A COVERING-GRAPH APPROACH TO EPIDEMICS ON SIS AND SIS-LIKE NETWORKS

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Abstract. In this paper we introduce a new class of epidemics on networks which we call SI(S/I). SI(S/I) networks differ from SIS networks in allowing an infected individual to become reinfected without first passing to the susceptible state. We use a covering graph construction to compare SIR, SIS, and SI(S/I) networks. Like the SIR networks that cover them, SI(S/I) networks exhibit infection probabilities that are monotone with respect to both transmission probabilities and the initial set of infectives. The same covering graph construction allows us to characterize the recurrent states in a SIS or SI(S/I) network with reinfection.

1. Introduction

Two standard network models for epidemics are the SIR and SIS models, which can each be considered deterministically or stochastically. The discrete stochastic SIS model does not in general exhibit monotonicity; that is, decreasing either transmission probabilities or the initial number of infected individuals may increase the total number of infections. Because monotonicity can be associated with herd immunity, models that admit monotonicity seem to be of real-world relevance. We introduce a stochastic SIS-like model that admits monotonicity with respect to both transmission probabilities and initial infectives. Moreover, there are in general no persistent epidemics on the stochastic SIS model, so that asymptotic behavior is uninteresting; we consider certain long-lived states that may be of qualitative interest.

In the SIR model, an individual is either susceptible (S), infective (I), or recovered (R). A susceptible individual may become infective if in contact with one or more infective individuals, and an infective individual will recover and no longer be capable of getting the disease. In the SIS model, an individual is either susceptible or infective, and an infective individual becomes susceptible again after recovering from the disease.

If births and deaths are neglected, a deterministic SIR model is given by the system

$$S' = -\beta SI, \quad I' = \beta SI - \gamma I, \quad R' = \gamma I$$

of ordinary differential equations, and a deterministic SIS model by the system

$$S' = -\beta SI + \gamma I, \quad I' = \beta SI - \gamma I$$

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of ordinary differential equations, where β is the transmission rate and γ is the recovery rate. The total population size is the constant $N = S + I + R$ in the first case and $N = S + I$ in the second. In both cases, $\mathcal{R}_0 = \frac{N\beta}{\gamma}$ is called the *basic reproductive number*. An initial condition with everyone susceptible except for a small number of infectives will proceed to an epidemic if $\mathcal{R}_0 > 1$ and will not if $\mathcal{R}_0 < 1$. The corresponding discrete deterministic SIS model is given by

$$S(n+1) = S(n) - \beta S(n)I(n) + \gamma I(n), \quad I(n+1) = I(n) + \beta S(n)I(n) - \gamma I(n).$$

In this model as well, the basic reprodutive number is $\mathcal{R}_0 = \frac{N\beta}{\gamma}$. For more information see, for example, Bailey's text [2] or Brauer's survey article [3] for the ordinary differential equations models and the Allen-Burgin article [1] for the discrete deterministic models.

The basic stochastic SIS network model consists of a labeled finite directed graph in which the vertices correspond to individuals, and the edges to contacts between individuals. Each edge is labeled with a transmission probability, and at any unit of time a vertex is labeled S or I. Epidemics proceed probabilistically. If no edge has probability 1, then since extinction is the single absorbing state it is well known from the theory of Markov processes that epidemics achieve extinction with probability 1. That is, there are no persistent epidemics on such networks. This stands in sharp contrast to the results for deterministic SIS networks.

An important real-world benefit of vaccination is herd immunity. By protecting those who are vaccinated, it reduces opportunities for disease transmission and thus confers probabilistic protection on those who are not vaccinated. In the deterministic SIR and SIS models above, a crucial benefit of vaccination is to reduce $\mathcal{R}_0$ by effectively reducing N. With a sufficient portion of the population vaccinated, $\mathcal{R}_0$ drops below 1 and there is no epidemic. Mathematically, herd immunity also manifests itself in terms of monotonicity properties: each person's chances of becoming infected should not go up as a result of either reducing the initial set of infective people or reducing transmission probabilities between people. Surprisingly, if transmission probabilities can vary with respect to time, SIR networks need not exhibit monotonicity. However, if these probabilities are held constant, then SIR networks are necessarily monotone [5].

A key ingredient for the proof of monotonicity in [5] is that in an SIR network each edge may transmit infection at most once. This allows for the use of percolation methods to study epidemics in SIR networks, and monotonicity follows readily from the percolation approach. By contrast, there is no limit to the number of times an edge may transmit on an SIS network. We used a covering graph argument in [5] to apply percolation methods to a generalization of SIR networks in which infectivity can last longer than a single time step. We now wish to use simlar covering graph methods to apply percolation techniques to study SIS networks. It turns out that these arguments require us to introduce a new class of SIS-like networks allowing for immediate reinfection. We call these SI(S/I) networks. As are SIR networks, SI(S/I) networks are monotone with respect to initial infectives and edge probabilities.

