Frontiers

The dynamics of epidemic spreading on signed networks

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ABSTRACT

Over the past two decades, epidemic spreading on complex network has been a vibrant and highly successful research avenue. The dynamics of epidemic spreading on signed networks has nonetheless received fairly little attention. Signed networks contain edges that are labeled as either positive or negative, in relation to their propensity to either accelerate or mitigate epidemic spreading. To that effect, we here propose a modified signed-susceptible-infectious-susceptible epidemiological model, which incorporates positive and negative transmission rates based on structural balance theory. We also consider dynamical transmission rates to determine the influence of structural balance on the dynamics of epidemic spreading. We use Erdős-Rényi random networks and Barabási-Albert scale-free networks, together with the Monte Carlo method, to determine the peak fraction of infected nodes and the epidemic thresholds. We also use the mean field analysis to show analytically the origin of the computationally obtained results, although of course the agreement is not perfect due to the impact of network structure.

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1. Introduction

Epidemic spreading in the complex networks has drawn wide attention from academic community. The epidemic spreading models have been applied in the propagations processes, such as epidemiological research, computer virus propagation and information transmission [1–5]. Pastor-Satorras and Vespignani [6] proposed a classical model that considers the degree of heterogeneity and approximates epidemic spreading in networks using a power-law degree distribution [3]. Faryad et al. showed that how the epidemic threshold changes with fixed infection strength adopting the spectral algorithm as a result of being coupled with another network [7]. Piet Van Mieghem proposed the N-intertwined virus spread model, which can flexibly extend the model to the entire heterogeneous setting and obtain many insights hidden in the exact Markov model [8]. Moreover, E. Cator transferred the whole analytic machinery of the N-intertwined mean-field approximation to the generalized SIS model and established the criterion to compute the epidemic threshold [9]. Motivated by [9], N-Intertwined Mean-Field Approximation [8] is proposed to scrutinize the network topology's effect on epidemic spreading, which simulates the epidemic dynamics using susceptible-infected-susceptible (SIS) processes.

Moving beyond networks with a single layer, recently a focus on epidemiological performance in interconnected networks [10] also caught researchers' attention; in this approach, different network layers have different infection rates [11,12]. Considering the classical SIS model, we assume homogenous infection and recovery rates; so, each node in the network's infection and recovery rates is the same. Only a few recent papers have considered how heterogeneous recovery and infection rates affect epidemic spreading. To investigate network epidemiology, researchers explored many network metrics, including the betweenness centrality [13]. In addition, correlations between network metrics have been studied for identifying a representative set of metrics in the past few years [14,15].

The research mentioned above focuses on epidemic spreading in networks, in which infections are accelerated by all the edges (such as friends, whether trusted or allies). However, there are negative relations that may inhibit epidemic spreading (such as rivals, whether distrusted or competitors) [16–20]. Generally speaking, a network with both relationships is called a signed network, where we mark a positive or negative sign on each edge—for example, two entities' partnership or competition [21–23]. An example is shown in Fig. 1. We have seen wide use of signed networks to model social networks, and in recent years, a throng

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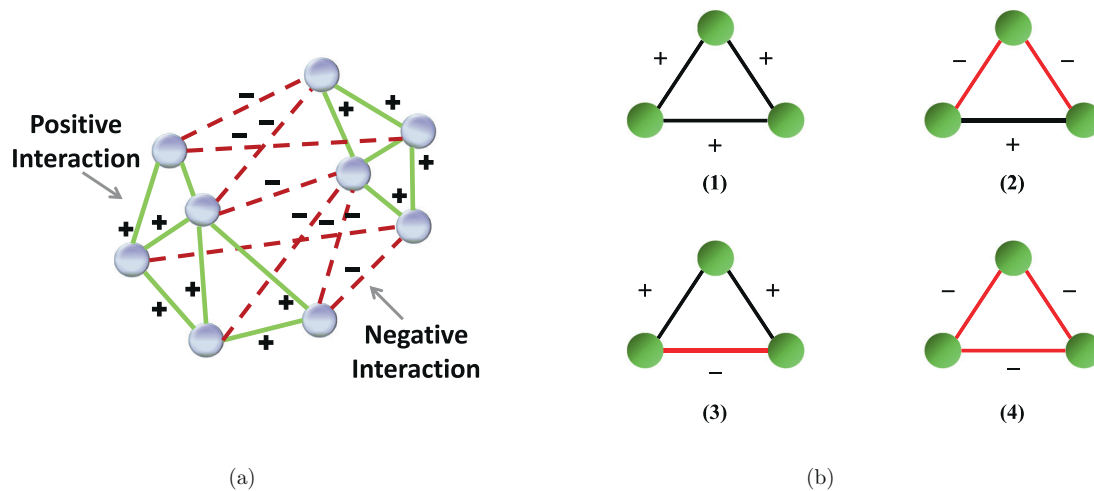 E-mail address: matjaz.perc@gmail.com (M. Perc).

