Bistability and Resurgent Epidemics in Reinfection Models

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***Abstract*—Spreading processes that propagate through local interactions have been studied in multiple fields (e.g., epidemiology, complex networks, social sciences) using the susceptible-infected-recovered (SIR) and susceptible-infected-susceptible (SIS) frameworks. SIR assumes individuals acquire full immunity to the infection after recovery, while SIS assumes individuals acquire no immunity after recovery. However, in many spreading processes individuals may acquire only partial immunity to the infection or may become more susceptible to reinfection after recovery. We study a model for reinfection called Susceptible-Infected-Recovered-Infected (SIRI). The SIRI model generalizes the SIS and SIR models and allows for study of systems in which the susceptibility of agents changes irreversibly after first exposure to the infection. We show that when the rate of reinfection is higher than the rate of primary infection, the SIRI model exhibits bistability with a small difference in the initial fraction of infected individuals determining whether the infection dies out or spreads through the population. We find this critical value and show that when the infection does not die out there is a resurgent epidemic in which the number of infected individuals decays initially and remains at a low level for an arbitrarily long period of time before rapidly increasing toward an endemic equilibrium in which the fraction of infected individuals is non-zero.**

***Index Terms*—Nonlinear systems, contagion dynamics, compartmental systems.**

I. Introduction

EPIDEMIOLOGICAL models [1] have been widely studied and successfully applied in many settings, including mobile networks [2], rumor spreading [3], and even viral video dynamics [4]. These compartmental models typically describe how the group sizes of different types of individuals evolve over time. The main appeal for these models is their high analytic tractability, which makes them a powerful framework for studying transient and steady-state system behaviors.

These models are also central to the understanding of contagious processes [5]–[7] and to the development of control and optimal resource allocation strategies that seek to inhibit or promote the spread of the process [8]–[10].

Two of the most successful and well-studied epidemiological models are the SIS and SIR models. In the SIS model individuals can be either susceptible or infected. Susceptible individuals become infected through contact with already infected individuals, and return to the susceptible state after recovering from the infection. The SIR model is similar to the SIS model except for the fact that recovered individuals acquire full immunity to the infection, meaning they cannot become infected again.

While the SIS (no immunity) and the SIR (full immunity) models have been extensively used and studied, they do not address many of the applicable real-world situations in which the susceptibility of individuals to primary infections is different from the susceptibility to secondary infections (i.e., reinfections). For instance, in the case of infectious diseases, a lower probability of reinfection corresponds to the development of partial immunity in which primary infections are more likely than secondary infections, such as in the case of influenza [11]. Alternatively, a higher probability of reinfection might correspond to a compromised immune system in which secondary infections are more likely, such as in the case of tuberculosis in particular populations [12].

In the spread of social behaviors, past experiences may lead to differences between primary and secondary infections. A lower probability of reinfection could be the result of a negative experience that reduces the propensity of an individual to further engage in the behavior, while a higher probability of reinfection could result from a positive experience that increases the propensity of an individual to engage in the behavior.

In this letter we study the role of susceptibility to reinfections by considering the spread of a contagious process using the SIRI (Susceptible-Infected-Recovered-Infected) model in which the rate of primary infections is different from the rate of secondary infections. The SIRI model contains the SIS and SIR models as special cases and allows for the study of systems in which individuals become more or less susceptible to the infection after first exposure.

In the theoretical biology literature reinfection models have been used to study the role of partial immunity and waning immunity across populations [13], while in the physics community spatial reinfection models have garnered attention due to their critical behavior connecting directed percolation and dynamic percolation [14]. A Markovian SIRI model on

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arbitrary networks is studied in [15], and it is shown through numerical simulations on random networks that the model exhibits bistability in which a low number of initially infected individuals leads to an infection-free steady-state while a much larger number of initially infected individuals leads to an endemic steady-state. We have found no other work in the literature that examines this bistability. In this letter we formalize the observation of [15] in the case of a well-mixed population by proving new results on the critical initial condition below which the infection dies out and above which solutions reach an endemic equilibrium.

Our contribution in this letter is a rigorous analysis of the SIRI model dynamics over its entire phase space. We identify and prove conditions for each of four different dynamical regimes exhibited by the SIRI model: infection-free, endemic, epidemic, and bistable. As far as we know this is the first such analysis of its kind. We prove that the bistability phenomenon occurs when secondary infections are more likely than primary infections. We prove that when the bistability condition leads to an endemic steady-state, the system exhibits a resurgent epidemic in which the number of infected individuals initially decreases before ramping up after an arbitrarily long delay.

