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 mortality (infected individuals can die prematurely) and morbidity (lower productivity and quality of life). Our model offers three main insights. First, a greater prevalence of diseases implies a lower savings-investment propensity because of mortality and morbidity. The extent to which preventive health investment can counter this depends on the prevalence rate and, specifically, on the strength of the disease externality. Second, infectious diseases can generate low-growth traps in which income alone can not push the economy out of underdevelopment. This is distinctly different from the development traps found by previous literature. Third, income *per se* does not cause health when prevalence is high. Successful interventions should therefore be health specific and when possible channeled via the public health delivery system.

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1 Introduction

Health and income are two fundamental issues confronting economists and policymakers alike. Beyond the fact that each is elemental to welfare it is perhaps their joint relationship that is most intriguing. Countries that are poor in per capita income are also more likely to be poor in health. This high positive correlation between a country's income and health status is well documented in the demographics, economics and epidemiology literatures (Soares, 2007). The most documented case is perhaps sub-Saharan Africa that experiences a disproportionate share of the global disease burden along with the lowest per capita income levels and growth rates over the last half century.

These observations beg the question, "do higher levels of health status improve economic development or does economic development improve health status?" What there is a consensus about is that the relationship runs in both directions. But which direction is more dominant? And what does this complex relationship suggest about policies that can effectively enhance growth?

Motivated by these questions we look into the health-development relationship through the lens of a general equilibrium model of infectious disease and growth. Epidemiological factors are introduced into a two-period overlapping generations model where disease transmission depends on incentives and rational behavior. Adult individuals may become infected upon exposure to randomly matched infected (older) individuals. Susceptibility from such encounters depends on two factors: preventive health investment and the disease ecology (climate, vectors, social practices). Diseases are costly because of mortality, which causes premature death in infected adults, and morbidity, which lowers productivity and quality-of-life. Since infected individuals face a higher mortality risk they are less inclined to save. Being less productive workers they are also less able to save. The combined effect renders the economy's savings-investment rate lower, the higher the disease prevalence.[1] But diseases do not evolve exogenously. Since they lower lifetime utility, their prevalence creates incentives for preventive investment. That investment, in turn, depends on the negative disease externality and ability to invest.

The key results from our theory is as follows. First, two types of long-run growth are possible: one where diseases are widespread and growth is low (possibly zero), and the other where diseases ultimately disappear and the economy enjoys a faster sustained improvement in living standards. Initial income, prevalence, and disease ecology determine which of these development paths attracts a particular country. Second, income does not cause health when infectious diseases are widespread, irrespective of the level of development. The disease externality becomes so high in this situation that it wipes out incentives to invest in prevention. An epidemic shock can therefore trip even

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

a wealthy economy into the slower growth path. Furthermore, overall foreign aid in the form of income transfers has little effect on health or development.[2] This is in stark contrast to low-growth traps found by previous literature (e.g., see Azariadis and Stachurski, 2005). It also means, general institutional changes that improve aggregate TFP can raise the growth rate but will have limited impact on disease eradication unless public health institutions improve too.[3] Third, numerical experiments based on our model that examine the efficacy of foreign aid in the form of health assistance to take the economy out of the low-growth path show that costs can be very large. Quick interventions and simultaneously targeting capital accumulation and preventive health can reduce this cost. Finally, the disincentive effect of diseases on savings-investment behavior can be strong enough to slow down growth rate convergence by several generations, even when all countries are converging to the same balanced growth path.

There has been a recent surge in research on health and development that is overwhelmingly empirical. Despite compelling microeconomic evidence that health is important for economic outcomes (see, e.g. Strauss and Thomas, 1998, and Deaton, 2003), the macroeconomic evidence has been mixed. Empirical works such as Bloom and Canning (2005) and Gallup and Sachs (2001) attribute Africa's persistent poverty to endemic infectious diseases particularly malaria. Gallup and Sachs, for example, estimate that malaria reduces per capita income in a malarious country by more than half compared to a non-malarious country. Lorentzen *et al.* (2006) find that adult mortality explains almost all of Africa's growth tragedy in the past forty years.

Other works, however, offer a more qualified view. Acemoglu and Johnson (forthcoming) use cross-country panel data and a novel instrument to control for the obvious endogeneity between health and income. They find very small if any positive effect of health on per capita GDP. These authors argue that the increase in population resulting from better health outweighs the productivity effects and therefore GDP per capita may have actually slightly decreased in their panel of countries. Weil (2007) uses microeconomic estimates of the effect of health on individual outcomes to construct macroeconomic estimates of the effect of (average) health on GDP per capita. His main finding is that eliminating health differences among countries will reduce the variance of log GDP per worker by about 10%. This estimate is economically significant but substantially smaller than estimates from cross-country growth regressions that Bloom and Canning (2005) and

[2] This is consistent with recent evidence. Rajan and Subramanian (forthcoming) examine the effects of aid on growth after correcting for the bias that aid typically goes to poorer countries, or to countries after poor performance. They find little robust evidence of a positive relationship between aid inflows into a country and its economic growth. Mishra and Newhouse (2007) empirically estimate the effects of aid on infant mortality using a dataset covering 118 countries from 1970 to 2004. These authors find that although overall foreign aid does not have a statistically significant effect on infant mortality, *health aid* does.

[3] This is consistent with evidence that the conquest of infectious diseases in many countries has been possible due to improvements in medicine and public health rather than income gains (Cutler *et al.*, 2006, and Soares, 2007).

Gallup and Sachs (2001) report.

Several theoretical papers have looked at health generally and at mortality specifically. Blackburn and Cipriani (1998), Boldrin *et al.* (2005), Chakraborty (2004), Cervellati and Sunde (2005), Doepke (2005), Kalemli-Ozcan (2002) and Soares (2005) variously consider the effect of declining infant mortality and improved longevity on fertility, human capital accumulation, the demographic transition and economic growth. Theoretical work on the microfoundation of diseases and economic growth is more limited. Momota *et al.* (2005) analyze the role of rational disease in giving rise to disease cycles in general equilibrium. Epidemic shocks in Lagerlöf (2003), and mortality declines triggered by nutritional improvements in Birchenall (2007), are used to explain the escape from Malthusian stagnation to modern economic growth. More generally, our paper is related to the Unified Growth Theory (Galor, 2005; Galor and Moav, 2002; Galor and Weil, 2000). In our model, a stagnant economy starts enjoying modern growth when prevalence rates fall sufficiently due to exogenous improvements in medicine, public health or the disease environment.

A novel feature of this paper compared to the literature cited above and the mathematical epidemiology is its microfounded disease behavior. The evolution of diseases is typically exogenous to human decisions in epidemiological models. As Geoffard and Philipson (1996) argue, ignoring the effect of rational behavior can convey an incorrect view of disease dynamics and the effectiveness of public health interventions.

The paper is organized as follows. In section 2, we specify a simplified version of the model to highlight some of the main forces driving our results. Its main advantage is that results can be presented with analytical solutions. This simplified version, however, although useful to understand why our model delivers predictions distinctly different from previous work, do not incorporate the key ingredient of our theory that is responsible for the main results. Section 3 incorporates it, and analyzes the complete version of the model. In particular, the section adds microfounded infection-transmission dynamics into the simplified version. This comes at the expense of having to approximate solutions and study dynamics with numerical methods. Section 5 uses the complete model predictions to shed additional light on the Africa's experience. Section 6 concludes.

2 A Simple Model

Our framework is 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,

[4] We do not explicitly model childhood. We do so because developing countries have enjoyed enormous life expectancy improvements over the past fifty years mainly due to sharp declines in infant and child mortality made possible by low-cost interventions and technology transfers. Adult mortality has declined relatively less and remains high in developing countries (World Bank, 1993). More importantly, this excessive adult mortality is mainly due to

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 Mechanism

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 an utility cost associated with being infected: he 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.

Young individuals can undertake preventive health investment, x_t, early in life. This may take the form of net food intake (that is, nutrients available for cellular growth), personal care and hygiene, accessing clinical facilities and related medical expenditure. It may even take the form of abstaining from risky behavior. What is key is that such investment is privately costly and improves resistance to infectious diseases. We model these costs in terms of income but just as likely they can be foregone utility (for instance, as in Geoffard and Philipson, 1996).

Diseases spread from infected older individuals to susceptible younger ones. In particular, a susceptible young person randomly meets $\mu > 1$ older individuals during the first half of his youth, before old infected agents start dying. Not all of these older individuals will be infected and not all encounters with infected people result in transmission. For the time being, let us assume that the probability of being infected (p_t) after these μ encounters is given by

$$p_t(x_t) = \mu \pi_t(x_t) i_t, \tag{1}$$

where $\pi_t(x_t)$ is the probability that a young individual gets infected in an encounter with an infected adult, and i_t is disease prevalence among adults. Furthermore, suppose that

$$\pi(x_t) = \begin{cases} \pi_1, & \text{if } x_t = x > 0 \\ \pi_0, & \text{if } x_t = 0. \end{cases} \tag{2}$$

Let $\mu\pi_1 < 1 < \mu\pi_0$. This assumption ensures an infected person infects more than one susceptible person in the absence of preventive investment but less than one (on average) if susceptible individuals undertake preventive investment.

infectious diseases which affect a disproportionate number of adults in poorer countries compared to their counterparts elsewhere. In the model, children's consumption is subsumed into their adult parent's. Since we focus on adult mortality, the effect of infant mortality on fertility decisions is ignored. Childhood morbidity from infectious diseases, however, can have lifelong repercussions on productivity and human capital. This morbidity effect is implicity incorporated below through cost of disease parameters.

Notice that equation (1) includes an important negative externality that characterizes infectious diseases. When an individual chooses preventive health investment *ex ante* – before he meets an infected older person – he does not take into account how his decision impacts the susceptibility of future generations. Furthermore, this externality is amplified by the random matching process.

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 in Africa, for example, show that reduction in all-cause mortality is considerably greater than the mortality reduction from malaria alone (see Abu-Raddad *et al.*, 2006).

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

Thirdly, within this disease ecology falls social norms and behavior. In several African societies for instance, social norms 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). Such norms 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 otherwise be circumspect in their social interactions may remain actively involved or simply hide their disease to avoid isolation.

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 in the model. More generally, infected individuals could invest in curative behavior that affects the length and severity of diseases. Incorporating such behavior should not qualitatively alter the model's predictions.

[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.

