Diseases, infection dynamics, and development

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ABSTRACT

The relationship between health and development is a subject of ongoing debate. This paper contributes to the debate by proposing a general equilibrium theory of infectious disease transmission, prevention investment, and rational behavior. Diseases cause premature death, labor productivity loss and lower quality of life. Higher disease prevalence lowers the average saving-investment propensity. The model offers two insights. First, infectious disease can plausibly generate an unconventional growth trap where income alone cannot push an economy out of underdevelopment. Second, even when countries converge to the same balanced growth path, the disease ecology significantly impairs 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, we incorporate rational disease behavior into a growth model.[1] 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 may cause premature death and lower the productivity and quality-of-life of the infected. The mortality effect makes infected individuals less inclined to save while morbidity makes them less able to do so.[2] Preventive investment can avert infection but its efficiency declines rapidly with prevalence due to disease externalities.

Two types of long-run growth equilibria 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 a particular country follows.

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 foreign 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).

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[1] Communicable diseases account for 55% of deaths in developing countries, 14% in developed countries (WHO, 2002). Analyzing this disease burden through rational behavior is vital so as not to paint an incorrect picture of disease dynamics or the effectiveness of public health interventions (Geoffard and Philipson, 1996).

[2] 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. (2008) for cross-country evidence.

Along the higher growth path, the causality between income and health runs in both directions: higher income assists prevention, lowers the incidence of communicable disease and generates stronger incentives for growth. This pattern is 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 alternatives that may be able to raise income growth more immediately.

Despite compelling microeconomic evidence that health is an essential component of economic well-being (see, e.g. 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 per capita. They argue that the increase in population resulting from better health outweighs the positive productivity effects. Weil (2007) uses microeconomic evidence 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 only 10%. 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 health and growth relationship.

This paper is related to several theoretical contributions incorporating mortality into growth models. Among others, Kalemli-Ozcan (2002), Chakraborty (2004), Doepke (2005) and Soares (2005) variously consider the effect of child and adult mortality on fertility, human capital, demographic change and economic growth. Theoretical work on infectious disease and growth is more limited. Improved disease resistance from better nutrition is used to explain the escape from Malthusian stagnation to modern economic growth in Birchenall (2007) while the focus of Momota et al.'s (2005) work is the possibility of disease cycles. Neither of these papers, however, clearly specify the disease transmission process or the role of prevention.[3]

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.[4] The calibration strategy and parameter choices are discussed in Section 3. Section 4 analyzes sub-Saharan Africa's twin problem 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.[5] 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 quality-of-life effect: the individual derives a utility flow of $\delta u(c)$ instead of $u(c)$ from a consumption bundle c, where $\delta \in (0,1)$. Thirdly, an infected young individual faces the risk of premature death and may not live through his entire old-age.

Young individuals can undertake preventive health investment, x_t, early in life. This takes the form of net food intake, 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.

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[3] Our paper is also related to the Unified Growth Theory proposed in Galor (2005). In the model, a stagnant economy starts enjoying modern economic growth when prevalence rates fall sufficiently due to exogenous improvements in medicine, public health and/or the disease environment.

[4] An *Online Supplementary Material* presents a simpler model with closed form solutions. Results there are more intuitive, but the simpler model generates a standard growth trap. The more general specification of disease transmission here delivers unique growth trap dynamics.

[5] Neither childhood nor child mortality are taken into account. 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 and remains high in developing countries (World Bank, 1993), 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.

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 infected (intergenerational transmission). In the second stage, young adults socialize among themselves with each young person interacting with μ_2 young adults (intragenerational transmission).

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, $i_t \pi(x_t)$. Not all older individuals are infected and not all encounters with infected people result in transmission. Hence, the probability p_t^1 of being infected after the first stage equals

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

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

In the second stage, an infected person does not get infected 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. The probability of getting infected in the second stage for someone who remained uninfected after the first is then

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

The proportion 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

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

It is important to note here that the negative externality associated with 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 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

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

This function satisfies the following properties: $\pi' < 0$, $\pi(0) = a$, $\pi(\infty) = 0$, and $\pi'(0) \to -\infty$ as $q \to 0$. 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.