Fig. 1. (a) An example of signed network. (b) The illustration of triangular structure balance. Here, (1) and (2) shows balanced triangles, while (3) and (4) shows unbalanced triangles. In each graph, the lines with '+' denote positive interactions, while the lines with '-' denote negative interactions.

of researchers focused their studies on signed networks. Antal et al. note that—regarding the theory of structural balance (as a concept)–to achieve social balance, interpersonal relationships develop in signed networks [24]. Ernesto Estrada [25] also analyzed the opinion spreading under the structural balance, which showed that negative edges played a key role in both the structure and dynamics of the network. Kunegis et al. also demonstrated that a signed network's Laplacian matrix is always a positive-semidefinite; and a signed network will only change to a positive-definite if it becomes unbalanced (i.e., it undergoes a period where it contains an odd number of negative edges) [26]. To predict the sign of each edge in signed networks, Leskovec et al. considered underlying ideas on how to determine signs within large social networks; their work yielded an approach with high prediction accuracy [22].

Although some research on epidemic spreading has been applied on signed networks, they rarely focus on the dynamics of "signed" topology and how they influence the epidemic. Thus, this paper introduces the Signed-SIS (denoted by S-SIS) epidemic spreading model and employs the Mean Field Analysis (denoted by MFA) to explore the spreading dynamics on signed networks. Specially, we propose a new Positive/Negative transmission rate based on structural balance theory, which is consistent with the real world. For a theoretical analysis of the S-SIS model, we study the dynamical infection rates rather than the usual constant infection rates to capture the potential influence of structural balance on the spreading behaviour. To verify our analysis, we apply the S-SIS model on Erdős-Rényi random network and scale-free network, particularly we explore the influence of the positive/negative edge density and degree correlation. Finally, the Monte Carlo simulation is utilized to verify our framework, especially on the infected node fraction and epidemic threshold in a metastable state. As a satisfactory theoretical tool, the results show that Mean Field Analysis approximates Monte Carlo simulation quite well.

2. The model

In this section, we propose the original Signed-SIS (S-SIS) model, which is used to study the epidemic spreading's dynamics in signed networks. Specially, we propose a new Positive/Negative transmission rate based on structural balance theory and study the dynamical infection rates rather than use a constant parameter to capture the potential influence of structural balance on the spreading behaviour.

2.1. Signed network and structural balance

Let's consider a signed, undirected graph $G = \{V, E, J\}$ to represent the signed network, where V is the set of nodes; $E \subseteq V \times V$ is the set of edges, and J is set of signs which illustrate the relationship between nodes. There are three types of signed relationships: (1) If $J_{ij} = +1$, the relationship between nodes i and j is friendly; (2) If $J_{ij} = -1$, the relationship between nodes i and j is a hostile relationship; (3) If $J_{ij} = 0$, there is no relationship between nodes i and j.

In signed networks, it is necessary to consider the network structure balance state, which was proposed by Heider [27] and improved by Cartwright et al. [28]. The theory of structural balance indicates that the balance among three different individuals can create the relationship of friends or enemies. From the social and psychological point of view, one can assume intuitively that there are four types of relationships: (1) a friend of my friend is my friend; (2) an enemy of my friend is my enemy; (3) a friend of my enemy is my enemy; (4) an enemy of my enemy is my friend. Nevertheless, in fact, it is natural to find that the relationship among three individuals can be expressed as an undirected signed network, in which the edge is marked as a symbol or sign, as shown in Fig. 1b.

2.2. Positive/negative transmission rate

In many existing social studies, friendly relationships positively spread their influence. When such friendly relationships are relatively straightforward, people are more inclined to trust and accept the opinions of their friends [27]. This situation is following the intuitive definition of the balanced structure. On the contrary, according to [28], people often oppose specific policies, not because they disagree with those policies, but there is disharmony or even conflict among their friends when voting. Furthermore, this disharmony or conflict, i.e., the unbalanced structure, makes him more likely to accept negative opinions. Therefore, based on the above conclusions, we can determine the transmission rate of opinions according to the degree of triangular balance of the positive/negative (+/-) edges in the signed network.

According to the above conclusions, we have the following definitions:

(1) The transmission rate of a positive edge is defined as the proportion of balanced triangles it participated. If a positive edge
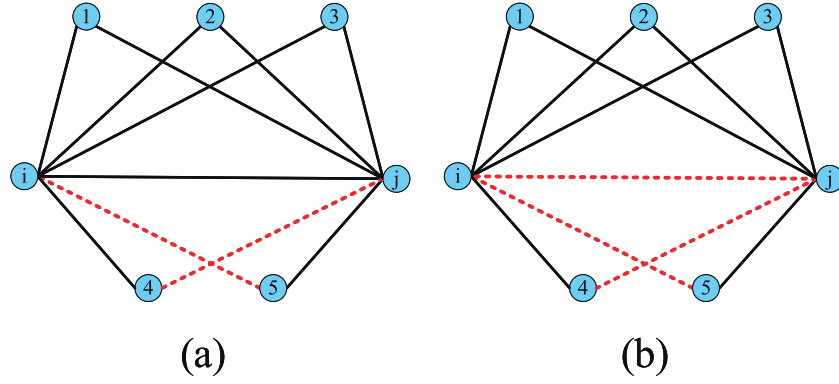
Fig. 2. The illustrations of calculating β_{ij}^+ and β_{ij}^-. Here, the solid black lines indicate positive relationships, and the dashed red lines indicate negative relationships. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

participates in more balanced triangles, the transmission rate of a positive edge is more significant;

(2) Similarly, a negative edge transmission rate is defined as the proportion of unbalanced triangles it participated. If a negative edge participates in more unbalanced triangles, the transmission rate of negative opinions is more significant.

