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Stability analysis of generalized epidemic models over directed networks

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Abstract— **In this paper we propose a generalized version of the Susceptible-Exposed-Infected-Vigilant (SEIV) disease spreading model over arbitrary directed graphs. In the standard SEIV model there is only one infectious state. Our model instead allows for the exposed state to also be infectious to healthy individuals. This model captures the fact that infected individuals may act differently when they are aware of their infection. For instance, when the individual is aware of the infection, different actions may be taken, such as staying home from work, causing less chance for spreading the infection. This model generalizes the standard SEIV model which is already known to generalize many other infection spreading models available. We use tools from nonlinear stability analysis to suggest a coordinate transformation that allows us to study the stability of the origin of a relevant linear system. We provide a necessary and sufficient condition for when the disease-free equilibrium is globally exponentially stable. We then extend the results to the case where the infection parameters are not homogeneous among the nodes of the network. Simulations illustrate our results.**

I. Introduction

The modeling and analysis of infectious diseases on complex networks is fast becoming an interesting and popular research topic. There are many works that focus on a myriad of different disease spreading models using various techniques for their analysis. Proper modeling and analysis of such systems is important in being able to determine when a certain disease in a community will naturally die out or cause an epidemic. Understanding the answer to this question will allow better decisions to be made regarding how much treatment or vaccine should be administered to different people.

In this paper we are interested in studying an epidemic model that generalizes many other models studied in the literature and also captures effects that human awareness may have. By considering arbitrary strongly connected directed graphs, we extend the applicability of our results beyond the motivating human disease spreading problem. For instance, our model is able to capture the spread of rumors or computer viruses through social networks, or the popularity of various products in a large market. We also consider node-dependent parameters which are able to capture natural inclinations or affinities that different individuals may have towards different diseases and/or products.

Literature review

The most widely studied infection model is the Susceptible-Infected-Susceptible (SIS) model. Earlier works often consider structured contact graphs such as homogeneous models that assume each individual has equal contact to everyone else [1]. A discrete-time model over arbitrary contact graphs is considered in [2]. One of the first works to consider a continuous-time SIS model over arbitrary contact graphs using mean field theory is [3]. A further analysis in [4] provides conditions on the disease and contact graph for global exponential stability of the disease-free equilibrium for both the continuous and discrete-time cases, i.e., conditions on when the disease will naturally die out. In case these conditions are not satisfied, the authors also show the existence of a nontrivial equilibrium where the disease never dies out. Unlike the above works, we consider arbitrary strongly connected directed networks. While undirected graphs are useful in modeling spreading of diseases among humans, directed graphs may be more appropriate when considering the spreading of computer viruses [5], [6] or products and information [7]. For instance, famous website or celebrities may have direct influence over many individuals while not being directly affected by them. Unlike the above works, we are also interested in relaxing the assumption that the infection parameters are homogeneous throughout the network. By allowing infection parameters to be different, we are able to capture the fact that individuals may respond to a given disease differently based on their personalities or bodies. This has very recently been rigorously studied for the two state SIS model [8].

An important factor in how infections can spread besides the contact graph and parameters such as infection rate and recovery rate is how humans adapt their behavior to the possibility of an emerging epidemic [9], [10]. A survey of results studying how disease spreading can be affected by changing human behavior is provided in [11]. The work [12] considers the possible effect of human behavior changes for the three state Susceptible-Alert-Infected-Susceptible (SAIS) model. In this model there are two healthy states and one infectious states; however, people in the alert state are aware of the possible epidemic and taking actions accordingly such as wearing masks or decreasing contact with other people. The authors are able to provide conditions for when the disease dies out, and additional conditions for when the non disease-free equilibrium is similar to that of the well known SIS model's equilibrium.

Another model that has recently become popular is the Susceptible-Exposed-Infected-Vigilant (SEIV) model. This model is appealing not only in that it can model diseases which have a latent period where a person can have the disease but not yet be infectious, but it also generalizes a large number of other studied models as shown in [13],

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[14]. A generalized version of the SEIV model in which birth and mortality are also considered are studied in [15], [16], but without considering graph interactions. Instead, we are interesting in studying another generalized version of the SEIV model over complex graphs in which human reaction to succumbing to the disease is also modeled, which also generalizes the popular SIS model.

