

Infectious Diseases, Optimal Health Expenditures and Growth *

Aditya Goenka[†] (National University of Singapore) Lin Liu[‡] (University of Rochester)

Manh-Hung Nguyen[§]
(Toulouse School of Economics)

May 21, 2010

Preliminary Version

Abstract: This paper develops a general framework to study the economic impact of infectious diseases by integrating epidemiological dynamics into a continuous time neo-classical growth model. There is a two way interaction between the economy and the disease: the incidence of the disease affects labor supply and investment in health capital can affect the incidence and recuperation from the disease. Thus, both the disease incidence and the income levels are endogenous. The dynamics of the disease make the control problem non-convex and thus, a new existence theorem is given. We fully characterize the local dynamics of the model. There can be multiple steady states, and as the underlying parameters change there can be bifurcations. There can also be steady states where the disease is endemic but the optimal response is not to spend any resources on controlling it. We also see how the endogenous variables change as some underlying economic parameters are varied.

Keywords: Epidemiology; Infectious Disease; Economic Growth; Bifurcation; Existence of equilibrium.

JEL Classification: C61, D51, E13, O41, E32.

*We would like to thank Murali Agastya, Michele Boldrin, Russell Cooper, Atsushi Kajii, Takashi Kamihigashi, Tomoo Kikuchi, Cuong Le Van, and seminar participants at the 2007 Asian General Equilibrium Theory Workshop, Singapore; 2008 European General Equilibrium Workshop, Paestum; FEMES 2008; NUS Macro Brown Bag Workshop; University of Paris I; University of Cagliari; City University London; SWIM Auckland 2010 for helpful comments and suggestions. The usual disclaimer applies.

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

[‡]Department of Economics, Harkness Hall, University of Rochester, Rochester, NY 14627, USA. Email: linliu@rochester.edu

[§]LERNA-INRA, Toulouse School of Economics, Manufacture des Tabacs, 21 Allée de Brienne, 31000 Toulouse, France. Email: mhnguyen@toulouse.inra.fr

1 Introduction

There is a growing interest in the economics literature on the effects of infectious diseases on economic outcomes. This literature, which is largely empirical, tries to quantify the impact of infectious diseases and account for the endogeneity of both health levels and income. In this literature there is a controversy on the size of impact of diseases on income: Some papers find the effect of control of diseases to be large (Bloom, *et al* (2009)), 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)). A central problem faced in this empirical literature is the endogeneity of income and the health. However, the underlying theoretical models in these papers largely look at steady state behavior with a fixed savings rate and exogenous labor supply while varying the disease incidence and thus, do not fully capture the endogeneity of economic decisions. This paper intends to provide a canonical theoretical framework modeling the joint determination of income and disease prevalence by integrating epidemiological dynamics into a continuous time neo-classical growth model. This paper is also related to some of the theoretical literature on the optimal control of diseases which develops models to evaluate welfare gains of disease control and eradication (e.g. Gersovitz and Hammer (2004), Barrett and Hoel (2004), d’Albis and Augeraud-Véron (2008)). These papers however, model disease dynamics but not the accumulation of capital.

As we would expect savings behavior to change in response to changes in disease incidence, it is important to incorporate this into the dynamic model to be able to correctly assess the impact of diseases on capital accumulation and hence, growth and income. As the prevalence of diseases is affected by health expenditure, which is an additional decision to the investment and consumption decision, this has to be modeled as well. Without modeling both physical and health capital accumulation and the evolution of diseases at the same time, it is hard to assess whether resource allocation is optimal¹. One of the key insights of the epidemiology literature (see Anderson and May (1991), Hethcote (2000), Hethcote (2009)), which models dynamic transmission of diseases, is that changes in infectivity changes the dynamic properties of diseases. Thus, we should also know how the dynamic properties of the economic variables change as disease incidence (which is endogenous) changes. As the literature does not model both disease dynamics and capital accumulation explicitly, the existing models are like a black-box: the very details of disease transmissions and the capital accumulation process that are going to be crucial in understanding their effects and for the formulation of public policy, are obscured. This paper develops a framework where disease incidence affects labor participation and hence, savings and output. In the model, the parameters of disease transmission, which is explicitly modeled using insights from the epidemiology literature, are also affected by health capital. Thus, there is a two-way interaction between diseases and the economy. The analysis shows that multiple steady states may exist and as the underlying parameters of the economy are varied, the nature of the steady states can change, i.e., there can be bifurcations.

In order to model the disease transmission explicitly we integrate the epidemiology literature (see Anderson and May (1991), Hethcote (2000), Hethcote (2009)) into dynamic economic analysis. In this paper we examine the effect of the canonical epidemiological structure for recurring diseases - *SIS* dynamics - in a continuous time growth model. *SIS* dynamics characterize diseases where upon recovery from the disease there is no subsequent immunity to the disease. This covers many major infectious diseases such as flu, tuberculosis, malaria, dengue, schistosomiasis, trypanosomiasis (human sleeping sickness), typhoid, meningitis, pneumonia, diarrhoea, acute haemorrhagic conjunctivitis, strep throat and sexually transmitted diseases (STD) such as gonorrhoea, syphilis, etc (see Anderson and May (1991)). As mentioned above, in our model we endogenize the epidemiological parameters by making them dependent on health capital: increases in health capital reduce the infectivity rate and increase the recovery rate from the disease. However, the disease dynamics are non-convex making the use of the Arrow-Mangasarian sufficiency conditions in optimal control problems difficult to use. Gersovitz and Hammer (2004) rely on simulations to argue that the first order conditions are in fact sufficient, while d’Albis and Augeraud-Véron (2008) assume that the disease dynamics are convex so that the problem does not arise in the first place. In this paper, we address the issue directly. We show that a solution to the optimal control problem does indeed exist following the method in d’Albis *et al* (2008).

Infectious diseases affect the economy mainly through three channels: labor productivity (Thirumurthy, *et al* (2007), Weil (2007)), human capital accumulation (Bell, *et al* (2003), Bleakley (2007), Kremer and Miguel (2004)) and population size (Kalemli-Ozcan, *et al* (2000), Young (2005)). An increase in disease prevalence will decrease all three variables. A decrease in the first two will have adverse

¹The model in Delfino and Simmons (2000) is an exception but it also uses fixed savings behavior and thus does not permit welfare comparisons. It does not include health capital.

effects on economic outcomes, but a decrease in the population size may have a positive effect contingent on the dependency ratio. In the paper we focus on the effect on labor productivity². However, the equilibrium outcomes capture the second and third channels in a way different from the literature as the effects are entirely endogenous. While we do not treat fertility (net population growth rate) as endogenous, we consider the effect of an exogenous variation of the net birth rate on economic outcomes. We find that there are multiple steady states: a disease free steady state always exists. It is unique when the net birth rate is high. The basic intuition is that individuals enter the economy at a faster rate than they contract the disease so that eventually it dies out. As the net birth rate decreases, there can be a steady state where the disease is endemic but there is no expenditure on health. Here due to the relatively high birth rate, the marginal returns to investing physical capital always dominate that of human capital: The high birth rates imply that there is low per capital physical capital on the one hand and the cost of an additional worker falling ill is low. This brings out the endogenous nature of the second and third mechanisms. As the net birth rate decreases further the rate of return dominance ceases to hold and in the endemic steady state there are positive health expenditures. Further decreases in the net birth rate increase health expenditures. The intuition is that it becomes increasingly costly for society if an additional worker falls ill, and thus, social health expenditures increase. This has two implications. First, if we look at the cross section relationship between health expenditures and growth, this is positive. This has led some to suggest health is a luxury good (Hall and Jones (2005)). In our framework, both health expenditures and income are endogenous. The link between the two is the effect of birth rates, where decreases in it cause both income and health expenditures to increase. The negative relationship between birth rates and income is well known (see for example Brander and Dowrick (1994) or the recent paper by Boldrin, *et al* (2009)). Second, the effect of changes in net birth rate on the economic variables - consumption, physical and health capital, and labor is non-linear. As the changes are endogenous, looking at Solow type models can be misleading.

In this paper we abstract away from disease related mortality. This is a significant assumption 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 making this assumption. These include several strains of influenza, meningitis, STDs (syphilis, gonorrhea), dengue, conjunctivitis, strep throat, etc. 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. In the paper we also consider the effect of changes in the discount rate on the variables of interest. As has been noted in the literature, increase in mortality increases discounting. When we vary the discount rate one interpretation is that there is a change in mortality. We show that if agents are more patient, i.e. longer lived, then physical and health capital increase. Furthermore, as mentioned above we also look at changes in net birth rate but it should be kept in mind that we are not capturing disease related mortality.

The paper is organized as follows: Section 2 describes the model and in Section 3 we establish existence of an optimal solution. Section 4 studies the steady state equilibria, and Section 5 contains the stability and bifurcation analysis of how the nature of the equilibria change as parameters are varied. Section 6 does comparative statics of steady states while varying discount and birth rates, and the last section concludes.

2 The Model

In this paper we study the canonical deterministic *SIS* model which divides the total population into two classes: susceptible (*S*) and infective (*I*) (see figure 1). Individuals who are healthy but susceptible can contract the disease - becoming infected and capable of transmitting the disease to other, i.e. infective. Upon recovery, individuals do not have any disease conferred immunity, and move back to the class of susceptible individuals. This model can be applied to the infectious diseases which are absent of immunity or which mutate rapidly such that people will be susceptible to the newly mutated strains of the disease even if they have immunity to the old ones. 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. Let $S(t)$ be the number of susceptibles at time t , $I(t)$ be the number of infectives 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

²See Goenka and Liu (2009) for inclusion of learning-by-doing human capital.

