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Analysis of a Networked SIS Multi-Virus Model with a Shared Resource

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Abstract: In this paper, we introduce a continuous-time competing virus model with a shared resource. We say that the system is in the healthy state if all the agents are healthy, and the shared resource is not contaminated. If the epidemic remains persistent, and the shared resource is contaminated, we say that the system is in the endemic state. First of all we show, under appropriate assumptions, that the model we introduce is well-posed. Secondly, we establish sufficient conditions for exponential (resp. asymptotic) eradication of a virus. Thirdly, for the single-virus case with a shared resource, we establish conditions that lead to existence of an endemic equilibrium. Finally, we provide a necessary and sufficient condition for uniqueness of the healthy state.

Keywords: Epidemic processes, Competing viruses, Shared resource, Exponential stability, Asymptotic Stability, Endemic equilibrium

1. INTRODUCTION

Epidemics, or more broadly spreading processes, have been of interest to the research community since Bernoulli's seminal paper (Bernoulli, 1760). The underlying goal behind these research efforts is to understand under what conditions would the epidemic become extinct, and use the knowledge of these conditions to design mitigation strategies. Towards this end, various models have been proposed and studied in the literature; susceptible-infected-susceptible (SIS), susceptible-infected-recovered (SIR), susceptible-exposed-infected-recovered (SEIR), etc. In this paper we focus on SIS models.

In an SIS model, an agent is either in the susceptible or infected state. A healthy agent could become infected at some infection rate β, scaled by the interactions it has with its neighbors. Each agent has its own healing rate δ, that is, the rate at which it recovers from the infection. It is assumed that the total number of agents in the network is large enough to ignore stochastic effects (Anderson and Robert, 1991) and that the number remains fixed (Lajmanovich and Yorke, 1976).

The analysis of SIS models has been a major research thrust in mathematical epidemiology over the last several decades; see for instance (Lajmanovich and Yorke, 1976; Fall et al., 2007; Khanafer et al., 2016). One of the drawbacks with the traditional SIS model is that it is not amenable for understanding epidemic spread when there is a shared resource in the network that could significantly worsen the spread; for instance public transit, a neighborhood supermarket, a community well, etc. A networked continuous-time *single-virus* SIS model that incorporates a shared resource was first proposed by Liu et al. (2019a), referred to as susceptible-infected-water-susceptible (SIWS) model. For the SIWS model, sufficient conditions for asymptotic convergence to the healthy state (defined as the state where each agent is healthy, and the shared resource is contamination-free) has been provided by Liu et al. (2019a). However, no theoretical guarantees were provided for the endemic behavior (i.e., where the virus remains persistent) of this model.

Yet another drawback with the SIS model is that it does not account for those scenarios where multiple strains of a virus could be simultaneously active within a population. In particular, it is possible that different virus strains *compete* with each other to infect the population. That is, each agent can be infected by one, and only one, of the multiple virus strains prevalent (Nowak, 1991). Such a phenomenon may also be exhibited in the context of opinion-spread, where incompatible ideas spread on different social networks (Sahneh and Scoglio, 2014). Another application is pathogen interaction on overlay networks with SIR dynamics (Funk and Jansen, 2010). Additionally, the notion of competing viruses could find applications to adoption of competing products in a marketplace, political stances, and alternative farming practices (Paré et al., 2020). Thus, in light of these shortcomings with the traditional SIS model, and consequently the SIWS model, we propose an extension.

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SIS models that account for multiple competing viruses have been a recent focus of the research community; see for instance (Wei et al., 2013; Watkins et al., 2016). A competing continuous-time time-invariant bi-virus model has been presented and studied by Liu et al. (2019b). Recently, by extending the setup in (Liu et al., 2019b) to also account for multiple competing viruses and time-varying topologies, a more general model has been presented by Paré et al. (2017). However, none of these works account for the presence of a shared resource in the network.

In this paper, we propose a SIS-type model that accounts for multiple competing viruses *and* a shared resource, which might (possibly) be contaminated. Our main contributions, then, are the following:

i) establish sufficient conditions for exponential convergence to the healthy state;
ii) establish a weaker sufficient condition for asymptotic convergence to the healthy state;
iii) establish conditions that give rise to an endemic behavior, and, thereby, show that the weaker sufficient condition is also necessary for uniqueness of the healthy state equilibrium.

Outline

The rest of this paper is organized as follows. We conclude the present section by listing all the needed notation. In Section 2 we derive the SIWS model that accounts for multiple competing viruses, and formally state the problem of interest. The main results are presented in Section 3. We illustrate our theoretical findings, via simulations, in Section 4. Finally, we summarize the results in Section 5.

