are conditions, such as sight and hearing, where economic theories don't appear to be relevant. What is also striking is the absence of effects among many chronic diseases where decision-making ability is believed pivotal. A limitation of the data, however, is that specific conditions are only identified for a subset of the sample that report having some health limitations. Nevertheless, this pattern of results suggests that the use of compulsory schooling laws as an instrument may be suspect. I also note that in a recent working paper, Clark and Royer (2007) use an even more sophisticated approach to analyze the effects of compulsory schooling law changes in the United Kingdom on mortality. Their findings also cast doubt on whether there is a strong causal connection between education and health.

Background and previous literature

Kitagawa and Hauser (1973) were the first to document the sharp differences in health in the United States by socioeconomic status. A large number of studies have since replicated this basic finding of a "gradient" in health by education or income, and this pattern has also been found in other countries.[2] For policymakers, a critical question is whether this gradient reflects a causal relationship that can be exploited to improve the long-term health of the population. For example, in a document soliciting research proposals on the pathways linking education to health, the National Institutes of Health (2003) cautioned that: "The association or pathway between formal education and either important health behaviors or diseases may not be causal. Instead it may reflect the

influence of confounding or co-existing determinants or may be bi-directional.''

A review of the literature on whether the education gradient in health is causal may be found in Grossman (2005). While these studies typically find an effect of more education leading to better health, in most cases it is questionable whether the instruments are truly exogenous. For example, Dhir and Leigh (1997) use parent schooling, parent income, and state of residence as instruments, all of which could plausibly affect long-term health independently of their effects through schooling. The innovation by Lleras-Muney (2005) to use changes in compulsory schooling laws early in the twentieth century appears to be more compelling, since it is more plausibly exogenous than instruments used in prior work. Nevertheless, other changes in public policy that coincided with changes in compulsory schooling laws might have led to long-run improvements in health. Cutler and Miller (2005) find that the introduction of clean water technologies during this period could explain as much as half of the concurrent decline in mortality. Similarly, many states introduced food programs in schools, recognizing that compulsory schooling was pointless if children were malnourished. Near the beginning of the twentieth century, Robert Hunter (1904) wrote in the book *Poverty*: "There must be thousands—very likely sixty or seventy thousand children—in New York City alone who often arrive at school hungry and unfitted to do well the work required. It is utter folly, from the point of view of learning, to have a compulsory school law which compels children, in that weak physical and mental state which results from poverty, to drag themselves to school and to sit at their desks, day in and day out, for several years, learning little or nothing.'' In response to this situation, Philadelphia, Boston, Milwaukee, New York, Cleveland, Cincinnati, and St. Louis all began large-scale programs to provide food in public schools during the 1900s and 1910s (Gunderson, 1971). Mazumder (2007) also provides suggestive evidence that the mechanism by which compulsory schooling laws might have improved long-term health was through school requirements for vaccination against smallpox. If improvements in nutrition and vaccination programs were coincident with changes in compulsory schooling laws, then these might explain some or all of the long-term health improvements that were associated with changes in these laws.

Supposing that it is true that more education leads to improved health, this finding raises an interesting question—namely, *how*, exactly, does education affect health? As Richard Suzman of the National Institute on Aging recently stated, "Education ... is a particularly powerful factor in both life expectancy and health expectancy, though truthfully, we're not quite sure why.''[3] Economists have proposed a variety of explanations. These theories typically emphasize the role of education in affecting various proximate determinants of health, including financial resources, knowledge and decision-making ability, and other behavioral characteristics that could lead to better health outcomes.

Financial resources come into play because better educated individuals may obtain higher paying and more stable jobs and thereby may be able to afford better quality health care and health insurance. With greater economic resources, they may also choose safer and more secure living and work environments. One might expect that if financial resources are the key factor behind the link between education and health, then we should expect to see virtually all forms of health conditions affected by exogenous sources of increased education.

The second explanation is that higher levels of schooling may lead to greater knowledge and an improved ability to process information and make better choices or take better advantage of technological improvements. In one widely cited paper, Goldman and Smith (2002) note that better educated patients may manage chronic conditions better. Those with more schooling adhere more closely to treatment regimens for human immunodeficiency virus (HIV) infection and diabetes, which can be fairly complex. For such conditions, the ability to form independent judgments and comprehend treatments is important, and apparently is fostered by schooling. Accordingly, Goldman and Smith (2002, p. 10934) state that "self-maintenance is an important reason for the very steep SES [socioeconomic status] gradient in health outcomes.'' Glied and Lleras-Muney (2003) argue that "the most educated make the best initial use of new information about different aspects of health,'' permitting them to respond more adeptly to evolving medical technologies.

Finally, it could be that education induces other kinds of behavioral changes. For example, the better educated may value the future more than the present compared with those with less education, and therefore, the better educated may take better care of their health (Becker and Mulligan, 1997). Others have argued that education improves one's perception of one's *relative* status in society and that improved social standing is associated with better health (Marmot, 1994).

Mortality analysis: Methodology and data

The first part of the analysis estimates the effects of education on mortality, using the approach developed by Lleras-Muney (2005). In the absence of a

large sample of data on individuals containing both education and lifespan, I use group-level data from successive U.S. *Decennial Censuses* to estimate mortality rates. Specifically, I use population estimates for groups defined by state of birth, gender, and year of birth to estimate the mortality rate across ten-year periods. The mortality rate at time t for birth cohort c of gender g born in state s, (M_{cgst}), is simply measured as the percentage decline in the population count (N_{cgst}) within these cells over the subsequent ten years:

$$1) \quad M_{cgst} = \frac{N_{cgst} - N_{cgst+1}}{N_{cgst}}.$$

I then model the mortality rate for each cell as follows:

$$2) \quad M_{cgst} = a + E_{cgst}\pi + W_{cs}\delta + \gamma_c + \alpha_s + \theta_{cr}$$
$$+ fem + \tau_t + \varepsilon_{cgst},$$

where E_{cgst} is the average education level for that cell at time t and W_{cs} measures a set of cohort and state-specific controls measured at age 14 intended to capture differences in other potential early life determinants of mortality (for example, manufacturing share of employment and doctors per capita). The model also includes a set of cohort dummies c, state of birth dummies s, interactions between cohort and region of birth θ_{cr}, a female dummy (*fem*), and year dummies τ_t.

