Multi-Layer SIS Model with an Infrastructure Network

Philip E. Paré, Axel Janson, Sebin Gracy, Ji Liu, Henrik Sandberg, and Karl H. Johansson

***Abstract*—In this paper, we develop a layered networked spread model for a susceptible-infected-susceptible (SIS) pathogen-borne disease spreading over a human contact network and an infrastructure network, and refer to it as a layered networked susceptible-infected-water-susceptible (SIWS) model.[1] We identify sufficient conditions for the existence, uniqueness and stability of various equilibria of the aforementioned model. Further, we study an observability problem, where, assuming that the measurements of the pathogen levels in the infrastructure network are available, we provide a necessary and sufficient condition for estimation of the sickness-levels of the nodes in the human contact network. Our results are illustrated through an in-depth set of simulations.**

***Index Terms*—Epidemic Processes, Infrastructure Networks, Stability, Observability**

I. Introduction

The spread of diseases has been a prominent feature of human civilization. The devastation that epidemics can bring worldwide, both from loss of life, and, less importantly, from hindrance to economic activity, has been brought into stark relief by the ongoing Covid-19 crisis. Consequently, understanding the causes of spread of diseases, and, as a result, possibly mitigating (or eradicating) the spread have been questions of longstanding interest for the scientific community. The earliest work in this area can be traced back to [1]. In recent times, modeling and analysis of spreading processes has attracted the attention of researchers across a wide spectrum ranging from mathematical epidemiology [1, 2] and physics [3] to the social sciences [4].

Various models have been proposed in the literature for studying spreading processes, and, in particular, epidemics. Nevertheless, a vast majority of such models factors in *only* person-to-person interaction. However, diseases can spread also through other medium, such as water [5]–[7] (or infected surfaces, e.g., in hospitals [8], public transit vehicles [9], etc). Water-borne pathogens could spread through infrastructure networks, water distribution systems (e.g., rivers, groundwater, and reservoirs) [10]. Moreover, while water quality issues are very prevalent in developing countries with less advanced plumbing and sewage infrastructure, such issues occasionally affect more prosperous countries as well. Notably, Sweden has had a number of water contamination incidents which have affected thousands of residents. For example, in Östersund in Northern Sweden, approximately $27,000$ people ($\sim$45% of the population) became ill and had a water-boil order for over two months as the result of Cryptosporidium contamination of the drinking water [11]. Thus, there is a need for epidemic models that also account for the spread of diseases as a consequence of contamination of shared infrastructure resources [2]. Observe that representing the entire network as a single layer (which, in context, means that the population nodes and resource nodes are treated on an equal footing) could possibly lead to erroneous conclusions [12]. Therefore, in order to better capture the coupled dynamical processes involved, it is prudent to devise an epidemic model that has two layers (one representing the interaction between the human population; another representing the interaction between the infrastructure resources), referred to as *multilayer networks* [13, 14].

The present paper relies on the susceptible-infected-susceptible (SIS) model. In an SIS model, an agent (resp. node), which can be interpreted as either an individual or, equivalently, a community, is either in the infected state or in the susceptible state. Assuming there is a non-trivial disease-spread in a population, an agent that is in the susceptible state, as a consequence of interactions with its neighbors, and depending on its infection rate, transitions to the infected state; an agent that is in the infected state recovers from the infections based on its healing rate. SIS networked models have been studied extensively in the literature [3, 15]–[19].

More recently, in order to account for the spread of diseases through infrastructure resources such as water distribution networks, a variant of the SIS model called the Susceptible-Infected-Water-Susceptible (SIWS) model has been developed in [20], and a multi-virus single resource SIWS model in [21]; an analogous model, inspired from the susceptible-infected-recovered (SIR) framework, had been proposed in [22]–[24]. The paper [20] provides sufficient conditions for global asymptotic stability (GAS) of the healthy state (see [20, Theorem 1]), but it does not provide any theoretical guarantees regarding endemic behavior. More recently, sufficient conditions for GAS of the healthy state, and also for the existence, uniqueness, and GAS of the endemic state have been provided in [21]; see [21, Theorem 2], and [21, Theorem 3], respectively. However, both [20] and [21] consider only the presence of a *single* resource. Notice that if there are *multiple* water resources being accessed by the population, then the spread of virus could be due to not only a) interaction between

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

[2]a node in an infrastructure network is referred to as resource.

an individual in a population node and another individual in (possibly) another population node, and b) interaction between an individual in a population node and a resource node in the infrastructure network, but also due to *interaction between two resource nodes in the infrastructure network*. The present paper aims to develop such a model (called the layered networked SIWS model), and provide an in-depth analysis of its various equilibria viz. existence, uniqueness, and stability. Based on the aforementioned analysis, we would also focus on understanding the effect on the endemic level of the population nodes in the presence of shared resource(s) as opposed to the absence of the same.

While the discussion insofar has been centered around modeling and analysis, another pressing challenge that health administration officials face is to estimate the sickness levels of the population. In particular, for large-scale modern societies it is not economically viable to install sensors in each and every household for measuring the respective household's infection levels. Given that the pathogen levels of infrastructure networks could be measured more easily, the following problem is of interest: under what conditions can we estimate the infection levels of individuals in the population by only measuring the pathogen levels in the infrastructure network? In fact, this problem has been of strong interest in the context of several epidemics such as Ebola [25], Zika [26], Covid [6, 7, 27], etc. It turns out that by employing system-theoretic notions such as observability[3], the aforementioned problem can be addressed by deploying as few sensors as possible. One of the earliest works in this direction is [28], where the problem of *which* subset of nodes in a network should be measured so as to improve observability of a SIS network is addressed; the condition therein involves checking the determinant of the inverse of the observability Grammian. The key theoretical tool that we would be using is the notion of local weak observability of non-linear systems[4].

Paper Contributions: For the layered networked SIWS model that accounts for the presence of multiple resources, our main contributions are as follows:

(i) We identify conditions such that regardless of whether or not a population node (resp. infrastructure resource) is infected or healthy, the model converges to the healthy state, i.e., conditions for GAS of the healthy state; see Theorem 1.

(ii) We provide conditions that guarantee the existence, uniqueness, and GAS of the endemic equilibrium; see Theorem 2.

(iii) We show that the endemic equilibrium in the population nodes for the layered networked SIWS model is greater than or equal to the endemic equilibrium of the population nodes in the networked SIS model, with at least one of the population nodes in the former having a strictly greater endemic level than in the latter; see Proposition 5.

(iv) Given knowledge of the pathogen levels in the infrastructure network, we provide a necessary and sufficient condition for estimating the sickness levels in the human contact network; see Theorem 3.

Additionally, we also have the following auxiliary contributions: a necessary, and sufficient, condition for the healthy state to be the unique equilibrium of the model; see Corollary 1. A sufficient (but not necessary) condition for local weak observability of the layered networked SIWS model, and, based off of this sufficient condition, we present a design of the observability matrix that results in the layered networked SIWS model being locally weakly observable; see Proposition 6 and Corollary 2, respectively.

A preliminary version of this paper appeared in [30]. The present paper involves a more comprehensive treatment by providing theoretical guarantees for the endemic behavior, studying a different observability problem, providing novel sufficient conditions for local weak observability, complete proofs of all assertions, and, finally, an in-depth set of simulations.

Paper Organization: The paper unfolds as follows. We conclude the present section by collecting all the notation used in the rest of the paper. The layered networked SIWS model is developed in Section II, where, we subsequently, also state the problems of interest. The analysis of the various equilibria of the model, namely stability of the healthy state and existence, uniqueness, and stability of the endemic state, is given in Section III. The observability problem is studied in Section IV. Simulations illustrating our theoretical findings are provided in Section V. Finally, some concluding remarks, together with some research directions of possible interest to the wider community, are provided in Section VI.

Notation: For any positive integer n, we use $[n]$ to denote the set $\{1, 2, \ldots, n\}$. The ith 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. For any vector $x \in \mathbb{R}^n$, we use $\mathrm{diag}(x)$ to denote the $n \times n$ diagonal matrix whose ith diagonal entry equals x_i. For any two sets $\mathcal{A}$ and $\mathcal{B}$, we use $\mathcal{A} \setminus \mathcal{B}$ to denote the set of elements in $\mathcal{A}$ but not in $\mathcal{B}$. 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 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., $s(M) = \max\{\mathrm{Re}(\lambda) \,:\, \lambda \in \sigma(M)\}$. Given a matrix A, $A \prec 0$ (resp. $A \preccurlyeq 0$) indicates that A is negative definite (resp. negative semidefinite), whereas $A \succ 0$ (resp. $A \succcurlyeq 0$) indicates that A is positive definite (resp. positive semidefinite).

II. The Model

In this section, we develop a distributed continuous-time pathogen model. This model will be hereafter referred to as *the layered networked SIWS model*; see Figure 1.

A. The Layered Networked SIWS Model

Consider a pathogen spreading over a two-layer network consisting of $n > 1$ groups of individuals and $m > 1$

[3]A system has the property of observability, if, given a series of output measurements, the initial state of the system can be uniquely determined.

[4]We say that two initial states are *indistinguishable* if the corresponding outputs are equal for all time instants. A system is locally weakly observable if one can instantaneously distinguish each initial state from its neighbors [29].

infrastructure resources. The individuals in a group could become contaminated as a consequence of their interactions with other infected individuals and/or as a consequence of their interactions with infected infrastructure resources.

