SOME ELEMENTARY PROPERTIES OF SIR NETWORKS
OR,
CAN I GET SICK BECAUSE YOU GOT VACCINATED?

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1. Introduction

An epidemic is sometimes said to spread like wildfire. Might it then be controlled by setting backfires? A fire is fueled by the available timber, an epidemic by the available susceptible population. Might an epidemic be averted by preemptively infecting some susceptible individuals?

To make sense of this question, we need to specify a model for disease transmission. The underlying phenomenon in the spread of disease is transmission from an infective to a susceptible individual. In the absence of medical omniscience, this is a probabilistic phenomenon. The nature of the pathogen, the state of health of the individuals and the duration and nature of their contact all contribute to determining this probability. In a group of people, these probabilities can be recorded in a social network; i.e., a directed labelled graph in which the vertices represent people and the edges are labelled with transmission probabilities. We consider two models in which these probabilities are time independent.(See below for details.)

We first consider the (discrete) SIR model, which was introduced by Kermack and McKendrick [8]. They formulated the differential equation governing a continuous model by first considering an associated discrete model that was fully mixed: one in which all individuals are in contact with each other, with identical transmission rates.

In this model: (i) an individual is either susceptible, infective, or recovered; (ii) a recovered individual stays recovered; (iii) an infective individual becomes recovered after a globally fixed time step; (iv) a susceptible individual who is not in contact with an infective individual stays susceptible; and (v) a susceptible individual who is in contact with one or more infective individuals either becomes infective or stays susceptible, with probability determined by the (per time step) transmission rate(s) of the contact(s).

After analyzing this model, we extend our results to what we call the S$\mathcal{I}$R model, in which conditions (iii) and (v) are relaxed. We relax (iii) by allowing different individuals to be infective for different lengths of time. We relax (v) by letting the transmission probabilities depend on the individual and on the number of time steps since the individual first became infective. In particular, this allows for a latent period and for the length of the latent period to vary from individual to

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individual. This model is time independent; the transmission probabilities depend on the number of time steps since the individual was infected, but they do not depend on when the individual was infected.

Returning to our analogy, we should point out that the use of backfires really corresponds to the case of disease transmission probabilities that are time dependent: firefighters, after setting a backfire, actively prevent its spread across the fire line. Our main result implies that in these models where the transmission probabilities are time independent, preemptive infections cannot help.

Of course, the real firebreaks against epidemics are vaccinations. While a backfire corresponds to deliberately switching individuals from susceptible to infective so that they will then become recovered, a safe vaccine switches the individual directly from susceptible to recovered. Is this safe for everyone else?

It is generally accepted that prophylactic vaccination benefits the group, not just the individual: vaccinating an individual can lower other people's chances of becoming infected. One can easily see that for realistic parameter values, this is always so in the Kermack-McKendrick model. (This can be derived from equation (20) of [8] or equation (9) of [1].) However, if we assume that the transmission probabilities can vary over time, a single vaccination can either avert or cause an epidemic. Consider, for instance, an individual P who is the sole contact between two communities, $\mathcal{A}$ and $\mathcal{B}$. Vaccinating P might block transmission from $\mathcal{A}$ to $\mathcal{B}$, thereby preventing an epidemic in $\mathcal{B}$. But what about vaccinating an individual Q in $\mathcal{A}$ who has close contact with P? Vaccinating Q might trigger an epidemic in $\mathcal{B}$ by causing P to be infected later, when the transmission probabilities are larger for edges emanating from P into $\mathcal{B}$.

In the real world, vaccination is highly effective as a public health measure. On average, administering a vaccination is more likely to reduce than increase the total number of infections. This must be the consequence of some generic properties of real-world social networks. We wonder what those properties are.

Here we assume that the transmission probabilities are time independent. In Section 3 we prove that, for our models, an individual's probability of getting the disease cannot be decreased by either infecting some individuals or increasing some transmission probabilities. It follows easily from the analysis that one cannot increase an individual's probability of getting infected by vaccinating some individuals.

Given an initial set of infectives in an otherwise susceptible population which is not fully mixed, we want to model the spread of disease on this social network. Since this happens probabilistically, there is no single scenario; rather, there are multiple scenarios with differing probabilities. As one begins to track these probabilities, one sees that the probabilities involved are no longer independent. The probability of transmission along an edge e whose origin is infective at time t depends on whether its target is still susceptible, and the likelihood of this being the case depends on the conditions at other vertices. In particular, the question is no longer local but potentially global.

One way to deal with these multiple possible time lines is to organize them into a tree or a directed acyclic graph. Each vertex of this graph represents a state of the network; i.e., a partition of the people into susceptible, infective and removed

states. The root is the initial situation. The edges of the graph give the ways in which the epidemic might proceed, each edge labelled with its probability[1].

Instead, we take the viewpoint that many of these problems become much more tractable if we pay less attention to the order of events. Rather than asking whether a given edge transmits infection at a given time, we ask whether a given edge transmits *if it ever comes into play.* Since a given edge can only transmit the disease once, in the course of an epidemic (or simulation) one can assume that a single edge either does or does not transmit the disease. Grassberger took this point of view in [6]; he considered the case that the social network is a cubic lattice and all of the transmission rates are the same, and noted that the problem of who gets infected could be interpreted in terms of bond percolation. This point of view has been considered for square lattices with random transmission rates in [11] by Sander et al., and for more general graphs by Newman, Meyers, and their coworkers (see, for example, [10] or [9], which each have extensive references).

