Analysis and Distributed Control of Periodic Epidemic Processes

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***Abstract*—This article studies epidemic processes over discrete-time periodic time-varying networks. We focus on the susceptible-infected-susceptible (SIS) model that accounts for a (possibly) mutating virus. We say that an agent is in the disease-free state if it is not infected by the virus. Our objective is to devise a control strategy which ensures that all agents in a network exponentially (respectively asymptotically) converge to the disease-free equilibrium (DFE). Toward this end, we first provide 1) sufficient conditions for exponential (respectively, asymptotic) convergence to the DFE and 2) a necessary and sufficient condition for asymptotic convergence to the DFE. The sufficient condition for global exponential stability (GES) [respectively global asymptotic stability (GAS)] of the DFE is in terms of the joint spectral radius of a set of suitably defined matrices, whereas the necessary and sufficient condition for GAS of the DFE involves the spectral radius of an appropriately defined product of matrices. Subsequently, we leverage the stability results in order to design a distributed control strategy for eradicating the epidemic.**

***Index Terms*—Discrete-time networks, distributed control strategy, epidemic processes, global asymptotic stability (GAS), global exponential stability (GES), susceptible-infected-susceptible (SIS) models, time-varying systems.**

I. Introduction

SPREADING processes, like epidemics, propagation of (mis)information in social networks, etc., often have significant consequences. For instance, the outbreak of Severe Acute Respiratory Syndrome (SARS) in 2003 in Hong Kong resulted in 286 deaths [1]. More recently, the increasing instances of coronavirus infections have severely affected normal life across multiple continents [2]. It is known that certain epidemics exhibit yearly seasonal patterns, such as meningococcal meningitis in Western Africa, which typically occurs between January and May of each year [3]. Furthermore, in the modern world, the networks that people have often recur with some periodicity, for example, professional networks during the day, personal networks at other times, and transportation networks. In this article, we will consider seasonal epidemic processes in periodic time-varying networks, and will be interested in the following natural question: how can the epidemic be eradicated? Answering this question is a two-step process: First, we need to know under what conditions do all the agents in a population become healthy. Second, given the knowledge of the convergence conditions, what measures can be adopted in order to guarantee that the epidemic is eradicated.

Modeling and analysis of spreading processes has attracted the attention of researchers across a wide spectrum ranging from mathematical epidemiology [4], [5] and physics [6] to the social sciences [7]. The primary objective behind these research efforts is to better understand *how* various diseases can spread through a population, which could then inform effective methods of management and control of the disease. In this pursuit, various models have been studied in the literature; here, we concern ourselves with susceptible-infected-susceptible (SIS) models.

In an SIS model, an agent is either in the susceptible or infected state. A healthy agent can, as a consequence of its neighbors being infected, become infected with some infection rate β. An infected agent can be cured, with a healing rate δ, thereby returning to the susceptible state. It is assumed that there is no entry into or exit from the population, that is, the number of agents in the network remains fixed [8], [9].

A. Related Works

The analysis of SIS epidemic models has attracted the attention of researchers over the last several decades; for the continuous-time case, see [6], [10], and [11], whereas for the discrete-time case, see [8], [12]–[14]. In this article, we consider a discrete-time setup and, therefore, mainly review the discrete-time literature. In this context, for the case of time-invariant graphs, the authors in [12] provide an epidemic threshold for the model equal to the inverse of the maximum eigenvalue of the matrix representing the graph structure. However, the result in [12] is restricted to homogeneous virus spread, i.e., the infection and healing rates of each agent are identical. The result in [12] has been further strengthened by accounting for directed and weighted graphs in [13]. In particular, the authors in [13, Theor. 5] establish that as long as the spectral radius of an appropriately defined matrix is strictly less than 1, the

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epidemic becomes extinct. However, note that the time-invariant discrete-time SIS model in [13] is different from the one in [14]. The disease-free equilibrium (DFE) and the nondisease free equilibrium (NDFE)[1] of several models have been studied in [8]. Moreover, the authors in [8] also provide existence, stability, and uniqueness conditions for the NDFE. A necessary and sufficient condition, in terms of the spectral radius of a matrix that is a function of the graph structure and the infection and healing rates, for global asymptotic stability (GAS) of the DFE has been established in [14]. To the best of our knowledge, for discrete-time time-invariant SIS models as in [14], a sufficient condition for global exponential stability (GES) of the DFE is missing in the existing literature.

The models in [8] and [12]–[14] suffer from the following limitation: they cannot account for highly complex settings, in particular, one where the interconnection between agents in a population (possibly) changes with time, for instance, real-world social and human-interaction networks. Such a scenario imposes a time-varying topology on the underlying graph, thus motivating the need for *time-varying* SIS models.

The interest in SIS models with time-varying topology is rather recent; for continuous-time setting, see [9], [16], and [17], while for discrete-time setting, see [18] and [19]. In the context of switched SIS models (both continuous-time and discrete-time), a sufficient condition for local exponential stability (respectively instability) of the DFE is provided in [19]. It turns out that the condition in [19, Theor. 2.2] implies GAS of the DFE for a continuous-time switched SIS model; see [16]. Following up on the work in [16] and [20], a switching susceptible-infected (SI) model is studied, albeit under the assumption of complete connectivity. In a similar vein, for a subset of random graphs, sufficient conditions for almost sure GES of the DFE are provided in [21].

In the continuous-time setting, for heterogeneous virus spread and directed graphs, under assumptions that the topology of the underlying graph does not change *too quickly*, sufficient conditions for exponential convergence to the DFE are provided in [9, Theor. 2]. The setup considered in this article differs from the aforementioned works in the following sense: First, we consider periodic discrete-time time-varying SIS models. That is, SIS models where the number of agents remains fixed, but the interconnection between them (possibly) changes with time. Second, we account for mutating viruses, that is, even the healing (respectively infection) rate of each agent can change with time. Finally, the interconnections between agents and the healing (respectively infection) rates repeat after every period.

As a first step toward designing a control strategy for eradicating epidemics in the aforementioned setup, we ask the following questions: First, what are the sufficient conditions for the DFE to be GES? Second, what are necessary and sufficient conditions for the DFE to be GAS? To the best of our knowledge, both the stated questions remain open. This article aims to answer these questions. The second step essentially involves comprehending how the dynamics of the spreading process can be *controlled* so as to ensure that all agents converge to the DFE exponentially (respectively asymptotically) fast. In this regard, various strategies have been proposed in the literature; see, for instance, [15] and [22], whereas for a survey of this subtopic, see [23]. In particular, the authors in [24] consider a directed, network comprising heterogeneous agents and propose a fully distributed Alternating Direction Method of Multipliers (ADMM) algorithm that allows for local computation of optimal investment required to boost the healing rate at each node. However, the ADMM algorithm in [24] involves heavy communication overhead, since every agent needs to share with its neighbors their local estimate of the full network. Overcoming the drawbacks with the ADMM algorithm in [24] and [25] proposes distributed discrete-time nonlinear algorithms for handling a class of distributed resource allocation problems. A decentralized algorithm that involves disconnecting nodes and increasing the healing rate subject to resource constraints has been proposed in [26]. This algorithm also accounts for control sparsity, that is, control resources can be allocated only to a subset of nodes, and not necessarily to the whole network. A distributed algorithm that, given resource limitations, ensures the eradication of an epidemic with a *specified* rate has been recently provided in [27]. The distributed control algorithms provided in [15], [24]–[27], and the results in [22] and [23] are for time-invariant SIS models. Similar techniques for the more general setting of the discrete-time, periodic, time-varying, mutating SIS model are, as far as we know, not available in the literature. This article closes this gap.

B. Contributions

The central premise of this article is: given that a seasonal epidemic is prevalent within a population with time-varying interconnection between the agents, how do we eradicate it? We answer this question in the following manner. First, we find conditions which ensure that regardless of the initial state of an agent, i.e., healthy or sick, *all* agents converge to the healthy state exponentially (respectively asymptotically) fast. Second, with the knowledge of the aforementioned conditions in hand, we show that by sufficiently boosting the healing rate of each agent, the epidemic can be eradicated in exponential (respectively asymptotic) time. More specifically, under assumptions of periodicity, we show that the following hold.

1) The joint spectral radius of an appropriately defined set of matrices being strictly less than 1 ensures GES of the DFE; see Theorem 1.
2) The joint spectral radius of an appropriately defined set of matrices being no greater than 1 ensures GAS of the DFE; see Theorem 2. A less restrictive condition that endows the DFE with the GES (respectively GAS) property requires the spectral radius of a suitably defined product of matrices to be strictly less than (respectively not greater than) 1; see Corollary 1 (respectively Corollary 3). In particular, for discrete-time SIS *time-invariant* models, we establish that the spectral radius of a suitably defined *matrix* being strictly less than 1 implies GES of the DFE; see Proposition 3.

[1]The NDFE is an equilibrium where the infection persists in the network, and is also referred to as the *endemic* equilibrium elsewhere in the literature; see for instance [14] and [15].

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3) The spectral radius, of a suitably defined product of matrices, being no greater than 1 is a necessary and sufficient condition for GAS of the DFE; see Theorem 3.
4) A novel distributed control strategy exponentially (respectively asymptotically) stabilizes the DFE; see Theorem 4 (respectively Corollary 4).

C. Outline

This article unfolds as follows: we conclude the present section by listing all the notation used in the sequel. The problems under investigation, and hence, the main objectives of this article, are stated in Section II, whereas the background material needed for developing the main results are provided in Section III. We present conditions for exponential convergence (respectively asymptotic convergence) to the DFE in Section IV (respectively Section V). We propose the distributed control strategy in Section VI. The simulations are provided in Section VII. Finally, we summarize this article, and highlight certain problems that could be of possible interest for future work in Section VIII.

