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How Education Produces Health: A Hypothetical Framework

by Peter Muennig — September 12, 2007

Background: *High school graduates live six to nine years longer than high school dropouts. Those with less education are more likely to die prematurely of cardiovascular disease, cancer, infectious disease, diabetes, lung disease, and injury than those with more education. Although there is growing evidence that the education-health relationship is causal, and some mechanisms linking education to health have been proposed, there is no gestalt for thinking about the health production function of education.*

Purpose: *The purpose of this article is to outline the mechanisms through which education may produce health.*

Design: *I explore the health risk factors that are more prevalent among those with lower educational attainment to ascertain whether such risk factors plausibly cause the diseases for which the less educated are at risk. To examine these relationships, I conduct a review of the public health, economics, endocrinology, sociology, neurosciences, and other literatures.*

Conclusions: *A remarkably clear path can be drawn between what we now believe to be the risk factors for disease and the primary causes of death among those with lower attainment. Although hypothetical, the pathways outlined in this article can be used as a basis for thinking about the health production function of education. These mechanisms may better allow policy makers to understand the relationship between education and health. They may also be used to guide future research on the health benefits of education. Finally, although the proposed pathways are hypothetical, there is good overall evidence that education produces health. Therefore, health benefits should be included as core outcome measures in future education research.*

INTRODUCTION

Education and health together account for a quarter of the gross domestic product in the United States and are perhaps the two most complex areas of domestic policy (Heffler et al. 2005; National Center for Education Statistics 2005). They are also intertwined. Not only is health critical for learning, but education also appears to be a major determinant of health (Lleras-Muney 2005). In fact, effective education interventions may produce significantly more health at a lower cost than all but a handful of health interventions (Muennig and Woolf , forthcoming; Muennig and Fahs 2001).

Still, schooling is not typically the first remedy that comes to U.S. policy makers' minds when confronted with skyrocketing Medicaid costs. Nor have education researchers historically collected relevant health outcomes, such as mortality rates or measures of morbidity, when studying education interventions. Education and health are perhaps not often associated in the minds of policy makers and researchers because, although there is good evidence that education improves health, the mechanisms through which it works are poorly understood (Lleras-Muney 2005; Groot and van den Brink 2004; Grossman 2005).

In this article, I critically examine interdisciplinary data to build a map of these causal pathways. I weave these strands of interdisciplinary data together to create a clear and intuitive picture of the relationship between education and health in the United States. I propose that not only do education-related risk factors interact to produce disease, but they also explain the very diseases responsible for most of the six-to-nine-year difference in life expectancy separating U.S. students with high school diplomas from those without.

The article begins with a brief but critical overview of the research exploring whether the relationship between education and premature mortality is causal. The primary causes of death from which the less educated are more likely to die are then explored. These causes of death point to six health risk factors associated with lower educational attainment: higher levels of stress, lower social standing, social deprivation, behavioral risk factors, lower health insurance coverage rates, and suboptimal cognitive skills.

In this article, the focus is educational attainment rather than education quality because it is the most common input used in the scientific literature. I also discuss genetic factors that might contribute to both health and educational attainment. The article concludes by tying these risk factors for disease together into a meaningful whole and outlines preliminary education

policy implications based on establishing the value of this education-as-health-intervention framework.

OVERVIEW OF CAUSAL RELATIONSHIPS

It has been recognized since the time of Hippocrates that social conditions shape the ecological niche in which people live or die (Wilkinson 1999; Lloyd 1983). Some 2,300 years after Hippocrates, Horace Mann proposed that education might serve as a tool for repairing social ills (Mann 1848). He observed that education is "the great equalizer," providing people with the knowledge and technical skills needed to survive. This equalizing force applies to health as well as income (Wong et al. 2002), an association that increasingly appears to be causal in nature (Lleras-Muney 2005; Mazumder 2007; Groot and van den Brink 2004; Grossman 2005).

Evidence of a causal relationship partially draws upon instrumental variable analyses. Lleras-Muney (2005) conducted what is perhaps the best known of these studies, using compulsory schooling laws to examine the causal relationship between educational attainment and mortality. Her instrumental variable analysis exploits the fact that compulsory schooling laws should have a direct influence on educational attainment but not on mortality rates. The analysis first examines the effect of compulsory schooling laws on educational attainment and then estimates the effect of these laws on mortality. The ratio of the effect of the laws on mortality to the effect on educational attainment produces a direct estimate of the causal effect of educational attainment on health. Using a synthetic cohort derived from census data, Lleras-Muney found that compulsory schooling and child labor laws reduced mortality rates by as much as 60%. Given that adjustment for income or occupation produced little effect on the analyses, it was hypothesized that improved cognition, manifested as improved medical decision making, was the causal factor through which educational attainment produces health (Glied and Lleras-Muney 2003).

Mazumder (2007) explored this hypothesis in a larger census sample and used data from the Survey of Income and Program Participation (SIPP). His analysis of the census data raises concerns that the instrument "might be picking up smooth cohort trends in educational attainment rather than discrete increases induced by more stringent compulsory schooling laws" (p. 3). Nonetheless, using the SIPP, he also finds a strong causal association between educational attainment and self-reported health. This latter finding is perhaps surprising because the cohort is older and contains only self-reported diseases among those with self-reported disability, potentially leaving out those subjects who are doing a good job of managing their disease.

Among those who survive to their golden years, those with less education are at a slight survival advantage relative to those with more education (Wong et al. 2002). Using this older SIPP cohort, Mazumder (2007) finds that complex diseases, which require a strong cognitive skill set to manage, were generally associated with poorer health outcomes among those with higher educational attainment. Mazumder (2007) further finds that compulsory vaccination against smallpox may explain much of the mortality effect observed by Lleras-Muney (2005).

