



Presidential Address

The Relationship Between Health and Schooling

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Many studies suggest that years of formal schooling completed is the most important correlate of good health. There is much less consensus as to whether this correlation reflects causality from more schooling to better health. The relationship may be traced in part to reverse causality and may also reflect “omitted third variables” that cause health and schooling to vary in the same direction. The past three and a half decades have witnessed the development of a large literature focusing on the issue just raised. I deal with that literature and what can be learned from it in this paper.

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“The one social factor that researchers agree is consistently linked to longer lives in every country where it has been studied is education. It is more important than race; it obliterates any effects of income” (Gina Kolata, *A Surprising Secret to Long Life: Stay in School*, *New York Times*, January 3, 2007, p. 1).

INTRODUCTION

Many studies suggest that years of formal schooling completed is the most important correlate of good health. This finding emerges whether health levels are measured by mortality rates, morbidity rates, self-evaluation of health, or psychological indicators of health and whether the units of observation are individuals or groups. There is much less consensus as to whether this correlation reflects causality from more schooling to better health. The relationship may be traced in part to reverse causality since a longer life expectancy increases the payoffs to investments in schooling and since healthier students may attend school for longer periods of time. The relationship may also reflect “omitted third variables” that cause health and schooling to vary in the same direction.

The past three and a half decades have witnessed the development of a large theoretical and empirical literature focusing on the issue just raised. I deal with that literature and what can be learned from it in this paper. Much of my paper deals with the empirical literature on the relationship between an individual’s own health and own schooling or between child health and parents’ schooling.

To motivate this discussion, I examine time-series data on completed schooling, infant mortality, and age-adjusted mortality in the United States from the early 1900s to the present in the next section. I then outline conceptual frameworks that generate causal relationships from health to schooling and from schooling to health in the subsequent and further sections, respectively. In the penultimate section, I call

attention to the role of "third variables." These are variables that may cause health and schooling to vary in the same direction and are difficult to measure.

In the three sections just mentioned, I address relevant empirical evidence. My emphasis is on studies that try to establish causality and to some extent on the difficulty of this undertaking. I cannot deal with the many contributions made to this literature in the past three and a half decades in the space allotted to me in this paper. Therefore, my strategy is to select several older papers that point to or question causal effects and several very recent ones that try to establish causality with refined econometric techniques. The reader is referred to Grossman [2006] for a more comprehensive review of the literature.

TRENDS IN HEALTH AND SCHOOLING

Table 1 contains trends in the health and educational attainment of the US population between 1910 and 2000. The table highlights the dramatic improvements in these outcomes in the past century. To be specific, the infant mortality rate fell by a factor of almost 20, and the age-adjusted mortality rate declined by a factor of almost three. At the same time, there was almost a 10-fold increase in the percentage of the population who completed 4 years of college or more.

To summarize the trends in Table 1, I have run regressions of each of the two mortality rates on the schooling variable and trend terms. Given the high correlation between schooling and time, the estimation of these regressions is perhaps more of an art than a science. In general my procedure is to experiment with linear, quadratic, and cubic trend specification and to select the one with the lowest residual variance. For infant mortality, the cubic model outperforms the other two. For age-adjusted mortality, the quadratic and cubic models do about the same. To be consistent, I select the cubic model for both outcomes. The schooling coefficient in the age-adjusted mortality regression is not, however, sensitive to this selection.

Table 1 Infant mortality rate, age-adjusted mortality rate, and educational attainment, United States, selected years, 1910–2000

<i>Year</i>	<i>Infant mortality rate (deaths per 1,000 live births)</i>	<i>Age-adjusted mortality rate (deaths per 100,000 population based on year 2000 standard population)</i>	<i>College graduates (percentage of persons aged 25 and older who completed 4 years of college or more)</i>
1910	131.8	2,317.2	2.7
1920	92.2	2,147.1	3.3
1930	69.0	1,943.8	3.9
1940	54.9	1,785.0	4.6
1950	33.0	1,446.0	6.2
1960	27.0	1,339.2	7.7
1970	21.4	1,222.6	11.0
1980	12.9	1,039.1	17.0
1990	9.7	938.7	20.3
2000	7.4	869.0	25.6

Sources: Infant and age-adjusted death rates taken from US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics (www.cdc.gov/nchs/datawh/statab/unpubd/mortabs/hist290.htm for infant mortality and www.cdc.gov/nchs/datawh/statab/unpubd/mortabs/hist293.htm for age-adjusted mortality). College graduates taken from US Department of Commerce, Bureau of the Census, *Statistical Abstract of the United States: 2004–2005, Mini Historical Statistics* (www.census.gov/statab/www/minihs.html).

