

## **Accounting for the Effect of Health on Economic Growth**

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### **Abstract**

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 use a variety of methods to construct estimates of the return to health, which I combine with cross-country and historical data on several health indicators including height, adult survival, and age at menarche. My preferred estimate of the share of cross-country variance in log income per worker explained by variation in health is 22.6%, roughly the same as the share accounted for by human capital from education, and larger than the share accounted for by physical capital. I present alternative estimates ranging between 9.5% and 29.5%. My preferred estimate of the reduction in world income variance that would result from eliminating health variations among countries is 36.6%.

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## 1. 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 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 raise the level of 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 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 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 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 answer the question of

how much of a gain in income for poor countries would result from an improvement in the health of their citizens.

Economists have identified a large number of channels through which health affects the level of output in a country. One channel, which I call the proximate 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. The effect of better health on population growth is ambiguous. In the short run, higher child survival may lead to more rapid population growth. Over longer horizons, however, lower infant and child mortality can lead to a more-than-offsetting decline in fertility, so that the Net Rate of Reproduction falls.<sup>1</sup> 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 allowing workers to work harder and more intelligently, holding constant the level of physical capital, education, the quality of institutions, and so on. Beyond the practical difficulty of fully accounting for all of the channels by which health affects income that are discussed above, there are two other reasons for taking this approach.

The first reason I restrict myself to looking at the proximate effects of health on income is the endogeneity of health itself. The mechanisms that lead to a positive dependence of health on

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<sup>1</sup>Bloom and Canning (2000), Kalemli-Ozcan, Ryder, and Weil (2000).

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>2</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 effects 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. Because there are no structural microeconomic estimates of most of the indirect effects of health on income discussed above, I cannot apply a similar methodology to look at these channels.

The second reason that I focus on only the proximate effects of health on income is to avoid the problem of double counting. Many of the indirect effects of health on national income that are discussed above run through channels, such as education and the accumulation of physical capital, that are already accounted for in conventional analyses of growth. If we account for the indirect effect of health on income through, for example, the channel of education, then it would only make sense to similarly account for the indirect effect of education on income through the channel of health. Such an exercise rapidly becomes untenable. Looking at the proximate effect of each of these factors on national income is a decomposition which, even if it does not answer all the questions we might be interested in, is conceptually clean.

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<sup>2</sup> 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 US) 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 (2000) takes the view that differences in the fundamental health environment between countries are not large, and that high level of disease in tropical countries is more a result than a cause of their poverty.

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, forthcoming, 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 productivity is by far the most significant source of income differences, explaining more than half of the variance of income. By accounting for variation in health among countries, I am able to explain a significant fraction of this residual productivity variation.

The rest of this paper is organized as follows. Section 2 discusses previous literature that examined the link between health and economic outcomes. Section 3 presents a theoretical framework for analyzing how health affects income at the individual and national level. Section 4 discusses the aggregate health indicators used in my analysis. Section 5 presents a variety of estimates of the return to health indicators. Section 6 looks at the magnitude of productivity differences among countries implied by differences in health outcomes, and Section 7 looks at the contribution of health variation to variation in GDP among countries. Section 8 concludes.

## **2. 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. Among the most important inputs into health are nutrition at various points in life (in utero, in childhood, and in adulthood), where nutrition includes both total calories and protein, as well as the presence of important micronutrients. Examples of other inputs into health are exposure to pathogens and medical care.

Health outcomes are characteristics that are determined both by an individual's health inputs and by his genetic endowment. Examples of health outcomes 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." Presumably human capital in the form of health is some combination of ability to work hard, cognitive function, and possibly other aspects of health. Of course, we do not observe human capital in the form of health directly at the individual level, but we can infer its determinants by looking at the determinants of wages. Nor can human capital in the form of health be observed at the level of countries.

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. Designating these measures as indicators is not meant to imply that they are not important. Indeed, for individuals, many health indicators (such as the probability of dying) are more important than ability to produce output. I call these measures indicators only because they are not direct measures of the aspect of health that I examine in this paper.

Comparisons of health among countries can be made by looking at either inputs to health or health indicators. Although we do not have good measures of all health inputs, the available data suggests that there are very large differences in many health inputs between poor and rich countries, with rich countries having better average values of almost all health inputs. 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 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. This is the basis for inferring that health differences play a proximate role in explaining income differences among countries.

A large microeconomic literature examines the effects of varying health inputs on either health outcomes themselves, human capital attributes that are contingent on health outcomes, or wages. In many studies, more than one of these groups of dependent variables is examined.<sup>3</sup>

A large number of studies have examined the long-run effects of childhood nutrition. Behrman et al. (2003) study the effect of nutrition interventions among children aged six through 24 months, using data from the well known INCAP experiment. They find that higher school completion and improved performance on an intelligence test among children who were exposed to the protein supplement Atole. Alderman, Hoddinott, and Kinsey (2003) examine the long-term consequences of early childhood nutritional status in Zimbabwe, using exposure to civil war and drought as instruments for malnutrition, and controlling for family fixed effects by looking at siblings not similarly exposed. They find that exposure to drought at ages 12-36 months led to a 2.3 cm. decline in adolescent height relative to siblings, as well as a reduction of 0.4 grades of schooling, while exposure to civil war had similar, though much smaller, effects. Maccini and Yang (2005), examining Indonesian women born 1953-1974, find that higher than average local rainfall in year of birth led to women being 0.5 centimeters taller and obtaining 0.32 more years of schooling. A small longitudinal intervention study in the Mexican village of Tezonteopan (Chavez, Martinez, and Soberanes, 1995) compares a group of children whose mothers received supplemental nutrition at the onset of pregnancy, and who themselves received supplemental nutrition through age 10, with a similar group of unsupplemented children. The gap in average IQ at age 18 between supplemented and unsupplemented groups was 10 points for men and 7 points for women, while the gap in average height was 7.0 centimeters for men and 9.6 cm. for women. Supplemented women also reached menarche an average of 1.5 years before unsupplemented women. Berhman and Rosenzweig (2004) using variation in birth weight among

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<sup>3</sup>See Thomas and Frankenberg (2002) for an extensive review.

monozygotic twins as an instrument, find significant effects of intrauterine nutrition on adult stature, schooling, and wages.

Among studies that have examined the effects of other aspects of children's health environment, Bleakley (2002) finds that hookworm eradication led to a significant increase in school enrollment in the American South, while Miguel and Kremer (2005) find that provision of deworming drugs reduced school absenteeism and stunting of growth among children in Kenya. Several other studies that have examined the effects of childhood nutrition and health inputs are discussed in Section 5.1.

A number of studies have also examined the effects of contemporaneous changes in nutrition or other health inputs. Strauss (1986) finds that the labor input of farm workers rises with their caloric consumption. Strauss (1997) finds that higher protein consumption raised hourly wages among urban workers in Brazil. Basta et al. (1979) find an increase in output of Indonesian rubber tappers provided with iron supplementation compared to those that received a placebo. Thomas et al. (2004) find significant positive effects of iron supplementation on physical health and economic success in a large, randomized trial. Gruber and Hanratty (1995) find that the introduction of national health insurance in Canada increased the productivity and wages of covered workers.

