2019

Rigorous Analysis of an Edge-Based Network Disease Model

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RIGOROUS ANALYSIS OF AN EDGE-BASED NETWORK DISEASE MODEL

by

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A thesis submitted in partial fulfillment of the requirements
for the Honors in the Major Program
in the Department of Mathematics
in the College of Sciences
at the University of Central Florida
Orlando, Florida

Spring Term
2019

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ABSTRACT

Edge-based network disease models, in comparison to the classic compartmental epidemiological models, better capture social factors affecting disease spread such as contact duration and social heterogeneity. We reason that there should exist infinitely many equilibria rather than only an endemic equilibrium and a disease-free equilibrium for the edge-based network disease model commonly used in the literature, as there do not exist any changes in demographic in the model. We modify the commonly used network model by relaxing some assumed conditions and factor in a dependency on initial conditions. We find that this modification still accounts for realistic dynamics of disease spread (such as the probability of contracting a disease based off your neighbors' susceptibility to the disease) based on the basic reproduction number. Specifically, if the basic reproduction number is below 1, then the infection dies out; while if the basic reproduction number is above 1, then there is possibility of an epidemic.

ACKNOWLEDGEMENTS

Thanks go to Dr. Zhisheng Shuai for his incredible patience and guidance.
Thanks go to Mr. Keith Carlson for his outlook and knowledge.
Thanks also goes to Kevin McCarthy and Connor Smith for their support and cookies throughout the research process.

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INTRODUCTION

Compartmental models have been widely used in mathematical epidemiology to understand the progression of communicable diseases. The observed population is partitioned into individual compartments, classified by their stage in the disease. Kermack and McKendrick first introduced their SIR model in a series of papers [1]–[3] as a means to describe the population flow between compartments or groups of individuals who are in the separate stages of susceptibility (S), infectiousness (I), and recovery (R).

The Kermack-McKendrick mass-action SIR model is simplistic in that dynamics into and out of compartments can be represented with a handful of ordinary differential equations, as depicted in Figure 1. The total population, N, is the summation of the populations in S, I, and R, yielding the base equation $N = S + I + R$. Population dynamics out of and into each compartment are considered with the following equations:

$$\dot{S} = -\beta IS, \quad \dot{I} = \beta IS - \gamma I, \quad \text{and} \quad \dot{R} = \gamma I,$$

where β is the constant rate of infection and γ is the constant rate of recovery.

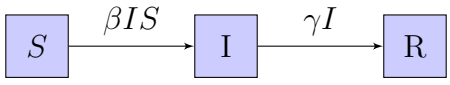

Figure 1: Kermack and McKendrick's mass-action SIR compartmental model

There have been numerous modifications of this core model. Some examples of modifications include differing populations, an inclusion of an exposure period in the SEIR model, and the return to susceptibility after infection, rather than recovery, in the SIS model; see, for example, [1], [4], [5].

Although the Kermack-McKendrick model has been incredibly useful in understanding

disease spread in relation to time, it can be too simplistic for capturing realistic social behaviors. This model assumes homogeneous mixing among individuals, and a fixed contact rate and duration. More realistic models would ideally account for variances in social dynamics, such as time-variable partnerships and differences in the number of contacts between individuals.

The epidemic processes of the compartmental model have classically been deterministic, in which randomness is not accounted for and that behaviors are largely constant. Stochastic models, on the other hand, account for this randomness through "probabilistic concepts" and "a distribution of possible behaviors" [5].

In relatively recent years, advances in stochastic epidemic compartmental models have involved network sciences. It is useful to consider an individual's sources of infections through a network model. Rather than considering flows of whole populations through the epidemic process, network disease models place emphasis on the individual's connections [6].

In network models, each individual is represented as a *node*, and connections with other individuals are represented as *edges*. These edges oftentimes represent real partnerships between individuals, making this approach particularly useful when studying diseases driven by social decisions such as sexually transmitted infections (STIs). The individuals a particular individual is connected to will be referred to as *contacts*, *neighbors*, or simply *partners*.

With a network approach to the compartmental model, it becomes easier to account for variations in mixing rates and behaviors in a population. In Joel C. Miller et. al's paper "Edge-based compartmental modelling for infectious disease spread" [7], they reason that to find the proportions of the populations that are susceptible, infected, and recovered, we can seek to understand the probability that some random individual contracts infection. This is equivalent to finding the probability that no partner of some randomly chosen test node u has ever transmitted infection to it. These probabilities are influenced by how many partners u has, how often their contacts change, and the probability that the partners are infected at

a given time. This is when heterogeneous mixing and variant contact rates come into effect [8]–[11].

