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Aditya Goenka Lin Liu and Manh-Hung Nguyen

Toulouse School of Economics

SIR Economic Epidemiological Models with Disease Induced Mortality*

Aditya Goenka† Lin Liu‡ Manh-Hung Nguyen§

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Abstract

This paper studies an optimal growth model where health expenditures (alternatively lockdowns) can be made to reduce infectivity of the disease when there is an infectious disease with SIR dynamics and infections can cause disease related mortality. We study implications of two different SIR models - with early mortality and with late mortality from the disease - on health outcomes, optimal response and on economic outcomes in equilibrium. We characterize the steady states and show how these vary when varying mortality. The outcomes are sensitive to the specification of the epidemiology model. We also study sufficiency conditions and provide the first results in economic models with SIR dynamics with and without disease related mortality - a class of models which are non-convex and have endogenous discounting so that no existing results are applicable.

Keywords: Infectious diseases, Covid-19, SIR model, mortality, sufficiency conditions, economic growth, lockdown, prevention, health expenditure.

JEL Classification: *E13, E22, D15, D50, D63, I10, I15, I18, O41, C61.*

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†Department of Economics, University of Birmingham, Email: a.goenka@bham.ac.uk

‡Management School, University of Liverpool, Email: lin.liu@liverpool.ac.uk

§Toulouse School of Economics, INRAE, University of Toulouse Capitole, Email: manh-hung.nguyen@tse-fr.eu

1 Introduction

The Covid-19 pandemic has brought the study of interaction of infectious disease with the economy, i.e. economic epidemiology models, to the frontiers of economic research. The first generation of economic epidemiology models typically studied SIS models without disease related mortality (see Bonds, et al. (2004), Bosi and Desmarchellier (2018), Delfino and Simmons (2004), Gersovitz and Hammer (2004), Goenka and Liu (2013, 2020), Goenka, Liu and Nguyen (2014), Toxvaerd (2019)). However, with Covid-19 the modeling of mortality has become important as this seems to be a driver of the policy responses adopted in many countries. [1]

This paper analyses the SIR model which has been used to model Covid-19 (Ferguson, et al. (2020)) when there is disease related mortality[2] in a neoclassical growth model so that the model is fully general equilibrium.[3] The literature has largely studied immediate effects of Covid-19 and in this paper we concentrate on the medium to long run effects, i.e. effects of the disease on the steady state.[4] Households can save through investing in capital and production of the single consumption good uses capital and labor. Only those that are not infected (i.e. those who are susceptible and those recovered from the disease) individuals can work. There are two effects of the disease: there is morbidity, i.e. those who are ill do not work, and there is mortality, so that a fraction of those who have contracted the disease die due to it. The contact rate is endogenous in the model and is decreasing in health expenditures, which can also be interpreted as self-isolation costs (Eichenbaum, et al. (2020).[5] In all our model the households are homogeneous and we do not model disease related externality where households do not take into account the effect of their decisions on the evolution of the disease in the population. The recent paper, Goenka and Liu (2020) explores in detail the effect of this health externality[6] in a dynamic general equilibrium model with health expenditures and we abstract from it to concentrate on the role of mortality in

[1] Boucekkine and Laffargue (2010) and Chakraborty, et al. (2010) model disease related mortality in the overlapping generations framework but did not use a compartment epidemiology model as in this paper and the emerging literature.

[2] Whether there is long-lasting immunity in Covid-19 is contentious. Long, et al. (2020) using data from China find evidence consistent with steep decline in 2-3 months. Similar results were found in a study in the US (Ibarrando, et al. (2020)). On the other hand Wajnberg, et al. (2020) and Sekine, et al. (2020) find evidence suggesting longer immunity. As in our companion paper we have studied issues related to the SIS model, here we study implications of the alternate, SIR model.

[3] Goenka, Liu and Nguyen (2020) in a companion paper study optimal lockdown and other issues in a SIS model with disease related mortality.

[4] There are other papers using the SIR model with disease related mortality e.g. Acemoglu, et al. (2020), Alvarez, et al. (2020), Eichenbaum, et al. (2020), and Jones, et al. (2020). These papers concentrate on short-run models and are not fully general equilibrium as all variables (wages, interest rate, and capital) do not adjust.

[5] Goenka and Liu (2020) and Goenka, Liu and Nguyen (2020) modeled optimal health expenditures in a similar growth framework. These papers treated health expenditures as accumulating in health capacity which is important for understanding response to Covid-19 and the mortality due to it. This introduces another state variable and in this paper to simplify the analysis we treat health expenditures as only a flow variable.

[6] This has been modeled in different ways in the literature, see Bosi and Desmarchellier (2018), Geoffard and Philipson (1996), Gersovitz and Hammer (2004), Goenka and Liu (2020), Hellwig, et al. (2020) and Toxvaerd (2019).

modeling diseases of the SIR type. In the model we use an extended welfare function that depends on the utility from consumption as well as a loss in welfare from disease related mortality as without it there can be counter-intuitive effects where increase in mortality is welfare improving.[7]

There are two main methodological issues that we feel have not received adequate attention which we study in the paper. The first is examining different modeling choices for the SIR model. Many of the recent papers are concerned with the immediate short run and have used the Kermack-McKendrick model with mass action. As discussed in the next section, for longer term analysis epidemiologists tend to use the standard or density dependence model where infections depend on the fraction and number of infectives in the population. The modeling choice is relevant as with disease related mortality the population size changes and this affects the epidemiological dynamics. This will in turn affect the equilibrium economic choices and outcomes. Even with this model the timing of disease mortality has important effects on dynamics of the disease. We examine two canonical specifications of the epidemiology model - one with early mortality so that there is immediate death of the infectives, and one with delayed mortality - where death takes place later when those who die from the disease are not also circulating in the population transmitting the disease (see Busenberg and van den Driessche (1990) and Keeling and Rohani (2008)). The timing of mortality affects epidemiology dynamics. We examine what the implications of the epidemiology modeling choices are on optimal choices and on the equilibrium steady state outcomes.

The second methodological issue is that we study the sufficiency conditions for the optimal control problem. It is well known that the SIR epidemiology dynamics are non-convex and thus, the standard Arrow or Mangasarian conditions do not apply (Gersovitz and Hammer (2004)). There are no results for the SIR model with and without mortality that can be used in economic models to our knowledge. Endogenous mortality adds another problem as the population becomes endogenous. We directly address this issue and given the special structure of the problem, we directly show the relevant transversality conditions and establish sufficiency by adapting the method of Leitmann and Stalford (1970) that was used for convex problems. Sufficiency for a neoclassical growth model with SIS disease dynamics without disease related mortality was established by Goenka, Liu and Nguyen (2014). The recent work, Goenka, Liu and Nguyen (2020) establishes sufficiency for the SIS model with disease related mortality. The problem is that the model is one of endogenous discounting which is non-convex. Thus, the transversality and sufficiency conditions for convex endogenous population models (see Boucekkine, et al. (2018)) do not apply. There are differences between the SIS and SIR models: that there is one less state variable for the epidemiology dynamics in the SIS models. The key to the proof in the current paper is to show the co-state variables associated with the bounded state variables converge to zero with time, and this implies a different transversality condition than in Goenka, Liu and Nguyen (2020). In Goenka, Liu and Nguyen (2020) paper the set up rules out the case of full labor supply in an endemic steady state. In the current paper this property does not hold and we have to rely on a different argument relying on stability properties of the steady state. As a special case, we obtain the sufficiency in the SIR model without disease related mortality.

[7]See Goenka, Liu and Nguyen (2020) for extensive discussion of the issues associated with modeling the objective function.

The model is a fully dynamic general equilibrium model and we characterize the Euler equations that govern the evolution of the economy. As our interest is beyond the very short run, we show that there are two steady states for the economy: a disease free and disease endemic steady state. The optimal health expenditure depends on a function of the parameters and the equilibrium values of the economic variables. This function is interpreted as the net marginal benefit of health expenditure (net of the marginal cost) its position determines whether the health expenditure is positive or not in a steady state, and if it is, its magnitude. Thus, the equilibrium reproduction rate, R_0^* will depend on both the infectivity of the disease and endogenous economic choices. As the models are too complex to solve analytically we study how the steady state values of variables of interest change as the rate of disease related mortality changes (and thus, the number of disease related deaths in equilibrium). The two models behave differently depending on the weight given to loss of welfare from disease related mortality. If there is no weight given to this loss, as increases in mortality self-limits the spread of the disease in the early mortality model (the sick die faster so the exit rate from the infective state increase limiting the spread of infections) even when the number of deaths in the two models are comparable. This leads to better disease and economic outcomes. This is driven by the greater incentive for health expenditures. When there is a positive weight given to welfare loss due to mortality the situation reverses. Even when number of deaths are similar, as there is no self-limiting behavior in the delayed mortality model, the expenditure on health is more aggressive leading to lower contact rates. The economic outcomes are better in the early mortality model in both specifications. The intuition is that in this model it is those that who are not working anyway that die leading to a lower loss in output. In addition, in the delayed mortality model as greater health expenditures are made to reduce the spread of infection, this further decreases consumption and investment in capital stock. The effect of increased mortality increasing economic outcomes in the early mortality model is similar to the gift of the dying in Young (2005) even though the exact mechanism is different (that paper had a Solow type model without health expenditures so the savings and investment do not adjust and the primary mechanism is the increase in per-capita capital stock). Thus, the details of the epidemiology modeling will affect economic implications.

The plan of the paper is as follows: Section 2 studies the SIR model with early and late mortality. Section 3 introduces the economic epidemiology model, and characterizes the Euler equations and steady states, and Section 4 does comparative statics of equilibrium steady state outcomes when mortality is varied. Section 5 studies the transversality and sufficiency conditions, and Section 6 concludes.

2 The Epidemiological Models

In this section, we discuss some issues concerning the SIR model which is used in the mathematical epidemiology literature. The simplest SIR model for a fixed population also known as the Kermack-McKendrick model is given by the following system of differential

$$\begin{aligned}\dot{S} &= -\alpha SI \\ \dot{I} &= \alpha SI - \gamma I \\ \dot{R} &= \gamma I,\end{aligned}$$

where S are the individuals who are healthy and susceptible to the disease, I are those infected and infective, and R are those who have recovered from the disease and have immunity from the disease. The parameters of the model are as follows: α is the adequate contact rate to transmit the disease, γ is the recovery rate from the disease. The model as specified is the mass action incidence where the transmission depends on the *number* of infective individuals. This is model is used for early stages of a disease, typically less than a year, where the population can be treated as fixed. An alternate specification is

$$\begin{aligned}\dot{S} &= -\frac{\alpha SI}{N} \\ \dot{I} &= \frac{\alpha SI}{N} - \gamma I \\ \dot{R} &= \gamma I.\end{aligned}$$

In this specification the transmission depends on the *fraction* of the infectives in the population. This is the standard incidence or density dependent incidence. If N is fixed then the two models are essentially similar.[8] When population is not constant either due to longer horizons where birth and deaths become significant or there is disease related mortality the choice of which model to use becomes significant.The epidemiology literature tends to use the standard incidence model (or some variation of it) for several reasons. There are also reasons why one would like to consider its use in economic models. First, the mass action model has a scale effect so that increases in population size will increase the rate of infections as the contacts increase linearly with population size (αN). However, the pattern of human interaction is relatively stable and invariant to population size. Second, Anderson and May (1991) estimated the contact rate for 5 diseases for population size 1,000 and 400,000 using the specification $\alpha N^{\nu} SI/N$ and found that $\nu \approx (0.03, 0.07)$ indicating that the standard incidence model is a better fit than the mass action incidence model. Third, as mortality increases in the mass action model there is an in-built dampening mechanism - as population size drops, so does the contact rate. Thus, the first model will imply a self-limiting behavior of epidemics due to mortality when the second will not. Fourth, if population is not growing then the threshold for existence of an endemic steady state depends inversely on initial population size - so that a larger initial condition makes it more likely the disease will be endemic (Mena-Lorca and Hethcote (1992)). Furthermore, if population is growing then eventually every one will get infected except when disease related mortality is higher

[8] In the first model, the number of contacts is αN, and the probability of a contact of an infective with a susceptible is $\alpha N(S/N)$ giving the number of new infections $\alpha N(S/N)I = \alpha SI$. In the second model, the number of contacts for a susceptible to catch the disease as $\alpha N(I/N)$ giving the number of new infections as $\alpha N(I/N)S = \alpha SI$.

than the natural population growth rate (Mena-Lorca and Hethcote (1992)). (See Brauer (2008), Mena-Lorca and Hethcote (1992), Hethcote (2000), and Keeling and Rohani (2008) for further discussion of the modeling in SIR models.)

As our focus is on infectious diseases with disease related mortality over the medium and long run we are concerned with models where population is varying both due to disease related mortality and with births and deaths (from other causes) in the population. Given the earlier discussion, we work with the standard incidence model which is canonical in the epidemiology literature.

