

Comments Welcome

## **Endemic Diseases and African Economic Growth: Challenges and Policy Responses**

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## 1. The Scope of the Problem

There is no question that Africa bears a heavy burden of disease. Life expectancy at birth in the year 2006 was 50 years in sub Saharan Africa. In the second least healthy region, South Asia, it was 64 years, while in high income countries it was 79. Over the last five decades, most of the developing world made remarkable strides in catching up to rich-country standards of health. In Africa, progress in health was already slow before the advent of HIV/AIDS, and since that time the continent has become sicker as the rest of the world continued to become healthier.

Table 1 shows the broad outlines of mortality in Africa, using data from the WHO's Global Burden of Disease project. In Africa, infectious and parasitic disease accounted for 43.1% of total deaths. In the next poorest region among the WHO's broad classifications, South Asia, the fraction of deaths due to infectious and parasitic diseases was 17.5%, while in Europe, the figure was 2.3%.<sup>1</sup>

In addition to being the least healthy part of the world, Africa is also the poorest. The question that naturally arises is how poverty and ill health interact. There is abundant evidence that being poor, either for a household or for a country, leads to worse health outcomes.<sup>2</sup> Families that are poorer spend less on food and medical care; countries that are poor devote fewer resources to public health.

In this paper, my concern is with causality that runs in the other direction, that is, with the economic effects of disease.<sup>3</sup> I will ignore completely the determinants of ill health, whether economic or non-economic. I will also not touch on the vital question of what policies that might be used to reduce the burden of disease. Instead, I will focus on the results of disease or its control. Throughout the paper, my emphasis will be on trying to estimate the *quantitative* effects of disease on different aspects of the economy. For economic analysis to be useful for policy making, this sort of quantification is crucial. There are many things that are "good for growth" or "good for welfare." But resources are limited. If economists are to make a contribution to wise policy making, they must

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<sup>1</sup> Estimates of the prevalence of HIV in Africa are particularly contentious. Most existing estimates have extrapolated from data from ante-natal clinics, with the obvious bias that pregnant women are a selected sample of people who have been having unprotected sex. Junh, Kalemli-Ozcan, and Turan (2008) have recently re-estimated prevalence in a number of African countries, using data from the Demographic and Health Surveys which took blood samples from respondents. In comparison to UNAIDS/WHO, the Junh, Kalemli-Ozcan, and Turan estimates average about one-quarter lower. For example, prevalence in Kenya is reduced from 12.0% to 8.7%, and in Zambia from 25.6% to 19.7%.

<sup>2</sup> See, for example, Pritchett and Summers (1996).

<sup>3</sup> I use the term "endemic" loosely in the title of this paper. Technically, malaria is currently endemic in some regions of Africa, while other areas are subject to repeated epidemics. HIV/AIDS may also be viewed as epidemic rather than endemic.

get into the business of saying how much good will result from some course of action, so that benefits of different possibilities can be compared.

## 2. The Economic Effects of Endemic Disease

### 2.1 Direct Effects of Endemic Disease on Utility

Analyses of economic growth usually proceed by examining gross domestic product (GDP), the sum of the value of goods and services produced in a country in a year, evaluated at market prices. But economists are well aware that people care about more than just the goods and services that they consume. As Robert Kennedy said, GDP “does not allow for the health of our children, the quality of their education, or the joy of their play. It does not include the beauty of our poetry or the strength of our marriages; the intelligence of our public debate or the integrity of our public officials.... It measures everything, in short, except that which makes life worthwhile.” For purposes of policy analysis, however, it is not enough to note that there are things other than GDP that we should care about. To make progress, one would ideally like to be able to *compare*, in comparable units, GDP with other things. Economists operationalize this comparison via the concept of “utility,” which is taken to be a function of both GDP and other aspects of life. In the context of endemic disease, these could include including people’s health status, how long they live, and whether their loved ones suffer illness or die.

Suppose that we measure utility as some function  $V$  of income per capita,  $Y$ , and health,  $S$  (where in practice  $S$  would be something like life expectancy at birth). We observe these at two points in time: 1960 and 2000. The traditional measure of economic growth that we observe in data is based only on income at the two points in time:

$$G = \frac{Y_{2000} - Y_{1960}}{Y_{1960}}$$

If health has improved over this period, this measure of income growth understates the true improvement in utility. We can get a better measure of growth by considering the extra income that we would have to give the person so that he would have the same utility he had in the second period, but with the mortality rates observed in the first. Call this extra income  $W(S_{1960}, S_{2000})$ . It is implicitly defined from the equation

$$V(Y_{2000} + W(S_{1960}, S_{2000}), S_{1960}) = V(Y_{2000}, S_{2000})$$

The properly measured growth of income is then the ratio of “full income” (actual income growth plus the imputed increase in income due to better health) would be

$$G = \frac{Y_{2000} + W(S_{2000}, S_{1960}) - Y_{1960}}{Y_{1960}}$$

Implementing this procedure requires knowing how to value health in terms of an income equivalent. Thus far, economists have only tried to measure the utility effect health improvements via the reduction in the probability of an individual losing his or her own life. To do this, they can rely on a widely used measure of how people trade off life risks against money, called the “value of a statistical life.” For example, a person who is indifferent between taking a one in 5,000 risk of dying and spending \$1,000 on avoiding the risk is putting a value \$5 million on his statistical life.

Unfortunately, measurement of the value of statistical lives is done almost exclusively in developed countries. For the United States, the value of a statistical life is estimated to be around six million dollars. Using a fair bit of economic theory, one can convert these measures into utility measures appropriate for poor countries as well, but there is the danger that the theory used for making this translation is subject to error, and it would be much better to have measures that were directly appropriate to poor countries.<sup>4</sup>

Taking the value of health improvements into account Becker, Philipson, and Soares (2005) calculate that in sub Saharan Africa over the period 1960-2000, the income equivalent of the gain in life expectancy (from 41 to 46 years) was \$72 per capita, which was almost as large as the gain in measured GDP per capita of \$103. Of course the four decades in question were a dismal period for both income and health in Africa. Had it not been for the effect of HIV, the gain in utility from life expectancy would have been far larger than the gain in conventionally measured income. The pattern of health having effects on utility that are significant in comparison to changes in conventionally measured income extends beyond Africa. For example, in south Asia, where life expectancy rose from 44 to 63 years and GDP per capita rose from \$892 to \$2,346, the income equivalent value of the rise in life expectancy (\$635) was almost half as large as the actual income gain.

As mentioned above, these calculations look only at the value individuals place on their own lives, ignoring the two other key aspects of health: utility loss from the death of loved ones, and pain and suffering associated with illness. These aspects could be quantitatively as important as loss of one’s one life, suggesting that the benefits of health gains measured here are significantly understated.

