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A Networked SIS Disease Dynamics Model with a Waterborne Pathogen

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Abstract— This paper proposes a distributed continuous-time epidemic model, called networked SIWS (Susceptible-Infected-Water-Susceptible) model, for an SIS type waterborne disease spreading over a network of multiple groups of individuals sharing a water source. A sufficient condition is obtained for the healthy state, at which all individuals are not infected and the water is not contaminated, to be globally asymptotically stable. The effects of the shared water source on the disease spreading are analyzed through the comparison of the basic reproduction number with the networked SIS model without water and demonstrated via simulations.

I. INTRODUCTION

The progress of an epidemic in a large population is an important issue for humans and a widely studied area in epidemiology [1]. Various epidemic models have been proposed to model such a process. Notable examples include SIS, SIR, and SEIR models [2]-[4]. Networked SIS models have recently received increasing attention [5]-[8], particularly in the control systems literature [9]-[13], to name a few. There are two types of networked SIS models. One type is called multi-group SIS epidemic models which have been studied in [6], [8]. The other type considers a system consisting of multiple interactive individuals, instead of groups, and studies the evolution of each individual's probability of being infected. Such models are described by either a discrete-time system [9], [14], [15] or a continuous-time system [5], [7], [12]. In [6], a continuous-time multi-group SIS model was proposed and studied for strongly connected graphs. The work of [7] proposed a networked Markov chain model, whose mean-field approximation is the same as the model in [6], and studied the case of undirected graphs. The same model over directed graphs was studied in [12] for both strongly and weakly connected graphs. For a survey of recent development of networked epidemic models, see [16].

Waterborne pathogens have caused diseases and other health problems worldwide, especially in developing countries [17]. Water systems (e.g., rivers, groundwater, and reservoirs) are important pathways for transmitting pathogens [18]. Therefore, some recent studies have considered the role of a water compartment in epidemic dynamic processes. For example, the paper [19] developed the SIWR model by adding a water compartment W in the classical SIR model. The result in [19] shows that the SIWR model can better predict the infectious period than the SIR model. Following this approach, the work of [20] developed a cholera model considering both direct and indirect disease transmission pathways via a water compartment. The work of [21] proposed a reaction-diffusion waterborne pathogen model and investigated the role of the reproduction number in epidemic dynamics. The paper [22] evaluated how the reproduction number affects the global dynamics behaviors in the reaction-diffusion waterborne pathogen model, and the paper [23] modeled disease dynamics of a waterborne pathogen on a random network.

In this paper, we propose an extension of the networked SIS model by adding a water compartment W, in which both person-person and person-water-person transmissions exist, and thus we call it the networked SIWS model. There are two ways to model and interpret a networked SIS model. One way is to regard each agent as a group of fully connected individuals and each agent's variable represents the proportion of infected individuals in the corresponding group. The other way is to treat each agent as a computer and its variable means the probability of the corresponding computer being infected. In both ways, the variables take values between zero and one. In this paper, since we are interested in studying the epidemic spreading of a waterborne disease over multiple groups of individuals, the first interpretation and its corresponding model derivation better fit our purpose.

The main contributions of this paper are three-fold. First, we propose a networked model for SIS-type waterborne diseases, called networked SIWS model, for a systme consisting of multiple groups of individuals with a shared water resource. Second, we obtain a sufficient condition for the healthy state to be globally asymptotically stable. Third, we compare the basic reproduction number of the networked SIWS model with that of the networked SIS model for non-waterborne diseases, and provide a set of simulations to demonstrate the behavior of the networked SIWS model differing from the networked SIS model.

The remainder of the paper is organized as follows. We begin with some notation in Subsection I-A which will be used throughout the paper. We present a multi-group derivation of the model in Section II. In Section III, we present stability analysis of the origin, or healthy state, providing a sufficient condition for convergence to the healthy state, and discuss the relationship between the sufficient condition and the basic reproduction number from the existing literature. In Section IV, we provide a set of simulations that illustrate the behavior of the model, consistent with the analysis as well as a surprising simulation which shows that, unlike the

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existing SIS-type epidemic models, the sufficient condition is not necessary for convergence to the healthy state. We conclude with some remarks in Section V.

A. Notation

For any positive integer n, we use [n] to denote the set $\{1, 2, \ldots, n\}$. The *i*th entry of a vector x will be denoted by x_i . We use 0 and 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 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 $a \ge b$ if $a_i \ge b_i$ for all $i \in [n]$, a > b if $a \ge b$ and $a \ne b$, and $a \gg b$ if $a_i > b_i$ for all $i \in [n]$. For a real square matrix M, we use $\sigma(M)$ to denote the spectrum of M, use $\rho(M)$ to denote the largest real part among the eigenvalues of M, i.e., $s(M) = \max \{\operatorname{Re}(\lambda) : \lambda \in \sigma(M)\}$.

II. THE MODEL

In this section, we propose a distributed continuous-time waterborne pathogen model, called networked SIWS model, as follows. The model follows the ideas in [6] and [19].

