Infectious Diseases, Human Capital and Economic Growth*

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DRAFT

Abstract

This paper investigates the joint determination of the transmission of infectious diseases, human capital accumulation and economic growth. We develop an economic epidemiological model by incorporating SIS epidemiological model into an endogenous growth model with human capital accumulation. Households choose how much to invest in human and physical capital, as well as in controlling the risk of infection. If an individual is infected, he is incapacitated and can neither work nor accumulate human capital. There are multiple balanced growth paths where the endogenous prevalence of the disease determines whether human capital is accumulated or not, i.e. whether there is sustained economic growth or a poverty trap. In the decentralized economy households fail to internalize the externality associated with controlling diseases. We further characterize the optimal solution and the subsidy that will decentralize it. With the optimal public health policy, economies are more likely to take off or grow at a higher rate. We show that there can be underinvestment in preventive health expenditures, and perversely for countries that are most afflicted with diseases and in a poverty trap, the optimal subsidy is lower than for growing economies. The poor health conditions are not only the result of tighter budget constraints, but more importantly the lack of incentives for investing in health capital.

JEL Classification: E19, I10, D90, O11.
Key Words: Infectious Diseases; Endogenous Growth; Epidemiology; Poverty Trap; Public Health Policy; Human Capital

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1 Introduction

The interaction of infectious diseases and economic well-being is a matter of importance for welfare and growth. Atleast since the formation of the Committee on Poverty and the Committee on Health set up by the National Constituent Assembly of revolutionary France in 1789 to examine public health policy on how diseases transmit (Susser and Stein (2009)), this been an important and controversial issue. However, economists have only recently begun to model the interaction of diseases and growth in dynamic general equilibrium models. This paper studies the joint determination of the transmission of infectious diseases, human capital and economic growth in a dynamic general equilibrium model. Can the incidence of diseases affect the growth paths of different economies? What is the effect of the externality associated with infectious diseases? How does it affect human capital accumulation? What is the optimal public health policy? These questions can be answered only with the help of detailed theoretical modeling where the variables are jointly determined.

To model the interaction of infectious diseases, human capital and economic growth we build on the Lucas (1988) model of endogenous growth where individuals allocate time between working and accumulating human capital. The difference in our environment is that individuals are exposed to the risk of being infected by infectious diseases that incapacitate them from working or accumulating human capital. The transmission of diseases builds on insights from the mathematical biology literature on epidemiology of infectious diseases. However, unlike the biology literature, there is a choice to spend resources, either private or public, to control the transmission of diseases by affecting their infectivity. As the diseases affect the ability to work and the productivity of human capital, their incidence affects the accumulation of human capital. This effect on human capital accumulation, in turn affects incentives to accumulate physical and health capital. Thus, we endogenize the three main objects of interest: the disease incidence through expenditures on health, human capital through its endogenous productivity, and income and welfare through both endogenous savings and allocation of savings to different forms of capital, and through the changing labor participation and productivity.

This paper is part of a larger project to incorporate epidemiology models into dynamic general equilibrium models (see Goenka and Liu (2012), and Goenka, Liu and Nguyen (2014)). The epidemiology models lend themselves to integration into economic models as they capture disease transmission via dynamical systems. In this paper we concentrate on recurring diseases, that is individuals can recover from the disease but recovery does not confer any subsequent immunity to either the same or other diseases. We adopt the canonical SIS epidemiological model.An individual is born healthy into a large household,[1] susceptible to the disease, S, may get infected and become infective, that is ill and capable of transmitting it to others, I, and then recover to become susceptible again, S. Models with more complicated epidemiological structure, however, bring little economic insight with added burden of complexity (see Hethcote (1994)).

We study the competitive equilibrium balanced growth paths in a decentralized economy

[1]The set-up of a large representative household is similar to the one commonly used in the labor search literature, which embeds a labor search structure into a dynamic general equilibrium model. As a result, economists can avoid from keeping track of the cross-sectional distribution of various economic variables. The model is reduced to a representative agent framework, though with heterogeneity within the household.

and show that the model generates multiple balanced growth paths (BGPs), where infectious diseases are either eradicated or endemic. In the disease-free case, countries grow at a faster rate, while in the disease-endemic cases, countries either grow at a slower rate or are in a poverty trap, depending on the investment in human capital, which in turn is influenced by the severity of the disease prevalence. The intuition is that marginal product of human capital investment depends on the effective labor force (or the disease prevalence), which itself is endogenously determined by the effective health capital. When the effective health capital is low and infectious diseases have high incidence, the return to human capital is extremely low. Thus, there is no incentive for human capital accumulation and countries are stuck in a poverty trap.

In the decentralized economy, households do not take into account how their own decisions affect the aggregate disease dynamics. Thus, they do not internalize the externality of infectious disease transmission. While this externality has been recognized (Geoffard and Philipson (1996), and Gersovitz and Hammer (2004)) it has not been modeled in a full dynamic general equilibrium environment. To examine the effect of public health policy we study the centralized economy where a social planner takes the disease externality into account. In this case, the effective health capital is higher, and thus, typically human capital accumulation and the growth rate are also higher.[2] However, public health policy does not guarantee sustained economic growth and there can be the situation where the disease incidence is so high that even in the planning outcome there is no human capital accumulation and economic growth. As delivery of effective public health programs remains a challenge in poor countries, we characterize the optimal subsidy that will decentralize the planning solution. The optimal subsidy is proportional and increasing in the size of the disease externality. It also depends on whether human capital is being accumulated or whether a country is growing or not. We show that there can be a perverse situation where countries that are most afflicted by infectious diseases will have lower subsidies than countries that grow, as the growth dividend from reducing the incidence of diseases is absent. Thus, for the least developed countries, the poor health conditions are not only the result of tighter budget constraints, but more importantly the lack of incentives for investing in health capital.

The macroeconomic effect of infectious diseases has been debated in the empirical literature. Most of the macro empirical literature, either using cross-country regressions or constructing macro effects from microeconomic estimates (Weil (2007)), and do not fully endogenize disease dynamics. So far, there is no consensus on the quantitative significance of effects of infectious diseases on the economy. Some papers find the effect of control of diseases to be large (Gallup and Sachs (2001), Bloom, et al. (2014)), while others find the effect is modest (Ashraf, et al. (2009)) or there might even be an adverse effect due to the dilution effect of a larger population and increase in dependency ratio (Acemoglu and Johnson (2007), Young (2005)). Largely using quasi-experiments, the microeconomic empirical literature tends to find larger effects of diseases on human capital (Bleakley (2007, 2010), Cutler, et al. (2010), Fortson (2011), Lucas (2010), and Miguel and Kremer (2004)).

[2]The underinvestment in preventive health expenditures predicted by the model is consistent with the evidence that there is underinvestment in preventive health by those who are most afflicted by the infectious diseases in LDCs, see Banerjee and Duflo (2011) and Tarozzi, et al. (2009).

However, how this may aggregate in the macroeconomy is not yet fully understood.

Recently, to understand the interactions in a general equilibrium framework, a few papers have started taking steps in building theoretical models by combining economic and epidemiological models. The papers that have used explicit epidemiology modeling (Bonds, et al. (2009), and Delfino and Simmons (2000)) look at Solow-type models with fixed savings decisions. Thus, how optimal choices change with disease incidence are not modeled, which also restricts the welfare implications that can be drawn. Azomahou, et al. (2016) look at the mechanism of HIV/AIDS incidence on human capital and growth in a calibrated Solow type model. However, they do not endogenize the disease dynamics or the optimal savings decision (see also Cuddington and Hancock (1994)).

In Goenka and Liu (2012), we endogenized savings and the labor-leisure choice, with only a one-way effect – diseases affect the economy. The paper showed that there can also be endogenous fluctuations in productivity and hence, equilibrium outcomes due to disease transmission. These can be stabilized by isolation and vaccination programs. However, to address endogeneity of both diseases and economic activities, we need to simultaneously model both capital accumulation and the epidemiological structure of the diseases. Goenka, Liu and Nguyen (2014) is a first step in this direction within a framework of a neo-classical growth model. In the current paper, we extend the analysis to an endogenous growth model, fully endogenizing both disease transmission and choices of physical, human and health capital investment.

There are two kinds of effects of diseases: disease related mortality and morbidity (illness). Much of the macroeconomic literature in explaining diseases and poverty, concentrate on premature death and its consequences on growth largely through health and pre-mature mortality and and its effect on fertility (e.g. Chakrabory, et al. (2010), Chakraborty, et al. (2014), Kalemli-Ozcan, Romer and Weil (2000), and Soares (2005)). This complementary approach followed in Chakraborty, et al. (2010), Chakraborty, et al. (2014), and Aksan and Chakraborty (2014) using a neo-classical overlapping generations framework focusses on the effect of pre-mature mortality (Chakraborty (2004)) due to infectious diseases on income and growth. In our paper, using a infinitely lived household framework, we focus on recurring diseases to study their effect on human capital accumulation. We thus, abstract from the fertility and mortality nexus.[3] to study interaction between incidence of diseases and capital choice decisions (physical, health, and human). This is motivated by macroeconomic stylized facts presented that show clustering of countries into three groups: low disease incidence, high income and growth, and high human capital (measured by educational attainment); higher disease incidence, lower income and growth, and lower educational attainment; and high disease incidence, low income and no growth, and low educational attainment. This is consistent with the microeconomic evidence that shows morbidity affects educational attainment (see Bleakley (2007, 2010), Cutler, et al. (2010), Lucas (2010), and Miguel and Kremer (2004)). The modeling enables us to study the dynamic general equilibrium effect of the disease externality and see how underinvestment in preventive health expenditures in

[3]The evidence of diseases on fertility is mixed. Some papers find evidence of the standard Beckerian channel of decrease in disease incidence increases demand for quality rather than quantity of children, and hence, fall in fertility (e..g. Bleakley and Lange (2009) who study hookworm eradication in southern USA. Others find no evidence of changes of disease incidence on fertility (e.g. Fortson (2009), and Kalemli-Ozcan and Turan (2011) who both study effect of HIV/AIDS in Africa.)

a decentralized economy can affect incentives to accumulate human capital and thus, the growth rate.

The paper is organized as follows. Section 2 provides the stylized facts on the relationship between disease incidence, income and growth, and educational attainment. Section 3 presents the economic epidemiology model, and Section 4 examines multiple balanced growth paths in the decentralized economy. Section 5 studies the centralized economy and optimal public health policy. Section 6 contains the model calibrations and simulations. Section 7 concludes.

2 The Empirical Facts

In this section, we present the cross-country evidence on the relationship among diseases, human capital, and growth which motivates this paper. Specifically, a cluster analysis is used to group countries based on various economic, educational, demographic, and health related indicator variables. The reason we adopt a cluster analysis rather than reduced-form regression is two-fold: economic growth, human capital and disease prevalence are simultaneously determined, causing an endogeneity problem; and there is an asymmetric effect of disease control, causing a non-linearity problem. These issues impose a challenge for reduced-form regression and can be a reason for the sensitivity of the estimates for the impact of disease control on the economy.

How to measure the burden of infectious diseases? The mortality rate is often used as a measure, both for reasons of humanity and easy data accessibility. However, morbidity caused by infectious diseases is at least as important as mortality (see Bleakley (2007, 2010) for impact of diseases with morbidity but low mortality). Diseases with a low mortality rate but a high morbidity rate have effects in terms of both the direct cost of treating, and indirect of cost of being disabled from the disease. As a result, World Health Organization (WHO) provides a summary measure - disability adjusted life year rates (DALY) - to give a better indication of the burden of diseases from both mortality and morbidity. It is calculated as the ratio of sum of the years of life lost due to premature mortality (YLL) and the years lost due to disability (YLD) in the population.[4] As this paper focuses more on disability caused by infectious diseases, ideally we should be using YLD as the measure for the burden of infectious diseases. However, since YLD is not available at the country level, we use DALY in the following cluster analysis. As countries bearing the heavier burden of infectious diseases - higher in DALY – are higher in both YLL and YLD. For the cluster analysis, our results should be robust to any of the above measurements. Moreover, as DALY at country level is only available for year 2000 and 2010, we also include mortality rate caused by infectious diseases in 1965. For educational attainment at the country level, we use the updated average schooling years from Barro and Lee (2013), which is available from 1965 to 2010 at 5 years intervals. The rest of data used for the cluster analysis is from

[4]YLL basically corresponds to the number of deaths caused by infectious diseases multiplied by the standard life expectancy at the age at which death occurs. To estimate YLD, the number of incident cases in a certain period is multiplied by the average duration of the diseases and a weight factor that reflects the severity of the disease on a scale from 0 (perfect health) to 1 (death). For more details, please refer to WHO website: http://www.who.int.

the World Bank database, including GDP per capita in year 1965 and 2012, average growth rate from year 1965 to 2012, life expectancy in year 1965 and 2012.

Through the cluster analysis, we classify all the countries into three groups, which we call developed countries, developing countries and least developed countries (LDCs). The LDCs are largely in the Sub-Saharan African Region.[5] Table 1 describes the mean and confidence intervals of one standard deviation for the selected variables used in the cluster analysis at each group level. The average growth rate for developed countries is around 1.86%, for developing countries it is around 1.79%. In contrast, the LDCs have the lowest growth rate, and in particular, some countries are stuck in the poverty trap with a negative average growth rate. In terms of the spread of infectious diseases, the LDCs bear the heaviest burden of infectious diseases. On average, for each individual 38% of his time is lost due to either premature death or disability caused by infectious diseases. As a comparison, an individual in developing countries loses 6.5% of his time due to infectious diseases and this number is 1.52% for developed countries. The life expectancy at birth in developed countries is significantly higher than the one in developing countries, which again is significantly higher than the one in the LDCs. For the educational attainment, developed countries have the highest educational levels with 7.04 average schooling years in 1965 and 11.20 in 2010, while the LDCs have the lowest educational levels with 1.22 in 1965 and 4.44 in 2010. Countries with heaviest burden of infectious diseases are countries with lowest GDP per capita and associated with lowest average schooling years, though on average GDP per capita, life expectancy and educational level have risen for the past few several decades. Thus, there is a positive relationship between disease control and economic development, which motivates the economic epidemiology model in this paper.

This cross-country evidence is consistent with the micro empirical studies in the literature. For instance, Bleakley (2007) evaluates the economic consequence of the successful eradication of hookworm disease from the American South, and finds that areas with higher level of hookworm infection prior to the intervention experienced greater increase in school enrolment, attendance and literacy. Miguel and Kremer (2004) evaluate a Kenyan project with deworming drugs targeting intestinal helminths, and find that the program substantially reduced school absenteeism. The evidence on eradication or control of malaria also indicates positive effects on schooling, health capital and subsequent income (Bleakley (2010), Lucas (2010). Cutler et al (2010) find weak effects of malaria eradication in India. These micro empirical studies focus on diseases where the burden is predominantly in the childhood. There is a concern that if there is child labor then part of the effect of decline in morbidity increases child labor supply. Our model is an infinitely-lived agent framework (as we want to abstract from mortality effects of diseases) and agents can accumulate human capital in any

[5]There are in total 67 countries for which we have the complete data. Here, we present the list of countries in each group. The developed countries include Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany, Greece, Iceland, Israel, Italy, Japan, Netherlands, New Zealand, Norway, Singapore, Spain, Sweden, Switzerland, United Kingdom and United States. The developing countries include Algeria, Argentina, Barbados, Bolivia, Chile, China, Colombia, Costa Rica, Ecuado, El Salvador, Guatemala, Guyana, Honduras, Hungary, India, Indonesia, Iran, Jamaica, Malaysia, Mexico, Pakistan, Panama, Paraguay, Peru, Philippines, Portugal, Syria, Thailand, Trinidad and Tobago, Tunisia, Turkey, Uruguay and Venezuela. The least developed countries are Cameroon, Gambia, Ghana, Kenya, Mali, Niger, Senegal, Sierra Leone, Sudan and Zambia.

