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ScienceDirect
Progress in Natural Science 18 (2008) 763–768
Progress in Natural Science
www.elsevier.com/locate/pnsc

Short communication

Epidemic spreading behavior in local-world evolving networks

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Received 31 October 2007; received in revised form 21 November 2007; accepted 23 November 2007

Abstract

The susceptible–infected–susceptible (SIS) epidemic model is presented which can be used to study the epidemic spreading behavior in the local-world evolving network model. Local-world evolving model displays a transition from the exponential network to the scale-free network with respect to the connectivity distribution. From theoretical analyses and numerical simulations, we find that the epidemic spreading behavior on local-world networks also takes on some kind of transitional behaviors. The transitional behavior is further verified by comparing the spreading behavior of local-world network with that of random and scale-free networks. Some feasible control strategies are also proposed to keep from the epidemic spreading on networks.
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Keywords: Epidemic spreading; Local-world model; Complex networks; Dynamics of systems

1. Introduction

Many social, biological and technological systems can be properly characterized by complex networks, in which nodes represent individuals or organizations and links mimic the interactions or connections among them [1–5]. Two seminal events motivate people's interests in complex networks: one is the small-world (SW) [6] effect, i.e. the average path length inside the network is very short and increasing slowly (usually logarithmically) with the network size; the other is the scale-free (SF) [7] property, i.e. the degree distribution displays power-law distribution $P(k) \sim k^{-\alpha}$ with $2 < \alpha \leqslant 3$, where $P(k)$ stands for the probability that any node has k links to other nodes.

Currently, many researchers from various fields have proposed multiple models of networks that typically seek to explain either how networks come to have the observed structure, or what the expected effect of that structure will be. Li and Chen [8] presented a local-world concept in modeling complex network, i.e. local-world evolving model. Local-world network model captures an important feature in the real-world network: preferential attachment mechanism works only within a local world instead of the whole network-wide, such as communication networks, world trade web, and biological networks [8].

Understanding the dynamic evolution on complex networked systems is one of our ultimate goals in this area [1–5]. Among them, an important issue is to study the effect of complex topology on the spreading dynamics. Many realistic applications, such as infectious disease [9] and computer virus [10], are correlated with the epidemics taking place on networks [11]. Recent studies [12–16] have opened out some valuable insights into these problems.

However, all these studies only consider the epidemic dynamics in the SW and SF complex network model. Since the local-world model network displays a transition from the exponential distribution to the power-law distribution, this model can capture some ubiquitous characteristics in the real complex systems. Therefore, it is necessary for us

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doi:10.1016/j.pnsc.2007.11.018

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to investigate the epidemic spreading behavior on this model.

In this paper, we use the SIS epidemic model to investigate the epidemic spreading behavior in local-world evolving networks. Large-scale numerical simulations agree well with the mean-field analysis. In addition, some feasible containment strategies are also put forward.

2. Local-world evolving network model

Li and Chen [8] first propose 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:

(i) *Growth:* Start with a small number m_0 of nodes and small number e_0 of links, and add a new node with m links connecting to the network at each time step t. Generally, we should also ensure that the m_0 nodes are connected at the initial state.

(ii) *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 $\Pi_{\text{Local}}(k_i)$:

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

Obviously, the LW size M must lie within $m - m_0 + t$, and there are two limiting cases for the LW model: $M = m$ and $M = m_0 + t$. When $M = m$, LW model reduces to exponential networks, and its degree distribution decays exponentially as $P(k) \sim e^{-k/m}$. When $M = m_0 + t$, LW model is entirely the same as the Barabási and Albert (BA) [7] SF network, and its degree distribution follows $P(k) \sim 2m^2/k^3$. That is to say, the LW size M can induce a significant effect on the degree distribution of the LW model when M increases from m to $m_0 + t$. We can clearly see from Fig. 1 that the degree distribution represents a transition between the exponential and power-law distributions. In Fig. 1 M increases from 4 to 40 with a constant value of $m = 4$ and $m_0 = 4$, $N = 10{,}000$. The result is averaged over 100 network implementations.

