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Equilibrium social activity during an epidemic ☆

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Abstract

During an infectious-disease epidemic, people make choices that impact transmission, trading off the risk of infection with the social-economic benefits of activity. We investigate how the qualitative features of an epidemic's Nash-equilibrium trajectory depend on the nature of the economic benefits that people get from activity. If economic benefits do not depend on how many others are active, as usually modeled, then there is a unique equilibrium trajectory, the epidemic eventually reaches a steady state, and agents born into the steady state have zero expected lifetime welfare. On the other hand, if the benefit of activity increases as others are more active ("social benefits") and the disease is sufficiently severe, then there are always multiple equilibrium trajectories, including some that never settle into a steady state and that welfare dominate any given steady-state equilibrium. Within this framework, we analyze the equilibrium impact of a policy that modestly reduces the transmission rate. Such a policy has no long-run effect on society-wide welfare absent social benefits, but can raise long-run welfare if there are social benefits and the epidemic never settles into a steady state.

JEL classification: C73; I12; I18

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1. Introduction

In 2005, a team of researchers led by Yale School of Medicine Professor Neel Gandhi descended on a rural hospital in KwaZulu Natal, South Africa to document the prevalence of drug-resistant tuberculosis (Gandhi et al., 2006). Of 542 patients diagnosed with active tuberculosis (TB), 53 had "extensively drug-resistant" (XDR) infections that were resistant to all of the first-line antibiotics typically used to treat TB as well as multiple second-line treatments. Worse yet, this XDR-TB strain was especially virulent: half of those with XDR-TB infection were dead within 16 days of identification, and only one survived for a full year.[1] "Totally-resistant" TB strains that are untreatable with any known antibiotic have been identified in Italy, Iran, India, and elsewhere (Velayati et al., 2013; Khawbung et al., 2021). Fortunately, none of these nightmare pathogens has yet succeeded in launching a global pandemic. But once that does happen, and an untreatable pandemic-potential TB strain arrives in places like Europe and the United States, what will happen next? What course will the epidemic take? And what long-term impact will this novel pathogen have on society-wide welfare, including not just the direct harms due to the disease but also the indirect economic and psycho-social harms associated with efforts to avoid infection?

How a novel infectious disease such as untreatable TB spreads through a human population and how much harm it inflicts on people's health and prosperity depends on people's behavior, which itself changes during the course of the epidemic. This feedback between human behavior and pathogen transmission determines the equilibrium trajectory of the epidemic. The field of economic epidemiology seeks to further our understanding of equilibrium epidemics through models of behavior during an epidemic. Such models can be used to analyze the path of a novel infectious disease (Farboodi et al. (2021), Garibaldi et al. (2020), Keppo et al. (2020), McAdams (2020), Toxvaerd (2020), and references therein), to evaluate policy options for managing an unfolding epidemic (on optimal lockdown policies, see Acemoglu et al. (2021), Alvarez et al. (2021), Bethune and Korinek (2020), Jones et al. (2021), and Rowthorn and Maciejowski (2020)), and to quantify the social value of new vaccines and treatments (Makris and Toxvaerd (2020)), among many other things—but only if these models adequately capture the underlying economic-epidemiological environment.

Many assumptions about the economic-epidemiological environment are implicit in any economic-epidemic model, including (i) ecological and epidemiological assumptions about the disease process itself and (ii) economic assumptions about agents' information, interactions, and payoffs. McAdams (2021) surveys the recent Covid-inspired literature, categorizing economic-epidemic models based on their assumptions about immune response, manner of transmission, and economic impacts. Avery et al. (2021) provides an insightful discussion of several of these modeling dimensions, focusing especially on how agent heterogeneity can impact the qualitative features of equilibrium outcomes.

[1] Most of those who died were also infected with HIV, but two health-care workers contracted XDR-TB within the hospital and they also died. Multiple XDR-TB strains were present, but 39 of 46 genotyped XDR-TB isolates were genetically related. Gandhi did not know about this strain before he arrived; it was sheer coincidence that he found it.

In this paper, we focus on the impact of an economic assumption that has not yet received much attention. Specifically, consider the social-economic activities that increase the risk of pathogen transmission ("transmissive activities" or simply "activity"). When an agent engages in these activities, does the benefit that they enjoy depend on whether others are also active? In other words, are the activities that drive transmission social in nature (such as working in-person at an office rather than virtually from home) or non-social (such as exercising in a gym). We show that incorporating social motivations into the economic model of an infectious-disease epidemic can have profound equilibrium implications for how the disease will progress and persist over time.

To illustrate the novel aspects of our analysis as clearly as possible, we employ an especially simple epidemiological model, a Susceptible-Infected-Removed (SIR) model with vital dynamics in which infected agents never recover from infection but may be "removed" due to death from the disease, agents also die at a constant rate from other causes, and there is a constant flow of newborn agents susceptible to infection. The special case in which no one dies from the disease is a Susceptible-Infected (SI) model. We develop our main findings first in the SI model, and then extend the analysis to allow for disease-induced death.

In the SI model, we first consider the benchmark case in which the economic benefits of activity do not depend on others' activity choices. For an epidemic that starts from an initial condition with low infection prevalence and sufficiently severe disease, we show:

(i) there is a unique equilibrium epidemic trajectory;
(ii) there is a unique steady-state equilibrium (SSE) and the unique equilibrium trajectory reaches this steady state in finite time; and
(iii) agents born into the SSE have the same expected lifetime welfare as if forbidden from ever engaging in transmissive activity ("zero welfare").

We then show that none of these key qualitative features of the equilibrium set are robust to the possibility that agents' individual benefit from activity may depend on others' activity. In particular, suppose that there are positive economic complementarities, so that agents gain more from being active when others are more active, what we refer to as "social benefits."[2] In that context, we show the following for any sufficiently severe disease:

(i) there are many equilibrium epidemic trajectories;
(ii) there is a unique SSE, which can be reached in finite time along an equilibrium trajectory, but many equilibrium trajectories never converge to a steady state; and
(iii) agents born into the SSE have zero welfare, but there are non-converging equilibrium trajectories in which agents' behavior oscillates over time and all agents have positive welfare at birth.

Interesting differences also arise in terms of equilibrium comparative statics, with policy-relevant implications. For example, consider the long-run impact of a policy that somewhat reduces the transmission rate, such as improving ventilation, providing free masks, or developing an imperfect immunotherapy or vaccine. In the benchmark case without economic complemen-

[2] The case with negative economic complementarities (e.g., congestion) is also of interest, but appears qualitatively similar to the benchmark case. We focus on the case with social benefits to streamline the exposition and highlight the novel aspects of our analysis as clearly as possible.

tarities, such a policy changes how many people are infected in the unique SSE (ironically, *more* people are infected in the new steady state when the disease is sufficiently severe) but newborn agents continue to have zero expected lifetime welfare. The societal benefits of the new policy are therefore transitory in nature, undone by agents' equilibrium behavioral response. By contrast, if there are social benefits to activity and the epidemic never settles into a steady state, then the policy can increase long-run society-wide welfare.

The most novel aspect of our analysis is that non-converging epidemic trajectories can emerge in equilibrium once there are social benefits associated with transmissive activity. In addition to the *qualitative* differences emphasized above, the possibility of non-converging equilibrium behavior can also have substantial *quantitative* implications. For instance, in the numerical example illustrated in Fig. 4(c), about 90% of the population is infected in the unique steady-state equilibrium of the epidemic but non-converging equilibrium trajectories also exist in which only about 20% of the population is infected in the long run. Predictions and policy recommendations derived from models that abstract from economic complementarities and/or that restrict attention to equilibrium trajectories that converge to a steady state could therefore be substantially off-base.

Relation to the literature. Like the vast majority of the recent Covid-inspired literature, this paper follows and builds on what we refer to as the "standard model," introduced in Geoffard and Philipson (1996) ("GP") and developed further by Reluga (2010) and others. In the literature following GP, agents know their own health status, transmission occurs whenever an infected person is randomly matched with a susceptible one, and the likelihood that any two agents are matched depends on how active they each choose to be. Most closely related is Toxvaerd (2019), who analyzes a Susceptible-Infected-Susceptible model with recovery and re-infection where strategic and forward-looking agents choose their individual level of exposure dynamically, under both centralized and decentralized decision making. By contrast, we focus on the decentralized case and work within a Susceptible-Infected-Removed model without recovery.

What distinguishes our paper from the rest of this literature is that we allow for economic complementarities of activity in a dynamic setting with forward-looking agents.[3] We find that complementarities can have novel and profound qualitative and quantitative implications for the set of equilibrium epidemic trajectories. Most notably, we show that any equilibrium trajectory that enters a steady state is welfare dominated by other equilibrium trajectories that never converge but instead eventually *oscillate* over time. Moreover, the difference in infection prevalence and welfare between oscillating and steady-state equilibria can be quite large in some cases. In environments where the benefits from activity depend on others' activity, such as when employees decide whether to work from home, analyses that abstract from complementarities and from non-convergent behavior may therefore generate inaccurate predictions and policy conclusions.

We appear to be the first in the economic literature[4] to analyze non-converging equilibrium epidemic trajectories, but a variety of mechanisms have been identified that can lead to equilibrium multiplicity. Kremer (1996) and Chen (2012) provide two interesting examples, where multiplicity arises as a result of a more complex transmission technology. In a pioneering early paper, Kremer (1996) considers a model in which agents control how many encounters they have, but who they meet depends on who else is looking to meet. In that context, multiple equilibria

[3] Other notable works such as Philipson and Posner (1993), Toxvaerd (2017) and Toxvaerd (2021) have analyzed complementarities in static or agent-myopic frameworks.

[4] Hethcote and Levin (1989) shows that, in some (non-economic) epidemiological models with constant activity, the epidemic need not ever converge to a steady state.

naturally arise due to a selection effect: If few uninfected people are looking to meet, then most encounters will be with infected people and it is an equilibrium for the uninfected to avoid others. On the other hand, if many uninfected people are looking to meet, then each encounter poses less exposure risk and hence becomes more attractive for the uninfected. Within the standard model, Chen (2012) shows that multiple equilibria can exist if there is crowding in transmission, more precisely, if the "contact rate" (encounter rate per unit of others' overall activity, typically assumed constant) is decreasing in overall activity.

Also related is Philipson and Posner (1993 "PP") and the insightful albeit relatively small literature that has followed, including Toxvaerd (2017) and Toxvaerd (2021). In their classic study of the AIDS epidemic, PP introduced a rich alternative modeling approach in which agents do not observe their own health status and transmission only occurs if, upon meeting, both agents consent to consummate their interaction. The need for mutual consent creates economic complementarities much as in our model, since the expected benefit and the exposure risk associated with activity both depend on others' willingness to consent. However, the implications of such complementarities on the equilibrium set have not hitherto received much attention in this literature. An earlier version of this paper, McAdams (2020), provides an algorithm to compute the set of equilibrium trajectories in a PP-esque model with asymptomatic infection and economic complementarities. However, the set of equilibria in that richer context is quite complex, making it difficult to draw clear insights from the analysis. For this reason, we focus here on a simpler model without asymptomatic infection.

The rest of the paper is organized as follows. Section 2 presents the model and some preliminary analysis. Section 3 considers the Susceptible-Infected model, corresponding to an untreatable disease from which people cannot recover but which does not kill them. Section 4 then extends the analysis to the Susceptible-Infected-Removed model, allowing infected agents to die from the disease. Section 5 considers a variety of equilibrium comparative statics, focused especially on equilibrium welfare during the endemic phase of the epidemic. Section 6 concludes.

2. Model and preliminary analysis

A disease-causing pathogen circulates among a population of hosts (or "agents") according to a standard Susceptible-Infected-Removed (SIR) model with vital dynamics. The pathogen first emerges at time 0, grows more prevalent during an initial "outbreak phase" and then potentially persists over the long run in an "endemic phase." Epidemiological dynamics depend on agents' economic choices whether or not to be socially active.

Vital dynamics. There is a unit-mass population of agents who die from other causes at rate $r > 0$, and each infected agent dies from the disease at rate $\gamma \geq 0$. There is constant flow r of newborn susceptible agents entering the population. Let $I(t)$ be the mass of infected agents and let $N(t)$ be the mass of living agents at time t. Vital dynamics are governed by the differential equation

$$N'(t) = r(1 - N(t)) - \gamma I(t) \tag{1}$$

Epidemiological dynamics. At each point in time $t \geq 0$, each agent in the population is either susceptible (health status $h_t = S$), infected ($h_t = I$), or dead before their time due to the disease ($h_t = R$ for "removed"). All agents are susceptible at birth, creating a flow of new hosts available to be infected. Let $h(t)$ be the mass of hosts in health state $h \in \{S, I, R\}$. Note that $R(t) = 1 - N(t)$ is the mass of agents who are dead at time t due to the disease but who would otherwise still be alive.

Susceptible and infected agents know their own health status and choose at each point in time whether or not to be "active." Susceptible hosts become infected if, while active, they encounter an infected host who is also active. For simplicity, we assume that activity is a zero-one decision and that inactive agents have zero exposure risk. Let $a_h(t)$ be the fraction of h-agents who choose to be active at time $t \geq 0$. Each active S-agent encounters an active I-agent and becomes infected themselves at rate $\beta a_I(t) I(t)$; the parameter β captures the transmissibility of the disease. An S-agent who chooses not to be active at time t is certain to remain in the susceptible state.

Infected agents have a dominant strategy to be fully active (details below). To simplify equations, we may therefore set $a_I(t) = 1$ for all t.[5]

Epidemiological dynamics are determined by the system of differential equations

$$S'(t) = -\beta a_S(t) S(t) I(t) + r(I(t) + R(t)) \tag{2}$$

$$I'(t) = \beta a_S(t) S(t) I(t) - (r + \gamma) I(t) \tag{3}$$

$$R'(t) = \gamma I(t) - r R(t) \tag{4}$$

as well as the adding-up condition $S(t) + I(t) + R(t) = 1$ for all $t \geq 0$ and initial condition $(S(0), I(0), R(0))$. Motivated by the emergence of a novel infectious disease, we focus on initial conditions of the form $I(0) \approx 0$ and $R(0) = 0$.

An *epidemic trajectory* (or simply "epidemic") $\mathcal{E}$ consists of an initial condition, an epidemic process $(S(t), I(t), R(t) : t \geq 0)$, and a susceptible-activity process $(a_S(t) : t \geq 0)$, where the epidemic process is determined from the initial condition and the susceptible-activity process according to the system of differential equations (2)-(4). To parse equation (3), note that there is mass $a_S(t) S(t)$ of active S-agents, each of whom encounters an active I-agent at rate $\beta I(t)$, creating overall flow $\beta a_S(t) S(t) I(t)$ of new infections. On the other hand, each infected agent dies at rate $r + \gamma$, creating a flow $(r + \gamma) I(t)$ out of the infected state. Equation (4) is a re-expression of the vital dynamics equation (1), while (2) follows directly from (3)-(4) and the adding-up condition.

The case when $r + \gamma \geq \beta$ is trivial and uninteresting since $I'(t) < 0$ and $\lim_{t \to \infty} I(t) = 0$ regardless of agent behavior. We therefore focus on the case when $r + \gamma < \beta$.

Special case: SI model. In the special case of a non-deadly disease ($\gamma = 0$ and $R(t) = 0$ for all t), the SIR model reduces to a Susceptible-Infected (SI) model. The SI model generates much simpler epidemiological dynamics than the SIR model. In the SI model in the "full-activity trajectory" in which susceptible agents are always active ($a_S(t) = 1$ for all t), the prevalence of infection $I(t)$ is monotonically increasing over time with $\lim_{t \to \infty} I(t) = 1 - \frac{r}{\beta}$. By contrast, in the SIR model, there are non-trivial transient dynamics early during the epidemic.

Economic dynamics. Each agent i seeks to maximize their expected lifetime continuation payoff $U_i(t)$, henceforth referred to as agent i's "welfare" at time t. Agent i gets flow payoff $u_i(t)$ equal to the sum of their "health flow payoff" and "social-economic flow payoff" (discussed below) while alive and has zero continuation payoff upon death.

[5] In reality, infected agents' transmissive activity may be constrained, e.g., if their sickness is incapacitating, if others can perceive that they are infected and shun transmissive contact (as with leprosy), and so on. Such effects can be incorporated into the transmission rate. In particular, if sickness is incapacitating for fraction s of I-agents, then only fraction $(1 - s)$ of them will be active and infection will spread as if the transmission rate were $\widehat{\beta} = (1 - s)\beta$.

Health: Health flow payoff is 0 if susceptible and $-d$ if infected; the parameter $d > 0$ captures the severity of the symptoms associated with the disease. Let $H_i(t)$ denote agent i's expected lifetime continuation health payoff (or simply "lifetime health") at time t.

Social-economic well-being ("wealth"): Social-economic flow payoff is b_0 for inactive agents and $b_0 + b_1 + b_2 A(t)$ for active agents, where we use shorthand

$$A(t) = a_S(t)S(t) + I(t) \tag{5}$$

for the mass of active hosts at time t. Let $W_i(t)$ denote agent i's expected lifetime continuation social-economic payoff (or simply "lifetime wealth") at time t.

The parameter $b_0 \geq 0$ captures the baseline benefit of being alive, including the benefits of all *safe activity* that does not put one at risk of exposure. $b_1 > 0$ captures the benefit of *public non-social activity* that puts an agent at risk of exposure to the virus but whose value to the agent does not depend on whether others are also active, e.g., going to the gym. $b_2 \geq 0$ captures the additional benefit of *public social activity* that arises when people are active at the same time. Such "social benefits" can arise for several sorts of reasons, such as (i) if people enjoy being around others, (ii) if the purpose of activity is to match with someone else and a better match can be found when more people are active, or (iii) if more aggregate activity leads to more individual opportunities to benefit from activity, e.g., if more stores open when more people are out looking to shop.

Equilibrium epidemics. An *equilibrium trajectory* (or "equilibrium epidemic") is one in which each living agent's activity choice at each time $t \geq 0$ maximizes their welfare given their time-t health status $h_t \in \{S, I\}$, the current state of the epidemic process, and the rest of the trajectory after time t. Because all agents must be optimizing along any equilibrium trajectory $\mathcal{E}$, all agents with the same health status must have the same welfare $U_h(t; \mathcal{E})$. Let $H_h(t; \mathcal{E})$ and $W_h(t; \mathcal{E})$ denote the lifetime health and lifetime wealth of h-agents at time t. Unless needed for clarity, we will usually suppress notation for the underlying epidemic trajectory; so, $U_h(t) = H_h(t) + W_h(t)$ for all $h \in \{S, I\}$ and all $t \geq 0$. Let $\Delta U(t) \equiv U_S(t) - U_I(t)$ be the "harm of infection" at time t, the amount by which an agent's lifetime welfare falls instantaneously at time t upon becoming infected.

Lemma 1 gathers together some basic facts that will be useful later in the analysis.

Lemma 1. *In any equilibrium epidemic: (i) if $a_S(t) \in (0, 1)$, then $b_1 + b_2 A(t) = \beta I(t) \Delta U(t)$; and (ii) if $a_S(t') < 1$ for all $t' \geq t$, then $U_S(t) = \frac{b_0}{r}$.*

Proof. (i) If $a_S \in (0, 1)$, then S-agents must be indifferent whether to be active. Being inactive guarantees that an S-agent will get baseline flow payoff b_0 plus continuation payoff $U_S(t)$. By contrast, being active increases the agent's flow payoff to $b_0 + b_1 + b_2 A(t)$ while causing them to transition to the infected state at rate $\beta I(t)$. Thus, each S-agent finds it strictly optimal to be active at time t when

$$b_1 + b_2 A(t) > \beta I(t) \Delta U(t), \tag{6}$$

and is indifferent whether to be active when $b_1 + b_2 A(t) = \beta I(t) \Delta U(t)$. (ii) If $a_S(t') < 1$ for all $t' \geq t$, then S-agents are indifferent at each point in time whether to be active and hence must get the same payoff as if they were to choose to be inactive for their entire lives. Because inactive S-agents get flow b_0 payoff and have expected lifetime $\frac{1}{r}$, we conclude that $U_S(t) = \frac{b_0}{r}$. □

Epidemics in the long run. We are especially interested in the prevalence of infection and agents' welfare in the long run of an equilibrium epidemic. However, as we will show, not every equilibrium epidemic settles into a long-run steady state.

Definition 1 *(Long-run infection range).* An epidemic trajectory $\mathcal{E}$ has "long-run infection range" $[\underline{I}^{\infty}, \overline{I}^{\infty}]$, where $\overline{I}^{\infty} \equiv \limsup_t I(t)$ and $\underline{I}^{\infty} \equiv \liminf_t I(t')$. If $\underline{I}^{\infty} = \overline{I}^{\infty} = I^{\infty}$, then $\mathcal{E}$ has "long-run infection prevalence" I^{∞}.

Suppose that an epidemic has long-run infection prevalence I^{∞}. Equation (4) implies that the mass of recovered agents must converge in the long run to $R^{\infty} = \frac{\gamma}{r} I^{\infty}$, and so the mass of susceptible agents must converge to $S^{\infty} = 1 - \frac{r+\gamma}{r} I^{\infty}$.

Welfare comparisons. In the course of our analysis, we will use two main notions to compare agents' welfare in different epidemic trajectories. The strongest sense in which we compare epidemic trajectories is that of "welfare dominance."

Definition 2 *(Welfare dominance).* Trajectory $\mathcal{E}'$ "welfare dominates" $\mathcal{E}$ if (i) $U_S(t;\mathcal{E}')S(t;\mathcal{E}') + U_I(t;\mathcal{E}')I(t;\mathcal{E}') \geq U_S(t;\mathcal{E})S(t;\mathcal{E}) + U_I(t;\mathcal{E})I(t;\mathcal{E})$ for all t and (ii) $U_h(t;\mathcal{E}') \geq U_h(t;\mathcal{E})$ for all $h \in \{S, I\}$ and all t.

Condition (i) means that the aggregate welfare of all living agents is always higher along trajectory $\mathcal{E}'$. Condition (ii) means that each living agent always prefers trajectory $\mathcal{E}'$ given their current health status.

We also compare epidemic trajectories based on the welfare of susceptible agents only. Because newborn agents are susceptible, S-agent welfare $U_S(t;\mathcal{E})$ is the welfare of someone born at time t, i.e., "newborn welfare."

