International Journal of Robust and Nonlinear Control

WILEY

RESEARCH ARTICLE

Mitigation of Discrete-Time SIS Networked Epidemics: A Local State Feedback Approach

Yuan Wang[1] | Sebin Gracy[2] | Hideaki Ishii[3] | Karl Henrik Johansson[4]

[1]School of Robotics, Hunan University, Changsha, China | [2]Department of Electrical Engineering and Computer Science, South Dakota School of Mines and Technology, Rapid City, South Dakota, USA | [3]Department of Information Physics and Computing, The University of Tokyo, Tokyo, Japan | [4]Division of Decision and Control Systems, School of Electrical Engineering and Computer Science, KTH Royal Institute of Technology, and Digital Futures, Stockholm, Sweden

Correspondence: Yuan Wang (yuanw@hnu.edu.cn)

Received: 14 October 2024 | **Revised:** 20 January 2025 | **Accepted:** 9 February 2025

Funding: This work was supported by National Natural Science Foundation of China Grant No. 92470202; by Provincial Natural Science Foundation of Hunan Grant No. 2024JJ6159; and by the Knut and Alice Wallenberg Foundation, Wallenberg Scholar under Grant 66469; by a Distinguished Professor Grant from the Swedish Research Council (Org: JRL, Project No: 3058); and by JSPS under Grant-in-Aid for Scientific Research Grant No. 22H01508.

Keywords: discrete-time networked SIS models | epidemic processes | mitigating epidemics

ABSTRACT

The paper deals with a discrete-time susceptible-infected susceptible (SIS) networked epidemic model. In the model, nodes represent populations and the network links possible transmission pathways of the disease between populations. Our aim is to design a feedback controller so that the fraction of infected in each population node remains below a prespecified value for all time instants. To this end, we introduce a distributed control law at the node level. This control law can be realized by the population following announcements made by local policymakers to enhance nonpharmaceutical interventions such as hand-washing, mask-wearing, and social distancing. We show that with the controller in place not only do the fraction of infected in each population node stay below the prespecified level but also the state of the disease dynamics converges either to the disease-free equilibrium or to a unique endemic equilibrium. It turns out that the endemic equilibrium is (element-wise) smaller than the unique endemic equilibrium of the uncontrolled system. The theoretical findings are illustrated by numerical examples.

1 | Introduction

Epidemics have posed serious challenges during the course of human civilization. Some of the most prominent examples in the past century include the Spanish flu 1918–1920, the Asian flu in the 1950s [1], and the recent COVID-19 pandemic. The toll of destruction that epidemics leave in their wake is enormous. For instance, the Spanish flu resulted in around 50 million deaths, set back the economy worldwide by a few decades, whereas the Justinian plague possibly led to the collapse of the Byzantine empire [2]. Unsurprisingly, the spread of diseases has drawn the attention of the scientific community, with the earliest work being a model for the smallpox virus formulated and analyzed by Daniel Bernouli [3]. Mathematical epidemiology, as a discipline, witnessed tremendous growth during the 20th century [4–8]. In recent years, problems in epidemiology have been investigated in different fields such as physics [9], computer science [10], economics [11], and so on. A fundamental quest behind such research efforts is to understand *what* causes a disease to spread, and *how* such a spread can be mitigated.

To this end, several models have been developed in the literature. These include, but are not limited to, susceptible-infected (SI), susceptible-infected-recovered (SIR), susceptible-infected

susceptible (SIS), and susceptible-exposed-infected-recovered (SEIR) models [12]. SIS models have been studied since the 1930s [13].

In the classic SIS model, each individual within a single population belongs to one of the two mutually exclusive health compartments: (i) susceptible to the disease (S) or (ii) infected by the disease (I). Infected individuals can transmit the disease to susceptible ones and recover from the disease. However, recovery does not necessarily confer long term immunity against reinfection. Note that disease spread is typically not constrained to a single city, region, or even country. Motivated by this observation, networked epidemic models have been proposed [12, 14]. In such models, each node represents a distinct population, and the edges connecting nodes signify disease transmission pathways between populations. Indeed, networked SIS models have been useful for studying the spread of diseases such as gonorrhea [15] and tuberculosis [16]. They have been analyzed extensively in the literature. Both time-invariant [17, 18] and time-varying networks [19–22] have been accounted for. In the context of time-invariant networks, both continuous-time [9, 17, 18] and discrete-time settings [10, 23–26] have been studied.

In this article, the focus is on *time-invariant discrete-time networked SIS models*, which were introduced in [26]. The behavior of this model is constrained by the reproduction number $\mathcal{R}_0$, which is determined by the recovery and infection rates, along with the adjacency matrix of the network topology (a precise definition of $\mathcal{R}_0$ appears in Section 2.1) [14, 27]. Specifically, when $\mathcal{R}_0 \leq 1$, the network dynamics converge to a healthy state, indicating the eradication of the disease from all nodes. Conversely, if $\mathcal{R}_0 > 1$, the disease persists and infects a fraction of individuals within each node in the network [26]. Conditions for the existence, uniqueness and stability of the endemic equilibrium have been presented [25, 28]. We note that all of the aforementioned works deal with finding conditions that result in the epidemic becoming extinct or persistent. From a public health perspective, it is of strong interest not to just know *when* a disease becomes endemic or extinct, but also *how* the spread dynamics can be manipulated so as to obtain a desired behavior. This is referred to as *control* of epidemics.

Control of SIS epidemics has received attention from the research community [29–31]. A vast majority of the literature on SIS-type models employ "one-shot" control strategies: Reduce $\mathcal{R}_0$ below one by manipulating the network through the removal of nodes or edges, or by altering the recovery rate or infection rates [29, 32, 33]. Note that one-shot approaches do *not* account for the current or past infection levels. Consequently, these are not necessarily well-suited as the epidemic evolves. One way to overcome this limitation is to employ state feedback mechanisms to dynamically alter the infection or recovery rates based on infection levels in the network [27, 34–37]. Furthermore, such mechanisms can be deployed in a decentralized fashion; each population node independently adjusts its parameters by solely relying on its own infection levels. Notice, however, that the aim of the control strategies in all of the aforementioned papers, assuming $\mathcal{R}_0 > 1$, is to drive the system to an endemic equilibrium that is (element-wise) smaller than the endemic equilibrium of the uncontrolled system. None of these strategies guarantee that, while the epidemic is in progress, the healthcare facilities would be commensurate with the number of infected cases, so that the epidemic remains manageable. More formally stated: the trajectories of the state, representing the fraction of infected individuals in each population, should never exceed a prespecified level (this level can be thought of as representative of the capacity of healthcare facilities in a node). Indeed, during the early phases of an epidemic, one of the primary objectives of health administration officials is not to overload the existing healthcare facilities, such as hospitals, nursing homes, etc. Such an objective would ensure that the number of patients who are seriously sick is not too high, and, therefore, they will receive the required medical treatment [38].

In this article, we address the existing limitations noted in the literature above by proposing a decentralized state feedback controller for the discrete-time networked SIS model which ensures that the fraction of infected individuals remains upper bounded by a prespecified value for all time instants. We adjust the effective infection rates via multiplicative gains which evolve adaptively, utilizing information about the current fraction of infected individuals; no information about the network, such as recovery or infection rates, is required to execute the algorithm. From a practical standpoint, the proposed feedback controller is useful in the development and evaluation of public health strategies for the containment and mitigation of infectious diseases via non-pharmaceutical interventions (NPIs).

The contribution of this work is as follows: First, we formulate a novel feedback control problem under the discrete-time networked SIS model (Problem 1), and propose a feedback controller to address the problem. Under certain assumptions, we show for the controlled system that the infected proportion for each node remains below a prespecified level for all time instants. Furthermore, the dynamics of the controlled system either asymptotically converges to the disease-free equilibrium (DFE) or to the endemic equilibrium (Theorem 1). Second, we show that the proposed control strategy drives the endemic equilibrium *closer* to the DFE. That is, the endemic equilibrium of the controlled system is strictly smaller in each component than that of the uncontrolled system (Theorem 2). We carry out extensive simulations to study how our proposed feedback controller performs under various uncertainties in real-world epidemic prevention scenarios. A preliminary version of this paper has appeared in a brief conference version [39], we considered the special case when all nodes have the same upper bound on the infection level. The current paper, besides relaxing the constraint on the upper bounds of the infection level, contains further theoretical results and more extensive discussions along with new numerical results.

The paper is organized as follows. The problem to be studied is introduced in Section 2. The problem is addressed in Section 3. Simulations evaluating our theoretical findings as well as the effects of uncertainties in the measurements and control are provided in Section 4. Finally, concluding remarks and some open problems are presented in Section 5.

Notation: Let $\mathbb{R}_+$ and $\mathbb{Z}_+$ denote the sets of nonnegative real numbers and nonnegative integers, respectively. For any positive integer n, we use $[n]$ to denote the set $\{1, 2, \ldots, n\}$. For any two vectors $\mathbf{a}, \mathbf{b} \in \mathbb{R}^n$, we write $\mathbf{a} > \mathbf{b}$ if $a_i > b_i$ for every $i \in [n]$. For any two matrices A and B, $A > B$ indicates that each entry

in A is strictly larger than the corresponding entry in B. For a matrix A, let $\rho(A)$ denote the spectral radius of A. A diagonal matrix is denoted as $\text{diag}(\cdot)$. We use $\mathbf{1} = [1, 1, \ldots, 1]^T$ and $\mathbf{0} = [0, 0, \ldots, 0]^T$ to denote the vectors of all-ones and all-zeros, respectively. Given a matrix A, $A \prec 0$ (resp. $A \preceq 0$) indicates that A is negative definite (negative semidefinite). A matrix is called *Metzler* if its off-diagonal entries are all nonnegative. A matrix $A \in \mathbb{R}^{n\times n}$ with all off-diagonal entries nonpositive is called an M-matrix if it can be written as $A = sI - B$, for some $s > 0$ and $B \geq \mathbf{0}_{n\times n}$ [40].

2 | Problem Formulation

In this section, we introduce the discrete-time networked SIS model, and formally state the problem being studied in this article.

2.1 | Discrete-Time Networked SIS Model

Consider a network with $n \geq 2$ large, distinct, well-mixed populations represented by a graph $\mathcal{G} = (\mathcal{V}, \mathcal{E})$. Each node $i \in \mathcal{V}$ denotes a population of fixed size comprising of individuals. The set $\mathcal{E}$ denotes the set of interconnections between the nodes. More precisely, $\mathcal{E} = \{(i, j) \in \mathcal{V} \times \mathcal{V} | a_{ij} \neq 0\}$, where the weight $a_{ij} > 0$ represents the strength of interconnection from node j to i (which can be considered as the frequency of human mobility among different geographic areas). Suppose that a virus is spreading over this network. Each individual, at any given time instant, belongs to one of the two health compartments: susceptible or infected. A node is said to be healthy if each individual in it is healthy; otherwise, we say it is infected. An individual that is infected by the virus recovers from the infection, and subsequently becomes susceptible to the virus. Let $\beta_i > 0$ and $\gamma_i > 0$ denote the local infection and recovery rates of each node, respectively. Let $x_i(t)$ denote the fraction of population i who are infected with the disease at time $t \in \mathbb{R}_+$.

The evolution of the fraction of infected individuals in node $i \in [n]$ can be represented by the scalar differential equation in [17]

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

Observe that the state $x_i(t)$ denotes the proportion of infected in population i at time $t \geq 0$ and thus $1 - x_i(t)$ is the proportion of susceptible in population i.

While the evolution of an epidemic is a continuous-time process, the outbreaks are often recorded in epidemiological reports that are compiled per day or week [41, 42]. Such a sampling of the system behavior has recently motivated the use of a discrete-time SIS model [26, 28]. The discrete-time version of (1) can be obtained by applying Euler's method:

$$x_i(k + 1) = x_i(k) + h\left[\beta_i(1 - x_i(k))\sum_{j=1}^{n} a_{ij}x_j(k) - \gamma_i x_i(k)\right] \tag{2}$$

where $h > 0$ is the sampling period and $k \in \mathbb{Z}_+$. Here, with a slight abuse of notation, $x_i(k)$ is the state at time kh. For system (2), if, for each $i \in [n]$, $x_i(k) = 0$ implies that $x_i(k') = 0$ for all $k' \geq k$, then we say that system (2) is in the *DFE*. If, for each $i \in [n]$, $x_i(k) = x_i^*$, where $0 < x_i^* < 1$, implies that $x_i(k') = x_i^*$ for all $k' \geq k$, then we say that system (2) is in the *endemic equilibrium*.

Let $\mathbf{x}(k) := [x_1(k), x_2(k), \ldots, x_n(k)]^T$, $\Xi := \text{diag}(\beta_i)$, $\Gamma := \text{diag}(\gamma_i)$, and $R := \Gamma^{-1}\Xi$. Note that R is a diagonal matrix with $[R]_{ii} = \beta_i/\gamma_i$. Then, system (2) can be written in vector form as

$$\mathbf{x}(k + 1) = \left[I + h\Xi(I - \text{diag}(\mathbf{x}(k)))A - h\Gamma\right]\mathbf{x}(k) \tag{3}$$

In the sequel, we refer to system (3) as the *uncontrolled system*. We make the following standing assumptions in the paper, which are common in the study of discrete-time networked SIS models [26, 28].

Assumption 1. For each $i \in [n]$, $\beta_i > 0$ and $\gamma_i > 0$.

Assumption 2. The graph $\mathcal{G}$ is strongly connected.

Assumption 3. For each $i \in [n]$, $x_i(0) \in [0, 1]$.

Assumption 4. For each $i \in [n]$, the sampling period h fulfills $h\gamma_i + h\beta_i\sum_{j=1}^{n} a_{ij} \leq 1$.

The graph $\mathcal{G}$ being strongly connected means that each node $i \in \mathcal{V}$ has a path to every other node $j \in \mathcal{V}$. Note that the adjacency matrix $A = [a_{ij}]_{n\times n}$ is irreducible if and only if the underlying graph $\mathcal{G}$ is strongly connected.

The Proposition below establishes the basic reproduction number $\mathcal{R}_0$, determining the long-term presence (or absence) of a disease.

Proposition 1. *Consider the uncontrolled system (3), under Assumptions 1–4. Define the matrix $M := I + h\Xi A - h\Gamma$, and let $\mathcal{R}_0 := \rho(M)$.*

1. *If $\mathcal{R}_0 \leq 1$, then the DFE is the unique equilibrium of (3) and $\lim_{k\to\infty} \mathbf{x}(k) = \mathbf{0}$.*
2. *If $\mathcal{R}_0 > 1$, then the DFE is unstable and, moreover, there exists another equilibrium $\mathbf{x}^* > \mathbf{0}$ such that $\lim_{k\to\infty} \mathbf{x}(k) = \mathbf{x}^*$ for every $\mathbf{x}(0) \in [0, 1]^n \setminus \mathbf{0}$.*

Proof. See Appendix section Proof of Proposition 1. □

Proposition 1 is a combination of Theorems 1, 2 and Proposition 2 in [26], as well as Theorem 1 in [28]. It establishes the existence, uniqueness and stability results for the equilibria of the uncontrolled system (3). The DFE is always an equilibrium of (3) – irrespective of the parameter choices. For certain parameter choices, the system possesses a unique endemic equilibrium [26]. Outside of these two equilibria, it possesses no other equilibria.

2.2 | Problem Statement

Proposition 1 guarantees that the networked SIS model converges to one of two equilibria, but does not guarantee that the state stays

below a certain value (smaller than one). In reality, during the early phases of an epidemic, one of the primary objectives of policymakers is to ensure that the local healthcare facilities are not overloaded. In other words, one seeks to ensure that the infection levels do not exceed a prespecified limit. One of the approaches towards achieving this objective involves modifying the infection rate of each node during the epidemic.

The vast majority of networked SIS models treat β_i as a fixed parameter [43, 44]. This means that the infection rate of a node remains constant throughout the epidemic. Therefore, to achieve the aforementioned objective, it is imperative that the overall infection rate be taken as the product of the intrinsic transmission rate β_i and the effective contact rate $g_i(\cdot)$, which can be actively modified based on measurements of infection within the population. Such a modification in the infection rate can be captured by adjusting (2) in the following manner:

$$x_i(k+1) = x_i(k) + hg_i(k)\beta_i(1 - x_i(k))\sum_{j=1}^{n} a_{ij}x_j(k) - h\gamma_i x_i(k) \quad (4)$$

where $g_i(k) \in [0, 1]$ is a control gain that represents the effectiveness of NPIs in lowering the acting infection rate. NPIs are typically represented by means of a manipulation of β_i [45–47]. Thus, $g_i(k)\beta_i$ can be considered the controlled infection rate, which should ensure that the infection levels stay below a prespecified level for all time instants.

Clearly, if $g_i(k)$ is taken to be a constant, then the controlled system (4) is in the same form as the uncontrolled system (2) with a modified infection rate $g_i\beta_i$. If policymakers have complete knowledge of networked epidemics (including the topology and all parameters β_i, γ_i, and a_{ij}), then a naive approach is to design g_i to ensure $\mathcal{R}_0 < 1$; this can be effectively done by the one-shot method based on Proposition 1.

