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# ACCOUNTING FOR THE EFFECT OF HEALTH ON ECONOMIC GROWTH\*

DAVID N. WEIL

I use microeconomic estimates of the effect of health on individual outcomes to construct macroeconomic estimates of the proximate effect of health on GDP per capita. I employ a variety of methods to construct estimates of the return to health, which I combine with cross-country and historical data on height, adult survival rates, and age at menarche. Using my preferred estimate, eliminating health differences among countries would reduce the variance of log GDP per worker by 9.9 percent and reduce the ratio of GDP per worker at the 90th percentile to GDP per worker at the 10th percentile from 20.5 to 17.9. While this effect is economically significant, it is also substantially smaller than estimates of the effect of health on economic growth that are derived from cross-country regressions.

## I. INTRODUCTION

People in poor countries are, on average, much less healthy than their counterparts in rich countries. How much of the gap in income between rich and poor countries is accounted for by this difference in health? The answer to this question is important both for evaluating policies aimed at improving health in developing countries and more generally for understanding the reasons why some countries are rich and some poor.

The United States government as well as several international organizations and private charities have recently embarked on ambitious efforts to improve health in developing countries. Included in these efforts are the Bush Administration's commitment of \$15 billion over five years to fight AIDS; the Roll Back Malaria partnership launched by the World Health Organization (WHO), World Bank, and other international organizations in 1998; and the recent creation of the independent Global Fund for AIDS, TB, and malaria. The primary justification for

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these programs is the potential to reduce suffering and premature death among the affected populations. However, an important secondary justification is the potential gain in economic development that is expected to follow from health improvements. For example, the report of the WHO's Commission on Macroeconomics and Health [2001] states

Improving the health and longevity of the poor is an end in itself, a fundamental goal of economic development. But it is also a *means* to achieving the other development goals relating to poverty reduction. The linkages of health to poverty reduction and to long-term economic growth are powerful, much stronger than is generally understood. The burden of disease in some low-income regions, especially sub-Saharan Africa, stands as a stark barrier to economic growth and therefore must be addressed frontally and centrally in any comprehensive development strategy.

My goal in this paper is to quantitatively assess the role that health differences play in explaining income differences between rich and poor countries and, thus, to calculate the income gain that would result from an improvement in the health of people living in poor countries.

Economists have identified several channels through which health affects the level of output in a country. One channel, which I call the proximate or direct effect of health, is that healthier people are better workers. They can work harder and longer and also think more clearly. Beyond this proximate effect of health, there are a number of indirect channels through which health affects output. Improvements in health raise the incentive to acquire schooling since investments in schooling can be amortized over a longer working life. Healthier students also have lower absenteeism and higher cognitive functioning and, thus, receive a better education for a given level of schooling. Improvements in mortality may also lead people to save for retirement, thus raising the levels of investment and physical capital per worker. Physical capital per worker may also rise because the increase in labor input from healthier workers will increase capital's marginal product. The effect of better health on population growth is ambiguous. In the short run, higher child survival leads to more rapid population growth. Over longer horizons, however, lower infant and child mortality may lead to a more-than-offsetting decline in fertility, so that the Net Rate of Reproduction falls (Bloom and Canning [2000], Kalemli-Ozcan, Ryder, and Weil [2000]). At a much longer horizon, Acemoglu, Johnson, and Robinson [2001] argue that the poor health environment in some

parts of the world led European colonizers to put in place extractive institutions which, in turn, reduce the level of output today. In this paper, I look only at health as a proximate determinant of a country's income—that is, I examine the effect of better health in enabling workers to work harder and more intelligently, holding constant the level of physical capital, education, the quality of institutions, and so on.

Examining the effect of health on economic growth is made difficult by the endogeneity of health itself. The mechanisms that lead to a positive dependence of health on income are fairly obvious. People who are richer can afford better food, shelter, and medical treatment. Countries that are richer can afford higher expenditures on public health.<sup>1</sup> Because health is endogenous, it is almost impossible to use aggregate data to determine the structural effect of health on income. I am able, however, to use structural microeconomic estimates of the direct effect of health on individual income, which, along with aggregate data on health differences among countries, are all that is required to measure the direct effect of health differences on income differences among countries.

Beyond looking at health's effect on growth, a second goal of this research is to examine the broader question of what determines a country's level of income. Recent research (see Caselli [2005] for a review) has used the technique of development accounting to parse variation in income among countries into the pieces explained by accumulation of physical capital and human capital in the form of education, as well as remaining residual variation due to differences in productivity. The conclusion from this literature is that residual productivity is by far the most

1. Pritchett and Summers [1996], using an instrumental variables procedure, find a significant effect of national income on health, as measured by infant and child mortality. The instruments that they use are terms of trade shocks, the ratio of investment to GDP, the black market premium, and the deviation of the exchange rate from PPP. A more contentious question is the degree to which average health varies among countries for reasons other than income. Gallup and Sachs [2001] argue that tropical areas have fundamentally worse health environments than do the temperate parts of the world. They claim, for example, that the fact that malaria has been eliminated in currently rich areas (such as Spain or the southern United States) but not in poor ones (such as sub-Saharan Africa) does not reflect differences in income, but rather the fact that malaria's grip is much stronger in Africa. Under this view, these fundamental differences in the health environment present a very strong obstacle to economic growth in the tropics. In contrast, recent work by Acemoglu, Johnson, and Robinson [2001] take the view that differences in the fundamental health environment between countries are not large and that the high level of disease in tropical countries is more a result than a cause of their poverty.

significant source of income differences, explaining more than half of the variance of income. Because existing analyses do not account for health, differences in income due to health are included in this productivity residual along with differences in income due to institutions, geography, culture, and so on. By accounting for variation in health among countries, I am able to explain a fraction of this residual productivity variation and produce a purified version of the productivity residual.

The rest of this paper is organized as follows. Section II discusses previous literature that has examined the link between health and economic outcomes. Section III presents a framework for analyzing how health affects income at the individual and national level. Section IV discusses the aggregate health indicators used in the analysis. Section V presents a variety of estimates of the return to health indicators. Section VI looks at the magnitude of productivity differences among countries implied by differences in health outcomes, and Section VII looks at the contribution of health variation to variation in GDP among countries. Section VIII concludes.

## II. BACKGROUND LITERATURE

Research examining the link between health and economic outcomes, at either the individual or national level, has generally examined two types of health measures: inputs into health and health outcomes. Inputs into health are the physical factors that influence an individual's health. These include nutrition at various points in life (*in utero*, in childhood, and in adulthood), exposure to pathogens, and the availability of medical care. Health outcomes are characteristics that are determined both by an individual's health inputs and by his genetic endowment. Examples include life expectancy, height, the ability to work hard, and cognitive functioning. For the purpose of explaining income differences among countries or individuals, the key health outcome of interest is how health affects the ability to produce output. I call this health outcome "human capital in the form of health." We do not observe human capital in the form of health directly, but presumably it is some combination of ability to work hard, cognitive function, and possibly other aspects of health. In contrast to human capital in the form of health, there are a number of health outcomes that can be observed at either the individual

level, the national level, or both. I refer to these health outcomes as health indicators.

Comparisons of health among countries can be made by looking at either inputs to health or health indicators. Rich countries have more of almost all health inputs than poor countries. To give some obvious examples, the fraction of the population with access to clean drinking water, the number of physicians per capita, and the nutrient composition of the diet all differ markedly between rich and poor countries. Similarly, rich countries today have better health inputs than they did in the past. Comparisons of health indicators tell much the same story as comparisons of inputs (these data are discussed further below). These facts suggest that *unobservable* health outcomes, including human capital in the form of health, are also better in rich than poor countries.

A large microeconomic literature examines the effects of varying health inputs on health outcomes themselves, human capital attributes that are contingent on health outcomes, and wages. In many studies, more than one of these groups of dependent variables is examined.<sup>2</sup> Several studies (see Behrman et al. [2003], Alderman, Hoddinott, and Kinsey [2006], Maccini and Yang [2005], and Chavez, Martinez, and Soberanes [1995]) have examined the long-run effects of childhood nutrition, using a variety of natural and man-made experiments that provide exogenous variation in nutrition. They find that better nutrition leads to improvements in school completion, IQ, height, and wages. Studies of the effect of adult nutrition (Strauss [1986], Strauss and Thomas [1997], Basta, Soekirman, and Scrimshaw [1979], and Thomas et al. [2004]) similarly find positive effects on labor input and wages. Bleakley [2007] and Miguel and Kremer [2004] find that treatment with deworming drugs increases school attendance. Several other studies that have examined the effects of childhood nutrition and health inputs are discussed in Section V.A.

Using these microeconomic estimates of the effects of variation in health inputs on wages, it is possible to calculate the contribution of variance in a single health input to variation in income among countries. For example, Behrman and Rosenzweig [2004], using variation in birth weight among monozygotic twins as an instrument, find significant effects of intrauterine nutrition

2. See Thomas and Frankenberger [2002] for an extensive review.

on adult wages. Using their estimate, one can show that eliminating variation in birth weight among countries would reduce the variance of log GDP per worker by 3.1 percent.<sup>3</sup>

Extending this methodology to encompass other health inputs is difficult. Comparing rich to poor countries, there are large differences along most of the dimensions considered in the microeconomic studies listed above and many more as well. A complete analysis of the effects of equalizing health inputs (and thus health) among countries would require data on how each of these inputs differed among countries as well as a microeconomic estimate of the effect of each input on labor productivity. Neither the data nor the relevant estimates to undertake this exercise currently exist. A further theoretical problem with conducting such an exercise is that to add together the different effects examined singly in microeconomic studies would require a very strong assumption of linearity, that is, that there are no interactions among the different health inputs.<sup>4</sup>

3. Behrman and Rosenzweig actually report a smaller number, but the two figures can be reconciled as follows. Define  $y_i$  as the log of GDP per worker in country  $i$ ,  $b_i$  as average birth weight, and  $\hat{y}_i$  as predicted log GDP per worker based on the equation

$$\hat{y}_i = \text{constant} + \beta b_i,$$

where the value of  $\beta$  is derived from the twins data. To assess how much of the world variance in log income is accounted for by variance in birth weight, Behrman and Rosenzweig look at the ratio  $\text{var}(\hat{y})/\text{var}(y) = \beta^2 \text{var}(b)/\text{var}(y)$ . Using their estimated values of  $\beta = 0.00413$  along with data for a cross-section of countries where  $\text{var}(y) = 1.16$  and  $\text{var}(b) = 32.6$ , this equation yields a value of 0.0005, which they report as "less than one percent."