Some recent works study control or resource allocation (such as a vaccine or cure) for simple models such as the SIS model [17], [18], [19]. By being able to properly analyze the generalized model we propose, we hope to facilitate control of the spreading of a given virus or infection in more general settings as well.

Statement of contributions

In this paper we propose a generalized version of the continuous-time Susceptible-Exposed-Infected-Vigilant (SEIV) model in which both the exposed and infected states are infectious. This model generalizes many models studied in the literature including SEIV, SIS, SIR, SIRS, SEIR, SEIS, and SIV [13], [14]. The reason for having two infectious states is to model human behavioral changes when infected with a disease. The exposed state corresponds to a person having the disease and being contagious, but not yet aware that they are sick. The infected state means the person is infected and aware of the disease, which means the person might behave differently. For instance, a person knowingly infected with a disease may have less contact with others due to staying home from work or school, yielding less chance of spreading the infection. After proposing the model in detail, we provide a useful coordinate change that allows us to study the stability of the origin of a relevant nonlinear system. By showing that the nonlinear system is upper-bounded by its linearization, we are able to provide necessary and sufficient conditions on the graph and parameters of the infection such that the disease dies out exponentially. We then extend the results to the case in which infection parameters are node dependent rather than constant throughout the entire network. Simulations illustrate our results.

Notation

We denote by $\mathbb{R}$ and $\mathbb{R}_{\geq 0}$ the sets of real and nonnegative real numbers, respectively. Given a graph $\mathcal{G}$, we denote by A the associated adjacency matrix. Given square matrices $M_1, \ldots, M_N$ where $M_i \in \mathbb{R}^{n_i \times n_i}$, we let $\operatorname{diag}(M_1, \ldots, M_N)$ denote the $n \times n$ block diagonal matrix with $M_1, \ldots, M_N$ on the diagonal where $n = \sum_{i=1}^{N} n_i$. The indicator function $\mathbf{1}_Z$ is 1 if Z is true, and 0 otherwise.

II. Model Description

Here we follow the idea of the N-intertwined SIS model developed in [3] and its extension to the SAIS model developed in [12] and consider its extension to the generalized SEIV model (G-SEIV) we are interested in.

We consider a virus spreading model with four states for each node: susceptible S, exposed E, infected I, and vigilant V. The susceptible state S corresponds to a healthy individual who is capable of being exposed to the disease. The exposed state E corresponds to an individual that has been exposed to the disease and is contagious, but not yet aware of this. The infected state I corresponds to an infected individual who is aware of the infection. Lastly, the vigilant state V corresponds to an individual that is not susceptible or infected by the disease. This can mean the individual has just recovered or been vaccinated and thus is not contagious, nor immediately susceptible to be infected.

Consider a network with with N nodes. For each node $i \in \{1, \ldots, N\}$ we define the random variable $X_i(t) \in \{S, E, I, V\}$ as the state of node i at a given time t. We consider a strongly connected contact graph $\mathcal{G}$ over which the disease can spread. A susceptible node is only able to become exposed if it has at least one neighbor that is either exposed or infected. The in and out-neighbors of an agent i on $\mathcal{G}$ are denoted by $\mathcal{N}_i^{\text{in}}$ and $\mathcal{N}_i^{\text{out}}$, respectively. A node i can only be infected by nodes in $\mathcal{N}_i^{\text{in}}$ and can only infect nodes in $\mathcal{N}_i^{\text{out}}$. We define the adjacency matrix A as $a_{i,j} = 1$ if node i can be infected by node j, i.e., $j \in \mathcal{N}_i^{\text{in}}$, and 0 otherwise. Note that by definition, this is equivalent to saying $i \in \mathcal{N}_j^{\text{out}}$.

Figure 1 shows a simple 5 node network with an underlying directed contact graph $\mathcal{G}$ shown with solid black lines. Depending on the states of the nodes and the contact graph, the dashed red lines show which nodes are being affected by an exposed or infected neighbor. As discussed above, a node i can only affect a neighboring $j \in \mathcal{N}_i^{\text{out}}$ if i is in either the exposed state E or infected state I.