The bottom line from this analysis is that quantitatively, changes in health in developing countries have effects on utility that are quite significant. It is possible for a country to achieve respectable “growth,” measured in utility terms, even when income does not change. Similarly, it is possible for a decay in health status to undo the positive effect on utility of income growth. In theory, policy decisions regarding the allocation of scarce resources between different uses (for example, more health clinics vs. improvements to infrastructure) could be based on the goal of achieving a utility-maximizing combination of increases in health and income. That is, the “bang for the

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<sup>4</sup> The value of a statistical life is certainly lower in poor countries than in rich ones. This is because in poor countries income is lower, and thus willingness or ability of individuals to pay to prevent risks to life is lower. It does not mean that lives are any less valuable in an objective sense.

buck” of a given piece of growth- or health-improving expenditure would be weighted by the increment in utility that each would achieve. Explicitly recognizing the tradeoff between income and health as targets for optimization may strike many readers as heartless. We are used to saying that life is priceless, and that economic considerations should take second place to saving lives. In practice, however, governments constantly make tradeoffs between health spending and other priorities. It is quite possible that an analysis along the lines described here would lead to the conclusion that health spending is too low.

Having said all this, in the rest of this paper I turn toward the question of how disease affects conventionally measured GDP. The claim that disease lowers GDP, and that improving health would raise measured GDP, has been a central part of discussions of health policy in Africa. If health improvements “pay for themselves” by raising output by more than they cost, then utility calculations like those just described are unnecessary.

## **2.2 Effects of Endemic Disease on Measured GDP: Theoretical Channels**

Disease affects economic outcomes through a number of different channels. The simplest channel, which I call the *proximate* effect of disease, is that people who suffer from disease are not as good workers as people who are healthy. For example, a person who is lying in bed suffering an acute bout of malaria is unable to supply any productive labor at all. Even a person who is healthy enough to come to work may provide a low level of labor input, either mentally or physically, because of the chronic effects of anemia, HIV, or any number of other conditions. People suffering disease can work fewer hours than those who are healthy, may work at a slower pace, and may be mentally less acute.

In addition to its proximate effect on worker productivity, disease also has a large number of indirect effects of the economy. Perhaps the most important of these are on the human capital accumulation of children living in countries with high levels of disease. There is good evidence that healthier children have lower rates of absenteeism and higher cognitive functioning, and thus are able to acquire more human capital for every year of schooling. Disease also affects the incentives that students face in deciding how many years of schooling to get: where adult mortality is high, the expected return on education is lower, and so children (or their families) will optimally invest less (evidence on this is discussed below). Another channel by which disease affects human capital accumulation is when one or both parents die, leaving a child orphaned.

A second channel through which disease affects economic outcomes is the size, growth rate, and age structure of the population. Except for HIV, the majority of the mortality burden of infectious disease in Africa falls on infants and young children. Thus the short-run effect of disease is to lower the Net Rate of Reproduction and the growth rate of population – and similarly a decrease in the mortality burden of disease will lead to more rapid population growth and a shift in the age structure toward dependent children. However, over time lower disease mortality will lead to reduced fertility, likely

in a more-than-compensating fashion, so that population growth falls and the age structure shifts toward a greater weight for working age adults. Thus the burden of disease may lead to population size or growth being lower in the short run but higher in the long run than it would otherwise be. The degree to which this effect on population size matters for economic outcomes depends on things like the determinants of capital accumulation and the role of fixed factors like land in the production function, which are discussed further below.

A third indirect effect of disease is on the labor supply of people who are not ill. Working age adults may be removed from the labor force to care for children or other family members. Similarly, children who are caring for ill family members will devote less time to schooling.

Endemic disease may affect the seasonal and spatial pattern of economic activity. Gallup and Sachs (2001) give examples of regions in Europe that were largely uninhabited prior to the eradication of malaria, and became productive agricultural regions afterward. Something similar occurred with the eradication of sleeping sickness on the island of Zanzibar in 1997. The need to shift the harvest cycle away from seasons in which malaria is most prevalent may alter the distribution of crops that farmers plant away from those that would otherwise be the most profitable. Improvements in mortality may also lead people to save for retirement, thus raising the levels of investment and physical capital per worker. Physical capital per worker may also rise because the increase in labor input from healthier workers will increase capital's marginal product.

An important aspect of these different effects is the time horizons over which they operate. Some of the proximate effect of disease operates at an immediate time horizon. If malaria is controlled, for example, fewer adults will suffer acute episodes of illness; additionally malaria-induced anemia will fall. Through both of these channels labor input will rise relatively rapidly. On the other hand, some of the effects of disease on worker productivity operate with a long lag. Cerebral malaria contracted in childhood produces lifelong disability. Control of malaria will eliminate the lower productivity due to this condition only with a very long time lag.

The indirect channels by which disease affects the economy also operate with a variety of lags. The burden of caring for ill children reduces labor input by adults in the short run. On the other hand, improvements in human capital accumulation among children due to a reduced disease burden will obviously not have economic effects until the children in question enter the labor force.

Of the channels discussed above, only a few have been well quantified. The effects of health on worker productivity, education, and population size, for example. For other potential effects of health on economic outcomes, there exist no firm estimates – nor do we know that we have a complete list of possible channels. This lack of information leads to a bifurcation of research strategies. One strategy is to try to estimate the total effect of health on economic outcomes directly, without examining the different channels through which this effect operates. The other strategy is to use the best estimates

of the different channels to simulate the overall effect of health on economic outcomes. Below I discuss these two strategies in turn.

### 3. Evidence from Cross Country Studies

#### 3.1 Evidence from Cross-Country Regressions: OLS

Cross-country regressions have become one of the major tools through which economists examine the determinants of economic growth and the relative levels of income across countries.

The theory underlying these regressions relies on two theoretical relationships. The first is a linear or log-linear relationship between a number of exogenous factors and the steady state level of income per capita in a country. Let  $y$  be income per capita (or alternatively income per working age adult). There is a long list of potential determinants of steady state income, including measures of investment rates, political institutions, geographic characteristics, ethnic fractionalization, and so on. Since our interest here is in the effects of disease, we will consider a simple specification in which disease enters explicitly, while a number of other covariates are allowed to enter as well. Thus we have

$$\ln(y_{ss,i}) = \beta_0 + \beta_1 disease_i + \sum_j \beta_j X_{j,i}$$

where *disease* is our measure of interest and the vector of  $X$  variables are additional controls.

The second piece of theory is a relationship between the growth rate of income and the gap between the current level of income in a country and the steady state level. This is called the speed of convergence, and is expressed in the equation<sup>5</sup>

$$\frac{\dot{y}}{y} = \lambda (\ln(y_{ss,i}) - \ln(y_i))$$

Substituting the equation for steady state into the equation for convergence speed, we get

$$\frac{\dot{y}}{y} = \lambda (\beta_0 + \beta_1 disease_i + \sum_j \beta_j X_{j,i} - \ln(y_i))$$

We can regress

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<sup>5</sup> Mankiw, Romer, and Weil (1992) derive this equation by linearizing the Solow model around the steady state level of income, showing that the parameter  $\lambda$  is a function of the shares of physical and human capital in the production function.

$$growth_i = \gamma_0 + \gamma_1 disease_i + \gamma_2 \ln(y_i) + \sum_j \delta_j X_{j,i} + \varepsilon_i$$

where

$$\gamma_1 = \lambda\beta_1 \quad \text{and} \quad \gamma_2 = -\lambda$$

The coefficient  $\gamma_2$  is often called the speed of conditional convergence.

