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EQUILIBRIUM SOCIAL DISTANCING

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This paper presents an economic model of an epidemic in which susceptible individuals may engage in costly social distancing in order to avoid becoming infected. Infected individuals eventually recover and acquire immunity, thereby ceasing to be a source of infection to others. Under non-cooperative and forward-looking decision making, equilibrium social distancing arises endogenously around the peak of the epidemic, when disease prevalence reaches a critical threshold determined by preferences. Spontaneous, uncoordinated social distancing thus acts to flatten the curve of the epidemic by reducing peak prevalence. In equilibrium, social distancing stops once herd immunity sets in, but acts to extend the duration of the epidemic beyond the benchmark of a non-behavioral epidemiological model. Comparative statics with respect to the model parameters indicate that the curve becomes flatter (i) the more infectious the disease is and (ii) the more severe the health consequences of the disease are for the individuals.

Equilibrium Social Distancing

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Abstract. This paper presents an economic model of an epidemic in which susceptible individuals may engage in costly social distancing in order to avoid becoming infected. Infected individuals eventually recover and acquire immunity, thereby ceasing to be a source of infection to others. Under non-cooperative and forward-looking decision making, equilibrium social distancing arises endogenously around the peak of the epidemic, when disease prevalence reaches a critical threshold determined by preferences. Spontaneous, uncoordinated social distancing thus acts to *flatten the curve* of the epidemic by reducing peak prevalence. In equilibrium, social distancing stops once herd immunity sets in, but acts to extend the duration of the epidemic beyond the benchmark of a non-behavioral epidemiological model. Comparative statics with respect to the model parameters indicate that the curve becomes flatter (i) the more infectious the disease is and (ii) the more severe the health consequences of the disease are for the individuals.

JEL Classification: C73, I18.

Keywords: Economic epidemiology, social distancing, non-pharmaceutical interventions, infection control.

"...[the Black Death made people] shun and flee from the sick and all that pertained to them, and thus doing, each thought to secure immunity for himself".

- Boccaccio's The Decameron (1353)

1. Introduction

The world is currently gripped by the COVID-19 pandemic. At the time of writing, there is no vaccine available against this virus and no antiviral therapies to increase the speed of recovery. The only available strategies to stem the spread of the disease are behavioral interventions such as *social distancing*. Social distancing refers to any non-pharmaceutical intervention, taken by individuals or by policy makers, which acts to decrease the contact rate between infected and susceptible individuals.[1] Reducing the

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[1]When social distancing is imposed on a sub-population by a government, it is often referred to as *quarantines*; when it is voluntary and chosen by individuals themselves, it is known as *self-isolation*.

contact rate is often held to be the central tool to *"flatten the curve"*, i.e. to reduce disease incidence and hence the number of infected individuals. In some countries such as the United Kingdom, governments have refrained from directly imposing restrictions on individuals and have instead appealed to citizens to act in the interests of society and to voluntarily withdraw from the public space. The question is then, when left to their own devices, how much social distancing will there be in equilibrium? And how does this depend on biological and preference parameters? The United Kingdom's response to the COVID-19 pandemic and the scientific research underlying it is heavily predicated on behavior changes that reduce the contact rate in the population (see Ferguson et al., 2020, Ferguson et al. 2006 and Halloran et a., 2008). The overwhelming focus of governments' responses to the epidemic on behavioral responses of the population makes it incumbent upon researchers to be clear about how and why individuals act as the epidemic unfolds. What are their constraints and incentives? Will they voluntarily comply with directions given by public health officials or do governments need to compel certain behaviors, as has now been seen across the world?

On current evidence, much of the thinking around social distancing is based on epidemiological simulations and modeling that eschew a nuanced analysis of human behavior in the face of epidemics. Specifically, most of the modeling is based on assumptions about how individuals will behave under a set of interventions such as travel restrictions, school closures and bans on sporting and cultural events and mass gatherings. But since behavior is the central issue, we must be careful about how we model it and strive to incorporate behavioral considerations more fully into our analysis of disease control. We cannot simply rely on traditional analyses that do not model behavior but augment these with ad-hoc interventions that rely on guesses about compliance rates. The standard epidemiological models are an excellent starting point for analysis, but must be made complete by fully integrating them with more sophisticated models of human decision-making and behavior. Empirical evidence shows that individuals indeed respond to disease outbreaks by changing behavior (see e.g. Kumar et al. 2012, Bayham et al., 2015, Bayham and Fenichel, 2016 and references therein).

Lauren Gardner, a public health expert and modeler of epidemics at Johns Hopkins, recently stated that

"When people change their behavior, [epidemiological] model parameters are no longer applicable."[2]

In other words, we must revisit the traditional models to fully account for human behavior. This paper is a contribution towards this goal.

This paper analyzes social distancing by means of a continuous-time, infinite-horizon

[2]https://www.nytimes.com/2020/03/13/us/coronavirus-deaths-estimate.html?referringSource=articleShare

economic-epidemiological model of an infectious disease. A closed population of individuals face a disease of the susceptible-infected-removed variety, which is an appropriate setting for analyzing the spread of COVID-19.[3] At each instant, individuals non-cooperatively decide whether to engage in costly social distancing and in doing so, trade off the benefits of social interactions against the risk of contracting the communicable disease.

I find that each individual's optimal strategy is described succinctly in terms of a threshold infection probability, which depends on aggregate disease prevalence. For sufficiently low disease prevalence, as may be found at the beginning or the end of an epidemic, the risks from social interactions are small and thus individuals choose not to socially distance themselves. For higher levels of disease prevalence, the risk of exposure may outweigh the benefits and so individuals switch to social distancing. In this case, aggregate equilibrium disease prevalence remains constant through time until sufficiently many individuals have gone through the cycle *susceptible* $\rightarrow$ *infected* $\rightarrow$ *recovered* to cause disease prevalence to fall without further social distancing. In a sense, individuals' equilibrium social distancing decisions act as a flow rate regulator between healthy and recovered individuals, where the underlying uncontrolled flow rates are determined by the biological features of the disease.

The analysis emphasizes that while the equilibrium extent of social distancing is not socially optimal, aggregate equilibrium infection across the epidemic is lower than what a traditional non-economic epidemiological analysis would suggest. In other words, a purely non-behavioral model would tend to overstate the severity of the epidemic relative to one that features rational behavior. While this by no means implies that equilibrium is socially optimal, it does mean that the worst-case scenario under a laissez-faire policy is not that predicted by purely biological considerations.[4]

To further contrast the predictions of the economic model with those of a purely epidemiological model, I characterize the equilibrium dynamics in terms of several properties of the aggregate disease dynamics, namely in terms of *peak prevalence, duration* and *final size distribution*. I find that equilibrium social distancing will tend to reduce peak prevalence, increase duration and decrease cumulative incidence, which can be thought of as an inverse measure of herd immunity. Interestingly, I find that the comparative statics predictions of the of economic model are the reverse of those in the uncontrolled epidemiological model. For example, peak prevalence and cumulative incidence are both *increasing* in the infectiousness of the disease in the biological model, whereas they are

[3] This model is also known as that of a general epidemic. See Kermack and McKendrick (1927) for the original treatment. Disease-induced deaths can be incorporated explicitly in the model but are not considered in order to simplify the exposition.

[4] In the present model, equilibrium will not be socially optimal since individuals do not internalize the positive externalities that flow from their decisions to socially distance themselves.

decreasing in the economic model. This is because the endogenously determined social distancing decisions of the individuals react to higher infectiousness by engaging in more protective behavior.

The formal economic analysis of social distancing is sparse. Sethi (1978) analyzes the problem of a social planner in the context of the simpler susceptible-infected-susceptible SIS model of disease in which recovered individuals are not immune to further infection. Chen et al. (2011), Gersovitz (2010) and Toxvaerd (2019) consider equilibrium social distancing in the SIS model under decentralized decision-making, while Rowthorn and Toxvaerd (2015) consider the interaction between social distancing and treatment with antivirals, both in equilibrium and under central planning. Toxvaerd and Rowthorn (2020) consider the equilibrium and socially optimal inducement of immunity via vaccination and treatment. Reluga (2010) analyzes a differential game model of social distancing with a finite horizon, no discounting and (eventual) vaccination. Chen (2012) studies social distancing in the susceptible-infected-recovered SIR model under more general matching functions than the standard mass-action specification used in the epidemiological literature and finds that for some specifications of the matching function, there may be scope for multiple Nash equilibria at each point in time, making it difficult to predict the course of the epidemic.