Miller et. al formulate an edge-based compartmental model with relatively simple assumptions about mixing and contact rates that they name the "Configuration Model". It is assumed that the network is static i.e. there are no changes in demographics resulting from births, non-disease deaths, or travel. From this model comes derivative models with variations in mixing and contact variables.

While current edge-based network disease models can be mathematically concise, capturing the dynamics of a whole system into a single differential equation, they only account for an endemic equilibrium and a disease-free equilibrium. As this model does not incorporate changes in demographics, we reason that the model should consider infinitely many equilibria [5], [12]. We generalize some concrete assumptions made and formulate a two-dimensional system to better capture the social dynamics of a network-based model. We then perform a rigorous analysis on the modified model, showing variance in behaviors of the infinitely many equilibria.

NETWORK EDGE-BASED COMPARTMENTAL MODEL

In this model, it is essential to fix our focus on the probabilities that a test node's neighbor is infected due to the likelihood that a neighbor has more contacts than a certain individual [13].

We begin by creating a network with N nodes in which some node u's degree, k_u, is assigned by some probability $P(k_u)$, and u is then given k_u stubs, or half-edges. Stubs are then randomly paired throughout the network to create partnerships between different nodes.

The probability that the test node u has a degree of k is given by $P(k)$, while the probability that a partner of test node u, which we shall refer to as v, has a degree of k is given by $P_v(k)$. The probability that one of u's stubs connects to a stub of v is proportional to k_v. Therefore we can find $P_v(k)$ to be

$$\begin{aligned} P_v(k) &= (\text{total number of degree-k stubs}) \div (\text{total number of network stubs}) \\ &= \frac{kP(k)N}{\sum_k kP(k)N} \\ &= \frac{kP(k)N}{\langle K \rangle N}, \text{ where } \langle K \rangle = \sum_k kP(k), \\ &= \frac{kP(k)}{\langle K \rangle}. \end{aligned}$$

We also define the following:

$$\begin{aligned} S = S(t) &= \text{proportion of population that is susceptible at time t,} \\ I = I(t) &= \text{proportion of population that is infected at time t,} \\ R = R(t) &= \text{proportion of population that is recovered at time t.} \end{aligned}$$

Note that these proportions are equivalent to the probabilities that u is in a given state, e.g.,

the probability that a random node is susceptible at time t is given by $S(t)$. Also note that $S(t)$ is ultimately the probability that none of u's partners have transmitted infection to u (if u is in the infected or recovered stages, one of its neighbors has clearly passed infection to it).

Define the probability that a randomly chosen partner v of u has *not* transmitted infection to u as $\theta(t)$. Note that this is different from simply finding the probability that v has obtained the infection, as it is possible for v to contract infection and not transmit to u. We find that the probability that u is susceptible, $s(t)$, is dependent on $\theta(t)$ and the degree of u, or the number of partners it has. For every additional partner u has, the probability one of its neighbors does *not* transmit infection compounds, and ultimately decreases. Thus,

$$s(k, \theta(t)) = \theta(t)^k.$$

We can then derive an equation for $S(t)$, which is akin to a probability generating function:

$$S(t) = \sum_k P(k) s(k, \theta(t)) = \psi(\theta(t)).$$

The flux between the compartments of populations that belong in S, I, and R follow the mass-action model; see Figure 2.

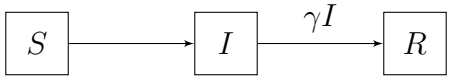

Figure 2: Flow/probability flux diagram of individuals on a static network, where γ is rate of recovery. Note that, unlike the mass-action model, there is no rate of infection term in the individual case as it is dependent on the number of connections each individual has and, thus, cannot be generalized in this model.

Define

$$\phi_S = P(\text{v is susceptible but has not transmitted infection}),$$
$$\phi_I = P(\text{v is infected but has not transmitted infection}),$$
$$\phi_R = P(\text{v is recovered but has not transmitted infection}).$$

Note that ϕ_I is the state when a partner has contracted infection but has not transmitted. The partner then can either transmit or recover before transmission.

