Multi-Competitive Time-Varying Networked SIS Model with an Infrastructure Network

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Abstract

The paper studies the problem of the spread of multi-competitive viruses across a (time-varying) population network and an infrastructure network. To this end, we devise a variant of the classic (networked) susceptible-infected-susceptible (SIS) model called the multi-competitive time-varying networked susceptible-infected-water-susceptible (SIWS) model. We establish a sufficient condition for exponentially fast eradication of a virus when a) the graph structure does not change over time; b) the graph structure possibly changes with time, interactions between individuals are symmetric, and all individuals have the same healing and infection rate; and c) the graph is directed and is slowly-varying, and not all individuals necessarily have the same healing and infection rates. We also show that the aforementioned conditions for eradication of a virus are robust to variations in the graph structure of the population network provided the variations are not too large. For the case of time-invariant graphs, we give a lower bound on the number of equilibria that our system possesses. Finally, we provide an in-depth set of simulations that not only illustrate the theoretical findings of this paper but also provide insights into the endemic behavior for the case of time-varying graphs.

Keywords: Epidemic processes, Networked SIS models,

1. Introduction

Epidemics have been a longstanding feature of human civilization. Some of the most prominent examples include the Spanish flu 1918–1920, the Asian flu in the 1950s (Jackson, 2009), 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 economies worldwide by a few decades, and led to social unrest in several parts of the world (Johnson and Mueller, 2002). 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 analysed by Daniel Bernoulli (Bernoulli, 1760). Mathematical epidemiology, as a discipline, witnessed tremendous growth during the 20th century; see (Becker, 1979; Hamer, 1906; Ross, 1911; Hethcote, 2000; Bailey et al., 1975), with (Bailey et al., 1975) being a key milestone. In recent years, problems in epidemiology have been investigated by several disciplines such as physics (Van Mieghem et al., 2008), computer science (Wang et al., 2003), economics (Bloom et al., 2018), and automatic control (Nowzari et al., 2016). A fundamental quest behind such research efforts is to *understand* what causes a disease to spread, and *how* the spreading can be mitigated.

To address the aforementioned quest, several models have been developed in the literature. These models include, but are not limited to, susceptible-infected (SI), susceptible-infected-recovered (SIR), susceptible-infected-susceptible (SIS), and susceptible-exposed-infected-recovered (SEIR) models. SIS models have been studied since (Kermack and McKendrick, 1932); for a thorough review, see (Hethcote, 2000). Notice that, while each of the epidemic models has its inherent advantages, only the SIS class of models admits reinfection. As such, it has been useful for studying the spread of diseases such as gonorrhea (Lajmanovich and Yorke, 1976), tuberculosis (Newman, 2003), etc. The present paper deals with networked SIS models.

Networked SIS models have been analyzed extensively in the literature. Substantial progress has been made in the context of time-invariant networks; see, (Khanafer et al., 2016; Fall et al., 2007; Van Mieghem et al., 2008; Allen, 1994; Wang et al., 2003; Peng et al., 2010; Ahn and Hassibi, 2013; Paré et al., 2020b; Chakrabarti et al., 2008; Gómez et al., 2010; Han et al., 2015; Gracy et al., 2020; Paré et al., 2020a). Time-invariant networked SIS models, while contributing massively towards our understanding of epidemics, cannot account for more complex settings, in particular one in which the agents are allowed

☆Research supported in part by the Rice Academy of Fellows, Rice University, and the National Science Foundation grants #2032258, #2238388, #2211815, and #2213568.

to move. Overcoming this shortcoming, a series of papers, spanning across several scientific communities such as automatic control, physics, complex networks, etc., have tackled networked SIS models with time-varying topology; see (Barrett et al., 2008; Vestergaard and Génois, 2015; Sun et al., 2015; Liu et al., 2018; Prakash et al., 2010; Bokharaie et al., 2010; Paré et al., 2021). The findings in (Barrett et al., 2008; Vestergaard and Génois, 2015; Sun et al., 2015; Liu et al., 2018) have only been supported by simulations; no analytical results have been provided, which greatly hampers our understanding of their behavior, and, consequently, our ability to predict and reason about system dynamics. On the automatic control side, the vast majority of the literature on networked SIS epidemics (including (Prakash et al., 2010; Bokharaie et al., 2010; Paré et al., 2018)) is centered on the case where there is only one virus circulating in the population. Recently, however, there has been significant attention on the more general case where multiple viruses are *simultaneously* circulating in a population. In fact, several scientific communities such as physics (Sahneh and Scoglio, 2014; Huang et al., 2021; Sagar et al., 2018), complex networks (Karrer and Newman, 2011), network science (Baingana and Giannakis, 2016), mathematical biology (Martcheva, 2009), have made significant advances on the topic of simultaneous spread of multiple competing viruses. However, note that most of these works have certain limitations: The paper (Martcheva, 2009) does not account for the presence of more than one node; the findings in (Karrer and Newman, 2011; Huang et al., 2021; Sagar et al., 2018) are based on simulations, and no analytical results have been provided. The paper (Baingana and Giannakis, 2016), although dealing with spreading processes over time-varying networks, does not focus on questions pertaining to stability. The paper (Sahneh and Scoglio, 2014) studies competing SIS epidemics but over time-invariant networks only. The same can be said about most of the recent literature in the automatic control community; see, for instance, (Janson et al., 2020; Ye, Anderson and Liu, 2022; Liu et al., 2019*a*; Paré et al., 2020c; Ye et al., 2023; Anderson and Ye, 2023). Paré et al. (2021) were the first to explore multiple competing SIS viruses over time-varying networks.

All of this literature (except for (Paré et al., 2020c)) involves continuous-time SIS models. Disease outbreaks are frequently recorded in epidemiological reports, which are compiled per day (as was observed during the COVID-19 crisis) (World Health Organization, 2021; Snow, 1855) or per week WHO (n.d.). Hence, it is immediate that the continuous-time spread process is sampled at discrete time intervals. Said sampling of the system behavior motivates the need for a discrete-time SIWS model. Other advantages of employing a discrete-time model are as follows: i) a discrete-time model possibly enables an easier comparison of experimental data with the predictions of a model, provided these predictions are given in discrete form; and ii) the numerical exploration of discrete-time epidemic models is fairly straightforward and consequently can be immediately implemented by non-mathematicians. The latter is of immense importance in the context of public health (Brauer et al., 2009). The paper (Paré et al., 2020c), while shedding more light on the behavior of the discrete-time networked multi-virus SIS model, does not account for the possibility of the interconnection graph being time-varying - hence, it is rather limited. Therefore, in the present paper, the focus is on providing a rigorous theoretical analysis for *competing, time-varying, discrete-time networked SIS models*. In particular, we seek to identify parameter-based conditions for convergence to the disease-free equilibrium. In so doing, the present paper substantially differs from the works in (Sagar et al., 2018; Karrer and Newman, 2011), where the analysis is, as previously mentioned, based on simulations.

The SIS model assumes that the spread of a virus can only happen due to individual-to-individual contact. However, (infectious) diseases could also spread due to the presence of a shared infrastructure network, such as a transportation network or a water distribution network. A classic example that illustrates the aforesaid scenario is the following: In Östersund in Northern Sweden, around 27,000 people became ill and had a water-boil order for more than two months due to Cryptosporidium contamination of the drinking water (Widerström et al., 2014). Therefore, there is a need for SIS models that also account for the possibility of the spread happening via multiple mediums. To this end, a time-invariant susceptible-infected-water-susceptible (SIWS)[1] model has been proposed in (Liu et al., 2019*b*), which has been subsequently expanded to account for the presence of multiple shared resources (Paré et al., 2022); to account for (continuous-time) time-varying dynamics with a single shared resource (meaning that the infection at the infrastructure level is just a scalar) by Gracy et al. (2022); and finally to also factor in the presence of multiple viruses and an infrastructure network in (Cui et al., 2022). However, single-virus SIWS model is limited, since it cannot capture the following realistic scenario: Different lineages of avian influenza viruses, namely lineage A and lineage B, are known to be competitive (Bahl et al., 2009). The typical means of spread for avian influenza viruses are via close contact with infected birds, and bodily fluid droplets. However, it has recently been observed that water bodies can also act as effective pathways for the spread of avian influenza. In fact, the amount of droppings that an infected (with avian influenza) duck sheds into water over a 24-hour period can possibly infect more than 10^3 ducks; see (Ahrens et al., 2022). To the best of our knowledge, there does not exist a model that captures the aforedescribed scenario. The goal of the present paper is to devise a discrete-time counterpart to the model by Gracy et al. (2022) but also account for the simultaneous presence of multiple viruses and *mul-*

[1]The "W" in SIWS denotes an arbitrary infrastructure network contamination, not necessarily restricted to a water distribution network.

tiple shared resources, and, unlike (Cui et al., 2022), also admit the possibility of the interconnection graph at the population level is time-varying.

Paper Contributions
The paper makes the following contributions.

i) We devise a discrete-time model that allows for the spread of multiple viruses both over a possibly time-varying population network and over an infrastructure network.

ii) Assuming that the graph is time-invariant, we identify a sufficient condition for the exponential stability of the eradicated state of virus r; see Theorem 1.

iii) For the case where the graph is time-varying, we identify a sufficient condition for the exponential stability of the eradicated state of virus r both when the spread is homogeneous[2] and also when the spread is heterogeneous but under the assumption that the graph is slowly-varying; see Theorem 2 and Theorem 3, respectively.

iv) We show that, for heterogeneous spread, the sufficient condition for exponential stability of the eradicated state of virus r identified in Theorem 2 is robust to variations in the graph structure of the population network, provided that the variations are not too large; see Theorem 4.

v) Assuming that the graph is time-invariant, we provide a lower bound on the number of equilibria that our system possesses; see Proposition 4.

An auxiliary contribution of the present paper is as follows: The sufficient condition for exponential eradication of a virus identified in Theorem 2 involves a strict inequality - the relaxation of this inequality also assures eradication of said virus except that the eradication is, in this case, asymptotic; see Proposition 3. A preliminary version of this paper has appeared in the proceedings of the 2023 IFAC World Congress; see (Gracy et al., 2023). In comparison to (Gracy et al., 2023), the present paper differs in the following aspects:

i) Complete proofs of all results, except that of Theorem 1.

ii) Theorem 4, Proposition 2, and Proposition 3 have not appeared previously.

iii) Section 6, and in particular Proposition 4, was not included in (Gracy et al., 2023).

iv) By allowing for the graph topology to change, we present an extended and interpretable set of simulations.

[2]We say that the spread of virus r is homogeneous if the healing (resp. infection) rate with respect to virus r is the same for all nodes in the network; otherwise, we say that the spread is heterogeneous.

Paper Outline

The paper is structured as follows: We conclude the present section by gathering all the notations that would be used in the sequel. We present the model and formally state the questions that the present paper investigates in Section 2. Section 3 deals with the case when the graph is time-invariant, whereas Section 4 tackles the case where the graph is time-varying. Section 5 explores the robustness of the conditions for exponential eradication of a virus even when there are variations in the graph structure, whereas Section 6 partially addresses the endemic behavior when the graph structure remains fixed. The theoretical findings are illustrated by numerical examples in Section 7,. Finally, a summary of the paper, along with some of the open questions of possible interest, is provided in Section 8.

Notations: Let $\mathbb{R}$ (resp. $\mathbb{Z}_{\geq 0}$) denote the set of real numbers (resp. non-negative integers). We denote the set of positive integers by $\mathbb{Z}_{+}$. For any positive integer n, we have $[n] = \{1, \ldots, n\}$. Given a matrix $A \in \mathbb{R}^{n \times n}$, a_{ij} denotes the i^{th} row and j^{th} column entry; $\rho(A)$ denotes its spectral radius, and $\lambda_{\min}(A)$ (resp. $\lambda_{\max}(A)$) denotes the minimum (resp. maximum) eigenvalue of A (real). A diagonal matrix is denoted as $\text{diag}(\cdot)$. The transpose of vector $x \in \mathbb{R}^n$ is denoted as $x^\top$. Euclidean norms are denoted by $\|\cdot\|$. Given a matrix A, $A \prec 0$ (resp. $A \preccurlyeq 0$) indicates that A is negative definite (resp. negative semidefinite), whereas $A \succ 0$ (resp. $A \succcurlyeq 0$) indicates that A is positive definite (resp. positive semidefinite). The notation $\{A(k)\}_a^b$ denotes a sequence of matrices $A(k)$, where $k \in \{a, a+1, \ldots, b-1, b\}$.

2. Problem Formulation

In this section, inspired by (Cui et al., 2022), we detail a model of multi-virus spread across a population network and a network of shared resources. Subsequently, we formally state the problems being investigated. Finally, we introduce pertinent assumptions and definitions that would be required in the sequel.

2.1. Model

Consider m competing viruses spreading over a network of n individuals. Suppose that these m viruses simultaneously spread also over an infrastructure network of q resource nodes. Observe that if $m = 1$, then there is no notion of competition. Hence, in the rest of this paper, we will focus on the case where $m \geq 2$. The spread of the r^{th} virus, where $r \in [m]$, in individual i can be represented as follows.

$$\begin{aligned}\dot{x}_i^r(t) = & -\delta_i^r x_i^r(t) + \Big((1 - \textstyle\sum_{\ell=1}^m x_i^\ell(t)) \times \\ & \big(\textstyle\sum_{j=1}^n \beta_{ij}^r x_j^r(t) + \sum_{j=1}^q \beta_{ij}^{wr} w_j^r(t) \big) \Big), \end{aligned} \quad (1)$$

where $\beta_{ij}^r = \beta_i^r a_{ij}^r$. The term β_i^r (resp. δ_i^r) denotes the infection (resp. healing rate) of individual i with respect to virus r, while $a^r_{ij} \geq 0$ denotes the strength of interconnection between nodes i and j for the spread of virus r. The term β^{wr}_{ij} is the resource-to-individual infection rate for individual i from resource j for virus r. Note that $x^r_i(t)$ is an approximation of the probability of infection with respect to virus r of individual i at time instant t.

