Disease and Development: The Role of Human Capital

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Abstract

This paper presents a model of human capital accumulation that allows for feedback effects between the consequences and the likelihood of suffering from particular diseases and the decisions to invest in knowledge, both in the form of schooling and on-the-job training.

I use a calibrated version of the model to estimate the long run impact of eradicating HIV/AIDS and malaria for a number of Sub-Saharan African countries. I find that the effect on output per worker can be substantial

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1 Introduction

There is no question that health improvements have contributed to the increase in the standard of living in many countries and, in particular, in less developed countries. It is also clear that there is a price tag associated with increasing health standards. Even though there is an intrinsic value associated with health, it is important from a policy perspective to have some sense of the economic impact of interventions directed to improving the health status of a population.

The literature that measures the contribution of improved health to economic performance has not produced, so far, definitive results. The empirically based studies that regress output on some measure of health tend to find large economic returns from health improvements (e.g. Sachs (2003)). Since, by its very nature, that approach is subject to the possibility of significant biases it is of interest to understand the predictions of more micro based models. The results from this approach are mixed. Some macro based models that allow for changes in population but model health improvements in a very stylized manner find that the economic impact of improving "health" is either relatively modest (e.g. see Weil (2007) and Ashraf, Lester and Weil (2008)) or directly negative (i.e. Acemoglu and Johnson (2007)). The findings of micro based studies (see the summary in Bleakley (2010)) suggest that, in the case of some diseases (e.g. malaria and hookworm) the economic benefits of eradication are substantial.

One channel through which disease can affect development that has not been thoroughly studied is the impact of health conditions on human capital acquisition. Existing studies consider the quantity of schooling as the only

measure of human capital and were not designed to incorporate the effects of quality and on-the-job training.

In this paper I develop a simple model of human capital accumulation, both in the form of schooling and on-the-job training, that can be used to estimate the impact of changes in health conditions on human capital accumulation decisions and economic performance. The aim is to study a model that is simple enough to be manageable and, at the same time, rich enough so that implications can be checked against micro observations. Moreover, I seek a model sufficiently flexible to capture the impact of diseases that affect the ability to learn or to work without necessarily having a large impact on adult death rates (e.g. malaria), as well as infectious diseases that significantly affect life expectancy (e.g. HIV/AIDS).

I consider three channels through which health influences human capital accumulation. First, an individual who has a given health condition can face a higher probability of permanent disability or death (e.g. if he is infected with AIDS). Second, a given health status can limit an individual's ability to supply effort (e.g. AIDS, malaria and other debilitating diseases). Finally, some diseases (e.g. malaria or intestinal worms) have a small impact on the probability of dying but significantly weaken individuals —and, in particular, children— to the point where the effectiveness of investment in human capital is diminished.

I calibrate the model using evidence from the U.S. to recover parameters of the human capital accumulation equation, and empirical studies of the impact of specific diseases on schooling and income to estimate how health conditions are mapped into the parameters of the model. I then use the

basic setting to study the benefits of eradicating malaria and HIV/AIDS and compare the outcomes with those that correspond to a standard increase in productivity for a small sample of African economies.

Even though I view the exercise as very preliminary there are some interesting results. I find that reducing the incidence of malaria and the rate of transmission of HIV/AIDS to one half of their current values will result, in the long run, in an increase in output per worker between 25% and just over 40%. This decrease in the disease environment has asymmetric effects on healthy and malarious individuals as it induces the latter to acquire proportionally more years of schooling, reducing the income gap between the two groups.

The response of schooling —a measure of quantity of human capital— and the stock of effective human capital per year of schooling —a measure of quality— to changes in HIV/AIDS prevalence and the incidence of malaria are very different, with the former accounting for over 70% of the increase in output per worker. Thus, models that ignore the quality dimension would tend to underestimate the beneficial effects of reducing the incidence of disease.

I also conduct a more traditional experiment: For each country I assume that productivity changes so that output increases by about 17%. Unlike the case of the reduction in the incidence of diseases, the role of schooling and quality of human capital are reversed, with the former accounting fro about 60% of the increase in output. Thus, in this type of models, estimates of the role of schooling drawn from episodes of growth not associated with changes in the disease environment do not provide an accurate guide about

the impact of improving health standards.

2 A Birds Eye View of Selected Health Indicators

Even though there is consensus that HIV/AIDS and malaria are two health conditions that significantly afflict many African countries, there is much less agreement about the fraction of individuals who are affected by those conditions. In Table I, I present some relatively homogeneous data on prevalence and incidence of HIV/AIDS and an index of malaria incidence rate. The first two columns (from Oster (2009)) report UNAIDS estimates of prevalence and incidence of HIV/AIDS approximately over the 1985-2007 period. More recent prevalence data from UNAIDS (column labeled Prev. (III)) suggests that the average estimates do not reflect in many cases the current situation. Oster (2009) also reports prevalence and incidence estimates inferred from mortality data (not shown here) for a subset of the countries in Table I and the values that she finds are smaller.

Irrespective of the preferred estimate, it is safe to conclude that there is a high degree of heterogeneity among African countries in the severity of the HIV/AIDS epidemic and that there are many countries in which a significant fraction of the population is HIV/AIDS positive. If HIV/AIDS has a large impact on productivity, the evidence is consistent with the view that the gains from eradication can be significant.

Table 1				
HIV/AIDS: Prevalence and Incidence				**Malaria**
Country	*Prev. (I)*	*Inc. (I)*	*Prev. (III)*	*Incidence Rate*
Angola	1.00	0.17	2.1	0.33
Burkina Faso	1.87	0.25	1.6	0.50
Cameroon	3.79	0.56	5.1	0.34
Cent. Af. Rep.	4.43	0.66	6.3	0.43
Congo	4.37	0.56	3.9	0.42
Cote d'Ivoire	4.24	0.57	3.9	0.41
Ethiopia	1.67	0.25	2.1	0.15
Ghana	1.35	0.20	1.9	0.40
Kenya	6.57	0.96	7.8	0.18
Malawi	8.79	1.31	11.9	0.45
Mali	0.86	0.14	1.5	0.46
Mozambique	5.94	1.01	12.5	0.39
Namibia	8.12	1.32	15.3	0.27
Nigeria	1.98	0.31	3.1	0.44
Sierra Leone	0.88	0.15	1.7	0.47
South Africa	8.95	1.49	18.1	0.0004
Tanzania	5.92	0.82	6.2	0.38
Zambia	12.63	1.82	15.2	0.39
Zimbabwe	18.95	2.51	15.3	0.07
Sources: Prev (I) and Inc. (I) from Oster (2009), Prev (III) from				
UNAIDS (2008), Malaria data from Korenromp (2005)				

The last column of the table presents estimates of the average number of malaria episodes per person per year. As in the case of HIV/AIDS there is heterogeneity in the impact which is not surprising given the significant influence that geographical factors have in the prevalence of the insects that transmit the malaria bacterium. Even though the incidence rate is a measure of exposure over a period of time it is highly correlated with the fraction of the population in high, medium and low transmission areas. As in the case of HIV/AIDS, malaria is a significant health problem in a large group of countries.

In interpreting the data it is necessary to take into account that all these measures reflect a mix of natural (or exogenous) disease conditions as well as behavioral responses and the impact of policies designed to mitigate or eliminate their effects.

3 A Simple Model of Human Capital Accumulation

Any model that succeeds in explaining the link between a particular health condition and individual decisions about how much human capital to acquire must take into account the effect of each health condition on morbidity — which determines the cost of acquiring human capital and the rate at which it can be utilized— and on the probability of dying or becoming totally disabled. As a first approximation, I will model the mortality effect as a change in the constant probability of dying. This amounts to assuming that lifetimes are exponential which is but a crude approximation to the data.

However, since the purpose of this paper is to describe a simple framework that can be used to match micro and macro evidence, this seems a reasonable first step.

The actual impact of health on economic conditions is probably dependent on the market structure and, it is possible, that markets are "more incomplete" is less developed countries[1]. However, a natural benchmark is the case in which individuals are able to invest according to their potential. In many cases, this provides an upper bound on the impact of diseases on human capital investment[2], and it is this case that this paper discusses.

In this context, consumption and human capital accumulation decisions can be studied separately. Thus, individuals maximize the present discounted value of income and then choose consumption according to that value.

