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RESEARCH ARTICLE

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Epidemic spreading behavior with time delay on local-world evolving networks

Abstract An improved susceptible-infected-susceptible (SIS) model in the local-world evolving network model is presented to study the epidemic spreading behavior with time delay, which is added into the infected phase. The local-world evolving model displays a transition from the exponential network to the scale-free network with respect to the degree distribution. Two typical delay regimes, i.e., uniform and degree-dependent delays are incorporated into the SIS epidemic model to investigate the epidemic infection processes in the local-world network model. The results indicate that the infection delay will promote the epidemic outbreaks, increase the prevalence and reduce the critical threshold of epidemic spreading. It is also found that local-world size M will considerably influence the epidemic spreading behavior with time delay in the local-world network through large-scale numerical simulations. Simulation results are also of relevance to fight epidemic outbreaks.

Keywords epidemic spreading, time delay, local-world model, complex networks, infection dynamics

1 Introduction

Many natural, social and technological systems can be properly described as complex networks, in which nodes represent the individuals or organizations and links mimic the interactions or connections among them [1–3]. Recently, the significant interest in complex networks could have been stimulated by two seminal works: one is the small-world (SW) effect [4], where the average path length inside the network is very short and increasing slowly (usually logarithmically) with the network size; the other is scale-free (SF) property [5], where the degree distribution displays power-law distribution $P(k) \sim k^{-\gamma}$ with $2 < \gamma \leqslant 3$ for the probability that a randomly selected node has k direct neighbors.

Understanding the evolution of dynamics taking place on complex networked systems is one of our ultimate research goals in this area [1–3]. Among them, an important issue is to study the effect of complex topology on the spreading dynamics. Many realistic applications, such as infectious disease [6], computer virus [7], rumor and information propagation [8] and so forth are all correlated with the epidemics on top of networks [9]. Recent works [10–12] have provided some valuable insights into these problems: for homogeneous networks there exists an epidemic threshold below which the infection cannot initiate a major outbreak; while for heterogeneous networks the critical threshold may tend to zero in the thermo-dynamic limit. Moreover, the dynamical evolution [13,14] of the fraction of infected nodes is also dominated by the topological structure of the network and the time-scale of the initial outbreaks is proportional to the ratio between the first and second moment of the network's degree distribution, i.e., $\tau \sim \langle k \rangle / \langle k^2 \rangle$.

However, all these researches only consider the epidemic dynamics without any time delay. In many real-world networks there are usually some time delays between spreading and response not only for the limited diffusion speed, but also for the competition and congestion among multiple signals [15,16]. Thus, the finite time delay for the information transmission between any two nodes may be very important. During the epidemic propagation, an infected individual can be recovered only after some periods which include the time to be found and sent to the hospital and cured, etc. Xu et al [17] studied the impact of time delay upon epidemic spreading on Watts and Strogatz (WS) [4] and Barabási and Albert (BA) [5] networks. In this paper, we will introduce the time delay into the standard susceptible-infected-susceptible (SIS) epidemic model to

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investigate how it influences the infection propagation behavior on local-world evolving networks [18]. The local-world model displays a transition from the exponential distribution to the power-law distribution and captures an important feature in the real-world network: preferential attachment mechanism that works only within a local-world instead of the whole network, such as communication networks, world trade web and biological networks [18]. A recent work [19] also indicates that numerous statistical characteristics of this model, such as clustering coefficient, diameter, efficiency, error and attack tolerance, are all closely correlated with local-world size (M). Is the epidemic spreading with time delay also related with this parameter? This is our motive and main work in this study. We will integrate the time delay into SIS epidemic dynamics to study the dissemination behavior running on the local-world evolving network.

The rest of this paper is organized as follows. First, we describe the local-world evolving network model briefly in Sect. 2, followed by a depiction of the SIS spreading model with time delay in Sect. 3. In Sect. 4 the epidemic spreading behaviors with the uniform delay and the degree-dependent delay are investigated in detail via numeric simulations. Finally, corresponding conclusions are drawn and further research directions are presented in Sect. 5.

