On Assessing Control Actions for Epidemic Models on Temporal Networks

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Published in:
IEEE Control Systems Letters

DOI:
10.1109/LCSYS.2020.2993104

Publication date:
2020

On Assessing Control Actions for Epidemic Models on Temporal Networks

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***Abstract*—In this letter, we propose an epidemic model over temporal networks that explicitly encapsulates two different control actions. We develop our model within the theoretical framework of activity driven networks (ADNs), which have emerged as a valuable tool to capture the complexity of dynamical processes on networks, coevolving at a comparable time scale to the temporal network formation. Specifically, we complement a susceptible–infected–susceptible epidemic model with features that are typical of nonpharmaceutical interventions in public health policies: i) actions to promote awareness, which induce people to adopt self-protective behaviors, and ii) confinement policies to reduce the social activity of infected individuals. In the thermodynamic limit of large-scale populations, we use a mean-field approach to analytically derive the epidemic threshold, which offers viable insight to devise containment actions at the early stages of the outbreak. Through the proposed model, it is possible to devise an optimal epidemic control policy as the combination of the two strategies, arising from the solution of an optimization problem. Finally, the analytical computation of the epidemic prevalence in endemic diseases on homogeneous ADNs is used to optimally calibrate control actions toward mitigating an endemic disease. Simulations are provided to support our theoretical results.**

***Index Terms*—Control of networks, epidemics, epidemiology, network analysis and control, predictive model.**

Manuscript received March 16, 2020; revised April 17, 2020; accepted May 4, 2020. Date of publication May 7, 2020; date of current version May 27, 2020. This work was supported in part by the National Science Foundation under Grant CMMI-1561134 and Grant CMMI-2027990, in part by the Compagnia di San Paolo, in part by MAECI ("Mac2Mic"), in part by the European Research Council under Grant ERC-CoG-771687, and in part by the Netherlands Organization for Scientific Research under Grant NWO-vidi-14134. Recommended by Senior Editor M. Arcak. *(Corresponding authors: Alessandro Rizzo; Maurizio Porfiri.)*

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Digital Object Identifier 10.1109/LCSYS.2020.2993104

I. Introduction

MATHEMATICAL models of epidemic outbreaks on networks have become increasingly popular in the last decades among scientists from many research fields [1]–[4]. The analysis of such models has allowed to gain new insight into the mechanisms that govern the spread of infectious diseases. Within the systems and controls community, many efforts have been put forward to leverage this insight and incorporate control actions in epidemic models to combat the spread of epidemic diseases [5], [6]. In particular, the rigorous analysis of epidemic models has elucidated the role of contact networks in shaping the evolution of epidemic outbreaks, informing effective control policies [7]–[9].

Most of the results on epidemic processes in the literature, in particular those that explicitly contemplate control actions, consider time-invariant networks as an exemplary social structure. However, many real networks have a time-varying structure that coevolves at a comparable time-scale to the epidemic process [10]. Understanding how temporal networks influence the propagation of epidemic diseases and how such knowledge can be used to design control policies is of paramount importance to help support public health administrations in contrasting epidemic outbreaks [10], [11].

Activity driven networks (ADNs) have emerged as a valuable mathematical framework to represent and study the coevolution of dynamical processes on and of networks [12], [13]. The main strength of ADNs lies in their simplicity, whereby the temporal nature of each individual is captured by a single parameter, that is, his/her propensity to generate interactions with others, called *activity*. Such a formulation allows for modeling heterogeneous temporal networks, enabling the analytical tractability of epidemic processes [12], [14] and the rigorous study of key features of real-world systems, such as burstiness [15] and higher-order relationships [16]. Other techniques to deal with temporal networks include Lyapunov stability [17], aggregated Markov processes [18], and temporally-switching networks [19].

In this letter, we propose an ADN-based epidemic model, which allows for the description at the microscopic level of epidemic processes coevolving with a temporal network, encapsulating two realistic control actions, which are typical of nonpharmaceutical interventions dictated by public health authorities. Specifically, we consider awareness campaigns and confinement. The former aims at increasing the awareness of the disease of uninfected individuals and, thus, induce

them to adopt self-protective behaviors [20], [21]. The latter is realized by isolating infected individuals (for instance, through quarantine), toward the interruption of the chain of contagions [22].