2.2 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

$$F(K^i, L^i) = A(K^i)^\alpha(\overline{k}L^i)^{1-\alpha}, \tag{3}$$

where A is a constant productivity parameter, and $\bar{k}$ denotes the average capital per effective unit of labor across firms.[6] For our model to generate 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 gross interest rate (R_t) are given respectively by

$$w_t = (1-\alpha)Ak_t \equiv w(k_t), \text{ and} \tag{4}$$

$$R_t = \alpha A \equiv R. \tag{5}$$

2.3 Preferences

Preferences and individual behavior are disease contingent. We first consider decisions of an uninfected individual whose health investment has successfully protected him from the disease. We assume, for the time being, that the period utility function is linear. The individual maximizes lifetime utility

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

subject to the budget constraints

$$c_{1t}^U = w_t - x_t - z_t^U \tag{7}$$

$$c_{2t+1}^U = R_{t+1}z_t^U, \tag{8}$$

where z denotes savings and x is given by decisions made early in period t.[7] Hereafter we tag variables by U and I to denote decisions and outcomes for uninfected and infected individuals, respectively.

[6] The choice of such a simple Ak growth mechanism is only for tractability. The basic story generalizes to other setups where savings behavior determines growth (in closed or open economies) via innovation and factor accumulation, as 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.

[7] 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.

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

$$\delta \left[c_{1t}^I + \beta \phi c_{2t+1}^I \right], \tag{9}$$

subject to

$$c_{1t}^I = (1-\theta) w_t - x_t - z_t^I \tag{10}$$

$$c_{2t+1}^I = R_{t+1} z_t^I + \tau_{t+1}, \tag{11}$$

where the new term 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.[8] Clearly transfers per surviving infected individual will be

$$\tau_{t+1} = \left(\frac{1-\phi}{\phi} \right) R_{t+1} z_t^I, \tag{12}$$

in equilibrium.

For simplicity suppose $\beta R > 1 > \phi \beta R$. Under this condition, infected individuals do not save at all while uninfected individuals save their entire labor income. That is, $c_{1t}^U = c_{2t+1}^I = z_t^I = \tau_{t+1} = 0$, $c_{1t}^I = (1-\theta) w_t - x_t$, $z_t^U = w_t - x_t$, and $c_{2t+1}^U = R_{t+1}(w_t - x_t)$. Substituting these into expected lifetime utility gives the two indirect utility functions

$$V^U(x_t) = \beta R_{t+1}(w_t - x_t), \text{ and} \tag{13}$$

$$V^I(x_t) = \delta \left[(1-\theta) w_t - x_t \right]. \tag{14}$$

At the beginning of period t, adults choose the optimal level of x_t to maximize expected lifetime utility. Recall that a young individual's probability of catching the disease is p_t given by (1). Hence, individuals choose x_t to maximize

$$p_t(x_t) V^I(x_t) + \left[1 - p_t(x_t) \right] V^U(x_t), \tag{15}$$

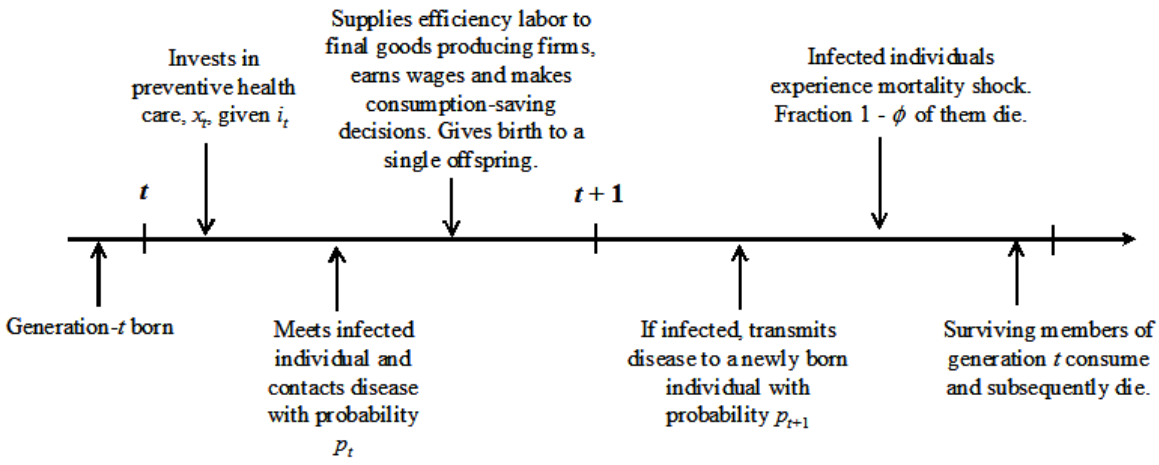
at the beginning of period t. Given that the prevention investment decision is either investing x units or nothing, it is straightforward to show that $x_t = x$ if and only if expected lifetime utility is higher in doing so

$$p_t(x) V^I(x) + \left[1 - p_t(x) \right] V^U(x) > p_t(0) V^I(0) + \left[1 - p_t(0) \right] V^U(0). \tag{16}$$

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

All savings are invested in capital, which are rented out to final goods producing firms, earning a return equal to the rental rate. The initial old generation is endowed with a stock of capital K_0 at $t = 0$. Finally, an exogenously specified fraction i_0 of old agents suffer from infectious diseases. The depreciation rate on capital is set equal to one. We summarize the timeline of events in Figure 1.

Figure 1: Timeline of Events



2.4 Dynamics

With a continuum of young agents of measure one, we can appeal to the law of large numbers and write down aggregate savings at t in this simple economy as the savings of uninfected individuals

$$S_t = z_t^U, \tag{17}$$

and the asset market clearing condition as

$$K_{t+1} = S_t. \tag{18}$$

To express this in terms of capital per efficiency unit of labor, note that efficiency-labor supply comprises of the labor of infected and uninfected individuals, that is,

$$L_{t+1} = (1-\theta)p_{t+1} + (1-p_{t+1}) = 1 - \theta p_{t+1}. \tag{19}$$

The higher the value of θ, the less productive are infected workers, and hence the less effective is labor supply.

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

$$k_{t+1} = \frac{[1 - p(k_t, i_t)]z^U(k_t, i_t)}{1 - \theta p\left(p(k_t, i_t)\right)}. \tag{20}$$

By the law of large numbers, equilibrium disease dynamics evolve according to

$$i_{t+1} = p(k_t, i_t). \tag{21}$$

Equations (20) and (21) describe the general equilibrium of this economy given initial conditions.

The dynamics of the system can be analyzed with the aid of a phase diagram. Three loci in (k_t, i_t) space determine the dynamics. The first two consist of the locus along which disease prevalence remains constant (the $\Delta i_t = 0$ line) and the locus for which capital per effective unit of labor remains constant (the $\Delta k_t = 0$ line). The third, is the general equilibrium version of (16) along which individuals are indifferent between investing and not investing in preventive care.

The assumption $\mu\pi_1 < 1 < \mu\pi_0$ implies, from equations (1) and (21), that for no (k_t, i_t), with $i_t \in (0, 1)$, is disease prevalence constant. More specifically, disease prevalence increases when $x_t = 0$ and decreases if $x_t = x$. This means prevalence dynamics will be governed by the locus of (k_t, i_t) pairs for which condition (16) holds with strict equality.

From expressions (1), (5), (13) and (14), we can write (16) as

$$\mu\pi_1 i_t \delta\left[(1-\theta)w_t - x\right] + \left(1 - \mu\pi_1 i_t\right)\beta R\left(w_t - x\right) > \mu\pi_0 i_t \delta(1-\theta)w_t + \left(1 - \mu\pi_0 i_t\right)\beta R w_t.$$

Substituting for equilibrium wages from (4) we obtain

$$i_t > \frac{\beta R x}{(1-\alpha)Ak_t\mu\left(\pi_0 - \pi_1\right)\left[\beta R - \delta(1-\theta)\right] + \mu\pi_1\left(\beta R - \delta\right)x} \equiv \Phi(k_t). \tag{22}$$

It is easy to show that:

(i) Φ is decreasing in k_t;

(ii) $\lim_{k\to\infty}\Phi(k) = 0$;

(iii) $\Phi(0) = \beta R/[\mu\pi_1\left(\beta R - \delta\right)] > 1$.

Hence the locus $i_t = \Phi(k_t)$ is downward sloping and has a positive intercept exceeding 1 at $k = 0$. The prevalence rate always rises for (k_t, i_t) pairs below and to the left of this locus and always decreases to the right of it.

We next determine the shape of the $\Delta k_t = 0$ schedule depending on whether or not individuals invest in preventive care.

Case 1: $x_t = 0$

Equations (1) – (4) and (20) imply

$$k_{t+1} = \left[\frac{1-\mu\pi_0 i_t}{1-\theta\mu\pi_{t+1}(\mu\pi_0 i_t)}\right](1-\alpha)Ak_t,$$

so that

$$k_{t+1} \geq k_t \Longleftrightarrow \frac{(1-\alpha)A-1}{[(1-\alpha)A-\theta\mu\pi_{t+1}]\,\mu\pi_0 i_t} \geq 1.$$

Let $(1-\alpha)A > \theta\mu\pi_0$ which ensures that the expression on the left is positive. Note that $\mu\pi_0 i_t$ goes to $\mu\pi_0 > 1$ as $i_t \to 1$. Hence the expression on the left becomes smaller than 1 for a sufficiently large disease prevalence. In addition, the expression becomes undefined as $i_t \to 0$. Hence there exists $\hat{\imath} \in (0,1)$ such that $\Delta k_t < 0$ for all $i_t > \hat{\imath}$ and $\Delta k_t > 0$ for all $i_t < \hat{\imath}$. This threshold is given by

$$\hat{\imath}(\pi_{t+1}) = \frac{(1-\alpha)A-1}{[(1-\alpha)A-\theta\mu\pi_{t+1}]\,\mu\pi_0}. \tag{23}$$

Note that the expression on the right-hand side in (23) depends on π_{t+1}, hence on x_{t+1}. As long as the economy is not too close to the $i_t = \Phi(k_t)$ line, time-t dynamics will place the economy to the left of this curve at $t+1$. In this case $\pi_{t+1} = \pi_0$. If, on the other hand, the economy is close to the $i_t = \Phi(k_t)$ line, i_{t+1} might fall to the right of this locus and $\pi_{t+1} = \pi_1$. The threshold $\hat{\imath}$ can be then discontinuous with $\hat{\imath}(\pi_1) < \hat{\imath}(\pi_0)$.