One straightforward way to estimate π in equation 2 would be through weighted least squares (WLS), with the weights corresponding to the population represented by each cell. However, this would produce a biased estimate because of omitted variables. Any number of factors could plausibly be associated with both higher education and lower mortality even at the group level. Therefore, I use two-stage least squares, where in the first stage, education is instrumented with the set of compulsory schooling laws, CL_{cs}, in place for each cohort and state of birth:

$$3) \quad E_{cgst} = b + CL_{cs}\rho + X_{cgst}\beta + W_{cs}\delta + \gamma_c + \alpha_s$$
$$+ \theta_{cr} + fem + \tau_t + u_{cgst}.$$

In Lleras-Muney (2005), the instruments for the compulsory schooling laws were constructed in the following way. The variable *childcom* measured the minimum required age for work minus the maximum age before a child is required to enter school, by state of birth and by the year the cohort is age 14. This

variable takes on one of eight values. A set of indicator variables were then used as instruments. In addition, an indicator for whether school continuation laws were in place in that state was also used. These laws required workers of school age to continue school part time. However, it probably makes more sense to match individuals to the laws concerning the maximum age for school *entry* around the age at which students start school, rather than to the laws in place when they were age 14. Therefore, I use a different set of data independently collected by Goldin and Katz (2003).[4] Goldin and Katz carefully compared their series with other codings of the compulsory schooling laws (for example, Lleras-Muney, 2005; and Acemoglu and Angrist, 2001) and resolved differences wherever possible. Since the Goldin and Katz data go back further in time, it is possible to match all of the cohorts to the school entry age laws in effect when the cohorts were younger than 14. I use these data to measure the required age for school entry when the cohorts were at age 8 instead of 14. In principle, incorporating these data should provide a better measure of the total years of compulsory schooling.

Several estimation samples are constructed for this part of the analysis. Initially, I produce a sample combining data from the 1 percent Integrated Public Use Microdata Series (IPUMS) from the 1960, 1970, and 1980 U.S. Censuses in order to replicate the basic results in Lleras-Muney (2005, 2006).[5] I then expand the analysis in stages. First, I replace the 1 percent samples in 1970 and 1980 with a 2 percent sample for 1970 and a 5 percent sample for 1980. Second, I also expand the periods by adding 5 percent samples for 1990 and 2000. Following the literature, I restrict the analysis to cohorts born between 1901 and 1925, top-code years of education at 18 starting in 1980, and exclude immigrants and blacks.[6] For the expanded samples, I also exclude cases where age, state of birth, and education are imputed by the U.S. Census Bureau. The descriptive statistics for the replication sample and the expanded sample are shown in table 1.

It is worth noting that the death rate for the 1970–80 period is quite a bit larger with the expanded sample but that the standard deviation is about 20 percent lower. There are now also five additional cells that had missing data when using just the 1 percent samples. The death rates for the 1980–90 and 1990–2000 periods are much higher because I follow these same cohorts when they are much older. Figure 1 plots the death rates by age for each U.S. Census year. This highlights the importance of controlling for age in the specifications, which is done by adding polynomials in age to the models.

TABLE 1

Summary statistics for Integrated Public Use Microdata Series samples

Variables	1960 1%, 1970 1%, and 1980 1% samples			1960 1%, 1970 2%, 1980 5%, 1990 5%, and 2000 5% samples		
	Mean	Standard deviation	Number of observations	Mean	Standard deviation	Number of observations
Ten year death rates						
Overall	0.108	0.136	4,792	0.213	0.173	8,636
1960–70	0.110	0.119	2,395	0.113	0.105	2,397
1970–80	0.105	0.152	2,397	0.154	0.125	2,400
1980–90	—	—	—	0.287	0.170	2,399
1990–2000	—	—	—	0.433	0.122	1,440
Individual characteristics						
Education	10.548	0.990	4,795	10.729	1.002	8,636
1960 dummy	0.471	0.499	4,795	0.325	0.469	8,636
1970 dummy	—	—	—	0.289	0.453	8,636
1990 dummy	—	—	—	0.142	0.349	8,636
Female	0.517	0.500	4,795	0.532	0.499	8,636
Age	50.366	8.482	4,795	56.811	11.287	8,636
Born in 1905	0.031	0.174	4,795	0.025	0.157	8,636
Born in 1910	0.038	0.191	4,795	0.031	0.174	8,636
Born in 1915	0.044	0.205	4,795	0.047	0.211	8,636
Born in 1920	0.048	0.213	4,795	0.052	0.222	8,636
Born in 1925	0.050	0.217	4,795	0.057	0.232	8,636
State of birth characteristics						
Percentage urban	53.523	21.279	4,795	53.778	21.153	8,636
Percentage foreign-born	11.737	8.523	4,795	11.562	8.430	8,636
Percentage black	8.983	11.901	4,795	8.945	11.787	8,636
Percentage employed in manufacturing	0.067	0.038	4,795	0.066	0.037	8,636
Annual manufacturing wage *($)*	7,171.39	1,343.09	4,795	7,206.15	1,353.57	8,636
Value of farm per acre *($)*	540.05	276.35	4,795	535.18	272.57	8,636
Per capita number of doctors	0.001	0.000	4,795	0.001	0.000	8,636
Per capita education expenditures *($)*	97.01	42.05	4,795	99.78	41.71	8,636
Number of school buildings per square mile	0.174	0.090	4,795	0.172	0.090	8,636

Notes: Summary statistics are for state of birth, cohort, and gender cells. All means and standard deviations use sample weights where the weights are the population estimates for the cell in the base period.

Source: Author's calculations based on data from the University of Minnesota, Minnesota Population Center, Integrated Public Use Microdata Series.