In contrast to these papers, I consider an SIR framework in which individuals are perfectly forward-looking but where each is small relative to a large population, thereby side-stepping the difficulties involved in differential games. Furthermore, in the present setting there is a unique equilibrium path through the epidemic, allowing me not only to predict of the course of the epidemic (within the model) but also to perform meaningful comparative statics with respect to biological and preference parameters. Last, Fenichel et al (2011) and Fenichel (2013) consider social distancing in the SIR model when individuals have concave utility functions. Fenichel (2013) considers the properties of decentralized equilibrium and socially optimal social distancing when susceptible, infected and recovered individuals can vary their exposure levels differentially. He argues that in such a setting, a second-best policy that requires all individuals to socially distance themselves to the same extent may be inferior to a laissez-faire policy. Here, the main focus is on the differences between the dynamics under equilibrium behavior and those in the uncontrolled epidemiological model. In addition, the dependence of the dynamics on preference parameters is explored and the present results are thus complementary to his analysis.

The paper is organized as follows. In Section 2, I present the economic-epidemiological model and briefly review the classical analysis of the susceptible-infected-recovered model. This is to set the stage for the subsequent analysis of individual decision-making and

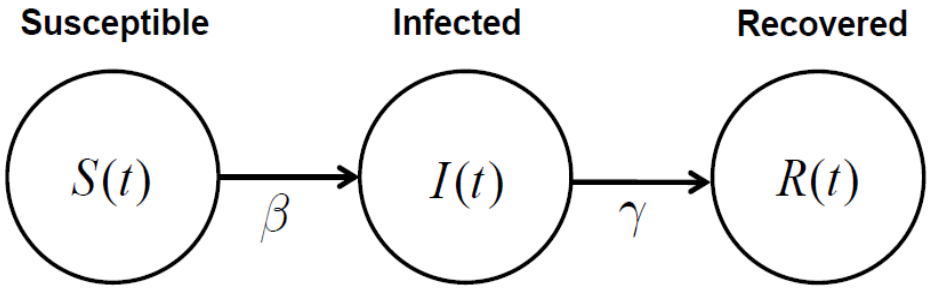

Figure 1: States and Flows in the SIR Model.

characterization of equilibrium dynamics under social distancing, contained in Section 3. In Section 4, I discuss the results and conclude.

2. THE MODEL

The model is an economic extension of the classical *susceptible-infected-recovered* model and is simple to describe. Time is continuous and runs indefinitely. A closed population consists of a continuum $[0, 1]$ of infinitely lived individuals who can at each instant $t \geq 0$ each be in one of three states, namely *susceptible* or *infected* or *recovered*. The measure of susceptible individuals is $S(t)$, the measure of infected (and infectious) individuals is $I(t)$ and the measure of recovered individuals is $R(t)$. Because the population size is normalized to one, these measures can be interpreted as fractions. Henceforth, $I(t)$ shall be referred to as *disease prevalence*.

At each instant, the population mixes homogeneously. This corresponds to pair-wise random matching where each individual has an equal chance of meeting any other individual, irrespective of the health status of the two matched individuals. A match between an infected and a susceptible individual may infect the susceptible. The rate at which infection is transferred in such a match, absent social distancing, is denoted by $\beta > 0$. This parameter captures the infectivity of the disease. Recovered individuals are immune to further infection and also cannot carry the disease. Coupled with the assumption of homogeneous mixing, this means that the aggregate rate at which susceptible individuals become infected is given by $\beta I(t)S(t)$. This means that the rate of new infection, or *disease incidence*, is proportional to disease prevalence. The basic model compartments with states and flow rates is illustrated in Figure 1.

Last, infected individuals spontaneously recover at rate $\gamma \geq 0$. This means that on aggregate, the rate at which recovery occurs is $\gamma I(t)$. Throughout, I will maintain the following assumption:

Assumption 1: $\beta > \gamma \geq 0$.

This assumption makes the analysis more interesting and will be explained below. To model the possibility of engaging in social distancing, assume that the individuals can affect the rate of infection by controlling the rate at which they expose themselves to infection. In particular, at each instant $t \geq 0$, each individual $i \in \mathcal{S}(t)$ non-cooperatively chooses exposure level $\varepsilon_i(t) \in [0,1]$, at personal cost $(1-\varepsilon_i(t))c \geq 0$. Effectively, this reduces the rate of infection for the individual to $\varepsilon_i(t)\beta I(t)$. This formalization captures the notion that, ceteris paribus, exposure is desirable. Equivalently, this means that engaging in social distancing is costly to the individual. In this analysis, infected and recovered (and therefore immune) individuals have no private benefits from social distancing and are assumed to not engage in any preventive efforts.

To complete the economic model, assume that the individuals in the susceptible, infected and recovered classes earn flow payoffs $\pi_{\mathcal{S}}$, $\pi_{\mathcal{I}}$ and $\pi_{\mathcal{R}}$ respectively and discount the future at rate $\rho > 0$. It will be assumed that

$$\pi_{\mathcal{S}} \geq \pi_{\mathcal{R}} \geq \pi_{\mathcal{I}}$$

In contrast to most of the literature on controlled epidemics, I allow for the possibility that $\pi_{\mathcal{S}} > \pi_{\mathcal{R}}$. This case captures the possibility of after-effects, i.e. that although an individual recovers from the disease, it may have negative long-term consequences on health and well-being to have been infected.

In what follows, I will impose the following restriction:

Assumption 2: $c < \frac{\beta}{(\rho+\gamma)(\rho+\beta)}\left[(\rho+\gamma)\,\pi_{\mathcal{S}} - \pi_{\mathcal{I}} + (\gamma/\rho)\pi_{\mathcal{R}}\right]$.

This assumption ensures that social distancing is state dependent in equilibrium.

2.1. The Epidemiological Benchmark. In this subsection, the classical SIR model will briefly reviewed. This is to help build intuition for the equilibrium analysis and to better contrast the equilibrium dynamics with those in the uncontrolled biological model.

The dynamics of the epidemic is described by the following system of differential equations:

$$\begin{aligned}
\dot{S}(t) &= -\beta I(t)S(t) && (1)\\
\dot{I}(t) &= I(t)\left[\beta S(t) - \gamma\right] && (2)\\
\dot{R}(t) &= \gamma I(t) && (3)\\
S(t) &= 1 - I(t) - R(t) && (4)\\
S(0) &= S_0 > \gamma/\beta, \quad I(0) = I_0, \quad S_0 + I_0 = 1 && (5)
\end{aligned}$$

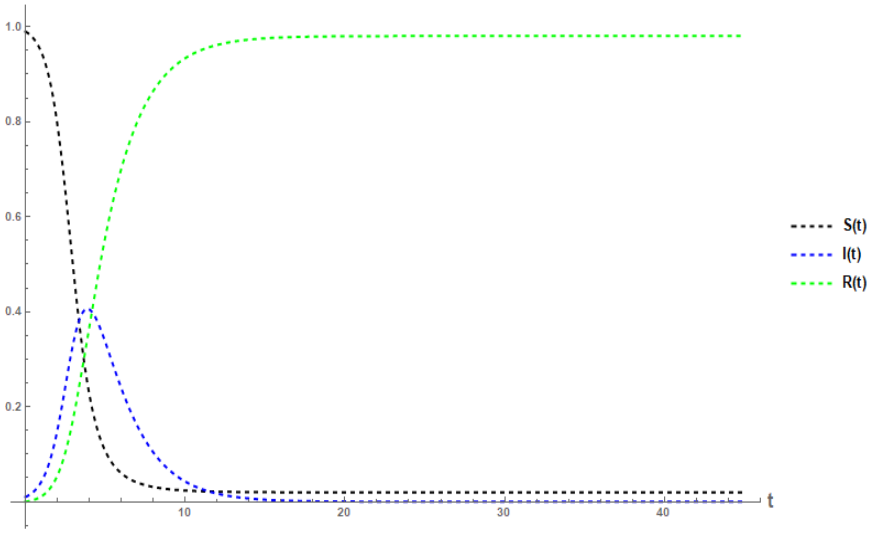

Figure 2: Dynamics of susceptible, infected and recovered in classical SIR model.

It follows from the equations that $\dot{S}(t) \leq 0$ and $\dot{R}(t) \geq 0$, but it turns out that the evolution of disease prevalence $I(t)$ is non-monotonic. The restriction that $S_0 > \gamma/\beta$ ensures that the epidemic can take hold in the population. With this assumption in place, the overall behavior of the system can be described as follows. The measure of susceptible individuals $S(t)$ decreases over time while the measure of recovered individuals increases over time. In contrast, the measure of infected individuals initially increases, peaks when $S(t) = \gamma/\beta$ and then tends to zero. The basic evolution of the uncontrolled, non-behavioral SIR epidemic is illustrated in Figure 2.

