



Brief paper

Epidemic population games and evolutionary dynamics[☆]

Nuno C. Martins*, Jair Certório, Richard J. La

Department of ECE and ISR, University of Maryland, College Park, MD, 20742, USA



ARTICLE INFO

Article history:

Received 25 January 2022

Received in revised form 3 October 2022

Accepted 22 February 2023

Available online xxxx

Keywords:

Epidemic

Population games

Evolutionary dynamics

Lyapunov stability

ABSTRACT

We propose a system theoretic approach to select and stabilize the endemic equilibrium of an SIRS epidemic model in which the decisions of a population of strategically interacting agents determine the transmission rate. Specifically, the population's agents recurrently revise their choices out of a set of strategies that impact to varying levels the transmission rate. A payoff vector quantifying the incentives provided by a planner for each strategy, after deducting the strategies' intrinsic costs, influences the revision process. An evolutionary dynamics model captures the population's preferences in the revision process by specifying as a function of the payoff vector the rates at which the agents' choices flow toward strategies with higher payoffs. Our main result is a dynamic payoff mechanism that is guaranteed to steer the epidemic variables (via incentives to the population) to the endemic equilibrium with the smallest infectious fraction, subject to cost constraints. We use a Lyapunov function not only to establish convergence but also to obtain an (anytime) upper bound for the peak size of the population's infectious portion.

© 2023 Elsevier Ltd. All rights reserved.

1. Introduction

This article has two main tenets: (i) We adopt a continuous-time susceptible-infectious-recovered-susceptible (SIRS) compartmental epidemic model (Pastor-Satorras, Castellano, Van Mieghem, & Vespignani, 2015) in which the aggregate decisions of a population of bounded-rationality agents determine the *transmission rate*, which we denote as $\mathcal{B}(t)$ at time t . We employ a population game approach in which the agents are nondescript and must choose from a set of available strategies $\{1, \dots, n\}$, $n \geq 2$. Each strategy will have an effect on $\mathcal{B}(t)$, but the agents' choices are guided by each strategy's payoff, or net reward, resulting from a payoff incentive, or reward, after the intrinsic cost of the strategy is deducted. The collective decision-making of the population follows an evolutionary dynamics model that captures the agents' preferences and assumes that the agents can repeatedly revise their strategies (see Section 1.1). (ii) We formulate and solve a design problem that seeks to steer via payoff incentives the agents' decisions to attain the smallest endemic prevalence of infections, subject to a limit on the long term incentives' cost. The problem envisages dynamic payoff mechanisms whose dynamics can be coupled with the state (epidemic variables) of the SIRS

model. We will refer to this coupled system as an *epidemic population game* (see Section 1.2).

1.1. Evolutionary dynamics model (EDM)

Each agent follows one strategy at a time, which it can revise repeatedly. A payoff vector $p(t)$ in \mathbb{R}^n whose entries quantify the net rewards of the available strategies influences the revision process, as typically the agents seek strategies with higher payoffs. Namely, we define

$$p(t) := r(t) - c, \quad (1)$$

where c is the vector whose ℓ th entry c_ℓ is the inherent cost of the ℓ th strategy, and $r(t)$ is a reward vector meant to incentivize the adoption of safer (costlier) strategies, where $r_\ell(t)$ is the ℓ th strategy's reward.

Rather than focusing on what each strategy may represent (see Remark 1), in our analysis we assume that a vector $\vec{\beta}$ in $\mathbb{R}_{>0}^n$ is given whose ℓ th entry $\vec{\beta}_\ell$ quantifies the effect of strategy ℓ towards $\mathcal{B}(t)$ according to

$$\mathcal{B}(t) = \vec{\beta}' x(t), \quad t \geq 0, \quad (2)$$

where $x(t)$ is the so-called *population state* taking values in the standard simplex \mathbb{X} defined below and whose ℓ th entry $x_\ell(t)$ is the proportion of the population adopting the ℓ th strategy at time t .

$$\mathbb{X} := \left\{ x \in [0, 1]^n \mid \sum_{i=1}^n x_i = 1 \right\}.$$

[☆] This work was supported by AFOSR, United States Grant FA9550-19-1-0315 and National Science Foundation (NSF) Grant 2135561. The material in this paper was not presented at any conference. This paper was recommended for publication in revised form by Associate Editor Aneel Tanwani under the direction of Editor Luca Zaccarian.

* Corresponding author.

E-mail addresses: nmartins@umd.edu (N.C. Martins), certorio@umd.edu (J. Certório), hyongla@umd.edu (R.J. La).

Following the standard approach in Sandholm (2010a, §4.1.2), the following *evolutionary dynamics model* (EDM) governs the dynamics of x in the large-population limit:

$$\dot{x}(t) = \mathcal{V}(x(t), p(t)), \quad t \geq 0, \quad (\text{EDM}a)$$

where the i th component of \mathcal{V} is specified as:

$$\begin{aligned} \mathcal{V}_i(x(t), p(t)) := & \underbrace{- \sum_{j=1, j \neq i}^n x_i(t) \mathcal{T}_{ij}(x(t), p(t))}_{\text{outflow switching out of strategy } i} \\ & + \underbrace{\sum_{j=1, j \neq i}^n x_j(t) \mathcal{T}_{ji}(x(t), p(t))}_{\text{inflow switching to strategy } i}. \end{aligned} \quad (\text{EDM}b)$$

A Lipschitz continuous map $\mathcal{T} : \mathbb{X} \times \mathbb{R}^n \rightarrow [0, \bar{\mathcal{T}}]^{n \times n}$, with upper bound $\bar{\mathcal{T}} > 0$, is referred to as *the revision protocol* and models the agents' strategy revision preferences. In Sandholm (2010a, Part II) and Sandholm (2015, §13.3–13.5) there is a comprehensive discussion on protocol types and the classes of bounded rationality rules they model.

Below, we define common protocol classes, which we will invoke in examples and to illustrate key concepts.

Definition 1. Any protocol is said to be of the *impartial pairwise comparison* (IPC) type (Sandholm, 2010b) if there is a map $\phi : \mathbb{R}_{\geq 0} \rightarrow [0, \bar{\mathcal{T}}]^n$, whose components satisfy $\phi_j(0) = 0$ and $\phi_j(v) > 0$ for $v > 0$, such that \mathcal{T} can be recast as:

$$\mathcal{T}_{ij}(x, p) = \phi_j([\tilde{p}_{ij}]_+), \quad (\text{3})$$

where $\tilde{p}_{ij} := p_j - p_i$. The well-known Smith's protocol (Smith, 1984) can be specified by $\phi_j^{\text{Smith}}([\tilde{p}_{ij}]_+) := \min\{\lambda[\tilde{p}_{ij}]_+, \bar{\mathcal{T}}\}$, $\lambda > 0$. For Smith's protocol, the switching rate from the i th to the j th strategy is proportional to the positive part of the payoff difference \tilde{p}_{ij} , up to the upper bound $\bar{\mathcal{T}}$.

Definition 2. Any protocol is said to be of the *separable excess payoff target* (SEPT) type (Sandholm, 2005) if there is a map $\phi : \mathbb{R}_{\geq 0} \rightarrow [0, \bar{\mathcal{T}}]^n$, whose components satisfy $\phi_j(0) = 0$ and $\phi_j(v) > 0$ for $v > 0$, such that \mathcal{T} can be recast as:

$$\mathcal{T}_{ij}(x, p) = \phi_j([\hat{p}_j]_+), \quad \hat{p}_j := p_j - \sum_{i=1}^n x_i p_i, \quad (\text{4})$$

where \hat{p} is the so-called excess payoff vector. The classic Brown-von Neumann-Nash (BNN) protocol (Brown & von Neumann, 1950) can be specified by $\phi_j^{\text{BNN}}([\hat{p}_j]_+) := \min\{\lambda[\hat{p}_j]_+, \bar{\mathcal{T}}\}$, $\lambda > 0$.

1.2. Epidemic Population Game (EPG)

Let θ and ζ be the daily birth and natural death rates (from all epidemic-unrelated causes), respectively, and define $g := \theta - \zeta$. Our time unit is *one day*, and g determines the daily population growth according to $N(t) = e^{gt}N(0)$, where $N(0)$ is large and $N(t)$ approximates the population's cardinality at time $t \geq 0$. The approximate daily birth and death rates in the U.S. in 2019 were respectively $\theta = 3.1 \times 10^{-5}$ and $\zeta = 2.4 \times 10^{-5}$.

Below, we specify an *epidemic population game* (EPG):

$$\dot{I}(t) = (\mathcal{B}(t)(1 - I(t) - R(t)) - \sigma)I(t), \quad (\text{EPG}a)$$

$$\dot{R}(t) = \gamma I(t) - \omega R(t), \quad (\text{EPG}b)$$

$$\dot{q}(t) = G(I(t), R(t), x(t), q(t)), \quad (\text{EPG}c)$$

$$\dot{r}(t) = H(I(t), R(t), x(t), q(t)), \quad (\text{EPG}d)$$

where $I(t)$, $R(t)$ and $S(t) := (1 - I(t) - R(t))$ take values in $[0, 1]$ and represent the proportions of the population which are infectious, have recovered and are susceptible to infection at time t , respectively. Specifically, they are the numbers of infectious, recovered and susceptible individuals at time t divided by $N(t)$. Here, (EPG a) and (EPG b) is a normalized SIRS model with $\sigma := \gamma + \theta$ and $\omega := \psi + \theta$, where γ and ψ denote the daily recovery rate and the daily rate at which recovered individuals become susceptible (due to waning immunity), respectively. The estimated mean recovery time and immunity duration for COVID-19 are respectively approximately 10 days and 2–9 months, yielding $\gamma \approx 0.1$ and $\psi \in [0.0037, 0.017]$.

