Suppressing traffic-driven epidemic spreading by efficient routing protocol

Han-Xin Yang\textsuperscript{1} and Zhi-Xi Wu\textsuperscript{2}
\textsuperscript{1}Department of Physics, Fuzhou University, Fuzhou 350108, China
\textsuperscript{2}Institute of Computational Physics and Complex Systems, Lanzhou University, Lanzhou, Gansu 730000, China

Abstract. Despite extensive work on the interplay between traffic dynamics and epidemic spreading, the control of epidemic spreading by routing strategies has not received adequate attention. In this paper, we study the impact of efficient routing protocol on epidemic spreading. In the case of infinite node-delivery capacity, where the traffic is free of congestion, we find that there exists optimal values of routing parameter, leading to the maximal epidemic threshold. This means that epidemic spreading can be effectively controlled by fine tuning the routing scheme. Moreover, we find that an increase in the average network connectivity and the emergence of traffic congestion can suppress the epidemic outbreak.

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1 Introduction

Epidemic spreading [1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12] and traffic dynamics [13, 14, 15, 16, 17, 18, 19, 20] on complex networks have attracted much attention in the past decade. For a long time, the two types of dynamical processes have been studied independently. However, in many cases, epidemic spreading is relied on the process of transportation. For example, a computer virus can spread over Internet via data transmission [21, 22]. 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 [23, 24, 25, 26, 27, 28, 29, 30, 31, 32]. 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 traffic-driven epidemic spreading model [33], 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.

Despite broad interests in traffic-driven epidemic spreading, the control of epidemic spreading by routing strategies has received little attention. In another recent work, 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 [34]. Later, Yang et al. found that epidemic spreading can be effectively controlled by a local routing strategy [35]. In the local routing protocol, each node does not know the whole network’s topological information and the packet is forwarded to a neighboring node $i$ with a probability that is proportional to the power of $i$’s degree [36]. It is noted that in the local traffic routing, the average traveling time of a packet $\langle T \rangle$ is proportional to the network size $N$ [37]. However, in global routing protocols such as the shortest-path routing, $\langle T \rangle$ usually increases approximately logarithmically with $N$ [38]. Thus, from
the view of transmission time, global routing protocols may be superior to local routing
protocols.

So far, the control of traffic-driven epidemic spreading by a global routing protocol
has not been studied. To address the above issue, we consider an efficient routing
strategy proposed by Yan et al. [39]. In the efficient routing protocol, each node in a
network is assigned a weight that is proportional to the power of its degree, where the
power exponent $\alpha$ is a tunable parameter. The efficient path between any two nodes is
corresponding to the route that makes the sum of the nodes’ weight (along the path)
minimal. It has been proved that the traffic throughput of the network can be greatly
improved by employing the efficient routing strategy as compared to the shortest-path
strategy [39]. In this paper, we intend to study how the the efficient routing protocol
affects traffic-driven epidemic spreading. Our preliminary results have shown that there
exists an optimal value of $\alpha$, leading to the maximal epidemic threshold.

The paper is organized as follows. In Sec. 2, we formalize the problem by
introducing the efficient routing strategy into traffic-driven epidemic spreading. In
Sec. 3, we investigate the epidemic spreading on scale-free networks by considering
two cases of node-delivering capacity, i.e., infinite capacity and finite capacity. Finally,
conclusions and discussions are presented in Sec. 4.

2. Model and Methods

Following the work of Meloni et al. [33], we incorporate the traffic dynamics into the
classical susceptible-infected-susceptible model [40] of epidemic spreading as follows.

(i) Efficient routing protocol. 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. For any path between nodes $i$ and $j$, $P(i \rightarrow j) : i \equiv x_1, x_2, \cdots, x_n \equiv j$, we define

\[ L(P(i \rightarrow j) : \alpha) = \sum_{l=1}^{n} k(x_l)^\alpha, \quad (1) \]