Health analysis: Methodology and data

The methodological approach changes only slightly when I turn to using individual-level data from the SIPP. Many of the outcomes in the SIPP are indicator variables that take on the value of 1 if a particular health problem is present and 0 otherwise. Therefore, I now use two-stage conditional maximum likelihood, or 2SCML (Rivers and Vuong, 1988), rather than IV.[7] Rivers and Vuong show that 2SCML has desirable statistical properties, is easy to implement, and produces a simple test for exogeneity. I continue to use IV for the few continuous dependent variables. Also, all of the analysis is now done using individual-level data. The statistical model is similar to equation 2, only now I use the latent variable framework:
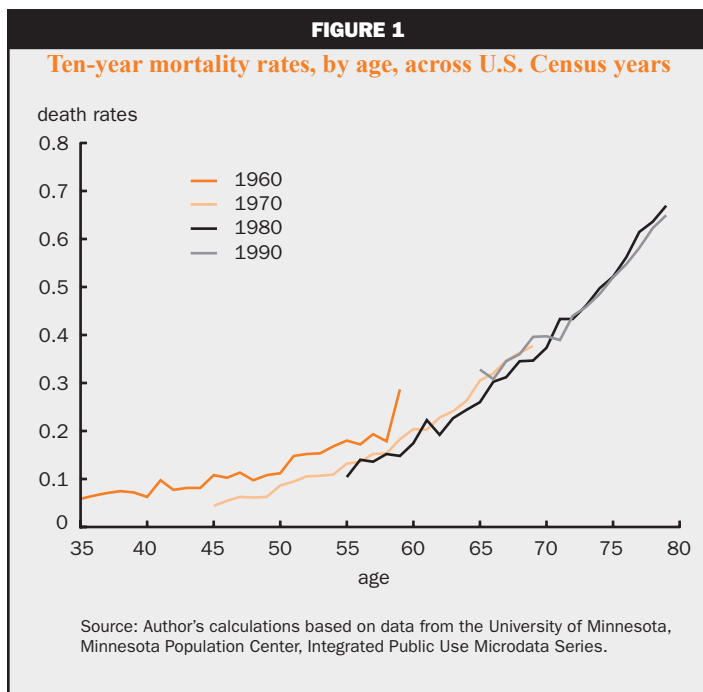
$$4) \quad y_{it}^* = a + E_i \pi + X_i \beta + W_{cs} \delta + \gamma_c + \alpha_s + trend_s + \tau_t + fem + \varepsilon_{it},$$

$$5) \quad y_{it} = 1 \text{ if } y_{it}^* > 0, \quad y_{it} = 0 \text{ if } y_{it}^* \leq 0.$$

In the first stage, I run a similar regression as before:

$$6) \quad E_i = b + CL_{cs} \rho + X_i \beta + W_{cs} \delta + \gamma_c + \alpha_s + trend_s + \tau_t + d + \varepsilon_{it}.$$

To implement 2SCML, I use the predicted residuals from equation 6, $\hat{\varepsilon}_{it}$, and I include it as an additional right-hand side variable (along with the actual value of E_i) when running the second stage probit. For comparability, I use the same sample restrictions and

FIGURE 1

Ten-year mortality rates, by age, across U.S. Census years

death rates

- 1960
- 1970
- 1980
- 1990

age

Source: Author's calculations based on data from the University of Minnesota, Minnesota Population Center, Integrated Public Use Microdata Series.

covariates as in the U.S. Census results, with only a few exceptions. I include a quadratic in age and use state-specific cohort trends to address concerns that region of birth interacted with cohort may not adequately control for state-specific factors that are smoothly changing over time.[8]

The sample is constructed by pooling individuals from the 1984, 1986–88, 1990–93, and 1996 SIPP panels. Each SIPP panel surveys approximately 20,000 to 40,000 households, and most panels are representative of the noninstitutionalized population.[9] Because participation in many programs is closely related to an individual's health and disability status, the SIPP routinely collects information on health and medical conditions. The SIPP is also ideally suited for this analysis because it contains the state of birth of all sample members, which allows me to implement the IV strategy of using compulsory schooling laws during childhood.

One especially useful outcome is self-reported health (SRH). The SRH is on a 1–5 scale, where 1 is "excellent," 2 is "very good," 3 is "good," 4 is "fair," and 5 is "poor." The SRH has been found to be an excellent predictor of mortality and changes in functional abilities among the elderly (Case, Lubotsky, and Paxson, 2002). I experiment with this measure in a few ways. First, I use it as a continuous variable. Second, I use indicators for being in poor health or in fair or poor health. Finally, I use the health utility scale that measures the differences between the categories in a health model using the *National Health Interview Survey*

(conducted by the U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics).[10]

I also examine some other general outcomes. These are whether the individual was hospitalized during the past year, the number of times she was hospitalized, the total number of nights spent in the hospital, and the number of days spent in bed in the past four months.

There are also questions dealing with functional activities, activities of daily living, and instrumental activities of daily living that are derived from the International Classification of Impairments, Disabilities, and Handicaps (ICIDH). I assembled a common set of questions that were consistently asked across surveys. These are whether the individual has "difficulty" with seeing, hearing, speaking, lifting, walking, and climbing stairs, as well as whether the person can perform any of these activities "at all." In addition, there is information on whether individuals have difficulty getting around inside the house, going outside of the house, or getting in or out of bed, as well as whether they need the assistance of others for these activities.

For a subset of individuals who report limited abilities in certain tasks or who have been classified as having a work disability ("health limitation"), detailed information is collected on a number of very specific health conditions including: arthritis or rheumatism; back or spine problems; blindness or vision problems; cancer; deafness or serious trouble hearing; diabetes; heart trouble; hernia; high blood pressure (hypertension); kidney stones or chronic kidney trouble; mental illness; missing limbs; lung problems; paralysis; senility/dementia/Alzheimer's disease; stiffness or deformity of limbs; stomach trouble; stroke; thyroid trouble or goiter; tumors (cyst or growth); or other.[11] Since the specific health ailments are only asked of specific subsamples, they probably only pick up on the most severe cases. Even though many of the sample individuals are not actually asked about these specific health conditions, I still include them in the estimation sample so that the sample is not a selected sample of only those in poor health. The summary statistics for these data are shown in table 2.

Mortality results

I begin by trying to match the estimates of the effect of education on ten-year mortality rates shown in