We look at examples where the network remains infected at very high levels for a very long time and spends most of that time in a particular state. Thus, while infection is not the long-term behavior of the system, it can be very long lived and spend most of this time in states that act like "conditional attractors". These

long lived behaviors, called quasi-stationary distributions by Daroch and Seneta in [4], may be of more practical importance than the network's eventual inevitable extinction. We also consider networks with spontaneous reinfection.

2. Background and definitions

2.1. **Social networks.** We consider the epidemiology of three sorts of networks, namely, SIR networks, SIS networks, and SI(S/I) networks. We give their definitions in parallel.

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

For SIR networks, we take $\mathbb{S} = \{S, I, R\}$; for SIS and SI(S/I) networks, we take $\mathbb{S} = \{S, I\}$. Elements of $\mathbb{S}$ are *compartments*, and $\mathbb{S}$ is the *compartmental model* or *set of compartments*. A *state* of such a network is an assignment of each vertex to a compartment, i.e. a function $\varphi : V \to \mathbb{S}$. That is, the set of states of $\mathcal{N}$ is $\mathrm{St}(\mathcal{N}) = \mathbb{S}^V$. In the case of an SIR network we say that a state is an *initial state* if it lies in $\{S, I\}^V$.

For a state φ of either an SIR network or an SIS network, we say that an edge e is *in play* if $\varphi(\partial_0(e)) = I$ and $\varphi(\partial_1(e)) = S$. For a state φ of an SI(S/I) network we say that an edge e is *in play* if $\varphi(\partial_0(e)) = I$. For each of the models, a vertex v is *in play* if it is the target of an edge that is in play.

Given states φ_1 and φ_2, we specify when the state φ_2 is a *possible successor* of φ_1. The rules here differ for the three kinds of networks.

For an SIR network φ_2 is a *possible successor* of φ_1 if the following hold:

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

For an SIS network φ_2 is a *possible successor* of φ_1 if the following hold:

(1) If $\varphi_1(v) = I$, then $\varphi_2(v) = S$.
(2) If $\varphi_2(v) = I$, then v is in play for φ_1.

For an SI(S/I) network φ_2 is a *possible successor* of φ_1 if the following holds:

(1) If $\varphi_2(v) = I$, then v is in play for φ_1.

For SIR, SIS, and SI(S/I) networks, we are using a standard simplification in which the infective period lasts a single time step. Thus SI(S/I) networks differ from SIS networks in allowing for immediate reinfection without an intervening return to the susceptible state.

An *epidemic* is a sequence $\varphi_1, \varphi_2, \ldots, \varphi_k$ (respectively $\varphi_1, \varphi_2, \ldots$) of states with φ_{i+1} a possible successor of φ_i for $1 \le i < k$ (respectively, $1 \le i$). The state φ with $\varphi(v) = S$ for all $v \in V$ is called the *clearance* state. We say that an epidemic *achieves extinction* if there is a nonnegative integer i such that φ_i is the clearance state and that an epidemic is *persistent* if it does not achieve extinction.

The probabilities on the edges of $\mathcal{N}$ induce a map f on the set of probability measures on $\mathrm{St}(\mathcal{N}) = \mathbb{S}^V$. 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

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

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

$$f(\mu_\varphi)(\{\psi\}) = \prod_{\{v \in P_\varphi(V) | \psi(v) = I\}} \mu(\varphi, v) \prod_{\{v \in P_\varphi(V) | \psi(v) = S\}} (1 - \mu(\varphi, v)). \tag{2}$$

Thus, given an initial condition φ_1, vertex $v \in V$, and time step $n \in \mathbb{N}$, we can ask (for example) what is the probability that v is in a given state at time n. $f^n(\mu_{\varphi_1})$ gives the probability distribution for all states at time step n and the probability that v is (say) infected at this time is $f^n(\mu_{\varphi_1})(\{\varphi \mid \varphi(v) = I\})$. In the case of SIS and SI(S/I) networks, we denote by $\mu(\varphi_1, n, v)$ the probability that v is infected at time n. In the case of SIR networks, we use this to denote the probability that v has become infected at or before time n.

For either SIR, SIS, or SI(S/I) networks, if we fix $\mathcal{N}$ then there is a partial order on the set of states in $V^{\{S,I\}}$. We say that $\varphi_0 \prec \varphi_1$ if $\varphi_0^{-1}(I) \subsetneq \varphi_1^{-1}(I)$. 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 also write $\mu_0 \prec \mu_1$.

2.2. **Percolation on social networks.** Our viewpoint here is that prior to the unfolding of an epidemic, a grand lottery is held to determine for each edge, whether it transmits if it ever comes into play. We use the notation of [5]. We define a *lottery* ζ to be an element of $\{0,1\}^E$. Those edges e with $\zeta(e) = 1$ are *winners*. The flat distribution on the cube $[0,1]^E$ induces a measure on the set $\{0,1\}^E$ of lotteries as follows. Define a map $t : [0,1]^E \to \{0,1\}^E$ by

$$t(x)(e) = \begin{cases} 1 & \text{if } x_e \leq \mu(e) \\ 0 & \text{otherwise} \end{cases}.$$

Thus, an edge e is a winner in the lottery $\zeta = t(x)$ if and only if $x_e \leq \mu(e)$. The flat distribution on $[0,1]^E$ pushes forward under t to a measure on $\{0,1\}^E$.

