

THRESHOLD BEHAVIOR OF EPIDEMICS IN REGULAR NETWORKS

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ABSTRACT

Current research is interested in identifying how topology impacts epidemics in networks. In this paper, we model SIS (susceptible-infected-susceptible) epidemics as a continuous-time Markov process and for which we can obtain a closed form description of the equilibrium distribution. Such distribution describes the long-run behavior of the epidemics. The adjacency matrix of the network topology is reflected explicitly in the formulation of the equilibrium distribution. Secondly, we are interested in analyzing the model in the regime where the topology dependent infection process opposes the topology independent healing process. Specifically, how will network topology affect the most probable long-run network state? We show that for k -regular graph topologies, the most probable network state transitions from the state where everyone is healthy to one where everyone is infected at a threshold that depends on k but not on the size of the graph.

Index Terms— reversible Markov process, regular networks, equilibrium distribution, SIS epidemics, limiting distribution

1. INTRODUCTION

We are interested in analyzing diffusion processes in a multi-agent system where agents' states are affected by their local neighborhood. We will motivate our problem by studying the spread of epidemics throughout a population. Such a process can also be used to study the spread of information, the spread of failures, etc. Traditional epidemics modeling assumes a homogenous population where each agent interacts with every other agent in the population. Such an approach uses mean-field approximations to analyze the diffusion process for a very large population ([1], [2]).

More recently, in conjunction with growing interest in network science, researchers have been interested in understanding how the topology of the interconnections in a population might affect the diffusion process. Usually, the diffusion process of interest is the SIS (susceptible-infected-susceptible) epidemics [1]. In such a process, an infected node may heal then be reinfected again. Many previous works focused on analyzing how epidemics will spread amongst specific graph topologies such as Erdős-Rényi networks, scale-free networks, etc [3].

In 2003, [4] introduced a general SIS epidemics model for arbitrary network topologies by modeling the diffusion process as a discrete time linear dynamic system; the state of the system is a vector whose elements correspond to a single agent in the population and whose values represents the probability that the agent is infected.

They found that the dynamics of the epidemics is dependent on the largest eigenvalue of the adjacency matrix that describes the topology of the network.

Alternatively, [5] modeled the SIS epidemics using a continuous-time Markov process where the state of the system is a binary-valued vector where each entry denotes the current state of an agent (i.e., 1 for infected, 0 for uninfected). Their results showed that the mean epidemics lifetime is related to the spectral radius of the adjacency matrix.

Both models assume that the only way an agent may become infected is through contamination from an infected neighbor. We account for possible infection sources external to the population (i.e., exogenous infection sources). Therefore, the epidemics is not eradicated if all the agents are healthy as an agent may spontaneously become infected.

Instead of the mean epidemics lifetime, we are interested in the long-run behavior of the epidemics process. For example, what is the probability that agent 1 and agent 2 will be infected while the rest of the population is healthy. What is the most likely network state in the long-run?

In section 2, we describe our continuous-time Markov process model in detail and provide the closed form equation of the limiting distribution for arbitrary network topologies. This result relates to works done by [6] but our formulation makes explicit how network topology affects the equilibrium distribution. In section 3, we focus on analyzing the most probable network configuration (i.e., the network state with the maximum equilibrium probability) for the regime where the topology dependent infection process opposes the topology independent healing process. We find that for regular networks, the critical point where the most probable network state exhibit threshold behavior is solely dependent on the degree of the network.

2. THE MODEL

2.1. Network of Agents

Consider a population of N agents whose relationship is represented by an unweighted, undirected, connected graph, $G(V, E)$, where V is the set of vertices and E is the set of edges. The topology of G is captured by the symmetric, $N \times N$ adjacency matrix, A . The state of the i th agent is denoted by n_i . Each agent can be in one of two possible states: healthy ($n_i = 0$) or infected ($n_i = 1$).

We assume that the network topology, G , remains static over time. Define $\mathbf{n}$ as the N -tuple collection of all the node states.

$$\mathbf{n} = [n_0, n_1, \dots, n_{N-1}]^T$$

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We will refer to $\mathbf{n}$ as the network state in this paper. Define $\mathcal{N} = \{\mathbf{n}\}, |\mathcal{N}| = 2^N$.