We begin by assuming that we have a discrete-time SIS network whose initial state $x_i(0)$ does not exceed the healthcare facility capacity, denoted by some constant $c_i \geq 1$ for node $i \in [n]$. This is summarized by the following assumption:

Assumption 5. For each $i \in [n]$, $x_i(0) \in [0, 1/c_i)$.

The problem studied in this paper can now be formally stated.

Problem 1. Consider the controlled networked SIS system (4) under Assumptions 1, 2, 5, and 4. Find a decentralized controller gain $g_i(k)$ to ensure that (i) for all $i \in [n]$ and $k \in \mathbb{Z}_+$, $x_i(k) \in [0, 1/c_i)$; and (ii) $\lim_{k\to\infty} x_i(k) = x_i^*$, where $x_i^* \geq 0$ for all $i \in [n]$.

3 | Mitigation of Discrete-Time SIS Networked Epidemics

In this section, we identify a decentralized controller gain $g_i(k)$ that solves Problem 1. Indeed, our solution is

$$g_i(k) = 1 - c_i x_i(k) \quad (5)$$

In Section 3.1, we show that Problem 1 is solved when $\mathcal{R}_0 \leq 1$, whereas in Section 3.2 we show that it is solved when $\mathcal{R}_0 > 1$. The control gain can be interpreted as follows: Susceptible individuals in population i (denoted by the term $1 - x_i(k)$) are possibly infected over the network by infectious individuals (denoted by the term $h\beta_i \sum a_{ij}x_j(k)$), with the total infection "force" adjusted by $g_i(k) \in [0, 1]$ to read as $hg_i(k)\beta_i\sum_{j=1}^{n} a_{ij}x_j(k)$ [34]. We note that the control law in (5) is decentralized in that each node can execute the control independently of other nodes.

Remark 1. In real-world scenarios, various factors may not be aligned with the problem statements. Two key factors are noncooperative populations and the use of piecewise constant control gain. Noncooperative populations arise when not everyone follows the proposed interventions, even if policymakers suggest measures to control the spread. However, our simulations (see Section 4) reveal that compliant populations can successfully address Problem 1, regardless of such noncooperative influences. Additionally, the efforts of compliant populations help lower the infected proportion for those not adhering to interventions. When applying the state feedback control, it may be more realistic to modify the value of the gain $g_i(k)$ only at a certain frequency rather than updating it at every discrete-time instant. Such a control approach will result in piecewise control gains and remains effective according to our simulations in Section 4.

Remark 2. In Section 4, we additionally consider the uncertainties arising when implementing the controller in real-world scenarios. Typical factors include noise and delays in measurements of infection, and uncertain updating periods in different populations. In an environment with noisy infection measurements, our proposed controller cannot address Problem 1, since the states cannot converge to an equilibrium, and therefore the second condition of Problem 1 is not satisfied. Nevertheless, simulation results show that our controller meets the first condition of Problem 1 through overestimating the current infection level, and then all infection levels stay below the desired bounds. In an environment with measurement delays, the results show that our solution can successfully address Problem 1 if the delays are small. However, if the measurement delays remain for a long time, then neither of the two conditions in Problem 1 is satisfied. For the uncertainties in the updating periods of the piecewise control gains, our simulations indicate that Problem 1 is solved when the updating periods vary within a small region. However, compared with the case of large fixed updating period, uncertainties in the intervals between updates have more negative effects on the system according to our simulations in Section 4.

Under the control law (5), the networked SIS model (4) becomes

$$x_i(k+1) = x_i(k) + h(1 - c_i x_i(k))\beta_i(1 - x_i(k))\sum_{j=1}^{n} a_{ij}x_j(k) - h\gamma_i x_i(k) \quad (6)$$

The controlled system (6) can be written in vector form as

$$\mathbf{x}(k+1) = [I + h\Xi(I - D \cdot \mathrm{diag}(\mathbf{x}(k))) \times (I - \mathrm{diag}(\mathbf{x}(k)))A - h\Gamma]\mathbf{x}(k) \quad (7)$$

where $D := \mathrm{diag}(c_i)$. We need the following assumption, which is a modified version of Assumption 4 for the sampling period h, to ensure that system (7) is well-defined.

Assumption 6. For each $i \in [n]$, the sampling period h fulfills $h\gamma_i + h\beta_i c_i \sum_{j=1}^{n} a_{ij} \leq 1$.

With Assumption 7 in place, we have the following result.

Lemma 1. *Consider the controlled system (7) under Assumptions 1, 5, and 6. Then $x_i(k) \in [0,1]$ for all $i \in [n]$ and $k \in \mathbb{Z}_+$.*

Proof. See Appendix section Proof of Lemma 1. □

It is worth noting that for the uncontrolled system (3), the set $[0,1]^n$ is positively invariant under Assumption 4 (see Lemma 1 in [26]). Moreover, in comparison, Assumption 6 requires a smaller sampling period h than Assumption 4 since $c_i \geq 1$.

Let us introduce some more notations. Let $\mathbf{v}$ be the eigenvector for RA corresponding to $\rho(RA)$ and v_i denote the i-th element of $\mathbf{v}$. By the Perron–Frobenius theorem [48], we know that $\mathbf{v} > \mathbf{0}$; here, we also scale it so that $\sum_{i\in[n]} v_i = 1$. Define $\alpha_i(k) := x_i(k)/v_i \geq 0, i \in [n]$. Let $\alpha_{\max}(k) := \max_{i\in[n]} \alpha_i(k)$, and $\overline{\alpha}_i := \alpha_{\max}(0)v_i$. We study a nontrivial disease-spread, that is, there exists at least one $i \in [n]$ such that $x_i(0) > 0$. This further implies that $\alpha_{\max}(0) > 0$, and hence $\overline{\alpha}_i > 0$. In the following subsections, we split the epidemic spreading into two cases [49].

3.1 | Weak Epidemic Spreading

In this subsection, we solve Problem 1 when $\mathcal{R}_0 \leq 1$. To do so, we need the following technical assumption on the sampling period h.

Assumption 7. For each $i \in [n]$, the sampling period h fulfills

$$h\beta_i \sum_{j=1}^{n} a_{ij} \leq \frac{1}{c_i} - \max\left\{ \frac{1}{c_i}\left(1 - \frac{1}{\rho(RA)}\right), \overline{x}_i^0, \hat{x}_i^0 \right\} \tag{8}$$

where

$$\overline{x}_i^0 := \frac{\left((c_i+1)\overline{\alpha}_i + 1\right) - \sqrt{[(c_i+1)\overline{\alpha}_i + 1]^2 - 4c_i\overline{\alpha}_i^2}}{2c_i\overline{\alpha}_i}$$

$$\hat{x}_i^0 := \frac{\left((c_i+1)\hat{\alpha}_i + 1\right) - \sqrt{[(c_i+1)\hat{\alpha}_i + 1]^2 - 4c_i\hat{\alpha}_i^2}}{2c_i\hat{\alpha}_i}$$

and $\hat{\alpha}_i := \overline{\alpha}_i \rho(RA)$.

Notice that $\overline{x}_i^0 < 1/c_i$, and similarly, $\hat{x}_i^0 < 1/c_i$. Hence, the right-hand side of (8) is positive, and thus we can always find h satisfying this condition. Assumption 7 is an additional assumption that is needed for solving Problem 1 using the proposed controller. We note that Assumption 4b is to guarantee that the states for the controlled system (7) remain within the region $[0,1]$. In the following propositions, we show that Assumption 7 guarantees the states in controlled system (7) to be strictly smaller than $1/c_i$. Combining Assumptions 4b and 7, we have $x_i(k) \in [0, 1/c_i)$ for controlled system (7).

With Assumption 7 in place, we have the following result.

Proposition 2. *Consider the controlled networked SIS system (7) under Assumptions 1, 2, 5, 6 and 7. If $\mathcal{R}_0 \leq 1$, then, for all $i \in [n]$ and $k \in \mathbb{Z}_+$, we have $0 \leq x_i(k) < 1/c_i$.*

Proof. See Appendix section Proof of Proposition 2. □

Note that under additional Assumptions 2 and 7, Proposition 2 shrinks the positive invariant set given in Lemma 1 from $[0,1]$ to $[0, 1/c_i)$ for all $i \in [n]$ and $k \in \mathbb{Z}_+$. However, Proposition 2 does not provide any guarantees regarding convergence to an equilibrium. From Proposition 1, we see that the uncontrolled system (3) has the stable DFE if $\mathcal{R}_0 \leq 1$. We would like to show that our controlled system exhibits convergence to the DFE, and we formally present it in the following proposition

Proposition 3. *Consider the controlled networked SIS system (7) under Assumptions 1, 2, 5, 6 and 7. If $\mathcal{R}_0 \leq 1$, then the DFE is asymptotically stable with the domain of attraction $[0, 1/c_i)$ for each $i \in [n]$.*

Proof. See Appendix section Proof of Proposition 3. □

Proposition 3 states that the state dynamics of the controlled system, regardless of the initial condition, converges to the DFE. The convergence results for $\mathcal{R}_0 \leq 1$ in Proposition 1 and Proposition 3 both indicate that the disease is eradicated eventually. However, we note that the controlled system has a faster eradication speed if the initial states as well as the networked epidemic parameters (including β_i, γ_i, and a_{ij}) are the same. An intuitive observation is that the control gain satisfies $g_i(k) = 1 - c_i x_i(k) \leq 1$, and the equality holds only when $x_i(k) = 0$. Thus, we know that the states in the controlled system is smaller than the states in the uncontrolled system (where the gain essentially satisfies $g_i(k) = 1$ for all $k \in \mathbb{Z}_+$). A comparison for convergence rates of the controlled and uncontrolled system dynamics is provided in Section 4.

Remark 3. Propositions 2 and 3 have the following interpretation. As long as the reproduction number is not larger than one, the fraction of infected would never exceed a prespecified bound. Therefore, assuming that the bound is commensurate with the healthcare facilities accessable for a population, the epidemic remains manageable in that the facilities do not get overwhelmed. As a comparison, even though the disease is eventually eliminated in uncontrolled system (3) if $\mathcal{R}_0 \leq 1$, it cannot ensure that the state trajectory of each node never exceeds the bound (see Section 4).

Note that for the uncontrolled system (3), the sufficient conditions (Assumptions 1–4) for asymptotic convergence to the DFE in Proposition 1 have been provided. While Proposition 3 provides a sufficient condition for convergence to the DFE, it does so under, among others, Assumption 7. It turns out that a similar convergence result can be obtained *without* Assumption 7, but with some additional conditions on c_i. This is established in the following proposition.

Proposition 4. *Consider the controlled networked SIS system (7) under Assumptions 1, 2, 5, and 6. If $\mathcal{R}_0 \leq 1$, then for any $1 < c_i < 3 + 2\sqrt{2}$, the DFE is asymptotically stable with the domain of attraction $[0, 1/c_i)$ for each $i \in [n]$.*

Proof. See Appendix section Proof of Proposition 4. □

3.2 | Strong Epidemic Spreading

In this subsection, we focus on the controlled networked epidemics for the strong epidemic case $\mathcal{R}_0 > 1$. A natural question is, whether the sufficient conditions in Propositions 2 and 3 can solve Problem 1 with $\mathcal{R}_0 > 1$. We first study the boundary of $x_i(k)$. It turns out that even when $\mathcal{R}_0 > 1$, the state trajectory of node i is bounded from above by $1/c_i$ for all times, under the same assumptions as in Proposition 2. We formally show this in the following proposition.

Proposition 5. *Consider the controlled system (7) under Assumptions 1, 2, 5, 6 and 7. If $\mathcal{R}_0 > 1$, then, for all $i \in [n]$ and $k \in \mathbb{Z}_+$, we have $x_i(k) < 1/c_i$.*

Proof. See Appendix section Proof of Proposition 5. □

We know that, from Proposition 1, when $\mathcal{R}_0 > 1$ the uncontrolled system (2) has two equilibria, namely the DFE $\mathbf{x}^* = \mathbf{0}$ and the endemic equilibrium $\mathbf{x}^* > \mathbf{0}$. Moreover, the endemic equilibrium for system (3) is globally asymptotically stable with the domain of attraction $[0, 1]^n \setminus \{\mathbf{0}\}$. We note that the stability result for the uncontrolled system (3) is proved in [28]. However, the approach in [28] cannot be applied directly for studying the existence of and the convergence to the endemic equilibrium for the controlled system (7). The main difficulty is that the Lyapunov candidate in the proof of Theorem 1 of [28] cannot be employed. Because of technical reasons, we restrict our analysis to a certain class of graphs.

Assumption 8. The weights of the graph $\mathcal{G}$ satisfy $(c_i - 1)a_{ii} > \sum_{j \neq i} a_{ij}$.

With Assumption 8, we provide a sufficient condition for the existence, uniqueness and convergence of the endemic equilibrium for (7).

Proposition 6. *Consider the controlled system (7) under Assumptions 1, 2, 5, 6, 7, and 8. If $\mathcal{R}_0 > 1$, then there exists a unique endemic equilibrium $\bar{\mathbf{x}}$ such that $0 < [\bar{\mathbf{x}}]_i < 1/c_i$ for each $i \in [n]$, and $c_i > 1$. Moreover, the endemic equilibrium is asymptotically stable with the domain of attraction $(0, 1/c_i)$.*

Proof. See Appendix section Proof of Proposition 6. □

Proposition 6 indicates that system (7) has two equilibria, namely, the DFE and the endemic equilibrium. Furthermore, the state asymptotically converges to $\bar{\mathbf{x}}$. At the endemic equilibrium, each population has a nontrivial proportion of infected which is strictly smaller than $1/c_i$.

Remark 4. Assumption 8 states that the weights of the interactions within a population should dominate those of the interactions outside the population. Such an assumption is consistent with what is often observed during epidemics: The frequency of interactions among individuals within a population is larger than that with individuals outside. In case of $(c_i - 1)a_{ii} \leq \sum_{j \neq i} a_{ij}$, we do not have a formal result as Proposition 6, and therefore the stability of endemic equilibrium under $\mathcal{R}_0 > 1$ is unclear. We leave the analysis of stability in case of $(c_i - 1)a_{ii} \leq \sum_{j \neq i} a_{ij}$ as an open problem in future research.

Remark 5. For the uncontrolled system (2), several procedures for computing the endemic equilibrium are available in the literature [9, 14]. Proposition 6 provides a sufficient condition for the existence of a unique endemic equilibrium $\bar{\mathbf{x}}$; it however makes no specific indications as to where in the state space $\bar{\mathbf{x}}$ would lie. Computing $\bar{\mathbf{x}}$ is beyond the scope of the present paper.

3.3 | Main Result

We are now ready to state the main result of the paper. It summarizes the results from Propositions 2–6. Recall that the controlled system (7) consists of the node dynamics in (6) and their local controls in (5).

Theorem 1. *Consider the controlled networked SIS system (6) under Assumptions 1, 2, 5, 6, 7and 8. The local state feedback control law (5) guarantees that $x_i(k) < 1/c_i$ for all $i \in [n]$ and $k \in \mathbb{Z}_+$. Furthermore, $\lim_{k\to\infty} x_i(k) = x_i^*$, where $x_i^* = 0$ if $\mathcal{R}_0 \leq 1$, and otherwise $x_i^* > 0$ for all $i \in [n]$.*

Proof. Define a vector $c := [c_1, c_2, \ldots, c_n]^\top$, where, for each $i \in [n]$, c_i is the prespecified upper bound on the infection level for node i. Note that for a given c where $\rho(M) \leq 1$, and $\rho(M) > 1$ fully characterize the behavior of system (6). For the case where $\rho(M) \leq 1$, Propositions 2 and 3 guarantee that for each $i \in [n]$ and for all k, $x_i(k) < 1/c_i$ for corresponding c_i, where $c_i > 1$, and $\lim_{k\to\infty} x_i(k) = 0$, respectively. Similarly, for the case where $\rho(M) > 1$, Propositions 5 and 6 guarantee that for each $i \in [n]$ and for all k, $x_i(k) < 1/c_i$ for corresponding $c_i > 1$, and $\lim_{k\to\infty} x_i(k) = x_i^*$, where $x_i^* > 0$, respectively. □

It is worthwhile to see how the endemic equilibrium of the controlled system (7) compares with that of the uncontrolled system (2).

Theorem 2. *Consider the controlled networked SIS system (7) under Assumptions 1, 2, 5, 6, 7 and 8. Let $\mathbf{x}^*$ and $\bar{\mathbf{x}}$ denote the unique endemic equilibria of the uncontrolled system (2) and the controlled system (7), respectively. If $\mathcal{R}_0 > 1$, then $\mathbf{x}^* > \bar{\mathbf{x}}$.*

Proof. See Appendix section Proof of Theorem 2. □

From Theorem 2, we see that, when the epidemic is persistent, the control strategy proposed in this paper leads to lower infection levels in each population.

