

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 (premature death) and morbidity (lower productivity and quality of life). The theory offers three main insights. First, higher disease prevalence implies lower saving-investment propensity. Preventive behavior can partially offset this when the prevalence rate and negative disease externality are relatively low. Secondly, infectious diseases can generate a low-growth trap where income alone cannot push an economy out of underdevelopment. This is distinctly different from development traps in the existing literature. Since income *per se* does not cause health in this equilibrium, successful interventions have to be health specific. Thirdly, a more favorable disease ecology propels the economy to a higher growth path where health and income co-evolve and infectious diseases disappear. Even so, diseases significantly slow down convergence. These results suggest the empirical relationship between health and income at the aggregate level may be more nuanced than realized.

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1 Introduction

Health and income are each elemental to welfare but it is perhaps their joint relationship that has most intrigued researchers. Countries that are poor in per capita income are also likely to be poor in health. This high positive correlation between income and health is well documented by demographers, economists and epidemiologists (Soares, 2007), with sub-Saharan Africa's prolonged stagnation and overwhelming disease burden offered as a case in point.

But does better health facilitate economic development? Or is it development that drives health improvements? While there is consensus that the relationship runs both ways, it is unclear if one direction is expected to be dominant. It also raises policy questions about how to effectively improve national health and economic growth.

This paper offers a theoretical framework tailored to address these issues. Motivated by evidence that a disproportionate share of developing country disease burden is due to infectious diseases,¹ we incorporate rational infectious disease behavior into a growth model.² Infection spreads from random exposure to disease vectors, susceptibility to which depends on preventive health investment and the disease ecology (climate, vectorial capacity, social practices). Diseases cause premature death as well as lower productivity and quality-of-life among the infected. The mortality effect makes infected individuals less inclined to save, morbidity makes them less able to do so.³ These costs create incentives for prevention which can partially offset them as long as the (negative) disease externality is not particularly severe.

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

Our second main result is that income does not cause health when diseases are widespread, *irrespective* of the level of development. The disease externality overwhelms incentives for prevention in this case and foreign aid in the form of income transfers has little effect on health or development.⁴ This result is in sharp contrast to development traps in the existing literature (see,

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

²Ignoring the effect of rational behavior can convey an incorrect view of disease dynamics and the effectiveness of public health interventions (Geoffard and Philipson, 1996). Philipson 2000 offers an excellent survey of the issues in economic epidemiology.

³There is some direct evidence that longevity 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.

⁴Rajan 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)

for example, Azariadis and Stachurski, 2005 for a review).

Numerical experiments show the cost of health aid needed to push an economy out of the low-growth equilibrium can be substantial. Simultaneously targeting capital accumulation and preventive health reduces this cost. Even when the economy converges to the high-growth path, the disincentive effect of diseases on saving-investment is sizeable enough to slow growth rate convergence by several generations.

These results shed some light on the “income versus public health” debate. On one side of the debate, McKeown (1976) and notably Fogel (1997) have argued that nutrition played a vital role in Britain’s mortality transition and facilitated economic growth. On the other side, Preston (1996) and more recently Cutler *et al.* (2006) and Soares (2007), present evidence that public health initiatives and medical improvements rather than income gains caused the worldwide mortality declines of the past century.

As long as an economy is converging to the higher growth path in our model, there is a two-way feedback between health and income. Higher income assists prevention, lowers the incidence of communicable diseases and generates stronger incentives for economic growth. We see this pattern as more relevant to the historical experience of Britain, Western Europe and its offshoots where diseases were not endemic and severely costly (except for brief spells). For developing countries, situated as they mostly are in the tropics, a development trap is more plausible. Since income deficiency is not the cause of poor health in this equilibrium, our theory suggests marked health improvements can occur only due to exogenous improvements in public health or medical innovations. Somewhat surprisingly, this escape from stagnation is at first accompanied by economic stagnation lasting several generations. The favorable disease environment initially creates stronger incentives for health investment over other forms of, directly growth-augmenting, capital investment.

There has been a recent surge in research on health and development. Despite compelling microeconomic evidence that health is important for economic outcomes (see, Strauss and Thomas, 1998, Deaton, 2003 for example), the macroeconomic evidence has been mixed. Empirical works such as Bloom and Canning (2005), Gallup and Sachs (2001) and Lorentzen *et al.* (2006) attribute Africa’s persistent poverty to endemic infectious diseases (like malaria) and 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 very small if any positive effect of health on income. These authors argue that the increase in population resulting from better health outweighs the productivity effects and therefore GDP per capita may have actually slightly decreased in their

empirically estimate the effects of aid on infant mortality using a dataset covering 118 countries from 1970 to 2004. They find that although overall foreign aid does not have a statistically significant effect on infant mortality, *health aid* does.

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. He finds that eliminating health differences among countries will reduce the variance of log GDP per worker by about 10% which is economically significant, but substantially smaller than estimates from cross-country growth regressions. While our theory offers a framework to parse this conflicting evidence, it is important to note that none of these empirical works explicitly allow for nonlinearities in the relationship between health and growth.

This paper is related to several theoretical works incorporating mortality in growth models. Among others, Blackburn and Cipriani (1998), Chakraborty (2004), Cervellati and Sunde (2005), Doepke (2005), Kalemli-Ozcan (2002) and Soares (2005) variously consider the effect of declining child and adult mortality on fertility, human capital, the demographic transition and economic growth. Theoretical work on the microfoundation of diseases and economic growth is more limited. Momota *et al.* (2005) generate disease cycles in a general equilibrium setup. 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 proposed in Galor (2005), Galor and Moav (2002) and Galor and Weil (2000). In our model, a stagnant economy starts enjoying modern economic growth when prevalence rates fall sufficiently due to exogenous improvements in medicine, public health or the disease environment.