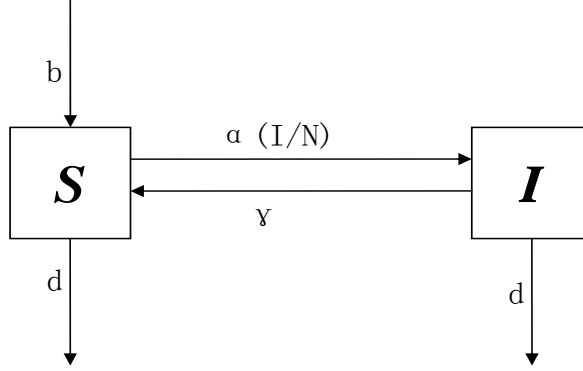


Figure 1: The transfer diagram for the SIS epidemiology model

of new cases per unit of time is $(\alpha I/N)S$. This is the standard model used in the epidemiology literature (Hethcote (2009)). 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 increases the pattern of interaction is going to be invariant. 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 $\gamma I(t)$.

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 . As mentioned above in this paper we do not study disease related mortality.

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 in the epidemiology literature is given by the following system of differential equations (Hethcote, 2009):

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

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

$$\dot{s}_t = (1 - s_t)(b + \gamma - \alpha s_t) \quad (1)$$

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 $\frac{b+\gamma}{\alpha} < 1$. Linearize the one-dimensional system around its equilibria and the Jacobians are $Ds|_{s_1^*} = \alpha - \gamma - b$ and $Ds|_{s_2^*} = \gamma + b - \alpha$. So if $b > \alpha - \gamma$ the system only has one disease-free steady state, which is stable, and if $b < \alpha - \gamma$ the system has one stable endemic steady state and one unstable disease-free steady state (refer to Figure 2). Hence, there is a bifurcation point, i.e. $b = \alpha - \gamma$, where the existence and stability of the equilibria changes. Equation (1) can be solved analytically³ and hence the dynamics we derive are global.

³Since $\dot{s}_t = (1 - s_t)(b + \gamma - \alpha s_t)$, with initial value $s_0 < 1$, is a Bernoulli differential equation, we can solve it and get an explicit unique solution: $s_t = 1 - \frac{e^{[\alpha - (\gamma + b)]t}}{\alpha - (\gamma + b) e^{[\alpha - (\gamma + b)]t} + \frac{1}{1 - s_0} - \frac{\alpha}{\alpha - \gamma - b}}$ (for $b \neq \alpha - \gamma$) and $s_t = 1 - \frac{1}{\alpha t + \frac{1}{1 - s_0}}$ (for $b = \alpha - \gamma$).

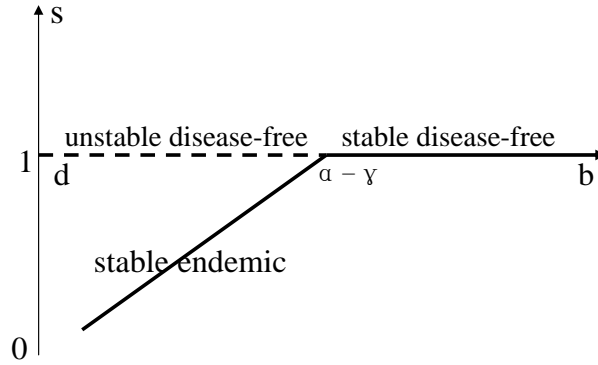


Figure 2: The bifurcation diagram for SIS model

In this paper, we endogenize the parameters α and γ in a two sector growth model. There is a population of size $N(t)$ growing over time at the rate of $b - d$. Each individual's labor is indivisible: We assume infected people cannot work and labor force consists only of healthy people with labor supplied inelastically.⁴ Thus, in time period t the labor supply is $L(t) = N(t) - I(t) = S(t)$ and hence, $L(t)$ inherits the dynamics of $S(t)$, that is,

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

in terms of the fraction of labor force $l_t = L_t/N_t$. It is unsatisfactory in an economic model that the epidemiological parameters are taken as exogenous as health expenditures may affect them⁵. Thus, we allow for health capital to affect the parameters, hence, allowing for a two-way interaction between the economy and the infectious diseases. Here we endogenize them by treating the contact rate and recovery rate as functions of health capital per capita h_t . This takes into account intervention to control the transmission of infectious diseases through their preventive or therapeutic actions. When health capital is higher people are less likely to get infected and more likely to recover from the diseases. We assume that the marginal effect diminishes as health capital increases. We further assume that the marginal effect is finite as health capital approaches zero, and so it is possible to have zero health capital when there is no infectious disease or the disease prevalence is extremely low.

Assumption 2: The epidemiological parameter functions $\alpha(h_t)$ and $\gamma(h_t): \mathfrak{R}_+ \rightarrow \mathfrak{R}_+$ satisfy:

1. $\alpha(h_t)$ is a C^∞ function with $\alpha'(h_t) \leq 0$, $\alpha''(h_t) \geq 0$, $\lim_{h_t \rightarrow 0} |\alpha'(h_t)| < \infty$, $\lim_{h_t \rightarrow \infty} \alpha'(h_t) \rightarrow 0$, $\alpha(h_t) \rightarrow \bar{\alpha}$ as $h_t \rightarrow 0$ and $\alpha(h_t) \rightarrow \underline{\alpha}$ as $h_t \rightarrow +\infty$;
2. $\gamma(h_t)$ is a C^∞ function with $\gamma'(h_t) \geq 0$, $\gamma''(h_t) \leq 0$, $\lim_{h_t \rightarrow 0} \gamma'(h_t) < \infty$, $\lim_{h_t \rightarrow \infty} \gamma'(h_t) \rightarrow 0$, $\gamma(h_t) \rightarrow \underline{\gamma}$ as $h_t \rightarrow 0$ and $\gamma(h_t) \rightarrow \bar{\gamma}$ as $h_t \rightarrow +\infty$.⁶

We assume physical goods and health are generated by different production functions. The output is produced using capital and labor, and is either consumed, invested into physical capital or spent in health expenditure. The health capital is produced only by health expenditure. For simplicity, we assume the depreciation rates of two capitals are the same and $\delta \in (0, 1)$. Thus, the physical capital k_t and health capital h_t are accumulated as follows.

$$\dot{k}_t = f(k_t, l_t) - c_t - m_t - \delta k_t - k_t(b - d) \quad (3)$$

$$\dot{h}_t = g(m_t) - \delta h_t - h_t(b - d). \quad (4)$$

The physical goods production function $f(k_t, l_t)$ and health capital production function $g(m_t)$ ⁷ are the usual neo-classical technologies. The health capital production function is increasing in health expenditure

⁴See Goenka and Liu (2010) for a model with an endogenous labor supply. This paper shows the dynamics are invariant to introduction of endogenous labor supply choice under certain conditions.

⁵The literature on rational epidemics as in Geoffard and Philipson (1996), Kremer (1996), Philipson (2000) looks at changes in epidemiology parameters due to changes in individual choices.

⁶For multiple equilibria analysis C^2 is required and for local stability and bifurcation analysis at least C^5 is required. Thus, for simplicity we assume all the functions to be smooth functions.

⁷This health capital production function could depend on physical capital as well. If we incorporate this effect, the qualitative result of this paper still hold.

but the marginal product is decreasing. The marginal product is finite as health expenditure approaches zero and so it is possible to have a corner solution with zero health expenditure.

Assumption 3: *The production function $f(k_t, l_t) : \mathfrak{R}_+^2 \rightarrow \mathfrak{R}_+$:*

1. $f(\cdot, \cdot)$ is C^∞ and homogenous of degree one;
2. $f_1 > 0, f_{11} < 0, f_2 > 0, f_{22} < 0, f_{12} = f_{21} > 0$ and $f_{11}f_{22} - f_{12}f_{21} > 0$;
3. $\lim_{k_t \rightarrow 0^+} f_1 = \infty, \lim_{k_t \rightarrow \infty} f_1 = 0$ and $f(0, l_t) = f(k_t, 0) = 0$.

Assumption 4: *The production function $g(m_t) : \mathfrak{R}_+ \rightarrow \mathfrak{R}_+$ is C^∞ with $g' > 0, g'' < 0, \lim_{m_t \rightarrow 0} g' < \infty$ and $g(0) = 0$.*

We further assume that all individuals are ex-ante identical. Utility function depends only on current consumption, c_t , is additively separable, and is discounted at the rate $\theta > 0$.

Assumption 5: *The instantaneous utility function $u(c_t) : \mathfrak{R}_+ \rightarrow \mathfrak{R}_+$ is C^∞ with $u' > 0, u'' < 0$ and $\lim_{c_t \rightarrow 0^+} u' = \infty$.*

We 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. As we look at the full insurance outcome, at time $t = 0$, the social planner maximizes $\int_0^\infty e^{-\theta t} u(c_t) dt$ ⁸ by choosing consumption c_t and health expenditure m_t , subject to the equations (2), (3) and (4), with the inequality constraints $m_t \geq 0, k_t, h_t \geq 0, 0 \leq l_t \leq 1 \forall t$ and initial conditions $k_0 > 0, h_0 \geq 0, l_0 > 0$ given. It is worthwhile noting here that we have irreversible health expenditure as it is unlikely to be true that the resource spent on public health could be recovered. For simplicity, we drop time subscript t when it is self-evident.

3 Existence of an optimal solution

We shall prove the existence of an optimal solution to the social planner problem

$$\max \int_0^\infty u(c) e^{-\theta t} dt \quad (5)$$

subject to (3), (4), (2), and

$$\begin{aligned} k &\geq 0, \quad m \geq 0, \quad h \geq 0, \quad 0 \leq l \leq 1 \\ k_0 &> 0, \quad h_0 \geq 0, \quad l_0 > 0 \text{ given.} \end{aligned} \quad (6)$$

The maximized Hamiltonian is non-concave, as the law of motion of labor is non-convex reflecting the increasing returns of infections, so that the Arrow sufficiency theorem is difficult to apply in our model⁹. The method of proof is to show that an optimal solution exists to the social planners problem by using the argument in d'Albis, et al. (2008) to show that the objective function is uniformly bounded from above on the set of feasible controls \mathcal{C} . By using the Dunford-Pettis criterion, the associated feasible sequences with \mathcal{C} (the maximizing sequences) will weakly converge in the topology $\sigma(L^1(e^{-\theta t}), L^\infty)$. This limit is feasible due to the Fatou Lemma and it will be the optimal solution of the social planner problem.