Case 2: $x_t = x$

Now the motion equation of capital (20) becomes

$$k_{t+1} = \left[\frac{1-\mu\pi_1 i_t}{1-\theta\mu\pi_{t+1}(\mu\pi_1 i_t)}\right][(1-\alpha)Ak_t - x],$$

which implies

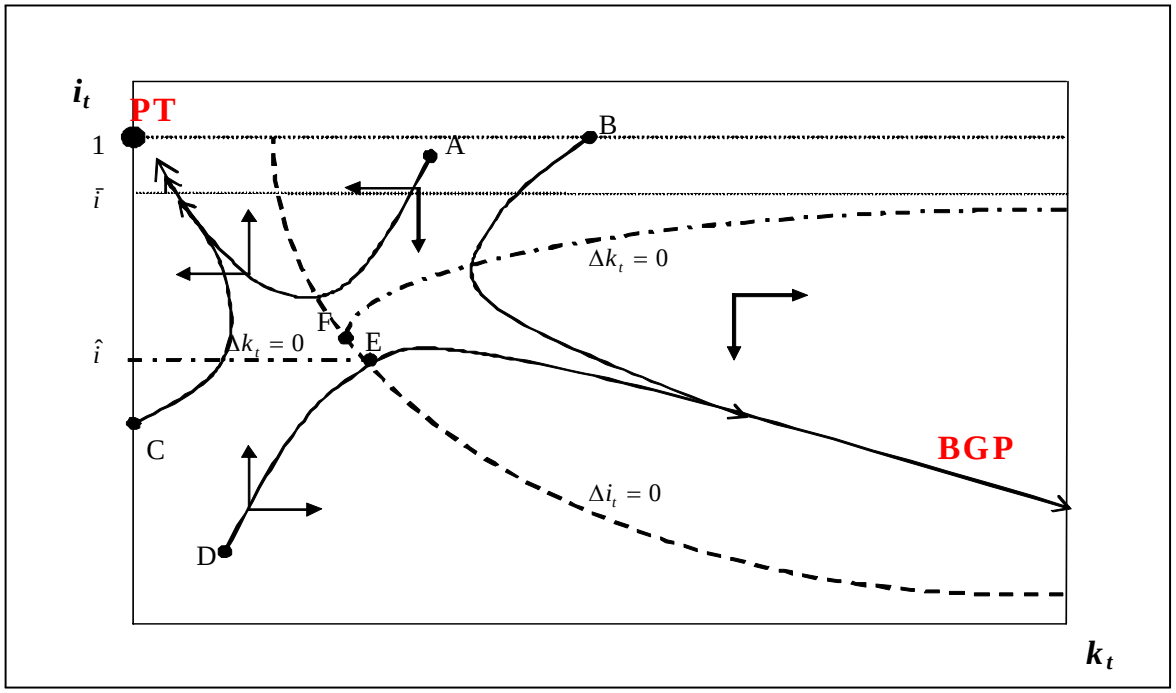
$$k_{t+1} \geq k_t \Longleftrightarrow k_t \geq \frac{(1-\mu\pi_1 i_t)x}{(1-\mu\pi_1 i_t)(1-\alpha)A-[1-\theta\mu\pi_{t+1}(\mu\pi_1 i_t)]} \equiv \Psi(i_t;\pi_{t+1}). \tag{24}$$

The denominator of Ψ is positive at $i_t = 0$ and declines monotonically with i_t. Hence Ψ increases with i_t. But the denominator of Ψ can become zero if

$$i_t = \bar{\imath} = \frac{(1-\alpha)A-1}{[(1-\alpha)A-\theta\mu\pi_1]\,\mu\pi_1}. \tag{25}$$

We obtain $\bar{\imath}$ by setting π_{t+1} equal to π_1 in the denominator of Ψ. This is the relevant probability since as the denominator approaches zero, Ψ goes to infinity and so does k_t on the $\Delta k_t = 0$ line and hence, preventive investment remains positive for any prevalence rate. Clearly $\bar{\imath} < 1$ if and

Figure 2: Phase Diagram for the Basic Model



only if $(1-\alpha)A < 1+\mu\pi_1(1-\theta\mu\pi_1)/(1-\mu\pi_1)$. Under this assumption, the $\Delta k_t = 0$ line is upward sloping with an asymptote at $\bar{\imath}$.

If $\bar{\imath} > 1$, on the other hand, k_t rises with i_t along the $\Delta k_t = 0$ line and coincides with the $i_t = 1$ line for capital stocks exceeding $(1-\mu\pi_1)x/[(1-\mu\pi_1)(1-\alpha)A - 1 + \theta\mu^2\pi_1^2]$. As before a discontinuity is possible near the $i_t = \Phi(k_t)$ locus. In particular, if i_{t+1} falls to the left of this locus for any (k_t, i_t) on the $\Delta k_t = 0$ schedule, then $\pi_{t+1} = \pi_0$ and k_t will be significantly smaller than if $\pi_{t+1} = \pi_1$ by (24).

The phase diagram shown in Figure 2 ignores these discontinuities in the $\Delta k_t = 0$ schedule since they have minor effects on long-run dynamics. Two stable attractors are present in Figure 2. The first is a zero-growth poverty trap (PT), and the second is a balanced growth path (BGP) with strictly positive growth. In addition, there exist two unstable steady states at points E and F. Note that E and F need not coincide given the discrete nature of disease-prevention and transmission.

There is no preventive investment in the poverty trap and disease prevalence is widespread. In addition, the capital stock in this trap is zero since infected individuals do not save. Economies that move along BGP, on the other hand, invest every period in prevention and asymptotically approach full eradication of infectious diseases and, from equation (24), a growth rate of output

per worker equal to $(1-\alpha)A-1$.

Recall that at $t=0$ the economy is endowed with K_0 units of capital owned by the initial old generation as well as with i_0, the prevalence rate of that generation. Hence both k_0 and i_0 are predetermined variables. Which of the stationary equilibria our model economy gravitates towards partly depends on these initial conditions. Economies that converge to PT are like A and C in Figure 2, with relatively low capital stock and large prevalence rates initially. Economies such as B and D in Figure 2 start with more favorable conditions to converge to BGP.

But initial conditions only partly determine convergence dynamics. For a given pair (k_0, i_0), the attractor that dominates an economy's dynamics depends on disease ecology and costs and technological productivity. Ecology determines susceptibility to infection, which depends on the number of encounters (μ) and the probability of catching the disease in each such encounter (π_0). Equation (22) implies that a larger $\mu\pi_0$ shifts the $i_t=\Phi(k_t)$ line to the left, whereas (23) suggests that $\hat{\imath}$ increases with $\mu\pi_0$ when $\mu\pi_0$ is not too large. Therefore, for low values of $\mu\pi_0$, a larger $\mu\pi_0$ tends to decrease the state space that leads to PT. For high values of $\mu\pi_0$, the effect is ambiguous.

Higher economic costs of infectious diseases have similar effects, shrinking the state space that leads to PT. A lower labor productivity (higher θ) shifts left the $i_t=\Phi(k_t)$ schedule and moves up the $\Delta k_t=0$ locus. A lower utility flow from ill-health (lower δ), on the other hand, shifts left the $i_t=\Phi(k_t)$ schedule. Note that the survival probability ϕ does not affect dynamics since only uninfected individuals save in this model.

Let us now look at the impact of parameters related to output or disease-prevention productivity. A larger technology parameter A shifts the $\Delta i_t=0$ and $\Delta k_t=0$ schedules to the left. The same is true for a smaller value of x. Finally, a smaller values of π_1 shifts the $\Delta i_t=0$ schedule to the left. All these shifts reduce the state space that leads to the trap. The logic for the result is the same as above.

Another aspect of the simplified version of the model that we want to emphasize is that the trap is a standard one, in the sense that financial aid can rescue any economy and take it to the balanced growth path. For example, if an economy located at PT in Figure 2 receives a grant that pushes it to a location like B, the economy will scape the trap and gravitate towards the BGP attractor. This also implies that a relatively capital rich nation can never fall into PT. As we will see in the next section, these properties are absent in the complete model.

We can conclude that infectious diseases can be a source of poverty traps. The possibility of multiple growth paths depends on the economy's average propensity to invest. This propensity is too low when everyone is infected, thus creating the trap, but takes on a higher value that allows sustained long-run growth for an uninfected population. Whether the economy falls into the

trap depends on initial state-variable values, and also on disease ecology. Relatively capital-poor economies with relatively large disease prevalence end up in the bad steady state. More adverse disease conditions as well as technologies with higher productivity tend to shrink the state space over which convergence to the no-growth high-prevalence path is possible.

3 The Complete Model

The interaction of diseases and economic behavior is not specific to the functional forms and assumptions used above. We offer a more general model in this section. The key modification is a more realistic disease transmission mechanism that drives the main predictions of our theory. In particular, the trap becomes stronger, possibly characterized by output growth, and income will not longer be able to take the economy out of that low-growth path. We also introduce some other modifications to technology and preferences that allow us to consider parametric values that are established in the existing literature. The added complexity, however, means the model has to be solved numerically.

3.1 Endogenous Disease Transmission

We begin by deriving the probability p_t of being infected after μ encounters instead of assuming it. Recall that diseases spread from infected older individuals to susceptible younger through a process of random matching. If encounters are independent, the probability of not getting infected during youth equals the product (across meetings) of not being infected. The probability of being infected after one match is the probability of meeting an infected individual (i_t) times the probability of getting infected by the encounter (π_t), that is, $i_t\pi(x_t)$. Hence, the probability of not being infected after μ matches is simply $[1 - i_t\pi(x_t)]^\mu$. Thus,

$$p_t = 1 - [1 - i_t\pi(x_t)]^\mu . \tag{26}$$

The main difference between equations (1) and (26) is that, in the latter, the negative externality associated with disease contagion rises exponentially with the number of encounters μ. This much stronger external effect is endogenous to the disease propagation process and will be important in understanding the results below.

We next modify the infection-probability function with a continuous form. In particular, given a preventive health investment x, the probability that a young individual gets infected from a matching is given by

$$\pi(x) = \frac{aq}{q+x},\ a \in (0,1),\ a > 1/\mu,\ q > 0. \tag{27}$$

Notice that this functional form fulfills the following properties: $\pi' < 0$, $\pi(0) = a$, $\pi(\infty) = 0$, and $\pi'(0)$ goes to $-\infty$ as q goes to zero. We restrict $a > 1/\mu$ so that disease prevalence rises over time in the absence of preventive investment.

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 parameter a is an evolutionary parameter which gives the probability of getting infected if agents do not invest in 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).

3.2 Concave Preferences and Natural Capital

Next we drop the linear utility function and assume that 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. The uninfected individual maximizes lifetime utility

$$u\left(c_{1t}^{U}\right) + \beta u\left(c_{2t+1}^{U}\right), \tag{28}$$

subject to (7) and (8), whereas the infected maximizes

$$\delta\left[u\left(c_{1t}^{I}\right) + \beta\phi u\left(c_{2t+1}^{I}\right)\right], \tag{29}$$

subject to (10) and (11). Remember that x_t is given by decisions made early in period t.

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

$$u'(c_{1t}^{U}) = \beta R_{t+1} u'(c_{2t+1}^{U}) \tag{30}$$

$$u'(c_{1t}^{I}) = \beta\phi R_{t+1} u'(c_{2t+1}^{I}), \tag{31}$$

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

We assume a CES utility function

$$u(c) = \frac{c^{1-\sigma} - 1}{1-\sigma},\ \sigma \in (0,1), \tag{32}$$

for analytical convenience. There is though a potential problem with this choice. While we have assumed zero utility from death, this function takes on negative values when consumption is less

than one (in the simulations below we set $\sigma = 1$). Since we think of death as the outcome that provides the lowest utility, consumption has to strictly exceed one.