The remainder of this paper is organized as follows. In section 2, we specify a simplified version of the model to illustrate the key mechanisms. This simple model does not encompass the complexities of real world disease behavior and its dynamics. These are incorporated into a more thorough analysis in Section 3. Section 4 attempts to understand sub-Saharan Africa's twin problems of disease and underdevelopment through the lens of our theory. Section 5 concludes by relating the model's implications with recent evidence on health and growth.

2 A Simple 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

⁵We do not explicitly model childhood nor do we take into account child mortality. Children's consumption is subsumed into their adult parent's. Our rationale for focusing on adult mortality is that the enormous life expectancy improvements enjoyed by developing countries over the past fifty years has been primarily due to sharp declines in infant and child mortality from low-cost interventions and technology transfers. Adult mortality has declined relatively less, remains high in developing countries (World Bank, 1993) and disproportionately so due to infectious diseases.

To the extent that child mortality is still a problem, we are ignoring its effect on fertility and investment in childhood human capital. Childhood morbidity from infectious diseases which have life-long repercussions is, however, implicitly

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 a utility loss from being infected: the individual derives a utility flow of $\delta u(c)$ instead of $u(c)$ from a consumption bundle c , where $\delta \in (0, 1)$. We interpret this as a quality-of-life effect. Thirdly, an infected young individual faces the risk of premature death and may not live through his entire old-age.

Young individuals can undertake preventive health investment, x_t , early in life. This takes 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 they can also take the form of utility costs as in Geoffard and Philipson (1996) for instance.

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

$$p(x_t) = \mu\pi(x_t)i_t, \quad (1)$$

where $\pi(x_t)$ 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

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

Let $\mu\pi_1 < 1 < \mu\pi_0$, that is, an infected person infects more than one susceptible person in the absence of prevention but less than one (on average) if susceptible populations engage in prevention.

Equation (1) exhibits a negative externality that characterizes communicable diseases. When an individual chooses preventive health investment *ex ante* – before he meets an infected 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.

incorporated in the cost of disease parameters.

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 vector more generally. For a disease like AIDS, it can be directly related to the number of sexual partners or needle-sharers.⁶ 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.

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,

⁶In 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.

we posit a firm-specific constant-returns technology exhibiting learning-by-doing externalities

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

where A is a constant productivity parameter, and \bar{k} denotes the average capital per effective unit of labor across firms.⁷ For our model to generate a balanced-growth path with strictly positive growth we assume that $(1 - \alpha)A > 1$.

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

$$w_t = (1 - \alpha)Ak_t \equiv w(k_t), \quad (4)$$

$$R_t = \alpha A \equiv R. \quad (5)$$

2.3 Preferences

Preferences and economic 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 now, that the period utility function is linear. The individual maximizes lifetime utility

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

subject to the budget constraints

$$c_{1t}^U = w_t - x_t - z_t^U \quad (7)$$

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

where z denotes savings and x is given by decisions made early in period t .⁸ Hereafter we tag variables by U and I to denote decisions and outcomes for uninfected and infected individuals, respectively.

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

$$\delta [c_{1t}^I + \beta \phi c_{2t+1}^I], \quad (9)$$

⁷The choice of a simple Ak mechanism is only for tractability. The story generalizes when saving behavior determines growth (in closed or open economies) via innovation and factor accumulation, as 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.

⁸Implicitly x is financed by zero-interest loans taken early in youth from the rest of the world and repaid after the labor market clears.

subject to

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

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

where 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.⁹ Clearly transfers per surviving infected individual will be

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

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, $c_{1t}^U = c_{2t+1}^U = z_t^U = \tau_{t+1} = 0$, $c_{1t}^I = (1 - \theta)w_t - x_t$, $z_t^U = w_t - x_t$, and $c_{2t+1}^U = R_{t+1}(w_t - x_t)$. Substituting these into expected lifetime utility gives the two indirect utility functions

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

$$V^I(x_t) = \delta [(1 - \theta)w_t - x_t]. \quad (14)$$

At the beginning of 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

$$p_t(x_t)V^I(x_t) + [1 - p_t(x_t)]V^U(x_t), \quad (15)$$

at the beginning of period t . Given that prevention is either investing x or nothing at all, the optimal decision is x if and only if expected lifetime utility is higher in doing so

$$p_t(x)V^I(x) + [1 - p_t(x)]V^U(x) > p_t(0)V^I(0) + [1 - p_t(0)]V^U(0). \quad (16)$$

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

⁹Alternatively, we could have assumed perfect annuities market with qualitatively similar results.

