Coupled Evolutionary Behavioral and Disease Dynamics Under Reinfec tion Risk

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Abstract—In this article, we study the interplay between epidemic dynamics and human decision-making for epidemics that involve reinfec tion risk; in particular, the susceptible–infected–susceptible (SIS) and the susceptible–infected–recovered–infected (SIRI) epidemic models. In the proposed game-theoretic setting, individuals choose whether to adopt protection or not based on the tradeoff between the cost of adopting protection and the risk of infec tion; the latter depends on the current epidemic prevalence and the fraction of individuals who adopt protection in the population. We define the coupled epidemic-behavioral dynamics by modeling the evolution of individual protection adoption behavior according to the replicator dynamics. For the SIS epidemic, we fully characterize the equilibria and their stability properties. We further analyze the coupled dynamics under timescale separation when individual behavior evolves faster than the epidemic, and characterize the equilibria of the resulting discontinuous hybrid system for both SIS and SIRI models. Numerical results illustrate how the coupled dynamics exhibits oscillatory behavior and convergence to sliding mode solutions in suitable parameter regimes.

Index Terms—Epidemic processes, population game, replicator dynamics.

I. INTRODUCTION

INFECTIOUS diseases or epidemics spread through human society via social interactions among infected and healthy individuals. There are two broad classes of epidemic models: 1) epidemics where an individual upon recovery is no longer at risk of future infection (e.g., the susceptible–infected–recovered (SIR) epidemic model) and 2) epidemics where recovery from infection does not lead to immunity from future infections [2], [3]. Motivated by a large number of infectious diseases, where recovered individuals are still at risk of future infection, including COVID-19 [4], we focus on the second class of epidemics in this work; in particular, the susceptible–infected–susceptible (SIS) and the susceptible–infected–recovered–infected (SIRI) [5] epidemic models. In the SIRI model, the infection rate of individuals who have been infected in the past is different from those who have never been infected which captures diseases that impart compromised or strengthened immunity after initial infec tion.

In the absence of suitable medicines or vaccines, particularly at the onset of an epidemic, individuals adopt protective measures (such as wearing masks or maintaining social distancing) in order to avoid becoming infected, often in a strategic and decentralized manner. Thus, the adoption of protective behavior both affects and is affected by the level of infec tion prevalence. Consequently, selfish adoption of protective behavior has been studied in the framework of game theory [6], [7]. For the SIS epidemic, both static or single-shot games to model vaccination decisions [8], [9] and dynamic games that model evolution of protective decisions [10], [11], [12], [13] have been analyzed in the recent past. Specifically, in [10], individuals in a well-mixed population decide whether or not to adopt protection (which eliminates the risk of infec tion) at a certain cost, and epidemic evolution under optimal protection strategies is studied. This setting is generalized in [11] and [13] to include network interactions. The COVID-19 pandemic led to renewed interest in this topic, with several recent papers exploring game-theoretic protection and vaccination strategies for the class of SIR epidemic and its variants [14], [15], [16]. While these works characterize Nash equilibrium strategies under various assumptions on the payoffs and population heterogeneity, they do not explore: 1) (evolutionary) learning dynamics for the decision-makers and 2) equilibria (and their stability) of the epidemic dynamics under game-theoretic strategies.

In particular, the problem of a large group of strategic individuals finding or learning equilibrium strategies is quite challenging [17], [18]. In settings with a large population of agents, evolutionary dynamics have been proposed and their convergence behavior to the set of equilibria has been analyzed [18], [19]. This class of learning dynamics involves repeated play of a static game, where the payoff functions remain unchanged. In contrast with the above settings, epidemic games exhibit a dynamically changing proportion of healthy and infected
individuals. A recent work [20] investigated this dynamic evolution of infection dynamics and strategic decisions for the class of susceptible-asymptomatic-infected-recovered (SAIR) epidemic in the framework of dynamic population games. Coupled evolution of disease and behavior has also been investigated in [21] and [22] for SIRS and SEIR epidemic models, respectively. While the setting in [21] allows for reinfection, the payoff function of an individual does not directly depend on the infection prevalence, rather the payoffs are governed by a dynamic reward term likely designed by a central authority. The authors in [23] considered a coupled SIS epidemic and evolutionary learning model based on mean dynamic and analyzed equilibria when disease dynamics is faster than learning dynamics. In a contemporary work [24], the authors considered a coupled SIS epidemic-behavioral setting in which the payoff contains a social influence factor and imitation dynamics which is used to model the evolution of behavior.

In this article, we build upon the above line of work, and consider the SIS and SIRI epidemic\(^1\) settings, where a large population of individuals chooses whether to adopt protection or to remain unprotected as the epidemic evolves. Adopting protection reduces the infection probability for healthy individuals and transmission probability for infected individuals.\(^2\) The cost of adopting protection is weighed with the instantaneous risk of becoming infected; the latter depends on the current epidemic prevalence and the proportions of individuals in different epidemic states adopting protection. We focus on the replicator dynamics [18], [19] to model the evolution of protection decisions and study their interaction with SIS and SIRI dynamics. Our choice is motivated by the fact that the replicator dynamics is one of the most well-studied evolutionary learning dynamics and has seen widespread applications in biological, environmental, and socioeconomic settings [25], [26], [27], [28], [29]. The major contributions of this work are stated as follows.

First, for the coupled SIS epidemic and replicator dynamics, we completely characterize the equilibria (existence and local stability) and show how the stability of different equilibrium points get exchanged as certain parameters change in Section II. We further explore the behavior of the coupled dynamics under timescale separation leading to a slow-fast dynamical system [30]. Second, for the coupled SIRI epidemic and replicator dynamics, we analyze possible equilibria under timescale separation in Section III. For both (SIS and SIRI) coupled dynamics, we specifically focus on the case where the replicator dynamics are faster.\(^3\) We characterize the asymptotic convergence of the infected proportion under game-theoretic strategies to an equilibrium by analyzing the resulting slow dynamics which takes the form of a variable structure system in the limiting regime. We also show that the infected proportion may converge to an endemic sliding mode of the hybrid dynamics for a certain range of parameters. Finally, we provide insights into the transient behavior of these coupled dynamics using numerical simulations in Section IV. Particularly, we illustrate the impact of the timescale separation parameter and oscillatory convergence to the equilibrium point. Finally, Section V concludes this article.

## II. COUPLED SIS EPIDEMIC AND EVOLUTIONARY BEHAVIORAL MODEL

In this section, we formally introduce the coupled evolution of the SIS epidemic and protection adoption behavior in a homogeneous large-population setting. Let the proportion of susceptible and infected individuals be \(s(t)\) and \(y(t)\), respectively. Both \(s(t), y(t) \in [0, 1]\) with \(s(t) + y(t) = 1\) for all \(t \geq 0\). We adopt a population game framework [18], where individuals choose whether to adopt protection against the epidemic or not; these actions are denoted by \(P\) and \(U\). Consequently, the population state at time \(t\) is defined as \(x(t) := [x_{SU}(t) \ x_{SP}(t) \ x_{IU}(t) \ x_{IP}(t)]\top \in \Delta_4\), where \(\Delta_4\) is the probability simplex in \(\mathbb{R}^4\), \(x_{SU}\) denotes the proportion of individuals who are susceptible and choose to remain unprotected, \(x_{IP}\) denotes the proportion of infected individuals who adopt protection, and so on. At time \(t\), we have \(x_{SU}(t) + x_{SP}(t) = s(t)\), \(x_{IU}(t) + x_{IP}(t) = y(t)\), and \(\mathbf{1}^\top x(t) = 1\), where \(\mathbf{1}\) is a vector of appropriate dimension with all entries 1. Thus, \(x(t)\) encodes the joint strategies of the entire population of agents.

Individuals choose their action to maximize their payoffs which depend on the population state \(x(t)\) (i.e., the joint strategy profile, which also includes information regarding infection prevalence). For an infected individual, there is no further risk of infection and, as a result, we define its payoff to be constant parameters given by \(-c_{IU}\) if it remains unprotected and \(-c_{IP}\) if it adopts protection. For instance, \(c_{IU} > 0\) captures the cost of breaking isolation/quarantine protocols for an infected individual. Thus, we assume \(c_{IU} > c_{IP} \geq 0\).

A susceptible individual trades off the cost of adopting protection, denoted by \(c_P > 0\), and expected cost of becoming infected. The latter is the product of the loss upon infection \(L > 0\)\(^4\) and the instantaneous probability of becoming infected which depends on its action and the strategies of other agents (captured in the population state). Specifically, let \(\beta_{SP}\) and \(\beta_P\) denote the probabilities of an infected individual causing a new infection if it is unprotected and protected, respectively. We impose the natural assumption \(\beta_{SP} > \beta_P \geq 0\) throughout this article. Consequently, the instantaneous probability of infection for an unprotected susceptible individual at population state \(x\) is given by \(\beta_{SP}x_{IU} + \beta_Px_{IP}\). Similarly, let a protected susceptible individual be \(\alpha \in (0, 1)\) times (less) likely to become infected compared to an unprotected susceptible individual. Thus, the instantaneous probability of infection for a protected susceptible individual at population state \(x\) is given by \(\alpha(\beta_{SP}x_{IU} + \beta_Px_{IP})\).