Consider an SIS type waterborne disease spreading over a network consisting of n > 1 groups of individuals, labeled 1 to n, and a water compartment shared among the n groups. The water compartment can be contaminated by infected individuals shedding the pathogen into it. We simulate the water compartment W as a reservoir-like water system with homogeneous water quality, assuming instantaneous pathogen diffusion process in W. An individual may be infected either by contact with contaminated water or by contact with infected individuals only in its own and neighboring groups. Neighbor relationships among the ngroups are described by a directed graph \mathbb{G} on n vertices with an arc (or a directed edge) from vertex j to vertex iwhenever the individuals in group i can be infected by those in group j. Thus, the neighbor graph \mathbb{G} has self-arcs at all n vertices, and the directions of arcs in \mathbb{G} represent the directions of epidemic contagion. It is assumed that \mathbb{G} is strongly connected. We also assume that each group has bidirectional connection with the water compartment, which implies that each group can contaminate the water if it has infected individuals, and the individuals in each group can in turn get infected by the water if it is contaminated.

Let $I_i(t)$ and $S_i(t)$ respectively denote the number of infected and susceptible individuals in group i at time $t \ge 0$. We assume that the total number of individuals in each group i, denoted by N_i , does not change over time. In other words, $S_i(t) + I_i(t) = N_i$ for all $i \in [n]$ and $t \ge 0$, which implies that the birth and death rates for each group are equal. Such an assumption simplifies the model and has been adopted in [6]. We leave the relaxed, and more realistic, scenarios without this assumption as future work.

Associate with each group *i* several parameters: curing rate γ_i , birth rate μ_i , death rate $\bar{\mu}_i$, person-to-person infection

rates α_{ij} (with the understanding that $\alpha_{ij} > 0$ whenever group j is a neighbor of group i and $\alpha_{ij} = 0$ otherwise), and water-to-person infection rates α_{iw} . As discussed earlier, since N_i is constant, there holds $\bar{\mu}_i = \mu_i$. We assume 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:

$$\dot{S}_{i}(t) = \mu_{i}N_{i} - \bar{\mu}_{i}S_{i}(t) + \gamma_{i}I_{i}(t) - \sum_{j=1}^{n} \alpha_{ij}\frac{S_{i}(t)}{N_{i}}I_{j}(t)
- \alpha_{iw}W(t)S_{i}(t)
= (\mu_{i} + \gamma_{i})I_{i}(t) - \sum_{j=1}^{n} \alpha_{ij}\frac{S_{i}(t)}{N_{i}}I_{j}(t)
- \alpha_{iw}W(t)S_{i}(t),$$
(1)
$$\dot{I}_{i}(t) = -\gamma_{i}I_{i}(t) - \bar{\mu}_{i}I_{i}(t) + \sum_{j=1}^{n} \alpha_{ij}\frac{S_{i}(t)}{N_{i}}I_{j}(t)
+ \alpha_{iw}W(t)S_{i}(t)
= (-\gamma_{i} - \mu_{i})I_{i}(t) + \sum_{j=1}^{n} \alpha_{ij}\frac{S_{i}(t)}{N_{i}}I_{j}(t)
+ \alpha_{iw}W(t)S_{i}(t),$$
(2)

where W(t) denotes the pathogen concentration in the water reservoir which evolves as

$$\dot{W}(t) = -\delta_w W(t) + \sum_{k=1}^n \zeta_k I_k(t),$$
 (3)

where δ_w denotes the decay rate of pathogen in the water, and ζ_k denotes the person-water contact rate of group k. Note that (1) and (2) implies 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 and for the purpose of analysis, we change the variables of the model as follows. First, we denote the portion of infected individuals in each group i by $x_i(t)$, and thus,

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

Second, define a new variable as

$$z(t) = \frac{\delta_w}{\sum_{k=1}^n \zeta_k N_k} W(t),$$

which can be regarded as an index describing the waterborne pathogen concentration. Set the following parameters:

$$\delta_i = \gamma_i + \mu_i, \quad \beta_{ij} = \alpha_{ij} \frac{N_j}{N_i}, \quad \beta_{iw} = \frac{\alpha_{iw}}{\delta_w} \sum_{k=1}^n \zeta_k N_k.$$

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

$$\dot{x}_{i}(t) = -\delta_{i}x_{i}(t) + (1 - x_{i}(t))\left(\sum_{j=1}^{n}\beta_{ij}x_{j}(t) + \beta_{iw}z(t)\right).$$
(4)

. . .

To proceed, let

$$c_i = \frac{\zeta_i N_i}{\sum_{k=1}^n \zeta_k N_k}.$$
(5)

Then, from (3), it follows that

$$\dot{z}(t) = \delta_w \left(-z(t) + \sum_{k=1}^n c_k x_k(t) \right).$$
(6)

This paper deals with the systems given by (4) and (6). We impose the following assumptions on the system parameters.

Assumption 1: Suppose that $\delta_i > 0$ for all $i \in [n]$, $\delta_w > 0$, $\beta_{ij} \ge 0$ for all $i, j \in [n]$, $\beta_{ij} > 0$ whenever group j is a neighbor of group i, the matrix $B = [\beta_{ij}]_{n \times n}$ is irreducible, $\beta_{iw} > 0$ for all $i \in [n]$, and $c_i > 0$ for all $i \in [n]$.