Notation

For any positive integer n, we use $[n]$ to denote the set $\{1, 2, ..., n\}$. The i^{th} entry of a vector x will be denoted by x_i. We use $\mathbf{0}$ and $\mathbf{1}$ to denote the vectors whose entries all equal 0 and 1, respectively, and use I to denote the identity matrix, while the sizes of the vectors and matrix are to be understood from the context. For a vector x we denote its diagonalization with $\text{diag}(x)$. For any two real vectors $a, b \in \mathbb{R}^n$, we write $a \geq b$ if $a_i \geq b_i$ for all $i \in [n]$, $a > b$ if $a \geq b$ and $a \neq b$, and $a \gg b$ if $a_i > b_i$ for all $i \in [n]$. For a real square matrix M, we use $\sigma(M)$ to denote the spectrum of M, use $\rho(M)$ to denote the spectral radius of M, and $s(M)$ to denote the largest real part among the eigenvalues of M, i.e., $s(M) = \max\{\text{Re}(\lambda) : \lambda \in \sigma(\text{M})\}$. We denote a subset A of a set B by $A \subset B$.

2. THE MODEL

Consider a network of n nodes, where each node represents a population subgroup, and a common resource W being shared among the n nodes. Suppose that there are m viruses active in the network. A node becomes infected as a consequence of either coming in contact with other infected nodes, or due to contact with the (possibly) contaminated shared resource. We assume that the viruses are competing with each other to infect each node in the network, which implies that, at a given time instant, an agent may get infected by no more than one virus. The spread of the competing m viruses among the n nodes can be represented by a directed graph $\mathcal{G}$, with existence of a

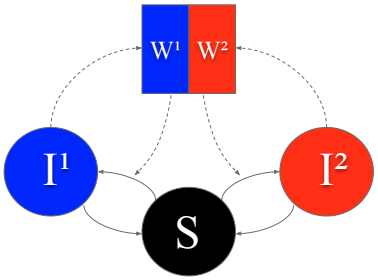

Fig. 1. Visualization of the model for the case when $m = 2$. An individual is either susceptible (S) or infected with virus 1 (I^1), or infected with virus 2 (I^2). The shared resource (W) is contaminated by individuals infected with either virus, and in turn augments the corresponding infection rate.

directed edge from node j to node i if individuals in node j can infect those in node i. We also assume that not only does a node get (possibly) infected due to contact with W but also that W could be contaminated whenever an infected node comes in contact with it. Thus, each node in $\mathcal{G}$ possibly has bidirectional connections with W.

Each population node contains N_i individuals and has a birth rate μ_i, and death rate $\bar{\mu}_i$. In each node, at time $t \geq 0$, $S_i(t)$ is the number of susceptible individuals, while $I_i^k(t)$ is the number of individuals infected by virus k. Individuals infected by virus k in node i have a recovery rate γ_i^k back to the susceptible state. We denote the node-to-node infection rate with respect to virus k by $\alpha_{ij}^k \geq 0$. Clearly, if node j is not connected to node i, then, for $k \in [m]$, $\alpha_{ij}^k = 0$. The shared resource W holds a viral mass of each type $k \in [m]$, which is denoted by $W^k(t)$, decaying at a rate δ_w^k and growing at a rate proportional to the sum of $I_i^k(t)$ scaled by ζ_i^k. We denote the resource-to-node infection rate for node i with respect to virus k by α_{iw}^k. A visualization of the model is provided in Figure 1.

The evolution of the number of susceptible and infected individuals in node i can be represented as follows:

$$\begin{aligned}
\dot{S}_i(t) =& \mu_i N_i - \bar{\mu}_i S_i(t) + \textstyle\sum_{k=1}^m \gamma_i^k I_i^k(t) \\
&- \textstyle\sum_{k=1}^m \big(\alpha_{iw}^k W^k(t) - \sum_{j=1}^n \alpha_{ij}^k \frac{I_j^k(t)}{N_i}\big) S_i(t), \\
\dot{I}_i^k(t) =& -\ (\bar{\mu}_i + \gamma_i^k) I_i^k(t) \\
&+ \big(\alpha_{iw}^k W^k(t) + \textstyle\sum_{j=1}^n \alpha_{ij}^k \frac{I_j^k(t)}{N_i}\big) S_i(t), \\
\dot{W}^k(t) =& -\delta_w^k W^k(t) + \textstyle\sum_{j=1}^n \zeta_j^k I_j^k(t).
\end{aligned} \tag{1}$$

We define new variables to simplify the system. Let:

$$p_i^k(t) = \frac{I_i^k(t)}{N_i},\ z^k(t) = \frac{\delta_w^k W^k(t)}{\sum_{j=1}^n \zeta_j^k N_j},\ \delta_i^k = \gamma_i^k + \mu_i,$$

$$\beta_{ij}^k = \alpha_{ij}^k \frac{N_j}{N_i},\ \beta_{iw}^k = \frac{\alpha_{iw}^k}{\delta_w^k} \textstyle\sum_{j=1}^n \zeta_j^k N_j, \text{ and } c_i^k = \frac{\zeta_i N_i}{\sum_{j=1}^n \zeta_j^k N_j}.$$

