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The Education Effect on Population Health: A Reassessment

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Within the demographic literature are numerous reports of consistent and significant associations between formal educational attainment and individual health outcomes and risks such as mortality, smoking, drug abuse, and accidents, as well as the contraction of many diseases. The vast majority of these reports conclude that more highly educated individuals are healthier and live longer.1 This association is so prevalent that after an extensive survey of the health literature, Mirowsky and Ross (2003, p. 6) conclude, "education has an enduring, consistent, and growing effect on health."

Macro demographic and development studies also identify a significant positive association between expanding mass education and population health; yet many, especially the earliest ones, did not control for other factors, such as economic development, that may also be linked to better health (see review by Feinstein 1993). As a consequence, it is frequently suggested that the association between expanding education and population health is spurious because of the impact of general social development (e.g., economic growth, quality of health care) on both. Thus, related factors of modernity are often assumed to pave the way for both mass education and healthier populations (e.g., Bloom 2007).

Social epidemiological research also reports extensive negative associations between education and disease, often after statistically controlling for indicators of socioeconomic status (e.g., Mackenbach et al. 2008; Martikainen et al. 2001; Steenland, Healey, and Thun 2002). But although education's causal role in health is recognized in the discipline of demography, overall it remains underappreciated. For example, demographic studies of health either conflate education with socioeconomic status in interpreting findings, or assume that education is only a proxy measure for socioeconomic status. Although educational attainment is associated with adult socioeconomic status, many studies, including those assessed below, suggest that schooling has a substantial effect on health independent of status attainment (Feinstein et al. 2006).

The low appreciation in demography of education's independent role as a causal agent fosters speculation that education is mostly a proxy for other aspects of socioeconomic status at the individual level or of economic development at the aggregate level (Desai and Alva 1998; Basu 1994; Hobcraft 1993; Kunstadter 1995). Consequently, the causal nature of the relationship between educational attainment and individual health, as well as between mass education and population health, has long been debated in demography, even as "universal primary education" (Goal 2) has become a core strategy of the UN's Millennium Development Program and massive development funds continue to target universal access to

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[1]Only the effects of formal education, or schooling, are considered here; informal learning and indigenous knowledge are beyond the scope of this article.

formal schooling throughout the developing world (e.g., Baker, Collins, and Leon 2009; Grossman 1972; Ross and Mirowski 1999; NRC 2005; UN 2009; UNESCO 2008).

The confusion over the causal role of education is likely due to three reasons. First, the educational revolution of the past 150 years was originally interpreted as an outcome, not a cause, of modernization. Second, there has yet to be a systematic assessment of the education effect on health across a full set of methodologically sophisticated studies, with controls for wealth and other dimensions of socioeconomic status. Third, hypotheses about the education effect on health have not considered the central schooling activity of cognitive development.

In addressing these shortcomings, we offer three contributions to illuminate the education effect on health. First, we summarize the dimensions of the worldwide educational revolution and reassess its potential influence on modernity and macro-demographic transitions in developed and developing countries. Second, to establish the independence and robustness of the education effect on individual health, we conduct a meta-analysis of peer-reviewed micro-level studies of the relationship between educational attainment and mortality. Lastly, in light of new multidisciplinary research on the cognitive impact of schooling, we propose a hypothesis about how education may be a powerful determinant of health and longevity.

Reassessing the mass education effect on population health

A large set of multidisciplinary studies from sociological, historical, and economic research examining educational development over the past 150 years yields three central findings about the expansion of formal education and its effect on modern society (Meyer 1977). First, it is likely that the unique event of widening access to primary and secondary education was one cause of modernity; second, the effects of schooling can be substantial, yet are underestimated in studies of highly educated societies; third, the timing and influence of the spread of education suggest it was an important cause of the demographic transition.

The educational revolution as a cause of modernity

The so-called educational revolution is marked by persistent worldwide growth in formal schooling both in terms of increasing school enrollment rates and in terms of upward expansion of formal educational attainment. Yet, when scholars identify the social forces responsible for the development of modern society, they rarely include the remarkable collective activity of schooling large proportions of children for long periods of time. Other transforming forces—industrial production, technology, science and medicine, improved nutrition, capitalism, the rise of the nation-state and democratic politics, large-scale warfare, decline of religion, and rise of rationalized bureaucracies— receive most of the credit. There is, however, growing evidence that the educational revolution played, and continues to play, a key role in the development of many features of modern societies. The evidence centers around 1) the demographic size and temporal dynamics of the educational revolution; 2) evidence on what did, and did not, initiate mass education; 3) results from econometric modeling of the causal impact of educational expansion on national economic development across the twentieth century; and 4) the intensive adoption of a common model of formal education capable of a uniform impact worldwide. We examine each of these in turn.

Size and speed of the educational revolution—Because the historical spread of formal education was so extensive and arose temporally with economic development and growing social complexity, it is unlikely to have been a result of these factors. At the beginning of the twenty-first century, formal education throughout the world is profoundly influential in terms of its recent growth, its claim to people's time and effort, and its impact

on their lives. What is demographically salient about the educational revolution has been both its relative newness to human society and its speed of growth. The enrollment of children and youth into primary, secondary, and post-secondary education over the past 200 years worldwide is, in historical terms, a recent social phenomenon. Once started, the rate of growth for each level of schooling rapidly becomes significant and sustained. The increasingly common practice of going to school and attending for a considerable number of years represents a new and massive change in human behavior. During the last 60 years, populations in low-income countries underwent a transition from virtually no access to schooling to wide access to primary and even secondary education, so that now an estimated 80 percent of all humans aged 15 or older have been schooled enough to both read and write a short statement about their lives, a population characteristic that would have been unthinkable just 50 years ago (UNESCO 2003).