We then find $\theta(t)$, the probability that neighbor v of u has not transmitted infection to u to simply be

$$\theta(t) = \phi_S(t) + \phi_I(t) + \phi_R(t). \tag{1}$$

Figure 3 represents the flow of the states a neighbor v can be in at time t. The rate of recovery is constant, with rate γ. The flux into ϕ_R is then $\gamma\phi_I$, so that $\dot{\phi_R} = \gamma\phi_I$. Flow between the ϕ_I and $1-\theta$ compartments represent v transmitting infection to u, which happens at rate β. The flux from ϕ_I to $1-\theta$ is $\beta\phi_I$, so flow out of the $1-\theta$ compartment into $\theta = \phi_S + \phi_I + \phi_R$ would be $-\beta\phi_I$. Thus, $\dot{\theta} = -\beta\phi_I$.

We now examine the probability that v is susceptible. This process is similar to the

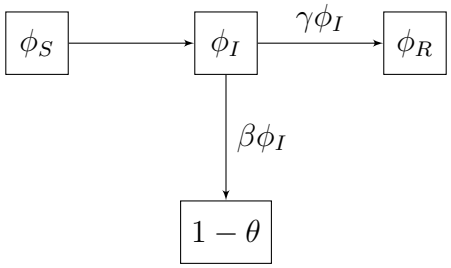

Figure 3: Flow/probability flux diagram for partners of u. β is constant in this context, as this flux diagram captures interactions between two specific individuals rather than generalizing across the network. β cannot be approximated across the network, as the rate of infection relies on the number of partners the neighbor has.

derivation of $S(t)$ in that susceptibility is based off the degree of the individual. If v has a degree of k, then it has $k-1$ possible paths of disease transmission since that u does not transmit to its neighbors. Taking the weighted average,

$$\begin{aligned}\phi_S &= \sum_k P_v(k)\theta^{k-1} \\ &= \sum_k \frac{kP(k)}{\langle K\rangle}\theta^{k-1} \\ &= \frac{\psi'(\theta)}{\psi'(1)}.\end{aligned}$$

For $\dot{\theta} = -\beta\phi_I$ and $\dot{\phi_R} = \gamma\phi_I$,

$$\frac{\dot{\theta}}{\dot{\phi_R}} = \frac{-\beta}{\gamma}.$$

We can solve this ordinary differential equation, obtaining

$$\gamma\theta(t) - \gamma\theta(0) = -\beta\phi_R(t) + \beta\phi_R(0).$$

In [7], it is assumed that at time $t = 0$, $\theta(t) \approx 1$ and $\phi_R(t) \approx 0$. It is then concluded that $\phi_R(t) = \frac{\gamma}{\beta}(1-\theta(t))$ and

$$\begin{aligned}\dot{\theta} &= -\beta\phi_I \\ &= -\beta(\theta - \phi_S - \phi_R) \\ &= -\beta(\theta - \frac{\psi'(\theta)}{\psi'(1)} - \frac{\gamma}{\beta}(1-\theta)) \\ &= -\beta\theta + \beta\frac{\psi'(\theta)}{\psi'(1)} + \gamma(1-\theta).\end{aligned}$$

With these assumptions, $\dot{\theta}$ is able to capture the dynamics of a SIR disease on a randomized social network in one equation.

The assumptions made to formulate ϕ_R are only correct when there is no disease at

the beginning of simulations. If there is already disease present at $t = 0$, then it cannot be assumed that $\theta(0) = 1$ or $\phi_R(0) = 0$. Furthermore, there are only two equilibria for this model: an endemic equilibrium and a disease-free equilibrium. For a model that does not account for demographic changes, it would be expected that there are infinitely many equilibria for the system for the infinitely many possible initial conditions.

To account for these inconsistencies, we modify the model by relaxing the assumptions on $\theta(t)$ and $\phi_R(t)$ at $t = 0$.

2.1 Model Formulation

Rather than solve the ordinary differential equations for $\dot{\theta}$ and $\dot{\phi_R}$, we simply maintain that $\dot{\phi_R} = \gamma\phi_I$ and $\dot{\theta} = -\beta\phi_I$. Substituting in $\phi_I = \theta - \phi_S - \phi_R$, we get the following system that shall serve as our new model:

$$\begin{aligned} \dot{\theta} &= -\beta\phi_I = -\beta\left(\theta - \frac{\psi'(\theta)}{\psi'(1)} - \phi_R\right), \\ \dot{\phi_R} &= \gamma\phi_I = \gamma\left(\theta - \frac{\psi'(\theta)}{\psi'(1)} - \phi_R\right). \end{aligned} \tag{2}$$