The problem with this measure is that because  $\hat{y}$  is not constructed by least squares, it is not orthogonal to the error term. That is, if  $\epsilon$  represents the factors other than birth weight that affect log wages, then in the equation  $y_i = \hat{y}_i + \epsilon_i$ , the two terms on the right-hand side are not orthogonal. A natural measure of the influence of birth weight on the variance of income is the proportional reduction in log variance that would result from equalizing birth weight among countries, that is,  $(\text{var}(\hat{y}) + 2 \text{cov}(\hat{y}, \epsilon))/\text{var}(y)$ . This can be expanded in turn as

$$\begin{aligned} \frac{\text{var}(\hat{y}) + 2 \text{cov}(\hat{y}, \epsilon)}{\text{var}(y)} &= \frac{2 \text{cov}(y, \hat{y}) - \text{var}(\hat{y})}{\text{var}(y)} = \frac{2\beta \text{cov}(y, b) - \text{var}(\hat{y})}{\text{var}(y)} \\ &= 2\beta \left( \frac{\text{cov}(y, b)}{\text{var}(b)} \right) \frac{\text{var}(b)}{\text{var}(y)} - \frac{\text{var}(\hat{y})}{\text{var}(y)}. \end{aligned}$$

The term  $\text{cov}(y, b)/\text{var}(b)$  is the coefficient from a regression of log GDP on average birth weight, which Behrman and Rosenzweig report as 0.136. Plugging this value along with the above data into this equation yields a value of 3.1 percent.

4. There are many cases where this required assumption of linearity is known not to hold. For example, three different inputs—nutrition, sanitary conditions, and access to medical care—are to some extent substitutes in combating

A second branch of the literature has attempted to answer the question “how much do differences in health contribute to differences in income” by looking at data on health outcomes rather than health inputs and examining data at the national rather than individual level. These papers present regressions of GDP per capita, GDP growth, or TFP on some measure of health outcomes as well as a standard set of controls. Bloom, Canning, and Sevilla [2004] report the results of thirteen such studies, which mostly reach similar quantitative results (see also Jamison, Lau, and Wang [2005]). Their own estimate, which comes from regressing residual productivity (after accounting for physical capital and education) on health measures in a panel of countries is that a one-year increase in life expectancy raises output by 4 percent.

Papers in this group suffer from severe problems of endogeneity and omitted variable bias. For example, Bloom, Canning, and Sevilla attempt to deal with the endogeneity of health and other inputs into production by using lagged values of these variables as instruments. The identifying assumption required for this strategy to work—that the error term in the equation generating health is serially correlated while the error term in the equation generating income is not—is not explicitly stated or defended.<sup>5</sup> More generally, the problem with the aggregate regression approach is that, at the level of countries, it is difficult to find an empirically usable source of variation in health, either in cross section or time series, that is not correlated with the error term in the equation determining income.<sup>6</sup>

In this paper I pursue the same question that is addressed by the aggregate regressions but using a different methodology.

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infectious diseases. Adding together the effects of improving one input at a time would overstate the net effect of improving all three.

5. See Mankiw [1995] for a discussion of this issue.

6. A few studies have attempted to solve the endogeneity problem by finding instruments for health. Sachs [2003] uses a geographically based measure of “malaria ecology” to instrument for the current prevalence of the disease and finds that malaria has a large effect on the level of GDP per capita. He is not able to look at the effect of overall health. There is the further problem that a high value of the malaria ecology index may be proxying for other omitted aspects of a tropical climate that negatively affect income. Acemoglu and Johnson [2006] construct a measure of countries’ predicted improvements in life expectancy during the decades around World War II based on the experiences of other countries with similar disease environments. Using these predicted values as instruments, they find that life expectancy improvements had a *negative* effect on GDP per capita and GDP per working-age person.



Specifically, I construct a framework in which estimates of the effect of variation in health inputs on individual wages can be used to generate estimates of how differences in health, as measured by observable outcomes, contribute to differences in national income. In other words, I use the available microeconomic estimates to create an estimate of the importance of health at the macroeconomic level.

### III. EMPIRICAL FRAMEWORK

#### III.A. Production and Wages

Start with a Cobb–Douglas aggregate production function that takes as its arguments capital and a composite labor input,

$$(1) \quad Y_i = A_i K_i^\alpha (H_i)^{1-\alpha}$$

where  $Y$  is output,  $K$  is physical capital,  $A$  is a country-specific productivity term, and  $i$  indexes countries. The labor composite,  $H_i$ , is determined by

$$(2) \quad H_i = h_i v_i L_i,$$

where  $h_i$  is per-worker human capital in the form of education,  $v_i$  is per-worker human capital in the form of health, and  $L_i$  is the number of workers.<sup>7</sup> As discussed earlier,  $v_i$  is not the totality of an individual's health; rather, it is only the aspects of individual health that are relevant for the production of output.

The wage paid to a unit of the labor composite,  $w_i$ , is its marginal product,

$$(3) \quad w_i = \frac{dY_i}{dH_i} = (1 - \alpha) A_i \left[ \frac{K_i}{H_i} \right]^\alpha.$$

The wage earned by worker  $j$  is a function of his own health and education, as well as the national wage of the labor composite. In logs,

$$(4) \quad \ln(w_{i,j}) = \ln(w_i) + \ln(h_{i,j}) + \ln(v_{i,j}) + \eta_{i,j},$$

7. Notice that implicit in this formulation is the notion that a worker with more education or health supplies more units of the same basic labor input as workers who are less educated or healthy. In the case of education, this assumption is hard to justify, since one worker with a Ph.D. is hardly a perfect substitute for four workers who have no education. In the case of health, the assumption may be marginally more satisfactory: one healthy worker who can work faster or longer may indeed be a substitute for several unhealthy workers.

where  $\eta_{i,j}$  is an individual-specific error term. Thus, individual wages are proportional to the individual's level of human capital in the form of health.

### III.B. Individual Health and Productivity

Let  $\mathbf{X}$  be a vector of inputs into individual health, such as nutrition at various points in life, exposure to pathogens, medical treatment, etc. I assume that individual health outcomes are functions of health inputs as well as a set of random errors. As mentioned earlier, there is a large number of health outcome characteristics, such as height, ability to work hard, age of death, and cognitive function, that are determined by health inputs. In the discussion that follows, I simplify notation by only working with two specific health outcomes:  $I$  (for indicator), which could be any observable element of health outcomes, such as height, and  $v$ , which is the health outcome that is relevant for labor productivity.

Consider a latent measure of health,  $z$ . Initially I take latent health to be a scalar, although below I discuss the implications of allowing it to be multidimensional. I assume that the relationship between the vector of health inputs,  $\mathbf{X}$ , and health outcomes such as  $I$  and  $v$  is mediated solely through changes in latent health. Following the existing microeconomic literature, I also restrict the effect of latent health  $z$  on health outcomes to be linear (or log-linear in the case of  $v$ ).

$$(5) \quad I_j = \text{constant} + \gamma_I z_j + \epsilon_{I,j}$$

$$(6) \quad \ln(v_j) = \text{constant} + \gamma_v z_j + \epsilon_{v,j}$$

Consider two individuals with different levels of latent health,  $z$ . As shown earlier, individual wages are proportional to human capital in the form of health. Thus, the expected gap in log wages (holding constant human capital in the form of schooling) is

$$(7) \quad \ln(w_2) - \ln(w_1) = \gamma_v(z_2 - z_1)$$

while the expected difference in health outcome  $I$  is given by

$$(8) \quad I_2 - I_1 = \gamma_I(z_2 - z_1).$$

Thus, if there were no error terms in equations (5) and (6), the

relation between the log wage gap to the gap in the indicator  $I$  would be

$$(9) \quad \ln(w_2) - \ln(w_1) = \frac{\gamma_v}{\gamma_I} (I_2 - I_1).$$

The ratio  $\gamma_v/\gamma_I$  is loosely referred to as the return to characteristic  $I$  (for example, the return to height) even though the characteristic has no economic return and is simply an indicator of underlying health. Put another way, making people taller will not increase their wages *per se*, but changes in latent health that produce both increases in wages and increases in height do so in the ratio given by the return to height. Below I use the notation  $\rho_I$  to refer to the return on outcome characteristic  $I$ .

Knowing the return to characteristic  $I$ , we can back out the difference in per-worker human capital in the form of health between countries, which is unobservable, by using data on an observable health indicator. For countries 1 and 2,

$$(10) \quad \ln(v_2) - \ln(v_1) = \rho_I(I_2 - I_1).$$

Equation (10) is the key that allows us to use cross-country data on observable health outcomes to infer the degree to which human capital in the form of health (the aspect of health that is relevant for producing output) varies across countries. The only difficulty is that we need an estimate of the return to the observable health outcome to implement this procedure.

One method for estimating return to a characteristic is if we have data at the country level on both  $I$  and  $v$  at two points in time. Then the return to the characteristic can be calculated directly as the ratio

$$(11) \quad \rho_I = \frac{\gamma_v}{\gamma_I} = \frac{\ln(v_{t+1}) - \ln(v_t)}{I_{t+1} - I_t}.$$

This is the approach taken in Section V.C below. Unfortunately, cases in which we can directly observe average  $v$  for a country are rare. The more common approach to estimating the return to health characteristics is to look at individual-level data.

