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Modeling optimal quarantines under infectious disease related mortality *

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Abstract

This paper studies optimal quarantines (can also be interpreted as lockdowns or self-isolation) when there is an infectious disease with SIS dynamics and infections can cause disease related mortality in a dynamic general equilibrium neoclassical growth framework. We characterize the optimal decision and the steady states and how these change with changes in effectiveness of quarantine, productivity of working from home, contact rate of disease and rate of mortality from the disease. A standard utilitarian welfare function gives the counter-intuitive result that increasing mortality reduces quarantines but increases mortality and welfare while economic outcomes and infections are largely unaffected. With an extended welfare function incorporating welfare loss due to disease related mortality (or infections generally) however, quarantines increase, and the decreasing infections reduce mortality and increase economic outcomes. Thus, there is no optimal trade-off between health and economic outcomes. We also study sufficiency conditions and provide the first results in economic models with SIS dynamics with 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, SIS model, mortality, sufficiency conditions, economic growth, lockdown, quarantine, self-isolation.

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

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1 Introduction

The Covid-19 pandemic has brought the study of interaction of infectious diseases, hence, epidemiology modeling and economic outcomes to the forefront of economic research. As for Covid-19 there are as yet no medical interventions to prevent and treat the disease, there is also an interest in the role of non-pharmaceutical interventions (NPI) to control the disease. The interaction of epidemiology modeling and economic outcomes predates the Covid-19 outbreak (see Bonds, et al. (2004), Goenka and Liu (2013, 2020), Goenka, Liu and Nguyen (2020), and Toxvaerd (2019)). However, these first generation of models concentrated on situations where there is no disease related mortality which in times of Covid-19 has become especially central to the problem.[1]

This paper studies optimal lockdown, i.e. where both the healthy (susceptible) and the infected (infectious) can be quarantineed. We model this in a neoclassical growth framework where the disease evolves according to SIS dynamics. This is motivated by the fact it is not well understood for how long is disease related immunity conferred for coronaviruses such as Covid-19. The evidence is preliminary and there is emerging evidence that subsequent immunity may not be long lasting.[2] As we are concerned about the medium to longer run in this paper we abstract from the temporary immunity phase (i.e. the state R).[3] Households can save through investing in capital and production of the single consumption good uses capital and labor. Only the healthy (susceptible) individuals can work. In this paper, motivated by Covid-19 we abstract from health expenditures that can be used for prevention and treatment[4] and the only way to control the disease is by quarantines. Quarantines are imperfect as a mechanism to control diseases as effectiveness or compliance of these varies. The productivity of those quarantined is reduced, and the labor supply is the fraction of the healthy not quarantined plus the reduced productivity of the healthy quarantined.[5] There is disease related mortality so a fraction of the infected die. The way the optimal quarantine decision is modeled, it can also be interpreted as the optimal decision to self-isolate. The distinction between self-isolation and a quarantine is that in the latter it is mandated rather than an individual decision. In all our model the households are homogeneous and we do not model disease related externality where households do not take into account the

[1]Chakraborty, et al. (2010) modeled disease related mortality in an overlapping generations framework but did not use a compartment epidemiology model.

[2]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 a modeling stategy Kissler, et al. (2020) use an SIRS model for medium run projections.

[3]This is also consisted with many of the other infectious diseases that are the main sources of disease related mortality, in particular malaria, tuberculosis, dengue, and influenza also do not have disease related immunity. While an individual may have immunity to a particular strain of influenza for a short period, the virus mutates and there is no lasting immunity. HIV/AIDS is a disease of SI class and its epidemiology is not captured by either SIS or SIR models.

[4]Goenka and Liu (2020) and Goenka, Liu and Nguyen (2020) modeled optimal health expenditures in a similar growth framework.

[5]In our model the productivity of all healthy who are quarantined drops but as there is only partial compliance, only a fraction of those quarantined are not relevant for disease dynamics.

effect of their decisions on the evolution of the disease in the population.[6] The preliminary evidence also suggests that the macroeconomic consequences of the two are similar.[7] For self-isolation, infections can be higher in heterogeneous populations for obvious reasons. Thus, the quarantine or lockdown can also be interpreted as optimally chosen self-isolation. There is an emerging literature on quarantines in economic epidemiology models but these these papers generally look at the very short-run and do not model capital accumulation.[8]

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 quarantine depends on a function of the parameters and the equilibrium values of the economic variables. The equilibrium reproduction rate, R_0^* will depend on both the infectivity of the disease and endogenous economic choices. As the degree of compliance, the drop in productivity from working at home, and contact and mortality rates of the disease are treated as parameters we vary them to see how the equilibrium economic and health outcomes vary with them. As compliance with quarantine increases, the optimal quarantine first increases and then decreases reflecting the fact that the impact of the product of degree of quarantine and compliance. Thus, increase in compliance eventually can be traded-off with a reduced quarantine. With increased productivity from working from home, there is a reduced trade-off between health and wealth, and the optimal quarantine increases. The effect of increasing the contact rate on the optimal quarantine is also what we would expect. However, in a pure utilitarian model where the welfare depends only on consumption, as mortality is increased there are counter-intuitive results: not only does the level of quarantine decrease but welfare and the economic outcomes increase. This is similar to the result in Young (2005) but in our model is driven by the fact that increased mortality results in fewer infections.[9]

This raises one of the two substantive methodological issues with incorporating the disease related mortality. What should be the welfare function? Thus, we extend the utilitarian welfare function by including a loss in welfare from infections and mortality (the latter is a fixed fraction of infections). This has also been done in other papers (Acemoglu, et al. (2020), Alvarez, et al. (2020), Jones, et al. (2020) for a partial list). We characterize the optimal quarantine with the extended welfare function and derive the steady states. As we would expect increasing the weight on welfare loss from infections increases the severity of quarantine. When we evaluate varying the parameters, the effects of varying compliance, home productivity, and contact rates are qualitatively similar with higher quarantines and better economic and health outcomes. However, the effect on increasing mortality is strikingly different, with higher mortality leading to more stringent lockdowns. The welfare initially decreases but eventually increases as stringent quarantines bring down infections. While in the utilitarian model economic variables and infections are not affected significantly

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

[7]See Andersen, et al. (2020) and Danske Bank (2020) for comparisons across Scandinavian countries.

[8]See for example Acemoglu, et al. (2020), Alvarez, et al. (2020), Eichenbaum, et al. (2020), Giannitsarou, et al. (2020), and Jones, et al. (2020).

[9]Young (2005) uses a Solow model so savings and investment do not adjust and the primary mechanism is the increase in per-capita capital stock.

from higher mortality, in the extended welfare function, there are decreased infections (due to more stringent quarantines) and better economic outcomes due to the decreased infections. Thus, in equilibrium, there is no trade-off between "health-wealth" trade-off.

The second methodological issue that has not received attention is the implications for evaluating sufficiency of first order conditions in optimal control problems. It is already known in the literature that epidemiology dynamics are not convex and the first order conditions to control problems need not be sufficient. This was first pointed out in the economics literature by Gersovitz and Hammer (2004) and sufficiency for SIS models without disease related mortality were provided by Goenka, Liu and Nguyen (2014). However, with disease related mortality, as population size changes with the level of infection, effectively the discount rate becomes endogenous. To our knowledge, while such models are being in the emerging literature, there are no established transversality and sufficiency conditions with endogenous discounting in a non-convex model. In this paper, we prove the transversality and sufficiency conditions for the economic SIS model with disease related mortality (Section 6). Following, Obstfeld (1990), as discounting is endogenous, we introduce another state variable for the rate of discount. 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. Thus, these are the first results for sufficiency with endogenous discounting for non-convex problems.

The plan of the paper is as follows: Section 2 introduces the economic epidemiology model, Section 3 studies the equilibrium steady states of the standard utilitarian model, Section 4 does comparative statics of equilibrium steady state outcomes, Section 5 studies the extended welfare model, Section 6 studies the transversality and sufficiency conditions, and Section 7 concludes.

2 The Economic Epidemiology Model

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

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 by the diseases. We assume that diseases follow the SIS dynamics (see the discussion in the Introduction).

We model the infectious disease as having two effects - reducing productivity of the infected and disease related mortality. We make the simplifying assumption that an infected

individual is incapacitated by the disease or that the productivity falls to zero. [10] We assume the labor is supplied inelastically.[11] If i is the fraction of household infected, the proportion ϕ of these succumb to the disease.

The representative household's preferences are given as:

$$\int_0^\infty e^{-\rho t} u(C) N_t dt = \int_0^\infty e^{-(\rho - b + d + \phi i)t} N_0 u(C) dt, \tag{1}$$

where ρ is the discount factor with $\rho > b - d$, and the initial size of household is assumed to be one.

Assumption 1. *The felicity function u, $u : \mathbb{R}_+ \to \mathbb{R}$ is $\mathcal{C}^2$ with $u' > 0$ and $u'' < 0$. The discount rate, $\rho > 0$.*

Physical capital accumulations follow the standard law of motion with the deprecation rate $\delta \in (0, 1)$.

In this paper we concentrate on the control of the disease through the imposition of a lockdown. This is motivated by Covid-19 and other coronaviruses including SARS and MERS for which there were are no vaccinations or proven prophylactic medicines or proven treatments for recovery at the time of writing the paper. All methods of control are non-pharmaceutical interventions (NPIs). The earlier paper Goenka and Liu (2013) studied imperfect vaccination and isolation to control the disease. In that paper the costs were not modeled and the interventions were ad-hoc to stabilize the disease rather than optimal choices. The paper Goenka, Liu and Nguyen (2014) modeled optimal reduction of infectivity and recovery from the disease through health expenditures. Goenka and Liu (2020) concentrated on reduction of infectivity from health expenditures[12] and distinguished between the decentralized case where households do not take into account the affect of their decisions on disease transmission, i.e. the disease externality, and the optimal public health policy.As this analysis is motivated by lockdowns as a method of disease control imposed by governments (or optimal self-imposed self-isolation) when there are no medical interventions we concentrate on the socially optimal solution abstracting away from these issues which have already been studied in our earlier work.

The way we model lockdown is that a fraction, θ, with $0 \leq \theta \leq 1$ of both susceptibles and infected population is quarantined. Thus, there is no effective track-and-trace-and-isolate

[10] How much productivity is affected varies across diseases. The recent comprehensive estimates of disability weights used to compute DALYs is one possible measure of affect on productivity (see Salomon, et al. (2012), Murray, et al. (2012)). For some specific diseases there are estimates in the economic literature on loss of income from which effect on productivity is imputed (e.g. Weisbrod, et al. (1974) study effect of five parasitic diseases on banana plantation workers in St. Lucia; Fox, et al. (2004) study loss of income to tea pickers infected with HIV/AIDS in Kenya). For Covid-19 many of the infected are asymptomatic and to the extent they are not isolated, their productivity is not affected. For symptomatic cases, even for "mild" cases that do not require hospitalization, the effect of the disease is debilitating and can have long lasting tail effects.