Denote $L^1(e^{-\theta t})$ the set of functions f such that $\int_0^\infty |f(t)| e^{-\theta t} dt < \infty$. The Dunford-Pettis criterion is :

⁸Alternatively instead of maximizing the representative agent's welfare we could maximize the total welfare by using $\int_0^\infty e^{-\theta t} e^{(b-d)t} N_0 u(c_t) dt$ (see the discussion in Arrow and Kurz (1970)). It is equivalent to having a lower discount factor. The qualitative results of this paper still remain although the optimal allocation may vary slightly.

⁹See Gersovitz and Hammer (2004) for more on sufficiency conditions in *SIS* dynamics models. d'Albis and Augeraud-Véron (2008) directly assume convexity of the law of motion.

Definition 1 Let B be a bounded subset of $L^1(e^{-\theta t})$. B is relative compact for the topology $\sigma(L^1, L^\infty)$ iff $\forall \varepsilon > 0, \exists \delta > 0$ such that $\int_K |f(t)| e^{-\theta t} dt < \varepsilon, \forall f \in B$ and $\forall K$ with $\int_K e^{-\rho t} dt < \delta$.

We need the following assumption:

Assumption 6: There exists $\gamma \geq 0, \xi \geq 0$ such that $-\gamma \leq \dot{k}/k, -\xi \leq \dot{h}/h$.

This assumption is automatically satisfied if the growth rates of physical capital and health capital are non-negative. If they are negative and converge, the Assumption 6 excludes the case that the growth rates of physical capital and health capital converge to $-\infty$. This assumption has been used in d'Albis, et al. (2008) and in LeVan and Vailakis (2003) in a discrete-time optimal growth model with irreversible investment.¹⁰

The sketch of the proof is as follows:

Step 1. We prove that x belong to $L^1(e^{-\theta t})$ for every variable x and $|\dot{x}|$ belong to $L^1(e^{-\theta t})$ if \dot{x} appears in the model. (assumptions A3, A4 in d'Albis et al. (2008)).

Indeed, since $\lim_{k \rightarrow \infty} f_1(k, l) = 0$, for any $\zeta \in (0, \theta)$ there exist a constant B such that $f(k, 1) \leq B + \zeta k$. If not, let $k = x + \epsilon$ where ϵ is a positive small real number and we suppose that $f(x + \epsilon, 1) > B + \zeta(x + \epsilon)$ for any B . Let $B = f(x, 1) - \zeta x$, we have $\frac{f(x + \epsilon, 1) - f(x, 1)}{\epsilon} > \zeta$. Taking the limit as $\epsilon \rightarrow 0$ we have $f_1(x, 1) > \zeta > 0 \forall x$. This implies $\lim_{x \rightarrow \infty} f_1(x, 1) \geq \zeta > 0$ which is a contradiction. Hence, we have

$$f(k, l) \leq f(k, 1) \leq B + \zeta k. \quad (7)$$

Since $\dot{k} = f(k, l) - c - m - k(\delta + b - d)$, it follows that

$$\dot{k} \leq f(k, l) \leq B + \zeta k.$$

Multiply by $e^{-\zeta \tau}$ we get $e^{-\zeta \tau} \dot{k} - \zeta k e^{-\zeta \tau} \leq B e^{-\zeta \tau}$. Thus,

$$e^{-\zeta t} k = \int_0^t \frac{\partial(e^{-\zeta \tau} k)}{\partial \tau} d\tau \leq \int_0^t B e^{-\zeta \tau} d\tau = \frac{-B e^{-\zeta t}}{\zeta} + \frac{B}{\zeta}.$$

This implies

$$k \leq \frac{-B}{\zeta} + \frac{B e^{\zeta t}}{\zeta}.$$

Thus, there exists a constant B' such that

$$k \leq B' e^{\zeta t}. \quad (8)$$

Therefore, note that $\zeta < \theta$,

$$\int_0^\infty k e^{-\theta t} dt \leq \int_0^\infty B' e^{(\zeta - \theta)t} dt < +\infty.$$

Moreover, since $-\dot{k} \leq k\gamma$ and $\dot{k} \leq B + \zeta k \leq B + \zeta B' e^{\zeta t}$ there exists a constant B'' such that $|\dot{k}| \leq B'' e^{\zeta t}$. Thus

$$\int_0^\infty |\dot{k}| e^{-\theta t} dt < \int_0^\infty B'' e^{(\zeta - \theta)t} dt < +\infty.$$

Because $-\dot{k} \leq k\gamma$ and $c = f(k, l) - \dot{k} - m - \delta k - k(b - d)$, it follows from (7) and (8) that

$$\begin{aligned} c &\leq f(k, l) + k(\gamma - \delta - b + d) \\ &\leq B + (\gamma - \delta - b + d + \zeta)k \\ &\leq B + (\gamma - \delta - b + d + \zeta)B' e^{\zeta t}. \end{aligned}$$

Thus, we can choose a constant B''' large enough such that

$$c \leq B''' e^{\zeta t}$$

¹⁰LeVan and Vailakis (2003) assume $0 \leq (1 - \delta)k_t \leq k_{t+1}$, and thus, $-\delta \leq (k_{t+1} - k_t)/k_t$ where $\delta > 0$ is a physical depreciation rate.

which implies

$$0 \leq \int_0^\infty ce^{-\theta t} dt \leq \int_0^\infty B''' e^{(\zeta-\theta)t} dt < +\infty.$$

Similarly, we can prove that $0 \leq \int_0^\infty me^{-\theta t} dt < +\infty$, i.e, m belongs to $L^1(e^{-\theta t})$.

By the similar arguments, it follows from (4),(2) and Assumptions 4, 6 that $|\dot{h}|, h, l$ belong to the space $L^1(e^{-\theta t})$.

Now we prove $|\dot{l}| \in L^1(e^{-\theta t})$. Since $0 \leq l \leq 1$, we have

$$\begin{aligned} |\dot{l}| &\leq b + |\gamma(h)| + |\alpha(h)| \\ &\leq b + |\gamma(h)| + |\alpha(0)|. \end{aligned}$$

Since $\lim_{h \rightarrow \infty} \gamma'(h) \rightarrow 0$, there exists a constant B'''' such that $\gamma(h) \leq B'''' + \zeta h$ where $\zeta \in (0, \theta)$. Thus, by the same argument as $k \in L^1(e^{-\theta t})$, since $h \in L^1(e^{-\theta t})$ we have $|\dot{l}| \in L^1(e^{-\theta t})$.

Step 2. We now use the results of d'Albis, et al. (2008).

Our assumption on the continuity of $u(c)$ implies, by Lemma 1 in d'Albis et al. (2008), $\int_0^\infty u(c)e^{-\theta t} dt$ is upper semicontinuous for the topology $\sigma(L^1(e^{-\theta t}), L^\infty)$. Moreover, since assumptions A3, A4 in d'Albis, et al. (2008) are satisfied in our model, $\int_0^\infty u(c)e^{-\theta t} dt$ is uniformly bounded from above on the set of feasible control \mathcal{C} . Let us consider feasible sequence $c(n)$ satisfies $\lim_{n \rightarrow \infty} \int_0^\infty u(c(n))e^{-\theta t} dt \stackrel{def}{=} \sup \int_0^\infty u(c)e^{-\theta t} dt$. Let $k(n), h(n), m(n), l(n)$ denote the feasible sequences associated with $c(n)$. In the step 1, we know that if $k(n), h(n), m(n), l(n)$ are feasible from $k_0 > 0, h_0 \geq 0, l_0 > 0$ then they belong to the space $L^1(e^{-\theta t})$. Thus, it follows from Corollary 11 in Dunford and Schwartz (1967), $c(n)$ and all associated feasible variables satisfy the Dunford-Pettis criterion and they have subsequences which weakly converge to the limit points in $L^1(e^{-\theta t})$. Denote c^* the limit of a subsequence of $c(n)$, denoted also $c(n)$ for simplicity. Since $\int_0^\infty u(c)e^{-\theta t} dt$ is upper semicontinuous for the topology $\sigma(L^1(e^{-\theta t}), L^\infty)$ we have $\int_0^\infty u(c^*)e^{-\theta t} dt \geq \lim_{n \rightarrow \infty} \int_0^\infty u(c(n))e^{-\theta t} dt = \sup \int_0^\infty u(c)e^{-\theta t} dt$. On the other hand, these limits sequences are feasible due to Fatou Lemma (see Theorem 1 in d'Albis, et al. (2008)). Therefore, they are optimal solutions of our problem.

Theorem 2 *Under Assumptions A.1-A.6, there exists a solution to the social planner's problem.*

4 Multiplicity of Steady State Equilibria

To analyze the equilibria, we look at the first order conditions to the optimal solution. This is valid as we know that these conditions are necessary and a solution exists, and thus a solution must satisfy these conditions. Note that we allow for corner solutions. As we will see that for some parameters there is a unique (steady state) solution to the first order conditions. For others there are multiple equilibria with one being a corner solution. In the next section, we study (local) stability properties of the different equilibria so that if we have an solution to the necessary first order conditions we can see how these evolve.

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 $l = 0$. The constraint $h \geq 0$ can be inferred from $m \geq 0$, and hence can be ignored. Now consider the central planner's maximization problem with irreversible health expenditure and inequality constraint $l \leq 1$. The current value Lagrangian for the optimization problem above is:

$$\begin{aligned} \mathcal{L} = & u(c) + \lambda_1(f(k, l) - c - m - \delta k - k(b - d)) + \lambda_2(g(m) - \\ & - \delta h - h(b - d)) + \lambda_3(1 - l)(b + \gamma(h) - \alpha(h)l) + \mu_1(1 - l) + \mu_2 m \end{aligned}$$

where λ_1, λ_2 and λ_3 are costate variables and μ_1 and μ_2 are the Lagrange multipliers for $l \leq 1$ and $m \geq 0$.