In order to ensure the last restriction, we modify the production technology. In particular, we consider in the complete model a production technology that incorporates $b > 0$. We can think of b as capturing non-produced natural endowments such as trees and animals:

$$F(K^i, L^i) = A(K^i)^\alpha(\overline{k}L^i)^{1-\alpha} + bL^i. \tag{33}$$

As long as b is sufficiently large, consumption will be always above one.

The inclusion of b only affects the equilibrium return to labor which is now

$$w_t = (1-\alpha)Ak_t + b \equiv w(k_t) \tag{34}$$

As will be clear later, $b > 0$ is not necessary to obtain the main results of the paper.

3.3 Preventive Investment Decision

Our next task is analyzing the preventive investment decision under these new assumptions. Given the Euler conditions (30) – (31) and the utility function (32), optimal savings for uninfected and infected individuals are

$$z_t^U = \left[\frac{\beta^{1/\sigma}R_{t+1}^{1/\sigma-1}}{1+\beta^{1/\sigma}R_{t+1}^{1/\sigma-1}}\right](w_t - x_t) \tag{35}$$

$$z_t^I = \left[\frac{(\beta\phi)^{1/\sigma}R_{t+1}^{1/\sigma-1}}{1+(\beta\phi)^{1/\sigma}R_{t+1}^{1/\sigma-1}}\right][(1-\theta)w_t - x_t] - \left[\frac{1}{1+(\beta\phi)^{1/\sigma}R_{t+1}^{1/\sigma-1}}\right]\frac{\tau_{t+1}}{R_{t+1}}. \tag{36}$$

Substituting these into lifetime utility gives the two indirect utility functions

$$V^U(x_t) = \frac{1}{1-\sigma}\left[\left(w_t - x_t - z_t^U\right)^{1-\sigma} + \beta\left(R_{t+1}z_t^U\right)^{1-\sigma}\right] - \frac{1}{1-\sigma} \tag{37}$$

$$V^I(x_t) = \frac{\delta}{1-\sigma}\left[\left((1-\theta)w_t - x_t - z_t^I\right)^{1-\sigma} + \beta\phi\left(R_{t+1}z_t^I\right)^{1-\sigma}\right] - \frac{\delta}{1-\sigma}, \tag{38}$$

contingent on prices, preventive health investment and disease realizations.

A young individual's probability of catching the disease is p_t given by (26). Hence, individuals choose x_t to maximize expected lifetime utility

$$p_t V^I(x_t) + [1-p_t]\,V^U(x_t), \tag{39}$$

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

$$-\mu\left[1 - i_t\pi_t\right]^{\mu-1}\pi'(x_t)i_t\left(V_t^U - V_t^I\right) \geq p_t\left(-\frac{\partial V_t^I}{\partial x_t}\right) + [1-p_t]\left(-\frac{\partial V_t^U}{\partial x_t}\right), \tag{40}$$

for $x_t \geq 0$. This states that for individuals to be willing to invest in disease prevention, the marginal benefit from living longer and experiencing a healthier life cannot be outweighed by the marginal cost of foregoing current income.[9]

Next substitute equilibrium prices and transfers into the saving functions to obtain

$$z_t^U = s^U \left[w(k_t) - x(w_t, i_t)\right] \equiv z^U(k_t, i_t), \tag{41}$$

and

$$z_t^I = s^I \left[(1-\theta)w(k_t) - x(w_t, i_t)\right] \equiv z^I(k_t, i_t), \tag{42}$$

where,

$$s^U \equiv \left[\frac{\beta^{1/\sigma} R^{1/\sigma - 1}}{1 + \beta^{1/\sigma} R^{1/\sigma - 1}}\right], \; s^I \equiv \left[\frac{\phi(\beta\phi)^{1/\sigma} R^{1/\sigma - 1}}{1 + \phi(\beta\phi)^{1/\sigma} R^{1/\sigma - 1}}\right]. \tag{43}$$

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 since they are less productive ($\theta > 0$). The third type of cost, a lower flow of utility ($\delta < 1$), can affect savings as well but the effect will operate through preventive investment.

Substituting the savings functions into indirect utility obtains

$$\begin{aligned} V_t^{U*} &= \frac{1}{1-\sigma}\left[\left(1 - s^U\right)^{1-\sigma} + \beta R^{1-\sigma}\left(s^U\right)^{1-\sigma}\right]\left(w(k_t) - x_t\right)^{1-\sigma} - \frac{1}{1-\sigma} \\ &\equiv \frac{\zeta^U \left(w(k_t) - x_t\right)^{1-\sigma}}{1-\sigma} - \frac{1}{1-\sigma}, \end{aligned} \tag{44}$$

and

$$\begin{aligned} V_t^{I*} &= \frac{\delta\phi^\sigma}{1-\sigma}\left[\left(\frac{1 - s^I}{\phi + (1-\phi)s^I}\right)^{1-\sigma} + \beta R^{1-\sigma}\left(s^I\right)^{1-\sigma}\right]\left((1-\theta)w(k_t) - x_t\right)^{1-\sigma} - \frac{\delta}{1-\sigma} \\ &\equiv \frac{\zeta^I \left((1-\theta)w(k_t) - x_t\right)^{1-\sigma}}{1-\sigma} - \frac{\delta}{1-\sigma}. \end{aligned} \tag{45}$$

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 (12) when making health investment decisions. Accordingly (40) becomes

$$p_t \zeta^I [(1-\theta)w(k_t) - x_t]^{-\sigma} + (1-p_t)\zeta^U [w(k_t) - x_t]^{-\sigma} \leq -\mu \left[1 - i_t \pi_t\right]^{\mu - 1} \pi'(x_t) i_t \left[V_t^{U*} - V_t^{I*}\right]. \tag{46}$$

Two possibilities arise depending on whether or not preventive investment yields positive returns. If (46) holds as a strict inequality at $x_t = 0$, optimal investment will be $x_t = 0$. The left-hand

[9] The first order conditions (30) – (31) and (40) 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.

side of (46) is the marginal utility cost of that investment, since health investment entails a lower current and, possibly, future consumption. The right-hand side constitutes the marginal benefit, in the form of higher net utility from lowering one's chance of catching the infectious disease. 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 the chance of leading a healthy life in such situations.

Rewriting (46) above, the condition for zero preventive investment is

$$\chi(k_t, i_t) = \zeta^U[1-p(0)]+\zeta^I(1-\theta)^{-\sigma}p(0)]w_t^{-\sigma}+\mu\left[1-i_t\pi(0)\right]^{\mu-1}\pi'(0)i_t\left\{V_t^U(0)-V_t^I(0)\right\}\geq 0. \quad (47)$$

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 (46) holds as an equality and

$$x_t = x(k_t, i_t), \quad (48)$$

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

3.4 Dynamics

As in the basic model, the weighted average of the savings of infected and uninfected individuals is given by $S_t = p_t z_t^I + (1-p_t)z_t^U$, the asset market clearing condition by $K_{t+1} = S_t$ and effective labor supply by $L_{t+1} = 1 - \theta p_{t+1}$.

Using optimal health investment $x(k_t, i_t)$, we can 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 directly increases the probability through the matching process but also tends to lower it by encouraging preventive investment. 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

$$k_{t+1} = \frac{p(k_t, i_t)z^I(k_t, i_t) + [1-p(k_t, i_t)]z^U(k_t, i_t)}{1-\theta p\left(p(k_t, i_t)\right)}, \quad (49)$$

while disease dynamics evolve as before

$$i_{t+1} = p(k_t, i_t). \tag{50}$$

Equations (49) and (50) describe the general equilibrium of this economy given initial conditions. Given the nonlinearities present in the above two equations, we characterize the disease dynamics numerically in the next subsection. As in the simpler version, there are two types of stationary equilibria, a development trap where output and capital per capita grow at a relatively low rate and there is widespread disease prevalence and a balanced growth path (BGP) along which per capita variables grow at a relatively high rate and infectious diseases disappear.

Even though dynamics can not be studied algebraically, it is easy to derive the growth rate along each of the two different balanced-growth paths. Define γ as the asymptotic growth rate of the economy's capital. When $i_t = 0$, the economy-wide saving propensity becomes s^U and, then, equation (49) implies

$$1 + \gamma^H \equiv (1-\alpha)As^U = \frac{\beta}{1+\beta}(1-\alpha)A. \tag{51}$$

In the numerical exercises, this number $1 + \gamma^H$ is always larger than one, which ensures sustained growth. If, on the other hand, $i_t = 1$ then the economy's saving rate equals s^I. Hence, (49) implies that long-run growth is

$$1 + \gamma^L \equiv (1-\alpha)As^I = \frac{\beta\phi^2}{1+\beta\phi^2}(1-\alpha)A. \tag{52}$$

This growth rate is zero if $(1-\alpha)As^I \leq 1$ but strictly positive when $(1-\alpha)As^I > 1$. Clearly the two growth rates above differ only because $\phi < 1$. It is through adult mortality alone that diseases impact long-run growth. Morbidity factors will turn out to matter only for convergence dynamics either by affecting savings directly (for θ) or indirectly (via x for δ).

3.5 Numerical Solutions

The central mechanism of our theoretical model is the interdependence between infectious diseases and economic outcomes. To identify how exactly the growth path is shaped by various economic and disease-specific conditions we rely on computational techniques. We first assign benchmark values to the model parameters and establish its dynamic properties through simulation exercises. Given the difficulty in assigning precise values to the disease related parameters, these numerical experiments should be seen as a method to dig deeper on the qualitative impact of infectious disease costs, policy interventions, and disease ecology and institutions on the economy's dynamics.

Table 1 presents the benchmark parameter values. The model features overlapping generations of agents who potentially live for two periods. To choose the length of one period, we use data on

Table 1: Benchmark Parameter Values

β	$0.99^{(31.5\times4)}$	α	0.67	θ	0.15	μ	5
σ	1	g_y	0.018	ϕ	0.47	q	0.14
b	1			δ	0.9	a	1

life expectancy at birth (LE). The 2005 Human Development Report (UNDP 2005) attributes 78 years of LE to OECD nations, average for the 2000-2005 period. If we focus on adults and consider the first 15 years as childhood, we obtain $(78-15)/2 = 31.5$ years for each period or generation.

We assign a value of $0.99^{31.5\times4}$ to the discount factor (β), that is, 0.99 per quarter which is standard in the real-business-cycle literature. We set the elasticity of substitution for consumption (σ) to 1 (log preferences). The production function has three parameters: the technology parameter A, the capital elasticity α, and the labor productivity coefficient b. We normalize b to 1 to ensure that consumption levels are bounded above one and, as a consequence, utility when alive remains positive. We calibrate α to 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 so as to reproduce an annual long-run growth rate in the low-prevalence steady state of 1.8%. This number is the average growth rate of GDP per capita between 1990 and 2003 for OECD nations in UNDP (2005). Therefore, A is chosen such that the balanced growth rate $s^U(1-\alpha)A$ equals $1.018^{31.5}$, which in turn implies that $A = 24.19$.