Viruses mutate over time, and the prevalence of an epidemic does not prevent the movement of people across cities, districts, etc. which might lead to the healing (resp. infection) rate changing over time. To account for such possibilities, we allow for the i) healing (resp. infection) rate to be time-varying; and ii) the set of neighbors that a node has to vary over time. Consequently, we need a more general form of (1), viz.

$$\begin{aligned}\dot{x}^r_i(t) =& -\delta^r_i(t)x^r_i(t) + \Big((1-\textstyle\sum_{\ell=1}^{m} x^\ell_i(t))\times \\ & \big(\textstyle\sum_{j=1}^{n} \beta_{ij}(t)^r x^r_j(t) + \sum_{j=1}^{q} \beta^{wr}_{ij}(t)w^r_j(t)\big)\Big), \quad (2)\end{aligned}$$

where $\beta^r_{ij}(t) = \beta^r_i(t)a^r_{ij}(t)$, and the concentration of the r^{th} virus in the j^{th} resource node at time t is described as:

$$\begin{aligned}\dot{w}^r_j(t) =& -\delta^{wr}_j w^r_j(t) + \textstyle\sum_{\ell=1}^{q} \alpha^r_{\ell j} w^k_\ell(t) - w^r_j(t)\sum_{\ell=1}^{q} \alpha^r_{j\ell} + \\ & \textstyle\sum_{\ell=1}^{n} c^{wr}_{j\ell}(t)x^r_\ell(t), \quad (3)\end{aligned}$$

where δ^{wr}_j denotes the healing rate of resource node j with respect to virus r; $\alpha^r_{j\ell}$ denotes the resource-to-resource infection rate for resource node ℓ from resource node j; and $c^{wr}_{j\ell}$ denotes the individual-to-resource infection rate for resource node j from individual ℓ.

A time-varying graph can be used to represent the spread of the m viruses over a possibly time-varying population network and an infrastructure network. More specifically, we define a m-layer graph $\mathcal{G}(k)$, where the vertices denote the individuals and the shared resource nodes, and layer r is the contact graph for the spread of virus r at time instant k, with $r \in [m]$. More precisely, there exists a directed edge from node j to node i in layer r, if individual j (resp. shared resource ℓ, with $\ell \in [q]$) can infect individual i (resp. shared resource ℓ) with virus r. For the sake of clarity of explanation, we define the following sets: $E^r(k) = \{(i,j) \mid i,j \in [n], a^r_{ji}(k) > 0\}$; $E^r_w = \{(\ell,j) \mid \ell,j \in [q], a^r_{\ell j} > 0\}$; $E^r_c = \{(j,\ell) \mid \ell \in [n], j \in [q], c^{wr}_{j\ell}(k) > 0\}$; and $E^r_b = \{(i,j) \mid i \in [n], j \in [q], \beta^{wr}_{ij}(k) > 0\}$. Finally, we define $\mathcal{E}^r(k) = E^r(k) \cup E^r_w \cup E^r_c(k) \cup E^r_b(k)$. Therefore, layer r of graph $\mathcal{G}$ at time k, denoted by $\mathcal{G}^r(k)$ is as follows: $\mathcal{G}^r(k) = (V, \mathcal{E}^r(k))$, with $|V| = n+q$.

By applying Euler's method (Atkinson, 2008) to (2) and (3), we obtain the following:

$$\begin{aligned}x^r_i(k+1) =& x^r_i(k) + h\big(-\delta^r_i(k)x^r_i(k) + (1-\textstyle\sum_{\ell=1}^{m} x^\ell_i(k))\times \\ & \big(\textstyle\sum_{j=1}^{n} \beta^r_{ij}(k)x^r_j(k) + \sum_{j=1}^{q} \beta^{wr}_{ij}(k)w^r_j(k)\big)\big) \quad (4)\\ w^r_j(k+1) =& w^r_j(k) + h\big(-\delta^{wr}_j w^r_j + \textstyle\sum_{\ell=1}^{q} \alpha^r_{\ell j} w^r_\ell \\ & - w^r_j \textstyle\sum_{\ell=1}^{q} \alpha^r_{j\ell} + \sum_{\ell=1}^{n} c^{wr}_{j\ell}(k)x^r_\ell\big), \quad (5)\end{aligned}$$

where h is the sampling parameter ($h > 0$). Define $x^r = [x^r_1 \ x^r_2 \ \dots \ x^r_n]$, $D^r(k) = \mathrm{diag}(\delta^r_i(k))_{i=1}^n$, $B^r(k) = [\beta^r_{ij}(k)]_{n\times n}$, $D^r_w = \mathrm{diag}(\delta^{wr}_j)_{j=1}^n$, $B^r_w = [\beta^{wr}_{ij}]_{n\times q}$, $C^r_w = [c^{wr}_{j\ell}]q \times n$, $A^r_w = [a^r_{\ell j}]_{q\times q}$, and $X^r = \mathrm{diag}(x^r)$. Therefore, in vector form, equations (4) and (5) can be written as follows:

$$\begin{aligned}x^r(k+1) =& x^r(k) + h\big(((I - \textstyle\sum_{\ell=1}^{m} X^\ell)B^r(k) - D^r(k))x^r(k)) \\ & + (I - \textstyle\sum_{\ell=1}^{m} X^\ell)B^r_w w^r(k)\big) \quad (6)\\ w^r(k+1) =& w^r(k) + h(-D^r_w w^r(k) + A^r_w w^r(k) + C^r_w(k)x^r(k)). \quad (7)\end{aligned}$$

Define the following.

$$\begin{aligned} z^r(k) &:= \begin{bmatrix} x^r(k) \\ w^r(k)\end{bmatrix}, \ X(z^r(k)) := \begin{bmatrix}\mathrm{diag}(x^r(k)) & 0 \\ 0 & 0\end{bmatrix}, \\ B^r_f(k) &:= \begin{bmatrix} B^r(k) & B^r_w(k) \\ C^r_w(k) & A^r_w - \mathrm{diag}(A^r_w)\end{bmatrix}, \text{ and} \quad (8) \\ D^r_f(k) &:= \begin{bmatrix} D^r(k) & 0 \\ 0 & D^r_w - \mathrm{diag}(A^r_w)\end{bmatrix}.\end{aligned}$$

Consequently, system (6)-(7) can be more compactly written as:

$$z^r(k+1) = z^r(k) + h\big(-D^r_f(k) + (I - \textstyle\sum_{\ell=1}^{m} X(z^\ell))B^r_f(k)\big)z^r(k), \quad (9)$$

with $r = 1, 2, \dots, m$.
It turns out system (9) has close connections to similar models devised in (Cui et al., 2022; Liu et al., 2019*b*; Paré et al., 2020a; Gracy et al., 2022). In order to explain those in a formal manner, we introduce the following remarks.

Remark 1 By setting $A_w = \mathbf{0}$, and $a_{ij}(k) = a_{ij}$ for all $k \in \mathbb{Z}_{\geq 0}$, (9) coincides with the model in (Cui et al., 2022). By setting $A_w = \mathbf{0}$, and $a_{ij}(k) = a_{ij}$ for all $k \in \mathbb{Z}_{\geq 0}$, $m = 1$, and $q = 1$, (9) is the discrete-time counterpart of the model in (Liu et al., 2019*b*).

Remark 2 Note that specialized to the case where $m = 1$, and $q = 1$, (9) is the discrete-time counterpart of the model in (Gracy et al., 2022), whereas specialized to the case where $m = 1$, and $a_{ij}(k) = a_{ij}$ for all $k \in \mathbb{Z}_{\geq 0}$, (9) is the discrete-time counterpart of the model in (Paré et al., 2022).

Remark 3 By setting $w^r(k) = \mathbf{0}$ for $r = 1, 2, \dots, q$, and $m = 1$, (9) collapses to the standard discrete-time time-varying networked SIS model studied in (Gracy et al., 2020).

The following remark provides additional intuition on the need for directed graphs while modeling disease spread.

Remark 4 It is quite natural to assume that the nature of disease transmission is undirected. Such an assumption is applicable provided that everyone is susceptible to the virus in the exact same fashion. That is, everyone gets affected by the virus to the same extent. Thus, the assumption of the graph being undirected is limited in the sense that it cannot account for situations where different individuals have different susceptibility levels to the same virus; for instance, some of the infected individuals might require hospitalization, while the others might be able to recover with only several days of rest. Hence, directed infection networks are important for more realistic modeling. Consequently, there is a whole body of literature that admits directed graphs in the modeling framework; please see for instance (Ye, Anderson and Liu, 2022; Anderson and Ye, 2023; Liu et al., 2019*a*; Gracy et al., 2020; Santos et al., 2015; Mei et al., 2017; Lin et al., 2021).

This paper deals with the stability analysis of the healthy state for the time-varying model in (9) and its time-invariant version. To this end, we need the following:

$$\begin{aligned} M_f^r(k) &:= I - hD_f^r(k) + hB_f^r(k) \qquad (10)\\ \hat{M}_f^r(k) &:= I - hD_f^r(k) + hB_f^r(k) - h\textstyle\sum_{\ell=1}^m X(z^\ell)B_f^r(k). \end{aligned}$$

Observe that the matrix $M_f^r(k)$ is the state matrix obtained by linearizing the dynamics of virus r around the eradicated state of virus r ($z^r(k) = 0$).

Define $\mathcal{D}^r := \{z^r(k) = [x^r(k)^\top, w^r(k)^\top]^\top \mid x^r(k) \in [0,1]^n, w^r(k) \in [0, w^r_{max}]^q\}$. Virus r is eradicated if $z^r(k) = 0$. The discrete-time multi-competitive layered networked SIWS model is in the disease-free equilibrium (DFE) if $z^r(k) = 0$, $\forall r \in [m]$.

2.2. Problem Statements

With respect to system (9), we aim to answer the following questions:

i) Suppose that, for some $r \in [m]$, $\mathcal{G}^r(k) = \mathcal{G}^r$ for all $k \in \mathbb{Z}_{\geq 0}$. Can we identify a sufficient condition such that, for any $z^r(0) \in \mathcal{D}^r$, $z^r(k)$ converges exponentially to its eradicated state, i.e., $z^r(k) = \mathbf{0}$?

ii) Suppose that for all $k \in \mathbb{Z}_{\geq 0}$
   i) $\beta_i^r(k)=\beta^r(k)$, and $\delta_i^r(k)=\delta^r(k)$ $\forall i \in [n]$;
   ii) $A^r(k)=A^r(k)^\top$ and $B_w^r(k)=C_w^r(k)^\top$.

   Can we identify a sufficient condition such that, for any $z^r(0) \in \mathcal{D}^r$, $z^r(k)$ converges exponentially to its eradicated state, i.e., $z^r(k) = \mathbf{0}$?

iii) Suppose that $A^r(k) \neq A^r(k)^\top$ for some $k \in \mathbb{Z}_{\geq 0}$, and that $\beta_i^r(k) \neq \beta_j^r(k)$ for some $i, j \in [n]$. Can we identify a sufficient condition such that, for any $z^r(0) \in \mathcal{D}^r$, $z^r(k)$ converges exponentially to its eradicated state, i.e., $z^r(k) = \mathbf{0}$?

iv) Suppose that, for some $r \in [m]$, for any $z^r(0) \in \mathcal{D}^r$, $z^r(k) = \mathbf{0}$ for all $k \geq k'$, for some $k' \in \mathbb{Z}_+$. Suppose that system (9) is perturbed via some suitably-defined additive perturbation. Can we identify a condition such that even for the perturbed system, for any $z^r(0) \in \mathcal{D}^r$, $z^r(k)$ converges exponentially to its eradicated state, i.e., $z^r(k) = \mathbf{0}$?

v) Suppose that, for some $r \in [m]$, $\mathcal{G}^r(k) = \mathcal{G}^r$ for all $k \in \mathbb{Z}_{\geq 0}$. What is a lower bound on the number of equilibria?

2.3. Standard stability notions and sufficient conditions

In this subsection, we will briefly recall some stability notions and results that are essential for understanding the findings in this paper. Consider a system, described as follows:

$$x(k+1) = f(k, x(k)), \qquad (11)$$

where $f : \mathbb{Z}_{\geq 0} \times \mathbb{R}^n \to \mathbb{R}^n$ is locally Lipschitz. Let $s(k, k_0, x_0)$ denote the solution of (11) corresponding to the initial condition $x(k_0) = x_0$. An equilibrium of (11) is said to be (uniformly) asymptotically stable if it is (uniformly) stable and (uniformly) attractive. Furthermore, an equilibrium of (11) is endowed with the property of GAS (resp. globally uniformly asymptotically stable (GUAS)) if, besides being asymptotically stable (resp. uniformly asymptotically stable), the system converges to that equilibrium for any initial state. We recall a sufficient condition for GUAS of an equilibrium of (11).

Lemma 1 (Vidyasagar, 2002, Section 5.9 Thm. 27) The DFE of system (11) is GUAS if there is a function $V : \mathbb{Z}_+ \times \mathbb{R}^n \to \mathbb{R}$ such that i) $V(k, 0) = 0$, and, for all $x \neq 0$, $V(k, x) > 0$, ii) V is decrescent, and radially unbounded, and iii) $-\Delta V$ (where the forward difference function $\Delta V : \mathbb{Z}_+ \times \mathbb{R}^n \to \mathbb{R}$ is defined as: $\Delta V(k, x) = V(k+1, x(k+1)) - V(k, x))$ is positive definite. ■

A stronger property is that of GES, which is defined as follows:

Definition 1 An equilibrium point of (11) is GES if there exist positive constants α and ω, with $0 \leq \omega < 1$, such that

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

We recall a sufficient condition for GES of an equilibrium of (11) in the following proposition:

Lemma 2 (Vidyasagar, 2002, Theorem 28, Section 5.9) Suppose there exists a function $V : \mathbb{Z}_+ \times \mathbb{R}^n \to \mathbb{R}$, and constants $a, b, c > 0$ and $p > 1$ such that $a\|x\|^p \leq V(k, x) \leq b\|x\|^p$, $\Delta V(k, x) := V(x(k+1)) - V(x(k)) \leq -c\|x\|^p$, $\forall k \in \mathbb{Z}_{\geq 0}$, and $\forall x \in \mathbb{R}^n$, then $x = 0$ is an exponentially stable equilibrium of (11). ■

3. Analysis of the Time-Invariant Case

In this section, we consider the case where the interconnection graph is time-invariant and identify conditions for exponential eradication of a virus.

3.1. Exponential eradication of a virus

Since the interconnection graph is time-invariant, i.e., $\mathcal{G}^r(k) = \mathcal{G}^r$ for all $k \in \mathbb{Z}_{\geq 0}$, the spread dynamics for virus r is as follows:

$$z^r(k+1) = z^r(k) + h\big(-D_f^r + (I - \textstyle\sum_{\ell=1}^m X(z^\ell))B_f^r\big)z^r(k). \qquad (12)$$

We make the following assumptions so that system (12) is well-defined.

Assumption 1 For all $i \in [n]$, $\sum_{\ell=1}^{m} x_i^\ell(0) \in [0,1]$.

Assumption 2 For all $i, j \in [n]$, $r \in [m]$ $\delta_i^r > 0$, $\beta_{ij}^r \geq 0$, $\beta_{ij}^{wr} \geq 0$. For all $r \in [m]$, $i \in [n]$, and $j \in [m]$, $\delta_j^{rw} > 0$ and $c_{ij}^{rw} \geq 0$ with at least one i such that $c_{ij}^{rw} > 0$.