The return to health outcome characteristics can be estimated at the individual level by using experimental or quasi-experimental variation in the vector of health inputs,  $\mathbf{X}$ . Consider some health input  $x$  that is an element of  $\mathbf{X}$ . If variation in  $x$  is

exogenous, its effect on either health outcomes ( $dI/dx$ ) or wages ( $dw/dx$ ) can be estimated without bias. From (5) and (6) we have

$$\frac{dw}{dx} = \frac{dz}{dx} \gamma_v,$$

$$\frac{dI}{dx} = \frac{dz}{dx} \gamma_I,$$

and thus,

$$(12) \quad \rho_I = \frac{\gamma_v}{\gamma_I} = \frac{dw/dx}{dI/dx}.$$

In other words, the return to a health outcome is just the ratio of the effect of varying the health input on wages to the effect of varying the health input on the particular health outcome. Because of the assumption that latent health is a scalar, the ratio of the change in any two health outcomes that results from changing a single element in the health input vector is the same as the ratio of the change in those two health outcomes that results from any change in the entire health input vector.

### *III.C. Bias from Assuming that Latent Health is Scalar*

The assumption that underlying health is scalar is obviously quite strong. It is more reasonable to assume that latent health is multidimensional and that health outcomes respond differentially to the various aspects of underlying health. The Appendix works through the algebra of how the measures constructed above are biased in the case in which there are two aspects of underlying health, only one of which is relevant for determining income but both of which affect the health indicator. The conclusion from this analysis is that the bias will depend on how the ratio of the changes in human capital in the form of health ( $v$ ) and the indicator ( $I$ ) induced by variation in health inputs among countries compares to the ratio of changes in these same variables induced by the experiment used to estimate the return to the health characteristic. For example, suppose that the return to height is estimated based on an experiment that has very little effect on human capital in the form of health but a large effect on height (this could be nutritional intervention at a time in life which is crucial for determining adult height). Suppose further that actual differences in health inputs among countries are

concentrated more on factors that affect human capital in the form of health (for example, micronutrient deficiencies *in utero*) but not height. In this case, the procedure presented already will understate the role of health, as proxied by the indicator height, in explaining differences in income among countries.

Similarly, the method of estimating the return to a health indicator by examining differences in  $v$  and the indicator within a single country over time and then applying this estimated return to cross-country data will be biased in the case in which the ratio of changes in  $v$  to changes in  $I$  that took place within the single country is not the same as differences in  $v$  and  $I$  induced by cross-country differences in health inputs.

I use these observations in discussing potential explanations for some of the divergent results found below. More generally, I address the problem of bias from health being multidimensional by considering a number of different health indicators and a number of different ways of estimating the return to health indicators.

The possibility that latent health is multidimensional raises conceptual problems that go beyond the potential biases in my estimates discussed earlier. If the assumption that latent health is a scalar is violated, then the concepts of “the return to health” and “the effect of health on economic growth” are not well defined, although one can still estimate how a specific change in health inputs affects individual wages or output. Even if latent health is multidimensional, however, one may still be able to sensibly discuss the return to a health *indicator*, such as the return to height. This would be the case if, for example, there were two dimensions of latent health (for example, health of the body and health of the mind) that had independent effects on log wages and each of these dimensions of health affected only a single health indicator.

#### IV. HEALTH INDICATORS

The framework above shows how observable health outcomes can be used to infer how human capital in the form of health varies among countries. The ideal indicator would have three characteristics. First, it would be related as closely as possible to the aspects of individual health that are relevant for labor productivity. Second, there would exist structural estimates of the return to this health characteristic, that is, how improvements in

overall health, as proxied by the relevant indicator, affect labor productivity. Finally, data on the indicator would be available for a broad cross section of countries.

In this paper, I use data on three indicators of health: average height of adult men, the adult survival rate (ASR) for men, and age of menarche (onset of menstruation) for women. None of these measures is ideal for the purposes at hand, but each has advantages.

#### IV.A. *Adult Height*

Adult height is a good indicator of the health environment in which a person grew up. Factors such as malnutrition and illness, both *in utero* and during childhood, result in diminished adult stature. Looking across individuals, there is also a large degree of variation in health that is not related to health, but much of this variation is washed out when one looks at population averages. Thus, the change in average height within a single country over time provides a good indicator of the change in the health environment (assuming a genetically stable population). And in settings where data such as income per capita are unavailable, height may serve as the best available measure of the standard of living.

Of course, the average height of adults is not a perfect indicator of the average *health* of adults, since height is almost completely determined by the time a person is in his or her mid-twenties. Thus, it is possible that the health environment in which an adult lives will be very different from the one in which he grew up. If one is looking at historical data from periods of time in which the environment was changing only slowly, or looking cross-sectionally at countries which differ greatly in their health environments, then this timing effect will not be a serious problem; however, if one looks at countries with rapidly changing health environments, it is a possible concern.

Even where the health environment is changing rapidly, and so adult height is not a good indicator of the health environment in which adults live, it is still the case that adult height provides a lot of information about adult health. The reason for this is that, as recent literature has shown [Fogel 1994], there is a "long reach" of childhood malnutrition and ill health into adulthood. Adults who are shorter because of a poor childhood environment have higher rates of many chronic illnesses in middle and old age. As I show below, the close correlation between adult height and

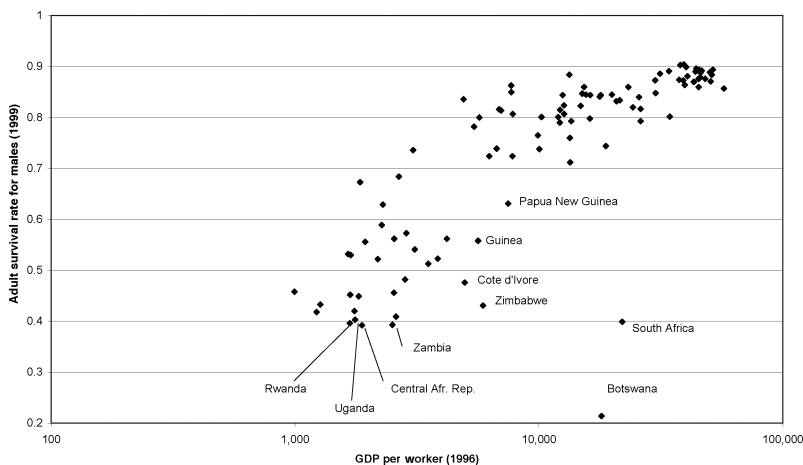


FIGURE I  
GDP per Worker versus Adult Survival Rate

adult mortality rates suggests that height is indeed a good indicator of health.

Height is the most frequently used health indicator in micro-economic studies of the relationship between health and income. Unfortunately, no comprehensive cross-country data set on height exists. Data are available for a modest number of countries. There are also relatively good historical data on height.

#### IV.B. Adult Survival Rate

The second measure of health I use is the adult survival rate (ASR): the fraction of fifteen-year-olds who will survive till sixty years of age, using the current life table.<sup>8</sup> These survival rates are available in consistent form for a large cross-section of countries. The ASR has the advantage of measuring survival during working years and, thus, seems likely to be a good measure of health during working years, which is what should be most relevant for determining the level of output per worker.

Figure I shows the relation between adult survival in 1999 and output per worker in 1996. Figure II shows the unweighted

8. Like the more common measure, life expectancy at birth, the ASR is based on a cross-sectional life table. Thus, it measures how many fifteen-year-olds would die before sixty years of age if, at each age, they experienced the mortality rates of men who are currently that age. Data are from the World Bank.

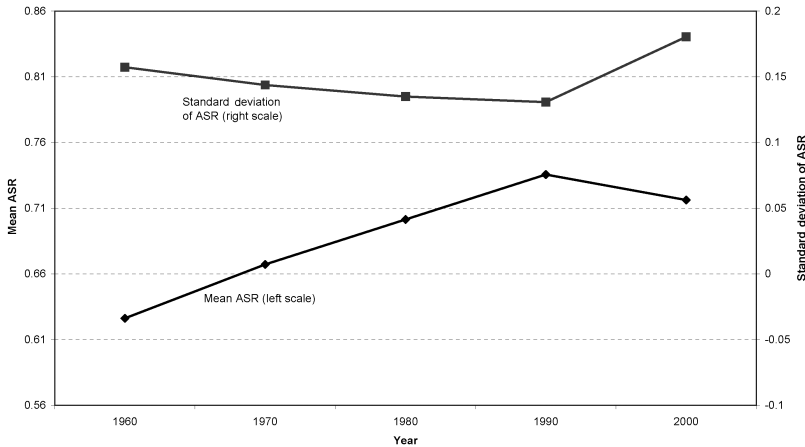


FIGURE II  
Adult Survival Rate

mean and standard deviation of the ASR over the period 1960–2000 for a sample of eighty countries in which survival and income data were available for the entire period. Mean ASR rose, and the standard deviation declined in the period up to 1990, reflecting a worldwide trend toward better health and the catching-up of the poorest countries toward rich country health levels, even though poor country incomes did not systematically grow faster than those in rich countries [Becker, Philipson, and Soares 2005]. The reversal of both of these trends between 1990 and 2000 reflects the impact of AIDS, which dramatically raised mortality rates in several African countries. The impact of AIDS can also be clearly seen in Figure I, in which the largest outliers in terms of low survival are all in sub-Saharan Africa. I discuss the impact of AIDS on my calculations further in Section VII.B.

There are historical data on ASR for a fair number of countries. Unfortunately, there are no good structural estimates linking an individual's health, as proxied by his survival, to wages.

#### IV.C. Age of Menarche

Of all the indicators I use, age at menarche (the onset of menstruation) is the one most foreign to the literature on economic growth. Delayed menarche serves as a good indicator of malnutrition in infancy and childhood. Thus, as countries grow wealthier, girls reach menarche earlier. As shown below, there is



one structural microeconomic estimate of the relation between age at menarche and wages. There is some historical data on the age of menarche in the currently wealthy countries.

To construct a cross-country data set on average age at menarche, I started with four published sources (Eveleth and Tanner [1990], Thomas et al. [2001], Parent et al. [2003], and Padez [2003]), each of which contains a compilation of data from fourteen to sixty-seven countries. When necessary (and possible) I followed the notes in these compilations to find the original studies from which the data came. I also found an additional thirty-two studies by following references and searching databases. I excluded observations that were based on highly non-representative samples (for example, a single economic class or a single locality) and also took only the most recent observation for each country. My data set has forty-nine observations.