The following remark highlights how our approach differs from other related works in control of SIS epidemics.

Remark 6. Technically, the feedback controllers in [27, 50] regard the recovery rate of each node as a local control input, that is, $\hat{\gamma}_i(t) = \gamma_i + u_i(t)$, where $\hat{\gamma}_i(t)$ is the new recovery rate achieved by, for instance, the administration of drugs, antibiotics, etc. In [50], the authors consider $u_i(t) = k_i x_i(t)$ with $k_i > 0$ and $\gamma_i = 0$. The work [27] studies a general class of feedback controller of the form $u_i(t) = g_i(x_i(t))$, where $g_i : [0, 1] \to \mathbb{R}_+$ is bounded,

smooth and monotonically nondecreasing, with $g_i(0) = 0$. On the other hand, in [34], the authors introduced a family of feedback controllers for both infection and recovery rates that adaptively evolve, and the controllers ensure that the state asymptotically converges to the DFE when $\mathcal{R}_0 > 1$. In contrast to these studies on continuous-time SIS models, our proposed feedback controller: a) operates within a discrete-time framework, so the magnitude of the sampling period significantly influences the control performance; b) incorporates prespecified upper boundary as a control objective for the node state trajectories; and c) treats the infection rate as the control variable, while differing from the form presented in [34].

4 | Numerical Example

In this section, we present several simulations to illustrate the effectiveness of the proposed control approach and also highlight its different characteristics.

The system settings used throughout the simulations are as follows. We work with a geometric random network of $n = 100$ population nodes. The nodes are randomly and uniformly located in an area of 100×100. Each node has a radius $r = 30$, within which it can interact with other nodes. The topology is depicted in Figure 1. We index the nodes from 1 to 100. The initial states are chosen such that $\mathbf{x}(0) \in [0, 0.1]^{100}$. We consider weights $a_{ij} \in [0, 1]$ satisfying Assumption 8 and normalize them according to the number of in-neighbors, ensuring $\sum_{i \in \mathcal{N}_i} a_{ij} = 1$.

Two different system settings will be used with the parameters shown in Table 1. The infection rates are randomly selected from the corresponding intervals in Table 1 such that we end up with an SIS model with heterogeneous transition rates, and we set the unit of the sampling period h as days. As a result, the two system

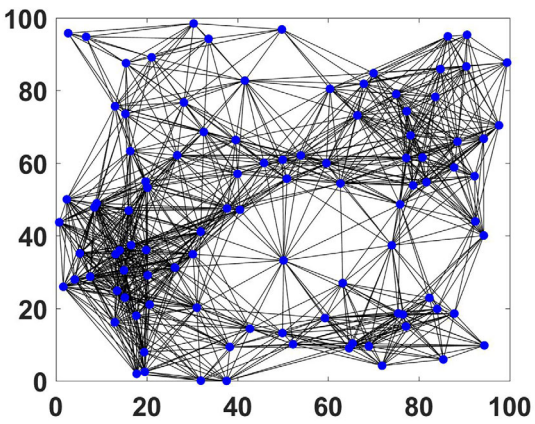

FIGURE 1 | Topology for all simulations.

TABLE 1 |

	β_i	γ_i	h (days)
Parameters I	(0.03, 0.04)	0.05	1
Parameters II	(0.08, 0.09)	0.01	1

settings have different properties. Under Parameters I, the reproduction number satisfies $\mathcal{R}_0 < 1$, whereas under Parameters II, we have $\mathcal{R}_0 > 1$. Additionally, we observe that Assumptions 1–4 are satisfied for the choices of model parameters in Parameters I (resp. Parameters II).

4.1 | Weak Epidemic Spreading

For the first simulation, we use the Parameters I (see the first line of Table 1) for all $i \in [100]$. This results in $\mathcal{R}_0 < 1$. The aim is to demonstrate that even when all the assumptions in Theorem 1 are satisfied and $\mathcal{R}_0 < 1$, the uncontrolled system cannot ensure that the state trajectory of each node remains under $1/c_i$. Then the time responses of the states of node 1 for uncontrolled system (3) are illustrated in Figure 2a. Notice that the initial state of node 1 is 0.04, which is below 0.05, and thus $x_i(0) \in [0, 1/20]$. If we choose $c_1 = 20$, then we have verified that Assumptions 5, 6, and 7 are satisfied. While keeping the other nodes uncontrolled, that is, $g_i(k) = 1$ for all $i \neq 1$, $k \in \mathbb{Z}_+$, we observe that the state of node 1 in the uncontrolled system (depicted by the black line) eventually converges to 0. However, during the time period $k \in [10, 70]$, it exceeds 0.05. In contrast, in the controlled system, the state of node 1 remains strictly below 0.05 and further converges to 0. With these simulations, we can verify the results of Propositions 2 and 3.

In the remaining simulations, we set the maximum local healthcare facility capacity $1/c_i$ for each population as follows: For $1 \leq i \leq 20$, we choose $1/c_i = 0.5$ (referred to as group 1); for $21 \leq i \leq 40$, we choose $1/c_i = 0.4$ (group 2); for $41 \leq i \leq 60$, we choose $1/c_i = 0.3$ (group 3); for $61 \leq i \leq 80$, we choose $1/c_i = 0.2$ (group 4); and for $81 \leq i \leq 100$, we choose $1/c_i = 0.1$ (group 5). All populations adopt the corresponding control law (5) unless otherwise specified. Given that the states of the uncontrolled system (3) and the controlled system (7) converge to the DFE if $\mathcal{R}_0 < 1$, we are interested in comparing the convergence speed between them. In this regard, we observe that the controlled states exhibit a faster convergence speed than the uncontrolled system, as depicted in Figure 2b. Moreover, populations with higher effectiveness of NPIs, indicated by a larger c_i, converge more rapidly to the DFE.

4.2 | Strong Epidemic Spreading

Next, we explore the controlled system with $\mathcal{R}_0 > 1$, by selecting Parameters II (see the second line of Table 1) to illustrate our theoretical results in Propositions 5, 6, and Theorem 2. In this parameter setting, it can be confirmed that the conditions in Assumptions 1, 2, 5, 6, 7, and 8 are fulfilled. Consistent with the result in Proposition 5, the states of the controlled system (7) are bounded from above by $1/c_i$, as shown in Figure 3a. Moreover, in line with Proposition 6, the states of the controlled system asymptotically converge to the endemic equilibrium. It is evident from Figure 3a that the endemic equilibrium of the controlled system (which is strictly smaller than 0.5) is smaller in every component than that of the uncontrolled system (depicted by the black lines settling at around 0.9). This observation is consistent with the result in Theorem 2.

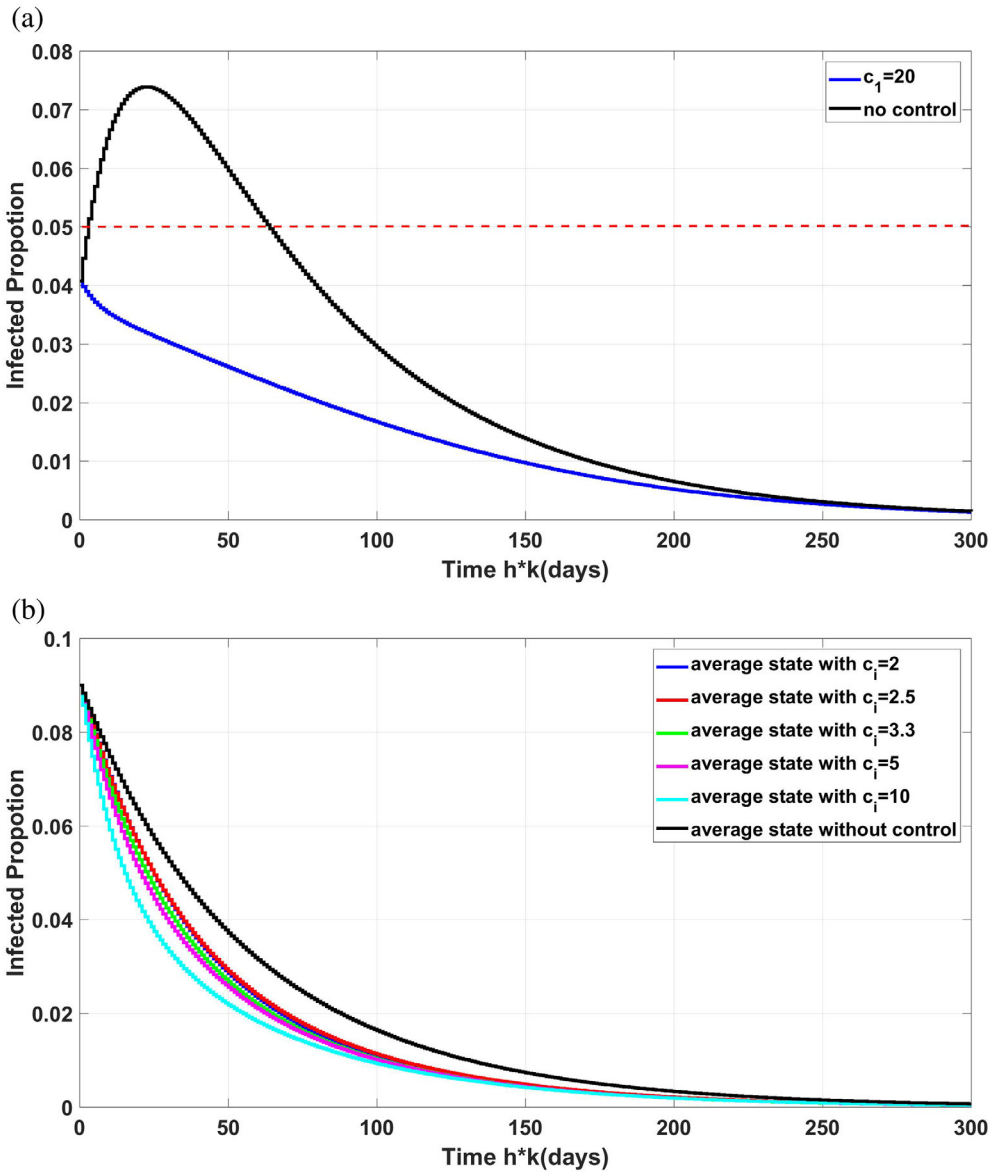

FIGURE 2 | Time responses when $\mathcal{R}_0 = 0.78 < 1$ (a) Time responses for node 1 when $\mathcal{R}_0 \leq 1$. (b) Time responses for systems (2) and (7) when $\mathcal{R}_0 \leq 1$.

4.3 | Effects of Uncertainties

In what follows, we examine the effects of various uncertainties in the system setting. These include uncertainties due to proportion of the population not adhering to the control law, noises and delays in measurements, as well as updating periods in the control gain. In all simulations, we focus on the scenario with Parameters II shown in Figure 3a.

4.3.1 | Noncooperative Population

In reality, even when policymakers propose NPIs to mitigate the spread, it is challenging to ensure that the entire populations adhere to these measures. Consequently, we examine the impact of noncooperative populations. We define population i as a noncooperative population if it does not adhere to the control law (5) and consistently sets $g_i(k) = 1$, indicating that no NPIs will be adopted in such a population. We randomly select five noncooperative populations, whereas the remaining population adheres to the control law (5). The time responses for all populations are illustrated in Figure 3b. The infected proportion in the noncooperative population exceeds its maximum local healthcare facility capacity, as indicated by the orange lines converging around 0.7. Conversely, the infected proportion for the compliant populations does not surpass the desired bound, and all converge to endemic equilibria. When compared with the time responses of the uncontrolled system (depicted by black lines converging around 0.9), the efforts of the compliant population contribute to lowering the endemic equilibrium for the noncooperative population.

4.3.2 | Measurement Noise

We next consider the imperfect state measurement $\hat{x}_i(k) = x_i(k) + w_i(k)$, where $w_i(k)$ is the bounded measurement noise with $|w_i(k)| \leq 0.1$. Here, a positive noise can be considered as

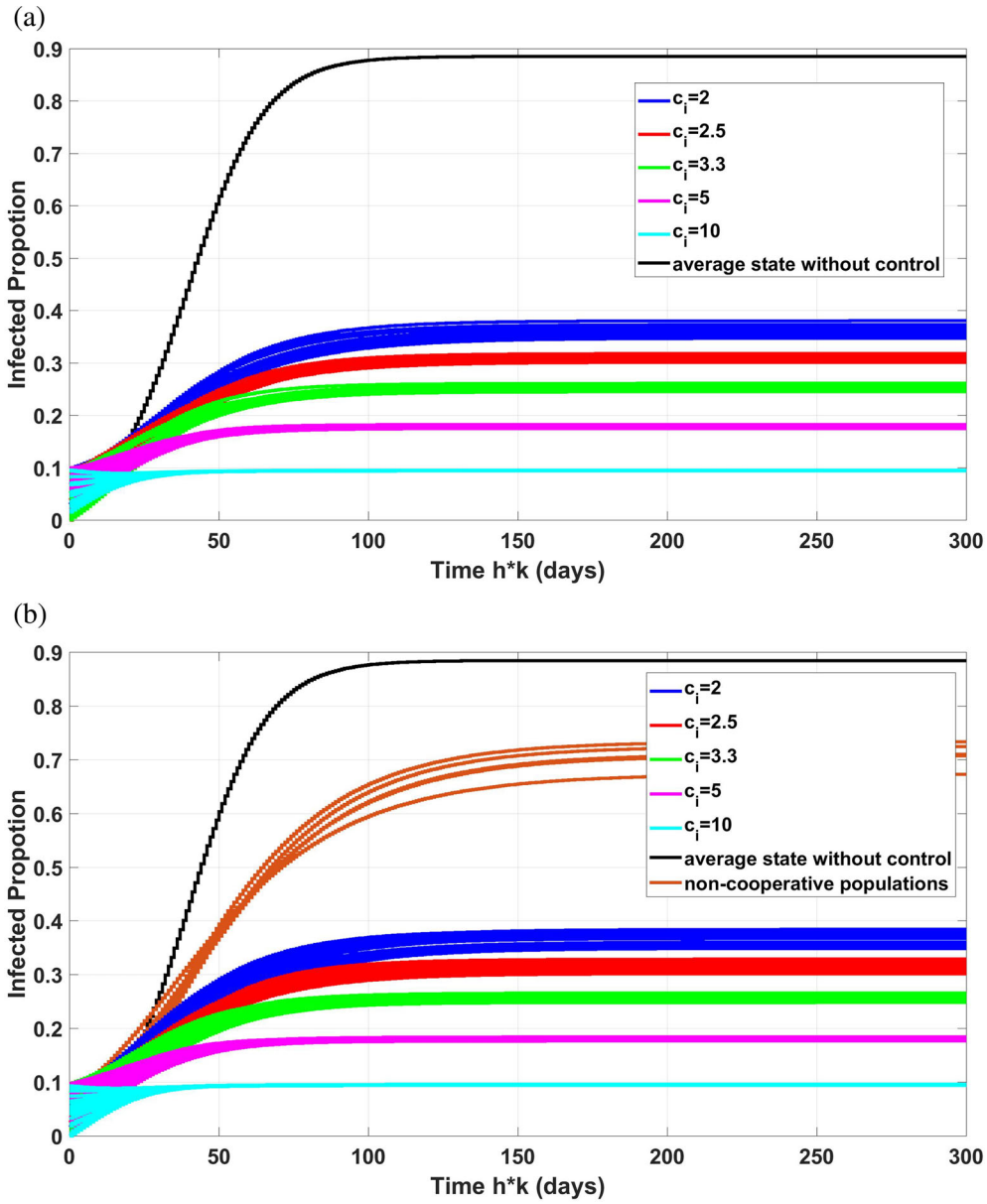

FIGURE 3 | Time responses when $\mathcal{R}_0 = 8.12 > 1$ (a) Time responses for systems (2) and (7) when $\mathcal{R}_0 > 1$. (b) Time responses for systems (2) and (7) when $\mathcal{R}_0 > 1$ with noncooperate populations.

an overestimation of the disease, and a negative noise is considered as an underestimation. Then, the control law (5) turns to be

$$g_i(k) = 1 - c_i \hat{x}_i(k) \tag{9}$$

Under a random positive noise $w_i(k) \in [0, 0.1]$ with uniform distribution, the resulting time responses for average states in groups 1–5 are displayed in Figure 4b. We see that the states continuously oscillate. However, the disease strictly stays below the prespecified levels for all time instants. As comparison, under a similar random but negative measurement noise $w_i(k) \in [-0.1, 0]$, the resulting time responses for average states in groups 1–5 are displayed in Figure 4b. Here, in addition to the oscillation, the states in groups 4 and 5 both exceed their local prespecified levels (0.2 for group 4 and 0.1 for group 5). Therefore, for the policymakers with uncertain infection measurement, overestimating the current infection level is better than underestimating it, since at least the infection level stays below the desired bound. In addition, the time responses displayed in Figure 4a,b show that the states in the group with larger c_i are oscillating with larger amplitudes. This indicates that the population with stricter NPIs (where c_i is large) is more fragile to the noise in infection measurement.