The techniques we employ here are very similar to techniques developed independently and contemporaneously by Anil Kumar and Madhav Marathe of the Network Dynamics and Simulation Science Laboratory at Virginia Tech. We are grateful to the Laboratory's Director, Stephen Eubank, for first directing our attention to this fascinating area.

2. Background and definitions

2.1. **SIR social networks.** We wish to study how disease spreads though a fixed population. Infection is necessarily transmitted from an infected to a susceptible person. In this model, we assume that the epidemic proceeds in discrete time steps, that infection resides in any person for one step, that the probability of transmission between any two people is time-invariant, and that recovery from infection confers lifetime immunity. This can be summarized in the following definitions.

An SIR *social network* is a labelled finite directed graph $\mathcal{N} = (G, \mu)$. The vertices, elements of $V = V(G)$, are *people.* The edges, elements of $E = E(G)$, are determined by their endpoints; that is, $E \subset V \times V \setminus \Delta$. (Here Δ denotes the diagonal.) The function $\mu : E \to [0, 1]$ assigns a probability to each edge. Given an edge $e = (p, q)$, we denote the *source* and *target* of this edge by $\partial_0(e) = p$ and $\partial_1(e) = q$.

A *state* of this network is a labelling of its vertices with labels $\{S, I, R\}$, i.e. $\varphi : V \to \{S, I, R\}$. Said another way, the states of $\mathcal{N}$ are $\mathrm{St}(\mathcal{N}) = \{S, I, R\}^V$. We will say that φ is an *initial state* if $\varphi(V) \subset \{S, I\}$. Given a state φ, we say that an edge e is *in play* if $\varphi(\partial_0(e)) = I$ and $\varphi(\partial_1(e)) = S$. We will say that a vertex is *in play* if it is the target of an edge that is in play.

Given states φ_1 and φ_2, the state φ_2 is a *possible successor* of φ_1 if it satisfies the following conditions:

1. If $\varphi_1(p) = R$, then $\varphi_2(p) = R$.
2. If $\varphi_1(p) = I$, then $\varphi_2(p) = R$.
3. If $\varphi_1(p) = S$, then $\varphi_2(p) \in \{S, I\}$.
4. If $\varphi_2(p) = I$, then p is in play for φ_1.

[1]To make this graph acyclic, we need to delete the self-edges. These occur at states which have no infectives. We can force this graph to be a tree at the cost of having multiple vertices representing the same state.

An *epidemic* is a sequence of states $\varphi_0, \ldots, \varphi_k$ where φ_{i+1} is a possible successor of φ_i for $i = 0, \ldots, k-1$. Notice that any sufficiently long epidemic becomes a constant sequence in which each person is either susceptible or recovered. It is not hard to see that the longest an epidemic can remain non-constant is at most one more than the length of the longest self-avoiding path in G.

The probabilities on the edges of $\mathcal{N}$ induce a map f on the set of probability measures on $\{S, I, R\}^V$. This map is determined by its values on those measures which concentrate all probability in a single state φ. Let μ_φ be the measure which concentrates all probability in φ. The support of $f(\mu_\varphi)$ is in the set of possible successor states of φ. Let us denote the set of edges that are in play for φ by $P_\varphi(E)$ and the set of vertices that are in play by $P_\varphi(V)$. We assume these edges infect or fail to infect independently. Then, if v is in play for φ, the *probability that φ infects* v is given by

$$\mu(\varphi, v) = 1 - \prod_{\substack{\{e \in P_\varphi(E) | \\ \partial_1(e) = v\}}} (1 - \mu(e)).$$

The measure $f(\mu_\varphi)$ is non-zero only on the possible successors of φ, and there it is given by

$$f(\mu_\varphi)(\{\psi\}) = \prod_{\substack{\{v \in P_\varphi(V) | \\ \psi(v) = I\}}} \mu(\varphi, v) \prod_{\substack{\{v \in P_\varphi(V) | \\ \psi(v) = S\}}} (1 - \mu(\varphi, v)).$$

Notice that this also induces a measure $\mu_{\varphi_1 n}$ on the set of all epidemics of length n begining at φ_1 given by

$$\mu_{\varphi_1 n}(\varphi_1, \ldots, \varphi_n) = \prod_{i=2}^{n} f(\mu_{\varphi_{i-1}})(\{\mu_{\varphi_i}\}).$$

We may think of a *condition* as defining a set of states; i.e., the set of states that satisfy that condition. For example, given a person p, the condition that p is infected or recovered defines the set $\{\varphi \mid \varphi(v) \in \{I, R\}\}$. Given an initial state φ and a condition C, the probability that C holds after n steps is $f^n(\mu_\varphi)(C)$. In particular, given an initial state φ the probability that person p is either infected or recovered after n steps is

$$\mu(\varphi, n, p) = f^n(\mu_\varphi)\left(\{\psi \mid \psi(p) \in \{I, R\}\}\right).$$

Implicit in this definition is a choice of $\mathcal{N}$. Since we will wish to vary this choice, we will further decorate this as $\mu_{\mathcal{N}}(\varphi, n, p)$.

If we fix $\mathcal{N}$, then there is a partial order on the set of initial states. We will say $\varphi_0 \prec \varphi_1$ if $\{v \mid \varphi_0(v) = I\} \subsetneq \{v \mid \varphi_1(v) = I\}$. There is also a partial order on the social networks on a fixed graph. Given two social networks $\mathcal{N}_0$ and $\mathcal{N}_1$ with the same underlying graph, G, we say $\mathcal{N}_0 \prec \mathcal{N}_1$ if $\mu_0 \neq \mu_1$ and for each $e \in E$, $\mu_0(e) \leq \mu_1(e)$. We will also write $\mu_0 \prec \mu_1$.