The payoff vector of individuals at population state \(x\) is now
defined as

\[
F(x) := \begin{bmatrix}
F_{SU}(x) \\
F_{SP}(x) \\
F_{TU}(x) \\
F_{TP}(x)
\end{bmatrix} = \begin{bmatrix}
-L(\beta_S x_{TU} + \beta_P x_{TP}) \\
-c_p - L\alpha(\beta_S x_{TU} + \beta_P x_{TP}) \\
-c_{TU} \\
-c_{TP}
\end{bmatrix}
\]  

where \(F_{SU}(x)\) denotes the payoff for an individual who is susceptible and unprotected at population state \(x\) (and, thus, depends on the joint strategy profile), and so on. Susceptible individuals who adopt protection pay a cost \(c_p\) but experience a reduced infection risk scaled by factor \(\alpha\), as discussed above.

While most of the past works have proceeded to directly analyze the equilibrium strategies after introducing the payoff function, we here consider the evolutionary learning dynamics under which individual protection decisions evolve. We first introduce some notation. Let \(z_S(t) \in [0,1]\) denote the fraction of susceptible individuals who remain unprotected, i.e., \(x_{SU}(t) = z_S(t)s(t)\) and \(x_{SP}(t) = (1 - z_S(t))s(t)\). Similarly, let \(z_I(t) \in [0,1]\) denote the fraction of infected individuals who remain unprotected. Due to the presence of both unprotected and protected individuals with different infection probabilities, the infected proportion evolves as

\[
\dot{y}(t) = (x_{SU}(t) + \alpha x_{SP}(t))(\beta_S x_{TU}(t) + \beta_P x_{TP}(t)) - \gamma y(t) = [(1 - y(t))(z_S(t) + \alpha(1 - z_S(t))) \\
\cdot (\beta_S z_I(t) + \beta_P(1 - z_I(t))) - \gamma] y(t) =: f_P(y(t), z_S(t), z_I(t))
\]

where \(\gamma\) is the rate of recovery for infected individuals. The above dynamics is analogous to the conventional scalar SIS epidemic dynamics with an effective infection rate \(\beta_{eff}(z_S, z_I) = (z_S + \alpha(1 - z_S))(\beta_S z_I + \beta_P(1 - z_I))\) which now depends on the efficacy of protection and the fractions that adopt protection. If \(\alpha = 1\) and \(\beta_S = \beta_P = \beta\), i.e., protection is not effective, the effective infection rate is \(\beta\), which is the setting in classical SIS epidemic without protection. Note further that in contrast with the classical SIS epidemic setting, \(\beta_{eff}(z_S, z_I)\) is time-varying as the fractions of susceptible and infected individuals adopting protection evolves with time in accordance with evolutionary learning dynamics, as discussed in the following.

We focus on the class of replicator dynamics [18], [25] in this work and assume that susceptible individuals only replicate the strategies of other susceptible individuals (likewise for infected individuals). For susceptible individuals, we obtain

\[
\dot{z}_S(t) = z_S(t)(1 - z_S(t)) [F_{SU}(x) - F_{SP}(x)] = z_S(t)(1 - z_S(t)) [c_p - L(1 - \alpha)(\beta_S z_I(t) + \beta_P(1 - z_I(t)))y(t)] =: f_S(y(t), z_S(t), z_I(t)).
\]

Similarly, for infected individuals, we have

\[
\dot{z}_I(t) = z_I(t)(1 - z_I(t)) (c_{TP} - c_{TU}) =: f_I(y(t), z_S(t), z_I(t)).
\]

Thus, (2)–(4) characterize the coupled evolution of the epidemic and population states which remain confined to the set \([0,1]^3\), as shown in the following.

**Lemma 1 (Invariant Set of Coupled SIS Epidemic):** For the coupled SIS epidemic and evolutionary behavioral dynamics defined by (2)–(4), the set \(\{(y, z_S, z_I) | (y, z_S, z_I) \in [0,1]^3\}\) is invariant.

**Proof:** It is easy to see that when \(y(t) = 0, \dot{y} = 0\) and when \(y(t) = 1, \dot{y} < 0\). Likewise, for \(z_I, z_S \in \{0,1\}, \dot{z}_I = \dot{z}_S = 0\).

The result follows from Nagumo’s theorem [31, Th. 4.7].

**A. Equilibrium Characterization and Stability Analysis**

We now examine the equilibrium points of the above coupled epidemic-replicator dynamics and their stability properties. First, we consider the evolution of \(z_I\) in (4) which does not depend on \(y\) and \(z_S\). There are two stationary points \(z_I = 0\) and \(z_I = 1\), and it is easy to see that for \(c_{TP} > c_{TU}\), \(z_I = 1\) is unstable and \(z_I = 0\) is exponentially stable with the basin of attraction \([0,1]\). It is also quite intuitive that if \(c_{TP} < c_{TU}\), infected individuals prefer to use protection and the strategy for infected individuals should converge to it.

Thus, in the rest of this section, we only focus on equilibria with \(z_I = 0\). We begin with introducing a few variables that will be used to define the equilibrium points

\[
y_0^* := 1 - \frac{\gamma}{\beta_p}; \quad y_1^* := 1 - \frac{\gamma}{\alpha \beta_p}; \quad y_{int} := \frac{c_p}{L(1 - \alpha) \beta_p};
\]

and \(z_{S, int}^* := \frac{1}{1 - \alpha} \left[ 1 - \frac{\gamma}{\beta_p(1 - y_{int})} \right] \).

We now define all possible equilibria \((y^*_1, z_S^*, z_I^*)\) of the coupled SIS epidemic and evolutionary behavior dynamics (2)–(4) corresponding to \(z_I = 0\)

\[
E_0 = (0, 0, 0), \quad E_1 = (0, 1, 0), \quad E_2 = (y_0^*, 1, 0), \quad E_3 = (y_{int}^*, z_S_{int}^*, 0), \quad E_4 = (y_1^*, 0, 0).
\]

At \(E_0\), everyone adopts protection and there is no infection. At \(E_1\), there is no infection, and susceptible individuals do not adopt protection. \(E_2\) is an endemic equilibrium, i.e., a fraction of the population is infected, and susceptible individuals continue to remain unprotected. \(E_3\) is an endemic equilibrium where a fraction of susceptible individuals adopts protection. Finally, \(E_4\) is an endemic equilibrium at which all susceptible individuals adopt protection. The existence and local stability of these equilibria are established in the following proposition, whose proof is presented in Appendix A.

**Proposition 1 (Equilibria and Stability):** For the equilibrium points of the coupled SIS epidemic and evolutionary behavioral dynamics (2)–(4) corresponding to \(z_I = 0\), the following statements hold.

1. \(E_0\) exists for all parameter regimes, and is unstable.
2. \(E_1\) exists for all parameter regimes, is locally stable if \(\beta_P < \gamma\), and is unstable, otherwise.
3. \(E_2\) exists only when \(\beta_P > \gamma\), is locally stable when \(y_0^* < y_{int}^*\), and is unstable otherwise.
4) \( E_3 \) exists only when \( \gamma < \beta_p \) and \( y_p^* < y_{int}^* < y_0^* \), and is locally stable.
5) \( E_4 \) exists only when \( \gamma < \alpha \beta_p \), is locally stable when \( y_p^* > y_{int}^* \), and is unstable otherwise.

**Remark 1:** While the above result shows local stability of the equilibrium points, we can show global attractivity if we restrict the coupled dynamics to the \((y, z_1)\) plane, i.e., by setting \( z_1 = 0 \) for the planar dynamics (2) and (3). Note that all equilibrium points except \( E_3 \) lie on the boundary of the invariant set \([0, 1]^2\) for the planar dynamics. Thus, when \( E_3 \) does not exist (i.e., in all regimes other than 4) in Proposition 1, it follows from index theory [32, Sec. 6.8] that no limit cycle exists. Since at any given set of parameter values, there is exactly one equilibrium point which is locally stable, it is globally attractive as well.

**Remark 2:** As the recovery rate \( \gamma \) increases, the infected proportion at the stable equilibrium decreases. In practice, \( \gamma \) is improved via direct intervention of authorities by augmenting healthcare facilities. When further resource augmentation is not possible, a more effective protection scheme with a smaller value of \( \alpha \) would result in a smaller value of \( y_p^* \). In addition, if \( \alpha \) is reduced further such that \( \gamma > \alpha \beta_p \), the dynamics exhibits a new stable endemic infection level \( y_{int}^* \), which also decreases in \( \alpha \).

Thus, our result shows that indirect intervention by facilitating availability of more effective protection schemes would significantly contribute toward a smaller endemic infection level under game-theoretic strategies.

**Remark 3:** While the above result holds under the assumption \( c_{IU} > c_{IP} \), the results for the case \( c_{IU} < c_{IP} \) are analogous with \( z_1 = 1 \) being the stable equilibrium for the infected population. In fact, when the proportion of infected agents adopting protection is a constant \( z_1^* \), then the results presented above would continue to hold by redefining \( \beta_p := \beta_0 z_1^* + \beta_p (1 - z_1^*) \).

Similarly, when \( \alpha = 0 \), \( y_p^* = -\infty \) and \( E_4 \) will cease to exist as an equilibrium point. The equilibrium behavior of the coupled dynamics will continue to be governed by the first four cases of the above proposition.

