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Control of epidemic spreading on complex networks by local traffic dynamics

Han-Xin Yang,[1,2,*] Wen-Xu Wang,[3] Ying-Cheng Lai,[3] Yan-Bo Xie,[2] and Bing-Hong Wang[2,4]
[1]*Department of Physics, Fuzhou University, Fuzhou 350002, China*
[2]*Department of Modern Physics, University of Science and Technology of China, Hefei 230026, China*
[3]*School of Electrical, Computer and Energy Engineering, Arizona State University, Tempe, Arizona 85287, USA*
[4]*Research Center for Complex System Science, University of Shanghai for Science and Technology and Shanghai Academy of System Science, Shanghai 200093, China*
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Despite extensive work on traffic dynamics and epidemic spreading on complex networks, the interplay between these two types of dynamical processes has not received adequate attention. We study the effect of local-routing-based traffic dynamics on epidemic spreading. For the case of unbounded node-delivery capacity, where the traffic is free of congestion, we obtain analytic and numerical results indicating that the epidemic threshold can be maximized by an optimal routing protocol. This means that epidemic spreading can be effectively controlled by local traffic dynamics. For the case of bounded delivery capacity, numerical results and qualitative arguments suggest that traffic congestion can suppress epidemic spreading. Our results provide quantitative insight into the nontrivial role of traffic dynamics associated with a local-routing scheme in the epidemic spreading.

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Understanding various dynamical processes on complex networks is a central theme in modern network science [1]. Two types of dynamical processes that have been studied extensively are epidemic spreading [2,3] and traffic dynamics [4]. Epidemic spreading is relevant to problems ranging from disease propagation in a human society to virus spreading on computer networks. Traffic dynamics is concerned mainly with how information or "packets" can be delivered efficiently from one location to another on a network and the conditions under which congestion may emerge.

The focus of previous works on spreading was mainly on how the network topology affects the epidemics. In fact, a typical approach in this area is to associate a link with a certain probability of infection. That is, if two nodes, one infected and another susceptible, are connected, then the susceptible node has a certain probability of being infected. The effect of network topology on epidemic spreading can then be conveniently characterized by the epidemic threshold, the critical probability of infection below which the virus dies out. What might happen in a realistic situation is that, even when there is a path connecting two nodes, infection will not propagate unless some kind of packet is delivered between the nodes. For example, a computer virus can spread through email exchanges. Without such an actual "delivery" or transportation process, even if there is a path linking two computers, an infected computer will not be able to infect the other one. Another example is air travel, which by acting as effective transportation may accelerate the propagation of annual influenza [5].

The first attempt to incorporate traffic dynamics in epidemic spreading was made recently by Meloni *et al.* [6], who introduced a theoretical approach to studying the result of an epidemic spreading process driven by the transport of a virus. In particular, they cast the susceptible-infected-susceptible (SIS) model [7] into a flow scenario where the contagion was carried by packets traveling across the network. A susceptible node is more likely to be infected if it receives more packets from infected neighbors, and packets are forwarded following the shortest path or the greedy algorithm [8].

In this Rapid Communication, we study traffic-driven epidemic spreading dynamics but under the assumption of local-routing protocol [9,10] in the sense that a node has knowledge only about its nearest neighbors' degrees. Our approach is to take one of the previously studied local-routing schemes and incorporate the resulting traffic dynamics into the SIS model for epidemic spreading. We find that the routing scheme can affect the epidemic dynamics in a significant manner. Quantitatively, our result can be explained, as follows. Let α be a general parameter characterizing the local-routing process, e.g., determining the probability that a packet is sent to which neighboring node. The issue of how traffic dynamics affects epidemic spreading can then be addressed by investigating how the epidemic threshold depends on α. We find numerically and analytically that, when the network is free of traffic congestion, the epidemic threshold is not a monotonic function of α. In fact, the threshold can be maximized by a proper choice of α. From the standpoint of control, our result means that a suitably designed or controlled local traffic-routing scheme can suppress the emergence of a large-scale epidemic on the network. Traffic congestion, on the other hand, tends to suppress epidemic spreading.

