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Traffic-driven epidemic spreading on networks of mobile agents

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Abstract – The question as to how traffic or transportation processes on complex networks can shape the dynamics of epidemic spreading is of great interest for a number of areas. We study traffic-driven epidemic spreading on networks of mobile agents by incorporating two routing strategies: random and greedy. We find that for the case of infinite agent delivery capacity, increasing the moving velocity has opposite effects on the outbreak of epidemic spreading for the two routing strategies. However, expanding the communication range among agents can increase the transportation efficiency but counterintuitively suppress epidemic spreading. For finite delivery capacity, the emergence of traffic congestion can effectively inhibit epidemic spreading for both routing strategies. We provide a mean-field theory to explain the numerical findings. Our results can provide insights into devising effective strategies to suppress the spreading of harmful epidemics on time-varying networks.

Introduction. – Epidemic spreading on complex networks [1–10] is a fundamental problem in network science with significant applications. Examples of spreading dynamics that can affect the daily life and functioning of a modern society include the propagation of diseases and large-scale spread of computer viruses. Most previous studies of epidemic spreading focused on the issue of how the network structure or topology can affect the dynamics in terms of quantities of basic interest such as the epidemic threshold.

A closely related problem in the study of complex networks is traffic dynamics [11–20], which is mainly concerned with the transport of packets from one place (source) to another (destination) on the network. Of particular interest is the role that complex-network topology plays in the traffic dynamics with respect to the occurrence of jamming or congestion. For example, as more packets are present on the network, one expects congestion to occur when the packet-generating rate exceeds a critical value and, in fact, most previous studies focused on how the network topology may affect this threshold and how it can be enhanced by perturbation or optimization methods. As traffic dynamics deals with the transport of physical materials or information on the network, one naturally expects a close interplay between traffic dynamics and epidemic spreading. For example, e-mail delivery is perhaps the most common way by which a virus can spread through a computer network, and fast propagation of infectious diseases among different countries is through air transport. However, in the field of network science and engineering, the two types of dynamical processes have been investigated mostly separately.

The recent pioneering work of Meloni *et al*. [21] introduced a theoretical approach to incorporating traffic dynamics in epidemic spreading. In particular, they cast the susceptible-infected-susceptible (SIS) model [22] into a transport scenario where contagion is carried by interaction packets traveling across the network. A susceptible node is more likely to be infected if it receives more packets from infected neighbors. It was found that the epidemic threshold depends on the traffic flow and in fact decreases as the flow increases. Inspired by this work, we recently investigated the effect of local-routing–based traffic dynamics on epidemic spreading [23]. Our finding was that there exists an optimal value of the routing parameter that can maximize the epidemic threshold. Besides the use of local routing protocol in the traffic dynamics, in the existing works [21,23,24], traffic-driven epidemic

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spreading was carried out on static complex networks where the network structure was fixed in time.

In this letter, motivated by the fact that real-world networks for which both traffic and spreading dynamics are of concern are time varying, we address the fundamental problem of traffic-driven epidemic spreading on networks of *mobile agents*. Examples of such dynamics on nonstationary, mobile networks abound, such as the spread of Bluetooth viruses over the network of mobile phone users [25]. A particularly challenging issue concerns the routing protocol on mobile networks. In this regard, in existing works of traffic dynamics on static networks, the types of routing protocols that have been studied include the shortest-path routing [26], a protocol based on the integration of static and dynamic information [27], a local routing strategy [28], and an efficient routing protocol [29]. In a recent work on traffic dynamics, we proposed a random-routing strategy to the traffic dynamic on networks of mobile agents [30]. In the present work, we will then consider a random-routing strategy for traffic-driven spreading dynamics. Our main finding is that, expanding the communication range of mobile agents, a key quantity characterizing the underlying nonstationary network can naturally make the transport dynamics more efficient but counterintuitively suppress infection spreading, in contrast to what occurs in the absence of traffic dynamics. For situations where the network has a finite delivery capacity, the emergence of traffic congestion can effectively inhibit epidemic spreading. To test the generality of our finding, we propose and investigate a different routing strategy, namely the greedy routing, and obtain essentially the same result. Our results provide insights into the role played by traffic dynamics in epidemic spreading and, in particular, suggest that it may be possible to control or manipulate traffic dynamics to effectively suppress spreading. This can be of value to devising emergency control or policy to mitigate or even eliminate spreading of harmful viruses or diseases on networks of critical interest.