2 Local-world evolving network model

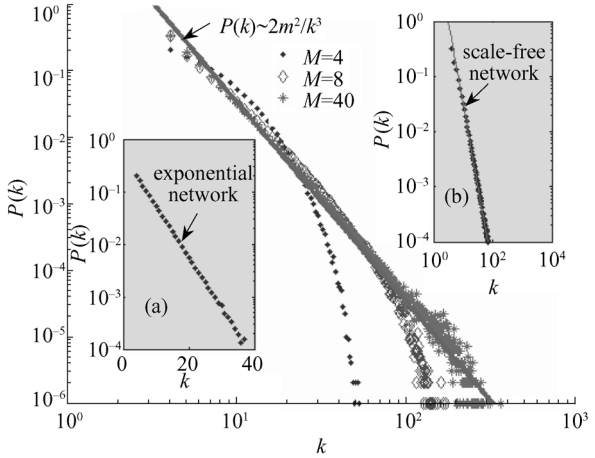
Li and Chen [18] firstly proposed a local-world (LW) evolving network, which captures the localization effect of real-world complex networks. The iterative algorithm of LW model is briefly described as follows:

1) Growth: start with a small number of m_0 nodes and a small number of e_0 links, and add a new node with m links into the network at each time step t. In addition, we should try to make m_0 nodes initially connected.

2) Local preferential attachment mechanism: at each time step t, before every new node is connected to the network, randomly select M nodes as its LW, then add m links between this new node and m nodes in the LW with a preferential attachment with probability $\Pi_{\text{Local}}(k_i)$:

$$\Pi_{\text{Local}}(k_i) = \Pi'(i \in \text{Local-world}) \frac{k_i}{\sum_{j \in \text{Local}} k_j} = \frac{M}{(m_0+t)} \frac{k_j}{\sum_{j \in \text{Local}} k_j}. \quad (1)$$

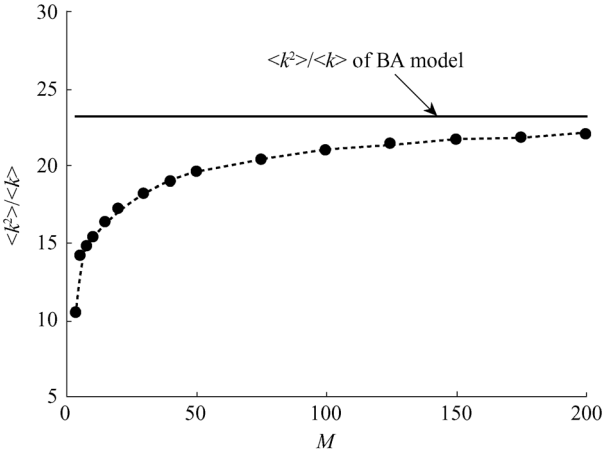
Obviously, the LW size M must lie between m and m_0+t, and there are two limiting cases for the LW model: $M=m$ and $M=m_0+t$. When $M=m$, the LW model is reduced to exponential networks, and its degree distribution decays exponentially as $P(k) \sim e^{-k/m}$. When $M=m_0+t$, the LW model is identical with the BA scale-free network, and its degree distribution follows as $P(k) \sim 2m^2/k^3$. So LW size M can induce a significant effect on the degree distribution of the LW model when M is increased from m to m_0+t. It is clearly seen in Fig. 1 that the degree distribution represents a transition between the exponential and power-law distributions. In Fig. 1, M is increased from 4 to 40 with a constant value of $N=10000$, $m_0=m=4$.



Inset (a): degree distribution in the log-linear scale with M=m=4
Inset (b): degree distribution in the log-log scale with M=40, m=4

Fig. 1 Degree distribution in the log-log scale of the LW evolving networks with $M=4$, 8, 40 and $m=4$ respectively

Furthermore, M also determines the heterogeneity ($\langle k^2\rangle/\langle k\rangle$) of the LW evolving model and it is reported in Fig. 2 that the degree of network heterogeneity grows with M and this result is also consistent with the very recent work of Sun et al. [19]. All these results are averaged over 100 network implementations in Figs. 1 and 2.