We note that the epidemic model in (EPG a) and (EPG b) is an adaptation of the mean-field approximation in Kermack and McKendrick (1927) and has been used extensively in the literature, e.g., Al-Radhawi, Sadeghi, and Sontag (2021), Amaral, de Oliveira, and Javarone (2021), Bauch (2005), Bauch and Earn (2004), Di Lauro, Kiss, and Miller (2021), d'Onofrio, Manfredi, and Poletti (2011), Godara, Herminghaus, and Heiderman (2021), Kabir and Tanimoto (2020), Nowzari, Preciado, and Pappas (2016), O'Regan, Kelly, Korobeinikov, O'Callaghan, and Pokrovskii (2010), Paré, Beck, and Basar (2020), Paré, Beck, and Nedić (2018), Pastor-Satorras et al. (2015) and Sontag (2021). In our derivation, we assume that newborns are susceptible and the disease death rate associated with the epidemic is zero (death rate is independent from the epidemic). This is a reasonable assumption when the number of deaths caused by the disease is negligible relative to that from all other causes.

Remark 1. Conceivably, the strategies could modulate the infection risk for susceptible individuals. If $x_i(t)S(t)$ is the proportion of the population at time t that is susceptible and adopts the i th strategy, then (2) is consistent with a rate of new infections of $\beta_i x_i(t)S(t)I(t)$ for such a sub-population and $\mathcal{B}(t)S(t)I(t)$ for the entire population.

Finally, (EPG c) and (EPG d) is a *payoff mechanism* we seek to design, where $r(t)$ appears in (1), and $q(t) \in \mathbb{R}^m$, $m \geq 1$.

1.3. Problem formulation and paper structure

The strategies' inherent costs decrease for higher transmission rates, and we order the entries of $\vec{\beta}$ and c as:

$$\vec{\beta}_i < \vec{\beta}_{i+1} \text{ and } c_i > c_{i+1}, \quad 1 \leq i \leq n-1.$$

We consider that $\vec{\beta}_1 > \sigma$, i.e., a transmission rate less than or equal to σ would be unfeasible or too onerous.

Henceforth, c and $\vec{\beta}$ satisfying the conditions above are assumed given and fixed. Hence, we can simplify our notation by omitting c and $\vec{\beta}$ from this point onward. We will use \tilde{c} defined below to specify cost constraints because for a planner seeking to promote the i th strategy it suffices to offer incentives to offset the differential \tilde{c}_i .

$$\tilde{c}_i := c_i - c_n, \quad 1 \leq i \leq n.$$

Definition 3. Given a cost budget c^* in $(0, \tilde{c}_1)$, we determine the optimal endemic transmission rate β^* as:

$$\beta^* := \min\{\vec{\beta}'x \mid \tilde{c}'x \leq c^*, x \in \mathbb{X}\}. \quad (5)$$

Main Problem: We seek to obtain Lipschitz continuous G and H for which the following hold for any $I(0)$ in $(0, 1]$, $R(0)$ in $[0, 1 - I(0)]$, $x(0)$ in \mathbb{X} , and $q(0)$ in \mathbb{R}^m :

$$\lim_{t \rightarrow \infty} (I, R, \mathcal{B})(t) = (I^*, R^*, \beta^*), \quad (\text{P1})$$

$$\limsup_{t \rightarrow \infty} x(t)'r(t) \leq c^*, \quad (\text{P2})$$

where, from Picard's Theorem, $\{(I, R, x, q)(t) \mid t \geq 0\}$ is the unique solution of the initial value problem for the closed loop system formed by (EDM) and (EPG). Here, the nontrivial endemic equilibrium for (EPGa) and (EPGb) is:

$$I^* := \eta(1 - \frac{\sigma}{\beta^*}), \quad R^* := (1 - \eta)(1 - \frac{\sigma}{\beta^*}), \quad \eta := \frac{\omega}{\omega + \gamma}.$$

We will seek G and H for which a *Lyapunov function for the overall system* exists. We will do so not only to establish (P1) but, crucially, also to leverage the Lyapunov function to obtain *anytime upper bounds* for $I(t)$. This is relevant because, as has been pointed out in studies (Godara et al., 2021; Sontag, 2021) employing $B(t)$ as a control variable, $I(t)$ tends to significantly overshoot its endemic equilibrium I^* when $I(0) < I^*$, unless the control policy prevents it.

To make **Remark 1** more concrete, consider a scenario with a set of available strategies, e.g., masking vs. vaccination. While masking may be cheaper than vaccination, it is likely less effective. If we wish to prevent the peak infection from exceeding a target threshold *without knowing the details of interactions*, it may be necessary at times to encourage individuals to get vaccinated by offering incentives (free vaccine) or rewards.

Remark 2. We interpret $r(t)'x(t)$ as the rate at which cost is accrued on average (per-agent) at time t . Hence, (P2) would guarantee that the long-term cost accrual rate a social planner would have to bear for employing G and H would not exceed c^* . Moreover, since I^* is an increasing function of β^* , (P1) would guarantee the smallest endemic infectious portion, subject to (P2).

Paper structure: In Section 2, we motivate our paradigm and compare it with previous work. In Section 3, we describe a choice for G and H , introduce a candidate Lyapunov function, and state **Theorem 1** asserting that our choice solves our Main Problem. We exemplify in Section 4 how to construct anytime bounds, which we also validate numerically via simulation. The article ends with brief conclusions in Section 5, and in the **Appendix** we prove **Theorem 1**.

2. Motivation and comparison to prior work

Modern theoretical epidemiology can be traced back to the early 20th century. For example, Kermack and McKendrick used a deterministic model to study the transmissions in a closed population, which is now known as the susceptible-infected-recovered (SIR) model, and demonstrated the existence of a critical threshold density of susceptible individuals for the occurrence of a major epidemic. Since then, many related compartmental models have been introduced with additional states, e.g., deceased (D), exposed (E), maternally-derived (M), vaccinated (V), and include SEIR/S, SIRD, SIRV, SIS, and MSIR, in addition to the SIRS model adopted for our study. A comprehensive survey on epidemic models and their dynamics can be found in Anderson and May (1991), Mei, Mohagheghi, Zampieri, and Bullo (2017), Nowzari et al. (2016).

A major aspect of epidemic processes is human behavior and the strategic interactions among individuals, which determine their decisions over time in response to their payoffs and in turn shape the course of epidemic processes. Game theory provides a natural framework and tools for studying such strategic interactions, and several recent studies adopted an *evolutionary* or *population game* framework, e.g., (Amaral et al., 2021; Arefin, Masaki, & Tanimoto, 2020; Bauch, 2005; Bauch & Earn, 2004; d'Onofrio et al., 2011; Kabir & Tanimoto, 2020) and Kuga and Tanimoto (2018). We refer an interested reader to Chang, Piraveenana, Pattison, and Prokopenko (2020) and references therein for a comprehensive survey of earlier studies and a more detailed

summary of work reported in Bauch (2005), Bauch and Earn (2004), d'Onofrio et al. (2011). Amaral et al. (2021) studied the effects of perceived risks, i.e., individual cost from infection, when individuals can choose to voluntarily quarantine or continue their normal life. They showed that increased perceived risks result in multiple infection peaks due to strategic interactions. Kabir and Tanimoto (2020) considered a similar setting and showed that naturally acquired shield immunity is unlikely to be effective in suppressing an epidemic without additional social measures with low costs for individuals.

In another line of related research, Di Lauro et al. (2021) and Sontag (2021) studied the problem of identifying when non-pharmaceutical interventions (NPIs), such as quarantine and lockdowns, should be put in place to minimize the peak infections; Di Lauro et al. (2021) studied the optimal timing for one-shot intervention, whereas Sontag (2021) considered a fixed number of complete lockdowns. Al-Radhawi et al. (2021) modeled media coverage, public health measures and other NPIs during a prolonged epidemic as feedback effects and examined the problem of tuning NPIs to regulate infection rates as an adaptive control problem. Using a singular-perturbation approach, the authors investigated the stability of disease-free and endemic steady states. Godara et al. (2021) considered the problem of controlling the infection rate to minimize the total cost till herd immunity is achieved in an SIR model. They formulated it as an optimal control problem subject to a constraint on the fraction of infectious population.

Although we do not consider epidemic processes on general networks here, their dynamics on networks have been studied extensively (see Mei et al., 2017; Nowzari et al., 2016; Paré et al., 2020; Pastor-Satorras et al., 2015 for a review of the literature), including time-varying networks (Paré et al., 2018) and the influence of network properties on epidemic processes, e.g., (La, 2019). Recently, the topic of mitigating disease or infection spread in a network has enjoyed much attention. In particular, researchers investigated optimal strategies using vaccines/immunization (prevention) (Preciado, Zargham, Enyioha, Jadbabaie, & Pappas, 2013), antidotes or curing rates (recovery) (Ottaviano, De Pellegrini, Bonaccorsi, & Van Mieghem, 2018) or a combination of both preventive and recovery measures (Nowzari, Preciado, & Pappas, 2017; Preciado, Zargham, Enyioha, Jadbabaie, & Pappas, 2014). For example, Preciado et al. (2013) studied the problem of partial vaccination via investments by each individual to reduce the infection rates, aiming to maximize the exponential decay rate to control the spread of an epidemic.

Our study advances the state-of-the-art in several directions: unlike the studies that aim to suppress epidemic spread (Nowzari et al., 2017; Ottaviano et al., 2018; Preciado et al., 2013, 2014), our goal is to design policies for minimizing the endemic transmission rate subject to a constraint on the long-term average cost a planner bears. Moreover, even though Di Lauro et al. (2021), Godara et al. (2021), Sontag (2021) investigated a related problem of managing infection rates during epidemics, these studies did not consider strategic interactions among many agents of bounded rationality, which can revise their strategies over time, leading to more complex dynamics. Finally, to the best of our knowledge, our study is the first to provide a methodology for designing policies that can guarantee (a) provable convergence to an equilibrium set (see **Theorem 1** and **Remark 5**) and (b) fulfill an *anytime* bound on $I(t)$ (see Eq. (24) and **Remark 6**). As we will discuss in detail, it is notable that these results hold under *any* revision protocol \mathcal{T} that satisfies some assumptions stated in the subsequent section *without the need to know the exact protocol*.