Both studies raise critical questions. Lleras-Muney's (2005) evidence that cognitive skills may be responsible for the production of health is supported by a number of academic studies (Gottfredson 2004; Glied and Lleras-Muney 2003; Cutler and Lleras-Muney 2006). However, her finding that education's indirect effects on health via occupation and income play a small role contradicts a large academic literature (Lynch, Kaplan, and Shema 1997; Smith 1999; Winkleby et al. 1992). It is also counterintuitive; one would not be surprised to learn that bankers tend to outlive coal miners.

Lleras-Muney's (2005) finding that income and occupation were not major explanatory variables does not jeopardize the validity of her work, but Mazumder's (2007) findings that cognitive skills are inversely correlated with health and that smallpox vaccination in school is a major explanatory variable in the education-health pathway suggest that the SIPP portion of his analysis suffered from serious confoundings. Mazumder's inverse association between cognitive skills and health status suggests that the more education people have, the *worse* they manage their diseases. Not only is this counterintuitive, but smallpox ceased to be a major cause of mortality by the turn of the 20th century (Fenner et al. 1988). In short, although the bulk of Mazumder's findings largely confirm those of Lleras-Muney (2005), his work also demonstrates the pitfalls of working with data from sources other than randomized trials of education interventions.

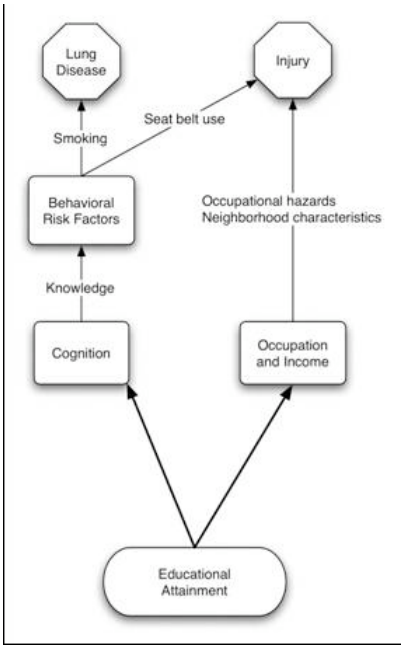
Randomized trials exist, but the health outcomes measured relate more to social pathologies than to definitive outcomes such as mortality. For instance, one small randomized trial of a prekindergarten intervention versus no program showed dramatically lower arrest rates and improved measures of parenting among subjects in the intervention group (Schweinhart 2004). These findings were supported by a similar quasi-experimental study with an active control group (Reynolds et al. 2001).

Beyond the handful of domestic and international instrumental variable studies and randomized studies with proximal outcome measures, few studies specifically examine the causal association between education and health (Lleras-Muney 2005; Groot and van den Brink 2004; Grossman 2005). However, there are a number of reasons to believe that the relationship between educational attainment and health is causal in nature. First, these studies do not reveal patterns in the relationship between health and "third" variables—those other than education—so little of the association is explained by unobserved heterogeneity in correlational analyses (Groot and van den Brink 2004). Second, because children and adolescents have very low rates of

chronic disease, sickness is not a major cause of low educational attainment. Thus, we can be more confident that most of the difference in life expectancy by educational attainment is explained by education rather than poor health. Third, the association is strong and consistent across cultures and time (Feldman et al. 1989; Elo and Preston 1996; Yen and Moss 1999), and fourth, as I note here, the primary causes of death for those with more and less education can be readily explained by the very risk factors for diseases for which less educated persons are most at risk (Wong et al. 2002).

I now attempt to connect the dots between educational attainment and the most common education-related diseases, focusing on the works of epidemiologists, sociologists, economists, urban planners, social psychologists, and even neuroimmunologists (Deaton 2002; Mechanic 2002; Sheridan et al. 1994; McEwen 1998; Sapolsky 2005; Cohen 1999; Syme 1987). Once connected, a rich picture emerges. Specifically, the risk factors most obviously linked to educational attainment are also risk factors for the diseases responsible for the six-to-nine-year gap in life expectancy between those with a high school diploma and those without (Wong et al. 2002).

These diseases include cardiovascular disease (35% of all deaths), cancer (27% of all deaths), infection (9% of all deaths), injury (5% of all deaths), lung disease (5% of all deaths), and diabetes (4% of all deaths). With the exception of injury, these risk factors overlap with respect to the diseases they cause (Figure 1). Many of these underlying risk factors are remarkably consistent with new evidence from the fields of psychology, neuroanatomy, neurophysiology, molecular biology, sociology, and epidemiology. Others, such as smoking or lower rates of health insurance, have been widely discussed (Mechanic 2002; Fuchs 2004). However, the interdisciplinary research helps us better understand and contextualize the linkages between these factors and educational attainment.



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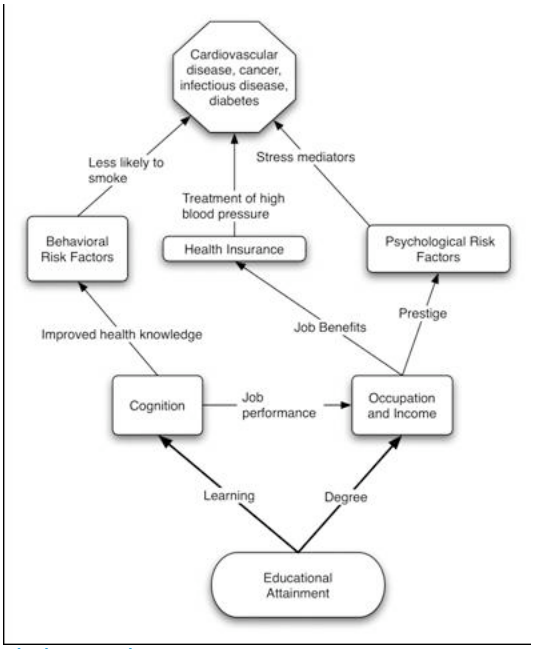

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Figure 1. Selected plausible pathways through which education works to improve health

The following sections set the stage by discussing the relationship between poor health and poor education outcomes. I then examine the evidence related to the six main pathways potentially linking education to health. I present them roughly in order, from the newest putative pathway (the stress hypothesis) to the most established (cognitive ability). I argue that the newer, more tentative linkages are no less important than traditional direct explanations, and I use them to fill in the gaps in the overall education-health picture.

