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Epidemic spreading on contact networks with adaptive weights

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HIGHLIGHTS

- We propose an epidemic SIS model on an adaptive and weighted contact network.
- Fixed weights setting can trigger the epidemic incidence.
- The adaptivity of weights cannot change the epidemic threshold but it can accelerate the disease decay.
- Strong adaptivity can suppress the epidemic globally to a low level, but cannot exterminate it.
- In contact patterns the frequency plays a more important role in epidemic spreading than the intensity.

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ABSTRACT

The heterogeneous patterns of interactions within a population are often described by contact networks, but the variety and adaptivity of contact strengths are usually ignored. This paper proposes a modified epidemic SIS model with a birth–death process and nonlinear infectivity on an adaptive and weighted contact network. The links' weights, named as 'adaptive weights', which indicate the intimacy or familiarity between two connected individuals, will reduce as the disease develops. Through mathematical and numerical analyses, conditions are established for population extermination, disease extinction and infection persistence. Particularly, it is found that the fixed weights setting can trigger the epidemic incidence, and that the adaptivity of weights cannot change the epidemic threshold but it can accelerate the disease decay and lower the endemic level. Finally, some corresponding control measures are suggested.

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1. Introduction

In epidemic processes, many infectious diseases spread directly from one individual to another via physical interactions. Therefore, contact networks with nodes representing individuals and links representing interactions have become a popular framework for investigating the epidemic spreading dynamics. Such networks can be used to visualize the outbreak of infection (Andre et al., 2007; Eames, 2008), to predict thresholds (Givan et al., 2011), to form a basis of modeling new approaches and to identify key individuals for intervention (Christley et al., 2005; Eames et al., 2009). Many numerical and analytical results have shown that the topologies of the underlying networks have a strong impact on the spread of infections, e.g., implying or indicating the absence of an epidemic threshold (Pastor-Satorras and Vespignani, 2001; Moreno et al., 2002) and the hierarchical spreading patterns of epidemic outbreaks (Barthelemy et al., 2005).

Recent studies have demonstrated that many large-scale systems, such as the Internet, biological and social networks, exhibit heterogenous topological properties, particularly scale-free network feature (Albert and Barabasi, 2002). In a thorough survey of human social mixing, heterogeneity has also been observed both in contact numbers and in link weights (Read et al., 2008). Based on contact networks, some epidemic models, such as SI (Barthelemy et al., 2005), SIS (Pastor-Satorras and Vespignani, 2001) and SIR (Moreno et al., 2002), have been investigated, yet there are some inappropriate assumptions such as closed populations, i.e., the total number of individuals stay invariant during the whole epidemic duration. Since some diseases can be persistent to last for an individual's life time, it is interesting to study the impact of birth and death on the spreading dynamics (Sanz et al., 2010; Liu et al., 2004; Zhang and Jin, 2011). In Sanz et al. (2010) an epidemiological model with constant birth and death rates was investigated to obtain dynamics of Tuberculosis-like infection. In Liu et al. (2004),

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it was suggested that empty nodes give birth to individuals with certain rate, which may be not very reasonable however. On the other hand, many current network models ignore the interaction strength and assume that each link is equivalent. Notice that many real networks are intrinsically weighted: their nodes and links have different weights, and the variations of their interaction strengths are essential for carrying out their basic functions. Examples include the internet traffic (Pastor-Satorras and Vespignani, 2004) and the flows of passengers in the airline network (Barrat et al., 2004a). The differences between links within a contact network can be described by link weights, which can represent the amount of time two individuals interact or the intimacy or proximity of their encounters (Boccaletti et al., 2006; Newman, 2004; Yan et al., 2005; Read et al., 2008; Barrat et al., 2004c). The link weights provide a way to assess the chance that the disease spreads along the links (Eames et al., 2009). The larger a weight is, the more intensively the two end-nodes communicate, so the more possible the susceptible individual becomes infected. Recently, it was shown that by using contact weights to evaluate an individual's influence on an epidemic process, individual infection risk can be estimated so that targeted interventions can be applied effectively (Eames et al., 2009). By assigning links' weights to denote familiarity, it was pointed out that the nodes with larger weights are preferential to be infected and that large dispersion of weights results in slower spreading (Yan et al., 2005). Further, it was found that the infectivity exponent has a stronger effect on the epidemic threshold and on the epidemic prevalence than the weight exponent (Chu et al., 2011). The usual assumption is that weights are constant and driven through the network connectivity, which is fixed as time goes on. For example, the weight between two nodes with degrees i and j are represented by a function of their degrees (Barrat et al., 2004a,b,c). However, as the disease progresses and the situation becomes severe, individuals tend to be more cautious in social contacts and employ some reactions such as decreasing the out-going visits, cutting down the meeting time, and reducing the intimacy. Such behaviors will change the strengths of nodes and the weights of links, which corresponds to an adaptive weighted network by nature.