Assumption 3 For all $r \in [m]$, $i \in [n]$ and $j \in [q]$, $w_j^r(0) \geq 0$ and $w_j^r(0) \leq w_{max}^r$, and $\sum_{\ell=1}^{n} c_{j\ell}^{wr}/\delta_j^{wr} \in [0, w_{max}^r]$.

Assumption 4 For all $i \in [n]$ (resp. $j \in [q]$), $r \in [m]$, $h\delta_i^r \in [0,1]$ (resp. $h\delta_j^r \in [0,1]$). Furthermore, $h \sum_{\ell=1}^{m} \left(\sum_{p=1}^{n} \beta_{ip}^\ell + \sum_{p=1}^{n} \beta_{ip}^{w\ell} w_{max}^\ell \right) \in [0,1]$.

The following lemma guarantees that the set $\mathcal{D}^r$ is positively invariant for system (12).

Lemma 3 (Cui et al., 2022, Lemma 1) Consider (12), and let Assumptions 1-4 hold. Then, $x_i^r(k) \in [0,1]$ for all $i \in [n]$, and $z_j^r(k) \in [0, w_{max}^r]$ for all $j \in [q]$, for all $k \in \mathbb{Z}_{\geq 0}$.

Recall that $x_i^r(k)$ is an approximation of the probability of infection for virus r of individual i, whereas $z_j^r(k)$ is the concentration of virus r in resource j; hence, if the states were to take values outside those in $\mathcal{D}^r$, then those states would not correspond to physical reality. Hence, for our subsequent stability results, we prove the system's eradicated state of virus r is stable with the domain of attraction $\mathcal{D}^r$, which is equivalent to global stability for this system. In particular, if the system's eradicated states are stable with the domain of attraction $\mathcal{D}^r$ for all $r \in [m]$, then the DFE is globally exponentially stable. Next, we provide a sufficient condition for the eradication of virus r.

Theorem 1 Let Assumptions 1-4 hold, and consider system (12). If $\rho(M_f^r) < 1$, with $r \in [m]$, then the eradicated state of virus r is exponentially stable, with domain of attraction $\mathcal{D}^r$.

Proof: See the proof of (Gracy et al., 2023, Theorem 1).
The following result is immediate.

Corollary 1 Consider system (12) under Assumptions 1-4. If $\rho(M_f^r) < 1$, for all $r \in [m]$, then the DFE is globally exponentially stable.

We now explain how Corollary 1 is related to a similar result in (Cui et al., 2022, Theorem 10). Corollary 1 provides guarantees for exponential convergence to the DFE, whereas (Cui et al., 2022, Theorem 10) guarantees only asymptotic convergence to the DFE. Thus, for the range of parameters that satisfy the conditions in Corollary 1 and in (Cui et al., 2022, Theorem 10), Corollary 1 provides stronger convergence guarantees (to the DFE). Moreover, Corollary 1, unlike (Cui et al., 2022, Theorem 10), does not require the graph to be strongly connected. On the other hand, (Cui et al., 2022, Theorem 10) allows for the spectral radius of M_f^r to be equal to one and yet achieves convergence, albeit asymptotic, to the DFE. Consequently, (Cui et al., 2022, Theorem 10) guarantees the eradication of viruses for a larger range of model parameters.

3.2. Reproduction number

The term $\rho(M_f^r)$ can be interpreted as the reproduction number for virus r. Define $M^r := I - hD + hB$; the term $\rho(M^r)$ denotes the reproduction number for virus r *assuming there is no infrastructure network*. It is natural to explore the relation between $\rho(M_f^r)$ and $\rho(M^r)$. To this end, we need the following assumption and proposition.

Assumption 5 The matrix B_f^r is irreducible for $r \in [m]$.

Proposition 1 (Cui et al., 2022, Proposition 4) Consider system (12) under Assumptions 2, 4, and 5. The reproduction number of the multi-virus SIS network with an infrastructure network is greater than the reproduction number of the multi-virus SIS network without the infrastructure network, i.e., $\rho(M_f^r) > \rho(M^r)$.

4. Exponential Eradication of a Virus: Time-Varying Case

This section studies the case where the population network is time-varying, i.e, we allow for $\mathcal{G}^r(k_0) \neq \mathcal{G}^r(k_1)$ for some $k_0 \neq k_1 \in \mathbb{Z}_{\geq 0}$. We rely on the model in (9). Before proceeding with the analysis, we need the following assumptions to ensure that (9) is well-defined.

Assumption 6 For all $k \in \mathbb{Z}_{\geq 0}$, $i, j \in [n]$, $r \in [m]$ $\delta_i^r(k) > 0$, $\beta_{ij}^r(k) \geq 0$, $\beta_{ij}^{wr}(k) \geq 0$. For all $r \in [m]$, $i \in [n]$, and $j \in [m]$, $\delta_j^{rw} > 0$ and $c_{ij}^{rw} \geq 0$ with at least one i such that $c_{ij}^{rw} > 0$.

Assumption 7 For all $k \in \mathbb{Z}_{\geq 0}$, $r \in [m]$, $i \in [n]$ and $j \in [q]$, $w_j^r(0) \geq 0$ and $w_j^r(0) \leq w_{max}^r$. Furthermore, $\sum_{\ell=1}^{n} c_{j\ell}^{wr}(k)/\delta_j^{wr}(k) \in [0, w_{max}^r]$.

Assumption 8 For all $i \in [n]$ (resp. $j \in [q]$), $k \in \mathbb{Z}_{\geq 0}$ and $r \in [m]$, $h\delta_i^r(k) \in [0,1)$ (resp. $h\delta_j^r(k) \in [0,1)$). Furthermore, $h \sum_{\ell=1}^{m} \left(\sum_{p=1}^{n} \beta_{ip}^\ell(k) + \sum_{p=1}^{n} \beta_{ip}^{w\ell}(k) w_{max}^\ell \right) \in [0,1]$.

Assumptions 6, 7, and 8 imply Assumptions 2, 3, and 4, respectively. The converse, however, is false. The following lemma establishes positive invariance of the set $\mathcal{D}^r$ for (9).

Lemma 4 (Cui et al., 2022, Lemma 4) Let Assumptions 1, 6-8 hold and consider (9). Then $x_i^r(k) \in [0,1]$, $\forall i \in [n]$, and $z_j^r(k) \in [0, w_{max}^r]$, $\forall j \in [q]$, $\forall k \in \mathbb{Z}_{\geq 0}$.

4.1. Homogeneous spread, symmetric undirected graphs

We focus on homogeneous virus spread (i.e., the infection rate for a virus is the same for every individual) in the layered network. The following theorem identifies a sufficient condition for the exponential eradication of a virus, irrespective of the initial infection levels in the network of individuals and in the network of shared resources, for the virus.

Theorem 2 Consider system (9) under Assumptions 1, 6-8. Suppose that, for all $k \in \mathbb{Z}_{\geq 0}$,

i) $\beta_i^r(k)=\beta^r(k)\ \forall i \in [n]$

ii) $\delta_i^r(k)=\delta^r(k)\ \forall i \in [n]$

iii) $A^r(k)=A^r(k)^\top$

iv) $B_w^r(k)=C_w^r(k)^\top$.

If $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) < 1$, where $r \in [m]$, then the eradicated state of virus r is exponentially stable with a domain of attraction $\mathcal{D}^r$.

Proof: Consider the Lyapunov function candidate $V(z^r, k) = \frac{1}{2} z^r(k)^\top z^r(k)$. It is immediate that $V(z^r, k) > 0$ for all k and $z^r(k) \neq 0$. Define $\Delta V(z^r, k) := V(z^r(k+1)) - V(z^r(k))$. Hence, for all $z^r \neq 0$, we have the following:

$$
\begin{aligned}
\Delta V(z^r) &= \frac{1}{2}\big(z^r(k+1)^\top z^r(k+1) - z^r(k)^\top z^r(k)\big)\\
&= \frac{1}{2}\big(z^\top (M_f^r(k) - \textstyle\sum_{\ell=1}^m Z^\ell B_f^\ell)^\top \qquad (13)\\
&\quad \times (M_f^r(k) - \textstyle\sum_{\ell=1}^m Z^\ell B_f^\ell) z^r - (z^r)^\top z^r\big)\\
&= \frac{1}{2}\big((z^r)^\top (M_f^r(k)^\top M_f^r - h M_f^r(k)^\top \textstyle\sum_{\ell=1}^m Z^\ell B_f^\ell\\
&\quad - h B_f^r(k)^\top \textstyle\sum_{\ell=1}^m Z^\ell M_f^r(k)\\
&\quad + h^2 B_f^r(k)^\top \textstyle\sum_{\ell=1}^m Z^\ell \sum_{\ell=1}^m Z^\ell B_f^r(k)) z^r - (z^r)^\top z^r\big). \qquad (14)
\end{aligned}
$$

Observe that

$$
\begin{aligned}
&(z^r)^\top \big(h^2 B_f^r(k)^\top \textstyle\sum_{\ell=1}^m Z^\ell \sum_{\ell=1}^m Z^\ell B_f^r(k)\\
&\quad - 2h M_f^r(k)^\top \textstyle\sum_{\ell=1}^m Z^\ell B_f^r(k)\big) z^r\\
&= (z^r)^\top \big(h^2 B_f^r(k)^\top \textstyle\sum_{\ell=1}^m Z^\ell \sum_{\ell=1}^m Z^\ell B_f^r(k)\\
&\quad - 2h^2 B_f^r(k)^\top \textstyle\sum_{\ell=1}^m Z^\ell B_f^r(k)\\
&\quad - 2h(I - hD_f^r(k)) \textstyle\sum_{\ell=1}^m Z^\ell B_f^r(k)\big) z^r\\
&< (z^r)^\top \big(h^2 B_f^r(k) \textstyle\sum_{\ell=1}^m Z^\ell \sum_{\ell=1}^m Z^\ell B_f^r(k)\\
&\quad - 2h^2 B_f^r(k)^\top \textstyle\sum_{\ell=1}^m Z^\ell B_f^r(k)\big) z^r \qquad (15)\\
&\leq (z^r)^\top \big(h^2 B_f^r(k) \textstyle\sum_{\ell=1}^m Z^\ell \sum_{\ell=1}^m Z^\ell B_f^r(k)\\
&\quad - h^2 B_f^r(k)^\top \textstyle\sum_{\ell=1}^m Z^\ell B_f^r(k)\big) z^r \qquad (16)\\
&= -(z^r)^\top \big(h^2 B_f^r(k)^\top \textstyle\sum_{\ell=1}^m Z^\ell (I - \sum_{\ell=1}^m Z^\ell) B_f^r(k)\big) z^r\\
&\leq 0, \qquad (17)
\end{aligned}
$$

where (15) follows by noting that i) due to Assumption 8 the matrix $(I - hD_f^r(k))$ is positive; and ii) due to Assumption 6, the matrix $B_f^r(k)$ is nonnegative; thus, implying that $-(z^r)^\top 2h(I - hD_f^r(k)) \sum_{\ell=1}^m Z^\ell B_f^r(k) z^r < 0$. Inequality (16) is a direct consequence of Lemma 4 and Assumption 6, whereas inequality (17) can be obtained by extending the claim in (Janson et al., 2020, Lemma 6) for m arbitrary, but finite, viruses, which is straightforward. Plugging (17) into (14) yields the following:

$$
\Delta V(z^r, k) \leq \frac{1}{2}(z^r)^\top \big(M_f^r(k)^\top M_f^r(k) - I\big) z^r. \qquad (18)
$$

It follows from the theorem assumptions that $M_f^r(k)$ is symmetric for all k, which implies that $M_f^r(k)^\top = M_f^r(k)$. Hence, it can be easily verified that $M_f^r(k)^\top M_f^r(k) = M_f^r(k) M_f^r(k)^\top$. Therefore, from (Horn and Johnson, 2012, page 114), we have that

$$
\begin{aligned}
\rho(M_f^r(k)^\top M_f^r(k)) &\leq \rho(M_f^r(k)^\top)\rho(M_f^r(k))\\
&= \rho(M_f^r(k))\rho(M_f^r(k))\\
&= \rho^2(M_f^r(k))\\
&< 1. \qquad (19)
\end{aligned}
$$

Inequality (19) is due to the following reason: By assumption, $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) < 1$, which, due to the definition of supremum, implies that $\rho(M_f^r(k)) < 1$ for all $k \in \mathbb{Z}_{\geq 0}$. Therefore, it is clear that $\rho^2(M_f^r(k)) < 1$.
Observe that $M_f^r(k)^\top M_f^r(k)$ is positive semi-definite, which implies that $\lambda_i(M_f^r(k)^\top M_f^r(k)) \geq 0$ for all $i \in [n+q]$. Therefore, $\lambda_{\max}(M_f^r(k)^\top M_f^r(k)) = \rho(M_f^r(k)^\top M_f^r(k))$. Consequently, from (19) we have that $\lambda_{\max}(M_f^r(k)^\top M_f^r(k)) < 1$. Applying Weyl's inequalities (Horn and Johnson, 2012, Corollary 4.3.15) to $M(k)^\top M(k) - I$, we obtain, for $i \in [n+q]$, $\lambda_i(M_f^r(k)^\top M_f^r(k) - I) \leq \lambda_i(M_f^r(k)^\top M_f^r(k)) - 1$. Since, for every $k \in \mathbb{Z}_{\geq 0}$, $\lambda_{\max}(M_f^r(k)^\top M_f^r(k)) < 1$, it follows that, for each $k \in \mathbb{Z}_{\geq 0}$, $\lambda_{\max}(M_f^r(k)^\top M_f^r(k) - I) < 0$. Plugging $\lambda_{\max}(M_f^r(k)^\top M_f^r(k) - I) < 0$ back into (18) and applying RRQ yields: $(z^r)^\top \big(M_f^r(k)^\top M_f^r(k) - I\big) z^r < 0$ for $z^r \neq 0$ and $k \in \mathbb{Z}_{\geq 0}$. Hence, it follows that, for $z^r \neq 0$ and $k \in \mathbb{Z}_{\geq 0}$, $\Delta V(z^r, k) < 0$. Exponential eradication of virus r with a domain of attraction $\mathcal{D}^r$, then, follows from (Vidyasagar, 2002, Theorem 28, Section 5.9). ☐

It turns out that there is an interesting ramification that Theorem 2 has on the possibility of eradication of virus r assuming there was no infrastructure network. We need the following assumption.

Assumption 9 The matrix $B_f^r(k)$ is irreducible for all $r \in [m]$ and $k \in \mathbb{Z}_{\geq 0}$.

We have the following result.

Proposition 2 Consider system (9) under Assumptions 6-9. It must be that $\sup\limits_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) > \sup\limits_{k\in\mathbb{Z}_{\geq 0}} \rho(M^r(k))$.