Definition 3 *(Better for newborns).* Trajectory $\mathcal{E}'$ has "higher newborn welfare" than $\mathcal{E}$ if, for all $t \geq 0$, $U_S(t;\mathcal{E}') > U_S(t;\mathcal{E})$, i.e., $\mathcal{E}'$ is better than $\mathcal{E}'$ for all agents at birth.

Note that, along an epidemic trajectory that is better from a welfare point of view, there may be more sickness and/or more death. For instance, it could be trajectory $\mathcal{E}'$ is better for newborns than trajectory $\mathcal{E}$ but $S(t;\mathcal{E}') < S(t;\mathcal{E})$ and/or $R(t;\mathcal{E}') > R(t;\mathcal{E})$ at some or all times $t > 0$. However, for that to be the case, any anticipated welfare losses due to increased sickness and/or accelerated death must be more than compensated by welfare gains due to increased social-economic activity for each newborn agent to view themselves as better off.

3. Susceptible-Infected (SI) analysis

This section develops our main findings within the context of the Susceptible-Infected (SI) model in which no one dies from the disease. Focusing on the SI model allows us to simplify the analysis in two main ways. First, since $\gamma = 0$, the system of differential equations (2)-(4) reduces to the single differential equation

$$I'(t) = \beta a_S(t) S(t) I(t) - r I(t) \tag{7}$$

plus the adding-up condition $S(t) + I(t) = 1$. Second, because agents live for expected length of time $\frac{1}{r}$ no matter what, the fact that being alive generates baseline flow benefit b_0 lifts all agents'

welfare by $\frac{b_0}{r}$ but otherwise has no effect on incentives or the equilibrium set. Without loss of generality, we may therefore set $b_0 = 0$ to simplify equations.

When an epidemic first emerges, infection is sufficiently rare that all agents have an incentive to be fully active and the prevalence of infection grows exponentially over time. There are two basic possibilities for how the epidemic then progresses: (i) *sustained full activity*, if all agents remain fully active forever; or (ii) *eventual social distancing* if, at some time, at least some susceptible agents choose not to be active.

The full-activity trajectory. Let $\overline{\mathcal{E}}$ be the epidemic trajectory that arises when S-agents are always active, and let $\overline{I}(t)$ be the time-t prevalence of infection along $\overline{\mathcal{E}}$. That is, $(\overline{I}(t) : t \geq 0)$ is determined by differential equation $\overline{I}'(t) = \beta \overline{S}(t)\overline{I}(t) - r\overline{I}(t)$, where $\overline{S}(t) = 1 - \overline{I}(t)$. As can be easily checked, $\overline{I}'(t) > 0$ for all t and $\lim_{t\to\infty} \overline{I}(t) = 1 - \frac{r}{\beta}$. Proposition 1 provides a simple condition that characterizes when $\overline{\mathcal{E}}$ is an equilibrium trajectory.

Proposition 1. *In the SI model, the full-activity trajectory $\overline{\mathcal{E}}$ is an equilibrium epidemic trajectory if and only if disease severity $d \leq \underline{d}$, where*

$$\underline{d} \equiv \frac{\beta(b_1 + b_2)}{\beta - r}. \tag{8}$$

Proof. Along the full-activity trajectory, all agents get flow economic payoff $b_1 + b_2$ but the health flow payoff for S-agents is d higher than for I-agents. Consequently, $\Delta U(t) = dL(t)$ where $L(t)$ is the expected length of time that an S-agent remains alive and uninfected. $L(t)$ is the mean of an exponential distribution with arrival rate $\beta I(t) + r$. Because infection prevalence $I(t)$ increases over time from approximately zero to $\lim_{t\to\infty} I(t) = 1 - \frac{r}{\beta}$, $L(t)$ decreases over time from approximately $\frac{1}{r}$ to $\lim_{t\to\infty} L(t) = \frac{1}{\beta - r + r} = \frac{1}{\beta}$.

Suppose that $d \leq \underline{d}$. By equation (6), S-agents find it optimal to be active at time t if and only if $\beta I(t)\Delta U(t) \leq b_1 + b_2$. Because $\Delta U(t) = dL(t)$, $L(t) < \frac{1}{\beta}$ for all t, and $I(t) < 1 - \frac{r}{\beta}$ for all t, we have $\beta I(t)\Delta U(t) < \frac{d(\beta - r)}{\beta}$ for all t. By equation (8) and the fact that $d \leq \underline{d}$, we conclude that $\beta I(t)\Delta U(t) < b_1 + b_2$ and hence that S-agents find it strictly optimal to be active at all times; so, $\overline{\mathcal{E}}$ is an equilibrium trajectory.

Suppose next that $d > \underline{d}$. Because $\lim_{t\to\infty} I(t) = 1 - \frac{r}{\beta}$ and $\lim_{t\to\infty} L(t) = \frac{1}{\beta}$, we have $\lim_{t\to\infty} \beta I(t)\Delta U(t) = \lim_{t\to\infty} \beta d I(t)L(t) = \frac{d(\beta - r)}{\beta} > b_1 + b_2$; so, S-agents strictly prefer not to be active sufficiently far into the epidemic and $\overline{\mathcal{E}}$ is not an equilibrium trajectory. □

Equilibrium uniqueness without social benefits of activity. If $b_2 = 0$, then there is a unique equilibrium trajectory and this equilibrium follows *either* the full-activity trajectory *or* an especially simple "rise-and-plateau trajectory" whereby all S-agents are active until the level of infection hits a critical threshold I^* at some time T^*, after which S-agents randomize whether to be active with just the right probability so that $I(t) = I^*$ for all $t \geq T^*$.

Definition 4. A "rise-and-plateau trajectory" is an epidemic trajectory that consists of (i) an outbreak phase during which all agents are active, followed by (ii) an endemic phase after time T in which the prevalence of infection is constant.

Proposition 2. *Consider the SI model. When $b_2 = 0$ and $d > \underline{d}$,[6] there is a unique equilibrium epidemic trajectory $\mathcal{E}^*$. Moreover, $\mathcal{E}^*$ is a rise-and-plateau trajectory with plateau infection level $I^* \equiv \frac{b_1 r}{\beta(d-b_1)}$.*

Proof. Proposition 2 follows directly from Proposition 8, which provides sufficient conditions for equilibrium uniqueness in the SIR model which include these as a special case. □

Equilibrium epidemics more broadly. A rise-and-plateau trajectory is the simplest path that an epidemic can take, but there are many other possibilities. Suppose that $\mathcal{E}$ is an equilibrium epidemic and that, in this trajectory, at least some S-agents are active at time $\tilde{t} > 0$.[7] Lemma 2 shows how to construct a *new* equilibrium epidemic trajectory $\widetilde{\mathcal{E}}$ by pasting together an initial portion of the full-activity trajectory $\overline{\mathcal{E}}$ with the remainder of the original trajectory $\mathcal{E}$ after time $\tilde{t}$.

Lemma 2. *In the SI model, suppose that $\mathcal{E}$ is an equilibrium epidemic trajectory with $a_S(\tilde{t}) > 0$ for some $\tilde{t} > 0$, and define t^O implicitly by $\overline{I}(t^O) = I(\tilde{t})$. Then $\widetilde{\mathcal{E}}$ is also an equilibrium epidemic trajectory, determined by the S-agent activity process $(\tilde{a}_S(t) : t \geq 0)$ as follows:*

- *$\tilde{a}_S(t) = 1$ for all $t \leq t^O$ ("pasted" initial outbreak)*
- *$\tilde{a}_S(t^O + x) = a_S(\tilde{t} + x)$ for all $x \geq 0$ (remainder of original trajectory)*

Proof. Because $\mathcal{E}$ is an equilibrium epidemic trajectory and $a_S(\tilde{t}) > 0$, S-agents find it optimal to be active at time t^O and agents' behavior after time t^O constitutes an equilibrium of the continuation game. We need to show (only) that S-agents find it optimal along $\widetilde{\mathcal{E}}$ to be active at times $t \leq t^O$.

Let $U_S(t; \widetilde{\mathcal{E}})$ denote the welfare of a S-agent i at time t along the trajectory $\widetilde{\mathcal{E}}$, assuming that others behave as prescribed along the trajectory (whether individually-optimal or not) and agent i plays an individually-optimal best response. Similarly, let $U_I(t; \widetilde{\mathcal{E}})$ be the welfare of optimizing I-agents, and let $\Delta U(t; \widetilde{\mathcal{E}}) = U_S(t; \widetilde{\mathcal{E}}) - U_I(t; \widetilde{\mathcal{E}})$ be the time-t harm of infection. Since all others are active at times $t < t^O$, agent i finds it optimal to be active if and only if

$$\beta I(t) \Delta U(t; \widetilde{\mathcal{E}}) \leq b_1 + b_2 \tag{9}$$

by inequality (6). (For ease of exposition, we henceforth drop "$\widetilde{\mathcal{E}}$ notation.")

Because S-agents find it optimal to be active at time t^O, we have $\beta I(t^O)\Delta U(t^O) \leq b_1 + A(t^O)b_2 \leq b_1 + b_2$; thus, condition (9) holds at time t^O.

Suppose for the sake of contradiction that condition (9) fails for some $t' < t^O$. By a simple continuity argument, there must be an interval $[t', t'')$ over which agent i strictly prefers not to be active but becomes indifferent whether to be active at time t'', for some $t'' \leq t^O$. In particular, (9) holds with equality at time t''.

Because S-agent i finds it optimal not to be active from time t' to t'', agent i gets zero economic payoff and is certain to avoid infection during this time. Since agent i survives from time t' to t'' with probability $e^{-r(t''-t')}$, we have $U_S(t') = e^{-r(t''-t')} U_S(t'')$. Moreover, $U_S(t'') \geq 0$ since S-agents can guarantee zero payoff by remaining inactive; so, $U_S(t') \leq U_S(t'')$.

[6] When $b_2 = 0$ and $d \leq \underline{d}$, the full-activity trajectory is the unique equilibrium (details omitted for space).

[7] In any equilibrium continuation trajectory starting from initial condition $I(t)$ at time t, there must be a future time $t' > t$ with some S-agent activity. If not, the prevalence of infection would fall to zero and all agents would eventually have a dominant strategy to be active, a contradiction.

What about infected agents? Because they are always active, I-agents' welfare takes the form $U_I(t') = \int_{t'}^{\infty} e^{-r(t-t')}(b_1 + A(t)b_2)dt - \frac{d}{r}$, where $\frac{1}{r}$ is agents' expected lifetime. Since all agents are active until time t^O, we have $A(t) = 1$ for all $t < t^O$ and, of course, $A(t) \leq 1$ for all $t > t^O$. $U_I(t)$ is therefore weakly decreasing over the interval $[t', t^O]$ and, in particular, $U_I(t'') \leq U_I(t')$.

All together, we have $U_S(t'') \geq U_S(t')$ and $U_I(t'') \leq U_I(t')$; so, $\Delta U(t'') \geq \Delta U(t')$. Since infection prevalence is rising, $I(t'') > I(t')$ and hence $\beta I(t')\Delta U(t') < \beta I(t'')\Delta U(t'') = b_1 + b_2$, where the second equality holds because S-agents are indifferent whether to be active at time t''. We conclude that S-agents strictly prefer to be active at time t', a contradiction. □

Discussion of Lemma 2: In Sections 3.1-3.2, we will construct equilibrium continuation trajectories starting from initial conditions in which infection is already widespread. Lemma 2 shows that these continuation trajectories can in fact be "reached" along equilibrium trajectories that start from an initial condition with rare infection and take an especially simple form, consisting of (i) an "outbreak phase" in which all agents are fully active followed immediately by (ii) an "endemic phase" in which further play follows the equilibrium continuation trajectory in question. See Propositions 4 and 5.

Bearing this in mind, the rest of this section focuses on situations in which infection is already widespread. We begin in Section 3.1 by characterizing what steady states can arise in equilibrium. Then in Section 3.2, we consider the simplest sort of non-converging trajectory, so-called "oscillating trajectories" in which S-agents alternate regularly between activity and inactivity, causing infection prevalence to rise and fall regularly over time.[8]

3.1. Steady-state equilibria

A "steady-state trajectory" is one with constant infection prevalence $I > 0$ and constant susceptible-agent activity a_S. This section provides a starting point for our analysis of the endemic phase of an infectious disease, by characterizing all steady-state trajectories that can arise in equilibrium. Our main finding is that, whenever the disease is sufficiently severe that $d > \underline{d}$ (defined in Proposition 1), there is a unique steady-state equilibrium and, in this steady state, susceptible agents' lifetime welfare is the same as if they were required to be inactive for their entire lives.

Definition 5. A "steady-state equilibrium (SSE)" with infection prevalence I is a steady-state trajectory that is also an equilibrium trajectory starting from initial condition $I(0) = I$.

If $a_S \leq \frac{r}{\beta}$ and all S-agents are active with probability a_S, then $I'(t) < I(t)(S(t) - 1) < 0$ for all t by equation (7), causing the level of infection to decline toward zero. This can never occur in equilibrium, as S-agents have an incentive to be active once $I(t) \approx 0$. We may therefore restrict attention to $a_S \in (\frac{r}{\beta}, 1]$. For each such activity level, there is a corresponding steady-state infection level $I^{SS}(a_S) \equiv 1 - \frac{r}{\beta a_S}$. Let $\mathcal{E}^{SS}(a_S)$ denote the steady-state trajectory with S-agent activity $a_S \in (\frac{r}{\beta}, 1]$ and infection prevalence $I^{SS}(a_S)$, and let $U_h(\mathcal{E}^{SS}(a_S))$ be agents' steady-state welfare in each health status $h \in \{S, I\}$.

[8] It is easy to show that, whenever an oscillating equilibrium trajectory exists, other non-converging equilibrium trajectories also exist without a regular oscillation. However, such equilibria are difficult to characterize and analyze, and do not appear to generate additional qualitative insights.

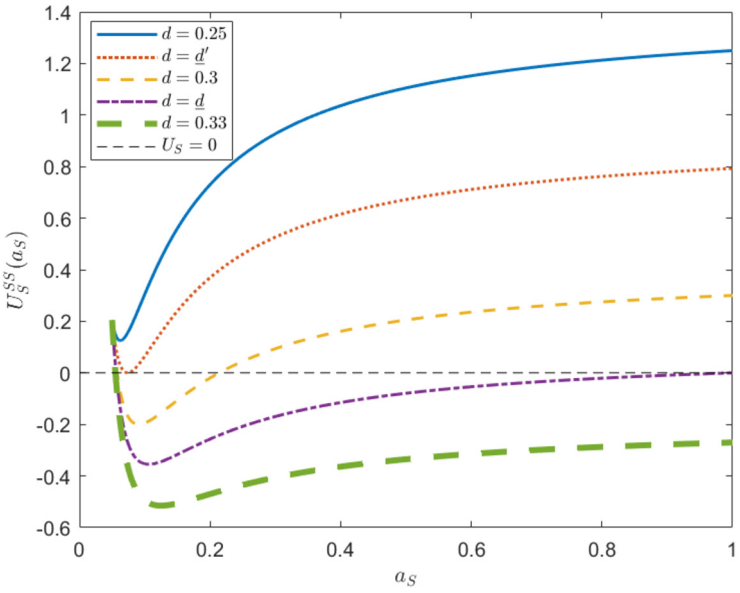


Fig. 1. Illustration of our characterization of all steady-state equilibria (SSEs), as disease severity d varies with other fixed parameters $\beta = 1$, $b_0 = 0$, $b_1 = 0.2$, $b_2 = 0.1$, and $r = 0.05$. Each zero of the function $U_S^{SS}(a_S)$ over $a_S \in (\frac{r}{\beta}, 1)$ corresponds to a partial-activity SSE. When d is sufficiently small (blue curve), the unique SSE has full activity. When d is sufficiently large (green curve), exactly one partial-activity SSE exists. In between (red, yellow, and purple curves), a full-activity SSE exists as well as one or two partial-activity SSEs. (For interpretation of the colors in the figure(s), the reader is referred to the web version of this article.)

Full-activity steady state. Here we show that the full-activity steady state $\mathcal{E}^{SS}(1)$ is an SSE if and only if $d \leq \underline{d}$. In this steady state, the infection level $I^{SS}(1) = 1 - \frac{r}{\beta}$, I-agents earn constant flow payoff $b_1 + b_2 - d$ and hence have welfare $U_I^{SS}(1) = \frac{b_1+b_2-d}{r}$. S-agents earn flow payoff $b_1 + b_2$ while uninfected, remain uninfected for average length of time $\frac{1}{(\beta-r)+r} = \frac{1}{\beta}$, become infected at rate $\beta I^{SS}(1) = \beta - r$, and have ex ante likelihood $\frac{\beta-r}{(\beta-r)+r} = \frac{\beta-r}{\beta}$ of becoming infected before death. Overall, then, S-agents have welfare $U_S^{SS}(1) = \frac{b_1+b_2}{\beta} + \frac{\beta-r}{\beta} U_I^{SS}(1) = \frac{b_1+b_2}{r} - \frac{d(\beta-r)}{\beta r}$ and the steady-state harm of infection is $\Delta U^{SS}(1) = \frac{d}{\beta}$. By the incentive condition (6), $\mathcal{E}^{SS}(1)$ is an equilibrium trajectory if and only if $b_1 + b_2 \geq \beta I^{SS}(1) \Delta U^{SS}(1) = \frac{d(\beta-r)}{\beta}$, which holds if and only if $d \leq \underline{d}$ as defined in (8).

Partial-activity steady states. Consider any $a_S \in (\frac{r}{\beta}, 1)$ and the partial-activity steady-state trajectory $\mathcal{E}^{SS}(a_S)$. This is an SSE if and only if S-agents are indifferent whether to be active. Because inactivity guarantees zero flow payoff, such indifference arises if and only if each S-agent has zero welfare along $\mathcal{E}^{SS}(a_S)$, i.e., $U_S^{SS}(a_S) = 0$. This observation provides a simple way to characterize the full set of SSEs, by identifying the set of activity levels $a_S \in (\frac{r}{\beta}, 1)$ such that S-agents have zero welfare in the steady-state trajectory $\mathcal{E}^{SS}(a_S)$.

Numerical example. Fig. 1 provides an illustration of our method of determining the set of SSEs, highlighting how the SSE set varies with disease severity d, in a numerical example. In particular, suppose that $\beta = 1$, $b_0 = 0$, $b_1 = 0.2$, $b_2 = 0.1$, and $r = 0.05$, given which $\underline{d}' \approx 0.274$ and $\underline{d} \approx 0.316$. There are three main possibilities for the SSE set, depending on disease severity.

- *low-severity disease:* If $d < \underline{d}'$ (e.g., $d = 0.25$), then $U_S^{SS}(a_S) > 0$ for all $a_S > \frac{r}{\beta}$. In this case, there is a unique SSE, which has full activity.
- *intermediate-severity disease:* If $\underline{d} < d < \underline{d}'$ (e.g., $d = 0.3$), then $U_S^{SS}(a_S) = 0$ at two activity levels less than one and $U_S^{SS}(1) > 0$. In this case, there are three SSEs, one with full activity and two with partial activity.
- *high-severity disease:* If $d > \underline{d}$ (e.g., $d = 0.33$), then $U_S^{SS}(a_S) = 0$ at a unique activity level less than one and $U_S^{SS}(1) < 0$. In this case, there is a unique SSE, which has partial activity.

Proposition 3 summarizes our main findings about the SSE set, focusing especially on the (surprisingly narrow) conditions under which there are multiple SSEs.

Proposition 3. *Consider the SI model. (i) An SSE exists. (ii) There is a unique SSE if and only if any of the following conditions hold: (a) there are no social benefits of economic activity, i.e., $b_2 = 0$; (b) agents are sufficiently short-lived that $r \geq \frac{\beta b_2}{b_1 + 2b_2}$; and/or (c) disease severity is greater than $\underline{d}$ or less than $\underline{d}'$, where*

$$\underline{d}' \equiv b_1 + 2r\frac{b_2}{\beta} + 2\sqrt{\frac{rb_2}{\beta}(b_1 + \frac{rb_2}{\beta})}. \tag{10}$$

Proof. *Part (i).* If $d \leq \underline{d}$, then a full-activity SSE exists. If $d > \underline{d}$, then $b_1 + b_2 < \beta I^{SS}(1)\Delta U^{SS}(1)$. However, because $\lim_{a_S \to r/\beta} I^{SS}(a_S) = 0$, we have $b_1 + b_2 > \beta I^{SS}(a_S)\Delta U^{SS}(a_S)$ for all $a_S \approx \frac{r}{\beta}$. By continuity, there must exist some $\hat{a}_S \in (\frac{r}{\beta}, 1)$ such that $b_1 + b_2 = \beta I^{SS}(\hat{a}_S)\Delta U^{SS}(\hat{a}_S)$, and $\mathcal{E}^{SS}(\hat{a}_S)$ is an ESS. This completes the proof of (i).

Part (ii). Define the following shorthand: $I(a_S) \equiv I^{SS}(a_S) = 1 - \frac{r}{\beta a_S}$ for infection prevalence in the steady-state trajectory $\mathcal{E}^{SS}(a_S)$; $A(a_S) = I(a_S) + a_S(1 - I(a_S)) = I(a_S) + \frac{r}{\beta}$ for population-wide activity; and $U_S(a_S; d)$ and $U_I(a_S; d)$ for the welfare of susceptible and infected agents, viewed here also as functions of disease severity d.

As discussed earlier: $\mathcal{E}^{SS}(1)$ is an SSE if and only if $U_S(1; d) \geq 0$, which holds whenever $d \leq \underline{d}$; and for each $a_S \in (\frac{r}{\beta}, 1)$, $\mathcal{E}^{SS}(a_S)$ is an SSE if and only if $U_S(a_S; d) = 0$. It remains for us to characterize when $U_S(a_S; d) = 0$ and show that SSE is unique under the stated conditions. We begin by deriving $U_I(a_S; d)$ and $U_S(a_S; d)$.

Infected-agent welfare: I-agents get flow payoff $b_1 + A(a_S)b_2 - d$ until they die. Since death arrives at rate r, each agent's expected length of life is $\frac{1}{r}$; so $U_I(a_S; d) = \frac{b_1 + A(a_S)b_2 - d}{r}$.

