Education and Population Health: A Reassessment

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Abstract

Within demographic research are numerous reports of consistent and significant associations between formal education attainment and a range of health risks such as smoking, drug abuse, accidents, the contraction of many diseases, and health outcomes such as mortality—almost all indicating the same conclusion: More educated individuals are healthier and live longer. The substantial number of studies reporting a robust education effect along with a weak appreciation of its role as a causal agent leads to confusion about the independent influence of formal schooling. To address the underappreciated effect of education on mortality, three contributions are provided: 1) a macro-level summary of the dimensions of the worldwide educational revolution and a reassessment of its role in macro-level demographic health transitions; 2) a meta-analysis of methodologically sophisticated studies of the effect of educational attainment on all-cause mortality to establish the independence and robustness of the education effect on health; and, 3) an improved hypothesis about how education is a powerful determinant of health in light of new multidisciplinary cognitive research.
Education and Population Health: A Reassessment

“Maternal education is the single most significant determinate of these marked differences in child mortality. These are also affected by a range of other socio-economic factors, but no other factor has the impact of maternal education and in their totality they do not even come close to explaining the effects of maternal education.”

J.C. Caldwell, from “Education as a Factor in Mortality Decline: An Examination of the Nigerian Data” (1979, p.408)

Introduction

It has been three decades since the publication of John Caldwell’s (1979) groundbreaking study of maternal education and child mortality in which he concludes from an analysis of Nigerian mothers and their children, that even a few years of maternal education significantly improved the chances of child survival. So compelling were the results that a whole research literature on maternal education and child mortality sprung up over the ensuing years (Cochrane, O’Hara, & Leslie 1982; Cochrane, 1979; Cochrane, O’Hara, & Leslie, 1982; Barrera, 1990; Buchmann, 1996). Unfortunately, from this auspicious beginning the evaluation and understanding of formal education as a major causal factor in morbidity and mortality remains underdeveloped in demography. Although still widely reported in empirical research, education’s positive association with health and longevity is underappreciated as a cause in its own right—or what is hereafter referred to as the “education effect.”

Within demographic research literatures are numerous reports of consistent and significant associations between formal education attainment and a range of individual health outcomes and risks such as mortality, smoking, drug abuse, accidents, and the contraction of many diseases; almost all indicate the same conclusion—more 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, 6) conclude, “education has an enduring, consistent, and growing effect on health.”

Macro demographic and development studies also identify a significant association between expanding mass education and population health, yet many, especially the earliest ones, did not control for other factors that also arise with more education and better health, such as economic development (see review by Feinstein, 1993). Therefore, it is frequently suggested that the association between expanding education and population health in nations 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 cause both mass education and healthier populations (e.g. Bloom, 2007).

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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 paper.
The substantial number of studies reporting a robust education effect along with a weak appreciation of its role as a causal agent leads to confusion about the independent influence of formal schooling, and fosters speculation that education is mostly a proxy for other dimensions of socioeconomic status at the individual level or economic development at the aggregate level (Desai and Alva 1998; Basu, 1994; Hobcraft, 1993, Kunstadter, 1995). Consequently the relationships between education and individual health, as well as mass education and population health continue to be debated (e.g. Grossman, 1972; Kitagawa & Hauser 1973).

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 of modernization, instead of as one of modernization’s main causes. Second, there has yet to be a systematic assessment of the education effect on health across a full set of methodologically sophisticated studies that control for wealth and other dimensions of socioeconomic status. Third, existing hypotheses about the education effect are limited in their ideas about how schooling influences health of individuals and populations.

In addressing these, three contributions are provided here to illuminate the education effect on health. First is a summary of the dimensions of the worldwide educational revolution and a reassessment of its potential causal influence on modernity and macro demographic transitions in developed and developing nations. Second, to establish the independence and robustness of the education effect on individual health, a meta-analysis is undertaken of peer-reviewed micro-level studies on the relationship between educational attainment and all-cause mortality. Lastly, in light of new multidisciplinary research on the cognitive impact of schooling, we propose an improved hypothesis about how education may be a powerful determinant of health and longevity.

I. Reassessment of the Mass Education Effect on Population Health

A large set of multidisciplinary studies from sociological, historical, and economics research examining educational development over the past 150 years yields three central findings about the expansion of formal education in populations 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 from highly schooled societies; and third, the timing and causal power of the growth of education suggests it was an important cause of the first demographic transition.

1. The Education Revolution as a Cause of Modernity

What is referred to as the “educational revolution” is marked by persistent worldwide growth in formal schooling both in terms of increasing school enrollment rates and an upward expansion of formal educational attainment. Yet, traditionally when scholars identify the large social forces that are responsible for the development of the modern society, they rarely, if ever, 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 causal credit.
There is, however, growing evidence that the education revolution played, and continues to play, a causal role in the development of many main features of modern societies. The evidence centers around A) the demographic size and temporal dynamics of the educational revolution; B) evidence on what did, and did not, initiated mass education; C) results from econometric modeling of the causal impact of educational expansion on national economic development across the 20th century; and, D) intensive worldwide adoption of a common model of education capable of common impact worldwide.

A) Size and Speed of the Educational Revolution: The historical spread of formal education was so extensive and arose temporally with economic development and growing social complexity that it is unlikely to have been a result of these factors. Since educational expansion is ubiquitous in all types of nations, the global historical dimensions of the education revolution are not as readily apparent. By the turn of the 21st century formal education in the world is immense in terms of it recent growth, its claim to people’s time and effort, and its level of impact on their lives. What is demographically salient about the educational revolution are both its relative newness to human society and speed of growth. The enrollment of children and youth into primary, secondary, and tertiary (i.e. higher education) schooling over the past two hundred years worldwide is anthropologically-speaking 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 is a new and massive change in human behavior. During the last 60 years, populations in low-income nations transitioned from virtually no access to schooling to wide access to primary and even secondary education, so that now an estimated 80% of all humans aged 15 or older have been schooled enough to both read and write a short statement about their life, a population characteristic that would have been unthinkable just 50 years ago (UNESCO 2004).