According to the Heider's balance theory [27], a balanced structure is achieved if the sign of all triangles of the network is positive, where the sign of a triangle (i, j, k) is defined as $J_{ij}J_{jk}J_{ki}$. In the theoretical view, the balanced or unbalanced status of a triangle (i, j, k) is due to the energy contribution $H_s = -J_{ij}J_{jk}J_{ki}$, as $H_s = +1(-1)$ represents that the triangle has an unbalanced (a balanced) edge configuration. Based on this theory, we can define the transmission rate of positive edge β_{ij}^+ and negative edge β_{ij}^- as

$$\beta_{ij}^+ = \frac{\sum_{k=1}^{N} \max\{0, J_{ij}J_{jk}J_{ki}\}}{\sum_{k=1}^{n} |J_{ij}J_{jk}J_{ki}|}; \tag{1}$$

and

$$\beta_{ij}^- = \frac{\sum_{k=1}^{N} \min\{0, J_{ij}J_{jk}J_{ki}\}}{\sum_{k=1}^{n} |J_{ij}J_{jk}J_{ki}|}. \tag{2}$$

To illustrate the calculation of β_{ij}^+ and β_{ij}^-, we show two small examples in Fig. 2. In this figure, the solid black lines indicate positive relationships, and the dashed red lines indicate negative relationships. For example (a), the positive edge between i and j participates in forming three balanced triangles (i.e. $(i, 1, j)$, $(i, 2, j)$, $(i, 3, j)$) and two unbalanced triangles (i.e. $(i, 4, j)$, $(i, 5, j)$). Thus, based on Eq. (1) and Eq. (2), there are $\beta_{ij}^+ = \frac{3}{5}$ and $\beta_{ij}^- = \frac{2}{5}$. For example (b), the negative edge between i and j participates in forming two balanced triangles (i.e. $(i, 4, j)$, $(i, 5, j)$) and three unbalanced triangles (i.e. $(i, 1, j)$, $(i, 2, j)$, $(i, 3, j)$). Thus, there are $\beta_{ij}^+ = \frac{2}{5}$ and $\beta_{ij}^- = \frac{3}{5}$.

2.3. The signed-SIS model

Our Signed-SIS (S-SIS) model concentrates on results for the following real-world scenario: For a business's network, there could be a positive edge (cooperative), negative edge (competitive), or unconnected (unrelated) relationship between two companies. Typically, a company will adopt a new technique to improve its own business if its partners or competitors successfully employed this technique. To simplify the calculation process, the competition or partnership between any two nodes does not change over time.

With this in mind, here we propose the S-SIS model. The signed network is composed of a set of $V = \{v_1, v_2, \ldots, v_N\}$ of N nodes and a set of positive edges and negative edges which the adjacency matrix is A and B. Rather than using a constant infection rate γ, which

is constant in SIS, in S-SIS, we apply $\xi_i(t)$ as the dynamic infection rate for each node. This infection status of neighbors at time t can determine the infection rate of node i during time $(t + \Delta t)$. The probability that node i infected through a positive edge at time t is defined as

$$\xi_i^+(t) = \gamma \left(\frac{\sum_{j=1}^{N} \beta_{ij}^+ X_j}{d_i^+} \right), \tag{3}$$

where β_{ij}^+ represents the transmission rate of positive edge ij, X_j denotes the infection state of node j (where 0 means susceptible, but 1 means infected), and d_i^+ represents i's positive degree. Likewise, the probability that node i infected through a negative edge at time t is defined as

$$\xi_i^-(t) = \gamma \left(\frac{\sum_{j=1}^{N} \beta_{ij}^- X_j}{d_i^-} \right), \tag{4}$$

where β_{ij}^- represent the transmission rate of negative edge ij, and d_i^- represents i's negative degree.

Based on Eqs. (3) and (4)' definitions, one can find that $\xi_i^+(t)/\xi_i^-(t) = \gamma$ is the maximum rate at which the positive and negative edges can infect one node. Moreover, $\xi_i^+(t)$ and $\xi_i^-(t)$ are proportional to β_{ij}^+ and β_{ij}^-, which also proportional to the number of balanced and unbalanced triangles it involves. This is why we can use the S-SIS model to study the influence of structural balance on the epidemic spreading.

2.4. Mean field analysis

For a theoretical analysis of the S-SIS model, we employ the Mean Field Analysis (denoted by MFA) inspired by Ref[8,29]. MFA can be used to compute the infection probability $v_i(t)$ of node i at any time t, even during a metastable state. According to the probabilities of being infected of each node, we can solve the metastable fraction of infection ρ. We also use MFA to obtain the epidemic threshold τ_c in signed networks.

