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ABSTRACT

In this review we synthesize what is known about the relationship between education and health. A large number of studies from both rich and poor countries show that education is associated with better health. While previous work has thought of the effect of education separately for rich and poor countries, we argue that there are insights to be gained by integrating the two. For example, education is associated with lower malnutrition in most countries, but in richer countries the educated have lower BMIs whereas in poor countries the educated have higher BMIs. This suggests that the behaviors associated with better health differ depending on the level of development. We illustrate this approach by comparing the effects of education on various health and health behaviors around the world, to generate hypotheses about why education is so often (but not always) predictive of health. Finally, we review the empirical evidence on the relationship between education and health, paying particular attention to causal evidence and evidence on mechanisms linking education to better health.

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In their seminal 1965 study, Kitagawa and Hauser documented that mortality in the United States fell with education. Since then a very large number of studies has confirmed the more educated enjoy longer lives: Cutler et al. (2008) for example compute that in 1980 at age 25 those with some college could expect to live another 54.4 years whereas life expectancy at 25 for those with a high school degree or less was only 51.6.

Not only are the differences in health by education large, by most measures these differences have been growing in recent years. For instance in 2000 those with some college lived 7 years longer than high school dropouts—thus the gap increased by 4 years since 1980 (Cutler et al 2008).¹ Education not only predicts mortality in the US, it is also a large predictor of health in most countries, regardless of their level of development (Strauss and Thomas, 1995). The more educated are also noticeably healthier while they are alive, as they report being in better health, having fewer health conditions and limitations (Cutler and Lleras-Muney 2010a, b). Children of educated parents are also in better health in both developed and developing countries (Strauss and Thomas 1995, Schultz 2007).

In this review we synthesize what is known about the relationship between education and health in both developed and developing countries. While previous work has thought of the effect of education separately for rich and poor countries, we argue that there are insights to be gained by integrating the two. For example, education is associated with lower mortality in most developed countries, and this relationship is similar regardless of the generosity of the social protections and health insurance systems that are in place. This suggests that access to care is not

¹ Other papers have documented increased gaps by education in other countries as well, for example education gradients in mortality since 1980 increased in Estonia (Leinsalu et al 2007), Sweden, Finland and Norway (Shkolnikov et al 2011), Russia (Plavinski et al 2003, Shkolnikov et al 1998), Denmark, England/Wales, and Italy (Machenback et al 2003). Caution must be taken when interpreting these trends however, as the number and composition of individuals within education categories has also changed substantially over time.

the main reason for the association in the first place. We illustrate this approach by comparing the effects of education on various health and health behaviors around the world to generate hypotheses about why education is so often (but not always) predictive of health.

We go on to examine theories for the relation between education and health. We then review the empirical evidence on this relationship paying particular attention to causal evidence and evidence on mechanisms linking education to higher health.

I. Stylized facts about education and health

To examine the link between education and health across countries, we combine data from three sources. Data on for most developing countries come from the Demographic and Health Surveys (DHS) for years between 2004 and 2009. Data for the US come from the Behavioral Risk Factor Surveillance System (BRFFS) for 2005. Data for Europe come from the Eurobarometer Surveys (2005 and 2009). We have a total of 61 countries in total. We matched each country to its per capita level of GDP in current US dollars reported by the World Bank. To create a consistent sample, we restrict attention to women ages 15-49 (the DHS does not collect data on men or older women). More details on the data construction are in the Data Appendix.

Education is measured as years of school in the DHS and the BRFFS, but the Eurobarometer only asks about the age at which a person finished schooling. We assume that years of schooling in the Eurobarometer data is five years less than age at which schooling was finished. Because some people take significant time off before finish schooling, we truncate schooling at 25 years. Although not ideal, this is the only standardized data source with a large number of countries.

For all of these countries we have measures of height (in centimeters) and weight (in kilos), which we use to construct Body Mass Index (BMI=weight/height squared), an indicator for being underweight (BMI<=18.5) and an indicator for being obese (BMI>=30). The data from the DHS comes from actual measures whereas the data for the US and Europe are self-reported. For all of the countries we also know whether the person is currently a smoker. For a few developing countries and all developed countries we know whether the person drinks alcohol. Finally only for developing countries, we have measures of hemoglobin levels and a measure of whether the person had a sexually transmitted disease in the last year.

To document basic patterns in the relationship between education and health, we estimate the following regression for each country in the sample:

$$(1) \quad H_{ic} = \beta_0 + \beta_{1c} * Education_{ic} + \mathbf{X}_i \alpha + \varepsilon_i$$

Where H_i is a health or health behavior indicator of individual i in country c , $Education$ is measured in years, and X_i contains basic demographics: age, age squared, marital status, ethnicity, race and religion dummies. For each country and outcome we obtain the regression coefficient β_{1c} , which we plot by level of GDP (in logs). All of the surveys have complex sampling design schemes, and we use the weights provided by the survey to compute means and to weight regressions.

It is difficult to interpret the coefficient of education in these regressions as causal since education and health could be both determined by unobservable factors. Also the coefficient on education might reflect the effect of health on schooling rather than the reverse. We discuss these issues much more below. For the time being we describe the correlations that we observe and hypothesize on the reasons for the patterns across countries.

We estimate linear models and look at the effect of years of schooling across all countries. It is not clear that years of schooling are comparable across countries since the quality of education differs widely by country and thus the actual education of individuals might differ even when years of school are comparable. Ideally one would use test scores or other measures of achievement (such as literacy and numeracy), as suggested by Hanusheck and Woessmann (2009), but these are not available here, or in most surveys. Also one might prefer to look at non-linear models, where the effect of education is allowed to vary depending on the level of education. Previous research has generally found that linear models are good approximations (Cutler and Lleras-Muney 2010a) though this refers to high income countries. Nevertheless our estimates are of interest since they mirror the standard estimates that are produced when looking at specific countries and times. Also we are restricting our attention to women because the DHS surveys collect information systematically on them but not necessarily for men. Prior research documents that correlations between education and health are similar for men and women, though in general correlations are stronger for men but this varies depending on the outcome (Cutler and Lleras-Muney 2010a).

Figure 1 shows the education gradient in BMI as it relates to average income. BMI is generally taken as an indicator of short term nutrition. The figure suggests a clear pattern by income: in poor countries, those with more education have higher BMIs whereas the opposite is true in rich countries. The crossover point is income of about \$3,000 per capita, roughly the income of Bolivia and Peru. However, the relationship between health and BMI is not monotonic: Higher weight (given height) is associated with lower mortality at low levels of weight, but after some threshold increased weight is associated with larger mortality. To

disentangle these effects we look at the effect of education on the likelihood of being underweight and on the likelihood of being obese: both of these are indicators of poor health.

Figure 2 shows the patterns for underweight. Overall education is associated with a decrease in undernutrition: most coefficients are either negative and statistically significant, or essentially zero (though there are a few exceptions). The effect of education is largest for the poorest countries and then becomes zero (or positive) as GDP rises. This is essentially due to the fact that there is very little undernutrition in countries that have reached middle levels of income, and there is no effect of education on malnutrition when the prevalence rates are low. This is more evident in Figure 3 which plots education coefficients against levels of malnutrition (the share of the population that is underweight).