Proof: Consider the matrix $M_f^r(k)$ and notice that, due to Assumption 9, it is irreducible, whereas due to Assumptions 6 and 8 it is nonnegative. Furthermore, it can be expressed as follows:

$$
M_f^r(k) = \begin{bmatrix} M^r(k) & hB_w^r(k) \\ hC_w^r(k) & I - hD_w^r(k) + hC_w^r(k) \end{bmatrix}.
$$

Note that $M^r(k)$ is a principal square submatrix of $M_f^r(k)$. Therefore, for a fixed k, from (Varga, 2000, Lemma 2.6), it follows that $\rho(M_f^r) > \rho(M^r)$. Note that the choice of k is arbitrary. Hence, for every $k \in \mathbb{Z}_{\geq 0}$, it must be that $\rho(M_f^r(k)) > \rho(M^r(k))$. Hence, by employing the definition of supremum, we obtain $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) > \sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M^r(k))$. $\square$

Note that $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M^r(k)) < 1$ is a sufficient condition for exponential convergence to the DFE, assuming there is no infrastructure network; see (Paré et al., 2020a, Theorem 1). Hence, Proposition 2 implies that eradicating a virus in the population network does not necessarily imply eradication of said virus in the layered network; this further underscores the challenges of combating epidemics that spread through multiple mediums.

The following remark explains how Propositions 1 and 2 are related.

Remark 5 Note that Proposition 1 deals with time-invariant spread, whereas Proposition 2 pertains to time-varying spread. On the one hand, if for system (12) we set $\beta_i^r = \beta^r$ and $\delta_i^r = \delta^r$ for all $i \in [n]$, $A^r = (A^r)^\top$ and $B_w^r = C_w^r$, then Proposition 2 implies Proposition 1. On the other hand, the findings of Proposition 1 are also applicable if, for instance, $\beta_i^r \neq \beta_j^r$ for some $i, j \in [n]$, while those of Proposition 2 do not apply for such cases; thus, for a given time instant k, Proposition 1 subsumes Proposition 2. To summarize, neither Proposition 2 nor Proposition 1 subsume each other.

Observe that Theorem 2 insists on a strict inequality for achieving (exponential) convergence to the DFE. It is quite natural to ask whether (or not) convergence can be achieved even if the inequality in Theorem 2 was relaxed by, for instance, possibly letting at least some of the point-wise eigenvalues of $M(k)$ lie *on* the unit disk. We have the following proposition.

Proposition 3 Consider system (9) under Assumptions 1, 6-8. Suppose that, for all $k \in \mathbb{Z}_{\geq 0}$,

i) $\beta_i^r(k) = \beta^r(k)\ \forall i \in [n]$

ii) $\delta_i^r(k) = \delta^r(k)\ \forall i \in [n]$

iii) $A^r(k) = A^r(k)^\top$

iv) $B_w^r(k) = C_w^r(k)^\top$.

If $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) \leq 1$, where $r \in [m]$, then the eradicated state of virus r is asymptotically stable with a domain of attraction $\mathcal{D}^r$.

In words, Proposition 3 says that for a virus with homogeneous infection parameters spreading over undirected graphs, under Assumptions 1, 6-9, if none of the point-wise eigenvalues of $M_f^r(k)$ lie outside the unit disk, then the healthy state is GAS.

Proof: Suppose that $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) \leq 1$, then either a) $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) < 1$, or b) $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) = 1$. We will consider both cases separately.

Case a) From Theorem 2, we know that $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) < 1$ implies GES of the eradicated state of virus r. Since GAS is a weaker notion than GES, $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) < 1$ also implies GAS of the eradicated state of virus r.

Case b:) Consider the same Lyapunov function $V(z^r, k)$ as in the proof of Theorem 2. Therefore, for $z^r(k) \neq 0$ and for each $k \in \mathbb{Z}_{\geq 0}$, we obtain the following:

$$\begin{aligned}
\Delta V(z^r, k) &= ((z^r)^\top \hat{M}_f^r(k)^\top \hat{M}_f^r(k)x - (z^r)^\top z^r) \\
&= (z^r)^\top (M_f^r(k)^\top M_f^r(k) - I - 2hB_f^r(k)^\top X(z^r(k))M_f^r \\
&\quad + h^2 B_f^r(k)^\top X(z^r(k))X(z^r(k))B_f^r(k))z^r. \qquad (20)
\end{aligned}$$

By applying the RRQ Theorem Horn and Johnson (2012) to the matrix $M_f^r(k)^\top M_f^r(k) - I$, we obtain

$$(z^r)^\top (M_f^r(k)^\top M_f^r(k) - I)z^r \leq \lambda_{\max}(M_f^r(k)^\top M_f^r(k) - I)\left\|z^r\right\|^2. \qquad (21)$$

By appealing to the same arguments employed for showing that $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) < 1$ implies negative definiteness of $M_f^r(k)^\top M_f^r(k) - I$ in the proof of Theorem 2, we can show that $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) = 1$ implies, for each $k \in \mathbb{Z}_{\geq 0}$, $\lambda_i(M_f^r(k)^\top M_f^r(k) - I) \leq 0$ where $i = 1, 2, \ldots, n+q$. Hence, from (21), it follows that $(z^r)^\top (M_f^r(k)^\top M_f^r(k) - I)z^r \leq 0$ for $z^r \neq 0$ and for each $k \in \mathbb{Z}_{\geq 0}$. Therefore, from (20), it follows that

$$\begin{aligned}
&\Delta V(z^r, k) \\
&\leq (z^r)^\top (h^2 B_f^r(k)^\top X(z^r(k))X(z^r(k))B_f^r(k) \\
&\quad - hB_f^r(k)^\top X(z^r(k))M_f^r(k) \\
&\quad - hB_f^r(k)^\top X(z^r(k))(I - hD_f^r(k) + hB_f^r(k)))z^r \\
&= (z^r)^\top (h^2 B_f^r(k)^\top X(z^r(k))X(z^r(k))B_f^r(k) \\
&\quad - hB_f^r(k)^\top X(z^r(k))M_f^r(k) \\
&\quad - h^2 B_f^r(k)^\top X(z^r(k))B_f^r(k) \\
&\quad - hB_f^r(k)^\top X(z^r(k))(I - hD_f^r(k)))z^r \\
&< (z^r)^\top (h^2 B_f^r(k)^\top X(z^r(k))X(z^r(k))B_f^r(k) \\
&\quad - hB_f^r(k)^\top X(z^r(k))M_f^r(k) \\
&\quad h^2 B_f^r(k)^\top X(z^r(k))B_f^r(k))z^r \qquad (22) \\
&= (z^r)^\top (-h^2 B_f^r(k)^\top X(z^r(k))(I - X(z^r(k)))B_f^r(k) \\
&\quad - hB_f^r(k)^\top X(z^r(k))M_f^r(k))z^r \\
&\leq -(z^r)^\top hB_f^r(k)^\top X(z^r(k))M_f^r(k))z^r \\
&\leq 0,
\end{aligned}$$

where inequality (22) is due to Assumptions 1 and 8. It is immediate that if $z^r(k) = 0$ then $\Delta V(z^r, k) = 0$. Since the matrix $X(z^r)$ is a diagonal matrix where, for $i = 1, \ldots, n$ $X(z^r)_{ii} = x_i^r$, and, for $i = n+1, \ldots, n+q$, $X(z^r)_{ii} = 0$, and since by Lemma 4 we know that $x_i^r(k) \in [0, 1]$ for all k, it follows that the matrix $X(z^r)$ is non-negative. By Assumptions 6-8, the matrices $B_f^r(k)$ and $D_f^r(k)$ are, for all $k \in \mathbb{Z}_{\geq 0}$, non-negative and positive, respectively. Hence, if $\Delta V(z^r, k) = 0$, then $z^r(k) = 0$. Thus, from Lemma 1, the eradicated state of virus r is GAS. □

4.2. Directed networks and heterogeneous spread

We have the following result.

Theorem 3 Consider system (9) under Assumptions 1, 6-8. Assume $\exists\ \alpha_1 > 0, L \in \mathbb{R}_+, \kappa \in \mathbb{R}_+$, such that

i) $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) \leq \alpha_1 < 1$;

ii) $||M_f^r(k)|| \leq L$, $\forall k \in \mathbb{Z}_{\geq 0}$; and

iii) $\sup_{k\in\mathbb{Z}_{\geq 0}} ||M_f^r(k+1) - M_f^r(k)|| \leq \kappa$.

If κ is sufficiently small, then the eradicated state of virus r is exponentially stable, with a domain of attraction $\mathcal{D}^r$.

We provide an explicit expression for κ later in the proof. The proof of Theorem 3 closely mirrors that of (Paré et al., 2020a, Theorem 2); it can be traced back to the linear work in (Desoer, 1970; Rugh, 1996).

Proof: Consider the discrete-time Lyapunov equation:

$$(M_f^r)^\top(k)Q(k+1)M_f^r(k) - Q(k+1) = -I_{n+q}. \tag{23}$$

Observe that I_{n+q} is symmetric and positive definite. Moreover, by assumption $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) < 1$. Therefore, the solution to (23) (say, $Q(k+1)$) exists, is unique, and is positive definite for all $k \in \mathbb{Z}_{\geq 0}$; see (Rugh, 1996, Theorem 23.7). Furthermore, from the proof of (Rugh, 1996, Theorem 24.8), a closed-form expression for the solution is as follows:

$$Q(k+1) = I_{n+q} + \textstyle\sum_{j=1}^{\infty}[(M_f^r)^\top(k)]^j(M_f^r)^j(k). \tag{24}$$

Consider the Lyapunov function $V(k, z^r) = (z^r)^\top Q(k)z^r$. Given that, for each $k \in \mathbb{Z}_{\geq 0}$, $Q(k)$ is positive definite, it follows that $V(z^r, k) > 0$ for all $k \in \mathbb{Z}_{\geq 0}$ and $z^r \neq 0$. The rest of the proof can be broken down into three steps: First, we find a constant $\gamma_1 > 0$ such that $\gamma_1||z^r||^2 \leq V(k, z^r)$ for all $k \in \mathbb{Z}_{\geq 0}$. Second, we find a constant $\gamma_2 > 0$ such that $V(k, z^r) \leq \gamma_2||z^r||^2$ for all $k \in \mathbb{Z}_{\geq 0}$. Finally, we prove that $\Delta V(k, z^r) < 0$ for all $z^r \neq 0$ and $k \in \mathbb{Z}_{\geq 0}$.

Step 1: From (24) it is immediate that $Q(k) \geq I$ for all k. Therefore, $(z^r)^\top z^r \leq (z^r)^\top Q(k)z^r$, and hence we have for all $k \in \mathbb{Z}_{\geq 0}$: $||z^r||^2 \leq V(z^r, k)$

Step 2: Our objective here is to find an upper bound on $V(k, x)$, which is independent of k. To this end, define $\mu := \frac{1-\alpha_1}{2}$. Therefore, the assumption $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) \leq \alpha_1$ implies that $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) \leq 1 - 2\mu$. It can be easily verified that $1 - \mu > 0$. By using Dunford's integral (Dunford and Schwartz, 1958, page 568) with the circle of radius $1 - \mu$ as contour, we have the following:

$$\begin{aligned} M_f^r(k)^P &= \frac{1}{2\pi j}\oint_C s^P(sI_{n+q} - M_f^r(k))^{-1}ds \\ &\leq \frac{1}{2\pi j}2\pi|s|\max_{|s|=1-\mu}\{s^F(sI_{n+q} - M_f^r(k))^{-1}\}. \end{aligned} \tag{25}$$

By taking the norm of both sides of (25), and evaluating at $|s| = 1 - \mu$ one obtains:

$$\begin{aligned} &||M_f^r(k)^P|| \leq (1-\mu)\max_{|s|=1-\mu}(|s|^P)||\max_{|s|=1-\mu}(sI_{n+q} - M_f^r(k))^{-1}|| \\ &||M_f^r(k)^P|| \leq (1-\mu)^{P+1}||\max_{|s|=1-\mu}(sI_{n+q} - M_f^r(k))^{-1}|| \\ &\leq (1-\mu)^{P+1}\max_{|s|=1-\mu}||(sI_{n+q} - M_f^r(k))^{-1}|| \\ &\leq (1-\mu)^{P+1}\max_{|s|=1-\mu}\left\{\frac{||(sI_{n+q} - M_f^r(k))||^{n+q-1}}{|\det(sI_{n+q} - M_f^r(k))|}\right\}, \end{aligned} \tag{26}$$

where (26) is due to (Kato, 1960, Lemma 1).

From (Horn and Johnson, 2012, pg.55) it is clear that, given a $s \in \mathbb{C}$, $\det(sI_{n+q} - M_f^r(k)) = (s - \lambda_j(M_f^r(k)))^{n+q}$. Notice that

$$\begin{aligned} |s - \lambda_j(M_f^r(k))| &\geq ||s| - |\lambda_j(M_f^r(k))|| && (27) \\ &\geq ||s| - (1-2\mu)| && (28) \\ &= \mu, && (29) \end{aligned}$$

where (27) follows from the reverse triangle inequality. We obtain inequality (28) in view of the following: Recall that $\sup_{k\in\mathbb{Z}_{\geq 0}} \rho(M_f^r(k)) \leq 1 - 2\mu$. By employing the definition of supremum, it must be that, for every k, each pointwise eigenvalue of $M_f^r(k) \leq 1 - 2\mu$, i.e., $|\lambda_i(M_f^r(k))| \leq 1 - 2\mu$, where $i = 1, 2, \ldots n + q$. Equality (29) is obtained by evaluating (28) at $|s| = 1 - 2\mu$, and, as a result, for $|s| = 1 - \mu$, $|\det(sI_{n+q} - M_f^r(k))| \geq \mu^{n+q}$.