3. A solution to main problem

In this section, we will propose a choice of G and H for (EPG) and in Section 3.5 we will state **Theorem 1**, which addresses the Main Problem, as stated in Section 1.3.

3.1. Cases I and II, and determining x^*

Before we proceed, we will introduce a definition, an assumption and a related remark.

Definition 4. [Cases I and II] Given c^* in $(0, \tilde{c}_1)$, one of two cases holds: **Case I** is defined by when $\tilde{c}_{i^*+1} < c^* < \tilde{c}_{i^*}$ for some positive $i^* \leq n - 1$. **Case II** occurs when $n \geq 3$ and $c^* = \tilde{c}_{i^*}$ for some $i^* \in \{2, \dots, n - 1\}$.

Notice that Case II is unlikely to appear in practice and is considered in our analysis for the sake of completeness.

Assumption 1. The following must hold when $n \geq 3$:

$$\frac{c_i - c_{i+1}}{\tilde{\beta}_{i+1} - \tilde{\beta}_i} > \frac{c_{i+1} - c_{i+2}}{\tilde{\beta}_{i+2} - \tilde{\beta}_{i+1}}, \quad 1 \leq i \leq n - 2. \quad (6)$$

According to (6), we assume that as the transmission rate decreases it becomes costlier to reduce it further.

Remark 3. Subject to Assumption 1, it follows from Karush–Kuhn–Tucker conditions that, for any given c^* in $(0, \tilde{c}_1)$, (5) has a unique solution we denote as:

$$x^* := \arg \min \{ \tilde{\beta}' x \mid \tilde{c}' x \leq c^*, x \in \mathbb{X} \}.$$

For Case I, with $\tilde{c}_{i^*+1} < c^* < \tilde{c}_{i^*}$, it results that $x_{i^*}^* = (c^* - \tilde{c}_{i^*+1})/(\tilde{c}_{i^*} - \tilde{c}_{i^*+1})$, $x_{i^*+1}^* = 1 - x_{i^*}^*$ and the other entries of x^* are zero, while for Case II, with $c^* = \tilde{c}_{i^*}$, we get that $x_{i^*}^* = 1$ and the other entries of x^* are zero. We also immediately conclude from $\beta^* = \tilde{\beta}' x^*$ that $\tilde{\beta}_{i^*} < \beta^* < \tilde{\beta}_{i^*+1}$ for Case I and $\beta^* = \tilde{\beta}_{i^*}$ for Case II.

3.2. Lyapunov-inspired choice for (EPGc) and (EPGd)

We start by defining (\hat{I}, \hat{R}) below, which can be interpreted as “reference” epidemic variables determined by the population state x via $\mathcal{B} = \beta' x$:

$$\hat{I} := \eta \left(1 - \frac{\sigma}{\mathcal{B}} \right), \quad \hat{R} := (1 - \eta) \left(1 - \frac{\sigma}{\mathcal{B}} \right). \quad (7)$$

A candidate Lyapunov function (to be described later in Section 3.4) motivated the following choice for (EPGc) and (EPGd):

$$G(I, R, x, q) := (\hat{I} - I) + \eta(\ln I - \ln \hat{I}) + \nu^2(\beta^* - \mathcal{B}) + \frac{\mathcal{B}}{\nu}(R - \hat{R})(1 - \eta - R), \quad (8)$$

$$H(I, R, x, q) := q\tilde{\beta} + r^*, \quad (9)$$

where $\nu > 0$ and $\rho^* > 0$ (see Section 3.2.1) are design parameters, and r^* is the following stationary reward vector:

$$r_i^* := \begin{cases} \tilde{c}_i - \rho^* & \text{if } x_i^* = 0 \\ \tilde{c}_i & \text{otherwise,} \end{cases} \quad 1 \leq i \leq n.$$

In Section 3.2.1, we describe the rules for selecting a valid ρ^* .

Definition 5 (Design Parameters). We refer to $\nu > 0$, $\rho^* > 0$ and c^* in $(0, \tilde{c}_1)$ as design parameters. Here, we recall that c^* determines β^* and x^* .

In the Appendix, our proof for the upcoming Theorem 1 will use the fact that, for the r^* chosen, x^* will be the only element x of \mathbb{X} that simultaneously satisfies $\tilde{\beta}' x = \beta^*$, and also maximizes $x'(r^* - c)$, which is equivalent to it being the best response to the equilibrium payoff $r^* - c$.

Notice that, when the epidemic is beginning or is effectively contained, (8) can be approximated simply as:

$$G(I, R, x, q) \underset{R, I \ll 1}{\approx} \eta(\ln I - \ln \hat{I}) + \nu^2(\beta^* - \mathcal{B}). \quad (10)$$

According to (EPGc), G will govern the dynamics of $q(t)$, which will indirectly regulate $\mathcal{B}(t)$ via the payoff $p(t)$ in (1) and H in (EPGd). Specifically, if the population’s agents adhere to a protocol, such as IPC or SEPT, that prioritizes strategies with higher payoffs, then lowering $q(t)$ would have the effect of decreasing more the payoffs of riskier strategies and hence steering the population towards safer strategies that lower $\mathcal{B}(t)$ and $\hat{I}(t)$. On the contrary, increasing $q(t)$ would incentivize higher $\mathcal{B}(t)$.

3.2.1. Rules for selecting a valid ρ^*

When $n = 2$, since c^* is in $(\tilde{c}_2, \tilde{c}_1)$, we have Case I and from Remark 3 we can further conclude that ρ^* is not present in r^* . To select a valid $\rho^* > 0$ for $n \geq 3$, proceed as follows: (i) For Case I, choose any $\rho^* > 0$. (ii) For Case II, select any $\rho^* \geq \max\{\beta_n - \beta^*, \beta^* - \beta_1\}$.

3.3. Nash stationarity and δ -passivity assumption

Definition 6 (Nash Stationarity). A protocol \mathcal{T} is “Nash stationary” if the following holds for all p in \mathbb{R}^n :

$$\mathcal{V}(x, p) = 0 \Leftrightarrow x \in \mathcal{M}(p), \quad (\text{NS})$$

where $\mathcal{M} : \mathbb{R}^n \rightarrow 2^{\mathbb{X}}$ is the following best response map¹:

$$\mathcal{M}(p) := \arg \max_{x \in \mathbb{X}} p' x, \quad p \in \mathbb{R}^n.$$

Therefore, for a protocol satisfying (NS), x is an equilibrium of (EDM) if and only if x is a best response to p . Any IPC or SEPT protocol satisfies (NS) (see Sandholm (2015, §13.5.3)).

Our analysis of the long-term evolution of $(\mathcal{I}, \mathcal{R})(t)$ and $(x, p)(t)$ will leverage the following assumption stemming from the δ -passivity concept originally proposed in Fox and Shamma (2013) and later generalized in Arcak and Martins (2021), Kara and Martins (2021), Park, Martins, and Shamma (2019).

Assumption 2. There exist a differentiable function $\mathcal{S} : \mathbb{X} \times \mathbb{R}^n \rightarrow \mathbb{R}_{\geq 0}$ and a Lipschitz continuous function $\mathcal{P} : \mathbb{X} \times \mathbb{R}^n \rightarrow \mathbb{R}_{\geq 0}$ that satisfy the following inequality for all x, p and u in \mathbb{X}, \mathbb{R}^n and \mathbb{R}^n , respectively:

$$\frac{\partial \mathcal{S}(x, p)}{\partial x} \mathcal{V}(x, p) + \frac{\partial \mathcal{S}(x, p)}{\partial p} u \leq -\mathcal{P}(x, p) + u' \mathcal{V}(x, p), \quad (11)$$

where \mathcal{S} and \mathcal{P} must also satisfy the equivalences below:

$$\mathcal{V}(x, p) = 0 \Leftrightarrow \mathcal{S}(x, p) = 0 \Leftrightarrow \mathcal{P}(x, p) = 0. \quad (12)$$

In addition, the following inequality (not required in standard δ -passivity) must hold:

$$\mathcal{P}(x, \alpha p) \geq \mathcal{P}(x, p), \quad \alpha \geq 1, \quad x \in \mathbb{X}, \quad p \in \mathbb{R}^n. \quad (13)$$

In (11), $\frac{\partial \mathcal{S}(x, p)}{\partial x}$ and $\frac{\partial \mathcal{S}(x, p)}{\partial p}$ are the row vectors of partial derivatives of $\mathcal{S}(x, p)$ with respect to the components of x and p , respectively. In the Appendix, (13) will be useful to cope with the lack of an a-priori bound for $|q(t)|$.

Based on the Lyapunov functions in Hofbauer and Sandholm (2009), the authors of Fox and Shamma (2013) determined, for main classes of protocols, explicit expressions for \mathcal{S} and \mathcal{P} , of which the following are important examples. Explicit constructions for \mathcal{S} and \mathcal{P} for a generalization of IPC protocols can be found in Kara and Martins (2021).

Example 1. For any IPC protocol (3) with non-decreasing $\{\phi_1, \dots, \phi_n\}$, the following satisfy (11)–(13):

$$\mathcal{S}^{\text{IPC}}(x, p) := \sum_{i=1, j=1}^{n, n} x_i \int_0^{[\tilde{p}_{ij}]_+} \phi_j(\nu) d\nu, \quad (14)$$

¹ A best response in our context may be interpreted in the mass-action sense (Nash, 1951) also discussed in Weibull (1995).

$$\mathcal{P}^{\text{IPC}}(x, p) := - \sum_{i=1, j=1}^{n, n} \mathcal{V}_i^{\text{IPC}}(x, p) \int_0^{[\hat{p}_{ij}]_+} \phi_j(v) dv,$$

where \mathcal{V}^{IPC} is obtained by substituting (3) into (EDMb). The analysis in Hofbauer and Sandholm (2009, below the expression for $\dot{\Psi}(x)$ on p.1691) can be used here to show that \mathcal{P}^{IPC} is non-negative and satisfies (11) and (12). A small modification of the same argument shows that (13) holds.