Susceptible-agent welfare: S-agents get flow payoff $a_S(b_1 + A(a_S)b_2)$ until they either die or become infected. Since infection arrives at rate $\beta a_S I(a_S) = \beta a_S - r$ and death at rate r, each S-agent remains susceptible for expected length of time $\frac{1}{(\beta a_S - r) + r} = \frac{1}{\beta a_S}$ and becomes infected prior to death with likelihood $\frac{\beta a_S - r}{\beta a_S} = I(a_S)$, in which case they have continuation welfare $U_I(a_S; d)$. All together, then,

$$U_S(a_S; d) = \frac{1}{\beta a_S} a_S \Big(b_1 + A(a_S)b_2 \Big) + I(a_S)U_I(a_S; d) \tag{11}$$

$$= \Big(b_1 + \Big(I(a_S) + \frac{r}{\beta} \Big) b_2 \Big) \Big(\frac{1}{\beta} + \frac{I(a_S)}{r} \Big) - d\frac{I(a_S)}{r} \tag{12}$$

Because $U_S(a_S; d)$ is linearly decreasing in d, there is a unique disease severity $d(a_S)$ given which $U_S(a_S; d(a_S)) = 0$ for any given $a_S \in (\frac{r}{\beta}, 1]$:

$$d(a_S) = \left(b_1 + \left(I(a_S) + \frac{r}{\beta}\right)b_2\right)\left(1 + \frac{r}{\beta I(a_S)}\right) \text{ for all } a_S \in \left(\frac{r}{\beta}, 1\right]. \tag{13}$$

Each partial-activity steady state $\mathcal{E}^{SS}(a_S)$ is an SSE if and only if disease severity $d = d(a_S)$ and the full-activity steady state $\mathcal{E}^{SS}(1)$ is an SSE if and only if $d \leq d(1) = \underline{d}$.

Lemma 3 establishes several useful facts about $d(a_S)$.

Lemma 3. *Consider the SI model. (i) If* $r \geq \frac{\beta b_2}{b_1+2b_2}$, *then* $d(a_S)$ *is strictly decreasing over* $a_S \in (\frac{r}{\beta}, 1]$. *(ii) If* $r < \frac{\beta b_2}{b_1+2b_2}$, *then* $-d(a_S)$ *is single-peaked with* $\arg\min_{a_S} d(a_S) \in (\frac{r}{\beta}, 1)$, *and* $\min_{a_S} d(a_S) = \underline{d}' < \underline{d}$, *where* $\underline{d}'$ *is defined in Proposition 3.*

Proof. We can re-write (13) as $d(a_S) = b_1 + 2r\frac{b_2}{\beta} + I(a_S)b_2 + \frac{rb_1+r^2\frac{b_2}{\beta}}{\beta I(a_S)}$. Since $I(a_S)$ is an increasing function of a_S, we can think of d also as a function of steady-state infection prevalence I, i.e., $d(I) = b_1 + 2r\frac{b_2}{\beta} + Ib_2 + \frac{rb_1+r^2\frac{b_2}{\beta}}{\beta I}$. Taking a derivative yields

$$d'(I) = b_2 - \frac{r(b_1 + r\frac{b_2}{\beta})}{\beta I^2}.$$

$d'(I) \gtrless 0$ when $I \gtrless \hat{I} \equiv \sqrt{\frac{r^2}{\beta^2} + \frac{b_1}{b_2}\frac{r}{\beta}}$. We conclude that $d(I)$ is strictly decreasing in I for all $I \in (0, \hat{I})$ and, if $\hat{I} < 1 - \frac{r}{\beta}$, strictly increasing in I for all $I \in (\hat{I}, 1 - \frac{r}{\beta})$. Or equivalently, $d(a_S)$ is strictly decreasing in a_S whenever $a_S \in (\frac{r}{\beta}, \frac{r}{\beta(1-\hat{I})})$ and strictly increasing in a_S whenever $a_S \in (\frac{r}{\beta(1-\hat{I})}, 1]$.

Note that $\hat{I}$ is strictly increasing in r, with $\hat{I} > 1 - \frac{r}{\beta}$ if and only if $r > \hat{r} \equiv \frac{\beta b_2}{b_1+2b_2}$. Suppose first that $r \geq \hat{r}$ so that $\hat{I} \geq 1 - \frac{r}{\beta}$. Since $I(a_S) < 1 - \frac{r}{\beta}$ for all $a_S \in (\frac{r}{\beta}, 1)$, we conclude that $d(a_S)$ is strictly decreasing over the whole interval $a_S \in (\frac{r}{\beta}, 1]$, as desired. Suppose next that $r < \hat{r}$, so that $\hat{I} < 1 - \frac{r}{\beta}$. Since $I(a_S)$ is strictly increasing with $\lim_{a_S \searrow r} I(a_S) = 0$ and $\lim_{a_S \nearrow 1} I(a_S) = 1 - \frac{r}{\beta}$, there exists $\hat{a}_S \in (\frac{r}{\beta}, 1)$ such that $I(\hat{a}_S) = \hat{I}$ and hence $I(a_S) > \hat{I}$ if and only if $a_S > \hat{a}_S$. We conclude that $d'(a_S) < 0$ for all $a_S \in (r, \hat{a}_S)$ and $d'(a_S) > 0$ for all $a_S \in (\hat{a}_S, 1)$. This establishes that $-d(a_S)$ is single-peaked over $a_S \in (\frac{r}{\beta}, 1)$, as desired.

Lastly, in the case when $r < \hat{r}$ so that $d(I)$ is non-monotone, define $\underline{d}' \equiv \min_I d(I) = d(\hat{I})$. The fact that $\underline{d}' = b_1 + 2r\frac{b_2}{\beta} + 2\sqrt{\frac{rb_2}{\beta}(b_1 + \frac{rb_2}{\beta})}$ can be verified directly through tedious algebra, but a more elegant approach is to recognize that the geometric mean of Ib_2 and $\frac{rb_1+r^2\frac{b_2}{\beta}}{\beta I}$ is $\sqrt{\frac{rb_2}{\beta}(b_1 + \frac{rb_2}{\beta})}$. The AM-GM Inequality therefore implies that $d(a_S) - b_1 - 2r\frac{b_2}{\beta} = Ib_2 + \frac{rb_1+r^2\frac{b_2}{\beta}}{\beta I} \geq 2\sqrt{\frac{rb_2}{\beta}(b_1 + \frac{rb_2}{\beta})}$, with the equality being realized only when $I = \hat{I}$. Thus, the global minimum of $d(I)$ is $b_1 + 2r\frac{b_2}{\beta} + 2\sqrt{\frac{rb_2}{\beta}(b_1 + \frac{rb_2}{\beta})}$, as desired. □

We are now ready to verify the specific conditions for SSE uniqueness in Proposition 3.

(a-b) Suppose first that $r \geq \frac{\beta b_2}{b_1+2b_2}$, including as a special case any situation with $b_2 = 0$. By Lemma 3, $d(a_S)$ is continuously decreasing over $a_S \in (\frac{r}{\beta}, 1]$, from $\lim_{a_S \searrow r} d(a_S) = \infty$ to $d(1) = \underline{d}$. We establish SSE uniqueness in two cases. First, for any $d > \underline{d}$, there is exactly one activity level a_S such that $d(a_S) = d$, which is between r and 1; thus, there is a unique SSE and this SSE has partial activity. Second, for any $d \leq \underline{d}$, no partial-activity SSE exists because $d(a_S) > \underline{d} \geq d$ for all $a_S < 1$. However, a full-activity SSE exists by Proposition 1.

(c) Suppose next that $r < \frac{\beta b_2}{b_1+2b_2}$. By Lemma 3, $d(a_S)$ is continuously decreasing over $a_S \in (\frac{r}{\beta}, \hat{a}_S)$, reaching its minimum at $d(\hat{a}_S) = \underline{d}'$, then is continuously increasing over $(\hat{a}_S, 1]$ with $d(1) = \underline{d}$. There are three main cases, in two of which there is a unique SSE. First, for any $d > \underline{d}$, there is exactly one SSE activity level $a_S(d)$ supported by disease severity d, and $a_S(d) \in (\frac{r}{\beta}, \hat{a}_S)$. Second, for any $d < \underline{d}'$, we have $d \in \mathcal{D}(1)$ but $d(a_S) > \underline{d}' \geq d$ for all $a_S < 1$; thus, the unique SSE has full activity. The main difference with part (i) of the proof is that multiple SSE exist whenever $d \in [\underline{d}', \underline{d}]$. In particular: a full-activity SSE exists over this entire range; and when $d \in (\underline{d}', \underline{d})$, two partial-activity SSE exist, one with activity less than $\hat{a}_S$ and the other with activity more than $\hat{a}_S$. This completes the proof of (ii). □

Rise-and-plateau epidemic trajectories. For any given partial-activity steady state with infection level I, each S-agent is active with probability $a_S = \frac{\beta}{r(1-I)} > 0$. Lemma 2 therefore implies that, starting from an initial condition in which infection is rare, an equilibrium epidemic trajectory exists in which all agents are active until the infection level reaches I, after which continuation play follows the partial-activity SSE in question. Proposition 4 summarizes this observation that rise-and-plateau equilibrium trajectories exist whenever a partial-activity SSE exists.

Proposition 4. *In the SI model, suppose that a partial-activity SSE exists with infection level I. Then a rise-and-plateau equilibrium trajectory exists in which that partial-activity SSE is played during the endemic phase.*

3.2. Oscillating equilibrium trajectories

> "Eat, drink, and be merry, for tomorrow we all stay home."
> – variation on a famous proverb, for those in an oscillating epidemic trajectory

This section expands our analysis to consider non-steady state "oscillating trajectories," whereby the endemic phase of the epidemic consists of alternating periods in which susceptible agents are all active and then all inactive. We have three main results. First, we characterize the full set of oscillating equilibrium trajectories (OETs), which can vary quite substantially in terms of endemic disease prevalence and agent welfare. Second, whenever a partial-activity steady-state equilibrium (SSE) exists, we show that "barely-oscillating trajectories" that approximate that SSE are also equilibrium trajectories, and that some of these nearby non-steady-state equilibrium trajectories Pareto dominate the SSE. Finally, we characterize the set of barely-oscillating equilibrium trajectories ("barely-OETs") and show that susceptible-agent welfare is maximized in the barely-OET with the least infection. This contrasts with our earlier finding that the SSE with the most infection Pareto dominates all other SSEs whenever there are multiple SSEs (corollary to Proposition 3).

Characterization of all oscillating equilibrium trajectories. *Outline of approach:* First, we define and describe all epidemiologically-feasible oscillating trajectories. In any such trajectory, we derive the prevalence and harm of infection at each point in time. This then allows us to derive necessary and sufficient incentive-compatibility conditions for that trajectory to arise in equilibrium.

Definition 6. An "oscillating epidemic trajectory" is one such that:

- infection prevalence oscillates with period length $T \equiv T_1 + T_2$, namely, $I(t+T) = I(t)$ for all $t \geq 0$, $I'(t) > 0$ for all $t \in (0, T_1)$, and $I'(t) < 0$ for all $t \in (T_1, T)$;
- S-agents alternate between all being active and all being inactive, i.e., $a_S(t) = 1$ for all $t \in (0, T_1)$ and $a_S(t) = 0$ for all $t \in (T_1, T)$, while I-agents are always active.

Note that, because infection prevalence follows the same oscillating pattern over each period of time $[KT, (K+1)T]$, S-agent activity in an oscillating epidemic trajectory must also repeat over time, alternating between "active periods" of length T_1 from KT to $KT + T_1$ and "inactive periods" of length T_2 from $KT + T_1$ to $(K+1)T$.

Feasible oscillating trajectories. Let $\underline{I} \equiv I(0)$ denote the minimal infection prevalence, reached at each time $t = KT$ for $K = 0, 1, 2, \ldots$. Infection dynamics during each active period are determined by $\underline{I}$ and the differential equation $I'(t) = (\beta(1 - I(t)) - r)I(t)$. Let $\overline{I} \equiv I(T_1)$ denote the maximal infection prevalence, reached for the first time at $t = T_1$. Infection dynamics during each inactive period are determined by $\overline{I}$ and the differential equation $I'(t) = -rI(t)$.

By definition, $I(0) = I(T)$ in any oscillating trajectory. This constrains the period lengths T_1, T_2 and oscillation range $(\underline{I}, \overline{I})$ that can feasibly arise. T_1 and T_2 are each determined by the amount of time it takes, respectively, to rise or fall between $\underline{I}$ and $\overline{I}$. In particular, because $\frac{d\log(I(t))}{dt} = \frac{I'(t)}{I(t)}$ equals $\beta(1 - I(t)) - r$ for all $t \in (0, T_1)$ and equals $-r$ for all $t \in (T_1, T)$, we have

$$\log(\overline{I}) - \log(\underline{I}) = \int_0^{T_1} (\beta(1 - I(t)) - r)dt = T_2 r \tag{14}$$

Since $I(t) < 1 - \frac{r}{\beta}$ at all times and $r > 0$, equation (14) uniquely determines T_1 and T_2.

Let $\mathcal{E}^O$ be shorthand for a feasible oscillating trajectory. For ease of notation, we will mostly suppress $\mathcal{E}^O$-notation in what follows, except where needed for clarity.

Infected-agent welfare. Infected agents have lifetime health (or simply "health") $H_I(t) = \frac{-d}{r}$ in any trajectory, but their lifetime wealth (or simply "wealth") $W_I(t)$ varies over time and depends on the trajectory. I-agents' wealth $W_I(0)$ at the start of each oscillation is determined by the fact that they get flow economic payoff $b_1 + b_2$ during each active period, flow economic payoff $b_1 + I(t)b_2$ during each inactive period, and continuation welfare $W_I(0)$ if still alive at the start of the next oscillation. That is,

$$\begin{aligned} W_I(0) &= \int_0^{T_1} (b_1 + b_2)e^{-rx}dx + \int_{T_1}^{T} (b_1 + I(x)b_2)e^{-rx}dx + e^{-rT}W_I(0) \\ &= \frac{\int_0^{T_1} (b_1 + b_2)e^{-rx}dx + \int_{T_1}^{T} (b_1 + I(x)b_2)e^{-rx}dx}{1 - e^{-rT}} \end{aligned} \tag{15}$$

In the same way, I-agents' wealth at times $t \in (0, T)$ is determined by their remaining flow economic payoffs until time T plus continuation payoff $W_I(0)$ if still alive at that time:

$$\begin{aligned} W_I(t) &= \int_0^{T_1 - t} (b_1 + b_2) e^{-rx} dx \\ &\quad + \int_{T_1}^{T} (b_1 + I(x) b_2) e^{-r(x-t)} dx + e^{-r(T-t)} W_I(0) \text{ for all } t \in [0, T_1] \\ &= \int_t^T (b_1 + I(x) b_2) e^{-r(x-t)} dx + e^{-r(T-t)} W_I(0) \text{ for all } t \in [T_1, T] \end{aligned} \tag{16}$$

I-agents' overall individual welfare $U_I(t) = H_I(t) + W_I(t)$ at each time $t \in [0, T]$, with $U_I(t) = U_I(t - T)$ for all $t > T$.

Susceptible-agent welfare. Consider an agent who is susceptible at time $t = 0$. Such an agent has health and wealth

$$\begin{aligned} H_S(0) &= \int_0^{T_1} \beta I(x) H_I(x) P(x) e^{-rx} dx + H_S(0) P(T) e^{-rT} \\ &= \frac{\int_0^{T_1} \beta I(x) H_I(x) P(x) e^{-rx} dx}{1 - P(T) e^{-rT}} \end{aligned} \tag{17}$$

$$\begin{aligned} W_S(0) &= \int_0^{T_1} (b_1 + b_2 + W_I(x) \beta I(x)) P(x) e^{-rx} dx + W_S(0) P(T) e^{-rT} \\ &= \frac{\int_0^{T_1} (b_1 + b_2 + W_I(x) \beta I(x)) P(x) e^{-rx} dx}{1 - P(T) e^{-rT}} \end{aligned} \tag{18}$$

where $P(t)$ is the probability that such an agent remains susceptible at time t, conditional on being alive at that time. Since S-agents are infected at rate $\beta I(t)$ during each active period and never infected during each inactive period, we have $P(t) = e^{-\int_0^t \beta I(x) dx}$ for all $t \in [0, T_1]$ and $P(t) = P(T_1)$ for all $t \in [T_1, T]$.

The health and wealth of S-agents at times $t \in (0, T)$ is determined by their remaining flow payoffs and potential transition to infection until time T, plus a continuation payoff if still alive and susceptible at that time:

$$\begin{aligned} H_S(t) &= \int_0^{T_1 - t} \beta I(t + x) H_I(t + x) \frac{P(t + x)}{P(t)} e^{-rx} dx \\ &\quad + H_S(0) \frac{P(T)}{P(t)} e^{-r(T-t)} \text{ for all } t \in [0, T_1] \\ &= H_S(0) \frac{P(T)}{P(t)} e^{-r(T-t)} \text{ for all } t \in [T_1, T] \end{aligned} \tag{19}$$

$$W_S(t) = \int_0^{T_1-t} (b_1 + b_2 + W_I(t+x)\beta I(t+x))\frac{P(t+x)}{P(t)}e^{-rx}dx$$
$$+ W_S(0)\frac{P(T)}{P(t)}e^{-r(T-t)} \text{ for all } t \in [0, T_1]$$
$$= W_S(0)\frac{P(T)}{P(t)}e^{-r(T-t)} \text{ for all } t \in [T_1, T] \quad (20)$$

S-agents' overall individual welfare $U_S(t) = H_S(t) + W_S(t)$ at each time $t \in [0, T]$, with $U_S(t) = U_S(t - T)$ for all $t > T$.

Incentive-compatibility (IC) conditions. At all times $t \in (0, T_1)$, S-agents must at least weakly prefer to be active given that all other agents are active. By inequality (6), this "active-IC condition" holds if and only if

$$b_1 + b_2 \geq \beta I(t)\Delta U(t) \text{ for all } t \in [0, T_1], \quad (21)$$

where $\Delta U(t) = U_S(t) - U_I(t)$ is the harm of infection at time t. Similarly, at all times $t \in (T_1, T)$, S-agents must at least weakly prefer not to be active given that other S-agents are not active. By inequality (6), this "inactive-IC condition" holds if and only if

$$b_1 + b_2 I(t) \leq \beta I(t)\Delta U(t) \text{ for all } t \in [T_1, T]. \quad (22)$$

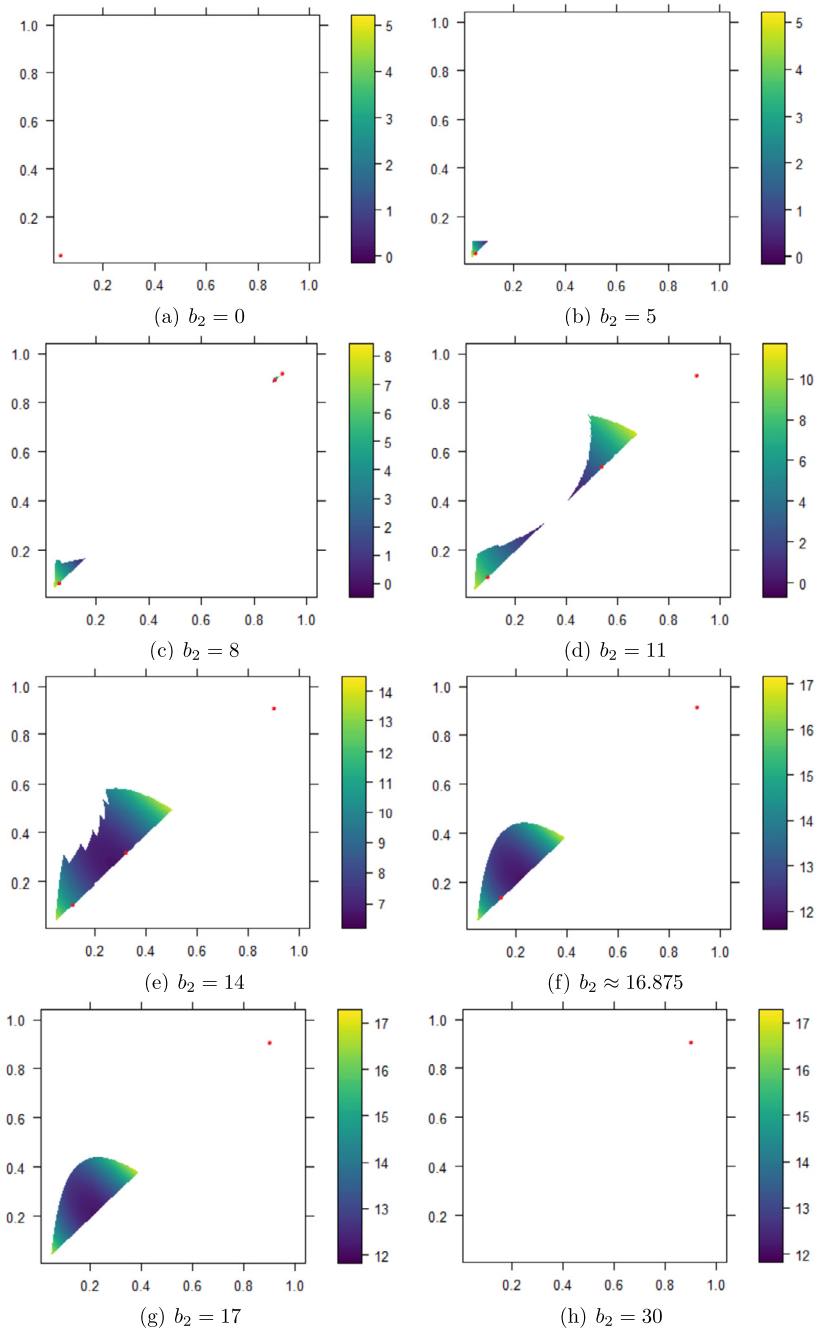
Inequalities (21),(22) allow us to compute all OETs given any model parameters.

Rise-and-oscillate epidemic trajectories. For any given OET, note that all S-agents are active at the start of the first active period at time $t = 0$. Lemma 2 therefore implies that, starting from the true initial condition in which infection is rare, an equilibrium epidemic trajectory exists in which all agents are active until the infection level reaches the oscillation's trough $\underline{I}$, after which continuation play follows the OET in question. Proposition 5 summarizes this observation that "rise-and-oscillate" equilibrium trajectories exist whenever the set of OETs is non-empty.

Definition 7. A "rise-and-oscillate trajectory" is an epidemic trajectory that consists of (i) an outbreak phase during which all agents are active, followed by (ii) an endemic phase in which the prevalence of infection oscillates over a fixed range.