A lottery decides which, if any, of the edges that are in play transmit infection and thus determines the course of an epidemic. This can be formalized in the obvious way as a map

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

The map ϵ and measure μ are compatible with f in the following sense:

$$f(\mu_\varphi)(\{\psi\}) = \mu(\{\zeta | \epsilon(\zeta, \varphi) = \psi\}).$$

It follows that given a lottery ζ, an initial state φ_1, and vertex v, the vertex v becomes infected in the course of an epidemic if and only if there is a path of

winners from $\varphi_1^{-1}(I)$ to v. In particular, $\mu(\varphi_1, n, v)$ is the measure of the set of such ζ.

2.3. **Comparing networks using covering graphs.** We now have three types of social networks—SIR, SIS, and SI(S/I)—and we wish to study the relationships among them. Our main tool for doing this will be the use of covering graphs. Given a social network $\mathcal{N} = (G, \mu)$ and a choice of compartmental model (SIR, SIS, or SI(S/I)), its transition graph $T(\mathcal{N})$ is the labeled directed graph whose vertices are the elements of $\mathrm{St}(\mathcal{N})$ and whose edges are the pairs (φ_0, φ_1) such that φ_1 is a successor of φ_0. An edge (φ_0, φ_1) is labeled with $f(\mu_{\varphi_0})(\varphi_1)$. We say that $\mathcal{N}_1$ *models* $\mathcal{N}_2$ if there is a forward invariant set $U \subset \mathrm{St}(\mathcal{N}_1)$ and a map $m : U \to \mathrm{St}(\mathcal{N}_2)$ which is onto and preserves probability, that is, if $\varphi_0 \in U$ then m restricted to the successors of φ_0 is a bijection to the successors of $m(\varphi_0)$. That is, for φ_1 a successor of φ_0, $f_{\mathcal{N}_1}(\mu_{\varphi_0})(\varphi_1) = f_{\mathcal{N}_2}(\mu_{m(\varphi_0)})(m(\varphi_1))$. Let $T(U)$ be the subgraph of $T(\mathcal{N}_1)$ with vertex set U. This makes $m : T(U) \to T(\mathcal{N}_2)$ a covering map. Note that we do not require $\mathcal{N}_1$ and $\mathcal{N}_2$ to have the same compartmental models.

3. Three comparison theorems

Having introduced the class of SI(S/I) social networks, we now wish to compare these with SIS networks. For each of these comparisons, we will make use of covering graph arguments.

Theorem 3.1. *Let $\mathcal{N} = (G, \mu)$ be a social network. Let $\varphi_1 : V \to \{S, I\}$, and let $n \in \mathbb{N}$. Using $\mu_{SIS}(\varphi_1, n, v)$ and $\mu_{SI(S/I)}(\varphi_1, n, v)$ to denote the corresponding probabilities under SIS and SI(S/I) propagation, we have $\mu_{SIS}(\varphi_1, n, v) \leq \mu_{SI(S/I)}(\varphi_1, n, v)$.*

Theorem 3.2. *Every SI(S/I) network is modeled by an SIS network which double covers it.*

Theorem 3.3. *SI(S/I) networks enjoy the following monotonicity properties:*

1. *Let $\mathcal{N}$ be an SI(S/I) network and $\varphi_1, \varphi_2 \in \mathrm{St}(\mathcal{N})$ with $\varphi_1 \prec \varphi_2$. Then for any vertex v and $n \geq 0$,*
$$\mu(\varphi_1, n, v) \leq \mu(\varphi_2, n, v).$$
2. *Given SI(S/I) networks $\mathcal{N}_0$ and $\mathcal{N}_1$ with $\mathcal{N}_0 \prec \mathcal{N}_1$, then for any initial state $\varphi_1 \in \{S, I\}^V$, vertex v, and $n \geq 0$,*
$$\mu_{\mathcal{N}_0}(\varphi_1, n, v) \leq \mu_{\mathcal{N}_1}(\varphi_1, n, v).$$

SIS networks do not in general exhibit monotonicity.

We postpone the proofs of Theorems 3.1 and 3.3, which follow from applying percolation methods to a covering graph.