Here, we model the epidemic process using a susceptible–alert–infected–susceptible (SAIS) model, which is an extension of the well-known susceptible–infected–susceptible (SIS) model, where alerted individuals take self-protective behaviors to reduce their susceptibility to the infection spreading. In the original implementation of the SAIS model, individuals become alerted by the presence of infected or alerted neighbors [23], [24]. An optimal control problem has been formulated for this model in [25]. A different implementation considers an adaptive scenario, in which alerted individuals change local contacts rather than adopting self-protective behaviors [26]. Preliminary results on the original SAIS models on ADNs can be found in [27], while the problem of cost-aware containment of epidemics is studied for the adaptive scenario in [28].

In our implementation, we assume that self-protective behaviors are triggered by control actions dictated by the public health authorities and are always successful in preventing contagion (which is appropriate for many sexually-transmitted diseases and parasite infections). However, these behaviors have a nonneglible cost, so that the individual adoption of such behaviors is the result of a personal cost-benefit assessment. In this vein, the use of pre-exposure prophylaxis for HIV prevention is a paradigmatic example of such a scenario [29]. Similar to [30], we include a state-dependent behavior in the ADN model, which models the effect of containment and isolation techniques.

In addition to the mathematical formalization of the model, the main theoretical contributions of this letter are: i) the analytical derivation of the epidemic threshold for the SAIS model on ADNs in the thermodynamic limit of large-scale populations through a mean-field argument; ii) the computation of the prevalence of the disease (that is, the fraction of infected individuals) in the endemic state for homogeneous populations; iii) the use of our analytical findings to rigorously evaluate the effectiveness of the two control actions, in the early stages of the epidemics; and iv) the formalization of an optimization problem to devise an optimal control policy as a mix of the two actions, both in the early stages of the epidemics and in the endemic regime of the disease. Simulations are presented to validate analytical findings and provide further insight into the epidemic process.

We gather here some notation used throughout this letter. We denote by $\mathbb{R}$, $\mathbb{R}_{\geq 0}$, and $\mathbb{R}_{>0}$ the set of real, real nonnegative, and strictly positive real numbers, respectively. Given a continuous-time function $x(t)$, we define $x(t^-) := \lim_{s \nearrow t} x(s)$ and $x(t^+) := \lim_{s \searrow t} x(s)$.

II. Model

We consider a population of individuals $\mathcal{V} = \{1, \dots, n\}$, each one identified by a node in an undirected temporal network [10]. Individuals generate time-varying interactions that are modeled through a temporal network $(\mathcal{V}, \mathcal{E}(t))$, where $\mathcal{E}(t)$ is the time-varying set of undirected links. Hence, $\{v, w\} \in \mathcal{E}(t)$ implies that nodes v and w interact at time t.

A. Activity Driven Networks

The temporal network is generated according to a continuous-time ADN model [13]. Each individual $v \in \mathcal{V}$ is characterized by an activity rate $a_v \in \mathbb{R}_{>0}$, which represents his/her propensity to interact with others. The network is dynamically generated as follows: i) at time $t = 0$, the link set is initialized as $\mathcal{E}(t) = \emptyset$. Each node $v \in \mathcal{V}$ is associated with a Poisson clock with rate equal to a_v, each one independent of the others; ii) time progresses until the click of any of the n Poisson clocks involved in the process clicks, iii) if the clock associated with node $v \in \mathcal{V}$ clicks at time t, the individual corresponding to node v is activated and he/she selects a fellow individual $w \in \mathcal{V}$ uniformly at random to connect with; iv) the undirected link $\{v, w\}$ is instantaneously added to $\mathcal{E}(t)$ and an instantaneous interaction (i.e., contagion or diffusion of information) is enabled; and v) the link is immediately removed from the set, the Poisson process associated with node v is re-initialized, and the process resumes from ii).