Proposition 5. *In the SI model, suppose that an OET exists with infection range* $[\underline{I}, \overline{I}]$. *Then a rise-and-oscillate equilibrium trajectory exists in which that OET is played during the endemic phase.*

Numerical example. Fig. 2 illustrates how the set of OETs and the set of SSEs vary with the importance of social interactions to agent welfare, as captured by the parameter b_2, in a numerical example with other parameters $\beta = 1$, $b_0 = 0$, $b_1 = 3$, $d = 12$, and $r = 0.1$. In each panel, the horizontal and vertical axes denote, respectively, the minimal infection-level $\underline{I}$ and maximal infection-level $\overline{I}$ during each oscillation. SSEs are shown in each panel as red dots on the 45°-line, while the set of OETs is the entire colored area above the 45°-line, with colors illustrating how average newborn welfare varies over the set of OETs. (In an oscillating trajectory with period length T, "average newborn welfare" equals $\frac{\int_0^T U_S(t)dt}{T}$.)



(a) $b_2 = 0$ (b) $b_2 = 5$

(c) $b_2 = 8$ (d) $b_2 = 11$

(e) $b_2 = 14$ (f) $b_2 \approx 16.875$

(g) $b_2 = 17$ (h) $b_2 = 30$

Fig. 2. Illustration of the set of all oscillating equilibrium trajectories (OETs, colored according to average newborn welfare) and steady-state equilibria (SSEs, red dots), as b_2 varies given other parameters $\beta = 1$, $b_0 = 0$, $b_1 = 3$, $d = 12$, and $r = 0.1$. In each panel, the horizontal and vertical axes represent $\underline{I}$ and $\overline{I}$, respectively, where $[\underline{I}, \overline{I}]$ is the range of oscillation.

Steady-state equilibria: In panels (a-b), there is a unique SSE with partial activity. In panels (c-e), there are two SSE with partial activity and one with full activity. In panel (f), there is one SSE with partial activity and one with full activity. In panels (g-h), there is a unique SSE with full activity.

Oscillating equilibrium trajectories: In panel (a), there are no social benefits of activity and hence no OET exists (Proposition 2). In panel (b), there is a single connected region of OETs, fully surrounding (i.e., "containing") the unique SSE. In panels (c-d), the set of OETs is the union of two connected regions, each containing one of the two partial-activity SSEs. In panels (e-f), the set of OETs is a single connected region containing both partial-activity SSEs. In panel (g), a set of OETs exists while partial-activity SSE does not. In panel (h), there are again no OETs. □

In every OET, the active-IC constraint $b_1 + b_2 \geq \beta I(t)\Delta U(t)$ must be satisfied at all times $t \in (0, T_1)$ and the inactive-IC constraint $b_1 + b_2 I \leq \beta I(t)\Delta U(t)$ must be satisfied at all $t \in (T_1, T)$. If the harm of infection $\Delta U(t)$ were constant, then checking these constraints would be simple. Since $I(t)$ is increasing during the active period and decreasing during the inactive period, one would only need to check the active-IC constraint at time T_1, when infection is highest, and the inactive-IC constraint at time 0, when infection is lowest. However, agents' welfare and hence the harm of infection varies throughout each oscillation and, specifically, may vary non-monotonically within the active period.

Because of these complications, an analytical characterization of the set of all OETs appears out of reach. However, we have been able to characterize and fruitfully analyze a special class of OETs—those with very short oscillations, in which the prevalence of infection remains essentially constant over time.

Definition 8 *(Barely-oscillating equilibrium trajectories).* Consider any sequence of oscillating trajectories $\{\mathcal{E}_k^O : k = 1, 2, ...\}$ with $\lim_{k\to\infty} \underline{I}_k = \lim_{k\to\infty} \overline{I}_k = I$. If every trajectory in the sequence is an equilibrium trajectory, then we refer to the limit of the sequence as a "barely-oscillating equilibrium trajectory" (or "barely-OET") and I as a "barely-OET infection level." Let $\mathcal{I}^{BO}$ be the set of all barely-OET infection levels.

In Fig. 2, the colored region is the set of all OETs and the set of barely-OETs is the colored portion of the 45° line.

Consider any oscillating trajectory $\mathcal{E}^O(I)$ in which the prevalence of infection is approximately equal to $I \in (0, 1 - \frac{r}{\beta})$ at all times. In such a trajectory, the active and inactive periods are extremely short and agents' welfare remains approximately constant over time, i.e., $U_h(t; \mathcal{E}^O(I)) \approx U_h^{BO}(I)$ for all t and each $h \in \{S, I\}$. Let $\Delta U^{BO}(I) \equiv U_S^{BO}(I) - U_I^{BO}(I)$ denote the harm of infection in this "barely-oscillating limit."

The active-IC and inactive-IC conditions (21),(22) hold throughout each oscillation if $b_1 + b_2 I < \beta I \Delta U^{BO}(I) < b_1 + b_2$, but not if either $\beta I \Delta U^{BO}(I) > b_1 + b_2$ (active-IC fails) or $\beta I \Delta U^{BO}(I) < b_1 + b_2 I$ (inactive-IC fails). Lemma 4 shows that these conditions are equivalent to even simpler conditions, expressed only in terms of S-agent welfare $U_S^{BO}(I)$.

Lemma 4. *Consider any $I \in (0, 1 - \frac{r}{\beta})$. $I \in \mathcal{I}^{BO}$ if $0 < U_S^{BO}(I) < \frac{b_2}{\beta}$ but not if $U_S^{BO}(I) < 0$ or $U_S^{BO}(I) > \frac{b_2}{\beta}$, where*

$$U_S^{BO}(I) = \frac{1}{\beta}(b_1 + b_2) + I\frac{b_1 + (I + \frac{r}{\beta})b_2 - d}{r}.$$

Proof. Consider any fixed time interval $T > 0$ and any oscillating trajectory such that $\underline{I} > I - \epsilon$ and $\overline{I} < I + \epsilon$ for some $\epsilon > 0$. Define $a_S(t;T) \equiv \frac{\int_t^{t+T} a_S(t)dt}{T}$; this is S-agents' *average* activity in the T-period after time t. So long as $\epsilon \approx 0$, we have $\underline{I} \approx \overline{I} \approx I$ and there is an approximately constant flow Ir of agents out of the infected state due to death. On the other hand, the average newly-infected flow is approximately $\beta a_S(t;T) I(1-I)$. Thus, $a_S(t;T) \approx a_S^{BO}(I) \equiv \frac{r}{\beta(1-I)}$ for all t. Similarly, average overall activity $A(t;T) \approx A^{BO}(I) \equiv I + a_S^{BO}(I)(1-I) = I + \frac{r}{\beta}$ for all t.

Infected agents are always active and get approximately constant average flow payoff $b_1 + A^{BO}(I)b_2 - d$ and hence have individual welfare $U_I(t) \approx U_I^{BO}(I)$ for all t, where

$$U_I^{BO}(I) \equiv \frac{b_1 + (I + \frac{r}{\beta})b_2 - d}{r}. \tag{23}$$

Susceptible agents are active in fraction $a_S^{BO}(I)$ of the time and, at those times, all agents in the population are active. Thus, S-agents get approximately constant average flow payoff $a_S^{BO}(I)(b_1 + b_2)$ while susceptible, plus approximate continuation payoff $U_I^{BO}(I)$ in the event that they become infected. Since S-agents die at rate r and become infected at rate $\beta a_S^{BO}(I) I$, they remain susceptible for expected length of time $\frac{1}{r + \beta a_S^{BO}(I) I} = \frac{1}{\beta a_S^{BO}(I)}$ and their ex ante likelihood of becoming infected is $\frac{\beta a_S^{BO}(I) I}{r + \beta a_S^{BO}(I) I} = I$. We conclude that S-agents have individual welfare $U_S(t) \approx U_S^{BO}(I)$ for all t, where

$$U_S^{BO}(I) \equiv \frac{1}{\beta a_S} a_S(b_1 + b_2) + I U_I^{BO}(I), \tag{24}$$

confirming the equation in the statement of the lemma. The harm of infection is also approximately constant, with $\Delta U^{BO}(t) \approx \Delta U^{BO}(I) = \frac{1}{\beta}(b_1 + b_2) - (1-I)U_I^{BO}(I)$ for all t.

The oscillating trajectory in question is an equilibrium trajectory if and only if the active-IC condition (21) holds throughout each active period and the inactive-IC condition (22) holds throughout each inactive period. Because $I(t)\Delta U(t) \approx I \Delta U^{BO}(I)$ for all t, this is true (for ϵ sufficiently small) whenever these inequalities are strictly satisfied in the barely-oscillating limit, i.e., whenever

$$b_1 + b_2 > \beta I \Delta U^{BO}(I) \tag{25}$$

$$b_1 + b_2 I < \beta I \Delta U^{BO}(I) \tag{26}$$

and not true whenever either is strictly violated in the limit. Next, observe that

$$\begin{aligned} b_1 + b_2 - \beta I \Delta U^{BO}(I) &= b_1 + b_2 - \beta I \left(\frac{1}{\beta}(b_1 + b_2) - (1-I) U_I^{BO}(I) \right) \\ &= (1-I) \left(b_1 + b_2 + \beta I U_I^{BO}(I) \right) = \beta (1-I) U_S^{BO}(I) \end{aligned}$$

Since $I > 0$, the strict active-IC condition (25) is equivalent to $U_S^{BO}(I) > 0$, while the strict inactive-IC condition (26) is equivalent to $U_S^{BO}(I) < \frac{b_2}{\beta}$. We conclude as desired that every oscillating trajectory with range of infection $[\underline{I}, \overline{I}] \subset [I - \epsilon, I + \epsilon]$ is an equilibrium trajectory (and hence $I \in \mathcal{I}^{BO}$) if S-agent welfare $0 < U_S^{BO}(I) < \frac{b_2}{\beta}$, but not if either $U_S^{BO}(I) < 0$ or $U_S^{BO}(I) > \frac{b_2}{\beta}$. $\square$

Next, we leverage Lemma 4 to establish several facts about the range of outcomes that can arise in barely-OETs. First, the set of barely-OET infection levels is either empty (as in panels (a,h) of Fig. 2), a single interval (as in panels (b,e,f,g)), or the union of two intervals (as in panels (c,d)). Second, whenever a partial-activity SSE exists with infection level I, that SSE is "surrounded" by a set of OETs, including some with more infection and some with less. Moreover, in this case, OETs always exist that Pareto dominate the SSE.

Proposition 6. *(i)* $\mathcal{I}^{BO} \subset (0, 1 - \frac{r}{\beta}]$ *is either empty, a single closed interval, or the union of two closed intervals. (ii) If a partial-activity SSE exists with infection-level* I*, then* $(I - \epsilon, I + \epsilon) \subset \mathcal{I}^{BO}$ *for some* $\epsilon > 0$*. (iii) Every partial-activity SSE is Pareto dominated by a non-empty open set of OETs. (iv)* $\max_{I \in \mathcal{I}^{BO}} U_S^{BO}(I) = U_S^{BO}(I^{min})$*, where* $I^{min} \equiv \min \mathcal{I}^{BO}$.

Proof. *(i)* By the proof of Lemma 4, $I \in \mathcal{I}^{BO}$ if $U_S^{BO}(I) \in (0, \frac{b_2}{\beta})$ but not if $U_S^{BO}(I) < 0$ or $U_S^{BO}(I) > \frac{b_2}{\beta}$. By inspection of (24), $U_S^{BO}(I)$ is a strictly convex continuous quadratic with $U_S^{BO}(0) > \frac{b_2}{\beta}$, as shown in Fig. 3. Thus, the set of infection levels satisfying the inactive-IC condition is a (potentially empty[9]) interval, while the set of infection levels violating the active-IC condition is a (potentially empty) interval within that interval. This implies immediately that *either* $\mathcal{I}^{BO} = \emptyset$ *or* $\mathcal{I}^{BO}$ is an interval, with interior (I^{min}, I^{max}) for some $0 < I^{min} < I^{max} \leq 1 - \frac{r}{\beta}$, *or* $\mathcal{I}^{BO}$ is the union of two intervals, with interior $(I^{min}, I') \cup (I'', I^{max})$ for some $0 < I^{min} < I' < I'' < I^{max} \leq 1 - \frac{r}{\beta}$. In addition, whenever $\mathcal{I}^{BO}$ is non-empty, a continuity argument (provided in Appendix A.1) establishes that the threshold infection levels $\{I^{min}, I', I'', I^{max}\}$ also belong to $\mathcal{I}^{BO}$, making it a closed set.

(ii) Suppose that a partial-activity SSE exists with infection level I. Let $a_S^{SS}(I) = \frac{r}{\beta(1-I)} < 1$ be the probability that S-agents are active, and let $A^{SS}(I) = I + a_S^{SS}(I)(1 - I) = I + \frac{r}{\beta}$ be the overall activity. For the same infection level to be maintained over time, average activity must be the same in barely-oscillating limit as in the steady state: $a_S^{BO}(I) = a_S^{SS}(I) \equiv a_S(I)$ and $A^{BO}(I) = A^{SS}(I) \equiv A(I)$. Since I-agents are always active themselves, they enjoy the same average amount of social activity and hence have the same lifetime wealth and hence welfare in the barely-oscillating limit: $U_I^{BO}(I) = U_I^{SS}(I)$. Similarly, S-agents are infected at the same average rate and hence have the same lifetime health: $H_S^{BO}(I) = H_S^{SS}(I)$. However, S-agents earn average flow economic payoff $a_S(I)(b_1 + b_2)$ in the barely-oscillating limit while they remain alive and susceptible, compared to $a_S(I)(b_1 + A(I)b_2)$ in the steady state. The difference between these flows, $a_S(I)(1 - A(I))b_2$, arises from all S-agents being active at the same time and hence maximizing the social benefit of their activity.

Because S-agents remain alive and susceptible on average for length of time $\frac{1}{r+\beta a_S(I)I} = \frac{1}{\beta a_S(I)}$ and $A(I) = I + \frac{r}{\beta}$, we have $U_S^{BO}(I) = U_S^{SS}(I) + (1 - I - \frac{r}{\beta})\frac{b_2}{\beta}$. In the SSE with infection-level I, agents must be indifferent whether to be active, i.e., $U_S^{SS}(I) = 0$ (proof of Proposition 1). We conclude that $U_S^{BO}(I) = (1 - I - \frac{r}{\beta})\frac{b_2}{\beta} \in (0, \frac{b_2}{\beta})$ and hence that $I \in \mathcal{I}^{BO}$. Moreover, because $U_S^{BO}(I)$ is continuous in I, we have immediately that $(I - \epsilon, I + \epsilon) \subset \mathcal{I}^{BO}$ for small enough $\epsilon > 0$, as desired.

[9] In any barely-OET, S-agents must find it optimal to be inactive when everyone else is inactive, a condition which cannot hold when disease severity d is sufficiently small and/or the non-social benefit of activity b_1 is sufficiently high. See Appendix A.2 for a complete characterization of the model parameters given which $\mathcal{I}^{BO}$ is empty.

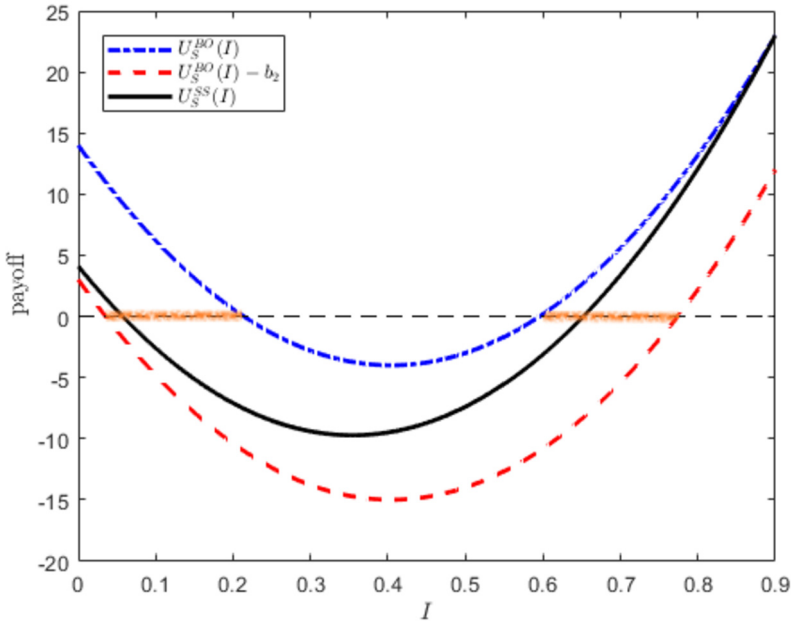


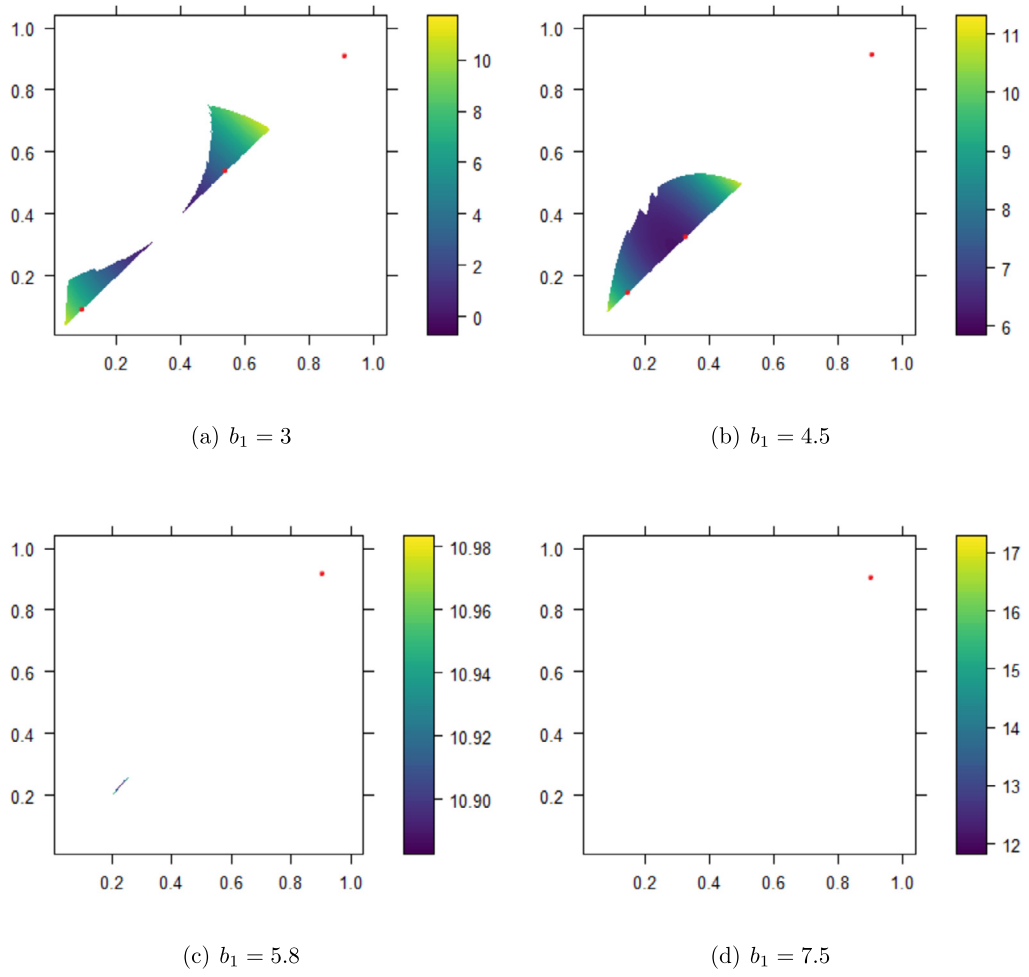
Fig. 3. An example in which $\mathcal{I}^{BO}$ consists of two disjoint intervals, given parameters $\beta = 1, b_1 = 3, b_2 = 11, d = 13, r = 0.1$.

(iii) Suppose that a partial-activity SSE exists with infection level I. Consider an oscillating trajectory $\mathcal{E}^O(\epsilon)$ with $\underline{I} \in (I, I + \epsilon)$ and $\overline{I} \in (\underline{I}, \underline{I} + \epsilon)$. By part (ii), $\mathcal{E}^O(\epsilon)$ is an equilibrium trajectory for all sufficiently small ϵ. $\mathcal{E}^O(\epsilon)$ Pareto dominates the partial-activity SSE because S-agents get positive individual welfare in $\mathcal{E}^O(\epsilon)$ compared to zero individual welfare in the SSE, while I-agents are strictly better off because there is more overall activity than in the SSE.[10]

(iv) For all $I \in \mathcal{I}^{BO}$, newborns have individual welfare $U_S^{BO}(I)$ in the barely-OET with infection-level I. By Lemma 4, $U_S^{BO}(I)$ is bounded above by $\frac{b_2}{\beta}$, and this upper bound is realized whenever the inactive-IC constraint is binding. Because $U_S^{BO}(I)$ is a continuous and strictly convex quadratic with $U_S^{BO}(0) > \frac{b_2}{\beta}$, this occurs at I^{min}, as desired. (If $I^{max} < 1 - \frac{r}{\beta}$, as in all of our numerical examples, then the inactive-IC constraint also binds at I^{max}. In that case, newborn welfare is also maximized at the highest barely-OET infection level.) □

Fig. 4 illustrates some of the key findings in Proposition 6. Panels (a-b) show two situations with two partial-activity SSEs, each contained within an interval of barely-OETs. (In panel (a), the set $\mathcal{I}^{BO}$ of barely-OETs consists of two intervals; in panel (b), it is a single interval.) Each of these partial-activity SSEs generates less welfare for newborns than the barely-OET with the same infection level, which themselves generate less newborn welfare than the barely-OETs with the least or the most amount of infection. Finally, consider panel (c). Proposition 6 establishes

[10] More precisely, let $A(t; \epsilon, T)$ denote the average overall activity in the T-period after time t, for any $T > 0$. For sufficiently small ϵ, $A(t; T)$ strictly exceeds the overall activity in the SSE at all times $t \geq 0$. Thus, I-agents accumulate strictly more economic payoffs over any given T-period when ϵ is sufficiently small.



(a) $b_1 = 3$ (b) $b_1 = 4.5$

(c) $b_1 = 5.8$ (d) $b_1 = 7.5$

Fig. 4. Illustration of all oscillating equilibrium trajectories (OETs) and steady-state equilibria (SSEs) as in Fig. 2, but now with varying b_1 and $\beta = 1$, $b_2 = 11$, $d = 12$, and $r = 0.1$.

that an interval of barely-OETs exists whenever a partial-activity SSE exists. Panel (c) shows that the converse is not true, as there is an interval of barely-OETs which does not contain any partial-activity SSE.