We have no guidance on the parameters governing disease transmission, including the prevention technology (π) and number of matches (μ). We choose the functional form (27) and set $a = 1$ as the benchmark. To assign values to μ and q, we require that a country can escape a low-growth trap if preventive investment represents at least 7.2% of its GDP. This percentage comes from dividing 34 by 475. The amount of 34 (current US$) is the minimum expenditure required for scaling up a set of essential interventions to fight diseases in least-developed countries estimated by WHO (2001a). 475 represents South-Saharan Africa's average GDP per capita, also in current US$, in 2001 estimated by UNDP (2003). For each value of μ, the procedure provides a value for q. Taking $\mu = 5$ as our benchmark, we obtain $q = 0.14$.[10] We also perform sensitivity analyses for (μ, q) equal to $(2, 0.55)$ and $(10, 0.06)$.

We have more guidance about the parameters that govern the cost of diseases to individuals. There are some estimates on how ill health affects utility (or quality of life). In particular, Viscusi and Evans (1990) estimate that for injuries severe enough to generate a lost workday with an average duration of one month, the marginal utility of income falls to 0.92 in a logarithmic utility

[10] This satisfies the condition that $a > 1/\mu$.

function model, although it can fall to 0.77 with a more flexible utility, where good health has a marginal utility of 1. This leads us to assign a benchmark value of 0.9 to the parameter δ.

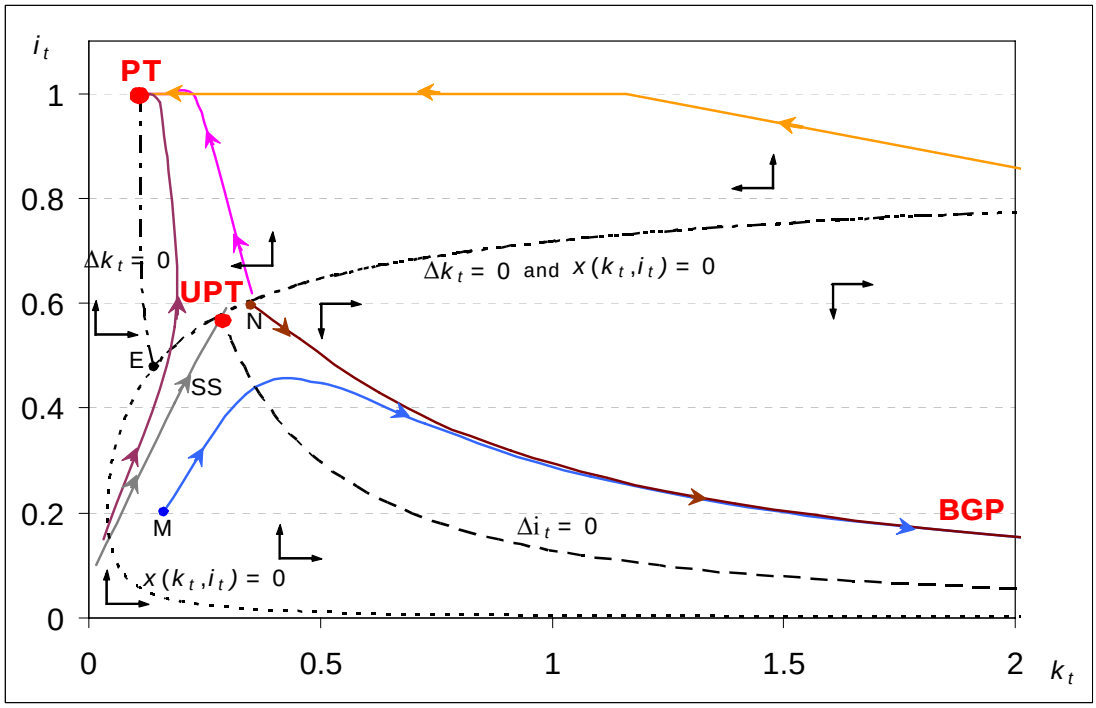
Regarding morbidity, Dasgupta (1993) finds that workers (in particular, farm workers in developing countries) are often incapacitated – too ill to work – for 15 to 20 days each year, and when they are at work, productivity may be severely constrained by a combination of malnutrition and parasitic and infectious diseases. His estimates suggest that potential income losses due to illness for poor nations are of the order of 15%. Focusing on specific diseases, Fox *et al.* (2004), study the impact of AIDS on labor productivity in Kenya and estimate that individuals affected by the illness suffer an earning loss of 16% in their second to last year of life, and 17% in their last year. Malaria infection does not seem to directly affect labor productivity of infected individuals when they are working, as Brohult *et al.* (1981) suggest. However, malaria usually causes anemia and loss of days of work, and therefore affects indirectly labor efficiency. For example, Khan (1966) and Winslow (1951) estimate a 20% reduction in work efficiency in Pakistan and a $5-10\%$ reduction in Southern Rhodesia. We assign an intermediate value 0.15 to θ.

Next, we calibrate the mortality parameter ϕ. According to WHO (2004), more than 90% of all deaths from infectious diseases are caused by a few diseases: lower respiratory infections, HIV/AIDS, diarrheal diseases, tuberculosis, malaria and measles. But their case-fatality rates differ substantially. For example, AIDS and tuberculosis are characterized by relatively high adult mortality. In particular, untreated pulmonary tuberculosis leads to death in about 50 percent of cases. With respect to AIDS, the Jamaican Ministry of Health estimates a case-fatality rate in Jamaica between 1982 and 2002 of 62% (NAC 2002). Other diseases, on the other hand, show lower mortality. For instance, the case-fatality rate during the malaria epidemic that hit Ethiopia in 1958 was estimated at 5%, with adults accounting for a relatively large proportion of cases (WHO 2003). From these examples it is evident that assigning a value to ϕ is a difficult task.

This difficulty increases due to disease complementarities (Dow *et al.* 1999): the probability of dying from infectious diseases is higher than the average probability across illnesses. Since we are interested in the overall adult mortality from all types of infectious diseases, microeconomic estimates are of limited help. We therefore rely on health aggregates to calibrate the mortality parameter. WHO (2001b) finds that fatalities from infectious diseases represent 53% of all deaths in Africa in 2001 for the male population between 15 and 80 years of age. We assign this value to the probability of dying from infectious diseases and pick $\phi = 0.47$.[11]

[11] This implies that life expectancy at birth is $15 + 1.47 \times 31.5 \simeq 61$ years in the high-prevalence steady-state. This is higher than life expectancies in sub-Saharan Africa, but we are ignoring non-infectious disease mortality.

Figure 3: Phase Diagram from the Complete Model with Benchmark Values



3.6 The Phase Portrait: Benchmark Case

Recall that the general equilibrium is described by the pair of difference equations (49) and (50), and the initial conditions (k_0, i_0). Figure 3 displays the phase diagram for the parameter values in Table 1. It plots the prevalence rate i_t against capital per effective unit of labor k_t.

Three different loci characterize dynamics. First, the $x(k_t, i_t) = 0$ 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 optimal investment is positive. The $x(k_t, i_t) = 0$ locus has its particular shape 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.[12]

The second locus, $\triangle k_t = 0$, plots (k_t, i_t) combinations along which k remains constant. It is given by equation (49) after imposing $k_{t+1} = k_t$. Capital per effective unit of labor declines above

[12] Our simulations suggest that, given any k, for any q arbitrarily close to zero (that is, for $\pi'(0)$ arbitrarily close to $-\infty$), there exists a value of i_t sufficiently close to 1 such that the optimal x_t is zero.

this locus and vice versa. The $\Delta k_t = 0$ line coincides with the $x(k_t, i_t) = 0$ curve to the right of point E. This is not a general result and depends on the choice of parameter values. For $q = 1$ and $\mu = 2$, for example, the $\Delta k_t = 0$ schedule would be located 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 $b > 0$.[13]

Note the U-shape of the $\triangle k_t = 0$ locus: the same infection rate can be associated with both high and low levels of the capital stock. This results from the tension between two effects of diseases on capital accumulation. 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 (49) 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. This shows up as a decrease in the denominator on the right-hand side of (49). The relative scarcity of efficiency labor causes its return to go up, as indicated in equation (34). This higher return may be high enough to actually increase savings and investment per effective unit of labor.

What is needed in the complete model for this positive effect to dominate is a relatively large stock of capital. To see this, set $x = 0$ since $\Delta k_t = 0$ coincides with the zero investment locus in our benchmark calibration. The $\Delta k_t = 0$ locus gives steady-state values of k for various (exogenous) values of i. This locus is now given by

$$k = \frac{p(i)s^I(1-\theta)w(k) + [1-p(i)]\, s^U w(k)}{1-\theta i}, \tag{53}$$

or,

$$\left[p(i)s^I(1-\theta) + \{1-p(i)\}s^U\right]\left(\frac{1}{1-\theta i}\right)\frac{w(k)}{k} = 1, \tag{54}$$

where $p(i) \equiv 1 - [1 - i\pi(0)]^\mu$. The first term (in brackets) on the left-hand side of (54) is a capital accumulation effect via discounting: as i decreases, $p(i)$ decreases and weight shifts to the higher savings propensity of the healthy. The second term is a capital dilution effect: as i decreases, there are more efficiency units of labor that lower the capital intensity (for a given K). When i decreases, the first term increases while the second term decreases. Since the $\Delta k_t = 0$ line is U-shaped, for any i there may exist two steady-state values, k_1 and $k_2 (> k_1)$ over a certain range of disease prevalence. At k_1, $\partial k/\partial i < 0$ while $\partial k/\partial i > 0$ at k_2. Let us consider a small neighborhood around k_1 for a moment. Since, over there, k_t goes up when i_t falls, $w/k = (1-\alpha)A + b/k$ goes down. It

[13] As we know from section 2, if production were not possible without capital (i.e., $b = 0$), the PT point on the $\triangle k_t = 0$ locus would be located at $(0, 1)$. However, as will become clear later, the model can generate multiplicity of balanced growth paths even in this case. Setting $b = 0$ simply prevents the existence of a poverty trap with zero growth and $k_t > 0$.

must then be the case that to maintain the steady-state equality above, the increase in the dilution effect is not enough to compensate for the increase in capital accumulation. So, at lower values of k, the capital accumulation effect dominates. Similar reasoning shows the capital dilution effect must dominate the accumulation effect at relatively higher values of k.[14]

The third locus characterizing dynamics is given by the downward sloping line, $\triangle i_t = 0$, defined by the equation

$$i_t = p(k_t, i_t), \tag{55}$$

along which the prevalence rate remains constant. It is defined wherever $x_t > 0$ and, in this area, $\triangle i_t < 0$ above the curve while $\triangle i_t > 0$ below it. To the left of the $x_t = 0$ schedule, preventive investment is zero, and the infection rate is always rising since $\mu\pi(0) = \mu a > 1$.