By assumption there also exists an L such that $||M_f^r(k)|| \leq L$, for all $k \in \mathbb{Z}_{\geq 0}$. Consequently, $||(sI_{n+q} - M_f^r(k))|| \leq (1 - \mu + L)$. Therefore, given that $|\det(sI_{n+q} - M_f^r(k))| \geq \mu^{n+q}$, we can rewrite (26) as follows:

$$||M_f^r(k)^P|| \leq \frac{(1-\mu)^{P+1}}{\mu^{n+q}}(1 - \mu + L)^{n+q-1}. \tag{30}$$

Define $m_1 := \frac{1-\mu}{\mu^{n+q}}(1 - \mu + L)^{n+q-1}$ and $p_1 := (1 - \mu)$. Therefore, (30) can be rewritten as:

$$||M_f^r(k)^P|| \leq m_1 p_1^P \quad \forall P, \forall k \in \mathbb{Z}_{\geq 0}. \tag{31}$$

Observe that taking norms on both sides of (24), and taking recourse to the triangle inequality and the submultiplicativity of matrix norms, we obtain:

$$||Q(k+1)|| \leq 1 + \textstyle\sum_{j=1}^{\infty} m_1^2 p_1^{2j} \leq \frac{m_1^2}{1-p_1^2}, \tag{32}$$

Note that $p_1 < 1$, then $p_1^2 < 1$, which implies (32). Since $Q(k)$ is symmetric $\forall k \in \mathbb{Z}_{\geq 0}$, by applying RRQ we have:

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

which implies

$$\begin{aligned} &\lambda_{\min}(Q(k))||z^r(k)||^2 \leq z^r(k)^\top Q(k)z^r(k) \\ &\leq \lambda_{\max}(Q(k))||z^r(k)||^2 \leq ||Q(k)|| \cdot ||z^r(k)||^2 && (33) \\ &\leq \frac{m^2}{1-p^2}||z^r(k)||^2, && (34) \end{aligned}$$

where (33) follows from (Horn and Johnson, 2012, Theorem 5.6.9), and (34) is due to (32). Then, $\forall k \in \mathbb{Z}_{\geq 0}$,

$$V(k, z^r) \leq \frac{m_1^2}{1-p_1^2}||z^r||^2. \tag{35}$$

Step 3: Define $\Delta V(k, z^r) := V(z^r(k+1)) - V(z^r(k))$. Hence, for $z^r \neq 0$, and $\forall k \in \mathbb{Z}_{\geq 0}$, we obtain the following:

$$\begin{aligned}\Delta V(k, z^r) &= (z^r)^\top (M_f^r(k)^\top Q(k+1) M_f^r(k) - Q(k)) z^r \\ &- 2h(z^r)^\top M_f^r(k)^\top Q(k+1) \textstyle\sum_{\ell=1}^m X(z^\ell) B_f^r z^r \\ &+ h^2 (z^r)^\top B_f^r(k)^\top \textstyle\sum_{\ell=1}^m X(z^\ell) Q(k+1) \sum_{\ell=1}^m X(z^\ell) B_f^r z^r.\end{aligned} \tag{36}$$

The matrix $M_f^r(k)^\top Q(k+1) M_f^r(k) - Q(k)$ is negative definite. Subtracting two successive instances of (23) results in

$$M_f^r(k)^\top Q(k+1) M_f^r(k) - M_f^r(k-1)^\top Q(k) M_f^r(k-1) = Q(k+1) - Q(k). \tag{37}$$

Adding and subtracting $M_f^r(k)^\top Q(k) M_f^r(k)$ to the LHS of (37), and rearranging of terms, leads to

$$\begin{aligned}&M_f^r(k)^\top (Q(k+1) - Q(k)) M_f^r(k) - (Q(k+1) - Q(k)) = \\ &M_f^r(k-1)^\top Q(k) M_f^r(k-1) - M_f^r(k)^\top Q(k) M_f^r(k).\end{aligned} \tag{38}$$

In a similar vein, by adding and subtracting $M_f^r(k-1)^\top Q(k) M_f^r(k)$ to the RHS of (38), we obtain

$$\begin{aligned}&M_f^r(k)^\top (Q(k+1) - Q(k)) M_f^r(k) - (Q(k+1) - Q(k)) \\ &= -((M_f^r(k)^\top - M_f^r(k-1)^\top) Q(k) M_f^r(k) \\ &\quad + M_f^r(k-1)^\top Q(k) (M_f^r(k) - M_f^r(k-1))).\end{aligned} \tag{39}$$

Define $R_1 := ((M_f^r(k))^\top - (M_f^r(k-1))^\top) Q(k) M_f^r(k) + (M_f^r(k-1))^\top Q(k) (M_f^r(k) - M_f^r(k-1))$. As a consequence, we have the following:

$$\begin{aligned}&||R_1|| \leq ||(M_f^r(k)^\top - M_f^r(k-1)^\top)) Q(k) M_f^r(k)|| + \\ &||M^\top(k-1) Q(k) (M(k) - M(k-1))|| \qquad (40)\\ &\leq ||(M_f^r(k)^\top - M_f^r(k-1)^\top)|| \cdot ||Q(k)|| \cdot ||M_f^r(k)|| \\ &+ ||M_f^r(k-1)^\top|| \cdot ||Q(k)|| \cdot ||M_f^r(k)^\top - M_f^r(k-1)^\top)||. \qquad (41)\end{aligned}$$

Note that inequality (40) comes from the triangle inequality of matrix norms, while inequality (41) follows from the submultiplicativity of matrix norms.

Since, for all $k \in \mathbb{Z}_{\geq 0}$, i) by assumption, there exists κ such that $||M_f^r(k+1) - M_f^r(k)|| \leq \kappa$, and ii) by (32), $||Q(k)|| \leq \frac{m_1^2}{1-p_1^2}$, it is clear from (41) that $||R_1|| \leq 2\kappa \frac{m_1^2}{1-p_1^2} L$. Notice that (39) is a discrete-time Lyapunov equation; the solution for which is given by

$$Q(k+1) - Q(k) = R_1 + \textstyle\sum_{j=1}^\infty [M_f^r(k)^\top]^j R_1 [M_f^r(k)]^j. \tag{42}$$

Taking the norm of both sides of (42) leads to

$$\begin{aligned}||Q(k+1) - Q(k)|| &\leq ||R_1||(1 + \textstyle\sum_{j=1}^\infty m_1^2 p_1^{2j}) \qquad (43)\\ &\leq 2\kappa \frac{m_1^4}{(1-p_1^2)^2} L. \qquad (44)\end{aligned}$$

where inequality (44) is a consequence of (43) being a convergent series. Next, pick $\sigma > 0$ such that $1 - \sigma < 1$. Hence, from inequality (44) it is clear that if $\kappa \leq \frac{(1-p_1^2)^2}{2m_1^4 L}(1-\sigma)$, then $||Q(k+1) - Q(k)|| \leq 1 - \sigma$. It turns out that $||Q(k+1) - Q(k)|| \leq 1 - \sigma$ implies, for $z^r \neq 0$ and $k \in \mathbb{Z}_{\geq 0}$,

$$z^r(k)^\top M_f^r(k)^\top Q(k+1) M_f^r(k) - Q(k) z^r(k) < 0. \tag{45}$$

Indeed, note that (23) can be rewritten as: $M_f^r(k)^\top Q(k+1) M_f^r(k) - Q(k) = -I_{n+q} + Q(k+1) - Q(k)$, for all $k \in \mathbb{Z}_{\geq 0}$. Therefore, for all $k \in \mathbb{Z}_{\geq 0}$, (45), can be written as:

$$\begin{aligned}&z^r(k)^\top (-I_{n+q} + Q(k+1) - Q(k)) z^r(k) \\ &\leq -||z^r(k)||^2 + z^r(k)^\top (Q(k+1) - Q(k)) z^r(k) \\ &\leq -||z^r(k)||^2 + \lambda_{\max}(Q(k+1) - Q(k)) ||z^r(k)||^2 \qquad (46)\\ &\leq -||z^r(k)||^2 + (1-\sigma)||z^r(k)||^2 \qquad (47)\\ &= -\sigma ||z^r(k)||^2 < 0, \qquad (48)\end{aligned}$$

where (46) follows from the definition of the induced norm of $(Q(k+1) - Q(k))^{\frac{1}{2}}$, (47) is due to the following reasons: a) the norm of a matrix is lower bounded by its spectral radius (Horn and Johnson, 2012, Theorem 5.6.9), and b) $||Q(k+1) - Q(k)|| \leq 1 - \sigma$, and finally (48) follows from the assumption that $\sigma > 0$.
Therefore, by plugging (45) in (36), it is immediate that

$$\begin{aligned}&\Delta V(k, z^r) < -2h(z^r)^\top M_f^r(k)^\top Q(k+1) \textstyle\sum_{\ell=1}^m X(z^\ell) B_f^r z^r \\ &\quad + h^2 (z^r)^\top B_f^r(k)^\top \textstyle\sum_{\ell=1}^m X(z^\ell) Q(k+1) \sum_{\ell=1}^m X(z^\ell) B_f^r z^r \\ &= (z^r)^\top \big(h^2 B_f^r(k)^\top \textstyle\sum_{\ell=1}^m Z^\ell Q(k+1) \sum_{\ell=1}^m Z^\ell B_f^r(k) \\ &\quad - 2h^2 B_f^r(k)^\top Q(k+1) \textstyle\sum_{\ell=1}^m Z^\ell B_f^r(k) \\ &\quad - 2h(I - hD_f^r(k)) Q(k+1) \textstyle\sum_{\ell=1}^m Z^\ell B_f^r(k)\big) z^r \\ &\leq (z^r)^\top \big(h^2 B_f^r(k) \textstyle\sum_{\ell=1}^m Z^\ell Q(k+1) \sum_{\ell=1}^m Z^\ell B_f^r(k) \\ &\quad - 2h^2 B_f^r(k)^\top Q(k+1) \textstyle\sum_{\ell=1}^m Z^\ell B_f^r(k)\big) z^r \qquad (49)\\ &\leq (z^r)^\top \big(h^2 B_f^r(k) \textstyle\sum_{\ell=1}^m Z^\ell Q(k+1) \sum_{\ell=1}^m Z^\ell B_f^r(k) \\ &\quad - h^2 B_f^r(k)^\top Q(k+1) \textstyle\sum_{\ell=1}^m Z^\ell B_f^r(k)\big) z^r \qquad (50)\\ &= -(z^r)^\top \big(h^2 B_f^r(k)^\top (I - \textstyle\sum_{\ell=1}^m Z^\ell) Q(k+1) \sum_{\ell=1}^m Z^\ell B_f^r(k)\big) z^r \\ &\leq 0, \qquad (51)\end{aligned}$$

where inequality (49), (50), and (51) are obtained using the same line of reasoning as in inequality (15), (16), and (17), respectively. Exponential eradication of virus r with a domain of attraction $\mathcal{D}^r$ is a direct consequence of (Vidyasagar, 2002, Theorem 28, Section 5.9). □

Note that the results in Sections 3 and 4 are reliant on exact knowledge of the system parameters. However, often times the system parameters could change over time due to a multitude of factors; in context, this would mean that the healing (resp. infection) rate of individuals could differ from the known values. This is referred to as *perturbation* of system parameters. It is of interest to know that, assuming a system with known virus dynamics is stable, how well the stability guarantees (obtained so far) translate to settings where there is some perturbation in the virus dynamics. The next section deals with the same.

5. Perturbation Analysis

In this section, we allow for some perturbations in the graph structure, i.e., the entries of $A^r(k)$, and, supposing that the unperturbed system is GES, we are interested in understanding when the same holds for the perturbed system. To this end, define $\bar{B}^r(k) := \text{diag}(\beta_i^r(k))$ with $i = 1, 2, \ldots, n$. Together with recalling that $A^r(k) = [a_{ij}^r(k)]$, it is immediate that $B^r(k) = \bar{B}^r(k)A^r(k)$. We have the following result.

Theorem 4 Consider system (9) under Assumptions 1, 6-8. Define the perturbed system as:

$$y^r(k+1) = (\hat{M}_f^r(k) + F^r(k))y^r(k), \tag{52}$$

where $F^r(k) = h(I - X(z^r(k)))B^r(k)\bar{\Delta}^r(k)$, $\bar{\Delta}^r(k) = [\begin{smallmatrix} \Delta^r(k) & \mathbf{0} \\ \mathbf{0} & \mathbf{0} \end{smallmatrix}]$, with $\Delta^r(k)$ denoting the perturbation in $A^r(k)$, for all $k \in \mathbb{Z}_{\geq 0}$. Suppose that there exists $\zeta \in \mathbb{R}_+$ such that, for all $k \in \mathbb{Z}_{\geq 0}$, $||F^r(k)|| \leq \zeta$. If, for system (9), the eradicated state of virus r is GES, and ζ is sufficiently small, then for system (52) the eradicated state of virus r is GES. ■

The proof is based on the techniques used for obtaining a similar result for the linear case; see (Rugh, 1996, Theorem 24.7).

Proof: Observe that by setting $F^r(k) = 0$ in system (52), we obtain system (9). By assumption, system (9) is GES. Therefore, by (Vidyasagar, 2002, Definition 15, Page 266), there exist constants η, $\alpha > 0$ and $\sigma < 1$ such that the following is satisfied:

$$\begin{aligned} ||y_0^r|| \leq \eta, k_0 \geq 0 \implies & ||s(k_0 + k, k_0, y_0^r)|| \\ & \leq \alpha ||y_0^r|| \sigma^k, \forall k \geq k_0 + 1, \end{aligned} \tag{53}$$

where $y^r(k_0) = y_0^r$. By viewing the term $F^r(k)y^r(k)$ as an input term, the complete solution to (52) is as follows:

$$\begin{aligned} y^r(k) = & s(k_0 + k, k_0, y_0^r) \\ & + \textstyle\sum_{j=k_0}^{k-1} s(k, j+1)F^r(j)y^r(j), \forall k \geq k_0 + 1. \end{aligned} \tag{54}$$

Taking norms on both sides of (54) yields:

$$||y^r(k)|| \leq ||s(k_0 + k, k_0, y_0^r)|| + ||\textstyle\sum_{j=k_0}^{k-1} s(k, j+1)F^r(j)y^r(j)|| \tag{55}$$

$$\leq \alpha \cdot \sigma^{(k-k_0)}||y_0^r|| + \textstyle\sum_{j=k_0}^{k-1} ||s(k, j+1)|| \cdot ||F^r(j)|| \cdot ||y^r(j)||, \tag{56}$$

where inequality (55) is due to the triangle inequality of matrix norms, whereas inequality (56) is a consequence of (53) and the submultiplicativity of matrix norms. Therefore, by Bellman-Gronwall inequality for sequences (Rugh, 1996, Lemma 24.5), we obtain for $k \geq k_0 + 1$:

$$\begin{aligned} \sigma^{-k}||y^r(k)|| & \leq \alpha\sigma^{-k_0}||y_0^r|| \prod_{j=k_0}^{k-1} [1 + \frac{\alpha}{\rho}||F^r(j)||] \\ & \leq \alpha\sigma^{-k_0}||y_0^r|| \prod_{j=k_0}^{k-1} [1 + \frac{\alpha}{\rho}\zeta], \end{aligned} \tag{57}$$

where inequality (57) is a consequence of the assumption that there exists $\zeta \in \mathbb{R}_+$ such that, for all $k \in \mathbb{Z}_{\geq 0}$, $||F^r(k)|| \leq \zeta$. Since $\prod_{j=k_0}^{k-1}[1 + \frac{\alpha}{\sigma}\zeta] \leq [1 + \frac{\alpha}{\sigma}\zeta]^{(k-k_0)}$, we can rewrite (57) as

$$||y^r(k)|| \leq \alpha(\sigma + \alpha\zeta)^{k-k_0}||y_0^r||. \tag{58}$$

Note that choosing $\zeta < \frac{1-\sigma}{\alpha}$ yields us a positive constant ω as in Definition 1. Furthermore, since $\alpha > 0$, it follows, from Definition 1, that for system (52) the eradicated state of virus r is GES. □

6. Endemic Behavior of the Model

The analysis so far has focused on identifying conditions guaranteeing convergence to the eradicated state of a virus. In this section, we will explore the endemic behavior of our model. Specifically, for the time-invariant case, we establish a lower bound on the number of equilibria that our system possesses. The analysis for the time-varying case is more complicated; we shed more light on the technical difficulties involved but provide no analytical results.