This letter is organized as follows. Section II introduces the SIRI model. In Section III we analyze the dynamics of the SIRI model over the entire phase space and prove conditions for the four dynamical regimes. In Section IV we study the bistable regime in more detail and introduce the concept of a resurgent epidemic with an arbitrarily long delay. We provide closing remarks and discuss future work in Section V.

II. Model Description

Consider a large population in which an individual can be in any of the following three states: *susceptible* (S), *infected* (I), or *recovered* (R). Susceptible and recovered individuals become infected through contact with already infected individuals at respective rates $\beta \geq 0$ and $\hat{\beta} \geq 0$, while infected individuals recover at a fixed rate $\delta \geq 0$:

$$\begin{gathered} S + I \xrightarrow{\beta} I + I \xleftarrow{\hat{\beta}} R + I \\ I \xrightarrow{\delta} R \end{gathered} \tag{1}$$

By assuming that interactions between any two individuals occur with the same probability (i.e., under homogeneous mixing conditions), we can model the system dynamics as

$$\begin{aligned} \dot{x}_S &= -\beta x_S x_I \\ \dot{x}_I &= \beta x_S x_I + \hat{\beta} x_R x_I - \delta x_I \\ \dot{x}_R &= -\hat{\beta} x_R x_I + \delta x_I, \end{aligned} \tag{2}$$

where x_S, x_I, and x_R represent the fractions of population that belong to the susceptible, infected, and recovered states, respectively. Note that $x_S + x_I + x_R = 1$, and this constraint is preserved under (2).

Table I shows the special cases of the SIRI model. Setting $\hat{\beta} = 0$ reduces the SIRI model to the SIR model, while setting $\hat{\beta} = \beta$ and redefining x_S as $x_S + x_R$ reduces the SIRI model to the SIS model. In between the SIR (full immunity) and the SIS (no immunity) models, the rate of secondary infections is larger than zero but lower than the rate of primary infections ($0 < \hat{\beta} < \beta$), and we say that recovered individuals have developed *partial immunity* to the infection, i.e., they are less likely to become reinfected. For example, in the spread of rumors, partial immunity might represent the scenario wherein individuals become less likely to spread new rumors, possibly due to negative consequences of an initially spread rumor. When the rate of secondary infections is larger than that of primary infections ($\beta < \hat{\beta} < \infty$), recovered individuals become reinfected more easily, and we say that recovered individuals have developed *compromised immunity* to the infection. In the example, this could represent the scenario wherein individuals become more willing to spread new rumors, possibly due to benefits from spreading a previous rumor. In the limit $\hat{\beta} \to \infty$, the rate of recovery is negligible compared to the rate of reinfection, and the SIRI model approximates the SI model where infected individuals can never recover. An exact equivalence with the SI model can be achieved by setting the recovery rate δ to zero and redefining x_S as $x_S + x_R$. This could represent the scenario wherein individuals cannot stop spreading rumors once they hear a rumor.

TABLE I
SPECIAL CASES OF THE SIRI MODEL

Parameter Value	Equivalent Model	Immunity Condition
$\hat{\beta} = 0$	SIR	Full Immunity
$0 < \hat{\beta} < \beta$	—	Partial Immunity
$\hat{\beta} = \beta$	SIS	No Immunity
$\beta < \hat{\beta} < \infty$	—	Compromised Immunity
$\delta = 0$	SI	No Recovery

III. Model Analysis

A. Epidemic Analysis

Here we derive conditions on model parameters that guarantee an epidemic, i.e., growth in number of infected individuals for a small initial number of infected individuals.

The dynamics for x_I in (2) can be written as

$$\dot{x}_I = \delta((R_0 x_S + R_1 x_R) - 1)x_I = \delta(R(x_S, x_R) - 1)x_I \tag{3}$$

where $R_0 \triangleq \beta/\delta$, $R_1 \triangleq \hat{\beta}/\delta$, and $R(x_S, x_R) \triangleq R_0 x_S + R_1 x_R$. The infection decays when $R < 1$, grows when $R > 1$, and neither grows nor decays when $R = 1$.