The reference line indicates the corresponding result of BA model

Fig. 2 Evolution of heterogeneity ($\langle k^2\rangle/\langle k\rangle$) of LW model with $M=4$, 5, 6, 7, 8,…, 200 respectively

3 SIS epidemic model with time-delay

3.1 Standard SIS model

SIS model is often utilized to describe a class of infectious diseases such as tuberculosis and gonorrhea [6], where the infected individuals can be cured, but may be unluckily infected again. In this model, an individual can only exist in two discrete states, namely susceptible or healthy and infected. The susceptible one can be infected with the probability v if it is connected to the infected individuals, while the infected one can be cured with the probability δ. Therefore, its dynamics can be determined by the following differential dynamics,

$$\frac{\mathrm{d}s}{\mathrm{d}t} = -vis + \delta i, \quad \frac{\mathrm{d}i}{\mathrm{d}t} = vis - \delta i, \tag{2}$$

where i and s denote the fraction of infected and susceptible ones, respectively. Moreover, i and s often satisfy the normalized condition $i+s=1$.

To our surprise, Pastor-satorras et al [10] combined the SIS model with BA heterogeneous networks and found that the critical threshold of SIS model tends to zero in the infinite network size. This new scenario may radically change a lot of drawn results and inspire many relevant researches [11–14] because the epidemic threshold is fundamental to the classical epidemiology.

3.2 SIS model with time-delay

In this paper, the time delay will be added into the above-mentioned SIS epidemic model. The disease propagation with delay is described by the following rules: a susceptible individual at time t will be infected with probability v at time $t+\Delta t$ if it is connected to one or more infected ones, where Δt is the time step of numerical simulations. Meanwhile, infected individuals at time t will be cured with probability δ at time $t+\Delta t+\tau_{\mathrm{Inf}}$, where τ_{Inf} stands for the delay time in the infected phase. We can define the effective spreading rate $\lambda = v/\delta$ and set $\delta = 1$ without loss of generality. We carried out large-scale numerical simulations while synchronously updating the whole network.

We introduce two typical delay regimes into the SIS model, uniform delay and degree-dependent delay. In the first regime, we consider that all the individuals have a uniform delay in the LW networks,

$$\tau_{\mathrm{Inf}} = \tau. \tag{3}$$

In other words, the delay time within individuals is independent of the degree fluctuations in the networks. While for the second one, we assume that the delay for each individual is different and related with its degree, namely, degree-dependent delay,

$$\tau_{\mathrm{Inf}}^{i} = \frac{k_i^{-\alpha}}{\alpha}, \tag{4}$$

where α is a tunable parameter. Obviously, the delay time of the ith node τ_i is inversely proportional to the node degree k_i, that is, the more connections a node has, the less delay time the node takes. It is intuitively conformant to the real-life cases in the social networks, where k_i denotes the extent of the activity of an individual. Those active people receive more attention in society, and they will be in time sent to a hospital for better cure if they are infected. Thus, it will perhaps take less time for them to become healthy again and it means shorter infection delay.

4 Simulation results and analysis

In Fig. 3, we plot the dynamical evolution of the fraction of infected individuals $i(t)$ (also named as infection density) under the uniform delay and degree-dependent delay. Four sub-figures denote various combinations between local-world size M and delay parameters, and are labeled as (a), (b), (c) and (d), respectively. The same representations are also found in Figs. 4 and 5.

It is clearly shown in Fig. 3 that the dynamical evolution of the infection process will quickly stabilize at an equilibrium point after some appropriate relaxation periods. The static prevalence ρ stands for the average fraction of infected individuals in the steady state. In sub-figures (a) and (c), M is set to be 4 and 40 respectively. These two sub-figures plot the evolution of infection density $i(t)$ under the uniform delay time. The results mean that the larger the delay time is, the higher the static prevalence ρ is. Both sub-figures (b) and (d) depict the dynamical evolution under the degree-dependent delay, in which M is also fixed to be 4 and 40 respectively. The simulations indicate that the smaller the tunable parameter α is, i.e., the larger delay time the node takes, the higher the static prevalence ρ is. Here, the effective spreading rate is $\lambda = 0.16$ and the initial infection ratio is 0.0001. From the previous numerical results, we can see that M has also a great influence on the heterogeneity of the LW model network and the heterogeneity increases with M. Thus, the dynamical evolution will more quickly rise to the equilibrium point if M becomes larger and larger. It is distinctly pointed out by the vertical line at time step $t = 10$ in (a) and (c), $t = 50$ in (b) and (d), respectively.