B. Epidemic Model

We consider an SAIS model [23], in which individuals can have three different states, depending on their health status and behavior. An individual can be either *susceptible* to the disease (denoted by S), *infected* by the disease (I), or he/she can be aware of the risks connected to the infection and consequently adopt *self-protective* behaviors, preventing him/herself from contracting the disease (P). We assume that individuals can be infected multiple times by the disease (this is often the case of parasites, such as lice and pinworms, and several bacterial sexually-transmitted infections). The SAIS model can have different implementations, depending on the specific mechanisms that govern the state transitions [23], [24], [27]. Here, we consider the following setup.

Each individual $v \in \mathcal{V}$ is given a ternary state $X_v(t) \in \{S, P, I\}$, which represents the individuals health state and behavior at time t. The state evolves in continuous time $t \in \mathbb{R}_{\geq 0}$ along with the network formation process, according to the following three mechanisms.

Contagion: A susceptible individual $(X_v(t^-) = S)$ who contacts an infected one (that is, $(v, w) \in \mathcal{E}(t)$ and $X_w(t) = I$) becomes infected according to a probabilistic mechanism. Specifically, he/she contracts the disease with probability $\lambda \in [0, 1]$, independent of the others.

Recovery: An infected individual $(X_v(t^-) = I)$ spontaneously recovers and becomes susceptible according to a Poisson clock with rate $\mu \in \mathbb{R}_{>0}$, independent of the others. We assume that recovered individuals assume self-protective behaviors $(X_v(t^+) = P)$.

Unprotecting: An individual with $X_v(t^-) = P$ spontaneously return to his/her regular behavior, becoming susceptible to the epidemics $(X_v(t^+) = S)$ according to a Poisson clock with rate $\nu \in \mathbb{R}_{>0}$, independent of the others. This modeling choice

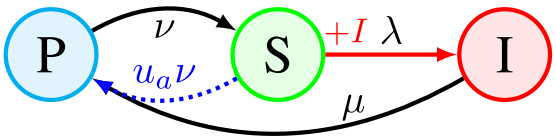


Fig. 1. Schematic of the state transitions of the SAIS model. The blue dotted arrow is due to the awareness control action.

is informed by the high social and economic costs of self-protective behaviors.

The state transitions induced by these mechanisms are represented in Fig. 1.

Remark 1: The model parameters have the following interpretation: λ is the infection probability after an unprotected contact with an infectious individual; μ governs the duration of illness, whose average duration is $1/\mu$; ν is monotonically positively correlated with the cost of self-protective behaviors, so that the larger ν, the faster individuals tend to stop adopting them (the average adoption time is equal to $1/\nu$).

C. Control Actions

Here, we present the two possible control actions that are implemented in our modeling framework.

Awareness: Through information campaigns, people can be prompted to take self-protective behaviors. We introduce a parameter $u_a \in \mathbb{R}_{\geq 0}$ that quantifies such an effort by public health administrations. Specifically, each susceptible individual ($X_v(t^-) = S$) starts adopting self-protective behaviors ($X_v(t^+) = P$) according to a Poisson clock with rate $u_a\nu$, independent of the others.

Confinement: The activity of infected individuals can be lowered through confinement strategies. We introduce a parameter $u_c \in [0, 1]$ that quantifies the effect of such policies. Specifically, we consider a state-dependent version of ADNs, similar to [14], [30], in which the Poisson clock that governs the activation of an infected individual $v \in \mathcal{V}$ with $X_v(t) = I$ has rate $a_v(1 - u_c)$.

III. Main Results

The SAIS epidemic process described in Section II induces an n-dimensional Markov process $X(t)$ in the state space $\{S, P, I\}^{\mathcal{V}}$, where the generic vth node has instantaneous transition rates summarized by the matrix

$$Q_v = \begin{bmatrix} \cdot & u_a\nu & \frac{\lambda a_v}{n}\sum_{w:X_w=I} 1 + (1-u_c)\frac{\lambda}{n}\sum_{w:X_w=I} a_w \\ \nu & \cdot & 0 \\ 0 & \mu & \cdot \end{bmatrix},$$

whose rows (columns) correspond to the three states S, P, and I, respectively. Hence, the probability that v changes his/her state from $h \in \{S, P, I\}$ to $k \in \{S, P, I\}$ is equal to

$$\mathbb{P}[X_v(t + \Delta t) = k \,|\, X_v(t) = h] = (Q_v)_{hk}\Delta t + o(\Delta t),$$

for any $h \neq k$. The diagonal entries of Q are equal to the opposite of the sum of the other two entries, to ensure that the rows of Q sum to 0.