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

Three features of the disease environment require elaboration. First, although we occasionally refer to *the* infectious disease, we think about communicable 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).

Second, 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. 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). 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 (Jaramillo, 1999). 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. We tag variables by U and I corresponding to uninfected and infected individuals, respectively.

First consider the decisions of an uninfected individual whose health investment has successfully protected him from the disease. This individual's problem is

$$\max_{\{z_t^U\}} \ \{u(c_{1t}^U) + \beta u(c_{2t+1}^U)\}, \quad \beta \in (0,1), \tag{5}$$

$$\text{s.t.:} \quad c_{1t}^U = w_t - x_t - z_t^U \quad \text{and} \quad c_{2t+1}^U = R_{t+1} z_t^U, \tag{6}$$

where z is savings, and w, R and x are given.[6]

An infected person faces a constant probability ϕ of surviving from the disease before reaching old-age. Normalizing utility from death to zero, the maximization problem is now

$$\max_{\{z_t^I\}} \ \{\delta[u(c_{1t}^I) + \beta\phi u(c_{2t+1}^I)]\}, \quad \delta,\phi \in (0,1), \tag{7}$$

$$\text{s.t.:} \quad c_{1t}^I = (1-\theta)w_t - x_t - z_t^I \quad \text{and} \quad c_{2t+1}^I = R_{t+1} z_t^I + \tau_{t+1}. \tag{8}$$

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

$$\tau_{t+1} = \left(\frac{1-\phi}{\phi}\right) R_{t+1} z_t^I. \tag{9}$$

The first-order conditions of the above problems are the familiar Euler equations

$$u'(c_{1t}^U) = \beta R_{t+1} u'(c_{2t+1}^U) \tag{10}$$

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

Hereafter we assume the analytically convenient CRRA utility function

$$u(c) = \frac{c^{1-\sigma}-1}{1-\sigma}, \quad \sigma > 0, \tag{12}$$

which is negative if c is less than one. Since utility from death is zero, we will assume that the aggregate technology is productive enough to ensure that consumption exceeds one.[8]

Saving is invested in capital which is rented to firms and fully depreciates in one period. At $t=0$, the initial old generation owns a stock of capital K_0, and the disease incidence rate is i_0. We summarize the timeline of events in Fig. 1.

2.3. Technology

A continuum of firms operates in perfectly competitive markets to produce the final good using capital (K) and efficiency units of labor (L) according to

$$F(K^i, L^i) = A(K^i)^\alpha (\overline{k}L^i)^{1-\alpha} + bL^i, \tag{13}$$

where A is a constant productivity parameter, and $\overline{k}$ denotes the average capital per effective unit of labor across firms, which represents a learning-by-doing externality.[9] Finally, $b > 0$ captures "natural endowments" such as trees and animals. For b sufficiently large, consumption will be above one, and then utility will never be negative.

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 $w_t = (1-\alpha)Ak_t + b \equiv w(k_t)$, and $R_t = \alpha A \equiv R$.

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[6] x is financed by zero-interest loans from the rest of the world, repaid after the labor market clears.

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

[8] An alternative is $u(c) = c^{1-\sigma}/(1-\sigma)$ with $\sigma \in (0,1)$ which does not entertain negative values. But this specification does not allow for values of $\sigma \geq 1$ that are supported by available estimates (Guvenen, 2006).