Our model can be described, as follows. (i) Local-routing protocol: Given a network, at each time step, R new packets are generated with randomly chosen sources and destinations, and each node can deliver at most C packets toward their destinations. To transport packets, each node performs a local search among its neighbors. If a packet's destination is found within the searched area, the packet is delivered directly to its target and then removed from the network. Otherwise, the packet is forwarded to node i, one of the neighbors of the searching node, according to the preferential probability [10] $\Pi_i = k_i^\alpha / \Sigma_j k_j^\alpha$, where the sum runs over the neighbors of the searching node, k_i is the degree of node i, and α is an adjustable

*hxyang01@gmail.com

parameter characterizing the local-routing scheme. For $\alpha > 0(<0)$, the packet has a larger probability to be delivered to a larger- (smaller)-degree neighbor of the searching node. When $\alpha = 0$, the packet is forwarded to a randomly chosen neighbor. The queue length of each agent is assumed to be unlimited and the first-in–first-out principle holds for the queue. (ii) Epidemic dynamics: After a transient time, the total number of delivered packets at each time will reach a steady value; then an initial fraction of nodes ρ_0 is set to be infected (e.g., we set $\rho_0 = 0.1$ in numerical experiments). The infection spreads in the network through packet exchanges. All packets in an infected node are infected, while all packets in a susceptible node are uninfected. A susceptible node has the probability β of being infected every time it receives an infected packet from an infected neighbor. With probability $1 - \beta$, the virus in an infected packet will be killed by antivirus software in a susceptible node. The infected nodes are recovered at rate μ (here, we set $\mu = 1$).

We study our model on scale-free networks which follow a power-law degree distribution $P(k) \sim k^{-\gamma}$. We construct scale-free networks with the uncorrelated configuration model (UCM) [11]: (i) Assign to each node i, in a set of N initially disconnected nodes, a number k_i of stubs, where k_i is drawn from the probability distribution $P(k) \sim k^{-\gamma}$ and subject to the constraints $m \leqslant k_i \leqslant \sqrt{N}$ and $\sum_i k_i$ even. (ii) Construct the network by randomly choosing stubs and connecting them to form edges, respecting the preassigned degrees and avoiding multiple and self-connections.

In this Rapid Communication, we set $m = 5$ and the network size $N = 2000$. In the UCM, the maximum connectivity of any node $k_c = \sqrt{N}$. Using again the continuous k approximation, the normalized connectivity distribution has the form $P(k) = (\gamma - 1)(m^{1-\gamma} - k_c^{1-\gamma})^{-1} k^{-\gamma}$.

We first consider the case where the node delivering capacity is unbounded, $C \to \infty$, so that traffic congestion will not occur in the network. Numerically, we have observed that, for a given value of α, the number of packets N_p in the network plateaus, say, at N_{sp}, after a transient time, where N_{sp} depends on α. A quantity that plays an important role in epidemic spreading is the average traveling time of a packet on the network $\langle T \rangle$, which can be calculated analytically by treating the process of a packet's wandering through the network as a biased random walk [12]. The mean first-passage time from node i to an arbitrary node that belongs to the nearest neighborhood of j is

$$T_{ij} = 1 + \frac{N\langle k^{\alpha+1}\rangle}{k_j}\left(\frac{\langle k\rangle}{\langle k^2\rangle}\right)^{\alpha+1}. \qquad (1)$$

We thus have

$$\langle T\rangle = \sum_{k_j} P(k_j)T_{ij} = 1 + N\langle k^{\alpha+1}\rangle\langle k^{-1}\rangle\left(\frac{\langle k\rangle}{\langle k^2\rangle}\right)^{\alpha+1}. \qquad (2)$$

