On the Endemic Behavior of a Competitive Tri-Virus SIS Networked Model

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Abstract—We study the endemic behavior of a multi-competitive networked susceptible-infected-susceptible (SIS) model. In particular, we focus on the case where there are three competing viruses (i.e., tri-virus system). First, we show that the tri-virus system is not monotone. Thereafter, we identify necessary conditions and a sufficient condition for local exponential convergence to a boundary equilibrium (exactly one virus is alive, and the other two are dead) and identify a special case that admits the existence and local exponential attractivity of a line of coexisting equilibria (at least two viruses are active). Finally, we identify a particular case (subsumed by the aforementioned special case) such that for all nonzero initial infection levels, the dynamics of the tri-virus system converge to a plane of coexisting equilibria.

I. INTRODUCTION

The study of spreading epidemic processes has been an active area of research for several decades. It has spanned multiple scientific disciplines such as physics [1], mathematics [2], economics [3], and computer science [4]. The central theme involving all such research directions is to derive a foundational understanding of what causes a disease to spread and to design effective mitigation (or eradication) strategies. Towards this end, various models have been proposed in the literature. This paper deals with the susceptible-infected-susceptible (SIS) model. More specifically, we are interested in networked SIS models, where each node in the network represents a large population and interconnections between nodes capture the possibility of the virus spreading between populations [5]–[8].

The vast majority of the literature on (networked) SIS models concerns the presence of a single virus. However, one often encounters several virus strains that might be circulating simultaneously in a community, as observed during the ongoing COVID-19 crisis. In a scenario where multiple viruses are present, these viruses could possibly be competing with each other. That is, assuming there are \( m > 1 \) viruses present, an individual (belonging to a population node) can be infected with at most one virus at any given time. More precisely, being infected with one virus precludes the possibility of being simultaneously infected with any of the other \( m - 1 \) viruses. This is commonly observed during the spread of multiple strains of a virus [9], [10]. Other settings where such a competing phenomenon is observed include, but are not limited to, the spread of competing opinions on different social networks, competing products in a market, and the spread of conflicting rumors [11]. This motivates the need for multi-competitive networked SIS models. The dynamics exhibited in the multi-virus setting are far richer than those in the single-virus setting, which complicates the analysis [12]–[15]. The case where \( m = 2 \) (also referred to as competitive bivirus spread) has been relatively well-explored in recent times; see [13]–[19]. However, settings accounting for the presence of more than two competing viruses have not been well studied. The paper [15] (also see [20] for the discrete-time version) proposes a multi-competitive networked SIS model, where \( m \) (with \( m \) being arbitrary but finite) viruses are simultaneously active. However, the analysis of the endemic behavior in [15], [20] is rather restrictive, in the sense that most of the results in [20] pertain to the existence of boundary equilibria whereas [15] focuses on certain special cases that give rise to a continuum of coexistence equilibria. Thus, to our knowledge, a comprehensive analysis accounting for \( m \) competing viruses, where \( m \) is some arbitrary but finite positive integer, is not available in the existing literature. Therefore, as a first step, in this direction, the present paper focuses on the special case when \( m = 3 \), i.e., the tri-virus competitive networked SIS model. The set of equilibria of such a system can be broadly classified into three categories: the disease-free equilibrium (all three viruses have been eradicated); the boundary equilibrium (two viruses are dead, and one is alive); and coexisting equilibria (at least two viruses infect separate fractions of every population node in the network).

Our contributions are as follows:

i) We show that the tri-virus system, unlike the bi-virus system, is not monotone; see Theorem 1. Consequently, one cannot leverage the rich literature on monotone dynamical systems (see [21], [22]), to study the limiting behavior of our model.

ii) We identify necessary conditions and a sufficient condition for local exponential convergence to a boundary equilibrium; see Theorem 2.

iii) We identify a special case that admits the existence of a line of coexisting equilibria (which, under certain conditions, is locally exponential attractive); see Theorem 3.

iv) For a special case, subsumed by that in iii), we provide a sufficient condition that ensures that, regardless of the initial non-zero infection levels, the dynamics of the tri-virus system converge to a plane of coexisting equilibria; see Theorem 4.

This paper is organized as follows. The model, background, and problem statements are detailed in Section II. We
show that the tri-virus system is not monotone in Section III, whereas Section IV deals with the case where at least one virus persists. Coexisting equilibria for nongeneric systems are illustrated in Section V. The theoretical findings are provided in Section VII.

