Modeling Multi-state Diffusion Process in Complex Networks: Theory and Applications

Yishi Lin John C.S. Lui
Dept. of Computer Science & Eng.
The Chinese Univ. of Hong Kong
Email: {yslin,cslui}@cse.cuhk.edu.hk

Kyomin Jung
Dept. of Electrical & Computer Engineering
Seoul National University
Email: kjung@snu.ac.kr

Sungsu Lim
Dept. of Mathematical Sciences
KAIST
Email: ssungssu@kaist.ac.kr

***Abstract*—There is a growing interest to understand the fundamental principles of how epidemic, ideas or information spread over large networks (e.g., the Internet or online social networks). Conventional approach is to use SIS model (or its derivatives). However, these models usually are over-simplified and may not be applicable in realistic situations. In this paper, we propose a generalized SIS model by allowing intermediate states between susceptible and infected states. To analyze the diffusion process on large graphs, we use the "*mean-field analysis technique*" to determine which initial condition leads to or prevents information or virus outbreak. Numerical results show our methodology can accurately predict the behavior of the phase-transition process for various large graphs (e.g., complete graphs, random graphs or power-law graphs). We also extend our generalized SIS model to consider the interaction of two competing sources (i.e., competing products or virus-antidote modeling). We present the analytical derivation and show experimentally how different factors, e.g., transmission rates, recovery rates, number of states or initial condition, can affect the phase transition process and the final equilibrium. Our models and methodology can serve as an essential tool in understanding information diffusion in large networks.**

I. Introduction

The spread of viruses, ideas or behaviors in networks has been widely studied using mathematical models of contagion [1], [2], [3]. Understanding these dynamical processes is of fundamental importance if we want to control and prevent the spread of diseases, or to maximize the influence of a product in online social networks [4]. One of the most studied contagion model is the *Susceptible-Infected-Susceptible* (SIS) model. In this model, each node in the network can be in one of these two states: "susceptible" or "infected". Using this model, one can describe the spread of contagions like flu or idea. When a node is in the "susceptible" state, it is subjected to influence by its neighboring nodes in the "infected" state, while only nodes in the infected state can influence their neighboring nodes.

However, the SIS model is often too restrictive. To illustrate, consider the case that a diffusion model is used to describe a product adoption [5], [6]. At some point of time after the product release, some people may have purchased the product while some may not. The consumer purchase decision process theory [7] suggests that there are five stages until a consumer makes a purchase and influences others. The states include "product recognition", "information search", "alternative evaluation", "purchase decision", and "post-purchase behavior". This implies that one needs to further divide the *susceptible* state into more states according to the degree of interest.

Contributions: We make several contributions in this paper. First, we propose a generalization of the SIS model by allowing the number of states to adoption (or infection) be more than one (or $k \geq 2$). In particular, the states can be from state 0 to state $k-1$, where the state $k-1$ is the *active* state: the node is infected and can influence its neighboring nodes. Nodes whose state is in 0 to $k-2$ can be promoted to a higher state if they are exposed to its infected neighbor. We analyze the influence spreading dynamics in complete and general graphs, according to which initial condition leads to or prevents a disease outbreak. Specifically, we use the *multidimensional mean-field method* to analyze our model and determine the condition of phase transition.

Our second contribution is to model the behavior and dynamics of *competing sources*. For instance, when there are more than one contagions, ideas or behaviors spreading in the network, how the interaction may affect the final state of the network? We use the generalized SIS model with two sources, one being dominant and the other being recessive, and they compete with each others at the same time. We formulate the dynamic process and show how different factors, such as different transmission rates or initial condition, may affect the phase transition results and final equilibrium.

Last but not least, we show how our methodology can predict the behavior of the diffusion accurately, and we illustrate several applications to show how to design a simple and effective vaccination or advertisement strategy.

II. Generalized SIS Model and Analysis

Let us present our multi-state SIS model. We first model the network as a fully connected undirected graph $G = (V, E)$. Any node $v \in V$ can be in one of $k \geq 2$ states: $\{0, 1, ..., k-1\}$. Only nodes in state $k-1$ (which we call the infected or active state) can increase the state value of

its neighbors, say node s, from state $j \in \{0, 1, \ldots, k-2\}$, to state $j+1$ with an infection rate of β_{j+1}. Each node can recover with a recovery rate of γ. Figure 1 depicts our generalized SIS model.

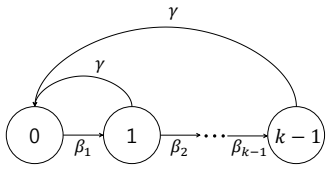


Figure 1. The generalized SIS model with $k \geq 2$ states.

We first briefly review the analysis of the classical SIS (or $k = 2$) model. For the classical SIS model, state 0 corresponds to *susceptible* (S) while state 1 corresponds to *infected* (I). The infection rate and the recovery rate are β and γ respectively. Let $x_0(t)$ and $x_1(t)$ be the fraction of nodes in state S and state I at time $t \geq 0$, respectively. Define $(x_0,\ x_1)$ be an equilibrium for this model (i.e., $\lim_{t\to\infty} x_i(t)$, $i = 1, 2$). Given that G is a fully connected graph, we have $\frac{dx_1}{dt} = \beta x_0 x_1 - \gamma x_1$ and $x_0(t) + x_1(t) = 1$. For this model, there are two possible equilibria, one is $(x_0, x_1) = (1, 0)$ and the other is $(x_0, x_1) = (\frac{\gamma}{\beta}, 1 - \frac{\gamma}{\beta})$.

For an arbitrary graph G, let A be the adjacency matrix of G. Let $x_0^{(i)}(t)$ and $x_1^{(i)}(t)$ be the probability of node i in state S and state I at time t, where $i \in V$, respectively. Let $(x_0^{(i)},\ x_1^{(i)})_{i\in V}$ be an equilibrium for this model. Then, $\frac{dx_1^{(i)}}{dt} = \beta x_0^{(i)} \sum_j A_{ij} x_1^{(j)} - \gamma x_1^{(i)} = 0$[1]. It can be shown that the condition for infection to die out over time is $\beta/\gamma < 1/\lambda_1$ where λ_1 is the largest eigenvalue of A. For detail, please refer [2], [8].

A. Ternary model for Generalized SIS

For the clarity of presentation, let us first consider a generalized SIS model with $k = 3$ states. Here, the state 1 represents that a node is exposed but *not* infected yet. For each state $s \in \{0, 1, 2\}$, let $x_s(t)$ be the fraction of nodes with state s at time t. Let (x_0, x_1, x_2) be an equilibrium for the model. Note that $x_0(t) + x_1(t) + x_2(t) = 1\ \forall t$. Using the mean-field analysis, we derive a system of differential equation that describes the system dynamics:

$$\frac{dx_2}{dt} = \beta_2 x_1 x_2 - \gamma x_2, \tag{1}$$

$$\frac{dx_1}{dt} = -\beta_2 x_1 x_2 + \beta_1 x_0 x_2 - \gamma x_1. \tag{2}$$

Setting $\frac{dx_2}{dt} = 0$, we have $(x_2 = 0)$ or $(x_2 \neq 0$ and $x_1 = \frac{\gamma}{\beta_2})$. We want to find the condition for the non-trivial equilibrium which is the second case. Setting $\frac{dx_1}{dt} = 0$, we have $-\beta_2 x_1 x_2 + \beta_1 x_0 x_2 - \gamma x_1 = 0$. This implies that

$$x_0 = \left(\frac{\beta_2 x_2 + \gamma}{\beta_1 x_2}\right) x_1. \tag{3}$$

Thus, if $x_2 \neq 0$, then $x_1 = \frac{\gamma}{\beta_2}$ and $x_0 = (\frac{\beta_2 x_2 + \gamma}{\beta_1 x_2})\frac{\gamma}{\beta_2}$. Since $\sum_{i=0}^{2} x_i = 1$, we have $1 = (\frac{\beta_2 x_2 + \gamma}{\beta_1 x_2})\frac{\gamma}{\beta_2} + \frac{\gamma}{\beta_2} + x_2$, or

$$\beta_1\beta_2 x_2^2 + [\gamma(\beta_1 + \beta_2) - \beta_1\beta_2]x_2 + \gamma^2 = 0 \tag{4}$$

Theorem *1:* Eq. (4) has real solution iff (1)$\gamma \neq \beta_1$ and $\beta_2 \geq \frac{\gamma\beta_1}{(\sqrt{\gamma}-\sqrt{\beta_1})^2}$; (2)$\gamma = \beta_1$ and $\beta_2 \leq \beta_1/4$.