Figure 4 shows the patterns for obesity. These patterns are very similar to the patterns for BMI: in poor countries, the effect of education on obesity is positive and significant, whereas it becomes negative and significant for richer countries. This pattern has been noted before (Stunkard and Sobel 1987, Stunkard 2007, Strauss and Thomas 1998) and it is more marked for women than men (our graphs only show patterns for women). Thus we observe that around the world more educated avoid malnutrition, but not always obesity. It is possible that when levels of nutrition are low, obesity is associated with increased survival since people are better able to fight infectious disease, and chronic problems are not large killers. But once infectious diseases fall, and chronic conditions become more important, the pattern reverses (conditional on knowledge that obesity is bad). It is also possible that girth is a status symbol or symbol of wealth in societies that are poor; but that in rich societies where knowledge of the health consequences is widespread, the opposite becomes true, as rich individuals will devote their resources to staying thin and fit. But the data strongly suggests that the effect of education

depends on the level of development and in particular where in the “nutrition transition” countries are.

Figure 5 shows the patterns for hemoglobin levels by income—though only for developing countries. Again we find that the effect of education is protective at low levels of income, and then decreases with GDP; this is again a function of the fact that on average hemoglobin levels rise with GDP. So in poor countries the more educated avoid malnutrition. But Figure 6 shows they do not always avoid disease: among very low income countries, there are more countries where education is associated with a higher incidence of STIs than countries where education is protective. But there is a trend by income again: education is more likely to be protective for higher levels for GDP. Recent work that looks at sexual behavior responses by education level in Africa also reports that the “effect of education” varies depending on the stages of the HIV epidemic (Iorio and Santaaulalia-Llopis 2011).

Figure 7 shows the patterns for the effect of education on smoking, the leading cause of preventable deaths worldwide (World Health Organization, 2009). In general the effect of education on smoking is negative, but for the poorest countries the coefficients tend to be very small. Also for many middle income countries there is a positive effect of education. It is unlikely that this reflects differential knowledge of the harms of smoking among the better educated. The danger of cigarette smoking is well known around the world even in the poorest countries: for example in Bangladesh 93% of smokers report that smoking causes lung cancer (International Tobacco Control Policy Evaluation Project). Rather, it may reflect the social acceptability of smoking as income increases, or the onset of public policies to reduce smoking at very high incomes. Also possible is that in some countries the effects of knowledge are counteracted by the effects of higher incomes, since smoking is a normal good. Again these

patterns suggest that the effect of education on smoking depends on the level of development defined both in terms of income and knowledge and will therefore vary over time and space. Table 1 presents some evidence of this “smoking transition” for the US. In 1949, high school dropouts are less likely to smoke than high school graduates or those with more education—the opposite of what we observe today. At this time dropouts also are more likely to think smoking is harmful. But between 1950 and 1970 the more educated became more likely to think smoking was harmful as knowledge of the harms of smoking emerged; and by 1969 they are also less likely to smoke.

Figure 8 shows the patterns for drinking. We do not have data on drinking for many developing countries, so we examine somewhat higher income countries. Alcohol appears to be a normal good. Education increases the odds of drinking alcohol in almost all the countries we examine. Modest alcohol consumption might not be detrimental to health so it is not necessarily clear that these coefficients have the “wrong” sign. Ideally we would study whether education lowers heavy drinking, which does fall with education levels in the US and the UK (Cutler and Lleras-Muney 2010a), but the data are not consistently available across countries.

The previous figures suggest important patterns by education, and could be taken as reflecting causal relationships from education to health. However we can also document here that education is partly determined by health by looking at height. Height is generally thought of as an excellent indicator of early childhood environment, as much of the variation in adult heights is determined by age 3 (Martorell and Habicht, 1986). Thus the coefficients of education on height from equation (1) most likely reflect the effect of health on the quantity and quality of education

individuals obtain, rather than the effect of education on final height.² Figure 9 shows the results for height. For almost all the countries we examine more educated people are taller and the relationship is generally statistically significant. And though the effect falls a bit with GDP, education is still very strongly associated with height, even in very rich countries (with a couple of interesting exceptions among the richest countries).

Summary

All told, the international data on health and education show several stylized facts. The clearest relationship is between income and the education gradient in nutritional intake. Poor countries are characterized by a mix of undernutrition and overnutrition. Many people are undernourished or anemic in poor countries, and these outcomes are strongly negatively related to education. Less educated people are more likely to be underweight and anemic; better educated people are more likely to be overweight or obese. In richer countries, where undernutrition is not very prevalent, there is no education gradient in undernutrition. In contrast in these countries the prevalence of obesity is large and there is a large positive education gradient in obesity. This suggests that education is protective for the outcomes that are known to be bad for health.

The link between education and height is also clear. In all countries – even the richest – better educated people are taller than less educated people. The magnitude of the relationship is large throughout the world.

² However, though most of height is determined at very young ages, there is another critical growth period in adolescence where the remaining differences in final adult height are determined. Thus it is possible some of the relationship is due to an effect of schooling on height.

The link between education and other measures of health is much less clear. The correlation between education and smoking is non-linear in income; the relationship between education and height or STIs is unrelated to income. These patterns demand more complex explanations.

II. Understanding the relationship between education and health

Education and health may be related for three reasons: poor health early in life may lead to less educational attainment; lower educational attainment may adversely affect subsequent health; or some third factor such as differences in discount rates may affect education and health-seeking behavior. We briefly discuss each of these pathways; more discussion is contained in our prior work (Cutler and Lleras-Muney, 2010a, b).

We start by emphasizing the most important common unobserved determinants of both education and health. The first is parental resources: parents with more resources (broadly construed to include wealth, social networks, knowledge, etc.) will devote part of them to improving the survival of their children (by investing in their health) and also to improving their future outcomes, which in turn means they will invest perhaps more on their children's education. Secondly, there are some important individual characteristics that theoretically are expected to increase both education and health. *Ceteris paribus*, more patient individuals are more likely to invest more in both education and health (Fuchs 1982). Also smarter individuals might be more likely to obtain more schooling and also have better health.

Effect of early life health on education

As the previous results indicate, there is a very strong correlation between early life indicator of health (such as height) and educational attainment—and this is true across all countries of the world. These correlations have been documented many times before, particularly in developing countries (Strauss and Thomas 1998). Since education is largely determined at young ages, this suggests that at least part of the correlation between education and health among adults is due to the fact that unhealthy children obtain few years of schooling and become unhealthy adults.

Recent studies show that the relationship we observe – shorter (and sicker) children obtain less education – is a causal one. Two types of studies investigate the causal effect of health shocks on human capital accumulation: some take advantage of so-called “natural experiments,” while others use randomized controlled trials to investigate the question.

Most studies that investigate this causal chain find support for it. For instance, Almond (2006) documents that individuals affected *in utero* by the 1918 influenza pandemic obtained fewer years of schooling than those not affected. Meng and Qian (2006) find that those born during the Great Famine in China had lower educational achievement than those born not during the Great Famine. Bleakley (2009) reports that malaria eradication in the US and various Latin American countries resulted in greater education, and Lucas (2009) finds similar effects of the eradication malaria campaigns in Paraguay and Sri Lanka; Maccini and Yang (2009) show that in Indonesia unusual amounts of rainfall in utero predict lower education levels. Finally Bleakley (2007) and Miguel and Kremer (2004) show that deworming campaigns had substantial effects on schooling in early 20th century American South and in Kenya respectively. Only the recent quasi-experimental study by Cutler et al. (2009) finds no effects of malaria eradication in India.