It is useful to separate the optimal human capital accumulation problem in two phases: schooling and working. I identify the schooling period —whose length is determined endogenously— as being characterized by specialization in human capital accumulation. This extreme view, i.e. that children in school do not work, can be shown to be inessential in closely related models. In future work, I will explore this issue in more detail. The working period is defined by the existence of a positive supply of labor. In what follows I work backwards to characterize the solution of the problem: I first study the

[1]See, however, the work of Townsend (XXXX) that shows that family structures come close to replicating the optimal allocation in some low income villages.

[2]For example, if due to incomplete markets a healthy individual cannot acquire any schooling, then a change in his health condition cannot possibly decrease the stock of human capital since he was at a corner. In general, very little can be said ex-ante about the impact of relaxing one condition in a second best situation.

problem faced by an individual who has already joined the labor force and then I discuss the choice of schooling.

3.1 The Post-Schooling Problem

The amount of human capital that an individual wants to supply to the market depends on his productivity as well as his chances of dying or being permanently disabled. I first analyze the optimal labor supply/human capital accumulation problem of an individual who has been infected (say of AIDS) at age τ and whose endowment of labor is $\upsilon_A \in (0,1)$. Let's denote the current level of human capital of such an individual by h. Then, the present discounted value of income, $V_A(h)$ is given by

$$V_A(h) = \max E\{\int_\tau^T e^{-r(a-\tau)}[w(\upsilon_A - n(a))h(a) - x(a)]da\}$$

subject to

$$\dot{h}(a) = z_h(n(a)h(a))^{\gamma_1}x(a)^{\gamma_2} - \delta_h h(a),$$

where the expectation is taken over the age of death, T. The assumption of exponential lifetime implies that the probability that an individual dies before age t is $P[T \leq t] = 1 - e^{-\lambda_A t}$. A simple calculation shows that the the value function is given by

$$V_A(h) = \bar{V}_A^0 + \bar{V}_A^1 h,$$

where

$$\begin{aligned}
\bar{V}_A^0 &= \frac{w}{\rho(\lambda_A)}\left[\frac{(1-\gamma)}{\gamma_1}[\frac{\upsilon_A z_h}{\rho(\lambda_A)+\delta_h}w^{\gamma_2}\gamma_1^{(1-\gamma_2)}\gamma_2^{\gamma_2}]^{\frac{1}{1-\gamma}}\right] \\
&= w w^{\frac{\gamma_2}{1-\gamma}} V_A^0 \\
\bar{V}_A^1 &= \frac{w\upsilon_A}{\rho(\lambda_A)+\delta_h} = wV_A^1,
\end{aligned}$$

which implies that

$$V_A^0 = \frac{1-\gamma}{\rho(\lambda_A)}[\frac{\upsilon_A}{\rho(\lambda_A)+\delta_h} z_h \gamma_1^{\gamma_1}\gamma_2^{\gamma_2}]^{\frac{1}{1-\gamma}}, \tag{1a}$$

$$V_A^1 = \frac{\upsilon_A}{\rho(\lambda_A)+\delta_h} \tag{1b}$$

where $\gamma = \gamma_1+\gamma_2$, $\rho(\lambda) = r+\lambda$, and $\lambda_j dt$ is the probability that an individual in state j dies in a small interval dt.

In this context two parameters summarize the impact of HIV/AIDS: life expectancy (given by $1/\lambda_A$), and a measure and the effective endowment of labor of an infected individual ($\upsilon_A \leq 1$).

Optimal investment implies that the stock of human capital of an a year old individual who was infected at age p and who was previously healthy evolves according to

$$h_{A,H}(a,p) = e^{-\delta_h(a-p)} h_H(\tau) + [1 - e^{-\delta_h(a-p)}] h_A^*, \tag{2}$$

where $h_H(\tau)$ is the stock of human capital of a healthy individual who becomes infected at age τ. The long run stock of human capital of an infected agent, h_A^*, is given by

$$h_A^* = \frac{1}{\delta_h}\left(\frac{\upsilon_A}{\rho(\lambda_A)+\delta_h}\right)^{\frac{\gamma}{1-\gamma}} w^{\frac{\gamma_2}{1-\gamma}} \left[z_h \gamma_1^{\gamma_1}\gamma_2^{\gamma_2}\right]^{\frac{1}{1-\gamma}}. \tag{3}$$

This expression shows the impact of the two parameters that define being infected on human capital accumulation: decreases in life expectancy (i.e. increases in λ_A) and in the effective time endowment, υ_A, reduce the long-run level of human capital.

Even though the evolution of human capital is of interest in itself, it is the effective amount supplied to the market that determines the level of

output, $h^e_{A,H}(a,p)$. Since $h^e_{A,H}(a,p) = (\upsilon_A - n_{A,H}(a,p))h_{A,H}(a,p)$, a simple calculation shows that net human capital in production is

$$h^e_{A,H}(a,p) = \upsilon_A \left[e^{-\delta_h(a-p)} h_H(p) + [(1 - e^{-\delta_h(a-p)}) - \frac{\delta_h \gamma_1}{\rho(\lambda_A) + \delta_h}] h^*_A \right] \quad (4)$$

The problem faced by a healthy individual who has left school is very similar. The only difference is that there are two sources of uncertainty: the time of death —governed by the Poisson parameter λ_H while still in the healthy state— and the infection probability which, over small periods of time, is given by ηdt. It is possible to show that the value function corresponding to the income maximization problem is

$$V_H(h) = \bar{V}^0_H + \bar{V}^1_H h,$$

where,

$$\bar{V}^0_H = \frac{1}{\rho(\lambda_H) + \eta} \left[w^{1+\frac{\gamma_2}{1-\gamma}} V^0_A \frac{\mu^{\frac{\gamma}{1-\gamma}} + \eta(\rho(\lambda_A) + \delta_h)}{\rho(\lambda_A) + \delta_h} \right], \quad (5a)$$

$$\bar{V}^1_H = w V^1_A \mu_H, \quad (5b)$$

and

$$\mu_H = \frac{\rho(\lambda_A) + \delta_h + \eta \upsilon_A}{\upsilon_A(\rho(\lambda_H) + \delta_h + \eta)} > 1. \quad (6)$$

It follows that

$$\bar{V}^0_H = w^{1+\frac{\gamma_2}{1-\gamma}} V^0_H,$$

where

$$V^0_H = \left[\frac{\mu_H^{\frac{1}{1-\gamma}} \rho(\lambda_A) + \eta}{\rho(\lambda_H) + \delta_h} \right] V^0_A,$$

$$V^1_H = V^1_A \mu_H$$

Let $h_H(6+s)$ be the stock of human capital that a healthy individual has at the end of his schooling period (i.e. when he is $6+s$ years old). Then, the stock of human capital at age a is

$$h_H(a) = e^{-\delta_h(a-6-s)}h_H(6+s) + [1 - e^{-\delta_h(a-6-s)}]h_H^*, \tag{7}$$

where

$$h_H^* = h_A^* \mu_H^{\frac{\gamma}{1-\gamma}}, \tag{8}$$

and the supply of effective human capital is given by

$$h_H^e(a) = e^{-\delta_h(a-6-s)}h_H(6+s) + \left[1 - e^{-\delta_h(a-6-s)} - \mu_H \frac{\upsilon_A \delta_h \gamma_1}{\rho(\lambda_A) + \delta_h}\right] h_H^*. \tag{9}$$

Changes in the parameters affecting the spread of HIV/AIDS have a significant impact on the human capital of healthy individuals. One way of summarizing their effect is through the elasticity of the long run level of human capital, h_H^* with respect to the disease parameters. Simple algebra shows that the elasticity with respect to the death rate associated with HIV/AIDS is given by:

$$\frac{\partial h_H^*}{\partial \lambda_A}\frac{\lambda_A}{h_H^*} = -\frac{\gamma}{1-\gamma}\frac{\upsilon_A}{\eta\upsilon_A + \rho(\lambda_A) + \delta_h}\frac{\lambda_A}{\rho(\lambda_A) + \delta_h}.$$

The actual values depend on our calibration but, for the subset of countries that we look at, the first term which is approximately equal to 13 dominates and it yields a large response of human capital to changes in mortality.