4.3.3 | Measurement Delays

As the third case of uncertainties, we examine the impact of delays in infection measurement since in practice the policymakers usually make decisions based on the historical data. In this case, the control law (5) becomes

$$g_i(k) = 1 - c_i x_i(k - \tau_i) \tag{10}$$

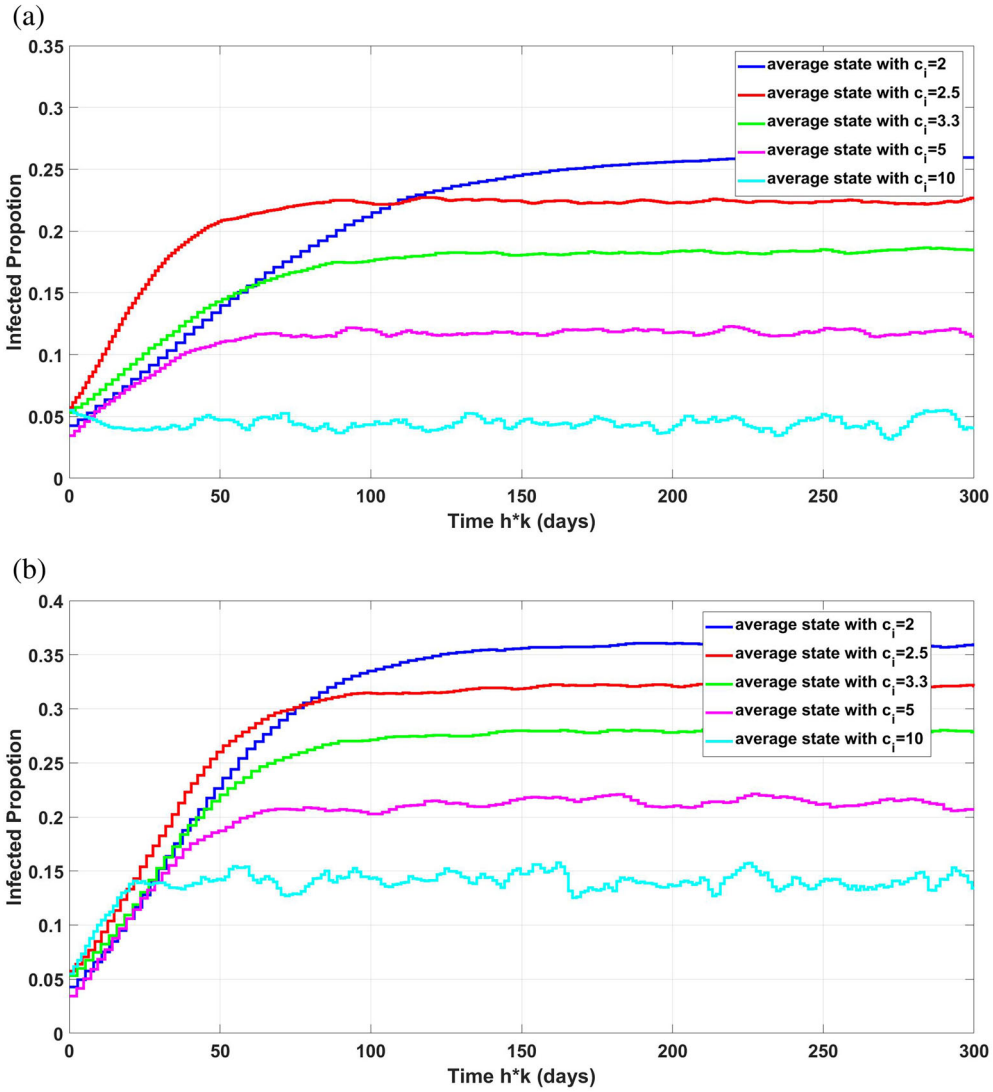

FIGURE 4 | Time responses with noisy infection measurement. (a) Time responses for systems (7) when $\mathcal{R}_0 > 1$ with positive noisy measurement. (b) Time responses for systems (7) when $\mathcal{R}_0 > 1$ with negative noisy measurement.

where $\tau_i \in \mathbb{Z}_+$ is the infection measurement delay in each population. We first examine small delays as $\tau_i \in [1, 8]$. The time responses of average states in groups 1–5 are depicted in Figure 5a. Here, we see that Problem 1 is successfully solved. If the measurement delays further increase as $\tau_i \in [8, 9]$, then the states in group 5 become unstable, as depicted in Figure 5b. As a point of reference, while policymakers can rely on recent data for decision making, it is advisable to refrain from utilizing overly outdated data. Another observation from Figure 5b is that the population with stricter NPIs is more fragile to the delays in infection measurements, since group 5 has the largest c_i.

4.3.4 | Updating Periods

Finally, we investigate the viability of employing piecewise constant and periodically updated control gains in the context of our discrete-time SIS networked model. While this scenario lacks theoretical results at present, we present preliminary simulations to illustrate that, even with appropriate periodic updating, the proposed method shows promise. For real-world implementations of public health interventions and measures to control epidemics, it is common practice for policymakers to implement certain interventions that remain in effect for extended durations, often spanning weeks or months. Observations are made on how these interventions impact the epidemic spreading process, and after reevaluation, new interventions are implemented (either more severe if the epidemic is still spreading strongly or less severe if the epidemic is receding). Implementing interventions gradually allows the population and medical staff to get used to them. Constantly changing interventions can create logistical challenges, so a phased approach helps smoother adaptation and understanding. Toward this end, we may adjust the dynamics in (5). In particular, with $T \in \mathbb{Z}_+$ being the updating period, we employ the control law given by

$$g_i(k) = 1 - c_i x_i(lT), \forall k \in [lT, (l+1)T), l \in \mathbb{Z}_+ \tag{11}$$

In other words, we update the control gain on the epidemic dynamics periodically, with a specific period length of T. Thus, $g_i(k)$ appears as a piecewise constant gain.

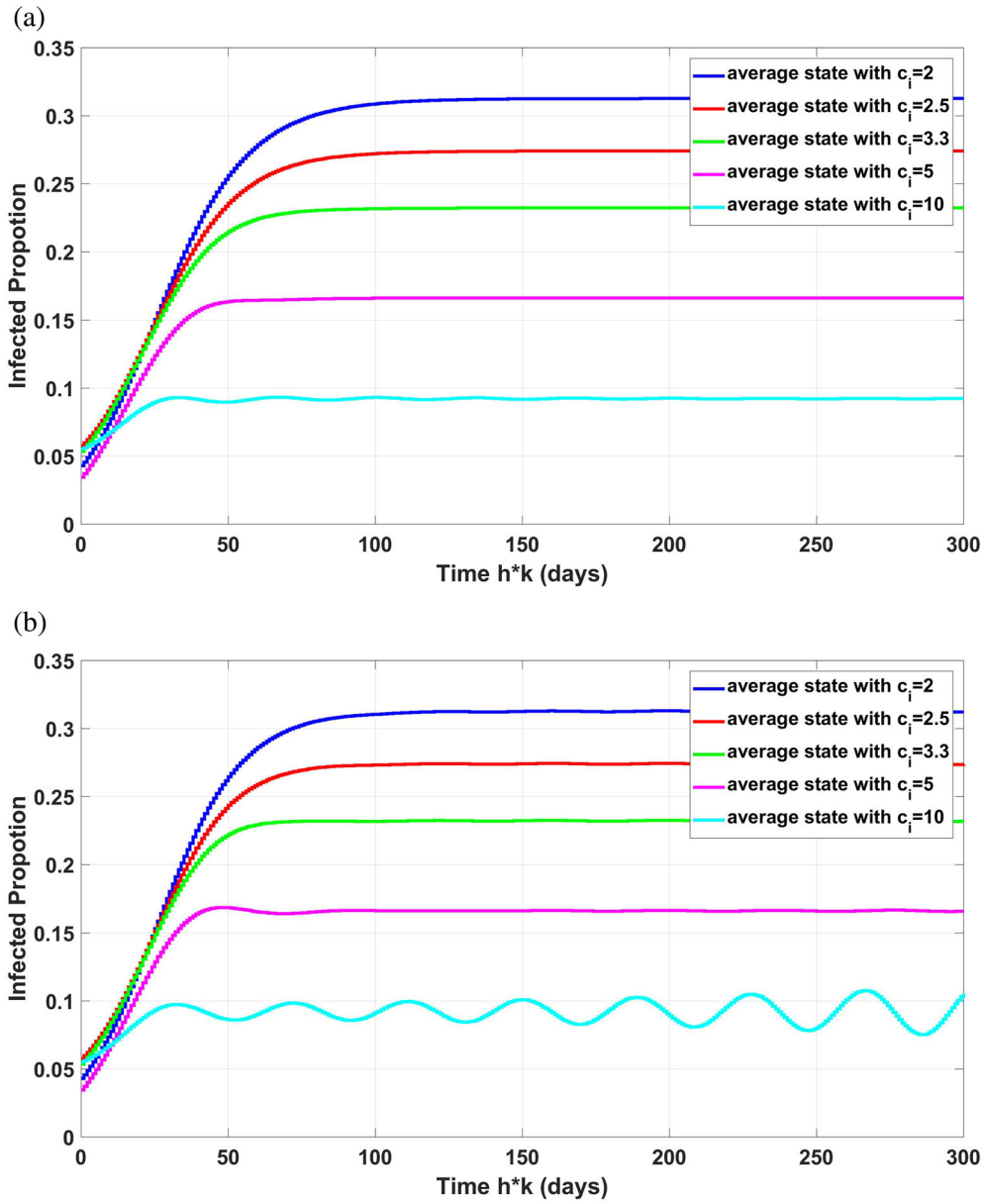

FIGURE 5 | Time responses with measurement delays. (a) Time responses for systems (7) when $\mathcal{R}_0 > 1$ with short measurement delay $\tau_i \in [1, 8]$. (b) Time responses for systems (7) when $\mathcal{R}_0 > 1$ with long measurement delay $\tau_i \in [9, 10]$.

In the simulation, we use this control law (11) with a period of $T = 12$. The resulting time responses for average states in groups 1–5 are displayed in Figure 6a. Here, we see that even with periodic updating of the gains, the disease is mitigated similarly as that in Figure 3a, where the period is $T = 1$. However, if we further increase the period to $T = 14$, then the system with the periodical control gain is unstable, as depicted in Figure 6b. The results presented here serve as a clear example that a poorly designed controller fails to address Problem 1 and cannot ensure the stability of the controlled systems.

Additionally, we discuss the effects of uncertain updating period T. Toward this end, we first choose the smaller uncertain updating period for groups 1-5, that is, $T_s \in [8, 10]$, for $s \in \{1, 2, 3, 4, 5\}$. Then the time responses of average states in groups 1–5 are shown in Figure 7a. Here, we see that the disease is mitigated similarly as that in Figure 3a. However, if the uncertain updating period is larger such that $T_s \in [10, 12]$, then the controlled system is unstable, and the states in group 5 exceed the desired bound, as we see in Figure 7b. An interesting observation is that the periodically controlled system addresses Problem 1 with large fixed updating period of $T = 12$, whereas the smaller but uncertain updating period of $T_s \in [10, 12]$ fails to solve the problem. Therefore, when policymakers have to implement their control laws with a large updating period, they should be careful with uncertainties in the period. As a comparison, groups 1–4

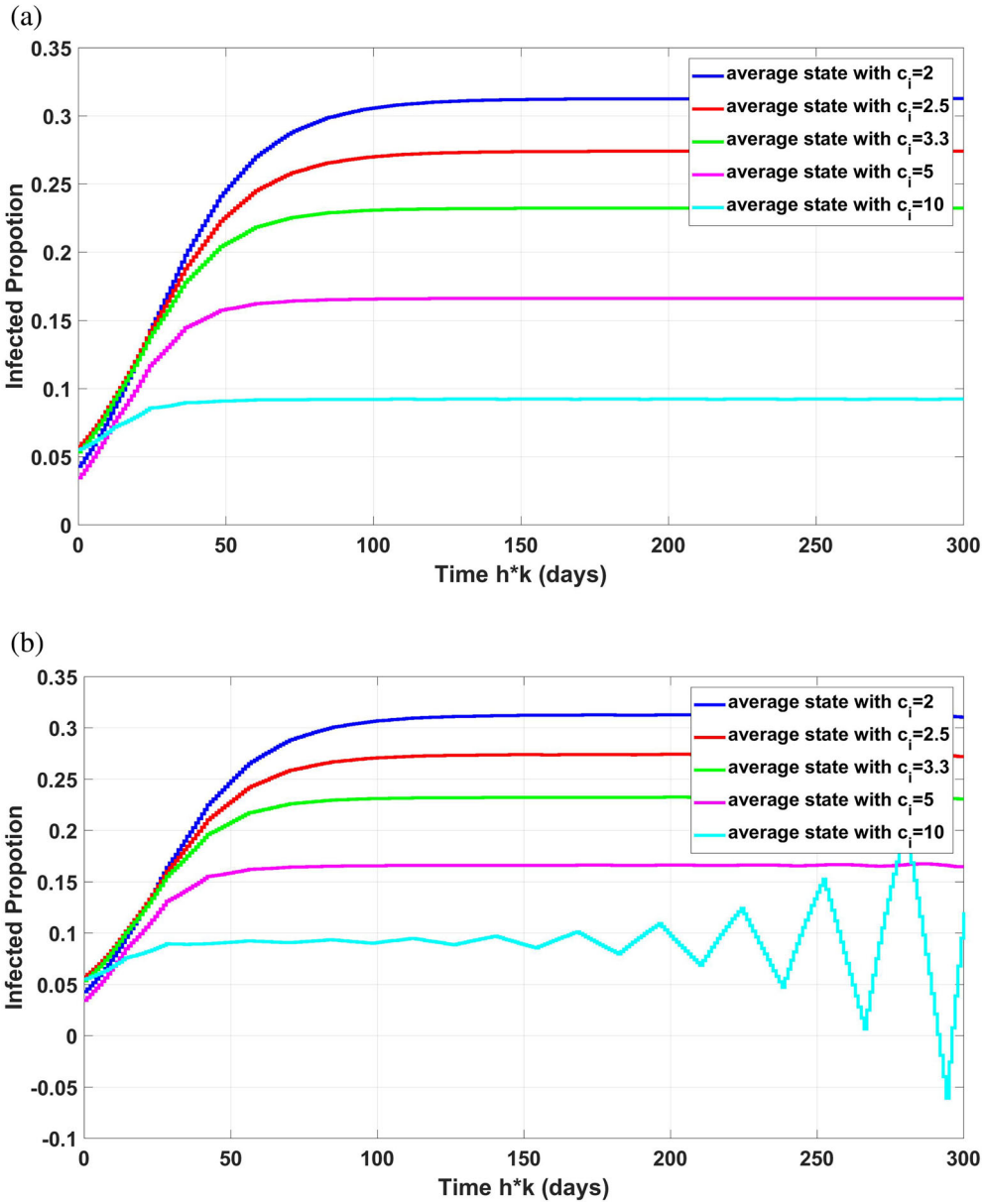

FIGURE 6 | Time responses when the updating period is long. (a) Time responses for systems (7) when $\mathcal{R}_0 > 1$ with large updating period $T = 12$. (b) Time responses for systems (7) when $\mathcal{R}_0 > 1$ with large updating period $T = 14$.

with small c_i solve Problem 1 under uncertain updating period of $T_s \in [10, 12]$. The results in Figure 7b clearly show that populations under stricter control laws are more sensitive to the uncertainties in the updating period and require policymakers to update their policies more frequently.

5 | Conclusion

This article studied the mitigation of discrete-time SIS epidemics spreading over a strongly connected network. We devised a local state feedback controller to guarantee that the infected proportion of each population stays below a prespecified level for all time instants. Furthermore, our controller guarantees that either the disease eventually becomes eradicated or, if it is persistent, the state converges to a unique endemic equilibrium, where the infected proportion of each population is not only strictly less than a prespecified level but also less than the endemic equilibrium of the uncontrolled system. One future work would be to extend the proposed approach to settings where multiple viruses are simultaneously active in each population. Another direction of interest would be to admit time-varying graphs and to consider edge removal or edge weight adjustment as an alternative control approach to address the proposed problem. Technically, our future work will moreover involve extending the stability results when $\mathcal{R}_0 > 1$, without relying on Assumption 8. Additionally, further formulation of the cost for the proposed control actions and solving the optimal control problems are of interest, and control barrier functions proposed in [31] can possibly be applied.

