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Competitive Networked Bivirus SIS spread over Hypergraphs

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Abstract— **The paper deals with the spread of two competing viruses over a network of population nodes, accounting for pairwise interactions and higher-order interactions (HOI) within and between the population nodes. We study the competitive networked bivirus susceptible-infected-susceptible (SIS) model on a hypergraph introduced in Cui et al. [1]. We show that the system has, in a generic sense, a finite number of equilibria, and the Jacobian associated with each equilibrium point is nonsingular; the key tool is the Parametric Transversality Theorem of differential topology. Since the system is also monotone, it turns out that the typical behavior of the system is convergence to some equilibrium point. Thereafter, we exhibit a tri-stable domain with three locally exponentially stable equilibria. For different parameter regimes, we establish conditions for the existence of a coexistence equilibrium (both viruses infect separate fractions of each population node).**

I. Introduction

The study of virus spread has been an active area of research for over two centuries. In particular, diverse scientific communities, such as physics [2], mathematics [3], computer science [4], automatic control [5], economics [6] , etc., have significantly aided in furthering our understanding of the complex mechanisms behind the spread of a virus. Fundamental to this effort has been the development of compartmental models where each individual is healthy and susceptible (S), infected with a virus (I), or has recovered from a viral infection (R). Two compartmental models, susceptible-infected-recovered (SIR) and susceptible-infected-susceptible (SIS) have garnered significant attention in several scientific disciplines, particularly in mathematical epidemiology. In contrast to the SIR model, the SIS model allows for the possibility of reinfection and is the focus of the present paper. More specifically, we will deal with networked SIS models, with each node in the network being representative of a large population, and the interconnection among the nodes denotes the possible spreading pathways for the virus. Networked SIS models have been studied in, among others, [7]–[9].

The existing literature on modeling virus spread typically relies on the assumption that there is just a single virus present. However, one often encounters scenarios where there are two viruses, say virus 1 and virus 2, circulating in a meta-population (i.e., a network of population nodes). In such a context, possibility is for the two viruses to compete; infection with virus 1 (resp. virus 2) precludes the possibility of simultaneous infection with virus 2 (resp. virus 1) - this is the focus of the present paper. We stress that the notion of competing viruses is not restricted to just epidemics; it manifests itself in, among others, product adoption in a marketplace and the spread of opinions in social networks [10].

Networked competitive multi-virus SIS models have been analyzed in substantial depth in recent times; see [3], [11]–[18]. A major drawback of networked competitive bivirus SIS models studied in the aforementioned papers is that they account only for pairwise interactions between individuals. In reality, interactions in social groups often involve more than two individuals - it is not unusual that an individual can *simultaneously* interact with more than one other individual. This motivates the need for higher-order networks such as hypergraphs [1], i.e., graphs where an edge can connect more than two nodes, which are quite effective in representing higher-order interactions (HOI) [20]. Inspired by the approach in [21], an SIS model on a hypergraph has been proposed and analyzed in [22]. However, the analytic results therein relied on certain restrictions on the network structure. Overcoming this drawback, a networked SIS model on a hypergraph has been devised and studied in considerable detail in [23]. However, the modeling frameworks in [21]–[23] are restrictive in the sense that none of these account for the possibility of more than one virus simultaneously circulating in a given population. Addressing this shortcoming, a competitive networked bivirus SIS model on a hypergraph has been developed and analyzed in [1]. The set of equilibria for the model in [1] can be broadly classified into three categories: the disease-free equilibrium (DFE) (both viruses have been eradicated), the boundary equilibria (one virus is dead, and the other is alive); and coexistence equilibria (two viruses infect separate fractions of every population node in the network). Nevertheless, the results in [1] have the following limitations: a) some of the findings therein have yet to be rigorously established, and b) the analysis, while improving our understanding of the existence and stability of various equilibria, is not exhaustive. The present paper aims to address the aforementioned gaps. Our contributions are as follows:

i) We show that the networked bivirus SIS system with

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[1] Simplicial networks (see [19] for more details) have also been used for studying HOI, see [20].

HOI has, in a generic sense, a finite number of equilibria. Furthermore, for each equilibrium, the associated Jacobian is a nonsingular matrix; see Theorem 1. In so doing, since our proof of Theorem 1 does not, unlike the proof of [1, Theorem 5.5], require the HOI infection rates to be set to zero, we establish the correctness of the claim raised in [1, Theorem 5.5]. Building off of Theorem 1 and leveraging the fact that the system is monotone as identified in [1, Theorem 5.5], we prove that the typical behavior of the bivirus SIS system with HOI is convergence to an equilibrium point; see Theorem 2.

ii) We identify a parameter regime that not only establishes the existence of three equilibria (a single-virus endemic equilibrium corresponding to virus 1 (resp. virus 2) and the DFE) but also guarantees that all of the said equilibria are locally exponentially stable at the same time; see Proposition 1.

iii) We identify a parameter regime, different from the one covered by Proposition 1, for the existence of a coexistence equilibrium. We do so under different configurations of the boundary equilibria, viz. both being unstable and both being stable; see Proposition 3 and Theorem 3, respectively.

Additionally, for the parameter regime covered by Proposition 1, we establish existence of a coexistence equilibrium; see Proposition 4.