Proof: The discriminant of this quadratic equation is $D = [\gamma(\beta_1+\beta_2)-\beta_1\beta_2]^2 - 4\gamma^2\beta_1\beta_2$. Let us derive the conditions for $D \geq 0$. First, we could write $D = 0$ as a quadratic equation of β_2, then we can express $D=0$ as:

$$\begin{aligned} D &= (\gamma(\beta_1 + \beta_2) - \beta_1\beta_2)^2 - 4\gamma^2\beta_1\beta_2 \\ &= (\gamma-\beta_1)^2\beta_2^2 + [2\gamma\beta_1(\gamma - \beta_1) - 4\gamma^2\beta_1]\beta_2 + \gamma^2\beta_1^2 = 0. \end{aligned}$$

If $\gamma = \beta_1$, the condition for $D \geq 0$ is

$$\beta_2 \leq \beta_1/4. \tag{5}$$

If $\gamma \neq \beta_1$, the discriminant of this quadratic equation of β_2, denoted by D'_{β_2}, is the following.

$$\begin{aligned} D'_{\beta_2} &= [2\gamma\beta_1(\gamma - \beta_1) - 4\gamma^2\beta_1]^2 - 4(\gamma - \beta_1)^2\gamma^2\beta_1^2 \\ &= 16\gamma^3\beta_1^2[\gamma - (\gamma - \beta_1)] = 16\gamma^3\beta_1^3. \end{aligned}$$

It is easy to see that $D'_{\beta_2} > 0$. Thus the equation $D = 0$ has two solutions, and we denote them by ρ_1 and ρ_2.

$$\begin{aligned} \rho_{1,2} &= \frac{-[2\gamma\beta_1(\gamma - \beta_1) - 4\gamma^2\beta_1] \pm 4\gamma^2\beta_1^2\sqrt{\gamma\beta_1}}{2(\gamma - \beta_1)^2} \\ &= \frac{\gamma\beta_1}{(\sqrt{\gamma} \pm \sqrt{\beta_1})^2}. \end{aligned}$$

If $\gamma \neq \beta_1$, $D \geq 0$ is equivalent to $\beta_2 \geq \frac{\gamma\beta_1}{(\sqrt{\gamma}-\sqrt{\beta_1})^2}$ or $\beta_2 \leq \frac{\gamma\beta_1}{(\sqrt{\gamma}+\sqrt{\beta_1})^2}$. Since $x_1 = \frac{\gamma}{\beta_2}$, it implies that $\beta_2 \geq \gamma$. Furthermore, we have $\frac{\gamma\beta_1}{(\sqrt{\gamma}+\sqrt{\beta_1})^2} < \gamma$ for $\beta_1 > 0$. Thus, if $\gamma \neq \beta_1$, $\beta_1 > 0$ and $\beta_2 \geq \gamma > 0$, $D \geq 0$ is equivalent to

$$\beta_2 \geq \frac{\gamma\beta_1}{(\sqrt{\gamma} - \sqrt{\beta_1})^2}. \tag{6}$$

■

Using this result, we can determine the *region for the phase transition* (i.e., Figure 2). The infection survives if (β_1, β_2) is in this region for $\gamma \neq \beta_1$. In Section II-B, we extend this condition to a general case of $k \geq 2$.

Stability analysis: Let us present the stability condition of the ternary model. Let $f_1(x_1, x_2) = \frac{dx_1}{dt} = -\beta_2 x_1 x_2 + \beta_1(1 - x_1 - x_2)x_2 - \gamma x_1$ and $f_2(x_1, x_2) = \frac{dx_2}{dt} = \beta_2 x_1 x_2 - \gamma x_2$. Define the Jacobian matrix $J = (\frac{df_i}{dx_j})_{i,j=1,2}$. Then, it can

[1]Note that this is a mean-field approximation. The right-hand side of the equation contains two average quantities, $x_0^{(i)}(t)$ and $x_1^{(j)}(t)$, and in multiplying these quantities we are implicitly assuming that the product of the average is equal to the average of their product. For a large graph G, this mean-field approximation is accurate. But for small networks, this may not hold since probabilities are not independent.

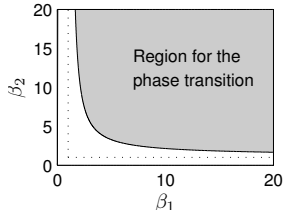


Figure 2. The phase transition region for the ternary model when $\gamma=1$.

be shown that a fixed point (or an equilibrium) of the system of differential equation defined by Eq. (1) and (2) is stable if the determinant (Det) of J is positive and the trace (Tr) of J is negative.

Theorem *2:* We have $Tr<0$ if $x_1=\frac{\gamma}{\beta_2}$ and $Det>0$ if $x_2>\frac{1}{2}(1-\frac{\gamma}{\beta_1}-\frac{\gamma}{\beta_2})$.

Proof: First, we show that $Det>0$ if $x_2>\frac{1}{2}(1-\frac{\gamma}{\beta_1}-\frac{\gamma}{\beta_2})$. We express differential of f_i:

$$\frac{df_1}{dx_1}=-\beta_2x_2-\beta_1x_2-\gamma,\ \frac{df_1}{dx_2}=-\beta_2x_1+\beta_1-\beta_1x_1-2\beta_1x_2,$$
$$\frac{df_2}{dx_1}=\beta_2x_2,\ \ \frac{df_2}{dx_2}=\beta_2x_1-\gamma,$$

If $x_1=\frac{\gamma}{\beta_2}$, the Jacobian matrix J is

$$J=\begin{bmatrix}-\beta_2x_2-\beta_1x_2-\gamma & -\gamma+\beta_1-\frac{\beta_1\gamma}{\beta_2}-2\beta_1x_2\\ \beta_2x_2 & 0\end{bmatrix}.$$

Thus, $Det=-\beta_2x_2(-\gamma+\beta_1-\frac{\beta_1\gamma}{\beta_2}-2\beta_1x_2)>0$ is equivalent to $0>-\gamma+\beta_1-\frac{\beta_1\gamma}{\beta_2}-2\beta_1x_2$, or

$$x_2>\frac{1}{2}(1-\frac{\gamma}{\beta_1}-\frac{\gamma}{\beta_2}). \tag{7}$$

Therefore, a non-trivial equilibrium (x_1,x_2) is stable if $x_2>\frac{1}{2}(1-\frac{\gamma}{\beta_1}-\frac{\gamma}{\beta_2})$, or a saddle point elsewhere. Note that $(x_1,x_2)=(0,0)$ is a stable equilibrium since the eigenvalues of J are less than zero. ∎

Application 1: Let us illustrate how one can apply the results of our ternary SIS model. Assume there is a large-scale computer virus spreading, the detection rate of an anti-virus software γ need to be high enough in order to prevent the spreading for the given (β_1,β_2). This leads to the following vaccination strategy: the value of γ need to guarantee that (1) Eq. (4) has no feasible solution of x_2 in $(0,1]$; (2) Or, all feasible solutions x_2 are not stable, i.e., $x_2\le\frac{1}{2}(1-\frac{\gamma}{\beta_1}-\frac{\gamma}{\beta_2})$.