Despite these efforts, there remain several problems with the menarche data. Some come from surveys that are not nationally representative, examining women from a few regions, or from the national capital and its environs, for example. There are also cases where the data refer to the median rather than the mean age. Finally, data are from years ranging as far back as 1957, although the vast majority are from the 1980s and 1990s.<sup>9</sup>

Figure III shows the relationship between GDP per capita in 1995 and age of menarche (for various years). The mean age of menarche in my data set is 13.3 years, with a standard deviation of 0.81 years. The five countries with the oldest measured age of menarche are Papua New Guinea (15.8 years), Haiti (15.4), Nigeria (15.0), Somalia (14.8), and Yemen (14.4). The United States has a mean age of 12.4 years, which is the sixth-youngest in the sample.

#### *IV.D. Comparisons of Health Indicators*

For the samples of countries that are used in the empirical analysis below, the correlation between ASR and the log of GDP per capita is .773; between age of menarche and log of GDP per capita is  $-.494$ ; and between ASR and age of menarche is  $-.495$ .

In countries that started developing earliest, there has been a long, gradual improvement in most health indicators, while

9. Because other data are missing, I use only forty-two observations on age of menarche in the analysis in Section VII, with the earliest observation coming from 1968. An annotated data set of the observations I used, as well as the broader data set of all the observations I found, is available upon request.

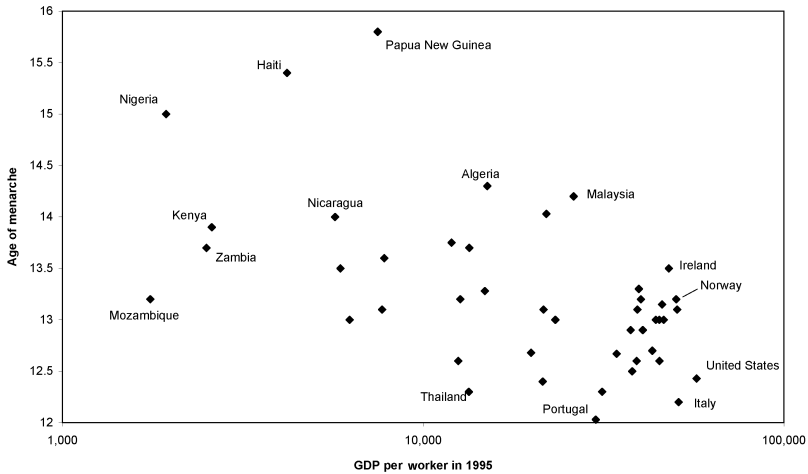


FIGURE III  
Age of Menarche versus GDP per Worker

recent episodes of rapid growth have been accompanied by rapid changes in the health indicators. In Sweden, whose experience is typical for Europe, height increased by 5.5 centimeters between 1820 and 1900 and a further 6.8 centimeters between 1900 and 1965 while ASR rose by 0.179 in the first period and 0.203 in the second. By contrast, in South Korea, the height of adult males rose by 4.8 centimeters and ASR rose by 0.188 over the thirty-three year period, 1962–1995 [Sohn 2000]. Similarly, among industrialized countries in Europe, there was a roughly linear decline in age at menarche of 0.2–0.3 years per decade over the period 1860–1980 [Eveleth and Tanner 1990]. By contrast, in South Korea, age of menarche fell from 16.8 to 12.7 between 1958 and 1998, a decline of more than one year per decade [Hwang et al. 2003].

One concern in looking at health indicators across countries is that there may exist genetic variation that influences the relationship between underlying health and any particular indicator. In the case of age of menarche, Tanner [1990] reports that holding nutrition and environment constant, Africans and Asians reach menarche earlier than do girls of north European descent; thus, estimates of the health gap of Europe and its offshoots versus the rest of the world based on age of menarche will tend to understate the true degree of variation in underlying health. In

the case of height, Steckel [1995] argues that although genetic differences can have some impact on differences in average heights between populations, observed differences are in fact largely attributable to environmental factors. (Despite Steckel's argument, I include fixed effects when I use panel data on average height.)

#### V. ESTIMATING THE RETURN TO HEALTH CHARACTERISTICS

As shown earlier, the return to a health indicator is equal to the change in wages resulting from a specific change in health inputs divided by the change in the health indicator resulting from the same change in health inputs. A naive approach to estimating the return to a health indicator would be to regress log wages on the indicator. Such an approach faces two problems [Schultz 2002]. First, the error term in the equation relating the health indicator to underlying health has a large variance. For example, when height is used as a health indicator, the error reflects genetic heterogeneity in the height that a healthy person attains. Using an indicator to measure underlying health is subject to measurement error, which biases downward the coefficient on the health indicator in a wage regression.

Second, there is likely to be a positive correlation between a person's health and unmeasured determinants of his wage. People with high wages are able to take better care of themselves. And even in the case of aspects of health that are determined early in life (for example, height and age of menarche), people from high-income families are well nourished and cared for as children, and they also carry into the labor market advantages, such as better schooling and family connections, that are not observed by the econometrician. The omission of these factors biases upward the coefficient on a health indicator in a wage regression.

Below I take three approaches to deriving unbiased estimates of the return to health,  $\rho$ .

##### V.A. *Estimating the Return to Health Using Exogenous Variation in Childhood Inputs*

Both of the problems in estimating the return to health discussed earlier can be overcome by using instrumental variables. What is needed is a variable that affects wages only through health, and which is uncorrelated with the unobserved

TABLE I  
STRUCTURAL ESTIMATES OF THE EFFECT OF HEALTH INDICATORS ON WAGES

Health indicator (unit)	Effect on ln (wage)	Sample	Country and year	Source
Height (centimeters)	0.080 (0.0056)	Males 18–60	Colombia (urban), 1991	Ribero and Nuñez [2000]
	0.094 (0.025)	Males 25–54	Ghana, 1987–89	Schultz [2002]
	0.078 (0.0083)	Males 20–60	Brazil, 1989	Schultz [2002]
	–0.261 (0.111)	Females 18–54	Mexico, 1995	Knaul [2000]

determinants of wages. One such type of variable that has been used in the literature is inputs into health in childhood that are not related to family income. These inputs are reflected in health indicators but do not increase wages except through their effect on health. By instrumenting for health indicators with inputs into health, the estimated coefficient in the regression will reflect only the true structural effect of health on wages.

Table I shows the coefficients on health indicators from a variety of individual-level instrumental variables analyses. The instruments used are generally inputs into health in childhood, such as the distance to local health facilities and the relative price of food in the worker's area of origin.<sup>10,11</sup> The regressions in Table I control for years of education as well as the health indicator, so any indirect effects on the level of schooling are not included in the coefficient on health. Further, in all of these regressions, the dependent variable is the log of the hourly wage. Thus, the extent to which good health allows a person to work more hours is not

10. Schultz [2002] also reports estimates in which instruments are parental education and ethnicity. I do not use these estimates because it is questionable whether the instruments can be excluded from the wage equation. He also reports results for the United States. However, because the regional price instruments do not provide a satisfactory basis for instrumenting for height in the wage regression, the IV estimates are not usable.

11. Schultz [2005] reports similar IV regressions for Côte d'Ivoire and Ghana in which both height and body mass index (BMI) are included on the right-hand side, the dependent variable being log male wages. The coefficients are as follows (with standard errors in parentheses): Côte d'Ivoire, height:  $-.011$  (0.019); BMI:  $.159$  (0.053); Ghana, height:  $.057$  (0.017); BMI:  $.079$  (0.041). Because my framework cannot accommodate multiple indicators of underlying health, I do not use these estimates, even though their basic thrust is similar to the estimates that I do use.

accounted for. For both of these reasons, the estimates may understate the effect of health on income.

However, a serious concern related to this set of estimates is the validity of the instruments used and specifically whether the instruments can be excluded from the wage equation. To the extent that good inputs into child health reflect family characteristics that will also lead to high wages through other channels, the effect of health on income estimated using this method will be biased upward. For this reason I treat these estimates as suspect and in my analysis rely primarily on the estimates in the next section.<sup>12</sup>

#### *V.B. Estimating the Return to Health Using Variation in Birth Weight between Twins*

My second method for estimating the return to health uses variation in birth weight between monozygotic twins. Monozygotic twins are genetically identical and also share all aspects of the family environment that might influence aspects of human capital other than health. However, within a pair of monozygotic twins there are variations in birth weight, which presumably reflect differences in intrauterine nutrition due to the location of the fetuses within the womb. These differences in birthweight can be treated as a quasi-experimental source of variation in health.

Behrman and Rosenzweig [2004] analyze a large data set of female monozygotic twins in the United States. In their sample, the average absolute value of the gap in weight is 10.5 ounces (compared to a mean birth weight of 90.2 oz.). Their main estimate (the within-monozygotic twins estimator) regresses the gap in the dependent variable (height, log wages, or schooling) between a pair of twins on the gap in fetal growth (measured in ounces per week of gestation). Conceptually, this is the same as

12. Because it is the only micro-based estimate of the return to age of menarche, I discuss the Knaul [2000] paper in greater detail. Knaul uses eleven instruments: four measures of the quality of housing (such as the percentage of the population with an earth floor or without toilet facilities), three measures of accessibility of health services (such as distance to a health center and number of physicians per capita), and four measures of the level and availability of education (such as literacy and distance to a secondary school). Unlike other papers in this literature, the measures are for the contemporary environment, not for the location where the respondent was raised. Thus, there is the possibility that these measures reflect endogenous migration in response to wages. However, Knaul reports that the results are unchanged looking at a sample of younger women who are less likely to have endogenously migrated in response to their economic situation. She also reports that the sign and significance of the menarche variable are similar in a subsample of women who live with their mothers.

regressing the dependent variable on fetal growth and a full set of family fixed effects. Behrman and Rosenzweig estimate that a one-unit difference in fetal growth leads to a difference of 0.657 years of schooling (standard error 0.211), 3.76 (0.43) centimeters of adult height, and 0.190 (0.077) gap in log wages. Dividing the reduced-form estimate of the effect of fetal growth on wages by the reduced-form estimate of the effect of fetal growth on height yields a TSLS estimate of the effect of health as proxied by height on wages. The estimate is 0.051, or slightly more than 5.1 percent per centimeter.

