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Multi-Layer Disease Spread Model with a Water Distribution Network

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Abstract— **This paper proposes a layered networked SIWS (Susceptible-Infected-Water-Susceptible) model, for an SIS-type waterborne disease spreading over a human contact network connected to a water distribution network that has a pathogen spreading in it. Conditions for local and global stability of the healthy state, where no one is sick and the water network is not contaminated, are provided. We also pose an observability problem, and show under certain conditions if you observe some of the human contact network you can recover the pathogen levels in the water network.**

I. Introduction

Diseases and other health problems have been caused by waterborne pathogens worldwide [1]. These pathogens spread through the water distribution systems (e.g., rivers, groundwater, and reservoirs) [2]. While water quality issues are very prevalent in developing countries with less advanced plumbing and sewage infrastructure, there are still problems in more 'developed' countries. 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 [3].

Recent work has considered adding a water compartment in traditional epidemic models. In [4], a SIWR (Susceptible-Infected-Water-Removed) model was proposed by adding a water compartment W in the classical SIR model. Extending this work, [5] proposed a cholera model that considers infection via a water compartment through both direct and indirect disease transmission pathways. In [6], the model from [4] is extended/modified by proposing a multi-group SIWS model, that is, there adding network-dependence in the human contact network but with only a single water compartment. In [7], the model from [4] is extended by proposing a network-dependent SIWR model, which, similarly, includes a human-contact network and a single water component that may be contaminated.

The above discussion considers the spread of waterborne pathogens through natural causes. However, the source of the outbreak could be the result of an attack by an adversary. Vulnerabilities of water networks have been highlighted from a cyber-physical systems perspective in the literature [8]–[10]. If an adversary attacks the water network of a city one would want to minimize the effect. Regardless of the source of the outbreak, observing the source of the contamination of the water so that the population can be protected as soon as possible is of utmost importance.

In this paper, we propose an extension of the networked SIWS model from [6] by adding a water distribution network, where, in addition to person-person and person-water-person transmissions, there exists water-water transmission of the pathogen, and thus we call it the layered networked SIWS model. There are two interpretations of networked SIS (Susceptible-Infected-Susceptible) models: 1) each node in the network is a group of fully connected individuals and the corresponding state variable represents the proportion of infected individuals in the corresponding group, or 2) each node is a single agent and the corresponding state variable represents the probability of the corresponding agent being infected. Both interpretations require the state variables to take values between zero and one. In this paper we study the epidemic spreading of a waterborne disease over multiple groups of individuals, mainly households, and therefore we employ the first interpretation.

Networked SIS spread models have been studied quite widely by the controls community in recent years [11], [12]. Recently it has been shown that these models capture real spread behavior in at least some applications [13], [14]. There has also been an interesting thread of research looking at competing/coupled viruses spreading on layered networks [15]–[17]. In addition to layered spread networks, there has also been layered network models proposed that explore the spread of products (modeled as an SIS process) coupled with a consensus, opinion dynamics, network model [18], [19]. To the best of our knowledge this is the first layered networked model comprised of a networked SIS spread component coupled with a water distribution network.

We use this model to formulate an interesting observability problem. If there is a water pathogen spreading in the water network but no sensors for detecting it, due to high costs, can you recover the water infection levels in the water distribution network by only measuring the level of sickness in the population? To the best of our knowledge the only work that has explored observability of SIS spread models is in [20], where the authors employ an empirical gramian approach.

The contributions of this paper are two fold: 1) we derive and analyze the layered networked SIWS model, and 2) we are interested in observing the spread of the pathogen and disease in the networks. More formally, we study the observability problem on this nonlinear system. Due to space limitations, some of the proofs are omitted in this version.

Notation: For any positive integer n, we use $[n]$ to denote the set $\{1, 2, \ldots, n\}$. We use $A^\top$ for the transpose of a matrix

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A. 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, while the sizes of the vectors and matrix are to be understood from the context. 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., $s(M) = \max\{\mathrm{Re}(\lambda) \ : \ \lambda \in \sigma(M)\}$.

II. The Model

In this section, we propose a distributed continuous-time waterborne pathogen model, called the layered networked SIWS model depicted in Figure 1.