The effect of changes in the transmission rate are, given our typical parameterization, are smaller. The relevant elasticity is

$$\frac{\partial h_H^*}{\partial \eta}\frac{\eta}{h_H^*} = -\frac{\gamma}{1-\gamma}\frac{\rho(\lambda_A) + \delta_h}{\eta\upsilon_A + \rho(\lambda_A) + \delta_h}\frac{\eta}{\rho(\lambda_H) + \delta_h + \eta}\left[1 - \frac{\upsilon_A(\rho(\lambda_H) + \delta_h)}{\rho(\lambda_A) + \delta_h}\right].$$

In both cases, improvements in the disease environment induce healthy individuals to increase their investment in human capital.[3]

Since infected individuals could also come from the population of malarious agents, their human capital stocks satisfy the obvious analogues of the expressions corresponding to healthy individuals. In particular, effective human capital at age a given that infection occurred at age p is

$$h^e_{A,M}(a,p) = \upsilon_A \left[e^{-\delta_h(a-p)} h_M(p) + [(1 - e^{-\delta_h(a-p)}) - \frac{\delta_h \gamma_1}{\rho(\lambda_A) + \delta_h}] h^*_A \right],$$

while the human capital of an HIV/AIDS free but malarious individual is

$$h^e_M(a) = \upsilon_M e^{-\delta_h(a-6-s)} h_M(6+s) + \upsilon_M \left[1 - e^{-\delta_h(a-6-s)} - \mu_M \frac{\upsilon_A \delta_h \gamma_1}{\rho(\lambda_A) + \delta_h} \right] h^*_M,$$

where

$$\mu_M = \frac{\upsilon_M(\rho(\lambda_A) + \delta_h) + \eta \upsilon_A}{\upsilon_A(\rho(\lambda_M) + \delta_h + \eta)} > 1,$$

as $\upsilon_M \geq \upsilon_A$, and

$$h^*_M = \frac{1}{\delta_h} \left(\frac{\upsilon_M}{\rho(\lambda_M) + \delta_h} \right)^{\frac{\gamma}{1-\gamma}} w^{\frac{\gamma_2}{1-\gamma}} [z_h \gamma_1^{\gamma_1} \gamma_2^{\gamma_2}]^{\frac{1}{1-\gamma}}.$$

For future reference we note that the relevant elements of the value function for a malarious individual are

$$V^1_M = \mu_M V^1_A,$$

and

$$V^0_M = \left[\frac{\mu_M^{\frac{1}{1-\gamma}} \rho(\lambda_A) + \eta}{\rho(\lambda_M) + \delta_h} \right] V^0_A$$

[3] See Jayachandran and Lleras-Muney (2009) for estimates of the effect of changes in life expectancy on investments in human capital.

3.2 The Schooling Decision

In this section I study the determinants of years of schooling as well as the quantity capital at the end of the schooling period. I consider the case in which, at the margin, individuals have to spend private resources to acquire schooling, and they can instantaneously adjust the level of market resources used in the production of schooling. I assume that the law of motion of human capital during the schooling period is

$$\dot{h}(a) = (1-\zeta_j) z_s (n(a)h(a))^{\gamma_1} x_s(a)^{\gamma_2},$$

where $1-\zeta_j$ is an indicator of the decrease in a parameter that measures ability to learn associated with health condition $j \in \{H, M, A\}$. As a matter of convention I assume that $\zeta_H = 0$.

I also consider the effect of early childhood human capital. To be precise I assume that the stock of human capital at age 6, h_E, is a function of the resources used in its production. The relationship is $h_E = h_B x_E^{\zeta}$.

The problem faced by an individual with health status j is

$$\max\{-x_E - \int_6^{6+s} e^{-\rho(\lambda_j)a} x(a) + e^{-\rho(\lambda_j)(6+s)}[w(V_j^0 w^{\frac{\gamma_2}{1-\gamma}} + V_j^1 h(6+s))]\}, \quad (10)$$

subject to

$$h_E = h_B x_E^{\xi}, \quad (11a)$$

$$\dot{h}(a) = (1-\zeta_j) z_s h(a)^{\gamma_1} x(a)^{\gamma_2}, \quad \text{for } 6 \leq a \leq 6+s. \quad (11b)$$

In the Appendix I show that the optimal level of human capital at the end

of the (endogenously chosen) schooling period is

$$\frac{1-\gamma_2}{\gamma_2}(\gamma_2(1-\zeta_j)z_s)^{\frac{1}{1-\gamma_2}}(h_j^*(s))^{\frac{\gamma_1}{1-\gamma_2}} = \frac{\rho(\lambda_j)}{w^{\frac{\gamma_2}{1-\gamma_2}}}\left[\frac{V_j^0}{(V_j^1)^{\frac{1}{1-\gamma_2}}}w^{\frac{\gamma_2}{1-\gamma}} + (V_j^1)^{-\frac{\gamma_2}{1-\gamma_2}}h_j^*(s)\right] \tag{12}$$

The left hand side of equation (12) is increasing and concave, while the right hand side is linear in human capital. Thus, it is possible that there is no positive solution to this equation. This can happen, for example, if an individual of type j has a very low ability to learn, i.e. high ζ_j. In that case, the equilibrium level of human capital at age 6 is h_E and the individual chooses zero schooling.

Generically, equation (12) has two solutions. There are two constraints that must be satisfied. First, no solution with $h_j^*(s) < h_E$ is a solution to our problem since during the schooling period capital does not depreciate. Second, the first order condition requires that, at the equilibrium $h_j^*(s)$, the net marginal gain from staying in school (the left hand side of equation (12)) intersects the marginal time cost (the right hand side of equation (12)) from above.

Inspection of this equation shows that:

1. *The effect of a disease that reduces ability to learn*: An increase in ζ_j reduces $h_j^*(s)$. If increases in ζ_j are viewed as the effect of some health condition (e.g. as a measure of days of schooling lost by a child infected with malaria) then the human capital of an infected individual will be lower than that of a healthy individual.

2. *The effect of an infectious debilitating disease*: In the context of this

model, decreases in υ_A —a measure of the debilitating effect of HIV/AIDS— and increases in η —the infection rate— correspond to a more severe effect of HIV/AIDS on the ability to produce. Simple algebra shows that if $\lambda_A < r+(1-\gamma_2)/(1-\gamma)$ —a condition satisfied by our calibrated values— then decreases in υ_A decrease $h_j^*(s)$. The effect of an increase in the rate of infection with HIV/AIDS, η, depends on the severity of infection. It can be shown that it unambiguously decreases the stock of human capital when η is large. For small values of η, the right hand side of equation (12)) pivots clockwise but, for our calibrated parameters still results in a decrease in $h_j^*(s)$. However, the effect of increases in η appear nonlinear, and they are stronger the larger the infection rate.

3. *The effect of the probability of disability/death*: If $j = A$ the right hand side of equation (12) is increasing in $\rho(\lambda_j)$ and increases in λ_j lower the amount of human capital at the end of the schooling period. If $j \in \{H, M\}$ and $\gamma_2 \leq 1/2$ (as it is in the calibration) the same results obtain for these two cases.

4. *The effect of economic growth*: If the right hand side of equation (12) is decreasing in the wage rate (as it turns out to be in the calibrated version), increases in w increase $h_j^*(s)$.

In order to determine the implications of the model for years of schooling —a measure of human capital with a readily available quantitative component— it is necessary to solve for the optimal human capital accumulation policy during the school years.

The derivation in the Appendix shows that the solution for years of schooling and the shadow price of human capital are given by

$$\left(h_j^*(s)\right)^{\frac{1-\gamma}{1-\gamma_2}} - (\xi q_E)^{\frac{\xi(1-\gamma_1)}{1-\xi}} h_B^{\frac{1-\gamma_1}{1-\xi}} \left(h_j^*(s)\right)^{-\frac{\gamma_1\gamma_2}{1-\gamma_2}} = \tag{13}$$
$$\frac{(1-\gamma_1)(1-\gamma_2)}{\rho(\lambda_j)\gamma_2}(\gamma_2(1-\zeta_j)z_s)^{\frac{1}{1-\gamma_2}}(wV_j^1)^{\frac{\gamma_2}{1-\gamma_2}}[1-e^{-\frac{\rho(\lambda_j)\gamma_2}{1-\gamma_2}s}].$$

and

$$wV_j^1(h_j^*(s))^{\gamma_1}e^{-\rho(\lambda_j)s} = \left[q_E^{1-\xi(1-\gamma_1)}h_B^{\gamma_1}\xi^{\gamma_1\xi}\right]^{\frac{1}{1-\xi}}. \tag{14}$$

Equations (12), (13) and 14) completely summarize the solution of the optimal level of schooling and human capital for an individual with health condition j. Equation (12) by itself pins down the individually optimal level of human capital at the end of the (endogenously determined) schooling period. Equations (13) and (14) jointly determine the shadow price of human capital at age 6, q_E, and the level of schooling, s.