Notation: We denote the set of real numbers by $\mathbb{R}$ and the set of nonnegative real numbers by $\mathbb{R}_+$. For any positive integer n, we use $[n]$ to denote the set $\{1, 2, ..., n\}$. 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. For a vector x, we denote the diagonal square matrix with x along the diagonal by $\mathrm{diag}(x)$. For any two 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 $i \in [n]$. Likewise, for any two matrices $A, B \in \mathbb{R}^{n\times m}$, we write $A \geq B$ if $A_{ij} \geq B_{ij}$ for all $i \in [n]$, $j \in [m]$, and $A > B$ if $A \geq B$ and $A \neq B$. For a square matrix M, we use $\sigma(M)$ to denote the spectrum of M, $\rho(M)$ to denote the spectral radius of M, and $s(M)$ to denote the spectral abscissa of M, i.e., $s(M) = \max\{\mathrm{Re}(\lambda) : \lambda \in \sigma(M)\}$.

A real square matrix A is called Metzler if all its off-diagonal entries are nonnegative. A matrix A is said to be an M-matrix if all of its off-diagonal entries are nonpositive, and there exists a constant $c > 0$ such that, for some nonnegative B and $c \geq \rho(B)$, $A = cI - B$. All eigenvalues of an M-matrix have nonnegative real parts. Furthermore, if an M-matrix has an eigenvalue at the origin, we say it is singular; if each eigenvalue has strictly positive parts, then we say it is nonsingular. If $A(= [a_{ij}]_{n\times n})$ is a nonnegative matrix, then $\rho(A)$ decreases monotonically with a decrease in a_{ij} for any $i, j \in [n]$. The matrix A is reducible if, and only if, there is a permutation matrix P such that $P^\top AP$ is block upper triangular; otherwise, A is said to be irreducible. If a nonnegative A is irreducible, and $Ax = y$ for $x > \mathbf{0}$, then $y > \mathbf{0}$, and y cannot have a zero in every position where x has a zero.

II. Problem Formulation

A. Model

Consider a network of n nodes. A node represents a well-mixed[2] population of individuals. We will assume that the size of the population is fixed. We suppose two viruses, say virus 1 and virus 2, are spreading over such a network. Throughout this paper, we will assume that the two aforementioned viruses are competing. Through pairwise or HOI as described in more detail below, an otherwise healthy individual in node i gets infected with virus 1 (resp. virus 2) due to contact with either other individuals in node i who are infected with virus 1 (resp. virus 2) and/or with other individuals in node j (where j is a neighbor of i) who are infected with virus 1 (resp. virus 2). When a single interaction is involved (i.e., between two individuals in node i or between an individual in node i and an individual in node j), we say that the infection is caused due to *pairwise interactions*. An individual in node i could also be infected with virus 1 (resp. virus 2) due to *simultaneous* interactions with infected individuals in nodes j and ℓ, where either a) $j = i$, and/or $\ell = i$, or b) j, ℓ are neighbors of i. Such interactions are referred to as *higher-order interactions* (HOI). The notion of competition implies that no individual can be simultaneously infected with virus 1 and virus 2.

We assume that the pairwise infection (resp. HOI) rate with respect to virus k is the same for all nodes, denoted by β_1^k (resp. β_2^k) for all $i \in [n]$ and $k \in [2]$ [3]. An individual infected with virus k recovers from said infection at a healing rate δ_i^k and immediately becomes susceptible to virus 1 or virus 2. All individuals within a node have the same healing rate with respect to virus k; individuals in different nodes possibly have different healing rates. We say that node i is healthy if all individuals in node i are healthy; otherwise, we say it is infected. Within the same node, it is possible for there to simultaneously exist a fraction of individuals that are infected with virus 1 and for a different fraction that is infected with virus 2.

As mentioned previously, diseases could spread due to pairwise interactions and HOI. In case of the former, if an individual in node j can infect an individual in node i with virus k, then, with $a_{ij}^k (\geq 0)$ denoting the strength of interactions between an individual in node j and an individual in node i with respect to the spread of virus k, we have that $a_{ij}^k > 0$; otherwise $a_{ij}^k = 0$. For the case of HOI, if an individual in node i gets infected with virus k due to simultaneous interactions with individuals in nodes j and ℓ (both of whom are infected), then, with $b_{ij\ell}^k$ denoting the strength of interaction that nodes j and ℓ together have

[2]Well-mixed means that the probability of any two individuals in a node interacting with each other is the same.

[3]Indeed, it is far more natural to have possibly different infection rates for each node; it is standard in the literature on classic SIS bivirus networked systems [11]–[16], [24]. As evident below, we do not impose constraints on the values of the nonnegative matrices capturing the interactions, and hence the analysis does not differ materially. We choose this particular notation to remain consistent with earlier literature on epidemic models with HOI [23].