[11] 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.

[12] The paper also considered the effect of education on affecting disease transmission. The channel is that education increases greater awareness and understanding of health risks.

(TTI) program that will isolate the infective (and those exposed to the infection).[13] The experience of quarantines shows that even with these in place, infections may or may not come down. While infections did come down in Italy and Spain under the quarantine, in UK they continued to remain significant. Thus, we model the effectiveness of the quarantine or compliance with the lockdown to reduce infections by the parameter δ_1, with $0 \leq \delta_1 \leq 1$. When $\delta_1 = 0$ the lockdown is not effective and with $\delta_1 = 1$ it is fully effective. In the paper we concentrate on partial compliance, $0 < \delta_1 < 1$. Effectiveness of the quarantine depends on both the sanctions for violating it and on compliance with it. The determinants of compliance with a lockdown are many with complex interactions between them.[14] In this paper we treat this as a parameter.

During a quarantine, susceptible individuals may continue to work from home. However, their productivity from working from home is likely to be affected. Some individuals are in occupations where they cannot work from home. The emerging evidence is that there is considerable heterogeneity on what occupations and who can work from home without loss of productivity. [15] Thus, we model the productivity of working from home by a parameter δ_2, with $0 \leq \delta_1 \leq 1$. When $\delta_2 = 0$ the productivity of working from home is zero and with $\delta_2 = 1$ it is as productive as absent a lockdown. There is an emerging literature on who can and who cannot work from home we treat this as a parameter.

Production: The production side of the model is a standard neo-classical growth model where households can invest in capital which is productive next period and depreciates at rate δ.[16] Households own representative firms that use capital and labor as inputs.

Assumption 2. *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,*

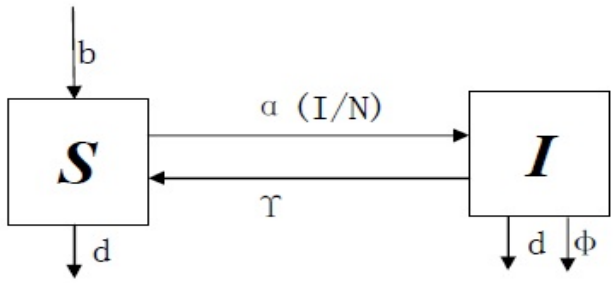
[13]There is diversity across countries on the effectiveness of TTI programs. Many countries do have test-and-track programs for Covid (e.g. Singapore, Korea, Germany, China) and many of the countries that have the largest number of infections do not have fully effective ones (e.g. US, UK, India, Brazil, Sweden, Russia, South Africa). Even with test-and-tracking, whether the infected and potentially infected can be isolated varies considerably and depends on personal compliance.

[14]The emerging literature on the determinants of compliance shows that some of the factors are trust of policy makers (Bargain and Aminjonov (2020), Vinck, et al. (2019)), civic engagement (Barrios, et al. (2020)), age (Belot, et al. (2020)), social capital (Borgonovi, Andrieu and Subramaniam (2020), Deopa and Fortunato (2020), Mazzona (2020)), political views (Brodeur, Grigoryeva, and Kattan(2020)), broader socio-economic determinanst including gender, political partisanship and risk tolerance (Fan, Orhun and Turjeman (2020), Papageorge, et al. (2020)).

[15]See Adams-Prassl, et al. (2020), Alipour,Falck and SchÃ$\frac{1}{4}$ller (2020), Bartik, et al. (2020), Dingell and Neiman (2020), Gottlieb, et al. (2020), Hensvik, Le Barbanchon and Rathelot (2020), Kalenkoski and Wulff Pabilonia (2020), Saltiel (2020)) for some examples of this emerging literature. The effect on productivity is affected by occupation and industry, number and age of children, care responsibilities, gender issues, access to technology, amongst other things.

[16]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.

Figure 1. The Transfer Diagram For the SIS Epidemiology Model with disease related mortality



Note: In a SIS epidemiology model, the total population is divided into three groups: the susceptible denoted as S and the infected denoted as I. The birth rate is b and newborns are born healthy and susceptible. All individuals irrespective of health status die at the rate d. The susceptible get infected at the rate $\alpha\frac{I}{N}$, the infected recover at the rate γ and might die at the rate ϕ as a result of being infected.

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 Epidemiology Model

For the epidemiology model we use a SIS model with standard incidence but with disease related mortality. An individual can be in one of two health states, S, where the individual is healthy and susceptible to the disease, or I where the individual is infected and infectious enough to transmit the disease.

Assumption 3. *The epidemiology model is given by the following system of differential equations :*

$$\begin{aligned}
\dot{S} &= bN - \frac{\alpha S(1-\delta_1\theta)I(1-\delta_1\theta)}{N} - dS + \gamma I \\
\dot{I} &= \frac{\alpha S(1-\delta_1\theta)I(1-\delta_1\theta)}{N} - \gamma I - dI - \phi I \\
\dot{N} &= (b-d)N - \phi I
\end{aligned}$$

The parameters in the model are b the birth rate, i.e. new flow of susceptibles, d the death or death or exit rate of infected which is not related to the infectious disease, α is the contact rate of adequate contacts that can transmit the disease, γ is the recovery rate from the disease, and ϕ is the mortality from infections due to the disease. We use the standard or density dependence model where the transmission of the disease depends on the fraction of infected rather than number of infected. In the second model, there are scale effects which

are seen in herd models but are thought not to characterize human interactions where the pattern of interactions is relatively invariant to population size. [17] In this paper, motivated by diseases such as Covid-19 for which there is no proven therapies or prophylactic treatment, we treat these as parameters.[18]

Since $N = S + I$, we define $s = S/N$ and $i = I/N$. We have $s + i = 1$ and $\dot{s} + \dot{i} = 0$. Therefore, the SIS epidemiological model can be reduced to:

$$\begin{aligned} \dot{i} &= \frac{\dot{I}}{N} - \frac{I}{N}\frac{\dot{N}}{N} \\ &= \alpha(1-\delta_1\theta)^2(1-i)i - bi - \gamma i - \phi i + \phi i^2, \end{aligned}$$

where the total population grows at the rate $b - d - \phi i$. Note that the population growth rate here is endogenous and affected by the prevalence of infectious diseases.

For the SIS epidemiological model, there are two steady states. One is the disease free steady state with $i^* = 0$ and diseases are fully eradicated. The disease free steady state always exists and it could be stable or not stable depending on parameters. The other steady state is disease endemic steady sate with $i^* = 1 - \frac{b+\gamma}{\alpha(1-\delta_1\theta)^2-\phi}$. The prevalence of diseases decreases when birth rate (b) or recovery rate (γ) increases, or when contact rate (α) decreases. When disease related death rate (ϕ) is higher, the fraction of the infected is smaller as these individuals cannot infect others. When there is wider lockdown (θ) or the efficacy of lockdown (δ_1) is higher, the fraction of the infected is also smaller. Note that disease endemic steady state exists if and only if $0 < \frac{b+\gamma}{\alpha(1-\delta_1\theta)^2-\phi} < 1$. This steady if it exists is stable and in that situation the disease free steady state is unstable. For details see Appendix 1.

We study simplest model where the only way to control infection, and hence, disease related mortality is through lock down θ in order to focus on the issues introduced by disease related mortality.

The total labor force is

$$L = (1 - \theta + \delta_2\theta)S$$

Individuals who are not infected and not quarantined can participate in the labor market with productivity equal to 1, that is, $(1 - \theta)S$. Moreover, people who are healthy but quarantined can work at home with productivity δ_2 ($0 \leq \delta_2 \leq 1$), that is, $\delta_2\theta S$). δ_2 captures the productivity of working at home. When $\delta_2 = 0$, people can not work at home. In this case, full lockdown ($\theta = 1$) is never desirable as the total output would be zero. When $\delta_2 = 1$, working at home does not reduce productivity at all, and full lockdown is always the best choice and the economy will be in a disease free steady state. In the paper, we focus on the case where $0 < \delta_2 < 1$.

The labor force is given as:

$$l = L/N = (1 - \theta + \delta_2\theta)(1 - i)$$

[17] For a further discussion of the SIS model see Goenka and Liu (2020).

[18] In earlier papers, Goenka, Liu and Nguyen (2014) and Goenka and Liu (2020) these were endogenized.

3 Standard utilitarian welfare model

The objective is to maximize the total welfare which is the discounted sum of utility only from consumption multiplied by the size of the population. This is the standard utilitarian welfare function used in economics. It is assuming that each household is weighted equally and there is perfect insurance within each household. Multiplying the household's utility by the population size is standard in the literature and will capture the effect of variation in the population size due to disease related mortality. The maximization problem is:

$$\begin{aligned} \max_{c,\theta} \quad & \int_0^\infty e^{-\rho t} u(c_t) N_t dt \\ = \quad & \int_0^\infty e^{-\int_0^t (\rho - b + d + \phi i(\tau)) d\tau} u(c_t) N_0 dt \end{aligned}$$

As the population size is varying, the discount factor becomes endogenous and varies with infections in the population. To solve this maximization problem with an endogenous discount factor, we define the following variable which is the effective discount rate (see Obstfeld (1990)),

$$\Delta(t) = \int_0^t (\rho - b + d + \phi i(\tau)) d\tau,$$

where

$$\dot{\Delta} = \rho - b + d + \phi i_t.$$

Note that with changes in infections, i, disease related mortality, ϕi changes the effective discount rate. Note that Δ is affected by a state variable. None of the existing results for sufficiency of the first order conditions to the optimal control will apply as discounting is endogenous and the problem is non-convex. The social planner problem becomes (we suppress the time subscript)

$$\max_{c,\theta} \int_0^\infty e^{-\Delta} u(c) N_0 dt$$

subject to

$$\dot{k} = f(k,l) - c - \delta k - (b - d - \phi i)k \tag{2}$$
$$\dot{i} = \alpha(1 - \delta_1 \theta)^2 (1 - i) i - bi - \gamma i - \phi i + \phi i^2 \tag{3}$$
$$\dot{\Delta} = \rho - b + d + \phi i \tag{4}$$
$$l = (1 - \theta + \delta_2 \theta)(1 - i) \tag{5}$$
$$0 \leq \theta \leq 1, \quad \text{and} \quad i \geq 0 \tag{6}$$

where the are parameters: $\rho, b, d, \delta, \alpha, \gamma, \phi, \delta_1, \delta_2$; the control variables: c, θ; and the state variables: k, i, l, Δ.

