Diseases and Development: A Theory of Infection Dynamics and Economic Behavior

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Abstract

We propose an economic theory of infectious disease transmission and rational behavior. Diseases are costly due to premature mortality and lost labor productivity and quality of life from morbidity. We calibrate the model to malaria and HIV in sub-Saharan Africa. The paper offers three insights. First, higher disease prevalence implies lower saving-investment propensity. Preventive behavior can partially offset this when the prevalence rate and disease externality are relatively low. Second, infectious disease can generate a low-growth trap where income alone cannot push an economy out of underdevelopment, a result that differs from development traps in the existing literature. Third, even when all economies converge to the same balanced growth path, infectious disease epidemiology can significantly impair the pace of economic development.

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1. Introduction

Motivated by evidence that a disproportionate share of the burden of ill health in developing countries comes from infectious diseases,[1] we incorporate rational disease behavior into a growth model.[2] Infection spreads from random exposure to disease vectors, susceptibility to which depends on prevention and the disease ecology (climate, vectorial capacity, culture and social practices). Diseases cause premature death as well as lower productivity and quality-of-life among the infected. The mortality effect makes infected individuals less inclined to save, while morbidity makes them less able to do so.[3] Preventive investment can help avert infectious diseases but its efficiency declines rapidly with the prevalence rate due to externalities.

Two types of long-run growth are possible in this economy. In one, diseases are widespread and growth is low, possibly zero. In the other, sustained improvement of living standards paves the way for a complete eradication of infectious disease. Initial income, disease prevalence and ecology determine which of these development paths attracts a particular country.

Income does not cause health in the low growth equilibrium *irrespective* of the economy's level of development. The disease externality overwhelms prevention incentives in this case and external assistance, for example financial aid, would have little impact on health or development. This result is in sharp contrast to development traps in the existing literature (Azariadis and Stachurski, 2005).

Along the higher growth path, the causality between income and health runs both ways: higher income assists prevention, lowers the incidence of communicable disease and generates stronger incentives for growth. We see this pattern as more relevant to the historical experience of Britain, Western Europe and its offshoots where infectious diseases were not endemic.

For developing countries, situated as they mostly are in the tropics and subject to a wide spectrum of disease vectors, a development trap is more plausible. Since income deficiency does not cause poor health in this equilibrium, our theory suggests marked health improvements can occur only from public health or medical innovations. Somewhat surprisingly this escape from ill health is at first accompanied by economic slowdown. The more favorable disease ecology raises returns on quantity of life faster than on quality of life (in the sense of Becker *et al.*, 2005): health investment is therefore initially preferred over other investments that can raise the growth rate more immediately.

There has been a recent surge in research on health and development. Despite compelling microeconomic evidence that health is important for economic outcomes (see, Strauss and Thomas, 1998, Deaton, 2003 for example), the macroeconomic evidence has been mixed. Empirical works such as Bloom and Canning (2005), Gallup and Sachs (2001) and Lorentzen *et al.* (2008) attribute Africa's persistent poverty to endemic diseases like malaria and to excessive adult mortality. Other works offer a more qualified view. Using a novel instrument to control for the endogeneity between health and income, Acemoglu and Johnson (2007) find small, if any, positive effect of health on income. These authors argue that the increase in population resulting from better health outweighs the productivity effects and therefore GDP per capita may have actually decreased slightly in their panel of countries. Weil (2007) uses microeconomic estimates on the effect of health on individual outcomes to construct macroeconomic estimates of the effect of (average) health on GDP per capita. He finds that eliminating health differences among countries will reduce the variance of log GDP per worker by about 10%

[1] Communicable diseases account for 55% of deaths in developing countries, 14% in developed countries (WHO, 2002).

[2] Ignoring the effect of rational behavior can convey an incorrect view of disease dynamics and the effectiveness of public health interventions (Geoffard and Philipson, 1996).

[3] There is some direct evidence that longevity has a non-trivial effect on saving and investment. See Deaton and Paxson (1994) for Taiwan, and Lorentzen *et al.* (2006) for cross-country evidence.

which is economically significant but substantially smaller than estimates from cross-country growth regressions. While our theory offers a framework to parse this conflicting evidence, it is important to note that none of these empirical works explicitly allow for nonlinearities in the relationship between health and growth.

This work is related to several theoretical contributions incorporating mortality into growth models. Among others, Kalemli-Ozcan (2002), Chakraborty (2004), Doepke (2005), Soares (2005) and Cordoba and Ripoll (2009) variously consider the effect of child and adult mortality on fertility, human capital, demographic change and economic growth. Theoretical work on the microfoundation of diseases and growth is more limited. Momota *et al.* (2005) generate disease cycles in a general equilibrium setup. Epidemic shocks in Lagerlöf (2003) and nutritional improvements in Birchenall (2007) are used to explain the escape from Malthusian stagnation to modern economic growth. None of these theories, however, are particularly clear about the disease transmission process and the role of prevention choices. Our paper is also related to the Unified Growth Theory proposed in Galor (2005). In our model, a stagnant economy starts enjoying modern economic growth when prevalence rates fall sufficiently due to exogenous improvements in medicine, public health or the disease environment.

The remainder of the paper is organized as follows. Section 2 specifies the economic environment and the disease transmission mechanism. It then presents optimal economic and health behavior followed by general equilibrium dynamics. The calibration strategy and parameter choices are discussed in section 3. Section 4 analyzes sub-Saharan Africa's twin problems of disease and underdevelopment through the lens of our theory. We focus on malaria and HIV/AIDS as the two most devastating diseases affecting the region. A brief discussion of alternative parameter values in section 5 establishes that the effect of ill health is substantial even when there are no traps in income levels. Section 6 concludes.

2. The Model

Consider a discrete time, infinite horizon economy populated by overlapping generations of families. Each individual potentially lives for two periods, adulthood and old-age.[4] As adults, individuals are endowed with one unit of labor which they supply inelastically to the market. The modification that we introduce to the standard model is the possibility of contracting an infectious disease early in life and prematurely dying from it.

2.1. Disease Transmission

Infectious diseases inflict three types of costs on an individual. First, he is less productive at work, supplying only $1-\theta$ units of efficiency labor instead of unity. Second, there is a utility loss from being infected: the individual derives a utility flow of $\delta u(c)$ instead of $u(c)$ from a consumption bundle c, where $\delta \in (0,1)$. We interpret this as a quality-of-life effect. Thirdly, an infected young individual faces the risk of premature death and may not live through his entire old-age.

[4] We do not explicitly model childhood nor do we take into account child mortality. Children's consumption is subsumed into their adult parent's. Our rationale for focusing on adult mortality is that the enormous life expectancy improvements enjoyed by developing countries over the past fifty years has been primarily due to sharp declines in infant and child mortality, low-cost interventions, and substantial technology transfers. Adult mortality has declined relatively less, remains high in developing countries (World Bank, 1993) and disproportionately so due to infectious diseases. To the extent that child mortality is still a problem, we are ignoring its effect on fertility and investment in childhood human capital. Childhood morbidity from infectious diseases which have life-long repercussions is, however, implicitly incorporated in the cost of disease parameters.

Young individuals can undertake preventive health investment, x_t, early in life. This takes the form of net food intake (nutrients available for cellular growth), personal care and hygiene, accessing clinical facilities and related medical expenditure. It may even involve abstaining from risky behavior. What is key is that prevention that improves disease resistance is privately costly. We assume these costs take the form of foregone consumption but they could also take the form of direct utility loss as in Geoffard and Philipson (1996) or Bhattacharya *et al.*'s (2007) work on sexually transmitted HIV.

Diseases spread from the infected to the susceptible in two stages. In the first stage, a susceptible young person randomly meets μ_1 older individuals some of whom may be carriers of infectious diseases (intergenerational transmission). In the second stage, young adults socialize among themselves with each young person interacting with μ_2 young adults (intragenerational transmission).

