# DISEASES AND DEVELOPMENT*

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**Abstract**

We propose an epidemiological overlapping generations model where the transmission and incidence of infectious diseases depend on economic incentives and rational behavior. The economic cost of diseases comes from their effect on mortality (infected individuals can die prematurely) and morbidity (lower productivity and quality of life). Our model offers two main insights: First, a greater prevalence of diseases implies a lower savings-investment propensity because of mortality and morbidity. The extent to which preventive health investment can counter this depends on the prevalence rate and, specifically, on the strength of the disease externality. Second, persistence of underdevelopment is possible as a consequence of the intrinsic nature of infectious diseases. Extensive calibration exercises reveal that income *per se* may not cause health when prevalence is high. Successful interventions should therefore be health specific and when possible channeled via the public health delivery system. The role of disease ecology and institutions is examined.

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# 1 Introduction

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

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

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

A key result from our theory is that two types of long-run growth are possible: one where diseases are widespread and growth is low (possibly zero), and the other where diseases ultimately disappear and the economy enjoys sustained improvement in living standards. Initial income, prevalence, and disease ecology determine which of these development paths attracts a particular country.

Extensive quantitative exercises reveal the following. First, both growth regimes are plausible

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

for reasonable parameter values. Second, income does not cause health when infectious diseases are widespread, irrespective of the level of development. The disease externality becomes so high in this situation that it wipes out incentives to invest in prevention. An epidemic shock can therefore trip even a wealthy economy to the slower growth path. Overall foreign aid in the form of income transfers has little effect on health or development.[2] It also means, general institutional changes that improve aggregate TFP can raise the growth rate but will have limited impact on disease eradication unless public health institutions improve too. This is consistent with evidence that the conquest of infectious diseases in many countries has been possible due to improvements in medicine and public health rather than income gains (Cutler *et al.*, 2006; Soares, forthcoming).

Third, income can have an effect on health, in contrast, when countries are converging to the high-growth low-disease balanced growth path. In particular, mortality and morbidity steadily decline as economic growth allows for better investment in preventive health. Fourth, numerical experiments that examine the efficacy of foreign aid in the form of health assistance show that costs can be large. Quick interventions and simultaneously targeting capital accumulation and preventive health can reduce this cost. Finally, the disincentive effect of diseases on savings-investment behavior can be strong enough to slow down growth rate convergence by several generations, even when all countries are converging to the same balanced growth path.

There has been a recent surge in research on health and development. Despite compelling microeconomic evidence that health is important for economic outcomes (see, e.g. Strauss and Thomas, 1997 and Deaton, 2003), the macroeconomic evidence has been somewhat mixed. Empirical works such as Bloom and Canning (2005) and Gallup and Sachs (2001) attribute Africa's persistent poverty to endemic infectious diseases particularly malaria. Gallup and Sachs, for example, estimate that malaria reduces per capita income in a malarious country by more than half compared to a non-malarious country. Lorentzen *et al.* (2006) find that adult mortality explains almost all of Africa's growth tragedy in the past forty years.

Other works, however, offer a more qualified view. Acemoglu and Johnson (2006) use cross-country panel data and a novel instrument to control for the obvious endogeneity between health and income. They find very small if any positive effect of health on per capita GDP. These authors argue that the increase in population resulting from better health outweighs the productivity effects and therefore GDP per capita may have actually slightly decreased in their panel of countries.

[2] This is consistent with recent evidence. Rajan and Subramanian (2005) examine the effects of aid on growth after correcting for the bias that aid typically goes to poorer countries, or to countries after poor performance. They find little robust evidence of a positive relationship between aid inflows into a country and its economic growth. Mishra and Newhouse (2007) empirically estimate the effects of aid on infant mortality using a dataset covering 118 countries from 1970 to 2004. These authors find that although overall foreign aid does not have a statistically significant effect on infant mortality, *health aid* does.

Weil (forthcoming) uses microeconomic estimates of the effect of health on individual outcomes to construct macroeconomic estimates of the effect of (average) health on GDP per capita. His main finding is that eliminating health differences among countries will reduce the variance of log GDP per worker by about 10%. This estimate is economically significant but substantially smaller than estimates from cross-country growth regressions that Bloom and Canning (2005) and Gallup and Sachs (2001) report.

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

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

We also depart from the existing theoretical literature by making adult mortality the centerpiece of our work. We do so for two reasons. First, developing countries have enjoyed enormous life expectancy improvements over the past fifty years mainly due to sharp declines in infant and child mortality made possible by low-cost interventions and technology transfers. Adult mortality has declined relatively less and remains high in developing countries (World Bank, 1993). More importantly, this excessive adult mortality is mainly due to infectious diseases which affect a disproportionate number of adults in poorer countries compared to their counterparts elsewhere. By now we have a good understanding how infant mortality impacts development; there is less clarity on the effects of adult mortality and less policy urgency as well. Secondly, some of the empirical evidence suggests that adult mortality may well be more instrumental in affecting economic development

than infant and child mortality (Lorentzen *et al.*, 2006; Chakraborty *et al.* 2006; Stoytcheva and Papageorgiou, 2006).

The paper is organized as follows. In section 2 we specify the model and analyze general equilibrium dynamics. In section 3 we use a set of benchmark values to calibrate the model and explore its dynamics. This section also presents numerical results on the quantitative effect of diseases on economic development. Section 4 examines two alternative cases to the benchmark and presents results from additional robustness checks. Africa's experience with diseases and its dismal growth experience are discussed within the model's framework in section 5. Section 6 concludes.

# 2 The Model

Our framework is a discrete time, infinite horizon economy populated by overlapping generations of families. Each individual potentially lives for two periods, adulthood and old-age.[3] As adults, individuals are endowed with one unit of efficiency labor which they supply inelastically to the market. The modification we introduce to the standard model is the possibility of contacting an infectious disease early in life and premature death from it.

## 2.1 Infectious Diseases

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

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

Diseases spread from infected older individuals to susceptible younger ones through a process of random matching. 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

[3] We do not explicitly model childhood. Children's consumption is subsumed into their adult parent's. Since we focus on adult mortality, the effect of infant mortality on fertility decisions is ignored. Childhood morbidity from infectious diseases, however, can have lifelong repercussions on productivity and human capital. This morbidity effect is implicity incorporated below through cost of disease parameters.

be infected and not all encounters with infected people result in transmission. In particular, given his preventive health investment $x_t$, the probability that a young individual gets infected from such a matching is $\pi(x_t)$, where $\pi' < 0$ and $-\pi'(0) > \infty$. We also restrict $\pi(0) > 1/\mu$ so that disease prevalence rises over time in the absence of preventive investment.

One example that satisfies these properties is

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

We use this function in calibration exercises later on. 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. Note that $\pi(0) = a$. We interpret $a$ as an evolutionary parameter which gives the probability of getting infected if agents do not invest in prevention. Factors that influence its value are the genetic evolution of humans and virus mutations. An example is the sickle-cell trait, a genetic mutation that provides partial defense against malaria and is carried by about 25% of the human population in areas severely affected by the disease (see, Galor and Moav, 2005, for references and additional examples).

Let $p_t$ denote the probability of being infected for a typical member of generation $t$. 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 ($\pi_t$), that is, $i_t\pi(x_t)$. Hence, the probability of not being infected after $\mu$ matches is simply $[1 - i_t\pi(x_t)]^\mu$. Thus,

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

Notice that equation (2) includes an important negative externality that characterizes infectious diseases. When an individual chooses preventive health investment *ex ante* – before he meets an infected older person – he does not take into account how his decision impacts the susceptibility of future generations. Furthermore, this externality is amplified by the random matching process: equation (2) implies that the probability of disease contagion rises exponentially with the number of encounters $\mu$.

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. There

is evidence for this. 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 (Gallup and Sachs, 2001).

Secondly, assuming diseases are transmitted directly from an infected to a susceptible person is a simplification. The parameter $\mu$ captures the disease ecology more generally. For a disease like AIDS, it can be directly related to the number of sexual partners or needle-sharers.[4] 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, $\mu$ has the more appropriate interpretation of the mosquito's vectoral 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 $\mu$. 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 Preferences

Preferences and individual behavior are disease contingent. We consider first decisions of an uninfected individual whose health investment has successfully protected him from the disease. The period utility function $u(c)$ is increasing, twice continuously differentiable with $u' > 0$, $u'' < 0$. In addition, it is homothetic, and current and future consumptions are normal goods. The individual maximizes lifetime utility

$$u\left(c_{1t}^{U}\right) + \beta u\left(c_{2t+1}^{U}\right),\ \beta \in (0,1), \tag{3}$$

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

subject to the budget constraints

$$c_{1t}^U = w_t - x_t - z_t^U \tag{4}$$

$$c_{2t+1}^U = R_{t+1} z_t^U, \tag{5}$$

where $w$ is the wage per efficiency unit of labor, $z$ denotes savings and $x$ is given by decisions made early in period $t$.[5] 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

$$\delta \left[ u\left(c_{1t}^I\right) + \beta\phi u\left(c_{2t+1}^I\right) \right], \tag{6}$$

subject to

$$c_{1t}^I = (1-\theta) w_t - x_t - z_t^I \tag{7}$$

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

where $\tau_{t+1}$ 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.[6] Clearly transfers per surviving infected individual will be

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

in equilibrium.