Rise-and-plateau barely-OETs. Because Proposition 5 applies to all OETs, an immediate implication is that for all infection levels $I \in \mathcal{I}^{BO}$, equilibrium epidemic trajectories exist in which all agents are active until the infection level reaches I, after which continuation play follows an OET that barely oscillates. These equilibrium trajectories are similar to the rise-and-plateau equilibrium trajectories of Proposition 4, in that infection prevalence is essentially constant during the endemic phase of the epidemic, but the long-run prevalence of infection (and agent welfare) can be very different. For instance, in the numerical example illustrated in Fig. 4(c), the prevalence of infection during the endemic phase is only about 20% of the population infected if agents are

able to coordinate on a barely-OET, compared to 90% infected if they settle into the unique SSE which has full activity.

4. Susceptible-infected-removed analysis

This section extends our analysis to the Susceptible-Infected-Removed (SIR) model,[11] in which infected agents are "removed" due to death at rate $\gamma > 0$. In summarizing our findings in the SIR model, it is helpful to discuss separately what we have been able to show about the endemic versus early phases of the epidemic.

Endemic phase: Our characterization of steady-state equilibria (SSEs) and barely-oscillating equilibrium trajectories (barely-OETs) in Sections 3.1-3.2 extends naturally from the SI model to the SIR model; see Propositions 9 and 10, which directly generalize Propositions 3 and 6. In particular, whenever disease symptoms are sufficiently severe, we show that (i) there is a unique SSE and that (ii) barely-OETs exist that welfare dominate that SSE.

Early-epidemic phase(s): The early phases of an equilibrium epidemic are more difficult to analyze in the SIR model, for two main reasons. *First,* for some parameter values, equilibrium trajectories with a simple rise-and-plateau or rise-and-oscillate structure cannot exist. For instance, consider an "apocalyptic epidemic" in which a novel infectious disease emerges that is very transmissible and very deadly. Uninfected agents may have a strong incentive to socially distance early during the epidemic while infection is raging but later on, when the population is dramatically reduced, survivors may be unlikely to encounter anyone at all and hence have an incentive to be fully active.[12] In apocalyptic epidemics absent any vaccine or treatment, the prevalence of infection must rise initially but then eventually fall to a very low level in any equilibrium trajectory, a pattern that does not arise in the rise-and-plateau and rise-and-oscillate equilibrium trajectories that have been our focus in this paper. Characterizing the more complex dynamics that can arise during an apocalyptic epidemic is beyond the scope of the present analysis but certainly worthy of future study.

Second, during the outbreak phase of an SIR epidemic as the mass of infection I increases, the rise in the mass of removed agents R lags the rise in infections. When an infection level I is first reached, R will therefore be relatively low and the mass of uninfecteds S will be relatively high compared to the steady state with the same amount of infection. So long as there are economic complementarities of social activity, the social-economic flow payoffs that agents earn (and their lifetime welfare) during the outbreak phase can therefore be substantially different than in the steady state with the same amount of infection. This in turn impacts agents' incentives, complicating whether or not the incentive-compatibility conditions for a rise-and-plateau or rise-and-oscillate equilibrium trajectory can be satisfied at the moment of transition between the outbreak phase and the endemic phase.

[11] The first version of this paper (McAdams (2020)) considers the Susceptible-Carriage-Infected-Recovered (SCIR) model, also allowing for asymptomatic infection, and provides an algorithm that implicitly characterizes all equilibrium epidemic trajectories in that context. We focus on the SI and SIR models here because the novel aspects of our analysis can be illustrated more clearly in these simpler disease models.

[12] The zombie apocalypse depicted in the hit TV show *The Walking Dead* illustrates this point. During the first several seasons of the show, zombies are everywhere and the main characters hole up in an abandoned prison to avoid exposure. But later after the first wave of zombies has mostly died off and the epidemic has entered its endemic phase, the main characters travel and forage with little fear of being infected.

In the SI model for any sufficiently severe disease, we showed how this transition can always be accomplished for any given barely-OET, allowing us to construct a rise-and-plateau equilibrium trajectory in which the outbreak phase is followed immediately by that barely-OET. However, this is not always true in the SIR model. As we show in a numerical example below, there are model parameters given which a barely-OET exists but no corresponding rise-and-plateau equilibrium exists.[13]

Equilibrium existence. Proposition 7 establishes that an equilibrium trajectory always exists in the SIR model. In those cases when rise-and-plateau equilibria do not exist, any equilibrium epidemic must therefore have a non-trivial *intermediate phase* after S-agents have begun distancing but before the epidemic has "settled down" into a long-run steady state or long-run oscillation. Characterizing what such intermediate phases could look like is of interest, but beyond the scope of this paper.

Proposition 7. *An equilibrium epidemic trajectory exists from any initial condition.*

Proof. The proof is in Appendix B.1. □

Sufficient conditions for uniqueness. Proposition 8 establishes that there is a unique equilibrium trajectory—and that this equilibrium follows a rise-and-plateau trajectory—so long as (i) there are no social benefits of activity, i.e., $b_2 = 0$, and (ii) disease symptoms are sufficiently severe, i.e., d is sufficiently large. Two infection-level thresholds play an important role in this analysis. First, let $\tilde{I}$ and $\tilde{R}$ be the long-run prevalence of infected and removed agents in the trajectory in which all agents are always active, implicitly defined by the steady-state conditions $\frac{I'(t)}{I(t)} = \beta(1-\tilde{I}-\tilde{R}) - (r+\gamma) = 0$ and $\frac{R'(t)}{R(t)} = \frac{\gamma \tilde{I}}{\tilde{R}} - r = 0$. In particular:

$$\tilde{I} \equiv \frac{r}{r+\gamma} - \frac{r}{\beta} \tag{27}$$

and $\tilde{R} = \frac{\gamma}{r}\tilde{I}$. Next, define I^* implicitly by the condition

$$b_1 - \beta I^* \left(\frac{b_0}{r} - \frac{b_0 + b_1 - d}{r+\gamma} \right) = 0. \tag{28}$$

Note that I^* is strictly decreasing in d and thus $I^* \leq \tilde{I}$ if and only if $d \geq \underline{d}(\gamma)$, where $\underline{d}(\gamma)$ is the threshold given which $I^* = \tilde{I}$.[14]

Proposition 8. *In the SIR model, suppose that $b_2 = 0$ and that $I^* \leq \tilde{I}$. Then there is a unique equilibrium trajectory, in which (i) $a_S(t) = 1$ until the first time t^* at which $I(t^*) = I^*$ and (ii) $I(t) = I^*$ for all $t \geq t^*$.*[15]

[13] On the other hand, this numerical example also shows that there is a wide range of parameters given which such rise-and-plateau equilibria do exist.

[14] As it turns out, this threshold is also the threshold for a partial-activity SSE to exist; see Proposition 9.

[15] To avoid confusion, please note that this equilibrium trajectory differs from the steady-state trajectory with infection level I^*. In particular, the mass of removed agents increases throughout the "plateau phase," eventually converging to its steady-state level, i.e., $\lim_{t\to\infty} R(t) = \frac{\gamma}{r} I^*$. In the same way, S-agent activity $a_S(t)$ increases over time toward its steady-state level.

Proof. *Preliminaries.* I-agent welfare: infected agents get flow payoff $b_0 + b_1 - d$ for expected duration $\frac{1}{r+\gamma}$; so, $U_I(t) = \frac{b_0+b_1-d}{r+\gamma}$ for all t. Lower bound on S-agent welfare: A susceptible agent who remains inactive earns flow payoff b_0 for expected duration $\frac{1}{r}$; so, $U_S(t) \geq \frac{b_0}{r}$ for all t and $U_S(t) = \frac{b_0}{r}$ if and only if S-agents weakly prefer to be inactive at all times $t' \geq t$. S-agent incentives and welfare dynamics: Define

$$X(t) \equiv b_1 - \beta I(t) \left(U_S(t) - \frac{b_0 + b_1 - d}{r + \gamma} \right). \tag{29}$$

At each time t, S-agents strictly prefer activity if $X(t) > 0$, strictly prefer inactivity if $X(t) < 0$, and are indifferent whether to be active if $X(t) = 0$. Moreover,

$$\begin{aligned} U_S'(t) &= rU_S(t) - b_0 - a_S(t) \left(b_1 - \beta I(t) \left(U_S(t) - U_I(t) \right) \right) \\ &= rU_S(t) - b_0 - a_S(t) X(t) \end{aligned} \tag{30}$$

Step 1: $I(t) \leq I^*$ for all t. Suppose that $I(t) > I^*$ for some t. Because $U_S(t) \geq \frac{b_0}{r}$ for all t and $b_1 + \beta I^* \left(\frac{b_0}{r} - \frac{b_0+b_1-d}{r+\gamma} \right) = 0$ by definition of I^*, $X(t) < 0$ and S-agents strictly prefer to be inactive. We conclude that $I'(t) < 0$ whenever $I(t) > I^*$ and hence that $I(t)$ can never exceed I^*, as desired.

Step 2: If $I(t) = I^*$, then $I(t') = I^*$ for all $t' \geq t$ and $U_S(t) = \frac{b_0}{r}$. Suppose that $I(t') = I^*$ and, without loss, suppose that this is the first time that the threshold I^* has been reached, i.e., $I(t) < I^*$ for all $t < t'$. By Step 1, $X(t') \leq 0$ since infections cannot rise above I^*. On the other hand, because infections were increasing just before t', S-agents must have at least weakly preferred to be active; so, $X(t') \geq 0$ and hence $X(t') = 0$. By the definition of I^* and equation (29), we have $U_S(t') = \frac{b_0}{r}$, then same as if $I(t)$ were to remain equal to I^* forever after t'. Now, suppose for sake of contradiction that $I(t)$ did not remain equal to I^* forever. $I(t)$ cannot increase by Step 1, so the only remaining possibility is that the trajectory sometimes falls below I^*. But an optimizing S-agent is always strictly better off when facing a trajectory where $I(t)$ is everywhere lower; so, it must be that $U_S(t') > \frac{b_0}{r}$, a contradiction.

Step 3: If $I(t) < I^*$, then $X(t) > 0$ and $U_S(t) > \frac{b_0}{r}$. Suppose that $I(t') < I^*$. A susceptible agent who is fully active after t' so long as $I(t) < I^*$ and fully inactive when $I(t) = I^*$ earns lifetime welfare strictly greater than $\frac{b_0}{r}$; so, it must be that $U_S(t') > \frac{b_0}{r}$.[16] This in turn implies that S-agents must strictly prefer to be active at some times after t'. Let t'' be the first time after t' at which S-agents begin to strictly prefer activity, i.e., (i) $X(t) \leq 0$ for all $t \in (t', t'')$ and (ii) $X(t) > 0$ for all $t \in (t'', t'' + \epsilon)$ for all small enough ϵ. We need to show that $t'' = t'$, since then S-agents must strictly prefer to be active at time t'.

Suppose for the sake of contradiction that $t'' > t'$. S-agents find it weakly optimal to be inactive during $[t', t'']$, but it is still possible that infection prevalence may rise during this period. However, it must be that $I(t'') < I^*$. To see why, note that $U_S'(t) = rU_S(t) - b_0$ so long as S-agents find it optimal to be inactive. Since $U_S(t') > \frac{b_0}{r}$, this implies $U_S'(t) > 0$ and hence $U_S(t'') > U_S(t') > \frac{b_0}{r}$; and we showed in Step 2 that $U_S(t) > \frac{b_0}{r}$ is only possible when $I(t) < I^*$.

[16] This point can also be made via proof by contradiction. Suppose that $U_S(t') = \frac{b_0}{r}$. Then $I(t') < I^*$ implies $X(t') > 0$ by (29), which implies $a_S(t') = 1$ because S-agents strictly prefer activity, which implies $U_S'(t') < 0$ by (30), a contradiction since $U_S(t)$ can never fall below $\frac{b_0}{r}$.

By continuity of $X(t)$, S-agents must be indifferent whether to be active at time t''. Thus, $\lim_{t\searrow t''} X(t) = 0$ and hence $\lim_{t\searrow t''} U_S'(t) = rU_S(t'') - b_0 > 0$. Moreover, because all S-agents are active immediately after t'', we have $\lim_{t\searrow t''} I'(t) > 0$. But then $\lim_{t\searrow t''} X'(t) < 0$ by equation (29), contradicting the presumption that $X(t) > 0$ immediately after t''. We conclude that $t' = t''$ as desired. □

Steady-state equilibria (SSE). As in the SI model, steady states with S-agent activity $a_S \leq \frac{r+\gamma}{\beta}$ cannot arise in equilibrium, since such low activity would drive infection prevalence to zero and S-agents strictly prefer to be active whenever infection is sufficiently rare; so, we may restrict attention to activity levels $a_S \in \left(\frac{r+\gamma}{\beta}, 1\right]$. What about infection levels? With full activity, the steady-state conditions $\beta a_S S = r + \gamma$ (see equation (2)) and $R = \frac{\gamma}{r} I$ (see equation (4)) imply that $I = \frac{r}{r+\gamma} - \frac{r}{\beta}$; we may therefore restrict attention to infection levels $I \in \left(0, \frac{r}{r+\gamma} - \frac{r}{\beta}\right]$.

Proposition 9 extends our key finding about the set of SSE to the SIR context.

Proposition 9. *There is a symptom-severity threshold $\underline{d}(\gamma)$ such that (i) the full-activity steady state is an SSE if and only if $d \leq \underline{d}(\gamma)$ and (ii) there is a unique SSE whenever $d > \underline{d}(\gamma)$ and, in this SSE, S-agents are partially active and newborn agents have the same lifetime welfare as if compelled to remain inactive for their entire lives. In particular:*

$$\underline{d}(\gamma) = \left(1 + \frac{r+\gamma}{r}\frac{r+\gamma}{\beta - r - \gamma}\right)\left(b_1 + b_2\left(\frac{r}{r+\gamma} + \frac{\gamma}{\beta}\right)\right) - \frac{\gamma}{r}b_0. \tag{31}$$

Proof. *Part (i).* Consider first the steady state with full activity ($a_S = 1$), infection level $I = \frac{r}{r+\gamma} - \frac{r}{\beta}$, and population-wide activity $A = S + I = 1 - R$. Infected agents get flow payoff $b_0 + b_1 + (1-R)b_2 - d$ until death, which occurs at rate $r + \gamma$; so, I-agents' steady-state welfare $U_I^{SS}(I) = \frac{b_0+b_1+(1-R)b_2-d}{r+\gamma}$. Susceptible agents get flow payoff $b_0 + b_1 + (1-R)b_2$ until death (rate r) or infection (rate βI). This susceptible period lasts on average for length of time $\frac{1}{r+\beta I}$, with infection occurring before death with ex ante likelihood $\frac{\beta I}{r+\beta I}$; so, S-agents' steady-state welfare $U_S^{SS}(I) = \frac{b_0+b_1+(1-R)b_2}{r+\beta I} + \frac{\beta I}{r+\beta I}U_I^{SS}(I)$. The harm of infection $\Delta U^{SS}(I) = U_S^{SS}(I) - U_I^{SS}(I) = \frac{b_0+b_1+(1-R)b_2}{r+\beta I} - \frac{r}{r+\beta I} \times \frac{b_0+b_1+(1-R)b_2-d}{r+\gamma}$. Collecting terms and leveraging the fact that $I = \frac{r}{r+\gamma} - \frac{r}{\beta}$ and hence that $r + \beta I = \frac{\beta r}{r+\gamma}$, this equation reduces to

$$\begin{aligned}\Delta U^{SS}(I) &= \frac{b_0 + b_1 + (1-R)b_2}{r+\beta I}\left(1 - \frac{r}{r+\gamma}\right) + \frac{r}{r+\beta I} \times \frac{d}{r+\gamma} \\ &= \frac{(b_0 + b_1 + (1-R)b_2)\gamma}{\beta r} + \frac{d}{\beta}\end{aligned} \tag{32}$$

By inequality (6), S-agents find it individually optimal to be active if $b_1 + (1-R)b_2 \geq \beta I \Delta U^{SS}(I)$. By (32), this inequality holds if and only if d is sufficiently small that

$$d \leq \frac{b_1 + (1-R)b_2}{I} - \frac{(b_0 + b_1 + (1-R)b_2)\gamma}{r} \tag{33}$$

Since $I = \frac{r}{r+\gamma} - \frac{r}{\beta}$ and $R = \frac{\gamma}{r+\gamma} - \frac{\gamma}{\beta}$, this inequality can be rewritten as

$$d \leq \underline{d}(\gamma) \equiv \left(\frac{1}{\frac{r}{r+\gamma} - \frac{r}{\beta}} - \frac{\gamma}{r}\right)\left(b_1 + \left(\frac{r}{r+\gamma} + \frac{\gamma}{\beta}\right)b_2\right) - \frac{\gamma}{r}b_0 \tag{34}$$

which in turn can be written more simply as in (31). We conclude that the full-activity steady state is an ESS if and only if $d \leq \underline{d}(\gamma)$, as desired.

Part (ii). Suppose that $d > \underline{d}(\gamma)$. By part (i), the steady state with $a_S = 1$ and $I = \frac{r}{r+\gamma} - \frac{r}{\beta}$ is not an SSE. We need to show that exactly one partial-activity steady state is an SSE. Recall from the proof of Lemma 1 that a partial-activity steady state is an SSE if and only if S-agents are indifferent whether to be active.

An S-agent who is inactive earns flow payoff b_0, dies at rate r, and does not become infected. An S-agent who is active earns flow payoff $b_0 + b_1 + b_2 A$, dies at rate r, and becomes infected at rate βI. Accounting for the harm of becoming infected while active, S-agents are indifferent whether to active if $b_0 = b_0 + b_1 + b_2 A - \beta I(U_S^{SS}(I) - U_I^{SS}(I))$, where $A = I + a_S S$. The indifference condition characterizing the set of ESSs is therefore

$$b_1 + b_2(I + a_S S) - \beta I(U_S^{SS}(I) - U_I^{SS}(I)) = 0 \tag{35}$$

If the partial-activity steady state in question is an ESS, $U_S^{SS}(I) = \frac{b_0}{r}$ by Lemma 1. Infected agents get flow payoff $b_0 + b_1 + Ab_2 - d$ and die at rate $r + \gamma$; so, $U_I^{SS}(I) = \frac{b_0 + b_1 + Ab_2 - d}{r+\gamma}$. Finally, by equation (3) and the steady-state condition $I'(t)/I(t) = 0$, we have $\beta a_S S = r + \gamma$ and hence $A = I + \frac{r+\gamma}{\beta}$. Indifference condition (35) can therefore be re-written as

$$X(I, d) \equiv \left(1 + \frac{\beta I}{r+\gamma}\right)\left(b_1 + b_2\left(I + \frac{r+\gamma}{\beta}\right)\right) - \frac{\beta I d}{r+\gamma} - \frac{\beta I \gamma b_0}{r(r+\gamma)} = 0. \tag{36}$$

Existence and uniqueness of a partial-activity steady state follows from three simple observations about the expression $X(I, d)$: (a) $\lim_{I \to 0} X(I, d) > 0$ for any $d > 0$; (b) $X\left(\overline{I}, d\right) < 0$ for all $d > \underline{d}(\gamma)$, where $\overline{I} \equiv \frac{r}{r+\gamma} - \frac{r}{\beta}$ is the infection level in the full-activity steady state and hence a strict upper bound on I in any partial-activity steady state; and (c) $X(I, d)$ is quadratic and convex in I for any given d. Together, observations (a-c) imply that, for all $d > \underline{d}(\gamma)$ there is exactly one infection level $I(d) \in (0, \overline{I})$ such that $X(I(d), d) = 0$.

Observations (a) and (c) are immediate from (36). To verify (b), note that $X\left(\overline{I}, \underline{d}(\gamma)\right) = 0$ by our construction of $\underline{d}(\gamma)$ in the proof of part (i). The fact that $X\left(\overline{I}, d\right) < 0$ for all $d > \underline{d}(\gamma)$ follows immediately from the fact that $X(I, d)$ is decreasing in d. □

Oscillating equilibrium trajectories (OETs). Epidemiological dynamics are more complex in the SIR model, since the mass of living agents $S(t) + I(t) = 1 - R(t)$ also changes over time. However, we can define "oscillating trajectories" much as in the SI model with alternating active and inactive periods; details in Appendix B.2. An oscillating trajectory is an equilibrium trajectory if and only if (i) $b_1 + b_2(1 - R(t)) \geq \beta I(t) \Delta U(t)$ during each active period ("active-IC condition," generalizing (21)) and (ii) $b_1 + b_2 I(t) \leq \beta I(t) \Delta U(t)$ during each inactive period ("inactive-IC condition," generalizing (22)), where $\Delta U(t) = U_S(t) - U_I(t)$ is the harm of infection at time t.

As in the SI model, the full set of OETs can only be characterized numerically, but clean analytical results are available if we focus on the limiting case of "barely-oscillating equilibrium trajectories (barely-OETs)" in which each oscillation period is infinitesimal and the level of infection remains constant over time.

In any barely-OET with infection level I, the mass of removed agents must converge in the long run to $R^\infty = \frac{\gamma}{r} I$. Following the notation in Section 3.2, let $\mathcal{I}^{BO}$ denote the range of infection-levels that can arise in a barely-OET in which the mass of removed agents has already

reached its steady-state level at time $t = 0$.[17] Lemma 5 (proven in Appendix B.3) is a direct extension of Lemma 4.

Lemma 5. *Consider any* $I \in \left(0, \frac{r}{r+\gamma} - \frac{r}{\beta}\right)$. $I \in \mathcal{I}^{BO}$ *if* $\frac{b_0}{r} < U_S^{BO}(I) < \frac{b_0}{r} + \frac{r+\gamma}{\beta r} b_2$ *but not if* $U_S^{BO}(I) < \frac{b_0}{r}$ *or* $U_S^{BO}(I) > \frac{b_0}{r} + \frac{r+\gamma}{\beta r} b_2$, *where*

$$U_S^{BO}(I) = \frac{1 - \frac{\gamma}{r} I}{r} b_0 + \frac{r+\gamma}{\beta r} \left(b_1 + b_2 \left(1 - \frac{\gamma}{r} I\right)\right) + I \frac{b_1 + \left(I + \frac{r+\gamma}{\beta}\right) b_2 - d}{r}.$$

The proof of Lemma 5 is essentially identical to that of Lemma 4, but with updated algebra accounting for the fact that (i) infected agents die at rate $r + \gamma$ rather than rate r and (ii) all living agents earn baseline flow payoff $b_0 \geq 0$.[18] All of our other key findings about barely-OETs in the SI model also extend to the SIR model. In particular, Proposition 10 (proven in Appendix B.4) directly extends Proposition 6.