F.O.Cs, Kuhn-Tucker conditions and transversality conditions are given by

$$c : \quad u'(c) = \lambda_1, \quad (9)$$

$$m : \quad m(\lambda_1 - \lambda_2 g') = 0 \quad m \geq 0 \quad \lambda_1 - \lambda_2 g' \geq 0 \quad (10)$$

$$k : \quad \dot{\lambda}_1 = -\lambda_1(f_1 - \delta - \theta - (b - d)) \quad (11)$$

$$h : \quad \dot{\lambda}_2 = \lambda_2(\delta + \theta + (b - d)) - \lambda_3(1 - l)(\gamma' - \alpha' l) \quad (12)$$

$$l : \quad \dot{\lambda}_3 = -\lambda_1 f_2 + \lambda_3(\theta + b + \gamma + \alpha - 2\alpha l) + \mu_1 \quad (13)$$

$$\mu_1 \geq 0 \quad 1 - l \geq 0 \quad \mu_1(1 - l) = 0 \quad (14)$$

$$\lim_{t \rightarrow \infty} e^{-\theta t} \lambda_1 k = 0 \quad \lim_{t \rightarrow \infty} e^{-\theta t} \lambda_2 h = 0 \quad \lim_{t \rightarrow \infty} e^{-\theta t} \lambda_3 l = 0. \quad (15)$$

The system dynamics are given by equations (2)-(4) and (9)-(15). If x is a variable, we use x^* to denote its steady state value. From $\dot{l} = 0$ we have either $l^* = 1$ (the disease-free case) or $l^* = \frac{\gamma(h)+b}{\alpha(h)}$ (the endemic case). For the disease free steady state $l^* = 1$, $\dot{\lambda}_2 = \lambda_2(\delta + b - d + \theta) = 0$. So $\lambda_2^* = 0$. Moreover $\lambda_1^* - \lambda_2^* g' = u'(c^*) > 0$ since by assumption g' is finite, equation (10) implies $m^* = 0$. Since $g(0) = 0$, we have $h^* = 0$ from equation (4). Hence, the epidemiology parameters take the values $\bar{\alpha}, \underline{\gamma}$. Thus, there is one disease-free steady state in which the disease is completely eradicated and there is no need for any health expenditure or health capital. In this case, the model reduces to the standard neo-classical growth model for the economic variables and to the mathematical biology model with exogenous parameters. Note that the disease-free steady state exists for all parameter values.

Proposition 1 *Under A.1–A.6, there always exists a unique disease-free steady state, which degenerates to the standard neo-classical growth model with $l^* = 1$, $m^* = 0$, $h^* = 0$, and k^* and c^* determined by:*

$$\begin{aligned} f_1(k^*, 1) &= \delta + \theta + b - d \\ f(k^*, 1) &= c^* + \delta k^* + k^*(b - d). \end{aligned}$$

For the endemic case, that is, $l^* = \frac{\gamma(h)+b}{\alpha(h)} < 1$, $\mu_1 = 0$. Since in a steady state, shadow prices are constant, $\dot{\lambda}_2 = 0$ and $\dot{\lambda}_3 = 0$. We have:

$$\lambda_2^* = \frac{u'(c^*) f_2(k^*, l^*) (1 - l^*) (\gamma'(h^*) - \alpha'(h^*) l^*)}{f_1(k^*, l^*) (\theta + \alpha(h^*) - b - \gamma(h^*))}$$

From equation (10) we know $\lambda_1 \geq \lambda_2 g'$ has to be satisfied, which is equivalent to:

$$f_1(k^*, l^*) \geq f_2(k^*, l^*) \frac{(1 - l^*) (\gamma'(h^*) - \alpha'(h^*) l^*)}{\theta + \alpha(h^*) - b - \gamma(h^*)} g'(m^*) \quad (16)$$

The left hand side of equation (16) is the marginal productivity of physical capital at an endemic steady state while the right hand side is marginal productivity of health capital. To see this the last term on the right hand side is the marginal productivity of health expenditure, the second term can be interpreted as the marginal contribution of health capital on labor supply, and the first term is the marginal productivity of labor. Denote the second term as $l'_\theta(h) := \frac{(1 - l)(\gamma'(h) - \alpha'(h)l)}{(\theta + \alpha(h) - b - \gamma(h))}$. Using $l^* = \frac{\gamma(h)+b}{\alpha(h)}$, in steady state this reduces to $l'_\theta(h) = -\frac{(\alpha'(\gamma + b) - \gamma'\alpha)(\alpha - (\gamma + b))}{\alpha^2(\alpha - (\gamma + b) + \theta)} > 0$. Equation (16) means if marginal productivity of physical capital is at least as high as marginal productivity of health capital, there will be no health expenditure. Otherwise health expenditure is positive.

From equation (10), there are two cases: $m^* = 0$ and $m^* > 0$. The first is termed as the endemic steady state without health expenditure and the second the endemic steady state with health expenditure. First, look at the case $m^* = 0$. $\dot{h} = 0$ implies $h^* = 0$ and thus, define $\underline{l} := \frac{b+\gamma}{\alpha}$. k^* is uniquely determined by:

$$f_1(k, \underline{l}) = \delta + b - d + \theta, \quad (17)$$

for each fixed \underline{l} due to the assumption 2. Since $m^* = 0$ and $h^* = 0$, equation (16) reduces to

$$f_1(k^*, \underline{l}) \geq f_2(k^*, \underline{l}) \frac{(1 - \underline{l})(\gamma'(0) - \alpha'(0)\underline{l})}{(\theta + \bar{\alpha} - b - \underline{\gamma})} g'(0). \quad (18)$$

Hence, this steady state exists only when marginal productivity of physical capital is no less than marginal productivity of health capital in steady state. In other words, despite the prevalence of the disease, if marginal productivity of physical capital investment is greater than marginal productivity of health capital, they will not invest in health. Thus, the prevalence of the disease is not sufficient (from purely an economic point of view) to require health expenditures. It is conceivable that in several situations this is indeed the case, and thus countries find it optimal to spend no resources on disease control. Moreover, the constraint on m is binding in the endemic steady state without health expenditure, and this exists only when $\underline{l} < 1$, that is,

$$d \leq b < \bar{\alpha} - \gamma. \quad (19)$$

As we treat the birth rate b as the varying parameter, we need to find a range for b such that equation (18) and (19) are both satisfied, i.e. there exists such endemic steady state without health expenditure. Rewrite equation (18) as

$$f_1(k^*, \underline{l})/f_2(k^*, \underline{l}) \geq g'(0)l'_\theta(0). \quad (20)$$

Note here k^* is a function of b and given by equation (17). From equation (17), we know $\frac{\partial k^*}{\partial b} = \frac{1 - f_{12}/\bar{\alpha}}{f_{11}}$.

Then

$$\begin{aligned} \frac{\partial [f_1(k^*, \underline{l})/f_2(k^*, \underline{l})]}{\partial b} &= \frac{(f_{11}f_2 - f_{12}f_1)\frac{\partial k^*}{\partial b} + (f_{12}f_2 - f_{11}f_{22})\frac{1}{\bar{\alpha}}}{f_2^2} \\ &= \frac{f_{11}f_2 - f_{12}f_1 + f_1(f_{12}f_{21} - f_{11}f_{22})/\bar{\alpha}}{f_{11}f_2^2} > 0. \end{aligned}$$

So the L.H.S. of equation (20) is an increasing function in b and it is strictly greater than 0. Furthermore

$$\frac{\partial [g'(0)l'_\theta(0)]}{\partial b} = g'(0) \left[-\frac{\alpha'(0)}{\alpha^2} + \frac{\theta(\theta\alpha'(0) + \bar{\alpha}(\alpha'(0) - \gamma'(0)))}{\alpha^2(\bar{\alpha} - \gamma - b + \theta)^2} \right].$$

Notice $\frac{\partial [g'(0)l'_\theta(0)]}{\partial b}$ decreases as b rises and $\lim_{b \rightarrow \bar{\alpha} - \gamma} \frac{\partial [g'(0)l'_\theta(0)]}{\partial b} = g'(0) \frac{\alpha'(0) - \gamma'(0)}{\alpha\theta} < 0$. It means the slope of the curve of R.H.S. of equation (20) is a decreasing function of b , and as b approaches $\bar{\alpha} - \underline{\gamma}$ the slope is negative. Moreover we have $\lim_{b \rightarrow \bar{\alpha} - \underline{\gamma}} g'(0)l'_\theta(0) = 0$.

Assumption 7: $\lim_{b \rightarrow d} g'(0)l'_\theta(0) > f_1(\hat{k}, \frac{d+\gamma}{\alpha})/f_2(\hat{k}, \frac{d+\gamma}{\alpha})$, where \hat{k} is determined by $f_1(\hat{k}, \frac{d+\gamma}{\alpha}) = \delta + \theta$.

Take the limit $b \rightarrow d$ on both sides of equation (20), R.H.S. is greater than L.H.S under assumption 7. So we can always find \hat{b} s.t. equation (20) holds at equality.

Proposition 2 *Under A.1 – A.7, there exists a unique endemic steady state without health expenditure whenever b lies within $[\hat{b}, \bar{\alpha} - \underline{\gamma}]$. And we have $m^* = 0$, $h^* = 0$, $l^* = \underline{l}$, and k^* , c^* determined by:*

$$\begin{aligned} f_1(k^*, \underline{l}) &= \delta + \theta + b - d \\ f(k^*, \underline{l}) &= c^* + \delta k^* + k^*(b - d). \end{aligned}$$

The endemic steady state without health expenditure is the same as a neo-classical steady state but with only a smaller labor force. Thus, there is lower consumption and production in the steady state. This will correspond to a Solow type steady state (savings rate constant) but just varying the labor supply which have been used in the literature (e.g. Young (2005)).