Then, assuming that the birth rates and the death rates are equal, (1) can be rewritten as:

$$\begin{aligned}
\dot{p}_i^k(t) =& -\delta_i^k p_i^k(t) + \big(1 - \textstyle\sum_{l=1}^m p_i^l(t)\big)\big(\beta_{iw}^k z^k(t) \\
&+ \textstyle\sum_{j=1}^n \beta_{ij}^k p_j^k(t)\big), \\
\dot{z}^k(t) =& \delta_w^k\big(-z^k(t) + \textstyle\sum_{i=1}^n c_i^k p_i^k(t)\big).
\end{aligned} \tag{2}$$

In matrix form, (2) can be rewritten as:

$$\begin{aligned}\dot{p}^k(t) &= \Big(\big(I - \textstyle\sum_{l=1}^m P^l(t)\big)B^k - D^k\Big)p^k(t) \\ &\quad + \big(I - \textstyle\sum_{l=1}^m P^l(t)\big)b^k z^k(t), \\ \dot{z}^k(t) &= \delta_w^k\big(-z^k(t) + c^k p^k(t)\big),\end{aligned} \tag{3}$$

where $P^k(t)$ is the diagonal matrix of $p^k(t)$, B^k is the $n \times n$-matrix with β_{ij}^k denoting the element corresponding to the i^{th} row and j^{th} column, D^k is the diagonal matrix with δ_i^k along the diagonal, b^k is a column vector with β_{iw}^k is element i, and c^k is a row vector with c_i^k as element i. To simplify further, define:

$$y^k(t) = \begin{bmatrix} p^k(t) \\ z^k(t) \end{bmatrix},\ y(t) = \begin{bmatrix} y^1(t) \\ \vdots \\ y^m(t) \end{bmatrix},\ B_w^k = \begin{bmatrix} B^k & b^k \\ \delta_w^k c^k & 0 \end{bmatrix},$$

$$D_w^k = \begin{bmatrix} D^k & 0 \\ 0 & \delta_w^k \end{bmatrix},\ X\big(y(t)\big) = \begin{bmatrix} \sum_{l=1}^m P^l(t) & 0 \\ 0 & 0 \end{bmatrix}.$$

Hence, (3) can be rewritten as:

$$\dot{y}^k(t) = \big(-D_w^k + B_w^k - X(y(t))B_w^k\big)y^k(t). \tag{4}$$

With the setup as given in (4) in place, we formally state the problems being investigated in this paper.

i) Under what conditions is a virus eradicated exponentially fast?
ii) Under what conditions is a virus eradicated asymptotically?
iii) Under what condition does a virus remain persistent in the population and the shared resource?

Before we address the aforementioned questions, we point out connections between the setup considered in the present work, and those in the existing literature.

Remark 1. Note that if $m = 1$, system (4) coincides with the model proposed by Liu et al. (2019a); see (Liu et al., 2019a, Equation (12)). ■

In the rest of this paper, when considering the single-virus case, i.e., $m = 1$, we drop the superscripts identifying the virus.

Defining $A^k(y(t)) = \big(-D_w^k + B_w^k - X(y(t))B_w^k\big)$, and by gathering all viruses into the same system, we can rewrite (4) as follows:

$$\dot{y}(t) = \begin{bmatrix} A^1\big(y(t)\big) & 0 & \dots & 0 \\ 0 & A^2\big(y(t)\big) & \dots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \dots & A^m\big(y(t)\big) \end{bmatrix} y(t). \tag{5}$$

Remark 2. Note that if we let $b^k = \mathbf{0}$ for all k in $[m]$, the influence of the shared resource on the network is nullified. Then the multi-virus dynamics of the population nodes in (5) are equivalent to the time-invariant multi-virus setup of Paré et al. (2017). ■

2.1 Assumptions

In order for the (4) to be well-defined and realistic, we make the following assumptions.

Assumption 3. Suppose that $\delta_i^k > 0, \delta_w^k > 0, \beta_{ij}^k \geq 0, \beta_{iw}^k \geq 0$ and $c_i^k \geq 0$ for all i, j in $[n]$ and k in $[m]$, with $c_l^k > 0$ for at least one l in $[n]$. ■

Note that if Assumption 3 holds, then B_w^k is a non-negative matrix and D_w^k is a positive diagonal matrix. A real square matrix M is said to be a Metzler matrix if the elements outside the main diagonal are non-negative. Thus $(B_w^k - D_w^k)$ is a Metzler matrix.

As a consequence of Assumption 3, we can restrict our analysis to the sets $S = \{y(t) : p^k(t) \in [0,1]^n,\ z^k(t) \in [0,\infty)\ \forall\, k \in [m]\}$ and $S^k = \{y^k(t) : p^k(t) \in [0,1]^n,\ z^k(t) \in [0,\infty)\}$. Since $p_i^k(t)$ is to be interpreted as a fraction of a population and $z^k(t)$ is supposed to be a non-negative quantity, these sets represent the sensible domain of the system. That is, for any $k \in [m]$, if $p^k(t)$ takes values outside the set $[0,1]^n$, then those values would lack physical meaning. The following lemma shows that once the dynamics of (5) enter the set S, it never leaves this set.

