

## **How Much of Cross-Country Income Variation is Explained by Health?**

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## **Abstract**

We use development accounting techniques to assess the contribution of health to differences in income per capita among countries. Rather than rely on regressions in aggregate data, we build up estimates of the effect of health starting from microeconomic data. We examine both a particular condition, anemia, and a proxy for general health, the adult survival rate. We find that differences in anemia explain 1.3% of the log variance of income per capita, and that differences in adult survival explain 19% of the log variance of income per capita. The latter figure is almost one-third of the variation in output that is left unexplained by other measures of factor accumulation.

## 1. Introduction

In this paper, we examine quantitatively the effect of health on income per capita as well as the contribution of health to differences in income among countries. Understanding the effect of health on income is important for two reasons. First, if health does have a large effect on income per capita, then this would be an important additional benefit of health improvement (beyond the direct effect on the quality of life) that should be taken into account when tallying up the costs and benefits of health interventions in developing countries. Second, for economists trying to understand differences in income per capita among countries, a proper accounting of the role of health is essential both for telling the complete story of economic growth and for evaluating the role of other factors that affect income. Specifically, accounting for health differences will reduce the size of the unexplained residual variance in income among countries that is currently attributed to productivity.

In this paper we examine both a single condition, anemia, and a measure of overall health, the adult survival rate. Figure 1 shows the relationship between income per capita and our two health measures. Both measures are strongly correlated with income, but using this data to infer the effect of health on economic outcomes presents two problems: first, health is itself endogenous. Thus an interpretation of Figure 1 as showing the structural effect of health on income would not make much sense. Second, a single measure may inappropriately proxy for other aspects of health. A regression of income on anemia, which is only one small aspect of overall ill health, fits almost as well as a regression of income on adult mortality, which is a more comprehensive (although still imperfect) measure.<sup>1</sup> Taking such regressions seriously, one might erroneously conclude that a health intervention that lowered anemia in poor countries to the same level as in rich countries would have the same effect on income as a health intervention that brought *mortality* in poor countries down to the same level as in rich countries.

For both of these reasons, one cannot simply use the aggregate relationship between health and income to determine how much health contributes to differences in income. Both of these problems can be addressed by starting from microeconomic estimates of the effect of health on productivity. This approach is directly modeled on recent analyses of the effect of education on income per capita (Hall and Jones, 1999; Klenow and Rodriguez-Clare, 1997). In

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<sup>1</sup>The regression are (standard errors in parentheses):

$$\ln(\text{output per worker in country } i / \text{output per worker in US}) = -0.0789 - 0.0472 \text{ Non-Pregnant Anemia}$$

(0.1812)      (0.0046)

$$N = 104, R^2 = 0.504$$

$$\ln(\text{output per worker in country } i / \text{output per worker in US}) = -6.893 + 0.00708 \text{ Adult Survival Rate}$$

(0.296)      (0.00040)

$$N = 104, R^2 = 0.753.$$

those studies, microeconomic estimates of the market return to schooling are used as direct measures of its productivity, and this measure of productivity is then used to find the contribution to of differences in average schooling to the variance of income per capita. In this study, we apply the same approach to health. Because we rely on microeconomic estimates of the structural effect of health on income, we are able to take aggregate health measures as given. In particular, our methodology makes no assumption about the fraction of variance in health that is itself an endogenous response to variation in income versus the fraction of variance in health that is due to variation in some underlying health environment, as might be shaped by geography.

## 2. Economic Framework

We use a straightforward extension of the development accounting framework popularized by Hall and Jones (1999) and Klenow and Rodriguez-Clare (1997). Start with a Cobb-Douglas aggregate production function that takes as its arguments capital,  $K$ , and a composite labor input,  $H$

$$(1) \quad Y = K^\alpha (A H)^{1-\alpha},$$

where  $A$  is a country-specific productivity term. The labor composite,  $H$ , is determined by

$$(2) \quad H = h v L,$$

where  $h$  is per-worker human capital in the form of education,  $v$  is per-worker human capital in the form of health, and  $L$  is the number of workers. (As is common in this literature, we assume away heterogeneity in considering the aggregation to national averages, but then turn around and exploit this heterogeneity to derive parameter estimates from microeconomic data.) Substituting (2) into (1), and rewriting so that the capital/output ratio (which is constant in a Solow-type steady state) appears on the right hand side,

$$(3) \quad \frac{Y}{L} = \left( \frac{K}{L} \right)^{\alpha/(1-\alpha)} A h v.$$

In steady state, cross-country variation in health human capital (like variation in education human capital) will translate one-for-one into variation in output per worker. Equation (3) can be used to measure the country specific productivity parameter,  $A$ , as a residual. Measurement of capital per worker is relatively straightforward. To measure human capital from education,  $h$ , and from health,  $v$ , we start with data on the education and health of the labor force. In order to convert these data into measures of human capital that are scaled in units of productivity, we have to use estimates from micro data.