Table 2 Infant and age-adjusted mortality regressions^a

	<i>Infant mortality rate</i>	<i>Age-adjusted mortality rate</i>
Percentage with 4 years of college or more	-1.617 (-5.06)	-28.950 (-3.96)
R^2	0.996	0.990
F-statistic	2,814.71	1,078.82

^aEach regression contains an intercept and a cubic time trend. *t*-statistics are given in parentheses. The sample size is 50 in the first regression and 49 in the second regression. See the text for more details.

The regression results are presented in Table 2. There are 50 observations in the infant mortality equation because the schooling variable was available in 1910, 1920, 1930, 1940, 1947, 1950, 1952, 1957, 1959, 1960, 1962, and 1964–2002 (the last year in which infant mortality was available when I compiled the series). There are 49 observations in the age-adjusted mortality equation because that series ended in 2001.

The schooling coefficient is negative and statistically significant in each of the two regressions. The growth in schooling “explains” approximately 30 percent of the reduction in infant mortality between 1910 and 2000 and approximately 48 percent of the reduction in age-adjusted mortality. This is solely an accounting explanation because it does not allow for reverse causality from health to schooling and does not consider the role of omitted third variables. I turn to these issues, especially the latter, in the remainder of this paper. I focus on theoretical frameworks that generate relationships between health and schooling and on supporting evidence from micro data.

CAUSALITY FROM HEALTH TO SCHOOLING

Students in poor health are almost certain to miss more days of school due to illness than their healthy peers and may also learn less while they are in school. Both factors suggest negative effects of poor health in childhood on school achievement and ultimately on years of formal schooling completed. Furthermore, this causal path may have long-lasting effects if past health is an input into current health status. Thus, even for non-students, a positive relationship between health and schooling may reflect causality from health to schooling in the absence of controls for past health. Health also may cause schooling because a reduction in mortality increases the number of periods over which the returns from investments in knowledge can be collected.

Edwards and Grossman [1979] document negative effects of low birthweight on school achievement test scores among children ages 6 through 11 years in Cycle II of the US Health Examination Survey, conducted between 1963 and 1965. Case et al. [2005] report a similar finding at a later stage in the life cycle in a unique data set: the 1958 British National Child Development Study. All children born in England, Scotland, and Wales in the week of March 3, 1958 have been followed in this study from birth through age 42. Parents were interviewed at the time of the birth, and health and socioeconomic data have been collected on panel members at ages 7, 11, 16, 23, 33, and 42. Their outcomes are the number of O-level exams passed at age

16 — a key predictor of completed schooling in England — and a measure of completed schooling by age 23 itself.

The Case et al. study goes beyond the Edwards–Grossman study because the results in the latter study suggest a more long-term or permanent effect of infant health on schooling. Neither study, however, controls for favorable genetic traits that may influence both outcomes. Behrman and Rosenzweig [2004] and Black et al. [2007] address this issue by examining the impact of differences in birthweight on differences in years of formal schooling completed among pairs of identical twins. The within-twin estimate of the effect of birthweight on schooling is positive and at least as large as the ordinary least squares (OLS) estimate in both studies.

CAUSALITY FROM SCHOOLING TO HEALTH

Productive efficiency

I [Grossman 1972a,b; 2000] develop a model in which schooling causes health because of a productive efficiency effect in the context of a model of the production of health and the demand for health. My model, which draws on a framework pioneered by Becker [1965] and further extended by Michael [1972; 1973], is somewhat complicated because it involves the selection of an optimal life cycle path of a durable stock of health capital and associated profiles of gross investment in that stock and inputs in the gross investment production function. My model also contains both investment and consumption motives for demanding health. As a consumption commodity, health is a direct source of utility. As an investment commodity, it determines the total amount of time in a period that can be allocated to work in the market and to the production of commodities in the non-market sector.