Figure 3 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 strictly positive rate. Vector fields indicate that the PT steady-state is a sink while UPT is a 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 SS 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. Notice that if i_t is relatively high (above the $x_t = 0$ locus), the economy always ends up at PT regardless of the value of k_t. In other words, even the richest economy could potentially slip into a new low-growth regime if the prevalence rate in the country becomes sufficiently large as a result of an exogenous disease shock for example.

Transition to the balanced growth path can exhibit interesting dynamics. In Figure 3, the trajectory starting from point M, initially shows slow growth and rising disease prevalence. The slow growth comes from the effect of diseases on mortality and productivity as well as lower savings due to a large portion of incomes being devoted to disease prevention. This preventive investment ultimately overcomes infectious diseases. The prevalence rate peaks and then declines monotonically as the economy takes-off into balanced growth. The take-off is fueled by capital accumulation shifting toward the higher savings of uninfected workers. In the limit, the growth rate converges to $\gamma^H \equiv (1-\alpha)s^U A - 1$. For a trajectory starting at point N, in contrast, the economy grows steadily

[14] The possibility that more adverse disease conditions can actually improve economic conditions is not novel to our model. In particular, 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 recent work by Acemoglu and Johnson (forthcoming). The interesting difference is the effect arises here not from mortality but morbidity.

as it converges to the balanced growth path and diseases decline monotonically.

Before concluding this section, it is important to compare this richer model's phase diagram (Figure 3) to the one of the stripped-down version (Figure 2). They are different mainly because now the contagion-probability function is continuous, the infected's saving propensity is not zero, and the external effect is stronger. More specifically, schedules $x(k_t, i_t) = 0$ and $\Delta i_t = 0$ no longer coincide in Figure 3 because of the continuous mapping between prevention investment and the disease-transmission probability. The capital level at the development trap is not zero since the infected individual's saving propensity is not zero. Finally, in Figure 3, the stronger negative externality arising from disease contagion causes that $x_t = 0$ and $\Delta i_t > 0$ for values of i_t sufficiently large. Key to understanding this point is recognizing that the return to preventive investment declines rapidly with μ. For instance, the probability of being infected after $\mu = 5$ matches becomes 1.0 for any $i_t\pi(x_t) \geq 0.5$, while it becomes 1.0 for any $i_t\pi(x_t) \geq 0.3$ after $\mu = 10$. Indeed, this last difference is the most important. In Figure 2, a very infected population converges to the high-growth path if the economy is sufficiently wealthy. However, in Figure 3, a highly infected population may never return to the high growth path regardless of its income level.

3.7 Two Alternative Cases

Next, we present two alternatives to our benchmark scenario by changing a and ϕ. First, we focus on the case where a is sufficiently low. Here the BGP is the unique steady state but economies with high prevalence rates go through a very slow convergence process. Secondly, we study what happens when ϕ is relatively high. In this case the development trap is no longer characterized by zero growth, and its existence do not depend on having $b > 0$. We conclude the section by performing robustness checks with respect to other parameter values.

Slow convergence without the low-growth trap

The existence of a low-growth trap depends on the value a takes. Recall that a positively affects the probability p_t of being infected after μ matches and, in particular, equals the probability of disease transmission in the absence of preventive investment. Hence as a falls, preventive investment becomes more efficient. When a falls sufficiently, diseases can be avoided at relatively low cost and the savings generated even at low incomes is enough to maintain a growing capital stock.

More specifically, for the benchmark parameterization, a PT still exists for $a \in (0.49, 1)$ though the prevalence rate falls below one. The low-growth trap vanishes when a falls below 0.49. For such low values, the $\triangle k_t = 0$ schedule disappears from the phase plane and optimal preventive investment is always positive ($x > 0$) for all (k, i) such that $k > 0.15$ and $i > 0.09$. As a result, no trap exists and all economies convergence to the unique BGP irrespective of initial conditions.

Figure 4: Phase Diagram for $a = 0.49$

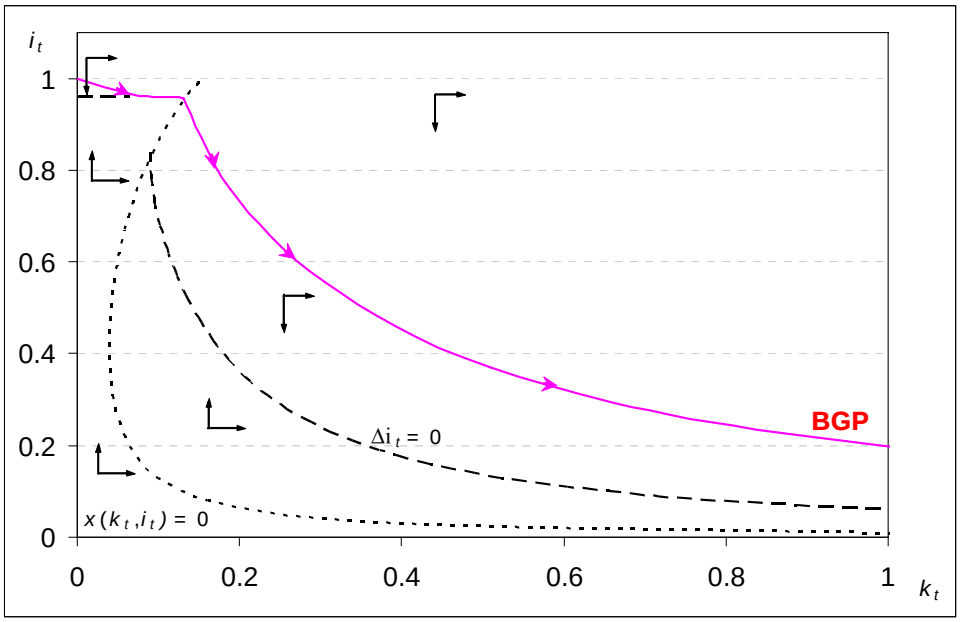


Figure 4 present the phase diagram for $a = 0.49$.

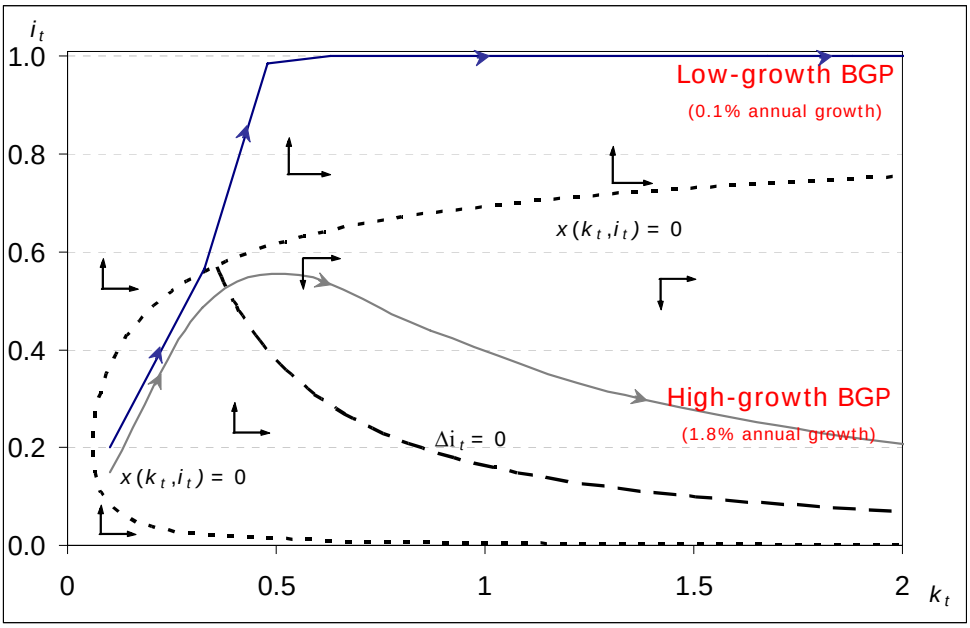
Multiple balanced-growth paths

The model's predictions are also sensitive to changes in the survival probability ϕ. The reason is that ϕ determines the rate at which infected individuals discount the future and, therefore, has a big impact on their saving propensity. When the survival probability is equal or higher than 0.72, the saving rate is sufficiently high to allow sustained growth in capital and output.[15]

For the next experiment, we assign a value of 0.73 to ϕ which implies that, in the low-growth trap, the long-run growth rate of output per capita will equal 0.1%, the average growth for sub-Saharan Africa from 1990 to 2003 (UNDP 2005). The phase diagram for this scenario is given in Figure 5. We observe that, as in the case where a is sufficiently low, the $\triangle k_t = 0$ schedule vanishes, implying that the capital stock grows from any point in the (k, i) plane. The figure illustrates dynamics for two economies: both start with the same level of physical capital but different prevalence rates (15% and 20%, respectively). The economy that starts with a prevalence rate of 15% experiences an increase in the infectious diseases during 2 generations, but eventually the prevalence rate drops to zero and the economy convergences to a balanced growth path with annual growth of 1.8%. The economy with an initial prevalence rate of 20%, show a continuous

[15] In the next subsection, we show that our benchmark results are robust to values of ϕ below 0.71.

Figure 5: Phase Diagram for $\phi = 0.73$



rise on the prevalence rate until everyone is infected. In the long-run, this economy does not invest in prevention and output per capita grows at 0.1% per year.

Notice that the fact that the $\triangle k = 0$ schedule plays no role in the results implies that neither does having a $b > 0$. We can see this from equation (54), and Figures 2 and 3. Given that $w/k = (1-\alpha)A + b/k$, the only significant role of b in the model dynamics is determining the location of the $\triangle k = 0$ schedule when $x = 0$.