This estimate includes the effect of health on wages through the channel of education and thus is not comparable to the return to health that is estimated in Section V.A, where education is held constant. To construct a comparable estimate, I start by using the Behrman and Rosenzweig results to produce a TSLS estimate of the effect of health as proxied by height on schooling. Dividing the effect of fetal growth on schooling by the effect of fetal growth on height yields an estimate of 0.175 years extra schooling per centimeter of height. To this figure, I apply a return to schooling of 0.10 (change in the log of wages per year of schooling), which represents a ballpark average of existing estimates [Card 1999]. This implies that each centimeter of height raises log wages by 0.018 through the channel of increased education. Subtracting this education effect from the total effect of height on wages derived above yields an estimate that each centimeter of height raises log wages by 0.033, holding education constant. This number has the same interpretation as the measures of the return to height derived in the previous section.

Black, Devereaux, and Salvanes [2007] perform a similar analysis using data on Norwegian men. Unlike Behrman and Rosenzweig, they do not have data on whether all the twins in their sample are monozygotic or dizygotic, although they demonstrate that among a subsample of twin pairs for which they do have information on zygosity, results for monozygotic twins are very similar to results for same-sex twins. For the full sample, their within-twin-pair estimate of the effect of log birth weight on log earnings is 0.24 (standard error of 0.08), and the estimate of the effect of log birth weight on height is 5.69 (standard error 0.56). The implied effect of health as proxied by height on wages is 4.2 percent per centimeter. Regressing years of education on log birth weight in their data yields a coefficient of 0.40 with a standard error of 0.19 [personal communication from Sandra

Black, April 2006], implying 0.07 years of extra schooling per centimeter of height. Applying the same return to schooling used above (0.10 log points per year of school) and subtracting this education effect from the estimated effect of height wages yields an estimate that each centimeter of height raises log wages by 0.035, holding education constant.

*V.C. Estimating the Return to Health Using Historical Data*

My final technique for estimating the return to health uses long-term historical data. Fogel [1997] analyzes caloric intake and measures of calorie demand in Great Britain over the period 1780–1980. His analysis takes into account both the total quantity of calories consumed and the distribution of these calories across the population. He also carefully accounts for use of calories in basal metabolic maintenance (which increased over this period as people got bigger) in order to calculate how many calories were left over for work. Fogel's conclusion is that increased calorie consumption had two significant impacts on labor supply. First, over this 200 year period, the fraction of the population that was simply too poorly nourished to work at all fell from 20 percent to zero, leading to an increase in labor input by a factor of 1.25. Second, among the adults who were working, increased caloric consumption allowed for a 56 percent increase in labor effort.<sup>13</sup> Combining these effects, improved nutrition raised labor input by a factor of 1.95.

As is clear from the above description, Fogel's analysis looks only at the effects of concurrent and childhood nutrition on a worker's productivity and also focuses solely on the effects of nutrition on the amount of energy a worker had available for expenditure. The analysis leaves out the effects of nutrition on aspects of productivity, such as mental abilities as well as the impact of deficiencies beyond total calories, for example, in micronutrient consumption. Similarly, the analysis excludes the effects of health improvements through sources other than improved nutrition, such as reduced exposure to disease agents and better medical care. (Fogel calculates that improvements in nutritional status accounted for 90 percent of the mortality decline in England between 1775 and 1875 and half of the decline be-

13. More specifically, Fogel finds that the number of calories *available* for work increased by 56 percent over this period and then further assumes, for lack of any data, that the division of energy output between work and "discretionary activities" remained constant.

tween 1875 and 1975.) For all these reasons, the estimate is likely to understate the improvement in worker productivity due to better health that took place over this period.

There are also biases in Fogel's calculation that run in the other direction. Specifically, the limiting factor in many workers' ability to produce output may be mental ability rather than energy or strength. If the component of mental ability related to physical health has changed less than overall energy output, then Fogel's calculation overstates the gain in labor productivity. There has also been a change in the mix of occupations over the period Fogel studies, away from those in which energy output is crucial (manual labor) toward those in which energy is less important. However, this change in occupational mix can be overstated: even among "knowledge workers," those who are energetic can produce more output than those who are lethargic.

For lack of a better benchmark, I assume in this section that Fogel's estimate captures the total health-induced increase in worker productivity over this period. To back out an estimate of the return to health, I compare the change in each health indicator to Fogel's estimate of the change in worker productivity. (This can be thought of as a regression with two data points.) The data on health indicators that I use are as follows. Over the period 1775–1995, average height in Great Britain rose by 9.1 centimeters [Fogel 1994]. In England over the 150 year period from 1832 to 1981, age at menarche declined by a total of 28.5 months [Wyshak and Frisch 1982].<sup>14</sup> I do not make any adjustment for the fact that the time periods applicable for the different sources do not completely match those used by Fogel, under the assumption that in each case the data are capturing the vast majority of the health transition that took place during the time period Fogel considered.

In Section III.A, I showed how the return to a health characteristic can be derived using data from a single country at two points in time. The key equation was

$$(11) \quad \rho_I = \frac{\gamma_v}{\gamma_I} = \frac{\ln(v_{t+1}) - \ln(v_t)}{I_{t+1} - I_t}.$$

Fogel's estimate that  $v$  increased by a factor of 1.95 implies that

14. Great Britain consists of England, Wales, and Scotland. Over the period in question, England accounted for roughly 80 percent of the population of Great Britain.



$$\ln(v_{t+1}) - \ln(v_t) = \ln(1.95) = 0.668.$$

In the case of height, dividing the change in  $\ln(v)$  by the change in height yields a value of  $\rho_{\text{height}} = 0.668/9.1 = 0.073$ ; in other words, the log of labor productivity rises by 0.073 for every centimeter increase in height. Using the same technique yields estimates that log wage rises by 0.281 per year reduction in age of menarche.

*V.D. Estimates of the Return to Health Used in the Rest of the Paper*

The above three methods for estimating the return to health characteristics produce a total of six estimates for the return to height: three from variation in childhood inputs (0.080, 0.094, and 0.078), two from twins (0.033 and 0.035), and one from long-run historical data (0.073). There are two estimates of the return to age of menarche, one from variation in childhood inputs (0.281) and one from historical data (0.261).

The most notable variation among the estimates is in the return to height, with the two estimates based on twins being less than half the estimates from other methods. A possible explanation for this difference is that the twins estimates come from wealthy countries while estimates based on childhood inputs come from poor countries (and the historical estimates are based on comparing a single country when it was rich to when it was poor). If nutrition primarily affects physical capabilities and if these capabilities are less important in rich than in poor countries, one would expect the twins-based estimates to be smaller. Alternatively, the estimates based on childhood inputs may simply be poorly identified and, thus, biased. In what follows, I take the conservative path of using the average of the two twins-based estimates (0.034) as a measure of the return to height. In the case of age of menarche, the two available estimates are quite close to each other, and I use their average (0.271). In Section VII.A of the paper, I discuss the sensitivity of my results to variations in the assumed return to health.

## VI. CROSS-COUNTRY PRODUCTIVITY VARIATION DUE TO HEALTH

Recall that the variable  $v$  measures the element of health that is relevant for labor productivity. To map from a given indicator of health, such as those discussed in Section IV, to the

aspect of health relevant for labor productivity, I use the coefficients derived in Section V. In this section, I focus on three health indicators: age of menarche, height, and adult survival.

In the case of age of menarche, matters are straightforward because both cross-sectional data and an estimate of the return to health as measured by menarche are available. The benchmark estimate of the return to menarche ( $-0.271$  per year) implies that, in my data set, a one standard deviation decline in the age of menarche results in a 24.5 percent increase in labor input per worker. Assuming a value of the capital share parameter  $\alpha$  in the production function of one-third (based on the results in Gollin [2002]), this increase in labor input per worker would raise GDP per worker by 15.4 percent. The gap between the earliest and latest age of menarche countries in my sample (3.7 years) translates into a difference in labor input per worker of a factor of 2.73, and a difference in GDP per worker of a factor of 1.95.

As mentioned above, analysis of cross-country data on health as proxied by height and adult survival faces two problems. ASR data are available for a large cross section of countries, but there is no estimate of the return to ASR; by contrast, there are good estimates of the return to height, but consistent height data are not available for a cross section of countries. In this section I take advantage of the fact that data on both height and ASR are available historically for a number of countries in order to map the structural coefficient on height into a coefficient that can be applied to the data on ASR.

In terms of the framework presented in Section III, I know the return to height,  $\gamma_v/\gamma_{\text{height}}$ , but would like to know the return to ASR, that is  $\gamma_v/\gamma_{\text{ASR}}$ . To convert the former to the latter, I need only multiply by the ratio  $\gamma_{\text{height}}/\gamma_{\text{ASR}}$ . This ratio can be constructed by taking advantage of the assumption that latent health  $z$  is a scalar variable that is linearly related to health outcomes. Specifically, at the country level,

$$(13) \quad \text{height}_{i,t} = \text{constant}_i + \gamma_{\text{height}} z_{i,t} + \epsilon_{i,t},$$

$$(14) \quad \text{ASR}_{i,t} = \text{constant} + \gamma_{\text{ASR}} z_{i,t} + \mu_{i,t},$$

where  $i$  indexes countries and  $t$  indexes time. Each equation includes a white noise error term due to measurement error. Further, I include country fixed effects in the equation for height to allow for genetic variation in how this health indicator is related to underlying health.