Now consider the endemic steady state with health expenditure, where $l^*(h) = \frac{\gamma(h)+b}{\alpha(h)} < 1$ with $\frac{\partial l^*(h)}{\partial h} = \frac{\gamma'\alpha - (\gamma+b)\alpha'}{\alpha^2}$ and $m^*(h) > 0$ given by $g(m) - \delta h - (b-d)h = 0$ with $\frac{\partial m^*(h)}{\partial h} = \frac{\delta+b-d}{g'(m)} > 0$. From equation (10), $\lambda_1 = \lambda_2 g'$ and equation (16) holds at equality. It implies marginal productivity of physical capital equals to marginal productivity of health capital. Moreover $k^*(h)$ is determined by

$$f_1(k, l^*(h)) = \delta + b - d + \theta,$$

that is, at the steady state marginal productivity of physical capital equals to the marginal cost. Since f_1 is strictly decreasing and lies in $(0, +\infty)$ for each $l^*(h)$, we can always find a unique $k^*(h)$ to satisfy the above equations. And $\frac{\partial k^*(h)}{\partial h} = -f_{12} \frac{\partial l^*(h)}{\partial h} / f_{11} > 0$.

So we only need to find a condition such that we can always find the solution h^* to the following equation:

$$f_2(k^*(h), l^*(h))g'(m^*(h)) \frac{(1-l^*(h))(\gamma'(h) - \alpha'(h)l^*(h))}{\theta + \alpha(h) - b - \gamma(h)} = \delta + b - d + \theta \quad (21)$$

Since $\lim_{h \rightarrow \infty} f_2g'(m)l'_\theta(h) = 0$ and $\lim_{h \rightarrow 0} f_2g'(m)l'_\theta(h) = f_2(k^*, l)g'(0)l'_\theta(0) > f_1(k^*, l) = \delta + b - d + \theta$ if $b \in [d, \hat{b})$ under A.7, equation (21) always has a solution. That is, under A.1-A.7 there always exists endemic steady state with health expenditure.

Moreover since $\frac{\partial f_2(k^*(h), l^*(h))}{\partial h} = \frac{f_{11}f_{22} - f_{12}f_{21}}{f_{11}} \frac{\partial l^*(h)}{\partial h} < 0$ and $\frac{\partial g'(m^*(h))}{\partial h} = g'' \frac{\partial m^*(h)}{\partial h} < 0$, $f_2(k^*(h), l^*(h))g'(m^*(h))$ is decreasing as h increases. If we impose additional assumption:

Assumption 8: $\alpha(\alpha''(\gamma + b) - \gamma''\alpha) > 2\alpha'(\alpha'(\gamma + b) - \gamma'\alpha)$.

Under A.2 and A.8 we can show

$$l''_\theta(h) = - \frac{(\alpha - \gamma - b + \theta)(\alpha - \gamma - b)[\alpha(\alpha''(\gamma + b) - \gamma''\alpha) - 2\alpha'(\alpha'(\gamma + b) - \gamma'\alpha)] + \alpha\theta(\alpha'(\gamma + b) - \gamma'\alpha)(\alpha' - \gamma')}{\alpha^3(\alpha - \gamma - b + \theta)^2} < 0$$

This strong assumption implies diminishing marginal productivity of health capital in controlling infectious diseases. So R.H.S. of equation (21) decreases as h increases and there exists a unique endemic steady state with health expenditure.

Proposition 3 *Under A.1 – A.8, there exists a unique endemic steady state with health expenditure whenever b lies within $[d, \hat{b})$. And $l^* = \frac{\gamma(h^*) + b}{\alpha(h^*)}$, and k^* , h^* , m^* and c^* determined by:*

$$\begin{aligned} f_1(k^*, l^*) &= \delta + \theta + b - d \\ f_2(k^*, l^*)g'(m^*) \frac{(1-l^*)(\gamma'(h^*) - \alpha'(h^*)l^*)}{\theta + \alpha(h^*) - b - \gamma(h^*)} &= \delta + \theta + b - d \\ f(k^*, l^*) &= c^* + m^* + \delta k^* + k^*(b - d) \\ g(m^*) &= (\delta + b - d)h^*. \end{aligned}$$

By investing in health expenditure we are able to control infectious disease since $l'_\theta(h)$ is positive. Compared with the disease-free case the economy has lower physical capital and a smaller labor force. The production will be lower, and there is expenditure allocated for health expenditure. Thus, clearly the consumption will be lower. However, the “savings rate” will change, as there will be expenditures on both physical and health capital.

5 Local Stability and Bifurcation

The dynamical system is given by equations (2)- (4), (9)- (15) and there are three equilibria. In order to examine their stability we linearize the system around each of the steady states. To simplify the exposition we make the following assumption.

Assumption 9: *The instantaneous utility function is in CES form: $u(c) = \frac{c^{1-\sigma} - 1}{1-\sigma}$.*

Substituting $\lambda_1 = u'(c) = c^{-\sigma}$ into equation (11), we get

$$\dot{c} = \frac{c}{\sigma}(f_1 - \delta - \theta - (b - d)). \quad (22)$$

5.1 The Disease-Free Case

At the disease-free steady state, $\lambda_1 > \lambda_2 g'$. Since all the functions in this model are smooth functions, by continuity there exists a neighborhood of the steady state such that the above inequality still holds. Thus, $m^* = 0$ in this neighborhood. Intuitively around the steady state the net marginal benefit of health investment is negative: the disease is eradicated and health investment only serves to reduce physical capital accumulation and hence, lower levels of consumption, and thus no resources are spent on eradicating diseases. As $m = 0$ in this steady state, we have a maximization problem with only one choice variable - consumption. The dynamic system is reduced to:

$$\begin{aligned}\dot{k} &= f(k, l) - c - \delta k - k(b - d) \\ \dot{h} &= -\delta h - h(b - d) \\ \dot{l} &= (1 - l)(b - \alpha(h)l + \gamma(h)) \\ \dot{c} &= \frac{c}{\sigma}(f_1 - \delta - \theta - (b - d)),\end{aligned}$$

with three state variables and one choice variable. This can also be simply derived by substituting $m = 0$ into the original dynamic system. By linearizing the system around the steady state, we have:

$$\mathcal{J}_1 = \begin{pmatrix} \theta & 0 & f_2^* & -1 \\ 0 & -\delta - (b - d) & 0 & 0 \\ 0 & 0 & \bar{\alpha} - (\underline{\gamma} + b) & 0 \\ \frac{c^*}{\sigma} f_{11}^* & 0 & \frac{c^*}{\sigma} f_{12}^* & 0 \end{pmatrix}.$$

The eigenvalues are $\Lambda_1 = -\delta - (b - d) < 0$, $\Lambda_2 = \frac{\theta - \sqrt{\theta^2 - 4c^* f_{11}^* / \sigma}}{2} < 0$, $\Lambda_3 = \frac{\theta + \sqrt{\theta^2 - 4c^* f_{11}^* / \sigma}}{2} > 0$, and $\Lambda_4 = \bar{\alpha} - (\underline{\gamma} + b)$. The sign of Λ_4 depends on b . We notice if $b = \bar{\alpha} - \underline{\gamma}$, \mathcal{J}_1 has a single zero eigenvalue. Thus, we have a non-hyperbolic steady state and a bifurcation may arise. In other words, the disease-free steady state possesses a 2-dimensional local invariant stable manifold, a 1-dimensional local invariant unstable manifold and 1-dimensional local invariant center manifold. In general, however, the behavior of trajectories in center manifold cannot be inferred from the behavior of trajectories in the space of eigenvectors corresponding to the zero eigenvalue. Thus we shall take a close look at the flow in the center manifold. As the zero eigenvalue comes from dynamics of l , and the dynamics of l and h are independent from the rest, we could just focus on the dynamics of l and h . By taking b as bifurcation parameter and following the procedures given by Wiggins (2002) and Kribs-Zaleta (2003), we are able to calculate the dynamics on the center manifold (See the Appendix for details):

$$\dot{z} = \bar{\alpha}z \left(z - \frac{1}{\bar{\alpha}} \tilde{b} \right). \quad (23)$$

The fixed points of (23) are given by $z = 0$ and $z = \frac{1}{\bar{\alpha}} \tilde{b}$, and plotted in figure 3. We can see the dynamics on the center manifold exhibits a transcritical bifurcation at $\tilde{b} = 0$. Hence, for $\tilde{b} < 0$, there are two fixed points; $z = 0$ is unstable and $z = \frac{1}{\bar{\alpha}} \tilde{b}$ is stable. These two fixed points coalesce at $\tilde{b} = 0$, and for $\tilde{b} > 0$, $z = 0$ is stable and $z = \frac{1}{\bar{\alpha}} \tilde{b}$ is unstable. Thus, an exchange of stability occurs at $\tilde{b} = 0$, i.e., $b = \bar{\alpha} - \underline{\gamma}$. Therefore, for the original dynamical system if $b > \bar{\alpha} - \underline{\gamma}$, there is a 3-dimensional stable manifold and a 1-dimensional unstable manifold, and if $b < \bar{\alpha} - \underline{\gamma}$, there is a 2-dimensional stable manifold and 2-dimensional unstable manifold. Moreover, while physical capital, health capital and labor force are given at any point in time, the consumption can jump. Thus, if $b > \bar{\alpha} - \underline{\gamma}$, the system is locally saddle stable and has a unique stable path; and if $b < \bar{\alpha} - \underline{\gamma}$, the system is locally unstable.