It is worth noting that the assumption of an irreducible matrix B is equivalent to that the neighbor graph \mathbb{G} is strongly connected.¹ From (5), it is clear that all c_i , $i \in [n]$, form a set of convex combination weights.

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 z is nonnegative.

Lemma 1: Suppose that Assumption 1 holds. Suppose that $x_i(0) \in [0,1]$ for all $i \in [n]$ and $z(0) \ge 0$. Then, $x_i(t) \in [0,1]$ for all $i \in [n]$ and $z(t) \ge 0$ for all $t \ge 0$.

Proof: Suppose that at some time τ , there holds $x_i(\tau) \in [0,1]$ for all $i \in [n]$ and $z(t) \geq 0$. First consider z(t). If $x_i(\tau) = 0$, then from (6) and Assumption 1, $\dot{z}(\tau) \geq 0$. It follows that $z(t) \geq 0$ for all times $t \geq \tau$. Next consider any index $i \in [n]$. If $x_i(\tau) = 0$, then from (4) and Assumption 1, $\dot{x}_i(\tau) \geq 0$. It follows that $x_i(t)$ will be in [0,1] for all times $t \geq \tau$. Since the above arguments hold for any $i \in [n]$, we have that $x_i(t) \in [0,1]$ for all $i \in [n]$ and $z(0) \geq 0$, the lemma follows by taking $\tau = 0$.

More can be said.

Lemma 2: Suppose that Assumption 1 holds. Suppose that $x_i(0) \in [0,1]$ for all $i \in [n]$ and $z(0) \ge 0$. Then, for any $\epsilon > 0$, there exists a finite time T_{ϵ} for which $z(t) < 1 + \epsilon$ for all $t \ge T_{\epsilon}$.

Proof: From Lemma 1, $x_k(t) \in [0, 1]$ for all $k \in [n]$ and t. Since $\sum_{k=1}^{n} c_k x_k(t)$ is a convex combination of $x_k(t)$, $k \in [n]$, it follows that $\sum_{k=1}^{n} c_k x_k(t) \leq 1$ for all t. Suppose that $z(0) \geq 1+\epsilon$ for some $\epsilon > 0$. Then, $-z(0)+\sum_{k=1}^{n} c_k x_k(0) \leq -\epsilon$, which implies that z(t) will decrease as $\delta_w > 0$. At any time t, as long as $z(t) \geq 1 + \epsilon$, from (6), $\dot{z}(t) \leq -\epsilon \delta_w$, which implies that $z(t) < z(0) - \epsilon \delta_w t$. Set $T_{\epsilon} = (z(0) - 1 - \epsilon)/(\epsilon \delta_w)$. Then, $z(T_{\epsilon}) < 1 + \epsilon$.

Lemma 2 implies that at any possible equilibrium, the value of z can only be equal to or less than one. We claim that z = 1 cannot hold at any equilibrium. To see this, suppose to the contrary that there exists an equilibrium at which z = 1. From (6) and Assumptions 1, since $\sum_{k=1}^{n} c_k x_k$ is a convex combination of all x_k , the equilibrium condition $\sum_{k=1}^{n} c_k x_k = z = 1$ implies that all $x_k = 1$ at this equilibrium. But it conflicts (4) at the equilibrium. Therefore,

¹ A directed graph is *strongly connected* if there is a directed path between each pair of ordered distinct vertices.

z must be strictly less than one at any equilibrium. The following lemma shows that z(t) is positively invariant in the set [0, 1].

Lemma 3: Suppose that Assumption 1 holds, and that $x_i(0) \in [0,1]$ for all $i \in [n]$ and $z(0) \ge 0$. Suppose that at some time $\tau \ge 0$, $z(\tau) \in [0,1]$. Then, $z(t) \in [0,1]$ for all $t > \tau$.

Proof: Following similar analysis as in the proof of Lemma 2, one can show that $\sum_{k=1}^{n} c_k x_k(t) \in [0,1]$ for all $t \ge 0$. Suppose that at time $\overline{\tau} > \tau$, there holds $z(\overline{\tau}) = 1$. From (5), it follows that $\dot{z}(\overline{\tau}) \le 0$. Suppose that at time $\underline{\tau} > \tau$, there holds $z(\underline{\tau}) = 0$. Similarly, one can show that $\dot{z}(\underline{\tau}) \ge 0$. Thus, it follows that $z(t) \in [0,1]$ for all $t > \tau$.

Motivated by the preceding discussion and without loss of generality, we now study the following continuous-time networked system with specified initial conditions:

$$\dot{x}_{i}(t) = -\delta_{i}x_{i}(t) + (1 - x_{i}(t))\left(\sum_{j=1}^{n}\beta_{ij}x_{j}(t) + \beta_{iw}z(t)\right),$$
$$x_{i}(0) \in [0, 1], \quad i \in [n], \tag{7}$$

$$\dot{z}(t) = \delta_w \left(-z(t) + \sum_{k=1}^n c_k x_k(t) \right), \quad z(0) \in [0, \infty), \quad (8)$$

where δ_i , δ_w , β_{ij} , β_{iw} , and c_i are model parameters satisfying Assumption 1.