We begin by deriving the probability p_t^1 of being infected after the first stage. Suppose the transmission rate of the disease from an infected old to a susceptible young is π_t. If encounters are independent, the probability of not getting infected during this stage equals the product (across meetings) of not being infected. The probability of being infected after one match is the probability of meeting an infected old individual (i_t) times the probability of getting infected by the encounter (π_t), that is, $i_t\pi(x_t)$. This reflects that not all of the older individuals are infected and not all encounters with infected people result in transmission. Hence,

$$p_t^1 = 1 - [1 - i_t\pi(x_t)]^{\mu_1}. \tag{1}$$

Applying the law of large numbers, this represents the proportion of young adults who are infected at the end of the first stage.

In the second stage, an infected person cannot get infected for a second time. For simplicity we assume that a young person who becomes newly infected in this stage does not go on to transmit the disease to susceptible members of his cohort. Hence the probability of getting infected in the second stage for someone who remained uninfected after the first one is

$$p_t^2 = 1 - [1 - p_t^1\pi(x_t)]^{\mu_2}. \tag{2}$$

The fraction of young adults who are infected after both stages consists of those who were infected in the first stage plus those who were not but subsequently were:

$$i_{t+1} = p_t^1 + (1 - p_t^1)p_t^2 \equiv p(i_t). \tag{3}$$

The rate of disease transmission is assumed identical for both types of encounters – the disease microbe does not distinguish between different types of disease vectors (humans in both cases here). It is also important to note that the negative externality associated with disease contagion rises exponentially with the number of encounters μ_1 and μ_2. This externality is endogenous to the disease propagation process and will be important in understanding some of the results below.

Next, we propose a parametric disease transmission function. Given preventive health investment x that takes on continuous values, the probability that a young individual gets infected from a matching with an infected individual in either stage is

$$\pi(x) = \frac{aq}{q + x},\ a \in (0, 1),\ q > 0. \tag{4}$$

This function satisfies the following properties: $\pi' < 0$, $\pi(0) = a$, $\pi(\infty) = 0$, and $\pi'(0)$ goes to $-\infty$ as q goes to zero. The parameter q captures the quality of national health institutions and possibly medical technology. As q falls, private preventive health investment becomes more productive. In

this sense, public and private health are complementary inputs. The evolutionary parameter a gives the probability of getting infected without prevention. Factors that influence its value are the genetic evolution of humans and virus mutations. An example is the sickle-cell trait, a genetic mutation that provides partial defense against malaria and is carried by about 25% of the human population in areas severely affected by the disease (see, Galor and Moav, 2005, for references and additional examples).

In order that disease prevalence can rise in some cases (presumably under low prevention), one infection should be able to cause multiple cases of infection. Suppose no one engages in prevention and the prevalence rate is low. The disease transmission function $p(i_t)$ can be approximated by

$$p(i_t) \simeq p_t^1 + p_t^2 \simeq a\mu_1(1 + a\mu_2)i_t \equiv \nu i_t. \tag{5}$$

The parameter ν is a *disease multiplier*, the cumulative number of cases per primary case in a relatively uninfected population. We require that $\nu > 1$ for which either μ_1 or μ_2 has to be sufficiently large.

Finally, once infection status is determined, consumption and saving choices are made in the usual manner. This is the simplest way to incorporate rational disease behavior. More generally, infected individuals could also undertake curative investment to reduce the length and severity of illness, the inclusion of which should not qualitatively alter the model's predictions

Several features of the disease environment should be noted. First, although we occasionally refer to *the* infectious disease, we want to think about such diseases more generally. In particular, people may be infected by any number of communicable diseases and what is relevant is the overall morbidity and mortality from such diseases. Even if a particular disease is typically not fatal among adults, it can turn out to be so when accompanied by morbidity from other illnesses. For example large-scale trials of insecticide-treated bednets show that reduction in all-cause mortality is considerably greater than the mortality reduction from malaria alone (see Sachs and Malaney 2002 and references therein).

Secondly, assuming diseases are transmitted directly from an infected to a susceptible person is a simplification. The parameter μ captures the disease vector more generally. For a disease like AIDS, it can be directly related to the number of sexual partners or needle-sharers.[5] It may also be related to population density (here exogenous) for a disease of the pulmonary system like tuberculosis. For a disease like malaria that is transmitted via infective mosquitoes, μ has the more appropriate interpretation of the mosquito's vectorial capacity.

Third, within this disease ecology falls social norms and behavior. For instance, social customs may limit the ability of a woman to deny sexual relationship with infected partners even when she is aware of her partner's HIV+ status (Gupta and Weiss, 1993; Wellings *et al.,* 2006). Norms such as these would naturally increase the rate of transmission μ. Likewise, tuberculosis is widely stigmatized in many societies especially when precise knowledge of its transmission and prevention is not available. Stigmatization can include job loss, divorce, being shunned by family members and even loss of housing (Jaramillo, 1999; Lawn, 2000). Infected individuals who would be otherwise circumspect in their social interactions may remain actively involved or simply hide their disease to avoid isolation.

2.2. Preferences

Preferences and economic behavior are disease contingent. The period utility function $u(c)$ is increasing, twice continuously differentiable with $u' > 0$, $u'' < 0$. In addition, it is homothetic, and current and future consumptions are normal goods.

[5] In a recent survey on global sexual behavior Wellings *et al.* (2006) argue that, contrary to popular perception, Africa's HIV/AIDS epidemic has more to do with poverty and mobility than promiscuity.

First consider decisions of an uninfected individual whose health investment has successfully protected him from the disease. This individual maximizes lifetime utility

$$u\left(c_{1t}^{U}\right)+\beta u\left(c_{2t+1}^{U}\right),\ \beta\in(0,1), \tag{6}$$

subject to the budget constraints

$$c_{1t}^{U}=w_t-x_t-z_t^{U}\text{ and }c_{2t+1}^{U}=R_{t+1}z_t^{U}, \tag{7}$$

where z denotes savings and x is given by decisions made early in period t.[6] Hereafter we tag variables by U and I to denote decisions and outcomes for uninfected and infected individuals respectively.

An infected individual faces a constant probability $\phi\in(0,1)$ of surviving from the disease before reaching old-age. Normalizing utility from death to zero, expected lifetime utility

$$\delta\left[u\left(c_{1t}^{I}\right)+\beta\phi u\left(c_{2t+1}^{I}\right)\right], \tag{8}$$

is now maximized subject to

$$c_{1t}^{I}=(1-\theta)w_t-x_t-z_t^{I}\text{ and }c_{2t+1}^{I}=R_{t+1}z_t^{I}+\tau_{t+1}. \tag{9}$$

Here τ_{t+1} denotes lump-sum transfers received from the government. We assume an institutional setup whereby the government collects and distributes the assets of the prematurely deceased among surviving *infected* individuals.[7] In equilibrium, transfers per surviving infected individual will be

$$\tau_{t+1}=\left(\frac{1-\phi}{\phi}\right)R_{t+1}z_t^{I}. \tag{10}$$

The first-order necessary conditions for optimal consumption are the familiar Euler equation for each type

$$u'(c_{1t}^{U})=\beta R_{t+1}u'(c_{2t+1}^{U}) \tag{11}$$

$$u'(c_{1t}^{I})=\beta\phi R_{t+1}u'(c_{2t+1}^{I}), \tag{12}$$

given the price vector (w_t,R_{t+1}) and preventive investment x_t.

Hereafter we assume the analytically convenient CRRA utility function

$$u(c)=\frac{c^{1-\sigma}-1}{1-\sigma},\ \sigma\in(0,1). \tag{13}$$

This function takes on negative values when consumption is less than one (in the calibration below we set $\sigma=1$). Since utility from death is zero, below we assume that the aggregate technology is "productive enough" to ensure that consumption exceeds one.