on node i with respect to the spread of virus k, we have that $b^k_{ij\ell} > 0$; else, $b^k_{ij\ell} = 0$. HOIs in SIS models are typically represented by directed hypergraphs [1], [23]. Specifically, we define a hypergraph G with 2 layers. The vertex set for each layer k, where $k = 1, 2$, is the set of population nodes. In layer k, there is a hyperedge (resp. edge) from nodes j and ℓ to node i (resp. from node j to node i) if, and only if, $b^k_{ij\ell} > 0$ (resp. $a^k_{ij} > 0$). Let $x^k_i(t)$ denote the fraction of individuals infected with virus k in agent i at time instant t. The evolution of this fraction can, therefore, be represented by the following scalar differential equation [1, Section 5], where, for $i = 1, 2, \ldots, n$, we have

$$\begin{aligned}\dot{x}^1_i =& -\delta^1_i x^1_i + \beta^1_1(1 - x^1_i - x^2_i)\textstyle\sum_{j=1}^n a^1_{ij}x^1_j + \\ & \beta^1_2(1 - x^1_i - x^2_i)\textstyle\sum_{j,\ell=1}^n b^1_{ij\ell}x^1_j x^1_\ell \\ \dot{x}^2_i =& -\delta^2_i x^2_i + \beta^1_1(1 - x^1_i - x^2_i)\textstyle\sum_{j=1}^n a^2_{ij}x^2_j + \\ & \beta^2_2(1 - x^1_i - x^2_i)\textstyle\sum_{j,\ell=1}^n b^2_{ij\ell}x^2_j x^2_\ell \end{aligned} \tag{1}$$

Define $D^1 = \operatorname{diag}(\delta^1_i)$, where $i \in [n]$, and define D^2 analogously. Define $X^1 = \operatorname{diag}(x^1_i)$, where $i \in [n]$, and define X^2 analogously. Let $A^1 = [a^1_{ij}]_{n\times n}$, and $A^2 = [a^2_{ij}]_{n\times n}$. Let $B^k_i = [b^k_{ij\ell}]_{n\times n}$, for each $i \in [n]$ and $k \in [2]$. Let $x^k = [x^k_1 \quad x^k_2 \quad \ldots \quad x^k_n]^\top$ for $k = 1, 2$.

Therefore, in vector form, equation (1) can be written as:

$$\begin{aligned}\dot{x}^1 =& -D^1x^1 + \beta^1_1(I - X^1 - X^2)A^1x^1 + \\ & \beta^1_2(I - X^1 - X^2)((x^1)^\top B^1_1 x^1, (x^1)^\top B^1_2 x^1, \ldots, (x^1)^\top B^1_n x^1)^\top \\ \dot{x}^2 =& -D^2x^2 + \beta^2_1(I - X^1 - X^2)A^2x^2 + \\ & \beta^2_2(I - X^1 - X^2)((x^2)^\top B^2_1 x^2, (x^2)^\top B^2_2 x^2, \ldots, (x^2)^\top B^2_n x^2)^\top \end{aligned} \tag{2}$$

Note that $(x^\top B_1 x, x^\top B_2 x, \ldots, x^\top B_n x)^\top = \begin{bmatrix} x^\top B_1 x \\ x^\top B_2 x \\ \vdots \\ x^\top B_n x \end{bmatrix}$.

We note that system (2) is a special case of [1, system 5.5] in the following sense: System (2) only accounts for a) the case where, for $k = 1, 2$, β^k_i and β^k_2 is identical for every node i, $i = 1, 2, \ldots, n$, and b) the case where virus 1 (resp. virus 2) spread only due to contact with the infected individuals. In contrast, the model in [1] (see [1, system 5.5]) allows for the possibility of β^k_i and β^k_2 being not necessarily the same for every node. Furthermore, it also allows for the possibility of the viruses to spread through additional mediums such as a water distribution network, a public transit network, etc.

Remark 1: Setting $x^1(0) = \mathbf{0}$ (resp. $x^2(0) = \mathbf{0}$) results in system (2) coinciding with the model used for studying the spread of a single virus over hypergraphs in [23].

The model in system (2) has three kinds of equilibria, viz. healthy state or disease-free equilibrium (DFE), $(\mathbf{0}, \mathbf{0})$; single-virus endemic equilibria corresponding to virus k, of the form $(\bar{x}^k, \mathbf{0})$, where $\mathbf{0} \ll \bar{x}^k \ll \mathbf{1}$ for $k = 1, 2$; and coexisting equilibria, $(\bar{x}^1, \bar{x}^2)$, where, as we will show in Lemma 1, $\mathbf{0} \ll \bar{x}^1, \bar{x}^2 \ll \mathbf{1}$, and, furthermore, $\bar{x}^1 + \bar{x}^2 \ll \mathbf{1}$. It is unknown whether the single-virus endemic equilibria corresponding to virus k are unique, in contrast to the classic bivirus SIS network model without HOI.