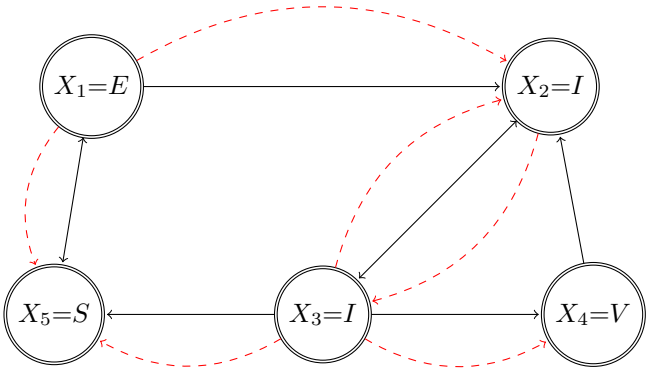

Fig. 1. An example of a 5 node network with underlying directed contact graph shown by solid black lines and effects of infection shown by dashed red lines.

Let δ_i be the rate of recovery when node i is infected. Let γ_i be the rate of becoming susceptible once the node has recovered. Let $\beta_{E,i}$ and $\beta_{I,i} \leq \beta_{E,i}$ correspond to the rates of susceptible node i being exposed through a contact graph $\mathcal{G}$ by an exposed or infected node, respectively. Let ε_i be the rate at which the exposed node becomes infected and let θ_i be the rate at which the susceptible node becomes vigilant. All the infection parameters are nonnegative.

The dynamics of the epidemic spread is then modeled

using the definition of the infinitesimal generator from [20],

$$\begin{aligned}
&P(X_i(t') = E | X_i(t) = S, X(t)) \approx \beta_{E,i}\Delta t Y_i(t) + \beta_{I,i}\Delta t Z_i(t),\\
&P(X_i(t') = V | X_i(t) = S, X(t)) \approx \theta_i \Delta t,\\
&P(X_i(t') = I | X_i(t) = E, X(t)) \approx \varepsilon_i \Delta t,\\
&P(X_i(t') = V | X_i(t) = I, X(t)) \approx \delta_i \Delta t,\\
&P(X_i(t') = S | X_i(t) = V, X(t)) \approx \gamma_i \Delta t,
\end{aligned} \tag{1}$$

where $t' = t + \Delta t$, and

$$\begin{aligned}
Y_i(t) &= \sum_{j \in \mathcal{N}_i^{\text{in}}} \mathbf{1}_{X_j(t)=E},\\
Z_i(t) &= \sum_{j \in \mathcal{N}_i^{\text{in}}} \mathbf{1}_{X_j(t)=I}.
\end{aligned}$$

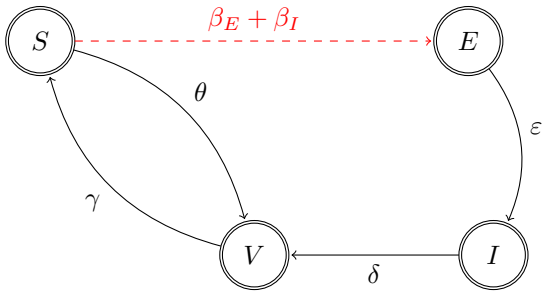

Fig. 2. Stochastic compartmental model for node 5 with one exposed and infected neighbor as shown in Figure 1.

Figure 2 shows the stochastic compartmental model for node 5, which is affected by one exposed and one infected node as seen in Figure 1. In this figure the solid black lines show the internal state transitions and the dashed red line corresponds to the node having one or more infected or exposed neighbors.

The state of the entire network $X(t)$ then lives in a 4^N dimensional space making it very hard to analyze directly. Instead, we utilize a mean-field approximation to reduce the complexity of the entire system. We do this by replacing Y_i and Z_i in (1) by their expected values $E[Y_i]$ and $E[Z_i]$, respectively.