The microeconomic studies discussed above are not generally interested in asking the question “how much does health contribute to income differences among countries?” However, in principle one can use their results to do just this. Specifically, using these microeconomic estimates of the effects of variation in health input on wages, it is possible to calculate the contribution of variance in single health input to variation in income among countries. For example, using Behrman and Rosenzweig’s estimate, variation in birthweight among countries contributes 1.6% to the log variance of world income per capita.<sup>4</sup>

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<sup>4</sup>Behrman and Rosenzweig actually report a smaller number, but the two figures can be reconciled as

Extending the Behrman and Rosenzweig 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. That

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follows. Define  $y_i$  as the log of GDP per capita in country  $i$ ,  $b_i$  as average birth weight, and  $\hat{y}_i$  as predicted GDP per capita 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  $\beta = .00413$  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 .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 birthweight that affect log wages, then in the equation

$$y_i = \hat{y}_i + \epsilon_i,$$

$\hat{y}$  and  $\epsilon$  are not orthogonal. The variance of  $y$  is affected by the covariance of  $\hat{y}$  and  $\epsilon$ :

$$\text{var}(y) = \text{var}(\hat{y}) + \text{var}(\epsilon) + 2 \text{cov}(\hat{y}, \epsilon)$$

A natural measure of the fraction of the variance in world income accounted for by variance in birth weight assigns to this factor not only its own variance, but half of the covariance with other factors as well. That is  $(\text{var}(\hat{y}) + \text{cov}(\hat{y}, \epsilon)) / \text{var}(y) = \text{cov}(y, \hat{y}) / \text{var}(y)$ . This can be expanded in turn as

$$\frac{\text{cov}(y, \hat{y})}{\text{var}(y)} = \beta \frac{\text{cov}(y, b)}{\text{var}(y)} = \beta \left( \frac{\text{cov}(y, b)}{\text{var}(b)} \right) \frac{\text{var}(b)}{\text{var}(y)}$$

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 0.0158

An alternative way to assess the importance of birth weight is to ask what would happen to the variance of world income if the variance of birth weight (and thus the covariance of birth weight with other factors) went to zero. From the above it is easy to calculate that in this case the variance of world income would fall by 3.1%.

is, workers in rich countries received better nutrition in utero, during childhood, and contemporaneously in comparison to poor country workers, they received better medical care throughout their lives, they are less exposed to debilitating diseases such as malaria, and so on. 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>5</sup>

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. In essence, these papers present regressions of GDP per capita (or GDP growth) on some measure of health outcomes, as well as a standard set of controls. Bloom, Canning, and Sevilla (2004) report the results of 13 such studies, which mostly reach similar quantitative results. 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%. Using a somewhat similar technique, Jamison, Lau, and Wang (forthcoming) estimate that the elasticity of GDP with respect to the adult survival rate (the fraction of 15 year olds who will reach age 65, using the current life table) is 0.5.

Papers in this group suffer from severe problems of endogeneity and/or omitted variable bias. For example, Bloom, Canning, and Sevilla attempt to deal with the endogeneity of health

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<sup>5</sup>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 infectious diseases. Adding together the effects of improving one input at a time would overstate the net effect of improving all three.

(and other inputs into production) by using lagged values of these variables as instruments. The identifying assumption is 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>6</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>7</sup>

In this paper I pursue the same question that is addressed by the aggregate regressions, but using a different methodology. 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 will use the available microeconomic estimates to create an estimate of the importance of health at the macroeconomic level.

### **3. Empirical Framework**

#### **3.1 Production and Wages**

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<sup>6</sup> Mankiw (1995).

<sup>7</sup> 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, Johnson, and Robinson (in preparation) 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 no effect on GDP per capita.

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

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

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. As discussed above,  $v_i$  is not the totality of individuals's health; rather, it is only the aspects of individual health that are relevant for production of output. Under relatively straightforward assumptions (see section 7), steady state output per worker in a country will be proportional to the level of  $v$ .

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

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

The wage earned by worker  $j$  will be a function of his own health and education as well as the national wage of the labor composite. In logs,<sup>8</sup>

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<sup>8</sup> 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.

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

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

### 3.2 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 above, there are a large number of health outcomes 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 underlying health itself. Thus

$$z = z(\mathbf{X})$$

$$I = I(z)$$

$$v = v(z)$$

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 underlying health,  $z$ . As shown above, individual wages are proportional to human capital in the form of health. Thus the expected gap in log wages (holding constant their human capital in the form of schooling) will be

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

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

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

The expected ratio of the log wage gap to the gap in height is

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

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 which 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 5.3 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 variations 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 equations (5) and (6) we have

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

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

And thus

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

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 will be the same as the ratio of the change in those two health outcomes that results from any change in the entire health input vector.

### 3.3 Bias from assuming that latent health is scalar

The assumption that underlying health is a scalar variable is obviously quite strong. In the limit we could imagine that there are as many elements of health as there are health outcome measures (that is, a person could be healthy in the sense of reaching his genetic maximum height; healthy in the sense of being able to work hard; and so on). Different outcome measures such as human capital in the form of health or any particular health indicator could respond differentially to the different aspects of underlying health.

The appendix works through the algebra of how the measures constructed above are biased in the case where 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 change in human capital in the form of health ( $v$ ) and the indicator ( $I$ ) that is induced by variation in health inputs among countries compares to the ratio of changes in these same variables that is 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 which affect human capital in the form of health (for example, micronutrient deficiencies in utero) but not height. In this case, the procedure presented above will understate the role of health, as proxied by the indicator height, in explaining differences in income among countries.

Similarly, using 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 where 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 attempt to deal with 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.

Finally it is worth mentioning that if these assumptions are violated, then the whole concept of the return to a health indicator (or even the return to health more generally) makes no sense. One can talk about how a specific change in health inputs will affect individual wages and

output. But because a given change in a health indicator can be accomplished via different changes in inputs, which will in turn have different effects on human capital in the form of health, it would not make sense to talk about how changes in a health indicator will result in changes in output.

#### **4. Health Indicators**

The framework above shows how observable health outcomes can be used as indicators 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 micro 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 four indicators of health: average height of adult men, body mass index (BMI) for 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.

##### **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 non-health related variation in height, 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 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 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 adult mortality rates suggests that height is indeed a good indicator of health.

Height is the most frequently used health indicator in microeconomic 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 is also relatively good historical data on height.

### **Body Mass Index**

As in the case of height there are good structural estimates linking health as proxied by BMI to wages, but there is not consistent data for a reasonable cross section of countries. There

is also less historical data on BMI than on height. For these reasons, BMI plays a relatively minor role in the analysis below.

### **Adult Survival Rate**

The third measure of health I use is the adult survival rate (ASR): the fraction of fifteen year olds who will survive to age 60, using the current life table.<sup>9</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 1 shows the relation between adult mortality in 1999 and output per worker in 1996. Figure 2 shows the unweighted mean and standard deviation of the ASR over the period 1960-2000 for a sample of 80 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, 2003). 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 1, 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 7.2.

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<sup>9</sup>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 years olds would die before age 60 if, at each age, they experienced the mortality rates of men who are currently that age. Data are from the World Bank.

There is 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.

### **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 14 to 67 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 32 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 49 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>10</sup>

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<sup>10</sup>Because other data are missing, I use only 41 observations on age of menarche in the analysis in Section 7, 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

Figure 3 shows a 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 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.

### **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 0.773; between age of menarche and log of GDP per capita is -0.494; and between ASR and age of menarche is -0.495.