I simplify my model while retaining the aspects required to study the impacts of schooling on the demand for health and health inputs by employing a static version of my pure investment model in which health does not enter the utility function directly. In the period at issue, say a year, the total amount of time that can be allocated to market and non-market production (h) is not fixed. Instead, it is a positive function of health (H) because increases in health lower the time lost from these activities due to illness and injury ($\partial h/\partial H \equiv G > 0$). Because the output of health has a finite upper limit of 8,760 h or 365 days times 24 h per day if the year is the relevant period, the marginal product of health falls as H rises ($\partial^2 h/\partial H^2 \equiv G_H < 0$). Health is produced with inputs of medical care (M) and the own time of the consumer (T):

$$(1) \quad H = e^{\rho S} F(M, T)$$

where S is the years of formal schooling completed, ρ the positive parameter, and F is the linear homogeneous in M and T . Schooling has a productive efficiency effect in the sense that an increase in S raises the amount of health obtained from given amounts of M and T . Since H is homogeneous of degree one in these two inputs, an increase in schooling raises their marginal products on average. The specification in equation (1) assumes that a one-unit increase in S raises each marginal product by the same percentage (ρ).

The consumer maximizes $Wh - \pi H$, where W is the wage rate and π is the marginal or average cost of producing health. The first-order condition for optimal H is

$$(2) \quad WG = \pi$$

Using this equation, one obtains formulas for the optimal percentage changes in the quantities of H and M caused by a one-unit increase in schooling (S):

$$(3) \quad \frac{\partial \ln H}{\partial S} = \varepsilon \rho$$

$$(4) \quad \frac{\partial \ln M}{\partial S} = (\varepsilon - 1)\rho$$

where

$$(5) \quad \varepsilon \equiv -\frac{G}{HG_H}$$

The effects summarized by equations (3) and (4) hold the wage rate and the price of medical care constant.

The parameter ε is the inverse of the absolute value of the elasticity of the marginal product of health (G) with respect to H . I [Grossman 1972a, b; 2000] show that ε is very likely to be smaller than one because the output of health has a finite upper limit. Given that this condition holds, an increase in schooling is predicted to increase the quantity of health demanded but lower the quantity of medical care demanded.

I [Grossman 1972b] report positive effects of schooling on self-rated health and negative effects of schooling on work-loss days due to illness and injury and on restricted activity days due to illness and injury in a nationally representative 1963 United States survey conducted by the Center for Health Administration Studies and the National Opinion Research Center of the University of Chicago. These findings control for the weekly wage rate, property income, age, and several other variables. In the demand function for medical care (measured by personal medical expenditures on doctors, dentists, hospital care, prescribed and non-prescribed drugs, non-medical practitioners, and medical appliances), the schooling coefficient is positive but not statistically significant. This finding is not consistent with the version of my pure investment model in which the inverse of the elasticity of the marginal product of health with respect to health is less than one in absolute value. But note that I was forced to use a very aggregate measure of medical care and had no information on health insurance. Since more generous health insurance coverage increases the quantity of care demanded and since coverage and schooling are positively related, my estimated schooling effect is biased away from zero.

Wagstaff [1986] provides more definitive evidence in favor of the productive efficiency hypothesis that I attempted to test. He uses the 1976 Danish Welfare Survey to estimate a multiple indicator version of my demand for health model. He performs a principal components analysis of 19 measures of non-chronic health problems to obtain four health indicators that reflect physical mobility, mental health, respiratory health, and presence of pain. He then uses these four variables as indicators of the unobserved stock of health. His estimation technique is the



so-called MIMIC (multiple indicators-multiple causes) model developed by Jöreskog [1973] and Goldberger [1974] and employs the maximum likelihood procedure contained in Jöreskog and Sörbom [1981]. His contribution is unique because it accounts for the multidimensional nature of good health both at the conceptual level and at the empirical level.

Wagstaff reports a positive and significant effect of schooling on his measure of good health and a negative and significant effect of schooling on the number of physician visits in the past 8 months. The latter result differs from mine. One factor that may account for the discrepancy is that Wagstaff has a much better measure of medical care utilization than I had. Another factor is that Wagstaff is able to control for variations in the price of a physician visit. Since money cost of medical care is heavily subsidized in Denmark, this price is given by the time required by survey respondents to travel to their physicians. A similar approach and similar results can be found in a study by Erbsland et al. [1995] with the 1986 West German Socio-economic Panel.

Allocative efficiency

In the productive efficiency approach, an increase in knowledge capital or schooling raises the efficiency of the production process in the non-market or household sector, just as an increase in technology raises the efficiency of the production process in the market sector. Some persons object to this approach. In the specific context of the production of health, Deaton [2002, p. 21] writes: "In many economic models of health, education is seen as enhancing a person's efficiency as a producer of health — a suggestive phrase, but not one that is very explicit about the mechanisms involved." In a study dealing with infant health production, Rosenzweig and Schultz [1982, p. 59] argue: "It is not clear...how education can actually alter marginal products of inputs...unless inputs are omitted from [the production function]. That is, it is doubtful that schooling can affect the production of... [health] without it being associated with some alteration in an input."