[9] The choice of this simple Ak mechanism that accommodates the possibility of endogenous growth is made for tractability. The story generalizes when saving behavior determines growth (in closed or open economies) via innovation and factor accumulation and to exogenous growth in which case the model's predictions will be in terms of income levels instead of growth rates.
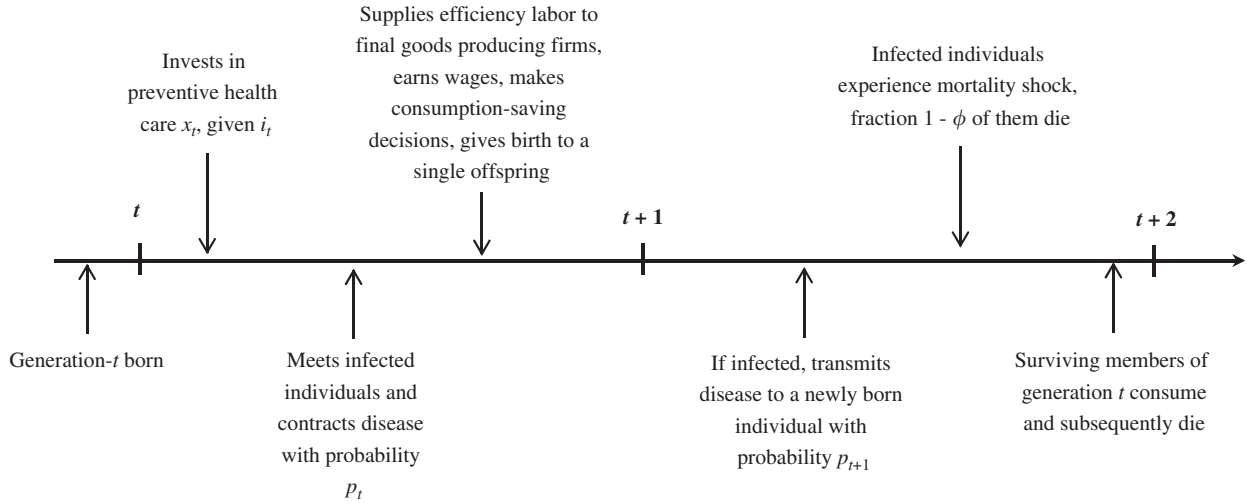
Fig. 1. Timeline of events. This figure outlines the sequence of economic and disease-related events for an individual born at $t-1$ who becomes an active adult in period t.

2.4. Preventive investment decision

Our next task is analyzing privately optimal prevention. Euler conditions (10), (11) and the utility function (12) provide optimal savings z_t^U and z_t^I, which substituted into lifetime utility give the indirect utility functions $V^U(x_t)$ and $V^I(x_t)$ contingent on prices, preventive health investment and disease realizations. Knowing that, at the beginning of period t, individuals choose x_t to maximize expected lifetime utility

$$p_t V^I(x_t) + (1-p_t)V^U(x_t), \tag{14}$$

where $p_t \equiv p(i_t)$ is a young individual's probability of catching the disease, given by (3).[10]

Substituting equilibrium prices and transfers into optimal savings obtains

$$z_t^U = s^U[w(k_t) - x(w_t, i_t)] \equiv z^U(k_t, i_t) \quad \text{and} \tag{15}$$

$$z_t^I = s^I[(1-\theta)w(k_t) - x(w_t, i_t)] \equiv z^I(k_t, i_t), \tag{16}$$

where

$$s^U \equiv \left(\frac{\beta^{1/\sigma} R^{1/\sigma-1}}{1 + \beta^{1/\sigma} R^{1/\sigma-1}} \right), \quad s^I \equiv \left[\frac{\phi(\beta\phi)^{1/\sigma} R^{1/\sigma-1}}{1 + \phi(\beta\phi)^{1/\sigma} R^{1/\sigma-1}} \right]. \tag{17}$$

Evidently $z_t^U > z_t^I$. 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 these expressions into the indirect utility functions leads to

$$V_t^{U*} = \zeta^U \frac{[w(k_t) - x_t]^{1-\sigma}}{1-\sigma} - \frac{1}{1-\sigma} \quad \text{and} \tag{18}$$

$$V_t^{I*} = \zeta^I \frac{[(1-\theta)w(k_t) - x_t]^{1-\sigma}}{1-\sigma} - \frac{\delta}{1-\sigma}, \tag{19}$$

for $\zeta^U \equiv (1-s^U)^{1-\sigma} + \beta R^{1-\sigma}(s^U)^{1-\sigma}$ and $\zeta^I \equiv \delta\phi^\sigma[\{(1-s^I)/[\phi+(1-\phi)s^I]\}^{1-\sigma} + \beta R^{1-\sigma}(s^I)^{1-\sigma}]$.