The steady growth in primary school enrollment throughout the second half of the nineteenth century and into the first few decades of the twentieth century led to an exponential climb worldwide from the 1940s on. As primary schooling reached large proportions of the world's population, enrollment in secondary schooling turned sharply upward in the 1960s. And then in the early 1970s enrollment in higher education began to grow. Mass higher education is the next wave of the educational revolution. For example, about half a million students, or just 1 percent of the youth age-cohort, were enrolled in higher education worldwide in 1900; yet a century later approximately 100 million were enrolled, representing 20 percent of the college-age cohort (Schofer and Meyer 2005). In a number of countries, enrollment rates now exceed 50 percent and in some have reached 80 percent of the age-cohort attending higher education (UNESCO 2004).

Causes of educational expansion—The spread of education over the past 150 years is generally unrelated to specific economic and social characteristics of nations. Contrary to the popular assumption that industrialization and urbanization precede expansion of primary and secondary schooling, extensive statistical analysis concludes that there is a lack of consistent historical ordering among these trends. Thus these two modernizing forces are unlikely to have been unidirectional causes of the educational revolution (e.g., Benavot 1992; Meyer, Ramirez, and Soysal 1992; Meyer et al. 1979). The same is true for political democratization, another factor often assumed to have caused the education revolution. Instead, these analyses show that the educational revolution, economic and urban development, and political democratization are co-determinants of one another, and the process occurs more on a global than a national level (Baker 1999).

For example, three main findings from a random effects GLS regression estimate modeling national characteristics and higher education enrollments over the twentieth century illustrate the interconnectedness of forces behind modern development (Schofer and Meyer 2005). First, as noted above, the results indicate that the wealth of a nation (i.e., its GDP) and degree of urbanization are only moderately related to growth in higher education over the century (see also Windolf 1997). Obviously it requires national resources and a certain level of development to expand higher education, but the effect is not overwhelming nor is it the whole story. Second, over the middle third of the last century, higher education enrollment expanded the fastest in countries most connected to the "world society," meaning a combination of factors that make up modern society including scientization of culture, rational national planning, democratization, and the globalized polity of multilateral agencies such as the United Nations and international nongovernmental agencies (Drori, Meyer, and Hwang 2006; Schofer 2004). Countries that embraced these cultural values and incorporated them into their national institutions experienced the most rapid expansion of higher education. Lastly, by 1960, enrollment growth rates began to converge in almost all countries, indicating that educational expansion leads to additional expansion, much like the

endogenous intensification of other trends of modernity such as capitalism and democratic politics.

Impact of educational expansion on national economic development— Econometric analysis of historical macro-economic development of several countries over the twentieth century shows that a significant force in economic development is the upward-spiraling interplay between rising human (chiefly educational) capital of the working population and technological change (Goldin and Katz 1998; Rubinson 1986). Historical studies also indicate that greater use of "pervasive skill-biased" technologies (e.g., continuous-process and batch-production technologies) was caused by the extensive growth in secondary education enrollment rates in the United States during the first half of the twentieth century. This complementary relationship between expanded education and economic development will likely continue in economies throughout the twenty-first century (e.g., Baker 2009; Berman, Bound, and Machin 1998; Murname and Levy 1996; Autor, Katz, and Krueger 1998).

Common model of education—There is considerable evidence that the form and organization of education have steadily converged on a worldwide model. Extensive comparative analyses in all regions of the world find a steady trend toward common educational practice in terms of organization, curricular content, teaching, learning, and values about education and its social role (e.g., Inkeles 1974; Baker and LeTendre 2005). So too, historical analysis suggests that the basic epistemological model and cultural ideas that sparked the expansion of primary and secondary schooling grew out of the spread of Western-style universities throughout Europe and North America over at least the last three centuries (Meyer, Ramirez, and Soysal 1992; Benavot and Riddle 1988). This shared model of education as a social institution creates the potential for a relatively uniform educational effect on development worldwide, similar to the universalizing effects of large-scale capitalism and widespread constitutional democracy that also have converged in form over time.

Underestimation of education effects in fully schooled societies

A second factor behind the under-appreciation of the education effect on health is that a substantial proportion of studies in highly educated countries such as the United States and countries in Western Europe and parts of Asia tend to underestimate the full impact of schooling. This occurs for two reasons.

First, because educational attainment has saturated populations in developed countries, studies using samples from these populations are restricted by limited variation in exposure to schooling, which continues to decrease over time. Currently, for example, the median educational attainment just exceeds the completion of secondary school (12+ years) in developed countries such as Japan, Sweden, and the US (OECD 2008). Furthermore, variation or inequality in educational attainment declined by 46 percent worldwide from 1980 to 2000, double the rate of decline in income and life expectancy inequality (Goesling and Baker 2008; see also Neumayer 2003).2 While there is no evidence that the education effect on health is other than linear, it is possible that the many studies of education and health done on samples from countries with high educational attainment and limited educational variation may not capture the more robust effects between the extremes of unschooled and moderately schooled populations, such as those reviewed below.

[2]Declining variation in life expectancy is found worldwide except for sub-Saharan Africa, and studies from that region were omitted from the meta-analysis below (see Goesling and Baker 2008 for details).