Notations: We denote the set of (nonnegative) real numbers by $\mathbb{R}_+ \times \mathbb{R}$. For any positive integer $k$, we use $[k]$ to denote the set $\{1, 2, \ldots, k\}$. The $i$th entry of a vector $x$ is denoted by $x_i$. The element in the $i$th row and $j$th column of a matrix $M$ is denoted by $M_{ij}$. We use $0$ and $I$ to denote the vectors whose entries all equal 0 and 1, respectively, and $I$ denotes the identity matrix. For a vector $x$, we denote the square matrix with $x$ along the diagonal by $\text{diag}(x)$. For any two real vectors $a, b \in \mathbb{R}^n$ we write $a \geq b$ if $a_i \geq b_i$ for all $i \in [n]$ and $b \neq 0$. Likewise, for any two real 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 $A \neq B$.

We use $\sigma^\text{red}(M)$ to denote the spectrum of a square matrix $M$, $\rho(M)$ to denote its spectral radius, and $s(M)$ to denote its spectral abscissa, i.e., $s(M) = \max\{\text{Re}(\lambda) : \lambda \in \sigma(M)\}$. A square matrix $A$ is said to be Hurwitz if $s(A) < 0$. A real square matrix $A$ is said to be Metzler if all its off-diagonal entries are nonnegative, and it is said to be an $M$-matrix if all of its off-diagonal entries are nonpositive, and all its eigenvalues have nonnegative real parts. If an $M$-matrix has an eigenvalue at the origin, then we say that it is singular; if each of its eigenvalues has strictly positive parts, then we say that it is nonsingular. The matrix $A$ is positive semidefinite if $x^T Ax \geq 0$ for all vectors $x$, and we denote this by $A \succeq 0$.

### II. Problem Formulation

In this section, we detail a model that captures the spread of multiple competing viruses across a population network.

#### A. Model

Consider a network of $n \geq 2$ nodes, where $m$ viruses compete with each other to infect the nodes (SIS models with $n = 1$ have been studied in [23, Sec. 2]). The notion of competition implies the presence of at least two (but possibly more) viruses. Throughout this paper, $m = 3$. In context, each node represents a well-mixed population of individuals with large and constant sizes. A well-mixed population means any two individuals in the population can interact with the same positive probability. This model assumes homogeneity within the population node and (possible) heterogeneity outside the population node, i.e., all individuals within a population node have the same infection (resp. healing) rates, but individuals in different population nodes need not necessarily have the same healing (resp. infection) rate [5].

Individuals in a population can be partitioned into four mutually exclusive health compartments: susceptible, infected with virus 1, infected with virus 2, or infected with virus 3. No individual can be simultaneously infected by more than one virus. A population node is healthy if all individuals belong to the susceptible compartment; otherwise, we say it is infected. An individual, belonging to population $i$ (where $i = 1, 2, \ldots, n$), in the susceptible compartment, can transition to the “infected with virus $k$” (for $k \in [m]$) compartment at a rate $\beta_{ki}$. An individual in population $i$ that is infected with virus $k$ recovers from it based on said individual’s healing rate with respect to virus $k$, i.e., $\delta_{ki}$.

The spread of $m$-competing viruses can be modeled using an $m$-layer graph $G$, where the vertices of the graph represent the population nodes. The $k$th layer denotes the contact graph for the spread of virus $k$, for each $k \in [m]$. For the graph $G$, there exists a directed edge from node $j$ to node $i$ in layer $k$ if, assuming an individual in population $j$ is infected with virus $k$, then said individual can infect at least one (but possibly more) healthy individual in node $i$. Let $E^k$ denote the edge set corresponding to the $k$th layer of $G$. We denote by $A^k$ (where $a_{ij} \geq 0$) the weighted adjacency matrix corresponding to layer $k$, with the elements in $A^k$ being in one-to-one correspondence with the existence (or lack thereof) of edges in layer $k$, i.e., $(i, j) \in E^k$ if, and only if, $a_{ij} \neq 0$. Let $x^k(t)$ denote the fraction of individuals infected with virus $k$ in agent $i$ at time instant $t$. The evolution of this fraction can, then, be represented by the following scalar differential equation [15, Eq. 4]:

$$\dot{x}^k(t) = -\delta_{ki}^k x^k(t) + (1 - \sum_{l=1}^{m} x^l(t)) \sum_{j=1}^{n} \beta_{ij}^k x^j(t),$$

where $\beta_{ij}^k = \beta_{ik}^k$. Define $x^k(t) = [x^k_1(t), \ldots, x^k_n(t)]^T$, $D^k = \text{diag}(\delta_{ki}^k)$, and $B^k = [\beta_{ij}^k]_{n \times n}$. Therefore, (1) can be written as

$$\dot{x}^k(t) = (-D^k + (1 - \sum_{l=1}^{m} \text{diag}(x^l(t)))) B^k x^k(t).$$

Defining $x(t) := [x^1(t), \ldots, x^m(t)]^T$, and $R^k(x(t)) := (-D^k + (1 - \sum_{l=1}^{m} \text{diag}(x^l(t)))) B^k$, the dynamics of the system of all $m$ viruses are given by

$$\dot{x}(t) = \begin{bmatrix} R^1(x(t)) & 0 & \ldots & 0 \\ 0 & R^2(x(t)) & \ldots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \ldots & R^m(x(t)) \end{bmatrix} x(t).$$

Note that by setting $m = 1$ in (3), one recovers the classic single-virus SIS model that has been studied extensively in the literature; see [1], [5], [7], [24]. Setting $m = 2$ yields the classic networked bi-virus SIS model, for which a plethora of results have been provided in [12]–[14], [16], [17]. In this paper, we are interested in the case when $m = 3$, i.e., the tri-virus networked competitive spread.

Define, for $k \in [3]$, $X^k = \text{diag}(x^k)$. Based on (2), the dynamics of the tri-virus system can be written as follows:

$$\dot{x}_1(t) = ((I - (X^1 + X^2 + X^3)) B^1 - D^1)x_1(t),$$

$$\dot{x}_2(t) = ((I - (X^1 + X^2 + X^3)) B^2 - D^2)x_2(t),$$

$$\dot{x}_3(t) = ((I - (X^1 + X^2 + X^3)) B^3 - D^3)x_3(t).$$

#### B. Assumptions and Preliminary lemmas

We need the following assumption to ensure that the aforementioned model is well-defined.

**Assumption 1:** Suppose that $\delta_{ki}^k > 0, \beta_{ij}^k \geq 0$ for all $i, j \in [n]$ and $k \in [3]$. 

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Assumption 2: The matrix $B^k$, for $k \in [3]$ is irreducible.

Observe that under Assumption 1, for all $k \in [3]$, $B^k$ is a nonnegative matrix and $D^k$ is a positive diagonal matrix. Moreover, recall that a square nonnegative matrix $M$ has the irreducibility property if, supposing $M$ is the (un)weighted adjacency matrix of a graph, the corresponding graph is strongly connected. Then, noting that non-zero elements in $B^k$ represent directed edges in the set $E^k$, we see that $B^k$ is irreducible whenever the $k^{th}$ layer of the multi-layer network $G$ is strongly connected.

Thanks to Assumption 1, we can restrict our analysis to the sets $D := \{x(t) : x_k(t) \in [0,1]^n, \forall k \in [3], \sum_{k=1}^{3} x^k \leq 1 \}$ and $D^k := \{x^k(t) \in [0,1]^n\}$. Since $x^k(t)$ is to be interpreted as a fraction of a population, these sets represent the sensible domain of the system. That is, if $x^k(t)$ takes values outside of $D^k$, then those values would lack physical meaning. The following lemma shows that $x(t)$ never leaves the set $D$.

Lemma 1: [15, Lemma 1] Let Assumption 1 hold. Then $D$ is positively invariant with respect to (3).

Lemma 2: [18, Lemma 7] Let Assumption 1 hold. Then $D \setminus \{0\}$ is positively invariant with respect to system (4)-(6).

Let $J(x^1, x^2, x^3)$ denote the Jacobian matrix of system (4)-(6) for an arbitrary point in the state space. Therefore, $J(x^1, x^2, x^3)$ is as given in (7).

The point, $\bar{0} = (0,0,0)$ is an equilibrium of (4)-(6), and it is referred to as the disease-free equilibrium (DFE). A sufficient condition for global stability of the DFE is as follows:

Proposition 1: [15, Lemma 2] Consider system (4)-(6) under Assumption 1. If $s(-D^k + B^k) \leq 0$, for each $k \in [3]$, then the DFE is asymptotically stable, with domain of attraction containing $D$.