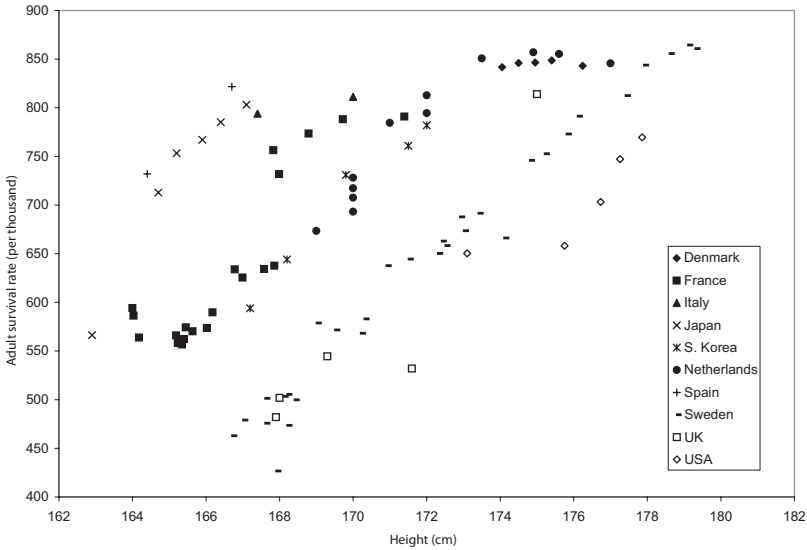


FIGURE IV  
Data on Height and Adult Survival

These two equations can be rearranged as follows:

$$(15) \quad \text{height}_{i,t} = \text{constant}_i + \frac{\gamma_{\text{height}}}{\gamma_{\text{ASR}}} \text{ASR}_{i,t} + \epsilon_{i,t} + \frac{\gamma_{\text{height}}}{\gamma_{\text{ASR}}} \mu_{i,t}.$$

Thus the ratio of coefficients  $\gamma_{\text{height}}/\gamma_{\text{ASR}}$  can be estimated by regressing height on ASR with a set of country fixed effects. To implement this regression, I constructed a data set with information from ten countries and data covering up to 180 years per country, for a total of ninety-three observations. The data are shown in Figure IV.<sup>15</sup>

Table II presents the results of this regression, as well as results from alternative specifications. Columns (1) and (2) show the basic regression of height on ASR, excluding and including country fixed effects. Excluding country fixed effects (column (1)) reduces the coefficient only slightly. Column (3) adds a linear time trend to the fixed effects regression, which reduces the coefficient on ASR by 40 percent of its initial value. Column (4) includes an interaction of ASR with year (normalized to be zero in

15. Data available upon request. Most of the data on height come from Arora [2001].

TABLE II  
REGRESSION OF HEIGHT ON ADULT SURVIVAL RATE

	(1)	(2)	(3)	(4)
Constant	156.0 (1.0)	157.5 (0.8)	166.2 (2.2)	165.2 (2.1)
Adult survival rate	21.1 (2.8)	26.4 (1.0)	16.6 (2.5)	19.2 (2.4)
Year			0.0292 (0.0068)	-0.0057 (0.0123)
Year $\times$ ASR				0.0719 (0.0216)
Country fixed effects	No	Yes	Yes	Yes
$R^2$	0.377	0.953	0.961	0.966

Standard errors in parentheses.  $N = 93$  for all regressions. Height is measured in centimeters. Year is normalized to be zero in 2000. In regressions with country fixed effects, the United States is the reference group.

2000) to allow for change over time in the relationship between ASR and height. The coefficient on the interaction term is positive and significant, implying that changes in ASR are associated with larger increases in height today than they were in the past. For example, a change of 0.1 in the ASR would produce an increase in height that was 0.71 centimeters greater in the year 2000 than it would have produced in the year 1900. The coefficient on ASR in this column, 19.2, implies that a difference in the adult survival rate of 0.1 (100 deaths per thousand) is associated with a difference in height of 1.92 centimeters. It is this estimate of the effect of height on ASR that I use in the rest of the paper.

The estimate of  $\gamma_{\text{height}}/\gamma_{\text{ASR}} = 19.2$  in column (4), along with the value of  $\rho_{\text{height}} = 0.034$  derived above, yields a value of  $\rho_{\text{ASR}} = 0.653$ .<sup>16</sup> This coefficient implies that an increase in the adult survival rate of 0.1 would translate into an increase in labor input per worker of 6.7 percent and thus GDP per worker of 4.4 percent. In my data, the adult survival rate ranges between 0.214 (Botswana) and 0.904 (Iceland). Applying the coefficient of  $\rho_{\text{ASR}} = 0.653$  implies that moving from the lowest to the highest level of

16. An earlier version of this paper, Weil [2005], reported an estimate of  $\rho_{\text{ASR}} = 1.90$ . The difference between that estimate and the one presented here is completely accounted for by two changes. First, in the previous version of the paper, I used a value of the estimated return to height of  $\rho_{\text{height}} = 0.072$ , which was the average of the five structural estimates I had available at the time; in this version, I use the value of 0.034 based on the two twins-based studies. Second, in the previous version, I used the estimate of  $\gamma_{\text{height}}/\gamma_{\text{ASR}} = 26.4$  from column (2) of Table II, rather than the value of 19.2 from column (4) of the same table, which I use in this version.

adult survival would raise labor input per worker by a factor of 1.59 and output per worker by a factor of 1.35.

Before proceeding further, it is useful to compare this derived effect of health on income to estimates using cross-country data, as discussed above. Bloom and Canning [2005] examine the effect of health on labor productivity, using the same approach as Bloom, Canning, and Sevilla [2004], which was discussed earlier. However, Bloom and Canning [2005] use the adult survival rate as their measure of health and further write their production function with human capital in the form of health multiplying labor input, as in (2). These changes make their results directly comparable to the effect of health on labor input that I measure here. They estimate a value of  $\rho_{ASR}$  of 2.8, with a 95 percent confidence interval of 1.2–4.3. Even the lower bound of their range is twice my estimate of  $\rho_{ASR} = 0.653$ .

#### VII. THE CONTRIBUTION OF HEALTH TO INCOME DIFFERENCES AMONG COUNTRIES

I now turn to examine the contribution of differences in health to differences in income among countries. Specifically, I extend the development accounting methodology of Klenow and Rodriguez-Clare [1997] and Hall and Jones [1999] to include a measure of health.

Start with the aggregate production function introduced above, in log-per capita terms:

$$(16) \quad \ln(y_i) = \ln(A_i) + \alpha \ln(k_i) + (1 - \alpha) \ln(h_i) + (1 - \alpha) \ln(v_i).$$

All the terms in this equation, with the exception of productivity,  $A_i$ , can be observed directly. Thus, this equation can be used to back out productivity as a residual. To implement this procedure, I use the estimates of  $v$  constructed in the previous section. Data for real GDP per worker, physical capital, and human capital in the form of education are drawn from Caselli [2005]. Output and physical capital are measured in 1996 while human capital is from 1995. As mentioned earlier, I use a value of one-third for  $\alpha$  based on the findings in Gollin [2002].

I take several approaches to answering the question of how much variation in income is explained by health. The upper part of Table III shows the full set of variances and covariances of the terms on the right-hand side of (16), all scaled by the variance of  $\ln(y)$ . From these terms, two summary statistics can be calcu-

TABLE III  
SHARES OF VARIATION IN OUTPUT PER WORKER ATTRIBUTABLE TO EACH FACTOR

Sample	ASR ( $N = 92$ )		Menarche ( $N = 42$ )		
	None	ASR	None	Age of menarche	ASR
Health Indicator Adjusted for:	(1)	(2)	(3)	(4)	(5)
var $(\ln(y))$	1.22	1.22	0.888	0.888	0.888
var $(\alpha \ln(k))/\text{var}(\ln(y))$	0.221	0.221	0.242	0.242	0.242
var $((1 - \alpha) \ln(h))/\text{var}(\ln(y))$	0.032	0.032	0.038	0.038	0.038
var $(\ln(A))/\text{var}(\ln(y))$	0.179	0.144	0.175	0.154	0.139
cov $(\alpha \ln(k), (1 - \alpha) \ln(h))/\text{var}(\ln(y))$	0.074	0.074	0.083	0.083	0.083
cov $(\ln(A), \alpha \ln(k))/\text{var}(\ln(y))$	0.161	0.137	0.150	0.111	0.123
cov $(\ln(A), (1 - \alpha) \ln(h))/\text{var}(\ln(y))$	0.048	0.040	0.040	0.028	0.032
var $((1 - \alpha) \ln(v))/\text{var}(\ln(y))$		0.004		0.021	0.005
cov $(\alpha \ln(k), (1 - \alpha) \ln(v))/\text{var}(\ln(y))$		0.024		0.039	0.027
cov $((1 - \alpha) \ln(h), (1 - \alpha) \ln(v))/\text{var}(\ln(y))$		0.008		0.012	0.008
cov $(\ln(A), (1 - \alpha) \ln(v))/\text{var}(\ln(y))$		0.015		0.000	0.015
Fraction of variance in $\ln(y)$ attributable to productivity	0.598	0.529	0.555	0.431	0.480
Proportional reduction in variance of $\ln(y)$ from eliminating health gaps		0.099		0.123	0.106

lated, which are presented at the bottom of Table III. The first is the fraction of variance in  $\ln(y)$  that is attributable to productivity. This is the sum of  $\text{var}(\ln(A))$  along with two times all the covariance terms that contain  $\ln(A)$ . The reduction in this term resulting from accounting for health is a measure of the success of health in reducing the fraction of income variance attributable to unobservable productivity. The second measure is the proportional reduction in the log variance of income that would be achieved by eliminating health gaps among countries. This is the sum of  $\text{var}(\ln(v))$  and two times all the covariance terms that contain  $\ln(v)$ .

