THRESHOLD OF SIS EPIDEMICS IN ALTERNATE SOCIAL NETWORKS

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We introduce the alternate social networks (ASN) model to study the epidemic threshold of SIS epidemic. The alternate social networks consist of a family network and a public network, mimicking the human contacts during some time (*e.g.* nighttime) and other time (*e.g.* daytime) respectively. Both the family network and the public network are constructed by a set of sub networks which can exhibit small-world properties, scale-free degree distribution or the household structure, representing various types of local interactions among social groups in modern society. Simulations show that the ASN has the essential characteristics of social networks, and the local fully connected structures (households) as well as the existence of local structures (publics) in the public network are two dominating ingredients for the epidemic threshold. Moreover, results show that the epidemic threshold in ASN is independent of the initial condition and the system size.

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1. Introduction

Classical mathematical models for the spread of a disease often ignore the patterns and structures of social and spatial interactions within a population [1]. However, it has been found recently that many social and natural systems share some important organising principles uncovered in the framework of complex network research [2–7], where the vertices represent the elements and the edges pairwise interactions between elements. They typically exhibit two distinct properties, the scale-free (SF) connectivity distributions [8] and the small-world (SW) properties [9]. These efforts dedicated

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to the structure of the networks help us understand the dynamics of the networks, which in its turn have a strong impact on the dynamics on the networks. Particularly interesting example where the interaction network plays capital role in the dynamics is the case of disease spreading. The extensive study of network-based models of epidemic spreading has given us valuable insights of how diseases spread [10–16].

The concept of thresholds, which is of important guiding significance to the disease eradication and vaccination, is a central part of the theoretical study of infectious diseases. In mathematical epidemiology [1], the epidemic threshold is a standard element, responsible for many of the standard conclusions drawn in epidemic modelling. Recently, the investigation of the susceptible-infected-susceptible (SIS) model on random SF networks find the absence of an epidemic threshold and its associated critical behaviour, which implies that random SF networks are prone to the spreading and the persistence of infections at whatever spreading rate the epidemic agents possess [17]. The clustering in random SF networks cannot restore a finite epidemic threshold [18]. Actually, it has been found that a SF degree distribution $P(k) \sim k^{-\gamma}$ with $2 < \gamma \leq 3$ in unstructured networks with assortative or disassortative mixing is a sufficient condition for a null epidemic threshold in the thermodynamic limit, and the degree correlations result therefore irrelevant for the epidemic spreading picture in these SF networks [19]. It has also been shown that for finite-size networks, finite thresholds for the spreading of an epidemic are always found [20, 21]. Moreover, research indicates that epidemic propagation depends equally on the infection scheme as well as the network structure, which says connectivity-dependent infection schemes can yield threshold effects even in scale-free networks where they would otherwise be unexpected [22]. Comparing this with the absence of a finite threshold in networks with purely random wiring, the study of SIS epidemic model in highly clustered SF networks suggests that high clustering (modularity) and degree correlations protect SF networks against the spreading of viruses, which means the existence of a finite epidemic threshold in these networks in the limit of infinite system size [23].

However, most of these investigations have been performed on the so-called scale-free networks, where some important factors, such as network hierarchy and the network evolution [11–16] are ignored, which have important influence on the epidemic threshold. In modern society, daily human activities are marked by strong habits with little day-to-day variety [24]. For instance, students will go to school in mornings, frequently contact with their classmates and teachers, and stay home with their parents after school; adults will go to their workplaces on time and stay there until off duty. In order to account for these interaction patterns, researchers [25–32] have paid increasing attention to the influence of the local structures in social networks

on the epidemic spreading. Others [33, 34] considered the case that a common set of nodes belong to two different networks. But in these work the influence of the household structure and the network hierarchy on the epidemic threshold has not been investigated yet. In this paper, we introduce the alternate social networks to model the potentially infective contacts, and we adopt the SIS model as a description of epidemic spreading in a closed finite population, focusing on the study of epidemic threshold in the alternate social networks. Studies show that the ASN has the essential characteristics of social networks, especially the local fully connected structures (household) and the high clustering coefficient, which were found to be two dominating ingredients for the epidemic threshold.