In countries that started developing earliest, there has been a long, gradual improvement in most health indicators, while 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 .179 in the first period and .203 in the second. By contrast, in South Korea, the height of adult males rose by 4.8 centimeters and ASR rose by 188 over the 33 year period 1962-95 (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

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upon request.

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 vs. 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 account for fixed effects when I use panel data on average height.)

## **5. Estimating the Return to Health Characteristics**

As shown above, 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 will reflect genetic heterogeneity in the height that a healthy person will attain. Using an indicator to measure underlying health will be subject to measurement error, which will bias 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 will be well nourished and cared for as children, and they will 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 will bias 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,  $\gamma$ .

### **5.1 Estimating the Return to Health Using Exogenous Variation in Childhood Inputs**

Both of the problems in estimating the return to health discussed above can be overcome by using instrumental variables. What is needed is a variable which affects wages only through health and which is uncorrelated with the unobserved determinants of wages. One such variable that has been used in the literature is inputs into health in childhood that are not related to family income. These inputs will be reflected in health indicators, but will 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 1 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>11 12</sup> The regressions in Table 1 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.

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<sup>11</sup> 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 U.S. 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.

<sup>12</sup> Schultz (2001) reports similar IV regressions for Côte d'Ivoire and Ghana in which *both* height and 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 (.019); BMI: .159 (.053); Ghana, height: .057 (.017); BMI: .079 (.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.

Thus the extent to which good health allows a person to work more hours, as well as to do better work during the hours employed, is not accounted for. For both of these reasons, the estimates may understate the effect of health on income.

## **5.2 Estimating the Return to Health Using Variation in Birth Weight Among Twins**

My second method for estimating the return to health uses results from Behrman and Rosenzweig's (2004) analysis of a large data set of female monozygotic twins in the United States. Monozygotic twins are genetically identical, and also share all aspects of the family environment that might influence health or other aspects of human capital. 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. In Behrman and Rosenzweig's sample, the average absolute value of the gap in weight is 10.5 ounces (compared to a mean birth weight of 90.2 oz.). These differences in birthweight can be treated as a quasi-experimental source of variation in health.

Behrman and Rosenzweig's 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 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 .657 years of schooling (standard error .211), 3.76 (0.43) centimeters of adult height, and .190 (.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 two stage least squares estimate of the effect of health as proxied by height on wages. The estimate is 0.051, or slightly more than 5.1% 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 5.1, where education is held constant. To construct a comparable estimate, I start by using the Behrman and Rosenzweig results to produce an 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 (see Card, 1999). This implies that each centimeter of height raises log wages by .018 through the channel of higher 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 .033 holding education constant. This number has the same interpretation as the measures of the return to height derived in the previous section.

The estimated return to health as proxied by height derived by this method differs by a factor of more than two from the values estimated in section 5.1. There are several possible explanations for the difference in the estimates. The Behrman-Rosenzweig estimates are for women in a wealthy country, while those reported in section 5.1 are for men in developing countries. If nutrition primarily affects physical capabilities, and if these capabilities are less important for women, in wealthy countries, or both, then one would expect the Behrman-Rosenzweig estimates to be smaller.

### **5.3 Calibrating the Return to Health Using Historical Data**

My final technique for deriving the return to health uses long-term historical data from the United Kingdom and relies on calibration rather than estimation. In a series of papers, Robert Fogel (see 1997 for a summary) analyzes caloric intake and measures of calorie demand in the UK 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% 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% 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% of the mortality decline in England between 1775 and 1875, and half of the decline between 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 will overstate 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 crucial.

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<sup>13</sup>More specifically, Fogel finds that the number of calories *available* for work increased by 56% 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.

(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. The data on health indicators that I use are as follows. Over the period 1775-1995, average height in the UK 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). Floud (1998, table 6) reports that mean BMI for British men aged 26-30 rose from 20.7 for the cohort born 1800-1819 to 24.9 for the cohort born 1960-79. 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 is capturing the vast majority of the health transition that took place during the time period Fogel considered.

In section 3.1, 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

$$\ln(v_{t+1}) - \ln(v_t) = \ln(1.95) = .668.$$

In the case of height, dividing the change in  $\ln(v)$  by the change in height yields a value of  $\rho_{\text{height}} = .668 / 9.1 = .073$ ; in other words, the log of labor productivity rises by .073 for every centimeter increase in height. Using the same technique yields estimates that log wage rises by .159 per unit

of BMI and by .281 per year reduction in age of menarche. These estimates are all quite close to the values derived in section 5.1.

#### **5.4 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 five estimates for the return to height: three from variation in childhood inputs (.080, .094, and .078), one from twins (.033), and one from historical calibration (.073). There similarly two estimates of the return to age of menarche: one from variation in childhood inputs (.281) and one from historical calibration (.261). In the case of BMI, there are also two available estimates, one from variation in childhood inputs (.180) and one from historical calibration (.159). In what follows I use as benchmark values the simple average of estimated returns for each characteristic, that is, .072 for height and .271 for age of menarche (I do not use the BMI estimates for lack of suitable cross-country data). Then in section 7.1 of the paper, I discuss the sensitivity of my results to variations in the assumed return to health.

### **6. Variation in Productivity Due to Health Among Counties**

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 4, to the aspect of health relevant for labor productivity, I use the coefficients derived in section 5. 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 (-.271 per year) implies that, in my data set a one standard deviation decline in the age of menarche results in a 24.5% increase in wages (and thus

steady state GDP per worker). The gap between the earliest and latest age of menarche countries in my sample (3.7 years) translates into a health gap of a factor of 2.73.

As mentioned above, analysis of cross-country data on health as proxied by height and adult survival faces two problems. ASR data is 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 is 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 3, I know the return to height,  $\frac{\gamma_v}{\gamma_{height}}$ , but would like to know the return to ASR, that is  $\frac{\gamma_v}{\gamma_{ASR}}$ . To convert the former to the latter, I need only multiply by the ratio  $\frac{\gamma_{height}}{\gamma_{ASR}}$ . This latter 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,

$$(13) \quad height_{i,t} = constant_i + \gamma_{height} z_{i,t} + \epsilon_{i,t}$$

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

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.

These two equations can be rearranged as follows:

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

Thus the ratio of coefficients  $\frac{\gamma_{\mathit{height}}}{\gamma_{\mathit{ASR}}}$  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 10 countries and data covering up to 180 years per country, for a total of 93 observations. The data are shown in Figure 4.<sup>14</sup>

Table 2 presents the results of this regression (in column 2), as well as results from alternative specifications. The coefficient of 26.4 in column 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 2.64 centimeters. Excluding country fixed effects (column 1) reduces the coefficient only slightly, while allowing for a linear time trend reduces the coefficient by 40% of its initial value, although it remains extremely significant. The final column includes an interaction of ASR with year (normalized to be zero in 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 itself is larger in the case when the interaction is included than when it is not.<sup>15</sup>

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<sup>14</sup> Data available upon request.

<sup>15</sup> Running the regression in column two backwards, that is, regressing ASR on height and a full set of country dummies, yields a coefficient of .0337 (.0013). Inverting this coefficient yields a conversion factor of 29.7, which is slightly higher than the estimate in column 2.