**B. Bifurcation Analysis**

The above proposition shows that the equilibrium points exchange stability properties as certain parameters, e.g., \( \gamma, \beta_p, \alpha \), vary. We now numerically explore the bifurcations associated with the transition of stability among the equilibria. For the numerical illustration we choose the parameter values in (2)-(4), as follows.

\[
\begin{array}{cccccc}
\beta_p & \alpha & \beta_0 & c_{IU} & L & c_{IP} \\
1 & 0.5 & 0.3 & 2 & 80 & 0.15 & 1
\end{array}
\]

We adopt the recovery rate \( \gamma \) as a bifurcation parameter and use the numerical continuation package MATCONT [33] to compute the bifurcation diagram shown in Fig. 1.

For \( \gamma \to 0^+ \), \( E_4 \) is the stable equilibrium, while \( E_0, E_1, \) and \( E_2 \) are unstable. As the value of \( \gamma \) is increased at point T3 in Fig. 1, \( E_4 \) exchanges stability to \( E_3 \) in a transcritical bifurcation. Note that unstable branch of \( E_3 \) is not visible since it is associated with negative values of \( z_S \). As \( \gamma \) is increased, the fraction of susceptible population that adopts protection decreases and at \( T_2, E_3 \) exchanges stability to \( E_2 \) in another transcritical bifurcation. Again, the unstable branch of \( E_3 \) at \( T_2 \) corresponds to \( z_s > 1 \) and is not visible in the diagram. Upon further increasing \( \gamma \), the fraction of infected population continues to decrease and at \( T_1, E_2 \) exchanges stability with the disease-free equilibrium \( E_1 \). The unstable branch of \( E_2 \) at \( T_1 \) corresponds to negative values of \( y \).

Another transcritical bifurcation takes place at \( T_0 (\gamma = \alpha \beta_p) \), where \( E_0 \) and \( E_4 \) cross. For \( \gamma < \alpha \beta_p \) (respectively, \( \gamma > \alpha \beta_p \)), \( E_0 \) has two (respectively, one) eigenvalues in the right-half plane. As \( E_4 \) approaches \( T_0 \) from \( y > 0 \), it has one eigenvalue in the right-half plane, while for \( y < 0 \) near \( T_0, E_4 \) is stable.

Thus, the transcritical bifurcation at \( T_0 \) corresponds to exchange of stable and unstable eigenvalues of two unstable equilibria.

It can be verified that each of the above bifurcations are indeed transcritical. For example, at \( \gamma = \beta_p \), \( E_1 \) and \( E_2 \) exchange stability. It follows from the proof of Proposition 1 that at \( \gamma = \beta_p \), \( J_{E1} = J_{E2} \) has only one eigenvalue at zero and the associated left and right eigenvectors are \( e_1^\top \) and \( e_1 \), where \( e_1 = [1 \ 0 \ 0]^\top \). Let \( \xi = [y \ z_S \ z_I]^\top \) and \( f(\xi) = [f_y(\xi) \ f_{z_S}(\xi) \ f_{z_I}(\xi)]^\top \). Then, it can be verified that \( e_1^\top (\partial^2 f / \partial \gamma \partial \xi)(e_1) = -1 \neq 0 \) at \( (\gamma, y, z_S, z_I) = (\beta_p, 0, 1, 0) \).

In addition, \( e_1^\top (\partial^2 \bigr( f / \partial \xi^2 \bigl( e_1, e_1 \bigl) = -2 \gamma \beta_p \neq 0 \) at \( (\gamma, y, z_S, z_I) = (\beta_p, 0, 1, 0) \). Thus, the bifurcation at \( \gamma = \beta_p \) is transcritical [34, Sec. 3.4]. The other bifurcations can be analyzed similarly.

**C. Coupled Epidemic-Behavioral Dynamics Under Timescale Separation**

We have thus far assumed that the epidemic and the replicator dynamics evolve at the same time-scale. However, it is not strictly necessary for the coupled dynamics. In order to obtain further insights into their behavior, we now study the coupled epidemic-behavioral dynamics (2)-(4) under timescale separation. In particular, we focus on the case in which the replicator dynamics evolves faster than the epidemic dynamics; indeed in the modern era, there is an increased awareness about infectious diseases due to publicly available testing data, awareness campaigns by public health authorities, and spread of information via social media, which shapes human response...
at a much faster time-scale. To this end, we model the coupled dynamics as a slow-fast system
\[
\begin{align*}
\dot{y}(t) &= f_p(y(t), z_S(t), z_I(t)) \\
\epsilon \dot{z}_S(t) &= f_S(y(t), z_S(t), z_I(t)) \\
\epsilon \dot{z}_I(t) &= f_I(y(t), z_S(t), z_I(t))
\end{align*}
\]
where \( \epsilon \in (0, 1) \) is a timescale separation variable [30].

At a given epidemic prevalence \( y \), we characterize the (stable) equilibria of the fast system involving the replicator dynamics with states \((z_S, z_I)\). For reasons discussed earlier, we focus on equilibria with \( z_I = 0 \). It is now easy to see that if \( y \neq y^*_{\text{int}} \), there are two equilibrium points: \((0, 0)\) and \((1, 0)\). If \( y = y^*_{\text{int}} \), then \((z_S, 0)\) is an equilibrium point of the fast system for any \( z_S \in [0, 1] \). Following analogous arguments, as in the proof of Proposition 1, it follows that \((0, 0)\) is locally stable for the fast system when \( y > y^*_{\text{int}} \) and \((1, 0)\) is locally stable for the fast system when \( y < y^*_{\text{int}} \). Consequently, we obtain the following reduced dynamics for the slow system which approximates the coupled dynamics (5) in the limit \( \epsilon \to 0 \) as:
\[
\dot{y}(t) = \begin{cases} 
(1 - y(t))\beta_p - \gamma & \text{if } y(t) < y^*_{\text{int}} \\
(1 - y(t))\alpha\beta_p - \gamma & \text{if } y(t) > y^*_{\text{int}}
\end{cases}
\]
and if \( y(t) = y^*_{\text{int}} \), we have
\[
\dot{y}(t) \in \{(1 - y^*_{\text{int}})\beta_p(zS + \alpha(1 - zS)) - \gamma \} y^*_{\text{int}} \mid zS \in [0, 1] \}.
\]

In particular, since the reduced dynamics is an instance of a discontinuous dynamical system, we define the dynamics at the point of discontinuity \( y = y^*_{\text{int}} \) as a differential inclusion which is also the convex combination of the dynamics on both sides of \( y = y^*_{\text{int}} \). It is easy to see that the right-hand side (RHS) of the dynamics is measurable and is locally essentially bounded, and therefore (6) admits a Filippov solution [35, Proposition 3]. We now establish the convergence of \( y(t) \) under (6).

**Proposition 2 (Trajectories under fast behavioral response):** For the epidemic dynamics (6) with \( y(0) \neq 0 \), the following statements hold.

1. If \( y^*_0 \leq 0 \), then \( y(t) \) monotonically decreases and converges to the origin.
2. If \( 0 < y^*_0 < y^*_{\text{int}} \), then \( y(t) \) monotonically converges to \( y^*_0 \).
3. If \( y^*_0 < y^*_{\text{int}} < y^*_p \), then \( y(t) \) converges to \( y^*_{\text{int}} \) which acts as a sliding mode of the dynamics.
4. If \( y^*_{\text{int}} < y^*_p \), then \( y(t) \) monotonically converges to \( y^*_p \).

The proof is omitted due to space constraints and is available in our preprint [36, Appendix A.2].

**Remark 4:** The dynamics in (6) potentially represents a class of nonpharmaceutical interventions, where authorities impose social distancing measures that reduces the infection rate by a factor \( \alpha \) when the infection prevalence exceeds a threshold. Thus, Proposition 2 is potentially of independent interest. Further, the above proposition generalizes analogous results obtained in prior works [10], [13] which assumed \( \alpha = 0 \), i.e., protection completely eliminates risk of infection.

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**III. COUPLED SIRI EPIDEMIC AND EVOLUTIONARY BEHAVIORAL MODEL**

In the SIS epidemic model, a recovered individual encounters the same infection rate as an individual who has never been infected. However, in many infectious diseases, initial infection could lead to compromised immunity [37] or it might even lead to reduced risk of future infection [4], [38]. The SIRI epidemic model [5], captures the above characteristics. In this section, we investigate the implications of game-theoretic protection decisions on the evolution of the SIRI epidemic dynamics.

In the SIRI epidemic model, an individual belongs to one of three possible compartments or states: 1) susceptible, 2) infected, and 3) recovered; while the proportions of individuals in each of the above states at time \( t \) are denoted by \( s(t), y(t), \) and \( r(t) \), respectively. The evolution of these proportions is given by
\[
\begin{align*}
\dot{s}(t) &= -\beta s(t)y(t) \\
\dot{y}(t) &= \beta s(t)y(t) + \beta r(t)y(t) - \gamma y(t) \\
\dot{r}(t) &= \gamma y(t) - \beta r(t)y(t)
\end{align*}
\]
where \( \beta > 0 \) is the rate at which susceptible individuals become infected if they encounter an infected individual, \( \beta > 0 \) is the rate at which recovered individuals become infected, and \( \gamma \) is the rate of recovery for infected individuals. The above transitions are depicted in Fig. 2.

Thus, the above model captures settings where an individual develops immunity upon becoming infected (\( \beta > \beta \)) and when the immunity of an individual is compromised upon infection (\( \beta < \beta \)). When \( \beta = \beta \) and susceptible and recovered states are combined, we recover the SIS epidemic model. The classical SIR model is also obtained as a special case when \( \beta = 0 \).