The above n + 1 differential equations can be combined into one equation in a compact form. Toward this end, let x(t) be the state vector in \mathbb{R}^n whose *i*th entry is $x_i(t)$, Dbe the $n \times n$ diagonal matrix whose *i*th diagonal entry is δ_i , B be the $n \times n$ matrix whose *i*th entry is β_{ij} , X(t) be the $n \times n$ diagonal matrix whose *i*th diagonal entry is $x_i(t)$, bbe the vector in \mathbb{R}^n whose *i*th entry is β_{iw} , and c be the vector in \mathbb{R}^n whose *i*th entry is $\delta_w c_i$. Then, from (7) and (8), it can be verified that

$$\dot{x}(t) = (-D + B - X(t)B) x(t) + (I - X(t)) bz(t), \quad (9)$$

$$\dot{z}(t) = -\delta_w z(t) + c^{\top} x(t), \quad (10)$$

which can be written as

$$\begin{bmatrix} \dot{x}(t) \\ \dot{z}(t) \end{bmatrix} = \begin{bmatrix} -D + B - X(t)B & (I - X(t))b \\ c^{\top} & -\delta_w \end{bmatrix} \begin{bmatrix} x(t) \\ z(t) \end{bmatrix}$$
(11)

In the special case when z(t) = 0 for all t, or equivalently, no water compartment exists, system (11) simplifies to

$$\dot{x}(t) = (-D + B - X(t)B)x(t), \tag{12}$$

which is the networked SIS model studied in [6].

Lemma 1 implies that for the x system defined in (9), the set $[0,1]^n$ is positively invariant. Lemma 2 and the fact that the value of z is strictly less than one at any equilibrium imply that the z system defined in (10) will enter the interval [0,1] for any nonnegative initial value. Lemma 3 indicates that z(t) is positively invariant in the set [0,1]. Therefore, the system (11) is positively invariant on the set $[0,1]^{n+1}$.

III. MAIN RESULTS

It can be seen that (x, z) = (0, 0) is an equilibrium of the system (11), which implies that no individual is infected and the water compartment is not contaminated. We call this equilibrium the *healthy state*. In this section, we study the stability of the healthy state. To state our main result, we need the following concept and result.

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 $\lim_{t\to\infty} x(t) = x^*$.

Proposition 1: Let x^* be an equilibrium of $\dot{x}(t) = f(x(t))$ and $\mathcal{E} \subset \mathcal{D}$ be a bounded domain containing x^* . Let $V : \mathcal{E} \to \mathbb{R}$ be a continuously differentiable function such that $V(x^*) = 0$, V(x) > 0 in $\mathcal{E} \setminus \{x^*\}$, $\dot{V}(x^*) = 0$, and $\dot{V}(x) < 0$ in $\mathcal{E} \setminus \{x^*\}$. If \mathcal{E} is an invariant set, then the equilibrium x^* is asymptotically stable with the domain of attraction \mathcal{E} .

The proposition can be proved using the same arguments in the proof of Lyapunov's stability theorem (see Theorem 4.1 and the discussion on page 122 in [24]).

To proceed, we define the following two matrices:

$$D_w = \begin{bmatrix} D & \mathbf{0} \\ \mathbf{0}^\top & \delta_w \end{bmatrix}, \quad B_w = \begin{bmatrix} B & b \\ c^\top & 0 \end{bmatrix}. \tag{13}$$

From Assumption 1, it is clear that D_w is a positive diagonal matrix, and that B_w is an irreducible nonnegative matrix, which implies that $(-D_w + B_w)$ is an irreducibel Metzler matrix.²

A. Local Stability of the Healthy State

Let (\tilde{x}, \tilde{z}) be an equilibrium of (11). Then, the Jacobian matrix of the equilibrium, denoted by $J(\tilde{x}, \tilde{z})$, is

$$J(\tilde{x},\tilde{z}) = \begin{bmatrix} -D + B - \bar{X}B - H_1 - H_2 & (I - \bar{X})b \\ c^{\top} & -\delta_w \end{bmatrix},$$

where \bar{X}, H_1, H_2 are diagonal matrices given by

$$X = \operatorname{diag} \left\{ \bar{x}_1, \bar{x}_2, \cdots, \bar{x}_n \right\},$$

$$H_1 = \operatorname{diag} \left\{ \sum_{j=1}^n \beta_{1j} \bar{x}_j, \sum_{j=1}^n \beta_{2j} \bar{x}_j, \cdots, \sum_{j=1}^n \beta_{nj} \bar{x}_j \right\},$$

$$H_2 = \operatorname{diag} \left\{ \beta_{1w} \bar{z}, \beta_{2w} \bar{z}, \cdots, \beta_{nw} \bar{z} \right\}.$$

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

$$J(\mathbf{0},0) = \begin{bmatrix} -D+B & b\\ c^{\top} & -\delta_w \end{bmatrix}.$$

Note that $J(\mathbf{0}, 0) = -D_w + B_w$ where D_w and B_w are given in (13). From Lemma 4, condition $\rho(D_w^{-1}B_w) < 1$ is equivalent to $s(-D_w + B_w) < 0$, which implies that $J(\mathbf{0}, 0)$ is a continuous-time sable matrix. Thus, we have the following result.