Let $\overline{I}$ denote the *peak prevalence* of the epidemic. The level $\overline{I}$ is the highest possible disease prevalence when there is no social distancing whatsoever. Peak prevalence for the SIR epidemic is

$$\overline{I} \equiv S_0 + I_0 - \frac{\gamma}{\beta} + \frac{\gamma}{\beta} \log\left(\frac{\gamma}{\beta S_0}\right) \tag{6}$$

The SIR model cannot be fully characterized analytically. Nevertheless, the limiting distribution of health states can be characterized, which shall prove useful in the analysis of the economic model below. Well-known steps lead to the central result in epidemiology that the final epidemic size is characterized by the equations[5]

$$S(\infty) = 1 - R(\infty) = S(0) \exp\left(-R(\infty)\mathcal{R}_0\right) \geq 0 \tag{7}$$

where $\mathcal{R}_0 \equiv \beta/\gamma$ is the *basic rate of reproduction*.

The basic rate of reproduction represents how many secondary infections are caused

[5]See Brauer and Castillo-Chavez (2012).

by the insertion of a single infected individual into a fully susceptible population. The second equation in (7) defines $R(\infty)$ implicitly and the first equation defines $S(\infty)$ as the residual, which is possible since $I(\infty) = 0$. The limiting proportions $S(\infty)$ and $R(\infty)$ are easily found for particular parameterizations of the model. As is to be expected, cumulative incidence $R(\infty)$ is an increasing function of the infectivity parameter β and a decreasing function of the rate of spontaneous recovery γ.

There are two important insights that follow from equation (7). First, in the limit the disease must die out and no infected individuals remain. Second, and more importantly, when the disease dies out, there is generically a positive measure of susceptibles remaining in the population. This shows that what causes the disease to die out is not that there is eventually a lack of susceptibles that can be infected. Rather, it dies out because the measure of recovered individuals, which must grow over time, becomes so large that the contact between infected and susceptible individuals becomes too rare for the infection to be passed on. Infected individuals have increasingly long sequences of matches with recovered individuals (or between themselves) and so, on expectation, will recover before having the opportunity to pass on the infection to a susceptible individual. Thus with increasing frequency, the chains of infection are broken. The remaining susceptible individuals are said to be protected by *herd* (or *population*) immunity as they benefit from the protection that the recovered individuals give.

The basic rate of reproduction plays a central role here. If $\mathcal{R}_0 < 1$, then infection cannot take hold in the population. If $\mathcal{R}_0 > 1$, then infection first flares up and then tapers off. In the characterization of equilibrium social distancing, the basic rate of reproduction will play a prominent role as well, not as an aim in itself, but as a feature of the equilibrium dynamics.

The economic version of the model inherits a number of simplifying assumptions from the classical model. First, there is only one disease and one level (or severity) of infection. In particular, this rules out the possibility of superinfection by different strains of the disease. Second, the *incubation period* has zero length. This means that the moment that an individual is infected coincides with the onset of symptoms, so no infected individual acts under the mistaken belief that he or she is susceptible. Last, once an individual becomes infected, he or she immediately becomes infectious to other individuals (i.e. the *latency period* has zero length). Relaxing any of these assumptions constitutes possible extensions of the present work.

Last, the analysis is based on the implicitly assumption that only susceptibles ever engage in social distancing, as strictly self-interested infected or recovered individuals face no risks from social interactions.

3. EQUILIBRIUM SOCIAL DISTANCING

In making a decision on how much preventive effort to engage in, the individual must trade off the net benefits of remaining susceptible (through costly prevention) and the net benefits of exposure (with its inherent risks of becoming infected). But since the transition from infected to recovered is beyond the influence of the individual, he or she may treat the problem as one with only two (health) states, namely *susceptible* and *non-susceptible*.

Let $\mathcal{S}(t)$ denote the set of susceptibles at time $t \geq 0$. For an individual $i \in \mathcal{S}(t)$, the social distancing decision influences his or her probability of becoming infected. Let $p_i(t) \in [0,1]$ denote that probability at instant $t \geq 0$. The problem to be solved by a susceptible individual is then given by

$$\max_{\varepsilon_i(t)\in[0,1]} \int_0^\infty e^{-\rho t} \left\{(1-p_i(t))[\pi_{\mathcal{S}} - (1-\varepsilon_i(t))c] + p_i(t)V_{\mathcal{I}}\right\} dt \tag{8}$$

$$s.t. \quad \dot{p}_i(t) = \varepsilon_i(t)\beta I(t)(1-p_i(t)), \quad p_i(0) = p_{i0} \tag{9}$$

where the value of transitioning into the infected state can be calculated as[6]

$$V_{\mathcal{I}} = \frac{1}{\rho+\gamma}\left[\pi_{\mathcal{I}} + \gamma\frac{\pi_{\mathcal{R}}}{\rho}\right] \tag{10}$$

The value can be understood as follows. Once infected, the individual experiences flow utility $\pi_{\mathcal{I}}$ until he or she recovers. From then on, the individual earns flow utility $\pi_{\mathcal{R}}$ in perpetuity. The recovery date is governed by a Poisson process with rate γ and cannot be influenced by the individual.[7] The value $V_{\mathcal{I}}$ is simply the expected discounted lifetime utility of an individual in the infected state. Last, observe that

$$\lim_{\gamma\to 0} V_{\mathcal{I}} = \frac{\pi_{\mathcal{I}}}{\rho}, \quad \lim_{\gamma\to\infty} V_{\mathcal{I}} = \frac{\pi_{\mathcal{R}}}{\rho} \tag{11}$$

In steady state, $\dot{p}_i(t) = 0$. Assuming that the agent has positive exposure, this means that in steady state, *either* the individual has become infected at some point in time $t \geq 0$ so $p_i(t) = 1$ (but has recovered since), *or* infection has died out so $I(t) = 0$ before the individual became infected, in which case he or she remains susceptible in perpetuity.

Note that no individual can influence the evolution of disease prevalence and this is thus taken as exogenously given. Thus each individual's problem is solved on the background of the aggregate evolution of the infectious disease. This is in turn described

[6] See the Appendix for the derivation.

[7] For equilibrium models with treatment augmented recovery, see Rowthorn and Toxvaerd (2015) for the SIS case with no immunity and Toxvaerd and Rowthorn (2020) for the SIR case with immunity.

by the following modified logistic growth equation, which is a function of the aggregate social distancing efforts across the population of susceptibles:

$$\dot{I}(t) = I(t)\left[\boldsymbol{\varepsilon}(t)\beta S(t) - \gamma\right], \quad \boldsymbol{\varepsilon}(t) \equiv \int_{i\in\mathcal{S}(t)} I(t)^{-1}\varepsilon_i(t)di \tag{12}$$

Letting $\eta(t)$ denote the current-value costate variable, the individual's current-value Hamiltonian is given by

$$H = p_i(t)\frac{1}{\rho+\gamma}\left[\pi_{\mathcal{I}} + \frac{\gamma}{\rho}\pi_{\mathcal{R}}\right] + (1-p_i(t))[\pi_{\mathcal{S}} - (1-\varepsilon_i(t))c] + \eta(t)\varepsilon_i(t)\beta I(t)(1-p_i(t)) \tag{13}$$

The optimality condition, supposing that $p_i(t) < 1$, is given by

$$\frac{\partial H}{\partial \varepsilon_i(t)} = \eta(t)\beta I(t) + c = 0 \tag{14}$$

Thus the privately optimal policy for an individual (i.e. his or her best response function) is given by

$$\varepsilon_i(t) = \begin{cases} 0 & for \quad -\eta(t)\beta I(t) > c \\ \varepsilon & for \quad -\eta(t)\beta I(t) = c \\ 1 & for \quad -\eta(t)\beta I(t) < c \end{cases} \tag{15}$$

for any constant $\varepsilon \in [0,1]$. At first blush, this may seem like a bang-bang type solution but as will become clear, in equilibrium the solution will have a *bang-singular-bang* nature. This means that for some intervals of time (or equivalently, for some levels of disease prevalence), the best response of individuals will be bang-bang and switch between $\varepsilon_i^*(t) = 0$ and $\varepsilon_i^*(t) = 1$. But during a phase around the peak of the epidemic, the best responses will be a singular solution determined by the aggregate measure of susceptibles remaining in the population.