A related literature explores the consequences of birth weight on adult outcomes and finds similar results suggesting that those born with lower birthweights have lower levels of education, income and health as adults (Case et al., 2005, Behrman and Rosenweig 2004, Black, Devereaux and Salvanes, 2007). Though these studies do not directly look at nutrition, but rather at extreme events that influence birth weight, in many cases nutrition and disease are the most likely intervening mechanisms.

Direct evidence on the effect of nutrition and disease on education is available from several randomized experiments. For example Bobonis et al. (2006), find that iron supplementation increases pre-school attendance in India, and Field et al. (2007) find that iodine supplementation *in utero* increases educational attainment. Hoddinott et al., (2008) and Maluccio et al. (2009) report that nutritional supplements before the age of 3 increased education and earnings in adulthood. Clarke et al (2008) show that treated children who randomly received malaria treatment in Kenya had improved health and increased cognitive ability.

Whether early life health affects education through morbidity at younger ages or expectation of life extension at older ages is unknown. Indeed, there is scant evidence on the extent to which expectations of longer life affects schooling. Jayachandran and Lleras-Muney (2010) find that when maternal mortality fell in Sri Lanka, girls' education increased (but not that of boys). But they cannot compute how much of the education-life expectancy relationship is accounted by this channel.

Overall, the evidence is consistent in showing that nutrition and disease shocks early in life are quite detrimental for human capital formation. Interestingly, a negative link between early life health and educational attainment is not the only theoretical possibility, since health affects the costs and benefits of schooling (Bleakley 2010). Sickness increases the cost of going

to school in terms of effort, and might also lower the returns to school if it lowers life expectancy. Thus, parents of sick children might optimally choose lower levels of schooling for those children. However illness also increases the cost of work, and might increase the returns to school (in terms of avoiding more physically demanding jobs). Thus it could be that the return to schooling increases as people become less healthy. However there is no empirical evidence of this alternative, though perhaps it explains why education and height are negatively related in two very rich countries: Finland and Luxembourg (see Figure 9). This discussion also underscores the fact that the observed relationship between height and education reflects not only the physical effects of disease in childhood, but also the behavioral responses of parents which might attenuate or exacerbate the effects of the health shock itself.

Given that health is an important determinant of schooling, and the fact that education and health could simply be determined by common factors such as parental resources, it is extremely challenging to document whether in addition to these well documented relationships, education itself affects health – the question we consider next.

The effect of education on health: theory

Theoretical foundations for a causal effect of education on health were first provided by the seminal work of Grossman (1972), based on the human capital model of Becker (1964). One key insight of Grossman’s model of health capital is that individuals derive utility from health directly (they do not like being sick) and indirectly by affecting labor market outcomes (sick individuals work less and earn less). The other essential feature of the model is the recognition that there is a “health production function” – that there are known factors that individuals (or institutions) can manipulate in order to affect health in predictable ways. These two features give

rise to a behavioral model where individuals demand medical care, food, and other goods and services because they are aware these factors will improve their health and ultimately increase their utility.³

In this type of model, education can affect health in a variety of ways. Most obviously, education affects the type of jobs that individuals get and the income they earn. A year of education raises income by about 7% (Card 2005), and this is true in both developed and developing countries. Higher incomes increase the demand for better health, but they affect health in other ways as well. Richer people can afford gyms and healthier foods; they can also afford more cigarettes. Further, when an individual's wage increases, it raises the opportunity cost of time: because many health inputs require time (such as exercise or doctor visits or cooking), in the short run wage increases might reduce health. Thus, the income associated with higher education may or may not improve health.

Highly educated people are also more likely to take jobs that provide health insurance and other benefits such as retirement accounts (Employee Benefit Research Institute, 2011). Although one expects these benefits to have a positive effect on health, it is theoretically possible that they do not. For example individuals with insurance could be less likely to care for themselves because they face lower financial costs in the event of a disease. Because the uncompensated costs of disease are large however (morbidity and premature mortality), we do not expect these indirect effects to dominate the access associated with better insurance.

Finally, more educated people work in different industries and occupations than less educated people. To the extent that job characteristics affect health, this may affect health as well. At the dawn of the industrial era, this relationship was undoubtedly positive. Early in the

³ See Strauss and Thomas (forthcoming) for an excellent exposition of the theoretical production of health over the life course and its determinants.

20th century the more educated were more likely to work in white collar occupations, which were substantially safer than working in agriculture or manufacturing (fewer accidents, exposure to chemicals, physical strain, etc). Today, this could still be true, but it is less clear. Most individuals work in the service sector and the better educated may have jobs that are worse for their health – for example, they spend more time sitting in front of computers, which could turn out to be bad: sitting (independently of exercise) has been recently shown to detrimental to health (Patel et al 2010).

Thus the effect of education on health, through its effect on the labor market, is ambiguous. Moreover, a positive association between education and disease can arise through the conscious choices of individuals: individuals may well know that exercise is needed to remain in good physical shape, but they may optimally trade-off some of their health for increased incomes when wages are high. At the extreme, when individuals have no other resources than their bodies to earn a living, they will optimally “use up” their bodies to earn a living: trading off higher lifetime earnings for shorter, sicker lives, as in Case and Deaton (2005). The theory of compensating differentials predicts just that: individuals can be “paid off” to accept risky occupations (Rosen 1986).

The second mechanism explored by Grossman is that education can affect the production function of health directly, acting as a “technology” parameter. This is the so-called “productive efficiency” mechanism (Kenkel, 1991), in contrast to the “allocative efficiency” mechanism which we have already described (the more educated will optimally chose different levels of health inputs because they face different prices and budget constraints). In its simplest formulation, productive efficiency posits that the better educated will have better health outcomes, even conditional on access to the same health inputs at the same prices. Better use of

information is the classic example. More educated individuals might be better at following doctor's instructions (because they might have better self control or more flexibility in their time use for example) or they might be more likely to believe the information produced by the scientific establishment and follow recommendations that follow from these (because they took science courses in school or know scientists directly).

Cutler and Lleras-Muney (2010a) provide an example of this pathway. Both more and less educated people strongly agree that one should wear a seatbelt while driving a car. But the less educated are much more likely to agree with the opposite of that statement as well: true or false, seatbelts are just as likely to harm as help you in an accident. It may be that better educated people have a deeper understanding of the risks in not wearing a seatbelt and the probabilities that go into a calculation of optimal seatbelt use. Another example concerns how successful individuals are at using certain health technologies, such as devices to help quit smoking. Conditional on making an attempt to quit smoking, the better educated are more likely to be successful quitters (Lillard et al 2007).

There is a third theoretical reason why education could be related to health: education could change the 'taste' for a longer, healthier life. Becker and Mulligan (1997) posit that education lowers individuals' discount rates, making them more "patient." There are two reasons for this. First, attending school per se is an exercise in delaying gratification, and school may teach patience; this may carry over into other aspects of life. Second, to the extent that individuals can "choose" or learn what to like (in other words if discount rates can be chosen), then those with more education have a greater incentive to choose patience, since they face steeper income profiles over their lifetimes. The same argument might be told about risk aversion.

Finally, education affects the peers that individuals spend time with, and different peer sets may encourage different health behaviors. This is particularly important in the context of health, given that many health behaviors have an important social component. For example, individuals generally drink together and often smoke together. More generally peers are thought to be essential in determining risky behaviors. Also peers and social networks are an important source of information, and of financial, physical and emotional support, and as such can affect whether individuals get sick and how well they fare when they do. If on average more educated individuals have more educated peers, they will have access to a greater set of resources. If more educated individuals are more likely to be better informed (because they learned so in school or because they remain better informed later), then peers will help individuals reinforce their knowledge, in a “multiplier” setting.