 $^2\mathrm{A}$ real square matrix is called Metzler if its off-diagonal entries are all nonnegative.

Proposition 2: Let Assumption 1 hold. If $\rho(D_w^{-1}B_w) < 1$, then the healthy state (0,0) of system (11) is locally exponentially stable.

B. Global Stability of the Healthy State

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

Theorem 1: Let Assumption 1 hold. If $\rho(D_w^{-1}B_w) \leq 1$, then the healthy state (0,0) of system (11) is asymptotically stable with the domain of attraction $x \in [0,1]^n$ and $z \in [0,\infty)$.

To prove this theorem, we need the following properties of Metzler matrices.

Lemma 4: [Proposition 1 in [25]] Suppose that Λ is a negative diagonal matrix in $\mathbb{R}^{n \times n}$ and N is an irreducible nonnegative matrix in $\mathbb{R}^{n \times n}$. Let $M = \Lambda + N$. Then, s(M) < 0 if and only if $\rho(-\Lambda^{-1}N) < 1$, s(M) = 0 if and only if $\rho(-\Lambda^{-1}N) = 1$, and s(M) > 0 if and only if $\rho(-\Lambda^{-1}N) > 1$.

Lemma 5: [Lemma 2.3 in [26]] 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 6: [Proposition 2 in [27]] Suppose that M is an irreducible Metzler matrix such that s(M) < 0. Then, there exists a positive diagonal matrix P such that $M^{\top}P + PM$ is negative definite.

Lemma 7: [Lemma A.1 in [12]] Suppose that M is an irreducible Metzler matrix such that s(M) = 0. Then, there exists a positive diagonal matrix P such that $M^{\top}P + PM$ is negative semi-definite.

Proof of Theorem 1: We first consider the case when $\rho(D_w^{-1}B_w) < 1$. By Lemma 4, in this case, $s(-D_w + B_w) < 0$. Since $(-D_w + B_w)$ is an irreducible Metzler matrix, by Lemma 6, there exists a positive diagonal matrix P such that $(-D_w + B_w)^\top P + P(-D_w + B_w)$ is negative definite. For convenience, define $y(t) = [x(t)^\top z(t)]^\top$. Consider the Lyapunov function $V(y(t)) = y(t)^\top Py(t)$. Then, from (11) and (13), when $y(t) \neq 0$, we have

$$\begin{split} \dot{V}(y(t)) &= 2y(t)^{\top} P \dot{y}(t) \\ &= 2y(t)^{\top} P(-D_w + B_w) y(t) \\ &+ 2y(t)^{\top} P \begin{bmatrix} -X(t)B & -X(t)b \\ \mathbf{0} & 0 \end{bmatrix} y(t) \\ &< -2y(t)^{\top} P \begin{bmatrix} X(t)B & X(t)b \\ \mathbf{0} & 0 \end{bmatrix} y(t) \\ &\leq 0. \end{split}$$

Thus, in this case, $\dot{V}(y(t)) < 0$ if $y(t) \neq 0$. In the last paragraph of Section II, we have shown that z(t) will enter the interval [0,1] for any nonnegative initial value, and that system (11) is positively invariant on the set $[0,1]^{n+1}$. From Lemma 1, Lemma 2, and Proposition 1, y = 0 is asymptotically stable with the domain of attraction $x \in [0,1]^n$ and $z \in [0,\infty)$.

Next we consider the case when $\rho(D_w^{-1}B_w) = 1$. By Lemma 4, $s(-D_w + B_w) = 0$. Since $(-D_w + B_w)$ is an irreducible Metzler matrix, by Lemma 7, there exists a positive diagonal matrix Q such that $(-D_w + B_w)^\top Q + Q(-D_w + B_w)$ is negative semi-definite. Consider the Lyapunov function $V(y(t)) = y(t)^\top Q y(t)$. Then, from (11) and (13), we have

$$\begin{split} \dot{V}(y(t)) &= 2y(t)^{\top}Q(-D_w + B_w)y(t) \\ &+ 2y(t)^{\top}Q \begin{bmatrix} -X(t)B & -X(t)b \\ \mathbf{0} & 0 \end{bmatrix} y(t) \\ &\leq -2y(t)^{\top}Q \begin{bmatrix} X(t)B & X(t)b \\ \mathbf{0} & 0 \end{bmatrix} y(t) \\ &= -2y(t)^{\top} \begin{bmatrix} \hat{Q} & 0 \\ \mathbf{0} & q \end{bmatrix} \begin{bmatrix} X(t)B & X(t)b \\ \mathbf{0} & 0 \end{bmatrix} y(t) \\ &= -2 \left(x(t)^{\top}\hat{Q}X(t)Bx(t) + x(t)^{\top}\hat{Q}X(t)bz(t) \right) \\ &\leq -2 \left(x(t)^{\top}\hat{Q}X(t)Bx(t) \right) \\ &\leq 0, \end{split}$$