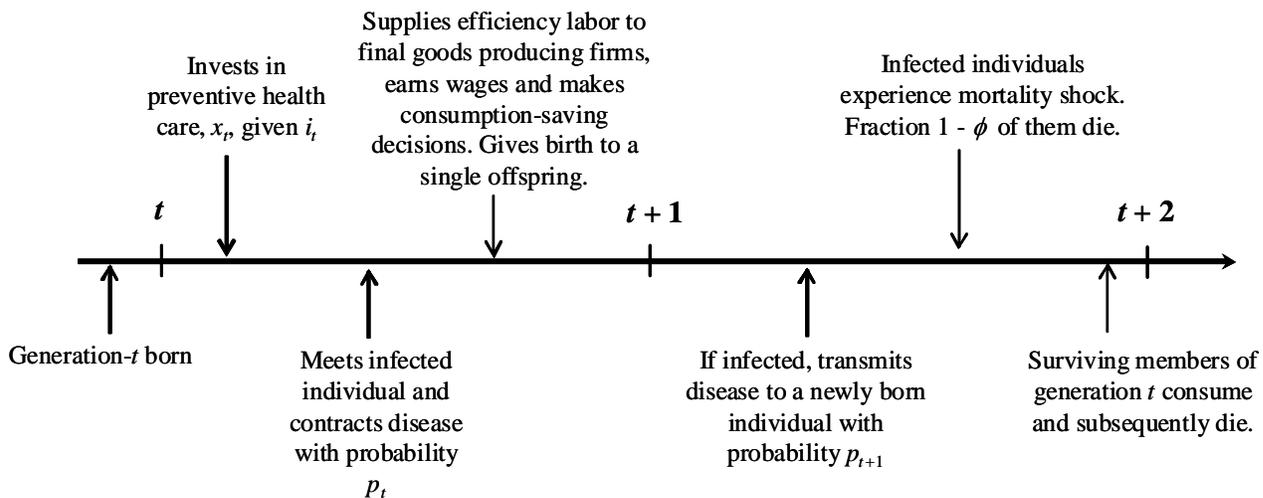


Figure 1: Timeline of Events

2.4 Dynamics

With a continuum of young agents of measure one, aggregate savings at t is

$$S_t = (1 - p_t)z_t^U, \quad (17)$$

and the asset market clearing condition

$$K_{t+1} = S_t. \quad (18)$$

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,

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

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 infection dynamics into the asset market clearing condition leads to

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

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

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

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 this. The first two consist of the locus along which disease prevalence remains constant ($\Delta i_t = 0$) and the locus for which capital per effective unit of labor remains constant ($\Delta k_t = 0$). The third locus is the general equilibrium version of (16) which separates positive preventive investment from zero investment.

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. Specifically, disease prevalence increases when $x_t = 0$ and decreases if $x_t = x$. From expressions (1), (5), (13) and (14) we can rewrite (16) as

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

Substituting for equilibrium wages from (4) we obtain

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

It is easy to show that Φ is decreasing in k_t , $\lim_{k \rightarrow \infty} \Phi(k) = 0$, and $\Phi(0) = \beta R / [\mu \pi_1 (\beta R - \delta)] > 1$. Hence the locus $i_t = \Phi(k_t)$ is downward sloping with 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 $\Delta k_t = 0$ locus depending on whether or not individuals invest in preventive care.

Case 1: $x_t = 0$

Equations (1) – (4) and (20) imply

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

so that

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

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 $i_t \rightarrow 1$. Hence the expression on the left becomes smaller than 1 for a sufficiently large disease prevalence. In addition, the expression becomes undefined as $i_t \rightarrow 0$. Hence there exists $\hat{i} \in (0, 1)$ such that $\Delta k_t < 0$ for all $i_t > \hat{i}$ and $\Delta k_t > 0$ for all $i_t < \hat{i}$. This threshold is given by

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

The expression on the right in (23) depends on π_{t+1} and 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{i} can therefore be discontinuous with $\hat{i}(\pi_1) < \hat{i}(\pi_0)$.

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

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

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

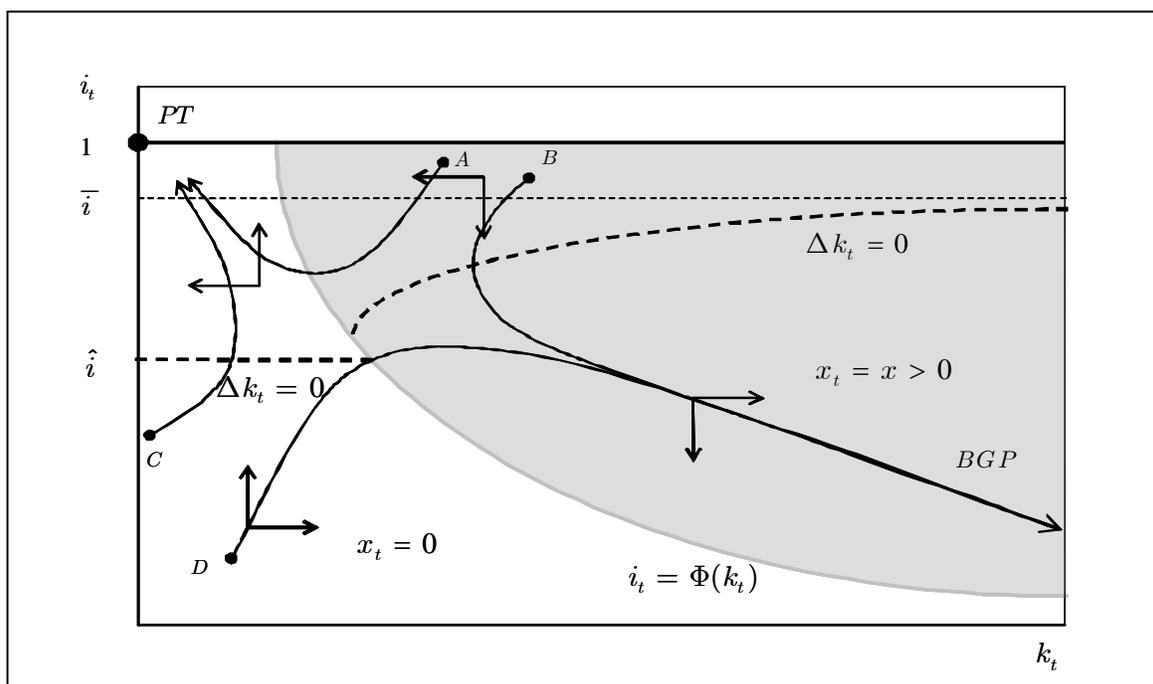
$$i_t = \bar{i} = \frac{(1 - \alpha)A - 1}{[(1 - \alpha)A - \theta\mu\pi_1] \mu\pi_1}. \quad (25)$$

We obtain \bar{i} 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 $\bar{i} < 1$ if and 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{i} .