2.2. **S$\mathcal{I}$R social networks.** In the SIR model, each individual can be infective for exactly one step. This is equivalent to assuming that there is no latency and that the period of infectivity is equal in all individuals and that this period is equal to the time step of the model. We now generalize this to a model to allow infection to persist and vary over time. We do this by assigning to each individual p a positive

integer $r(p)$ and a sequence of infective states[2] $\mathcal{I}_p = I_1, \ldots, I_{r(p)}$. In each state I_i with $i < r(p)$, p can either recover or progress to the next infective state I_{i+1}. We formalize this as follows. An S$\mathcal{I}$R social network is a 4-tuple

$$\mathcal{M} = (G, \iota, \rho, \mu)$$

where

1. $G = (V, E)$ is a directed graph.
2. For each $p \in V$, $\iota(p) = \mathcal{I}_p = I_1, \ldots, I_{r(p)}$.
3. For each $p \in V$ and $1 \leq i < r(p)$, $\rho(p, i)$ is the probability that p progresses from state I_i to state I_{i+1}. For notational convenience, we take $\rho(p, r(p)) = 0$ and $\rho(p, 0) = 1$.[3]
4. For each $e = (p, q) \in E$ and $1 \leq i \leq r(p)$, $\mu(p, i, q)$ is the probability that if p is in state i and q is susceptible, then p infects q.

The *states* of $\mathcal{M}$ are

$$\mathrm{St}(\mathcal{M}) = \{\varphi : V \to \{S\} \cup \mathcal{I} \cup \{R\} \mid \text{ for each } p \in V,\ \varphi(p) \in \{S\} \cup \mathcal{I}_p \cup \{R\} \ \},$$

where $\mathcal{I} = I_1, I_2, \ldots$. A state φ is an *initial state* if for each $p \in V$, $\varphi(p)$ is either S or $I_1 \in \mathcal{I}_p$. If $(p, q) \in E$ with $\varphi(p) \in \mathcal{I}_p$ and $\varphi(q) = S$, we say that (p, q) is *in play* and q is *in play*. Again, we denote by $P_\varphi(V)$ the set of vertices in play and by $P_\varphi(E)$ the set of edges in play. We say that φ_2 *is a possible successor of* φ_1 if it satisfies the following:

1. If $\varphi_1(p) = R$, then $\varphi_2(p) = R$.
2. If $\varphi_1(p) = I_{r(p)}$, then $\varphi_2(p) = R$.
3. If $\varphi_1(p) = I_j$, where $j < r(p)$, then $\varphi_2(p) \in \{I_{j+1}, R\}$.
4. If $\varphi_1(p) = S$, then $\varphi_2(p) \in \{S, I_1\}$.
5. If $\varphi_2(p) = I_1$, then p is in play for φ_1.

As before, we can define an epidemic to be a sequence of states each of which is a successor of the previous state. Once again, assuming independence of all events (infections and recoveries), we have a map f on the set of measures on $\mathrm{St}(\mathcal{M})$. For any φ where $q \in V$ is in play, we can compute the probability that φ infects q. For each infective p such that (p, q) is in play, there is $i = i(p)$ so that $\varphi(p) = I_i \in \mathcal{I}_p$, and we will abuse notation by dropping the dependency of i on φ and p. We then have

$$\mu(\varphi, q) = 1 - \prod_{(p,q) \in P_\varphi(E)} (1 - \mu(p, i, q)).$$

[2]If one wishes to assign biological meaning to these states, this is an abuse of notation since the I_i of $\mathcal{I}_p$ may not be the same as the I_i of $\mathcal{I}_q$. Mathematically, the biological information is encoded in the probabilities and we do not need to distinguish the I_i of different vertices.

[3]*A priori*, this appears to be a loss of generality since exposure might confer immunity without passing through an infective state. However, we can model this by setting $\mu(p, 1, q) = 0$.

Again $f(\mu_\varphi)$ is non-zero only on successors of φ, and here it is given by

$$f(\mu_\varphi)(\{\psi\}) = \left(\prod_{\substack{\{v\in P_\varphi(V)|\\ \psi(v)=I_1\}}} \mu(\varphi,v)\right)\left(\prod_{\substack{\{v\in P_\varphi(V)|\\ \psi(v)=S\}}} (1-\mu(\varphi,v))\right)$$
$$\left(\prod_{\substack{\{v|\varphi(v)=I_i,\\ \psi(v)=I_{i+1}\}}} \rho(v,i)\right)\left(\prod_{\substack{\{v|\varphi(v)=I_i,\\ \psi(v)=R\}}} (1-\rho(v,i))\right).$$

Again this induces a corresponding measure $\mu_{\varphi_1 n}$ on the set of epidemics of length n starting at φ_1.

Once again, we have a partial order on the set of initial states given by $\varphi_0 \prec \varphi_1$ if $\varphi_0^{-1}(\mathcal{I}) \subsetneq \varphi_1^{-1}(\mathcal{I})$. We also have a partial order on the set of S$\mathcal{I}$R social networks with a fixed graph and fixed labeling ι. If $\mathcal{M}_0 = (G, \iota, \rho_0, \mu_0)$ and $\mathcal{M}_1 = (G, \iota, \rho_1, \mu_1)$ then $\mathcal{M}_0 \prec \mathcal{M}_1$ if $\mathcal{M}_0 \neq \mathcal{M}_1$, $\rho_0(p,i) \leq \rho_1(p,i)$ for each (p,i), and $\mu_0(p,i,q) \leq \mu_1(p,i,q)$ for each (p,i,q).