Finally, in Table IV, I look at nonvariance measures of dispersion of income: specifically, the ratio of GDP per worker in the

TABLE IV  
EFFECT OF ELIMINATING HEALTH GAPS ON INCOME RATIOS

Sample	Health measure	Income ratio	Raw data	Eliminating health gaps
ASR	ASR	90/10	20.47	17.88
		90/50	3.21	3.08
		50/10	6.37	5.80
Menarche	ASR	90/10	10.05	9.21
		90/50	1.75	1.71
		50/10	5.74	5.39
Menarche	Menarche	90/10	10.05	7.76
		90/50	1.75	1.82
		50/10	5.74	4.25

country at the 90th percentile of the income distribution to GDP per worker of the country at the 10th percentile of the income distribution, which I label  $y_{90}/y_{10}$ . I further decompose this into the 90th percentile/median ratio, and the median/10th percentile ratio. These measures of dispersion are calculated both for the raw data and then under the assumption that health differences among countries were eliminated. The latter measure is constructed by summing the terms on the right-hand side of (16), excluding the term  $(1 - \alpha) \ln(v_i)$ , which captures the effect of health.

The analyses in Tables III and IV are carried out using both ASR and age of menarche to construct country-specific estimates of human capital in the form of health,  $v_i$ . Data to carry out the analysis using ASR are available for ninety-two countries, and for analysis using age of menarche they are available for forty-two countries (which are a subset of the ASR sample). I also show results using ASR as a measure of health in the sample of countries for which menarche data are available.

The ASR estimates in Table III imply that eliminating health gaps among countries would reduce the variance of log output per worker among countries by 9.9 percent (or 10.6 percent when ASR is used as a health measure in the menarche sample); using menarche as a health measure, the reduction would be 12.3 percent. Accounting for health using ASR reduces the fraction of variance in  $\ln(y)$  attributable to residual productivity by seven

percentage points; using menarche, the reduction is twelve percentage points. The fact that the menarche and ASR estimates are fairly similar, given that they are constructed using different data sources and different methodologies, is encouraging.

The results in Table IV are largely in line with Table III. Using ASR as a health measure, the elimination of health gaps in the ASR sample would reduce the 90/10 income ratio from 20.5 to 17.9, a decline of 12.7 percent of its initial value. Not surprisingly, the largest part of this reduction would come from the lower part of the distribution. The 50/10 income ratio would fall from 6.4 to 5.8 (a decline of 8.9 percent), while the 90/50 ratio would fall from 3.2 to 3.1 (a decline of 4.1 percent). Thus, health's effect on GDP is strongest among the poorer half of countries. As in Table III, the importance of health is somewhat larger using menarche as a measure than using ASR.

The consistent message from Tables III and IV is that health has an economically important effect in determining income differences among countries but that this effect is far from the dominant source of cross-country income variation. As seen in Table III, using either health measure, health is less important than both human capital from education and physical capital (especially the latter) as a explainer of income differences among countries. Similarly, residual productivity is still left as the most important determinant of income differences among countries, although according to some of my estimates (specifically, within the menarche sample), productivity no longer ranks as being more important than all other factors taken together. A world in which health was equalized among countries would still have 90 percent of the cross-country income variance left intact.

#### *VII.A. Sensitivity Analysis*

My estimates of the proximate effects of health on GDP differences among countries are built up from data on how health indicators vary among countries and estimates of the return to health. In the case of health as measured by ASR, the health indicator itself is of relatively high quality and is measured on a uniform basis, and so the most likely source of error is in the calculation of the return to health. This is particularly true because in this case the return to health is itself the product of two



other estimates: the return to height and my estimated mapping between changes in ASR and changes in height.

As discussed in Section V.D, the various methods that I used produced six estimates of the return to height. In the calculations above, I used the value of  $\rho_{\text{height}} = 0.034$  (3.4 percent per centimeter), the average of the two estimates based on twins, which I considered the most trustworthy because they are based on a well-identified experiment. As an alternative, I recalculated my results using a value of  $\rho_{\text{height}} = 0.066$ , which is the average of all six estimates. This, in turn, implies a value of the return to adult survival,  $\rho_{\text{ASR}} = 1.27$ , compared to my benchmark estimate of  $\rho_{\text{ASR}} = 0.653$ . Using this alternative measure, the proportional reduction in the variance of  $\ln(y)$  that results from eliminating health gaps among countries rises to 18.4 percent, as compared to 9.9 percent in the base case. Similarly, using this alternative value for  $\rho_{\text{ASR}}$ , eliminating health gaps among countries would lower the 90/10 income ratio from 20.5 to 15.9 (a reduction of 22.1 percent of its initial value) as compared to a reduction of 12.7 percent of its initial value in the base case. By either of these measures, increasing the value of  $\rho_{\text{ASR}}$  slightly less than two-fold raises the implied contribution of health to income differences by a factor of slightly less than two. Thus, although the measures of health's importance that I calculate are not exactly linear in the estimate of  $\rho_{\text{ASR}}$  used, they are not far from it in the range being considered here. Similar roughly proportional results are obtained if the other factor that goes into determining  $\rho_{\text{ASR}}$ , that is, the mapping from changes in ASR into changes in height,  $\gamma_{\text{height}}/\gamma_{\text{ASR}}$ , is varied among the different values estimated in Table II.

In the case of health as measured by age of menarche, the estimated return to menarche is arguably less likely a source of error than mismeasurement of menarche itself. As mentioned above, my data on menarche come from a variety of years, and in some cases refer to the median rather than the mean. A regression of age of menarche on a dummy for median and year of measurement yields the following (standard errors in parentheses):

$$\begin{array}{rcc} \text{Age of menarche} = & 51.98 & -0.105 \text{ median} & -0.0195 \text{ year} \\ & (25.46) & (0.264) & (0.0128) \\ & & & R^2 = 0.054 \\ & & & (N = 49) \end{array}$$

Using the residuals from this regression in the accounting exer-

cise above implies that eliminating health gaps would reduce the variance of  $\ln(y)$  by 12.0 percent, which is hardly different from the original estimate of 12.3 percent.

### *VII.B. The Changing Relationship between Adult Survival and Income*

As mentioned in Section III.C, my method for finding the return to a health outcome,  $\rho$ , is biased in the case in which the ratio of changes in worker productivity to changes in the indicator induced by the experiment used to measure  $\rho$  is different from the ratio of changes in worker productivity to changes in the indicator in cross-country data. In the case of the ASR, there is reason to worry that just such a bias may be present, due to AIDS. As shown in Figure I, a significant fraction of the variance in ASR in the year 1999 is due to high AIDS mortality in developing countries. Young [2005] argues that, in comparison to other diseases, AIDS has a disproportionately large effect on mortality relative to its affect on morbidity (that is, the health of workers). Specifically, in the case of sub-Saharan Africa, where advanced medical treatment for AIDS is rare, individuals infected with HIV experience a long asymptomatic period during which their labor productivity is unaffected by the disease, following which they sicken and rapidly die. If this is the case, my estimates of the variation in human capital in the form of health among countries, using adult survival to measure health, will be overstated.

One way to address this issue would be to look at data on how adult survival would vary among countries in the absence of AIDS. Such data do not exist, however. As an alternative, I look at data from the period before AIDS became widespread. Specifically, using data on output per worker and ASR, I construct some of the measures that were examined earlier: the proportional reduction in the log variance of GDP per worker and the proportional reductions in the 90/10, 90/50, and 50/10 income ratios that would result from eliminating health gaps among countries.

I construct these measures for every decade over the period 1960–2000. In the year 2000, there were 2.4 million AIDS deaths in Africa, compared to 450,000 in 1990 and almost none in 1980. The data are a panel of eighty countries for which data on GDP per worker and adult survival were available over the period

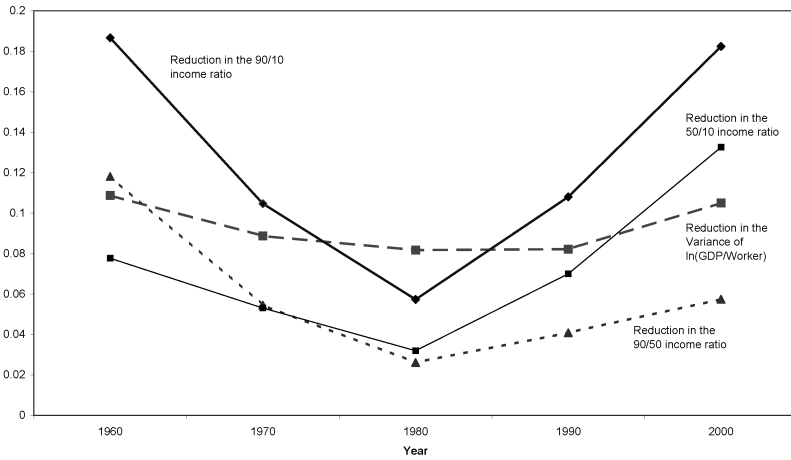


FIGURE V  
The Effect of Eliminating Health Gaps among Countries

1960–2000.<sup>17</sup> I use the benchmark estimate of the return to adult survival of  $\rho = 0.653$  derived above. Figure V shows the results. Comparing 1990 and 2000, there is indeed a significant increase (2.3 percentage points, or 22 percent of the 2000 value) in the proportional reduction in income variance that would result from eliminating health gaps. If we assume that this entire increase was due to AIDS and that AIDS had no effect at all on the labor input of workers, then the other calculations in Tables III and IV would have to be adjusted downward accordingly.

Besides addressing the issue of bias induced by AIDS, Figure V is interesting in its own right. It shows that in the period prior to 1980, convergence in health among countries significantly reduced the fraction of world income variation attributable to health. Because this convergence in health was due to progress against a number of diseases, it is less likely that the bias in measuring the return to health discussed earlier is present, and, thus, it is less likely that decline in health's importance as a determinant of GDP variation is a statistical illusion.

17. GDP per worker is the variable RGDPWOK from Heston, Summers, and Aten [2002]. Adult Survival is from the World Development Indicators database of the World Bank.

### VII.C. Indirect Effects of Health on Income

As discussed in the introduction, the approach taken in the rest of this paper has been to examine only the proximate or direct effects of health on GDP per worker. In other words, I have considered the counterfactual of improving worker health while holding constant the amount of physical capital per worker, the average level of education, the quality of institutions, and so on.