Proposition 10. *(i)* $\mathcal{I}^{BO} \subset (0, \frac{r}{r+\gamma} - \frac{r}{\beta}]$ *is either empty, a single closed interval, or the union of two closed intervals. (ii) If a partial-activity SSE exists with infection-level* I, *then* $(I - \epsilon, I + \epsilon) \subset \mathcal{I}^{BO}$ *for some* $\epsilon > 0$. *(iii) Every partial-activity SSE is welfare dominated by a non-empty open set of OETs. (iv)* $\max_{I \in \mathcal{I}^{BO}} U_S^{BO}(I) = U_S^{BO}(I^{min})$, *where* $I^{min} \equiv \min \mathcal{I}^{BO}$.

Early-epidemic dynamics. In this paper, we focus on the range of *endemic* epidemic outcomes that can arise in equilibrium, after the disease is well-established and the population as a whole has settled into a steady state or an oscillating trajectory. Of course, the beginning phases of the epidemic are also of interest. In the SI model, we showed by construction that for any sufficiently severe disease, equilibrium epidemic trajectories exist with an especially simple "rise-and-plateau" structure, in which the infection level rises during an outbreak phase until a threshold level I is reached, after which continuation play follows a partial-activity SSE or barely-OET with constant infection level I and constant S-agent activity level. Such rise-and-plateau equilibrium trajectories do not always exist in the SIR model.

Numerical examples. Fig. 5 illustrates the qualitative features of rise-and-plateau equilibrium trajectories in the SIR model in a numerical example with parameters $\gamma = 0.08$, $r = 0.1$, $\beta = 1$, $d = 5$, $b_0 = 1$, $b_1 = 1$, and $b_2 = 5$. During the initial *outbreak phase*, all agents are active until a time is reached (vertical dashed line) at which infection prevalence hits a target level I^∞, here 0.20. The epidemic then transitions directly to an *endemic phase* in which the mass of infected agents remains equal to I^∞. Few people have died from the disease when the epidemic enters its "plateau phase," but the mass of removed agents (blue line) gradually increases over time toward its long-run level $R^\infty = \frac{\gamma}{r} I^\infty = 0.16$. As $R(t)$ increases and $I(t)$ remains constant, the mass of susceptible agents falls and S-agent activity must increase in order to keep infections constant.

[17] If $R(0) < R^\infty$ at the start of a barely-oscillating trajectory, then there will be more S-agents and these S-agents will need to be active less often initially, compared to the long run. We explore the implications of this complication later in a numerical example.

[18] In the SI model, introducing $b_0 > 0$ shifts up all agents' welfare by $\frac{b_0}{r}$ but has no effect on agents' incentives and hence no effect on the equilibrium set. Once people can die from the disease, however, b_0 impacts susceptible agents' incentive to avoid infection. It is therefore without loss to focus on the case with $b_0 = 0$ in the SI model, but not in the SIR model.

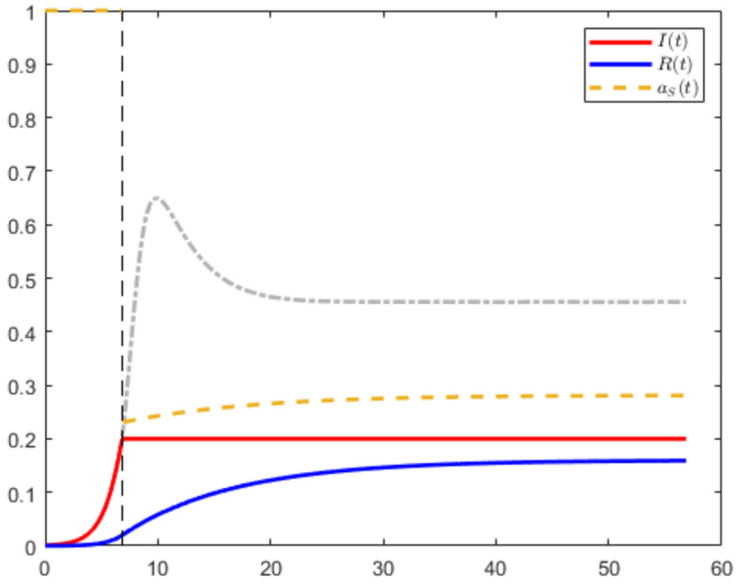


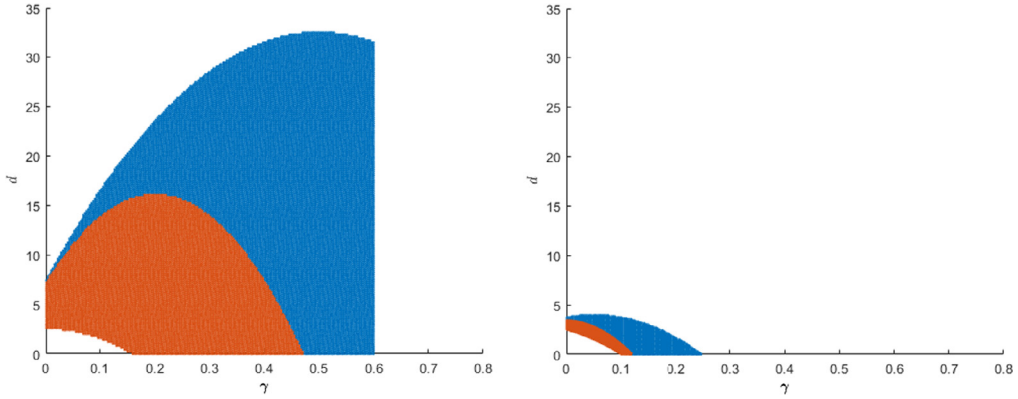
Fig. 5. A rise-and-plateau equilibrium trajectory in the SIR model with parameters $(\gamma, r, \beta, d, b_0, b_1, b_2) = (0.08, 0.1, 1, 5, 1, 1, 5)$ and plateau infection prevalence $I^\infty = 0.20$.

This can be seen in the dashed yellow line, as average S-agent activity $a_S(t)$ increases from 0.23 to its long-run level of about 0.28.[19] Absent any distancing, the mass of infections would follow the gray curve and the long-run mass of infection would be $\frac{r}{r+\gamma} - \frac{r}{\beta} = \frac{41}{90} \approx 46\%$.

Fig. 6 illustrates how, unlike in the SI model, it is possible for a barely-OET to exist with infection level I but for no rise-and-plateau equilibrium trajectory to exist in which the plateau phase is a barely-OET with that level of infection.[20] In this example, we fix the parameters $r = 0.1$, $\beta = 1$, $b_0 = 1$, $b_1 = 1$, and $b_2 = 5$ but vary the parameters for disease severity d and disease-death rate γ. For each (d, γ) pair, we first check the conditions of Proposition 10 to determine whether a barely-OET exists with constant infection prevalence $I^\infty \in \{0.05, 0.2\}$ and constant mass of removed agents $R^\infty = \frac{\gamma}{r} I^\infty$, i.e., is $0.05 \in \mathcal{I}^{BO}$ and/or is $0.2 \in \mathcal{I}^{BO}$? We then numerically determine whether the rise-and-plateau trajectory with barely-oscillating infection level $I^\infty \in \{0.05, 0.2\}$ is an equilibrium trajectory. In Fig. 6(a-b): the orange regions are the parameter ranges in which a rise-and-plateau trajectory exists with barely-oscillating infection level $I^\infty = 0.05$ and $I^\infty = 0.2$, respectively, similar to that shown in Fig. 5; the blue regions are the parameter ranges in which a barely-OET exists with that level of infection but the corresponding rise-and-plateau trajectory is not an equilibrium; and the uncolored regions are those in which a barely-OET does not exist with that level of infection.

[19] In the equilibrium trajectory illustrated here, the endemic phase consists of a barely-oscillating trajectory in which the proportion of time in which S-agents are active ("average S-agent activity") itself changes over time. See Appendix B.2 for more details on oscillating trajectories in the SIR model.

[20] We focus on rise-and-plateau equilibria with a barely-OET during the endemic phase for analytical simplicity. More generally in a rise-and-oscillate trajectory with range $[\underline{I}, \overline{I}]$, each active and inactive period will last for a different length of time as the endemic phase progresses. The active-IC and inactive-IC conditions therefore need to be checked separately throughout each oscillation, compared to just checking them throughout one oscillation in the SI model.



(a) $I^\infty = 0.05$ (b) $I^\infty = 0.2$

Fig. 6. Disease-severity parameters (γ, d) given which a rise-and-plateau equilibrium trajectory exists with plateau infection prevalence I^∞ in the SIR model (orange region) or a barely-OET exists with constant infection prevalence I^∞ (orange and blue regions), for $I^\infty \in \{0.05, 0.2\}$ and with other parameters fixed at $(r, \beta, b_0, b_1, b_2) = (0.05, 1, 1, 1, 5)$.

In the SI model, we showed that anytime an OET exists (no matter how big the oscillation), a rise-and-oscillate equilibrium trajectory also exists in which that OET is the endemic phase; see Lemma 2 and Proposition 5. But here we see that this is sometimes not true in the SIR model, even when we restrict attention to the simplest oscillations in which infection prevalence is approximately constant over time. Sometimes, a barely-OET exists but the corresponding rise-and-plateau trajectory is not an equilibrium. To gain intuition, let t^* be the time during the outbreak phase at which $I(t^*) = I^\infty$. As previously discussed, relatively few agents have been removed by time t^* compared to the long run, because the rise in deaths lags the rise in infections during an SIR outbreak. Consequently, the mass of susceptible agents is relatively high, which causes infections to increase relatively quickly during each active period at the beginning of the endemic phase. Active periods must therefore be relatively short (else infection prevalence would increase), which in turn reduces the welfare of susceptible agents. The harm of infection at time t^* is therefore lower than in the long run, which reduces agents' willingness to temporarily cease activity at time t^* as the rise-and-plateau trajectory requires them to do. In cases where agents' incentive to remain active is too strong for them to be willing to cease activity at time t^*, any equilibrium trajectory that eventually converges to the desired barely-OET must initially overshoot infection level I^*.

Remark: Susceptible-Infected-Recovered analysis. The SI-Recovered model differs from the SI-Removed model in that agents who enter the "R" compartment are still alive and immune to infection, and hence able to engage in social-economic activity at no risk. Our methodology in this section can be readily adapted to the SI-Recovered model with only minor changes in algebra. Appendix C presents the corresponding results. Interestingly, we show that the set of SSEs and barely-OETs in the SIRecovered model corresponds exactly to the set of SSEs and barely-OETs in the SI model with suitably modified parameters, with the ex ante likelihood of infection, the harm of infection, and newborn welfare all being the same. All qualitative and quantitative findings in Sections 3.1-3.2 about endemic-equilibrium outcomes in the SI model therefore carry over directly to the SIRecovered model.

5. Endemic-disease equilibrium comparative statics

This section considers how changing model parameters impacts equilibrium welfare in the endemic phase of the epidemic. In particular, we focus on (i) maximal newborn welfare in any steady-state equilibrium, called "SSE newborn welfare" and denoted here by U^{SSE}, and (ii) maximal newborn welfare in any barely-oscillating equilibrium trajectory,[21] called "barely-OET newborn welfare" and denoted here by U^{BOET}.

The most general version of our model has several parameters which could naturally be impacted by public policies, technological discoveries, shifts in cultural practices, pathogen mutation, and other sorts of "interventions": $\beta > 0$, the transmission rate; $d > 0$, the flow disease cost incurred by infected agents; $\gamma \geq 0$, the rate at which infected agents die due to disease; b_1, the benefit of transmissive activity, not including any social benefits; and b_2, the social benefit of activity. (For present purposes, we view the natural death rate r and the baseline benefit of being alive b_0 as fixed parameters.) Before considering equilibrium comparative statics associated with changing these parameters, it is helpful to review our main findings vis-a-vis SSE newborn welfare and barely-OET newborn welfare.

SSE newborn welfare. Our analysis showed that there is a threshold disease severity $\underline{d}$ such that (i) $U^{SSE} = \frac{b_0}{r}$ if $d \geq \underline{d}$ and (ii) $U^{SSE} > \frac{b_0}{r}$ if $d < \underline{d}$. The threshold $\underline{d}$ is provided in equation (8) for the SI model and in equation (31) for the SIR model, repeated here for convenience:

$$\underline{d} = \left(1 + \frac{r+\gamma}{r}\frac{r+\gamma}{\beta - r - \gamma}\right)\left(b_1 + b_2\left(\frac{r}{r+\gamma} + \frac{\gamma}{\beta}\right)\right) - \frac{\gamma}{r}b_0 \tag{37}$$

We focus here on the case of *severe sickness*, meaning that $d > \underline{d}$ and given which there is a unique partial-activity SSE (Proposition 9).

Consider an intervention that changes the parameters $(d, \gamma, \beta, b_1, b_2)$ to $(d^A, \gamma^A, \beta^A, b_1^A, b_2^A)$, and let $\underline{d}^A$ be the disease-severity threshold given these new parameters. (The superscript "A" is mnemonic for *A*fter the intervention.) There are three basic possibilities for how SSE newborn welfare may be affected.

Possibility #1: Disease elimination and maximal welfare. If $\beta^A \leq \gamma^A + r$, then the prevalence of infection will fall toward zero no matter what people do. In the long run, newborn agents will be able to enjoy their lives as if the disease did not exist. In particular, newborn agents get lifetime welfare of approximately $\frac{b_0 + b_1^A + b_2^A}{r} > 0$ with the intervention rather than $\frac{b_0}{r}$ without it.

Possibility #2: Full-activity SSE and improved welfare. Suppose next that $\beta^A > r + \gamma^A$ but $d^A < \underline{d}^A$. In this case, the disease is never eliminated in equilibrium but SSE newborn welfare is now strictly higher than $\frac{b_0}{r}$.

Possibility #3: Unique partial-activity SSE and unchanged welfare. Lastly, suppose that $d^A > \underline{d}^A$, including any intervention that only slightly changes model parameters. In this case, any society-wide benefits associated with the intervention are short-lived, undermined by agents' behavioral responses. In particular, SSE newborn welfare remains equal to $\frac{b_0}{r}$.

For the rest of this section, we will focus on this last case, so that the intervention in question has no impact on SSE newborn welfare. However, such interventions can impact barely-OET newborn welfare.

[21] The full set of OETs is of interest, but barely-OETs are especially convenient to analyze since all agents have the same welfare at birth. In an OET, those born at an oscillation peak have lower welfare than those born at a trough.

Barely-OET newborn welfare. When $d > \underline{d}$ so that a partial-activity SSE exists, we showed that barely-OETs also exist that welfare dominate that partial-activity SSE (Proposition 10). Moreover, we characterized the maximal newborn welfare U^{BOET} that can be achieved in any barely-OET, repeated here for convenience:

$$U^{BOET} = \frac{b_0}{r} + \frac{b_2}{\beta}\frac{r+\gamma}{r}. \tag{38}$$

Long-term impact of improved ventilation and other interventions. Poor ventilation has been implicated as an important factor in tuberculosis transmission; see e.g. Miller-Leiden et al. (1996) for experimental evidence and Du et al. (2020) for a case study of an epidemic on a Taiwanese college campus. Improved ventilation may in some cases be sufficiently effective to stop an epidemic in its tracks, but what if the impacts are more modest? Better ventilation reduces susceptible agents' likelihood of becoming infected for any given infection level I and susceptible-activity level a_S. However, S-agents naturally respond to such improved protection by becoming more active, which increases their likelihood of being exposed. Indeed, because S-agents must be indifferent whether to be active in the unique SSE, modestly improving ventilation ironically causes the prevalence of infection in the unique SSE to increase; also, newborn SSE welfare remains $\frac{b_0}{r}$, the same as before the intervention. On the other hand, if agents are able to coordinate their activity and the epidemic progresses according to a barely-OET, newborn welfare can be as high as $\frac{b_0}{r} + \frac{b_2}{\beta^A}\frac{r+\gamma}{r}$, strictly higher than before the intervention.

Other sorts of interventions can be analyzed in a similar way. So long as the disease causes sufficiently severe sickness that a partial-activity SSE exists, any intervention that modestly changes some or all of the models' parameters will have no impact on newborn SSE welfare. On the other hand, by equation (38), barely-OET newborn welfare is decreasing in the transmission rate β, increasing in the mortality rate γ, increasing in the benefits of social activity b_2, and independent of symptom severity d and the benefits of non-social activity b_1. Thus, interventions that impact some combination of $(\beta, d, \gamma, b_1, b_2)$ can impact barely-OET newborn welfare U^{BOET}. For instance:

- universal immunotherapy and/or vaccination (decreasing β, d, and/or γ, depending on whether the immunotherapy / vaccine prevents infection, prevents illness, and/or prevents death) may or may not increase U^{BOET}, depending on whether the ratio $\frac{r+\gamma}{\beta}$ increases or decreases;
- universal masking (decreasing β as infections are prevented and potentially also decreasing b_2 if social interactions become less fulfilling) may or may not increase U^{BOET}, depending on whether the ratio $\frac{b_2}{\beta}$ increases or decreases;
- closing social public spaces (decreasing b_2) decreases U^{BOET}, but closing non-social public spaces (decreasing b_1) has no effect on U^{BOET};
- pathogen evolution that increases disease mortality (increasing γ) increases U^{BOET}; and
- pathogen evolution that only affects symptom severity (d may increase or decrease) has no effect on U^{BOET}.

Most of these comparative statics are intuitive, but some readers may be surprised that barely-OET newborn welfare *increases* as the pathogen becomes more deadly. To gain intuition, note that increased disease mortality accelerates the decline in infection prevalence during the inactive

phases of an oscillating trajectory.[22] This effect shortens the inactive phase of each oscillation and allows the uninfected to enjoy more of their lives with one another—an effect that becomes relatively more important when the social benefits of activity are larger.

6. Concluding remarks

This paper explores the impact of social benefits (i.e. economic complementarities) of transmissive activity on the equilibrium course of an infectious-disease epidemic. We show that the qualitative nature of equilibrium epidemics and the quantitative predictions derived from equilibrium analysis can hinge critically on whether or not there are social benefits. For analysts and policy-makers, it is therefore essential to use models that accurately account for the extent of economic complementarities.

To illustrate the equilibrium impact of social benefits in the clearest way possible, we consider a hypothetical infectious disease (loosely motivated by the emerging threat of totally-resistant tuberculosis) from which infected people never recover and in which transmission dynamics follow a standard Susceptible-Infected-Removed ("SIRemoved") epidemiological model with an equal flow of births and non-disease deaths. Within this context, the equilibrium impact of social benefits is easiest to see in the special case in which the disease causes severe symptoms but infected people do not die of the disease.

Without social benefits: We show that there is a unique equilibrium epidemic trajectory, which follows an especially simple rise-and-plateau pattern. During the first part of the epidemic ("outbreak phase"), all agents are active and the prevalence of infection increases until a critical level is reached. The epidemic then enters a steady state ("endemic phase") in which susceptible agents are indifferent whether to be active and have subsequent welfare the same as if barred from all activity for the rest of their lives.

With social benefits: There is still a unique equilibrium trajectory that follows a rise-and-plateau pattern and, if the epidemic were to follow that trajectory, there would be relatively little loss in abstracting from social benefits. However, we show that many other equilibrium epidemic trajectories also exist that never settle into a steady state, including some that welfare dominate the equilibrium trajectory that settles into the unique equilibrium steady state. Moreover, as we show in numerical examples, the long-run prevalence of infection and population-wide welfare can be very different in these other equilibrium trajectories.

A strength of our approach is that our model is highly tractable and can potentially be enriched and extended in several interesting directions, a few of which we mention here.

Lockdowns and other activity restrictions. We consider an equilibrium model in which agents do not face any endogenous[23] constraints related to their activity. Policies that restrict (or tax) agents' activity can induce better epidemic outcomes, especially if they can target infected agents. In particular, if the planner can identify who is infected, newborn welfare in the long run would be maximized by perfectly quarantining all infected agents and allowing uninfected agents to be fully active, since then the disease can be eliminated even as economic activity is

[22] For instance, in the zombie epidemic of the TV show *Walking Dead*, higher mortality weeds out the zombie horde more quickly, allowing survivors to spend less time in hiding.

[23] Exogenous constraints that remain the same throughout the epidemic, such as debilitating illness that reduces infected agents' ability to leave the home, can be easily incorporated by appropriately adjusting the transmission rate.

fully enjoyed by those who remain uninfected. Several authors have recently examined how to intervene optimally during an epidemic (e.g., Alvarez et al. (2021), Bethune and Korinek (2020), and Rowthorn and Maciejowski (2020)) but, following the hitherto-standard modeling approach, these authors abstracted from the possibility of social benefits from activity. In future work, it would be interesting to revisit this "optimal lockdown" literature within an extended framework allowing for social benefits.

Time-limited epidemics with immunity after infection. In this paper, we assume that there is a steady flow of new susceptible agents (as infected agents die and new ones are born), supporting indefinite transmission of the disease.[24] However, some epidemics spread so rapidly that the host population is essentially fixed and no steady state with a positive amount of infection is ever reached. For instance, the measles virus spread rapidly across the island of Tahiti on three separate occasions in 1929, 1951, and 1960 (Rosen (1962)), each time quickly disappearing after causing a sharp outbreak. Motivated by the Covid-19 outbreak, several authors have recently analyzed such time-limited epidemics from a game-theory perspective, characterizing the equilibrium course of the epidemic; see e.g., Farboodi et al. (2021), McAdams (2020), and Toxvaerd (2020). A common feature of these equilibrium-epidemic models is that there is a period of time in the middle of the epidemic in which the prevalence of infection is roughly constant, what McAdams (2021) refers to as "epidemic limbo." During this limbo period, the level of infection is sufficiently high that susceptible agents prefer to reduce their transmissive activity—but not enough to drive down the level of infection. If there are social benefits associated with activity, our analysis can be easily adapted to show that, once epidemic limbo has been reached, there are in fact many potential equilibrium trajectories for the rest of the epidemic. Understanding the set of equilibrium-epidemic outcomes that can arise in a time-limited epidemic when there are social benefits is an important direction for future work.

