Social Distancing and the SIR Model with Temporary Immunity*

Peter Prastakos†

Advisors: Larry Samuelson and Philipp Strack

April 21, 2021

Abstract

We modify the Susceptible-Infected-Recovered (SIR) model for infectious diseases by first adding an inflow into the susceptible class through births as well as an outflow from the susceptible, infected, and recovered classes into a deceased class, distinguishing the fatalities caused by the infectious disease from all remaining fatalities. We find a disease-free equilibrium and an endemic equilibrium and perform some simulations and comparative statics to the model. We then account for the possibility of temporary immunity, adding a flow from the recovered to the susceptible class. After computing the new steady states, we analyze their stability. We establish analytical conditions for the local stability of the disease-free equilibrium and study the stability of the endemic equilibrium through simulations. We then perform comparative statics on the endemic equilibrium. Finally, we introduce social distancing to the model through the use of a central planner. We establish conditions under which a planner can select the optimal level of social distancing and solve for it. We conclude by applying the optimal policy conditions in a specific example.

Keywords: Social Distancing, SIR model, Temporary Immunity

*I would like to thank my thesis advisors, Prof. Samuelson and Prof. Strack, for their invaluable guidance, expertise, and encouragement throughout my senior year. All errors are my own.

†Department of Economics & Department of Mathematics, Class of 2021, Yale University

1 Introduction

As the current COVID-19 pandemic has shown, infectious diseases can have a very formidable impact on society, both purely from a medical as well as an economic standpoint. For an extensive period when no vaccines were available for COVID-19, policy makers instead had to rely primarily on so-called non-pharmaceutical interventions (NPIs) such as lockdowns or social distancing measures. Common to these measures is that they work by creating physical distance between people, thereby avoiding the spread of the disease from infected to susceptible individuals. It is no exaggeration to say that these measures have come at an enormous economic and social cost. During the deepest recession since the Great Depression, businesses have been struggling to keep afloat, the unemployment rate has skyrocketed, the elderly and ill have had access to health care curtailed and many children have had their schooling and education put on hold or greatly diminished in quality. From any number of perspectives, the current pandemic has been a calamity unlike any in recent memory.

With COVID-19 in mind, the goal of this paper is to study how an infectious disease progresses over time, analyzing its steady states and studying how the use of an NPI might impact its evolution. While we seek to study infectious diseases in generality, we also wish to make some assumptions for the model that are realistic given the epidemiological literature on COVID-19. Specifically, after monitoring healthy individuals for more than 35 years, Edridge et al. (2020) found that reinfection with the same seasonal coronavirus occurred frequently at 12 months after infection, and this result may reveal common characteristics applicable to all human coronaviruses. Thus, we will study infectious diseases under the assumption that there exists a possibility of reinfection (i.e. recovered individuals have temporary instead of permanent immunity). We also seek to consider the fatalities caused by the infectious disease as a key part of the model.

Related to our study, Taylor and Carr (2009) have investigated the case of temporary immunity where a fraction of recovered people becomes susceptible following some time delay. However, their model does not consider fatalities caused by the disease, and, coupled with the assumption that the birth rate and death rate are equal, allows for the assumption of constant population size, which we will not have. With regards to the study of NPIs, Kruse and Strack (2020) investigate the question of how to optimally engage in social distancing in order to minimize the spread of an infectious disease. Unlike the dynamical nature of their model as well as most of the models in the current literature, we will focus on studying the steady states in the evolution of the disease, and we will evaluate the optimal policy for social distancing accordingly.

We begin by presenting the standard Susceptible-Infected-Recovered (SIR) model for infectious diseases by Kermack and McKendrick (1927), assuming permanent immunity for those who recover from the disease. We modify the model to account for births and deaths in the population, distinguishing fatalities caused by the infectious disease from other fatalities. We find two steady states: a disease-free equilibrium, where the number of infected people is 0, and an endemic equilibrium, where the number of infected people is a function of the infection rate, the recovery rate, the birth and overall death rate, and the case-fatality rate of the infectious disease. We then present some simulations to observe the evolution of the disease under different choices of parameters, and we perform some comparative statics on the endemic equilibrium. As intuitively expected, we find that the number of infected people at the steady state increases as the transmission rate increases, and decreases as the recovery rate and case-fatality rate of the disease increase.

We then introduce our assumption of temporary immunity and analyze the steady states of the augmented model. We find that again, there exists a disease-free and an endemic equilibrium, and then we proceed to evaluate their stability. We establish conditions under which the disease-free equilibrium is locally stable, but we are unable to analytically find such conditions for the endemic equilibrium. Instead, we present numerous simulations that seem to indicate such conditions may exist. We conclude the steady state analysis with some comparative statics, finding that the number of infected people at the steady state is increasing in the transmission rate, decreasing in the recovery and case-fatality rate, and increasing in the rate at which recovered people lose their immunity and become resusceptible.

Finally, we introduce social distancing to the model through the use of a central planner. We establish conditions under which a planner can select the optimal level of social distancing and solve for it. We then conclude by applying the optimal policy conditions in a specific example.

2 Related Literature

There has been significant literature in epidemiology with regards to the SIR model of infectious diseases as well as some of its variations. Kermack and McKendrick (1927) present the original model, and Taylor and Carr (2009) modify it to include the possibility for temporary immunity following some fixed time delay. As aforementioned, though this paper introduces births and deaths into the model, it does not distinguish fatalities caused by the infectious disease from other fatalities, and, coupled with the assumption of the birth rate and death rate being equal, this allows for a constant population size over time.

In the field of optimal control, Rowthorn and Toxvaerd (2012) investigate the model under the assumption that individuals can be either be susceptible, or infected, but they can never be immune. In analyzing the optimal policy, they distinguish between prevention and treatment and find that, while an optimal path cannot end at a point of maximal prevention, it must end at a point of either maximal or minimal treatment. Adding the possibility of immunity, Toxvaerd and Rowthorn (2020) investigate the optimal policy of treatment and vaccination in relation to herd immunity. They find that optimal treatment involves intervention at early stages of the epidemic, while optimal vaccination defers intervention to later stages. Kruse and Strack (2020) solve the optimal control problem of social distancing under the basic SIR model, showing that an optimal policy might delay measures that decrease the transmission rate substantially to create herd-immunity and that engaging in social distancing sub-optimally early can increase the number of fatalities. Reluga (2010) formulates a differential game to study the potential value of social distancing as a mitigation measure, calculating the equilibrium behaviors under a variety of cost functions. Assuming there exists a time at which vaccination becomes immediately available to the population, the paper uses numerical methods to calculate the total costs of the an epidemic under equilibrium behaviors as a function of time until mass vaccination, and shows that the window of opportunity for vaccine development lengthens as the efficiency of social distancing improves. Toxvaerd (2020) sidesteps the difficulties involved in differential games by assuming each individual is perfectly forward-looking but insignificantly small relative to the size of the population. In investigating the dynamics of social distancing under equilibrium behavior, and finds that equilibrium social distancing erises endogenously around the peak of the epidemic, when disease prevalence reaches a critical threshold determined by preferences.