Notice however, that just as personal financial resources can have an ambiguous effect on health, so can peers’. A peer group that focuses on sedentary lifestyles and lack of long-term investment may encourage that same behavior among all members of the peer group.

Beyond the Grossman model, there are other theories that also predict associations between education and health. The most prominent is that education predicts rank in society, and those with higher rank are in better health than those with lower rank. In small hierarchical groups, such as apes and (perhaps) humans, those at the top will have access to more resources and greater control over their lives in general, whereas those at the bottom will have both fewer resources and control. As a consequence, those at the bottom will suffer more “stress” and this in turn lowers immune responses and increases the likelihood of short term illness and long term chronic disease.

This theory has been shown to be accurate among mammals and other species (Sapolsky 2004), and has been tested experimentally with animals to rule out genetic factors as the main explanation (for example, the top of one hierarchy will suffer in health if they are transferred into a different group where they have a lower place in the hierarchy). Although it is not entirely clear whether and how this theory applies to humans in large modern societies – where reference groups are multiple and they are chosen endogenously – it provides another rationale by which education may affect health. Note that this theory has an interesting prediction: if all that matters is relative rank in society, a society with higher average levels of education may have no better outcomes than a society with lower average levels of education.

Education may also affect health because the things that kids do while in school are different than what they do outside of school. Though this is a trivial observation, this so-called “incarceration effect” is extremely important to consider. For example, children in school may have less exposure to criminal activity or poor role models.

Finally there are other possibilities. The more educated could inadvertently be better or worse off because of biological processes that are not well understood. For example, more educated women have higher mortality rates of cancers of the reproductive system. It has been hypothesized that this “wrong” gradient emerges because more educated women have fewer children, and having children turns out to be protective from certain cancers (Willett et al., 2010). Overall, Cutler and Lleras-Muney (2010b) report that education appears to lower mortality even after all health behaviors are accounted for, which suggests that some of these non-behavioral mechanisms might be important – though it is not obvious that all important health behaviors can be observed.

Of course it is very likely that many of these mechanisms are at play at any one time and place, and that in combination they will yield complex patterns. The patterns reported by de Walque (2006) about education and HIV in Africa are an interesting case in point—he reports that “education predicts protective behaviors like condom use, use of counseling and testing, discussion among spouses and knowledge, but it also predicts a higher level of infidelity and a lower level of abstinence.” In this example it would appear as if the educated seek out information at higher rates, know more, and use their information and resources to purchase protection; but also have some higher risky behaviors, perhaps because of their higher incomes or lower risk (they can “afford” it).

Evidence on the causal effect of education

A large number of early studies found supporting evidence for the Grossman model using largely descriptive tools (Grossman 2000). The usual prediction tested was that education and health were positively correlated. Clearly they are; the literature struggled with instruments for wages to determine causality. However, these studies were not entirely convincing about whether education had a causal effect on health, since descriptive methods and imperfect instruments are not well-suited to establishing causality.

A second generation of studies attempted to provide clearer evidence of a causal link between education and health again using “natural experiments.” Many of these studies make use of compulsory schooling as a source of plausibly exogenous variation of education to investigate whether more school improves adult health. The intuition for this approach is simple: some individuals are forced to attend school longer because of compulsory school legislation, and researchers can examine whether the health of those that are forced to obtain more schooling

improves relative the health of those not required to stay in school. Studies in the US (Lleras-Muney, 2005), Denmark (Arendt, 2005), Netherlands (Van Kippersluis et al 2011), Sweden (Spasojevic, 2004), the UK (Silles 2009, and Powdthavee 2009) and Germany (Kempton et al. 2011) using changes in compulsory schooling find that indeed these laws ultimately improved the health of the affected populations. On the other hand, recent work by Clark and Royer (2010), Oreopolous (2006), and Jurges et al (2009) finds no effect of compulsory schooling laws on health in England, and Albouy and Lequien (2009) find no effects in France.

The literature that has estimated the effect of education on health behaviors using natural experiments is also mixed. For example, Sander (1995a, b), De Walque (2004), Kenkel, Lillard and Mathios (2006) and Grimard and Parent (2007), Jurges et al (2011) find that schooling lowers smoking rates but Grimard and Parent (2007) and Kempton et al (2011) find no evidence that schooling affects smoking behavior.

It is difficult to interpret this conflicting evidence. All of the papers that find positive effects of education on health use natural experiments to construct IV estimates of the impact of education. They tend to find effects that are larger than OLS. Although this has been generally interpreted as reflecting heterogeneity of treatment effects (those that are affected by the legislation have larger returns), the alternative interpretation is that the “natural experiment” did not in fact work as well as an experiment, and there is still substantial bias in the education estimates, as thoroughly discussed in Rosenzweig and Wolpin (2000). For example Mazumder (2010) shows that the results in Lleras-Muney are not robust to the inclusion of state-specific trends. However there is very little variation left once these controls are added, so it is not clear whether the effects are truly overestimated, or whether the variation in the laws is not sufficient to estimate an effect of education. This discussion underscores the limitations of IV studies in

general. From a methodological point of view, the paper that makes the fewest assumptions is the Clark and Royer Regression-Discontinuity study, and this paper finds no effects of education on health.

Also interesting to note is that these papers all report impacts along different margins, not just because of the obvious reason that they study different times and places, but because the “experiments” themselves are different. In the UK, the changes in compulsory schooling were strictly followed and an entire cohort of individuals was forced to obtain almost one more year of schooling as a result. In the US by contrast, the laws that are typically studied increased educational attainment by 0.05 of a year—that is only 1 in 20 individuals obtained one more year of schooling. There are two important differences here. First the affected population in the US is a small sample among those that were potentially affected—it is indeed possible returns are different for this subset. Second in the US only a few individuals in a given cohort and place were affected, but entire cohorts were affected in the UK. If, for example, education matters because of rank, then in the US those that stayed in school had their rank increased relative to the counterfactual of no compulsory schooling law, but this is not clear in the UK.

It is also theoretically possible that the effect of education varies over time and place, and that the results from the previous studies correctly document this variation. Indeed, the international evidence suggests that the returns to education do vary across space. It is notable that the two studies that find no effects of education (Clark and Royer in the UK and Albouy and Lequien in France) study cohorts during and shortly after WWII, a time when the income returns to education were falling and generally low.

The fact that the effect of education on labor market earnings itself is causal (Card 2005) also suggests a positive effect of schooling: if schooling is rewarded in the labor market because

it raises productivity, how does it do so? Whatever general human capital is learnt in school and rewarded in the labor market might also be useful in the production of health, since it is useful in the production of goods. If education makes better workers by making workers better decision makers or better able to deal with complexity or uncertainty (as has been hypothesized by Nelson and Phelps, 1975, Schultz 1975, Rosenzweig 1995), then these abilities can be used in other domains, in particular for health.

One central conclusion of this discussion is that looking into the specific mechanisms by which education affects health would improve our understanding of the education-health link substantially. We review what is known about this next, after describing the latest attempts to infer causality in the literature.