The evolution of the current-value multiplier is given by

$$\dot{\eta}(t) = \rho\eta(t) - \frac{\partial H}{\partial p_i(t)} \tag{16}$$

$$= \eta(t)[\rho + \varepsilon_i(t)\beta I(t)] + [\pi_{\mathcal{S}} - \frac{\pi_{\mathcal{I}} + (\gamma/\rho)\pi_{\mathcal{R}}}{\rho+\gamma} - (1-\varepsilon_i(t))c] \tag{17}$$

Using the indifference condition (14) with the equation $\dot{\eta}(t) = 0$ to eliminate $\eta(t)$ yields the critical threshold of disease prevalence

$$I^* \equiv \frac{\rho c}{\beta\left(\pi_{\mathcal{S}} - \frac{\pi_{\mathcal{I}} + (\gamma/\rho)\pi_{\mathcal{R}}}{\rho+\gamma} - c\right)} \tag{18}$$

	β	γ	c	ρ	$\pi_{\mathcal{S}}$	$\pi_{\mathcal{I}}$	$\pi_{\mathcal{R}}$
I^*	$-$	$+$	$+$	$+/-$	$-$	$+$	$+$
$\overline{I}$	$+$	$-$	0	0	0	0	0

Table 1: Comparative statics of peak prevalence levels.

Under Assumption 2, $I^* \in (0,1)$.[8] Now the optimal strategy of a susceptible individual can be expressed in terms of disease prevalence as follows:

$$\varepsilon_i(t) = \begin{cases} 0 & for \quad I(t) > I^* \\ \varepsilon & for \quad I(t) = I^* \\ 1 & for \quad I(t) < I^* \end{cases} \tag{19}$$

for any constant $\varepsilon \in [0,1]$.

The comparative statics of the threshold value I^* and peak prevalence $\overline{I}$ are listed in Table 1.

It is noteworthy that peak prevalence $\overline{I}$ for the purely biological model is increasing in the infectiousness of the disease β, while the maximum equilibrium prevalence I^* is in fact decreasing in β. Similarly, peak prevalence is decreasing in the recovery rate γ in the biological model but increasing in γ in the economic model. Thus the economic and the biological models offer sharply different predictions about how the characteristics of the disease will influence the course of the epidemic. That increased infectiousness will decrease individuals' incentives to self-protection is a feature also seen in the work of Philipson and Posner (1993), Geoffard and Philipson (1996), Fenichel (2013) and Toxvaerd (2019). It can be understood as follows.

Controlling for behavior, i.e. *holding behavior fixed*, higher infectiousness necessarily leads to more infected individuals. This is intuitive and in fact what the classical epidemiological model predicts. But in the behavioral model, behavior is *not fixed* but endogenously determined and changes as the environment changes. This is because the society in the economic model is populated by utility maximising individuals who each weigh the costs and benefits of social distancing. For these individuals, as the infectiousness increases, social distancing becomes more attractive because exposure now leads to a higher probability of becoming infected. As a result, individuals respond to increased infectiousness by scaling back exposure and socially distancing themselves. On aggregate, this behavioral response acts to curb disease incidence and hence decrease peak prevalence.

[8] Assumption 2 ensures that $I^* < 1$. The assumption also implies the weaker condition $(\rho+\gamma)\,c < [(\rho+\gamma)\,\pi_{\mathcal{S}} - \pi_{\mathcal{I}} + (\gamma/\rho)\pi_{\mathcal{R}}]$, which ensures that $I^* > 0$.

3.1. No Social Distancing Scenario. Next, I turn to the characterization of the equilibrium social distancing choices and the concomitant behavior of the dynamics of the epidemic. There are two cases to consider, namely $I^* < \overline{I}$ and $I^* \geq \overline{I}$ and each scenario will be characterized in turn. When $I^* \geq \overline{I}$, the disease is not very serious as seen from the perspective of individuals themselves and thus they never engage in any social distancing. That is, disease prevalence in the uncontrolled biological model never reaches levels that prompt individuals to engage in preventive effort. The equilibrium path of disease prevalence therefore exactly coincides with that in the classical SIR model, with infection peaking at $\overline{I}$. This is not a trivial case, since it highlights an important feature of continual prevention. Namely, in this type of equilibrium, it is quite possible that a very large proportion will become infected at some point along the way, and that all individuals know this. The key reason for there not being an incentive to engage in social distancing is that the intertemporal distribution of infections is sufficiently spread out, i.e. the infection curve is already sufficiently flat, such that at no given moment is the probability of infection sufficiently high to merit costly prevention. In a nutshell, what matters for prevention is the *intensity* of the epidemic rather than the *duration* of the epidemic.

3.2. Social Distancing Scenario. In the case where $I^* < \overline{I}$, equilibrium becomes more complicated. Denote by $S^*(t)$, $I^*(t)$ and $R^*(t)$ the paths of susceptible, infected and recovered individuals under equilibrium social distancing and define the following threshold values:

$$\underline{t} \equiv \min\{t \geq 0 : I(t) = I^*\} \tag{20}$$

$$\overline{t} \equiv \min\{t \geq 0 : S^*(t) = \gamma/\beta\} \tag{21}$$

Disease prevalence will be defined as *naturally decreasing* if disease incidence is negative in the absence of social distancing. In other words, disease prevalence is naturally decreasing when even with no preventive behaviour, there are sufficiently few remaining susceptibles to ensure that the number of infected individuals declines. Then, the uncontrolled epidemic becomes naturally decreasing at time $\widetilde{t}$, defined implicitly by

$$\widetilde{t} \equiv \min\{t \geq 0 : S(t) = \gamma/\beta\}$$

Because social distancing induces (weakly) lower disease incidence for all t, it has to be that $I^*(t) \leq I(t)$. But this implies that

$$S^*(\widetilde{t}) > S(\widetilde{t}) = \gamma/\beta$$

In other words, at the point in time at which disease prevalence on the uncontrolled path starts decreasing, the equilibrium disease incidence would be positive (i.e. disease prevalence would *increase*) were the individuals to cease social distancing. This means that in a sense, in equilibrium social distancing prolongs the duration of the epidemic. To use a phrase much discussed in recent policy debates, in equilibrium the individuals will act to *"flatten the curve"* out of an uncoordinated desire for self-preservation.

Equilibrium behavior can now be characterized as follows:

Proposition: If $I^* < \overline{I}$, then in a symmetric equilibrium, exposure at time $t \geq 0$ for each individual $i \in \mathcal{S}(t)$ is given by

$$\varepsilon_i^*(t) = \begin{cases} \frac{\gamma}{\beta S(t)} & for \quad t \in (\underline{t}, \overline{t}) \\ 1 & for \quad t \notin (\underline{t}, \overline{t}) \end{cases} \tag{22}$$

Proof: From the best response function of the individuals, it follows that if $I(t) > I^*$, then all individuals will engage in full social distancing, thereby bringing down disease incidence. Similarly, if $I(t) < I^*$, then all individuals will fully expose themselves to infection, thereby increasing disease incidence. There are two cases to consider. If disease prevalence is naturally decreasing, then full exposure will continue to be optimal indefinitely. If disease prevalence is not naturally decreasing, then it will move towards the level I^*. Therefore, in equilibrium, disease prevalence must remain constant until it becomes naturally decreasing. Setting $\dot{I}(t) = 0$ yields the required aggregate exposure level (and thus the individual mixing probabilities) as

$$\boldsymbol{\varepsilon}^*(t) = \frac{\gamma}{\beta S(t)} \tag{23}$$

and the result follows ■

The Proposition shows that in equilibrium, individuals engage in no social distancing until a sufficiently large proportion of the population has become infected. Once it has taken sufficient hold, they switch to a mixed strategy equilibrium in which they attach increasingly high probability to no social distancing. The probability increases as the measure of susceptibles decreases. One can view the strategy of individuals as akin to a thermostat that switches off and on as the temperature is above or below a desired level. An immediate result of the Proposition is as follows:

Corollary: During the equilibrium social distancing phase, individuals gradually reduce their social distancing efforts despite the infection probability not decreasing.

This result is noteworthy because it shows that in equilibrium, during the social distancing phase it is the measure of remaining susceptibles that determines the level of

social distancing, *not the number of infected individuals*. In fact, during this plateau phase, disease incidence stays constant at the critical threshold I^* and equilibrium social distancing effort decreases as the measure of susceptibles decreases. During the social distancing phase, i.e. when the best responses are on the singular segment, efforts to decrease exposure to infection are strategic substitutes in that any individual would respond to more social distancing by others with an increase in exposure. The mixed strategy-singular solution nature of equilibrium during the social distancing phase is similar in nature to the steady state equilibrium behavior in Toxvaerd (2019). In that paper, the disease is of the susceptible-infected-susceptible variety and thus individuals cannot obtain immunity. As a consequence, in steady state $S(t)$ remains constant through time and thus the mixed strategy weights in the singular solution are constant. In contrast, with immunity, the measure of susceptibles must decrease over time, explaining why the equilibrium mixing probabilities must change as time progresses and the state of the epidemic changes.