B. Generalized Multi-state SIS model for complete graphs

We first analyze the general SIS model for $k\ge2$ and the underlying network is a complete graph. Extension for general will follow. For each $s\in\{0,1,\ldots,k-1\}$, let $x_s(t)$ be the fraction of nodes in state s at time t. Let $(x_0,x_1,\ldots,x_{k-1})$ be an equilibrium for the model. Then, we obtain the following system of differential equation.

$$\frac{dx_{k-1}}{dt}=\beta_{k-1}x_{k-2}x_{k-1}-\gamma x_{k-1}, \tag{8}$$
$$\frac{dx_s}{dt}=-\beta_{s+1}x_sx_{k-1}+\beta_sx_{s-1}x_{k-1}-\gamma x_s\ \forall s\in\{1,..,k\text{–}2\}, \tag{9}$$
$$\frac{dx_0}{dt}=-\beta_1x_0x_{k-1}+\gamma(1-x_0). \tag{10}$$

Setting $\frac{dx_{k-1}}{dt}=0$, we have $(x_{k-1}=0)$ or $(x_{k-1}\ne0$ and $x_{k-2}=\frac{\gamma}{\beta_{k-1}})$. We set $\frac{dx_s}{dt}=0$ for any $1\le s\le k-2$, or $-\beta_{s+1}x_{k-1}x_s+\beta_sx_{k-1}x_{s-1}-\gamma x_s=0$. This implies that

$$x_{s-1}=\left(\frac{\beta_{s+1}x_{k-1}+\gamma}{\beta_sx_{k-1}}\right)x_s=\left(\prod_{j=s}^{k-2}\frac{\beta_{j+1}x_{k-1}+\gamma}{\beta_jx_{k-1}}\right)x_{k\text{–}2}. \tag{11}$$

Thus, if $x_{k-1}\ne0$, the condition $\sum_{i=0}^{k-1}x_i=1$ is equivalent to $1=x_{k-1}+\frac{\gamma}{\beta_{k-1}}+\sum_{s=1}^{k-2}(\prod_{j=s}^{k-2}\frac{\beta_{j+1}x_{k-1}+\gamma}{\beta_jx_{k-1}})\frac{\gamma}{\beta_{k-1}}$, which is a $(k-1)$-dimension equation of x_{k-1}. Multiplying $\beta_1\beta_2\ldots\beta_{k-1}x_{k-1}^{k-2}$ on both sides, we have

$$\beta_1\beta_2\ldots\beta_{k-1}x_{k-1}^{k-2}=(\beta_1x_{k-1}+\gamma)\ldots(\beta_{k-1}x_{k-1}+\gamma). \tag{12}$$

This holds for any $k\ge2$, and one can check that this argument holds via mathematical induction on k.

Now let us consider a special case in which the infection rates are increasing geometrically with a growth rate $\alpha>1$ so that $\beta_{k-1}=\alpha\beta_{k-2}=\ldots=\alpha^{k-2}\beta_1$. Then, for nonzero β_1, Eq. (12) is equivalent to $\frac{1}{x_{k-1}}=(1+\frac{\gamma}{\beta_1x_{k-1}})\ldots(1+\frac{\gamma}{\beta_{k-1}x_{k-1}})$. For simplicity, we take $\beta=\beta_1$. Substituting y with $\frac{1}{\beta x_{k-1}}$, we have $\beta y=(1+\gamma y)(1+\frac{\gamma}{\alpha}y)\ldots(1+\frac{\gamma}{\alpha^{k-2}}y)$.

Let $g_1(y)=\beta y$ and $g_2(y)=(1+\gamma y)(1+\frac{\gamma}{\alpha}y)\ldots(1+\frac{\gamma}{\alpha^{k-2}}y)$. Then, these two functions of y are positive, monotone increasing, and convex for $y>0$ since $g_2(y)=0$ has only negative solutions $y=-\gamma,-\gamma\alpha,\ldots,-\gamma\alpha^{k-2}$. Thus, $g_1(y)=g_2(y)$ has at most two solutions. Moreover, for a fixed α, there is a *tipping point* β_t (or equivalently epidemic threshold) so that the equation has no solution if $\beta<\beta_t$ and has two solutions if $\beta>\beta_t$.

For instance, for the case that $\alpha=1$ (i.e., the infection rates are homogeneous), $g_1(y)=g_2(y)$ is equivalent to $\beta y=(1+\gamma y)^{k-1}$. Note that the slopes of $g_1(y)$ and $g_2(y)$ are the same at the tipping point with $\beta=\beta_t$. Since $\frac{d}{dy}\beta y=\beta$ and $\frac{d}{dy}(1+\gamma y)^{k-1}=\gamma(k-1)(1+\gamma y)^{k-2}$, $y=\frac{1}{\gamma}\{(\frac{\beta}{\gamma(k-1)})^{1/(k-2)}-1\}$ at the intersecting point. Substituting y with $\frac{1}{\gamma}\left((\frac{\beta}{\gamma(k-1)})^{1/(k-2)}-1\right)$, we have $\frac{\beta}{\gamma}\{(\frac{\beta}{\gamma(k-1)})^{1/(k-2)}-1\}=(\frac{\beta}{\gamma(k-1)})^{(k-1)/(k-2)}$. When β is nonzero, we can derive the value of β as:

$$\frac{\beta}{\gamma}\left[\left(\frac{\beta}{\gamma(k-1)}\right)^{1/(k-2)}-1\right]=\left(\frac{\beta}{\gamma(k-1)}\right)^{(k-1)/(k-2)}$$
$$\frac{\beta}{\gamma}\left[\frac{\beta^{\frac{1}{k-2}}}{\gamma^{\frac{1}{k-2}}(k-1)^{\frac{1}{k-2}}}-1\right]=\frac{\beta}{\gamma}\left[\frac{\beta^{\frac{1}{k-2}}}{r^{\frac{1}{k-2}}(k-1)^{\frac{k-1}{k-2}}}\right]$$

$$\beta^{\frac{1}{k-2}}\left[\frac{1}{\gamma^{\frac{1}{k-2}}(k-1)^{\frac{1}{k-2}}}-\frac{1}{\gamma^{\frac{1}{k-2}}(k-1)^{\frac{k-1}{k-2}}}\right]=1$$

$$\beta=\gamma\frac{(k-1)^{(k-1)}}{(k-2)^{(k-2)}}. \quad (13)$$

Application 2: Consider a computer virus outbreaks in a network which is represented by G. We can devise an effective vaccination strategy from Eq. (8)-(10). For example, we can provide an anti-virus software with a suitable detection rate of γ. When $\gamma > \beta\frac{(k-2)^{(k-2)}}{(k-1)^{(k-1)}}$, the fraction of nodes at the infected state converges to zero for large t. Furthermore, when k increases (i.e., it takes more phases to activate a virus), the threshold for γ decreases. Thus it is easier to control the outbreak when k is larger. On the other hand, if we want to promote a product in an online social network, then decreasing k is crucial.