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

$$\begin{aligned} u'(c_{1t}^U) &= \beta R_{t+1} u'(c_{2t+1}^U) \\ u'(c_{1t}^I) &= \beta\phi R_{t+1} u'(c_{2t+1}^I), \end{aligned} \tag{10}$$

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

We assume a CES utility function

$$u(c) = \frac{c^{1-\sigma} - 1}{1-\sigma}, \ \sigma \in (0,1). \tag{11}$$

for analytical convenience and because it is appropriate for the calibration exercise later. There is though a potential problem with this choice. While we have assumed zero utility from death, this

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

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

function takes on negative values when consumption is less than one. Since we think of death as the outcome that provides the lowest utility, consumption has to strictly exceed one. The choice of our production technology, later on, will ensure that.

Optimal savings for uninfected and infected individuals are

$$z_t^U = \left[\frac{\beta^{1/\sigma} R_{t+1}^{1/\sigma-1}}{1+\beta^{1/\sigma} R_{t+1}^{1/\sigma-1}}\right](w_t - x_t) \tag{12}$$

$$z_t^I = \left[\frac{(\beta\phi)^{1/\sigma} R_{t+1}^{1/\sigma-1}}{1+(\beta\phi)^{1/\sigma} R_{t+1}^{1/\sigma-1}}\right][(1-\theta)w_t - x_t] - \left[\frac{1}{1+(\beta\phi)^{1/\sigma} R_{t+1}^{1/\sigma-1}}\right]\frac{\tau_{t+1}}{R_{t+1}}. \tag{13}$$

Substituting these into lifetime expected utility gives the two indirect utility functions

$$V^U(x_t) = \frac{1}{1-\sigma}\left[\left(w_t - x_t - z_t^U\right)^{1-\sigma} + \beta\left(R_{t+1} z_t^U\right)^{1-\sigma}\right] - \frac{1}{1-\sigma} \tag{14}$$

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

contingent on prices and preventive health investment.

At the beginning of $t$, adults choose the optimal level of $x_t$ to maximize expected lifetime utility. Recall that $i_t$ denotes the fraction of old agents who are infected. Given the random matching process mentioned above, a young individual's probability of catching the disease is $p_t$ given by (2). Hence, individuals choose $x_t$ to maximize

$$p_t V^I(x_t) + [1-p_t]\, V^U(x_t), \tag{16}$$

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

$$-\mu\left[1 - i_t\pi_t\right]^{\mu-1}\pi'(x_t) i_t\left(V_t^U - V_t^I\right) \geq p_t\left(-\frac{\partial V_t^I}{\partial x_t}\right) + [1-p_t]\left(-\frac{\partial V_t^U}{\partial x_t}\right), \tag{17}$$

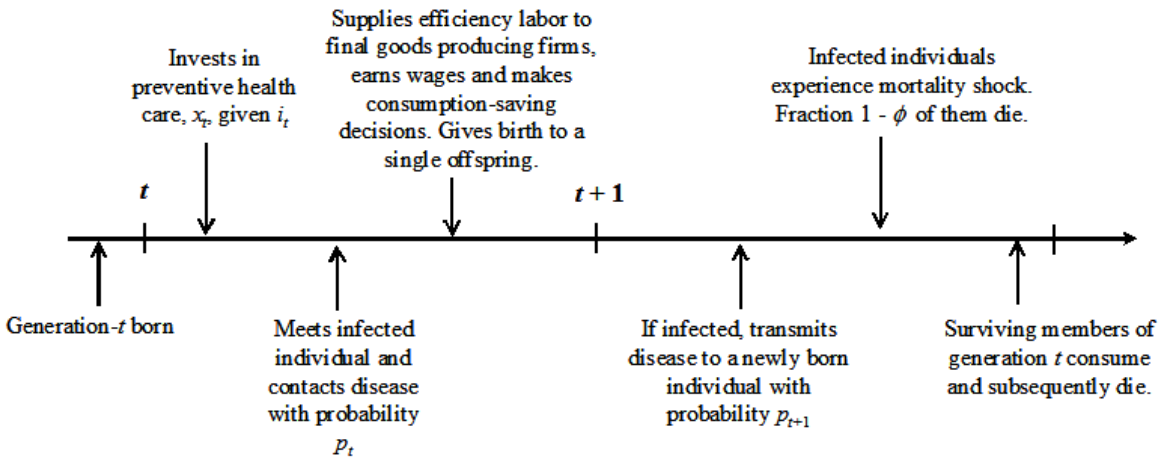
for $x_t \geq 0$. This states that for individuals to be willing to invest in disease prevention, the marginal benefit from living longer and experiencing a healthier life cannot be outweighed by the marginal cost of foregoing current income.[7]

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

We summarize the timeline of events in Figure 1.

[7] The first order conditions (10) and (17) are necessary but not sufficient since preferences can become non-convex with endogenous $p$. When we calibrate the model we verify that second order conditions are satisfied for the parameter values and functional forms that we choose.

Figure 1: Timeline of Events




## 2.3 Technology

A continuum of firms operate in perfectly competitive markets to produce the final good using capital and efficiency units of labor. To accommodate the possibility of endogenous growth we posit a firm-specific constant-returns technology exhibiting learning-by-doing externalities

$$F(K^i, L^i) = A(K^i)^\alpha(\bar{k}L^i)^{1-\alpha} + bL^i, \tag{18}$$

where $A$, $b > 0$, and $\bar{k}$ denotes the average capital per effective unit of labor across firms.[8] We can think of $b$ as capturing natural endowments such as trees and animals that do not need to be produced. In addition, the optimal amount of consumption is always above one if $b$ is sufficiently large. As mentioned in the previous subsection, this is important given our choice of a CES utility function and the assumption of zero utility from death.

Standard factor pricing relationships under such externalities imply that

$$w_t = (1-\alpha)Ak_t + b \equiv w(k_t) \tag{19}$$

$$R_t = \alpha A \equiv R. \tag{20}$$

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

## 3 General Equilibrium

We begin by substituting equilibrium prices and transfers into the saving functions to obtain

$$z_t^U = s^U \left[w(k_t) - x(w_t, i_t)\right] \equiv z^U(k_t, i_t), \tag{21}$$

and

$$z_t^I = s^I \left[(1-\theta)w(k_t) - x(w_t, i_t)\right] \equiv z^I(k_t, i_t), \tag{22}$$

where,

$$s^U \equiv \left[\frac{\beta^{1/\sigma} R^{1/\sigma - 1}}{1 + \beta^{1/\sigma} R^{1/\sigma - 1}}\right], \; s^I \equiv \left[\frac{\phi(\beta\phi)^{1/\sigma} R^{1/\sigma - 1}}{1 + \phi(\beta\phi)^{1/\sigma} R^{1/\sigma - 1}}\right]. \tag{23}$$

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

Substituting the savings functions into indirect utility obtains

$$\begin{aligned} V_t^{U*} &= \frac{1}{1-\sigma}\left[\left(1 - s^U\right)^{1-\sigma} + \beta R^{1-\sigma}\left(s^U\right)^{1-\sigma}\right]\left(w(k_t) - x_t\right)^{1-\sigma} - \frac{1}{1-\sigma} \\ &\equiv \frac{\zeta^U \left(w(k_t) - x_t\right)^{1-\sigma}}{1-\sigma} - \frac{1}{1-\sigma}, \end{aligned} \tag{24}$$

and

$$\begin{aligned} V_t^{I*} &= \frac{\delta\phi^\sigma}{1-\sigma}\left[\left(\frac{1 - s^I}{\phi + (1-\phi)s^I}\right)^{1-\sigma} + \beta R^{1-\sigma}\left(s^I\right)^{1-\sigma}\right]\left((1-\theta)w(k_t) - x_t\right)^{1-\sigma} - \frac{\delta}{1-\sigma} \\ &\equiv \frac{\zeta^I \left((1-\theta)w(k_t) - x_t\right)^{1-\sigma}}{1-\sigma} - \frac{\delta}{1-\sigma}. \end{aligned} \tag{25}$$

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 (9) when making health investment decisions. Accordingly (17) becomes

$$p_t \zeta^I [(1-\theta)w(k_t) - x_t]^{-\sigma} + (1-p_t)\zeta^U [w(k_t) - x_t]^{-\sigma} \leq -\mu \left[1 - i_t \pi_t\right]^{\mu - 1} \pi'(x_t) i_t \left[V_t^{U*} - V_t^{I*}\right]. \tag{26}$$

Two possibilities arise depending on whether or not preventive investment yields positive returns. If (26) holds as a strict inequality at $x_t = 0$, optimal investment will be $x_t = 0$. The left-hand of side (26) is the marginal utility cost of that investment, since health investment entails a lower current and, possibly, future consumption. The right-hand side constitutes the marginal benefit, in the form of higher net utility from lowering one's chance of catching the infectious disease. Optimal

health investment is zero as long as the utility cost dominates, that is, returns to health investment are negative at $x_t = 0$. Our assumption that an individual cannot infinitely lower his disease risk through finite health investments, $\pi'(0) > -\infty$, ensures that such a possibility can arise. 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 (26) above, the condition for zero preventive investment is

$$\chi(k_t, i_t) = \zeta^U[1-p(0)]+\zeta^I(1-\theta)^{-\sigma}p(0)]w_t^{-\sigma}+\mu\left[1-i_t\pi(0)\right]^{\mu-1}\pi'(0)i_t\left\{V_t^U(0)-V_t^I(0)\right\} \geq 0. \quad (27)$$

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 (26) holds as an equality. Optimal health investment in the interior solution

$$x_t = x(k_t, i_t), \quad (28)$$

satisfies $\partial x/\partial k > 0$ (income effect, as a higher $k$ increases lifetime labor income) and $\partial x/\partial i > 0$ (preventive investment is higher when the disease externality is greater).