The the equilibrium path for disease prevalence and the associated equilibrium social distancing efforts are illustrated in Figure 3, which also displays the path of disease prevalence in the uncontrolled biological model for comparison. As can be seen in the figure, at the early stages of the epidemic, individuals choose not to make any social distancing efforts (i.e. they choose to fully expose themselves). This reflects the fact that as disease prevalence is initially very low (and thus the infection risk from exposure commensurately small), individuals do not find social distancing measures worthwhile. Similarly, when the epidemic has run its course and infection has almost died out, individuals will again opt for full exposure. But at the height of the epidemic, during the phase in which the uncontrolled epidemic would have peaked, individuals spontaneously act and engage in social distancing, causing a dampening effect on disease incidence and prevalence.

Corollary: The equilibrium trajectory of the disease during the social distancing phase is characterized by the system of differential equations

$$\dot{S}^*(t) = -\beta\boldsymbol{\varepsilon}^*(t)I^*S^*(t) = -\beta\left(\frac{\gamma}{\beta S^*(t)}\right)I^*S^*(t) = -\gamma I^* \tag{24}$$

$$\dot{I}^*(t) = 0 \tag{25}$$

$$\dot{R}^*(t) = \gamma I^* \tag{26}$$

As an aside, the model also an equilibrium in asymmetric strategies. All that is required on the equilibrium path during the social distancing phase is that on the horizontal segment of the curve, aggregate exposure must equal $\boldsymbol{\varepsilon}^*(t)$; it does not matter how this comes about. Since on this segment the individuals are indifferent between full social distancing and full exposure, they are willing to mix between strategies. But it is also

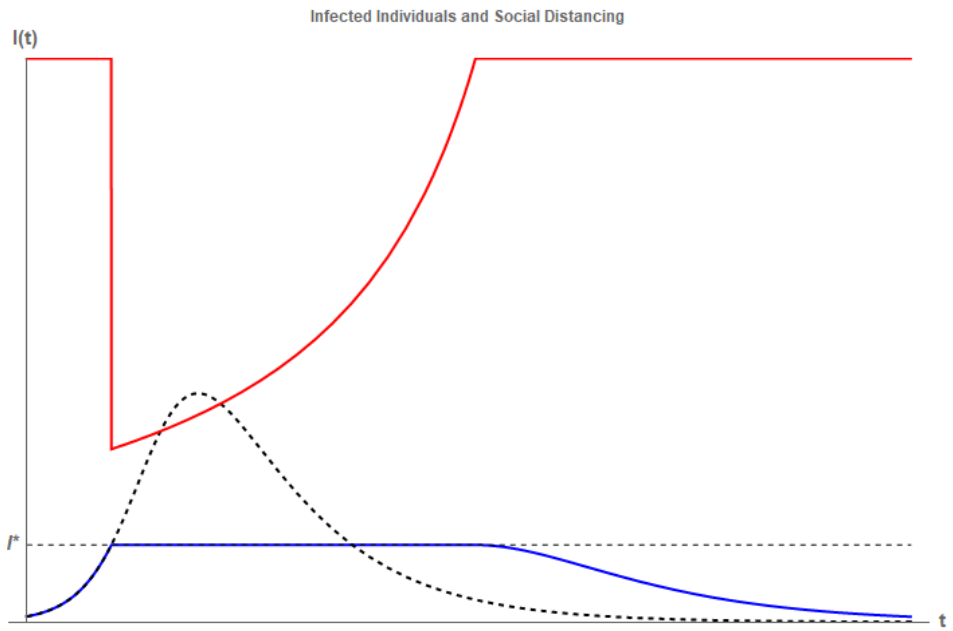

Figure 3: Equilibrium disease prevalence and social distancing across stages of epidemic. The blue curve shows disease prevalence and the red curve shows aggregate exposure.

consistent with equilibrium to have a fraction $\varepsilon^*(t)$ exposing themselves fully and have the remainder $(1 - \varepsilon^*(t))$ fully socially distancing themselves.

For ease of comparison of disease paths between the non-controlled biological model and the equilibrium under social distancing, Figures 4, 5 and 6 show the evolution of individuals in each health state separately, while Figure 9 at the end of the paper shows all the paths superimposed.

The dynamic equations in the Corollary show another interesting feature, namely that during the social distancing phase, the measure of susceptible individuals decreases linearly at a rate $-\gamma I^*$, while the measure of recovered individuals increases linearly at rate γI^*. In addition, one sees that the rates of change are proportional to the critical threshold I^*. In other words, we can relate the speed of change over time during the social distancing phase to the magnitude of the biological and preference parameters. For example, an increase in infectivity β will cause the susceptibles to decrease more sharply and the recovered to increase more sharply. Similarly, the more severe the disease is, as measured by lower flow utility while infected $\pi_{\mathcal{I}}$, will likewise make susceptibles decrease faster and that of the recovered increase faster. The linear segments on the $S^*(t)$ and $R^*(t)$ curves can be verified in Figures 4 and 6.

Next, consider how a change in the preference parameters influences the social distancing decisions in equilibrium and how they alter the trajectory of the disease over time. We will do this in terms of effects on peak prevalence and on the duration of the plateau phase with elevated disease prevalence. To trace the effects of changes in the

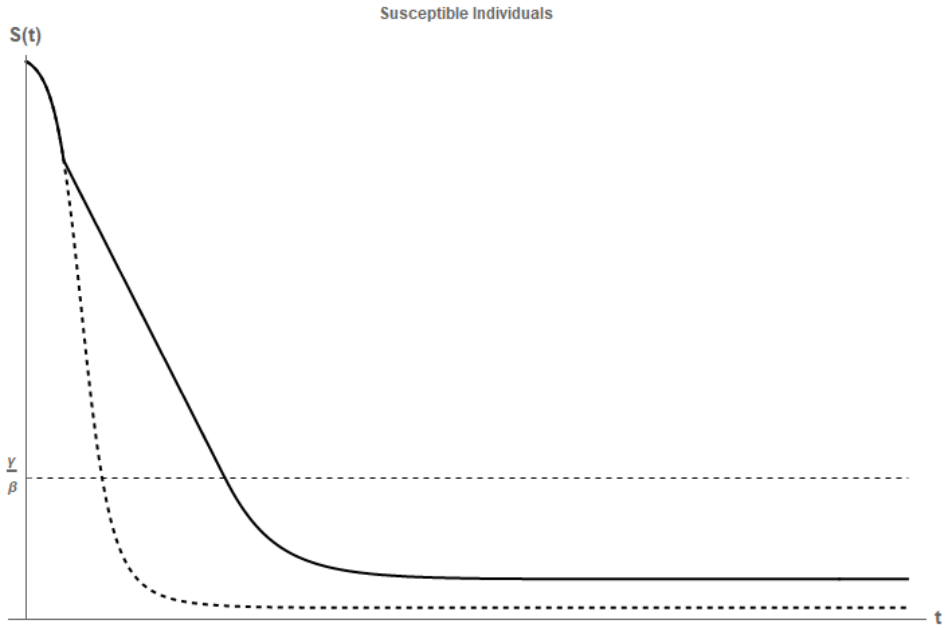

Figure 4: Paths of susceptible individuals across epidemic. Dashed curve shows path in epidemiological model; solid curve shows equilibrium path in economic model.

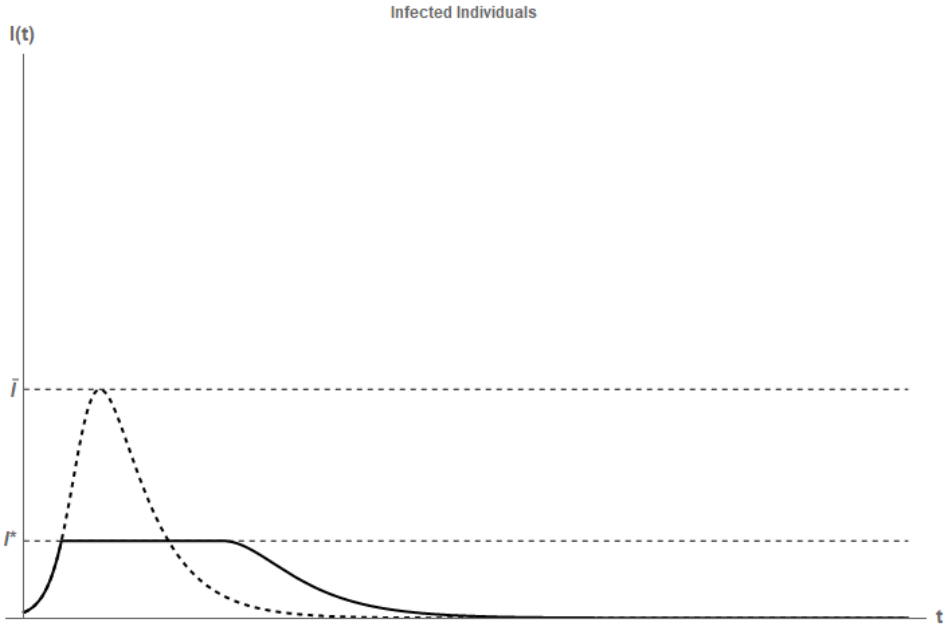

Figure 5: Paths of infected individuals across epidemic. Dashed curve shows path in epidemiological model; solid curve shows equilibrium path in economic model.