All saving is invested in capital which is rented out to final goods producing firms. The initial old generation is endowed with a stock of capital K_0 at $t=0$. The depreciation rate on capital is set equal to one and an exogenously specified fraction i_0 of the initial old are infected. We summarize the timeline of events in Figure 1.

[6] Implicitly x is financed by zero-interest loans taken early in youth from the rest of the world and repaid after the labor market clears.

[7] Alternatively, we could have assumed perfect annuities market with qualitatively similar results.

2.3. Technology

A continuum of firms operate in perfectly competitive markets to produce the final good using capital (K) and efficiency units of labor (L). To accommodate the possibility of endogenous growth, we posit a firm-specific constant-returns technology exhibiting learning-by-doing externalities. To ensure that consumption exceeds one, we consider the production technology[8]

$$F(K^i, L^i) = A(K^i)^\alpha(\overline{k}L^i)^{1-\alpha} + bL^i, \tag{14}$$

where A is a constant productivity parameter, and $\bar{k}$ denotes the average capital per effective unit of labor across firms. $b > 0$ captures "natural endowments" such as trees and animals that allow for positive consumption levels in the absence of physical capital. For b sufficiently large, consumption will be above one. For our model to generate a balanced growth path with strictly positive growth we assume that $(1-\alpha)A > 1$.

Standard factor pricing relationships under such externalities imply that the wage per effective unit of labor (w_t) and interest factor (R_t) are given respectively by

$$w_t = (1-\alpha)Ak_t + b \equiv w(k_t),$$

$$R_t = \alpha A \equiv R.$$

Note that $b > 0$ is not necessary to obtain the main results in this section but will be helpful with the calibration exercise later on.

2.4. Preventive Investment Decision

Our next task is analyzing privately optimal prevention. Given the Euler conditions (11), (12) and the utility function (13), optimal savings for uninfected and infected individuals are

$$z_t^U = \left(\frac{\beta^{1/\sigma}R_{t+1}^{1/\sigma-1}}{1+\beta^{1/\sigma}R_{t+1}^{1/\sigma-1}}\right)(w_t - x_t)$$

$$z_t^I = \left[\frac{(\beta\phi)^{1/\sigma}R_{t+1}^{1/\sigma-1}}{1+(\beta\phi)^{1/\sigma}R_{t+1}^{1/\sigma-1}}\right][(1-\theta)w_t - x_t] - \left[\frac{1}{1+(\beta\phi)^{1/\sigma}R_{t+1}^{1/\sigma-1}}\right]\frac{\tau_{t+1}}{R_{t+1}}.$$

Substituting these into lifetime utility gives the two indirect utility functions

$$V^U(x_t) = \frac{1}{1-\sigma}\left[\left(w_t - x_t - z_t^U\right)^{1-\sigma} + \beta\left(R_{t+1}z_t^U\right)^{1-\sigma}\right] - \frac{1}{1-\sigma} \tag{15}$$

$$V^I(x_t) = \frac{\delta}{1-\sigma}\left\{\left[(1-\theta)w_t - x_t - z_t^I\right]^{1-\sigma} + \beta\phi\left(R_{t+1}z_t^I\right)^{1-\sigma}\right\} - \frac{\delta}{1-\sigma}, \tag{16}$$

contingent on prices, preventive health investment and disease realizations.

A young individual's probability of catching the disease is $p_t \equiv p(i_t)$ given by (3). Individuals choose x_t to maximize expected lifetime utility

$$p_tV^I(x_t) + (1-p_t)\,V^U(x_t), \tag{17}$$

[8] The choice of a simple Ak mechanism is only for tractability. The story generalizes when saving behavior determines growth (in closed or open economies) via innovation and factor accumulation as, for example in Aghion *et al.* (2006), and also to exogenous growth frameworks in which case the model's predictions will be in terms of income levels instead of growth rates.

at the beginning of period t. The first order condition for this is

$$-\mu\left(1-i_t\pi_t\right)^{\mu-1}\pi'(x_t)i_t\left(V_t^U-V_t^I\right)\geq p_t\left(-\frac{\partial V_t^I}{\partial x_t}\right)+(1-p_t)\left(-\frac{\partial V_t^U}{\partial x_t}\right), \tag{18}$$

for $x_t \geq 0$, the expression holding with equality only for positive prevention. For individuals to be willing to invest in disease prevention, the marginal benefit from living longer and experiencing a healthier and more productive life cannot be outweighed by the marginal cost of foregoing current income.[9]

Substituting equilibrium prices and transfers into the saving functions obtains

$$z_t^U = s^U\left[w(k_t)-x(w_t,i_t)\right]\equiv z^U(k_t,i_t),$$

and

$$z_t^I = s^I\left[(1-\theta)w(k_t)-x(w_t,i_t)\right]\equiv z^I(k_t,i_t),$$

where,

$$s^U\equiv\left(\frac{\beta^{1/\sigma}R^{1/\sigma-1}}{1+\beta^{1/\sigma}R^{1/\sigma-1}}\right),\ s^I\equiv\left[\frac{\phi(\beta\phi)^{1/\sigma}R^{1/\sigma-1}}{1+\phi(\beta\phi)^{1/\sigma}R^{1/\sigma-1}}\right]. \tag{19}$$

Evidently $z_t^U > z_t^I$: given the wage per efficiency unit of labor and preventive investment, the infected save less since their effective discount rate is lower ($\phi < 1$) and they are less productive ($\theta > 0$). The third type of cost, a lower utility flow ($\delta < 1$) affects savings indirectly through preventive investment.

Substituting optimal saving into (15) and (16) leads to

$$V_t^{U*}=\zeta^U\frac{\left[w(k_t)-x_t\right]^{1-\sigma}}{1-\sigma}-\frac{1}{1-\sigma}, \tag{20}$$

and

$$V_t^{I*}=\zeta^I\frac{\left[(1-\theta)w(k_t)-x_t\right]^{1-\sigma}}{1-\sigma}-\frac{\delta}{1-\sigma}, \tag{21}$$

for $\zeta^U\equiv\left(1-s^U\right)^{1-\sigma}+\beta R^{1-\sigma}\left(s^U\right)^{1-\sigma}$and $\zeta^I\equiv\delta\phi^\sigma\left[\{(1-s^I)/[\phi+(1-\phi)s^I]\}^{1-\sigma}+\beta R^{1-\sigma}\left(s^I\right)^{1-\sigma}\right]$. We then substitute equilibrium prices and savings into the first order condition for preventive health investment. Note that individuals do not take into account equilibrium transfers given by (10) when making health investment decisions. Accordingly (18) becomes

$$p_t\zeta^I[(1-\theta)w(k_t)-x_t]^{-\sigma}+(1-p_t)\zeta^U[w(k_t)-x_t]^{-\sigma}\leq-\mu\left(1-i_t\pi_t\right)^{\mu-1}\pi'(x_t)i_t\left(V_t^{U*}-V_t^{I*}\right). \tag{22}$$

Two possibilities arise depending on whether or not prevention yields positive returns. If (22) holds as a strict inequality at $x_t = 0$, optimal investment will be $x_t = 0$. The left-hand side of (22) is the marginal utility cost of that investment, since health investment entails a lower current consumption. The right-hand side constitutes the marginal benefit in the form of higher net utility from lowering one's chance of contracting diseases. Optimal health investment is zero as long as the utility cost dominates, that is, returns to health investment are negative at $x_t = 0$. Intuitively we expect this to occur at levels of low income and high prevalence rates. Private actions have a negligible impact on leading a healthy and more productive life in such situations.

[9] The first order conditions (11), (12), and (18) are necessary but not sufficient since preferences can become non-convex with endogenous p. We verify that second order conditions are satisfied for the parameter values and functional forms we choose later on.