Finally, we combine the Kuhn–Tucker first order condition for preventive health investment from (14) with equilibrium prices and savings. Noting that individuals do not take into account equilibrium transfers given by (9) when making health investment decisions, we get

$$p_t \zeta^I[(1-\theta)w(k_t) - x_t]^{-\sigma} + (1-p_t)\zeta^U[w(k_t) - x_t]^{-\sigma} \leq -\mu(1 - i_t\pi_t)^{\mu-1}\pi'(x_t)i_t(V_t^{U*} - V_t^{I*}). \tag{20}$$

Two possibilities arise. Optimal health investment is zero as long as its marginal utility cost (the left-hand side of (20)) dominates its marginal benefit (the right-hand side of the same expression), that is, as long as the returns to health investment are negative when $x_t=0$. Intuitively we expect this to occur at levels of low income and high prevalence rates.

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[10] Because preferences can become non-convex with endogenous p, we verify that second order conditions are also satisfied for the parameter values and functional forms we choose later on.

Private actions have a negligible impact on leading a healthy and more productive life in such situations. If, on the other hand, the LHS of (20) at $x_t=0$ is dominated by the RHS, prevention investment will take on strictly positive values.

Rewriting (20) above, the condition for zero preventive investment becomes

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

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 (20) 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 $S_t=p_tz_t^I+(1-p_t)z_t^U$. 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 $p_t = p[x(k_t,i_t),i_t] \equiv p(k_t,i_t)$. 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

$$k_{t+1} = \frac{p(k_t,i_t)z^I(k_t,i_t)+[1-p(k_t,i_t)]z^U(k_t,i_t)}{1-\theta p(p(k_t,i_t))}, \tag{21}$$

while disease dynamics evolve according to

$$i_{t+1} = p(k_t,i_t). \tag{22}$$

Eqs. (21) and (22) 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 Eq. (21) implies

$$1+\gamma^H \equiv \frac{\beta}{1+\beta}(1-\alpha)A. \tag{23}$$

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 (21) implies that long-run growth is

$$1+\gamma^L \equiv \frac{\beta\phi^2}{1+\beta\phi^2}(1-\alpha)A. \tag{24}$$

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 as well 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 $k^* = bs^I/[1-(1-\alpha)s^IA]$.[11]

Further analyses about how transition to the two BGPs is shaped by economic and disease conditions require quantitative methods. A main goal of these exercises is studying whether, as some suggest, infectious diseases contribute to the economic underdevelopment of some economies such as sub-Saharan Africa (SSA). For this purpose, 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 parameter values under different disease scenarios. Consider first parameters unrelated to infectious disease. To choose the length of one period in the model, we use data on U.S. life expectancy at age 15 (LE15). Life tables in

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[11] Note that $b > 0$ is not necessary to obtain the main results in this section. When $b=0$, the low-growth trap still exists.

Table 1
Parameter values.

Non-disease parameters			Malaria	HIV/AIDS	HIV/AIDS with Malaria
β	$0.99^{(31.5 \times 4)}$	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

Parameter values employed for the simulations. The first two columns correspond to parameters unrelated to infectious diseases, while the last four report parameters calibrated to each of the three disease environments considered in Sections 3 and 4.

the World Health Statistics 2008 attribute 63 years of LE15 in year 2000. This implies 31.5 years for each period or generation, and leads us to assign a value of $0.99^{31.5 \times 4}$ to the discount factor (β), that is, 0.99 per quarter.