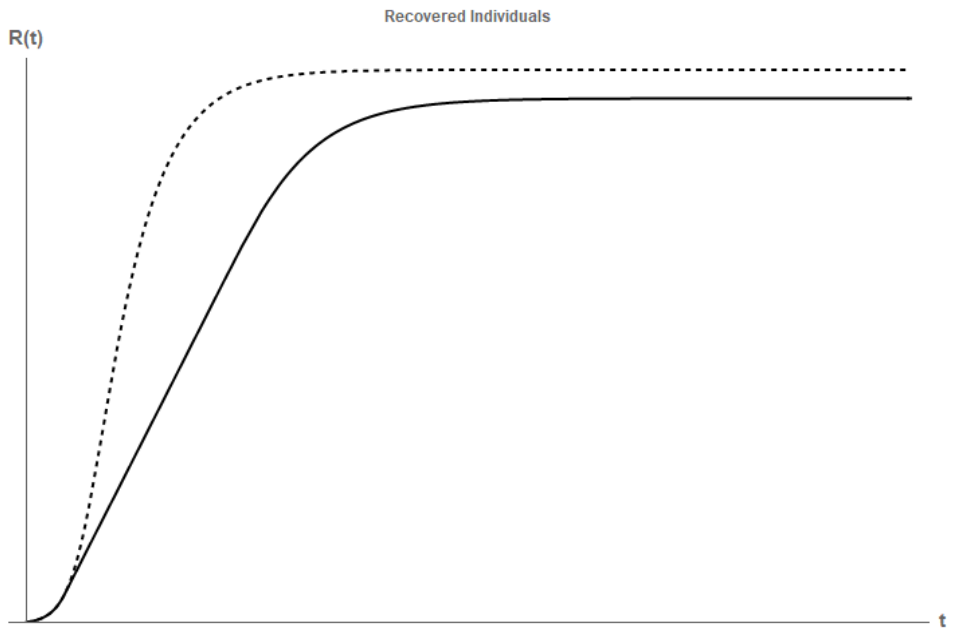

Figure 6: Paths of recovered individuals across epidemic. Dashed curve shows path in epidemiological model; solid curve shows equilibrium path in economic model.

preference parameters, we can simply determine how they influence the critical threshold I^* and then see what effect this has on the aggregate dynamics. Since the method of analysis is the same for each of the parameters, I will perform this exercise only for a change in the flow payoff $\pi_{\mathcal{I}}$ that an individual earns when it is infected. This case is illustrated in Figure 7, which shows the effects on the trajectory of infected people when $\pi_{\mathcal{I}}$ is lowered. This corresponds to making the disease more severe in that it has more dire health consequences. All other parameters are kept fixed. The benchmark case is shown as a solid black curve while the modified case is shown as a solid blue curve. The dashed black curve shows the uncontrolled benchmark for reference. Because the disease is now more severe, individuals have reduced tolerance to infection. This is reflected in a downward shift in the critical threshold I^*. But this means that social distancing kicks in earlier in equilibrium and also serves to extend the duration of the epidemic (in the sense described earlier). As will be explored further below, this also has consequences for the limiting distribution of the epidemic. As will become clear, although the phase of relatively high disease prevalence is thus increased, the actual number of infected individuals across the epidemic (i.e. cumulative incidence) actually decreases.

Analysis of the limiting distribution. Till now, I have focused on the evolution of the epidemic across time, which is useful to analyze inter-dependencies between the compartments, growth rates and time-domain properties of the disease. But the time dimension is less useful for determining the limiting properties of the disease, such as the

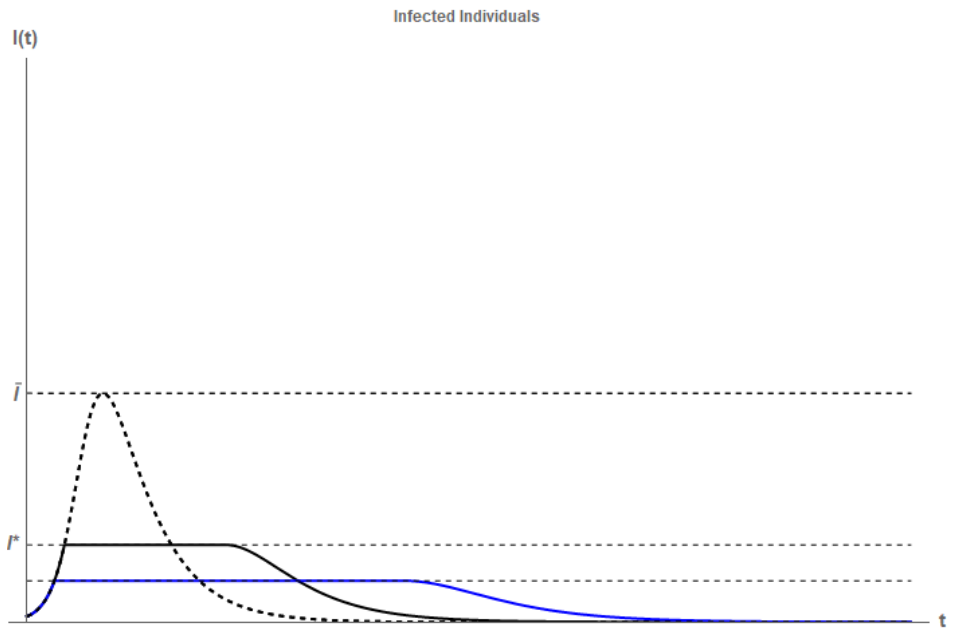

Figure 7: How parameters change the intensity and duration of the epidemic under equilibrium social distancing. Plot shows the evolution of susceptible individuals in the benchmark model in solid black and the evolution when the disease is more severe ($\pi_{\mathcal{I}}$ is lower) in solid blue.

cumulative incidence (i.e. the total number of infected individuals across the epidemic) and how the eventual number of immune and susceptible individuals depend on the initial conditions. For that purpose, and to further compare the equilibrium disease dynamics with social distancing to those under the uncontrolled biological model, it is useful to consider the evolution of infections in the $(S(t), I(t))$-plane. This is done in Figure 8. For an arbitrary point in this diagram, the measure of recovered individuals $R(t)$ is residually determined. For this purpose, note that the dynamics in $(S(t), I(t))$-space are characterized by the equation[9]

$$I(t) = S_0 + I_0 - S(t) + \frac{\gamma}{\beta} \log\left(\frac{S(t)}{S_0}\right) \tag{27}$$

To understand the figure, assume that $R_0 = 0$ and pick an initial point (S_0, I_0). This point is denoted by a on the curve. I will first describe the uncontrolled dynamics in the absence of social distancing and then contrast them with the dynamics in equilibrium. Starting from the initial point a, infection picks up and the susceptible population decreases, moving the state of the system along the dashed curve peaking at point f and ending in some point $(S(\infty), I(\infty))$, denoted by g. There are two important points to

[9] See Hethcote and Waltman (1973), who uses this type of diagram to illustrate the effects of an initial pulse vaccination of a fraction of the susceptible population.

notice.

First, the curve showing the uncontrolled dynamics has its maximum at $S(t) = \gamma/\beta$, *irrespective of the initial condition.* In other words, all the curves describing the epidemic in $(S(t), I(t))$-plane achieve their maximum at the same value of susceptibles. This will turn out to be important for the characterization of the equilibrium dynamics. If one chooses another initial state (S_0', I_0') with $I_0' > I_0$, indicated by e, then the resulting curve is simply shifted upwards and never intersects the initial curve. The dynamics under this higher initial disease prevalence settles on a lower value for $S(\infty)$, as can be verified from the equation that characterizes the final distribution. But note that the shifted curve *also* has its maximum at $S(t) = \gamma/\beta$.

Second, generically, the curves intersect the $S(t)$-axis at a point $(S(\infty), I(\infty))$ at which $I(\infty) = 0$ and $S(\infty) > 0$. In other words, the disease dies out asymptotically, some individuals remain susceptible and $R(\infty) = 1 - S(\infty)$ become infected at some point during the epidemic but eventually recover. The limiting fraction $R(\infty)$ measures the aggregate incidence (or total case count) of the epidemic. Next, consider the movement along the $I(t)$-dimension. As is clear from the curve, infection initially increases to the point when $S(t) = \gamma/\beta$ and then decreases.