Figure 4 shows the relationship of the static prevalence ρ versus the effective spreading rate λ for $M = 4$ and that of ρ versus $1/\lambda$ for $M = 40$ at two different delay regimes. In sub-figures (a) and (c), it is easily found that the static prevalence ρ increases greatly with the time delay τ_{Inf}, which causes the epidemic threshold λ_C to become smaller in the LW network with $M = 4$ and the scaling effect to get

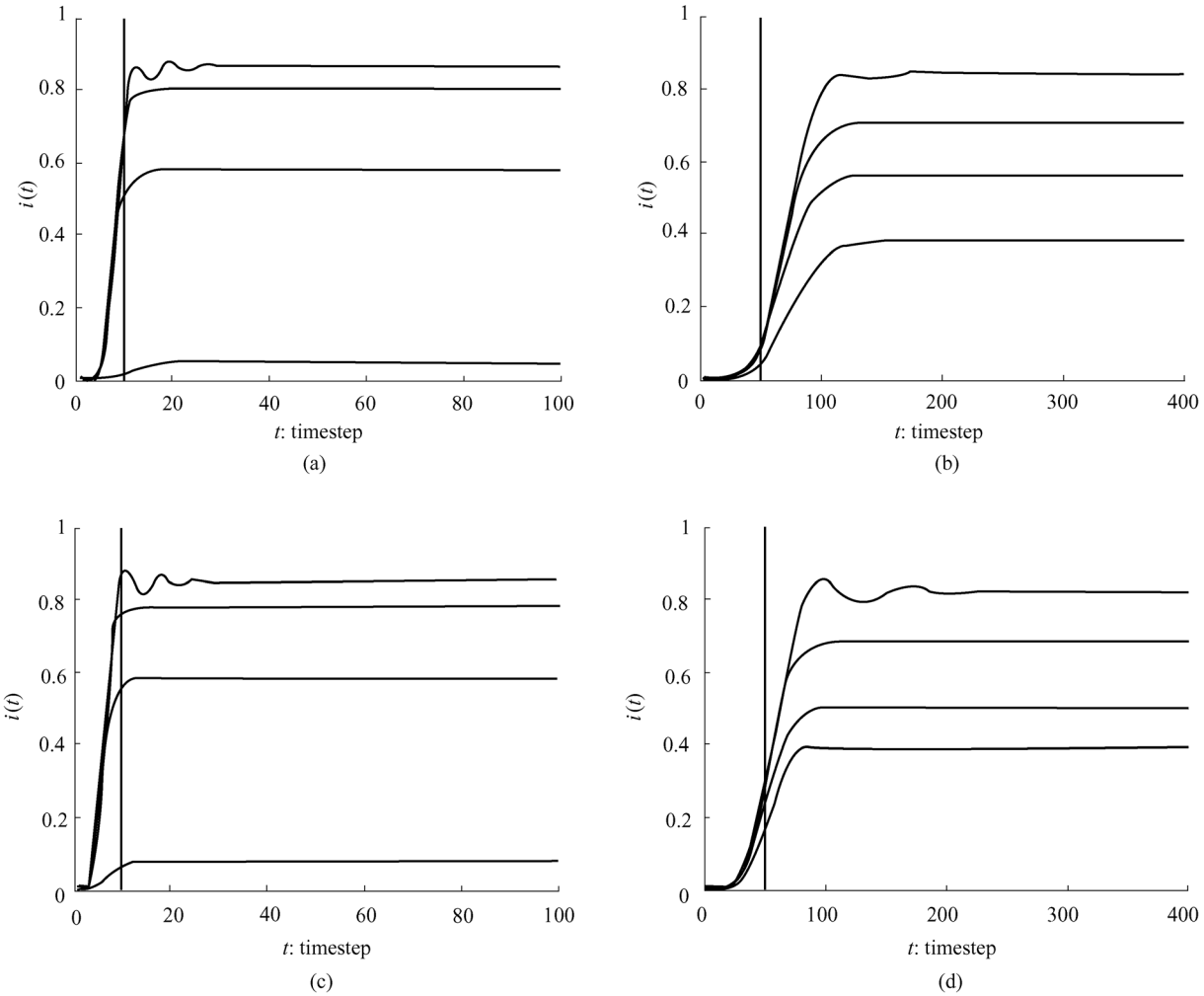


Fig. 3 Dynamical evolution of the fraction of the infected individuals $i(t)$ as time in the LW evolving networks. (a) Effect of the uniform delay time $\tau_{\text{Inf}} = 0, 2, 4, 6$ (from bottom to top) when $M = 4$; (b) effect of the degree-dependent parameter $\alpha = 0.45, 0.35, 0.25, 0.15$ (from bottom to top) when $M = 4$; (c) effect of the uniform delay time $\tau_{\text{Inf}} = 0, 2, 4, 6$ (from bottom to top) when $M = 40$; (d) effect of the degree-dependent parameter $\alpha = 0.45, 0.35, 0.25, 0.15$ (from bottom to top) when $M = 40$

weaker in the LW network with $M = 40$. In sub-figure (b), we can observe that the epidemic threshold becomes smaller in the case $M = 4$ when τ_{Inf} increases. While for sub-figure (d), the scaling effect gets weaker under $M = 40$ when the tunable parameter α decreases from 0.45 to 0.15. The numerical results are qualitatively conformable for these two different delay strategies. In fact, it is clearly seen from Eq. (4) that the delay time of every node is inverse to the tunable parameter α in the second regime. The decrease of α means the increase of the time delay, thus the prevalence is higher.