2. The model

The alternate social networks model (ASN) is depicted in Fig. 1, which consists of two sub models: the public network model (PN) and the family network model (FN). We consider a closed finite population of N individ-

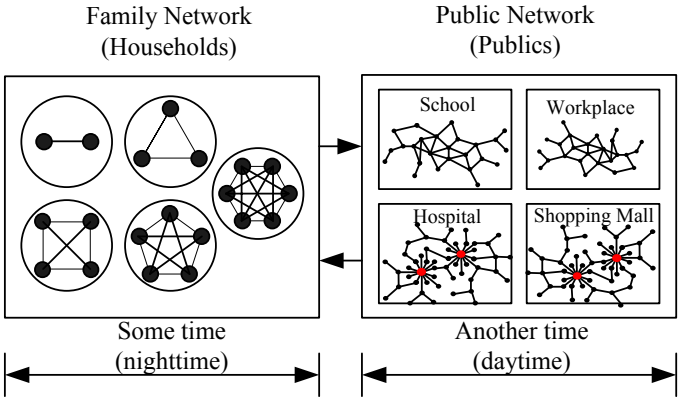

Fig. 1. Schema of the alternate social networks. The family network and the public network are formed during some time (*e.g.* nighttime) and another time (*e.g.* daytime), respectively. Dots represent individuals and lines between dots represent contacts between individuals that could potentially lead to disease transmission. Each household in the family network is fully connected, while in the public network each sub network can be a small-world or a scale-free network. For the family network, the size of sub networks is uniformly distributed, while for the public network, it is drawn from a power-law distribution (see text).

uals, which is in our model randomly partitioned into several subgroups, namely publics or households according to different time. For simplicity, it is assumed that each subgroup is isolated from others, so only contacts

between individuals in the same subgroup are allowed. These subgroups can be modelled by sub networks with a certain topology. The set of sub networks formed during some time (*e.g.* daytime) and during another time (*e.g.* nighttime) are then referred to as the public network (PN) and the family network (FN), respectively. For the FN, the size $N_{\rm fn}$ of the sub networks is uniformly distributed in the interval $[2, 6]$, while for the PN the size $N_{\rm pn}$ of the sub networks is drawn from the power-law distribution $P(N_{\rm pn}) \sim N_{\rm pn}^{-4}$ in the range $[50, 150]$ according to the fact that the size distribution of workgroups has power-law form [11]. In this way, the population is modelled in turn by FN, each sub network of which is fully connected, and PN, of which the sub network can be a small-world network (SWN), a scale-free network (SFN), or some other network models. In this paper, we have considered two cases:

- Case A: All sub networks of PN are SFNs;
- Case B: All sub networks of PN are SWNs.

For Case A, each SFN has been generated by BA model in [8]: *(i)* Start from an initial network of m fully interconnected nodes. *(ii)* At each time step, a new node with m edges that link to m different old nodes is added into the system. The connection probability of a new node to already present node i is given by $\Pi(k_i) = k_i / \sum_j j k_j$. Finally, a network with an average degree $\langle k \rangle = 2m$ is generated, and the expected degree distribution follows a power law $P(k) = 2m^2 k^{-3}$ in the thermodynamic limit.

For Case B, each SWN has been generated by WS model in [9]: *(i)* Start from a one dimensional ring lattice with desired size satisfying the periodic boundary condition and connect each node to its $\langle k \rangle = 2m$ neighbours; *(ii)* Rewire each edge of the lattice with probability p, ignoring the self-connection and duplication of edges.

With this method of modelling of the social interactions, the ASN can exhibit some essential characteristics of real social networks, such as small-world topology of connections, scale-free distribution of degree (for case A), a large clustering coefficient, a hierarchical structure and the network evolution (change from PN to FN, and so on). It should be noted that the ASN can be extended to take into account more factors to model the real contact networks. For example, it may be a more feasible way to model the public network by taking a mixture of Case A and B, where the SFN will be used to model the subgroup if there is some super-spreaders.