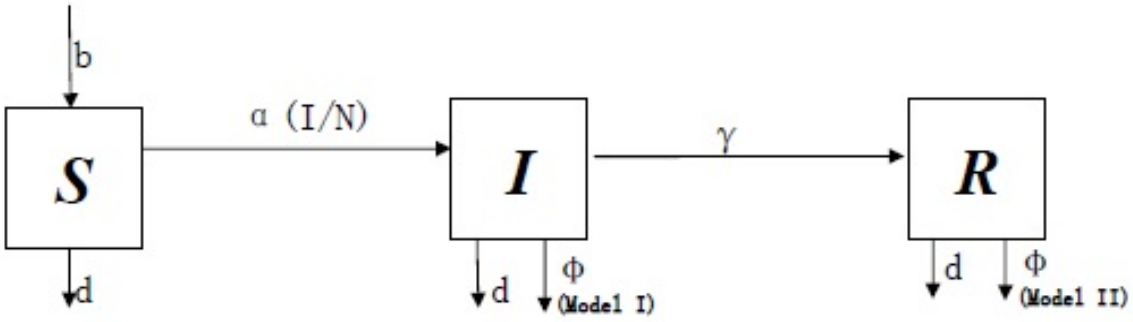
The transfer diagram for the SIR epidemiological models with disease induced mortality is shown in Figure 1 for two variations of the model. Now there are newborns (new entrants to the population) with the birth rate, b, with newborns healthy and susceptible. All individuals irrespective of health status die at the rate d. The susceptible get infected at the rate $\alpha\frac{I}{N}$, and the infected recover at the rate γ.

The two models differ only in how we model disease induced mortality. In model I - the model of Early Mortality - the infected dies at the rate ϕ due to infectious diseases,[9] while in model II, the recovered dies at the rate ϕ due to infectious diseases. Thus, model II is a model with Delayed Mortality. In the first model, if mortality is high the period of infectiousness is reduced which in turn will reduce new infections. To see this note that new infections are due to fraction of I. The outflow from I is given by $\gamma + \phi$ (holding the exogenous mortality d constant) which reduces the stock of infectives and thus, new infections. Thus, there is a self-limiting effect of increase in mortality. In addition, diseases where the mortality is late in the infectiousness period the second model is also better modeling choice (Keeling and Rohani (2008)).

The pure SIR epidemiological models with varying population size are well studied in mathematical biological literature (see Busenberg and van den Driessche (1990), Hethcote (2008), Keeling and Rohani (2008) and Mena-Lorca and Hethcote (1992)).

[9] ϕ is similar to the Infection Fatality Rate which also covers those that are untested and asymptomatic rather than the Case Fatality Rate which depends on the number of individuals diagnosed with the disease.

Figure 1. The Transfer Diagram For the SIR Epidemiology Model with mortality



Note: In a SIR epidemiology model with disease related mortality, the total population is divided into three groups: the susceptible denoted as S, the infected denoted as I and the recovered denoted as R. The birth rate is b and newborns are born healthy and susceptible. All individuals irrespective of health status die at the rate d. The susceptible get infected at the rate $\alpha I/N$, the infected recover at the rate γ. In model I, the infected die at the rate ϕ due to disease, while in model II, the recovered die at the rate ϕ due to disease.

Assumption 1. *We assume the parameters in the SIR models:*

1. *The demographic parameters b and d are non-negative, and $b \geq d + \phi$;*
2. *The recovery rate $\gamma > 0$;*
3. *The disease induced mortality rate $\phi \geq 0$.*

The first assumption ensures that the total population in the models grows over time, regardless of the level of disease prevalence, otherwise there may be extinction of the population. In a SIR model with varying population size the dynamic properties depend on the evolution of the population size. We make the above assumption which is consistent with existence of steady states with a growing population. Busenberg and van den Driessche (1990) have a weaker condition that depends on the properties of the steady state fractions of susceptibles and infectives. Mena-Lorca and Hethcote (1992) only study the first model and have a weaker condition that depends only on the parameters and not on the steady state.[10] As we want to use a unified condition for the two models which is also consistent with the properties of the economic epidemiology model we assume this stronger condition which is meaningful and easy to interpret. We assume the recovery rate is positive as this is an SIR model. In the following analysis, we study how the model predictions vary when we change disease mortality rate ϕ. When $\phi = 0$, it is a special case where the two versions of SIR models coincide. We do not impose assumption on the contact rate α here, as it is endogenous in the economic epidemiological models in the next section. The contact

[10]Essentially in the first model below they assume $b \geq d + \phi i^{\infty}$ and in the second model, $b \geq d + \phi r^{\infty}$.

rate in the epidemiology literature is also understood to depend on behavioral and social considerations.

2.1 Model I: Early Mortality

The model I is given by the following system of differential equations :

$$\begin{aligned}\dot{S} &= bN - \frac{\alpha SI}{N} - dS \\ \dot{I} &= \frac{\alpha SI}{N} - \gamma I - dI - \phi I \\ \dot{R} &= \gamma I - dR \\ \dot{N} &= (b-d)N - \phi I\end{aligned}$$

In this model the outflow of susceptibles is due to new infections, $\alpha(I/N)S,\ \alpha > 0$ and death due to other causes $dS, d > 0$. All individuals are born healthy and thus there is a flow into the susceptible class, $bN,\ b > 0$.[11] The infectives grow by $\alpha(I/N)S$ and infectives recover at rate $\gamma > 0$ or succumb to the disease at rate $\phi > 0$. There is also mortality from other causes given by dS. The individuals who recover from the disease have subsequent immunity from the disease and inflow is given by γI and there is mortality from other causes dR. The population changes by net births, $(b-d)N$ and disease related mortality, ϕI. Since $N = S + I + R$, we define $s = S/N$ and $i = I/N$ and rewrite the model as:[12]

$$\dot{i} = \alpha si - \gamma i - bi - \phi i + \phi i^2 \tag{1}$$

$$\dot{s} = b - bs - \alpha si + \phi si \tag{2}$$

$$\frac{\dot{N}}{N} = (b-d) - \phi i. \tag{3}$$

Note that the proportion of the recovered $r = R/N = 1 - s - i$.

Proposition 1. *(Busenberg and van den Driessche (1990), Mena-Lorca and Hethcote (1992)) Consider the epidemiological model given by equation (1) - (3). Then*

1. *The disease free steady state with $s^\infty = 1$ and $i^\infty = 0$ always exists. It is stable when $\frac{\alpha}{b+\gamma+\phi} \leq 1$, and unstable when $\frac{\alpha}{b+\gamma+\phi} > 1$;*
2. *When $\frac{\alpha}{b+\gamma+\phi} > 1$, there exists a unique endemic steady state with $0 < s^\infty < 1$ and $0 < i^\infty < 1$, which is stable. The endemic steady state (s^∞, i^∞) is the solution to the*

[11] These are new entrants to the population.

[12] We denote the steady state of a variable x as x^∞ to distinguish it from the optimal value in a trajectory in the later part of the paper which is denoted as x^*.

following system of equations:

$$\alpha(\alpha-\phi)s^2-[\alpha(\gamma+b+\phi)-\phi(\phi+\gamma)]s+b\phi=0 \tag{4}$$

$$\phi i+\alpha s-(\phi+\gamma+b)=0. \tag{5}$$

Note that even though fraction of susceptibles, s, is given by a quadratic equation there is only one admissible solution in the range $0 \leq s \leq 1$.

2.2 Model II: Delayed Mortality

The model II is given by the following system of differential equations :

$$\begin{aligned}
\dot{S} &= bN-\frac{\alpha SI}{N}-dS \\
\dot{I} &= \frac{\alpha SI}{N}-\gamma I-dI \\
\dot{R} &= \gamma I-dR-\phi R \\
\dot{N} &= (b-d)N-\phi R
\end{aligned}$$

With delayed mortality, the model differs from the earlier one in that mortality from the disease is among the recovered, ϕR rather than in the class of infectives. It may seem strange that a fraction of the recovered population is dying from the disease rather than those infected from the disease but the model is valid with a suitable interpretation. Whether infectives are asymptomatic or have mild or severe symptoms it is their coming into contact with susceptibles which leads to transmission of the disease. In Covid-19 it is those with severe symptoms which die from the disease and it seems that a large part of this mortality has taken in either hospitals or care homes. Thus, one can think of them not circulating in the population and thus, one can treat this group in the recovered group from a modeling point of view. This is consistent with the evidence that risk of infections in the community are higher than that in hospitals, i.e. nosocomial infections (Carter, et al. (2020)). The mortality in the recovered group also covers pre-mature mortality of those who have been treated and free of the disease. While Covid-19 is too recent to have data on this, there are several other infectious diseases where those who have successfully been treated have shorter conditional life expectancies: for example for TB it is 3.6 years (Hoger, et al. (2016), and for HIV it is 6.8 years (9.5 years without any co-morbidities) (Marcus, et al. (2020)).[13] There is emerging evidence that Covid-19 induces damage to lungs, Ground-glass opacities and lesions, and long term lung fibrosis (Bernheim, et al. (2020), Hosseiny, et al. (2020), and Li and Xia (2020)) and to the heart tissue (see PÃ¨rez-Bermejo, et al. (2020)) and thus, one may expect impairment of life expectancy . Thus, while there is only a morbidity effect on infectives there is an increased mortality effect on the recovered consistent with the delayed mortality model.

[13]For other infectious diseases while there are estimates for mortality, estimates on effect on life expectancy for someone who has been treated are not available.

Figure 3. Simulation for the SIS epidemiological models varying disease induced mortality rate ϕ (high contact rate α)

The panels clockwise are s, i, r and $\phi(1-s-i)$. The blue color line is the simulation for model I, while the red color line is the simulation for model II. The parameter values are $b = 0.02, d = 0.01, \alpha = 0.15$ and $\gamma = 0.1$. The disease induced mortality rate ϕ varies from 0 to 1%.

3 The Economic Epidemiology Models

The model is based on the growth model with SIS disease dynamics in Goenka and Liu (2013, 2020) and Goenka, Liu and Nguyen (2014, 2020), to include disease related mortality and to model lockdowns. In this paper we use an underlying neoclassical growth model with SIR dynamics. To avoid keeping track of the cross-sectional distribution of the healthy, infected and recovered individuals, and to stay close to the canonical growth model, we adopt the framework of a large representative household.

Households: We assume the economy is populated by a continuum of non-atomic identical households who are the representative decision-making agents. In the absence of the disease, the size of the population in each household grows over time at the rate of $b - d \geq 0$, where b is the birth rate and d is the death rate. Within each household, an individual is either healthy or infected or recovered from the diseases. We assume that diseases follow the SIR dynamics (see the discussion in Section 2).

We model the infectious disease as having two effects - reducing productivity of the infected and disease related mortality. We make the simplifying assumption that when an infected individual is incapacitated by the disease the productivity falls to zero. [14] We assume the labor is supplied inelastically.[15]

We use an extended welfare function (see Goenka, Liu and Nguyen (2020) for a discussion) to avoid counter-intuitive effects due to increasing mortality.[16] When there is no disease prevalence, the disutility from disease mortality is of course zero. There is full insurance within the household so that consumption of all members irrespective of health status is the same. The representative household's preferences are given as:

$$\begin{aligned} & \int_0^\infty e^{-\rho t}[u(c_t) - \chi\nu(D_t)]N_t dt \\ = & \int_0^\infty e^{-\int_0^t(\rho - b + d + \phi i(\tau))d\tau}[u(c_t) - \chi\nu(D_t)]N_0 dt \end{aligned} \tag{11}$$

where ρ is the discount factor with $\rho > b - d$, the initial size of household is assumed to be one. $\nu(D)$ is the loss in welfare from disease related mortality, D, and $\chi \geq 0$ is the weight.

Assumption 2. *The felicity function u, $u : \mathbb{R}_+ \to \mathbb{R}$ is $\mathcal{C}^2$ with $u' > 0$ and $u'' < 0$. The discount rate, $\rho > 0$.*

Assumption 3. *The loss from mortality function $\nu(D) : \mathbb{R}_+ \to \mathbb{R}$ is a convex function with $\nu' > 0$ and $\nu'' \geq 0$ and $\nu(0) = 0$.*

[14] How much productivity is affected varies across diseases and see Goenka, Liu and Nguyen (2020) for a discussion of this. If we assume partial decrease in productivity the qualitative results are not affected.

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

[16] This is also consistent with the welfare function used in models which are partial equilibrium, e.g. Acemoglu, et al. (2020), and Alvarez, et al. (2020). See Goenka, Liu and Nguyen (2020) for further discussion.

Production: The production side of the model is a standard neoclassical growth model where households can invest in capital which is productive next period and depreciates at rate δ.[17] Households own representative firms that use capital and labor as inputs.

Assumption 4. *The production function* $f(k,l)$, $f:\mathbb{R}^2_+ \to \mathbb{R}$ *is* $\mathcal{C}^2$ *with*

1. $f_k > 0, f_l > 0$,
2. f *is concave and homogeneous of degree 1,*
3. *with* $f(0,\cdot) = f(\cdot,0) = 0$.
4. $\lim_{k\to 0} f_k(k,\cdot) = \lim_{l\to 0} f_l(\cdot,l) = \infty$; $\lim_{k\to\infty} f_k(k,\cdot) = 0$.
5. *The physical capital depreciates at the rate* $\delta \in (0,1]$.

