Traffic-driven epidemic spreading on scale-free networks with tunable degree distribution

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We study the traffic-driven epidemic spreading on scale-free networks with tunable degree distribution. The heterogeneity of networks is controlled by the exponent $\gamma$ of power-law degree distribution. It is found that the epidemic threshold is minimized at about $\gamma = 2.2$. Moreover, we find that nodes with larger algorithmic betweenness are more likely to be infected. We expect our work to provide new insights into the effect of network structures on traffic-driven epidemic spreading.

Keywords: traffic-driven epidemic spreading; scale-free networks; degree heterogeneity

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1. Introduction

Epidemic spreading \[1,2,3,4,5,6,7,8,9,10,11,12\] and traffic transportation \[12,13,14,15,16,17,18,19,20,21,22,23\] on complex networks have attracted much attention in the past decade. In many cases, epidemic spreading is relied on the process of transportation. For example, a computer virus can spread over Internet via data transmission. Another example is that air transport tremendously accelerates the propagation of infectious diseases among different countries.

The first attempt to incorporate traffic into epidemic spreading is based on metapopulation model \[24,25,26,27,28,29\]. This framework describes a set of spatially structured interacting subpopulations as a network, whose links denote the traveling path of individuals across different subpopulations. Each subpopulation consists of a large number of individuals. An infected individual can infect other individuals in the same subpopulation. In a recent work, Meloni et al. proposed another

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traffic-driven epidemic spreading model, in which each node of a network represents a router in the Internet and the epidemic can spread between nodes by the transmission of packets. A susceptible node will be infected with some probability every time it receives a packet from an infected neighboring node.

The routing strategy plays an important role in the traffic-driven epidemic spreading. Meloni et al. observed that when travelers decide to avoid locations with high levels of prevalence, this self-initiated behavioral change may enhance disease spreading. Yang et al. found that epidemic spreading can be effectively controlled by a local routing strategy, a greedy routing or an efficient routing protocol. For a given routing strategy, the traffic-driven epidemic spreading is affected by network structures. It has been found that the increase of the average network connectivity can slow down the epidemic outbreak. Besides, the epidemic threshold can be enhanced by the targeted cutting of links among large-degree nodes or edges with the largest algorithmic betweenness.

Many real networks display a power-law degree distribution: \( P(k) \sim k^{-\gamma} \), with the exponent typically satisfying \( 2 < \gamma \leq 3 \). It has been found that the exponent of power-law degree distribution plays an important role in opinion dynamics and evolutionary games. In this paper, we study how the exponent of power-law degree distribution affects the traffic-driven epidemic spreading. Our preliminary results have shown that there exists an optimal value of exponent, leading to the minimum epidemic threshold.

The paper is organized as follows. In Sec. 2, we introduce scale-free networks with tunable degree distribution. In Sec. 3, we describe the traffic-driven epidemic spreading model. The results and discussions are presented in Sec. 4. Finally, we give a brief conclusion in Sec. 5.

2. Scale-free networks with tunable degree distribution

We adopt the algorithm proposed by Dorogovtsev et al. to generate the scale-free networks with tunable degree distribution.

Initially, there are \( m \) fully connected nodes. At each time, a newly added node makes \( m \) links to \( m \) different nodes already present in the network. The probability \( \Pi_i \) that the new node will be connected to an old node \( i \) is:

\[
\Pi_i = \frac{k_i + Am}{\sum_j (k_j + Am)},
\]

where \( k_i \) is the degree of node \( i \), the sum runs over all old nodes, and \( A \) is a tunable parameter (\( A > -1 \)). After a long evolution time, this algorithm generates a scale-free network following the power-law degree distribution \( P(k) \sim k^{-\gamma} \) with the degree exponent \( \gamma = 3 + A \). Particularly, this algorithm produces the Barabasi-Albert network model when \( A = 0 \). The average degree of the network \( \langle k \rangle = 2m \).
Fig. 1. (Color online) The degree distribution $P(k)$ for different values of $\gamma$. The inset shows the degree heterogeneity $H$ as a function of $\gamma$. Each data results from an average over 100 different network realizations.