If $R_0 > 1$, a small fraction of initially infected individuals can spread the infection in a population with no recovered individuals ($x_R = 0$). To see this, consider the dynamics for x_I around a point where $x_R = 0$ and $x_I \approx 0$:

$$\dot{x}_I = (\beta - \delta)x_I = \delta(R_0 - 1)x_I. \tag{4}$$

Similarly, if $R_1 > 1$ a small fraction of initially infected individuals can spread the infection in a population with no susceptible individuals ($x_S = 0$). To see this, consider the dynamics for x_I around a point where $x_R \approx 1$ and $x_I \approx 0$:

$$\dot{x}_I = (\hat{\beta} - \delta)x_I = \delta(R_1 - 1)x_I. \tag{5}$$

We can investigate the effect of introducing a small fraction of infected individuals in a population with both susceptible

and recovered individuals by looking at the linearized dynamics of x_I around an infection-free point $x_S = 1 - \bar{x}_R$, $x_I = 0$, and $x_R = \bar{x}_R$,

$$\dot{x}_I = \delta(R_0 + (R_1 - R_0)\bar{x}_R - 1)x_I. \tag{6}$$

For initial conditions where $\bar{x}_R = 0$, by (4) the initial infection spreads if $R_0 > 1$. If all individuals become infected, i.e., $\bar{x}_R = 1$, by (5) the infection spreads through the recovered population if $R_1 > 1$.

When $0 < \bar{x}_R < 1$, the infection spreads if $R_0 + (R_1 - R_0)\bar{x}_R > 1$. So, if $R_0 > R_1$, i.e., $\beta > \hat{\beta}$, as in the case of partial immunity, the effective spreading power of the infection decreases with the fraction of recovered individuals $\bar{x}_R$. That is, recovered individuals are less prone to the infection than susceptible individuals, which makes it harder for the infection to spread. And if $R_0 < R_1$, i.e., $\beta < \hat{\beta}$, as in the case of compromised immunity, recovered individuals are likely to become reinfected and facilitate the spread of the infection through the population.

B. Equilibrium Points and Stability Analysis

The dynamics of the SIRI model (2) evolve on the 2-simplex $\Delta_2 \triangleq \{(x_S, x_I, x_R) \in [0,1]^3 | x_S + x_I + x_R = 1\}$, and the corresponding reduced dynamics can be expressed as

$$\begin{aligned} \dot{x}_S &= -\beta x_S x_I \\ \dot{x}_I &= (\hat{\beta} - \delta)x_I + (\beta - \hat{\beta})x_S x_I - \hat{\beta}x_I^2. \end{aligned} \tag{7}$$

This reduced model (7) has one continuum of equilibria and an isolated equilibrium point:

1) *Infection-Free Equilibria (IFE):* $x_S = x_S^*$, $x_I = 0$,
2) *Endemic Equilibrium (EE):* $x_S = 0$, $x_I = 1 - \delta/\hat{\beta}$.

The IFE is a continuum of equilibria corresponding to the boundary of Δ_2 where $x_I = 0$ and $x_S^* \in [0,1]$, while the EE corresponds to the case in which every individual is either in the infected or recovered state. The SIRI model does not have an equilibrium point where all three states S, I, and R coexist.

We now show how the steady-state solution x_S^* at a point in the IFE depends on the initial conditions.

Theorem 1: The fraction of susceptible individuals x_s^* at a point in the IFE is given by the implicit equation

$$\left(\frac{x_S^*}{x_{S0}}\right)^{R_1/R_0}\left(x_{I0} + x_{S0} - \frac{R_1 - 1}{R_1}\right) - x_S^* + \frac{R_1 - 1}{R_1} = 0,$$

where x_{I0} and x_{S0} are the initial fractions of infected and susceptible individuals, respectively.

Proof: Dividing the two equations in (7) we get an expression for dx_I/dx_S:

$$\frac{dx_I}{dx_S} = \frac{(\hat{\beta} - \beta)}{\beta} - \frac{\hat{\beta} - \delta}{\beta x_S} + \frac{\hat{\beta}x_I}{\beta x_S}$$

with solution

$$x_I = -x_S + \frac{\hat{\beta} - \delta}{\hat{\beta}} + k x_S^{\hat{\beta}/\beta} \tag{8}$$

where the value of k can be found by setting $t = 0$, yielding

$$\frac{x_I + x_S - (\hat{\beta} - \delta)/\hat{\beta}}{x_S^{\hat{\beta}/\beta}} = \frac{x_{I0} + x_{S0} - (\hat{\beta} - \delta)/\hat{\beta}}{x_{S0}^{\hat{\beta}/\beta}}. \tag{9}$$

In the limit $t \to \infty$, $x_I(\infty) = 0$. Simplifying and making the substitution $x_S^* = x_S(\infty)$ we get the implicit equation

$$\left(\frac{x_S^*}{x_{S0}}\right)^{\hat{\beta}/\beta}\left(x_{I0} + x_{S0} - \frac{\hat{\beta} - \delta}{\hat{\beta}}\right) - x_S^* + \frac{\hat{\beta} - \delta}{\hat{\beta}} = 0.$$