We denote by $[S_i(t), E_i(t), I_i(t), V_i(t)]^T$ the probability vector associated with node i being in each of these states, i.e.,

$$\begin{aligned}
S_i(t) + E_i(t) + I_i(t) + V_i(t) &= 1,\\
S_i(t), E_i(t), I_i(t), V_i(t) &\geq 0,
\end{aligned} \tag{2}$$

for all $i \in \{1, \dots, N\}$. The G-SEIV model we consider in this paper is then given by

$$\begin{aligned}
\dot{S}_i(t) &= \gamma_i V_i(t) - \theta_i S_i(t)\\
&\quad - S_i(t)\left(\sum_{j \in \mathcal{N}_i^{\text{in}}} \beta_{E,i} E_j(t) + \beta_{I,i} I_j(t)\right),\\
\dot{E}_i(t) &= S_i(t)\left(\sum_{j \in \mathcal{N}_i^{\text{in}}} \beta_{E,i} E_j(t) + \beta_{I,i} I_j(t)\right) - \varepsilon_i E_i(t),\\
\dot{I}_i(t) &= \varepsilon_i E_i(t) - \delta_i I_i(t),\\
\dot{V}_i(t) &= \delta_i I_i(t) + \theta_i S_i(t) - \gamma_i V_i(t),
\end{aligned} \tag{3}$$

It is easy to see that

$$\dot{S}_i(t) + \dot{E}_i(t) + \dot{I}_i(t) + \dot{V}_i(t) = 0 \tag{4}$$

for all $i \in \{1, \dots, N\}$ and $t \in \mathbb{R}_{\geq 0}$.

For simplicity, we now consider only identical infection parameters $\delta, \gamma, \beta_E, \beta_I, \varepsilon, \theta$ for all nodes $i \in \{1, \dots, N\}$. We refer to this as the homogeneous parameters case. We will later relax this assumption.

Due to constraints (2) and (4), one of the equations (3) is redundant. By setting $S_i(t) = 1 - E_i(t) - I_i(t) - V_i(t)$, we can describe the system by

$$\begin{aligned}
\dot{E}_i(t) &= (1 - E_i(t) - I_i(t)\\
&\quad - V_i(t))\left(\sum_{j \in \mathcal{N}_i^{\text{in}}} \beta_E E_j(t) + \beta_I I_j(t)\right) - \varepsilon E_i(t)\\
\dot{I}_i(t) &= \varepsilon E_i(t) - \delta I_i(t)\\
\dot{V}_i(t) &= \delta I_i(t) + \theta(1 - E_i(t) - I_i(t) - V_i(t)) - \gamma V_i(t).
\end{aligned} \tag{5}$$

III. Stability analysis of G-SEIV model

We are interested in studying the disease-free equilibrium of (5) defined by $E_i(t) = I_i(t) = 0$. However, this does not mean that $V_i(t)$ will go to 0 as it depends on the parameters θ and γ. Thus, we find a change of coordinates by solving for the equilibrium point V_i^e. Solving for V_i^e in

$$0 = \delta I_i(t) + \theta(1 - E_i(t) - I_i(t) - V_i^e(t)) - \gamma V_i^e(t),$$

we get

$$V_i^e(t) = \frac{\theta + (\delta - \theta) I_i(t) - \theta E_i(t)}{\theta + \gamma}.$$

For simplicity, we drop the explicit dependence on time when not important. This gives us a suitable coordinate change

$$r_i = V_i - \frac{\theta + (\delta - \theta) I_i - \theta E_i}{\theta + \gamma}. \tag{6}$$

Then, the system (5) can be rewritten in the new coordinates as

$$\dot{E}_i = \Big(\frac{\gamma}{\theta+\gamma} - \frac{\gamma}{\theta+\gamma}E_i - \frac{\delta+\gamma}{\theta+\gamma}I_i - r_i\Big)\sum_{j\in\mathcal{N}_i^{\text{in}}}(\beta_E E_j + \beta_I I_j) - \varepsilon E_i$$
$$\dot{I}_i = \varepsilon E_i - \delta I_i$$
$$\dot{r}_i = -\frac{\varepsilon\delta}{\theta+\gamma}E_i - \frac{\delta(\theta-\delta)}{\theta+\gamma}I_i - (\theta+\gamma)r_i + \frac{\theta\gamma}{(\theta+\gamma)^2}\sum_{j\in\mathcal{N}_i^{\text{in}}}(\beta_E E_j + \beta_I I_j) - \frac{\theta}{\theta+\gamma}\Big(\Big(\frac{\gamma}{\theta+\gamma}E_i + \frac{\delta+\gamma}{\theta+\gamma}I_i + r_i\Big)\sum_{j\in\mathcal{N}_i^{\text{in}}}(\beta_E E_j + \beta_I I_j)\Big).$$