New advanced sectors of education are spurred on by the earlier growth of the subsequent sector. The steady growth in primary school enrollment throughout the second half of the 19th century and into the first few decades of the 20th century began a logarithmic climb worldwide from 1940. As primary schooling reached large proportions of the world’s population twenty years later, enrollment in secondary schooling turned sharply up in the 1960s. And then in the early 1970s enrollment in higher education began to grow. What began with the expansion of primary schooling in the middle of 19th century continues to spread, as mass higher education is the next wave of the educational revolution. For example, about half a million students, or just 1% of the youth age-cohort were enrolled in higher education worldwide in 1900, yet a century later approximately 100 million were enrolled, representing 20% of the college-aged cohort (Schofer and Meyer 2005). In a number of nations, enrollment rates are now rapidly climbing past 50% and in some reaching 80% of the age-cohort attending higher education (UNESCO, 2007).

B) Causes of Educational Expansion: The growth 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 modeling concludes that this is a lack of consistent historical ordering among these trends, these two modernizing forces are unlikely unidirectional causes of the educational revolution (e.g., Benavot, 1992; Meyer, Ramirez, & Soysal, 1992, Meyer Tyack, Nagel, 1979). The same is true for political democratization of nations, another
factor often assumed to have caused the education revolution. Instead, what these analyses show is that the education revolution, economic and urban development, and political democratization are co-determinants of one another, and the causal process occurs more on a global than national level (Baker 1999).

For example, three main findings of a random effects GLS regression estimate of a detailed model of national characteristics and higher education enrollments over the 20th century illustrates 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. GDP) and degree of urbanization are only moderately related to growth in higher education over the century (see also Windolf, 1997). Obviously it takes national resources and a certain level of development to undertake large amounts of 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 nations most connected to the “world society,” meaning a combination of factors that make up modern western culture including scientization of culture, rational national planning for progress, democratization, and the globalized world polity of multilateral agencies such as the United Nations and INGOs (international non-government agencies) (Drori, Meyer, & Hwang, 2006; Suárez, 2007). Nations 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 (not absolute levels) begin to converge in almost all nations, indicating that educational expansion leads to additional expansion, much like the endogenous intensification of other trends of modernity such as capitalism and democratic polities.

C) Causal Impact of Educational Expansion on National Economic Development: Econometric modeling of historical macro-economic development of several nations over the 20th century shows that a significant force in economic development is the upward spiraling interplay between rising human (i.e. chiefly educational) capital of the working population and technological change (Goldin, Katz 1998; Rubinson, 1986). Detailed historical studies also indicate that greater use of “pervasive skill-biased” (e.g. continuous-process and batch production technologies) was caused by the extensive growth in secondary education enrollment rates in the U.S. during the first half of the 20th century. This complimentary relationship between expanded education and economic development will likely continue in economies throughout the rest of the century (e.g. Baker 2009; Berman, Bound, Machin 1998; Levy and Murname 1996; Auter, Katz, Krueger 1998).

D) Common Model of Education: There is considerable evidence that rather than a result of unique national histories and cultures, the form and organization of formal education has steadily converged on a worldwide model. Extensive comparative analyses across education systems in all regions of the world find a steady trend towards isomorphic educational practice in terms of the organization, operation, curricular content, teaching, learning, and deeply held values about education and its social role (e.g. Inkles, 1974; Baker and LeTendre 2005). So too, there is historical analysis suggesting that the basic epistemological model and cultural ideas that sparked the expansion of primary and secondary schooling grew out of the spread of a western-style university throughout Europe and North America over the last three centuries (Meyer, Ramirez, Soysal, 1992; Benavot, Riddle, 1988). This shared model of education as a social institution
creates the potential for a relatively uniform education effect on development worldwide, similar to the universalizing effects of large-scale capitalism and widespread democracy that also have converged in form over time.

2. Underestimation of Schooling Effects in Fully Schooled Societies

A second factor behind the under-appreciation of the education effect on health is that the significant number of studies in highly schooled nations such as the U.S., Western Europe, and parts of Asia tend to underestimate the full impact of education. This occurs for two reasons.

First, as is obvious from the historical development of the education revolution, educational attainment has saturated populations in developed nations and thus studies using samples from these populations are restricted as to variation in exposure to schooling, and even this limited variation is decreasing over time. Currently, for example, the medium educational attainment is just over the completion of secondary school (12+ years) in developed nations such as the U.S., Sweden, and Japan (OECD 2008). Furthermore, variation or inequality in educational attainment has declined by 46% worldwide from just 1980 to 2000, double the rate of decline in income and life expectancy inequality (Goesling and Baker 2008; see also Neumayer 2003). While there is no evidence that the education effect on health is other than linearly monotonic, it is possible that the many studies of education and health done on samples from heavily schooled nations with high attainment and limited education variation within their populations may not capture the more robust effects between the extremes of unschooled and moderately schooled as those reviewed below.