Table 1. Cluster Analysis for Three Grouped Countries

	Developed Countries	Developing Countries	Least Developed Countries
	Panel A: Economic Growth and Development		
Growth rate (1965-2012)	1.86%	1.79%	0.31%
	[1.20% 2.54%]	[0.62% 2.96%]	[−0.50% 1.12%]
GDP per capita 1965	9394.8	1498.6	361.2
	[4908.7 13880.8]	[196.8 2800.1]	[201.3 520.9]
GDP per capita 2012	24621.8	3498.9	410.4
	[15010.7 34232.9]	[888.2 6109.5]	[253.5 571.4]
	Panel B: Burden of Infectious Diseases and Demographics		
DALY 2010	1.52%	6.50%	38.02%
	[0.43% 2.61%]	[2.39% 10.59%]	[24.82% 51.22%]
Life expectancy 1965	70.5	55.8	40.8
	[68.2 72.8]	[49.1 62.5]	[34.5 47.1]
Life expectancy 2012	79.6	72.8	54.6
	[76.8 82.5]	[69.1 76.5]	[49.0 60.2]
	Panel C: Educational Attainment		
Years of schooling 1965	7.04	2.86	1.22
	[5.45 8.62]	[1.58 4.13]	[0.38 2.05]
Years of schooling 2010	11.20	7.80	4.44
	[9.67 11.87]	[5.83 8.84]	[1.83 6.17]

Note: The table describes the mean and confidence intervals of one standard deviation for variables used in the cluster analysis for each group of countries. The variables included in the table are growth rate from 1965 to 2012, GDP Per capita in year 1965 and 2012 (constant 2000 US$), DALY rate for year 2010, life expectancy in year 1965 and 2012, quality adjusted average schooling years for age 15+ from Barro and Lee (2013) for year 1965 and 2010.

period. This is consistent with the evidence as increase in human capital will subsequently increase income, but it also takes a more general view of human capital accumulation through non-schooling acquisition of skills.

3 The Model

3.1 SIS Epidemiology Model

Epidemiological modeling refers to dynamic modeling where the population is divided into groups based on their epidemiological status (e.g. S, susceptible and I, infective), and flows between the groups are specified by differential equations (as we develop the model in continuous time). Depending on the given disease, there are different disease transmission mechanisms with possibly more epidemiology states. In this paper we model recurring diseases where having the disease does not confer subsequent immunity. For these diseases, the SIS model is the canonical model.[6]

The total population, N, is divided into two groups: S, the susceptible (healthy and susceptible to the disease) and I, the infective (infected and capable of transmitting the disease).[7] Individuals are born at the rate b,[8] healthy and susceptible to the disease. We assume homogeneous mixing so that the likelihood of any individual contracting the disease is the same.[9] There is horizontal incidence of the disease i.e. transmission from peers. Let α be the average number of adequate contacts of a person to catch the disease per unit time or the contact rate. Then, the number of new cases per unit of time is $\alpha(I/N)S$, depending on the *fraction of the infected*. This contact structure is the standard incidence or frequency dependant model, commonly used in the epidemiology literature for human diseases. It is adopted as the pattern of human interaction is relatively stable and invariant to the size of the population.[10] The contact rate α is the key parameter and reflects two different aspects of disease transmission: the biological infectivity of the disease and the pattern of social interaction. Changes in either will change α.[11] The recovery of individuals is governed by the parameter γ and the total number of individuals who recover from the disease at each time period is γI. Upon recovery, individuals move back to the class of susceptible individuals.[12] Each individual faces the exogenous death rate, d, irrespective of

[6]Having more epidemiology states does not add significant additional insight at the cost of considerable complexity. For more details on the epidemiology models, see Hethcote (1994, 2005).

[7]The model is in continuous time. All variables are functions of time. However, we omit the subscript 't' throughout the paper, if no confusion caused.

[8]Birth is understood to mean entry to the *labor population* either through birth or migration. We abstract from the age structure in the paper.

[9]Thus, the network structure where who contacts whom which is important for disease where there is a choice of whom to contact, is abstracted away from, see Goyal and Vigier (2014)

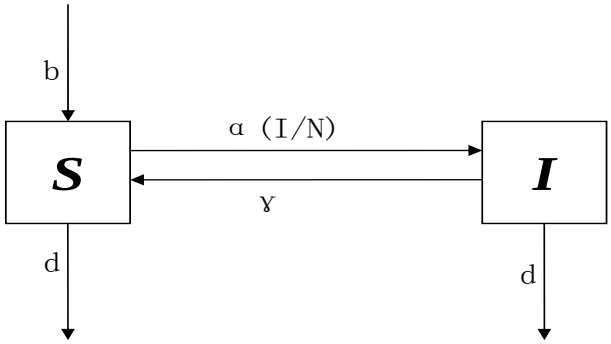
[10]Naively, it might seem plausible that the population density and hence the contact rate would increase with population size, but the daily contact patterns of people are often similar in large and small communities, cities and regions. For human diseases the contact rate seems to be only very weakly dependent on the population size. The other commonly used model, i.e., new cases equal to αIS, is used typically for herd animals. For more discussion about the form of the incidence, see Hethcote (2005).

[11]We endogenize α in the model, see section 2.2.

[12]Upon recovery, individuals may or may not develop immunity to the disease. Even though they have immunity to the disease, they are still susceptible to mutations of the disease, or other types of infectious diseases. One of the leading examples is flu: The flu virus mutates and each year there are new strains of flu diseases discovered. Immunity from one type of flu does not typically confer immunity to other strains.

health status.[13] Figure 1 describes the transfer diagram for the SIS model.

Figure 1. The Transfer Diagram For the SIS Epidemiology Model



Note: In a SIS epidemiology model, the total population is divided into two groups: the susceptible denoted as S and the infected denoted as I. The birth rate is b and newborns are born healthy and susceptible. All individuals irrespective of health status die at the rate d. The susceptible get infected at the rate $\alpha\frac{I}{N}$ and the infected recover at the rate γ. For more details, see Hethcote (2005).

The SIS epidemiology model is given by the following system of differential equations (Hethcote, 2005):

$$\begin{aligned}
&\dot{S} = bN + \gamma I - \alpha(I/N)S - dS \\
&\dot{I} = \alpha(I/N)S - \gamma I - dI \\
&N = S + I \\
&S, I \geq 0\,;\ S_0, I_0 > 0 \text{ given.}
\end{aligned}$$

The first equation shows that the change in the number of the susceptibles equals the inflow of newborns, bN, and the recovered, γI, minus the outflow due to both being infected, $\alpha(I/N)S$, and death, dS. Similarly, the second equation shows that the change in the number of the infected is the difference between the inflow of newly infected, $\alpha(I/N)S$, and the outflow of the those recovered, γI and dead, dI. As the total population consists of the susceptibles and the infected, letting $s = S/N$ be the fraction of the susceptibles we can simplify the dynamical system to:

$$\dot{s} = (b + \gamma)(1 - s) - \alpha(1 - s)s \tag{1}$$

with the total population growing at the rate $b - d$.[14] Note that the probability for a healthy individual to contract diseases is $\alpha(1 - s)$, depending on the contact rate α and the fraction

[13] Introducing disease-related mortality rate will make the discount factor non-linear and endogenous, since population growth is affected by the composition of the healthy and infected individuals, which are both endogenous variables. This will become clear in the following subsection, see equation (4). Nevertheless, we do comparative statics of varying death rate or life expectancy, see Section 5.

[14] Let $i = I/N$ be the fraction of the infected and $i = 1 - s$. We can rewrite the SIS epidemiological model as:

of the infected $(1-s)$ in the population. We maintain the assumption that $b-d \geq 0$, that is, the net population growth is always non-negative.

The SIS epidemiology model admits multiple steady states. One steady state is the disease-free steady state ($s^* = 1$), and the other is the disease-endemic steady state $\left(s^* = \frac{b+\gamma}{\alpha}\right)$. We note that the former exists for all parameter values, while the latter exists only when $\frac{b+\gamma}{\alpha} < 1$.[15] The epidemiology model described so far is a biological one with the disease transmission as given. In the economic epidemiology model we endogenize the disease transmission through health expenditures that affect infectivity of the disease, and study how this interacts with choices on physical and human capital.

3.2 The Economic Epidemiology Model

The model follows the Lucas (1988) endogenous growth model with human capital accumulation, where we incorporate the dynamics of disease transmission. To avoid keeping track of the cross-sectional distribution of the healthy and infected individuals, and to stay close to the canonical endogenous growth model, we adopt the framework of a large representative household.

The Households: We assume the economy is populated by a continuum of non-atomic identical households who are the representative decision-making agents. The size of the population in each household grows over time at the rate of $b-d \geq 0$. Within each household, an individual is either healthy or infected by the diseases. Each household is assumed to be sufficiently large so that the proportion of the household in each disease status is identical to the corresponding population proportion. Thus, within a household, the proportion of healthy individuals is s and the proportion of infected individuals is $1-s$. Each household understands and anticipates how the disease evolves and is fully forward-looking with regard to its possible future states as well as its present situation. However, following Gersovitz and Hammer (2004) the household considers itself small relative to the population and believes that the disease status within the household does not affect the proportion of infectives in the entire population. In particular, the household takes as given the proportion of the population that is infected, denoted as Π, and thinks the probability for the healthy individuals to contract disease is $\alpha\Pi$, rather than $\alpha(1-s)$. As a result, the disease transmission dynamics perceived by the households is now given as follows:

$$\dot{s} = (b+\gamma)(1-s) - \alpha \mathbf{\Pi} s.$$

This captures the idea that the household is small relative to the population and does not take into account the externality on disease transmission. It is competitive "disease taking" looking only at private benefits/costs and not social benefits/costs. This distinguishes the competitive model from the social planner's problem where this externality is taken into

$$\begin{aligned} \dot{s} &= b - ds - \alpha i s + \gamma i - s(b-d) \\ \dot{i} &= \alpha i s - \gamma i - di - i(b-d). \end{aligned}$$

Since $i = 1-s$, one of these equations is redundant.

[15] When both steady state co-exist, that is $\frac{b+\gamma}{\alpha} < 1$, the disease-free steady state is unstable.

account. The two different formulations also help distinguish between private health (where the externality is ignored) and optimal public health expenditure (where it is internalized).

There is a two-way interaction between the economy and the disease. On the one hand, diseases have direct adverse effects on the economy by reducing the labor force participation. Being infected with a disease affects the productivity of an individual. We make the simplifying assumption that an infected individual is incapacitated by the disease or that the productivity falls to zero. That is, the infected are unable to work or accumulate human capital.[16] We assume the labor is supplied inelastically.[17] For each household labor supply L is given by the proportion of the healthy individuals, and its dynamics inherits the dynamics of s:

$$\dot{L} \quad = \quad (b+\gamma)(1-L) - \alpha\Pi L. \tag{2}$$

Households take the interest rate R and wage W as given, rent out physical capital K and choose the fraction of of time to spend in work, $u \in [0,1]$, and in accumulating human capital, $(1-u)$. Thus, they provide effective labor supply eLu, where e is the average human capital. The income is either consumed C, invested in physical capital I_K or health capital I_H. Thus, the budget constraint is:

$$C + I_K + I_H \quad = \quad RK + WeuL. \tag{3}$$

We further assume there is full insurance within each household and all individuals have the same consumption irrespective of their health status. This is indeed optimal, if the household welfare aggregator is concave. The representative household's preferences are given as:

$$\int_0^\infty e^{-\rho t} u(C) N_t dt = \int_0^\infty e^{-(\rho - b + d)t} u(C) dt, \tag{4}$$

where ρ is the discount factor with $\rho > b - d$, and the initial size of household is assumed to be one. For analytical convenience, we assume the felicity function to take the following form: $u(C) = \log(C)$.[18]

Health and physical capital accumulations follow the standard laws of motion with the

[16] How much productivity is affected varies across diseases. The recent comprehensive estimates of disability weights used to compute DALYs is one possible measure of affect on productivity (see Salomon, et al. (2012), Murray, et al. (2012)). For some specific diseases there are estimates in the economic literature on loss of income from which effect on productivity is imputed (e.g. Baldwin and Weisbrod (1974) study effect of five parasitic diseases on banana plantation workers in St. Lucia; Fox, et al. (2004) study loss of income to tea pickers infected with HIV/AIDS in Kenya). The burden of diseases varies considerably, and the estimates in these studies are annualized. Our model is however, an aggregated continuous time model making it difficult to use these estimates. Assuming that the productivity falls to an intermediate level but not to zero will not affect the qualitative results.

[17] In Goenka and Liu (2012) we endogenize the labor-leisure choice with SIS disease dynamics and show that the dynamics are invariant under standard assumptions.

[18] The adoption of the usual CES utility function affects the quantitative results of the paper, but not the qualitative results. For simplicity of exposition, we use log utility.

deprecation rate δ:[19]

$$\dot{K} = I_K - \delta K - (b-d)K \quad (5)$$
$$\dot{H} = I_H - \delta H - (b-d)H. \quad (6)$$

The law of motion for human capital is given as:

$$\dot{e} = \psi e L(1-u), \quad (7)$$

where ψ is the effectiveness of human capital accumulation. The linearity in the above equation, i.e. non diminishing returns on human capital accumulation, implies human capital is the engine of economic growth. Unlike the standard endogenous growth model (Lucas (1988)), here it depends on the effective time spent in accumulation human capital $L(1-u)$, and both the components L and u are affected by the severity of disease prevalence.

In the paper we concentrate on preventive expenditures for controlling the infectivity of the disease via the contact rate α. In Goenka, Liu and Nguyen (2014) the recuperation rate γ is endogenized but as these two enter additively, for ease of exposition, we abstract away from the latter.