The wage to a unit of the labor composite,  $w$ , is simply its marginal product,

$$(4) \quad w = (1 - \alpha) K^\alpha (A H)^{1-\alpha}.$$

The wage earned by worker  $j$  will be a function of his own health and education, in logs:

$$(5) \quad \ln(w_j) = \ln(w) + \ln(h_j) + \ln(v_j)$$

The studies mentioned above have used “Mincer regressions” of individual wages on years of education to map the relationship between years of schooling and human capital from education,  $h$ . We are similarly interested in the relationship between available measures of health and the level of human capital in the form of health,  $v$ . Unlike the case for education, the measures that we consider are not inputs into the production of health, but rather indicators of health. The next section discusses the indicators that we use and our method for converting them into productivity equivalents.

### **3. Measures of Health and the Effect of Health on Labor Input**

#### **3.1 Anemia**

Anemia is defined as a low level of hemoglobin in the blood, resulting in reduced transportation of oxygen to the tissues in the body. Iron deficiency anemia, the most common form, results from insufficient dietary intake of iron and/or the presence of diseases such as malaria (which attacks red blood cells) and helminth infections such as hookworm (which lead to intestinal bleeding). Anemia can retard growth, reduce cognitive function, and increase morbidity and mortality rates. Anemia also affects a person’s stamina, making him or her tire more easily, thus causing workers to be less productive.

In this study we focus on the direct, short run effect of anemia on worker productivity – thus our estimates of the economic burden of anemia will be an understatement of the total effect of the disease. For example, if impaired cognitive function of an anemic child prevents her from investing in human capital that would be productive later in life, this effect will be missing from our estimates.

Anemia is not an all or nothing thing. Rather, the severity of the condition depends on the concentration of hemoglobin in the blood, measured in grams per deciliter (g/dl). We use the conventional cutoffs of 12.0 g/dl for non-pregnant women and 11.0 g/dl for pregnant women as the thresholds for anemia. To calculate the effect of anemia on productivity for the population as a whole, we need to know not only the fraction of workers who fall below these thresholds, but also how far below them they fall – in other words, for each country we need to know the

*distribution* of hemoglobin concentrations, or at least the part of the distribution which lies below the threshold.

Our starting point in estimating the distribution of hemoglobin in each country is data on the fraction of women who are anemic (Micronutrient Initiative, 2002; World Bank 1993; World Health Organization, 1992). Some of the available data applies to non-pregnant women, and some to pregnant women. We convert the observations for pregnant anemia to non-pregnant anemia using coefficients from a regression run in the subsample of countries for which both types of data are available.<sup>2</sup> Based on the work of Viteri and Torun (1974), we assume that the distribution of hemoglobin is normal. As an estimate of the standard deviation of the distribution, we use the average standard deviation from a sample of 25 Indian states examined by Shastry (2002). The value is 0.989. Given the data on standard deviation and the fraction of women who are anemic, we can then fit a normal curve for the distribution of hemoglobin for the entire population.<sup>3</sup>

To convert our estimates of blood hemoglobin to measures of productivity, we rely on a series of studies that have conducted randomized trials of iron supplementation. The key number we extract from these studies is the elasticity of productivity with respect to blood hemoglobin among anemic workers. The value we use is 1.5 (Levin, 1985; Shastry, 2002). We assume that there is no effect of hemoglobin concentration on productivity among the non-anemic. The productivity of a worker with hemoglobin concentration  $H_i$ , relative to a worker who is not anemic, holding other inputs to productivity such as education constant, is

$$(6) \quad \begin{aligned} v_i / v_{\text{non-anemic}} &= (H_i / 12)^{1.5} && \text{if } H_i < 12 \\ &= 1 && \text{otherwise} \end{aligned}$$

Thus a woman with blood hemoglobin of 10 g/dl would have 76% of the labor input of a non-anemic woman.