Letting $E = [E_1^T, \ldots, E_N^T]^T$, $I = [I_1^T, \ldots, I_N^T]^T$, and $r = [r_1^T, \ldots, r_N]^T$, we are able to rewrite the state equations separating the linear and nonlinear components

$$\begin{bmatrix} \dot{E} \\ \dot{I} \\ \dot{r} \end{bmatrix} = Q\begin{bmatrix} E \\ I \\ r \end{bmatrix} + \begin{bmatrix} H_E \\ 0 \\ H_r \end{bmatrix}, \tag{7}$$

where Q is defined as

$$\begin{bmatrix} \frac{\gamma}{\theta+\gamma}\beta_E A - \varepsilon\mathbf{I} & \frac{\gamma}{\theta+\gamma}\beta_I A & 0 \\ \varepsilon\mathbf{I} & -\delta\mathbf{I} & 0 \\ \frac{1}{\theta+\gamma}(\frac{\theta\gamma}{\theta+\gamma}\beta_E A - \varepsilon\delta\mathbf{I}) & \frac{1}{\theta+\gamma}(\frac{\theta\gamma}{\theta+\gamma}\beta_I A - \delta(\theta-\delta)\mathbf{I}) & -(\theta+\gamma)\mathbf{I} \end{bmatrix},$$

with $\mathbf{I}$ being the N-dimensional identity matrix and

$$H_{E,i} = -\Big(\frac{\gamma}{\theta+\gamma}E_i + \frac{\delta+\gamma}{\theta+\gamma}I_i + r_i\Big)\sum_{j\in\mathcal{N}_i^{\text{in}}}(\beta_E E_j + \beta_I I_j) \tag{8}$$

$$H_{r,i} = -\frac{\theta}{\theta+\gamma}\Big(\Big(\frac{\gamma}{\theta+\gamma}E_i + \frac{\delta+\gamma}{\theta+\gamma}I_i + r_i\Big)\sum_{j\in\mathcal{N}_i^{\text{in}}}(\beta_E E_j + \beta_I I_j)\Big). \tag{9}$$

Using (6) to plug V_i back into (8) and (9), we get

$$H_{E,i} = -(E_i + I_i + V_i)\sum_{j\in\mathcal{N}_i^{\text{in}}}(\beta_E E_j + \beta_I I_j) \tag{10}$$

$$H_{r,i} = -\frac{\theta}{\theta+\gamma}\left((E_i + I_i + V_i)\sum_{j\in\mathcal{N}_i^{\text{in}}}(\beta_E E_j + \beta_I I_j)\right). \tag{11}$$

Since $E_i, I_i, V_i \geq 0$ for all $i \in \{1, \ldots, N\}$ at all times, we see that $H_{E,i}, H_{r,i} \leq 0$ at all times. Letting $\mathbf{X} = [E^T, I^T, r^T]^T$ and $H = [H_E^T, \mathbf{0}, H_r^T]^T$, this means that the linear system

$$\dot{\mathbf{X}} = Q\mathbf{X} \tag{12}$$

upper bounds the original nonlinear system (7),

$$Q\mathbf{X} + H \leq Q\mathbf{X}. \tag{13}$$

Thus, if we can find conditions for which Q is Hurwitz, we have found the necessary and sufficient condition such that the origin of the nonlinear system (7) is globally exponentially stable, and thus the disease-free set of the original system (3) is as well. Sufficiency follows from (13) and necessity follows from [21, Theorem 4.15].

The following result characterizes the condition for which Q is Hurwitz causing the disease-free equilibrium to be globally exponentially stable.