### 3.1 Dynamics

With a continuum of young agents of measure one we can appeal to the law of large numbers and write down aggregate savings at $t$ as a weighted average of the savings of infected and uninfected individuals

$$S_t = p_t z_t^I + (1-p_t)z_t^U, \quad (29)$$

and the asset market clearing condition as

$$K_{t+1} = S_t. \quad (30)$$

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

$$L_{t+1} = (1-\theta)p_{t+1} + (1-p_{t+1}) = 1-\theta p_{t+1}. \quad (31)$$

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

Using optimal health investment $x(k_t, i_t)$, we can express the equilibrium probability of getting infected as $p_t = p\left(x(k_t, i_t), i_t\right) \equiv p(k_t, i_t)$. For the functions we choose and numerical values

we assign to parameters, we can establish that $\partial p_t/\partial k_t > 0$ and $\partial p_t/\partial i_t > 0$. The first result ($\partial p_t/\partial k_t > 0$) is simply an income effect: preventive investment rises with affluence. Two opposing effects are embedded in the second result ($\partial p_t/\partial i_t > 0$). Disease prevalence directly increases the probability via equation (2), but also tends to lower it by encouraging greater preventive investment ($\partial x/\partial i > 0$). This indirect effect is not sufficiently strong to overturn the externality effect.

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

$$k_{t+1} = \frac{p(k_t, i_t)z^I(k_t, i_t) + [1 - p(k_t, i_t)]z^U(k_t, i_t)}{1 - \theta p\left(p(k_t, i_t)\right)}. \tag{32}$$

Equilibrium disease dynamics, in turn, evolve according to

$$i_{t+1} = p(k_t, i_t), \tag{33}$$

by the law of large numbers.

Equations (32) and (33) describe the general equilibrium of this economy given initial conditions. Analytical results, particularly for the dynamics, are hard to establish. We examine this numerically when we calibrate the model below. For now, we foresee that there are two types of stationary equilibria. The first one is a development trap where output and capital per capita grow at a relatively low rate and there is widespread disease prevalence. The second one is a balanced growth path along which per capita variables grow at a relatively high rate and infectious diseases disappear.

As we show below, both stationary equilibria are stable. 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. It also depends on the disease ecology. More adverse disease conditions expand the state space over which convergence to the slow growth high prevalence growth path is possible.

Intuitively, the possibility of multiple growth paths depends on the economy's average propensity to invest. This propensity is a low $s^I$ when everyone is infected and takes on the higher value $s^U$ for an uninfected population. For our calibrated values, a saving rate of $s^U$ implies balanced growth equal to

$$1 + \gamma^H \equiv (1 - \alpha)As^U = \frac{\beta}{1 + \beta}(1 - \alpha)A > 1, \tag{34}$$

whereas $s^I$ implies that long-run growth is

$$1 + \gamma^L \equiv (1 - \alpha)As^I = \frac{\beta\phi^2}{1 + \beta\phi^2}(1 - \alpha)A. \tag{35}$$

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 differ only because $\phi < 1$. In other words, it is adult mortality alone that

Table 1: Benchmark Parameter Values

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| $\beta$ | $0.99^{(31.5\times4)}$ | $\alpha$ | 0.67 | $\theta$ | 0.15 | $\mu$ | 5 |
| $\sigma$ | 1 | $g_y$ | 0.018 | $\phi$ | 0.47 | $q$ | 0.14 |
| $b$ | 1 | | | $\delta$ | 0.9 | $a$ | 1 |

causes long-run growth to be slower when diseases are widespread.[9] Interestingly, even if $\phi = 1$, two economies with differing initial income and disease prevalence can grow at similar rates but experience diverging incidence of infectious diseases. That implies large persistent welfare gaps as long as quality of life depends on morbidity from infectious diseases (that is, $\delta < 1$).

# 4 Dynamics and Numerical Experiments

The central mechanism of our theoretical model is the interdependence between infectious diseases and economic outcomes. But the exact nature of the growth path will be shaped by various economic and disease-specific conditions. Since the model does not offer analytically tractable results we rely on computational techniques. We first calibrate the model and establish its dynamic properties under a set of benchmark values. We then conduct simulation exercises to assess the cost of infectious diseases, the impact of policy interventions, and how parameters governing the disease ecology and institutions matter.

## 4.1 Benchmark Calibration

Table 1 presents our 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 $0.99^{31.5\times4}$ to the discount factor ($\beta$), that is, 0.99 per quarter which is standard in the real-business-cycle literature. We set the elasticity of substitution for consumption ($\sigma$) to 1 (log preferences). The production function has three parameters: the technology parameter $A$, the capital elasticity $\alpha$, and the labor productivity coefficient $b$. We normalize $b$ to 1 to ensure that consumption levels are bounded above one and, as a consequence, utility when alive remains positive. We calibrate $\alpha$ to 0.67; we are then looking at a broad concept of capital that includes physical, human and organizational capital. The value for $A$, in turn, is chosen so as to reproduce

[9] The parameters $\theta$ and $\delta$ affect convergence dynamics since they either affect savings directly (for $\theta$) or indirectly via $x$ (for $\delta$).

an annual long-run growth rate in the low-prevalence steady state of 1.8%. This number is the average growth rate of GDP per capita between 1990 and 2003 for OECD nations in UNDP (2005). Therefore, $A$ is chosen such that the balanced growth rate $s^U(1-\alpha)A$ equals $1.018^{31.5}$, which in turn implies that $A = 24.19$.

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

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

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 (1952) 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 $\theta$.

Also difficult to calibrate is the mortality parameter $\phi$. According to WHO (2004), more than

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

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 $\phi$ 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]

### 4.2 The Phase Portrait: Benchmark Case

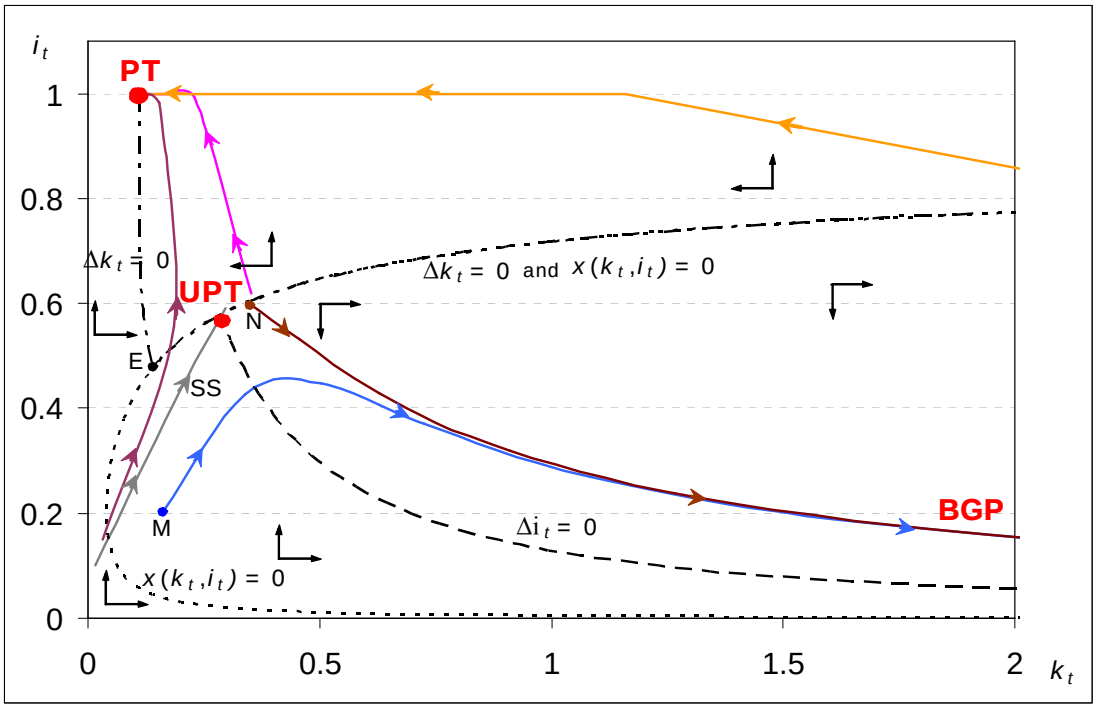
Recall that the general equilibrium is described by the pair of difference equations (32) and (33), and the initial conditions $(k_0, i_0)$. Figure 2 displays the phase diagram for the parameter values in Table 1. It plots the prevalence rate $i_t$ against capital per effective unit of labor $k_t$.