The following is the governing equation of MFA in our S-SIS model at node i:

$$\frac{dv_i(t)}{dt} = -v_i(t)\delta + (1 - v_i(t)) \left[\gamma \left(\frac{\sum_{j=1}^{N} \beta_{ij}^+ X_j}{d_i^+} \right) \sum_{j=1}^{N} a_{ij} v_j(t) + \gamma \left(\frac{\sum_{j=1}^{N} \beta_{ij}^- X_j}{d_i^-} \right) \sum_{j=1}^{N} b_{ij} v_j(t) \right]. \tag{5}$$

Next, we explain the terms of the equation:

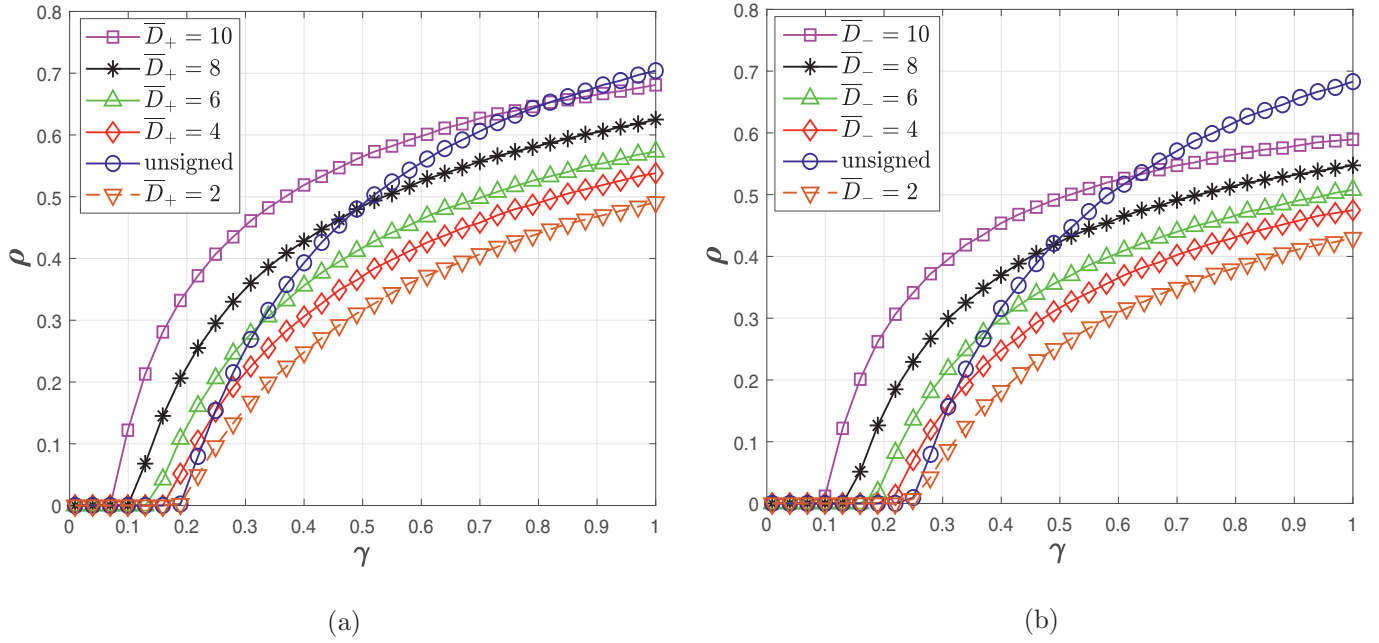- **Term** $(-v_i(t)\delta)$: At a rate of δ, a node recovers when its i is infected with probability $v_i(t)$.

Fig. 3. The metastable fractions of infected nodes ρ corresponds to infection rates γ with different (a) positive and (b) negative degrees on Erdös-Rényi signed networks.
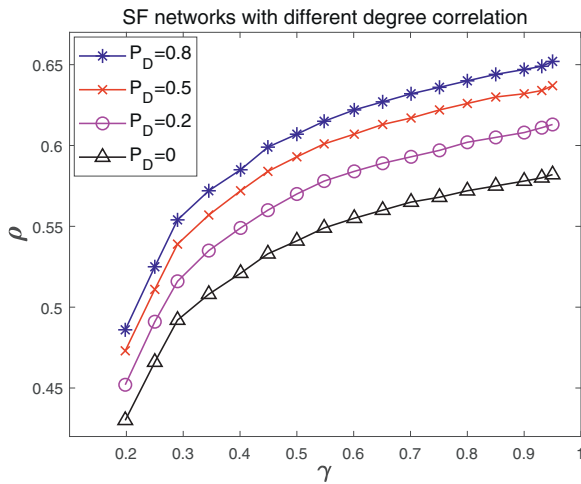

Fig. 4. The metastable fractions of infected nodes ρ corresponds to infection rates γ with different degree correlation p_D on scale-free signed networks.