TABLE 2

Summary statistics for *Survey of Income and Program Participation* sample

Variables	Mean	Standard deviation	Number of observations
Outcomes			
Self-reported health (1 is excellent, 5 is poor)	3.084	1.138	26,030
Poor health	0.119	0.324	26,030
Fair or poor health	0.357	0.479	26,030
Health index (1–100 scale)	67.992	24.842	26,030
Hospitalized in last year	0.180	0.384	26,484
Days in bed, last four months	3.937	17.030	25,223
Number of times hospitalized	0.282	1.029	22,229
Number of nights in hospital	1.908	7.898	26,274
Trouble seeing	0.136	0.342	20,853
Trouble hearing	0.152	0.359	20,845
Trouble speaking	0.021	0.144	20,834
Trouble lifting	0.237	0.425	20,837
Trouble walking	0.289	0.453	20,799
Trouble with stairs	0.276	0.447	20,820
Trouble getting around outside the home	0.129	0.335	17,401
Trouble getting around inside the home	0.059	0.235	17,643
Trouble getting in/out of bed	0.079	0.270	17,636
Trouble seeing at all	0.023	0.149	20,811
Trouble hearing at all	0.013	0.114	20,819
Trouble speaking at all	0.003	0.052	15,138
Trouble lifting at all	0.115	0.319	20,789
Trouble walking at all	0.154	0.361	20,723
Trouble with stairs at all	0.116	0.321	20,775
Needs help getting around outside	0.088	0.283	13,610
Needs help getting around inside	0.024	0.154	13,893
Needs help getting in/out of bed	0.025	0.156	13,868
Work limitation due to health conditions	0.423	0.494	19,073
Arthritis	0.129	0.335	19,073
Back	0.062	0.242	19,073
Blind	0.026	0.159	19,073
Cancer	0.016	0.125	19,073
Deaf	0.023	0.149	19,073
Deformity	0.027	0.162	19,073
Diabetes	0.030	0.170	19,073
Heart	0.090	0.287	19,073
Hernia	0.006	0.080	19,073
Hypertension	0.036	0.185	19,073
Kidney	0.005	0.067	19,073
Lung	0.043	0.203	19,073
Mental illness	0.005	0.067	19,073
Missing limb	0.003	0.056	19,073
Paralysis	0.006	0.075	19,073
Senility	0.007	0.084	19,073
Stomach	0.010	0.099	19,073
Stroke	0.021	0.144	19,073
Thyroid	0.003	0.056	19,073
Other	0.066	0.247	19,073
Individual characteristics			
Education	11.432	3.208	26,030
Female	0.580	0.494	4,795
Age	72.079	5.606	4,795

Source: Author's calculations based on data from the U.S. Census Bureau, *Survey of Income and Program Participation*.

Lleras-Muney (2006).[12] Using WLS, Lleras-Muney's estimate is –0.036, and using IV, her estimate is –0.063. These estimates imply huge effects. For example, the IV estimate implies that one additional year of education would reduce the ten-year mortality rate by about 60 percent.[13] In table 3, I show the results of the replication exercise, as well as the effects of expanding the sample and employing additional robustness checks.

TABLE 3			
New estimates of effects of education on mortality			
Sample and specification	**WLS**	**IV**	**Number of observations**
A. 1960–80			
1960–1980 1%:			
No age controls, region × cohort	–0.036 (0.004)	–0.072 (0.025)	4,792
1960 1%, 1970 2%, and 1980 5%:			
No age controls, region × cohort	–0.045 (0.004)	–0.045 (0.024)	4,797
With age cubic, region × cohort	–0.039 (0.004)	–0.047 (0.024)	4,797
With age cubic × Census year, region × cohort	–0.040 (0.004)	–0.047 (0.024)	4,797
With age cubic × Census year, state × cohort trend	–0.048 (0.004)	–0.016 (0.024)	4,797
B. 1960–2000			
1960 1%, 1970 2%, and 1980–2000 5%:			
With age cubic × Census year	–0.034 (0.003)	–0.026 (0.015)	8,636
With age cubic × Census year, state × cohort trend	–0.036 (0.003)	–0.012 (0.016)	8,636
C. 1960–2000, by Census year			
1960 1%, 1970 2%, and 1980–2000 5% with age cubic × Census year:			
Estimated effect for 1960–70	–0.025 (0.006)	–0.081 (0.052)	2,397
Estimated effect for 1970–80	–0.061 (0.005)	–0.023 (0.033)	2,400
Estimated effect for 1980–90	–0.043 (0.004)	0.023 (0.029)	2,399
Estimated effect for 1990–2000	–0.012 (0.005)	0.027 (0.039)	1,440
D. 1960–2000, by age			
1960 1%, 1970 2%, and 1980–2000 5% with age cubic × Census year:			
35–54 year olds	–0.017 (0.005)	–0.067 (0.036)	2,879
55–64 year olds	–0.039 (0.005)	0.063 (0.053)	2,398
65–89 year olds	–0.030 (0.003)	–0.047 (0.023)	3,071
E. 1960–2000, by cohort			
1960 1%, 1970 2%, and 1980–2000 5% with age cubic × Census year:			
Cohorts born in 1901–12	–0.019 (0.004)	–0.203 (0.125)	3,644
Cohorts born in 1913–25	–0.017 (0.004)	0.025 (0.023)	4,992

Notes: WLS means weighted least squares. IV means instrumental variables. The dependent variable is the ten-year mortality rate; table entries are the coefficient on education. All specifications include year dummies, cohort dummies, state of birth dummies, region of birth interacted with cohort, and an intercept (except for panel A, fifth row, and panel B, second row). Estimates are weighted using the number of observations in the cell in the base year. Standard errors, shown in parentheses, are clustered at the state of birth and cohort level.

In the first row of panel A of table 3, I match the WLS estimate of –0.036 exactly, although my IV estimate of –0.072 is slightly larger. It is also worth pointing out that the partial F statistic on the first stage regression is reasonable at 7.5.[14] The second row of panel A uses the 1960 (1 percent) sample, as well as the larger samples for 1970 (2 percent) and 1980 (5 percent), and utilizes the Goldin and Katz (2003) data for constructing the instruments. I find that the WLS estimate rises to –0.045 and that the IV estimates drop considerably to –0.045. Had I used the Lleras-Muney data for constructing the instruments, the estimate would be exactly the same at –0.045. However, the standard error would have declined by about 25 percent relative to the first row, suggesting that expanding the sample provides considerably more precision. In the third and fourth rows of panel A, I control for age and find that this lowers the WLS estimates a little and increases the IV estimates a little. In the fifth row, I drop the region of birth interactions with cohort and instead use state-specific linear (cohort) trends. This raises the WLS estimate to –0.048, but I now find that the IV coefficient is sharply lower at –0.016 and is no longer statistically significant. However, the fact that the standard error does not rise suggests that the precision is the same when including the state-specific trends.

In panel B of table 3, I add data from the 5 percent samples of the 1990 and 2000 U.S. Censuses. With this larger data set, I construct death rates over four ten-year periods and therefore follow cohorts over a longer period with a considerably larger sample. Given that the sample also tracks the cohorts later in life when mortality rates are much higher, the age controls are essential. I use a cubic in age, although I find that the results are not very sensitive to the choice of the polynomial. Since medical technology and other health-related factors might change over time, I have also interacted the cubic in age with the U.S. Census year. In this specification (the first row of panel B), I now find that the WLS estimate is about –0.034 and that the IV estimate is –0.026. Both of these estimates are a bit more plausible than the ones mentioned previously. The IV estimate is now significant at the 10 percent level, but not at the 5 percent level. With this larger sample, the inclusion of state-specific cohort trends again results in a point estimate that is much smaller in magnitude (–0.012) and not statistically distinguishable from zero (the second row of panel B), despite a similar degree of precision.

