Infectious Diseases and Endogenous Growth

Aditya Goenka*
(National University of Singapore)

Lin Liu†
(University of Rochester)

September 30, 2009

Abstract: We study an endogenous economic growth model where there is a prevalence of infectious diseases. The mechanism of growth is human capital accumulation model through learning by doing as in Lucas (1988). The dynamics of the spread of infectious diseases, which depends on the ratio of health and physical capital, is modeled explicitly by incorporating the *SIS* epidemiology model: Recovering from the disease does not confer subsequent immunity from it. Labor is indivisible with only the healthy people working. Working contributes to the accumulation of human capital through learning by doing. In the model with disease prevalence, the disease affects the effectiveness of human capital accumulation adversely and hence, the long run growth rate. Moreover we find that the growth rate depends on the proportion of the healthy population, which is in turn determined by the full range of the parameters. The decentralized economy is investigated by assuming each household takes as given the proportion of the population that is infected. The results indicate that there is underinvestment in health capital due to this negative externality.

Keywords: Infectious Diseases; Endogenous Growth; Epidemiology.
JEL Classification: E19, I10, E19, D90, O11.

*Correspondence to A. Goenka, Department of Economics, National University of Singapore, AS2, Level 6, 1 Arts Link, Singapore 117570, Email: ecsadity@nus.edu.sg

†Department of Economics, Harkness Hall, University of Rochester, Rochester, NY 14627, USA, Email: lliu18@mail.rochester.edu

1 Introduction

The history of mankind is inseparable from infectious diseases, which have adverse effects on the entire population and society. The recent outbreak of Swine flu has reiterated the impact of infectious diseases on economies. Historically, malaria has been claimed to be responsible for half of all human deaths from an infectious disease since the Stone Age. Smallpox killed 300 million people in the twentieth century alone. With time some of the diseases have been largely controlled controlled, e.g. small pox, owing to medical improvements and vaccination campaigns, but others continue to afflict the population. Diseases such as HIV/AIDS, malaria and some strains of influenza have received the most attention by economists and policy makers due to the high associated mortality. These are also diseases which cannot be eliminated by vaccination campaigns. There are other serious debilitating diseases which continue to play significant roles in human populations. Schistosomiasis is vector borne (by helminths) and is endemic in 74 countries and infects over 200 million people. While it is rarely fatal it is a chronic disease which can damage internal organs and in children impair growth and cognitive development. In tropical countries (especially in Africa) it is the second most important parasitic disease after malaria (see The Carter Centre (2008)). Trypanosomiasis (human sleeping sickness) is another endemic disease in tropical countries. In 1993 the World Bank estimated it to be the third most important disease in terms of economic impact in Africa (World Bank (1993)). A more recent paper extrapolates updated incidence levels and extrapolates them globally to estimate that it results in 10 million quality life adjusted years annually second only to malaria in terms of parasitic diseases (Moore, *et al*, 1999). There are other diseases which continue to be significant in both developing and developed countries. For example, while syphilis is relatively uncommon in the US incidence increased 75% between 1985-1990 (Waseem and Aslam (2007)), and between 2005-2006 increased by 118% (CDC (2008b)). Gonorrhea is much more common with over 700,00 cases annually in the US alone with an infection rate of 120.6 per 100,000 (CDC (2008a)). There is also a feeling that there may be "emerging infectious diseases", that is new infectious diseases which are previously unknown (Wald (2008)).

The incidence of infectious diseases is typically higher in developing countries which face tighter budget constraints, lack public health infrastructure and also lie in geographical regions where the parasites which cause the dis-

eases are prevalent. Moreover, outbreak of social strife can lead to increase in incidence of the disease (see discussion in Moore, *et al* (1999) in the context of trypanosomiasis. Pavlovsky (1966) in his work developed the concept of doctrine of natural nidality - or natural focus based on his study of encephalitis, relapsing fever, tularemia and plague. Many diseases have their natural focus in wild animals and arthropods which occur in a certain terrain. When the human interface with this terrain increases due to the development process and landscape modifications, then the diseases may cross-over to the humans (see also Meade and Earickson (2005) and Henley (2007) who discusses malaria in Indonesia). The development process usually involves changes in landscape use. Thus, it is important to know the interaction of continuing economic growth (endogenous growth) and infectious diseases. Intensive farming methods have been blamed for the recent outbreak of swine flu, avian flu, and of SARS.

There is a growing economic literature studying this issue (Bell *et al* (2003), Delfino and Simmons (2000), Gersovitz and Hammer (2003), Goenka and Liu (2006), (2007), Young (2005)). However, the interaction of infectious diseases with macroeconomic performance especially long run economic growth is not well understood. The classical economic growth literature ignores the impact of infectious diseases on the long run economic growth (see for example Aghion and Howitt (1997), and Barro and Xala-i-Martin(2003)). Usually technological progress, human capital accumulation, variety expansion and quality improvement are considered to be the source of economic growth, in which health plays no role. However, health may affect the long run economic growth rate indirectly through the other mechanisms, such as, reducing the effectiveness of human capital accumulation. On the other hand, even if the effect of diseases is incorporated into the economic model (e.g. Lopez-Casanovas et al (2005)), the dynamics of infectious diseases is not explicitly modeled especially as health expenditures on controlling infectious diseases interact in complex ways with accumulation of physical and human capital. In this paper we incorporate insights from the mathematical biology literature on epidemiology of infectious diseases (see Anderson and May (1991), Brauer and Castillo-Chavez (2001), Hethcote (2000), (2005)) into an endogenous growth model. These epidemiology models lend themselves into integration into dynamic economic models as they capture disease transmission via dynamical systems. The literature on economic models of epidemiology (see the survey by Philipson (2000)) looks at rational choices of individuals in the face of the diseases and how these choices may affect the

spread of the disease. These choices are very important in case of diseases generated by one-to-one contact such as STDs. They are less applicable to the diseases considered in this paper where short of quarantine (isolation of the healthy population), there may be no way to avoid infection from the disease.