Moreover, most network models assume that each infected individual can establish contacts with all its neighbors, namely, the infectivity of each infected node equals to its degree. But in practical situations, an individual cannot contact with all its acquaintances within a short time, especially when he/she is ill. To take this fact into account, it was suggested that the infectivity is a constant (Zhou et al., 2006). Later, a piecewise linear infectivity was introduced (Fu et al., 2008). Actually, infectivity can admit much more complicated nonlinear expressions for different populations and epidemics (Zhang and Fu, 2009).

In this paper, motivated by the above observations, we propose a modified SIS model with birth and death of individuals, which would be more reasonable and precise to analyze a long-lasting epidemic spreading in an open population. To account for different cases of transmission and infectivity, we introduce general forms of the weight function and infectivity function. The weights correspond to the intimacy or familiarity between two connected individuals, whose role is reflected by the infection rate. Particularly, due to people's health-conscious behavior, the weights will reduce as the disease propagates, which we call 'adaptive weights'. We investigate the threshold, dynamics and propagation behavior of the model, and analyze the influence of weights on epidemic spreading.

The rest of this paper is organized as follows. In Section 2, we build the model via dynamical differential equations. Then we present a global analysis of the model in Section 3. In Section 4, we have some discussions on related issues and perform some numerical analysis. We finally conclude the paper in Section 5.

2. Model formulation

To simulate the process of interaction, a complex network N is established and individuals are spatially distributed on this network, where each node of N is either vacant or occupied by one individual. In an epidemic spreading process, every node has three optional states: vacant state, healthy individual occupation, infected individual occupation (Liu et al., 2004). Each node can change its state at a certain rate. Individuals can generate offsprings into neighboring vacant sites at a birth rate b. In other words, a birth event occurs at a vacant node next to a non-vacant node at rate b, which also depends on the number of neighboring individuals. Due to the physiological limitation, it is assumed that, at each time step, every individual generates the same birth contacts A, here A is a constant. Furthermore, it is assumed that healthy (infected) individuals give birth to healthy (infected) children. Meanwhile, a healthy individual can be infected through contact if it is connecting to an infected one, while an infected individual can be cured at rate μ. All individuals die at rate d, namely, the disease is not fatal. If an individual dies, the occupied node becomes vacant.

In order to account for the heterogeneity of contact patterns, it is needed to consider the difference of node degrees. Let $S_k(t)$ and $I_k(t)$ denote the densities of susceptible and infected individuals with degree k at time t, respectively. $S(t)=\sum_k P(k)S_k(t)$ and $I(t)=\sum_k P(k)I_k(t)$ are the average densities of susceptible and infected individuals, respectively, where $P(k)$ is the probability that a randomly chosen node has degree k. Let the density of nodes with the same degree be unity after normalization, and then the density of the vacant nodes with degree k is $1-S_k(t)-I_k(t)$. Therefore, the evolution processes of $S_k(t)$ and $I_k(t)$ are governed by the following differential equations:

$$\begin{cases} \dfrac{dS_k(t)}{dt} = bk[1-S_k(t)-I_k(t)]\sum_i \dfrac{A}{i}P(i|k)S_i(t) - dS_k(t) - kS_k(t)\Theta_k(t) + \mu I_k(t), \\ \dfrac{dI_k(t)}{dt} = bk[1-S_k(t)-I_k(t)]\sum_i \dfrac{A}{i}P(i|k)I_i(t) + kS_k(t)\Theta_k(t) - (d+\mu)I_k(t), \end{cases} \tag{2.1}$$

where

$$\Theta_k(t) = \sum_i P(i \mid k)\frac{\varphi(i)}{i}\lambda_{ik}I_i(t), \tag{2.2}$$

with initial conditions

$$\{(S_k(0),I_k(0)) \mid 0 \le S_k(0) \le 1, 0 \le I_k(0) \le 1, 0 < S_k(0)+I_k(0) \le 1, k = 1,2,\ldots\}$$

and b,d,μ, and λ are positive constants. The meanings of the parameters and variables in model (2.1) are as follows:

- $P(i|k)$ is the probability that a node of degree k is connected to a node of degree i. This paper focuses on degree uncorrelated networks. Hence, $P(i|k)=iP(i)/\langle k\rangle$, where $\langle k\rangle=\sum_i iP(i)$ is the average degree of the network. For a general function $\delta(k)$, it is defined as $\langle \delta(k)\rangle=\sum_i \delta(i)P(i)$.
- $bk[1-S_k(t)-I_k(t)]\sum_i (A/i)P(i|k)S_i(t)$ represents the new born susceptible individuals per unit time, which is proportional to the connectivity degree k, the densities of vacant nodes $(1-S_k(t)-I_k(t))$ and susceptible individuals $S_k(t)$. The factor $1/i$ accounts for the probability that one of the neighboring individual of a vacant node, with degree i, will activate this node at the present time step. $bk[1-S_k(t)-I_k(t)]\sum_i (A/i)P(i|k)I_i(t)$ has a similar meaning. Without loss of generality, we set $A=1$.
- d is the natural death rate. So $1/d$ is the average life span. There is no disease-related death, since the disease is assumed not fatal.

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