If $\bar{i} > 1$, 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. 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

Figure 2: Phase Diagram for the Simple Model



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 or large prevalence rates initially. Economies such as B and D in Figure 2 start with more favorable initial conditions to converge to BGP .

But initial conditions only partly determine convergence dynamics. For a given (k_0, i_0) , the attractor that dominates dynamics depends on disease ecology and costs and technological productivity. We postpone a discussion of these factors for the next section. For now, one aspect of the dynamics worth emphasizing is that PT is a standard trap in the sense that financial aid can propel an economy from 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 capital stock comparable to B , the economy will start converging to 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, this aspect of the poverty trap does not

generalize.

We conclude this section by noting 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 to sustain perpetual growth when everyone is infected but takes on a higher value, allowing sustained growth, for an uninfected population. Relatively capital-poor economies with relatively large disease prevalence end up in the trap.

3 The Complete Model

This section generalizes the simpler model above by incorporating a more realistic disease transmission mechanism. While the trap can exhibit positive rates of output growth now, it also gets stronger in that income transfers can no longer deliver an economy from it to the higher growth path.

We also modify the technology and preferences in order to consider parametric values established in the 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,

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

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 stronger externality 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 that takes on continuous values, the probability that a young individual gets infected from a matching is

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

This function fulfills the following properties: $\pi' < 0$, $\pi(0) = a$, $\pi(\infty) = 0$, and $\pi'(0)$ goes to $-\infty$ as q goes to zero. As before we restrict $\mu\pi(0) = \mu a > 1$.

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

3.2 Preferences and Production Technology

Next we assume $u(c)$ 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

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

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

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

subject to (10) and (11). As before 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:

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

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

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

We assume a CES utility function

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

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 exceed one.

In order to ensure the last restriction, we modify the production technology to

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

where $b > 0$ captures “natural endowments” such as trees and animals that allow for positive consumption levels in the absence of physical capital. For b sufficiently large, consumption will be above one. The equilibrium return to labor now becomes

$$w_t = (1 - \alpha)Ak_t + b \equiv w(k_t). \quad (34)$$

As will be clearer below, $b > 0$ is not necessary to obtain the main results in this section.

3.3 Preventive Investment Decision

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

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

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

Substituting these into lifetime utility gives the two indirect utility functions

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

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

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

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

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

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

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.¹⁰

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

$$z_t^U = s^U [w(k_t) - x(w_t, i_t)] \equiv z^U(k_t, i_t), \quad (41)$$

and

$$z_t^I = s^I [(1 - \theta)w(k_t) - x(w_t, i_t)] \equiv z^I(k_t, i_t), \quad (42)$$

where,

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

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

Substituting the savings functions into indirect utility obtains

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

and

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

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

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

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

¹⁰The 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.

(46) is the marginal utility cost of that investment, since health investment entails a lower current consumption. The right-hand side constitutes the marginal benefit, in the form of higher net utility from lowering one's chance of contracting diseases. Optimal health investment is zero as long as the utility cost dominates, that is, returns to health investment are negative at $x_t = 0$. Intuitively we expect this to occur at levels of low income and high prevalence rates. Private actions have a negligible impact on the chance of leading a healthy life in such situations.

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

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

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

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

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 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

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

while disease dynamics evolve as before

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

Table 1: Benchmark Parameter Values

β	0.99 ^(31.5×4)	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

Equations (49) and (50) 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 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 convergence dynamics cannot be studied algebraically, it is easy to derive the balanced growth rates. 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

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

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

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

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

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 parameters and establish dynamic properties through simulation. Given the difficulty of assigning precise values to the disease related parameters, these numerical experiments should be seen as a way to dig deeper into the qualitative impact of disease costs, ecology, policy interventions and institutions.

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

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 $0.99^{31.5 \times 4}$ to the discount factor (β), that is, 0.99 per quarter which is standard in the real-business-cycle literature. We set the elasticity parameter $\sigma = 1$ in the utility function (log preferences). The production function has three parameters: the technology parameter A , the capital elasticity α , and the labor productivity coefficient b . We normalize $b = 1$ to ensure that consumption levels are bounded above one and, as a consequence, utility when alive remains positive. We set $\alpha = 0.67$; we are then looking at a broad concept of capital that includes physical, human and organizational capital. The value for A , in turn, is chosen so as to reproduce an annual long-run growth rate of 1.8% in the low-prevalence steady state. 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 $s^U(1 - \alpha)A = 1.018^{31.5}$, 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 – where 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), and 475 (current US\$) is sub-Sahara Africa’s average GDP per capita, also 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 $q = 0.14$ which satisfies the condition that $a > 1/\mu$. We also perform sensitivity analyses for (μ, q) using $(2, 0.55)$ and $(10, 0.06)$.

We have more guidance on parameters that govern the cost of diseases. 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 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

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 .

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 prevention is positive. The $x(k_t, i_t) = 0$ locus has its particular shape because of

¹¹This implies that life expectancy at birth is about 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.

the way prevalence and income affect incentives. For low levels of disease prevalence ($i_t \rightarrow 0$), the risk of catching an infection is so low that prevention is not necessary. At high levels of disease prevalence ($i_t \rightarrow 1$), in contrast, the productivity of prevention becomes vanishingly small as the disease externality from sequential matching outweighs the benefits from prevention.¹²

The $\Delta k_t = 0$ locus on Figure 3 is given by equation (49) after imposing $k_{t+1} = k_t$. Capital per effective unit of labor declines above 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 $b > 0$.