D. Notation

Let $\mathbb{R}$ (respectively $\mathbb{Z}_{\geq 0}$) denote the set of real numbers (respectively nonnegative integers). We denote by $\mathbb{Z}_+$ the set of positive integers. For a pair of integers $a, b \in \mathbb{Z}_+$, $a \bmod b$ indicates a modulo b. For any positive integer n, we have $[n] = \{1, \ldots, n\}$ and $[n]^- = \{0, \ldots, n-1\}$. Given a matrix $A \in \mathbb{R}^{n\times n}$, a_{ij} denotes the entry corresponding to the ith row and jth column; and $\rho(A)$ denotes its spectral radius. Given a matrix A, supposing its spectrum is real, $\lambda_{\min}(A)$ (respectively $\lambda_{\max}(A)$) denotes the minimum (respectively maximum) eigenvalue of A. A diagonal matrix is denoted $\operatorname{diag}(\cdot)$. Given a vector $x \in \mathbb{R}^n$, its transpose is denoted $x^\top$ and its average $\bar{x} := \frac{1}{n}\sum_{i=1}^n x_i$. The Euclidean norm is denoted by $\|\cdot\|$, whereas the infinity norm is indicated by $\|\cdot\|_\infty$. Given a sequence of matrices $A(k+p)$, $A(k+p-1)$, …, $A(k+1)$, $A(k)$, their product $A_{k+p+1:k}$ is defined as $A_{k+p+1:k} = A(k+p)\cdot A(k+p-1)\cdots A(k+1)\cdot A(k)$. Given a matrix A, $A \prec 0$ (respectively $A \preccurlyeq 0$) indicates that A is negative definite (respectively negative semidefinite), whereas $A \succ 0$ (respectively $A \succcurlyeq 0$) indicates that A is positive definite (respectively positive semidefinite).

II. Problem Formulation

Consider a possibly time-varying epidemic network of n agents, where the interpretation of *time-varying* is as follows: the set of agents remains fixed, whereas the interconnections among the agents could possibly be time-varying. Due to the possibly time-varying nature of the interconnections, the healing rate and infection rate of each agent might also be time-dependent, that is, mutating. Thus, the continuous-time dynamics of each agent can be represented as follows:

$$\dot{x}_i(t) = (1-x_i)\beta_i(t)\sum_{j=1}^n a_{ij}(t)x_j - \delta_i(t)x_i(t) \tag{1}$$

where i represents the ith agent, x_i is the infection level, and for every $t \in \mathbb{R}$, $\beta_i(t) \geq 0$ (respectively $\delta_i(t) \geq 0$) denotes the infection (respectively healing) rate. Assuming there exists a directed edge from agent j to agent i at time t, the corresponding edge weight is denoted by $a_{ij}(t) > 0$. If $a_{ij}(t) = 0$, then there does not exist an edge from agent j to agent i at time t. Intuitively, one can think of x_i as an approximation of the probability of agent i being infected, and $1 - x_i$ represents an approximation of the probability of agent i being healthy. The state can also be interpreted as the proportion of subpopulation i that is infected. Therefore, for the remainder of the article, we assume that the initial values of each node's state lie in the interval [0,1].

The model in (1) was introduced in [9, eq. (10)], whereas, for the time-invariant case, it has been also proposed in [14, eq. (1)].

The virus outbreaks that motivate this work are often recorded in epidemiological reports that are compiled per day [28], [29] or week [30]. This sampling of the system behavior motivates the use of a discrete-time SIS model [14]. The model is obtained by applying the Euler's method [31] to (1),

$$x_i(k+1) = x_i(k) + h\Bigg((1-x_i(k))\beta_i(k)\sum_{j=1}^n a_{ij}(k)x_j(k) - \delta_i(k)x_i(k)\Bigg) \tag{2}$$

where h is the sampling parameter, and, therefore, $h > 0$. Observe that system (2) is a discrete-time nonlinear time-varying system, and quite naturally its stability analysis differs considerably from that of discrete-time linear time-varying systems.

The spread of diseases in a network can be modeled using a graph: the nodes representing the agents or subpopulations, and the edges representing the interaction among them. More formally, let $G_k = (V, E_k)$ represent such a network, where $V = \{1, 2, \ldots, n\}$ is the vertex set and $E_k = \{(x_i, x_j) \mid a_{ij}(k) \neq 0\}$ is the edge set.

The model in (2) can be written in a matrix form as

$$x(k+1) = x(k) + h((I - X(k))B(k)A(k) - D(k))x(k) \tag{3}$$

where $X(k) = \operatorname{diag}(x(k))$, $B(k) = \operatorname{diag}(\beta_i(k))$, $D(k) = \operatorname{diag}(\delta_i(k))$, and $A(k) = [a_{ij}(k)]$, for every $i, j \in [n]$. Let us define $\bar{B}(k) := B(k)A(k)$, with its entries being denoted as $\bar{\beta}_{ij}(k)$. Then, (3) can be rewritten as

$$x(k+1) = x(k) + h((I - X(k))\bar{B}(k) - D(k))x(k). \tag{4}$$

Observe that $\bar{\beta}_{ij}(k)$ represents the infection rate and nearest-neighbor graph both at time instant k. That is, assuming $\beta_i(k) > 0$ and that there exists an edge from vertex j to vertex i (i.e., $a_{ij}(k) > 0$), $\bar{\beta}_{ij}(k)$ scales the weight on the edge from vertex j to vertex i by $\beta_i(k)$.

Since we are interested in seasonal epidemics, we restrict our attention to discrete-time *periodic* SIS models. Thus, we have the following assumption.

Assumption 1: Given some period $p \in \mathbb{Z}_+$, $B(k+p) = B(k)$, $A(k+p) = A(k)$, and $D(k+p) = D(k)$ for all $k \geq 0$.

The DFE is defined as the state where $x_i(k) = 0$ for all $i \in [n]$, which, from (4), implies that $x_i(\kappa) = 0$ for all $\kappa \geq k$, for all $i \in [n]$. We are interested in ensuring that, irrespective of the initial condition of an agent, i.e., healthy or sick, the system

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should exponentially (respectively asymptotically) converge to the DFE.

Throughout this article, we interchangeably use the terms "healthy state" and "DFE," and likewise the terms "convergence to the DFE" and "eradication of the virus."

With the above-described setup in place, the objectives of this article are as follows.

1) For the system with dynamics as given in (4), find sufficient condition(s) such that the DFE is the only equilibrium and GES.
2) For the system with dynamics as given in (4), find necessary and sufficient condition such that the DFE is the only equilibrium and GAS.
3) Based on the knowledge of the graph topologies, infection rates and the conditions for exponential (respectively asymptotic) convergence to the DFE, develop a distributed control strategy such that the DFE can be exponentially (respectively asymptotically) stabilized.

We make the following assumptions.

Assumption 2: We have $h\delta_i(k) \geq 0$ and $\bar{\beta}_{ij}(k) \geq 0$ for every $i, j \in [n]$, $k \in [p]^-$. ■

Assumption 3: For every $i, j \in [n]$ and $k \in [p]^-$, $h\delta_i(k) \leq 1$ and $h\sum_j \bar{\beta}_{ij}(k) \leq 1$. ■

Assumption 2 says that, for each agent, the healing and infection rates are non-negative. Assumption 3 is required for ensuring that our model is well-defined.

Lemma 1: For the system in (4), under the conditions of Assumptions 2 and 3 and if $x_i(0) \in [0, 1]$, for all $i \in [n]$, then $x_i(k) \in [0, 1]$ for all $i \in [n]$ and $k \geq 0$. ■

The proof is along similar lines as that of [14, Lemma 1], and, hence, is skipped. □

Lemma 1 ensures that the set $[0, 1]^n$ is positively invariant, i.e., once a trajectory of (4) enters the set $[0, 1]^n$, it stays within the set $[0, 1]^n$ for all future time instants.

III. Preliminaries

In this section, we recall various notions of stability of discrete-time deterministic systems [32, Sec. 5.9], which will be used in the sequel. Additionally, we collect some useful results from the literature that facilitate the development of our main results.

Consider a system, described as follows:

$$x(k+1) = f(k, x(k)) \tag{5}$$

where $f : \mathbb{Z}_{\geq 0} \times \mathbb{R}^n \to \mathbb{R}^n$ is locally Lipschitz. We say that an equilibrium of the abovementioned equation is (uniformly) asymptotically stable if it is (uniformly) stable and (uniformly) attractive. An equilibrium is said to be GAS [respectively globally uniformly asymptotically stable (GUAS)] if in addition to being asymptotically stable (respectively uniformly asymptotically stable) the system converges to that equilibrium for any initial condition. We recall a sufficient condition for GUAS of an equilibrium of (5).

Lemma 2: [32, Sec. 5.9 Th. 27] The DFE of system (5) is GUAS if there is a function $V : \mathbb{Z}_+ \times \mathbb{R}^n \to \mathbb{R}$ such that i) $V(k, 0) = 0$, and, for all $x \neq 0, V(k, x) > 0$, ii) V is decrescent, and radially unbounded, and iii) $-\Delta V$ (where the forward difference function $\Delta V : \mathbb{Z}_+ \times \mathbb{R}^n \to \mathbb{R}$ is defined as: $\Delta V(k, x) = V(k + 1, x(k + 1)) - V(k, x)$) is positive definite. ■

A stronger notion of stability is that of GES, which is defined as follows.