The total labor force is

$$L = N - I \Rightarrow l = \frac{N-1}{N} = 1 - i. \tag{12}$$

The contact rate, which is key to determine the prevalence of infectious disease, is affected by health expenditure. That is, each household can control infection by investing in health expenditure $h = H/N$. This can be thought of preventive measures taken to prevent the transmission of the disease. This is the specification used in Goenka, Liu and Nguyen (2014) and Goenka and Liu (2020). The first paper also modeled expenditures that increase recovery rates from the disease but we abstract from this in this paper as there are no known therapies for recovery from Covid-19.[18] Eichenbaum, et al. (2020) interpret this loss of output, h in our context, as the cost of a lockdown which also reduces the transmission rate α. In Goenka, Liu and Nguyen (2020) we have a different way of modeling a lockdown or quarantine where a fraction of the non-infective population has to work from home with a reduced productivity.

Assumption 5. *The contact rate is a function of* h *- that is,* $\alpha(h)$. *We assume*

- $\alpha'(h) < 0$;
- $\alpha(h) \to \bar{\alpha}$ *when* $h \to 0$;

[17] Goenka and Liu (2020) have an endogenous growth model where there is human capital accumulation and households choose time to work and time for human capital accumulation. It uses SIS dynamics without disease related mortality.

[18] Antivirals and anti-inflammatory drugs are now known to reduce mortality but these are inexpensive and widely available prior to the outbreak. While ventilators are used in severe cases, there is an emerging view that their use can complicate recovery and in fact cause ventilator induced lung injury (Marini and Gattinoni (2020).

The law of evolution of capital stock is:

$$\dot{K} = f(K, N - I) - C - H - \delta K.$$

We define for each household, physical capital per capita $k = K/N$ and consumption per capita $c = C/N$. Since the two models differ in the growth rate of total population N, which is affected by death rate caused by infectious diseases, the maximization problems which is set up in terms of per household are different. To be clear, we discuss each of the models in turn.

3.1 Model I: Early Mortality

In model I, the growth rate of total population is $b - d - \phi i$. Then, the law of motion for physical capital can be rewritten as:

$$\dot{k} = f(k, 1 - i) - c - h - \delta k - (b - d - \phi i)k \tag{13}$$

The planning problem is to maximize discounted welfare. Each household derives utility from consumption goods - $u(c)$ and disutility from disease related death - $\nu(\phi i)$ with a weight χ, which is non-negative. Each household discounts future at the rate ρ. The objective function is:

$$\begin{aligned} & \max \int_0^\infty e^{-\rho t}[u(c) - \chi\nu(\phi i)]N dt \\ = \; & \max \int_0^\infty e^{-\Delta}[u(c) - \chi\nu(\phi i)]N_0 dt \end{aligned} \tag{14}$$

where we define the following variable which is the effective discount rate (see Uzawa (1968). In Das (2003) discounting depends on consumption and in Boucekkine, et al. (2018) on population similar to this paper).

$$\begin{aligned} \Delta &= \int_0^t (\rho - b + d + \phi i(\tau))d\tau \\ \dot{\Delta} &= \rho - b + d + \phi i. \end{aligned} \tag{15}$$

Thus, the social planner maximizes equation (14) subject to equation (13), (12), (1), (2) and (15) with the constraints

$$0 \leq i \leq 1, \quad 0 \leq s \leq 1, \quad h \geq 0.$$

The control variables are c, h, and state variables are k, i, s, Δ. Define $\lambda_1, \lambda_2, \lambda_3$ and λ_4 are co-state variables for k, i, s and Δ, respectively. μ_1, μ_2 and μ_3 are Lagrangian multipliers for $i \geq 0$, $s \leq 1$ and $h \geq 0$. [19]

[19] We can ignore the constraints $i \leq 1$ and $s \geq 0$.

The problem is one where there is endogenous discounting in a non-convex model so it is well known that the usual sufficiency conditions do not apply. We establish the sufficiency conditions for this model in section 5.[20] The necessary and sufficient F.O.Cs are as follows:

$$
\begin{aligned}
c: \quad & e^{-\Delta}u'(c) = \lambda_1 && (16)\\
h: \quad & \lambda_1 = \alpha'(h)si(\lambda_2 - \lambda_3) + \mu_3 && (17)\\
k: \quad & \dot{\lambda_1} = -\lambda_1[f_1(k, 1-i) - \delta - (b - d - \phi i)] && (18)\\
i: \quad & \dot{\lambda_2} = e^{-\Delta}\chi\phi\nu'(\phi i) + \lambda_1(f_2(k, 1-i) - k\phi) - \lambda_2(\alpha(h)s - \gamma - b - \phi + 2\phi i) + \\
& +\lambda_3(\alpha(h)s - \phi s) - \lambda_4\phi - \mu_1 && (19)\\
s: \quad & \dot{\lambda_3} = \alpha(h)i(\lambda_3 - \lambda_2) + \lambda_3(b - \phi i) + \mu_2 && (20)\\
\Delta: \quad & \dot{\lambda_4} = e^{-\Delta}[u(c) - \chi\nu(\phi i)] && (21)\\
& \mu_1 \geq 0, \quad i \geq 0, \quad \mu_1 i = 0\\
& \mu_2 \geq 0, \quad s \leq 1, \quad \mu_2(1-s) = 0\\
& \mu_3 \geq 0, \quad h \geq 0, \quad \mu_3 h = 0
\end{aligned}
$$

3.1.1 Steady states

Proposition 3. *There always exists a unique disease free steady state with $s^\infty = 1$, $i^\infty = 0$ and $h^\infty = 0$. The economic variables k^∞ and c^∞ are determined by*

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

The disease free steady state is stable when $\frac{\bar{\alpha}}{b+\gamma+\phi} \leq 1$, and unstable when $\frac{\bar{\alpha}}{b+\gamma+\phi} > 1$.

Proof. From $\dot{i} = 0$ and $\dot{s} = 0$, we have one disease free steady state with $i^\infty = 0$ and $s^\infty = 1$. From equation (17), we have

$$\mu_3 = \lambda_1 - \alpha'(h)si(\lambda_2 - \lambda_3) = \lambda_1 > 0$$

Therefore, μ_3 is strictly positive and implies $h^\infty = 0$. From equation (18), we have

$$\frac{\dot{\lambda_1}}{\lambda_1} = -[f_1(k, 1) - \delta - b + d].$$

Moreover, from equation (16), we have

$$\frac{\dot{\lambda_1}}{\lambda_1} = -(\rho - b + d) + \frac{u''(c)}{u'(c)}\dot{c}.$$

[20] Goenka, Liu and Nguyen (2014) prove sufficiency in a SIS model without disease related mortality and Goenka, Liu and Nguyen (2020) show sufficiency in a SIS model with disease related mortality. These are the only sufficiency conditions for optimal control in economic epidemiology models we are aware of.

Since the economy is bounded, all economic variables including k, c and l are constant in the steady state. That is, $\dot{c} = 0$ in the steady state. Thus, combing the above two equations, we have

$$f_1(k, 1) = \rho + \delta,$$

from which we can solve for k^∞. □

Definition 1. *Define the function* $G^I(h)$ *:*

$$G^I(h) = \alpha'(h)si(\tilde{\lambda}_2 - \tilde{\lambda}_3) - u'(c), \tag{22}$$

where

$$\alpha(\alpha - \phi)s^2 - [\alpha(\gamma + b + \phi) - \phi(\phi + \gamma)]s + b\phi = 0 \tag{23}$$

$$\alpha s + \phi i = \phi + \gamma + b \tag{24}$$

$$f_1(k, 1 - i) = \rho + \delta \tag{25}$$

$$f(k, 1 - i) = c + h + \delta k + (b - d - \phi i)k \tag{26}$$

$$M_1 = \chi\phi\nu'(\phi i) + u'(c)[f_2(k, 1 - i) - k\phi] + \phi\frac{(u(c) - \chi\nu(\phi i))}{\rho - b + d + \phi i} \tag{27}$$

$$\tilde{\lambda}_2 = \frac{M_1(\alpha(h)i + \rho + d)}{(\alpha(h)s - \rho - \gamma - d - \phi + \phi i)(\alpha(h)i + \rho + d) - (\alpha(h)s - \phi s)\alpha(h)i} \tag{28}$$

$$\tilde{\lambda}_3 = \frac{M_1\alpha(h)i}{(\alpha(h)s - \rho - \gamma - d - \phi + \phi i)(\alpha(h)i + \rho + d) - (\alpha(h)s - \phi s)\alpha(h)i}. \tag{29}$$

Proposition 4. *If* $\frac{\bar{\alpha}}{b+\gamma+\phi} > 1$ *and* $G^I(h)|_{h=0} \leq 0$*, there exists a disease endemic steady state with no health expenditure* $h^\infty = 0$*. Given the optimal* h^∞*, the steady state variables* $s^\infty, i^\infty, k^\infty$ *and* c^∞ *are determined by equations (23) - (26).*

Proof. If $\frac{\bar{\alpha}}{b+\gamma+\phi} \leq 1$, the infectious diseases are eradicated and only the disease free steady state exists. Therefore, the disease endemic steady states only exist when $\frac{\bar{\alpha}}{b+\gamma+\phi} > 1$.

From equation (17), we have

$$\mu_3 = \lambda_1 - \alpha'(h)si(\lambda_2 - \lambda_3),$$

where λ_1 is the marginal cost (MC) of health expenditure and $\alpha'(h)si(\lambda_2 - \lambda_3)$ is marginal benefit (MB) of health expenditure. When $G^I(h)|_{h=0} \leq 0$, that is, the MC is larger than the MB, we have a corner solution with $h^\infty = 0$ and $\alpha(h^\infty) = \bar{\alpha}$. Once we know h^∞, we can solve for the steady state $s^\infty, i^\infty, k^\infty$ and c^∞ with equations (23) - (26). □

Proposition 5. *If* $\frac{\bar{\alpha}}{b+\gamma+\phi} > 1$ *and* $G^I(h)|_{h=0} > 0$*, there exists a disease endemic steady state with positive health expenditure, which is determined by* $G^I(h^\infty) = 0$*. Given the optimal* h^∞*, the steady state variables* $s^\infty, i^\infty, k^\infty$ *and* c^∞ *are determined by equation (23) - (26).*

Proof. If $\frac{\bar{\alpha}}{b+\gamma+\phi} \leq 1$, the infectious diseases are eradicated and only the disease free steady state exists. Therefore, the disease endemic steady states only exist when $\frac{\bar{\alpha}}{b+\gamma+\phi} > 1$.

From equation (17), we have

$$\mu_3 = \lambda_1 - \alpha'(h)si(\lambda_2 - \lambda_3),$$

where λ_1 is MC of health expenditure and $\alpha'(h)si(\lambda_2 - \lambda_3)$ is MB of health expenditure. When $G^I(h)|_{h=0} > 0$, there is an interior solution ($h^\infty > 0$) determined by:

$$\lambda_1 = \alpha'(h)si(\lambda_2 - \lambda_3). \tag{30}$$

In the steady state, all economic variables converge to the steady state, and all co-state variables grow at the same rate with $\frac{\dot{\lambda_1}}{\lambda_1} = \frac{\dot{\lambda_2}}{\lambda_2} = \frac{\dot{\lambda_3}}{\lambda_3} = \frac{\dot{\lambda_4}}{\lambda_4}$.

From equation (16), we have

$$\frac{\dot{\lambda_1}}{\lambda_1} = -(\rho - b + d + \phi i).$$

Combined with equation (18), we have

$$f_1(k, 1-i) = \rho + \delta. \tag{31}$$

From equation (21), we have

$$\frac{\lambda_4}{\lambda_4} = \frac{e^{-\Delta}[u(c) - \chi\nu(\phi i)]}{\lambda_4} = -(\rho - b + d + \phi i)$$
$$\lambda_4 = \frac{e^{-\Delta}[u(c) - \chi\nu(\phi i)]}{-(\rho - b + d + \phi i)}$$

From equation (20), we have

$$\frac{\dot{\lambda_3}}{\lambda_3} = \alpha(h)i(1 - \frac{\lambda_2}{\lambda_3}) + [b - \phi i] = -(\rho - b + d + \phi i)$$

From equation (19), we have

$$\frac{\dot{\lambda_2}}{\lambda_2} = \frac{e^{-\Delta}\chi\phi\nu'(\phi i)}{\lambda_2} + \frac{\lambda_1[f_2(k,1-i)-k\phi]}{\lambda_2} - [\alpha(h)s - \gamma - b - \phi + 2\phi i] + \\ + \frac{\lambda_3}{\lambda_2}[\alpha(h)s - \phi s] - \frac{\lambda_4\phi}{\lambda_2}$$

Define

$$M_1 = \chi\phi\nu'(\phi i) + u'(c)[f_2(k,1-i)-k\phi] + \phi\frac{(u(c)-\chi\nu(\phi i))}{\rho - b + d + \phi i}$$

Then, we have

$$\tilde{\lambda}_2 = \frac{\lambda_2}{e^{-\Delta}} = \frac{M_1(\alpha(h)i+\rho+d)}{(\alpha(h)s-\rho-\gamma-d-\phi+\phi i)(\alpha(h)i+\rho+d)-(\alpha(h)s-\phi s)\alpha(h)i}$$
$$\tilde{\lambda}_3 = \frac{\lambda_3}{e^{-\Delta}} = \frac{M_1\alpha(h)i}{(\alpha(h)s-\rho-\gamma-d-\phi+\phi i)(\alpha(h)i+\rho+d)-(\alpha(h)s-\phi s)\alpha(h)i}$$

Substitute λ_1, λ_2 and λ_3 into equation (17), we have

$$G^I(h) = \alpha'(h)si(\tilde{\lambda}_2 - \tilde{\lambda}_3) - u'(c).$$

Then, we can solve for h^∞ with $G^I(h) = 0$. Once we know h^∞, we can solve for the steady state $s^\infty, i^\infty, k^\infty$ and c^∞ with equations (23) - (26).