Example 2. The following satisfy (11)–(13) for a SEPT protocol (4) for which $\{\phi_1, \dots, \phi_n\}$ are non-decreasing:

$$\begin{aligned} \mathcal{S}^{\text{SEPT}}(x, p) &:= \sum_{j=1}^n \int_0^{[\hat{p}_j]_+} \phi_j(v) dv, \\ \mathcal{P}^{\text{SEPT}}(x, p) &:= \sum_{i, j=1}^{n, n} \phi_i([\hat{p}_i]_+) p_j \mathcal{V}_j^{\text{SEPT}}(x, p), \end{aligned} \quad (15)$$

where $\mathcal{V}^{\text{SEPT}}$ is obtained by substituting (4) into (EDMb). Here, one can use the analysis in Fox and Shamma (2013, (62)) to show that (11) holds. Nash stationarity and positive correlation as defined and established in Sandholm (2005) lead to (12). A straightforward argument using the equalities in Sandholm (2005, proof of Lemma 3.2(ii)) leads to (13).

3.4. A candidate Lyapunov function and its properties

We start with the following reparameterization:

$$\mathcal{I}(t) := \mathcal{B}(t)I(t), \quad \mathcal{R}(t) := \mathcal{B}(t)R(t), \quad (16a)$$

$$\hat{\mathcal{I}}(t) := \mathcal{B}(t)\hat{I}(t), \quad \hat{\mathcal{R}}(t) := \mathcal{B}(t)\hat{R}(t), \quad (16b)$$

$$\tilde{\mathcal{I}}(t) := \hat{\mathcal{I}}(t) - \mathcal{I}(t), \quad \tilde{\mathcal{R}}(t) := \hat{\mathcal{R}}(t) - \mathcal{R}(t), \quad (16c)$$

where $\hat{I}(t)$ and $\hat{R}(t)$ are defined in (7). Using (8), (9) and (16), we rewrite (EPG) as follows:

$$\dot{\mathcal{I}}(t) = \mathcal{I}(t)(\tilde{\mathcal{I}}(t) + \tilde{\mathcal{R}}(t)) + I(t)\dot{\mathcal{B}}(t), \quad (17a)$$

$$\dot{\mathcal{R}}(t) = \omega\tilde{\mathcal{R}}(t) - \gamma\tilde{\mathcal{I}}(t) + R(t)\dot{\mathcal{B}}(t), \quad (17b)$$

$$\dot{q}(t) = G(I(t), R(t), x(t), q(t)), \quad (17c)$$

$$r(t) = H(I(t), R(t), x(t), q(t)) = q(t)\vec{\beta} + r^*, \quad (17d)$$

$$p(t) = q(t)\vec{\beta} + r^o, \quad r^o := r^* - c. \quad (17e)$$

Here, we modified the representation of the SIRS model in O'Regan et al. (2010, between (3) and (4)) to obtain (17a)–(17b), and we substituted (17d) into (1) to obtain (17e).

We proceed to define the candidate Lyapunov function:

$$\mathcal{L}(\mathcal{Y}) := \mathcal{S}(x, p) + \mathcal{I}(\mathcal{I}, \mathcal{R}, \mathcal{B}), \quad \mathcal{Y} \in \mathbb{Y}, \quad (18)$$

$$\mathcal{Y} := (\mathcal{I}, \mathcal{R}, x, q), \quad \mathcal{B} := \vec{\beta}'x,$$

where \mathcal{I} is defined below, \mathcal{S} satisfies (11)–(13), and \mathcal{Y} taking values in \mathbb{Y} defined below is the state of the complete system comprising (EDM) and (17) :

$$\begin{aligned} \mathbb{Y} &:= \{(\mathcal{I}, \mathcal{R}, x, q) \mid x \in \mathbb{X}, q \in \mathbb{R}, \vec{\beta}_1 \leq \mathcal{B} \leq \vec{\beta}_n, \\ &0 < \mathcal{I} \leq \mathcal{B}, 0 \leq \mathcal{R} \leq \mathcal{B} - \mathcal{I}\}. \end{aligned}$$

Here, \mathcal{I} defined below is a modification of the Lyapunov function in O'Regan et al. (2010), with $\tilde{\mathcal{I}} := \hat{\mathcal{I}} - \mathcal{I}$, and $\tilde{\mathcal{R}} := \hat{\mathcal{R}} - \mathcal{R}$:

$$\mathcal{I}(\mathcal{I}, \mathcal{R}, \mathcal{B}) := \hat{\mathcal{I}} \ln \frac{\hat{\mathcal{I}}}{\mathcal{I}} - \tilde{\mathcal{I}} + \frac{1}{2\gamma} \tilde{\mathcal{R}}^2 + \frac{\omega^2}{2} (\mathcal{B} - \beta^*)^2.$$

Notation convention: We note that \mathcal{I} depends on the given design parameters c^* , ω and ρ^* . But, to simplify our notation we decided not to indicate this dependence.

Remark 4. Notice that \mathcal{I} is convex and nonnegative, and $\mathcal{I}(\mathcal{I}, \mathcal{R}, \mathcal{B}) = 0$ if and only if $(\mathcal{I}, \mathcal{R}, \mathcal{B}) = (\mathcal{I}^*, \mathcal{R}^*, \beta^*)$, where $\mathcal{I}^* := \beta^* I^*$ and $\mathcal{R}^* := \beta^* R^*$. Furthermore, (11)–(13) and (NS) imply that $\mathcal{L}(\mathcal{Y}) = 0$ if and only if $(\mathcal{I}, \mathcal{R}, \mathcal{B}) = (\mathcal{I}^*, \mathcal{R}^*, \beta^*)$, and $x \in \mathcal{M}(q\vec{\beta} + r^o)$.

After taking derivatives, and using (11)–(13) and (17), we get:

$$\frac{d}{dt} \mathcal{L}(\mathcal{Y}(t)) \leq -\mathcal{P}(x(t), p(t)) - \tilde{\mathcal{I}}(t)^2 - \frac{\omega}{\gamma} \tilde{\mathcal{R}}(t)^2. \quad (19)$$

3.5. A stability concept and main result

We will leverage \mathcal{L} and (19) to establish global asymptotic stability of an equilibrium set in the following sense.

Definition 7. A set $\mathbb{E} \subset \mathbb{Y}$ is said to be globally asymptotically stable (GAS) if it satisfies the conditions (GASa)–(GASc) for \mathcal{L} defined in (18), with \mathcal{Y} being the state of the feedback system formed by (EDM) and (17):

(GASa) It holds that $\mathcal{Y} \in \mathbb{E} \Leftrightarrow \mathcal{L}(\mathcal{Y}) = 0$.

(GASb) Given any $\mathcal{Y}(0)$ in \mathbb{Y} , $\{\mathcal{Y}(t) \mid t \geq 0\}$ has at least one accumulation point in \mathbb{E} .

(GASc) Given any $\mathcal{Y}(0)$ in \mathbb{Y} , all accumulation points of $\{\mathcal{Y}(t) \mid t \geq 0\}$ are in \mathbb{E} .

The following is our main convergence result, which we prove in the [Appendix](#).

Theorem 1. Let the protocol defining (EDM) and the design parameters $\omega > 0$, $\rho^* > 0$ (valid according to Section 3.2.1) and c^* in $(0, \tilde{c}_1)$ be given. If (NS) and [Assumptions 1–2](#) hold, then the set \mathbb{E}^* defined below is GAS:

$$\mathbb{E}^* := (\mathcal{I}^*, \mathcal{R}^*, x^*) \times \mathfrak{Q}^*, \quad \mathfrak{Q}^* := \begin{cases} \{0\} & (\text{Case I}) \\ [-\zeta_2^*, \zeta_1^*] & (\text{Case II}), \end{cases}$$

where $\zeta_1^* := \rho^*(\vec{\beta}_n - \beta^*)^{-1}$, and $\zeta_2^* := \rho^*(\beta^* - \vec{\beta}_1)^{-1}$.

Under the conditions of [Theorem 1](#), for any $\mathcal{Y}(0)$ in \mathbb{Y} , the theorem guarantees that $\mathcal{Y}(t)$ tends to \mathbb{E}^* . Consequently, since $\vec{\beta}_n \geq \mathcal{B}(t) \geq \vec{\beta}_1 > 0$, and from (16), the following holds subject to [Theorem 1](#)'s conditions:

$$(I, R, x, q)(t) \xrightarrow[t \rightarrow \infty]{} (I^*, R^*, x^*) \times \mathfrak{Q}^*, \quad (20)$$

implying (P1) as defined in our Main Problem. This will also imply (P2) for Case I. For the rather unlikely Case II, we infer that $\lim_{t \rightarrow \infty} r(t)x(t) \leq c^* + \zeta_1^* \beta^*$.

From [Theorem 1](#) and (GASa), we conclude for Case I that $\mathcal{L}(\mathcal{Y}) = 0$ holds if and only if $(\mathcal{I}, \mathcal{R}, x, q) = (\mathcal{I}^*, \mathcal{R}^*, x^*, 0)$. Hence, based on this fact, [Remark 4](#) and (19) we can view \mathcal{L} for Case I as a Lyapunov function associated with the equilibrium $(\mathcal{I}^*, \mathcal{R}^*, x^*, 0)$.

Remark 5 (Universality of Theorem 1). The protocol \mathcal{T} is absent from (17c)–(17e), and [Theorem 1](#) guarantees that \mathbb{E}^* is GAS for any protocol satisfying (NS) and [Assumption 2](#). These facts are relevant when the exact protocol is uncertain, but enough of its structure is known to establish that it satisfies (NS) and [Assumption 2](#). The IPC protocol class is a case in point as (NS) and [Assumption 2](#) hold for any $\tilde{\mathcal{T}} > 0$ and any ϕ satisfying the monotonicity condition in [Example 1](#), which can be interpreted as presuming (quite plausibly) that the population's agents switch from strategy i to j with a rate that does not decrease when \tilde{p}_{ij} increases. A similar observation could be made about protocols in the SEPT class satisfying the conditions of [Example 2](#).