Note that the dimension of the state-space of the Markov process $X(t)$ grows exponentially with n and the expression of Q_v depends on the state of the other nodes. This joint dependence along with exponential growth of the state-space hinder the analysis of $X(t)$ for large-scale systems. Following [3], we study a continuous-state deterministic mean-field relaxation of the dynamics. Instead of the evolution of the individuals' state, we study the probability for each individual to attain each of the three states, that is, $s_v(t) := \mathbb{P}[X_v(t) = S]$, $p_v(t) := \mathbb{P}[X_v(t) = P]$, and $i_v(t) := \mathbb{P}[X_v(t) = I]$. These three probabilities are governed by a system of $3n$ ordinary differential equations (ODEs) [3], which can be obtained from $[\dot{s}_v\, \dot{p}_v\, \dot{i}_v] = [s_v\, p_v\, i_v]Q_v$, $\forall\, v \in \mathcal{V}$, yielding

$$\begin{aligned} \dot{s}_v &= -u_a\nu s_v + \nu p_v \\ &\quad - \lambda s_v\left[a_v\frac{1}{n}\sum_{w\in\mathcal{V}} i_w + (1-u_c)\frac{1}{n}\sum_{w\in\mathcal{V}} a_w i_w\right], \\ \dot{p}_v &= u_a\nu s_v - \nu p_v + \mu i_v, \\ \dot{i}_v &= -\mu i_v + \lambda s_v\left[a_v\frac{1}{n}\sum_{w\in\mathcal{V}} i_w + (1-u_c)\frac{1}{n}\sum_{w\in\mathcal{V}} a_w i\right]. \end{aligned} \quad (1)$$

The following result proves that the system of ODEs in Eq. (1) is well-defined, that is, $(s_v(t), p_v(t), i_v(t))$ is always a probability vector.

Lemma 1: The set $\{(s_v, p_v, i_v) : s_v, p_v, i_v \geq 0,\ s_v + p_v + i_v = 1,\ \forall\, v \in \mathcal{V}\}$ is positive invariant under Eq. (1).

Proof: We immediately verify that, if one of the variables is equal to 0, then its derivative is always nonnegative. Hence, the nonnegative orthant is a positive invariant set. We further observe that $\dot{s}_v + \dot{p}_v + \dot{i}_v = 0$, preserving the sum of the three variable for each node v, which proves our claim. ■

As a consequence of Lemma 1, only $2n$ of the ODEs from Eq. (1) are linearly independent.

Before presenting our main results, we introduce some more notation. We define the average activity of the whole population and its second moment as

$$\alpha_1 := \frac{1}{n}\sum_{v\in\mathcal{V}} a_v, \qquad \alpha_2 := \frac{1}{n}\sum_{v\in\mathcal{V}} a_v^2.$$

Similarly, we define the macroscopic variables

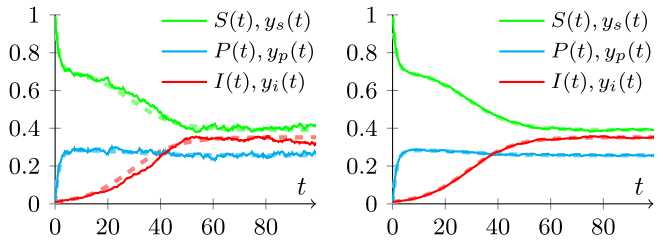
$$y_s := \frac{1}{n}\sum_{v\in\mathcal{V}} s_v, \quad y_p := \frac{1}{n}\sum_{v\in\mathcal{V}} p_v, \quad y_i := \frac{1}{n}\sum_{v\in\mathcal{V}} i_v, \quad (2)$$

that is, the average probability for a randomly selected node to attain any of the three states.