3. Traffic-driven epidemic spreading model

Following the work of Meloni et al. [30], we incorporate the traffic dynamics into the classical susceptible-infected-susceptible model [43] of epidemic spreading as follows.

In a network of size $N$, at each time step, $\lambda N$ new packets are generated with randomly chosen sources and destinations (we call $\lambda$ as the packet-generation rate), and each node can deliver at most $C$ packets towards their destinations. Packets are forwarded according to a given routing algorithm. The queue length of each agent is assumed to be unlimited. The first-in-first-out principle applies to the queue. Each newly generated packet is placed at the end of the queue of its source node. Once a packet reaches its destination, it is removed from the system. After a transient time, the total number of delivered packets at each time will reach a steady value, then an initial fraction of nodes $\rho_0$ is set to be infected (we set $\rho_0 = 0.1$ in numerical experiments). The infection spreads in the network through packet exchanges. Each susceptible node has the probability $\beta$ of being infected every time it receives a packet from an infected neighbor. The infected nodes recover at rate $\mu$ (we set $\mu = 1$ in this paper).

4. Results and discussions

In the following, we carry out simulations systematically by employing traffic-driven epidemic spreading on scale-free networks with tunable degree distribution. We set the size of the network $N = 3000$ and the packet-generation rate $\lambda = 0.5$. Moreover, we assume that the node-delivering capacity $C$ is infinite, so that traffic congestion
will not occur in the network. Packets are forwarded according to the shortest-path routing protocol.

Figure 1 shows the degree distribution $P(k)$ for different values of $\gamma$. One can see that there are more small-degree nodes and less large-degree nodes in network as $\gamma$ increases. Following Ref. [44] we quantify the degree heterogeneity of a network as

$$H = \frac{\langle k^2 \rangle - \langle k \rangle}{\langle k \rangle}. \quad (2)$$

From the inset of Fig. 1 one can see that the degree heterogeneity $H$ decreases as $\gamma$ increases, indicating that the generated network becomes more homogeneous for larger exponent.

Figure 2 shows the density of infected nodes $\rho$ as a function of the spreading rate $\beta$ for different values of the exponent $\gamma$. One can observe that there exists an epidemic threshold $\beta_c$, beyond which the density of infected nodes is nonzero and increases as $\beta$ is increased. For $\beta < \beta_c$, the epidemic goes extinct and $\rho = 0$. Figure 3 shows the dependence of the epidemic threshold $\beta_c$ on the exponent $\gamma$ for different values of the average degree $\langle k \rangle$ of the network. One can observe a nonmonotonic behavior. For different values of $\langle k \rangle$, $\beta_c$ is minimized for $\gamma \approx 2.2$.

According to the analysis of Ref. [30], the epidemic threshold for uncorrelated networks is

$$\beta_c = \frac{\langle b_{\text{alg}} \rangle}{\langle b_{\text{alg}}^2 \rangle} \frac{1}{\lambda N}, \quad (3)$$

where $b_{\text{alg}}$ is the algorithmic betweenness of a node [15,46] and $\langle \cdot \rangle$ denotes the aver-
Traffic-driven epidemic spreading on scale-free networks with tunable degree distribution

Fig. 3. (Color online) The epidemic threshold $\beta_c$ as a function of the exponent $\gamma$ for different values of $\langle k \rangle$. Each data point results from an average over 30 different realizations.