In addition to natural experiments described above, there are a variety of experimental interventions that have been carried out and can be used to infer the effect of education on health, typically in low income countries. Duflo, Dupas and Kremer (2011) study the effects of a program than randomly distributed school uniforms – a significant cost associated with school – among upper primary level students in Kenya. The intervention successfully increased levels of schooling for both genders by a substantial amount (the dropout rate fell by 18 percent). Seven years later, treated girls had significantly lower rate of marriage and pregnancy, but the treatment had no had effect on sexually transmitted diseases. Duflo et al. also study a randomized intervention to add HIV information to the curriculum of some students. We can think of this as an intervention that affects the content or quality of school rather than the quantity. Interestingly this intervention had no effect on sexually transmitted diseases, but the rate of unwed teenage pregnancies fell.

Baird et al (BMO 2010) look at the effects of conditional cash transfers on schooling, pregnancy and marriage rates in Malawi. Conditional cash transfers are transfer programs where the receipt of income is conditional on certain behaviors, generally related to health or schooling. Unconditional cash transfers do not have any strings attached. Like Duflo et al. they find that the conditional cash transfer program resulted in lower levels of sexual activity, teen pregnancy and marriage rates among young girls in the short term, in addition to increasing schooling.

Jensen and Lleras-Muney (2010) study an intervention in the Dominican Republic that informed 14-year old boys about the labor market returns to school. The intervention successfully increased schooling by 0.2 years, and significantly decreased work in the formal labor market. They also find that treated boys started drinking heavily later and were less likely to smoke than untreated boys.

These studies suggest that education affects specific health behaviors, but not all behaviors. However even here, it is not clear that one can infer that education is the ultimate cause of the changes in health behavior we observe. The gold standard for establishing causality would call for randomly assigning individuals to various levels of education. Clearly, this approach is unethical and unfeasible. Instead these studies look at an “intent-to-treat” intervention, where individuals are randomly “incentivized” to obtain different levels of education. With this design it is possible to estimate the effect of education on health, if (1) the intervention successfully raises education levels and (2) the random incentives that are provided to increase schooling affect health only through education (the exclusion restriction assumption).

In this light, consider whether randomized interventions that potentially raise schooling can be used to estimate the causal effect of schooling. Typically interventions are designed so that reasonably sized effects on education can be detected with the chosen sample. But even if

this requirement is met and the intervention increases education levels, the intervention must induce students to attend school but not directly or indirectly impact any other determinant of health. It is difficult to design an intervention that meets this assumption. Providing scholarships to those that are credit constrained is equivalent to increasing income in the short run, which directly or indirectly is likely to affect health. Providing uniforms is not quite like providing income, but it increases incomes indirectly, by substituting for household spending. The more constrained individuals are in their consumption, and the higher the effect of the intervention on schooling is, the more likely it is that the income effects of the intervention are large. Finally informing misinformed students of the returns to school affects the present discounted value of earnings of all participants, regardless of whether they are induced to attend school or not. Since health (and its determinants) is likely to depend on permanent rather than temporary (current) income, this intervention also fails the exclusion restriction assumption.

Another important limitation of randomized interventions is that in the short run, schooling is not expected to affect health since the young are generally in excellent health and because health is a stock. Instead we expect the health effects to emerge slowly and cumulate. But it is difficult and expensive to follow individuals for many years; the interventions above follow individuals for several years but on average the participants are still quite young at the last follow up (for example in the Jensen and Lleras-Muney, the intervention takes place when boys are 14 and they are 18 when they are last interviewed). The interventions then look at health behaviors but it is not clear how these effects will eventually translate into for example mortality.

There are only 2 studies of randomized education interventions that follow individuals over a long period of time. One looks at the participants Perry Preschool School program (PPP) 37 years later (Muennig et al 2009) and the other looks at the participants of the Carolina

Abecedarian (ABC) Project at age 21 (Muennig et al 2011). Both of these interventions occurred early in childhood, and they have been shown to have had persistent effects on wages and other outcomes (Heckman et al., 2010). The results from these two studies are again in conflict: the treated students in the ABC program had significantly better health than the controls, but that was not true in the PPP program, although in both cases the treated appear to have better health behaviors. These results are to be taken with caution though as in both cases the number of observations consists of only about 100 individuals.

Thus simple randomized trials cannot conclusively nail down the question of whether education affects health. But it is possible to make progress on this question by investigating mechanisms through which interventions affect education, by designing more complex randomized interventions, or by making stronger assumptions theoretically. We discuss the evidence on mechanisms next and conclude with a series of observations on what questions could be explored in future research.

III. Evidence on the mechanisms linking education and health

To be convincing, studies of the effect of education on health will need to understand the pathways that link the two. Because there are a large number of potential mechanisms, this is a difficult task (e.g. Cunha et al., 2006). In addition, the evidence on mechanisms is somewhat weaker than the evidence on causality, since we often have to make assumptions about what constitutes a mechanism.

Some studies have attempted to look at why education matters for health. Consider the research cited above by Duflo, et al. on the effect of education on sexual behaviors and fertility. An important reason why education improves outcomes for girls is that it delays marriage and

fertility, since the common practice is for girls to marry soon after finishing school. This in turn means girls will have fewer years of “exposure” to get pregnant, and thus fewer children over their lifetime. Also girls in school have children later, which is beneficial because reproduction during the early teenage years is riskier for the health of the mother and the infant compared to reproduction in prime adult years (Trussell and Pebley 1984).

The results from the randomized trial in the Dominican Republic analyzed by Jensen and Lleras-Muney seem also to be driven in part by the incarceration effect: most boys that are not in school start working or are idle – the set of people that they interact with when they are not in school is different from their peers in school, and “treated boys” (those given the message about the value of education and stay in school longer) report that their peers are significantly less likely to drink and smoke. Notice further that early exposure to a different set of peers could have important long term consequences, as smoking and drinking are addictive behaviors that affect youth’s physical and mental development.

Consider now the natural experiments that use compulsory schooling as an instrument for education. In the US in the 1910s, the time period analyzed by Lleras-Muney, children who were not in school were either idle or working. The main occupation for children 10-15 at the time was agriculture (Bureau of the Census, 1924). Agricultural work is substantially more hazardous to health than school work. Thus it is possible the health effects of forcing children to stay in school during this period are driven by the difference in health hazards across environments. However by the 1940s the types of jobs adolescents engaged in when they were not in school were substantially different, and perhaps not as hazardous. This may explain why the returns to post-World War II compulsory education in the UK were smaller.

However the evidence suggests that the effect of education is not limited to this incarceration effect alone. Duflo, et al. find that the delay in marriage extends well beyond the increase in years of schooling generated by the intervention, so at least in this case, incarceration alone cannot explain the observed effects.

Another possibility is that education matters (sometimes) for health because schools directly provide information on how to improve health, and it is the health information itself, rather than being in school that affects behaviors. More educated individuals are indeed better informed about health risks in developed countries (Cutler and Lleras-Muney, 2010a). And when information first becomes available, it seems to first become known to the more educated, who in turn seem to be the first to respond. Aizer and Stroud (2010) show that educated mothers stopped smoking at higher rates after the 1964 Surgeon General Report first widely publicized the harms of smoking, and their babies' health increased more as a result. De Walque (2004) shows that smoking for the best educated declined in the 1950s, before the Surgeon General's report, as the dangers of smoking were increasingly discovered. Similarly de Walque (2006) documents that there was no relationship between education and HIV in Uganda in 1990, but one emerged by 2000 after a decade of information campaigns on prevention. And Anderberg, Chevalier and Wadsworth (2011) describe how when information was first (incorrectly) reported about possible autism risks associated with the MMR vaccines, vaccination rates fell more in areas with more educated individuals. In fact, in some cases it appears as if all of the effect of education is explained by information: for example Thomas et al. (1991) report that most of the effect of maternal education on child height can be explained by differences in information.