Note the parabolic $\Delta k_t = 0$ locus: the same infection rate can be associated with both high and low levels of capital per effective worker. This results from a tension between two effects. Diseases have a negative effect on capital accumulation via their effect on mortality (which lowers incentive to save) and productivity (which lowers ability to save). This is what the numerator on the right-hand side of equation (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.

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

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

where $p(i) \equiv 1 - [1 - i\pi(0)]^\mu$. The first term on the left-hand side of (53) is the effect of diseases on capital accumulation: as i decreases, investment shifts towards the higher savings propensity of the healthy. 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 U-shaped, 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 . Hence, at lower values of k , the capital accumulation effect dominates while the dilution effect dominates at relatively higher values of k .¹³

¹²Our 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.

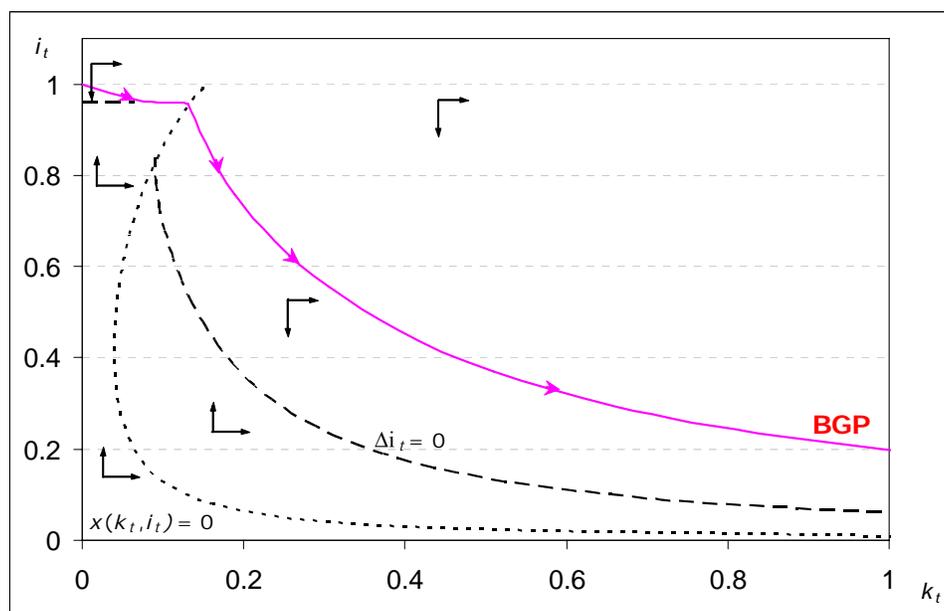
¹³The possibility that more adverse disease conditions can actually improve economic conditions is not novel to

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 can slip into a low-growth regime if the prevalence rate becomes sufficiently large, from an exogenous disease shock for instance.

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. Prevalence 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 γ^H . For a trajectory starting at point N , in contrast, the growth is steady as it converges to the BGP and diseases abate.

Unlike Figure 2, the capital stock in the development trap Figure 3 is not zero since infected individuals do save. Also, in Figure 3, the stronger externality from disease contagion induces zero prevention even at high income levels when the prevalence rate is sufficiently high. Key to understanding this point is recognizing that the return to prevention declines rapidly with μ . For instance, the probability of being infected after $\mu = 5$ matches becomes 1 for any $i_t\pi(x_t) \geq 0.5$, while it becomes 1 for any $i_t\pi(x_t) \geq 0.3$ when $\mu = 10$. Indeed, this last point is important. In Figure 2, a highly infected population converges to the high-growth path if the economy is sufficiently wealthy. In contrast, in Figure 3, it may never converge to the high growth path regardless of income level.

A remaining question is how disease ecology and costs interact with initial conditions to determine the growth path. Ecology determines susceptibility to infection, which depends on the number of encounters (μ) and the probability of catching the disease in each such encounter (a, q). As we increase μ (which also means we lower q because their values are jointly determined), the state space within which people invest in prevention shrink and, consequently, it is easier to end up in the trap. Higher values of a have a similar effect. The effect of disease costs, on the other hand, depends on which parameter we look at. Higher mortality risk (lower ϕ) has the same effect as higher μ , making a trap more probably for given initial conditions. But as morbidity costs increase (higher θ , lower δ), it elicits stronger preventive behavior which makes PT a less likely equilibrium outcome.