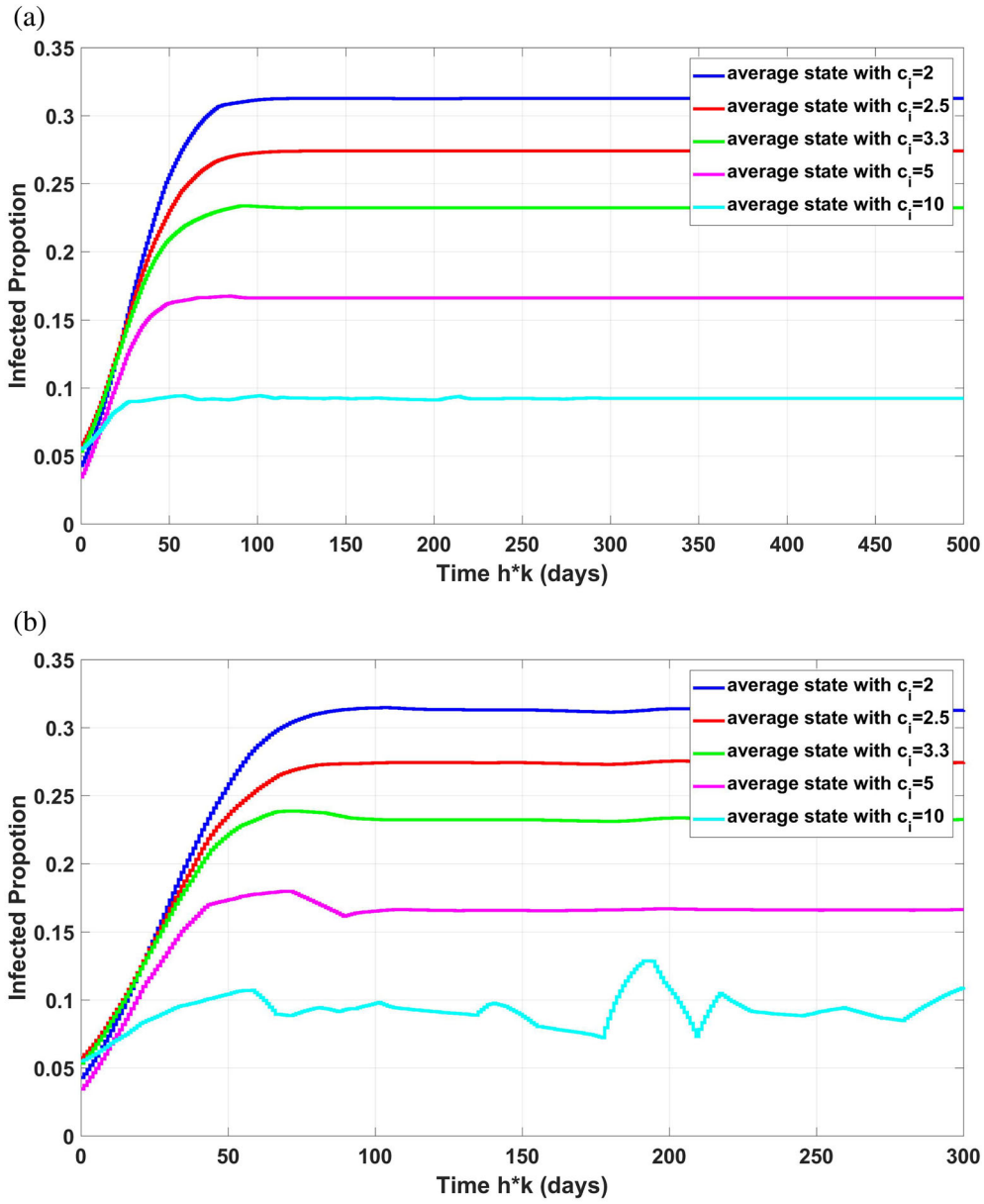

FIGURE 7 | Time responses when the updating period is uncertain. (a) Time responses for systems (7) when $\mathcal{R}_0 > 1$ with $T_s \in [8, 10]$. (b) Time responses for systems (7) when $\mathcal{R}_0 > 1$ with $T_s \in [10, 12]$.

Acknowledgments

This work was supported by National Natural Science Foundation of China Grant No. 92470202; by Provincial Natural Science Foundation of Hunan Grant No. 2024JJ6159; and by the Knut and Alice Wallenberg Foundation, Wallenberg Scholar under Grant 66469; by a Distinguished Professor Grant from the Swedish Research Council (Org: JRL, Project No: 3058); and by JSPS under Grant-in-Aid for Scientific Research Grant No. 22H01508.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

References

1. C. Jackson, "History Lessons: The Asian Flu Pandemic," *British Journal of General Practice* 59, no. 565 (2009): 622–623.

2. J. Piret and G. Boivin, "Pandemics Throughout History," *Frontiers in Microbiology* 11 (2021): 631736.

3. D. Bernoulli, "Essai d'une Nouvelle Analyse de la mortalité causée Par la Petite vérole, et Des Avantages de l'inoculation Pour la prévenir," *In Memoires de Mathematiques et de Physique. Academic Royale Des Science* (1760): 1–45.

4. N. Becker, "The Uses of Epidemic Models," *Biometrics* 35, no. 1 (1979): 295–305.

5. W. H. Hamer, "The Milroy Lectures on Epidemic Disease in England – The Evidence of Variability and Persistence of Type," *Lancet* 1 (1906): 733–739.

6. R. Ross, *The Prevention of Malaria*, 2nd ed. (Murray, 1911).

7. H. W. Hethcote, "The Mathematics of Infectious Diseases," *SIAM Review* 42, no. 4 (2000): 599–653.

8. J. Radcliffe and N. Bailey, *The Mathematical Theory of Infectious Diseases and Its Applications* (Charles Griffin & Company Ltd, 1975).

9. P. Van Mieghem, J. Omic, and R. Kooij, "Virus Spread in Networks," *IEEE/ACM Transactions on Networking* 17, no. 1 (2008): 1–14.

10. Y. Wang, D. Chakrabarti, C. Wang, and C. Faloutsos, "Epidemic Spreading in Real Networks: An Eigenvalue Viewpoint," in *Proceeding 22nd International Symposium on Reliable Distributed Systems* (IEEE, 2003), 25–34.

11. D. E. Bloom, D. Cadarette, and J. Sevilla, "Epidemics and Economics," *Finance & Development* 55 (2018): 2.

12. M. Gattoa, E. Bertuzzob, L. Maria, et al., "Spread and Dynamics of the COVID-19 Epidemic in Italy: Effects of Emergency Containment Measures," *Proceedings of the National Academy of Sciences of the United States of America* 117, no. 19 (2020): 10484–10491.

13. W. O. Kermack and A. G. McKendrick, "Contributions to the Mathematical Theory of Epidemics. II.–the Problem of Endemicity," *Proceedings of the Royal Society of London* 138, no. 834 (1932): 55–83.

14. W. Mei, S. Mohagheghi, S. Zampieri, and F. Bullo, "On the Dynamics of Deterministic Epidemic Propagation Over Networks," *Annual Reviews in Control* 44 (2017): 116–128.

15. A. Lajmanovich and J. A. Yorke, "A Deterministic Model for Gonorrhea in a Nonhomogeneous Population," *Mathematical Biosciences* 28, no. 3-4 (1976): 221–236.

16. M. E. Newman, "The Structure and Function of Complex Networks," *SIAM Review* 45, no. 2 (2003): 167–256.

17. A. Fall, A. Iggidr, G. Sallet, and J. J. Tewa, "Epidemiological Models and Lyapunov Functions," *Mathematical Modelling of Natural Phenomena* 2, no. 1 (2007): 62–83.

18. A. Khanafer, T. Başar, and B. Gharesifard, "Stability of Epidemic Models Over Directed Graphs: A Positive Systems Approach," *Automatica* 74 (2016): 126–134.

19. B. A. Prakash, H. Tong, N. Valler, M. Faloutsos, and C. Faloutsos, "Virus Propagation on Time-Varying Networks: Theory and Immunization Algorithms," in *Proceeding Joint European Conference on Machine Learning and Knowledge Discovery in Databases* (Springer, 2010), 99–114.

20. V. Bokharaie, O. Mason, and F. Wirth, "Spread of Epidemics in Time-Dependent Networks," in *Proceeding 19th International Symposium on Mathematical Theory of Networks and Systems–MTNS* (IFAC, 2010), 1717–1719.

21. S. Gracy, P. E. Paré, H. Sandberg, and K. H. Johansson, "Analysis and Distributed Control of Periodic Epidemic Processes," *IEEE Transactions on Control of Network Systems* 8, no. 1 (2020): 123–134.

22. P. E. Paré, S. Gracy, H. Sandberg, and K. H. Johansson, "Data-Driven Distributed Mitigation Strategies and Analysis of Mutating Epidemic Processes," in *Proceeding 59th IEEE Conference on Decision and Control* (IEEE, 2020a), 6138–6143.

23. L. J. Allen, "Some Discrete-Time SI, SIR, and SIS Epidemic Models," *Mathematical Biosciences* 124, no. 1 (1994): 83–105.

24. C. Peng, X. Jin, and M. Shi, "Epidemic Threshold and Immunization on Generalized Networks," *Physica A: Statistical Mechanics and its Applications* 389, no. 3 (2010): 549–560.

25. H. J. Ahn and B. Hassibi, "Global Dynamics of Epidemic Spread Over Complex Networks," in *Proceedings 52nd IEEE Conference on Decision and Control* (2013), 4579–4585.

26. P. E. Paré, J. Liu, C. L. Beck, B. E. Kirwan, and T. Başar, "Analysis, Estimation, and Validation of Discrete-Time Epidemic Processes," *IEEE Transactions on Control Systems Technology* 28, no. 1 (2020): 79–93.

27. M. Ye, J. Liu, B. D. O. Anderson, and M. Cao, "Applications of the Poincare-Hopf Theorem: Epidemic Models and Lotka-Volterra Systems," *IEEE Transactions on Automatic Control* 67, no. 4 (2022): 1609–1624.

28. F. Liu, S. Cui, X. Li, and M. Buss, "On the Stability of the Endemic Equilibrium of a Discrete-Time Networked Epidemic Model," *IFAC-PapersOnLine* 53, no. 2 (2020): 2576–2581.

29. V. M. Preciado, M. Zargham, C. Enyioha, A. Jadbabaie, and G. J. Pappas, "Optimal Resource Allocation for Network Protection Against Spreading Processes," *IEEE Transactions on Control of Network Systems* 1, no. 1 (2014): 99–108.

30. E. Ramírez-Llanos and S. Martínez, "Distributed Discrete-Time Optimization Algorithms With Applications to Resource Allocation in Epidemics Control," *Optimal Control Applications and Methods* 39, no. 1 (2018): 160–180.

31. B. A. Butler and P. E. Paré, "Optimal Safety-Critical Control of Epidemics," *IEEE Control Systems Letters* 7 (2023): 1819–1824.

32. J. C. Miller and J. M. Hyman, "Effective Vaccination Strategies for Realistic Social Networks," *Physica A: Statistical Mechanics and its Applications* 386 (2007): 780–785.

33. S. Jafarizadeh and D. Veitch, "Optimal Curing Resource Allocation for Epidemic Spreading Processes," *Automatica* 150 (2023): 110851.

34. L. Walsh, M. Ye, B. D. O. Anderson, and Z. Sun, "Decentralised Adaptive-Gain Control for Eliminating Epidemic Spreading on Networks," *Automatica* 174 (2025): 112143.

35. A. S. Bizyaeva, M. O. Arango, Y. Zhou, S. Levin, and N. E. Leonard, "Active Risk Aversion in SIS Epidemics on Networks," in *Proceedings American Control Conference* (2024), 4228–4233.

36. Y. Zhou, S. A. Levin, and N. E. Leonard, "Active Control and Sustained Oscillations in actSIS Epidemic Dynamics," *IFAC-PapersOnLine* 53, no. 5 (2020): 807–812.

37. M. Ogura and V. M. Preciado, "Epidemic Processes Over Adaptive State-Dependent Networks," *Physical Review* 93 (2016): 062316.

38. F. D. Rossa, D. Salzano, A. D. Meglio, et al., "A Network Model of Italy Shows That Intermittent Regional Strategies Can Alleviate the COVID-19 Epidemic," *Nature Communications* 11 (2020): 5106.

39. Y. Wang, S. Gracy, H. Ishii, and K. H. Johansson, "Suppressing the Endemic Equilibrium in SIS Epidemics: A State Dependent Approach," in *Proceedings 11th IFAC Symposium on Biological and Medical Systems* (IFAC, 2021), 163–168.

40. A. Berman, *Plemmons RJ* (SIAM, 1994).

41. World Health Organization, "Coronavirus (COVID-19) Outbreak," 2023, https://www.who.int/westernpacific/emergencies/covid-19.

42. J. Snow, *On the Mode of Communication of Cholera* (John Churchill, 1855).

43. C. Nowzari, V. M. Preciado, and G. J. Pappas, "Analysis and Control of Epidemics: A Survey of Spreading Processes on Complex Networks," *IEEE Control Systems Magazine* 36, no. 1 (2016): 26–46.

44. P. E. Paré, D. Vrabac, H. Sandberg, and K. H. Johansson, "Analysis, Online Estimation, and Validation of a Competing Virus Model," *Proceedings American Control Conference* (2020c): 2556–2561.

45. S. Moore, E. M. Hill, M. J. Tildesley, L. Dyson, and M. J. Keeling, "Vaccination and Non-pharmaceutical Interventions for COVID-19: A Mathematical Modelling Study," *Lancet Infectious Diseases* 21 (2021): 793–802.

46. G. N. Wong, Z. J. Weiner, A. V. Tkachenko, A. E. Elbanna, S. Maslov, and N. Goldenfeld, "Modeling COVID-19 Dynamics in Illinois Under Nonpharmaceutical Interventions," *Physical Review X* 10, no. 5 (2020): 041033.

47. H. Tian, Y. Liu, Y. Li, et al., "An Investigation of Transmission Control Measures During the First 50 Days of the COVID-19 Epidemic in China," *Science* 368 (2020): 638–642.

48. R. Varga, *Matrix Iterative Analysis* (Springer, 2000).

49. A. C. Fowler and T. D. Hollingsworth, "Simple Approximations for Epidemics With Exponential and Fixed Infectious Periods," *Bulletin of Mathematical Biology* 77 (2015): 1539–1555.

50. J. Liu, P. E. Paré, A. Nedić, C. Y. Tang, C. L. Beck, and T. Başar, "Analysis and Control of a Continuous-Time Bi-Virus Model," *IEEE Transactions on Automatic Control* 64, no. 12 (2019): 4891–4906.

51. A. Rantzer, "Distributed Control of Positive Systems," in *Proceeding 50th IEEE Conference on Decision and Control and European Control Conference* (IEEE, 2011), 6608–6611.

52. M. Vidyasagar, *Nonlinear Systems Analysis, SIAM Review* (SIAM, 2002).

53. M. A. Khamsi and W. A. Kirk, *An Introduction to Metric Spaces and Fixed Point Theory* (Wiley, 2001).

54. Z. Qu, *Cooperative Control of Dynamical Systems: Applications to Autonomous Vehicles* (Springer, 2009).

Appendix A

Proof of Proposition 1. We now introduce Theorems 1, 2 and Proposition 2 in [26], as well as Theorem 1 in [28] and state them as four lemmas in the sequel. □

Lemma 2. ([26]). *Suppose that Assumptions 1–4 hold for (3), If* $\mathcal{R}_0 \le 1$, *then the DFE is asymptotically stable with the domain of attraction* $[0,1]^n$.

Lemma 3. ([26]). *Under Assumptions 1 – 4, the DFE is the unique equilibrium of (3) if and only if* $\mathcal{R}_0 \le 1$.

Lemma 4. ([26]). *Let Assumptions 1 – 4 hold. If* $\mathcal{R}_0 > 1$, *then (3) has two equilibria,* $\mathbf{0}$ *and* $\mathbf{x}^*$, *where* $\mathbf{x}^* \gg 0$.

Lemma 5. ([28]). *Suppose that Assumptions 1 – 4 hold. Given the networked discrete-time SIS model with* $\mathcal{R}_0 > 1$ *on graph* $\mathcal{G}$, *the endemic equilibrium is asymptotically stable with the domain of attraction* $[0,1]^n \setminus \mathbf{0}$ *and the DFE is stable if and only if* $\mathbf{x}(0) = \mathbf{0}$.

Clearly, we have Proposition 1 from Lemmas 2–5.

Proof of Lemma 1. We use an induction argument. Observe that, from Assumption 5, $x_i(0) \in [0,1]$ for each $i \in [n]$. We assume that for all $k \le k'$ $x_i(k) \in [0,1]$ for each $i \in [n]$. We must show that, for each $i \in [n]$, $x_i(k'+1) \in [0,1]$. As the first step, we show $x_i(k'+1) \le 1$. To this end, from (6), we have

$$\begin{aligned} x_i(k'+1) &= (1-h\gamma_i)x_i(k') + h\beta_i \\ &\quad \times \sum_{j=1}^{n} a_{ij}x_j(k')(1-x_i(k'))(1-c_ix_i(k')) \\ &\le (1-h\gamma_i)x_i(k') + h\beta_i \sum_{j=1}^{n} a_{ij}(1-x_i(k')) \qquad \text{(A1)} \\ &< x_i(k') + \big(1-x_i(k')\big) = 1 \qquad \text{(A2)} \end{aligned}$$

The inequality (A1) is a consequence of $1 - c_ix_i(k') \le 1$ and $x_j(k') \le 1$, whereas the inequality in (A2) comes from the fact that from Assumptions 1 and 6 we have $h\gamma_i \le 1$ and $h\beta_i \sum_{j=1}^{n} a_{ij} < 1$. Finally, by inductive assumption, $x_i(k') \in [0,1]$, we obtain $x_i(k'+1) < 1$.