Proof of Theorem 3.2. Let $\mathcal{N}_2 = ((V_2, E_2), \mu_2)$ be an SI(S/I) network. We take (as an SIS network) $\mathcal{N}_1 = (G_1, \mu_1)$ to be the bipartite labeled directed graph given by

$$\begin{aligned} V_1 &= V_2 \times \mathbb{Z}_2 \\ E_1 &= \{((p, i), (q, i+1)) \mid (p, q) \in E_2,\ i \in \mathbb{Z}_2\} \\ \mu_1((p, i), (q, i+1)) &= \mu_2(p, q). \end{aligned}$$

G_1 double covers G_2. For $i \in \mathbb{Z}_2$ we take

$$\begin{aligned} \mathrm{St}_i(\mathcal{N}_1) &= \{\varphi \in \mathrm{St}(\mathcal{N}_1) \mid \varphi^{-1}(I) \subset V_2 \times \{i\}\} \\ U &= \mathrm{St}_0(\mathcal{N}_1) \cup \mathrm{St}_1(\mathcal{N}_1). \end{aligned}$$

U is forward invariant. Note that SIS and SI(S/I) propagation coincide on U since every edge from an infective leads to a susceptible. Thus U double covers $\mathrm{St}(\mathcal{N}_2)$ preserving probabilities, so $\mathcal{N}_1$ models $\mathcal{N}_2$ as required. □

4. A More elaborate covering graph

4.1. Definition and basic properties.

Definition 4.1. Let $\mathcal{N} = (G, \mu)$ be an SIS or SI(S/I) network. The *tower network* $\widetilde{\mathcal{N}} = (\widetilde{G}, \widetilde{\mu})$ is the infinite network given by the following data:

$$\begin{aligned} \widetilde{V} &= V \times \mathbb{N} \\ \widetilde{E} &= \{((p,n),(q,n+1)) \mid (p,q) \in E,\ n \in \mathbb{N}\} \\ \widetilde{G} &= (\widetilde{V}, \widetilde{E}) \\ \widetilde{\mu}((p,n),(q,n+1)) &= \mu(p,q) \end{aligned}$$

We take $\widetilde{G}_{[m,n]}$ to be the induced subgraph of $\widetilde{G}$ whose vertices are $V \times \{m, \dots, n\}$ and we take $\widetilde{\mathcal{N}}_{[m,n]}$ to be the corresponding social network.

We leave unspecified for now whether $\widetilde{\mathcal{N}}$ is an SIR, SIS or SI(S/I) network.

The alert reader will have noticed that $\widetilde{\mathcal{N}}$ is not, strictly speaking, a social network since the underlying graph is not finite. However, we can think of $\widetilde{G}$ as

$$\widetilde{G} = \cup_{n=1}^{\infty} \widetilde{G}_{[1,n]}.$$

The properties we discuss (e.g., the probability that (v,n) becomes infected) depend only on finite subsets of $\widetilde{G}$. Accordingly, we are untroubled by this abuse of terminology.

We now turn to the case where we consider $\mathcal{N}$ as an SI(S/I) network and $\widetilde{\mathcal{N}}$ as an SIR network.

Definition 4.2. Suppose that $\varphi_1, \varphi_2, \dots$ is a finite or infinite epidemic on $\mathcal{N}$. The *corresponding epidemic* on $\widetilde{\mathcal{N}}$ is the sequence

$$\Phi(\varphi_1, \varphi_2, \dots) = \psi_1, \psi_2, \dots$$

where ψ_n is defined by

$$\psi_n(v,j) = \begin{cases} S & \text{if } n < j \\ \varphi_n(v) & \text{if } n = j \\ R & \text{if } j < n \text{ and } \varphi_j(v) = I \\ S & \text{otherwise} \end{cases}$$

A *valid initial state* of $\widetilde{\mathcal{N}}$ is an initial state in which all infective vertices are confined to $G_{[1,1]}$. A *valid state* is one which can arise in the course of an epidemic starting at a valid initial state.

The idea behind the tower network is that a vertex (v, n) of $\widetilde{\mathcal{N}}$ corresponds to the vertex v of $\mathcal{N}$ at time n (where the starting time is 1 and the time step is 1). While a vertex v of $\mathcal{N}$ might get infected more than once, vertices of $\widetilde{\mathcal{N}}$ do not get reinfected since a vertex (v, n) can only be infective at time n. This allows us to use percolation methods on $\widetilde{\mathcal{N}}$ and apply the results to $\mathcal{N}$.

Lemma 4.3. *$\widetilde{G}$ is a covering graph of G. The following hold:*

1. *The map $\varphi_1 \mapsto \psi_1$ is a bijection between $St(\mathcal{N})$ and the set of valid initial states of $\widetilde{\mathcal{N}}$.*
2. *The edge $((p, n), (q, n + 1))$ is in play for ψ_n if and only if (p, q) is in play for φ_n.*
3. *ψ_{n+1} is a possible successor of ψ_n. In particular a corresponding epidemic is an epidemic.*
4. *The epidemic $\varphi_1, \varphi_2, \ldots$ infects vertex v at time n if and only if the corresponding epidemic infects vertex (v, n) at time n.*
5. *As φ_{n+1} varies over all possible successors of φ_n, ψ_{n+1} varies over all possible successors of ψ_n. In particular, Φ is a bijection between the set of epidemics on $\mathcal{N}$ and the set of epidemics on $\widetilde{\mathcal{N}}$ which start in a valid initial state.*
6. *The map Φ preserves probability. That is to say,*

$$f(\mu_{\varphi_n})(\{\varphi_{n+1}\}) = f(\mu_{\psi_n})(\{\psi_{n+1}\})$$

Proof. (1) Consider the map that takes $\varphi \in \mathrm{St}(\mathcal{N}) = \{S, I\}^V$ to the valid initial state ψ defined by $\psi(v, 1) = \varphi(v)$ and $\psi(v, n) = S$ for $n > 1$. This map is clearly a bijection and is none other than the restriction of Φ.