C. Generalized Multi-state SIS Model in General Graphs

Let us consider a general graph $G = (V, E)$ with a generalized SIS model of $k \geq 2$. Let A be the adjacency matrix of G. For each $s \in \{0, 1, ..., k-1\}$, let $\langle x_s^{(i)}(t)\rangle$ be the average probability that node i is in state s at time t. Let $\langle x_{s_1}^{(i)}(t), x_{s_2}^{(j)}(t)\rangle$ be the average probability that node i is in state s_1 and node j is in state s_2. In general graph, the transmission rate is the rate at which infection will be transmitted between an infected individual and a susceptible individual. We denote the transmission rate by β_i' for each $i \in \{0, 1, ..., k-1\}$. Then, for each node $i \in V$, we obtain the following differential equation via mean-field analysis.

$$\frac{d\langle x_{k-1}^{(i)}\rangle}{dt}=\beta'_{k-1}\sum_j A_{ij}\langle x_{k-2}^{(i)}, x_{k-1}^{(j)}\rangle-\gamma\langle x_{k-1}^{(i)}\rangle, \quad (14)$$

$$\frac{d\langle x_s^{(i)}\rangle}{dt}=-\beta'_{s+1}\sum_j A_{ij}\langle x_s^{(i)}, x_{k-1}^{(j)}\rangle + \beta'_s\sum_j A_{ij}\langle x_{s-1}^{(i)}, x_{k-1}^{(j)}\rangle-\gamma\langle x_s^{(i)}\rangle \quad \forall s \in \{1, \ldots, k-2\}. \quad (15)$$

Note that we have $x_0^{(i)} = 1 - \sum_{s=1}^{k-1} x_s^{(i)}$ for each node i.

Now we give an approximation to the above true equation in which we assume that $\langle x_{s_1}^{(i)}(t), x_{s_2}^{(j)}(t)\rangle = \langle x_{s_1}^{(i)}(t)\rangle\langle x_{s_2}^{(j)}(t)\rangle$. Let $\mathbf{x_s}$ be a column vector containing $x_s^{(i)}$ for all nodes i, i.e. $\mathbf{x_s} = (x_s^{(1)}, ..., x_s^{(|V|)})$. We write the approximation equations in matrix form. For simplicity, we omit the angle brackets.

$$\frac{d\mathbf{x_{k-1}}}{dt}=\beta'_{k-1}\mathbf{x_{k-2}}\circ(A\mathbf{x_{k-1}})-\gamma\mathbf{x_{k-1}}, \quad (16)$$

$$\frac{d\mathbf{x_s}}{dt}=(\beta'_s\mathbf{x_{s-1}}-\beta'_{s+1}\mathbf{x_s})\circ(A\mathbf{x_{k-1}})-\gamma\mathbf{x_s}\ \forall s\in\{1,..,k-2\}, \quad (17)$$

$$\mathbf{x_0}=\mathbf{1}-\sum_{s=1}^{k-1}\mathbf{x_s^{(i)}}. \quad (18)$$

where $A \circ B$ is the Hadamard product of matrix A and matrix B, $(A \circ B)_{ij} = (A)_{ij}(B)_{ij}$. For any given graph G and initial fraction of nodes in each states s, i.e. $x_s^{(i)}(0)$, we can numerically calculate the probability of each nodes in each states as a function of time.

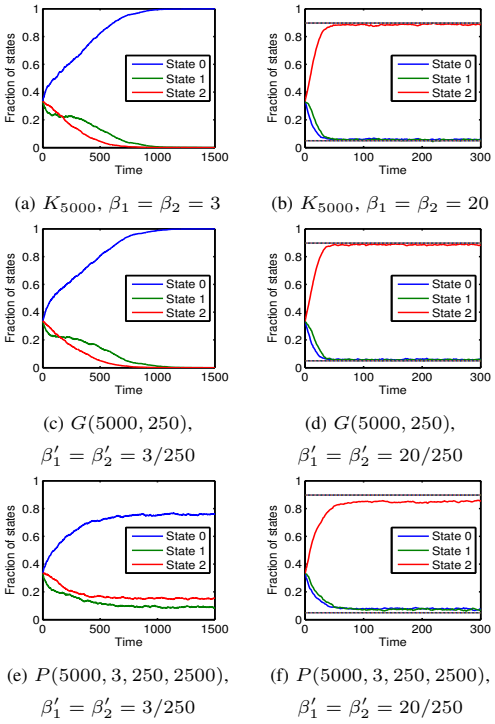
III. Numerical results for Generalized SIS Model

We conduct a set of numerical experiments using the generalized SIS model and study the dynamics of fractions of states for different network datasets. Our network datasets include (i) a *complete graph* K_N with N nodes, (ii) a *Erdös-Rényi random graph* $G(N, M)$ with N nodes and M edges, and (iii) a *random power law graph* $P(N, \theta, d, m)$ with N nodes, the exponent θ, the expected average degree d and the maximum degree m [9]. In our experiments, the initial state value of each node is chosen independently and uniformly at random from $\{0, 1, \ldots, k-1\}$ according to a given initial rate.

Note that although our analysis focuses on case (i), our results can be applied to the case (ii) with some constant factor with respect to M. Figure 3 compares the dynamics for the ternary model with different β_1 and β_2 values. Figure 3(a) shows that if there is no non-trivial stationary equilibrium, then (x_0, x_1, x_2) converges to $(1, 0, 0)$ for large t. However, if there is a stationary equilibrium, then there is a possibility that (x_1, x_2) converges to another point. In Figure 3(b), the dotted lines represent a stationary non-trivial equilibrium $(x_1, x_2) = (0.05, 0.897)$ and (x_1, x_2) converges to it. Figure 3(c) and 3(d) show the analysis holds for $\beta_1' = \beta_1/M$ and $\beta_2' = \beta_2/M$ ($M = 250$). It is interesting to note that for the case (iii), our method still succeeds to predict the equilibrium condition with $\beta_1' = \beta_1/d$ and $\beta_2' = \beta_2/d$ (d=250), as shown in Figure 3(e) and 3(f).

IV. General SIS Model with Competing Sources

Previous work usually consider only one contagion source which spreads virus (or information) in a network. However, in practice, there might be more than one kind of contagions, ideas or behaviors spreading at the same time. In this section, we consider two *competing sources*: a and b, which simultaneously spread their influence in a network. We extend the generalized SIS model discussed in section II to a model with two competing sources. Without loss of generality, we assume source b is more dominant than source a such that even a node is on its way to become activated by source a, this node is still possible to being influenced by its neighbors activated by source b and become activated by source b eventually. The converse is not true, i.e. source a has no such power over b. One application of such model is to consider a spreading of virus a. To eliminate such virus spreading, an antidote (source b) is introduced into the network. We first present the formal analysis of a ternary model in a large complete graph. Then we formulate the model with two competing sources under a general graph.



(a) K_{5000}, $\beta_1 = \beta_2 = 3$ (b) K_{5000}, $\beta_1 = \beta_2 = 20$

(c) $G(5000, 250)$, $\beta_1' = \beta_2' = 3/250$ (d) $G(5000, 250)$, $\beta_1' = \beta_2' = 20/250$

(e) $P(5000, 3, 250, 2500)$, $\beta_1' = \beta_2' = 3/250$ (f) $P(5000, 3, 250, 2500)$, $\beta_1' = \beta_2' = 20/250$

Figure 3. Numerical results: The dynamics of x_0, x_1 and x_2 over time where $\gamma = 1$. (Initial fraction of nodes in different states: $x_0(0) = x_1(0) = x_2(0) = 1/3$)