2.2 Model Analysis

2.2.1 Positively Invariant Region

To show our refined model is valid, we find a positively invariant region such that all solutions will obey biological realities. We begin this approach by defining

$$g(\theta) = \theta - \phi_S = \theta - \frac{\psi'(\theta)}{\psi'(1)}. \tag{3}$$

To make certain our model is biologically feasible, we require that $g(\theta) \geq 0$, as $g(\theta) = \phi_I + \phi_R$. Since $g(1) = 1 - \frac{\psi'(1)}{\psi'(1)} = 0$, we require that $g'(\theta) < 0$ for θ near 1. That is, $g'(1) = 1 - \frac{\psi''(1)}{\psi'(1)} < 0$,

implying $\frac{\psi''(1)}{\psi'(1)} > 1$. We rely on assumption that $\frac{\psi''(1)}{\psi'(1)} > 1$ to ensure our feasible region is positive.

The following lemma is required to define the feasible region.

Lemma 1. *Assume* $\frac{\psi''(1)}{\psi'(1)} > 1$. *Then*

(i) There exists a $\bar{\theta}$ *such that* $\bar{\theta} = \sup\{\hat{\theta} \in (0,1) \,|\, g(\hat{\theta}) = 0\}$.

(ii) $g'(\bar{\theta}) \geq 0$.

Proof.

(i) Given $\epsilon = -\frac{g'(1)}{2} > 0$, there exists $\delta > 0$ such that for all $h \in (0, \delta)$,

$$\left| \frac{g(1) - g(1-h)}{h} - g'(1) \right| < \epsilon$$
$$-\epsilon < \frac{g(1) - g(1-h)}{h} - g'(1) < \epsilon$$
$$-\epsilon + g'(1) < \frac{g(1) - g(1-h)}{h} < \epsilon + g'(1)$$

$$\frac{g'(1)}{2} + g'(1) < \frac{g(1) - g(1-h)}{h} < -\frac{g'(1)}{2} + g'(1) = \frac{g'(1)}{2} < 0.$$

This condition holds for all $h \in (0, \delta)$. Choose $h < 1$. Therefore,

$$\frac{g(1) - g(1-h)}{h} < 0.$$

But since $h > 0$,

$$g(1) - g(1-h) < 0$$
$$g(1) < g(1-h).$$

Recall that $g(1) = 0$, so $0 < g(1-h)$. For some particular $h^* \in (0, \delta)$, let $\theta^* = 1 - h^*$. Then $g(\theta^*) > 0$. Since $g(0) < 0$ and $g(\theta^*) > 0$ for $\theta^* > 0$, by continuity, there exists $\hat{\theta} \in (0, \theta^*)$ such that $g(\theta^*) = 0$. Thus, there exists at least one root on $(0, 1)$. It suffices to define

$$\bar{\theta} = \sup\{\hat{\theta} \in (0,1) \,|\, g(\hat{\theta}) = 0\}.$$

(ii) Since $g(1) = 0$ and $g'(1) < 0$, then there must exist some $\theta_1 \in (1-\epsilon, 1)$ such that $g(\theta_1) > 0$ for some arbitrarily small ϵ.

Suppose, for sake of contradiction, that $g'(\bar{\theta}) < 0$. Then there exists $\theta_0 \in (\bar{\theta}, \bar{\theta} + \delta)$ such that $g(\theta_0) < 0$ for some δ arbitrarily small. Notice that $\theta_0 < \theta_1$ and $g(\theta_1) > 0$. Then there must exist $\theta_c \in (\theta_0, \theta_1)$ such that $g(\theta_c) = 0$.

This is a direct contradiction to the definition of $\bar{\theta}$ being the supremum of all roots of $g(\theta)$. Thus, $g'(\bar{\theta}) \geq 0$.

□

Now we are able to define the feasible region for our model to be

$$\Gamma = \left\{ (\theta, \phi_R) \,|\, \bar{\theta} \leq \theta \leq 1, 0 < \phi_R \leq \theta - \frac{\psi'(\theta)}{\psi'(1)} \right\} \tag{4}$$

where $\bar{\theta}$ is defined as in Lemma 1. We are secured a feasible region that is positive and biologically realistic; see Figure 4.