As the second step, we show that $x_i(k'+1) \ge 0$. We split this into two cases. First, suppose that $0 \le x_i(k') < 1/c_i$. Then from (6), and since Assumption 6 also implies $h\gamma_i \le 1$, we have $x_i(k'+1) \ge (1 - h\gamma_i)x_i(k') \ge 0$. Next, suppose that $1/c_i \le x_i(k') \le 1$. Together with inductive assumption, we have

$$\begin{aligned} x_i(k'+1) &\ge (1-h\gamma_i)x_i(k') - (c_i-1)h\beta_i \sum_{j=1}^{n} a_{ij}x_i(k') \\ &= (1-h\gamma_i - (c_i-1)h\beta_i \sum_{j=1}^{n} a_{ij})x_i(k') \\ &> (1-h\gamma_i - c_ih\beta_i \sum_{j=1}^{n} a_{ij})x_i(k') \ge 0. \end{aligned}$$

Hence, for system (7), $x_i(k) \in [0,1]$, $\forall i \in [n], \forall k \in \mathbb{Z}_+$.

Proof of Proposition 2. We now introduce a lemma needed in the sequel. □

Lemma 6. *Under Assumption 2,* $\rho(M) \le 1$ *if and only if* $\rho(RA) \le 1$.

Proof. Note that, from Assumption 2, the matrix RA is irreducible nonnegative. Recall that $\mathbf{v}$ denotes the eigenvector of RA associated with the eigenvalue $\rho(RA)$. Suppose that $\rho(RA) \le 1$. Since Γ is a positive diagonal matrix, we have $I + h\Gamma(\rho(RA) - 1) \le I$. Hence,

$$(I + h\Gamma(\rho(RA) - 1))\mathbf{v} \le \mathbf{v}$$

Since $RA\mathbf{v} = \rho(RA)\mathbf{v}$, we have

$$(I + h\Gamma(RA - I))\mathbf{v} \le \mathbf{v}$$

We have that $M = I + h\Gamma(RA - I)$, and $M\mathbf{v} \le \mathbf{v}$. Hence, it follows that $\rho(M)\mathbf{v} \le \mathbf{v}$. Since M is irreducible nonnegative, $\rho(M) \le 1$.

Conversely, suppose that $\rho(M) \le 1$. From Assumption 2, we know that M is an irreducible nonnegative matrix. Thus we have a positive eigenvector $\mathbf{x}_{\rho(M)} > \mathbf{0}$ and

$$M\mathbf{x}_{\rho(M)} = (I + h\Gamma(RA - I))\mathbf{x}_{\rho(M)} \le \mathbf{x}_{\rho(M)}$$

where the inequality is a consequence of $\rho(M) \le 1$. Thus, $\Gamma(RA - I)\mathbf{x}_{\rho(M)} \le \mathbf{0}$. Since Γ is a positive diagonal matrix, Γ^{-1} exists, and, therefore, we have $RA\mathbf{x}_{\rho(M)} \le \mathbf{x}_{\rho(M)}$, which implies that $\rho(RA) \le 1$. □

Recall that $\mathbf{v}$ is the eigenvector corresponding to the largest eigenvalue of RA. From (6) we obtain

$$\begin{aligned} x_i(k+1) &= x_i(k) + h(\beta_i(1-c_ix_i(k))(1-x_i(k)) \times \sum_{j=1}^{n} a_{ij}x_j(k) - \gamma_ix_i(k)) \\ &= x_i(k) + h(\beta_i(1-c_ix_i(k))(1-x_i(k)) \times \sum_{j=1}^{n} a_{ij}\alpha_j(k)v_j - \gamma_ix_i(k)) \qquad \text{(A3)} \\ &\le x_i(k) + h\gamma_i([R]_{ii}(1-c_ix_i(k))(1-x_i(k))\alpha_{\max}(k) \times \sum_{j=1}^{n} a_{ij}v_j - x_i(k)) \qquad \text{(A4)} \\ &= x_i(k) + h\gamma_i((1-c_ix_i(k))(1-x_i(k)) \times \alpha_{\max}(k)\rho(RA)v_i - x_i(k)) \qquad \text{(A5)} \end{aligned}$$

where (A3) comes from the fact that $\alpha_j(k) = \frac{x_j(k)}{v_j}$, with v_j being the j-th element of the eigenvector $\mathbf{v}$. The inequality in (A4) is due to the fact that $\alpha_{\max}(k) = \max_{j\in[n]} \alpha_j(k)$. Since $RA\mathbf{v} = \rho(RA)\mathbf{v}$, it follows that $[R]_{ii}\sum_{j=1}^{n} a_{ij}v_j = \rho(RA)v_i$, and thus we obtain (A5).

By assumption, $\rho(M) \le 1$, and thus from Lemma 6, we know that $\rho(RA) \le 1$. Hence, from (A5), we have

$$\begin{aligned} x_i(k+1) &\le x_i(k) + h\gamma_i\left((1-c_ix_i(k))(1-x_i(k))\alpha_{\max}(k)v_i - x_i(k)\right) \qquad \text{(A6)} \\ &\le x_i(k) + h\gamma_i\left((1-x_i(k))\alpha_{\max}(k)v_i - x_i(k)\right) \\ &= (1-h\gamma_i)x_i(k) + h\gamma_i\alpha_{\max}(k)v_i - h\gamma_ix_i(k)\alpha_{\max}(k)v_i \\ &\le (1-h\gamma_i)\alpha_{\max}(k)v_i \end{aligned}$$

$$+ h\gamma_i\alpha_{\max}(k)v_i - h\gamma_i x_i(k)\alpha_{\max}(k)v_i = \alpha_{\max}(k)v_i - h\gamma_i x_i(k)\alpha_{\max}(k)v_i$$
$$< \alpha_{\max}(k)v_i \tag{A7}$$

Since $x_i(k+1) = \alpha_i(k+1)v_i$, we have that $\alpha_i(k+1) < \alpha_{\max}(k) < \alpha_{\max}(0)$ for each $i \in [n]$.

From (A6), and since $\overline{\alpha}_i = \alpha_{\max}(0)v_i > 0$, we have that

$$x_i(k+1) < x_i(k) + h\gamma_i\left((1 - c_i x_i(k))(1 - x_i(k))\overline{\alpha}_i - x_i(k)\right)$$

Recall that by Assumption 5, we have $x_i(0) < 1/c_i$ for each $i \in [n]$. Note that $\overline{x}_i^0 < 1/c_i$ from Assumption 7. In what follows, we will show that if $\overline{x}_i^0 < x_i(k) < 1/c_i$, then $x_i(k+1) < x_i(k)$, and if $0 \le x_i(k-1) < \overline{x}_i^0$ and $x_i(k) \ge \overline{x}_i^0$, then $x_i(k) < 1/c_i$ for each $i \in [n]$ and $k \in \mathbb{Z}_+$. Then, we have $0 \le x_i(k) < 1/c_i$ for each $i \in [n]$ and $k \in \mathbb{Z}_+$.

Case (i): Suppose that $\overline{x}_i^0 < x_i(k) < 1/c_i$. It is immediate that $(1 - c_i x_i(k))(1 - x_i(k))\overline{\alpha}_i - x_i(k) < 0$ from Assumption 7. Hence, it follows that $x_i(k+1) < x_i(k) < 1/c_i$.

Case (ii): Suppose that $x_i(k) < \overline{x}_i^0$. If $x_i(k)$ does not exceed the interval $(0, \overline{x}_i^0]$, it is clear that $0 < x_i(k) < 1/c_i$, $\forall k \in \mathbb{Z}_+$. Let k' denote the first time instant at which $x_i(k')$ exceeds the interval $(0, \overline{x}_i^0]$. This implies that $x_i(k'-1)$ is within the interval $(0, \overline{x}_i^0]$, and therefore $x_i(k'-1) \le \overline{x}_i^0$. From (6) we obtain $x_i(k') - x_i(k'-1) < h\beta_i\sum_{j=1}^n a_{ij}$, which, from Assumption 7, further implies that $x_i(k') - x_i(k'-1) < 1/c_i - \overline{x}_i^0$. Consequently, since $x_i(k'-1) < \overline{x}_i^0$, we obtain $x_i(k') < 1/c_i$.

Proof of Proposition 3. The proof is inspired by [26], Theorem 1. We first introduce two lemmas. □

Lemma 7. ([51]). *Suppose that M is an irreducible nonnegative matrix such that $\rho(M) < 1$. Then, there exists a positive diagonal matrix P such that $M^T PM - P \prec 0$.*

Lemma 8. ([26]). *Suppose that M is an irreducible nonnegative matrix such that $\rho(M) = 1$. Then, there exists a positive diagonal matrix P such that $M^T PM - P \prec 0$.*

Proof of Proposition 3. Due to Assumption 2 and $a_{ij} \ge 0$, we know that A is an irreducible nonnegative matrix. From Assumptions 1 and 6, it follows that M is irreducible nonnegative. We separately consider the cases $\rho(M) < 1$ and $\rho(M) = 1$.

Case (i) ($\rho(M) < 1$): From Lemma 7, we know that there exists a positive diagonal matrix P_1 such that $M^T P_1 M - P_1 \prec 0$. Consider the Lyapunov candidate $V_1(\mathbf{x}) = \mathbf{x}^T P_1 \mathbf{x}$. It is immediate that $V_1(\mathbf{x}) > 0$ for every $\mathbf{x} \ne \mathbf{0}$. Let $\Delta V_1(\mathbf{x}(k)) := V_1(\mathbf{x}(k+1)) - V_1(\mathbf{x}(k))$, $X(k) := \mathrm{diag}(\mathbf{x}(k))$, $B(k) := h\Xi X(k)(D + I - DX(k))$ and $\hat{M}(k) := M - B(k)A$. For $\mathbf{x}(k) \ne \mathbf{0}$,

$$\begin{aligned}
\Delta V_1(\mathbf{x}(k)) &= \mathbf{x}^T(k+1)P_1\mathbf{x}(k+1) - \mathbf{x}^T(k)P_1\mathbf{x}(k)\\
&= \mathbf{x}^T(k)\left(\hat{M}^T(k)P_1\hat{M}(k) - P_1\right)\mathbf{x}(k)\\
&= \mathbf{x}^T(k)\left((M - B(k)A)^T P_1(M - B(k)A) - P_1\right)\mathbf{x}(k)\\
&= \mathbf{x}^T(k)\big(M^T P_1 M - P_1 - M^T P_1 B(k)A\\
&\quad - A^T B^T(k)P_1 M + A^T B^T(k)P_1 BA\big)\mathbf{x}(k)
\end{aligned} \tag{A8}$$

Since $M^T P_1 M - P_1$ is negative definite, we have

$$\begin{aligned}
\Delta V_1(\mathbf{x}(k)) < \mathbf{x}^T(k)\big(&-M^T P_1 B(k)A - A^T B^T(k)P_1 M\\
&+A^T B^T(k)P_1 B(k)A\big)\mathbf{x}(k)
\end{aligned} \tag{A9}$$

Plugging $M = I + h\Xi A - h\Gamma$ into (A9), and due to Assumption 6, we have

$$\begin{aligned}
\Delta V_1(\mathbf{x}(k)) < \mathbf{x}^T(k)\big(&-P_1 B(k)A - h\Xi A^T P_1 B(k)A + h\Gamma P_1 B(k)A\\
&-A^T B^T(k)P_1 - h\Xi A^T B^T(k)P_1 A\\
&+h\Gamma A^T B^T(k)P_1 + A^T B^T(k)P_1 B(k)A\big)\mathbf{x}(k)
\end{aligned}$$

$$\begin{aligned}
&= \mathbf{x}^T(k)\big((h\Gamma - I)P_1 B(k)A + (h\Gamma - I)A^T B^T(k)P_1\\
&\quad - h\Xi A^T P_1 B(k)A - h\Xi A^T B^T(k)P_1 A + A^T B^T(k)P_1 B(k)A\big)\mathbf{x}(k)\\
&\le \mathbf{x}^T(k)\big(-h\Xi A^T P_1 B(k)A - h\Xi A^T B^T(k)P_1 A\\
&\quad + A^T B^T(k)P_1 B(k)A\big)\mathbf{x}(k)
\end{aligned} \tag{A10}$$

$$= \mathbf{x}^T(k)A^T\left(-h\Xi P_1 B(k) - h\Xi B^T(k)P_1 + B^T(k)P_1 B(k)\right)A\mathbf{x}(k) \tag{A11}$$

Note that (A10) holds since P_1 and $B(k)$ are both positive diagonal matrices and A is a nonnegative matrix. The term $h\Gamma - I$ is, due to Assumption 6, nonpositive. Since $B(k)$ and P_1 are diagonal and $P_1 B(k) = B(k)P_1$, from (A11),

$$\Delta V_1(\mathbf{x}(k)) < \mathbf{x}^T(k)A^T(-2h\Xi + B(k))P_1 B(k)A\mathbf{x}(k). \tag{A12}$$

Next we consider the matrix $\overline{B}(k) := -2h\Xi + B(k)$. Observe that

$$\begin{aligned}
\overline{B}(k) &= -2h\Xi + B(k)\\
&= -2h\Xi + h\Xi X(k)(D + I - DX(k))\\
&= h\Xi\left[X(k)(D + I - DX(k)) - 2I\right].
\end{aligned}$$

Clearly, $\overline{B}$ is a diagonal matrix and its i-th element is $[\overline{B}(k)]_{ii} = h\beta_i\left[-c_i x_i^2(k) + (c_i + 1)x_i(k) - 2\right]$.

As a consequence of Assumptions 1–7, from Proposition 2 we have

$$[\overline{B}(k)]_{ii} \le h\beta_i \cdot \left[-\frac{1}{c_i} + 1 + \frac{1}{c_i} - 2\right] = -1 < 0 \tag{A13}$$

which means that $\overline{B}$ is negative. Since A, P_1 and $B(k)$ are all nonnegative matrices, from (A12) we conclude that $\Delta V_1(\mathbf{x}(k)) < 0$. Therefore, from [52], $x_i(k)$ converges asymptotically to the DFE for this case.

Case (ii) ($\rho(M) = 1$): Due to Lemma 8, the condition $\rho(M) = 1$ guarantees the existence of a positive diagonal matrix P_2 such that $M^T P_2 M - P_2 \prec 0$. Consider the Lyapunov candidate given by $V_2(\mathbf{x}(k)) = \mathbf{x}^T(k)P_2\mathbf{x}(k)$. By an analysis similar to that for the case of $\rho(M) < 1$, we obtain

$$\Delta V_2(\mathbf{x}(k)) \le \mathbf{x}^T(k)A^T(-2h\Xi + B(k))P_1 B(k)A\mathbf{x}(k)$$

Clearly, if $\mathbf{x} = \mathbf{0}$, then $\Delta V_2(\mathbf{x}(k)) = 0$. Observe that the matrices P_1 and $B(k)$ are positive, and A are nonnegative, whereas from (A13) we know that the matrix $\overline{B}(k)$ is negative. Hence, it follows that $\Delta V_2(\mathbf{x}(k)) = 0$ if and only if $\mathbf{x} = \mathbf{0}$. Therefore, from [52] it follows that the state converges asymptotically to the DFE. □

Proof of Proposition 4. The proof is very similar to that of Proposition 3, and, in the interest of space, we only mention the key differences when $\rho(M) < 1$. Let $V_1(\mathbf{x}(k)) = \mathbf{x}^T(k)P_1\mathbf{x}(k)$ be the Lyapunov candidate, where P_1 is the same positive diagonal matrix as in the proof of Proposition 3. Then, the analysis is the same until (A12). Under Assumptions 1–6, if $1 < c_i < 3 + 2\sqrt{2}$, then

$$[\overline{B}(k)]_{ii} \le h\beta_i \cdot \frac{c_i^2 - 6c_i + 1}{2c_i} < 0$$

where $\overline{B}(k)$ is as defined in the proof of Proposition 3. Hence, it is immediate that $\Delta V_1(\mathbf{x}(k)) < 0$ for all $\mathbf{x}(k) \ne \mathbf{0}$. □

Proof of Proposition 5. By similar reasoning as in the proof of Proposition 2, we obtain (A5). Let $\chi_i^0 := \max\{\frac{1}{c_i}\left(1 - \frac{1}{\rho(RA)}\right), \hat{x}_i^0\}$, and note that $\chi_i^0 < 1/c_i$ from Assumption 7. Recall that by Assumption 5, we have $x_i(0) < 1/c_i$ for each $i \in [n]$. In what follows, we will show that if $\chi_i^0 < x_i(k) < 1/c_i$, then $x_i(k+1) < x_i(k)$, and if $0 \le x_i(k-1) < \chi_i^0$ and $x_i(k) \ge \chi_i^0$, then $x_i(k) < 1/c_i$ for each $i \in [n]$ and $k \in \mathbb{Z}_+$. As a consequence, we have $0 \le x_i(k) < 1/c_i$ for each $i \in [n]$ and $k \in \mathbb{Z}_+$.