4. Using \mathcal{L} to obtain anytime bounds

We start by using (19) to state (a) below, and (b) follows from (18), for $t \geq 0$:

$$\alpha := \mathcal{L}(\mathcal{Y}(0)) \stackrel{(a)}{\geq} \mathcal{L}(\mathcal{Y}(t)) \stackrel{(b)}{\geq} \mathcal{S}(\mathcal{I}(t), \mathcal{R}(t), \mathcal{B}(t)). \quad (21)$$

Although in Section 3.4 we adopted the convention of not indicating in our notation that \mathcal{S} depends on the design parameters, here it will be useful to remember that it does and this includes dependence on v . In fact, we will soon outline a method for selecting $v > 0$ based on (21).

Assuming that ρ^* and c^* are pre-selected, while v can vary to meet an overshoot specification, we now proceed to construct an upper-bound for $I(t)/I^*$. Obtaining an upper bound for $I(t)/I^*$ is important because, although Theorem 1 guarantees that $\mathcal{Y}(t)$ will converge to \mathbb{E}^* , the theorem offers no guarantees on the transient behavior of $I(t)$. Using $\pi_v^*(\alpha)$ defined below, we can leverage (21) to obtain the anytime bound $I(t) \leq I^* \pi_v^*(\alpha)$, $t \geq 0$.

Definition 8. Given the parameters specifying (EDM) and (17), we seek to perform the following optimization:

$$\pi_v^*(\alpha) := \frac{1}{I^*} \sup \{ \mathcal{B}^{-1} \mathcal{I} \mid \mathcal{S}(\mathcal{Y}) \leq \alpha, \mathcal{Y} \in \mathbb{Y} \}, \quad (22)$$

where we reverse (16a) to write $I = \mathcal{I}/\mathcal{B}$ and $I^* = \mathcal{I}^*/\beta^*$. Using the fact that \mathcal{S} is convex (see Remark 4), we conclude that (22) is a quasi-convex program that can be swiftly solved using available software.

From Remark 4, we can immediately conclude that for any given $v > 0$, it holds that $\pi_v^*(0) = 1$ and $\pi_v^*(\alpha)$ is an increasing continuous function of $\alpha \geq 0$.

4.1. Bounds when $\mathcal{Y}(0)$ is endemic equilibrium ($n=2$)

Throughout this subsection, consider that $n = 2$ and $\mathcal{Y}(0)$ is an endemic equilibrium point for which $(\mathcal{I}, \mathcal{R})(0) = (\hat{\mathcal{I}}, \hat{\mathcal{R}})(0)$, $\mathcal{B}(0) = \beta' x(0) =: \beta^0$, and $q(0) = 0$. Namely, the system starts at an equilibrium that could have resulted from the prior use of (17) in which β^0 (instead of β^*) was the endemic transmission rate. We proceed by observing that, since the entries of $p(0) = r^0$ are identical (both are equal to $-c_n$), any $x(0) \in \mathbb{X}$ is in $\mathcal{M}(p(0))$, which implies $\mathcal{S}(x(0), p(0)) = 0$. Hence, in this case, by direct substitution into (18), we obtain $\alpha = \mathcal{L}(\mathcal{Y}(0)) = \frac{1}{2} v^2 (\beta^0 - \beta^*)^2$, which using (21) leads, for $t \geq 0$, to the following inequalities:

$$(\mathcal{B}(t) - \beta^*)^2 \stackrel{(a)}{\leq} \frac{2}{v^2} \mathcal{S}(\mathcal{Y}(t)) \stackrel{(b)}{\leq} \tilde{\beta}^2, \quad \tilde{\beta} := \beta^0 - \beta^*. \quad (23)$$

Based on (a) and (b) in (23), it readily follows that $|\mathcal{B}(t) - \beta^*| \leq |\tilde{\beta}|$ and if $\beta^* < \beta^0$ then $\mathcal{B}(t) \leq \beta^0$.

From (b) in (23), we also obtain (see Proposition 1):

$$I(t) \leq I^* \times \pi_v^*(\frac{1}{2} v^2 \tilde{\beta}^2), \quad t \geq 0. \quad (24)$$

Remark 6. [Universality of (24)] Analogously to Remark 5, it is pertinent to observe that since the computation of $\pi_v^*(\alpha)$ for a given α does not require knowledge of the protocol \mathcal{T} , (24) remains valid for any (EDM) satisfying the conditions of Theorem 1.

The following proposition indicates that v plays a key role in bounding the overshoot of $I(t)/I^*$.

Proposition 1. (i) For any $\check{v} \geq v > 0$, it holds that $\pi_{\check{v}}^*(\frac{1}{2} \check{v}^2 \tilde{\beta}^2) \geq \pi_v^*(\frac{1}{2} v^2 \tilde{\beta}^2)$. (ii) Furthermore, it holds that $\pi_v^*(\frac{v^2}{2} \tilde{\beta}^2) \geq \frac{n}{I^*} (1 - \sigma \tilde{\beta}^{-1}) > 1$, $\tilde{\beta} := \min\{|\tilde{\beta}| + \beta^*, \tilde{\beta}_2\}$.

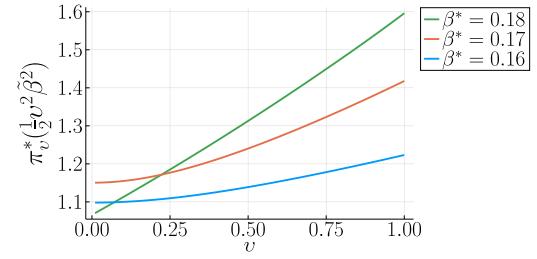


Fig. 1. Plot of $\pi_v^*(\frac{1}{2} v^2 \tilde{\beta}^2)$ in Example 3 as a function of v for varied β^* (other parameters of Example 3 are unchanged).

Proof. Express the constraint defining $\pi_v^*(\frac{1}{2} v^2 \tilde{\beta}^2)$ as $\mathcal{S}(\mathcal{Y}) - \frac{1}{2} v^2 (\mathcal{B} - \beta^*)^2 \leq \frac{1}{2} v^2 (\tilde{\beta}^2 - (\mathcal{B} - \beta^*)^2)$, where for any given \mathcal{B} the left-hand side is a (convex) function of $(\mathcal{I}, \mathcal{R})$. Using the same steps leading to (a)–(b) in (23) we infer that $\tilde{\beta}^2 - (\mathcal{B} - \beta^*)^2 \geq 0$. Hence, we can establish (i) by observing that increasing v does not tighten the constraint. To show (ii) it suffices to select $\mathcal{I} = \hat{\mathcal{I}}$, $\mathcal{R} = \hat{\mathcal{R}}$ and $\mathcal{B} = \tilde{\beta}$ as a feasible solution. \square

We will use the following example to illustrate the validity of our bounds. Our time unit will be one day.

Example 3. Consider that $g = 0$, $\sigma = 0.1$ (infectiousness period ~ 10 days), $\gamma = \sigma$, and $\omega = 0.005$ (immunity period ~ 200 days). The problem parameters are $\tilde{\beta}_1 = 0.15$, $\tilde{\beta}_2 = 0.19$, while the cost vector is $c_1 = 0.2$, $c_2 = 0$. We select $c^* = 0.1$, which gives $\beta^* = 0.17$, $x_1^* = x_2^* = 0.5$, and $(I^*, R^*) \approx (1.96\%, 39.22\%)$. We assume that $x_{1(0)} = 1$, $(I(0), R(0)) = (\hat{I}(0), \hat{R}(0)) = (1.60\%, 31.75\%)$, and $\beta^0 = \mathcal{B}(0) = 0.15$. Our goal is to design G and H so that $I(t) \leq 1.344 \times I^*$. Since $n = 2$, ρ^* is irrelevant (see Section 3.2.1) and we can use (24) to select v .

Example 3 would describe the case in which expensive measures were previously ($t < 0$) in place, but a planner seeks from $t = 0$ onward to relax those measures to reduce the normalized cost rate from $r'(0)x(0) = 0.2$ to a long-term limit of $c^* = 0.1$. From our numerical results (see Fig. 1 for $\beta^* = 0.17$), we determine that $\pi_{0.806}^*(\frac{1}{2}(0.806 \times 0.02)^2) \approx 1.3436$ and conclude from Proposition 1 that any positive $v \leq 0.806$ will guarantee for any protocol satisfying the conditions of Theorem 1 that $I(t) \leq 1.344 \times I^*$ holds, as required in Example 3. All figures were generated using Certório (2022).² Fig. 2(a) illustrates for $v \in \{0.806, 0.316\}$ that the required bound indeed holds for a Smith's protocol. Fig. 2(a) suggests that (24) may be conservative. However, since (24) must be valid for any protocol (not just Smith's) satisfying the conditions of Theorem 1, we do not know how conservative it may be. It is worth noting that, as illustrated in Fig. 2(a), one could have significantly exceeded a 34.4% overshoot by selecting $v \geq 2$.

From the previous discussion, one could be tempted to select a very small $v < 0.806$ expecting to perhaps eliminate any overshoot. However, as Fig. 2(b) illustrates, smaller v may lead to slower convergence, which would keep $x(t)'r(t)$ higher for longer. Thus, selecting the largest v for which the required overshoot constraint is guaranteed by (24) could be a sensible approach. In the case of Example 3 this approach would yield $v = 0.806$.

4.2. Bounds when $\mathcal{Y}(0)$ is endemic equilibrium ($n \geq 3$)

Consider that $n \geq 3$ and that $\mathcal{Y}(0)$ satisfies the conditions specified in Section 4.1. Following the same argumentation as

² The code that generates all figures, and other supplementary scripts, can also be found at github.com/jcert/EPG.