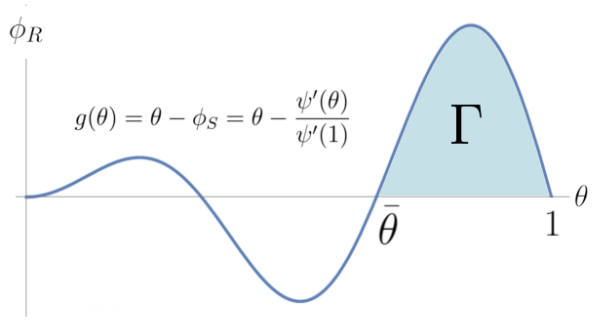

Figure 4: The defined feasible region Γ as bounded above by $\phi_R = g(\theta)$. The shape of the curve should vary based off network structure.

We now show that Γ is positively invariant, or that all initial conditions that begin in Γ shall stay in and have solutions in Γ.

Theorem 1. *Assume* $\frac{\psi''(1)}{\psi'(1)} > 1$. *Then the feasible region* Γ *is positively invariant.*

Proof. Assume that at some time $\bar{t}$, $\phi_R(\bar{t}) = \theta(\bar{t}) - \frac{\psi'(\theta(\bar{t}))}{\phi'(1)}$.

Then

$$\begin{aligned}\dot{\phi_R}(\bar{t}) &= \gamma(\theta(\bar{t}) - \frac{\psi'(\theta(\bar{t}))}{\psi'(1)} - \phi_R(\bar{t})) \\ &= \gamma\left[\theta(\bar{t}) - \frac{\psi'(\theta(\bar{t}))}{\psi'(1)} - \left(\theta(\bar{t}) - \frac{\psi'(\theta(\bar{t}))}{\psi'(1)}\right)\right] \\ &= 0,\end{aligned}$$

and

$$\begin{aligned}\dot{\theta}(\bar{t}) &= -\beta(\theta(\bar{t}) - \frac{\psi'(\theta(\bar{t}))}{\psi'(1)} - \phi_R(\bar{t})) \\ &= -\beta\left[\theta(\bar{t}) - \frac{\psi'(\theta(\bar{t}))}{\psi'(1)} - \left(\theta(\bar{t}) - \frac{\psi'(\theta(\bar{t}))}{\psi'(1)}\right)\right] \\ &= 0.\end{aligned}$$

Thus, all solutions tending toward the curve $\phi_R(t) = \theta(t) - \frac{\psi'(\theta(t))}{\phi'(1)}$ will stop and never leave

the region Γ through it.

Assume that the solution leaves Γ through the θ axis. Then, at some $\hat{t}$, $\phi_R(\hat{t}) = 0$. Thus,

$$\dot{\phi_R}(\hat{t}) = \gamma \left[\theta(\hat{t}) - \frac{\psi'(\theta(\hat{t}))}{\psi'(1)} - \phi_R(\hat{t})\right] = \gamma(\theta(\hat{t}) - \phi_S(\hat{t})).$$

Since for all t, $\theta = \phi_S + \phi_I + \phi_R$, then $\phi_S \leq \theta$, implying $0 \leq \theta - \phi_S$.

As $\gamma > 0$ for all t, then

$$\dot{\phi_R}(\hat{t}) = \gamma(\theta(\hat{t}) - \phi_S(\hat{t})) \geq 0.$$

Therefore, $\dot{\phi_R}(\hat{t}) \geq 0$.

It can also be seen that

$$\dot{\theta}(\hat{t}) = -\beta \left[\theta(\hat{t}) - \frac{\psi'(\theta(\hat{t})}{\psi'(1)} - \phi_R(\hat{t})\right] = -\beta(\theta(\hat{t}) - \phi_S(\hat{t})) < 0.$$

□

2.2.2 Model Dynamics

In mathematical epidemiology, the basic reproductive number is the average number of secondary infections generated over the course of infection. In general, if $\mathcal{R}_0 < 1$, then the disease will die out before infection spreads. If $\mathcal{R}_0 > 1$, then there is a possibility that infection will spread in the population. We define the basic reproductive number of this model to be

$$\mathcal{R}_0 = \frac{\beta}{\beta + \gamma} \frac{\psi''(1)}{\psi'(1)}.$$

The following lemma characterizes $\mathcal{R}_0 > 1$.