With this framework as background, we can turn to the interpretation of cross country regressions involving health that have appeared in the literature. The most prominent example of such a regression is Gallup and Sachs (2001). The disease measure that they use is the product of the fraction of a country's population living in regions with high malaria risk in 1965 times the fraction of malaria cases that were due to *P. falciparum*. The index is in the range of 0.75-1.00 for most of sub Saharan Africa. The dependent variable is the average rate of income growth over the period 1965-1990. Additional controls include measures of geography, quality of institutions, schooling, and life expectancy (to control for other diseases).

Gallup and Sachs's estimate for the coefficient  $\gamma_1$  is -1.3%. The interpretation is that *ceteris paribus* a country with a malaria index of 1.0 would grow 1.3% per year more slowly than a country with an index of zero – or, alternatively, that wiping out malaria in a country where the index was 1.0 would raise the growth rate of income by 1.3% per year. The conclusion that malaria has this large an effect on growth was famously enshrined in the Abuja declaration of 2000, signed by 57 African heads of state.

An alternative way of presenting the Gallup and Sachs result is to use their estimate of the conditional convergence parameter,  $\gamma_2$ , which is -2.6. Dividing the estimate of  $\gamma_1$  by that of  $\gamma_2$  (and changing the sign) yields an estimate for  $\beta_1$  of 0.5. The interpretation is that going from a malaria index of one to a malaria index of zero would raise the log of steady state income per capita by 0.5, implying that the *level* of steady state income would rise by 65%. Expressed this way, the Gallup and Sachs result is somewhat easier to compare to other measures of the role that disease plays in affecting economic growth.

Following in the footsteps of Gallup and Sachs, a number of researchers have used cross country regressions to examine the role of disease in affecting economic growth. Rather than looking at individual diseases, these papers generally used life expectancy at birth or sometimes the adult survival rate (the probability of living from age 15 to age 60 using the current life table) as a summary measure of the state of population health. Life expectancy is affected not only by the presence of disease but also by nutrition, the quality of medical care, and other factors. However, it has the advantage of being easily observed and standardized its measurement. The dependent variable in these analyses has been the growth of GDP per capita, GDP per working age adult, or the level of productivity, factoring out contributions from physical capital and human capital in the form of education. Bloom, Canning, and Sevilla (2004) summarize

the result of 13 such studies, which reach largely similar results. Their own estimate is that an increase in life expectancy by one year raises steady state output per capita by four percent. (The paper controls for accumulation of physical capital and human capital in form of schooling, so any effect health that run through these channels are not included in the estimated effect).

It is instructive to compare the results in Bloom, Canning, and Sevilla regarding health generally with the findings of Gallup and Sachs regarding malaria. To do this, we need to know the contribution of malaria to low life expectancy. We can do such a calculation using data from Ashraf, Lester, and Weil (2008). They report life expectancy at birth in Zambia, a relatively typical country in sub-Saharan Africa, was 37.0 years in 2001. Eliminating all deaths from malaria would raise life expectancy by 1.6 years. Thus, from the Bloom, Canning, and Sevilla estimate, output per capita would rise by 6.4%. By contrast, using a figure of 0.81 as the malaria index for Zambia (based on the 1994 data in Figure 4 of Gallup and Sachs), the Gallup and Sachs estimate implies that eradicating the disease would raise steady state output per capita by 50%.<sup>6</sup> This exercise shows that even though the Gallup and Sachs and Bloom, Canning, and Sevilla estimates are similar in spirit – in that both show a large effect of health on growth – their quantitative implications are quite different. Some of this difference might be explained by malaria not being a typical disease in terms of its relative effects on life expectancy, on the one hand, and economic outcomes, on the other. However, as will be discussed further below, malaria's most unusual characteristic is its high mortality burden (primarily on children) relative to its relatively low morbidity burden on adults. Thus, if anything, one would expect that analyzing the effects of malaria reduction using the Bloom, Canning, and Sevilla estimate of the effect to life expectancy on income would produce an estimate that was too *large*, not one that was so small relative to that of Gallup and Sachs.

Taking a step back, there is a more general problem with papers like these that simply regress income on disease prevalence or life expectancy, which is that the health measure is likely correlated with the error term in the estimated equation. Either low income itself or the presence factors that lower income (such as bad institutions) may lead to a high presence of disease. In other words, the regression is subject to biases from endogeneity and omitted variables. To give an extreme example, the Panama Canal Zone is a naturally highly malaria-prone region – so much so that the French were turned back in their attempt to build a canal by the disease (along with yellow fever). However, under control of the United States from 1903 to 1979, with the resources and institutions of a developed country, the disease was kept well in check (and has remained so subsequently under Panamanian control). These same resources and institutions raised income directly. If this is the case in other countries as well, then the presence of malaria will be correlated with steady state income, and the estimate of the regression coefficient  $\gamma_1$  will be biased. Although many of the papers in this literature include controls for

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<sup>6</sup> The calculation is  $\exp(0.5*0.81)=1.499$ .

institutions on the right hand side, these proxies are all rather imperfect, and so the worry about omitted variable bias persists.<sup>7</sup>

### 3.2 Evidence from Cross-Country Regressions: Instrumental Variables

The best way to deal with the problems of endogeneity and omitted variables bias that afflict the studies discussed above is to find an instrument for disease, that is, an exogenous variable that affects the level of disease in a country and has no effect on income other than through the channel of disease.

In the case of malaria, a potentially good instrument is the “malaria ecology index” created by Kiszewski *et al.* (2004), which measures the susceptibility of a country’s climate to mosquito breeding as well as the prevalence of mosquito species that feed only on humans. By construction, the malaria ecology index is supposed to be unrelated to any human activity that might affect the prevalence of the disease. Figure 1 shows a scatterplot of the malaria ecology index and the actual incidence of malaria, measured as the fraction of the population that was exposed to risk of malaria in 1994. The figure shows that at intermediate levels of the index there is a good deal of observed variation in malaria risk. For example, Spain and India have roughly similar levels of malaria ecology, but (as of 1994) 66% of India’s population was at risk for the disease vs. none in Spain. Similarly, Brazil ranks much higher in the malaria index than does Pakistan, but has a much lower rate of the disease. Thus for countries in this range of the index, the endogeneity of malaria, or its determination by omitted variables, would be a serious concern. However, the figure also shows that at more extreme ranges of the index, either high or low, there is far less variability in malaria outcomes. The only country with a high level of the malaria ecology index that has wiped out the disease is Mauritius, which has the unusual characteristics of being a small, isolated island where control of the malaria vector was relatively easy.