Asymptomatic infection and diagnostic testing. Many disease-causing pathogens, including HIV and SARS-CoV-2, can spread without causing noticeable symptoms. However, due to recent advances in diagnostic technology, it may be possible for agents to test themselves to determine their own health status, and show these test results to others to prove their status. In this context, an agent's incentive to invest in getting tested depends on the prevalence of infection (while infection is rare, no one will bother getting tested) and whether others have the ability and incentive to exclude them from social activity unless they can show a recent negative test. In future work, it would be interesting to extend our model to allow for asymptomatic infection and account for agents' equilibrium incentive to learn about and share their own infection status with others.

Limited synchronization of activity. Our findings highlight how the endemic burden of an infectious disease critically depends on whether or not agents in the population are able to synchronize their aggregate activity, alternating between periods with more and then less activity. Such synchronization seems unrealistic at a global level, but is routinely achieved to a limited extent at smaller scales, such as schools, workplaces, and social pods. Bearing this in mind, it would be valuable to extend our analysis to a richer setting in which individuals interact not just with the "general population" but also in social groups within which coordinated activity is possible. Even

[24] A less important simplifying assumption is that there is no recovery from infection. Our analysis can be easily extended to a more general Susceptible-Infected-Recovered-Susceptible (SIRS) model with recovery, adaptive immunity after recovery, and potential loss of immunity. However, little additional economic insight emerges from this more complex epidemiological model.

if the general population settles into a low-welfare equilibrium steady state, those within such a social group can improve their own welfare by coordinating on an oscillating pattern for their own activity with one another.

The politics of activity restrictions. Our analysis focuses on the welfare of newly-born agents, but agents' preferences change once they become infected. This could have significant implications for the politics of public health during an emerging epidemic, as infected agents prefer for others' activity to be as high as possible when there are social benefits of activity. In future work, it would be interesting to model the political dynamics of an infectious-disease outbreak more explicitly, accounting for the evolving preferences of the host population.

Appendix A. SI model

This Appendix provides further details and omitted proofs for our SI analysis in Section 3.

A.1. Omitted steps in proof of Proposition 6

To complete the proof of Proposition 6(i), we need to show that $\mathcal{I}^{BO}$ is closed whenever non-empty, i.e., we need to show that $\{I^{min}, I', I'', I^{max}\}$ are all barely-OET infection levels. Define shorthand $\underline{U} \equiv \min_{I\in(0,1-r]} U_S^{BO}(I)$. There are three relevant cases. First, if $\underline{U} \geq \frac{b_2}{\beta}$, then $\mathcal{I}^{BO}$ is either empty or a (closed) singleton with $\mathcal{I}^{BO} = \arg\min U_S^{BO}(I)$.

Second, suppose that $\underline{U} \in (0, b_2)$. $\mathcal{I}^{BO}$ is a single interval with $I^{min} \equiv \inf \mathcal{I}^{BO}$ and $I^{max} \equiv \sup \mathcal{I}^{BO}$. We need to show that I^{min} and I^{max} are in $\mathcal{I}^{BO}$, making it a closed set. To show that $I^{min} \in \mathcal{I}^{BO}$, we need to show that there is a sequence of oscillating equilibrium trajectories with limit-infection level I^{min}. We do so with a standard diagonal argument. Consider a sequence $\epsilon_l \to 0$. For each l, there exists $I_l \in (I^{min}, I^{min} + \epsilon_l)$ such that $I_l \in \mathcal{I}^{BO}$. We may therefore define a sequence $\{\mathcal{E}^O_{l,k} : k = 1, 2, ...\}$ consisting only of equilibrium oscillating trajectories and with limit-infection level I^{min}. Now consider the diagonal sequence $\{\mathcal{E}^O_{k,k} : k = 1, 2, ...\}$. Each element of this sequence is an equilibrium trajectory, and the sequence has limit-infection level I^{min}; thus, $I^{min} \in \mathcal{I}^{BO}$. The fact that $I^{max} \in \mathcal{I}^{BO}$ can be shown in the same way, but now using an infection-level sequence $(I_l \in (I^{max} - \epsilon_l, I^{max}) : l = 1, 2, ...)$ that converges to I^{max} from below.

Finally, suppose that $\underline{U} \leq 0$ and define $I' \leq I''$ by $U_S^{BO}(I') = U_S^{BO}(I'')$. (If $\underline{U} = 0$, then $I' = I''$; otherwise, $I' < I''$.) Our diagonal-limit argument now shows that $\{I', I''\} \subset \mathcal{I}^{BO}$, using infection-level sequences that converge to I' from below and to I'' from above.

A.2. Exact conditions for non-existence of barely-OET

This subsection explicitly characterizes the parameter range given which $\mathcal{I}^{BO}$ is empty.

As is illustrated in Fig. 3, the incentive compatible constraints for barely-OET are quadratic in I with positive vertical intercept. Furthermore, the range of infection levels that satisfy the inactive-IC condition supporting barely-oscillating trajectories is an interval, while the range that violates the active-IC condition is a proper subset of that interval. Thus, $\mathcal{I}^{BO}$ is empty if and only if inactive-IC is violated for all $I \in (0, 1 - \frac{r}{\beta}]$. For convenience, we re-write inactive-IC as a function of $I \in (0, 1 - \frac{r}{\beta}]$:

$$\frac{b_1 + b_2}{\beta} + \frac{I}{r}\left(b_1 + \left(I + \frac{r}{\beta}\right)b_2 - d\right) < \frac{b_2}{\beta}.$$

Rewriting the inequality yields the quadratic form

$$\frac{\beta}{r}b_2 I^2 + \left[\frac{\beta}{r}(b_1 - d) + b_2\right]I + b_1 < 0$$

Defining the left-hand-side of the above inequality as $f(I)$, which is a convex parabola passing through point $(0, b_1)$ (at the left limit). For the inactive-IC to be violated for all $I \in (0, 1 - \frac{r}{\beta}]$, we equivalently seek for parametric conditions to make $f(I) \geq 0\ \forall I \in (0, 1 - \frac{r}{\beta}]$. There are three cases to discuss.

Case 1: $d \leq \frac{r}{\beta}b_2 + b_1$ (the axis of symmetry of $f(I)$ is less than or equal to 0). In this case, $f(I)$ is monotonically increasing for all $I \in (0, 1 - \frac{r}{\beta})$. Then $f(I) \geq f(0) = b_1 > 0$. Therefore, the inactive-IC is always violated in this case.

Case 2: $d \geq (2 - \frac{r}{\beta})b_2 + b_1$ (the axis of symmetry is weakly larger than $1 - \frac{r}{\beta}$). In this case, $f(I)$ is monotonically decreasing for all $I \in (0, 1 - \frac{r}{\beta}]$. Thus $f(I) \geq 0\ \forall I \in (0, 1 - r)$ if and only if

$$f(1 - \frac{r}{\beta}) = \frac{\beta}{r}b_2\Big(1 - \frac{r}{\beta}\Big)^2 + \left[\frac{\beta}{r}(b_1 - d) + b_2\right]\Big(1 - \frac{r}{\beta}\Big) + b_1 \geq 0,$$

which gives $d \leq \frac{b_1}{1-\frac{r}{\beta}} + b_2$. For the set of feasible d's to be non-empty, we require

$$\frac{b_1}{1 - \frac{r}{\beta}} + b_2 \geq (2 - \frac{r}{\beta})b_2 + b_1,$$

yielding

$$\frac{b_1}{b_2} \geq \frac{(1 - \frac{r}{\beta})^2}{\frac{r}{\beta}}.$$

Case 3: $d \in (\frac{r}{\beta}b_2 + b_1, (2 - \frac{r}{\beta})b_2 + b_1)$ (the axis of symmetry is within the interval of $(0, 1 - \frac{r}{\beta}]$). In this case, the minimum of $f(I)$ is obtained at the axis of symmetry. The range of d establishing non-existence of OET is then characterized the inequality that requires the minimum to be positive, i.e.,

$$\min_I f(I) = \frac{4\frac{\beta}{r}b_1 b_2 - (\frac{\beta}{r}(b_1 - d) + b_2)^2}{4\frac{\beta}{r}b_2} \geq 0.$$

Combining with the restriction of Case 3, we conclude that

$$d \leq \frac{r}{\beta}b_2 + b_1 + 2\sqrt{\frac{r}{\beta}b_1 b_2}.$$

To sum up, the conditions for non-existence of barely-OET are characterized as follows:

1. $d \leq \frac{r}{\beta}b_2 + b_1$; or
2. $d > \frac{r}{\beta}b_2 + b_1$, and
   (a) $d < \frac{b_1}{1-\frac{r}{\beta}} + b_2$, if $d \geq (2 - \frac{r}{\beta})b_2 + b_1$ and $\frac{b_1}{b_2} > \frac{(1-\frac{r}{\beta})^2}{\frac{r}{\beta}}$;
   (b) $d < \frac{r}{\beta}b_2 + b_1 + 2\sqrt{\frac{r}{\beta}b_1 b_2}$, if $d < (2 - \frac{r}{\beta})b_2 + b_1$.

Fig. 4 panel (c) and (d) can be viewed as a numerical illustration for 2(b). Under parameters of $\beta = 1, d = 12, b_2 = 11, r = 0.1$ ($\frac{r}{\beta}b_2 + \hat{b}_1 + 2\sqrt{\frac{r}{\beta}\hat{b}_1 b_2} = d$ gives $\hat{b}_1 = 6.0007$), panel (c) ($b_1 = 5.8$) is the case that OETs are on the brink of existence (which still has a notable range of OETs), while partial-activity SSE does not exist. Panel (d) ($b_2 = 7.5$) shows non-existence of OET.

Appendix B. SIR(emoved) model

This Appendix provides further details and omitted proofs for our SIR analysis in Section 4.

B.1. Proof of Proposition 7

We establish equilibrium existence by an application of Glicksberg's fixed-point theorem (Glicksberg (1952)). SIR epidemiological dynamics $(S(\cdot), I(\cdot))$ and population-wide activity $A(\cdot) = a_S(\cdot)S(\cdot) + I(\cdot)$ are determined by the initial condition $(S(0), I(0))$ and the S-agent activity process $a_S(\cdot)$; as shorthand, say that $(S(\cdot), I(\cdot))$ is "generated" by $a_S(\cdot)$. Define a correspondence $\mathcal{F}$ that maps $a_S(\cdot)$ to the set of activity processes $\mathcal{A}_S(\cdot; a_S(\cdot))$ that are individually optimal for an S-agent in a trajectory with dynamics generated by $a_S(\cdot)$. An equilibrium trajectory exists if and only if $\mathcal{F}$ has a fixed point. To establish existence by Glicksberg's theorem, it suffices to show that $\mathcal{F}$ is convex-valued and has a closed graph.

Consider an S-agent i who believes that the epidemic is generated by $a_S(\cdot)$. $\mathcal{A}_S(\cdot; a_S(\cdot))$ is the set of activity processes that are individually optimal for agent i while uninfected. Let $U_S(t; a_S(\cdot))$ be agent i's welfare at time t, assuming individually-optimal activity at all future times. Similarly, let $U_I(t; a_S(\cdot))$ be the time-t welfare of an optimizing infected agent and $\Delta U(t; a_S(\cdot)) = U_S(t; a_S(\cdot)) - U_I(t; a_S(\cdot))$ agent i's time-t harm of infection. As shorthand, define $Y(t) \equiv b_1 + b_2 A(t) - \beta I(t)\Delta U(t; a_S(\cdot))$ By inequality (6) for each t: $\widehat{a}_S(t) = 1$ for all $\widehat{a}_S(\cdot) \in \mathcal{A}_S(\cdot; a_S(\cdot))$ if $Y(t) > 0$; $\widehat{a}_S(t) = 0$ for all $\widehat{a}_S(\cdot) \in \mathcal{A}_S(\cdot; a_S(\cdot))$ if $Y(t) < 0$; and $\widehat{a}_S(t) \in [0, 1]$ for all $\widehat{a}_S(\cdot) \in \mathcal{A}_S(\cdot; a_S(\cdot))$ if $Y(t) = 0$. Convexity of the set $\mathcal{A}_S(\cdot; a_S(\cdot))$ is immediate.

We establish that $\mathcal{F}$ has a closed graph with respect to the functional-space norm $\|a_S(\cdot)\| \equiv \int_0^\infty e^{-rt} a_S(t)$. Consider any convergent sequence $a_S^k(\cdot) \to a_S(\cdot)$. Let $(S^k(\cdot), I^k(\cdot))$ be the epidemiological dynamics generated by $a_S^k(\cdot)$ and let $\Delta U^k(t)$ be the corresponding harm of infection for an optimizing agent given those epidemiological dynamics. Observe first that $\lim_{k\to\infty} S^k(t) = S(t)$ and $\lim_{k\to\infty} I^k(t) = I(t)$ for all t, where $(S(\cdot), I(\cdot))$ are generated by $a_S(\cdot)$. The welfare of optimizing agents is therefore also continuous in $a_S(\cdot)$, implying that $\lim_{k\to\infty} \Delta U^k(t) = \Delta U(t; a_S(\cdot))$ for all t and hence also $\lim_{k\to\infty} Y^k(t) = Y(t)$ for all t.

Now, let $\widehat{a}_S^k(\cdot)$ be a selection from $\mathcal{A}_S(\cdot; a_S^k(\cdot))$ and suppose that $\widehat{a}_S^k(\cdot) \to \widehat{a}_S(\cdot)$. We need to show that $\widehat{a}_S(\cdot) \in \mathcal{A}_S(\cdot; a_S(\cdot))$. Because $\lim_{k\to\infty} \int_0^\infty e^{-rt}(\widehat{a}_S^k(t) - \widehat{a}_S(t))dt = 0$, the set of times at which $\lim_{k\to\infty} \widehat{a}_S^k(t) \neq \widehat{a}_S(t)$ has zero measure. For each t in the remaining full-measure set, there are three possibilities: (i) $\widehat{a}_S(t) = 0$, in which case $\widehat{a}_S^k(t) \in [0, \epsilon)$ for all sufficiently large k, implying that $Y^k(t) \leq 0$ and hence $Y(t) \leq 0$, making non-activity optimal in the trajectory generated by $a_S(t)$; (ii) $\widehat{a}_S(t) = 1$, in which case $\widehat{a}_S^k(t) \in (1 - \epsilon, 1]$ for all sufficiently large k, implying $Y^k(t) \geq 0$ and $Y(t) \geq 0$, making activity optimal; and (iii) $\widehat{a}_S(t) \in (0, 1)$, in which case $\widehat{a}_S^k(t) \in (0, 1)$ for all sufficiently large k, implying $Y^k(t) = 0$ and $Y(t) = 0$, making an optimizing S-agent indifferent whether to be active. We conclude that $\widehat{a}_S(\cdot) \in \mathcal{A}_S(\cdot; a_S(\cdot))$ is an optimal activity rule when the trajectory is generated by $a_S(\cdot)$, as desired. □

B.2. Omitted details on oscillating trajectories in the SIR model

Definition and construction of oscillating trajectories. In the SIR model, an "oscillating trajectory" is more complex to define than in the SI model. We do so as follows. *First,* fix a range of oscillation $[\underline{I}, \overline{I}]$ and an initial condition $(I(0), R(0))$ with $I(0) = \underline{I}$ and $\overline{I} < \frac{r}{r+\gamma} - \frac{r}{\beta}$.[25] The first "active period" begins at time $T_{0,1} = 0$. *Second*, differential equations $I'(t) = \beta(1 - S(t) - I(t))I(t) - (r+\gamma)I(t)$ and (4) determine the path of $(I(t), R(t))$ during the first active period while $a_S(t) = 1$. Let $T_{1,1}$ be the first time at which $I(t) = \overline{I}$, the end of the first active period. *Third*, differential equations $I'(t) = -(r+\gamma)I(t)$ and (4) determine the path of $(I(t), R(t))$ during the first inactive period while $a_S(t) = 0$. Let $T_{0,2}$ be the next time at which $I(t) = \underline{I}$, the end of the first inactive period. *Finally*, continue in the same way to construct the length of the second active period $[T_{0,2}, T_{1,2}]$ and the second inactive period $[T_{1,2}, T_{0,3}]$, and so on for all subsequent oscillations.

Note that the mass of removed agents $R(t)$ need not oscillate at the start of an oscillating trajectory. Indeed, as we discuss in the main text and illustrate in Fig. 5, $R(t)$ may be much lower than its long-run oscillation range at the beginning of an oscillating trajectory, in which case $R(t)$ could increase steadily throughout the first several oscillations.

Equilibrium verification and agent welfare. A given oscillating trajectory is an equilibrium if and only if the active-IC inequality (39) holds throughout each active period and the inactive-IC inequality (40) holds throughout each inactive period:

$$b_1 + b_2(1 - R(t)) \geq \beta I(t)\Delta U(t) \text{ for all } k = 0, 1, ..., \text{ and all } t \in [T_{0,k}, T_{1,k}] \tag{39}$$

$$b_1 + b_2 I(t) \leq \beta I(t)\Delta U(t) \text{ for all } k = 0, 1, ..., \text{ and all } t \in [T_{1,k}, T_{0,k+1}] \tag{40}$$

where $\Delta U(t) = U_S(t) - U_I(t)$ is the harm of infection at time t.

In order to check these inequalities, it is necessary first to compute the welfare of susceptible and infected agents at each point along the trajectory. In the SI model, this computation is simplified by the fact that each oscillation is identical and hence agents' welfare is the same at the start of each oscillation. By contrast, in the SIR model, computing welfare at any given point in time requires integrating over the entire continuation trajectory. From a conceptual point of view, however, it is trivial to extend the welfare computation provided in Section 3.2 to the SIR context. We omit the tedious details here to save space.

B.3. Proof of Lemma 5

For an arbitrary infection level $I \in \mathcal{I}^{BO}$, consider any fixed time interval $T > 0$ and any oscillating trajectory such that $\underline{I} > I - \epsilon$, $\overline{I} < I + \epsilon$, and $R(t)$ uniquely pinned down by the epidemiological dynamics (4). Define, for any fixed time interval $[t, t+T]$ with some $t \geq 0$, $T > 0$, the S-agents' *average* activity as $a_S(t;T) \equiv \frac{\int_t^{t+T} a_S(t)dt}{T}$. As long as $\epsilon \approx 0$, $\underline{I} \approx \overline{I} \approx I$ and $R(t) \approx R(I) \equiv \frac{\gamma}{r} I$, for all t. Therefore, $a_S(t;T) \approx a_S^{BO}(I)$ is given by $\beta a_S^{BO}(I) SI = I(r+\gamma)$, i.e. $a_S^{BO}(I) = \frac{r+\gamma}{\beta S}$ where $S(t) \approx S(I) = 1 - I - R(I) \equiv \frac{r+\gamma}{r} I$. Average overall activity is then $A(t;T) \approx A^{BO}(I) \equiv I + a_S^{BO}(I)S = I + \frac{r+\gamma}{\beta}$. Then we have the corresponding term to (23): the individual welfare of infected agents, $U_I(t)$, is approximately

[25] Recall that $\frac{r}{r+\gamma} - \frac{r}{\beta}$ is the long-run level of infection absent any distancing. $\overline{I} < \frac{r}{r+\gamma} - \frac{r}{\beta}$ therefore ensures that infection prevalence will eventually reach $\overline{I}$ when all agents are active.

$$U_I^{BO}(I) = \frac{b_0 + b_1 + b_2(I + \frac{r+\gamma}{\beta}) - d}{r+\gamma}.$$

In a barely-OET, the susceptible agents get an approximately constant average flow payoff $b_0 + a_S^{BO}(I)(b_1 + b_2(1 - \frac{\gamma}{r}I))$, plus continuation value $U_I^{BO}(I)$ when infected. They remain susceptible for expected length of time $\frac{1}{r+\beta a_S^{BO}(I)I} = \frac{1-\frac{r+\gamma}{r}I}{r}$, and their ex ante likelihood of becoming infected is $\frac{\beta a_S^{BO}(I)I}{r+\beta a_S^{BO}(I)I} = \frac{r+\gamma}{r}I$. Therefore we obtain the corresponding term to (24): S-agents have individual welfare $U_S(t)$, approximately,

$$\begin{aligned} U_S^{BO}(I) =& \frac{b_0(1-\frac{r+\gamma}{r}I)}{r} + \frac{r+\gamma}{\beta r}\left(b_1 + b_2\left(1-\frac{\gamma}{r}I\right)\right) + \frac{r+\gamma}{r}IU_I^{BO}(I) \\ =& \frac{1-\frac{\gamma}{r}I}{r}b_0 + \frac{r+\gamma}{\beta r}\left(b_1 + b_2\left(1-\frac{\gamma}{r}I\right)\right) + I\frac{b_1 + \left(I + \frac{r+\gamma}{\beta}\right)b_2 - d}{r}, \end{aligned}$$

confirming the equation in the statement of the lemma. The harm of infection is then

$$\begin{aligned} \Delta U^{BO}(t) \approx \Delta U^{BO}(I) =& \frac{b_0(1-\frac{r+\gamma}{r}I)}{r} \\ &+ \frac{r+\gamma}{\beta r}\left(b_1 + b_2\left(1-\frac{\gamma}{r}I\right)\right) - \left(1 - \frac{r+\gamma}{r}I\right)U_I^{BO}(I), \end{aligned}$$

for all t. The (strict) IC conditions which correspond to (25) and (26) are

$$\begin{aligned} b_1 + b_2(I+S) = b_1 + b_2\left(1-\frac{\gamma}{r}I\right) &> \beta I \Delta U^{BO}(I) \\ b_1 + b_2 I &< \beta I \Delta U^{BO}(I). \end{aligned}$$

Note that

$$\begin{aligned} & b_1 + b_2\left(1-\frac{\gamma}{r}I\right) - \beta I \Delta U^{BO}(I) \\ &= \left(1 - \frac{r+\gamma}{r}I\right)\left(b_1 + b_2\left(1-\frac{\gamma}{r}I\right) + \beta I U_I^{BO}(I)\right) - \beta I \frac{b_0(1-\frac{r+\gamma}{r}I)}{r} \\ &= \left(1 - \frac{r+\gamma}{r}I\right)\frac{\beta r}{r+\gamma}\left(U_S^{BO}(I) - \frac{b_0}{r}\right). \end{aligned}$$

Therefore we can rewrite the final IC conditions, after applying the usual continuity argument, as $U_S^{BO}(I) > \frac{b_0}{r}$ and $U_S^{BO}(I) < \frac{b_0}{r} + \frac{r+\gamma}{\beta r}b_2$. Therefore, $I \in \mathcal{I}^{BO}$ if $\frac{b_0}{r} < U_S^{BO}(I) < \frac{b_0}{r} + \frac{r+\gamma}{\beta r}b_2$, but not if $U_S^{BO}(I) < \frac{b_0}{r}$ or $U_S^{BO}(I) > \frac{b_0}{r} + \frac{r+\gamma}{\beta r}b_2$. □

B.4. Proof of Proposition 10

By Lemma 5, whenever $U_S^{BO}(I) < \frac{b_0}{r}$ or $U_S^{BO}(I) > \frac{b_0}{r} + \frac{r+\gamma}{\beta r}b_2$, $\mathcal{I}^{BO}$ is empty. Now we focus on the cases where $\mathcal{I}^{BO}$ is non-empty. Note that $U_S^{BO}(I)$ is continuous, quadratic and strictly convex with $U_S^{BO}(0) > \frac{b_0}{r} + \frac{r+\gamma}{\beta r}b_2$. Applying the same argument of Appendix A.1 with the bound of the quadratic function $U_S^{BO}(I)$ replaced by $\frac{b_0}{r} + \frac{r+\gamma}{\beta r}b_2$, we establish that $\mathcal{I}^{BO}$ has to be closed whenever non-empty. Combined with the condition that $\mathcal{I}^{BO} \subset (0, \frac{r}{r+\gamma} - \frac{r}{\beta}]$, it follows that (i) and (iv) hold.