Lemma 2. *Assume* $\frac{\psi''(1)}{\psi'(1)} > 1$. *If* $\mathcal{R}_0 > 1$, *then there exists a unique* $\tilde{\theta} \in (\bar{\theta}, 1)$ *such that*

(i) $\beta + \gamma = \beta \frac{\psi''(\tilde{\theta})}{\psi'(1)}$

(ii) $\beta + \gamma > \beta\frac{\psi''(\theta)}{\psi'(1)}$ *for* $\theta \in [\bar{\theta}, \tilde{\theta})$

(iii) $\beta + \gamma < \beta\frac{\psi''(\theta)}{\psi'(1)}$ *for* $\theta \in (\tilde{\theta}, 1]$

Proof.

(i) By assumption, $\mathcal{R}_0 > 1$, thus,

$$\beta\frac{\psi''(1)}{\psi'(1)} > \beta + \gamma.$$

Define $h(\theta) = \beta + \gamma - \beta\frac{\psi''(\theta)}{\psi'(1)}$.

At $\theta = 1$,

$$h(1) = \beta + \gamma - \beta\frac{\psi''(1)}{\psi(1)} < 0,$$

and at $\bar{\theta}$,

$$h(\bar{\theta}) = \beta + \gamma - \beta\frac{\psi''(\bar{\theta})}{\psi(1)} = \beta\left[1 - \frac{\psi''(\bar{\theta})}{\psi(1)}\right] + \gamma.$$

In Lemma 1, it is found that $g'(\bar{\theta}) = 1 - \frac{\psi''(\bar{\theta})}{\psi'(1)} \geq 0$.

Thus,

$$h(\bar{\theta}) \geq 0 + \gamma > 0.$$

Since $\bar{\theta} < 1$, $h(\bar{\theta}) > 0$ and $h(1) < 0$, then there must exist a point $\widetilde{\theta} \in (\bar{\theta}, 1)$ such that

$$h(\widetilde{\theta}) = 0 = \beta + \gamma - \beta\frac{\psi''(\widetilde{\theta})}{\psi'(1)}.$$

Thus, at $\theta = \widetilde{\theta}$, $\beta + \gamma = \beta\frac{\psi''(\widetilde{\theta})}{\psi'(1)}$.

(ii) It can be seen that for all t, $h'(\theta) < 0$ ($h(\theta)$ is monotonically decreasing). Since $h(\bar{\theta}) > 0$ and $h(\widetilde{\theta}) = 0$, then for all $\theta \in [\bar{\theta}, \widetilde{\theta})$, $h(\theta) > 0$ or $\beta + \gamma > \beta\frac{\psi''(\widetilde{\theta})}{\psi'(1)}$.

(iii) Since $h(\widetilde{\theta}) = 0$ and $h(1) < 0$, then for all $\theta \in (\widetilde{\theta}, 1]$, $h(\theta) < 0$ or $\beta + \gamma < \beta\frac{\psi''(\widetilde{\theta})}{\psi'(1)}$. □

From the model and as previously discussed, it can be seen that any point on the curve $\phi_R = g(\theta)$ is an equilibrium point of (2.2.2), denoted as (θ^*, ϕ_R^*). Note that $\phi_R^* = g(\theta^*)$.

The following theorem uses Lemma 2 to classify the stability of the equilibria.

Theorem 2. *Assume* $\frac{\psi''(1)}{\psi'(1)} > 1$.

(i) If $\mathcal{R}_0 < 1$, *then all equilibria* (θ^*, ϕ_R^*) *are stable.*

(ii) If $\mathcal{R}_0 > 1$, *then all equilibria* (θ^*, ϕ_R^*) *with* $\theta^* \in [\bar{\theta}, \widetilde{\theta})$ *are stable, and all equilibria* (θ^*, ϕ_R^*) *with* $\theta^* \in (\widetilde{\theta}, 1]$ *are unstable.*

Proof.

(i) For the system

$$\begin{aligned}
\dot{\theta} &= -\beta \left(\theta(t) - \frac{\psi'(\theta(t))}{\psi'(1)} - \phi_R(t) \right), \\
\dot{\phi_R} &= \gamma \left(\theta(t) - \frac{\psi'(\theta(t))}{\psi'(1)} - \phi_R(t) \right),
\end{aligned}$$

the Jacobian matrix is

$$J = \begin{bmatrix} -\beta + \beta \frac{\psi''(\theta)}{\psi'(1)} & \beta \\ \gamma - \gamma \frac{\psi''(\theta)}{\psi'(1)} & -\gamma \end{bmatrix}.$$

The determinant of the Jacobian matrix is as follows

$$\begin{aligned}
\det J &= \left(-\beta + \beta \frac{\psi''(\theta)}{\psi'(1)} \right) (-\gamma) - \beta(\gamma - \gamma \frac{\psi''(\theta)}{\psi'(1)}) \\
&= \beta\gamma - \beta\gamma \frac{\psi''(\theta)}{\psi'(1)} - \beta\gamma + \beta \frac{\psi''(\theta)}{\psi'(1)} \\
&= 0.
\end{aligned}$$

Since $\det J = 0$, then at least one eigenvalue is zero, say $\lambda_1 = 0$.