3.3 The Distribution of the Population

In this section I derive the steady state distribution of the population. It is convenient to separately keep track of the number of healthy individuals, those who are infected with malaria (but not HIV/AIDS) and those who have HIV/AIDS. To simplify the discussion, I analyze separately those who are infected with HIV/AIDS who were healthy before from those who already had malaria.

3.3.1 The Healthy and HIV/AIDS Population

I assume that healthy individuals die at the rate λ_H and they become infected at the rate η. Infected individuals die at the rate λ_A. I concentrate on the balanced growth distribution and assume that population is growing at a constant rate. Thus, for example, the number of healthy individuals who are at most a years old at time t is $N^H(a,t) = \Phi^H(a)G(t)$, with $\dot{G}(t) = gG(t)$ for some g.

The evolution of the population satisfies

$$\frac{\partial N^H}{\partial a}(a,t) + \frac{\partial N^H}{\partial t}(a,t) = -\lambda_H N^H(a,t) - \eta N^H(a,t) + B_H G(t),$$

where B_H is a measure of the birth rate of healthy individuals. Given the steady state assumption, the previous equation is equivalent to

$$\dot{\Phi}^H(a) = -(\lambda_H + g + \eta)\Phi^H(a) + B_H,$$

which has a unique solution that satisfies $\Phi^H(0) = 0$ given by

$$\Phi^H(a) = \frac{B_H}{\lambda_H + g + \eta}(1 - e^{-(\lambda_H + g + \eta)a}).$$

The density of this distribution is $\phi^H(a)$ which is

$$\phi^H(a) = B_H e^{-(\lambda_H + g + \eta)a}$$

Consider next the HIV/AIDS infected population consisting of individuals who were previously healthy. The fraction of infected individuals who are at most a years old who were infected at age p (of course, $p \leq a$) at time t is $N_H^A(a,p,t) = \Psi_H^A(a,p)G(t)$. This measure of the population must satisfy the following partial differential equation

$$\frac{\partial N_H^A}{\partial a}(a,p,t) + \frac{\partial N_H^A}{\partial t}(a,p,t) = -\lambda_A N_H^A(a,p,t) + \eta N^H(p,t),$$

which is equivalent to

$$\frac{\partial \Psi_H^A}{\partial a}(a,p) = -(\lambda_A + g)\Psi_H^A(a,p) + \eta \Phi^H(p)$$

Let the density of that distribution be denoted $\psi_H^A(a,p)G(t)$. Then,

$$\frac{\partial \psi_H^A}{\partial a}(a,p) = -(\lambda_A + g)\psi_H^A(a,p).$$

This expression says that the relative size of the cohort of age a who became infected at age p shrinks because of deaths (given by the exit parameter λ_A) and the rate of population growth. The solution to this equation is

$$\psi_H^A(a,p) = e^{-(\lambda_A+g)a}\tilde{\psi}_H^A(p),$$

for some function $\tilde{\psi}_H^A(p)$. To pin down the boundary condition, note that $\psi_H^A(p,p)$ is the inflow of individuals from the healthy population to the HIV/AIDS infected population. Thus, it must be the case that

$$\psi_H^A(p,p) = \eta \phi^H(p),$$

which implies that

$$\tilde{\psi}_H^A(p) = \eta \phi^H(p) e^{(\lambda_A+g)p}.$$

The density of HIV/AIDS individuals of age a who were infected at age p is then

$$\psi_H^A(a,p) = e^{-(\lambda_A+g)(a-p)}\eta\phi^H(p) = \eta B_H e^{-(\lambda_A+g)(a-p)}e^{-(\lambda_H+g+\eta)p}.$$

The total fraction of the population in this category is

$$\Psi_H^A = \int_0^\infty [\int_0^a \psi_H^A(a,p)dp]da = \frac{\eta}{\lambda_A+g}\frac{B_H}{\lambda_H+g+\eta}$$

Consider next the population born with HIV/AIDS. A calculation that parallels that for healthy individuals implies that the fraction of the population in this category that is at most a years old is

$$\tilde{\Phi}^A(a) = \frac{B_A}{\lambda_A + g}\left(1 - e^{-(\lambda_A+g)a}\right),$$

where B_A is a measure of the rate of individuals born with HIV/AIDS. The density of this distribution is

$$\phi^A(a) = B_A e^{-(\lambda_A+g)a}.$$

3.3.2 The Malaria and HIV/AIDS Population

The evolution of the population that is infected with malaria exclusively parallels the distribution of the healthy population. The relevant fraction is

$$\Phi^M(a) = \frac{B_M}{\lambda_M + g + \eta}(1 - e^{-(\lambda_M+g+\eta)a}),$$

where B_M is a measure of individuals born (or infected early in life) with malaria, and $1/\lambda_M$ is the life expectancy of an individual infected with malaria.

Finally the density of the HIV/AIDS infected population that was previously malarial is given by

$$\psi_M^A(a,p) = \eta B_M e^{-(\lambda_A+g)(a-p)} e^{-(\lambda_M+g+\eta)p}$$

and the total mass is

$$\Psi_M^A = \frac{\eta}{\lambda_A + g}\frac{B_M}{\lambda_M + g + \eta}.$$

The model implies that the total number of HIV/AIDS individuals is

$$\Phi^A(a) = \tilde{\Phi}^A(a) + \Psi_H^A(a,a) + \Psi_M^A(a,a)$$

3.3.3 The Equilibrium Growth Rate

In order to determine the equilibrium growth rate it is necessary to impose that the measures of births be consistent with birth rates. Let $m(i,j)\beta^i$ be the birth rate to an individual in state i of a child in state j, and let $\bar{\Phi}^j = \lim_{a\to\infty} \Phi^j(a)$, with $i,j \in \{H,M,A\}$. Then,

$$\begin{aligned} B_H &= m(H,H)\beta^H\bar{\Phi}^H + m(A,H)\beta^A\bar{\Phi}^A + m(M,H)\beta^M\bar{\Phi}^M, \\ B_M &= m(H,M)\beta^H\bar{\Phi}^H + m(A,M)\beta^A\bar{\Phi}^A + m(M,M)\beta^M\bar{\Phi}^M \\ B_A &= m(A,A)\beta^A\bar{\Phi}^A, \end{aligned}$$

where I assume that an infected individual can give birth to a healthy child, an infected child or a malarious child. Since

$$\begin{aligned} \bar{\Phi}^H &= \frac{B_H}{\lambda_H + g + \eta}, \\ \bar{\Phi}^M &= \frac{B_M}{\lambda_M + g + \eta} \\ \bar{\Phi}^A &= \frac{B_A}{\lambda_A + g} + \frac{\eta}{\lambda_A + g}\left[\frac{B_H}{\lambda_H + g + \eta} + \frac{B_M}{\lambda_M + g + \eta}\right], \end{aligned}$$

and imposing the normalization (equivalent to fixing the size of the initial population)

$$\bar{\Phi}^H + \bar{\Phi}^M + \bar{\Phi}^A = 1,$$

this system can be solved for the growth rate. Rather than describing the more general setting, I discuss a simple but reasonable parameterization.

3.3.4 A Special Case

In order to highlight the forces at work, I will consider an interesting special case in the rest of the paper. I assume that the birth rates and death proba-

bilities of malarious and healthy individuals are the same[4], and I denote by $m(H)$ the fraction of all births to a healthy (and a malarious) person who are healthy. I denote by $m(A)$ the fraction of births to an HIV/AIDS positive that result in an infected child. I assume that the distribution between healthy and malarious is the same for HIV/AIDS and others.

I let $\beta^A = \varpi\beta^H$ for some factor ϖ. In the calibration below I find that $\varpi < 1$. Finally, I set $\Phi{=}\bar{\Phi}^A$. With these conventions the relevant steady state conditions are,

$$\begin{aligned} B_H &= m(H)\beta^H(1-\Phi) + m(H)\varpi\beta^H(1-m(A))\Phi, \\ B_M &= (1-m(H))\beta^H(1-\Phi) + (1-m(H))\varpi\beta^H(1-m(A))\Phi, \\ B_A &= m(A)\varpi\beta^H\Phi, \end{aligned}$$

and,

$$(\lambda_H + g + \eta)(1-\Phi) = B_H + B_M, \tag{15a}$$

$$\Phi = \frac{B_A}{\lambda_A + g} + \frac{\eta}{\lambda_A + g}[1-\Phi]. \tag{15b}$$

Simple manipulation of these equations show that the equilibrium level of HIV/AIDS prevalence, Φ, and the population growth rate, g, are the solutions to the following two equations:

$$g = G^1(\Phi) \equiv (\beta^H - \lambda_H)(1-\Phi) + (\varpi\beta^H - \lambda_A)\Phi, \tag{16}$$

$$g = G^2(\Phi) \equiv (\beta^H - \lambda_H - \eta) + \varpi\beta^H(1-m(A))\frac{\Phi}{1-\Phi}. \tag{17}$$

[4]Even though death rates from malaria are higher than those for the population at large they are much more significant early on in life before significant investments in human capital have taken place.