MECHANISMS LINKING HEALTH AND EDUCATION

Before exploring each mechanism, it is important to consider the environmental factors giving rise to poor outcomes in both health and education. Poor health and poor education are tightly intertwined concepts. Native-born parents lacking a high school diploma tend to have lower-than-average income, less healthy children, and children who are themselves at risk of dropping out of high school. Factors contributing to higher dropout rates among such children include attendance at poorly funded schools, exposure to lead paint, abuse, overcrowded living conditions, and a host of other health and environmental factors (Chen, Matthews, and Boyce 2002; Ross and Wu 1996; Wilkinson 1999; De Bellis et al. 1999). The challenge, therefore, is to tease apart the primary ways in which education produces health independent of the influence of these factors on health.

For instance, a lead abatement intervention may improve both health and educational attainment in children, but we would not point to the lead abatement program as causing any observed improvements in both health and education. On the other hand, if a child received an educational intervention that induced him or her to graduate from high school, that individual will be more likely to obtain a quality job and enjoy the benefits of health insurance, a quality job, a lower stress lifestyle, good housing stock, and other health-inducing social benefits. In the latter case, it was the education, not the intervention, that is central to the production of health.

STRESS

Early associations between “Type A” lifestyles and heart disease led to the popular misperception that affluence and education naturally led to stressful lifestyles (Haynes et al. 1978). Wealthier, more educated persons indeed have stress in their lives. But although those with a postgraduate degree report higher levels of having “too many things to do” (relative to high school dropouts), nationally representative self-report surveys suggest that the better educated also have fewer concerns about money, health, leisure time, environmental noise, or problems with children (Taylor 2002). Self-report surveys also suggest that those with more education enjoy lower levels of anger, distress, aches, pains, and other factors that interfere with subjective quality of life than do those with less education (Ross and Van Willigen 1997). Finally, those with more education are more likely to have fulfilling, rewarding jobs, a high sense of control in life, and higher levels of social support, all of which are associated with higher self-reported health and physical functioning (Ross and Wu 1995).

If real, how might these differences in self-reported stressors and annoyances translate into poor health? Functional magnetic resonance imaging (fMRI) studies offer one view. These studies expose small numbers of subjects to stimuli that are intended to evoke an emotional response or a cognitive appraisal of an event. The scanner then detects which areas of the brain receive an increase in blood flow, and statistical testing is performed to ascertain which areas are significantly more active than others. fMRI studies suggest that when one is exposed to a stressor, such as persistent horn honking or a difficult boss, parts of the brain that give these perceptions emotional valence become activated (LeDoux 2003; Roy 2004). These emotional centers, collectively known as the limbic system, in turn activate other circuits in the brain responsible for regulating heart rate, blood pressure, and the production of stress mediators (Rauch et al. 1997).

We can observe the effects of these neural processes on the body by examining the relationship between levels of stress mediators in the blood and educational attainment. When subjects of differing social class (and, by extension, educational attainment) are exposed to mild stressors such as a line-tracing task, persons with a low income and low education tend to exhibit an abnormal stress response (Maes et al. 1998; Markowe et al. 1985; Ridker et al. 2000; Sapolsky 2005; Wamala et al. 1999; Cohen, Tyrrell, and Smith 1991; McEwen 1998). Many of these stress mediators (e.g., cortisol, interleukin-6, catecholamines, C-reactive protein, and fibrinogen) are putative risk factors for cardiovascular disease, hypertension, diabetes, and infectious disease—four of the education-related diseases in Figure 1 (McEwen and Mirsky 2002; McEwen 1998). Nevertheless, it is possible that these stress mediators are merely markers of other processes or that their association with education-related disease is simply the result of spurious association (Davey Smith, Harbord, and Ebrahim 2004).

In addition to leading to high blood pressure, diabetes, and disruption of the immune system, stress may cause oxidative damage, which increases the rate of human cell aging (Epel et al. 2004; Cherkas et al. 2006). The first study in this area recruited unstressed subjects and compared them with subjects with a chronically ill child to ensure large differentials in self-reported stress. They then examined a marker of cell aging, called the telomere, to ascertain whether there was an association between self-reported stress and cell age in the immune system. They found that subjects with high levels of self-reported stress show chromosomal changes that are consistent with an entire decade of additional biological life relative to those with the lower levels. This study was potentially confounded by their selection criteria; parents with a chronically ill child may be more likely to suffer from genetic disease and therefore be more prone to premature cell aging.

A subsequent study overcame this limitation by examining twins who had different levels of educational attainment (along with other markers of socioeconomic status [SES]). Although the outcome measures they included in their study were different, this group of researchers effectively confirmed the findings of Epel et al. (2004). These authors found that the increased aging among less educated, poorer twins could be accounted for by increased rates of smoking and obesity and lower rates of exercise. Although these authors did not fully disentangle these behavioral risk factors from psychosocial stress, the study raises the possibility that behavioral risk factors and stress are intertwined.

In a related series of studies, subjects with various forms of stress, including job-induced stress, were found to have higher levels of DNA damage and higher blood levels of a marker for cancer (Irie, Asami, Nagata, Miyata, et al. 2001; Irie, Asami, Nagata, Ikeda, et al. 2001). This damage is thought to occur when the acute stress response causes the physiological release of oxidative chemicals into the bloodstream.