Given the preliminary knowledge about the relationship between the infectious diseases and economic growth, it is important to study this two-way relationship more carefully in order to provide a theoretical base for examining the determinants of long term economic growth. This paper integrates epidemiological dynamics into a dynamic economic model. It examines the effect of the canonical epidemiological structure - SIS dynamics (See Figure 1) - in the Lucas(1988) endogenous growth model with human capital accumulation through learning by doing. The epidemiological structure is as follows: An individual is born healthy or susceptible to the disease, i.e., in state S. He/she can become infective, i.e., that is infected with the disease and capable of transmitting it to others, and transit to state I, according to a law of motion. While infective he/she can recover according to some laws of motion. In the simplest model, contracting disease does not confer immunity, and thus, the individual transits to the state of being susceptible, i.e., S. This covers several infectious diseases such as flu, conjunctivitis, STDs such as gonorrhea and syphilis, tuberculosis, SARS, strep throat, encephalitis, etc. Other diseases such as dengue, malaria, schistosomiasis and trypanosomiasis also have an SIS nature but it should be kept in mind that they are vector borne diseases. The SIS dynamics are the canonical dynamics on which more complicated models such as $MSEIR$, etc. are based on. Thus, it is a natural point in understanding epidemiological dynamics in economic models. Moreover, in order to capture the effect of the economy on the disease transmission, it is endogenized in that both contact rate and recovery rate, the key parameters in epidemiology model depend on economic variables. Thus, people can influence the disease transmission through preventive and therapeutic behaviors by investing more in health. In the model we also incorporate the fact that higher physical capital could be detrimental to the immune system such that healthy people could easily get infected or the infected have difficulty in getting recovered due to the pollution caused by more plants etc. Thus, for simplicity we take epidemiological parameters as a function of the ratio of health and physical capital.

In this paper we want to study the effect of infectious diseases on the growth rate. There are many factors identified as the determinants of eco-

nomic growth, such as technological progress, human capital accumulation, innovation etc. The most natural way to model the effect of diseases would be through its interaction with the human capital channel. Given that diseases have the effect of debilitating infected individuals, human accumulation with learning by doing is the most relevant one in examining the effect of diseases. We assume only the healthy people work with labor supplied inelastically. Thus, we abstract away from the labour-leisure choice as is commonly done in these class of models. Since the human capital accumulation depends on the effective labor force, disease prevalence is likely to have an adverse effect on it, and hence, on the long run growth rate. We show that in the balanced growth path, health, physical, human capital and consumption all grow at the same rate. We further find that the growth rate depends on the proportion of healthy people, which is in turn determined by the full range of parameters. This is unlike the Lucas (1988) model where some parameters like discount rate or risk aversion preference, etc. only have level effect rather than growth effects. In our paper, all the parameters have growth effect because they determines the optimal ratio of health and physical capital and hence the proportion of the healthy people in a balanced growth path and long run growth rate. In the paper, the decentralized economy is also investigated by assuming each household takes as given the proportion of the population that is infected. The results indicate that there is underinvestment in health capital due to a negative externality, as compared to the centralized economy.

The paper is organized as follows: Section 2 describes the basic model, Section 3 examines the balanced growth path, and Section 4 investigates the decentralized economy.

2 The Basic Model

In this paper we use the SIS epidemiological structure. We assume that individuals are born healthy and susceptible to the disease. There is homogeneous mixing so that the likelihood of any individual contracting the disease is the same, irrespective of age. Thus, there is only horizontal incidence of the disease i.e. from peers. The contact structure is the standard incidence or frequency dependant model. Let $S(t)$ be the number of susceptibles at time t, $I(t)$ be the number of infectives/infected and $N(t)$ be the total population size. The fractions of individuals in the susceptible and

infected class are $s(t) = S(t)/N(t)$ and $i(t) = I(t)/N(t)$, respectively. 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 $(\alpha I/N)S$. This is the standard model used in the epidemiology literature (Hethcote (2005)). The basic idea is that the pattern of human interaction is relatively stable and what is important is the *fraction of infected people* rather than the total number. If the population increased the pattern of interaction is going to be invariant. The latter model (i.e., new cases equal to $\alpha I(t)S(t)$) is used typically for herd animals or for very densely populated urban areas. The parameter 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. The recovery of individuals is governed by the parameter γ and the total number of individuals who recover from the disease at time t is $\gamma I(t)$. This corresponds to exponentially distributed recovery time, i.e. $P(t) = e^{-\gamma t}$ is the fraction of infected class still infected t periods after becoming infected and $1/\gamma$ is the mean waiting time.

Many epidemiology models assume total population size to be constant when the period of interest is short, i.e. less than a year, or when natural births and deaths and immigration and emigration balance each other. As we are interested in long run effects, we assume that there is a constant birth rate b, and a constant (natural) death rate d. In this paper we abstract away from disease related mortality. This is a significant assumptions as it shuts down the demographic interaction. This assumption is made for two reasons. First, several SIS diseases have low mortality so there is no significant loss by this assumption. These include several strains of influenza, meningitis, STDs (syphilis, gonorrhea), dengue, conjunctivitis, strep throat, etc (Anderson and May (1991)). For example, between January-September 2005, there were 9,540 cases of Dengue fever in Singapore out of which there were 8 mortalities; January-May 2005, Thailand - 8900 cases and 16 mortalities; January-August 2005, Indonesia - 43,509 cases and 605 mortalities (Chen (2005)). Secondly, from an economic modeling point of view we can use the standard discounted utility framework with an exogenous discount rate if mortality is exogenous. Despite the simplification, we show several new insights emerge from incorporating epidemiological dynamics in economic models.

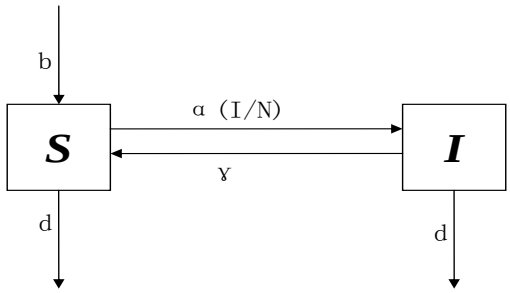

Figure 1: The transfer diagram for the SIS epidemiology model

Assumption 1: The birth rate b and death rate d are positive constant scalars with $b - d \geq 0$.