Second and related, there is a widely held misperception that weak between-schools 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, LeTendre, 2005). The former refers to the effect of going to one school versus another often on achievement scores and is called “between-school effects” that are frequently studied with a focus on differences in instructional resources. Starting with the American Coleman Report (1966) and the British Plowden Report (1971), a large research literature reports only modest between-school effects (and relatively stronger between-family SES effects). The large between-school effects once found in poorer nations, presumably with greater variation in school quality, have recently also declined (Baker, Goesling, LeTendre 2002). Given the global trend towards isomorphic operation of schooling including political pressure towards reduction of 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. Oreopoulos, Salvanes, 2009).
3. Timing of Education Revolution and the First Demographic Transition

A third reason for the under-appreciation of the educational revolution’s effect on population health is its relative exclusion from scholarship on independent causal factors behind historical demographic transitions.

The First Demographic Transition (hereafter, FDT), made up of the anthropologically novel decline in crude death rates and crude birth rates among populations in Western Europe and North America beginning in the 19th century, is considered a foundational transformation of population dynamics that heralded a new kind of society. Across demographical, economic, and medical historical 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 creating 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, and significant advances in scientifically derived medical technology occurred considerably after the onset of the FDT (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.

[Figure One about here]

Problematically for the materialist causal argument, the first stages of the industrial revolution in Western Europe began in the 18th century and by the mid-19th century had already made 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 economic arguments to reconcile cross-national variation in financial growth with the near universal trends of the FDT in the region. For example, like other scholars, Pritchett and Summers (1996) posit that improvement in household socioeconomic status is responsible for the reduction in mortality during this transition. Yet in England during the 1850s the real income was almost 50 percent above that of France, but this did not give the English population comparatively better health during this period (Caldwell, 2006).

The same is true for materialist arguments about science and medical technology. In the 18th and 19th century there were a number of medical breakthroughs that contributed to the decline in mortality including smallpox vaccination and germ theory, however the majority of the life-saving medical discoveries did not occur until after 1935 (Le Fanu 1999: xvii). Thus, the timing of the majority of medical interventions was decades after the already rapid and widespread decline in mortality. So too, arguments about improved nutrition within populations are weakened by inconsistencies of nations that shared similar nutritional profiles however had divergent mortality trajectories (McKeown 1976; Caldwell 1990).

By contrast, the timings of the educational revolution and the FDT are aligned—as increasing proportions of the population gained access to basic schooling, birth and death rates declined. The crude birth rate and crude death rate and primary and secondary school rates from 1816 to
1830 remained relatively stable, but from 1830 to 1850, as the primary and secondary enrollment rates per capita quickly increased, both birth and death rates began to rapidly decline. In addition to the nations that experienced the FDT, there is evidence that educational expansion among populations elsewhere in the world is temporally related to similar demographic transitions. For example, since the early 1950s, low- and middle-income countries experienced a rapid increase in life expectancy at birth from 40 years to 65 years by 2005, and starting just at the end of the World War II most of these nations began significant strides towards wide access to basic schooling (World Bank, 2007).

Further too, for the FDT and others since, it is generally observed that child mortality declines occur slightly before fertility declines, thus bringing into question initially popular causal arguments about higher material investment in the fewer children per household. Also this internal timing in demographic transitions tends to be a problem for the related argument that fertility decline is the 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 (Caldwell, 1979).

Certainly the FDT was likely due to an array of population factors (e.g. Kirk 1996), but if the pervasive education revolution was one cause of modernity, and if the main type of schooling spread so widely by the revolution potentially changes behaviors and attitudes, then this timing supports the consideration of education as one major cause of the FDT and similar demographic transitions occurring later in other parts world.

I. Meta-analysis of the Education Effect on All-causes Adult Mortality

Many studies in different nations across all levels of development show a relationship between education and health. In fact, even with relatively small amounts of formal schooling (2-3 years) in less developed regions, observable differences arise between schooled and non-schooled individuals in health behavior (de Walque, Nakiyingi-Miiro, Busingye, & Whitworth, 2005; Kenkel, 1991; Becker, Agopian, & Yeh, 1992). The association is not limited to developing contexts, but has also been widely observed in developed regions. Inequality in mortality gradients in a number of developed countries have also been attributed in part to inequalities in education for these countries (Kunst, Mackenbach, 1994). Even though this association is widely reported, to our knowledge there has never been a formal meta-analysis of the relationship between education and mortality that establishes its independence and size as an effect across the literature. While there are extensive literatures documenting the relationship between education and other measures of health than mortality, we have chosen to look at all-cause mortality among adults as a clearly defined and directly comparable outcome across developing and developed countries.

Procedure: A meta-analysis is not an all-inclusive review of studies; instead the technique statistically assesses the effect of an independent variable on a dependent variable (i.e. education on mortality), across studies employing the best methodological procedures possible (Glass, McGaw, & Smith, 1981). Meta-analysis can also statistically assess publication bias by estimating influences of study characteristics on the estimated effect sizes.
The sample for our meta-analysis is derived from all issues published between 1995 and 2005 of 22 demographic, social sciences, and health journals that were relevant studies. In order for a study to be included as a case in the meta-analysis it must meet the following criteria: 1) an original empirical study on adult mortality (e.g. re-analyses, reviews, comments, and editorials were excluded); 2) inclusion of formal education attainment of subjects as an independent variable; 3) a sample size of at least 100 adult subjects; 4) multivariate statistical conditioning on demographic factors such as gender, age, and residence; and, 5) statistical conditioning on either economic resources or socio-economic status. An initial search of titles and abstracts generated a list of 112 studies for consideration. Upon close examination, 51 study-cases met all the selection criteria (study-cases are marked with a * in the references section). Most study-cases include analyses of all-cause mortality of sub-samples, which are also analyzed below.

Among the selected study-cases, 16 (31%) were studies on North American samples, 21 (41%) on European samples, and 14 (27%) on Asian samples. Forty-four study-cases (86%) included at least 1,000 subjects; and 27 (53%) used nationally-representative samples of adults collected by a governmental agency, while all others used data collected by investigators.