A. Ternary model in a large complete graph

Let us first consider a model with two competing sources "a" and "b" as depicted in Figure 4. In this model, the underlying network is a large complete graph $G = (V, E)$. Any node $v \in V$ can be in one of 5 states: $\{0, a_1, a_2, b_1, b_2\}$. Nodes in state a_2 and b_2 are in activation state for a and b respectively. Nodes activated by source a (or b) can change the state value of its neighbors, say node s, which is in state 0 or a_1 (0 or b_1), to state a_1 or a_2 (b_1 or b_2). Furthermore, to represent the dominant behavior of source b, nodes in state b_2 can change their neighboring nodes in state a_1 to state b_1 with a non-zero probability. Nodes in state a_1 or a_2 can independently recover with a rate γ_a (recovery rate). Similarly, nodes in state b_1 or b_2 can recover with a rate γ_b. Assume that the underlying network is a complete graph. For each state $s \in \{0, a_1, a_2, b_1, b_2\}$, let $x_s(t)$ be the fraction of nodes in state s at time t. The dynamics of the system are:

$$\frac{dx_{a_1}}{dt} = \alpha_1 x_0 x_{a_2} - \alpha_2 x_{a_1} x_{a_2} - \lambda x_{a_1} x_{b_2} - \gamma_a x_{a_1}, \quad (19)$$

$$\frac{dx_{a_2}}{dt} = \alpha_2 x_{a_1} x_{a_2} - \gamma_a x_{a_2}, \quad (20)$$

$$\frac{dx_{b_1}}{dt} = \beta_1 x_0 x_{b_2} - \beta_2 x_{b_1} x_{b_2} + \lambda x_{a_1} x_{b_2} - \gamma_b x_{b_1}, \quad (21)$$

$$\frac{dx_{b_2}}{dt} = \beta_2 x_{b_1} x_{b_2} - \gamma_b x_{b_2}, \quad (22)$$

$$x_0(t) = 1 - x_{a_1}(t) - x_{a_2}(t) - x_{b_1}(t) - x_{b_2}(t). \quad (23)$$

For the above system, there exists no closed-form solution, but one can solve it numerically so to understand the impact and dynamics of these two competing sources. We will illustrate our numerical results in later section.

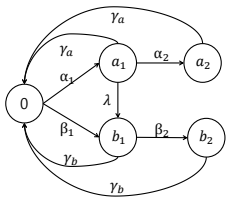


Figure 4. A ternary SIS model with two competing sources.

B. General Multi-state model in large complete graph

Now we proceed to analyze a general SIS with two competing sources. Let $\mathcal{M} \geq 2$ and $\mathcal{N} \geq 2$ be the number of additional states for sources a and b. Noted that there is one additional initial state 0, so the total number of states is $\mathcal{M} + \mathcal{N} + 1$. For example, in ternary model, we have $\mathcal{M} = \mathcal{N} = 2$. Figure 5 depicts the state-transition diagram of this general multi-state model.

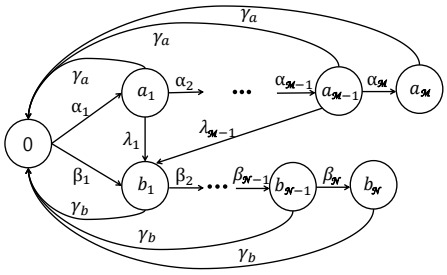


Figure 5. The generalized SIS model with two competing sources.

Let say a node i is activated by the dominant source b and one of its neighboring node j is in state a_i ($1 \leq i \leq \mathcal{M}-1$), we assume that node i can change node j from state a_i to state b_1 with probability λ_i per unit time. In some real world cases, for a node in state a_k ($1 \leq k \leq \mathcal{N}-1$), a higher value of k implies that the node is closer to being activated by the source a. In this case, we may have $\lambda_1 \geq \lambda_2 \geq ... \geq \lambda_{\mathcal{M}-1}$. Take consumer purchase decision process as an example, suppose $\mathcal{M} = 3$, a_1 is the stage that a customer have heard about a new product and a_2 is the stage that he is planing to make a purchase. For this example, a customer in stage a_1 is easier to change his mind if he is exposed to a more dominant product, say b. In other word, we can assume that $\lambda_1 \geq \lambda_2$. Note that $x_0 = 1 - \sum_{i=1}^{\mathcal{M}} x_{a_i} - \sum_{j=1}^{\mathcal{N}} x_{b_j}$. The dynamics of the system can be specified by:

$$\frac{dx_{a_1}}{dt} = \alpha_1 x_0 x_{a_{\mathcal{M}}} - \alpha_2 x_{a_1} x_{a_{\mathcal{M}}} - \lambda_1 x_{a_1} x_{b_{\mathcal{N}}} - \gamma_a x_{a_1}, \quad (24)$$

$$\frac{dx_{a_i}}{dt} = \alpha_i x_{a_{i-1}} x_{a_{\mathcal{M}}} - \alpha_{i+1} x_{a_i} x_{a_{\mathcal{M}}} - \lambda_i x_{a_i} x_{b_{\mathcal{N}}} - \gamma_a x_{a_i} \quad \forall i \in \{2, \ldots, \mathcal{M}-1\}, \quad (25)$$

$$\frac{dx_{a_{\mathcal{M}}}}{dt} = \alpha_{\mathcal{M}} x_{a_{\mathcal{M}-1}} x_{a_{\mathcal{M}}} - \gamma_a x_{a_{\mathcal{M}}}, \quad (26)$$

$$\frac{dx_{b_1}}{dt} = \beta_1 x_0 x_{b_{\mathcal{N}}} - \beta_2 x_{b_1} x_{b_{\mathcal{N}}} + \sum_{l=1}^{\mathcal{M}-1} \lambda_l x_{a_l} x_{b_{\mathcal{N}}} - \gamma_b x_{b_1}, \quad (27)$$

$$\frac{dx_{b_j}}{dt} = \beta_j x_{b_{j-1}} x_{b_{\mathcal{N}}} - \beta_{j+1} x_{b_j} x_{b_{\mathcal{N}}} - \gamma_b x_{b_j} \ \forall j \in \{2, .., \mathcal{N}-1\}, \quad (28)$$

$$\frac{dx_{b_\mathcal{N}}}{dt} = \beta_\mathcal{N} x_{b_{\mathcal{N}-1}} x_{b_\mathcal{N}} - \gamma_b x_{b_\mathcal{N}}. \quad (29)$$

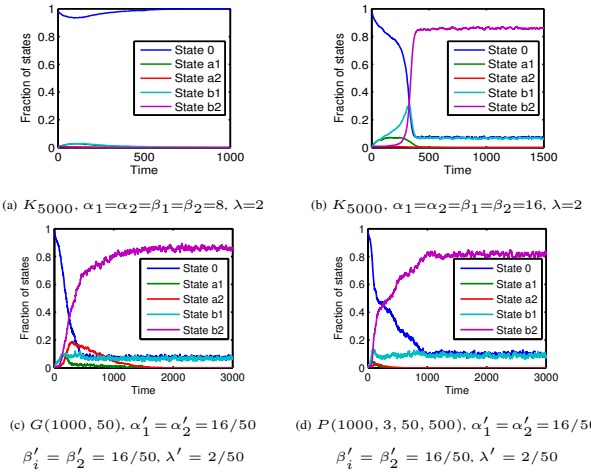
It is easy to verify that the ternary model discussed in Sec. IV-A is a special case of this model, where $\mathcal{M}=\mathcal{N}=2$.

C. Multi-state model in general graph

In a complete graph, we assume that the contact is possible with the entire population. However, for general graphs, only an activated node can influence its neighbors. In this model, the transmission rate is the rate that a source being transmitted between two nodes, one activated and one non-activated, and they are connected by an edge in the graph. In a complete graph, the transmission rate is the rate of contacts between an activated node and all others, whereas in general graph it is the rate of contacts between neighboring nodes. We denote the transmission rate here by α'_i $(1\le i\le\mathcal{M})$, β'_j $(1\le j\le\mathcal{N})$ and λ'_l $(1\le l\le\mathcal{M}-1)$. For example, assume node i and node j are connected. Suppose node i is in state $b_\mathcal{N}$ (activated by source b) and node j is in state 0, node j can change from state 0 to b_1 with rate β'_1. Suppose another node k is in state a_1. The rate that node k will change to state b_1 is λ'_1. Noted that the transmission rate in complete graph and general graph is slightly different with each other.