Substituting $R_1 = \hat{\beta}/\delta$ completes the proof. ∎

Corollary 1: Given an initial condition $x_I = x_{I0}$, $x_S = 1 - x_{I0}$, where $0 < x_{I0} < 1$, the fraction of susceptible individuals x_s^* at the IFE is given by the implicit equation

$$\frac{1}{R_1}\left(\frac{x_s^*}{1 - x_{I0}}\right)^{R_1/R_0} - x_s^* + \frac{R_1 - 1}{R_1} = 0. \tag{10}$$

Proof: The proof follows by setting $x_{S0} = 1 - x_{I0}$ in Theorem 1. ∎

Before we state the main theorem of this letter, we define the quantity $M \triangleq (1 - R_1)/(R_0 - R_1)$ which we use throughout the rest of this section.

Theorem 2 (Behavioral Regimes of SIRI): Given an initial condition $x_I = x_{I0}$, $x_S = 1 - x_{I0}$, where $0 < x_{I0} < 1$, the SIRI model (7) exhibits four different dynamical behaviors:

1) *Infection-Free:* If $R_0 < 1$ and $R_1 < 1$, then all solutions reach a point in the IFE as $t \to \infty$, and x_I decays monotonically to zero.
2) *Endemic:* If $R_0 > 1$ and $R_1 > 1$, then all solutions reach the EE as $t \to \infty$.
3) *Epidemic:* If $R_0 > 1$ and $R_1 \leq 1$, then all solutions reach a point in the IFE as $t \to \infty$ and, at equilibrium, $x_S^* < M$. For initial conditions where $x_{I0} \geq (\beta - \delta)/\beta$, x_I decays monotonically to zero. While for initial conditions where $x_{I0} < (\beta - \delta)/\beta$, x_I grows initially and reaches a maximum value:
$$x_I^{max} = \frac{R_0 - R_1}{R_1 (R_0^{R_0}(1 - x_{I0})^{R_1})^{1/(R_0 - R_1)}} + \frac{R_1 - 1}{R_1},$$
before decaying to zero as $t \to \infty$.
4) *Bistable:* If $R_0 \leq 1$, $R_1 > 1$, then x_I decays initially. Moreover, there is a critical initial fraction of infected individuals
$$x_{IC} = 1 - M(R_0 M)^{-\frac{R_0}{R_1}}. \tag{11}$$
Solutions with initial condition $x_{I0} < x_{IC}$ reach a point in the IFE as $t \to \infty$ and x_I decays monotonically to zero. Solutions with initial conditions $x_{I0} > x_{IC}$ reach the EE as $t \to \infty$.

Before proving Theorem 2, we prove three lemmas.

Lemma 1: The EE is an equilibrium point of (7) if and only if $R_1 \geq 1$. Moreover, the EE is locally stable.

Proof: To show necessity, note that at the EE we have $x_I = 1 - \delta/\hat{\beta} = 1 - 1/R_1$ which is nonnegative only if $R_1 \geq 1$.

Sufficiency follows from the fact that $x_I = 1 - 1/R_1$, $x_s = 0$ and $x_R = 1 - x_I$ is an equilibrium point of (7).

The Jacobian of (7) around the EE is given by

$$J = \begin{bmatrix} -\beta(\hat{\beta} - \delta)/\hat{\beta} & 0 \\ (\beta - \hat{\beta})(\hat{\beta} - \delta)/\hat{\beta} & -(\hat{\beta} - \delta) \end{bmatrix}$$

which is Hurwitz if $R_1 > 1$. ∎

The following lemma shows that in the epidemic and bistable regimes, the IFE contains both locally stable and unstable equilibrium points.

Lemma 2: The following holds true for the IFE:

1) If $R_0 < 1$ and $R_1 < 1$, then all points in the IFE are locally stable.
2) If $R_0 > 1$ and $R_1 > 1$, then all points in the IFE are unstable.
3) If $R_0 > 1$ and $R_1 \leq 1$, points in the IFE with $x_S^* < M$ are locally stable and points with $x_S^* > M$ are unstable.
4) If $R_0 \leq 1$ and $R_1 > 1$, points in the IFE with $x_S^* > M$ are locally stable and points with $x_S^* < M$ are unstable.