Sachs (2003) uses the malaria ecology index as an instrument for the level of malaria in regressions looking at a cross section of countries. The dependent variable is the log of GDP per capita in 1995, and controls include measures of the quality of institutions. Unlike the Gallup and Sachs paper, there are no controls for schooling (one of the channels through which malaria might affect income) or life expectancy (to proxy for non-malaria diseases). The striking finding in this paper is that the effect of malaria on income is much *larger* than in Gallup and Sachs. Sachs estimates coefficients on the falciparum malaria index are in the neighborhood of one, implying that eliminating the disease in parts of sub Saharan Africa where the index is near one would raise GDP per capita by a factor of 2.7! In other words, this result is much larger than the already large effect of malaria estimated by Gallup and Sachs. A potential explanation for this finding

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<sup>7</sup> Bloom, Canning, and Sevilla attempt to deal with the endogeneity of health and other inputs into production by using lagged values of these variables as instruments. The identifying assumption required for this strategy to work – that the error term in the equation generating health is serially correlated while the error term in the equation generating income is not – is not explicitly stated or defended.

is that the malaria ecology index may be proxying for other omitted aspects of a tropical climate that negatively affect income.

Two other papers have used Instrumental Variables techniques to assess the effect of disease on economic growth, reaching conclusions that differ significantly from Sachs. The first, Werker, Ahuja, and Wendell (2007) examines the economic impact of HIV/AIDS in sub Saharan Africa. As an instrument for the spread of HIV, they use data on the male circumcision rate, which varies significantly among countries for cultural reasons. The authors show that a low circumcision rate is a good predictor of the extent to which HIV spread in the population, and that it is uncorrelated with other factors likely to have affected growth. In the second stage of their analysis, they show that HIV, as predicted by circumcision, has no effect on the level of GDP per capita, although it is correlated with a slowdown in educational gains and an increase in poverty as measured by malnutrition. On the face of it, it is hard to understand how a disease like HIV, which kills primarily productive adults, could *not* have a large negative impact on growth. However, Young (2005), in a very controversial paper, argues that this is exactly what one would expect in an economy with a very high ratio of workers to both physical capital and land.<sup>8</sup>

The other paper that uses IV to examine the effect of disease on growth is Acemoglu and Johnson (2007). The goal of the authors is see how changes in health, proxied by life expectancy at birth, fed into changes in income. To identify the effect of changes in health, the authors examine the international epidemiological transition that took place roughly 1940-1980. They instrument for mortality reductions in a given country by first looking at the breakdown of causes of death in the country in 1940, then adjusting for progress against the diseases that were particularly important in that country that took place in the rest of the world. They begin by showing that their measure of predicted mortality declines is correlated with actual changes in life expectancy. In the second stage, however, they find that predicted increases in life expectancy actually led to *decreases* in GDP per capita. Their explanation for this surprising finding is that while increases in health may indeed have made workers more productive, their more important effect was on population. Rapid population growth, in the presence of fixed land and slowly adjusting physical capital, led to declining income. Indeed, Acemoglu and Johnson show that, instrumented with predicted mortality, increases in life expectancy led to quite large increases in population.

As in the case of Sachs (2003), the instrumental variables procedure, although it can theoretically deal with the problems of omitted variable and endogeneity bias, still leaves us with many worries about the results regarding Acemoglu and Johnson's conclusions. Ashraf, Lester, and Weil (2008) show that the increases in population that Acemoglu and Johnson find resulting from increases in life expectancy are *too* large, in comparison with the predictions of simple demographic model. This suggests that there

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<sup>8</sup> Young uses a simulation model that has some commonalities to the model of Ashraf, Lester, and Weil (2007) described below. However, a key piece of his model is that HIV will have a *negative* effect of fertility. He presents evidence supporting this assumption. However, Juhn, Kalemli-Ozcan, and Turan (2008) take issue with this finding.

may be a spurious correlation between improvements in life expectancy and other factors that affected growth.

#### **4. Evidence from Individual Channels**

As mentioned above, the cross-country regression methodology has the characteristic that it encompasses all the different channels by which health might affect income into a single regression coefficient. An alternative research strategy is to grasp the nettle, actually trying to measure, and then add together, all of the different channels by which disease affects output. There are several potential pitfalls in following this path. First, the strategy only works to the extent that the researcher can quantify all of the important effects of disease on economic outcomes. As discussed in Section 2 of this paper, there are a large number of theoretical channels by which health affects income, and only some of them have been studied in any detail. Thus one cannot be sure that an important part of the story is missing. A second problem is that what evidence is available about the different channels by which health affects income is not in a form that can easily be aggregated; a good deal of effort has to go into making the units in which disease and its economic effects consistent. A third problem is that there are important interactions among diseases; in the African context the most notable of these is between HIV and tuberculosis. A final hurdle is that one cannot simply add together the different individual channels by which health affects income in order to learn the total effect of health on income: considerations of general equilibrium often have to be dealt with.

Potentially compensating for these disadvantages is the fact that in studying individual channels through which disease exerts economic impacts it is far easier to achieve identification than it is when looking at the overall effects of health, for two reasons. First, individual channels can be identified by looking at data on individual people, rather than on large units like countries. There are far more people than countries! Second, there are far more well-identified experiments (both natural and man-made) that can be used to learn the effect of health on individual outcomes than is the case for data at the country level.

In section 4.1, I discuss some of the data on individual effects of disease on economic outcomes. Section 4.2 then discusses how these can be put together into a simulation model to give an estimate of the aggregate effect of disease.

##### **4.1 Quantifying Some of the Major Effects of Disease on Growth**

###### *Disease and Labor Productivity*

One channel through which disease affects economic outcomes is through the productivity of workers. People who are sick cannot work as effectively as those who are healthy. They are physically weaker and mentally less acute. Sometimes they cannot work at all. The lower labor input due to disease reduces total output.

There are several possible approaches to quantifying the effect of disease on labor input. One approach is to look at the prevalence of diseases among working age adults, on a disease-by-disease basis. Prevalence is defined as the fraction of adults who have a disease at a point in time. It is in turn a function of disease incidence and duration. Annual incidence is the average number of new cases per person per year and duration is how long the condition lasts, on average. Thus if the incidence of disease is 0.5 cases per person per year in a certain population and the duration is two weeks (roughly 0.04 years), then the prevalence will be 0.02 – that is, two percent of people will have the disease at a given time. Table 2 shows data on disease incidence and prevalence in Africa for the five most important of the infectious and parasitic diseases.<sup>9</sup> These measures are shown for the population overall, and then for two specific age groups: 0-4, and 15-44. The latter group contains the bulk of the working-age population. Several points are notable from the table. First, diseases differ significantly in the ages at which prevalence is focused. Diarrhoeal disease, malaria, and childhood diseases are much more prevalent among children than adults; for tuberculosis and HIV, the opposite is true. This suggests that in terms of looking at the direct effect of disease on labor input, the latter two diseases will be far more important. Second, focusing on malaria, which has been the subject of a much of the literature on the economic effects of disease, the prevalence among adults is strikingly low: just one-quarter of one percent of adults are suffering malaria at any point in time. This prevalence figure is in turn the product of a 17.5% incidence and an extremely short duration (less than one week). In the case of malaria, however, adult productivity may be affected by disease episodes that took place decades earlier. Among females aged 15-44, the prevalence of malaria cases (240 per 100,000) is far lower than the prevalence of the neurological sequelae of cerebral malaria (617 per 100,000). Even though the incidence of the latter disease among adults is zero, the duration of childhood cases is for the rest of one's life. This point about childhood diseases potentially affecting adult productivity may be important beyond malaria.