As it turns out, if the inequalities are strict, i.e., if $s(-D^k + B^k) < 0$, for each $k \in [3]$, then the DFE is actually exponentially stable, with $D$ in the domain of attraction, see [18, Theorem 1].

It turns out that for every eigenvalue condition in Proposition 1 that is violated, one obtains an equilibrium of the form $(\bar{x}, \bar{x}, \ldots, \bar{x})$ in $D$, where $\bar{x}$ is the single-virus endemic equilibrium corresponding to virus $k$. This is formalized in the following proposition.

Proposition 2: [6, Theorem 2.1] Consider system (4)-(6) under Assumptions 1 and 2. For each $k \in [3]$, there exists a unique single-virus endemic equilibrium $(\bar{x}, \ldots, \bar{x}, \ldots, \bar{x})$ in $D$, with $\bar{x} \ll \bar{x} \ll 1$ if, and only if, $s(B^k - D^k) > 0$.

Analytic methods for computing the single-virus endemic equilibria have been provided in [1, Theorem 5].

The equilibria of the form $(\bar{x}, \ldots, \bar{x}, \ldots, \bar{x})$ are referred to as the boundary equilibria. The equilibria of the form $(\bar{x}, \bar{x}, \bar{x})$, where at least $\bar{x}$ and $\bar{x}$ $(i, j \in [3], i \neq j)$ are nonnegative vectors with at least one positive entry in each of $\bar{x}$ and $\bar{x}$ are referred to as coexistence equilibria. It turns out that such vectors are strictly positive; see [18, Lemma 6].

C. Monotone dynamical systems and competitive bivirus networked SIS models

Observe that for the case when $m = 2$, system (3) is, under Assumption 2, monotone [17, Lemma 3.3] (and, assuming homogeneous recovery rates, also in [16, Theorem 18]). That is, setting $m = 2$ for system (3), suppose that $(x_A^0(0), x_A^0(0))$ and $(x_B^0(0), x_B^0(0))$ are two initial conditions in $int(D)$ satisfying i) $x_A^1(0) > x_B^1(0)$ and ii) $x_A^2(0) < x_B^2(0)$. Since the bivirus system is monotone, it follows that, for all $t$, i) $x_A^1(t) > x_B^1(t)$ and ii) $x_A^2(t) < x_B^2(t)$. Further, it has been shown that, for almost all choices of $D^0$, $B^i$, $i = 1, 2$, system (3) has a finite number of equilibria [17, Theorem 3.6]. Therefore, from [22, Theorems 2.5 and 2.6], we know that for almost all initial conditions in $D$, system (3) with $m = 2$ converges to a stable equilibrium point. The set of initial conditions for which said convergence does not occur (in which case it is either already at an unstable equilibrium, assuming such an equilibrium exists, or in the stable manifold of an unstable equilibrium, or on a nonattractive limit cycle, or in the stable manifold of a nonattractive limit cycle) has measure zero. Leveraging the fact that the bivirus system is monotone, conditions guaranteeing the existence of, and local convergence to, a finite set of coexistence equilibria have been recently provided in [25]. It is unknown if the notion of monotone dynamical systems applies for the case when $m = 3$. Consequently, understanding the limiting behavior of tri-virus systems remains partly open.

Note that, for the case when $m = 2$, a sufficient condition for local exponential convergence to a boundary equilibrium has been identified in [17, Theorem 3.10], whereas for the $m = 3$ case no such condition has been identified. Further, certain special (nongeneric) scenarios have been identified which lead to the existence of a continuum of coexistence equilibria; see [14, Theorems 6 and 7]. Improving upon these results, a broader scenario (but again nongeneric) that accounts for a larger class of parameters has been identified, which leads to not only the existence but also local exponential attractivity, of a continuum of coexistence equilibria; see [17, Proposition 3.9]. Analogous results for the $m = 3$ case are as yet unavailable.

D. Problem Statements

Based on the above discussions, our objective in the present paper is to answer the following questions:

i) Is the tri-virus system monotone?

ii) Identify a necessary condition and a sufficient condition for local exponential convergence to a boundary equilibrium?

iii) Identify sufficient conditions for the existence and local attractivity of a continuum of coexisting equilibria?

iv) What are special case(s) where, irrespective of the non-zero initial infection levels, the tri-virus dynamics converge to a continuum of coexisting equilibria?

