Annual Reviews in Control 44 (2017) 116–128

Contents lists available at ScienceDirect

Annual Reviews in Control

journal homepage: www.elsevier.com/locate/arcontrol

Review article

On the dynamics of deterministic epidemic propagation over networks☆

Wenjun Mei[a,*], Shadi Mohagheghi[b], Sandro Zampieri[c], Francesco Bullo[a]

[a] Mechanical Engineering and Center for Control, Dynamical-Systems and Computation, University of California at Santa Barbara, USA
[b] Department of Electrical and Computer Engineering, University of California at Santa Barbara, USA
[c] Department of Information Engineering, University of Padova, Italy

ARTICLE INFO

Article history:
Received 6 May 2017
Revised 9 September 2017
Accepted 17 September 2017
Available online 28 September 2017

Keywords:
Network propagation model
Nonlinear dynamical system
Phase transition
Mathematical epidemiology

ABSTRACT

In this work we review a class of deterministic nonlinear models for the propagation of infectious diseases over contact networks with strongly-connected topologies. We consider network models for Susceptible-Infected (SI), Susceptible-Infected-Susceptible (SIS), and Susceptible-Infected-Recovered (SIR) settings. In each setting, we provide a comprehensive nonlinear analysis of equilibria, stability properties, convergence, monotonicity, positivity, and threshold conditions. For the network SI setting, specific contributions include establishing its equilibria, stability, and positivity properties. For the network SIS setting, we review a well-known deterministic model, provide novel results on the computation and characterization of the endemic state (when the system is above the epidemic threshold), and present alternative proofs for some of its properties. Finally, for the network SIR setting, we propose novel results for transient behavior, threshold conditions, stability properties, and asymptotic convergence. These results are analogous to those well-known for the scalar case. In addition, we provide a novel iterative algorithm to compute the asymptotic state of the network SIR system.

Contents

☆ This material is based upon work supported by, or in part by, the U.S. Army Research Laboratory and the U.S. Army Research Office under grant no. W911NF-15-1-0577.
* Corresponding author.
E-mail addresses: wenjun_mei@umail.ucsb.edu (W. Mei), shadi.mohagheghie@gmail.com (S. Mohagheghi), zampi@dei.unipd.it (S. Zampieri), bullo@engineering.ucsb.edu (F. Bullo).

https://doi.org/10.1016/j.arcontrol.2017.09.002
1367-5788/

1. Introduction

1.1. Problem motivation and description

Propagation phenomena appear in numerous disciplines. Examples include the spread of infectious diseases in contact networks, the transmission of information in communication networks, the diffusion of innovations in competitive economic networks, cascading failures in power grids, and the spreading of wild-fires in forests.

One important class of models of propagation phenomena are scalar deterministic models. These models have been widely studied, e.g., see the survey by Hethcote (2000). These models qualitatively capture some dynamic features, including phase transitions and asymptotic states. However, shortcomings of scalar models are also prominent: for example, scalar models are typically based on the assumption that individuals in the population have the same chances of interacting with each other. This assumption overlooks the internal structure of the network over which the propagation occurs, as well as the heterogeneity of individuals in the network. Both these aspects play critical roles in shaping the dynamical behavior of the propagation processes.

In a general formulation, propagation is a dynamical process on a complex network. Each network node has a state taking value in a discrete set and state changes are influenced by the nodes' neighbors in the network. Many relevant research questions arise naturally, including: how to model the local dynamics at each node, how to identify model parameters, how to estimate the state of such a dynamical system, and how to analyze the system transient and asymptotic properties.

Various types of models have been proposed to describe propagation processes over complex networks; one key distinguishing feature of these models is whether the propagation dynamics is assumed to be stochastic or deterministic. Deterministic network epidemic models were originally proposed in the late1970s in the seminal works (Hethcote, 1978; Lajmanovich & Yorke, 1976). These models are of great research value, as attested by the large literature focusing on them (see below). Moreover, they can be considered as approximations of certain Markov-chain models, e.g., see Sahneh, Scoglio, and Mieghem (2013).

In this paper, we review three key continuous-time deterministic models for epidemic propagation over networks. Depending upon the nodal dynamics, i.e., the disease propagation behavior, deterministic epidemic propagation models are classified as: the *Susceptible-Infected* (SI) model, the *Susceptible-Infected-Susceptible* (SIS) model and the *Susceptible-Infected-Recovered* (SIR) model; basic representations of these models are illustrated in Fig. 1. In this work we focus on transient and asymptotic behavior of these three continuous-time dynamical models over networks. It is our key objective to relate the structure of the network to the function of the network (i.e., the transient and asymptotic behavior of the propagation phenomenon).

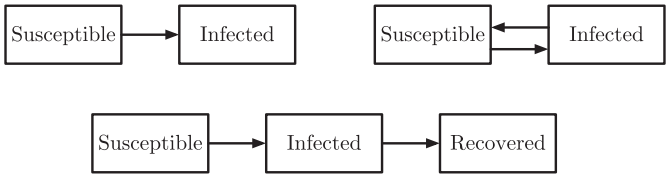

Fig. 1. Three basic models of infectious diseases: SI, SIS and SIR.

1.2. Literature review on deterministic epidemic models over networks

The literature on epidemic propagation is exceedingly vast. This paper focuses on deterministic models over networks and on their dynamical behavior. Accordingly, this subsection reviews the literature on deterministic epidemic models. Unless specified, the works and results reviewed in what follows are all for the deterministic models. For readers interested in Markov-chain models and in the mean-field approximation method, we refer to Nowzari, Preciado, and Pappas (2016); Pastor-Satorras, Castellano, VanMieghem, and Vespignani (2015); Ruhi and Hassibi (2015); Sahneh et al. (2013), Bullo (2017, Chapter 17). (Note that Markov-chain network epidemic models and their deterministic approximating models are different in some of the dynamical properties, such as the epidemic threshold and the asymptotic behavior.)

The dynamics of several classic scalar epidemic models, i.e., the population models without network structure, are surveyed in detail by Hethcote (2000). Among the different metrics discussed, identifying the *effective reproduction number* R is of particular interest to researchers; R is the expected number of individuals that a randomly infected individual can infect during its infection period. In these scalar models, whether an epidemic outbreak occurs or the disease dies down depends upon whether $R > 1$ or $R < 1$, i.e., upon whether the system is above or below the so-called *epidemic threshold*. Here by epidemic outbreak we mean an exponential growth of the fraction of the infected population for small time. The *basic reproduction number* R_0 is the effective reproduction number in a fully-healthy susceptible population. In what follows we focus our review on deterministic network models.

The earliest work on the (continuous-time heterogeneous) SIS model on networks is Lajmanovich and Yorke (1976). This work proposes an n-dimensional model on a contact network and analyzes the system's asymptotic behavior. This article proposes a rigorous analysis of the threshold for the epidemic outbreak, which depends on both the disease parameters and the spectral radius of the contact network. For the case when the basic reproduction number is above the epidemic threshold, this paper establishes the existence and uniqueness of a nonzero steady-state infection probability, called the endemic state. In what follows we refer to the model by Lajmanovich and Yorke (1976) as the *network SIS model*; it is also known as the multi-group or multi-population SIS model.

Allen (1994) proposes and analyzes a discrete-time network SIS model. This work appears to be the first to revisit and formally reproduce, for the discrete-time case, the earlier results by Lajmanovich and Yorke (1976); see also the later work by Wang, Chakrabarti, Wang, and Faloutsos (2003). This work confirms the existence of an epidemic threshold, as a function of the spectral radius of the contact network. Further recent results on the discrete-time model are obtained by Ahn and Hassibi (2013) and by Ruhi and Hassibi (2015).

Mieghem, Omic, and Kooij (2009) argue that the (continuous-time) network SIS model is in fact the mean-field approximation of the original Markov-chain SIS model of exponential dimension; this claim is rigorously proven by Sahneh et al. (2013). Mieghem et al. (2009) refer to this model as the intertwined SIS model and write the endemic state as a continued fraction.

The works of Fall, Iggidr, Sallet, and Tewa (2007) and Khanafer, Başar, and Gharesifard (2016) discuss the continuous-time network SIS model in a more modern language. Fall et al. (2007) refer to this model as the n-group SIS model and

apply Lyapunov techniques and Metzler matrix theory to establish existence, uniqueness, and stability of the equilibrium points below and above the epidemic threshold. Khanafer et al. (2016) use positive system theory in their analysis and extend the existence, uniqueness, and stability results to the setting of weakly connected digraphs.

Numerous extensions of these basic results on the network SIS model and other related works have appeared over the years. For example, the estimation of the epidemic threshold in contact networks with power-law degree distributions has been studied both by mathematically rigorous analysis, see Chung, Lu, and Vu (2003), and by numerical simulation, see Castellano and Pastor-Satorras (2010). The deterministic network SIS models without mean-field approximation and with second-order mean-field approximation have been analyzed by Schwartz and Stone (2013) and Ogura and Preciado (2017), respectively.

An early work by Hethcote (1978) proposes a general multi-group SIR model with birth, death, immunization, and de-immunization. The epidemic threshold and the equilibria below/above the threshold are characterized. For the simplified model without birth/death and de-immunization, Hethcote (1978) proves that the system converges asymptotically to an all-healthy state. Guo, Li, and Shuai (2008) consider a generalized network SIR model with vital dynamics, that is, with birth and death. They characterize the basic reproduction number and, through a careful Lyapunov analysis, show the existence and global asymptotic stability of an endemic state above the threshold. Youssef and Scoglio (2011) study a special case of the network SIR model under the name of individual-based SIR model over undirected networks. Through a simulation-based analysis, the epidemic threshold is given as a function of the spectral radius of the network.

There are also some extensions and related studies regarding the network SIR model. Sharkey (2008) investigates the deterministic network SIR model without mean-field approximation. Castellano and Pastor-Satorras (2010) point out that the (mean-field) network SIR predicts a vanishing threshold for a certain class of power-law distributed networks, which is inconsistent with the corresponding stochastic SIR model. Sharkey, Kiss, Wilkinson, and Simon (2015) show that, different from the network SIR model with mean-field approximation, the so-called pair-based approach gives an exact description of the stochastic SIR process for the tree topology.

To the best of our knowledge, no works have comprehensively characterized the properties of the network SI model.

We conclude by mentioning other surveys and textbook treatments. In Mesbahi and Egerstedt (2010), the stability of equilibria for the SEIR model is reviewed through Lyapunov and graph theory. The additional state E represents the exposed population, i.e., the individuals who are infected but not infectious. Newman (2010, Chapter 17), Easley and Kleinberg (2010, Chapter 21), and Barrat, Barthlemy, and Vespignani (2008, Chapter 9) review various heterogeneous epidemic models. The recent survey by Nowzari et al. (2016) presents various epidemic models and addresses many solved and open problems in the control of epidemic spreading.