The Jacobian of system (2) evaluated at an arbitrary point, (x^1, x^2), in the state space is as given in (3).

$$J(x^1, x^2) = \begin{bmatrix} J_{11} & J_{22} \\ J_{21} & J_{22} \end{bmatrix}, \tag{3}$$

where

$$\begin{aligned} J_{11} =& -D^1 + \beta^1_1(I - X^1 - X^2)A^1 - \operatorname{diag}(\beta^1_1 A^1 x^1) + \\ & \beta^1_2(I - X^1 - X^2)O_1(x^1) - \beta^1_2 O_2(x^1) \end{aligned} \tag{4}$$

$$J_{12} = -\operatorname{diag}(\beta^1_1 A^1 x^1) - \beta^1_2 \operatorname{diag}((x^1)^\top B^1_i x^1)_{i=1,2,\ldots,n} \tag{5}$$

$$J_{21} = -\operatorname{diag}(\beta^2_1 A^2 x^2) - \beta^2_2 \operatorname{diag}((x^2)^\top B^2_i x^2)_{i=1,2,\ldots,n} \tag{6}$$

$$\begin{aligned} J_{22} =& -D^2 + \beta^2_1(I - X^1 - X^2)A^2 - \operatorname{diag}(\beta^2_1 A^2 x^2) + \\ & \beta^2_2(I - X^1 - X^2)O_3(x^2) - \beta^2_2 O_4(x^2) \end{aligned} \tag{7}$$

The terms $O_1(x^1)$, $O_2(x^1)$, $O_3(x^2)$ and $O_4(x^2)$ are as given in (8), (9), (10) and (11), respectively.

We will need the following assumptions to ensure the model is well-defined in the sense that the states take values that correspond to physical reality in the epidemiological context.

Assumption 1: The matrix D^k, for $k = 1, 2$, is a positive diagonal matrix. The matrix A^k, for $k = 1, 2$, is nonnegative. The matrix B^k_i is nonnegative for all $i \in [n]$ and $k \in [2]$.

Observe that for classic bivirus systems, some works have also considered the case where D^k is a nonnegative matrix; see, for instance, [13].

Assumption 2: The matrix A^k, for $k = 1, 2$, is irreducible.

Assumption 2 guarantees that the graph representing the pairwise interactions is strongly connected.

We define the set $\mathcal{D}$ as follows:

$$\mathcal{D} := \{(x^1, x^2) \mid x^k \geq \mathbf{0}, k = 1, 2, \textstyle\sum_{k=1}^2 x^k \leq \mathbf{1}\}. \tag{12}$$

It is known that the set $\mathcal{D}$ is positively invariant, and that the DFE is always an equilibrium for system (2); see [1, Lemma 5.1]. The fact that $\mathcal{D}$ is positively invariant guarantees that the state values $x^k_i, k \in [2], i \in [n]$, always stay in the $[0, 1]$ interval. Since the states represent fractions of an infected population node, if the states were to take values outside the $[0, 1]$ interval, then those would not correspond to physical reality.

B. Problem Statements

With respect to system (2), we aim to answer the following questions in this paper

i) What is the typical behavior the trajectories exhibit as time goes to infinity?
ii) Can we identify a parameter regime such that multiple equilibria are simultaneously stable?
iii) Can we identify sufficient conditions for the existence of a coexistence equilibrium? Furthermore, can we establish the stability properties of such an equilibrium based on knowledge of the stability properties of the boundary equilibria?

C. Preliminary Lemmas and analysis of healthy state

In this subsection, we establish a result on the nature of equilibria of system (1).

Lemma 1: Consider system (2) under Assumptions 1 and 2. If $\bar{x} = (\bar{x}^1, \bar{x}^2) \in \mathcal{D}$ is an equilibrium of (2), then, for each $k \in [2]$, either $\bar{x}^k = \mathbf{0}$, or $\mathbf{0} \ll \bar{x}^k \ll \mathbf{1}$. Moreover, $\sum_{k=1}^2 \bar{x}^k \ll \mathbf{1}$.

$$O_1(x^1) = \big[(B_1^1 + (B_1^1)^\top)x^1 \qquad (B_2^1 + (B_2^1)^\top)x^1 \qquad \dots \qquad (B_n^1 + (B_n^1)^\top)x^1\big] \tag{8}$$

$$O_2(x^1) = \operatorname{diag}((x^1)^\top B_i^1 x^1)_{i=1,2,\dots,n} \tag{9}$$

$$O_3(x^2) = \big[(B_1^2 + (B_1^2)^\top)x^2 \qquad (B_2^2 + (B_2^2)^\top)x^2 \qquad \dots \qquad (B_n^2 + (B_n^2)^\top)x^2\big] \tag{10}$$

$$O_4(x^2) = \operatorname{diag}((x^2)^\top B_i^2 x^2)_{i=1,2,\dots,n} \tag{11}$$

Proof: See proof of [25, Lemma 1].

It can be seen that $(\mathbf{0}, \mathbf{0})$ is an equilibrium of (2), and is referred to as the disease-free equilibrium (DFE). Sufficient conditions for convergence to the DFE have been provided in [1].

III. Monotone dynamical systems and competitive bivirus networked SIS models with HOI

Monotone dynamical systems (MDS) are a class of systems that has found resonance in mathematical epidemiology; one of the major reasons for this is the fact that MDS, assuming that they generically have a finite number of equilibria, converge to a (stable) equilibrium point for almost all initial conditions. Here, the term "almost all" means: for all but a set of parameter values that has measure zero. An algebraic or semi-algebraic set defines this set of exceptional values. It is known that under Assumptions 1 and 2, system (2) is monotone; see [1, Theorem 5.5]. That is, suppose that $(x_A^1(0), x_A^2(0))$ and $(x_B^1(0), x_B^2(0))$ are two initial conditions in int($\mathcal{D}$) satisfying i) $x_A^1(0) > x_B^1(0)$ and ii) $x_A^2(0) < x_B^2(0)$. Since system (2) is monotone, it follows that, for all $t \in \mathbb{R}_{\geq 0}$, i) $x_A^1(t) \gg x_B^1(t)$ and ii) $x_A^2(t) \ll x_B^2(t)$. Note that [1, Theorem 5.5] also claims that system (2), in a generic sense, has a finite number of equilibria. However, the proof for said assertion is the proof for the case where $\beta_2^k = 0$, for $k = 1, 2$, in [14, Theorem 3.6], which is only a nongeneric case. Since the proof for finiteness of equilibria in [1, Theorem 5.5] is not complete, it leaves open the issue of generic convergence to an equilibrium point. To remedy this, we provide a different proof for generic finiteness of equilibria that does not rely on $\beta_2^k = 0$ for $k = 1, 2$.