In the thermodynamic limit of large populations, $n \to \infty$, the temporal evolution of the stochastic SAIS process at the population level can be approximated by the macroscopic variables in Eq. (2) for any finite time-horizon within an arbitrary precision[1] [3], [32], [33]. This is due to the absence of a fixed connectivity pattern in the ADN mechanisms, so that individuals interact in an anonymous fashion [14]. Thus, the epidemic *prevalence* $I(t)$ can be approximated as

$$I(t) := \frac{1}{n}\left|\{v \in \mathcal{V} : X_v = I\}\right| \approx y_i,$$

[1]For finite populations and long time-horizons, the behavior of stochastic epidemic models and their deterministic relaxations may show large deviations, since stochastic models always reach a disease-free state in a time that may grow exponentially in the population size [31].



(a) $n = 1,000$ (b) $n = 10,000$

Fig. 2. Simulations of the stochastic SAIS model (solid curves) and its deterministic approximation (dashed curves) at the population level for increasing population sizes. Activities a_is are uniformly distributed in $[0, 1]$; other parameters are fixed as $\mu = 1/7$, $\lambda = 0.43$, $\nu = 0.5$, $u_a = 0.4$, and $u_c = 0.3$.

and, similarly, the fraction of susceptible $S(t) \approx y_s$ and self-protective individuals $P(t) \approx y_p$. Hence, for sufficiently large populations and limited time-horizons, we can study the behavior of the epidemics at the population level through Eq. (1), as showed in Fig. 2. In view of these considerations, we focus on the analysis of the epidemic process for large populations by studying Eq. (1).

A first, key question we aim to elucidate is whether a small or local outbreak of the infectious disease will quickly extinguish or whether it will spread, yielding to a pandemic. From a mathematical point of view, answering such a question in the thermodynamic limit corresponds to finding under which conditions the disease-free equilibrium is a (local) asymptotically stable equilibrium of Eq. (1). Such conditions establish the so-called *epidemic threshold* of the model [1], which is characterized in the following result.

Theorem 1: In the thermodynamic limit of $n \to \infty$, the epidemic threshold for the SAIS model in Eq. (1) is equal to

$$\sigma := \frac{2(1+u_a)}{(2-u_c)\alpha_1 + \sqrt{u_c^2\alpha_1^2 + 4(1-u_c)\alpha_2}}. \tag{3}$$

If $\lambda/\mu < \sigma$, the disease-free state $y_i = 0$ is locally asymptotically stable.

Proof: From Eq. (1), we observe that

$$(s_v, p_v, i_v) = \left(\frac{1}{1+u_a}, \frac{u_a}{1+u_a}, 0\right), \quad \forall v \in \mathcal{V}, \tag{4}$$

is the disease-free equilibrium, that is, the unique equilibrium point of Eq. (1) with $y_i = 0$, and it is globally asymptotically stable on the disease-free manifold $i_v = 0$, for all $i \in \mathcal{V}$. To study its local stability, we follow a technique similar to the one proposed in [14]. We introduce two ancillary variables

$$z_1 := y_s - \frac{1}{1+u_a}, \quad \text{and} \quad z_2 = \frac{1}{n}\sum_{v\in\mathcal{V}} a_v i_v,$$

that is, the difference between the average probability that an individual is susceptible and the corresponding quantity in the disease-free equilibrium, and the average activity of infected individuals, respectively. We conclude that the disease-free equilibrium is (locally) asymptotically stable if and only if the origin is (locally) asymptotically stable for the system of ODEs composed by z_1, y_i, and z_2. From Eq. (1), we derive the system of ODEs for these three variables and we linearize them about the disease-free equilibrium, obtaining

$$\begin{aligned}
\dot{z}_1 &= -\nu(1+u_a)z_1 - \left(\frac{\lambda\alpha_1}{1+u_a} + \nu\right)y_i - \frac{\lambda(1-u_c)}{1+u_a}z_2,\\
\dot{y}_i &= \left(\frac{\lambda\alpha_1}{1+u_a} - \mu\right)y_i + \frac{\lambda(1-u_c)}{1+u_a}z_2,\\
\dot{z}_2 &= \frac{\lambda\alpha_2}{1+u_a}y_i + \left(\frac{\lambda(1-u_c)\alpha_1}{1+u_a} - \mu\right)z_2.
\end{aligned}$$