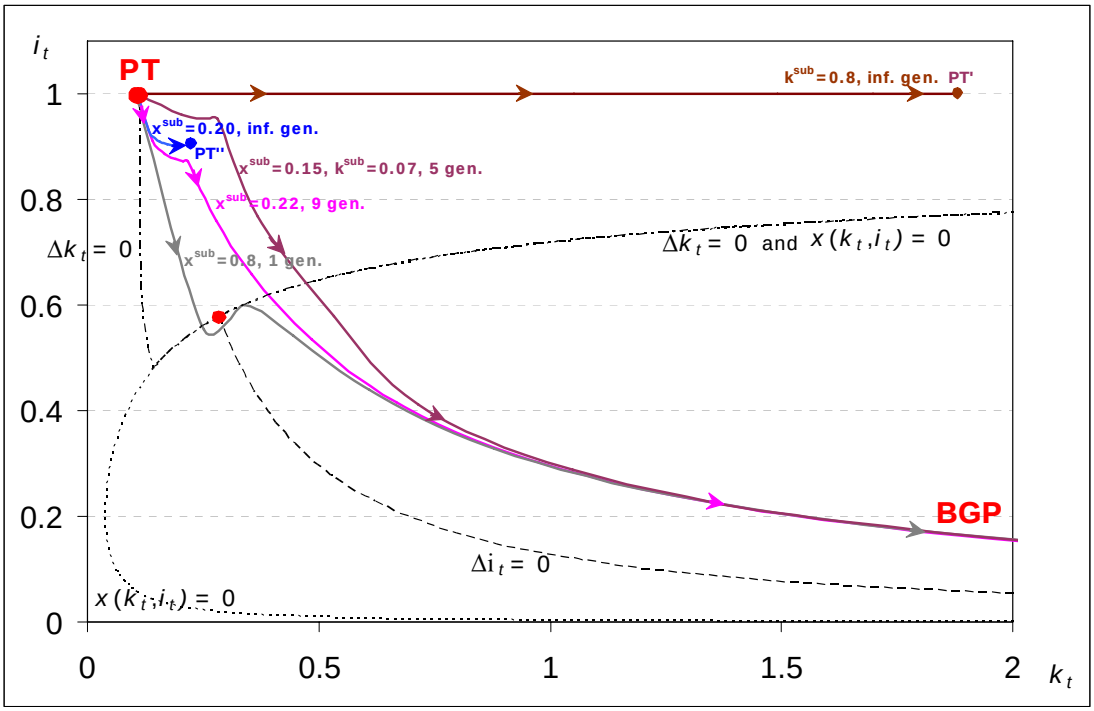
3.8 Policy Analysis

As we just saw, economies converge to either a poverty trap or a balanced growth path depending on initial conditions. For an economy that ends up at PT, an interesting question is whether subsidies are effective in taking it out of the trap. We explore this issue next. Figure 6 shows dynamics induced by international health subsidies (x^{sub}) and international capital-investment subsidies (k^{sub}).[16] The label to the right of each line denotes the characteristics of the policy package (x^{sub}, k^{sub}) and the number of generations during which it is implemented.

An immediate consequence of the model's dynamics described in the previous section is that

[16] Our experiments only consider the effect of pure subsidies from foreign sources, that is, foreign aid. However, the same qualitative results are obtained if subsidies are granted by the domestic government and financed with lump-sum taxes.

Figure 6: Subsidies to Health and Capital Investment



no k subsidy alone can take the economy to the BGP. As shown in Figure 6, when international donors supply $k^{sub} = 0.8$ (which amounts to 22% of GDP at PT) to each generation, an economy that starts at PT only moves to a slightly higher income poverty trap, PT'. Also, insufficient funds to help prevention may reduce the long-run prevalence rate below one but the economy grows enough to reach a higher income poverty trap. This is illustrated in Figure 6 when $x^{sub} = 0.20$ and the economy escapes PT but moves to PT''. Escaping the trap is, on the other hand, possible through health subsidies alone, provided that x^{sub} is large enough.

Given the method used to calibrate the model parameters, a x^{sub} equal to 0.22 (7.2% of GDP at PT) is the minimum amount required to take the economy from PT to BGP. The minimum health subsidy required will constitute our policy benchmark to which we compare other policy scenarios. In particular, a health subsidy of 0.22 has to be provided for at least 9 generations to achieve that goal. In addition, important scale economies are associated with x^{sub} in the sense that the number of subsidized generations required to escape the trap falls rapidly with x^{sub}. For instance, if we double preventive subsidies (i.e., $x^{sub} = 0.44$), the subsidy has to be provided for only 3 generations instead of 9. When $x^{sub} = 0.8$, this falls to only 1 generation.

Even though capital subsidies *per se* cannot take the economy to BGP, they can improve

the effectiveness of health subsidies. This is true provided that x^{sub} is sufficiently large – for the benchmark parameterization, x^{sub} needs to be at least 0.11. As shown in Figure 6, if instead of allocating 0.22 units of international aid only to health prevention, we choose $(x^{sub}, k^{sub}) = (0.15, 0.07)$, the required number of subsidized generations falls to 5. If instead of $(x^{sub}, k^{sub}) = (0.44, 0)$, we allocate these subsidies equally to capital and health investment so that $(x^{sub}, k^{sub}) = (0.22, 0.22)$, the number of generations declines from 3 to 2. But this type of complementarity between capital accumulation and health aid becomes weaker as x^{sub} becomes larger and the balance shifts in favor of health aid. For example, policy packages $(x^{sub}, k^{sub}) = (0.8, 0)$ and $(x^{sub}, k^{sub}) = (0.7, 0.1)$ need to be applied only during one generation, but a package $(x^{sub}, k^{sub}) = (0.6, 0.2)$ requires at least 2 generations.

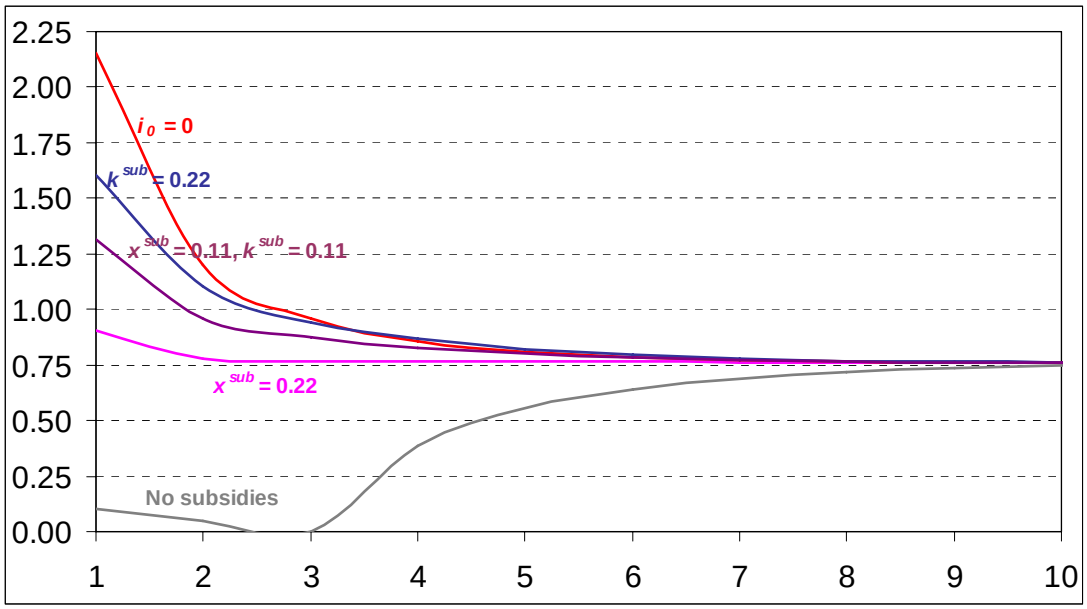
Next, we study policy in the two alternative cases considered in section 3.7. Both of them correspond to economies that are always growing.

Policy under the slow convergence case

An important question here is whether costs of infectious diseases remain large even when the economy is converging towards the high-growth path. Suppose that $a = 0.49$ and the economy starts developing from initial values $K_0 = 0.09$ and $i_0 = 0.96$. Remember that, in this case, there is no poverty trap. A prevalence rate of 0.96 is the maximum that the economy can endogenously reach for $a = 0.49$. Figure 7 presents time paths of the growth rate for different policy packages implemented every period. The comparison line $i_0 = 0$ presents the disease-free scenario. The effect of infectious diseases on the economy can be still substantial. If the economy does not receive international aid, growth-rate convergence takes several centuries. This case is represented in Figure 7 by the *no subsidies* time path. Growth rates are close to zero during the first 3 generations and do not reach half that for $i_0 = 0$ until generation 5. Indeed, growth even becomes negative when the economy starts investing in prevention with generation 3. Therefore, we conclude that even when development traps do not exist, the cost of infectious diseases is still sizeable.

Another result that comes out of Figure 7 is that subsidies to capital accumulation in this case are always more effective in raising the growth rate. In Figure 7 the package $(x^{sub}, k^{sub}) = (0, 0.22)$ takes the economy's growth rates closer to the $i_0 = 0$ path rather than the alternative packages $(0.11, 0.11)$ and $(0.22, 0)$. Nevertheless, it is important to note that subsidizing only capital may not be optimal if we take into account the evolution of life expectancy. It is straightforward that a package that includes x^{sub} will decrease the number of infected and, therefore, increase life expectancy at birth in period t. However, a package that only subsidizes capital accumulation does not impact the current generation. Hence, a central planner who values the life expectancy of different generations could choose an intermediate policy that includes both types of subsidies.

Figure 7: Effect of Subsidies without Poverty Trap ($a = 0.49$)



Policy under the multiple balanced-growth path case

Regarding policy effectiveness, our main results do not change. To abandon the low-growth trap, the economy needs investment in prevention, capital subsidies alone cannot help. In the case where $\phi = 0.65$, the economy requires a health subsidy of 0.11 for at least 3 generations to escape the trap. Capital subsidies do not provide much help because health subsidies are already very effective.

This scenario is also useful to illustrate the impact of changes in the institutional environment that, we can think, is captured by the parameter A. Like the survival probability, A directly affects long-run growth. However, changes in A could have at most a modest impact on a developing nation's long-run growth and health. Suppose that, initially, $\phi = 0.72$ and $A = 19$. The latter implies an annual steady-state growth rate of 1% in a zero prevalence economy instead of the benchmark 1.8%. In this case, the poverty trap appears at $(k, i) = (0.6, 1)$. In addition, assume that there is an improvement in the institutional environment that provides higher protection of property rights and enforcement of contracts, causing A to rise to 24.19.[17] An economy that was previously located in the poverty trap would now experiences a modest increase in long-run growth from 0% to 0.2% and no change in its population's health status.

[17] It makes sense to assume that its benchmark value (24.19) is the maximum which A can take. The reason is that A also affects economies that move along the high-growth steady state and are, therefore, on the technology and institutional frontier.

3.9 Summary of Results

We can summarize results from the numerical experiments as follows. First, unless the probability of infection is relatively low for zero preventive investment, there are two stable balanced-growth paths, one characterized by a low growth rate (which equals zero if the survival probability is relatively small) and the other by a relatively high growth rate. Second, any economy, regardless of its income level, can diverge towards the low-growth attractor (when it exists) if prevalence becomes sufficiently high. Third, subsidies to capital accumulation or non-health-related institutional improvements cannot fully substitute for health aid. In other words, a minimum health aid is a prerequisite to escape from the development trap. Nevertheless, health subsidies are strictly preferred to policy packages that combine capital and health aid only when a massive preventive investment is undertaken or the prevention technology offers relatively high returns. Finally, diseases impose a very high cost in terms of lost growth, regardless of which attractor dominates the economy's dynamics.

