

# Diffusion in Social Networks as SIS Epidemics: Beyond Full Mixing and Complete Graphs

June Zhang, *Student Member, IEEE*, and José M. F. Moura, *Fellow, IEEE*

**Abstract**—Peer influence and interactions between agents in a population give rise to complex, nonlinear behaviors. This paper adopts the SIS (susceptible-infected-susceptible) framework from epidemiology to analytically study how network topology affects the diffusion of ideas/opinions/beliefs/innovations in social networks. We introduce the *scaled SIS process*, which models peer influence as neighbor-to-neighbor infections. We model the scaled SIS process as a continuous-time Markov process and derive for this process its closed form equilibrium distribution. The adjacency matrix that describes the underlying social network is explicitly reflected in this distribution. The paper shows that interesting population asymptotic behaviors occur for scenarios where the individual tendencies of each agent oppose peer influences. Specifically, we determine how the most probable configuration of agent states (i.e., the population configuration with maximum equilibrium distribution) depends on both model parameters and network topology. We show that, for certain regions of the parameter space, this and related issues reduce to standard graph questions like the maximum independent set problem.

**Index Terms**—Complete multipartite graph, diffusion process,  $k$ -regular graph, Markov process, maximum independent set, SIS epidemics, social influence, social networks.

## I. INTRODUCTION

SOCIAL networks capture relationships, which may give insights to peer influence, between agents in a population. We are interested in studying how the network topology affects the asymptotic behavior of diffusion processes in social networks. To study this effect, we model the diffusion process of ideas/opinions/beliefs/innovations over a finite-sized, static, connected social network as an epidemics process.

We call our model the *scaled SIS process*. We assume that each agent can be in one of two possible states: as a *spreader*, the agent influences its non-spreader neighbors to adopt the disseminating idea/opinion/belief/innovation; and as an *adopter*,

where the agent is a non-spreader and is susceptible to influences. We choose the SIS (susceptible-infected-susceptible) epidemics model framework because we assume that agents may be spreaders or adopters interchangeably. The scaled SIS process captures micro interactions at the agent level that reflect the following evolutions in the diffusion process: 1) topology independent healing (i.e., spreaders spontaneously become adopters); 2) topology independent infection (i.e., adopters spontaneously become spreaders); and 3) topology dependent infection (i.e., adopters become spreaders through peer-to-peer influence).

At the abstract level, epidemics can and have been used for modeling the propagation of social contagions in networks since the stochastic evolution mechanisms of both processes are similar [1]–[6]. The primary challenge is in how to incorporate the network topology constraint as epidemiology traditionally focus on studying full mixing populations (i.e., a population where every agent can interact directly with everyone else); there is still no clear consensus on how to extend the analysis to heterogeneous network topologies [7].

While it may be easier to incorporate network topology in simulation based models, simulating epidemics on large networks is resource consuming and infeasible for moderately sized populations [5]. Models that can be mathematically analyzed are not as restricted by computation resources. Existing analytical methods for modeling SIS epidemics over heterogeneous networks account for topology in one of two fashions: 1) topology is characterized through statistical properties such as degree distribution and degree correlations. These methods consider the diffusion process in the limit of large networks using mean field analysis [2], [5], [8]–[10]; and 2) topology is captured exactly by the adjacency matrix of the underlying network, which is static and finite-sized. The epidemics is treated as a Markov process and the infection rate is assumed to be dependent on the number of infected neighbors [11]–[16]. These works usually attempt only to either characterize mean epidemic lifetime (discounting either healing or exogenous infection so that the asymptotic state of the system is known a priori), or analyze macroscopic behaviors such as the fraction of infected agents in asymptotic time. We are interested in going beyond these moment characteristics; in particular, we are interested in the microscopic characterization of the asymptotic state of the network (i.e., the configuration of all jointly held agent opinions in the network).

The *scaled SIS process* that we consider is similar in some aspects to the class of models in [11]–[16], but differs in important respects. First, for a given range of the model parameters, the

Manuscript received September 15, 2013; revised February 05, 2014; accepted March 26, 2014. Date of publication April 02, 2014; date of current version July 16, 2014. This work was supported in part by the Air Force Office of Scientific Research (AFOSR) under Grant FA95501010291, and by the National Science Foundation (NSF) under Grants CCF1011903 and CCF1018509. The guest editor coordinating the review of this manuscript and approving it for publication was Prof. Patrick Wolfe.

J. Zhang is with the Department of Electrical and Computer Engineering, Carnegie Mellon University, Pittsburgh, PA 15213 USA (e-mail: junez@andrew.cmu.edu).

J. M. F. Moura is with the Department of Electrical and Computer Engineering, Carnegie Mellon University, Pittsburgh, PA 15213 USA, and also with the Center for Urban Science and Progress (CUSP), New York University (NYU), NY 10012 USA (e-mail: moura@ece.cmu.edu).

Color versions of one or more of the figures in this paper are available online at <http://ieeexplore.ieee.org>.

Digital Object Identifier 10.1109/JSTSP.2014.2314858

scaled SIS process can either mimic the behavior of traditional diffusion processes or can, for another choice of the parameters, account for opposition behavior that may arise in local interactions; for example agents may be less likely to adopt an opinion shared by many of their neighbors (or in epidemics, agents may seek measures to protect their health when they observe many of their neighbors becoming infected). Second, the scaled SIS process leads to a reversible Markov process, which makes it more amenable to analysis. Third, we are able to derive the equilibrium distribution of the process, which quantifies exactly how the time asymptotic opinions of each agent are jointly likely. We will show that the most likely configuration of jointly held agent opinions exhibits threshold behavior that depends on the model parameters and the network topology; further, for some classes of graphs, we can mathematically characterize the impact of the topology on this threshold behavior. For other classes of graphs, the threshold behavior is more complicated and the most probable configuration may be any from a given set of configurations. Finally we demonstrate that, in a specific region of parameter values, the most probable configuration corresponds to the solution of the maximum independent set graph problem.

We describe the scaled SIS process in detail in Section II. Section III shows that it can be modeled as a reversible continuous-time Markov process and presents its closed form equilibrium distribution. In Section IV, we pose the problem of solving for the most probable configuration (i.e., the configuration of all agent states that maximizes the equilibrium distribution). Sections V and VI discuss the impact of network topologies on the most probable configuration under different parameter regimes. Section VII shows how model parameters affect the probability of the most probable configuration. Finally, Section VIII concludes the paper.

## II. THE MODEL

This section presents the network and shows how we model the diffusion process in social networks as an epidemics process. Section II-A describes the network of agents. Section II-B introduces the scaled SIS process, and Section II-C details the transition rate matrix associated with the corresponding continuous-time Markov process.

### A. Network of Agents

The population of  $N$  agents is represented by an unweighted, undirected, simple graph,  $G(V, E)$ , where  $V$  is the set of nodes representing the agents, and  $E$  is the set of edges representing the dependencies, contacts, or relationships amongst the agents. The topology of  $G$  is captured by the symmetric,  $N \times N$  adjacency matrix,  $A$ . We assume that  $G$  is connected and that it remains static over time.

The agents connected to the  $i^{\text{th}}$  node are its *neighbors*; the degree,  $k_i$ , is the total number of neighbors of agent  $i$ . In this paper, we will consider graphs of arbitrary topology and graphs with certain structures such as:

- **k-regular graph.** The network  $G(V, E)$  is a  $k$ -regular graph if and only if  $k_i = k, \forall i = 1, 2, \dots, N$ . The complete graph is a  $k$ -regular graph with degree  $N - 1$ .
- **Bipartite graph.** Partition the vertex set into 2 sets:  $V = \{V_1, V_2\}$  where  $|V_1| = N_1, |V_2| = N_2$ . We will refer to the

vertex sets  $V_i$  as *islands* or *supernodes*. The graph  $G(V, E)$  is a bipartite graph if and only if: for any pairs of nodes  $a, b \in V_i$  for  $i \in \{1, 2\}, \{a, b\} \notin E$ .

- **Complete multipartite graph.** Partition the vertex set into  $m$  supernodes:  $V = \{V_1, \dots, V_m\}$  where  $|V_1| = N_1, |V_2| = N_2$ , etc. The graph  $G(V, E)$  is a complete multipartite graph if and only if

- 1) For any pairs of nodes  $a, b \in V_i$  for any  $i \in \{1, \dots, m\}, \{a, b\} \notin E$ .
- 2) For any pairs of nodes  $a \in V_i, b \in V_j$ , and  $i \neq j$ , if  $\{a, b\} \in E$ , then  $\{a, c\} \in E, \forall c \in V_j$ ; islands  $V_i$  and  $V_j$  are connected. We define the indicator function,  $\mathbb{1}(V_i \sim V_j) = 1$  if  $V_i$  and  $V_j$  are connected and 0 otherwise. For any node in island  $V_i$ , we call  $\sum_{j, j \neq i} \mathbb{1}(V_i \sim V_j) N_j$  the *inter-degree*,  $k_i^{\text{inter}}$ , of node  $i$  (i.e., the total number of edges to nodes in other islands).

The **complete bipartite graph** is a complete multipartite graph with only 2 islands.

- **Complete multipartite graph with  $k$ -regular islands.** The graph  $G(V, E)$  is a complete multipartite graph with  $k$ -regular islands if and only if it is a complete multipartite graph and, within the  $i^{\text{th}}$  island, each node is connected to  $k_i^{\text{intra}}$  other nodes in the same island. We will refer to  $k_i^{\text{intra}}$  as the *intra-degree* of the node. The total degree of a node in  $G(V, E)$  is its intra-degree plus its inter-degree.

### B. Scaled SIS Process

The state of the  $i^{\text{th}}$  agent in the network is denoted by  $x_i$ . If  $x_i = 0$ , the agent is an *adopter*. The adopter holds the nondisseminating opinion. In the terminology of epidemics, this agent is healthy and is a *susceptible*. If  $x_i = 1$ , the agent is a *spreader*. The spreader holds the disseminating opinion. It is infected; we call infected agents *infectives*. The *network configuration* is the  $N$ -tuple collection of all the individual agent states.

$$\mathbf{x} = [x_1, x_2, \dots, x_N]^T$$

We will refer to  $\mathbf{x}$  as the *configuration* or the *network state* interchangeably and to the individual elements of  $\mathbf{x}$  as either the *agent state* or the *node state*. The set of all possible network configurations is the network state space  $\mathcal{X} = \{\mathbf{x}\}$ , cardinality  $|\mathcal{X}| = 2^N$ .

We identify two special configurations:

$$\begin{aligned} \mathbf{x}^0 &= [0, 0, \dots, 0]^T, \text{ all the agents are susceptibles} \\ \mathbf{x}^N &= [1, 1, \dots, 1]^T, \text{ all the agents are infectives.} \end{aligned}$$

The network configuration,  $\mathbf{x}$ , changes with time as agents adopt the nondisseminating opinion (i.e., they heal) or the disseminating opinion (i.e., they become infected). We represent this SIS epidemics over the network by a stochastic process  $X(t), t \geq 0$ ; at any point in time,  $X(t) = \mathbf{x}$ , for some  $\mathbf{x} \in \mathcal{X}$ . We assume that every agent in the population behaves in the following manner:

- 1) **Healing.** An infective heals in a length of time,  $T$ , that is exponentially distributed with rate  $\mu > 0$ . Borrowing terminology from the field of system reliability,  $T$  is the

*downtime*. Once healed, the agent is susceptible to reinfection. Since healing is a spontaneous action and the parameter  $\mu$  is the same for all the agents, it is considered to be network topology independent. We will refer to  $\mu$  as the *healing rate*. Since  $T$  is exponentially distributed, the average downtime is

$$E[T] = \frac{1}{\mu}. \quad (1)$$

- 2) *Infection*. A susceptible becomes infected in a length of time,  $\hat{T}$ , that is exponentially distributed with rate  $\lambda\gamma^d > 0$ , where  $d$  is the number of infected neighbors of the susceptible agent; the scaled SIS process is dependent on the network topology due to  $d$ . From system reliability,  $\hat{T}$  is the *uptime*. The average uptime is

$$E[\hat{T}] = \frac{1}{\lambda\gamma^d}. \quad (2)$$

We separate the adoption of the disseminating opinion into two types as per standard epidemics model: exogenous and endogenous infection. Exogenous infection accounts for the scenario where adopters spontaneously become spreaders without peer influence. Endogenous infection accounts for the scenario where adopters become spreaders due to influence from having neighbors who are spreaders.

- a) *Exogenous Infection*. When  $d = 0$ , the infection rate is  $\lambda > 0$ . We can interpret  $\lambda$  as the *exogenous infection rate*, where the source of infection is external to the population. Since this infection is a spontaneous action, the parameter  $\lambda$  is the same for all the agents. This is the *topology independent infection rate*.
- b) *Endogenous Infection*. We call  $\gamma^d$  the *endogenous infection rate*, where the source of infection is due to contagion from infected neighbors. Since it is dependent on the number of infected neighbors, it is limited by the total number of neighbors. Therefore, it may be different for different agents depending on the network topology. Therefore, we will refer to  $\gamma$  as the *topology dependent infection rate*.

We assume that these exponentially distributed downtime and uptime are all independent and that multiple events (i.e., infection, healing) can not occur simultaneously.