Consider an SIS-type waterborne pathogen spreading over a two-layer network consisting of $n > 1$ groups of individuals and $m > 1$ water compartments which can be contaminated by infected individuals shedding the pathogen into them. We assume that the water compartments are reservoir-like water systems with homogeneous water quality and an instantaneous pathogen diffusion processes. An individual can be infected either by coming into contact with contaminated water or infected individuals.

Let $I_i(t)$ and $S_i(t)$ respectively denote the number of infected and susceptible individuals in group i at time $t \geq 0$. We assume that the total number of individuals in each group i, denoted by N_i, does not change over time, i.e., $S_i(t) + I_i(t) = N_i$ for all $i \in [n]$ and $t \geq 0$, which implies that the birth and death rates for each group are equal. This assumption simplifies the model and has been adopted in [21]. We associate with each group i curing rate γ_i, birth rate μ_i, death rate $\bar{\mu}_i$ (which equals μ_i), person-to-person infection rates a_{ij} and water-to-person infection rates a^w_{ij}. It is assumed that individuals are susceptible at birth even if their parents are infected. The evolution of the numbers of infected and susceptible individuals in each group i is as follows (which follows the ideas in [21] and [4]):

$$\begin{aligned}\dot{S}_i(t) &= \mu_i N_i - \bar{\mu}_i S_i(t) + \gamma_i I_i(t) - \sum_{j=1}^{n} a_{ij} \frac{S_i(t)}{N_i} I_j(t) \\ &\quad - \sum_{j=1}^{m} a^w_{ij} w_j(t) S_i(t) \\ &= (\mu_i + \gamma_i) I_i(t) - \sum_{j=1}^{n} a_{ij} \frac{S_i(t)}{N_i} I_j(t) \\ &\quad - \sum_{j=1}^{m} a^w_{ij} w_j(t) S_i(t), \qquad (1)\end{aligned}$$

$$\dot{I}_i(t) = -\gamma_i I_i(t) - \bar{\mu}_i I_i(t) + \sum_{j=1}^{n} a_{ij} \frac{S_i(t)}{N_i} I_j(t) + \sum_{j=1}^{m} \alpha^w_{ij} w_j(t) S_i(t)$$

$$\begin{aligned}&= (-\gamma_i - \mu_i) I_i(t) + \sum_{j=1}^{n} a_{ij} \frac{S_i(t)}{N_i} I_j(t) \\ &\quad + \sum_{j=1}^{m} a^w_{ij} w_j(t) S_i(t), \qquad (3)\end{aligned}$$

where $w_j(t)$ denotes the pathogen concentration in the jth water reservoir and evolves as

$$\dot{w}_j = -\delta^w_j w_j + \sum_{k=1}^{n} \zeta^w_{jk} I_k + \sum_{k=1}^{m} \alpha_{kj} w_k - w_j \sum_{k=1}^{m} \alpha_{jk}, \quad (4)$$

where δ^w_j denotes the decay rate of the pathogen in the water, ζ^w_{jk} denotes the person-water contact rate of group k to water node j, and α_{kj} represents the flow of the pathogen from node k to node j in the water network. It is easy to check from (1) and (3) that $\dot{S}_i(t) + \dot{I}_i(t) = 0$, which is consistent with the assumption that N_i is a constant.

To simplify the model, we define the portion of infected individuals in each group i by

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

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), (3), and (4), it follows that

$$\dot{x}_i = -\delta_i x_i + (1 - x_i) \left(\sum_{j=1}^{n} \beta_{ij} x_j + \sum_{j=1}^{m} \beta^w_{ij} w_j \right) \quad (5)$$

$$\dot{w}_j = -\delta^w_j w_j + \sum_{k=1}^{m} \alpha_{kj} w_k - w_j \sum_{k=1}^{m} \alpha_{jk} + \sum_{k=1}^{n} c^w_{jk} x_k, \quad (6)$$

Note that depending on the pathogen δ^w_j may be zero. Note also that if there is only one node in the water network, the model reduces to a slightly more general version of the model proposed in [6]. Finally if $w_j(t) = 0$ for all t and all $j \in [m]$, or equivalently, there is no water distribution network, the model reduces to the regular networked SIS model [11].

We impose the following assumptions on the system parameters.