The statements by Deaton and by Rosenzweig and Schultz point to an allocative efficiency effect of education. This pertains to situations in which the more educated pick a different mix of inputs to produce a certain commodity than the less educated. The mix selected by the more educated gives them more output of that commodity than the mix selected by the less educated. As the quotes by Deaton and Rosenzweig and Schultz cited above imply, education will have no impact on outputs unless it alters inputs, and education coefficients in production functions will be zero if all relevant inputs are included. Theoretical underpinnings of the allocative efficiency approach are contained in Rosenzweig and Schultz [1982], Kenkel [2000], Glied and Lleras-Muney [2003], de Walque [2005; 2007], and other studies discussed by Grossman [2006]. These treatments correctly recognize the multivariate nature of the health production function and include a variety of market goods inputs, such as diet, cigarette smoking, and alcohol use, in addition to medical care. Some of these inputs have negative marginal products in the production of health. For example, cigarette smoking lowers health but raises utility at least for some consumers because it simultaneously produces the commodity "smoking pleasure" that is a positive source of utility. Hence, models of allocative efficiency incorporate joint production in the non-market sector. Some of these models replace a generic time input with time allocated to such activities as exercise and weight control.

Typically, approaches to allocative efficiency assume that the more educated have more information about the true nature of the production function. For example, the more educated may have more knowledge about the harmful effects of smoking or about what constitutes an appropriate diet. In addition, they may respond to new knowledge more rapidly. These approaches also pay attention to the role of endowed or inherited health. Clearly, a favorable endowment raises current health. At the same time, the demand for inputs with positive marginal products falls while the demand for inputs with negative marginal products may rise.

Situations in which new information becomes available or in which new medical technologies are introduced provide the best setting to explore and test the allocative efficiency hypothesis since most treatments of this effect assume that the more educated respond more rapidly to these new developments. The spread of the HIV/AIDS epidemic since the early 1980s provides one such setting. de Walque [2007] finds that, after more than a decade of prevention campaigns about the dangers of the HIV/AIDS epidemic in Uganda, there has been a significant change in the HIV/education gradient. In 1990 no relationship existed, but by 2000 education lowers the risk of being HIV positive among young individuals. He also reports a positive relationship between schooling and condom use during the recent period, which may partially explain his findings. Not enough time in the AIDS epidemic has elapsed to examine whether a permanent relationship between the prevalence and severity of the disease and schooling has emerged. The weakening of this relationship would provide further support for the allocative efficiency hypothesis, while its persistence would provide support for productive efficiency effects or for the role of third variables (see the next section).

Glied and Lleras-Muney [2003] focus on relationships between schooling and mortality from each of 55 diseases that account for mortality from all diseases and on relationships between schooling and cancer mortality from each of 81 different cancer sites. Their principal result is that negative effects of schooling on mortality are largest for diseases and cancer sites in which progress has been the most rapid.

OMITTED THIRD VARIABLES

Since health and schooling are both endogenous, unobserved third variables may cause both of these outcomes to vary in the same direction. The third-variable hypothesis has received the most amount of attention in assessing whether schooling causes health because it is related to the hypothesis that the positive effect of schooling on earnings, explored in detail by Mincer [1974] and in hundreds of studies since his seminal work (see Card [1999; 2001] for reviews of these studies), is biased upwards by the omission of ability. Fuchs [1982] identifies time preference as the third variable. He argues that persons who are more future oriented (who have a high degree of time preference for the future or discount it at a modest rate) attend school for longer periods of time and make larger investments in their own health and in the health of their children. Thus, the effects of schooling on these outcomes are biased if one fails to control for time preference.

Endogenous taste models make important modifications to the interpretation of time preference in a health outcome equation. For example, proponents of the time preference hypothesis assume that a reduction in the rate of time preference for the present causes years of formal schooling to rise. On the other hand, Becker and Mulligan [1997] argue that causality may run in the opposite direction: namely, an



increase in schooling may *cause* the rate of time preference for the present to fall (may *cause* the rate of time preference for the future to rise). They point out that the *higher* the present value of lifetime utility, the smaller the rate of time preference for the present. Hence, consumers have incentives to make investments that *lower* the rate of time preference for the present.