In the paper the interpretation of reducing α are health expenditures to control infectivity of the disease. We make α a function of effective health capital, $q = \frac{H}{K}$. An increase in health capital, H, reduces infectivity of the diseases by improving protection to infections, both physically and by strengthening the immume system. We take it to include investments related to health, such as improved sanitation. On the other hand, increases in physical capital, K, is modeled as increasing infectivity. This incorporates increased stress which impairs immunity, pollution, and increased economic activity that can increase exposure to diseases. More pollution can increase the incidence of diseases (Chauhan and Johnston (2003)); increased greenhouse gases change weather patterns leading to outbreaks of new diseases and spread of diseases where they were not prevalent (McMichael, et. al. (2006)); increased hyper-hygenic environments may reduce exposure to viruses and in childhood leading to greater illnesses later in life (McMichael (2003)); viruses causing the infection may mutate and become resistant to existing interventions (such as resistance to antibiotic and MRSA due to increased usage but not correct usage, which would be a part of health capital, (Goossens, et. al. (2004), Levy and Marshall (2004)); insect vectors become resistant to insectisides (Hemingway and Ranson (2000)); expansion of economic activity may change the natural nidality of diseases (Patz, et. al. (2003), Pavlovsky (1966)), in particular increased dams and irrigation leads to spread of schistosomiasis (Steinmann, et al. (2006)). Specifically, the contact rate is assumed to be a decreasing function of the ratio of health and physical capital, $\alpha\left(\frac{H}{K}\right)$. This is also required to guarantee a balanced growth path. If changing pattern of interaction is costly, for example loss of income by reduced activity then

[19] Having a common depreciation rate δ for both health and physical capital is inessential and the assumption is made for the sake of simplicity.

this is also compatible with the model.[20]

Assumption 1. *Define the effective health capital* $q := \frac{H}{K}$. *The contact rate* $\alpha(q)$ *is a* C^2 *function:*

1. $\alpha' < 0$, $\alpha'' > 0$ *and* $\lim_{q\to 0} \alpha' \to -\infty$, $\lim_{q\to\infty} \alpha' \to 0$;
2. *Let* $\overline{\alpha}$ *and* $\underline{\alpha}$ *be the upper and lower bound, respectively.*

$$\frac{b+\gamma}{\overline{\alpha}} < \frac{\rho - b + d}{\psi} < \frac{b+\gamma}{\underline{\alpha}} < 1.$$

The first assumption implies that contact rate is decreasing and concave in the effective health capital. The Inada condition is not necessary for the analysis but in its absence there can be another equilibrium where the disease is prevalent but there are no positive health expenditures, which we want to rule out here.[21] Eradication of endemic diseases is difficult and smallpox is the first, and so far the only infectious disease, to have been eradicated. It was largely due to a long-run coordinated vaccination program involving WHO and national assumptions. In the absence of sustained public efforts, diseases that were previously controlled can re-emerge as in the case of leprosy in India (Gokhale (2013)) and measles in the western countries. Most SIS diseases are also not amenable to effective vaccination strategies making their eradication problematic. Thus, we assume $\frac{b+\gamma}{\underline{\alpha}} < 1$, which implies an endemic disease cannot be eradicated by private health expenditures alone, and the disease free steady state is unstable. The assumption $\frac{b+\gamma}{\overline{\alpha}} < \frac{\rho-b+d}{\psi} < \frac{b+\gamma}{\underline{\alpha}}$ ensures that controlling diseases is relevant for the growth of a country. For countries afflicted by infectious diseases, when $\frac{\rho-b+d}{\psi} \leq \frac{b+\gamma}{\overline{\alpha}}$, all of them have a positive economic growth rate, and when $\frac{\rho-b+d}{\psi} \geq \frac{b+\gamma}{\underline{\alpha}}$, all of them are in the poverty trap, regardless of whether they control the diseases or not. Thus, to have an interesting economic problem, we assume $\frac{b+\gamma}{\overline{\alpha}} < \frac{\rho-b+d}{\psi} < \frac{b+\gamma}{\underline{\alpha}}$. This will become clearer in the following analysis.

The Firms: There are many perfectly competitive firms that maximize profit by choosing physical capital and effective labor as inputs. We assume the Cobb-Douglas production function $Y = AK^{\beta}(eLu)^{1-\beta}$, where A is the total factor productivity and $\beta \in (0,1)$ is the capital share. Thus, we have:

$$R = \beta A K^{\beta-1}(eLu)^{1-\beta} \tag{8}$$
$$W = (1-\beta)AK^{\beta}(eLu)^{-\beta}. \tag{9}$$

Competitive Equilibrium: A competitive equilibrium is a feasible allocation $\{C, K, H, I_K, I_H, L, u, e\}$ and a price system $\{R, W\}$ such that, given prices:

[20] In Kremer's model (1996) increased sexual contact increases utility so the cost is directly in terms of utility. In his model there are no long run dynamics of changing behavior which in our framework is modelled as changing the health capital.

[21] See Goenka, Liu and Nguyen (2014) for analysis of the corner solution with no health expenditure in the absence of this Inada condition.

- Households maximize equation (4) by choosing consumption C, health expenditure I_H, physical capital investment I_K and time allocation u, subject to the constraints equation (2) – (3), (5) – (7), and $0 \leq u \leq 1$, $0 \leq L \leq 1$, $I_H \geq 0$, with e_0, K_0, H_0, and L_0 given;
- Firms maximize profits, given by equation (8) and (9);
- The capital market, labor market and goods market clear;
- Since each household is representative of the population, in equilibrium

$$\Pi = 1 - L. \tag{10}$$

4 Competitive Equilibria

In this section, we analyze the competitive equilibrium balanced growth paths (BGPs). The current value Hamiltonian for the household's optimization problem is:

$$\begin{aligned} H = & \log(C) + \lambda_1[RK + WeuL - C - I_H - \delta K - (b-d)K] + \lambda_2[I_H - \delta H - (b-d)H] + \\ & + \lambda_3 \psi e L(1-u) + \lambda_4[(b+\gamma)(1-L) - \alpha\left(\frac{H}{K}\right)\Pi L] + \theta_1(1-u) + \theta_2(1-L) + \theta_3 I_H, \end{aligned}$$

where $\lambda_1, \lambda_2, \lambda_3$ and λ_4 are costate variables or shadow value of increments to physical capital, health capital, human capital and labor supply, respectively. θ_1, θ_2 and θ_3 are the Lagrange multiplies for the inequality constraints.[22]

On the margin, goods must be equally valuable in their use as consumption, physical capital investment and health expenditure:

$$\begin{aligned} & \frac{1}{C} = \lambda_1 = \lambda_2 + \theta_3 \\ & \theta_3 \geq 0, \quad I_H \geq 0, \quad \theta_3 I_H = 0; \end{aligned} \tag{11}$$

and labor time must be equally valuable in either production or human capital accumulation:

$$\begin{aligned} & \lambda_1 WeL = \lambda_3 \psi e L + \theta_1, \\ & \theta_1 \geq 0, \quad 1 - u \geq 0, \quad \theta_1(1-u) = 0. \end{aligned} \tag{12}$$

[22] It has been recognized in the literature that SIS dynamics are not concave which can make the Hamiltonian non-concave, and difficult to check whether the maximized Hamiltonian is concave or not. Thus, the usual Mangasarian and Arrow sufficiency conditions cannot be used. Goenka, Liu and Nguyen (2014) investigate this issue in detail. They show that if the growth rate of capital is bounded from below, $\dot{K}/K \geq -\kappa, \kappa > 0$, then there is a solution to the maximization problem. It relies on showing that the feasible set is relatively compact in $L^1(e^{-(\rho-b+d)t})$. They then show that the first order conditions to the maximization problems are indeed optimal. Thus, we work with the first order conditions in this paper. (See also d'Albis et al (2008)). There is an additional problem due to the decentralized decision problem. For existence in the Lucas (1988) model also see d'Albis and Le Van (2006)). These methods can be adapted to the model, but is beyond the scope of the current paper.

The changes of shadow values satisfy the following conditions:

$$\dot{\lambda}_1 = (\rho - b + d)\lambda_1 - \lambda_1(R - (\delta + b - d)) - \lambda_4\alpha'\left(\frac{H}{K}\right)\frac{H}{K^2}\Pi L \tag{13}$$

$$\dot{\lambda}_2 = (\rho - b + d)\lambda_2 + \lambda_2(\delta + b - d) + \lambda_4\alpha'\left(\frac{H}{K}\right)\frac{1}{K}\Pi L \tag{14}$$

$$\dot{\lambda}_3 = (\rho - b + d)\lambda_3 - \lambda_3\psi L(1-u) - \lambda_1 W u L \tag{15}$$

$$\dot{\lambda}_4 = (\rho - b + d)\lambda_4 - \lambda_3\psi e(1-u) + \lambda_4\left[b + \gamma + \alpha\left(\frac{H}{K}\right)\Pi\right] - \lambda_1 W e u + \theta_2$$

$$\theta_2 \geq 0, \quad 1 - L \geq 0, \quad \theta_2(1-L) = 0. \tag{16}$$

Thus, the competitive equilibrium is described by equation (2) – (3), (5) – (7), (8) – (10) and (11) – (16), along with the TVCs.[23]

From the epidemiology dynamics, there are two types of BGPs. One is the disease-free case with $L^* = 1$, where infectious diseases are eradicated, and all individuals are healthy and working. The other is the disease-endemic case with $L^* = \dfrac{b+\gamma}{\alpha(q^*)} < 1$, where infectious diseases are prevalent, and a fraction of individuals are infected and unable to work. These two cases mirror the two steady states in the pure SIS epidemiology model. The difference is that here households can influence disease transmission through choices on health expenditures which are in themselves determined endogenously.

Since for the disease-endemic case labor is a function of effective health capital, for easy exposition, in a BGP, we define the continuous function $L(q)$ such that

$$L(q) = \frac{b+\gamma}{\alpha(q)},$$

which is increasing in q. We further define the unique critical value $\hat{q}$ such that

$$L(\hat{q}) = \frac{b+\gamma}{\alpha(\hat{q})} = \frac{\rho - b + d}{\psi}.$$

Proposition 1. *There exist both a disease-free BGP and a disease-endemic BGP.*

1. *There exists a disease-free BGP with $L^* = 1$, $u^* = \frac{\rho-b+d}{\psi}$, and growth rate $g = \psi - (\rho - b + d)$;*

2. *There exists a disease-endemic case with $L^* = L(q^*)$.*

   (a) *If $L^* > \frac{\rho-b+d}{\psi}$ or $q^* > \hat{q}$, there exists a disease-endemic BGP, with $u^* = \frac{\rho-b+d}{\psi L^*}$ and $g = \psi L^* - (\rho - b + d)$;*

   (b) *If $L^* \leq \frac{\rho-b+d}{\psi}$ or $q^* \leq \hat{q}$, there exists a disease-endemic poverty trap, with $u^* = 1$ and $g = 0$.*

[23] See the proof of Proposition 1 in the Appendix for more details.

Moreover, the effective health capital q^* is determined by the equation

$$G(q^*) = max\{G_L(q), G_R(q)\} = 0,$$

where

$$\begin{aligned} G_L(q) &= -\frac{1-\beta}{\beta}\alpha'(q)(1-L(q))(1+q) - \alpha(q) - (\rho - b + d), \quad \textit{and} \\ G_R(q) &= -\frac{1-\beta}{\beta}\alpha'(q)(1-L(q))(1+q)\frac{\psi L(q)}{\rho - b + d} - \alpha(q) - (\rho - b + d). \end{aligned}$$

Proof. See the Appendix. □

G_L is the net marginal value of labor when there is no human capital accumulation, and G_R is the net marginal value of labor when there is human capital accumulation. Both are functions of effective health capital, q. As the choice of human capital accumulation is endogenous, for any q the higher of the two will be chosen. The equilibrium effective health capital q^* is determined when the upper contour of the two is equal to zero (see below).

In the disease-free case, infectious diseases are completely eradicated, and thus health expenditure for controlling diseases is zero. The maximization problem degenerates to the standard Lucas (1988) model, where countries undergo positive growth path if the effectiveness of human capital accumulation is larger than the effective discount rate, that is, $\psi > \rho - b + d$.

The intuition for the determinants of economic growth when diseases are endemic, is similar to the disease-free case. Human capital accumulation is the driving force for growth, which depends on the relative magnitude of marginal value of time use in education and production. Assuming all the time is allocated for production and the growth rate is zero, the marginal value of additional du unit of time in education is $\lambda_3 \psi e L du$, and the marginal cost is the value associated with loss in production, $\lambda_1(1-\beta)AK^{\beta}(eL)^{1-\beta}du$. Therefore, more time is devoted to education if the former is larger than the latter. By $\dot{\lambda}_3 = 0$ as the growth rate is assumed to be zero, we have $\lambda_3(\rho - b + d) = \lambda_1(1-\beta)AK^{\beta}(eL)^{-\beta}L$, and thus there is a positive growth only if

$$\psi L^* > \rho - b + d.$$

This implies that when the effectiveness of human capital accumulation, now proportional to the labor supply, is larger than the effective discount rate, the country undergoes positive growth path. Compared with the disease-free case, here marginal value of time use in education depends on the proportion of healthy individuals in a household. As a result, higher disease prevalence reduces the effectiveness of human capital accumulation, and more time is allocated for production rather than education, and there is slower growth. In the extreme case, all the time is allocated for production and there is a poverty trap.

Whether countries undergo growth or are in a poverty trap is directly linked to the severity of disease prevalence, which itself is endogenously determined by the effective health capital. We now look at how the effective health capital, q^*, is determined. When infectious diseases

are endemic, health expenditure is strictly positive, and we have $\lambda_1 = \lambda_2$ and

$$\lambda_1 \beta A K^{\beta-1} (euL)^{1-\beta} + \lambda_4 \alpha'(q) \frac{H}{K^2}(1-L)L = -\lambda_4 \alpha'(q) \frac{1}{K}(1-L)L, \tag{17}$$

by combining equation (13) and (14). It implies that the marginal value of physical capital investment equals the marginal value of health expenditure. We further show that along the BGPs, consumption, physical, health and human capital all grow at the same rate $g = \psi L(1-u)$, and $\frac{\dot{\lambda}_1}{\lambda_1} = \frac{\dot{\lambda}_3}{\lambda_3} = -g$, $\frac{\dot{\lambda}_4}{\lambda_4} = 0$. Through some manipulations, equation (15) is given as:

$$\lambda_3 \psi L(1-u) + \lambda_1 (1-\beta) A K^{\beta} (euL)^{-\beta} uL = \lambda_3 (\rho - b + d + g), \tag{18}$$

that is, marginal value of human capital, consisting of its contributing to both human capital accumulation and production, equals to marginal cost. Similarly, equation (16) becomes:

$$\lambda_1 (1-\beta) A K^{\beta} (euL)^{-\beta} eu - \lambda_4 (b + \gamma + \alpha(q)(1-L)) + \lambda_3 \psi e (1-u) = \lambda_4 (\rho - b + d), \tag{19}$$

that is, marginal value of labor supply, consisting of its contribution to production, evolution of labor force participation and human capital accumulation, equals to marginal cost. Divide both sides of the above equation by λ_4, substitute into equations (17) and (18), and we have:

$$\begin{aligned} &-\frac{1-\beta}{\beta}\alpha'(q)(1-L(q))(1+q) - \alpha(q) - \frac{1-\beta}{\beta}\alpha'(q)(1-L(q))(1+q)\frac{\psi L(q)(1-u)}{\rho - b + d} \\ &= \rho - b + d, \end{aligned} \tag{20}$$

which is a function of both the effective health capital q and the fraction of time allocated for production u. Hence, equation (20) along with equation (12) determine the equilibrium q^* and u^*.

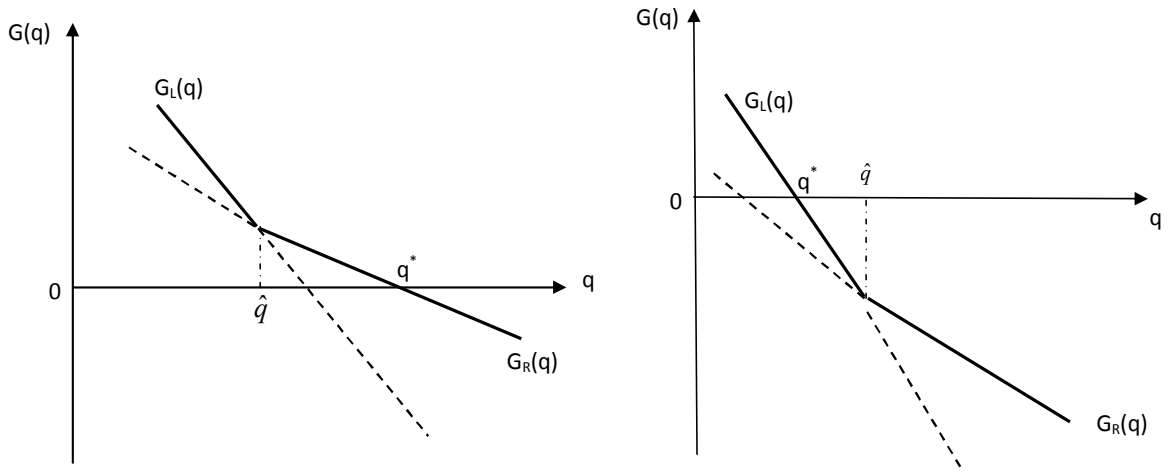
There are two cases. One is the poverty trap with $u^* = 1$. Equation (20) simplifies to $G_L(q) = 0$, suggesting q^* is chosen such that marginal cost of labor is equal to its marginal value, consisting of the first two terms in the L.H.S. of equation (20). Because there is no economic growth, the third term disappears. This case exists only if $\psi L^* \leq \rho - b + d$ or $q^* \leq \hat{q}$. The other case is a positive economic growth path with $u^* = \frac{\rho - b + d}{\psi L^*}$ and $g = \psi L^* - (\rho - b + d)$. q^* is determined by the equation $G_R(q) = 0$, derived by substituting u^* into equation (20). This case exists only if $\psi L^* > \rho - b + d$ or $q^* > \hat{q}$. Moreover, $G_L(q) > G_R(q)$ if $q < \hat{q}$, $G_L(q) < G_R(q)$ if $q > \hat{q}$, and $G_L(q) = G_R(q)$ if $q = \hat{q}$. Combining the two cases, q^* is determined by the upper contour of the functions G_L and G_R. That is, it is determined by the function $G(q) = \max\{G_L(q), G_R(q)\} = 0$. Since the function G is continuous, $\lim_{q \to 0} G = +\infty$ and $\lim_{q \to \infty} G < 0$, by intermediate value theorem, there exists a $q^* > 0$ such that $G(q) = 0$, that is, there exists an endemic-disease case.