It is important to emphasize that the speeds along these different uncontrolled disease curves differ and depend on the initial conditions and the parameters β and γ (see Hethcote and Waltman, 1973 for details). As will be shown below, the speed of movement in equilibrium is lower than that in the biological model, as social distancing serves to reduce the speed by suppressing disease incidence.

Next, I turn to the dynamics under endogenous social distancing. In Figure 8, I plot the horizontal line corresponding to the equilibrium cutoff $I^* < \bar{I}$. In equilibrium, the initial dynamics from the point a coincide with those of the biological model until the point b, where $I(t) = I^*$. At that point, the equilibrium and uncontrolled biological paths diverge as the individuals start to socially distance themselves. They do so to an extent that keeps $I(t)$ constant at the critical level. Thus in equilibrium after the initial stage, the dynamics move horizontally leftward till the point c, at which no further social distancing is desired by the individuals. As described above, this happens when the infection becomes naturally decreasing. But on the diagram, one readily verifies this must happen at a point where $S^*(t) = \gamma/\beta$. Thus once point c is reached, individuals cease social distancing and thus the dynamics going forward coincide with those of the uncontrolled biological model but with the modified initial condition $(S_0, I_0) = (\gamma/\beta, I^*)$. The equilibrium path thus ends at point d, which can be confirmed to lie strictly to the right of point g.

In both the uncontrolled biological model and in the model with social distancing,

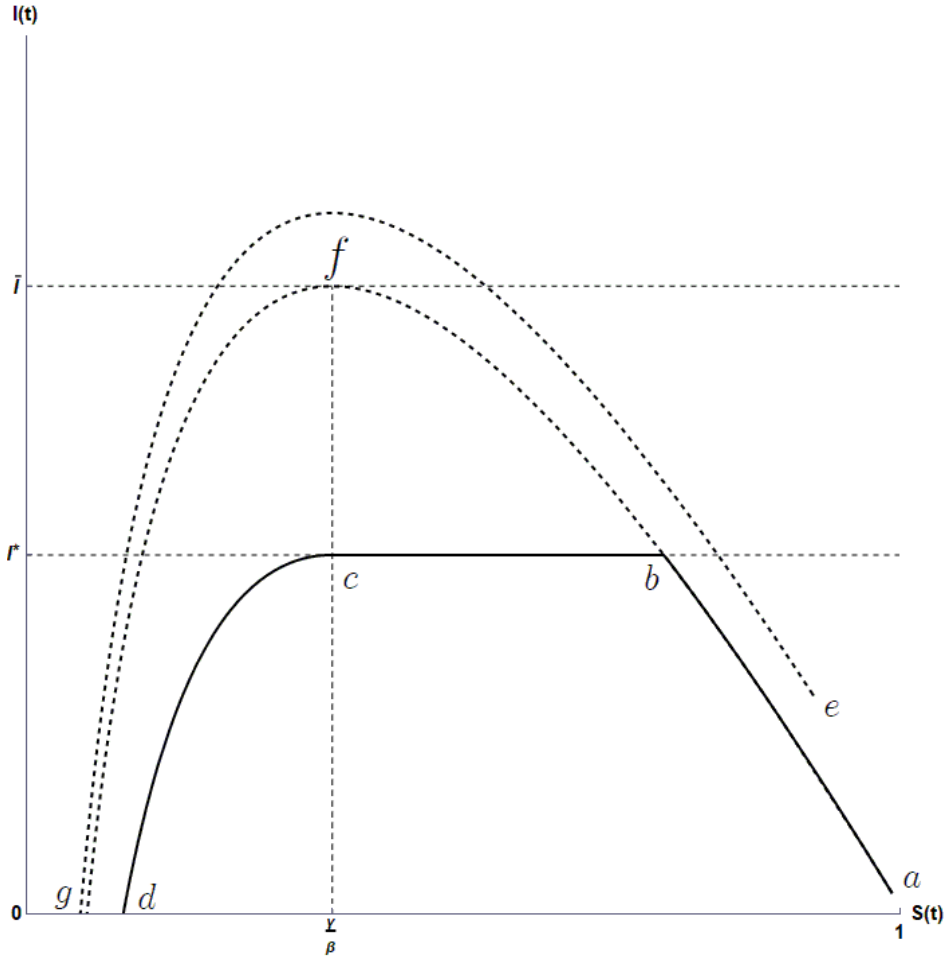

Figure 8: Disease dynamics in the $(S(t), I(t))$-plane. Dashed curve shows path in epidemiological model; solid curve shows equilibrium path in economic model.

the infection dies out only asymptotically. Thus the end of the disease cannot be said to occur faster under social distancing than it otherwise would have under a purely non-behavioral model. What is possible though is to determine when the disease starts decreasing naturally under the two scenarios. In the diagram, it is clear that this happens when the fraction of remaining susceptibles reaches the critical threshold γ/β. But note that along the uncontrolled biological trajectory, the speed of movement along the curve in the $(S(t), I(t))$-plane is

$$\begin{aligned} \dot{S}(t) &= -\beta I(t) S(t) && (28) \\ &= -\beta \left[S_0 + I_0 - S(t) + \frac{\gamma}{\beta} \log\left(\frac{S(t)}{S_0}\right) \right] S(t) && (29) \end{aligned}$$

In contrast, during the social distancing phase, the speed of movement is

$$\dot{S}(t) = -\beta I^* S(t) \tag{30}$$

But since $I^*(t) \leq I^*$ for all $t \geq 0$, it follows that the starting point of declining infection happens with a delay under social distancing. Another way to see that infection is suppressed under social distancing is to recall that in the uncontrolled biological model, the churn rate is

$$\frac{\beta S(t)}{\gamma} \tag{31}$$

In other words, each individual who recovers is replaced by $\beta S(s)/\gamma$ new infected individuals. The dependence of this conversion ratio on the fraction of susceptibles $S(t)$ is exactly what causes infection to first increase and then decrease. In contrast, under social distancing, the churn rate is

$$\frac{\boldsymbol{\varepsilon}^*(t)\beta S(t)}{\gamma} = \frac{\frac{\gamma}{\beta S(t)}\beta S(t)}{\gamma} = 1 \tag{32}$$

In other words, equilibrium exposure is set such that each individual who recovers is replaced by exactly one new individual who is infected.

The suppression of incidence in equilibrium also has effects on the progression of susceptible and recovered individuals. Once social distancing kicks in, as fewer people become infected, the measure of susceptibles declines less steeply. At the same time, the measure of recovered grows less rapidly.

Note that since the curves in the $(S(t), I(t))$-plane do not intersect, we can use the intersection between the lines $I(t) = I^*$ and $S(t) = \gamma/\beta$ to determine the effects of changes in parameters on the final size distribution. E.g., for two distinct such intersection points (which differ because we vary one of the underlying parameters), we can rank the resulting

	β	γ	c	ρ	$\pi_{\mathcal{S}}$	$\pi_{\mathcal{I}}$	$\pi_{\mathcal{R}}$
$S^*(\infty)$	$+$	$-$	$-$	$+/-$	$+$	$-$	$-$
$R^*(\infty)$	$-$	$+$	$+$	$-/+$	$-$	$+$	$+$
$S(\infty)$	$-$	$+$	0	0	0	0	0
$R(\infty)$	$+$	$-$	0	0	0	0	0

Table 2: Comparative statics of final size distributions.

limiting distributions by following the curves from the initial intersection points to the $S(t)$-axis to find the corresponding $S(\infty)$-values.

Using the comparative statics of the critical threshold I^* in Table 1, we find the corresponding effects that the biological and preference parameters have on the final size distribution in the uncontrolled biological model and in the equilibrium in the economic model. These are given in Table 2.

These comparative statics again show that the results from the equilibrium model may reverse those of the uncontrolled biological model. For example, increasing infectiousness β or decreasing the recovery rate γ leads to higher cumulative incidence in the biological model but to lower cumulative incidence in the economic model. The comparative statics with respect to the preference parameters have no biological counterpart and so for these, no comparison is possible.

4. Discussion

This paper has considered the equilibrium amount of social distancing in the context of the well-known SIR epidemiological model. While simple, this model allows for an intuitive and clean analysis of the tradeoffs involved in individuals' decision-making on social distancing. There are several ways in which the analysis can be enriched. First, rather than consider a linear cost of social distancing, other cost structures can be considered. The main insights are robust to this extension. With increasing convex costs of social distancing, individuals would continually adjust to increasing disease prevalence in the population.[10] Second, the paper has not offered a full welfare analysis of the equilibrium. It is immediately clear that the equilibrium is in fact not social welfare maximizing. The reasons is a classical one in this type of model, namely that the individuals in the population do not internalize the positive externalities flowing from social distancing. Third, and most interestingly, the analysis has been based on the assumption of a well-mixed population in which all that matters are the fractions of susceptible, infected and recovered. A richer model would consider a population with explicit social structure. This would open up for the possibility that the incentives to socially distance oneself may

[10]Note also that although costs (and thus the current-value Hamiltonian) is linear, the symmetric equilibrium is characterised by a singular solution during the social distancing phase. Thus exposure levels in fact vary continuously with the state during this phase.

depend on one's position in the social network. Such an analysis may also be useful in informing policy, such as the socially optimal design and micro-targeting of quarantines.