- **Term** $(1 - v_i(t)) \left[\gamma \left(\frac{\sum_{j=1}^{N} \beta_{ij}^+ X_j}{d_i^+} \right) \sum_{j=1}^{N} a_{ij} v_j(t) \right]$: When the probability of node i in healthy is $1 - v_i(t)$, the rate that the node might be infected by any of its infected positive neighbors is $\gamma \left(\frac{\sum_{j=1}^{N} \beta_{ij}^+ X_j}{d_i^+} \right)$. We denote the likelihood of node i's positive neighbors infecting the node at time t as $\sum_{j=1}^{N} a_{ij} v_j(t)$, where a_{ij} is an element in the positive network's adjacency matrix (denoted as A).

- **Term** $(1 - v_i(t)) \left[\gamma \left(\frac{\sum_{j=1}^{N} \beta_{ij}^- X_j}{d_i^-} \right) \sum_{j=1}^{N} b_{ij} v_j(t) \right]$: When the probability of node i being healthy is $1 - v_i(t)$, the node might be affected by each negative neighbors, which is impacted by the $\gamma \left(\frac{\sum_{j=1}^{N} \beta_{ij}^- X_j}{d_i^-} \right)$ rate, where b_{ij} represents the adjacency matrix's element in a negative network (denoted as B). $\sum_{j=1}^{N} b_{ij} v_j(t)$

gives node i's likelihood of becoming infected at time t by negative neighbors.

Specially, the metastable state of the epidemic spreading is of significant interest to us, which can reach convergence quickly and keep it for an extremely long time [30]. Because the initial condition doesn't affect a Markov chain's metastable state [30], the initial value of Eq. (5) could be any value between 0 and 1. When we implement MFA, we choose 0.5 as the initial values $v_i(0)$ for all nodes. For a network, we see that the final solution $V(t)$ consists of N elements, and we calculate the metastable fraction of infection ρ as the average of $V(t)$ using the following Eq. (6)

$$\rho = \frac{\sum_{i=1}^{N} v_{i\infty}}{N}. \tag{6}$$

3. Simulation results

In this section, we apply the S-SIS model on signed Erdős-Rényi random networks and signed Scale-Free networks. We explore the S-SIS model's epidemic prevalence under two control constraints–namely, the average positive/negative degree $\overline{D}_+/\overline{D}_-$ on signed ER networks and degree correlation probability p_D on signed SF networks. We mainly focus on exploring the effect of these control constraints on the epidemic threshold τ_c and the fraction of infected nodes ρ in a metastable state. Finally, we compare the performance of the Mean Field Analysis with Monte Carlo simulation to verify our analysis.

3.1. The S-SIS model on signed erdös-Rényi random networks

Here, we study the S-SIS model's properties on signed Erdős-Rényi (ER) random networks and concentrate on how the average positive/negative degree $\overline{D}_+/\overline{D}_-$ affect the overall spread of epidemics. The results are shown in Fig. 3, in which the metastable fractions of infected nodes ρ correspond to different infection rates γ. One can notice in Fig. 3 that all signed networks' epidemic thresholds are smaller than the unsigned network; and as $\overline{D}_+/\overline{D}_-$ increases, the threshold decreases.