1.3. Statement of contribution

This paper reviews, in a comprehensive and coherent manner, deterministic models and dynamical behavior of SI, SIS and SIR epidemic phenomena over networks. This review includes known results from the literature as well as several novel results. We discuss SI, SIS and SIR models in three subsequent corresponding sections. Each section starts by reviewing the well-known results for the corresponding scalar models; these are the models in which variables represent an entire well-mixed population or nodes of an all-to-all unweighted graph. The core of each section is a discussion about multi-group network models. We provide a tutorial treatment with comprehensive statements and proofs for the deterministic network SI, SIS and SIR models.

We first analyze the network SI model. We analyze its asymptotic convergence, positivity of infection probabilities, initial and asymptotic growth rates, and the stability of equilibria. We show that in the network SI model, the system does not display a threshold and, with the exception of the trivial no-epidemics equilibrium, all the trajectories converge to the full contagion state. While these results are not technically difficult, they are novel here in the sense that, to the best of our knowledge, the properties of the network SI model have never before been formally characterized.

Next we focus on the network SIS model. Our presentation includes known results from Lajmanovich and Yorke (1976) (see also Fall et al., 2007; Khanafer et al., 2016) regarding the epidemic threshold, the system's behavior below the epidemic threshold, the existence and uniqueness of the endemic state for systems above the epidemic threshold, and the asymptotic stability of the endemic state. Moreover, we provide a novel provably-correct iterative algorithm for computing the fraction of infected individuals converging to the endemic state. This algorithm also provides an alternative proof for the existence and uniqueness of the endemic state for systems above the epidemic threshold. We argue that this alternative proof is more concise that the those proposed in the previous works of Fall et al. (2007), Lajmanovich and Yorke (1976), Khanafer et al. (2016). In addition, we present novel Taylor expansions for the endemic state near the epidemic threshold and in the limit of high infection rates. These novel Taylor expansions shed light on these previously poorly-understood regimes. Finally, we show that the spread of infection takes place instantaneously upon infecting at least one node in the network.

Finally, for the network SIR model, we review some known results on the monotonicity of the individuals' susceptible probabilities and the system's asymptotic behavior from (Hethcote, 1978). More importantly, we provide the several novel results: We present novel transient behavior and system properties. First, we propose new threshold conditions above which the epidemic grows initially, and below which it exponentially dies down. The initial rate of growth above the threshold is given in terms of network characteristics, initial conditions, and infection parameters. Moreover, we show that our proposed weighted average of the infected population, obtained by the entries of dominant eigenvector of an irreducible quasi-positive matrix, captures information regarding the distribution of infection in the system. We also establish positivity of the infection probabilities. Finally, we provide a novel iterative algorithm to compute the asymptotic state of the network SIR model, with any arbitrary initial condition. For the iterative algorithm, the existence and uniqueness of the fixed point, and the convergence of the iteration are rigorously proved. Our results are analogous to the scalar SIR model properties and are valid for any arbitrary network topologies. In comparison with (Youssef & Scoglio, 2011), our treatment builds on their numerical results but our result is more general in that it does not depend upon specific initial conditions and graph topologies, and establishes numerous properties, including the novel characterization of epidemic threshold.

1.4. Organization

Section 2introduces our model set-up and some preliminary notations. The SI, SIS and SIR models are presented, respectively, in Sections 3, 4, and 5. Section 6 is the conclusion.

2. Model set-up and notations

For the scalar models, we use the notation $x(t)$ ($s(t)$ and $r(t)$ resp.) for the fraction of infected (susceptible and recovered resp.) individuals in the population at time t. The rest of this section is about the notations and basic model set-up for the network epidemic model.

(a) *Contact network:* The epidemics are assumed to propagate over a weighted digraph $G = (V, E)$, where $V = \{1, \dots, n\}$ and E is the set of directed links. Nodes of G can be interpreted as either single individuals in the contact network or as homogeneous populations of individuals at each location/node in the contact network. $A = (a_{ij})_{n\times n}$ denotes the adjacency matrix associated with G. For any $i, j \in V$, a_{ij} characterizes the contact strength from node j to node i. For $(i, j) \in E$, $a_{ij} > 0$ and for $(i, j) \notin E$, $a_{ij} = 0$. In this paper, G is assumed to be strongly connected.

(b) *Node states and probabilities:* For different epidemic propagation models, the set of possible node states are distinct. For network SI or SIS models, each node can be in either the "susceptible" or "infected" state, while in the network SIR model, there is an additional possible node state: "recovered." For a graph in which the nodes are single individuals, let $s_i(t)$ ($x_i(t)$ and $r_i(t)$ resp.) be the probability that individual i is in the susceptible (infected and recovered resp.) state at time t. Alternatively, if the nodes are considered to be the populations, then $s_i(t)$ ($x_i(t)$ and $r_i(t)$ resp.) is interpreted as the fraction of susceptible (infected and recovered resp.) individuals in population i. In this paper, without loss of generality, we adopt the interpretation of nodes as single individuals.

(c) *Frequently used notations:* The symbol $\mathbb{R}$ denotes the set of real numbers, while $\mathbb{R}_{\geq 0}$ denotes the set of non-negative real numbers. The symbol ϕ denotes the empty set. For any two vectors $x, y \in \mathbb{R}^n$, we write

$$\begin{aligned} x \ll y, &\quad \text{if } x_i < y_i \text{ for all } i \in \{1, \dots, n\},\\ x \leq y, &\quad \text{if } x_i \leq y_i \text{ for all } i \in \{1, \dots, n\}, \text{ and}\\ x < y, &\quad \text{if } x \leq y \text{ and } x \neq y. \end{aligned}$$

We adopt the shorthand notations $\mathbb{1}_n = [1, \dots, 1]^\top$ and $\mathbb{0}_n = [0, \dots, 0]^\top$. Let I_n denote the $n \times n$ identity matrix. Given $x = [x_1, \dots, x_n]^\top \in \mathbb{R}^n$, let $\mathrm{diag}(x)$ denote the diagonal matrix whose diagonal entries are $x_1, \dots, x_n$. For an irreducible nonnegative matrix A, let $\lambda_{\max}(A)$ denote the dominant eigenvalue of A that is equal to the spectral radius $\rho(A)$. Moreover, we let $v_{\max}(A)$ ($u_{\max}(A)$ resp.) denote the corresponding entry-wise strictly positive left (right resp.) eigenvector associated with $\lambda_{\max}(A)$, normalized to satisfy $\mathbb{1}_n^\top v_{\max}(A) = 1$ (resp. $\mathbb{1}_n^\top u_{\max}(A) = 1$). The Perron–Frobenius theorem for irreducible matrices guarantees that $\lambda_{\max}(A)$, $v_{\max}(A)$ and $u_{\max}$ are well defined and unique. Where not ambiguous, we will drop the (A) argument and, for example, write

$$v_{\max}^\top A = \lambda_{\max} v_{\max}^\top \quad \text{and} \quad A u_{\max} = \lambda_{\max} u_{\max},$$

with $v_{\max} \gg \mathbb{0}_n$ and $\mathbb{1}_n^\top v_{\max} = 1$; $u_{\max} \gg \mathbb{0}_n$ and $\mathbb{1}_n^\top u_{\max} = 1$.

3. Susceptible-Infected model

In this section, we first review the classic scalar susceptible-infected (SI) model, and then present and characterize the network SI model.

3.1. Scalar SI model

The scalar SI model assumes that the growth rate of the fraction of the infected individuals is proportional to the fraction of the susceptible individuals, multiplied by a so-called *infection rate* $\beta > 0$. The model is given by

$$\dot{x}(t) = \beta s(t) x(t) = \beta\big(1 - x(t)\big)x(t). \tag{1}$$

This is the well-established logistic equation. The following results can be found for example in the textbook of Brauer and Castillo-Chavez (2012).

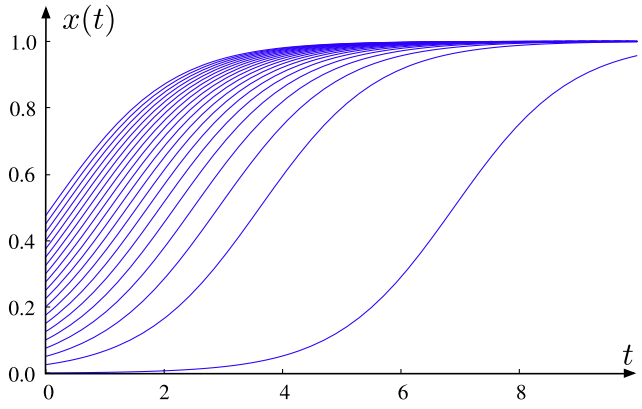

Fig. 2. Evolution of the (lumped deterministic) SI model ($\beta = 1$) from small initial fraction of infected individuals.

Lemma 3.1 (Dynamical behavior of the SI model)**.** *Consider the scalar SI model (1) with $\beta > 0$. The solution from initial condition $x(0) = x_0 \in [0, 1]$ is*

$$x(t) = \frac{x_0 e^{\beta t}}{1 - x_0 + x_0 e^{\beta t}}. \tag{2}$$

All initial conditions $0 < x_0 < 1$ result in the solution $x(t)$ being monotonically increasing and converging to the unique equilibrium 1 as $t \to \infty$.

Solutions to Eq. (1) with different initial conditions are plotted in Fig. 2. The SI model (1) results in an evolution akin to a logistic curve, and is also called the logistic equation for population growth.

3.2. Network SI model

The network SI model on a weighted digraph with the adjacency matrix $A \in \mathbb{R}_{\geq 0}^{n\times n}$ is given by

$$\dot{x}_i(t) = \beta\big(1 - x_i(t)\big)\sum_{j=1}^{n} a_{ij} x_j(t), \tag{3}$$

or, in equivalent vector form,

$$\dot{x}(t) = \beta\Big(I_n - \mathrm{diag}\big(x(t)\big)\Big)Ax(t), \tag{4}$$

where $\beta > 0$ is the infection rate. Alternatively, in terms of the fractions of susceptibile individuals $s(t) = \mathbb{1}_n - x(t)$, the network SI model is

$$\dot{s}(t) = -\beta\,\mathrm{diag}(s(t))A(\mathbb{1}_n - s(t)). \tag{5}$$

The network SI model is a particular case of the widely-studied network SIS model, which is to be discussed in the next section. The dynamical properties of the network SI model are not difficult to analyze, but, to the best of our knowledge, have not been formally presented in any previous literature. We present the results on the transient and asymptotic behavior of the network SI model, as well as the proof, in the following theorem.