In the utility function, we set $\sigma = 1$. The parameter b is normalized to 1 to ensure that utility when alive remains non-negative. The chosen value for α is 0.67, reflecting a broad concept of capital. Finally, the parameter A is set equal to 24.18 so as to reproduce in the low-prevalence steady-state an annual long-run growth rate of 1.8%, the average growth rate of GDP per capita between 1990 and 2003 for OECD nations in UNDP (2005).

Remaining parameters are disease related. Their calibration depends on three scenarios: malaria in pre-HIV SSA, the HIV/AIDS case, and HIV/AIDS in a 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. 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. This implies 0.4–1 mosquito bites per human per day. SSA is, however, widely infested with the *Anopheles gambiae* which is capable of 158–174 bites per human per night. 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%. Also important is that the recovery rate from malaria among infected individuals is between 0.01 and 0.05 per day.[12]

With these estimates in mind, the general strategy is to choose values that minimize the bias towards poverty trap dynamics. In the language of our model, a match between an infected and a susceptible individual is intermediated through an initially *non-infective* mosquito. For malaria transmission in this encounter, the mosquito has to take a blood meal from the infected human, become a carrier of the disease, and then transmit the parasite when biting the susceptible person. We assume that the mosquito dies after the encounter.

In principle the transmission probability in the absence of prevention, a, could take on 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, there is an encounter at least every $1/(0.35 \times 2) = 1.4$ days. Hence we choose $a = 0.25 \times (1 - 1.4 \times 0.03) = 0.240$. The number of matches should limit the number of expected exposures to the disease to take into account the possibility of acquired immunity. This is implemented by setting $\mu_1 = \mu_2 = 41$, which means that an individual will be exposed to the parasite $82 \times 0.240 = 20$ times on average, and that mosquitoes do not discriminate by age.[13]

Estimates of the quality-of-life impact of malaria δ come from disability weights in the Burden of Disease Project. A disability weight for a specific disease is a scaling factor that ranges from zero (fully healthy) to one (worst possible health state). It is derived from patient surveys on subjective valuations of the impact of the disease. WHO (2008) calculates a disability weight of 0.19 for malaria which leads us to assign a value of 0.81 to δ.

Labor efficiency loss due to malaria is hard to measure because a worker's health and productivity are hard to observe.[14] Based on Bleakley's (2003) analysis of the impact of malaria in the U.S. South we set $\theta = 0.1$.

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[12] These values were obtained from Ruan et al. (2008), Molineaux and Gramiccia (1980), and Gallup and Sachs (2001).

[13] Gu et al.'s (2003) review of the literature on immunity acquisition reports that susceptibility to malaria declines by 66% after 20 exposures.

[14] If we were not restricted to specific infectious diseases, Weil's (2007) morbidity estimates would offer an alternative way to calibrate productivity loss from ill health more generally.

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. Hence ϕ should reflect the mortality experience from all diseases, not infectious disease alone. We choose the value of ϕ that delivers, in the steady-state where everyone is infected, a life expectancy of 49.5 years. This corresponds to SSA's average life expectancy at age 15 in 1990.[15] 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 needed is q, the productivity of the prevention technology. 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 the 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 implies that $q=0.0286$.

3.2. HIV/AIDS

The main transmission channel of the HIV virus in Africa has been unsafe sex (Schmid et al., 2004). For this reason, evidence related to sexual intercourse among heterosexual couples is used to calibrate the HIV/AIDS case.

Consistent with previous work, Wawer et al. (2005) obtain a mean coital frequency of 8–10 per month in Uganda. They also cite literature that finds transmission probabilities per coital act between 0.0001 and 0.1, with an average value of about 0.0012. Based on this information we assign a value of 3402 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, however, the distinction between inter- and intragenerational matches was not relevant. For HIV it is since the majority of sexual encounters occur among people of similar age groups. Hallett 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. Based on this 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, the disability weights computed by the WHO equal 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, estimates of the effect of HIV/AIDS on labor efficiency in SSA are hard to find, an exception being Fox et al. (2004)'s study of the impact of the disease on commercial agricultural 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, suggesting $\theta = 0.17$. Following the approach discussed above, the survival parameter ϕ is calibrated to the 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 sustainable 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). 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

The third and final case we consider is the presence of both malaria and HIV. Malaria infection (in regions with stable malaria) can facilitate HIV transmission as it has been shown to increase HIV concentration in the blood (WHO, 2004). HIV, in turn, increases susceptibility to malaria due to compromised immunity. In fact, malaria is often the proximate cause of death among HIV+ patients in SSA.