Becker and Mulligan then show that the marginal costs of investments that lower time preference fall and the marginal benefits rise as income or wealth rises. Marginal benefits also are greater when the length of life is greater. Hence, the equilibrium rate of time preference falls as the level of education rises because education raises income and life expectancy. Moreover, the more educated may be more efficient in making investments that lower the rate of time preference for the present — a form of productive efficiency not associated with health production.

In a widely cited study, Farrell and Fuchs [1982] reject the hypothesis that schooling causes cigarette smoking — a key determinant of unfavorable health outcomes — because completed schooling at age 24 has the same negative effect on smoking participation at age 17 as it does at age 24 in a sample of persons, all of whom were at least high school graduates and were high school seniors at age 17. They conclude that the observed relationship is due solely to time preference. de Walque [2005] applies a difference-in-differences estimation methodology to explore this issue in more detail. Like Farrell and Fuchs, he focuses on the impact of completing college on the probability of smoking before and after completion. Unlike them, he examines participation at much older ages than 24.

To simplify his approach, assume that data are available on the same persons at ages 17 ($t = 0$) and 45 ($t = 1$). Let d be a dichotomous variable that identifies people who graduate from college. The following linear probability model for smoking participation (c_t) is estimated:

$$(6) \quad c_t = \phi_0 + \phi_1 t + \alpha d + \beta dt$$

The coefficient α captures unmeasured characteristics that differ between the treatment group (those who graduate from college) and the control group (those who do not graduate from college). The coefficient (ϕ_1) captures time or age effects that are common to both groups. The coefficient (β) captures the true causal effect of the program (graduating from college) on cigarette smoking.

de Walque's estimates of α and β are negative and statistically significant. The ratio of β to α is approximately equal to 0.4 for the cohort born between 1950 and 1959. It is tempting to conclude that time preference accounts for 70 percent of the impact of college completion on smoking participation (given by the sum of α and β) and that true causality from schooling to smoking accounts for 30 percent of the observed relationship. But note that at least part of the effect summarized by α may reflect direct or indirect (through time preference) factors associated with parents' schooling.

Becker and Mulligan's [1997] endogenous time preference model contains theoretical support for the last proposition. Parents can raise their children's future health, including their adulthood health, by making them more future oriented. Years of formal schooling completed is a time-invariant variable beyond approximately age 25 or 30, while adult health is not time invariant. Thus, parents probably have a more important direct impact on the former than the latter. By making investments that raise their offspring's schooling, parents also induce them

to make investments that lower their rate of time preference for the present and discourage them from smoking.

Definitive estimates of the partial effects of schooling and time preference on health would treat both as endogenous in a system of equations that allows for causality between schooling and time preference in both directions. Given difficulties in measuring time preference and in identifying this system, no attempts have been made to estimate it. There is, however, an extremely promising line of research that treats schooling as endogenous and estimates the causal effect of schooling on health by the method of instrumental variables. This line of research does not attempt to distinguish between the direct effect of schooling on health and the indirect effect that operates through time preference. The latter variable is treated as the disturbance term in the health equation and is assumed to be correlated with schooling. The idea is to find instruments that are correlated with schooling but not correlated with time preference. These variables serve as instruments for schooling in estimation of health equations by two-stage least squares and its variants.

Lleras-Muney [2005], Currie and Moretti [2003], and Chou et al. [2007] are three examples of this line of research. [For other examples, see Grossman 2006]. Lleras-Muney [2005] employs compulsory education laws in effect from 1915 to 1939 to obtain consistent estimates of the effect of education on mortality in synthetic cohorts of successive US Censuses of Population for 1960, 1970, and 1980. This instrument is highly unlikely to be correlated with unobserved determinants of health, especially because she controls for state of birth and other state characteristics at age 14. Her OLS estimates suggest that an additional year of schooling lowers the probability of dying in the next 10 years by 1.3 percentage points. Her IV estimate is much larger: 3.6 percentage points.

Currie and Moretti [2003] examine the relationship between maternal education and low birthweight among US white women with data from individual birth certificates from the Vital Statistics Natality files for 1970–2000. They use information on college openings between 1940 and 1990 to construct an availability measure of college in a woman's 17th year as an instrument for years of college schooling. They find that the negative effect of maternal schooling on low birthweight increases in absolute value when it is estimated by instrumental variables. They also find that the negative IV coefficient of maternal schooling in an equation for the probability of smoking during pregnancy exceeds the corresponding OLS coefficient in absolute value. Since prenatal smoking is the most important modifiable risk factor for poor pregnancy outcomes in the United States, they identify a very plausible mechanism via which more schooling causes better birth outcomes. Their results suggest that the increase in maternal education between the 1950s and the 1980s accounts for 12 percent of the 6 percentage point decline in the incidence of low birthweight in that period.