Case (i): Suppose that $\chi_i^0 < x_i(k) < 1/c_i$. Then, we have

$$x_i(k+1) < x_i(k) + h\gamma_i\left((1-x_i(k))\alpha_{\max}(k)v_i - x_i(k)\right) \tag{A14}$$

$$< \alpha_{\max}(k)v_i \tag{A15}$$

where (A14) comes from $\frac{1}{c_i}\left(1-\frac{1}{\rho(RA)}\right) < x_i(k) < 1/c_i$, which implies that $(1-c_ix_i(k))\rho(RA) < 1$. The inequality (A15) follows from the same reason as that which leads to inequality (A7) from (A6). Since $x_i(k+1) = \alpha_i(k+1)v_i$, we have $\alpha_i(k+1) < \alpha_{\max}(k) < \alpha_{\max}(0)$ for each $i \in [n]$. Since $\alpha_{\max}(k)v_i \le \alpha_{\max}(0)v_i = \overline{\alpha}_i$, from (A5), we have that $x_i(k+1) \le x_i(k) + h\gamma_i\left((1-c_ix_i(k))(1-x_i(k))\rho(RA)\overline{\alpha}_i - x_i(k)\right)$. From Assumption 7, it is immediate that if $\hat{x}_i^0 < x_i(k) < 1/c_i$, then $(1-c_ix_i(k))(1-x_i(k))\rho(RA)\overline{\alpha}_i - x_i(k) < 0$. Hence it follows that $x_i(k+1) < x_i(k) < 1/c_i$, if $\chi_i^0 < x_i(k) < 1/c_i$.

Case (ii): Suppose that $x_i(k) \le \chi_i^0$. If $x_i(k)$ does not exceed the interval $(0, \chi_i^0]$, it is clear that $x_i(k) < 1/c_i$, $\forall k \in \mathbb{Z}_+$. Let k' denote the first time instant at which $x_i(k')$ exceeds the interval. This implies that $x_i(k'-1)$ is within the interval, and therefore $x_i(k'-1) \le \chi_i^0$. From (6) we obtain, $x_i(k') - x_i(k'-1) < h\beta_i\sum_{j=1}^n a_{ij}$, which, from Assumption 7, further implies that $x_i(k') - x_i(k'-1) < 1/c_i - \chi_i^0$. Consequently, since $x_i(k'-1) < \chi_i^0$, we obtain $x_i(k') < 1/c_i$. □

Proof of Proposition 6. We separate the proof into three parts: existence of the endemic equilibrium, uniqueness of the endemic equilibrium and stability of the endemic equilibrium. The existence and uniqueness proofs are inspired by [17, 50] while the stability analysis is inspired by [28].

1. **Existence of an endemic equilibrium**

By (7), an equilibrium $\mathbf{x}^* = [x_1^*, x_2^*, \ldots, x_n^*]^T$ satisfies

$$\mathbf{x}^* = (I + h\Xi(I - DX^*)(I - X^*)A - h\Gamma)\mathbf{x}^*$$

where $X^* = \mathrm{diag}(\mathbf{x}^*)$. Hence, it follows that

$$(I - RA)\mathbf{x}^* = DRX^*X^*A\mathbf{x}^* - (I + D)RX^*A\mathbf{x}^*$$

Furthermore, we have $\left(I + \mathrm{diag}((D+I)RA\mathbf{x}^*) - \mathrm{diag}(DRA\mathbf{x}^*)X^*\right)\mathbf{x}^* = RA\mathbf{x}^*$. Let

$$H(\mathbf{x}^*) := I + \mathrm{diag}((D+I)RA\mathbf{x}^*) - \mathrm{diag}(DRA\mathbf{x}^*)X^*$$

It can be immediately seen that $H(\mathbf{x}^*)$ is a positive diagonal matrix with $[H(\mathbf{x}^*)]_{ii} \ge 1$, and as a consequence, $H^{-1}(\mathbf{x}^*)$ exists. Thus, we have

$$\mathbf{x}^* = H^{-1}(\mathbf{x}^*)RA\mathbf{x}^* \tag{A16}$$

By assumption, $\rho(M) > 1$. Hence, from Lemma 6 it follows that $\rho(RA) > 1$. By the Perron-Frobenius theorem for irreducible nonnegative matrices [48], there exists $\mathbf{v} > \mathbf{0}$ corresponding to $\rho(RA)$ such that $RA\mathbf{v} = \rho(RA)\mathbf{v}$.

We can choose a small ε satisfying for each $i \in [n]$, $0 < \varepsilon v_i < \frac{(c_i+1)\rho(RA) - \sqrt{(c_i-1)^2(\rho(RA))^2 + 4c_i\rho(RA)}}{2c_i\rho(RA)}$. Therefore, we obtain: $0 < 1 + (c_i+1)\rho(RA)\varepsilon v_i - c_i\rho(RA)(\varepsilon v_i)^2 < \rho(RA)$, and thus $\varepsilon v_i < \frac{\rho(RA)\varepsilon v_i}{1+(c_i+1)\rho(RA)\varepsilon v_i - c_i\rho(RA)(\varepsilon v_i)^2}$.

Since $[RA\mathbf{v}]_i = \rho(RA)v_i$, it follows that

$$\varepsilon v_i < \frac{[RA\varepsilon\mathbf{v}]_i}{1 + (c_i+1)[RA\varepsilon\mathbf{v}]_i - c_i[RA\varepsilon\mathbf{v}]_i \cdot \varepsilon v_i}$$

Hence, we have $\varepsilon\cdot\mathbf{v} < H^{-1}(\varepsilon\cdot\mathbf{v})RA(\varepsilon\cdot\mathbf{v})$.

Define the map $f_i(\mathbf{x}) : \mathbb{R}_+^n \to \mathbb{R}_+$ by

$$f_i(\mathbf{x}) = \frac{[RA\mathbf{x}]_i}{1 + (c_i+1)[RA\mathbf{x}]_i - c_i[RA\mathbf{x}]_i \cdot [\mathbf{x}]_i}$$

Since RA is an irreducible nonnegative matrix, we can check that with the domain of $(0,1]^n$, it holds $1 + (c_i+1)[RA\mathbf{x}]_i - c_i[RA\mathbf{x}]_i \cdot [\mathbf{x}]_i > 0$, Moreover, for any $\mathbf{x}_1 < \mathbf{x}_2$, we have $f_i(\mathbf{x}_1) < f_i(\mathbf{x}_2)$. It is immediate that for every $i \in [n]$,

$$f_i(\mathbf{1}) = \frac{[RA\mathbf{1}]_i}{1 + [RA\mathbf{1}]_i} < 1$$

Hence, $\mathbf{1} > H^{-1}(\mathbf{1})RA\cdot\mathbf{1}$. Based on Brouwer's fixed point theorem [53], we can find a fixed point $\overline{\mathbf{x}}$ of $f_i(\mathbf{x})$ that $f_i(\overline{\mathbf{x}}) = \overline{x}_i$, where $\overline{\mathbf{x}} = [\overline{x}_1, \overline{x}_2, \ldots, \overline{x}_n]$ and $\overline{x}_i \in (\varepsilon v_i, 1)$ is given by

$$\overline{x}_i = \frac{[RA\overline{\mathbf{x}}]_i}{1 + (c_i+1)[RA\overline{\mathbf{x}}]_i - c_i[RA\overline{\mathbf{x}}]_i \cdot \overline{x}_i}$$

Since a fixed point of $f_i(\mathbf{x})$ is equivalent to an equilibrium of node i of (7), and since $\overline{\mathbf{x}} = H^{-1}(\overline{\mathbf{x}})RA\overline{\mathbf{x}}$, we have that $\overline{\mathbf{x}}$ is the equilibrium of (7). Moreover, we have $\overline{x}_i \in (0,1)$ for each $i \in [n]$.

We next show that $0 < \overline{x}_i < 1/c_i$ for each $i \in [n]$ by contradiction. Suppose that for some $i_0 \in [n]$, $1/c_{i_0} \le \overline{x}_{i_0} < 1$. Each element of $\overline{\mathbf{x}}$ satisfies

$$\overline{x}_i = \overline{x}_i + h\left(\beta_i(1 - c_{i_0}\overline{x}_i)(1-\overline{x}_i)\cdot\sum_{j=1}^n a_{ij}\overline{x}_j - \gamma_i\overline{x}_i\right).$$

Then we have

$$\beta_i(1 - c_i\overline{x}_i)(1-\overline{x}_i)\sum_{j=1}^n a_{ij}\overline{x}_j - \gamma_i\overline{x}_i = 0. \tag{A17}$$

Since $1/c_{i_0} \le \overline{x}_{i_0} < 1$, we know that

$$\beta_{i_0}(1 - c_{i_0}\overline{x}_{i_0})(1-\overline{x}_{i_0})\sum_{j=1}^n a_{i_0 j}\overline{x}_j - \gamma_{i_0}\overline{x}_{i_0} < 0,$$

which means that $\overline{x}_{i_0}$ is not an endemic equilibrium. We obtain a contradiction of the assumption that $1/c_{i_0} < \overline{x}_{i_0} < 1$. Thus we have $0 < \overline{x}_i < 1/c_i$ for each $i \in [n]$.

1. **Uniqueness of the endemic equilibrium**

We prove uniqueness by a contradiction argument. Suppose that there is another endemic equilibrium $\overline{\mathbf{x}}^* = [\overline{x}_1^*, \overline{x}_2^*, \ldots, \overline{x}_n^*]$. Let $\zeta = \max_{i\in[n]}\frac{\overline{x}_i^*}{\overline{x}_i}$. We need to show that $\zeta \le 1$. By way of contradiction, assume that $\zeta > 1$. This implies that $\overline{\mathbf{x}}^* \le \zeta\overline{\mathbf{x}}$ and there exists an i_0 so that $\overline{x}_{i_0}^* = \zeta\overline{x}_{i_0}$. Then, for the node i_0, based on (A16) and $f(\overline{x}_{i_0}^*) = f(\zeta\overline{x}_{i_0})$, we have

$$\begin{aligned}\overline{x}_{i_0}^* &= \frac{[RA\overline{\mathbf{x}}^*]_{i_0}}{1 + (c_{i_0}+1)[RA\overline{\mathbf{x}}^*]_{i_0} - c_{i_0}[RA\overline{X}^*\overline{\mathbf{x}}^*]_{i_0}} \\ &= \frac{[RA\zeta\overline{\mathbf{x}}]_{i_0}}{1 + (c_{i_0}+1)[RA\zeta\overline{\mathbf{x}}]_{i_0} - c_{i_0}[RA\mathrm{diag}(\zeta\overline{\mathbf{x}})\zeta\overline{\mathbf{x}}]_{i_0}} \\ &= \frac{\zeta[RA\overline{\mathbf{x}}]_{i_0}}{1 + (c_{i_0}+1)\zeta[RA\overline{\mathbf{x}}]_{i_0} - c_{i_0}\zeta^2[RA\overline{X}\overline{\mathbf{x}}]_{i_0}}\end{aligned} \tag{A18}$$

Let

$$\begin{aligned}g(\overline{x}_{i_0}) &:= (1 + (c_{i_0}+1)\zeta[RA\overline{\mathbf{x}}]_{i_0} - c_{i_0}\zeta^2[RA\overline{X}\overline{\mathbf{x}}]_{i_0}) \\ &\quad - (1 + (c_{i_0}+1)[RA\overline{\mathbf{x}}]_{i_0} - c_{i_0}[RA\overline{X}\overline{\mathbf{x}}]_{i_0}) \\ &= (c_{i_0}+1)[RA\overline{\mathbf{x}}]_{i_0}(\zeta-1) - c_{i_0}[RA\overline{\mathbf{x}}]_{i_0}\cdot\overline{x}_{i_0}(\zeta-1)(\zeta+1) \\ &= [RA\overline{\mathbf{x}}]_{i_0}(\zeta-1)(c_{i_0} + 1 - c_{i_0}\zeta\overline{x}_{i_0} - c_{i_0}\overline{x}_{i_0})\end{aligned}$$

Since $\zeta\overline{x}_{i_0} \le 1$, $0 < \overline{x}_{i_0} < 1/c_{i_0}$ and $\zeta > 1$, we have

$$g(\overline{x}_{i_0}) \ge [RA\overline{\mathbf{x}}]_{i_0}(\zeta-1)(1 - c_{i_0}\overline{x}_{i_0}) > 0$$

Thus, from (A18), we have

$$\overline{x}_{i_0}^* < \frac{\zeta[RA\overline{\mathbf{x}}]_{i_0}}{1 + (c_{i_0}+1)[RA\overline{\mathbf{x}}]_{i_0} - c_{i_0}[RA\overline{X}\overline{\mathbf{x}}]_{i_0}} = \zeta\overline{x}_{i_0} = \overline{x}_{i_0}^*$$

Then we obtain a contradiction of the assumption that $\zeta > 1$, thus implying $\zeta \le 1$. This means that if there exists another equilibrium $\overline{\mathbf{x}}^*$, it must satisfy $\overline{\mathbf{x}}^* \le \overline{\mathbf{x}}$.

By exchanging the roles of $\overline{\mathbf{x}}^*$ and $\overline{\mathbf{x}}$, by a similar analysis as before, we obtain $\overline{\mathbf{x}}^* \ge \overline{\mathbf{x}}$. This implies $\overline{\mathbf{x}}^* = \overline{\mathbf{x}}$, thus concluding the proof of uniqueness.

1. **Stability of the endemic equilibrium**

If $\mathbf{x}(0) = \mathbf{0}$, it follows from (7) that $\mathbf{x}(k) = \mathbf{0}, \forall k \in \mathbb{Z}_+$. Thus if $\mathbf{x}(0) = \mathbf{0}$, the trajectories of $\mathbf{x}(k)$ stay at the origin and do not converge to the endemic equilibrium.

If $\mathbf{x}(0) \in [0, 1/c_i)^n \setminus \{\mathbf{0}\}$, let, for all $i \in [n]$, $y_i(k) = x_i(k) - \overline{x}_i$ and $\Delta y_i(k) = y_i(k+1) - y_i(k)$. Substituting $x_i(k) = y_i(k) + \overline{x}_i$ into (6) yields

$$\begin{aligned}
\Delta y_i(k) &= h\beta_i\big(1 - c_i\overline{x}_i - c_i y_i(k)\big)\big(1 - \overline{x}_i - y_i(k)\big) \\
&\quad \times \sum_{j=1}^{n} a_{ij}\big(y_j(k) + \overline{x}_j\big) - h\gamma_i\overline{x}_i - h\gamma_i y_i(k) \\
&= -h\gamma_i y_i(k) + h\beta_i\big(1 - c_i\overline{x}_i\big)\big(1 - \overline{x}_i\big) \times \sum_{j=1}^{n} a_{ij} y_j(k) + h\beta_i\big(c_i y_i^2(k) \\
&\quad + (2c_i\overline{x}_i - (c_i + 1))y_i(k)\big)\sum_{j=1}^{n} a_{ij} x_j(k) \\
&\quad + h\left(\beta_i(1 - c_i\overline{x}_i)(1 - \overline{x}_i)\sum_{j=1}^{n} a_{ij}\overline{x}_j - \gamma_i\overline{x}_i\right)
\end{aligned}$$

From (A17), and since $x_j(k) = y_j(k) + \overline{x}_j$, we have

$$\begin{aligned}
\Delta y_i(k) &= h\beta_i\big(1 - c_i\overline{x}_i\big)\big(1 - \overline{x}_i\big)\sum_{j=1}^{n} a_{ij} y_j(k) - h\gamma_i y_i(k) \\
&\quad + h\beta_i\left(c_i y_i^2(k) + (2c_i\overline{x}_i - (c_i + 1))y_i(k)\right) \times \sum_{j=1}^{n} a_{ij}(y_j(k) + \overline{x}_j)
\end{aligned} \tag{A19}$$

From (A19), we have the following:

$$\begin{aligned}
y_i(k+1) &= \left[(1 - h\gamma_i) + h\beta_i(2c_i\overline{x}_i - (c_i + 1))\sum_{j=1}^{n} a_{ij}\overline{x}_j\right] \\
&\quad \times y_i(k) + hc_i\beta_i\sum_{j=1}^{n} a_{ij}\overline{x}_j y_i^2(k) + h\beta_i\big[\big(1 - c_i\overline{x}_i\big)\big(1 - \overline{x}_i\big) + (2c_i\overline{x}_i \\
&\quad - (c_i + 1))y_i(k) + c_i y_i^2(k)\big]\sum_{j=1}^{n} a_{ij} y_j(k)
\end{aligned} \tag{A20}$$