6.1. Time-invariant case

Assuming $m = 1$, if $\rho(M_f) > 1$, then there exists an endemic equilibrium, $\bar{z}$, where $\bar{z} \gg \mathbf{0}$. Furthermore, for all initial conditions in $\mathcal{D} \setminus \{\mathbf{0}\}$, the dynamics of system (12) asymptotically converges to $\bar{z}$; see (Cui et al., 2022, Theorem 3). For the multi-competitive case (i.e., $m \geq 1$), we have the following result.

Proposition 4 Consider system (12) under Assumptions 1-5. If $\rho(M_f^r) > 1$, for all $r \in [m]$, then system (12) has at least $m+1$ equilibria, viz. $\mathbf{0}$, $(\bar{z}^1, \mathbf{0} \ldots, \mathbf{0}) \ldots, (\mathbf{0} \ldots, \mathbf{0}, \bar{z}^m)$, where for each $r \in [m]$ $\bar{z}^r \gg \mathbf{0}$.

Proof: From (Cui et al., 2022, Theorem 3) we know that for each $r \in [m]$ such that $\rho(M_f^r) > 1$, there exists an endemic equilibrium, $(\mathbf{0} \ldots, \mathbf{0}, \bar{z}^r, \mathbf{0}, \ldots, \mathbf{0})$, where $\bar{z}^r \gg \mathbf{0}$. Hence, since, by assumption, $\rho(M_f^r) > 1$, for all $r \in [m]$, it follows that there exists m such endemic equilibria. Coupled with the observation that $\mathbf{0}$ is always an equilibrium of system (12), it is clear that said system has at least

$m+1$ equilibria. □

Note that Proposition 4, since it also accounts for the influence of infrastructure network, is a more general version of (Paré et al., 2020c, Proposition 2). Proposition 4 provides a lower bound on the number of equilibria that system (12) possesses. It, however, makes no comment regarding the stability (or lack thereof) of the various endemic equilibria. It does imply that the healthy state is an unstable equilibrium; see (Cui et al., 2022, Theorem 3). An exact characterization of the endemic equilibria, $(\mathbf{0}\ldots,\mathbf{0},\bar{z}^r,\mathbf{0},\ldots,\mathbf{0})$, where $\bar{z}^r \gg \mathbf{0}$, remains, to the best of our knowledge, unavailable. For the special case, where $w(k)=\mathbf{0}$, (in which case we have a multi-competitive discrete-time networked SIS model), by leveraging the fact that a) both a continuous-time dynamic system and its discrete-time counterpart share the same equilibria ((Cui et al., 2022)), and b) the equilibrium $(\mathbf{0}\ldots,\mathbf{0},\bar{z}^r,\mathbf{0},\ldots,\mathbf{0})$, where $\bar{z}^r \gg \mathbf{0}$, is the endemic equilibrium corresponding to the single-virus system obtained by setting $z^\ell=\mathbf{0}$ for all $\ell \in [m]\setminus r$, an exact characterization of the equilibrium point $(\mathbf{0}\ldots,\mathbf{0},\bar{z}^r,\mathbf{0},\ldots,\mathbf{0})$ has been provided in (Mei et al., 2017, Theorem 4.3, statement (iii)(b)).

Suppose that $m=2$. Then, another kind of equilibrium that could possibly exist is of the form $(\hat{z}^1,\hat{z}^2)$ such that $\hat{z}^k \gg \mathbf{0}$ for $k=1,2$ and $\hat{z}^1+\hat{z}^2 \ll \mathbf{1}$. A sufficient (resp. necessary) condition for the existence of such an equilibrium (hereafter referred to as coexistence equilibrium) has been provided in (Cui et al., 2022, Theorem 12) (resp. (Cui et al., 2022, Theorem 13)). Note that (Cui et al., 2022, Theorem 12) guarantees existence, but makes no comments on the stability of the coexisting equilibrium. Furthermore, it assumes that the equilibria, $(\bar{z}^1,\mathbf{0})$ and $(\mathbf{0},\bar{z}^2)$ are unstable. It is unknown whether (or not) any coexistence equilibrium could exist for other stability configurations of the boundary equilibria. We will explore this further in Section 7.

It turns out that it is impossible to have multiple coexistence equilibria of system (12) that differ in one, and only one, coordinate. We have the following result.

Lemma 5 Consider system (12) under Assumptions 1-5, and suppose that $m=2$. Suppose that $(\hat{z}^1,\hat{z}^2)\in\mathcal{D}$ and $(\tilde{z}^1,\tilde{z}^2)\in\mathcal{D}$ be coexistence equilibria of system (12). If $\hat{z}^1=\tilde{z}^1$, then $\hat{z}^2=\tilde{z}^2$.

Proof: By assumption, $(\hat{z}^1,\hat{z}^2)\in\mathcal{D}$ and $(\tilde{z}^1,\tilde{z}^2)\in\mathcal{D}$ are coexistence equilibria. Therefore, by employing the definition of the fixed point of a discrete map to equation (12) for $r=1$ yields the following:

$$\mathbf{0} = -D_f^1\hat{z}^1 + ((I-\hat{Z}^1-\hat{Z}^2))B_f^1\hat{z}^1 \tag{59}$$

$$\mathbf{0} = -D_f^1\tilde{z}^1 + ((I-\tilde{Z}^1-\tilde{Z}^2))B_f^1\tilde{z}^1. \tag{60}$$

From (59) and (60), and since, by assumption, $\hat{z}^1=\tilde{z}^1$

$$\begin{aligned}\hat{z}^1 &= (D_f^1)^{-1}((I-\hat{Z}^1-\hat{Z}^2)))B_f^1\hat{z}^1\\ &= (D_f^1)^{-1}((I-\hat{Z}^1-\tilde{Z}^2))B_f^1\hat{z}^1,\end{aligned}$$

which, since i) $\hat{z}^1 \gg \mathbf{0}$ and ii) by Assumption 5 it follows that for each $i\in[n+q]$, $\sum_{j=1}^n [B_f^1]_{ij} > 0$, which implies that $\hat{Z}^2=\tilde{Z}^2$, i.e., $\hat{z}^2=\tilde{z}^2$. □

6.2. Time-varying case

Endemic behavior for time-varying epidemics is quite a challenging problem - even more so when $m\geq 1$. Assuming $m=1$ and, for some $p\in\mathbb{Z}_+$, $B(k)=B(k+p)$, $D(k)=D(k+p)$, setting $w_i(k)=0$ for all $k\in\mathbb{Z}_{\geq 0}$, it has been shown that violation of a certain eigenvalue condition results in the healthy state being unstable; see (Gracy et al., 2020, Proposition 5). In fact, simulations indicate that the aforementioned setting gives rise to the existence of a limit cycle that contains p states. A sufficient condition for the existence of limit cycles for switched continuous-time SIS epidemics has been provided in (Mason et al., 2014, Theorem 6.2), whereas for the case of discrete-time time-varying SIS epidemics existence (or lack thereof) of limit cycles remain open. One approach towards solving this problem may possibly rely on the celebrated Poincaré-Hopf theorem for discrete-time nonlinear systems; see (Ye, Liu, Anderson and Cao, 2022, Theorem 6); the difficulty with this approach lies primarily in identifying a compact and contractible manifold that fulfills the conditions in (Ye, Liu, Anderson and Cao, 2022, Theorem 6). Due to the analytical difficulty of this problem, we explore it via simulations in Section 7.

7. Simulations

We consider 2 competitive viruses spreading over 10 population nodes with an infrastructure network of 5 resources, i.e., $n=10,\ q=5,\ m=2$. For all the simulations we assume that all the resource nodes are connected to all the population nodes. We set the sampling period $h=0.001$. For all simulations, we plot the infection level of each node in the population and resource network for both viruses. The blue (resp. red) and green (resp. black) lines represent the infection level with respect to virus 1 (resp. virus 2) in the population and in the resource, respectively. We split the simulations into two subsections: 1) the illustrative simulations where confirm the analytic results in the paper, and 2) exploratory simulations where we show unproven behavior of the model.

7.1. Illustrative Simulations

Time Invariant Case: First, we assume that $a_{ij}^r=1$, for all $i,j\in[10]$, $r\in[2]$, and $\alpha_{ij}=1$ for all $i,j\in[5]$. The healing and infection rates for the population and infrastructure network are as follows: $\beta_i^1=0.07,\delta_i^1=3,\delta_j^{w1}=2$ for virus 1, and $\beta_i^2=0.3,\delta_i^2=2,\delta_j^{w2}=1$ for virus 2, for all $i\in[10], j\in[5]$. We assume that all the resource nodes are connected to all the population nodes. The individual-to-resource infection rate for both viruses is c_{jl}^{wr}=1 for all $j\in[5],\ l\in[10]$. The resource-to-individual infection rate for each virus are $\beta_{ij}^{w1}=0.05$ and $\beta_{ij}^{w2}=0.01$ for

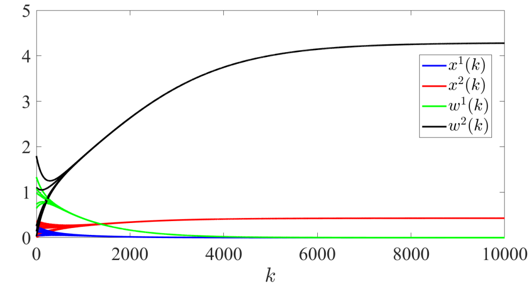

Figure 1: Since $\rho(M_f^1) < 1$, virus 1 is eradicated. For this simulation, we allow $\rho(M_f^2) > 1$ and it converges to an endemic equilibrium.

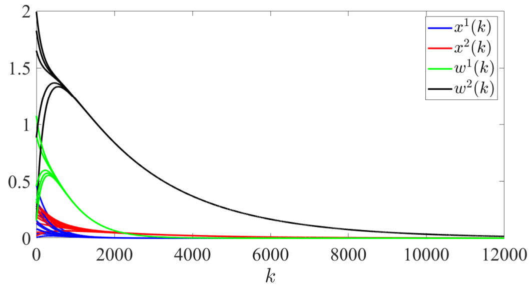

Figure 2: Since $\rho(M_f^1) < 1$ and $\rho(M_f^2) < 1$ both viruses are eradicated.

all $i \in [10]$, $j \in [5]$. Note that, for a given virus, the rate at which the population contaminates the infrastructure network is different from how the resource nodes contaminate the individuals.

The aforementioned choice of parameters results in $\rho(M_f^1) = 0.9994$ and $\rho(M_f^2) = 1.0012$. Consequently, consistent with the result in Theorem 1, the virus 1 is asymptotically eradicated across the network. Since $\rho(M_f^2) > 1$, virus 2 converges to some positive equilibrium which is consistent with (Cui et al., 2022, Theorem 3); see Figure 1.

Choosing a lower infection rate for virus 2, specifically, $\beta_i^2 = 0.08$, for all $i \in [10]$, and keeping the same values for the other parameters results in $\rho(M_f^1) = 0.9994$ and $\rho(M_f^2) = 0.9996$. Therefore, consistent with Corollary 1, both viruses are exponentially eradicated; see Figure 2. Observe that, even when the spectral radius for a particular virus is close to the unit circle, the virus can still be eradicated, thus indicating that the claim in Theorem 1 could possibly be established without the strict inequality.

Homogeneous spread in symmetric undirected graphs: For this set of simulations, we still assume that all the population nodes are connected to all the resource nodes. However, the interconnection graph for the population and resource networks are changing at each time step. At each layer, we randomly position each node in a 2-dimensional plane and it is connected to only those nodes that are within a given radius with an edge-weight of 1. Thus, $A^r(k) = A^r(k)^\top$ and $A_w^r(k) = A_w^r(k)^\top$. We also periodically change the infection and healing rates for each

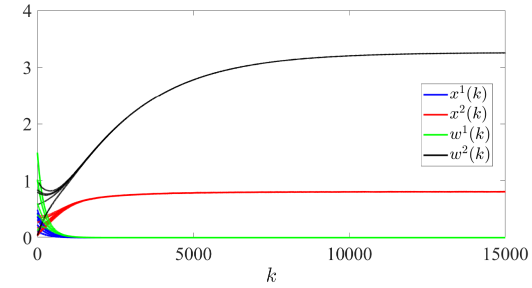

Figure 3: Simulations with a periodic change of homogeneous healing and infection rates in a symmetric time-varying network. Since $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^1(k)) < 1$, virus 1 is eradicated. Also, we allow $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^2(k)) > 1$ and the infection level of virus 2 across the population and resource nodes converges to an endemic equilibrium.

virus, where $\beta_i^1(k) \in [0.01, 0.1]$, $\beta_i^2(k) \in [0.2, 0.6]$, $\delta_i^1(k) \in [3,4]$, $\delta_i^2(k) \in [0.5, 2]$ for all $i \in [10]$, $\delta_j^{w1}(k) \in [3,4]$, $\delta_j^{w2}(k) \in [0.01, 1]$ for all $j \in [5]$, and $\beta_{ij}^{w1}(k) \in [0, 0.01]$, $\beta_{ij}^{w2}(k) \in [0.01, 0.3]$ for all $i \in [10]$, $j \in [5]$. Note that we allow virus 1 to not be transmitted at some time instances from the resource to the population nodes. We use the same values of $\beta_{ij}^{wr}(k)$ to construct the matrix $C_w^r(k)$, so that $B_w^r(k) = C_w^r(k)^\top$, for all $r \in [2]$.