3. Results

3.1. **Monotonicity of the SIR model.** Given a social network as described above, how can you decrease the probability that a particular person gets infected? One possibility is by inoculating certain individuals. In the model, this corresponds to assigning them state R. Is it possible to decrease any individual's probability of getting infected by deliberately infecting certain individuals? Since an infected person becomes recovered after one time step and can then never be infected again, it is conceivable that infecting an individual could lessen someone else's chance of becoming infected later. Alternatively, could you lessen an individual's chance of being infected by increasing some of the edge transmission rates?

The following theorem shows that both of these are impossible in this model.

Theorem 3.1. *The following monotonicity properties hold:*

1. *Given φ_0 and φ_1 in $\{S,I\}^V$ with $\varphi_0 \prec \varphi_1$, then for any person p and $n \geq 0$,*
$$\mu(\varphi_0, n, p) \leq \mu(\varphi_1, n, p).$$
2. *Given $\mathcal{N}_0$ and $\mathcal{N}_1$ with $\mathcal{N}_0 \prec \mathcal{N}_1$, then for any initial state $\varphi_0 \in \{S,I\}^V$, person p and $n \geq 0$,*
$$\mu_{\mathcal{N}_0}(\varphi_0, n, p) \leq \mu_{\mathcal{N}_1}(\varphi_0, n, p).$$

Now, vaccination of an individual p can be modelled as

- removing p from any potential set of initial infectives, and
- setting to 0 the transmission probabilities on edges to or from p.

Corollary 3.2. *In the SIR social network model, vaccination has no collateral damage.* □

We start by proving some lemmas.

Lemma 3.3. *Given any edge e and any epidemic $\varphi_0, \ldots, \varphi_k$, there is at most one state φ_j such that e is in play.*

Proof. For e to be in play at state φ_j, we must have $\varphi_j(\partial_0(e)) = I$. This implies that $\varphi_t(\partial_0(e)) = S$ for $t < j$ and $\varphi_t(\partial_0(e)) = R$ for $t > j$. □

This suggests the following map:

$$\epsilon : \{0,1\}^E \times \{S,I,R\}^V \to \{S,I,R\}^V.$$

Given $\zeta \in \{0,1\}^E$ and $\varphi \in \{S,I,R\}^V$ we take $\epsilon(\zeta,\varphi) = \psi$ where

$$\psi(v) = \begin{cases} R & \text{if } \varphi(v) = R \\ R & \text{if } \varphi(v) = I \\ I & \text{if there is } e \text{ such that } v = \partial_1(e), \text{ where } e \text{ is in play and } \zeta(e) = 1 \\ S & \text{otherwise} \end{cases}$$

We will also use $\epsilon_\zeta(\varphi)$ to denote $\epsilon(\zeta,\varphi)$.

We define a probability measure on $\{0,1\}^E$ by

$$\mu(\zeta) = \prod_{\{e|\zeta(e)=1\}} \mu(e) \prod_{\{e|\zeta(e)=0\}} (1-\mu(e)).$$

It is not hard to check that

$$\sum_{\zeta\in\{0,1\}^E} \mu(\zeta) = 1.$$

Lemma 3.4. *These have the following properties:*

1. *For any φ and ζ, $\epsilon(\zeta,\varphi)$ is a possible successor of φ.*
2. *Every possible successor of φ arises in this manner.*
3. *Given ζ and φ, the sequence $\epsilon_\zeta^i(\varphi)$ is an epidemic.*
4. *Every epidemic arises in this manner.*
5. *Given ζ, φ and ψ,*
$$f(\mu_\varphi)(\{\psi\}) = \mu(\{\zeta|\epsilon(\zeta,\varphi) = \psi\}).$$
6. *Given ζ, φ, n and ψ,*
$$f^n(\mu_\varphi)(\{\psi\}) = \mu\left(\{\zeta \mid \epsilon_\zeta^n(\varphi) = \psi\}\right).$$
Likewise, given a condition C,
$$f^n(\mu_\varphi)(C) = \mu\left(\{\zeta \mid \epsilon_\zeta^n(\varphi) \in C\}\right).$$

Proof. To see that ϵ produces only successor states, observe that for each vertex, ϵ preserves the property of being recovered, turns infected vertices into recovered vertices, infects only vertices that are in play, and preserves the property of being susceptible for those vertices which are in play that it does not infect and for those vertices which are not in play.

We wish to see that every successor state arises in this manner. To see this, notice that choosing a successor state corresponds to choosing which subset of the vertices that are in play to infect. We choose ζ in the following manner. If v is in play and we do not wish to infect v, then for each edge e which is in play with $\partial_1(e) = v$, we set $\zeta(e) = 0$. If v is in play and we wish to infect v, we choose at least one edge e which is in play with $\partial_1(e) = v$ and set $\zeta(e) = 1$.

It now follows immediately that the sequence $\epsilon_\zeta^i(\varphi)$ is an epidemic. Now Lemma 3.3 implies that given φ we can choose ζ to produce any epidemic which starts at φ.