Model. – In our model, N agents move on a square-shaped continuous cell of size L with periodic boundary conditions. Agents change their directions θ of motion during the time evolution, but the moving speed v is the same for all agents. Initially, agents are randomly distributed on the cell. After each time step, the position and moving direction of agent i are updated according to

$$x_i(t+1) = x_i(t) + v\cos\theta_i(t), \tag{1}$$

$$y_i(t+1) = y_i(t) + v\sin\theta_i(t), \tag{2}$$

$$\theta_i(t) = \Psi_i, \tag{3}$$

where $x_i(t)$ and $y_i(t)$ are the coordinates of the agent at time t, and Ψ_i is an N-independent random variable uniformly distributed in the interval $[-\pi, \pi]$. Each agent has the same communication radius r. At time t, two agents i and j can communicate with each other only if the distance between them is less than r,

$$\sqrt{[x_i(t) - x_j(t)]^2 + [y_i(t) - y_j(t)]^2} < r. \tag{4}$$

The neighborhood of agent i's at time t is the agents within agent i's communication area at the time.

Rules for implementing the traffic dynamics are as follows. At each time step, the whole system generates R packets, with randomly chosen sources and destinations (agents), and each agent can deliver at most C packets toward its destination. To forward a packet, an agent performs a local search within its communication radius for one of its neighbors at the particular time. If the packet's destination is found within the searched area, it will be delivered directly to the destination. Otherwise, the packet will be forwarded to a selected agent in the searched area according to a specific routing protocol. We consider the following two routing strategies: i) *random routing* by which the packet is forwarded to a randomly chosen agent in the searched area; and ii) *greedy routing* in which the packet is forwarded to the agent whose distance from destination is the shortest. Compared with the random-routing protocol, the greedy-routing protocol requires additional information about agents' positions. For simplicity, we assume that the queue length of each agent is assumed to be unlimited. The first-in-first-out principle holds for the queue. Each newly generated packet is placed at the end of the queue of its source agent. Once a packet reaches its destination, it will be removed from the system.

Epidemic spreading as driven by the above-defined traffic dynamics occurs according to the following rules. After a transient time, as the total number of delivered packets at each time reaches a steady value, an initial fraction ρ_0 of agents 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. For instance, at a given time t, if agent i is infected and a packet is delivered from agent i to a susceptible agent j, then at the next time step, agent j will be infected with probability β. The infected agents are recovered at rate μ (here we set $\mu = 1$ for simplicity).

Results. – In our simulations, we set the number of agents to be $N = 1500$ and the size of the square region to be $L = 10$. To gain insight, we first consider the scenario that each agent's delivering capacity is infinite, $C \to \infty$ so that traffic congestion will not occur in the network. In the case of infinite delivering capacity, the average traveling time of a packet $\langle T \rangle$ is independent of the packet-generation rate R. Figure 1 shows the dependence of $\langle T \rangle$ on the moving speed v and the communication radius r for the random- and greedy-routing strategies, respectively. For a fixed value of r, $\langle T \rangle$ decreases with v for random routing but $\langle T \rangle$ increases with v for greedy routing (fig. 1(a)). For a fixed value of v, $\langle T \rangle$ decreases as r increases for both routing strategies (fig. 1(b)). From fig. 1,