In the remaining panels of table 3, I examine how the effects vary by year, age, and cohort. In panel C, I separately estimate the education coefficient for each U.S. Census year. Since the specification includes a full set of cohort dummies, these are equivalent to age controls when using a single U.S. Census year. Although the WLS estimates are significant in all years, they peak in 1970–80 at –0.061 and drop to only –0.012 by 1990–2000. The IV estimates have large standard errors, so they are likely to be imprecisely estimated. Nonetheless, the point estimate is large only for 1960–70 and is actually positive for 1980–90 and 1990–2000. In panel D, I stratify the sample by three age ranges: 35–54, 55–64, and 65–89. Here I observe different patterns between the WLS and IV specifications. The WLS estimates suggest that the largest effect may be for those aged 55–64, while the IV estimates are largest for those aged 35–54. Given the imprecision of the estimates, I cannot draw any meaningful inferences regarding the age pattern.

Panel E of table 3, however, provides a striking result when using the IV specification. It appears that the entire effect of education on mortality arising from compulsory schooling laws is due to cohorts born in 1901–12, who constitute just over 40 percent of the sample. In fact for those born in 1913–25, the point estimate is actually positive.

Interpreting the mortality results

I interpret the results in the fifth row of panel A and the second row of panel B of table 3 as suggesting that I cannot reject the null hypothesis that the effect of education on mortality is zero. In other words, education has no causal effect on mortality once I adequately control for state time trends. An alternative view might be that once one includes state time trends, the coefficient is smaller but still negative, and that the standard errors are simply too large to estimate the effect precisely, and therefore, I cannot rule out a causal effect. One might be concerned, for example, that the instruments are highly collinear with the time trends. However, as I have shown, the standard errors do not rise when including the time trends. In any case, this alternative interpretation of the results would implicitly start with the hypothesis that there *is* a causal effect and that the results here do not offer sufficient evidence to reject *that* hypothesis—a strong assumption given that the literature has yet to successfully identify a causal effect.

If one takes seriously the point estimates shown in the fifth row of panel A and the second row of panel B of table 3 (despite their statistical insignificance), then this implies that the causal effects of education on mortality are much smaller than previously thought. A more reasonable estimate then is that an additional year of schooling lowers mortality risk over a ten-year period by about 10 percent. This is still a large

effect that might reflect the true causal effect. Still, it bears repeating that using the current research design, I am unable to reject the hypothesis that the true effect is actually zero.

My analysis also suggests that, upon closer inspection, the results are driven by cohorts born very early in the century and their mortality experience during the 1960–70 period. One possible explanation could be that the effect of education stayed roughly constant but that compulsory schooling laws had their biggest effect on those born earlier in the century. However, I have run the first-stage regressions by these cohort groupings and found that the partial F statistics on the instruments are actually much higher for the 1913–25 cohorts. This suggests that the schooling laws may actually have been more binding for the later cohorts, casting doubt on this alternative explanation.

Health outcome results

Table 4 presents the results using the microdata on health outcomes using the SIPP. The first column shows the effects of education using a simple probit (or ordinary least squares, or OLS), which does not account for endogeneity. The second column presents the 2SCML (or IV) estimates using the compulsory schooling laws as instruments. Given the possible effects of education on mortality and the fact that outcomes in the SIPP are not observed until at least 1984, one might not expect any remaining health effects to be apparent. As it turns out, I do find significant effects using the instruments for several broad health outcomes. The first row of panel A shows that self-reported health measured as a continuous variable is affected by education. The IV estimate of –0.23 is more than twice the OLS estimate of –0.09. In the fourth column using a Hausman test of exogeneity, I can reject that the OLS and IV coefficients are the same at the 7 percent level (shown as 0.074 in the table). Translating the SRH into a health index on a 1–100 scale following Johnson and Schoeni's (2007) approach, the IV estimate implies that an increase in schooling by one year improves the health index by 4.5 points, or about 7 percent evaluated at the mean (third column). I also estimate that the probability of being in fair or poor health is reduced by 8.2 percentage points with an additional year of schooling, a fairly large effect that is statistically different from the naive probit at the 18 percent significance level. I do not find, however, that any of the measures of hospitalization or days spent in bed are significant when accounting for endogeneity.

Looking across a variety of measures of physical function, I find that, while all of the naive probit estimates are significant and of the expected sign, the

two-stage estimates are typically not significant. Those who have an additional year of schooling because of compulsory schooling laws are no less likely to have trouble lifting, walking, climbing stairs, getting around outside the house, getting around inside the house, or getting into or out of bed. In fact for many of these outcomes, the coefficients are actually positive, suggesting they have a greater propensity for worse health. On the other hand, those with greater schooling associated with compulsory schooling laws are dramatically less likely to experience problems with seeing, hearing, or speaking. In almost all of these cases, the differences between the simple probit and the 2SCML estimates are very large and statistically different at about the 10 percent level. For example, the 2SCML estimates imply that an additional year of schooling reduces the probability of having trouble "seeing" by 5.6 percentage points. In this sample, the mean rate of this health outcome is 13.6 percent. These results might suggest that the channel by which general health is compromised for those with less schooling may be related to sensory functions.

Next, I estimate results based on the incidence of specific health conditions. Recall that these conditions are only identified for subsets of individuals and that the screening criteria changed across SIPP survey years. Also recall that all individuals are included regardless of whether they were screened for this question, so as to avoid using a sample of only those in poor health. Generally, the underlying health conditions were only asked of individuals who reported particular kinds of activity limitations, reported having a work disability, or reported being in fair or poor health. This is captured by the variable "health limitation," which, not surprisingly, is significant under both probit and 2SCML. When I turn to the estimated likelihood of having one of the underlying health conditions, the probit estimates once again are significant in every case. The 2SCML estimates, however, are only negative and significant for four outcomes: back or spine problems; stiffness or deformity of a limb; diabetes; and senility/dementia/Alzheimer's disease. It is important to point out that "trouble seeing," "trouble hearing," and "trouble speaking" were never used as screening criteria for asking about an underlying health condition. This likely explains why blindness and deafness are not significant within the subsamples.