Furthermore, the following lemma guarantees the uniqueness of q^*.

Lemma 1. *If $\alpha''(q)$ is large enough,*[24] *the function $G(q)$ is monotonically decreasing, and there exists a unique q^* such that $G(q) = 0$. If $q^* \leq \hat{q}$, it is a disease-endemic poverty trap; if $q^* > \hat{q}$, it is a diseases-endemic BGP.*

Proof. See the Appendix. □

Figure 2. Determination of Equilibrium Effective Health Capital: Disease-endemic BGP (Left Panel) and Disease-endemic Poverty Trap (Right Panel)



Note: The figure describes the two scenarios in the disease-endemic case. It depicts the function $G(q)$ – the upper contour of the functions $G_L(q)$ and $G_R(q)$, which determines the equilibrium effective health capital q^*. If q^* is greater than the critical value $\hat{q}$, countries grow at a positive rate with disease endemic, shown in the left panel; and if q^* is less than the critical value $\hat{q}$, countries are stuck in a poverty trap with disease endemic, shown in the right panel.

Figure 2 describes the two scenarios under the disease-endemic case. In both panels, the functions $G_L(q)$ and $G_R(q)$ are monotonically decreasing in q, and intersect at the point $\hat{q}$. The function $G(q)$ is given by the upper contour of both functions. The left panel gives the disease-endemic BGP with $q^* > \hat{q}$, and the right panel gives the disease-endemic poverty trap with $q^* < \hat{q}$. This suggests that whether there is positive or zero economic growth depends on the function $G(q)$, which in turn depends on all the economic, demographic and epidemiological parameters.

[24] We assume $\alpha''(q)$ is large enough, that is,

$$\alpha''(q) > -\alpha'(q)\ \max\{\frac{\beta}{(1-\beta)(1-L(q))(1+q)} + \frac{L(q)}{1-L(q)} \cdot \frac{\alpha'(q)}{\alpha(q)} + \frac{1}{1+q},$$
$$\frac{\beta}{(1-\beta)(1-L(q))(1+q)} \cdot \frac{\rho - b + d}{\psi L(q)} + \frac{L(q)}{1-L(q)} \cdot \frac{\alpha'(q)}{\alpha(q)} + \frac{1}{1+q} - \frac{\alpha'(q)}{\alpha(q)}\}.$$

Proposition 2. *When infectious diseases are endemic, countries are more likely to undergo a positive economic growth path, if:*

1. *Capital share, β, is smaller;*
2. *Households are more patient, i.e. ρ is smaller;*
3. *Death rate, d, is lower or life expectancy increases;*
4. *Effectiveness of human capital accumulation, ψ, is higher.*

Proof. See the Appendix. □

When labor becomes more important in production, that is, capital share is smaller, households care more about labor force participation rate and spend more on health expenditure. When households becomes more patient, they are more willing to postpone consumption and invest more in health capital. As result of this, labor force participation rate increases and hence countries are more likely to be in a growth path. When effectiveness of human capital accumulation is higher, it is more profitable to spend time in investing human capital rather than production, and the possibility of taking off increases. Nevertheless, the effects of changing the birth rate and recovery rate are ambiguous. On the one hand, due to the assumption that all newborns are healthy, higher birth rate is beneficial for controlling diseases (as is a higher recovery rate). On the other hand, when diseases are not severe and the fraction of the infected is low, there is less chance for the healthy individuals to catch diseases, which lowers the incentive for diseases control and hence reduces the health expenditure. The different deep parameters in the model reinforce the different capital choices and hence of growth. For the poorest countries, the constellation of parameters seem to work in the same direction to reduce the possibility of growth.

The following lemma details the resource allocation for each type of countries.

Proposition 3. *The resources are allocated as follows:*

1. *For countries in a disease-free BGP, the saving rate is $\beta\left(1-\frac{\rho-b+d}{\psi+b-d+\delta}\right)$;*
2. *For countries in a disease-endemic BGP, the saving rate is $\beta\left(1-\frac{\rho-b+d}{\psi L^*+b-d+\delta}\right)$, of which $\frac{q^*}{1+q^*}$ fraction is invested in health expenditure;*
3. *For countries in a disease-endemic poverty trap, the saving rate is $\beta\left(1-\frac{\rho-b+d}{\rho+\delta}\right)$, of which $\frac{q^*}{1+q^*}$ fraction is invested in health expenditure.*

Proof. See the Appendix. □

Since $\psi+b-d+\delta > \psi L^*+b-d+\delta > \rho+\delta$, Proposition 3 implies that countries in a disease-free BGP in fact have the highest saving rate and countries in a disease-endemic poverty trap have the lowest saving rate. For the countries with diseases eradicated, all the

savings are invested in physical capital as infectious diseases are eradicated and there is no need to spend resources in combating infectious diseases.[25] For the countries afflicted by infectious diseases, $\frac{q^*}{1+q^*}$ fraction is invested in health expenditure and the rest is invested in physical capital.

To sum up, as the result of the introduction of SIS epidemiological model, there are multiple competitive equilibria, in which infectious diseases are either be eradicated or are endemic. In the disease-free case, countries grow at a fast rate,[26] while in the disease-endemic case, countries either grow at a slow rate or are in a poverty trap, depending on the investment in human capital accumulation - the engine of economic growth, which is affected by the severity of disease prevalence. Therefore, countries with lower disease prevalence are more likely to invest in human capital, and hence be in a economic growth path. The intuition is that as the incidence of disease prevalence goes down, households expect a larger proportion to be healthy which increases the rate of return on human capital accumulation. This has the natural effect of increasing its accumulation. It implies that projections of the economic burden of disease which largely focus on lost productivity and cost of treatment are going to underestimate the cost as they do not account for the changed incentives for human capital accumulation and thus not account for the change in the growth rate.

5 Optimal Public Health Policy

In this section, we examine the centralized economy and characterize the optimal public health policy where a social planner takes into account the effect of controlling diseases at the household level on the aggregate disease dynamics. We then characterize the subsidy that decentralizes the centralized outcome.

5.1 Centralized Economy

The centralized economy differs from the decentralized one in that social planner takes into account that the intervention can effectively control the proportion of the infected in total population. Recall that in the decentralized economy household takes the proportion of the infected in total population as fixed, shown in equation (2). The social planner's maximization problem is essentially similar to the one we considered above with the only

[25]This does not contradict the fact that the developed countries have a high health expenditure to GDP ratio. The estimation results from cross-country panel data in the OECD countries suggest that technological progress and variation in medical practice are major determinants in the level and growth of health expenditure. More importantly, these countries are largely affected by non-communicable diseases or chronic illness, instead of infectious diseases. Health expenditure in our set-up are the resources spent on combating infectious diseases and rich countries in the model do not spend anything on it as the diseases have already been eradicated.

[26]However, we know from the disease dynamics in section 2.1 disease-free equilibrium is not stable if $b+\gamma<\alpha$. Since $b+\gamma<\alpha(q)$ for all q by the assumption, economic growth with disease eradicated is not a stable BGP. This explains why in developed countries, even though diseases are eradicated people are still concerned about the possible outbreak of infectious diseases.

difference being in the law of motion for labor force participation, which is now given as:

$$\dot{L} \quad = \quad (b+\gamma)(1-L) - \alpha(1-L)L.$$

In the following analysis, the superscript c is used in denoting variables in the centralized economy.

Proposition 4. *In a centralized economy,*

1. *There exists a disease-free BGP with the growth rate* $g^c = \psi - (\rho - b + d)$;
2. *There exists a disease-endemic case with* $L^{*,c} = L(q^{*,c})$.
   (a) *If* $L^{*,c} > \frac{\rho-b+d}{\psi}$ *or* $q^{*,c} > \hat{q}$, *it is a BGP with* $u^{*,c} = \frac{\rho-b+d}{\psi L^{*,c}}$, *and* $g^c = \psi L^{*,c} - (\rho - b + d)$;
   (b) *If* $L^{*,c} \leq \frac{\rho-b+d}{\psi}$ *or* $q^{*,c} \leq \hat{q}$, *it is a poverty trap.*

Moreover, the effective health capital $q^{*,c}$ *is determined by the equation*

$$G(q) + b + \gamma = 0.$$

Proof. The proof is similar to the proof of Proposition 1. □

Similar to the decentralized case, there always exists a disease-free balance growth path. Since the social planner and the households only differ in how they view the impact of their behavior on the disease transmission, there is no difference between the optimal growth path and competitive equilibrium path when diseases are eradicated.

There also exists a disease-endemic case. The effective health capital is optimally chosen according to:

$$\lambda_1(1-\beta)AK^{\beta}(euL)^{-\beta}eu - \lambda_4\left(b+\gamma+\alpha(q)(1-L)-\alpha(q)L\right) + \lambda_3\psi e(1-u) = \lambda_4(\rho - b + d).$$

The right hand side of the above equation is marginal cost of labor supply and the left hand side is marginal value of labor supply, consisting of its contribution to production, the evolution of labor force participation and human capital accumulation. Compared with equation (19), since social planner takes into account the positive externality of disease control, the marginal value of labor is always higher in the centralized than the decentralized economy, exactly by the amount $\lambda_4\alpha(q)L$ or $\lambda_4(b+\gamma)$. Thus, in the centralized economy, the effective health capital $q^{*,c}$ is determined by the equation $G(q) + b + \gamma = 0$, which is higher in the centralized economy than the decentralized one. It suggests infectious diseases are better controlled in the centralized economy and the labor force participation rate is higher. Thus, with an effective public health policy, it is more likely that countries can escape the poverty trap or grow at a faster rate.

To be more specific, Figure 3 describes three different scenarios for the comparison between the decentralized and centralized economies. In all the panels, the solid line is the

function $G(q)$, determining the effective health capital q^* in the decentralized economy, and the dashed line is the function $G(q) + b + \gamma$, determining the effective health capital $q^{*,c}$ in the centralized economy. The critical value $\hat{q}$ for the positive growth is the same in both economies. In the upper panel, the country is in a positive balanced growth path with the decentralized economy, while it grows at a faster rate with the centralized economy. That is, $q^{*,c} > q^* > \hat{q}$, $L^{*,c} > L^* > \frac{\rho-b+d}{\psi}$ and $g^c > g > 0$. In this case, the saving rates in both economies are given as $\beta(1 - \frac{\rho-b+d}{\psi L+b-d+\delta})$, $\frac{q}{1+q}$ fraction of which is invested in health expenditure. Notice these are increasing functions of labor supply. Thus, the centralized economy has a higher saving rate and investment rate for health expenditure, is better in controlling infectious diseases (lower $\alpha(q)$), and hence, grows at a faster rate.

In the bottom left panel, the decentralized economy is stuck in a poverty trap, while the centralized economy is in a positive growth path. That is, $q^{*,c} > \hat{q} \geq q^*$, $L^{*,c} > \frac{\rho-b+d}{\psi} \geq L^*$ and $g^c > g = 0$. In this case, because individuals fail to take into account the positive externality of disease control, the economy is stuck in the poverty trap, which otherwise would have taken off in a centralized economy. The saving rate in the centralized economy is given as $\beta(1 - \frac{\rho-b+d}{\psi L^{*,c}+b-d+\delta})$, while in the decentralized economy, it is given as $\beta(1 - \frac{\rho-b+d}{\rho+\delta})$. Since $\psi L^{*,c} + b - d + \delta > \rho + \delta$, the saving rate is higher in the centralized economy, more resources are allocated for controlling infectious diseases, and hence the country escapes the poverty trap.

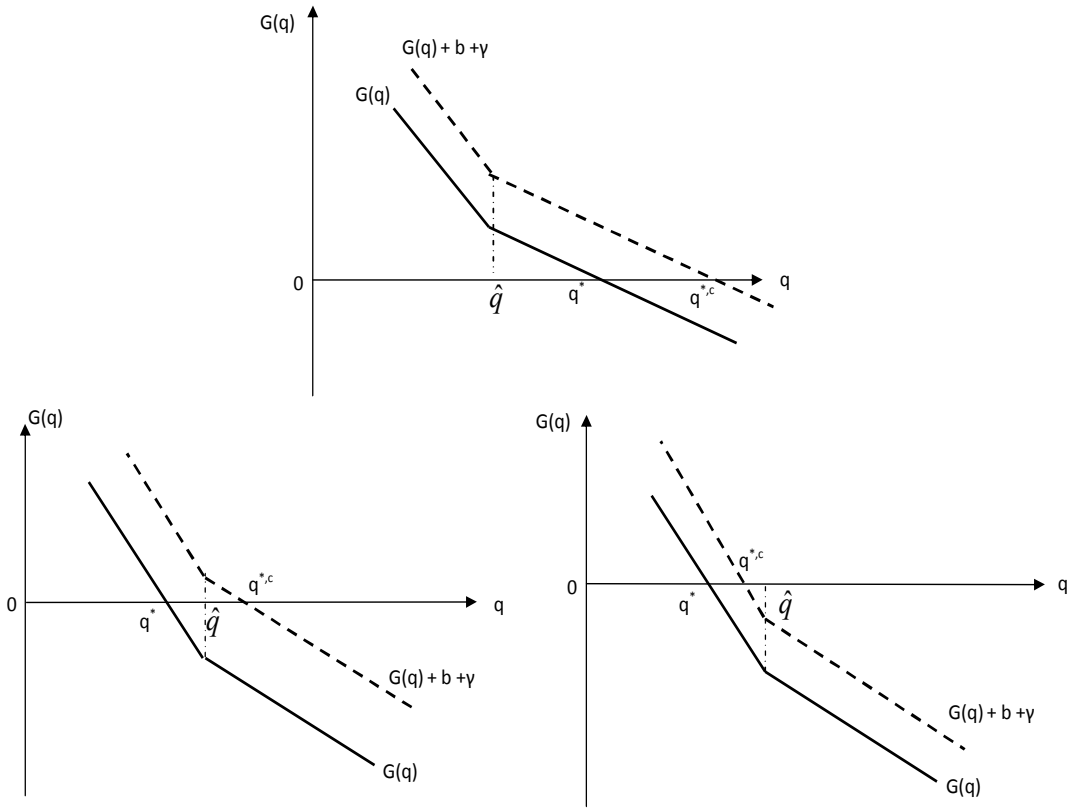
In the bottom right panel, both the centralized and decentralized economies are in the poverty trap, but the centralized economy has larger proportion of healthy individuals than the decentralized one. That is, $\hat{q} \geq q^{*,c} > q^*$, $\frac{\rho-b+d}{\psi} \geq L^{*,c} > L^*$ and $g^c = g = 0$. The saving rates in both economies are given as $\beta(1 - \frac{\rho-b+d}{\rho+\delta})$, of which $\frac{q}{1+q}$ fraction is invested for controlling infectious diseases. Thus, both economies share the same saving rate, of which centralized economy spends more in health expenditure than the decentralized ones. The prevalence of infectious diseases is less severe in the centralized economy. However, the effectiveness of human capital accumulation is still not large enough for justifying its time allocation, and hence there is no economic growth. In this case, the welfare comparison between two economies is ambiguous. The output and consumption in both economies are given as:[27]

$$Y^{*,j} = A^{\frac{1}{1-\beta}} \left(\frac{\rho+\delta}{\beta}(1+q^{*,j}) \right)^{-\frac{\beta}{1-\beta}} L(q^{*,j}), \quad \text{and} \quad C^{*,j} = \left(1 - \beta \frac{\delta+b-d}{\rho+\delta} \right) Y^{*,j},$$

depending on $q^{*,j}$, where $j = c$ for the centralized economy and $j = nil.$ for the decentralized economy. Even though labor force participation rate is higher in the centralized economy, which increases the production, the investment in physical capital is less compared with to decentralized ones, which leads to lower production. Thus, the overall effect on the total production and consumption is ambiguous. The calibration and simulation exercise (in Section 5) suggests that output, consumption and hence welfare are in fact higher in the centralized economy.