The estimate of  $\frac{\gamma_{height}}{\gamma_{ASR}} = 26.4$  in column (2), along with the value of  $\rho_{height}=.072$

derived above, yields a value of  $\rho_{ASR} = 1.90$ . This coefficient implies that a difference in the adult survival rate of 0.1 would translate into an increase in labor input per worker, and thus steady state GDP per worker, of 21%.

Before proceeding further, it is useful to compare this derived effect of health on income to existing estimates. Bloom, Canning, and Sevilla (2004) estimate that a one-year change in life expectancy at birth ( $e_0$ ) raises worker productivity (and thus steady state GDP per worker) by 4%. To map between  $e_0$  and ASR, I use the Brass generalized life table. Moving from  $e_0=55$  to  $e_0=60$ , ASR rises from .675 to .739, an increase of .064. Thus using the technique introduced above,

$$\rho_{ASR} = \frac{\gamma_v}{\gamma_{ASR}} = \frac{\gamma_v}{\gamma_{e_0}} \frac{\gamma_{e_0}}{\gamma_{ASR}} = \rho_{e_0} \frac{\Delta e_0}{\Delta ASR} = .04 \frac{5}{0.064} = 3.125$$

This estimate is 64 percent larger than the value of  $\rho_{ASR}$  that I derived, implying that a difference of 0.1 in the adult survival rate would translate into an increase in labor input per worker of 37%.<sup>16</sup> Figure 5 shows their and my estimates of the structural effect of health as proxied by ASR on GDP per worker, superimposed on the raw data on ASR and GDP.

## 7. Contribution of Health to Income Differences Among Countries

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<sup>16</sup> Bloom and Canning (2005) use the adult survival rate, rather than life expectancy, as a right hand side variable in cross-country panel regressions. They estimate a value of  $\rho_{ASR}$  of 2.8.

I now turn to examine the contributions 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:

$$Y_i = K_i^\alpha (A_i h_i v_i L_i)^{1-\alpha}.$$

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 assess the role of health, productivity, and other factors in accounting for income variation among countries, I start by rearranging the production function as follows:

$$(16) \quad \frac{Y_i}{L_i} = \left( \frac{K_i}{L_i} \right)^\alpha (A_i h_i v_i)^{1-\alpha} = \left( \frac{K_i}{Y_i} \right)^{\frac{\alpha}{1-\alpha}} A_i h_i v_i$$

The first transformation simply adjusts for labor force. The second adjusts for endogenous variations in the capital/labor ratio that result from differences in productivity, health, or education among countries (as would occur, for example, if countries had fixed investment rates). Starting with the left-most and right-most terms in the above equation, I can take logs and variances to do a decomposition of the sources of variation in  $\ln(Y/L)$  among countries.

I take two approaches to answering the question of how much of the variance of income is explained by variation in health. First, I follow Klenow and Rodriguez-Clare (1997) in simply

dividing the covariance evenly between factors.<sup>17</sup> Thus, for example, the fraction of the variance in output per worker due to health will be given by

$$(17) \quad \frac{\text{Var}(\ln(v)) + \text{Cov}\left(\ln(v), \frac{\alpha}{1-\alpha} \ln\left(\frac{K}{Y}\right)\right) + \text{Cov}(\ln(v), \ln(h)) + \text{Cov}(\ln(v), \ln(A))}{\text{Var}\left(\ln\left(\frac{Y}{L}\right)\right)}$$

As an alternative, I ask the question, How much would the variance of income fall if all variation in health were eliminated? The answer is that variance would fall by the variance of health as well as all the covariances of health with other factors, that is,

$$(18) \quad \frac{\text{Var}(\ln(v)) + 2 \times \left( \text{Cov}\left(\ln(v), \frac{\alpha}{1-\alpha} \ln\left(\frac{K}{Y}\right)\right) + \text{Cov}(\ln(v), \ln(h)) + \text{Cov}(\ln(v), \ln(A)) \right)}{\text{Var}\left(\ln\left(\frac{Y}{L}\right)\right)}$$

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<sup>17</sup>Klenow and Rodriguez-Clare justify their procedure as follows. Starting with the equation in the text, take logs and look at the covariance of  $\ln(Y/L)$  with each side:

$$\text{Var}(\ln(Y/L)) =$$

$$\text{Cov}(\ln(Y/L), \frac{\alpha}{1-\alpha} \ln(K/Y)) + \text{Cov}(\ln(Y/L), \ln(A)) + \text{Cov}(\ln(Y/L), \ln(h)) + \text{Cov}(\ln(Y/L), \ln(v))$$

Dividing both sides by  $\text{Var}(\ln(Y/L))$ , the four terms on the right hand side can be interpreted as the fractions of the variance of output per worker that are attributable to each factor. So, for example, the fraction of variance due to health would be

$$\frac{\text{Cov}(\ln(Y/L), \ln(v))}{\text{Var}(\ln(Y/L))}$$

Expanding this term by substituting for  $\ln(Y/L)$  gives the same expression that I present in the text.

To implement this procedure, I use the estimates of  $\nu$  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. I use a value of one-third for  $\alpha$  (based on the findings in Gollin, 2002). The choice of  $\alpha$  affects the division of variance between physical capital ( $K/Y$ ) and productivity ( $A$ ), but does not affect the fraction of variance attributed to health

Columns (1) and (2) of Table 3 show a decomposition of the shares of the variation in output per worker attributable to each factor, excluding or including health as measured by age of menarche. Columns (4) and (5) show the variance decomposition of output per worker using the measure of health constructed using data on ASR and the estimate of  $\rho_{ASR}$  derived in section 6. The bottom row of the table shows the implied reduction in the variance of  $\ln(\text{GDP}/\text{worker})$  that would result from eliminating variance in health using the different health measures and the different samples.

Table 3 shows that variation in health does indeed have a large effect on variation in output per worker. Using the menarche method, health accounts for 10.8% of the variation in log GDP per worker, while using the ASR method health accounts for 22.6% of the variation in log GDP per worker. The latter figure is roughly the same as the share accounted for by human capital from education, and larger than the share accounted for by physical capital. Even the former figure implies that health variations are an important source of income variation among countries. In both cases, the implied reductions in income variance from eliminating health differences among countries are more than half again as large: 16.9% in the case of menarche and 36.6% in the case of ASR. While these latter figures may seem excessively large, it is worth remembering that they are variances, and thus have a square term. In the case of the ASR measure, for example, the implied reduction in the standard deviation of log GDP per worker from an elimination of health gaps is 20.4%.

The results in Table 3 also modify the conclusions reached by Hall and Jones (1999) and Klenow and Rodriguez-Clare (1997) regarding the importance of productivity differences in explaining differences in output between countries. Because my procedure does not affect the shares of variation attributable to education and accumulation of physical capital, in comparison to these earlier studies, any of the variance that is explained by health in my procedure would be attributed to productivity if health were not measured. Thus using either health indicator, the importance of productivity is significantly reduced. Productivity is still left as the most important determinant of income differences, but it no longer ranks as being more important than all other factors taken together.

Finally, a puzzling point about the results in Table 3 is that menarche and ASR yield such different estimates of the importance of health. Column (3) of the table shows that the difference between the ASR and menarche estimates is not a result of the different samples used. Even using the menarche sample, the fraction of the variance of output explained by health is more than twice as large using the ASR method as using the menarche method.

## **7.1 Robustness and Sensitivity**

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.