But information cannot be the whole explanation; we observe differences in health behaviors by education even when there are no differences in information by education. For

example in the experiment in the Dominican Republic analyzed by Jensen and Lleras-Muney, there were no differences in the extent to which smoking and drinking were perceived as harmful by the treated and the control boys, and yet the treated boys smoked and drank less. Similarly in developed countries today knowledge of the harms of smoking is nearly universal, and though there are some small differences by education in knowledge, these differences are very small compared to the differences in smoking rates by education. In the Duflo et al. study, curriculum interventions alone had little impact on behavior. Finally, observational studies suggest that a small portion of the effect of education on behaviors is due to differences in knowledge (Kenkel 1991, Meara 2001, Cutler and Lleras-Muney 2010a). It appears that when knowledge first becomes available on how to improve health, it substantially increases education disparities. But in the long run, information diffuses and other factors are more important in explaining the associations between education and health. This again suggests important dynamics in the health-education relationship.

In this sense, information may be like other innovations in health. For example Lleras-Muney and Lichtenberg (2006) report that the more education are more likely to use recently approved drugs than the less educated, and this appears to be driven by those with chronic conditions who use drugs repeatedly, suggesting that learning is an important component of the education effect. Similarly in developing countries more educated individuals are generally more likely to adopt new innovations (Foster and Rozenweig 2010). Whether the initial advantage of the educated fades away or gets stronger with time might depend in turn on the type of health technology. For example some medication regimes are difficult to adhere to, and the educated might have a permanent advantage at using them—this is the case for diabetes type 1 (Goldman and Smith 2002). Other innovations instead are “de-skilling,” such as the birth control pill

(Rozenweig and Schultz 1989), in which case eventually the less educated catch up. The results from Cohen, Dupas and Schaner (2011) provide some interesting evidence on this point: when access to malaria treatment improves, the gap in access between the educated and the uneducated falls. However the educated still behave quite differently from the uneducated in their treatment-seeking behaviors: they appear to be more likely to know the likelihood that they have malaria and they are more likely to visit a health care center and less likely to use other treatments when their symptoms are worse. This is not true among illiterate individuals.

The evidence from randomized interventions suggests some mechanisms are important whereas others are not—but of course as the discussion above suggests the extent to which any findings are generalizable is not clear. Some of the effect of schooling is through the incarceration effect as we already discussed. Another important mechanism is income, as the Malawi conditional cash transfer intervention suggests. Finally peers are also important. Jensen and Lleras-Muney find that discount rates, risk aversion and health information were not affected by the intervention they study, even as schooling increased. Instead they observe that it is the income and the behavior of students' peers that are affected and most plausibly explain the changes in smoking and drinking.

Interestingly, this evidence is consistent with the exploratory findings in Cutler and Lleras-Muney (2010a) who consider a comprehensive set of mechanisms to explain health behaviors in the US and the UK. They can account for about 70% of the effect of education (in a statistical sense), through resources (30%), family and friends (10%), and information (10%) and cognition (20%). On the other hand risk aversion, discounting, stress and other personality traits did not appear to mediate the relationship between education and behaviors – though the noise in these measures gives one some pause. Conti et al (2010) who use a more structural approach also

find that education appears to be causally related to many health behaviors and outcomes, with selection explaining (roughly) half of the association between education and outcomes.

IV. Summary

On balance, the literature we review highlights a wealth of interactions between education and health. Education appears to be causally related to health in many settings, but not always, and the reverse is true as well.

Equally important, this review highlights some unanswered issues. The most important issue is to understand in more detail how education translates into health. To what extent is education associated with specific knowledge, with cognitive ability in general, or with different social settings, either during school or after? Some evidence on this may come from looking at the quality of education individuals receive. Most of the literature has looked at the impact of additional years of schooling. Yet many of the theories say that the quality of the years should matter as well. This has not been explored in any great detail.

Simple experimental designs that randomly encourage individuals to obtain schooling can be useful in providing further evidence of causality on health and health behaviors—but they cannot conclusively answer the question of whether education alone is responsible for the observed effects because in general it is difficult to satisfy the exclusion restriction that is needed to reach such conclusions. However more sophisticated designs such as the ones discussed in Ludwig, Kling and Mullanaithan (2011) could be implemented to help identify mechanisms and causality both. For example one could design an experiment with three treated groups, where individuals are given unconditional cash transfers (cash-only group), conditional cash transfers if they attend school (attendance group), and conditional cash transfers for both going to school

and obtaining good grades (performance group). Under the assumption that all treatments induce changes in education, income and grades, we can learn about the separate effects of education, income and learning on health. By comparing the controls to the cash-only group one can estimate the effects of income on health and health behaviors. By comparing the outcomes of the cash-only group and the attendance group one can obtain an estimate of the effects of attendance. Finally by comparing the performance group and the attendance group one can learn about the effects of education content.

Further, it is vitally important to understand the translation from intention into action. In developed countries, almost everyone knows the behaviors that are good for health and would like to behave healthier. Yet people systematically fail at this task. How do we understand these failures, and what types of interventions would reduce them? In a way, this is asking for a benchmark by which to compare education. Improving health by inducing more education is costly; many people do not enjoy schooling, and forcing additional years of schooling comes at a price. If we can replicate the impact of education on health using other methods, this would be very attractive.

In sum, the burgeoning literature on education and health is just the beginning. A review written a decade from now will ideally have many more specific conclusions to draw.

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Figure 1: Coefficient of Education on BMI by GDP