We denote by $I_i(t)$ and $S_i(t)$ the number of infected and susceptible individuals, respectively, in group i at time $t \geq 0$. We denote by N_i the total number of individuals in group i, and assume that N_i does not change over time, i.e., $S_i(t) + I_i(t) = N_i$ for all $i \in [n]$ and $t \geq 0$, This assumption implies that the birth and death rates for each group are equal. Thus, it simplifies the model. The healing rate of each group i is denoted by γ_i, the birth rate by μ_i, the death rate by $\bar{\mu}_i$ (which equals μ_i), the person-to-person infection rates by a_{ij} and the infrastructure-to-person infection rates by a^w_{ij}. We denote by $w_j(t)$ the pathogen concentration in the jth infrastructure resource, with δ^w_j denoting the corresponding decay rate of the pathogen, ζ^w_{jk} denoting the person-infrastructure contact rate of group k to infrastructure node j, and α_{kj} representing the flow of the pathogen from node k to node j in the infrastructure network. In the rest of this paper, we will assume that all of the aforementioned parameters are nonnegative. We assume that the individuals are susceptible at birth regardless of whether (or not) their parents are infected. The evolution of the numbers of infected and susceptible individuals in each group i is, consistent with the ideas in [31, 32], as follows:

$$\begin{aligned}
\dot{S}_i(t) &= \mu_i N_i - \bar{\mu}_i S_i(t) + \gamma_i I_i(t) - \textstyle\sum_{j=1}^n a_{ij} \frac{S_i(t)}{N_i} I_j(t) \\
&\quad - \textstyle\sum_{j=1}^m a^w_{ij} w_j(t) S_i(t) \\
&= (\mu_i + \gamma_i) I_i(t) - \textstyle\sum_{j=1}^n a_{ij} \frac{S_i(t)}{N_i} I_j(t) \\
&\quad - \textstyle\sum_{j=1}^m a^w_{ij} w_j(t) S_i(t), \qquad (1) \\
\dot{I}_i(t) &= -\gamma_i I_i(t) - \bar{\mu}_i I_i(t) + \textstyle\sum_{j=1}^n a_{ij} \frac{S_i(t)}{N_i} I_j(t) \\
&\quad + \textstyle\sum_{j=1}^m \alpha^w_{ij} w_j(t) S_i(t) \\
&= (-\gamma_i - \mu_i) I_i(t) + \textstyle\sum_{j=1}^n a_{ij} \frac{S_i(t)}{N_i} I_j(t) \\
&\quad + \textstyle\sum_{j=1}^m a^w_{ij} w_j(t) S_i(t), \qquad (2) \\
\dot{w}_j &= -\delta^w_j w_j + \textstyle\sum_{k=1}^n \zeta^w_{jk} I_k + \sum_{k=1}^m \alpha_{kj} w_k \\
&\quad - w_j \sum_{k=1}^m \alpha_{jk}. \qquad (3)
\end{aligned}$$

It is clear from (1) and (2), that $\dot{S}_i(t) + \dot{I}_i(t) = 0$, which is consistent with our assumption that N_i is a constant.

We simplify the model further by defining the fraction of infected individuals in each group i as

$$x_i(t) = \frac{I_i(t)}{N_i}.$$

By defining the following parameters

$$\delta_i = \gamma_i + \mu_i, \quad \beta_{ij} = a_{ij} \frac{N_j}{N_i}, \quad \beta^w_{ij} = N_i a^w_{ij}, \quad c^w_{jk} = \zeta^w_{jk}/N_k$$

and from (1), (2), and (3), it follows that

$$\begin{aligned}
\dot{x}_i &= -\delta_i x_i + (1 - x_i) \left(\textstyle\sum_{j=1}^n \beta_{ij} x_j + \sum_{j=1}^m \beta^w_{ij} w_j \right), \qquad (4) \\
\dot{w}_j &= -\delta^w_j w_j + \textstyle\sum_{k=1}^m \alpha_{kj} w_k - w_j \sum_{k=1}^m \alpha_{jk} + \sum_{k=1}^n c^w_{jk} x_k. \qquad (5)
\end{aligned}$$

Note that, we also allow for the healing rate of an infrastructure resource j, δ^w_j, to be zero.

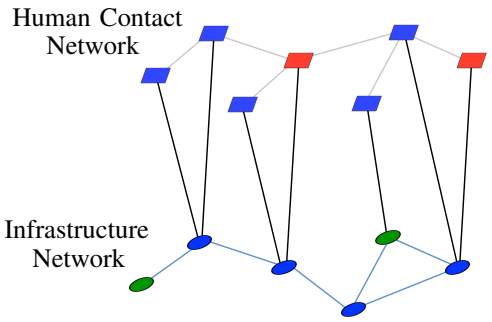

Fig. 1: Multi-layered SIWS model: The disease (depicted by red) spreads between household nodes (squares) and the pathogen (green) spreads through infrastructure network nodes (circles). Blue indicates healthy. The model permits transmission from the infrastructure network to the human contact network, vice versa, and not necessarily symmetrically. We use the terms "infrastructure network" and "water network" interchangeably.

The model from (4)-(5) in vector form becomes:

$$\begin{aligned}
\dot{x} &= (B - XB - D)x + (I - X)B_w w \qquad (6) \\
\dot{w} &= -D_w w + A_w w + C_w x, \qquad (7)
\end{aligned}$$

where $B = [\beta_{ij}]_{n \times n}$, $X = \operatorname{diag}(x)$, $B_w = [\beta^w_{ij}]_{n \times m}$, A_w has off-diagonal entries equal to α_{kj} and diagonal entries equal to $-\sum_k \alpha_{kj}$, and $C_w = [c^w_{jk}]_{m \times n}$. Therefore, the columns of A_w sum to zero.

System (6)-(7) could be written more compactly using

$$\begin{aligned}
z(t) &:= \begin{bmatrix} x(t) \\ w(t) \end{bmatrix}, \; X(z(t)) := \begin{bmatrix} \operatorname{diag}(x(t)) & 0 \\ 0 & 0 \end{bmatrix}, \\
B_f &:= \begin{bmatrix} B & B_w \\ C_w & A_w - \operatorname{diag}(A_w) \end{bmatrix}, \text{ and} \qquad (8) \\
D_f &:= \begin{bmatrix} D & 0 \\ 0 & D_w - \operatorname{diag}(A_w) \end{bmatrix}.
\end{aligned}$$

With the new notations in place, (6)-(7) can be rewritten as:

$$\dot{z} = \big(- D_f + (I - X(z)) B_f \big) z. \qquad (9)$$

Remark 1. *We highlight how the model considered in the present paper is connected with similar models in the existing literature*

i) If $m = 1$, (9) coincides with the model in [20], and with the multi-virus model in [21], when the latter is particularized for the single-virus case.

ii) If $w_j(t) = 0$ for all t and all $j \in [m]$, or equivalently, there is no coupled infrastructure network, (9) reduces to the regular networked SIS model in [33].

The spread of viruses over infrastructure networks has been studied in [34], but the model therein only accounts for spread between the nodes in infrastructure network; the coupling with a human population network is not considered. Note that, in contrast to [34], the model in (9) admits three media for spread, namely, population-population, population-resource, and resource-resource.

B. Problem Statements

In the sequel, for the model in (9), we will be interested in addressing the following problems:

(i) Identify a condition such that $z(t)$ converges asymptotically to the healthy state, i.e., $z = \mathbf{0}$.

(ii) Under what conditions does there exist an endemic equilibrium $\hat{z} > \mathbf{0}$, and under such conditions, does the system converge asymptotically to $\hat{z}$ from any non-zero initial condition?

(iii) Let $\tilde{z} = \begin{bmatrix}\tilde{x}^\top & \tilde{w}^\top\end{bmatrix}^\top$, where $\tilde{x}$ (resp. $\tilde{w}$) denotes the endemic equilibrium of the population nodes (resp. the shared resources). Let $\hat{x}$ denote the unique endemic equilibrium of the SIS model without a shared resource. What is the relation between $\hat{x}$ and $\tilde{x}$?

(iv) Identify a necessary, and sufficient, condition such that $x(0)$ can be uniquely recovered given $z(t)$.

C. Positivity Assumptions

We impose the following assumptions on the parameters.

Assumption 1. *Suppose that $\delta_i > 0$ for all $i \in [n]$, $\delta_j^w + \sum_k \alpha_{jk} > 0$ for all $j \in [m]$, $\beta_{ij} \geq 0$ for all $i, j \in [n]$, and $\beta_{ij} > 0$ whenever group j is a neighbor of group i.*

Assumption 1 says, among other things, that the healing and infection rate of each population group is strictly positive.

Since each x_i represents the fraction of infected individuals in group i, it is immediate that the initial value of x_i is in $[0, 1]$, because otherwise the value of x_i will lack physical meaning for the epidemic model considered here. Similarly, it is also natural to assume that the initial value of w_j (measured, for instance, in milligrams per litre) is nonnegative. Hence, we can restrict our analysis to the set:

$$\mathcal{D} \coloneqq \{y(t) : x(t) \in [0, 1]^n, w(t) \in [0, \infty)^m\}. \tag{10}$$

The following lemma establishes that, under Assumption 1, the set $\mathcal{D}$ is positively invariant.