Rewriting (22) above, the condition for zero preventive investment becomes

$$\chi(k_t, i_t) = \left\{\zeta^U[1-p(0)] + \zeta^I(1-\theta)^{-\sigma}p(0)\right\} w_t^{-\sigma} + \mu\left[1 - i_t\pi(0)\right]^{\mu-1}\pi'(0)i_t\left[V_t^U(0) - V_t^I(0)\right] \geq 0.$$

We note that $\partial\chi/\partial k > 0$ and $\partial\chi/\partial i > 0$, that is, private returns from preventive health investment are negative at low values of k and high values of i.

For (k_t, i_t) combinations such that $\chi(k_t, i_t) < 0$ optimal investment in health will be positive. In this case (22) holds as an equality and

$$x_t = x(k_t, i_t),$$

where $\partial x/\partial k > 0$ (income effect) and $\partial x/\partial i > 0$ (higher disease prevalence encourages preventive investment).

2.5. Balanced Growth

Aggregate savings is the weighted average of the saving of infected and uninfected individuals $S_t = p_t z_t^I + (1-p_t)z_t^U$. The asset market clearing condition is $K_{t+1} = S_t$ and effective labor supply $L_{t+1} = 1 - \theta p_{t+1}$.

Using optimal health investment $x(k_t, i_t)$, we express the equilibrium probability of getting infected as $p_t = p\left[x(k_t, i_t), i_t\right] \equiv p(k_t, i_t)$. For the functions we choose and numerical values we assign to parameters, we can establish that $\partial p_t/\partial k_t > 0$ and $\partial p_t/\partial i_t > 0$. The first result ($\partial p_t/\partial k_t > 0$) is simply an income effect operating through preventive investment. Two opposing effects are embedded in the second result ($\partial p_t/\partial i_t > 0$). Disease prevalence increases directly the probability through the matching process but also tends to lower it by encouraging prevention. This indirect effect is not sufficiently strong to overturn the externality effect.

Substituting the equilibrium probability and prevalence dynamics into the asset market clearing condition leads to

$$k_{t+1} = \frac{p(k_t, i_t)z^I(k_t, i_t) + [1 - p(k_t, i_t)]z^U(k_t, i_t)}{1 - \theta p\left(p(k_t, i_t)\right)}, \tag{23}$$

while disease dynamics evolve according to

$$i_{t+1} = p(k_t, i_t). \tag{24}$$

Equations (23) and (24) describe the general equilibrium of this economy given initial conditions. Given the nonlinearities present in the two equations above, we characterize the dynamics numerically in the next section. There are two types of stationary equilibria: a development trap where output and capital per capita grow at a relatively low rate and infectious diseases are widespread, and a Balanced Growth Path (BGP) along which per capita variables grow at a relatively high rate and infectious diseases disappear.

It is straightforward to derive the steady-states. Define γ as the asymptotic growth rate of capital and output per person. When $i = 0$, the economy-wide saving propensity becomes s^U and equation (23) implies

$$1 + \gamma^H \equiv \frac{\beta}{1+\beta}(1-\alpha)A. \tag{25}$$

In the quantitative exercise below $1 + \gamma^H$ is always larger than one, ensuring sustained growth. When, on the other hand, $i = 1$ the economy's saving rate equals s^I. Hence (23) implies that long-run growth is

$$1 + \gamma^L \equiv \frac{\beta\phi^2}{1+\beta\phi^2}(1-\alpha)A. \tag{26}$$

Clearly the two growth rates γ^H and γ^L differ only because $\phi < 1$. It is through adult mortality alone that diseases impact long-run growth. Morbidity factors matter only for convergence dynamics either by affecting savings directly (for θ) or indirectly (via x for δ).

Note that γ^L is zero if $(1-\alpha)As^I \leq 1$, strictly positive otherwise. In the former case, the steady-state level of capital per effective labor is $k^* = bs^I/[1-(1-\alpha)s^I A]$.

We rely on quantitative methods to identify how transition to the two BGPs is shaped by economic and disease conditions. First, we establish the dynamic properties of a benchmark model. Then we apply our theory to sub-Saharan Africa (SSA) to examine whether, as some suggest, infectious diseases contribute to its economic underdevelopment.

We implement the following identification strategy. The model is first calibrated to pre-HIV SSA using malaria as the focal disease. Then it is recalibrated to post-HIV SSA. The malaria case serves as a benchmark illustrating the type of dynamics an infectious disease can induce. The HIV-malaria scenario digs deeper into SSA's disease burden and highlights the enormity of health challenges when there are disease complementarities.

3. Calibration

Table 1 presents the parameter values for different scenarios. Let us start with parameters that are unrelated to infectious diseases. The model features overlapping generations of agents who potentially live for two periods. To choose the length of one period, we use data on life expectancy at age 15 (LE15) for the U.S.. Life tables in the World Health Statistics 2008 attribute 63 years of LE15 in year 2000. This implies 31.5 years for each period or generation.

We assign a value of $0.99^{31.5\times 4}$ to the discount factor (β), that is, 0.99 per quarter which is standard in the real business cycle literature. We set the elasticity parameter $\sigma = 1$ in the utility function (log preferences). The production function has three parameters: the usual total factor productivity parameter A, output elasticity of capital α, and the productivity term b. We normalize $b = 1$ to ensure that consumption levels are bounded above one and, as a consequence, utility when alive remains positive. We set $\alpha = 0.67$. We are then looking at a broad concept of capital that includes physical, human and organizational capital. The value for A, in turn, is chosen to reproduce an annual long-run growth rate of 1.8% in the low-prevalence steady state. This is the average growth rate of GDP per capita between 1990 and 2003 for OECD nations as reported in UNDP (2005). Hence A is chosen such that $s^U(1-\alpha)A = 1.018^{31.5}$, that is, $A = 24.18$.

The remaining parameters are disease related. Calibration of these depends on three scenarios: malaria in pre-HIV SSA, the HIV/AIDS case, and HIV/AIDS in a highly malaria infected economy.

3.1. Malaria

Malaria is transmitted by the *Anopheles* mosquito that carries the plasmodium parasite from infected to healthy people. For this we need to consider the frequency of mosquito bites and the probability that a bite by an infective mosquito transmits the disease. According to the epidemiological literature, these numbers depend on the mosquito type. With less efficient vectors, human bites per mosquito ranges from 0.2 to 0.5 per day, while the ratio of mosquitoes to humans is about 2.[10] This implies between 0.4 and 1 mosquito bites per human per day. SSA is, however, widely infested with the immensely efficient *Anopheles gambiae* which is capable of 158-174 bites per human per night (Molineaux and Gramiccia 1980, Gallup and Sachs 2001).

[10] This literature is summarized in Ruan *et al.* (2008).

Another important factor in malaria epidemiology is the progressive acquisition of immunity. The evidence on this in low and unstable transmission areas is fragmented. In high transmission areas, prevalence peaks at a young age and declines sharply thereafter. Gu *et al.*'s (2003) review the literature suggests that susceptibility to malaria declines by 66% after 20 exposures.

Finally, we refer to Ruan *et al.* (2008) for estimates on the transmission probability. If a vector bites an infected human, the probability that the mosquito becomes a carrier of the disease is around 50%. In turn, when an infective mosquito bites a non-infected human, the probability of transmission is also about 50%. Ruan *et al.* also report that the recovery rate from malaria among infected individuals is between 0.01-0.05 per day.

With these estimates in mind, we assign values to the disease parameters. The general strategy is to choose values that minimize the bias towards poverty trap dynamics. In the language of our model, an encounter between infected and susceptible individuals is a match consisting of two people and a mosquito vector. For malaria transmission in this encounter, a non-infective mosquito has to take a blood meal from an infected human, become a carrier of the disease, and finally transmit the parasite when biting the susceptible person. We assume that the mosquito dies after the encounter.