To prove (ii) and (iii), consider a partial-activity SSE with infection level I, characterized by $U_S^{SS}(I) = \frac{b_0}{r}$. Note that in a barely-OET, S-agents remain alive and susceptible on average for length of time $\frac{1}{r+\beta a_S^{BO}(I)I} = \frac{r+\gamma}{r}\frac{1}{\beta a_S^{BO}(I)}$, during which they earn a flow of additional benefit

$$a_S^{BO}(I)\left(\left(1-\frac{\gamma}{r}I\right)-A^{BO}(I)\right)b_2 = a_S^{BO}(I)\left(1-(r+\gamma)\left(\frac{1}{\beta}+\frac{I}{r}\right)\right)b_2$$

as compared to an SSE with the same infection prevalence. Therefore

$$U_S^{BO}(I) = U_S^{SS}(I) + \frac{r+\gamma}{\beta r}\left(1-(r+\gamma)\left(\frac{1}{\beta}+\frac{I}{r}\right)\right)b_2,$$

which exactly corresponds to the proof of Proposition 6. When $U_S^{SS}(I) = \frac{b_0}{r}$, $U_S^{BO}(I)$ satisfies the strict IC constraints, i.e., $U_S^{BO}(I) \in (\frac{b_0}{r}, \frac{b_0}{r} + \frac{r+\gamma}{\beta r}b_2)$, since

$$(r+\gamma)\left(\frac{1}{\beta}+\frac{I}{r}\right) = \frac{r+\gamma}{\beta} + I + \frac{\gamma}{r}I = a_S S + I + R \in (0,1).$$

Hence (ii) is proven. As S-agents earn a strictly positive flow of additional benefit in the barely-OET than in the SSE, (iii) is also proven. This completes the proof. □

B.5. Omitted details on rise-and-plateau trajectories with endemic barely-OET

Here we provide additional analysis and results regarding rise-and-plateau trajectories in the SIR model, where susceptible agents barely oscillate in the endemic phase. Denoted as $\mathcal{E}^{BO}(I, I(0))$, a trajectory starting with initial infection prevalence $I(0) > 0$ and $R(0) = 0$, such that susceptible agents remain fully active until $I(\hat{t}) = I \in (I(0), \tilde{I})$, and barely oscillate thereafter so that $I(t) = I$. The following analysis underlies our numerical work in the examples provided in the text.

We first investigate whether $\mathcal{E}^{BO}(I, I(0))$ is incentive compatible after $\hat{t}$, proceeding in the following steps.

Step 1. Let $f(x) \equiv \frac{r+\gamma}{\beta(1-I-x)}$, which is increasing in x. We know the following:

(1) The expected length of time that an S-agent remains susceptible has range $[\frac{1}{r+f(0)I}, \frac{1}{r+f(\frac{\gamma}{r}I)I}]$.
(2) When susceptible, the agent's activity flow has range $[f(0), f(\frac{\gamma}{r})]$.
(3) The ex ante likelihood of the agent being infected has range $[\frac{f(0)I}{r+f(0)I}, \frac{f(\frac{\gamma}{r}I)I}{r+f(\frac{\gamma}{r}I)I}]$.
(4) The average overall activity is equal to $I + \frac{r+\gamma}{\beta}$.

Step 2. For $t > \hat{t}$, we will use $U_I^{BO}(I;t)$ and $U_S^{BO}(I;t)$ to denote the continuation payoff of an infected agent and a susceptible one respectively. t is needed as a parameter since the measure of R over time converges to $\frac{\gamma}{r}I$ but is not constant. Observe that

- (a) By (4), $U_I^{BO}(I;t)$ is given by

$$U_I^{BO}(I;t) = \frac{b_0+b_1+(I+\frac{r+\gamma}{\beta})b_2}{r+\gamma} - \frac{d}{r+\gamma}.$$

- (b) A range of $U_S^{BO}(I;t)$ can be obtained from (1)-(3) and (a) since we can write

$$\begin{aligned} U_S^{BO}(I;t) =& \hat{L}_S^{BO}(I;t) \times (b_0 + (I + \frac{r+\gamma}{\beta})b_2) \\ &+ b_1 \int_t^{\infty} \beta I \hat{a}_S^{BO}(I;t') e^{-\beta I \int_t^{t'} \hat{a}_S^{BO}(I;x)dx} dt' \\ &+ \hat{K}_S^{BO}(I;t) \times U_I^{BO}(I;t), \end{aligned}$$

  $\hat{L}_S^{BO}(I;t)$ and $\hat{K}_S^{BO}(I;t)$ denote the (future) expected length of remaining susceptible and the ex ante likelihood of being infected, evaluated at time t; $\hat{a}_S^{BO}(I;t)$ denotes the average activity flow at time t. Their ranges correspond to (1)-(3) while $U_I^{BO}(I;t)$ is given by (a).

Step 3. The IC constraints for susceptible agents are

$$\begin{aligned} b_1 + b_2(I + \frac{r+\gamma}{\beta \hat{a}_S^{BO}(I;t)}) >& I\Delta U^{BO}(I;t) \equiv U_S^{BO}(I;t) - U_I^{BO}(I;t) \\ b_1 + b_2 I <& I\Delta U^{BO}(I;t). \end{aligned}$$

From Step 2 we can already obtain a range of $\Delta U^{BO}(I;t)$. Let $\Delta\overline{U}(I)$ denote its upper bound and $\Delta\underline{U}(I)$ its lower bound. We also know that $I + \frac{r+\gamma}{\beta \hat{a}_S^{BO}(I;t)} > 1 - \frac{\gamma}{r} I$ since $R(t)$ only converges to from below, but never reaches, $\frac{\gamma}{r} I$. A sufficient condition for the IC constraints regardless of t is then

$$\begin{aligned} b_1 + b_2(1 - \frac{\gamma}{r} I) >& I\Delta\overline{U}(I) \\ b_1 + b_2 I <& I\Delta\underline{U}(I). \end{aligned}$$

Suppose that the above condition is satisfied for some $I = I^* \in (0,1)$ (which is also the measure of infected agents in some barely-OET). This means that $\mathcal{E}^{BO}(I^*, I(0))$ is incentive compatible starting from $t = \hat{t}$. We now show that $\mathcal{E}^{BO}(I^*, I(0))$ is also incentive compatible before $t = \hat{t}$.

Consider one susceptible agent i and suppose that for some $t' \geq 0$ and $\epsilon > 0$ such that $t' + \epsilon < \hat{t}$, being inactive is i's best response for all $t \in [t', t' + \epsilon)$. Note that the (other) susceptible agents choose full activity and that $I(t) < I^*$, whenever $t < \hat{t}$: this means that as long as i chooses to be active for a positive measure of time on $[t' + \epsilon, \hat{t})$, her continuation value at $t' + \epsilon$ is strictly positive. Indeed, being active at any $t \in [t' + \epsilon, \hat{t})$ means, as compared to any $t \geq \hat{t}$, (weakly) more benefit from higher social activity level and (strictly) less risk from lower infection level. Therefore in i's best response she will never remain inactive until $\hat{t}$. It also implies that $U_S(t' + \epsilon)$ (i's continuation value at $t' + \epsilon$) is strictly positive.

Suppose WLOG that i will be active at $t = t' + \epsilon$. It implies that

$$b_1 + b_2 A(t' + \epsilon) = b_1 + b_2(1 - R(t' + \epsilon)) \geq \beta I(t' + \epsilon)(U_S(t' + \epsilon) - U_I(t' + \epsilon)).$$

However, we know that (1) $U_S(t') < U_S(t' + \epsilon)$ since i supposedly chooses to remain inactive from t' to $t' + \epsilon$, (2) $U_I(t') > U_I(t' + \epsilon)$ since all agents remain active before $I(t)$ reaches I^*, and (3) $I(t') < I(t' + \epsilon)$. Therefore

$$b_1 + b_2(1 - R(t')) > b_1 + b_2(1 - R(t' + \epsilon)) > \beta I(t')(U_S(t') - U_I(t')),$$

which means that i should become active at t', a contradiction. Therefore the whole trajectory $\mathcal{E}^{BO}(I^*, I(0))$ is an equilibrium one.

Now we show that our argument for a rise-and-barely-oscillate equilibrium trajectory in the SI model extends to the SIR model, provided that γ does not exceed a positive threshold.

Proposition 11. *Fix $(\beta, b_0, b_1, b_2, r, d)$, and suppose that I^* lies in the interior of $\mathcal{I}^{BO}$ in the SI model. Then there exists $\overline{\gamma} > 0$ such that $\forall \gamma < \overline{\gamma}$, $\mathcal{E}^{BO}(I^*, I(0))$ is an equilibrium trajectory for all $I(0) \in (0, I^*)$.*

Proof. Since I^* lies in the interior of $\mathcal{I}^{BO}$, we know that I^* can be reached in the SI model, which means that $\mathcal{E}^{BO}(I^*, I(0))$ is feasible $\forall I(0) < I^*$ when γ is sufficiently small. To see this, take some $\epsilon > 0$ and let γ be such that $\gamma I^* - r\epsilon < 0$. This implies the following: suppose that in the SIR dynamics with some $I(0) \in (0, I^*)$, $R(0) = 0$ and full activity, $I(t)$ never reaches I^* when it increases from $I(0)$; then $R(t)$ can never go beyond ϵ during this time. However, when ϵ is sufficiently small (note that the bound of ϵ here does not depend on $I(0)$), if $R(t)$ never exceeds ϵ, $I(t)$ must reach I^* at some t since I^* is in the interior of $\mathcal{I}^{BO}$. We thus conclude that $\mathcal{E}^{BO}(I^*, I(0))$ must be feasible for all $I(0) \in (0, I^*)$. Furthermore as γ becomes sufficiently small, both $\Delta\overline{U}(I^*)$ and $\Delta\underline{U}(I^*)$ get arbitrarily close to $\Delta U^{BO}(I^*)$ in the SI model, which, again given I^* is in the interior of $\mathcal{I}^{BO}$, implies that $\mathcal{E}^{BO}(I^*, I(0))$ is incentive compatible for all $I(0) \in (0, I^*)$. This completes the proof. □

Appendix C. SIR(ecovered) model

This appendix discusses how our endemic-equilibrium analysis in the SI model can be adapted directly to a Susceptible-Infected-Recovered model in which no one dies from the disease and agents who recover from infection have subsequent immunity to infection.

C.1. Susceptible-infected-recovered analysis

This section extends the endemic-disease analysis of Sections 3.1-3.2 to the SIR(ecovered) model. The key difference of this variation from the SIR(emoved) model is that R-agents remain alive and active and hence continue to enjoy benefit flow $b_0 + b_1$ and are free from health cost flow $-d$. We focus here on how high and how low the long-run prevalence of infection can be in an equilibrium epidemic; the analysis for the outbreak phase is analogous to the SIR(emoved) model in the main text.

Steady-state equilibria. Consider a steady state with masses S, I, and R of susceptible, infected, and recovered agents, respectively. By equation (4), the steady-state condition $R'(t) = 0$ requires that $\gamma I = rR$. Since $S + I + R = 1$, this in turn implies that $S = 1 - \frac{r+\gamma}{r} I$ and $R = \frac{\gamma}{r} I$. By equation (3), the steady-state condition $I'(t) = 0$ requires that $\beta a_S S = r + \gamma$ and hence that S-agent activity $a_S = \frac{r+\gamma}{\beta(1-\frac{r+\gamma}{r} I)}$. Overall, then, there is at most one steady state with mass I of infected agents.

Let $\mathcal{I}^{SSE}(\beta, \gamma, d, r, b_0, b_1, b_2)$ be the set of infection levels I that can be supported in an steady-state equilibrium in the SIR model with transmission rate β, recovery rate γ, disease severity d, death rate r, and payoff parameters (b_1, b_2). Proposition 12 establishes that $\mathcal{I}^{SSE}(\beta, \gamma, d, r, b_0, b_1, b_2)$ is identical to the set of infection levels that can arise in an SSE in an SI model up to a re-scaling, with a suitably-reduced transmission rate ($\widehat{\beta} < \beta$) and disease

severity ($\widehat{d} < d$), and other parameters unchanged. Moreover, as we show in the proof of this result, population-wide economic activity and population-wide suffering from the disease are also identical in corresponding SSEs across the two models.

Proposition 12. $\mathcal{I}^{SSE}(\beta, \gamma, d, r, b_0, b_1, b_2) = \frac{r}{\gamma+r} \times \mathcal{I}^{SSE}(\widehat{\beta}, 0, \widehat{d}, r, b_0, b_1, b_2)$, *where* $\widehat{\beta} = \beta\frac{r}{\gamma+r}$ *and* $\widehat{d} = d\frac{r}{\gamma+r}$.

Proof. Consider the SIR model with parameters $(\beta, \gamma, d, r, b_0, b_1, b_2)$ and suppose that an SSE exists with infection level I. The steady-state epidemiological conditions require that $S = 1 - \frac{r+\gamma}{r}I$, $R = \frac{\gamma}{r}I$, and $a_S = \frac{r+\gamma}{\beta(1-\frac{r+\gamma}{r}I)}$ (discussed earlier). If $a_S < 1$ so that the SSE has partial activity, then incentive-compatibility (IC) holds when S-agents have individual welfare $U_S = 0$ in the steady state, since then agents are indifferent whether or not to be active. Alternatively, if $a_S = 1$ so that the SSE has full activity, then IC holds when $U_S \geq 0$.

From an economic perspective, an SSE is characterized by

$$\begin{aligned} &b_1 + b_2(a_S S + I + R) + \beta I(U_I^{SS}(I) - \frac{b_0}{r}) \\ =&b_1 + b_2(r+\gamma)(\frac{I}{r} + \frac{1}{\beta}) + \beta I(U_I^{SS}(I) - \frac{b_0}{r}) = 0, \end{aligned}$$

where

$$U_I^{SS}(I) = \frac{1}{r}(b_0 + b_1 + b_2(r+\gamma)(\frac{I}{r} + \frac{1}{\beta})) - \frac{d}{r+\gamma}.$$

Therefore an overall condition for $I \in \mathcal{I}^{SSE}(\beta, \gamma, d, r, b_1, b_2)$ is

$$\frac{\beta I + r}{\beta I r}(b_1 + b_2(r+\gamma)(\frac{I}{r} + \frac{1}{\beta})) = \frac{d}{r+\gamma}.$$

For every $\hat{I} \in \mathcal{I}^{SSE}(\widehat{\beta}, 0, \widehat{d}, r, b_1, b_2)$, letting $I = \frac{r}{\gamma+r}\hat{I}$, a condition characterizing I is

$$\frac{\widehat{\beta}\frac{\gamma+r}{r}I + r}{\widehat{\beta}\frac{\gamma+r}{r}Ir}(b_1 + b_2 r(\frac{\frac{\gamma+r}{r}I}{r} + \frac{1}{\widehat{\beta}})) = \frac{\widehat{d}}{r}.$$

It is straightforward to verify that these two conditions are identical when $\widehat{\beta} = \beta\frac{r}{\gamma+r}$ and $\widehat{d} = d\frac{r}{\gamma+r}$. □

Barely-oscillating equilibrium trajectories. As in Section 3.2, define an oscillating trajectory as one in which (i) S-agents alternate regularly between periods of full activity and zero activity and (ii) the prevalence of infection rises from $\underline{I}$ to $\overline{I}$ during each active period and then falls from $\overline{I}$ to $\underline{I}$ during each inactive period. Here we focus on "barely-oscillating trajectories" in which $\underline{I} \approx \overline{I}$, so that the level of infection remains approximately constant over time.

$\mathcal{I}^{BO}(\beta, \gamma, d, r, b_1, b_2)$ be the set of infection levels that can be supported in a barely-oscillating equilibrium trajectory in the SIR model with parameters $(\beta, \gamma, d, r, b_1, b_2)$. Proposition 13 establishes that this, too, is exactly the same in the SIR model and the "corresponding SI model" discussed earlier, with suitably-reduced transmission rate and disease severity.

Proposition 13. $\mathcal{I}^{BO}(\beta, \gamma, d, r, b_0, b_1, b_2) = \frac{r}{\gamma+r} \times \mathcal{I}^{BO}(\widehat{\beta}, 0, \widehat{d}, r, b_0, b_1, b_2)$, *where* $\widehat{\beta} = \beta\frac{r}{\gamma+r}$ *and* $\widehat{d} = d\frac{r}{\gamma+r}$.

Proof. Define S-agents' "average" activity as $a_S^{BO}(I)$. In any barely-OET, a_S^{BO} must satisfy

$$\beta a_S^{BO} SI = I(r+\gamma)$$

which means the average inflow and outflow of the infected measure is the same. With $S+I+R=1$, the above condition gives $a_S^{BO} = \frac{r+\gamma}{\beta(1-\frac{r+\gamma}{r}I)}$. The average overall activity is then

$$A^{BO} = I + R + a_S^{BO}(I)(1-I-R) = \frac{r+\gamma}{r}(I+\frac{r}{\beta}).$$

Then the individual welfare of infected agents is

$$U_I^{BO}(I) = \frac{b_0+b_1+A^{BO}(I)b_2}{r} - \frac{d}{r+\gamma} = \frac{b_0+b_1+\frac{r+\gamma}{r}(I+\frac{r}{\beta})b_2}{r} - \frac{d}{r+\gamma}.$$

Since S-agents die at rate r and become infected at rate $\beta a_S^{BO}(I)I$, they remain susceptible for expected length of time $\frac{1}{r+\beta a_S^{BO}(I)I} = \frac{r+\gamma}{r}\frac{1}{\beta a_S^{BO}(I)}$ and their ex ante likelihood of becoming infected is $\frac{\beta a_S^{BO}(I)I}{r+\beta a_S^{BO}(I)I} = \frac{r+\gamma}{r}I$. We conclude that S-agents have individual welfare

$$\begin{aligned} U_S^{BO}(I) =& \frac{b_0(1-\frac{r+\gamma}{r}I)}{r} + \frac{r+\gamma}{r}\frac{1}{\beta a_S^{BO}(I)}(a_S^{BO}(I)(b_1+b_2)) + \frac{r+\gamma}{r}IU_I^{BO}(I) \\ =& \frac{b_0(1-\frac{r+\gamma}{r}I)}{r} + \frac{r+\gamma}{\beta r}(b_1+b_2+\beta IU_I^{BO}(I)) \end{aligned}$$

The harm of infection is then given by

$$\Delta U^{BO}(I) = \frac{b_0(1-\frac{r+\gamma}{r}I)}{r} + \frac{r+\gamma}{\beta r}(b_1+b_2) - (1-\frac{r+\gamma}{r}I)U_I^{BO}(I).$$

The IC constraints for susceptible agents are

$$\begin{aligned} b_1+b_2 >& \beta I\Delta U^{BO}(I) \\ b_1+b_2\frac{r+\gamma}{r}I <& \beta I\Delta U^{BO}(I). \end{aligned}$$

Note that

$$\begin{aligned} b_1+b_2-\beta I\Delta U^{BO}(I) =& (1-\frac{r+\gamma}{r}I)(b_1+b_2+\beta IU_I^{BO}(I)) - \beta I\frac{b_0(1-\frac{r+\gamma}{r}I)}{r} \\ =& (1-\frac{r+\gamma}{r}I)\frac{\beta r}{r+\gamma}(U_S^{BO}(I)-\frac{b_0}{r}). \end{aligned}$$

Therefore we can rewrite the IC constraints as

$$\begin{aligned} U_S^{BO}(I) >& \frac{b_0}{r} \\ U_S^{BO}(I) <& \frac{b_0}{r} + \frac{r+\gamma}{\beta r}b_2. \end{aligned}$$

Therefore, $\forall I \in \mathcal{I}^{BO}(\beta,\gamma,d,r,b_0,b_1,b_2)$,

$$0 < \frac{r+\gamma}{\beta r}(b_1+b_2+\beta IU_I^{BO}(I)) - \frac{b_0(r+\gamma)I}{r^2} < \frac{r+\gamma}{\beta r}b_2,$$

which is equivalent to

$$0 < b_1 + b_2 + \beta I\left(\frac{b_1 + \frac{r+\gamma}{r}(I + \frac{r}{\beta})b_2}{r} - \frac{d}{r+\gamma}\right) < b_2.$$

On the other hand, for every $\widehat{I} \in \mathcal{I}^{BO}(\widehat{\beta}, 0, \widehat{d}, r, b_1, b_2)$

$$0 < b_1 + b_2 + \widehat{\beta} I\left(\frac{b_1 + (I + \frac{r}{\widehat{\beta}})b_2 - \widehat{d}}{r}\right) < b_2.$$

Let $\widehat{\beta} = \beta\frac{r}{\gamma+r}$ and $\widehat{d} = d\frac{r}{\gamma+r}$, then there is a one-to-one mapping from $\mathcal{I}^{BO}(\widehat{\beta}, 0, \widehat{d}, r, b_0, b_1, b_2)$ to $\mathcal{I}^{BO}(\beta, \gamma, d, r, b_0, b_1, b_2)$ established by $I = \frac{r}{r+\gamma}\widehat{I}$. □

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