where \hat{Q} is the *n*th principal subarray of Q, which is an $n \times n$ positive diagoal matrix, and q is the (n+1)th diagonal entry of Q. We claim that $\dot{V}(y(t)) < 0$ if $y(t) \neq \mathbf{0}$. To establish this claim, we first consider the case when $y(t) \gg \mathbf{0}$, i.e., $x(t) \gg 0$ and z(t) > 0. Since B is nonnegative and irreducible because of Assumption 1, $Bx(t) \gg \mathbf{0}$. Since \hat{Q} is a positive diagonal matrix, it follows that $x(t)^{\top}\hat{Q}X(t)Bx(t) > 0$, so $\dot{V}(y(t)) < 0$. Next we consider the case when $y(t) > \mathbf{0}$ and y(t) has at least one zero entry. If $(-D_w + B_w)^{\top}Q + Q(-D_w + B_w)$ does not have an eigenvalue at zero, then $(-D_w + B_w)^{\top}Q + Q(-D_w + B_w)$ is negative definite, which implies that $y(t)^{\top}((-D_w + B_w)^{\top}Q + Q(-D_w + B_w))y(t) < 0$ when $y(t) > \mathbf{0}$ and, thus, in this case,

$$\dot{V}(y(t)) = 2y(t)^{\top}Q(-D_w + B_w)y(t) + 2y(t)^{\top}Q \begin{bmatrix} -X(t)B & -X(t)b \\ \mathbf{0} & 0 \end{bmatrix} y(t) \leq 2y(t)^{\top}Q(-D_w + B_w)y(t) < 0.$$

Now suppose that $(-D_w + B_w)^\top Q + Q(-D_w + B_w)$ has an eigenvalue at zero. Since $(-D_w + B_w)$ is an irreducible Metzler matrix and Q is a positive diagonal matrix, $(-D_w+B_w)^{\top}Q+Q(-D_w+B_w)$ is a symmetric irreducible Metzler matrix. Since $(-D_w + B_w)^\top Q + Q(-D_w + B_w)$ is negative semi-definite, it follows that $s((-D_w + B_w)^\top Q +$ $Q(-D_w + B_w)) = 0$. By Lemma 5, it follows that zero is a simple eigenvalue of $(-D_w + B_w)^\top Q + Q(-D_w + B_w)$ and it has a unique (up to scalar multiple) strictly positive eigenvector corresponding to the eigenvalue zero. Thus, $y(t)^{\top} \left((-D_w + B_w)^{\top} Q + Q(-D_w + B_w) \right) y(t) < 0$ when y(t) > 0 and y(t) has at least one zero entry. Therefore, V(y(t)) < 0 if $y(t) \neq 0$. Using the same arguments used in the case when $ho(D_w^{-1}B_w) < 1$ and from Lemma 1, Lemma 2, and Proposition 1, y = 0 is asymptotically stable with the domain of attraction $x \in [0, 1]^n$ and $z \in [0, \infty)$.

C. Basic Reproduction Number

For the networked SIS model (12), $\rho(D^{-1}B)$ is called the basic reproduction number. It has been shown in [6] 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. For the networked SIWS model (11), Theorem 1 implies that when $\rho(D_w^{-1}B_w) \leq 1$, the model will asymptotically converge to the healthy state for all initial conditions, which implies that the healthy state is the unique equilibrium in this case, and extensive simulations show that when $\rho(D_w^{-1}B_w) > 1$, the model has an epidemic equilibrium. Thus, we call $\rho(D_w^{-1}B_w)$ the basic reproduction number of the networked SIWS model (11), and compare its value with that of the networked SIS model (12), $\rho(D^{-1}B)$, to reveal the effect of the shared water compartment. Note that

$$D_w^{-1}B_w = \begin{bmatrix} D^{-1} & \mathbf{0} \\ \mathbf{0}^{ op} & rac{1}{\delta_w} \end{bmatrix} \begin{bmatrix} B & b \\ c^{ op} & 0 \end{bmatrix}$$

 $= \begin{bmatrix} D^{-1}B & D^{-1}b \\ rac{1}{\delta_w}c^{ op} & 0 \end{bmatrix}.$

Since $D_w^{-1}B_w$ is an irreducible nonnegative matrix by Assumption 1, $\rho(D_w^{-1}B_w) > \rho(D^{-1}B)$ because of the following lemma.

Lemma 8: [Lemma 2.6 in [26]] Suppose that N is an irreducible nonnegative matrix. If M is a principal square submatrix of N, then $\rho(M) < \rho(N)$.

We thus have proved the following result.

Proposition 3: Suppose that Assumption 1 holds. Then, the basic reproduction number of the networked SIWS model (11) is greater than that of the networked SIS model (12).

The proposition implies that the shared water compartment yields a more vulnerable system to SIS type diseases.

