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Stability of SIS Spreading Processes in Networks With Non-Markovian Transmission and Recovery

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***Abstract*—Although viral spreading processes taking place in networks are often analyzed using Markovian models, in which both the *transmission* and the *recovery* times follow exponential distributions, empirical studies show that, in many real scenarios, the distribution of these times is not necessarily exponential. To overcome this limitation, we first introduce a generalized susceptible–infected–susceptible spreading model that allows transmission and recovery times to follow *phase-type* distributions. In this context, we derive a lower bound on the exponential decay rate toward the infection-free equilibrium of the spreading model without relying on mean-field approximations. Based on our results, we illustrate how the particular shape of the transmission/recovery distribution influences the exponential rate of convergence toward the equilibrium.**

***Index Terms*—Complex networks, Markov processes, multiagent systems, stochastic systems.**

I. Introduction

UNDERSTANDING the dynamics of spreading processes in complex networks is a challenging problem with a wide range of practical applications in epidemiology and public health [3], information propagation in social networks [21], or cyber-security [38]. During the last decade, significant progress has been made toward understanding the relationship between the topology of a network and the dynamics of spreading processes taking place over the network (see [28] and [35] for recent surveys). A common approach to investigate this relationship is by modeling spreading processes using networked Markov processes, such as the networked susceptible–infected–susceptible (SIS) model [44]. Based on these Markovian models, it is then possible to find an explicit relationship between epidemic thresholds and network eigenvalues in static topologies [18], [34], [44], [46]; as well as in multilayer [10], [40]; time-varying [29], [33]; and adaptive [23], [30] networks. Markovian models also allow us to design optimal strategies for containment of spreading processes taking place in static, uncertain, and temporal networks (see, e.g., [13] and [37]).

A consequence of using Markovian models in the analysis of spreading processes is that both *transmission times* (i.e., the time it takes for an infection to be transmitted from an infected node to one of its neighbors) and *recovery times* (i.e., the time it takes for an infected node to recover) follow exponential distributions. However, empirical studies show that, in many real networks, the distribution of transmission and recovery times is not necessarily exponential. For example, the transmission time of messages in Twitter, or news in other social media outlets, follows (approximately) a log-normal distribution [21], [45]. In the context of human contact networks, the transmission of various infectious diseases [4], [7], [17], [26] or the time it takes to recover from the influenza virus [42] is often nonexponential.

Since realistic transmission and recovery times often follow nonexponential distributions, it is of practical importance to understand the role of these distributions on the dynamics of the spread. In this direction, the authors in [45] illustrated, via numerical simulations, that nonexponential transmission times can have a substantial effect on the dynamics of the spread. Motivated by this study, several approximative methods for quantifying the steady-state fraction of infected nodes have been proposed in the literature. In this direction, the authors in [5] analyzed spreading processes with general transmission and recovery times using mean-field approximations. In [16] and [25], simple but yet analytically solvable spreading models with nonexponential transmission times were studied. Moment-closure approximations for analyzing spreading processes with nonexponential transmission and recovery times were proposed in [19] and [36]. Under the assumption that recovery times follow an exponential distribution, the analytical framework in [41] enables us to reduce nonexponentially distributed transmission times into exponentially distributed counterparts without changing the steady state of the spread. The authors in [27] used a mean-field approximation to derive stability conditions for the infection-free equilibrium of a spreading process with three compartments and non-Markovian transition dynamics. However, either their conditions are conservative or they guarantee only the local stability of the infection-free equilibrium.

In this paper, we propose a tractable but rigorous approach to analyze the *transient* of SIS spreading processes over arbitrary networks with general (nonexponential) transmission and recov-

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ery times. In this direction, we first introduce the *generalized networked susceptible–infected–susceptible* (GeNeSIS) model, which allows for transmission and recovery times following arbitrary phase-type distributions (see, e.g., [2]). Defined as the *exit time* of time-homogeneous Markov processes, phase-type distributions form a dense family in the space of positive-valued distributions [9]. Therefore, the GeNeSIS model allows us to theoretically analyze arbitrary transmission and recovery times within an arbitrary accuracy [2]. We are particularly interested in quantifying the exponential decay rate of the spread toward the infection-free equilibrium, in other words to eradicate the viral spreading process. The key tool used in our derivations is a vectorial representation of phase-type distributions, which we use to bound the exponential decay rate toward the infection-free equilibrium in the stochastic dynamics of the GeNeSIS model.

The rest of this paper is organized as follows. In Section II, we introduce elements of graph theory and stochastic differential equations with Poisson jumps. In Section III, we describe a generalized SIS model over networks with arbitrary transmission and recovery times. In Section IV, we provide a vectorial representation of the GeNeSIS model, which we use in Section V to analyze the exponential decay rate toward the infection-free equilibrium. We validate the effectiveness of our results via numerical simulations in Section VI, where we also illustrate the effect of nonexponential transmission/recovery times in the dynamics of the spread.

II. MATHEMATICAL PRELIMINARIES

Let $\mathbb{R}$ and $\mathbb{N}$ denote the set of real numbers and positive integers, respectively. For a positive integer n, define $[n] = \{1, \dots, n\}$. For a real function f, let $f(t^-)$ denote the limit of f from the left at time t. We let I_n and O_n denote the $n \times n$ identity and zero matrices. By $\mathbb{1}_p$ and 0_p, we denote the p-dimensional vectors, whose entries are all ones and zeros, respectively. A real matrix A (or a vector as its special case) is said to be nonnegative, denoted by $A \geq 0$, if A is nonnegative entrywise. The notation $A \leq 0$ is understood in the obvious manner. For a square matrix A, the maximum real part of its eigenvalues, called the *spectral abscissa* of A, is denoted by $\eta(A)$. We say that A is Metzler if the off-diagonal entries of A are all nonnegative. It is easy to see that, if A is Metzler, then $e^{At} \geq 0$ for every $t \geq 0$. The Kronecker product [15] of two matrices A and B is denoted by $A \otimes B$, and the Kronecker sum of two square matrices $A \in \mathbb{R}^{p\times p}$ and $B \in \mathbb{R}^{q\times q}$ is defined by

$$A \oplus B = A \otimes I_q + I_p \otimes B.$$

Given a collection of n matrices $A_1, \dots, A_n$ having the same number of columns, the matrix obtained by stacking the matrices in vertical (A_1 on top) is denoted by $\operatorname{col}(A_1, \dots, A_n)$.

An undirected graph is a pair $\mathcal{G} = (\mathcal{V}, \mathcal{E})$, where $\mathcal{V} = \{1, \dots, n\}$ is the set of nodes, and $\mathcal{E} \subset \mathcal{V} \times \mathcal{V}$ is the set of edges, consisting of distinct and unordered pairs $\{i, j\}$ for $i, j \in \mathcal{V}$. We say that a node i is a neighbor of j (or that i and j are adjacent) if $\{i, j\} \in \mathcal{E}$. The set of neighbors of node i is denoted by $\mathcal{N}_i$. The adjacency matrix of $\mathcal{G}$ is defined as the $n \times n$ matrix whose (i, j)th entry is 1 if and only if nodes i and j are adjacent, 0 otherwise.

We let $P(\cdot)$ denote the probability of events. The expectation of a random variable is denoted by $E[\cdot]$. A Poisson counter [6, Ch. 4] of rate $\lambda > 0$ is denoted by N_λ. In this paper, we extensively use a specific class of stochastic differential equations with Poisson jumps, described below. For each $i \in [m]$, let $f_i : \mathbb{R} \times \mathbb{R} \to \mathbb{R}$ be a continuous function, N_{λ_i} be a Poisson counter, and $\boldsymbol{\kappa}_i$ be a continuous-time, real, and stationary stochastic process defined over the probability space Ω. All the above stochastic processes are assumed to be independent of each other. Then, we say that a real and right-continuous function x is a solution of the stochastic differential equation

$$dx = \sum_{i=1}^{m} f_i(x(t), \boldsymbol{\kappa}_i(t))\, dN_{\lambda_i} \tag{1}$$

if x is constant on any interval, where none of the counters N_{λ_1}, $\dots$, N_{λ_m} jumps, and

$$x(t) = x(t^-) + f_i(x(t^-), \boldsymbol{\kappa}_i(t))$$

when N_{λ_i} jumps at time t. This definition can naturally be extended to the vector case. In the following, we present two lemmas for this class of stochastic differential equations. The first lemma states a version of Itô's formula.