The trace τ of a 2×2 matrix is the sum of the two eigenvalues. Since $\lambda_1 = 0$, then $\tau = \lambda_1 + \lambda_2 = 0 + \lambda_2 = \lambda_2$.

If $\tau = 0$, then $\lambda_2 = 0$. Thus $\lambda_1 = \lambda_2 = 0$.

The trace of J for the system is

$$\tau = -\beta + \beta\frac{\psi''(\theta)}{\psi(1)} - \gamma.$$

Recall that when $\mathcal{R}_0 < 1$, $\beta + \gamma > \beta\frac{\psi''(\theta)}{\psi'(1)}$, implying that $-\beta - \gamma + \beta\frac{\psi''(\theta)}{\psi'(1)} = \tau < 0$. Thus, when $\mathcal{R}_0 < 1$, all equilibrium points are stable.

(ii) Recall from Lemma 2 that there exists a $\tilde{\theta} \in (\bar{\theta}, 1)$ such that

$$h(\widetilde{\theta}) = \beta + \gamma - \beta\frac{\psi''(\widetilde{\theta})}{\psi'(1)} = 0.$$

Thus at $\widetilde{\theta}$, $\tau = 0$.

For $\theta \in [\bar{\theta}, \widetilde{\theta})$,

$$h(\theta) = \beta + \gamma - \beta\frac{\psi''(\theta)}{\psi'(1)} > 0,$$
$$\tau = -\beta - \gamma + \beta\frac{\psi''(\theta)}{\psi'(1)} < 0,$$

and for $\theta \in (\widetilde{\theta}, 1]$,

$$h(\theta) = \beta + \gamma - \beta\frac{\psi''(\theta)}{\psi'(1)} < 0,$$
$$\tau = -\beta - \gamma + \beta\frac{\psi''(\theta)}{\psi'(1)} > 0.$$

Therefore, all equilibrium for $\theta \in [\bar{\theta}, \widetilde{\theta})$ are stable as $\tau < 0$ and all equilibrium for $(\widetilde{\theta}, 1]$ are unstable as $\tau > 0$.

□

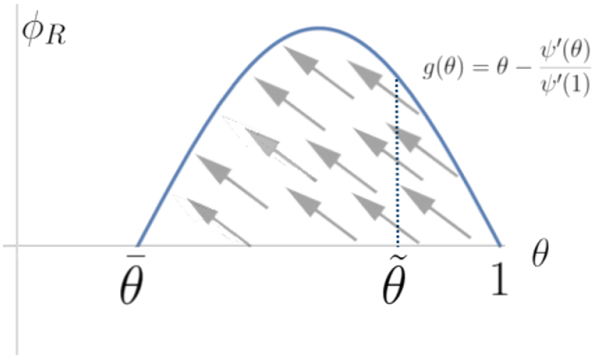

Figure 5: Phase portrait of (2) for $\mathcal{R}_0 > 1$

A phase portrait of the model when $\mathcal{R}_0 > 1$ can be seen in Figure 5. Any initial conditions that start in Γ will remain in Γ (positive invariance). All equilibrium points from $[\bar{\theta}, \widetilde{\theta})$ are stable, while all equilibrium points from $(\widetilde{\theta}, 1]$ are unstable.

2.2.3 Biological Interpretations

In order to biologically interpret our results from Theorem 2, we rewrite system (2.2.2) in terms of θ and ϕ_I. Specifically, it follows from $\phi_I = \theta - \phi_S - \phi_R$ that

$$\begin{aligned}
\dot{\phi_I} &= \dot{\theta} - \dot{\phi_S} - \dot{\phi_R} \\
&= -\beta\phi_I - \frac{\psi''(\theta)}{\psi'(1)}\dot{\theta} - \gamma\phi_I \\
&= -\beta\phi_I - \frac{\psi''(\theta)}{\psi'(1)}(-\beta\phi_I) - \gamma\phi_I \\
&= \phi_I\left(-\beta - \gamma + \beta\frac{\psi''(\theta)}{\psi'(1)}\right).
\end{aligned}$$