[27]Since human capital is indeterminate in the case of poverty trap, we assume it is given by its initial level normalized to 1, that is, $e_0 = 1$.

Figure 3. Determination of the Equilibrium Effective Health Capital: The Comparison Between the Competitive Equilibria and Optimal Paths



Note: The figure describes three different scenarios for the comparison between the decentralized and centralized economies. In all the panels, the solid line is the function $G(q)$, determining the effective health capital q^* in the decentralized economy, and the dashed line is the function $G(q) + b + \gamma$, determining the effective health capital $q^{*,c}$ in the centralized economy. The critical value $\hat{q}$ for the positive growth is the same in both economies. In the upper panel, we have $q^{*,c} > q^* > \hat{q}$ and both the decentralized and centralized economies grow at a positive rate; in the bottom left panel, we have $q^{*,c} > \hat{q} > q^*$ and the centralized economies grow at a positive rate, while the decentralized economy is in a poverty trap; in the bottom right panel, we have $\hat{q} > q^{*,c} > q^*$ and both the centralized and decentralized economies are stuck in a poverty trap.

5.2 Optimal Health Subsidy

Compared with the decentralized economy, the centralized economy, taking into account the positive externatlity of controlling infectious diseases, either has a higher growth rate, or is more likely to take off, or has a higher consumption level even in a poverty trap. This provides a justification for introducing effective public health policy. One of the issue with infectious diseases is that households do not account for the effect of their actions on the transmission of the disease. The evidence indicates that households seem to underinvest in preventive health care (e.g. Banerjee and Duflo (2011) who discuss preventive health care in general and Tarozzi, et al. (2009) who focus on use of insectiside-treated

bednets for prevention of malaria). What is the nature of the subsidy that will induce households to internalize preventive health expenditures? This is especially important as countries that are most afflicted with infectious diseases have weak public health delivery mechanisms. While external aid is often discussed in the context of controlling diseases, whether it is actually delivered for the specific need is an open question. There are, of course international health organizations (e.g. WHO) and NGOs (e.g. Carter Foundation that has worked for eradication of Guinea Worm in sub-Saharan Africa, Gates Foundation, and the earlier Rockefeller Foundation that played a key role of eradication of hookworm in southern U.S.A. (Bleakley (2007)). However, a market solution via balanced (self-financing) public health policy is more sustainable. Here we focus on health subsidies. In fact, any policy distorting marginal benefit of physical capital investment and health expenditure can be equally effective in obtaining the optimal path under the centralized economy, for instance, proportionate capital income tax, educational subsidy, etc. However, these are harder to motivate as the effect appears indirect. Also note that the disease free steady state is locally unstable and thus, external aid that does not change the inherent disease dynamics (untied or lump-sum aid) may help control diseases the outcome is not stable. If the aid is targeted to change the relative cost of health expenditure (i.e. act like a subsidy) then the following exercise also applies except it need not be self-financing.

We assume for each unit of private health investment, there is a proportional health subsidy τ, and the law of motion for health capital now is:

$$\dot{H} = (1+\tau)I_H - \delta H - (b-d)H. \tag{21}$$

The public health expenditure is financed through a lump-sum tax T, and the budget constraint is:

$$C + I_K + I_H = RK + WeuL - T. \tag{22}$$

Households maximize equation (4) by choosing consumption C, health expenditure I_H, physical capital investment I_K and time allocation u, subject to the constraints equation (2), (5), (7), (21) and (22). In equilibrium, the period-by-period balance budget (balancedness) implies $T = \tau I_H$.[28] The rest is the same as the competitive equilibrium, defined in Section 3.

We solve the maximization problem and the first order conditions are similar to equations (11)- (16). The only difference is the equation (11) with positive health expenditure which is now given as:

$$\lambda_1 = (1+\tau)\lambda_2.$$

We see that because there is the additional τ unit health subsidy for each unit of private health expenditure, marginal value of physical capital investment (the L.H.S. of the above equation) equals to $(1+\tau)$ times marginal value of private health expenditure (the R.H.S. of the above equation).

[28] An external aid subsidy would be $\tau I_H = \Xi$, where Ξ is the external aid budget and $T = 0$.

The following proposition gives the optimal subsidy, in the sense that it is chosen such that the allocations in the decentralized economy with public health subsidy coincide with the optimal path in the centralized economy.

Proposition 5. *Let $q^{*,c}$ be the optimal effective health capital in the centralized economy, defined in Proposition 4*

1. *When the optimal path is a disease-endemic BGP with $g = \psi L(q^{*,c}) - (\rho - b + d)$, the optimal health subsidy is:*

$$\tau = (1 + q^{*,c}) \frac{b + \gamma}{\alpha(q^{*,c}) - (b + \gamma) + \rho - b + d} \cdot \frac{\rho + \delta + g}{\rho + \delta - (1 + q^{*,c}) \frac{b+\gamma}{\alpha(q^{*,c}) - (b+\gamma) + \rho - b + d} g};$$

2. *When the optimal path is a disease-endemic poverty trap, the optimal health subsidy is:*

$$\tau = (1 + q^{*,c}) \frac{b + \gamma}{\alpha(q^{*,c}) - (b + \gamma) + \rho - b + d}.$$

Proof. See the Appendix. □

We can see that the optimal subsidy is proportional to the externality $b + \gamma$, and the larger the externality the bigger the subsidy. Moreover, the optimal subsidy is higher when the optimal path is a BGP than poverty trap. There can be the perverse case where if an economy is in a poverty trap in the centralized solution due to high incidence of diseases, the optimal subsidy will be smaller than in an economy where the disease incidence is lower and which is growing. Thus, countries that are most afflicted by diseases may have the least incentive to control them. The reason is that there is additional distortion resulting from positive growth rate, and hence the larger the public health subsidy.

6 Calibrations and Simulations

The marriage of the economic and epidemiological models provides us a framework in understanding the close link between the poverty and diseases. In this section, we calibrate the model for the LDCs and examine the impact of increasing effectiveness of controlling diseases and rising life expectancy. There has been considerable improvement in health conditions in the LDCs. This has mostly been attributed to the foreign aid, direct investment and the spillover effects from the technological progress and medical improvement in the developed world, all of which can be taken as exogenous from the perspective of the poor countries. However, the health conditions in Africa today still considerably lag behind the world average, and it is interesting to know whether the LDCs can escape from the vicious cycle of diseases and poverty as a a result of these changes. Note that the analysis here focuses on the evolution of the growth paths before and after the change, and the transitional dynamics

are ignored due to the complicated dynamical system.[29]

6.1 Calibrations

Both the model and empirical evidence show that the various growth paths countries undertake are closely related to the prevalence of infectious diseases, which in turn depends on all the fundamental economic, demographic and epidemiological parameters in the model. So different sets of model parameters can be calibrated by targeting countries in different stages of growth paths. As we are interested in the close link between diseases and poverty and how countries can escape this vicious cycle, the calibration and simulation exercise are aimed for the LDCs, mainly those in the Sub-Saharan African region.

The following parameters are chosen in line with the literature: discount rate $\rho = 0.055$, capital share $\beta = 0.36$, depreciation rate $\delta = 0.05$, and the scale parameter in the production function A is normalized to 1. Some economists believe that the capital share is typically higher in the LDCs, while others (e.g. Gollin(2002)) show that the share is generally the same with the developed countries, taking into account of self-employing sectors where income, accruing whether to labor or to capital, is in practice treated as capital income in previous inquiries. So we set capital share of the LDCs to be the relative upper bound of the estimates for the developed countries. Compared with the developed countries, both fertility rate and death rate are much higher in the Sub-Saharan Africa. Using the statistics from the World Health Organization (See WHO(2013)), we set the birth rate $b = 3.5\%$ and death rate $d = 1.85\%$, which implies 55 years of life expectancy. We could not calibrate the effectiveness of human capital accumulation ψ directly for the LDCs, as the model equilibrium does not depend on it due to the poverty trap. Nevertheless, in the developed countries, the effectiveness of human capital accumulation is calibrated to be 0.05 (Lucas (1988)). In the LDCs, human capital formation has received increasing attention for the past few decades. Increased spending on education from both the national budgets and foreign aid has led to educational progress, which has allowed them to narrow the gap with the rest of the world, particularly in the primary education. However, they are still far behind in terms of the quality of the education and schooling. Thus, we assume that the effectiveness in the LDCs is roughly eighty percent of the one in the developed countries, and we set $\psi = 0.04$.

In calibrating the disease related parameters, some papers (e.g. Chakraborty et al. (2010)) take a more micro approach, that is, calibrating epidemiological parameters to transmission of a single infectious disease. Nevertheless, we take a macro approach by targeting the key macroeconomic variables, as we do not think it is convincing to argue that any single infectious disease can be accountable for the underdevelopment in the Sub-Saharan Africa. The calibration requires a specific functional form for contact rate. As far as we know, there are no papers on estimating what this function is likely to be. So we choose the functional form of contact rate $\alpha(q) = q^{-0.5}/a$, where a is the effectiveness of controlling disease. It assumes contact rate is decreasing in effective health capital, and marginal

[29]The dynamical system is of eight dimensional system, which is too complicated to study the transitional dynamics. So we only focus on comparative statics here. Goenka and Liu (2012) are able to characterize the full global dynamics as there is only a one-way interaction which simplifies the dynamics. Goenka, Liu and Nguyen (2014) have a full analytic characterization of local dynamics in the neo-classical version of the model, and show that the disease endemic steady state is saddle-point stable under reasonable assumptions.

benefit of controlling diseases decreases as effective health capital increases. This leaves us with two disease-related model parameters: the effectiveness of controlling disease, a and the recovery rate, γ. They are calibrated to match two key disease-related macro variables. One is the years loss due to infectious diseases. The average DALY of the LDCs shown in Table 1 suggests that 38.02% of time is lost due to infectious diseases. Note that this number represents the loss from both mortality and morbidity from infectious diseases. Moreover, the statistics provided by the WHO including a wide array of countries indicate that roughly one-third of DALY is due to morbidity. This suggests in the LDCs, around 14% of time is lost due to morbidity from infectious diseases. The other macro variable used for calibration is the health expenditure as a share of GDP in controlling infectious diseases. There is an extensive literature on health expenditure in the developed countries. However, evidence from developing countries is relatively scarce. Health care expenditure in the LDCs varies over time and across countries. On average, low-income countries spend around 3% of GDP on health, though increasing over years. This consists of government health expenditure, private out-of-pocket health expenditure and external aids. It is estimated that external aids is around $10-20\%$ of total health expenditure, and government funding takes up the half of the rest of health expenditure (See Gottret and Schieber (2006), Xu, et al. (2011) and etc.). So we target the health expenditure ratio in a decentralized economy to be around 1.5% of GDP. This is likely to be the upper bound of private health expenditure ratio in the LDCs.

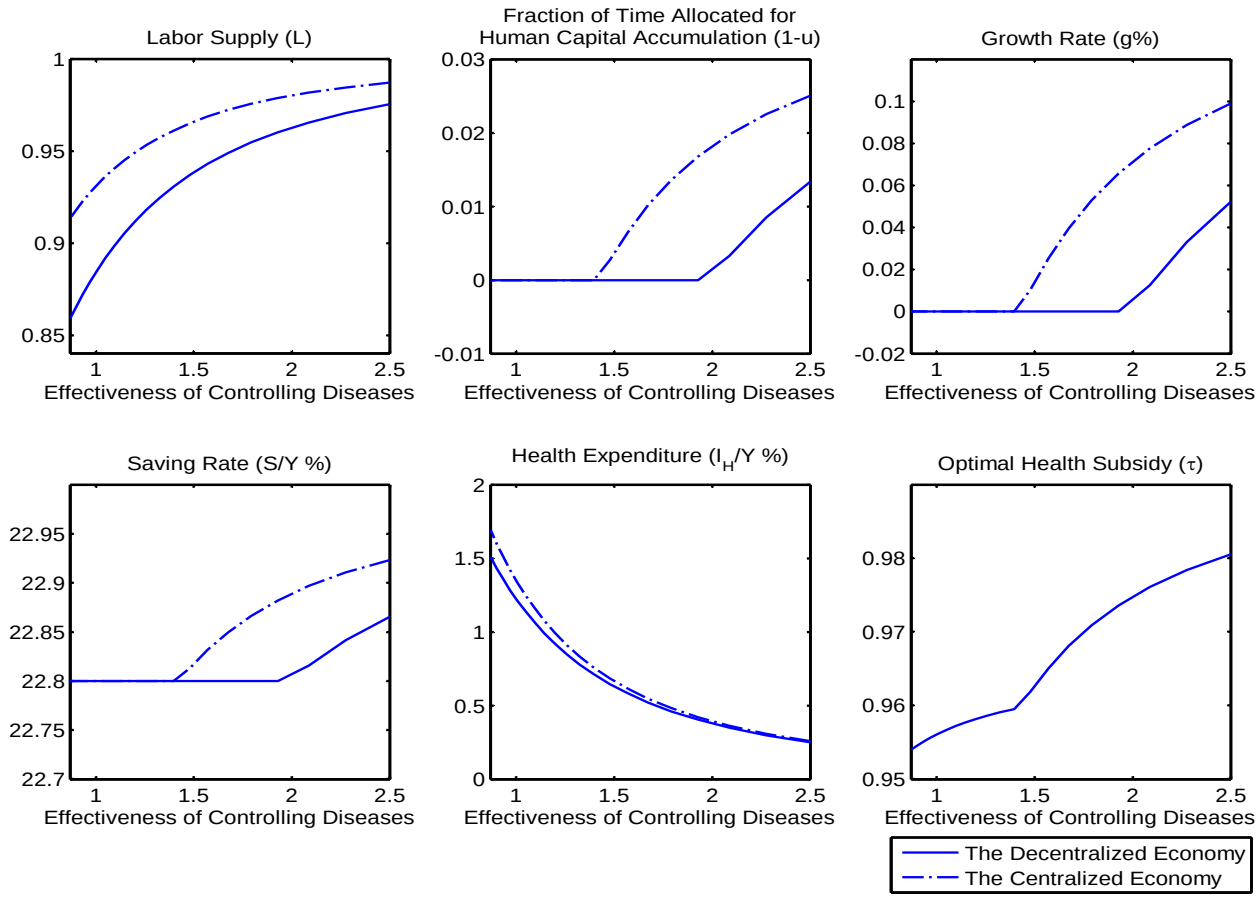
6.2 Impact of Increasing Effectiveness of Controlling Diseases

In this subsection, we examine the impact of increasing effectiveness of controlling diseases in the LDCs by the parameter – a. Figure 4 depicts the evolution of economic variables when a increases. The solid line presents the change for the decentralized economy, while the dashed line shows the change for the centralized economy. When a is at its initial level of 0.85, the proportion of health individuals or the effective labor supply is 86%, and the health expenditure as a share of GDP is 1.5%. These are directly the result of calibration. In addition to the low level of physical capital, indicating high marginal return to production, the prevalence of infectious diseases significantly reduces the marginal return to human capital accumulation. This implies all the time is allocated to production, instead of human capital accumulation. The saving rate is around 22.8%, lying in the reasonable range of saving rates in the LDCs, 10% to 25%. The large portion of saving is used for investment of physical capital, rather than health capital. Thus, with lower effectiveness of controlling diseases, countries are in a poverty trap. In the decentralized case, when a increases from 0.85 to 2, a smaller portion of the saving is actually spent on health expenditure, but the effective labor supply increases substantially. The marginal benefit of human capital accumulation is still not large enough for justifying its time allocation, and hence there remains no economic growth. Nevertheless, all these start changing when a increases further above the critical value 2. Countries start investing in human capital accumulation and transit from a poverty trap to an equilibrium with positive economic growth. Therefore, with increasing effectiveness of controlling diseases, the LDCs can eventually escape the poverty trap.

