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Epidemic spreading on weighted complex networks

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ABSTRACT

Nowadays, the emergence of online services provides various multi-relation information to support the comprehensive understanding of the epidemic spreading process. In this Letter, we consider the edge weights to represent such multi-role relations. In addition, we perform detailed analysis of two representative metrics, outbreak threshold and epidemic prevalence, on SIS and SIR models. Both theoretical and simulation results find good agreements with each other. Furthermore, experiments show that, on fully mixed networks, the weight distribution on edges would not affect the epidemic results once the average weight of whole network is fixed. This work may shed some light on the in-depth understanding of epidemic spreading on multi-relation and weighted networks.

1. Introduction

Epidemic spreading based on complex networks, where nodes represent individuals and links denote their interactions, has attracted an increasing attention in recent years [1–3]. Generally, disease propagation can be modeled as a kind of dynamic process in which an item is transmitted from an infected individual to a susceptible individual via the link between them [4]. Motivated by previous pioneering works that many real networks exhibit the small-world phenomenon and scale-free property, more and more results of spreading dynamics on those networks are presented recently [5,6]. The spreading process on the scale-free network indicates that a highly heterogeneous structure would lead to both the absence of the epidemic threshold [7,8] and the hierarchical spreading of epidemic outbreak [9]. Further study of the susceptible-infected-susceptible (SIS) model on the scale-free network shows that the vanishing of epidemic threshold stems from the node with the largest degree rather than the scale-free nature [10]. More general, the epidemic threshold for SIS model on an arbitrary undirected graph is determined by the largest eigenvalue of the adjacency matrix [11,12]. On the small-world network, most infection occurs locally because of the high-level cluster and the disease spreads rapidly into large regions of the population for the short path lengths [13,14]. Analysis of the susceptible-infected-recovered model (SIR) on small-world networks presents that a phase transition between two different regimes occurs at a particular rewiring parameter p_c [15], and such critical transition is also found in the spreading on dynamical small-world networks [16]. In addition, the epidemic propagation on the real-network structure also draws much attention, such as sexually transmitted disease on the sexual contact networks [17], mobile phone viruses on the multimedia messaging systems [18], disease transmission between human beings and mosquitos [19], and so on.

However, the aforementioned researches mostly consider the simplest case of networks with only one type of links. In fact, there exist various real-world complex networks, which are characterized by inherent multi-relation connections [20–22], such as blood relationship, romantic relationship, friend relationship, work relationship in the social contact network. The role of hybrid relations in the spreading process could be very different [23]. Some disease propagation would be more likely to be promoted among family members such as the HIV, while some contagions such as H7N9 [24] are prone to transmit among the staffs in the slaughter house or chicken farm. It is obvious that with the existence of the multiple relationship, the network structure becomes more complex and diverse, leading to more special spreading dynamics. Failure cascading of the network coupled with connectivity links and dependency links [25–27] demonstrates that the network disintegrates in a form of a first-order phase transition for a high density of dependency links, whereas the network disintegrates in a second-order transition for a low density of dependency links. Though multi-relation networks attract more and more attention, it is still unclear how the multi-relationship affects the epidemic spreading dynamic for the complex network structure. It is a reasonable way to treat the multi-relation network with assigning different weights for each relation. Li et al. [28] proposed a binary-relation network model, representing colleague and friend

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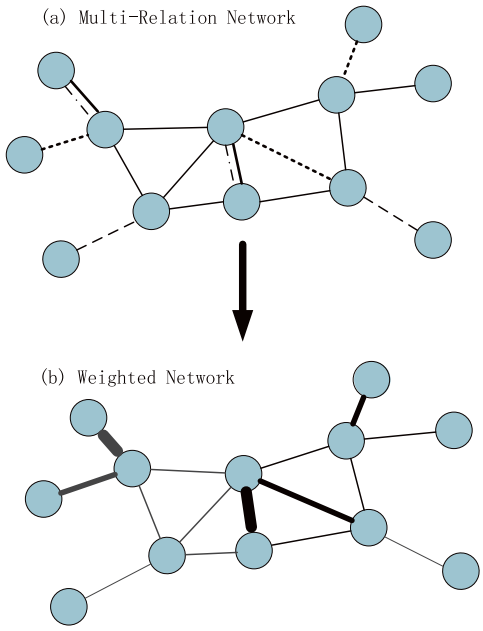

Fig. 1. (Color online.) Illustration of (a) multi-relation network where each type of line corresponds to one kind of relation; and (b) the corresponding weighted network, where the thickness of link represents the size of weight.

relationship by setting different weights of the corresponding links, and the epidemic spreading process demonstrates that the outbreak threshold is suppressed by the closer relationship.