IV. SIMULATIONS

The Village of Richmond was the government center of Staten Island for nearly two centuries. Now there is an interactive museum/site called the Historic Richmond Town [28]. We used a subset of the map of the current setting given in Figure 1, excluding the smaller buildings, for the neighbor graph in our simulations. Richmond Creek, which flows out of Orbach Lake, is the water source for the simulations.

For the simulations we made the simplifying assumption that the underlying interaction graph for each household is determined by its nearest proximity neighbors, acknowledging that more general interaction graphs are also possible. We set the infection rate matrix B as a binary matrix corresponding to the edges in Figure 2, i.e., $\beta_{ij} = 1$ whenever j is a neighbor of i. Note that we chose an undirected neighbor graph for convenience. The state of the system is the proportion of the households that are infected with the disease, and the dynamics follow (7) and (8).

For each node *i* representing the *i*th group of individuals, we used the color blue $(b = [0 \ 0 \ 1])$ and red $(r = [1 \ 0 \ 0])$ to depict its completely healthy state $(x_i = 0, \text{ i.e., all its})$



Fig. 1. Map of Historic Richmond Town [28]



Fig. 2. The epidemic equilibrium when $\delta_i = 2$, $\delta_w = 1$, $\beta_{iw} = 1$ and $c_i = 1$ for all $i \in [n]$. Note that all the households are at least somewhat sick.

individuals are healthy) and completely epidemic state ($x_i = 1$, i.e., all its individuals are infected), respectively. At each time t, its color is given by

$$(1 - x_i(t))b + x_i(t)r.$$

Thus, any partially infected group will be depicted by a purple node. In the sequel, we will present three simulations.

In the first simulation, we set $\delta_i = 2$, $\delta_w = 1$, $\beta_{iw} = 1$ and $c_i = 1$ for all $i \in [n]$. Then, $\rho(D_w^{-1}B_w) > 1$, and the system converges to an epidemic equilibrium, as shown in Figure 2. The equilibrium appears to be independent of initial conditions, as long as one of the initial states is nonzero, which implies that at least one household is (partially)



Fig. 3. The healthy equilibrium when $\delta_i = 10$, $\delta_w = 1$, $\beta_{iw} = 1$ and $c_i = 1$ for all $i \in [n]$.

infected, or the water source is (partially) contaminated.

In the second simulation, we set $\delta_i = 10$, $\delta_w = 1$, $\beta_{iw} = 1$ and $c_i = 1$ for all $i \in [n]$. Then, $\rho(D_w^{-1}B_w) < 1$, and the system converges to the healthy state for all initial conditions, as shown in Figure 3.

In the last simulation, we set $\delta_i = 8$, $\delta_w = 1$, $\beta_{iw} = 1$ and $c_k = 1$ for all $i \in [n]$. Then, $\rho(D_w^{-1}B_w) > 1$, but the system may still converge to the healthy state (the same as the second simulation). However, the system appears to be dependent on the initial conditions. For example, if $x_1(0) = .5$ and the rest of the states are initialized as zeros, the system converges to the healthy state, but if z(0) = 1 and x(0) = 0, the system converges to an epidemic equilibrium. This is a very interesting behavior which is distinct from the networked SIS model (12). It appears that the linear decay rate of the z state can, in some cases, dominate the system when $\rho(D_w^{-1}B_w) > 1$. The behavior and observation add an intrigue that merits future research.

V. CONCLUDING REMARKS

In this paper, we have proposed a network-dependent, continuous-time SIWS epidemic model, which captures a networked system of multiple groups of individuals with a shared water source that can be contaminated. We have obtained the basic reproduction number for the networked model and shown that the healthy state is globally asymptotically stable if the number is less than or equal to one. We have illustrated the behavior of the model via simulations on a small population with a contaminated water source.

As an immediate future direction, we plan to extend the model to reflect more realistic scenarios. For instance, the decay rate parameter δ_w is assumed to be a positive constant for the convenience of theoretical analysis. We remark that in a more general sense, one can also consider time-varying or even state-dependent decay rate $\delta_w(t)$. Time-varying or state-dependent decay rate can be used to capture the interactions between infected populations and self-cleaning capabilities in

water reservoir, which provides a better modelling for reallife systems. We will show that, as long as $\delta_w(t) > 0$, the main results on equilibrium points and stability conclusions are not affected.

For future work, we would like to analyze the stability of epidemic equilibrium, which from the simulations appears to be unique. We want to analyze the interesting case illuminated in the simulations where $\rho(D_w^{-1}B_w) > 1$ but the system still converges to the healthy state. We also want to explore the possibility of allowing time-varying graph structures in the model. Another interesting direction for future work is to relax the assumption that each group has a constant population, allowing sub-populations in each group to flow between each other. We are currently developing a SIWR (Susceptible-Infected-Water-Removed) model and would like to develop new water models to capture interactions in modern water systems, such as networked water resources.