Lemma II.1: Assume that x is a solution of (1). Let g be a real continuous function. Then, $y(t) = g(x(t))$ is a solution of the stochastic differential equation

$$dy = \sum_{i=1}^{m} \left[g\left(x(t) + f_i\left(x(t), \boldsymbol{\kappa}_i(t)\right)\right) - g(x(t))\right] dN_{\lambda_i}$$

i.e., y satisfies

$$y(t) = y(t^-) + g\left(x(t^-) + f_i(x(t^-), \boldsymbol{\kappa}_i(t))\right) - g(x(t^-))$$

if the Poisson counter N_{λ_i} jumps at time t and is constant over any interval in which none of the counters $N_{\lambda_1}, \dots, N_{\lambda_m}$ jumps.

Proof: Assume that the counter N_{λ_i} jumps at time t. Since x is the solution of the stochastic differential equation (1), it follows that $y(t) - y(t^-) = g\left(x(t^-) + f_i(x(t^-), \boldsymbol{\kappa}_i(t))\right) - g(x(t^-))$, as desired. ■

We also state the following lemma concerning the expectation of the solution to the stochastic differential equation (1).

Lemma II.2: Assume that x is a solution of (1). If the functions $f_1, \dots, f_m$ are affine with respect to the second variable, then

$$\frac{d}{dt} E[x(t)] = \sum_{i=1}^{m} E\left[f_i(x(t), E[\boldsymbol{\kappa}_i(t)])\right] \lambda_i. \tag{2}$$

Proof: By the assumption, for each $i \in [m]$, we can take real functions $f_{i,1}$ and $f_{i,2}$ such that $f_i(a, b) = f_{i,1}(a) + b f_{i,2}(a)$ for every $a, b \in \mathbb{R}$. Let $t \geq 0$ and $h > 0$ be arbitrary. We have the following three possibilities: 1) no counter jumps on the time interval $[t, t + h]$; 2) exactly one counter jumps in the interval; or 3) more than one counter jumps in the interval. The first case happens with probability $1 - (\lambda_1 + \dots + \lambda_m)h + o(h)$. For the second case, for each $i \in [m]$, one and the only one counter N_{λ_i} jumps on the time interval $[t, t + h]$ with probability $\lambda_i h + o(h)$.

In this case, we have

$$\begin{aligned} x(t+h) &= x(t) + f_i(x(t), \boldsymbol{\kappa}_i(\tau)) \\ &= x(t) + f_{i,1}(x(t)) + \boldsymbol{\kappa}_i(\tau) f_{i,2}(x(t)) \end{aligned}$$

for some $\tau \in [t, t+h]$ and therefore

$$\begin{aligned} E[x(t+h)] &= E[x(t)] + E[f_{i,1}(x(t))] \\ &\quad + E[\boldsymbol{\kappa}_i(\tau)] E[f_{i,2}(x(t))] \\ &= E[x(t)] + E\big[f_i(x(t), E[\boldsymbol{\kappa}_i(t)])\big] \end{aligned}$$

because $\boldsymbol{\kappa}_i$ is a stationary stochastic process. Finally, the third case occurs with probability $o(h)$. Summarizing, we have shown that

$$\frac{E[x(t+h)] - E[x]}{h} = \frac{o(h)}{h} + \sum_{i=1}^{m} E\big[f_i(x(t), E[\boldsymbol{\kappa}_i(t)])\big]\lambda_i$$

which proves (2) in the limit of $h \to 0$. ■

III. SIS Model With General Transmission and Recovery Times

The aim of this section is to introduce the GeNeSIS model, which will allow us to analyze the effect of nonexponential transmission and recovery times in the spreading dynamics.

A. GeNeSIS Model

We start by giving a brief overview of the standard SIS model (see, e.g., [28] and [35]). Let $\mathcal{G} = (\mathcal{V}, \mathcal{E})$ be an undirected and unweighted graph with n nodes. In the SIS model, at a given (continuous) time $t \geq 0$, each node can be in one of two possible states: *susceptible* or *infected*. If a neighbor of node i is infected, then this neighbor can infect node i with an instantaneous rate β_i, where $\beta_i > 0$ is called the *transmission rate* of node i. Therefore, while being infected, the neighbor attempts to infect node i with the interevent times following an exponential distribution of rate β_i. On the other hand, when a node i is infected, it can randomly transition to the susceptible state with an instantaneous rate $\delta_i > 0$, called the *recovery rate* of node i. This implies that the time it takes for an infected node i to recover follows an exponential distribution of rate δ_i.

Before we introduce the GeNeSIS model, we introduce the following notations. We describe the state of a node $i \in \mathcal{V}$ by a $\{0, 1\}$-valued continuous-time stochastic process, denoted by $z_i = \{z_i(t)\}_{t\in\mathbb{R}}$. We say that node i is susceptible (respectively, infected) at time t if $z_i(t) = 0$ (respectively, $z_i(t) = 1$). We assume that the function z_i is continuous from the right for all $i \in [n]$. Under this assumption, we say that node i becomes infected (respectively, becomes susceptible) at time t if $z_i(t^-) = 0$ and $z_i(t) = 1$ (respectively, $z_i(t^-) = 1$ and $z_i(t) = 0$). It is assumed that all nodes are susceptible before time $t = 0$, i.e., $z_i(t) = 0$ for $t < 0$. We now introduce the GeNeSIS model as follows.

Definition III.1: We say that the family $z = \{z_i\}_{i\in[n]}$ of stochastic processes is a *GeNeSIS model* if there exist a subset $\mathcal{V}_0 \subset [n]$, as well as random variables

$$0 = \tau_0^{ji}(t) < \tau_1^{ji}(t) < \cdots$$

and $\boldsymbol{\rho}^i(t) > 0$ satisfying the following conditions for all $i \in [n]$, $j \in \mathcal{N}_i$, and $t \geq 0$.

a) Node i becomes infected at time $t = 0$ if and only if $i \in \mathcal{V}_0$, i.e., $\mathcal{V}_0$ is the initially infected subset.

b) Assume that node i becomes infected at time t. Then, node i remains infected during the time interval $[t, t + \boldsymbol{\rho}^i(t))$ and becomes susceptible at time $t + \boldsymbol{\rho}^i(t)$, i.e., the random variable $\boldsymbol{\rho}^i(t)$ is the *recovery time* of node i;

c) If node i becomes infected at time t, then, until its recovery, the node attempts to infect node $j \in \mathcal{N}_i$ at times $\{t + \tau_k^{ji}(t)\}_{k\in\mathbb{N}}$, i.e., if node j is susceptible at time $t + \tau_k^{ji}(t)$ for any $k \in \mathbb{N}$, then node j becomes infected.

Remark III.2: We call the random increments $\{\tau_k^{ji}(t) - \tau_{k-1}^{ji}(t)\}_{j\in\mathcal{N}_i,\, t\geq 0,\, k\in\mathbb{N}}$ the *transmission times* of node i, since the difference $\tau_k^{ji}(t) - \tau_{k-1}^{ji}(t)$ represents the time between infection attempts from an infected node i toward a neighboring node j. Note that, when all the recovery and transmission times follow exponential distributions, the GeNeSIS model recovers the standard networked SIS model described at the beginning of this subsection.

Notice that the origin (i.e., $z_i = 0$ for all $i \in \mathcal{V}$) is an absorbing state of the GeNeSIS dynamics. In what follows, we will refer to the origin as the *infection-free* equilibrium. The aim of this paper is to quantify the transient dynamics of the generalized SIS model according to the following definition.