Second and related, there is a widely held misperception that weak between-school effects on educational achievement indicate a general lack of an education effect. The misunderstanding stems from blurring the considerable distinction between "school effects" and "effects of schooling" (Baker and LeTendre 2005). The former, called a "between-school effect," refers to the effect on achievement of going to one school versus another, and is frequently studied with a focus on differences in instructional resources. Starting with the Coleman Report in the US (1966) and the Plowden Report in the UK (1967), a large research literature reports only modest between-school effects (and relatively stronger between-family socioeconomic status effects). Also, the large between-school effects once found in poorer countries, then with greater variation in school quality, have recently declined (Baker, Goesling, and LeTendre 2002). Given the global trend toward common operation of schooling, including political pressure to reduce between-school differences in resources described above, weak between-school effects are to be expected. But to conclude from weak between-school effects that any effect of schooling is also weak is technically inaccurate and belies the evidence of the robust effects of schooling.

Contrary to weak between-school effects, there is considerable evidence of robust effects of schooling, or in other words the influence of exposure to different levels of education, the most extreme being unschooled versus schooled. A set of studies reports consistent significant and sizable education effects on an array of health behaviors and attitudes of adults after controlling for income (e.g., Baker, Leon, Collins 2010; Goesling 2007; Oreopoulos and Salvanes 2009; Peters et al. 2010).

Timing of the educational revolution and the first demographic transition

A third reason for the under-appreciation of the effect of the educational revolution on population health is, with a few notable exceptions, its limited inclusion in scholarship on independent factors behind historical demographic transitions (e.g., Caldwell 1979, 1989).

The first demographic transition (hereafter, FDT), referring to the initial extensive decline in crude death rates and crude birth rates among populations in Western Europe and North America beginning in the nineteenth century, is considered a fundamental transformation of population dynamics that heralded a new kind of society. Across demographic, economic, and medical accounts of the cause of the FDT, materialist arguments—improving production and technological advances—reign supreme (Caldwell 1979; McKeown 1976). Various aspects of the scientific and industrial revolutions are hypothesized as having created the material conditions necessary for the FDT. But at the same time, it is often noted that the timing implied by materialist arguments is not optimal for causal logic. The industrial revolution began considerably before the onset of the FDT, and significant advances in scientifically derived medical technology occurred considerably after its onset (e.g., Caldwell 2006). By contrast, as illustrated in Figure 1, the beginning and intensification of the FDT closely parallels the beginning and expansion of the educational revolution.

Problematically for the materialist causal argument, the first stages of the industrial revolution in Western Europe began in the eighteenth century and, by the mid-nineteenth century, had already exerted a significant impact on increased production of many everyday commodities, too early to be a cause of the FDT (Hobsbawm 1983). It has also been difficult for scholars to reconcile cross-national variation in economic growth with the near universal trends of the FDT in the region. For example, Pritchett and Summers (1996) posit that improvements in average household wealth were responsible for the reduction in mortality during this transition. Yet real income in England during the 1850s was almost 50 percent above its level in France, but this did not give the English population a comparative advantage in health during this period (Caldwell 2006).

The same is true for materialist arguments about science and medical technology. Some medical breakthroughs in the eighteenth and nineteenth centuries, including smallpox vaccination and the germ theory of disease, contributed to the decline in mortality; however, the majority of life-saving medical discoveries did not occur until after 1935 (Le Fanu 1999). Thus, most medical interventions occurred decades after the already rapid and widespread decline in mortality. So, too, arguments about improved nutrition within populations are weakened by inconsistencies across countries that shared similar nutritional profiles but had divergent mortality trajectories (McKeown 1976; Caldwell 1990).

By contrast, the timing of the educational revolution and the decline in the crude death rate that characterized the FDT is closely aligned: as increasing proportions of the population gained access to basic schooling, death rates declined. The crude death rate and primary and secondary school enrollment rates from 1815 to 1830 remained relatively stable, but from 1830 to 1850, as primary and secondary enrollment rates per capita quickly increased, death rates began to fall rapidly. The initial decline in death rates was due to decreases in infant and child mortality, and as research on developing countries undergoing demographic transition clearly concludes, a robust relationship exists between maternal education and child survival (e.g., Caldwell 1979; Caldwell and Caldwell 1993). And, as we argue below, the relationship between schooling and mortality may improve women's health and that of their children principally through enhancing thinking and reasoning skills.

The onset of fertility change is another component of the FDT; and while it is beyond the focus of this article, a similar argument can be made about a causal education effect on declining birth rates, both in the countries experiencing the FDT and more recently in developing countries (e.g., Caldwell 1980; Cochrane 1979; Teitelbaum 1984). Also, historical demographic research has considered how fertility decline in the FDT was related to the diffusion of ideas concerning the ability, capacity, and appropriateness of controlling fertility, an argument much in alignment with effects of rising mass education during this period (Coale and Watkins 1986).

In addition to countries that historically experienced the FDT, there is evidence that educational expansion among populations elsewhere in the world is temporally related to similar demographic transitions. For example, low- and middle-income countries experienced a rapid increase in life expectancy at birth from 40 years in the early 1950s to 65 years by 2005, and starting just after World War II these countries began significant strides toward wide access to basic schooling for all children (World Bank 2007). Changes in the lives of girls and women are a focal point at the beginning of transitions, when fertility and infant mortality rapidly decline. Nevertheless, considerable research suggests that before, during, and after population transformations, education is independently related to life expectancy (Mirowsky and Ross 2003). For example, two recent studies show that the association between education and mortality at the individual level continues to shape population dynamics even as norms of education reach into postsecondary levels. Strand et al. (2010), reporting on a 65-year longitudinal study in Norway, where inequalities in access to health services are minimal, find that the mortality gap between males with basic education and males with the highest levels of education widens in younger cohorts. And Brown et al. (2010) find that educational expansion in the United States has "rectangularized" the human survival curve.