Previous works that model the SIS epidemics over networks [12], [16] use the infection rate form  $\lambda + \gamma d$  where  $d$  is the number of infected neighbors of the susceptible agent. This leads to the following interpretation regarding the uptime

$$\hat{T} = \min(\alpha, \beta),$$

where  $\alpha \sim \exp(\lambda)$  and  $\beta \sim \exp(\gamma d)$ . Then  $\hat{T} \sim \exp(\lambda + \gamma d)$ . In our model, we consider the uptime,  $\hat{T}$ , as an exponentially distributed random variable that is scaled by the exogenous infection rate. Then  $\hat{T} \sim \exp(\lambda\gamma^d)$ . This is the reason why we refer to  $X(t)$  as the *scaled SIS process*.

There are several reasons why the infection rate form  $\lambda\gamma^d$  is a feasible alternative to  $\lambda + \gamma d$ : 1) both rates either increase or decrease monotonically depending on the value of  $\gamma$ ; 2) multiplicative type relationships have been shown as one of the hall-

marks characterizing network behavior [1], [2]; finally and very importantly, 3) in quantifying analytically the role of the population topology, we will see in Section III that changing the infection rate to  $\lambda\gamma^d$  will make the underlying process more amenable to analysis.

### C. Continuous-Time Markov Process

Under the assumptions stated in the previous section, the network state  $X(t)$  is a time-homogenous, irreducible, finite state, continuous-time Markov process, [17]. Each state of the Markov process is a particular network configuration  $\mathbf{x}$ ; the state space of the Markov process is the network state space,  $\mathcal{X}$ . The continuous-time Markov process is characterized by an *infinitesimal matrix*, also known as the *rate matrix*,  $Q$ . The infinitesimal matrix is a nonnegative, asymmetric  $2^N \times 2^N$  matrix.

The summation of each row of  $Q$  equals to 0, therefore  $Q_{i,i} = -\sum_{j \neq i} Q_{i,j}$ . The off-diagonal entry,  $Q_{i,j}$ , corresponds to the transition rate between 2 configurations  $\mathbf{x} = i$ ,  $\mathbf{x}' = j$ , where  $i$  and  $j$  are the decimal representations of  $\mathbf{x}$  and  $\mathbf{x}'$  respectively. There are 3 possible transition types:

- 1) Markov process transitions from a network configuration  $\mathbf{x} = [x_1, x_2, \dots, x_j, \dots, x_k = 0, \dots, x_N]^T$  to a configuration,  $\mathbf{x}'$ , where susceptible agent  $k$  becomes an infective:

$$\mathbf{x}' = [x_1, x_2, \dots, x_k = 1, \dots, x_N]^T.$$

$X(t)$  jumps from network state  $\mathbf{x}$  to the network state  $\mathbf{x}'$  with transition rate

$$Q_{i,j} = q(\mathbf{x}, \mathbf{x}') = \lambda\gamma^{d_k}, \quad (3)$$

where  $d_k = \sum_{j=1}^N \mathbb{1}(n_j = 1)A_{jk}$  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  $G$ .

- 2) Markov process transitions from a network configuration  $\mathbf{x} = [x_1, x_2, \dots, x_j = 1, \dots, x_k, \dots, x_N]^T$  to a configuration,  $\mathbf{x}'$ , where infective agent  $j$  heals into a susceptible:

$$\mathbf{x}' = [x_1, x_2, \dots, x_j = 0, \dots, x_N]^T.$$

$X(t)$  jumps from network state  $\mathbf{x}$  to the network state  $\mathbf{x}'$  with transition rate:

$$Q_{i,j} = q(\mathbf{x}, \mathbf{x}') = \mu. \quad (4)$$

- 3) Besides the above 1) and 2), no other transitions are possible from network configurations  $\mathbf{x}$  to  $\mathbf{x}'$ ; then  $Q_{i,j} = q(\mathbf{x}, \mathbf{x}') = 0$  and  $Q_{j,i} = q(\mathbf{x}', \mathbf{x}) = 0$ .

### III. EQUILIBRIUM DISTRIBUTION OF SCALED SIS PROCESS, $X(t)$

The process  $X(t)$  is a stationary, irreducible, finite-state continuous-time Markov process. Then, its equilibrium distribution,  $\pi$ , always exists and is unique. Furthermore,  $\pi$  is the limiting distribution of the process [17]:

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

For a continuous-time Markov process, the equilibrium distribution satisfies the full balanced equation [17]:

$$\pi(\mathbf{x}) \sum_{\mathbf{x}' \in \mathcal{X}} q(\mathbf{x}, \mathbf{x}') = \sum_{\mathbf{x}' \in \mathcal{X}} \pi(\mathbf{x}') q(\mathbf{x}', \mathbf{x}), \quad \mathbf{x} \in \mathcal{X}. \quad (5)$$

The equilibrium distribution can be found by solving the eigenvalue/eigenvector problem  $\pi Q = 0$  (i.e.,  $\pi(\mathbf{x})$  is the left eigenvector corresponding to the null eigenvalue of  $Q$ ). Perron-Frobenius theory guarantees that, for the irreducible Markov process,  $X(t)$ , such a nonnegative eigenvector exists [18]. However, directly finding this eigenvector may be computationally infeasible since the dimension of the  $Q$  matrix is exponential in the size of the network population. For example, a population of 30 agents will have a  $Q$  matrix that is  $2^{30} \times 2^{30}$ ; solving this eigenvector/eigenvalue problem would be computationally intractable.

We prove in this section that, by assuming the rate forms of (3) and (4),  $X(t)$  is a special type of Markov process known as a *reversible* Markov process: a Markov process whose stochastic behavior forward in time is the same as its stochastic behavior reversed in time. Reversible Markov processes are useful because their equilibrium distribution,  $\pi(\mathbf{x})$ , satisfies the detailed balance equation [17]

$$\pi(\mathbf{x}) q(\mathbf{x}, \mathbf{x}') = \pi(\mathbf{x}') q(\mathbf{x}', \mathbf{x}), \quad \mathbf{x}, \mathbf{x}' \in \mathcal{X} \quad (6)$$

in addition to the full balanced (5). We can utilize the detailed balanced equation to find the equilibrium distribution,  $\pi(\mathbf{x})$  for  $X(t)$ , as we show next.

*Theorem III.1: The scaled SIS process,  $X(t)$ , is a reversible Markov process and the equilibrium distribution is*

$$\pi(\mathbf{x}) = \frac{1}{Z} \left( \frac{\lambda}{\mu} \right)^{1^T \mathbf{x}} \gamma^{\mathbf{x}^T A \mathbf{x} / 2}, \quad \mathbf{x} \in \mathcal{X} \quad (7)$$

where  $Z$  is the partition function defined as

$$Z = \sum_{\mathbf{x} \in \mathcal{X}} \left( \frac{\lambda}{\mu} \right)^{1^T \mathbf{x}} \gamma^{\mathbf{x}^T A \mathbf{x} / 2}. \quad (8)$$

According to the equilibrium distribution (7), the long run probability of the network configuration,  $\mathbf{x}$ , is completely described by 2 statistics:  $1^T \mathbf{x} = \sum_{i=1}^N x_i$ , the total number of infected nodes in the network configuration, and  $\mathbf{x}^T A \mathbf{x} / 2 = (1/2) \sum_{i=1}^N \sum_{j=0}^{N-1} A_{ij} x_i x_j$ , the total number of edges where both end nodes are infected; we will refer to these edges as *infected edges*. Note that  $0 \leq 1^T \mathbf{x} \leq N$  and  $0 \leq \mathbf{x}^T A \mathbf{x} / 2 \leq |E|$ , where  $N$  is the total number of agents in the network and  $|E|$  is the total number of edges. Reference [14] derived a generalized equilibrium distribution, which accounts for more than 2 agents states. We provide a more topology dependent intuition to the equilibrium distribution and will use it to address different questions.

We can interpret the equilibrium distribution in (7) as the product of two terms: a topology independent term and a topology dependent term. The topology independent term is determined by a ratio of the parameters  $\lambda, \mu$  and by  $1^T \mathbf{x}$ , the total number of infectives in configuration  $\mathbf{x}$ ; these values are independent of the topology. The topology dependent term explicitly accounts for the network topology in the form of the adjacency matrix,  $A$ . It is determined by the topology

dependent infection rate  $\gamma$  and  $\mathbf{x}^T A \mathbf{x} / 2$ , the total number of infected edges in configuration  $\mathbf{x}$ .

*Proof:* To prove Theorem III.1, we use the following theorem regarding *reversible* Markov processes.

*Theorem III.2 (Theorem 1.3 in [17]): A stationary Markov process is reversible if and only if there exists a collection of positive numbers  $\pi(\mathbf{x}), \mathbf{x} \in \mathcal{X}$ , summing to unity that satisfy the detailed balance (6). When there exists such a collection  $\pi(\mathbf{x}), \mathbf{x} \in \mathcal{X}$ , it is the equilibrium distribution of the process.*

We show now that (7) satisfies the detailed balance equation (6) and is therefore the equilibrium distribution.

- 1) Consider the scenario where the Markov process jumps from state  $\mathbf{x}$ , where the  $k^{\text{th}}$  agent is susceptible, to the state  $\mathbf{x}'$  where it becomes an infective:

$$\mathbf{x}' = [x_1, x_2, \dots, x_k = 1, \dots, x_N]^T.$$

By (3),  $q(\mathbf{x}, \mathbf{x}') = \lambda \gamma^{d_k}$  where  $d_k = \sum_{j=1}^N 1(x_j = 1) A_{jk}$  is the total number of infected neighbors of node  $k$ . Using (7), the LHS of (6) is

$$\begin{aligned} \pi(\mathbf{x}) q(\mathbf{x}, \mathbf{x}') &= \frac{1}{Z} \left( \frac{\lambda}{\mu} \right)^{1^T \mathbf{x}} \gamma^{\mathbf{x}^T A \mathbf{x} / 2} (\lambda \gamma^{d_k}) \\ &= \frac{1}{Z} \left( \frac{\lambda^{(1^T \mathbf{x} + 1)}}{\mu^{1^T \mathbf{x}}} \right) \gamma^{\mathbf{x}^T A \mathbf{x} / 2 + d_k}. \end{aligned}$$

By (4),  $q(\mathbf{x}', \mathbf{x}) = \mu$ . We know that in state  $\mathbf{x}'$  there is one more additional infected node than in state  $\mathbf{x}$ . Furthermore, transitioning from  $\mathbf{x}$  to  $\mathbf{x}'$ , we gain as many additional infected edges as the number of infected neighbors of agent  $k$ :

$$\frac{(\mathbf{x}')^T A (\mathbf{x}')}{2} = \frac{\mathbf{x}^T A \mathbf{x}}{2} + d_k.$$

Therefore, the RHS of (6) is

$$\begin{aligned} \pi(\mathbf{x}') q(\mathbf{x}', \mathbf{x}) &= \frac{1}{Z} \left( \frac{\lambda}{\mu} \right)^{1^T \mathbf{x} + 1} \gamma^{\mathbf{x}^T A \mathbf{x} / 2 + d_k} (\mu) \\ &= \frac{1}{Z} \left( \frac{\lambda^{(1^T \mathbf{x} + 1)}}{\mu^{1^T \mathbf{x}}} \right) \gamma^{\mathbf{x}^T A \mathbf{x} / 2 + d_k}. \end{aligned}$$

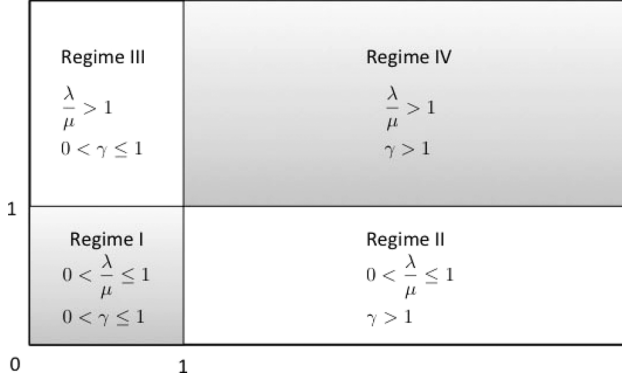
Since the RHS of (6) is the same as its LHS, the detailed balance condition (6) is satisfied.

- 2) Consider the scenario where the Markov process jumps from  $\mathbf{x}$ , where the  $j^{\text{th}}$  agent is infective, to the state  $\mathbf{x}'$  where it is healed:

$$\mathbf{x}' = [x_1, x_2, \dots, x_j = 0, \dots, x_N]^T.$$

We know that in state  $\mathbf{x}'$  there is one fewer infected node than in state  $\mathbf{x}$ . Furthermore, transitioning from  $\mathbf{x}$  to  $\mathbf{x}'$ , we lose as many infected edges as the number of infected neighbors of node  $j$ . Therefore, the LHS of (6) is

$$\begin{aligned} \pi(\mathbf{x}) q(\mathbf{x}, \mathbf{x}') &= \frac{1}{Z} \left( \frac{\lambda}{\mu} \right)^{1^T \mathbf{x}} \gamma^{\mathbf{x}^T A \mathbf{x} / 2} (\mu) \\ &= \frac{1}{Z} \left( \frac{\lambda^{1^T \mathbf{x}}}{\mu^{(1^T \mathbf{x} - 1)}} \right) \gamma^{\mathbf{x}^T A \mathbf{x} / 2}. \end{aligned}$$

Fig. 1. Parameter regimes of the scaled SIS process,  $X(t)$ .

The RHS of (6) is

$$\begin{aligned} \pi(\mathbf{x}')q(\mathbf{x}', \mathbf{x}) &= \frac{1}{Z} \left( \frac{\lambda}{\mu} \right)^{1^T \mathbf{x} - 1} \gamma^{(\mathbf{x}^T A \mathbf{x} / 2 - d_j)} (\lambda \gamma^{d_j}) \\ &= \frac{1}{Z} \left( \frac{\lambda^{1^T \mathbf{x}}}{\mu^{(1^T \mathbf{x} - 1)}} \right) \gamma^{\mathbf{x}^T A \mathbf{x} / 2}. \end{aligned}$$

The detailed balance condition in (6) is again satisfied.