Assumption 1. *Suppose that $\delta_i > 0$ for all $i \in [n]$, $\delta^w_j \neq \sum_k \alpha_{kj}$ 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.*

The model from (5)-(6) in vector form becomes:

$$\dot{x} = (B - XB - D)x + (I - X)B_w w \quad (7)$$

$$\dot{w} = -D_w w + A_w w + C_w x, \quad (8)$$

where $B = [\beta_{ij}]_{n \times n}$, $X = \mathrm{diag}(x)$, $B_w = [\beta^w_{ij}]_{n \times m}$, 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.

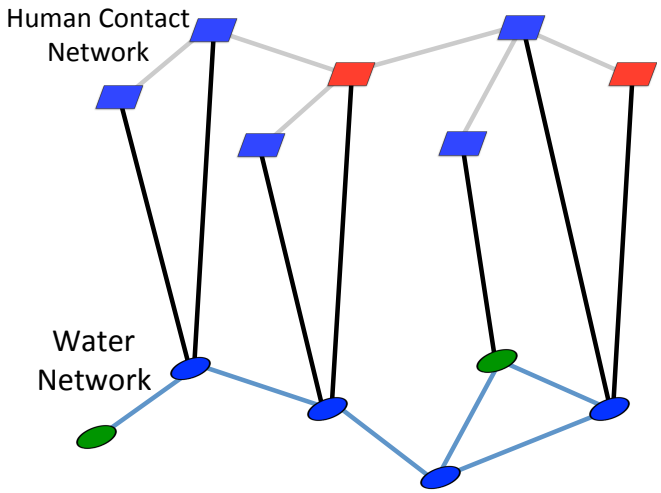


Fig. 1: Multi-layered SIWS model: The disease (depicted by red) spreads between household nodes (squares) through the human contact network and the pathogen (green) spreads through water network (nodes depicted by circles. Blue indicates healthy and the model allows for transmission from the water network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

Since each x_i represents the proportion of infected individuals in group i, it is natural to assume that the initial value of x_i is in $[0,1]$, or the value of x_i will lack physical meaning of the epidemic model considered here. Similarly, it is also natural to assume that the initial value of w_j is nonnegative.

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 (6) 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 (5) and Assumption 1, $\dot{x}_i(\tau) \geq 0$. If $x_i(\tau) = 1$, then again from (5) 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 $\tau = 0$. ∎

In the next section we solve the following problem.

Problem 1. *Given the model in (5)-(6), find conditions for convergence to the healthy state, that is, where all nodes in both the human contact network and the water distribution network are healthy.*

III. Analysis

In this section we analysis the new proposed model both locally and globally to solve Problem 1.

A. Local Stability of the Healthy State

Consider $(\tilde{x}, \tilde{w})$, an equilibrium of (7)-(8). The Jacobian matrix of the equilibrium, denoted by $J(\tilde{x}, \tilde{w})$, is

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

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

$$\tilde{X} = \text{diag}\left\{\tilde{x}_1, \tilde{x}_2, \cdots, \tilde{x}_n\right\},$$

$$F_1 = \text{diag}\left\{\sum_{j=1}^{n} \beta_{1j}\tilde{x}_j, \sum_{j=1}^{n} \beta_{2j}\tilde{x}_j, \cdots, \sum_{j=1}^{n} \beta_{nj}\tilde{x}_j\right\},$$

$$F_2 = \text{diag}\left\{\sum_{j=1}^{n} \beta^w_{1j}\tilde{w}_j, \sum_{j=1}^{n} \beta^w_{2j}\tilde{w}_j, \cdots, \sum_{j=1}^{n} \beta^w_{nj}\tilde{w}_j\right\}.$$

In the case when $\tilde{x} = \mathbf{0}$ and $\tilde{w} = \mathbf{0}$, i.e., at the healthy state,

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

If either $B_w = 0$ or $C_w = 0$, i.e., the water does not affect the population or humans will not contaminate the water 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 (7)-(8) 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 [22] the healthy state $(\mathbf{0}, \mathbf{0})$ of (5)-(6) is locally exponentially stable. ∎

Note that $J(\mathbf{0}, \mathbf{0}) = B_f - D_f$ where

$$B_f = \begin{bmatrix} B & B_w \\ C_w & A_w - \text{diag}(A_w) \end{bmatrix}, \tag{9}$$

$$D_f = \begin{bmatrix} D & 0 \\ 0 & D_w - \text{diag}(A_w) \end{bmatrix}. \tag{10}$$

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 (7)-(8) is locally exponentially stable.*

To prove the proposition, we need the following lemma.