Let $G=(V,E)$ be the underlying general graph, where A be the adjacency matrix of G. Let $\mathcal{M}\ge 2$ and $\mathcal{N}\ge 2$ be the number of additional states of sources a and b. For each $s\in\{0,a_1,...,a_\mathcal{M},b_1,...,b_\mathcal{N}\}$, let $x_s^{(i)}(t)$ be the average probability that node i is in state s at time t. Let $\langle x_{s_1}^{(i)}(t), x_{s_2}^{(j)}(t)\rangle$ be the average probability that node i is in state s_1 and node j is in state s_2 at time t. And we use approximation $\langle x_{s_1}^{(i)}, x_{s_2}^{(j)}\rangle \approx x_{s_1}^{(i)} x_{s_2}^{(j)}$ to close the equations at the level of one-variable average. We can express the system dynamics in matrix form:

$$\begin{aligned}
\frac{d\mathbf{x_{a_1}}}{dt} &= \alpha'_1\mathbf{x_0}\circ(A\mathbf{x_{a_\mathcal{M}}}) - \alpha'_2\mathbf{x_{a_1}}\circ(A\mathbf{x_{a_\mathcal{M}}}) \\
&\quad -\lambda'_1\mathbf{x_{a_1}}\circ(A\mathbf{x_{b_\mathcal{N}}}) - \gamma_a\mathbf{x_{a_1}}, && (30)\\
\frac{d\mathbf{x_{a_k}}}{dt} &= \alpha'_k\mathbf{x_{a_{k-1}}}\circ(A\mathbf{x_{a_\mathcal{M}}}) - \alpha'_{k+1}\mathbf{x_{a_k}}\circ(A\mathbf{x_{a_\mathcal{M}}}) \\
&\quad -\lambda'_k\mathbf{x_{a_k}}\circ(A\mathbf{x_{b_\mathcal{N}}}) - \gamma_a\mathbf{x_{a_k}} && (31)\\
&\quad \forall k\in\{2,..,\mathcal{M}-1\}, && (32)\\
\frac{d\mathbf{x_{a_\mathcal{M}}}}{dt} &= \alpha'_\mathcal{M}\mathbf{x_{a_{\mathcal{M}-1}}}\circ(A\mathbf{x_{a_\mathcal{M}}}) - \gamma_a\mathbf{x_{a_\mathcal{M}}}, && (33)\\
\frac{d\mathbf{x_{b_1}}}{dt} &= \beta'_1\mathbf{x_0}\circ(A\mathbf{x_{b_\mathcal{N}}}) - \beta'_2\mathbf{x_{b_1}}\circ(A\mathbf{x_{b_\mathcal{N}}}) \\
&\quad + \sum_{l=1}^{\mathcal{M}-1}\lambda'_l\mathbf{x_{a_l}}\circ(A\mathbf{x_{b_\mathcal{N}}}) - \gamma_b\mathbf{x_{b_1}}, && (34)\\
\frac{d\mathbf{x_{b_k}}}{dt} &= \beta'_k\mathbf{x_{b_{k-1}}}\circ(A\mathbf{x_{b_\mathcal{N}}}) - \beta'_{k+1}\mathbf{x_{b_k}}\circ(A\mathbf{x_{b_\mathcal{N}}}) && (35)\\
&\quad -\gamma_b\mathbf{x_{b_k}}\ \forall k\in\{2,..,\mathcal{N}-1\}, && (36)\\
\frac{d\mathbf{x_{b_\mathcal{N}}}}{dt} &= \beta'_\mathcal{N}\mathbf{x_{b_{\mathcal{N}-1}}}\circ(A\mathbf{x_{b_\mathcal{N}}}) - \gamma_b\mathbf{x_{b_\mathcal{N}}}, && (37)\\
\mathbf{x_0} &= \mathbf{1} - \sum_{i=1}^{\mathcal{M}}\mathbf{x_{a_i}} - \sum_{j=1}^{\mathcal{N}}\mathbf{x_{b_j}}. && (38)
\end{aligned}$$



(a) K_{5000}, $\alpha_1{=}\alpha_2{=}\beta_1{=}\beta_2{=}8$, $\lambda{=}2$ (b) K_{5000}, $\alpha_1{=}\alpha_2{=}\beta_1{=}\beta_2{=}16$, $\lambda{=}2$

(c) $G(1000, 50)$, $\alpha'_1=\alpha'_2=16/50$, $\beta'_i=\beta'_2=16/50$, $\lambda'=2/50$ (d) $P(1000, 3, 50, 500)$, $\alpha'_1=\alpha'_2=16/50$, $\beta'_i=\beta'_2=16/50$, $\lambda'=2/50$

Figure 6. Simulation results: The dynamics of x_0, x_{a_1}, x_{a_2}, x_{b_1} and x_{b_2} over time where $\gamma_a=\gamma_b=1$. (Initial fraction of nodes in different states: $x_0(0)=0.98$, $x_{a_2}(0)=0.01$ and $x_{b_2}(0)=0.01$)

In fact, the above derivation is equivalent to Eqs.(24)-(29) for complete graphs when $\alpha_i=(N-1)\alpha'_i$ for all $1\le i\le\mathcal{M}$, $\lambda_l=(N-1)\lambda'_l$ for all $1\le l\le\mathcal{M}-1$ and $\beta_j=(N-1)\beta'_j$ for all $1\le j\le\mathcal{N}$. For detailed derivation, please refer [10].

V. Numerical Results For Competing Sources

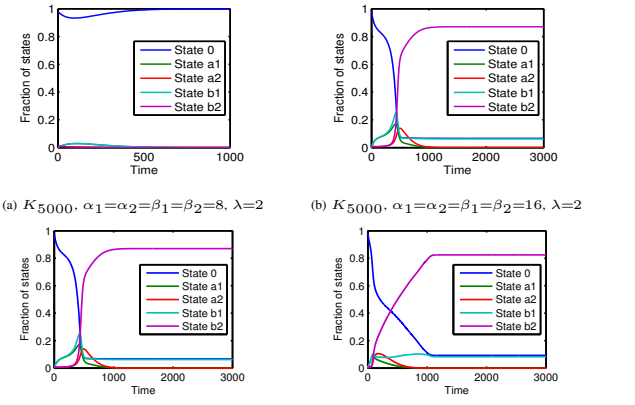
Here, we provide numerical results when the network has two competing sources. Similar to Sec.III, we consider networks of (i) a complete graph K_N, (ii) a *Erdös-Rényi random graph* $G(N,M)$ with N nodes and M edges, and (iii) a random power law graph $P(N,\theta,d,m)$. The initial state value of each node is chosen independently and uniformly according to a given initial rate.

A. Ternary model

Results for ternary model: Figure 6 shows the simulation results of ternary model in a complete graph, an ER random graph and a random power law graph. We can observe that when transmission rates α_i and β_i are not sufficiently large, the epidemic will eventually die out. In Figure 6 (b)-(d), we have $\alpha=50\alpha'$, $\beta=50\beta'$ and $\lambda=50\lambda'$. Thus, the expected number of contacts of an activated nodes remain the same, and we observe similar phase transition and equilibrium in these three networks.

Figure 7 shows the numerical results given by solving Eq. (19) to Eq. (23) for complete graph and Eq. (30) to Eq. (38) for general graph. Comparing Fig. 7 with Fig. 6, we can see that the numerical results of solving the differential equations are very close to the numerical results given by simulation. We discussed in Sec.IV that Eq. (30)-(38) for general graph is an approximation. However, from the experiment results, we can see that the approximation agrees very well with the simulation results in Fig. 6.