Lemma 4. Let Assumption 3 hold. Then S is positively invariant with respect to (5), and if $y(t) \in S$ for all $t \geq 0$, S^k is positively invariant with respect to (4).

Proof. Note that if $p_i^k(t) = 1$ then $\dot{p}_i^k(t) < 0$, so if $p_i^k(0) \leq 1$ then $p_i^k(t) \leq 1$ for all $t \geq 0$. Further, if $p_i^k(t) = 0$ and $y(t) \geq \mathbf{0}$ then $\dot{p}_i^k(t) \geq 0$ for all $t \geq 0$. Similarly, if $z^k(t) = 0$ and $y(t) \geq \mathbf{0}$ then $\dot{z}^k(t) \geq 0$. Thus $y(t) \geq \mathbf{0}$ if $y(0) \geq \mathbf{0}$ for all $t \geq 0$. □

3. MAIN RESULTS

In this section we present the equilibria of the system and some stability results, under suitable assumptions.

We say that a virus $k \in [m]$ is in the eradicated state if $y^k = \mathbf{0}$. It is immediate that $y^k = \mathbf{0}$ is an equilibrium of (4) with respect to virus k. If all $k \in [m]$ viruses are in the eradicated state then we say the system is in the healthy state $y = \mathbf{0}$, which is an equilibrium of (5). Note that when $m = 1$, the notions of healthy state and eradicated state coincide.

We present a sufficient condition for exponential stability of an eradicated state in the following theorem.

Theorem 5. Consider (4) under Assumption 3 and assume that $y(0) \in S$. Suppose that for some virus k we have that $s(B_w^k - D_w^k) < 0$. Then the eradicated state of virus k is exponentially stable with a domain of attraction containing S^k. ■

Proof: See Appendix. □

Theorem 5 answers question i) in Section 2. Note that for the same condition as in Theorem 5, (Liu et al., 2019b, Proposition 2) showed only *local* exponential stability, whereas Theorem 5 holds globally, on the sensible domain. Hence, Theorem 5 is a stronger version of (Liu et al., 2019b, Proposition 2) when $m = 1$, and more general since it applies to the competing virus case. In particular, Theorem 5 says that insofar that the matrix $(B_w^k - D_w^k)$ is Hurwitz, then, irrespective of the initial condition of the network, virus k is eradicated exponentially fast.

Observe that while Theorem 5 guarantees exponential eradication of virus k, the condition there is rather stringent. It is, therefore, pertinent to ask whether (or not) eradication of virus k can be achieved even if the condition

in Theorem 5 were to be relaxed. In such a case, one would naturally expect the speed of eradication to decrease. The following theorem provides a sufficient condition for asymptotic eradication of virus k.

Theorem 6. Consider (4). Let Assumption 3 hold and assume that $y(0) \in S$. Suppose that for some virus k we have $s(B_w^k - D_w^k) \leq 0$, $c_i^k > 0$, $\beta_{iw}^k > 0$ for all $i \in [n]$ and that the matrix B^k is irreducible. Then the eradicated state of virus k is asymptotically stable with a domain of attraction containing S^k. ■

Proof: See Appendix. □

Theorem 6 answers question ii) in Section 2. Observe that, particularized for the single-virus case, Theorem 6 coincides with (Liu et al., 2019a, Theorem 1). In order to understand Theorem 6 from an epidemiological standpoint, we need the following lemma for irreducible non-negative matrices:

Lemma 7. (Liu et al., 2019b, Proposition 1) Suppose that Λ is a negative diagonal matrix and N is an irreducible non-negative square matrix. Let $M = \Lambda + N$. Then, $s(M) < 0$ if and only if $\rho(-\Lambda^{-1}N) < 1, s(M) = 0$ if and only if $\rho(-\Lambda^{-1}N) = 1$, and $s(M) > 0$ if and only if $\rho(-\Lambda^{-1}N) > 1$. ■

Observe that, by Lemma 7, the condition in Theorem 6 is equivalent to $\rho((D_w^k)^{-1}B_w^k) \leq 1$. Therefore, we can interpret $\rho((D_w^k)^{-1}B_w^k)$ as a generalization of the reproduction number of the virus in the network, that is, the number of agents that become infection by an infected agent on the average. As such Theorem 6 states that if the reproduction number of virus k is less than or equal to one, then virus k will asymptotically converge to its eradicated state.

It is natural to ask what sort of behavior (4) would exhibit when the condition in Theorem 6 is violated. The next theorem addresses this question when $m = 1$.