The dynamics in (7) admits a continuum of equilibria which are infection free (IFE) and may also have an isolated endemic equilibrium (EE), where infection level is nonzero. Mathematically, at the IFE, we have \( s = s^*, y = 0, r = 1 - s^* \), where \( s^* \in [0, 1] \). At the EE, we have \( s = 0, y = 1 - \frac{3}{\hat{\beta}}, r = \frac{\gamma}{\hat{\beta}} \). The existence and stability of these equilibria were analyzed in [5]. We now introduce a population game model to capture how individuals adopt protective behavior, followed by analyzing the resulting evolution of the SIRI epidemic.

**A. Population Game Model of Protection Adoption**

We build upon the formulation in the previous section. Consider a large population of individuals, each in one of three possible infection states, who choose whether to adopt...
protection or remain unprotected. The population state $x := [x_{SG} \ x_{SP} \ x_{I} \ x_{IP} \ x_{Rg} \ x_{RP}]^{\top} \in \Delta_{6}$, where $x_{SG}$ denotes the proportion of recovered individuals who remain unprotected, $x_{RP}$ denotes the proportion of recovered individuals who adopt protection, and the other states are as described earlier. At time $t$, we have $x_{SG}(t) + x_{SP}(t) = s(t), x_{IP}(t) = y(t)$, and $x_{Rg}(t) + x_{RP}(t) = r(t)$, and $t^{\top}x(t) = 1$.

A susceptible individual becomes infected at a rate $\beta_{p} \geq 0$ ($\beta_{p} < 0$) if it comes in contact with an infected individual who adopts protection (remains unprotected). Similarly, a recovered individual becomes reinfected at rate $\beta_{p} \geq 0$ ($\beta_{p} < 0$) if it comes in contact with an infected and unprotected (protected) individual. A susceptible or recovered individual is $\alpha \in (0, 1)$ times less likely to become infected (reinfected) if it adopts protection compared to an unprotected individual with same disease status. Building upon the discussion in the previous section, we define the payoff vector as

$$F(x) := \begin{bmatrix} F_{SG}(x) \\ F_{SP}(x) \\ F_{I}(x) \\ F_{IP}(x) \\ F_{Rg}(x) \\ F_{RP}(x) \end{bmatrix} = \begin{bmatrix} -L(\beta_{S}x_{I} + \beta_{p}x_{IP}) \\ -c_{p} - L(\beta_{S}x_{I} + \beta_{p}x_{IP}) \\ -c_{I} \\ -c_{I} \\ -L(\beta_{S}x_{I} + \beta_{p}x_{IP}) \\ -c_{p} - L(\beta_{S}x_{I} + \beta_{p}x_{IP}) \end{bmatrix}$$

where $L, c_{p}, c_{I},$ and $c_{IP}$ are as defined earlier.

The payoffs for susceptible and infected individuals defined above coincide with the payoffs in case of the SIS epidemic stated in (1). Due to risk of reinfection, recovered individuals behave in a similar manner as susceptible individuals and evaluate the tradeoff between cost of adopting protection and the instantaneous infection risk while choosing their protection status. As before, we assume $c_{I} > c_{IP} \geq 0$.

We denote the time-varying proportions of susceptible, infected, and recovered individuals who choose to remain unprotected $z_{S}, z_{I},$ and $z_{R}$, respectively. In particular, we have $x_{SG} = z_{p}r^{\top}, x_{SP} = (1 - z_{R})r^{\top},$ and so on. These proportions evolve according to the replicator dynamics with payoffs defined in (8). We now state the coupled evolution of disease and evolutionary behavioral dynamics as follows:

$$\dot{s} = -(\beta_{S}z_{I} + \beta_{p}(1 - z_{I}))(z_{S} + \alpha(1 - z_{S}))sy$$

$$\dot{y} = (\beta_{S}z_{I} + \beta_{p}(1 - z_{I}))(z_{S} + \alpha(1 - z_{S}))sy$$

$$+ (\beta_{p}z_{I} + \beta_{S}(1 - z_{I}))z_{R} + \alpha(1 - z_{R}))ry - \gamma y$$

$$\dot{r} = -(\beta_{S}z_{I} + \beta_{p}(1 - z_{I}))z_{R} + \alpha(1 - z_{R}))ry + \gamma y$$

$$\dot{z}_{S} = z_{S}(1 - z_{S})(c_{p} - L(1 - \alpha)(\beta_{S}z_{I} + \beta_{p}(1 - z_{I})))$$

$$\dot{z}_{I} = z_{I}(1 - z_{I})(c_{IP} - c_{I})$$

$$\dot{z}_{R} = z_{R}(1 - z_{R})(c_{p} - L(1 - \alpha)(\beta_{p}z_{I} + \beta_{S}(1 - z_{I})))$$

Lemma 2 (Invariant Set for Coupled SIRI Dynamics): For the coupled SIRI epidemic and evolutionary behavioral dynamics defined by (9), the set $\{(s, y, r, z_{S}, z_{I}, z_{R})|(s, y, r, z_{S}, z_{I}, z_{R}) \in [0, 1]^{6}\}$ is invariant. The proof follows from identical arguments as the proof of Lemma 1 and is omitted due to space constraints.

A complete characterization of the equilibria and their stability properties for the above dynamics is prohibitive due to the dynamics being high-dimensional and the presence of a large number of equilibrium points. In order to gain insights into epidemic evolution and convergence of infected proportion under game-theoretic decision-making, we analyze the coupled dynamics via timescale separation arguments. Specifically, we analyze in detail the case when the behavioral dynamics is much faster than the disease dynamics, derive the stable equilibria of the fast system (consisting of variables $z_{S}, z_{I}, z_{R}$) followed by analyzing the equilibria and convergence behavior of the reduced dynamics for the slow system (consisting of variables $s, y, r$).

B. Equilibria of the Replicator Dynamics Evolving on the Faster Timescale

At a given infection prevalence $y$, we now characterize the equilibria of the behavioral dynamics (9d)-(9f).

Proposition 3: Let $\tilde{y}_{int} := \frac{c_{p}}{L(1 - \alpha)\beta_{p}}$ and $\tilde{y}_{int} := \frac{c_{p}}{L(1 - \alpha)\beta_{S}}$.

Then, the following statements hold for dynamics (9d)-(9f).

1) Any equilibrium with $z_{I} = 1$ is unstable.

2) For the dynamics (9d) and (9e), $(z_{S}, z_{I})$ with values

$\alpha (1, 0)$ is the stable equilibrium when $y < \tilde{y}_{int}$;

$b (0, 0)$ is the stable equilibrium when $y > \tilde{y}_{int}$;

c) any $z_{S} \in [0, 1], z_{I} = 0$ is an equilibrium when $y = \tilde{y}_{int}$.

3) For the dynamics (9e) and (9f), $(z_{I}, z_{R})$ with values

$\alpha (0, 1)$ is the stable equilibrium when $y < \tilde{y}_{int}$;

$b (0, 0)$ is the stable equilibrium when $y > \tilde{y}_{int}$;

c) any $z_{R} \in [0, 1], z_{I} = 0$ is an equilibrium when $y = \tilde{y}_{int}$.

The proof is straightforward and is omitted. In fact, the stable equilibria of the above behavioral dynamics corresponds to the Nash equilibrium strategies of the underlying population game defined by the payoff vector (8) at a given infection prevalence $y$. In particular, when $y = \tilde{y}_{int}$, susceptible individuals are indifferent between adopting protection or remaining unprotected. When $y > \tilde{y}_{int}$, it is optimal to adopt protection which leads to $z_{S} = 0$ being the stable equilibrium, and when $y < \tilde{y}_{int}$, it is optimal to remain unprotected which leads to $z_{S} = 1$ being stable. Stability of $z_{R}$ is analogous.

We now analyze the epidemic dynamics under timescale separation when the population instantly converges to the Nash equilibrium strategies depending on the current value of $y$. We consider two cases: 1) $\beta_{p} > \beta_{S}$; and 2) $\beta_{p} < \beta_{p}$ separately.

C. Epidemic Evolution Under Strengthened Immunity Upon Infection

We first consider the case when $\beta_{p} > \beta_{S}$, i.e., compared to a recovered individual, a susceptible individual becomes infected at a higher rate from a protected infected individual. In other words, initial infection strengthens the immunity of the individual against subsequent infections. In this case, we have $y_{int}^{*} < \tilde{y}_{int}$. The reduced epidemic dynamics for infected and recovered subpopulations, where $z_{S}$ and $z_{R}$ are replaced by their
stable equilibrium values in accordance with Proposition 3, is a hybrid system given by
\[
y \in [0, y_{\text{int}}) : \begin{cases}
\dot{y} = [\beta_p (1-r-y) + \hat{\beta}_p r - \gamma]y \\
\dot{r} = [-\hat{\beta}_p r + \gamma]y
\end{cases}
\] (10a)
y = y_{\text{int}} : \begin{cases}
\dot{y} \in \{[(z_S + \alpha (1-z_S) \beta_p (1-r-y) + \hat{\beta}_p r - \gamma)]y \mid z_S \in [0,1]\}
\dot{r} \in \{[-\hat{\beta}_p (z_S + \alpha (1-z_S)r + \gamma)]y
\end{cases}
\] (10b)
y \in (y_{\text{int}}, \hat{y}_{\text{int}}) : \begin{cases}
\dot{y} = [\alpha \beta_p (1-r-y) + \hat{\beta}_p r - \gamma]y \\
\dot{r} = [-\alpha \hat{\beta}_p r + \gamma]y
\end{cases}
\] (10c)
y \in (\hat{y}_{\text{int}}, 1) : \begin{cases}
\dot{y} = [\alpha \beta_p (1-r-y) + \hat{\beta}_p r - \gamma]y \\
\dot{r} = [-\alpha \hat{\beta}_p r + \gamma]y.
\end{cases}
\] (10d)
\]

In particular, when \( y < y_{\text{int}} < \hat{y}_{\text{int}} \), it follows from the previous subsection that the stable equilibrium of the fast system [behavioral replicator dynamics (9d)–(9f)] is \( z_S = 1, z_I = 0, z_R = 1 \). Setting these values in (10b) and (10c), together with the observation \( s = 1 - y - r \), yields the disease dynamics stated in (10a). The dynamics for moderate and high prevalence of infection stated in (10c) and (10e) are obtained in an analogous manner. The dynamics at points of discontinuities are defined via differential inclusions. As before, it is easy to see that (10) admits a Filippov solution.