Given that nonlinear systems can have complex equilibria patterns, including a continuum of equilibria for the classic bivirus network model, we establish that for generic parameter matrices, system (1) has a finite number of equilibria. We use arguments very much like those in [15]. Essentially because the healthy equilibrium and the single-virus boundary equilibria can be conveniently studied using single-virus techniques, it is easily established that there are no continua of equilibria confined to any boundary, i.e. any continuum of equilibria necessarily includes a continuum of coexistence equilibria. Therefore, we focus on showing that such equilibria cannot exist for generic parameter values. The tool is the Parametric Transversality Theorem, see [26, see p. 145]. The main result is as follows:

Theorem 1: Consider the model of (2), under Assumptions 1 and 2. With any fixed matrices A^k and nonnegative B_i^k, and the exclusion of a set of values for the entries of D^1, D^2 of measure zero, the number of coexistence equilibrium points is finite, and the associated vector field zero is nondegenerate, i.e. the associated Jacobian is nonsingular. Similarly, with any fixed D^1, D^2 and B_i^k, and the exclusion of a set of values for the entries of A^1, A^2 of measure zero, the same properties of equilibrium points hold.

Proof: See proof of [25, Theorem 1]. □

Theorem 1, coupled with the fact that system (2) is monotone, allows us to leverage Hirsch's generic convergence theorem [27] to draw conclusions on the limiting behavior of system (2) outside of the specific conditions identified in [1, Theorem 5.2, statement 1]. We have the following result.

Theorem 2: Consider system (2) under Assumptions 1 and 2. For all initial conditions $(x^1(0), x^2(0)) \in \mathcal{D}$ except possibly for a set of measure zero, the system (2) will converge to an equilibrium. If the system does not converge to an equilibrium, it is on a nonattractive limit cycle.

In words, Theorem 2 establishes that the typical behavior of system (2) is convergence to *some* equilibrium; this could be healthy, or (one of the possibly many) single-virus boundary equilibria, or a coexistence equilibrium. It further says that limit cycles, if any, are nonattractive. No more complicated behavior is allowed; chaos can be ruled out, see [28]. Thus, Theorem 2 answers question i) raised in Section II.

IV. Existence and local stability of boundary equilibria

In this section, we identify a parameter regime that permits three equilibria of the bivirus system (2) to be simultaneously locally exponentially stable. Subsequently, for a parameter regime different from the one mentioned above, we identify a condition for the existence and instability of a boundary equilibrium. Finally, when there is only one virus, we identify a condition for the existence and local exponential stability of an endemic equilibrium.

Proposition 1: Consider system (2) under Assumptions 1 and 2. Define, for $k = 1, 2$, $\mathbf{1}_{B^k} \in \{0, 1\}^n$ by $(\mathbf{1}_{B^k})_i = 1$ if $B_i^k \neq \mathbf{0}$; otherwise $(\mathbf{1}_{B^k})_i = 0$. Suppose that the following conditions are fulfilled for $k = 1, 2$:

a) $\rho(\beta_1^k (D^k)^{-1} A^k) < 1$, and

b) $\min_{i.s.t. B_i^k \neq \mathbf{0}} \big(\frac{\beta_1^k}{\delta_i^k} (A^k \mathbf{1}_{B^k})_i + \frac{\beta_2^k}{2\delta_i^k} \mathbf{1}_{B^k}^\top B_i \mathbf{1}_{B^k} \big) > 2.$

Then, the following statements are true:

i) The DFE is locally exponentially stable.

ii) there exist equilibria $\bar{x}^k \gg \mathbf{0}$ such that $\bar{x}_i^k \geq \frac{1}{2}$ for $k = 1, 2$, for any i such that $B_i^k \neq \mathbf{0}$.

iii) Any such equilibrium point $(\bar{x}^1, \mathbf{0})$ is locally exponentially stable; and

iv) Any such equilibrium point $(\mathbf{0}, \bar{x}^2)$ is locally exponentially stable.

Proof: See proof of [25, Proposition 1].

Proposition 1 answers question ii) raised in Section II. Proposition 1 guarantees the existence and simultaneous local exponential stability of three equilibria, whereas [1, Theorem 5.3], assuming that an endemic equilibrium exists, guarantees its local stability. Furthermore, the possibility of the DFE being locally stable simultaneously is alluded to; see [1, Remark 10]. On the other hand, for a parameter regime different from the one covered in Proposition 1, assuming that the terms corresponding to HOI are sufficiently small, [1, Theorem 5.3] secures global stability of the endemic equilibrium. Notice also that an exact characterization of the region og attraction of locally stable equilibria, $(\bar{x}^1, \mathbf{0})$ and $(\mathbf{0}, \bar{x}^2)$, is not available. The following remarks are in order.