Figure 4: Phase Diagram for $a = 0.49$ 

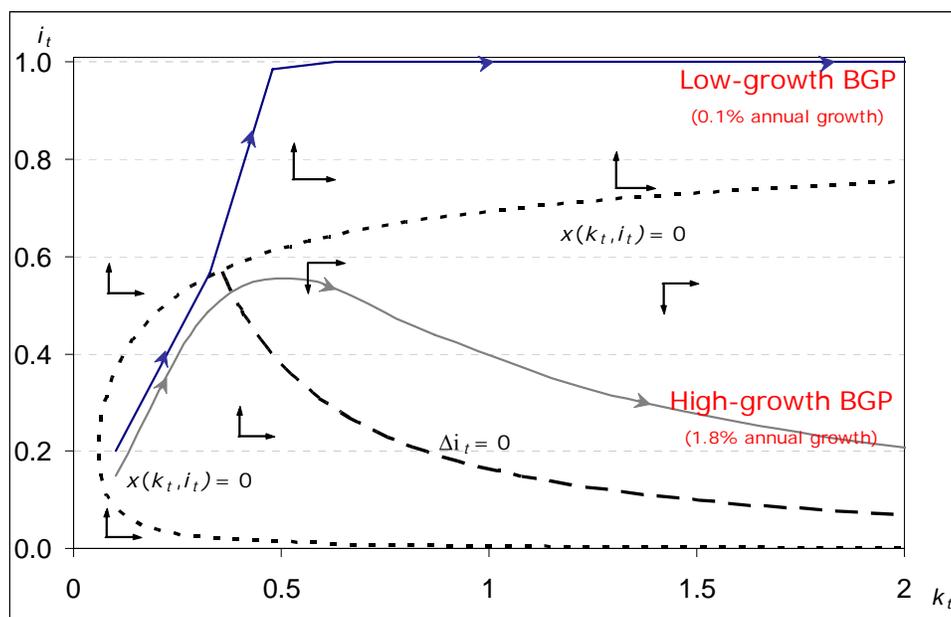
3.7 Two Alternative Cases

We present two alternatives to our benchmark scenario by changing a and ϕ . First, we examine 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 consider how the development trap is no longer characterized by zero growth when ϕ is relatively high. We conclude the section by performing some robustness checks.

Slow Convergence without a Low-Growth Trap

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 prevention. 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* 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 $\Delta k_t = 0$ schedule disappears from the phase plane and optimal preventive investment is always positive for all $(k, i) > (0.15, i > 0.09)$. Hence, no trap exists and all economies converge to the unique *BGP* irrespective of initial conditions as Figure 4 shows.

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

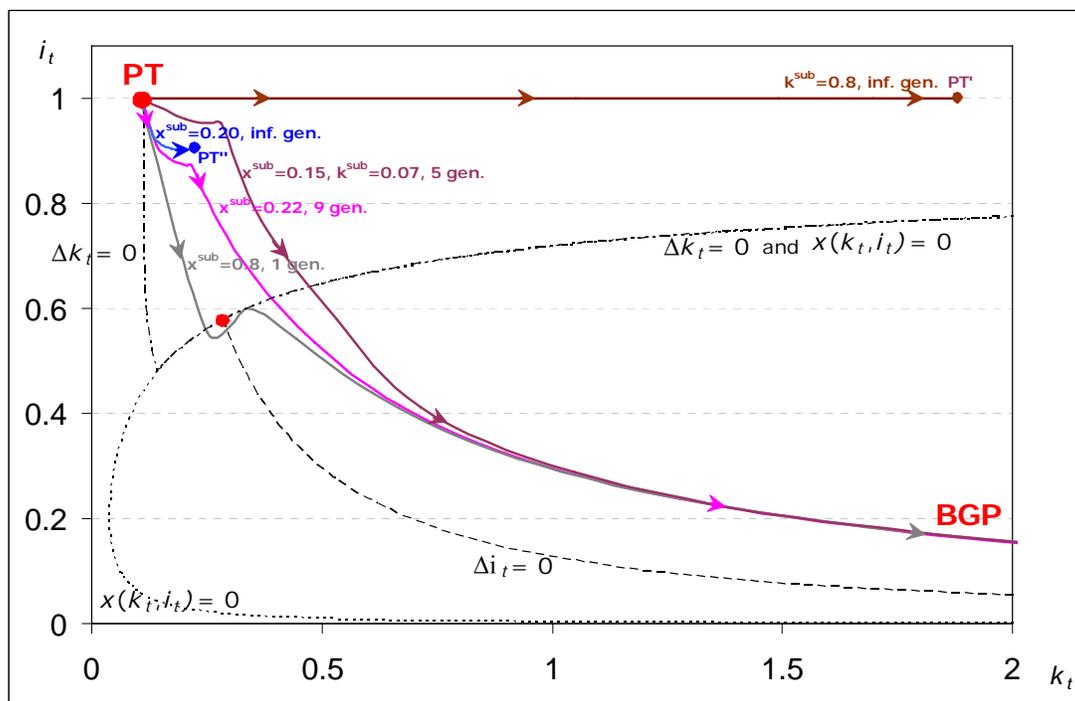
Multiple Balanced-Growth Paths

The model's predictions are also sensitive to the survival probability ϕ since it determines the rate at which infected individuals discount the future and, therefore, has a significant effect on their saving propensity. When the survival probability exceeds 0.72, the saving rate is high enough to sustain output growth.

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 shown in Figure 5. As with sufficiently low a , the $\Delta k_t = 0$ schedule vanishes and positive growth occurs 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 disease prevalence for 2 generations, after which it abates as the economy converges to an annual growth rate of 1.8%. The economy with an initial prevalence rate of 20%, shows a continuous rise in prevalence 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.¹⁴

¹⁴The fact that the $\Delta k = 0$ schedule plays no role in the results implies that neither does a positive b . The only significant role b plays in the dynamics is determining the location of the $\Delta k = 0$ schedule when $x = 0$.

Figure 6: Subsidies to Health and Capital Investment



3.8 Policy Analysis

For an economy that ends up at PT , an interesting question is whether external subsidies can take it out of the trap. Figure 6 shows dynamics induced by international health (x^{sub}) and capital-investment subsidies (k^{sub}).¹⁵ The label to the right of each line denotes the characteristics of the policy package (x^{sub}, k^{sub}) and the number of generations over which it is implemented.

An immediate consequence of the model's dynamics is that no k subsidy alone can take the economy to the BGP . For example, as Figure 6 shows, when international donors supply $k^{sub} = 0.8$ (22% of GDP at PT) to each generation, an economy that starts at PT only moves to a slightly higher income trap, PT' . The figure also illustrates how funds like $x^{sub} = 0.20$ that are insufficient for prevention can reduce the long-run prevalence rate below one but increase income levels only slightly, from PT to PT'' . Escaping the trap is possible through health subsidies alone provided x^{sub} is large enough.