From (A17), we have

$$\beta_i\sum_{j=1}^{n} a_{ij}\overline{x}_j = \frac{\gamma_i\overline{x}_i}{(1 - c_i\overline{x}_i)(1 - \overline{x}_i)}. \tag{A21}$$

Substituting (A21) into (A20), we have

$$\begin{aligned}
y_i(k+1) &= \left[(1 - h\gamma_i) + h\frac{\gamma_i(2c_i\overline{x}_i - (c_i + 1))\overline{x}_i}{(1 - c_i\overline{x}_i)(1 - \overline{x}_i)}\right] y_i(k) \\
&\quad + h\frac{c_i\gamma_i\overline{x}_i}{(1 - c_i\overline{x}_i)(1 - \overline{x}_i)} y_i^2(k) + h\beta_i[\big(1 - c_i\overline{x}_i\big)\big(1 - \overline{x}_i\big) \\
&\quad + (2c_i\overline{x}_i - (1 + c_i))y_i(k) + c_i y_i^2(k)]\sum_{j=1}^{n} a_{ij} y_j(k) \\
&= \left[1 - h\gamma_i \cdot \frac{1 - c_i\overline{x}_i^2}{(1 - c_i\overline{x}_i)(1 - \overline{x}_i)}\right] y_i(k) + h\gamma_i \\
&\quad \cdot \frac{c_i\overline{x}_i}{(1 - c_i\overline{x}_i)(1 - \overline{x}_i)} y_i^2(k) + h\beta_i\big[1 - c_i(y_i(k) + \overline{x}_i)\big] \\
&\quad \times \big[1 - (y_i(k) + \overline{x}_i)\big]\sum_{j=1}^{n} a_{ij} y_j(k) \\
&= \left[1 - h\gamma_i \cdot \frac{1 - c_i\overline{x}_i \cdot (\overline{x}_i + y_i(k))}{(1 - c_i\overline{x}_i)(1 - \overline{x}_i)}\right] y_i(k) \\
&\quad + h\beta_i\big[1 - c_i(y_i(k) + \overline{x}_i)\big]\big[1 - (y_i(k) + \overline{x}_i)\big] \times \sum_{j=1}^{n} a_{ij} y_j(k) \\
&= \left[1 - h\gamma_i \cdot \frac{1 - c_i\overline{x}_i x_i(k)}{(1 - c_i\overline{x}_i)(1 - \overline{x}_i)}\right] y_i(k) \\
&\quad + h\beta_i\big(1 - c_i x_i(k)\big)\big(1 - x_i(k)\big)\sum_{j=1}^{n} a_{ij} y_j(k)
\end{aligned} \tag{A22}$$

Note that (A22) holds since $x_i(k) = y_i(k) + \overline{x}_i$. Let $\mathbf{y}(k) = [y_1(k), y_2(k), \ldots, y_n(k)]^T$, and recall that $X(k) = \mathrm{diag}(\mathbf{x}(k))$, hence, we write (A22) in matrix form to obtain $\mathbf{y}(k+1) = \Phi(k)\mathbf{y}(k)$, where

$$\begin{aligned}
\Phi(k) &= I - h\Gamma\mathrm{diag}\left(\frac{1}{(1 - c_i\overline{x}_i)(1 - \overline{x}_i)}\right) + h\Gamma\mathrm{diag}\left(\frac{c_i\overline{x}_i}{(1 - c_i\overline{x}_i)(1 - \overline{x}_i)}\right) X(k) \\
&\quad + h\Xi(I - DX(k))(I - X(k))A.
\end{aligned}$$

Construct a matrix F such that

$$F = I - h\Gamma\mathrm{diag}\left(\frac{1}{(1 - c_i\overline{x}_i)(1 - \overline{x}_i)}\right) + h\Xi A.$$

We can see that $[F]_{ij} = h\beta_i a_{ij}, \forall i \ne j$ and

$$\begin{aligned}
[F]_{ii} &= 1 - h\gamma_i\frac{1}{(1 - c_i\overline{x}_i)(1 - \overline{x}_i)} + h\beta_i a_{ii} \\
&= 1 - h\beta_i\sum_{j=1}^{n} a_{ij}\frac{\overline{x}_j}{\overline{x}_i} + h\beta_i a_{ii} = 1 - h\beta_i\sum_{j\ne i} a_{ij}\frac{\overline{x}_j}{\overline{x}_i}.
\end{aligned}$$

We can find a $\mathbf{u} = \left[1, \frac{\overline{x}_2}{\overline{x}_1}, \frac{\overline{x}_3}{\overline{x}_1}, \ldots, \frac{\overline{x}_n}{\overline{x}_1}\right]^T$ such that $F\mathbf{u} = \mathbf{u}$. Note that matrix F is Metlzer since it has nonnegative off-diagonal elements. We must consider the following two cases based on the diagonal elements of F.

Case (i): If for each $i \in [n]$, $h\beta_i\sum_{j\ne i} a_{ij}\frac{\overline{x}_j}{\overline{x}_i} < 1$, then $[F]_{ii} > 0$. Therefore, F is irreducible nonnegative. By construction, $\mathbf{u} > \mathbf{0}$. Then we have $\rho(F) = 1$. Thus, by the Perron-Frobenius theorem for irreducible nonnegative matrices [48], Theorem 2.7, we can find a left eigenvector $\mathbf{v}^T > \mathbf{0}^T$ such that $\mathbf{v}^T F = \mathbf{v}^T$.

Case (ii): If there exists i such that $h\beta_i\sum_{j\ne i} a_{ij}\frac{\overline{x}_j}{\overline{x}_i} > 1$, then F is not necessarily an irreducible nonnegative matrix, and, hence the Perron-Frobenius theorem cannot be applied directly here. We construct another auxiliary matrix F' such that $[F']_{ij} = h'\beta_i a_{ij}, \forall i \ne j$, and $[F']_{ii} = 1 - h'\beta_i\sum_{j\ne i} a_{ij}\frac{\overline{x}_j}{\overline{x}_i}$, where h' is sufficiently small and meets case (i). We know that there exists a left eigenvector $\mathbf{v}^T > \mathbf{0}^T$ such that $\mathbf{v}^T F' = \mathbf{v}^T$. Let $\mathbf{v} = [v_1, v_2, \ldots, v_n]$, then we have

$$v_i\left(1 - h'\beta_i\sum_{j\ne i} a_{ij}\frac{\overline{x}_j}{\overline{x}_i}\right) + h'\sum_{j\ne i}\beta_j a_{ji} v_j = v_i.$$

Therefore, we have

$$\sum_{j\ne i}\beta_j a_{ji} v_j = v_i\beta_i\sum_{j\ne i} a_{ij}\frac{\overline{x}_j}{\overline{x}_i}. \tag{A23}$$

Then, based on (A23), we can show that for each $i \in [n]$,

$$[\mathbf{v}^T F]_i = v_i\left(1 - h\beta_i\sum_{j\neq i}a_{ij}\frac{\overline{x}_j}{\overline{x}_i}\right) + h\sum_{j\neq i}\beta_j a_{ji}v_j$$
$$= v_i + h\left(\sum_{j\neq i}\beta_j a_{ji}v_j - v_i\beta_i\sum_{j\neq i}a_{ij}\frac{\overline{x}_j}{\overline{x}_i}\right) = v_i.$$

Thus, in both Cases (i) and (ii), we have $\mathbf{v}^T F = \mathbf{v}^T$.

Construct an auxiliary system as follows

$$\mathbf{z}(k+1) = \Phi(k)\mathbf{z}(k) \tag{A24}$$

where $\mathbf{z}(0) = |\mathbf{y}(0)|$. Since $\Phi(k)$ is a nonnegative matrix, we have $\mathbf{z}(k) \geq \mathbf{0}$ and $-\mathbf{z}(k) \leq \mathbf{y}(k) \leq \mathbf{z}(k)$ for all k. Then $\mathbf{y}(k)$ is asymptotically stable if the origin is asymptotically stable for $\mathbf{z}(k)$. Consider the Lyapunov candidate $V(k) = \mathbf{v}^T\mathbf{z}(k)$, and it is easy to check that $V(k) \geq 0$ since $\mathbf{v}^T > \mathbf{0}^T$, $\mathbf{z}(k) \geq \mathbf{0}$. We note that $V(k) = 0$ only when $\mathbf{z}(k) = \mathbf{0}$. Then, we have

$$\begin{aligned} V(k+1) - V(k) &= \mathbf{v}^T(\mathbf{z}(k+1) - \mathbf{z}(k)) \\ &= \mathbf{v}^T(\Phi(k) - I)\mathbf{z}(k) \\ &= \mathbf{v}^T(\Phi(k) - F)\mathbf{z}(k) \end{aligned} \tag{A25}$$

We further discuss the properties of $\Phi(k) - F$. Note that

$$\Phi(k) - F = h\Gamma\mathrm{diag}\left(\frac{c_i\overline{x}_i}{(1-c_i\overline{x}_i)(1-\overline{x}_i)}\right)X(k) + h\Xi X(k)(DX(k) - (D+I))A.$$

Observe that $[\Phi(k) - F]_{ij} = h\beta_i(c_i x_i(k) - (c_i+1))a_{ij} < 0, i \neq j$. We next discuss the diagonal element $[\Phi(k) - F]_{ii}$:

$$\begin{aligned} [\Phi(k) - F]_{ii} &= hx_i(k)\left[\frac{c_i\gamma_i\overline{x}_i}{(1-c_i\overline{x}_i)(1-\overline{x}_i)} + \beta_i(c_i x_i(k) - (c_i+1))a_{ii}\right] \\ &= hx_i(k)\left[c_i\beta_i\sum_{j=1}^{n}a_{ij}\overline{x}_j + \beta_i(c_i x_i(k) - (c_i+1))a_{ii}\right] \quad \text{(A26)} \\ &< h\beta_i x_i(k)\left[\sum_{j=1}^{n}a_{ij} - c_i a_{ii}\right] \quad \text{(A27)} \\ &= h\beta_i x_i(k)\left[\sum_{j\neq i}a_{ij} - (c_i-1)a_{ii}\right] < 0 \quad \text{(A28)} \end{aligned}$$

We note that (A26) is obtained on (A21). Moreover, we know that $\overline{x}_i < 1/c_i, \forall i$ and $x_i(k) < 1/c_i$ based on Proposition 5, which indicates (A27). Based on Assumption 8, we have (A28). Then we know that $\Phi(k) - F$ is a matrix in which every element is negative. Hence, we have $V(k+1) - V(k) \leq 0$, where from (A25), the equality holds if and only if $\mathbf{z}(k) = 0$. Thus, from [52], the auxiliary system (A24) converges asymptotically to the origin. Hence, the endemic equilibrium is asymptotically stable for any non-zero initial condition that meets Assumption 5. □

Proof of Theorem 2. The proof is inspired by that of [27], Lemma 5. We need the following lemmas to prove the claim in Theorem 2.

Lemma 9. ([40]). *Suppose that $P \in \mathbb{R}^{n\times n}$ has all off-diagonal entries nonpositive. Then, the following statements are equivalent*:

1. *P is an M-matrix.*
2. *The eigenvalues of P have nonnegative real parts.*

Lemma 10. ([54]). *Suppose that P is a singular irreducible M-matrix. If Q is a nonnegative diagonal matrix with at least one positive diagonal element, then the eigenvalues of $P + Q$ have strictly positive real parts.*

Proof of Theorem 2. By (7) and Proposition 6, there exists a unique equilibrium $\overline{\mathbf{x}} = [\overline{x}_1, \overline{x}_2, \ldots, \overline{x}_n]^T > \mathbf{0}$ satisfying

$$\overline{\mathbf{x}} = \left(I + h\Xi(I - D\overline{X})(I - \overline{X})A - h\Gamma\right)\overline{\mathbf{x}}$$

where $\overline{X} = \mathrm{diag}(\overline{\mathbf{x}})$. Let $\alpha \in [0,1]$ be a variable, and we consider the following system:

$$\mathbf{x}(k+1) = \left[I + h\Xi(I - \alpha D\overline{X})(I - \overline{X})A - h\Gamma\right]\mathbf{x}(k).$$

By arguments similar to those in the proof of Proposition 6, we know that there exists a unique equilibrium $\mathbf{x}_\alpha^* > \mathbf{0}$ satisfying $\mathbf{x}_\alpha^* = (I + h\Xi(I - \alpha DX^*)(I - X^*)A - h\Gamma)\mathbf{x}_\alpha^*$. Notice that $\mathbf{x}_0^* = \mathbf{x}^*$, which is the endemic equilibrium of the uncontrolled system (2) and $\mathbf{x}_1^* = \overline{\mathbf{x}}$. Then, by arguments similar to those leading to (A16), we have

$$\left(H_\alpha(\mathbf{x}_\alpha^*) - RA\right)\mathbf{x}_\alpha^* = \mathbf{0} \tag{A29}$$

where $H_\alpha(\mathbf{x}_\alpha^*) := I + \mathrm{diag}\left((\alpha D + I)RA\mathbf{x}_\alpha^*\right) - \mathrm{diag}(\alpha DRA\mathbf{x}_\alpha^*)X_\alpha^*$. From (A29), we have

$$\left(H_\alpha(\mathbf{x}_\alpha^*) - RA\right)\frac{d\mathbf{x}_\alpha^*}{d\alpha} + \frac{d}{d\alpha}\left(H_\alpha(\mathbf{x}_\alpha^*)\right)\mathbf{x}_\alpha^* = \mathbf{0} \tag{A30}$$

The matrix $-H_\alpha(\mathbf{x}_\alpha^*) + RA$ is an irreducible Metzler matrix since the off-diagonal entries are all nonnegative. Then from [48], Lemma 2.3 and (A29), we have that 0 is the simple eigenvalue of $-H_\alpha(\mathbf{x}_\alpha^*) + RA$, and moreover, 0 is the largest real part among the eigenvalues. We conclude using Lemma 9 that $H_\alpha(\mathbf{x}_\alpha^*) - RA$ is a singular irreducible M-matrix.

The second term in the left-hand side of (A30) satisfies

$$\begin{aligned} \frac{d}{d\alpha}\left(H_\alpha(\mathbf{x}_\alpha^*)\right)\mathbf{x}_\alpha^* &= \mathrm{diag}\left(DRA\mathbf{x}_\alpha^*\right)\left(I - X_\alpha^*\right)\mathbf{x}_\alpha^* + [\mathrm{diag}(\alpha DRA\mathbf{x}_\alpha^*) \\ &\quad + \mathrm{diag}(RA\mathbf{x}_\alpha^*) - \mathrm{diag}(2\alpha DRA\mathbf{x}_\alpha^*)X_\alpha^*]\frac{d\mathbf{x}_\alpha^*}{d\alpha} \end{aligned} \tag{A31}$$

Define $G_\alpha := \mathrm{diag}(\alpha DRA\mathbf{x}_\alpha^*) + \mathrm{diag}(RA\mathbf{x}_\alpha^*) - \mathrm{diag}(2\alpha DRA\mathbf{x}_\alpha^*)X_\alpha^* = \mathrm{diag}(RA\mathbf{x}_\alpha^*)\left[\alpha D(I - X_\alpha^*) + (I - \mathrm{diag}(\alpha D\mathbf{x}_\alpha^*))\right]$. If $\alpha D \leq I$, then it is immediate that G_α is a positive diagonal matrix since $\mathbf{x}_\alpha^* < I$. If $\alpha D > I$, then, by arguments similar to those in (A17), we obtain a contradiction. Therefore, we have $[\mathbf{x}_\alpha^*]_i < 1/(\alpha c_i)$, which means $\mathrm{diag}(\alpha D\mathbf{x}_\alpha^*) < I$. Thus, G_α is a positive diagonal matrix.

Then from (A30) and (A31), we have $\left(H_\alpha(\mathbf{x}_\alpha^*) - RA + G_\alpha\right)\frac{d\mathbf{x}_\alpha^*}{d\alpha} = -\mathrm{diag}\left(DRA\mathbf{x}_\alpha^*\right)\left(I - X_\alpha^*\right)\mathbf{x}_\alpha^*$. Let $K_\alpha := H_\alpha(\mathbf{x}_\alpha^*) - RA + G_\alpha$. Based on Lemma 10, it follows immediately that K_α is a nonsingular M-matrix, which yields that $K_\alpha^{-1} > \mathbf{0}_{n\times n}$ [40], Theorem 2.7. This means that $\frac{d\mathbf{x}_\alpha^*}{d\alpha} = -K_\alpha^{-1}\mathrm{diag}\left(DRA\mathbf{x}_\alpha^*\right) \times \left(I - X_\alpha^*\right)\mathbf{x}_\alpha^* < \mathbf{0}$. Thus, we have $\mathbf{x}_0^* = \mathbf{x}^* > \overline{\mathbf{x}} = \mathbf{x}_1^*$.