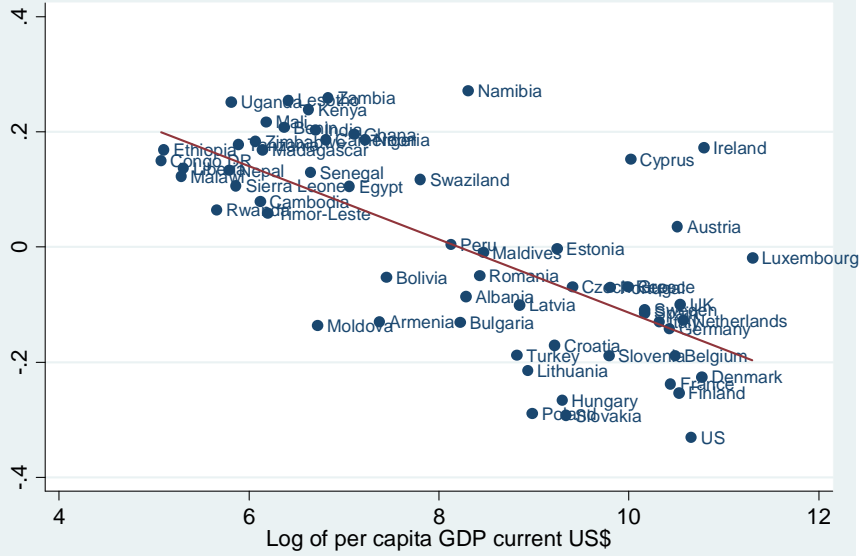


Figure 2: Coefficient of Education on Underweight by GDP

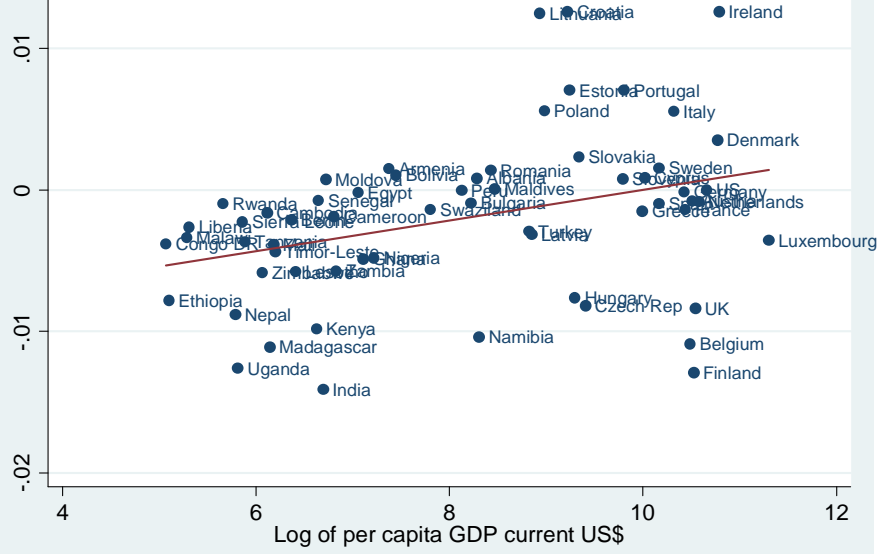
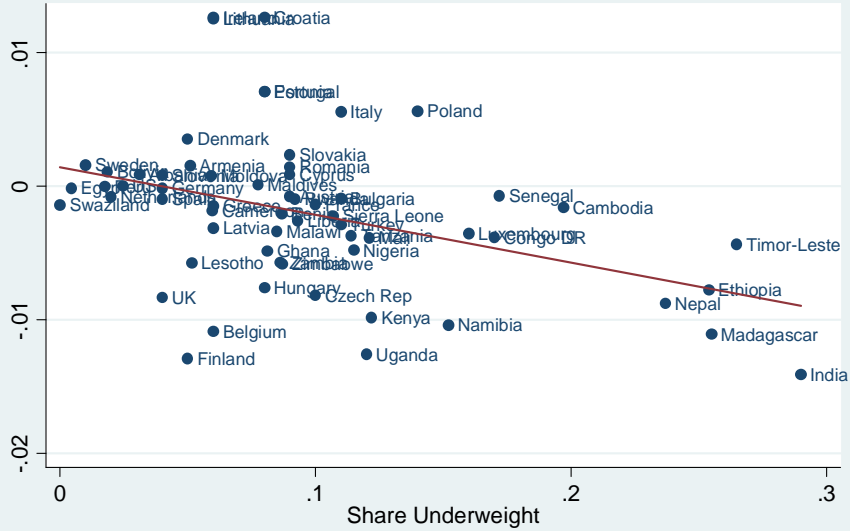


Figure 3: Coefficient of Education on Underweight by underweight level



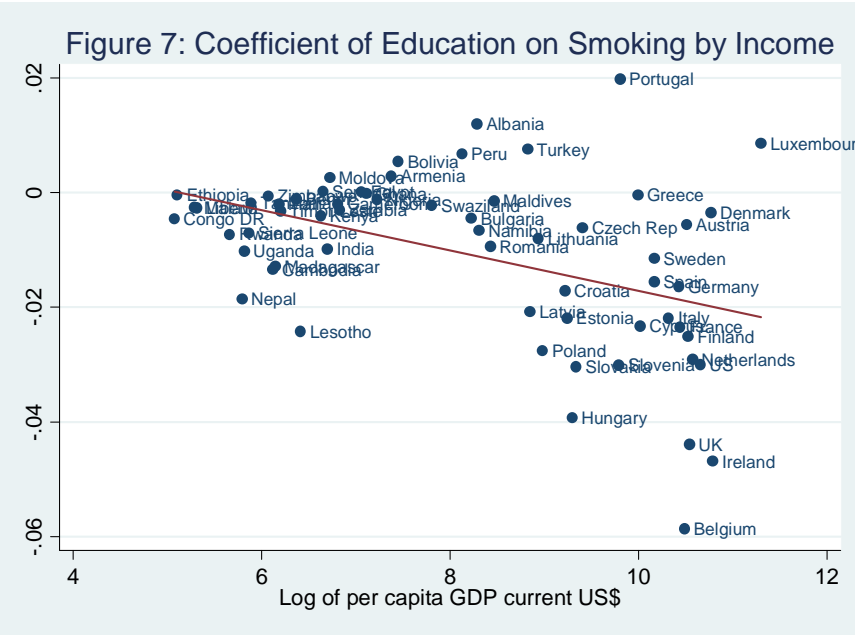
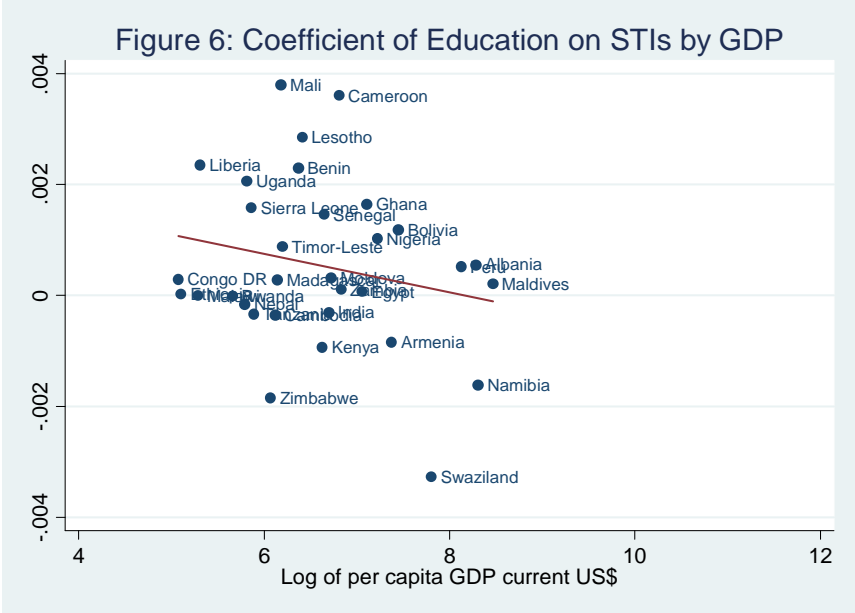