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

$$\begin{aligned} &dS/dt = bN - dS - \alpha SI/N + \gamma I \\ &dI/dt = \alpha SI/N - (\gamma + d)I \\ &dN/dt = (b - d)N \\ &S, I, N \geq 0 \, \forall t; S_0, I_0, N_0 > 0 \, \text{given.} \end{aligned}$$

Since $N(t) = S(t) + I(t)$, we can simplify the model in terms of the susceptible fraction s_t

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

with the total population growing at the rate $b - d$. In the pure epidemiology model there are two steady state equilibria ($\dot{s_t} = 0$) given by: $s_1^* = 1$ and $s_2^* = \frac{b+\gamma}{\alpha}$. We notice s_1^* (the disease-free steady state) exists for all parameter values while s_2^* (the endemic steady state) exists only when $\frac{b+\gamma}{\alpha} < 1$.

We now introduce the economic model and focus on the centralized economy. The key feature is modeling the labor supply. There is a population of size $N(t)$ growing over time at the rate of $b - d$. Each individual's labor is indivisible (Hansen, 1985 and Rogerson, 1988). We assume infected people cannot work and labor force consists only of healthy people with labor supplied inelastically. Thus in time period t the labor supply is

$L(t) = N(t) - I(t) = S(t)$ and henceforth $L(t)$ inherits the dynamics of $S(t)$, that is,

$$\dot{l}_t = (1 - l_t)(b + \gamma - \alpha l_t), \tag{2}$$

in terms of the fraction of labor force $l_t = L_t/N_t$. There is a two-way interaction between the economy and the disease. On the one hand, diseases have adverse effects on the economy by reducing the labor force participants. On the other hand, the economy will also have impact on the disease transmission. The existing epidemiology models, from an economists point of view, do not go far enough as they treat the parameters of the disease dynamics as biological constants. In this paper, the disease transmission is endogenized in that both contact rate and recovery rate, the key parameters in epidemiology model, do depend on economic variables. Thus, people could intervene in the diseases transmission through preventive and therapeutic behaviors by investing more in health. At the same time, higher physical capital could be detrimental to the immune system such that healthy people could easily get infected or the infected have difficulty in recovering from the disease, due to the pollution caused by more plants etc. Thus, for simplicity we assume the contact rate α and the recovery rate γ is a function of $\frac{h}{k}$, the ratio of health and physical capital. Hence, this takes into account the impact of human being's intervention on the diseases transmission dynamics. So when health capital is higher or physical capital is lower, people are less likely to get infected and more likely to recover from the diseases. As usual, the marginal effect diminishes as $\frac{h}{k}$ increases and Inada conditions are also imposed.

Assumption 2: The epidemiological parameter functions $\alpha(\frac{h}{k})$ and $\gamma(\frac{h}{k})$: $\Re_+ \times \Re_+ \to \Re_+$:

1. $\alpha(\frac{h}{k})$ is a C^2 function with $\alpha' \leq 0$, $\alpha'' \geq 0$, $\lim_{\frac{h}{k}\to 0} \alpha' \to -\infty$, $\lim_{\frac{h}{k}\to \infty} \alpha' \to 0$, $\alpha \to \overline{\alpha}$ as $\frac{h}{k} \to 0$ and $\alpha \to \underline{\alpha}$ as $\frac{h}{k} \to +\infty$;
2. $\gamma(\frac{h}{k})$ is a C^2 function with $\gamma' \geq 0$, $\gamma'' \leq 0$, $\lim_{\frac{h}{k}\to 0} \gamma' \to +\infty$, $\lim_{\frac{h}{k}\to \infty} \gamma' \to 0$, $\gamma \to \underline{\gamma}$ as $\frac{h}{k} \to 0$ and $\gamma \to \overline{\gamma}$ as $\frac{h}{k} \to +\infty$;

The economic model follows Lucas(1988) endogenous growth model with learning by doing human capital accumulation. This is a centralized economy and the average human capital, given by $e(t)$ affects labor productivity. The

effective labor supply is $e(t)L(t)$. The output is produced using capital and effective labor and the production function is given as $AK(t)^{\beta}(e(t)L(t))^{1-\beta}$, which exhibits constant return to scale in physical and human capital. The output is either consumed $c(t)N(t)$, invested into physical capital $\dot{K(t)}$ or spent in health expenditure $m(t)N(t)$, which contributes to health capital $H(t)$. For simplicity, we assume full depreciation of physical and health. Thus the physical capital $K(t)$ and health capital $H(t)$ are accumulated as follows:

$$\dot{K(t)} = AK(t)^{\beta}(e(t)L(t))^{1-\beta} - c(t)N(t) - m(t)N(t)$$
$$\dot{H(t)} = m(t)N(t).$$

The effort people spend on working or the amount of time spent working contributes to the accumulation of human capital. This is a natural assumption where the time lost to work due to work will lead to lower skill and dexterity in carrying out the various tasks and hence, lead to lower labor productivity and hence, a lower effective labor supply. Thus, the growth of average human capital $\dot{e(t)}$ is determined by the level already attained and the time spent working. In order for human capital to be the engine of growth, as in Lucas (1988) there are no diminishing returns to the accumulation of human capital and for simplicity we assume the change of human capital is linear in level already attained and the effort on working, that is,

$$\dot{e(t)} = \delta e(t) l_t,$$

where δ is the effectiveness of human capital accumulation.