Lemma 1. *Suppose that Assumption 1 holds. Suppose that $x_i(0) \in [0, 1]$ for all $i \in [n]$ and $w_j(0) \geq 0$ for all $j \in [m]$. Then, $x_i(t) \in [0, 1]$ for all $i \in [n]$ and $w_j(t) \geq 0$ for all $j \in [m]$, for all $t \geq 0$.*

Proof: Suppose that at some time τ, $x_i(\tau) \in [0, 1]$ for all $i \in [n]$ and $w_j(\tau) \geq 0$ for all $j \in [m]$. First consider any index $j \in [m]$. If $w_j(\tau) = 0$, then from (5) and Assumption 1, $\dot{w}_j(\tau) \geq 0$. Therefore $w_j(t) \geq 0$ for all $t \geq \tau$.

Now consider any index $i \in [n]$. If $x_i(\tau) = 0$, then from (4) and Assumption 1, $\dot{x}_i(\tau) \geq 0$. If $x_i(\tau) = 1$, then again from (4) and Assumption 1, $\dot{x}_i(\tau) < 0$. Therefore, $x_i(t)$ will be in $[0, 1]$ for all times $t \geq \tau$.

Since the above arguments hold for any $i \in [n]$ and any $j \in [m]$, we have that $x_i(t) \in [0, 1]$ for all $i \in [n]$ and $w_j(t) \geq 0$ for all $j \in [m]$, $t \geq \tau$. Since it is assumed that $x_i(0) \in [0, 1]$ for all $i \in [n]$ and $w_j(0) \geq 0$ for all $j \in [m]$, the lemma follows by setting $\tau = 0$. □

III. Stability analysis of the equilibria

In this section, we analyze the equilibria of the proposed model and their stability both locally and globally.

A. Local Stability of the Healthy State

Consider $(\tilde{x}, \tilde{w})$, an equilibrium of (6)-(7). The Jacobian matrix of the equilibrium, denoted by $J(\tilde{x}, \tilde{w})$, is

$$J(\tilde{x}, \tilde{w}) = \begin{bmatrix} B - \tilde{X}B - D - F_1 - F_2 & (I - \tilde{X})B_w \\ C_w & -D_w + A_w \end{bmatrix}, \tag{11}$$

where $\tilde{X}, F_1, F_2$ are diagonal matrices given by

$$\tilde{X} = \operatorname{diag}\left(\tilde{x}_1, \tilde{x}_2, \cdots, \tilde{x}_n\right), \tag{12}$$

$$F_1 = \operatorname{diag}\left(\textstyle\sum_{j=1}^n \beta_{1j}\tilde{x}_j, \sum_{j=1}^n \beta_{2j}\tilde{x}_j, \cdots, \sum_{j=1}^n \beta_{nj}\tilde{x}_j\right), \tag{13}$$

$$F_2 = \operatorname{diag}\left(\textstyle\sum_{j=1}^n \beta_{1j}^w\tilde{w}_j, \sum_{j=1}^n \beta_{2j}^w\tilde{w}_j, \cdots, \sum_{j=1}^n \beta_{nj}^w\tilde{w}_j\right). \tag{14}$$

In the case when $\tilde{x} = \mathbf{0}$ and $\tilde{w} = \mathbf{0}$, i.e., at the healthy state (also referred to as the disease-free equilibrium),

$$J(\mathbf{0}, \mathbf{0}) = \begin{bmatrix} B - D & B_w \\ C_w & A_w - D_w \end{bmatrix} = B_f - D_f.$$

If either $B_w = 0$ or $C_w = 0$, i.e., the pathogen does not affect the population or humans can not contaminate the infrastructure network by using it, we have the following result.

Proposition 1. *If $s(B - D) < 0$, $s(A_w - D_w) < 0$, and $B_w = 0$ or $C_w = 0$, then the healthy state $(\mathbf{0}, \mathbf{0})$ of (6)-(7) is locally exponentially stable.*

Proof: If $B_w = 0$ or $C_w = 0$ then $J(\mathbf{0}, \mathbf{0})$ is a triangular matrix (lower or upper, respectively), and therefore the spectrum of the matrix is equal to the union of the spectrum of the two block matrices on the diagonal. Consequently, if $s_1(B - D) < 0$ and $s_1(A_w - D_w) < 0$ then $J(\mathbf{0}, \mathbf{0})$ is Hurwitz and by Lyapunov's indirect method [35] the healthy state $(\mathbf{0}, \mathbf{0})$ of (6)-(7) is locally exponentially stable. □

For nonzero B_w and C_w, we have the following result.

Proposition 2. *Let Assumption 1 hold. If $\rho(D_f^{-1}B_f) < 1$ and B_f is irreducible, then the healthy state $(\mathbf{0}, \mathbf{0})$ of (6)-(7) is locally exponentially stable.*

Proof: See Appendix. □

B. Global Stability of the Healthy State

To state our first main result, we need the following concept. Consider an autonomous system $\dot{x}(t) = f(x(t))$, where $f : \mathcal{D} \to \mathbb{R}^n$ is a locally Lipschitz map from a domain $\mathcal{D} \subset \mathbb{R}^n$ into $\mathbb{R}^n$. Let $\tilde{x}$ be an equilibrium of the system and $\mathcal{E} \subset \mathcal{D}$ be a domain containing $\tilde{x}$. The equilibrium $\tilde{x}$ is called asymptotically stable with the domain of attraction $\mathcal{E}$ if for any $x(0) \in \mathcal{E}$, there holds $\lim_{t\to\infty} x(t) = \tilde{x}$.

The global stability of the healthy state is characterized by the following theorem.

Theorem 1. *Let Assumption 1 hold. If $\rho(D_f^{-1}B_f) \leq 1$ and B_f is irreducible, then the healthy state of (6)-(7) is asymptotically stable with the domain of attraction $\mathcal{D}$, with $\mathcal{D}$ given in (10).*

Proof: See Appendix. □

Theorem 1 addresses Question (i) in Section II-B.

C. Reproduction Number

In epidemiology the reproduction number, R_0, is the average number of people that become infected from one infected individual. If $R_0 > 1$ the disease will lead to an outbreak; if $R_0 \leq 1$ the disease will die out. For the networked SIS model with no water resources, it has been shown that $\rho(D^{-1}B)$ is the reproduction number, and that if $\rho(D^{-1}B) \leq 1$, the model will asymptotically converge to the healthy state for all initial conditions, and if $\rho(D^{-1}B) > 1$, the model will asymptotically converge to a unique epidemic state for all initial conditions except for the healthy state [31].

For the layered networked SIWS model (6)-(7), Theorem 1 implies that when $\rho(D_f^{-1}B_f) \leq 1$, the model will asymptotically converge to the healthy state for all initial conditions, which implies that the healthy state is the unique equilibrium. We call $\rho(D_f^{-1}B_f)$ the basic reproduction number of the layered networked SIWS model (6)-(7), and compare its value with that of the networked SIS model, $\rho(D^{-1}B)$, to illustrate the effect of the water distribution network. Note that

$$\begin{aligned} D_f^{-1}B_f &= \begin{bmatrix} D^{-1} & 0 \\ 0 & (D_w - \mathrm{diag}(A_w))^{-1} \end{bmatrix} \begin{bmatrix} B & B_w \\ C_w & A_w - \mathrm{diag}(A_w) \end{bmatrix} \\ &= \begin{bmatrix} D^{-1}B & D^{-1}B_w \\ (D_w - \mathrm{diag}(A_w))^{-1}C_w & (D_w - \mathrm{diag}(A_w))^{-1}A_w - \mathrm{diag}(A_w) \end{bmatrix}. \end{aligned}$$

We need the following lemma.

Lemma 2. *[36, Lemma 2.6] Suppose that N is an irreducible nonnegative matrix. If M is a principal square submatrix of N, then $\rho(M) < \rho(N)$.*

Since $D_f^{-1}B_f$ is an irreducible nonnegative matrix by Assumption 1, and since $D^{-1}B$ is a principal square submatrix of $D_f^{-1}B_f$), from Lemma 2 it follows that $\rho(D_f^{-1}B_f) > \rho(D^{-1}B)$. Therefore we have the following result.

Proposition 3. *Suppose that Assumption 1 holds. Then, the basic reproduction number of the layered networked SIWS model (6)-(7) is greater than that of the networked SIS model.*

Proposition 3 implies that eradication of the disease in the population in itself does not guarantee that the system is disease-free. That is, the presence of infrastructure network makes the system more vulnerable to SIS-type diseases than otherwise.

D. Analysis of the Endemic Behavior

It turns out that the condition in Proposition 1 being violated results in the instability of the healthy state $(\mathbf{0}, \mathbf{0})$ of (6)-(7), as we show in the following proposition.

Proposition 4. *Suppose that $B_w = 0$ or $C_w = 0$. If $s(B - D) > 0$ or $s(A_w - D_w) > 0$, then the healthy state $(\mathbf{0}, \mathbf{0})$ of* (6)-(7) *is unstable.*

Proof: Since by assumption, $B_w = 0$ (resp. $C_w = 0$), it follows that the Jacobian matrix of the equilibrium evaluated at the healthy state, i.e., $J(\mathbf{0}, \mathbf{0})$, is a block lower triangular (resp. upper triangular) matrix. Hence, the eigenvalues of $J(\mathbf{0}, \mathbf{0})$ are same as those of matrices $B - D$ and $A_w - D_w$. Consequently, if $s(B-D) > 0$ and/or $s(A_w - D_w) > 0$, then $s(J(\mathbf{0}, \mathbf{0})) > 0$. Hence, the healthy state $(\mathbf{0}, \mathbf{0})$ of (6)-(7) is unstable. □

Simulations indicate the existence of an endemic state (also referred to as the endemic equilibrium) when the eigenvalue condition in Theorem 1 is violated (see Figure 5 in Section V), a rigorous result, however, remains missing. Therefore, we consider the following variant of Assumption 1.