Three different loci characterize dynamics. First, the $x(k_t, i_t) = 0$ line represents combinations of $(k_t, i_t)$ for which the optimal decision is not to invest in prevention. The same decision is also optimal in the area to the left of $x(k_t, i_t) = 0$ while to its right optimal investment is positive. The $x(k_t, i_t) = 0$ locus has its particular shape because of the way prevalence and income affect incentives. For low levels of disease prevalence ($i_t \to 0$), the risk of catching an infection is so low that prevention is not necessary. At high levels of disease prevalence ($i_t \to 1$), in contrast, the productivity of prevention becomes vanishingly small as the disease externality from sequential matching outweighs the benefits from prevention.[12] Key to understanding this last point is recognizing that the return to preventive investment declines rapidly with $\mu$. For instance, the probability of being infected after $\mu = 5$ matches becomes 1.0 for any $i_t\pi(x_t) \geq 0.5$, while it becomes 1.0 for any $i_t\pi(x_t) \geq 0.3$

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

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

Figure 2: Phase Diagram for Benchmark Values




after $\mu = 10$.

The second locus, $\triangle k_t = 0$, plots $(k_t, i_t)$ combinations along which $k$ remains constant. It is given by equation (32) 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 $b > 0$. [13]

Note the U-shape of the $\triangle k_t = 0$ locus: the same infection rate can be associated with both high and low levels of the capital stock. This results from a tension between two effects of diseases on capital accumulation. Diseases have a negative effect on capital accumulation via their effect on mortality (which lowers incentive to save) and productivity (which lowers ability to save). This is what the numerator on the right-hand side of equation (32) represents. But diseases can also

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

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 (32). The relative scarcity of efficiency labor causes its return to go up, as indicated in equation (19). This higher return may be high enough to actually increase savings and investment per effective unit of labor.

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

$$k = \frac{p(i)s^I(1-\theta)w(k) + [1-p(i)]\, s^U w(k)}{1-\theta i}, \tag{36}$$

or,

$$\left[p(i)s^I(1-\theta) + \{1-p(i)\}s^U\right]\left[\frac{1}{1-\theta i}\right]\frac{w(k)}{k} = 1, \tag{37}$$

where $p(i) \equiv 1 - [1 - i\pi(0)]^\mu$. The first term on the left-hand side of (37) is a capital accumulation effect via discounting: as $i$ decreases, $p(i)$ decreases and weight shifts to the higher savings propensity of the healthy. The second term is a capital dilution effect: as $i$ decreases, overall worker productivity increases which lowers the capital intensity (for a given $K$). Since the $\Delta k_t = 0$ line is U-shaped, for any $i$ there may exist two steady-state values, $k_1$ and $k_2 (> k_1)$ over a certain range of disease prevalence. At $k_1$, $\partial k_1/\partial i < 0$ while $\partial k_2/\partial i > 0$ at $k_2$. Let us consider $k_1$ for a moment. When $i$ decreases, the first term increases while the second term decreases. Since $k_1$ goes up, $w/k = (1-\alpha)A + b/k$ goes down. In other words, to maintain the steady-state equality above, the decrease in the dilution effect is not enough to compensate for the increase in capital accumulation. So, at lower value of $k$, the capital accumulation effect dominates. Similar reasoning shows the capital dilution effect must dominate the accumulation effect at relatively higher values of $k$.

The possibility that disease prevalence can actually improve economic conditions is not novel to our model. In particular, it echoes historical accounts of how the Black Death pandemic in 14th century Europe may have left its survivors better-off by easing population pressure from agriculture. Young's (2005) analysis of the economic consequences of Africa's AIDS epidemic follows a similar argument as does the effect of higher life expectancy in Acemoglu and Johnson (2006). The interesting difference is the effect arises here not from mortality but morbidity. Even then, whether or not this effect dominates in equilibrium depends on the dynamics of infectious diseases.

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

by the equation

$$i_t = p(k_t, i_t), \tag{38}$$

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

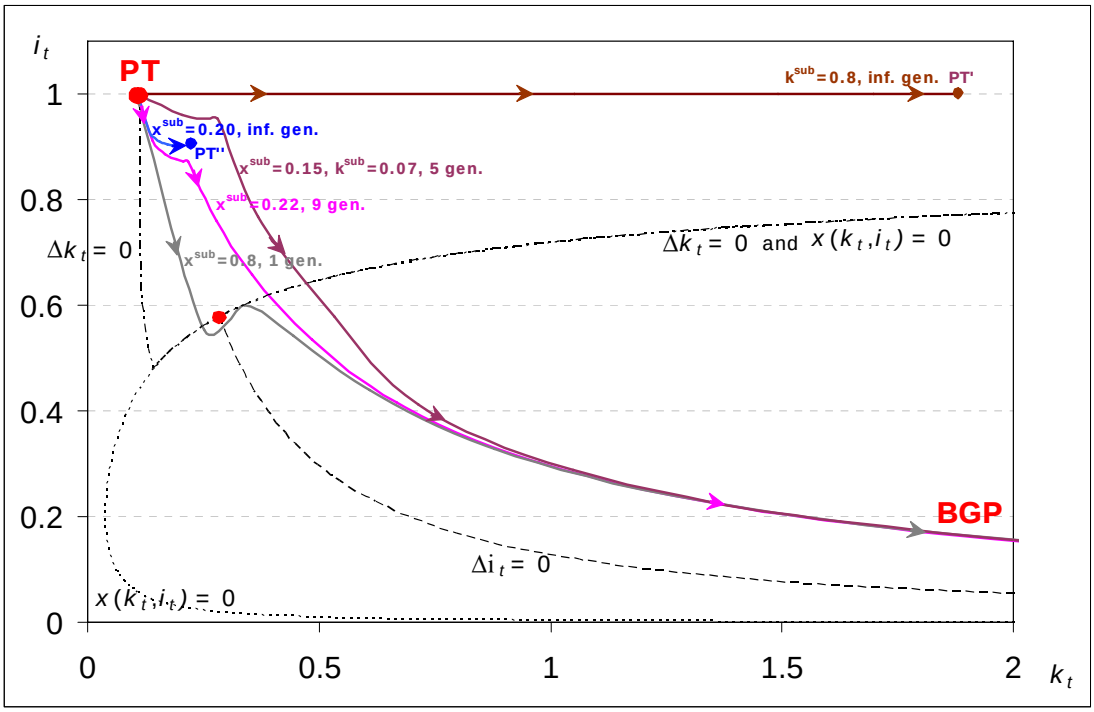
Figure 2 shows multiple steady states. There are two poverty traps with zero growth, one stable ($PT$) and the other unstable ($UPT$). There also exists a stable balanced growth path ($BGP$) along which the economy grows at a strictly positive rate. Vector fields indicate that the $PT$ steady-state is a sink while $UPT$ is a saddle-point. Since both the initial prevalence rate $i_0$ and the initial capital per efficiency labor $k_0$ are pre-determined, $PT$ is asymptotically stable but $UPT$ is not. In particular, sequences of $(k_t, i_t)$ which do not start exactly on the saddle-arm $SS$ converge either to $PT$ or diverge to a sustained growth path along which infectious diseases disappear asymptotically. The saddle path therefore acts as a threshold until it meets the $x = 0$ locus, at which point, the continuation of that locus becomes the effective threshold. Notice that if $i_t$ is relatively high (above the $x_t = 0$ locus), the economy always ends up at $PT$ regardless of the value of $k_t$. In other words, even the richest economy could potentially slip into a new low-growth regime if the prevalence rate in the country becomes sufficiently large as a result of an exogenous disease shock for example.

Transition to the balanced growth path can exhibit interesting dynamics. In Figure 2, the trajectory starting from point $M$, initially shows slow growth and rising disease prevalence. The slow growth comes from the effect of diseases on mortality and productivity as well as lower savings due to a large portion of incomes being devoted to disease prevention. This preventive investment ultimately overcomes infectious diseases. The prevalence rate peaks and then declines monotonically as the economy takes-off into balanced growth. The take-off is fueled by capital accumulation shifting toward the higher savings of uninfected workers. In the limit, the growth rate converges to $\gamma^H \equiv (1-\alpha)s^U A - 1$. For a trajectory starting at point $N$, in contrast, the economy grows steadily as it converges to the balanced growth path and diseases decline monotonically.

### 4.3 Policy in the Benchmark Case

As we just saw, economies converge to either a poverty trap or a balanced growth path depending on initial conditions. For an economy that ends up at $PT$, an interesting question is whether subsidies are effective in taking it out of the trap. We explore this issue next. Figure 3 shows dynamics induced by international health subsidies ($x^{sub}$) and international capital-investment

Figure 3: Subsidies to Health and Capital Investment



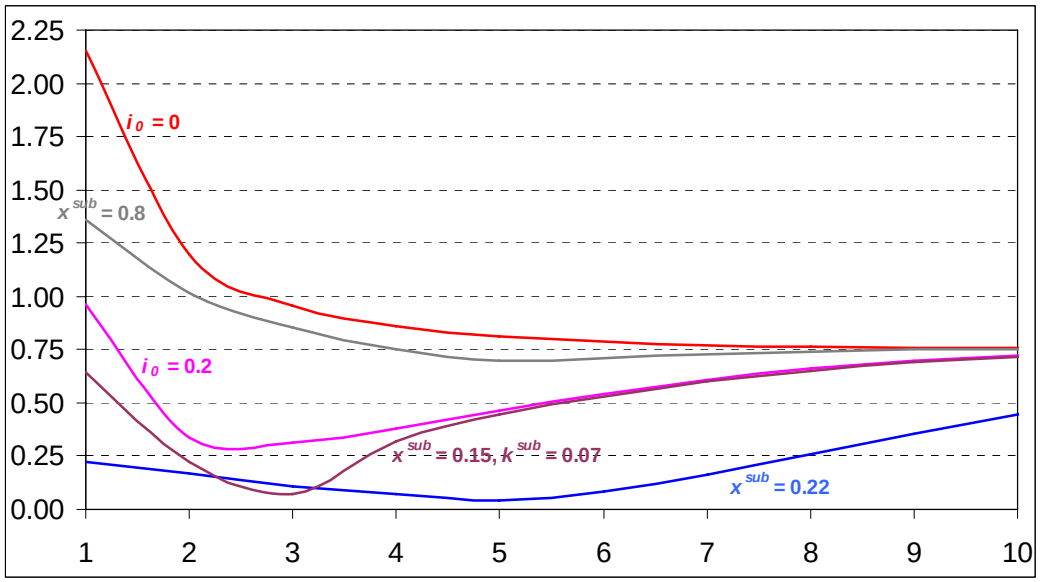

subsidies ($k^{sub}$).[14] The label to the right of each line denotes the characteristics of the policy package ($x^{sub}, k^{sub}$) and the number of generations during which it is implemented.