Theorem 8. Consider (4) under Assumption 3 with $m = 1$. Suppose that B_w is irreducible and $s(B_w - D_w) > 0$. Then there exists at least one non-zero equilibrium in S. ■

Proof: See Appendix. □

Theorem 8 partly addresses question iii) in Section 2. It provides sufficient conditions for the existence of a non-zero equilibrium in a single-virus system, which was not shown by Liu et al. (2019b). By Lemma 7, if the condition in Theorem 8 is satisfied, then $\rho(D_w^{-1}B_w) > 1$, thus implying that the virus remains persistent in the population and contaminates the resource, further supporting the generalized reproduction number interpretation.

Combining Theorems 6 and 8, we immediately obtain a necessary and sufficient condition for uniqueness of the eradicated state in a single-virus system.

Theorem 9. Consider (4) under Assumption 3 with $m = 1$. Suppose that B is irreducible, $c_i > 0$ and $\beta_{iw} > 0$ for all $i \in [n]$. Then the healthy state is the unique equilibrium of (4) in S if, and only if, $s(B_w - D_w) \leq 0$. ■

4. SIMULATIONS

In this section, we present simulations to illustrate our theoretical findings. To this end, we use the city of Stockholm as the setting. In particular, the major districts of Stockholm, namely, Kungsholmen, Vasastan, Östermalm, Norrmalm, Gamla Stan and Södermalm, are taken to be the population nodes, and we view the public transportation system SL (Storstockholms Lokaltrafik) as the shared resource; see Figure 2.

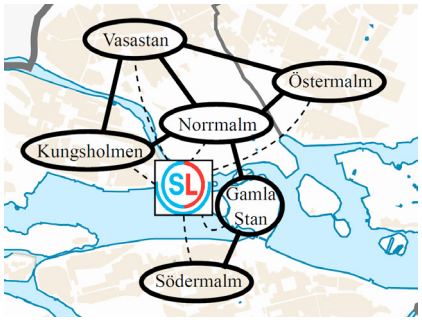

Fig. 2. A map of the relevant districts in Stockholm (CC (2016)). All districts are connected to the public transportation system (SL).

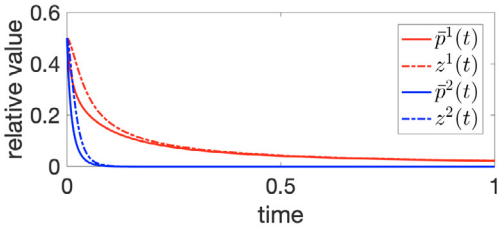

Fig. 3. Simulation with two viruses, both (red and blue) approaching eradication. The average infection rate by virus k in the network is denoted by $\bar{p}^k(t)$.

We consider two competing viruses spreading across the city, namely virus 1 and virus 2. Thus, $n = 6$, and $m = 2$. The spread parameters β_{ij}^k, for $k \in [2]$, are taken to be 1 if district i is adjacent to district j, and 0 otherwise. We also assume that each district is connected to the shared resource, the public transportation system, with $\beta_{iw}^k = 1$ and $c_i^k = 1/6$, for all $i \in [6]$, and each virus $k \in [2]$. As such the matrix B_w^k is irreducible for both viruses $k \in [2]$.

We begin by considering the case where the shared resource is contaminated by both viruses ($z^k(0) = 0.5$), and in each population node, half of the population is infected by virus 1 and the other half is infected by virus 2 ($p_i^k(0) = 0.5$). Further, we set $\delta_i^1 = 4.6$, $\delta_w^1 = 4.6$, $\delta_i^2 = 10$, and $\delta_w^2 = 10$. As a consequence, $s(B_w^1 - D_w^1) = -0.005$, and $s(B_w^2 - D_w^2) = -4.6$. In line with the result in Theorem 5, both the viruses are eradicated exponentially fast; see Figure 3. That is, both the viruses spreading across the above-mentioned districts of Stockholm (see Figure 2) disappear, and therefore the population in these districts become healthy. It can be observed that virus 2 is eradicated much more quickly than virus 1, which occurs since $s(B_w^2 - D_w^2)$ is much farther away from the origin than $s(B_w^1 - D_w^1)$.

Next we consider the case where initially all population nodes are infection-free ($p^k(0) = \mathbf{0}$), while the shared resource is contaminated by both viruses ($z^k(0) = 0.5$). Further, we set $\delta_i^1 = 1$, $\delta_w^1 = 1$, $\delta_i^2 = 5$, and $\delta_w^2 = 5$. That is, the recovery rate of virus 1 is slower compared to

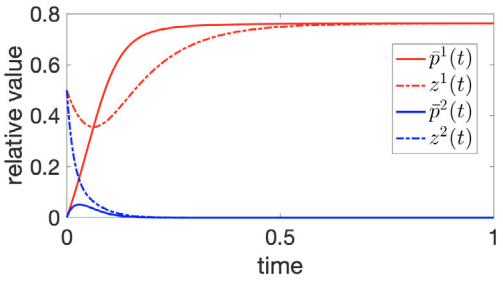

Fig. 4. Simulation with two viruses: one (highlighted in red) staying endemic, and one (highlighted in blue) approaching eradication. The average infection rate of virus k in the network is denoted by $\bar{p}^k(t)$.