A subanalysis by Irie et al. (Irie, Asami, Nagata, Miyata, et al. 2001; Irie, Asami, Nagata, Ikeda, et al. 2001) of these data found that effective cognitive coping skills for stress (measured using a validated instrument) were associated with lower levels of DNA damage. Given that increased educational attainment is associated with improved cognitive coping skills and social network size, it is plausible that education may reduce the incidence of disease though this association (Ross and Van Willigen 1997; Wilkinson 1999). The positive correlation between social network size and health has been a longstanding enigma in public health that may be explained by the effects of psychological support on human physiology (Berkman et al. 2000).

Certainly, it is difficult to separate the psychological and material effects of social support. Some of the sources of stress that cause these physiological disruptions—fewer marketable skills, fewer networking connections, and the inability to navigate institutions—both arise from and lead to lower occupational class and lower earnings (Link and Phelan 1995). Further complicating matters is growing evidence that health is also influenced by the way we see ourselves through other's eyes.

SOCIAL STANDING

Social identity theory suggests that individuals tend to categorize other people on the basis of characteristics such as educational attainment as a means of self comparison (Turner 1987; Austin and Worchel 1979). Differences in relative standing—whether due to less education or less wealth—can be a source of anger, envy, or stress. The long human history of bloody wars fought over social class, such as the Bolshevik Revolution, suggests both that these feelings can be powerful and that they can have direct consequences for health and mortality. It has been suggested that the modern manifestations of lower social standing include both internalized physiological disruptions and crime (Wilkinson 1999). This hypothesis was initially drawn from

heavily confounded ecological studies assessing the effects of income inequality on mortality and crime (Lynch et al. 2004), but it is now supported by a stronger base of outcome measures and research designs.

The idea that social status affects health came to light in studies of government workers in England (Marmot, Shipley, and Rose 1984). These studies found that, among persons with good jobs in the same government department and equal access to healthcare, occupational class was still inversely linked to premature mortality. More surprisingly, this "health gradient" extended all the way into white-collar jobs; even those with high-level jobs were at greater risk of premature mortality than those in the most prestigious jobs. Similar gradients were subsequently found for income and education, and these gradients have been observed in a wide variety of cultural and economic contexts (Marmot 2004). In fact, it is possible that the social prestige conferred by an educational degree is a more important determinant of life expectancy than the skills acquired with each year of education (see Figure 2) (Backlund, Sorlie, and Johnson 1999).

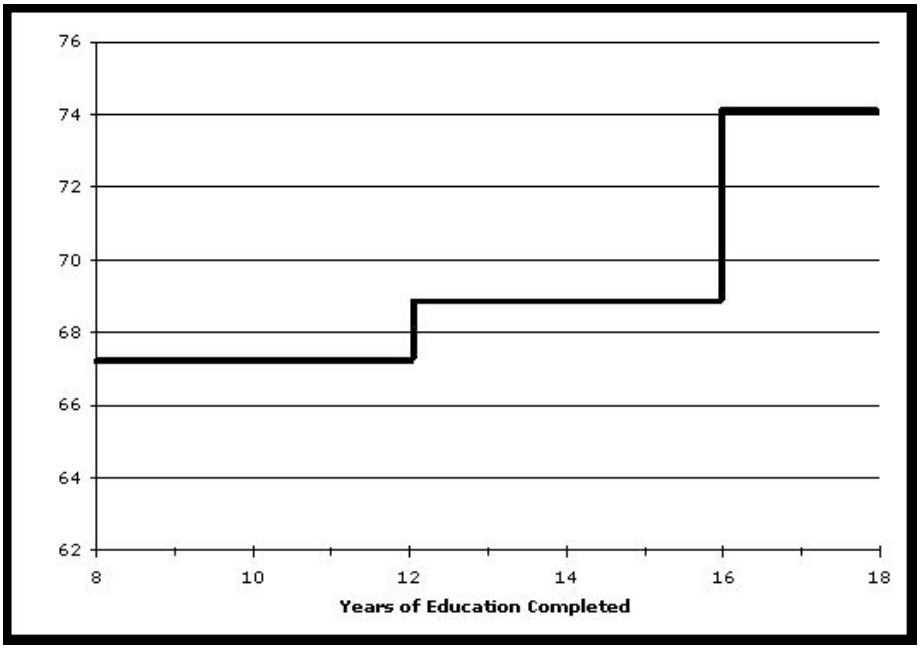

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Figure 2. Change in life expectancy with educational attainment among males. Life expectancy remains relatively flat between educational milestones but increases greatly when degrees are conferred. (Chart is adapted from Backlund et al. 1999 after converting mortality risks to life expectancy values. All data points are adjusted for age, race, household size, marital status, employment status, and occupation. A similar relationship is seen for females, but the data are not presented here for simplicity.)

Perhaps the most convincing evidence to support the notion that social standing matters comes from animal studies. Primates low in social standing, measured in part by the size of the animals, have higher levels of cholesterol and higher levels of stress mediators (Sapolsky 2005). When a dominant male is removed, the subordinate's laboratory tests improve. Likewise, when a dominant male is put among still larger males, his laboratory tests deteriorate. Such findings are, of course, only consistent among stable social hierarchies. Dominant primates whose authority is constantly challenged show opposite results, but most human primate hierarchies (e.g., corporate or government offices) more closely resemble stable nonhuman primate hierarchies than unstable ones.

Likewise, when primates are randomly assigned to receive an inoculation of a placebo saline solution or a solution containing the cold virus, susceptibility to the common cold is greater in lower ranking primates (both human and nonhuman) than higher-ranking primates (Cohen 1999). In this study by Cohen, lower-ranking primates were more likely to develop cold symptoms, which were confirmed by blood tests. Those higher-ranking animals and humans who did develop symptoms tended to produce less mucous and had a shorter duration of cold symptoms. These data provide powerful support for correlational studies, which show that persons who perceive themselves to be of lower social status show higher levels of stress hormones and immune dysfunction (Goodman et al. 2005; Adler et al. 2000).

