



WP-AD 2010-28

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Edita / Published by: Instituto Valenciano de Investigaciones Económicas, S.A.

Depósito Legal / Legal Deposit no.: V-3478-2010

Impreso en España (septiembre 2010) / Printed in Spain (September 2010)

Diseases, infection dynamics and development^{*}

Shankha Chakraborty, Chris Papageorgiou
and Fidel Pérez Sebastián^{**}

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, a result that differs 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 infectious diseases are eradicated. Even so, diseases can significantly slow down convergence to this growth path. Taken together, our results suggest that the empirical relationship between health and income at the aggregate level may be more nuanced than realized.

Keywords: Infectious Disease, Rational Disease Behavior, Morbidity, Mortality, Productivity, Economic Development.

JEL Classification: O40, O47

^{*} For helpful comments and suggestions we thank Costas Azariadis, Michele Boldrin, Angus Deaton, David DeJong, Allan Drazen, Eric Fisher, Oded Galor, Nicola Gennaioli, Simon Johnson, Peter Lorentzen, Jenny Minier, Emily Oster, Marla Ripoll, Andrés Rodríguez-Clare, Richard Rogerson, Rodrigo Soares, Antonio Spilimbergo, David Weil and seminar participants at various universities this paper was presented including Alberta, Copenhagen, Cyprus, Melbourne, Navarra, Pittsburgh, USC, Sydney, Syracuse, Melbourne, Vienna, Zaragoza and the Institute for Advanced Studies, Vienna. We also thank participants at the 2004 conference on “75 Years of Development Research” at Cornell, the 2004 Midwest Macro Meetigs at Iowa State, the 2005 CMSG in Vancouver, the 2005 NEUDC at Brown, the 2006 Demographics and Economic Development conference at Stanford, the 2006 Minerva-DEGIT conference in Jerusalem, the 2008 AEA meetings in New Orleans and the 2008 SED meetings in Cambridge. Research assistance from Hui-Hui Lin is gratefully acknowledged.

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1 Introduction

Motivated by evidence that a disproportionate share of developing country disease burden is due to infectious diseases,¹ we incorporate rational disease behavior into a growth model.² Infection spreads from random exposure to disease vectors, susceptibility to which depends on preventive health investment and the disease ecology (climate, vectorial capacity, culture and social practices). Diseases cause premature death as well as lower productivity and quality-of-life among the infected. The mortality effect makes infected individuals less inclined to save, 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, 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.

Income does not cause health in the low growth equilibrium *irrespective* of the economy's level of development. The disease externality overwhelms prevention incentives in this case and foreign assistance in the form of income transfers has little impact on health or development. This result is in sharp contrast to development traps in the existing literature (see Azariadis and Stachurski, 2005 for a recent review).

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 suggests public health initiatives and medical improvements, rather than income gains, caused worldwide mortality declines over 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 coun-

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.

tries, 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 slowdown 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 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 looks at 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.⁴ As adults, individuals are endowed with one unit of labor which they supply inelastically to the market. The modification that we introduce to the standard model is the possibility of contracting an infectious disease early in life and prematurely dying from it.

2.1 Disease Transmission 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,

⁴We 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 incorporated in the cost of disease parameters.

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.

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.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*

⁵In 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.

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, 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.⁶ 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

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

⁶The 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.

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 .⁷ 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

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

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.⁸ 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, $c_{1t}^U = c_{2t+1}^U = z_t^U = \tau_{t+1} = 0$, $c_{1t}^I = (1 - \theta)w_t - x_t$, $z_t^I = w_t - x_t$, and $c_{2t+1}^I = R_{t+1}(w_t - x_t)$. 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

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

⁷Implicitly x is financed by zero-interest loans taken early in youth from the rest of the world and repaid after the labor market clears.