To see (5), we need to look at the relationship between $\{0,1\}^E$ and $\{0,1\}^{E'}$, where $E' \subset E$. There is a map $\tau : \{0,1\}^E \to \{0,1\}^{E'}$, where $\tau(\zeta)$ is restriction of ζ

to E'. We have a measure μ' on $\{0,1\}^{E'}$ given by taking the above products only over edges in E'. It follows that for any set $X \subset \{0,1\}^{E'}$, $\mu'(X) = \mu(\tau^{-1}(X))$.

Now consider a vertex v which is in play, and take E' to be the set of edges which are in play and whose target is v. It is not hard to see that the μ' measure of those $\zeta \in \{0,1\}^{E'}$ which infect v is the same as the probability that φ infects v. This is the same as the μ measure of those $\zeta \in \{0,1\}^{E}$ which infect v. Applying this to every v which is in play gives the result.

Finally, the first part of (6) follows from (5) by induction. The second part of (6) follows from the first by summing over individual states. □

Lemma 3.5. *Suppose we are given ζ, φ_0, n and v, and that $\varphi_0(V) \subset \{S, I\}$. Then*

1. *$\epsilon_\zeta^n(\varphi_0)(v) = I$ if and and only if $\zeta^{-1}(1)$ contains a directed path from an infected vertex of φ_0 to v and the shortest such path has length n.*
2. *$\epsilon_\zeta^n(\varphi_0)(v) \in \{I, R\}$ if and only if $\zeta^{-1}(1)$ contains a directed path from an infected vertex of φ_0 to v of length at most n.*
3. *The epidemic induced by ζ starting at state φ_0 infects v if and and only if $\zeta^{-1}(1)$ contains a directed path from an infected vertex of φ_0 to v.*

Proof. The first statement can be proved by induction on n. Our induction hypothesis is as follows. Suppose the shortest path in $\zeta^{-1}(1)$ from the infected vertices of φ_0 to v has length n. (If there is no such path, we will say its length is ∞.) Then $\epsilon_\zeta^i(v) = S$ for $i < n$ and $\epsilon_\zeta^n(v) = I$ when $n < \infty$.

For $n = 0$, this reduces to the assumptions on φ_0. If it holds for $n-1$, then it follows immediately for n by applying ϵ_ζ to $\epsilon_\zeta^{n-1}(\varphi_0)$.

The second and third statements now follow immediately. □

We are now prepared to prove Theorem 3.1.

Proof. (Theorem 3.1) We are given a fixed social network, $\mathcal{N}$. We must show that given φ_0 and φ_1 with $\varphi_0 \prec \varphi_1$, a person p and $n \geq 0$, $\mu(\varphi_0, n, p) \leq \mu(\varphi_1, n, p)$. Now,

$$\mu(\varphi_0, n, p) = \mu\left(\{\zeta \mid \text{there is a path in } \zeta^{-1}(1) \text{ of length at most } n \text{ from } \varphi_0^{-1}(I) \text{ to } p\}\right)$$

and

$$\mu(\varphi_1, n, p) = \mu\left(\{\zeta \mid \text{there is a path in } \zeta^{-1}(1) \text{ of length at most } n \text{ from } \varphi_1^{-1}(I) \text{ to } p\}\right).$$

Since $\varphi_0^{-1}(I) \subsetneq \varphi_1^{-1}(I)$, the result follows.

Next, we are given two social networks $\mathcal{N}$ and $\mathcal{N}'$ differing only in their respective edge probabilities, with $\mu \prec \mu'$. We have an initial state φ_0, a person p and $n \geq 0$. We must prove that $\mu(\varphi_0, n, p) \leq \mu'(\varphi_0, n, p)$. We consider the set

$$Z = \{\zeta \mid \text{there is a path in } \zeta^{-1}(1) \text{ of length at most } n \text{ from } \varphi_0^{-1}(I) \text{ to } p\}.$$

It suffices to show that $\mu \prec \mu'$ implies $\mu(Z) \leq \mu'(Z)$, which we do in the following lemma. □

Lemma 3.6. *If $\mu \prec \mu'$ then $\mu(Z) \leq \mu'(Z)$.*

Proof. For convenience, we order $E = (e_1, \ldots, e_m)$. Consider a machine which carries out a lottery to choose an element of $\{0,1\}^E$ according to probability μ. Such a machine would consist of the m-dimensional unit cube $[0,1]^E$ and a way of choosing $(z_1, \ldots, z_m)$ with a flat probability distribution. The machine then emits ζ according to the formula

$$\zeta(e_i) = \begin{cases} 1 & \text{if } z_i \leq \mu(e_i) \\ 0 & \text{otherwise} \end{cases}$$

Let $\{\sigma_1, \ldots, \sigma_s\}$ be the set of paths from $\varphi^{-1}(I)$ to v of length less than or equal to n. For each σ_i there is a subset $S_i \subset [0,1]^E$ so that $\zeta^{-1}(1)$ includes σ_i if and only if the machine chooses a value in S_i. This set is

$$S_i = \{(z_1, \ldots, z_m) \mid z_j \leq \mu(e_j) \text{ if } e_j \text{ is an edge of } \sigma_i \ \}.$$

The machine chooses ζ which infects v in n or fewer steps if and only if it chooses an element of $S = \cup S_i$. The probability of doing so is $\mathrm{vol}(S)$.

If we now perform the same procedure using the edge probabilities given by μ' to produce $S' = \cup S_i'$, then in each case $S_i \subset S_i'$ so that $S \subset S'$, and hence $\mathrm{vol}(S) \leq \mathrm{vol}(S')$. □

3.2. **Symmetry in the SIR model.** We say that $\mathcal{N} = (G, \mu)$ is *symmetric* if $\mu(p,q) = \mu(q,p)$ for each $(p,q) \in E(G)$. In a symmetric social network, individuals are "equally mutually infective." A similar property holds on the level of groups of individuals. These two assertions are made precise in the following theorem.

