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. They are also intertwined. Not only is health critical for learning, but also education appears to be a major determinant of health.

This article begins with a brief 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.

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. Some 2300 years after Hippocrates, Horace Mann proposed that education might serve as a tool for repairing social ills. He observed that education is “the great equalizer,” providing people with the knowledge and technical skills needed to survive. While Mann was referring to income and social prestige, education also appears to be an equalizing force in health.

Evidence of a causal relationship draws upon a single, randomized, controlled trial, instrumental variable analyses, and a large number of prospective studies. The lone, randomized, controlled trial with long-term follow-up is the Perry Preschool Program, a study consisting of 123 students randomized to 2.5 h of preschool at ages 3 and 4 years or no intervention. Although the study was small,
children randomized to the preschool intervention were much more likely to report better health status and fewer behavioral risk factors for health (such as seatbelt use or smoking). By age 40, two of the children in the experimental group had died and five in the control group had died.

Most evidence arises from natural experiments, which primarily rely on 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 (2005) 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.

This same methodology has since been replicated in a number of other countries with similar results. It was also later explored using a larger census sample as well as using data from the Survey of Income and Program Participation (SIPP). This subsequent 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. Nonetheless, using the SIPP, a strong causal association between educational attainment and self-reported health was also found.

Beyond the handful of domestic and international natural experiments and randomized studies with proximal outcome measures, there are few studies specifically examining the causal association between education and health. 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. 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, and fourth, the primary causes of death between 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.

The education–health association has been explored by epidemiologists, sociologists, economists, urban planners, social psychologists, and even neuroimmunologists. Once this work is 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 6–9-year gap in life expectancy between those with and without a high school diploma (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. However, the interdisciplinary research helps us better understand and contextualize the linkages between these factors and educational attainment as well.

The following sections set the stage by discussing the relationship between poor health and poor education outcomes, then explore the evidence related to the six main pathways potentially linking education to health.

Health, Education, and Poverty

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. 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 both improve health and educational attainment in children, but we would point to the lead-abatement program as the causative agent for any observed improvements in both health and education, not to the additional education received by the children in the abatement program. On the other hand, if a child received an educational

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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, which was 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. Wealthier, more educated persons indeed have stress in their lives. However, while 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. 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. 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.

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. 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.

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 (McEwen, 1998). 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. 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 (Muennig, 2007). 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.

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 and possibly increases one’s risk of cancer. The first study in this area recruited unstressed subjects and compared them to subjects with a chronically ill child to ensure large differentials in self-reported stress (Epel et al., 2004). 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)). While the outcome measures they included in the second study were different, this group of researchers effectively confirmed the finding that stress increases cell aging. 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. While 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. This damage is thought to occur when the acute stress response causes the physiological release of oxidative chemicals into the bloodstream. A subanalysis 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.

**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. Differences in relative standing—which 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. This hypothesis was initially drawn from heavily confounded ecological studies assessing the effects of income inequality on mortality and crime, 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. 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. 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).

These findings are corroborated by animal studies. Primates, low in social standing, measured in part by the size of the animals and dominance behavior, have higher levels of cholesterol and higher levels of stress mediators. 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.

The implications of social-standing research for education policy are unclear. All of human society is hierarchical, 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.
Science and Medicine

mortality: The National Longitudinal Mortality Study.

A comparison of the relationships of education and income with occupation. A similar relationship is seen for females, but the data are not presented here for simplicity. Adapted from Backlund, E., Sorlie, P. D. and Johnson, N. J. (1999). Mortality risks were converted to life expectancy values. All data points are adjusted for age, race, household size, marital status, employment status, and education. A comparison of the relationships of education and income with mortality: The National Longitudinal Mortality Study. Social Science and Medicine 49, 1373–1384.

Social Deprivation

For children with poorly educated parents, the effects of social deprivation manifest early in life. Harlow’s (1964) classical work on animal bonding suggests that an individual’s psychosocial troubles can begin with poor parenting. 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 fairing 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. 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 correctly 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. This does not portend a healthy psychological milieu in which children or adults can thrive. 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. Many childhood events, such as exposure to lead paint and abuse, are largely irreversible and simultaneously affect cognitive development and health. Lead paint has long been known to be a neurotoxin, and even small quantities of lead cause intelligence quotient (IQ) deficits. Likewise, children who suffer from physical and emotional abuse have been shown to have smaller brain structures than children who do not.

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

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. Although 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. Data from the aforementioned Perry Preschool Program randomized controlled trial (along with many other quasi-experimental studies) confirms much higher rates of crime, injury, and death among those receiving less education.

The Perry Preschool Program randomized experiment suggests that these early deprivations are somewhat reversible with early intervention. By age 5, 67% of the children in the experimental 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 US$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. Thus, the intervention appeared to modulate the intergenerational transfer of social pathology.