We interpret this phenomenon analytically: The epidemic threshold is equal approximately to the network spectral radius's
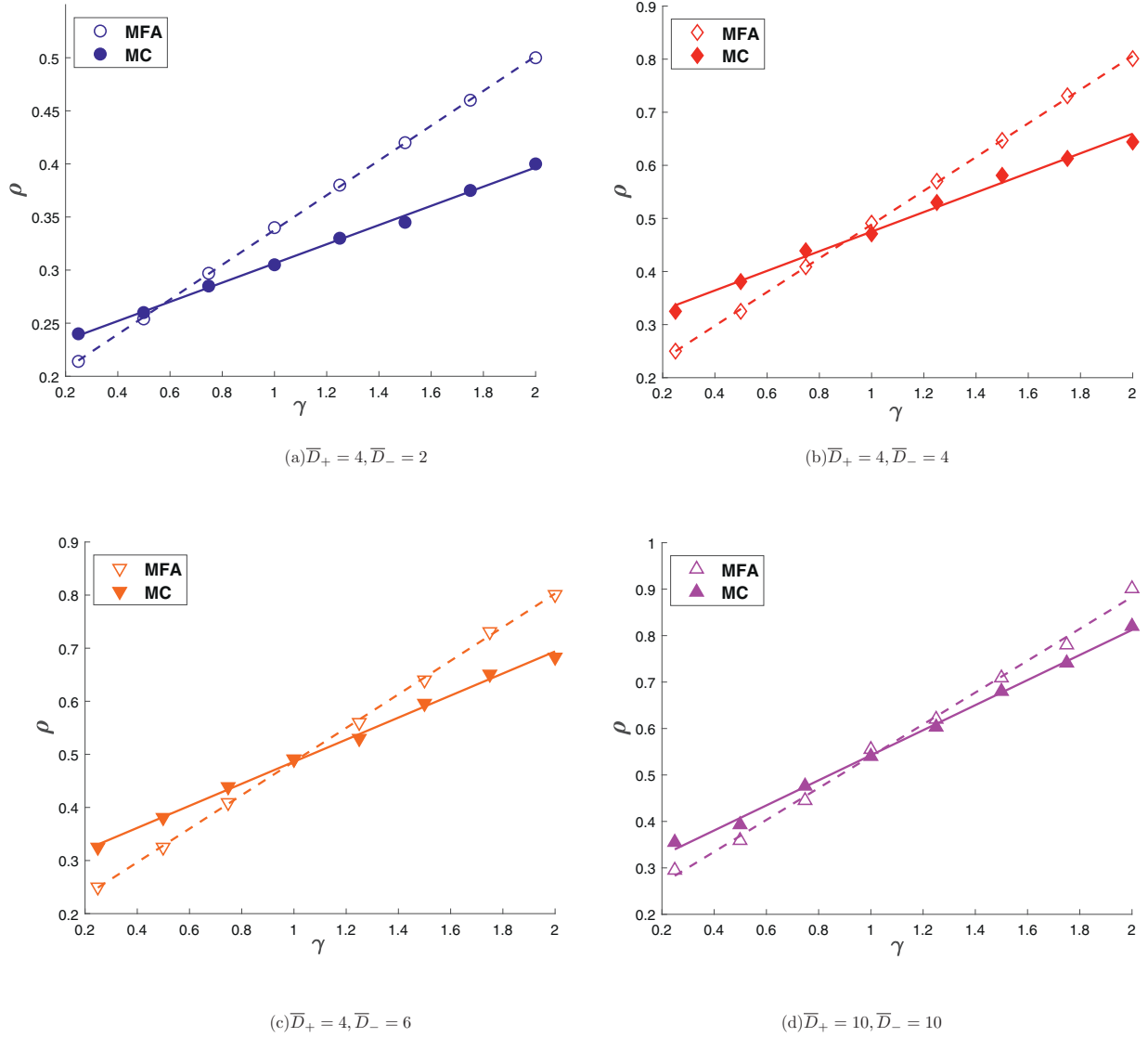
(a) $\overline{D}_+ = 4, \overline{D}_- = 2$

(b) $\overline{D}_+ = 4, \overline{D}_- = 4$

(c) $\overline{D}_+ = 4, \overline{D}_- = 6$

(d) $\overline{D}_+ = 10, \overline{D}_- = 10$

Fig. 5. The metastable fractions of infected nodes ρ corresponds to infection rates γ with different positive and negative degrees from (a) to (d) on Erdös-Rényi signed networks. The solid markers indicate MC simulation results, while the hollow markers are MFA approximation results. We depict linear fitting line for the simulation results using dotted lines.

reciprocal, in which we define the spectral radius as an adjacency matrix's largest eigenvalue. Because of the signed network, we consider it to be a two-layer interconnected network. Here, $\tau_c^{signed} = \frac{1}{\lambda_{\max}(A+B)}$ approximates the network's epidemic threshold, where $A + B$ represents the network's adjacency matrix [31]. We also regard the unsigned network—which has a null negative network—as a signed network. Thus, we can deduce that the epidemic threshold of an unsigned network is $\tau_c^{unsigned} = \frac{1}{\lambda_{\max}(A)} = \frac{1}{\lambda_{\max}(A+0B)}$. Based on [31]-[32], we have the following lemma:

$$\lambda_{max}(A) \leq \lambda_{max}(A + B) \leq \lambda_{max}(A) + \lambda_{max}(B). \tag{7}$$

It follows, then, that $\tau_c^{signed} \leq \tau_c^{unsigned}$ if negative adjacent matrix B is not empty. Moreover, we find that if $\overline{D}_+$ unchanged, with the increase of $\overline{D}_-$, τ_c^{signed} will decrease. This is because with the increase of the number of edges, τ_c will rapidly converge to 0 logarithmically in ER networks [33]. Since $\overline{D} = \frac{2L}{N}$, one can find that increasing the number of negative edges leads a higher value of $\overline{D}_-$.

Comparing with other signed networks, we notice that signed networks with denser negative edges (i.e., $\overline{D}_-$) have larger ρ at the

same infection rate in a metastable state. According to our analysis, we can also explain our results as follows. Based on Eq. (3) and Eq. (4), the infection rate ξ_i^+ and ξ_i^- of node i are proportionate to transmission rate β_{ij}^+ and β_{ij}^-, respectively. With the increasing number of negative edges, nodes will have more unbalanced triangles since the positive edges $\overline{D}_+$ is unchanged. The increase of $\overline{D}_-$ will increase the number of unbalanced triangles and does not destroy the existing balanced triangles. This will increase the negative transmission rate ξ_i^-, and result in a larger infection proportion ρ.

To prove our findings, we deduce the theoretical result of $v_{i\infty}$ in the MFA form. To simplify the proof, we use a completely mixed assumption [1]. That is to say, at any time t, all nodes have a roughly similar positive degree ($\overline{D}_+$) and negative degree ($\overline{D}_-$). Furthermore, each node has the same proportion of balanced and unbalanced triangles, and we simplify the β_i^+ and β_i^- as ξ^+ and ξ^-, respectively.