Definition 1: An equilibrium point of (5) is GES if there exist positive constants α and η, with $0 \leq \eta < 1$, such that

$$\|x(k)\| \leq \alpha \|x(k_0)\| \eta^{(k-k_0)} \ \forall k, k_0 \geq 0 \quad \forall x_{k_0} \in \mathbb{R}^n.$$

We recall a sufficient condition for GES of an equilibrium of (5) in the following proposition.

Lemma 3: [32, Sec. 5.9 Th. 28] Suppose there exists a function $V : \mathbb{Z}_+ \times \mathbb{R}^n \to \mathbb{R}$, and constants $a, b, c > 0$ and $p > 1$ such that $a\|x\|^p \leq V(k, x) \leq b\|x\|^p$, $\Delta V(k, x) \leq -c\|x\|^p$ $\forall k \geq 0$, and $\forall x \in \mathbb{R}^n$, then $x = 0$ is an exponentially stable equilibrium of (5). ■

The initial values are in the domain $[0, 1]^n$, since otherwise they do not correspond to reality for the model under consideration. Consequently, we can say that the DFE of system (4) is GES if the condition in Definition 1 (respectively Lemma 3) is satisfied for all $x_{k_0} \in [0, 1]^n$. Similarly, we say that the DFE of system (4) is GAS if the condition in Lemma 2 is satisfied for all $x_{k_0} \in [0, 1]^n$.

The following lemmas will be needed for proving the sufficiency results in the sequel.

Lemma 4: [33, Proposition 1] Suppose that M is a nonnegative matrix such that $\rho(M) < 1$. Then there exists a diagonal matrix $P \succ 0$ such that $M^\top PM - P \prec 0$. ■

Lemma 5: [14, Lemma 3] Suppose that M is an irreducible nonnegative matrix such that $\rho(M) = 1$. Then there exists a diagonal matrix $P \succ 0$ such that $M^\top PM - P \preccurlyeq 0$. ■

The following proposition is used for proving the necessity result in the sequel.

Proposition 1: [32, Sec. 5.9 Th. 42] Consider the autonomous system

$$x(k+1) = f(x(k)). \tag{6}$$

Define $A = [\frac{\partial f}{\partial x}]_{x=0}$. If A has at least one eigenvalue with magnitude greater than 1, then $x = 0$ is an unstable equilibrium of (6). ■

Given that this article concerns periodic systems, we now recall a result concerning the time invariance of the spectrum of the state transition matrix.

Proposition 2: [34, p. 157] Consider the discrete-time p-periodic time-varying autonomous system

$$x(k+1) = A(k)x(k). \tag{7}$$

Let $A_{k+p:k}$ denote the corresponding state transition matrix. The spectrum of $A_{k+p:k}$ is independent of k. ■

One of the approaches toward studying stability issues in time-varying networks relies on the notion of *joint spectral radius*—first introduced by Rota and Strang in [35]—of a set of matrices; see for instance [16], [19]. In the sequel, we will explore the relation between the joint spectral radius of an appropriately defined set of matrices and GES (respectively GAS) of the DFE.

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We define the following:

$$M(k) := I - hD(k) + h\bar{B}(k) \tag{8}$$

$$\hat{M}(k) := I + h((I - X(k))\bar{B}(k) - D(k))$$

$$M_{k+p:k} := M(k+p-1)M(k+p-2)\cdots M(k). \tag{9}$$

Observe that the linearizing system (4) around the DFE yields a linear time-varying periodic system, whose state matrix is $M(k)$.

Let $\mathcal{M} = \{M(0), M(1), \ldots, M(p-1)\}$ denote a set of p matrices $M(k)$, where $k \in [p]^-$. As was defined in [19], the joint spectral radius of $\mathcal{M}$, denoted by $\rho(\mathcal{M})$, is

$$\rho(\mathcal{M}) = \lim_{p\to\infty} \sup \|M(p-1)M(p-2)\cdots M(0)\|^{\frac{1}{p}},$$

$$\text{where } M(k) \in \mathcal{M} \quad \forall k \in [p]^-. \tag{10}$$

That is, $\rho(\mathcal{M})$ is the largest eigenvalue of the product of p matrices in $\mathcal{M}$ amongst *all* products of p matrices in $\mathcal{M}$.

IV. Exponential Convergence to the DFE

In this section, we present sufficient conditions for GES of the DFE. In the context of epidemic outbreaks, these conditions guarantee the eradication of the epidemic exponentially fast. Recalling the understanding of joint spectral radius in (10), the following result gives a sufficient condition for the DFE to be GES.

Theorem 1: Consider (4) under Assumptions 1–3. If $\rho(\mathcal{M}) < 1$, then the DFE is GES. ■

Proof: See the Appendix, where additionally we establish an upper bound on the rate of convergence. □

The result in Theorem 1, albeit restricted to periodic systems, is relevant in its own right: Theorem 1 gives a sufficient condition for GES of the DFE, whereas the same condition in [19, Th. 2.2], particularized for periodicity assumptions, guarantees only *local* exponential stability of the DFE. Moreover, the proof technique is entirely different.

Notice that checking the condition on the joint spectral radius in Theorem 1 essentially entails asking the following question: given a set of matrices, say $\mathcal{R}$, is each and every product of matrices within $\mathcal{R}$ stable? Answering this question is known to be NP-hard; see [36, Corollary 2]. Hence, we are motivated to seek a different condition that is computationally tractable.

The following corollary is an immediate consequence of Proposition 2 and the proof of Theorem 1, and provides a less restrictive sufficient condition for GES of the DFE.

Corollary 1: Consider (4) under Assumptions 1–3. If, for some $k \in [p]^-$, $\rho(M_{k+p:k}) < 1$, then the DFE is GES. ■

For the continuous-time setting, [9, Th. 2] gives a sufficient condition for GES of the DFE, under the assumption that the rate of change of topology is suitably bounded. To the best of our knowledge, for discrete-time time-varying SIS epidemics, Theorem 1 and Corollary 1 are the first results for GES of the DFE. Moreover, unlike [9, Th. 2], neither Theorem 1 nor Corollary 1 rely on any restrictions on *how large* the variations in topology can be.

The following remark provides an epidemiological interpretation of the implications of Theorem 1 (and Corollary 1).

Remark 1: The result in Corollary 1 is useful in the following sense: Subject to virus mutation and the underlying sequence of graph topologies repeating with some period p, we can conclude that the virus will be eradicated. Notice that this result is irrespective of how the aforementioned parameters vary with time; in particular, even if at times, compared to the healing rates, the infection rates are dominant. The same has been illustrated via simulations in Section VII. ■

Recall that [13, Theor. 5] provides a sufficient condition for exponential convergence to the DFE. However, the model in [13] is different from (4); healing (respectively infection) rates are non-negative and cannot be greater than 1, and the edge-weights and the spreading parameters are not allowed to vary with time. Additionally, there are certain restrictions on the underlying time-invariant graph; in particular, all self-loops have weight equal to 1, and no edge can have weight greater than 1. Consequently, [13, Theor. 5] is less general than Theorem 4.

To the best of our knowledge, for the SIS model in (4), even when particularized for the *time-invariant* case (thus, yielding the model in [14]), a sufficient condition for exponential convergence to the DFE does not exist in the literature. It can be immediately seen that the condition in Corollary 1 can be specialized for the time-invariant setting, as discussed next. First, note that time-invariant systems are periodic systems with periodicity $p = 1$. Hence, $M_{k+p:k} = M$ for every $k \in \mathbb{Z}_{\geq 0}$, which implies that the condition in Corollary 1 is satisfied if $\rho(M) < 1$. Therefore, we have the following proposition.

Proposition 3: Consider the nonmutating, static graph topology version of (4), that is, where $p = 1$. If $\rho(M) < 1$, then the DFE is GES. ■

Proposition 3 establishes GES of the DFE, whereas [14, Theor. 2], under the same condition as in Proposition 3, establishes only GAS of the DFE. Hence, Proposition 3 is a stronger version of [14, Theor. 2].

Notice that, on one hand, the conditions in Theorem 1 and Corollary 1 involve strict inequalities. On the other, they guarantee faster convergence to the DFE. An obvious question that one can ask is the following: is it possible to relax the strict inequalities in Theorem 1 and Corollary 1 at the cost of *slower* convergence? In the context of epidemiology, the motivation for doing so goes along the following lines: depending on the severity of the epidemic in question, there might be scenarios, for instance the common cold, where a positive answer to the question: "will the disease die out?" suffices, and one is not too concerned with the *speed* with which the epidemic disappears. We investigate the same in the following section.

V. Asymptotic Convergence to the DFE

It turns out that if the inequality in Corollary 1 and, therefore, in Theorem 1 were not necessarily strict, then the DFE is GAS. Furthermore, if the inequality in Corollary 1 were to be reversed, then the healthy state is an unstable equilibrium. Thus, in this section, we establish a sufficient condition, and a necessary and sufficient condition for GAS of the DFE.

We begin by noting that an immediate consequence of Theorem 1 is the following.

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Corollary 2: Consider (4) under Assumptions 1–3. If $\rho(\mathcal{M}) < 1$, then the DFE is GAS. ■

It turns out that the DFE is endowed with the property of GAS even if $\rho(\mathcal{M}) = 1$. To prove this claim, we need, besides the assumptions in Corollary 2, the following assumptions.