With the aforementioned choice of parameters , we obtain $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^1(k)) = 0.9972$ and $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^2(k)) = 1.0047$. Therefore, virus 1 is eradicated both in the population and resource network, consistent with the result in Theorem 2; see Figure 3. Now, we change the set from which the parameters are selected to $\beta_i^1(k) \in [0.1, 0.2]$, $\beta_i^2(k) \in [0.2, 0.6]$, $\delta_i^1(k) \in [1,3]$, $\delta_i^2(k) \in [0.5, 2]$ for all $i \in [10]$, $\delta_j^{w1}(k) \in [1,4]$, $\delta_j^{w2}(k) = 1$ for all $j \in [5]$, and $\beta_{ij}^{w1}(k) \in [0.01, 0.2]$, $\beta_{ij}^{w2}(k) \in [0.01, 0.3]$ for all $i \in [10]$, $j \in [5]$. The new choices of parameters result in $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^1(k)) = 1$ and $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^2(k)) = 1.0047$. Again, the infection levels of virus 1 in both networks attain the healthy state which is consistent with the result in Proposition 3; see Figure 4. However, in Figures 3 and 4, the contamination of virus 2 across both networks asymptotically approaches an endemic equilibrium when $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^2(k)) > 1$. Through these two cases, we illustrate that for time-varying networks with homogeneous spread and symmetric graphs the eradicated state of virus r, for some $r \in [2]$, may possibly be stable under a wider range of model parameters than that identified in Proposition 3 - this is not surprising since, in the continuous-time setting, it suffices for the linearized system to be Hurwitz on average; see Paré et al. (2018). A corresponding result for the discrete-time setting is yet to be obtained – it is an ongoing focus. Separately, there is a possibility that when the reproduction number of virus r, for some $r \in [2]$, is larger than one, then virus r becomes endemic.

Heterogeneous spread in directed networks: For the simulation in Figure 5 we have that $\beta_i^1(k) \in [0.01, 0.045]$, $\beta_i^2(k) \in [0.2, 0.6]$, $\delta_i^1(k) \in [2,3]$, $\delta_i^2(k) \in$

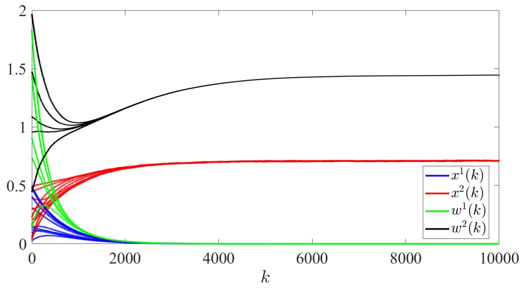

Figure 4: Simulation making changes in the healing and infection rates in contrast to the system in Figure 3. Since $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^1(k)) = 1$, virus 1 attains the DFE. As in the system of Figure 3, $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^2(k)) > 1$ and the contamination of virus 2 becomes endemic across all population and resource nodes.

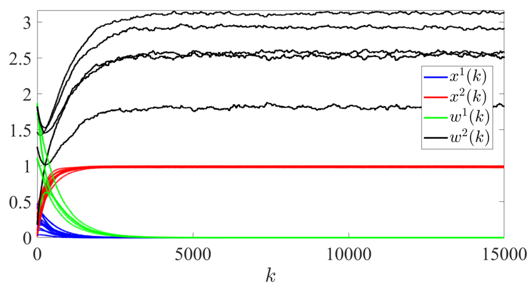

Figure 5: Since $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^1(k)) < 1$, virus 1 is eradicated. Still, virus 2 converges to an endemic equilibrium even when $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^2(k)) > 1$. Observe that, in contrast to the systems in Figure 3 and 4, the resource nodes converge to different equilibria.

$[0.05, 0.2]$ for all $i \in [10]$, and $\delta_j^{wr}(k) \in [1,2]$ for all $j \in [5]$, $r \in [2]$. The entries in matrices A^r and A_w^r are uniformly picked at random from the sets $[1,4]$ and $[1,2]$, respectively. Following the same strategy as in simulation for Figures 3 and 4, the entries of these matrices are changed at each step depending on how far the nodes are from each other in the population and infrastructure network. If they are beyond a certain radius, the corresponding entries are set to zero. Moreover, we assume that all the population nodes are connected to the resource nodes. Choosing the parameters as described before results in $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^1(k)) = 0.9993$ and $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^2(k)) = 1.0091$. Thus, consistent with the result in Theorem 3, virus 1 is eradicated. Observe that, virus 2 appears to converge to an endemic equilibrium which is not necessarily the same for all the nodes (e.g. the resource nodes converge to different endemic equilibrium while all the nodes in the population get the highest possible rate of infection with virus 2; see Figure 5).

To further explore the stability of the system, we increase the infection rate of the population of virus 1 and allow $\beta_i^1(k) = 0$ at random time steps for some $i \in [10]$. That is, there will be some nodes in the population that will only be contaminated with virus 1 by their interaction with the resource nodes. Also, in some time instances, we randomly pick population and resource nodes that are not able to heal themselves from virus 2, i.e., $\delta_i^2(k)$, $\delta_j^{w2} = 0$ for some $i \in [10]$, $j \in [5]$. With this parameter selection, Theorem 3 does not apply since the condition in Assumption 6 is violated. We obtain that $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^1(k)) = 1.0015$, and $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^2(k)) = 1.0090$. Two main insights can be taken from this system: despite $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^1(k)) > 1$, virus 1 is eradicated. Secondly, when the network is not capable of healing itself at some steps and $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^2(k)) > 1$, the infection levels for virus 2 converge to an endemic equilibrium; see Figure 6. Given that the system in Figure 6 has much lower healing rates for virus 2 than the system in Figure 5, the endemic equilibrium that the resource nodes attain is much higher.

Perturbation Analysis: Based on Theorem 4, we

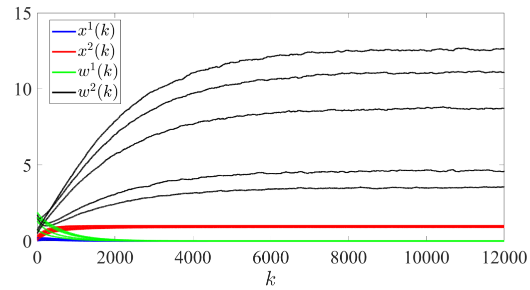

Figure 6: Simulations where some nodes in the network are not capable of healing themselves from virus 2 at random time instances. Nonetheless, even this system converges to some endemic equilibrium for virus 2. Also, it is able to eradicate virus 1 having $\sup_{k\in\mathbb{Z}_{\geq 0}}(M_f^1(k)) > 1$.

consider a system with parameters chosen for the simulations depicted in Figure 5. The entries of the matrix $\Delta^r(k)$ are uniformly picked at random from the set $[0, 1]$ at each step k. We use a logarithmic scale to search what is the maximum norm of $\bar{\Delta}^1(k)$ such that the eradicated state of virus 1 is GES as in the system without perturbation. With the given parameter selection, we obtain that $\|F^1(k)\| \leq 0.0773$ and $\zeta = 0.2361$; see Figure 7. Now, we deliberately increase the norm of the perturbation matrix $\Delta^1(k)$ to understand the behavior of the system. As expected, for a higher perturbation in the edges of the population network for the transmission of virus 1, the condition on the norm of $F^1(k)$ is violated (e.g. $\|F^1(k)\| \leq 0.3313$). With an increase of 4.24 times the norm of $\bar{\Delta}^1(k)$, virus 1 becomes endemic across the network and virus 2 is eradicated whereas in the original system the opposite occurs; see Figure 8. In the case of this system, we can argue that the network, despite the perturbations, is still stable under a wider range of parameters which is a behavior common to all the case studies we have presented in this section. Therefore, for this general setting with time-varying, asymmetric, heterogeneous spread, we can argue that the sufficient conditions established in this work guarantee the eradication of a virus, however, results on the endemic behavior of competing viruses with

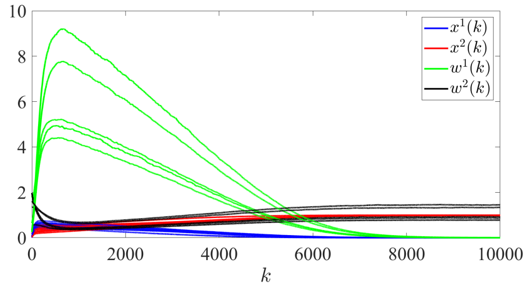

Figure 7: Simulation employing the same parameters as the system in Figure 5. For virus 1, $\zeta = 0.2361$ and it is GES since $\|F^1(k)\| \leq 0.0773$ while virus 2 still reaches an endemic equilibrium across the network.

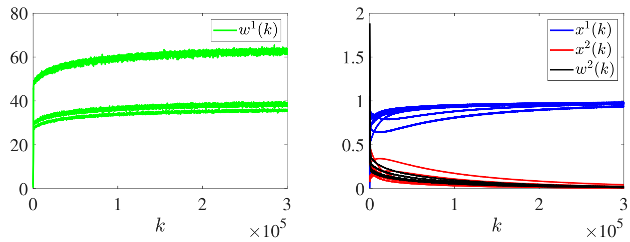

Figure 8: Resource nodes dynamics for virus 1 with the perturbation matrix $\Delta^1(k)$ increased by a factor of 4.24. As expected, the condition in Proposition 4 is violated ($\|F^1(k)\| > \zeta$). Virus 1 converges to an endemic equilibrium, but virus 2 is eradicated as a consequence of the perturbation.

infrastructure networks require future investigation.

7.2. Exploratory Simulations

Endemic Behavior for Time-Varying Case: So far, we have illustrated sufficient conditions for a virus to converge to the DFE for both types of virus spread (homogeneous/heterogeneous) and graphs (undirected/directed). In the previous simulations, either both viruses die out or one of the virus reaches an endemic equilibrium and the other one dies out. However, the situation where multiple viruses simultaneously remain endemic in the system is not considered. Hence, we now aim to investigate if it is possible for both viruses to simultaneously exhibit endemic behaviour when the graph is randomly varying at each time step for both the population and infrastructure networks. To this end, we create independently, identically sampled random graphs, the same way graphs were created in Section 7.1 "Homogeneous spread in symmetric undirected graphs." For the viral parameters, we use the following intervals for the healing and infection rates $\beta_i^1(k) \in [0.1, 50]$, $\beta_i^2(k) \in [0.2, 25]$, $\delta_i^1(k) \in [2, 3]$, $\delta_i^2(k) \in [2, 3]$ for all $i \in [10]$ while $\delta_i^1(k), \delta_j^{wr}(k)$ for all $i \in [5]$, $j \in [5]$, $r \in [2]$ are the same as in the simulation in Figure 5. Also, the entries of the matrices B_w^r and C_w^r are uniformly picked at random from the sets $[0, 0.4]$ and $[0.3]$, respectively. With the aforementioned parameter selection, we obtain that $\sup_{k \in \mathbb{Z}_{\geq 0}}(M_f^1(k)) = 1.3865$

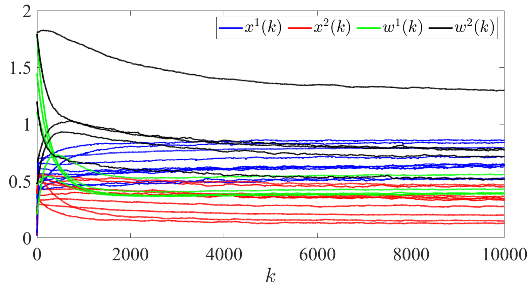

Figure 9: Simulation where the infection rates for both viruses are increased with respect to those used for the system in Figure 5. Even though the matrices A^r and A_w^r are randomly created at each step, both viruses coexist and reach an endemic equilibrium on average for both the population and infrastructure network.

and $\sup_{k \in \mathbb{Z}_{\geq 0}}(M_f^2(k)) = 1.1376$. Both viruses coexist in both the population and infrastructure network; see Figure 9. The key takeaway for this scenario is that even though the spectral radius for each virus is greater than one and the graph $\mathcal{G}^r(k)$ is randomly generated at each time step k, the system does not become chaotic. Note that Figure 9 is just one instantiation of the simulation. For other iterations of the simulations, both viruses did not always survive.

Periodic Epidemic Process: Next, we shift our focus to a particular class of time-varying systems, namely periodic time-varying systems. In (Gracy et al., 2020), a discrete-time periodic networked single-virus SIS model was analyzed. The authors showed through simulations the existence of a limit-cycle behavior when a sufficient condition for convergence to the DFE is violated. Here, we aim to check by simulations if it is the case that a limit cycle could exist and that it is possible for our system to reach this limit-cycle even when there are multiple (competitive) viruses present and the spread also gets exacerbated due to the presence of an infrastructure network. To this end, inspired by the example in (Gracy et al., 2020), we use a 64-node population network (i.e., $n = 64$) and we allow the adjacency matrix of this population network to vary periodically with periodicity three. For simplicity, we use homogeneous, non-mutating virus parameters with $\beta_i^1(k) = 10$, $\beta_i^2(k) = 3$, $\delta_i^1(k) = 33$ and $\delta_i^2(k) = 10$ for all $i \in [64]$. Also, $\delta_j^{w1}(k) = 1$ and $\delta_j^{w2}(k) = 5$ for all $j \in [5]$. We use the matrices B_w^r and C_w^r, for $r = 1, 2$, from the simulation in Figure 9. It turns out that with such a choice of model parameters, the system gives rise to a limit cycle. Furthermore, it can be seen that the system converges to the aforementioned limit cycle for both viruses at all the nodes in the population network; see Figure 10. Also, the infection level of both viruses attains a limit-cycle in the resource nodes, however, virus 1 starts increasing with no upper bound; see Figure 11.

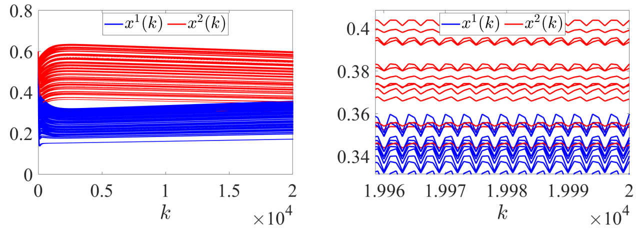

Figure 10: Infection level for the two viruses in the population network whose adjacency matrix varies periodically with periodicity three. Note that the infection levels converge to a limit cycle for both viruses at all nodes.

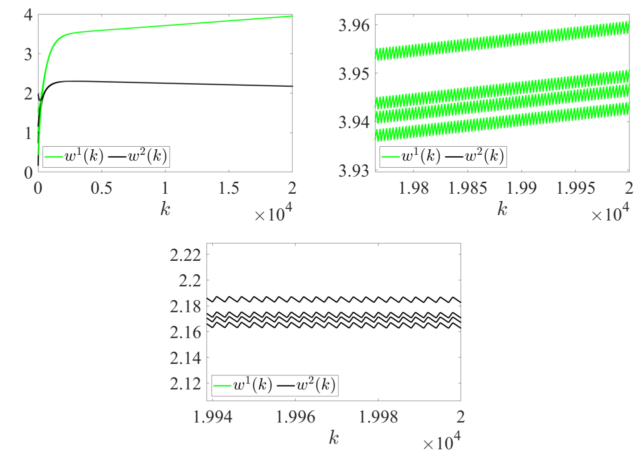

Figure 11: Contamination levels of the resource nodes with a periodic time-varying matrix A_w^r. The concentration of virus 1 in all the resource nodes increases unboundedly.