Likewise, one nonrandomized age-matched trial of 1359 children showed decreased arrest rates at age 20 years among those children assigned to a prekindergarten (plus family interventions and health services) intervention group relative to children receiving a less-intensive preschool intervention. While 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

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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.

Change in life expectancy with educational attainment relative to years of education completed.
a casino opening on social pathology rates among Native American on a reservation. The investigators examined the rate of mental health diagnoses among 1420 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 8 years of follow-up, results showed the rate of conduct and oppositional/defiant disorders, such as physical abuse, among the newly rich families fell to levels seen in families that 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.

To the extent that educational attainment improves social networks and reduces social pathology, we would expect education interventions to improve health through reduced negative emotions and improved behavioral risk factors.

**Behavioral Risk Factors**

The combined effects of smoking, eating poorly, and lack of exercise are thought to explain somewhere between 12% and 50% of the association between educational attainment (or other socioeconomic risk factors) and mortality (Lantz et al., 1998). 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. 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.

It has been pointed out that most people, regardless of their level of educational attainment, know that smoking, drinking, and eating greasy food is not healthy (Mechanic, 2002). One might speculate, however, that, in addition to reducing fatalism, education could increase the total exposure to preventative 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.

As increased educational attainment improves income, it may also exert positive effects on health behavior through upward mobility to neighborhoods where healthier foods are available in stores and their consumption is normative. 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. 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.

Finally, prospective examination of the contacts that people make in their lives suggest that improved diet and exercise as well as reduced smoking and obesity rates are contagious. That is, when an individual develops a contact with a peer that does not smoke or drink, he or she is much less likely to smoke or drink (Christakis and Fowler, 2007). Thus, the effects of educational attainment on behavioral risk factors appear to be transmissible to less-educated peers.

**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.

Environment also plays a role; while 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. 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 of the interaction of the two (Boomsma et al., 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. 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.

Second, virtually all of 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 7 years. 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.

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 be 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. Massachusetts, Pennsylvania, and California all have planned health insurance reforms that are primarily driven by concerns surrounding child health. Nonetheless, while 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 bachelors degree lack health insurance, compared with 27.6% of those without a high school diploma. This lower rate of insurance among those with less education is probably attributable to fact that less-educated persons tend to have lower-quality jobs, which usually do not offer health insurance.

Access to medical care increases access to medications and treatments that are known to reduce morbidity and mortality. 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 through these modalities.

In the 1982 Rand health insurance experiment, 3958 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 3 or 5 years, and then evaluated for health outcomes, including mortality. These authors found that mandatory patient contributions reduced healthcare utilization 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 of 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). These medications, being expensive, are almost exclusively used by persons with health insurance. Recent correlational analyses show a 25–67% reduction in mortality among the uninsured (Muennig et al., 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.

Given that preventive modalities, such as antihypertensive medications, seem to matter most, and that these modalities reduce the incidence of disease 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 3–9 months of the roughly 6–9-year difference in life expectancy between those with and without a high school diploma. It should be noted though, that the effects of educational attainment on cognition should synergize with health insurance effects; in addition to improving access to medications, more-educated people are also more likely to comply with prescribed dosages and intervals.

In sum, in industrialized nations, healthcare likely plays a small but significant and growing role in reducing health disparities by SES. While health disparities by educational attainment are smaller in nations that offer health insurance, these countries also tend to offer more social services like childcare 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. Moreover, increasing health insurance would be significantly less cost effective than implementing effective education interventions, such as small class sizes (Muennig and Woolf, 2007).

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 of itself prove to be an effective education intervention. After all, it is difficult to for children to learn if they are sick. Moreover, while health may improve educational attainment, increasing educational attainment may improve the utilization 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. 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. In addition to increasing the uptake and proper use of health technologies, improved cognition also likely reduces day-to-day errors that can lead to automotive or household accidents. 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, that is, a value assigned to consequences and events far in a person’s future, such as retirement funds or smoking-induced lung cancer.

Conclusions

Lower educational attainment likely affects health both through a higher-stress lifestyle and through material deprivation. Social stressors both partially originate from, and are compounded by, a higher likelihood of exposure to social pathology, weaker social support networks, and lower social standing. Likewise, lower rates of health insurance, weaker cognitive coping mechanisms, lower job quality, and an inability to fully understand medical diagnoses or treatments all seem to play a role.

Less explored are the effects of fatalism, anger, and hostility among those with less educational attainment. These negative emotional styles may contribute to higher rates of smoking, drinking, and eating poorly among those with less education. Recent evidence suggests that influences from educated friends and family members also play a large role in behavioral risk factors.

In sum, the 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 6–9 years of life expectancy separating those with and without a high school diploma.

See also: Education Production Functions: Concepts; Family Environment in the Production of Schooling; Human Capital; The External Benefits of Education.

Bibliography


Further Reading


Relevant Websites

http://www.macses.ucsf.edu – MacArthur Network on SES and Health summarizes the seminal works in pertaining to the education-health relationship, and provides information on many of the key researchers in this area.