Assumption 4: We have $h \neq 0$ and, for all $k \in [p]^-$, there exists $i \neq j$ such that $\bar{\beta}_{ij}(k) > 0$. ■

Assumption 5: For each $k \in [p]^-$, the graph G_k is strongly connected. ■

Assumption 4 rules out scenarios wherein an agent is infected, yet since it is not connected to any of the other agents in the network, it does not transmit the virus. Assumption 5 implies that the adjacency matrix $\bar{B}(k)$, where $k \in [p]^-$, is irreducible, i.e., $\bar{B}(k)$ cannot be permuted to a block upper triangular matrix.

Proposition 4: Consider (4) under Assumptions 1–5. If $\rho(\mathcal{M}) = 1$, then the DFE is GAS. ■

Proof: See Appendix □

Combining Corollary 2 and Proposition 4, we readily obtain the following result.

Theorem 2: Consider (4) under Assumptions 1–5. If $\rho(\mathcal{M}) \leq 1$, then the DFE is GAS. ■

Theorem 2 establishes asymptotic stability of the equilibrium point $x = 0$, even for the case when $\rho(\mathcal{M}) = 1$. Thus, it differs from [19, Theor. 2.2] wherein no conclusions can be drawn when $\rho(\mathcal{M}) = 1$.

It is well-known that the condition in Theorem 2 is undecidable; see [37, Theor. 2]. That is, given a finite set of matrices $\mathcal{R}$, it is impossible to construct an algorithm that gives a correct binary answer to the question: is $\rho(\mathcal{R}) \leq 1$?

Observe that from Proposition 2 and the proof of Theorem 2, the following less restrictive sufficient condition is immediate.

Corollary 3: Consider (4) under Assumptions 1–5. If, for some $k \in [p]^-$, $\rho(M_{k+p:k}) \leq 1$, then the DFE is GAS. ■

It turns out that condition in Corollary 3 is also necessary.

Proposition 5: Consider (4) under Assumptions 1–5. The DFE is asymptotically stable only if, for some $k \in [p]^-$, $\rho(M_{k+p:k}) \leq 1$. ■

Proof: See Appendix. □

Combining Corollary 3 and Proposition 5, readily yields the following.

Theorem 3: Consider (4) under Assumptions 1–5. The DFE of system (4) is GAS if, and only if, for some $k \in [p]^-$, $\rho(M_{k+p:k}) \leq 1$. ■

For the continuous-time setting, it has been shown that the switched SIS model admits a limit cycle if the condition in Theorem 3 is violated; see [16, Theor. 6.4]. While our simulations suggest that, for the discrete-time setting, violating the condition in Theorem 3 could lead to the existence of a limit cycle (see Section VII), this conjecture remains open. The main difficulty in proving this claim stems from the fact that the celebrated Poincaré–Benedickson Theorem—which forms the underpinning for the proof in the continuous-time case—does not seem to have a discrete-time counterpart.

Theorem 3 is a more general version of [14, Theor. 2], as discussed next.

Remark 2: If system (4) is time-invariant or equivalently, $p = 1$, then the condition in Theorem 3 coincides with the condition in [14, Theor. 2]. To see this, consider the following argument: since $p = 1$, for every $k \in \mathbb{Z}_{\geq 0}$ $M_{k+p:k} = M$. Therefore, $\rho(M_{k+p:k}) = \rho(M)$. Hence, $\rho(M_{k+p:k}) \leq 1$ if and only if $\rho(M) \leq 1$, which is the same as the condition in [14, Th. 2]. ■

Notice that the objective insofar has been to find conditions that ensure exponential (respectively asymptotic) convergence to the DFE. Knowledge of the stability conditions enables health administration officials to determine *how* the model parameters in (4) should be adjusted so as to completely stop the spreading of a disease. We focus on the same in the following section.

VI. Distributed Control Strategy

In this section, we show that increasing the healing rate of each agent by a sufficiently high amount ensures eradication of the epidemic. For time-invariant continuous-time SIS models, (local) techniques for eliminating the spread of epidemics have been provided in [15, Sec. V]. Inspired by the same, we explore similar strategies for the periodic, mutating setup, as in this article. More specifically, in the sequel, we study how to influence the healing rate of each agent so that the DFE is exponentially (respectively asymptotically) stabilized. Toward this end, we consider the following healing rates:

$$\delta_i(k) = \sum_{j=1}^{n} \bar{\beta}_{ij}(k) + \gamma_i \quad \forall k \in [p]^-, i \in [n] \tag{11}$$

where for each $i \in [n]$, $\gamma_i > 0$.

It turns out that choosing healing rates as in (11) together with appropriate assumptions on γ_i ensures that the DFE of (4) is GES. Hence, we have the following theorem.

Theorem 4: Consider (4) under Assumptions 1–2. Suppose that, for each $i \in [n]$, γ_i in (11) satisfies $h \sum_{j=1}^{n} \bar{\beta}_{ij}(k) + h\gamma_i \leq 1$. Then for healing rates as in (11), the DFE of (4) is GES. ■

Proof: See Appendix. □

Observe that if $\gamma_i = 0$ $\forall i \in [n]$ in (11), then, from the proof of Theorem 4, and that of Proposition 4, it follows that the DFE is GAS; thus, leading us to the following corollary.

Corollary 4: Consider (4) under Assumptions 1–5. For healing rates of the form

$$\delta_i(k) = \sum_{j=1}^{n} \bar{\beta}_{ij}(k)$$

for each $i \in [n]$ and $k \in [p]^-$, the DFE of (4) is GAS. ■

The key insight that can be gleaned from Theorem 4 and Corollary 4 is that there exist sufficiently large, yet finite, time-varying healing rates such that the DFE can be stabilized. This could inform healthcare professionals of disease-response techniques, as explained in the following remark.

Remark 3: The distributed control strategy proposed in Theorem 4 (respectively Corollary 4) may be interpreted in the following sense: if the healing rate of each agent is suitably increased—for instance by injecting sufficiently high dosages of antidote—then the virus will be eradicated exponentially (respectively asymptotically) fast. While the control strategy is extreme, i.e., no constraints are imposed, it informs decision

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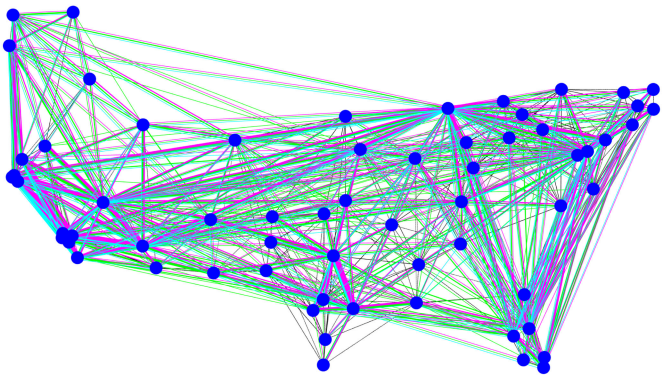

Fig. 1. Final condition for simulation with $\delta = 35$. All nodes are in the DFE, depicted by blue.

makers of the best way to respond if ample resources are available and encourages the stockpiling of resources to combat possible outbreaks. ■

VII. Simulations

The main challenge with new infectious diseases is that it is often unclear how they spread and how contagious they are [1]. Therefore, motivated by diseases like SARS and COVID-19 and to further understand the implications of the results from the previous sections, we present simulations over a network of 64 cities in the United States. The default graph structure is a binary, nearest-neighbor graph depicted in Fig. 1 by black dotted edges. For the periodic parts of the network we aggregate the Southwest Airlines flights between the cities, split by departure time in the morning (0:00–8:00), the day (8:00–16:00), and the evening (16:00–24:00), which are depicted in Fig. 1 by green, magenta, and cyan, respectively, and the edge weights are scaled by the number of flights. The maximum number flights in and out of one city during the day is 125; therefore, we set $h = 0.005$ to meet Assumption 3. We use the initial condition of Albuquerque completely infected and every other city completely healthy (however, the results are independent of nonzero initial condition). In the plots of the network, blue (b) represents healthy and red (r) represents infected. The coloring of each node i at time k follows:

$$x_i(k)r + (1 - x_i(k))b. \tag{12}$$

Since our interest lies in eradicating the virus, we explore the results on the stability of the DFE from Sections IV and V via simulations. For simplicity we use homogeneous, nonmutating virus parameters with $\beta = 1$. We also suppose that the healing rate of each agent is the same, i.e., $\delta_i = \delta$ for each $i \in [n]$. Recall from Proposition 2, that the spectrum of $M_{k+p:k}$ is independent of k. When $\delta = 35$, $\rho(M_{k+3:k}) = 0.9815$. Consistent with the results in Section IV, the system converges to the DFE; see Fig. 1. When $\delta = 33.765$, $\rho(M_{k+3:k}) = 1.000$. In line with the results in Section V, the system with $\rho(M_{k+3:k}) = 1.000$ converges at a much slower rate than the other; see Fig. 2. For both of these systems $\rho(M_0) < 1$, while $\rho(M_1)$ and $\rho(M_2)$ are greater

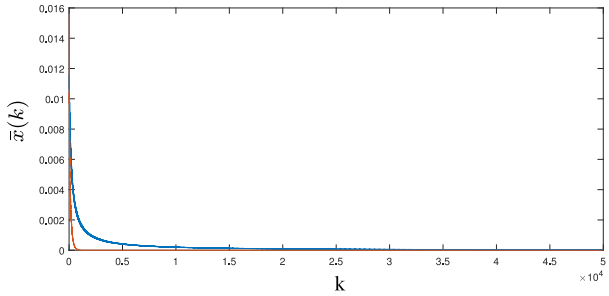

Fig. 2. Average infection level of the cities over time. Blue is for the system with $\rho(M_{k+3:k}) = 1.000$ and red is for $\rho(M_{k+3:k}) < 1.000$.