It should be noted that previous research on the demographic transition has not entirely ignored education. But the focus has been mostly on an indirect effect, such as longer schooling raising females' age at marriage and childbearing, or children's time spent in school (instead of working) raising costs for families and hence leading to lower fertility and greater maternal care (Becker and Tomes 1976). Although these indirect facets are clear in

theory, there is little empirical evidence for these mechanisms. For this reason, as described below, we hypothesize that schooling more directly influences individual behavior and population change by enhancing higher-order cognition.3 Furthermore, although the FDT was likely the result of various population characteristics, the pervasive education revolution was one cause of modernity, and this timing supports the view of education as a major cause of the FDT and similar demographic transitions occurring later than in the West in other parts of the world (e.g., Caldwell 2006; Kirk 1996).

Meta-analysis of the education effect on adult mortality

Many studies in countries at all levels of development show a correlation between education and health. Even with small amounts of formal schooling (2–3 years) in less developed regions, observable differences in health outcomes arise in comparison to non-schooled individuals (de Walque et al. 2005; Kenkel 1991; Becker, Agopian, and Yeh 1992). The association has also been widely observed in developed regions. To our knowledge, however, no meta-analysis of the relationship between education and mortality has been conducted to establish the education effect.

There are three reasons why our meta-analysis uses adult mortality as the health outcome. First, the mortality gradient is a clearly defined and directly comparable outcome across countries and cultures and is a general measure of overall health. Second, inequalities in mortality gradients in a number of developed countries have already been attributed in part to inequalities in education (e.g., Kunst and Mackenbach 1994). Third, there is a well-established and methodologically sophisticated research literature on mortality among adults.

A meta-analysis is not an all-inclusive review of studies; instead the technique statistically assesses the effect of an independent variable (education) on a dependent variable (mortality) across studies employing the best available methodological procedures (Glass, McGaw, and Smith 1981). Since it can be assumed that studies that found a significant effect of education are more likely to be published than those with a null effect, a meta-analysis also statistically assesses the likelihood of publication bias. An Egger regression asymmetry test for publication bias across studies was performed. Also, to adjust estimates of pooled effect sizes for any effects of the heterogeneity in qualities of studies (e.g., sample size, study location, measurement difference), a meta-regression analysis was estimated.

Searching and selecting articles for the meta-analysis

Various search engines were applied to databases of scientific publications—JSTOR, PubMed, National Bureau of Economic Research (NBER), Pro-Quest, PsycINFO, Science Direct—from 1995 to 2005, using different words conveying the combination of the two key variables: education and mortality.4 Additionally, Google Scholar was searched on “education” and “mortality” and all possible synonyms. The searches yielded about 700 documents including book chapters, working papers, conference proceedings, and peer-reviewed journal articles. Dropping duplicate listings and all documents not published in peer-reviewed scientific journals left 112 articles. A close reading of each of these was assessed by the following scientific criteria for inclusion in the meta-analysis.

[3]Further too, for the first demographic transition and other transitions since, it is generally observed that child mortality declines occur slightly before fertility declines, thus calling into question causal arguments about higher material investment in the fewer children per household. Also this sequence in demographic transitions tends to be a problem for the related argument that fertility decline is the only intervening factor between mother’s education and reduced child mortality. Both of these causal problems raise the possibility that growing access to education was at least one causal factor influencing both parts of the transition (e.g., Caldwell 1979).

[4]The key terms used for those concepts were: For education: education, schooling, or educated. For mortality: death, fatality, mortality, or loss. And, these key terms were combined for all searches.

To be included in the meta-analysis, a study had to meet all of the following scientific criteria: 1) an original empirical study on adult mortality as dependent variable (e.g., re-analyses, reviews, comments, and editorials were excluded); 2) inclusion of formal educational attainment of subjects as an independent categorical variable; 3) a sample size of at least 100 adults; and 4) adequate statistical estimate to extract the education effect size (relative risk, odds ratios, or hazard ratio) and corresponding confidence interval including multivariate statistical conditioning on relevant demographic factors (e.g., age, sex, race).5 While many studies meeting these criteria also statistically conditioned on economic resources or socioeconomic status, a number did not. Therefore to establish an independent education effect, we included studies that statistically conditioned on economic resources or socioeconomic status.

Applying these criteria yielded 29 studies from 14 demographic, epidemiological, and medical journals.6 While some studies report just a single education effect on mortality, others report effects for unique sub-samples in the study (e.g., age cohorts, sex), for a total of 69 reported effects across a sample of more than 20 million adults. See Appendix A for details of the studies included in the analysis.

Among the selected studies, eight (28 percent) are studies on North American samples, 18 (62 percent) on European samples, and three (10 percent) on Asian and Middle Eastern samples. Twenty-seven studies (93 percent) included at least 1,000 subjects. Nineteen of the 29 (66 percent) used regionally or nationally representative samples of adults collected by a governmental agency, while the rest used data collected by investigators. Nineteen studies (66 percent) conditioned on socioeconomic status (e.g., occupational status) or economic resources (e.g., income and household possessions).