Lemma 2. [Proposition 1 in [17]] *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: From Lemma 2, 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 (5)-(6) is locally exponentially stable. ■

B. Global Stability of the Healthy State

To state our 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 x^* be an equilibrium of the system and $\mathcal{E} \subset \mathcal{D}$ be a domain containing x^*. The equilibrium x^* is called asymptotically stable with the domain of attraction $\mathcal{E}$ if for any $x(0) \in \mathcal{E}$, there holds $\lim_{t\to\infty} x(t) = x^*$.

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 $(\mathbf{0}, \mathbf{0})$ of (7)-(8) is asymptotically stable with the domain of attraction $x \in [0, 1]^n$ and $w \in [0, \infty)^n$.*

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 compartments, it is 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 [21].

For the layered networked SIWS model (7)-(8), 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. Extensive simulations (some of which are included in Section V) show that when $\rho(D_f^{-1}B_f) > 1$, the model has an epidemic equilibrium. Therefore, we call $\rho(D_f^{-1}B_f)$ the basic reproduction number of the layered networked SIWS model (7)-(8), 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

$$\begin{aligned} D_f^{-1}B_f &= \begin{bmatrix} D^{-1} & 0 \\ 0 & (D_w - \text{diag}(A_w))^{-1} \end{bmatrix} \\ &\qquad \times \begin{bmatrix} B & B_w \\ C_w & A_w - \text{diag}(A_w) \end{bmatrix} \\ &= \begin{bmatrix} D^{-1}B & D^{-1}B_w \\ (D_w - \text{diag}(A_w))^{-1}C_w & F_3 \end{bmatrix}, \end{aligned}$$

where $F_3 = (D_w - \text{diag}(A_w))^{-1}A_w - \text{diag}(A_w)$. We need the following lemma.

Lemma 3. [Lemma 2.6 in [23]] *Suppose that N is an irreducible nonnegative matrix. If M is a principal square submatrix of N, then $\rho(M) < \rho(N)$.*

Since $D_w^{-1}B_w$ is an irreducible nonnegative matrix by Assumption 1, $\rho(D_w^{-1}B_w) > \rho(D^{-1}B)$. Therefore we have the following result.

Proposition 3. *Suppose that Assumption 1 holds. Then, the basic reproduction number of the layered networked SIWS model (7)-(8) is greater than that of the networked SIS model.*

The proposition implies that the water distribution network makes the system more vulnerable to SIS-type diseases.

IV. Observability Problem

The problem we want to solve is motivated by the question of when, if you do not have sensors to detect waterborne pathogens in your water network, can you use measurements of the sickness level of people, or households, who drink the water to discover the contamination level of the water, or the source of the contamination (the initial condition of the water pathogen levels). We introduce the following notation:

$$y = Cx, \tag{11}$$

where C is a measurement matrix.

Problem 2. *Given B, D, A_w, B_w, C_w, D_w, C, and measurements y, find conditions for when $w(0)$ can be recovered.*

We derive conditions for when the system is locally weakly observable appealing to the rank of the Jacobian of the Lie derivatives applying the results from [24]. Consequently, here are the Lie derivative calculations:

$$\begin{aligned} y &= Cx \\ \dot{y} &= C\dot{x} = C[\underbrace{(B - XB - D)}_{F_x}x + \underbrace{(I - X)B_w}_{F_w}w] \\ \ddot{y} &= C\ddot{x} = C[F_x\dot{x} + F_w\dot{w} - \dot{X}(Bx + B_w w)] \\ y^{(3)} &= Cx^{(3)} = C[F_x\ddot{x} + F_w\ddot{w} - \ddot{X}(Bx + B_w w) \\ &\qquad - 2\dot{X}(B\dot{x} + B_w\dot{w})] \\ &\vdots \\ y^{(m+n)} &= Cx^{(m+n)} = C[F_x x^{(m+n-1)} + F_w w^{(m+n-1)} \\ &\qquad - X^{(m+n-1)}(Bx + B_w w) - \cdots], \end{aligned}$$

where $\dot{x}$ and $\dot{w}$ are defined in (7) and (8),

$$\begin{aligned} \ddot{w} &= (A_w - D_w)\dot{w} + C_w\dot{x}, \\ \dot{X} &= \text{diag}(\dot{x}), \\ \ddot{X} &= (\tilde{B}_x + \tilde{B}_w)(F_x - \tilde{B}_{xw}), \end{aligned}$$

with $\tilde{B}_x = \text{diag}(Bx)$ and $\tilde{B}_w = \text{diag}(B_w w)$.