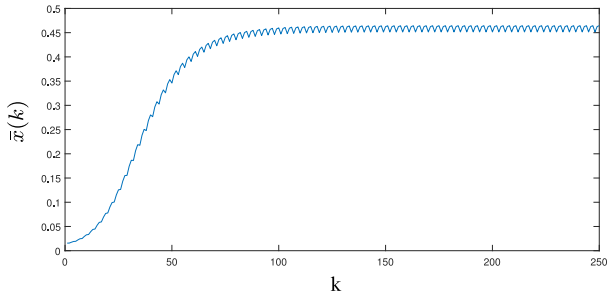

Fig. 3. Average infection level of the cities over time for the simulation with $\delta_i(k) = 10$, for every $k \in \{0, 1, 2\}$, $i \in [n]$.

than 1. Thus, we see that even if the infection rates dominate the healing rates for the majority of the time, the virus can still be eradicated. This insight offers hope for control algorithm design; having actuator capabilities for some portion of the period might be sufficient to eradicate a virus.

Next, we focus on illustrating the instability result from Section V. Our simulations, consistent with the result in Proposition 5, exemplify that when $\rho(M_{k+p:k}) > 1$, the DFE is an unstable equilibrium. Moreover, although it still needs to be rigorously proven, our simulations show the existence of limit-cycle behavior, thus, implying *persistence* of an outbreak when $\rho(M_{k+p:k}) > 1$.

For this simulation, we employ the same parameters as the simulations in Fig. 2 except $\delta = 10$. This system has $\rho(M_{k+3:k}) = 1.4015$ and converges to a limit cycle with three states. This limit-cycle behavior is illustrated via the average infection level plotted in Fig. 3. The values of the three limit cycle states are quite close to each other; therefore, we only plot one in Fig. 4. Simulations show that the limit cycle is independent of the initial condition, given that $x(0) \neq 0$. This finding implies that if, when factoring in the network connections, the infection rates dominate the healing rates sufficiently, the virus can pervade the network. Therefore, intervention is essential.

In order to see how well the distributed control technique from Section VI performs, we implement it here. In the context of the simulation, the algorithm can be interpreted as a strategy for boosting the healing rates of the more susceptible cities, which could be implemented by deploying mobile treatment clinics, distributing medicine/antidote, and installing

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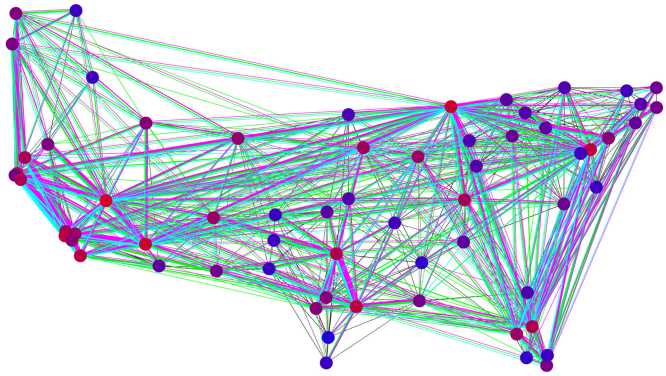

Fig. 4. One of the limit cycle states for the simulation with $\delta_i(k) = 10$. All cities become at least partially infected, depicted by the reddish purple color, following (12).

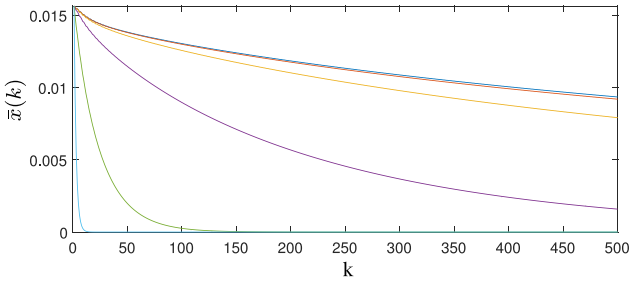

Fig. 5. Average infection level of the cities over time with the healing rates set to (11) with $\gamma_i = \{0, 0.01, 0.1, 1.0, 10, 100\}$, for every $i \in [n]$.

hand-washing stations in airports and other public places. For this simulation, we keep the model parameters the same as the previous simulations except we set the healing rates using (11) with $\gamma_i = \gamma = \{0, 0.01, 0.1, 1.0, 10, 100\}$, for every $i \in [n]$, and $h = 0.004$. Consistent with the results in Theorem 4 and Corollary 4, the system converges to the DFE in exponential time for nonzero γ and in asymptotic time when $\gamma = 0$. Here, we explore the effect of γ on the convergence rate. The average level of infection for each γ value is shown in Fig. 5. We see that for this system $\gamma = 0.01$ behaves very similarly to $\gamma = 0$, while $\gamma \geq 10$ eradicates the virus relatively quickly. Therefore, if there are enough resources available to boost the healing rates of the cities, the virus can be eradicated.

However, in certain situations there may not be enough resources to implement such viral-combatant measures during every time step. Nevertheless, as we saw in the simulations in Fig. 2, it is not necessary that the healing rates dominate the infection rates at every time step, or even a majority of the time, in order for the virus to be eradicated. In this set of simulations, we explore the effectiveness of the distributed control strategy proposed in Section VI when the redesign of the healing parameters can only be implemented for part of the period. Given our flight example, the constraint can be interpreted as there only being enough resources to boost the healing rates of the cities during the day, however, not in the early morning or at night. Therefore, we implement the controller from (11) but only during the work

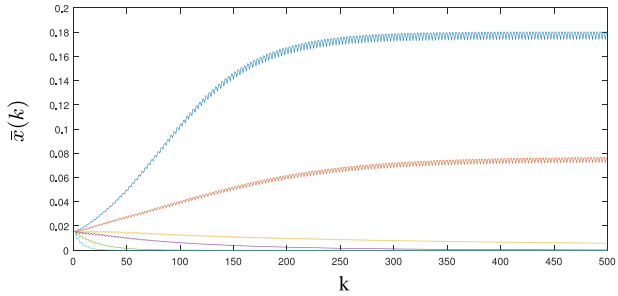

Fig. 6. Average infection level of the cities over time with the healing rates for the work day period (8:00–16:00) set to (11) with $\gamma_i = \{0, 10, 19.7, 25, 50, 100\}$ and for the other two periods $\gamma_i = 10, \forall i \in [n]$.

day (8:00–16:00). For the other two periods, we set $\delta = 10$ for every city, similar to the simulation in Fig. 3 that displayed the limit-cycle behavior. We run a set of simulations with different γ values in (11), $\gamma = \{0, 10, 19.7, 25, 50, 100\}$, where $\gamma = \gamma_i$ for every $i \in [n]$. We plot the average infection level for each simulation in Fig. 6. As would be expected, a greater γ value is needed to eradicate the virus than when actuation is allowed for all three periods. However, even when no control actuation is available for the majority of the periods, the virus can be eradicated for this system if, for the work day period, $\gamma \geq 19.7$. As would be expected, in the case where the equality holds, $\rho(M_{k+p:k}) = 1$. This result gives hope that, even when there are constraints on the distributed control strategy, the virus can be eradicated.

VIII. Conclusion

Considering discrete-time periodic time-varying networks with a mutating virus, this article has dealt with the problem of designing a control strategy that ensures exponential (respectively asymptotic) convergence to the healthy state. Our approach was the following: we first provided conditions for exponential (respectively asymptotic) convergence to the DFE. Thereafter, we exploited the proven conditions for the design of a distributed control strategy.

Note that we have restricted our attention to periodic time-varying systems. Hence, a line of future research could be to remove the periodicity assumption. Second, this article operated under the assumption that there was a *single* virus that was infecting the population. Generalizing this setup to account for *multiple*—not necessarily two—competing viruses could be an appealing line of investigation. Third, this article dealt with a *deterministic* model; an inherent drawback with deterministic models is that they do not account for the possibility that the system dynamics can be corrupted by noise. Consequently, deterministic models limit our understanding of the potential behaviors involved and a future direction is to study the stochastic version of the periodic SIS model. Finally, our simulations indicate, under the assumption that the condition in Theorem 3 is violated, the possible existence of a limit-cycle behavior. However, a rigorous proof (or a counterexample) for this conjecture remains missing.

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APPENDIX

Proof of Theorem 1

We use the cyclic reformulation of a linear periodic system; see [38, Sec. 6.3]. Specifically, define

$$\tilde{M} = \begin{bmatrix} 0 & 0 & \cdots & 0 & M(p-1) \\ M(0) & 0 & \cdots & 0 & 0 \\ 0 & M(1) & \cdots & 0 & 0 \\ \vdots & \vdots & \ddots & \vdots & \vdots \\ 0 & 0 & \cdots & M(p-2) & 0 \end{bmatrix}. \quad (13)$$

Note that

$$\tilde{M}^p = \begin{bmatrix} M_{p:0} & 0 & \cdots & 0 \\ 0 & M_{p+1:1} & \cdots & 0 \\ \vdots & \ddots & \ddots & \vdots \\ 0 & & 0 & M_{2p-1:p-1} \end{bmatrix}. \quad (14)$$

Since $\tilde{M}^p$ is a block diagonal matrix, the eigenvalues of $\tilde{M}^p$ are the eigenvalues of $M_{p:0}$, $M_{p+1:1}$, …, $M_{2p-1:p-1}$. By assumption, $\rho(\mathcal{M}) < 1$. Hence, from (10), it follows that, for all $k \in [p]^-$, $\rho(M_{p:k}) < 1$, and therefore, $\rho(\tilde{M}^p) < 1$. Since the eigenvalues of $\tilde{M}$ are the pth roots of eigenvalues of $\tilde{M}^p$, it follows that $\rho(\tilde{M}) < 1$.