Remark 2: Proposition 1 sheds light on an interesting phenomenon that bivirus spread over hypergraph admits (but bivirus spread over a normal graph does not exhibit): the possibility of the presence of a parameter regime that permits three equilibria, namely the DFE and the two boundary equilibria, to be simultaneously stable. This is an extension to the single virus case studied in [23], which permitted the simultaneous stability of the DFE and (since there is only one in this case) an endemic equilibrium.

Remark 3: It is known that, assuming $\beta_2^k = 0$ for $k = 1, 2$, the condition $\rho(\beta_1^k (D^k)^{-1} A^k) \leq 1$ guarantees that the DFE is the only equilibrium of system (2); see [24, Lemma 2]. However, as Proposition 1 shows, that is not necessarily true when considering bivirus SIS spread over hypergraphs.

Proposition 1 guarantees existence of boundary equilibria for the case when $\rho(\beta_1^k (D^k)^{-1} A^k) < 1$, for $k = 1, 2$. It is natural to ask if one is assured of existence even if the spectral radii of relevant quantities are larger than one. Specifically, if $\rho(\beta_1^k (D^k)^{-1} A^k) > 1$ for some $k \in [2]$, and if , for some $\ell \in [2]$ such that $\ell \neq k$, $\rho(\beta_1^\ell (D^\ell)^{-1} A^\ell) < 1$, then, since the dynamics of virus ℓ converges to the DFE, system (2) effectively has the dynamics of a single-virus system in the long run. Therefore, in addition to the DFE, there exists a boundary equilibrium of the form $(\bar{x}^k, \mathbf{0})$; see [23, Theorem 5.2, statement vii)]. The following proposition addresses this issue.

Proposition 2: Consider system (2) under Assumptions 1 and 2. Suppose that, for all $k \in [2]$, $\rho(\beta_1^k (D^k)^{-1} A^k) > 1$. Then system (2) has at least three equilibria, namely the DFE, a single virus endemic equilibrium corresponding to virus 1 $(\bar{x}^1, \mathbf{0})$, and a single virus endemic equilibrium corresponding to virus 2 $(\mathbf{0}, \bar{x}^2)$. Furthermore, if $s(-D^i + \beta_1^i (I - \bar{X}^k) A^i) > 0$ for $i, k \in [2]$ such that $i \neq k$, then the equilibrium points $(\bar{x}^1, \mathbf{0})$ and $(\mathbf{0}, \bar{x}^2)$ are unstable.

Proof: See proof of [25, Proposition 2]. □

Remark 4: Proposition 2 (resp. Proposition 1) guarantees the existence (resp. existence and local exponential stability) of the equilibrium points, $(\bar{x}^1, \mathbf{0})$ and $(\mathbf{0}, \bar{x}^2)$. It turns out that it is possible to compute these points iteratively; see [23, Theorem 5.3].

V. (Non)existence of Coexistence equilibria

This section identifies sufficient conditions for the existence (resp. nonexistence) of coexistence equilibria. Specifically, for investigating existence, we consider two parameter regimes, viz. for $k = 1, 2$, i) $s(-D^k + \beta_i^k A^k) > 0$, and ii) $s(-D^k + \beta_i^k A^k) < 0$. Further, for the parameter regime i), we consider two stability configurations of the boundary equilibria, viz. a) both being unstable and b) both being stable; for parameter regime ii), we consider the case where both boundary equilibria are stable.

Proposition 3: Consider system (2) under Assumptions 1 and 2. Let $(\bar{x}^1, \mathbf{0})$ and $(\mathbf{0}, \bar{x}^2)$ denote a single-virus endemic equilibrium corresponding to virus 1 and virus 2, respectively. Suppose that the following conditions are satisfied:

i) $s(-D^1 + \beta_1^1 A^1) > 0$;
ii) $s(-D^2 + \beta_1^2 A^2) > 0$;
iii) $s(-D^1 + \beta_1^1 (I - \bar{X}^2) A^1) > 0$; and
iv) $s(-D^2 + \beta_1^2 (I - \bar{X}^1) A^2) > 0$.

Then there exists at least one equilibrium of the form $(\hat{x}^1, \hat{x}^2)$ such that $\mathbf{0} \ll \hat{x}^1, \hat{x}^2 \ll \mathbf{1}$ and $\hat{x}^1 + \hat{x}^2 \ll \mathbf{1}$.

Proof: See proof of [25, Proposition 3].

Proposition 3 is implied by [1, Theorem 5.4], which, assuming $\beta_2^k = 0$ for $k = 1, 2$, is the same as [12, Theorem 5]. The proof technique in [1, Theorem 5.4] is quite involved since it primarily relies on fixed point mapping, Perron Frobenius theory, etc. Our proof is significantly shorter. Note that [29, Theorem 2] is a key ingredient of our proof strategy. In light of Theorem 1, one could perhaps leverage [29, Theorem 2] to obtain a lower bound on the number of coexistence equilibria for the stability configuration of boundary equilibria given in Proposition 3, as has been done for classic bivirus networked SIS models; see [15, Corollary 3.9, statement 2]. Subsequently, one could possibly exploit the properties of MDS to conclude that there must exist a locally exponentially stable coexistence equilibrium.