Theorem 3.7. *Suppose $\mathcal{N}$ is symmetric. Then the following symmetry properties hold:*

1. *Let $p_0, p_1 \in V(G)$, and let φ_i, $i = 0, 1$, be the states defined by*

$$\varphi_i(p) = \begin{cases} I & \text{if } p = p_i \\ S & \text{otherwise} \end{cases}$$

Then for any $n \geq 0$,

$$\mu(\varphi_0, n, p_1) = \mu(\varphi_1, n, p_0).$$

2. *Let $P_0, P_1 \subset V(G)$, and let φ_i, $i = 0, 1$, be the states defined by*

$$\varphi_i(p) = \begin{cases} I & \text{if } p \in P_i \\ S & \text{otherwise} \end{cases}$$

For $i = 0, 1$, let

$$C_i = \{\varphi \mid \text{there exists } p \in P_i \text{ such that } \varphi(p) \in \{I, R\} \ \}.$$

Then for any $n \geq 0$,

$$f^n(\mu_{\varphi_0})(C_1) = f^n(\mu_{\varphi_1})(C_0).$$

Proof. Given $\zeta \in \{0,1\}^E$ and $n \geq 0$, $\epsilon_\zeta^n(\varphi_0)(p_1) \in \{I, R\}$ if and only if $\zeta^{-1}(1)$ contains a directed path from p_0 to p_1 of length less than or equal to n. Likewise, $\epsilon_\zeta^n(\varphi_1)(p_0) \in \{I, R\}$ if and only if $\zeta^{-1}(1)$ contains a directed path from p_1 to p_0 of length less than or equal to n. The symmetry of $\mathcal{N}$ makes these two events equally likely. This proves the first property.

Transmission from P_0 to P_1 takes place exactly for those ζ for which $\zeta^{-1}(1)$ contains a path from some person in P_0 to some person in P_1. The second property now follows by the same argument. □

3.3. **The S$\mathcal{I}$R model.** We will now generalize these results to S$\mathcal{I}$R social networks. The key here is that given an S$\mathcal{I}$R social network $\mathcal{M}$, we can build an SIR social network $\mathcal{N}$ whose epidemiology encodes that of $\mathcal{M}$. The underlying graph of $\mathcal{N}$ covers that of $\mathcal{M}$.

Theorem 3.8.

1. *Let $\mathcal{M}$ be an S$\mathcal{I}$R social network, let $p \in V$ be a person in this network, let $\varphi_0, \varphi_1 \in \mathrm{St}(\mathcal{M})$ be initial states satisfying $\varphi_0 \prec \varphi_1$, and let $n \geq 0$. Then $\mu(\varphi_0, n, p) \leq \mu(\varphi_1, n, p)$.*
2. *Let $\mathcal{M}_0 \prec \mathcal{M}_1$ be S$\mathcal{I}$R social networks, and suppose that φ is an initial state, $p \in V$ and $n \geq 0$. Then $\mu_{\mathcal{M}_0}(\varphi, n, p) \leq \mu_{\mathcal{M}_1}(\varphi, n, p)$.*

We start by constructing the *covering SIR social network* $\mathcal{N} = (\tilde{G}, \mu)$ for an S$\mathcal{I}$R social network $\mathcal{M} = (G, \iota, \rho, \mu)$. For each $p \in V$, we take $V_p = \{p_1, \ldots, p_r\}$, where $\mathcal{I}_p = I_1, \ldots, I_{r(p)}$ and $r = r(p)$. We take $\tilde{G} = (\tilde{V}, \tilde{E})$, where

$$\tilde{V} = \amalg_{p \in V} V_p,$$

and

$$\tilde{E} = \{(p_i, q_1) \mid (p, q) \in E, 1 \leq i \leq r(p)\} \cup (\amalg_{p \in V} \{(p_i, p_{i+1}) \mid 1 \leq i < r(p)\})$$

We say that V_p is the *stack over* p, that these vertices *project* to p, and that each edge of the form (p_i, q_1) *projects* to (p, q). For each edge of the form (p_i, q_1) and each edge of the form (p_i, p_{i+1}) we take

$$\mu(p_i, q_1) = \mu(p, i, q)$$
$$\mu(p_i, p_{i+1}) = \rho(p, i)$$

To understand the terminology and the underlying idea behind the construction, think of the graph G as being horizontal and the graph $\tilde{G}$ as lying above G. Over a vertex $p \in V$ there are vertices $p_1, \ldots, p_r$ (where $r = r(p)$) and "vertical" edges (p_i, p_{i+1}) for $1 \leq i < r(p)$. Over an edge (p, q) there are *diagonal* edges (p_i, q_1) for $1 \leq i \leq r(p)$.