2.2. Continuous-Time Markov Chain SIS Model

Let $X(t) = \mathbf{n}$ be the state of the network at time $t, t \geq 0$. The evolution of $X(t)$ models a SIS epidemics on the network consisting of 2 types of events:

1. *Healing*. Infected agents are healed in a length of time that is exponentially distributed with rate $\mu > 0$. The healed agent is susceptible to reinfection. The parameter μ is considered to be network topology independent as we assume that μ is the same for all agents.
2. *Infection*. We separate the infection process into two types: exogenous and endogenous infection.
 - (a) *Exogenous Infection*. An uninfected node may spontaneously develop infection in a length of time that is exponentially distributed with rate $\lambda > 0$. We assume that λ is the same for all the agents; the parameter is network topology independent.
 - (b) *Endogenous Infection*. An uninfected node becomes infected by transmission from infected neighbors (assuming that all infected individuals are equally contagious). The parameter $\gamma > 0$ is the endogenous infection rate due to a single infected neighbor. We assume that γ is independent of λ . However, since the overall endogenous infection rate is dependent on the total number of infected neighbors, it is different for different agents and is therefore network topology dependent.

Under these conditions, we model $X(t)$ as a finite state, continuous-time Markov chain with state space $\mathcal{N} = \{\mathbf{n}\}$.

Adapting the notation from [7], we define 2 operators on a state of the Markov process, $\mathbf{n} = [n_0, n_1, \dots, n_j, \dots, n_k, \dots, n_{N-1}]^T$

$$H_k \mathbf{n} = [n_0, n_1, \dots, n_k = 1, \dots, n_{N-1}]^T$$

$$H_{j\bullet} \mathbf{n} = [n_0, n_1, \dots, n_j = 0, \dots, n_{N-1}]^T$$

The operator H_k defines the operation that node k becomes infected. If node k is already infected, the operator does nothing. The operator $H_{j\bullet}$ defines the operation that node j is healed. If node j is already uninfected, the operator does nothing.

There are two types of state transitions in the Markov process corresponding to healing and infection events respectively:

- 1) $X(t)$ jumps to the network state where the j th node ($j = 0, \dots, N-1$) is healed with transition rate:

$$q(\mathbf{n}, H_{j\bullet} \mathbf{n}) = \mu, \quad \mathbf{n} \neq H_{j\bullet} \mathbf{n} \quad (1)$$

Borrowing terminology from the field of system reliability, the time, T , it takes for the j th node to heal is referred to as the *downtime*. The average downtime for agent j is

$$E[T_j] = \frac{1}{\mu} \quad (2)$$

- 2) $X(t)$ jumps to the network state where the k th node ($k = 0, 1, \dots, N-1$) is infected with transition rate

$$q(\mathbf{n}, H_k \mathbf{n}) = \lambda \gamma^{d_k} \quad (3)$$

where $d_k = \sum_{j=0}^{N-1} \mathbb{1}(n_j = 1) A_{jk}$, is the number of infected neighbors of node k . The symbol $\mathbb{1}(\cdot)$ is the indicator function, and

$A = [A_{jk}]$ is the adjacency matrix of G . We will refer to the time, $\hat{T}$, it takes for the k th node to become infected as the *uptime*. The average uptime for agent k is

$$E[\hat{T}_k] = \frac{1}{\lambda \gamma^{d_k}} \quad (4)$$

When $d_k = 0$, we will refer to $E[\hat{T}_k] = \frac{1}{\lambda}$ as the *topology independent uptime*.

In previous SIS Markov models ([8, 5]), the rate of infection is addition (i.e., $\lambda + \gamma d_k$). In this model, the rate of infection is multiplicative.

2.3. Rate Matrix, Q

Using the rates defined in (1) and (3), we can generate the rate or infinitesimal matrix, Q . The infinitesimal matrix is an asymmetric $2^N \times 2^N$ matrix. $Q_{i,j}$ corresponds to the transition rate between 2 network states $\mathbf{i}, \mathbf{j} \in \mathcal{N}$ where i and j are the decimal scalar representation of $\mathbf{i}$ and $\mathbf{j}$ respectively.