We now characterize the equilibria \((y^*, r^*)\) of the dynamics in (10). The following three types of equilibria are possible:

\[ E1 = (0, r^*), \quad \text{for} \quad r^* \in [0,1], \]

\[ E2 = \left(1 - \frac{\gamma}{\hat{\beta}_p}, \frac{\gamma}{\hat{\beta}_p}\right), \quad E3 = \left(1 - \frac{\gamma}{\alpha \hat{\beta}_p}, \frac{\gamma}{\alpha \hat{\beta}_p}\right). \]

E1 corresponds to a continuum of IFE, where the proportion of infected population is 0, while the proportion of recovered population depends on the initial condition and the values of the other parameters. This set of equilibria always exists for the dynamics in (10). E2 and E3 are endemic equilibrium points with a nonzero proportion of infected population and the susceptible proportion being 0. The existence and stability of all these equilibria as well as a sliding mode solution are established as follows.

**Proposition 4 (Equilibria and Stability of (10)):** For the SIRI epidemic under game-theoretic protection stated in (10), the following statements hold for any \( y(0) \in (0,1] \).

1) If \( \gamma > \beta_p \), E2 and E3 are not equilibrium points, the set of the IFE is globally asymptotically stable, and \( y(t) \) decays monotonically to 0.
2) If \( \beta_p < \gamma < \beta_p \), E2 and E3 are not equilibrium points, the sets of IFE with \( r^* \leq \frac{\beta_p - \gamma}{\beta_p - \hat{\beta}_p} \) are unstable and globally asymptotically stable, respectively.
3) If \( \beta_p[1 - \hat{y}_{\text{int}}] < \gamma < \beta_p \), E3 is not an equilibrium point, the set of the IFE is unstable, and E2 is an (almost) globally asymptotically stable equilibrium point.
4) If \( \alpha \beta_p[1 - \hat{y}_{\text{int}}] < \gamma < \beta_p[1 - y_{\text{int}}] \), then E2 and E3 are not equilibrium points, the set of the IFE is unstable and \( (y_{\text{int}}, 1 - y_{\text{int}}) \) acts as a sliding mode of (10) with \( y(t) \to \hat{y}_{\text{int}} \) as \( t \to \infty \).
5) If \( \gamma < \alpha \beta_p[1 - \hat{y}_{\text{int}}] \), E2 is not an equilibrium point, E3 is an (almost) globally asymptotically stable equilibrium point, and the set of the IFE is unstable.

The proof is presented in Appendix B and exploits the notion of input-to-state stability [39]. Note that in a certain parameter regime (Case 4 in the above proposition), the infection free equilibria are not stable, endemic equilibria do not exist and infected fraction converges to a sliding mode of the hybrid dynamics. This sliding mode corresponds to an equilibrium of the original coupled dynamics (9) given by \( s = 0, y = \hat{y}_{\text{int}}, r = 1 - \hat{y}_{\text{int}}, z_S = 0, z_I = 0, z_R = \frac{1}{1 - \alpha \hat{\beta}_p} \). It is easy to see that for the range of \( \gamma \) in Case 4, \( z_R \in (0,1) \), i.e., the outcome is an intermediate level of protection adoption by recovered individuals.

**D. Epidemic Evolution Under Compromised Immunity**

We now consider the case when \( \beta_p < \hat{\beta}_p \), i.e., initial infection leads to compromised immunity against future infections. Here, we have \( y_{\text{int}} > \hat{y}_{\text{int}} \). The reduced epidemic dynamics for infected and recovered subpopulations is given by

\[
y \in [0, \hat{y}_{\text{int}}) : \begin{cases}
\dot{y} = [\beta_p (1-r-y) + \hat{\beta}_p r - \gamma]y \\
\dot{r} = [-\hat{\beta}_p r + \gamma]y
\end{cases}
\] (11a)
y \in (\hat{y}_{\text{int}}, y_{\text{int}}) : \begin{cases}
\dot{y} = [\beta_p (1-r-y) + \alpha \hat{\beta}_p r - \gamma]y \\
\dot{r} = [-\alpha \hat{\beta}_p r + \gamma]y
\end{cases}
\] (11b)
y \in (y_{\text{int}}, 1) : \begin{cases}
\dot{y} = [\alpha \beta_p (1-r-y) + \alpha \hat{\beta}_p r - \gamma]y \\
\dot{r} = [-\alpha \hat{\beta}_p r + \gamma]y.
\end{cases}
\] (11c)

and the dynamics at \( y = y_{\text{int}} \) and \( y = \hat{y}_{\text{int}} \) can be written in terms of differential inclusions similar to (10). The above hybrid dynamics is obtained by setting \( z_S \) and \( z_R \) values to their stable equilibrium values in accordance with Proposition 3. The equilibria of (11) coincide with those of (10), i.e., the following equilibria exist:

\[ E1 = (0, r^*), \quad \text{for} \quad r^* \in [0,1], \]

\[ E2 = \left(1 - \frac{\gamma}{\beta_p}, \frac{\gamma}{\beta_p}\right), \quad E3 = \left(1 - \frac{\gamma}{\alpha \beta_p}, \frac{\gamma}{\alpha \beta_p}\right). \]

The existence and local stability of all these equilibria as well as a sliding mode solution are established as follows with the proof presented in Appendix C.

**Proposition 5 (Equilibria and Stability of (11)):** For the SIRI epidemic under game-theoretic protection stated in (11), the following statements hold.
1) If $\gamma > \hat{\beta}_p$, E2 and E3 are not equilibrium points, the set of the IFE is globally asymptotically stable, and $y(t)$ decays monotonically to 0.

2) If $\hat{\beta}_p [1 - \hat{y}_{\text{int}}] < \gamma < \hat{\beta}_p$, E2 is an equilibrium point which is locally asymptotically stable, and E3 is not an equilibrium point.

3) If $\alpha \hat{\beta}_p [1 - \hat{y}_{\text{int}}] < \gamma < \hat{\beta}_p [1 - \hat{y}_{\text{int}}]$, then E2 and E3 are not equilibrium points, and $(\hat{y}_{\text{int}}, 1 - \hat{y}_{\text{int}})$ acts as a sliding mode of (11).

4) If $\gamma < \alpha \hat{\beta}_p [1 - \hat{y}_{\text{int}}]$, E2 is not an equilibrium point, E3 is a locally asymptotically stable equilibrium point.

In addition, for statements 2–4 above, 1) if $\gamma < \beta_p$, then all points in the IFE E1 are unstable; 2) if $\gamma > \beta_p$, then the sets of the IFE with $r^* \leq \frac{\beta_p - \gamma}{\beta_p - \beta_p}$ are locally stable and unstable, respectively.

**Remark 5:** Note that when $\gamma > \beta_p$ and $\gamma$ satisfies any of the conditions in statements 2–4 of Proposition 5, the hybrid dynamics exhibits bistability, where both the IFE and an endemic equilibrium or attractive sliding mode coexist and are stable. In contrast, when $\beta_p > \beta_p$, multiple stable equilibria do not coexist. Due to the coexistence of multiple stable equilibria, characterizing the respective regions of attraction remains a direction for future research.

**IV. NUMERICAL RESULTS**

**A. SIS Epidemic Setting**

Herein, we first investigate the SIS epidemic setting. We simulate the dynamics in (5) for $\epsilon \in \{0.01, 0.1, 1\}$. The same model parameters, as in Section II-B, are selected and $\gamma$ is selected as 0.1 so that E3 is the stable equilibrium point. Fig. 3 (right panel) shows the time-evolution of $y$ and $z_S$. More oscillatory behavior is observed as $\epsilon$ becomes smaller, i.e., the behavioral dynamics becomes faster. To understand this, we focus on $z_S$ dynamics (3) with

$$\Delta F = c_p - L(1 - \alpha)(\beta_0 z_I(t) + \beta_p (1 - z_I(t)))y(t)$$

as a dynamic parameter. To this end, we illustrate $z_S$ trajectories in $z_S - \Delta F$ plane in Fig. 3 (left panel). Recall that if $\Delta F$ is a positive (respectively, negative) constant, then $z_S = 1$ (respectively, $z_S = 0$) is a stable equilibrium point. Accordingly, $z_S = 0$ and $z_S = 1$ are marked blue and red in Fig. 3 (left panel), when they are stable and unstable, respectively.