3.1 Characterization of Steady States

As the effective discount rate varies with the rate of infections (which are not monotonic) there can be a time-consistency problem. To avoid this we work the present value Hamiltonian with this additional state variable (see Obstfeld (1990)) which is:

$$\begin{aligned}\mathcal{L} = \quad & e^{-\Delta}u(c) + \lambda_1\{f(k,(1-\theta+\delta_2\theta)(1-i)) - c - \delta k - (b-d-\phi i)k\} + \\ & +\lambda_2\{\alpha(1-\delta_1\theta)^2(1-i)i - bi - \gamma i - \phi i + \phi i^2\} + \\ & +\lambda_3\{\rho - b + d + \phi i\} + \mu_1\theta + \mu_2(1-\theta) + \mu_3 i,\end{aligned}$$

where $\lambda_1 - \lambda_3$ are costate variables and $\mu_1 - \mu_3$ are Lagrange multipliers.

The necessary and sufficient first order conditions (see Section 6 the results on sufficiency) are:

$$\begin{aligned}
c: \quad & e^{-\Delta}u'(c) = \lambda_1 && (7)\\
\theta: \quad & -\lambda_1 f_2(k,l)(1-\delta_2)(1-i) - \lambda_2 2(1-\delta_1\theta)\delta_1\alpha(1-i)i + \mu_1 - \mu_2 = 0 && (8)\\
k: \quad & -\dot{\lambda}_1 = \lambda_1[f_1(k,l) - \delta - b + d + \phi i] && (9)\\
i: \quad & -\dot{\lambda}_2 = -\lambda_1 f_2(k,l)(1-\theta+\delta_2\theta) + \lambda_1\phi k + \lambda_2\alpha(1-\delta_1\theta)^2(1-2i) + \\
& +\lambda_2[-b-\gamma-\phi+2\phi i] + \lambda_3\phi + \mu_3 && (10)\\
\Delta: \quad & \dot{\lambda}_3 = e^{-\Delta}u(c) && (11)\\
& \mu_1 \geq 0, \quad \theta \geq 0, \quad \mu_1\theta = 0 && (12)\\
& \mu_2 \geq 0, \quad 1-\theta \geq 0, \quad \mu_2(1-\theta) = 0 && (13)\\
& \mu_3 \geq 0, \quad i \geq 0, \quad \mu_3 i = 0 && (14)
\end{aligned}$$

Proposition 1. *There always exists a unique disease free steady state with $i^* = 0$, $\theta^* = 0$, $l^* = 1$ and k^* and c^* are determined by*

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

Proof. From $\dot{i} = 0$, we have one disease free steady state $i^* = 0$ and thus $\mu_3 > 0$. From equation (8), we have

$$\mu_1 - \mu_2 = \lambda_1 f_2(k,l)(1-\delta_2).$$

If $\delta_2 < 1$, $\mu_1 > \mu_2 \geq 0$. Therefore, μ_1 is strictly positive and implies $\theta^* = 0$. Then, from

equation (5), $l^* = 1$. From equation (9), we have

$$\frac{\dot{\lambda}_1}{\lambda_1} = -[f_1(k,1) - \delta - b + d].$$

Moreover, from equation (7), we have

$$\frac{\dot{\lambda}_1}{\lambda_1} = -(\rho - b + d) + \frac{u''(c)}{u'(c)}\dot{c}.$$

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

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

from which we can solve for k^*. c^* is derived from equation (2) with $\dot{k} = 0$ in the steady state. □

Proposition 2. *Define the function:*

$$G(\theta) = -\tilde{\lambda}_2 2(1-\delta_1\theta)\delta_1\alpha(1-i)i - \tilde{\lambda}_1 f_2(k,l)(1-\delta_2)(1-i), \tag{15}$$

where

$$i = 1 - \frac{b+\gamma}{\alpha(1-\delta_1\theta)^2 - \phi} \tag{16}$$

$$l = (1-\theta+\delta_2\theta)\frac{b+\gamma}{\alpha(1-\delta_1\theta)^2 - \phi} \tag{17}$$

$$f_1(k,l) = \rho + \delta \tag{18}$$

$$c = f(k,l) - \delta k - (b - d - \phi i)k \tag{19}$$

$$\tilde{\lambda}_1 = \lambda_1/e^{-\Delta} = u'(c) \tag{20}$$

$$\tilde{\lambda}_2 = \lambda_2/e^{-\Delta} = \frac{u'(c)[-f_2(k,l)(1-\theta+\delta_2\theta) + \phi k] + u(c)\phi/g}{-g + b + \gamma + \phi - 2\phi i - \alpha(1-\delta_1\theta)^2(1-2i)} \tag{21}$$

$$g = -(\rho - b + d + \phi i). \tag{22}$$

There are three scenarios:

- *If $G(\theta)|_{\theta=0} < 0$, then $\theta^* = 0$;*
- *If $G(\theta)|_{\theta=1} > 0$, then $\theta^* = 1$;*
- *Otherwise, θ^* is determined by $G(\theta^*) = 0$.*

Given the optimal θ^*, an endemic steady state exists if $0 < \frac{b+\gamma}{\alpha(1-\delta_1\theta^*)^2-\phi} < 1$.

Proof. From $\dot{i} = 0$, we have one endemic steady state with $i^* = 1 - \frac{b+\gamma}{\alpha(1-\delta_1\theta)^2-\phi}$. The steady state exists only if $0 < \frac{b+\gamma}{\alpha(1-\delta_1\theta)^2-\phi} < 1$. Then, we have $\mu_3 = 0$. From equation (5), $l^* = (1-\theta+\delta_2\theta)\frac{b+\gamma}{\alpha(1-\delta_1\theta)^2-\phi}$. From equation (9), we have

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

Moreover, from equation (7), we have

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

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

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

from which we can solve for k^*. c^* is derived from equation (2) with $\dot{k} = 0$ in the steady state.

Next, we need to solve for λ_1 and λ_2. From equation (10), we have

$$-\frac{\dot{\lambda_2}}{\lambda_2} = \frac{\lambda_1}{\lambda_2}f_2(k,l)(1-\theta+\delta_2\theta) + \frac{\lambda_1}{\lambda_2}\phi k + \alpha(1-\delta_1\theta)^2(1-2i) + (-b-\gamma-\phi+2\phi i) + \frac{\lambda_3}{\lambda_2}\phi.$$

Thus, all co-state variables λ_1, λ_2 and λ_3 grow at the same rate:

$$g = \frac{\dot{\lambda_1}}{\lambda_1} = \frac{\dot{\lambda_2}}{\lambda_2} = \frac{\dot{\lambda_3}}{\lambda_3} = -(\rho - b + d + \phi i)$$

Since $g = \frac{\dot{\lambda_3}}{\lambda_3} = \frac{e^{-\Delta}u(c)}{\lambda_3}$, we have

$$\lambda_3 = \frac{e^{-\Delta}u(c)}{g}.$$

Substitute all these into equation (10), we can solve for λ_2:

$$\lambda_2 = e^{-\Delta}\frac{u'(c)[-f_2(k,l)(1-\theta+\delta_2\theta)+\phi k] + u(c)\phi/g}{-g+b+\gamma+\phi-2\phi i-\alpha(1-\delta_1\theta)^2(1-2i)}.$$

From equation (8), we have

$$\begin{aligned}\mu_2 - \mu_1 &= -\lambda_2 2(1-\delta_1\theta)\delta_1\alpha(1-i)i - \lambda_1 f_2(k,l)(1-\delta_2)(1-i) \\ &= e^{-\Delta}\, G(\theta),\end{aligned}$$

where

$$\begin{aligned}G(\theta) &= -\tilde{\lambda}_2 2(1-\delta_1\theta)\delta_1\alpha(1-i)i - \tilde{\lambda}_1 f_2(k,l)(1-\delta_2)(1-i) \\ &= -\frac{u'(c)[-f_2(k,l)(1-\theta+\delta_2\theta)+\phi k]+u(c)\phi/g}{-g+b+\gamma+\phi-2\phi i-\alpha(1-\delta_1\theta)^2(1-2i)}\,[2(1-\delta_1\theta)\delta_1\alpha(1-i)i] \\ &\quad -u'(c)f_2(k,l)(1-\delta_2)(1-i)\end{aligned}$$

Moreover, equations (12) and (13) imply:

1) If $G(\theta) < 0$ when $\theta = 0$, that is, marginal benefit of lockdown is smaller than marginal cost, the endemic steady state is the one with no lockdown ($\theta^* = 0$).

2) If $G(\theta) > 0$ when $\theta = 1$, that is, marginal benefit of lockdown is larger than marginal cost, the endemic steady state is the one with full lockdown ($\theta^* = 1$);

3) Otherwise, the endemic steady state is the one with partial lockdown ($0 < \theta^* < 1$), where θ^* is determined by solving $G(\theta^*) = 0$. □

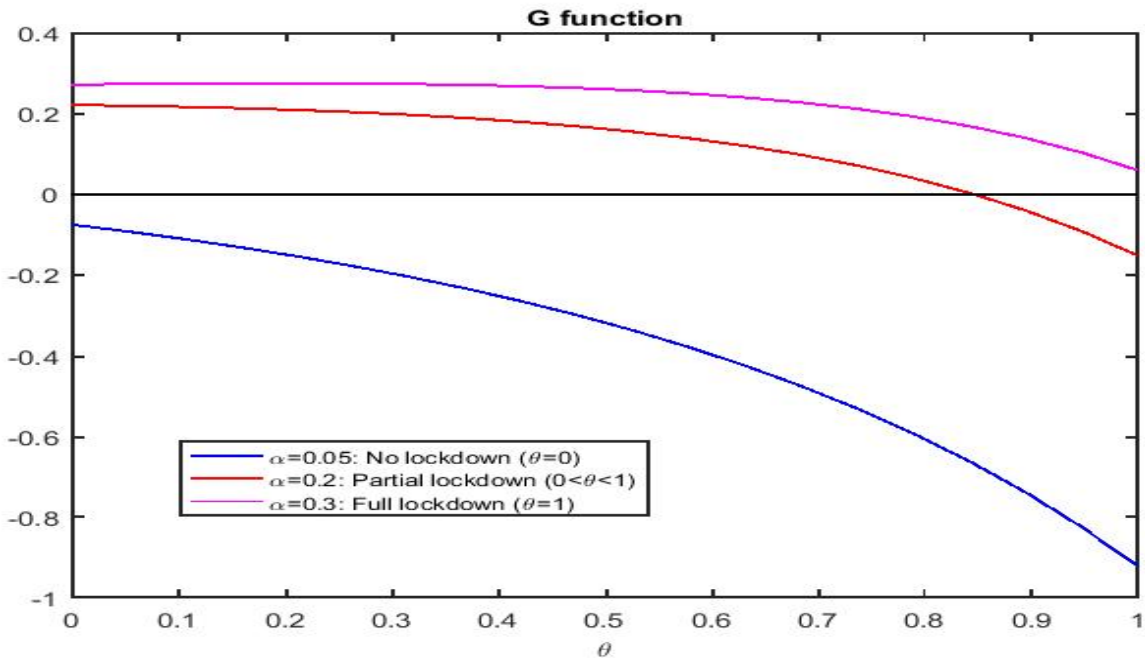
Whether an endemic steady state exists on what the optimal choice of quarantines, θ^*, is in equilibrium. The proposition gives a stronger sufficient condition that does not depend on the choice of θ.