Of course, it must be noted that subjects in these cold studies were randomized on the basis of exposure to the virus rather

than social status. Thus, it is possible that social status (observed in animals or self-reported perceived social status in humans) was confounded by some other variable. In fact, although it is implied in these studies that social status affects health by acting as a stressor, it is unclear whether this stressor is different in quality or physiological effects from other stressors, such as the inability to pay the rent (Cohen, Doyle, and Skoner 1999). Moreover, it is difficult to disentangle social status from other social effects, such as sociability or social network size (Cohen et al. 2003).

These distinctions may not be relevant to education policy, but the overall implications of the research certainly are. For instance, these findings beg the question, "Social standing relative to what?" All of human society is hierarchical, after all, and the top and bottom of the hierarchy may be better delimited by those in one's immediate environment than by the relative social distance between the wealthiest and poorest members of society (Mechanic 2002; Deaton 2002).

If absolute social inequality matters, improving the number of high school graduates could positively affect public health by skewing the distribution of social credentials toward higher education. There will always be high school dropouts as a reference point for having the lowest social status. If social standing is measured relative to one's peers, however, increasing the number of high school graduates could theoretically be either helpful or harmful. For instance, it is difficult to weigh the net benefits of the freshly minted high school graduate against the losses in social status suffered by his or her peers in the community who did not graduate.

Another logical question that arises in response to the linkage between educational attainment and social status is whether the health effects of low social status, childhood stress, or other psychosocial factors are reversible (Goodman et al. 2005). Certainly, there is good evidence that, for the population as a whole, both childhood and adult social factors play an important role in determining health and longevity (Pensola and Martikainen 2003). However, there is likely a subset of children who are exposed to such harsh social conditions that they cannot overcome their social deprivations.

SOCIAL DEPRIVATION

For children with poorly educated parents, the effects of social deprivation manifest early in life. Harlow's classical work on animal bonding, along with subsequent studies, suggests that an individual's psychosocial troubles can begin with poor parenting (Higley et al. 1993; Harlow and Suomi 1971). For instance, socially deprived monkeys tend to be more hostile and have predilections that lead them toward risk-taking and alcohol consumption in experimental situations. These problems show a dose-response effect, with monkeys raised by a furry stuffed doll that administers milk faring better than those raised by a similarly equipped surrogate mother made of chicken wire.

These problems can begin before birth; behavioral problems are also observed among the offspring of monkeys stressed by prolonged physical restraint during pregnancy (Clarke and Schneider 1997). Likewise, humans raised in busy orphanages with less social bonding tend to make few friends and exhibit more social pathologies. Nicolae Ceausescu famously relied on orphans to supply the Romanian secret police because they had few social contacts, and he judged that they would be able to kill without remorse.

From childhood to death, persons born into poverty face a series of psychological and social obstacles that often include neglect, abuse, failing schools, bullying, low graduation rates, low wages, and often multiple jobs (Chen, Matthews, and Boyce 2002; Ross and Wu 1996). This does not portend a healthy psychological milieu in which children or adults can thrive (Wilkinson 1999). Educational and economic deprivations such as these are associated with a cascade of biochemical mediators in childhood, which may lead to cardiovascular disease later in life (Goodman, Slap, and Huang 2003). Many childhood events, such as exposure to lead paint and abuse, are largely irreversible and simultaneously affect cognitive development and health (De Bellis et al. 1999; Canfield et al. 2003). Lead paint has long been known to be a neurotoxin, and even small quantities of lead cause IQ deficits. Likewise, children who suffer from physical and emotional abuse have been shown to have smaller brain structures than children who do not (De Bellis et al. 1999).

It is possible that weaker social networks are merely markers for childhood deprivations and that such traumas are the primary reason that weak social networks are associated with poor health. However, it is likely that poor social networks are also independent risk factors for poor health (Berkman et al. 2000).

Social deprivations and their associated feelings of anger or hostility, like other psychosocial stressors, are associated with higher rates of cardiovascular disease, infectious disease, lung disease, and diabetes (Cassel 1976; Wilkinson 1999; Kubzansky et al. 2001; Yan et al. 2003; Cohen 1999). Though speculative, risk-taking and anger may also partially explain higher mortality; persons of lower educational attainment sustain significantly more injuries and display higher rates of smoking and drinking (Lantz et al. 1998; Smith, Shipley, and Rose 1990; Pastor et al. 2002).

A relevant policy question, then, is whether a school intervention or other social programs can break an intergenerational cycle of low educational attainment and social deprivation. There is some evidence that this is the case, at least with respect to

proxy measures of cognitive ability and health, such as IQ scores, criminality, child rearing, and drug use (Schweinhart 2004; Reynolds et al. 2001). Perhaps the strongest positive evidence is provided by the High Scope/Perry preschool program evaluation. This experiment randomly assigned 123 low-income African American children to either receive a high-quality preschool education or no intervention. By age five, 67% of the children in the intervention group, relative to just 28% of the children in the control group, had a measured IQ greater than 90. By age 40, those in the intervention group were a third less likely to have multiple arrests and were a third more likely to have earnings over $20,000 per year. Relative to those who did not receive the intervention, males who received the intervention were nearly twice as likely to raise their own children, less likely to use drugs, and more likely to report satisfactory relationships with their children.

Likewise, one nonrandomized age-matched trial of 1,539 children showed decreased arrest rates at age 20 among those children assigned to a prekindergarten (plus family interventions and health services) intervention group relative to children receiving a less intensive preschool intervention (Reynolds et al. 2001). Although the rates of social pathology were reduced in both instances, it is unknown whether the benefits of these preschool interventions extended to other health measures.