III. IS THE TRI-VIRUS SYSTEM MONOTONE?

In this section, we seek to conclusively answer whether (or not) system (3) with $m = 3$ is monotone.

In order to answer this question, we construct a graph associated with the Jacobian of system (4)-(6), say $G$. The construction follows the outline provided in [26]. More

1Almost all means that for all but a set of parameter values that has measure zero and it is defined by an algebraic or semi-algebraic set.
specifically, the graph $G$ has $3n$ nodes. The edges of $G$ are based on the entries in the Jacobian matrix $J(x^1, x^2, x^3)$. Specifically, if $[J(x^1, x^2, x^3)]_{ij} \leq 0$ for $i \neq j$, then we draw an edge labelled with “−” sign; if $[J(x^1, x^2, x^3)]_{ij} \geq 0$ for $i \neq j$, then we draw an edge labelled with “+” sign. Thus, $G$ is a signed graph. Note that $G$ has no self-loops. As an aside, also observe that since $x^k(t) \geq 0$ for $k \in [3]$ and $t \in \mathbb{R}^+$, it is immediate that the sign of the elements in $J(x^1, x^2, x^3)$ does not change with the argument, so $G$ is the same for all points in the interior of $D$.

We also need the following concepts from graph theory. A signed graph is said to be consistent if every undirected cycle in the graph has a net positive sign, i.e., it has an even number of “−” signs [26]. We have the following result.

**Theorem 1:** System (4)-(6) is not monotone.

**Proof:** The Jacobian $J(x^1, x^2, x^3)$ is a block matrix, with all blocks along the off-diagonal being negative diagonal matrices. Pick any node $i$, where $i \in \{1, 2, \ldots, n\}$. Since all blocks along the off-diagonal of $J(x^1, x^2, x^3)$ are negative diagonal matrices, there exists an edge from node $j$ to node $i$ or node $j \in n$, an edge from node $i \in n$ to node $j$, and an edge from node $i \in 2n$. Each of these edges have a “−” sign. Hence, a loop starting from node $i$, traversing through nodes $i \in n$, $i \in 2n$, and back to node $i$ is a 3-length cycle that has an odd number of negative signs. Therefore, the signed graph $G$ is not consistent [26, page 62]. It follows that the system (4)-(6) is not monotone [26, page 63].

The tri-virus system not being monotone is in sharp contrast to the bi-virus setting, which is known to be monotone [17]. The fact that a bivirus system is monotone coupled with the fact that for almost all choices of $D^k$, $B^k$, $k = 1, 2$, the bivirus system has a finite number of equilibria allows one to draw general conclusions on the limiting behavior of bivirus dynamical systems. By extending the algebraic geometry arguments in the proof of [17, Theorem 3.6], it follows that even for the tri-virus system, for almost all choices of $D^k$, $B^k$, $k = 1, 2, 3$, there exists a finite number of equilibria. The details are omitted here in the interest of space. Nonetheless, due to the findings of Theorem 1, one cannot draw upon the rich literature on monotone dynamical systems (see [22]) to study the limiting behavior of system (4)-(6). In general, for non-monotone systems, no dynamical behavior, including chaos, can be ruled out [26].

Another possible consequence of the lack of monotonicity is as follows: It is known that setting $D^k = I$ for $k \in [2]$ has no bearing on either the location of equilibria of system (3) with $m = 2$ nor on their (local) stability properties [17, Lemma 3.7]. That is, for a) bivirus systems, where $D^k = I$ and $B^k = (D^k)^{-1}B^k$ for $k \in [2]$, and b) bivirus systems, where $D^k$s are arbitrary positive diagonal matrices, the location of equilibria are the same for both bivirus systems a) and b). Plus, local stability of an equilibrium in bivirus system a) implies, and is implied by, that in bivirus system b). For system (3) with $m = 3$, by extending the arguments from [17, Lemma 3.7], it is straightforward to show that the location of the equilibria is the same when, for $k \in [3]$, $D^k = I$ and $B^k = (D^k)^{-1}B^k$, and when $D^k$ (resp. $B^k$) are arbitrary positive diagonal (resp. nonnegative) matrices with the $D^k$s not necessarily being equal to each other. However, since the tri-virus system is not monotone, the arguments for stability of equilibria used in [17, Lemma 3.7] cannot be adapted. As such, for the tri-virus case, preservation of stability properties remains an open question when the healing rates for all nodes with respect to all viruses are ‘scaled’ in the manner above to become unity.