REFERENCES

- [1] R. M. Anderson and R. M. May. *Infectious Diseases of Humans*. Oxford University Press, 1991.
- [2] R. Pastor-Satorras and A. Vespignani. Epidemic spreading in scalefree networks. *Physical Review Letters*, 86(14):3200–3203, 2001.
- [3] W. O. Kermack and A. G. McKendrick. A contribution to the mathematical theory of epidemics. *Proceedings of the Royal Society* A, 115(772):700–721, 1927.
- [4] M. Y. Li and J. S. Muldowney. Global stability for the SEIR model in epidemiology. *Mathematical Biosciences*, 125(2):155–164, 1995.
- [5] A. Lajmanovich and J. A. Yorke. A deterministic model for gonorrhea in a nonhomogeneous population. *Mathematical Biosciences*, 28(3-4):221–236, 1976.
- [6] A. Fall, A. Iggidr, G. Sallet, and J. J. Tewa. Epidemiological models and Lyapunov functions. *Mathematical Modelling of Natural Phenomena*, 2(1):55–73, 2007.
- [7] P. Van Mieghem, J. Omic, and R. Kooij. Virus spread in networks. *IEEE/ACM Transactions on Networking*, 17(1):1–14, 2009.
- [8] Z. Shuai and P. van den Driessche. Global stability of infectious disease models using Lyapunov functions. *SIAM Journal on Applied Mathematics*, 73(4):1513–1532, 2013.
- [9] H. J. Ahn and B. Hassibi. Global dynamics of epidemic spread of over complex networks. In *Proceedings of the 52nd IEEE Conference* on Decision and Control, pages 4579–4585, 2013.
- [10] V. M. Preciado, M. Zargham, C. Enyioha, A. Jadbabaie, and G. Pappas. Optimal resource allocation for network protection against spreading processes. *IEEE Transaction on Control of Network Systems*, 1(1):99– 108, 2014.
- [11] E. Ramírez-Llanos and S. Martínez. A distributed algorithm for virus spread minimization. In *Proceedings of the 2014 American Control Conference*, pages 184–189, 2014.
- [12] A. Khanafer, T. Başar, and B. Gharesifard. Stability of epidemic models over directed graphs: a positive systems approach. *Automatica*, 74:126–134, 2016.
- [13] P. E. Paré, C. L. Beck, and A. Nedić. Epidemic processes over timevarying networks. *IEEE Transactions on Control of Network Systems*, 5(3):1322–1334, 2018.
- [14] D. Chakrabarti, Y. Wang, C. Wang, J. Leskovec, and C. Faloutsos. Epidemic spreading in real networks. ACM Transactions on Information and System Security, 10(4):Article 13, 2008.
- [15] V. S. Bokharaie, O. Mason, and F. Wirth. Spread of epidemics in time-dependent networks. In *Proceedings of the 19th International Symposium on Mathematical Theory of Networks and Systems*, pages 1717–1719, 2010.
- [16] C. Nowzari, V. M. Preciado, and G. J. Pappas. Analysis and control of epidemics: A survey of spreading processes on complex networks. *IEEE Control Systems Magazine*, 36(1):26–46, 2016.

- [17] L. Vermeulen, N. Hofstra, C. Kroeze, and G. Medema. Advancing waterborne pathogen modelling: lessons from global nutrient export models. *Current Opinion in Environmental Sustainability*, 14:109–120, 2015.
- [18] A. Kough, C. Paris, D. Behinger, and M. Butler. Modelling the spread and connectivity of waterborne marine pathogens: the case of PaV1 in the Caribbean. *ICES Journal of Marine Science*, 72:i139–i146, 2015.
- [19] J. H. Tien and D. J. D. Earn. Multiple transmission pathways and disease dynamics in a waterborne pathogen model. *Bulletin of Mathematical Biology*, 72:1506–1533, 2010.
- [20] Z. Shuai and P. van de Driessche. Global dynamics of cholera models with differential infectivity. *Mathematical Biosciences*, 234:118–126, 2011.
- [21] L. Zhang, Z.-C. Wang, and Y. Zhang. Dynamics of a reaction-diffusion waterborne pathogen model with direct and indirect transmission. *Computers and Mathematics with Applications*, 72:202–215, 2016.
- [22] J. Zhou, Y. Yang, and T. Zhang. Global dynamics of a reactiondiffusion waterborne pathogen model with general incidence rate. *Journal of Mathematical Analysis and Applications*, 466:835–859, 2018.
- [23] M. Li, J. Ma, and P. van de Driessche. Model for disease dynamics of a waterborne pathogen on a random network. *Mathematical Biology*, 71:961–977, 2015.
- [24] H. K. Khalil. Nonlinear Systems. Prentice Hall, 2002.
- [25] J. Liu, P. E. Paré, A. Nedić, C. Y. Tang, C. L. Beck, and T. Başar. On the analysis of a continuous-time bi-virus model. *IEEE Transactions on Automatic Control.* conditionally accepted, available at arXiv:1603.04098v2.
- [26] R. Varga. Matrix Iterative Analysis. Springer-Verlag, 2000.
- [27] A. Rantzer. Distributed control of positive systems. In Proceedings of the 50th IEEE Conference on Decision and Control, pages 6608–6611, 2011.
- [28] Main village richmond town site. https:// www.historicrichmondtown.org/collections/ main-village. Accessed: 2018-09-24.