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

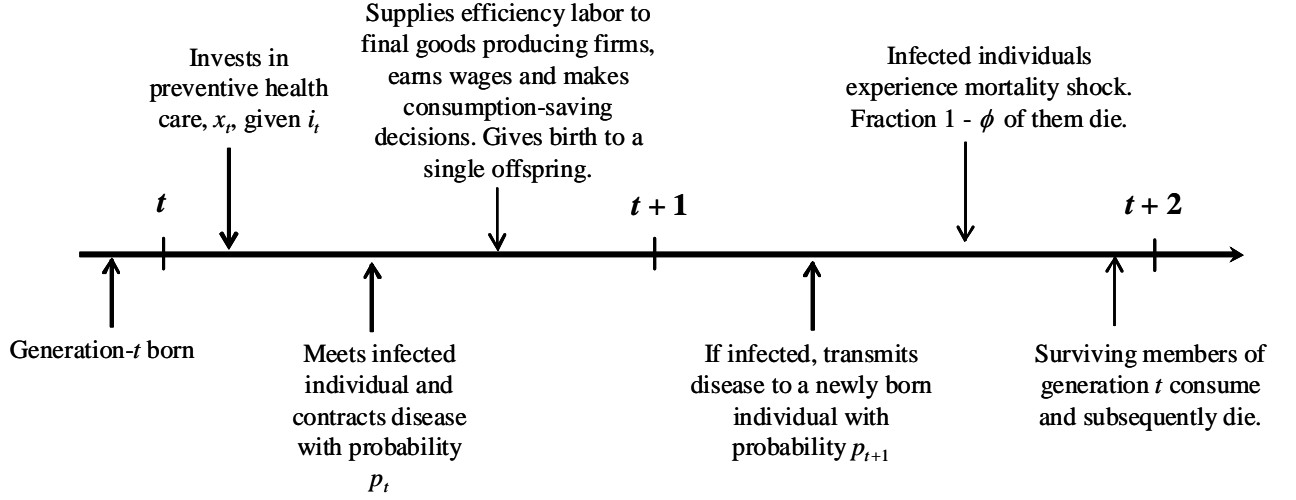


Figure 1: Timeline of Events

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.

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

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

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

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

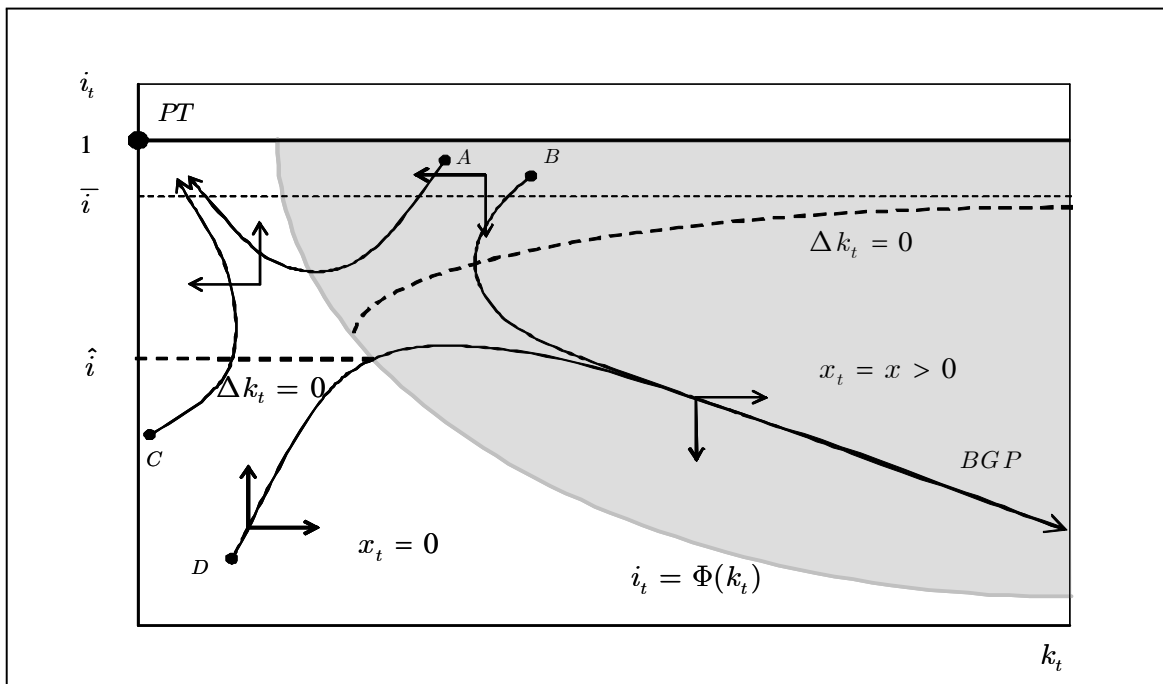
The denominator of Ψ is positive at $i_t = 0$ and declines monotonically with i_t . Hence Ψ increases with i_t . But the denominator of Ψ can become zero if

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

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

Figure 2: Phase Diagram for the Simple Model



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 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,

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

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 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.⁹

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

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

⁹The 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.

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 (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

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

while disease dynamics evolve as before

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

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

Table 1: Benchmark Parameter Values

β	$0.99^{(31.5 \times 4)}$	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

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 $\phi = 0.47$.¹⁰

¹⁰This implies that life expectancy at birth is about 61 years in the high-prevalence steady-state. This is higher

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 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.¹¹

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.