To be used in assessing the economic effect of disease, data on prevalence must be combined with some measure of the degree to which being ill lowers labor input. There are a few direct measures of this effect. For example, Fox *et al.* (2004), studying tea-pickers in Kenya, find that workers earned roughly 17% less in each of the two years before they exited the labor force than a control group. To turn this finding into a measure of the effect of on labor input, one would have to use a number of auxiliary assumptions, including how long individuals are HIV positive before leaving the labor force entirely, and how long they live after leaving the labor force. To give an admittedly crude example, suppose that individuals are HIV positive for ten years before dying, of which one year is spent out of the labor force entirely, two are spent with reduced productivity as measured by Fox *et al.*, and seven are spent with unaffected labor input. The average productivity of an HIV person is thus

$$\frac{7 + 2 \times 0.83}{10} = .866,$$

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<sup>9</sup> Unfortunately, the data are for 1990 – the update of the Global Burden of Disease database is still underway.

or 87% the level of a person who is HIV negative. Combined with an adult prevalence of 2.42%, this implies a reduction in labor input of less than a third of one percent for the typical African country. Even an adult prevalence of one third would imply a reduction in labor input of only 4.5%.

As an alternative to looking at direct productivity measures, Ashraf, Lester, and Weil (2008) explore using “disability weights” created by the Global Burden of Disease project as a measure of how disease affects labor input. Although disability weights are explicitly created as a measure of the utility effect of disease, rather than the productivity effect, they are a useful measure of the relative severity of diseases. On a scale where perfect health is zero and death is one, examples of disability weights are blindness (.600), severe iron deficiency anemia (.093), HIV (.136), AIDS (.505), tuberculosis sero-negative for HIV (.264), malaria episodes (.136), and neurological sequelae of malaria (.473). It is notable that these weights show relatively low weights for both HIV and tuberculosis, the two infectious diseases with the highest prevalence for adults. If the disability weights are reasonable reflections of the severity of disease from the point of view of labor input, it would indicate that the direct economic effects of disease via this channel are modest.

An alternative approach to quantifying the effects of disease on labor productivity is to use data on an aggregate measure of health, rather than looking disease by disease. This has the advantage of being comprehensive, which is important, given the large number of diseases that afflict Africa. On the other hand, a summary measure lumps together diseases which may have different economic effects. For example, using life expectancy as a summary measure lumps together malaria, which has a large effect on childhood mortality but, as shown above, little effect on adult productivity, with tuberculosis, which probably has a greater relative impact on productivity than it has on mortality. Weil (2007) applies such an approach, using the adult survival rate as a summary measure of health. Weil starts with evidence on the effect of health on wages, derived from well-identified microeconomic studies. From this, he extracts a key parameter relating health and labor input. His measure implies that an increase in the adult survival rate of 0.1 would translate into an increase in labor input per worker of 6.7%. Ashraf, Lester, and Weil (2008) show how this coefficient can be applied to assess the effect of health differences among countries using life expectancy data. Consider the effect of going from life expectancy at birth being equal to 40 to life expectancy being 60. The corresponding change in the ASR would be from 0.50 to 0.72. Using Weil’s coefficient, this implies an increase in labor input per adult of 15%.

Ashraf, Lester, and Weil (2008) also explore a second method for using summary data to calculate the loss of labor input due to disease, in this case using data on Years Lost to Disability (YLDs) from the Global Burden of Disease project. YLDs take into account the incidence and duration of disease as well as the disability weights discussed above. Data are available by region and age group. Thus, for example, according to the data, the average male in the 30-44 age group in the AFRO E region loses 0.135 years of disability free life every year, of which 0.040 years are due to infectious and parasitic diseases (half of which is due to HIV). Ashraf, Lester, and Weil estimate the effect on

YLDs, and thus implicitly on labor input, of moving Africa's disease environment in such a way that life expectancy at birth rose from 40 to 60 years. Their estimate using this method is that the increase in labor input would be roughly half of what they calculated using the coefficient from Weil (2007).

### *Disease and Education*

In addition to the direct effect of making adult workers less productive, an important channel by which disease may affect economic outcomes is through the schooling, and thus human capital, of children. This can happen through many different channels. The increase in adult mortality induced by disease reduces the incentives of families to invest in the education of their children, since the likely return on such an investment (higher wages during working years) is reduced (Kalemli-Ozcan, Ryder, and Weil, 2000). Higher adult mortality also raises the number of orphans, who may receive less schooling than children living with their parents.<sup>10</sup> There is also evidence that healthier children are more likely to benefit from schooling due to reduced absenteeism and greater mental alertness while in school. Finally, children may also be kept out of school to provide care for family members who are ill.

In the African context, two of the most important diseases in affecting school attendance are malaria and HIV. Malaria exerts a particularly heavy burden on children; in areas where malaria is endemic, adults develop partial immunity. The effect of malaria on school attendance can be examined by looking at cases in which malaria has been rapidly eradicated or at least greatly reduced. Lucas (2007a) examines malaria eradication in Sri Lanka. Rapid deployment of DDT in the years after World War II, along with pre-existing variation in malaria intensity that resulted from climate factors allow for a differences-in-differences comparison of education in cohorts born before and after eradication. The measure of malaria in her data is the "spleen rate," which is the percentage of school-age children with palpably enlarged spleens. She estimates that reducing the spleen rate from 100 percent to zero would raise primary education by 1.79 years. Although spleen rate is no longer used as a measure of malaria, one can construct a mapping from it to reported incidence. Lucas (2007b) calculates that Zambia's reported malaria incidence rate of 33.1% would imply a spleen rate of 10.3% among children. Her estimates imply that eradicating malaria would raise increase primary schooling by 0.18 years. Bleakley (2007) conducts a similar analysis using data from several countries in South America where malaria was largely eradicated in the period after World War II. He finds that for the same reduction in malaria in Zambia analyzed by Lucas, schooling would rise by 0.25 years.

Fortson (2008) examines how HIV has affected schooling in seven sub-Saharan countries. She measures HIV prevalence in communities using blood-test data in the Demographic and Health Surveys. Her estimate is that a rise adult HIV prevalence from zero to ten percent reduced completed schooling by 0.5 years. Ashraf, Lester, and Weil

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<sup>10</sup> Case and Ardington (2006) find that, even holding household income constant, orphans in South Africa receive one quarter year less schooling.

(2008) use Fortson's estimates to construct a measure of the effect of life expectancy more generally on schooling. Their estimate is that raising life expectancy at birth from 40 to 60 would increase schooling by .386 years.

### *Disease and Population*

The channel running from disease through demography to economic outcomes is complex. Historically, and even today with the exception of HIV, the burden of excess mortality associated with endemic disease has been concentrated on the very young. For this reason, there is a tight link between mortality and fertility rates: in response to the risk of child death, parents have more children. Indeed, it is possible for childhood disease to actually raise the net rate of reproduction because parents more than compensate for risks of child mortality.