Since behavioral dynamics is fast, $y$ is quasi-stationary and $z_I$ very quickly converges to zero. In Fig. 3 (left panel), the initial fraction of infected population is sufficiently high such that $\Delta F < 0$, then the fast behavioral dynamics quickly converges to $z_S = 0$ (the bottom solid blue line), i.e., every susceptible individual adopts protection. This results in a decrease in the fraction of infected population and increases $\Delta F$. As $\Delta F$ becomes positive, $z_S = 0$ becomes unstable and $z_S$ quickly jumps to $z_S = 1$ (the top solid blue line), and a similar process repeats which again drives $\Delta F$ to negative values. This process leads to the highly oscillatory behavior seen in Fig. 3. Eventually, trajectories converge such that $\Delta F = 0$ and $z_S$ settles to equilibrium value $z_S^{*\text{int}}$.

**B. SIRI Epidemic Setting**

We first consider the case where initial infection leads to strengthened immunity. In order to highlight a wide range of transient behavior of the coupled dynamics, we choose parameter values as $\beta_p = 0.3, \hat{\beta}_p = 0.2, \beta_0 = 0.4, \hat{\beta}_0 = 0.25, L = 75, \alpha = 0.6, c_p = 2, c_{\text{pr}} = 2$, and $c_{\text{pr}} = 1.5$. For this set of parameter values, we have $\hat{y}_{\text{int}} = 0.2222$ and $\hat{y}_{\text{int}} = 0.3333$. Fig. 4 shows the evolution of infected proportion $y(t)$ for three different values of the recovery rate $\gamma$ with initial states as $y(0) = 0.2, r(0) = 0.01, z_S(0) = z_I(0) = z_R(0) = 0.5$. In particular, we compute the trajectories of the dynamics in (9) with $\epsilon$ multiplied to the RHS of (9a)–(9c) via the Runge–Kutta fourth-order method with spacing 0.05. When $\epsilon = 1$, epidemic and replicator dynamics evolve at the same timescale while a smaller value of $\epsilon$ signifies a faster evolution of behavior compared to disease evolution.

When $\gamma = 0.15 \in (\hat{\beta}_p - \beta_{\text{int}}, \beta_p)$, it follows from Proposition 4 that points at the infection free equilibria are not stable, E3 does not exist while E2 is a locally stable equilibrium. The plot in the left panel of Fig. 4 shows indeed $y(t)$ converges to the endemic infection level at E2 in our simulations. Furthermore, the convergence is not monotonic. When $\gamma = 0.1, y(t)$ converges to the endemic infection level $\hat{y}_{\text{int}}$ in accordance with Case 4 of Proposition 4. The convergence is in an oscillatory manner similar to the setting illustrated above for the SIS epidemic. Finally, when $\gamma = 0.078 < \alpha \hat{\beta}_p [1 - \hat{y}_{\text{int}}], y(t)$ converges to the endemic infection level at E3.

Finally, we consider the case where initial infection leads to compromised immunity. We choose parameter values as $\beta_p = 0.25, \hat{\beta}_0 = 0.4, \hat{\beta}_0 = 0.35, L = 125, \alpha = 0.6, c_p = 2, c_{\text{pr}} = 2$, and $c_{\text{pr}} = 1$. For this set of parameter values, we
Evolution of infected proportion in the SIRI model for different values of recovery rate $\gamma$ and timescale separation parameter $\epsilon$ in accordance with Proposition 4 when initial infection leads to strengthened immunity.

Evolution of infected proportion and bistable behavior in the SIRI model for different values of $\gamma$ and $\epsilon$ when initial infection leads to compromised immunity in accordance with Proposition 5.

Finally, when $\beta_p = 0.15 > \gamma$, we no longer have bistable behavior and $y(t)$ converges to $\hat{y}_{\text{int}}^*$ for all values of $\epsilon$ even when $y(0) = 0.001$, as shown in the right panel of Fig. 5.

V. Conclusion

In this article, we propose and analyze a novel model that captures the interaction of epidemic propagation dynamics with behavioral dynamics of human protection adoption. For the coupled SIS epidemic-replicator dynamics, we characterized the equilibrium points, their stability properties, and the associated bifurcations. For both SIS and SIRI epidemic models, we further analyzed the coupled dynamics under timescale separation, and established global convergence results to the equilibrium of the reduced epidemic dynamics. Numerical results showed that the relative speed of evolutionary learning compared to the disease dynamics plays a critical role in the transient behavior of the coupled dynamics, which may induce highly oscillatory behavior, and in the bistable regime of the SIRI epidemic, it may have a strong influence on which equilibrium the infected proportion converges to.

Thus, our results highlight that in order to influence and control epidemic prevalence, it is critical to understand not only the equilibrium behavior of humans but also the transient evolution of human behavior in a comparable time-scale as the disease dynamics. We plan to build upon the results derived in this article, and design dynamic intervention schemes (for example, dynamically varying the cost of protection) that guarantee convergence of the disease dynamics to desired equilibrium points. Similarly, it would be interesting to consider a setting where the the payoff of infected individuals also depends on the proportion of infected individuals, for instance due to social or peer influence. Another possible factor is bounded rationality of agents that may lead to a proportion of infected agents not adopting protection. We hope that our work stimulates further investigations along the above lines.

APPENDIX

A. Proof of Proposition 1

Proof: It can be verified that $E_0$ and $E_1$ are always equilibria of the coupled dynamics. The Jacobian matrix at $E_0$ and $E_1$

\[^6\text{See [36, Appendix A] for detailed expressions of the elements of the Jacobian matrix which are omitted from here due to space constraints.}\]
are

$$J_{E0} = \begin{bmatrix} \alpha \beta_p - \gamma & 0 & 0 \\ 0 & c_p & 0 \\ 0 & 0 & c_{IP} - c_{IU} \end{bmatrix},$$

$$J_{E1} = \begin{bmatrix} \beta_p - \gamma & 0 & 0 \\ 0 & -c_p & 0 \\ 0 & 0 & c_{IP} - c_{IU} \end{bmatrix}.$$

$J_{E0}$ has a positive eigenvalue if $c_p > 0$. Thus, for any nonzero cost of adopting protection, $E0$ is not a stable equilibrium point of the coupled dynamics. Likewise, $E1$ is stable if $\beta_p < \gamma$, and unstable, otherwise.

We now analyze existence and local stability of equilibrium points, where infection is endemic. It can be verified that $E2$ exists only when $\beta_p > \gamma$ as otherwise $y_0^* < 0$. The Jacobian matrix at $E2$ is

$$J_{E2} = \begin{bmatrix} d_1 & d_2 & d_3 \\ 0 & -[c_p - L(1 - \alpha)\beta_p y_0] & 0 \\ 0 & 0 & c_{IP} - c_{IU} \end{bmatrix},$$

where $d_1 = (1 - 2y_0^*)\beta_p - \gamma$, $d_2 = y_0^*(1 - y_0^*)(1 - \alpha)\beta_p > 0$, and $d_3 = y_0^*(1 - y_0^*)\beta_p > 0$. Thus, $J_{E2}$ is an upper triangular matrix. Furthermore, the first diagonal entry is

$$-(1 - 2y_0^*)\beta_p - \gamma = \left(1 - 2 \left(1 - \frac{\gamma}{\beta_p}\right)\right)\beta_p - \gamma = \left(-1 + 2\frac{\gamma}{\beta_p}\right)\beta_p - \gamma = \beta_p - \gamma < 0$$

in the regime where $E2$ exists. Therefore, $E2$ is stable when $c_p > L(1 - \alpha)\beta_p y_0^* \Longleftrightarrow y_0^* < y_{int}$.

It can be verified that $E3 = (y_{int}^*, z_{S, int}^*, 0)$ is an equilibrium point. We now examine the conditions under which $y_{int}^* \in (0, 1)$ and $z_{S, int}^* \in (0, 1)$. By definition, $y_{int}^* > 0$. We note that

$$z_{S, int}^* > 0 \iff \frac{\gamma}{\alpha \beta_p} > 1 - y_{int}^* \iff y_{int}^* > 1 - \frac{\gamma}{\alpha \beta_p}$$

and

$$z_{S, int}^* < 1 \iff \frac{\gamma}{\beta_p} < 1 - y_{int}^* \iff y_{int}^* > 1 - \frac{\gamma}{\beta_p}.$$

Thus, $E3$ exists when $y_0^* < y_{int}^*$. Note that the third row of the Jacobian of the dynamics at $E3$, $J_{E3}$, would be $[0 \ 0 \ c_{IP} - c_{IU}]$ as before, and as a result, $c_{IP} - c_{IU} < 0$ would be an eigenvalue. Thus, we focus on the 2 x 2 submatrix containing the first two rows and columns of the Jacobian matrix which simplifies to

$$J_{E3} = \begin{bmatrix} \frac{-\gamma y_{int}^*}{1 - z_{S, int}^*} & d_4 \\ -z_{S, int}^* (1 - z_{S, int}^*)L(1 - \alpha)\beta_p & 0 \end{bmatrix},$$

where $d_4 = y_{int}^*(1 - y_{int}^*)(1 - \alpha)\beta_p > 0$. For the above matrix, the sum of the eigenvalues is negative and the determinant is positive, and as a result, $J_{E3}$ is Hurwitz. Therefore, $E3$, when it exists, is a stable equilibrium of the coupled dynamics.