- 3) If  $q(\mathbf{x}, \mathbf{x}') = 0$ , then  $q(\mathbf{x}', \mathbf{x}) = 0$ , and the detailed balance condition is satisfied trivially. ■

Theorem III.1 gives the equilibrium distribution of the scaled SIS process. It involves only two statistics: the number of infected nodes  $1^T \mathbf{x}$  and the number of infected edges  $\mathbf{x}^T A \mathbf{x} / 2$ . In practice, it may not be trivial to find the partition function (8). Still, knowledge of the equilibrium distribution  $\pi(\mathbf{x})$  is useful to address many interesting questions.

#### IV. MOST PROBABLE CONFIGURATION, $\mathbf{x}^*$

If the size of the population is large, the computation of the partition function (8) is infeasible. We focus our analysis on finding the most probable configuration,  $\mathbf{x}^*$  of  $X(t)$  where

$$\mathbf{x}^* = \arg \max_{\mathbf{x} \in \mathcal{X}} \pi(\mathbf{x}) = \arg \max_{\mathbf{x} \in \mathcal{X}} \left\{ \left( \frac{\lambda}{\mu} \right)^{1^T \mathbf{x}} \gamma^{\mathbf{x}^T A \mathbf{x} / 2} \right\}. \quad (9)$$

Solving for  $\mathbf{x}^*$  is a quadratic integer programming problem. Since  $A$ , the adjacency matrix, is an indefinite matrix, the problem is in general NP-hard. We will show that we can solve (9) under specific parameter regimes and for certain network topologies. In addition, under particular conditions, the solution to (9) corresponds to the maximum independent set problem, a well-studied graph theoretic problem [19].

##### A. Parameter Regimes

The equilibrium distribution of the scaled SIS process,  $X(t)$ , is dependent on two positive parameters:  $\lambda/\mu$  and  $\gamma$ . We divide the parameter space into 4 regimes, as shown in Fig. 1.

- I) **Healing Dominant:**  $0 < \lambda/\mu \leq 1$ ,  $0 < \gamma \leq 1$
- II) **Endogenous Infection Dominant:**  $0 < \lambda/\mu \leq 1$ ,  $\gamma > 1$
- III) **Exogenous Infection Dominant:**  $\lambda/\mu > 1$ ,  $0 < \gamma \leq 1$
- IV) **Infection Dominant:**  $\lambda/\mu > 1$ ,  $\gamma > 1$

We first provide intuition regarding these parameter regimes. When  $0 < \lambda/\mu < 1$ , the average downtime of an agent, given

by (1), on average, a standalone agent is in the infected state shorter than it is in the susceptible state. In the context of social contagion, we interpret this to mean that single agents inherently prefer to be adopters instead of spreaders. When  $\lambda/\mu > 1$ , the average downtime of an agent is longer than the average topology independent uptime; on average, the standalone agent is infected for longer than it is healthy. This means that agents prefer to be spreaders instead of adopters. When  $\lambda/\mu = 1$ , the average downtime and the average topology independent uptime are the same.

When  $\gamma > 1$ , additional infected neighbors of a healthy agent will decrease the same agent's average uptime (2); additional infections exert a loading effect on the population. For example, in the case of epidemics, a single infection will be followed quickly by additional infections. In the context of social contagion, having more neighbors who are spreaders will positively influence an adopter agent to adjust its opinion to that of the spreaders.

On the contrary, when  $0 < \gamma < 1$ , additional infected neighbors of a healthy agent will increase the average uptime of the agent. This means that additional infections will actually strengthen the population. For example, in a system with active countermeasures, an increase in the number of infected neighbors is a signal to booster the susceptible agent's defense; therefore, its uptime may increase. Another way to interpret this scenario is that having more spreaders as neighbors will negatively influence an adopter agent to switch its opinion. We can easily model this behavior with the infection rate  $\lambda \gamma^d$ ; this would be more difficult with the form  $\lambda + \gamma d$ . When  $\gamma = 1$ , then the state of the node is immune to the states of the node's neighbors.

Since the network topology affects only the  $\gamma$  term, we will refer to the process controlled by the  $\gamma$  parameter as the *topology dependent process* and the one controlled by  $\lambda/\mu$  as the *topology independent process*. When the topology dependent process and the topology independent process agree with each other, as in regimes I and IV, the solution for (9) can be solved easily.

In regime I), Healing Dominant,  $0 < \lambda/\mu \leq 1$ ,  $0 < \gamma \leq 1$ , the healing process dominates both the exogenous infection and the endogenous infection processes. Therefore, the most probable configuration in equilibrium is  $\mathbf{x}^* = \mathbf{x}^0 = [0, 0, \dots, 0]^T$ , the configuration where all the nodes are susceptibles. This holds for any network topology,  $A$ , and  $\pi(\mathbf{x}^0) = 1/Z$ .

In regime IV), Infection Dominant,  $\lambda/\mu > 1$ ,  $\gamma > 1$ , both exogenous infection and endogenous infection processes dominate the healing process. Therefore, the most probable configuration is  $\mathbf{x}^* = \mathbf{x}^N = [1, 1, \dots, 1]^T$ , the configuration where all the nodes are infectives. This holds for any network topology,  $A$ , and

$$\pi(\mathbf{x}^N) = \frac{1}{Z} \left( \frac{\lambda}{\mu} \right)^N \gamma^{(1/2)|E|}.$$

Since the network topology plays no role in solving (9) in these two regimes, we do not discuss them further. For the rest of the paper, we focus our analysis on regimes II) Endogenous Infection Dominant and III) Exogenous Infection Dominant, where the topology independent process opposes the topology dependent process. This introduces an additional complexity

into the system since there is now an opposing process which counters the influence of the diffusion process in some respect. More accurately, the diffusion process that we model in regimes II) and III) can be classified as *diffusion-with-opposition* processes. We will show for these two regimes, the solution to (9) exhibits phase transition that is dependent on both the network topology and model parameters.

## V. REGIME II) ENDOGENOUS INFECTION DOMINANT:

$$0 < \lambda/\mu \leq 1, \gamma > 1$$

In this regime, the topology independent infection rate  $\lambda$  is smaller than the topology independent healing rate  $\mu$ . If we do not account for the topology dependent infection process through the population (i.e.,  $\gamma = 1$ ), then the most probable network configuration would be  $\mathbf{x}^0 = [0, 0, \dots, 0]^T$ . In this scenario, agents individually prefer to be adopters instead of spreaders.

However, since  $\gamma > 1$ , additional infected agents will increase the topology dependent infection rate; in the social contagion context, peer influence favors the conversion of adopters to spreaders. To maximize the probability in (9), we seek the configurations that minimize the number of infected nodes while maximizing the number of infected edges; hence, nodes with high degree are important. Intuitively, if  $\lambda/\mu$  is very small and  $\gamma$  is close to 1, then the behavior of the process will be dictated by the topology independent process; a good guess for  $\mathbf{x}^*$  is  $\mathbf{x}^0 = [0, 0, \dots, 0]^T$ . When  $\gamma$  is very large and  $\lambda/\mu$  is close to 1, the process behavior will be dictated by the topology dependent process; a good guess for  $\mathbf{x}^*$  is  $\mathbf{x}^N = [1, 1, \dots, 1]^T$ .

In the next section, we will show that, for  $k$ -regular, complete multipartite, and complete multipartite with  $k$ -regular island networks, we can derive the exact threshold for which the solution of (9) goes from  $\mathbf{x}^0$  to  $\mathbf{x}^N$ . More interestingly, we observe that, for other types of network topologies, the most probable configuration,  $\mathbf{x}^*$ , may correspond to configurations other than  $\mathbf{x}^0$  or  $\mathbf{x}^N$  for certain ranges of the parameters; we will refer to these configurations as the *spurious most probable configurations*. In Section V-E, we will point out some properties of these spurious configurations using example topologies.

### A. $k$ -Regular Graph

Consider the  $k$ -regular graph,  $G(V, E)$ . We partition the state space,  $\mathcal{X}$ , as

$$\mathcal{X} = \mathcal{X}_0 \cup \mathcal{X}_1 \cup \dots \cup \mathcal{X}_N,$$

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

**Lemma V.1 (Proof in Appendix A):** When  $0 \leq \lambda/\mu \leq 1, \gamma > 1$  and for  $s = 0, 1, \dots, N, \mathbf{x}^s \in \mathcal{X}_s$ , the unnormalized equilibrium distribution for a  $k$ -regular graph is upperbounded by an exponential function:

$$\pi(\mathbf{x}^s) \propto \left(\frac{\lambda}{\mu}\right)^s \gamma^{\mathbf{x}^{s^T} \mathbf{A} \mathbf{x}^s / 2} \leq \left(\frac{\lambda}{\mu} \gamma^\beta\right)^s, \quad (10)$$

where  $\beta = k/2$ . Furthermore, the relationship (10) holds with equality for  $\mathbf{x}^0$  and  $\mathbf{x}^N$ .

**Theorem V.1:** For a  $k$ -regular graph, when  $0 \leq \lambda/\mu \leq 1, \gamma > 1$ , a threshold exists for the most probable network configuration,  $\mathbf{x}^*$ . Let

$$\beta = \frac{k}{2}.$$

Then

- 1)  $\lambda\gamma^\beta > \mu$  if and only if  $\mathbf{x}^*$  is unique and  $\mathbf{x}^* = \mathbf{x}^N$ .
- 2)  $\lambda\gamma^\beta < \mu$  if and only if  $\mathbf{x}^*$  is unique and  $\mathbf{x}^* = \mathbf{x}^0$ .
- 3)  $\lambda\gamma^\beta = \mu$  if and only if  $\mathbf{x}^*$  is no longer the unique maximizer, and  $\mathbf{x}^N$  and  $\mathbf{x}^0$  are both maximizers.

**Proof:**

- 1)  $\lambda\gamma^\beta > \mu$

**Sufficiency:** If  $\lambda\gamma^\beta > \mu$ , we show that  $\mathbf{x}^*$  is unique and  $\mathbf{x}^* = \mathbf{x}^N$ .

When  $(\lambda/\mu)\gamma^\beta > 1$ , the RHS of (10) is maximized when  $s = N$ . Since this is a growing exponential function, it is also the unique maximizer. By Lemma V.1,  $\mathbf{x}^* = \mathbf{x}^N$ , and the relationship in (10) holds with equality.

**Necessity:** If  $\mathbf{x}^*$  is unique and  $\mathbf{x}^* = \mathbf{x}^N$ , we show that  $\lambda\gamma^\beta > \mu$ .

Since  $\mathbf{x}^* = \mathbf{x}^N$ , it follows from Lemma V.1 that the bounding exponential function reaches a maximum at  $s = N$ , and this bound is met with equality. The RHS is monotonically increasing only when  $(\lambda/\mu)\gamma^\beta > 1$ .

- 2)  $\lambda\gamma^\beta < \mu$

**Sufficiency:** If  $\lambda\gamma^\beta < \mu$ , we show that  $\mathbf{x}^*$  is unique and  $\mathbf{x}^* = \mathbf{x}^0$ .

When  $(\lambda/\mu)\gamma^\beta < 1$ , the RHS of (10) is maximized when  $s = 0$ . Since this is a decaying exponential function, it is also the unique maximizer. By Lemma V.1,  $\mathbf{x}^* = \mathbf{x}^0$ , and the relationship in (10) holds with equality.

**Necessity:** If  $\mathbf{x}^*$  is unique and  $\mathbf{x}^* = \mathbf{x}^0$ , we show that  $\lambda\gamma^\beta < \mu$ .

Since  $\mathbf{x}^* = \mathbf{x}^0$ , it follows from Lemma V.1 that the bounding exponential function reaches a maximum at  $s = 0$  and this bound is met with equality. The RHS is monotonically decreasing only when  $(\lambda/\mu)\gamma^\beta < 1$ .

- 3)  $\lambda\gamma^\beta = \mu$

**Sufficiency:** If  $\lambda\gamma^\beta = \mu$ , we show that  $\mathbf{x}^*$  is no longer the unique maximizer, and  $\mathbf{x}^N$  and  $\mathbf{x}^0$  are both maximizers.

When  $(\lambda/\mu)\gamma^\beta = 1$ , the RHS of (10) is 1 regardless of  $s$ . We know that this is satisfied with equality for  $\mathbf{x}^0$  and  $\mathbf{x}^N$ . Therefore,  $\mathbf{x}^*$  is no longer the unique maximizer since  $\mathbf{x}^N$  and  $\mathbf{x}^0$  are both maximizers.

**Necessity:** If  $\mathbf{x}^*$  is no longer the unique maximizer, and  $\mathbf{x}^N$  and  $\mathbf{x}^0$  are both maximizers, we show that  $(\lambda/\mu)\gamma^\beta = 1$ .