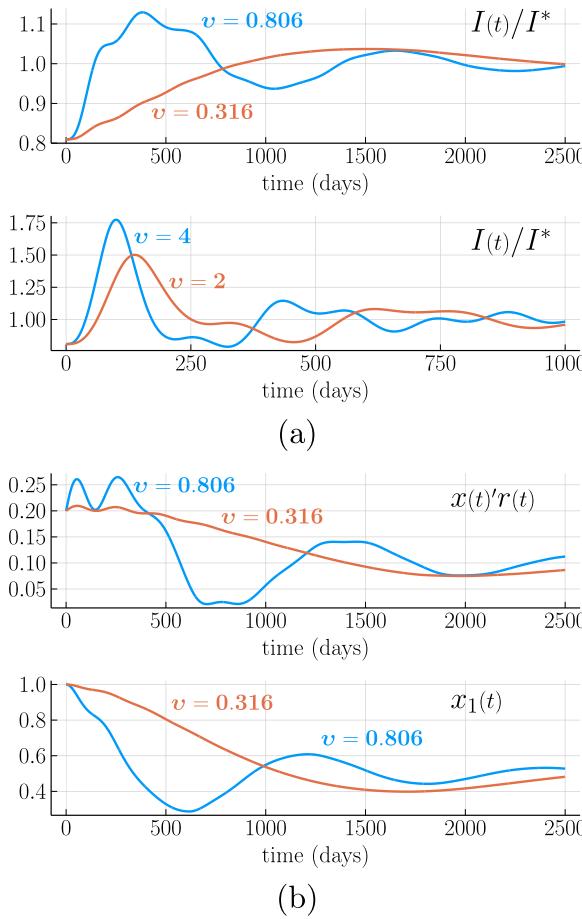


Fig. 2. Simulation for Example 3 using v as shown, and a Smith's protocol specified by $\lambda = 0.1$ and $\bar{\tau} = 0.1$.

in Section 4.1, we conclude that $\mathcal{S}(x(0), p(0)) = 0$ when the support of $x(0)$ is included in that of x^* , in which case (23) remains valid. If this condition on the support of $x(0)$ does not hold, then (b) in (23) is no longer valid and we should instead use:

$$\frac{v^2(\mathcal{B}(t) - \beta^*)^2}{2} \stackrel{(a)}{\leq} \mathcal{S}(\mathcal{Y}(t)) \stackrel{(b)}{\leq} \frac{v^2\tilde{\beta}^2}{2} + \mathcal{S}(x(0), p(0)),$$

where $\mathcal{S}(x(0), p(0))$ can be computed by direct substitution when an explicit formula for \mathcal{S} is known. We can use (14) to compute $\mathcal{S}(x(0), p(0))$ for any IPC protocol, such as for Smith's protocol as shown below:

$$\begin{aligned} \mathcal{S}^{\text{Smith}}(x(0), p(0)) &= \sum_{i=1, j=1}^{n, n} x_i(0) \Phi(r_j^0 - r_i^0), \\ \Phi(v) &:= \begin{cases} \frac{1}{2}[v]_+^2 & \text{if } v \leq \bar{\tau} \\ [v]_+ \bar{\tau} & \text{if } v > \bar{\tau}, \end{cases} \quad v \in \mathbb{R}. \end{aligned}$$

5. Conclusions and future directions

We put forth a system theoretic methodology to model and regulate the endemic prevalence of infections for the case where the decisions of a population of strategically interacting agents determine the epidemic transmission rate. Our main result is a dynamic payoff mechanism that is guaranteed to steer the epidemic variables (via incentives to the population) to an endemic equilibrium characterized by the lowest prevalence of infections, subject to cost constraints. Using a Lyapunov function we used to

prove convergence, we established an upper bound for the size of the population's infectious fraction.

Subject to the conditions of Theorem 1, a planner using the incentive policy specified by the proposed G and H , which in some cases admits the approximation for G in (10), will steer the population to the lowest endemic infectious fraction for a given budget constraint (see Remark 2). In addition, as discussed in Section 4, the planner can tune v to obtain a desired upper bound of the peak size of the population's infectious portion (see Remark 6).

Although Theorem 1 is rather general (Remark 5), there are protocols for which it is not valid. These include imitation (Sandholm, 2010a, §5.4) and perturbed best response (PBR) (Sandholm, 2010a, §6.2) protocols, which are not Nash stationary. Moreover, the so-called replicator protocol (a particular case of imitation protocol) is not δ -passive (Park, Shamma, & Martins, 2018, Proposition III.5) and, while PBR is δ -passive (Park et al., 2018, Proposition III.8), it is not clear whether it would satisfy (13). Hence, investigating modifications of G , H and Theorem 1 for these protocols is an important future research direction.

Appendix. Proof of Theorem 1

In order to prove Theorem 1, subsequently we show that \mathbb{E}^* is GAS (see Definition 7).

Important notes: (i) We assume that $\mathcal{Y}(0)$ is arbitrarily selected in \mathbb{Y} and kept fixed throughout the proof. (ii) We will introduce more notation and definitions as needed. (iii) We will make extensive use of the best response map \mathcal{M} . We will also refer repeatedly to \mathbb{E}^* , \mathcal{Q}^* , ζ_1^* , and ζ_2^* as defined in the statement of Theorem 1.

Proof structure: We follow a LaSalle approach (LaSalle, 1960) with four steps: **Step 1:** We show that \mathbb{E}^* satisfies (GASa); **Step 2:** We prove that $\{q(t) \mid t \geq 0\}$ is bounded for any $\mathcal{Y}(0)$ in \mathbb{Y} ; **Step 3:** We show that \mathbb{E}^* satisfies (GASb); **Step 4:** We prove that \mathbb{E}^* satisfies (GASc).

A.1. Step 1: showing that \mathbb{E}^* satisfies (GASa)

We start by defining $\bar{x} := [0 \ \dots \ 1]'$ and $\underline{x} := [1 \ \dots \ 0]'$. Now, define \mathbb{A}^* as follows:

$$\mathbb{A}^* := \{(x, q) \mid x \in \mathcal{M}(q\bar{\beta} + r^0), \bar{\beta}'x = \beta^*\}$$

or use (12), and (NS) to rewrite \mathbb{A}^* with $p = q\bar{\beta} + r^0$ as:

$$\mathbb{A}^* = \{(x, q) \in \mathbb{X} \times \mathbb{R} \mid \mathcal{S}(x, p) = 0, \bar{\beta}'x = \beta^*\}. \quad (\text{A.1})$$

Since, by Remark 3, β^* satisfies $\bar{\beta}_1 < \beta^* < \bar{\beta}_n$, subsequently we will be able to show the following equality:

$$\mathbb{A}^* = \{x^*\} \times \mathcal{Q}^*. \quad (\text{A.2})$$

We now proceed to prove (A.2) for Cases I and II:

Case I: Let $\{i^*, i^* + 1\}$ be the support of x^* (see Remark 3). If $q = 0$ then we conclude, from the fact that $\{i^*, i^* + 1\}$ is the support of any x in $\mathcal{M}(r^0)$, that $\{x \in \mathcal{M}(r^0) \mid \beta^* = \bar{\beta}'x\} = \{x^*\}$. If $q > 0$ and x is any element in $\mathcal{M}(q\bar{\beta} + r^0)$, then $x_1 = \dots = x_{i^*} = 0$ and, consequently, $\bar{\beta}'x > \beta^*$. Analogously, if $q < 0$ then $\bar{\beta}'x < \beta^*$ for all x in $\mathcal{M}(q\bar{\beta} + r^0)$.

Case II: Let i^* be the support of x^* . If $-\zeta_2^* \leq q \leq \zeta_1^*$, then $\{x \in \mathcal{M}(q\bar{\beta} + r^0) \mid \beta^* = \bar{\beta}'x\} = \{x^*\}$. If $q > \zeta_1^*$, then $\mathcal{M}(q\bar{\beta} + r^0) = \{\bar{x}\}$, which would not be viable for \mathbb{A}^* because $\bar{\beta}'\bar{x} = \beta_n > \beta^*$. A similar argument shows that $q < -\zeta_2^*$ is not viable for \mathbb{A}^* .

The proof for Step 1 is concluded by using (A.1) and (A.2) in conjunction with (18) and Remark 4. Or, equivalently, the following holds for $\mathbb{E}^* = (\mathcal{I}^*, \mathcal{R}^*) \times \mathbb{A}^*$:

$$\mathcal{Y} \in \mathbb{E}^* \Leftrightarrow \mathcal{L}(\mathcal{Y}) = 0. \quad (\text{A.3})$$

A.2. Step 2: proving that $\{q(t) \mid t \geq 0\}$ is bounded

Start by selecting and keeping fixed throughout the proof two positive constants $\zeta_1 > \zeta_1^*$ and $\zeta_2 > \zeta_2^*$.

We proceed with the following additional definitions:

$$\mathbb{P}_1 := \left\{ \vec{\beta} + \gamma r^0 \mid 0 \leq \gamma \leq \zeta_1^{-1} \right\},$$

$$\mathbb{P}_2 := \left\{ -\vec{\beta} + \gamma r^0 \mid 0 \leq \gamma \leq \zeta_2^{-1} \right\}.$$

Remark 7. Notice that if p is in \mathbb{P}_1 then $\mathcal{M}(p) = \{\bar{x}\}$, and if p is in \mathbb{P}_2 then $\mathcal{M}(p) = \{x\}$.

We are now ready to state the following lemma.