In the uniform case, the relationship between the static prevalence ρ and delay time τ_{Inf} is illustrated in Figs. 5(a) and 5(c) under two different effective spreading rate $\lambda = 0.16$ (red triangle marks) and 0.24 (green square marks), where M is set to 4 and 40, respectively. For the degree-dependent case, the static prevalence ρ and tunable parameter α are drawn in Figs. 5(b) and 5(d) under the same spreading rate and LW size. Again, it is clearly seen that the static prevalence ρ increases with the augmentation of delay time τ_{Inf} or the reduction of tunable parameter α.

In Fig. 6, we report the threshold λ_{C} as a function of the delay time τ_{Inf} or tunable parameter α for two different time delay regimes. Similarly, the critical threshold λ_{C} decreases with τ_{Inf} in the uniform delay and increases with α for the degree-dependent case. At the same time, the local-world size M leads to the increase of heterogeneity, and thus induces the smaller epidemic threshold. It can be seen from Fig. 6 that the epidemic threshold of $M = 40$ is usually smaller than that of $M = 4$.

In the above-mentioned simulations, LW network parameters are set as $N = 10000$, $m_0 = m = 4$. The initial infected ratio is 0.05 from Figs. 4 to 6, while this ratio is 0.0001 in Fig. 3. Generally, we performed 10 independent network realizations and 20 SIS dynamics with time delay. In addition, from Figs. 3 to 6, we can also see that the LW