Coding the selected studies

In addition to estimating the pooled education effect size from all 69 reported effects, comparing effects from six subsets of samples illuminates various aspects of the influence of education on mortality. To calculate these, we coded each of the study samples by specific characteristics: conditioned on socioeconomic status (SES) and economic resources (1=yes, 0=no); continent of study country (1=North America, 2=Europe, 3=Asia and Middle East); sex (1=male, 2=female, 3=male and female); sample age cohort (1=all ages, 2=30 years or older, 3=50 years or older); representativeness of sample (1= nationally representative, 2=regionally representative, 3=non-representative); and data collection method (1=cross-sectional, 2=merging of demographic characteristics with death records, 3=direct tracking of subjects). To estimate the reliability of the various pooled effect sizes (i.e., a lack of publication bias) we regressed education effect sizes on five characteristics of studies found to have significant difference in size of effect: representativeness of sample (1=nationally or regionally representative sample, 0=otherwise); type of mortality (0=specific cause of death, 1=all causes of death); continent of study country (dummy per continent); conditioned on SES (1=yes, 0=no); and study design (1=direct tracking of subjects, 0=merged official records and cross-sectional).

[5]In a few cases several selected studies reported effect sizes from the same public data. In such cases one study was randomly selected for inclusion in the meta-analysis.

[6]The final 29 studies meeting all the selection criteria came from 14 journals: *Addiction*, *American Journal of Epidemiology*, *American Journal of Public Health*, *Annals of Epidemiology*, *Cancer Causes and Control*, *Epidemiology*, *European Journal of Cancer*, *European Journal of Epidemiology*, *European Journal of Public Health*, *International Journal of Epidemiology*, *Journal of Clinical Epidemiology*, *Journal of Epidemiology and Community Health*, *Preventive Medicine*, and *Social Science and Medicine*.

The size of the education effect

The 69 reported education effect sizes are variously expressed as relative risk, hazard ratios, or odds ratios. Since effect sizes expressed as odds ratios are not strictly comparable to those expressed as relative risk/hazard ratios, odds ratios were converted and expressed as relative risk for comparison (Zhang and Yu 1998). Also, because of some variation in the design and heterogeneity among sampled populations across studies, we used a random-effects model to estimate the pooled effect sizes, where each effect size is weighted by the inverse of the variance, made up of within- and between-study variances (DerSimonian and Laird 1986). This estimation model does not assume a singular true effect size across studies; instead a random-effects model assumes that there is a distribution of true effect sizes, and the pooled effect size is the average of the population of true effects. The pooled relative risks compare low education (i.e., no education through primary schooling) against high education (i.e., secondary through postsecondary education), and are interpreted as the increase in the probability that subjects with primary education or less died over the course of the study in comparison to individuals with secondary education or higher.

All of the 69 reported education effect sizes indicate the same association: less education is associated with a higher likelihood of death. Table 1 shows the pooled estimation of the effect sizes calculated across all reported effects, and also for various effects from sub-samples. Across all reported effect sizes, the pooled education effect size on mortality is expressed as an average relative risk of 1.46, meaning that people with no education or lower secondary schooling have a 46 percent higher probability of dying than people with high school or higher education.

An illustrative example of the overall education effect is a study that followed 400,000 American men and women from 1979 to 1989 using the National Longitudinal Mortality Study (NLMS) (Backlund, Sorlie, and Johnson 1999). This study reports that after conditioning on a host of demographic factors, women with primary education or less were 33 percent more likely to have died over the course of the study than college-educated women, and men with primary education or less were 42 percent more likely to have died than college-educated men.

A more rigorous test of the independence of the education effect is found among studies that condition on socioeconomic status and economic resources, two factors widely assumed to be more important for health than education, and for which education is assumed to serve only as a proxy. Yet, as reported in the second set of comparisons in Table 1, after conditioning on socioeconomic status the pooled education effect size is even larger than the effect for all studies. After controlling for socioeconomic status and economic resources, less-educated persons are 67 percent more likely to die earlier than the more educated.[7] For example, Winkleby and Cubbin's (2003) study of the US National Health Interview Survey merged with the National Death Index reports that after controlling for socioeconomic status (i.e., occupational status), white women with nine years of schooling or less have a 44 percent higher probability of death than white women with high school or higher education, and for less-educated white men the probability of earlier death was 49 percent greater than for more highly educated white men.

Similarly, if education has a robust effect on health, then effect sizes should be larger in samples with more variation among persons with lower levels of educational attainment, including adults with no schooling. Because Asian and Middle Eastern countries expanded education later than countries in North America and Europe, there should be larger

[7]The pooled effect sizes among comparisons that did not condition on socioeconomic status or economic resources are smaller, with a standard deviation of .06 versus .70 among studies that did condition.

education effect sizes in the former. This is shown in the third comparison in Table 1. The five education effect sizes from studies of Asian and Middle Eastern samples report larger effects than those from Europe and North America. For example, a study of age cohorts in South Korea using the National Health and Nutrition Examination Survey reported that Korean adults with no formal education were four times more likely to die than those with at least a college education (Khang and Kim 2005).

In the same vein, it is well documented that the educational revolution has incorporated successive generations of males and females into greater amounts of schooling. Although many developing countries are lagging in universal education for females, much historical research finds that once initiated, mass Western-style education has similar effects on both sexes (e.g., Tyack and Hansot 1990). This is shown in the fourth comparison in Table 1, which indicates that education has approximately the same effect size on mortality for both sexes. For example, Mackenback et al. (2004), using nationally and regionally representative datasets from European countries, report that less-educated Danish men and women have respectively a 33 percent and 29 percent higher probability of dying earlier than their highly educated counterparts. The same study reports similar education effects for less-educated compared to better-educated Flemish men (36 percent) and women (30 percent).