The algorithmic betweenness of a node is the number of packets passing through that node when the packet-generation rate $\lambda = 1/N^{45,46}$. For the shortest-path routing protocol, the algorithmic betweenness is equal to the topological betweenness ($b_{\text{alg}} = b_{\text{top}}$) and $\langle b_{\text{alg}} \rangle = \langle D \rangle / (N - 1)$, where $\langle D \rangle$ is the average topological distance of a network. Here, the topological betweenness of a node $k$ is defined as

$$b_{\text{top}}^k = \frac{1}{N(N-1)} \sum_{i \neq j} \sigma_{ij}(k) \sigma_{ij},$$

where $\sigma_{ij}$ is the total number of shortest paths going from $i$ to $j$, and $\sigma_{ij}(k)$ is the number of shortest paths going from $i$ to $j$ and passing through $k$. The average topological distance of a network is given by $\langle D \rangle = \sum_{i \neq j} d_{ij} / [N(N-1)]$, where $d_{ij}$ is the shortest distance between $i$ and $j$. Combining Eq. (3) and Eq. (4), we are able to calculate the theoretical value of the epidemic threshold $\beta_c$. In Fig. 4 one can notice that for a given $\langle k \rangle$, the theoretical value of $\beta_c$ increases with the exponent $\gamma$. However, in the simulation results, $\beta_c$ decreases with $\gamma$ when $\gamma < 2.2$.

To understand the deviation between numerical results and theoretical analysis, we study traffic flow and infection probability of the nodes with different degrees. From Fig. 5(a), one can see that the algorithmic betweenness $b_{\text{alg}}(k)$ increases with the degree $k$ and the relationship between them follows a power-law form as $b_{\text{alg}}(k) \sim k^\nu$ when $k$ is large. Figure 5(b) shows the density of infected nodes $\rho_k$ as a function of the degree $k$ for different values of $\gamma$. One can see that $\rho_k$ increases with $k$. Combining Figs. 5(a) and (b), one can find that the algorithmic betweenness is positively correlated with the probability of being infected.

In the theoretical analysis, it is assumed that there is sufficient number of nodes...
Fig. 4. (Color online) The theoretical prediction of $\beta_c$ as a function of $\gamma$ for different values of the average degree $\langle k \rangle$ of a network. Each curve results from an average over 30 different realizations.

Fig. 5. (Color online) (a) The algorithmic betweenness $b_{\text{alg}}(k)$ and (b) the density of infected nodes $\rho_k$ as a function of the degree $k$ for different values of $\gamma$. For each value of $\gamma$, we set the spreading rate $\beta = \beta_c + 0.02$. The average degree of the network $\langle k \rangle = 8$. Each data point results from an average over 30 different realizations.

within each degree class $k$. However, when the exponent $\gamma$ is very small (i.e., $\gamma < 2.2$), the network becomes highly heterogeneous and almost all nodes connect to the initial $m$ nodes. As a result, these hubs carry almost all the traffic flow and the epidemic threshold totally depends on only a few hubs. Due to the uncertainty of infection, all the $m$ hubs may simultaneously become susceptible when the spreading rate $\beta$ is small. To make sure at least one hub is infected, the spreading rate $\beta$
must be much higher than the theoretical prediction, leading to deviation between numerical observations and theoretical predictions of the epidemic threshold.

5. Conclusion

In conclusion, we have studied traffic-driven epidemic spreading on scale-free networks with tunable degree distribution. The heterogeneity of networks decreases as the exponent $\gamma$ of the power-law degree distribution increases. It is interesting to find that the epidemic threshold is minimized at about $\gamma = 2.2$. Besides, we find that the nodes with larger degree have higher traffic flow and thus are more likely to be infected. For $\gamma > 2.2$, both simulation results and theoretical analysis show that the epidemic threshold increases with $\gamma$. For $\gamma < 2.2$, the network becomes so heterogeneous that the epidemic threshold totally depends on only a few hubs. To ensure at least one hub is infected, the spreading rate $\beta$ must be set to be a relatively high value, leading to an enhancement of the epidemic threshold. We hope our results can be useful to understand the effect of network structures on traffic-driven epidemic spreading.

Acknowledgments

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