In order to understand the epidemic spreading process on multi-relation networks in-depth, in this Letter, we construct multi-relation networks with considering various relation-levels as different weights, where link weight follows some given distributions (see Fig. 1). Then, we perform SIS and SIR models on the proposed weighted networks, where the links with the same weight shows the same transmit capacity. Focusing on the outbreak threshold and epidemic prevalence, theoretical analysis based on the mean-field approximation illustrates that multiple relations would result in the decrease of the outbreak threshold and brings more infections in the final state. Detailed analysis indicates that the epidemic spreading result just depends on the average level of relationship rather than the link weight distribution. In addition, Monte Carlo simulation agrees well with the theoretical results.

2. Model

In this Letter, we consider that there are n kinds of relations in the network where the multiple relations can be represented as the link weight. Fig. 1 shows such a illustration of a multi-relation network and its corresponding weighted network. In general, we set the links with discrete weights as $w = 1, 2, \ldots, n$ to identify each type of relationship, and the link with higher weight means closer relationship, through which disease is more likely to transmit. In order to illustrate the spreading effects of different relations, we investigate two sets of weight distributions, one of which follows uniform distribution, the other follows the Poisson distribution. In addition, we assume that all links are fully mixed and the same type of links are distributed uniformly in the network.

Consequently, we adopt SIS and SIR models on the small-world network (WS network with randomness probability $p_s = 0.3$) [29] and scale-free network (BA network) [30], respectively, where the network size is set to be $N = 10^4$, and the average degree is $\langle k\rangle = 8$. In general, we set the recovery probability $\mu = 1$, initial infected density $I_0 = 0.01$ and define the transmit probability for links with $w = 1$ as λ. We assume that transmit probability through the edge with weight w (λ_w) is equivalent to the infected probability that w infected individuals (I) simultaneously influence the susceptible individual (S) [31], which can be obtained by:

$$\lambda_w = 1 - (1-\lambda)^w. \tag{1}$$

According to the mean-field approximation, for an arbitrary edge the successful transmission probability in one timestep is:

$$\beta = \sum_w p_w\left(1 - (1-\lambda)^w\right), \tag{2}$$

where p_w is the proportion of links with weight w.

In general, λ is very small, thus Eq. (2) can be simplified to:

$$\beta \approx \alpha\lambda, \tag{3}$$

where α is the average weight of all links in the network.

3. Outbreak threshold

In order to understand the epidemic outbreak threshold with the multi-relation effect, we use a method of percolation theory, for disease spreading can be seen as a growing percolation process [32]. For the case of uncorrelated networks, the probability that an edge links to a node with degree k is $\frac{kp(k)}{\langle k\rangle}$, where $p(k)$ is the degree distribution of the observed network, and $\langle k\rangle$ is the average degree. In addition, we assume that as long as the epidemic has not spread out yet, the infected node with degree k has only one ingoing link and $k-1$ outgoing links [32]. And the average number of susceptible nodes infected by an already infected node i is:

$$\langle n_i\rangle = \beta\sum_k \frac{p(k)k(k-1)}{\langle k\rangle} + \pi, \tag{4}$$

where π is the contribution of the probability to reinfect the ancestor (the node that infected node i, corresponding to i's ingoing link) [33].

For the **SIR** model where the reinfection is forbidden, the disease spreads directionally down a tree structure and $\pi = 0$. If an infected individual infects at least one other individual on average, the epidemic can reach an endemic state. Therefore, we have $\langle n_i\rangle = 1$ at the epidemic threshold [32,33], leading to:

$$\lambda_c(\mathrm{SIR}) = \frac{\langle k\rangle}{\alpha(\langle k^2\rangle - \langle k\rangle)}. \tag{5}$$

For the **SIS** model where the reinfection is allowed, things get more complicated. We define π_t as the probability that j infects i if i has infected j yet. In this model, we set the recovery probability $\mu = 1$, which means that the infected node remains the infected state in just one step. Therefore, the interval that j remains infected and i remains susceptible is only 1, which leads to $\pi_t = \beta$. Incorporating the effect of competition between j and the other descendants of i, the reinfected probability π for the system can be calculated as following [32]:

$$\pi = \pi_t\sum_{k'=0}^{\kappa-1}\binom{\kappa-1}{k'}\frac{(\beta\pi_t)^{k'}(1-\beta\pi_t)^{\kappa-1-k'}}{k'+1}, \tag{6}$$

where $\kappa - 1 = \sum_k \frac{p(k)k(k-1)}{\langle k\rangle}$ is the branching factor that represents the average number of nodes influenced by node i and k' represents the infected neighbors of node i.

Neglect the high-order term, we can obtain that $\pi \approx \beta$. According to Eq. (4), the epidemic threshold of SIS model is:

$$\lambda_c(\mathrm{SIS}) = \frac{\langle k\rangle}{\alpha\langle k^2\rangle}. \tag{7}$$