The Jacobian matrix of the system,

$$\begin{bmatrix}
-\nu(1+u_a) & -\frac{\lambda\alpha_1}{1+u_a} - \nu & -\frac{\lambda(1-u_c)}{1+u_a}\\
0 & -\mu + \frac{\lambda\alpha_1}{1+u_a} & \frac{\lambda(1-u_c)}{1+u_a}\\
0 & \frac{\lambda\alpha_2}{1+u_a} & -\mu + \frac{\lambda\alpha_1(1-u_c)}{1+u_a}
\end{bmatrix},$$

has eigenvalues equal to $-\nu(1+u_a) < 0$ and to

$$-\mu + \frac{\alpha_1(2-u_a)}{2(1+u_a)}\lambda \pm \frac{\lambda}{1+u_a}\sqrt{\frac{u_c^2}{4}\alpha_1^2 + (1-u_c)\alpha_2}.$$

The largest eigenvalue is negative if and only if $\lambda/\mu < \sigma$, from Eq. (3), which yields the claim. Note that the ancillary variable z_2 is necessary to explicitly study the ODE that governs y_i, while z_1 shifts the equilibrium point of y_s to the origin, simplifying the analytical computations. ■

Remark 2: For $u_a = u_c = 0$, Eq. (3) reduces to the threshold for the standard SIS model on ADNs [12], [13]; and for $u_a = 0$ it reduces to the threshold of the SIS model in the presence of behavioral changes due to infection [14], [30]. In general, the epidemic threshold is monotonically increasing in u_a and u_c, as shown in Fig. 3(a). From the epidemic threshold, one can derive the basic reproduction number as $\mathcal{R}_0 = \lambda/\mu\sigma$.

In the limit case of homogeneous ADNs (that is, $a_v = \alpha_1$, $\forall v \in \mathcal{V}$), a complete analysis of the thermodynamic limit $n \to \infty$ of the epidemic process can be performed. Note that there might be realistic scenarios (for instance, during a lockdown) in which the population's heterogeneity could be strongly reduced and the population treated as homogeneous.

Theorem 2: Consider the SAIS model in Eq. (1) in the thermodynamic limit of $n \to \infty$ on a homogeneous ADN with $a_v = \alpha_1$, for all $v \in \mathcal{V}$. If

$$\frac{\lambda}{\mu} \le \sigma_h := \frac{1+u_a}{\alpha_1(2-u_c)}, \tag{5}$$

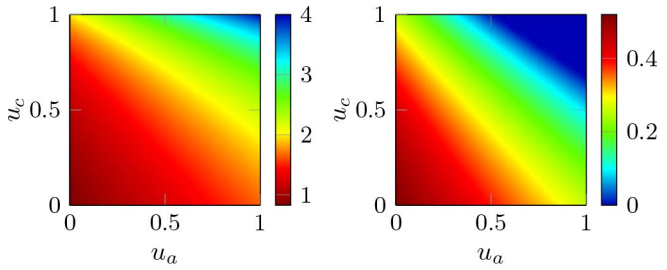
then the system in Eq. (1) converges to the disease-free equilibrium in Eq. (4). If $\lambda/\mu > \sigma_h$ and $y_i(0) > 0$, the system in Eq. (1) converges to

$$\bar{y}_s = \frac{\mu}{\lambda\alpha_1(2-u_c)}, \quad \bar{y}_i = \frac{\nu}{\mu+\nu} - \frac{\mu\nu(1+u_a)}{\lambda\alpha_1(2-u_c)(\mu+\nu)}, \tag{6}$$

and $\bar{y}_p = 1 - \bar{y}_s - \bar{y}_i$.