Since $X(t)$ is an irreducible, finite-state continuous-time Markov process, the equilibrium distribution, π , always exists and is the unique equilibrium distribution. Furthermore, π is also the limiting distribution of the process [7].

$$\pi(\mathbf{n}) = \lim_{t \rightarrow \infty} P(X(t) = \mathbf{n}), \quad \mathbf{n} \in \mathcal{N}$$

The equilibrium distribution can be found by solving $\pi Q = 0$. However, this is computational infeasible for networks with large number of agents. We have shown previously for a similar SIS model that symmetries in network topologies decrease computation requirement [8].

2.4. Reversibility and Equilibrium Distribution

Some continuous-time Markov process possess the property that the stochastic behavior of the process forward in time is the same as the behavior of the process reversed in time. These Markov processes are called *reversible* processes. There exists a simple necessary and sufficient condition relating the equilibrium distribution and the reversibility condition:

Theorem 2.1 (From [7]). *A stationary Markov process is reversible if and only if there exists a collection of positive number $\pi(j), j \in \mathcal{L}$, summing to unity that satisfy the detailed balance conditions*

$$\pi(j)q(j, k) = \pi(k)q(k, j), \quad j, k \in \mathcal{L}$$

When there exists such a collection $\pi(j), j \in \mathcal{L}$, it is the equilibrium distribution of the process.

Using the detailed balance conditions, the equilibrium distribution may be ‘guessed’.

Theorem 2.2 (Proof in Appendix A). *$X(t)$ is a reversible Markov process and the equilibrium distribution is*

$$\pi(\mathbf{n}) = \frac{1}{Z} \left(\frac{\lambda}{\mu} \right)^{1^T \mathbf{n}} \gamma^{\frac{\mathbf{n}^T A \mathbf{n}}{2}}, \quad \mathbf{n} \in \mathcal{N} \quad (5)$$

where Z is the normalization constant and is defined as

$$Z = \sum_{\mathbf{n} \in \mathcal{N}} \left(\frac{\lambda}{\mu} \right)^{1^T \mathbf{n}} \gamma^{\frac{\mathbf{n}^T A \mathbf{n}}{2}} \quad (6)$$

The equilibrium distribution is the product on two terms: a topology independent term and a topology dependent term. The topology independent term consists of the topology independent model parameters $\frac{\lambda}{\mu}$ and $1^T \mathbf{n}$, the number of infected nodes in the network state $\mathbf{n}$. The topology dependent term explicitly accounts for the network topology in the form of the adjacency matrix, A . It consists of the topology dependent parameter γ and $\frac{\mathbf{n}^T A \mathbf{n}}{2}$, the number of edges where both end nodes are infected in network state $\mathbf{n}$. Note that $0 \leq 1^T \mathbf{n} \leq N$ and $0 \leq \frac{\mathbf{n}^T A \mathbf{n}}{2} \leq E$, where N is the total number of nodes in the network and E is the total number of edges.

3. MOST PROBABLE LONG-RUN NETWORK STATE, $\mathbf{n}^*$

We can analyze the long-run behavior of the SIS epidemics by solving for the entire equilibrium distribution using Theorem 2.2. However, the normalization constant, Z , may be cumbersome to calculate for large networks.

Instead of looking at the full equilibrium distribution, we focus our attention on the most probable network state, $\mathbf{n}^*$, where

$$\mathbf{n}^* = \arg \max_{\mathbf{n} \in \mathcal{N}} \pi(\mathbf{n}) = \arg \max_{\mathbf{n} \in \mathcal{N}} \left(\frac{\lambda}{\mu} \right)^{1^T \mathbf{n}} \gamma^{\frac{\mathbf{n}^T A \mathbf{n}}{2}} \quad (7)$$

We will show that $\mathbf{n}^*$ exhibits a threshold behavior on the modeling parameters $\left(\frac{\lambda}{\mu}, \gamma \right)$. In particular, we can show how the topology of k -regular graph network will affect $\mathbf{n}^*$ when the model parameters are in the following regime: $0 < \frac{\lambda}{\mu} \leq 1, \gamma > 1$.