The epidemic spreading is modelled using the standard susceptible-infected-susceptible (SIS) mechanism [1]. Each individual can be in either a susceptible or an infected state. We start with an initial density ρ_0 of infected individuals. The dynamics of the model are such that at every time

step: (1) Susceptible individuals become infected with probability λ if at least one of the neighbours is infected. (2) Infected vertices, on the other hand, recover and become susceptible again with probability one. Although this simple epidemic dynamics is inpractical in real world and also different from the normal one in which the infection probability is dependent on the density of infected individuals, it is feasible to study the epidemic threshold, as we can see in [23].

Here, we define the network active time n for PN and FN, which means that the epidemic system will be continuously updated n time-steps when the network (PN or FN) is active. Thus, the above spreading mechanism will be iterated for n_{p} time-steps on PN, then for another n_{f} time-steps on FN, and so on. This process can be readily iterated in numerical simulations until the system reaches its stationary state or the epidemic dies out. It should be noted that the time step in our model is different from most epidemic simulation models where the time step is assumed to be equivalent to one 24-hour period in the real world. Although this time step in our model may be unreasonable in real world, it is easy to implemented and still effective for qualitative study of the epidemic threshold according to our research. Moreover, it allows us to study the different roles of PN and FN by tuning the network active time n. Actually, one can easily extend the spreading mechanism to a practical form by taking the time step as micro time step (thus one 24-hour period is divided into several micro time steps) and changing a bit the updating rules (1) and (2).

3. Results

Extensive numerical simulations have been performed in this section, with synchronous dynamics used. In order to study the SIS model in ASN, we first generate the network using the algorithm described in the preceding section for both settings of Cases A and B, with $N = 10^4$, $[a_{\mathrm{p}}, b_{\mathrm{p}}] = [50, 150]$, and $[a_{\mathrm{f}}, b_{\mathrm{f}}] = [2, 6]$. For Case A, PN shows a power law degree distribution with the exponent being in the interval $2 < \lambda < 3$. Analysis based on a fit yields an exponent $\lambda = 2.58 \pm 0.02$ for $m = 5$. For Case B, we take $p = 0.01$ as the rewiring probability. Initially, a fraction of randomly selected individuals $\rho_0 = 0.1\%$ is infected, and then we update individuals' state synchronously in turn on PN for n_{p} time-steps and on FN for n_{f} time-steps until the system relax into steady state where the prevalence ρ attains its stationary value. It is worth recalling that results are collected over 200 independent simulations, each of which corresponds to a different realization of the network and different initially infected individuals and the prevalence in the stationary state is computed as the average over all surviving trials. The results obtained are depicted in Fig. 2 where the stationary state

prevalence has been drawn as a function of the spreading rate λ for different values of the average connectivity. Only when λ is increased above a value λ_c is a significant prevalence observed for both of Cases A and B. The effect of the topological properties of Case A becomes clear when comparing the shape of the prevalence curves with that obtained for BA model with $m = 5$. In the later case no change of behaviour is apparent as the prevalence and its slope varies smoothly when λ is increased. The average connectivity can strongly affect the epidemic spreading, such as the epidemic threshold and the prevalence. It also can be found that Case A is much like Case B when regarding the shape of the prevalence curves, and the prevalence value of ρ for them is much lower than those obtained for BA and WS models.