With specific applications to COVID-19, Acemoglu et al. (2020) numerically study targeted lockdowns in a multi-group SIR model whose infection, hospitalization, and fatality rates vary between groups between the "young," "middle-aged," and "old." For baseline parameter values for COVID-19 applied to the US, they find that optimal policies differentially targeting different age groups (which have different levels of risk) significantly outperform optimal uniform policies and most of the gains can be realized by having stricter lockdown policies on the oldest group. Ellison (2020) provides a quick survey of results on the classic SIR model under heterogeneity and suggests some implications for COVID-19, presenting both a version of heterogeneity that assumes uniform matching between individuals, where the probability that any two agents are randomly matches is proportional to the product of their activity levels, as well as one that assumes homophilic matching, in which agents are more likely to interact with others in their own subpopulation.

3 Model

3.1 Basic SIR

One of the most fundamental models for infectious diseases is the SIR model, which classifies individuals to be Susceptible (S), Infected (I), or Recovered (R), the latter of which assumes that once someone recovers from the virus, they become permanently immune and are hence removed from the population of susceptible people. The ordinary differential equations that describe the model are as follows:

$$\frac{dS}{dt} = -\beta S(t)\frac{I(t)}{N}$$

$$\frac{dI}{dt} = \beta S(t)\frac{I(t)}{N} - \gamma I(t)$$

$$\frac{dR}{dt} = \gamma I(t)$$

where t is time, β is the infection rate, γ is the recovery rate, $S(t)$ is the number of susceptible people, $I(t)$ is the number of people infected, $R(t)$ is the number of people who have recovered and developed immunity to the infection, and N is the total population size (note $\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$ and thus N is constant).

In this paper, we wish to study the model while accounting for changes to the population size, i.e. assuming there is a certain flow into the susceptible class as well as a flow out of all three classes into a deceased class. We also wish to distinguish the fatalities caused by the infectious disease from the remaining fatalities. Since much of the concern with the current COVID-19 pandemic is that people die from it, a model that does not specifically incorporate the case-fatality rate would not be too useful in studying such a disease.

Thus, we modify the model as follows:

$$\frac{dS}{dt} = \mu - \beta S(t)I(t) - \mu S(t) \tag{1}$$

$$\frac{dI}{dt} = \beta S(t)I(t) - (\mu + \mu_i + \gamma)I(t) \tag{2}$$

$$\frac{dR}{dt} = \gamma I(t) - \mu R(t) \tag{3}$$

where μ denotes the birth rate as well as the death rate for all causes except the infectious disease being studied (the simplifying assumption to set the two rates to be equal to each other was made for normalization purposes to be explained in the steady state analysis that shortly follows), and μ_i denotes the case fatality rate specific to the disease.

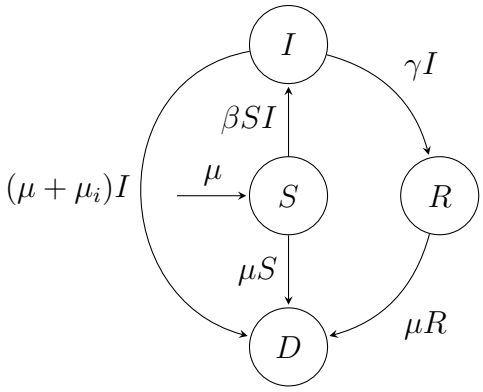


Figure 1: A flow diagram that describes the modified SIR model (where we have added a D node for clarity to reflect the deceased class).

The intuition for the $\beta S(t)I(t)$ term is as follows: consider a round-robin tournament, where everyone gets in contact with everyone else; then every susceptible person gets in contact with every infected person, and that is all multiplied by the probability of transmission per contact to get the total flow from the susceptible class to the infected class at a particular instance. For comparison, in the standard SIR model presented above, the corresponding term $\beta S(t)\frac{I(t)}{N}$ is interpreted as the number of infected people multiplied by the probability of interacting with an infected person (which is seen as the proportion of infected people in the population) multiplied by the probability of such an interaction leading to the transmission of the disease. However, since population size is not constant in our modified model, this modelling choice would make our steady-state analysis uninformative (as will become clear shortly), and thus we opt for the round-robin scenario.

To find the 2 steady states, we solve the system of linear equations created by setting all 3 equations to 0. Starting from equation (2), we have that $0 = \beta S^* I^* - (\mu + \gamma)I^*$, and hence $I^* = 0$ solves the system. Then, from equation (3), we have that $R^* = 0$, and from (1), we have that $S^* = 1$. For the remainder of the analysis we will refer to this steady state as the **disease-free equilibrium (DFE)**. Without the assumption that the birth rate and general death rate are equal, we would have that, in this particular steady state, $S^* = b/\mu$ (where b is the birth rate and μ is the death rate). However, since this steady state is not as instructive as one where $I^*, R^* > 0$ (i.e. the disease exists), we make the normalization that $b = \mu$ to simplify computations that are nonessential.

We will now explain the modelling choice for the entry term into the susceptible class, $+\mu$. At first glance, the most intuitive choice for the rate of entry into the population should be a linear term, $\mu N(t)$, where $N(t) = S(t) + I(t) + R(t)$ is the population size, and μ is the birth rate. However, in that case, we wouldn't be able to make useful steady state observations - if we were to consider $\frac{dN}{dt}$, where we will now denote δ as the general death

rate to distinguish it from μ, we would have

$$\frac{dN}{dt} = (\mu - \delta)N(t) - \mu_i I(t).$$

Plugging in $I^* = 0$ from equation (2), we have that if $\mu > \delta$, $N(t)$ diverges to $+\infty$, and if $\mu < \delta$, $N(\infty) = 0$ (i.e. everybody dies). Thus there does not exist a unique DFE due to the addition of population size as an extra variable. Intuitively, one would also expect environmental factors, such as the carrying capacity of the environment, to adjust as the population size diverges to ∞, increasing death rate and decreasing the birth rate so as to limit the population, and the reverse to happen if the population approaches zero. Hence, the decision to make the entry term constant, similarly to the aforementioned round-robin modelling choice for the disease transmission, not only makes the steady-state analysis useful, but is also the appropriate modelling choice given our intuition of the population dynamics.

We now proceed to find the second steady state of the system, which we will refer to as the **endemic equilibrium (EE)** for the remainder of the analysis. To find the second steady state, we assume $I^* \neq 0$ in equation (2) and solve for S^*. Then, plugging into (1) and in turn (3), we have that

$$S^* = \frac{\mu + \mu_i + \gamma}{\beta}, I^* = \frac{\mu}{\mu + \mu_i + \gamma} - \frac{\mu}{\beta}, R^* = \frac{\gamma}{\mu + \mu_i + \gamma} - \frac{\gamma}{\beta}.$$

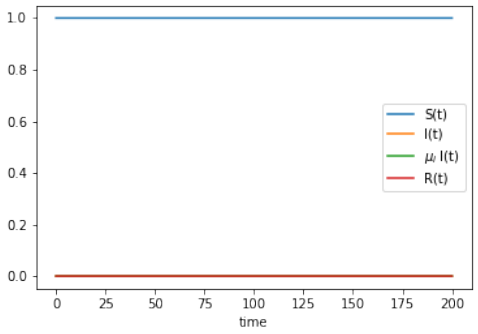
Having fully described the model and discovered the two unique steady states, we present some simulations of the evolution of the system under different parameter values (Figure 2; see Appendix A.1 for code). We observe that if $\beta < \mu + \mu_i + \gamma$, the system cannot converge to the EE since otherwise we have $I^*, R^* < 0$, which is impossible. In fact, running numerous simulations as in Appendix A.1 (adjusting the time span), we observe that when $\beta < \mu + \mu_i + \gamma$, the system converges to the DFE, and when $\beta > \mu + \mu_i + \gamma$, the system converges to the EE (when $\beta = \mu + \mu_i + \gamma$ the EE and DFE coincide).