As is common in meta-analyses, even though each study-case meets the selection criteria, there is variation among details of measurement and reported statistical indicators of effects that limit the degree to which they are statistically comparable (Allen & Preiss, 1990). For example, among the study-cases mortality is reported in several metrics including time to death, survival time, and cause of death. The study-cases also report effects of education as rate ratios, hazard ratios or relative risks, which are difficult to compare with odds ratios. Therefore, two levels of meta-analytic assessments of the education effect are reported. First, we report on the full sample of study-cases and then proceed to a more detailed analysis of only those studies that are fully comparable. Lastly, we assess the possibility of publication bias among these study-cases.

**[Table One about here]**

*Results from General Meta-Assessment:* Table 1 provides the pooled estimation of the effect sizes for the studies used in the analysis. The groups compared in the analysis for the relative risks are; low educated (no education or primary) against highly educated (middle school or tertiary education). Thus, the relative risk is interpreted as how much more probable is that a low educated person dies than a highly educated. Hence, the pooled effect size was calculated using all the studies (n=31), and was disaggregated by different sub-populations. The method used to calculate the pooled effect sizes was the random effect model proposed by DerSimonian and Laird (1986). This method adjusts the standard errors of each study to add the variation or diversity among them.

The overall effect of education on mortality has a relative risk of 1.42 (95% CI: 1.33 – 1.50) which suggests that people with no education or primary schooling have a 42% higher probability to die than highly educated people. The same pattern can be seen if we check each of the different pooled effect sizes estimated for the different groups, since each of the effect sizes are statistically significant (see Table 2).
Even after conditioning on economic resources and demographic characteristics, 46 (90%) of the study-cases show that exposure to formal education as a child reduces the risks of all-cause adult mortality (p<.05). Collectively, these study-cases represent approximately 20 million respondents from various nations. An illustrative example is Mostafa and van Ginneken’s (2000) longitudinal study on over 10,000 Bangladeshi adults from 1982 to 1999, which reports that after controlling for a host of demographic factors and socioeconomic status, unschooled subjects were 14% more likely to have died over the course of the study than schooled subjects.

**Results from Meta-Analysis of Most Comparable Effects of Education:** The fully comparative measurement and statistical methodology of 31 study-cases reported 142 educational effects on unique sub-samples, and across these we calculated a random effects estimation of the pooled effect of education on the relative risk of death across (DerSimonian and Laird 1987). As displayed in Figure 2, the pooled education effect size is 1.41 (p<.001), indicating that individuals with more than a primary education have almost a 40% reduction in the relative risk of death after conditioning on socio-economic status and other demographic characteristics.

**Analysis of Possible Publication Bias:** Before this estimate of the pooled education effects in Table 1 can be considered reliable, it is important to consider whether study characteristics are influencing the results. The influence of various characteristics of study-cases on effect sizes is examined by regressing the 142 education effects on unique sub-samples on nine study characteristics. Using the Egger Test (1997) of publication bias, we find little bias in the pooled effect size of education, as this overall indicator of publication bias is statistically insignificant (t=-.48, p=.63). There were two statistically significant study characteristics however both are modest in size. The study-cases using a nationally representative sample reported slightly higher education effect sizes and secondly earlier study-cases reported slightly smaller effects. Even in light of these modest biases, the general lack of publication bias confirms the conclusion that a consistent and robust effect of education is found throughout the research literature on all-cause mortality.

**III. Towards an Improved Hypothesis about the Education Effect**

Speculations about the causal mechanisms behind the association between education and health generally fall into one of two types. The first focus on a literal, but limited view of education by suggesting that schooling provides basic information about health with some literacy to do tasks such as read medical instructions and follow directions (Cleland and Van Ginneken, 1988). The second group of speculations suggests that formal education somehow instills new attitudes that lead to better health and longevity (see Hobcraft 1993 for review). These include individual psychological qualities such as delayed-gratification, changed interactive patterns within families, and an increased attitude of personal empowerment. As noted above, formal education likely transforms individuals in many ways and these ideas about schooling’s causal mechanism in health may be part of the answer, although these hypotheses have not been extensively tested. Yet the main limitation with these two types of causal arguments is that neither addresses the main activities of learning, keeping the schooling process a black box—literal interpretations of information-transfer though literacy peek into the box but go no further, and ideas about attitude change skip over primary learning processes all together.
At its core, schooling is a unique environment in which students spend long periods of time engaged in cognitive activities, and we propose that this fact is the key to understanding the causal mechanism behind the education effect on health. Learning to read, write a language, and use numbers, even under rudimentary conditions, are not only specific skills, each result from considerable abstract cognitive exercise that may transform how schooled individuals think, reason, and problem-solve (e.g. Martinez 1999). And it is this meta-cognitive enhancement from schooling that may prove to be the most powerful mechanism in creating the education effect.

Although there is a very large research literature on school achievement as the learning of the curriculum, until recently the potential for a meta-cognitive enhancement from literacy, numeracy, and knowledge acquisition has been less appreciated (Blair et al. 2004; Martinez 1999). So too, schooling has always included rote learning, memorization, and recitation that on the surface appear not to enhance cognitive thinking and reasoning skills. But as reviewed below, there is growing evidence that much of what occurs in the learning of school subjects imparts general cognitive abilities that are not readily found among otherwise normal unschooled individuals. And the evidence points to a robust meta-cognitive effect that often goes unnoticed since schooling is now so ubiquitous in many places. 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, anthropologically-speaking over most of the course of human society the large mass of non-elite people lived in a very concrete world. The relatively recent advent of mass education is the single largest non-family intervention for children, and a considerable extent of its core process is a cognitive intervention.