The fifth comparison indicates a larger pooled effect size among studies using samples from age cohorts 50 and younger than those using samples of 51 years or older. This is likely a consequence of upwardly expanding normative levels of education among younger cohorts that widens the education gap for individuals who complete less education.

The next comparison in Table 1 indicates the broadness of the education effect. Studies employing nationally representative samples reported larger effect sizes. And lastly, studies using direct tracking of subjects over time report larger education effect sizes than those matching and merging data on demographic factors with mortality, most likely because of reduced random error with the former method.

These reported pooled education effect sizes are reliable estimates from a valid sample of the studies undertaken about this relationship. An Egger regression asymmetry test (Egger et al. 1997) of publication bias detected no overall evidence of bias ($t = -.48$, $p = .63$). To examine this further, we regressed all 69 reported education effect sizes on five study characteristics; only education effects estimated on cohorts of 51 years and older proved different (smaller education effects) at a statistically significant level (see Appendix B for meta-regression results).

Collectively, the pooled education effects on mortality are derived from a set of studies representing over 20 million subjects from various world regions. Employing rigorous methodology and conditioning for a range of demographic factors, including in most cases economic resources and socioeconomic status, the results from the meta-analysis confirm an independent, consistent, and substantial effect of education on adult mortality. This conclusion begs the question, What causal mechanism is most responsible for the education effect on health to the point that it is so robustly associated with longevity?

Toward a cognitive hypothesis about the education effect on health

As described in the first section above, formal education has evolved into a pervasive social institution. Therefore educational attainment and related performance can influence individuals in a number of ways, including determination of adult social status and material resources acquired through formal educational degrees; change of self-image vis-à-vis others; investment of considerable time during childhood and adolescence; and inculcation of greater future orientation. Each of these mechanisms has been used by past research to

explain the education effect on health, yet the limited empirical evidence supporting cognitive mechanisms of education prompts us to focus on the processes of learning and cognitive development that occur through schooling.

Unlike many theorized hypotheses about the education effect, there is growing scientific evidence of a cognitive impact on mortality. Starting with Gottfredson's (1997) argument about general intelligence and its implications for everyday life, recent research indicates an association between intelligence, as measured on standard IQ instruments, and health outcomes, many of which specifically address mortality (Batty, Deary, and Gottfredson 2007; Deary et al. 2008; Gale et al., 2010). For example, Lager, Bremberg, and Vågerö (2010) find in a 65-year Swedish longitudinal study that higher early IQ (measured at age ten) reduced mortality risk among males.

Children enter school with differing levels of genetically endowed intelligence and differing influences of early parenting. In light of this, some interpret the evidence on cognitive effects to mean that intelligence is a non-malleable quality mostly unchanged by environmental factors, and that educational attainment mostly verifies intelligence. Yet some evidence indicates that without the enhancement of cognitive skills through formal schooling, the effect of intelligence could be greatly muted. Research on the relationship between IQ and mortality establishes the influence of cognition, but what remains to be considered is the additional influence of formal education on cognitive skills. For example, a recent longitudinal study by Herd (2010) supports the perspective that, when controlling for childhood IQ, formal education increases cognitive human capital, which helps to explain the strong relationship between educational attainment and health outcomes in later life.

Schooling is a unique environment in which students spend long periods of time engaged in cognitive activities, and we propose that this process is the key to understanding the causal mechanism behind the education effect on health. Learning to read, write, and use numbers, even under rudimentary conditions, is the result of considerable abstract cognitive exercise that likely transforms how educated individuals think, reason, and solve problems. These are skills that cognitive science refers to collectively as "meta-cognition" (e.g., Martinez 2000). Further, there is evidence that schooling progressively becomes more cognitive in its demands as students move to higher grades. As other research has pointed out, formal education enmeshes individuals in a uniquely different cognitive process in comparison to that related to subsistence-level farming, early industrial factory work, and other pre-modern activities. With the exception of sacred and magical beliefs, the large mass of non-elite people over most of the course of human history lived in a very concrete world.

Progressive meta-cognitive enhancement derived from schooling may prove to be a powerful mechanism in creating the education effect on health behaviors. The relatively recent advent of mass education is the single largest non-family institution for children, and a considerable extent of its core process is a cognitive intervention. A robust meta-cognitive effect of this institution may have gone largely unnoticed given that the education revolution makes schooling almost universal, yet there is recent evidence of such an effect from schooling. As we noted above, the educational revolution has spread worldwide a relatively standardized version of Western-style schooling that includes significant potential for cognitive change in individuals. Therefore a cognitive hypothesis is a promising route to understanding how the education effect works across the globe. Our *schooling-enhanced cognition/health hypothesis* proposes that through the teaching of literacy, numeracy, and other academic subjects, schooling transforms general intelligence into higher-order cognitive skills that promote risk assessment and decision making abilities related to health.

Recent research on neuro-development and higher-order cognition establishes evidence supporting the schooling-enhanced cognition/health hypothesis.