With $0 < \frac{\lambda}{\mu} \leq 1$, the topology independent healing process dominates the topology independent infection process. Without the diffusion process, the agents are more likely to be healthy than infected. However, since $\gamma > 1$, additional infected agents will exert an adverse effect on health of the population. What role does network topology play in this tug-of-war between the topology independent process and the topology dependent process?

3.1. K-Regular graph

In a k -regular network, each node has k neighbors. Partition the state space, $\mathcal{N}$

$$\mathcal{N} = \mathcal{N}_0 \cup \mathcal{N}_1 \cup \dots \mathcal{N}_N$$

where $\mathcal{N}_s = \{\mathbf{n} \in \mathcal{N} \mid 1^T \mathbf{n} = s\}$ is the set of network states with s infected agents and $\mathbf{n}^s \in \mathcal{N}_s$. Note that $\mathcal{N}_0 = \{\mathbf{n}^0\}$ and $\mathcal{N}_N = \{\mathbf{n}^N\}$.

Lemma 3.1 (Proof in Appendix B). *When $0 \leq \frac{\lambda}{\mu} \leq 1, \gamma > 1$ and for $s = 0, 1, \dots, N, \mathbf{n}^s \in \mathcal{N}_s$, the unnormalized equilibrium distribution is upperbounded by an exponential function:*

$$\pi(\mathbf{n}^s) \propto \left(\frac{\lambda}{\mu} \right)^s \gamma^{\frac{\mathbf{n}^s T A \mathbf{n}^s}{2}} \leq \left(\frac{\lambda}{\mu} \gamma^{\frac{k}{2}} \right)^s \quad (8)$$

Furthermore, the relationship holds with equality for $\mathbf{n}^0$ and $\mathbf{n}^N$.

Theorem 3.1. *When $0 \leq \frac{\lambda}{\mu} \leq 1, \gamma > 1$, a threshold exists for the most probable network configuration*

1. $\frac{\lambda}{\mu} \gamma^{\frac{k}{2}} > 1$ if and only if $\mathbf{n}^*$ is unique and $\mathbf{n}^* = \mathbf{n}^N = [1, 1, \dots, 1]^T$

2. $\frac{\lambda}{\mu} \gamma^{\frac{k}{2}} < 1$ if and only if $\mathbf{n}^*$ is unique and $\mathbf{n}^* = \mathbf{n}^0 = [0, 0, \dots, 0]^T$
3. $\frac{\lambda}{\mu} \gamma^{\frac{k}{2}} = 1$ if and only if $\mathbf{n}^*$ is no longer the unique maximizer: $\mathbf{n}^* = \mathbf{n}^N, \mathbf{n}^* = \mathbf{n}^0$

Proof. For sufficiency in case 1 of Theorem 3.1: if $\frac{\lambda}{\mu} \gamma^{k/2} > 1$, then $\mathbf{n}^*$ is unique and $\mathbf{n}^* = \mathbf{n}^N$.

When $\frac{\lambda}{\mu} \gamma^{k/2} > 1$, the RHS of (8) is maximized when $s = N$. Since this is a growing exponential function, it is also the unique maximizer. By Lemma 3.1, $\mathbf{n}^* = \mathbf{n}^N$. $\square$

Proof. For necessity in case 1 of Theorem 3.1: if $\mathbf{n}^*$ is unique and $\mathbf{n}^* = \mathbf{n}^N$, then $\frac{\lambda}{\mu} \gamma^{k/2} > 1$

Since $\mathbf{n}^* = \mathbf{n}^N$, it follows from Lemma 3.1 that the bounding exponential function reaches a maximum at $s = N$. By the monotonicity property, we know that this is satisfied only when $\frac{\lambda}{\mu} \gamma^{k/2} > 1$. $\square$

Proof. For sufficiency in case 2 of Theorem 3.1: If $\frac{\lambda}{\mu} \gamma^{k/2} < 1$, then $\mathbf{n}^*$ is unique and $\mathbf{n}^* = \mathbf{n}^0$.

When $\frac{\lambda}{\mu} \gamma^{k/2} < 1$, the RHS of (8) is maximized when $s = 0$. Since this is a decaying exponential function, it is also the unique maximizer. By Lemma 3.1, $\mathbf{n}^* = \mathbf{n}^0$. $\square$

Proof. For necessity in case 2 of Theorem 3.1: If $\mathbf{n}^*$ is unique and $\mathbf{n}^* = \mathbf{n}^0$, then $\frac{\lambda}{\mu} \gamma^{k/2} < 1$.