Since, by Assumptions 2–3, $M(k)$ is nonnegative, it follows that $\tilde{M}$ is also nonnegative. Therefore, from Lemma 4, there exists a diagonal matrix $Q_1 \succ 0$ such that $\tilde{M}^\top Q_1 \tilde{M} - Q_1 \prec 0$. Let the diagonal blocks of Q_1 be denoted by $[Q_1]_k \in \mathbb{R}^{N\times N}$, for all $k \in [p]$. By defining $P_1(k) = [Q_1]_{k+1}$, for all $k \in [p]^-$, it is immediate that $M(k)^\top P_1(k+1)M(k) - P_1(k) \prec 0$ for all $k \in [p]^-$.

Consider the following Lyapunov function $V_1(k,x) = x^\top P_1(k)x$. Since $Q_1 \succ 0$ and diagonal, each of the blocks along its diagonal must be positive definite. This implies that, for all $k \in [p]^-$ and for $x \neq 0$, $x^\top P_1(k)x > 0$, and hence, $V_1(k,x) > 0$. Since for all $k \in [p]^-$ $P_1(k)$ is positive definite, each eigenvalue of $P_1(k)$ is real and positive. Then, since $P_1(k)$ is also symmetric, by applying the Rayliegh–Ritz Theorem [39], we obtain

$$\lambda_{\min}(P_1(k))I \leq P_1(k) \leq \lambda_{\max}(P_1(k))I$$

and hence

$$\lambda_{\min}(P_1(k))\|x\|^2 \leq V_1(k,x) \leq \lambda_{\max}(P_1(k))\|x\|^2. \quad (15)$$

Define $\sigma_1 := \min_{k\in[p]^-} \lambda_{\min}(P_1(k))$, and

$$\sigma_2 := \max_{k\in[p]^-} \lambda_{\max}(P_1(k)). \quad (16)$$

Since for $k \in [p]^-$ $\lambda_{\min}(P_1(k)) > 0$ and $\lambda_{\max}(P_1(k)) > 0$, it follows that $\sigma_1 > 0$ and $\sigma_2 > 0$. Thus, we have found positive constants σ_1, σ_2 such that for all $k \in [p]^-$

$$\sigma_1\|x\|^2 \leq V_1(k,x) \leq \sigma_2\|x\|^2. \quad (17)$$

Define $\Delta V_1(k,x) = V_1(x(k+1)) - V_1(x(k))$. For $x \neq 0$, and for all $k \in [p]^-$, one obtains the following:

$$\begin{aligned}\Delta V_1(k,x) &= x^\top \hat{M}^\top(k)P_1(k+1)\hat{M}(k) - x^\top P_1(k)x \\ &= x^\top(M^\top(k)P_1(k+1)M(k) - P_1(k))x \\ &\quad - 2hx^\top \bar{B}^\top(k)X(k)P_1(k+1)M(k)x \\ &\quad + h^2 x^\top \bar{B}^\top(k)X(k)P_1(k+1)X\bar{B}(k)x. \quad (18)\end{aligned}$$

From Assumptions 2–3 and Lemma 1, the following is satisfied:

$$x^\top(h^2 x^\top \bar{B}^\top(k)X(k)P_1(k+1)X(k)\bar{B}(k)$$
$$-2hx^\top \bar{B}^\top(k)X(k)P_1(k+1)M(k))x \leq 0.$$

Hence, from (18), we obtain

$$\Delta V_1(k,x) \leq x^\top(M^\top(k)P_1(k+1)M(k) - P_1(k))x.$$

Recall that, for all $k \in [p]^-$, $M^\top(k)P_1(k+1)M(k) - P_1(k)$ is negative definite, and therefore, $M^\top(k)P_1(k+1)M(k) - P_1(k)$ is symmetric with all real and negative eigenvalue. Hence, applying Rayleigh–Ritz Theorem yields: for all $k \in [p]^-$

$$\Delta V_1(k,x) \leq \lambda_{\max}(M^\top(k)P_1(k+1)M(k) - P_1(k))\|x\|^2.$$

Since $M^\top(k)P_1(k+1)M(k) - P_1(k)$ is negative definite, $P_1(k) - M^\top(k)P_1(k+1)M(k)$ is positive definite, and hence, we obtain $\lambda_{\max}(M^\top(k)P_1(k+1)M(k) - P_1(k)) = -\lambda_{\min}(P_1(k) - M^\top(k)P_1(k+1)M(k))$, which leads to

$$\Delta V_1(k,x) \leq -\lambda_{\min}(P_1(k) - M^\top(k)P_1(k+1)M(k))\|x\|^2. \quad (19)$$

Defining

$$\sigma_3 := \max_{k\in[p]^-} \lambda_{\min}(P_1(k) - M^\top(k)P_1(k+1)M(k)) \quad (20)$$

it follows from (19) that $\Delta V_1(k,x) \leq -\sigma_3\|x\|^2$. Since, for all $k \in [p]^-$, $P_1(k) - M^\top(k)P_1(k+1)M(k)$ is positive definite, it follows that $\sigma_3 > 0$.

Thus, there exists positive constants, σ_1, σ_2, and σ_3, such that for $x \neq 0$ and for all $k \in [p]^-$,

$$\sigma_1\|x\|^2 \leq V_1(k,x) \leq \sigma_2\|x\|^2 \quad (21)$$

$$\Delta V_1(k,x) \leq -\sigma_3\|x\|^2. \quad (22)$$

By Assumption 1, $M(k+p) = M(k)$ for every $k \in \mathbb{Z}_{\geq 0}$. Hence, over every successive interval of size p, the matrix $\tilde{M}$ remains the same. This implies that $P_1(k+p) = P_1(k)$ for every $k \in \mathbb{Z}_{\geq 0}$. Hence, we can use the *same* Lyapunov function over *every* successive interval of size p. Thus, repeating the same analysis as in the interval $[0, p-1]$ for every successive interval of size p results in inequalities (21) and (22) being satisfied for all $k \in \mathbb{Z}_{\geq 0}$ and for all $x \in [0,1]^n$. Therefore, from Lemma 3, the system converges exponentially fast to the DFE, for all $x(0) \in [0,1]^n$. □

Next, we explore the rate of convergence to the DFE.

Proposition 6 (Rate of Convergence): Under the assumptions of Theorem 1, the rate of convergence to the DFE is upper

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bounded by an exponential with rate $\sqrt{1-\frac{\sigma_3}{\sigma_2}}$, where σ_2 and σ_3 are defined in (16) and (20), respectively. ■

Proof: The expression for the rate $\sqrt{1-\frac{\sigma_3}{\sigma_2}}$ follows directly from (21) to (22) and [40, Theor. 23.3]. Now we show that the rate is well-defined, that is, $0 \leq \sqrt{1-\frac{\sigma_3}{\sigma_2}} < 1$. To see this, consider the following: notice that, since $\sigma_2 > 0$ and $\sigma_3 > 0$, it suffices to show that $\sigma_2 \geq \sigma_3$. Toward this end, observe that, for all $k \in [p]^-$, both $P_1(k)$ and $M^\top(k)P_1(k+1)M(k)$ are symmetric. Applying Weyl's inequalities [39, Corollary 4.3.15] to $P_1(k) - M^\top(k)P_1(k+1)M(k)$, one obtains, for all $k \in [p]^-$ and $i \in [n]$:

$$\begin{aligned}&\lambda_i(P_1(k) - M^\top(k)P_1(k+1)M(k))\\ &\quad\leq \lambda_i(P_1(k)) + \lambda_{\max}(-M^\top(k)P_1(k+1)M(k))\end{aligned}$$

which implies

$$\lambda_{\max}(P_1(k)) \geq \lambda_{\max}(P_1(k) - M^\top(k)P_1(k+1)M(k)) \tag{23}$$

$$\geq \lambda_{\min}(P_1(k) - M^\top(k)P_1(k+1)M(k)) \tag{24}$$

where (23) holds because $-M^\top(k)P_1(k+1)M(k)$ is negative semidefinite, implying $\lambda_{\max}(-M^\top(k)P_1(k+1)M(k)) \leq 0$. By definitions of σ_2 and σ_3, and since (24) is satisfied for all $k \in [p]^-$, it follows that $\sigma_2 \geq \sigma_3$. □

Proof of Proposition 4

By assumption, $\rho(\mathcal{M}) = 1$. This implies, from the definition of joint spectral radius, that, for all $k \in [p]^-$, $\rho(M_{p:k}) \leq 1$, and therefore, $\rho(\tilde{M}^p) \leq 1$. Since the eigenvalues of $\tilde{M}$ are the pth-roots of eigenvalues of $\tilde{M}^p$, it follows that $\rho(\tilde{M}) \leq 1$. For the case where $\rho(\tilde{M}) < 1$, from the proof of Theorem 1, the DFE of (4) is GAS. Hence, in the rest of the proof, we focus on the case where $\rho(\tilde{M}) = 1$.