In principle, we could equate a (transmission probability in the absence of prevention) to the product of the two human-mosquito-human transmission probabilities, $0.5 \times 0.5 = 0.25$. But disease transmission in the aggregate also depends on the recovery rate of humans, 0.03 per day on average. Given that less efficient mosquitoes bite individuals around 0.35 times a day and there are 2 vectors per human, we can argue that the there is an encounter at least every $1/(0.35 \times 2) = 1.4$ days. Hence, we pick $a = 0.25 \times (1 - 1.4 \times 0.03) = 0.240$. The number of matches is set so as to limit the number of expected exposures to the disease to 20. This is done to take into account the possibility of acquired immunity. Hence we set $\mu_1 + \mu_2 = 82$ implying that an individual will be exposed to the parasite $82 \times 0.240 = 20$ times on average. Since mosquitoes do not discriminate by age, we assign $\mu_1 = \mu_2 = 41$.

Estimates of the quality-of-life impact of malaria, δ, come from the disability weights computed for the Burden of Disease Project by the WHO. Disability weights are a scaling factor that ranges from zero (fully healthy) to one (worst possible health state). They are derived from patient survey data on subjective valuations of the impact of a disease. WHO (2008) offers a disability weight for malaria of 0.19. We then assign a value of 0.81 to δ.

Labor efficiency loss due to malaria are hard to measure directly because workers' health and productivity are hard to observe.[11] Based on Bleakley's (2003) analysis of the impact of malaria in the U.S. South we set $\theta = 0.9$.

The other cost imposed by malaria is premature mortality. In our model, ϕ represents the survival probability of infected agents. This parameter is key for agents' saving decisions and, therefore, growth. This leads us to consider that ϕ should reflect actual mortality experience from all diseases, not infectious disease alone. We choose the value of ϕ that delivers, in the BGP where everyone is infected, a life expectancy of 49.5 years. This corresponds to the average life expectancy at age 15 in SSA in 1990.[12] Assuming that when people die prematurely they do so at the beginning of their second period and that the size of each generation is one, this pins down the value of ϕ at 0.57.

The last parameter we need to calibrate is q, the productivity of the prevention technology. Preven-

[11] Weil's (2007) finding that cross-country health differences do not explain a significant portion of income differences is based on the effect of morbidity alone in partial equilibrium. A positive impact of health on income is to be expected in this case but our analysis shows it is not the more substantive margin: morbidity at best affects the income level, not its growth rate. If we were not restricted to specific infectious diseases, Weil's work would offer an alternative way to calibrate productivity loss from ill health more generally.

[12] We exclude from SSA the African horn because the incidence of malaria and HIV/AIDS is negligible in this region.

tion devices against malaria include mosquito coils and repellents, aerosol sprays, and most importantly bed nets. Here we concentrate on insecticide-treated bed nets, the most commonly advocated tool. After reviewing the literature on field trials Choi *et al.* (1995) conclude that the use of treated bed nets decreases malaria incidence by approximately 50%. Treated bednets cost around $8 in 2005 US dollars and maintain efficacy for two years (Becker-Dreps *et al.* 2009). According to U.N. statistics available online, nominal GDP per capita in 2005 in SSA was $\$1,063$. Using 5% as discount factor, a biannual preventive investment in bed nets represents 0.4% of national income. To use this information for calibrating q, we take as the economy's income the minimum steady-state level attainable in the malaria case, which equals 7.144. This is consistent with our general strategy of minimizing bias towards the poverty trap in that we assign to q a value that maximizes the productivity of prevention. A reduction in the probability of disease transmission of 50% when 0.4% of income is invested requires that $q = 0.0286$.

3.2. HIV/AIDS

The transmission mechanism of the HIV virus is different from the malaria parasite. The HIV virus enters a susceptible person's body through four main channels: sexual intercourse with an infected individual, intravenous injection using unclean shared needles, blood transfusion, and mother-to-child transmission. The main channel of HIV transmission in Africa has been unsafe sex (Schmid *et al.* 2004) so we focus on evidence relating to sexual intercourse among heterosexual couples.

Consistent with previous work, Wawer *et al.* (2005) obtain a mean coital frequency of $8-10$ per month in Uganda. They also cite previously published work that finds transmission probabilities per coital act are between 0.0001 and 0.1, with an average value of about 0.0012. Importantly, in studies carried out in SSA, reported condom use was relatively low even when condoms were offered free of charge. Based on this information we assign a value of $3,402$ to the total number of encounters in each period ($\mu_1 + \mu_2$) obtained by multiplying the mid point estimate of monthly coital acts and the number of months in each period. We choose $a = 0.005$ by incorporating a small upward correction to the mean transmission probability. Instead of taking the sample average, we choose a slightly larger value (well below the mid point estimate) since the sample average is based on both unprotected and protected sexual intercourse whereas only the first is relevant for a.

For malaria, the distinction between inter- and intragenerational matches was not relevant. For HIV, however, it may be since the majority of sexual encounters occur among people of similar age groups. Hallet *et al.* (2007) find that in approximately 25% of partnerships between men and women in Zimbabwe the age difference was 10 years or more, in line with other estimates that the authors reference. Hence we assume that 25% of encounters are intergenerational which implies that $\mu_1 = 851$ and $\mu_2 = 2,551$.

For quality-of-life effect of AIDS, we use again the disability weights computed by the WHO. These are 0.135 for people who are HIV+ but have not developed the illness, and 0.505 for individuals with AIDS. We choose an intermediate value 0.3 for δ. As with malaria, measures of the effect of HIV/AIDS on labor efficiency in SSA are scarce. One of the few studies is Fox *et al.* (2004) that looks at the impact of the disease on the productivity of commercial agriculture workers in Kenya. These authors estimate that tea pluckers who died or retired due to AIDS earned about 17% less in the two years previous to termination. We therefore set $\theta = 0.17$. Following the approach discussed above, we calibrate the survival parameter ϕ to LE15 in 2006. For that year, LE15 in SSA was 46 years, implying $\phi = 0.46$.

On prevention, we look at condom use, "a critical element in a comprehensive, effective and sustain-

able approach to HIV prevention and treatment" according to UNAIDS. The effectiveness of condoms in reducing heterosexual transmission of HIV is around 90% (Davis and Weller 1999, Pinkertona and Abramson 1997). This means that the probability of transmission with prevention is about 10 times smaller than without it. Another relevant statistic is the cost of prevention. An estimate of the international price of condoms is \$0.025 (Shelton and Johnston 2001) although this is a lower bound that does not include distribution costs or cost of information campaigns. With an average of 108 coital acts per year, the total annual cost is \$2.7, about 0.4% of Africa's 2001 current per capita income. Following the same strategy as in the case of malaria, q has to be 0.0015 to achieve a reduction of 90% in the infection transmission probability when 0.4% of the minimum steady-state income level achievable in the HIV case (3.467) is invested in prevention.

3.3. Dual infection: HIV and Malaria

Finally, to illustrate how disease complementarity affects the spread of a new infection such as HIV, we use data on the impact of malaria on HIV transmission. A high incidence of malaria can facilitate the spread of HIV and *vice versa*. Abu-Raddad *et al.* (2006) review the evidence and report that the probability of coital transmission of the HIV virus is 2.45 times larger among malaria patients. During malarial infection, however, sexual activity reduces by about 10%. Accordingly we revise the values for a and the number of encounters. The former parameter in the HIV case when malaria is widespread among the population becomes $0.005 \times 2.45 = 0.01225$ while the latter becomes $3,402 \times (1 - 0.1)$ with $\mu_1 = 765$ and $\mu_2 = 2,296$. The remaining parameters are the same.

The effect of disease complementarity is clear from these parameter choices alone. Consider the disease multiplier in equation (5). The value of ν in the malaria case is 107 implying that one infected person is capable of causing 107 new malarial infections under no prevention and low prevalence of the disease. For HIV/AIDS, ν is about 59, that is, an HIV+ person is capable of causing 59 additional cases whether directly through his own sexual activities or indirectly through subsequent rounds of infection. For dual infection in HIV and malaria, however, ν is 273. This has to be interpreted now as the number of cumulative malarial *and* HIV infections that are caused by a malarial *or* HIV+ person. This is higher than the sum of the only-malaria and only-HIV multipliers: malaria sufferers have less resistance to the HIV virus and HIV+ individuals with compromised immune systems are more susceptible to malaria. In fact this value of ν is an underestimate. Higher prevalence rates amplify disease complementarities due to the exponential nature of disease transmission which the linear approximation of equation (5) abstracts from.