□

Under assumption in Proposition 5, there exists an endemic steady state. We denote by the value of the contact rate around the steady state as $\tilde{\alpha} = \alpha(h^\infty)$.

Proposition 6. *If $\tilde{\alpha} > \phi + \gamma + b$ and $h^* \to h^\infty$ for any h^* in the neigborhood of h^∞, the steady state $s^\infty > 0, i^\infty > 0$ is unique with $s^* \to s^\infty$ and $i^* \to i^\infty$ when $t \to \infty$.*

As in Proposition 5, the steady state s^∞ is determined by the following equation $P(s) = 0$ where $P(s) = \tilde{\alpha}(\tilde{\alpha}-\phi)s^2 - [\tilde{\alpha}(\gamma+b+\phi) - \phi(\phi+\gamma)]s + b\phi$. We have $P(0) = b\phi > 0$. We take two points $\hat{s}_1 = \frac{b+\gamma}{\tilde{\alpha}} \in (0,1)$ and $\hat{s}_2 = \frac{b+\gamma+\phi}{\tilde{\alpha}} \in (0,1)$. Due to Assumption 1, $b > d + \phi \geq \phi$ and from the condition for existence of an endemic steady we have $\tilde{\alpha} > \phi+\gamma+b \geq \phi$. Thus,

$$P(\hat{s}_1) = \tilde{\alpha}(\tilde{\alpha}-\phi)(\frac{b+\gamma}{\tilde{\alpha}})^2 - [\tilde{\alpha}(\gamma+b+\phi) - \phi(\phi+\gamma)]\frac{b+\gamma}{\tilde{\alpha}} + b\phi$$
$$= -\frac{\phi}{\tilde{\alpha}}[b(b+\gamma-\phi) + \gamma(\tilde{\alpha}-\phi)] < 0.$$

Moreover,

$$\begin{aligned} P(\hat{s}_2) &= \tilde{\alpha}(\tilde{\alpha}-\phi)(\frac{b+\gamma+\phi}{\tilde{\alpha}})^2 - [\tilde{\alpha}(\gamma+b+\phi)-\phi(\phi+\gamma)]\frac{b+\gamma+\phi}{\tilde{\alpha}} + b\phi \\ &= \frac{b\phi}{\tilde{\alpha}}(\tilde{\alpha}-\phi-\gamma-b) > 0. \end{aligned}$$

Therefore, $P(0)P(\hat{s}_1) < 0, P(\hat{s}_1)P(\hat{s}_2) < 0$, the equation $P(s) = 0$ has two solutions s_1^∞ and s_2^∞ such that $0 < s_1^\infty < \hat{s}_1 < s_2^\infty < \hat{s}_2 < 1$.

Because the associated steady state i^∞ is given by

$$i = \frac{\phi+\gamma+b}{\phi} - \frac{\tilde{\alpha}}{\phi}s \in [0,1],$$

we have a condition for s_1^∞ and s_2^∞ :

$$\frac{b+\gamma}{\tilde{\alpha}} \leq s_{1,2}^\infty \leq \frac{b+\gamma+\phi}{\tilde{\alpha}}. \tag{32}$$

Therefore s_1^∞ violates the condition (32) since we have shown that $s_1^\infty < \hat{s}_1 = \frac{b+\gamma}{\tilde{\alpha}}$. There exists only the steady state $0 < s_2^\infty < \hat{s}_2 = \frac{b+\gamma+\phi}{\tilde{\alpha}}$. Thus, the associated steady state

$$i_2^\infty = \frac{\phi+\gamma+b}{\phi} - \frac{\tilde{\alpha}}{\phi}s_2^\infty > 0.$$

We now prove the convergence of i^* and s^* to the steady state when setting the value of the contact rate at $\tilde{\alpha}$.

The Jacobian matrix from equation (1) - (2) is

$$J = \begin{pmatrix} \alpha(h)s - \gamma - b - \phi + 2\phi i & \alpha(h)i \\ -\alpha(h)s + \phi s & -b - \alpha(h)i + \phi i \end{pmatrix}$$

At the steady state, $\dot{i} = \dot{s} = 0$ so $\tilde{\alpha}s - \gamma - b - \phi + 2\phi i = \phi i$ and $-b - \tilde{\alpha}i + \phi i = \frac{b-bs-\tilde{\alpha}si+\phi si}{s} - \frac{b}{s} = \frac{-b}{s}$. Then the matrix evaluated at steady state (i_2^∞, s_2^∞) is rewritten as follows

$$J = \begin{pmatrix} \phi i_2^\infty & \tilde{\alpha}i_2^\infty \\ -\tilde{\alpha}s_2^\infty + \phi s_2^\infty & \frac{-b}{s_2^\infty} \end{pmatrix}.$$

By assumption 1 we have

$$\text{Trace } J = \phi i_2^\infty - \frac{b}{s_2^\infty} < b - \frac{b}{s_2^\infty} = \frac{-b(1-s_2^\infty)}{s_2^\infty} < 0.$$

Let denote $P(s) = As^2 - Bs + \Gamma$ where

$$\begin{aligned} A &= \tilde{\alpha}(\tilde{\alpha} - \phi) > 0, \\ B &= \tilde{\alpha}(\gamma + b + \phi) - \phi(\phi + \gamma) = (\tilde{\alpha} - \phi)(\phi + \gamma) + \tilde{\alpha}b > 0, \\ \Gamma &= b\phi > 0 \end{aligned}$$

which satisfies $B^2 > 4A\Gamma$ and $A(s_2^\infty)^2 = Bs_2^\infty - \Gamma$. Thus

$$\begin{aligned} \det J &= i_2^\infty[\frac{-\phi b}{s_2^\infty} + \tilde{\alpha}(\tilde{\alpha} - \phi)s_2^\infty)] \\ &= i_2^\infty \frac{-\Gamma + A(s_2^\infty)^2}{s_2^\infty} = i_2^\infty \frac{Bs_2^\infty - 2\Gamma}{s_2^\infty}. \end{aligned}$$

Moreover, the steady state s_2^∞ is the larger solution so

$$s_2^\infty > \frac{s_1^\infty + s_2^\infty}{2} = \frac{B}{2A}$$

which implies

$$Bs_2^\infty - 2\Gamma > \frac{B^2}{2A} - 2\Gamma = \frac{B^2 - 4A\Gamma}{2A} > 0.$$

Therefore we have $\det J > 0$ and Trace $J < 0$. The matrix J has two negative eigenvalues and hence the steady state (i_2^∞, s_2^∞) is stable.

We know that a steady state exists and this Proposition shows that the epidemiological variables, s, i in the economic epidemiology model which satisfy the first order necessary conditions converge to the unique steady state value of these variables. Busenberg and van den Driessche (1990) also show convergence of the pure epidemiology model to a steady state which is unique. As we have to work with an α that varies along a trajectory satisfying the first order conditions we need verify this for the economic epidemiology model. Our argument is different and we also show that the steady state is unique.

3.2 Model II: Delayed Mortality

In model II, the growth rate of total population is $b - d - \phi(1 - s - i)$. Then, the law of motion for physical capital can be rewritten as:

$$\dot{k} = f(k, 1 - i) - c - h - \delta k - (b - d - \phi(1 - s - i))k. \tag{33}$$

As before the objective is to maximize total welfare. Each household derives utility from consumption goods - $u(c)$ and disutility from disease related death - $\nu(\phi(1 - s - i))$ with a weight χ, which is non-negative. Each household discounts future at the rate ρ. The

objective function is:

$$\max \int_0^\infty e^{-\rho t}[u(c) - \chi\nu(\phi(1-s-i))]Ndt \tag{34}$$

$$= \max \int_0^\infty e^{-\Delta}[u(c) - \chi\nu(\phi(1-s-i))]N_0 dt \tag{35}$$

where

$$\begin{aligned} \Delta &= \int_0^t (\rho - b + d + \phi(1 - s(\tau) - i(\tau)))d\tau \\ \dot{\Delta} &= \rho - b + d + \phi(1-s-i). \end{aligned} \tag{36}$$

Thus, the social planner maximizes equation (35) subject to equation (33), (12), (6), (7) and (36) with the constraints

$$i \geq 0, \quad s \leq 1, \quad h \geq 0.$$

The control variables are c, h, and state variables are k, i, s, Δ. Define $\lambda_1, \lambda_2, \lambda_3$ and λ_4 are co-state variables for k, i, s and Δ, respectively. μ_1, μ_2 and μ_3 are Lagrangian multipliers for $i \geq 0$, $s \leq 1$ and $h \geq 0$.

The F.O.Cs are as follows:

$$c: \quad e^{-\Delta}u'(c) = \lambda_1 \tag{37}$$

$$h: \quad \lambda_1 = \alpha'(h)si(\lambda_2 - \lambda_3) + \mu_3 \tag{38}$$

$$k: \quad \dot{\lambda_1} = -\lambda_1[f_1(k, 1-i) - \delta - (b - d - \phi(1-s-i))] \tag{39}$$

$$i: \quad \dot{\lambda_2} = -e^{-\Delta}\chi\phi\nu'(\phi(1-s-i)) + \lambda_1(f_2(k,1-i) + k\phi) - \lambda_2[\alpha(h)s - \gamma - b + \phi(1-s-i) - \phi i] + \\ + \lambda_3(\alpha(h)s + \phi s) + \lambda_4\phi - \mu_1 \tag{40}$$

$$s: \quad \dot{\lambda_3} = -e^{-\Delta}\chi\phi\nu'(\phi(1-s-i)) + \lambda_1 k\phi - \lambda_2[\alpha(h)i - \phi i] + \lambda_3[b + \alpha(h)i - \phi(1-s-i) + \phi s] + \\ + \lambda_4\phi + \mu_2 \tag{41}$$

$$\Delta: \quad \dot{\lambda_4} = e^{-\Delta}[u(c) - \chi\nu(\phi(1-s-i))] \tag{42}$$

$$\mu_1 \geq 0, \quad i \geq 0, \quad \mu_1 i = 0$$
$$\mu_2 \geq 0, \quad s \leq 1, \quad \mu_2(1-s) = 0$$
$$\mu_3 \geq 0, \quad h \geq 0, \quad \mu_3 h = 0$$

3.2.1 Steady States

Proposition 7. *There always exists a unique disease free steady state with $s^\infty = 1$, $i^\infty = 0$ and $h^\infty = 0$. The economic variables k^∞ and c^∞ are determined by*

$$f_1(k, 1) = \rho + \delta$$
$$f(k, 1) = c + \delta k + (b - d)k.$$

The disease free steady state is stable when $\frac{\bar{\alpha}}{b+\gamma+\phi} \leq 1$*, and unstable when* $\frac{\bar{\alpha}}{b+\gamma+\phi} > 1$*.*

Proof. The proof is omitted here, as it is the same as the proof for the disease free steady state in Model I. □

Definition 2. *Define a function* $G^{II}(h)$ *:*

$$G^{II}(h) = \alpha'(h)si(\tilde{\lambda}_2 - \tilde{\lambda}_3) - u'(c), \tag{43}$$

where

$$\alpha^2 s^2 - [\alpha(\gamma + b - \phi) + \phi\gamma]s - b\phi = 0 \tag{44}$$

$$i = \frac{b - bs + \phi s - \phi s^2}{s(\alpha + \phi)} \tag{45}$$

$$f_1(k, 1-i) = \rho + \delta \tag{46}$$

$$f(k, 1-i) = c + h + \delta k + (b - d - \phi(1 - s - i))k \tag{47}$$

$$M_2 = -\chi\phi\nu'(\phi(1-s-i)) + u'(c)k\phi - \frac{\phi(u(c) - \chi\nu(\phi(1-s-i)))}{(\rho - b + d + \phi(1-s-i))} \tag{48}$$

$$M_3 = -\chi\phi\nu'(\phi(1-s-i)) + u'(c)[f_2(k, 1-i) + k\phi] - \frac{\phi(u(c) - \chi\nu(\phi(1-s-i)))}{(\rho - b + d + \phi(1-s-i))} \tag{49}$$

$$\tilde{\lambda}_2 = \frac{M_2(\alpha(h)s + \phi s) - M_3(\alpha(h)i + \rho + d + \phi s)}{(\alpha(h)i - \phi i)(\alpha(h)s + \phi s) - (\alpha(h)i + \rho + d + \phi s)(\alpha(h)s - \rho - \gamma - d - \phi i)} \tag{50}$$

$$\tilde{\lambda}_3 = \frac{M_2(\alpha(h)s - \rho - \gamma - d - \phi i) - M_3(\alpha(h)i - \phi i)}{(\alpha(h)i - \phi i)(\alpha(h)s + \phi s) - (\alpha(h)i + \rho + d + \phi s)(\alpha(h)s - \rho - \gamma - d - \phi i)}. \tag{51}$$

Proposition 8. *If* $\frac{\bar{\alpha}}{b+\gamma+\phi} > 1$ *and* $G^{II}(h)|_{h=0} \leq 0$*, there exists a disease endemic steady state with no health expenditure* $h^\infty = 0$*. Given the optimal* h^∞*, the steady state variables* $s^\infty, i^\infty, k^\infty$ *and* c^∞ *are determined by equations (44) - (47).*

Proof. The proof is omitted here as it is similar to the proof for the disease endemic steady state with corner solution in Model I. □

Proposition 9. *If* $\frac{\bar{\alpha}}{b+\gamma+\phi} > 1$ *and* $G^{II}(h)|_{h=0} > 0$*, there exists a disease endemic steady state with positive health expenditure, which is determined by* $G^{II}(h^\infty) = 0$*. Given the optimal* h^∞*, the steady state variables* s^∞, i^*, k^∞ *and* c^∞ *are determined by equation (44) - (47).*

Proof. The proof is brief here, as it is similar to the proof for the disease endemic steady state with interior solution in Model I.