Surprisingly, both kidney problems and hypertension appear to be positively associated with more schooling. This is especially notable because these are two outcomes for which self-management and recent technological advances appear to be especially important. According to appendix table B of

			TABLE 4		
		Estimates of effects of education on health outcomes			
Dependent variable	**OLS/probit**	**IV/2SCML**	**IV/2SCML effect size**	**Exogeneity test *p* value**	**Number of observations**
A. General health outcomes					
Self-reported health (1 is excellent, 5 is poor)	−0.0941 (0.0023)	−0.2289 (0.0745)	−0.074	0.074	26,030
Health index (1–100 scale)	1.9674 (0.0511)	4.5345 (1.6738)	0.067	0.131	26,030
Fair or poor health	−0.0359 (0.0010)	−0.0824 (0.0343)	−0.230	0.176	26,030
Poor health	−0.0141 (0.0006)	−0.0269 (0.0206)	−0.226	0.533	26,030
Hospitalized in last year	−0.0049 (0.0008)	−0.0268 (0.0241)	−0.149	0.364	26,484
Days in bed, last four months	−0.3310 (0.0364)	2.1526 (1.4848)	0.547	0.074	25,223
Number of times hospitalized	−0.0101 (0.0024)	−0.0944 (0.0884)	−0.335	0.329	22,229
Number of nights in hospital	−0.0730 (0.0186)	−1.0828 (0.7668)	−0.567	0.185	26,289
B. Functional limitations/activities of daily living/instrumental activities of daily living					
Trouble seeing	−0.0122 (0.0007)	−0.0559 (0.0254)	−0.412	0.085	20,853
Trouble hearing	−0.0103 (0.0007)	−0.0499 (0.0247)	−0.329	0.109	20,845
Trouble speaking	−0.0019 (0.0002)	−0.0192 (0.0079)	−0.909	0.039	20,573
Trouble lifting	−0.0198 (0.0009)	−0.0055 (0.0330)	−0.023	0.667	20,837
Trouble walking	−0.0251 (0.0011)	0.0130 (0.0325)	0.045	0.242	20,797
Trouble with stairs	−0.0250 (0.0010)	−0.0066 (0.0324)	−0.024	0.993	20,820
Trouble getting around outside the home	−0.0120 (0.0008)	−0.0146 (0.0257)	−0.114	0.918	17,401
Trouble getting around inside the home	−0.0048 (0.0005)	0.0051 (0.0208)	0.087	0.635	17,463
Trouble getting in/ out of bed	−0.0056 (0.0006)	0.0013 (0.0230)	0.016	0.764	17,621
Trouble seeing at all	−0.0020 (0.0002)	−0.0078 (0.0084)	−0.343	0.490	20,589
Trouble hearing at all	−0.0008 (0.0001)	−0.0100 (0.0045)	−0.758	0.060	20,256
Trouble speaking at all	0.0000 (0.0001)	−0.0008 (0.0001)	−0.284	0.000	7,516
Trouble lifting at all	−0.0100 (0.0007)	−0.0029 (0.0250)	−0.025	0.775	20,789
Trouble walking at all	−0.0148 (0.0008)	0.0107 (0.0260)	0.069	0.328	20,723
Trouble with stairs at all	−0.0114 (0.0006)	0.0071 (0.0202)	0.061	0.359	20,775
Needs help getting around outside	−0.0066 (0.0007)	0.0044 (0.0153)	0.050	0.470	13,598

TABLE 4 (CONTINUED)

Estimates of effects of education on health outcomes

Dependent variable	OLS/probit	IV/2SCML	IV/2SCML effect size	Exogeneity test *p* value	Number of observations
Needs help getting around inside	−0.0010 (0.0002)	0.0108 (0.0078)	0.446	0.125	13,757
Needs help getting in/out of bed	−0.0011 (0.0003)	0.0092 (0.0080)	0.372	0.191	13,794
C. Specific health conditions					
Health limitation	−0.0250 (0.0013)	−0.0743 (0.0348)	−0.175	0.157	19,073
Arthritis	−0.0088 (0.0008)	−0.0043 (0.0217)	−0.034	0.836	19,012
Back	−0.0028 (0.0005)	−0.0349 (0.0167)	−0.561	0.061	18,924
Blind	−0.0014 (0.0003)	0.0145 (0.0084)	0.557	0.060	18,454
Cancer	−0.0007 (0.0002)	0.0025 (0.0078)	0.161	0.677	18,569
Deaf	−0.0003 (0.0002)	−0.0041 (0.0064)	−0.179	0.568	18,422
Deformity	−0.0006 (0.0002)	−0.0159 (0.0066)	−0.591	0.018	18,821
Diabetes	−0.0023 (0.0003)	−0.0258 (0.0082)	−0.868	0.007	18,688
Heart	−0.0062 (0.0006)	−0.0014 (0.0194)	−0.016	0.804	19,025
Hernia	−0.0003 (0.0001)	0.0023 (0.0037)	0.362	0.454	17,179
Hypertension	−0.0031 (0.0004)	0.0376 (0.0124)	1.053	0.000	18,683
Kidney	−0.0001 (0.0001)	0.0042 (0.0027)	0.938	0.072	16,593
Lung	−0.0037 (0.0005)	0.0203 (0.0152)	0.472	0.106	19,060
Mental illness	−0.00009 (0.00008)	−0.0002 (0.0424)	−0.045	0.932	15,794
Missing limb	−0.00007 (0.00005)	−0.0019 (0.0016)	−0.580	0.155	14,565
Paralysis	−0.00011 (0.00006)	0.0016 (0.0020)	0.287	0.348	17,301
Senility	−0.00005 (0.00002)	−0.0015 (0.0006)	−0.214	0.070	17,993
Stomach	−0.0006 (0.0002)	0.0069 (0.0060)	0.695	0.195	17,701
Stroke	−0.0008 (0.0003)	0.0084 (0.0090)	0.397	0.295	18,918
Thyroid	−0.0000001 (0.000000)	0.000001 (0.000000)	0.000	0.000	14,559
Other	−0.0023 (0.0005)	−0.0013 (0.0152)	−0.019	0.947	19,060

Notes: OLS means ordinary least squares. IV means instrumental variables. 2SCML means two-stage conditional maximum likelihood. Standard errors, shown in parentheses, are clustered at the state of birth and cohort level.

Glied and Lleras-Muney (2003), treatment of kidney infections experienced substantial innovation. Among the 56 causes of death, kidney disease experienced the fastest decline in age-adjusted mortality from 1986 to 1995—falling more than 9 percent per year (Glied and Lleras-Muney, 2003, p. 8, appendix table B). Accordingly, a steep (negative) gradient between education and kidney disease would presumably be expected. It is therefore of note that the 2SCML specification finds an increase in the incidence of kidney problems among those with high education. Treatment of diabetes is "often considered the prototype for chronic disease management" (Goldman and Smith, 2002). My findings, which analyze a broad range of health conditions and chronic diseases, would suggest that, insofar as the formal schooling is concerned, diabetes appears to be an exception. In the SIPP data, diabetes enters in the expected direction; that is, increases in schooling appear to reduce the incidence of severe cases of diabetes.