The Neurological and Cognitive Impact of Schooling

As reviewed above, the educational revolution has spread worldwide a relatively similar version of western schooling that includes significant potential for cognitive change in individuals. Therefore a cognitive hypothesis is a promising route to answer Caldwell and Caldwell’s (1979) question about how the education effect works: *Through the teaching of basic literacy, numeracy, and other academic subjects schooling enhances higher-order cognitive skills (such as reasoning, novel problem-solving, effortful thinking, and task planning) that in turn help individuals transform basic facts into deeper knowledge that enhances risk assessment and decision-making skills about health.* This proposes a Schooling-Enhanced Cognition-Health model of the widely reported education effect.

Recent research on neuro-development and higher-order cognitive skills establishes three interrelated sets of evidence pointing to cognitive effects of schooling as a plausible major causal mechanism behind the education effect on health:

1. **Neuro-development of high-order cognitive skills occurs at least through late adolescence and is highly responsive to environmental stimulation, such as that which routinely occurs in formal education.** Contrary to an older assumption that most neuro-development is finished before the child begins school, recent research using a variety of methodologies finds that higher-order cognitive skills are developed independently from general intelligence (Blair, 2006; Duncan, Burgess, & Emslie, 1995; Eslinger, Flaherty-Craig, & Benton, 2004; Shallice & Burgess, 1991;
The ability of functional magnetic resolution imaging (fMRI) technique 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 mathematics problems that require some reasoning from primary-school mathematics curricula, the brain areas associated with higher-order cognitive skills are activated (i.e. recruited activations in the superior parietal cortices most prominently, the dorsolateral prefrontal, occipital–temporal, and premotor/supplementary cortices, the basal ganglia, and insula) (Eslinger et al. 2009). The study also found a clear age effect in usage, younger students who did not readily know the answers elicited these high-order thinking areas more than older students who instead activated areas of the brain associated with long-term memory. Combining this type of evidence with the large research literature indicating that the structure and functions of the brain progressively emerge out of use, or what is called “activity-dependent development” or “neural plasticity” (e.g. Hubel and Weisel 1962; Quartz and Sjenowski 1997). This suggests that even simple cognitive activities during earlier schooling can have profound effects on neurological functioning.

2. Exposure to schooling is monotonically and linearly associated with enhanced higher-order cognitive skills. Research comparing normally-developed schooled and unschooled adults, finds that within a genetically determined range of capability, schooling can significantly enhance meta-cognitive skills such as categorization, logical deduction, and IQ (e.g. Cole 2003, Luria 1976, Nisbet 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 .3 to .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 finds that even small amounts of schooling as a child yields higher-order cognitive skills among adults net of work conditions and more general social and economic status (Christian, Bachman, & Morrison, 2001; Cole 2003, Luria 1976, Stevensen et al., 1991; Stevenson, Chen, & Booth, 1990).

3. Higher-order cognitive skills are associated with better risk assessment and decision-making skills. Experiments on risk assessment and the use of effective heuristics for decision making repeatedly find that such skills are positively associated with numeracy and higher-order cognitive skills (e.g. De Bruine de Bruin, W., Parker, A., Fischhoff, B. 2007; Peters et al 2006). Risk assessment and decision-making skills are an important component of reasoning about health and deciding on protective behavior.

In addition to the neurological evidence on the meta-cognitive effects of schooling, two recent studies test the Schooling-Enhanced Cognition-Health model. The first study used structural equation modeling of Demographic Health Survey data from nine sub-Saharan African nations, and found that, net of control variables, there is a robust, positive effect of education (ranging from unschooled to some secondary schooling) on condom use among sexually risky adults (Baker, Leon, Collins in-print). Although formal education increases 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.

In the second study, data collected from a naturally-occurring experiment on school attendance among 181 rural unschooled and schooled Ghanaian adults found education as a child was associated with current reports of taking more protective health behaviors (Peters et al. in-print). 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.

Conclusion

The spread of education and the increase in the duration of expected years of school attendance is a profoundly new social activity that continues to have far reaching consequences for population development. An assessment of findings from a diverse set of disciplines over the past three decades concludes that the education revolution was a central cause in the rise of the modern society in general and the FDT specifically, which are two large-scale transitions that have significantly improved human health over the past century. As illustrated by the meta-analysis of recent studies of education and all-cause mortality, exposure to schooling is a dominant covariate of an individual’s life expectancy, and given the extensive statistical conditioning on other factors of individuals in these studies, it is likely that formal education is a primary cause of health. Lastly, the Schooling-Enhanced Cognition-Health model put forth as a potential explanation of education’s direct causal effect on health is compatible with the main discoveries from research on neuro-development and the psychology of decision making. Further, the hypothesis is consistent with a small, but uniform, set of findings on schooling effects on basic cognitive skill enhancement and in turn on health behaviors. Taken as a whole, this new way to think about the education effect and health offers several advantages for future population science.

First, an appreciation of the causal influence of formal schooling in population dynamics related to health is potentially a key insight into a broader model of the impact of the education revolution on human society. For example, sociological research on status attainment has also tended to treat schooling as a black box. Although educational attainment, both of parents and children, is now the central stratifying factor of individuals in these studies, it is still often considered mostly a proxy for social class in sociology (Baker 2009). The theoretical focus is on “attainment” to the extent that education’s only function is in providing a type of master credential for use in the labor market, and given that individual educational attainment is associated (although far from perfectly) with social origin, much is made of the social status reproductive nature of education, although the there is a recent trend of disconnection of social origin and adult status attained through education (Baker forthcoming; Hout 1988; Torche 2010). Education does indeed play a central role in social status attainment, but this does not rule out the possibility that education can play additional causal roles based more on what happens in the black box of schooling. The effect of exposure to schooling on health, net of what educational attainment might afford in social status and material resources, is illustrative of this and is an important issue for further research in population science. Also, in addition to health, future research on the education effect on other demographic processes would
lend even more credence to this broader model of education’s role in the formation of society.