Chou et al. [2007] exploit a natural experiment to estimate the causal impact of parental education on child health in Taiwan. In 1968, the Taiwanese government extended compulsory education from 6 to 9 years. From that year through 1973, the government opened 254 new junior high schools, an 80 percent increase, at a differential rate among regions. My colleagues and I form treatment and control groups of women who were age 12 or under on the one hand and between the ages of 13 and 20 on the other hand in 1968. Within each region, we exploit variations across cohorts in new junior high school openings to construct an instrument for schooling. We employ this instrument to estimate the causal effects of mother's or father's schooling on the incidence of low birthweight and mortality of infants born



to women in the treatment and control groups in the period from 1978 through 1999. Mother's schooling does indeed cause favorable infant health outcomes. IV estimates are similar in magnitude to OLS estimates. The increase in schooling associated with the reform saved almost 1 infant life in 1,000 live births, resulting in a decline in infant mortality of approximately 11 percent.

The findings of the three studies just reviewed suggest causality from more schooling to better health. The finding that the IV estimates sometimes exceed the OLS estimates may arise because the instruments are based on policy interventions that affect the educational choices of persons with low levels of education [Card 2001]. If different individuals face different health returns to education, IV estimates reflect the marginal rate of return of the group affected by the policies [Angrist et al., 1996]. Card [2001] points out: "For policy evaluation purposes...the average marginal return to schooling in the population may be less relevant than the average return for the group that will be impacted by a proposed reform. In such cases, the best available evidence may be IV estimates of the return to schooling based on similar earlier reforms (p. 1157)."

A second explanation of the larger IV than OLS estimates is that the schooling variable contains random measurement error, which leads to a downward bias in the OLS estimates. As long as the instruments for schooling are not correlated with this error, the IV procedure eliminates this bias [Card 1999; 2001]. A third explanation is that there may be spillover effects in the sense that the health outcome of an individual depends on the average schooling of individuals in his area as well as on his own schooling or that of his parents [Acemoglu 1996; Acemoglu and Angrist 2000].

A cautionary note in interpreting schooling effects in recent IV studies is that the impact of the specific instrument employed on schooling in the first stage typically is modest. Of course, it is possible that small exogenous changes in schooling can have large changes on health in certain settings while having small or no effects on health in other settings. More research in this important area clearly is in order.

CONCLUSIONS

I conclude with some suggestions for future research. A number of the studies mentioned in this paper and in Grossman [2006] bear on the somewhat controversial but highly influential work by Barker [1995] and the less controversial studies by De Stavola et al. [2000] because they report effects of parents' schooling on children's current health status, with past health status held constant. Barker [1995] suggests that in utero growth and adolescent growth affect heart disease at age 75. De Stavola et al. [2000] report that estrogen in utero affects birth size, and large female babies with elevated estrogen have a much higher incidence of pre-menopausal breast cancer. In their study with the 1958 British National Child Development Survey discussed in *Casualty from schooling to health* section, Case et al. [2005] find that men who experienced poor health in utero and at ages 7, 16, 23, and 33 have lower health at age 42, with parents' schooling and socioeconomic status held constant. But self-rated health at age 42 is positively related to parents' schooling, unless own schooling is included in the regression analysis.

In Grossman [2006], I stress the significant effects of parent's schooling and own schooling on current health, with past health held constant. On the other hand, Case and her colleagues stress the significant effects of past health on current health, with



the schooling variables held constant. In general, their results and others suggest a long-term association between parents' attributes and children's attributes including health. Schooling is part of this relationship, but uncoupling the causal links associated with genetic and behavioral factors is very difficult. Clearly, breaking into this complicated bundle is a challenge for future research.

A second challenge is to specify and estimate a model in which time preference can be identified as a potential mechanism via which schooling affects health. Consider the value of this undertaking in the context of the formulation of public policy. Suppose that most of the effect of schooling on health operates through time preference. Then school-based programs to promote health knowledge in areas characterized by low levels of income and education may have much smaller payoffs than programs that encourage the investments in time preference made by the more educated. Indeed, in an ever-changing world in which new information constantly becomes available, general interventions that encourage future-oriented behavior may have much larger rates of return in the long run than specific interventions designed, for example, to discourage cigarette smoking, alcohol abuse, or the use of illegal drugs.

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