4. Dynamics

4.1. Malaria

We first analyze the dynamic behavior of the economy using malaria as the benchmark disease. Recall that the general equilibrium is described by the pair of difference equations (23) and (24) and the initial conditions (k_0, i_0). Figure 2 illustrates the phase portrait for the parameter values for malaria in Table 1. It plots the prevalence rate i_t against capital per effective unit of labor k_t.

The $x(k_t, i_t) = 0$ schedule (dotted line) represents combinations of (k_t, i_t) for which the optimal decision is not to invest in prevention. The same decision is also optimal in the area to the left of $x(k_t, i_t) = 0$ while to its right prevention is positive. The $x(k_t, i_t) = 0$ locus has a particular shape composed of two different pieces that show two different values of i_t for each k_t because of the way prevalence and income affect incentives. For low levels of disease prevalence ($i_t \to 0$), the

risk of catching an infection is so low that prevention is not necessary. At high levels of disease prevalence ($i_t \to 1$), in contrast, the productivity of prevention becomes vanishingly small as the disease externality from sequential matching outweighs the benefits from prevention.

The $\triangle k_t = 0$ locus on Figure 2 comes from imposing $k_{t+1} = k_t$ on equation (23). Capital per effective unit of labor declines over time above this locus and vice versa. The $\Delta k_t = 0$ line more or less coincides with the $x(k_t, i_t) = 0$ curve to the right of point E. Although we obtain this result in all scenarios presented in the paper it is not a general result and depends on parameter values. For $q = 1$ and $\mu = 2$, for example, the $\Delta k_t = 0$ schedule would be located well below the $x(k_t, i_t) = 0$ curve to the right of a point E. The locus is not defined for low values of k_t since such values are precluded by $b > 0$.

Note the parabolic shape of the $\triangle k_t = 0$ locus: the same prevalence rate can be associated with both high and low levels of capital per effective worker. This results from a tension between two effects. Diseases have a negative effect on capital accumulation via their effect on mortality (which lowers incentive to save) and productivity (which lowers ability to save). This is what the numerator on the right-hand side of equation (23) represents. But diseases can also have a positive effect in general equilibrium. When the prevalence rate goes up, the labor force becomes more debilitated and less effective (denominator on the right-hand side of (23)). The relative scarcity of efficiency labor raises its return which may be high enough to actually raise saving and investment per effective unit of labor.

This positive effect dominates at a relatively large capital intensity. To see this, set $x = 0$ since $\Delta k_t = 0$ coincides with the zero investment locus. The $\Delta k_t = 0$ locus gives steady-state values of k for exogenous values of i. Rearranging terms, this locus is

$$\left\{p(i)s^I(1-\theta) + [1-p(i)]\, s^U\right\}\left(\frac{1}{1-\theta i}\right)\frac{w(k)}{k} = 1.$$

The first term on the left-hand side of this expression is the effect of diseases on capital accumulation: as i decreases, more investment comes from the higher saving propensity healthy individuals. The second term on the left-hand side is the capital dilution effect: a decrease in i increases the efficiency supply of labor which dilutes capital intensity (for a given aggregate capital stock). Since the $\Delta k_t = 0$ line is parabolic, for any i there may exist two steady-state values k_1 and $k_2 > k_1$. At k_1, $\partial k/\partial i < 0$ while $\partial k/\partial i > 0$ at k_2. In other words, the capital accumulation effect dominates at lower values of k while the dilution effect dominates at relatively higher values.[13]

To completely characterize dynamics we now turn to the third locus given by the downward sloping line, $\triangle i_t = 0$, defined by

$$i_t = p(k_t, i_t), \tag{27}$$

along which the prevalence rate remains constant. It is defined wherever $x_t > 0$ with the prevention rate decreasing above the curve and increasing below it. Above the $x_t = 0$ schedule preventive investment is zero which implies the prevalence rate is always rising since $\nu > 1$.

Figure 2 shows multiple steady states. There are two poverty traps with zero growth, one stable (PT) and the other unstable (UPT). There also exists a stable Balanced Growth Path (BGP) along which the economy grows at a positive rate. Vector fields indicate that PT is a sink while UPT is a

[13] The possibility that more adverse disease conditions can actually improve economic conditions is not novel to our model. It echoes historical accounts of how the Black Death pandemic in 14th century Europe may have left its survivors better-off by easing population pressure from agriculture. Young's (2005) analysis of the economic consequences of Africa's AIDS epidemic follows a similar argument as does the combined effect of several other infectious diseases on life expectancy and growth in Acemoglu and Johnson (2007).

saddle-point. Since both the initial prevalence rate i_0 and the initial capital per efficiency labor k_0 are pre-determined, PT is asymptotically stable but UPT is not. In particular, sequences of (k_t, i_t) which do not start exactly on the saddle-arm (not shown) that leads from the left up to UPT converge either to PT or diverge to a sustained growth path along which infectious diseases disappear asymptotically. The saddle path therefore acts as a threshold until it meets the $x = 0$ locus, at which point, the continuation of that locus becomes the effective threshold.

Transition to BGP can exhibit interesting dynamics. In Figure 2, the trajectory starting from point M initially shows slow growth and rising disease prevalence. The slow growth comes from the effect of disease on mortality and labor productivity as well as lower saving due to prevention. But prevention ultimately overcomes infectious diseases. The prevalence rate peaks and then declines monotonically as the economy takes-off into sustained growth, its growth rate converging asymptotically to γ^H. For a trajectory starting at point N, in contrast, growth is steady as the economy converges to BGP and diseases abate.

It is instructive to understand how disease ecology and costs interact with initial conditions to shape the growth trajectory. Ecology determines susceptibility to infection and depends on the number of encounters (μ) and the probability of contracting a disease in each such encounter (a, q). As we decrease μ, q or a, the transmission probability falls and the state space within which people invest in prevention expands. This facilitates convergence towards BGP making a poverty trap less likely.

For sufficiently large values of these parameters, on the other hand, the model generates a poverty trap that differs from those commonly discussed in the literature (Azariadis and Stachurski, 2005). Table 2 reports numerical results that facilitate comparison across different disease scenarios. For the malaria benchmark, the $x = 0$ and $\triangle k = 0$ schedules intersect $i = 1$ at $k = 171,678$. This means that, in principle, a sufficiently rich country never ends up in the poverty trap, a result similar to the existing literature. But it requires the country to be $581,257$ times richer than a country at PT, an income gap far in excess of what we observe in the data.

When we pick $a = 1$ (second column in Table 2), the $x = 0$ and $\triangle k = 0$ lines asymptote at $i < 1$.[14] This means that for large a (equivalently large q), the model produces a trap in which it is not lack of income that predisposes countries towards PT. No matter how high income per effective worker is, there is always a high enough prevalence rate that triggers implosion to the poverty trap. The fact that such a poverty trap is empirically plausible is also a novel contribution on the quantitative side. While Graham and Temple (2006) argue that the variable-returns-to-scale poverty trap model can account for up to a quarter of cross-country income variation, Caucutt and Kumar's (2008) analysis of several other poverty trap models finds them lacking in explaining African underdevelopment. Neither of these quantitative studies, however, look specifically at the disease and development explanation for Africa.

Indeed key to understanding the new poverty trap dynamics in this model is recognizing that the return to prevention declines rapidly with μ_1 and μ_2. For instance, the probability of being infected after $\mu_1 + \mu_2 = 5$ matches becomes 1 if the probability of getting infected from a single match ($i_t \pi_t$) exceeds 50%. When $\mu_1 + \mu_2 = 10$, it reaches 1 even if the probability of getting infected from a single match drops to 30%. What drives non-ergodicity is this negative externality of communicable diseases.