Definition III.3: The *exponential decay rate* of the GeNeSIS model is defined by

$$\lambda = -\sup_{\mathcal{V}_0\subset[n]} \limsup_{t\to\infty} \frac{\log \sum_{i=1}^{n} E[z_i(t)]}{t}.$$

Since the sum $\sum_{i=1}^{n} E[z_i(t)]$ equals the expected number of infected nodes at time t, the decay rate λ quantifies how fast the infectious spreading process dies out in the network (in average). Besides quantifying the impact of contagious spreading processes over networks [12], [20], [45], the exponential decay rate has been used as a standard tool for measuring the performance of strategies aiming to contain epidemic outbreaks [1], [13], [37], [46]. We further remark that, although exponential *distributions* are not necessarily appropriate for modeling realistic transmission and recovery times as discussed in Section I, the exponential *decay rate* is still a valid quantity for measuring the spreading capability of epidemic processes.

B. Phase-Type Transmission and Recovery Times

In this paper, we consider the GeNeSIS model with transmission and recovery times following phase-type distributions [2]. In what follows, we briefly describe this class of probability distributions. Consider a time-homogeneous Markov process x in continuous time with $p + 1$ ($p \in \mathbb{N}$) states (also called *phases*) such that the states $1, \ldots, p$ are transient and the remaining state $p + 1$ is absorbing. The infinitesimal generator of the process is then necessarily of the form

$$\begin{bmatrix} T & b \\ 0 & 0 \end{bmatrix}, \quad b = -T\mathbb{1}_p \tag{3}$$

where $T \in \mathbb{R}^{p\times p}$ is an invertible Metzler matrix with nonpositive row sums. Let

$$\begin{bmatrix} \phi \\ 0 \end{bmatrix} \in \mathbb{R}^{p+1} \quad (\phi \in \mathbb{R}^p)$$

denote the initial distribution of the Markov process x, i.e.,

$$P(x(0) = m) = \begin{cases} \phi_m, & m \in [p] \\ 0, & m = p+1. \end{cases}$$

Then, the time to absorption into the state $p+1$ is a random variable following a *phase-type distribution*, which we denote by the pair (ϕ, T). In the rest of this paper, we make the following assumption on the distribution of (random) transmission and recovery times in the GeNeSIS model.

Assumption III.4: Transmission and recovery times of all nodes follow phase-type distributions (ϕ, T) and (ψ, R), respectively.

The class of phase-type distributions include various distributions of theoretical and practical interests. For example, if we choose the parameters $p = 1$, $T = -\beta$, and $\phi = 1$, then the phase-type distribution (ϕ, T) equals an exponential distribution with mean $1/\beta$. A phase-type distribution can also represent various classes of distributions including the Erlang, Coxian, and hyperexponential distributions [8]. Furthermore, it is known that the set of phase-type distributions is dense in the set of positive-valued distributions [9]. Therefore, it is possible to approximate an arbitrary distribution by a phase-type distribution within any given accuracy. Moreover, there are efficient numerical algorithms for finding the parameters of an approximating phase-type distribution [2]. Hence, under Assumption III.4, the GeNeSIS model allows us to efficiently approximate realistic spreading processes having non-Markovian transmission and recovery distributions.

IV. VECTORIAL REPRESENTATIONS

The aim of this section is to introduce a vectorial representation of the GeNeSIS model under Assumption III.4. We start our exposition by providing a vectorial representation of an arbitrary phase-type distribution (see Section IV-A) and then present a vectorial representation of the GeNeSIS model (see Section IV-B) that shall be used in Section V for analyzing the decay rate of the spreading model.

A. Vectorial Representation of Phase-Type Distributions

In what follows, we use the following notation: For $m, m' \in [p]$, let $E_{mm'}$ denote the $p \times p$ matrix whose entries are all zeros, except for its (m, m')th entry being 1. Also, let e_m denote the mth vector of the canonical basis in $\mathbb{R}^p$ (i.e., all the entries of e_m are zeros, except for the mth entry being 1). Finally, given a probability distribution ϕ on $[p]$, we say that an $\mathbb{R}^p$-valued random variable x follows the distribution ϕ, denoted by $x \sim \phi$, if

$$P(x = e_m) = \phi_m$$

for every $m \in [p]$.

By identifying the state space of the underlying Markov process by the set of vectors $\{e_1, \dots, e_p, 0\} \subset \mathbb{R}^p$, the following proposition allows us to represent the phase-type distribution as the *exit time* (see [32, p. 117]) of a vectorial stochastic differential equation.

Proposition IV.1: Let (ϕ, T) be a phase-type distribution having $p+1$ phases (i.e., $T \in \mathbb{R}^{p\times p}$) and define the vector $b \in \mathbb{R}^p$ as in (3). Consider the $\mathbb{R}^p$-valued stochastic differential equation

$$dx = \sum_{m=1}^{p}\sum_{m'=1}^{p}(E_{m'm} - E_{mm})x\, dN_{T_{mm'}} - \sum_{m=1}^{p} E_{mm}\, x\, dN_{b_m} \tag{4}$$

with random initial condition $x(0) \sim \phi$. Then, the random variable

$$\begin{aligned} \boldsymbol{\rho} &= \min\{t > 0 : x(t) = 0\} \\ &= \min\{t > 0 : \exists\, m \in [p] \text{ such that } x(t^-) = e_m, \\ &\qquad \text{and } N_{b_m} \text{ jumps at time } t\} \end{aligned} \tag{5}$$

follows (ϕ, T).

Proof: A detailed investigation of the differential equation (4) shows that the solution x of (4) is a Markov process with state space $\{e_1, \dots, e_p, 0\} \subset \mathbb{R}^p$ and infinitesimal generator given by (3). This fact specifically shows that the second equality in (5) is true. Furthermore, since $x(0)$ follows the probability distribution ϕ, the time to absorption of the stochastic process x into the absorbing state 0 follows the phase-type distribution (ϕ, T). This completes the proof of the proposition.

The stochastic differential equation (4) shall be used for describing phase-type recovery events in the proof of our first main result (see Theorem V.2). On the right-hand side of the stochastic differential equation (4), the first term represents the transitions between nonabsorbing states, while the second term represents the transitions into the absorbing state.

In order to derive stochastic differential equations for transmission events, we appropriately modify the second term in the stochastic differential equation (4) and derive a vectorial representation for *renewal sequences* (see, e.g., [6, Ch. 9]) whose interrenewal times follow a phase-type distribution.

Proposition IV.2: Let (ϕ, T) be a phase-type distribution. Let $e_\phi = \{e_\phi(t)\}_{t\geq 0}$ be independent and identically distributed random variables such that $e_\phi(t)$ follows the distribution ϕ for all $t \geq 0$. Consider the $\mathbb{R}^p$-valued stochastic differential equation

$$\begin{aligned} dx = {} & \sum_{m=1}^{p}\sum_{m'=1}^{p}(E_{m'm} - E_{mm})x\, dN_{T_{mm'}} \\ & + \sum_{m=1}^{p}(e_\phi(t)e_m^\top - E_{mm})x\, dN_{b_m} \end{aligned} \tag{6}$$

with a random initial state $x(0)$ following the distribution ϕ. Define $\tau_0 = 0$ and let $0 < \tau_1 < \tau_2 < \cdots$ be the (random) times at which $x(t^-) = e_m$ and the counter N_{b_m} jumps for some $m \in [p]$. Then, the increments $\{\tau_k - \tau_{k-1}\}_{k\in\mathbb{N}}$ are independent and identically distributed random variables following (ϕ, T).