On the one hand, since diabetes is also associated with loss of limbs and poor vision, the diabetes result could be a plausible explanation for those findings. On the other hand, kidney problems and hypertension, which are also commonly associated with diabetes, go in the wrong direction. Further, there is no well-established connection between diabetes and speech, hearing, and back problems. An alternative explanation for the diabetes result could be that states that had higher compulsory schooling levels also promoted nutritional policies that might have reduced adult onset of diabetes. Overall, however, one conclusion that may be drawn from this table is that there is little support for the "decision-making" hypothesis.

I would also note that explanations for the link between education and health that focus on better health care access due to more financial resources (for example, from higher income and a better paying occupation) or unobserved time preferences do not appear to be consistent with these results. These explanations would likely imply that many outcomes ought to be affected, not just a few.

There are two important limitations to this analysis. First, I observe individuals only if they have survived into the 1980s and 1990s when they are anywhere between the ages of 59 and 83. This sample is almost certainly positively selected on education and health, so it is unclear to what extent they may be generalized. I suspect that because of this selection, my results are biased against finding any effects of education on improving health, making it still surprising that there are very large negative coefficients on the incidence of several negative health outcomes. Second, because

specific health conditions are only asked of those who report an activity limitation or being in fair or poor health, some individuals with a particular condition may not be captured in the analysis. Nonetheless, it may be even more meaningful to identify the effects of education on specific conditions that were severe enough to cause an activity limitation.

Conclusion

In this article, I expand upon the growing literature that attempts to identify whether there is a causal effect of education on health. I closely examine the effects of education induced by compulsory schooling laws early in the twentieth century on long-term health, using several approaches. First, I revisit the results in Lleras-Muney (2005, 2006) by expanding the U.S. Census sample and employing a variety of robustness checks. The main finding is that the effects of education on mortality induced by changes in compulsory schooling laws are not robust to including state-specific time trends, suggesting that a causal interpretation is unwarranted.

Second, I use the SIPP to identify not only general health effects but also specific health outcomes that were induced by changes in state compulsory schooling laws to see if these outcomes correspond to our existing theories of how education affects health. The results suggest that there is a large effect of education on general health status arising from compulsory schooling laws that are robust to state time trends. However, I find that, with the important exception of diabetes, none of the other specific health conditions that are associated with education (for example, vision, hearing, speaking ability, back problems, deformities, and senility) correspond to the leading theories of how education improves health (for example, technological improvements, better decision-making, lower discount rates, higher income). This suggests that either our theories are incorrect or that the compulsory schooling laws are suspect instruments. An important caveat, however, is that the SIPP analysis uses a sample of older individuals who are almost surely positively selected on education and health. While this likely makes it more difficult to detect effects of education on improved health, it also raises questions as to how far one can generalize these results.

A few other studies have begun to implement strategies to better identify the causal effects of education on health with mixed findings. In a working paper, Clark and Royer (2007) use differences in compulsory schooling laws affecting very narrowly defined birth cohorts in the United Kingdom, combined with *individual*-level mortality data and find very small

effects of education on mortality, which are consistent with the results here. In another working paper, Deschenes (2007) uses plausibly exogenous variation based on cohort size in the U.S. and estimates a statistically significant and large effect of education on mortality using a grouped estimator. Deschenes' estimates suggest that an additional year of schooling adds an additional year to life expectancy. Because we are still only in the early stages of our understanding of this important issue, it is important to conduct replication and extension exercises on the small number of studies that have used more credible research strategies.

NOTES

[1]Kolata (2007).

[2]For example, Deaton and Paxson (2004) document that there is a strong association between education and health in the United Kingdom.

[3]See Lyman (2006). The National Institute on Aging is part of the National Institutes of Health.

[4]The results from using the Lleras-Muney (2005) instruments instead of the Goldin and Katz (2003) instruments are not very different, and are in an earlier version of this article, Mazumder (2007).

[5]The IPUMS are from the University of Minnesota, Minnesota Population Center.

[6]Lleras-Muney (2002) found no effect of compulsory schooling laws on the education levels of blacks.

[7]I thank Jay Bhattacharya for this suggestion. In a previous version of the article, I found very similar results using two-stage least squares for the dichotomous outcomes.

[8]I generally found that the IV results were larger and more significant when using the state trends than when using region of birth interacted with cohort. The ordinary least squares results were virtually identical under either specification.

[9]The 1990 and 1996 panels include an oversample of poorer households. The restriction to the noninstitutionalized population means that those living in nursing homes are not included in the survey. However, more than 90 percent of the disabled and more than 80 percent of those requiring long-term care live outside of institutions; for further details, see http://aspe.hhs.gov/daltcp/reports/rn11.htm.

[10]See Johnson and Schoeni (2007) and the citations therein for a discussion of this approach.

[11]I pool responses from the 1984, 1990–93, and 1996 SIPPs in order to maximize sample size. Unfortunately, different criteria were used across the SIPP survey years to select the subsamples for which specific health conditions were asked. For example, in 1996 the health conditions were asked of those who reported being in fair or poor health. I found that it was important to combine all of the subsamples in all of the years in order to have enough power to identify effects. There are also an additional set of ten outcomes that are not used because they were not available in the 1984 SIPP. Experimentation with a smaller sample suggests that the conclusions are not altered by dropping these other outcomes.

[12]Note that these are estimates from errata that correct the previous estimates in Lleras-Muney (2005). See Mazumder (2007) for more details.

[13]The mean ten-year mortality rate in Lleras-Muney (2005) is 10.6 percent, so a reduction of 6.3 percentage points implies a 59 percent reduction in mortality.

[14]The partial F statistic rises to 9.07 when using the expanded sample.

REFERENCES

Acemoglu, D., and J. Angrist, 2001, "How large are human capital externalities? Evidence from compulsory schooling laws," in *NBER Macroeconomics Annual 2000*, B. S. Bernanke and K. S. Rogoff (eds.), Cambridge, MA: MIT Press, pp. 9–59.

Becker, G. S., and C. B. Mulligan, 1997, "The endogenous determination of time preference," *Quarterly Journal of Economics,* Vol. 112, No. 3, August, pp. 729–758.

Case, A., D. Lubotsky, and C. Paxson, 2002, "Economic status and health in childhood: The origins of the gradient," *American Economic Review*, Vol. 92, No. 5, December, pp. 1308–1334.