**IV. PERSISTENCE OF ONE OR MORE VIRUSES**

If one or more of the eigenvalue conditions in Proposition 1 is violated, then at least one of the viruses persists in the population. This, in turn, gives rise to a richer possible set of behaviors, as we will see in the rest of this paper.

This section identifies necessary conditions and a sufficient condition for local exponential convergence to a boundary equilibrium. While similar results exist for the case when $m = 2$ (see [17, Theorem 3.10]), to the best of our knowledge, no such result exists for the $m = 3$ case. The following theorem addresses this gap and establishes that the local stability (resp. instability) of a boundary equilibrium corresponding to virus $i$ is dependent on whether (or not) the state matrix corresponding to viruses $j$ and $k$, linearized around the single-virus endemic equilibrium of virus $i$, is Hurwitz.

**Theorem 2:** Consider system (4)-(6) under Assumptions 1 and 2. The boundary equilibrium $(\tilde{x}^1, 0, 0)$ is locally exponentially stable if, and only if, each of the following conditions are satisfied:

i) $\rho((I - \tilde{X}^1)(D^2)^{-1}B^2) < 1$; and

ii) $\rho((I - \tilde{X}^1)(D^3)^{-1}B^3) < 1$.

If $\rho((I - \tilde{X}^1)(D^2)^{-1}B^2) > 1$ or $\rho((I - \tilde{X}^1)(D^3)^{-1}B^3) > 1$, then $(\tilde{x}^1, 0, 0)$ is unstable.

The proof follows the strategy outlined for the $m = 2$ case in [17, Theorem 3.10].

**Proof:** Please see proof of [27, Theorem 2].

Analogous results for the boundary equilibria $(0, \tilde{x}^2, 0)$ and $(0, 0, \tilde{x}^3)$ can be similarly obtained.

**V. COEXISTENCE EQUILIBRIA FOR NONGENERIC TRI-VIRUS NETWORKS: EXISTENCE AND ATTRACTIVITY**

**A. Existence and attractivity of a continuum of equilibria**

Proposition 2 and Theorem 2, deal with the existence and local exponential convergence to an equilibrium where one, and only one, virus is alive; the rest have become extinct. Here, we are interested in identifying a scenario that guarantees the existence and local exponential attractivity of a continuum of coexisting equilibria (i.e., at least two, but
possibly all three, viruses exist in some sort of balance with each other), when all three viruses persist in a population. We assume $D^k = I, k = 1, 2, 3$, for ease of describing the phenomenon of a continuum of coexistence equilibria.

Let $z$ denote the single-virus endemic equilibrium corresponding to virus 1, with $Z = \text{diag}(z)$. Therefore, since $D^1 = I$, the vector $z$ fulfills the following:

$$-I + ((I - Z)B^3)z = 0. \quad (8)$$

Furthermore, since $z$ is an endemic equilibrium, from [18, Lemma 6] it follows that $0 \ll z \ll 1$. Let $C$ be any nonnegative irreducible matrix for which $z$ is also an eigenvector corresponding to eigenvalue one. That is, $Cz = z$. Therefore, from [28, Theorem 2.7], it follows that $\rho(C) = 1$, and that the vector $z$, up to a scaling, is the unique eigenvector of $C$ with all entries being strictly positive. Define

$$B^2 := (I - Z)^{-1}C. \quad (9)$$

We have the following result.

**Theorem 3:** Consider system (4)-(6) under Assumption 1. Suppose that $D^k = I$ for $k \in [3]$. Suppose that $B^1$ and $B^3$ are arbitrary nonnegative irreducible matrices; and vector $z$ and matrix $B^2$ are as defined in (8) and (9), respectively. Then, a set of equilibrium points of the trivirus equations is given by $(\beta_1z, (1 - \beta_1)z, 0)$ for all $\beta_1 \in [0, 1]$. Furthermore,

i) if $s(-I + (I - Z)B^3) < 0$, then the equilibrium set $(\beta_1z, (1 - \beta_1)z, 0)$, with $\beta_1 \in [0, 1]$, is locally exponentially attractive.

ii) if $s(-I + (I - Z)B^3) > 0$, then the equilibrium set $(\beta_1z, (1 - \beta_1)z, 0)$, with $\beta_1 \in [0, 1]$, is unstable.