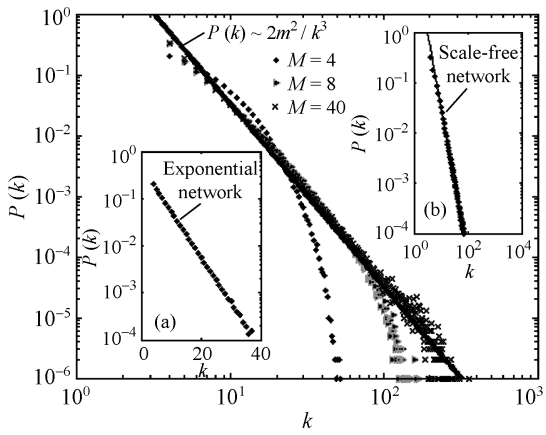

Fig. 1. Degree distribution comparison in the log–log scale of the LW evolving networks with $m = 4$ and $M = 4$, 8, and 40, respectively. The inset (a) degree distribution in the log-linear scale with $M = m = 4$, the inset (b) degree distribution in the log–log scale with $M = 40$ and $m = 4$.

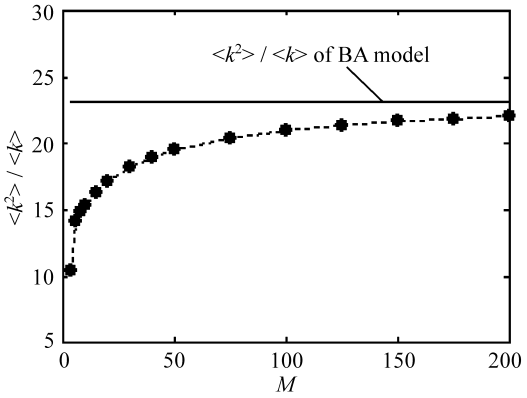

Fig. 2. Evolution of heterogeneity of LW model with $M = 4$, 5, 6, 7, 8,...,200, respectively. The reference line indicates the corresponding result of BA model.

In addition, M also determines the heterogeneity ($\langle k^2\rangle/\langle k\rangle$) of LW evolving model. Fig. 2 shows that the degree of network heterogeneity grows with M. The very recent work [17,18] also indicates that M is relevant to various statistical characteristics of LW model network, such as clustering coefficient, diameter, efficiency, error or attack tolerance, and synchronizability.

3. SIS epidemic dynamics on LW model

SIS model assumes that the individual can only exist in two discrete states: susceptible (or healthy) and infected. A susceptible individual will be infected with the probability β if it is connected to one or more infected nodes, and the infected one will be cured with the probability γ. Thus, the individuals will still run stochastically through the cycles: susceptible → infected → susceptible.

We can see from Section 2 that the LW network displays high heterogeneity when local-world size $M > m$. Therefore, it is necessary to write down the infection equation for various $i_k(t)$. In SIS model, the equation can be described as

$$\frac{di_k(t)}{dt} = \beta k[1 - i_k(t)]\Theta_k(t) - \gamma i_k(t) \quad (2)$$

where β is the spreading rate, γ is the curing rate, k is the degree of the vertex, the densities of infected and susceptible individuals are $i_k(t)$ and $[1 - i_k(t)]$, respectively. $\Theta_k(t)$ is the probability that the neighbor of any node with degree k is infected.

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For uncorrelated heterogeneous networks [11], $\Theta_k(t)$ is independent of the degree of vertex, and every infected neighbor may be the initial seeds (infected at $t=0$) or be infected at $t>0$. Thus, $\Theta_k(t)$ equals to the sum of the following two terms:

$$\Theta_k(t)=\Theta(t)=\frac{\sum_{k'}k'P(k')i_{k'}(0)}{\langle k\rangle}+\frac{\sum_{k'}(k'-1)P(k')[i_{k'}(t)-i_{k'}(0)]}{\langle k\rangle} \quad (3)$$

where $\langle k\rangle$ is the average degree and becomes the proper normalization factor dominated by the total edges.