One of the two measures of health's importance that I use, the fraction of variance explained by health, is by construction linear in the estimated return to health. Thus, it is easy to assess the effects of imprecision in estimating the two components of the return to ASR on my

results. For example, if instead of my baseline estimate of the return to height (7.2% per centimeter) I use the estimate derived from the Behrman and Rosenzweig data on twins (3.3% per centimeter), the fraction of the variance in log GDP explained by health will fall from 22.6% to 10.3%. Using the highest estimated return to height in my data (9.4% per centimeter), the fraction of variance in log income explained would be 29.5%. The estimated fraction of variance in log GDP explained by health is also linear in the estimate of  $\frac{\gamma_{height}}{\gamma_{ASR}}$  – that is, the mapping from

changes in ASR into changes in height. In the calculations above, I use the estimate of this ratio excluding a time trend (26.4). Using instead the estimate including a time trend (16.6), along with the baseline estimate of the return to height, the fraction of variance of log GDP accounted for by health falls from 22.6% to 14.2%. Using the lowest combination of estimates (3.3% for the return to height and 16.6 for the mapping from ASR to height), the fraction of log variance of output explained by health is 6.5%.

The other measure of health’s importance that I construct, the reduction in the variance of log income that would result from equating health among countries, is not linear in the return to health. However, because the bulk of this term is due to the covariance of health with other inputs rather than health’s own variance (see equations 17 and 18), in practice this measure also varies roughly linearly with the return to health. Moving from the low to the benchmark estimates of the return to height (3.3% to 7.2% – a factor of 2.18), the reduction in log variance of output that results from an elimination of health gaps among countries rises from 18.9% to 36.6% – a factor of 1.94. Using the lowest combination of estimates of the return to height and the mapping from ASR to height, the reduction in the variance of log output that would result from an elimination of health gaps is 12.3%.

In the case of health as measured by age of menarche, the estimated return to menarche is arguably less likely as 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):

$$\begin{array}{rcccc} \text{Age of Menarche} = & 51.98 & -0.105 & \text{median} & -0.0195 & \text{year} & R^2 = .054 \\ & (25.46) & (0.264) & & (0.0128) & & N = 49 \end{array}$$

Using the residuals from this regression in the accounting exercise above implies that age at menarche explains 10.6% of the variance in the log of GDP per capita, which is hardly different from my original estimate of 10.8%.

## 7.2 The Changing Relationship Between ASR and Income

As mentioned in Section 3.3, my method for finding the return to a health outcome,  $\rho$ , is biased in the case where the ratio of changes in worker productivity to changes in the indicator induced by the experiment used to measure  $\rho$  is different than the ratio of changes in worker productivity to changes in the indicator in cross-country data. In the case of the Adult Survival Rate, there is reason to worry that just such a bias may be present, due to AIDS. As shown in Figure 1, a significant fraction of the variance in ASR in the year 1999 is due to high AIDS mortality in developing countries. Young (2004) argues that, in comparison to other diseases, AIDS has a disproportionately large effect on mortality in comparison 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 the same two measures of health's importance that were examined in Table 3 – the fraction of the variance of log GDP per worker that is attributable to health and the reduction in the variance of log GDP per worker that would result from an elimination of health gaps – 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 80 countries for which data on GDP per worker and Adult Survival were available over the period 1960-2000.<sup>18</sup> I use the benchmark estimate of the return to adult survival of  $\rho = 1.9$  derived above. Figure 6 shows the results. There is indeed a significant rise (5.4 percentage points, or 29%) in the implied effect of health on GDP between 1990 and 2000. If we assume that this entire rise was due to AIDS, and that AIDS had no effect at all on the labor input of workers, then the fraction of income variance explained by health calculated in Table 3 would have to be reduced to 17.4%, which is still quite large. Even if this adjustment is applied to the lowest estimated return to ASR discussed in Section 7.1 (that is, the combination of the lowest estimates of the return to height and the mapping from ASR to height), the implied fraction of the variance in log GDP explained by health is 9.5%, which is hardly insignificant.

Besides addressing the issue of bias induced by AIDS, Figure 6 is interesting in its own right. It shows that in the period prior to 1990, convergence in health among countries significantly reduced the fraction of world income variation attributable to health. Because this reduction in mortality was due to progress against a number of diseases, it is less likely that the bias in measuring the return to health discussed above is present, and thus it is less likely that decline in health's importance as a determinant of GDP variation is a statistical illusion.

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<sup>18</sup>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.

## 8. Conclusion

People in rich countries are on average healthier than people in poor countries. In this paper I have sought to determine the extent to which these gaps in health can help explain the income gaps themselves. Knowing the role that health plays in explaining income differences among countries is important both for policy makers who are considering interventions to improve health in poor countries and for economic researchers seeking a fuller picture of how income gaps arise.

Determining health's role in accounting for income differences among countries is made difficult by the endogeneity of health. In particular, the endogeneity of health makes it almost impossible to use aggregate data to estimate the structural effect of health on income. 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 effect of health on income. In other words, I use microeconomic results to answer the macroeconomic question of how much of income variation among countries is explained by health. My methodology has the advantage of being able to incorporate and compare results from a large number of microeconomic studies that have examined different health interventions and different health indicators. The methodology can also encompass historical data on health and labor productivity.

The effects of health on income that I estimate are large. My baseline estimate, using the Adult Survival Rate for men as a measure of health, is that health explains 22.6% of the variance of log GDP per worker, and that eliminating health gaps among countries would reduce the variance of log GDP per worker by 36.6%. A parallel set of estimates, using the age of menarche as a health indicator, are slightly less than half as large. The conclusion that health is an important determinant of income variation is robust to using a variety of different microeconomic and historically calibrated 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.

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. As discussed in the introduction, it is conceptually difficult, if not impossible, to answer the question “how much of the variation in income among countries is explained by health?” in a framework that takes account of these indirect effects. On the other hand, the question “how much would the variance of income fall if health gaps were eliminated?” raises no such conceptual problem. Nor is there any doubt that accounting for health's indirect effects would yield a larger answer than the one I have presented here. How much larger remains a question for future work.

## Appendix: Allowing Latent Health to be Two Dimensional

As a way of thinking about the biases resulting from the assumption that latent health is uni-dimensional, 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

$$(A.1) \quad \ln(v_2) - \ln(v_1) = \gamma_v(z(\mathbf{X}_1) - z(\mathbf{X}_2))$$

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

$$(A.2) \quad I_2 - I_1 = \gamma_I^z(z(\mathbf{X}_1) - z(\mathbf{X}_2)) + \gamma_I^\zeta(\zeta(\mathbf{X}_1) - \zeta(\mathbf{X}_2))$$

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

$$(A.3) \quad \hat{\rho}_I = \frac{\frac{dw}{dx}}{\frac{dI}{dx}} = \frac{\frac{dz}{dx} \gamma_v}{\frac{dz}{dx} \gamma_I^z + \frac{d\zeta}{dx} \gamma_I^\zeta}$$

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

$$(A.4) \quad \hat{\rho}_I (I_2 - I_1) = \frac{\frac{dz}{dx} \gamma_v}{\frac{dz}{dx} \gamma_I^z + \frac{d\zeta}{dx} \gamma_I^\zeta} \left[ \gamma_I^z (z(X_1) - z(X_2)) + \gamma_I^\zeta (\zeta(X_1) - \zeta(X_2)) \right]$$

which can be rearranged to give

$$(A.5) \quad \hat{\rho}_I (I_2 - I_1) = \left[ \gamma_v (z(X_1) - z(X_2)) \right] \frac{\left( 1 + \frac{\gamma_I^\zeta (\zeta(X_1) - \zeta(X_2))}{\gamma_I^z (z(X_1) - z(X_2))} \right)}{1 + \frac{\gamma_I^\zeta \frac{d\zeta}{dx}}{\gamma_I^z \frac{dz}{dx}}}$$

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 equation (A.1). 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 3.2 and applied in Section 5.3) where the return to health is calibrated 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 ratio of differences in these two elements of latent health between countries.