It is straightforward to check that the there is always a unique solution such that $\Phi \in (0,1)$ and $g > 0$. It follows that the impact of increases in the (exogenous) demographic factors on HIV/AIDS prevalence, Φ, and the population growth rate, g, are

Factor	Φ	g
η	+	-
$m(A)$	+	-
λ_A	+	+
λ_H	+	-
β^H	-	+
ϖ	+	+

From the perspective of the issues I am interested in, the two effects worth emphasizing are those associated with transmission of HIV/AIDS: lower transmission rates (η), and lower rates of infection in newborns ($m(A)$) both decrease the prevalence of HIV/AIDS but they increase population growth rate which, in general equilibrium has a negative impact on output per worker.

4 Equilibrium

As a first pass, I will study an open economy that takes the interest rate as given. The aggregate stock of human capital available for production on a per capita basis is given by

$$h^e = \sum_{j\in\{H,M,A\}} \int_0^\infty h_j^e(a)\phi^j(a)da + \sum_{j\in\{H,M\}} \int_0^\infty [\int_0^a h_{A,j}^e(a,p)\psi_j^A(a,p)dp]da,$$

Letting s_j be the level of schooling for type j, the fraction of the population working is

$$\varphi = \sum_{j\in\{H,M,A\}} \int_{6+s_j}^{\infty} \phi^j(a)da + \sum_{j\in\{H,M\}} \int_{6+s}^{\infty} [\int_0^a \psi_j^A(a,p)dp]da.$$

Profit maximization requires that

$$r + \delta_k + g = zF_k(\kappa, 1),$$

where κ is the physical-human capital ratio. It follows that

$$w = zF_h(\kappa, 1),$$

and output per worker is

$$\bar{y} = \frac{zF(\kappa,1)h^e}{\varphi}.$$

Unlike the models discussed by Young (2005), Weil (2007) and Ashraf et. al. (2009), I assume that savings adjust so as to keep the capital-human capital ratio equal to the user cost of capital. If the population growth rate does not change in response to changes a health, the capital-human capital ratio is unchanged and the change in output is completely determined by changes in the stock of human capital and the labor force participation rate.

5 Calibration

This section briefly describes the approach that I use to match the model with the data. I use the parameters of the human capital accumulation technology from Manuelli and Seshadri (2009). I assume that the technology is Cobb-Douglas with a capital coefficient equal to 1/3. The calibrated values are in Table A.1 in the Appendix. Then, I use different studies on the impact of malaria and HIV/AIDS to calibrate the disease specific parameter.

Population Parameters As indicated before, I assume that the birth rates for healthy and malarious individuals are the same, that is, $\beta^H = \beta^M$. As a first pass I also assume that the probability that a child is infected with malaria (is born malarious) is independent of the health status of the mother. This is a crude approximation since it is likely that common geographical factors can result in malarious mothers having a higher proportion of children with malaria. However, relaxing this requires access to more detailed information that is not readily available. These assumptions imply

$$\begin{aligned} \beta^H &= \beta^M, \\ m(M) &= m(H,M) = m(A,M) = m(M,M). \end{aligned}$$

Since the emphasis of the paper is on the effects of human capital, it seems reasonable to assume that $\lambda_H = \lambda_M$ since most of the differences in mortality associated with malaria occur early on in life before significant investments in human capital take place. Thus, as a first approximation, I just ignore this source of heterogeneity.

The fertility behavior of HIV/AIDS individuals relative to non-HIV/AIDS agents is a controversial subject and the empirical literature has not settled the issues related to the overall impact of the disease on birth rates. Young (2005) argues that an increase in the prevalence of HIV/AIDS lowers fertility. More recent analysis by Fortson (2009) and Juhn et. al. (2009) are consistent with the view that birth rates for infected women are about 20% lower and that the prevalence of HIV/AIDS has little impact on the fertility of healthy

women[5]. Thus, as a first approximation, I assume

$$\beta^A = 0.80 * \beta^H.$$

In order to estimate expected lifetimes of healthy individuals, λ_H, the transmission rate of HIV/AIDS, η, and the fraction of children who are born with HIV/AIDS, $m(A)$. I use aggregate demographic data and the proportion of all individuals who are younger than 15 years of age and have HIV/AIDS.

Life expectancy is a weighted average of the life expectancy of healthy and infected individuals. Since the emphasis is on the effect of human capital, I use an estimate of life expectancy at age 5 rather than life expectancy at birth since most of the investments take place after that age. Let P_A be the observed HIV/AIDS prevalence rate, and let T be life expectancy. Then, given that λ_A is known (I discuss this parameter later), then

$$T = \frac{1 - P_A}{\lambda_H} + \frac{P_A}{\lambda_A},$$

is used to calibrate λ_H.

Total fertility rate per person (not woman) is the product of the instantaneous birth rate, β^H, and the life expectancy of different types of individuals. This rate satisfies

$$F = \beta^H \frac{1 - P_A}{\lambda_H} + 0.80 * \beta^H \frac{P_A}{\lambda_A},$$

which is used to calibrate β^H.

Assuming that the population who is HIV/AIDS at age 14 has been born with that condition, we have that

$$\Phi^A(14) = \frac{0.80 * \beta^H m(A) P_A}{\lambda_A + g}(1 - e^{(\lambda_A + g) * 14}),$$

[5]See also the findings and the discussion in Kalemli-Ozcan (2009a) and (2009b).

and this equation can be used to calibrate $m(A)$.

Finally, the prevalence rate satisfies (see equation (15b))

$$P_A = \Phi^A(14) + \frac{\eta}{\lambda_A + g}(1 - P_A),$$

which I use to calibrate η.

One problem with any estimate assuming that the country is in a steady state is that, potentially, there are inconsistencies as some steady state conditions are not imposed/used. In this case, I am not imposing the steady state condition for the growth rate of population growth. To be internally consistent (but not necessarily consistent with the calibration), I calculate the growth rate implied by the model and the calibrated parameters as

$$g = \beta^H(1 - \Phi) + 0.80 * \beta^H\Phi - \lambda_H(1 - \Phi) - \lambda_A\Phi.$$

Finally, given these estimates the model implies that the fraction of malarious births is[6]

$$1 - m(H) = \frac{(\lambda_H + g + \eta)P_M}{\beta^H(1 - .2P_A)},$$

where P_M is a measure of incidence of malaria.

The results for a subset of countries are displayed in Table A.2 in the Appendix

AIDS: Calibrating the Parameters Since the objective of this paper is to illustrate how the model can be disciplined, I will use some simple estimates of the parameters that are relevant. Whenever possible I double

[6]To be precise, $1 - m(H)$ should be viewed as a measure of the fraction of children who survive to age 5 who are malarious since my calibration essentially ignores what happens before age 5.

check the calibrated values for consistency. Salomon (2006) presents data on incidence, prevalence and mortality estimates corresponding to the year 2000. Since

$$\frac{\text{Mortality}}{100,000} = \lambda_A P_A$$

Using a simple average of male and females values Salomon's data implies

Concept	AFRO D	AFRO E	World
λ_A	0.10	0.094	0.08

According to Salomon, Gakidou and Murray (undated) a good approximation to the distribution of death times conditional on infection is given by the Weibull distribution. Using this distribution, the probability that an infected individual will die before τ years after infection is

$$W(\tau) = 1 - \exp\{-\varkappa_0 \tau^{\varkappa_1}\}.$$

If this probability is to match the probability according to the exponential distribution, it must be the case that

$$\varkappa_0 \tau^{\varkappa_1 - 1} = \lambda_A.$$

Since for the reported parameters the mean of the Weibull distribution is 10, the estimate of λ_I according to this criterion would be 0.08. Finally, if instead of matching the cumulative probability I chose to match the mean, then the estimate of λ_A is 10. Young (2005) presents data on cumulative survival rates. He shows that the probability of surviving more than 10 years after infection is approximately 0.40. Thus, another estimate of λ_A is given by

$$\lambda_A = -\frac{\ln(0.40)}{10} = 0.091$$

It seems that even though it is not clear that the data come from a steady state distribution —and much less from an exponential— a reasonable estimate of λ_A is somewhere between 0.08 and 0.10, with values for high infection areas closer to the upper bound.