To understand the effects of coinfection, or disease complementarity, ideally one has to consider how the presence of malaria raises susceptibility to HIV and vice versa. This would require a different model since we will have to distinguish between patients suffering from clinical malaria, those who are HIV+ and those who are co-infected. Additionally we will

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[15] The African horn is excluded because the incidence of malaria and HIV/AIDS is negligible in this region.
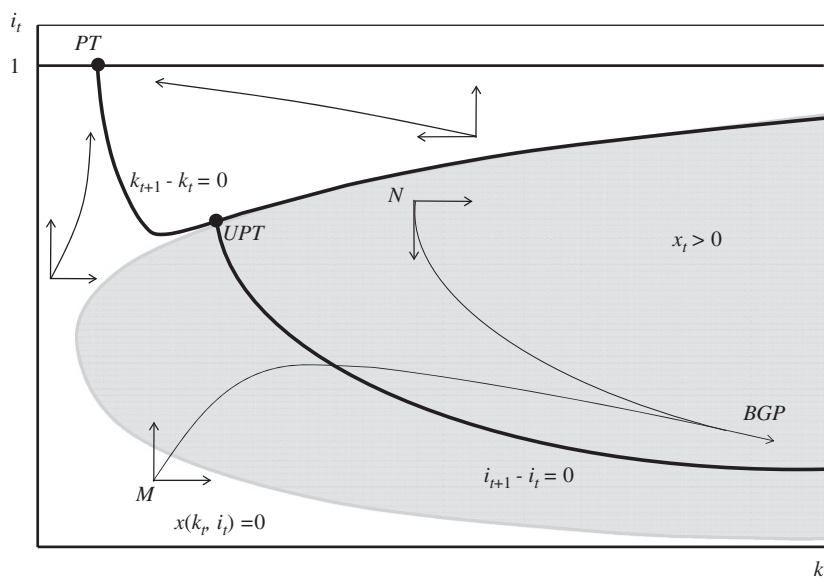
Fig. 2. Phase diagram. The dynamics of capital per effective worker (k) and prevalence rate (i) is qualitatively based on the parameter values for malaria reported in Table 1. Prevention is zero outside the shaded region. There are three stationary sates: the asymptotically stable poverty trap (*PT*) and balanced growth path (*BGP*) and the unstable intermediate trap (*UPT*).

need to calibrate ($a,\mu_1,\mu_2,\phi,\delta$) for each patient type. We adopt a simpler approach and consider the effect of malaria on HIV transmission alone.[16]

Abu-Raddad et al. (2006) 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 $3402 \times (1-0.1)$ with $\mu_1 = 765$ and $\mu_2 = 2296$. The remaining parameters are the same.

The effect of disease complementarity can be seen from these parameter choices alone. Approximate the disease transmission function (3) by $p(i_t) \simeq a\mu_1(1+a\mu_2)i_t \equiv vi_t$ where v is a *disease multiplier*, the cumulative number of cases per primary case in a relatively uninfected population. The value of v in the malaria case is 107: one infected person is capable of causing 107 new malarial infections under no prevention and low prevalence of the disease. For HIV/AIDS, v 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, v 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 and exceeds the sum of the only-malaria and only-HIV multipliers.

4. Dynamics

The economy's dynamic behavior is qualitatively similar for the different sets of parameter values reported in Table 1. The simplest way to understand the model's dynamics is using a phase portrait. Fig. 2 illustrates the phase portrait between the prevalence rate i_t and capital per effective unit of labor k_t.