Suppose that $\rho(\tilde{M}) = 1$. Since by Assumptions 2–5, $M(k)$, for each $k \in [p]^-$, is irreducible and nonnegative, it follows that $\tilde{M}$ is also irreducible and nonnegative. Hence, from Lemma 5, there exists a positive diagonal matrix Q_2 such that $\tilde{M}^\top Q_2 \tilde{M} - Q_2 \preccurlyeq 0$. By defining, for all $k \in [p]^-$, $P_2(k) = [Q_2]_{k+1}$, it is immediate that $M(k)^\top P_2(k+1)M(k) - P_2(k) \preccurlyeq 0$, where $k \in [p]^-$.

Consider the Lyapunov candidate function $V_2(k,x) = x^\top P_2(k)x$. Observe that, by analogous reasoning as in proof of Theorem 1, for all $k \in [p]^-$ and for $x \neq 0$, $V_2(k,x) > 0$. By mimicking the steps involved in establishing the right-hand side of inequality (17), and invoking [32, Def. 22, p 266], it can be seen that $V_2(k,x)$ is decrescent. Define $\Delta V_2(k,x) = V_2(x(k+1)) - V_2(x(k))$. For $x \neq 0$, from (4), one obtains

$$\begin{aligned}\Delta V_2(k,x) &= x^\top \hat{M}^\top(k)P_2(k+1)\hat{M}(k)x(k) - x^\top P_2(k)x\\ &= x^\top(M^\top(k)P_2(k+1)M(k) - P_2(k))x\\ &\quad - 2hx^\top \bar{B}^\top(k)X(k)P_2(k+1)M(k)x(k)\\ &\quad + h^2 x^\top \bar{B}^\top(k)X(k)P_2(k+1)X(k)\bar{B}(k)x\\ &\leq h^2 x^\top \bar{B}^\top(k)X(k)P_2(k+1)X(k)\bar{B}(k))x\\ &\quad - 2hx^\top \bar{B}^\top(k)X(k)P_2(k+1)M(k)x\\ &= h^2 x^\top \bar{B}^\top(k)X(k)P_2(k+1)X(k)\bar{B}(k)x\\ &\quad - hx^\top \bar{B}^\top(k)X(k)P_2(k+1)M(k)x\\ &\quad - h^2 x^\top \bar{B}^\top(k)X(k)P_2(k+1)\bar{B}(k)x\\ &\quad - hx^\top \bar{B}^\top(k)X(k)P_2(k+1)(I - hD(k))x\\ &\leq h^2 x^\top \bar{B}^\top(k)X(k)P_2(k+1)X(k)\bar{B}(k)x\\ &\quad - hx^\top \bar{B}^\top(k)X(k)P_2(k+1)M(k)x\\ &\quad - h^2 x^\top \bar{B}^\top(k)X(k)P_2(k+1)\bar{B}(k)x\\ &\leq -h^2 x^\top \bar{B}^\top(k)X(k)P_2(k+1)(I - X(k))\bar{B}(k)x\\ &\quad - hx^\top \bar{B}^\top(k)X(k)P_2(k+1)M(k)x\\ &\leq -hx^\top \bar{B}^\top(k)X(k)P_2(k+1)M(k)x\\ &\leq 0.\end{aligned}$$

It can be immediately seen that if $x = 0$, then for all $k \in [p]^-$, $\Delta V_2(k,x) = 0$. For every $k \in \mathbb{Z}_{\geq 0}$, by Assumptions 2 and 5, $\bar{B}(k)$ (and hence $M(k)$) is nonzero and nonnegative, whereas, from Lemma 5, $P_2(k)$ is a positive diagonal matrix. Hence, if, for all $k \in [p]^-$, $-hx^\top \bar{B}^\top(k)X(k)P_2(k+1)M(k)x = 0$, then $x = 0$.

For reasons, similar to those outlined in the proof of Theorem 1, the aforesaid analysis can be repeated over *every* successive interval of size p, which yields $V_2(k,x) > 0$ and $\Delta V_2(k,x) \leq 0$ for every $k \in \mathbb{Z}_{\geq 0}$. Moreover, it can be immediately seen that $V_2(k,x)$ is radially unbounded, since $V_2(k,x) = \|P_2(k)^{\frac{1}{2}}x\|^2$. Therefore, from Lemma 2, the DFE is GAS. □

Proof of Proposition 5

System (4) can be represented as a nonlinear *time-invariant* system, using the technique provided in [41, Sec. IV-C]. Toward this end, we first write (4) using (9) as

$$x(pq+k+1) = \hat{M}(pq+k)x(pq+k) \tag{25}$$

where p is the periodicity of system (4), and q is any nonnegative integer. Recall from (9) that

$$\begin{aligned}\hat{M}(pq+k) &= M(pq+k) - hX(pq+k)\bar{B}(pq+k)\\ &= M(k) - hX(pq+k)\bar{B}(k)\end{aligned} \tag{26}$$

where (27) is a consequence of Assumption 1. Define

$$\hat{M}_k(pq) := M(k \bmod p) - hX(pq+k)\bar{B}(k \bmod p). \tag{27}$$

By concatenating the state vector over an interval of size p, we define a new state variable $y \in \mathbb{R}^{pn}$,

$$y(pq) := \begin{bmatrix} y_1(pq) \\ y_2(pq) \\ \vdots \\ y_p(pq) \end{bmatrix} = \begin{bmatrix} x(pq) \\ x(pq+1) \\ \vdots \\ x(pq+p-1) \end{bmatrix}. \tag{28}$$

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TABLE I
DEFINITION OF $\bar{\tilde{M}}(X)$

$$\bar{\tilde{M}}(X) := \begin{bmatrix} \hat{M}_{p-1}(pq)\cdots\hat{M}_0(pq) & 0 & \cdots & 0 \\ 0 & \hat{M}_p(pq)\cdots\hat{M}_1(pq) & \cdots & 0 \\ \vdots & \vdots & \ddots & 0 \\ 0 & 0 & \cdots & \hat{M}_{2p-2}(pq)\cdots\hat{M}_{p-1}(pq) \end{bmatrix}$$

We are interested in studying how $y(pq)$ evolves for $q \in \{0, 1, 2, \ldots\}$. From the abovementioned equation, we have

$$y(p(q+1)) = \begin{bmatrix} y_1(p(q+1)) \\ y_2(p(q+1)) \\ \vdots \\ y_p(p(q+1)) \end{bmatrix} = \begin{bmatrix} x(p(q+1)) \\ x(p(q+1)+1) \\ \vdots \\ x(p(q+1)+p-1) \end{bmatrix}.$$

From (25), (27), and (28), we know that

$$\begin{aligned} x(p(q+1)) &= \hat{M}_{p-1}(pq)\cdots\hat{M}_0(pq)x(pq) \\ x(p(q+1)+1) &= \hat{M}_p(pq)\cdots\hat{M}_1(pq)x(pq+1) \\ &\vdots \\ x(p(q+1)+p-1) &= \hat{M}_{2p-2}(pq)\cdots\hat{M}_{p-1}(pq)x(pq+p-1). \end{aligned}$$

Hence, we can rewrite the dynamics as

$$y(p(q+1)) = \bar{\tilde{M}}(X)y(pq) \tag{29}$$

where $\bar{\tilde{M}}(X)$ is defined in Table I. Note that as a consequence of (27), the dynamics of the system in the abovementioned equation, for any $q \in \mathbb{Z}_+$, depend only on the matrices $M(k)$ and $\bar{B}(k)$, for all $k \in [p]^-$, and the state vector. Therefore, (29) is a discrete-time nonlinear time-invariant system. Linearizing (29) around $y = 0$ yields the following:

$$y(p(q+1)) = \tilde{M}^p y(pq) \tag{30}$$

where $\tilde{M}^p$ is defined in (14).

By way of contraposition, assume that, for all $k \in [p]^-$, $\rho(M_{k+p:k}) > 1$. This assumption implies that $\rho(\tilde{M}^p) > 1$, since $\tilde{M}^p$ is a block diagonal matrix. Therefore, from Proposition 1, $y = 0$ is an unstable equilibrium of system (29). Since, by (28), $y = 0$ corresponds to the DFE of (4), the DFE is an unstable equilibrium of system (4). ■

Proof of Theorem 4

Consider healing rates as in (11). Define $\hat{B}(k) = \operatorname{diag}(\sum_{j=1}^n \bar{\beta}_{ij}(k) + \gamma_i)$. Then substituting (11) into (2), and rewriting the system in the matrix form yields:

$$x(k+1) = x(k) + h((I - X(k))\bar{B}(k) - \hat{B}(k))x(k). \tag{31}$$

Define $M^1(k) = I - h\hat{B}(k) + h\bar{B}(k)$. As a consequence of Assumptions 2 and since, for each $i \in [n]$, γ_i is chosen such that $h\sum_{j=1}^n \bar{\beta}_{ij}(k) + h\gamma_i \leq 1$, for all $k \in [p]^-$, $M^1(k)$ is non-negative. Moreover, since $h > 0$, and $\gamma_i > 0\ \forall i \in [n]$, each row i of $M^1(k)$ satisfies $\sum_{j=1}^n [M^1(k)]_{ij} < 1$ for all $k \in [p]^-$.

By building $\tilde{M}^1$ analogous to $\tilde{M}$ in (13), the structure of $\tilde{M}^1$ immediately gives that $\tilde{M}^1$ is non-negative and that each row satisfies $\sum_{j=1}^n [\tilde{M}^1]_{ij} < 1$. Thus, by definition of the infinity norm of a matrix, $\|\tilde{M}^1\|_\infty < 1$, which, due to [39, Th. 5.6.9], further implies that $\rho(\tilde{M}^1) < 1$. From the proof of Theorem 1, it is clear that $\rho(\tilde{M}^1) < 1$ ensures that the DFE is GES. □

REFERENCES

[1] L. S. Hung, "The SARS epidemic in Hong Kong: What lessons have we learned?" *J. Roy. Soc. Med.*, vol. 96, no. 8, pp. 374–378, Jul. 2003.