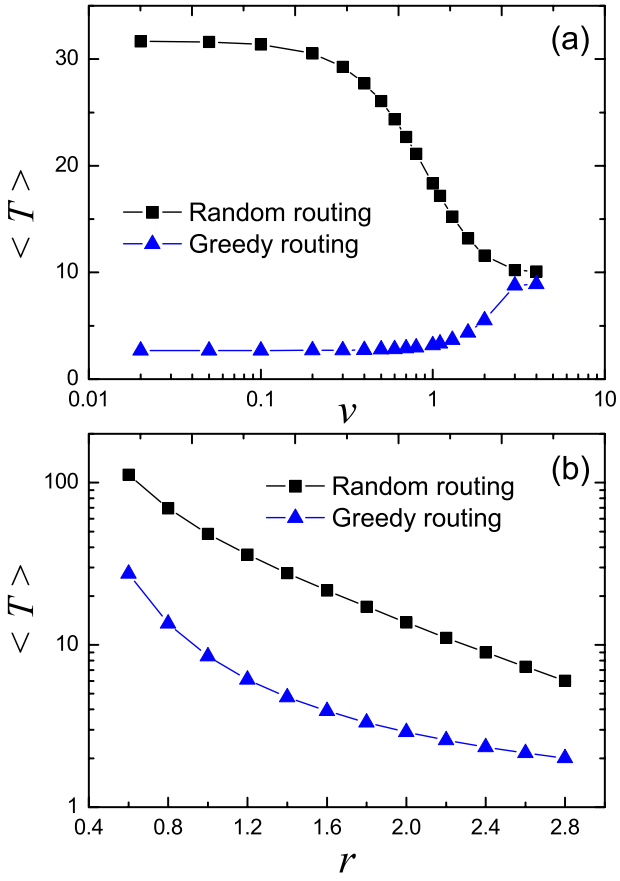

Fig. 1: (Color online) (a) Average traveling time $\langle T\rangle$ as a function of the moving speed v for random routing and greedy routing. The communication radius is $r = 1.8$. (b) Dependence of $\langle T\rangle$ on the communication radius r for different values of v. Each data point results from an average over 50 different realizations, and this will be the case for all subsequent figures.

we can also see that, the average traveling time for greedy routing is shorter than that for random routing with the same values of other parameters. From the viewpoint of transport, greedy routing is then more efficient than random routing.

Figure 2 shows the density of infected agents ρ as a function of the spreading rate β for different values of v and r. We observe that, there exists an epidemic threshold β_c, beyond which the density of infected agents ρ is nonzero and increases as β is increased. For $\beta < \beta_c$, the virus is extinct and $\rho = 0$. From fig. 2, we find that ρ for random routing is higher than that for greedy routing, indicating that random routing makes the network of mobile agents more susceptible to epidemic spreading.

Figure 3(a) shows the epidemic threshold β_c as a function of the moving speed v for random- and greedy-routing strategies. We see that, β_c increases with v for random routing but decreases with v for greedy routing. Figure 3(b) shows the dependence of β_c on the communication radius r for random and greedy routing. One can observe that β_c increases with r for both cases. Figure 3

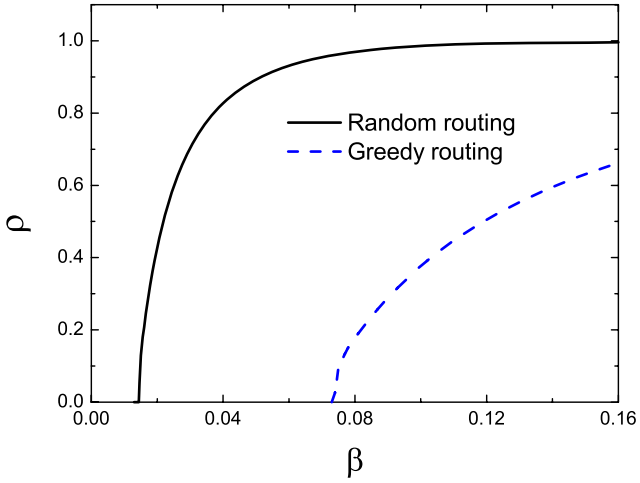

Fig. 2: (Color online) For the idealized case of infinite delivering capacity for each node, density of infected agents ρ as a function of the spreading rate β for random- and greedy-routing strategies. The packet-generation rate is $R = 4000$, and other parameters are $v = 1.1$ and $r = 1.4$. Each curve is a result of averaging over 50 different realizations.

indicates that the epidemic threshold for random routing is smaller than that for greedy routing, demonstrating that greedy routing can suppress the outbreak of epidemic more effectively than random-routing strategy. In the following, we will provide a simple argument for the numerically obtained behavior of the epidemic threshold β_c. The number of packets passing through an agent approximately follows the Poisson distribution for both routing strategies, as we have verified numerically. The mean-field theory is thus applicable to calculating β_c. At each time step, the average number of packets that an agent delivers is $R\langle T\rangle/N$. Following the mean-field approximation [22], the rate equation for the epidemic dynamics can be written as

$$\frac{\mathrm{d}\rho(t)}{\mathrm{d}t} = -\rho(t) + \frac{R\langle T\rangle}{N}\beta\rho(t)[1-\rho(t)]. \tag{5}$$

After imposing the stationarity condition $\mathrm{d}\rho(t)/\mathrm{d}t = 0$, we obtain

$$\rho\left[-1 + \frac{R\langle T\rangle}{N}\beta(1-\rho)\right] = 0. \tag{6}$$

From eq. (6), we obtain the epidemic threshold for the case of infinite agent delivery capacity:

$$\beta_c = \frac{N}{R\langle T\rangle}. \tag{7}$$

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. (7) because of the difficulty in calculating $\langle T\rangle$ theoretically). We see that our simple argument can explain the numerical results of β_c reasonably well.