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Table 1: Structural Estimates of the Effect of Health Indicators on Wages				
Health Indicator (unit)	Effect on ln(wage)	sample	country and year	Source
height (cm)	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)
BMI (kg/m <sup>2</sup> )	0.180 (0.069)	males 18-65	Brazil, 1996-97	Rivera and Currais (forthcoming)
Age of Menarche (yrs)	-0.261 (0.111)	females 18-54	Mexico, 1995	Knaul (2000)

Table 2: 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			.0292 (.0068)	-.0057 (.0123)
Year * ASR				.0719 (.0216)
Country Fixed Effects?	no	yes	yes	yes
R <sup>2</sup>	.377	.953	.961	.966
<p>Note: Standard errors in parentheses. N=93 for all regressions. Height is measured in cm. Year is normalized to be zero in 2000. In regressions with country fixed effects, the United States is the reference group.</p>				

Table 3: Shares of Variation in Output per Worker Attributable to Each Factor					
Health Indicator:	none	Age of Menarche	ASR	none	ASR
Sample:	Menarche sample (N = 42)			ASR sample (N = 92)	
	(1)	(2)	(3)	(4)	(5)
physical capital, $K/Y$	.211	.211	.211	.185	.185
human capital from education, $h$	.242	.242	.242	.232	.232
health, $v$		.108	.243		.226
productivity, $A$	.547	.439	.305	.583	.357
Implied Reduction in Variance of $\ln(\text{GDP}/\text{Worker})$ from Eliminating Variance in Health		.169	.389		.366

**Figure 1:  
GDP per Worker vs Adult Survival Rate**

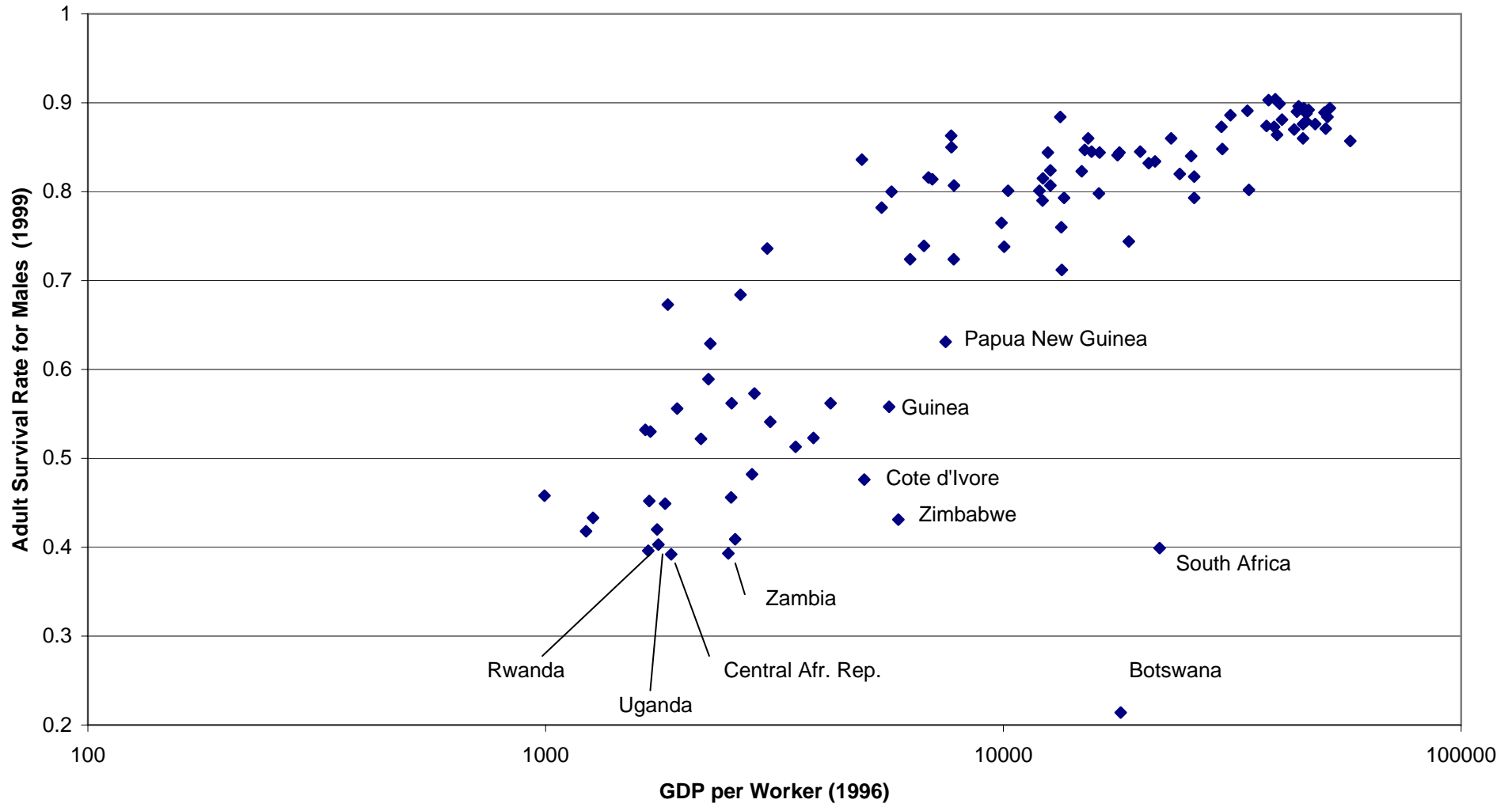
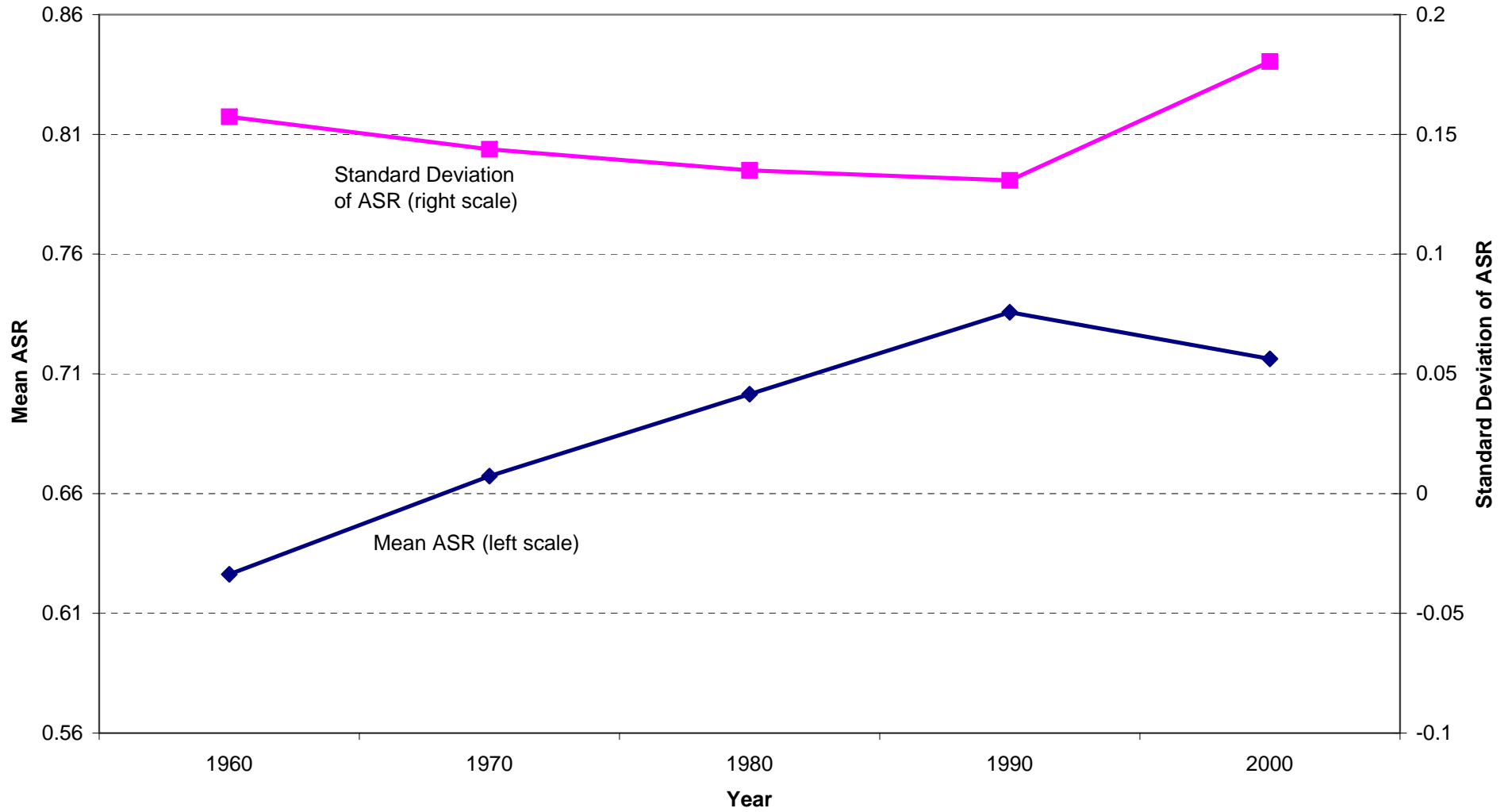