the recovery rate of virus 2. With these settings in place, we obtain $s(B_w^1 - D_w^1) = 2.9$, and $s(B_w^2 - D_w^2) = -0.3$. With reference to Figure 4, it can be seen that virus 2 is eradicated exponentially fast, consistent with the result in Theorem 5. In contrast, it can be seen that virus 1 appears to approach a non-zero equilibrium, thereby not only validating Theorem 8, but also suggesting that the result in Theorem 8 can be extended to the multi-virus setting. Moreover, changing the initial state of virus 1 ($y^1(0)$) did not cause the equilibrium to change, suggesting that the aforementioned non-zero equilibrium could be unique. With respect to Figure 2, Figure 4 says that since one of the viruses persists, the population in the above-mentioned districts of Stockholm and the SL system remain infected/contaminated.

5. CONCLUSION

The present paper introduced a SIS model accounting for multiple competing viruses in the presence of a shared contaminative resource. We provided conditions under which a virus is eradicated exponentially fast (resp. asymptotically). Subsequently, for the single-virus case, we provided conditions under which the virus remains endemic in the population and the resource. Finally, we established a necessary and sufficient condition for the healthy state to be the unique equilibrium.

REFERENCES

Anderson, R.M. and Robert, M. (1991). May. infectious diseases of humans: dynamics and control. *Oxford Science Publications*, 36, 118.

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*, 1–45.

CC (2016). `https://commons.wikimedia.org/wiki/File:Sweden_Stockholm_City_location_map.svg`. Mikey641 and OpenStreetMap contributors / CC BY-SA `https://creativecommons.org/licenses/by-sa/2.0` Accessed: 2020-24-06.

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.

Funk, S. and Jansen, V.A. (2010). Interacting epidemics on overlay networks. *Physical Review E*, 81(3), 036118.

Khalil, H. (2002). *Nonlinear Systems*. Pearson Education. Prentice Hall.

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.

Liu, J., Paré, P.E., Du, E., and Sun, Z. (2019a). A networked SIS disease dynamics model with a waterborne pathogen. In *2019 American Control Conference (ACC)*, 2735–2740. IEEE.

Liu, J., Paré, P.E., Nedich, A., Tang, C.Y., Beck, C.L., and Basar, T. (2019b). Analysis and control of a continuous-time bi-virus model. *IEEE Transactions on Automatic Control*.

Nowak, M. (1991). The evolution of viruses. competition between horizontal and vertical transmission of mobile genes. *Journal of theoretical biology*, 150(3), 339–347.

Paré, P.E., Vrabac, D., Sandberg, H., and Johansson, K.H. (2020). Analysis, online estimation, and validation of a competing virus model. In *American Control Conference*.

Paré, P.E., Liu, J., Beck, C.L., Nedić, A., and Başar, T. (2017). Multi-competitive viruses over static and time-varying networks. In *2017 American Control Conference (ACC)*, 1685–1690. IEEE.

Rantzer, A. (2011). Distributed control of positive systems. In *Proceedings of the 50th IEEE Conference on Decision and Control and European Control Conference*, 6608–6611.

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

Watkins, N.J., Nowzari, C., Preciado, V.M., and Pappas, G.J. (2016). Optimal resource allocation for competitive spreading processes on bilayer networks. *IEEE Trans. Control of Network Systems*, 5(1), 298–307.

Wei, X., Valler, N.C., Prakash, B.A., Neamtiu, I., Faloutsos, M., and Faloutsos, C. (2013). Competing memes propagation on networks: A network science perspective. *IEEE J. Selected Areas in Communications*, 31(6), 1049–1060.

APPENDIX

Proof of Theorem 5: We need the following proposition to prove the claim in Theorem 5.

Proposition 10. Let $\mathbb{G} \subset \mathbb{R}^n$. Consider

$$\dot{x}(t) = f(t, x), \tag{6}$$

where $f : [0, \infty) \times \mathbb{G} \to \mathbb{R}^n$ is a locally Lipschitz map. Let $x^* = \mathbf{0}$ be an equilibrium of (6) and $\mathbb{E} \subset \mathbb{G}$ be a positively invariant and connected set with respect to (6), containing x^*. Let $V : [0, \infty) \times \mathbb{E} \to \mathbb{R}$ be a continuously differentiable function such that:

$$k_1\|x\|^a \leq V(t, x) \leq k_2\|x\|^a,$$
$$\dot{V}(t, x) \leq -k_3\|x\|^a,$$

$\forall t > 0$ and $\forall x \in \mathbb{E}$, where k_1, k_2, k_3 and a are positive constants. Then the equilibrium x^* is exponentially stable with a domain of attraction containing $\mathbb{E}$. ■

This proposition can be proven using (Khalil, 2002, Theorem 4.10) and the discussion on pages 122, 317-320 in (Khalil, 2002).