Proof: The Jacobian for the linearized system about $x_I = 0$, $x_S = x_S^*$ is

$$J = \begin{bmatrix} 0 & -\beta x_S^* \\ 0 & (\beta - \hat{\beta})x_S^* + \hat{\beta} - \delta \end{bmatrix} \tag{12}$$

The zero eigenvalue has eigenvector $[1, 0]^T$ corresponding to the invariant subspace $x_I = 0$. The second eigenvalue $J_a = (\beta - \hat{\beta})x_s^* + (\hat{\beta} - \delta)$ determines the local stability of points in the IFE.

To prove 1, assume $R_0 < 1$ and $R_1 < 1$. If $R_0 > R_1$, then $\beta > \hat{\beta}$ and $J_a < 0$ for any $0 \leq x_S^* \leq 1$ and all points in the IFE are locally stable. If $R_0 < R_1$ then $\beta < \hat{\beta}$ and $\max J_a = \beta - \delta < 0$ and all points in the IFE are locally stable.

To prove 2, assume $R_0 > 1$ and $R_1 > 1$. If $R_0 > R_1$, $J_a > 0$ for any $0 \leq x_S^* \leq 1$ and all points in the IFE are unstable. If $R_0 < R_1$ then $\max J_a = \beta - \delta > 0$ and all points in the IFE are unstable.

To prove 3, assume $R_0 > 1$ and $R_1 \leq 1$. It follows that $J_a < 0$ if $0 < x_S^* < M$ and $J_a > 0$ if $M < x_S^* < 1$, which is equivalent to 3.

To prove 4, assume $R_0 \leq 1$ and $R_1 > 1$. It follows that $J_a < 0$ if $M < x_S^* < 1$ and $J_a > 0$ if $0 < x_S^* < M$, which is equivalent to 4. ∎

We rule out the existence of periodic orbits in the SIRI model as this has the implication that any solution starting on Δ_2 must end at either a point in the IFE or the EE.

Lemma 3: The SIRI model does not exhibit non-trivial periodic orbits on Δ_2.

Proof: We rule out the existence of periodic orbits by contradiction. Suppose there is a periodic solution of (7) on Δ_2. Then $x_S(t) = x_S(t')$ for some $t' > t$. Since x_S is nonincreasing in Δ_2, this implies that $\dot{x}_S \equiv 0$ on $[t, t']$ which holds if and only if $x_I x_S \equiv 0$ on $[t, t']$.

If $x_I = 0$ at any time $\bar{t} \in [t, t']$, then the system is at a point in the IFE at time $\bar{t}$. Then $x_I \equiv 0$ on $[\bar{t}, t']$ and the solution is not a non-trivial periodic orbit. If $x_S \equiv 0$ on $[t, t']$, then $x_R = 1 - x_I$ and the dynamics of (7) can be reduced to a single first order ODE. Because periodic orbits cannot take place in a first order system, we have a contradiction. ∎

Proof of Theorem 2: Assume $R_0 < 1$ and $R_1 < 1$. By Lemma 2 the IFE are the only equilibria of the system and by Lemma 3 there are no periodic solutions. These two statements imply that all solutions reach a point in the IFE as $t \to \infty$. To show that all solutions x_I are monotonically decreasing, note that $R < 1$ for any $x_S, x_I \in [0, 1]$. From (3) it follows that $\dot{x}_I < 0$ for $x_I \neq 0$. This completes the proof for 1.

Assume $R_0 > 1$ and $R_1 > 1$. By Lemmas 2 and 1 all points in the IFE are unstable and the EE is locally stable. By Lemma 3 there are no periodic solutions. These statements imply that all solutions reach the EE as $t \to \infty$. This completes the proof for 2.

Assume $R_0 > 1$ and $R_1 \leq 1$. Following the same argument as in the proof of 1, all solutions reach a point in the IFE as $t \to \infty$. From Lemma 2, equilibrium points in the IFE for which $x_S^* > M$ are unstable. Therefore solutions reach points in the IFE where $x_S^* < M$.

At the initial condition $x_I = x_{I0}$, $x_S = 1 - x_{I0}$, with $0 < x_{I0} < 1$, the initial rate of change of x_I by (7) is $((\beta - \delta) - \beta x_{I0})x_{I0}$. It follows that x_I grows initially if $x_{I0} < (\beta - \delta)/\beta$ and decays initially if $x_{I0} > (\beta - \delta)/\beta$.

Points along the solution where $\dot{x}_I = 0$ belong to the x_I-nullcline and satisfy $R = 1$ if $x_I \neq 0$. Since all solutions reach a point in the IFE, solutions that grow initially must reach a maximum value x_I^{max} where $R = 1$. Since solutions cannot intersect, this implies that solutions that decay initially do not change sign and continue to decay monotonically until they reach a point in the IFE.