In addition to its impact on life expectancy HIV/AIDS can potentially reduce an individuals ability to work. The estimates are controversial and in many cases emphasize the loss of income in the last few years. I consider two possible values of υ_A, 0.01 and 0.05 which corresponds to a decrease in the endowment of labor of 1% and 5% respectively.

Malaria As in the case of HIV/AIDS the effect of malaria is completely summarized by two parameters: the learning ability while in schooling, $1 - \zeta_M$, and the effective endowment of labor, υ_M. Bleakley (2010a) estimates the impact of eradication on average income in the U.S. around 1920 and in Brazil, Colombia and Mexico in the 1950s. He estimates that the gains in regional income income from complete malaria eradication are somewhere between 10% (U.S.) and 30% (Brazil). He finds a small impact on years of schooling. A low estimate is that complete eradication increases schooling by 0.10-0.20 years. Lucas (2010) reports the impact of a given decrease in the prevalence of malaria on school attainment in Paraguay and Sri Lanka. She estimates that a 10% decrease in prevalence increases schooling by 10%. Thus, this corresponds approximately to an increase in one year of completed schooling associated with malaria eradication.

I use the U.S. in 1920 as my basic economy since around that time malaria was eradicated. I assume that life expectancy is 54 years and adjust the wage

rate so that individuals choose to stay in school for 6.7 years which is an estimate of average schooling around that time. I consider two estimates of the impact of school attendance due to malaria: the low estimate assumes that malaria reduces school attendance by 0.2 years, and the high estimate that schooling decreases by a full year. The following values of the parameters (ζ_M, υ_M) are consistent with these equilibrium changes in schooling for an economy parameterized to mimic the U.S. in the pre eradication period.

Malaria Parameters		
Calibration	ζ_M	υ_M
$\Delta s = -0.2$	0.003	0.93
$\Delta s = -1.0$	0.001	0.91

These calibrated parameters should be viewed as a first (rough) approximation. They imply that income of a malarious worker at age thirty one relative to the income of a healthy worker is 14% and 23% lower in the case of $\Delta s = -0.2$ and $\Delta s = -1.0$, respectively. These implications are consistent with Bleakley's findings even though more work is needed to understand how malaria affects age-earnings profiles.

6 Disease and Development

In this section I report the implications of the model for some experiments that capture the impact of reducing the burden of disease and simple economic development. The basic strategy is simple: I allocate to each country its population parameters (from Table A.2) but I assume that technological parameters are the same across countries. I then choose for each country a

level of TFP so that the model's predictions for output per worker coincide with the estimate in version 6.3 of Summers-Heston Dataset. Throughout I consider the high incidence of malaria and HIV/AIDS. To be precise, I assume that $(\zeta_M, \upsilon_M, \upsilon_A) = (0.001, 0.91, 0.95)$

6.1 The Role of Human Capital

Before reviewing the predictions of the model for the impact of changes associated with health policy, it is useful to understand the channels through which disease and economics interact. In Table 2 I present estimates of some measures of quality and quantity of human capital. The column labeled $\Delta(h^*/s)$ displays the difference in human capital per year of schooling between malarious and healthy children. Formally,

$$\Delta(h^*/s) = \frac{h^*_M(s^*_M)/s^*_M}{h^*_H(s^*_H)/s^*_H}.$$

The column labeled s^*_M/s^*_H reports the ratio of years of schooling between malarious and healthy children given the other conditions prevailing in the country. Finally, $\bar{h}^e/s$ is an index of the amount of human capital per worker supplied to the market per year of formal schooling. Even though the units themselves do not have an interpretation, the indicator is comparable across countries and I normalized Ghana's level to one.

Table 2. Human Capital and Schooling				
Country	y	$\Delta(h^*/s)$	$\frac{s_M^*}{s_H^*}$	$\bar{h}^e/s$
Cameroon	1.98	0.85	0.87	1.15
Ghana	1	0.92	0.82	1.00
Kenya	1.26	0.89	0.81	0.93
Malawi	0.82	0.96	0.75	0.80
Mozambique	1.24	0.88	0.82	0.83
Zambia	1.37	0.89	0.81	0.89
Zimbabwe	1.47	0.96	0.76	0.90

In the context of this model if one individual wants to adjust his stock of human capital there are three possible channels: years of schooling, quality of schooling and on the job training. The results in Table 2 suggest that the three dimensions move in the same direction. For example, a value of $\Delta(h^*/s)$ less than one implies that the quality of schooling chosen by individuals exposed to malaria is lower, per year of schooling, than the quality acquired by healthy individuals. Thus, malarious individuals not only acquire fewer years of schooling but they also have less human capital per year of schooling. The countries in this sample differ in terms of the average amount of human capital supplied to the market per year of schooling and the ranking, with the exception of Ghana, are positively correlated with the level of output per worker.

6.2 Disease: Reducing HIV/AIDS Transmission

The first exercise that I consider models the situation in which, through behavioral or policy changes, the rate of transmission of HIV/AIDS can be cut in half. To be precise, the experiment takes the basic estimates from the calibrated version of the model and recomputes the equilibrium with a value of η which is half the calibrated value.[7]

In Table 3, I present the predictions of the model (in the form of percentage changes relative to the base case) for the changes in output per worker, $\Delta\bar{y}$, output per capita, Δy, levels of schooling, $(\Delta s, \Delta s_H, \Delta s_M)$, an indicator of prevalence of HIV/AIDS, Φ, and the index of average human capital per year of schooling.

Table 3. Lower Transmission Rate (%)

Country	$\Delta\bar{y}$	Δy	$\Delta\Phi$	Δs	Δs_H	Δs_M	$\Delta(\bar{h}^e/s)$
Cameroon	9.2	9.6	-2.5	1.8	0.6	3.1	7.7
Ghana	4.0	4.0	-1.0	1.2	0.1	1.9	2.9
Kenya	14.2	14.4	-4.1	4.4	2.9	7.4	9.5
Malawi	19.5	17.5	-5.8	13.6	7.7	16.6	3.4
Mozambique	21.4	20.8	-6.2	9.4	5.7	10.8	10.5
Zambia	23.8	22.9	-7.3	11.6	7.3	13.4	10.2
Zimbabwe	24.3	25.1	-7.7	8.6	7.0	15.2	15.1

The effects decreasing HIV/AIDS transmission on output per worker can be quite large. In countries where HIV/AIDS does not affect a large fraction

[7]Even though this is a significant reduction it seems within the boundaries of what is possible. McNeil (2010) reports that a vaginal gel used by women reduced the infection rate by 39% on average and by 54% among those women who used the gel consistently.

of the population (e.g. Ghana) the gains are modest. At the other end, in countries with a significant fraction of HIV/AIDS individuals in the population the gains in output per worker exceed 30% in the long run. As a rough approximation, the model implies that a 1% decrease in the prevalence of HIV/AIDS through the decrease in the transmission rate increases long run output on average by 4.0%, with the impact slightly lower for high prevalence areas.

The impact on average schooling is a combination of increases in schooling by healthy and malarious individuals and a change in the composition of the population in favor of the former. In general, I find that the increase in years of schooling for malarious children exceed, and in some cases significantly so, the corresponding increase for healthy children.

The results in Table 3 suggest that even though average schooling increases, this changed is dwarfed by the increase in human capital per year of schooling. The decrease in the probability of infection has the same effect as an increase in effective life expectancy which corresponds to an increase in the utilization rate of human capital. This results in more schooling but also higher quality and more investment in on-the-job training.

As a separate exercise I considered reducing the mother to child infection rate to one half of the calibrated value. For this set of countries the impact on all variables was minimal. The largest effect on output per worker occurs in Zimbabwe and it amounts to an increase in output per worker of just above 0.1%. For that reason, I do not report the results of the exercise.

6.3 Disease: Increasing Life Expectancy associated with AIDS

Table 4 shows the effects of doubling life expectancy for individuals that become infected with HIV/AIDS. The results are in some dimensions similar to those corresponding to lower infections rates: changes in output per worker (in the steady state) are large for countries in which HIV/AIDS affects a significant fraction of the population. The distributional effects are also significant as malarious individuals increase their schooling more than healthy individuals. The largest increases in human capital occur in the quantity (schooling) dimension, as the effective amount of human capital per year of schooling, $\bar{h}^e/s$, displays smaller increments.