The key to understanding the dynamic relationship between health and population is the observation that fertility responds to changes in mortality only with a lag. Thus health improvements that lead to falling child mortality will result in an initial period of more rapid population growth before fertility has fully adjusted, even if in the long run better health actually lowers net fertility. These fluctuations in the growth rate of population may have direct economic effects, as stressed by Acemoglu and Johnson (2007). Further, fluctuation in population growth will be matched by changes in the age structure of the population. As stressed by Bloom and Williamson (1998), a country that rapidly lowers fertility experiences a period of "demographic dividend" in which the ratio of working age population to dependents (children and elderly) is unusually high. Bloom and Williamson claim that accumulation of capital during this window of low dependency can trigger lasting economic growth.

Recently, the interaction of HIV with fertility has received a lot of attention. Young (2005) has argued that, unlike most diseases, HIV should actually *lower* fertility, for several reasons. First, many of the measures that individuals take to avoid HIV, such as using condoms, also reduce the probability of conception. Second, HIV infection lowers a woman's fecundity. Finally, because HIV kills mostly adults, it lowers demand for children, since their parents may not be alive to raise them. Young (2007) finds evidence in African data for a negative effect of HIV prevalence on fertility. However, Juhn, Kalemli-Ozcan, and Turan (2008), using data in which individual HIV status is measured by a blood test, find that while being HIV positive lowers an individual's own fertility rate, the community level of HIV does not lower community fertility.

## **4.2 Simulating the Effects of Disease on Growth**

The section above describes some of the individual channels which link disease and economic growth at the aggregate level. Ashraf, Lester, and Weil (2008) present a discussion of several other channels including changes in the average experience of workers, accumulation of physical capital, and congestion of fixed natural resources such

as land. These different pieces are then put together into a dynamic simulation model, in which one can change health status and watch income per capita evolve (along with such endogenous variables as population size and age structure, the experience and schooling of the labor force, and the stock of physical capital). Readers interested in all the details of these simulations can refer to Ashraf, Lester, and Weil. Before presenting simulation results, I flag a few of the key points that come up in that paper.

One of the most important considerations in looking at the effect of disease on economic outcomes is the time horizon over which one is looking. Some effects of disease, such as the direct effect on labor productivity, are immediate. But other effects work with a time lag. For example, to the extent that the damage disease does to labor productivity takes place in childhood (as in the case of neurological sequelae of malaria), an improvement in health will not start to raise labor productivity for at least a decade. Unfortunately, no good estimates exist regarding the fraction of productivity gains due to disease eradication that accrue to current workers, as compared to those that accrue to new generations that come along after the improvement. This is a key parameter, but Ashraf, Lester, and Weil, in their base case scenario, arbitrarily set it to 50% for lack of any evidence. A second source of lags in the impact of health on the economy runs through the channel of education. Unlike the health effect, this one is easily accounted for mechanically by keeping track of different birth cohorts and “aging” them through the simulation model. The accumulation of physical capital adds yet another aspect to the temporal dynamics.

Population dynamics are particularly important in affecting the economic impact of health improvements. As Acemoglu and Johnson point out, an improvement in health and child survival that reduces population growth in the long run may nonetheless raise it in the short run. The simulations below make a particular assumption about the response of fertility to declining mortality when health improves: we assume that fertility adjusts linearly over a period of 50 years to restore the net rate of reproduction to its pre-shock value.

A third issue that arises in simulating the economic effect of health improvements is the role of fixed factors of production like land in the production function. If health improvements lead to rapid population growth, any economic benefits of more productive workers can be undone by the decline in the land/labor ratio, a la Malthus. Whether this effect is quantitatively important depends on many things, including the structure of economy. An economy open to trade with the rest of the world (and thus able to import resources), will be less subject to a land constraint. Even for an economy with a large subsistence agriculture sector, the degree to which the land constraint binds depends on the availability of inputs such as fertilizer, as well as the degree of substitutability of other inputs (capital and labor) for land. Even 200 years after Malthus wrote, and four decades after worries regarding the “population bomb” reached their apogee, economists know much less about the importance of the land constraint than they should.<sup>11</sup>

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<sup>11</sup> On the interaction of population, land, and agricultural productivity in Africa see Weil (2008).

## *Simulation Results*

Figures 2 and 3 show simulation results for two health improvements. The first is a rise in life expectancy at birth from 40 to 60. This is roughly the size of the post World War II international epidemiological transition discussed by Acemoglu and Johnson, and it is also roughly the size of the gap in health between sub Saharan Africa and middle income countries. Thus the results can be interpreted as answering the question “What would the economic effects of suddenly raising the health status of Angola (life expectancy of 42 years in 2006) to match that of Myanmar (life expectancy of 62).” The simulation uses the estimate of the effect of health on worker productivity derived from Weil (2007), as discussed above. The other parameter values are discussed in Ashraf, Lester, and Weil (2008).

As seen in Figure 2, output per worker follows an odd path, initially rising from the improvement in worker productivity due to better health, then falling due to capital and land dilution from faster population growth, and eventually rising again as population growth slows and the benefits of better health through schooling and the productivity of later-born cohorts phase in. The long-run effect of better health is to raise output per capita by roughly 15 percent relative to the baseline of no health improvement. However, for the first thirty years following a health improvement, income per capita is *lower* than it would have been had health not improved. This is primarily the result of a temporary rise in the number of dependent children relative to working age adults.

Figure 3 shows the results of simulations of the effects of eradicating either malaria or tuberculosis. In this case, the model is set up to match the demographic structure and health situation (including age-specific rates of disease prevalence and mortality) of Zambia in the year 2000. Instead of using disability weights, Ashraf, Lester, and Weil make the strong assumption that individuals suffering from tuberculosis or malaria (including neurological sequelae of cerebral malaria contracted in childhood) simply supply no labor at all. The figure shows that the two health improvements would have similar long run results, raising income per capita by about two percent. The short-run paths are quite different, however. In the case of malaria eradication, income per capita initially dips to almost 1.5 percent below its pre-eradication level, and does not get back to its pre eradication level until some 40 years into the simulation. In the case of tuberculosis, by contrast, income rises immediately. Part of the explanation for this divergence is demographic. The biggest mortality impact of malaria is on very young children; in the case of tuberculosis, it is working age adults. Thus eradicating malaria raises the dependency ratio in the short run while eradicating tuberculosis lowers it. Further, the productivity gains from eradicating tuberculosis are realized immediately, as adult morbidity falls. In the case of malaria, productivity rises only when children who were not affected by the disease enter the labor force, and similarly as the cohort of workers, some of whom were affected by the sequelae of childhood cerebral malaria are replaced by workers not affected.