Let us now do some comparative statics to the endemic equilibrium. We have that

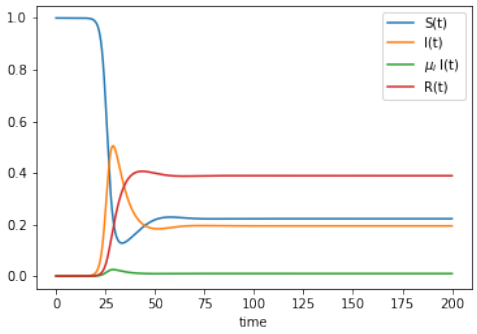
$$\frac{\partial S^*}{\partial \gamma} = \frac{\partial S^*}{\partial \mu} = \frac{\partial S^*}{\partial \mu_i} = \frac{1}{\beta} > 0$$

$$\frac{\partial S^*}{\partial \beta} = -\frac{\mu + \mu_i + \gamma}{\beta^2} < 0$$

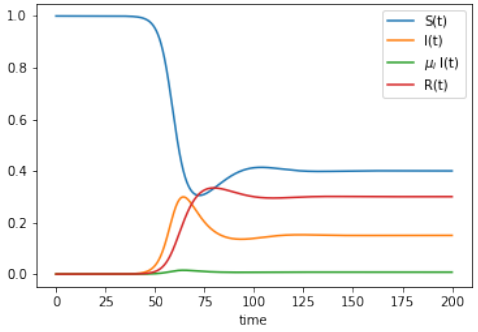
Thus, an increase in the birth/general death rate, the disease case fatality rate, or the recovery rate increases the number of susceptible people at the steady state, while an increase in the infection rate does the opposite.



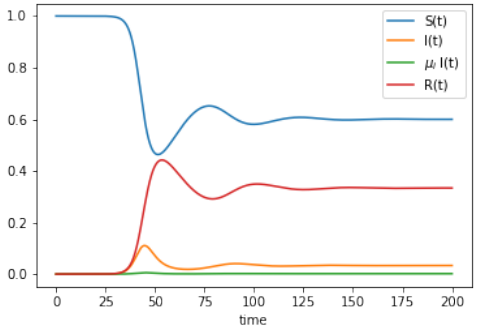
(a) $\beta = 0.1, \gamma = 0.9, \mu = \mu_i = 0.05$



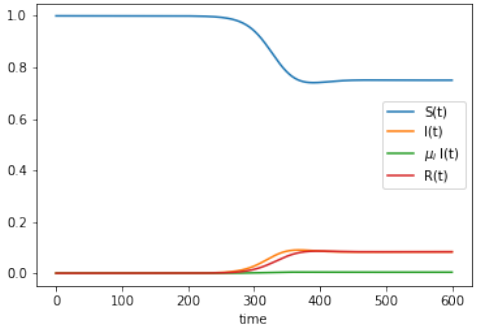
(b) $\beta = 0.9, \gamma = 0.1, \mu = \mu_i = 0.05$



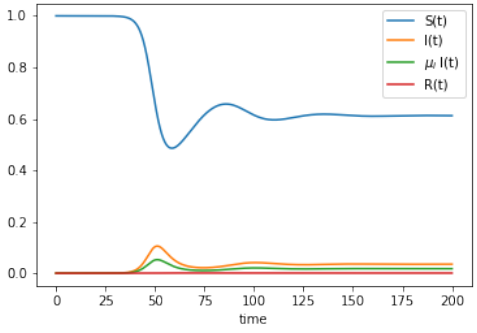
(c) $\beta = 0.5, \gamma = 0.1, \mu = \mu_i = 0.05$



(d) $\beta = 1, \gamma = 0.5, \mu = \mu_i = 0.05$



(e) $\beta = 0.2, \gamma = \mu = \mu_i = 0.05$



(f) $\beta = 0.9, \gamma = 0.001, \mu_i = 0.5, \mu = 0.05$

Figure 2: Evolution of modified SIR model under different parameters with initial conditions $I(0) = 1 \times 10^{-8}$,$S(0) = 1 - I(0), R(0) = 0$

In a similar vein, we have that

$$\frac{\partial I^*}{\partial \beta} = \frac{\mu}{\beta^2} > 0$$

$$\frac{\partial I^*}{\partial \gamma} = \frac{\partial I^*}{\partial \mu_i} = -\frac{\mu}{(\mu + \mu_i + \gamma)^2} < 0$$

$$\frac{\partial I^*}{\partial \mu} = \frac{\mu_i + \gamma}{(\mu + \mu_i + \gamma)^2} - \frac{1}{\beta}$$

As expected, an increase in the infection rate increases the number of infected people at the steady state, while an increase in the recovery rate or case fatality rate decreases it. The birth/death rate has an inconclusive effect on I^*. If β is small enough (i.e. $\beta < \frac{(\mu+\mu_i+\gamma)^2}{\mu_i+\gamma}$), an increase in the birth/death rate yields a decrease in I^*. Using some numerical estimates from Toda (2020) as well as Center for Disease Control and Prevention (2019a) and Johns Hopkins Coronavirus Resource Center (2021), we have that $\frac{\partial I^*}{\partial \mu}$ is empirically positive (see Appendix A.2 for code).

Finally, we have,

$$\frac{\partial R^*}{\partial \beta} = \frac{\gamma}{\beta^2} > 0$$

$$\frac{\partial R^*}{\partial \mu} = \frac{\partial R^*}{\partial \mu_i} = -\frac{\gamma}{(\mu + \mu_i + \gamma)^2} < 0$$

$$\frac{\partial R^*}{\partial \gamma} = \frac{\mu + \mu_i}{(\mu + \mu_i + \gamma)^2} - \frac{1}{\beta}$$

Here we have some results that may seem nontrivial: an increase in the infection rate increases the number of recovered people at the steady state, while an increase in the case fatality rate or birth/death rate has the opposite effect. A possible intuitive explanation may be that, if more people get infected, more people can become recovered at the steady state, and if more people die from the virus, fewer of the infected people can recover. Perhaps more surprisingly, an increase in the recovery rate has an inconclusive effect on the number of recovered (while our intuition might have expected a strictly positive partial derivative). Again, we have that if β is small enough (that is, $\beta < \frac{(\mu+\mu_i+\gamma)^2}{\mu_i+\mu}$), an increase in γ has a *negative* effect on the number of recovered people at the EE. In fact, this seems to be the case for COVID-19 using the aforementioned data estimates (A.2).