Given the method used to calibrate the parameters μ and q , a x^{sub} equal to 0.22 (7.2% of GDP

¹⁵These experiments consider only the effect of pure subsidies from foreign sources, that is, foreign aid. Similar qualitatively results obtain if subsidies are financed domestically with lump-sum taxes.

at PT) is the minimum required to take the economy from PT to BGP . This minimum will be our 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., $x^{sub} = 0.44$), it has to be provided for only 3 generations instead of 9. When $x^{sub} = 0.8$, this falls to only 1 generation.

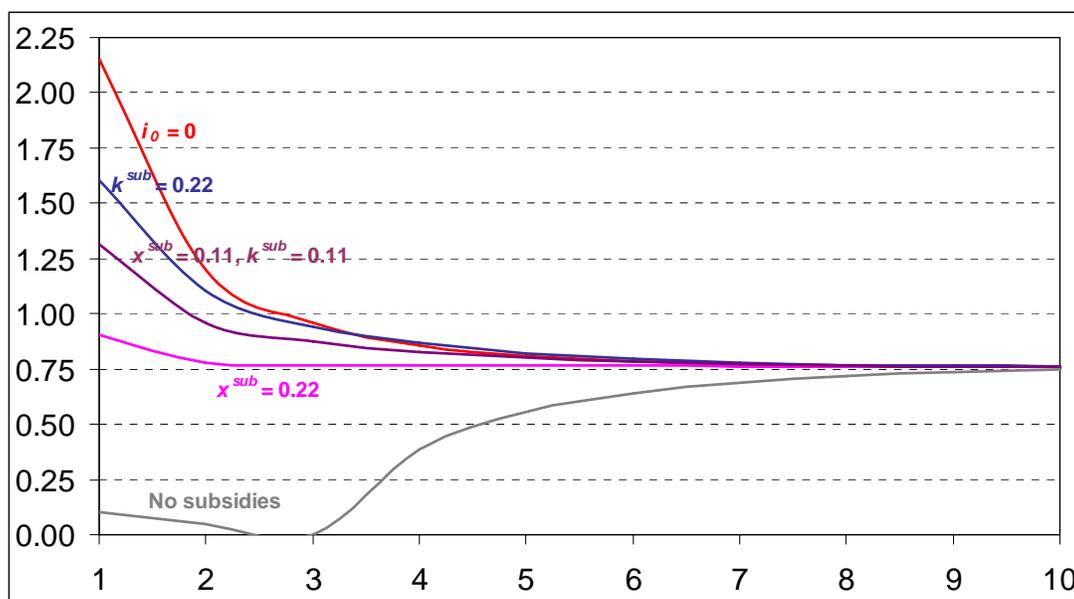
Even though capital subsidies alone cannot take the economy to BGP , they can improve the effectiveness of health subsidies. This is true provided that x^{sub} is sufficiently large – for the benchmark parameterization, x^{sub} needs to be at least 0.11. In Figure 6, if instead of allocating 0.22 units 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.

We study next policy in the two alternative cases considered in section 3.7 where the economy is always growing.

The Case of Slow Convergence

An important question here is how large disease costs are as the economy converges to the high-growth path. Suppose $a = 0.49$ and economic development starts from $K_0 = 0.09$ and $i_0 = 0.96$. Recall 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 figure shows how substantial the cost of diseases can be. If the economy does not receive international aid, growth-rate convergence takes several centuries. This case is represented in 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 when the disease externality is low enough to make this worthwhile.

We can also use the *no subsidies* line to understand the effect of exogenous health improvements

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

on an economy located at PT . Suppose such health improvements, for example large-scale eradication of disease vectors, cause a to fall from 1 to 0.49. This effectively makes income less important in disease transmission. Based on our previous analysis we know this will trigger a long-run growth take-off. What Figure 7 suggests is that this take-off is preceded by slow growth (not unlike what the economy experienced at PT) lasting several generations. In this sense, Acemoglu and Johnson's (2007) finding that health improvements do not lead to faster growth may be, at least for their relatively short sample period, more general than the mechanism they emphasize. Here the slowdown occurs because a lower a initially creates stronger incentives for health investment that dominate other types of (growth promoting) investment. Returns to health investment, during this phase, mainly take the form of direct utility gains realized from longer and healthier lives.

Returning to our policy analysis, another result that comes out of Figure 7 is that capital subsidies are now always more effective in raising growth. 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, subsidizing only capital may not be optimal if we take into account life expectancy. A package that includes x^{sub} will decrease the number of infected and, therefore, contemporaneously increase life expectancy. In contrast, a package that only subsidizes capital accumulation does not impact the current generation. Hence, a social planner who values longevity across generations may choose an intermediate policy that includes both types of subsidies.

The Case of Multiple Balanced-Growth Paths

Regarding policy effectiveness, our main results do not change when there are multiple *BGPs*. To abandon the low-growth trap, capital subsidies alone cannot help. In the case where $\phi = 0.65$, a health subsidy of 0.11 for at least 3 generations is needed to escape the trap. Capital subsidies do not provide much help since health subsidies are already very effective.

This scenario is also useful to illustrate the impact of changes in the institutional environment that can be captured by A . Like the survival probability, A directly affects long-run growth. However, changes in A have 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, suppose an improvement in the institutions that protect property rights and enforce contracts cause A to rise to 24.19.¹⁶ An economy that was previously located in the poverty trap would now experience a modest increase in long-run growth from 0% to 0.2% and no change in its population's health status. This, of course, delinks institutional improvements that increase A from improvements in public health systems that lower q . We briefly discuss the plausibility of this in the following section.

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 growth trajectory.

¹⁶It 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.