Proof: Since the stochastic differential equation given in the proposition is equivalent to the one in Proposition IV.1 on the time interval $[0, \tau_1)$, the random variable τ_1 has the same probability distribution as the random variable $\boldsymbol{\rho}$ defined in (5) and, therefore, follows the phase-type distribution (ϕ, T) by Proposition IV.1. Furthermore, by the definition of τ_1, there exists an $m \in [p]$ such that $x(\tau_1) = (e_\phi(\tau_1)e_m^\top - E_{mm})x(\tau_1^-) + x(\tau_1^-) = e_\phi(\tau_1)$, which follows ϕ. Therefore, by the same argument as above, we see that the random increment $\tau_2 - \tau_1$ also follows (ϕ, T). An induction completes the proof. ■

Using Propositions IV.1 and IV.2 as the machinery for describing phase-type recovery and transmission events, in the next subsection, we present a set of vectorial stochastic differential equations for describing the whole GeNeSIS spreading model.

B. Vectorial Representation of the Generalized SIS Model

In this section, we use Propositions IV.1 and IV.2 to provide a vectorial representation of the GeNeSIS model under Assumption III.4. Let $A = [a_{ij}]_{i,j}$ be the adjacency matrix of the graph $\mathcal{G}$. Define the vectors b and d by (3) and

$$d = -R\mathbb{1}_q \in \mathbb{R}^q$$

respectively. For $\ell, \ell' \in [q]$, let $F_{\ell\ell'}$ denote the $q \times q$ matrix whose entries are all zeros, except for its (ℓ, ℓ')th entry being 1. Also, let f_ℓ denote the ℓth vector in the canonical basis of $\mathbb{R}^q$. Finally, for $i, j \in [n]$ and $\gamma > 0$, we let N_γ^{ij} and N_γ^i denote independent Poisson counters with rate γ. The next theorem is the first main result of this paper.

Theorem IV.3: For each $i \in [n]$ and $j \in \mathcal{N}_i$, let $e_\phi^{ji} = \{e_\phi^{ji}(t)\}_{t\geq 0}$ and $f_\psi^i = \{f_\psi^i(t)\}_{t\geq 0}$ be independent and identically distributed random variables such that

$$e_\phi^{ji}(t) \sim \phi, \quad f_\psi^i(t) \sim \psi$$

for all $t \geq 0$. Let x^{ji} and y^i be, respectively, the $\mathbb{R}^p$- and $\mathbb{R}^q$-valued stochastic processes satisfying the following stochastic differential equations:

$$\begin{aligned} dx^{ji} = & \sum_{m=1}^{p}\sum_{m'=1}^{p}(E_{m'm} - E_{mm})x^{ji}\, dN_{T_{mm'}}^{ji} \\ & + \sum_{m=1}^{p}(e_\phi^{ji} e_m^\top - E_{mm})x^{ji}\, dN_{b_m}^{ji} - x^{ji}\sum_{\ell=1}^{q} y_\ell^i\, dN_{d_\ell}^i \\ & + e_\phi^{ji}(1 - \mathbb{1}_q^\top y^i)\sum_{k=1}^{n} a_{ik}\sum_{m=1}^{p} x_m^{ik}\, dN_{b_m}^{ik}, \end{aligned} \tag{7}$$

$$\begin{aligned} dy^i = & \sum_{\ell=1}^{q}\sum_{\ell'=1}^{q}(F_{\ell'\ell} - F_{\ell\ell})y^i\, dN_{R_{\ell\ell'}}^i - y^i\sum_{\ell=1}^{q} y_\ell^i\, dN_{d_\ell}^i \\ & + f_\psi^i(1 - \mathbb{1}_q^\top y^i)\sum_{k=1}^{n} a_{ik}\sum_{m=1}^{p} x_m^{ik}\, dN_{b_m}^{ik}, \end{aligned} \tag{8}$$

where for an initially infected subset $\mathcal{V}_0 \subset [n]$, the initial conditions satisfy

$$\begin{cases} x^{ji}(0) \sim \phi,\ y^i(0) \sim \psi, & \text{if } i \in \mathcal{V}_0 \\ x^{ji}(0) = 0_p,\ y^i(0) = 0_q, & \text{otherwise.} \end{cases} \tag{9}$$

Then, the GeNeSIS model in Definition III.1 with transmission and recovery times following, respectively, phase-type distributions (ϕ, T) and (ψ, R) can be equivalently described as the family of stochastic processes $z = \{z_i\}_{i=1}^n$, where

$$z_i(t) = \mathbb{1}_q^\top y^i(t) \tag{10}$$

for all $t \geq 0$ and $i \in [n]$.

The representations of the GeNeSIS model as the set of stochastic differential equations (7) and (8) allow us to analyze the model via symbolic computations, as will be illustrated in Section V. Before proceeding to the proof of Theorem IV.3, we provide an intuitive explanation of the theorem. As shown in Corollary IV.5, the variable x^{ji} is related to spread of the infection from an infected node i to a susceptible node j, while y^i controls the recovery process of node i. Specifically, on the right-hand side of the differential equation (7), the first two terms have the same structure as in (6) and correspond to renewal sequences of transmissions. The third and fourth terms represent the recovery and infection of node i, respectively. Similarly, on the right-hand side of the differential equation (8), the first two terms correspond to the phase-type recovery and have almost the same structure as in (4), while the remaining last term is related to the infection of the node i.

In order to prove Theorem IV.3, we first state the following lemma.

Lemma IV.4: The following statements are true for all $i \in [n]$, $j \in \mathcal{N}_i$, and $t \geq 0$.

1) $\mathbb{1}_p^\top x^{ji}(t) = \mathbb{1}_q^\top y^i(t)$.
2) $x^{ji}(t) \in \{0_p, e_1, \dots, e_p\}$.
3) $y^i(t) \in \{0_q, f_1, \dots, f_q\}$.

Proof: To prove the first statement, fix $i \in [n]$ and $j \in \mathcal{N}_i$, and let

$$\epsilon = \mathbb{1}_p^\top x^{ji} - \mathbb{1}_q^\top y^i.$$

From (7) and (8), we obtain that

$$d\epsilon = \epsilon\sum_{\ell=1}^{q} y_\ell^i\, dN_{d_\ell}^i.$$

This equation implies that ϵ is constant over $[0, \infty)$ because $\epsilon(0) = 0$. Therefore, we have $\epsilon(t) = \epsilon(0) = 0$ for every $t \geq 0$, completing the proof of the first statement.

Let us prove the second and third statements. Notice that x^{ji} and y^i are piecewise constant since they are the solutions of the stochastic differential equations (7) and (8). Moreover, their values can change only when one of the Poisson counters in the stochastic differential equations jumps. Finally, if we let

$$U = \{0_p, e_1, \dots, e_p\}$$
$$V = \{0_q, f_1, \dots, f_q\}$$

then (9) shows $x^{ji}(0) \in U$ and $y^i(0) \in V$. Therefore, it is sufficient to show that the jump of any Poisson counter leaves the sets U and V invariant.