(2) By definition, $j = n$ is the only value of j for which ψ_j may assign I to (v, n). Further, it assigns I if and only if $\varphi_n(v) = I$. Since $\mathcal{N}$ is an SI(S/I) network, in this case all edges out of v are in play. Since ψ_n assigns S for all $(q, n + 1)$, when $\psi_n(v, n) = I$ all edges out of (v, n) are in play.

(3) We need to check that ψ_{n+1} is a possible successor of ψ_n. By assumption, φ_{n+1} infects only vertices which are in play for φ_n. Since ψ_{n+1} infects $(v, n + 1)$ exactly when φ_{n+1} infects v, by (2), ψ_{n+1} infects only vertices which are in play for ψ_n. By construction, vertices which are infected by ψ_n are assigned R by ψ_{n+1}, and recovered vertices remain recovered.

(4) This is immediate from the definition.

(5) Since $\psi_{n+1}(v, n+1) = \varphi_{n+1}(v)$, the map carrying possible successors φ_{n+1} of φ_n to ψ_{n+1} is injective. Given a possible successor of ψ_n, (2) ensures that the corresponding candidate φ_{n+1} is a possible successor of φ_n.

(6) This now follows from (2) and the fact that $\mu((p, n), (q, n + 1)) = \mu(p, q)$.

□

Corollary 4.4. *$\widetilde{\mathcal{N}}$ models $\mathcal{N}$.*

Proof. This follows by taking U to be the set of valid states. □

Note that the proof of Lemma 4.3 (2) is precisely where we need the distinction between SIS and SI(S/I) networks.

4.2. **Applications.**

Proof of Theorem 3.3. The result for SI(S/I) networks now follows by Lemma 4.3 and the corresponding monotonicity result for SIR networks, Theorem 3.1 of [5].

To see that SIS networks do not in general enjoy monotonicity with respect to the set of initial infectives, consider a network $\mathcal{N} = (G, \mu)$ with two initial states: a state φ_1 such that each individual is expected to be infected more than once, and the state ψ_1, with all individuals infected. For ψ_1, extinction is immediately achieved after one time step. Thus, going from φ_1 to ψ_1 increases the initial set of infectives but decreases the number of expected infections for each vertex.

We can turn this into a counter-example to monotonicity with respect to edge probabilities. Expand G to a new graph $\widehat{G}$ by appending a single vertex $\widehat{v}$ to G, with edges from $\widehat{v}$ to each vertex of G. Leave the probabilities on all edges of G unchanged but define $\widehat{\mu}_1$ on $\widehat{G}$ by setting the probability on each new edge to be 1, and $\widehat{\mu}_2$ by assigning probability 1 to each edge from $\widehat{v}$ to $\varphi_1^{-1}(I)$ and 0 to all other edges from $\widehat{v}$. For $\widehat{\mathcal{N}}_1 = (\widehat{G}, \widehat{\mu}_1)$ and $\widehat{\mathcal{N}}_2 = (\widehat{G}, \widehat{\mu}_2)$, consider the initial state with $\widehat{v}$ infective and all other vertices susceptible. For $\widehat{\mathcal{N}}_1$, extinction is achieved after two time steps. But for $\widehat{\mathcal{N}}_2$, after the first time step the course of the infection depends only on $\mathcal{N} = (G, \mu)$, which is in state φ_1. Thus, going from $\widehat{\mathcal{N}}_1$ to $\widehat{\mathcal{N}}_2$ decreases edge probabilities but increases the number of expected infections. □

Proof of Theorem 3.1. Suppose we are given a social network $\mathcal{N}$. We would like to know the conditions under which the initial state φ_1 leads to vertex v being infected at time n under SI(S/I) propagation. This happens when (v, n) becomes infected in the course of the corresponding infection of $\widetilde{\mathcal{N}}$, and this happens for those lotteries ζ which provide at least one path of winners from the set of initial infectives in $V \times \{1\}$ to (v, n).

For a path of ζ to produce infection of (v, n) under SIS propagation, it must fulfill the additional condition that for each edge $e = ((q, i), (r, i + 1))$ of this path (r, i) must be susceptible. This makes the set of ζ which produce infection of v at time n under SIS propagation a subset of those that produce infection of v at time n under SI(S/I) propagation. The result now follows. □

4.3. **Ping-pong infection is the only persistence.** Ping-pong infection occurs when two individuals repeatedly infect each other. When they do this with probability 1, this gives a persistent epidemic. We shall see that in SIS and SI(S/I) networks, this is essentially the only form of persistence.