Since $\mathbf{n}^* = \mathbf{n}^0$, it follows from Lemma 3.1 that the bounding exponential function reaches a maximum at $s = 0$. By the monotonicity property, we know that this is satisfied only when $\frac{\lambda}{\mu} \gamma^{k/2} < 1$. $\square$

Proof. For sufficiency in case 3 of Theorem 3.1: If $\frac{\lambda}{\mu} \gamma^{\frac{k}{2}} = 1$ then $\mathbf{n}^*$ is no longer the unique maximizer. $\mathbf{n}^* = \mathbf{n}^N, \mathbf{n}^* = \mathbf{n}^0$.

When $\frac{\lambda}{\mu} \gamma^{k/2} = 1$, the RHS of (8) is 1 regardless of s . We know that this is satisfied with equality for $\mathbf{n}^0$ and $\mathbf{n}^N$. Therefore, $\mathbf{n}^*$ is no longer the unique and $\mathbf{n}^* = \mathbf{n}^N, \mathbf{n}^* = \mathbf{n}^0$. $\square$

Proof. For necessity in case 3 of Theorem 3.1: If $\mathbf{n}^*$ is no longer the unique maximizer and $\mathbf{n}^* = \mathbf{n}^N, \mathbf{n}^* = \mathbf{n}^0$, then $\frac{\lambda}{\mu} \gamma^{k/2} = 1$.

When $\frac{\lambda}{\mu} \gamma^{k/2} = 1$, the RHS of (8) is the same for $s = 0$ and $s = N$ when $\frac{\lambda}{\mu} \gamma^{k/2} = 1$. By Lemma 3.1, $\mathbf{n}^* = \mathbf{n}^N, \mathbf{n}^* = \mathbf{n}^0$ is also satisfied. $\square$

4. CONCLUSION

By modifying the transition rates so that the diffusion effect is multiplicative rather than linear, the continuous-time Markov process that describes the SIS epidemics becomes a reversible process. A closed form expression for the equilibrium distribution, up to a constant multiplier, is obtained for arbitrary network topology; the equilibrium distribution is separable into a topology independent term and a topology independent term.

Network topology plays an important role when the topology independent process opposes the topology dependent process (e.g.,

topology dependent process tends toward cascading infection but individual nodes tend to heal faster than they become infected). For a k -regular network, we prove that relationship between the model parameters and the degree of the network defines a critical threshold for the most probable network state; the size of the network however, is not a contributing factor.

The threshold defines where the most probable network state transitions from the configuration where everyone is healthy to the configuration where everyone is infected. At the threshold, the behavior is unpredictable. Our results show that networks with high degree will have a lower threshold than a network with low degree. This matches our intuition that a network with higher degree is more well connected; therefore it is easier for the topology dependent process to dominate the topology independent process.

5. REFERENCES

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A. PROOF OF THEOREM 2.2

Proof. Using Theorem 2.1. We will now prove that Equation (5) satisfies the detailed balance condition and is therefore the equilibrium distribution.

$$\pi(\mathbf{n})q(\mathbf{n}, H_k \mathbf{n}) = \pi(H_k \mathbf{n})q(H_k \mathbf{n}, \mathbf{n}), \quad \forall \mathbf{n} \in \mathcal{N} \quad (9)$$

and

$$\pi(\mathbf{n})q(\mathbf{n}, H_{j\bullet} \mathbf{n}) = \pi(H_{j\bullet} \mathbf{n})q(H_{j\bullet} \mathbf{n}, \mathbf{n}), \quad \forall \mathbf{n} \in \mathcal{N} \quad (10)$$

The total number of infected nodes in the entire network is $1^T \mathbf{n} = n$. The number of infected neighbors of node k is

$$d_k = \sum_{j=0}^{N-1} 1(n_j = 1)A_{jk} \quad (11)$$

In state $H_k \mathbf{n}$, we gain 1 additional infected node: $1^T H_k \mathbf{n} = n + 1$. Furthermore, we gain as many additional balanced infected edges as the number of infected neighbor of node k :

$$\frac{(H_k \mathbf{n})^T A(H_k \mathbf{n})}{2} = \frac{\mathbf{n}^T A \mathbf{n}}{2} + d_k$$