When we compare the centralized economy with the decentralized ones, the interesting

observations are the following. The first is that the centralized economy or the introduction of public health policy does not necessarily guarantee economic growth. It can be seen that the centralized economy is also in a poverty trap when a is below 1.48. Secondly, when effectiveness of controlling diseases increases, the centralized economy starts taking off way before the decentralized economy does. Thirdly, for the resource allocation, when both economies are in the poverty trap, the saving rates are in fact the same, though more is spent for health expenditure in the centralized economy. This is because of the fact that social planner takes into account the positive externality of controlling disease. Lastly, the optimal health subsidy is convex in shape. The reason is that the positive externality from controlling diseases becomes increasingly larger as a increases, due to the additional distortion from the growth.

Figure 4. The Evolution of Growth Paths Due to Increasing Effectiveness of Controlling Diseases



Note: The figure depicts the evolution of economic variables when the effectiveness of controlling diseases increases. The solid line presents the change for the decentralized economy, while the dashed line shows the change for the centralized economy. The variables included are labor supply (L), fraction of time allocated for human capital accumulation ($1-u$), growth rate (g), saving rate (S/Y), health expenditure ratio (I_H/Y) and optimal public health subsidy (τ).

6.3 Impact of Rising Life Expectancy

In this subsection, we examine the effects of rising life expectancy, In the paper, we emphasize the interaction between diseases transmission and human capital investment, instead of the interaction between disease transmission and demographics so as to focus on the role of morbidity.[30] The examination of effect of increase in life expectancy gives us a glimpse of how the demographic transition affects the disease control, and hence human capital investment and economic growth.

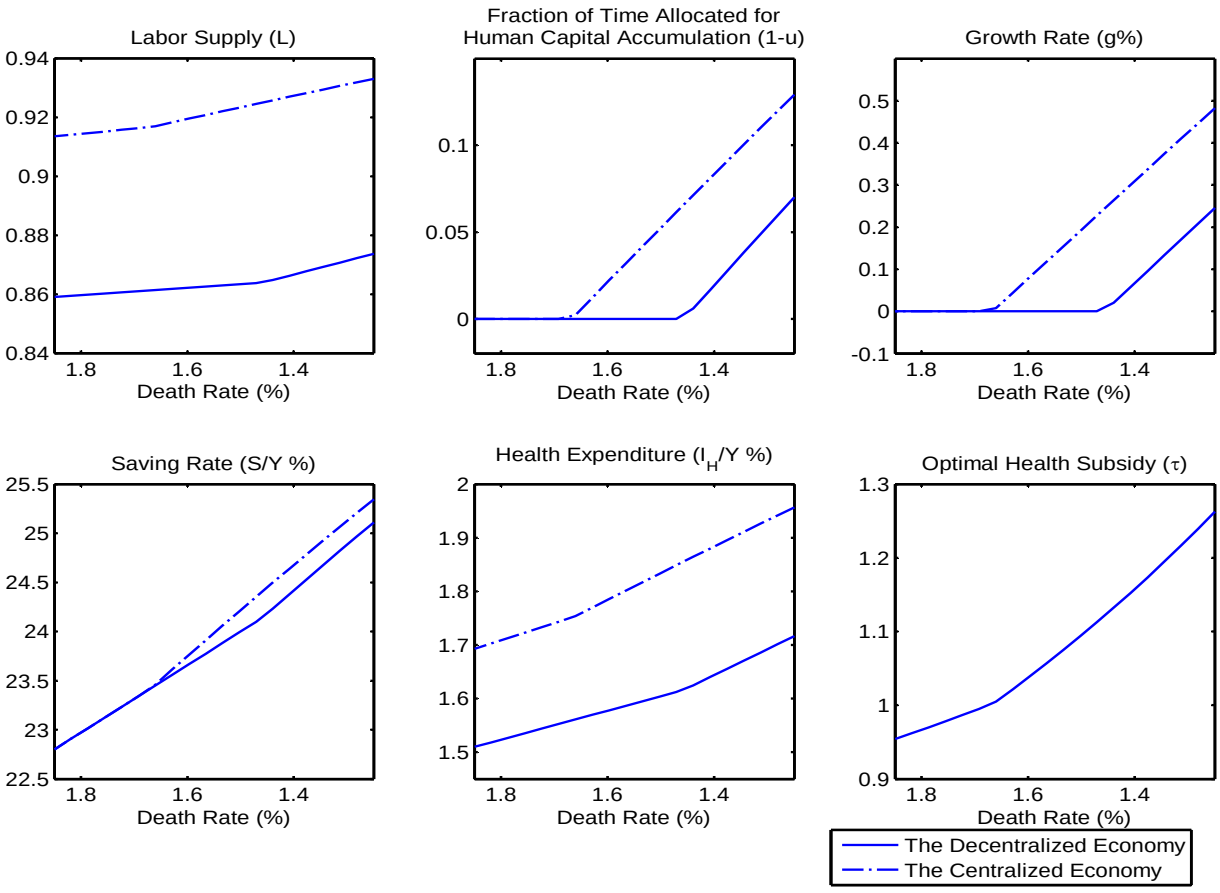
Figure 5 depicts the evolution of economic variables when mortality rate drops from 1.85% to 1.25%, that is, life expectancy increases from 55 years to 80 years. In the decentralized case, when the mortality rate decreases from 1.85% to 1.48% or life expectancy increases from 55 to 67 years, households become more patient and save more. However, only a small portion of the increased saving is spent on health expenditure. This can be seen from the fact that saving rate increases by 1.2%, while health expenditure ratio increases only by 0.1%. As a result, the effective labor supply remains relatively the same with an indiscernible increase. The marginal benefit of human capital accumulation is still not large enough for justifying its time allocation, and hence there remains no economic growth. Nevertheless, all these start changing when mortality rate drops further below the critical value 1.48% or life expectancy increases above 67 years. Households save even more. Moreover, the increment in saving rate as a result of rising life expectancy increases, which is shown by the increase of slope for the saving curve. Similarly, health expenditure ratio increases as well, which leads the effective labor supply to rise. There is also the relative change of the slope above and below the critical mortality rate, for both the health expenditure ratio and the effective labor supply in Figure 5. Countries start investing in human capital accumulation and transit from a poverty trap to an equilibrium with positive economic growth. Therefore, with a prospect life expectancy increasing to 68.5 years by 2050, the LDCs can eventually escape the poverty trap.

Similar to the previous sub-section, when we compare the centralized economy with the decentralized ones, we find that the centralized economy does not necessarily guarantee economic growth; when mortality rate declines, the centralized economy starts taking off way before the decentralized economy does; when both economies are in the poverty trap, the saving rates are in fact the same, though more is spent for health expenditure in the centralized economy; and the optimal health subsidy is convex in shape. The reason is that the positive externality from controlling diseases becomes increasingly larger as life expectancy increases, due to the additional distortion from the growth. Thus, the fact that poor countries in a poverty trap are short of public health expenditure is not only because they have tighter budget constraints, but more importantly they lack incentives for investing in health capital.

As life expectancy increases, more health expenditure is allocated for controlling infectious diseases and labor force participation rate rises, which increase output and consumption level. However, on the other hand, as the result of direct effect of declining death rate, the

[30] See Chakraborty, et al. (2010) for example. These papers examine the effect of infectious diseases on the economy by taking mortality as endogenous, but with simplified disease dynamics. Soares (2005) also looks at effect of exogenous change of longevity on human capital but the mechanism is via endogenous fertility rather than through health expenditures and control of diseases.

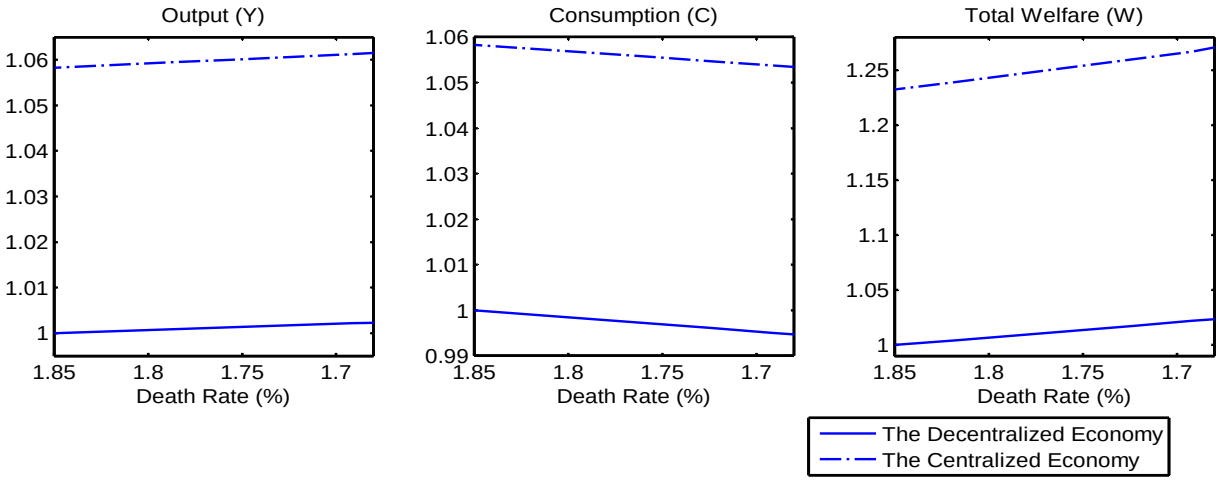
Figure 5. The Evolution of Growth Paths Due to Rising Life Expectancy



Note: The figure depicts the evolution of economic variables when mortality rate drops from the initial level of 1.8% to 0.8%. The solid line presents the change for the decentralized economy, while the dashed line shows the change for the centralized economy. The variables included are labor supply (L), fraction of time allocated for human capital accumulation ($1-u$), growth rate (g), saving rate (S/Y), health expenditure ratio (I_H/Y) and optimal public health subsidy (τ).

consumption level decreases. The reason is that more people alive diffuse the resource allocation and lower consumption level for each individual. This is so called Malthusian effect. It is not clear in the model which effect dominates. One thing to note is that the Malthusian effect can take place only when a country is in a poverty trap. For the other situations, a decrease in the death rate or increase in life expectancy unambiguously increases growth through the mechanism of increased incentives for saving due to the decrease in the effective discount rate. Figure 6 depicts the change of output, consumption and welfare when mortality rate drops from the initial level of 1.85% to 1.68%. That is, both centralized and decentralized economies are in a poverty trap. The solid line presents the change for the decentralized economy, while the dashed line shows the change for the centralized economy. The welfare is calculated as the discounted stream of utility in the steady state. In each panel, the value is normalized such that it is 1 for the initial level of mortality rate 1.85% in the decentralized economy. Compared with the decentralized economy, output, consumption

Figure 6. The Change of Welfare Due to Rising Life Expectancy



Note: The figure depicts the change of welfare when mortality rate drops from the initial level of 1.8% to 1.65%. That is, both centralized and decentralized economies are in a poverty trap. The solid line presents the change for the decentralized economy, while the dashed line shows the change for the centralized economy. The variables included are output (Y), consumption (C) and welfare (W). In each panel, the value is normalized such that it is 1 for the initial level of mortality rate 1.8% in the decentralized economy.

and welfare are much higher in the centralized economy, though both are in a poverty trap. Furthermore, as life expectancy rises, output increases slightly. However, due to Malthusian effect, consumption level declines. But, welfare increases as people now live longer.

7 Conclusions

This paper develops an endogenous growth model with human capital formation where the prevalence of an infectious disease causes ill-health and incapicitates individuals from working as well as accumulating human capital. There is an endogenous choice of health expenditure to prevent infectious diseases. The paper focuses on the effects of morbidity (ill-health) and thus chooses to use an infinitely lived agent framework. There are multiple balanced growth paths where the endogenous prevalence of the disease determines whether human capital is accumulated or not, i.e. whether there is sustained economic growth or a poverty trap. This mirrors the cross-country empirical evidence. The paper also shows that an exogenous demographic transition could lead to a take-off from poverty trap to a positive growth. It shows that beyond the mortality effects of diseases such as HIV/AIDS and malaria, the so-called "forgotten disease" that are endemic, do not cause significant mortality, and afflict primarily the poor, could be an important determinant of poverty traps by affecting the amortization of physical, human and health capital. This affects the the size and allocation of savings amongst the different types of capital which will be missed in models that treat as exogenous.

References

[1] Acemoglu, D. and Johnson, S. (2007): "Disease and development: The effect of life expectancy on economic growth," *Journal of Political Economy*, 115: 925-985.

[2] Ashraf, Q.H., Lester, A., and Weil, D.N. (2009): "When does improving health raise GDP?" D. Acemoglu, K. Rogoff, and M. Woodford (Eds.) *NBER Macroeconomics Annual 2008*, Chicago: The University of Chicago Press.

[3] Azomahou, T.T., Bouccekkine, R., and Diene, B. (2016) "HIV/AIDS and development: A reappraisal of the productivity and factor accumulation effects," *American Economic Review: Papers and Proceedings*, 106(5): 472-477.

[4] Banerjee, A., and Duflo, E. (2011) *Poor Economics*, Philadelphia: Perseus Books Group.

[5] Barro, Robert and Jong-Wha Lee, 2013, "A New Data Set of Educational Attainment in the World, 1950-2010." Journal of Development Economics, Vol 104, pp.184-198.

[6] Bleakley, H. (2007): "Disease and development: Evidence from hookworm eradication in the American South," *Quarterly Journal of Economics*, 122: 73-117.

[7] Bleakley, H. (2010): "Malaria eradication in the Americas: A retrospective analysis of childhood exposure," *American Economic Journal: Applied Economics* 2: 1-45.

[8] Bleakley, H. and Lange, F. (2009) "Chronic disease burden and the interaction of education, fertility and growth," *Review of Economic Statistics* 91: 52-65.

[9] Bloom, D.E., Canning, D. and Fink, G. (2014) Diseases and development revisited, *Journal of Political Economy*, 122: 1355-1366.