Finally, there is evidence that other social interventions reduce social pathology as well. In one natural experiment, Costello et al. (2003) examined the effects of a casino opening on social pathology rates among Native Americans on a reservation. The investigators examined the rate of mental health diagnoses among 1,420 children in the community according to income level before and after the casino redistributed large amounts of cash to Native American families registered by the tribe. After eight years of follow-up, results showed that the rate of conduct and oppositional/defiant disorders, such as physical abuse, among the newly rich families fell to levels seen in families who were well-off before the intervention. On the other hand, rates of conduct and oppositional/defiant disorders remained high among those families not receiving the income distributions.

Given that social pathology appears to be highly associated with behavioral risk factors, another speculation is that smoking and alcohol consumption may have also been lower among those receiving the education or social interventions mentioned compared with those who did not (Coombs, Wellisch, and Fawzy 1985). The adoption of healthy behaviors may be facilitated by optimism and inhibited by anger, hostility, or fatalism (Kubzansky et al. 2001; Yan et al. 2003). Therefore, to the extent that education reduces social pathology, it may lower emotional barriers to quitting smoking, eating healthier foods, or exercising. These questions were not, however, pursued.

BEHAVIORIAL RISK FACTORS

The combined effects of smoking, eating poorly, and lack of exercise are thought to explain 12%–30% of the association between educational attainment (or other socioeconomic risk factors) and mortality (Lantz et al. 1998; Smith, Shipley, and Rose 1990). About 44% of white males without a high school diploma and 32% of white males with a high school diploma (but no higher degree) are self-reported smokers (Pastor et al. 2002). Although the overall prevalence of smoking is lower among Hispanics, African Americans, and females, the percent reduction in smoking is similar across these populations among those who have a high school diploma relative to those who do not.

As Mechanic (2002) notes, most people, regardless of their level of educational attainment, know that smoking, drinking, and eating greasy food is not healthy. However, one might speculate that, in addition to reducing fatalism, education could increase the total exposure to preventive health information, thus normalizing it. Education might similarly improve cognitive appraisal of this information. For instance, people may be more likely to eat healthy food if they understand that cholesterol and saturated fat clog arteries and can visualize these arteries instead of just having a vague, abstract notion that these substances are harmful. Better-educated persons might also be better equipped to balance health information against messages from the fast food, tobacco, and alcohol industries.

Because increased educational attainment improves income, it may also exert positive effects on health behavior via upward mobility to neighborhoods where healthier foods are available in stores and their consumption is normative (Morland et al. 2002). Given lower rates of crime, wealthier neighborhoods also afford more opportunity for exercise. One large randomized, controlled multisite trial evaluated the health, crime, and other social effects of vouchers for housing that allowed recipients to move out of low-income neighborhoods (Kling et al. 2004). Five years after randomization, those who received the vouchers had significant reductions in obesity and improvements in measures of mental health relative to those randomized to receive no vouchers.

GENETIC RISK FACTORS

Individual characteristics are determined by a combination of genetic predispositions and environmental influences. Genetic predispositions that influence health behaviors may also influence success in school. For instance, twin studies suggest that a person’s ability to cope with stress is partly determined by genetic factors; further, the probability of having a life event perceived as stressful in the first place has also been partly linked to genetic factors (Wang et al. 2005).

Environment also plays a role; although twin siblings reared apart show some concordance on measures of IQ and income, twins who fall on hard times socioeconomically have been found to have more cardiovascular risk factors (e.g., higher blood pressure and cholesterol levels) than identical siblings not raised in poverty (Lichtenstein and Pedersen 1997; Plassman et al. 1995; Krieger et al. 2005). Twin studies strongly suggest that measures of health status and cognitive ability are partially environmentally determined. Unfortunately, such studies cannot produce a reliable estimate of the contributions of genes, environmental factors, or the interaction of the two (Boomsma, Busjahn, and Peltonen 2002).

First, twins reared apart are nonetheless exposed to similar environments in utero and sometimes the same environment in childhood, both of which are strong predictors of adult mortality by SES and social pathology (Chen, Matthews, and Boyce 2002). Therefore, upon comparing adult twins reared apart, it is difficult to disentangle genetic effects from early environmental effects. For instance, fetal alcohol and lead paint exposure are both risk factors for delayed development, and both are strongly associated with parental educational attainment (Streissguth et al. 2004).

Second, virtually all the conceivable genetic contribution to the education-health gradient described here can be attributed to a large number of genetic foci. The expression of these genes, in turn, is highly influenced by environmental factors in a dose-response manner. Consider a hypothetical experiment in which a group of fetuses is randomized to ideal childhood conditions, and another group is randomized to a harsh childhood. Among children with loving parents, excellent schools, and ideal nutritional intake, genetics will play a major role in determining which children do well in school and live a long and prosperous life. Among children with abusive parents and peers who are exposed to drugs, unhealthy food, lead paint, and bad schools, the degree of exposure to positive environmental conditions (such as preschool programs or mentors) will be a major determinant of their longevity and prosperity. In these latter situations, genetics play a small role in explaining educational attainment.

In a groundbreaking twin study, Turkheimer et al. (2003) set out to test this hypothetical scenario using biometric analyses. They examined the contribution of (1) genetics, (2) shared environment, and (3) nonshared environment on IQ as measured by the Wechsler Intelligence Scale for Children administered at age seven. Their models examined the interaction of these three characteristics and SES using the National Collaborative Perinatal Project data set. This prospective study included 48,197 pregnant women and their 59,397 children. Their measure of SES was based on a linearly combined measure of parental educational attainment, occupation, and income. Their intent was to measure the interaction between genotype and environmental conditions as measured by SES. They accounted for the possibility that SES is genetically determined by including the main effect of the moderating variable in their model. They found that, among poor families, IQ was determined almost entirely by the childhood environment. Among wealthy families, on the other hand, genes were almost entirely predictive of IQ. Of course, this study's conclusions hinge on the assumption that SES is an adequate proxy measure of a harsh versus an ideal childhood environment (Turkheimer et al. 2003).

