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# Strong ties promote the epidemic prevalence in susceptible–infected–susceptible spreading dynamics



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#### HIGHLIGHTS

- Strong and weak ties play far different roles in epidemic spreading on networks.
- Strong ties promote the epidemic prevalence in networks.
- The distribution pattern of weights mainly contributes to the results.

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#### ABSTRACT

We propose a weighted susceptible–infected–susceptible model on complex networks, where the weight of an edge is defined by the topological proximity of the two associated nodes. Each infected individual is allowed to select a limited number of neighbors to contact, and a tunable parameter is introduced to control the preference to contact through high-weight or low-weight edges. Simulation results on six real networks show that the epidemic prevalence can be largely promoted when strong ties are favored. By comparing with two statistical null models, we show that the distribution pattern of weights, rather than the topological structure, mainly contributes to the observations. Further analysis suggests that the weight–weight correlation strongly affects the results: high-weight edges are more significant in keeping high epidemic prevalence when the weight–weight correlation is positive.

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#### 1. Introduction

Early before the classification of social ties was proposed, in 1954, Rapoport [1] was aware of "well-known fact that the likely contacts of two individuals who are closely acquainted tend to be more overlapping than those of two arbitrarily selected individuals". This argument became one cornerstone of social network theory. In 1973, ties in social networks, generally, come in two varieties: strong and weak, which have been first proposed by Granovetter [2]. Strong ties connect with the people you really trust, and people whose social circles tightly overlap with your own. Often, they are also the people most like you. Weak ties, conversely, connect with mere acquaintances and often provide access to novel information. Tie strength usually plays a vital role in many real networks and is crucial to understand dynamical processes on the networks [3]. Weak ties display an important bridging function [4,5], while strong ties are more likely to activate the flow of referral information and more influential than weak ties [6]. In addition, weak ties could play a more significant role than strong ties to keep

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the stability [7], maintain the connectivity [8] and uncover the missing information [9,10], while strong ties can be better utilized to enhance the human resource flexibility [11], to provide accurate recommendation [12,13], and so on.

Despite the qualitative distinction between strong and weak ties, tie strength could be quantitatively described by edge weight—the edges with high weights are considered to be strong. In a number of social networks, edges are often associated with weights that differentiate them in terms of their intensity, capacity or the frequency of recent contacts [2,14]. For technological and biological networks, weights often refer to the functions performed by edges, e.g., the amount of traffic flowing along connections in world-wide airport networks [14], the number of synapses and gap junctions in neural networks [15] and the carbon flow between species in food webs [16].

Weight plays a significant role in disparate networked dynamics, such as transportation [17], synchronization [18], percolation [19], and so on. In this paper, we concentrate on the effects of weights on epidemic spreading [20–22]. Yan et al. [23] investigated the epidemic spreading in weighted scale-free networks and showed that the more homogeneous weight distribution of the network is, the more quickly epidemic spreads on it (see also an analogous result [24] on epidemic threshold for unweighted networks). This finding was further demonstrated by an edge-based mean-field solution [25]. Chu et al. [26] showed that weight distribution has strong impacts on both epidemic threshold and prevalence. Karsai et al. [27] considered the contact process in weighted scale-free networks, in which the weight of an edge connecting two high-degree nodes is relatively small. Yang et al. [28] further showed that in the contact process, the epidemic prevalence can be maximized by setting the edge weight inversely proportional to the degree of the receiving node. Baronchelli and Pastor-Satorras [29] considered the diffusive dynamics on weighted networks and shed light on the validity of mean-field theory on weighted networks. Rattana et al. [30] proposed a pairwise-type approximation for epidemic dynamics on weighted networks. Hu et al. [31] studied the effects of distance in spreading dynamics, where the distance between two locations can be mapped to weight assigned to the corresponding link.

In this paper, we propose a weighted susceptible–infected–susceptible (SIS) epidemic spreading model, with a tunable parameter controlling the preference of spreading: whether or not an infected individual prefers to contact others through edges with high weights. Simulation results on six real networks show that the preferential contacts through strong ties could largely improve the epidemic prevalence. We compare such results with two statistical null models, where the topological structure and weight distribution are randomized respectively. The aforementioned strong tie effects are qualitatively the same under randomized topology while are vanished if the weights are randomly redistributed, indicating that the distribution pattern of weights mainly contributes to the observations. Further analysis suggests that the weight–weight correlation strongly affects the results, with the optimal value of the controlling parameter monotonously depending on the correlation strength.

#### 2. Model

The SIS model [32–35] is suitable to describe the cases when individuals cannot acquire immunity after recovering from the disease, such as influenza, pulmonary tuberculosis and gonorrhea. Here we consider a networked discrete-time SIS model, where nodes are in two discrete states, "susceptible" or "infected". In the traditional model, each infected node will contact all its neighbors once at each time step, and therefore the infectivity of each node is proportional to its degree. In the real world, individuals may be only able to contact limited neighbors within one time step [36]. For example, salesman in network marketing processes will not make referrals to all his acquaintances due to the limited money and time [37]. In sexual contact networks, although a few individuals have hundreds of sexual partners, their sexual activities are not far beyond a normal level due to the physiological limitations [38]. Therefore, in the present model we assume that every individual has the same infectivity [36,39]. Without loss of generality, at each time step, each infected node will select one of its neighbors to contact. If the selected neighbor has been infected already, nothing happens, otherwise it will be infected with probability  $\alpha$ . Meanwhile, each infected node will become susceptible in the next time step with probability  $\beta$ . In the following study, we fix  $\alpha = 0.4$  and  $\beta = 0.1$ , and we have already checked that the specific choices of  $\alpha$  and  $\beta$  will not change the qualitative results reported in this paper if the set of parameters allows a non-zero fraction of infected nodes in the stable state.

The probability that an infected node *i* will select its neighbor *j* is

$$p_{ij} = \frac{s_{ij}^{b}}{\sum_{l \in I_i} s_{ll}^{b}},$$
(1)

where  $\Gamma_i$  is the set of neighbors of node *i*,  $s_{ij}$  denotes the tie strength between *i* and *j*, and *b* is a tunable parameter. If b = 0, the infected node randomly selects a neighbor to contact, equivalent to the unweighted SIS model [39]. If b > 0, strong ties are favored to constitute the paths of spreading, while if b < 0, weak ties are favored. According to Eq. (1), the infection rates among neighbors are generally inhomogeneous. Some recent works [42–46] consider a related problem where the infection rate (characterized by the distribution of infection times) to each neighbor is inhomogeneous, and shows remarkable impacts of such temporal correlation of contacts on the epidemic threshold and spreading speed. In these works, the impacts of a class of links are ignored, while this paper emphasizes the specific role played by the strong ties.

In different contexts, the strength of a tie may have different definitions and measures [10,47], which may depend on external information to network topology. For general networks, one may be not aware of external information and thus it

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