While this calculation flows directly from the microeconomic studies of the effect of health on individual wages that I start with, one would also like to know total effect of health on income, that is, the sum of the direct effect and any indirect effects of health through other channels. Here I consider two such indirect effects: first, the effect of improved health in raising the level of education that individuals attain, and, second, the effect of health in raising the quantity of physical capital per worker. Healthier individuals have an incentive to get more education because they can amortize their investment over more working years (note that healthier students also learn more for every year that they are in school, but this effect was already incorporated into my estimated return to health). A healthier workforce, which supplies more efficiency units of labor per worker, also attracts more physical capital.

To adjust for the increase in years of schooling that results from improved health, I go back to the twins studies discussed in Section V.B. There, I started with TSLS estimates of the effect of height on log wages and then adjusted for part of the wage increase that was due to education by subtracting an estimate of the effect of height on education multiplied by an estimate of the return to education. Here I simply use the unadjusted TSLS estimate of the return to height. The two studies discussed in Section V.B reported returns to height of 5.1 and 4.2 percent per centimeter. I use the average of these two, 4.65 percent per centimeter. With this estimated return to height, I construct a measure of  $v_i$ , redefined to be human capital in the form of health *inclusive* of the effect of health in raising years of schooling.

To adjust for the increase in physical capital that results from healthier workers, I follow Hall and Jones [1999] in decomposing output per worker, holding constant the capital/output ratio, as would be the case in the steady state of a Solow model.

Specifically, the production function can be rearranged as

$$(17) \quad \ln(y_i) = \frac{1}{1-\alpha} \ln(A_i) + \frac{\alpha}{1-\alpha} \ln\left(\frac{k_i}{y_i}\right) + \ln(h_i) + \ln(v_i).$$

I use the adult survival rate to proxy for health. As a measure of health's importance, I focus on the proportional reduction in the variance of  $\ln(y)$  from eliminating health gaps among countries. This can be calculated as the sum of the variance of  $\ln(v)$  plus twice the covariance of  $\ln(v)$  with all the other terms on the right-hand side of (17), all divided by the variance of  $\ln(y)$ . The direct effect reported in Table III is 9.9 percent. The sum of the direct and indirect effects considered here is 19.3 percent. In other words, the two indirect effects considered here are roughly as important as the direct effect of health. Adjusting for schooling but not physical capital, the effect is 13.3 percent, while adjusting for physical capital but not schooling, the effect is 14.5 percent.

Although this result seems to suggest that the combined direct and indirect effects of health are significantly larger than the direct effect analyzed in the rest of the paper, it is important to note that I have considered only some of the indirect effects of health. Most significant among the effects that are left out is that health improvements can trigger a rise in population growth, which will negatively impact output per worker both through the "Solow" channel (dilution of the capital stock) and the "Malthus" channel (reductions in the quantity of land per worker). Acemoglu and Johnson [2006] argue that the negative effects of population on income explain their puzzling finding that, in a cross-country regression, improvements in life expectancy reduce income per capita and income per worker. A second important consideration that is ignored in the above calculation is the dynamic aspects of the effect of health improvements on the economy. Increases in schooling due to better health will not affect output until the children who get more schooling enter the labor force. Similarly, the calculation of the effects of health on capital assumed that the capital/output ratio was in steady state. In fact the adjustment of the capital stock following a health shock would take many decades. Finally, the demographic effects of a change in health, including an initial increase in population growth and subsequent changes in the age structure of the population, would play out over a very long period of time. Ashraf, Lester, and Weil [2006] undertake a detailed analysis of the different indirect

channels through which health affects output, taking these dynamic considerations explicitly into account.

### VIII. CONCLUSION

In this paper I have presented a methodology by which properly identified estimates of the effect of variation in health inputs on individual income and health outcomes can be applied to cross-country data on health indicators in order to estimate the direct effect of health on income. My baseline estimate, using the adult survival rate for men as a measure of health, is that eliminating health gaps among countries would reduce the variance of log GDP per worker by 9.9 percent. Eliminating health gaps would also reduce the ratio of income of the country at the 90th percentile of the income distribution to income at the 10th percentile by 12.7 percent of its initial value, with the bulk of the decline taking place in the median/10th percentile ratio. A parallel set of estimates, using the age of menarche as a health indicator, show health playing a slightly larger role. The conclusion that health is an important determinant of income variation is robust to using a variety of different microeconomic and historically based estimates of the return to health as well as to using alternative estimates of the mapping between different health indicators and adjusting for the role of AIDS in affecting mortality in the 1990s.

While the effect of health on income that I estimate is economically significant, it is also much smaller than existing estimates derived from cross-country regressions. My estimates do not match the characterization of ill health as a major stumbling block to economic development as described in the WHO report on macroeconomics and health cited in the introduction.

A limitation of the methodology that I present is that it can only examine the proximate effect of health—that is, people working harder, longer, or more intelligently—on GDP per worker. In addition to this proximate effect, there are a number of indirect channels through which health affects a country's output. Several of these indirect channels, such as the effect of better health in encouraging the accumulation of human and physical capital, could have positive impacts on income that are as large as the direct channel that I have measured in this paper. However, there is one indirect channel that works in the opposite direction: the effect of better health in increasing population growth.

Higher population growth lowers GDP per capita through the mechanical effect of raising the ratio of children to adults; it can also lower GDP per worker through reductions in the quantities of physical capital and land per worker.

In principle, all of the indirect effects of health on income that are discussed above can be quantitatively assessed using a combination of microeconomic estimates and macroeconomic models. Such an analysis will also have to account for the different speeds at which health affects the economy through various channels. Putting all of these effects together to paint a picture of the total effect of health improvements on income remains a topic for future research.

#### APPENDIX: ALLOWING LATENT HEALTH TO BE TWO DIMENSIONAL

As a way of thinking about the biases resulting from the assumption that latent health is unidimensional, I here consider the case in which there are two latent health characteristics, one of which,  $z$ , is relevant to an individual's productivity in the labor market and the other of which,  $\zeta$ , is not. I assume that both aspects of health can affect the observed health indicator,  $I$ . Specific variations in the vector of health inputs,  $\mathbf{X}$ , will affect one or both of the aspects of underlying health.

Consider two countries with vectors of health inputs  $\mathbf{X}_1$  and  $\mathbf{X}_2$ . The true difference in their levels of human capital in the form of health will be

$$(18) \quad \ln(v_2) - \ln(v_1) = \gamma_v(z(\mathbf{X}_1) - z(\mathbf{X}_2)).$$

Let  $\gamma_I^z$  be the coefficient giving the effect of the latent health measure  $z$  on the observable health outcome  $I$ , and similarly  $\gamma_I^\zeta$  be the coefficient giving the effect of the latent health measure  $\zeta$  on the observable outcome  $I$ . The difference in  $I$  is given by

$$(19) \quad I_2 - I_1 = \gamma_I^z(z(\mathbf{X}_1) - z(\mathbf{X}_2)) + \gamma_I^\zeta(\zeta(\mathbf{X}_1) - \zeta(\mathbf{X}_2)).$$

Using microeconomic data in which one element of the health input vector,  $x$ , is varied exogenously, the estimated return to characteristic  $I$  is

$$(20) \quad \hat{\rho}_I = \frac{dw/dx}{dI/dx} = \frac{(dz/dx)\gamma_v}{(dz/dx)\gamma_I^z + (d\zeta/dx)\gamma_I^\zeta}.$$

Thus the estimated difference in human capital in the form of health is

$$(21) \quad \hat{\rho}_I(I_2 - I_1) = \frac{(dz/dx)\gamma_v}{(dz/dx)\gamma_I^z + (d\zeta/dx)\gamma_I^\zeta} \\ \times [\gamma_I^z(z(\mathbf{X}_1) - z(\mathbf{X}_2)) + \gamma_I^\zeta(\zeta(\mathbf{X}_1) - \zeta(\mathbf{X}_2))],$$

which can be rearranged to give

$$(22) \quad \hat{\rho}_I(I_2 - I_1) = [\gamma_v(z(\mathbf{X}_1) - z(\mathbf{X}_2))] \\ \times \frac{(1 + (\gamma_I^\zeta/\gamma_I^z)((\zeta(\mathbf{X}_1) - \zeta(\mathbf{X}_2))/(z(\mathbf{X}_1) - z(\mathbf{X}_2)))}{1 + (\gamma_I^\zeta/\gamma_I^z)(d\zeta/dx)/(dz/dx)}.$$

The term in square brackets in this expression is the actual gap in human capital in the form of health between the two countries from (18). The other part of the expression is the bias in estimating this gap. Several points can be noted about this bias term:

- If  $\gamma_I^\zeta = 0$ , that is, the aspect of health that is irrelevant for  $v$  is also irrelevant for the indicator  $I$ , then there is no bias.
- If  $\gamma_I^\zeta > 0$ , then the sign of the bias will depend on how the variation in the two dimensions of health induced by the experiment being used to estimate the return to the characteristic (that is, the ratio of  $d\zeta/dx$  to  $dz/dx$ ) compares to the actual variation in the two dimensions between two countries (that is, the ratio of  $(\zeta(\mathbf{X}_1) - \zeta(\mathbf{X}_2))$  to  $(z(\mathbf{X}_1) - z(\mathbf{X}_2))$ ). If these two ratios are equal, then once again there is no bias. The gap in  $v$  between two countries will be overstated in cases where variation in the aspect of health that is irrelevant for producing output ( $\zeta$ ), relative to variation in the relevant aspect of health ( $z$ ), is larger among countries than the variation induced by the experiment being used to estimate the return to health.
- If there is bias present from the above cause, then the absolute size of this bias will be magnified, the greater is the ratio  $\gamma_I^\zeta/\gamma_I^z$ , that is, the more that the aspect of health that is irrelevant to output affects the observable health indicator relative to the effect of the relevant measure of health on the observable indicator.

One can do similar algebra to deal with the case (described in Section III.B and applied in Section V.C) where the return to health is estimated by examining changes over time in  $v$  within a single country. The estimated gap in  $v$  between countries will be



biased if the ratio of changes in the different elements of latent health ( $z$  and  $\zeta$ ) that took place in the relevant historical experiment does not match the ratio of differences in these two elements of latent health between countries.

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