Finally, the effect of disease costs depends on which parameter we look at. As morbidity costs increase (higher θ, lower δ), it elicits stronger preventive behavior which makes PT a less likely equilibrium outcome. Higher mortality risk (lower ϕ), however, makes a trap more probable for given initial conditions. The model's predictions are actually sensitive to ϕ because it determines the rate

[14] In particular, our simulations show that for i sufficiently close to 1 the economy converges to PT for any k that GAUSS can handle, that is, for $k \leq 10^{308}$.

at which infected individuals discount the future and, therefore, has a significant effect on their saving propensity. In the malaria case, when the survival probability is at least 0.72, the saving rate is high enough to sustain output growth and the poverty trap vanishes.

To summarize, there are two stable balanced growth paths for disease parameters calibrated to SSA's malaria burden. One path is characterized by a low growth rate (which equals zero if the survival probability is sufficiently small and prevention investment is sufficiently effective) and the other by a relatively high growth rate. Second, unless the probability of contagion can be reduced sufficiently, any economy regardless of its income level can diverge towards the low-growth attractor (when it exists) if prevalence becomes sufficiently high due to an exogenous epidemic shock for instance.

4.2. HIV/AIDS

It is important to understand whether other major infectious diseases, HIV/AIDS for SSA for example, can generate effects as powerful as malaria.

The phase portrait for the HIV/AIDS parameter values is qualitatively similar to before in that it shows non-ergodicity of growth paths. For this reason it has been omitted. Instead Table 2 reports some quantitative results. The prevalence rates when $k = 0.00$ (for computational reasons a value sufficiently close to zero) along the $x = 0$ schedule are 0.039 for malaria (first column) and 0.181 for HIV/AIDS (third column). In addition, the value of k at which the zero prevention locus intersects $i = 1$ is much higher under malaria than under HIV/AIDS. Consequently the state space that leads to BGP under HIV/AIDS is significantly larger.

This occurs even though the three costs that the disease imposes on infected individuals are *larger* with HIV/AIDS. Dominating these costs is the efficacy of HIV/AIDS prevention. Recall that the probabilities of contagion without prevention are 0.240 and 0.005 for malaria and HIV, respectively. For the same preventive investment, HIV susceptibility falls by 90% compared to 50% for malaria. In other words, HIV is more difficult to spread than malaria and easier to avoid. For example, Table 2 reports that if $\phi = 0.82$ it takes 21 generations for a highly HIV-infected economy that starts from the PT to lower prevalence rates and register rapid growth, compared to 53 for malaria.

As we argued in the calibration section, the distinction between intra- and intergenerational encounters is important for HIV. It can be shown that if we decrease the number of encounters between old and young agents (μ_1), increasing by the same magnitude those among the young (μ_2), the state space leading to BGP expands. That is, the intergenerational transmission dominates the whole disease transmission process. This is not surprising since HIV is caused by direct human-to-human contact: if μ_1 were zero, the prevalence rate would ultimately drop to zero when all members of the infective cohort died from natural aging and the negative effect of the disease would last for only a single generation.

4.3. Dual Infection

Finally, consider how the HIV epidemic interacts with the endemic nature of malaria in SSA. Here too the phase diagram is omitted since it is qualitatively similar to that for malaria. Looking at Table 2 (fourth column) and following the same logic as in the previous section, it becomes clear that the interaction effect gives rise to the worst outcome of the three. The area that leads to BGP now shrinks considerably and the model implies that HIV will spread and perpetuate more in areas with a high malaria incidence. In fact, even if the survival rate were higher, equal to 0.82, an economy located at PT would take an inordinately long time (100 generations) before prevalence rates begin to fall.

Complementarity between malaria and HIV reinforce the health costs. Abu-Raddad *et al.*, (2006) estimate that the interaction between malaria and HIV has been responsible for 8,500 excess HIV infections and 980,000 excess malaria episodes in Kenya. The authors note that such co-infection, in turn, made it easier for malaria to spread to areas with high HIV prevalence. This type of complementarity extends to other infectious diseases. Sub-Sahara Africans are four to five times more likely than Americans to become infected with HIV for a given unprotected sexual relationship with an HIV+ partner. Oster (2005) attributes this difference to a higher incidence of untreated bacterial STDs in SSA. Open sores from these STDs increase the transmission efficiency of the HIV virus.[15]

5. Alternative Scenarios

Understanding how the economy's dynamic behavior depends on alternative parameter values can inform us about the applicability of the model to other countries where the cost of infectious diseases may not be as severe as in SSA. We briefly return to our benchmark malaria case and consider alternative values of two key parameters, a and ϕ. The former guides the virulence of an infectious disease while the latter shapes its long term effect.

As a falls, preventive investment becomes more efficient. When a falls sufficiently, diseases can be avoided at relatively low cost and the saving generated even at low incomes is enough to maintain a growing capital stock. For example, when a drops from 0.240 to 0.071 (Table 2) the poverty trap equilibrium disappears and instead all economies converge to a unique balanced growth path.

The model's predictions are also sensitive to the survival probability ϕ which determines the average saving-investment propensity and, therefore, growth in the development trap. For our benchmark calibration this growth rate was zero. When ϕ increases the development trap vanishes and the economy either converges to a high-growth path or to a growth trap in which the economy grows at a sustained positive rate but given the "income neutrality" of the trap it does not escape infectious diseases. This never happens for the malaria case, given the other parameter values. But it can when a is concurrently higher. For $\phi = 0.82$ (LE15 of 57), for example, if a is sufficiently close to 1 the poverty trap turns into a growth trap.

Now imagine these parameter changes to be exogenous health improvements occuring in an impoverished country. Figure 3 shows the growth of income per worker per generation (%) against generational time. All three paths start at $k = 0.2$, marginally below the poverty trap capital stock. The time path labeled $i_0 = 0$ is for a poor economy without infectious diseases. This economy converges to the balanced (annual) growth rate of 1.8% and takes about six generations to get there.

The intermediate path labeled $i_0 = 1, \phi = 0.82$ pertains to an economy that starts out close to PT with high prevalence rates. An exogenous medical breakthrough, e.g., one that makes malaria treatments highly effective, increases the survival rate dramatically from 0.57 to 0.82. The time path illustrates that this economy initially grows relatively rapidly before settling down to a steady (annual) growth rate of 0.8% which, of course, is worse than the one enjoyed by the first economy because of the growth trap.

The lowest time path labeled $i_0 = 1, a = 0.098$ illustrates the consequences of another breakthrough that makes it relatively easier to avoid infection. This can be a medical innovation that improves

[15] Institutions are presumably relevant too. Our work shows that the quality of public health systems and medical technology (q), rather than general institutional capability and aggregate productivity (A), are relevant for solving the twin problems of health and poverty. Moreover, as the WHO's experience with battling malaria in Nigeria's Garki district demonstrates (see Molineuax and Gramiccia 1980), existing medical technologies and best practice q's may be inadequate for SSA's disease problem.

resistance to malaria by more than twice (e.g. vaccines widely available free of charge) or a large-scale eradication of disease vectors (e.g. due to public health innovations). This change eliminates the poverty trap and triggers a growth take-off. What Figure 3 shows is that this take-off is preceded by declining growth lasting six generations. This suggests that Acemoglu and Johnson's (2007) finding, that exogenous health improvements did not lead to faster growth over the past two generations (their sample spans 1940–2000), may be more general than the mechanism they emphasize. Here the slowdown occurs because a lower a initially creates stronger incentives for health investment, but these stronger incentives do not generate prevention investment until k is large enough. More importantly, when prevention investment starts flowing, it dominates other types of (growth promoting) investment. Returns to health investment, during this adjustment phase, are enjoyed mainly in the form of direct welfare gains from longer and healthier lives. These quantity of life gains dominate what Becker *et al.* (2005) call quality of life gains, that is, welfare gains in the form of higher future consumption through investment in capital.