The results here are indicative of the broader range presented in Ashraf, Lester, and Weil (2008). That paper emphasizes the degree to which findings are sensitive to

choices of parameters regarding the different links between health and growth and well as their phase-in speeds. However, even allowing for a quite broad range of parameter inputs, the basic message from the exercise is not changed. The broad conclusion from these exercises is that, while disease eradication or health improvements in general do have positive economic consequences, they are relatively small and long in coming. Certainly the finding that eradicating malaria would raise GDP per capita by only 2% is in extremely sharp contrast to Gallup and Sachs's results.

## **Conclusion: Priorities for Research, Lessons for Policy**

Research on the effects of disease on economic outcomes, either in Africa or more broadly around the world, is in an unsettled state. In this paper I have reviewed some of the most recent research which has used a variety of approaches and, unfortunately, come to a variety of conclusions. The reader who wishes to do so will be able to find support for a favored conclusion, whether that be that disease has an enormous impact on economic growth or that disease has little or no such impact. For the reader who does not have a preferred conclusion in mind – for example, a policy maker looking at allocate scarce resources amongst many pressing demands – things are more complicated.

### *Research Priorities*

The greatest research priority I see is investigating some of the unmeasured channels through which health affects economic outcomes. As discussed above, there exist reasonable estimates of the direct impact of disease on worker productivity, schooling, and demographics. Also as shown above, putting together estimates of these different channels produces a result that is quite small in comparison to some cross country growth regressions. While it is possible that the regressions are wrong, it is also possible that important pieces are missing in the simulation exercise. To give some examples

- It is clear that disease in general, and particularly diseases like HIV and tuberculosis that strike down working age adults, will create a high level of uncertainty in the lives of families and the operation of firms. For example, a firm that is constantly losing prime age employees to HIV will lose knowledge capital, and will also have to hire excessive staff as a precaution. These effects may significantly lower productivity.
- Anecdotal evidence suggests that malaria as well as other diseases may distort the decisions of farmers regarding what crops are planted away from what would be optimal in the absence of disease.
- In a high disease environment, some industries such as tourism, may never get off the ground and similarly firms may be reluctant to invest in large facilities.

All of the above (and many like them) could in principle be quantified and incorporated into a simulation model of the type described here.

As mentioned above, another priority area for research regarding the economic effects of health improvements is the role of the land constraint (and land congestion more generally) in affecting output. Acemoglu and Johnson explain their surprising result that better health led to negative growth by invoking a Malthusian effect of the falling land/labor ratio. If this story is correct, it should be possible to confirm it using other data. If it can't be confirmed, casts doubt on the Acemoglu and Johnson result.

### *Lessons for Policy*

Given the inconclusive state of research on the economic effects of disease, one might think that there was not useful lesson for practical policy makers to take away from this paper. But in fact there is, for the reason that many policy makers, or at least people who write for the policy making community, seem to have embraced as certain a conclusion that this analysis shows to be highly questionable: specifically the conclusion that eradicating disease or improving health more generally will produce large economic benefits in the conventional sense. In other words, the idea espoused in the Abuja declaration. They summary of evidence presented in this paper does not prove that the idea is wrong, but it certainly removes the presumption that it is right.

This being said, the analysis in section 2.1 of the paper should also serve as a reminder that, in terms of welfare-maximizing policy, whether disease affects conventional economic outcomes is largely irrelevant. The benefits of longer life, of not seeing loved ones dying, and of averting the suffering of disease, when translated into money-equivalent terms, are simply enormous.

Another potentially useful result of this analysis from a policy making point of view is to highlight the interaction of disease with population issues. If health improvements do not lead to increases in living standards, part of the reason may be because health improvements lead to rapid population growth, which undoes the positive productivity effects of better health. If this is the case, it suggests that the economic benefits of better health can be unlocked by pairing health improvements with, for example, better access to and education regarding family planning. Another policy that can unlock the economic benefits of better health is openness to foreign investment, which can mitigate the shortage of capital (and thus jobs) that arises when population grows rapidly and workers become more productive.

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**Table 1: Morality Burden of Disease in WHO AFRO Region, 2004**

Total Deaths	11,248	
<b>Communicable Diseases, Maternal and Perinatal Conditions, and Nutritional Deficiencies</b>	<b>7,682</b>	<b>68.30%</b>
Infectious and Parasitic Disease	4,849	43.11%
<i>HIV/AIDS</i>	1,651	14.68%
<i>Diarrhoeal diseases</i>	1,005	8.93%
<i>Malaria</i>	806	7.17%
<i>Tuberculosis</i>	405	3.60%
<i>Childhood Cluster*</i>	356	3.17%
Respiratory Infection	1,437	12.78%
Maternal Disease	259	2.30%
Perinatal Disease	977	8.69%
Nutritional Deficiencies	159	1.41%
<b>Noncommunicable Disease</b>	<b>2,797</b>	<b>24.87%</b>
<b>Injuries</b>	<b>769</b>	<b>6.84%</b>

\*Pertussis, Poliomyelitis, Diphtheria, Measles, and Tetanus

Source: Global Burden of Disease Estimates, 2008.

[http://www.who.int/healthinfo/global\\_burden\\_disease/en/](http://www.who.int/healthinfo/global_burden_disease/en/)

**Table 2: Disease Incidence and Prevalence, Africa, 1990**

<b>Incidence</b>			
	New Cases Per Person Per Year: Total Population	New Cases Per Person Per Year: 0 to 4 years old	New Cases Per Person Per Year: 15 to 44 years old
HIV	0.0021	0.0012	0.0044
Malaria	0.3649	1.2000	0.1750
Diarrhoeal diseases	1.2800	5.0000	0.3000
Childhood diseases	0.0429	0.1960	0.0002
Tuberculosis*	0.0022	0.0008	0.0033

<b>Prevalence</b>			
	% of People with the Disease: Total Population	% of People with the Disease: 0 to 4 years old	% of People with the Disease: 15 to 44 years old
HIV	1.07%	0.11%	2.42%
Malaria	0.50%	1.64%	0.24%
Diarrhoeal diseases	2.35%	8.97%	0.51%
Childhood diseases	0.49%	1.20%	0.29%
Tuberculosis*	0.43%	0.11%	0.61%

\*HIV negative cases only

Source: Murray, Christopher J.L. and Lopez, Alan D., *Global Health Statistics*, Harvard School of Public Health, 1996.