3.2 SIR model with temporary immunity

The SIR model, albeit being used widely in epidemiological/economic literature, does not seem to realistically account for diseases that do not have permanent recovery (one of which is suspected to be COVID-19). Thus we introduce a modified version of the SIR model that

can account for temporary immunity.

The system of equations that describe the model are as follows:

$$\frac{dS}{dt} = \mu - \beta S(t)I(t) - \mu S(t) + \sigma R(t) \tag{4}$$

$$\frac{dI}{dt} = \beta S(t)I(t) - (\mu + \mu_i + \gamma)I(t) \tag{5}$$

$$\frac{dR}{dt} = \gamma I(t) - (\mu + \sigma)R(t) \tag{6}$$

where σ denotes the return rate to the susceptible class, and the rest is as in the traditional SIR model in section 3.1. We still hold the assumption that the birth rate is equal to the death rate for all causes except the disease of interest, as discussed in 3.1.

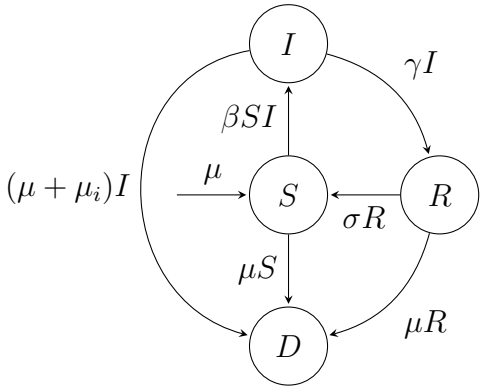


Figure 3: A flow diagram that describes the modified SIRS model (where the D node reflecting the deceased class).

The process to solve for the two unique steady states is exactly as in the basic SIR model. Starting from equation (5), we have that either $I^* = 0$, in which case we get that $R^* = 0$ from (6) and then $S^* = 1$ from (4), or $I^* \neq 0$, in which case we get that

$$S^* = \frac{\mu + \mu_i + \gamma}{\beta}$$

from (5), and plugging that in to (4), as well as $R^* = \frac{\gamma I^*}{\mu+\sigma}$ from (6), we get

$$I^* = \frac{\mu(1 - \frac{\mu+\mu_i+\gamma}{\beta})}{\mu + \mu_i + \frac{\mu\gamma}{\mu+\sigma}}, R^* = \frac{\gamma\mu(1 - \frac{\mu+\mu_i+\gamma}{\beta})}{(\mu + \mu_i)(\mu + \sigma) + \mu\gamma}.$$

We will now analyze the stability of the steady states. First, let us review some notation from point-set topology. For a space $(X, ||\cdot||)$, an open ball of radius r centered around x,

is defined as the set

$$B_r(x) := \{y : ||y - x|| < r\}.$$

We define a steady state $x \in X$ to be **locally stable** if there exists $r > 0$ and $B_r(x) \subseteq X$ such that for any initial point $y \in B_r(x)$, the system will converge to x over time. In other words, if any y in some neighborhood of x (including x) was selected as the initial point of the system at $t = 0$, the system should converge to x over time.

One way to test for steady state stability of a system of differential equations, is to check whether each eigenvalue of the Jacobian matrix evaluated at the steady state is negative (or has a negative real part). If any eigenvalue is positive (or has a positive real part), then the steady state is unstable. Otherwise, the test is inconclusive.

In this case, the Jacobian matrix in $\mathbb{R}^{3x3}$ is

$$J = \begin{bmatrix} -\beta I(t) - \mu & -\beta S(t) & \sigma \\ \beta I(t) & \beta S(t) - \mu - \mu_i - \gamma & 0 \\ 0 & \gamma & -\mu - \sigma \end{bmatrix}$$

For the steady state $(S^*, I^*, R^*) = (1, 0, 0)$, we have that

$$J^* = J\Big|_{(S^*,I^*,R^*)} = \begin{bmatrix} -\mu & -\beta & \sigma \\ 0 & \beta - \mu - \mu_i - \gamma & 0 \\ 0 & \gamma & -\mu - \sigma \end{bmatrix}$$

Solving for the eigenvalues, we get that

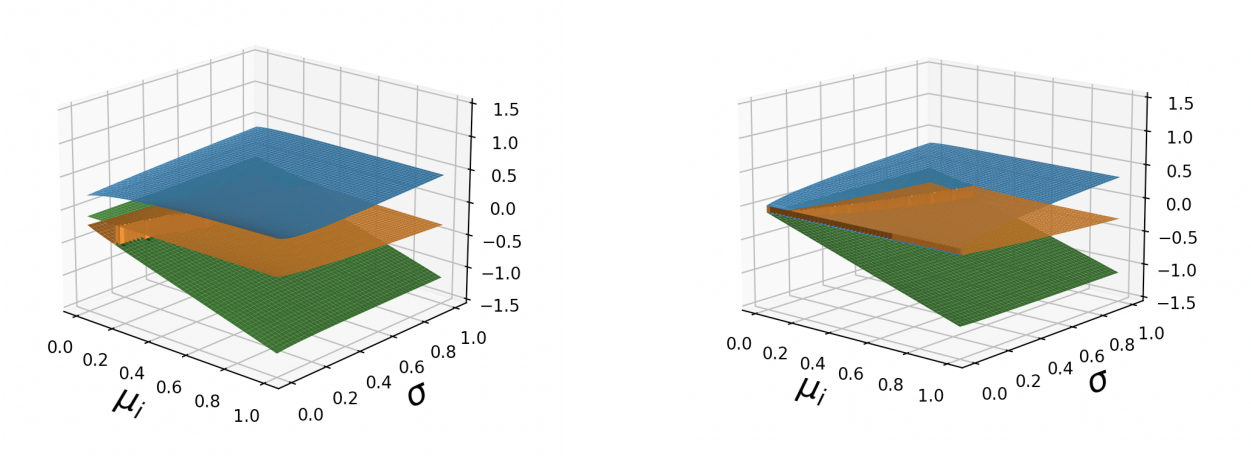
$$\lambda_1 = -\mu, \lambda_2 = -\sigma - \mu, \lambda_3 = \beta - \mu - \mu_i - \gamma.$$

Thus, under the assumption that $\mu, \mu_i, \gamma, \beta, \sigma > 0$, we observe that the steady state $(S^*, I^*, R^*) = (1, 0, 0)$ is locally stable if $\beta < \mu + \mu_i + \gamma$ and unstable if $\beta > \mu + \mu_i + \gamma$.