We further assume there is full insurance and so each individual has the same consumption irrespective of his health status. This is consistent to the fact that we are looking at the optimal solution and given concavity of the period utility function, any efficient allocation will involve full insurance. Each individual's utility function is CES. The central planner maximizes discounted streams of total welfare with the discount rate, ρ [1]. For simplicity, we drop time subscript t when it is self-evident. The central planner maximizes total welfare by choosing both consumption c and health expenditure

[1] see Arrow and Kurz (1970) for a discussion of maximizing total as opposed to representative consumer welfare. The qualitative results do not change if the other objective is used.

m, and the optimization problem in per capita terms is given as follows:

$$\max \int_0^\infty e^{-(\rho-b+d)t} \frac{c_t^{1-\sigma}-1}{1-\sigma} N_0 dt$$

s.t.

$$\dot{k} = Ak^\beta (le)^{1-\beta} - c - m - k(b-d) \tag{3}$$

$$\dot{e} = \delta e l \tag{4}$$

$$\dot{l} = (1-l)(b + \gamma(\frac{h}{k}) - \alpha(\frac{h}{k})l) \tag{5}$$

$$\dot{h} = m - h(b-d) \tag{6}$$

$$k_t, h_t, e_t \geq 0, m \geq 0, 0 \leq l_t \leq 1 \, \forall t$$

$$k_0, h_0, e_0, l_0 > 0 \, \text{given.}$$

3 The Balanced Growth Path

We are interested in the balanced growth paths, where physical capital, human capital and health capital grow at a constant rate while the proportion of labor force among the total population remains constant. Define $g_k = \frac{\dot{k}}{k}$, $g_e = \frac{\dot{e}}{e}$ and $g_h = \frac{\dot{h}}{h}$. From the Inada conditions we can rule out $k = 0$, and the constraint $l \geq 0$ is not binding since $\dot{l} = b + \gamma > 0$ whenever $l = 0$. e is strictly greater than 0 as $e_0 > 0$ and $\frac{\dot{e}}{e} = \delta l > 0$. The constraint $h \geq 0$ can be inferred from $m \geq 0$, and hence can be ignored. So there are only two constraints $l \leq 1$ and $m \geq 0$ needed to be taken care of. Moreover we know when l is strictly less than 1, m has to be strictly positive since the diseases is prevalent and it is always optimal to invest in health given by the Inada assumption imposed on the epidemiological parameter functions $\alpha(\cdot)$ and $\gamma(\cdot)$. And $l^* = 1$ implies $m = 0$ as the disease is eradicated and there is no need for any health expenditure in controlling the diseases. Thus, the optimization problem degenerates to the one in Lucas(1988), and in the BGP the growth rate is given by $g = \delta$. Moreover this BGP exists for all parameter values.

Proposition 1 *Under A.1 − A.2 there always exists a unique disease-free BGP with growth rate $g = \delta$.*

Now consider the maximization problem with an interior solution. The current value Lagrangian is given by:

$$\mathcal{H} = \frac{c_t^{1-\sigma}-1}{1-\sigma} + \lambda_1[Ak^\beta(le)^{1-\beta} - c - m - k(b-d)] + \lambda_2[\delta el] + \\ + \lambda_3[(1-l)(b+\gamma(\frac{h}{k}) - \alpha(\frac{h}{k})l)] + \lambda_4[m - h(b-d)]$$

The necessary conditions and TVCs are:

$$c : c^{-\sigma} = \lambda_1 \tag{7}$$
$$m : \lambda_1 = \lambda_4 \tag{8}$$
$$k : \dot{\lambda_1} = \rho\lambda_1 - \lambda_1\beta Ak^{\beta-1}(le)^{1-\beta} + \lambda_3(1-l)(\gamma' - \alpha' l)\frac{h}{k^2} \tag{9}$$
$$e : \dot{\lambda_2} = (\rho - b + d)\lambda_2 - \lambda_1 A(1-\beta)k^\beta l^{1-\beta}e^{-\beta} - \lambda_2\delta l \tag{10}$$
$$l : \dot{\lambda_3} = (\rho - b + d)\lambda_3 - \lambda_1 A(1-\beta)k^\beta l^{-\beta}e^{1-\beta} - \lambda_2\delta e - \\ - \lambda_3(2\alpha l - \alpha - \gamma - b) \tag{11}$$
$$h : \dot{\lambda_4} = \rho\lambda_4 - \lambda_3(1-l)(\gamma' - \alpha' l)/k \tag{12}$$
$$\lim_{t\to\infty} e^{-(\rho-b+d)}\lambda_1 k = 0 \quad \lim_{t\to\infty} e^{-(\rho-b+d)}\lambda_2 e = 0 \tag{13}$$
$$\lim_{t\to\infty} e^{-(\rho-b+d)}\lambda_3 l = 0 \quad \lim_{t\to\infty} e^{-(\rho-b+d)}\lambda_4 h = 0. \tag{14}$$

For the interior case or endemic case, $l^* = \frac{b+\gamma(\frac{h}{k})}{\alpha(\frac{h}{k})} < 1$ by letting equation (5) equal to zero. And l strictly increases as h/k increases. So the endemic BGP exists only when $\lim_{\frac{h}{k}\to 0}\frac{b+\gamma(\frac{h}{k})}{\alpha(\frac{h}{k})} < 1$, that is $b < \overline{\alpha} - \underline{\gamma}$. Moreover, l is constant in the BGP implies h/k is also constant over the BGP, i.e. h and k grow at the same rate. From equation (21) and (21), $\frac{\dot{\lambda_1}}{\lambda_1} = \frac{\dot{\lambda_4}}{\lambda_4} = -\sigma g_c$. Then dividing equation (21) by λ_4 on both sides and rearranging, we have:

$$\frac{\lambda_3}{\lambda_1} = \frac{\rho + \sigma g_c}{(1-l)(\gamma' - \alpha' l)}k. \tag{15}$$

From equation (21),

$$\frac{\dot{\lambda_1}}{\lambda_1} = \rho - \beta Ak^{\beta-1}(le)^{1-\beta} + \frac{\lambda_3}{\lambda_1}(1-l)(\gamma' - \alpha' l)\frac{h}{k^2},$$

and using equation (21) and (15),

$$-\sigma g_c = \rho - \beta Al^{1-\beta}(\frac{e}{k})^{1-\beta} + (\rho + \sigma g_c)\frac{h}{k},$$

that is,

$$\beta A l^{1-\beta}(\frac{e}{k})^{1-\beta} = (\rho + \sigma g_c)(1 + \frac{h}{k}). \tag{16}$$

So we get a function of both $\frac{e}{k}$ and $\frac{h}{k}$. Moreover, $\frac{e}{k}$ is constant, i.e. $g_e = g_k$. From equation (3) and (6), we have

$$\frac{\dot{k}}{k} = A l^{1-\beta}(\frac{e}{k})^{1-\beta} - \frac{c}{k} - \frac{\dot{h}}{h}\frac{h}{k} - (b-d)\frac{h}{k} - (b-d).$$