According to this assumption, we rewrite Eq. (5) as

$$\frac{dv_i(t)}{dt} = (1 - v_i(t))[\gamma \cdot \xi^+ \cdot v_i(t) \cdot \overline{D}_+ \cdot v_i(t) + \gamma \cdot \xi^- \cdot v_i(t) \cdot \overline{D}_- \cdot v_i(t)] - \delta v_i(t). \tag{8}$$

(a)SF networks $P_D = 0.2$ (b)SF networks $P_D = 0.6$
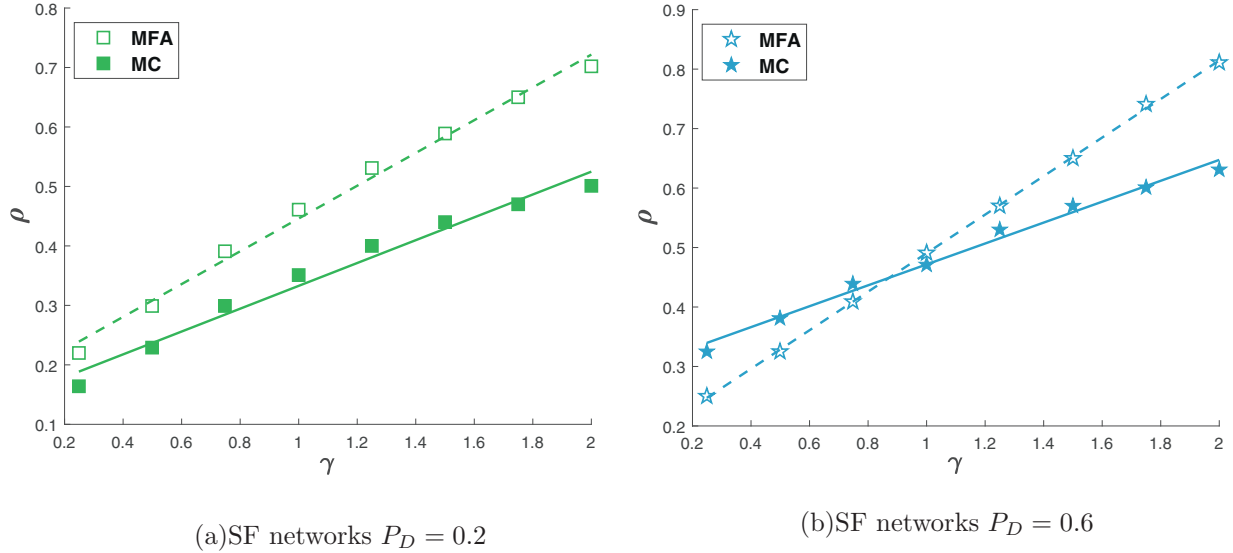
Fig. 6. The metastable fractions of infected nodes ρ corresponds to infection rates γ with (a) $P_D = 0.2$ and (b) $P_D = 0.6$ on scale-free signed networks. The solid markers indicate MC simulation results, while the hollow markers are MFA approximation results. We depict linear fitting line for the simulation results using dotted lines.

In a metastable state, we have $\frac{dv_{i\infty}}{dt} = 0$ and we can write Eq. (8) as

$$\delta v_{i\infty} = (1 - v_{i\infty}) \cdot v_{i\infty}^2 \cdot \gamma (\xi^+ \cdot \overline{D}_+ + \xi^- \cdot \overline{D}_-). \tag{9}$$

Let $\delta = 1$. Furthermore, let us simplify Eq. (9) by omitting $v_{i\infty} = 0$ (representing the state of absorption) as the trivial solution:

$$(1 - v_{i\infty}) \cdot v_{i\infty} = \frac{1}{\gamma (\xi^+ \cdot \overline{D}_+ + \xi^- \cdot \overline{D}_-)}. \tag{10}$$

Considering that $v_{i\infty} \leq 1$, we give the solution of Eq. (9) as

$$v_{i\infty} = \sqrt{\frac{1}{4} - \frac{1}{\gamma (\xi^+ \cdot \overline{D}_+ + \xi^- \cdot \overline{D}_-)}} + \frac{1}{2}. \tag{11}$$

As Eq. (11) shows, when all other parameters remain the same, $v_{i\infty}$ rises up in signed ER networks with the increase of $\overline{D}_-$ and $\overline{D}_+$. Moreover, the larger value of transmission rate ξ^+ and ξ^- will also lead to a greater value of $v_{i\infty}$. So as $\rho = \frac{\sum_{i=1}^{N} v_{i\infty}}{N}$ demonstrates, the increase of negative edges (positive edges) may facilitate epidemics' spread in signed ER networks when the number of positive edges (negative edges) remains constant. In conclusion, we can find that this corresponds to the simulation results in Fig. 3.