We refer to a sequence of edges $(v_0, v_1), (v_1, v_2), \ldots, (v_{n-1}, v_0)$ with probability 1 on each edge as a *cycle*. In the SIS case we require $n > 1$; we allow for $n = 1$ in the SI(S/I) case. If $\mathcal{N}$ does not contain a cycle, we say it is *cycle free*. In the SIS case we say it is *persistent* if it has at least one infected vertex and at least one uninfected vertex. In the SI(S/I) case we say it is *persistent* if it contains at least one infected vertex. The next theorem generalizes the familiar concept of ping-pong infection.

Theorem 4.5. *Suppose that $\mathcal{N} = (G, \mu)$ is either an SIS or SI(S/I) network. Then with probability 1 all epidemics achieve extinction if and only if $\mathcal{N}$ is cycle free.*

Proof. In the SI(S/I) case, if any vertex of a cycle becomes infected, infection will travel around that cycle forever. It is not hard to see that the same is true in the SIS case if at least one member of a cycle is infected and one is not. To see this, notice that in this case there must be an edge from an infected to an uninfected

member of this cycle. Thus, at least one uninfected member of the cycle becomes infected, and each infected member must return to the susceptible state.

Now suppose that $\mathcal{N}$ does not contain a cycle. It then follows that $\widetilde{\mathcal{N}}$ has a longest path σ which has probability 1 on each of its edges. Suppose the length of this path is k. Fix n and consider $\widetilde{\mathcal{N}}_{[n,n+k+1]}$, the induced subgraph of $\widetilde{\mathcal{N}}$ whose vertex set is $V \times [n, n+k+1]$. Since there is no path of length $k+1$ with probability 1 on each edge, for each pair (p, n) and $(q, n+k+1)$, any path from (p, n) to $(q, n+k+1)$ contains an edge with probability less than 1. Hence, the probability of transmission from (p, n) to $(q, n+k+1)$ is less than 1. Consequently, the probability of transmission from all of $V \times \{n\}$ to $(q, n+k+1)$ is less than 1, and so the probability that there is any infected vertex of $V \times \{n+k+1\}$ is also less than 1. Further, since edge probabilities are constant, this probability of attaining extinction between time n and time $n+k+1$ is bounded away from 0 independent of n. It follows that there is $r < 1$ so that the probability of persistence of infection in $\mathcal{N}$ for $k+1$ steps is less than r. Since n was arbitrary, the probability of infection persisting for $m(k+1)$ steps is less than r^m. Thus the network achieves extinction with probability 1. □

Notice that in the course of proving Theorem 4.5 we have shown the following.

Proposition 4.6. *Suppose $\mathcal{N}$ is a cycle-free SIS or SI(S/I) network. Then there are C and $r < 1$ so that for any initial condition φ, the probability that an epidemic with initial state φ has not achieved extinction after n time steps is less than Cr^n. In particular, the probability that a vertex v is infected at step n is less than Cr^n.* □

5. Shortcomings of Theorem 4.5

5.1. **Long-lived behaviors.** There is something unsatisfactory in Theorem 4.5. As the following example shows, the expected time to extinction can be extraordinarily long, rendering Theorem 4.5 useless as a practical guide to network behavior.

Example 5.1. We take $\mathcal{N} = \mathcal{N}(n, p)$ to be the network consisting of the full graph on n vertices with probability p on each edge. We include self-edges at every vertex. We consider SI(S/I) propagation on $\mathcal{N}$. By Theorem 3.2, the full bipartite graph $\mathcal{K} = \mathcal{K}(n, p)$ includes the same dynamics under SIS propagation. Depending on n and v, we can expect infection on $\mathcal{N}$ to persist for many steps. If there is at least one infected vertex, then at the following step, the expected number of infected vertices is at least np and the chance of extinction is at most $(1-p)^n$.

Taking $n = 100$ and $p = .99$ gives a very conservative estimate that the expected time to extinction is greater than 10^{200}. With very high probability, during the course of the epidemic the network spends most of its time with 99 or 100 infected vertices. In fact, it spends most of this time with 100 infected vertices.

There are several things to be learned from this example. The first is that while extinction is the only attracting state of the system, it can take an extremely long time to reach that state. The second is that prior to extinction, there are states that act like attractors or stationary points.

Implicit in our discussion of the transition graph $T(\mathcal{N})$ is the Markov chain viewpoint. Given a cycle-free SIS or SI(S/I) network, we enumerate $\mathrm{St}(\mathcal{N})$ as $\varphi_1, \ldots, \varphi_N$ with $N = 2^{\|V\|}$ and take φ_1 to be clearance. Taking P to be the transition matrix and

$$p_1 = [p_{11} \ldots p_{1N}]$$

to be an initial distribution, this distribution evolves over time as $p_n = p_1 P^n$, and Theorem 4.5 is simply the assertion that

$$\lim_{n\to\infty} p_1 P^n = [1\ 0 \dots 0].$$

In [4], Darroch and Seneta consider this phenomenon of long-lived states in a Markov process with a single absorbing state. They give several approaches to defining such "almost-stationary" distributions, which they call quasi-stationary distributions. Here we consider the approach that they call stationary conditional distributions. These states can be described in terms of conditional probabilities, i.e., the probability of finding the system in a particular state given that it has not reached extinction.