Proof of Theorem 5: By Lemma 4 we know that S is positively invariant with respect to (5), and since $y(0) \in S$ we have $y(t) \in S$ for all $t \geq 0$. Given that $(B_w^k - D_w^k)$ is Metzler, (Rantzer, 2011, Proposition 2) states that there exists a positive diagonal matrix Q^k such that $((B_w^k - D_w^k)^T Q^k + Q^k(B_w^k - D_w^k))$ is negative definite. Define the Lyapunov function candidate $V(y^k(t)) = y^k(t)^T Q^k y^k(t)$ with S^k as the domain. This function is positive definite, so, by the Rayleigh-Ritz Theorem, there exist constants $a = \min_i(Q^k)_{ii} > 0$, $b = \max_i(Q^k)_{ii} > 0$ such that:

$$a\|y^k(t)\|^2 \leq V(y^k(t)) \leq b\|y^k(t)\|^2. \tag{7}$$

Differentiating $V(y^k(t))$ with respect to time yields:

$$\begin{aligned}\dot{V}(y^k(t)) &= 2y^k(t)^T Q^k \dot{y}^k(t)\\ &= 2y^k(t)^T Q^k \Big(B_w^k - D_w^k - X\big(y(t)\big)B_w^k\Big)y^k(t)\\ &\leq 2y^k(t)^T Q^k \big(B_w^k - D_w^k\big)y^k(t)\\ &= y^k(t)^T \big((B_w^k - D_w^k)^T Q^k + Q^k(B_w^k - D_w^k)\big)y^k(t).\end{aligned}$$

Note that $y^k(t)^T Q^k X\big(y(t)\big)B_w^k y^k(t) \geq 0$ since the matrices in this product are non-negative while $y(t) \in S$. Since $((B_w^k - D_w^k)^T Q^k + Q^k(B_w^k - D_w^k))$ is negative definite, by the Rayleigh-Ritz theorem there exists a constant $c = s\big((B_w^k - D_w^k)^T Q^k + Q^k(B_w^k - D_w^k)\big) < 0$ such that:

$$\dot{V}(y^k(t)) \leq c\|y^k(t)\|^2. \tag{8}$$

Since by Lemma 4, S^k is a positively invariant set with respect to (4), from (7) and (8), it follows that $V(y^k(t))$ fulfills the requirements of Proposition 10. Therefore, the eradicated state of virus k is exponentially stable with a domain of attraction containing S^k. □

Proof of Theorem 6: Note that if $s(B_w^k - D_w^k) < 0$, Theorem 5 implies exponential stability of the eradicated state with a domain of attraction including S^k, in turn implying asymptotic stability with the same domain of attraction. Because of this the rest of the proof assumes that $s(B_w^k - D_w^k) = 0$. By Lemma 4 we know that S is positively invariant with respect to (5), and since $y(0) \in S$ we have $y(t) \in S$ for all $t \geq 0$. Then the trajectories of $y^k(t)$ are bounded above by the trajectories $\bar{y}(t)$ of a similar single-virus system with the same parameters and positively invariant set S^k. Since $s(B_w^k - D_w^k) = 0$ implies $\rho((D_w^k)^{-1}B_w^k) = 1$ by Lemma 7, the requirements for (Liu et al., 2019a, Theorem 1) are fulfilled by extension for the single-virus system, meaning that its healthy state is asymptotically stable with a domain of attraction containing S^k. Given that $\mathbf{0} \leq y^k(t) \leq \bar{y}(t)$, the eradicated state for virus k is asymptotically stable with a domain of attraction containing S^k. □

Proof of Theorem 8: Define a map $T(x) : \mathbb{R}_+^{n+1} \to \mathbb{R}_+^{n+1}$ such that:

$$\begin{aligned}T(x) = &(I + \operatorname{diag}(D_w^{-1}B_w x))^{-1}\\ &\times (D_w^{-1}B_w x + \operatorname{diag}(D_w^{-1}B_w x)[\mathbf{0}\, x_{n+1}]^T).\end{aligned}$$

Since $\operatorname{diag}(D_w^{-1}B_w x)$ is a non-negative diagonal matrix, the inverse of $(I+\operatorname{diag}(D_w^{-1}B_w x))$ exists, and, hence, $T(x)$ is well-defined. Note that the components of $T(x)$ are:

$$T_i(x) = \frac{(D_w^{-1}B_w x)_i}{1 + (D_w^{-1}B_w x)_i}, \text{ for } i \in [n],$$

$$T_{n+1}(x) = \frac{(D_w^{-1}B_w x)_{n+1}x_{n+1} + (D_w^{-1}B_w x)_{n+1}}{1 + (D_w^{-1}B_w x)_{n+1}}.$$