For  $s = 0$ , the RHS of (10) equals to 1 and by Lemma V.1 this bound is met with equality. If  $\mathbf{x}^N$  and  $\mathbf{x}^0$  are both maximizers, this can only be achieved when  $(\lambda/\mu)\gamma^\beta = 1$ . ■

### B. Complete Bipartite Graph

Consider the complete bipartite graph,  $G(V, E) = K_{N_1, N_2}$ . We partition the state space,  $\mathcal{X}$ , as

$$\mathcal{X} = \mathcal{X}_{0,0} \cup \mathcal{X}_{1,0} \cup \mathcal{X}_{0,1} \cup \mathcal{X}_{1,1} \cup \dots \mathcal{X}_{N_1, N_2},$$

where

$$\mathcal{X}_{s_1, s_2} = \{\mathbf{x} \in \mathcal{X} \mid s_1 \text{ infectives in } V_1, s_2 \text{ infectives in } V_2\}. \quad (11)$$

Denote a configuration belonging to a partition as

$$\mathbf{x}^{s_1, s_2} \in \mathcal{X}_{s_1, s_2}.$$

*Lemma V.2 (Proof in Appendix B):* For the complete bipartite graph, all the network configurations,  $\mathbf{x}^{s_1, s_2}$ , belonging to the same partition,  $\mathcal{X}_{s_1, s_2}$ , have the same equilibrium probability.

*Lemma V.3 (Proof in Appendix C):* When  $0 \leq \lambda/\mu \leq 1, \gamma > 1$  and for  $s_1 = 0, 1, 2, \dots, N_1, s_2 = 0, 1, 2, \dots, N_2$ , the unnormalized equilibrium distribution for a complete bipartite graph is upperbounded by an exponential function:

$$\pi(\mathbf{x}^{s_1, s_2}) \propto \left(\frac{\lambda}{\mu}\right)^{s_1 + s_2} \gamma^{s_1 s_2} \leq \left(\frac{\lambda}{\mu} \gamma^\beta\right)^{s_1 + s_2}, \quad (12)$$

where  $\beta = N_1 N_2 / (N_1 + N_2)$ . Furthermore, the relationship holds with equality for  $\mathbf{x}^{s_1, s_2} = \mathbf{x}^N$  and  $\mathbf{x}^{s_1, s_2} = \mathbf{x}^0$ .

*Theorem V.2:* For a complete bipartite graph, when  $0 \leq \lambda/\mu \leq 1, \gamma > 1$ , a threshold exists for the most probable network configuration. Let

$$\beta = \frac{N_1 N_2}{N_1 + N_2}.$$

Then

- 1)  $\lambda\gamma^\beta > \mu$  if and only if  $\mathbf{x}^*$  is unique and  $\mathbf{x}^* = \mathbf{x}^N$ .
- 2)  $\lambda\gamma^\beta < \mu$  if and only if  $\mathbf{x}^*$  is unique and  $\mathbf{x}^* = \mathbf{x}^0$ .
- 3)  $\lambda\gamma^\beta = \mu$  if and only if  $\mathbf{x}^*$  is no longer the unique maximizer;  $\mathbf{x}^* = \mathbf{x}^N, \mathbf{x}^* = \mathbf{x}^0$ .

*Proof:* The proof follows that of Theorem V.1. Note that when  $N_1 = N_2$ , the complete bipartite graph is also a  $k$ -regular graph, and  $k = N_1$ ,

$$\beta = \frac{N_1 N_1}{N_1 + N_1} = \frac{N_1}{2}.$$

### C. Complete Multipartite Graph

Consider the complete multipartite graph,  $G(V, E) = K_{N_1, N_2, \dots, N_m}$ , where the intra-island degree,  $k^{\text{intra}} = 0$  for all nodes.

We partition the state space,  $\mathcal{X}$ , as

$$\mathcal{X} = \mathcal{X}_{0,0,\dots,0} \cup \mathcal{X}_{1,0,\dots,0} \cup \mathcal{X}_{2,0,\dots,0} \cup \dots \cup \mathcal{X}_{N_1, N_2, \dots, N_m}, \quad (13)$$

where  $\mathcal{X}_{s_1, s_2, \dots, s_m}$  is the set of network configurations where there are  $s_1$  infected nodes in island 1,  $s_2$  infected nodes in island 2, and so forth.

$$\mathbf{x}^{s_1, s_2, \dots, s_m} \in \mathcal{X}_{s_1, s_2, \dots, s_m}. \quad (14)$$

For brevity, we state without proof the generalized version of Lemma V.2. For the complete multipartite graph, all the configurations,  $\mathbf{x}^{s_1, s_2, \dots, s_m}$ , belonging to the same partition,  $\mathcal{X}_{s_1, s_2, \dots, s_m}$ , have the same equilibrium probability.

*Lemma V.4 (Proof in Appendix D):* When  $0 \leq \lambda/\mu \leq 1, \gamma > 1$ , and for  $s_1 = 0, 1, 2, \dots, N_1, s_2 = 0, 1, 2, \dots, N_2, \dots, s_m = 0, 1, 2, \dots, N_m$ , the unnormalized equilibrium distribution for a complete multipartite graph is upperbounded by an exponential function:

$$\begin{aligned} \pi(\mathbf{x}^{s_1, s_2, \dots, s_m}) &\propto \left(\frac{\lambda}{\mu}\right)^{\sum_{p=1}^m s_p} \gamma^{(\mathbf{x}^{s_1, s_2, \dots, s_m})^T A \mathbf{x}^{s_1, s_2, \dots, s_m} / 2} \\ &\leq \left(\frac{\lambda}{\mu} \gamma^\beta\right)^{\sum_{p=1}^m s_p}, \end{aligned} \quad (15)$$

where

$$\beta = \frac{\sum_{i=1}^m N_i \left( \sum_{j \neq i} \mathbb{1}(V_i \sim V_j) N_j \right)}{2 \sum_{p=1}^m N_p}.$$

Furthermore, the inequality holds with equality for  $\mathbf{x}^{s_1, s_2, \dots, s_m} = \mathbf{x}^N$  and  $\mathbf{x}^{s_1, s_2, \dots, s_m} = \mathbf{x}^0$ .

*Theorem V.3:* For the complete multipartite graph, when  $0 \leq \lambda/\mu \leq 1, \gamma > 1$ , a threshold exists for the most probable network configuration. Let

$$\beta = \frac{\sum_{i=1}^m N_i \left( \sum_{j \neq i} \mathbb{1}(V_i \sim V_j) N_j \right)}{2 \sum_{p=1}^m N_p}.$$

Then

- 1)  $\lambda\gamma^\beta > \mu$  if and only if  $\mathbf{x}^*$  is unique and  $\mathbf{x}^* = \mathbf{x}^N$ .
- 2)  $\lambda\gamma^\beta < \mu$  if and only if  $\mathbf{x}^*$  is unique and  $\mathbf{x}^* = \mathbf{x}^0$ .
- 3)  $\lambda\gamma^\beta = \mu$  if and only if  $\mathbf{x}^*$  is no longer the unique maximizer;  $\mathbf{x}^* = \mathbf{x}^N, \mathbf{x}^* = \mathbf{x}^0$ .

*Proof:* The proof follows that of Theorem V.1. Note that the complete bipartite graph is a special case of the complete multipartite graph where  $m = 2$  and  $\sum_{j \neq 1} \mathbb{1}(V_1 \sim V_j) N_j = N_2$  and  $\sum_{j \neq 2} \mathbb{1}(V_2 \sim V_j) N_j = N_1$ . ■

### D. Complete Multipartite Graph With $k$ -Regular Supernodes

Consider the complete multipartite graph with  $k$ -regular supernodes,  $G(V, E)$ , where the intra-island degree,  $0 < k^{\text{intra}} \leq N_i, \forall i = 1, \dots, m$ . We adopt the same notation as in Section V-C.

*Lemma V.5 (Proof in Appendix E):* When  $0 \leq \lambda/\mu \leq 1, \gamma > 1$ , and for  $s_1 = 0, 1, 2, \dots, N_1, s_2 = 0, 1, 2, \dots, N_2, \dots, s_m = 0, 1, 2, \dots, N_m$ , the unnormalized equilibrium distribution for the complete multipartite graph with regular islands is upperbounded by an exponential function:

$$\begin{aligned} \pi(\mathbf{x}^{s_1, s_2, \dots, s_m}) &\propto \left(\frac{\lambda}{\mu}\right)^{\sum_{p=1}^m s_p} \gamma^{(\mathbf{x}^{s_1, s_2, \dots, s_m})^T A \mathbf{x}^{s_1, s_2, \dots, s_m} / 2} \\ &\leq \left(\frac{\lambda}{\mu} \gamma^\beta\right)^{\sum_{p=1}^m s_p}, \end{aligned} \quad (16)$$

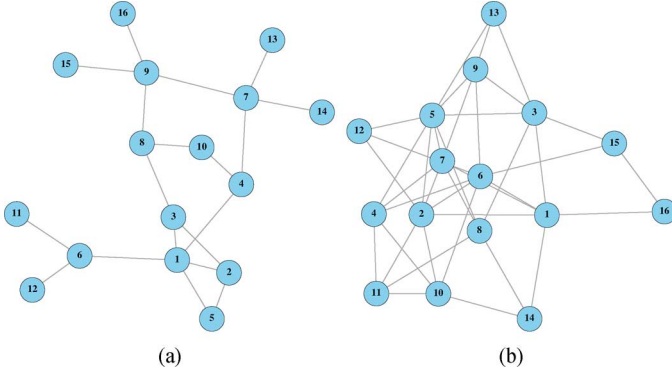


Fig. 2. Example Network Topologies. (a) Graph 1. (b) Graph 2.

where

$$\beta = \sum_{i=1}^m \frac{k_i^{\text{intra}}}{2} + N_i \left( \frac{\sum_{j \neq i} \mathbb{1}(V_i \sim V_j) N_j}{2 \sum_{p=1}^m N_p} \right).$$

Furthermore, the relationship holds with equality for  $\mathbf{x}^{s_1, s_2, \dots, s_m} = \mathbf{n}^N$  and  $\mathbf{x}^{s_1, s_2, \dots, s_m} = \mathbf{x}^0$ .

**Theorem V.4:** For the complete multipartite graph with  $k$ -regular islands, when  $0 \leq \lambda/\mu \leq 1, \gamma > 1$ , a threshold exists for the most probable network configuration. Let

$$\beta = \sum_{i=1}^m \frac{k_i^{\text{intra}}}{2} + N_i \left( \frac{\sum_{j \neq i} \mathbb{1}(V_i \sim V_j) N_j}{2 \sum_{p=1}^m N_p} \right).$$

Then

- 1)  $\lambda\gamma^\beta > \mu$  if and only if  $\mathbf{x}^*$  is unique and  $\mathbf{x}^* = \mathbf{x}^N$ .
- 2)  $\lambda\gamma^\beta < \mu$  if and only if  $\mathbf{x}^*$  is unique and  $\mathbf{x}^* = \mathbf{x}^0$ .
- 3)  $\lambda\gamma^\beta = \mu$  if and only if  $\mathbf{x}^*$  is no longer the unique maximizer;  $\mathbf{n}\mathbf{x}^* = \mathbf{x}^N$ ,  $\mathbf{x}^* = \mathbf{x}^0$ .

*Proof:* The proof follows that of Theorem V.1. ■

#### E. General Graph Topology

Unlike the more structured network topologies (e.g.,  $k$ -regular graph, complete multipartite graph, complete multipartite graph with regular islands) considered in the previous sections, we can not bound the equilibrium distribution (7) of the process  $X(t)$  by a monotonic function for other topologies. Therefore,  $\mathbf{x}^*$  may be configurations other than  $\mathbf{x}^0$  or  $\mathbf{x}^N$ . For example, consider Graph 1 shown in Fig. 2(a) and Graph 2 shown in Fig. 2(b). Graph 1 is a 16 node Erdős-Rényi random graph, while Graph 2 is a 16 node Watts-Strogatz graph [1].

If we hold the  $\lambda$  and  $\mu$  parameters constant, but vary the parameter  $\gamma$ , then the most probable configuration changes. Fig. 3 shows the resultant  $\mathbf{x}^*$  for different parameter values. We set  $\lambda = 1, \mu = 2$  while varying  $\gamma$  from 1 to 4 with incremental step sizes as shown on the X-axis. On the Y-axis, we plot the total number of infectives in  $\mathbf{x}^*$ . When the number of infectives is 0,  $\mathbf{x}^* = \mathbf{x}^0$ ; when the number of infectives is 16,  $\mathbf{x}^* = \mathbf{x}^N$ . As  $\gamma$  increases,  $\mathbf{x}^*$  switches from  $\mathbf{x}^0$  to  $\mathbf{x}^N$  as per our intuition. Fig. 4 shows the resultant  $\mathbf{x}^*$  plot for Graph 2 with  $\lambda = 1, \mu = 2$ , and  $\gamma$  going from 1 to 3.

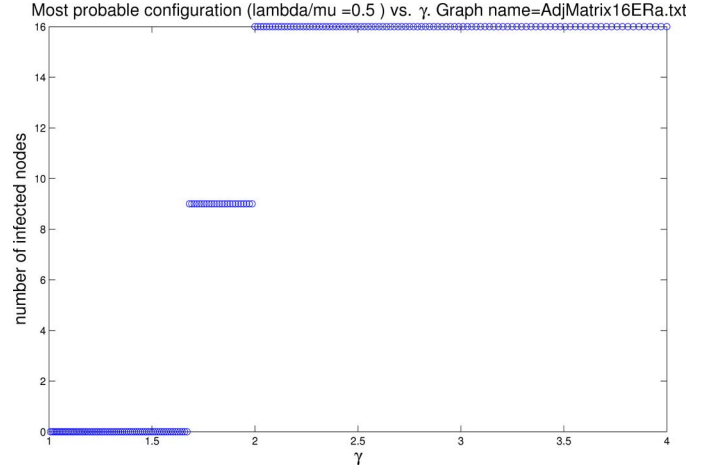


Fig. 3. Most Probable Configuration  $\mathbf{x}^*$  for Graph 1.