The neurological and cognitive impact of schooling

Neurological development of higher-order cognitive skills occurs at least through late adolescence and is highly responsive to environmental stimulation, such as routinely occurs in formal education. Contrary to an older assumption that most neurological development is complete before a child begins school, recent research finds that higher-order cognitive skills can be developed by interactions with the environment well into early adulthood, if not longer (Blair et al. 2005; Duncan, Burgess, and Emslie 1995; Eslinger, Flaherty-Craig, and Benton 2004; Shallice and Burgess 1991; Waltz et al. 1999). The ability of functional magnetic resolution imaging (fMRI) to measure activations of different regions in the brain while subjects perform cognitive tasks has generated the most compelling evidence on this point. For example, in a recent developmental fMRI experiment, when 8–19-year-olds solved problems that require some reasoning from primary-school mathematics, the brain areas associated with higher-order cognition are activated (Eslinger et al. 2009). The study also found a clear age effect: younger students who did not readily know the answers activated these higher-order cognitive areas more than older students who more likely activated areas of the brain associated with long-term memory. Combining this evidence with findings that the structure and functions of the brain progressively emerge out of use—or what is called “activity-dependent development” or “neural plasticity”—suggests that even simple cognitive activities during early schooling can have long-lasting effects on neurological functioning (e.g., Hubel and Weisel 1962; Quartz and Sjenowski 1997).

Exposure to schooling is monotonically and linearly associated with enhanced higher-order cognition. Research comparing schooled and unschooled adults finds that within a genetically determined range of capability, schooling significantly enhances meta-cognitive skills such as categorization, logical deduction, and IQ (e.g., Blair et al. 2005; Cole 2003; Luria 1976; Nisbet et al. 1993). A meta-analysis of over 50 studies using naturalistic observation, post-hoc statistical comparisons, and cohort-sequential analysis estimates the education effect to range from 0.3 to 0.6 of an IQ point for every year of school completed. Given that the standard deviation of most IQ tests is 15 points, multiple years in school results in a sizable increase in IQ (Ceci 1991). Perhaps most convincing, quasi-experimental studies of unschooled and schooled adults in subsistence-level farming communities find that even small amounts of schooling as a child yield higher-order cognitive skills among adults net of work conditions and more general social and economic status (Christian, Bachman, and Morrison 2001; Cole 2003; Luria 1976; Stevensen et al. 1991; Stevenson, Chen, and Booth 1990).

Higher-order cognition is associated with enhanced risk assessment and decision making skills. Experiments on risk assessment and the use of effective strategies for decision making repeatedly find that such skills are positively associated with numeracy and higher-order cognition (e.g., Bruine de Bruin, Parker, and Fischhoff 2007; Peters et al. 2006). Risk assessment and decision-making skills are important components of reasoning related to health and making choices about protective behavior. Lastly, demographic research has identified some of the general social benefits of higher education that appear to be unrelated to cognition, such as evidence that education increases supportive relationships and greater social support, which are associated with positive health and decreased risk of mortality (e.g., House, Landis, and Umberson 1988; Kelsey et al. 2000). But recent neurological research shows as well that social skills are closely associated with higher-order cognitive capabilities of the kind that can be enhanced through schooling (e.g., Eslinger et al. 2007).

In addition to the neurological evidence on the meta-cognitive effects of schooling, two recent studies offer direct tests of our schooling-enhanced cognition/health hypothesis. We conclude with a brief discussion of each.

The first study is a response to a major limitation of the IQ and mortality research on samples from highly educated populations, namely that individuals with more general intelligence tend to achieve higher educational levels. To counter this, data were collected from a naturally occurring experiment on school attendance among 181 rural unschooled and schooled adults in Ghana, and analysis of these data find that education during childhood was associated with current reports of adopting more protective health behaviors (Peters et al. 2010). After conditioning on economic resources and other demographic characteristics, a structural-equation model showed that subjects' cognitive skills, numeracy, and decision making skills increased with exposure to schooling, and these enhanced skills mediated the effects of education on protective health behaviors related to HIV infection. Importantly, among this sample there was no evidence of selection effects, since schooling access and attainment were unrelated to cognitive qualities of subjects as children.

The second study extended this finding to large nationally representative samples. Structural-equation modeling of Demographic and Health Survey data from nine sub-Saharan African countries finds, net of control variables, a robust, positive effect of education (ranging from unschooled to some secondary schooling) on condom use among adults engaged in sexually risky behavior (Baker, Leon, and Collins 2010). Although formal education was also found to increase acquisition of basic facts and the inculcation of positive attitudes, these commonly assumed effects of formal education have only weak influence on condom use. Instead, education robustly influences health reasoning ability, and this factor mediates a significant proportion of the education effect on condom use.

This finding, that well-informed reasoning about the risk of disease and the adoption of prevention strategies occur over the life course, is likely to be the causal mechanism behind the associations between education and mortality reported in the meta-analysis above. Intelligence certainly plays a causal role in reducing mortality, but formal schooling adds significant value to innate ability in the form of higher-order cognitive skills crucial to decisions about health.

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FIGURE 1. Timing of the onset of the first demographic transition and the educational revolution in England and Wales, France, Sweden, and the United States

NOTE: CBR and CDR are weighted based on total population and averaged across countries. The school enrollment rates are from the only historical source of which the authors are aware. Although the limited nature of historical data makes it difficult to judge their reliability, these data are widely used for historical education analysis.

SOURCE: For birth and death rates: Human Mortality Database (2009); for school enrollment rates: Arthur Banks Education Statistics Cross-National Time-Series Data Archive (CNTS) Copyright 2008.