5.2 The Endemic Case Without Health Expenditures

For the endemic steady state with no health expenditures, $\lambda_1 \geq \lambda_2 g'$ and $m^* = 0$. By continuity, this will also hold in a small neighborhood of the steady state. Thus, it is similar to the disease-free case except that $l^* < 1$. Linearize the system around the steady state:

$$\mathcal{J}_2 = \begin{pmatrix} \theta & 0 & f_2^* & -1 \\ 0 & -\delta - (b - d) & 0 & 0 \\ 0 & (1 - l^*)(\gamma'^* - \alpha'^* l^*) & \bar{\alpha} - (\underline{\gamma} + b) & 0 \\ \frac{c^*}{\sigma} f_{11}^* & 0 & \frac{c^*}{\sigma} f_{12}^* & 0 \end{pmatrix}.$$

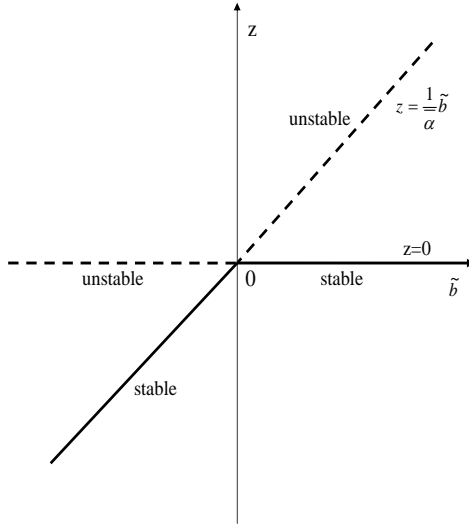


Figure 3: The transcritical bifurcation diagram

The eigenvalues are $\Lambda_1 = -\delta - (b - d) < 0$, $\Lambda_2 = \frac{\theta - \sqrt{\theta^2 - 4c^* f_{11}/\sigma}}{2} < 0$, $\Lambda_3 = \frac{\theta + \sqrt{\theta^2 - 4c^* f_{11}/\sigma}}{2} > 0$, and $\Lambda_4 = (\underline{\gamma} + b) - \bar{\alpha} < 0$. So it has 3-dimensional stable manifold and 1-dimensional unstable manifold. Since the system has three state variables and one choice variable, it is locally saddle stable and has a unique stable path. Moreover, this corresponds to the stable steady state $z = \frac{1}{\alpha} \tilde{b}$ when $\tilde{b} < 0$ in figure 3. This also explains why when \tilde{b} decreases and crosses 0, the stable disease-free steady state undergoes a bifurcation into one unstable disease-free steady state and one stable endemic steady state without health expenditure.

5.3 The Endemic Case With Health Expenditures

For the endemic case with health expenditures, the dynamical system is given by equations (2)- (4), (9)-(15) with $\lambda_1 = \lambda_2 g'$, $m^* > 0$ and $l^* < 1$. By $\lambda_1 = \lambda_2 g' + \lambda_2 g'' \dot{m}$, the system reduces to:

$$\begin{aligned}
 \dot{k} &= f(k, l) - c - m - \delta k - k(b - d) \\
 \dot{h} &= g(m) - \delta h - h(b - d) \\
 \dot{l} &= (1 - l)(b + \gamma(h) - \alpha(h)l) \\
 \dot{c} &= \frac{c}{\sigma}(f_1 - \delta - (b - d) - \theta) \\
 \dot{m} &= (c^\sigma \lambda_3 g'(m)(1 - l)(\gamma' - \alpha'l) - f_1) \frac{g'(m)}{g''(m)} \\
 \dot{\lambda}_3 &= -c^{-\sigma} f_2 + \lambda_3 \theta - \lambda_3 (2\alpha(h)l - b - \gamma(h) - \alpha(h)).
 \end{aligned}$$

We now have a higher dimensional system than the earlier two cases as $m > 0, h > 0$. Linearizing around the equilibrium the Jacobian is given by:

$$\mathcal{J}_3 = \begin{pmatrix} \theta & 0 & f_2^* & -1 & -1 & 0 \\ 0 & q_1 & 0 & 0 & g'^* & 0 \\ 0 & q_2 & q_3 & 0 & 0 & 0 \\ q_4 & 0 & q_5 & 0 & 0 & 0 \\ q_6 & q_7 & q_8 & q_9 & f_1^* & q_{10} \\ q_{11} & q_{12} & q_{13} & q_{14} & 0 & q_{15} \end{pmatrix} \quad (24)$$

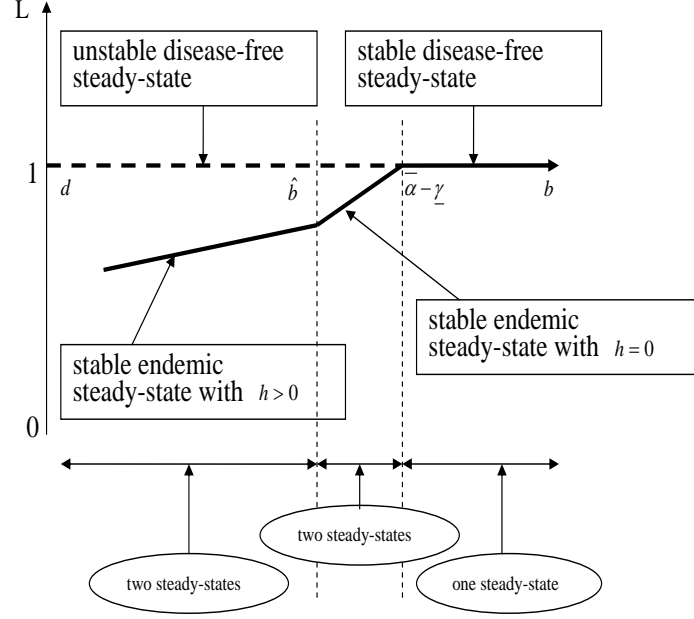


Figure 4: The local stability and bifurcation diagram

where

$$\begin{aligned}
q_1 &= -\delta - (b - d), & q_2 &= (1 - l^*)(\gamma'^* - \alpha'^* l^*), & q_3 &= b + \gamma^* - \alpha^*, & q_4 &= c^* f_{11}^* / \sigma \\
q_5 &= c^* f_{12}^* / \sigma, & q_6 &= -f_{11}^* \frac{g'^*}{g''^*}, & q_7 &= \frac{f_1^* (\gamma''^* - \alpha''^* l^*)}{\gamma'^* - \alpha'^* l^*} \frac{g'^*}{g''^*}, \\
q_8 &= \left(\frac{f_1^* (2\alpha'^* l^* - \alpha'^* - \gamma'^*)}{(1 - l^*)(\gamma'^* - \alpha'^* l^*)} - f_{12}^* \right) \frac{g'^*}{g''^*}, & q_9 &= \frac{\sigma f_1^*}{c^*} \frac{g'^*}{g''^*}, & q_{10} &= \frac{f_1^*}{\lambda^3} \frac{g'^*}{g''^*} \\
q_{11} &= -\frac{f_{12}^*}{c^* \sigma}, & q_{12} &= -\lambda_3^* (2\alpha'^* l^* - \gamma'^* - \alpha'^*), & q_{13} &= -\frac{f_{22}^*}{c^* \sigma} - 2\lambda_3^* \alpha^*, & q_{14} &= \frac{\sigma f_2^*}{c^* \sigma + 1}, & q_{15} &= \frac{f_2^*}{c^* \sigma \lambda_3^*}.
\end{aligned}$$

Since the Jacobian matrix is high dimensional, it is difficult to solve for the eigenvalues analytically. We specify functional forms and parameter values to calculate eigenvalues numerically. How we choose functional forms and parameter values are discussed more in details in section 6.2. We get three positive eigenvalues and three negative eigenvalues which shows that the system is saddle-point stable. Robustness of the result is checked for a wide range of parameters.

The local stability and bifurcation of the dynamic system are summarized in Figure 4. The solid line represents the equilibrium which is locally stable, while the dashed line means the equilibrium is locally unstable. So when birth rate b is very high, which is greater than $\bar{\alpha} - \underline{\gamma}$, there is only one disease-free steady state which is locally stable. This is the case the birth rate is relatively high and the disease is eradicated in the long run. When b decreases to exactly $\bar{\alpha} - \underline{\gamma}$, the stable disease-free equilibrium goes through a transcritical bifurcation to two equilibria: one is the unstable disease-free steady state and the other is the stable endemic steady state without health expenditure. We further show these two equilibria coexist when $b \in [\hat{b}, \bar{\alpha} - \underline{\gamma})$. If $b \in [d, \hat{b})$, the endemic steady state with no health expenditures disappears as when birth rate becomes extremely low and diseases prevalence becomes worse, it is always optimal to invest in health. Thus, the system has one unstable disease-free equilibrium and one stable endemic equilibrium with health expenditure.

6 Comparative Statics

We now explore how the steady state properties of the model change as the parameters are varied. Here we only examine the endemic steady states with or without health expenditure because as mentioned above, the disease-free case reduces to the standard neo-classical model.

6.1 The discount factor θ

In the endemic case without health expenditure,

$$\frac{dk^*}{d\theta} = \frac{1}{f_{11}} < 0, \quad \text{and} \quad \frac{dc^*}{d\theta} = \frac{\theta}{f_{11}} < 0.$$

The disease prevalence $l^* = \frac{\gamma+b}{\alpha}$ remains unchanged.

In the endemic case with health expenditure, we have $\frac{\partial m}{\partial h} = \frac{\delta+(b-d)}{g'(m)} > 0$ and $\frac{\partial l'_\theta(h)}{\partial \theta} = \frac{-l'_\theta(h)}{\alpha(h)-(\gamma+b)+\theta} < 0$. Let $D = f_{11}(f_{22}g'(m)l'l'_\theta + f_2g'(m)l''_\theta + f_2g''(m)\frac{\partial m}{\partial h}l'_\theta) - f_{12}l'f_{21}l'_\theta g' > 0$. By the multi-dimensional implicit function theorem, we have:

$$\begin{aligned} \frac{dk^*}{d\theta} &= \frac{1}{D}(f_{22}g'(m)l'l'_\theta + f_2g'(m)l''_\theta + f_2g''(m)\frac{\partial m}{\partial h}l'_\theta - f_{12}l'(1 - f_2g'\frac{\partial l'_\theta}{\partial \theta})) < 0, \\ \frac{dh^*}{d\theta} &= \frac{1}{D}(f_{11}(1 - f_2g'\frac{\partial l'_\theta}{\partial \theta}) - f_{21}g'(m)l'_\theta) < 0, \\ \text{and thus } \frac{dl^*}{d\theta} &= l'\frac{dh^*}{d\theta} < 0, \\ \frac{dc^*}{d\theta} &= (f_1 - \delta_k - (b-d))\frac{dk^*}{d\theta} + (f_2l' - \delta_h - (b-d))\frac{dh^*}{d\theta} < 0. \end{aligned}$$

Therefore in the endemic steady state without health expenditure variation in discount factor has no effect on the spread of infectious diseases, since without health expenditure the mechanism of disease spread is independent of individual's behavior. The smaller discount factor only leads to higher physical capital and consumption in exactly the same way as the change in the neo-classical model. In the endemic steady state with health expenditure, as the discount rate decreases, that is as the people become more patient, they tend to spend more resources in preventing from being infected or getting better treatment. Hence, the rise in health capital leads to a larger labor force, and both physical capital and consumption will increase.