Let $t > 0$ be arbitrary and assume that $x^{ji}(t^-) \in U$ and $y^i(t^-) \in V$. The stochastic differential equations (7) and (8) have the following five different types of Poisson counters: $N^{ik}_{b_m}$, $N^i_{d_\ell}$, $N^{ji}_{T_{m\,m}}$, $N^i_{R_{\ell\ell}}$, and $N^{ji}_{b_m}$. Careful investigations of the stochastic differential equations (7) and (8) show that any of these counters leave the sets U and V invariant as follows. For example, when $N^{ik}_{b_m}$ jumps at time t, we have that

$$\begin{aligned} &x^{ji}(t) \\ &= x^{ji}(t^-) + e^{ji}_\phi(t)(1 - \mathbb{1}_q^\top y^i(t^-))a_{ik}x^{ik}_m(t^-) \\ &= \begin{cases} x^{ji}(t^-), & \text{if } \mathbb{1}_q^\top y^i(t^-) = 1, a_{ik} = 0, \text{or } x^{ik}_m(t^-) = 0 \\ e^{ji}_\phi(t), & \text{otherwise.} \end{cases} \end{aligned} \tag{11}$$

Also, we have

$$\begin{aligned} y^i(t) &= y^i(t^-) + f^i_\psi(t)(1 - \mathbb{1}_q^\top y^i(t^-))a_{ik}x^{ik}_m(t^-) \\ &= \begin{cases} y^i(t^-), & \text{if } \mathbb{1}_q^\top y^i(t^-) = 1, a_{ik} = 0, \text{or } x^{ik}_m(t^-) = 0 \\ f^i_\psi(t), & \text{otherwise.} \end{cases} \end{aligned} \tag{12}$$

Therefore, this jump leaves U and V invariant. Similarly, when $N^i_{d_\ell}$ jumps at time t, we obtain

$$\begin{aligned} x^{ji}(t) &= x^{ji}(t^-) - x^{ji}(t^-)y^i_\ell(t^-) \\ &= \begin{cases} 0, & \text{if } y^i_\ell(t^-) = 1 \\ x^{ji}(t^-), & \text{otherwise.} \end{cases} \end{aligned} \tag{13}$$

Hence, $x^{ji}(t) \in U$. We also have $y^i(t) \in V$ because

$$\begin{aligned} y^i(t) &= y^i(t^-) - y^i(t^-)y^i_\ell(t^-) \\ &= \begin{cases} 0, & \text{if } y^i_\ell(t^-) = 1 \\ y^i(t^-), & \text{otherwise.} \end{cases} \end{aligned} \tag{14}$$

The cases of other counters can be analyzed in a similar manner, and therefore, the proofs are omitted. ■

The next corollary of Lemma IV.4 clarifies the roles of the variables x^{ji} and y^i and the various Poisson counters in the stochastic differential equations (7) and (8).

Corollary IV.5: The following statements are true for every $i \in [n]$, $j \in \mathcal{N}_i$, and $t \geq 0$.

1) Node i attempts to infect node j at time t, if and only if, $x^{ji}_m(t^-) = 1$ and $N^{ji}_{b_m}$ jumps at time t for some $m \in [p]$.
2) Node i becomes susceptible at time t, if and only if, $y^i_\ell(t^-) = 1$ and $N^i_{d_\ell}$ jumps at time t for some $\ell \in [q]$.

Proof: From the proof of Lemma IV.4, the value of $z_i = \mathbb{1}_p^\top x^{ji} = \mathbb{1}_q^\top y^i$ can change from 0 to 1 when and only when a counter $N^{ik}_{b_m}$ jumps. Therefore, from (11) and (12), we see that node i becomes infected at time $t \geq 0$ if and only if $z_i(t^-) = 0$, node i is adjacent to node k, $x^{ik}_m(t^-) = 1$, and $N^{ik}_{b_m}$ jumps at time t for some $m \in [p]$. Therefore, node k attempts to infect node i at time t if and only if $x^{ik}_m(t^-) = 1$ and $N^{ik}_{b_m}$ jumps at time t for some $m \in [p]$, as desired.

Similarly, we see that the value of z^i can change from 1 to 0 when and only when a counter $N^i_{d_\ell}$ jumps. Therefore, (13) and (14) imply that node i becomes susceptible at time t if and only if $y^i_\ell(t^-) = 1$ and the counter $N^i_{d_\ell}$ jumps at time t for some $\ell \in [q]$, showing the second assertion of the corollary. ■

Now, we are ready to prove Theorem IV.3.

Proof of Theorem IV.3: We prove that the family of stochastic processes $z = \{z_i\}_{i=1}^n$, defined by (7)–(10), satisfies items a)–c) in Definition III.1, as well as Assumption III.4. Item a) is true by the given initial conditions. Let us prove item b). Assume that node i becomes infected at time $t \geq 0$; hence, we have that either $t = 0$ or $t > 0$. In this proof, we only consider the case $t = 0$, as the other case can be proved in a similar way. Let $\boldsymbol{\rho}$ be the earliest time at which $y^i(\boldsymbol{\rho}) = 0_q$. Then, on the time interval $[0, \boldsymbol{\rho})$, the stochastic differential equation (8) is equivalent to

$$dy^i = \sum_{\ell=1}^q \sum_{\ell'=1}^q (F_{\ell'\ell} - F_{\ell\ell})y^i \, dN^i_{R_{\ell\ell}} - \sum_{\ell=1}^q F_{\ell\ell}y^i \, dN^i_{d_\ell}$$

since $y^i y^i_\ell = F_{\ell\ell}y^i$. Moreover, the vector $y^i(0)$ follows the distribution ψ. Therefore, by Proposition IV.1, we see that $\boldsymbol{\rho}$ follows the phase-type distribution (ψ, R), proving item b) in Definition III.1 under Assumption III.4 for $t = 0$. A similar discussion using Proposition IV.2 shows that the family of stochastic processes z satisfies item c) in Definition III.1 under Assumption III.4. This completes the proof of the theorem. ■

Before presenting our analysis of the decay rate in the next section, we state another corollary of Lemma IV.4.

Corollary IV.6: The family of stochastic processes

$$\xi = \{x^{ji}, y^i\}_{i\in[n], j\in\mathcal{N}_i} \tag{15}$$

where x^{ji} and y^i are the solutions of the stochastic differential equations (7) and (8), is a Markov process. Moreover, the states of ξ are all transient except for the absorbing state α at which $x^{ji} = 0$ and $y^i = 0$ for all $i \in [n]$ and $j \in \mathcal{N}_i$. Furthermore, the size of the state space of ξ equals

$$N_\xi = \prod_{i=1}^n (1 + p^{|\mathcal{N}_i|}q)$$

where $|\mathcal{N}_i|$ denotes the size of the neighbor set $\mathcal{N}_i$.

Proof: A careful investigation of the proof of Lemma IV.4 shows the first and second claims. The third claim is an immediate consequence from the constraint $\mathbb{1}_p^\top x^{ji}(t) = \mathbb{1}_q^\top y^i(t)$ that was proved in Lemma IV.4. ■

V. DECAY RATE ANALYSIS

In this section, we use our previous results to bound the exponential decay rate of the GeNeSIS model under Assumption III.4. We begin by presenting a characterization of the decay rate in terms of the eigenvalues of a matrix, whose size grows exponentially with respect to the model parameters. To overcome the difficulty of computing the eigenvalues of a very large matrix, we then present an alternative bound on the decay rate based on the representation of the GeNeSIS model as the stochastic differential equations (7) and (8).

Throughout this section, we consider the GeNeSIS model with transmission and recovery times following, respectively, phase-type distributions (ϕ, T) and (ψ, R) (i.e., satisfying Assumption III.4). The following proposition illustrates the computational difficulty in computing the exponential decay rate of the GeNeSIS model.

Proposition V.1: Let $Q \in \mathbb{R}^{N_\xi \times N_\xi}$ be the transition rate matrix of the Markov process ξ (see (15) for the definition of ξ), and let $r < 0$ be the maximum real part of the nonzero eigenvalues of Q. Then, the exponential decay rate of the GeNeSIS model is given by

$$\lambda = -r.$$

Proof: Since $\xi(t) \neq \alpha$ if and only if at least one node is infected at time t, we have the inequality

$$E[z_i(t)] \leq P(\xi(t) \neq \alpha) \leq \sum_{i=1}^{n} E[z_i(t)]$$

for all $i \in [n]$ and $t \geq 0$. Therefore, the exponential decay rate of the GeNeSIS model is determined by that of the function $P(\xi(t) \neq \alpha)$ of t. By Corollary IV.6, a basic argument on Markov processes (see [44] for the case of exponential transmission and recover times) shows that: 1) if $\lambda < -r$, there exists a constant $C > 0$ such that $P(\xi(t) \neq \alpha) \leq Ce^{-\lambda t}$ for all t and any initial state $\xi(0)$; and 2) if $\lambda > -r$, there exists an initial state of ξ such that the function $P(\xi(t) \neq \alpha)$ cannot be bounded from above by the exponential function $Ce^{-\lambda t}$ for any value of C. These observations immediately prove the claim of the proposition. ■

Proposition V.1 yields the exact value of the decay rate, since the proposition uses the transition rate matrix of the whole Markov process ξ that exactly describes the GeNeSIS model. However, Proposition V.1 is not easily applicable in practice because the dimension N_ξ of the matrix Q grows exponentially as

$$N_\xi \geq \prod_{i=1}^{n} (p^{|\mathcal{N}_i|} q) = p^{2m} q^n \tag{16}$$

where m denotes the number of the edges in the network. The following theorem overcomes this computational difficulty by providing a lower bound on the growth rate in terms of the eigenvalues of a matrix whose size grows *linearly* with respect to the parameters in the GeNeSIS model.