Table 4. Higher Life Expectancy - AIDS (%)

Country	$\Delta\bar{y}$	Δy	Δs	Δs_H	Δs_M	$\Delta(\bar{h}^e/s)$
Cameroon	10.8	9.9	1.9	0.6	4.3	7.9
Ghana	4.5	4.2	1.3	0.1	2.5	2.8
Kenya	16.5	14,2	4.8	3.4	10.1	9.0
Malawi	23.5	17.0	16.1	9.3	23.2	0.8
Mozambique	26.2	20.6	11.0	6.9	15.2	8.7
Zambia	29.9	22.4	13.7	9.0	19.3	7.7
Zimbabwe	32.5	29.3	9.5	8.5	22.8	18.1

6.4 Disease: Reducing the Incidence of Malaria

Table 5 displays the predictions of the model when the incidence of malaria among young children is reduced by one half. As equations (12), (13) and

14) show, individual choices are not affected by this policy. The only channel through which it has an impact on output is through composition effects.

Table 5. Lower Incidence of Malaria (%)

Country	$\Delta\bar{y}$	Δy	Δs	$\Delta(\bar{h}^e/s)$
Cameroon	7.7	6.6	2.4	4.1
Ghana	13.3	12.1	4.6	2.7
Kenya	4.6	3.6	2.4	1.2
Malawi	9.4	6.3	8.0	0.0
Mozambique	8.4	6.0	4.9	1.1
Zambia	7.4	4.8	4.9	0.0
Zimbabwe	1.4	1.0	1.1	0.0

As expected, the largest impact is to be found in the countries in which malaria incidence is high. From a quantitative point of view, the economic impact is smaller than the one found for the transmission of HIV/AIDS but still significant. Most of the changes are associated with increases in average schooling driven by an increase in the fraction of non-malarious individuals. These results are consistent with a number of empirical studies that find positive but small effects of malaria eradication.[8]

[8] See, for example, Barosfsky et. al. (2011), Bleakley (2010a), Cutler et. al (2010), Lucas (2010). Weil (2010) is also skeptical that malaria had a large impact on African development in the pre-colonial era. For an alternative model in which the presence of malaria can result in multiple steady states see Gollin et. al. (2007).

6.5 Disease: Combined Effect

Table 6 displays the results of jointly halving the transmission rate of HIV/AIDS, the incidence of malaria, and the mother to child transmission.

Table 6. Combined Effect (% change)

Country	$\Delta\bar{y}$	Δy	Δs	Δs_H	Δs_M	$\Delta(\bar{h}^e/s)$
Cameroon	23.9	23.1	4.0	0.5	5.2	18.5
Ghana	20.6	19.31	5.6	0.1	3.11	13.0
Kenya	29.5	28.0	7.6	3.7	12.3	19.0
Malawi	45.7	38.1	24.4	10.4	28.0	10.9
Mozambique	49.2	44.2	16.2	7.6	18.3	24.2
Zambia	52.9	46.6	19.2	9.8	22.9	23.1
Zimbabwe	47.9	47.5	11.7	8.8	26.5	32.0

The combined effects on output per worker of this "better" disease environment are large and range from 20% to over 50%. Even though keeping in mind that these are steady state values and, hence, that discounting will likely reduce their present value, it is hard not to view these findings as possibilities to induce economic growth in African countries. Given the constant interest rates, the increases in output require substantial increases in capital. Since the model is silent about domestic savings due to the constant interest rate, it is possible that attaining these large gains will require significant capital inflows.

In terms of the drivers of economic growth the results indicate that a large fraction of the increase in output per worker is associated with increases in the quality of human capital as measured by its value per year of schooling

(over 70%), while increases in years of education account for less than 30% of the total change.

As in the case of the individual changes in the disease environment, the combined effect seems to have a positive effect on income distribution as measured by years of education: malarious children increase their schooling proportionally more than healthy children and some of these differences are substantial.

6.6 Development

In this section I explore the consequences of a 3% increase in the marginal product of labor. In the context of this model this does not correspond to the conventionally measured real wage since the latter is given by the product of the marginal product of labor and the stock of human capital supplied to the market. Since human capital is not a simple function of schooling as quality is endogenous, adjustments for schooling do not map conventional measures of hourly wages into marginal product. This change in the marginal product roughly corresponds to a 2% increase in true TFP and yields increases in output per worker of about 17%.

The results are in Table 7.

Table 6. Development (% change)						
Country	$\Delta\bar{y}$	Δy	Δs	Δs_H	Δs_M	$\Delta(\bar{h}^e/s)$
Cameroon	16.9	13.2	7.6	7.1	8.7	2.2
Ghana	17.0	13.8	11.5	10.3	13.3	-1.0
Kenya	16.9	12.6	9.5	8.9	11.8	-0.1
Malawi	16.9	11.2	14.1	11.9	17.5	-5.4
Mozambique	16.9	12.1	9.3	8.3	10.9	-0.5
Zambia	16.9	11.3	9.8	8.7	11.8	-1.6
Zimbabwe	16.9	13.0	11.3	11.0	15.6	-1.5

There are several interesting observations. First, the quality dimension of human capital appears less responsive to increases in productivity than to changes in the disease environment as they account for a smaller fraction of the increase in output per worker. Second, even an across the board productivity change has differential impacts when it comes to schooling between malarious and non malarious individuals. In all cases the response of years of education to a change in the marginal product of capital is larger for the poorer and less educated individuals. Finally, the increase in schooling reduces labor force participation and this implies that output per capita does not increase as much as output per worker. The differences vary by country and appear to be driven by demographics and the initial level of schooling.

7 Conclusion and Directions for Further Research

In this paper I developed a model that can be used to evaluate the effect of changes in the disease environment on output per worker. The version that I discuss is very stylized and it could, and should, be adapted to specific country and disease conditions. However, the exercise shows that it is feasible to use micro estimates of the impact of diseases on schooling and income to calibrate the model so that it can be used in policy analysis.

I find that for a small sample of African economies the potential effects on output per worker of decreasing the rate of transmission of HIV/AIDS and the incidence of malaria to half their current levels are large and range somewhere between 20% and 50%. The model suggests that improvements in the disease environment are not neutral and they affect human capital investment decisions differentially.

Given the preliminary nature of the exercise, it seems safe to view the results as suggesting orders of magnitude. Nevertheless it provides an alternative to the conventional wisdom that disease eradication does not have a large impact on output.

Much work remains to be done since the model, as implemented in this draft, has limitations. First, it assumes an unrealistic distribution of lifetimes. This was done to facilitate the computation and to highlight the role of the disease parameters in influencing human capital choices. More realistic representations of the disability/death effects of some diseases are feasible but require more complex models and are more computationally demanding.

The model takes an "average" view of the effects of malaria. It is relatively straightforward to account for the intensity and frequency of malaria episodes (details are available from the author) and this would allow to better match the theory with the data. I hope to make progress in this direction in the near future.

The model also abstracted away from capital market imperfections that may limit an individual's ability to invest in human capital for reasons unrelated to the disease environment. Incorporating this type of imperfection is feasible but requires a more careful modeling of the role of the family as a substitute to the market. I also considered the case in which the interest rate is constant. However, it is possible to endogeneize it in a way that incorporates the effect of population growth and the number of children along the lines of Manuelli and Seshadri (2009).

The paper is salient about the welfare effects since it takes changes of the disease environment as exogenous and does not quantify its costs. For example, even in "simple" cases like malaria, it is not always straightforward to discover the impact on health and economic outcomes of particular interventions (see, for example, Ashraf et. al. (2010)).

The analysis was restricted to steady states but dynamics that do not change the interest rate are feasible to model but computationally more demanding. Last, but not least, the model can accommodate significant amounts of heterogeneity between individuals and regions within a country. However, it seems best that these extensions be performed in the context of a country and disease specific analysis.