For the steady state $(S^*, I^*, R^*) = (\frac{\mu+\mu_i+\gamma}{\beta}, \frac{\mu(1-\frac{\mu+\mu_i+\gamma}{\beta})}{\mu+\mu_i+\frac{\mu\gamma}{\mu+\sigma}}, \frac{\gamma\mu(1-\frac{\mu+\mu_i+\gamma}{\beta})}{(\mu+\mu_i)(\mu+\sigma)+\mu\gamma})$, we have that

$$J^* = J\Big|_{(S^*,I^*,R^*)} = \begin{bmatrix} \frac{\mu(\mu+\mu_i+\gamma-\beta)}{\mu+\mu_i+\frac{\mu\gamma}{\mu+\sigma}} - \mu & -\mu - \mu_i - \gamma & \sigma \\ \frac{\mu(\beta-\mu-\mu_i-\gamma)}{\mu+\mu_i+\frac{\mu\gamma}{\mu+\sigma}} & 0 & 0 \\ 0 & \gamma & -\mu - \sigma \end{bmatrix}$$

Solving for the eigenvalues here is very computationally intensive. The characteristic



(a) $\beta = 0.1, \gamma = 0.5, \mu = 0.1$ (b) $\beta = 0.4, \gamma = 0.3, \mu = 0.1$

Figure 4: Plot of the 3 eigenvalues of the Jacobian matrix at the EE, for varying values of μ_i and σ and fixed values of β, γ, and μ. For both (a) and (b), we observe that one eigenvalue (blue plane) is clearly positive for the entire range of μ_i and σ, and thus the EE is unstable for these choices of fixed parameters.

polynomial (denoted $f(x)$) is

$$f(x) = -\frac{1}{\mu(\gamma+\mu+\sigma)+(\mu+\sigma)\mu_i}(\gamma\mu\sigma(\mu+\sigma)(-\beta+\gamma+\mu+\mu_i)+(x+\mu+\sigma)$$
$$(-\mu(\mu+\sigma)(\gamma+\mu+\mu_i)(-\beta+\gamma+\mu+\mu_i)+x(\mu(-\gamma\sigma+\beta(\mu+\sigma)+x(\gamma+\mu+\sigma))+x(\mu+\sigma)\mu_i)))$$

Using the assumption that $\mu, \mu_i, \gamma, \beta, \sigma > 0$, the polynomial simplifies to

$$f(x) = \gamma\mu\sigma(\mu+\sigma)(-\beta+\gamma+\mu+\mu_i)+(x+\mu+\sigma)(-\mu(\mu+\sigma)(\gamma+\mu+\mu_i)$$
$$(-\beta+\gamma+\mu+\mu_i)+x(\mu(-\gamma\sigma+\beta(\mu+\sigma)+x(\gamma+\mu+\sigma))+x(\mu+\sigma)\mu_i))$$

However, this remains a cubic function and thus computing the roots does not yield informative constraints for stability (to see this, run the command `Roots[f(x)==0, x]` in Mathematica, replacing $f(x)$ with the polynomial above). Figure 4 shows examples of certain choices of parameters for which the endemic equilibrium is unstable (see Appendix B.1 for code).

The natural question one might ask then is how the system progresses in situations where the endemic equilibrium is unstable, as in Figure 4. First, we observe that if $\beta < \mu + \mu_i + \gamma$, we have that $\frac{\mu+\mu_i+\gamma}{\beta} > 1$, which implies that, if the system were at the endemic equilibrium, $I^* = \frac{\mu(1-\frac{\mu+\mu_i+\gamma}{\beta})}{\mu+\mu_i+\frac{\mu\gamma}{\mu+\sigma}} < 0$, which is impossible. Thus, we have that the system can converge to the EE only if $\beta > \mu + \mu_i + \gamma$, and the EE is unstable if $\beta < \mu + \mu_i + \gamma$. Figure 5 shows the

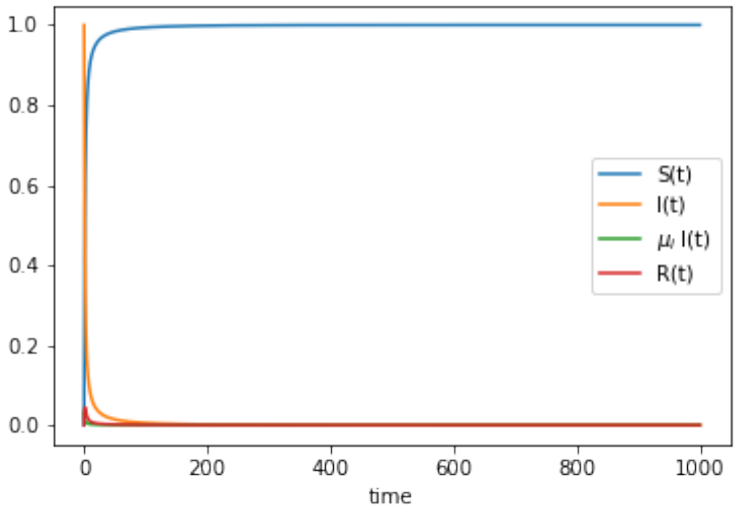


Figure 5: Evolution of the system with initial conditions $S(0) = 0.0001, I(0) = 0.9999, R(0) = 0$, where $\beta = 0.92, \mu = 0.8, \mu_i = 0.03, \gamma = 0.1, \sigma = 0.15$. Note $\beta < \mu + \mu_i + \gamma$, and the initial conditions were purposely selected to show what happens even in extreme cases where the initial conditions are very far from the DFE.

time path of the variables when the EE is unstable, where we see that the system converges to the DFE. This immediately resolves our inquiries regarding the instability of the EE in Figures 4a and 4b.

However, we have only observed sufficient, not necessary, conditions under which the EE is unstable. Since we cannot derive conditions for the local stability of the EE algebraically, we seek to do so through simulations. We begin by hypothesizing that the EE is locally stable if $\beta > \mu + \mu_i + \gamma$, as this intuitively seems like a natural choice. The simulation in Appendix B.2 seems to indicate that it is quite plausible this is the case. In the simulation, we run through all combinations of values of the parameters in (approximate) increments of 0.1 from 0 to 1 for a total of 10^5 combinations, and for those combinations that have $\beta > \mu + \mu_i + \gamma$, we set the initial conditions $(S(0), I(0), R(0))$ to be close to the endemic equilibrium, $(\frac{\mu+\mu_i+\gamma}{\beta}, \frac{\mu(1-\frac{\mu+\mu_i+\gamma}{\beta})}{\mu+\mu_i+\frac{\mu\gamma}{\mu+\sigma}}, \frac{\gamma\mu(1-\frac{\mu+\mu_i+\gamma}{\beta})}{(\mu+\mu_i)(\mu+\sigma)+\mu\gamma})$. By definition of local stability, it should then follow that for initial conditions that are very close to the EE, the system converges to the EE. Indeed, we find that for all combinations where $\beta > \mu + \mu_i + \gamma$ the last elements in our S, I, and R arrays get very close to S^*, I^*, R^*. We note that, trivially, since such a numerical simulation is by nature not analytical, it cannot prove our hypothesis, but it does provide some support for it.

Now, we shall proceed with some comparative statics. As before, we have that

$$\frac{\partial S^*}{\partial \gamma} = \frac{\partial S^*}{\partial \mu} = \frac{\partial S^*}{\partial \mu_i} = \frac{1}{\beta} > 0$$

$$\frac{\partial S^*}{\partial \beta} = -\frac{\mu + \mu_i + \gamma}{\beta^2} < 0.$$

We now also have that

$$\frac{\partial S^*}{\partial \sigma} = 0.$$

Thus, perhaps surprisingly, we observe that an increase in the return rate to the susceptible class does not have an impact on the number of susceptible people at the steady state.