where $k(x_l)$ is the degree of node $x_l$ and $\alpha$ is a tunable parameter. For any given $\alpha$,
the efficient path between $i$ and $j$ is corresponding to the route that makes the sum
$L(P(i \rightarrow j) : \alpha)$ minimum. Packets are delivered following the efficient path. When
$\alpha = 0$, the efficient path recovers the traditional shortest path. Once a packet reaches
its destination, it is removed from the system. The queue length of each node 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 $\rho_0$ 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. 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).
Figure 1. The epidemic threshold $\beta_c$ as a function of $\alpha$ for different values of $\langle k \rangle$. The symbols are from numerical simulations. The solid, dashed, and dotted curves correspond to the theoretical prediction from Eq. (2) for $\langle k \rangle = 4, 6$ and 10, respectively. The inset shows the optimal value $\alpha_{\text{opt}}$ of the routing parameter as a function of node degree $\langle k \rangle$. The packet-generation rate $\lambda = 1$. Each data point results from an average over 100 different realizations.

3. Main results and Analysis

In the following, we carry out simulations systematically by employing traffic-driven epidemic spreading on the Barabási-Albert (BA) scale-free networks [41]. The size of the BA network is set to be $N = 2000$. In the case where the node-delivering capacity is infinite ($C \to \infty$), traffic congestion will not occur in the network. When the node-delivering capacity is finite, traffic congestion can occur if the packet-generating rate $\lambda$ exceeds a critical value [35]. Therefore infinite and finite node-delivering capacity are considered respectively in the following sections.

3.1. Infinite node-delivering capacity

Previous studies have shown that there exists an epidemic threshold $\beta_c$, below which the epidemic goes extinct [33, 35]. Figure 1 shows the dependence of $\beta_c$ on $\alpha$ for different values of the average degree $\langle k \rangle$ of the network. We find that for each value of $\langle k \rangle$, there exists an optimal value of $\alpha$, hereafter denoted by $\alpha_{\text{opt}}$, leading to the maximum $\beta_c$. The inset of Fig. 1 shows that $\alpha_{\text{opt}}$ decreases from 0.7 to 0.5 as $\langle k \rangle$ increases from 4 to 16.

According to the analysis in Ref. [33], the epidemic threshold for uncorrelated
Figure 2. The epidemic threshold $\beta_c$ as a function of $\langle k \rangle$ for different values of $\alpha$. The packet-generation rate $\lambda = 1$. Each data point results from an average over 100 different realizations.

Networks is

$$\beta_c = \frac{\langle b_{\text{alg}} \rangle}{\lambda N},$$  \hspace{1cm} (2)

where $b_{\text{alg}}$ is the efficient algorithmic betweenness of a node \cite{42, 43} and $\langle \cdot \rangle$ denotes the average of all nodes. The efficient algorithmic betweenness of a node represents the average number of packets passing through that node at each time step when the packet-generation rate $\lambda = 1/N$. In this paper, the efficient algorithmic betweenness of a node $k$ can be calculated as

$$b^k_{\text{alg}} = \frac{1}{N(N-1)} \sum_{i \neq j} \frac{\sigma_{ij}(k)}{\sigma_{ij}},$$  \hspace{1cm} (3)

where $\sigma_{ij}$ is the total number of efficient paths going from $i$ to $j$, and $\sigma_{ij}(k)$ is the number of efficient paths going from $i$ to $j$ and passing through $k$. From Fig. 1, one can see that the theoretical predictions agree with numerical results qualitatively.

Next, we study the effect of the average degree of the network on the traffic-driven epidemic spreading. Figure 2 shows the epidemic threshold $\beta_c$ as a function of the average degree of the network $\langle k \rangle$ for different values of $\alpha$. From Fig. 2, we find that for each value of $\alpha$, $\beta_c$ increases with $\langle k \rangle$, in contrast to the behavior of spreading dynamics in the absence of traffic \cite{44}. This phenomenon can be understood as follows. An increase in the average degree of the network shortens the average time steps that a packet spends traveling from its source to its destination and decreases the number of packages passing through each node, leading to a decrease in the infection probability of each node.

Figure 3 shows the epidemic threshold $\beta_c$ as a function of the packet-generation rate $\lambda$ for different values of $\alpha$. One can see that $\beta_c$ scales inversely with $\lambda$, as predicted by
Figure 3. The epidemic threshold $\beta_c$ as a function of the packet-generation rate $\lambda$ for different values of $\alpha$. The average degree of the network $\langle k \rangle = 6$. The slope of the fitted line is about -1. Each data point results from an average over 100 different realizations.