We next consider the case of finite delivery capacity, where traffic congestion can now occur when the packet-generation rate R exceeds a critical value R_c [12,30].

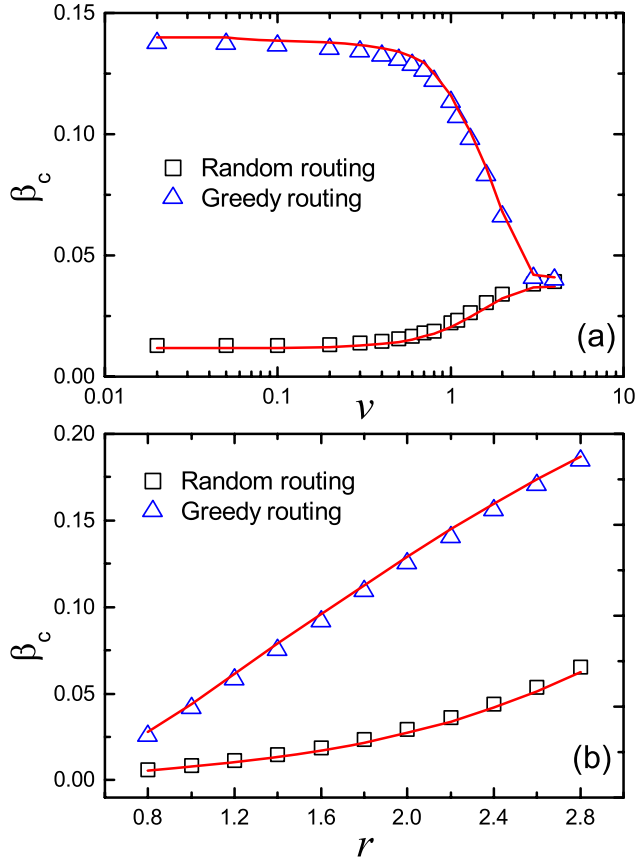

Fig. 3: (Color online) For infinite node delivering capacity and $R = 4000$, (a) epidemic threshold β_c as a function of the moving speed v for random- and greedy-routing strategies, for $r = 1.8$, (b) β_c *vs.* communication radius r for random and greedy routing for $v = 1.1$. The curves are the theoretical prediction from eq. (7).

Figure 4 shows the epidemic threshold β_c as a function of R for two kinds of routing strategies and different values of the delivering capacity C. We see that β_c scales inversely with R for small R or large C for both routing strategies, as predicted by eq. (7). For $C = 10$, β_c decreases to a steady value as R increases for both routing strategies. For $R \leqslant R_c$, β_c is almost the same for the cases of both finite and infinite delivery capacities. However, for $R > R_c$, the value of β_c is larger for $C = 10$ than that for $C \to \infty$, indicating that traffic congestion can suppress epidemic spreading on networks of mobile agents.

In the case of finite delivery capacity, the lower limit of the epidemic threshold can be estimated as follows. For sufficiently large values of R, all agents will become congested and each agent can deliver only C packets at each time step. Equation (5) thus should be modified to be

$$\frac{\mathrm{d}\rho(t)}{\mathrm{d}t} = -\rho(t) + C\beta\rho(t)[1-\rho(t)]. \tag{8}$$

Imposing the stationarity condition, $\mathrm{d}\rho(t)/\mathrm{d}t = 0$ and we obtain the epidemic threshold for the case of finite agent