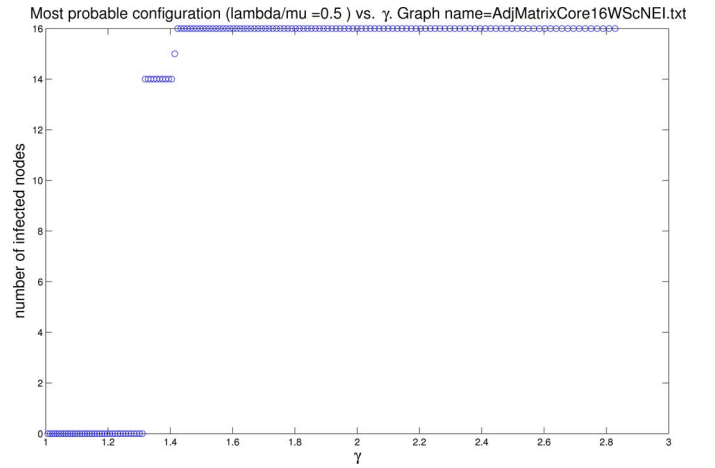


Fig. 4. Most Probable Configuration  $\mathbf{x}^*$  for Graph 2.

For a small range of  $\gamma$  values the number of infectives in  $\mathbf{x}^*$  is neither 0 nor 16. In Graph 1, it is 9, while for Graph 2, it can be 14 or 15, but not 16. This is indicative of the existence of solutions to (9) which are not  $\mathbf{x}^0$  or  $\mathbf{x}^N$ . We call these solutions the *spurious most probable configuration*. Fig. 5(a) shows the spurious configurations for Graph 1 where 9 agents are infected but 7 agents remain healthy. Fig. 5(b) and (c) show these spurious configurations for Graph 2.

We summarize the following observations regarding these spurious most probable network configurations.

- 1) Not all unstructured topologies exhibit these spurious  $\mathbf{x}^*$ .
- 2) Spurious  $\mathbf{x}^*$  occur as transitional configurations as  $\mathbf{x}^*$  goes from  $\mathbf{x}^0$  to  $\mathbf{x}^N$ .
- 3) For the same set of parameter values, spurious  $\mathbf{x}^*$  may be a set of equally probable configurations.

#### VI. REGIME III) EXOGENOUS INFECTION DOMINANT:

$$\lambda/\mu > 1, 0 < \gamma \leq 1$$

In this regime, the topology independent infection rate,  $\lambda$ , is larger than the topology independent healing rate,  $\mu$ . If we did not account for the topology dependent infection process through the population (i.e., if  $\gamma = 1$ ), then the most probable



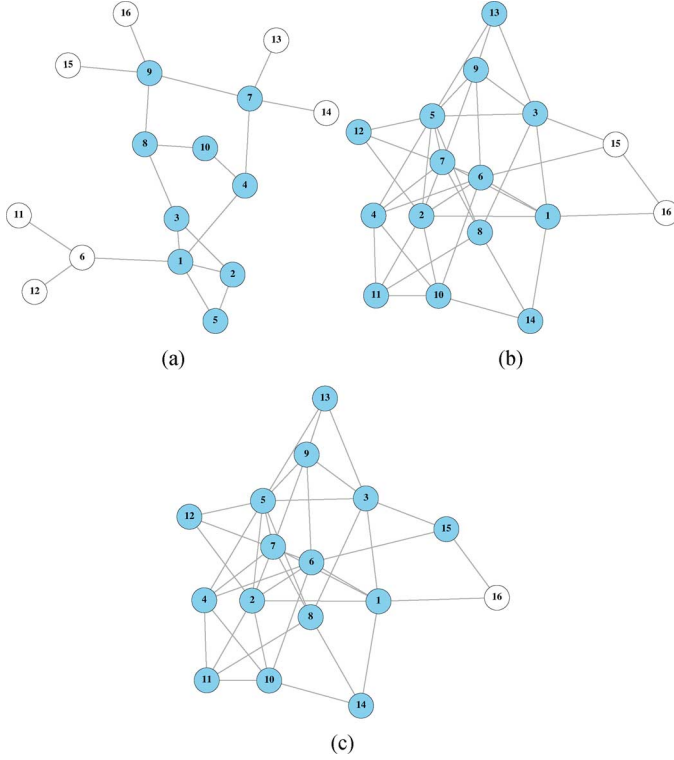


Fig. 5. Spurious  $\mathbf{x}^*$  (Blue (Gray) = Infectives, White = Susceptibles). (a) Spurious  $\mathbf{x}^*$  for Graph 1 with 9 infectives. (b) Spurious  $\mathbf{x}^*$  for Graph 2 with 14 infectives. (c) Spurious  $\mathbf{x}^*$  for Graph 2 with 15 infectives.

network configuration would be  $\mathbf{x}^* = \mathbf{x}^N = [1, 1, \dots, 1]^T$ . In this scenario, agents individually prefer to be spreaders instead of adopters.

However, since  $0 < \gamma \leq 1$ , additional infected agents will decrease the topology dependent infection rate; in the social contagion context, peer influence actually inhibits the conversion of adopters to spreaders. To maximize the probability in (9), we seek network configurations that maximize the number of infected nodes while minimizing the number of infected edges; unlike the most probable network configuration in regime II, infected nodes are likely to be separated from one another. We will show that the graph theoretic concept of *independent sets* plays a major role in this regime. An independent set is a subset of nodes such that the induced subgraph is composed entirely of isolated nodes. The maximum independent set is the largest possible independent set for a given graph [20]. The maximum independent set is also the largest maximal independent set (i.e., an independent set which is not a subset of any other independent set).

Intuitively, if  $\lambda/\mu$  is large and  $\gamma$  is close to 1, then the epidemics will be dictated by the topology independent process; a good guess for  $\mathbf{x}^*$  is  $\mathbf{x}^N = [1, 1, \dots, 1]^T$ . When  $\gamma$  is small and  $\lambda/\mu$  is close to 1, the epidemic behavior will be dictated by the topology dependent process. But unlike regime II, where  $0 < \lambda/\mu \leq 1, \gamma > 1$ , the topology dependent process can not overcome the topology independent process; we will prove that  $\mathbf{x}^*$  can never be  $\mathbf{x}^0 = [0, 0, \dots, 0]^T$  for any network topology.

**Theorem VI.1:** For any network topology, when  $\lambda/\mu > 1, 0 < \gamma \leq 1, \mathbf{x}^* \neq \mathbf{x}^0 = [0, 0, \dots, 0]^T$  for any feasible parameter values.

*Proof:* We prove this by contradiction. Suppose that there is a network topology for which  $\mathbf{x}^* = \mathbf{x}^0$ . Therefore,

$$\pi(\mathbf{x}^0) = \frac{1}{Z} > \pi(\mathbf{x}), \quad \forall \mathbf{x} \in \mathcal{X} \setminus \mathbf{x}^0.$$

We know that, for any network configuration with 1 infected agent,  $\mathbf{x}^1$ , the equilibrium is  $\pi(\mathbf{x}^1) = (1/Z)(\lambda/\mu)$  regardless of network topology. Since  $\lambda/\mu > 1$ ,

$$\pi(\mathbf{x}^1) = \frac{1}{Z} \left( \frac{\lambda}{\mu} \right) > \frac{1}{Z} = \pi(\mathbf{x}^0),$$

which contradicts the premise that  $\mathbf{x}^* = \mathbf{x}^0$ . ■

This theorem shows how regime III) Exogenous Infection Dominant behaves differently from regimes II) Endogenous Infection Dominant. In regime II, since  $0 < \lambda/\mu \leq 1$ , without the effects of the topology dependent process, the most probable configuration  $\mathbf{x}^* = \mathbf{x}^0$ . However, since  $\gamma > 1$ , regardless of network topology, we can find a threshold value for  $\gamma$  such that  $\mathbf{x}^*$  switches from  $\mathbf{x}^0$  to  $\mathbf{x}^N$ ; in the most probable configuration, if  $\gamma$  is larger than this threshold, then the endogenous infection process  $\gamma$  is capable of spreading the virus throughout the network.

In regime III however, as Theorem VI.1 states, no matter how small we set  $\gamma$  is, there is no way to drive  $\mathbf{x}^*$  from  $\mathbf{x}^N$  to  $\mathbf{x}^0$  for any network topology; in the most probable configuration, the endogenous infection process is incapable of eliminating the contagion from the network.

**Theorem VI.2:** For any network topology and when  $\lambda/\mu > 1, 0 < \gamma \leq 1$ , sort the degrees so that  $k_1 \geq k_2 \geq \dots \geq k_N$ , if  $\lambda\gamma^{k_1} > \mu$ , then  $\mathbf{x}^* = \mathbf{x}^N = [1, 1, \dots, 1]^T$ .

*Proof:* Suppose that there is a network topology for which  $\mathbf{x}^* = \mathbf{x}', \mathbf{x}' \neq \mathbf{x}^N$ . This means that  $\pi(\mathbf{x}') > \pi(\mathbf{x}^N)$ .

What are possible  $\mathbf{x}'$ ? Since  $\lambda/\mu > 1, 0 < \gamma \leq 1$ , we should consider configurations which either have more infected nodes or less infected edges than  $\mathbf{x}^N$ . However, only the latter option is possible. In addition, having less than  $|E|$  infected edges means that we need to reduce the number of infected nodes in the configuration as well. Consider

$$\mathcal{X}' = \left\{ \mathbf{x} \in \mathcal{X} \mid 1^T \mathbf{x} = N - 1, \frac{\mathbf{x}^T A \mathbf{x}}{2} = |E| - k_1 \right\}.$$

Note that since  $1 \leq (\lambda/\mu)\gamma^{k_1} \leq (\lambda/\mu)\gamma^{k_2} \leq \dots \leq (\lambda/\mu)\gamma^{k_N}$ ,  $\pi(\mathbf{x}') \geq \pi(\mathbf{x}), \forall \mathbf{x} \in \mathcal{X} \setminus \mathbf{x}^N$ .

Realize that

$$\frac{\pi(\mathbf{x}^N)}{\pi(\mathbf{x}')} = \frac{\left(\frac{\lambda}{\mu}\right)^N \gamma^{|E|}}{\left(\frac{\lambda}{\mu}\right)^{N-1} \gamma^{|E|-k_1}} = \frac{\lambda}{\mu} \gamma^{k_1} > 1.$$

This implies that  $\pi(\mathbf{x}^N) > \pi(\mathbf{x}')$  which contradicts the premise that  $\mathbf{x}^* = \mathbf{x}', \mathbf{x}' \neq \mathbf{x}^N$ . ■

**Theorem VI.3:** For any network topology and when  $\lambda/\mu > 1, 0 < \gamma \leq 1$ , if in addition  $\lambda\gamma < \mu$ ,  $\mathbf{x}^*$  are the network

configuration(s) with maximum number of infected nodes and 0 infected edges.

*Proof:* Let

$$\mathcal{X}' = \{\mathbf{x} \in \mathcal{X} \mid \mathbf{x}^T \mathbf{A} \mathbf{x} = 0, \text{ and } 1^T \mathbf{x} \text{ is maximum}\}. \quad (17)$$

The set  $\mathcal{X}'$  is also known as the maximum independent set [20]. Since  $\lambda/\mu > 1$ , we know that

$$\pi(\mathbf{x}') = \frac{1}{Z} \left( \frac{\lambda}{\mu} \right)^{1^T \mathbf{x}'} \geq \pi(\mathbf{x}), \quad \forall \mathbf{x} \in \{\mathbf{x} \in \mathcal{X} \mid \mathbf{x}^T \mathbf{A} \mathbf{x} = 0\}. \quad (18)$$

Consider  $\mathbf{x}' \in \mathcal{X}'$  and  $\mathbf{x} \notin \mathcal{X}'$ . Let  $\bar{\mathcal{X}}_s$  represent the set of configurations in  $\mathcal{X} \setminus \mathcal{X}'$  with  $s$  infected nodes.

- 1) If  $1^T \mathbf{x}' \geq 1^T \mathbf{x}$ , then since  $\lambda/\mu > 1, 0 < \gamma \leq 1$ , we know that  $\pi(\mathbf{x}') > \pi(\mathbf{x})$ .
- 2) If  $1^T \mathbf{x}' = m < 1^T \mathbf{x}$ , then we know that  $\mathbf{x}^T \mathbf{A} \mathbf{x} / 2 \geq 1$  by our definition of  $\mathcal{X}'$ . Consider the sets  $\bar{\mathcal{X}}_{m+k}$  and  $\bar{\mathcal{X}}_{m+k+1}$  where  $1 \leq k \leq N - m - 1$ . Suppose that all the configurations in  $\bar{\mathcal{X}}_{m+k}$  have at least  $q \geq 1$  edges. Condition (17) also implies that each additional infected node will result in at least one additional infected edge; therefore all the configurations in  $\bar{\mathcal{X}}_{m+k+1}$  will have at least  $q + 1$  infected edges.