Substituting equation (16) into the above equation, we have a function of both $\frac{c}{k}$ and $\frac{h}{k}$:

$$\frac{c}{k} = [\frac{1}{\beta}(\rho + \sigma g_c) - g_k - (b-d)](1 + \frac{h}{k}). \tag{17}$$

So $\frac{c}{k}$ is a constant, i.e. c and k grow at the same rate. Furthermore from equation (21), $g_e = \delta l^*$. Thus physical capital, human capital, health capital and consumption all grow at the same rate $g = \delta l^*$ in the BGP. In equation (15), since $\frac{\lambda_3}{\lambda_1} = \text{Constant} \times k$,

$$\frac{\dot{\lambda_3}}{\lambda_3} = \frac{\dot{\lambda_1}}{\lambda_1} + \frac{\dot{k}}{k} = (1-\sigma)g.$$

Dividing both sides of equation (21) by λ_3 and substituting $\frac{\lambda_3}{\lambda_1}$ and $\frac{\dot{\lambda_3}}{\lambda_3}$, we get

$$(1-\sigma)g = \rho - b + d - \frac{(1-l)(\gamma' - \alpha' l)(1-\beta)}{\beta l}(1 + \frac{h}{k}) - \frac{\lambda_2}{\lambda_3}\delta e - (2\alpha l - \alpha - \gamma - b). \tag{18}$$

From the above equation, since $\frac{\lambda_2}{\lambda_3}\delta e = \text{Constant}$, $\frac{\dot{\lambda_2}}{\lambda_2} = \frac{\dot{\lambda_3}}{\lambda_3} - \frac{\dot{e}}{e} = -\sigma g$. Furthermore from equation (21),

$$\frac{\dot{\lambda_2}}{\lambda_2} = \rho - b + d - \frac{\lambda_1}{\lambda_2} A(1-\beta) k^{\beta} l^{1-\beta} e^{-\beta} - \delta l.$$

From equation (15) $\lambda_1 = \frac{(1-l)(\gamma' - \alpha' l)}{(\rho + \sigma g)k}\lambda_3$, we have

$$\frac{\lambda_2}{\lambda_3} = \frac{(1-l)(\gamma' - \alpha' l)A(1-\beta)k^{\beta-1}l^{1-\beta}e^{-\beta}}{(\rho + \sigma g)(\rho - b + d - \delta l + \sigma g)}.$$

Substituting this into equation (18), we get

$$\rho - b + d - \frac{(1-l)(\gamma' - \alpha' l)(1-\beta)}{\beta}(1 + \frac{h}{k})(\frac{1}{l} + \frac{\delta}{\rho - b + d - \delta l + \sigma g}) -$$
$$- (2\alpha l - \alpha - \gamma - b) = (1-\sigma)g.$$

Since $l = \frac{b+\gamma(\frac{h}{k})}{\alpha(\frac{h}{k})}$ and $g = \delta l$, the above equation is a function of $\frac{h}{k}$. Define $x = \frac{h}{k}$ and rewrite the above equation as:

$$\frac{[\rho - b + d - (1-\sigma)\delta l(x) + \alpha(x) - b - \gamma(x)][\rho - b + d - (1-\sigma)\delta l(x)]}{(1+x)[\rho - b + d + \sigma\delta l(x)]} =$$
$$= \frac{1-\beta}{\beta}\frac{(1-l(x))(\gamma'(x) - \alpha'(x)l(x))}{l(x)}. \tag{19}$$

As long as we can solve the above equation for x, we can get the proportion of healthy people l and the growth rate g. So next we want to show there exists at least one solution x which satisfies equation (19).

Assumption 3: $\rho > b - d + (1-\sigma)\delta$.

Clearly both the L.H.S. and R.H.S. of the equation (19) are decreasing functions of x. Moreover, $l(x) = \frac{b+\gamma(x)}{\alpha(x)}$ is increasing in x and $l(x) \leq 1$ has to be satisfied. By assumption, $\lim_{x\to 0} \frac{b+\gamma(x)}{\alpha(x)} < 1$ and and we examine $\lim_{x\to\infty} \frac{b+\gamma(x)}{\alpha(x)}$, case by case.

Case 1: $\lim_{x\to\infty} \frac{b+\gamma(x)}{\alpha(x)} \leq 1$. As x goes to 0, the L.H.S. goes to a positive finite number under A.3, while x goes to ∞ the L.H.S. goes to 0. For the R.H.S., as x approaches 0 it goes to $+\infty$ while x approaches ∞ it goes to 0. Moreover as x goes to ∞ the R.H.S. converges to 0 faster than the L.H.S. does since $\lim_{x\to\infty}(1+x)(\gamma'(x) - \alpha'(x)l(x)) = 0$ by assuming $\lim_{x\to\infty} \frac{[\alpha'(x)]^2}{\alpha''(x)} \to 0$ and $\lim_{x\to\infty} \frac{[\gamma'(x)]^2}{\gamma''(x)} \to 0$. This assumption could be satisfied by taking the following functional forms: $\alpha(x) = ae^{-x}$ and $\gamma(x) = r(1-e^{-x})$. Thus as shown in figure 2 at least there exists one intersection of the two curves given by the L.H.S. and the R.H.S. of the equation (19).

Case 2: $\lim_{x\to\infty} \frac{b+\gamma(x)}{\alpha(x)} > 1$. There exists $\overline{x}$ such that $\frac{b+\gamma(x)}{\alpha(x)} = 1$. As x goes to 0, the L.H.S. goes to a positive finite number under A.3, while the

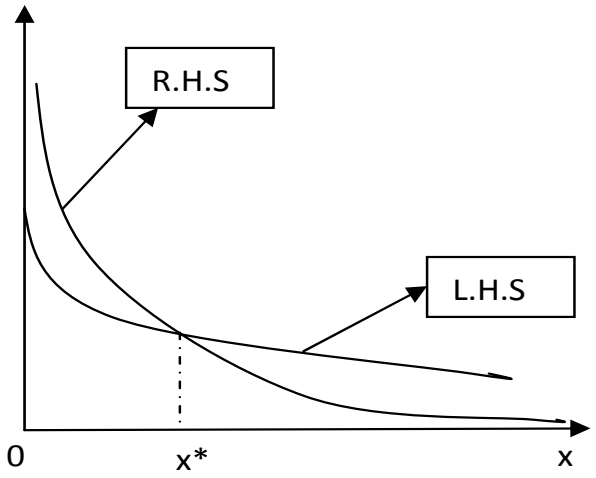

Figure 2: The curves of the L.H.S. and the R.H.S (Case 1).