[2] E. Dong, H. Du, and L. Gardner, "An interactive web-based dashboard to track COVID-19 in real time," *Lancet Infectious Diseases*, vol. 20, no. 5, pp. 533–534, May 2020.

[3] M. Pascual and A. Dobson, "Seasonal patterns of infectious diseases," *PLoS Med.*, vol. 2, no. 1, Jan. 2005, Art. no. e5.

[4] D. Bernoulli, "Essai d'une nouvelle analyse de la mortalité causée par la petite vérole, et des avantages de l'inoculation pour la prévenir," *Histoire de l'Acad., Roy. Sci.(Paris) avec Mem*, pp. 1–45, 1760.

[5] H. W. Hethcote, "The mathematics of infectious diseases," *SIAM Rev.*, vol. 42, no. 4, pp. 599–653, Jan. 2000.

[6] P. V. Mieghem, J. Omic, and R. Kooij, "Virus spread in networks," *IEEE/ACM Trans. Netw.*, vol. 17, no. 1, pp. 1–14, Feb. 2009.

[7] D. Easley and J. Kleinberg, *Networks, Crowds, Markets*, vol. 8. Cambridge, U.K.: Cambridge Univ. Press, Jul. 2010.

[8] H. J. Ahn and B. Hassibi, "Global dynamics of epidemic spread over complex networks," in *Proc. 52nd IEEE Conf. Decis. Control*, 2013, pp. 4579–4585.

[9] P. E. Paré, C. L. Beck, and A. Nedić, "Epidemic processes over timevarying networks," *IEEE Trans. Control Netw. Syst.*, vol. 5, no. 3, pp. 1322–1334, Sep. 2018.

[10] A. Khanafer, T. Başar, and B. Gharesifard, "Stability of epidemic models over directed graphs: A positive systems approach," *Automatica*, vol. 74, pp. 126–134, Dec. 2016.

[11] A. Fall, A. Iggidr, G. Sallet, and J. J. Tewa, "Epidemiological models and Lyapunov functions," *Math. Model. Natural Phenomena*, vol. 2, no. 1, pp. 62–83, 2007.

[12] Y. Wang, D. Chakrabarti, C. Wang, and C. Faloutsos, "Epidemic spreading in real networks: An eigenvalue viewpoint," in *Proc. 22nd Int. Symp. Reliable Distrib. Syst.*, 2003, pp. 25–34.

[13] C. Peng, X. Jin, and M. Shi, "Epidemic threshold and immunization on generalized networks," *Physica A: Statist. Mechanics Appl.*, vol. 389, no. 3, pp. 549–560, Feb. 2010.

[14] P. E. Paré, J. Liu, C. L. Beck, B. E. Kirwan, and T. Başar, "Analysis, estimation, and validation of discrete-time epidemic processes," *IEEE Trans. Control Syst. Technol.*, vol. 28, no. 1, pp. 79–93, Jan. 2020.

[15] J. Liu, P. E. Pare, A. Nedic, C. Y. Tang, C. L. Beck, and T. Basar, "Analysis and control of a continuous-time bi-virus model," *IEEE Trans. Autom. Control*, vol. 64, no. 12, pp. 4891–4906, Dec. 2019.

[16] O. Mason, F. R. Wirth, M. A. Rami, and V. Bokharaie, "Stability criteria for SIS epidemiological models under switching policies," *Discrete Continuous Dynamical Syst. Ser.*, vol. 19, no. 9, p. 2865–2887, 2014.

[17] P. E. Paré, J. Liu, C. L. Beck, A. Nedić, and T. Başar, "Multi-competitive viruses over static and time-varying networks," in *Proc. Amer. Control Conf.*, 2017, pp. 1685–1690.

[18] B. A. Prakash, H. Tong, N. Valler, M. Faloutsos, and C. Faloutsos, "Virus propagation on time-varying networks: Theory and immunization algorithms," in *Mach. Learn. Knowl. Discovery Databases*. Berlin, Germany: Springer, 2010, pp. 99–114.

[19] V. Bokharaie, O. Mason, and F. Wirth, "MTNS-85: Seventh international symposium on the mathematical theory of networks and systems," *Signal Process.*, vol. 7, no. 1, pp. 87–98, Sep. 1984.

[20] Q. Liu, "The threshold of a stochastic susceptible-infective epidemic model under regime switching," *Nonlinear Anal., Hybrid Syst.*, vol. 21, pp. 49–58, Aug. 2016.

Authorized licensed use limited to: KTH Royal Institute of Technology. Downloaded on March 03,2021 at 09:50:12 UTC from IEEE Xplore. Restrictions apply.

[21] M. Ogura and V. M. Preciado, "Stability of spreading processes over time-varying large-scale networks," *IEEE Trans. Netw. Sci. Eng.*, vol. 3, no. 1, pp. 44–57, Jan. 2016.
[22] E. A. Enns, J. J. Mounzer, and M. L. Brandeau, "Optimal link removal for epidemic mitigation: A two-way partitioning approach," *Math. Biosci.*, vol. 235, no. 2, pp. 138–147, Feb. 2012.
[23] C. Nowzari, V. M. Preciado, and G. J. Pappas, "Analysis and control of epidemics: A survey of spreading processes on complex networks," *IEEE Control Syst.*, vol. 36, no. 1, pp. 26–46, Feb. 2016.
[24] C. Enyioha, A. Jadbabaie, V. Preciado, and G. Pappas, "Distributed resource allocation for control of spreading processes," in *Proc. Eur. Control Conf.*, Jul. 2015, pp. 2216–2221.
[25] E. Ramírez-Llanos and S. Martínez, "Distributed discrete-time optimization algorithms with applications to resource allocation in epidemics control," *Optimal Control Appl. Methods*, vol. 39, no. 1, pp. 160–180, Jul. 2017.
[26] J. A. Torres, S. Roy, and Y. Wan, "Sparse resource allocation for linear network spread dynamics," *IEEE Trans. Autom. Control*, vol. 62, no. 4, pp. 1714–1728, Apr. 2016.
[27] V. S. Mai, A. Battou, and K. Mills, "Distributed algorithm for suppressing epidemic spread in networks," *IEEE Control Syst. Lett.*, vol. 2, no. 3, pp. 555–560, Jul. 2018.
[28] World Health Organization (WHO), Accessed: 2020-02-03, *novel coronavirus 2019-nCoV*. [Online]. Available: https://www.who.int/westernpacific/emergencies/novel-coronavirus
[29] J. Snow, *On Mode Commun. Cholera*. Ongar, U.K.: John Churchill.
[30] *Ebola Virus Disease Democratic Republic of the Congo*, 2020. [Online]. Available: https://www.who.int/csr/don/30-january-2020-ebola-drc/en/
[31] K. E. Atkinson, "Current awareness in phytochemical analysis," *Phytochemical Anal.*, vol. 19, no. 1, pp. 91–98, Jan. 2008.
[32] M. Vidyasagar, Nonlinear Systems Analysis, vol. 42. Philadelphia, PA, USA: SIAM, Jan. 2002.
[33] A. Rantzer, "Distributed control of positive systems," in *Proc. IEEE Conf. Decis. Control Eur. Control Conf.*, Dec. 2011, pp. 6608–6611.
[34] S. Bittanti, "Deterministic and stochastic linear periodic systems," in *Time Series and Linear Systems*. New York, NY, USA: Springer-Verlag, pp. 141–182.
[35] G. Rota and W. G. Strang, "A note on the joint spectral radius," *Indagationes Mathematicae (Proc.)*, vol. 63, pp. 379–381, 1960.
[36] J. N. Tsitsiklis and V. D. Blondel, "The Lyapunov exponent and joint spectral radius of pairs of matrices are hard? When not impossible? To compute and to approximate," *Math. Control, Signals, Syst.*, vol. 10, no. 1, pp. 31–40, Mar. 1997.
[37] V. D. Blondel and J. N. Tsitsiklis, "The boundedness of all products of a pair of matrices is undecidable," *Syst. Control Lett.*, vol. 41, no. 2, pp. 135–140, Oct. 2000.
[38] S. Bittanti and P. Colaneri, *Periodic Control System*, vol. 5108985. Hoboken, NJ, USA: Wiley, Feb. 2018.
[39] R. A. Horn and C. R. Johnson, in *Matrix Analysis*. Cambridge, U.K.: Cambridge Univ. Press, 2009.
[40] W. J. Rugh, *Linear System Theory* , vol. 2. Upper Saddle River, NJ, USA: Prentice-Hall 1996.
[41] M. Ye, J. Liu, B. D. O. Anderson, C. Yu, and T. Basar, "Evolution of social power in social networks with dynamic topology," *IEEE Trans. Autom. Control*, vol. 63, no. 11, pp. 3793–3808, Nov. 2018.

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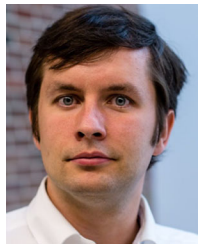

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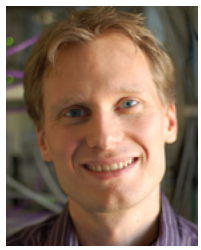

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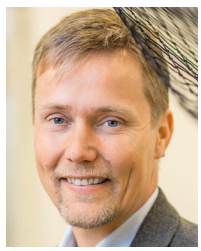

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