Figure 4. The infection probability of nodes $\rho$ as a function of the algorithmic betweenness $b_{\text{alg}}$ for different values of $\alpha$. The average degree of the network $\langle k \rangle = 6$. Each data point results from an average over 100 different realizations.

Eq. (2), indicating that the increase of traffic flow facilitates the outbreak of epidemic. The similar result has also been found in Ref. [33].

An interesting issue is how the algorithmic betweenness $b_{\text{alg}}$ affects the infection probability of nodes $\rho$. Figure 4 features the dependence of $\rho$ on $b_{\text{alg}}$ for different values of $\alpha$. From Fig. 4, one can observe that the algorithmic betweenness of the nodes is positively correlated with the risk of them being infected.
3.2. Finite node-delivering capacity

In the case of finite node-delivering capacity, traffic congestion occurs when the packet-generating rate exceeds a critical value $\lambda_c$, which can be estimated as \[ \lambda_c = \frac{C}{Nb_{\text{alg}}^\text{max}}, \] (4)

where $b_{\text{alg}}^\text{max}$ is the largest algorithmic betweenness of the network.

Figure 5 shows the epidemic threshold $\beta_c$ as a function of the packet-generation rate $\lambda$ for finite and infinite $C$. One can see that when $\lambda \leq \lambda_c$, $\beta_c$ is identical for both cases of the finite and infinite delivery capacities. However, for $\lambda > \lambda_c$, $\beta_c$ is larger in the case of finite capacity than that in the infinite capacity case, indicating that traffic congestion can suppress the outbreak of epidemic. This result is consistent with that in Ref. [33]. The above phenomenon can be explained as follows. Once a node becomes congested, it cannot deliver the total packets in its queue at each time step. A decrease in the number of delivered packets can help nodes reduce the probability of being infected.

Figure 6 shows the epidemic threshold $\beta_c$ as a function of $\alpha$ for different values of the packet-generation rate $\lambda$. One can observe that for small values of $\lambda$ (e.g., $\lambda = 1$ or $\lambda = 1.5$), there exists an optimal value of $\alpha$, leading to the maximal $\beta_c$. However, when $\lambda$ is large enough (e.g., $\lambda = 4$), $\beta_c$ decreases with the increase of $\alpha$. The inset of Fig. 6 shows the critical packet-generating rate $\lambda_c$ as a function of $\alpha$. One can see $\lambda_c$ is maximized by an optimal value of $\alpha$. 

Figure 5. The epidemic threshold $\beta_c$ as a function of the packet-generation rate $\lambda$ for finite and infinite $C$. The average degree of the network $\langle k \rangle = 6$ and the routing parameter $\alpha = 0.7$. The critical packet-generating rate for $C = 10$ is $\lambda_c \approx 0.7$. Each data point results from an average over 100 different realizations.
Figure 6. The epidemic threshold $\beta_c$ as a function of $\alpha$ for different values of the packet-generation rate $\lambda$. The average degree of the network $\langle k \rangle = 6$ and the node-delivering capacity $C = 50$. The inset shows that the critical packet-generating rate $\lambda_c$ as a function of $\alpha$. When $\lambda = 1$, $\lambda > \lambda_c$ for $\alpha < 0.4$ and $\alpha > 2.2$. When $\lambda = 1.5$, $\lambda > \lambda_c$ for $\alpha < 0.5$ and $\alpha > 1.6$. When $\lambda = 4$, $\lambda > \lambda_c$ for all the values of $\alpha$. Each data point results from an average over 100 different realizations.

4. Conclusions and Discussions

In conclusion, we have studied the impact of efficient routing protocol on traffic-driven epidemic spreading. We find that the epidemic threshold increases with the average degree of the network when other parameters are fixed. Besides, we find that nodes with larger algorithmic betweenness are more likely to be infected. Both analytic and numerical results show that, there exists an optimal value of routing parameter, leading to the maximal epidemic threshold. This means that epidemic spreading can be controlled by fine tuning the routing scheme. We hope our results can be useful to understand and control spreading dynamics.

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