Since  $0 < \gamma \leq 1$ , the configuration(s) with the maximum equilibrium probability in  $\bar{\mathcal{X}}_{m+k}$  should have the fewest number of infected edges amongst all the configurations in  $\bar{\mathcal{X}}_{m+k}$ , similarly for  $\bar{\mathcal{X}}_{m+k+1}$ .

$$\begin{aligned} \max_{\mathbf{x} \in \bar{\mathcal{X}}_{m+k}} \pi(\mathbf{x}) &= \frac{1}{Z} \left( \frac{\lambda}{\mu} \right)^{m+k} \gamma^q, \\ \text{and} \\ \max_{\mathbf{x} \in \bar{\mathcal{X}}_{m+k+1}} \pi(\mathbf{x}) &= \frac{1}{Z} \left( \frac{\lambda}{\mu} \right)^{m+k+1} \gamma^{q+1}. \end{aligned} \quad (19)$$

With  $\lambda\gamma < \mu$ , we see that

$$\max_{\mathbf{x} \in \bar{\mathcal{X}}_{m+k}} \pi(\mathbf{x}) > \max_{\mathbf{x} \in \bar{\mathcal{X}}_{m+k+1}} \pi(\mathbf{x}). \quad (19)$$

Equation (19) implies that the configuration with the largest equilibrium probability in  $\{\bar{\mathcal{X}}_s \mid s = m+1, m+2, \dots, N\}$  is the configuration with  $m+1$  infected nodes and 1 infected edge:

$$\max_{\mathbf{x} \in \{\bar{\mathcal{X}}_s \mid s=m+1, m+2, \dots, N\}} \pi(\mathbf{x}) = \frac{1}{Z} \left( \frac{\lambda}{\mu} \right)^{m+1} \gamma.$$

With  $\lambda\gamma < \mu$ ,  $\pi(\mathbf{x}') > \max_{\mathbf{x} \in \bar{\mathcal{X}}_{m+1}} \pi(\mathbf{x})$ .

Since we showed for both cases 1) and 2), we conclude that  $\mathbf{x}^* = \mathbf{x}'$ . ■

*Corollary VI.4 (Proof in Appendix F):* For the complete graph,  $K_N$ , when  $\lambda/\mu > 1, 0 < \gamma \leq 1$ , if in addition  $\lambda\gamma < \mu$ ,  $\mathbf{x}^* \in \{\mathbf{x} \in \mathcal{X} \mid 1^T \mathbf{x} = 1\}$ .

*Corollary VI.5 (Proof in Appendix G):* For the bipartite graph, when  $\lambda/\mu > 1, 0 < \gamma \leq 1$ , if in addition  $\lambda\gamma < \mu$ ,  $\mathbf{x}^* \in \{\mathbf{x} \in \mathcal{X} \mid 1^T \mathbf{x} = \max\{N_1, N_2\}, \mathbf{x}^T \mathbf{A} \mathbf{x} = 0\}$ .

1) *Maximum Independent Set Problem:* In regime III, theorem VI.3 shows that in the parameter space,

$\lambda/\mu > 1, 0 < \gamma \leq 1$ , and  $\lambda\gamma < \mu$ , the most probable configuration  $\mathbf{x}^*$  for the scaled SIS process in equilibrium is the maximum independent set. It is known that the maximum independent set problem, in addition to other graph theoretic problems such as minimum vertex cover, maximum matching, is NP-hard for general graph topologies [20]. However, polynomial algorithms do exist for a special class of graphs called perfect graphs [21]. Well-known examples of perfect graphs are: complete graphs, bipartite graphs, and chordal graphs. We do not pursue this further here since it requires more in depth discussion of graph theory concepts.

## VII. PROBABILITY OF THE MOST PROBABLE NETWORK CONFIGURATION

Thus far, we focused on the most probable configuration,  $\mathbf{x}^*$ , which solves the optimization problem (9). In this section, we analyze how  $\pi(\mathbf{x}^*)$ , the maximum probability of the equilibrium distribution, varies with parameter values.

*Lemma VII.1 (Proof in Appendix H):* Consider two non-negative vectors  $\mathbf{x} = [x_1, \dots, x_n]$  and  $\mathbf{y} = [y_1, \dots, y_n]$ . We use  $\mathbf{x}^\delta = [x_1^\delta, \dots, x_n^\delta]$  and  $\mathbf{y}^\zeta = [y_1^\zeta, \dots, y_n^\zeta]$  to denote entrywise power. We denote entrywise product as  $\mathbf{x} \circ \mathbf{y} = [x_1 y_1, \dots, x_n y_n]$ . If  $\delta > 1$  and  $\zeta > 1$ , then

$$\left\| \frac{\mathbf{x}^\delta \circ \mathbf{y}^\zeta}{(\mathbf{x}^\delta)^T (\mathbf{y}^\zeta)} \right\|_\infty \geq \left\| \frac{\mathbf{x} \circ \mathbf{y}}{\mathbf{x}^T \mathbf{y}} \right\|_\infty.$$

*Theorem VII.1:* For any network topology  $G(V, E)$ , consider a scaled SIS process  $X(t)$  over the network and an alternate scaled SIS process  $X'(t)$  over the same network. Process  $X(t)$  is parameterized by  $(\lambda, \mu, \gamma)$  and process  $X'(t)$  parameterized by  $(\lambda', \mu', \gamma')$  with corresponding equilibrium distributions  $\pi(\mathbf{x})$  and  $\pi(\mathbf{x}')$  and most probable network configurations,  $\mathbf{x}^*$  and  $\mathbf{x}'^*$ .

- **I) Healing dominant:**  $0 < \lambda/\mu \leq 1, 0 < \gamma \leq 1$ . If  $\lambda'/\mu' < \lambda/\mu$  and/or  $\gamma' < \gamma$ , then  $\pi(\mathbf{x}'^*) \geq \pi(\mathbf{x}^*)$ .
- **II) Endogenous infection dominant:**  $0 < \lambda/\mu \leq 1, \gamma > 1$ . If  $\lambda'/\mu' < \lambda/\mu$  and/or  $\gamma' > \gamma$ , then  $\pi(\mathbf{x}'^*) \geq \pi(\mathbf{x}^*)$ .
- **III) Exogenous infection dominant:**  $\lambda/\mu > 1, 0 < \gamma \leq 1$ . If  $\lambda'/\mu' > \lambda/\mu$  and/or  $\gamma' < \gamma$ , then  $\pi(\mathbf{x}'^*) \geq \pi(\mathbf{x}^*)$ .
- **IV) Infection dominant:**  $\lambda/\mu > 1, \gamma > 1$ . If  $\lambda'/\mu' > \lambda/\mu$  and/or  $\gamma' > \gamma$ , then  $\pi(\mathbf{x}'^*) \geq \pi(\mathbf{x}^*)$ .

*Proof:* We will prove Theorem VII.1 for regime IV; the other 3 regimes uses similar arguments. We want to show that when  $\lambda/\mu > 1, \gamma > 1$ , if  $\lambda'/\mu' > \lambda/\mu$  and/or  $\gamma' > \gamma$ , then  $\pi(\mathbf{x}'^*) \geq \pi(\mathbf{x}^*)$ .

We can express the parameters of  $X'(t)$  as scaled versions of the parameters of  $X(t)$  where  $\lambda' = \lambda^\delta, \mu' = \mu^\delta, \gamma' = \gamma^\zeta$ . Depending on if the parameters in  $X'(t)$  are larger, or smaller or equal than the parameters of  $X(t)$ , we can set  $\delta > 1$  or  $0 < \delta \leq 1$ , and likewise for  $\zeta$ .

Next, take the logs of all the parameters so that  $\bar{\lambda} = \log_2 \lambda$ ,  $\bar{\mu} = \log_2 \mu$ ,  $\bar{\gamma} = \log_2 \gamma$ ,  $\bar{\lambda}' = \delta \bar{\lambda}$ ,  $\bar{\mu}' = \delta \bar{\mu}$ ,  $\bar{\gamma}' = \zeta \bar{\gamma}$ .

Rewriting the equilibrium distribution (7) for  $X(t)$  using the log parameters, we get

$$\pi(\mathbf{x}) = \frac{1}{Z} 2^{(\bar{\lambda} - \bar{\mu}) 1^T \mathbf{x} + \bar{\gamma} \mathbf{x}^T \mathbf{A} \mathbf{x} / 2}, \quad \mathbf{x} \in \mathcal{X} \quad (20)$$

and for  $X'(t)$  as

$$\pi'(\mathbf{x}) = \frac{1}{Z'} 2^{\delta(\bar{\lambda}-\bar{\mu})1^T \mathbf{x} + \delta \bar{\gamma} \mathbf{x}^T A \mathbf{x}/2}, \quad \mathbf{x} \in \mathcal{X}. \quad (21)$$

It's easier to see the relationship between  $\pi(\mathbf{x})$  and  $\pi'(\mathbf{x})$  when we rewrite  $\pi'(\mathbf{x})$  as

$$\pi'(\mathbf{x}) = \frac{1}{Z'} \left( 2^{(\bar{\lambda}-\bar{\mu})1^T \mathbf{x}} \right)^\delta \left( 2^{\bar{\gamma} \mathbf{x}^T A \mathbf{x}/2} \right)^\zeta, \quad \mathbf{x} \in \mathcal{X}.$$

Since the equilibrium is a PMF, there is a bijective mapping from  $\pi(\mathbf{x})$  to the finite length vector  $\mathbf{x} \circ \mathbf{y}/\mathbf{x}^T \mathbf{y}$  and from  $\pi'(\mathbf{x})$  to the vector  $\mathbf{x}^\delta \circ \mathbf{y}^\zeta/(\mathbf{x}^\delta)^T (\mathbf{y}^\zeta)$ . Using Lemma VII.1, we conclude that

$$\pi(\mathbf{x}'^*) = \max_{\mathbf{x}' \in \mathcal{X}} \pi(\mathbf{x}') \geq \pi(\mathbf{x}^*) = \max_{\mathbf{x} \in \mathcal{X}} \pi(\mathbf{x})$$

■

## VIII. CONCLUSION

We introduced the scaled SIS process for modeling the diffusion of ideals/opinions/beliefs/innovations in a population whose relationships and influences can be characterized by a static, undirected network. We showed that, under specific rate assumptions, the scaled SIS process is a reversible continuous-time Markov process, for which we determined analytically its equilibrium distribution. This equilibrium distribution exhibits explicitly, through the adjacency matrix defining the local interaction among agents, the impact of the topology.

We then derived the most probable configuration, the configuration of jointly held opinions with the maximum equilibrium probability. Interpreting the equilibrium distribution as the product of a topology dependent process (i.e., a term that includes the adjacency matrix of the network) and a topology independent process allowed us to breakdown the solution space into 4 regimes. The two most interesting regimes reflect an opposing process that counters the diffusion of ideas in the population.

The rest of the paper focused on analyzing these two regimes. In regime II) Endogenous Infection Dominant, as the number of spreaders increase, the diffusion rate increases thereby increasing the number of spreaders further. However the diffusion process is tempered by the innate preference of each agent to be an adopter instead of a spreader. For structured network topologies (i.e.,  $k$ -regular, complete multipartite, and complete multipartite with  $k$ -regular islands graphs), we derived the threshold below which all agents are adopters and above which all agents are spreaders. For other network topologies, the most probable configuration may be a combination of adopters and spreaders. We call these solutions the spurious configurations.

In regime III) Exogenous Infection Dominant, increasing the number of neighbor spreaders decreases the diffusion rate; this may be the case in populations where agents consciously choose to act in the opposite fashion as their neighbors. However, in this regime, the innate preference of each agent is to be a spreader

instead of an adopter. We showed that the most probable configuration also exhibits phase transition behavior though it is more complicated than in regime II. In regime III, the most probable configuration can never be the configuration where all the agents are adopters. We also proved that, in a range of parameter values, the most probable configuration corresponds to the solution of the maximum independent set problem.

In the future, we will further explore how network topology affects the existence of spurious configurations, and how agents corresponding to the maximum independent set are 'special' through the role they may play in the diffusion process. We will also study the relationships between the equilibrium distribution and the spectral characteristics of the underlying network. We will fit empirical data to this model and estimate the parameter and/or network topology from full or partial observations of the diffusion process.

## APPENDIX A

### PROOF OF LEMMA V.1

*Lemma A.1:* When  $0 \leq \lambda/\mu \leq 1, \gamma > 1$  and for  $s = 0, 1, \dots, N, \mathbf{x}^s \in \mathcal{X}_s$ , the unnormalized equilibrium distribution for a  $k$ -regular graph is upperbounded by an exponential function:

$$\pi(\mathbf{x}^s) \propto \left( \frac{\lambda}{\mu} \right)^s \gamma^{\mathbf{x}^{sT} A \mathbf{x}^s/2} \leq \left( \frac{\lambda}{\mu} \gamma^\beta \right)^s, \quad (22)$$

where  $\beta = k/2$ . Furthermore, the relationship holds with equality for  $\mathbf{x}^0$  and  $\mathbf{x}^N$ .

*Proof:* For the  $k$ -regular graph

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

and

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

Then

$$\begin{aligned} \mathbf{x}^{sT} A \mathbf{x}^s &\leq sk, \text{ if } k \leq s \\ \mathbf{x}^{sT} A \mathbf{x}^s &\leq s^2 \leq sk, \text{ if } k \geq s. \end{aligned} \quad (23)$$

With  $\gamma > 1$  and (23), we deduce that

$$\left( \frac{\lambda}{\mu} \right)^s \gamma^{\mathbf{x}^{sT} A \mathbf{x}^s/2} \leq \left( \frac{\lambda}{\mu} \right)^s \gamma^{sk/2}, \quad \forall s = 0, 1, \dots, N. \quad (24)$$

When  $s = 0$ , equality is satisfied in (24). When  $s = N$ ,  $\mathbf{x}^{sT} A \mathbf{x}^s/2 = kN/2$ ; hence equality is also satisfied. ■

### APPENDIX B PROOF OF LEMMA V.2

*Lemma B.1:* For the complete bipartite graph, if  $\mathbf{x}_1^{s_1, s_2}, \mathbf{x}_2^{s_1, s_2} \in \mathcal{X}_{s_1, s_2}$ , then  $\pi(\mathbf{x}_1^{s_1, s_2}) = \pi(\mathbf{x}_2^{s_1, s_2})$ .