Second, a broader theoretical model of how education can influence health can generate better explanations of seemingly paradoxical empirical patterns in population trends in health. For example, early in the HIV/AIDS pandemic in sub-Saharan Africa widespread misinformation about viral transmission suppressed the cognitive effect of education, and as a result more educated males with access to more social and material resources leading to sexual opportunities were infected at such a high rate that education was declared a risk factor (e.g. Watkins and Swidler 2009). But as biologically and medically correct information entered the region, education among younger cohorts turned to its usual function as a social vaccine and lower infection rates (Baker, Collins, Leon 2009). The indirect effects of education through increased social and material status likely caused the risk factor trend, while the direct effect of education through better reasoning about risk and disease has likely led to the shift to education acting as social vaccine later on in the pandemic (Peters et al. in-print).

Third, over the past fifty years a significant intellectual undertaking has been the extensive scientific study of cognition and neurological development, dubbed the “cognitive revolution” (Miller 2003). And much like sociology and until recently economics, by and large population science has not taken advantage of this growing understanding of human neurological capacity. The centrality of education effects on health at both the individual and population levels along with the Schooling-Enhanced Cognition-Health model offers one avenue for population science to explore and benefit from the findings and methods of the cognitive revolution.

Fourth, going beyond measuring educational attainment is a potential future advantage for population science. Since so much demographic research on health is conducted on data from large omnibus surveys, a deeper appreciation of the education effect suggests that one innovation would be to include cognitive measures in these surveys related to the Schooling-Enhanced Cognition-Health model. Just recently, measures of literacy have been incorporated to the Demographic and Health Survey. Similarly there are a number of relatively easy-to-administer reliable and valid measures of basic executive functioning skills in terms of working memory, spatial relationships, and reasoning, and numeracy. Performance on cognitive tests are known to be associated with prior education and current decision making skills (e.g. Baker, Leon, Collins in-print; Peters et al. in-print). Incorporation of such cognitive tasks would allow further analysis of cognition as a mechanism through which education influences health and move population science towards a great understanding of how education is a catalyst of social change.

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i Declining variation in life expectancy is found worldwide except for sub-Saharan Africa and the region was withheld from this calculation (See Goesling and Baker 2008 for details).

iii Populations studied in the 51 study-cases are from Bangladesh, Belgium, Canada, Denmark, England, Estonia, Finland, France, Germany, India, Italy, Israel, Japan, Korea, Norway, Russia, Spain, Sweden, Switzerland, Taiwan, and The Netherlands.

iv In 3 of study-cases reporting a statistically significant effect, the education effect was reduced to non-significant when additional controls were added to subsequent models that were positively correlated with an individual’s education: spousal education (Egeland 2002), occupation (Van Rossum et al 2000, McDonough et al. 1997). And in 5 study-cases an initial education effect turned non-significant when measures of current health (all related to prior education) were added to a subsequent model (Seeman et al 2004, Kilander et al 2001, Liu et al. 1998, Rogers 1995, Zimmer et al. 2005).

v Because study-cases differ as to their national-context, sampling design, and sample size estimation of pooled education effects used a random effect model, which assumes a distribution of effect sizes instead of one true effect size assumed by a fixed effect model.

vi The 9 characteristics are nationally represented sample, world region, gender studied, sample size, cause of death studied, modal age in sample, missing age cohort, longitudinal design, a missing education category. Detailed results are available upon request from authors.

vii The 9 characteristics are nationally represented sample, world region, gender studied, sample size, cause of death studied, modal age in sample, missing age cohort, longitudinal design, a missing education category. Detailed results are available upon request from authors.

viii An assessment of the information-transfer and attitude-change consequences of schooling is beyond the scope of this paper, but other research finds that these outcomes have a weak influence on health knowledge and proactive behavior strategies, particularly in comparison to cognitive outcomes.
Figures and Tables
Table 1. Pooled Relative Risk Ratios from 142 Reported Education effect Sizes from 31 Study-cases*

<table>
<thead>
<tr>
<th></th>
<th>Number of effect sizes</th>
<th>ES (RR)</th>
<th>ES - log(RR) -</th>
<th>Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Lower bound</td>
</tr>
<tr>
<td>All samples</td>
<td>142</td>
<td>1.41</td>
<td>0.35</td>
<td>1.33</td>
</tr>
<tr>
<td><strong>By gender</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Only females</td>
<td>59</td>
<td>1.40</td>
<td>0.34</td>
<td>1.35</td>
</tr>
<tr>
<td>Only males</td>
<td>66</td>
<td>1.38</td>
<td>0.32</td>
<td>1.25</td>
</tr>
<tr>
<td>Male &amp; females</td>
<td>17</td>
<td>1.40</td>
<td>0.34</td>
<td>1.24</td>
</tr>
<tr>
<td><strong>By age</strong>*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Middle age and senior samples</td>
<td>120</td>
<td>1.45</td>
<td>0.37</td>
<td>1.36</td>
</tr>
<tr>
<td>Only senior sample</td>
<td>16</td>
<td>1.17</td>
<td>0.16</td>
<td>1.07</td>
</tr>
<tr>
<td><strong>By continent</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>America</td>
<td>13</td>
<td>1.35</td>
<td>0.30</td>
<td>1.23</td>
</tr>
<tr>
<td>Europe</td>
<td>86</td>
<td>1.33</td>
<td>0.28</td>
<td>1.30</td>
</tr>
<tr>
<td>Asia</td>
<td>43</td>
<td>1.60</td>
<td>0.47</td>
<td>1.28</td>
</tr>
<tr>
<td><strong>By type of mortality</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All causes</td>
<td>97</td>
<td>1.30</td>
<td>0.52</td>
<td>1.28</td>
</tr>
<tr>
<td>Specific mortality</td>
<td>45</td>
<td>1.68</td>
<td>0.27</td>
<td>1.41</td>
</tr>
</tbody>
</table>

*The DerSimonian and Laird method was used to calculate the pooled effect sizes using a random effects approach.