Proof: Let us consider the macroscopic variables defined in Eq. (2). From Eq. (1), we derive the system of ODEs that govern their evolution which, in this case, reduces to

$$\begin{aligned}
\dot{y}_s &= -u_a\nu y_s + \nu y_p - \lambda\alpha_1(2-u_c)y_s y_i,\\
\dot{y}_p &= u_a\nu y_s - \nu y_p + \mu y_i,\\
\dot{y}_i &= -\mu y_i + \lambda\alpha_1(2-u_c)y_s y_i,
\end{aligned} \tag{7}$$



(a) Threshold (b) Prevalence

Fig. 3. (a) Epidemic threshold and (b) endemic prevalence for different values of u_a and u_c, from Theorems 1 and 2, respectively. In (a), activities a_is are uniformly distributed in [0, 1] and $\nu = 0.5$; in (b) we fix $\alpha_1 = 0.5$, $\nu = 0.5$, $\lambda = 0.43$, and $\mu = 1/7$.

where one equation is a linear combination of the other two, since $y_s+y_p+y_i = 1$. We analyze the planar system comprising the first and the last equations in Eq. (7). We observe that the epidemic threshold σ from Theorem 1 reduces to the expression in Eq. (5). Introducing the function $\phi(y_s, y_i) = (y_s y_i)^{-1}$, we observe that

$$\frac{\partial(\phi \dot{y}_s)}{\partial y_s} + \frac{\partial(\phi \dot{y}_i)}{\partial y_i} = -\frac{\nu(1-y_i)}{y_s^2 y_i} < 0,$$

almost everywhere in the planar domain. Bendixson-Dulac criterion is used to exclude the existence of limit cycles [34]. Since the system is planar and the domain bounded (Lemma 1), Eq. (7) necessarily converges to an equilibrium point. From a direct computation, we find that, for $\lambda/\mu < \sigma_h$, the disease-free equilibrium $(1/(1+u_a), u_a/(1+u_a), 0)$ is the unique equilibrium point of Eq. (7) and, thus, it is globally asymptotically stable. If $\lambda/\mu > \sigma_h$, the disease-free is still an equilibrium point, but it becomes unstable (that is, asymptotically stable only on the disease-free manifold $y_i = 0$), while a second equilibrium point is found in correspondence to the coordinates in Eq. (6). Its asymptotic stability is a straightforward consequence of the instability of the disease-free equilibrium, together with the absence of other equilibrium points and of closed trajectories. Finally, convergence to the disease-free equilibrium for $\lambda/\mu = \sigma_h$ is established by observing that Eq. (6) coincides with the disease-free equilibrium in Eq. (4). ■

Remark 3: Unsurprisingly, from Eq. (6), we observe that the endemic prevalence is monotonically decreasing with respect to the two control actions u_a and u_c, as shown in Fig. 3(b).

IV. Analysis of Control Actions

The theoretical results in Section III can be leveraged to elucidate the effect of different control actions that can be taken to contrast an epidemic outbreak. First, we consider an epidemic outbreak in its early stages. In this scenario, we aim to determine which control action should be prioritized and, in general, what is the optimal strategy to maximize the epidemic threshold, thus hindering the diffusion of the infectious disease. Second, we consider a disease that has already reached its endemic state. Assuming a homogeneous population, our theoretical results are used to understand how a control policy should be designed to mitigate the epidemic prevalence, in the endemic state.

A. Early Stages of an Epidemic Outbreak

From the analytical expression of the epidemic threshold in Eq. (3), one could infer which control action may be more effective to contrast the inception of an epidemic outbreak. Toward this aim, we examine the sensitivity of Eq. (3) with respect to the two control parameters, thereby obtaining

$$\frac{\partial \sigma}{\partial u_a} = \frac{2}{(2-u_c)\alpha_1 + \sqrt{u_c^2\alpha_1^2 + 4(1-u_c)\alpha_2}},$$
$$\frac{\partial \sigma}{\partial u_c} = \frac{2(1+u_a)\left(\alpha_1 + \frac{2\alpha_2 - u_c\alpha_2^2}{\sqrt{u_c^2\alpha_1^2+4(1-u_c)\alpha_2}}\right)}{\left((2-u_c)\alpha_1 + \sqrt{u_c^2\alpha_1^2 + 4(1-u_c)\alpha_2}\right)^2}. \quad (8)$$

By comparing the two quantities in Eq. (8), we observe that $\frac{\partial \sigma}{\partial u_a}(0,0) > \frac{\partial \sigma}{\partial u_c}(0,0)$. Hence, we conclude that, in the very early stages of the epidemic outbreak, public health administrations may found more effective to prioritize the promotion of self-protective behaviors, rather than actions to confine all the infected individuals.

