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Epidemic spreading driven by biased random walks

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HIGHLIGHTS

- We study the dynamics of epidemic spreading driven by biased random walks.
- We obtain the optimal parameters of our epidemic model.
- We investigate the impact of network structure on our model.

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ABSTRACT

Random walk is one of the basic mechanisms of many network-related applications. In this paper, we study the dynamics of epidemic spreading driven by biased random walks in complex networks. In our epidemic model, infected nodes send out infection packets by biased random walks to their neighbor nodes, and this causes the infection of susceptible nodes that receive the packets. Infected nodes recover from the infection at a constant rate λ, and will not be infected again after recovery. We obtain the largest instantaneous number of infected nodes and the largest number of ever-infected nodes respectively, by tuning the parameter α of the biased random walks. Simulation results on model and real-world networks show that spread of the epidemic becomes intense and widespread with increase of either delivery capacity of infected nodes, average node degree, or homogeneity of node degree distribution.

1. Introduction

Unexpected epidemic outbreaks in biological systems [1,2] and the spread of computer viruses in technology systems [3–5] result in a lot of death or great damage to related systems. The study of epidemiological models has a long history, especially in the field of social science [6,7]. The SIR (susceptible–infected–removed) model and the SIS (susceptible–infected–susceptible) model are two representative models which capture the basic properties of epidemic spreading by defining some transitions among several disease states [8,9]. In the SIR model, a susceptible individual becomes infected at some rate when the individual has contact with infected individuals. An infected individual recovers from the disease at a constant rate and is assumed to get a permanent immunity. Therefore, the SIR model always terminates when all infected individuals are recovered. However, in the SIS model there are only two states: susceptible and infected. A recovered individual can get infected again. If the fraction of infected individuals is large enough, the disease will spread indefinitely, otherwise it will die out after a period of time. In the past, epidemic models were discussed under the homogeneous mixing hypothesis [10], which assumes that at each time point an arbitrary individual has an equal opportunity to contact everyone in the population. Recently, results from the area of network science demonstrated that most real-world network systems

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have heterogeneous topological structures [11–13], and this caused many mathematicians and physicists to explore epidemic models on heterogeneous random networks [14–20] by means of mean-field approximation [21–23], generating functions formalism [24], and percolation theory [25]. It was found that the epidemic threshold is absent for random networks with strongly heterogeneous degree distribution, which means an epidemic always has a finite probability to survive indefinitely [11,21]. Most recently, much attention has been paid to the study of epidemic spreading in multiplex networks. For example, it was found that the critical point for the spread of a disease in a multiplex network is lower than the critical point in each isolated network [26,27]. Also, many researchers obtained the interrelation between the spread of an epidemic and the information awareness of the epidemic on multiplex networks [19,28].

Besides network structures, traffics in networks also have great impacts on epidemic spreading. For instance, in the Internet computer viruses are transmitted from a node to another with data packets. Without transmission of packets, viruses do not spread even if the two nodes are physically connected. In world city networks, air traffics speed the spread of diseases among different spatial areas. The combination of epidemic spreading and traffic dynamics was first considered in the metapopulation model [29] which characterizes the dynamics of systems composed of subpopulations. Then, Meloni et al. [30] studied the impact of traffic dynamics on the spread of viruses in the Internet, in which information packets are transmitted with the shortest paths. Later, many mechanisms were proposed to suppress traffic-driven epidemic spreading, for instance controlling the traffic flow [31], the routing strategy [32,33], the heterogeneous curing rate [34], and deleting some particular edges [35], etc.

Random walk is a basic mechanism related to many spreading processes [36–41]. For example, a mobile phone virus may randomly dial some phone numbers from a directory. Some computer viruses propagate randomly by email or other online communication tools. Therefore, the role of random walks in epidemic spreading should be explored. We propose an epidemic model driven by biased random walks. In our model, infected nodes send out a constant number of infection packets to their neighbor nodes through biased random walks. Susceptible nodes are infected after receiving the infection packets, and then are removed from the set of infected nodes with a constant rate. We investigate the spreading properties and the optimal control parameters of our model, as well as the influence of network structures on our model.

2. SIR model

The SIR model is one of the traditional epidemic spreading models in literature. In the SIR model, there are three types of nodes: susceptible nodes, infected nodes and removed nodes. A susceptible node can contract the infection. An infected node was previously a susceptible node that got infected by the disease. A removed node is the one that recovered from the disease, and was removed from the set of infected nodes. Assuming that the numbers of susceptible, infected and removed nodes at time t are S, I and R respectively. There are three basic elements in the SIR model as follows [8]:

(1) $S + I + R = N$. N is the number of nodes in the network.
(2) An arbitrary infected node infects the susceptible nodes by the ratio β. The number of new infected nodes is $\beta * S * I$.
(3) The number of new removed nodes is proportional to the total number of infected nodes I, which is $\lambda * I$.

According to these three elements, the dynamics of the SIR model can be expressed as follows [8]:

$$\begin{cases} \dfrac{\mathrm{d}I}{\mathrm{d}t} = \beta SI - \lambda I, \\ \dfrac{\mathrm{d}S}{\mathrm{d}t} = -\beta SI, \\ \dfrac{\mathrm{d}R}{\mathrm{d}t} = \lambda I. \end{cases} \tag{1}$$

When time t is large enough, all the infected nodes will become removed nodes, and the epidemic spreading stops.

The SIR model is based on the assumption that a node in the network contacts every other node with equal probability. However, in real situations, individuals often have heterogeneous numbers of contacts [11]. A few individuals have a larger number of contacts, and they will get more contacts according to the "rich-get-richer" mechanism, while most of the individuals have a few contacts.

3. Epidemic model driven by biased random walks

In real situation, epidemics are often spreading with other physical quantities on networks. In the Internet, a virus cannot spread from a node to another node unless there is a transport of infection packets between the two nodes. Additionally, in city networks even roads within cities are physically well connected, an epidemic cannot spread among cities unless infectious individuals travel among the cities. Therefore, based on the SIR model, we further consider the transport of infection packets in networks.

In our model, a randomly selected node is infected initially. In one time step, each of the infected nodes sends C infection packets independently to its neighbor nodes through biased random walks. An infection packet diffuses from one infected node to a neighbor node with a probability that is a function of the neighbor node's connectivity [42,43], in contrast to the