[10] Bonds, M.H., Keenan, D.C., Rohani, P., and Sachs, J.D. (2009) "Poverty trap formed by the ecology of infectious diseases," *Proceedings of the Royal Society B*, doi:10.1098/rspb.2009.1778

[11] Chakrabory, S. (2004) "Endogenous lifetime and economic growth" *Journal of Economic Theory*, 116: 119-137.

[12] Chakrabory, S., Papageorgiou, C., and Perez-Sebastian, F., (2010) "Diseases, infection dynamics and development" *Journal of Monetary Economics*, 859-872.

[13] Chakrabory, S., Papageorgiou, C., and Perez-Sebastian, F., (2014) "Health cyles and health transitions" *Macroeconomic Dynamics*, Forthcoming.

[14] Chauhan, A., and Johnston, S. L (2003). "Air pollution and infection in respiratory illness," *British Medical Bulletin*, 68(1): 95-112.

[15] Cuddington, J.T. and Hancock, J.D. (1994) "Assessing the Impact of AIDS on the growth path of the Malawian economy, " *Journal of Development Economics* 43 (2): 363-68.

[16] Cutler, D., Fung, W., Kremer, M., Singhal, M. and Vogl, T. (2010) "Early-life malaria exposure and adult outcomes: Evidence from malaria eradication in India," *American Economic Journal: Applied Economics* 2: 72-94.

[17] d'Albis, H., Gourdel, P. and Le Van, C. (2008): "Existence of solutions in continuous-time optimal growth models," *Economic Theory*, 37(2): 321-333.

[18] D'Albis, H., and Le Van, C. (2006) On the existence of a competitive equilibrium in the Lucas (1988) model, *Journal of Mathematical Economics*, 42: 46-55, 2006.

[19] Delfino, D. and P.J. Simmons (2000): "Positive and normative issues of economic growth with infectious diseases," Discussion Papers in Economics 2000/48, University of York.

[20] Epstein, P.R. (2001), " Climate change and emerging infectious diseases," *Microbes and Infection*, 3: 747-754.

[21] Fortson, J.G. (2009): "HIV/AIDS and fertility," *American Economic Journal: Applied Economics*, 170-194.

[22] Fortson, J.G. (2011): "Mortality risk and human capital investment: The impact of HIV/AIDS in Sub-Saharan Africa, *Review of Economics and Statistics* 93: 1-15.

[23] Fox, M.P., Rosen, S., MacLeod, W.B., Wasunna, M., Bii, M., Foglia, G., Simon, J.L. (2004) "The impact of HIV/AIDS on labor productivity in Kenya," *Tropical Medicine and International Health*, 9: 318-324.

[24] Geoffard, P-Y. and Philipson, T. (1996): "Rational Epidemics and Their Public Control," *International Economic Review*, 37(3): 603-24.

[25] Gersovitz, M. and Hammer, J.S., (2004): "The economical control of infectious diseases", *The Economic Journal*, 1-27.

[26] Goenka, A., and Liu, L. (2012): "Infectious diseases and endogenous fluctuations," *Economic Theory*, 50(1), 125-149.

[27] Goenka, A., Liu, L., and Nguyen, M-H. (2014): "Infectious diseases and economic growth," *Journal of Mathematical Economics*, 50: 34-53.

[28] Gokhale, K. (2013) "Leprosy return shows neglect in India of ancient blight," Bloomberg News (18/09/2013) (http://www.bloomberg.com/news/2013-09-17/leprosy-return-shows-neglect-in-india-of-ancient-blight.html)

[29] Gollin, D. (2002) "Getting Income Shares Right," *Journal of Political Economy*, 110: 458-474.

[30] Goossens, H., Ferech, M., Van der Stichele, R., Elseviers, M., (2004) "Outpatient antibiotic use in Europe and association with resistance: a cross-national database study ," *Lancet*, 365, Issue 9459, 12-18 February, 579-579.

[31] Gottret, P., and Schieber, G. (2006) "Health Financing Revisited: A Practioner's Guide", The World Bank.

[32] Goyal, S. and Vigier, A. (2015) "Infection, protection, and epidemics," *Journal of Public Economics*, forthcoming.

[33] Hemingway, J., and Ranson, H. (2000) "Insecticide resistance in insect vectors of human disease, *Annual Review of Entomology*, 45: 371-391.

[34] Hethcote, H.W. (1994): "A thousand and one epidemic models, "In S.A. Levin (Ed.) *Frontiers in Theoretical Biology*, Vol. 100 of Lecture Notes in Biomathematics: 504-515, Berlin: Springer-Verlag.

[35] Hethcote, H.W. (2005): "The basic epidemiology models, Epidemiology models with variable population size, and Age-structured epidemiology models, "IMS Lecture note series, National University of Singapore.

[36] Kalemli-Ozcan, S., Ryder, H., and Weil, D.N. (2000): "Mortality decline, human capital investment, and economic growth, *Journal of Development Economics*," 62: 1-23.

[37] Kalemli-Ozcan and Turan, B. (2011): "HIV and fertility revisited," *Journal of Development Economics* 96: 61-65.

[38] Kremer, M. (1996): "Integrating behavioral choice into epidemiological models of the AIDS epidemic," *Quarterly Journal of Economics*, 111: 549-573.

[39] Levy, S.B., and Marshall, B. (2004) "Antibacterial resistance worldwide: causes, challenges and responses," *Nature Medicince*, 10, S122 - S129.

[40] Lucas, A. (2010) "Malaria eradication and educational attainment: Evidence from Paraguay and Sri Lanka," *American Economic Journal: Applied Economics* 2: 46-71.

[41] Lucas, Robert E. (1988): "On the mechanics of economic development," *Journal of Monetary Economics* 22: 3-42.

[42] McMichael, A.J. (2003) "Environmental and social influences on emerging infectious diseases: past, present and future," *Philosophical Proceedings of the Royal Society B: Biological Sciences*, DOI: 10.1098/rstb.2004.1480.

[43] McMichael, A.J., Woodruff, R.E., and Hales, S. (2006) "Climate change and human health: present and future risks," *Lancet* 367: 859-69.

[44] Miguel, E. and Kremer, M. (2004): "Worms: Identifying Impacts on Education and Health in the Presence of Treatment Externalities," *Econometrica*, 72: 159-217.

[45] Murray, C.J.L., et al. (2012): "Disability-adjusted life years (DALYs) for 291 diseases and injuries in 21 regions, 1990-2010: A systematic analysis of Global Burden of Disease Study 2010," *The Lancet* 380: 2197-2223.

[46] Patz, J. A., Githeko, A. K., McCarty, J. P., Hussein, S., Confalonieri, Ulisses E. C., Wet, N. de. (2003) "Climate change and infectious diseases," in McMichael, A. J., Campbell-Lendrum, Diarmid., Ebi, K. L., CorvalÃ¡n, Carlos., Githeko, A. K., Woodward, A., Scheraga, J. D. *Climate change and human health: risks and responses*, Geneva, OMS, p.103-132.

[47] Pavlovsky, E.N. (1966): *Natural Nidality of Transmissible Diseases*, edited by N.D. Levine, translated by F.K. Plous. Urbana and London: University of Illinois Press.

[48] Salomon, J.A. et al (2012) "Common values in assessing health outcomes from disease and injury: disability weights measurement study for the Global Burden of Disease Study 2010," *The Lancet* 380: 2129-2143.

[49] Soares, R. (2005): "Mortality Reductions, Educational Attainment, and Fertility Choice," *American Economic Review*, 95(3): 580-601.

[50] Steinmann, P., Keiser, J., Bos, R., Tanner, M., and Utzinger, J. (2006) "Schistosomiasis and water resources development: systematic review, meta-analysis, and estimates of people at risk," *Lancet*, 6(7): 411-425.

[51] Susser, M, and Stein, Z. (2009) *Eras in epidemiology: The evolution of ideas*, Oxford: Oxford University Press.

[52] Tarozzi, A., Mahajan, A., Yoong, J., and Blackburn, B. (2009) "Commitment mechanisms and compliance with health-protecting behavior: Preliminary evidence from Orissa, India," *American Economic Review: Papers and Proceedings* 99(2): 231-235.

[53] United Nations Population Fund (UNPFA) (2012): *Population Dynamics in the Least Developed Countries: Challenges and Opportunities for Development and Poverty Reduction.*

[54] Weil, D. (2007): "Accounting for the effect of health on economic growth," *Quarterly Journal of Economics*, 122: 1265-1308.

[55] World Health Organization (2013): *World Health Statistics*, available on the WHO web site (www.who.int).

[56] Xu, K., Saksena, P. and Holly, A. (2011): "The Determinants of Health Expenditure: A Country-Level Panel Data Analysis" *World Health Organization* working paper.

[57] Young, A. (2005): "The Gift of the Dying: The Tragedy of AIDS and the Welfare of Future African Generations." *Quarterly Journal of Economics* 120: 243-266.

A Appendix

A.1 Proof of Proposition 1: Existence of the BGPs

The competitive equilibrium is described by equation (2) - (3), (8) - (10) and (11) - (16). In addition, the following TVCs: $\lim_{t\to\infty} e^{-(\rho-b+d)t}\lambda_1 K = 0$, $\lim_{t\to\infty} e^{-(\rho-b+d)t}\lambda_2 H = 0$, $\lim_{t\to\infty} e^{-(\rho-b+d)t}\lambda_3 e = 0$, and $\lim_{t\to\infty} e^{-(\rho-b+d)t}\lambda_4 L = 0$ have to be satisfied in the equilibrium.

We rewrite the dynamical system of the decentralized economy as follows:

$$C + [\dot{K} + (\delta + b - d)K] + [\dot{H} + (\delta + b - d)H] = AK^{\beta}(euL)^{1-\beta} \tag{A.1}$$

$$\dot{e} = \psi e L(1-u) \tag{A.2}$$

$$\dot{L} = (b+\gamma)(1-L) - \alpha\left(\frac{H}{K}\right)(1-L)L \tag{A.3}$$

$$\frac{1}{C} = \lambda_1 \tag{A.4}$$

$$\lambda_1 = \lambda_2 + \theta_3, \quad \theta_3 \geq 0, \quad I_H \geq 0, \quad \theta_3 I_H = 0 \tag{A.5}$$

$$\lambda_1(1-\beta)AK^{\beta}(euL)^{-\beta}eL = \lambda_3\psi eL + \theta_1, \quad \theta_1 \geq 0, \quad 1-u \geq 0, \quad \theta_1(1-u) = 0 \tag{A.6}$$

$$\dot{\lambda}_1 = (\rho - b + d)\lambda_1 - \lambda_1(\beta AK^{\beta-1}(euL)^{1-\beta} - (\delta + b - d)) - \lambda_4\alpha'\left(\frac{H}{K}\right)\frac{H}{K^2}(1-L)L \tag{A.7}$$

$$\dot{\lambda}_2 = (\rho - b + d)\lambda_2 + \lambda_2(\delta + b - d) + \lambda_4\alpha'\left(\frac{H}{K}\right)\frac{1}{K}(1-L)L \tag{A.8}$$

$$\dot{\lambda}_3 = (\rho - b + d)\lambda_3 - \lambda_3\psi L(1-u) - \lambda_1(1-\beta)AK^{\beta}(euL)^{-\beta}uL \tag{A.9}$$

$$\begin{aligned}\dot{\lambda}_4 = {} & (\rho - b + d)\lambda_4 - \lambda_3\psi e(1-u) + \lambda_4\left(b + \gamma + \alpha\left(\frac{H}{K}\right)(1-L)\right) \\ & -\lambda_1(1-\beta)AK^{\beta}(euL)^{-\beta}eu + \theta_2, \quad \theta_2 \geq 0, \quad 1-L \geq 0, \quad \theta_2(1-L) = 0\end{aligned} \tag{A.10}$$

Disease-free case: In this case, infectious diseases are eradicated, all individuals are healthy, and health expenditure for disease control is zero, $I_H = 0$. Otherwise, if $I_H > 0$, we have $\theta_3 = 0$ and $\lambda_1 = \lambda_2$. Combining equations (A.7) and (A.8), we obtain $\lambda_1\beta AK^{\beta-1}(eu)^{1-\beta} = 0$, which contradicts $\lambda_1 = \frac{1}{C} > 0$.

Differentiating both sides of equation (A.4) and we get $-\frac{\dot{C}}{C} = \frac{\dot{\lambda}_1}{\lambda_1}$. Dividing both sides of equation (A.7) by λ_1, we get $\frac{\dot{\lambda}_1}{\lambda_1} = \rho + \delta - \beta AK^{\beta-1}(eu)^{1-\beta}$. Since u is a constant along BGP, it implies growth rates of human capital and physical capital are the same. Similarly, by dividing both sides of equation (A.1) by K, growth rates of physical capital and consumption are the same. So consumption, physical and human capital all grow at the same rate $g = \psi(1-u)$, given by equation (A.2). Dividing both sides of equation (A.9) by λ_3, we have $\frac{\dot{\lambda}_3}{\lambda_3} = \frac{\dot{\lambda}_1}{\lambda_1} = -g$.

If $u^* = 1$, $g = 0$ and $\dot{\lambda}_1 = \dot{\lambda}_3 = 0$. From equation (A.6), $\theta_1 \geq 0$ and $\lambda_1(1-\beta)AK^{\beta}e^{-\beta} > \lambda_3\psi$. From equation (A.9), $\lambda_1(1-\beta)AK^{\beta}e^{-\beta} = \lambda_3(\rho - b + d)$. So we have $\psi < \rho - b + d$, contradicting the assumption $\psi > \rho - b + d$. So, u^* is strictly less than one, $\theta_1 = 0$ and $\lambda_1(1-\beta)AK^{\beta}e^{-\beta} = \lambda_3\psi$. Substitute this into equation (A.9), and we get $g = \psi - (\rho - b + d)$ and $u^* = \frac{\rho - b + d}{\psi}$.

Disease-endemic case: In this case, infectious diseases are prevalent and $L(q) = \frac{b+\gamma}{\alpha(q)}$. Since L^* is a constant along BGP, q^* is also a constant, implying physical and health capital grow at the

same rate. Due to the Inada condition in Assumption 1, health expenditure is strictly positive. So in equation (A.5) $\theta_3 = 0$ and $\lambda_1 = \lambda_2$. Then, we could rewrite equations (A.7) - (A.10) as:

$$\frac{\dot{\lambda}_1}{\lambda_1} = \rho - b + d - [\beta A(\frac{euL}{K})^{1-\beta} - (\delta + b - d)] - \frac{\lambda_4}{\lambda_1}\alpha'(q)\frac{H}{K^2}(1-L)L \tag{A.11}$$

$$\frac{\dot{\lambda}_1}{\lambda_1} = \rho - b + d + (\delta + b - d) + \frac{\lambda_4}{\lambda_1}\alpha'(q)\frac{1}{K}(1-L)L \tag{A.12}$$

$$\frac{\dot{\lambda}_3}{\lambda_3} = \rho - b + d - \psi L(1-u) - \frac{\lambda_1}{\lambda_3}(1-\beta)A\left(\frac{euL}{K}\right)^{-\beta} uL \tag{A.13}$$

$$\frac{\dot{\lambda}_4}{\lambda_4} = \rho - b + d - \frac{\lambda_3}{\lambda_4}\psi e(1-u) + (b + \gamma + \alpha(q)(1-L)) - \frac{\lambda_1}{\lambda_4}(1-\beta)A\left(\frac{euL}{K}\right)^{-\beta} eu \tag{A.14}$$

By some manipulations, we can see that consumption, physical, health and human capital grow at the same rate $g = \psi L(q^*)(1-u^*)$, $\frac{\dot{\lambda}_1}{\lambda_1} = \frac{\dot{\lambda}_3}{\lambda_3} = -g$ and $\frac{\dot{\lambda}_4}{\lambda_4} = 0$. Substitute these into equations (A.11)-(A.13), we have

$$\frac{\lambda_1}{\lambda_3} = \frac{\rho - b + d}{(1-\beta)AK^{\beta}(euL)^{-\beta}uL}, \quad \text{and} \quad \frac{\lambda_4}{\lambda_1} = -\frac{g + \rho + \delta}{\alpha'(q)\frac{1}{K}(1-L)L}.$$

Then substitute these into equation (A.14) and we obtain:

$$-\frac{1-\beta}{\beta}\alpha'(q)(1-L(q))(1+q) - \alpha(q) - \frac{1-\beta}{\beta}\alpha'(q)(1-L(q))(1+q)\frac{\psi L(q)(1-u)}{\rho - b + d} = \rho - b + d, \tag{A.15}$$

which is a function of both q and u. Moreover, from equation (A.6), we have:

$$\begin{aligned}\theta_1 &= \lambda_1(1-\beta)A\left(\frac{euL}{K}\right)^{-\beta} eL - \lambda_3\psi eL \\ &= \lambda_3\frac{e}{u}[(\rho - b + d) - \psi uL] \\ &\geq 0\end{aligned}$$

Since $\frac{\lambda_1}{\lambda_3} > 0$ and $\lambda_1 > 0$, we have $\lambda_3 > 0$. So equation (A.6) reduces to

$$\rho - b + d - \psi uL \geq 0, \quad u \leq 1, \quad \text{and} \quad (\rho - b + d - \psi uL)(1-u) = 0. \tag{A.16}$$

Therefore, equations (A.15) and (A.16) determines (u^*, q^*).