The quantity $\langle k^\theta \rangle$ can be calculated by $\langle k^\theta \rangle = \int_m^{k_c} P(k)k^\theta dk$. We then have $\langle k^{\gamma-1}\rangle = (\gamma - 1)(m^{1-\gamma} - k_c^{1-\gamma})^{-1} \ln(k_c/m)$ and $\langle k^\theta \rangle = (\gamma - 1)(m^{1-\gamma} - k_c^{1-\gamma})^{-1}(k_c^{\theta-\gamma+1} - m^{\theta-\gamma+1})/(\theta - \gamma + 1)$ (for $\theta \neq \gamma - 1$). Substituting these relations into Eq. (2) yields $\langle T \rangle$. Figure 1 shows $\langle T \rangle$ as a function of α for different values of γ. We see that $\langle T \rangle$ is apparently not a monotonic function of α. In fact, $\langle T \rangle$ attains its minimum for $\alpha \approx 0.5$, which can be understood by noting that the packets' searching areas will increase if they are delivered to hubs. As a result, some proper positive value of α helps packets find their destinations more quickly. However, too large values of α make packets traverse among hubs, increasing the traveling time. Note that, since the delivery capacity is unbounded, at each time step each node can deliver all packets in its queue. The value of N_{sp} can thus be determined as $N_{sp} = R\langle T \rangle$.

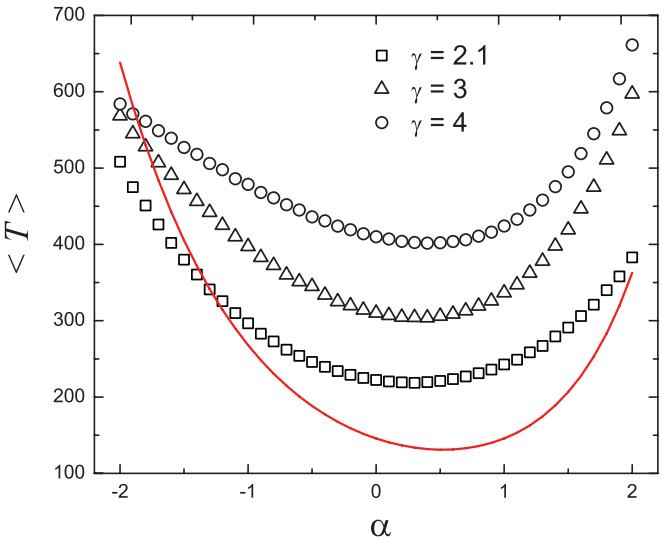

FIG. 1. (Color online) Average traveling time $\langle T \rangle$ of a packet as a function of α for different values of γ. Each data point results from an average over 100 different realizations. The curve is the theoretical prediction derived from Eq. (2) for $\gamma = 3$.

We now simulate the epidemic process driven by traffic dynamics. Figure 2 shows the density of infected nodes ρ as a function of the spreading rate β for different values of α. We observe that for each value of α, there exists an epidemic threshold β_c, beyond which the density of infected nodes ρ is nonzero and increases as β is increased. For $\beta < \beta_c$, the epidemic dies out and $\rho = 0$. Figure 3 shows the dependence of β_c on α for different values of γ. One can observe a nonmonotonic behavior. For different values of γ, β_c is maximized for $\alpha \approx -0.5$ (we have checked that the optimal value of α corresponding to maximal β_c is almost unchanged for $2 < \gamma < 5$). In the following we provide an analytic derivation of the relationship between β_c and α.

Based on the heterogeneous mean-field theory [2], the rate equation for the epidemic dynamics is $d\rho_k/dt = -\rho_k(t) + \beta n_k[1 - \rho_k(t)]\Theta(t)$, where the first term is the recovery rate of infected nodes, and the second term represents the probability that a node with k links belongs to the susceptible class $[1 - \rho_k]$ and gets the infection via packets traveling from infected nodes. The traveling process is determined by the spreading probability β, the number of packets n_k received by a node of degree k at each time step, and the probability $\Theta(t)$ that a packet travels through a link pointing to an infected node. The probability $\Theta(t)$ takes the form $\Theta(t) = \Sigma_k P(k)n_k\rho_k/\Sigma_k P(k)n_k$. This expression, when combined with the stationary solution of ρ_k, $\rho_k = \beta n_k\Theta/(1 + \beta n_k\Theta)$,