4 Calibration and Simulation

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 examine the impact of varying parameters, i.e. changing efficacy of lockdown measure (compliance), productivity of working at home and disease related mortality. The analysis here focuses on the 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 model shows that the economy is closely related to the prevalence of infectious diseases. This in turn depends on all the fundamental economic, demographic and epidemiological parameters in the model. The following parameters are chosen in line with the literature: discount rate $\rho = 0.055$, capital share $\beta = 0.36$, depreciation rate $\delta = 0.05$, and the scale parameter in the production function A is normalized to 1. The utility function is of CES form $U(c) = \frac{c^{1-\sigma}}{1-\sigma}$ and we choose $\sigma = 1$, that is, the utility function is log utility. Using the statistics from the World Health Organization (See WHO (2013)), we set the birth rate $b = 1\%$ and death rate $d = 0.5\%$. For the baseline case we set $\phi = 0.05$, $\alpha = 0.2$, $\gamma = 0.005$.

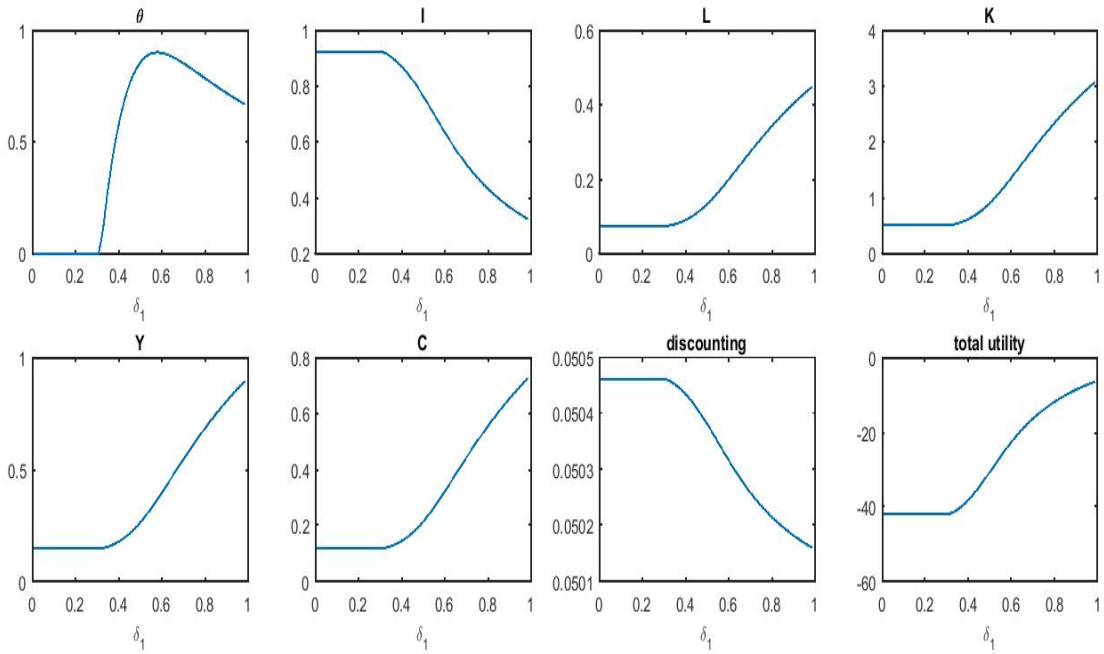
Figure 2. G function



Note: This figure plots G for 3 values of α and calculates the optimal amount of quarantine, θ^*.

There are two disease-related model parameters: the contact rate, α and the recovery rate, γ. These parameters determines the severity of disease prevalence. The higher the contact rate, the easier it is to transmit the disease and the higher the disease prevalence. The higher the recovery rate, the less severe the disease and the lower the disease prevalence. We set $\gamma = 0.005$ and examine how the G function, which determines the lockdown indicator θ, change as the contact rate α varies. Figure 2 displays G as a function of θ, when $\alpha = 0.05, 0.2$ and 0.3. That is, how the net marginal benefit of lockdown changes when the lockdown measure varies. We can see when α is very low ($\alpha = 0.05$), the G function is below the zero line throughout. It implies the marginal cost of lockdown is significantly higher than the marginal benefit, and the optimal choice is no lockdown. When the contact rate increases, the G function shifts up gradually, suggesting the net marginal benefit increases. When $\alpha = 0.2$, the optimal choice is a partial lockdown. And when the contact rate is very high $\alpha = 0.3$, the marginal benefit outweighs the marginal cost and a full lockdown is the best response. In these case, once we know θ^* we can calculate the reproduction rate $i^* = 1 - \frac{1}{R_0^*} = 1 - \frac{b+\gamma}{\alpha(1-\delta_1\theta)^2 - \phi}$. Thus, $R_0^* = \frac{\alpha(1-\delta_1\theta^*)^2 - \phi}{b+\gamma}$. This is illustrated in the simulations below where as the parameters change, so does θ^* and the R_0^* illustrated is the equilibrium reproduction rate with the optimal (partial) quarantine in place.

Figure 3. Steady state varying δ_1



Note: This figure equilibrium steady values of the endogenous variables as compliance or efficacy of lockdown, δ_1 is varied. The blue line plots the steady state values in the standard utilitarian welfare function and the red line for the extended welfare function.

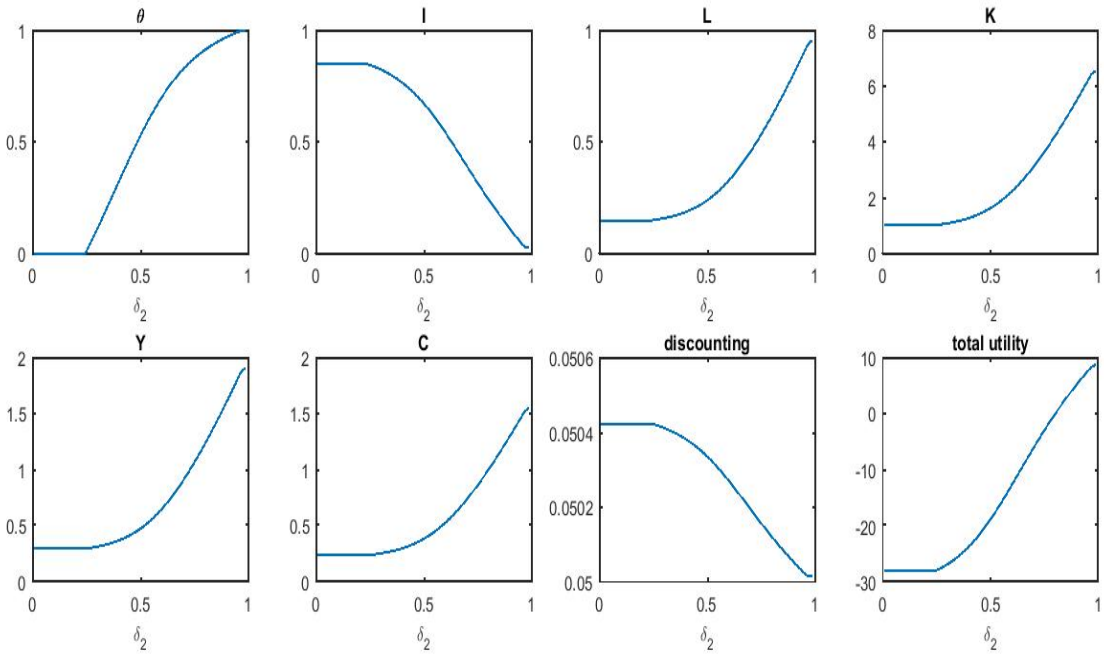
4.1 Impact of increasing efficacy of lockdown measure

We examine the impact of increasing effectiveness or compliance with quarantine – δ_1 on equilibrium steady state values of the endogenous variables in an endemic steady state. As we would expect, with low compliance, the optimal policy is not to have any quarantines and the disease circulates unchecked. However, as compliance increase, the optimal policy is to increase the quarantine and then to decrease it. The fraction of the infectives in the population depends of the product of compliance and the quarantine ($\theta\delta_1$) and when compliance is very high, the quarantine can be eased. However, infections are always decreasing in a convex way leading to better economic outcomes which increase concavily and to lower mortality.

4.2 Impact of rising productivity of working at home

Now we examine the impact of increasing productivity with quarantine – δ_2 on equilibrium steady state values of the endogenous variables in an endemic steady state. When productivity from working from home is low, the optimal policy is not to have quarantines as the loss in output from the quarantine decreases reducing the conflict with controlling inflation. As productivity from working from home increases, the optimal policy is to have more strict

Figure 4. Steady state varying δ_2



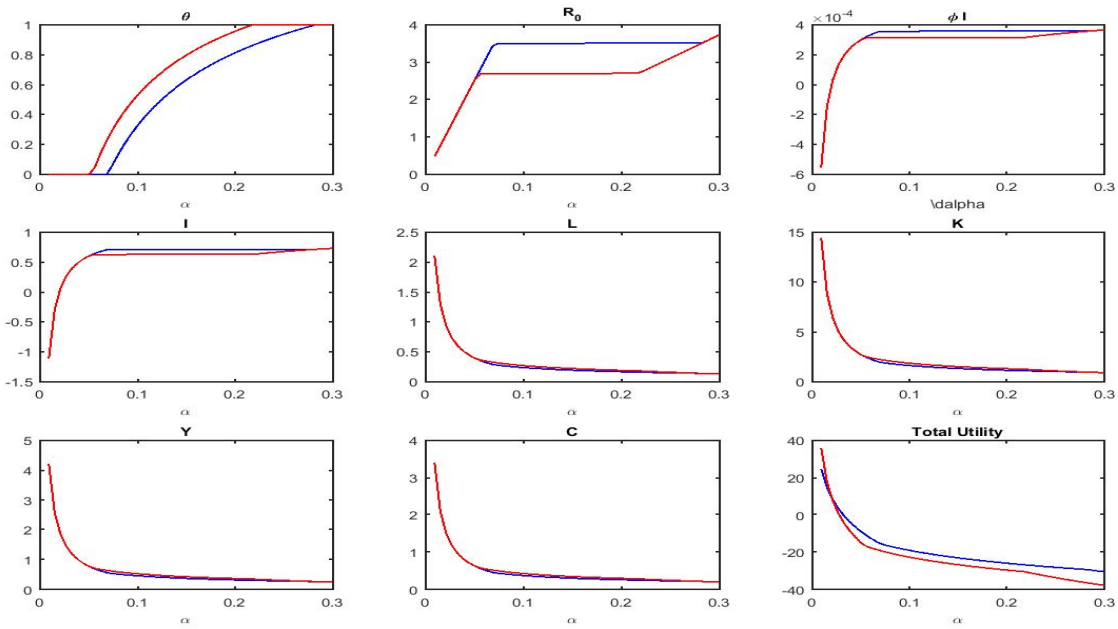
Note: This figure equilibrium steady values of the endogenous variables as productivity from working from home, δ_2 is varied. The blue line plots the steady state values in the standard utilitarian welfare function and the red line for the extended welfare function.