*Proof:* By definition (11),  $1^T \mathbf{x}_1^{s_1, s_2} = s_1 + s_2$  and  $1^T \mathbf{x}_2^{s_1, s_2} = s_1 + s_2$ . Furthermore, if the underlying topology is a complete bipartite graph, we know that  $(\mathbf{x}_1^{s_1, s_2})^T A \mathbf{x}_1^{s_1, s_2} = s_1 s_2$  and  $(\mathbf{x}_2^{s_1, s_2})^T A \mathbf{x}_2^{s_1, s_2} = s_1 s_2$ . Since the number of infected nodes and the number of infected edges are the same, then  $\pi(\mathbf{x}_1^{s_1, s_2}) = \pi(\mathbf{x}_2^{s_1, s_2})$ . ■

### APPENDIX C PROOF OF LEMMA V.3

*Lemma C.1:* When  $0 \leq \lambda/\mu \leq 1, \gamma > 1$  and for  $s_1 = 0, 1, 2, \dots, N_1, s_2 = 0, 1, 2, \dots, N_2$ , the unnormalized equilibrium distribution for a complete bipartite graph is upper-bounded by an exponential function:

$$\pi(\mathbf{x}^{s_1, s_2}) \propto \left(\frac{\lambda}{\mu}\right)^{s_1 + s_2} \gamma^{s_1 s_2} \leq \left(\frac{\lambda}{\mu} \gamma^\beta\right)^{s_1 + s_2}, \quad (25)$$

where  $\beta = N_1 N_2 / (N_1 + N_2)$ . Furthermore, the relationship holds with equality for  $\mathbf{x}^{s_1, s_2} = \mathbf{x}^N$  and  $\mathbf{x}^{s_1, s_2} = \mathbf{x}^0$ .

*Proof:* Proving the inequality in (25) is equivalent to showing that

$$\frac{s_1 s_2}{s_1 + s_2} \leq \frac{N_1 N_2}{N_1 + N_2}, \text{ for } s_1 = 0, 1, \dots, N_1, s_2 = 0, 1, \dots, N_2, \text{ excluding } s_1 = 0, s_2 = 0. \quad (26)$$

Equation (26) implies that  $N_1 N_2 / (N_1 + N_2) - s_1 s_2 / (s_1 + s_2) \geq 0$ , which we can restate as

$$\frac{N_1 N_2 (s_1 + s_2) - s_1 s_2 (N_1 + N_2)}{(N_1 + N_2)(s_1 + s_2)} \geq 0. \quad (27)$$

The denominator of the LHS of (27) is always positive, so we only need to consider the numerator. Realize that we can rewrite the numerator term of the LHS of (27) as  $N_1 s_1 (N_2 - s_2) + N_2 s_2 (N_1 - s_1)$ . When  $s_1 = 0, 1, \dots, N_1 - 1, s_2 = 0, 1, \dots, N_2 - 1$ ,

$$N_1 s_1 (N_2 - s_2) + N_2 s_2 (N_1 - s_1) > 0. \quad (28)$$

When  $s_1 = N_1, s_2 = N_2$

$$N_1 s_1 (N_2 - s_2) + N_2 s_2 (N_1 - s_1) = 0. \quad (29)$$

Therefore, (26) is satisfied. Since  $\gamma > 1$ , we can conclude that

$$\left(\frac{\lambda}{\mu} \gamma^{s_1 s_2 / (s_1 + s_2)}\right)^{s_1 + s_2} \leq \left(\frac{\lambda}{\mu} \gamma^{N_1 N_2 / (N_1 + N_2)}\right)^{s_1 + s_2}, \quad (30)$$

for  $s_1 = 0, 1, \dots, N_1, s_2 = 0, 1, \dots, N_2$ . Equality is satisfied for  $s_1 = N_1, s_2 = N_2$ , which corresponds to the network configuration  $\mathbf{x}^N$ .

Consider the special case of  $s_1 = 0, s_2 = 0$ . If we rewrite  $((\lambda/\mu) \gamma^{s_1 s_2 / (s_1 + s_2)})^{s_1 + s_2}$  as  $(\lambda/\mu)^{s_1 + s_2} \gamma^{s_1 s_2}$ , then the relationship in (30) is also satisfied with equality. ■

### APPENDIX D PROOF OF LEMMA V.4

*Lemma D.1:* When  $0 \leq \lambda/\mu \leq 1, \gamma > 1$ , and for  $s_1 = 0, 1, 2, \dots, N_1, s_2 = 0, 1, 2, \dots, N_2, \dots, s_m = 0, 1, 2, \dots, N_m$ , the unnormalized equilibrium distribution for a complete multipartite graph is upper-bounded by an exponential function:

$$\begin{aligned} \pi(\mathbf{x}^{s_1, s_2, \dots, s_m}) &\propto \left(\frac{\lambda}{\mu}\right)^{\sum_{p=1}^m s_p} \gamma^{\sum_{i=1}^m s_i \left(\sum_{j \neq i} \mathbb{1}(V_i \sim V_j) s_j\right) / 2} \\ &\leq \left(\frac{\lambda}{\mu} \gamma^\beta\right)^{\sum_{p=1}^m s_p}, \end{aligned} \quad (31)$$

where

$$\beta = \frac{\sum_{i=1}^m N_i \left(\sum_{j \neq i} \mathbb{1}(V_i \sim V_j) N_j\right)}{2 \sum_{p=1}^m N_p}.$$

Furthermore, the inequality holds with equality for  $\mathbf{x}^{s_1, s_2, \dots, s_m} = \mathbf{x}^N$  and  $\mathbf{x}^{s_1, s_2, \dots, s_m} = \mathbf{x}^0$ .

*Proof:* This proof follows the same reasoning as the proof of Lemma V.3 in appendix C; certain steps are skipped for brevity. Proving the inequality in (31) is equivalent to showing that for  $s_1 = 0, 1, 2, \dots, N_1, s_2 = 0, 1, 2, \dots, N_2, \dots, s_m = 0, 1, 2, \dots, N_m$  (excluding the case  $s_1 = 0, s_2 = 0, \dots, s_m = 0$ , which we will handle separately),

$$\beta - \frac{\sum_{i=1}^m s_i \left(\sum_{j \neq i} \mathbb{1}(V_i \sim V_j) s_j\right)}{2 \sum_{p=1}^m s_p} \geq 0. \quad (32)$$

Realize that the sign of the LHS of (32) is determined by

$$\begin{aligned} &\left(\sum_{i=1}^m N_i \left(\sum_{j \neq i} \mathbb{1}(V_i \sim V_j) N_j\right)\right) \left(\sum_{p=1}^m s_p\right) \\ &- \left(\sum_{i=1}^m s_i \left(\sum_{j \neq i} \mathbb{1}(V_i \sim V_j) s_j\right)\right) \left(\sum_{p=1}^m N_p\right), \end{aligned} \quad (33)$$

which we can rewrite as

$$\sum_{i=1}^m \sum_{p=1}^m N_i s_p \left(\sum_{j \neq i} \mathbb{1}(V_i \sim V_j) (N_j - s_j)\right). \quad (34)$$

Equation (34) is positive when  $s_i = 0, 1, \dots, N_i, \forall i = 1, \dots, m$  and is 0 when  $s_i = N_i, \forall i = 1, \dots, m$ , which means that (32) is satisfied.

Consider the special case of  $s_1 = 0, s_2 = 0, \dots, s_m = 0$ . The inequality in (31) is satisfied with equality trivially. ■

### APPENDIX E PROOF OF LEMMA V.5

*Lemma E.1:* When  $0 \leq \lambda/\mu \leq 1, \gamma > 1$ , and for  $s_1 = 0, 1, 2, \dots, N_1, s_2 = 0, 1, 2, \dots, N_2, \dots, s_m = 0, 1, 2, \dots, N_m$ , the unnormalized equilibrium distribution for the complete multipartite graph with regular islands is upperbounded by an exponential function:

$$\begin{aligned} \pi(\mathbf{x}^{s_1, s_2, \dots, s_m}) &\propto \left(\frac{\lambda}{\mu}\right)^{\sum_{p=1}^m s_p} \gamma^{(\mathbf{x}^{s_1, s_2, \dots, s_m})^T A \mathbf{x}^{s_1, s_2, \dots, s_m} / 2} \\ &\leq \left(\frac{\lambda}{\mu} \gamma^\beta\right)^{\sum_{p=1}^m s_p}, \end{aligned} \quad (35)$$

where

$$\beta = \sum_{i=1}^m \frac{k_i^{\text{intra}}}{2} + N_i \left( \frac{\sum_{j \neq i} \mathbb{1}(V_i \sim V_j) N_j}{2 \sum_{p=1}^m N_p} \right).$$

Furthermore, the relationship holds with equality for  $\mathbf{x}^{s_1, s_2, \dots, s_m} = \mathbf{n}^N$  and  $\mathbf{x}^{s_1, s_2, \dots, s_m} = \mathbf{x}^0$ .

*Proof:* Recall that  $N_i$  is the total number of nodes in island  $i$ . There are  $m$  islands so the total number of nodes in the entire network is  $N = \sum_{i=1}^m N_i$ . The number of infected nodes in the  $i$ th island is  $s_i$ . Without loss of generality, we can label the nodes in such a way that  $x_1, \dots, x_{N_1}$  refers to the state of all the nodes in island 1 and  $x_{N_1+1}, \dots, x_{N_1+N_2}$  refers to the state of all the nodes in island 2, and so forth.

Proving (35) is equivalent to proving that for  $s_1 = 0, 1, 2, \dots, N_1, s_2 = 0, 1, 2, \dots, N_2, \dots, s_m = 0, 1, 2, \dots, N_m$  (excluding the case  $s_1 = 0, s_2 = 0, \dots, s_m = 0$ , which we will handle separately),

$$\frac{(\mathbf{x}^{s_1, s_2, \dots, s_m})^T A \mathbf{x}^{s_1, s_2, \dots, s_m}}{2 \sum_{p=1}^m s_p} \leq \beta. \quad (36)$$

We will partition the rows of  $A$  into blocks corresponding to each island. Let  $A_{[i]}$  denote the rows of the  $i$ th block where each block corresponds to different islands,  $A = \{A_{[i]}\}_{i=1}^m$ . Following the same reasoning as the proof of Lemma V.1 in appendix A, we can deduce the following bound for each island

$$A_{[i]} \mathbf{x}^{s_1, s_2, \dots, s_m} = \begin{cases} k_i^{\text{intra}} + \sum_{j \neq i} \mathbb{1}(V_i \sim V_j) s_j, & \text{if } k_i^{\text{intra}} < s_i \\ s_i + \sum_{j \neq i} \mathbb{1}(V_i \sim V_j) s_j, & \text{if } k_i^{\text{intra}} \geq s_i. \end{cases}$$

Recognize that then

$$\begin{aligned} (\mathbf{x}^{s_1, s_2, \dots, s_m})^T A \mathbf{x}^{s_1, s_2, \dots, s_m} &\leq \sum_{i=1}^m s_i \left( k_i^{\text{intra}} + \sum_{j \neq i} \mathbb{1}(V_i \sim V_j) s_j \right). \end{aligned} \quad (37)$$

We can rewrite the RHS of (37) as

$$\sum_{i=1}^m s_i (k_i^{\text{intra}}) + \sum_{i=1}^m s_i \left( \sum_{j \neq i} \mathbb{1}(V_i \sim V_j) s_j \right). \quad (38)$$

Divide the LHS and RHS of (37) by  $2 \sum_{p=1}^m s_p$ . We obtain the new relationship

$$\begin{aligned} &\frac{(\mathbf{x}^{s_1, s_2, \dots, s_m})^T A \mathbf{x}^{s_1, s_2, \dots, s_m}}{2 \sum_{p=1}^m s_p} \\ &\leq \frac{k_i^{\text{intra}} \sum_{i=1}^m s_i + \sum_{i=1}^m s_i \left( \sum_{j \neq i} \mathbb{1}(V_i \sim V_j) s_j \right)}{2 \sum_{p=1}^m s_p}. \end{aligned} \quad (39)$$

Since the largest possible  $s_i$  is  $N_i$ , we can upper bound the RHS of (39) with

$$\sum_{i=1}^m \frac{k_i^{\text{intra}}}{2} + \frac{\sum_{i=1}^m N_i \left( \sum_{j \neq i} \mathbb{1}(V_i \sim V_j) N_j \right)}{2 \sum_{p=1}^m N_p}. \quad (40)$$

This means that

$$\begin{aligned} &\frac{(\mathbf{x}^{s_1, s_2, \dots, s_m})^T A \mathbf{x}^{s_1, s_2, \dots, s_m}}{2 \sum_{p=1}^m s_p} \\ &\leq \sum_{i=1}^m \frac{k_i^{\text{intra}}}{2} + \frac{\sum_{i=1}^m N_i \left( \sum_{j \neq i} \mathbb{1}(V_i \sim V_j) N_j \right)}{2 \sum_{p=1}^m N_p}, \end{aligned} \quad (41)$$

which is the relationship we want to prove in (36). Note that the first term of the RHS of (41) is the same threshold that we derived for  $k$ -regular graphs and the second term is the same term we derived for complete multipartite graphs. ■

### APPENDIX F PROOF OF COROLLARY VI.4

*Corollary F.1:* For the complete graph,  $K_N$ , when  $\lambda/\mu > 1, 0 < \gamma \leq 1$ , if in addition  $\lambda\gamma < \mu, \mathbf{x}^* \in \{\mathbf{x} \in \mathcal{X} \mid 1^T \mathbf{x} = 1\}$ .