Table 2. Meta-regression analysis to evalulate bias in the effect sizes

<table>
<thead>
<tr>
<th></th>
<th>Coef.</th>
<th>SE</th>
<th>RR</th>
<th>Confidence interval (RR)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Lower</td>
</tr>
<tr>
<td>All causes of mortality</td>
<td>-0.11</td>
<td>0.08</td>
<td>0.90</td>
<td>-0.27</td>
</tr>
<tr>
<td>National sample</td>
<td>0.23</td>
<td>0.08</td>
<td>1.26</td>
<td>0.08</td>
</tr>
<tr>
<td>Europe</td>
<td>0.16</td>
<td>0.09</td>
<td>1.17</td>
<td>-0.03</td>
</tr>
<tr>
<td>Asia</td>
<td>0.14</td>
<td>0.11</td>
<td>1.15</td>
<td>-0.08</td>
</tr>
<tr>
<td>More than 10,000 people</td>
<td>-0.01</td>
<td>0.08</td>
<td>0.99</td>
<td>-0.17</td>
</tr>
<tr>
<td>Female sample</td>
<td>-0.12</td>
<td>0.09</td>
<td>0.89</td>
<td>-0.29</td>
</tr>
<tr>
<td>Male sample</td>
<td>-0.16</td>
<td>0.09</td>
<td>0.85</td>
<td>-0.33</td>
</tr>
<tr>
<td>Unspecified educational levels</td>
<td>-0.10</td>
<td>0.07</td>
<td>0.90</td>
<td>-0.24</td>
</tr>
<tr>
<td>Older cohort (above 60)</td>
<td>-0.35</td>
<td>0.09</td>
<td>0.70</td>
<td>-0.52</td>
</tr>
<tr>
<td>Missing age cohort</td>
<td>-0.13</td>
<td>0.13</td>
<td>0.88</td>
<td>-0.38</td>
</tr>
<tr>
<td>Longitudinal study</td>
<td>-0.13</td>
<td>0.08</td>
<td>0.88</td>
<td>-0.28</td>
</tr>
<tr>
<td>Constant</td>
<td>0.36</td>
<td>0.15</td>
<td>1.43</td>
<td>0.06</td>
</tr>
</tbody>
</table>