The average level of productivity of workers in a country, relative to the case where there was no anemia, can be found by integrating the above equation multiplied by the density of hemoglobin concentrations. The second column of Table 1 shows the prevalence of anemia, and

<sup>2</sup> The regression is (standard errors in parentheses):

$$\text{Non-pregnant Anemia} = 0.176 + 0.869 \text{ Pregnant Anemia}$$

$$(5.049) \quad (0.116)$$

N = 27, R<sup>2</sup> = 0.693.

<sup>3</sup> Anemia lowers productivity in both men and women, and our data on the effect of anemia on productivity comes from studies of both genders. However, the data on prevalence of anemia all comes from women, who are at higher risk of anemia because of blood loss during menstruation. If anemia is not as prevalent in men as in women, our results will overstate the impact of anemia on overall labor input.

the third column of the table shows the value of our productivity measure for selected countries. In the countries where anemia is most severe, gains from its elimination would be significant. In India, the country with the highest rate of anemia, the average level of productivity is 0.896 relative to a non-anemic population. Thus eliminating anemia would raise output per worker by 11%. By contrast, in the United States, eliminating anemia would raise output per worker by one third percent.

Country	Anemia Prevalence, Non-Pregnant Women	Average Productivity Relative to No Anemia	Adult Survival Rate	Average Productivity Relative to U.S.
India	76.7	.896	763.5	.896
Tanzania	69.7	.914	555.7	.618
Haiti	55.8	.942	647.2	.727
Senegal	48.0	.954	421.3	.485
Sri Lanka	45.1	.958	815.7	.983
Algeria	36.7	.969	807.4	.969
Brazil	29.7	.976	806.9	.968
Japan	17.6	.988	891.7	1.127
United States	5.0	.997	825.0	1

Using this data on  $v$  we can do a variance decomposition exercise along the lines of Klenow and Rodriguez-Clare (1997). The results are shown in Table 2. The table shows that differences in health capital due to anemia explain 1.3% of the cross-country variation in the log of output per worker.

Factor	Percentage Contribution to Variation in $\ln(\text{Output}/\text{Worker})$
Physical Capital	20.1
Human Capital from Education	21.6
Human Capital from Health	1.3
Productivity	57.0

An important point to note about this result is that we are *not* using anemia as a measure of health more generally. That is, the result does not say that health variation explains 1.3% of output variation. Rather, we are looking only at the effect of anemia itself.

### 3.2 Adult Survival Rate

Having examined a single condition, we now go to the opposite extreme and examine the effect of health in general. The measure of health we use is the adult survival rate (ASR), which is the probability that a 15 year old will live to age 60, using the current life table. Using data on survival of working age adults is appropriate because we are interested in the productivity of workers. The key assumption in looking at this measure as an indicator of the health of workers is that the fraction of people who die gives a good measure of the health of those who remain alive. One can think of cases where this assumption was problematic: Some conditions may have an impact on mortality that is disproportionate to their effect on the health of living workers. Nonetheless, the ASR is probably the best measure of adult health that is available for a large cross section of countries.

In the case of anemia, we were able to draw on microeconomic studies in which individual output and blood hemoglobin were observed. In the case of adult survival, no comparable data is available, and so we have to use other means to scale data on survival into a measure of productivity. We assume that the relationship between ASR and human capital in the form of health has the same exponential structure found in the literature on education

$$(7) \quad \ln(v) = \text{constant} + \phi \text{ ASR}.$$

Weil (2002) uses a variety of techniques to derive estimates of  $\phi$ , which yield fairly similar results. Here we present a brief outline of one of them. To derive a value for  $\phi$ , we use information on height, which is often used as an indicator of health. 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. Available micro data allows an estimate of the effect of health, as proxied by height, on wages. We then use historical data on changes in mortality and height in a single country to map from the effect of height on wages into the effect of ASR on wages.

We assume that the relation between height and human capital in the form of health has the same form as that for ASR:

$$(8) \quad \ln(v) = \text{constant} + \gamma \text{ Height}$$

Savedoff and Schultz (2000) report various estimates of the coefficient  $\gamma$  derived from individual level wage regressions estimated using instrumental variables. The average coefficient for males is 0.06, or six percent per centimeter of height. This is the value that we use.