Appendix

Determining Human Capital The problem faced by an individual with health status j is

$$\max\{-x_E - \int_6^{6+s} e^{-\rho(\lambda_j)a}x(a) + e^{-\rho(\lambda_j)(6+s)}[w(V_j^0 w^{\frac{\gamma_2}{1-\gamma}} + V_j^1 h(6+s))]\},$$

subject to

$$\begin{aligned} h_E &= h_B x_E^{\xi}, \\ \dot{h}(a) &= (1-\zeta_j)z_s h(a)^{\gamma_1} x(a)^{\gamma_2}, \quad \text{for } 6 \le a \le 6+s. \end{aligned}$$

The Hamiltonian for this problem (before age $6+s$) is simply

$$H = -x + q((1-\zeta_j)z_s h^{\gamma_1} x^{\gamma_2}).$$

where the costate variable satisfies

$$\dot{q}(a) = \rho(\lambda_j)q(a) - q(a)[\gamma_1(1-\zeta_j)z_s h^{\gamma_1} x^{\gamma_2} h^{-1}].$$

The boundary conditions are

$$q(6+s) = wV_j^1,$$

and that the value of the Hamiltonian at the boundary point (that is, at age $6+s$) must equal the negative of the impact on the continuation value. Formally, it is given by

$$\begin{aligned} -x(6+s) + q(6+s)[(1-\zeta_j)z_s h(6+s)^{\gamma_1} x(6+s)^{\gamma_2}] \\ = \rho(\lambda_j)[w(V_j^0 w^{\frac{\gamma_2}{1-\gamma}} + V_j^1 h(6+s))]. \end{aligned}$$

Let the stock of human capital of an individual of type j at age $6+s$ be denoted $h_j^*(s)$.Since the optimal choice of investment in school quality requires that

$$x(a) = q(a)\gamma_2(1-\zeta_j)z_s h(a)^{\gamma_1} x(a)^{\gamma_2},\ 6 \leq a \leq 6+s,$$

the condition for the optimal time to stop going to school is

$$\frac{1-\gamma_2}{\gamma_2}(\gamma_2(1-\zeta_j)z_s)^{\frac{1}{1-\gamma_2}}(h_j^*(s))^{\frac{\gamma_1}{1-\gamma_2}} = \frac{\rho(\lambda_j)}{w^{\frac{\gamma_2}{1-\gamma_2}}}\left[\frac{V_j^0}{(V_j^1)^{\frac{1}{1-\gamma_2}}}w^{\frac{\gamma_2}{1-\gamma}} + (V_j^1)^{-\frac{\gamma_2}{1-\gamma_2}}h_j^*(s)\right],$$

which corresponds to equation (12) in the text.

The Optimal Length of Schooling Since (see Seshadri and Manuelli (2009a)) it is possible to show that the expression $q(a)h^{\gamma_1}(a)$ satisfies

$$q(a)h^{\gamma_1}(a) = q_E h_E^{\gamma_1} e^{\rho(\lambda_j)(a-6)},\ \text{for } 6 \leq a \leq 6+s.$$

In particular, the number of years of schooling, s, must satisfy the $a = 6+s$ version of that expression, namely,

$$wV_j^1(h_j^*(s))^{\gamma_1} = q_E h_E^{\gamma_1} e^{\rho(\lambda_j)s}. \tag{19}$$

The left hand side of this expression is known (and so is essentially, h_E). However, the shadow price of human capital at age 6, q_E, needs to be determined. In particular, it influences the evolution of human capital. Since, along a solution path, it must be the case that

$$\dot{h}(a) = (1-\zeta_j)z_s h(a)^{\gamma_1}[q(a)\gamma_2(1-\zeta_j)z_s h(a)^{\gamma_1}]^{\frac{\gamma_2}{1-\gamma_2}},$$

then

$$\dot{h}(a) = (1-\zeta_j) z_s h(a)^{\gamma_1} (\gamma_2 (1-\zeta_j) z_s)^{\frac{\gamma_2}{1-\gamma_2}} (q_E h_E^{\gamma_1})^{\frac{\gamma_2}{1-\gamma_2}} e^{\frac{\rho(\lambda_j)\gamma_2}{1-\gamma_2}(a-6)}.$$

The solution to the differential equation that determines the evolution of human capital during the schooling period. This equation is given by

$$\dot{h}(a) = M h(a)^{\gamma_1} e^{\frac{\rho(\lambda_j)\gamma_2}{1-\gamma_2}(a-6)},$$

where

$$M = z_s (\gamma_2 z_s)^{\frac{\gamma_2}{1-\gamma_2}} (q_E h_E^{\gamma_1})^{\frac{\gamma_2}{1-\gamma_2}}.$$

The solution (that satisfies $h(6) = h_E$) is given by

$$h(a) = \left[K(e^{\frac{\rho(\lambda_j)\gamma_2}{1-\gamma_2}(a-6)} - 1) + h_E^{1-\gamma_1} \right]^{\frac{1}{1-\gamma_1}},$$

where

$$K = \frac{(1-\gamma_1)(1-\gamma_2)}{\rho(\lambda_j)\gamma_2} z_s (\gamma_2 z_s)^{\frac{\gamma_2}{1-\gamma_2}} (q_E h_E^{\gamma_1})^{\frac{\gamma_2}{1-\gamma_2}}.$$

Since this candidate $h(a)$ must hold for all $a \in [6, 6+s]$, it must the be that that $h(6+s) = h_j^*(s)$. Imposing this terminal condition, we obtain that

$$\left(h_j^*(s)\right)^{1-\gamma_1} - h_E^{1-\gamma_1} = K(e^{\frac{\rho(\lambda_j)\gamma_2}{1-\gamma_2}6} - 1). \tag{20}$$

Equations (20) and (19) jointly determine the equilibrium values of schooling, s, and the shadow price of human capital at age 6, q_E. Since the latter is only needed to determine the amount invested in early childhood human capital, it is convenient to present an expression that, given the value of $h_j^*(s)$ determines years of schooling. Using the value of $q_E h_E^{\gamma_1}$ from equation (19) in equation (20) I get (after some algebra)

$$\begin{aligned} & \left(h_j^*(s)\right)^{\frac{1-\gamma}{1-\gamma_2}} - h_E^{1-\gamma_1} \left(h_j^*(s)\right)^{-\frac{\gamma_1\gamma_2}{1-\gamma_2}} \\ = \; & \frac{(1-\gamma_1)(1-\gamma_2)}{\rho(\lambda_j)\gamma_2} z_s (\gamma_2 z_s)^{\frac{\gamma_2}{1-\gamma_2}} (w V_j^1)^{\frac{\gamma_2}{1-\gamma_2}} [1 - e^{-\frac{\rho(\lambda_j)\gamma_2}{1-\gamma_2} s}] \end{aligned}$$

which is equation (13) in the text. Equation (19) and the optimal h_E imply that

$$wV_j^1(h_j^*(s))^{\gamma_1}e^{-\rho(\lambda_j)s} = \left[q_E^{1-\xi(1-\gamma_1)}h_B^{\gamma_1}\xi^{\gamma_1\xi}\right]^{\frac{1}{1-\xi}}.$$

which is equation (14) in the text.

Calibration The common calibrated values are

Table A.1: Common Parameters

γ_1	γ_2	δ_h	z_s	z_h	h_B	r	α	δ_k
.63	.30	.018	.33	.37	1.64	.0531	.33	.071

In Table A.2 I present the estimates of the relevant population parameters when I use a measure of life expectancy that is an approximation to life expectancy conditional on reaching age 5. The column labeled $\hat{g}$ is the internally consistent population growth rate, that is, the growth rate implied by the model.

Table A.2: Calibrated Population Parameters						
Country	$T(5)$	λ_H	β^H	$m(A)$	η	$m(H)$
Angola	54	.018	.061	.149	.003	.736
Burkina Faso	58	.017	.053	.138	.002	.542
Cameroon	59	.016	.039	.218	.006	.664
Cent. Af. Rep.	58	.016	.042	.219	.007	.605
Congo	58	.017	.059	.166	.002	.642
Cote d'Ivoire	60	.016	.038	.327	.004	.596
Ethiopia	63	.016	.054	.231	.002	.870
Ghana	64	.015	.031	.216	.002	.530
Kenya	60	.016	.042	.239	.010	.774
Malawi	56	.016	.052	.196	.016	.522
Mali	58	.017	.057	.181	.002	.612
Mozambique	58	.015	.045	.152	.016	.559
Namibia	65	.013	.026	.296	.019	.551
Nigeria	58	.017	.048	.186	.004	.620
Sierra Leone	54	.018	.061	.125	.002	.682
South Africa	55	.015	.026	.210	.002	.999
Tanzania	56	.016	.048	.207	.007	.663
Zambia	52	.017	.052	.183	.020	.587
Zimbabwe	46	.019	.037	.272	.018	.916

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