By setting $\dot{x}_I = 0$ in (7), we can express x_S^{max} (i.e., the maximum value x_S) in terms of x_I^{max} as

$$x_S^{max} = \frac{\delta + \hat{\beta}(x_I^{max} - 1)}{\beta - \hat{\beta}}. \tag{13}$$

Then substituting (13) into (9) and simplifying, we get

$$x_I^{max} = \frac{R_0 - R_1}{R_1 (R_0^{R_0} (1 - x_{I0})^{R_1})^{1/(R_0 - R_1)}} + \frac{R_1 - 1}{R_1}. \tag{14}$$

This completes the proof for 3.

Assume $R_0 \leq 1$, $R_1 > 1$. It follows that at the initial condition $\dot{x}_I < 0$ for any $0 < x_{I0} \leq 1$ and x_I decays initially. A necessary condition for the solution to reach a point in the IFE is for the fraction of susceptible individuals at steady-state x_S^* to satisfy the implicit equation (10) and for the IFE point $(x_S^*, 0)$ to be locally stable.

By Lemma 2 any point in the IFE with $x_S^* < M$ is unstable. Therefore we require $x_S^* > M$. Solving (10) for x_{I0} shows that the necessary condition is satisfied if $x_{I0} < x_{IC} \triangleq 1 - M(R_0 M)^{-R_0/R_1}$. Thus, $x_{I0} < x_{IC}$ is a necessary condition for the solution to reach a point in the IFE.

To prove sufficiency, we show that $\dot{x}_I$ does not change sign when $x_{I0} < x_{IC}$ and therefore solutions with $x_{I0} < x_{IC}$ decrease monotonically.

If $\dot{x}_I$ changes sign, then x_I has a minimum value where $R = 1$. To be a valid minimum of $x_I \in [0, 1]$, we require a minimum value $x_I^{min} \in [0, 1]$. If $x_I^{min} = 0$, then we must have $R = 1$ at $(x_S^*, 0)$. This condition is satisfied when $x_S^* = M$, or equivalently when $x_{I0} = x_{IC}$. Thus, any solution with $x_{I0} < x_{IC}$ cannot have $x_I^{min} > 0$ and $x_I \to 0$ monotonically as $t \to \infty$.

From the discussion above, it follows that a necessary and sufficient condition for the solution not to reach a point in the IFE is $x_{I0} > x_{IC}$. Due to the invariance of Δ_2 and the impossibility of periodic orbits, this implies that $x_{I0} > x_{IC}$ is a necessary and sufficient condition for solutions to reach the EE. This completes the proof. ∎

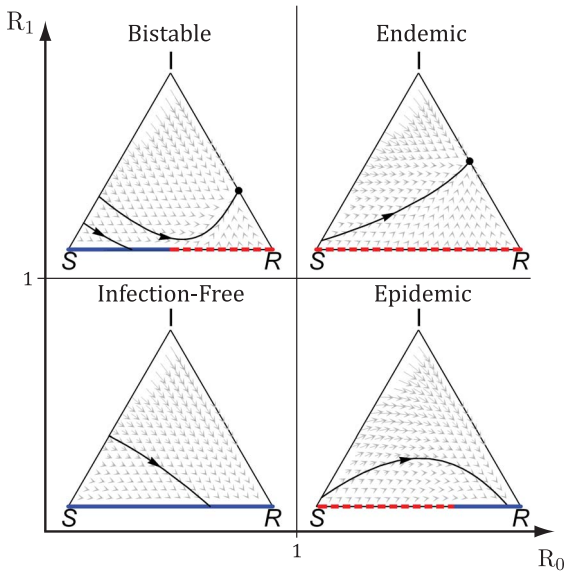

Fig. 1. The four different behavioral regimes of the SIRI model plotted on Δ_2. The four plots are arranged in the R_0, R_1 parameter space to illustrate the four corresponding regimes.

Figure 1 summarizes the results of Theorem 2. In each quadrant of the R_0, R_1 parameter space, we show a simulation of the corresponding dynamics on Δ_2. The bottom boundary of Δ_2 represents the IFE. The solid blue and dashed red lines correspond to locally stable and unstable points in the IFE, respectively. The thinner black lines are example trajectories. We show two trajectories in the bistable case corresponding to a trajectory with $x_{I0} = 0.15$ that reaches a point in the IFE and a trajectory with $x_{I0} = 0.3$ that reaches the EE.