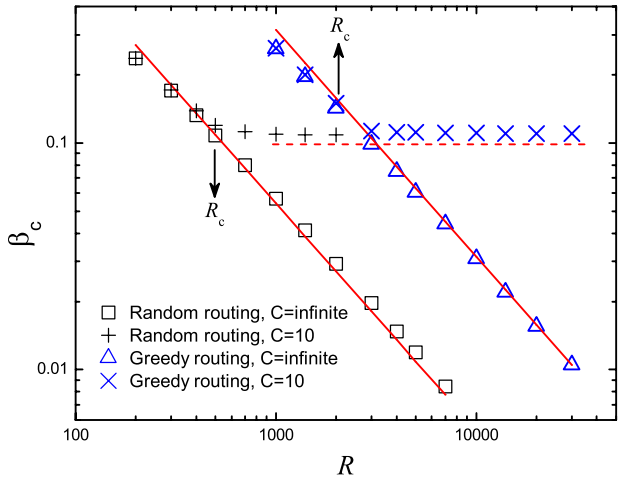

Fig. 4: (Color online) Epidemic threshold β_c as a function of the packet-generation rate R for different routing strategies and different values of agent's delivering capacity C. The parameters are $v = 1.1$ and $r = 1.4$. For $C = 10$, the critical packet-generating rate R_c is about 500 and 2000 for random and greedy routing, respectively. The solid and dashed line are the theoretical prediction from eq. (7) and eq. (9), respectively.

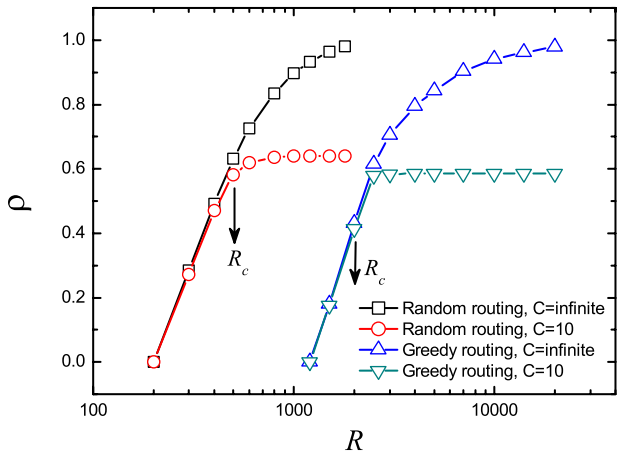

Fig. 5: (Color online) Density of infected agents ρ as a function of the packet-generation rate R for different routing strategies and different values of agent's delivering capacity C. The parameters are $v = 1.1$, $r = 1.4$, and $\beta = 0.2$. For $C = 10$, the critical packet-generating rate R_c is about 500 and 2000 for random and greedy routing, respectively.

delivery capacity:

$$\beta_c = \frac{1}{C}. \tag{9}$$

Figure 4 shows that the theoretical prediction of β_c is consistent well with that of simulation result.

Figure 5 shows the density of infected agents ρ as a function of the packet-generation rate R for two kinds of routings and different values of agent's delivering capacity C. In the case of free flow ($R \leqslant R_c$), ρ is almost the same for finite and infinite delivery capacity. In contrast, in the presence of congestion ($R > R_c$), the value of ρ is lower

for $C = 10$ than that for $C \to \infty$. As R is increased, ρ increases to 1 for infinite delivery capacity. However, for $C = 10$, ρ increases to a bounded value less than 1 as R increases. Combining fig. 4 and fig. 5, we conclude that the emergence of traffic congestion can effectively inhibit epidemic spreading on networks of mobile agents.

Conclusion. – We have studied quantitatively how traffic dynamics can drive epidemic spreading on nonstationary networks of mobile agents for two types of routing strategies: random and greedy. The interplay between epidemic spreading and traffic dynamics has been explored in terms of motion velocity, communication radius and forwarding capacity. We find that the greedy-routing strategy is more effective than the random strategy in enhancing transmission efficiency and suppressing epidemic spreading. In the idealized case of infinite delivery capacity, increasing moving velocity can decrease the epidemic threshold for greedy routing but the opposite is true for random routing. Interestingly, while expanding the communication range favors the transportation of packets, epidemic spreading can be suppressed, in contrast to the behavior of spreading dynamics in the absence of traffic. For finite delivery capacity, traffic congestion can serve to restrain epidemic spreading for both routing strategies. Theoretical insights into these phenomena can be obtained from mean-field theory, especially in terms of the dependence of the outbreak threshold on moving velocity, communication radius and packet-generation rates. The interplay between epidemic spreading and traffic dynamics has gained momentum recently, and the findings presented here can be useful to understand and control spreading dynamics in time-varying complex-networked systems of mobile individuals.

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