Given that $D_w^{-1}B_w$ is a non-negative matrix, $y \geq z$ implies that $T(y) \geq T(z)$. A fixed point of $T(x)$ is any point $x \in \mathbb{R}_+^{n+1}$ such that:

$$\begin{aligned}x = &(I + \operatorname{diag}(D_w^{-1}B_w x))^{-1}\\ &\times (D_w^{-1}B_w x + \operatorname{diag}(D_w^{-1}B_w x)[\mathbf{0}; x_{n+1}]).\end{aligned} \tag{9}$$

Multiplying (9) by $(I + \operatorname{diag}(D_w^{-1}B_w x))$ gives:

$$D_w^{-1}B_w x + \operatorname{diag}(D_w^{-1}B_w x)[\mathbf{0}; x_{n+1}] = (I + \operatorname{diag}(D_w^{-1}B_w x))x. \tag{10}$$

Using the identity $\operatorname{diag}(u)v = \operatorname{diag}(v)u$ we see that (10) is equivalent to:

$$D_w^{-1}B_w x + \operatorname{diag}([\mathbf{0}; x_{n+1}])D_w^{-1}B_w x = (I + \operatorname{diag}(x)D_w^{-1}B_w)x. \tag{11}$$

For a given $x \in \mathbb{R}_+^{n+1}$, define $X(x)$ to be its diagonalization with the final element x_{n+1} set to zero. As such, by subtracting $\operatorname{diag}([\mathbf{0}x_{n+1}]^T)D_w^{-1}B_w x$ from (11), we obtain:

$$D_w^{-1}B_w x = (I + X(x)D_w^{-1}B_w)x. \tag{12}$$

Since $X(x)$ and D_w^{-1} are diagonal matrices, they commute. Furthermore, by pre-multiplying (12) with D_w, and suitably rearranging terms, we obtain:

$$(B_w - D_w - X(x)B_w)x = 0. \tag{13}$$

A solution of equation (13) is clearly an equilibrium of (4) with $m = 1$. As such it suffices to show that $T(x)$ has a non-zero fixed point in S. We will now show that at least one such fixed point exists. Since $s(B_w - D_w) > 0$, by Lemma 7, $\rho(D_w^{-1}B_w) > 1$. Further, given that B_w is an irreducible non-negative matrix and D_w^{-1} is a positive diagonal matrix, $D_w^{-1}B_w$ is an irreducible non-negative matrix. Hence, by the Perron-Frobenius theorem, $\lambda^* = \rho(D_w^{-1}B_w)$ is a simple eigenvalue of $D_w^{-1}B_w$, with an eigenspace spanned by a vector $x^* \gg \mathbf{0}$. Thus, there exists some constant $\epsilon > 0$ such that, for all $i \in [n+1]$, we have $\epsilon x_i^* \leq (\lambda^* - 1)/\lambda^*$, which implies that $1 \leq \lambda^*/(1 + \lambda^* \epsilon x_i^*)$. Hence, $\epsilon x_i^* \leq \lambda^* \epsilon x_i^*/(1 + \lambda^* \epsilon x_i^*)$, which further implies:

$$\epsilon x_i^* \leq \frac{(D_w^{-1}B_w \epsilon x^*)_i}{1 + (D_w^{-1}B_w \epsilon x^*)_i}. \tag{14}$$

Noting that $(D_w^{-1}B_w \epsilon x^*)_{n+1}\epsilon x_{n+1}^* > 0$, we have:

$$\epsilon x_{n+1}^* \leq \frac{(D_w^{-1}B_w \epsilon x^*)_{n+1}\epsilon x_{n+1}^* + (D_w^{-1}B_w \epsilon x^*)_{n+1}}{1 + (D_w^{-1}B_w \epsilon x^*)_{n+1}}. \tag{15}$$

Due to the inequalities (14) and (15), we have $T(\epsilon x^*) \geq \epsilon x^*$. Since $y \geq z$ implies $T(y) \geq T(z)$, it follows that for any $x \geq \epsilon x^*$ we have $T(x) \geq \epsilon x^*$. Consider $T(\mathbf{1})$ for $i \in [n]$,

$$T_i(\mathbf{1}) = \frac{(D_w^{-1}B_w \mathbf{1})_i}{1 + (D_w^{-1}B_w \mathbf{1})_i} \leq 1. \tag{16}$$

For $i = n + 1$, we have:

$$T_{n+1}(\mathbf{1}) = \frac{2[c\ 0]\mathbf{1}}{1 + [c\ 0]\mathbf{1}} \leq 1. \tag{17}$$

Due to the inequalities (16) and (17), we have $T(\mathbf{1}) \leq \mathbf{1}$. Since $y \geq z$ implies that $T(y) \geq T(z)$ it follows that $T(x) \leq \mathbf{1}$ if $x \leq \mathbf{1}$. Applying Brouwer's fixed-point theorem, there is at least one fixed point $\tilde{x}$ of $T(x)$ such that $\epsilon x^* \leq \tilde{x} \leq \mathbf{1}$. Since $\tilde{x}$ is also a solution of equation (13), there is at least one non-zero equilibrium for (4) in S. □