Theorem III.1 (Global exponential stability of disease-free equilibrium) *Given a directed contact graph $\mathcal{G}$ with adjacency matrix A, the disease-free equilibrium of* (3) *with homogeneous parameters is globally exponentially stable if and only if*

$$\lambda_{max}(A) < \frac{\delta\varepsilon(\theta+\gamma)}{\gamma(\delta\beta_E + \varepsilon\beta_I)}. \tag{14}$$

Proof: It is clear that there are N eigenvalues of Q given by $\lambda = -(\theta+\gamma)$. The remaining $2N$ eigenvalues come from the matrix

$$Q_{12} = \begin{bmatrix} \frac{\gamma}{\theta+\gamma}\beta_E A - \varepsilon\mathbf{I} & \frac{\gamma}{\theta+\gamma}\beta_I A \\ \varepsilon\mathbf{I} & -\delta\mathbf{I} \end{bmatrix} \in \mathbb{R}^{2N\times 2N},$$

Let $\mathbf{X}_{12} = [E^T, I^T]^T$, since dynamics of $\mathbf{X}_{12}$ don't depend on r,

$$\dot{\mathbf{X}}_{12} = Q_{12}\mathbf{X}_{12}.$$

In the case that A is symmetric (i.e., the contact graph is undirected), there exists $A = \mathbf{V}D\mathbf{V}^T$ where $\mathbf{V}^T\mathbf{V} = \mathbf{I}$ and $D = \text{diag}\,(d_1, \ldots, d_N)$ where $d_i = \lambda_i(A)$. Then,

$$\mathcal{V}^T\dot{\mathbf{X}}_{12} = \mathcal{V}^T Q_{12}\mathcal{V}\mathcal{V}^T\mathbf{X}_{12},$$

where

$$\mathcal{V} = \begin{bmatrix} \mathbf{V} & 0 \\ 0 & \mathbf{V} \end{bmatrix}$$

Letting $\mathbf{X}_3 = [(\mathbf{V}^T E)^T, (\mathbf{V}^T I)^T]^T$,

$$\dot{\mathbf{X}}_3 = W\mathbf{X}_3$$

where $W = \text{diag}\,(W_1, \ldots, W_N)$ is a $2N \times 2N$ block diagonal matrix with

$$W_i = \begin{bmatrix} \frac{\gamma}{\theta+\gamma}\beta_E d_i - \varepsilon & \frac{\gamma}{\theta+\gamma}\beta_I d_i \\ \varepsilon & -\delta \end{bmatrix}$$

for $i \in \{1, \ldots, N\}$.

The eigenvalues of W_i are then given by

$$\lambda(W_i) = \frac{1}{2}\left(\frac{\gamma}{\theta+\gamma}\beta_E d_i - (\delta+\varepsilon)\right) \pm \frac{1}{2}\left[\left(\delta+\varepsilon-\frac{\gamma}{\theta+\gamma}\beta_E d_i\right)^2 - 4\left(\varepsilon\delta - \frac{\gamma}{\theta+\gamma}(\delta\beta_E + \varepsilon\beta_I)d_i\right)\right]^{1/2}.$$

From this we can see that for the real parts of the eigenvalues to be negative we need

$$d_i < \max\left\{\frac{(\theta+\gamma)(\delta+\varepsilon)}{\gamma\beta_E}, \frac{\delta\varepsilon(\theta+\gamma)}{\gamma(\delta\beta_E+\varepsilon\beta_I)}\right\},$$

for all $i \in \{1,\dots,N\}$. Finally, noticing that

$$\frac{\delta\varepsilon(\theta+\gamma)}{\gamma(\delta\beta_E+\varepsilon\beta_I)} \leq \frac{(\theta+\gamma)(\delta+\varepsilon)}{\gamma\beta_E},$$

justifies the result.

In the case that A is not symmetric, we are not able to analytically compute the eigenvalues of Q_{12} exactly. Let

$$Q_{12} = \begin{bmatrix} w & x \\ y & z \end{bmatrix},$$

where

$$\begin{aligned} w &= \frac{\gamma}{\theta+\gamma}\beta_E A - \varepsilon\mathbf{I}, \\ x &= \frac{\gamma}{\theta+\gamma}\beta_I A, \\ y &= \varepsilon\mathbf{I}, \\ z &= -\delta\mathbf{I}. \end{aligned}$$

We can then reorder the states $\mathbf{X}_{12}$ such that

$$\dot{\mathbf{X}}_{21} = Q_{21}\mathbf{X}_{21},$$

where $\mathbf{X}_{21} = [I^T, E^T]^T$, and

$$Q_{21} = \begin{bmatrix} z & y \\ x & w \end{bmatrix}.$$

Using an LU block decomposition, this is

$$Q_{21} = \begin{bmatrix} \mathbf{I} & 0 \\ xz^{-1} & \mathbf{I} \end{bmatrix}\begin{bmatrix} z & 0 \\ 0 & w - xz^{-1}y \end{bmatrix}\begin{bmatrix} \mathbf{I} & z^{-1}y \\ 0 & \mathbf{I} \end{bmatrix}.$$