An immediate consequence of the model's dynamics described in the previous section is that no $k$ subsidy alone can take the economy to the $BGP$. As shown in Figure 3, when international donors supply $k^{sub} = 0.8$ (which amounts to 22% of GDP at $PT$) to each generation, an economy that starts at $PT$ only moves to a slightly higher income poverty trap, $PT'$. Also, insufficient funds to help prevention may reduce the long-run prevalence rate below one but the economy grows enough to reach a higher income poverty trap. This is illustrated in Figure 3 when $x^{sub} = 0.20$ and the economy escapes $PT$ but moves to $PT''$. Escaping the trap is, on the other hand, possible through health subsidies alone, provided that $x^{sub}$ is large enough.

Given the method used to calibrate the model parameters, a $x^{sub}$ equal to 0.22 (7.2% of GDP at $PT$) is the minimum amount required to take the economy from $PT$ to $BGP$. The minimum health subsidy required will constitute our policy benchmark to which we compare other policy

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

Figure 4: Growth Rate per Generation, Starting from $K = 0.094$




scenarios. In particular, a health subsidy of 0.22 has to be provided for at least 9 generations to achieve that goal. In addition, important scale economies are associated with $x^{sub}$ in the sense that the number of subsidized generations required to escape the trap falls rapidly with $x^{sub}$. For instance, if we double preventive subsidies (i.e., $x^{sub} = 0.44$), the subsidy has to be provided for only 3 generations instead of 9. When $x^{sub} = 0.8$, this falls to only 1 generation.

Even though capital subsidies *per se* cannot take the economy to *BGP*, they can improve the effectiveness of health subsidies. This is true provided that $x^{sub}$ is sufficiently large – for the benchmark parameterization, $x^{sub}$ needs to be at least 0.11. As shown in Figure 3, 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.

An additional perspective on the cost of infectious diseases is offered by Figure 4. The chart displays growth rates of per capita GDP per generation under different scenarios. All of these

growth paths share two common characteristics. At $t = 0$ they each start with the capital stock associated with the poverty trap $PT$ and they all converge to the long-run growth of 75% (that is, an average annual growth rate of 1.8%). Line $i_0 = 0$ corresponds to an economy with an initial prevalence rate equal to zero and that, consequently, never suffers from infectious diseases (transition dynamics exist due to diminishing average product of $k$). Line $i_0 = 0.2$ represents the time path of a second economy that has a relatively small initial prevalence rate of 20% and never receives subsidies. Notice that this economy convergences to $BGP$. However, the cost imposed on it by infectious diseases is large in terms of lost growth. Compared to $i_0 = 0$ path, growth rates along the $i_0 = 0.2$ path are less than half during the first 4 generations.

Economies that escape $PT$ through subsidies can nevertheless suffer for a while. Think about an economy near $PT$ at $t = 0$ that starts receiving a subsidy $x^{sub} = 0.22$ every generation. This economy converges to the $BGP$ very slowly. As we see in Figure 4 (see $x^{sub} = 0.22$ line), the initial growth rate is more than 10 times lower than in the zero prevalence case. Note also that the $x^{sub} = 0.22$ path does not reach the half life of the convergence process until the $10^{th}$ generation. In other words, an economy that receives the minimum perpetual subsidy needed to escape the trap will experience growth rates that are less than half of that of the developed world for about three centuries.

The figure also illustrates how $k$ subsidies complement $x$ subsidies. But starting from $PT$, even a package $(x^{sub}, k^{sub}) = (0.15, 0.07)$ that can push the economy to the $BGP$ requires 4 generations (more than one century) to reach its half life. We can accelerate the convergence by raising the subsidies. For example, a massive subsidy towards preventive health ($x^{sub} = 0.8$ line) generates growth rates that are close to their $i_0 = 0$ counterparts. It is important to note, however, that this is true only if the subsidy is supplied every period. Recall that package $(x^{sub}, k^{sub}) = (0.8, 0)$ needs to be implemented only for one generation to move the economy from $PT$ towards $BGP$. If the package is cancelled in period 2, the convergence path would be close to the $i_0 = 0.2$ line; consequently, reaching half life would again required more than a century.

Our main results from these benchmark exercises can be summarized as follows: $(i)$ there are two stable steady states, a zero-growth trap and a balanced high-growth path, $(ii)$ any economy, regardless of its income level, can diverge towards the trap if the prevalence rate becomes sufficiently high for exogenous reasons, $(iii)$ when an economy is at the trap, subsidies to capital accumulation cannot fully substitute for health aid; specifically, a minimum value of $x^{sub}$ is indispensable in order to take the economy out of the trap, $(iv)$ policy packages that rescue the economy from $PT$ in only one generation are the least costly, $(v)$ if such massive health improvements are not possible, a policy package that combines both types of subsidies is preferred, and $(vi)$ when a developing

nation shows high prevalence rates, the cost in terms of growth rates can be relatively large – reaching half of the process can take several centuries even when the economy is converging to the $BGP$.

Finally, we want to conclude this section with a word of caution. Given uncertainty about our functional choices and disease-related parameter values, ours results are better interpreted qualitatively than quantitatively. While we can be confident about the direction of changes or the relative values of these changes in the experiments above, the exact magnitudes should be interpreted cautiously.

# 5 Two Alternative Cases

In this section we present two alternatives to our benchmark scenario by changing $a$ and $\phi$. First, we focus on the case where $a$ is sufficiently low. Here the $BGP$ is the unique steady state but economies with high prevalence rates go through a very slow convergence process. Secondly, we study what happens when $\phi$ is relatively high. In this case the development trap is no longer characterized by zero growth. We conclude the section by performing robustness checks with respect to other parameter values.

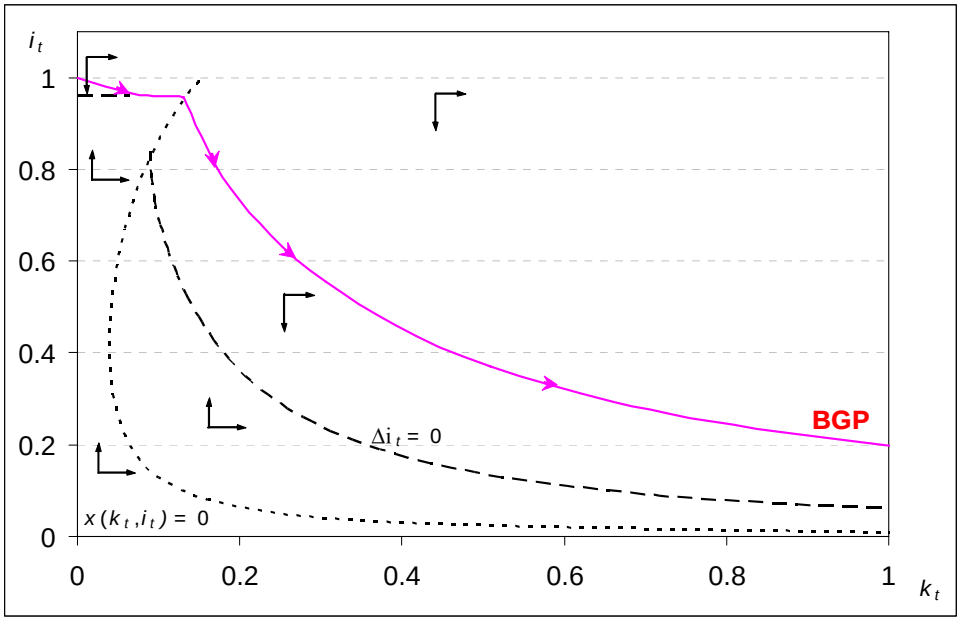
## 5.1 Slow Convergence without the Low-Growth Trap

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

More specifically, for the benchmark parameterization, a $PT$ still exists for $a \in (0.49, 1)$ though the prevalence rate falls below one. The low-growth trap vanishes when $a$ falls below 0.49. For such low values, the $\triangle k_t = 0$ schedule disappears from the phase plane and optimal preventive investment is always positive ($x > 0$) for all $(k, i)$ such that $k > 0.15$ and $i > 0.09$. As a result, no trap exists and all economies convergence to the unique $BGP$ irrespective of initial conditions. Figure 5 present the phase diagram for $a = 0.49$.

