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Traffic-driven SIR epidemic spreading in networks

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HIGHLIGHTS

- We study traffic-driven SIR epidemic spreading in networks.
- Homogeneous load distribution facilitates the epidemic spreading.
- Large-degree nodes have dual effects on the epidemic spreading.
- Traffic congestion blocks the epidemic spreading.

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ABSTRACT

We study SIR epidemic spreading in networks driven by traffic dynamics, which are further governed by static routing protocols. We obtain the maximum instantaneous population of infected nodes and the maximum population of ever infected nodes through simulation. We find that generally more balanced load distribution leads to more intense and wide spread of an epidemic in networks. Increasing either average node degree or homogeneity of degree distribution will facilitate epidemic spreading. When packet generation rate ρ is small, increasing ρ favors epidemic spreading. However, when ρ is large enough, traffic congestion appears which inhibits epidemic spreading.

1. Introduction

As the increase of connectivity in and between different complex systems, spread of many diseases or viruses is becoming more and more prevalent in our society [1–4]. For instance, outbreaks of many infectious diseases, including Severe Acute Respiratory Syndromes (SARS), Swine flu (H1N1), and the recent Ebola virus, caused great damage and loss of life. The spread of computer and mobile phone viruses brought about a great deal trouble to human life and serious damage to economy. Understanding the intrinsic mechanisms of those spreading processes and designing efficient control strategies become very important and urgent tasks, which bring together a lot of researchers from areas of biology, sociology, mathematics, physics, engineering, etc. [3].

Mathematical modeling of epidemic spreading has a long history of more than two hundred years [4]. Generally, the population is divided into several classes: susceptible, infected and recovered individuals. Susceptible individuals represent those who can contract the infection. Infected individuals were previously susceptible individuals and got infected by the disease. Recovered individuals are those who have recovered from the infection. In the susceptible–infected–susceptible (SIS) model [4], infected individuals can recover from the disease and become susceptible individuals again. While in the susceptible–infected–recovered (SIR) model [4], infected individuals no longer get infected after recovery from the disease, which are assumed to get the permanent immunity. In classical epidemiology, a common assumption is that individuals in

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a class are treated similarly, and have equal probability to contact with everyone else [3,4]. However, the recent abundance of data demonstrates that both the connectivity pattern and the contact rate are heterogeneous among real-world complex networks [3,5], which means the traditional deterministic differential equations and many other related results of epidemic processes are inadequate in real-world situations. This greatly stimulates the research of epidemics on real-world complex networks [4]. Due to the complexity of real-world networks, the mean-field approach [6–8] and the generating function approach [9] are used to drive the analytic results of epidemics spreading. One of the remarkable results obtained by Pastor-Satorras and Vespignani [4,6] shows that in the limit of a network of infinite size, the epidemic threshold of the SIS model tends to zero asymptotically in scale-free networks with power-law parameter in (2, 3]. For SIR model, it was found that in the thermodynamic limit, not only the threshold tends to vanish, but also the time for the stabilization of the infection becomes very small [10,11]. By using the message-passing approach, Karrer and Newman [12] calculated the probabilities for any node and any time to be in state S, I, and R on tree structure. Many other explicit results of SIR model are obtained by mapping the SIR model to the percolation process [9,13,14]. Also, effects of degree correlations [15], clustering [16,17], weights and directions of edges [7,18,19] on epidemic spreading are broadly discussed. On the other hand, various efficient immunization protocols [4,20,21] have been designed for controlling the spread of epidemics on networks. Recently much attention has been transferred to epidemic spreading in temporal and multiplex networks [22–24].

Addition to diseases or viruses, there are usually many other substances spreading in networks like information packets, goods, ideas, etc., which depend on the specific types of the networks. Epidemic spreading is often coupled with the delivery of these substances. For example, HIV spreads through the exchange of body fluids among individuals in contact networks. Computer viruses spread with the delivery of information packets in computer networks. Flu often spreads by air traffics among different spatial areas. Therefore, understanding the mechanisms of these coupled spreading processes and how these processes affect each other is significant for designing efficient epidemic immunization strategies. Meloni et al. [25] first studied the effects of traffic flow on epidemic spreading. They found that the epidemic threshold in the SIS model decreases as flow increases, and emergence of traffic congestion slows down the spread of epidemics. Then, Yang et al. [26,27] further studied the relation between traffic dynamics and the SIS epidemic model, and found that the epidemic can be controlled by fine tuning the local or global routing schemes. Furthermore, they obtained that the epidemic threshold can be enhanced by cutting some specific edges in the network [28]. The impacts of traffic dynamics on SIR epidemic model have not been reported in literature. We study the traffic-driven SIR spreading dynamics in complex networks. We focus on the instantaneous size of infected population, and the final size of ever infected population. Based on these two properties, we study how the packets transmission process governed by given routing protocols affects the epidemic spreading.

2. Model

Our model includes two coupled processes: packet delivery process and the epidemic spreading process. We will introduce our model in the context of computer networks.

2.1. Packet delivery process

We assume that nodes in the network are identical which can generate, receive and deliver information packets. Each node has a queue obeying the First-In–First-Out (FIFO) rule for storing packets. The length of the queue is set infinite. Load of a node is the number of packets in its queue. Every node generates packets at a rate ρ. For example if $\rho = 1.5$, each time an arbitrary node generates one packet definitely and another one with probability 0.5. The destination nodes of the packets are chosen randomly, and the packets will be removed from the network after arriving at the destination nodes. The transmission of packets is governed by the efficient routing protocol proposed by Yan et al. [29]. For an arbitrary path p of length l between node s and d, denoted as $\langle s, n_1, n_2, \ldots, n_{l-1}, d\rangle$, its routing cost is defined as follows:

$$\phi(p) = \sum_{i=1}^{l-1} k_i^{\alpha} \tag{1}$$

where k_i is the degree of node i, and α is a control parameter. The sum runs over all the intermediate nodes of path p. The efficient paths for delivering packets are defined to be those which have the minimum routing costs. If there are many efficient paths between two nodes, we randomly chose one for delivering packets. According to Eq. (1), α determines the routing cost of a path. When $\alpha > 0$, paths with large-degree nodes usually have large routing costs. Thus, efficient paths tend to be those paths composed of small-degree nodes when $\alpha > 0$, and vice versa. Each time a node can deliver at most C packets. When $C = \infty$, all the packets can be delivered without delay, there is no traffic congestion. The overall load of the network is constant after a short transient time. When C is a constant value, there is a critical packet generation rate ρ_c. When $\rho < \rho_c$, there is no traffic congestion. When $\rho > \rho_c$, the network generates more packets than it can deliver. As a result, the overall load of the network increases with time, which is the traffic congestion phenomenon. We use the order parameter to characterize the traffic congestion, which is as follows [30]:

$$\psi = \frac{1}{\rho N} \lim_{t\to\infty} \frac{\langle w(t+\Delta t) - w(t)\rangle}{\Delta t} \tag{2}$$