It is clear that the first and last matrices have $2N$ eigenvalues at 1. The matrix Q_{21} is then Hurwitz if and only if the middle matrix is Hurwitz. Since this matrix is diagonal and $z = -\delta\mathbf{I}$ is clearly Hurwitz, we are only interested in conditions when

$$\begin{aligned} w - xz^{-1}y &= \frac{\gamma}{\theta+\gamma}\beta_E A - \varepsilon\mathbf{I} + \frac{1}{\delta}\frac{\gamma}{\theta+\gamma}\beta_I\varepsilon A \\ &= \frac{\gamma}{\theta+\gamma}\left(\beta_E + \frac{\varepsilon}{\delta}\beta_I\right)A - \varepsilon\mathbf{I} \end{aligned}$$

is Hurwitz. From here it is easy to see that we arrive at the same condition (14) as the undirected case. ∎

We now consider the original dynamics (3) with heterogeneous parameters. By associating different parameters at each node, we are able to capture the fact that different individuals may have different levels of tolerance against a particular disease or infection due to varying personalities or bodies. We assume that the parameters are bounded for all i,

$$\begin{aligned} &\delta^- \leq \delta_i \leq \delta^+, && \gamma^- \leq \gamma_i \leq \gamma^+, \\ &\beta_E^- \leq \beta_{E,i} \leq \beta_E^+, && \beta_I^- \leq \beta_{I,i} \leq \beta_I^+, \\ &\varepsilon^- \leq \varepsilon_i \leq \varepsilon^+, && \theta^- \leq \theta_i \leq \theta^+. \end{aligned} \tag{15}$$

The following characterizes our result that extends Theorem III.1 to the heterogeneous case. Its proof has been omitted due to space restrictions.

Theorem III.2 (Global exponential stability for heterogeneous parameters) *Given a directed contact graph $\mathcal{G}$ with adjacency matrix A, the disease-free equilibrium of* (3) *is globally exponentially stable if*

$$\lambda_{max}(A) < \frac{\delta^+\varepsilon^+\theta^+}{\gamma^-(\delta^-\beta_E^- + \varepsilon^-\beta_I^-)} + \frac{\delta^+\varepsilon^+}{\delta^-\beta_E^- + \varepsilon^-\beta_I^-}. \tag{16}$$

We note here that the result of Theorem III.2 is only a sufficient condition unlike that of Theorem III.1. This is due to the bounds (15) used to ensure the matrix Q is Hurwitz. The following necessary condition is obtained in a similar fashion by exchanging the upper and lower bounds in (16).

Corollary III.3 (Necessary condition for global exponential stability) *Given a directed contact graph $\mathcal{G}$ with adjacency matrix A, if the disease-free equilibrium of* (3) *is globally exponentially stable, then*

$$\lambda_{max}(A) < \frac{\delta^-\varepsilon^-\theta^-}{\gamma^+(\delta^+\beta_E^+ + \varepsilon^+\beta_I^+)} + \frac{\delta^-\varepsilon^-}{\delta^+\beta_E^+ + \varepsilon^+\beta_I^+}.$$

IV. Simulations

For our simulations we consider an undirected Erdos-Renyi graph with $N = 40$ nodes and connection probability $p = 0.1$ defined by adjacency matrix A. For the particular graph we consider, we have $\lambda_{\max}(A) = 4.5111$. For simplicity, we consider homogeneous infection parameters. We leave the parameters $\delta = 1, \varepsilon = 0.9, \gamma = 0.25$, and $\theta = 0.25$ fixed and vary the infection rates β_I and $\beta_E = \beta_I + 0.1$.