*Adjusted R-square: .28

F (11,130): 5.06 (.000)
## APPENDIX

### Studies included in the Meta-analysis

<table>
<thead>
<tr>
<th>N</th>
<th>Year of publication</th>
<th>Author(s)</th>
<th>Country(ies)</th>
<th>Type of mortality</th>
<th>Age group</th>
<th>Number of effect sizes</th>
<th>Effect size or Range of the effect sizes (RR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2002</td>
<td>Grace M Egeland et al</td>
<td>Norway</td>
<td>Coronary heart disease</td>
<td>35-56</td>
<td>1</td>
<td>1.00</td>
</tr>
<tr>
<td>2</td>
<td>2001</td>
<td>Pelka Martikainen et al</td>
<td>Finland</td>
<td>Lung cancer</td>
<td>50-69</td>
<td>1</td>
<td>1.32</td>
</tr>
<tr>
<td>3</td>
<td>2002</td>
<td>M Osler et al</td>
<td>Denmark</td>
<td>All causes</td>
<td>Not specified</td>
<td>2</td>
<td>1.02-1.13</td>
</tr>
<tr>
<td>4</td>
<td>2003</td>
<td>M. A. Winkleby et al</td>
<td>United States</td>
<td>All causes</td>
<td>25-64</td>
<td>6</td>
<td>1.05-4.76</td>
</tr>
<tr>
<td>5</td>
<td>2002</td>
<td>E. Regidor et al</td>
<td>Spain</td>
<td>Infectious diseases</td>
<td>35-64</td>
<td>4</td>
<td>2.41-2.82</td>
</tr>
<tr>
<td>6</td>
<td>2002</td>
<td>M Son et al</td>
<td>Korea</td>
<td>Work related (Manual &amp; Non-manual workers)</td>
<td>20-64</td>
<td>4</td>
<td>1.84-5.37</td>
</tr>
<tr>
<td>7</td>
<td>1999</td>
<td>Eric Backlund et al</td>
<td>United States</td>
<td>All causes</td>
<td>25-64</td>
<td>2</td>
<td>1.33-1.42</td>
</tr>
<tr>
<td>8</td>
<td>2004</td>
<td>Johan P. Mackenbach et al</td>
<td>England, Norway, Denmark, Finland, Belgium, Switzerland</td>
<td>All causes</td>
<td>40-89</td>
<td>20</td>
<td>1.17-1.42</td>
</tr>
<tr>
<td>9</td>
<td>2003</td>
<td>Mail Leinsala et al</td>
<td>Estonia</td>
<td>All causes</td>
<td>20 or more</td>
<td>2</td>
<td>1.37-2.23</td>
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<tr>
<td>10</td>
<td>1997</td>
<td>Cameron A. Mustard et al</td>
<td>Canada</td>
<td>All causes</td>
<td>0 or more</td>
<td>1</td>
<td>1.38</td>
</tr>
<tr>
<td>11</td>
<td>2004</td>
<td>Orly Manor et al</td>
<td>Israel</td>
<td>Heart diseases: cardiovascular, ischemic heart, cerebrovascular,</td>
<td>45-89</td>
<td>30</td>
<td>0.80-9.68</td>
</tr>
<tr>
<td>12</td>
<td>2004</td>
<td>F.V.A. van Oort et al</td>
<td>Netherlands</td>
<td>All causes</td>
<td>15-54</td>
<td>2</td>
<td>1.27-1.66</td>
</tr>
<tr>
<td>13</td>
<td>2002</td>
<td>Judith A. Long et al</td>
<td>United States</td>
<td>All causes</td>
<td>72 or more</td>
<td>1</td>
<td>0.50</td>
</tr>
<tr>
<td>14</td>
<td>2004</td>
<td>Sofia Malutina et al</td>
<td>Russia</td>
<td>All causes</td>
<td>25-64</td>
<td>6</td>
<td>0.89-1.41</td>
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<tr>
<td>15</td>
<td>1997</td>
<td>Jan Sundquist et al</td>
<td>Sweden</td>
<td>All causes</td>
<td>25-64</td>
<td>2</td>
<td>1.29-1.72</td>
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<tr>
<td>16</td>
<td>2001</td>
<td>Lena Kilander et al</td>
<td>Sweden</td>
<td>All causes</td>
<td>50</td>
<td>1</td>
<td>1.01</td>
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<tr>
<td>17</td>
<td>2004</td>
<td>Roberto Gnavi et al</td>
<td>Italy</td>
<td>All causes</td>
<td>20 or more</td>
<td>4</td>
<td>1.14-1.7</td>
</tr>
<tr>
<td>18</td>
<td>2002</td>
<td>Tiina Pennola et al</td>
<td>Finland</td>
<td>All causes</td>
<td>30-34</td>
<td>2</td>
<td>2.49-2.90</td>
</tr>
<tr>
<td>19</td>
<td>2003</td>
<td>Peter Franksa et al</td>
<td>United States</td>
<td>All causes</td>
<td>21 and over</td>
<td>1</td>
<td>1.14</td>
</tr>
<tr>
<td>20</td>
<td>2003</td>
<td>Maria Fencia Vescio et al</td>
<td>Italy</td>
<td>All causes</td>
<td>20-75</td>
<td>4</td>
<td>0.82-1.33</td>
</tr>
<tr>
<td>21</td>
<td>2003</td>
<td>Martin Bobuk et al</td>
<td>Russia</td>
<td>All causes</td>
<td>Not specified</td>
<td>4</td>
<td>1.21-2.61</td>
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<tr>
<td>22</td>
<td>2005</td>
<td>Giovani Menveli et al</td>
<td>France</td>
<td>All causes</td>
<td>35-59</td>
<td>6</td>
<td>1.20-2.10</td>
</tr>
<tr>
<td>23</td>
<td>2005</td>
<td>DH Jaffe et al</td>
<td>Israel</td>
<td>All causes</td>
<td>70 or more</td>
<td>4</td>
<td>1.02-1.27</td>
</tr>
<tr>
<td>24</td>
<td>2000</td>
<td>Augustine et al</td>
<td>United States</td>
<td>Suicide</td>
<td>15 or more</td>
<td>1</td>
<td>1.50</td>
</tr>
<tr>
<td>25</td>
<td>2005</td>
<td>Martijn Haakman et al</td>
<td>Finland, Norway, England, Belgium, Austria, Switzerland</td>
<td>All causes</td>
<td>45 or more</td>
<td>16</td>
<td>1.20-1.43</td>
</tr>
<tr>
<td>26</td>
<td>2005</td>
<td>Young-Ho Khang et al</td>
<td>Korea</td>
<td>All causes</td>
<td>30 or more</td>
<td>3</td>
<td>1.60-2.15</td>
</tr>
<tr>
<td>27</td>
<td>1999</td>
<td>Orly Manor et al</td>
<td>Israel</td>
<td>All causes</td>
<td>45-89</td>
<td>2</td>
<td>0.98-1.11</td>
</tr>
<tr>
<td>28</td>
<td>2003</td>
<td>T. Laitikainen et al</td>
<td>Finland</td>
<td>All causes</td>
<td>25-64</td>
<td>1</td>
<td>1.57</td>
</tr>
<tr>
<td>29</td>
<td>1999</td>
<td>Hans Munkel</td>
<td>Finland</td>
<td>Alcohol related</td>
<td>35-69</td>
<td>4</td>
<td>1.36-1.86</td>
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<tr>
<td>30</td>
<td>2000</td>
<td>Caroline TM Van Rossum et al</td>
<td>Netherlands</td>
<td>All causes</td>
<td>55 or more</td>
<td>4</td>
<td>1.20-1.70</td>
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<td>31</td>
<td>1997</td>
<td>Peggy McDonough et al</td>
<td>United States</td>
<td>All causes</td>
<td>45-64</td>
<td>1</td>
<td>1.35</td>
</tr>
</tbody>
</table>
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NATIONAL BUREAU OF ECONOMIC RESEARCH


Plavinskaya, S. L. & Klimov, A. N. (2003) Social factors and increase in mortality in Russia in the 1990s: Prospective, British Medical Journal, 326, 7401; Health Module pg. 1240


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