Theorem V.2: Define the $(npq) \times (npq)$ matrix

$$\mathcal{A} = (\phi b^\top) \otimes A \otimes (\psi \mathbb{1}_q^\top) + I_{np} \otimes R^\top + (T^\top + \phi b^\top) \otimes I_{nq} \tag{17}$$

where A is the adjacency matrix of the graph $\mathcal{G}$ and the vector b is defined in (3). Then, we have

$$\lambda \geq -\eta(\mathcal{A})$$

where $\eta(\mathcal{A})$ is the spectral abscissa of $\mathcal{A}$.

Before providing a proof of Theorem V.2, we below present a series of corollaries of the theorem. The proofs of the corollaries are straightforward and, therefore, omitted. The first corollary gives a bound on the decay rate of the GeNeSIS model with exponential transmission times and phase-type recovery times.

Corollary V.3: Assume that the transmission times follow an exponential distribution with mean $1/\beta$. Define the $(nq) \times (nq)$ matrix

$$\mathcal{A}_\beta = \beta A \otimes (\psi \mathbb{1}_q^\top) + I_n \otimes R^\top.$$

Then, the decay rate satisfies $\lambda \geq -\eta(\mathcal{A}_\beta)$.

The next corollary deals with the dual case with phase-type transmission times and exponential recovery times.

Corollary V.4: Assume that the recovery times follow an exponential distribution with mean $1/\delta$. Define the $(np) \times (np)$ matrix

$$\mathcal{A}_\delta = (\phi b^\top) \otimes A + (T^\top + \phi b^\top) \otimes I_n - \delta I_{np}.$$

Then, the decay rate satisfies $\lambda \geq -\eta(\mathcal{A}_\delta)$.

As the special case of Theorem V.2, as well as Corollaries V.3 and V.4, we can prove the following bound on the decay rate of the standard SIS model.

Corollary V.5 (see[12] and [37]): Assume that the transmission and recovery times follow exponential distributions with means $1/\beta$ and $1/\delta$, respectively. Define the $n \times n$ matrix

$$\mathcal{A}_{\beta,\delta} = \beta A - \delta I_n.$$

Then, the decay rate satisfies $\lambda \geq -\eta(\mathcal{A}_{\beta,\delta})$.

The above corollaries suggest an intuitive understanding of the terms in the matrix $\mathcal{A}$ defined in (17). Comparing the expressions of the matrices $\mathcal{A}_\delta$ and $\mathcal{A}_{\beta,\delta}$, we see that the role of the exponential transmission rate β is played by the $p \times p$ matrix $\phi b^\top$ in the case of phase-type transmission times. On the other hand, the second term of the matrix $\mathcal{A}_\delta$, namely, $(T^\top + \phi b^\top) \otimes I_n$, can be understood as a correction term that arises independently of the topology of the network. Similarly, comparing the matrices $\mathcal{A}_\beta$ and $\mathcal{A}_{\beta,\delta}$, we see that the second term $I_n \otimes R^\top$ of the matrix $\mathcal{A}_\beta$ represents the effect of phase-type recoveries. On the other hand, we can understand the matrix $\psi \mathbb{1}_q^\top$ as a correction term resulting from using phase-type recovery times.

We now give the proof of Theorem V.2.

Proof of Theorem V.2: Combining (7) and (8), we obtain the following $\mathbb{R}^{p+q}$-valued stochastic differential equation:

$$\begin{aligned} d\begin{bmatrix} x^{ji} \\ y^i \end{bmatrix} &= \sum_{m=1}^{p} \sum_{m'=1}^{p} \begin{bmatrix} (E_{m'm} - E_{mm}) x^{ji} \\ 0_{nq} \end{bmatrix} dN_{T_{mm'}}^{ji} \\ &\quad + \sum_{m=1}^{p} \begin{bmatrix} (e_\phi^{ji} e_m^\top - E_{mm}) x^{ji} \\ 0_{nq} \end{bmatrix} dN_{b_m}^{ji} \\ &\quad + \sum_{\ell=1}^{q} \sum_{\ell'=1}^{q} \begin{bmatrix} 0_{np} \\ (F_{\ell'\ell} - F_{\ell\ell}) y^i \end{bmatrix} dN_{R_{\ell\ell'}}^{i} \\ &\quad + \sum_{\ell=1}^{q} \begin{bmatrix} -x^{ji} y_\ell^i \\ -y^i y_\ell^i \end{bmatrix} dN_{d_\ell}^{i} \\ &\quad + \sum_{k=1}^{n} \sum_{m=1}^{p} a_{ik} \begin{bmatrix} e_\phi^{ji} (1 - \mathbb{1}_q^\top y^i) x_m^{ik} \\ f_\psi^i (1 - \mathbb{1}_q^\top y^i) x_m^{ik} \end{bmatrix} dN_{b_m}^{ik}. \end{aligned}$$

Then, we apply Itô's formula in Lemma II.1 using the function

$$g\left(\begin{bmatrix} x^{ji} \\ y^i \end{bmatrix}\right) = w^{ji} = x^{ji} \otimes y^i$$

to obtain the following $\mathbb{R}^{pq}$-valued stochastic differential equation (after tedious, but simple, algebraic manipulations):

$$\begin{aligned} dw^{ji} ={}& \sum_{m=1}^{p}\sum_{m'=1}^{p}\big((E_{m'm} - E_{mm}) \otimes I_q\big) w^{ji}\, dN^{ji}_{T_{mm'}} \\ &+ \sum_{m=1}^{p}\big((e^{ji}_{\phi} e_m^\top - E_{mm}) \otimes I_q\big) w^{ji}\, dN^{ji}_{b_m} \\ &+ \sum_{\ell=1}^{q}\sum_{\ell'=1}^{q}\big(I_p \otimes (F_{\ell'\ell} - F_{\ell\ell})\big) w^{ji}\, dN^{i}_{R_{\ell\ell'}} \\ &+ \sum_{\ell=1}^{q}(-y^i_\ell w^{ji})\, dN^i_{d_\ell} \\ &+ \sum_{k=1}^{n}\sum_{m=1}^{p} a_{ik}(e^{ji}_\phi \otimes f^i_\psi)(1 - \mathbb{1}_{pq}^\top w^{ji})(e_m^\top \otimes \mathbb{1}_q^\top) w^{ik}\, dN^{ik}_{b_m}. \end{aligned} \tag{18}$$

For brevity, we omit the details of this derivation. Define

$$\omega_{ji}(t) = E[w^{ji}(t)]$$

for $t \geq 0$. Then, using Lemma II.2, from (18), we can derive the $\mathbb{R}^{pq}$-valued differential equation

$$\begin{aligned} \frac{d\omega_{ji}}{dt} ={}& \Big[(T^\top + B) \otimes I_q + (\phi b^\top - B) \otimes I_q \\ &+ \big(I_p \otimes (R^\top + D)\big)\Big]\omega_{ji} - (I_p \otimes D)\omega_{ji} \\ &+ (\phi \otimes \psi)(e_m^\top \otimes \mathbb{1}_q^\top) \sum_{m=1}^{p} b_m \sum_{k=1}^{n}(a_{ik}\omega_{ik}) - \epsilon_{ji} \end{aligned} \tag{19}$$

where B and D are the $n \times n$ diagonal matrices having the diagonals $b_1, \dots, b_n$ and $d_1, \dots, d_n$, respectively, and the $\mathbb{R}^{pq}$-valued function ϵ_{ji} is defined by

$$\epsilon_{ji}(t) = \sum_{k=1}^{n}\sum_{m=1}^{p} b_m a_{ik}(\phi \otimes \psi) E[\mathbb{1}_{pq}^\top w^{ji}(t)(e_m^\top \otimes \mathbb{1}_q^\top) w^{ik}(t)] \tag{20}$$

for every $t \geq 0$.