6.2 The birth rate b

The other two parameters are the death rate d and the birth rate b . As they enter in difference, we look at variations in b . In the endemic case without health expenditure,

$$\frac{dl^*}{db} = \frac{1}{\alpha} > 0, \quad \frac{dk^*}{db} = \underbrace{\frac{1}{f_{11}}}_{-} + \underbrace{\frac{f_{12}}{-\alpha f_{11}}}_{+} \quad ?, \quad \text{and} \quad \frac{dc^*}{db} = \underbrace{\frac{\theta - kf_{11}}{f_{11}}}_{-} + \underbrace{\frac{\theta f_{12} - f_2 f_{11}}{-\alpha f_{11}}}_{+} \quad ?.$$

This is because a rise of the birth rate has two effects. First, it has a negative effect as more needs to be invested to maintain the same capital per capita. Second, there is a positive effect. The proportion of healthy people increase due to more healthy newborns, and thus a higher labor force leads to higher physical capital and consumption. Hence the two effects are offsetting each other and the net effect is unclear in general.

In the endemic case with health expenditure, by the implicit function theorem

$$\begin{aligned} \frac{dk^*}{db} &= \frac{1}{D} \underbrace{(f_{22}g'l'l'_\theta + f_2g'l''_\theta + f_2g''\frac{\partial m}{\partial h}l'_\theta - f_{12}l')}_- + \underbrace{-\frac{1}{D}f_2f_{12}g'\frac{1}{\alpha}l''_\theta}_+ + \underbrace{\frac{1}{D}f_2f_{12}g'l'\frac{\partial l'_\theta}{\partial b}}_? \quad ? \\ \frac{dh^*}{db} &= \frac{1}{D} \underbrace{(f_{11} - f_{21}g'l'_\theta)}_- + \frac{1}{D} \frac{1}{\alpha} \underbrace{g'l'_\theta(f_{21}f_{12} - f_{11}f_{22})}_- + \frac{1}{D} \underbrace{(-f_{11}f_2g'\frac{\partial l'_\theta}{\partial b})}_? \quad ? \\ \text{and then } \frac{dl^*}{db} &= \frac{1}{\alpha} + l'(h) \frac{dh^*}{db} \quad ? \end{aligned}$$

where $\frac{\partial l'_\theta}{\partial b} = -\frac{\alpha'}{\alpha^2} + \frac{\theta(\theta\alpha' + \alpha(\alpha' - \gamma'))}{\alpha^2(\alpha - (\gamma + b) + \theta)^2}$.¹¹

Therefore the effect of a rise in birth rate is ambiguous. The basic reasoning is similar to the endemic case without health expenditure above, but here it becomes more complex by involving changes in health capital. First, it has a negative effect: The marginal cost of physical capital and health capital will increase which leads lower physical capital and health capital. Second, since people are born healthy the labor force is increasing, which means marginal productivity of physical capital is increasing and hence physical capital needs to rise. On the other hand the higher labor force causes marginal productivity of labor to decline and hence health capital needs to decrease. Third, because of more healthy newborns, the marginal benefit of health is changing. The marginal benefit of health to labor force is increasing ($\partial l'/\partial b > 0$), whereas the discounted marginal benefit of health to labor force $\partial l'_\theta/\partial b$ is unclear.

To see the effect of the change in b for a parametrized example, We specify the following functional forms:

$$f(k, l) = Ak^{a_1}l^{1-a_1}; \quad g(m) = (m + \phi_1)^{\phi_2} - \phi_1^{\phi_2}; \quad \alpha(h) = \alpha_1 + \alpha_2 e^{-\alpha_3 h}; \quad \gamma(h) = \gamma_1 - \gamma_2 \exp^{-\gamma_3 h}.$$

The parameter values are chosen as follows: $A = 1, a = 0.36, \sigma = 1, \delta = 0.05, \theta = 0.05$ and $d = 0.005$ by convention. Since there are no counterpart for health related functions in economic literature we choose the following parameters which satisfy all the assumptions we made earlier: $\phi_1 = 2, \phi_1 = 0.1, \alpha_1 = \alpha_2 = 0.023, \alpha_3 = 1, \gamma_1 = \gamma_2 = \gamma_3 = 1$. So we have $\bar{\alpha} = 0.046$ and $\underline{\gamma} = 0$. We vary b from 0.5% to 5%, which is the range of birth rates for all the countries in the world, and $\hat{b} = 4\%$. So if $b \in [4.6\%, 5\%]$ there is only disease free steady state, if $b \in [4\%, 4.6\%]$ there is endemic steady state without health expenditure and if $b \in [0.5\%, 4\%]$ there is endemic steady state with health expenditure. We can see from the Figure 5 that as b decreases, from the disease free steady state, the endemic steady state with no health expenditure emerges, and if it decreases further the endemic steady state with positive health expenditure emerges. The capital stock decreases, and as b decreases, it starts increasing due to the increasing health expenditures. This is mirrored in the effect on consumption. One of the interesting implications of this is that there will be a positive relationship between capital and hence output and health capital, and consumption and health capital. Thus, one may be led to think that there is a causal relationship between income and health capital - that health is a luxury good. However, the link is through the birth rate. If we were to look at the relationship between birth rate and health expenditure there would be the negative relationship which drives the link between income and health capital. The intuition is that as the birth rate falls the cost of the marginal worker falling ill becomes higher and this leads to an increase in health expenditure and hence health capital.

7 The Conclusion

This paper developed a framework to study the interaction of infectious diseases and economic growth by establishing a link between economic growth model and epidemiology model. We find that there are multiple steady states. Furthermore by examining the local stability we explored how the equilibrium properties of the model change as the parameters are varied. Although the model we present here is elementary, it provides a fundamental framework for considering more complicated model. It is important to understand the basic relationship between disease prevalence and economic growth before we go even

¹¹Note $\partial l'/\partial b = -\alpha'^2 > 0$, but it is not clear $\partial l'_\theta/\partial b$ takes the positive sign or the negative sign.

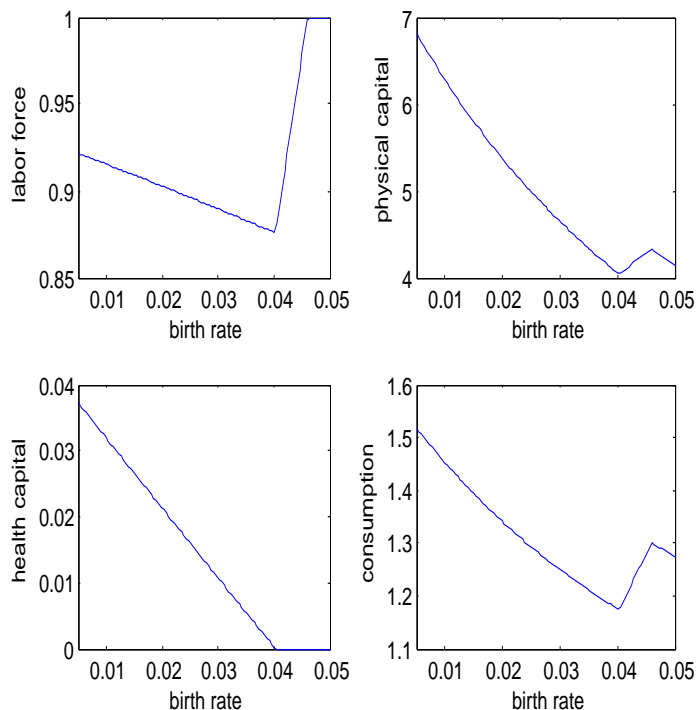


Figure 5: Comparative statics by varying birth rate b

further to consider more general models. The model also points the link between the health expenditures and income - both of which are endogenous - may be driven by parameters of population - as the birth rate drops the cost of a marginal worker becoming ill increases which leads to a negative relationship between population growth and health expenditures (controlling for disease induced mortality). An epidemiology model including control procedures, such as screening, tracing infectors, tracing infectives, post-treatment vaccination and general vaccination can be used to study the economic cost and benefit analysis of disease control. Moreover, the prevalence for many diseases varies periodically because of seasonal changes in the epidemiological parameters. It may also be one of the reasons of economic fluctuations. In addition the parameters can be estimated and used to analyzed the economic effects of some specific infectious diseases in detail.

In a companion paper, Goenka and Liu (2010) we examine a discrete time formulation of a similar model. In that paper, however, there is only a one way interaction between the disease and the economy. The disease affects the labor force as in this model, but the labor supply by healthy individuals is endogenous and the epidemiology parameters are treated as biological constants. We find that under standard assumptions the dynamics of the model with and without endogenous labor are topologically conjugate. Thus, there may be no loss in generality in using an exogenous labor-leisure choice as in this paper. This simplifying assumption of a one-way interaction, the dynamics become two-dimensional and we can study the global dynamics. The key result is that as the disease becomes more infective, cycles and then eventually chaos emerges. Here, we endogenize the epidemiology parameters. Thus, it is a framework to study optimal health policy. However, the dynamical system becomes six dimensional and we have to restrict our analysis to local analysis of the steady state. In Goenka and Liu (2009) we incorporate learning-by-doing into a similar model as the current paper. We find that the growth rate is reduced by disease incidence. However, unlike Lucas (1988) the growth rate depend on all the economic parameters of the model as the human and physical capital choice depends on these. Thus, even small differences in the disease prevalence or in the economic fundamentals can have long run effects.