These numerical experiments reinforce what we saw before, that infectious diseases impose large economic costs by causing development *or* growth traps. Even without multiple attractors, the cost is substantial: it takes six generations for the third economy in Figure 3 to enjoy greater or similar rates of progress as the first.

6. Conclusions

This paper establishes that poor health due to infectious disease can have a first-order effect on economic growth. By explicitly incorporating disease behavior and prevention in a general equilibrium model of growth, the theory reveals the power of disease externalities. These externalities are the source of a unique and previously unexplored poverty trap that can also impact relatively wealthy nations. In other words, the disease-development trap in our model is distinct from traps typically tied to history and initial conditions in the existing literature.

A calibration of the model to sub-Saharan Africa's disease burden shows that multiplicity of growth paths is empirically plausible. Specifically, quantitative results based on malaria and HIV in sub-Saharan Africa show that while each disease has a large negative impact on development (especially malaria), the challenge of tackling multiple diseases is a formidable one due to disease complementarities. Even when all economies converge to the same long run growth rate, countries that are exposed to infectious diseases take much longer to experience robust growth.

Our work offers theoretical foundation to a predominantly empirical health and development literature. This is necessary on the macroeconomic side where the evidence on the relationship between health and income is mixed. Existing models either assume exogenous health or *ad hoc* disease transmission mechanisms that are not tied to microfoundations. A natural next step is to explore the policy implications of this model and shed light on the ongoing debate about the effectiveness of health aid and alternative interventions.

The model also offers several testable predictions that empiricists can exploit. First, it implies that while mortality has growth effects, morbidity at best has a level effect on income. Second, both mortality and morbidity costs are important drivers of saving and prevention behavior in diseased environments. Third, morbidity can generate dilution effects on capital intensity that are stronger at relatively higher levels of development. Perhaps most importantly, the theory suggests that health can generate nonlinearities in the growth process.

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Table 1: Benchmark Parameter Values

Non-disease Parameters			**Malaria**	**HIV/AIDS**	**HIV/AIDS with Malaria**
β	$0.99^{(31.5\times4)}$	ϕ	0.57	0.46	0.46
σ	1	θ	0.1	0.17	0.17
b	1	δ	0.81	0.7	0.7
α	0.67	μ_1	41	851	765
g_y	0.018	μ_2	41	2551	2296
		q	0.0286	0.0015	0.0015
		a	0.240	0.005	0.01225

Table 2: Key Numerical Results

	Malaria	**a = 1**	**HIV/AIDS**	**HIV/AIDS with Malaria**
Value of k at PT	0.254	0.254	0.102	0.102
Value of i for $k = 0.00$ on the $x = 0$ line*	0.039	0.002	0.181	0.033
Value of k for $i = 1$ on the $x = 0$ line*	171,678	∞	65	1,396,932,300
Value of ϕ for which PT becomes growth trap	*none*	0.72	*none*	*none*
Value of ϕ for which multiplicity vanishes	0.72	*none*	0.72	0.72
# gens. till i falls starting at PT for $\phi = 0.82$	53	*none*	21	100
Value of a for which multiplicity vanishes	0.071	—	0.003	0.003

*Refers to the upper segment of the $x = 0$ locus in Figure 2.

Figure 1: Timeline of Events

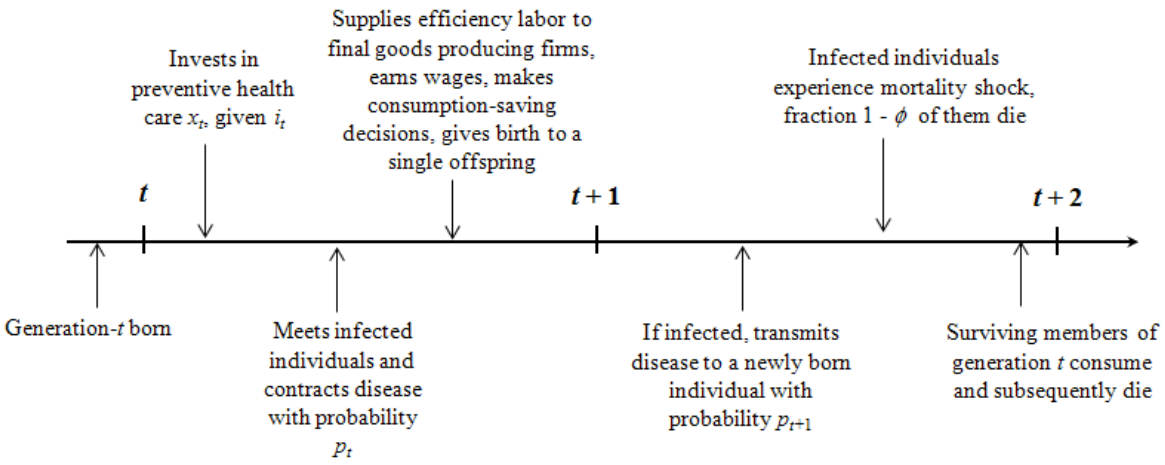

Figure 2: Phase Diagram for the Malaria Case

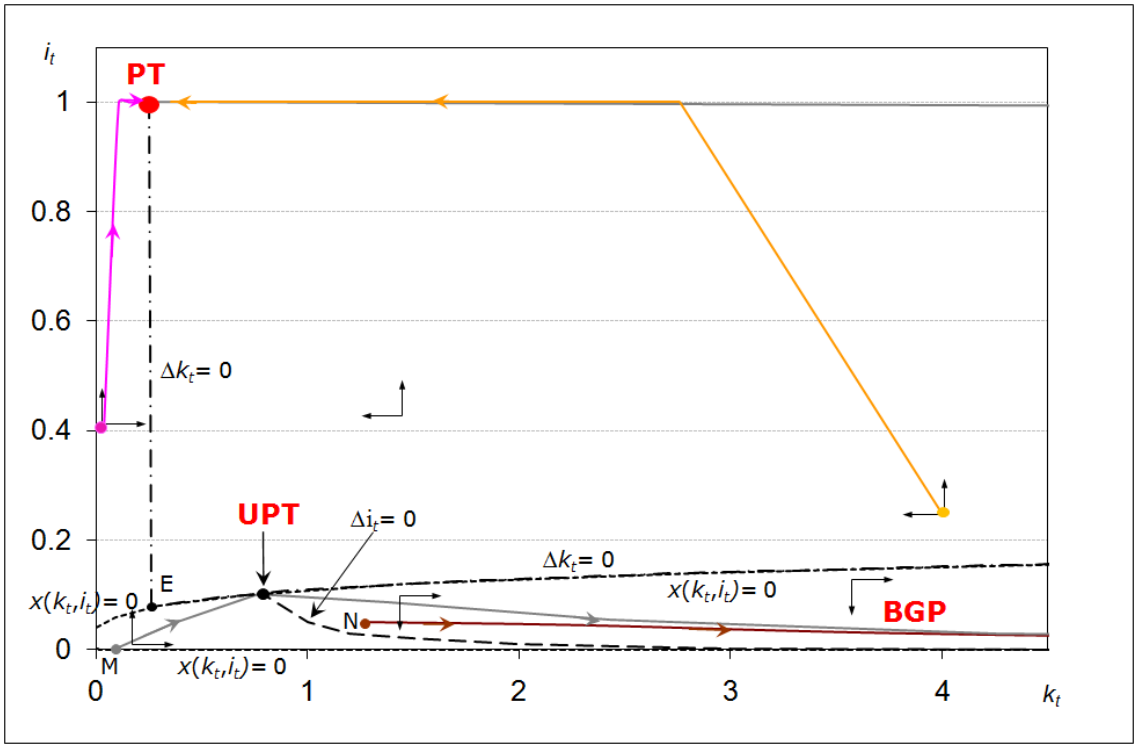

Figure 3: Output Growth per Generation