In another compelling study, Korean adoptees—who were essentially randomly assigned to families of varying SES—were studied later in life (Sacerdote 2004). Education and health outcomes were then compared with those of their new parents and siblings. Clearly, Korean children adopted into non-Asian families tend to stand out in social situations, and this likely exerts an influence on their development. Nonetheless, adoptees assigned to better-educated parents do better in school, go further, and are healthier by some measures than adoptees assigned to parents with less education. Most strikingly, adoptees were just as likely as their nonadopted siblings to take up smoking and drinking, providing strong evidence that these behavioral risk factors might not be inherited.

In sum, both genetic factors and environmental factors influence characteristics of individuals that are critical in determining both SES and health status later in life. Study design problems make it difficult to quantify the effect of genetics on health. However, both current scientific evidence and logic suggest that genetic factors will be stronger determinants of health among more affluent children and much weaker determinants among poor children. Thus, intellectual capacity will likely be optimized when educational interventions are targeted toward low-income families and schools in low-income communities. Clearly, to maximize environmental variables, it is also important to optimize other aspects of childhood and adult environmental conditions, such as access to healthy foods, good housing, safe transportation, and medical care.

HEALTH INSURANCE

Of these childhood environmental characteristics, medical care for children has received almost as much attention as education interventions of late; Massachusetts, Pennsylvania, and California have all planned health insurance reforms that are primarily driven by concerns surrounding child health. Nonetheless, although it is clear that families with poorly educated parents simultaneously lack health insurance and suffer from poor health outcomes, there is as of yet limited evidence showing that the possession of health insurance is causally linked to improved health status.

Among 18–64-year-olds, 7.3% of persons with at least a bachelor's degree lack health insurance, compared with 27.6% of those without a high school diploma (Muennig and Woolf 2007). This lower rate of insurance among those with less education is

probably attributable to the fact that less educated persons tend to access lower-quality jobs, which tend not to offer health insurance.

Access to medical care increases access to medications and treatments that are known to reduce morbidity and mortality (Hadley 2003). Of those diseases prominent in the education gradient, access to medications that reduce cholesterol, blood pressure, and diabetes may be most important (see Figure 1). The best evidence of the efficacy of health insurance to date suggests that insurance may improve health *exclusively* through these very modalities.

In the 1982 Rand Health Insurance Experiment, 3,958 healthy but uninsured subjects were randomly assigned to either receive a premium health insurance policy or a policy that required financial contributions on the part of patients before they could receive care (Brook et al. 1983). Subjects were assigned to their insurance plans for three or five years and then evaluated for health outcomes, including mortality. These authors found that mandatory patient contributions reduced healthcare use relative to those who had no such requirement, but this barrier to care only produced a calculated 10% increased risk of death among high-risk subjects with hypertension. No changes were found in other measures of health outside an improvement in vision through corrective lenses.

This study is somewhat dated, however, and over the past 24 years, a wide range of medications that prevent heart disease, infections, and cancer have become available (e.g., statins, vaccines, and smoking cessation technologies, respectively). Because they are expensive, these medications are almost exclusively used by persons with health insurance. Recent correlational analyses show a 25%–67% reduction in mortality among the uninsured (Muennig, Franks, and Gold 2005). However, this reduction in mortality might be explained by model endogeneity or other factors associated with insurance, such as the economic protection and peace of mind that health insurance affords, rather than the benefits associated with receiving medical attention itself (Ross and Mirowsky 2000).

Given that preventive modalities, such as antihypertensive medications, seem to matter most and that these modalities reduce the incidence of disease almost exclusively in the education-health gradient (Figure 1), health insurance seems a logical contender for an explanatory variable in the education-health gradient. If accurate, this 25%–66% reduction in mortality would account, at most, for three to nine months of the roughly six-to-nine-year difference in life expectancy between those with a high school diploma and those without (Muennig, Franks, and Gold 2005; Wong et al. 2002).

In sum, in industrialized nations, healthcare likely plays a small but significant and growing role in reducing health disparities by SES (Muennig, Franks, and Gold 2005). Although health disparities by educational attainment are smaller in nations that offer health insurance, these countries also tend to offer more social services like child care and parental leave, making it difficult to disentangle the effect of health insurance from the effects of other social programs on reducing educational disparities in health (Marmot and Wilkinson 2006). Moreover, increasing health insurance would be significantly less cost-effective than implementing effective education interventions, such as small class sizes (Muennig and Woolf 2007; Muennig, Franks, and Gold 2005).

Nonetheless, universal health insurance coverage may be one potential policy approach to addressing the health-education gradient. The findings of the Rand Health Insurance Experiment notwithstanding, universal healthcare may, in and of itself, prove to be an effective education intervention. After all, it is difficult to for children to learn if they are sick. Moreover, although health may improve educational attainment, increasing educational attainment may improve the use of healthcare among those who are already insured.

ENHANCED COGNITIVE ABILITY

Even among those with access to medical care, knowledge of screening test availability is predictive of the use of such tests (Brown et al. 1990; Davis et al. 1996; Glied and Lleras-Muney 2003). Those with more education are also more likely to be compliant with their medications and otherwise manage their disease better than those with less education (Gallagher, Viscoli, and Horwitz 1993; Goldman and Smith 2002). In addition to increasing the uptake and proper use of health technologies, improved cognition also would likely reduce day-to-day errors that can lead to automotive or household accidents (Gottfredson 2004). In addition to increasing one's access to health information and improving one's comprehension of the information, cognitive ability may also influence the so-called future discount rate (i.e., a value assigned to consequences and events far in a person's future, such as retirement funds or smoking-induced lung cancer) (Grossman 2005).