Figure 2: Adult Survival Rate



**Figure 3**  
**Age of Menarche vs GDP per Worker**

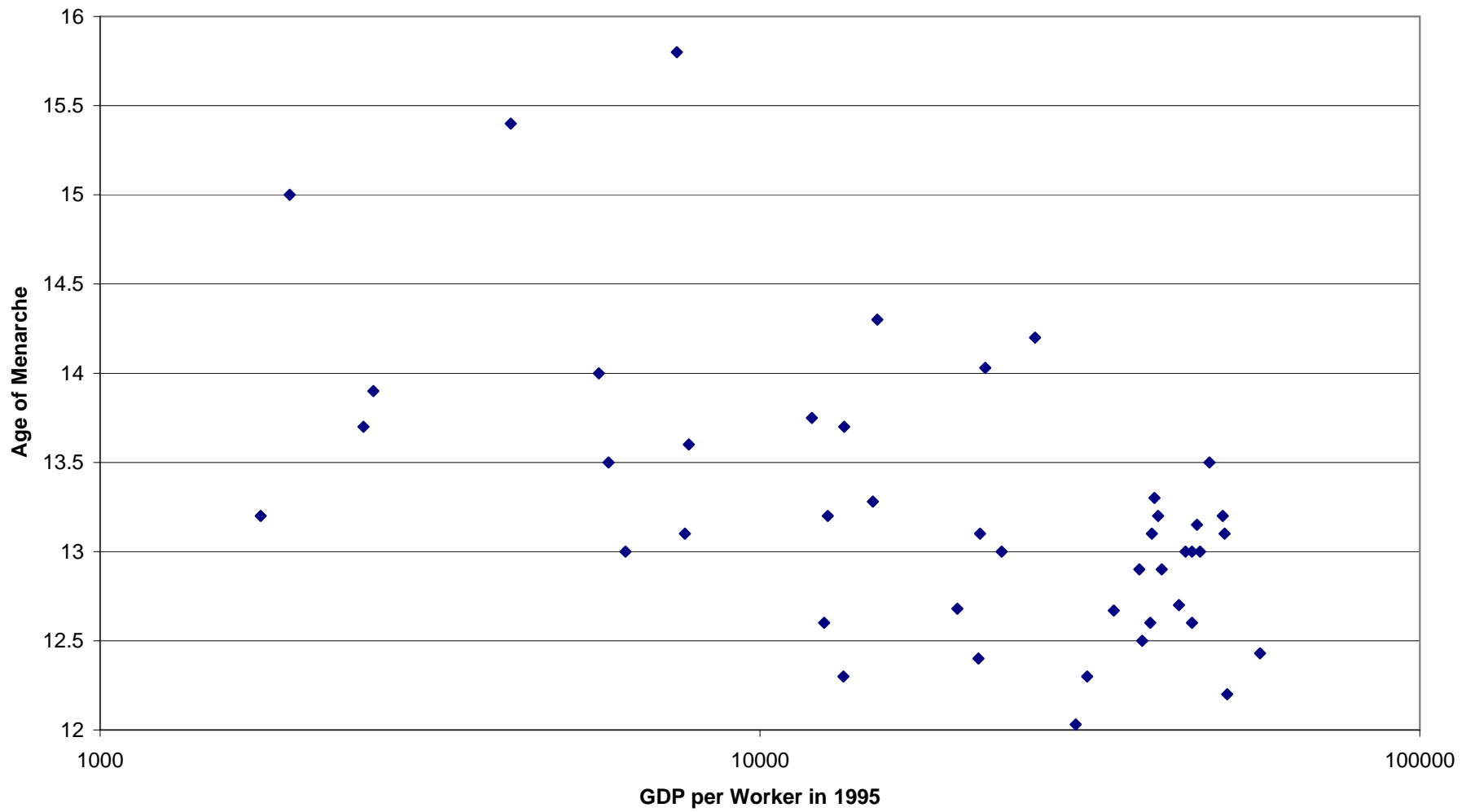
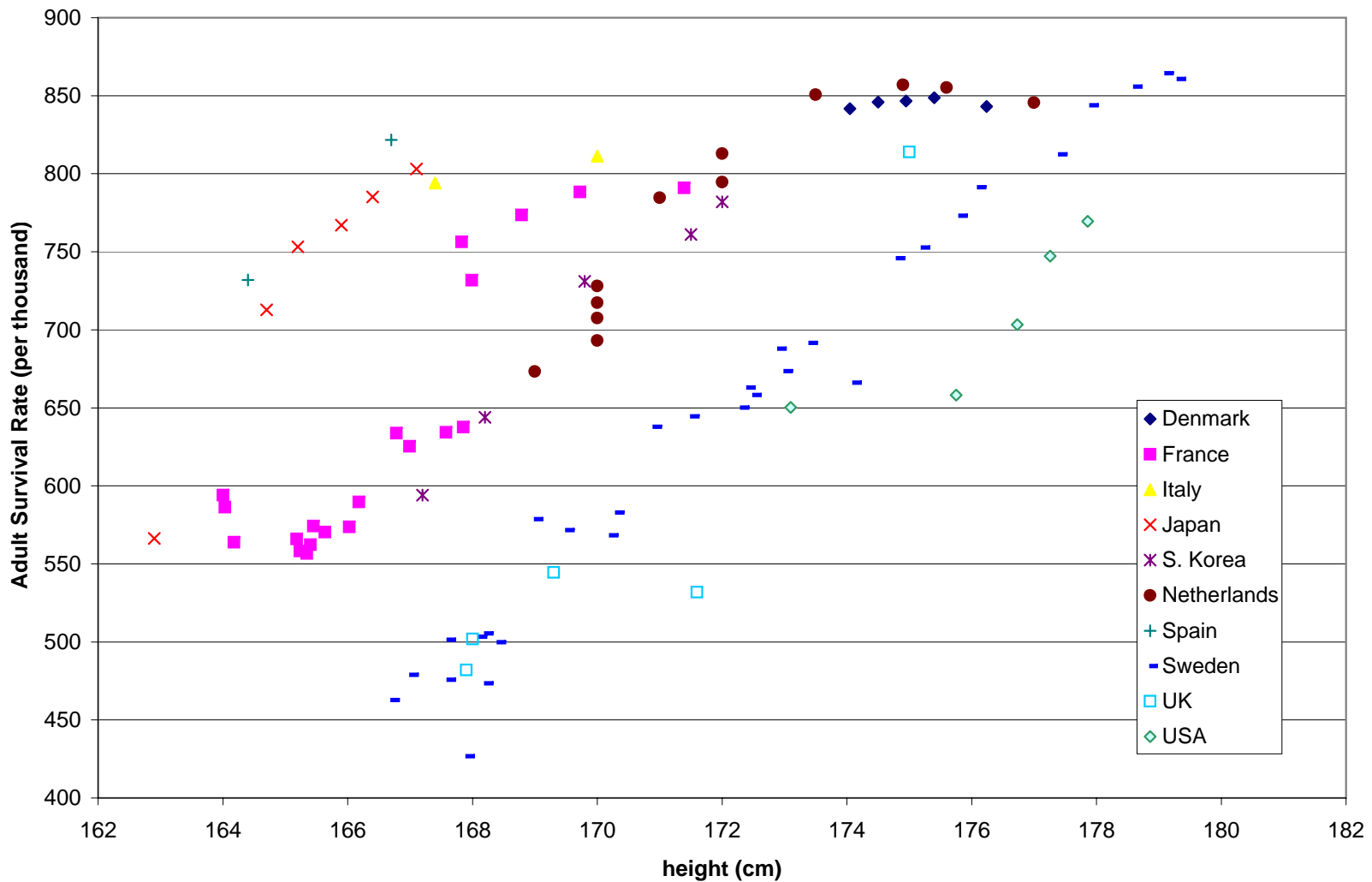