From equation (37) and(39), we have

$$f_1(k, 1-i) = \rho + \delta.$$

From equation (40) and (42), we have

$$\lambda_4 = \frac{e^{-\Delta}[u(c) - \chi\nu(\phi(1-s-i))]}{-(\rho - b + d + \phi(1-s-i))}.$$

From equation (41) and (40), we have

$$\begin{aligned}
\frac{\dot{\lambda_2}}{\lambda_2} &= \frac{-e^{-\Delta}\chi\phi\nu'(\phi(1-s-i))}{\lambda_2} + \frac{\lambda_1[f_2(k,1-i)+k\phi]}{\lambda_2} - [\alpha(h)s - \gamma - b + \phi(1-s-i) - \phi i] + \\
&+ \frac{\lambda_3}{\lambda_2}[\alpha(h)s + \phi s] + \frac{\lambda_4\phi}{\lambda_2} \\
\frac{\dot{\lambda_3}}{\lambda_3} &= \frac{-e^{-\Delta}\chi\phi\nu'(\phi(1-s-i))}{\lambda_3} + \frac{\lambda_1 k\phi]}{\lambda_3} + [b + \alpha(h)i - \phi(1-s-i) + \phi s] + \\
&- \frac{\lambda_2}{\lambda_3}[\alpha(h)i - \phi i] + \frac{\lambda_4\phi}{\lambda_3}
\end{aligned}$$

Define

$$\begin{aligned}
M_2 &= -\chi\phi\nu'(\phi(1-s-i)) + u'(c)k\phi - \frac{\phi(u(c) - \chi\nu(\phi(1-s-i)))}{(\rho - b + d + \phi(1-s-i))} \\
M_3 &= -\chi\phi\nu'(\phi(1-s-i)) + u'(c)[f_2(k,1-i)+k\phi] - \frac{\phi(u(c) - \chi\nu(\phi(1-s-i)))}{(\rho - b + d + \phi(1-s-i))}.
\end{aligned}$$

Then, we have

$$\begin{aligned}
\tilde{\lambda}_2 &= \frac{\lambda_2}{e^{-\Delta}} = \frac{M_2(\alpha(h)s + \phi s) - M_3(\alpha(h)i + \rho + d + \phi s)}{(\alpha(h)i - \phi i)(\alpha(h)s + \phi s) - (\alpha(h)i + \rho + d + \phi s)(\alpha(h)s - \rho - \gamma - d - \phi i)} \\
\tilde{\lambda}_3 &= \frac{\lambda_3}{e^{-\Delta}} = \frac{M_2(\alpha(h)s - \rho - \gamma - d - \phi i) - M_3(\alpha(h)i - \phi i)}{(\alpha(h)i - \phi i)(\alpha(h)s + \phi s) - (\alpha(h)i + \rho + d + \phi s)(\alpha(h)s - \rho - \gamma - d - \phi i)}
\end{aligned}$$

Then, we can substitute λ_1, λ_2 and λ_3 into equation (38) and get

$$G^{II}(h) = \alpha'(h)si(\tilde{\lambda}_2 - \tilde{\lambda}_3) - u'(c).$$

Then, we can solve for h^∞ with $G^I(h) = 0$. Once we know h^∞, we can solve for the steady state $s^\infty, i^\infty, k^\infty$ and c^∞ with equations (44) - (47). □

Proposition 10. *If an endemic steady state exists, i.e. when $\tilde{\alpha} > \phi + \gamma + b$, the steady state*

is unique $s^\infty > 0$ *and* $i^\infty > 0$ *with* $s^* \to s^\infty$ *and* $i^* \to i^\infty$ *when* $t \to \infty$.

Proof. The proof follows that of Proposition 6 and is omitted for brevity. □

4 Comparison on Effects of Varying Disease Induced Mortality Rate

The marriage of the economic and epidemiological models provides us a framework in understanding the close link between the the economy and disease prevalence. As the model is too complex for closed form solutions, in this section, we calibrate the model and run comparative statics. We focus on examining the effects of varying disease induced mortality rate ϕ, rather than other parameters, for instance birth rate, recovery rate and etc. The reason is that the effects of varying other parameters are similar in both models. In the paper, we want to highlight the difference in model implications when we model disease related mortality differently. Moreover, the analysis here focuses on the equilibrium steady states before and after the change as we want to capture the medium to longer term effects when investment and returns to labor and capital have adjusted.

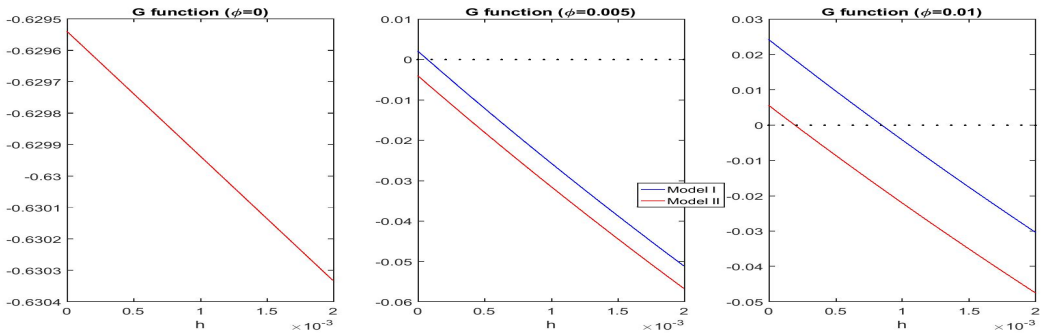
The following functional forms and parameters are chosen in line with the literature. The production function is assumed to be Cobb Douglas: $y = f(k, 1-i) = Ak^\beta(1-i)^{1-\beta}$ with $A = 1$ and $\beta = 0.36$. Physical capital depreciates at the rate $\delta = 0.05$ and discount rate $\rho = 0.055$. The utility function is of the CES form $U(c) = \frac{c^{1-\sigma}}{1-\sigma}$ and we choose $\sigma = 1$, that is, the utility function is log utility. We assume the disutility from disease induced death takes the form: $\nu(D) = D^{\nu_0}$ with $\nu_0 = 1.2$ in line with the assumptions in the paper. In the simulation, we vary the weight we attach to this disutility χ. We set the birth rate $b = 2\%$ and death rate $d = 1\%$. The recovery rate is $\gamma = 0.1$.

We do not have enough empirical evidence in suggesting for functional form for contact rate function. Therefore, the contact rate function is chosen in line with the assumption on $\alpha(h)$ and large enough to generate an endemic steady state in the simulation. We assume contact rate function is a power function: $\alpha(h) = \epsilon_0(h + \epsilon_2)_1^\epsilon$ with $\epsilon_0 = 0.2, \epsilon_1 = -0.2$ and $\epsilon_2 = 0.0215$.

Figure 4 depicts G function for both model I and II with different disease mortality rates ϕ. The disutility weight $\chi = 0$, that is, the objective is the standard utility which only depends on the level of consumption. [21] The blue color line is the simulation for model I, while the red color line is the simulation for model II. For all the three panels, when health expenditure h increases, the net marginal benefit of health expenditure decreases. Moreover, comparing the three panels, we find that with high disease related mortality ϕ, the net marginal benefit of health expenditure in controlling disease prevalence is higher. The first panel is for the case when $\phi = 0$. As we know, when $\phi = 0$, that is, there is no disease related mortality, both models are essentially the same, and the blue and red color lines coincide. And when $h = 0$, the net marginal benefit of health expenditure is negative. It implies marginal benefit of health expenditure is smaller than marginal cost, and the optimal health expenditure is zero. Thus, we have an endemic steady state with

[21] We plot G with $\chi > 0$ below.

Figure 4. A depiction of G function in Model I and II ($\chi = 0$)



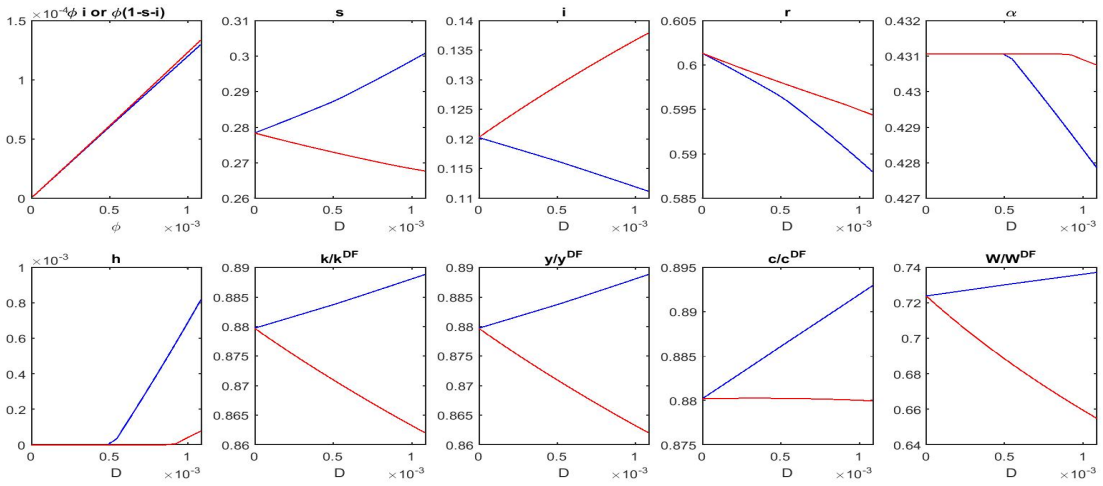
The figure depicts G function, which is the marginal benefit of health expenditure minus the marginal cost of health expenditure. The blue color line is the simulation for model I, while the red color line is the simulation for model II. $\chi = 0$

corner solution, in both models. The second panel is for the case when $\phi = 0.5\%$. For given level of health expenditure h, the net marginal benefit is higher for model I than model II. And when $h = 0$, the net marginal benefit of health expenditure is positive in model I, and it implies we have an endemic steady state with interior solution. In contrast, when $h = 0$, the net marginal benefit of health expenditure is negative in model II, and it implies we have an endemic steady state with corner solution. The third panel is for the case when $\phi = 1\%$. And when $h = 0$, the net marginal benefit of health expenditure is positive in both models, and it implies we have an endemic steady state with interior solution in both models. For given level of health expenditure h, the net marginal benefit is higher for model I than model II. Thus, model I has larger health expenditure in the steady state than model II.

To further investigate how the steady state variables change as we change the disease induced mortality rate, we provide simulation results in Figure (5) and (7). Figure (5) is the scenario when disutility weight $\chi = 0$, while Figure (7) is the scenario when disutility weight χ is positive. Again, the blue color line is the simulation for model I, while the red color line is the simulation for model II. In all panels except the left panel of the top row, we plot change in steady state variables against change in disease induced death rate in the total population, that is, $D = \phi i$ in model I and $D = \phi(1 - s - i)$ in model II, rather than against the disease induced death rate ϕ. The left panel of the top row shows that the disease induced death rate in the total population D increases almost one by one, as disease induced death rate ϕ increases in both models. Thus, it does not matter much if we plot all the rest panels in terms of D or ϕ. However, for easy comparison and explanation, we choose to plot in term of the disease induced death rate in the total population D.

Figure (5) is the scenario when the household only derives utility from consumption. The prevalence of infectious diseases affect the economy mainly because of three reasons: (i) infected can not participate in labor force, (ii) that investing in health in controlling diseases can squeeze the economic resources and (iii) the disease related mortality changes population size. This has two consequences: first, the per capita amount of resources change and second, the change in population affects the discount rate. When there is no death from

Figure 5. The simulation for Model I and II ($\chi = 0$)



The figure shows changes in steady state as we vary disease induced mortality rate ϕ. The blue color line is the simulation for model I, while the red color line is the simulation for model II. Here, the weight $\chi = 0$.

diseases ($\phi = 0$), both models are the same, which predict zero health expenditure as the net marginal benefit of health expenditure is negative, shown in Figure 4. As the disease induced death rises, there is an increasing incentive in investing in heath expenditure to control disease spreading. It is more so in model I than in model II, shown in the left panel of the bottom row. When disease death rate rises gradually, health expenditure becomes positive in model I, and when death rate rises further, health expenditure becomes positive in model II. This change in health expenditure directed mirrors change in contact rate α.