Theorem 3.2 (Dynamical behavior of network SI model)**.** *Consider the network SI model (4) with $\beta > 0$. For strongly connected graph with adjacency matrix A, the following statements hold:*

(i) *if* $x(0), s(0) \in [0, 1]^n$, *then* $x(t), s(t) \in [0, 1]^n$ *for all* $t > 0$. *Moreover,* $x(t)$ *is monotonically non-decreasing (here by monotonically non-decreasing we mean* $x(t_1) \leq x(t_2)$ *for all* $t_1 \leq t_2$*). Finally, if* $x(0) > \mathbb{0}_n$, *then* $x(t) \gg \mathbb{0}_n$ *for all* $t > 0$;

(ii) *the model (4) has two equilibrium points:* $\mathbb{0}_n$ *(no epidemic), and* $\mathbb{1}_n$ *(full contagion);*

  (a) *the linearization of model (4) about the equilibrium point* $\mathbb{0}_n$ *is* $\dot{x} = \beta A x$ *and it is exponentially unstable;*

  (b) *let* $D = \mathrm{diag}(A\mathbb{1}_n)$ *be the degree matrix. The linearization of model (5) about the equilibrium* $\mathbb{0}_n$ *is* $\dot{s} = -\beta D s$ *and it is exponentially stable;*

(iii)] *each trajectory with initial condition* $x(0) \neq \mathbb{0}_n$ *converges asymptotically to* $\mathbb{1}_n$, *that is, the epidemic spreads monotonically to the entire network.*

Proof.

(i) The fact that, if $x(0), s(0) \in [0, 1]^n$, then $x(t), s(t) \in [0, 1]^n$ for all $t > 0$ means that $[0, 1]^n$ is an invariant set for the differential equation (4). This is the consequence of Nagumo's theorem (see Blanchini and Miani, 2015, Theorem 4.7), since for any x belonging on the boundary of the set $[0, 1]^n$, the vector $\beta\Big(I_n - \mathrm{diag}\big(x\big)\Big)Ax$ is either tangent, or points inside the set $[0, 1]^n$.
Observe that the invariance of the set $[0, 1]^n$ implies that $\dot{x}(t) \geq \mathbb{0}_n$ and so $x(t_1) \leq x(t_2)$ for all $t_1 \leq t_2$.
We want to prove now that, if $x(0) > 0_n$, then $x(t) \gg 0_n$ for all $t > 0$. If by contradiction there is $i \in \{1, \ldots, n\}$ and $T > 0$ such that $x_i(T) = 0$, then the monotonicity of $x_i(t) = 0$ would imply that $x_i(t) = 0$ for all $t \in [0, T]$, which would yield $\dot{x}_i(t) = 0$ for all $t \in [0, T]$. By (3) this would imply that $x_j(t) = 0$ for all $t \in [0, T]$ for all j such that $a_{ij} > 0$. We could iterate this argument and using the irreducibility of A we would get the contradiction that $x(t) = 0$ for all $t \in [0, T]$ concluding in this way the proof of (i).

(ii) Regarding statement (ii), note that $\mathbb{0}_n$ and $\mathbb{1}_n$ are clearly equilibrium points. Let $\bar{x} \in [0, 1]^n$ be an equilibrium and assume that $\bar{x} \neq \mathbb{1}_n$. Then there is i such that $\bar{x}_i \neq 1$. Since $\beta\big(1 - \bar{x}_i\big)\sum_{j=1}^n a_{ij}\bar{x}_j = 0$, then $\sum_{j=1}^n a_{ij}\bar{x}_j = 0$ which implies that $\bar{x}_j = 0$ for all j such that $a_{ij} > 0$. By iterating this argument and using the irreducibility of A we get that $\bar{x} = 0$ concluding only $\mathbb{0}_n$ and $\mathbb{1}_n$ are equilibrium points. Statements (ii)a and(ii)b are obvious. Exponential stability of the linearization $\dot{s} = -\beta D s$ is obvious, and the Perron–Frobenius theorem implies the existence of the unstable positive eigenvalue $\rho(A) > 0$ for the linearization $\dot{x} = \beta A x$.

(iii) Consider the function $V(x) = \mathbb{1}_n^\top(\mathbb{1}_n - x)$; this is a smooth function defined over the compact and forward invariant set $[0, 1]^n$ (see statement (i), since $\dot{V} = -\beta\mathbb{1}_n^\top\big(I_n - \mathrm{diag}(x)\big)Ax$, we know that $\dot{V} \leq 0$ for all x and $\dot{V}(x) = 0$ if and only if $x \in \{\mathbb{0}_n, \mathbb{1}_n\}$. The LaSalle Invariance Principle implies that all trajectories with $x(0)$ converge asymptotically to either $\mathbb{1}_n$ or $\mathbb{0}_n$. Additionally, note that $0 \leq V(x) \leq n$ for all $x \in [0, 1]^n$, that $V(x) = 0$ if and only if $x = \mathbb{1}_n$ and that $V(x) = n$ if and only if $x = \mathbb{0}_n$. Therefore, all trajectories with $x(0) \neq \mathbb{0}_n$ converge asymptotically to $\mathbb{1}_n$.

□

In the next two paragraphs we present the "initial-time" ("final-time" resp.) approximation of the solution to the network SI model, i.e., the approximated solution to Eq. (4), or Eq. (5) equivalently, when t is sufficiently small (large resp.). These results are novel.

For the adjacency matrix A, there exists a non-singular matrix T such that $A = TJT^{-1}$, where J is the Jordan normal form of A. Since A is non-negative and irreducible, according to Perron–Frobenius theorem, the first Jordan block $J_1 = (\lambda_{\max})_{1\times 1}$ and $\lambda_{\max} > Re(\lambda_i)$ for any other eigenvalue λ_i of A. Consider now the onset of an epidemic in a large population characterized by a small initial infection $x(0) = x_0$ much smaller than $\mathbb{1}_n$. The system evolution is approximated by $\dot{x} = \beta A x$. This "initial-times" linear evolution satisfies

$$x(t) = e^{\beta A t}x(0) = Te^{\beta J t}T^{-1}x(0) = e^{\beta\lambda_{\max}t}\Big(T\mathbb{e}_1\mathbb{e}_1^\top T^{-1}x(0) + o(1)\Big),$$

where $\mathbb{e}_1$ is the first standard basis vector in $\mathbb{R}^n$ and $o(1)$ denotes a time-varying vector that vanishes as $t \to +\infty$. Let u_1 denote the first column of T and let $v_1^\top$ denote the first row of T^{-1}. Since $AT = TJ$ and $T^{-1}A = JT^{-1}$, one can check that u_1 (v_1 resp.) is the right (left resp.) eigenvector of A associated with the eigenvalue $\lambda_{\max}$. Since $T^{-1}T = I_n$, we have $v_1^\top u_1 = 1$. therefore,

$$\begin{aligned} x(t) &= e^{\beta\lambda_{\max}t}\Big(u_1 v_1^\top x(0) + o(1)\Big) \\ &= e^{\beta\lambda_{\max}t}\Big(\frac{v_{\max}^\top x(0)}{v_{\max}^\top u_{\max}}u_{\max} + o(1)\Big). \end{aligned} \tag{6}$$

That is, the epidemic initially experiences exponential growth with rate $\beta\lambda_{\max}$ and with distribution among the nodes given by the eigenvector $u_{\max}$.

Now suppose that at some time T, for all i we have that $x_i(T) = 1 - \epsilon_i$, where each ϵ_i is much smaller than 1. Then, for time $t > T$, the approximated system for $s(t)$ is given by:

$$\dot{s}_i(t) = -\beta d_i s_i(t) \quad \Rightarrow \quad s_i(t) = \epsilon_i e^{-\beta d_i(t-T)},$$

where, for any $i \in \{1, \ldots, n\}$, $d_i = \sum_{j=1}^n a_{ij}$ denotes the out-degree of node i in the network. From the discussion above, we conclude that the initial infection rate is proportional to the eigenvector centrality, and the final infection rate is proportional to the degree centrality.

4. Susceptible-Infected-Susceptible model

In this section we review the Susceptible-Infected-Susceptible (SIS) epidemic model. In addition to the existence of an infection process with rate $\beta > 0$, this model assumes that the infected individuals recover to the susceptible state at so-called *recovery rate*$\gamma > 0$.

4.1. Scalar SIS model

In the scalar SIS model, the population is divided into two fractions: the infected $x(t)$ and the susceptible $s(t)$, with $x(t) + s(t) = 1$, obeying the following dynamics:

$$\dot{x}(t) = \beta s(t)x(t) - \gamma x(t) = (\beta - \gamma - \beta x(t))x(t). \tag{7}$$

The dynamical behavior of system (7) given below can be found in Hethcote (2000).

Lemma 4.1 (Dynamical behavior of the SIS model)**.** *For the SIS model (7) with* $\beta > 0$ *and* $\gamma > 0$*:*

(i) *the closed-form solution to equation (7) from initial condition* $x(0) = x_0 \in [0, 1]$, *for* $\beta \neq \gamma$, *is*

$$x(t) = \frac{(\beta - \gamma)x_0}{\beta x_0 - e^{-(\beta-\gamma)t}(\gamma - \beta(1 - x_0))}; \tag{8}$$

(ii) *if* $\beta \leq \gamma$, *all trajectories converge to the unique equilibrium* $x = 0$ *(i.e., the epidemic disappears);*

(iii) *if* $\beta > \gamma$, *then each trajectory from an initial condition* $x(0) > 0$ *converges to the exponentially stable equilibrium* $x^* = (\beta - \gamma)/\beta$, *which is called the* endemic state.

Case (iii) corresponds to the case in which epidemic outbreaks take place and a steady-state epidemic contagion persists. The basic reproduction number in this deterministic scalar SIS model is given by $R_0 = \beta/\gamma$. Simulations regarding to Lemma 4.1(ii) and (iii) are shown in Fig. 3.

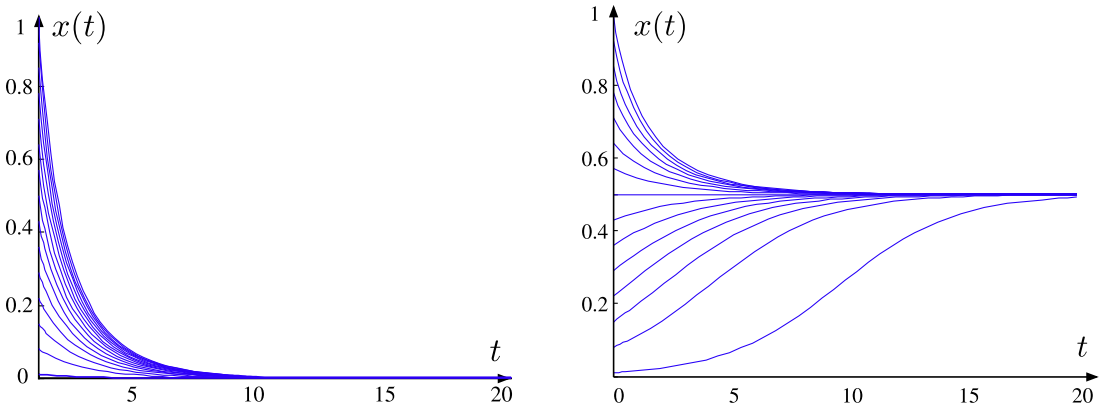

Fig. 3. Evolution of the scalar SIS model with varying initial fraction of infected individuals. Top figure: $\beta = 0.5 < \gamma = 1$. Bottom figure: $\beta = 0.8 > \gamma = .4$.