quarantines. With stricter quarantines infections fall and the economic variables increases and mortality decreases. While this paper has homogeneous households its implications are consistent with the emerging literature on working from home for different segments of the population (Brown and Ravallion (2020), Lekfuangfu, et al. (2020), Lewandowski, Lipowska and Magda (2020), Mongey, Pilossoph and Weinberg (2020)). For households who have higher productivity from working at home during a quarantine, the infection rates will be lower and the economic outcomes will be better than those who cannot. This suggests that differentials may emerge across different segments of the population in terms of economic and health impact and they may also have different views on desirability of a quarantines. Note that we are plotting the optimal quarantine and outcomes, so while for households with low home productivity, while the utility rates are lower and infection rates are higher, the optimal response is still to have lower (or no) quarantines.

4.3 Impact of infectivity of the disease

In figure 5 we examine the effect of varying the infectivity rate, α. The results are in line with what we expect: as the infectivity of the disease increases, after a threshold the quarantine also increases. While the severity of the quarantine increase, the level of infections

Figure 5. Steady state varying α



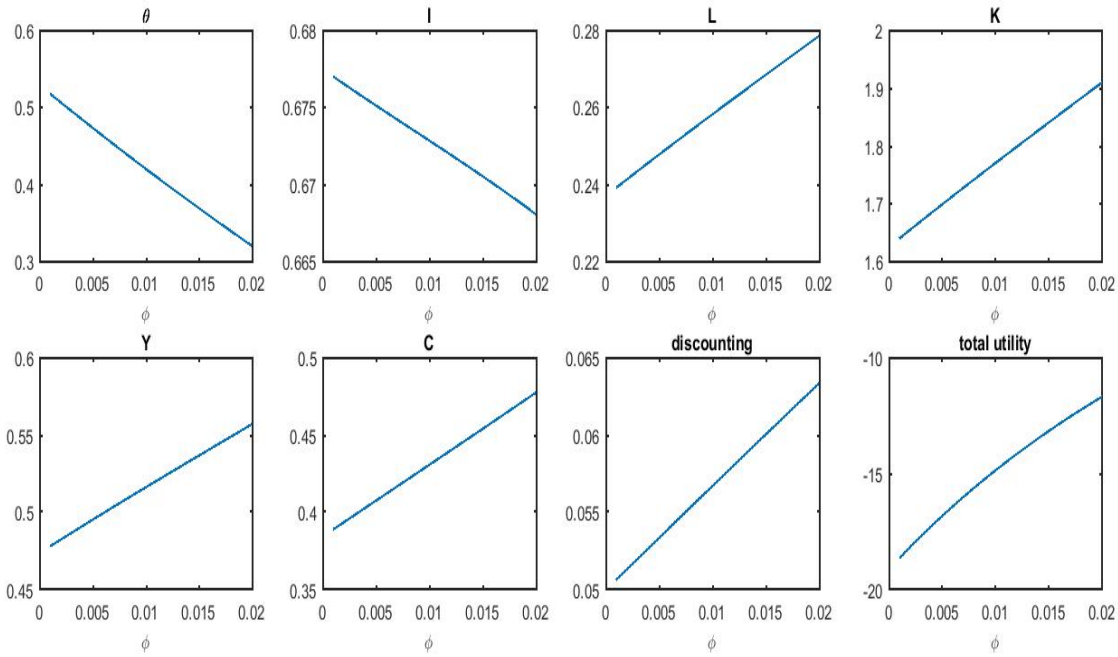
Note: This figure equilibrium steady values of the endogenous variables as infectivity from disease, α is varied. The blue line plots the steady state values in the standard utilitarian welfare function and the red line for the extended welfare function.

and mortality remains relatively constant as the infections are driven by the product $\alpha\theta^*$. However, the effect of increasing quarantine drives down the economic variables.

4.4 Impact of varying disease related death rate

In Figure 6 we vary the disease related mortality ϕ. For the utilitarian model (blue line in the figures). This gives the striking result that increasing disease related mortality decreases the intensity of the quarantines and increases total welfare even when accounting for the higher mortality and shrinking population size as there is an increase in discounting. This is similar to the "gift of the dying" in Young (1994). That paper had a Solow model and the primary effect was increase in the capital-labor ratio. While we also have increase in per capita capital stock when savings rates are changing, in our model, one of the key effects is that the increased disease related mortality leads to lower infections. Thus, conditional on survival, welfare increases. It, however, leads to the disturbing conclusion that if disease is more fatal, it is better to let it run its course even if there will be higher mortality - this is reflected in the figure for discounting which is $e^{-\int_0^t(\rho-b+d+\phi i^*(\tau)d\tau}$.

Figure 6. Steady state varying ϕ



5 An extended model with the disutility from infections and mortality

As the standard economic model gives a counter-intuitive result that increasing mortality increases social welfare, we extend the welfare function to include a welfare loss due to infections. As in the epidemiology model, the disease related mortality is a fraction of infectives, it includes welfare loss due to disease related mortality. We extend the welfare function to:

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

where $\nu(i)$ is the disutility from infections, and $\chi \geq 0$ is the weight.

Assumption 4. *$\nu(i) : \mathbb{R}_+ \to \mathbb{R}$ is a convex function with $\nu' > 0$ and $\nu'' \geq 0$ and $\nu(0)$.*

When there is no disease prevalence, the disutility from disease mortality is of course zero. For the case of loss from mortality only, we can write it as $\nu(\phi i)$ which is equivalent to the objective as ϕ is a constant in this paper.

There are several recent papers that also include such an extended welfare function and

we discuss a few of them that received prominence in the emerging literature.[19] Alvarez, Lippi and Argente (2020) use the objective[20]:

$$\int_0^\infty e^{-(\rho+\kappa)t}\left[(N_t-(L_t+S_t)\theta w)-\dot{N}_t\eta+\frac{\kappa}{\rho}N_t w\right]dt$$

where κ is the probability of finding a vaccine in a unit of time, η is the monetary loss of a mortality (see Alvarez, et al. (2020, p. 7)). They assume constant wages, w, and effectively discounting is at the interest rate, r, so that $r=\rho$. In our model wages are endogenous as $w=f_l$ and we keep track of the changing population size which makes the discounting endogenous. If we were to write the total welfare when there is no infectious disease, this is the total discounted welfare in the neo-classical steady state and the minimizing the loss as in Alvarez, et al. (2020) is equivalent to maximizing welfare via a lock-down in the scenario where diseases are prevalent. The timing of arrival of an effective vaccine is complex, as for most of the diseases with significant mortality including malaria, dengue, HIV/AIDS, diarrhoea, and all coronaviruses circulating including SARS and MERS there are no effective vaccines.[21] Even for diseases such as tuberculosis for which BCG vaccines exist coverage is mixed and there are issues of disease externalities and compliance (see Geoffard and Philipson (1996)). A recent survey also showed that only half of the UK population would definitely have the ccaccination if one was to become available and one-sixth would definitely not have one (Duffy, et al. (2020)). Thus, we do not model vaccinations in this paper.

Acemoglu, et al. (2020, especially p. 13-14) look at the efficient frontier between lives lost which in our model and economic loss. They also are concerned with the situation where a vaccine is found at time T after which there is no loss due to infections. As they are concerned with a short-run and the current scenario of low interest rates, there is no discounting. The economic loss is also a weighted wage loss from those quarantined and those isolated and the loss in expected wages of those who have premature disease mortality. The loss due to mortality is the total number of mortalities.

Jones, Phillipon and Venkateswaran (2020) have a discrete time dynamic stochastic general equilibrium model where the welfare function effectively use (see p. 4) is that of a representative household:

$$u(c_t,l_t,i_t,D_t)=l\log(c_t)+i(log(c_t)-u_k)-u_D D_t,$$

where D_t is the disease related deaths in a time period. Thus, it is a weighted average of utilities individuals who work, the disutility of those unable to work which is set equal to u_k and the welfare loss due to mortality, u_D.[22] This is also an extended welfare function but it

[19]We recognize that this is a only a very selective survey of the emerging literature and that they have different structures and modeling of epidemiology.

[20]We have changed notation in the cited equations to be close to that in our paper

[21]Smallpox is the only infectious disease to have been eradicated with a vaccination program. The vaccine was developed in 1796 by Edward Jenner and the last case was observed in 1977.

[22]Their model as the Eichenbaum, et al. (2020) paper includes endogenous labor-leisure choice and only a fraction of the infectives cannot work.

does not explicitly keep track of the changing population size though discounting but only through the loss in welfare from mortality.

Eichenbaum, Rebelo and Trabandt (2020) have a discrete-time dynamic stochastic general equilibrium model without capital. The cost of infection is a consumption tax. Each consumer is heterogenous and maximized discounted expected utility conditional of being in a given health state. If an individual is to die, there is no continuation utility. While this paper is a disaggregated model and does not model maximization total welfare, it is closer to the pure utilitarian model.

Giannitsarou, Kissler and Toxvaerd (2020) have a model where there is disease related mortality in an SEIRS model with disease related mortality where the objective is to maximize (constant) flow utility of the different classes where there is a cost to social distancing. Their welfare function is closer to the spirit of a utilitarian welfare as there is no explicit cost to infection and of mortality. These are implicit in the different flow utilities and changing sizes of the groups.

In summary, the first three papers use an extended welfare function as we do and looking at the welfare loss as in the first two papers[23] is equivalent to maximizing welfare (as done in the last two papers). How to weight the loss from mortality is an important one and different papers have followed different routes. Eichenbaum, et al. (2020) have an estimate close to EPA numbers while others have advocated using a value of statistical life measure (see Hall, Jones and Klenow (2020) and Holden, et al. (2020)).