Assumption 2. *Assume that $\delta_i > 0$, $\delta_j^w > 0$, $\beta_{ij} \geq 0$, $\beta_{ij}^w \geq 0$, and that, for $j \neq k$, $\alpha_{jk} \geq 0$, with $\alpha_{jj} = -\sum_{k \neq j}^{m} \alpha_{jk}$.*

Assumption 2 states that the system parameters, with the exception of the rate of flow of pathogen within a resource node, are nonnegative. It is easy to show that Assumption 2 implies Assumption 1, and, is, thus, more restrictive. Hence, we only need Assumption 2 in the sequel.

Theorem 2. *Consider* (9) *under Assumption 2. Suppose that B_f is irreducible and $\rho(D_f^{-1}B_f) > 1$. Then there exists a unique endemic equilibrium $\tilde{z} \gg \boldsymbol{0}$. Furthermore, $\tilde{z}$ is asymptotically stable with the domain of attraction $\mathcal{D} \setminus \{\boldsymbol{0}\}$, with $\mathcal{D}$ given in* (10)*.*

Proof: See Appendix. □

Theorem 2 says that as long as the reproduction number of the layered SIWS network is greater than one, then, assuming that there is at least one node (population or infrastructure) that is infected initially, the spreading process converges to a unique proportion in each population node, and a unique infection level in each infrastructure node. Thus, Theorem 2 addresses Question (ii) in Section II-B. Note that Theorem 2 improves upon [21, Theorem 3] since it also accounts for *multiple* shared resources.

Combining Theorems 1 and 2 yields a necessary, and sufficient, condition for the healthy state to be the unique equilibrium of (6)-(7). Hence, we have the following result:

Corollary 1. *Consider the layered networked SIWS model in* (6)-(7) *under Assumption 2. Suppose that B_f is irreducible. Then the healthy state is the unique equilibrium in the domain $\mathcal{D}$ if, and only if, $\rho(D_f^{-1}B_f) \leq 1$.*

Rewriting the condition in Corollary 1 in view of [19, Proposition 1] tells us that insofar the linearized state matrix of system (9) (linearized around the healthy state) is Hurwitz, the healthy state is the *only* equilibrium of system (9).

Remark 2. *Assuming that $\rho(D_f^{-1}B_f) \leq 1$, if the weights on the multi-layer network increase or if new edges are added to the multi-layer network, then the entries in the B_f matrix increase. Consequently, since $D_f^{-1}B_f$ is irreducible nonnegative, from [19, Lemma 5, 4)] it follows that if the weights are increased sufficiently well, then $\rho(D_f^{-1}B_f) > 1$. Hence, the system transitions from the healthy to endemic state due to the changes in the structure of the multi-layer network.*

A very pertinent question that could arise at this point is as follows: focusing solely on the population, is there a relation between the endemic equilibrium of the layered networked SIWS model, and that of the networked SIS model. In order to answer this, we recall the latter:

$$\dot{x} = (B - XB - D)x. \tag{15}$$

In order to ensure that the model in (15) is well-defined, we need to particularize Assumption 2, for the setting without shared resource(s). This is given as follows:

Assumption 3. *Suppose that $\delta_i > 0$ and $\beta_{ij} \geq 0$ for all $i, j \in [n]$.*

Let $\hat{x}$ denote the unique endemic equilibrium of (15) and recall that $\tilde{z} = [\tilde{x}^\top \ \tilde{w}^\top]^\top$ denotes the unique endemic equilibrium of (9). With this notation and Assumption 3 in place, we present the following result.

Proposition 5. *Consider* (9) *under Assumption 2, and* (15) *under Assumption 3. Suppose that B_f and B are irreducible, and that $\rho(D_f^{-1}B_f) > 1$, and $\rho(D^{-1}B) > 1$. Then $\tilde{x} > \hat{x}$.*

Proof: See Appendix. □

Proposition 5 says that the endemic level in each of the population nodes for the layered networked SIWS model is greater than or equal to the endemic level of the population nodes in the absence of shared resource(s). As such, it addresses Question (iii) in Section II-B.

IV. Observability Problem

In this section, we aim to address the following question: Assuming there are not enough tests available to measure the sickness levels of the population, can measurements of the pathogen levels in the water network, be used to estimate the sickness levels of the population, or the source of the outbreak (the initial states of the system)? We introduce the following notation:

$$y = Gw, \tag{16}$$

where $G \in \mathbb{R}^{q \times m}$ is a measurement matrix, with $q \in \mathbb{Z}_+$ denoting the number of measurements. The problem posed in Question (iv) could be re-written as follows: Given B, D, A_w, B_w, C_w, D_w, G, and measurements y, find conditions for when $x(0)$ can be recovered.

We derive conditions such that, given measurements of pathogen levels in the water network, it is possible to uniquely recover the initial state of population network. Towards this end, we appeal to the rank of the Jacobian of the Lie derivatives, and apply the results from [29]. Consequently, the Lie derivative calculations are as follows:

$$\begin{aligned}
y &= Gw \\
\dot{y} &= G\dot{w} = GC_w x + G\underbrace{(A_w - D_w)}_{\check{A}_w} w \\
\ddot{y} &= G\ddot{w} = GC_w\big(\underbrace{(B - XB - D)}_{F_x} x + \underbrace{(I - X)B_w}_{F_w} w\big) \\
&\qquad + G\check{A}_w C_w x + G\check{A}_w^2 w \\
y^{(3)} &= Gw^{(3)} = G\big(C_w\dddot{x} + \check{A}_w C_w \ddot{x} + \check{A}_w^2 C_w \dot{x} + \check{A}_w^3 C_w x + \check{A}_w^3 w\big) \\
&= G\big(\check{A}_w^3 w + \check{A}_w^2 C_w x + C_w F_x^2 x + \check{A}_w C_w F_x x \\
&\qquad + \check{A}_w C_w F_w w + C_w F_w C_w x + C_w F_x F_w w \\
&\qquad + C_w F_w \check{A}_w w - C_w \dot{X}(Bx + B_w w)\big) \\
&\ \ \vdots \\
y^{(m+n)} &= Gw^{(m+n)} = G\big(\check{A}_w^{m+n} w + \check{A}_w^{m+n-1} C_w x \\
&\qquad + C_w F_x^{(m+n-1)} x + \cdots\big),
\end{aligned}$$

where $\dot{x}$ and $\dot{w}$ are defined in (6) and (7) and $\dot{X} = \mathrm{diag}(\dot{x})$.

We explore the case when we assume that all nodes in the human contact network are initially healthy, that is, $x(0) = \mathbf{0}$. This case is especially interesting because the early part of an outbreak is when tests are the scarcest and, therefore, using water sensors could be of most utility. Thus, we explore the Jacobian of the above Lie derivatives evaluated at $x(0) = \mathbf{0}$, called $\mathcal{O}$, where $\mathcal{O} =$

$$\begin{bmatrix}
0 & G \\
GC_w & G\check{A}_w \\
GC_w(\underbrace{F_{x_0} - \check{B}_w}_{X_x}) & G(\check{A}_w^2 + C_w B_w) \\
G(\check{A}_w^2 C_w + \check{A}_w C_w X_x & G(\check{A}_w^3 + \check{A}_w C_w B_w + C_w(B_w F_{w_0} \\
+ C_w(X_x^2 - \check{B}_w B - \check{B})) & + X_x B_w - \check{B}_w B_w)) \\
\vdots & \vdots
\end{bmatrix}, \tag{17}$$

with $F_{x_0} = (B - D)$, $F_{w_0} = (A_w - D_w)$, $\check{B}_w = \mathrm{diag}(B_w w)$ and $\check{B} = \mathrm{diag}(BB_w w + B_w F_{w_0} w)$. Note, $\mathcal{O}$ has $q(n+m)$ rows and $n+m$ columns.

Therefore, from [29, Theorems 3.1 and 3.12], and since the system is analytic, we have the following theorem.

Theorem 3. *The layered networked SIWS model in* (6)-(7) *with measurements in* (16) *is locally weakly observable at $x(0) = \mathbf{0}$ if and only if $\mathcal{O}$, as defined in* (17)*, has full rank.*

Observe that Theorem 3 provides a necessary and sufficient condition for checking whether (or not) the layered networked SIWS model is locally weakly observable at $x(0) = \mathbf{0}$, and thus answers Question (iv) in Section II-B. However, the condition therein involves checking the rank of the $\mathcal{O}$ matrix, which in turn, involves too many computations, since $\mathcal{O}$ has $q(n+m)$ rows. This drawback motivates us to seek a simpler, easier to check, sufficient condition for the layered networked SIWS model to be locally weakly observable at $x(0) = \mathbf{0}$, and is presented next.