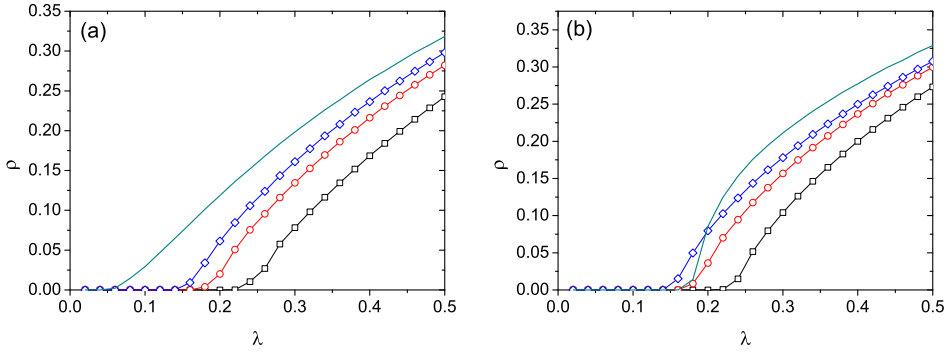

Fig. 2. Prevalence (fraction of infected individuals in the stationary state) as a function of the spreading rate λ for (a) Case A with $m = 3$ (squares), 5 (circles), and 7 (diamonds), and BA model with $m = 5$ (solid curve), (b) Case B with $m = 3$ (squares), 5 (circles), and 7 (diamonds), and WS model with $m = 5$ (solid curve). The other parameters are $N = 10^4$, $\rho_0 = 0.1\%$, $p = 0.01$, $n_p = 1$, $n_f = 1$,$[a_p, b_p] = [50, 150]$, $[a_f, b_f] = [2, 6]$.

In order to understand the role of the topology we consider the clustering coefficient [9], which is defined as follows. Consider a node i with total degree k_i. Between the k_i nodes that i is linked with, at most $k_i(k_i - 1)/2$ links are possible. Let C_i denote the fraction of links that actually exist among the neighbours of i. The clustering coefficient C is the average of C_i taken over all N nodes i in the network. Note that all links are considered as bidirectional when calculating the clustering coefficient. Here, C is calculated only for PN due to the fact that FN is just a group of independent fully connected households. Fig. 3 shows that for Cases A and B the clustering coefficient C towards a high stationary value as the network size N is increased. For the sake of comparison, in Fig. 3 the clustering coefficient of the BA model and WS model are plotted as well, which can be instead quantitatively described by [35]:

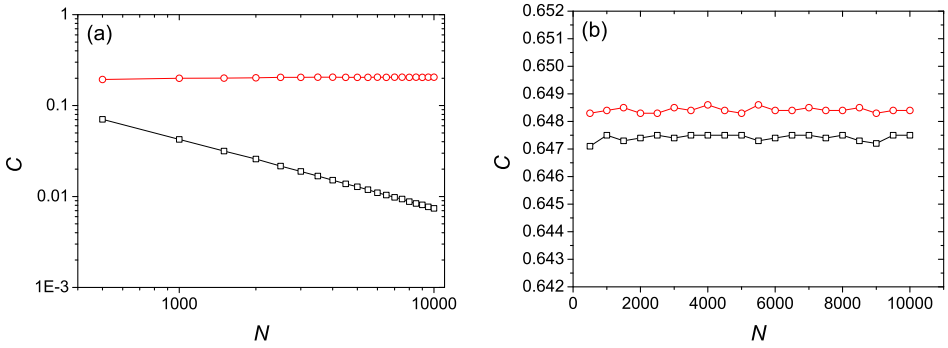

Fig. 3. Dependence of the clustering coefficient C on the size of network of (a) PN (Case A) (circles) and the BA model (squares), (b) PN (Case B) (circles) and the WS model (squares). The other parameters are $m = 5$, $p = 0.01$, $[a_{\rm p}, b_{\rm p}] = [50, 150]$. Results are averaged over 200 independent simulations, each of which corresponds to a different realization of the network.

$$C = \frac{m^2(m+1)^2}{4(m-1)} \left[\ln\left(\frac{m+1}{m}\right) - \frac{1}{m+1} \right] \frac{[\ln(N)]^2}{N}\,, \tag{1}$$

and [36]:

$$C(p) = \frac{3(\langle k\rangle - 2)}{4(\langle k\rangle - 1)}(1-p)^3\,, \tag{2}$$

respectively. Thus the Cases A and B generate networks with a higher clustering coefficient than the corresponding BA and WS models. This result indicates that dividing the whole population into subgroups will result in a network with high clustering coefficient. Further analysis finds that the value of clustering coefficient for Cases A and B towards a higher value as the average connectivity increases (Fig. 4). From Fig. 2 we can see the epidemic threshold is strongly related with the average connectivity. These results suggest that high clustering of the ASN can protect the network against the spreading of diseases. It should be pointed out that the high clustering coefficient in our model is rooted in the existence of local structure (sub network) in the network, which is different from the work [23], where the network model is the so-called structured scale-free network.