It can be verified that $E4$ exists when $y_0^* \in (0, 1)$ or equivalently, when $\gamma < \alpha \beta_p$. The Jacobian matrix at $E4$ is

$$J_{E4} = \begin{bmatrix} (1 - 2y_0^*)\alpha \beta_p - \gamma & d_5 & d_6 \\ 0 & d_7 & 0 \\ 0 & 0 & c_{IP} - c_{IU} \end{bmatrix}$$

where $d_5 = y_0^*(1 - y_0^*)(1 - \alpha)\beta_p > 0$, $d_6 = y_0^*(1 - y_0^*)\alpha(\beta_0 - \beta_p) > 0$, and $d_7 = c_p - L(1 - \alpha)\beta_p y_0^*$. Thus, $J_{E4}$ is an upper triangular matrix with the first diagonal entry

$$(1 - 2y_0^*)\alpha \beta_p - \gamma = \gamma - \alpha \beta_p < 0$$

in the regime, where $E4$ exists. Thus, $E4$ is stable if $c_p < L(1 - \alpha)\beta_p y_0^* \iff y_0^* > y_{int}$.

This concludes the proof.

B. Proof of Proposition 4

**Proof:** We leverage results on equilibrium existence and stability of the classical SIRI epidemic shown in [5] in our proof. These results are omitted from this manuscript due to space constraints and are collected in our preprint [36, Th. 1]. We prove each of the cases in the following.

Case 1: $\gamma > \beta_p$. Since $\alpha \in (0, 1)$ together with $\gamma > \beta_p > \hat{\beta}_p$, we have $1 - \frac{\alpha \beta_p}{\gamma} < 1 - \frac{\gamma}{\beta_p} < 0$. In other words, the endemic infection level is negative at $E2$ and $E3$. As a result, $E2$ and $E3$ are not equilibrium points. The local stability of the IFE depends on the dynamics (10). When $1 < \frac{\beta_p - \beta_0}{\beta_p - \beta_0 - \beta_p} < 1$ for the dynamics (10a). Since $\alpha < 1$, we have $R_0 < 1$ and $R_1 < 1$ for the dynamics (10c) and (10e) as well. As a result, following [36, Th. 1, Case 1], the set of IFE of (10) is locally stable and $y(t)$ decays monotonically to 0.

Case 2: $\beta_p < \gamma < \beta_0$. $E2$ and $E3$ not being equilibrium points follows from the arguments in Case 1 above. We have $R_0 < \frac{\beta_p - \beta_0}{\beta_p - \beta_0 - \beta_p} < 1$ and $R_1 < \frac{\beta_p - \beta_0}{\beta_p - \beta_0 - \beta_p}$ for the dynamics (10a). Following [36, Th. 1, Case 3], the set of the IFE with $s^* < \frac{1 - R_1 - R_0}{1 - R_0}$ or equivalently with $s^* > 1 - \frac{1 - R_1 - R_0}{1 - R_0}$ is locally stable, and unstable otherwise.

We now argue that any trajectory of (10) with $y(0) \in (0, 1)$ converges to a stable IFE. Recall that any IFE point $(y = 0, r = \bar{r})$ is unstable if $\bar{r} \in [0, \frac{\beta_p - \beta_0}{\beta_p - \beta_0 - \beta_p}]$ [5, Lemma 2].

Suppose $s(0) < \bar{s} : = 1 - \frac{\beta_p - \beta_0}{\beta_p - \beta_0 - \beta_p}$. Consequently, we have $r(0) < \frac{\beta_p - \beta_0}{\beta_p - \beta_0 - \beta_p}$. Since the possible IFE at the above initial condition are unstable, the vector field around $y = 0$ points toward increasing value of $y$. Let $y(0) \neq 0$ and let $t_0, \epsilon_0 > 0$ be suitable constants such that $y(t) \geq \epsilon_0$ for all $t \geq t_0$ until $s(t) > \bar{s}$. Thus, there exists $c > 0$ such that under (10)

$$\dot{s} = -(\dot{y} + \bar{r}) \leq -c \beta_p \epsilon_0 s \Rightarrow s(t) \leq s(0) e^{-c \beta_p \epsilon_0 t}$$

until $s(t) > \bar{s}$. As a result, there exists $T_0$ such that $s(t) \leq \bar{s}$ for $t > T_0$. We exploit this property to prove convergence to a stable IFE. First, we show that if $y(t) > \bar{s}_{int}$, infected proportion eventually decreases. Indeed, we have

$$\dot{y} = [\alpha \beta_p s + \alpha \beta_p (1 - s - y) - \gamma] y$$
\[
\begin{align*}
= [\alpha(\beta_\bar{p} - \beta_p)s + \alpha\beta_\bar{p}(1 - y) - \gamma]y \\
\leq [\alpha(\beta_\bar{p} - \beta_p)s + \alpha\beta_\bar{p}(1 - y) - \gamma]y \\
= [\alpha(\gamma - \beta_\bar{p}) + \alpha\beta_\bar{p}(1 - y) - \gamma]y \\
= [-\alpha\beta_\bar{p}y + (1 - \alpha)\gamma]y < 0
\end{align*}
\]

since \( \alpha \in (0, 1) \). In other words, when \( s(t) \leq \bar{s} \) for \( t > T_0 \), \( \dot{y}(t) < 0 \) for \( y > \bar{y}_{\text{int}} \), and as a result, the trajectory will eventually remain confined to (10a) and (10c).

Next, we show that if \( y(t) \in (\bar{y}_{\text{int}}, \bar{y}_{\text{int}}^*), \) infected proportion eventually decreases as well. In this regime, we have
\[
\begin{align*}
\dot{y} &= [\alpha\beta_p s + \beta_\bar{p}(1 - s - y) - \gamma]y \\
&= [(\alpha\beta_p - \beta_\bar{p})s + \beta_\bar{p}(1 - y) - \gamma]y \\
&< [(\beta_\bar{p} - \beta_p)s + \beta_\bar{p}(1 - y) - \gamma]y \\
&= [\gamma - \beta_\bar{p} + \beta_\bar{p}(1 - y) - \gamma]y = -\beta_\bar{p}y^2 < 0
\end{align*}
\]
when \( s(t) \leq \bar{s} \) for \( t > T_0 \). Finally, when the trajectory is eventually confined to (10a), i.e., \( y(t) \in (0, \bar{y}_{\text{int}}) \), we have
\[
\begin{align*}
\dot{y} &= [\beta_\bar{p}s + \beta_\bar{p}(1 - s - y) - \gamma]y \\
&\leq [(\beta_\bar{p} - \beta_p)s + \beta_\bar{p}(1 - y) - \gamma]y \\
&= [\gamma - \beta_\bar{p} + \beta_\bar{p}(1 - y) - \gamma]y = -\beta_\bar{p}y^2 < 0.
\end{align*}
\]
Therefore, when \( s(t) \leq \bar{s} \), \( y(t) \) is monotonically decreasing for all \( y \in (0, 1) \) and thus, the infected proportion asymptotically converges to an IFE.

Case 3: \( \beta_\bar{p}[1 - \bar{y}_{\text{int}}] < \gamma < \beta_p \). For the dynamics (10a), we have \( R_0 = \frac{\gamma}{\beta_p} > 1 \) and \( R_1 = \frac{\gamma}{\beta_\bar{p}} > 1 \) in this regime. Following [36, Th. 1], we conclude that all points in the IFE are unstable. Since E3 is the endemic equilibrium for (10e), its existence as an endemic equilibrium for (10) requires
\[
1 - \frac{\gamma}{\alpha\beta_p} \geq \bar{y}_{\text{int}}^* \iff \alpha\beta_p[1 - \bar{y}_{\text{int}}] > \gamma
\]
which is not satisfied in this parameter regime as \( \alpha < 1 \).

Now observe that E2 is the endemic equilibrium for both (10a) and (10c), but not for the dynamics (10e). Thus, for E2 to be an equilibrium for the hybrid system (10), we must have
\[
0 < 1 - \frac{\gamma}{\beta_p} < \bar{y}_{\text{int}}^*
\]
or, equivalently, \( \beta_\bar{p} > \gamma \) and \( \beta_p[1 - \bar{y}_{\text{int}}^*] < \gamma \), i.e., precisely the parameter regime in this case. Further, for both (10a) and (10c), \( R_1 = \frac{\gamma}{\beta_\bar{p}} > 1 \), and as a result from [36, Th. 1], E2 is locally stable. Let \( y_{E2} = 1 - \frac{\gamma}{\beta_\bar{p}} \).

It remains to show that the infected proportion \( y(t) \) converges to the endemic level at E2. Since all points on the IFE are unstable, the vector field at the IFE points toward increasing value of \( y(t) \). Let \( y(0) \neq 0 \) and let \( t_0, e_0 > 0 \) be suitable constants such that \( y(t) \geq e_0 \) for all \( t \geq t_0 \). Thus, there exists \( c > 0 \) such that under (10)
\[
\dot{s} = -(\dot{y} + \dot{r}) \leq -c\beta_p e_y s \Rightarrow s(t) \leq s(0)e^{-c\beta_p e_y t}.
\]
As a result, for any \( \epsilon_s > 0 \), there exists \( T_{\epsilon_s} \) such that \( s(t) \leq \epsilon_s \) for \( t > T_{\epsilon_s} \). In other words, \( s(t) \) acts as a vanishing input to the dynamics of infected proportion.

\[
\begin{align*}
\dot{y} &= [\alpha\beta_p s + \alpha\beta_\bar{p}(1 - s - y) - \gamma]y \\
&\leq [\alpha(\beta_\bar{p} - \beta_p)e_s + \alpha\beta_\bar{p}(1 - y - y_{E2})]y \\
&< [\alpha(\beta_\bar{p} - \beta_p)e_s + \beta_\bar{p}(y_{E2} - y)]y.
\end{align*}
\]
Since the second term is strictly negative for \( y > \bar{y}_{\text{int}} \), there exists an \( e_s \) and \( T_0 \) such that \( \dot{y}(t) < 0 \) for \( t > T_0 \), and as a result, the trajectory will eventually remain confined to (10a) and (10c). We now consider the following two subcases.