What we find more interesting is the sharp difference in change of steady states as disease death rate rises, when we compare the two models in Figure (5). When disease death rate D rises, in model I the proportion of susceptible (s) increases and the proportion of the infected (i) decreases. This is consistent with the epidemiological model. When there is additional death from disease, the total population decreases, which leads to an increase in the proportion of susceptibles. Increase in mortality of infectives decreases the proportion of infectives in the population. This is the self-limiting effect of mortality on pandemics. However, in the economic model the health expenditure will also change so the effect is not immediately obvious. To explore the change in economic variables, we plot them as a proportion of the disease free steady state, which implies how much worse off of the disease endemic steady state is as compared to the disease free steady state. As we can see, for model I, as disease death rate D increases, physical capital stock, total output, consumption and total welfare all increase. This seems to be counter intuitive. In fact, this is like a gift of dying effect. In model I, the individuals who die due to infectious diseases are those who are infected, infectious and can not work, but consume. So from a purely economic point of view (when the objective is only about maximizing consumption per capita), the society is better off when disease mortality rate is high. However, the phenomena of gift of dying is

absent in model II. In model II, when disease mortality rate D increases, the proportion of the susceptible decreases and the proportion of the infected increases, which is in line with the epidemiological model. Moreover, when disease death rate D increases, physical capital stock, total output, consumption and total welfare all decrease. This is because in model II, increasing mortality does not limit the spread of infections and the pandemic gets worse rather than better. This effect not only directly affects per capita availability of resources but also increases discounting. Thus, this leads to lower investment in capital and in health.

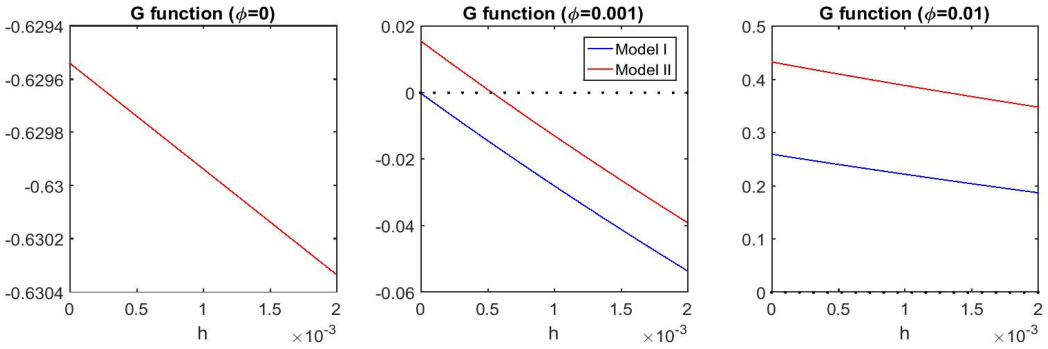
We now look at the case where there is a welfare loss from mortality, $\chi > 0$. We plot the case of $\chi = 100$ in Figure 6.[22]. Now as compare to Figure 4 the position of the G functions reverse in model I and model I with increasing mortality rate ϕ. (The first panel is for $\phi = 0$ where the two models are the same.) As ϕ becomes positive and increase the net marginal benefit of health expenditure in model II is always higher. The intuition is that the increase in mortality has no self-limiting effect on the pandemic in model II, and as mortality loss leads to a drop in welfare, the net marginal benefit is also higher. This gap widens the larger is the weight on welfare loss from mortality.

Figure 7 differs from Figure 5 in that Figure 7 incorporates directly the disutility from disease related death, where we set $\chi = 100$. There are two main differences between Figure 5 and 7. One is the change in health expenditure. When disease death rate D rises, it is more likely for model II to have a positive health expenditure. The higher health expenditure leads to a larger fall in the contact rate α in model II. The other difference is the change in total welfare. For model I, even though physical capital, total output and consumption rise as disease death rate D increases, the total welfare decreases as D increases. This is because with positive weight on disease related death, the rise in disutility from death outweighs the rise in utility from consumption, which leads the total welfare to fall. For model II, as mortality rate increases, the infections decrease as health expenditure rises more sharply. The effect on total number of deaths is similar. As there is no self-limiting effect of mortality on infections, the proportion of infective increases which decreases output (more individuals not working) and consumption. The capital stock decreases in model II as opposed to model I as output is decreasing and health expenditure increasing which depresses investment. There is a sharper fall in welfare in model II primarily from the fall in consumption (recall that mortality loss in both models are similar).

To sum up, differences in epidemiological modeling matters not only for disease evolution but also the differing implications for evolution of the disease has major differences for the equilibrium economic outcomes. As the difference in modeling mortality affects evolution of the infective population, the optimal health expenditure is affected. To what extent depends on how much weight is given to the welfare loss due to mortality. This affects other economic outcomes and the welfare in equilibrium. Thus, there is an a non-trivial interaction between economic and epidemiological choices in understanding the effects of pandemics on welfare and economic outcomes.

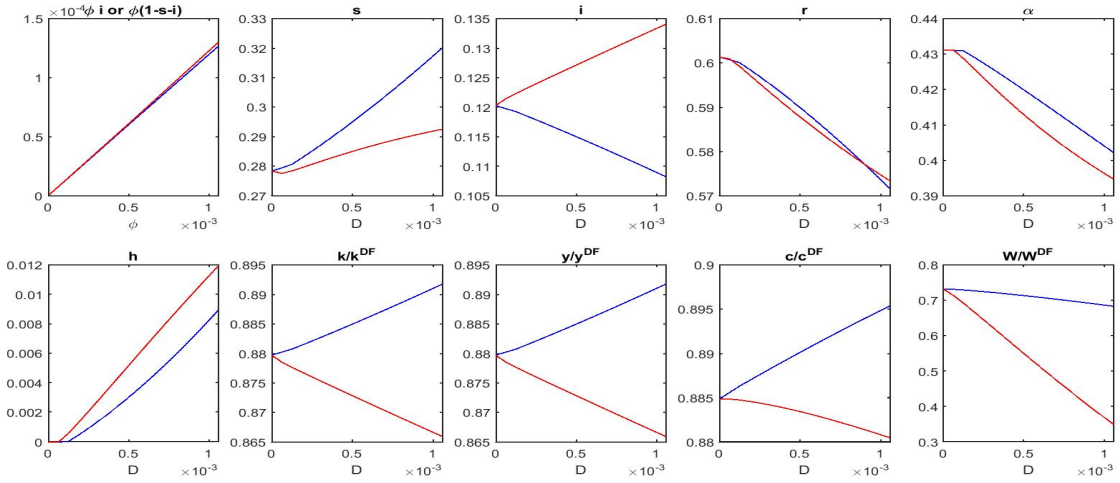
[22]We do not plot additional figures for more values of χ as the results are qualitatively similar.

Figure 6. A depiction of G function in Model I and II ($\chi = 100$)



The figure depicts G function, which is the marginal benefit of health expenditure minus the marginal cost of health expenditure. The blue color line is the simulation for model I, while the red color line is the simulation for model II. $\chi = 100$

Figure 7. The simulation for Model I and II ($\chi = 100$)



The figure shows changes in steady state as we vary disease induced mortality rate ϕ. The blue color line is the simulation for model I, while the red color line is the simulation for model II. Here, the weight $\chi = 100$.

5 Sufficient conditions

In this section we study the sufficiency of the first order conditions with disease related mortality. It is well known in the literature that with SIS or SIR dynamics the constraints are not convex and it is unclear if either the Arrow or the Mangasarian sufficiency conditions will be satisfied (Gersovitz and Hammer (2003))). Goenka, Liu and Nguyen (2014) provided a sufficiency result in a neo-classical framework, such as in the current paper, with SIS dynamics but no disease mortality.[23] and Goenka, Liu and Nguyen (2020) show sufficiency in the SIS with disease related mortality. We give the first sufficiency result for the SIR model. The result is with disease related mortality and the case without disease related mortality is a special case. We give the result for Model I, and the proof for Model II is similar and omitted. The problem is non-trivial because including disease related mortality effectively makes the effective discount rate, Δ, endogenous. The Hamiltonian is non-concave so in this situation the Arrow and Mangasarian conditions do not apply (see below) as well as conditions for endogenous population models that are convex (for example Boucekkine, et al. (2018)). While the proof follows the strategy of the SIS disease related model, there are now two state variables for disease evolution, s, i as opposed to only one for the SIS model. The control variable h is also different from the control variable for quarantine in the same paper. In the SIR model one has to also due to the presence of two state variables s, i for the disease we cannot directly show the appropriate transversality condition directly as in the SIS model and have to convergence of these variables to steady state values.

We directly show the inequality of local optimality of the Hamiltonian along *any* interior path that satisfies the first order necessary and transversality conditions. This is done by adapting the method of Leitmann and Stalford (1971). As a corollary, the disease endemic steady state will be locally optimal. Optimality of the disease free steady state is not in question as it is the neoclassical steady state.

Denote the state variables $\mathbf{x}_t^* = (k_t^*, i_t^*, s_t^*, \Delta_t^*)$ where $\mathbf{x}_0^* = (k_0^*, i_0^*, s_0^*, \Delta_0^*)$, the control variables $\mathbf{z}_t^* = (c_t^*, h_t^*)$ and co-state variables $\lambda_t = (\lambda_{1,t}, \lambda_{2,t}, \lambda_{3,t}, \lambda_{4,t})$.

The Hamiltonian becomes

$$\begin{aligned} H(\mathbf{x}_t, \mathbf{z}_t, \lambda_t) &= e^{-\Delta}[u(c) - \chi\nu(\phi i)] + \lambda_1\{f(k, (1-i) - c - h - \delta k - (b - d - \phi i)k\} + \\ &\quad + \lambda_2\{\alpha(h)si - \gamma i - bi - \phi i + \phi i^2\} + \lambda_3\{b - bs - \alpha(h)si + \phi si\} + \lambda_4\{\rho - b + d + \phi i\} \\ &= e^{-\Delta}[u(c) - \chi\nu(\phi i)] + < \lambda_t, \dot{\mathbf{x}}_t > \end{aligned}$$

where $< \mathbf{x}, \mathbf{y} >= \sum_1^n x_j y_j$ is the dot product of two vectors $\mathbf{x} = (x_1, .., x_n), \mathbf{y} = (y_1, ..., y_n)$.

[23] This paper also included the additional state variable health capital which can reduce contact rate and increase recovery rate.

The first-order necessary conditions are satisfied at $(\mathbf{x}_t^*, \mathbf{z}_t^*)$

$$e^{-\Delta}u'(c) = \lambda_1 \tag{52}$$
$$\lambda_1 = \alpha'(h)si(\lambda_2 - \lambda_3) \tag{53}$$
$$-\dot{\lambda_1} = \lambda_1[f_1(k, 1-i) - \delta - b + d + \phi i] \tag{54}$$
$$-\dot{\lambda_2} = e^{-\Delta}\chi\phi\nu'(\phi i) + \lambda_1(f_2(k, 1-i) - k\phi) - \lambda_2(\alpha(h)s - \gamma - b - \phi + 2\phi i) + \\ +\lambda_3(\alpha(h)s - \phi s) - \lambda_4\phi \tag{55}$$
$$\dot{\lambda_3} = \alpha(h)i(\lambda_3 - \lambda_2) + \lambda_3(b - \phi i) \tag{56}$$
$$\dot{\lambda_4} = e^{-\Delta}[u(c) - \chi\nu(\phi i)] \tag{57}$$

The standard transversality conditions are

$$\lim_{t\to\infty} \lambda_{j,t}x_{j,t}^* = 0, j = 1, .., 3. \tag{58}$$

Remark 1. The Hamiltonian is not jointly concave in state and control variables if the welfare function is positive, i.e. if $u(c) - \nu(\phi i) > 0$. In particular, the condition for the Hessian matrix to be semi-negative definite which requires the principal minors $M_j (j = 1, ..., 6)$ alternate in sign, starting with a negative determinant does not satisfied in our model.

Let us rewrite the Hamiltonian as $H(k, i, \Delta, s, h, c)$ then it is easy to check, the first minor $M_1 = |H_{kk}| = \lambda_1 f_{11} < 0$ and suppose that $M_2 = \begin{vmatrix} H_{kk} & H_{ki} \\ H_{ik} & H_{ii} \end{vmatrix} > 0$. We then have

$$\begin{aligned} M_3 &= \begin{vmatrix} H_{kk} & H_{ki} & H_{k\Delta} \\ H_{ik} & H_{ii} & H_{i\Delta} \\ H_{\Delta k} & H_{\Delta i} & H_{\Delta\Delta} \end{vmatrix} = \begin{vmatrix} H_{kk} & H_{ki} & 0 \\ H_{ik} & H_{ii} & H_{i\Delta} \\ 0 & H_{\Delta i} & H_{\Delta\Delta} \end{vmatrix} \\ &= H_{\Delta\Delta}M_2 + (-1)^{2+3}H_{\Delta i}\begin{vmatrix} H_{kk} & 0 \\ H_{ik} & H_{i\Delta} \end{vmatrix} \\ &= H_{\Delta\Delta}M_2 - H_{\Delta i}^2 H_{kk}. \end{aligned}$$

Because $H_{\Delta\Delta} = e^{-\Delta}[u(c) - \chi\nu(\phi i)] > 0, H_{kk} < 0$, we have

$$M_3 = H_{\Delta\Delta}M_2 - H_{\Delta i}^2 H_{kk} > 0 \text{ if } M_2 > 0.$$

So the condition for the Hessian being semi-negative definite fails.