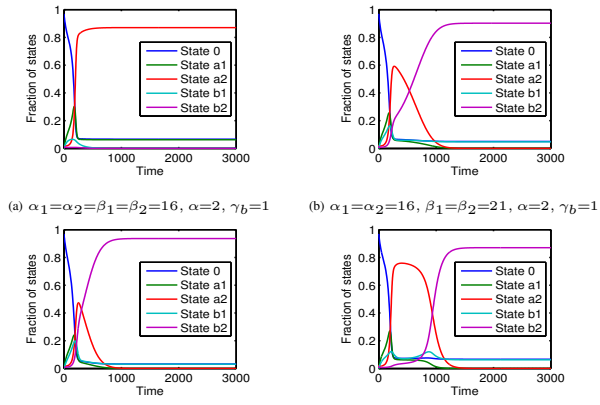
From now on, we show only the experiments based on complete graph K_{5000}. We use numerical results given by



(a) K_{5000}, $\alpha_1{=}\alpha_2{=}\beta_1{=}\beta_2{=}8$, $\lambda{=}2$ (b) K_{5000}, $\alpha_1{=}\alpha_2{=}\beta_1{=}\beta_2{=}16$, $\lambda{=}2$

(c) $G(1000, 50)$, $\alpha'_1{=}\alpha'_2{=}16/50$, $\beta'_1{=}\beta'_2{=}16/50$, $\lambda'{=}2/50$ (d) $P(1000, 3, 50, 500)$, $\alpha'_1{=}\alpha'_2{=}16/50$, $\beta'_1{=}\beta'_2{=}16/50$, $\lambda'{=}2/50$

Figure 7. Numerical results: The dynamics of x_0, x_{a_1}, x_{a_2}, x_{b_1} and x_{b_2} over time where $\gamma_a{=}\gamma_b{=}1$. (Initial fraction of nodes in different states: $x_0(0) = 0.98$, $x_{a_2}(0) = 0.01$ and $x_{b_2}(0) = 0.01$)



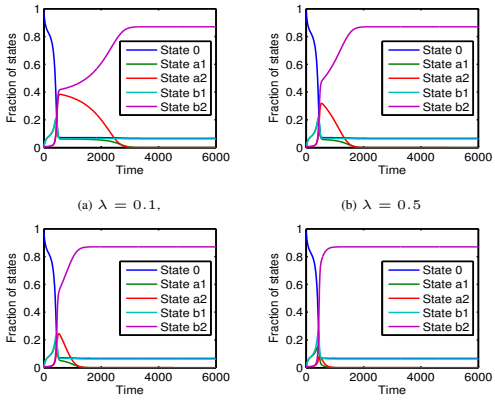
(a) $\alpha_1{=}\alpha_2{=}\beta_1{=}\beta_2{=}16$, $\alpha{=}2$, $\gamma_b{=}1$ (b) $\alpha_1{=}\alpha_2{=}16$, $\beta_1{=}\beta_2{=}21$, $\alpha{=}2$, $\gamma_b{=}1$

(c) $\alpha_1{=}\alpha_2{=}\beta_1{=}\beta_2{=}16$, $\lambda{=}2$, $\gamma_b{=}0.5$ (d) $\alpha_1{=}\alpha_2{=}\beta_1{=}\beta_2{=}16$, $\lambda{=}31$, $\gamma_b{=}1$

Figure 8. Numerical results: The dynamics of x_0, x_{a_1}, x_{a_2}, x_{b_1} and x_{b_2} over time where the graph is K_{5000} and $\gamma_a = 1$. (Initial fraction of nodes in different states: $x_0(0){=}0.97$, $x_{a_2}(0){=}0.02$ and $x_{b_2}(0){=}0.01$)

Eq. (19) to Eq. (23). For ER random graph and power law graph, the results could also be applied.

Impact of delay in deploying source b**:** Figure 8 shows the numerical results for which $x_{a_2}(0){=}2x_{b_2}(0){=}0.02$. By setting different initial fraction of infected nodes for sources a and b, we can examine the impact of delay in the phase-transition process. For example, if $x_{a_2}(0) > x_{b_2}(0)$, we can assume that source b is introduced later than a. In Fig. 8 (a), we can see that for product b, if $\beta_{1,2}$ and λ are not large enough, it cannot compete well with product a. Eventually, the fraction of nodes in state b_2 will approach zero. On the other hand, for a product b, if it is far more superior than product a (i.e., λ and $\beta_{1,2}$ are sufficiently large, or γ_b is small), potential buyers can be easily persuaded to eventually adopt product b. Fig. 8(b) (c) and (d) correspond to these situations, i.e., if source b is sufficiently superior, it will be the dominant source even if it is introduced to the network later than source a.



(a) $\lambda = 0.1$, (b) $\lambda = 0.5$

(c) $\lambda = 1$ (d) $\lambda = 3$

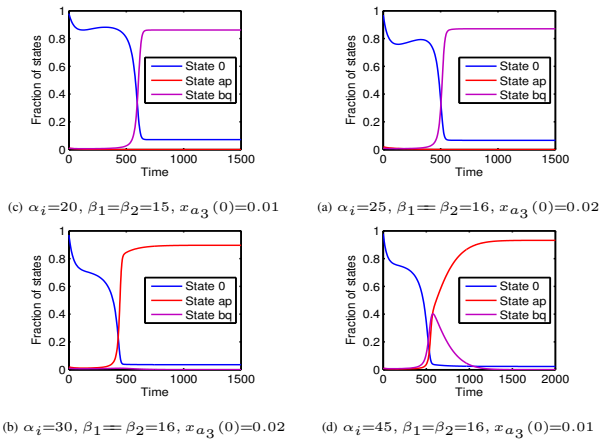
Figure 9. Numerical results: The dynamics of x_0, x_{a_1}, x_{a_2}, x_{b_1} and x_{b_2} over time where the graph is K_{5000} ,$\alpha_1 = \alpha_2 = \beta_1 = \beta_2 = 16$ and $\gamma_a = \gamma_b = 1$. (Initial fraction of nodes in different states: $x_0(0) = 0.98$, $x_{a_2}(0) = 0.01$ and $x_{b_2}(0) = 0.01$)

Impact of λ**:** λ plays an important role for source b to be dominant and it is shown in Figure 9. Suppose the fraction of nodes in state a_2 and state b_2 are equal initially. Intuitively speaking, a larger λ means nodes in state a_1 have higher probability of being changed to state b_1. From the figure, we can observe that the larger the λ is, the sooner the network reaches its equilibrium. In addition, suppose we fix $\alpha_{1,2}$ and $\beta_{1,2}$ such that at equilibrium the fraction of nodes in state a_2 is zero. In this scenario, the value of λ only has impact on the time taken to reach the equilibrium, and it has negligible impact on the fraction of nodes in different states.

Impact of $\mathcal{M}$ **and** $\mathcal{N}$**:** For multi-state model with one source, as we have discussed in Sec. II, when k (i.e. the number of steps to activate a node from state 0 increases, the threshold for γ decreases so to prevent a phase transition. In other words, if the recovery rate γ remains the same, and if we want to maximize the influence of a source, decreasing k is important.

For multi-state model with two competing sources, similar conclusion can be made. Since source b is the dominant one, we could assume that $\mathcal{N} \leq \mathcal{M}$ (i.e. it takes less phases for a node to be activated by dominant source b). For the ease of presentation, we only show the fraction of nodes in state 0, state $a_{\mathcal{M}}$ and state $b_{\mathcal{N}}$.