4.2. Network SIS model

In this section we study the network SIS model which is closely related to the original "multi-group SIS model" proposed by Lajmanovich and Yorke (1976); see also the intertwined SIS model in Mieghem et al. (2009).

The network SIS model with infection rate β and recovery rate γ is given by:

$$\dot{x}_i(t) = \beta(1 - x_i(t)) \sum_{j=1}^{n} a_{ij} x_j(t) - \gamma x_i(t), \tag{9}$$

or, in equivalent vector form,

$$\dot{x}(t) = \beta\big(I_n - \operatorname{diag}(x(t))\big)Ax(t) - \gamma x(t). \tag{10}$$

In the rest of this section we study the dynamical properties of this model. We start by defining the monotonically-increasing functions

$$f_+(y) = y/(1+y), \quad \text{and} \quad f_-(z) = z/(1-z),$$

for $y \in \mathbb{R}_{\geq 0}$ and $z \in [0, 1[$. Note that $f_+(f_-(z)) = z$ for all $z \in [0, 1)$. For vector variables $y \in \mathbb{R}^n_{\geq 0}$ and $z \in [0, 1)^n$, we write $F_+(y) = (f_+(y_1), \ldots, f_+(y_n))$, and $F_-(z) = (f_-(z_1), \ldots, f_-(z_n))$.

4.2.1. Behavior of system below the threshold

In this subsection, we characterize the behavior of the network SIS model in a regime we describe as "below the threshold." The results presented in the theorem below can be found in Fall et al. (2007), Lajmanovich and Yorke (1976), Khanafer et al. (2016). Historically, it is meaningful to attribute this theorem to Lajmanovich and Yorke (1976), even if the language adopted here is more modern.

Theorem 4.2 (Dynamical behavior of the network SIS model: Below the threshold)**.** *Consider the network SIS model (9), with $\beta > 0$ and $\gamma > 0$, over a strongly connected digraph with adjacency matrix A. Let $\lambda_{\max}$ and $v_{\max}$ be the dominant eigenvalue of A and the corresponding normalized left eigenvector respectively. If $\beta\lambda_{\max}/\gamma < 1$, then*

(i) *if $x(0), s(0) \in [0, 1]^n$, then $x(t), s(t) \in [0, 1]^n$ for all $t > 0$. Moreover, if $x(0) > \mathbb{0}_n$, then $x(t) \gg \mathbb{0}_n$ for all $t > 0$;*
(ii) *there exists a unique equilibrium point $\mathbb{0}_n$, the linearization of (9) about $\mathbb{0}_n$ is $\dot{x} = (\beta A - \gamma I_n)x$ and it is exponentially stable;*
(iii) *from any $x(0) \neq \mathbb{0}_n$, the weighted average $t \mapsto v_{\max}^\top x(t)$ is monotonically and exponentially decreasing, and all the trajectories converge to $\mathbb{0}_n$.*

Proof.

(i) As in Theorem 3.2 the first part is the consequence of Nagumo's theorem. Then define $y(t) := e^{\gamma t}x(t)$. Notice that this variable satisfies the differential equation $\dot{y}(t) = \beta \operatorname{diag}(s(t))Ay(t)$. From the same arguments used in the proof of the point (i) of Theorem 3.2 we argue that $y(t) \gg \mathbb{0}_n$ for all $t > 0$. From this it follows that also $x(t) \gg \mathbb{0}_n$ for all $t > 0$.

(ii) Assume that x^* is an equilibrium point. It is easy to see that $x^* \ll \mathbb{1}_n$. Let $\hat{A} = \beta A/\gamma$. Observe moreover that x^* is an equilibrium point if and only if $\hat{A}x^* = F_-(x^*)$ or, equivalently, if and only if $F_+\big(\hat{A}x^*\big) = x^*$. This means that x^* is an equilibrium if and only if it is a fixed point of $\mathcal{F}$, where $\mathcal{F}(x) := F_+\big(\hat{A}x\big)$. For $x \in [0, 1]^n$, note $F_+(\hat{A}x) \leq \hat{A}x$ because $f_+(z) \leq z$. Moreover, $\mathbb{0}_n \leq x \leq y$ implies that $\mathbb{0}_n \leq \mathcal{F}(x) \leq \hat{A}y$. Therefore, if $\mathbb{0}_n \leq x$, then $\mathcal{F}^k(x) \leq \hat{A}^k x$, for all k. Since $\hat{A}$ is Schur stable, then $\lim_{k\to\infty} \mathcal{F}^k(x) = 0$. This shows that the only fixed point of $\mathcal{F}$ is zero.
Next, the linearization of Eq. (10) is verified by dropping the second-order terms. The linearized system is exponentially stable at $\mathbb{0}_n$ for $\beta\lambda_{\max} - \gamma < 0$ because $\lambda_{\max}$ is larger, in real part, than any other eigenvalue of A by the Perron–Frobenius theorem for irreducible matrices.

(iii) Finally, regarding statement(iii), define $y(t) = v_{\max}^\top x(t)$ and note that $\big(I_n - \operatorname{diag}(z)\big)v_{\max} \leq v_{\max}$ for any $z \in [0, 1]^n$. Therefore,

$$\dot{y}(t) \leq \beta v_{\max}^\top Ax(t) - \gamma v_{\max}^\top x(t) = (\beta\lambda_{\max} - \gamma)y(t) < 0.$$

By the Grönwall-Bellman comparison lemma, $y(t)$ is monotonically decreasing and satisfies $y(t) \leq y(0)e^{(\beta\lambda_{\max}-\gamma)t}$ from all initial conditions $y(0)$. This concludes our proof of statement(iii).

□

4.2.2. Behavior of system above the threshold

We present the dynamical behavior of the network SIS model above the threshold as follows. Statement (i) of the theorem below is a straightforward result from Eq. (10). Historically, the existence of a unique endemic state and its global attractivity properties, i.e., statements (ii), (iii), (iii)a and (iv) in the theorem below, are due to Lajmanovich and Yorke (1976), and can be found in Fall et al. (2007), Khanafer et al. (2016). To the best our knowledge, the Taylor expansions in parts (iii)b and(iii)c and the algorithm in part (iii)d are novel. In addition, compared with the previous works (Fall et al., 2007; Khanafer et al., 2016; Lajmanovich & Yorke, 1976), construction of the algorithm in part (iii)d provides an alternative and more concise proof for the existence and uniqueness of the endemic state, and the convergence of any solution starting with $x(0) \in (0, 1)^n$ to this endemic state.

Theorem 4.3 (Dynamical behavior of the network SIS model: Above the threshold)**.** *Consider the network SIS model (9), with $\beta>0$ and $\gamma>0$, over a strongly connected digraph with adjacency matrix A. Let $\lambda_{\max}$ be the dominant eigenvalue of A and let $v_{\max}$ and $u_{\max}$ be the corresponding normalized left and right eigenvectors respectively. Let $d=A\mathbb{1}_n$. If $\beta\lambda_{\max}/\gamma>1$, then*

(i) *if $x(0), s(0)\in[0,1]^n$, then $x(t), s(t)\in[0,1]^n$ for all $t>0$. Moreover, if $x(0)>\mathbb{0}_n$, then $x(t)\gg\mathbb{0}_n$ for all $t>0$;*

(ii) *$\mathbb{0}_n$ is an equilibrium point, the linearization of system (10) at $\mathbb{0}_n$ is unstable due to the unstable eigenvalue $\beta\lambda_{\max}-\gamma$ (i.e., there will be an epidemic outbreak);*

(iii) *besides the equilibrium $\mathbb{0}_n$, there exists a unique equilibrium point x^*, called the* endemic state*, such that*

(a) $x^*\gg\mathbb{0}_n$,

(b) $x^*=\delta a u_{\max}+O(\delta^2)$ *as* $\delta\to0^+$, *where* $\delta:=\beta\lambda_{\max}/\gamma-1$ *and*

$$a=\frac{v_{\max}^T u_{\max}}{v_{\max}^T\operatorname{diag}(u_{\max})u_{\max}},$$

(c) $x^*=\mathbb{1}_n-(\gamma/\beta)\operatorname{diag}(d)^{-1}\mathbb{1}_n+O(\gamma^2/\beta^2)$, *at fixed A, as $\gamma/\beta\to0^+$, where $d=A\mathbb{1}_n$,*

(d) *define a sequence $\{y(k)\}_{k\in\mathbb{N}}\subset\mathbb{R}^n$ by*

$$y(k+1):=F_+\left(\frac{\beta}{\gamma}Ay(k)\right). \tag{11}$$

If $y(0)\geq0$ is a scalar multiple of $u_{\max}$ and satisfies either $0<\max_i y_i(0)\leq1-\gamma/(\beta\lambda_{\max})$ or $\min_i y_i(0)\geq1-\gamma/(\beta\lambda_{\max})$, then

$$\lim_{k\to\infty}y(k)=x^*.$$

Moreover, if $\max_i y_i(0)\leq1-\gamma/(\beta\lambda_{\max})$, then $y(k)$ is monotonically non-decreasing; if $\min_i y_i(0)\geq1-\gamma/(\beta\lambda_{\max})$, then $y(k)$ is monotonically non-increasing.

(iv) *the endemic state x^* is locally exponentially stable and its domain of attraction is $[0,1]^n\setminus\mathbb{0}_n$.*

Note: statement (ii) means that, near the onset of an epidemic outbreak, the exponential growth rate is $\beta\lambda_{\max}-\gamma$ and the outbreak tends to align with the dominant eigenvector $u_{\max}$; for more details see the discussion leading up to the approximate evolution (6). The basic reproduction number for this deterministic network SIS model is given by $R_0=\beta\lambda_{\max}/\gamma$. The network SI model discussed in Section 3 describes the limit behavior of the network SIS model as $\gamma/\beta\to0^+$. Statement(iii)c in Theorem 4.3 indicates that $x^*\to\mathbb{1}_n$ as $\gamma/\beta\to0^+$, which is consistent with statement (iii) in Theorem 3.2.

Proof of selected statements in Theorem 4.3..

(i) This point can be proved as done in point (i) of Theorem 3.2.

(ii) This follows from the same analysis of the linearized system as in the proof of Theorem 4.2(ii).