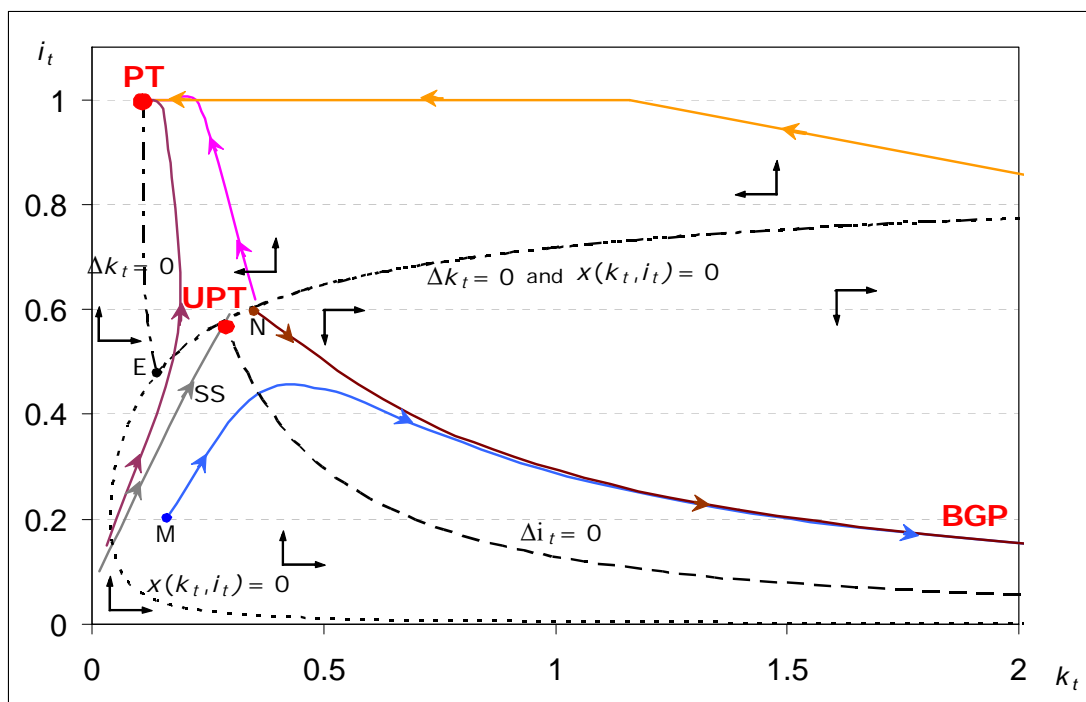
This positive effect dominates at a relatively large capital intensity. To see this, set $x = 0$ since $\Delta k_t = 0$ coincides with the zero investment locus. The $\Delta k_t = 0$ locus gives steady-state values of k for exogenous values of i . Rearranging terms, this locus is

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

than life expectancies in sub-Saharan Africa, but we are ignoring non-infectious disease mortality.

¹¹Our 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.

Figure 3: Phase Diagram for the Complete Model under Benchmark Parameter Values



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 .¹²

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

$$i_t = p(k_t, i_t), \quad (54)$$

along which the infection rate remains constant. It is defined wherever $x_t > 0$ and, in this area, the infection rate is decreasing above the curve while it is increasing below it. To the left of the

¹²The possibility that more adverse disease conditions can actually improve economic conditions is not novel to our model. It echoes historical accounts of how the Black Death pandemic in 14th century Europe may have left its survivors better-off by easing population pressure from agriculture. Young's (2005) analysis of the economic consequences of Africa's AIDS epidemic follows a similar argument as does the combined effect of several other infectious diseases on life expectancy and growth in recent work by Acemoglu and Johnson (2007).

$x_t = 0$ schedule preventive investment is zero which implies the infection rate is always rising since $\mu a > 1$.