8 Appendix: Center Manifold Calculation

Here, we introduce the procedure of calculating center manifold instead of the calculation part itself. We use $\dot{x} = g(x, b)$ to denote the dynamic system, where $x = (k, h, l, c)^T \in \mathbb{R}_+^4$, and $g : \mathbb{R}_+ \times \mathbb{R}_+^4 \rightarrow \mathbb{R}_+^4$ is the

vector field. Moreover, we use x^* to denote its equilibrium point, and so $g(x^*, b) = 0$. Bifurcation occurs when $b^* = \bar{\alpha} - \gamma$. We assume $g(x, b)$ to be at least C^5 . We follow the procedure given by Wiggins (2003) and Kribs-Zaleta (2002):

1. Using $\tilde{x} = x - x^*$ and $\tilde{b} = b - b^*$, we transform the dynamical system into $\dot{\tilde{x}} = g(\tilde{x} + x^*, \tilde{b} + b^*)$ with the equilibrium point $\tilde{x}^* = 0$ and bifurcation point $\tilde{b}^* = 0$. Then we linearize the system at point 0 to get $\dot{\tilde{x}} = D_x g(x^*, b^*)\tilde{x} + D_b g(x^*, b^*)\tilde{b} + R(\tilde{x}, \tilde{b})$, where $R(\tilde{x}, \tilde{b})$ is the high order term;
2. Let $A = D_x g(x^*, b^*)$, $B = D_b g(x^*, b^*)$ and calculate matrix A's eigenvalues, corresponding eigenvectors matrix TA (placing the eigenvector corresponding to zero eigenvalue first) and its inverse TA^{-1} . By transformation $\tilde{x} = TA \cdot y$, we get $y = TA^{-1} \cdot A \cdot TA \cdot y + TA^{-1} \cdot B \cdot \tilde{b} + TA^{-1} \cdot R(TA \cdot y, \tilde{b})$, where $TA^{-1} \cdot A \cdot TA$ is its Jordan canonical form;
3. We separate y into two vectors y_1 , the first term, and y_2 , the rest terms, and then we can rewrite the system as:

$$\begin{aligned} y_1' &= \Gamma_1 y_1 + \tilde{R}_1(TA \cdot y, \tilde{b}) \\ y_2' &= \Gamma_2 y_2 + \tilde{R}_2(TA \cdot y, \tilde{b}); \end{aligned}$$

Since $TA^{-1} \cdot B \neq 0$, we separate it into two vectors Δ_1 with only one element, and Δ_2 with the rest, and form a system as:

$$\begin{pmatrix} y_1 \\ \tilde{b} \\ y_2 \end{pmatrix}' = \underbrace{\begin{pmatrix} \Gamma_1 & \Delta_1 & 0 \\ 0 & 0 & 0 \\ 0 & \Delta_2 & \Gamma_2 \end{pmatrix}}_C \underbrace{\begin{pmatrix} y_1 \\ \tilde{b} \\ y_2 \end{pmatrix}}_{y_b} + \underbrace{\begin{pmatrix} \tilde{R}_1(TA \cdot y, \tilde{b}) \\ 0 \\ \tilde{R}_2(TA \cdot y, \tilde{b}) \end{pmatrix}}_{\tilde{R}_b(TA \cdot y, \tilde{b})};$$

4. In order to put matrix C into Jordan canonical form, we make another linear transformation $y_b = TC \cdot z$, and get $\dot{z} = TC^{-1} \cdot C \cdot TC \cdot z + TC^{-1} \cdot \tilde{R}_b(TA \cdot TC \cdot z, \tilde{b})$, where $z = (z_1, \tilde{b}, z_2, z_3, z_4)$. Therefore, we can now write the system as:

$$\begin{aligned} z_1' &= \Pi_1 z_1 + \hat{R}_1(z_1, z_2, z_3, z_4, \tilde{b}) \\ z_2' &= \Pi_2 z_2 + \hat{R}_2(z_1, z_2, z_3, z_4, \tilde{b}) \\ z_3' &= \Pi_3 z_3 + \hat{R}_3(z_1, z_2, z_3, z_4, \tilde{b}) \\ z_4' &= \Pi_4 z_4 + \hat{R}_4(z_1, z_2, z_3, z_4, \tilde{b}) \\ \tilde{b}' &= 0; \end{aligned}$$

5. Take $z_i = h_i(z_1, \tilde{b})$ ($i = 2, 3, 4$) as a polynomial approximation to the center manifold, and differentiate both sides w.r.t. t :

$$\Pi_i z_i + \hat{R}_i(z_1, h_2, h_3, h_4, \tilde{b}) = D_{z_1} h_i(z_1, \tilde{b})[\Pi_1 z_1 + \hat{R}_1(z_1, h_2, h_3, h_4, \tilde{b})].$$

And then solve for the center manifold by equating the coefficient of each order;

6. Finally, we write the differential equation for the dynamical system on the center manifold by substituting $h_i(z_1, \tilde{b})$ in $\hat{R}_1(z_1, z_2, z_3, z_4, \tilde{b})$, and get the system:

$$\begin{aligned} z_1' &= \Pi_1 z_1 + \hat{R}_1(z_1, h_2(z_1, \tilde{b}), h_3(z_1, \tilde{b}), h_4(z_1, \tilde{b}), \tilde{b}) \\ \tilde{b}' &= 0. \end{aligned}$$

However, in our economic epidemiology model as dynamics of l and h is independent of the rest of system dynamics, we could just simply calculate their dynamics on the center manifold, which is given by:

$$\dot{z}_1 = \bar{\alpha} z_1 (z_1 - \frac{1}{\bar{\alpha}} \tilde{b}).$$

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. Anderson, R.M. and May, R.M. (1991) *Infectious Diseases of Humans*, Oxford: Oxford Science Publications.
3. Arrow, K.J. and Kurz, M. (1970) *Public investment, the rate of return, and optimal fiscal policy*, Baltimore: The Johns Hopkins Press.
4. Ashraf, Q.H., Lester, A., and Weil, D.N. (2009) When does improving health raise GDP? In D. Acemoglu, K. Rogoff, and M. Woodford (Eds.) *NBER Macroeconomics Annual 2008*, Chicago: The University of Chicago Press.
5. Barrett, S. and Hoel, M. (2004) Optimal disease eradication, mimeo. University of Oslo.
6. Bell, C., Devarajan, S. and Gersbach, H., (2003) The long-run economic costs of AIDS: theory and an application to South Africa, *Working paper*, The World Bank.
7. Bleakley, H. (2007) Disease and development: Evidence from hookworm eradication in the American South, *Quarterly Journal of Economics*, 122: 73-117.
8. Boldrin, M., Jones, L. and Khan, A. (2009) Three equations generating an industrial revolution, WUSTL, mimeo.
9. Bloom, D.E., Canning, D. and Fink, G. (2009) Diseases and development revisited, NBER Working Paper No. 15137.
10. Brander, J.A. and Dowrick, S. (1994) The Role of Fertility and Population in Economic Growth: Empirical Results: From Aggregate Cross-National Data, *Journal of Population Economics*.
11. d'Albis, H. and Augeraud-Véron, E. (2008): The optimal prevention of epidemics, Mimeo., LERNA, Toulouse.
12. 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.
13. Delfino, D. and Simmons, P.J. (2000) Positive and normative issues of economic growth with infectious diseases, Discussion Papers in Economics 2000/48, University of York.
14. Dunford, N. and J.T. Schwartz, (1967) *Linear operators*, New York: Interscience Publishers.
15. Geoffard, P.-Y. and Philipson, T. (1997) Rational epidemics and their rational control, *International Economic Review*, 37: 603-624.
16. Gersovitz, M. and Hammer, J.S. (2004) The economical control of infectious diseases, *The Economic Journal* : 1-27.
17. Goenka, A., and Liu, L. (2010) Infectious diseases and endogenous fluctuations, *Economic Theory*, Forthcoming.
18. Goenka, A., and Liu, L. (2009) Infectious diseases and endogenous growth, National University of Singapore, mimeo.
19. Hall, R.E. and Jones, C. (2007) The value of life and the rise in health spending, *Quarterly Journal of Economics*, 122 (1): 39-72.
20. Hethcote, H.W. (2000) The mathematics of infectious diseases, *SIAM Review* 42: 599-653.
21. Hethcote, H.W. (2009) The basic epidemiology models, in S. Ma and Y. Xia (Eds.) *Mathematical understanding of infectious disease dynamics*, Singapore: World Scientific.
22. 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.

23. Kribs-Zaleta, C.M. (2002) Center manifolds and normal forms in epidemic models, in C. Castillo-Chavez, *et al* (Eds.) *Mathematical Approaches for Emerging and Reemerging Infectious Diseases: An Introduction* : 269-86.
24. Kremer, M. (1996) Integrating Behavioral Choice into Epidemiological Models of the AIDS Epidemic, *Quarterly Journal of Economics*, 111: 549-573.
25. Kremer, M. and Miguel, E. (2004) Worms: Identifying Impacts on Education and Health in the Presence of Treatment Externalities, *Econometrica* 72: 159-217.
26. Le Van, C. and Vailakis, Y (2003) Existence of a competitive equilibrium in a one sector growth model with irreversible investment, *Economic Theory*, 22: 743-771.
27. Lucas, Robert E. (1988): "On the mechanics of economic development," *Journal of Monetary Economics* 22: 3-42.
28. Philipson, T. (2000) Economic epidemiology and infectious diseases, in J. Newhouse and T. Culyer (Eds.) *Handbook of Health Economics*, New York: North-Holland.
29. Thirumurthy, H., Zivin, J.G., and Goldstein, M. (2007) The economic impact of AIDS treatment: Labor supply in Western Kenya, *Journal of Human Resources*, 43: 511-552.
30. Weil, D. (2007) Accounting for the effect of health on economic growth, *Quarterly Journal of Economics*, 122.
31. Wiggins, S., (2003) *Introduction to applied nonlinear dynamical systems and chaos*, New York: Springer-Verlag.
32. 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.