The $x(k_t,i_t) = 0$ schedule divides the state space into two regions: (k_t,i_t) pairs in the locus shaded region where prevention is positive; and pairs on and below the locus for which the optimal decision is not to invest in prevention. For low levels of disease prevalence ($i_t \to 0$), the risk of getting infected is so low that prevention is not necessary. At high levels of disease prevalence ($i_t \to 1$), in contrast, prevention becomes vanishingly less productive as the disease externality from sequential matching outweighs the benefits from prevention.

The $\Delta k_t = 0$ schedule in Fig. 2 comes from imposing $k_{t+1} = k_t$ on Eq. (21). Capital per effective unit of labor declines over time above this line and vice versa. The $\Delta k_t = 0$ locus more or less coincides with the $x(k_t,i_t) = 0$ line at high prevalence rates. Although this is true in all scenarios presented in the paper, it is not a general result and depends on parameter values.

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[16] A complementary exercise would be to study the impact of HIV on malaria transmission. Since HIV introduces only negative effects, the poverty trap attractor would only get stronger. When we look at the effect of malaria on HIV, on the other hand, the conclusion is not as obvious. By reducing the number of sexual encounters, malaria slows down the scope for HIV transmission which counteracts the higher risk of contracting the virus.

Note the parabolic shape of the $\Delta k_t = 0$ 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 Eq. (21) 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 (21)). 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 $x_t = 0$ 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

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

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.[17]

The third locus in Fig. 2 is the downward sloping line, $\Delta i_t = 0$, defined, from Eq. (22), by

$$i_t = p(k_t, i_t), \tag{25}$$

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. In the non-shaded region preventive investment is zero and the prevalence rate is always rising.

The phase diagram 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. Sequences of (k_t, i_t) which do not start exactly on the positively sloped saddle-arm (not shown) leading 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 $x_t = 0$ locus, at which point, the continuation of that locus becomes the effective threshold.

Transition to *BGP* can exhibit interesting dynamics. In Fig. 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.

To understand better how a specific disease affects dynamics and how the disease ecology and costs interact with initial conditions (k_0, i_0) to shape the growth trajectory, we turn to Table 2 which reports quantitative results for the various alternatives.

4.1. Malaria

The upper segment of the $x(k_t, i_t) = 0$ schedule in Fig. 2 plays a key role since any (k_t, i_t) pair above that line is attracted towards *PT*.

Disease ecology determines susceptibility to infection. It depends on the number of encounters (μ_1, μ_2) and the probability of contracting a disease in each such encounter (a, q). As we decrease μ_1, μ_2, q or a, the transmission probability falls, the state space within which people invest in prevention opens up and the upper segment of the $x_t = 0$ locus moves up. 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 that the $x_t = 0$ schedule intersects $i = 1$ at $k = 171,678$ for malaria. This means, in principle, a sufficiently wealthy country never ends up in the poverty trap, similar to the existing literature on poverty traps. But it does require 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.

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[17] 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).

Table 2
Key quantitative 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 k for $i=1$ on the $x=0$ locus	171,678	∞	65	1,396,932,300
Value of ϕ at w. 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, $\phi=0.82$	53	None	21	100
Value of a for which multiplicity vanishes	0.071	—	0.003	0.003

Simulation results that compare the phase diagrams across the three disease environments. The second column of results corresponds to the malaria calibration except for the value of a.

For $a=1$ (second column in Table 2), the $x=0$ locus asymptotes at $i<1$.[18] 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 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 unable to explain 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, consider the effect of disease costs on long-run outcomes. This depends on which parameter we look at. As morbidity costs increase (higher θ, lower δ), it elicits stronger prevention 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 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.

4.2. HIV/AIDS

Can other infectious diseases, for instance HIV/AIDS in SSA, generate effects as powerful as malaria? The phase portrait for the HIV/AIDS parameter values is qualitatively similar to the malaria case in that it exhibits non-ergodic dynamics. Quantitatively though, the state space leading to BGP under HIV/AIDS is significantly larger. One indication of this is that the value of k at which the zero prevention locus intersects $i=1$ is much higher under malaria than under HIV/AIDS (Table 2).