Additionally, we have,

$$\frac{\partial I^*}{\partial \beta} = \frac{\mu(\gamma + \mu + \mu_i)}{\beta^2(\mu + \mu_i + \frac{\gamma\mu}{\mu+\sigma})} > 0$$

$$\frac{\partial I^*}{\partial \gamma} = -\frac{\mu^2(1 - \frac{\mu+\mu_i+\gamma}{\beta})}{(\mu+\sigma)(\mu + \mu_i + \frac{\mu\gamma}{\mu+\sigma})^2} - \frac{\mu}{\beta(\mu + \mu_i + \frac{\mu\gamma}{\mu+\sigma})} < 0$$

$$\frac{\partial I^*}{\partial \sigma} = \frac{\gamma\mu^2(1 - \frac{\mu+\mu_i+\gamma}{\beta})}{(\mu+\sigma)^2(\mu + \mu_i + \frac{\mu\gamma}{\mu+\sigma})^2} > 0$$

$$\frac{\partial I^*}{\partial \mu_i} = -\frac{\mu(1 - \frac{\mu+\mu_i+\gamma}{\beta})}{(\mu + \mu_i + \frac{\mu\gamma}{\mu+\sigma})^2} - \frac{\mu}{\beta(\mu + \mu_i + \frac{\mu\gamma}{\mu+\sigma})} < 0$$

$$\frac{\partial I^*}{\partial \mu} = -\frac{\mu(1 - \frac{\gamma+\mu+\mu_i}{\beta})(1 - \frac{\gamma\mu}{(\mu+\sigma)^2} + \frac{\gamma}{\mu+\sigma})}{(\mu + \mu_i + \frac{\gamma\mu}{\mu+\sigma})^2} - \frac{\mu}{\beta(\mu + \mu_i + \frac{\gamma\mu}{\mu+\sigma})} + \frac{1 - \frac{\gamma+\mu+\mu_i}{\beta}}{\mu + \mu_i + \frac{\gamma\mu}{\mu+\sigma}}$$

where the signs for $\frac{\partial I^*}{\partial \gamma}, \frac{\partial I^*}{\partial \sigma}$, and $\frac{\partial I^*}{\partial \mu_i}$ come from using the observation that $\frac{\mu+\mu_i+\gamma}{\beta} < 1$. Thus, as in section 3.1, we have that an increase in the infection rate leads to an increase in the number infected at the EE, while an increase in the recovery rate or case fatality rate decreases it, and an increase in the birth/death rate has an inconclusive effect on I^*. We now also have that an increase in the return rate σ leads to a increase in the number infected at the EE, which aligns with our basic intuition.

Finally, we have,

$$\frac{\partial R^*}{\partial \beta} = \frac{\gamma\mu(\mu + \mu_i + \gamma)}{\beta^2(\gamma\mu + (\mu + \mu_i)(\mu + \sigma))} > 0$$

$$\frac{\partial R^*}{\partial \sigma} = -\frac{\gamma\mu(\mu + \mu_i)(1 - \frac{\gamma+\mu+\mu_i}{\beta})}{(\gamma\mu + (\mu + \mu_i)(\mu + \sigma))^2} < 0$$

$$\frac{\partial R^*}{\partial \mu_i} = -\frac{\gamma\mu(1-\frac{\gamma+\mu+\mu_i}{\beta})(\mu+\sigma)}{(\gamma\mu+(\mu+\mu_i)(\mu+\sigma))^2} - \frac{\gamma\mu}{\beta(\gamma\mu+(\mu+\mu_i)(\mu+\sigma))} < 0$$

$$\frac{\partial R^*}{\partial \gamma} = -\frac{\mu(\gamma^2\mu + 2\gamma(\mu+\mu_i)(\mu+\sigma) - (\beta-\mu-\mu_i)(\mu+\mu_i)(\mu+\sigma))}{\beta(\gamma\mu+(\mu+\mu_i)(\mu+\sigma))^2}$$

$$\frac{\partial R^*}{\partial \mu} = \frac{-\gamma\sigma(\gamma\mu_i+(\mu+\mu_i)^2) + \beta\gamma(-\mu^2+\mu_i\sigma)}{\beta(\gamma\mu+(\mu+\mu_i)(\mu+\sigma))^2}$$

Thus, as expected, we note that an increase in the infection rate increases the number of recovered people at the EE, while an increase in the case fatality rate or return rate has the opposite effect. Finally, a change in the recovery rate or birth/death rate has an inconclusive effect on R^*.

3.3 Adding social distancing through central planning

We will now introduce social distancing to the model through the transmission rate β. We consider the problem of a central planner who is seeking to find the optimal social distancing policy that can be implemented from a set of possible policies. We do this, by no longer consider β as solely capturing the epidemiological rate of infection, but also capturing the selected amount of investment in social distancing. That is, β, which we will now denote as β_s for clarity, is selected by the social planner from a set $B = [\underline{\beta}, \overline{\beta}] \subset [0,1]$, where $\underline{\beta} < \overline{\beta}$. The choice of $\underline{\beta}$ is then the transmission rate that corresponds to the maximum investment in social distancing, and $\overline{\beta}$ is the transmission rate that corresponds to the minimum investment. Just as before, our system of ordinary differential equations is:

$$\frac{dS}{dt} = \mu - \beta_s S(t)I(t) - \mu S(t) + \sigma R(t) \tag{7}$$

$$\frac{dI}{dt} = \beta_s S(t)I(t) - (\mu+\mu_i+\gamma)I(t) \tag{8}$$

$$\frac{dR}{dt} = \gamma I(t) - (\mu+\sigma)R(t) \tag{9}$$

Note now that, in contrast with the previous models, the number of susceptible, infected, and recovered people at the steady state (S^*, I^*, R^*) is dependent on the planner's choice of β_s. We thus have that the steady state *functions* are $(S^*(\beta_s), I^*(\beta_s), R^*(\beta_s)) = (1,0,0)$ and

$$(S^*(\beta_s), I^*(\beta_s), R^*(\beta_s)) = (\frac{\mu+\mu_i+\gamma}{\beta_s}, \frac{\mu(1-\frac{\mu+\mu_i+\gamma}{\beta_s})}{\mu+\mu_i+\frac{\mu\gamma}{\mu+\sigma}}, \frac{\gamma\mu(1-\frac{\mu+\mu_i+\gamma}{\beta_s})}{(\mu+\mu_i)(\mu+\sigma)+\mu\gamma}).$$

In order to evaluate the optimal policy, we introduce a utility function $u\colon B \to \mathbb{R}$ as well

as a cost function $c\colon B \to [0, \infty)$. Both u and c then are functions of the planner's choice of β_s. We will assume that the planner utility function is only directly impacted by the number of infected people at the steady state, I^*, and is indifferent about S^* or R^*. Note that the fatalities cause by the disease are proportional to I^*, so such a utility function can be interpreted as the central planner only caring about deaths. Additionally, we will make the reasonable assumptions that c is decreasing (higher β_s would imply less investment in social distancing and thus a smaller economic cost of social distancing), convex (the marginal cost of investing in social distancing should be increasing), twice differentiable (for optimization purposes), and $c(\overline{\beta}) = 0$ (the cost of not implementing any social distancing measures and keeping the default infection rate should be 0).