It is standard that $0 \leq k_t \leq \max\{k_0, \hat{k}\}$ where $\hat{k}$ is the maximum sustainable capital

stock[24]. Then is c_t is bounded by a constant[25], $c_t \leq A$, and hence

$$u(c) - \chi\nu(\phi i) \leq u(A) + \chi\nu(\phi) < +\infty \tag{59}$$

Similarly, h_t is also bounded by A.

The proof proceeds via three Lemmas.

Lemma 1. *We have*

$$\lim_{t\to\infty} \lambda_{4,t}(\Delta_t - \Delta_t^*) = 0.$$

Proof. Consider any feasible path $(\mathbf{x}_t, \mathbf{z}_t)$ with the same initial condition $\mathbf{x}_0^*$.

It follows from (57) that

$$\lambda_{4,t} = \lambda_{4,0} + \int_0^t e^{-\Delta_\tau^*}[u(c_\tau^*) - \chi\nu(\phi i_\tau^*)]d\tau.$$

The transversality condition (70) implies

$$\lim_{t\to\infty}[\lambda_{4,0} + \int_0^t e^{-\Delta_\tau^*}[u(c_\tau^*) - \chi\nu(\phi i_\tau^*)]d\tau]\Delta_t^* = 0.$$

Since $\lim_{t\to\infty} \Delta_t^* = +\infty$, the identity above is satisfied only if

$$\lambda_{4,0} = -\int_0^t e^{-\Delta_\tau^*}[u(c_\tau^*) - \chi\nu(\phi i_\tau^*)]d\tau$$

which in turn implies

$$\begin{aligned}\lambda_{4,t} &= -\int_0^\infty e^{-\Delta_\tau^*}[u(c_\tau^*) - \chi\nu(\phi i_\tau^*)]d\tau + \int_t^0 -e^{-\Delta_\tau^*}[u(c_\tau^*) - \chi\nu(\phi i_\tau^*)]d\tau \\ &= -\int_t^\infty e^{-\Delta_\tau^*}[u(c_\tau^*) - \chi\nu(\phi i_\tau^*)]d\tau.\end{aligned}$$

[24]Definition of maximal capital stock is $\hat{k} \in (0, \infty)$ such that $f(k, l) > k$ for all $k \in (0, \hat{k})$ and $f(k, l) < k$ for all $k > \hat{k}$. It implies $k \leq \max\{k_0, \hat{k}\} := \bar{k}$.

[25]If investement is irreversible, then $c_t \leq f(k_t, l_t) \leq f(\hat{k}, 1) := A$. Otherwise, as in Goenka, Liu and Nguyen (2014), we can assume that there exists $\kappa \geq 0, \kappa \neq \infty$ such that $-\kappa \leq \dot{k}/k$. This reasonable assumption implies that it is not possible that the growth rate of physical capital converges to $-\infty$ rapidly and is weaker than those used in the literature (see, e.g. Chichilnisky (1981)). Let us define the net investment $\iota = \dot{k} + (\delta + b - d)k = f(k, l) - c - m$, it then implies there exists $\kappa \geq 0, \kappa \neq \infty$ such that $\iota + [\kappa - (\delta + b - d)]k \geq 0$. If the standard assumption 2 (v) in Chichilnisky (1981) holds (non-negative investment, $\iota \geq 0$) then it holds with $\kappa = \delta + b - d$. Therefore, assuming non-negative investment is stronger in the sense that κ can take any value except for infinity. And we have $c_t \leq f(\bar{k}, 1) + \kappa\bar{k} := A$.

For any Δ, since $d\Delta = (\rho - b + d + \phi i)dt$ we have

$$\int_t^\infty e^{-\Delta_\tau} d\tau = \int_t^\infty \frac{e^{-\Delta_\tau} d\Delta_\tau}{\rho - b + d + \phi i_\tau}.$$

Let denote $q_\tau = \Delta_\tau$, if $\tau = t$ then $q_t = \Delta_t$. If $\tau = \infty$ then $q_\infty = \Delta_\infty = \infty$.
Since $0 \leq i \leq 1$ we get

$$\begin{aligned} & \frac{1}{\rho - b + d + \phi} \int_{\Delta_t}^\infty e^{-q} dq \leq \int_t^\infty e^{-\Delta_\tau} d\tau \leq \frac{1}{\rho - b + d} \int_{\Delta_t}^\infty e^{-q} dq \\ \Leftrightarrow \quad & \frac{e^{-\Delta_t}}{\rho - b + d + \phi} \leq \int_t^\infty e^{-\Delta_\tau} d\tau \leq \frac{e^{-\Delta_t}}{\rho - b + d}. \end{aligned} \tag{60}$$

It follows from (59), (60) and using the l'Hôpital's rule we have

$$\begin{aligned} 0 \quad \leq \quad & \lim_{t\to\infty} \Delta_t \int_t^\infty e^{-\Delta_\tau^*} [u(c_\tau^*) - \chi\nu(\phi i_\tau^*)] d\tau \leq (u(A) + \chi\nu(\phi)) \lim_{t\to\infty} \Delta_t \int_t^\infty e^{-\Delta_\tau^*} d\tau \\ \leq \quad & (u(A) + \chi\nu(\phi)) \lim_{t\to\infty} \frac{\Delta_t e^{-\Delta_t^*}}{\rho - b + d} \\ = \quad & \frac{u(A) + \chi\nu(\phi)}{\rho - b + d} \lim_{t\to\infty} \frac{\Delta_t}{e^{\Delta_t^*}} = \frac{u(A) + \chi\nu(\phi)}{\rho - b + d} \lim_{t\to\infty} \frac{\dot{\Delta}_t}{\dot{\Delta}_t^* e^{\Delta_t^*}} \\ = \quad & \frac{u(A) + \chi\nu(\phi)}{\rho - b + d} \lim_{t\to\infty} \frac{\rho - b + d + \phi i}{\rho - b + d + \phi i^*} \frac{1}{e^{\Delta_t^*}} = 0 \end{aligned}$$

because

$$\frac{\rho - b + d}{\rho - b + d + \phi} \leq \frac{\rho - b + d + \phi i}{\rho - b + d + \phi i^*} \leq \frac{\rho - b + d + \phi}{\rho - b + d} \text{ and } e^{\Delta_t^*} \to \infty \text{ as } t \to \infty.$$

Therefore, for any feasible Δ_t,

$$\lim_{t\to\infty} \lambda_{4,t} \Delta_t = -\lim_{t\to\infty} \Delta_t \int_t^\infty e^{-\Delta_\tau} [u(c_\tau) - \chi\nu(\phi i_\tau)] d\tau = 0. \tag{61}$$

Together with (70) we have

$$\lim_{t\to\infty} \lambda_{4,t} (\Delta_t - \Delta_t^*) = 0.$$

Note that , since $\lim_{t\to\infty} \Delta_t = \infty$ so from (61) we get $\lim_{t\to\infty} \lambda_{4,t} = 0$.

□

Lemma 2. *We have*

$$\begin{aligned} &i) \quad \lim_{t\to\infty} \lambda_1(k^* - k) \leq 0, \\ &ii) \quad \lim_{t\to\infty} \lambda_2(i^* - i) = 0, \\ &iii) \lim_{t\to\infty} \lambda_3(s^* - s) = 0. \end{aligned}$$

Proof. i) From (52) we get $\lambda_1 \geq 0$. Therefore $\lambda_1 k \geq 0$ and (58) implies

$$\lim_{t\to\infty} \lambda_1(k^* - k) \leq 0.$$

ii) From (58) and Proposition 6, we have $0 = \lim_{t\to\infty} \lambda_2 i^* = i^\infty \lim_{t\to\infty} \lambda_2$ therefore

$$\lim_{t\to\infty} \lambda_2 = 0.$$

As i is bounded, we have

$$\lim_{t\to\infty} \lambda_2(i^* - i) = 0.$$

Similarly, we have

$$\lim_{t\to\infty} \lambda_3 = 0 \text{ and hence } \lim_{t\to\infty} \lambda_3(s^* - s) = 0.$$

□

In the SIS model there is only one state variable, i, describing the disease dynamics and we can show the appropriate transversality condition for it directly (see Goenka, Liu and Nguyen (2020, Lemma 2)). In the SIR model there are two variables and we need the additional property of convergence of the disease state variables, s, i, to the steady state in order to derive the limiting properties of the co-state variables.

We will adapt the method developed by Leitmann and Stalford (1971)[26] for a sufficiency condition to our (non-convex) infinite-horizon optimal control problem for the endogenous discounting problem. The Leitmann-Stalford result allows for potential non-convexities but not endogenous discounting. This condition is weaker than standard Arrow-Mangasarian sufficient conditions (see Theorem V, Peterson and Zalkind (1978), page 595).

[26] We re-state the Leitmann and Stalford result. (Leitmann-Stalford, 1971): Consider the problem:

$$\max \int_0^\infty g_0(x(t), z(t))$$

subject to

$$\dot{x}(t) = g(x(t), z(t)), x(0) = x_0, z(t) \in Z$$

Define the Hamiltonian

$$H(x, z, \lambda) = g_0(x(t), z(t)) + < \lambda, g(x(t), z(t)) >$$

Define the augmented Hamiltonian $\bar{H}(\mathbf{x}_t, \mathbf{z}_t, \lambda_t) = H(\mathbf{x}_t, \mathbf{z}_t, \lambda_t) + \langle \dot{\lambda}_t, \mathbf{x}_t \rangle$ and $M(\mathbf{x}_t, \lambda_t) = \max_{\mathbf{z}_t} \bar{H}(\mathbf{x}_t, \mathbf{z}_t, \lambda_t)$ as the augmented maximized Hamiltonian.

We need the following Lemma.

Lemma 3. *We have $\bar{H}(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t) \geq \bar{H}(\mathbf{x}_t^*, \mathbf{z}_t, \lambda_t)$ for all $\mathbf{z}_t$. In other word, given $\mathbf{x}_t^*$ then $\mathbf{z}_t^* = \arg\max \bar{H}(\mathbf{x}_t^*, \mathbf{z}_t, \lambda_t)$ and thus $M(\mathbf{x}_t^*, \lambda_t) = \bar{H}(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t)$.*

Proof. We have

$$\begin{aligned} & \bar{H}(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t) - \bar{H}(\mathbf{x}_t^*, \mathbf{z}_t, \lambda_t) \\ = \; & e^{-\Delta^*}[u(c_t^*) - u(c_t)] - \lambda_1(c_t^* - c_t) - \lambda_1(h_t^* - h_t) + (\lambda_2 - \lambda_3)s^*i^*(\alpha(h^*) - \alpha(h)) \end{aligned}$$

As $u(c)$ is concave we have

$$e^{-\Delta^*}[u(c_t^*) - u(c_t)] \geq e^{-\Delta^*}u'(c_t^*)(c_t^* - c_t) = \lambda_1(c_t^* - c_t). \tag{62}$$

Since $\alpha(h)$ is convex, we have

$$\alpha(h^*) - \alpha(h) \leq \alpha'(h^*)(h^* - h).$$

From (52) and (53), $0 < e^{-\Delta}u'(c) = \lambda_1 = \alpha'(h)si(\lambda_2 - \lambda_3)$ we have $s^*i^*(\lambda_2 - \lambda_3) < 0$. Therefore,

$$s^*i^*(\lambda_2 - \lambda_3)(\alpha(h^*) - \alpha(h)) \geq s^*i^*(\lambda_2 - \lambda_3)\alpha'(h^*)(h^* - h) = \lambda_1(h^* - h). \tag{63}$$

It follows from (62) and (63) that $\bar{H}(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t) - \bar{H}(\mathbf{x}_t^*, \mathbf{z}_t, \lambda_t) \geq 0$.

□

In line with Leitmann and Stalford(1971), we will use the following assumption.

Assumption 6. *Assume that*

$$\bar{H}(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t) \geq \bar{H}(\mathbf{x}_t, \mathbf{z}_t, \lambda_t) \tag{64}$$

where $< \lambda, g(x(t), z(t)) >$ is the inner product in $\mathbb{R}^n$. Let $z^* \in Z$, let $x^* = x(z^*)$ be the corresponding trajectory, and let $\lambda : [0, \infty) \to R^n$ be absolutely continuous. Let following conditions be fulfilled for every $z \in Z$ and $x = x(z)$

$$i) \int_0^\infty e^{-\rho t}[H(x^*(t), z^*(t), \lambda(t)) - H(x(t), z(t), \lambda(t)) + < \dot{\lambda}, x^*(t) - x(t) >]dt \geq 0,$$

and

$$ii) \lim_{t \to \infty} \langle \lambda, x^*(t) - x(t) \rangle \leq 0.$$

Then (x^*, z^*) is an optimal solution.