Observe that in Propostion 3 the demonstration of the existence of a coexistence equilibrium point $(\hat{x}^1, \hat{x}^2)$ relies on the assumption that both boundary equilibria are unstable. We now present a different condition that also guarantees the existence of a coexistence equilibrium point $(\hat{x}^1, \hat{x}^2)$ even when both boundary equilibria are stable.

Theorem 3: Consider system (2) under Assumptions 1 and 2. Let $(\bar{x}^1, \mathbf{0})$ and $(\mathbf{0}, \bar{x}^2)$ denote a single-virus endemic equilibrium corresponding to virus 1 and virus 2, respectively. Suppose that the following conditions are satisfied:

i) $s(-D^1 + \beta_1^1 A^1) > 0$;
ii) $s(-D^2 + \beta_1^2 A^2) > 0$;

Suppose that both $(\bar{x}^1, \mathbf{0})$ and $(\mathbf{0}, \bar{x}^2)$ are locally exponentially stable. Then there exists at least one equilibrium of the form $(\hat{x}^1, \hat{x}^2)$ such that $\mathbf{0} \ll \hat{x}^1, \hat{x}^2 \ll \mathbf{1}$ and $\hat{x}^1 + \hat{x}^2 \ll \mathbf{1}$, such that $(\hat{x}^1, \hat{x}^2)$ is either neutrally stable or unstable.

Proof: See proof of [25, Theorem 3]. □

Proposition 3 and Theorem 3 partially answer question iii) raised in Section II-B. Observe that neither of these results

covers the case where one boundary equilibrium is locally exponentially stable, and the other is unstable.

We next consider a different parameter regime, namely $s(-D^k + \beta_1^k A^k) < 0$. We have the following result.

Proposition 4: Consider system (2) under Assumptions 1 and 2. Define, for $k = 1, 2$, $\mathbf{1}_{B^k} \in \{0,1\}^n$ by $(\mathbf{1}_{B^k})_i = 1$ if $B_i^k \neq \mathbf{0}$; otherwise $(\mathbf{1}_{B^k})_i = 0$. Suppose that the following conditions are fulfilled for $k = 1, 2$:

a) $\rho(\beta_1^k (D^k)^{-1} A^k) < 1$, and

b) $\min_{i.s.t. B_i^k \neq \mathbf{0}} \left(\frac{\beta_1^k}{\delta_i^k} (A^k \mathbf{1}_{B^k})_i + \frac{\beta_2^k}{2\delta_i^k} \mathbf{1}_{B^k}^\top B_i \mathbf{1}_{B^k} \right) > 2.$

Then there exists at least one equilibrium of the form $(\hat{x}^1, \hat{x}^2)$ such that $\mathbf{0} \ll \hat{x}^1, \hat{x}^2 \ll \mathbf{1}$ and $\hat{x}^1 + \hat{x}^2 \ll \mathbf{1}$ that is either neutrally stable or unstable.

Proof: See proof of [25, Proposition 4]. □

Note that Proposition 4 guarantees the existence of at least one coexistence equilibrium. Given that system (2) is monotone, and since, from Theorem 1, it is known that for each of the equilibrium points the associated Jacobian is nonsingular, the conditions in Proposition 4 guarantee the existence of an odd number of coexistence equilibria, each of which must be unstable. The proof for the same follows from a Brouwer degree argument; see [30]. In fact, for the special case where $\beta_2^k = 0$ for $k = 1, 2$, for the same stability configuration as in Theorem 3 and Proposition 4, a lower bound on the number of coexistence equilibria has been recently provided; see [15, Corollary 3.9, statement 3].

VI. Numerical Examples

We present a series of simulations highlighting interesting phenomena that can emerge when HOIs are incorporated. We use the following bivirus system with HOIs. The network has $n = 5$ nodes, and we set $D^1 = D^2 = I$. The pairwise interactions are captured by two-cycle graphs with self-loops, with infection matrices:

$$A^1 = \begin{bmatrix} 1 & 0 & 0 & 0 & 1 \\ 1 & 1 & 0 & 0 & 0 \\ 0 & 1 & 1 & 0 & 0 \\ 0 & 0 & 1 & 1 & 0 \\ 0 & 0 & 0 & 1 & 1 \end{bmatrix}, \qquad A^2 = (A^1)^\top. \tag{13}$$

The HOI are captured by the following set of hyperedges with unit weight:

$$\text{virus } 1 : (1,2,3), (2,3,1), (3,2,1), (1,4,5), (4,5,1), (5,4,1)$$
$$\text{virus } 2 : (1,2,4), (2,4,1), (4,2,1), (1,3,5), (3,5,1), (5,3,1).$$

In other words, this corresponds to the following $b_{ij\ell}^k$ entries being equal to 1, with all other entries of B_i^k equal to 0: b_{123}^1, b_{231}^1, b_{321}^1, b_{145}^1, b_{451}^1, b_{541}^1, and b_{124}^2, b_{241}^2, b_{421}^2, b_{135}^2, b_{351}^2, b_{531}^2. In our simulations, we randomly sample $x_i^k(0)$ from a uniform distribution $(0,1)$, and then normalize the vectors $x^1(0)$ and $x^2(0)$ to ensure that $(x^1(0), x^2(0)) \in \text{int}(\mathcal{D})$. The β_i^k are varied to yield different stability properties for the system in (2).