Clark, D., and H. Royer, 2007, "The effect of education on longevity: Evidence from the United Kingdom," Case Western Reserve University, working paper.

Cutler, D., and G. Miller, 2005, "The role of public health improvements in health advances: The twentieth-century United States," *Demography*, Vol. 42, No. 1, February, pp. 1–22.

Deaton, A., and C. Paxson, 2004, "Mortality, income, and income inequality over time in Britain and the United States," in *Perspectives on the Economics of Aging*, D. A. Wise (ed.), Chicago: University of Chicago Press, pp. 247–280.

Deschenes, O., 2007, "The effect of education on adult mortality: Evidence from the baby boom generation," University of California, Santa Barbara, working paper.

Dhir, R., and J. P. Leigh, 1997, "Schooling and frailty among seniors," *Economics of Education Review*, Vol. 16, No. 1, February, pp. 45–57.

Glied, S., and A. Lleras-Muney, 2003, "Health inequality, education, and medical innovation," National Bureau of Economic Review, working paper, No. 9738, June.

Goldin, C., and L. F. Katz, 2003, "Mass secondary schooling and the state," National Bureau of Economic Review, working paper, No. 10075, November.

Goldman, D. P., and J. P. Smith, 2002, "Can patient self-management help explain the SES health gradient?," *Proceedings of the National Academy of Sciences*, Vol. 99, No. 16, August 6, pp. 10929–10934.

Grossman, M., 2005, "Education and nonmarket outcomes," National Bureau of Economic Review, working paper, No. 11582, August.

Gunderson, G. W., 1971, "The National School Lunch Program: Background and development," U.S. Department of Agriculture, Food and Nutrition Service, report, available at www.fns.usda.gov/cnd/Lunch/AboutLunch/ProgramHistory.htm.

Hunter, R., 1904, *Poverty*, New York: Macmillan.

Johnson, R. C., and R. F. Schoeni, 2007, "The influence of early-life events on human capital, health status, and labor market outcomes over the life course," University of California, Berkeley, Institute for Research on Labor and Employment, working paper, No. iirwps-140-07, January 2.

Kitagawa, E. M., and P. M. Hauser, 1973, *Differential Mortality in the United States: A Study in Socioeconomic Epidemiology*, Cambridge, MA: Harvard University Press.

Kolata, G., 2007, "A surprising secret to a long life: Stay in school," *New York Times,* January 3, available at www.nytimes.com/2007/01/03/health/03aging.html.

Lleras-Muney, A., 2006, "Erratum: The relationship between education and adult mortality in the United States," *Review of Economic Studies*, Vol. 73, No. 3, p. 847.

_____**,** 2005, "The relationship between education and adult mortality in the United States," *Review of Economic Studies*, Vol. 72, No. 1, pp. 189–221.

_____**,** 2002, "Were compulsory attendance and child labor laws effective? An analysis from 1915 to 1939," *Journal of Law and Economics*, Vol. 45, No. 2, part 1, October, pp. 401–435.

Lyman, R., 2006, "Census report foresees no crisis over aging generation's health," *New York Times,* March 10, available at www.nytimes.com/2006/03/10/national/10aging.html.

Mazumder, B., 2007, "How did schooling laws improve long-term health and lower mortality?," Federal Reserve Bank of Chicago, working paper, No. WP-2006-23, revised January 24, 2007.

Marmot, M. G., 1994, "Social differences in health within and between populations," *Daedalus*, Vol. 123, No. 4, pp. 197–216.

National Institutes of Health, 2003, "Pathways linking education to health," report, Bethesda, MD, No. RFA OB-03-001, January 8, available at http://grants1.nih.gov/grants/guide/rfa-files/RFA-OB-03-001.html.

Rivers, D., and Q. H. Vuong, 1988, "Limited information estimators and exogeneity tests for simultaneous probit models," *Journal of Econometrics*, Vol. 39, No. 3, November, pp. 347–366.

ERRATUM, Corrected Table 3: New Estimates of effects of education on mortality

		IV	Number of observations
A. 1960 - 1980			
1960-1980 1%			
No age controls, region X cohort	-0.036	-0.072	4792
	(0.004)	(0.025)	
1960 1%, 1970 2%, and 1980 5%:			
No age controls, region × cohort	-0.045	-0.045	4797
	(0.004)	(0.024)	
With age cubic, region × cohort	-0.039	-0.047	4797
	(0.004)	(0.024)	
With age cubic × Census year, region × cohort	-0.039	-0.047	4797
	(0.004)	(0.024)	
With age cubic × Census year, state × cohort trend	-0.040	0.003	4797
	(0.004)	(0.038)	
B. 1960 - 2000			
1960 1%, 1970 2%, and 1980–2000 5%:			
With age cubic × Census year	-0.034	-0.029	8636
	(0.003)	(0.015)	
With age cubic × Census year, state × cohort trend	-0.035	0.006	8636
	(0.003)	(0.031)	
C. 1960–2000, by Census year			
1960 1%, 1970 2%, and 1980–2000 5% with age cubic:			
Estimated effect for 1960–70	-0.025	-0.081	2397
	(0.006)	(0.052)	
Estimated effect for 1970–80	-0.061	-0.023	2400
	(0.005)	(0.033)	
Estimated effect for 1980–90	-0.043	0.023	2399
	(0.004)	(0.029)	
Estimated effect for 1990–2000	-0.012	0.027	1440
	(0.005)	(0.039)	
D. 1960–2000, by age			
1960 1%, 1970 2%, and 1980–2000 5% with age cubic × Census year:			
35–54 year olds	-0.017	-0.064	2879
	(0.005)	(0.036)	
55–64 year olds	-0.039	0.063	2398
	(0.005)	(0.053)	
65–89 year olds	-0.031	-0.052	3359
	(0.003)	(0.022)	
E. 1960–2000, by cohort			
1960 1%, 1970 2%, and 1980–2000 5% with age cubic × Census year:			
Cohorts born in 1901–12	-0.019	-0.200	3644
	(0.004)	(0.124)	
Cohorts born in 1913–25	-0.017	0.029	4992
	(0.004)	(0.023)	

Notes: WLS means weighted least squares. IV means instrumental variables. The dependent variable is the ten-year mortality rate; table entries are the coefficient on education. All specifications include year dummies, cohort dummies, state of birth dummies, region of birth interacted with cohort, and an intercept (except for panel A, fifth row, and panel B, second row). Estimates are weighted using the number of observations in the cell in the base year. Standard errors, shown in parentheses, are clustered at the state of birth and cohort level.