Remark 2. Assumption 6 is weaker than assumption on the concavity of maximized Hamiltonian $M(\mathbf{x}_t, \lambda_t)$ in $\mathbf{x}_t$ as in Arrow's sufficiency condition. Indeed, assuming $M(\mathbf{x}_t, \lambda_t)$ is concave in $\mathbf{x}_t$: Since $M(\mathbf{x}_t, \lambda_t) \geq \bar{H}(\mathbf{x}_t, \mathbf{z}_t, \lambda_t)$ and by Lemma 3 $M(\mathbf{x}_t^*, \lambda_t) = \bar{H}(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t)$ and

$$\begin{aligned} \bar{H}_{x_{j,t}}(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t) &= H_{x_{j,t}}(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t) + \dot{\lambda}_{j,t} \\ &= -\dot{\lambda}_{j,t} + \dot{\lambda}_{j,t} = 0 \end{aligned}$$

we get

$$\begin{aligned} \bar{H}(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t) - \bar{H}(\mathbf{x}_t, \mathbf{z}_t, \lambda_t) &\geq M(\mathbf{x}_t^*, \lambda_t) - M(\mathbf{x}_t, \lambda_t) \\ &\geq < M_x(\mathbf{x}_t^*, \lambda_t), \mathbf{x}_t^* - \mathbf{x}_t^* > \\ &= < \bar{H}_x(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t), \mathbf{x}_t^* - \mathbf{x}_t > \\ &= 0 \end{aligned}$$

Also, if the Hamiltonian is jointly concave in state and control variables as in the Mangasarian sufficient condition, we easily get (64) by the properties of a concave function and the FOCs (16)-(21)

$$\bar{H}(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t) - \bar{H}(\mathbf{x}_t, \mathbf{z}_t, \lambda_t) \geq < \bar{H}_x(\mathbf{x}_t^*, \mathbf{z}_\mathbf{t}^*, \lambda_t), \mathbf{x}_t^* - \mathbf{x}_t > + < \bar{H}_z(\mathbf{x}_t^*, \mathbf{z}_\mathbf{t}^*, \lambda_t), \mathbf{z}_t^* - \mathbf{z}_t >= 0.$$

However, in our model, the Hamiltonian is not jointly concave if the welfare function is positive, i.e. if $u(c) - \chi\nu(\phi i) > 0$. (see Remark 1 above). □

Remark 3. The SIR model without disease related mortality is a special case and with exogenous discounting Assumption 6 is not needed. □

We are now ready to prove the sufficient condition.

Proposition 11. *Consider the maximization problem (14) and suppose that an interior continuous* $(\mathbf{x}_t^*, \mathbf{z}_t^*)$ *and associated costate variables* λ_t *exist and satisfy (16)-(21). Then under Assumptions 1-6,* $(\mathbf{x}_t^*, \mathbf{z}_t^*)$ *is a locally optimal solution of (P).*

Proof. The results of Lemmas 1- 3 yield

$$\lim_{t\to\infty} \lambda_{1,t}(k_t^* - k_t) + \lim_{t\to\infty} \lambda_{2,t}(i_t^* - i_t) + \lim_{t\to\infty} \lambda_{3,t}(\Delta_t^* - \Delta_t) \leq 0. \tag{65}$$

Assumption A5 implies

$$H(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t) - H(\mathbf{x}_t, \mathbf{z}_t, \lambda_t) + < \lambda_t, \mathbf{x}_t^* - \mathbf{z}_t^* > \geq 0. \tag{66}$$

Taking integral over (66) we get

$$\int_0^\infty \{H(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t) - H(\mathbf{x}_t, \mathbf{z}_t, \lambda_t)] + \langle \dot{\lambda}_t, \mathbf{x}_t^* - \mathbf{x}_t \rangle\} dt \geq 0$$

$$\Leftrightarrow \int_0^\infty e^{-\Delta^*}[u(c^*) - \chi\nu(\phi i^*)]dt - \int_0^\infty e^{-\Delta}[u(c) - \chi\nu(\phi i)]dt + \int_0^\infty \{< \lambda_t, \dot{\mathbf{x}}_t^* - \dot{\mathbf{x}}_t > + \langle \dot{\lambda}_t, \mathbf{x}_t^* - \mathbf{x}_t \rangle\} dt$$

$$\Leftrightarrow \int_0^\infty e^{-\Delta^*}[u(c^*) - \chi\nu(\phi i^*)]dt - \int_0^\infty e^{-\Delta}[u(c) - \chi\nu(\phi i)]dt \geq -\lim_{t\to\infty} \langle \lambda_t, \mathbf{x}_t^* - \mathbf{x}_t \rangle. \tag{67}$$

Therefore, it follows from (65) that

$$\int_0^\infty e^{-\Delta^*}[u(c^*) - \chi\nu(\phi i^*)]dt - \int_0^\infty e^{-\Delta}[u(c) - \chi\nu(\phi i)]dt \geq 0$$

and we get the sufficient condition.

□

Corollary 1. *The disease endemic steady state with positive health expenditures is locally optimal.*

As the endemic steady state satisfies the necessary conditions, we have shown that it is indeed optimal.

Remark 4. The proof showing that the endemic steady state with zero health expenditure in Model I, as well as the two steady states in Model II follow similar arguments and are omitted for brevity.

□.

5.1 Discussion

We are using the FOCs and the standard transversality conditions

$$\lim_{t\to\infty} \lambda_{j,t} x_{j,t}^* = 0, j = 1, .., 3. \tag{68}$$

to provide a direct proof for sufficient condition. Note that this condition holds only at the optimal path $x_{j,t}^*$. For any admissible path $x_{j,t}$ it may not be satisfied. Moreover, λ_t is only identified by the FOCs at optimal solutions $(\mathbf{x}_t^*, \mathbf{z}_t^*)$.

The literature also used a transversality condition where along the optimal paths

$$\lim_{t\to\infty} H = 0. \tag{69}$$

The transversality condition (69) is taken from Michel (1982) for a constant discount rate. In general, these two conditions are not equivalent. Six and Wirl (2015) in a pollution model with endogenous discounting model using the convergence of the state variable to a steady state show that if (58) holds then (69) also holds. We now also show a similar result

but for our non-convex model based on the usual transversality condition and additional assumptions on function $\alpha(h)$.

Lemma 4. *If $\alpha'(0) \neq -\infty$ and $\alpha'' > 0$ then the usual transversality condition (58) implies the (69) transversality condition.*

Proof. As $0 \leq h^* \leq A$ and $\alpha'' > 0$, we have $\alpha'(0) \leq \alpha'(h^*) \leq \alpha'(A) \leq 0$. Therefore, $0 \leq |\alpha'(h^*)s^*i^*\lambda_2| \leq |\alpha'(0)s^*i^*\lambda_2|$. Therefore $\lim_{t\to\infty} |\alpha'(h^*)s^*i^*\lambda_2| = 0$. Similarly, $\lim_{t\to\infty} |\alpha'(h^*)s^*i^*\lambda_3| = 0$. Thus from (53) we have $\lim_{t\to\infty} \lambda_1 = \lim_{t\to\infty} \alpha'(h^*)si(\lambda_2 - \lambda_3) = 0$.

We have

$$\begin{aligned}
&\lim_{t\to\infty} H = \\
&\lim_{t\to\infty} e^{-\Delta^*}[u(c^*) - \chi\nu(\phi i^*)] + \lim_{t\to\infty} \lambda_1\{f(k^*, 1-i^*) - c^* - h^* - \delta k^* - (b - d - \phi i^*)k^*\} \\
&+ \lim_{t\to\infty} \lambda_2 i^* E + \lambda_3 F + \lim_{t\to\infty} \lambda_4\{\rho - b + d + \phi i^*\}
\end{aligned}$$

where

$$\begin{aligned}
E &= [\phi - \alpha(h^*)(1 - \delta_1\theta^*)^2]i^* - b - \gamma - \phi, \\
F &= b - bs - \alpha(h^*)s^*i^* + \phi s^*i^*.
\end{aligned}$$

It is easy to see that

$$\begin{aligned}
0 &\leq |E| = \left|[\phi - \alpha(h^*)(1 - \delta_1\theta)^2]i^* - b - \gamma - \phi\right| \leq \alpha(h^*) + b + \gamma + 2\phi < \infty, \\
0 &\leq |F| = |b - bs - \alpha(h^*)s^*i^* + \phi s^*i^*| \leq 2b + \alpha(A) + \phi < \infty.
\end{aligned}$$

Moreover, using the results of Lemma 1 and Lemma 2 ($\lim_{t\to\infty} \lambda_1 = \lim_{t\to\infty} \lambda_2 = \lim_{t\to\infty} \lambda_3 = \lim_{t\to\infty} \lambda_4 = \lim_{t\to\infty} e^{-\Delta^*} = 0$) with the fact that $k^*, c^*, h^*, i^*, u(c^*), \nu(\phi i^*)$ and f are bounded, it implies that the transversality condition (69) is satisfied. □

Note we only assume that $\alpha'(0) < 0$ for existence of steady state (Assumption 5). The assumption that $\alpha'(0) \neq -\infty$ is ruling out an Inada type condition on the α function. This is consistent with our results on the existence of a steady state with zero health expenditure. We did not need to impose a condition on the second derivative of α for the earlier results.

Note that the transversality condition (69) can also imply the usual transversality condition (58) but we need more assumptions. For example, Aseev and Kryazhimskiy (2004) show that (69) implies (58) if additional assumptions on the constraint of state variables imposed. (see Corollary 4, page 1111).

For the sufficiency, we assume only (58) holds. However, since our model is non-convex with endogenous discounting, this condition is not enough for a sufficiency as the framework of the earlier results do not hold. Using the special structure of the autonomous problem, we provide a direct proof of sufficiency by proving the transversality condition for state variables for any admissible x_t,

$$\lim_{t\to\infty} \lambda_{j,t}(x^*_{j,t} - x_{j,t}) \leq 0. \tag{70}$$

These kind of tranversality conditions were assumed directly in Cartigny and Michel (2003), Acemoglu (2009) (Theorem 7.11, page 246) for a sufficiency condition but for convex problems and standard discounting. This condition is difficult to check because the admissible path $x_{j,t}$ does not necessarily satisfy the FOCs while the co-state $\lambda_{j,t}$ is only determined at the optimal path $x^*_{j,t}$. We do not get any information for $x_{j,t}$ from two standard transversality conditions (58) and (69). However, if $x_{j,t}$ is bounded, then the condition $\lim_{t\to\infty} \lambda_{j,t} = 0$ implies (70). If $\lambda_{j,t} \geq 0$ and $x_{j,t} \geq 0$ then (58) implies (70). Thus, Acemoglu (2009) (Theorem 7.14) makes this assumption as $\lim_{t\to\infty} \lambda_{j,t}x_{j,t} \geq 0$. In our model, the co-state variable associated with the infective is negative so this inequality is only satisfied as a zero identity which will be proven in our model.

Finally, it is crucial when we check the maximality of the Hamiltonian we can decompose it into two parts: the first just relies on the separability of control and state variables and the concavity in control variables of the objective function, and thus, using standard results the difference between the candidate solution and any other solution is non-negative; and a term that depends on the co-state and the state variables as given above. Recall, the non-concavity in the problem arises from the law of evolution of state variables and the Hamiltonian is also non-concave. As indicated, we show this term converges to a negative value, and we are able to obtain sufficiency of the first order conditions.

6 Conclusion

Due to the ongoing Covid-19 pandemic there is an increasing interest in economic epidemiology which study the interaction of infectious diseases using compartmental disease modeling and economic outcomes. Many papers use the Kermack-McKendrick model with mass action to model the epidemiology. However, this model due to the linear effect on infections of population size may not be the most suitable to look at medium to long run effects as population size change due to birth and deaths - both due to the disease and other causes. The standard incidence model does not have this effect and is the model of choice by epidemiologists. We investigated two models: where there are early deaths so that deaths of infectives which shortens the duration of the epidemics and late deaths when the individuals who succumb to the disease are not circulating in the population - either because they are hospitalized or there is pre-mature mortality of the recovered population. It seems that in Covid-19 an increasing part of the mortality is of the later type. These are early days of the pandemic and long run effects of the disease on pre-mature mortality are not fully understood. We studied the optimal response which can be either interpreted as preventive health expenditures or self-isolation. In a fully general equilibrium neoclassical growth model, the optimal response and equilibrium outcomes are sensitive to the modeling choice as they have very different implications for disease evolution. This generates different equilibrium effects even when mortality is the same in both models. Thus, economists should pay close attention to what choices are made for modeling infectious diseases.

In economic epidemiology models, the interest is in optimal choices. The models with

SIR dynamics are non-convex and if there is disease related mortality, discounting is also endogenous. Thus, the usual conditions for sufficiency of first order conditions do not apply. We present the first results on sufficiency for this model. The SIR model without disease related mortality is a special case. The results differ from the SIS model as there are now two state variables, s, i to describe disease dynamics rather than just one. As a result the conditions rely on convergence of the disease variables to a steady state which we establish.

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