(iii) We begin by establishing two properties of the map $x\mapsto F_+(\hat{A}x)$, for $\hat{A}=\beta A/\gamma$. First, we claim that, $y\gg z\geq\mathbb{0}_n$ implies $F_+(\hat{A}y)\gg F_+(\hat{A}z)$. Indeed, note that G being connected implies that the adjacency matrix A has at least one strictly positive entry in each row. Hence, $y-z\gg\mathbb{0}_n$ implies $\hat{A}(y-z)\gg\mathbb{0}_n$ and, since f_+ is monotonically increasing, $\hat{A}y\gg\hat{A}z$ implies $F_+(\hat{A}y)\gg F_+(\hat{A}z)$.
Second, we observe that, for any $0<\alpha<1$ and $z>0$, we have $f_+(\alpha z)\geq z$ if and only if $z\leq1-1/\alpha$. Suppose $y(0)$ is a scalar multiple of $u_{\max}$ and $0<\max_i y_i(0)\leq1-\gamma/(\beta\lambda_{\max})$. We have

$$F_+(\hat{A}y(0))_i=f_+\left(\frac{\beta\lambda_{\max}}{\gamma}y_i(0)\right)\geq y_i(0).$$

Therefore, the sequence $\{y(k)\}_{k\in\mathbb{N}}$ defined by equation (11) satisfies $y(1)\geq y(0)$, which in turn leads to $y(2)=F_+(\hat{A}y(1))\geq F_+(\hat{A}y(0))=y(1)$, and by induction, $y(k+1)=F_+(\hat{A}y(k))\geq y(k)$ for any $k\in\mathbb{N}$. Such sequence $\{y(t)\}$ is monotonically non-decreasing and entry-wise upper bounded by $\mathbb{1}_n$. Therefore, as k diverges, $y(k)$ converges to some $x^*\gg\mathbb{0}_n$ such that $F_+(\hat{A}x^*)=x^*$. This proves the existence of an equilibrium $x^*=\lim_{k\to\infty}y(k)\gg\mathbb{0}_n$ as claimed in statements (iii)a and (iii)d.

Similarly, for any $0<\alpha<1$ and $z>0$, $f_+(\alpha z)\leq z$ if and only if $z\geq1-1/\alpha$. Following the same line of argument in the previous paragraph, one can check that the $\{y(k)\}_{k\in\mathbb{N}}$ defined by equation (11) is monotonically non-increasing and converges to some x^*, if $y(0)$ is a scalar multiple of $u_{\max}$ and satisfies $\min_i y_i(0)\geq1-\gamma/(\beta\lambda_{\max})$.

Now we establish the uniqueness of the equilibrium $x^*\in[0,1]^n\setminus\{\mathbb{0}_n\}$. First, we claim that an equilibrium point with an entry equal to 0 must be $\mathbb{0}_n$. Indeed, assume y^* is an equilibrium point and assume $y_i^*=0$ for some $i\in\{1,\dots,n\}$. The equality $y_i^*=f_+(\sum_{j=1}^n a_{ij}y_j^*)$ implies that also any node j with $a_{ij}>0$ must satisfy $y_j^*=0$. Because G is connected, all entries of y^* must be zero. Second, by contradiction, we assume there exists another equilibrium point $y^*\gg\mathbb{0}_n$ distinct from x^*. Let $\alpha:=\min_j\{y_j^*/x_j^*\}$ and let i such that $\alpha=y_i^*/x_i^*$. Then $y^*\geq\alpha x^*\gg\mathbb{0}_n$ and $y_i^*=\alpha x_i^*$. Notice that we can assume with no loss of generality that $\alpha<1$ otherwise we exchange x^* and y^*. Observe now that

$$\begin{aligned}\left(F_+(\hat{A}y^*)-y^*\right)_i&=f_+\left((\hat{A}y^*)_i\right)-\alpha x_i^*\\&\geq f_+\left(\alpha(\hat{A}x^*)_i\right)-\alpha x_i^* &&(\hat{A}\geq\mathbb{0}_{n\times n})\\&>\alpha f_+\left((\hat{A}x^*)_i\right)-\alpha x_i^* &&(0<\alpha<1\text{ and }z>0)\\&=\alpha\left(F_+(\hat{A}x^*)-x^*\right)_i=0. &&(x^*\text{is an equilibrium})\end{aligned}$$

Therefore, $\left(F_+(\hat{A}y^*)-y^*\right)_i>0$, which contradicts the fact that y^* is an equilibrium.

Now we prove (iii)b. Observe first that, since taking

$$y(0)=\left(1-\frac{\gamma}{\beta\lambda_{\max}}\right)\frac{u_{\max}}{\max_i\{u_{\max,i}\}}=\frac{\delta}{\delta+1}\frac{u_{\max}}{\max_i\{u_{\max,i}\}}$$

then $y(k)$ is monotonically non-decreasing and converges to x^*, and since taking instead

$$y(0)=\left(1-\frac{\gamma}{\beta\lambda_{\max}}\right)\frac{u_{\max}}{\min_i\{u_{\max,i}\}}=\frac{\delta}{\delta+1}\frac{u_{\max}}{\min_i\{u_{\max,i}\}}$$

then $y(k)$ is monotonically non-increasing and converges to x^*, we can argue that

$$\frac{\delta}{\delta+1}\frac{u_{\max}}{\max_i\{u_{\max,i}\}}\leq x^*\leq\frac{\delta}{\delta+1}\frac{u_{\max}}{\min_i\{u_{\max,i}\}}$$

This implies that x^* is infinitesimal as a function of δ. Consider the expansion $x^*(\delta)=x_1\delta+x_2\delta^2+O(\delta^3)$. Since the equilibrium x^* satisfies the equation

$$(\delta+1)\left(I_n-\operatorname{diag}(x^*)\right)Ax^*-\lambda_{\max}x^*=0$$

by substituting the expansion and equating to zero the coefficient of the term δ we obtain the equation

$$Ax_1-\lambda_{\max}x_1=0$$

which proves that x_1 is a multiple of $u_{\max}$, namely $x_1=au_{\max}$ for some constant a. By equating to zero the coefficient of the term δ^2 we obtain instead the equation

$$Ax_1+Ax_2-\operatorname{diag}(x_1)Ax_1-\lambda_{\max}x_2=0$$

Using the fact that $x_1=au_{\max}$ we argue that

$$a\lambda_{\max}u_{\max}+Ax_2-a^2\lambda_{\max}\operatorname{diag}(u_{\max})u_{\max}-\lambda_{\max}x_2=0$$

By multiplying on the left by $v_{\max}^T$ we obtain

$$a\lambda_{\max} v_{\max}^T u_{\max} - a^2 \lambda_{\max} v_{\max}^T \operatorname{diag}(u_{\max}) u_{\max} = 0$$

which proves that

$$a = \frac{v_{\max}^T u_{\max}}{v_{\max}^T \operatorname{diag}(u_{\max}) u_{\max}}$$

Point (iii)c can be proved in a similar way. Indeed, define $\epsilon := \gamma/\beta$. Since

$$\left(1 - \frac{\epsilon}{\lambda_{\max}}\right) \frac{u_{\max}}{\max_i\{u_{\max,i}\}} \leq x^* \leq \left(1 - \frac{\epsilon}{\lambda_{\max}}\right) \frac{u_{\max}}{\min_i\{u_{\max,i}\}}$$

we can argue that the expansion $x^*(\epsilon) = x_0 + x_1\epsilon + O(\epsilon^2)$ as ϵ tends to zero is such that $x_0 \gg \mathbb{0}_n$. Since the equilibrium x^* satisfies the equation

$$\big(I_n - \operatorname{diag}(x^*)\big) A x^* - \epsilon x^* = 0$$

by substituting the expansion and equating to zero the coefficient of the term ϵ^0 we obtain the equation

$$Ax_0 - \operatorname{diag}(x_0) A x_0 = 0$$

which proves that $x_0 = vectorones[n]$. By equating to zero the coefficient of the term ϵ^1 we obtain instead the equation

$$Ax_1 - \operatorname{diag}(x_1) A x_0 - \operatorname{diag}(x_0) A x_1 - x_0 = 0$$

Using the fact that $x_0 = \mathbb{1}_n$ we argue that

$$\operatorname{diag}(A\mathbb{1}_n) x_1 + \mathbb{1}_n = 0$$

which yieds the thesis.

(iv) For this point we refer to Fall et al. (2007), Lajmanovich and Yorke (1976) or Khanafer et al. (2016, Theorems 1 and 2) in the interest of brevity. □

Remark 4.4. The network SI model can be regarded as the limit case of the network SIS model with vanishing curing rate $\gamma \to 0^+$. According to Theorem 4.2 and 4.3, for any strongly connected digraph and any fixed infection rate $\beta > 0$, the quantity $\beta\lambda_{\max}/\gamma$ is always above the threshold in the limit $\gamma \to 0^+$. Moreover, statement (iii)(c) indicates that, as $\gamma \to 0^+$, the endemic state x^* satisfies $x^* \to \mathbb{1}_n$. Therefore, the behavior of the network SI model is the same as that for the network SIS model in the limit $\gamma \to 0^+$.

5. Network Susceptible-Infected-Recovered model

In this section we review the Susceptible-Infected-Susceptible (SIR) epidemic model.

5.1. Scalar SIR model

In this model individuals who recover from infection are assumed not susceptible to the epidemic any more. In this case, the population is divided into three distinct groups: $s(t)$, $x(t)$, and $r(t)$, denoting the fraction of susceptible, infected, and recovered individuals, respectively, with $s(t) + x(t) + r(t) = 1$. We write the (Susceptible-Infected-Recovered) SIR model as:

$$\begin{aligned} \dot{s}(t) &= -\beta s(t) x(t), \\ \dot{x}(t) &= \beta s(t) x(t) - \gamma x(t), \\ \dot{r}(t) &= \gamma x(t). \end{aligned} \tag{12}$$

The following results on the dynamical behavior of the scalar SIR model can be found in Hethcote (2000).

Lemma 5.1 (Dynamical behavior of the SIR model)**.** *Consider the SIR model (12). From each initial condition $s(0) + x(0) + r(0) = 1$ with $s(0) > 0$, $x(0) > 0$ and $r(0) \geq 0$, the resulting trajectory $t \mapsto (s(t), x(t), r(t))$ has the following properties:*

(i) *$s(t) > 0$, $x(t) > 0$, $r(t) \geq 0$, and $s(t) + x(t) + r(t) = 1$ for all $t \geq 0$;*

(ii) *$t \mapsto s(t)$ is monotonically decreasing and $t \mapsto r(t)$ is monotonically increasing;*

(iii) *$\lim_{t\to\infty}(s(t), x(t), r(t)) = (s_\infty, 0, r_\infty)$, where r_∞ is the unique solution to the equality*

$$1 - r_\infty = s(0) \mathrm{e}^{-\frac{\beta}{\gamma}\left(r_\infty - r(0)\right)}; \tag{13}$$

(iv) *if $\beta s(0)/\gamma < 1$, then $t \mapsto x(t)$ monotonically and exponentially decreases to zero as $t \to \infty$;*

(v) *if $\beta s(0)/\gamma > 1$, then $t \mapsto x(t)$ first monotonically increases to a maximum value and then monotonically decreases to 0 as $t \to \infty$; the maximum fraction of infected individuals is given by:*

$$x_{\max} = x(0) + s(0) - \frac{\gamma}{\beta}\left(\log(s(0)) + 1 - \log\left(\frac{\gamma}{\beta}\right)\right).$$

As mentioned before, we describe the behavior in statement (v) as an epidemic outbreak, an exponential growth of $t \mapsto x(t)$ for small times. The effective reproduction number in the deterministic scalar SIR model is $R = \beta s(t)/\gamma$. Note that the basic reproduction number $R_0 = \beta/\gamma$ does not have predict power in this model.