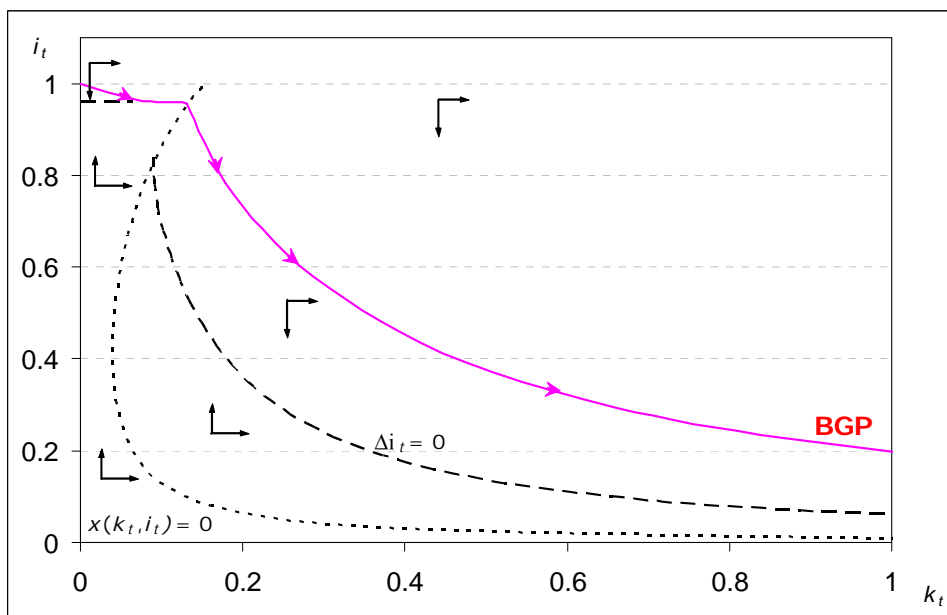
Figure 3 shows multiple steady states. There are two poverty traps with zero growth, one stable (PT) and the other unstable (UPT). There also exists a stable balanced growth path (BGP) along which the economy grows at a strictly positive rate. Vector fields indicate that the PT steady-state is a sink 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 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).

Figure 4: Phase Diagram for $a = 0.49$



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.

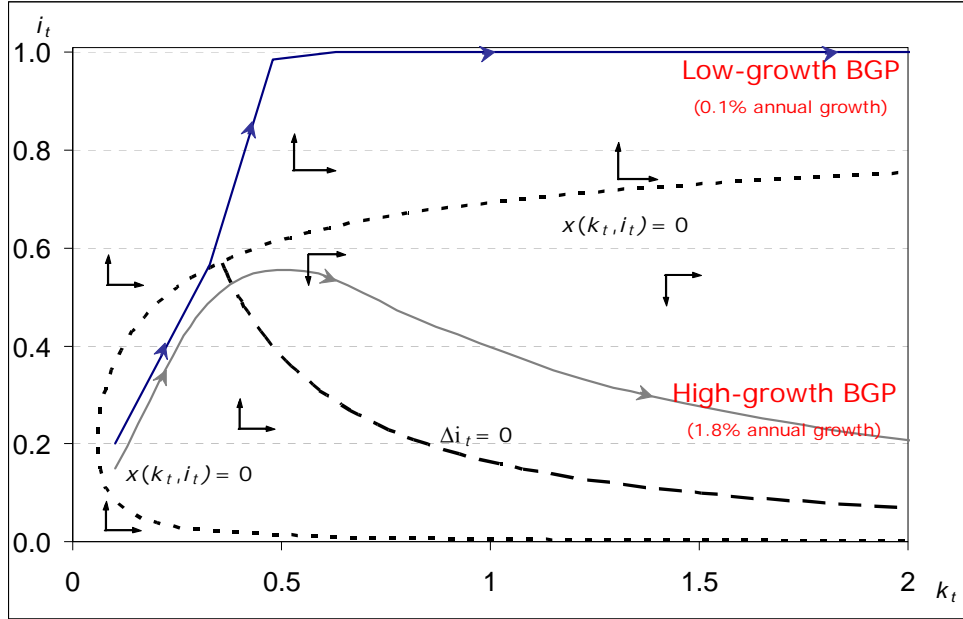
3.7 Two Alternative Cases

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,

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



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.

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 a 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.¹³

4 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 have to be health specific, for instance 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 recently, Glennerster *et al.* (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 two recent contributions, Weil (2007) and Acemoglu and Johnson (2007), that suggest the effect of health on development is relatively minor. Weil focuses on the impact of *morbidity* in partial equilibrium. In the context of our model,

¹³The 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 $x = 0$.

this is a novel attempt to estimate θ more precisely than we do. A positive impact of health on income is expected in this case but our model shows morbidity is not the sole driving force.

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, comprises of nations with relatively better prospects of escaping a development-trap. Nations that converge to the higher growth path in our model all end up with the same long-run growth rate, independently of initial LE or income, as Acemoglu and Johnson find. If we had used a neoclassical technology, the model's implications would have been similar in terms of long-run income levels. Our theory may even shed light on Acemoglu and Johnson's finding that LE improvements can lead to a short-run decline in GDP. While we do not account for population growth, our numerical simulations show that escape from the disease trap is accompanied by very low growth rates, sometimes negative, of income per worker. This effect is further amplified by a morbidity-related effect on GDP since healthier cohorts begin contributing to capital accumulation only several decades later.

The model presented here offers several testable predictions that empiricists can exploit. First, it implies that while mortality has growth effects, morbidity can at best have a level effect in the long run. 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 at higher levels of development. Perhaps most importantly, the theory suggests the impact of health will show up in the data through important nonlinearities in the growth process.

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