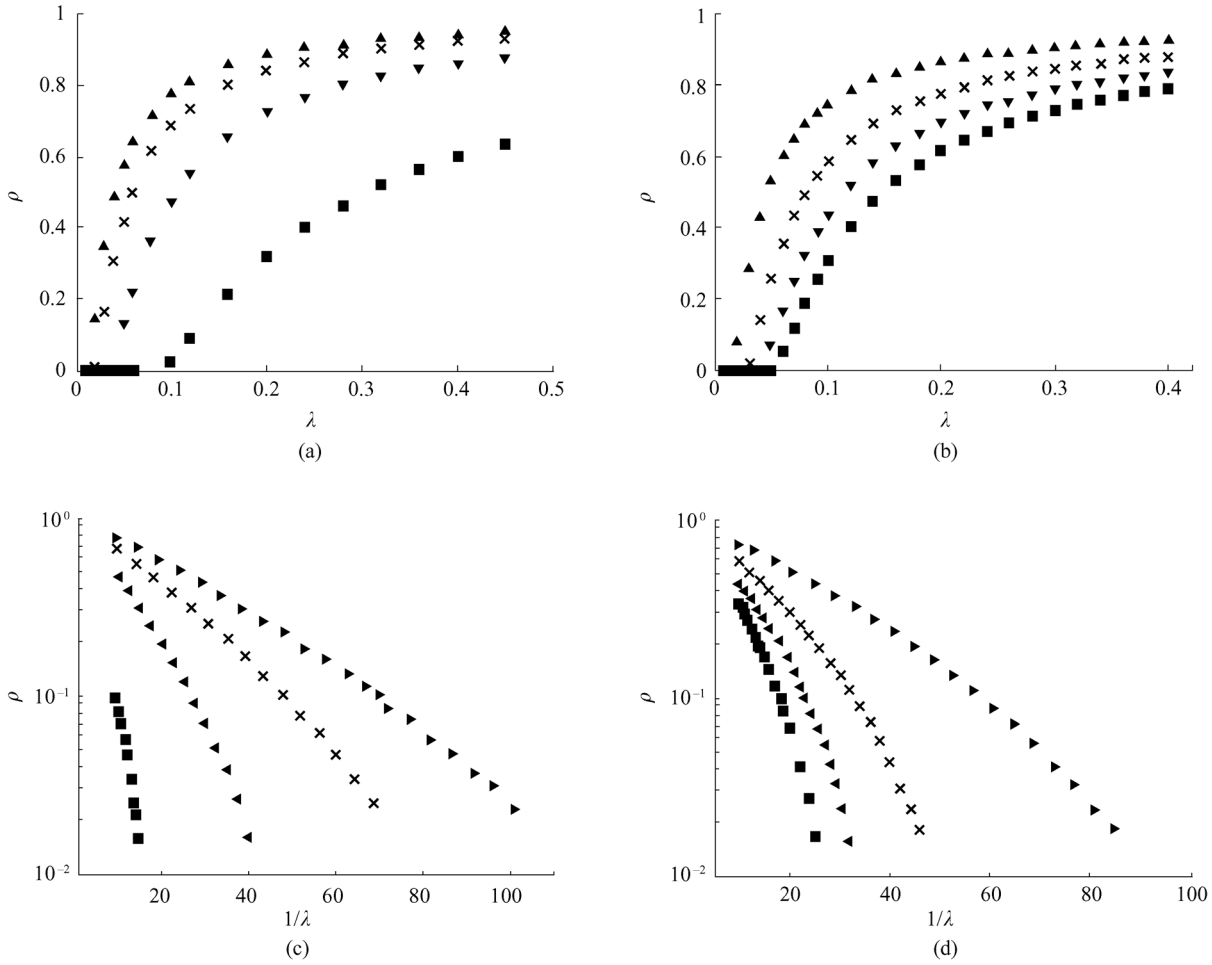


Fig. 4 Relationship between ρ and λ under two different delay schemes. (a) Effect of the uniform delay time $\tau_{\mathrm{Inf}} = 0, 2, 4, 6$ (from bottom to top) when $M = 4$; (b) effect of the degree-dependent parameter $\alpha = 0.45, 0.35, 0.25, 0.15$ (from bottom to top) when $M = 4$; (c) effect of the uniform delay time $\tau_{\mathrm{Inf}} = 0, 2, 4, 6$ (from bottom to top) when $M = 40$; (d) effect of the degree-dependent parameter $\alpha = 0.45, 0.35, 0.25, 0.15$ (from bottom to top) when $M = 40$

size M can have a substantial effect on the epidemic spreading taking place on top of LW networks. Since the degree distribution reduces to the exponential one when $M = m$, the spreading behavior with time delay is similar to that of exponential networks [17]. While $M >> m$, the LW model approaches the BA scale-free networks, correspondingly the propagating behavior with time delay is approximately identical to that of the BA model [17]. That is to say, the heterogeneity of the network structure will become larger as M increases, thus the epidemic spreading behavior on top of LW networks will also quicken.