Thus, rather than starting with an arbitrary distribution considering the recursion

$$p_1 = [p_{11} \dots p_{1N}] \mapsto p_2 = p_1 P = [p_{21} \dots p_{2N}],$$

we start with a distribution $p_1 = [0\ p_{12} \dots p_{1N}]$ and consider the recursion

$$p_1 \mapsto C(p_1) = \frac{1}{p_{21}}[0\ p_{22} \dots p_{2N}],$$

the result of conditioning on non-extinction. (Note that this requires that extinction not be the only possible outcome starting at p_1.) A distribution is then *almost stationary* if it is stationary for C. A distribution or set of distributions is a *conditional attractor* if it is an attractor for C. It is well known that P has a single eigenvector with eigenvalue 1, and all other eigenvalues have norm less than 1. It is not hard to see that each eigenvector (other than clearance) is almost stationary and that the subspace spanned by the eigenvectors of the second largest eigenvalue or eigenvalues is a conditional attractor. For generic P there will be a single such attractor. An extensive bibliography on quasi-stationary distributions is given at

`http://www.maths.uq.edu.au/~pkp/papers/qsds/qsds.pdf`.

5.2. **Spontaneous reinfection.** There is another reason that Theorem 4.5 may not be appropriate for some real-world applications. Real-world networks are neither constant nor isolated over the long term. This argues for including spontaneous infection from outside the network. (Indeed, we can assume that this is how the network got into its initial state.) Formally, we define an *SI(S/I) network with reinfection* to be the pair $\mathcal{N}^* = (\mathcal{N}, \bar{\mu})$, where $\mathcal{N}$ is an SI(S/I) network and $\bar{\mu} : V \to [0, 1]$ gives the probability of spontaneous infection of each vertex. We assume that all infections take place or fail to take place independently. This gives the following modifications to our basic results concerning SI(S/I) networks.

(1) A vertex v is in play if it is the target of an edge that is in play or if $\bar{\mu}(v) > 0$. If $\bar{\mu}(v) > 0$, for all $v \in V$, all vertices are always in play.
(2) A state φ_2 is a $\mathcal{N}^*$ possible successor of a given state φ_1 if and only if every infected vertex v of φ_2 is either in play for $\mathcal{N}$ or has $\bar{\mu}(v) > 0$. In particular, if $\mathcal{N}$ has no edges with probability 1 and $0 < \bar{\mu}(v) < 1$ for all $v \in V$, then every state is a $\mathcal{N}^*$ possible successor of every other state.
(3) Following equation 1, the probability that φ infects v in $\mathcal{N}^*$ is given by
$$\mu^*(\varphi, v) = 1 - (1 - \mu(\varphi, v))(1 - \bar{\mu}(v))$$
(4) Equation 2 changes only in that $\mu^*(\varphi, v)$ is substituted for $\mu(\varphi, v)$ and different vertices are in play.

Proposition 5.2. *Suppose that $\mathcal{N}^*$ consists of the SI(S/I) network $\mathcal{N}$ together with a reinfection function $\bar{\mu}(v)$. Then there is an SI(S/I) network $\widehat{\mathcal{N}}$ which models $\mathcal{N}^*$.*

Proof. We take $\widehat{\mathcal{N}}$ to consist of $\mathcal{N}$ together with an additional vertex $\widehat{v}$. We append edges $(\widehat{v}, v)$ with transmission probabilities $\bar{\mu}(v)$ and an edge $(\widehat{v}, \widehat{v})$ with probability 1. We then model $\mathcal{N}^*$ using the set of states in which $\widehat{v}$ is infected. It is clear that the obvious bijection carries $f_{\widehat{\mathcal{N}}}$ to $f_{\mathcal{N}^*}$. □

5.3. **Spontaneous reinfection, covering graphs and lotteries.** We would like to adapt the method of covering graphs and lotteries to networks with spontaneous reinfection. We do this by applying these methods to the covering graph $\widetilde{\widehat{\mathcal{N}}}$ of the network $\widehat{\mathcal{N}}$ of Proposition 5.2. We then have

$$\widetilde{\widehat{V}} = \widetilde{V} \cup \{\widehat{v}\} \times \mathbb{N}$$
$$\widetilde{\widehat{E}} = \widetilde{E} \cup E_{\text{vertical}} \cup E_{\text{reinfection}}$$

where

$$E_{\text{vertical}} = \{((\widehat{v}, n), (\widehat{v}, n+1)) \mid n \in \mathbb{N}\}$$
$$E_{\text{reinfection}} = \{((\widehat{v}, n), (v, n+1)) \mid n \in \mathbb{N},\ \bar{\mu}(v) \neq 0\}$$

We have

$$\widetilde{\mu}(E_{\text{vertical}}) = 1$$
$$\widetilde{\mu}((\widehat{v}, n), (v, n+1)) = \bar{\mu}(v)$$
$$\widetilde{\mu}((p, n), (q, n+1)) = \mu(p, q)$$

As before, a lottery ζ of $\widetilde{\widehat{\mathcal{N}}}$ causes (v, n) to become infected if and only if there is a path of winners from an initial infective to (v, n). The measure of such ζ gives the probability that v is infected at time n in $\widehat{\mathcal{N}}$.