Differentiating Eq. (3), the following equation is acquired:

$$\frac{\mathrm{d}}{\mathrm{d}t}\Theta(t)=\frac{\sum_{k'}(k'-1)P(k')\frac{\mathrm{d}}{\mathrm{d}t}i_{k'}(t)}{\langle k\rangle} \quad (4)$$

Neglecting the terms of order $O(i^2)$ of Eq. (2) and simplifying the result, we obtain

$$\frac{\mathrm{d}i_k(t)}{\mathrm{d}t}\approx(\beta k-\gamma)\Theta(t) \quad (5)$$

Then substituting Eq. (5) into Eq. (4), we have

$$\frac{\mathrm{d}\Theta(t)}{\mathrm{d}t}\approx\frac{\sum_{k'}(k'-1)P(k')(\beta k'-\gamma)\Theta(t)}{\langle k\rangle}=\frac{\beta\langle k^2>-(\beta+\gamma)\langle k\rangle+\gamma}{\langle k\rangle}\Theta(t) \quad (6)$$

Combining Eq. (6) with Eq. (5) to solve them in the case of the uniform initial condition $i_k(t=0)=i_0$, we can acquire the analytical solution of Eq. (2)

$$i_k(t)\approx i_0\left[\frac{(\beta k-\gamma)\langle k\rangle}{\beta\langle k^2\rangle-(\beta+\gamma)\langle k\rangle+\gamma}\left(\mathrm{e}^{\frac{t}{\tau_H}}-1\right)+1\right] \quad (7)$$

Thus, the total infection density is

$$i(t)=\sum_k P(k)i_k(t)\approx i_0\left[\frac{\beta\langle k\rangle^2-\gamma\langle k\rangle}{\beta\langle k^2\rangle-(\beta+\gamma)\langle k\rangle+\gamma}\left(\mathrm{e}^{\frac{t}{\tau_H}}-1\right)+1\right] \quad (8)$$

where $\tau_H=\frac{\langle k\rangle}{\beta\langle k^2\rangle-(\beta+\gamma)\langle k\rangle+\gamma}$.

For LW networks, M determines the heterogeneity of the network and $\frac{\langle k^2\rangle}{\langle k\rangle}$ increases with M as shown in Fig. 2. According to Eq. (8), the time scale of the epidemic outbreak τ_H is inversely related to $\frac{\langle k^2\rangle}{\langle k\rangle}$ at the initial stage and so τ_H will decrease with M. This result means that the dynamical evolution will become much quicker for larger local-world size M. Moreover, the dynamical process will also infect more quickly for higher β or lower γ. At the same time, the infection density in the steady state (static prevalence) will be augmented as β increases or as γ decreases. All these theoretical analyses will be verified by the numerical results in the following section.

4. Numerical simulations

4.1. Simulation of epidemic spreading in LW networks

Fig. 3 shows the effect of various infection rates on the dynamic spreading behavior under the constant cure rate. Larger infection rate will accelerate the dynamical evolution and increase the infection density during the steady state. Fig. 3(a) and (b) represent the epidemic processes for $M=4$ and 40, respectively.

We can also find from Fig. 3 that the local-world size M will influence the dynamic spreading process under the same infection and cure rate. The heterogeneity of LW network increases with M, and thus the time scale τ_H becomes smaller and the epidemic process will infect more quickly during the initial time. At a specified time step the infected fraction will be larger which can be seen clearly at $t=500$ in Fig. 3. From bottom to top the infection rate β is set to be 0.001, 0.005, and 0.025 respectively, while the cure rate γ is fixed to be 0.005.

Fig. 4 plots the effect of various cure rates on the dynamic spreading behavior under the constant infection rate. Larger cure rate will slow down the dynamical evolution and decrease the infection density during the steady state. Fig. 4(a) and (b) represent the epidemic processes for $M=4$ and 40, respectively.