Proposition 6. *Suppose that the matrices G and GC_w have full column rank. Then the layered networked SIWS model in* (6)-(7) *with measurements in* (16) *is locally weakly observable at $x(0) = \mathbf{0}$.*

Proof: Define the matrices

$$\mathcal{G} := \begin{bmatrix} 0 & G \\ GC_w & G\check{A}_w \end{bmatrix} \text{ and } \mathcal{F} := \begin{bmatrix} G & 0 \\ G\check{A}_w & GC_w \end{bmatrix}. \tag{18}$$

Observe that since $\mathcal{F}$ is just a permutation of the block columns of $\mathcal{G}$, $\mathrm{rank}(\mathcal{F}) = \mathrm{rank}(\mathcal{G})$. Since $\mathcal{F}$ is a block lower triangular matrix, $\mathrm{rank}(\mathcal{F}) \geq \mathrm{rank}(G) + \mathrm{rank}(GC_w)$. By assumption, matrices G and GC_w have full column rank, which implies that $\mathrm{rank}(\mathcal{F}) \geq n + m$. Observe also that the total number of columns in $\mathcal{F}$ equals $n + m$. Therefore, it follows that $\mathrm{rank}(\mathcal{F}) \leq n+m$. Hence, $\mathrm{rank}(\mathcal{F}) = n+m$, i.e., $\mathcal{F}$ has full column rank. Now note that $\mathcal{F}$ is a submatrix of $\mathcal{O}$, that has the *same* number of columns as $\mathcal{O}$. Also observe that adding more rows to $\mathcal{F}$ does not lead to matrix $\mathcal{F}$ becoming rank deficient. This implies that matrix $\mathcal{O}$ has full column rank, and therefore, from Theorem 3, we conclude that the layered networked SIWS model in (6)-(7) with measurements in (16) is locally weakly observable at $x(0) = \mathbf{0}$. □

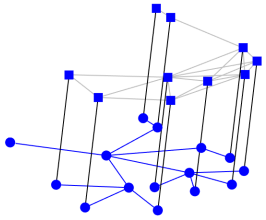

Fig. 2: The contact network of population and resource nodes used for simulations, represented by squares and circles, respectively.

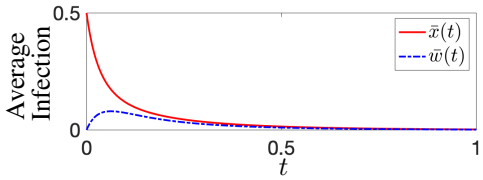

Fig. 3: $D = 5I$, $D_w = 5I$, and the matrices B, B_w, and C_w are binary. Since $\rho(D_f^{-1}B_f) < 1$, the virus gets eradicated.

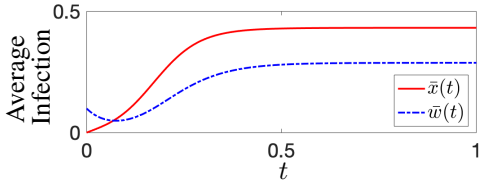

Fig. 4: $D = 2I$, $D_w = 2I$, and the matrices B, B_w, and C_w are binary. Since $\rho(D_f^{-1}B_f) > 1$, the virus becomes endemic.

Given that both Proposition 6 and Theorem 3 provide sufficient conditions for local weak observability, it is natural to ask how the two conditions are related. The following remark addresses this question.

Remark 3. *Proposition 6 implies Theorem 3. The converse, however, is not true. To see this, consider the following example: Let $n = 2, m = 2$. With $G = I$, $D = I$, $D_w = I$,*

$$B = \begin{bmatrix} 1 & 1 \\ 1 & 2 \end{bmatrix}, \; B_w = \begin{bmatrix} 1 & 0 \\ 1 & 0 \end{bmatrix},$$
$$C_w = \begin{bmatrix} 1 & 1 \\ 1 & 1 \end{bmatrix}, \; A_w = \begin{bmatrix} 1 & 1 \\ 1 & 1 \end{bmatrix},$$

it is clear that GB_w does not have full column rank, so the conditions for Proposition 6 are not met. However, allowing $w = (w_1, w_2)$ to be free, we obtain

$$GW_w = \begin{bmatrix} 1 - 2w_1 & 1 \\ 2 - 2w_1 & 1 \end{bmatrix}.$$

Therefore, independent of the value of w, the rightmost column of $\mathcal{O}$ is linearly independent of the other three columns of $\mathcal{O}$, that is, $\mathcal{O}$ has full column rank. Thus, the condition in Theorem 3 is met. ■

We now highlight an interesting consequence of Proposition 6.

Corollary 2. *Let $n \geq m$. If $G = I_{m\times m}$ and C_w has full column rank, then the layered networked SIWS model in* (6)-(7) *with measurements in* (16) *is locally weakly observable at $x(0) = \mathbf{0}$.*

Proof: Suppose that, by assumption, $G = I_{m\times m}$. Consequently, $\text{rank}(G) = m$. Moreover, $GC_w = C_w$, and hence $\text{rank}(GC_w) = \text{rank}(C_w)$. Since, by assumption, $\text{rank}(C_w) = n$, it follows that the conditions in Proposition 6 are satisfied, and hence the result follows. □

Observe that the result in Corollary 2 could potentially inform sensor placement (in the infrastructure network) strategies for detecting infection levels of the population in the layered networked SIWS model.

V. Simulations

For all simulations, we consider a network of 10 population nodes and 15 resource nodes. This network is depicted in Fig. 2, with population nodes as squares and resource nodes as circles. We denote the average infection proportion of the virus across the population nodes by $\bar{x}(t)$, and the average contamination across the resource nodes by $\bar{w}(t)$. The terms β_{ij}, β_{ij}^w, and α_{ij} are all binary, i.e. equal to one whenever nodes i and j are neighbors, for all simulations. For the simulations in Fig. 3, Fig. 4, and Fig. 5 we set c_{ij}^w to be binary which results in the network being irreducible. By choosing $D = 5I$, $D_w = 5I$, we see that $\rho(D_f^{-1}B_f) < 1$. Consequently, consistent with the result in Theorem 1, the virus is asymptotically eradicated across the network; see Fig. 3. Choosing $D = 2I$, $D_w = 2I$ results in $\rho(D_f^{-1}B_f) > 1$. Therefore, consistent with the result in Theorem 2, the virus becomes endemic across all population and resource nodes, asymptotically approaching some positive equilibrium; see Fig. 4. Choosing $D = 4I$ and D_w equal to a zero matrix, except for one diagonal entry equal to 100, Assumption 1 is fulfilled but Assumption 2 is violated. Therefore Theorem 2 does not apply, despite $\rho(D_f^{-1}B_f) > 1$, yet the virus still appears to converge to some positive equilibrium; see Fig. 5.

For the simulations depicted in Fig. 6 we chose $D = 3I$, $D_w = 0.2I$. Since D_w is a positive diagonal matrix, the resource network requires some non-zero c_{ij}^w to sustain a positive level of contamination. Choosing $c_{ij}^w = 0$ for all i, j ensures that the contamination across all resource nodes decays to zero; see the blue curve in the left of Fig. 6. However, B is an irreducible matrix, and we still have $\rho(D^{-1}B) > 1$. Therefore, the infection levels in the population network converge to an endemic equilibrium, consistent with the results in [19, 31]; see the red curve in the left of Fig. 6. Setting c_{ij}^w to be binary as before results in the contamination of the resource network converging to a positive equilibrium; see the blue curve in the right of Fig. 6. Consistent with the result in Proposition 5, it can be seen that in the absence of contamination in the resources, the endemic state in the population is smaller, whereas if the resources are also contaminated then the endemic state in the population is larger; see the red curves in the left of and the right of Fig. 6, respectively.

Finally, for the simulation in Fig. 7 we set matrices B, B_w and C_w to be binary. The healing rates for each node in the population and the resource are set to the same value and varied. Fig. 7 illustrates how the equilibrium of the system changes as a function of the healing rate (left) and the spectral radius $\rho(D_f^{-1}B_f)$ (right).

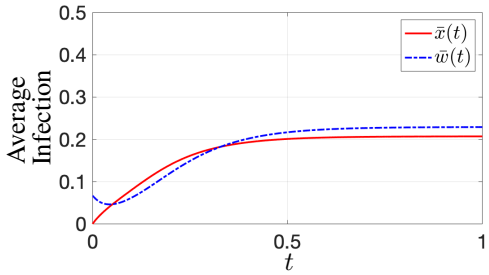

Fig. 5: Simulation only employing Assumption 1. We choose $D = 4I$, while the matrices B, B_w, and C_w are binary. We choose $[D_w]_{ij} = 100$ if $i = j = 15$; $[D_w]_{ij} = 0$, otherwise. Observe that, in contrast to the system in Fig. 4, for this simulation we allow some of the resource nodes to not be able to heal itself. Nonetheless, even this system converges to some endemic equilibrium, thus indicating that the claim in Theorem 2 could possibly be established under less restrictive assumptions on the healing rate.

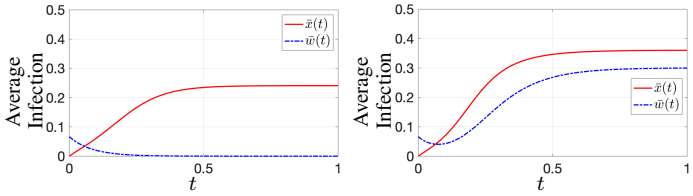

Fig. 6: Simulations without and with population-to-resource contact. On the left, $C_w = 0$, so the contamination of the resource nodes decays to zero. On the right, C_w is non-zero; thus the virus becomes endemic in the resource nodes as well.