3.2. The S-SIS model on signed scale-Free networks

Next, we study the performance of the S-SIS model in signed Scale-Free (SF) networks. Since the construction of SF networks does not use the properties $\overline{D}_+$ and $\overline{D}_-$, in this part, we focus on the signed SF networks that are degree-correlated, where node i's positive degree d_i^+ relates to its negative-degree d_i^-. We develop a method for creating signed SF networks that are degree-correlated and investigate how the degree correlation p_D influences τ_c (the epidemic threshold) and ρ (the metastable fraction of infected nodes).

We first introduce our method for creating signed SF networks that are degree-correlated: (1) First, we generate the positive network use the ordinary Barabási-Albert Scale-free network model [34]. Here, the network size is $N = 1000$, and the connectivity exponent is 2.5 within our simulations. (2) Next, we generate the negative network by copying the positive network's degree sequence. (3) Given p_D (the degree-correlation), a p_D fraction of nodes are selected randomly, where the negative and positive degrees are kept the same. Subsequently, we shuffle the remaining

degree-sequence elements and randomly reallocate the remaining $1 - p_D$ fraction's negative degrees of nodes. (4) We repeat step (1) to create the negative network.

In Fig. 4, we notice that when degree-correlated p_D is larger, the metastable fraction of infected nodes in signed SF networks is also higher. This means we enhance the degree-correlation that may facilitate the epidemic spreading in our framework. These observations may be interpreted as follows. As we mentioned, generally speaking, if p_D is large, a hub node in a negative network will have a large positive degree. Thus, when p_D is large, the likelihood of a hub node linked by positive edges and becomes infected in a negative network is larger than when p_D is small. Since hubs have many neighbours, they still can facilitate many other nodes having higher infection rates, resulting in a higher metastable fraction of infected nodes at larger p_D.

3.3. Monte carlo simulations

To further verify the effectiveness of our analysis, we designed another method, i.e., Monte Carlo simulations with discrete-time [30], to approximate the S-SIS model's epidemic processes. In the MC simulation, each final result has at least 200 implementations on average. We apply $\Delta t = 0.01s$ as the sample time when $\gamma < 1$ (indicating each time step in the simulation is 0.01 seconds in the real world), and $\Delta t = 0.001s$ when $\gamma > 1$. At first, we set a random selection of 10% infected nodes for time $t = 0$. Then the probability is $\Delta t \cdot \xi_i^+$ at each time step that the infected positive neighbour may infect any healthy node i, or $\Delta t \cdot \xi_i^-$ that the infected negative neighbour may infect the healthy node i. We calculate ξ_i^+ and ξ_i^- based on Eqs. (3) and (4), respectively, along with the recovery probability at each time step of each infected node $\Delta t \cdot \delta$. To calculate an accurate metastable fraction of infection ρ, we run a large enough number of time steps. For every 20 time steps, we record ρ and check the difference between adjacent records. We know that we have reached the metastable state once the procedures check the difference for less than a predetermined value at least five times consecutively.

Fig. 5 shows the simulation results of both MC's and MFA method on ER networks, which illustrates the linear fitting of γ^* with a metastable fraction of infection ρ. One can find that MFA approximating MC simulation well in all different values of $\overline{D}_+$ and $\overline{D}_-$, which verified the effectiveness of our framework. Especially,

with the increase of the density of positive and/or negative networks, i.e., increase the value of $\overline{D}_+$ or $\overline{D}_-$, we observe that the MFA fits MC simulation better. Just as shown in Fig. 5a to 5d, the linear fitting lines of MFA and MC get more close when the $\overline{D}_+$ and $\overline{D}_-$ is increasing.

Similar to signed ER networks, we also perform both MC's and MFA simulation on SF networks, and observe the influence of degree-correlation on their linear fitting. The simulation results are shown in Figs. 6a and 6b. We find that with the increase of p_D, the MFA and MC simulation fit better. Additionally, crossing points (γ^*, ρ^*) appears in Fig. 6b.

4. Conclusion

This paper proposes a novel signed epidemic spreading model (denoted as S-SIS) to study the influence of structural balance on epidemic transmission in signed networks. Specially, we propose a new Positive/Negative transmission rate based on structural balance theory, which is consistent with the real world. For a theoretical analysis of the S-SIS model, we study the dynamical infection rates rather than the usual constant infection rates to capture the potential influence of structural balance on the spreading behaviour. Finally, we develop MC simulations that observe S-SIS's performance under varying constraints and scenarios and derive an MFA method (based on solutions surrounding nodes' metastable likelihood of infection) to analyze the model theoretically. We demonstrate that MFA approximates MC simulation results quite accurately.

This work has broader application to other real-world scenarios, for instance, whether a company will opt to adopt a new technology based on its novelty and opportunities, whether competitors are adopting it, and social media rumours. Based on this scenario, we had the following observations: (1) Even if certain companies rapidly adopt and use new technology in response to competitors' use, that does not necessarily mean we will see a larger percentage of companies adopting the new technique. (2) The competitive nature of companies sometimes may result in a higher percentage of adoption than networks focused on cooperative relationships (unsigned networks), especially those with indicators of being more reticent to adopt new approaches.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgment

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