Lemma 1. Given any $\epsilon > 0$, there is $\delta > 0$ such that:

$$\max \left\{ \|x - \bar{x}\| \mid x \in \mathbb{X}, \min_{p \in \mathbb{P}_1} \mathcal{P}(x, p) \leq \delta \right\} < \epsilon, \quad (\text{A.4a})$$

$$\max \left\{ \|x - \underline{x}\| \mid x \in \mathbb{X}, \min_{p \in \mathbb{P}_2} \mathcal{P}(x, p) \leq \delta \right\} < \epsilon. \quad (\text{A.4b})$$

Proof of Lemma 1. We will prove the lemma by showing that assuming it was not valid would lead to a contradiction. Hence, without loss of generality, assume that there was $\epsilon^* > 0$ for which no $\delta > 0$ satisfying (A.4a) existed. (The case in which no $\delta > 0$ satisfying (A.4b) existed would have been analogous.) In order to reach a contradiction, we start by noticing that under the assumption the following would hold:

$$\|x^{(\ell)} - \bar{x}\| \geq \epsilon^* \text{ and } \mathcal{P}(x^{(\ell)}, p^{(\ell)}) \leq \frac{1}{\ell}, \quad \ell \geq 1$$

for a sequence $(x^{(\ell)}, p^{(\ell)})$ satisfying:

$$x^{(\ell)} \in \arg \max \left\{ \|x - \bar{x}\| \mid x \in \mathbb{X}, \min_{p \in \mathbb{P}_1} \mathcal{P}(x, p) \leq \frac{1}{\ell} \right\},$$

$$p^{(\ell)} \in \arg \min_{p \in \mathbb{P}_1} \mathcal{P}(x^{(\ell)}, p).$$

We proceed by noting that since the sequence $(x^{(\ell)}, p^{(\ell)})$ would take values in the compact set $\mathbb{X} \times \mathbb{P}_1$, it would have an accumulation point $(x^*, p^*) \in \mathbb{X} \times \mathbb{P}_1$. By continuity of $\|\cdot\|$ and \mathcal{P} , the pair (x^*, p^*) would satisfy: (i) $\|x^* - \bar{x}\| \geq \epsilon^*$ and (ii) $\mathcal{P}(x^*, p^*) = 0$. However, since $p^* \in \mathbb{P}_1$, we can use Remark 7, (NS), (ii) and (12) to conclude that $x^* = \{\bar{x}\}$, which would contradict (i). \square

Since \mathcal{L} is lower bounded, we infer from (19) that $\tilde{\mathcal{I}}(t)$ and $\tilde{\mathcal{R}}(t)$ are square-integrable. Also, $\tilde{\mathcal{I}}(t)$ and $\tilde{\mathcal{R}}(t)$ are uniformly continuous (bounded derivatives). Thus, we conclude using Barbalat's Lemma (Farkas & Wegner, 2016, Theorem 1) that

$$\lim_{t \rightarrow \infty} \tilde{\mathcal{I}}(t)^2 + \tilde{\mathcal{R}}(t)^2 = 0. \quad (\text{A.5})$$

Using a similar argument and (13), we conclude that

$$\lim_{t \rightarrow \infty} \mathcal{P}(x(t), \bar{p}(t)) = 0, \quad \bar{p}(t) := \frac{p(t)}{\max\{1, |q(t)|\}}. \quad (\text{A.6})$$

Namely, from (13) and (19) we infer that $\mathcal{P}(x(t), \bar{p}(t))$ is integrable. In order to use Barbalat's Lemma to prove (A.6), it suffices to establish uniform continuity of $\mathcal{P}(x(t), \bar{p}(t))$ (as a function of t). To do so, we observe that (i) $\tilde{\mathcal{I}}(t) \geq \eta(\beta_1 - \sigma) > 0$ and (ii) according to (19) $\mathcal{L}(\mathcal{Y}(t))$ remains bounded. It follows from (i) and (ii) that $\ln \mathcal{I}(t)$ is also bounded, and, from (8) and (17c), that $\dot{q}(t)$ is bounded and $\bar{p}(t)$ is uniformly continuous. Since from (EDM) it holds that $\|\dot{x}\|_\infty \leq n\bar{\tau}$, we conclude that $x(t)$ is also uniformly continuous. Consequently, $\mathcal{P}(x(t), \bar{p}(t))$ is uniformly continuous.

Define $\xi := \nu^2 \min\{\beta_n - \beta^*, \beta^* - \beta_1\}$ and select $\epsilon > 0$ such that (i) $\nu^2(\vec{\beta}'x - \beta^*) > \frac{2}{3}\xi$ for all x in \mathbb{X} satisfying $\|x - \bar{x}\| < \epsilon$ and (ii) $\nu^2(\vec{\beta}'x - \beta^*) < -\frac{2}{3}\xi$ for all x in \mathbb{X} satisfying $\|x - \underline{x}\| < \epsilon$.

From Lemma 1, we know that there is $\delta > 0$ such that (A.4a)–(A.4b) hold. Furthermore, from (A.6) we know that there is κ such that, for all $t \geq \kappa$, we have $\mathcal{P}(x(t), \bar{p}(t)) \leq \delta$. Consequently, we conclude that: (a) if $q(t) \geq \zeta_1$ and $t \geq \kappa$, then $\bar{p}(t)$ is in \mathbb{P}_1 and $\min_{p \in \mathbb{P}_1} \mathcal{P}(x(t), p) \leq \mathcal{P}(x(t), \bar{p}(t)) \leq \delta$ and (b) if $q(t) \leq -\zeta_2$ and $t \geq \kappa$ then $\bar{p}(t)$ is in \mathbb{P}_2 and $\min_{p \in \mathbb{P}_2} \mathcal{P}(x(t), p) \leq \mathcal{P}(x(t), \bar{p}(t)) \leq \delta$. Hence, combining (i), (a), and (A.4a) we arrive at (A.7a), and from (ii), (b), and (A.4b) we infer (A.7b).

$$q(t) \geq \zeta_1 \implies \nu^2(\mathcal{B}(t) - \beta^*) > \frac{2}{3}\xi, \quad t \geq \kappa. \quad (\text{A.7a})$$

$$q(t) \leq -\zeta_2 \implies \nu^2(\mathcal{B}(t) - \beta^*) < -\frac{2}{3}\xi, \quad t \geq \kappa. \quad (\text{A.7b})$$

From (8), (17c), and (A.5), and the fact that $\hat{\mathcal{I}}(t) \geq \eta(\beta_1 - \sigma) > 0$, we can select $\underline{t} \geq \kappa$ satisfying:

$$|\dot{q}(t) + \nu^2(\mathcal{B}(t) - \beta^*)| < \frac{1}{3}\xi, \quad t \geq \underline{t}. \quad (\text{A.8})$$

From (A.7), and (A.8), we can finally conclude that (i) if $q(t) \geq \zeta_1$ and $t \geq \underline{t}$, then $\dot{q} < -\frac{1}{3}\xi$ and (ii) if $q(t) \leq -\zeta_2$ and $t \geq \underline{t}$, then $\dot{q} > \frac{1}{3}\xi$. Hence, we can conclude that there is $\bar{t} \geq \underline{t}$ such that the following holds:

$$-\zeta_2 \leq q(t) \leq \zeta_1, \quad t \geq \bar{t}. \quad (\text{A.9})$$

A.3. Step 3: showing that \mathbb{E}^* satisfies (GASb)

Subsequently, we will show by construction the existence of an accumulation point of $\{\mathcal{Y}(t) \mid t \geq 0\}$ in \mathbb{E}^* .

Remark 8. Before we proceed, we observe that since ζ_1 and ζ_2 were any arbitrarily selected constants satisfying $\zeta_1 > \zeta_1^*$ and $\zeta_2 > \zeta_2^*$, we can infer from (A.9) that any accumulation point of $\{q(t) \mid t \geq 0\}$ must be in \mathcal{Q}^* .

We start by observing that continuity of $\dot{q}(t)$ and (A.9) imply that 0 is an accumulation point of $\{\dot{q}(t) \mid t \geq 0\}$. Consequently, from (8), (17c), and (A.5), and the fact that $\hat{\mathcal{I}}(t) \geq \eta(\beta_1 - \sigma) > 0$, we conclude that $(\mathcal{I}^*, \mathcal{R}^*, \beta^*)$ is an accumulation point of $\{(\mathcal{I}, \mathcal{R}, \mathcal{B})(t) \mid t \geq \bar{t}\}$.

Let $t^{(n)}$ be a sequence of times such that $(\mathcal{I}, \mathcal{R}, \mathcal{B})(t^{(n)})$ converges to $(\mathcal{I}^*, \mathcal{R}^*, \beta^*)$. Then, the sequence $(\mathcal{I}, \mathcal{R}, \mathcal{B}, x, q)(t^{(n)})$ also has an accumulation point $(\mathcal{I}^*, \mathcal{R}^*, \beta^*, \bar{x}, \dot{q})$ because from (A.9) we know that, for $t \geq \bar{t}$, the pair $(x, q)(t)$ takes values in a compact set $\mathbb{X} \times [-\zeta_2, \zeta_1]$. We now proceed to observe that by continuity of \mathcal{P} and (A.6), it must be that $\mathcal{P}(\bar{x}, \frac{1}{\max\{|\dot{q}|, 1\}}(\dot{q}\vec{\beta} + r^0)) = 0$. Consequently, from (NS), (12), the fact that $\beta^* = \vec{\beta}'\bar{x}$ and Remark 8, we conclude that (\bar{x}, \dot{q}) must be in the set \mathbb{A}^* characterized in (A.2) for Cases I and II.

A.4. Step 4: showing that \mathbb{E}^* satisfies (GASc)

In Step 3, we constructed an accumulation point e^* of $\{\mathcal{Y}(t) \mid t \geq 0\}$ in \mathbb{E}^* . Hence, there is a sequence $t^{(n)}$ such that $\lim_{n \rightarrow \infty} \mathcal{Y}(t^{(n)}) = e^*$. However, from (A.3) and the continuity of \mathcal{L} , we conclude that $\lim_{n \rightarrow \infty} \mathcal{L}(\mathcal{Y}(t^{(n)})) = \mathcal{L}(e^*) = 0$. Furthermore, since (19) guarantees that $\mathcal{L}(\mathcal{Y}(t))$ is non-increasing, we conclude that the following holds:

$$\lim_{t \rightarrow \infty} \mathcal{L}(\mathcal{Y}(t)) = 0. \quad (\text{A.10})$$

Now take any candidate accumulation point \mathcal{Y}^* in \mathbb{Y} . From (A.10) and the continuity of \mathcal{L} it follows that $\mathcal{L}(\mathcal{Y}^*) = 0$, which from (A.3) implies that $\mathcal{Y}^* \in \mathbb{E}^*$.