5.4. **Spontaneous reinfection and Markov chains.** It is clear that in the presence of spontaneous reinfection, clearance is no longer an absorbing state.

Given a network with spontaneous reinfection $\mathcal{N}^* = (\mathcal{N}, \bar{\mu})$, we say that $\mathcal{N}^*$ is *cycle free* if $\mathcal{N}$ is cycle free and there is no vertex v such that $\bar{\mu}(v) = 1$.

We say that a state of $\mathcal{N}^*$ is *spontaneous* if it can arise in the course of an epidemic starting in the infection-free state.

Proposition 5.3. *Let $\mathcal{N}^* = (\mathcal{N}, \bar{\mu})$ be a cycle-free SI(S/I) network with reinfection. Then a state of $\mathcal{N}^*$ is recurrent if and only if it is spontaneous. These form an aperiodic set of communicating states. In particular, $\mathcal{N}^*$ has a unique stationary distribution.*

Proof. We claim that there is $k > 0$ such that for any state φ, there is a positive probability that $\mathcal{N}^*$ arrives at clearance within k steps starting at φ. This follows by recapitulating the proof of Theorem 4.5 and using the additional observation that at each step there is a positive probability that no reinfection occurs. In particular, there is $\epsilon > 0$ such that for each state, there is probability at least ϵ that clearance occurs within k steps. It follows that clearance recurs almost surely. Now every spontaneous state can with positive probability arise in the course of an epidemic

starting at clearance. It follows that these are all recurrent and communicate with clearance. Further, since clearance can immediately follow clearance, this recurrent class is aperiodic.

It remains to check that these are the only recurrent states. Let us suppose that φ can occur in an epidemic with initial state φ_1, but cannot occur in an epidemic starting with clearance. Let us suppose that it occurs at time n. It follows that it results from a lottery which contains a path in $\widetilde{\mathcal{N}}_{[1,n]}$ from some infected vertex of φ_1 to an infected vertex (v, n). By the argument of Proposition 4.6, there are C and $r < 1$ so that the probability of such an epidemic is bounded above by Cr^n. In particular, the probability of state φ falls off exponentially as a function of n. However, any recurrent state occurs almost surely for arbitrarily high n. Hence φ is not recurrent. □

6. Discussion

In a previous paper [5], we considered the SIR model for epidemics in social networks. Using bond percolation, we established that SIR epidemic models satisfy monotonicity with respect to edge probabilities and with respect to the number of infectives. In this paper we extend this analysis to SIS and SIS-like epidemic models. The bond percolation technique does not apply directly to an SIS model, but it does apply to a suitable covering graph of an SIS or SI(S/I) model. Switching to a covering graph to use bond percolation was done in [5] for the S$\mathcal{I}$R model, and it seems to be implicit in [6]. We use covering graphs more systematically here as a tool for comparing different compartmental models and extending results across compartmental models.

In the SIS model, an infective individual must return to susceptibility before becoming infective again. In the simplified model where the infective period only lasts a single time step, this means that an infective individual at one time step cannot be infective at the next time step. We introduce an SIS-like model called the SI(S/I) model. The SI(S/I) model differs from the SIS model precisely in allowing an infective individual to be reinfected while still infective. Although we haven't found this model in the literature, from personal experience we know that an infected computer can get reinfected with a virus while it already has the virus. (Some computer viruses disable reinfections but other viruses don't.) While SIS models do not in general satisfy monotonicity with respect to edge probabilities or the number of infectives, SI(S/I) models satisfy monotonicity with respect to both.

When used in conjunction with covering graphs, the Markov chain approach that we use here allows us to relate social networks with different compartmental models. This is convenient for a theoretical study of these networks, since results for networks with one compartmental model may be extended to networks with other models. Unfortunately, the Markov chain analysis that we use here is intractable from a computational viewpoint. For a social network $\mathcal{N} = (G, \mu)$ with either the SIS or the SI(S/I) model, if G has n vertices then the transition graph $T(\mathcal{N})$ has 2^n vertices. This exponential growth in the number of vertices prevents explicit computation of the transition graph for large networks. Indeed, computing the probability of an individual becoming infected in an SIR network is NP-hard (see the paper [7] by Delgado-Eckert and Shapiro). Still, covering graphs give structural insight into the relationships between these different compartmental models.

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