Case 3A: \( y_{E2} \in (0, \bar{y}_{\text{int}}) \). Following analogous arguments as above, we first claim that there exists \( T'_0 \) such that \( \dot{y}(t) < 0 \) for \( t > T'_0 \), and as a result, the trajectory eventually remains confined to (10a). Observe now that the dynamics of infected proportion in (10a) can be viewed as a perturbed system with \( s(t) = 1 - r(t) - y(t) \) playing the role of a vanishing input. We now show that the equilibrium point E2 is input-to-state stable (ISS) [39]. Let \( V(y) = (y - y_{E2})^2 \) be the candidate ISS-Lyapunov function. Under the dynamics (10a), we have
\[
\dot{V}(y) = 2(y - y_{E2})[(\beta_\bar{p}s + \beta_\bar{p}(1 - s - y) - \beta_\bar{p}(1 - y_{E2})]y \\
= 2(y - y_{E2})[(\beta_\bar{p} - \beta_p)s - \beta_\bar{p}(y - y_{E2})]y \\
= 2(y - y_{E2})[\beta_\bar{p} - \beta_p)ys - 2\beta_\bar{p}yV(y) \\
\leq -2(\beta_\bar{p} - \delta)\bar{y}V(y)
\]
when \( |y(t) - y_{E2}| \geq \delta^{-1}(\beta_\bar{p} - \beta_p)|s(t)| \). It follows from [39] that \( y_{E2} \) is ISS for the dynamics of infected proportion when \( y(t) \in (0, y_{\text{int}}) \). As \( s(t) \to 0 \) as \( t \to \infty \), we have \( y(t) \to y_{E2} \).

Case 3B: \( y_{E2} \in (y_{\text{int}}, \bar{y}_{\text{int}}^*) \). First, observe that if \( y(t) \in (0, y_{\text{int}}) \), we have
\[
\begin{align*}
\dot{y} &= [\beta_\bar{p}s + \beta_\bar{p}(1 - s - y) - \gamma]y \\
&= [(\beta_\bar{p} - \beta_p)s + \beta_\bar{p}(1 - y) - \gamma]y \\
&> [(\beta_\bar{p} - \beta_p)s + \beta_\bar{p}(1 - y_{\text{int}}) - \beta_\bar{p}(1 - y_{E2})]y \\
&= [(\beta_\bar{p} - \beta_p)s + \beta_\bar{p}(y_{E2} - y_{\text{int}})]y > 0
\end{align*}
\]
since \( y_{E2} > y_{\text{int}} \) in this subcase. Consequently, \( y(t) \) remains confined to (10c). Let \( V(y) = (y - y_{E2})^2 \) as before. Under the dynamics (10c), we have
\[
\begin{align*}
\dot{V}(y) &= 2(y - y_{E2})[(\alpha\beta_p s + \beta_\bar{p}(1 - s - y) - \beta_\bar{p}(1 - y_{E2})]y \\
&= 2(y - y_{E2})[(\alpha\beta_p - \beta_p)s - \beta_\bar{p}(y - y_{E2})]y \\
&= 2(y - y_{E2})(\alpha\beta_p - \beta_p)ys - 2\beta_\bar{p}yV(y) \\
&\leq -2(\beta_\bar{p} - \delta)\bar{y}V(y)
\end{align*}
\]
when \( |y(t) - y_{E2}| \geq \delta^{-1}|\alpha\beta_p - \beta_\bar{p}|s(t) \) which implies that \( y_{E2} \) is ISS for the dynamics of infected proportion when \( y(t) \in (y_{\text{int}}, \bar{y}_{\text{int}}^*) \). Since \( s(t) \to 0 \) as \( t \to \infty \), we have \( y(t) \to y_{E2} \).
Case 4: \(\alpha \hat{\beta}_p (1 - \hat{y}_{\text{int}}) < \gamma < \hat{\beta}_p [1 - \hat{y}_{\text{int}}]\). It follows from Case 3 above that for E2 to be an equilibrium of the hybrid dynamics, we must have \(\hat{\beta}_p [1 - \hat{y}_{\text{int}}] < \gamma\) which is not satisfied in this regime. Similarly, for E3 to be an equilibrium, we must have \(\alpha \hat{\beta}_p [1 - \hat{y}_{\text{int}}] > \gamma\) which is not satisfied in this regime. IFE being unstable follows from previous arguments as \(R_0 > 1\) and \(R_1 > 1\) for the dynamics (10a).

We now focus on the dynamics at the neighborhood of \((\hat{y}_{\text{int}} - 1 - \hat{y}_{\text{int}})\). For \(y = \hat{y}_{\text{int}} + \epsilon_1, r = 1 - \hat{y}_{\text{int}} - \epsilon_2\) with a sufficiently small \(\epsilon_1, \epsilon_2 > 0\), we have

\[
\dot{r} = -\alpha \hat{\beta}_p (1 - \hat{y}_{\text{int}} - \epsilon_2) + \gamma (\hat{y}_{\text{int}} + \epsilon_1) > 0 \\
\dot{y} = [\alpha \hat{\beta}_p (1 - \hat{y}_{\text{int}} - \epsilon_2) - \gamma (\hat{y}_{\text{int}} + \epsilon_1) < 0
\]

since \(\gamma > \alpha \hat{\beta}_p [1 - \hat{y}_{\text{int}}]\). Similarly, at \(y = \hat{y}_{\text{int}} - \epsilon_1, r = 1 - \hat{y}_{\text{int}} + \epsilon_2\) with a sufficiently small \(\epsilon_1, \epsilon_2 > 0\), we have

\[
\dot{r} = [\hat{\beta}_p (1 - \hat{y}_{\text{int}} + \epsilon_2) + \gamma (\hat{y}_{\text{int}} - \epsilon_1) < 0 \\
\dot{y} = [\hat{\beta}_p (1 - \hat{y}_{\text{int}} + \epsilon_2) - \gamma (\hat{y}_{\text{int}} - \epsilon_1) > 0
\]

since \(\gamma < \hat{\beta}_p [1 - \hat{y}_{\text{int}}]\). Since the differential inclusion in (10d) is a convex combination of the dynamics in (10c) and (10e), \((\hat{y}_{\text{int}} - 1 - \hat{y}_{\text{int}})\) acts as a sliding mode of (10).

Following identical arguments as Case 3B above, it can be shown that when \(y(t) \in (0, \hat{y}_{\text{int}}), \dot{y} > 0\) and when \(y(t) > \hat{y}_{\text{int}}, \dot{y} < 0\) for some \(t > T_0\). Consequently, \(y(t) \rightarrow y_{\text{int}}\) as \(t \rightarrow \infty\).

Case 5: \(\gamma < \alpha \hat{\beta}_p [1 - \hat{y}_{\text{int}}]\). It follows from Case 3 that for E3 to be an equilibrium of the hybrid dynamics, we require \(\gamma < \alpha \hat{\beta}_p [1 - \hat{y}_{\text{int}}]\) and \(\gamma < \alpha \hat{\beta}_p\), both of which are satisfied in this regime. Further, for (10e), \(R_1 = \frac{\hat{\beta}_p}{\alpha} > 1\), and consequently, E3 is locally stable. The IFE being unstable and E2 not being an equilibrium point follows from previous arguments.

Following identical arguments as Case 3B above, it can be shown that when \(y(t) < \hat{y}_{\text{int}}, \dot{y} > 0\) and when \(y(t) > \hat{y}_{\text{int}}, \dot{y}_{\text{endemic infection level}}\) at \(E_3\) is ISS for the infection dynamics with \(s(t)\) being the vanishing input.

\[\square\]

C. Proof of Proposition 5

\textbf{Proof:} The proofs of statements 1–4 follows from analogous arguments as the proof of Proposition 4 and is omitted. We now focus on establishing the claims regarding the stability of IFE. Note that local stability of the IFE depends on the dynamics (11a). For statements 2–4, we have \(\gamma < \hat{\beta}_p\), and as a result, \(R_1 = \frac{\hat{\beta}_p}{\alpha} > 1\) for (11a).

When \(\gamma < \hat{\beta}_p\), we have \(R_0 = \frac{\hat{\beta}_p}{\gamma} > 1\) for the dynamics (11a). Following [36, Th. 1, Case 3], all points in the IFE are unstable. When \(\gamma > \beta_p\), we have \(R_0 = \frac{\hat{\beta}_p}{\gamma} < 1\) for the dynamics (11a). It follows from [36, Th. 1, Case 4] that IFE with:

\[
s^* > \gamma - \hat{\beta}_p \gamma \beta_p - \gamma \beta_p > \beta_p - \gamma \beta_p - \gamma P \frac{\hat{\beta}_p}{\beta_p - \gamma P} - \gamma P
\]

are locally stable and IFE with \(r^* > \beta_p \gamma - \beta_p - \gamma P\) are unstable.

\[\square\]
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