In the rest of the paper we will specialize the welfare function to make ν a function of disease related mortality so as to be closer to the existing papers. Note, that the loss due to economic loss due to the infection is already incorporated in the constraints and the loss due to the change in population size is coming from the fact that we evaluated total rather than per capita utility. Thus, the objective problem becomes:

$$\begin{aligned} \max_{\{c,\theta\}} \quad & \int_0^\infty e^{-\rho t}[u(c) - \chi\nu(\phi i)]N dt \qquad (23) \\ = \quad & \int_0^\infty e^{-\int_0^t(\rho - b + d + \phi i(\tau))d\tau}[u(c) - \chi\nu(\phi i)]N_0 dt \end{aligned}$$

where $\nu(\phi i)$ is the loss in welfare from disease mortality, and χ is the weight. The constraints are as before, i.e. (2-6). The F.O.Cs are the same as the baseline model, except the following two equations:

$$\begin{aligned} i: \quad & -\dot{\lambda}_2 = -e^{-\Delta}\chi\phi\nu'(\phi i) - \lambda_1 f_2(k,l)(1-\theta+\delta_2\theta) + \lambda_1\phi k + \lambda_2\alpha(1-\delta_1\theta)^2(1-2i) + \\ & +\lambda_2[-b-\gamma-\phi+2\phi i] + \lambda_3\phi + \mu_3 \qquad (24) \\ \Delta: \quad & \dot{\lambda}_3 = e^{-\Delta}[u(c) - \chi\nu(\phi i)] \qquad (25) \end{aligned}$$

The disease free steady state is the same as the baseline model. The model only differs from the baseline model in incorporating the loss in welfare from disease mortality into the

[23] Acemoglu, et al. (2020) do not maximize welfare but only calculate the efficient frontier.

objective function.

Proposition 3. *Define the function:*

$$G^D(\theta) = -\tilde{\lambda}_2^D 2(1-\delta_1\theta)\delta_1\alpha(1-i)i - \tilde{\lambda}_1 f_2(k,l)(1-\delta_2)(1-i), \tag{26}$$

where

$$\begin{aligned}
&i = 1 - \frac{b+\gamma}{\alpha(1-\delta_1\theta)^2 - \phi} \\
&l = (1-\theta+\delta_2\theta)\frac{b+\gamma}{\alpha(1-\delta_1\theta)^2 - \phi} \\
&f_1(k,l) = \rho + \delta \\
&c = f(k,l) - \delta k - (b - d - \phi i)k \\
&\tilde{\lambda}_1 = \lambda_1/e^{-\Delta} = u'(c) \\
&\tilde{\lambda}_2^D = \lambda_2^D/e^{-\Delta} = \frac{u'(c)[-f_2(k,l)(1-\theta+\delta_2\theta) + \phi k] + u(c)\phi/g}{-g+b+\gamma+\phi-2\phi i - \alpha(1-\delta_1\theta)^2(1-2i)} + \\
&+\frac{\chi[-\phi\nu(\phi i) - \nu(\phi i)\phi/g]}{-g+b+\gamma+\phi-2\phi i - \alpha(1-\delta_1\theta)^2(1-2i)} \\
&g = -(\rho - b + d + \phi i).
\end{aligned} \tag{27}$$

There are three scenarios:

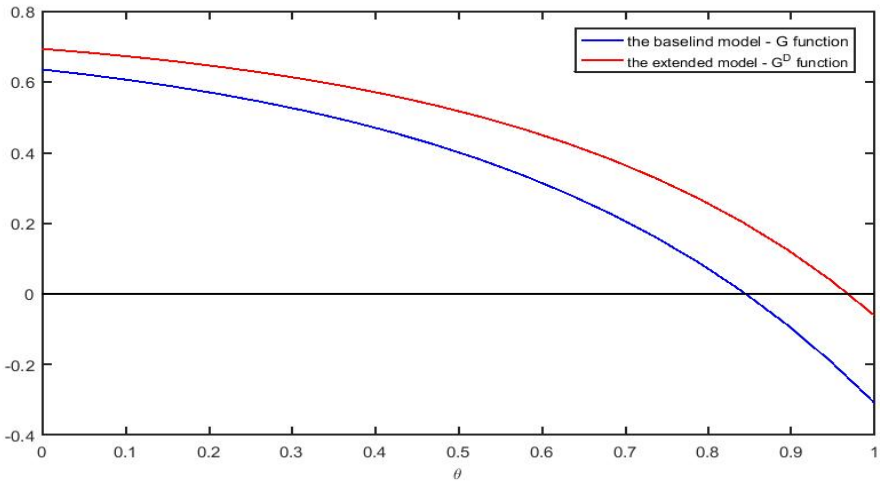
- *If $G^D(\theta)|_{\theta=0} < 0$, then $\theta^* = 0$;*
- *If $G^D(\theta)|_{\theta=1} > 0$, then $\theta^* = 1$;*
- *Otherwise, θ^* is determined by $G^D(\theta^*) = 0$.*

Given the optimal θ^*, an endemic steady state exists if $0 < \frac{b+\gamma}{\alpha(1-\delta_1\theta^*)^2-\phi} < 1$.

Proof. The proof mirrors the proof of Proposition 1 and is omitted for brevity. □

The endemic steady state in the extended model differs from the baseline model in the marginal utility of controlling disease, that is $-\lambda_2^D$. If we compare equation (28) and (21), we have $\lambda_2^D < \lambda_2$ when χ is positive. This is because when we take into account the disutility from the disease death, there is additional loss from disease prevalence, which is captured by the second term in equation (28). In other words, when we are able to control disease prevalence and reduce the infection rate, there is additional marginal utility gain. Therefore, the additional incentive from controlling the infection implies that the steady state level of lockdown is higher in the extended model than the baseline one. Figure 7 depicts function G^D in the extended model and function G in the baseline model. We can see that with every θ, the net marginal utility from additional lockdown is higher for the extended model. Thus, the steady state level of lockdown (where the curve intersects with zero line) is higher for the extended model.

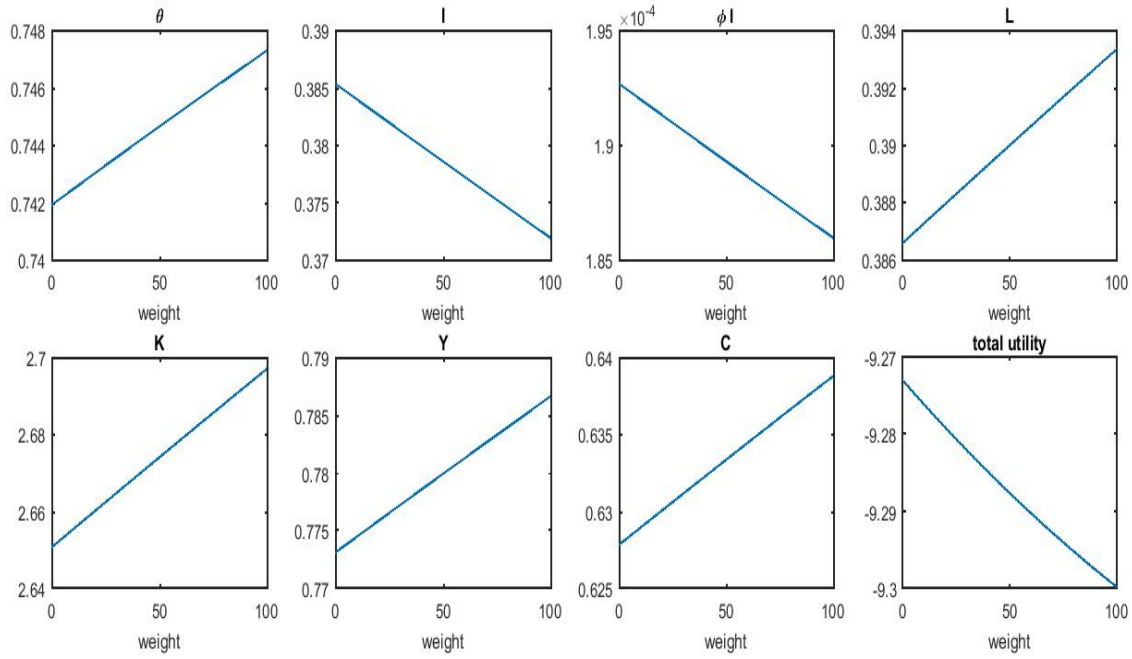
Figure 7. The determination of θ - G and G^D function



Furthermore, we look at how the economy at the endemic steady state change when we vary the weight given to loss in welfare due to mortality, χ, shown in Figure 8. When the weight increases, the level of lockdown increases. As a result, the infection rate and the disease related death drop. The labor force increases and so do capital, output and consumption. However, the total utility decreases when the weight increases, as households cares more about the disease mortality despite of the drop in death.

If we compare the results or varying the compliance to quarantine (3), varying productivity from working at home (5), and disease related mortality (6), the equilibrium steady state values for the utilitarian model is in blue and for the extended welfare function is in red. The equilibrium outcomes for the first two are similar with better disease and economic outcomes for extended welfare function. However, for varying mortality, the results are strikingly different: now under the extended welfare function, increasing mortality increases severity of the quarantine leading to greater control of the disease, lower mortality and better economic outcomes - labor force, capital, output and consumption. Thus, there is no trade-off in equilibrium between economic outcomes and disease control. While total utility initially decreases, it eventually increases as disease is controlled more aggressively.

Figure 8. The effect of varying welfare weight χ



6 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.[24] However, given the recent Covid-19 epidemic and concern with mortality how incorporating mortality in the welfare function will affect the sufficiency conditions has not been addressed in the literature to our knowledge. The problem becomes 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).

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

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

The Hamiltonian becomes

$$\begin{aligned} H(\mathbf{x}_t, \mathbf{z}_t, \lambda_t) &= e^{-\Delta}[u(c) - \chi\nu(\phi i)] + \lambda_1\{f(k, (1-\theta+\delta_2\theta)(1-i)) - c - \delta k - (b-d-\phi i)k\} + \\ &\quad +\lambda_2\{\alpha(1-\delta_1\theta)^2(1-i)i - bi - \gamma i - \phi i + \phi i^2\} + \lambda_3\{\rho - b + d + \phi i\} \\ &= e^{-\Delta}[u(c) - \chi\nu(\phi i)] + < \lambda_t, \dot{\mathbf{x}}_t > \end{aligned}$$

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

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

$$e^{-\Delta}u'(c) = \lambda_1 \tag{28}$$

$$-\lambda_1 f_2(k, (1-\theta+\delta_2\theta)(1-i))(1-\delta_2)(1-i) - \lambda_2 2(1-\delta_1\theta)\delta_1\alpha(1-i)i = 0 \tag{29}$$

$$-\dot{\lambda}_1 = \lambda_1[f_1(k,l) - \delta - b + d + \phi i] \tag{30}$$

$$\begin{aligned} -\dot{\lambda}_2 &= -\lambda_1 f_2(k, (1-\theta+\delta_2\theta)(1-i))(1-\theta+\delta_2\theta) + \lambda_1\phi k + \lambda_2\alpha(1-\delta_1\theta)^2(1-2i) + \\ &+\lambda_2[-b-\gamma-\phi+2\phi i] - e^{-\Delta}\chi\phi\nu'(\phi i) \end{aligned} \tag{31}$$

$$\dot{\lambda}_3 = e^{-\Delta}[u(c) - \chi\nu(\phi i)] \tag{32}$$

Remark 1. The Hamiltonian is not jointly concave in state and control variables if the welfare function is positive, i.e. if $u(c) - \nu(i) > 0$. In particular, the condition for the Hessian matrix to be semi-negative definite which requires the principal minors $M_j (j = 1, ..., 5)$ alter in sign, starting with a negative determinant does not satisfied in our model.