Based on the above-mentioned analyses and simulations, some control measures are necessary when we are faced with epidemic diseases. First, we should take effective steps to cure the disease as soon as possible because the delay in the infected phase might promote its outbreak. Second, we should try to constrain the infected individuals' contact ranges, namely, to decrease the local-world (M) of the newly added vertices. We can modify the topology of the contact network, such as quarantine the infected patients or computers with virus, to slow down the dynamical contagion process and control further spreading of the disease.

5 Conclusions

In this paper we have studied the epidemic spreading behavior with time delay on complex dynamical networks with local preferential attachment. The results indicate that the infection delay will greatly push forward the epidemic outbreaks and increase the static infection prevalence. So it is necessary for us to find and cure the infective ones as soon as possible, and it requires that we take immediate measures to decrease the delay time of the disease. Thus, it is of high importance to control the diffusion of infectious diseases in the initial outbreaks for highly heterogeneous networks. Furthermore, it is verified via simulations that the local-world size M also affects the behaviors of epidemic spreading in the LW network,

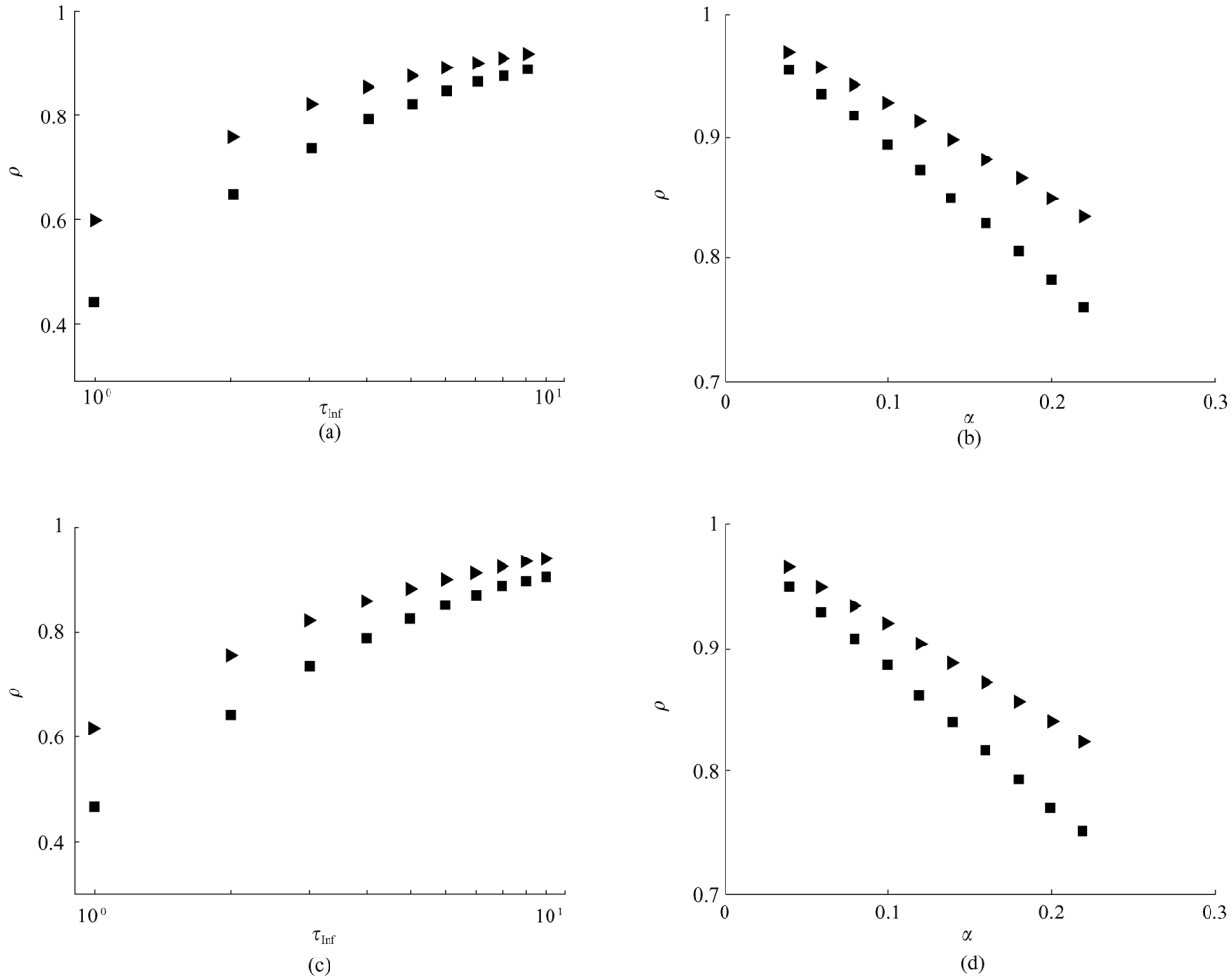


Fig. 5 Static prevalence as a function of delay time parameters. (a) Static prevalence ρ vs. τ_{Inf} in the uniform case when $M = 4$; (b) static prevalence ρ vs. α in the degree-dependent case when $M = 4$; (c) static prevalence ρ vs. τ_{Inf} in the uniform case when $M = 40$; (d) static prevalence ρ vs. α in the degree-dependent case when $M = 40$

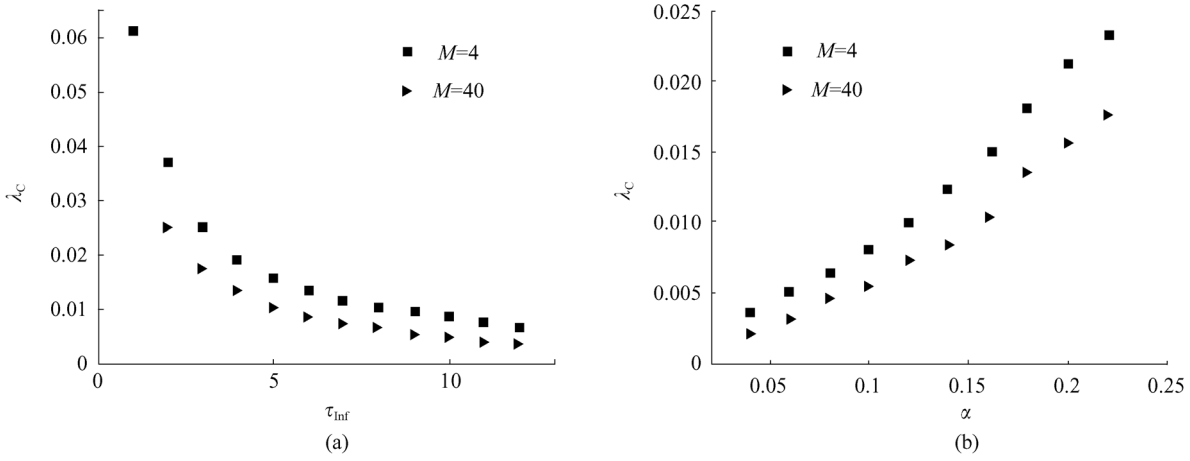


Fig. 6 Critical epidemic threshold as a function of delay parameters. (a) Threshold vs. τ_{Inf}; (b) threshold vs. degree-dependent parameters α

and this prompts that we should try to confine or quarantine the infected individuals when we are combating epidemic or pandemic outbreaks.

Current analyses and results provide an intuitive description of the spreading phenomena with time delay in complex networks, and it will help us to understand

various real-world propagation mechanisms and behaviors. However, all results are acquired by large-scale numerical simulations. The theoretical analyses are worth exploring in the future since time delay is a ubiquitous phenomenon in the natural and engineering fields.

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