5.2. Network SIR model

The network SIR model on a graph with adjacency matrix A is given by

$$\begin{aligned} \dot{s}_i(t) &= -\beta s_i(t) \textstyle\sum_{j=1}^n a_{ij} x_j(t), \\ \dot{x}_i(t) &= \beta s_i(t) \textstyle\sum_{j=1}^n a_{ij} x_j(t) - \gamma x_i(t), \\ \dot{r}_i(t) &= \gamma x_i(t), \end{aligned}$$

where $\beta > 0$ is the infection rate and $\gamma > 0$ is the recovery rate. Note that the third equation is redundant because of the constraint $s_i(t) + x_i(t) + r_i(t) = 1$. Therefore, we regard the dynamical system in vector form as:

$$\dot{s}(t) = -\beta \operatorname{diag}(s(t)) A x(t), \tag{14a}$$

$$\dot{x}(t) = \beta \operatorname{diag}(s(t)) A x(t) - \gamma x(t). \tag{14b}$$

We state our main results of this section below. Weaker versions of statements (i)a and (i)b are due to Hethcote (1978). To the best of our knowledge, statements (i)c, (ii), (iii), (iv) and (v) are novel.

Theorem 5.2 (Dynamical behavior of the network SIR model)**.** *Consider the network SIR model (14), with $\beta > 0$ and $\gamma > 0$, over a strongly connected digraph with adjacency matrix A. For $t \geq 0$, let $\lambda_{\max}(t)$ and $v_{\max}(t)$ be the dominant eigenvalue of the non-negative matrix $\operatorname{diag}(s(t))A$ and the corresponding normalized left eigenvector, respectively. The following statements hold:*

(i) *if $x(0) > \mathbb{0}_n$, and $s(0) \gg \mathbb{0}_n$, then*
  (a) *$t \mapsto s(t)$ and $t \mapsto x(t)$ are strictly positive for all $t > 0$,*
  (b) *$t \mapsto s(t)$ is monotonically decreasing, and*
  (c) *$t \mapsto \lambda_{\max}(t)$ is monotonically decreasing,*

(ii) *the set of equilibrium points is the set of pairs $(s^*, \mathbb{0}_n)$, for any $s^* \in [0, 1]^n$, and the linearization of model (14) about $(s^*, \mathbb{0}_n)$ is*

$$\begin{aligned} \dot{s}(t) &= -\beta \operatorname{diag}\left(s^*\right) A x, \\ \dot{x}(t) &= \beta \operatorname{diag}\left(s^*\right) A x - \gamma x; \end{aligned} \tag{15}$$

(iii) *(behavior below the threshold) let the time $\tau \geq 0$ satisfy $\beta\lambda_{\max}(\tau) < \gamma$. Then the weighted average $t \mapsto v_{\max}(\tau)^\top x(t)$, for $t \geq \tau$, is monotonically and exponentially decreasing to zero;*

(iv) *(behavior above the threshold) if $\beta\lambda_{\max}(0) > \gamma$ and $x(0) > \mathbb{0}_n$, then,*

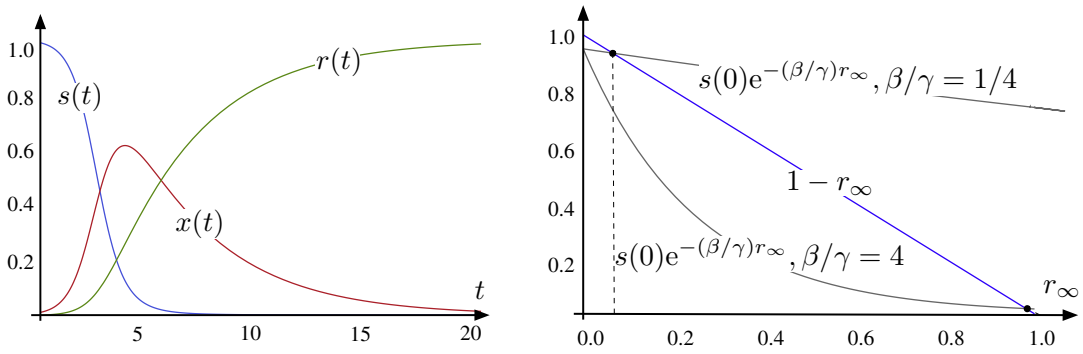

Fig. 4. Left figure: evolution of the scalar SIR model from small initial fraction of infected individuals (and zero recovered); parameters $\beta = 2$, $\gamma = 1/4$ (case (iv) in Lemma 5.1). Right figure: intersection between the two curves in Eq. (13) with $s(0) = 0.95$, $r(0) = 0$ and $\beta/\gamma \in \{1/4, 4\}$. If $\beta/\gamma = 1/4$, then $0.05 < r_\infty < 0.1$. If $\beta/\gamma = 4$, then $0.95 < r_\infty$.

(a) *(epidemic outbreak) for small time, the weighted average* $t \mapsto v_{\max}(0)^\top x(t)$ *grows exponentially fast with rate* $\beta\lambda_{\max}(0) - \gamma$, *and*
(b) *there exists* $\tau > 0$ *such that* $\beta\lambda_{\max}(\tau) < \gamma$;

(v) *each trajectory converges asymptotically to an equilibrium point, that is,* $\lim_{t\to\infty} x(t) = \mathbb{0}_n$ *so that the epidemic asymptotically disappears.*

The effective reproduction number in the deterministic network SIR model is $R(t) = \beta\lambda_{\max}(t)/\gamma$. When $R(0) > 1$, we have an epidemic outbreak, i.e., an exponential growth of infected individual for short time. In any case, the theorem guarantees that, after at most finite time, $R(t) < 1$ and the infected population decreases exponentially fast to zero.

Proof. Regarding statement (i)a, $s(t) \gg \mathbb{0}_n$ is due to the fact that Ax is bounded and $s(t)$ is continuously differentiable to t. The statement that $x(t) \gg \mathbb{0}_n$ for all $t > 0$ is proved in the same way as Theorem 4.2 (i). Statement (i)b is the immediate consequence of $\dot{s}_i(t)$ being strictly negative. From statement (i)a we know that each $s_i(t)$ is positive, and from A being irreducible and $x(0) \neq \mathbb{0}_n$ we know that $\sum_{j=1}^n a_{ij}x_j$ is positive. Therefore, $\dot{s}_i(t) = -\beta s_i(t)\sum_{j=1}^n a_{ij}x_j(t) < 0$ for all $i \in V$ and $t \geq 0$.

For statement(i)c, we start by recalling the following property from Meyer (2001, Example 7.10.2): for B and C nonnegative square matrices, if $B \leq C$, then $\rho(B) \leq \rho(C)$. Now, pick two time instances t_1 and t_2 with $0 < t_1 < t_2$. Let $\alpha = \max_i s_i(t_2)/s_i(t_1)$ and note $0 < \alpha < 1$ because $s(t)$ is strictly positive and monotonically decreasing. Now note that,

$$\operatorname{diag}(s(t_1))A > \alpha \operatorname{diag}(s(t_1))A \geq \operatorname{diag}(s(t_2))A,$$

so that, using the property above, we know

$$\rho(\operatorname{diag}(s(t_1))A) > \alpha\rho(\operatorname{diag}(s(t_1))A) \geq \rho(\operatorname{diag}(s(t_2))A).$$

This concludes the proof of statement (i)c.

Regarding statement (ii), note that a point (s^*, x^*) is an equilibrium if and only if:

$$\mathbb{0}_n = -\beta \operatorname{diag}\left(s^*\right)Ax^*, \quad \text{and}$$
$$\mathbb{0}_n = \beta \operatorname{diag}\left(s^*\right)Ax^* - \gamma x^*.$$

Therefore, each point of the form $(s^*, \mathbb{0}_n)$ is an equilibrium. On the other hand, summing the last two equalities we obtain $\mathbb{0}_n = \gamma x^*$ and thus x^* must be $\mathbb{0}_n$. As a straightforward result, the linearization of model (14) about any equilibrium point $(s^*, \mathbb{0}_n, \mathbb{1}_n - s^*)$ is given inEq. (15).

Regarding statement (iii), multiplying $v_{\max}(\tau)^\top$ from the left on both sides of Eq. (14b) we obtain:

$$\begin{aligned}\tfrac{d}{dt}\left(v_{\max}(\tau)^\top x(t)\right) &= v_{\max}(\tau)^\top\left(\beta \operatorname{diag}\left(s(t)\right)Ax(t) - \gamma x(t)\right)\\ &\leq v_{\max}(\tau)^\top\left(\beta \operatorname{diag}\left(s(\tau)\right)Ax(t) - \gamma x(t)\right)\\ &= (\beta\lambda_{\max}(\tau) - \gamma)v_{\max}(\tau)^\top x(t).\end{aligned}$$

Therefore, we obtain

$$v_{\max}(\tau)^\top x(t) \leq (v_{\max}(\tau)^\top x(0))\mathrm{e}^{(\beta\lambda_{\max}(\tau)-\gamma)t}.$$

The right-hand side exponentially decays to zero when $\beta\lambda_{\max}(\tau) < \gamma$. Therefore, $v_{\max}(\tau)^\top x(t)$ also decreases monotonically and exponentially to zero for all $t > \tau$.

Regarding statement (iv)a, note that based on the argument in (i)a, we only need to consider the case when $x(0) \gg \mathbb{0}_n$. Left-multiplying $v_{\max}(0)^\top$ on both sides of Eq. (14b), we obtain:

$$\begin{aligned}\frac{d}{dt}\left(v_{\max}(0)^\top x(t)\right)\Big|_{t=0} &= v_{\max}(0)^\top\left(\beta \operatorname{diag}\left(s(t)\right)Ax(t) - \gamma x(t)\right)\Big|_{t=0}\\ &= (\beta\lambda_{\max}(0) - \gamma)v_{\max}(0)^\top x(0).\end{aligned}$$

Since $\beta\lambda_{\max}(0) - \gamma > 0$, the initial time derivative of $v_{\max}(0)^\top x(t)$ is positive. Since $t \mapsto v_{\max}(0)^\top x(t)$ is a continuously differentiable function, there exists $\tau' > 0$ such that $\frac{d}{dt}\left(v_{\max}(0)^\top x(t)\right) > 0$ for any $t \in [0, \tau']$.