An important question here is whether costs of infectious diseases remain large even in absence of a development trap. Suppose that $a = 0.49$ and the economy starts developing from initial values $K_0 = 0.09$ and $i_0 = 0.96$. A prevalence rate of 0.96 is the maximum that the economy can endogenously reach for $a = 0.49$. Figure 6 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.

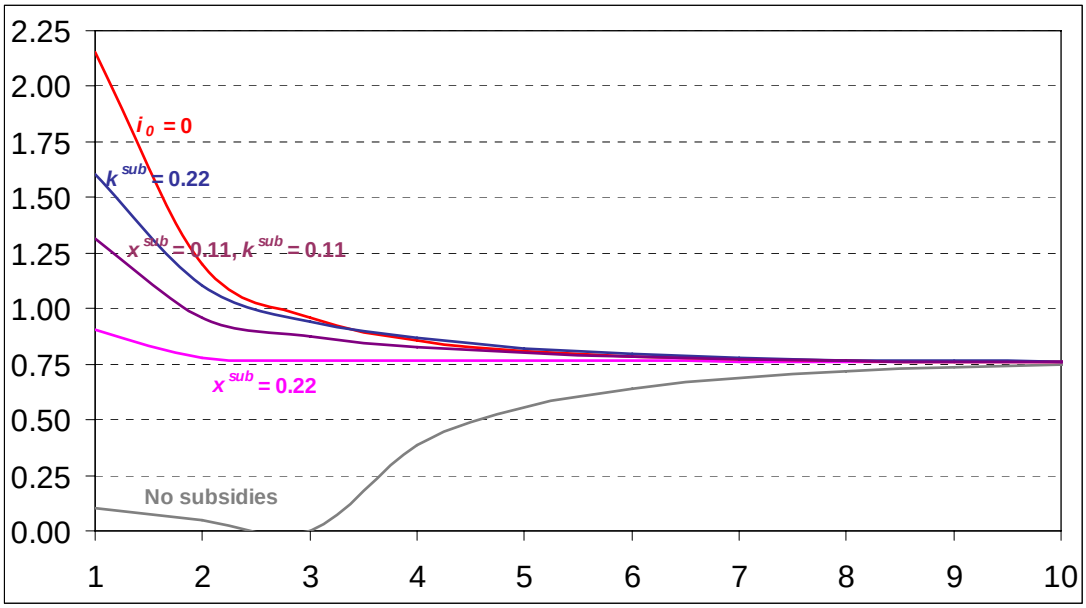
Figure 5: Phase Diagram for $a = 0.49$




The effect of infectious diseases on the economy is smaller than before but still substantial. If the economy does not receive international aid, growth-rate convergence takes several centuries. This case is represented in Figure 6 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.

It is clear that policies are now significantly more effective. Moreover, in contrast to the benchmark, subsidies to capital accumulation are always more effective in raising the growth rate. In Figure 6 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 $(0.22, 0)$. It is important to note however, that subsidizing capital only may not be optimal if we take into account the evolution of life expectancy. Suppose that the economy is located at $i = 0.96$ and $K = 0.09$. Furthermore, assume that life expectancy at birth ($LE$) is 50 years for the first generation if no subsidies are granted. As Figure 7 shows, a subsidy package $(x^{sub}, k^{sub}) = (0, 0.22)$ leaves $LE$ for generation 1 unchanged at 50 years because the effect of $k^{sub}$ does not impact the current generation. The package $(x^{sub}, k^{sub}) = (0.22, 0)$, in contrast, generates a $LE$ for generation 1 of 60 years which is a substantial difference. For generation 3 onwards, $k^{sub} = 0.22$ generates slightly higher $LE$ than $x^{sub} = 0.22$, but the difference is never above 2 years. Therefore, a central planner who values the

Figure 6: Effect of Subsidies without Poverty Trap ($a = 0.49$)




$LE$ of different generations could choose an intermediate policy such as $(x^{sub}, k^{sub}) = (0.11, 0.11)$.

We conclude that when development traps do not exist, the cost of infectious diseases is still sizeable. But capital subsidies are more productive in speeding up growth-rate convergence than in the benchmark model.

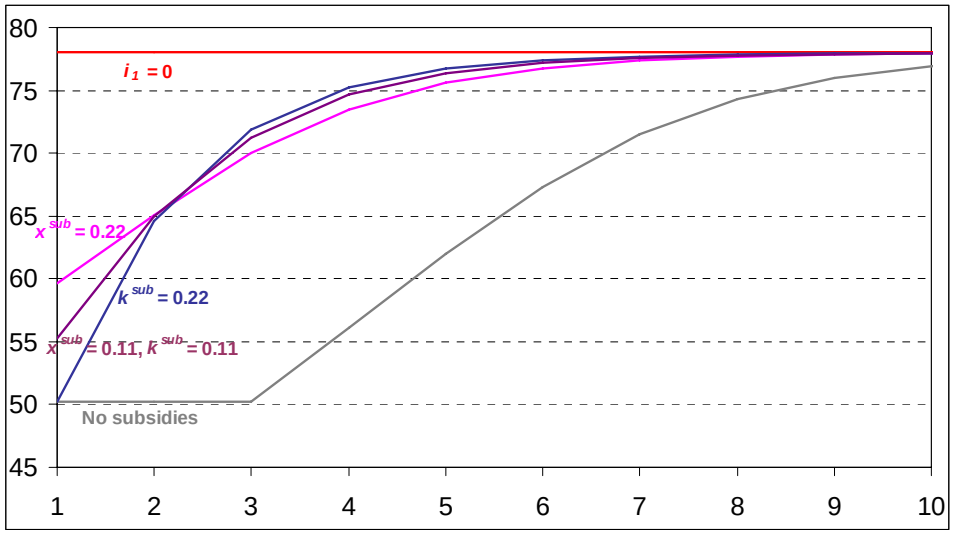
### 5.2 Multiple Balanced-Growth Paths

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

For the next experiment, we assign a value of 0.73 to $\phi$ which implies that, in the low-growth trap, the long-run growth rate of output per capita will equal 0.1%, the average growth for sub-Saharan Africa from 1990 to 2003 (UNDP 2005). The phase diagram for this scenario is given in Figure 8. We observe that, as in the case where $a$ is sufficiently low, the $\triangle k_t = 0$ schedule vanishes, implying that the capital stock grows from any point in the $(k, i)$ plane. The figure illustrates dynamics for two economies: both start with the same level of physical capital but different prevalence rates (15% and 20%, respectively). The economy that starts with a prevalence

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

Figure 7: Evolution of Life Expectancy for $a = 0.49$ under Different Policy Packages

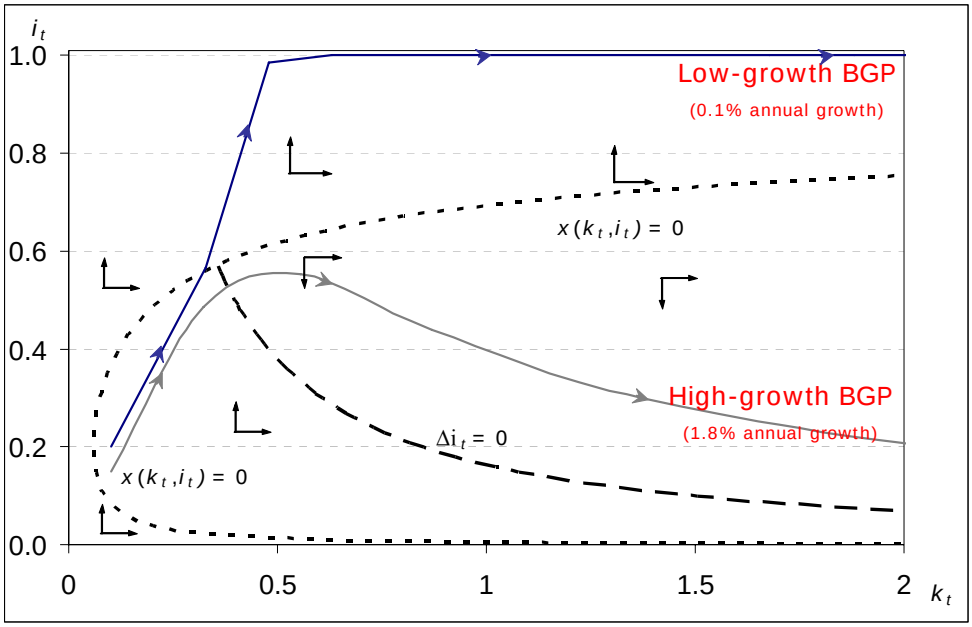



rate of 15% experiences an increase in the infectious diseases during 2 generations, but eventually the prevalence rate drops to zero and the economy convergences to a balanced growth path with annual growth of 1.8%. The economy with an initial prevalence rate of 20%, show a continuous rise on the prevalence rate until everyone is infected. In the long-run, this economy does not invest in prevention and output per capita grows at 0.1% per year.

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

Like the survival probability, the parameter $A$ directly affects long-run growth. However, it makes sense to assume that its benchmark value (24.19) is the maximum which $A$ can take. The reason is that $A$ also affects economies that move along the high-growth steady state and are, therefore, on the technology and institutional frontier. Given this, it is easy to illustrate that changes in $A$ could have at most a modest impact on a developing nation's long-run growth and health. Suppose that, initially, $\phi = 0.72$ and $A = 19$. The latter implies an annual steady-state growth rate of 1% in a zero prevalence economy instead of the benchmark 1.8%. In this case, the poverty trap appears at $(k, i) = (0.6, 1)$. In addition, assume that there is an improvement in the institutional environment that provides higher protection of property rights and enforcement of

Figure 8: Phase Diagram for $\phi = 0.73$




contracts, causing $A$ to rise to 24.19. An economy that was previously located in the poverty trap would now experiences a modest increase in long-run growth from 0% to 0.2% and no change in its population's health status.