Definition 3.9. *Recall that $\varphi \in \mathrm{St}(\mathcal{M})$ is an* initial state *if for each $p \in V$, $\varphi(v)$ is either S or $I_1 \in \mathcal{I}_p$. We will say that $\varphi_1 \in \mathrm{St}(\mathcal{N})$ is a* valid initial state *if*

1. *$\varphi_1(\tilde{V}) \subset \{S, I\}$.*
2. *Each vertex p with $\varphi_1(p) = I$ is the initial vertex in its stack; i.e., p is of the form p_1.*

Lemma 3.10. *Suppose that φ_1 is a valid initial state of $\mathcal{N}$ and that $\varphi_1, \ldots, \varphi_n$ is an epidemic. Then for each stack $V_p = \{p_1, \ldots, p_r\}$ and each i, $1 \leq i \leq n$, the string of labels in that stack, $\varphi_i(p_1)\varphi_i(p_2)\ldots\varphi_i(p_r)$, takes the form $R^a I^b S^c$ where*

1. *$a + b + c = r(p)$*
2. *$0 \leq a \leq r(p)$*
3. *$0 \leq b \leq 1$*
4. *$0 \leq c \leq r(p)$*

Proof. By assumption for $i = 1$ this string has either the form S^r or IS^{r-1}. Notice that by construction, infection can only arrive at the stack via p_1, and it can only be passed up the stack. Induction now shows that at each point in time, the R's occupy an initial segment of the stack, there is at most one infected vertex which must follow the R's, and any remaining vertices are susceptible. □

Definition 3.11. *We will say that a state $\varphi \in \mathrm{St}(\mathcal{N})$ is* valid *if it satisfies the conclusions of Lemma 3.10. We use $\mathrm{Val}(\mathcal{N})$ to denote the set of valid states.*

In particular, the valid initial states of $\mathcal{N}$ are valid.

Using the values a, b, c of the previous Lemma we can define a map

$$\Phi : \mathrm{Val}(\mathcal{N}) \to \mathrm{St}(\mathcal{M})$$

by

$$(\Phi(\varphi))(p) = \begin{cases} S & \text{if } c = r(p) \\ I_{a+1} & \text{if } b = 1 \\ R & \text{if } a > 0,\ b = 0 \end{cases}$$

Proposition 3.12.

1. *Suppose $\varphi \in \mathrm{Val}(\mathcal{N})$ and $p \in V$. Then φ is determined on V_p by $(\Phi(\varphi))(p)$ unless $(\Phi(\varphi))(p) = R$ and $r(p) > 1$.*
2. *Φ is a bijection between the initial states of $\mathcal{M}$ and the valid initial states of $\mathcal{N}$. We call φ and $\Phi(\varphi)$* corresponding initial states.
3. *If $\varphi \in \mathrm{Val}(\mathcal{N})$ then the possible successors of φ are in $\mathrm{Val}(\mathcal{N})$.*
4. *If φ_1 is a possible successor state of φ_0 in $\mathrm{Val}(\mathcal{N})$, then $\Phi(\varphi_1)$ is a possible successor state of $\Phi(\varphi_0)$.*
5. *Φ induces a bijection on epidemics which start in (valid) initial states. That is to say, given an epidemic $\psi_1, \ldots, \psi_n \in \mathrm{St}(\mathcal{M})$, there is a unique $\varphi_1 \in \mathrm{Val}(\mathcal{N})$ such that $\Phi(\varphi_1) = \psi_1$ and there is a unique epidemic $\varphi_1, \ldots, \varphi_n \in \mathrm{St}(\mathcal{N})$ such that $\Phi(\varphi_i) = \psi_i$ for $1 \leq i \leq n$. This epidemic necessarily lies in $\mathrm{Val}(\mathcal{N})$.*
6. *Φ carries the function f on probability measures on $\mathrm{Val}(\mathcal{N})$ to the function f on probability measures on $\mathrm{St}(\mathcal{M})$.*
7. *Given a valid initial state φ_1 of $\mathcal{N}$ and an epidemic $\varphi_1, \ldots, \varphi_n$,*

$$\mu_{\varphi_1 n}(\varphi_1, \ldots, \varphi_n) = \mu_{\Phi(\varphi_1)n}(\Phi(\varphi_1), \ldots, \Phi(\varphi_n))$$

Proof.

1. If $(\Phi(\varphi))(p) = S$, then $a = b = 0$, $c = r(p)$. In the case where $(\Phi(\varphi))(p) = I_i \in \mathcal{I}_p$, $a = i - 1$, $b = 1$ and $c = r(p) - i$. However, when $(\Phi(\varphi))(p) = R$ and $r(p) > 1$, we cannot determine a and c.
2. When φ is an initial state, $R \notin (\Phi(\varphi))(V)$, so the value of φ is determined in each stack V_p by the value of $(\Phi(\varphi))(p)$.
3. Given a stack $V_p = \{p_1, \ldots, p_r\}$ and a valid state φ, we consider the string of labels $\varphi(p_1) \ldots \varphi(p_r)$.
   (a) If this string is $S^{r(p)}$, then in any successor state, this string is either $S^{r(p)}$ or $IS^{r(p)-1}$.
   (b) If this string is $R^a I S^c$ then in any successor state, the string is either $R^{a+1}S^c$ or $R^{a+1}IS^{c-1}$.
   (c) If this string is $R^a S^c$ with $a > 0$, then in any successor state the string is unchanged.

4. This follows by observing that each possible change of state in the stack over p that we have just enumerated corresponds to a unique possible change of state at $p \in V$.
5. We have already seen the existence and uniqueness of φ_1. Using the previous observation, an induction shows the existence of each successive φ_i. *A priori*, uniqueness might fail in the stack over some p if $\psi_i(p) = R$. However, in this case φ_{i-1} determines the values that φ_i must take over p.
6. Once started in corresponding initial states, each edge that is in play in $\mathcal{N}$ corresponds to an edge that is in play in $\mathcal{M}$ and these bear the same probability of transmitting infection. Likewise each stack in which a vertex is infected lies over an infected vertex of $\mathcal{M}$ which is in the corresponding state, and these bear the same probability of recovery.
7. This follows from the previous step by induction.