B. Optimal Control Policies

The explicit expression for the epidemic threshold can be further used to devise an optimal control policy in which a total budget must be used to fund a mix of the two control actions. Such a decision problem can be formalized through the following optimization problem:

Problem 1 (Minimize Outbreak Risk):

$$\begin{aligned} &\text{minimize} \quad f_1(u_a, u_c) = -\sigma(u_a, u_c), \\ &\text{subject to} \quad g(u_a, u_c) \le B, \\ &\qquad u_a \in \mathbb{R}_{\ge 0}, \quad u_c \in [0,1], \end{aligned}$$

where the function $\sigma(u_a, u_c)$ is defined in Eq. (3), $g(u_a, u_c)$ is the cost function for exerting control actions u_a and u_c, and $B \in \mathbb{R}_{\ge 0}$ is the total budget.

We observe that the objective function is concave, which hinders the use of standard optimization tools. However, since the objective function is monotonically decreasing with respect to both variables, its minimum is necessarily attained for values of u_a and u_c that satisfy the equality constraints $g(u_a, u_c) = B$. Thus, Problem 1 can be reduced to a one-dimensional problem by writing the constraint with respect to one of the variables and solving with respect to the other (analytically or numerically).

C. Minimizing Endemic Prevalence

For homogeneous populations, Theorem 2 provides an explicit computation of the epidemic prevalence of endemic diseases. Similar to Problem 1, we can formulate an optimization problem to minimize the endemic prevalence, given a total budget that should be divided between the two control actions, as follows:

Problem 2 (Minimize Prevalence):

$$\begin{aligned} &\text{minimize} \quad f(u_a, u_c) = \bar{y}_i(u_a, u_c), \\ &\text{subject to} \quad g(u_a, u_c) \le B, \end{aligned}$$

$$u_a \in \mathbb{R}_{\geq 0}, \quad u_c \in [0, 1],$$

where the function $\bar{y}_i(u_a, u_c)$ is defined in Eq. (6), $g(u_a, u_c)$ is the cost function for exerting control actions u_a and u_c, and $B \in \mathbb{R}_{\geq 0}$ is the total budget.

Similar to Problem 1, the objective function is concave, but the solution is attained when the equality constraint is verified, allowing to reduce Problem 2 to a one-dimensional minimization. We propose the following example to clarify this approach.

Example 1: Let us assume a linear control cost function $u_a + c_c u_c \leq B$, for some $c_c \in \mathbb{R}_{\geq 0}$. We set $u_a = B - c_c u_c$ and analytically solve the one-dimensional minimization problem obtained, determining the optimal solution

$$u_c^* = \begin{cases} \max\{1, B/c_c\} & \text{if } c_c < (1+B)/2, \\ 0 & \text{if } c_c \geq (1+B)/2, \end{cases}$$

and $u_a^* = B - c_c u_c^*$. This result suggests that, if the cost for implementing containment policies is sufficiently small with respect to the budget, containment policies should always be implemented.

V. Conclusion

In this letter, we have proposed an epidemic model on activity driven networks that encapsulates two different control actions, typically adopted by public health authorities: enforcing individuals to take self-protective behaviors and containment techniques to reduce the social interactions generated by infected individuals. Through a mean-field analysis of the model in the thermodynamic limit of large populations, we have derived the epidemic threshold of the model and, for homogeneous populations, we have computed the epidemic prevalence in the endemic state. Our theoretical results shed light on the role of the two control actions in shaping the evolution of the epidemic process, allowing for the formalization of optimization problems that can assist in the design of control policies to contrast the epidemic outbreak in its early stages or to minimize the prevalence of an endemic disease.

Beside extending the analysis of the two optimization problems formulated in this letter, we plan to include further features of real-world epidemics, such as mobility and more complex human behaviors, toward mathematical modeling of realistic epidemic outbreaks.

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