R.H.S. goes to $+\infty$. As x goes to $\overline{x}$, the L.H.S. goes to a positive number while the R.H.S. goes to 0. Thus as shown in figure 3 at least there exists one intersection of the two curves given by the L.H.S. and the R.H.S. of the equation (19).

Proposition 2 *Under $A.1 - A.3$ there exists an endemic disease BGP with growth rate $g = \delta l(\frac{h}{k})$ when $b < \overline{\alpha} - \underline{\gamma}$, and the optimal health and physical capital ratio $\frac{h}{k}$ is determined by the equation (19).*

In addition, TVCs need to be satisfied in the BGP, that is, both equation (21) and (21) are satisfied. And it is equivalent to have

$$\begin{aligned}\lim_{t\to\infty} e^{-(\rho-b+d)t}\lambda_1(t)k(t) &= \lim_{t\to\infty} e^{-(\rho-b+d)t}\lambda_1(0)e^{-\sigma\delta lt}k(0)e^{\delta lt} \\ &= \lim_{t\to\infty} \lambda_1(0)k(0)e^{(-\rho+b-d+\delta l(1-\sigma))t} \\ &= 0\end{aligned}$$

to be true. And it is clearly satisfied under assumption 3.

Therefore there are two balanced growth paths. One is disease free BGP, which exists for all the parameter values and is the same as Lucas(1988) model with growth rate equal to the effectiveness of human capital accumulation δ. Thus only the parameter δ has growth effect while all the other

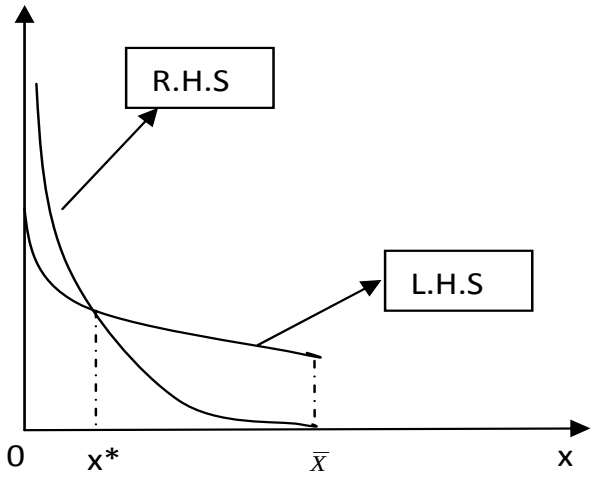

Figure 3: The curves of the L.H.S. and the R.H.S (Case 2).

parameters have level effects. And the higher the effectiveness of human capital accumulation, the higher the growth rate. The other BGP is endemic BGP, which exists only when birth rate is relatively low, that is, $b < \overline{\alpha} - \underline{\gamma}$. The growth rate under endemic BGP is the product of both effectiveness of human capital accumulation and the proportion of labor force, i.e. δl. Moreover the proportion of labor force is a function of the optimal health and physical capital ration $\frac{h}{k}$, which is determined by equation (19). The same mechanism as in the Lucas paper - human capital accumulation drives long term growth. However, here the disease prevalence affects the human capital accumulation adversely and hence, the long run growth rate. Moreover the growth rate depends on the proportion of the healthy population, which is in turn determined by the full range of the parameters. So all the parameters have growth effects. Next we want to explore how the growth rate along the endemic BGP changes as some of the important parameters vary.

For the equation (19) as the discount factor ρ increases the L.H.S shifts up and hence the optimal health and physical capital ratio decreases. So higher discount factor leads to lower growth rate. Higher discount factor means people are less patient, who care more about the current consumption rather than the steam of future income. So they invest less in health and end up with higher disease prevalence and lower growth rate. Similarly if the intensiveness of physical capital in the production function β increases,

the R.H.S. of equation (19) shifts down and $\frac{h}{k}$ decreases. So the higher the β, the lower the growth rate. This is because physical capital becomes relatively more important than human capital. So people invest more in physical capital instead of health capital, and $\frac{h}{k}$ drops which leads to lower growth rate. For the change in birth rate, it's more complicated because it plays a role in the diseases transmission process and also has impact on real discount factor $\rho - b + d$ in the maximization problem. From analytical result the effect of change in birth rate is ambiguous. So we run the simulation. The epidemiological parameter functions we choose are $\alpha = \alpha_0 e^{-x}$ and $\gamma = \gamma_0(1 - e^{-x})$. And the other parameters are as follows: $\rho = 0.05$, $d = 0.005$, $\sigma = 1$, $\delta = 0.2$ and $\beta = 0.36$. We change birth rate from $b = 0.01$ to $b = 0.02$. We find both the L.H.S. and the R.H.S. shift down and it's likely the intersection $\frac{h}{k}$ decreases. Thus the higher the birth rate, the lower the health investment and the lower the growth rate.