**Proof:** See proof of [27, Theorem 3].

Theorem 3 is not in conflict with the claim for finiteness of equilibria presented in Sec. III. The elements in matrices $D^i, B^i, i = 1, 2, 3$, are either a priori fixed to a specific value or they are not. In the case of the latter, these are allowed to take any value in $\mathbb{R}_+$. The dimension of the space of free parameters equals the number of free parameters in the tri-virus system. Each choice of free parameters yields a realization of (4)-(6). The set of choices of free parameters that fall within the special case identified by Theorem 3 has measure zero.

**B. Global convergence to a plane of coexisting equilibria**

Section V-A dealt with the existence and attractivity (resp. instability) of a line of coexisting equilibria. Moving beyond this, it is of natural interest to identify scenario(s) where a plane of coexisting equilibria could exist, and furthermore, seek condition(s) that guarantee stability of such a plane of coexisting equilibria. The present section deals with this issue.

We consider a case where three identical copies of a virus are spreading over the same graph as formalized next.

**Assumption 3:** Let the following conditions hold

i) All three viruses are spreading over the same graph.

ii) For all $i \in [n], \beta_i^1 = \beta_i^2 = \beta_i^3 > 0$.

iii) For all $i = j \in [n]$ and $(i, j) \in E, \beta_{ij}^1 = \beta_{ij}^2 = \beta_{ij}^3$.

Note that for the special case identified in Assumption 3, assuming that the setting in [15] is restricted to the tri-virus case, the existence of a plane of coexisting equilibria has been secured by [15, Corollary 3]. However, [15, Corollary 3] does not provide guarantees for even local (let alone global) convergence to the said plane. To address this shortcoming, first consider the system

$$\dot{x}(t) = (-D + (I - \text{diag}(\tilde{x}))B)x(t), \quad (10)$$

where $\tilde{x}$ is the unique endemic equilibrium of the single virus SIS system associated with $(D, B)$ and $B$ irreducible.

We have the following result.

**Theorem 4:** Let Assumptions 1, 2 and 3 hold, and consider (4)-(6). Further, suppose that $\rho(D^{-1}B) > 1$. Then

i) For all initial conditions satisfying $x^1(0) > 0_n, x^2(0) > 0_n, x^3(0) > 0_n$, we have that $\lim_{t \to \infty} (x^1(t), x^2(t), x^3(t)) \in \mathcal{E}$ exponentially fast, where

$$\mathcal{E} = \{(x^1, x^2, x^3)|\alpha_1 x^1 + \alpha_2 x^2 + \alpha_3 x^3 = \tilde{x}, \sum_{i=1}^3 \alpha_i = 1\},$$

and $\tilde{x}$ is the unique endemic equilibrium of the single virus SIS dynamics defined by $(D, B)$.

ii) Every point on the connected set $\mathcal{E}$ is a coexistence equilibrium.

**Proof:** Please see proof of [27, Theorem 4].

**VI. NUMERICAL ANALYSIS**

We choose $D^i = I_n$ for $i = 1, 2, 3$, with the following $B^i$ matrices, where $\beta_{ij}^k$ are constants that are changed depending on the simulation example being presented.

$$B^1 = \begin{bmatrix} 0 & 0 & 0 & 1.5 \\ 1.5 & 0 & 0 & 0 \\ 0 & 1.5 & 0 & 0 \\ 0 & 0 & 1.5 & 0 \end{bmatrix}, \quad B^2 = \begin{bmatrix} 0 & 1.5 + \beta_{12}^2 & 0 & 0 \\ 0 & 0 & 1.5 & 0 \\ 0 & 0 & 0 & 1.5 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad B^3 = \begin{bmatrix} 0 & 1 & 0.5 + \beta_{13}^3 & 0 \\ 0 & 1 + \beta_{23}^3 & 0 & 1 \\ 0 & 0.5 & 0 & 1 \\ 0.3 + \beta_{33}^3 & 0 & 1.2 & 0 \end{bmatrix}.$$  

Initial conditions are obtained as follows. First, for $i \in [n]$ and $s \in [4]$, we sample a value $p_{is}$ from a uniform distribution $(0, 1)$. Then, for $i \in [n]$ and $k \in [3]$ we set $x^k_i(0) = p_{is}^k / \sum_{s=1}^4 p_{is}^k$, which ensures that the initial conditions are in $D$ but otherwise randomized.