### 5.3 Further Robustness Analysis

Finally we perform robustness analysis using alternative parameter values related to disease transmission ($\mu$, $q$), costs ($\delta$, $\theta$) and the production technology ($\alpha$, $A$). We also examine robustness to values of $\phi$ lower than 0.71, validating our parametric choice in the case presented above. Results for a subset of the experiments appear in Table 2.

Table 2 specifically shows robustness checks with respect to pairs of $(\mu, q)$ that are compatible with our calibration method, $(2, 0.55)$ and $(10, 0.06)$. The same type of steady states exist for these new values and the location of the $PT$, $(k, i) = (0.11, 1)$, is not affected. The reason is that $\mu$ and $q$ affect savings only through preventive investment but at $PT$, $x = 0$. Table 2 also provides information on the location of $UPT$. This is useful in order to know the state space over which dynamics lead to $PT$. The new $UPT$ associated with $(\mu, q) = (2, 0.55)$ is $(k, i) = (0.34, 0.79)$. It is easy to deduce, if we compare this to the $UPT$ in Figure 2, that the state-space which leads to $PT$ shrinks. The opposite is true for $(\mu, q) = (10, 0.06)$. The difference here is mainly due to $\mu$. The

Table 2: Robustness Checks for Key Parameters

| **Cases** | $PT$ | | $UPT$ | | $x^{sub}$ * | | $x^{sub}$ & $k^{sub}$ † | | | Gen. till half-life, $x^{sub}_{Min}$ ‡ |
|---|---|---|---|---|---|---|---|---|---|---|
| | $k$ | $i$ | $k$ | $i$ | $x^{sub}_{Min}$ | Gen. | $x^{sub}$ | $k^{sub}$ | Gen. | |
| Benchmark | 0.11 | 1.00 | 0.29 | 0.58 | 0.22 | 9 | 0.15 | 0.07 | 5 | 9 |
| $(\mu, q) = (2, 0.55)$ | 0.11 | 1.00 | 0.34 | 0.79 | 0.22 | 22 | 0.15 | 0.07 | 7 | 26 |
| $(\mu, q) = (10, 0.06)$ | 0.11 | 1.00 | 0.33 | 0.41 | 0.22 | 7 | 0.15 | 0.07 | 5 | 6 |
| $q = 0.06$ | 0.11 | 1.00 | – | – | 0.06 | 4 | 0.04 | 0.05 | 4 | 3 |
| $\alpha = 0.34$ | 0.11 | 1.00 | 0.43 | 0.58 | 0.26 | 13 | 0.16 | 0.10 | 8 | 15 |
| $A = 19$ | 0.09 | 1.00 | 0.39 | 0.58 | 0.29 | 20 | 0.19 | 0.10 | 6 | 35 |
| $\delta = 1$ | 0.11 | 1.00 | 038 | 0.57 | 0.24 | 23 | 0.16 | 0.06 | 12 | 24 |
| $\theta = 0$ | 0.11 | 1.00 | 0.36 | 0.59 | 0.25 | 13 | 0.15 | 0.10 | 7 | 15 |
| $\phi = 0.65$ | 0.71 | 1.00 | – | – | 0.11 | 3 | 0.11 | 0.01 | 3 | 5 |

Notes:

* Minimum health-prevention subsidy (no k subsidy) to escape PT, and minimum number of subsidized generations

† Package ($x^{sub}$,$k^{sub}$) and minimum number of subsidized generations to escape PT

‡ Generation that reaches half-life growth for the $i = 0$ case, starting from PT and receiving $x^{sub}_{Min}$ health aid per period

number of meetings between susceptible young and possibly infected old individuals determines the strength of the negative externality. As we increase $\mu$, the state-space within which people invest in preventive health gets smaller and, as a consequence, it is easier to end up in the trap.

Subsidies turn out to be more effective as $q$ declines. Hence, when $(\mu, q) = (10, 0.06)$, it is easier to fall into the trap but also cheaper to escape from it. In particular, Table 2 shows that, compared to the benchmark, the minimum health subsidy needs to be provided only to the first 7 generations, instead of 9, and reaches half the growth rate for $i_0 = 0$ by the $6^{th}$ instead of the $9^{th}$ generation. Of course, although cheaper to eliminate, infectious diseases remain very costly when $(\mu, q) = (10, 0.06)$. Similarly, we can deduce from the table that infectious diseases are more costly than in the benchmark case and subsidies less effective for $(\mu, q) = (2, 0.55)$. Finally, a combined policy package such as $(x^{sub}, k^{sub}) = (0.15, 0.07)$ continues to be more productive in escaping from $PT$ than only $x^{sub} = 0.22$.[16]

As before, the complementarity effect of capital-aid on health subsidies becomes weaker as the effectiveness of health subsidies rises. Consider the row corresponding to $q = 0.06$ in Table 2. In this case, prevention is very effective. A subsidy $x^{sub} = 0.06$ supplied during only 4 generations could take a trapped economy to $BGP$ and no combination $(x^{sub}, k^{sub})$ such that $x^{sub} + k^{sub} = 0.06$ could do better. We also see that the more expensive package $(x^{sub}, k^{sub}) = (0.04, 0.05)$, needs to

[16] We also considered $(\mu, q) = (2, 0.06)$ with lower values of both $\mu$ and $q$. $PT$ still exists but $UPT$ is eliminated. In addition, it is harder to fall into the trap and easier to escape. Physical capital subsidies do not complement health subsidies much since preventive investment is already very productive.

be provided at least for 4 generations.

Table 2 also shows that qualitative results are not very sensitive to changes in the other parameters $\alpha$, $\delta$, $\theta$, and $A$ either. This is also true for changes in $\phi$ provided that it does not exceed 0.71. In particular, we try $\alpha = 0.34$, $\delta = 1$, $\theta = 0$, $q = 0.06$, $A = 19$, and $\phi = 0.65$. The most remarkable findings come from the morbidity and mortality parameters. Table 2 shows that the disease cost from reduced morbidity is more severe. In particular, the minimum $x^{sub}$ and number of generations needed to escape $PT$ as well as the area that leads to the trap increase as the utility cost disappears (that is, $\delta = 1$) and the effective labor lost $\theta$ falls to zero. The reason is that people feel less pressure to invest in prevention as morbidity costs fall. As expected, the cost of getting out of $PT$ falls fast with $\phi$. For example, when $\phi = 0.65$ a subsidy $x^{sub} = 0.11$ provided for 3 generations is sufficient to escape the trap. However, like morbidity, a greater survival probability implies that the state-space that leads to $PT$ expands. But the reason now is quite different: it is simply the result of a higher value of capital in the poverty trap.

We summarize the results of section 5 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, health subsidies are strictly preferred to policy packages that combine capital and health aid, not only when a massive preventive investment is undertaken but also when the prevention technology offers relatively high returns. Third, our main results remains strong. In particular, any economy, regardless of its income level, can diverge towards the low-growth attractor (when it exists) if prevalence becomes sufficiently high. As a consequence, a minimum health aid is a prerequisite to escape from this development trap. In addition, diseases impose a very high cost in terms of lost growth, regardless of which attractor dominates the economy's dynamics.

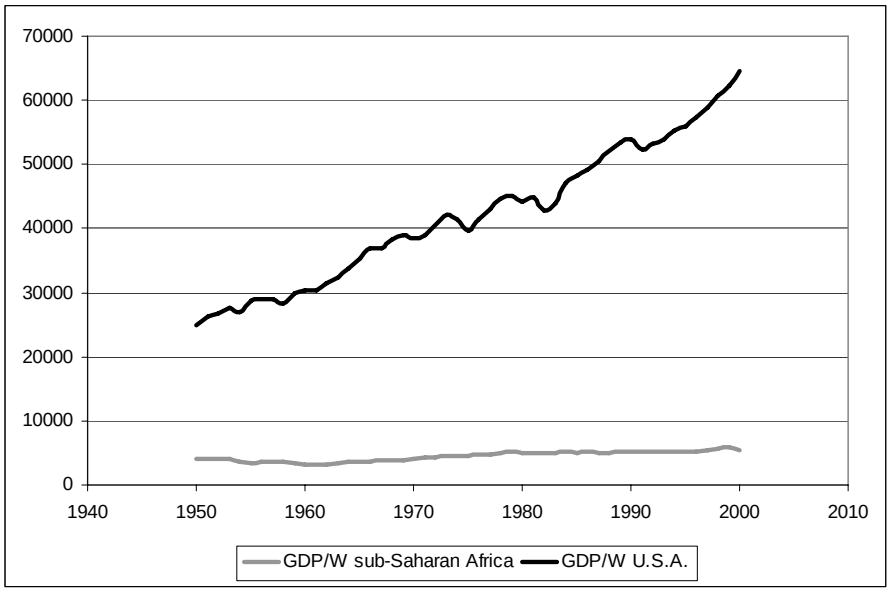
# 6 Diseases and Africa

Among the growth facts development economists agree on is sub-Saharan Africa's (SSA) divergence from the rest of the world. Figure 9 portrays one version of this divergence: SSA's output per worker grew at an average annual rate of 0.6% during $1950 - 2000$ against 1.9% for the US (Penn World Table 6.2).