Another important parameter is the network active time n. By tuning this parameter, one can study the different influences of the PN and FN on the epidemic spreading. Two configurations have been considered: (1) $n_{\rm p}$ varies while $n_{\rm f}$ fixed (Fig. 5(a)); (2) $n_{\rm f}$ varies with $n_{\rm p}$ fixed (Fig. 5(b)). It can be found that the network is more prone to the spreading of disease when $n_{\rm p}$ is changed from 1 to 3, but no obvious change observed for higher value of $n_{\rm p}$. Contrarily, the increase in $n_{\rm f}$ can effectively protect the network

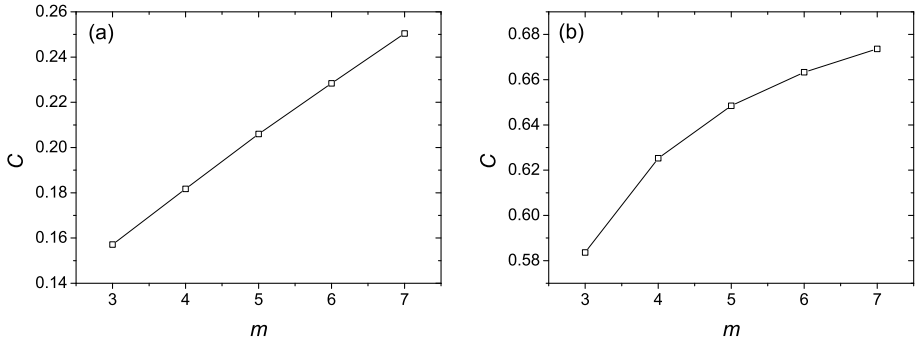

Fig. 4. Dependence of the clustering coefficient C on the parameter m for PN with (a) Case A, (b) Case B. The other parameters are $N = 10^4$, $p = 0.01$, $[a_{\rm p}, b_{\rm p}] = [50, 150]$. Results are averaged over 200 independent simulations, each of which corresponds to a different realization of the network.

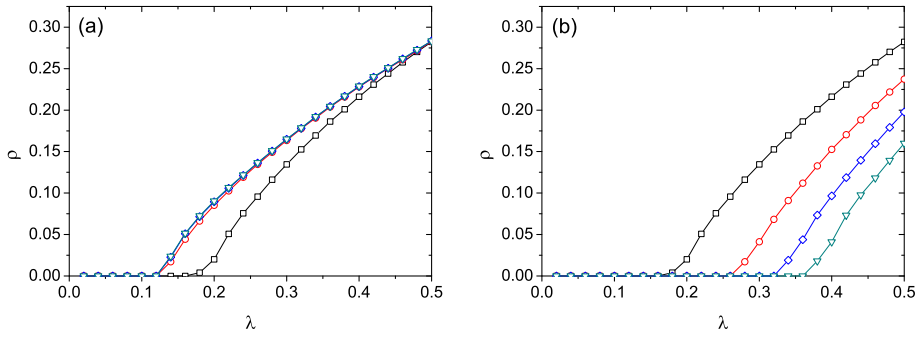

Fig. 5. Prevalence as a function of the spreading rate λ for Case A, with (a) $n_{\rm p} = 1$ (squares), 3 (circles), 5 (diamonds), 7 (triangles), and $n_{\rm f}$ fixed to 1. (b) $n_{\rm f} = 1$ (squares), 3 (circles), 5 (diamonds), 7 (triangles), and $n_{\rm p}$ fixed to 1. The other parameters are $N = 10^4$, $\rho_0 = 0.1\%$, $p = 0.01$, $m = 5$, $[a_{\rm p}, b_{\rm p}] = [50, 150]$, and $[a_{\rm f}, b_{\rm f}] = [2, 6]$.