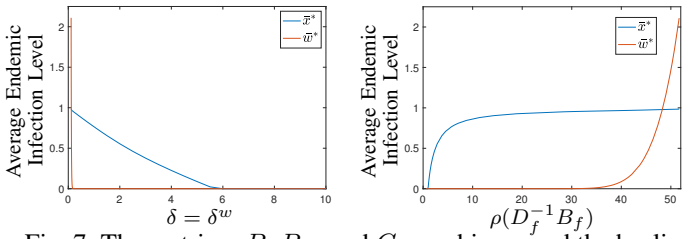

Fig. 7: The matrices B, B_w, and C_w are binary and the healing rates are equal and vary. We see how the equilibria of the system change as a function of the healing rate (left) and $\rho(D_f^{-1}B_f)$ (right).

VI. Conclusion

In this paper, we have developed a multi-network-dependent, continuous-time SIWS epidemic model, also referred to as a layered networked SIWS model. This model captures a networked system, which can be interpreted as individual people or multiple groups of individuals, coupled with an infrastructure network, which can be understood as a contaminated water (or some other utility) distribution network. We have analyzed the stability of the healthy state, both locally and globally. We compared the basic reproduction number of the model with the standard networked SIS model without a pathogen. We have established conditions for the existence, uniqueness, and stability of an endemic equilibrium. We have also provided a necessary and sufficient condition for the healthy state to be the only equilibrium of this model. Lastly, we have established conditions under which the initial infection levels of the shared resources could be recovered based on the measurements of the infection levels of the population.

One line of future investigation could focus on understanding the spread of diseases in infrastructure networks with time-varying topologies. Another problem of interest would be to develop control algorithms that exploit the topology of the infrastructure network for virus mitigation. Still on the topic of control of epidemics, it would be interesting to mitigate (resp. eradicate) epidemics subject to constraints on the availability of healing resources. Likewise, as mentioned previously, studying local weak observability under partial measurements remains a very interesting and challenging problem.

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APPENDIX

Proof of Prosition 2

To prove Proposition 2, we need the following lemma.

Lemma 3. *[19, Proposition 1] Suppose that N is an irreducible nonnegative matrix in $\mathbb{R}^{n\times n}$ and Λ is a negative diagonal matrix in $\mathbb{R}^{n\times n}$. Let $M = N + \Lambda$. 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 $\rho(-\Lambda^{-1}N) > 1$.*

Proof of Proposition 2: By Assumption 1, $\delta_j^w + \sum_k \alpha_{kj} > 0$ for all $j \in [m]$, D_f is invertible. From Lemma 3, the condition $\rho(D_f^{-1}B_f) < 1$ is equivalent to $s(B_f - D_f) < 0$, which implies that $J(\mathbf{0}, \mathbf{0})$ is a continuous-time stable matrix. Thus, by Lyapunov's indirect method the healthy state $(\mathbf{0}, \mathbf{0})$ of (6)-(7) is locally exponentially stable. □

Proof of Theorem 1

To prove the claim in Theorem 1, we need the following lemmas.

Lemma 4. *[36, Lemma 2.3] Suppose that M is an irreducible Metzler matrix. Then, $s(M)$ is a simple eigenvalue of M and there exists a unique (up to scalar multiple) vector $x \gg \mathbf{0}$ such that $Mx = s(M)x$.*

Lemma 5. *[37, Proposition 2] Suppose that M is an irreducible Metzler matrix such that $s(M) < 0$. Then, there exists a positive diagonal matrix P such that $M^\top P + PM \prec 0$.*

Lemma 6. *[18, Lemma A.1] Suppose that M is an irreducible Metzler matrix such that $s(M) = 0$. Then, there exists a positive diagonal matrix P such that $M^\top P + PM \preccurlyeq 0$.*

Lemma 7. *[35] Let $\tilde{x}$ be an equilibrium of $\dot{x}(t) = f(x(t))$ and $\mathcal{E} \subset \mathcal{D}$ be a bounded domain containing $\tilde{x}$. Let $V : \mathcal{E} \to \mathbb{R}$ be a continuously differentiable function such that $V(\tilde{x}) = 0$, $V(x) > 0$ in $\mathcal{E} \setminus \{\tilde{x}\}$, $\dot{V}(\tilde{x}) = 0$, and $\dot{V}(x) < 0$ in $\mathcal{E} \setminus \{\tilde{x}\}$. If $\mathcal{E}$ is an invariant set, then the equilibrium $\tilde{x}$ is asymptotically stable with the domain of attraction $\mathcal{E}$.*

Proof of Theorem 1: Recalling the notation in (8), we first consider the case when $\rho(D_f^{-1}B_f) < 1$. By Lemma 3, in this case, $s(B_f - D_f) < 0$. Since $(B_f - D_f)$ is an irreducible Metzler matrix, by Lemma 5, there exists a positive diagonal matrix P such that $(B_f - D_f)^\top P + P(B_f - D_f)$ is negative definite. Consider the Lyapunov function $V(z(t)) = z(t)^\top P z(t)$. Then, from (6)-(9), when $z(t) \neq \mathbf{0}$, we have

$$\begin{aligned}
\dot{V}(z(t)) &= 2z(t)^\top P \dot{z}(t) \\
&= 2z(t)^\top P(B_f - D_f)z(t) \\
&\quad + 2z(t)^\top P \begin{bmatrix} -X(t)B & -X(t)B_w \\ 0 & 0 \end{bmatrix} z(t) \\
&< -2z(t)^\top P \begin{bmatrix} X(t)B & X(t)B_w \\ 0 & 0 \end{bmatrix} z(t) \\
&\leq 0,
\end{aligned}$$

where the strict inequality holds by Lemma 5 since $2z(t)^\top P(B_f - D_f)z(t) = z(t)^\top (B_f - D_f)^\top P + P(B_f - D_f)z(t)$. Thus, in this case, $\dot{V}(z(t)) < 0$ if $z(t) \neq \mathbf{0}$. From Lemma 1 and Lemma 7, the healthy state is asymptotically stable with domain of attraction $\mathcal{D}$, with $\mathcal{D}$ given in (10).

Next we consider the case when $\rho(D_f^{-1}B_f) = 1$. By Lemma 3, $s(B_f - D_f) = 0$. Since $(B_f - D_f)$ is an

irreducible Metzler matrix, by Lemma 6, there exists a positive diagonal matrix Q such that $(B_f - D_f)^\top Q + Q(B_f - D_f)$ is negative semi-definite. Consider the Lyapunov function $V(z(t)) = z(t)^\top Q z(t)$. Then, from (6)-(9), we have

$$\begin{aligned}\dot{V}(z(t)) &= 2z(t)^\top Q(B_f - D_f)z(t) \\ &\quad + 2z(t)^\top Q \begin{bmatrix} -X(t)B & -X(t)B_w \\ 0 & 0 \end{bmatrix} z(t) \\ &\leq -2z(t)^\top Q \begin{bmatrix} X(t)B & X(t)B_w \\ 0 & 0 \end{bmatrix} z(t) \\ &= -2z(t)^\top \begin{bmatrix} Q_1 & 0 \\ 0 & Q_2 \end{bmatrix} \begin{bmatrix} X(t)B & X(t)B_w \\ 0 & 0 \end{bmatrix} z(t) \\ &= -2\left(x(t)^\top Q_1 X(t)Bx(t) + x(t)^\top Q_1 X(t)B_w w(t)\right) \\ &\leq 0,\end{aligned}$$

where Q_1 is the nth principal subarray of Q, which is an $n \times n$ positive diagonal matrix, and Q_2 is the $m \times m$ positive diagonal matrix that is composed of the rest of the block diagonal entries of Q. We claim that $\dot{V}(z(t)) < 0$ if $z(t) \neq \mathbf{0}$. To establish this claim, we first consider the case when $z(t) \gg \mathbf{0}$. Since B_f is irreducible and non-negative we have $B_f z(t) \gg \mathbf{0}$. As such, $Bx(t) + B_w w(t) \gg \mathbf{0}$, and due to Q_1 being a positive diagonal matrix, it follows that $x(t)^\top Q_1 X(t)(Bx(t) + B_w w(t)) > 0$. Thus, $\dot{V}(z(t)) < 0$.

Next we consider the case when $z(t) > \mathbf{0}$ and $z(t)$ has at least one zero entry. If $(B_f - D_f)^\top Q + Q(B_f - D_f)$ does not have an eigenvalue at zero, then $(B_f - D_f)^\top Q + Q(B_f - D_f)$ is negative definite, which implies that $z(t)^\top \left((B_f - D_f)^\top Q + Q(B_f - D_f)\right) z(t) < 0$ when $z(t) > \mathbf{0}$ and, thus, in this case,

$$\begin{aligned}\dot{V}(z(t)) &= 2z(t)^\top Q(B_f - D_f)z(t) \\ &\quad + 2z(t)^\top Q \begin{bmatrix} -X(t)B & -X(t)B_w \\ 0 & 0 \end{bmatrix} z(t) \\ &\leq 2z(t)^\top Q(B_f - D_f)z(t) < 0.\end{aligned}$$

Now suppose that $(B_f - D_f)^\top Q + Q(B_f - D_f)$ has an eigenvalue at zero. Since $(B_f - D_f)$ is an irreducible Metzler matrix and Q is a positive diagonal matrix, $(B_f - D_f)^\top Q + Q(B_f - D_f)$ is a symmetric irreducible Metzler matrix. Since $(B_f - D_f)^\top Q + Q(B_f - D_f)$ is negative semi-definite, $s((B_f - D_f)^\top Q + Q(B_f - D_f)) = 0$. By Lemma 4, zero is a simple eigenvalue of $(B_f - D_f)^\top Q + Q(B_f - D_f)$ and it has a unique (up to scalar multiple) strictly positive eigenvector corresponding to the eigenvalue zero. Thus, $z(t)^\top \left((B_f - D_f)^\top Q + Q(B_f - D_f)\right) z(t) < 0$ when $z(t) > \mathbf{0}$ and $z(t)$ has at least one zero entry (because the only vector for which it equals zero is the strictly positive eigenvector). Therefore, $\dot{V}(z(t)) < 0$ if $z(t) \neq \mathbf{0}$. From Lemma 1 and Lemma 7, the healthy state is asymptotically stable with domain of attraction $\mathcal{D}$, with $\mathcal{D}$ given in (10). □

Proof of Theorem 2

To prove the claim in Theorem 2, we will be making use of the following variant of the Perron-Frobenius theorem for irreducible matrices.