Weil (2002) shows that in the available data, adult survival rates are linearly related to adult male height:

$$(9) \quad ASR_t = \text{constant} + \theta \text{ Height}_t$$

We can derive the value of the coefficient  $\theta$  by comparing two points in time,  $s$  and  $t$ ,

$$(10) \quad \theta = \frac{(ASR_t - ASR_s)}{(\text{Height}_t - \text{Height}_s)}$$

We implement this calculation using data for Sweden for the two years 1775 and 1975.<sup>4</sup> The implied value of  $\theta$  is 33.5 – in other words, a one-centimeter increase in average height is associated with a rise in 3.35% in the probability of surviving from age 15 to age 60.

Using our estimates, we can solve for a value for  $\phi = \gamma / \theta = .06 / 33.5 = .00179$ . This says that a rise in the adult survival rate by 100 would raise the log of wages by 0.179. A change in the ASR by 318 (as took place in Sweden over this period) would raise wages by a factor of 1.77.

Using this estimate of  $\phi$ , we can convert cross-country data on adult survival into a measure of human capital from health scaled in units of productivity. Specifically, using equation (7) we can calculate the value of  $\nu$  in each country relative to a benchmark. The last two columns of Table 2 show data on ASR and the calculated values of  $\nu$  relative to the United States. Comparing the richest to the poorest countries in our sample,  $\nu$  differs by roughly a factor of two. In other words, if health in the poorest countries were to instantly increase to the level of health in the richest countries, steady state income per capita in the poor countries would double.

Using the calculated values of  $\nu$  we can do the same variance decomposition exercise that we presented above. Table 3 shows the results. Our finding is that human capital from health is only slightly less important than human capital from education in terms of the fraction of the variance of income that it explains. Accounting for health also reduces by roughly one-third the fraction of variance that is explained by differences in productivity. That accounting for health should reduce this variance is not surprising, since by construction “productivity” was everything that was not previously being accounted for.

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<sup>4</sup> The data are 1775: height 168.1 cm, ASR 539; 1975: height 177.6 cm, ASR 867. Sources: Fogel (1997), Keyfitz and Flieger (1968).

<b>Table 3: Variance Decomposition Using Adult Survival as a Measure of Health</b>	
Factor	Percentage Contribution to Variation in ln(Output/Worker)
Physical Capital	20.1
Human Capital from Education	21.6
Human Capital from Health	19.0
Productivity	39.3

#### **4. Conclusion**

In this paper we have presented a general methodology to account for the direct effect of health on income per capita. We found that variation in a single component of health, anemia, accounts for 1.3% of the variance of log income per capita in a large sample of countries. Variation in the adult survival rate, a proxy for general adult health, accounts for 19.0% of the variance in log income per capita.

Our methodology is obviously applicable to a variety of different diseases and health conditions. In each case, one needs data on the prevalence of the condition and an estimate of the structural effect of the condition on worker productivity. While we are enthusiastic about pursuing such an agenda, it is important to point out some of the pitfalls that await.

First, there is the issue of how different conditions and diseases are related or interact. The assumption implicit in our analysis of anemia was that this single condition had a multiplicative effect on productivity. Such an assumption is reasonable looking at one condition at a time, but in considering two or more conditions, interactions, both positive and negative, may be important. Similarly, when considering more than one condition, there is the danger of double counting. One of the benefits of eliminating malaria, for example, would be to reduce the prevalence of anemia.

A second drawback of our methodology is the difficulty of assessing long-run effects of health improvements. As the case of anemia shows, it is relatively easy to measure the short-run effect of a disease on productivity by examining the outcome of experiments in which the diseases was eliminated. However, such an approach may miss many of the most significant channels through which disease affects productivity. Diseases that affect physical or mental development may affect income with a lag of decades.

A third drawback of our methodology is that by focusing on the direct effects of health on productivity, we may be missing an important part of the story. For example, ill health may limit

human capital accumulation in the form of education. Similarly, high mortality, which we used as a measure of ill health, may also have indirect effects on output by raising fertility and limiting the incentive to invest in physical or human capital.

Finally, waiting in the wings are problems of general equilibrium, which, unlike many of the previous considerations mentioned, may serve to moderate, rather than amplify, the effects estimated here. Consider the example of a health intervention that improves cognitive function. The effect of such an intervention on individual productivity (which is what we learn from examining microeconomic data) may be large in the context of a society where individuals with high cognitive function are rare. The effect of raising cognitive function for all individuals may be much smaller, since in such a case high cognitive function would lose its scarcity value.

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Figure 1  
Mortality, Anemia, and Income per Capita