It is also widely recognized that SSA experiences a disproportionate share of the global disease burden, especially of infectious diseases. These diseases contribute about 30% to the overall disease burden (DALYs) worldwide, but in Africa 64% (Global Burden of Disease 2002, WHO). Much of the burden falls on infants and children. For instance, only 78% of newborns in SSA survive to

age 15 compared to 99% in developed countries. But infectious diseases take their toll on adults too. The probability of death between ages 15 and 60 is $40\% - 60\%$ for sub-Saharan African men compared to less than 15% in developed countries (Murray and Lopez, 1997; Gakidou *et al.*, 2004). About 53% of SSA's high adult mortality is due to infectious and parasitic diseases, the leading contributors being HIV/AIDS, tuberculosis and malaria (WHO, 2001b).

Figure 9: Africa's Income Divergence, $1950 - 2000$ (PWT 6.2)




We think infectious diseases can be conceptually and quantitatively important in explaining Africa's stagnation. Our theory shows the prevalence of diseases can slow down convergence and when these diseases are particularly virulent and costly (as they were for the benchmark calibration), the economy's long-run growth path can be fundamentally altered. These implications are consistent with Africa's growth experience: SSA suffers the world's highest incidence of infectious diseases and the slowest growth of output per worker. These facts can be interpreted both in terms of a low growth poverty trap and slow convergence to the same balanced growth path SSA is converging to as the rest of the world (see for example the 'no subsidies' line in Fig 6).

Infectious diseases, though, are not new and have not affected Africa alone through history. Epidemics and pestilence routinely plagued Western Europe, with considerable human cost, until the dawn of the Industrial Revolution. But diseases seem to have had little adverse consequence for Europe's subsequent industrialization.[17] It is then natural to ask what it is about Africa's

[17] The Black Death epidemic for example made little dent to Europe's economies (Clark, 2001).

environment that limits its growth potential. A look at the epidemiological literature provides some clues about how the model's driving forces – economic costs and disease transmission – differ substantially between Europe and Africa.

Consider malaria which ranks third among the continent's infectious disease threats but has caused much less destruction elsewhere (Dunn, 1993). African malaria is a chronic persistent problem not episodic like the plague, influenza or small pox that affected pre-industrial Europe in waves. This is due to several reasons. First, much of Africa's malarial fatality is due to *plasmodium falciparum*, the deadliest strain of the malaria parasite. This strain was absent or rare elsewhere. Secondly, the prevalence and severity of the disease depends on temperatures. Where temperatures drop during the winters, as in temperate climates, the prevalence is much reduced. In other words, temperate zones offer a natural check against a rampant outbreak of the disease. Thirdly, the stability of the disease in Africa makes it difficult to control. Malaria control efforts have faltered in the continent due to the 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 instrumental in eliminating the disease from many temperate regions during the early part of the twentieth century, and now to antimalarial drugs (Nchinda, 1998).

A similar difference emerges in the transmission of HIV in SSA. 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 in Africa, plays an important role and has been a challenge for health policies. A recent study estimates that the interaction between malaria and HIV may have been responsible for 8,500 excess HIV infections and 980,000 excess malaria episodes in Kenya (Abu-Raddad *et al.*, 2006). Such co-infection may have also made it easier for malaria to spread to areas with high HIV prevalence.

Humans in the Old World acquired immunity and resistance over thousands of years as a consequence of its temperate climate and domestication of animals (Diamond, 1999), endowing those populations with lower values of $a$ (the probability of getting infected in the absence of preventive investment) which would have increased the efficacy of preventive behavior. This evolution of $a$ on its own could well have freed Europe from the tyranny of diseases. Think, for example, of a population heterogenous in its disease resistance: less resistant individuals would die without being able to pass on their genes. Over time this lowers the average $a$ for the population and allows the

economy to escape diseases and stagnation.

In the case of Africa this immunization process may have been confounded by its encounter with the West and its diseases (Diamond, 1999).[18] Colonization introduced new diseases to non-immune populations in eastern, central, and southern Africa that were relatively more isolated than western Africa. Previously endemic diseases often took the form of epidemics so much so that the period $1880-1920$ has been described as a time of tumultuous "ecological disaster" (Lyons 1993). By the mid-nineteenth century, tropical Africans were afflicted by most of the diseases of the temperate Old World and as we saw above for HIV and malaria, the concurrence of multiple infectious diseases could easily have amplified morbidity and mortality costs ($\theta$, $\phi$ and $\delta$).[19]

Another determinant of the efficiency of preventive behavior in our model is the parameter $q$, which is inversely related to medical technology and public health systems. The epidemiological literature offers evidence why $q$ may have been substantially higher in Africa than in Europe. If colonialism brought new diseases to Africa, public health practices of the colonial powers did not help matters. Often large-scale medical campaigns were launched against single illnesses which were expensive but made little dent on the overall problem. In some cases, disease-specific knowledge was either absent or had limited transferability in Africa's virulent climate (Dunn, 1993).[20] The importance of public health systems in eradicating communicable diseases relates to the McKeown-Preston debate. McKeown (1976) held that nutrition played a vital role in Britain's mortality transition, more so than public health innovations and improvements in medicine. This view has been challenged by Preston (1996), among others, who presents evidence suggesting public health initiatives initially, and medical treatment improvements like vaccination later, were largely behind the rapid mortality decline in developed countries (see also Cutler *et al.*, 2006). Public health intervention meant, to a large extent, public subsidization of disease prevention like filtering and chlorinating water, draining swamps, and building sanitation systems. Modern Africa's disease problem has surely been exacerbated by its instability and chronic institutional failures. The great diseases, plagues and subsistence crises disappeared from western Europe by the early 1700's partly

[18] Africa's battle between man and diseases forms the basis of McNeill's (1998) stark thesis on the history of disease parasites. McNeill argues that the evolution of early humans in Africa's tropical climate led to the emergence of an ecological balance between man and micro parasite. It was only when humans began to migrate to colder climates in Europe and elsewhere that the battle for primacy between man and parasite started. In other words, Africa's infectious disease problem is simply nature's way of ensuring that no one species dominated. The reach of the tsetse fly and the sleeping sickness it transmits, for instance, still determines the range of human activity in many parts of the continent.

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

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

because of the increasing stability of governments which enhanced administrative efficiency (Kunitz, 1993). Not so in Africa, where wars have been extraordinarily common. Public health systems are widely ineffective due to corruption, lack of provision and in some cases, lack of skilled manpower (World Bank, 1993). One reason people continue to die from infectious disease is because they do not know what causes the disease, how it spreads or often confuse early symptoms for more benign illnesses.

Finally, Kiple (1993b) points out that another reason why infectious diseases have been costlier for Africa is the lack of appropriate nutrition. African soils are typically acidic, nitrogen deficient and deprived of minerals, especially calcium and phosphorus. As a result, crops were protein and mineral deficient. Since the sub-Saharan African diet was predominantly vegetarian, it was nutrition-deficient. Animal protein was relatively scarce. Few animals were available either because they were quickly hunted down or because they 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. Europeans, on the other hand, tackled diseases more effectively because of the nutritional quality of a diet derived both from fertile agriculture and domestication of animals (Diamond, 1999). These factors may affect $q$ in the sense that food intake would boost immunity less for African populations than for Europe.

A central lesson of our study is that health subsidies are vital to lower prevalence and generate faster growth due to important externalities associated to disease transmission. Moreover a dramatic improvement in the disease environment, not an increase in income, is needed. This opens the door to medical technology and public health initiatives which reduce $q$. Interventions in the form of vaccination, nutritional supplement, information campaigns, environmental improvements are, of course, easiest to channel through the public health delivery system. We find that general institutional improvements that raise aggregate productivity, $A$, have limited impact: these would modestly raise the growth rate and income levels, without lowering disease prevalence for a country in the low-growth path. Since diseases have a quality-of-life effect, welfare gains from faster growth will be smaller.

# 7 Conclusions

We care about health not only because it fundamentally affects quality of life but also because it may affect productivity. This paper makes the case that poor health due to infectious diseases has first-order effects in explaining the persistence of underdevelopment. More precisely, we present an epidemiological model where the transmission and incidence of infectious diseases depend on economic incentives and rational behavior. Incorporating rational behavior into a micro-founded

disease structure is our key theoretical innovation.

Our model's main insights are a consequence of the lower savings-investment propensity associated with mortality and morbidity. Preventive health investment can counter this effect to some extent but that depends on the strength of the negative externality from infectious diseases. The lower saving propensity of infected individuals implies that a low-growth trap can exist. Calibration exercises reveal that, compared to development traps studied by previous literature, the disease trap is different because it can attract any economy, regardless of its income level, when prevalence becomes sufficiently large. This strong finding is generated by the negative externality intrinsically associated with the transmission of infectious diseases. An important consequence of this is income *per se* does not cause health when prevalence is high. Successful interventions should therefore be health specific (e.g. in the form of vaccination or nutritional supplements) and channeled via the public health delivery system.

Our calibrations also reveal that unlike general institutional improvements that have limited impact, institutional improvements of the quality of the health sector (public and/or private) is instrumental in raising aggregate productivity.

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