In state $H_{j\bullet} \mathbf{n}$, we loose an infected node: $1^T H_{j\bullet} \mathbf{n} = n - 1$. Furthermore, we loose as many balanced infected edges as the number of infected neighbors of node j :

$$\frac{(H_{j\bullet} \mathbf{n})^T A(H_{j\bullet} \mathbf{n})}{2} = \frac{\mathbf{n}^T A \mathbf{n}}{2} - d_j$$

Using the transition rates defined by (1), (3) and the proposed form of the equilibrium distribution $\pi(\mathbf{n})$ in (5), the detailed balance equations are satisfied.

$$\frac{1}{Z} \left(\frac{\lambda}{\mu} \right)^n \gamma^{\frac{\mathbf{n}^T A \mathbf{n}}{2}} \lambda \gamma^{d_k} = \frac{1}{Z} \left(\frac{\lambda}{\mu} \right)^{n+1} \gamma^{\frac{\mathbf{n}^T A \mathbf{n}}{2} + d_k} (\mu) \quad (12)$$

and

$$\frac{1}{Z} \left(\frac{\lambda}{\mu} \right)^n \gamma^{\frac{\mathbf{n}^T A \mathbf{n}}{2}} (\mu) = \frac{1}{Z} \left(\frac{\lambda}{\mu} \right)^{n-1} \gamma^{\frac{\mathbf{n}^T A \mathbf{n}}{2} - d_j} \lambda \gamma^{d_j} \quad (13)$$

□

B. PROOF OF LEMMA 3.1

We want to show that for a k -regular graph and $0 \leq \frac{\lambda}{\mu} \leq 1, \gamma > 1$, where s is the number of infected nodes in the graph

$$\left(\frac{\lambda}{\mu} \right)^s \gamma^{\frac{\mathbf{n}^s T A \mathbf{n}^s}{2}} \leq \left(\frac{\lambda}{\mu} \gamma^{\frac{k}{2}} \right)^s, \quad s = 0, 1, \dots, N, \mathbf{n}^s \in \mathcal{N}_s$$

Furthermore, the relationship holds with equality for $\mathbf{n}^0$ and $\mathbf{n}^N$.

Proof. Note that for the k -regular graph

$$A \mathbf{n}^s = \begin{bmatrix} \sum_{i=1}^N A_{1i} n_i^s \\ \sum_{i=1}^N A_{2i} n_i^s \\ \vdots \\ \sum_{i=1}^N A_{Ni} n_i^s \end{bmatrix} \leq \begin{bmatrix} k \\ k \\ \vdots \\ k \end{bmatrix}, \quad \text{if } k \leq s$$

and

$$A \mathbf{n}^s = \begin{bmatrix} \sum_{i=1}^N A_{1i} n_i^s \\ \sum_{i=1}^N A_{2i} n_i^s \\ \vdots \\ \sum_{i=1}^N A_{Ni} n_i^s \end{bmatrix} \leq \begin{bmatrix} s \\ s \\ \vdots \\ s \end{bmatrix}, \quad \text{if } k \geq s$$

$$\mathbf{n}^{sT} A \mathbf{n}^s \leq sk, \text{ if } k \leq s \quad (14)$$

$$\mathbf{n}^{sT} A \mathbf{n}^s \leq s^2 \leq sk, \text{ if } k \geq s \quad (15)$$

With $\gamma > 1$, we know that

$$\left(\frac{\lambda}{\mu} \right)^s \gamma^{\frac{\mathbf{n}^s T A \mathbf{n}^s}{2}} \leq \left(\frac{\lambda}{\mu} \right)^s \gamma^{\frac{sk}{2}}, \forall s = 0, 1, \dots, N$$

When $s = 0$, equality is clearly satisfied. When $s = N$, all the nodes in the graph are infected. Then the number of edges whose end nodes are infected is $\frac{kN}{2}$. Hence the equality is also satisfied. □