Lemma 8. *[38, Chapter 8.3] [36, Theorem 2.7]Suppose that N is an irreducible nonnegative matrix. Then,*

(i) $r = \rho(N)$ is a simple eigenvalue of N.
(ii) There is an eigenvector $\zeta \gg \mathbf{0}$ corresponding to the eigenvalue r.
(iii) $x > \mathbf{0}$ is an eigenvector only if $Nx = rx$ and $x \gg \mathbf{0}$.

Proof of Theorem 2: The proof is split in three parts: First we show existence of an endemic equilibrium provided the conditions in Theorem 2 are satisfied. Subsequently, we show that this equilibrium is unique, and that for all non-zero initial conditions the dynamics converge asymptotically to this equilibrium.

Part 1 -Proof of existence

Note that if $z \geq \mathbf{0}$, $\mathrm{diag}(D_f^{-1}B_f z)$ is a nonnegative diagonal matrix, and therefore the inverse of $(I + \mathrm{diag}(D_f^{-1}B_f z))$ exists. Define a map $T(z) : \mathbb{R}_+^{n+m} \to \mathbb{R}_+^{n+m}$ such that

$$T(z) = (I + \mathrm{diag}(D_f^{-1}B_f z))^{-1}(D_f^{-1}B_f z + \mathrm{diag}(D_f^{-1}B_f z)\begin{bmatrix}\mathbf{0} \\ w\end{bmatrix}).$$

Observe that the components of $T(y)$ are

$$T_i(z) = \frac{(D_f^{-1}B_f z)_i}{1 + (D_f^{-1}B_f z)_i}, \text{ for } i \in [n],$$

$$T_j(z) = \frac{(D_f^{-1}B_f z)_j z_j + (D_f^{-1}B_f z)_j}{1 + (D_f^{-1}B_f z)_j}, \text{ for } j \in [n+m]\backslash[n].$$

Note that the scalar function $s/(1+s)$ is increasing in s, and that $D_f^{-1}B_f$ is a nonnegative matrix. Therefore, $v \geq z$ implies $T(v) \geq T(z)$. Notice that a fixed point of $T(z)$ fulfills

$$z = (I + \mathrm{diag}(D_f^{-1}B_f z))^{-1}(D_f^{-1}B_f z + \mathrm{diag}(D_f^{-1}B_f z)\begin{bmatrix}\mathbf{0} \\ w\end{bmatrix}). \tag{19}$$

Multiplying (19) by $(I + \mathrm{diag}(D_f^{-1}B_f z))$ gives us

$$D_f^{-1}B_f z + \mathrm{diag}(D_f^{-1}B_f z)\begin{bmatrix}\mathbf{0} \\ w\end{bmatrix} = (I + \mathrm{diag}(D_f^{-1}B_f z))z. \tag{20}$$

Using the identity $\mathrm{diag}(u)v = \mathrm{diag}(v)u$, (20) is equivalent to

$$D_f^{-1}B_f z + \mathrm{diag}(\begin{bmatrix}\mathbf{0} \\ w\end{bmatrix})D_f^{-1}B_f z = (I + \mathrm{diag}(z)D_f^{-1}B_f)z. \tag{21}$$

Recall that the definition of $X(z)$ means that subtracting $\mathrm{diag}(\begin{bmatrix}\mathbf{0} \\ w\end{bmatrix})D_f^{-1}B_f z$ from (21) yields

$$D_f^{-1}B_f z = (I + X(z)D_f^{-1}B_f)z. \tag{22}$$

Since $X(z)$ and D_f^{-1} are diagonal matrices, they commute. Furthermore, by pre-multiplying (22) with D_f, and suitably rearranging terms, we obtain

$$(-D_f + (I - X(z))B_f)z = 0. \tag{23}$$

A solution of equation (23) is clearly an equilibrium of (9). As such, it suffices to show that $T(z)$ has a fixed point $\tilde{z} \gg \mathbf{0}$. We will now show that at least one such fixed point exists.

We have $\rho(D_f^{-1}B_f) > 1$. Note that $D_f^{-1}B_f$ is an irreducible nonnegative matrix. Hence, by Lemma 4, $\lambda^* = \rho(D_f^{-1}B_f)$

is a simple eigenvalue of $D_f^{-1}B_f$ and the eigenspace of λ^* is spanned by a vector $z^* \gg \mathbf{0}$. Then, since $\lambda^* > 1$, there exists some $\epsilon > 0$ such that, for all $i \in [n+m]$, we have $\epsilon z_i^* \leq (\lambda^* - 1)/\lambda^*$, which implies that $1 \leq \lambda^*/(1 + \lambda^* \epsilon z_i^*)$. Hence, $\epsilon z_i^* \leq \lambda^* \epsilon z_i^*/(1 + \lambda^* \epsilon z_i^*)$, and thus

$$\epsilon z_i^* \leq \frac{(D_f^{-1}B_f \epsilon z^*)_i}{1 + (D_f^{-1}B_f \epsilon z^*)_i}, \text{ for all } i \in [n]. \tag{24}$$

Noting that $(D_f^{-1}B_f \epsilon z^*)_j \epsilon z_j^* > 0$ for all $j \in [n+m]\backslash[n]$, we also have

$$\epsilon z_j^* \leq \frac{(D_f^{-1}B_f \epsilon z^*)_j \epsilon z_j^* + (D_f^{-1}B_f \epsilon z^*)_j}{1 + (D_f^{-1}B_f \epsilon z^*)_j}, \tag{25}$$

for all $j \in [n+m]\backslash[n]$. Due to the inequalities (24) and (25), we have $T(\epsilon z^*) \geq \epsilon z^*$. Since $z \geq r$ implies $T(z) \geq T(r)$, it follows that for any $z \geq \epsilon z^*$ we have $T(z) \geq \epsilon z^*$. Define the vector

$$\mathbf{z} := \begin{bmatrix} \mathbf{1} \\ \mathbf{w} \end{bmatrix},$$

where $\mathbf{w} := -(A_w - D_w)^{-1} C_w \mathbf{1}$. Note that $(A_w - D_w)$ is invertible because of it being diagonally dominant. Consider $T_i(\mathbf{z})$ for $i \in [n]$ while noting that $s/(1+s)$ is bounded from above by 1 for any positive s. Then

$$T_i(\mathbf{z}) = \frac{(D_f^{-1}B_f \mathbf{z})_i}{1 + (D_f^{-1}B_f \mathbf{z})_i} \leq 1, \text{ for all } i \in [n]. \tag{26}$$

Before considering $T_j(\mathbf{z})$ for $j \in [n+m]\backslash[n]$, first note that

$$\begin{aligned}
&(D_w - \mathrm{diag}(A_w))^{-1} \begin{bmatrix} C_w & A_w - \mathrm{diag}(A_w) \end{bmatrix} \begin{bmatrix} \mathbf{1} \\ \mathbf{w} \end{bmatrix} \\
&= (D_w - \mathrm{diag}(A_w))^{-1} C_w \mathbf{1} - (D_w - \mathrm{diag}(A_w))^{-1} \\
&\qquad \times (A_w - \mathrm{diag}(A_w))(A_w - D_w)^{-1} C_w \mathbf{1} \\
&= (D_w - \mathrm{diag}(A_w))^{-1} (I - (A_w - \mathrm{diag}(A_w))(A_w - D_w)^{-1}) \\
&\qquad \times C_w \mathbf{1} \\
&= (D_w - \mathrm{diag}(A_w))^{-1} ((A_w - D_w) - (A_w - \mathrm{diag}(A_w))) \\
&\qquad \times (A_w - D_w)^{-1} C_w \mathbf{1} \\
&= -(D_w - \mathrm{diag}(A_w))^{-1} (D_w - \mathrm{diag}(A_w))(A_w - D_w)^{-1} C_w \mathbf{1} \\
&= -(A_w - D_w)^{-1} C_w \mathbf{1} \\
&= \mathbf{w}.
\end{aligned}$$

Hence,

$$T_j(\mathbf{z}) = \frac{\mathbf{z}_j(1 + \mathbf{z}_j)}{1 + \mathbf{z}_j} = \mathbf{z}_j, \text{ for all } j \in [n+m]\backslash[n]. \tag{27}$$

Due to (26) and (27), we have $T(\mathbf{z}) \leq \mathbf{z}$. Since $v \geq w$ implies $T(v) \geq T(w)$, it follows that $T(z) \leq \mathbf{z}$ if $z \leq \mathbf{z}$. By Brouwer's fixed-point theorem, there is at least one fixed point of $T(z)$ in the domain $\{z : \epsilon z^* \leq z \leq \mathbf{z}\}$. In conclusion, the map $T(z)$ has at least one fixed point in the domain $\{z : \epsilon z^* \leq z \ll \mathbf{z}\}$, and therefore (9) has at least one equilibrium $\tilde{z} \gg \mathbf{0}$. □