4 Diseases in Africa

Sub-Saharan Africa (SSA) is characterized by low growth and high infectious-disease prevalence. Therefore this region's experience is consistent with the key theoretical implication in this paper. But why is it that SSA suffers from such a high disease burden? This section examines, through the lens of our theory, particularities of the region's disease problems.¹⁷

Disease Ecology

According to our theory, SSA is more likely to gravitate towards a low-growth, high-prevalence attractor because its disease ecology is characterized by higher fatality rates (lower ϕ), higher exposure to the disease (higher μ), higher probability of contracting diseases if exposed (higher a), and lower efficacy of preventive actions (higher q).

Infectious diseases in SSA have been more virulent and destructive than elsewhere. Consider malaria which is a chronic disease and not episodic like the plague, influenza or small pox that affected pre-industrial Europe in waves. In the first place, much of SSA'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 SSA 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. Sub-Saharan 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

¹⁷SSA's GDP per worker grew at a dismal 0.6% average annual growth rate during 1950 – 2000 (Penn World Table 6.2). At the same it experienced a disproportionate share of the global infectious disease burden – it is estimated that infectious diseases contribute about 64% to SSA's overall disease burden (DALYs) in contrast to only 30% worldwide (Global Burden of Disease; WHO, 2002). Further, the probability of death of sub-Saharan African men between the ages 15 – 60 was 40% – 60% compared to less than 15% in developed countries (Murray and Lopez, 1997; Gakidou *et al.*, 2004). About 53% of the adult mortality is attributed to infectious and parasitic diseases, HIV/AIDS, tuberculosis and malaria being the leading contributors (WHO, 2001b).

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, in turn, 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 was also a contributing factor (Diamond, 1999). Colonization introduced new diseases to non-immune 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 “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, the concurrence of multiple infectious diseases could have then easily decreased the efficacy of preventive actions and increased the probability of contagion.

Finally, Kiple (1993) points to another reason for the lower efficacy of preventive investment (higher q in our model). 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

Our theory predicts that a lower value of q can propel a country towards the high steady state. This opens the door for medical technology and public health intervention to play a role in disease eradication and escape from underdevelopment. For instance, interventions in the form of vaccination, nutritional supplements, information campaigns, environmental improvements can all reduce q , and are best channeled through the public health system. SSA’s health institutions have been

¹⁸The flow of diseases went also in the other direction. Through the slave trade, during 1500–1900, African diseases like malaria, yellow fever and hookworm appeared in the warmer parts of the Americas, significantly affecting those populations (Patterson, 1993).

slow to respond in practice¹⁹ and medical technology has been scarce. Consistent with our theory, these two factors contributed to the persistence of low growth and high disease burden.

One would expect q and A (productivity and general institutional capability) to be correlated. Countries that have better institutions (A) are more likely to also have more efficient public health systems and readily adopt frontier medicine and medical technologies (q). 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 SSA. Compounding such conditions, Africa's public health systems are ineffective due to corruption, inadequate provision and, quite often, lack of skilled manpower (World Bank, 1993).

Unfortunately for SSA, a higher A may not bring a lower q if improved health interventions associated with stronger institutions are not effective. Nigeria's Garki Project illustrates that this is a real possibility. Under this project, 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). The best-practice q 's were simply inadequate for the disease problem.

5 Conclusions

This paper makes the case that poor health due to infectious diseases has first-order effects on economic development. The theory explicitly incorporates disease behavior in a general equilibrium framework. A simplified version of the model first illustrates how mortality and morbidity create the possibility of a development trap through their effect on saving-investment incentives. A more realistic version of the model with less restrictive preferences and disease transmission dynamics reveals a much stronger disease externality: any economy, regardless of income, can be attracted to a low-growth trap for a sufficiently large prevalence rate.

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). This is consistent with the work of Rachel Glennerster and Michael Kremer on vaccine research (2000, forthcoming). For example, Kremer (2002), and more

¹⁹Not only did colonialism bring new diseases to SSA, public health practices of the colonial powers also 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).

recently, Glennester, Kremer and Williams (2006) and Bernt *et al.* (forthcoming) devise 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) are instrumental in raising aggregate productivity.

We hope that our work can offer theoretical foundations 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. Consider the two recent contributions, Weil (2007) and Acemoglu and Johnson (2007), which suggest the effect of health on development may be relatively minor. Weil focuses on the impact of *morbidity* in partial equilibrium. In relation to our model, this would be a novel attempt to more accurately estimate θ . Hence, a positive impact of health on income is expected but not the sole driving force as we show here.

Acemoglu and Johnson, on the other hand, focus on *mortality* in general equilibrium and, in particular, on the impact of life expectancy (LE) on income per capita and its growth rate. They find that changes in LE have no significant impact on long-run economic growth. However, their main sample excludes Africa (for data quality reasons) and, therefore, consists of nations with relatively low chances of realizing a development-trap. But nations that move towards the *BGP* in our model end up with the same long-run growth rate, independently of initial levels of LE, as Acemoglu and Johnson find. If we had used a neoclassical production function, the model’s implications would have been that LE does not predict long-run GDP per capita in the higher-income steady-state. We can even use our theory to understand Acemoglu and Johnson’s finding 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 our simulation exercises very low growth rates, sometimes negative, following the escape from the trap. This effect is further amplified by a morbidity-related effect on GDP and because healthier cohorts will contribute to capital accumulation only several decades later.

The model presented here offers several testable predictions that empiricists can exploit. First, in the long run, it implies while mortality has growth effects, morbidity can at best have a level effect. Second, both mortality and morbidity are important determinants of the saving rate and disease prevention. Third, morbidity can generate dilution effects on capital intensity and these effects are stronger for higher levels of development. Lastly and perhaps most importantly, the effect of health should show up in the data through important nonlinearities in the growth process.

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