References

Al-Radhawi, M. A., Sadeghi, M., & Sontag, E. D. (2021). Long-term regulation of prolonged epidemic outbreaks in large populations via adaptive control: a singular perturbation approach. *arXiv:2103.08488*.

Amaral, M. A., de Oliveira, M. M., & Javarone, M. A. (2021). An epidemiological model with voluntary quarantine strategies governed by evolutionary game dynamics. *Chaos, Solitons & Fractals*, 143, Article 110616.

Anderson, R. M., & May, R. M. (1991). *Infectious diseases of humans: Dynamics and control*. Oxford Science Publications.

Arcak, M., & Martins, N. C. (2021). Dissipativity tools for convergence to Nash equilibria in population games. *IEEE Transactions on Control of Network Systems*, 8(1), 39–50.

Arefin, M. R., Masaki, T., & Tanimoto, J. (2020). Vaccinating behaviour guided by imitation and aspiration. *Proceedings of the Royal Society of London, Series A (Mathematical and Physical Sciences)*, 476(2239), Article 20200327.

Bauch, C. T. (2005). Imitation dynamics predict vaccinating behaviour. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, 272(1573), 1669–1675.

Bauch, C. T., & Earn, D. J. D. (2004). Vaccination and the theory of games. *Proceedings of the National Academy of Sciences*, 101(36), 13391–13394.

Brown, G. W., & von Neumann, J. (1950). *Solutions of games by differential equations: Tech. rep.*, RAND Corporation.

Certório, J. (2022). *jcrt/EPG: v1.1*. Zenodo, <http://dx.doi.org/10.5281/zenodo.7098545>.

Chang, S. L., Piraveenana, M., Pattison, P., & Prokopenko, M. (2020). Game theoretic modelling of infectious disease dynamics and intervention methods: a review. *Journal of Biological Dynamics*, 14(1), 57–89.

Di Lauro, F., Kiss, I. Z., & Miller, J. C. (2021). Optimal timing of one-shot interventions for epidemic control. *PLoS Computational Biology*, 17(3), Article e1008763.

d’Onofrio, A., Manfredi, P., & Poletti, P. (2011). The impact of vaccine side effects on the natural history of immunization programmes: An imitation-game approach. *Journal of Theoretical Biology*, 273(1), 63–71.

Farkas, B., & Wegner, S.-A. (2016). Variations on Barbalat’s Lemma. *American Mathematical Monthly*, 123(8), 825–830.

Fox, M. J., & Shamma, J. S. (2013). Population games, stable games, and passivity. *Games*, 4, 561–583.

Godara, P., Herminghaus, S., & Heiderman, K. M. (2021). A control theory approach to optimal pandemic mitigation. *PLoS One*, 16(2), 1–16.

Hofbauer, J., & Sandholm, W. H. (2009). Stable games and their dynamics. *Journal of Economic Theory*, 144(4), 1665–1693.e4.

Kabir, K. A., & Tanimoto, J. (2020). Evolutionary game theory modelling to represent the behavioural dynamics of economic shutdowns and shield immunity in the COVID-19 pandemic. *Royal Society Open Science*, 7(9), Article 201095.

Kara, S., & Martins, N. C. (2021). Pairwise comparison evolutionary dynamics with strategy-dependent revision rates: stability and delta-passivity (Expanded version). *arXiv:2107.02835*.

Kermack, W. O., & McKendrick, A. G. (1927). A contribution to the mathematical theory of epidemics. *Proceedings of the Royal Society of London, Series A (Mathematical and Physical Sciences)*, 115(772), 700–721.

Kuga, K., & Tanimoto, J. (2018). Which is more effective for suppressing an infectious disease: imperfect vaccination or defense against contagion?. *Journal of Statistical Mechanics: Theory and Experiment*, 2(2), 023407.

La, R. J. (2019). Influence of clustering on cascading failures in interdependent systems. *IEEE Transactions on Network Science and Engineering*, 6(3), 351–363.

LaSalle, J. P. (1960). Some extensions of Liapunov’s second method. *IRE Transactions on Circuit Theory*, 7(4), 520–527.

Mei, W., Mohagheghi, S., Zampieri, S., & Bullo, F. (2017). On the dynamics of deterministic epidemic propagation over networks. *Annual Reviews in Control*, 44, 116–128.

Nash, J. F. Jr. (1951). *Annals of mathematics second series: vol. 54, Non-cooperative games* (no. 2), (pp. 286–295).

Nowzari, C., Preciado, V. M., & Pappas, G. J. (2016). Analysis and control of epidemics: a survey of spreading processes on complex networks. *IEEE Control Systems Magazine*, 26–44.

Nowzari, C., Preciado, V. M., & Pappas, G. J. (2017). Optimal resource allocation for control of networked epidemic models. *IEEE Transactions on Control of Network Systems*, 4(2), 159–169.

O’Regan, S. M., Kelly, T. C., Korobeinikov, A., O’Callaghan, M. J., & Pokrovskii, A. V. (2010). Lyapunov functions for SIR and SIRS epidemic models. *Applied Mathematics Letters*, 23(4), 446–448.

Ottaviano, S., De Pellegrini, F., Bonacorsi, S., & Van Mieghem, P. (2018). Optimal curing policy for epidemic spreading over a community network with heterogeneous population. *J. Complex Netw.*, 6(5), 800–829.

Paré, P. E., Beck, C. L., & Basar, T. (2020). Modeling, estimation, and analysis of epidemics over networks: an overview. *Annual Reviews in Control*, 50, 345–360.

Paré, P. E., Beck, C. L., & Nedić, A. (2018). Epidemic processes over time-varying networks. *IEEE Transactions on Control of Network Systems*, 5(3), 1322–1334.

Park, S., Martins, N. C., & Shamma, J. S. (2019). From population games to payoff dynamics models: A passivity-based approach. In *Proc. of IEEE conf. decision control* (pp. 6584–6601).

Park, S., Shamma, J. S., & Martins, N. C. (2018). Passivity and evolutionary game dynamics. In *Proc. of IEEE conf. decision control* (pp. 3553–3560).

Pastor-Satorras, R., Castellano, C., Van Mieghem, P., & Vespignani, A. (2015). Epidemic processes in complex networks. *Reviews of Modern Physics*, 87(3), 925–979.

Preciado, V. M., Zargham, M., Enyioha, C., Jadbabaie, A., & Pappas, G. (2013). Optimal vaccine allocation to control epidemic outbreaks in arbitrary networks. In *Proc. of IEEE conf. decision control* (pp. 7486–7491).

Preciado, V. M., Zargham, M., Enyioha, C., Jadbabaie, A., & Pappas, G. J. (2014). Optimal resource allocation for network protection against spreading processes. *IEEE Transactions on Control of Network Systems*, 1(1), 99–108.

Sandholm, W. H. (2005). Excess payoff dynamics and other well-behaved evolutionary dynamics. *Journal of Economic Theory*, 124(2), 149–170.

Sandholm, W. H. (2010a). *Population games and evolutionary dynamics*. MIT Press.

Sandholm, W. H. (2010b). Pairwise comparison dynamics and evolutionary foundations for Nash equilibrium. *Games*, 1(1), 3–17.

Sandholm, W. H. (2015). In H. P. Young, & S. Zamir (Eds.), *Handbook of game theory*, vol. 4 (pp. 703–775). North Holland.

Smith, M. J. (1984). The stability of a dynamic model of traffic assignment: an application of a method of Lyapunov. *Transportation Science*, 18(3), 245–252.

Sontag, E. D. (2021). An explicit formula for minimizing the infected peak in an SIR epidemic model when using a fixed number of complete lockdowns. *International Journal of Robust and Nonlinear Control*, 1–24.

Weibull, J. W. (1995). *The mass-action interpretation of nash equilibrium: Working paper series no. 427*, Research Institute of Industrial Economics.



Nuno C. Martins received his Ph.D. degree in Electrical Engineering and Computer Science with a minor in Mathematics from Massachusetts Institute of Technology (MIT), Cambridge, in 2004. He is Professor of Electrical Engineering in the Electrical and Computer Engineering Department of the University of Maryland at College Park, where he also holds a joint appointment with the Institute for Systems Research. He was Director of the Maryland Robotics Center from 2012 until 2014. Prof. Martins received the 2006 American Automatic Control Council O. Hugo Schuck Award, a National Science Foundation CAREER Award in 2007, the 2008 IEEE CSS Axelby Award for the best paper in the IEEE Transactions on Automatic Control, the 2010 Outstanding ISR Faculty Award, the 2010 George Corcoran Award from the ECE Department / UMD and he was an UMD/ADVANCE Leadership Fellow in 2013. He served as Associate Editor for Systems and Control Letters (Elsevier), Automatica and the IEEE Control Systems Society Conference Editorial Board. He was also a program Vice-Chair for the IEEE Conference on Decision and Control in 2013 and 2014.



Jair Certório received his B.S. degree in Electrical Engineering from Universidade Estadual de Maringá, Brazil, in 2018. He is currently working towards a Ph.D. degree in Electrical and Computer Engineering from the University of Maryland, College Park.



Richard J. La received his Ph.D. degree in Electrical Engineering from the University of California, Berkeley in 2000. Since 2001 he has been on the faculty of the Department of Electrical and Computer Engineering at the University of Maryland, where he is currently a Professor. He is currently an associate editor for IEEE/ACM Transactions on Networking, and served as an associate editor for IEEE Transactions on Information Theory and IEEE Transactions on Mobile Computing.