Let us rewrite the Hamiltonian as $H(k, i, \Delta, c, \theta)$ 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

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

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

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

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

6.1 Transversality conditions

The standard transversality conditions are

$$\lim_{t\to\infty} \lambda_{j,t} x_{j,t}^* = 0, j = 1, .., 3. \tag{33}$$

Note that this condition holds only at the optimal solution $x^*_{j,t}$, not for any admissible path $x_{j,t}$. Moreover, λ_t is only identified by the FOCs at $(\mathbf{x}^*_t, \mathbf{z}^*_t)$.

Many studies in literature on endogenous discounting used a weaker transversality condition where along the optimal paths

$$\lim_{t\to\infty} H = 0. \tag{34}$$

The transversality condition (34) is taken from Michel (1982) for a constant discount rate. Six and Wirl (2015) in a pollution model with endogenous discounting model[25] using the convergence of the state variable to a steady state show that if (33) holds then (34) also holds. We will also show this but our model is non-convex and the state variables need not converge to a steady state.

For the sufficiency, we assume only (33) 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. We provide a direct proof of sufficiency by proving the following transversality condition for state variables for any admissible x_t,

$$\lim_{t\to\infty} \lambda_{j,t}(x^*_{j,t} - x_{j,t}) \leq 0. \tag{35}$$

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 (33) and (34). However, if $x_{j,t}$ is bounded, then the condition $\lim_{t\to\infty} \lambda_{j,t} = 0$ implies (35). If $\lambda_{j,t} \geq 0$ and $x_{j,t} \geq 0$ then (33) implies (35). 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.

In the following, based on the standard transversality conditions (33) and special structure of the model on the convexity in control variables (but not in state variables), and the boundedness of state variables we are able to prove the transversality condition (35).

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

[25]The Six and Wirl (2015) model has one state and one control variable and is convex (see Appendix B in that paper).

stock[26]. Then is c_t is bounded by a constant[27], $c_t \leq A$, and hence

$$u(c) - \chi\nu(\phi i) \leq u(A) + \chi\nu(\phi) < +\infty \tag{36}$$

The proof proceeds via three Lemmas.

Lemma 1. *We have*

$$\lim_{t\to\infty} \lambda_{3,t}(\Delta_t - \Delta_t^*) = 0.$$

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

It follows from (32) that

$$\lambda_{3,t} = \lambda_{3,0} + \int_0^t e^{-\Delta_\tau^*}[u(c_\tau^*) - \chi\nu(\phi i_\tau^*)]d\tau.$$

The transversality condition (35) implies

$$\lim_{t\to\infty}[\lambda_{3,0} + \int_0^t e^{-\Delta_\tau^*}[u(c_\tau^*) - \chi\nu(\phi i_\tau^*)]d\tau]\Delta_t^* = 0.$$

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

$$\lambda_{3,0} = -\int_0^t e^{-\Delta_\tau^*}[u(c_\tau^*) - \chi\nu(\phi i_\tau^*)]d\tau$$

which in turn implies

$$\begin{aligned}\lambda_{3,t} &= -\int_0^\infty e^{-\Delta_\tau^*}[u(c_\tau^*) - \chi\nu(\phi i_\tau^*)]d\tau + \int_t^0 -e^{-\Delta_\tau^*}[u(c_\tau^*) - \chi\nu(\phi i_\tau^*)]d\tau \\ &= -\int_t^\infty e^{-\Delta_\tau^*}[u(c_\tau^*) - \chi\nu(\phi i_\tau^*)]d\tau.\end{aligned}$$

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

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

[26]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 $k \leq \max\{k_0, \hat{k}\} := \bar{k}$.

[27]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 $c_t \leq f(\bar{k}, 1) + \kappa\bar{k} := A$.

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

$$\begin{aligned} & \frac{1}{\rho - b + d + \phi} \int_{\Delta_t}^{\infty} e^{-q} dq \le \int_t^{\infty} e^{-\Delta_\tau} d\tau \le \frac{1}{\rho - b + d} \int_{\Delta_t}^{\infty} e^{-q} dq \\ \Leftrightarrow \quad & \frac{e^{-\Delta_t}}{\rho - b + d + \phi} \le \int_t^{\infty} e^{-\Delta_\tau} d\tau \le \frac{e^{-\Delta_t}}{\rho - b + d}. \end{aligned} \tag{37}$$

It follows from (36), (37) and using the l'Hôpital's rule we have

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

because

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

Therefore, for any feasible Δ_t,

$$\lim_{t\to\infty} \lambda_{3,t} \Delta_t = -\lim_{t\to\infty} \Delta_t \int_t^{\infty} e^{-\Delta_\tau} [u(c_\tau) - \chi\nu(\phi i_\tau)] d\tau = 0. \tag{38}$$

Together with (35) we have

$$\lim_{t\to\infty} \lambda_{3,t} (\Delta_t - \Delta_t^*) = 0.$$

Note that , since $\lim_{t\to\infty} \Delta_t = \infty$ so from (38) we get $\lim_{t\to\infty} \lambda_{3,t} = 0$.

□

Lemma 2. *We have*

$$\begin{aligned} & i) \quad \lim_{t\to\infty} \lambda_{1,t} (k_t^* - k_t) \le 0, \\ & ii) \quad \lim_{t\to\infty} \lambda_{2,t} (i_t^* - i_t) = 0. \end{aligned}$$

Proof. i) From (28) we get $\lambda_1 \geq 0$. Therefore $\lambda_{1,t}k_t \geq 0$ and (33) implies

$$\lim_{t\to\infty} \lambda_{1,t}(k_t^* - k_t) \leq 0.$$

ii) By (29) we get $\lambda_2 \leq 0$. Since we are considering the interior solutions, it follows from (29) that

$$\lambda_1 \frac{f_2(k^*, (1-\theta^* + \delta_2\theta^*)(1-i^*))(1-\delta_2)}{2(1-\delta_1\theta^*)\delta_1\alpha} = -\lambda_2 i^* \to 0 \tag{39}$$

by the transversality condition (33).

Because $0 < \theta^*, l^* = (1-\theta^* + \delta_2\theta^*)(1-i^*) < 1, 2(1-\delta_1\theta^*)\delta_1\alpha < 2\delta_1\alpha$ then

$$\frac{f_2(k^*, (1-\theta^* + \delta_2\theta^*)(1-i^*))(1-\delta_2)}{2(1-\delta_1\theta^*)\delta_1\alpha} > \frac{f_2(k^*, 1)(1-\delta_2)}{2\delta_1\alpha}.$$

When $l = 1$, then problem becomes a neoclassical model, our standard assumptions on production f implies k^* converges to a positive steady state thus $\frac{f_2(k^*,1)(1-\delta_2)}{2\delta_1\alpha} > 0$ as $t \to \infty$. Therefore, (39) implies

$$\lim_{t\to\infty} \lambda_1 = 0. \tag{40}$$

On the other hand, as $\theta^* \leq 1$ we have

$$\frac{f_2(k^*, (1-\theta^* + \delta_2\theta^*)(1-i^*))(1-\delta_2)}{2(1-\delta_1\theta^*)\delta_1\alpha} \leq \frac{f_2(k^*, l^*)(1-\delta_2)}{2(1-\delta_1)\delta_1\alpha}$$

Since

$$\begin{aligned} l^* &= (1-\theta^* + \delta_2\theta^*)(1-i^*) \geq \delta_2(1-i^*) \\ &\Rightarrow i^* \geq \frac{\delta_2 - l^*}{\delta_2}. \end{aligned}$$

Therefore

$$\begin{aligned} 0 &\leq -\lambda_2 = \lambda_1 \frac{f_2(k^*, (1-\theta^* + \delta_2\theta^*)(1-i^*))(1-\delta_2)}{2(1-\delta_1\theta^*)\delta_1\alpha i^*} \\ &\leq \lambda_1 \frac{f_2(k^*, l^*)(1-\delta_2)}{2(1-\delta_1)\delta_1\alpha i^*} \leq \lambda_1 \frac{f_2(k^*, l^*)(1-\delta_2)\delta_2}{2(1-\delta_1)\delta_1\alpha(\delta_2 - l^*)} \\ &\leq \lambda_1 \frac{f_2(k^*, l^*)(1-\delta_2)\delta_2}{2(1-\delta_1)\delta_1\alpha(\delta_2 - l^*)}. \end{aligned} \tag{41}$$

We assume that there is no scenario of full lockdown where there is no disease at all. Note that $\delta_2 = l^*$ if and only if $i^* = 0$ (no disease) and $\theta^* = 1$ (full lockdown). Therefore, it is impossible that $2(1-\delta_1)\delta_1\alpha(\delta_2 - l^*) \to 0$. Moreover, if $l^* = 0$ then $i^* = 1$ then steady

state $i^\infty = 1$, which is a contradiction with the result in Proposition 3, where an endemic steady state exists if $0 < i^\infty < 1$.[28] Therefore, $f_2(k^*, l^*)(1-\delta_2)\delta_2 < \infty$ as $t \to \infty$.