Remark 1: The transient dynamics in the infection-free and endemic regimes depend on the ratio R_0/R_1. When $R_0/R_1 > 1$ recovered individuals inhibit the spread of the infection, leading to concave trajectories in Δ_2. In contrast, when $R_0/R_1 < 1$, recovered individuals facilitate the spread, leading to convex trajectories in Δ_2.

IV. Resurgent Epidemics

In this section we study the bistable regime in more detail and show that when the initial condition is above the critical value solutions exhibit a resurgent epidemic in which the infection initially decreases before increasing after an arbitrarily long period of time.

Theorem 3 (Resurgent Epidemic): Consider a solution in the bistable regime with initial condition $x_{IC} < x_{I0} < 1$ such that the solution reaches the EE as $t \to \infty$. For that solution, the fraction of infected individuals decreases initially, reaches a minimum value

$$x_I^{min} = \frac{R_0 - R_1}{R_1 (R_0^{R_0}(1 - x_{I0})^{R_1})^{1/(R_0 - R_1)}} + \frac{R_1 - 1}{R_1},$$

and then increases until it reaches the EE.

Proof: Assume $R_0 < 1$, $R_1 > 1$, and $x_{I0} > x_{IC}$. From (4) we get that the initial fraction of infected individuals decays exponentially while from result 4 of Theorem 2 we get that the solution reaches the EE as $t \to \infty$.

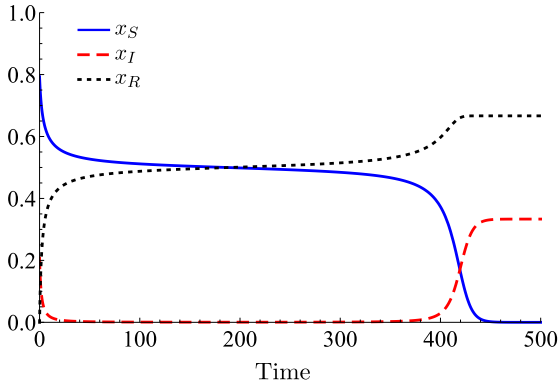

Fig. 2. Resurgent epidemic for $\beta = 0.5$, $\delta = 1$, $\hat{\beta} = 1.5$, and $x_{I0} = 0.207$.

Similar to the analysis for x_I^{max} in the epidemic case, any minimum of x_I must satisfy $R = 1$. This is only satisfied along the portion of the x_I-nullcline between the points $(M, 0)$, which separates the IFE into locally stable and unstable sets, and $(\frac{R_1 - 1}{R_1}, 0)$, which corresponds to the EE. We refer to this portion of the x_I-nullcline as Λ.

To show that all trajectories reach a minimum, note that if a trajectory passes through a point in Λ, that point will correspond to x_I^{min}, the minimum value of x_I along the trajectory. Solving (9) for x_{I0} we get

$$x_{I0} = 1 - Q(R_0 Q)^{-R_0/R_1} \tag{15}$$

where $Q = (1 + R_1(x_I - 1))/(R_0 - R_1)$.

Setting $x_I = 0$ in (15) yields a lower bound on the initial condition x_{I0} that results in a trajectory with a minimum value $x_I^{min} \in [0, 1]$, while setting $x_I = (R_1 - 1)/R_1$ in (15) yields an upper bound on the initial condition x_{I0} that results in a trajectory with a minimum $x_I^{min} \in [0, 1]$.

When $x_I = 0$, $Q = M$ and we get $x_{I0} = x_{IC}$, that is, we recover (11), the critical value for bistability. When $x_I = (R_1 - 1)/R_1$, $Q = 0$ and $x_{I0} = 1$. This shows that any solution with $x_{IC} < x_{I0} < 1$ achieves a minimum value $x_I^{min} \in [0, 1]$.

Finally, note that the same analysis used to find (14) is valid in the bistable case, except that the resulting equation describes the minimum value x_I^{min}. ■

Figure 2 shows a simulation that exhibits resurgent epidemics with $\beta = 0.5$, $\delta = 1$, and $\hat{\beta} = 1.5$. The initial fraction of infected individuals x_{I0} was set to 0.207. The infection decays at first, reaching a value close to zero after 20 time units. The infection stays close to zero for over 350 time units before increasing towards an endemic state where $x_I = 0.33$.

The time it takes before the resurgent epidemic is observed depends on the difference between the initial condition x_{I0} and the critical value x_{IC}. If x_{I0} is close to x_{IC} then the minimum value x_I^{min} will be close to zero and the rate of growth of x_I will be very slow, leading to long time periods where the infection appears to be under control before the epidemic resurges.