The social planner thus has the following problem:

$$\max_{\beta_s} u(\beta_s) - c(\beta_s) = \max_{\beta_s} -I^*(\beta_s) - c(\beta_s) = \max_{\beta_s} \frac{\mu(\frac{\mu+\mu_i+\gamma}{\beta_s} - 1)}{\mu + \mu_i + \frac{\mu\gamma}{\mu+\sigma}} - c(\beta_s). \tag{10}$$

Note here we are considering the EE steady state since in the case of no disease, the central planner can simply set $\beta_s = \overline{\beta}$ and have no social distancing in order to solve the maximization.

We now seek to find conditions for concavity of equation (10). We assume positive values for all parameters. Let $\alpha_1 = \frac{\mu}{\mu+\mu_i+\frac{\mu\gamma}{\mu+\sigma}}$, $\alpha_2 = \mu + \mu_i + \gamma$. Then in order to perform the maximization, we want to ensure $\alpha_1\alpha_2(\beta_s)^{-1} - \alpha_1 - c(\beta_s)$ is concave. For a twice-differentiable function, we have that a sufficient condition for concavity is the second derivative being non-positive. Taking the first derivative, we get

$$-\frac{\alpha_1\alpha_2}{\beta_s^2} - c'(\beta_s) \tag{11}$$

and then taking the second we get

$$\frac{\alpha_1\alpha_2}{\beta_s^3} - c''(\beta_s).$$

Thus, a sufficient condition for optimality is that $c''(\beta_s) \geq \frac{\alpha_1\alpha_2}{\beta_s^3}$. Thus, not only does the cost function need to be convex, but it needs to be convex enough (the second derivative has a greater lower bound than 0).

Assuming then that the above inequality holds, we have, from equation (11) that the optimal choice of β_s is any point $\beta^* \in [\underline{\beta}, \overline{\beta}]$ that solves

$$c'(\beta^*) = -\frac{\mu(\mu + \mu_i + \gamma)}{(\beta^*)^2(\mu + \mu_i + \frac{\mu\gamma}{\mu+\sigma})}. \tag{12}$$

Let us now apply this result in an example and confirm it using a simulation. Let $[\underline{\beta}, \overline{\beta}] = [0.1, 0.9]$. Using α_1, α_2 as before, let us assume

$$c''(\beta_s) = \frac{\alpha_1\alpha_2}{\beta_s^4} > \frac{\alpha_1\alpha_2}{\beta_s^3}.$$

Integrating twice (setting constants to 0), we have that

$$c(\beta_s) = \frac{\alpha_1\alpha_2}{6\beta_s^2} = \frac{\mu(\mu+\mu_i+\gamma)}{6\beta_s^2(\mu+\mu_i+\frac{\mu\gamma}{\mu+\sigma})}.$$

Since concavity holds by construction, we use equation (12) to find the optimal choice of β_s. We thus have

$$c'(\beta^*) = -\frac{\mu(\mu+\mu_i+\gamma)}{3(\beta^*)^3(\mu+\mu_i+\frac{\mu\gamma}{\mu+\sigma})} = -\frac{\mu(\mu+\mu_i+\gamma)}{(\beta^*)^2(\mu+\mu_i+\frac{\mu\gamma}{\mu+\sigma})}$$

so $\beta^* = \frac{1}{3} \in [0.1, 0.9]$. The simulation in Appendix C.1 fully aligns with our computed value of β^*.

4 Conclusion

This paper investigated the impact of certain modifications to the SIR model for infectious diseases that seem to more accurately represent infectious diseases like COVID-19. After adding births and deaths into the model and distinguishing the fatalities caused by the infectious disease from other fatalities, we observed a disease-free and an endemic equilibrium, established conditions under which the system converges to each steady state through simulations, and performed comparative statics on the endemic equilibrium, finding that I^* is increasing in the infection rate β, and decreasing in the recovery rate γ and the case fatality rate μ_i.

We then augmented the model by allowing for the possibility of recovered individuals to become reinfected through a flow $\sigma R(t)$ from the recovered into the susceptible class. We again found two equilibria, and we analyzed their local stability. We observed that the disease-free equilibrium is locally stable if $\beta < \mu + \mu_i + \gamma$ by analyzing the signs of the eigenvalues of the Jacobian matrix at the DFE. Though we were not able to make a similar algebraic observation for the eigenvalues of the matrix at the EE, we noted that the EE is (vacuously) unstable if $\beta < \mu + \mu_i + \gamma$ since we assume the number of infected, susceptible, and recovered people must always be nonnegative. We also provided some support for our hypothesis that the EE is locally stable if $\beta > \mu + \mu_i + \gamma$ through numerical simulations. We

then performed some comparative statics to the model, and observed that, as in the previous model, I^* is decreasing in μ_i and γ and increasing in β and σ, as intuitively expected.

Finally, we introduced social distancing to the model through a central planner who can control the transmission rate β_s in some interval $[\underline{\beta}, \overline{\beta}]$. After assuming the central planner only cares about deaths at the steady state (so $u(\beta_s) = I^*(\beta_s)$) and the cost function is twice-differentiable, convex, decreasing, and $c(\overline{\beta}) = 0$, we established some sufficient conditions under which the planner can find the optimal transmission rate $\beta^* \in [\underline{\beta}, \overline{\beta}]$. We then solved the optimization for an explicit example of a cost function.

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A Appendix for Section 3.1

A.1 Figure 2 Simulations

```
import numpy as np
from scipy.integrate import odeint
import matplotlib.pyplot as plt


beta = [0.1, 0.9, 0.5, 1, 0.2, 0.9]
gamma = [0.9, 0.1, 0.1, 0.5, 0.05, 0.001]
mu = [0.05]*6
mu_i = [0.05, 0.05, 0.05, 0.05, 0.05, 0.5]
for n in range(0,6):
    def sir(x,t):
        s = x[0]
        i = x[1]
        r = x[2]
        dsdt = mu[n] - beta[n]*s*i - mu[n]*s
        didt = beta[n]*s*i - (mu[n]+gamma[n]+mu_i[n])*i
        drdt = gamma[n]*i - mu[n]*r
        return [dsdt,didt,drdt]

    x0 = [0.99999999, 0.00000001, 0] #initial conditions used in Toda
    (2020)
    t = np.linspace(0, 200, 10000)
    x = odeint(sir,x0,t)

    s=x[:,0]
    i=x[:,1]
    r=x[:,2]
    plt.plot(t,s, label="S(t)")
    plt.plot(t,i, label="I(t)")
    plt.plot(t,mu_i[n]*i, label="$\mu_i$ I(t)")
    plt.plot(t,r, label="R(t)")
    plt.xlabel('time')
    plt.legend()
    plt.savefig(str(n)+".png")

    plt.show()
```