Last, the population in this model has been assumed to be homogeneous. With a population that is heterogeneous along some dimension, the qualitative nature of the analysis would be similar, but aggregate social distancing would change more continuously, creating a gradual increase and subsequent decrease in social distancing. To see this, suppose that individuals were heterogeneous in how much they suffered from infection. In that case, different individuals would have different tolerances to infection risk and this would mean that each individual would start socially distance itself at different levels of disease prevalence. Initially, only the very risk intolerant in the population would start socially distancing but as prevalence increases further, additional individuals would join them. In this manner, aggregate social distancing would be phased in more smoothly than in the homogeneous population case, in which all switch to social distancing at the same time. The heterogeneous population case also suggests the interesting possibility of free-riding by the more risk tolerant on the efforts of the less risk tolerant. As the latter start socially distancing themselves, disease incidence is curbed somewhat, thus protecting those individuals who have not yet reached their individual social distancing thresholds. In fact, disease prevalence may be curbed so much by the initial social distancing efforts of the risk intolerant that the most tolerant may never have to engage in any social distancing and they would in effect be free-riding on the preventive efforts of those who do.

A. APPENDIX

In this Appendix, I derive the value of transitioning into the infected state. Let the recovery date for an infected individual be denoted by T. This date arrives according to a Poisson process with rate $\gamma \geq 0$.

The value we seek to characterize, namely the net present value of being infected at instant $t \geq 0$ is

$$V_{\mathcal{I}} = \int_t^T e^{-\rho u} \pi_{\mathcal{I}} du + \int_T^\infty e^{-\rho u} \pi_{\mathcal{R}} du \tag{33}$$

Consider a utility flow π which can take two values, $\pi_{\mathcal{I}}$ and $\pi_{\mathcal{R}}$. At time t, the utility flow starts off with $\pi = \pi_{\mathcal{I}}$. Over the time interval $[t, t+dt)$, the probability of the utility flow switching to $\pi_{\mathcal{R}}$ is γdt and so the probability of the utility flow not switching $\pi_{\mathcal{R}}$ is $1 - \gamma dt$. Define

$$V_{\mathcal{I}}^t = E_t \int_t^\infty e^{-\rho(u-t)} s_u du \tag{34}$$

and assume that $s_t = \pi_{\mathcal{I}}$. We do so because for the case where $s_t = \pi_{\mathcal{R}}$, we know that the utility flow gets stuck at $\pi_{\mathcal{R}}$ and so

$$E_t \int_t^\infty e^{-\rho(u-t)} \pi_{\mathcal{R}} du = \pi_{\mathcal{R}} E_t \int_t^\infty e^{-\rho(u-t)} du = \frac{\pi_{\mathcal{R}}}{\rho} \tag{35}$$

Observe also that $V_{\mathcal{I}}^t$ is independent of t, by virtue of the infinite horizon and so $V_{\mathcal{I}}^t = V_{\mathcal{I}}$. Therefore

$$V_{\mathcal{I}} = \pi_{\mathcal{I}} dt + (1 - \gamma dt) e^{-\rho dt} V_{\mathcal{I}} + \gamma dt e^{-\rho dt} \frac{\pi_{\mathcal{R}}}{\rho} \tag{36}$$

$$V_{\mathcal{I}} = \pi_{\mathcal{I}} dt + [1 - (\rho + \gamma) dt] V_{\mathcal{I}} + \gamma dt \frac{\pi_{\mathcal{R}}}{\rho} + o(dt) \tag{37}$$

$$0 = \pi_{\mathcal{I}} dt - (\rho + \gamma) V_{\mathcal{I}} dt + \gamma dt \frac{\pi_{\mathcal{R}}}{\rho} + o(dt) V_{\mathcal{I}} dt \qquad = \frac{\pi_{\mathcal{I}} + \gamma \frac{\pi_{\mathcal{R}}}{\rho}}{\rho + \gamma} dt + o(dt). \tag{38}$$

In the continuous-time limit, we obtain

$$V_{\mathcal{I}} = \frac{1}{\rho + \gamma} \left[\pi_{\mathcal{I}} + \gamma \frac{\pi_{\mathcal{R}}}{\rho} \right] \tag{39}$$

and the result follows ■

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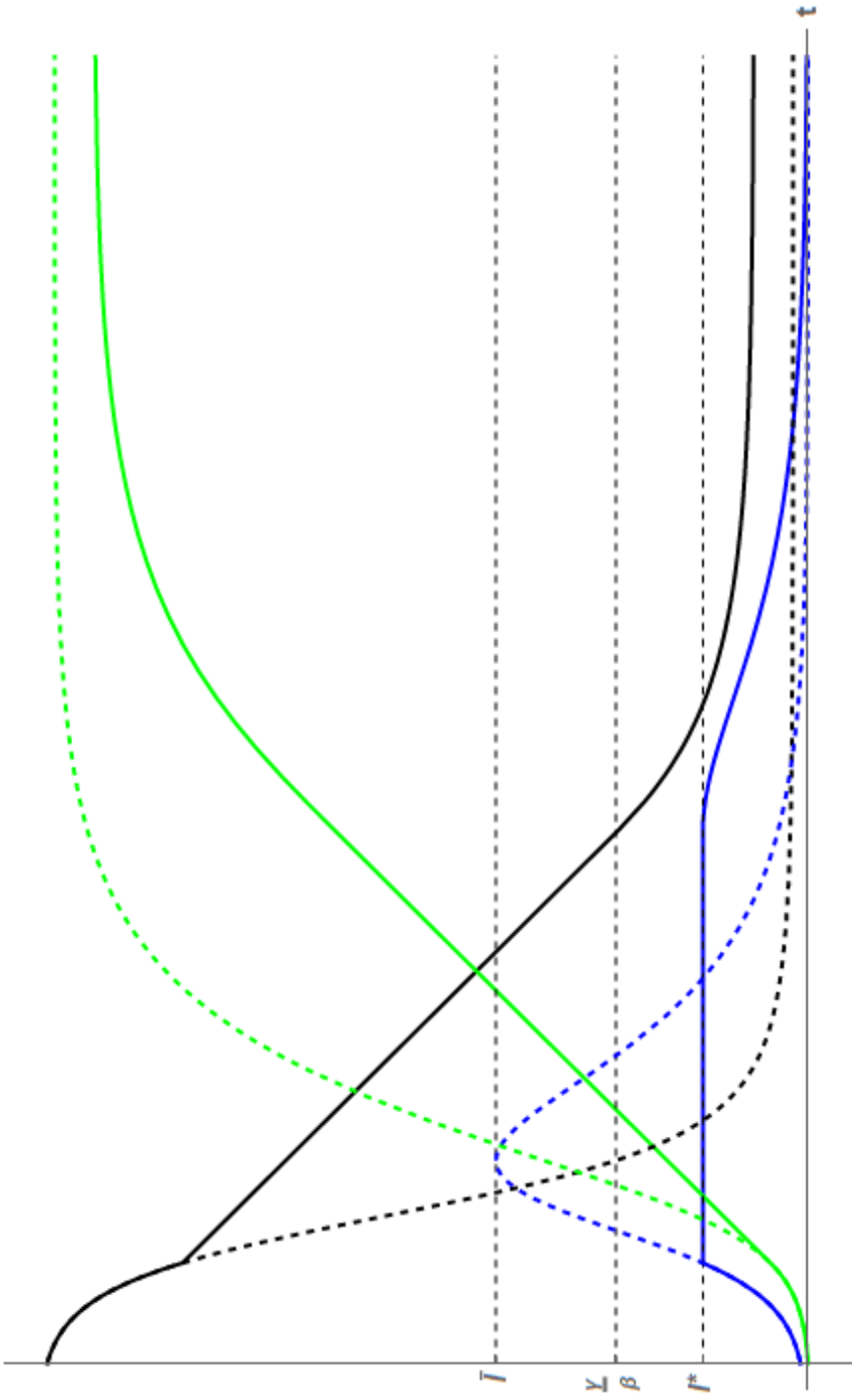

Figure 9: Paths of susceptible, infected and recovered individuals across epidemic. Dashed curves shows paths in epidemiological model; solid curves show equilibrium paths in economic model.