Figure 8: Coefficient of Education on Drinking by Income

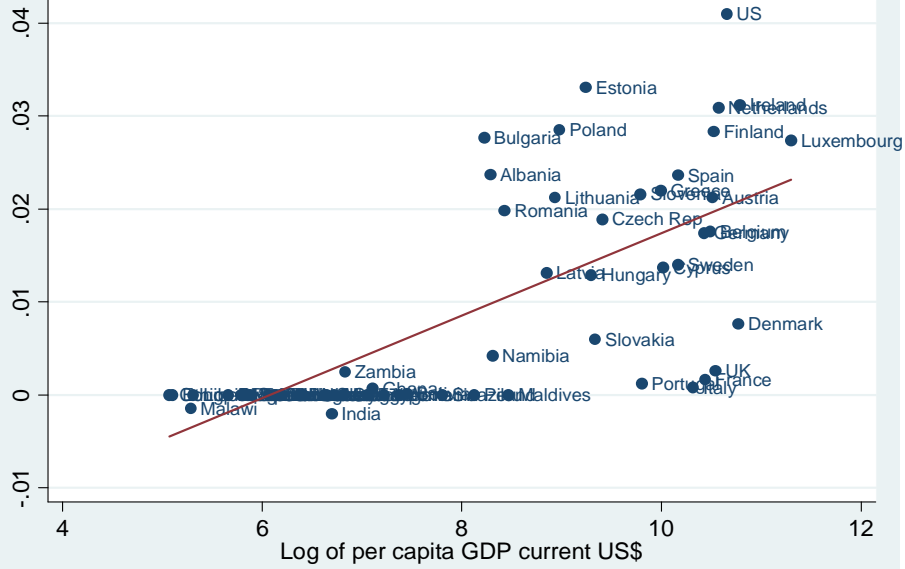


Figure 9: Coefficient of Education on Height by Income

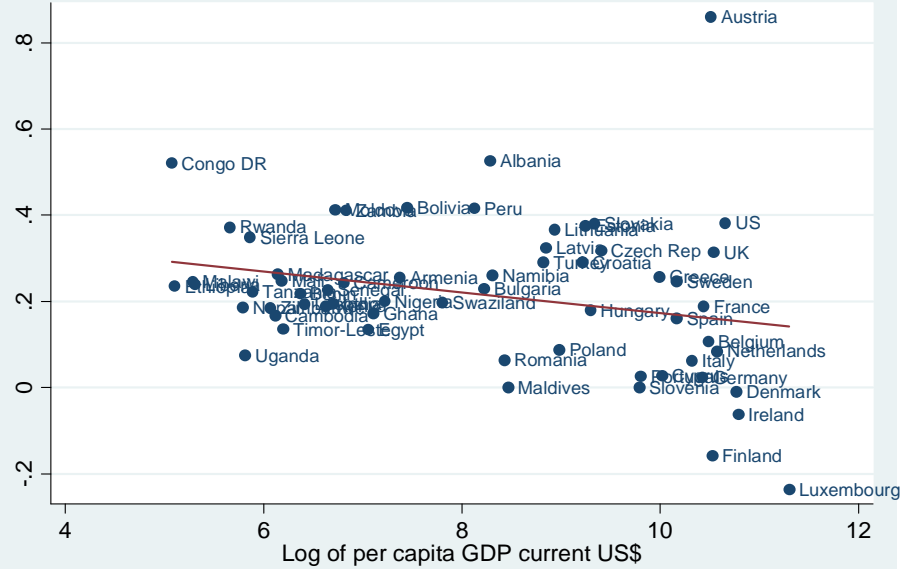


Table 1: The evolution of knowledge and smoking gradients in education in the US 1949-1969

Year of survey:	1949	1954	1957	1969
<u>Panel A: effect of education on knowledge</u>				
Dependent variable:	"Do you think cigarette smoking is harmful or not?"	What is your opinion--do you think cigarette smoking is one of the causes of lung cancer, or not?		
<HS	.057*	-.054*	-.065**	-0.041
Some college	0.012	0.032	.116**	0.045
College+	0.021	0.067	.172**	.111**
<u>Panel B: Effect of education on smoking</u>				
Dependent variable:	Current Smoker?			
<HS	-.056*	-0.016	0.024	.054*
Some college	0.019	-0.026	-0.008	0.011
College+	-0.045	-0.061	-0.003	-.076*

All regressions are adjusted for age, sex and race. Individuals with a high school degree only are the reference group.

Data Appendix

1. DHS Surveys

We selected 31 countries with either a DHS-IV or a DHS-V survey that includes data on a woman's anthropometry (height and weight), education level, and her drinking or smoking habits. All surveys contain nationally representative samples of ever-married women between the ages of 15 and 49.

Height is the respondent's height in centimeters. Body Mass Index (BMI) is computed as weight (in kilos) divided by height (in meters) squared. Underweight is equal to one if the person's BMI is less than or equal to 18.5; obese is equal to one if the person's BMI is greater than or equal to 30. Anemia is coded 1 if the person is anemic at all, irrespective of the level of anemia (slight, moderate, severe). Hemoglobin is the individual's hemoglobin level in g/dl adjusted for altitude. Anemia and hemoglobin were considered unknown if hemoglobin levels were below 5 or greater than 50. If the adjusted hemoglobin level was not available, the unadjusted level was used. Smoke is coded 1 if the individual has currently smokes, 0 if not. STI is equal to 1 if the individual had a sexually transmitted infection in the last 12 months. Drink is a binary variable if the individual has ever or recently consumed alcohol (this varies by country).

Regressions control for age, age2, education, married, religion dummies, and ethnicity dummies. Age and education are measured in years. Religion and ethnicity dummies are country specific. Marital status is 1 if the woman is married or living with a partner as if married, and 0 otherwise. All means and regression coefficients were computed taking survey design into account, unless strata or sample weights were not provided by the survey.

2. Eurobarometer data

Our European data are drawn from 2 waves of the Standard Eurobarometer. Women's anthropometry (height, weight, BMI, probability of being underweight or obese) are drawn from Eurobarometer 64.3, which was collected in November-December 2005. All other outcome variables of interest (alcohol consumption, smoking, physical activity and sport, and fruit consumption) are drawn from Eurobarometer 72.3, which was collected in October 2009. Both surveys contain nationally representative samples of women between the ages of 15 and 49 in 29 European countries.

Height is the respondent's height in centimeters. Body Mass Index (BMI) is computed as weight (in kilograms) divided by height (in meters) squared. Underweight is equal to one if the respondent's BMI is less than 18.5; Obese is equal to one if the respondent's BMI is greater than or equal to 30. Currently Smokes is equal to one if the respondent currently smokes, and is zero otherwise; Consumed Alcohol in Past Year is equal to one if the respondent has consumed any alcoholic beverages in the past 12 months.

Regressions control for age, age2, education level and marital status. Age is measured in years. Marital status is 1 if the woman is married or living with a partner, and 0 otherwise. Education level is the age at which the respondent left school, in years. All means and regression coefficients were computed using the post-stratification weights provided with the surveys.

3. Behavioral and Risk Factors Survey for the US

For the US we use the 2005 wave of the Behavioral And Risk Factor survey, which contains height, weight, drinking and smoking. We again keep only women ages 15 to 49.

Height is the respondent's height in centimeters. Body Mass Index (BMI) is computed as weight (in kilograms) divided by height (in meters) squared. Underweight is equal to one if the respondent's BMI is less than 18.5; Obese is equal to one if the respondent's BMI is greater than or equal to 30. Currently Smokes is equal to one if the respondent currently smokes, and is zero otherwise. A person is said to drink if they drank any alcohol in the last 30 days.

Regressions control for age, age2, education level and marital status. Age is measured in years. Marital status is 1 if the woman is married or living with a partner, and 0 otherwise. Education level is measured in years of school. Race and ethnicity dummies are included. All means and regression coefficients were computed using the post-stratification weights provided with the surveys.

4. GDP data

The GDP per capita data comes from the World Bank, using the GDP per capita (current US\$) indicator. When the dataset comes from a survey taken over multiple years, the GDP per capita figure is the mean during that period.