4 Decentralized Economy

Thus far we have examined the endogenous growth model with infectious diseases in a centralized economy framework. The next step is to consider a similar model but under a decentralized framework. Then we compare how the results from the decentralized economy differ from those in the centralized case. This is important as there is an externality from learning by doing which may not be fully internalized by individuals especially if the health status affects the size of the externality. In this extended model, we follow the simplified assumption in Gersovitz and Hammer (2004). We assume the economy is populated with many identical households, which are taken as the representative decision-making agents. Each household is assumed to be sufficiently large that the proportion of the household in each disease status is identical to the corresponding population proportion. Moreover the household understands and anticipates how the epidemic will evolve and is fully forward-looking with regard to its possible future statues as well as its present situation. The only difference between the social planner and the representative household is that the household is assumed to be small relative to the population as a whole. Thus, a household believes that the proportion of the household in any disease status does not affect the proportion of the population as a whole that is in that status. In particular, the household takes as given the proportion of the population that is infected, which equals

to the probability, π, that any random contact is with a person. The SIS epidemiology model is now given as follows:

$$dS/dt = bN - dS - \alpha S\pi + \gamma I$$
$$dI/dt = \alpha S\pi - (\gamma + d)I.$$

So equation (5) becomes:

$$\dot{l} = (1-l)(\gamma(\frac{h}{k}) + b) - \alpha(\frac{h}{k})\pi l. \tag{20}$$

Moreover, we say e_t is the average human capital for the household and the growth of human capital $\dot{e}_t$ is linear in e_t and determined by the effective human capital accumulation δ and labor force participation l_t, that is, $\dot{e}_t = \delta e_t l_t$. We further assume there is full insurance within the household and so all individuals in the household have the same consumption irrespective of health status. The utility function is CES and discounted at the rate ρ $(0 < \rho < 1)$. The optimization problem for the household is given as follows:

$$\begin{aligned}
&\max \int_0^\infty e^{-\rho t} \frac{c_t^{1-\sigma} - 1}{1-\sigma} dt \\
&\text{s.t.} \\
&\dot{k} = Ak^\beta (le)^{1-\beta} - c - m - k(b-d) \\
&\dot{e} = \delta e l \\
&\dot{l} = (1-l)(\gamma(\frac{h}{k}) + b) - \alpha(\frac{h}{k})\pi l \\
&\dot{h} = m - h(b-d) \\
&k_t, h_t, e_t \geq 0, m_t \geq 0, 0 \leq l_t \leq 1 \, \forall t \\
&k_0, h_0, e_0, l_0 > 0 \, \text{given.}
\end{aligned}$$

The same as the centralized case, there always exists disease-free BGP with growth rate $g = \delta$ in the decentralized economy. Along disease-free BGP the disease is fully eradicated and has no impact on the economy. And the only difference between the social planner and the individual household lies in how the household thinks about the impact of his behavior on the distribution of different diseases status among total population. Thus this difference does not lead to any discrepancy between the optimal solutions of two cases. And we have exactly the same growth rate in the disease-free BGP.

Similarly to the centralized case, there also exists an endemic BGP, which should be a more interesting case to look at. In order to only focus on how the social planner and the individual household react to the disease prevalence differently, we assume the social planner maximizes the representative agents's utility rather than the total welfare onwards. It could be easily done just by replacing $\rho - b + d$ with ρ in the centralized case . In the decentralized economy, with these modification the household's current-value Hamiltonian is:

$$\begin{aligned}\mathcal{H} &= \frac{c_t^{1-\sigma}-1}{1-\sigma} + \lambda_1[Ak^\beta(le)^{1-\beta} - c - m - k(b-d)] + \lambda_2[\delta el] + \\ &\quad + \lambda_3[(1-l)(\gamma(\frac{h}{k}) + b) - \alpha(\frac{h}{k})\pi l] + \lambda_4[m - h(b-d)]\end{aligned}$$

As usual, we get necessary conditions and TVCs:

$$\begin{aligned}&c : c^{-\sigma} = \lambda_1 \\ &m : \lambda_1 = \lambda_4 \\ &k : \dot{\lambda_1} = (\rho + b - d)\lambda_1 - \lambda_1\beta A k^{\beta-1}(le)^{1-\beta} + \lambda_3[(1-l)\gamma' - \alpha'\pi l]\frac{h}{k^2} \\ &e : \dot{\lambda_2} = \rho\lambda_2 - \lambda_1 A(1-\beta)k^\beta l^{1-\beta}e^{-\beta} - \lambda_2\delta l \\ &l : \dot{\lambda_3} = \rho\lambda_3 - \lambda_1 A(1-\beta)k^\beta l^{-\beta}e^{1-\beta} - \lambda_2\delta e + \lambda_3(\gamma + b + \alpha\pi) \\ &h : \dot{\lambda_4} = (\rho + b - d)\lambda_4 - \lambda_3[(1-l)\gamma' - \alpha'\pi l)/k \\ &\lim_{t\to\infty} e^{-\rho}\lambda_1 k = 0 \quad \lim_{t\to\infty} e^{-\rho}\lambda_2 e = 0 \\ &\lim_{t\to\infty} e^{-\rho}\lambda_3 l = 0 \quad \lim_{t\to\infty} e^{-\rho}\lambda_4 h = 0.\end{aligned}$$

Since each household is representative of society, we must have

$$\pi = 1 - l.$$

Then we substitute the above equation into the necessary conditions and we find the only difference between the social planner's problem and the individual household's problem is the co-state equation for λ_3:

$$\dot{\lambda_3} = \rho\lambda_3 - \lambda_1 A(1-\beta)k^\beta l^{-\beta}e^{1-\beta} - \lambda_2\delta e + \lambda_3(\gamma + b + \alpha(1-l)).$$

For the centralized economy if the social planner only maximizes the representative agent's utility, the equation (19) could be rewritten as:

$$\frac{[\rho - (1-\sigma)\delta l(x) + \alpha(x) - b - \gamma(x)][\rho - (1-\sigma)\delta l(x)]}{(1+x)[\rho + \sigma\delta l(x)]} =$$

$$= \frac{1-\beta}{\beta}\frac{(1-l(x))(\gamma'(x)-\alpha'(x)l(x))}{l(x)}. \tag{21}$$

Similarly, in the decentralized economy we could get the following equation, which determines the ratio between health capital and physical capital $x = \frac{h}{k}$,

$$\frac{[\rho-(1-\sigma)\delta l(x)+\gamma(x)+b+\alpha(x)(1-l(x))][\rho-(1-\sigma)\delta l(x)]}{(1+x)[\rho+\sigma\delta l(x)]} = \\ = \frac{1-\beta}{\beta}\frac{(1-l(x))(\gamma'(x)-\alpha'(x)l(x))}{l(x)}. \tag{22}$$

Compared with the equation (21), the difference lies in the numerator in the L.H.S. of the equation, that is, the term $\gamma(x)+b+\alpha(x)(1-l(x))$. Moreover from the equation (20) we have $l^* = \frac{\gamma+b}{\gamma+b+\alpha\pi}$. Since $\pi = 1-l$, we get $l^* = \frac{\gamma+b}{\alpha}$. Then we substitute this into equation (22) and the term $\gamma(x)+b+\alpha(x)(1-l(x))$ becomes $\alpha(x)$. The L.H.S and R.H.S. of both equation (21) and (22) are shown in figure 4. Clearly the ratio of health and physical capital in decentralized economy x_d^* is smaller than the one in centralized economy x_c^*. This arises as in the decentralized economy with the negative externality, households ignore the impact of their behavior on the population as a whole. Thus they invest less in health capital and more in physical capital.