Figure 1: Malaria Ecology versus Incidence of Malaria

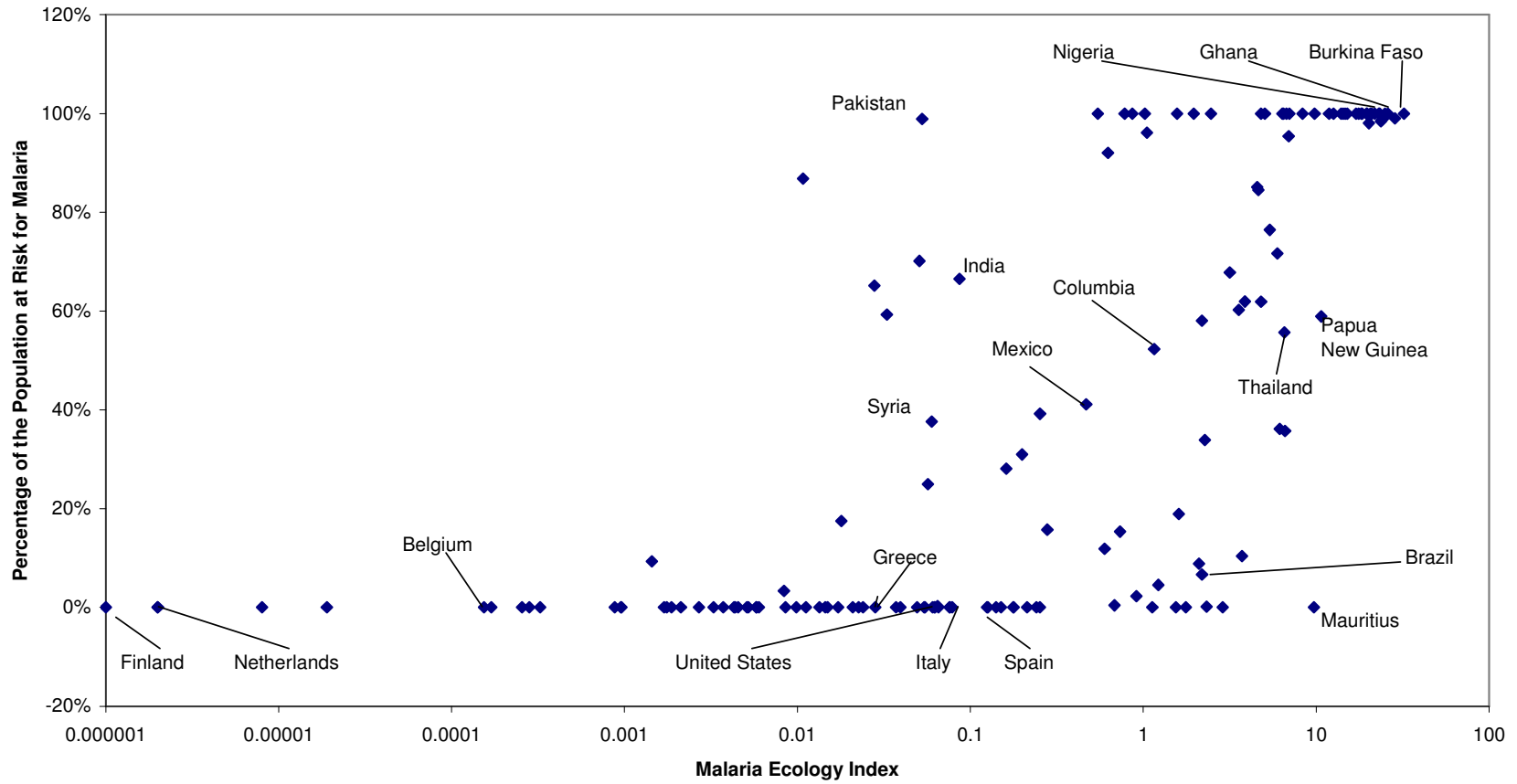


Figure 2: Simulated Effect of a Rise in Life Expectancy from 40 to 60

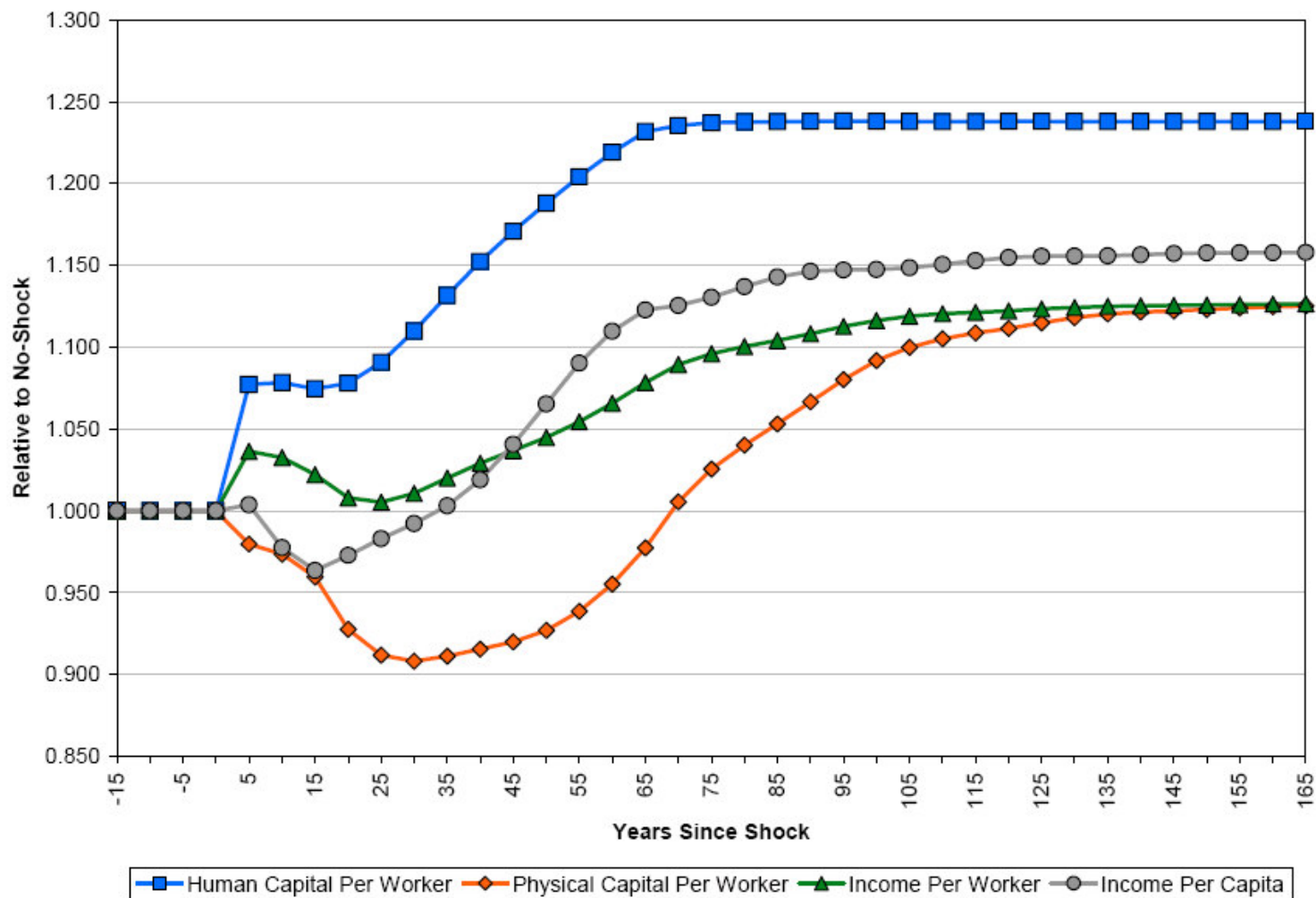


Figure 3: GDP per Capita Under Disease Eradication Scenarios