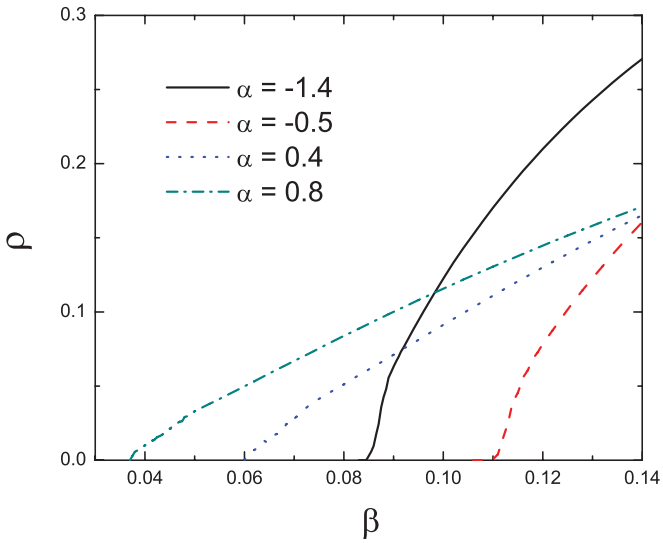

FIG. 2. (Color online) Density of infected nodes ρ as a function of the spreading rate β for different values of α. Each curve results from an average over 100 different realizations. The packet-generation rate is $R = 50$ and networks have $\gamma = 3$.

gives the following self-consistent equation for Θ:

$$\Theta = \frac{1}{\Sigma_k P(k) n_k} \sum_k \frac{P(k)\beta n_k^2 \Theta}{1 + \beta n_k \Theta}. \tag{3}$$

The trivial solution is $\Theta = 0$. In order to obtain a nontrivial solution, we impose the condition

$$\frac{1}{\Sigma_k P(k) n_k} \frac{d}{d\Theta} \left(\sum_k \frac{P(k)\beta n_k^2 \Theta}{1 + \beta n_k \Theta} \right) \Bigg|_{\Theta=0} > 1, \tag{4}$$

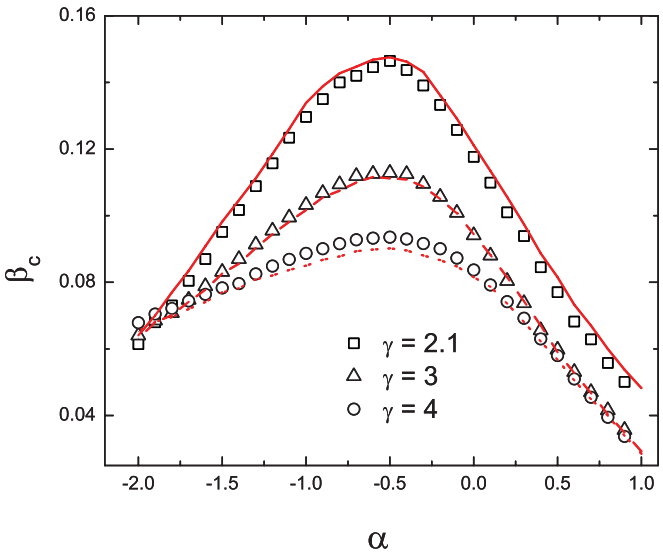

FIG. 3. (Color online) Epidemic threshold β_c as a function of α for different values of γ. The packet-generation rate is $R = 50$. Each data point results from an average over 100 different realizations. The curves are theoretical predictions from Eq. (5). The solid, dashed, and dotted curves correspond to the theoretical prediction for $\gamma = 2.1$, 3, and 4, respectively.

from which the epidemic threshold is obtained as $\beta_c = \langle n_k \rangle / \langle n_k^2 \rangle$. From the analysis in Ref. [10], the relationship between the number of packets n_k received by a node and its degree k is $n_k = Ak^{1+\alpha}$, where A is a constant. Since the delivery capacity is unbounded, at each time step, the total number of delivered packets can be written as $N_{sp} = \sum_k N P(k) n_k$. Using the relation $N_{sp} = R\langle T \rangle$, we obtain $R\langle T \rangle = AN\langle k^{1+\alpha} \rangle$, which gives $A = R\langle T \rangle / (N\langle k^{1+\alpha} \rangle)$ and consequently, $n_k = R\langle T \rangle k^{1+\alpha} / (N\langle k^{1+\alpha} \rangle)$. Substituting this expression into $\beta_c = \langle n_k \rangle / \langle n_k^2 \rangle$, we get the epidemic threshold as

$$\beta_c = \frac{N}{R\langle T \rangle} \frac{\langle k^{\alpha+1} \rangle^2}{\langle k^{2\alpha+2} \rangle}. \tag{5}$$

Utilizing the formulas for $\langle k^{\gamma-1} \rangle$ and $\langle k^{\theta} \rangle$ (for $\theta \neq \gamma - 1$), we can calculate β_c. The comparison between numerical and theoretical values of β_c is shown in Fig. 3 [we use the numerical values of $\langle T \rangle$ in Eq. (5) to better predict numerical results].