**Example 1:** We set $\beta_{13}^3 = 0, \beta_{12}^2 = -0.1, \beta_{22}^3 = -0.1, \beta_{31}^3 = 0.1$. We obtain $\rho((I - \tilde{x^1})(D^2)^{-1}B^2) = 0.9829$ and $\rho((I - \tilde{x^3})(D^3)^{-1}B^3) = 0.99624$, where $\tilde{x^1} = \text{diag}(\tilde{x}^1)$. Thus, $(\tilde{x^1}, 0, 0)$ is locally exponentially stable. Figure 1a shows convergence to $(\tilde{x^1}, 0, 0)$. Also, $\rho((I - \tilde{x^2})(D^1)^{-1}B^1 = 1.0174, \rho((I - \tilde{x^3})(D^3)^{-1}B^3 = 1.0127$, and $\rho((I - \tilde{x^3})(D^3)^{-1}B^3 = 1.0033$, and $\rho((I - \tilde{x^2})(D^2)^{-1}B^2) = 0.9863$. Hence, $(0, \tilde{x^2}, 0)$ and $(0, 0, \tilde{x^3})$ are both unstable (Thm. 2). Simulations suggest $(\tilde{x^1}, 0, 0)$ is globally attractive for initial conditions in the interior of $D$.

**Example 2:** We set $\beta_{13}^3 = 0, \beta_{12}^2 = -0.1, \beta_{22}^3 = -0.1, \beta_{31}^3 = 0.15$. Thus, $\rho((I - \tilde{x^1})(D^2)^{-1}B^2) = 0.9829$ and $\rho((I - \tilde{x^1})(D^3)^{-1}B^3 = 1.0037$. Then, $(0, 0, \tilde{x^3})$ is locally exponentially stable (Thm. 2), while the two other boundary equilibria are unstable; see Fig. 1b. Additional simulations suggest $(0, 0, \tilde{x^3})$ is globally attractive for initial conditions in the interior of $D$.  

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Example 3: We set \( \beta_3^3 = 0.05, \beta_2^2 = \beta_2^3 = \beta_3^3 = 0 \). Thus, we have a line of equilibria \( (\beta z, (1 - \beta z), 0) \), with \( z = \frac{1}{3} \) and \( \beta_1 \in [0, 1] \) (Thm. 3). Since \( \rho((I - Z)B^3) = 1.0043 \), this line of equilibria is unstable; see Fig. 1c. Then, \((0, 0, \bar{x}^3)\) is locally exponentially stable (Thm. 2). Additional simulations suggest that \((0, 0, \bar{x}^3)\) is globally attractive for initial conditions in the interior of \( D \).

Example 4: We set \( \beta_3^3 = -0.1, \beta_2^2 = \beta_2^3 = \beta_3^3 = 0 \). Thus, we have a line of equilibria \( (\beta_1 z, (1 - \beta_1 z), 0) \), with \( z = \frac{1}{3} \). Now, however, \( \rho((I - Z)B^3) = 0.9911 \), and this line of equilibria is locally exponentially attractive (Thm. 3); see Figure 1d. The boundary equilibrium \((0, 0, \bar{x}^3)\) is unstable (Thm. 2). Simulations suggest that \((\beta_1 z, (1 - \beta_1 z), 0)\) is globally attractive for initial conditions in the interior of \( D \), while the value of \( \beta_1 \) is dependent on the initial conditions.

VII. CONCLUSION

The present paper studied competitive tri-virus spread. We showed that, unlike the bivirus system, the tri-virus system is not monotone. We identified some necessary conditions and a sufficient condition for local exponential convergence to a boundary equilibrium. A special case that admits the existence and local exponential attractivity of a continuum of coexisting equilibria was identified. Finally, we identified another special case where, irrespective of the initial non-zero infection levels, the tri-virus dynamics converge to a plane of coexisting equilibria. No dynamical behavior can be ruled out for the tri-virus. Therefore, establishing the existence or otherwise of limit cycles is a line of future investigation, as well as the study of the endemic behavior of time-varying tri-virus systems.

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