Now, for every $i \in [n]$, we take an arbitrary $j_i \in \mathcal{N}_i$. We then define the function ω_i by $\omega_i = \omega_{j_i i}$, as well as the $\mathbb{R}^{npq}$-valued function

$$\omega = \mathrm{col}(\omega_1, \dots, \omega_n).$$

Notice that, from (18), for each $i \in [n]$, all the stochastic processes in the set $\{w^{ji}\}_{j \in \mathcal{N}_i}$ follow the same stochastic differential equation and, therefore, present the same probability distribution. This implies that $\omega_i = \omega_{ji}$ for every $j \in \mathcal{N}_i$. Therefore, we can rewrite the last summation appearing in (19) as $\sum_{k=1}^n a_{ik}\omega_{ik} = \sum_{k=1}^n a_{ik}\omega_k = (A_i \otimes I_{pq})\omega$, where A_i denotes the ith row of the adjacency matrix A. Then, from (11), it follows that

$$\begin{aligned} \frac{d\omega_i}{dt} ={}& \big(T^\top \otimes I_q + I_p \otimes R^\top + (\phi b^\top) \otimes I_q\big)\omega_i \\ &+ \big(A_i \otimes (\phi b^\top) \otimes (\psi \mathbb{1}_q^\top)\big)\omega - \epsilon_{j_i i}. \end{aligned}$$

Defining

$$\epsilon = \mathrm{col}(\epsilon_{j_1 1}, \dots, \epsilon_{j_n n})$$

we obtain the differential equation

$$\frac{d\omega}{dt} = \mathcal{A}'\omega - \epsilon$$

for the matrix $\mathcal{A}' = I_n \otimes (T^\top \otimes I_q + I_p \otimes R^\top + (\phi b^\top) \otimes I_q) + A \otimes (\phi b^\top) \otimes (\psi \mathbb{1}_q^\top)$.

Since $\mathcal{A}'$ is Metzler, we have that $e^{\mathcal{A}'t} \geq 0$ for every $t \geq 0$. Also, since both $x^{ji}(t)$ and $y^i(t)$ are nonnegative for all $i \in [n]$, $j \in \mathcal{N}_i$, and $t \geq 0$, we have that $\epsilon(t) \geq 0$ for every $t \geq 0$. Therefore, it follows that

$$\omega(t) = e^{\mathcal{A}'t}\omega(0) - \int_0^t e^{\mathcal{A}'(t-\tau)}\epsilon(\tau)\, d\tau \leq e^{\mathcal{A}'t}\omega(0).$$

This inequality implies that $\omega(t)$ converges to zero as $t \to \infty$ with a decay rate at least $-\eta(\mathcal{A}')$ since $\omega(t) \geq 0$ for all $t \geq 0$. On the other hand, for each $i \in [n]$ and $j \in \mathcal{N}_i$, we have

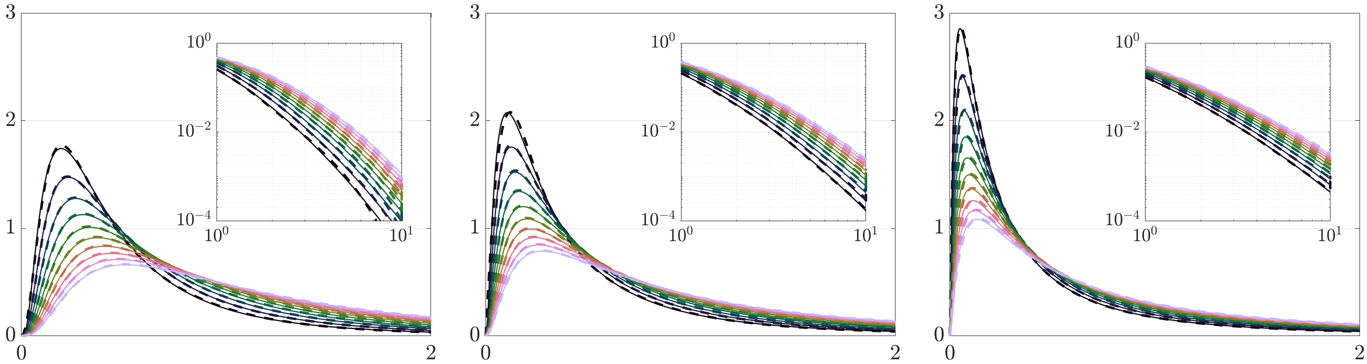
$$\mathbb{1}_{pq}^\top w^{ji}(t) = (\mathbb{1}_p^\top x^{ji}(t))(\mathbb{1}_q^\top y^i(t)) = \mathbb{1}_q^\top y^i(t) = z_i(t)$$

from Lemma IV.4. Therefore, we have $E[z_i(t)] = \mathbb{1}_{pq}^\top \omega_{ji}(t)$, which shows the exponentially fast convergence of $E[z_i(t)]$ toward zero with a decay rate at least $-\eta(\mathcal{A}')$. Furthermore, using the fact that two Kronecker products $F \otimes G$ and $G \otimes F$ are similar for square matrices F and G, we can show that the matrices $\mathcal{A}'$ and $\mathcal{A}$ are similar. This completes the proof of the theorem. ■

Remark V.6: Unlike the necessary and sufficient condition in Proposition V.1, the condition in Theorem V.2 is only sufficient. This conservatism arises from ignoring the higher order term ϵ_{ji} in (20). The inclusion of these higher order terms into the analysis (see, e.g., [31] and [39]) would allow us to reduce the conservatism, at the cost of increasing the dimension of the matrix $\mathcal{A}$.

VI. NUMERICAL SIMULATIONS

In this section, we illustrate the effectiveness of our results with numerical simulations in a real social network having $n = 247$ nodes and 940 edges. We focus on log-normal transmission and recovery times, which are observed in empirical studies, including information spread on online social networks [11], [43] and human epidemiology [22], [26]. In our simulations, we illustrate the effect of using exponential distributions to model transmission and recovery times that, in reality, follow log-normal distributions. In particular, we analyze how using standard Markovian models with exponential rates induce errors in the computation of the decay rate. Furthermore, we are also interested in how the variances of log-normal distributions, which cannot be incorporated into the standard Markovian



(a) (b) (c)

Fig. 1. Approximations by phase-type distributions. Solid: Probability density functions of the log-normal distributions for μ in the range $[0.5 : 0.1 : 1.5]$. Dashed: Probability density functions of phase-type distributions. The darker the colors of the plots, the smaller the values of μ. (a) Log-normal distributions (solid lines) with mean μ and variance μ^2, and their phase-type approximations (dashed lines). (b) Log-normal distributions (solid lines) with mean μ and variance $2\mu^2$, and their phase-type approximations (dashed lines). (c) Log-normal distributions (solid lines) with mean μ and variance $4\mu^2$, and their phase-type approximations (dashed lines).