Part 2 – Proof of uniqueness

We will now prove that the endemic equilibrium is unique. Suppose that there are two endemic equilibria, $\tilde{z}$ and $\tilde{\mathbf{z}}$. Note that, by similar arguments as in [21, Lemma 6], $\tilde{z} \gg \mathbf{0}$ and $\tilde{\mathbf{z}} \gg \mathbf{0}$. Let $\kappa = \max_{i \in [n+m]} \tilde{z}_i / \tilde{\mathbf{z}}_i$. It turns out that κ is given by

$$\kappa = \max_{i \in [n]} \tilde{z}_i / \tilde{\mathbf{z}}_i. \tag{28}$$

To see this, assume by way of contradiction that $\kappa = \tilde{z}_{n+j}/\tilde{\mathbf{z}}_{n+j}$ for some $j \in [m]$, and thus $\kappa > \tilde{z}_i/\tilde{\mathbf{z}}_i$, for all $i \in [n]$. Since both $\tilde{z}$ and $\tilde{\mathbf{z}}$ are equilibria of system (9), it follows that, for each $j \in [m]$

$$\begin{aligned}
\tilde{z}_{n+j} &= \textstyle\sum_i^n c_{ji} \tilde{x}_i + \sum_{k,k \neq j}^m \alpha_{kj} \tilde{w}_k, \\
\tilde{\mathbf{z}}_{n+j} &= \textstyle\sum_i^n c_{ji} \tilde{\mathbf{x}}_i + \sum_{k,k \neq j}^m \alpha_{kj} \tilde{\mathbf{w}}_k.
\end{aligned} \tag{29}$$

Since we have that $\kappa > \tilde{z}_i/\tilde{\mathbf{z}}_i$, for all $i \in [n]$, then $\kappa \tilde{\mathbf{z}}_i > \tilde{z}_i$, for all $i \in [n]$. Since by assumption $\kappa = \tilde{z}_{n+j}/\tilde{\mathbf{z}}_{n+j}$ for some $j \in [m]$, it follows that, for each $k \in [m]$, $\tilde{z}_{n+k} \leq \kappa \tilde{\mathbf{z}}_{n+k}$. Then, (29) yields

$$\begin{aligned}
\tilde{z}_{n+j} &= \textstyle\sum_i^n c_{ji}^w \tilde{x}_i + \sum_{k,k \neq j}^m \alpha_{kj} \tilde{w}_k \\
&< \kappa \textstyle\sum_i^n c_{ji}^w \tilde{\mathbf{x}}_i + \sum_{k,k \neq j}^m \alpha_{kj} \tilde{\mathbf{w}}_k \\
&= \kappa \tilde{\mathbf{z}}_{n+j}.
\end{aligned}$$

Hence, for all $j \in [m]$, $\kappa > \tilde{z}_{n+j}/\tilde{\mathbf{z}}_{n+j}$, which contradicts the assumption that $\kappa = \tilde{z}_{n+j}/\tilde{\mathbf{z}}_{n+j}$, for some $j \in [m]$. Therefore, κ must be given by (28). Now, by (28) we know that $\tilde{z} \leq \kappa \tilde{\mathbf{z}}$. For some $j \in [n]$ we have $\tilde{z}_j = \kappa \tilde{\mathbf{z}}_j$. Assume, by way of contradiction, that $\kappa > 1$. Then, since an equilibrium of (9) also constitutes a fixed point of $T(z)$, we have

$$\begin{aligned}
\tilde{z}_j &= (D_f^{-1}B_f \tilde{z})_j / (1 + (D_f^{-1}B_f \tilde{z})_j) \\
&\leq (D_f^{-1}B_f \kappa \tilde{\mathbf{z}})_j / (1 + (D_f^{-1}B_f \kappa \tilde{\mathbf{z}})_j) && (30) \\
&< \kappa (D_f^{-1}B_f \tilde{\mathbf{z}})_j / (1 + (D_f^{-1}B_w \tilde{\mathbf{z}})_j) && (31) \\
&= \kappa \tilde{\mathbf{z}}_j && (32) \\
&= \tilde{z}_j, && (33)
\end{aligned}$$

where (30) follows from $\tilde{z} \leq \kappa \tilde{\mathbf{z}}$ and that $T(v) \geq T(w)$ whenever $v \geq w$, (31) follows from the assumption $\kappa > 1$, and (32) follows from the fact that $\tilde{\mathbf{z}}$ is an equilibrium of (9). Note that (33) is a contradiction, following from our assumption that $\kappa > 1$. Hence, $\kappa \leq 1$, meaning that $\tilde{z} \leq \tilde{\mathbf{z}}$. Switching the roles of $\tilde{z}$ and $\tilde{\mathbf{z}}$, we see that $\tilde{\mathbf{z}} \leq \tilde{z}$. Therefore, $\tilde{z} = \tilde{\mathbf{z}}$, and thus the equilibrium is unique.

Part 3– Proof of asymptotic convergence: The proof of asymptotic convergence is quite similar to that of [21, Theorem 3, part 3], and is, in the interest of space, omitted here. For details, please see proof of [39, Theorem 2].

Proof of Proposition 5

We need the following result to proceed ahead.

Lemma 9. *Consider system* (15) *under Assumption 3. If* $\rho(D^{-1}B) > 1$*, then there exists a unique endemic equilibrium* $\hat{x}$ *such that* $\mathbf{0} \ll \hat{x} \ll \mathbf{1}$.

Proof: The result follows by particularizing [21, Theorem 3] for the networked SIS model. □

Proof of Proposition 5: By assumption, the matrices B_f and B are irreducible. Moreover, $\rho(D_f^{-1}B_f) > 1$, and $\rho(D^{-1}B) > 1$. Therefore, from Theorem 2, and from Lemma 9, we know that there exists a unique endemic equilibrium $\tilde{z} = [\begin{smallmatrix} \tilde{x} \\ \tilde{w} \end{smallmatrix}]$ for (9), and a unique endemic equilibrium

$\hat{x}$ for (15), respectively. Moreover, $\mathbf{0} \ll \hat{x} \ll \mathbf{1}$.
Note that since $\hat{x}$ is an equilibrium of (15), we have

$$(I - \hat{X})D^{-1}B\hat{x} = \hat{x}. \tag{34}$$

Consider a solution $z(t) = (x(t), w(t))$ to (9) for $t \geq 0$, with $x_i(0) \in [0, 1]$ and $w_j(0) \geq 0$ for all $i \in [n]$, $j \in [m]$. By Lemma 1 we have $x_i(t) \in [0, 1]$ and $w_j(t) \geq 0$ for all $i \in [n]$, $j \in [m]$ and $t \geq 0$. Suppose that, for some $t \geq 0$, $x(t) \geq \hat{x}$, with $x_i(t) = \hat{x}_i$ for some $i \in [n]$. Then

$$\begin{aligned}
\dot{x}_i(t) &= (1 - x_i(t))(D_f^{-1}B_f z(t))_i - x_i(t) \\
&= (1 - \hat{x}_i)(D^{-1}Bx(t) + D^{-1}B_w w(t))_i - \hat{x}_i \\
&\geq (1 - \hat{x}_i)(D^{-1}B\hat{x})_i - \hat{x}_i \qquad (35) \\
&= 0, \qquad (36)
\end{aligned}$$

where (35) follows from $D^{-1}B_w w(t) \geq \mathbf{0}$, and (36) follows from (34). Since the same argument holds for any t and $i \in [n]$ we have $x(t) \geq \hat{x}$ for all $t \geq 0$ if $x(0) \geq \hat{x}$. Furthermore, due to (a): $\hat{x} \ll \mathbf{1}$, and (b): $\hat{z}$ being the unique equilibrium of (9) with a region of attraction including $\{z = (x, w) : \mathbf{1} \geq x \geq \hat{x}\}$, we must have $\tilde{x} \geq \hat{x}$. In order to show $\tilde{x} \neq \hat{x}$, assume by way of contradiction that $\tilde{x} = \hat{x}$. Note that

$$\tilde{x} = (I - \tilde{X})(D^{-1}B\tilde{x} + D^{-1}B_w\tilde{w}). \tag{37}$$

With the assumption that $\tilde{x} = \hat{x}$, (37) is equivalent to

$$\begin{aligned}
\hat{x} &= (I - \hat{X})(D^{-1}B\hat{x} + D^{-1}B_w\tilde{w}) \\
&< (I - \hat{X})D^{-1}B\hat{x} \qquad (38) \\
&= \hat{x}, \qquad (39)
\end{aligned}$$

where (38) is due to the following: (i): $D_f^{-1}B_f$ is an irreducible Metzler matrix, (ii): $\tilde{w} \gg \mathbf{0}$, and (iii): $\hat{x} \ll \mathbf{1}$, so we have $(I - \hat{X})D^{-1}B_w\tilde{w} > \mathbf{0}$. Moreover, (39) follows from (34). Clearly, (39) is a contradiction, and therefore $\tilde{x} > \hat{x}$. □

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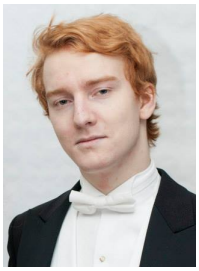

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