There are two scenarios. If $u^* = 1$, growth rate $g = 0$. Equation (A.15) simplifies to:

$$G_L(q) = -\frac{1-\beta}{\beta}\alpha'(q)(1-L(q))(1+q) - \alpha(q) - (\rho - b + d) = 0.$$

Since $u^* = 1$, we have $\theta_1 \geq 0$, implying $\psi L(q^*) \leq \rho - b + d$. That is, a disease-endemic poverty trap exists if $q^* \leq \hat{q}$.

If $u^* < 1$, we have $\lambda_1(1-\beta)AK^{\beta}(eL)^{1-\beta}u^{-\beta} = \lambda_3\psi eL$ and $u^* = \frac{\rho-b+d}{\psi L^*}$. q^* is determined by:

$$G_R(q) = -\frac{1-\beta}{\beta}\alpha'(q)(1-L(q))(1+q)\frac{\psi L(q)}{\rho-b+d} - \alpha(q) - (\rho-b+d) = 0.$$

Since $u^* < 1$, we have $\psi L(q^*) > \rho - b + d$. That is, a disease-endemic BGP exists if $q^* > \hat{q}$.

If we compare the two functions $G_L(q)$ and $G_R(q)$, we find that $G_L(q) > G_R(q)$ if $q < \hat{q}$, $G_L(q) < G_R(q)$ if $q > \hat{q}$, and $G_L(q) = G_R(q)$ if $q = \hat{q}$. Thus q^* is determined by function

$$G(q) = \max\{G_L(q), G_R(q)\} = 0.$$

Furthermore, the function G is continuous, $\lim_{q\to 0} G = +\infty$ and $\lim_{q\to\infty} G < 0$. By intermediate value theorem, there exists a $q^* > 0$ such that $G(q) = 0$, that is, there exists an endemic-disease case. If $q^* \le \hat{q}$, it is a poverty trap, and if $q^* > \hat{q}$, it is a positive growth path.

A.2 Proof of Lemma 1: Uniqueness of q^*

Since the functions $G_L(q)$ and $G_R(q)$ are differentiable, we have

$$\begin{aligned}
\frac{\partial G_L(q)}{\partial q} &= -\frac{1-\beta}{\beta}\alpha'(q)(1-L(q))(1+q)\left[\frac{\alpha''(q)}{\alpha'(q)} + \frac{L(q)}{1-L(q)}\cdot\frac{\alpha'(q)}{\alpha(q)} + \frac{1}{1+q}\right] - \alpha'(q);\\
\frac{\partial G_R(q)}{\partial q} &= -\frac{1-\beta}{\beta}\alpha'(q)(1-L(q))(1+q)\frac{\psi L(q)}{\rho-b+d}\left[\frac{\alpha''(q)}{\alpha'(q)} + \frac{L(q)}{1-L(q)}\cdot\frac{\alpha'(q)}{\alpha(q)} + \frac{1}{1+q} - \frac{\alpha'(q)}{\alpha(q)}\right] - \alpha'(q).
\end{aligned}$$

We further assume $\alpha''(q)$ is big enough, that is:

$$\begin{aligned}
\alpha''(q) > -\alpha'(q)\ \max\{&\frac{\beta}{(1-\beta)(1-L(q))(1+q)} + \frac{L(q)}{1-L(q)}\cdot\frac{\alpha'(q)}{\alpha(q)} + \frac{1}{1+q},\\
&\frac{\beta}{(1-\beta)(1-L(q))(1+q)}\cdot\frac{\rho-b+d}{\psi L(q)} + \frac{L(q)}{1-L(q)}\cdot\frac{\alpha'(q)}{\alpha(q)} + \frac{1}{1+q} - \frac{\alpha'(q)}{\alpha(q)}\}.
\end{aligned}$$

Therefore, under Assumption 1, we can show that both functions $G_L(q)$ and $G_R(q)$ are monotonically decreasing in q. Moreover, since $G(q) = G_L(q)$ when $q < \hat{q}$, $G(q) = G_R(q)$ when $q > \hat{q}$, and $G(q) = G_L(q) = G_R(q)$ when $q = \hat{q}$, function $G(q)$ is also monotonically decreasing in q. Thus, there exists a unique q^* such that $G(q) = 0$.

A.3 Proof of Proposition 2: Comparative Statics

Since $\psi\frac{b+\gamma}{\alpha(\hat{q})} = \rho - b + d$, we have

$$\begin{aligned}
&\frac{\partial\hat{q}}{\partial\rho} = -\frac{\alpha(\hat{q})}{\alpha'(\hat{q})(\rho-b+d)} > 0; \quad \frac{\partial\hat{q}}{\partial d} = \frac{-\alpha(\hat{q})}{\alpha'(\hat{q})(\rho-b+d)} > 0;\\
&\frac{\partial\hat{q}}{\partial\psi} = \frac{b+\gamma}{\alpha'(\hat{q})(\rho-b+d)} < 0.
\end{aligned}$$

Define $\hat{G} = G(\hat{q}) = -\frac{1-\beta}{\beta}\alpha'(\hat{q})(1-L(\hat{q}))(1+\hat{q}) - \alpha(\hat{q}) - (\rho-b+d)$. If $\hat{G} \le 0$, it is a disease-

endemic poverty trap, and if $\hat{G} > 0$, it is a disease-endemic BGP.

When $\alpha''(q)$ is big enough, we have $\frac{\partial \hat{G}}{\partial \hat{q}} < 0$. Then,

$$\begin{aligned}
&\frac{d\hat{G}}{d\beta} = -(-\frac{1}{\beta^2})\alpha'(\hat{q})(1-L(\hat{q}))(1+\hat{q}) < 0;\\
&\frac{d\hat{G}}{d\rho} = \frac{\partial \hat{G}}{\partial \rho} + \frac{\partial \hat{G}}{\partial \hat{q}} \cdot \frac{\partial \hat{q}}{\partial \rho} = -1 + \frac{\partial \hat{G}}{\partial \hat{q}} \cdot \frac{\partial \hat{q}}{\partial \rho} < 0;\\
&\frac{d\hat{G}}{dd} = \frac{\partial \hat{G}}{\partial d} + \frac{\partial \hat{G}}{\partial \hat{q}} \cdot \frac{\partial \hat{q}}{\partial d} = -1 + \frac{\partial \hat{G}}{\partial \hat{q}} \cdot \frac{\partial \hat{q}}{\partial d} < 0;\\
&\frac{d\hat{G}}{d\psi} = \frac{\partial \hat{G}}{\partial \hat{q}} \cdot \frac{\partial \hat{q}}{\partial \psi} > 0.
\end{aligned}$$

That is, when infectious diseases are endemic, countries are more likely to undergo a positive economic growth path, if capital share (β) is smaller; households are more patient (i.e. ρ is smaller); life expectancy rises (i.e. d is smaller), or effectiveness of human capital accumulation (ψ) is higher.

A.4 Proof of Proposition 3: Resource Allocation

Here, we provide resource allocation in a decentralized economy.

1) Countries in the disease-free BGP: Substitute $L^* = 1$ and $\frac{\lambda_1}{\lambda_1} = -g$ into equation (A.7), we have $\beta A(\frac{K}{eu})^{\beta-1} = g + \rho + \delta$. Then

$$\begin{aligned}
\frac{I_K}{Y} &= \frac{\dot{K} + (\delta + b - d)K}{Y} = (\frac{\dot{K}}{K} + \delta + b - d)\frac{1}{A}(\frac{K}{eu})^{1-\beta} = \beta\frac{g+\delta+b-d}{g+\rho+\delta};\\
\frac{C}{Y} &= 1 - \frac{I_K}{Y} = 1 - \beta\frac{g+\delta+b-d}{g+\rho+\delta}.
\end{aligned}$$

Substitute $g = \psi - (\rho - b + d)$, and we obtain the results for countries in the disease-free BGP.

2) Countries in the disease-endemic BGP: Combing equations (A.11) and (A.12), we have $\beta A(\frac{K}{euL})^{\beta-1} = (g + \rho + \delta)(1 + q)$. Then

$$\begin{aligned}
\frac{I_K}{Y} &= \frac{\dot{K} + (\delta + b - d)K}{Y} = (\frac{\dot{K}}{K} + \delta + b - d)\frac{1}{A}(\frac{K}{euL})^{1-\beta} = \beta\frac{g+\delta+b-d}{g+\rho+\delta}\cdot\frac{1}{1+q};\\
\frac{I_H}{Y} &= \frac{\dot{H} + (\delta + b - d)H}{Y} = (g + \delta + b - d)\cdot\frac{H}{K}\cdot\frac{K}{Y} = \beta\frac{g+\delta+b-d}{g+\rho+\delta}\cdot\frac{q}{1+q};\\
\frac{C}{Y} &= 1 - \frac{I_K}{Y} - \frac{I_H}{Y} = 1 - \beta\frac{g+\delta+b-d}{g+\rho+\delta}.
\end{aligned}$$

Substitute $g = \psi L^* - (\rho - b + d)$, and we obtain the results for countries in the disease-endemic BGP.

3) Countries in the disease-endemic poverty trap: Similar to countries in the diseases-endemic BGP, we substitute into $g = 0$ and obtain the results for countries in the poverty trap.

A.5 Proof of Proposition 5: Optimal Public Health Subsidy

In the following analysis, the superscript τ is used in denoting variables in the decentralized economy with the health subsidy.

In a decentralized economy with health subsidy,

1. *There exists a unique disease-free BGP with the growth rate* $g^\tau = \psi - (\rho - b + d)$;
2. *There exists a unqie disease-endemic case with* $L^{*,\tau} = L(q^{*,\tau})$.
   (a) *If* $L^{*,\tau} > \frac{\rho-b+d}{\psi}$ *or* $q^{*,\tau} > \hat{q}$, *it is a BGP with* $u^{*,\tau} = \frac{\rho-b+d}{\psi L^{*,\tau}}$, *and* $g^c = \psi L^{*,\tau} - (\rho - b + d)$;
   (b) *If* $L^{*,\tau} \leq \frac{\rho-b+d}{\psi}$ *or* $q^{*,\tau} \leq \hat{q}$, *it is a poverty trap.*

   The effective health capital $q^{*,\tau}$ *is determined by the equation*

$$G^\tau(q) = \max\{G_L^\tau(q), G_R^\tau(q)\} = 0,$$

   where

$$\begin{aligned} G_L^\tau(q) &= -\frac{1-\beta}{\beta}\alpha'(q)(1-L(q))(1+q+\tau) - \alpha(q) - (\rho - b + d), \quad \textit{and} \\ G_R^\tau(q) &= -\frac{1-\beta}{\beta}\alpha'(q)(1-L(q))(1+q+\frac{\rho+\delta}{\rho+\delta+(1+\tau)g}\tau)\frac{\psi L(q)}{\rho-b+d} - \alpha(q) - (\rho - b + d). \end{aligned}$$

The proof is the similar to the proof of Proposition 1, and hence ignored here. When infectious diseases are eradicated, there is no health expenditure and thus no need for the health subsidy. The disease-free BGP is the same as those in the decentralized economy shown in Proposition 1 and the centralized economy shown in Proposition 4. When infectious diseases are endemic, the effective health capital $q^{*,\tau}$ is determined by the equation $G^\tau(q) = 0$. Compared with $G(q) = 0$ in Proposition 1, the difference lies in the first term in the net marginal benefit, which is distorted by the relative marginal value of physical capital investment and health expenditure, due to the subsidy τ. We can rewrite $G^\tau(q)$ as follows:

$$\begin{aligned} G_L^\tau(q) &= G_L(q) + \left[-\frac{1-\beta}{\beta}\alpha'(q)(1-L(q))\tau\right], \quad \text{and} \\ G_R^\tau(q) &= G_R(q) + \left[-\frac{1-\beta}{\beta}\alpha'(q)(1-L(q))\frac{\psi L(q)}{\rho-b+d}\frac{\rho+\delta}{\rho+\delta+(1+\tau)g}\tau\right]. \end{aligned}$$

Clearly with the health subsidy, countries are more likely to be in the positive economic growth path.

The subside τ is chosen such that $q^{*,\tau}$ determined by equation $G^\tau(q) = 0$ is the same as $q^{*,c}$ determined by equation $G(q) + b + \gamma = 0$. Let $q^* = q^{*,\tau} = q^{*,c}$.

If $q^* \leq \hat{q}$, we know $G(q^*) + b + \gamma = 0$, which implies $-\frac{1-\beta}{\beta}\alpha'(q^*)(1-L(q^*))(1+q^*) - \alpha(q^*) -$

$(\rho - b + d) + b + \gamma = 0$. From $G^{\tau}(q^*) = 0$, we have

$$\begin{aligned}
\tau &= \frac{\alpha(q^*) + \rho - b + d}{-\frac{1-\beta}{\beta}\alpha'(q^*)(1 - L(q^*))} - (1 + q^*) \\
&= \frac{\alpha(q^*) + \rho - b + d}{\frac{\alpha(q^*) - (b+\gamma) + \rho - b + d}{1+q^*}} - (1 + q^*) \\
&= (1 + q^*)\frac{b + \gamma}{\alpha(q^*) - (b + \gamma) + \rho - b + d}.
\end{aligned}$$

Similarly, if $q^* > \hat{q}$, we know $G(q^*) + b + \gamma = 0$, which implies $-\frac{1-\beta}{\beta}\alpha'(q^*)(1 - L(q^*))(1 + q^*)\frac{\psi L^*}{\rho - b + d} - \alpha(q^*) - (\rho - b + d) + b + \gamma = 0$. From $G^{\tau}(q^*) = 0$, we have

$$\frac{\rho + \delta}{\rho + \delta + (1 + \tau)g}\tau = (1 + q^*)\frac{b + \gamma}{\alpha(q^*) - (b + \gamma) + \rho - b + d},$$

and

$$\tau = (1 + q^*)\frac{b + \gamma}{\alpha(q^*) - (b + \gamma) + \rho - b + d} \cdot \frac{\rho + \delta + g}{\rho + \delta - (1 + q^*)\frac{b+\gamma}{\alpha(q^*) - (b+\gamma) + \rho - b + d}g}.$$