Regarding statement(iv)b, since $\dot{s}(t) \leq \mathbb{0}_n$ and is lower bounded by $\mathbb{0}_n$, we conclude that the limit $\lim_{t\to+\infty} s(t)$ exists. Moreover, since $s(t)$ is monotonically non-increasing, we have $\lim_{t\to+\infty} \dot{s}(t) = 0$, which implies either $\lim_{t\to+\infty} s(t) = \mathbb{0}_n$ or $\lim_{t\to+\infty} x(t) = \mathbb{0}_n$. If $s(t)$ converges to $\mathbb{0}_n$, then $\dot{x}(t)$ converges to $-\gamma x(t)$. Therefore, there exists $T > 0$ such that $\beta\lambda_{\max}(T) < \gamma$, which leads to $x(t) \to \mathbb{0}_n$ as $t \to +\infty$; If $s(t)$ converges to some $s^* > \mathbb{0}_n$, then $x(t)$ still converges to $\mathbb{0}_n$. Therefore, for any $(s(0), x(0))$, the trajectory $(s(t), x(t))$ converges to some equilibria with the form $(s^*, \mathbb{0}_n)$, where $s^* \geq \mathbb{0}_n$. Let

$$s(t) = s^* + \delta_s(t), \quad \text{and } x(t) = \mathbb{0}_n + \delta_x(t).$$

We know that $\delta_s(t) \geq 0$ and $\delta_x(t) \geq 0$ for all $t \geq 0$. Moreover, $\delta_s(t)$ is monotonically non-increasing and converges to $\mathbb{0}_n$, and there exists $\tilde{T} > 0$ such that, for any $t \geq T$, $\delta_x(t)$ is monotonically non-increasing and converges to $\mathbb{0}_n$.

Let λ^* and v^* denote the dominant eigenvalue and the corresponding normalized left eigenvector of matrix $\operatorname{diag}(s^*)A$, respectively, that is, $v^{*\top}\operatorname{diag}(s^*)A = \lambda^* v^{*\top}$. First let us suppose $\beta\lambda^* - \gamma > 0$, then the linearized system of (12) around $(s^*, \mathbb{0}_n)$ is written as

$$\dot{\delta}_s = -\beta \operatorname{diag}(s^*)A\delta_x,$$
$$\dot{\delta}_x = \beta \operatorname{diag}(s^*)A\delta_x - \gamma\delta_x.$$

Since $\beta\lambda^* - \gamma > 0$, the linearized system is exponentially unstable, which contradicts the fact that $\big(\delta_s(t), \delta_x(t)\big) \to (\mathbb{0}_n, \mathbb{0}_n)$ as $t \to +\infty$. Alternatively, suppose $\beta\lambda^* - \gamma = 0$. By left multiplying $v^{*\top}$ on both sides of the equation for $\dot{x}(t)$ in (12), we obtain

$$v^{*\top}\dot{\delta}_x = (\beta\lambda^* - \gamma)(v^{*\top}\delta_x) + \beta v^{*\top}\operatorname{diag}(\delta_s)A\delta_x = \beta v^{*\top}\operatorname{diag}(\delta_s)A\delta_x \geq \mathbb{0}_n,$$

which contradicts $\delta_x(t) \to \mathbb{0}_n$ as $t \to +\infty$. Therefore, we conclude that $\beta\lambda^* - \gamma < 0$. Since $\lambda_{\max}(t)$ is continuous on t, we conclude that there exists $\tau < +\infty$ such that $\beta\lambda_{\max}(t) - \gamma < 0$.

□

Remark 5.3. Consider the network SIR model as a parameterized dynamical system, with the curing rate γ as the parameter. The network SI model can be regarded the network SIR dynamics with $\gamma = 0$ and zero initial fraction of recovered individuals. However, due to the specific bifurcation behavior of the network SIR model at $\gamma = 0$, the dynamical properties of the network SIR model with $\gamma = 0$ are qualitatively different from the case when $\gamma > 0$. When $\gamma = 0$, the set given by statement (ii) of Theorem 5.2 is only a subset of the equilibrium set. Points in the set of pairs $(\mathbb{0}_n, x^*)$ are also the equilibria of the network SIR with $\gamma = 0$. In addition, while statement (iv)a of Theorem 5.2 on the initial epidemic outbreak is still true, statements (iv)b and (v) on the eventual decay no longer hold for $\gamma = 0$.

In what follows, we present a novel result on an iterative algorithm that computes the limit state $\lim_{t\to\infty}(s(t), 0, r(t))$ of the network SIR model (14) as a function of an arbitrary initial condition $(s(0), x(0), r(0))$.

Note that, for the scalar SIR model (12), if we define

$$V\big(s(t), x(t)\big) := s(t)e^{\frac{\beta}{\gamma}\left(1 - x(t) - s(t)\right)}.$$

Simple calculations result in $dV\big(s(t), x(t)\big)/dt = 0$, which implies that the trajectories are on the level sets of V and in the set $\{(s, x) \in \mathbb{R}^2 \mid s \geq 0, x \geq 0, s + x \leq 1\}$. Here, we apply a similar approach to the network SIR system (14). Let

$$V_i(s, r) := s_i e^{\frac{\beta}{\gamma}\sum_{j=1}^n a_{ij} r_j}, \quad \text{for any } i \in \{1, \dots, n\}.$$

One can check that, along any trajectory of dynamics (14), $dV_i/dt = 0$ for any $i \in \{1, \dots, n\}$. Therefore, the trajectories $(s(t), r(t))$ lie on the level curves of the functions $V_i(s, r)$ for $i \in \{1, \dots, n\}$.

Let $s(\infty) := \lim_{t\to+\infty} s(t)$, $x(\infty) := \lim_{t\to+\infty} x(t)$, and $r(\infty) := \lim_{t\to+\infty} r(t)$. Notice that $x(\infty) = \mathbb{0}_n$ and so $r(\infty) = \mathbb{1}_n - s(\infty)$. Since $dV_i/dt = 0$ for any $i \in \{1, \dots, n\}$, we have

$$s_i(\infty) = s_i(0)e^{-\frac{\beta}{\gamma}\sum_{j=1}^n a_{ij}\left(1 - r_j(0)\right)} e^{\frac{\beta}{\gamma}\sum_{j=1}^n a_{ij} s_j(\infty)}. \tag{16}$$

Given any initial condition $(s(0), r(0))$, the right-hand side of equation (16) defines a map

$$H(s) := e^{\frac{\beta}{\gamma}\operatorname{diag}\left(A(s - \mathbb{1}_n + r(0))\right)} s(0), \tag{17}$$

and $s(\infty)$ is a fixed point of H, that is, $s(\infty) = H\big(s(\infty)\big)$. The following theorem is novel.

Theorem 5.4 (Existence, uniqueness, and algorithm for the asymptotic point)**.** *Consider the network SIR model (14), with positive rates β and γ and with initial condition $(s(0), x(0), r(0))$ satisfying $s(0) \gg \mathbb{0}_n$, $x(0) > \mathbb{0}_n$, $r(0) \geq \mathbb{0}_n$ and $s(0) + x(0) + r(0) = \mathbb{1}_n$. Let $\big(s(\infty), \mathbb{0}_n, r(\infty)\big)$ be the asymptotic state of system (14). The map $H : \mathbb{R}^n \to \mathbb{R}^n$ defined by equation (17) has the following properties:*

(i) *there exists a unique fixed point s^* of the map H in the set $\{s \in \mathbb{R}^n \mid \mathbb{0}_n \leq s \leq \mathbb{1}_n - r(0)\}$. Moreover, $s^* = s(\infty)$ and $r(\infty) = \mathbb{1}_n - s^*$; and*

(ii) *any sequence $\{y(k)\}_{k\in\mathbb{N}}$ defined by $y(k+1) = H(y(k))$ and initial condition $\mathbb{0}_n \leq y(0) \leq \mathbb{1}_n - r(0)$ converges to the unique fixed point s^*.*

Proof. Since A is a non-negative matrix, and $s(0) \leq \mathbb{1} - r(0)$, one can easily observe that, if $\mathbb{0}_n \leq p \leq q \leq \mathbb{1}_n - r(0)$, then $\mathbb{0}_n \leq H(\mathbb{0}_n) \leq H(p) \leq H(q) \leq H(\mathbb{1}_n - r(0)) \leq \mathbb{1}_n - r(0)$. According to the Brower Fixed Point Theorem, the map H has at least one fixed point.

Define the sequence $\{p(k)\}_{k\in\mathbb{N}}$ by $p(k+1) = H(p(k))$ and $p(0) = \mathbb{0}_n$. Since

$$\mathbb{1}_n - r(0) \geq p(1) = H(\mathbb{0}_n) = e^{\frac{\beta}{\gamma}\operatorname{diag}\left(-A\mathbb{1}_n + Ar(0)\right)} s(0) \geq p(0),$$

we have $\mathbb{1}_n - r(0) \geq p(2) = H(p(1)) \geq H(p(0)) = p(1)$ and, by induction, $\mathbb{1}_n - r(0) \geq p(k+1) \geq p(k)$ for any $k \in \mathbb{N}$. Since $p(k)$ is non-decreasing and upper bounded by $\mathbb{1}_n - r(0)$, we conclude that the limit $p^* = \lim_{k\to\infty} p(k)$ exists, and p^* is a fixed point of the map H.

Similarly, define a sequence $\{q(k)\}_{k\in\mathbb{N}}$ by $q(k+1) = H(q(k))$ and $q(0) = \mathbb{1}_n - r(0)$. One can check that $q(k)$ is non-increasing and that $q^* = \lim_{k\to\infty} q(k)$ is a fixed point of map H. Moreover, since $p(0) \leq q(0)$, we have $p(k) \leq q(k)$ for any $k \in \mathbb{N}$ and thereby $p^* \leq q^*$.

If $p^* = q^*$, then, for any $\mathbb{0}_n \leq y(0) \leq \mathbb{1}_n - r(0)$, the sequence $\{y(k)\}_{k\in\mathbb{N}}$ defined by $y(k+1) = H(y(k))$ satisfies $p(k) \leq y(k) \leq q(k)$ for any $k \in \mathbb{N}$. Therefore, $y^* = \lim_{k\to\infty} y(k)$ exists and $y^* = p^* = q^*$, which implies that the fixed point of map H is unique. According to Eq. (16), $s(\infty)$ is the unique fixed point. This concludes the proof for statement (i) and (ii).