To study this phenomenon in more detail, we define the time to resurgence t_{RS} as the time it takes x_I to decay from x_{I0} to the minimum value x_I^{min}. Figure 3 shows t_{RS} versus $x_{I0} > x_{IC}$ for the same parameters as in Figure 2. As the difference $x_{I0} - x_{IC}$ goes to zero, t_{RS} goes to infinity.

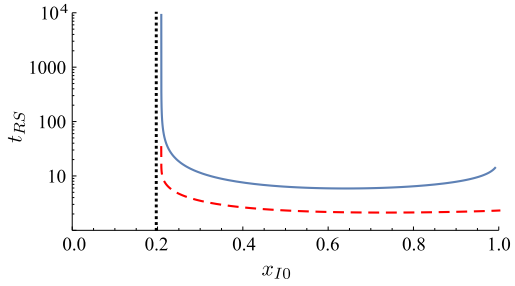

Fig. 3. Numerical simulations (solid) and lower bound in Theorem 4 (dashed) for time to resurgence t_{RS} versus initial condition x_{I0} for $\beta = 0.5$, $\delta = 1$, $\hat{\beta} = 1.5$. The dotted line shows the critical initial condition $x_{IC} = 0.206$.

Theorem 4 (Time to Resurgence): Consider a solution in the bistable regime that exhibits a resurgent epidemic. The time to resurgence $t_{RS} \triangleq t_{min} - t_0$ satisfies the lower bound

$$t_{RS} \geq \frac{\log x_{I0} - \log x_I^{min}}{\delta - \beta},$$

where t_0 is the initial time and t_{min} is the time at which $x_I = x_I^{min}$. Moreover, $t_{RS} \to \infty$ as $x_{I0} - x_{IC} \to 0_+$.

Proof: Recall that close to the initial condition, the dynamics of x_I are given by (4) with solution $x_I(t) = x_{I0}e^{(\beta-\delta)t}$, where $\beta - \delta < 0$. Setting $x_I = x_I^{min}$ and solving for t, we find the time t_d it takes (4) to decay from x_{I0} to x_I^{min}:

$$t_d = \frac{\log x_I^{min} - \log x_{I0}}{\beta - \delta}. \tag{16}$$

Along the solution, $x_I(t) \geq x_{I0}e^{-(\delta-\beta)t}$, which implies $t_{RS} \geq t_d$. In the limit $x_{I0} \to x_{IC}$, $\log x_I^{min} \to -\infty$ and $t_d \to \infty$. ■

V. Conclusion and Future Directions

We have studied the SIRI model for reinfection. We prove that the model has four different behavioral regimes determined by the values R_0 and R_1 that describe the susceptibility of individuals to primary and secondary infections. When both R_0 and R_1 are below or above the critical value of 1, the SIRI model behaves like the SIS model: if $R_0 \leq 1$ and $R_1 \leq 1$ the infection dies out, and if $R_0 > 1$ and $R_1 > 1$ the infection spreads. When $R_0 > 1$ and $R_1 \leq 1$, the SIRI model behaves qualitatively like the SIR model and the infection spreads initially in an epidemic that reaches a maximum number of infected individuals before dying out. Finally, when $R_0 \leq 1$ and $R_1 > 1$, the model displays bistability in which initial conditions below a critical value lead to an infection-free equilibrium while initial conditions above the critical value lead to the infection spreading through the population. We prove that, in the latter case, solutions exhibit a resurgent epidemic in which the infection decreases at first and reaches a minimum value before rapidly increasing after a long delay.

Possible extensions of the SIRI model include an SIRS-like model in which recovering individuals pass through an additional stage with full immunity before transitioning to the recovered state at a fixed rate.

Our results have implications for spreading processes where individuals adapt after first exposure. Common control strategies focus on preventative measures that seek to minimize the number of exposed individuals. However, the resurgent epidemic phenomenon shows that if reinfections are more likely than primary infections, these control strategies might fail at preventing the spread of the process. More effective control strategies should complement prevention of infection with post-exposure treatment and reinfection prevention.

Our results hold under the restrictive assumption of homogeneous interactions. Although in most cases individuals tend to interact in a non-homogeneous manner with other individuals in the population, our assumption provides invaluable intuition into the dynamics, and the analytical tractability of the model has allowed us to obtain rigorous results. The SIS and SIR models have been successfully adapted to network topologies [5], [16], [17]. In ongoing work we are designing and analyzing SIRI dynamics on networks.

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