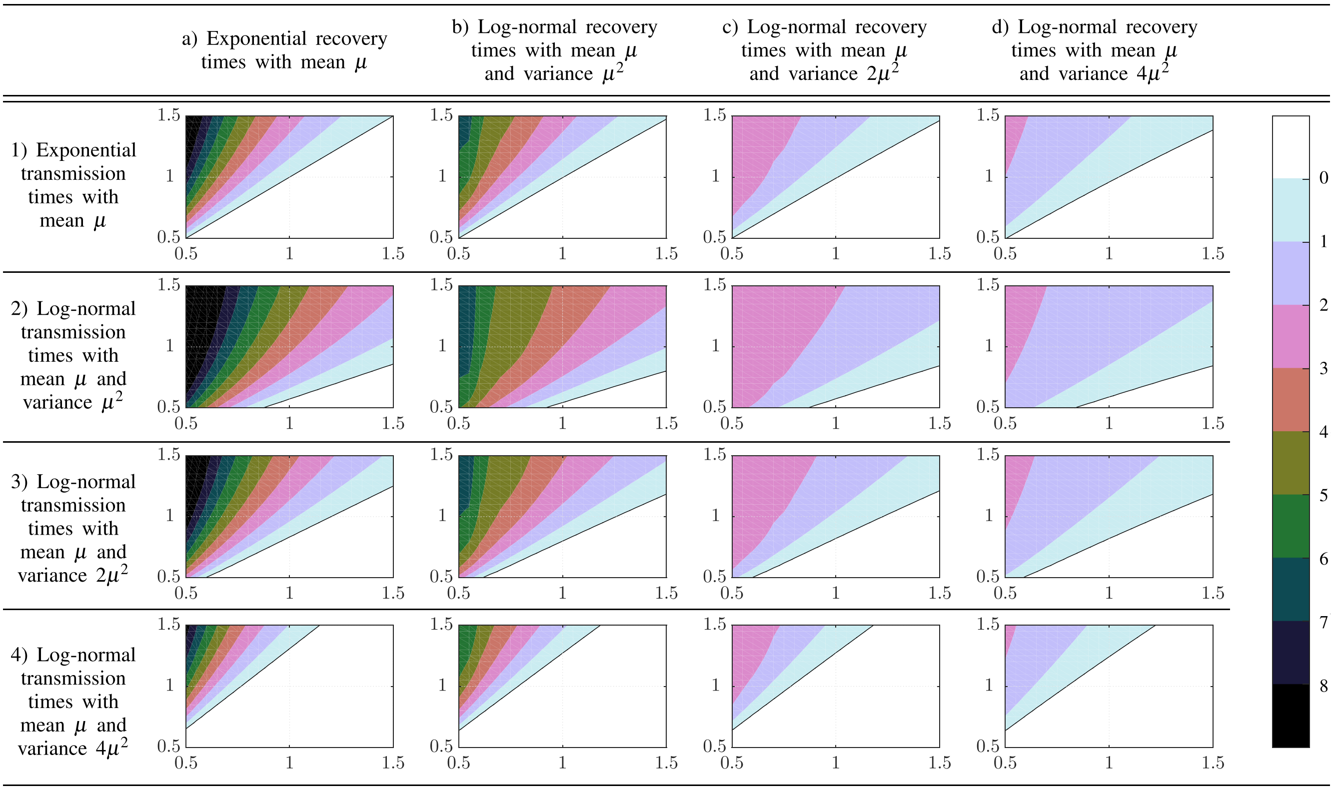


Fig. 2. Lower bound $-\eta(\mathcal{A})$ on the exponential decay rate of the generalized SIS model. Horizontal axes: μ. Vertical axes: μ divided by the spectral radius of the graph. The white region corresponds to unstable epidemics; the darker the region, the faster the epidemics converges toward the infection-free equilibrium. (a) Exponential recovery times with mean μ. (b) Log-normal recovery times with mean μ and variance μ^2. (c) Log-normal recovery times with mean μ and variance $2\mu^2$. (d) Log-normal recovery times with mean μ and variance $4\mu^2$.

model, affect the decay rates. For this purpose, we use the following four distributions to model transmission and recovery times in the GeNeSIS model: (*i*) the exponential distribution with mean μ (and, hence, variance μ^2); (*ii*) the log-normal distribution with mean μ and variance μ^2; (*iii*) the log-normal distribution with mean μ and variance $2\mu^2$; and (*iv*) the log-normal distribution with mean μ and variance $4\mu^2$.

In order to analyze the decay rate of the GeNeSIS model whose transmission and recovery times follow one of these four distributions, we first approximate the three log-normal distributions in (*ii*)–(*iv*) using phase-type distributions (as described in Section III) having $p = 10$ phases. Fig. 1 shows the probability density functions of the (exact) log-normal distributions, as well as the fitted phase-type distributions, when the value of the parameter μ is in the range $[0.5 : 0.1 : 1.5]$. We notice that, since the inequality (16) shows that the size of the exact state transition matrix satisfies $N_\xi > 10^{2127}$, it is not practical to use Proposition V.1 to compute the exact decay rate.

Using the proposed phase-type distributions, we apply Theorem V.2 to analyze the decay rate of the GeNeSIS model when the transmission/recovery times follow one of the four distributions described above. We compute the decay rate $-\eta(\mathcal{A})$ for each one of the possible 16 combinations of transmission/recovery distributions when μ is in the range $[0.5 : 0.05 : 1.5]$. In the subfigures of Fig. 2, we include contour plots of the decay rates for these 16 cases. In the figure, the values of the decay rates are indicated by different colors. The darker the color, the faster the epidemics converges to the infection-free equilibrium.

We remark that, even though the four distributions used in our simulations have the same mean, the resulting GeNeSIS models exhibit different decay rates. We can furthermore observe that the variances of the distributions used to model transmission and recovery times can dramatically affect the decay rate of the GeNeSIS model, as was previously indicated by numerical simulations [45] and mean-field approximations [5]. For a fixed recovery distribution (i.e., for a fixed column in the table in Fig. 2), the colored region (indicated by black solid lines) shrinks as we increase the variance of the log-normal distribution modeling the transmission times (see rows 2–4 in Fig. 2). From this observation, we see that the heavier the tail of the transmission distribution, the slower the extinction of the spreading process, as was numerically confirmed in [45]. We also observe that for a fixed transmission distribution (i.e., for a fixed row in the table), the colored region remains almost unaltered as we increase the variance of the distribution modeling the recovery time, confirming the validity of the mean-field analysis in [5] for the case of exponential transmission times. We can furthermore observe that the exponential decay rate $-\eta(\mathcal{A})$ increases more abruptly inside the region as we decrease this variance as indicated by steeper gradients. The above observations illustrate the effectiveness of the proposed framework for analyzing the decay rate of epidemics in networks with non-Poissonian transmission and/or recovery distributions.

VII. CONCLUSION

In this paper, we have analyzed the dynamics of an SIS model of spreading over arbitrary networks with phase-type transmission and recovery times. Since phase-type distributions form a dense family in the space of positive-valued distributions, our results allow us to theoretically analyze arbitrary transmission and recovery times within an arbitrary accuracy. In this context, we have derived conditions for this generalized spreading model to converge toward the infection-free equilibrium (i.e., to eradicate the spread) with a given exponential decay rate. We have specifically provided a *transient* analysis of the *stochastic* spreading dynamics over arbitrary networks without relying on mean-field approximations. Our results illustrate that the particular shape of the transmission/recovery distribution heavily influences the exponential decay rate of the convergence toward the infection-free equilibrium. Through numerical simulations, we have specifically observed that our results allow us to theoretically confirm some observations previously obtained by numerical simulations and mean-field approximations.

A possible direction for future research is considering time-varying (temporal) networks [14], [24], in which transmission and recovery events follow non-Poissonian distributions. Another interesting research direction is developing an optimal resource allocation strategy for non-Markovian epidemic spreading processes. Although we can find in the literature various research efforts [1], [13], [37], [46] for designing containment methodologies for networked epidemic spreading processes, many of them are based on decay rates that are derived under the Markovian assumption on transmission and recovery events. In this direction, it is of practical interest to investigate how the non-Markovianity of spreading dynamics alters the optimal allocation strategies that have been investigated in the literature.

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