When the node delivery capacity is finite, traffic congestion can occur. There now exists a critical packet-generating rate R_c, above which congestion can occur in the sense that, for $R \leqslant R_c$, N_p reaches a constant value, but for $R > R_c$, N_p tends to increase continuously with time. Figure 4 shows the epidemic threshold β_c as a function of R for finite and infinite C. One can see that β_c scales inversely with the packet-generation rate R for unbounded C, as predicted by Eq. (5). However, β_c decreases to a steady value as R increases for bounded C. For $R \leqslant R_c$, β_c is identical for bounded and unbounded delivery capacities, which is understandable because of the absence of congestion in both cases. Our theoretical formula of β_c for the unbounded delivery-capacity case is thus applicable for the corresponding bounded case. However, for $R > R_c$, we observe that the value of β_c is larger for the bounded than the unbounded case, indicating that traffic congestion can suppress the epidemic dynamics. Qualitatively, this can be understood by noting that, once a node becomes congested, the number of packets in its queue will exceed its

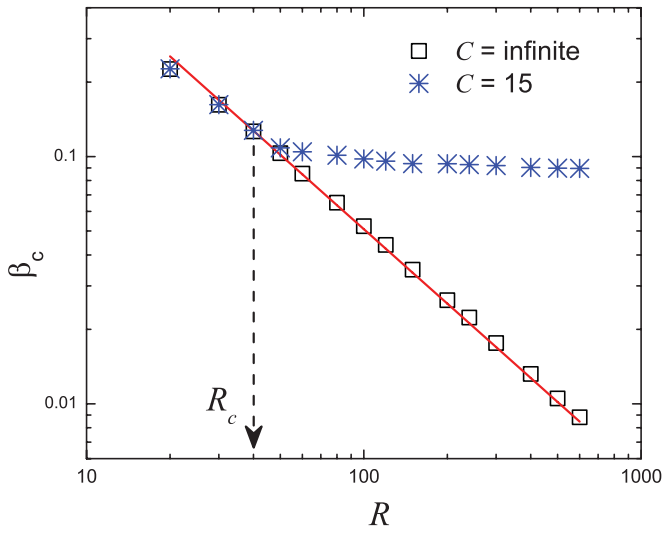

FIG. 4. (Color online) Epidemic threshold β_c as a function of the packet-generation rates R for $C = 15$ and $C \to \infty$. We set $\alpha = -1$ and $\gamma = 3$. The line is the theoretical prediction from Eq. (5). For $C = 15$ and $\alpha = -1$, the critical packet-generating rate is $R_c = 40$.

delivery capacity because the node can deliver only C packets at each time step. A decrease in the number of delivered packets can slow down the process of epidemic spreading. To promote spreading, the rate β must be correspondingly larger than that for the unbounded delivery-capacity case.

In conclusion, we studied the effects of a local-routing scheme on traffic-driven epidemic spreading on scale-free networks. For the case of unbounded node delivery capacity so that the traffic is never congested, we derived a theory to predict the epidemic threshold as a function of a basic parameter characterizing the routing process. The relation was found to be nonmonotonic, where an optimal value of the routing parameter can maximize the epidemic threshold. This means that epidemic spreading can be controlled by fine tuning the routing scheme. For the case of bounded node delivery capacity in the presence of traffic congestion, we argued with numerical support that congestion can in fact be beneficial if one wishes to suppress epidemic spreading. The interplay between traffic dynamics and epidemic spreading turns out to be quite interesting, and the possibility that the former can be used to control and/or suppress the latter can be of broad interest to a number of fields, such as computer science and public health.

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