A.2 Comparative statics using COVID-19 data

```
import numpy as np
#mu 0.008697 (death), 0.0114 (birth), average 0.01 (annual; Source: https://www.cdc.gov/nchs/fastats/births.htm and https://www.cdc.gov/nchs/fastats/deaths.htm)
#mui 0.018 (Source: https://coronavirus.jhu.edu/data/mortality)
#beta 0.38 (Toda, 2020)
#gamma 0.1 (Toda, 2020)

#Note 1: While the model distinguishes fatalities caused by the infectious disease from other fatalities, there doesn't exist such a clear distinction in the data. Thus, some fatalities allocated to COVID-19 in the data may in fact be from other causes.
#Note 2: General mortality rate is from 2019 data, while COVID-19 data is from 2020. Thus the time periods do not exactly coincide, but should give a reasonable estimate.

#converting to daily rates when necessary to match the calibration in Toda (2020)
mu = np.linspace(0.008/365, 0.012/365, num=10)
mui= np.linspace(0.01/452, 0.03/452, num=10) #452 is number of days since Jan 21,2020 - first confirmed case of COVID-19 in the US (approximate)
beta = np.linspace(0.2, 0.4, num=10)
gamma = np.linspace(0.08, 0.12, num=10)
countIpos = 0
countRneg = 0
for i in mu:
    for j in beta:
        for k in mui:
            for l in gamma:
                dIdmu = (k+l)/(i+k+l)**2 - 1/j
                dRdgamma = (k+i)/(i+k+l)**2 - 1/j
                if dIdmu > 0:
                    countIpos = countIpos+1
                if dRdgamma < 0:
                    countRneg = countRneg+1
print(countIpos)
print(countRneg)
```

B Appendix for Section 3.2

B.1 3D plot of eigenvalues

```
%matplotlib auto

from typing import Tuple
import argparse
import numpy as np
from numpy import linalg as LA
import matplotlib.pyplot as plt


class EEStability:
    def __init__(self,
                 beta: float,
                 mu: float,
                 gamma: float,
                 N: int = 1000,
                 mu_i_min: float = 1e-8,
                 mu_i_max: float = 1.-1e-8,
                 sigma_i_min: float = 1e-8,
                 sigma_i_max: float = 1.-1e-8) -> None:
        """Namespace for simulation of EE stability"""
        self.beta = beta
        self.mu = mu
        self.gamma = gamma

        self.N = N
        self.mu_i = np.linspace(mu_i_min, mu_i_max, N)
        self.sigma = np.linspace(sigma_i_min, sigma_i_max, N)

        self.matrices = None
        self.vals = None


    def get_matrices(self) -> np.array:
       """Returns Jacobian at EE"""
       self.matrices = np.zeros((self.N,self.N,3,3))
       for i in range(self.N):
           for j in range(self.N):
```

```
                self.matrices[i,j,0,0] = self.mu * (self.mu+self.mu_i[i]+
self.gamma-self.beta) / \
                                                (self.mu + self.mu_i[i] + (self
.mu*self.gamma) / (self.mu + self.sigma[j])) \
                                                 - self.mu
                self.matrices[i,j,0,1] = -self.mu - self.mu_i[i] - self.
gamma
                self.matrices[i,j,0,2] = self.sigma[j]
                self.matrices[i,j,1,0] = self.mu * (self.beta - self.mu -
self.mu_i[i] - self.gamma) / \
                                               (self.mu + self.mu_i[i] + (self.mu*
self.gamma) / (self.mu + self.sigma[j]))
                self.matrices[i,j,1,1] = 0.
                self.matrices[i,j,1,2] = 0.
                self.matrices[i,j,2,0] = 0.
                self.matrices[i,j,2,1] = self.gamma
                self.matrices[i,j,2,2] = -self.mu - self.sigma[j]
        return self.matrices


    def SVD(self) -> Tuple[np.array,np.array,np.array]:
        self.vals = np.zeros((self.N,self.N,3))
        for i in range(self.N):
            for j in range(self.N):
                self.vals[i,j,:], _ = LA.eig(self.matrices[i,j,:,:])
        return self.mu_i, self.sigma, self.vals


    def plot(self, **kwargs) -> None:
        mu_i, sigma = np.meshgrid(self.mu_i, self.sigma)
        fig = plt.figure()
        ax = fig.add_subplot(projection='3d')
        ax.plot_surface(mu_i, sigma, self.vals[:,:,0], label=r"$\lambda_1$
")
        ax.plot_surface(mu_i, sigma, self.vals[:,:,1], label=r"$\lambda_2$
")
        ax.plot_surface(mu_i, sigma, self.vals[:,:,2], label=r"$\lambda_3$
")
        ax.set_xlabel(r"$\mu_i$", fontsize=20)
        ax.set_ylabel(r"$\sigma$", fontsize=20)
        ax.set_zlim(-1.5, 1.5)
        plt.show()

```

```

def main():
    example_params = {"beta": 0.1,
                      "mu": 0.1,
                      "gamma": 0.5}
    simulation = EEStability(**example_params)
    simulation.get_matrices()
    simulation.SVD()
    simulation.plot()


main()
```

B.2 Analysis of Stability of EE through Simulations

```
import numpy as np
from scipy.integrate import odeint
import matplotlib.pyplot as plt


a = np.linspace(0.001, 1, 10)
b = np.linspace(0.001, 1, 10)
eecase=0
eecorrectcase=0
for mu in a:
    for mu_i in a:
        for beta in b:
            for gamma in a:
                for sigma in a:
                    if beta > mu+mu_i+gamma:
                        eecase = eecase+1
                        s_star= (mu+mu_i+gamma)/beta
                        i_star = mu*(1-(mu+mu_i+gamma)/beta)/(mu+mu_i+mu*gamma/(mu+sigma))
                        r_star = mu*gamma*(1-(mu+mu_i+gamma)/beta)/((mu+mu_i)*(mu+sigma)+mu*gamma)
                        def sir(x,t):
                            s = x[0]
                            i = x[1]
                            r = x[2]
                            dsdt = mu - beta*s*i - mu*s + sigma*r
                            didt = beta*s*i - (mu+gamma+mu_i)*i
```

```
                                drdt = gamma*i - (mu+sigma)*r
                                return [dsdt,didt,drdt]

                            x0 = [s_star+0.0001, i_star-0.0001, r_star]

                            t = np.linspace(0, 12000, 10000)
                            x = odeint(sir,x0,t)

                            s=x[:,0]
                            i=x[:,1]
                            r=x[:,2]
                            if abs(s[9999]-s_star) < 0.001 and abs(i[9999]-
    i_star) < 0.001:
                                eecorrectcase= eecorrectcase+1
print(eecase)
print(eecorrectcase)
```

C Appendix for Section 3.3

C.1 Computing the optimal transmission rate β^*

```
import numpy as np

mu = 0.1
mu_i = 0.05
gamma = 0.4
sigma = 0.3
beta = np.linspace(0.1,0.9,200)
max_function = [0]*200

for n in range(0,200):
    max_function[n] = mu*((mu+mu_i+gamma)/beta[n]-1)/(mu+mu_i+(mu*gamma)/(
    mu+sigma))-mu*(mu+ mu_i+ gamma)/(6*beta[n]*beta[n]*(mu+mu_i+(mu*gamma)
    /(mu+sigma)))

maxval = 0
for n in range(0,200):
    if max_function[n] > max_function[maxval]:
        maxval = n
print(beta[maxval])
#output is 0.3331658291457287 which is about 1/3
```