Taking the limit of both side of (41), together with (40), we have

$$\lim_{t\to\infty} \lambda_{2,t} = 0. \tag{42}$$

Because i is bounded, we have $\lim_{t\to\infty} \lambda_{2,t} i_t^* = \lim_{t\to\infty} \lambda_2 i = \lim_{t\to\infty} \lambda_{2,t}(i_t^* - i_t) = 0$. □

Michel's theorem (Michel (1982)) assumes a constant discount rate for the condition (34). We now show that it holds also for endogenous discounting based on the usual transversality conditions,

Lemma 3. *The usual transversality condition (33) implies the (34) transversality condition.*

Proof. We have

$$\begin{aligned}
&\lim_{t\to\infty} H = \\
&\lim_{t\to\infty} e^{-\Delta^*}[u(c^*) - \chi\nu(\phi i^*)] + \lim_{t\to\infty} \lambda_1\{f(k^*, (1-\theta^* + \delta_2\theta^*)(1-i^*)) - c^* - \delta k^* - (b - d - \phi i^*)k^*\} \\
&+ \lim_{t\to\infty} \lambda_2 i^* B + \lim_{t\to\infty} \lambda_3\{\rho - b + d + \phi i^*\}
\end{aligned}$$

where

$$B = [\phi - \alpha(1-\delta_1\theta^*)^2]i^* - b - \gamma - \phi.$$

It is easy to see that

$$0 \le |B| = \left|[\phi - \alpha(1-\delta_1\theta)^2]i - b - \gamma - \phi\right| \le \alpha + b + \gamma + 2\phi < \infty.$$

Note that

$$-\lim_{t\to\infty} |\lambda_2 i^* B| \le \lim_{t\to\infty} \lambda_2 i^* B \le \lim_{t\to\infty} |\lambda_2 i^* B| = 0$$

because of (42).

Moreover, using the results of Lemma 2 and Lemma 3 ($\lim_{t\to\infty} \lambda_{3,t} = \lim_{t\to\infty} \lambda_{1,t} = \lim_{t\to\infty} e^{-\Delta^*} = 0$) with the fact that $k^*, c^*, i^*, u(c^*), \nu(\phi i^*)$ and f are bounded, it implies that the transversality condition (34) is satisfied. □

6.2 Sufficiency condition

We adapt the method developed by Leitmann and Stalford (1971) for a sufficiency condition to our (non-convex) infinite-horizon optimal control problem for the endogenous discounting

[28] We denote the optimal steady state value of a variable x as x^∞ to distinguish from the optimal path of the variable which is denoted by x^*.

problem. This condition is weaker than standard Arrow-Mangasarian sufficient conditions (see Theorem V, Peterson and Zalkind (1978), page 595).

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 4. *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

$$\begin{aligned}
& \bar{H}(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t) - \bar{H}(\mathbf{x}_t^*, \mathbf{z}_t, \lambda_t) \\
= \; & e^{-\Delta^*}[u(c_t^*) - u(c_t)] - \lambda_1(c_t^* - c_t) \\
& + \lambda_1[f(k^*, l^*) - f(k^*, \hat{l})] + \lambda_2[(1 - \delta_1\theta^*)^2 - (1 - \delta_1\theta)^2]\alpha(1 - i^*)i^* \\
= \; & \lambda_1[f(k^*, l^*) - f(k^*, \hat{l})] + \lambda_2[D(\theta^*) - D(\theta)]\alpha(1 - i^*)i^*
\end{aligned}$$

where $l^* = (1 - \theta^* + \delta_2\theta^*)(1 - i^*), \hat{l} = (1 - \theta + \delta_2\theta)(1 - i^*)$ and $D(\theta) = (1 - \delta_1\theta)^2$.

Since $D(\theta)$ is convex, we have

$$D(\theta^*) - D(\theta) \leq D'(\theta^*)(\theta^* - \theta).$$

which implies

$$\lambda_2[D(\theta^*) - D(\theta)] \geq \lambda_2 D'(\theta^*)(\theta^* - \theta) = -\lambda_2 2(1 - \delta_1\theta^*)\delta_1(\theta^* - \theta). \tag{43}$$

because $\lambda_2 \leq 0$.

On the other hand, since $f(k, l)$ is concave with respect to k and l,

$$f(k^*, l^*) - f(k^*, \hat{l}) \geq f_2(k^*, l^*)(l^* - \hat{l}) = -f_2(k^*, l^*)(1 - \delta_2)(1 - i^*)(\theta^* - \theta).$$

Since $\lambda_1 \geq 0$,

$$\lambda_1[f(k^*, l^*) - f(k^*, \hat{l})] \geq -\lambda_1 f_2(k^*, l^*)(1 - \delta_2)(1 - i^*)(\theta^* - \theta). \tag{44}$$

As $u(c)$ is concave we have

$$e^{-\Delta^*}[u(c_t^*) - u(c_t)] \geq e^{-\Delta^*} u'(c_t^*)(c_t^* - c_t). \tag{45}$$

It follows from (28),(29), (43), (44), and (45) that

$$
\begin{aligned}
& H(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t) - H(\mathbf{x}_t^*, \mathbf{z}_t, \lambda_t) \\
\geq\ & [e^{-\Delta^*} u'(c_t^*) - \lambda_{1,t}](c_t^* - c_t) \\
& -[\lambda_1 f_2(k^*, l^*)(1-\delta_2)(1-i^*) + \lambda_2 2(1-\delta_1\theta^*)\delta_1\alpha(1-i^*)i^*](\theta^* - \theta) \\
=\ & 0.
\end{aligned}
$$

□

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

Assumption 5. *Assume that*

$$
\bar{H}(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t) \geq \bar{H}(\mathbf{x}_t, \mathbf{z}_t, \lambda_t) \tag{46}
$$

Remark 2. Assumption A5 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 4 $M(\mathbf{x}_t^*, \lambda_t) = \bar{H}(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t)$ 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 (46) by the properties of a concave function and the FOCs (7)-(11)

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

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

Remark 3. In a model with exogenous discounting and the objective consist of only control variables as in Goenka, Liu and Nguyen (2014), this assumption satisfies so the authors did not need A5 for the proof of sufficiency. □

We are now ready to prove the sufficient condition.

Proposition 4. *Consider the maximization problem (23) and suppose that an interior continuous* $(\mathbf{x}_t^*, \mathbf{z}_t^*)$ *and associated costate variables* λ_t *exist and satisfy (2)-(5) and (7)-(11). Then under assumptions A2-A5,* $(\mathbf{x}_t^*, \mathbf{z}_t^*)$ *is a locally optimal solution of (P).*

Proof. The results of Lemma 1 and Lemma 2 yield

$$\lim_{t\to\infty} \lambda_{1,t}(k_t^* - k_t) + \lim_{t\to\infty} \lambda_{2,t}(i_t^* - i_t) + \lim_{t\to\infty} \lambda_{3,t}(\Delta_t^* - \Delta_t) \leq 0. \tag{47}$$

Assumption A5 implies

$$H(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t) - H(\mathbf{x}_t, \mathbf{z}_t, \lambda_t) + < \lambda_t, \mathbf{x}_t^* - \mathbf{z}_t^* > \geq 0. \tag{48}$$

Taking integral over (48) we get

$$\begin{aligned}
& \int_0^\infty \{H(\mathbf{x}_t^*, \mathbf{z}_t^*, \lambda_t) - H(\mathbf{x}_t, \mathbf{z}_t, \lambda_t)] + \langle \dot{\lambda}_t, \mathbf{x}_t^* - \mathbf{x}_t \rangle\} dt \geq 0 \\
\Leftrightarrow \quad & \int_0^\infty e^{-\Delta^*}[u(c^*) - \chi\nu(\phi i^*)]dt - \int_0^\infty e^{-\Delta}[u(c) - \chi\nu(\phi i)]dt + \int_0^\infty \{< \lambda_t, \dot{\mathbf{x}}_t^* - \dot{\mathbf{x}}_t > + \langle \dot{\lambda}_t, \mathbf{x}_t^* - \mathbf{x}_t \rangle\} dt \\
\Leftrightarrow \quad & \int_0^\infty e^{-\Delta^*}[u(c^*) - \chi\nu(\phi i^*)]dt - \int_0^\infty e^{-\Delta}[u(c) - \chi\nu(\phi i)]dt \geq -\lim_{t\to\infty} \langle \lambda_t, \mathbf{x}_t^* - \mathbf{x}_t \rangle. \qquad (49)
\end{aligned}$$

Therefore, it follows from (47) 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 BGP with lockdown is locally optimal.*

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

Using the special structure of the autonomous problem we show that $\lim_{t\to\infty} \langle \lambda_t, \mathbf{x}_t^* - \mathbf{x}_t \rangle \leq 0$. This condition is needed to check (local) optimality of a path that satisfies the necessary conditions. This is crucial as 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.

7 Conclusion

This paper studied the effect of disease related mortality in an SIS model where the only way to control the incidence of the disease is via a lockdown which can be interpreted either as an optimally mandated quarantine or a self-imposed isolation chosen by the household. The changing population size due to disease related mortality makes discounting endogenous and raises two methodological issues. When we compare the equilibrium outcomes with a utilitarian welfare function or with an extended welfare function that incorporates loss due to disease related mortality, the former gives counter intuitive results. Thus, there is to be a case to use the latter. While there is a trade-off in the welfare function, in equilibrium there is no trade-off between health outcomes and economic outcomes. The second issue is that with endogenous discounting in a model which is non-convex due to disease dynamics, none of the existing sufficiency conditions apply. Using the special structure of the model we directly demonstrate the sufficiency still holds. As epidemiology models generate non-convex laws of motion for the state variables, care should be taken in the literature to show that the results based on first order conditions are meaningful.

8 Appendix 1

Given θ^*, the disease dynamic is given by:

$$H = \dot{i} = \alpha(1-\delta_1\theta)^2(1-i)i - bi - \gamma i - \phi i + \phi i^2.$$

We know that there are two steady states when $H = 0$ given by:

$$i^* = 0, \text{ and } i^* = 1 - \frac{b+\gamma}{\alpha(1-\delta_1\theta)^2 - \phi}.$$

Differentiating, we have

$$\frac{\partial H}{\partial i} = -2[\alpha(1-\delta_1\theta)^2 - \phi]i + \alpha(1-\delta_1\theta)^2 - b - \gamma - \phi.$$

In a disease free steady state:

$$\frac{\partial H}{\partial i}|_{i^*=0} = \alpha(1-\delta_1\theta)^2 - b - \gamma - \phi.$$

Thus, if $\alpha(1-\delta_1\theta)^2 - b - \gamma - \phi < 0$ the disease free steady state is stable and if $\alpha(1-\delta_1\theta)^2 - b - \gamma - \phi > 0$ it is unstable.

For the disease endemic steady state,

$$0 < i^* < 1 \Rightarrow 0 < 1 - \frac{b+\gamma}{\alpha(1-\delta_1\theta)^2 - \phi} < 1.$$

Checking its stability:

$$\frac{\partial H}{\partial i}|_{i^*>0} = -\alpha(1-\delta_1\theta)^2 - b - \gamma - \phi.$$

Thus, if $\alpha(1-\delta_1\theta)^2 - b - \gamma - \phi > 0$ then the disease endemic steady state is exists and is stable while the disease free steady state is unstable. When $\alpha(1-\delta_1\theta)^2 - b - \gamma - \phi < 0$, the disease endemic steady state does not exist and the disease free steady state is stable.

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