We explore the case when we assume that all nodes in the human contact network are initially healthy, that is, $x(0) = \mathbf{0}$. Therefore, we explore the Jacobian of the above Lie derivatives evaluated at $x(0) = \mathbf{0}$, called $\mathcal{O}$,

$$\begin{bmatrix} C & 0 \\ C\underbrace{[F_{x_0} - \tilde{B}_w]}_{X_x} & CB_w \\ C\underbrace{[X_x^2 - \tilde{B}_w B - \tilde{B}]}_{X_{xx}} & C\underbrace{[B_w F_{w_0} + X_x B_w - \tilde{B}_w B_w]}_{W_w} \\ C[F_{x_0} X_{xx} - \tilde{D}B - \tilde{D}_w & C[B_w F_{w_0}^2 + X_x W_w - 2\tilde{B}_w^2 B_w \\ -2\tilde{B}_w^2 B - 2\tilde{B}_w B X_x & -\tilde{B}_w(B_w F_{w_0} + B B_w) \\ -2\tilde{B}X_x - \mathrm{diag}(BB_w w) & -2\tilde{B}W_w - \tilde{D}B_w + B_w C_w B_w] \\ +B_w(F_{w_0} C_w + C_w X_x)] & \\ \vdots & \vdots \end{bmatrix}, \tag{12}$$

where

$$\begin{aligned} F_{x_0} &= (B - D) \\ F_{w_0} &= (A_w - D_w), \\ \tilde{B} &= \mathrm{diag}(BB_w w + B_w F_{w_0} w), \\ \tilde{D} &= \mathrm{diag}(F_{x_0} B_w w), \\ \tilde{D}_w &= \mathrm{diag}(BX_x B_w w - B^2 F_{w_0} w \\ &\qquad + B_w F_{w_0}^2 w + B_w C_w B_w w). \end{aligned}$$

Therefore, from Theorems 3.1 and 3.12 in [24], and since the system is analytic, we have the following theorem.

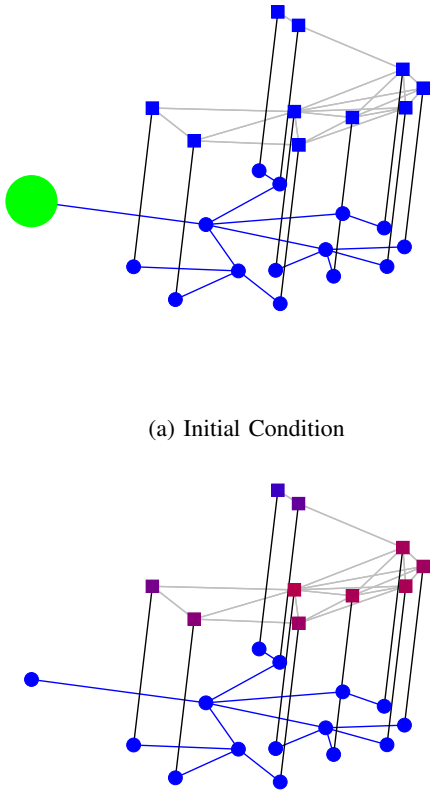
Theorem 2. *The layered networked SIWS model in* (7)-(8) *with measurements in* (11) *is weakly locally observable at* $x(0) = \mathbf{0}$ *if and only if* $\mathcal{O}$*, as defined in* (12)*, has full rank.*

V. Simulations

For the simulations we depict healthy nodes (both human and water) as blue. The water network and circle nodes are depicted in blue for healthy water and the nodes are green when the water is contaminated, and the spectrum between. If the value of w_j is greater than one, the node is all green and the diameter of the node is increased proportionally to the size of w_j. The human, or household, nodes are depicted by squares. Their color ranges between blue and red, indicating healthy and sick, and the spectrum between. The human contact network is depicted by gray edges. The connection from the water network to the human network is depicted by black edges.