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 risk of contagion without prevention is 0.240 for malaria and 0.005 for HIV. 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. 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. In other words, 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 a single generation alone.

4.3. Dual infection

Finally, consider how the HIV epidemic interacts with the endemic nature of malaria in SSA. 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

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[18] Simulations show that for i sufficiently close to 1 the economy converges to PT for any k that GAUSS can handle, that is, for $k \leq 10^{308}$.
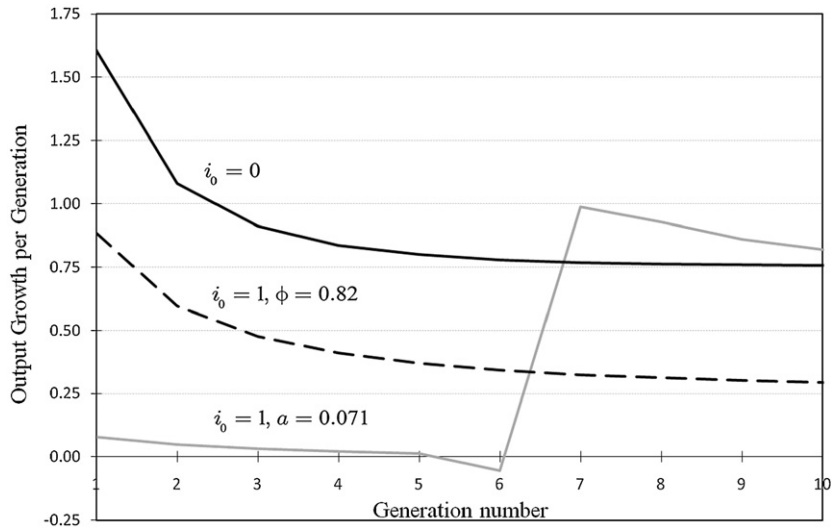
Fig. 3. Output growth per generation. Percentage growth of income per worker per generation against generational time. The benchmark parameter values employed are the ones reported in Table 1 for malaria. All simulations start at k=0.2, marginally below the poverty trap capital stock.

spread and perpetuate in areas of high malaria. 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 8500 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, one aggravating factor being open sores from untreated bacterial STDs in SSA.[19]

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, the latter 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 sustain growth. For example, when a drops from 0.240 to 0.071 (Table 2) the poverty trap disappears and all economies converge to the 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 where the economy grows at a sustained positive rate but, given the "income neutrality" of the trap, does not escape infectious disease. 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 occurring in an impoverished country. Fig. 3 shows the percentage 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 $i_0=0$ (solid, in black) pertains to 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 $i_0 = 1, \phi = 0.82$ (dotted, in black) 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,

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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 $i_0=1, a=0.098$ (solid, in gray) illustrates the consequences of another breakthrough that makes it relatively easier to avoid infection. This can be a medical innovation that improves 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. public health innovations). This change eliminates the poverty trap and triggers a growth take-off. What Fig. 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 encourage prevention until k is large enough. When individuals start engaging in prevention, it dominates other types of growth promoting investment in k. 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 quantitative exercises reinforce that infectious diseases impose large economic costs by causing under-development *or* growth traps. Even without multiple attractors, the cost is substantial: it takes six generations for the third economy in Fig. 3 to enjoy similar growth rates as the first economy.

6. Conclusions

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 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, especially the effectiveness of health aid versus alternative interventions. In a companion paper we have begun to venture along this path (Chakraborty et al., 2010).

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 in disease-prone environments. Third, morbidity can dilute capital intensity, an effect that is stronger at relatively higher levels of development. Perhaps most importantly, the theory suggests that health can generate nonlinearities in the growth process.

Appendix A. Supplementary material

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.jmoneco.2010.08.004.

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