Now we eliminate the case $p^* < q^*$ by contradiction. First of all we prove that $q^* \ll \mathbb{1}_n - r(0)$. Let $N_i = \{j \mid a_{ij} > 0\}$ and $\mathcal{I}(k) = \big\{i \,\big|\, q_i(\tau) < 1 - r_i(0) \text{ for any } \tau \geq k\big\}$. We have $\mathcal{I}(0) = \phi$. Since $x(0) > \mathbb{0}_n$, we have $q(1) = s(0) < 1 - r(0)$, that is, there exists i such that $q_i(1) < 1 - r_i(0)$. Moreover, since $q(k)$ is non-increasing, we have $q(k) \leq q(1)$ for any $k \geq 1$. Therefore, for any i such that $q_i(1) < 1 - r_i(0)$, it satisfies $q_i(k) \leq q_i(1) < 1 - r_i(0)$ for any $k \geq 1$. Since $j \notin \mathcal{I}(1)$ if $q_j(1) = s_j(0) = 1 - r_j(0)$, we conclude that $\mathcal{I}(1) = \{i \mid s_i(0) < 1 - r_i(0)\}$. Moreover, for any given $k \geq 1$, since, for any i such that $N_i \cap \mathcal{I}(k) \neq \phi$,

$$\begin{aligned} q_i(k+1) &= H(q(k))_i = e^{\frac{\beta}{\gamma}\sum_{j=1}^n a_{ij}\left(q_j(k) - 1 + r_j(0)\right)} s_i(0) \\ &< s_i(0) \leq 1 - r_i(0); \end{aligned}$$

and for any i such that $N_i \cap \mathcal{I}(k) = \phi$ and $i \notin \mathcal{I}(k)$,

$$\begin{aligned} q_i(k+1) &= H(q(k))_i = e^{\frac{\beta}{\gamma}\sum_{j=1}^n a_{ij}\left(q_j(k) - 1 + r_j(0)\right)} s_i(0) = s_i(0) \\ &= 1 - r_i(0), \end{aligned}$$

we have $\mathcal{I}(k+1) = \{i \mid N_i \cap \mathcal{I}(k) \neq \phi\} \cup \mathcal{I}(k)$ for any $k \geq 1$. Because the graph associated with A is strongly connected, we can argue that $\mathcal{I}(k)$ contains all the indices when k is large enough. Therefore, $q^* \ll \mathbb{1}_n - r(0)$.

Now suppose $p^* < q^*$. Let

$$\alpha = \min_j \frac{1 - r_j(0) - p_j^*}{q_j^* - p_j^*}, \quad \text{and} \quad w = (1 - \alpha)p^* + \alpha q^*.$$

We have $\alpha > 1$, $\mathbb{0}_n \leq w < \mathbb{1}_n - r(0)$, and $w_i = 1 - r_i(0)$ for any i such that $\alpha_i = \big(1 - r_i(0) - p_i^*\big)/(q_j^* - p_j^*)$. Let $\mu = 1/\alpha$. Thereby $q^* = \mu w + (1 - \mu)p^*$, where $0 < \mu < 1$. This means that q^* is a convex combination of p^* and w. Since $H(s)_i$ is a strictly convex function of s, we obtain that

$$\begin{aligned} q_i^* = H\big(\mu w + (1 - \mu)p^*\big)_i &< \mu H(w)_i + (1 - \mu)p_i^* \leq \mu\big(1 - r_i(0)\big) \\ &+ (1 - \mu)p_i^* = q_i^*. \end{aligned}$$

In the last inequality, we used the fact that $H(w)_i \leq 1 - r_i(0)$ for any $0 \leq w \leq \mathbb{1}_n - r(0)$. The previous inequality yields a contradiction. □

In the rest of this section, we present some numerical results for the network SIR model for the famous Krackhardts advice network illustrated in Fig. 5. This network reflects the data collected

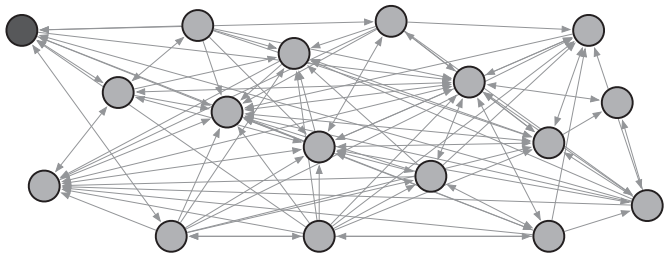

Fig. 5. Main strongly-connected component of the Krackhardt digraph with 17 nodes.

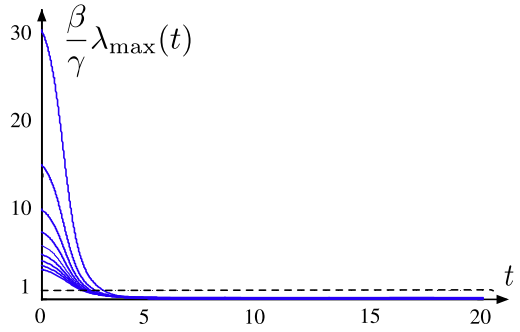

Fig. 6. Evolution of the spectral radius of $(\beta/\gamma)\,\mathrm{diag}(s(t))A)$ over the strongly connected digraph in Fig. 5. The parameter γ takes value in $.1, .2, \ldots, .9$, corresponding respectively to the curves from up to down in the time interval [0, 5].

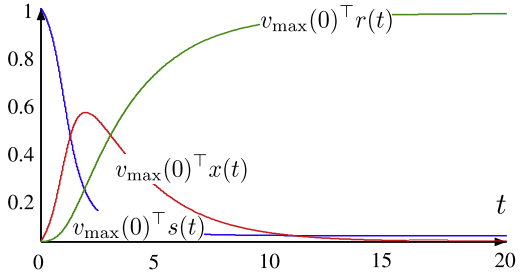

Fig. 7. Evolution of the network SIR model from initial condition consisting of one node fully infected individual (the dark-gray node in Fig. 5), 16 fully healthy individuals, and zero recovered fraction. The effective reproduction number satisfies $R(0) = 3.57$.

by Krackhardt (1987) on the cognitive social structure of the management personnel in a high-tech machine manufacturing firm. In the network, each node represents an individual, and each directed link (i, j) means that individual i seeks advice from individual j. We refer the interested readers to Krackhardt (1987) for more details.

Consider the epidemic spreading process on the Krackhardts advice network. The associated adjacency matrix A is binary. Unless otherwise stated, the system parameters are set as $\beta = 0.5$ and $\gamma = 0.4$. As for initial condition, we select one node fully infected (the dark-gray node in Fig. 5, say, with index 1), 16 fully healthy individuals, and zero recovered fraction—corresponding to $x(0) = e_1$, $r(0) = \mathbb{0}_n$, and $s(0) = \mathbb{1}_n - x(0)$. These parameters lead to an initial effective reproduction number $R(0) = 3.57$.

Fig. 6 illustrates the time evolution of $(\beta/\gamma)\lambda_{\max}(t)$ with varying network parameters. Note that each evolution starts above the threshold, reaches the threshold value 1 in finite time, and converges to a final value below 1. Fig. 7 illustrates the behavior of the average susceptible, average infected and average recovered quantities in populations starting from a small initial infection fraction and with an effective reproduction number above 1 at time 0. Note that the evolution of the infected fraction of the population displays a unimodal dependence on time, like in the scalar model.

6. Conclusion

This paper provides a comprehensive and consistent treatment of deterministic nonlinear continuous-time SI, SIS, and SIR propagation models over contact networks. We investigated the asymptotic behaviors (vanishing infection, steady-state epidemic, and full contagion). We studied the transient propagation of an epidemic starting from small initial fractions of infected nodes. We presented conditions under which a possible epidemic outbreak occurs or the infection monotonically vanishes for arbitrary fixed topology graphs. We introduced a network SI model and analyzed its behavior. Network SIS model sections includes improved properties over previously proposed works. New transient behavior, threshold condition, and system properties for the network SIR model were proposed. In addition, for the network SIR model, we provide a novel iterative algorithm to compute the asymptotic state of the system. In all cases, we show the results for network models are appropriate generalizations of those for the respective scalar models.

There are numerous potential future research directions regarding the deterministic network epidemic processes and the literature is still growing rapidly. Recent progress in this area includes but is not limited to the modeling and analysis of epidemic spreading on time-varying networks, e.g., see Ogura and Preciado (2016), Paré, Beck, and Nedić (2017), the optimal immunization strategies, e.g., see Nowzari, Preciado, and Pappas (2017), Ramírez-Llanos and Martínez (2017), and the competitive propagation of multiple virus/memes, e.g., see Goyal, Heidari, and Kerans (2014), Mei and Bullo (2017), Paré, Liu, Beck, Nedić, and Başar (2017).

Finally, we point out that, although the network SI, SIS, and SIR models have attracted enormous attention by researchers working on network epidemics, they are not the only deterministic models of epidemic spreading processes on networks. For example, there is another class of deterministic network models, referred to as the *multi-city model* or the *epidemic model in a patchy environment*. This class of models considers each node in the network as a city obeying the scalar SIS or SIR dynamics. The disease is spread via the traffic flows between those cities. We refer the interested reader to Arino and van den Driessche (2010), Colizza, Barrat, Barthélemy, and Vespignani (2005), Wang and Zhao (2004) for detailed treatments.

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Wenjun Mei received his Bachelor of Science in Theoretical and Applied Mechanics from Peking University in 2011. He is now a PhD candidate in the Department of Mechanical Engineering at the University of California, Santa Barbara. His research interests include modeling, analysis and control of dynamical systems on complex networks.

Shadi Mohagheghi received her Bachelor's Degree in Electrical and Electronics Engineering from University of California, Los Angeles in 2015. She is now a PhD student in the Department of Electrical and computer Engineering at the University of California, Santa Barbara. Her research interests include dynamical systems, adaptive systems, estimation and detection, and sensor networks.

Sandro Zampieri is Full Professor in Automatic Control at the Department of Information Engineering of the University of Padova since 2002. He received the Laurea degree in Electrical Engineering and the PhD degree in System Engineering from the University of Padova, Italy, in 1988 and 1993, respectively. His research interests include automatic control and dynamical systems theory, and in particular distributed control and estimation and networked control and control under communication constraints.
He has delivered several invited seminars and he was member of the Technical Program Committee for several international conferences. He was general chair of the 1st IFAC Workshop on Estimation and Control of Networked Systems 2009, program chair of the 3rd IFAC Workshop on Estimation and Control of Networked Systems 2012 and publication chair of the IFAC World Congress 2011. He served as an Associate Editor of the Siam Journal on Control and Optimization on 2002–2004 and as the chair of the IFAC technical committee "Networked systems" on 2005–2008. Since 2012 he is serving as an Associate Editor of IEEE Transactions of Automatic Control.

Francesco Bullo is a Professor with the Mechanical Engineering Department and the Center for Control, Dynamical Systems and Computation at the University of California, Santa Barbara. He was previously associated with the University of Padova, the California Institute of Technology, and the University of Illinois.
His research interests focus on network systems and distributed control with application to robotic coordination, power grids and social networks. He is the coauthor of Geometric Control of Mechanical Systems (Springer, 2004) and Distributed Control of Robotic Networks (Princeton, 2009); his forthcoming Lectures on Network Systems is available on his website. He received best paper awards for his work in IEEE Control Systems, Automatica, SIAM Journal on Control and Optimization, IEEE Transactions on Circuits and Systems, and IEEE Transactions on Control of Network Systems. He is a Fellow of IEEE and IFAC. He has served on the editorial boards of IEEE, SIAM, and ESAIM journals, and will serve as IEEE CSS President in 2018.