The β_{ij}, β_{ij}^w, and α_{kj} are all binary. We set $D = 2I$, $D_w = 0.5I$, and $C_w = 0$, that is the people are not allowed to contaminate the water network. In this simulation, the B matrix is symmetric and the water network is a directed tree graph flowing from the first node which represents the reservoir. The initial condition has all the people healthy and the reservoir (the first water node) contaminated, $x(0) = \mathbf{0}$ and $w(0) = [1\ 0 \cdots 0]^\top$; see Figure 2a. The system has $\rho(D_f^{-1}B_f) > 1$. The system converges to a unique endemic equilibrium with $x(0) \gg \mathbf{0}$ and $w(0) = \mathbf{0}$; see Figure 2b.

Using the same parameters as the above simulation except allowing nonzero C_w with $C_w = B_w^\top$ gives noticeably different results; see Figure 3. The contaminant remains in the water system and the human contact network becomes more infected than the system in Figure 2. Both sets of simulations converged to the healthy state when D was increased such that $\rho(D_f^{-1}B_f) \leq 1$, consistent with the results in Section III.



(a) Initial Condition

(b) Equilibrium

Fig. 2: The human contact network is depicted by gray edges. The water network and nodes are depicted in blue for healthy water and green when the water is contaminated. The human, or household, nodes are depicted by squares. Their color ranges between blue and red, indicating healthy and sick, and the spectrum between. On the top is the initial condition, $x(0) = \mathbf{0}$ and $w(0) = [1\ 0 \cdots 0]^\top$, with the reservoir contaminated, depicted by the green node. On the bottom is the equilibrium with $x(0) \gg \mathbf{0}$ and $w(0) = \mathbf{0}$.

Using the same parameters as the simulation in Figure 2, except allowing the water network to be undirected changes the dynamics significantly; see Figure 4. The contaminant in the water system goes unstable very quickly (depicted by the whole frame being green) and the human contact network becomes completely infected. Increasing D did not decrease the effect of the instability since C was zero. Increasing D_w sufficiently did remove the stability and, if D was also increased sufficiently such that $\rho(D_f^{-1}B_f) \leq 1$, the system would converge to the healthy state, consistent with the results in Section III.

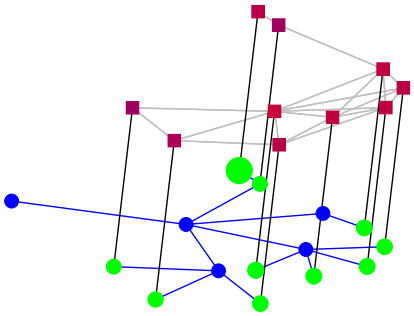


Fig. 3: Using the same parameters as the simulation in Figure 2 except allowing the internetwork links to be undirected ($C_w = B_w^\top$) the system appears to have this unique endemic state.

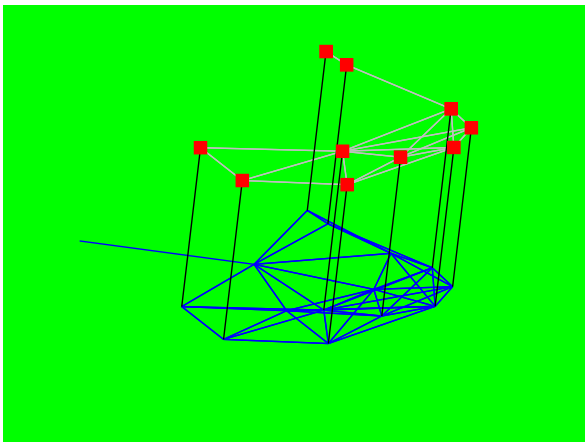


Fig. 4: Using the same parameters as the simulation in Figure 2 except allowing A_w to be undirected, the water states grow unboundedly and all the nodes in the human contact network converge to 1.

VI. Conclusion

In this paper, we have proposed a multi-network-dependent, continuous-time SIWS epidemic model, which captures a networked system of multiple groups of individuals with a water distribution network that can be contaminated. We have analyzed the stability of the healthy state, compared the basic reproduction number of the model with the standard networked SIS model without water, and discussed an observability problem of the system. For future work, we would like to analyze the number and stability of epidemic equilibria. We also want to explore the possible estimation and control techniques to detect and suppress a waterborne disease outbreak in finite time.

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