8. Conclusion

We studied the spread of multiple competing viruses over a population network and an infrastructure network using a discrete-time time-varying multi-competitive layered networked SIWS model. We first provided a sufficient condition for the eradication of a virus in exponential time. By relaxing said condition, we showed that virus eradication will still be possible, although it would happen asymptotically. Subsequently, for the case where the graph structure varies over time, under the assumption of homogeneous spread, we identified a sufficient condition for the eradication of a virus in exponential time. Thereafter, for the case when the graph is slowly varying, we provided a sufficient condition for exponential eradication of a virus even when the spread is not necessarily homogeneous. Moreover, we proved that the aforementioned sufficient condition is robust to variations in the graph structure of the population network provided that the variations are not too large. Finally, under the assumption that the spread is time-invariant, we provided a lower bound on the number of equilibria that our system possesses.

There are several interesting open problems. In no particular order, first, one could, inspired by the simulations in Section 7.2, aim to develop a comprehensive understanding of the endemic behavior for the time-varying case viz. existence and attractivity of endemic equilibrium and/or limit cycle, chaos, etc. Second, designing feedback control schemes such that the virus gets eradicated exponentially quickly could be of interest. Another line of research could involve identifying conditions for estimating the infection level in the population, given knowledge of infection levels in (a part of) the infrastructure network.

References

Ahn, H. J. and Hassibi, B. (2013), 'Global dynamics of epidemic spread over complex networks', *in Proc. 52nd IEEE Conference on Decision and Control,* pp. 4579–4585.

Ahrens, A. K., Selinka, H.-C., Mettenleiter, T. C., Beer, M. and Harder, T. C. (2022), 'Exploring surface water as a transmission medium of avian influenza viruses–systematic infection studies in mallards', *Emerging microbes & infections* **11**(1), 1250–1261.

Allen, L. J. (1994), 'Some discrete-time SI, SIR, and SIS epidemic models', *Mathematical Biosciences* **124**(1), 83–105.

Anderson, B. D. and Ye, M. (2023), 'Equilibria analysis of a networked bivirus epidemic model using poincaré–hopf and manifold theory', *SIAM Journal on Applied Dynamical Systems* **22**(4), 2856–2889.

Atkinson, K. E. (2008), *An Introduction to Numerical Analysis*, John Wiley & Sons.

Bahl, J., Vijaykrishna, D., Holmes, E. C., Smith, G. J. and Guan, Y. (2009), 'Gene flow and competitive exclusion of avian influenza a virus in natural reservoir hosts', *Virology* **390**(2), 289–297.

Bailey, N. T. et al. (1975), *The Mathematical Theory of Infectious Diseases and Its Applications*, Charles Griffin & Company Ltd, Bucks, U.K.

Baingana, B. and Giannakis, G. B. (2016), 'Tracking switched dynamic network topologies from information cascades', *IEEE Transactions on Signal Processing* **65**(4), 985–997.

Barrett, C. L., Bisset, K. R., Eubank, S. G., Feng, X. and Marathe, M. V. (2008), Episimdemics: an efficient algorithm for simulating the spread of infectious disease over large realistic social networks, *in* 'SC'08: Proceedings of the 2008 ACM/IEEE Conference on Supercomputing', IEEE, pp. 1–12.

Becker, N. (1979), 'The uses of epidemic models', *Biometrics* pp. 295–305.

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

Bloom, D. E., Cadarette, D. and Sevilla, J. (2018), 'Epidemics and economics', *Finance & Development* **55**(002).

Bokharaie, V. S., Mason, O. and Wirth, F. (2010), 'Spread of epidemics in time-dependent networks'.

Brauer, F., Feng, Z. and Castillo-Chavez, C. (2009), 'Discrete epidemic models', *Mathematical Biosciences & Engineering* **7**(1), 1–15.

Chakrabarti, D., Wang, Y., Wang, C., Leskovec, J. and Faloutsos, C. (2008), 'Epidemic thresholds in real networks', *ACM Transactions on Information and System Security (TISSEC)* **10**(4), 1–26.

Cui, S., Liu, F., Jardón-Kojakhmetov, H. and Cao, M. (2022), 'Discrete-time layered-network epidemics model with time-varying transition rates and multiple resources', *arXiv preprint arXiv:2206.07425* .

Desoer, C. (1970), 'Slowly varying discrete system $x_{i+1} = A_i x_i$', *Electronics Letters* **6**(11), 339–340.

Dunford, N. and Schwartz, J. T. (1958), *Linear Operators Part I: General Theory*, Interscience publishers New York.

Fall, A., Iggidr, A., Sallet, G. and Tewa, J.-J. (2007), 'Epidemiological models and Lyapunov functions', *Mathematical Modelling of Natural Phenomena* **2**(1), 62–83.

Gómez, S., Arenas, A., Borge-Holthoefer, J., Meloni, S. and Moreno, Y. (2010), 'Discrete-time markov chain approach to contact-based disease spreading in complex networks', *Europhysics Letters* **89**(3), 38009.

Gracy, S., Morarescu, I. C., Varma, V. S. and Paré, P. E. (2022), Analysis and on/off lockdown control for time-varying SIS epidemics with a shared resource, *in* 'Proc. 2022 European Control Conference (ECC)', IEEE, pp. 1660–1665.

Gracy, S., Paré, P. E., Sandberg, H. and Johansson, K. H. (2020), 'Analysis and distributed control of periodic epidemic processes', *IEEE Transactions on Control of Network Systems* **8**(1), 123–134.

Gracy, S., Wang, Y., Paré, P. E. and Uribe, C. A. (2023), Multi-competitive virus spread over a time-varying networked SIS model with an infrastructure network, *in* 'Proc. 2023 IFAC World Congress (IFAC WC)', pp. 19–24.
URL: *https://sciencedirect.com/science/article/pii/S2405896323017913*

Hamer, W. H. (1906), *Epidemic Disease in England: The Evidence of Variability and of Persistency of Type*, Bedford Press.

Han, S., Preciado, V. M., Nowzari, C. and Pappas, G. J. (2015), 'Data-driven network resource allocation for controlling spreading processes', *IEEE Transactions on Network Science and Engineering* **2**(4), 127–138.

Hethcote, H. W. (2000), 'The mathematics of infectious diseases', *SIAM Review* **42**(4), 599–653.

Horn, R. A. and Johnson, C. R. (2012), *Matrix Analysis*, Cambridge University Press.

Huang, H., Chen, Y. and Ma, Y. (2021), 'Modeling the competitive diffusions of rumor and knowledge and the impacts on epidemic spreading', *Applied mathematics and computation* **388**, 125536.

Jackson, C. (2009), 'History lessons: The Asian flu pandemic', *British Journal of General Practice* **59**(565), 622–623.

Janson, A., Gracy, S., Paré, P. E., Sandberg, H. and Johansson, K. H. (2020), 'Networked multi-virus spread with a shared resource: Analysis and mitigation strategies', *arXiv preprint arXiv:2011.07569* .

Johnson, N. P. and Mueller, J. (2002), 'Updating the accounts: Global mortality of the 1918-1920 "Spanish" influenza pandemic', *Bulletin of the History of Medicine* pp. 105–115.

Karrer, B. and Newman, M. E. (2011), 'Competing epidemics on complex networks', *Physical review E* **84**(3), 036106.

Kato, T. (1960), 'Estimation of iterated matrices, with application to the von Neumann condition', *Numerische Mathematik* **2**(1), 22–29.

Kermack, W. O. and McKendrick, A. G. (1932), 'Contributions to the mathematical theory of epidemics. ii.—the problem of endemicity', *Proceedings of the Royal Society of London. Series A, containing papers of a mathematical and physical character* **138**(834), 55–83.

Khanafer, A., Başar, T. and Gharesifard, B. (2016), 'Stability of epidemic models over directed graphs: A positive systems approach', *Automatica* **74**, 126–134.

Lajmanovich, A. and Yorke, J. A. (1976), 'A deterministic model for gonorrhea in a nonhomogeneous population', *Mathematical Biosciences* **28**(3-4), 221–236.

Lin, Y., Xuan, W., Ren, R. and Liu, J. (2021), On a discrete-time network sis model with opinion dynamics, *in* '2021 60th IEEE Conference on Decision and Control (CDC)', IEEE, pp. 2098–2103.

Liu, J. et al. (2019*a*), 'Analysis and control of a continuous-time bi-virus model', *IEEE Transactions on Automatic Control* **64**(12), 4891–4906.

Liu, J. et al. (2019*b*), A networked SIS disease dynamics model with a waterborne pathogen, *in* 'Proceedings of the 2019 American Control Conference (ACC)', IEEE, pp. 2735–2740.

Liu, Q.-H., Xiong, X., Zhang, Q. and Perra, N. (2018), 'Epidemic spreading on time-varying multiplex networks', *Physical review E* **98**(6), 062303.

Martcheva, M. (2009), 'A non-autonomous multi-strain sis epidemic model', *Journal of biological dynamics* **3**(2-3), 235–251.

Mason, O., Wirth, F. R., Rami, M. A. and Bokharaie, V. (2014), 'Stability criteria for SIS epidemiological models under switching policies', *Discrete and Continuous Dynamical Systems Ser.* **19**(9), 2865–2887.

Mei, W., Mohagheghi, S., Zampieri, S. and Bullo, F. (2017), 'On the dynamics of deterministic epidemic propagation over networks', *Annual Reviews in Control* **44**, 116–128.

Newman, M. E. (2003), 'The structure and function of complex networks', *SIAM Review* **45**(2), 167–256.

Nowzari, C., Preciado, V. M. and Pappas, G. J. (2016), 'Analysis and control of epidemics: A survey of spreading processes on complex networks', *IEEE Control Systems Magazine* **36**(1), 26–46.

Paré, P. E., Beck, C. L. and Nedić, A. (2018), 'Epidemic processes over time-varying networks', *IEEE Trans. on Control of Network Systems* **5**(3), 1322–1334.

Paré, P. E., Gracy, S., Sandberg, H. and Johansson, K. H. (2020a), 'Data-driven distributed mitigation strategies and analysis of mutating epidemic processes', *Proc. 59th IEEE Conference on Decision and Control,* pp. 6138–6143.

Paré, P. E., Janson, A., Gracy, S., Liu, J., Sandberg, H. and Johansson, K. H. (2022), 'Multilayer SIS model with an infrastructure network', *IEEE Transactions on Control of Network Systems* **10**(1), 295–307.

Paré, P. E., Liu, J., Beck, C. L., Kirwan, B. E. and Başar, T. (2020b), 'Analysis, estimation, and validation of discrete-time epidemic processes', *IEEE Transactions on Control Systems Technology* **28**(1), 79–93.

Paré, P. E., Liu, J., Beck, C. L., Nedić, A. and Başar, T. (2021), 'Multi-competitive viruses over time-varying networks with mutations and human awareness', *Automatica* **123**, 109330.

Paré, P. E., Vrabac, D., Sandberg, H. and Johansson, K. H. (2020c), 'Analysis, online estimation, and validation of a competing virus model', *in Proc. American Control Conference,* pp. 2556–2561.

Peng, C., Jin, X. and Shi, M. (2010), 'Epidemic threshold and immunization on generalized networks', *Physica A: Statistical Mechanics and its Applications* **389**(3), 549–560.

Prakash, B. A., Tong, H., Valler, N., Faloutsos, M. and Faloutsos, C. (2010), 'Virus propagation on time-varying networks: Theory and immunization algorithms', *Joint European Conference on Machine Learning and Knowledge Discovery in Databases* pp. 99–114.

Ross, R. (1911), *The Prevention of Malaria, 2nd edn*, London, UK: Murray.[Google Scholar].

Rugh, W. J. (1996), *Linear System Theory*, Vol. 2, Prentice Hall Upper Saddle River, NJ.

Sagar, V., Zhao, Y. and Sen, A. (2018), 'Effect of time varying transmission rates on the coupled dynamics of epidemic and awareness over a multiplex network', *Chaos: An Interdisciplinary Journal of Nonlinear Science* **28**(11).

Sahneh, F. D. and Scoglio, C. (2014), 'Competitive epidemic spreading over arbitrary multilayer networks', *Physical Review E* **89**(6), 062817.

Santos, A., Moura, J. M. and Xavier, J. M. (2015), 'Bi-virus SIS epidemics over networks: Qualitative analysis', *IEEE Transactions on Network Science and Engineering* **2**(1), 17–29.

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

Sun, K., Baronchelli, A. and Perra, N. (2015), 'Contrasting effects of strong ties on sir and sis processes in temporal networks', *The European Physical Journal B* **88**, 1–8.

Van Mieghem, P., Omic, J. and Kooij, R. (2008), 'Virus spread in networks', *IEEE/ACM Transactions On Networking* **17**(1), 1–14.

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

Vestergaard, C. L. and Génois, M. (2015), 'Temporal gillespie algorithm: fast simulation of contagion processes on time-varying networks', *PLoS computational biology* **11**(10), e1004579.

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

Wang, Y., Chakrabarti, D., Wang, C. and Faloutsos, C. (2003), 'Epidemic spreading in real networks: An eigenvalue viewpoint', *in Proc. 22nd International Symposium on Reliable Distributed Systems* pp. 25–34.

WHO (n.d.), 'Ebola virus disease – democratic republic of the congo', `https://www.who.int/csr/don/30-january-2020-ebola-drc/en/`. Accessed: 2020-02-03.

Widerström, M., Schönning, C., Lilja, M., Lebbad, M., Ljung, T., Allestam, G., Ferm, M., Björkholm, B., Hansen, A., Hiltula, J. et al. (2014), 'Large outbreak of Cryptosporidium hominis infection transmitted through the public water supply, Sweden', *Emerging Infectious Diseases* **20**(4), 581.

World Health Organization (2021), 'Novel coronavirus (2019-nCoV)', https://www.who.int/westernpacific/emergencies/novel-coronavirus. Accessed: 2021-11-15.

Ye, M., Anderson, B. D. O., Janson, A., Gracy, S. and Johansson, K. H. (2023), 'Competitive epidemic networks with multiple survival-of-the-fittest outcomes', *Systems & Control Letters* . Note: Under Review.
URL: *https://arxiv.org/pdf/2111.06538.pdf*

Ye, M., Anderson, B. D. O. and Liu, J. (2022), 'Convergence and equilibria analysis of a networked bivirus epidemic model', *SIAM Journal on Control and Optimization* **60**(2), S323–S346.

Ye, M., Liu, J., Anderson, B. D. O. and Cao, M. (2022), 'Applications of the Poincare-Hopf Theorem: Epidemic models and Lotka-Volterra systems', *IEEE Transactions on Automatic Control* **67**(4), 1609–1624.