4 Diseases and Africa

Sub-Saharan Africa's (SSA) output per worker grew at an average annual rate of only 0.6% during $1950-2000$ against 1.9% for the US (Penn World Table 6.2). The continent also experiences a disproportionate share of the global infectious disease burden. Infectious diseases contribute about 64% to Africa's overall disease burden (DALYs), but worldwide only 30% (Global Burden of Disease; WHO, 2002). The probability of death of South-Saharan African men between ages $15-60$ is $40\%-60\%$ compared to less than 15% in developed countries (Murray and Lopez, 1997; Gakidou *et al.*, 2004). About 53% of this high adult mortality is due to infectious and parasitic diseases, HIV/AIDS, tuberculosis and malaria being the leading contributors (WHO, 2001b).

SSA is, therefore, characterized by low growth and high infectious-disease prevalence. Given that our theory offers a potential explanation for this scenario, this section examines some factors, through the lens of our theory, that help us understand it better.

Disease Ecology

According to our theory, Africa has a much larger probability of being gravitating towards a low-growth, high-prevalence trap attractor because its disease ecology is characterized by higher fatality rates (lower ϕ), higher exposure to the disease (bigger μ), higher probability of contracting diseases if exposed (higher $\pi(0) = a$), and lower efficacy of preventive actions (larger q).

Infectious diseases in Africa have been more virulent and destructive than elsewhere. Consider, for example, malaria which is a chronic persistent problem in SSA and not episodic like the plague,

influenza or small pox that affected pre-industrial Europe in waves. In the first place, much of Africa's malarial fatality is due to *plasmodium falciparum*, the deadliest strain of the malaria parasite that was absent or rare elsewhere. Secondly, the prevalence and severity of the disease depends on the continent's tropical climate. Low winter temperatures in temperate climates cut short the life-cycle of mosquitoes and offer a natural check. Third, the stability of the disease in Africa makes it difficult to control: control efforts have faltered due to the unusually high vectorial capacity of mosquitoes, some 2000 times higher than the critical value required to stop transmission (Gallup and Sachs, 2001). This problem has been compounded by rapidly spreading resistance, initially to DDT which was relatively efficient in eliminating the disease from many temperate regions during early-to-mid twentieth century, and now to antimalarial drugs (Nchinda, 1998).

A similar difference underlies HIV transmission. 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 diseases increase the transmission efficiency of the HIV virus. Such complementarity between various infectious diseases, each virulent on its own, plays an important role and has been a challenge for Africa's health policies. A recent study estimates that the interaction between malaria and HIV may have been responsible for 8,500 excess HIV infections and 980,000 excess malaria episodes in Kenya (Abu-Raddad *et al.*, 2006). Such co-infection may have also made it easier for malaria to spread to areas with high HIV prevalence.

The higher probability of contagion among Africans also results from the stronger immunity and resistance acquired by humans in the Old World over thousands of years as a consequence of its temperate climate and domestication of animals (Diamond, 1999). This evolution of a on its own could well have freed Europe from the tyranny of diseases. Imagine, for example, a population heterogenous in its disease resistance: as less resistant individuals die prematurely without being able to reproduce, the average a for the population would decrease over time, allowing an escape from diseases and stagnation.

The encounter with colonialism (Diamond, 1999) could have contributed as well. Colonization introduced new diseases to non-immuned populations in eastern, central, and southern Africa that were relatively more isolated than western Africa.[18] Previously endemic diseases often took the form of epidemics so much so that the period $1880-1920$ has been described as a time of tumultuous "ecological disaster" (Lyons 1993). By the mid-nineteenth century, tropical Africans were afflicted by most of the diseases of the temperate Old World and as we saw above for HIV and malaria,

[18] The flow of diseases was not one way. Through the slave trade, between $1500-1900$ African diseases like malaria, yellow fever and hookworm appeared in the warmer parts of the Americas, significantly affecting those populations (Patterson, 1993).

the concurrence of multiple infectious diseases could have then easily decreased the efficacy of preventive actions, and increased the probability of contagion and the mortality rate.

Finally, Kiple (1993) points out to another reason for the lower efficacy of preventive investment. African soils are typically acidic, nitrogen deficient and deprived of essential minerals. As a result, crops were protein and mineral deficient. Animal protein was relatively scarce and the few animals that were available were either quickly hunted down or fell prey to illnesses from tsetse flies. Although some animals were raised in West Africa, there was a taboo against drinking goat's milk and eating eggs. The sub-Saharan African diet, in other words, was nutrition deficient whereas it was through a nutrition-rich diet derived from fertile agriculture and domestication of animals that Europeans tackled diseases effectively (Diamond, 1999).

Institutional Factors

The theory above predicts that a lower value of the parameter q can propel a country into the better equilibrium. This opens the door to medical technology, but also to improvements in the public health system. Among others, interventions in the form of vaccination, nutritional supplement, information campaigns, environmental improvements can contribute to reduce q, and are best channeled through the public health delivery system. We now argue that Africa's institutional factors have advanced relatively much less than the western world's in that direction and have, therefore, according to our theory, contributed to the persistence of low economic growth and a high disease burden.

Even as colonialism brought new diseases to Africa, public health practices of the colonial powers made matters worse in some cases. For example, large-scale expensive medical campaigns were launched against single illnesses that made little dent on the overall problem. In some cases, disease-specific knowledge was either absent or knowledge gained from Europe had limited transferability to Africa (Dunn, 1993).[19]

But the main public health problem is a more contemporary one. Clearly, q and A must be correlated: countries that have better institutions are likely to also have more efficient public health systems and a higher adoption rate of frontier medicine. For example, the great diseases, plagues and subsistence crises disappeared from western Europe by the early 1700's partly due to the increasing stability of governments which enhanced administrative efficiency (Kunitz, 1993). In contrast, wars have been extraordinarily common and governments chronically unstable in Africa. Under such conditions, public health systems are ineffective due to corruption, inadequate provision and, quite often, lack of skilled manpower (World Bank, 1993).

[19] Even then, by the 1960's the African population was healthier because of the public health measures and western medical practices in effect since colonial times.

Unfortunately for Africa, a higher A may not bring a lower q if improved health interventions associated with stronger institutions are not effective. The following case illustrates that this is a very real possibility. Under the Garki Project in Nigeria, massive resources and manpower were devoted by the WHO and Nigerian government towards eradicating malaria from the Garki district in the 1960s. Despite steady and frequent public health interventions over seven years, there was no significant change in the malarial parasite rate in the population: existing technologies and knowledge turned out to have little effect on the transmission efficiency of mosquitoes (Gallup and Sachs, 2001; Molineaux and Gramiccia, 1980). Basically, the frontier q's turned to be inadequate to address the Garki district's disease problem.

The bottom line is that, since income *per se* is not the cause of ill-health in the worse equilibrium, general institutional improvements that only increase A offer limited scope of escaping high mortality and relatively slow growth.

5 Conclusions

We care about health not only because it fundamentally affects quality of life but also because it may affect economic outcomes. This paper makes the case that poor health due to infectious diseases has first-order effects on economic development.

The epidemiological model presented here explicitly incorporates rational disease behavior in a general equilibrium framework. A simplified version presented first shows how a lower savings-investment propensity associated with high mortality and morbidity caused by infectious diseases creates the possibility of a development trap. This trap is similar to the ones previously found in the literature in the sense that sufficiently wealthy economies can never fall into it.

A more general version of the model allows for less restrictive preferences and disease transmission dynamics. This exercise has revealed a much stronger negative externality intrinsically associated with the transmission of infectious diseases. Because of that, the model predicts that any economy, regardless of its income level, can be attracted to a low-growth trap if prevalence becomes sufficiently large.

An important consequence of these results is that income *per se* does not cause health when prevalence is high. Successful interventions should therefore be health specific (e.g. in the form of vaccination or nutritional supplements) and channeled via the public health delivery system. This is consistent with the work of Michael Kremer and Rachel Glennerster on vaccine research (e.g. 2000, forthcoming). For example, Kremer (2002), and more recently, Glennester, Kremer and Williams (2006) and Bernt *et al.* (forthcoming) try to device proposals "... to incentive private sector R&D investments in products for diseases concentrated in poor countries." Certainly, health aid in the

form of effective vaccination or drugs to cure major diseases in poor countries like malaria, and tuberculosis is a way out of the low-growth poverty trap present in our model. Our experiments also reveal that unlike general institutional improvements that have limited impact, institutional improvements of the quality of the health sector (public and/or private) is instrumental in raising aggregate productivity.

We hope that our work can offer theoretical foundations to a health and development literature that is overwhelmingly empirical, usually analyzing data of poor quality. This is specially urgent in its macroeconomic side, which provides mixed results regarding the relationship between health and income. Let us focus on two main contributions that deliver opposing results to see how our theory can help reconcile them: Weil (2007) and Acemoglu and Johnson (forthcoming in JPE). The former focuses on the impact of morbidity in partial equilibrium. We could think about it, in terms of our model, as a very interesting attempt to more accurately estimate θ. Hence, a positive impact of health on income was expected. The latter paper, on the other side, focuses on mortality in general equilibrium and, in particular, on the impact of life expectancy (LE) on income per capita and economic growth. We shall argue that, in light of our theory, Acemoglu and Johnson's results are not completely surprising because of the specific cross-section and time-series dimensions of their sample.

These last two authors find that changes in LF have no significant impact on long-run economic growth. However, their main sample excludes Africa – African data is only used tentatively due to bad quality. Their main sample is, therefore, mainly composed of nations with relatively low probability of being gravitating towards a development-trap equilibrium. But then notice that, in our model, nations that move towards the BGP end up with the same long-run growth rate, independently of its initial level of LE. Their results have also the same prediction regarding income. It is straightforward that if we had used a neoclassical growth framework instead, our theory would say that LE does not predict long-run GDP per capita in the good equilibrium. We could even argue that our theory can help to explain why Acemoglu and Johnson find that initial LE is related to a short-run decline in GDP. Even though we do not have population growth in the model, we have found in the simulations very low growth rates, sometimes negative, following the scape of the bad equilibrium. Basically, growth takes time since the economy needs first take care of the disease problem. This effect is further amplified because there is a morbidity related effect on GDP, and because healthier cohorts will contribute to capital accumulation only several decades later.

The theory also offers several testable predictions that empiricists can exploit. The most important ones are the following. First, in the long run, the model implies that a good candidate to have growth effects is mortality, and that morbidity can have at most level effects. Second, both

mortality and morbidity are important determinants of the saving rate and prevention investment. Third, morbidity can also generate dilution effects on capital intensity and these effects are stronger for higher levels of development. Last but perhaps more important, the effects of health on capital accumulation that dominate dynamics should show up in the data through important nonlinearities in the growth process.

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