We conduct a set of experiments where $\mathcal{M}{=}3$ and $\mathcal{N}{=}2$. Figure 10 shows the numerical results. Figure 10 (b) shows even if $\alpha_{1,2,3}$ is larger than $\beta_{1,2}$ and $x_{a_3}(0) = 2x_{b_2}(0)$, source a may still die out. For an application of this model, assume source a and b are two products. If product b enters the market later than product a, or customers who brought product b cannot contact as much potential customers as those who brought product a, decreasing the phase for product b is *crucial*. Figure 10 (c) and (d) show that if source a takes more phases to activate nodes, it needs to have larger



(c) α_i=20, β_1=β_2=15, $x_{a_3}(0)$=0.01 (a) α_i=25, β_1=β_2=16, $x_{a_3}(0)$=0.02

(b) α_i=30, β_1=β_2=16, $x_{a_3}(0)$=0.02 (d) α_i=45, β_1=β_2=16, $x_{a_3}(0)$=0.01

Figure 10. Numerical results: The dynamics of x_0, $x_{a_{\mathcal{M}}}$ and $x_{b_{\mathcal{N}}}$ over time where the graph is K_{5000}, $\mathcal{M}=3$, $\gamma_a=\gamma_b=1$ and $\lambda_i=2$ $(1\leq i\leq \mathcal{M}-1)$. (Initial fraction of nodes in different states: $x_{b_2}(0)=0.01$)

transmission rate in order to counter the dominance of b.

VI. Related Work and Conclusion

Problem of epidemic modeling on large networks has gained considerable attention lately. It is crucial not only to gain the fundamental insights on how the disease, ideas or behavior spread, but also how products get promoted in social networks. One of the most studied epidemic models is the Susceptible-infected-susceptible (SIS) model [11], [12], [13]. A series of works focus on the analysis of SIS model in different network [14], [15], [16], [17]. Some other famous models, such as SEIR and SEIV model, that introduce the "exposed" state have also been proposed.

However, in practice, a more general SIS model is needed. For example, one "exposed" state between healthy and infectious states may not be enough for modeling disease spreading. For viral marketing, the consumer purchase decision process theory [7] suggests that there are five stages until a consumer buys a product and influences others. This motivates us to study and analyze a generalized SIS model that allows multi-susceptible states before getting infected. However, previous work cannot be easily extended on our generalized multi-state SIS model. We use mean-field technique to analyze our generalized model, and show that our methodology predicts the diffusion accurately under variety of graphs.

Recently, there is a thread of research focusing on modeling and analyzing competing process. Melnik et al. [18] proposed a model of a multi-stage complex contagion, in which agents at different stages exert different amounts of influence on their neighbors. Our work focused on the generalized SIS model, in which the phase-transition process is different from the cascade model used in [18]. Newman [19], Aspnes el al. [20], Beutel et al. [21] and Prakash et al. [22] studied the scenario that two sources are competing but their models are defined differently from ours since they did not consider intermediate stages between susceptible and infected states. Our model considers multiple susceptible states.

In our early research [23], we presented the idea of the generalized SIS model. We build on that work by analyzing more on the generalized SIS model. We also analyze two competing sources, one dominant and one regressive, under the generalized SIS model which allows multi-intermediate states. And we allow the nodes being exposed to the regressive source change to being influenced by the dominant source.

References

[1] M. E. J. Newman, "The spread of epidemic disease on networks," *Phys. Rev. E*, vol. 66, p. 016128, 2002.

[2] A. Ganesh, L. Massoulie, and D. Towsley, "The effect of network topology on the spread of epidemics," in *proc. of INFOCOM'05*, 2005.

[3] B. A. Prakash, D. Chakrabarti, M. Faloutsos, N. Valler, and C. Faloutsos, "Threshold conditions for arbitrary cascade models on arbitrary networks," in *proc. of ICDM'11*, 2011.

[4] Y. K. Lui, B. Zhao, and J. C. S. Lui, "On modeling product advertisement in large scale online social networks," *IEEE/ACM Transactions on Networking*, vol. 20, no. 5, pp. 1412–1425, 2002.

[5] S. Bhagat, A. Goyal, and L. V. S. Lakshmanan, "Maximizing product adoption in social networks," in *proc. of WSDM'12*, 2012.

[6] K. Jung, B. Y. Kim, and M. Vojnović, "Distributed ranking in networks with limited memory and communication link," in *proc. of ISIT'12*, 2012.

[7] R. D. Blackwell, P. W. Miniard, and J. F. Engel, "Consumer behavior," *South-Western College Publishing, 10th edition*, 2005.

[8] D. Chakrabarti, Y. Wang, C. Wang, J. Leskovec, and C. Faloutsos, "Epidemic thresholds in real networks," *ACM TISSEC*, vol. 10, no. 4, 2008.

[9] F. Chung and L. Lu, "Connected components in random graphs with given expected degree sequence," *Ann. Combin.*, vol. 6, pp. 125–145, 2002.

[10] Y. Lin, J. C. S. Lui, K. Jung, and S. Lim, "Stochastic analysis of diffusion process in complex networks," in *Tech. Report*, 2013.

[11] R. M. Anderson, R. M. May, and B. Anderson, *Infectious diseases of humans: dynamics and control*. Wiley Online Library, 1992, vol. 28.

[12] N. T. Bailey *et al.*, *The mathematical theory of infectious diseases and its applications*. Charles Griffin & Company Ltd, 5a Crendon Street, High Wycombe, Bucks HP13 6LE., 1975.

[13] D. J. Daley, J. Gani, and J. J. M. Gani, *Epidemic modelling: an introduction*. Cambridge University Press, 2001, vol. 15.

[14] V. M. Eguiluz and K. Klemm, "Epidemic threshold in structured scale-free networks," *Physical Review Letters*, vol. 89, no. 10, p. 108701, 2002.

[15] T. Gross, C. J. D. DLima, and B. Blasius, "Epidemic dynamics on an adaptive network," *Physical review letters*, vol. 96, no. 20, p. 208701, 2006.

[16] D. López-Pintado, "Diffusion in complex social networks," *Games and Economic Behavior*, vol. 62, no. 2, pp. 573–590, 2008.

[17] R. Parshani, S. Carmi, and S. Havlin, "Epidemic threshold for the susceptible-infectious-susceptible model on random networks," *Physical review letters*, vol. 104, no. 25, p. 258701, 2010.

[18] S. Melnik, J. A. Ward, J. P. Gleeson, and M. A. Porter, "Multi-stage complex contagions," *Chaos: An Interdisciplinary Journal of Nonlinear Science*, vol. 23, no. 1, pp. 013 124–013 124, 2013.

[19] M. E. Newman, "Threshold effects for two pathogens spreading on a network," *Physical review letters*, vol. 95, no. 10, p. 108701, 2005.

[20] J. Aspnes, N. Rustagi, and J. Saia, "Worm versus alert: Who wins in a battle for control of a large-scale network?" in *Principles of Distributed Systems*. Springer, 2007, pp. 443–456.

[21] A. Beutel, B. A. Prakash, R. Rosenfeld, and C. Faloutsos, "Interacting viruses in networks: can both survive?" in *Proceedings of the 18th ACM SIGKDD international conference on Knowledge discovery and data mining*. ACM, 2012, pp. 426–434.

[22] B. A. Prakash, A. Beutel, R. Rosenfeld, and C. Faloutsos, "Winner takes all: competing viruses or ideas on fair-play networks," in *Proceedings of the 21st international conference on World Wide Web*. ACM, 2012, pp. 1037–1046.

[23] S. Lim, K. Jung, and J. C. Lui, "Phase transition of multi-state diffusion process in networks." MAMA2013.