□

We are now prepared to prove Theorem 3.8.

Proof. (Theorem 3.8) We start by building a covering graph for each of these S$\mathcal{I}$R social networks. In each case, we have a corresponding initial state in the covering SIR network. The key observation is that p becomes infected during an epidemic in the S$\mathcal{I}$R model if and only if $p_1 \in V_p$ becomes infected in the corresponding epidemic in the covering graph. Since the probabilities of corresponding epidemics are equal, we appeal to the theorem in the SIR case and are done. □

It is not hard to give a similar generalization of Theorem 3.7. Here the definition of symmetry requires that the following properties hold for each $(p, q) \in E$.

1. The edge (q, p) is in E.
2. The vertices p and q have the same number of infective states.
3. For each i with $1 \leq i < r(p)$, $\rho(p, i) = \rho(q, i)$.
4. For each i with $1 \leq i \leq r(p)$, $\mu(p, i, q) = \mu(q, i, p)$.

We wish to mention in passing one more method of modeling S$\mathcal{I}$R social networks with SIR social networks. Let us fix an S$\mathcal{I}$R social network $\mathcal{M}$. Given an edge (p, q) that is in play, we can compute the cumulative probability that p infects q during the course of p's infectivity, assuming that q is not infected first from some other source. Given that p enters state I_1, the probability that it enters state I_i without previously infecting q is

$$\prod_{j=1}^{i-1} \rho(p, i)(1 - \mu(p, i, q)).$$

Since infection of q from each of the infective states $I_i \in \mathcal{I}_p$ constitute disjoint events, the cumulative probability is

$$\mu(p, q) = \sum_{i=1}^{r(p)} \mu(p, i, q) \prod_{j=1}^{i-1} \rho(p, i)(1 - \mu(p, i, q)).$$

(The product is empty for $j = 1$.) We will call $\mathcal{N} = (G, \mu)$ the *cumulative infectivity SIR model* for $\mathcal{M}$. We leave to the reader the proof that starting in corresponding initial states p is ultimately infected in $\mathcal{M}$ with the same probability of ultimately being infected in $\mathcal{N}$. This model has the advantage of being simpler than the covering model. Its drawback is that the number of steps it takes in any given

epidemic for p to be infected in $\mathcal{N}$ tells us neither the number of steps nor the number of transmissions required in $\mathcal{M}$.

4. Discussion

In this paper, we have considered two social network models of epidemiology, the SIR and S$\mathcal{I}$R models. The discrete SIR model was introduced by Kermack and McKendrick in [8] as theoretical justification for their differential equations SIR model for epidemics. The discrete model, which is based upon a graph with a vertex for each individual and an edge for each pair of individuals who are in contact with each other, enables one to model populations in which the patterns and extents of contacts between individuals can vary greatly. With the advent of powerful computers, it has been increasingly used for simulations; this has sparked renewed theoretical interest in these models.

A number of studies in the last few years use discrete social networks to consider vaccination strategies for combating epidemics. In [5], Ferrari et al. contrast the effects of prior epidemics (which thin out nodes with large contacts) with random vaccinations for the SIR model on small world, Poisson, and scale-free graphs. In [3], Dezsö and Barabási use numerical and analytical results for the SIS model on a scale-free network to show that immunizing the highly-connected nodes can reduce the epidemic threshold. (In the SIS model, individuals are either susceptible or infectious, and an infectious individual becomes susceptible again.) In [2], Colizza et al. consider the effects of different vaccination strategies on large-scale simulations with the SLIR model. (Here L represents a latent state.) They are mainly interested in influenza, and take into account air traffic and the worldwide diversity of stockpile sizes of anitviral drugs. Hartvigsen et al. are also interested in the SIR model for influenza in [7]. They use numerical simulations with various underlying graphs to test five different vaccination strategies. Since they found little difference due to population size in their simulations, for the paper they chose a population size of 10,000. In [4], Eubank et al. describe their work using EpiSims to model epidemics in large networks corresponding to urban populations. They use their model of Portland to test several different vaccination strategies.

Here we have used bond percolation methods to establish some elementary properties of SIR networks. By ignoring the time at which a given edge might transmit infection, these methods avoid a thicket of conditional probabilites that grows up around any attempt to trace the propagation of the probabilities through the network. These methods require transmission probabilities that do not vary over time. The methods break down, and the results are false, if transmission probabilities are allowed to vary over time — as they surely do in reality.

Our results in the SIR and S$\mathcal{I}$R models imply that I cannot get sick because you got vaccinated. Yet we have seen that this conclusion need not be true if transmission probabilities vary over time. We suspect that an individual vaccination occasionally does have the peverse effect of raising another's odds of infection. However, real-world experience is that vaccination creates herd immunity. This suggests that on average, the collateral benefit of vaccination outweights any collateral damage. The example we gave in the introduction seems fragile. One has the reaction, "Sure, that could happen, but I bet most of the time, it doesn't." What properties of social networks would supply a rigorous justification for this feeling?

We have used a sort of covering graph to extend our results from the SIR model to the S$\mathcal{I}$R model. Since in the SIS model an edge may transmit infection more than once, the SIS model is not obviously interpretable in terms of bond percolation. However, we are currently working on using a covering SIR social network to model the repeated infection that is possible in SIS social networks. We hope to extend our results to this case.

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