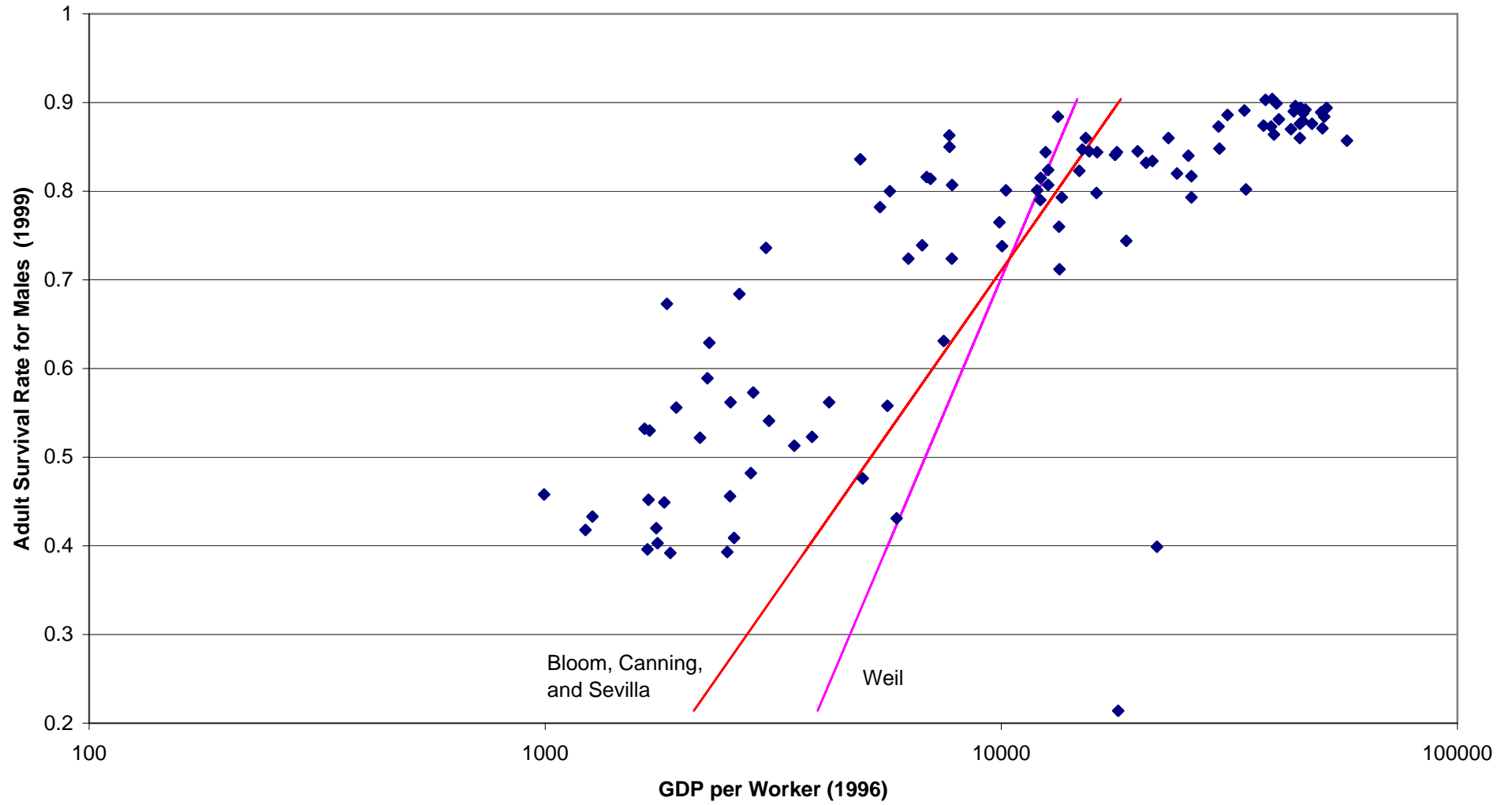


Figure 4: Data on Height and Adult Survival



**Figure 5:**  
**Estimates of the Effect of ASR on GDP**



**Figure 6**  
**The Effect of Health on GDP per Worker**