TABLE 1

Pooled relative risk ratios from 69 reported education effect sizes from 29 studies

	Number of samples	Pooled effect size (as relative risk)	Pooled effect size (as coefficient)	Confidence interval	
				Lower bound	Upper bound
All samples	69	1.46	0.38	1.33	1.59
By SES control[a]					
Conditioning on SES[b]	33	1.67	0.52	1.26	2.22
No conditioning on SES[c]	36	1.32	0.28	1.28	1.36
By continent					
North America[b]	17	1.29	0.25	1.22	1.35
Europe[b]	47	1.38	0.32	1.34	1.42
Asia and Middle East[c]	5	2.61	0.96	1.68	4.06
By sex					
Only females[b]	27	1.40	0.33	1.32	1.48
Only males[b]	31	1.46	0.38	1.24	1.71
Males and females[b]	11	1.49	0.40	1.29	1.73
By age[d]					
50 or under[b]	63	1.49	0.40	1.36	1.63
51 or older[b]	5	1.07	0.07	0.77	1.48
By representativeness					
National or regional[b]	49	1.56	0.44	1.39	1.74
Non-representative[c]	20	1.26	0.23	1.17	1.36
By data collection method					
Merged mortality records and cross-sectional[b]	45	1.31	0.27	1.28	1.35
Direct tracking[c]	24	1.85	0.62	1.36	2.52

[a] The socioeconomic status variables included in the meta-analysis were related to income (e.g., individual), social class (e.g., occupation), and assets at home (e.g., index of amenities).

[b] No significant differences between pooled education effect sizes.

[c] Pooled education effect size is statistically different from other studies in category ($p<.05$).

[d] One study in the meta-analysis did not indicate the age range of the sample.

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APPENDIX A

Studies included in the meta-analysis

Year of publication	Author(s)	Country(ies)	Type of mortality	Age group	Number of subsamples (effect sizes)	Effect size or range of effect sizes (RR)
1999	Backlund, Sorlie, and Johnson	United States	All causes	25–64	2	1.33–1.42
2003	Bobak et al.	Russia	All causes	25 and older	2	1.89–2.61
2002	Egeland et al.	Norway	Coronary heart disease	35–56	1	1.00
2003	Franks, Gold, and Fiscella	United States	All causes	21 and older	1	1.14
2004	Gnavi et al.	Italy	All causes	20 and older	2	1.35–1.7
2005	Khang and Kim	South Korea	All causes	30 and older	1	2.15
2001	Kilander et al.	Sweden	All causes	50	1	1.01
2000	Kposowa	United States	Suicide	15 and older	1	1.50
2003	Laatikainen et al.	Finland	All causes	25–64	1	1.57
2003	Leinsalu, Vågerö, and Kunst	Estonia	All causes	20 and older	2	1.37–2.23
2002	Long et al.	United States	All causes	72 and older	1	0.50
2004	Mackenbach et al.	Ten European populations	All causes	40–89	20	1.17–1.45
1999	Makela	Finland	Alcohol related	35–69	2	1.43–1.86
2004	Malyutina et al.	Russia	All causes	25–64	2	1.13–1.41
2004	Manor et al.	Israel	Heart diseases	45–89	2	1.21–2.54
2001	Martikainen et al.	Finland	Lung cancer	50–69	1	1.32
1997	McDonough et al.	United States	All causes	45–64	1	1.35
2005	Menvielle et al.	France	All causes	35–59	2	1.80–2.10
1997	Mustard et al.	Canada	All causes	0 and older	1	1.38
2002	Osler and Prescott	Denmark	All causes	20 and older	1	1.13
2002	Pensola and Valkonen	Finland	All causes	30–34	1	2.90
2002	Regidor et al.	Spain	Infectious diseases	35–64	2	2.73–2.82
2002	Son et al.	South Korea	Work related (manual and non-manual workers)	20–64	2	3.06–5.37
2002	Steenland, Henley, and Thun	United States	All causes	45 and older	4	1.14–1.28
1997	Sundquist and Johansson	Sweden	All causes	25–64	2	1.29–1.72
2004	Van Oort, Van Lenthe, N and Mackenbach	Netherlands	All causes	15–54	1	1.66
2000	Van Rossum et al.	Netherlands	All causes	55 and older	2	1.30–1.50

Year of publication	Author(s)	Country(ies)	Type of mortality	Age group	Number of subsamples (effect sizes)	Effect size or range of effect sizes (RR)
2003	Vescio, Smith, and Giampaoli	Italy	All causes	20–75	2	1.00–1.33
2003	Winkleby and Cubbin	United Statea	All causes	25–64	6	1.05–4.76

NOTE: Relative risk is the ratio of the likelihood of the event (mortality) occurring in the exposed group versus a non-exposed group, or in this case the likelihood of death among those with low versus high levels of education.

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Appendix B

Meta-regression analysis to identify possible bias in effect sizes

	Coefficient	Standard error	Relative risk (RR)	Confidence interval (RR)	
				Lower	Upper
All causes of mortality[a]	0.04	0.11	1.04	0.83	1.31
National or regional sample[b]	0.12	0.07	1.12	0.98	1.29
North America[c]	−0.69**	0.16	0.50	0.37	0.69
Europe[c]	−0.42**	0.15	0.66	0.49	0.89
Direct tracking[d]	−0.25**	0.09	0.78	0.65	0.93
Conditioning on SES[e]	0.07	0.08	1.07	0.91	1.26
Constant	0.85	0.16			

Adjusted R-square: .51 F (6,62): 9.63 (.000)

*p<.05;

**p<0.01

[a]Reference group was specific cause of mortality.

[b]Reference group was non-national samples.

[c]Reference group was Asian samples.

[d]Reference group was non-longitudinal studies.

[e]Reference group was no control for SES.

NOTE: Relative risk is the ratio of the likelihood of the event (mortality) occurring in the exposed group versus a non-exposed group, or in this case the likelihood of death among those with low versus high levels of education.