We denote by

$$s = \lambda_{\max}(A)\frac{\gamma(\delta\beta_E + \varepsilon\beta_I)}{\delta\varepsilon(\theta+\gamma)}$$

the strength of the infection for a given graph and infection parameters. Note that the condition of Theorem III.1 is equivalent to $s < 1$. In Figure 3 we show the trajectories of the infection over time for varying parameters of β_I and $\beta_E = \beta_I + 0.1$. We use uniformly random initial conditions for all 40 nodes of the network and look at the total probability of infection for each node $P_i(t) = E_i(t) + I_i(t)$. We plot the minimum and maximum probabilities of infection over time given by $\min_{i\in\{1,\dots,N\}} P_i(t)$ and $\max_{i\in\{1,\dots,N\}} P_i(t)$, respectively, and the average given by $\frac{1}{N}\sum_{i=1}^N P_i(t)$.

In Figure 3 we consider $\beta_I = 0.05$ ($s = 0.5430$), $\beta_I = 0.40$ ($s = 2.3948$), and $\beta_I = 1.10$ ($s = 6.0983$). As supported by Theorem III.1, we see that in Figure 3(a) the epidemic dies out exponentially, but in (b) and (c) it reaches a nontrivial equilibrium that grows with s.

In Figure 4 we vary β_I from 0 to 1 with $\beta_E = \beta_I + 0.1$ and plot the steady state values of the minimum, maximum, and average probabilities of infection. The bifurcation that occurs at $s = 1$ is supported by Theorem III.1.

V. Conclusions

In this paper we have proposed an epidemic model that generalizes many known models in the literature including the well known SIS and SIR models. Further we consider

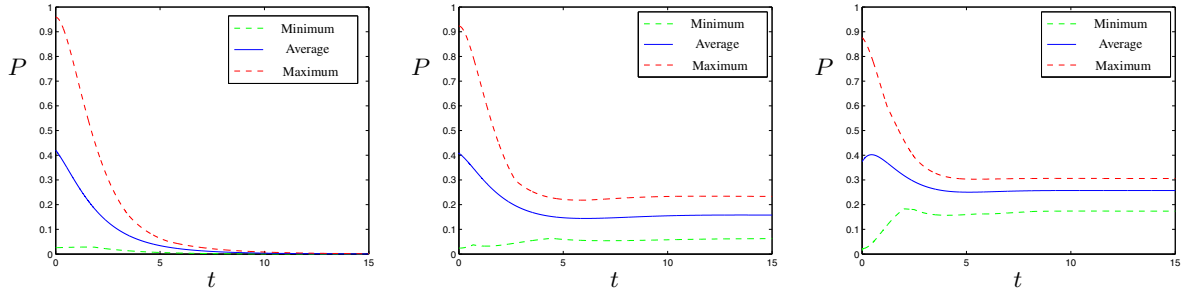

Fig. 3. Plots of the minimum, maximum, and average probabilities of infection over time for (a) $\beta_I = 0.05$, (b) $\beta_I = 0.40$, and (c) $\beta_I = 1.10$.

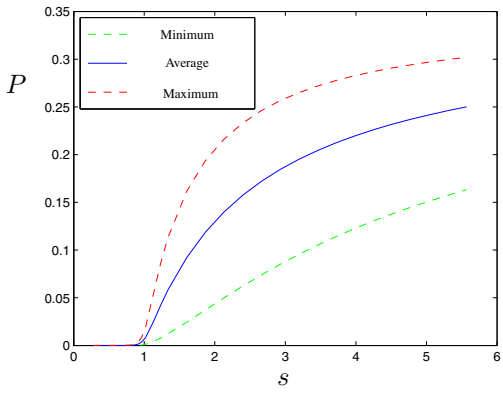

Fig. 4. Plots of the minimum, maximum, and average probabilities of infection $P_i = E_i + I_i$ at steady state for various infection strengths s.

both arbitrary directed graphs and non-homogeneous infection parameters to further amplify the applicability of the proposed model. We have rigorously studied the disease-free equilibrium of the mean-field approximation of the model and identified the necessary and sufficient condition for global exponential stability in the case of homogeneous parameters. For heterogeneous parameters, we are able to show both a sufficient condition and a separate necessary condition for global exponential stability. We illustrate the results through simulations of a 40 node Erdos-Renyi graph.

In future works involving analysis we are interested in determining the existence of a nontrivial non-disease-free equilibrium in the case that the conditions of this paper are not satisfied and studying the stability of this equilibrium. We are also interested in using this analysis to facilitate control of the epidemic given different measurements of the state and methods of control.

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