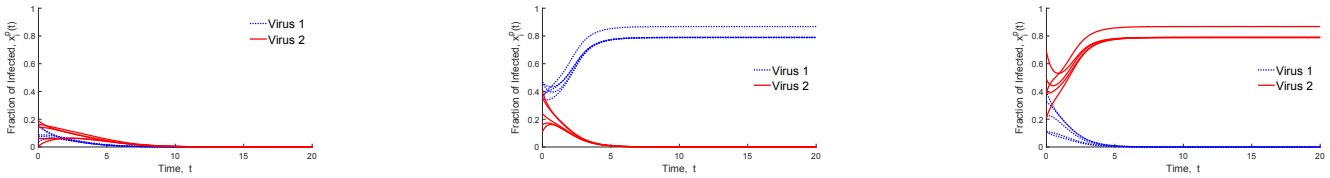
Example 1: We set $\beta_1^1 = \beta_2^1 = 0.2$ and $\beta_1^2 = \beta_2^2 = 5$. This ensures the inequalities of both conditions for Proposition 1 are satisfied. As can be observed from Fig. 1a, for initial conditions close to the DFE, the trajectories converge to the locally exponentially stable DFE, $(x^1 = \mathbf{0}, x^2 = \mathbf{0})$. In Figs. 1b and 1c, the initial conditions are further in the interior of $\mathcal{D}$, and depending on the particular initial condition, we observe convergence to a boundary equilibrium where one of the two viruses is extinct, $(\bar{x}^1, \mathbf{0})$ or $(\mathbf{0}, \bar{x}^2)$ for some positive $\bar{x}^1 > 0.5 \times \mathbf{1}$ and $\bar{x}^2 > 0.5 \times \mathbf{1}$. That is, both boundary equilibria are simultaneously locally exponentially stable. Interestingly, without HOIs, it is impossible for a bivirus system to have the DFE, $(\bar{x}^1, \mathbf{0})$, and $(\mathbf{0}, \bar{x}^2)$ all locally exponentially stable [31, Section E].

Example 2: We set $\beta_1^1 = \beta_2^1 = 2$ and $\beta_1^2 = 3$ and $\beta_2^2 = 2.4$. As illustrated in Figs. 2a and 2b, there are two locally exponentially stable two boundary equilibria $(\bar{x}^1, \mathbf{0})$ or $(\mathbf{0}, \bar{x}^2)$, and we converge to either depending on the initial conditions. However, the DFE is unstable, and no trajectories in $\mathcal{D}$ converge there except if one starts at the DFE. This simulation highlights are interesting observation: for a standard bivirus system with no HOIs, examples of systems with two locally stable boundary equilibria have not been identified until recently and are not straightforward to construct [15], [32].

VII. Conclusion

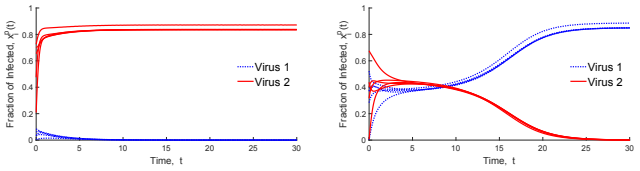
This paper analyzed a networked competitive bivirus SIS model that also accounts for the possibility of HOI among the nodes. By taking recourse to the Parametric Transversality Theorem of differential topology, we showed that the bivirus system with HOI has, for generic parameter values, a finite number of equilibria. Furthermore, the Jacobian matrices associated with each of the equilibria are nonsingular. This finding, coupled with the knowledge that the system is monotone, enabled us to establish that the typical behavior that our system exhibits is convergence to some equilibrium. Subsequently, we identified a parameter regime that ensures the existence of multiple boundary equilibria and simultaneous stability of the same along with that of the DFE. For the special case where only one virus is circulating in the metapopulation, we guarantee the existence and local stability of an endemic equilibrium; our result does not impose any restrictions on the model parameters besides those covered by Assumptions 1 and 2. Thereafter, for different parameter regimes, we identified conditions that guarantee the existence of a coexistence equilibrium.

There are multiple research lines that merit further investigation. One such problem would be identifying conditions that guarantee not just existence but also uniqueness of a coexistence equilibrium. Yet another problem would be investigating nongeneric scenarios that could give rise to a continuum of coexistence equilibria. Other matters we are developing include relaxing of key assumptions, and considering time-scale adjustments. Finally, while the analysis in the present paper relies on time-invariant hypergraphs, it may be interesting to see how, for instance, the conditions for convergence to DFE would alter (if at all) when one allows for the hypergraphs to be possibly time-varying. Moving away from equlibrium analysis, a problem of interest would be to study the transient dynamics of the model presented here.



(a) (b) (c)

Fig. 1: Trajectories of the system (2) set out in *Example 1*, for different initial conditions in panels (a), (b) and (c).



(a) Example 2 (b) Example 2

Fig. 2: Trajectories of the system (2) set out in *Example 2*, for different initial conditions in panel (a) and (b).

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