Thus, system (2.2.2) is equivalent to the following system:

$$\dot{\theta} = -\beta \left(\theta - \frac{\psi'(\theta)}{\psi'(1)} - \phi_R \right),$$
$$\dot{\phi_I} = \phi_I \left(-\beta - \gamma + \beta \frac{\psi''(\theta)}{\psi'(1)} \right).$$

Now, define our effective reproduction number

$$\mathcal{R}_e = \frac{\beta}{\beta + \gamma} \frac{\psi''(\theta(0))}{\psi'(1)},$$

which incorporates the initial state of $\theta = \theta(t)$. When $\theta \approx 1, \mathcal{R}_e$ becomes $\mathcal{R}_0$.

Theorem 3. *Assume* $\frac{\psi''(1)}{\psi'(1)} > 1$. *Then the following statements hold.*

(i) If $\mathcal{R}_e < 1$, *then* $\phi_I(t)$ *decreases for* $t \geq 0$.

(ii) If $\mathcal{R}_e > 1$, *then* $\phi_I(t)$ *increases and then decreases.*

Proof. Notice that $\dot{\phi_I} < 0$ if and only if $\frac{\beta}{\beta+\gamma} \frac{\psi''(\theta)}{\psi'(1)} < 1$. If $\mathcal{R}_e < 1$, then $\frac{\beta}{\beta+\gamma} \frac{\psi''(\theta(t))}{\psi'(1)} < 1$ for all $t \geq 0$.

If $\mathcal{R}_e > 1$, then $\frac{\beta}{\beta+\gamma} \frac{\psi''(\theta(t))}{\psi'(1)}$ decreases as t increases, and equals 1 whenever $\theta = \widetilde{\theta}$. That is, ϕ_I increases then decreases. □

CONCLUSION AND FUTURE STUDY

By defining a positively invariant region for all initial conditions and their solutions to exist within, we are able to ensure our model will remain biologically and mathematically feasible. We found that when $\mathcal{R}_0 < 1$, then the following are equivalent: all equilibrium points are stable, ϕ_I will always decrease for all time, and the disease on the network will die out before becoming an epidemic. We also found that when $\mathcal{R}_0 > 1$, then the following are equivalent: there exists a $\widetilde{\theta}$ such that all equilibrium points in $[\bar{\theta}, \widetilde{\theta})$ are stable and all initial conditions starting in this range will lead to the disease dying out before becoming an epidemic; and that all equilibrium points in $(\widetilde{\theta}, 1]$ are unstable and all initial conditions starting in this range will lead to the disease becoming an epidemic before dying out.

The endemic equilibrium and the disease-free equilibrium that exist in the commonly used network edge-based disease models also exist as two solutions in our model; see Figure 6. However, we are able to account for infinitely many initial conditions with infinitely many possible solutions. Biologically speaking, we are able to use our model to simulate the spread of infection with any amount of preexisting disease on the network. In this way, we are able to incorporate the intended behavior results of previous edge-based network disease models in a more accommodating approach.

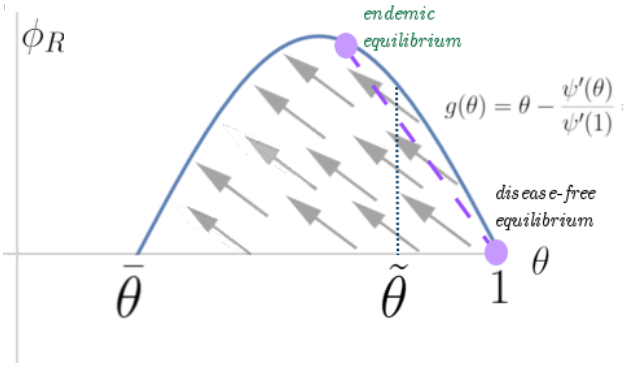

Figure 6: The endemic equilibrium and disease-free equilibrium when $\mathcal{R}_0 > 1$ as solutions in the modified model.

There are a few potential avenues of future study. It is worth investigating the correlation of network structure with the graph of $g(\theta)$. It is possible that different network structures such as scale-free and small-world networks yield different feasible regions whose dynamics warrant future study. Furthermore, numerical simulations may also be run in order to understand the shape of the $g(\theta)$ graphs of randomized networks. We may also take the approach of Miller et. al. in applying variations in social heterogeneity and contact rates, rather than focusing on a static network.

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