against the epidemic spreading. In Fig. 6 we plot the threshold value of spreading rate λ as a function of the parameter $n_{\rm f}$, which show that large $n_{\rm f}$ can effectively prevent the disease spreading. These results imply that staying at home can greatly reduce the risk of being infected. Moreover, it brings to us that the fully connected structures (households) that coexist with the large SF or SW sub networks is another dominating ingredient for the epidemic threshold. This result allows claiming that although SF networks are measured in real societies the existence of FN or households that are fully disconnected from each other prevents the spreading of the disease across the whole network, which is in contrast to the previously reported behaviour on single SF network.

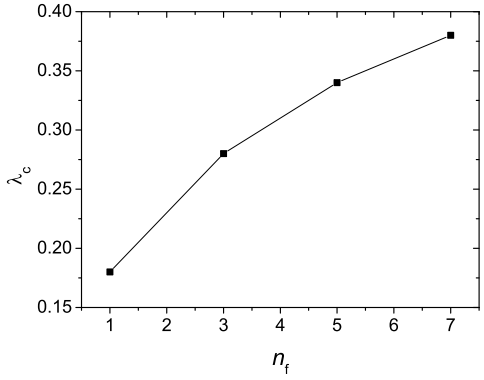

Fig. 6. Threshold value of spreading rate λ as a function of the parameter $n_{\rm f}$ for the Case A, with $N = 10^4$, $n_{\rm p} = 1$, $\rho_0 = 0.1\%$, $m = 5$, $[a_{\rm p}, b_{\rm p}] = [50, 150]$, and $[a_{\rm f}, b_{\rm f}] = [2, 6]$.

Finally, we studied the influence of the initial condition on the epidemic spreading by setting $\rho_0 = \text{const.}$ and $\rho_0 = 1/N$. In both cases, the prevalence curves are the same regardless of the initial condition of ρ_0. This indicates that the value of epidemic threshold and the prevalence is independent of the initial condition. We also have investigated the finite-size effect on the epidemic spreading and found that the epidemic threshold for our model is independent of the system size or the size distribution of PN over a considered range of values.

4. Conclusions

This paper introduces the alternate social networks (ASN) of human interactions to study the dynamics of the SIS epidemic. The ASN consists of a family network (FN) and a public network (PN), to model the human interactions during nighttime and daytime, respectively. In the current study, two versions of ASN, Cases A and B, have been considered. The networks generated by Case A still follow a power law degree distribution with the exponent being in the interval $2 < \gamma < 3$, but with a high clustering coefficient regardless of the system size, that is, the small-world properties are restored. Thus, the ASN model has the essential characteristics of real social networks, including the small-world property (large clustering coefficient), scale-free distribution of degree (for case A), a hierarchical structure and the network evolution.

Our simulations show that the high clustering of the ASN can protect the network against the spreading of diseases, and also make Case A much like the Case B. However, it should be pointed out that the high clustering

coefficient in our model is rooted in the existence of local structures (sub networks) in the PN, even when the sub networks are generated by the BA model, which is different from the work [23], where the network model is the so-called structured scale-free network.

Moreover, we found that the PN is more prone to the epidemic spreading than the FN, which suggested that the fully connected structures (households) that coexist with the large SF or SW sub networks is another dominating ingredient for the epidemic threshold. This result allows claiming that although SF networks are measured in real societies the existence of FN or households that are fully disconnected from each other prevents the spreading of the disease across the whole network, which is in contrast to the previously reported behaviour on single SF network.

Finally, our results show that the existence or not of an epidemic threshold for the SIS model on the ASN does not dependent on the initial condition and is irrelevant with the finite-size effect.

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