5 The conclusion

This paper examines an endogenous growth model where the engine of growth is learning by doing. In this framework, the effect of infectious disease can be naturally seen: individuals who are ill cannot work and hence, do not accumulate human capital by learning by doing. This affects the average human capital and has a growth effect. As the proportion of individuals who are working depends on the parameters of the model, and this determines the growth rate, the full set of parameters have growth effects. This results also highlight that endemic diseases even if they do not lead to mortality, can have long run effects through this mechanism. As the set of institutions in a country may determine the parameters α and γ through the efficacy of the public health system, the results also indicate that more effective institutions through the health of individuals can have growth effects.

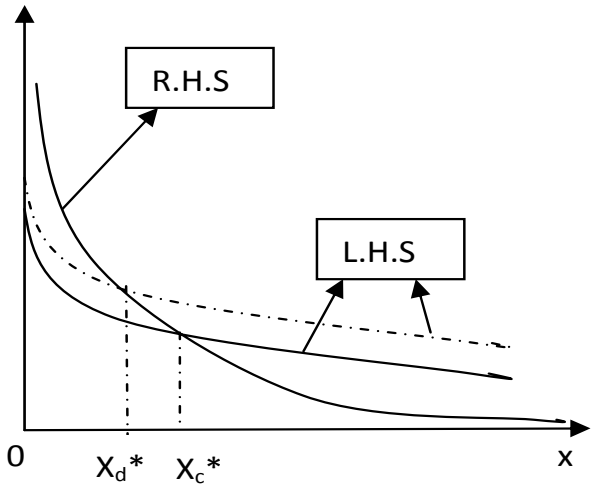

Figure 4: The comparison between the decentralized economy and the centralized economy

References

1. Aghion. P. and Howitt, P. (1997): *Endogenous growth theory*, Cambridge: MIT Press.

2. Arrow, K.J. and Kurz, M. (1970): *Public investment, the rate of return, and optimal fiscal policy*, Baltimore and London: The Resources for the Future, Inc.

3. Barro, R. and Sala-i-Martin (2003): *Economic growth*, 2nd Ed., Cambridge: MIT Press.

4. Bell, C., Devarajan, S. and Gersbach, H., (2003): "The long-run economic costs of AIDS: theory and an application to South Africa", *Working paper*.

5. Brauer, F., C. Castillo-Chavez (2001): *Mathematical models in population biology and epidemiology*, Berlin: Springer-Verlag.

6. The Carter Center: "Schistosomiasis Control Program," ⟨http://www.cartercenter.org/health/schistosomiasis/index.htm⟩. Retrieved on 1 September 2008.

7. Centre for Disease Control (2008a): "Gonorrhea - CDC Fact sheet," ⟨http://www.cdc.gov/std/gonorrhea/stdfact-gonorrhea.htm⟩. Retrieved on 1 September 2008.

8. Centre for Disease Control (2008b): "Syphilis - CDC Fact sheet," ⟨http://www.cdc.gov/std/syphilis/STDFact-Syphilis.htm⟩. Retrieved on 1 September 2008.

9. Chen, I-C.M. (2005) "Recent changes in dengue epidemiology in Singapore: The view from outside," Paper presented at *Mathematical Modeling of Infectious Diseases: Dynamics and Control*, (15 Aug - 9 Oct 2005), Institute of Mathematical Sciences, National University of Singapore.

10. 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.

11. Gersovitz, M. and Hammer, J.S., (2003): "Infectious disease, public policy, and the marriage of economics and eqidemiology", *The world bank research observer,18(2)* : 129-57.

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

13. Goenka, A., and Liu, L. (2006): "Infectious diseases and endogenous fluctuations," mimeo. National University of Singapore.

14. Goenka, A., and Liu, L. (2007): "Infectious diseases and economic growth," mimeo. National University of Singapore.

15. Hansen, G.D. (1985): "Indivisible labor and the business cycle," *Journal of Monetary Economics*, 16: 309-327.

16. 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.

17. Hethocote, H.W. (2000): "The mathematics of infectious diseases," *SIAM Review* 42: 599-653.

18. 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.

19. Lopez-Casasnovas, G., B. Rivera, and L. Currais (2005): *Health and Economic growth* Cambridge: MIT press.

20. Henley, D. (2007): *Malaria and malaria control in Indonesia, 1850-2000 Lessons from the past?*, unpublished manuscript, KITLV Leiden.

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

22. Meade, M.S. and R.J. Earickson (2005): *Medical Geography*, Guildford: Guilford Press.

23. Moore, A,, M. Richer, M. Enrile, E. Losio, J. Roberts and D. Levy (1999): "Resurgence of sleeping sickness in Tambura county, Sudan," *American Journal of Tropical Medicince and Hygiene*, 61(2): 315-318.

24. 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.

25. Philipson, T. (2000): "Economic epidemiology and infectious diseases," in J. Newhouse and T. Culyer (Eds.) *Handbook of Health Economics*, New York: North-Holland.

26. Rogerson, R. (1988): "Indivisible labor, lotteries and equilibrium," *Journal of Monetary Economics* 21: 3-16.

27. Wald, P. (2008): *Contagious: Cultures, carriers, and the outbreak narrative*, Durham and London: Duke University Press.

28. Waseem, M. and M. Aslam (2007): "Syphilis" ⟨ http://www.emedicine.com/ped/topic2193.htm ⟩. Retrieved on 1 September 2008.

29. World Development Report (1993): *Investing in Health*, The World Bank, New York: Oxford University Press.

30. 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.