CONCLUSIONS

There is evidence to suggest that lower educational attainment is associated with a higher-stress lifestyle. The lower social standing and weaker social support networks available to less educated persons exacerbate this stress. Social stressors both partially originate from, and are compounded by, a higher likelihood of exposure to social pathology. They are also exacerbated by lower rates of health insurance, weaker cognitive coping mechanisms, and an inability to fully understand medical diagnoses

or treatments. One might hypothesize that these high rates of stress, poor coping mechanisms, and social pathology among those with less education arise as a direct result of poorly educated persons' inability to obtain a well-paying job that offers health insurance and other benefits.

It may be further hypothesized that higher rates of stress and abuse compounded by weaker social networks and poor cognitive coping mechanisms serve as explanatory variables for the higher rates of fatalism, anger, and hostility among those with less educational attainment. What have been called "negative emotional styles" may contribute to higher rates of smoking, drinking, and eating poorly among those with less education (Yan et al. 2003). The uptake of behavioral risk factors may further be facilitated by poor coping skills and a weaker understanding of how poor diet and exercise harm health. It is probably not coincidental that the very psychosocial factors linked to educational attainment are also major medical risk factors for cardiovascular disease, cancer, infection, injury, lung disease, and diabetes—the diseases contributing most to the six to nine years of life expectancy separating those with a high school diploma from those without.

That a high school diploma is the minimum social credential required to obtain a good job is not generally disputed. Nor is it radical to suggest that a living wage is needed to obtain adequate housing, medical care, and nourishment, or that the social environment is a major contributor to psychosocial stressors. What is new is our understanding of the linkages between psychosocial stressors and biopsychological disruptions that may both directly cause disease and increase risk-taking behavior. In this article, I attempted to knit this new scientific knowledge together with existing understandings of education-related risk factors. My aim was to create an overall picture of the hypothetical linkages between education and health.

Certainly, the linkages I propose here are as complex as they are intuitive. Many of the linkages have not been shown to be causal, and the gestalt I provide should be considered hypothetical. Indeed, although the evidence linking educational attainment to health overall suggests a causal relationship between the two, causality remains unproved. Nevertheless, the evidence is strong enough to build a case for adding health to the long list of the expected benefits of education interventions.

One critical remaining question is how we might estimate the magnitude of benefits associated with specific education interventions, such as preschool programs or small class sizes. These data are partly missing because education researchers have historically omitted relevant health outcomes from education studies and because health researchers often fail to disaggregate the effects of educational attainment on measured health outcomes.

A bigger problem is that each education intervention is unique. Generic outcomes, such as lives saved per additional high school graduate produced by an intervention, may not be easily generalized to specific interventions, which differ with respect to the cognitive skills they confer and the demographic characteristics of the students they affect. For instance, in a large, randomized controlled trial of small class size, low-income students responded more vigorously to education interventions than other students (Finn, Gerber, and Achilles 2001). This may partially reflect the large influence of environmental variables on education outcomes in this group (Turkheimer et al. 2003).

Still, estimates of health benefits will likely produce an impact on the overall predicted costs and benefits of expensive education interventions (Muennig and Woolf 2007). For instance, the value of a quality-adjusted year of life (one year of life lived in perfect health) has been estimated to be upwards of $428,000 (Hirth et al. 2000). A final consideration is that a legislator may be more compelled to vote for a law that is lifesaving than one made in the name of economic benefits alone.

Recognizing the importance of health to policy making, the United Kingdom has embarked on a program of education and social reform in the name of their predicted health benefits (Roberts 2000). This program, Sure Start, combines parental counseling and social services that are intuitively linked, such as daycare and job training, with a preschool intervention.

In focusing on social conditions rather than individual risk factors for disease, Sure Start tests a contentious idea in public health, economics, and sociological literatures. The debate centers on whether policy should focus on targeting risk factors for disease, such as smoking, or targeting the social conditions that give rise to these risk factors in the first place, such as poverty arising from failing schools (Deaton 2002; Mechanic 2002).

With respect to the former approach, some have suggested that legislative approaches can bypass the cognitive barriers associated with low educational attainment (Link and Phelan 1995). For instance, if people do not respond to messages that lead paint is bad because they do not have the knowledge, resources, or will to act on this information, then mandatory lead abatement programs are preferable to lead education campaigns. Certainly, lead-poisoned children are less likely to rank among the responders to preschool programs or small class sizes. Moreover, lead abatement is itself a type of educational intervention. But most risk-factor-based approaches, such as improving access to medications, treat the symptoms of poor cognition rather than the cause: namely, poor schooling.

Given that there is clear evidence that educational interventions are most effective for low-income students (Finn, Gerber, and Achilles 2001), many such students are capable and ready to succeed but just need proper support. In addition to adequate

schooling, others will need more intensive psychosocial interventions so that their social and educational opportunities better match those of their more privileged peers. Certainly, privileged students will always have access to greater resources than students from low-income backgrounds. But if lack of access to knowledge, prestige, power, and social connections is the root cause of the social conditions that first caught Hippocrates's attention over 2,000 years ago, it may well make more sense to heed Mann's advice and focus our efforts on curing the disease by investing in education rather than treating the costly symptoms later on.

ACKNOWLEDGMENTS

I would like to thank the W. T. Grant Foundation for providing financial support for this study. I would also like to thank Jessica Simon for outstanding research assistance and Clive Belfield, Cecilia Rouse, and Hank Levin for their insight on study design. Thanks to Mitch Wong, Bruce Link, Celina Su, Steven Woolf, and Marilyn Sinkwicz for their thoughts on the causal linkages between education and health.

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