*Proof:* For the complete graph, every node is connected to every other node. Therefore, if there are more than 2 infectives in a network configuration, at least 1 infected edge will be created. The maximum number of infectives that result in 0 infected edges is 1. ■

### APPENDIX G PROOF OF COROLLARY VI.5

*Corollary G.1 (Proof in Appendix):* For the bipartite graph, when  $\lambda/\mu > 1, 0 < \gamma \leq 1$ , if in addition  $\lambda\gamma < \mu, \mathbf{x}^* \in \{\mathbf{x} \in \mathcal{X} \mid 1^T \mathbf{x} = \max\{N_1, N_2\}, \mathbf{x}^T A \mathbf{x} = 0\}$ .

*Proof:* Without loss of generality, we assume that  $N_1 \geq N_2$ . We will prove that  $\mathbf{x}^* \in \{\mathbf{x} \in \mathcal{X} \mid 1^T \mathbf{x} = N_1, \mathbf{x}^T A \mathbf{x} = 0\}$  by contradiction. Suppose that  $\mathbf{x}'$  is a configuration where  $\pi(\mathbf{x}') > \pi(\mathbf{x}^*)$ .

*Case I:* Consider when  $1^T \mathbf{x}' < N_1$ . By the definition of a bipartite graph, we can obtain 0 infected edges by placing

all the infected agents in  $V_1$ . Since  $\lambda/\mu > 1$ , we know that  $\pi(\mathbf{x}') < \pi(\mathbf{x}^*)$ , which contradicts the supposition.

*Case 2:* Consider when  $1^T \mathbf{x}' = N_1 + d > N_1$ ,  $d = 1, 2, \dots, N_2$ . Since  $|V_1| = N_1$ , the minimum number of infected edges that will be created is  $d$  (i.e., we place all  $N_1$  infected nodes in  $V_1$  which generates no infected edges for a bipartite graph and the rest of the infected nodes in  $V_2$ ). Therefore

$$\pi(\mathbf{x}') = \left(\frac{\lambda}{\mu}\right)^{N_1+d} \gamma^d, \quad d = 1, 2, \dots, N_2$$

whereas

$$\pi(\mathbf{x}^*) = \left(\frac{\lambda}{\mu}\right)^{N_1}.$$

Realize that

$$\frac{\pi(\mathbf{x}')}{\pi(\mathbf{x}^*)} = \left(\frac{\lambda}{\mu}\right)^d \gamma^d, \quad d = 1, 2, \dots, N_2.$$

Since  $\lambda\gamma < \mu$ ,  $\pi(\mathbf{x}') < \pi(\mathbf{x}^*)$ , which contradicts the supposition. ■

#### APPENDIX H PROOF OF LEMMA VII.1

*Lemma H.1:* Consider two nonnegative vectors  $\mathbf{x} = [x_1, \dots, x_n]$  and  $\mathbf{y} = [y_1, \dots, y_n]$ . We use  $\mathbf{x}^\delta = [x_1^\delta, \dots, x_n^\delta]$  and  $\mathbf{y}^\zeta = [y_1^\zeta, \dots, y_n^\zeta]$  to denote entrywise power. We denote entrywise product as  $\mathbf{x} \circ \mathbf{y} = [x_1 y_1, \dots, x_n y_n]$ . If  $\delta > 1$  and  $\zeta > 1$ , then

$$\left\| \frac{\mathbf{x}^\delta \circ \mathbf{y}^\zeta}{(\mathbf{x}^\delta)^T (\mathbf{y}^\zeta)} \right\|_\infty \geq \left\| \frac{\mathbf{x} \circ \mathbf{y}}{\mathbf{x}^T \mathbf{y}} \right\|_\infty. \quad (42)$$

*Proof:* Without loss of generality, assume that  $x_1 = \|\mathbf{x}\|_\infty$  and  $y_1 = \|\mathbf{y}\|_\infty$ . Since  $\delta > 1$  and  $\zeta > 1$ , then  $x_1^\delta = \|\mathbf{x}^\delta\|_\infty$  and  $y_1^\zeta = \|\mathbf{y}^\zeta\|_\infty$ . Furthermore, as all the terms are nonnegative,  $x_1 y_1 = \|\mathbf{x} \circ \mathbf{y}\|_\infty$  and  $x_1^\delta y_1^\zeta = \|\mathbf{x}^\delta \circ \mathbf{y}^\zeta\|_\infty$ .

To show (42) is equivalent as showing

$$\frac{x_1^\delta y_1^\zeta}{\sum_{i=1}^n x_i^\delta y_i^\zeta} - \frac{x_1 y_1}{\sum_{i=1}^n x_i y_i} \geq 0. \quad (43)$$

We can write the LHS of (43) as

$$x_1 y_1 \left( \frac{x_1^{\delta-1} y_1^{\zeta-1} \left( \sum_{i=1}^n x_i y_i \right) - \left( \sum_{i=1}^n x_i^\delta y_i^\zeta \right)}{\left( \sum_{i=1}^n x_i^\delta y_i^\zeta \right) \left( \sum_{i=1}^n x_i y_i \right)} \right). \quad (44)$$

The sign of (44) is determined by  $x_1^{\delta-1} y_1^{\zeta-1} \left( \sum_{i=1}^n x_i y_i \right) - \left( \sum_{i=1}^n x_i^\delta y_i^\zeta \right)$ , which we can rearrange into

$$\begin{aligned} & x_1^\delta y_1^m + x_1^{\delta-1} y_1^{\zeta-1} \left( \sum_{i=2}^n x_i y_i \right) - x_1^\delta y_1^\zeta - \left( \sum_{i=2}^n x_i^\delta y_i^\zeta \right) \\ &= x_1^{\delta-1} y_1^{\zeta-1} \left( \sum_{i=2}^n x_i y_i \right) - \left( \sum_{i=2}^n x_i^\delta y_i^\zeta \right) \\ &= \sum_{i=2}^n x_i y_i \left( x_1^{\delta-1} y_1^{\zeta-1} - x_i^{\delta-1} y_i^{\zeta-1} \right). \end{aligned} \quad (45)$$

Since all the terms are nonnegative and  $x_1^\delta y_1^\zeta$  is the maximum, expression (45) is either greater than or equal to 0. This implies that

$$\frac{x_1^\delta y_1^\zeta}{\sum_{i=1}^n x_i^\delta y_i^\zeta} - \frac{x_1 y_1}{\sum_{i=1}^n x_i y_i} \geq 0. \quad \blacksquare$$

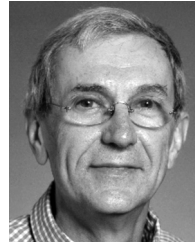
#### REFERENCES

- [1] M. O. Jackson, *Social and Economic Networks*. Princeton, NJ, USA: Princeton Univ. Press, 2008.
- [2] A. Barrat, M. Barthelemy, and A. Vespignani, *Dynamical Processes on Complex Networks*. Cambridge, U.K.: Cambridge Univ. Press, 2008, vol. 1.
- [3] D. J. Watts and P. S. Dodds, "Influentials, networks, and public opinion formation," *J. Consumer Res.*, vol. 34, no. 4, pp. 441–458, 2007.
- [4] A. Vespignani, "Modelling dynamical processes in complex socio-technical systems," *Nature Phys.*, vol. 8, no. 1, pp. 32–39, 2011.
- [5] L. Danon, A. P. Ford, T. House, C. P. Jewell, M. J. Keeling, G. O. Roberts, J. V. Ross, and M. C. Vernon, "Networks and the epidemiology of infectious disease," *Interdisc. Perspect. Infect. Diseases*, 2011.
- [6] C. J. Kuhlman, V. A. Kumar, M. V. Marathe, S. Ravi, and D. J. Rosenkrantz, "Effects of opposition on the diffusion of complex contagions in social networks: An empirical study," in *Social Computing, Behavioral-Cultural Modeling and Prediction*. New York, NY, USA: Springer, 2011, pp. 188–196.
- [7] F. Ball, "The threshold behaviour of epidemic models," *J. Appl. Probabil.*, pp. 227–241, 1983.
- [8] A. Santos and J. M. F. Moura, "Emergent behavior in large scale networks," in *Proc. 50th IEEE Conf. Decision Control and Eur. Control Conf. (CDC-ECC)*, 2011, pp. 4485–4490.
- [9] T. House and M. J. Keeling, "Insights from unifying modern approximations to infections on networks," *J. R. Soc. Interface*, vol. 8, no. 54, pp. 67–73, 2011.
- [10] R. Pastor-Satorras and A. Vespignani, "Epidemic spreading in scale-free networks," *Phys. Rev. Lett.*, vol. 86, no. 14, p. 3200, 2001.
- [11] M. Draief, A. Ganesh, and L. Massoulié, "Thresholds for virus spread on networks," in *Proc. 1st Int. Conf. Perform. Eval. Methodol. Tools*, New York, NY, USA, 2006.
- [12] A. Ganesh, L. Massoulié, and D. Towsley, "The effect of network topology on the spread of epidemics," in *Proc. IEEE INFOCOM '05. 24th Annu. Joint Conf. IEEE Comput. Commun. Soc.*, Mar. 2005, vol. 2, pp. 1455–1466.
- [13] D. Chakrabarti, Y. Wang, C. Wang, J. Leskovec, and C. Faloutsos, "Epidemic thresholds in real networks," *ACM Trans. Inf. Syst. Secur.*, vol. 10, no. 4, pp. 1:1–1:26, 2008.
- [14] C.-K. Chau, V. Pappas, K.-W. Lee, and A. Tantawi, "Self-organizing processes of collective behavior in computer networks," 2009 [Online]. Available: [https://www.usukitacs.com/papers/5196/paper\\_4.pdf](https://www.usukitacs.com/papers/5196/paper_4.pdf)
- [15] Y. Wang, D. Chakrabarti, C. Wang, and C. Faloutsos, "Epidemic spreading in real networks: An eigenvalue viewpoint," in *Proc. 22nd Int. Symp. Reliab. Distrib. Syst.*, 2003, pp. 25–34.
- [16] J. Zhang and J. M. F. Moura, "Accounting for topology in spreading contagion in non-complete networks," in *Proc. IEEE Int. Conf. Acoust., Speech, Signal Process. (ICASSP)*, Mar. 2012, pp. 2681–2684.
- [17] F. P. Kelly, *Reversibility and Stochastic Networks*. New York, NY, USA: Wiley, 1979, vol. 40.
- [18] R. A. Horn and C. R. Johnson, *Matrix Analysis*. Cambridge, U.K.: Cambridge Univ. Press, 2012.
- [19] Y. Song, "On the Independent Set and Common Subgraph Problems in Random Graphs," *ArXiv*, 2013 [Online]. Available: [arXiv:1308.1556](https://arxiv.org/abs/1308.1556)
- [20] J. Abello, S. Butenko, P. M. Pardalos, and M. G. C. Resende, "Finding independent sets in a graph using continuous multivariable polynomial formulations," *J. Global Optimiz.*, vol. 21, no. 2, pp. 111–137, 2001.
- [21] Y. Okamoto, T. Uno, and R. Uehara, "Linear-time counting algorithms for independent sets in chordal graphs," in *Graph-Theoretic Concepts in Computer Science*. New York, NY, USA: Springer, 2005, pp. 433–444.



**June Zhang** received her B.S. with Highest Honor in Electrical and Computer Engineering from the Georgia Institute of Technology in 2005 and a M.S. in Electrical and Computer Engineering from Stanford University in 2008. She is currently a Ph.D. student in Electrical and Computer Engineering at Carnegie Mellon University.

She was a recipient of the Georgia Hope Scholarship and the National Science Foundation Graduate Research Fellowship. Her research interests are stochastic processes, network science, human computer interaction, and design methodology. She is currently very fond of koalas.



**José M. F. Moura** is the Philip and Marsha Dowd University Professor at Carnegie Mellon University in the Departments of Electrical and Computer Engineering and Biomedical Engineering (by courtesy). He holds an EE degree from Instituto Superior Técnico (IST, Portugal) and MSc, EE, and DSc degrees from the Massachusetts Institute of Technology. He has held visiting professor appointments with MIT and NYU and was on the faculty of IST (Portugal). At CMU, he founded and directs the Information and Communication Technologies Institute (ICTI) that

manages the CMU/Portugal Program and co-founded and co-directs the Center for Sensed Critical Infrastructure Research (CenSIR). With CMU colleagues, he cofounded SpiralGen that commercializes, under a CMU license, the Spiral technology ([www.spiral.net](http://www.spiral.net)). His interests are on statistical signal and image processing and data science. He holds eleven issued patents and has published over 450 papers.

Dr. Moura's professional volunteer activities include having been an IEEE Board Director, IEEE Division IX Director, member of several IEEE Boards, President of the IEEE Signal Processing Society (SPS), and Editor-in-Chief for the TRANSACTIONS ON SIGNAL PROCESSING. He has been on the Editorial Boards of several IEEE and ACM Journals.

He received several awards including the IEEE Signal Processing Society Technical Achievement Award and the IEEE Signal Processing Society Award for outstanding technical contributions and leadership in signal processing. Moura is a Fellow of the IEEE, a Fellow of AAAS, a corresponding member of the Academy of Sciences of Portugal, and a member of the US National Academy of Engineering.