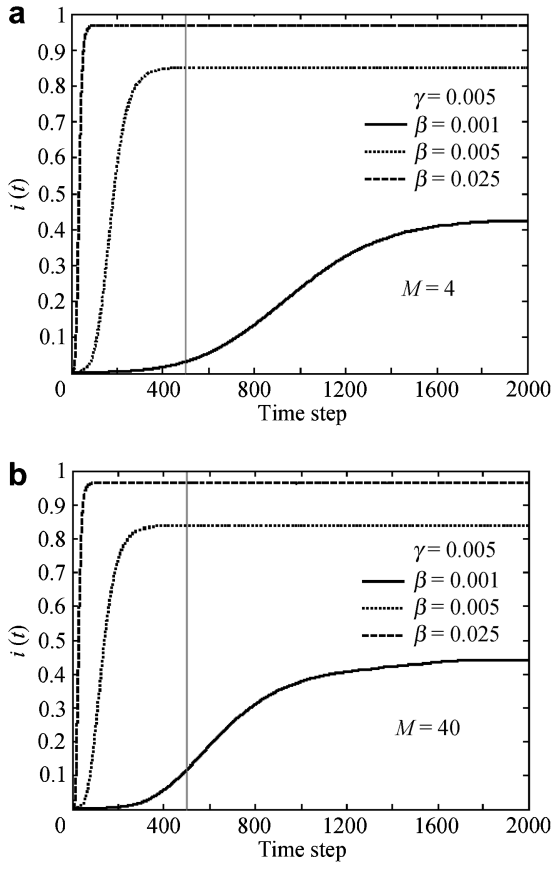

Fig. 3. Effect of various infection rates on dynamic spreading processes of LW network under the same cure rate. (a) $M=4$ and (b) $M=40$.

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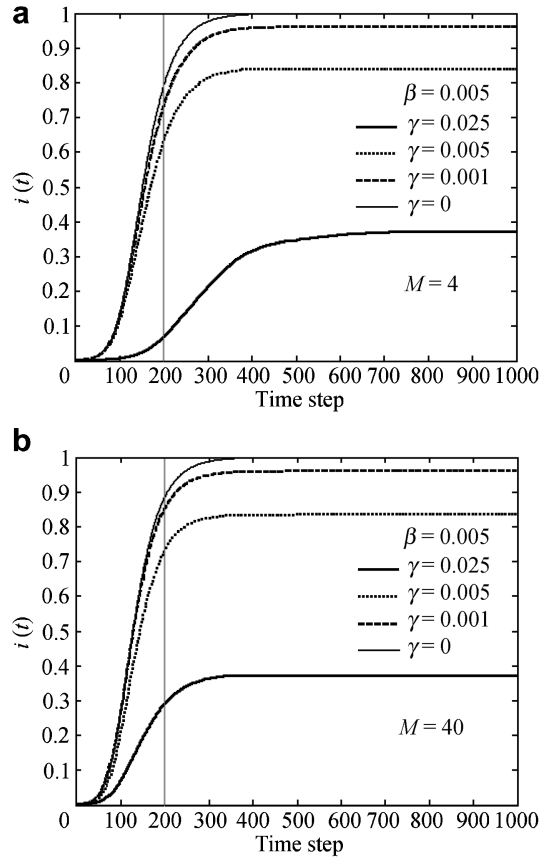

Fig. 4. Effect of various infection rates on dynamic spreading processes of LW network under the same cure rate. (a) $M = 4$ and (b) $M = 40$.

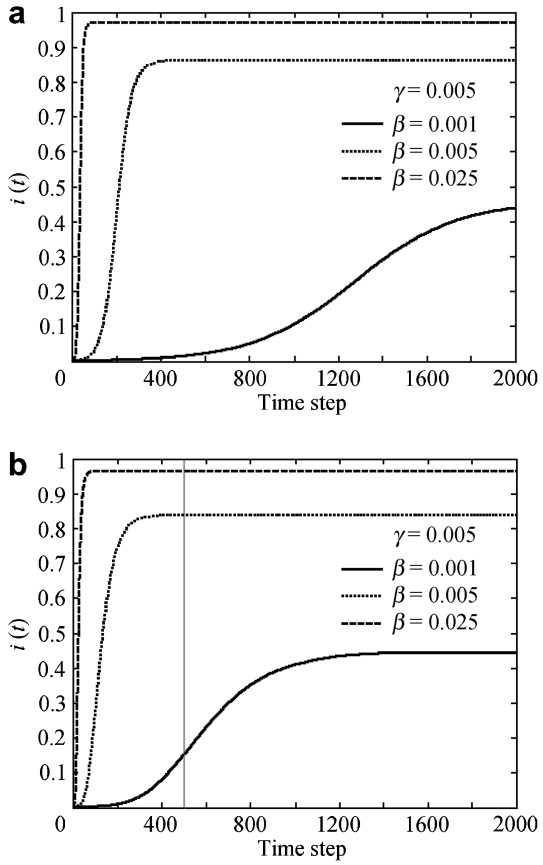

Fig. 5. Effect of various infection rates on dynamic spreading processes of ER (a) and BA (b) network under the same cure rate.

Similarly, M will also affect the dynamic process of epidemic spreading in the LW model network. It is distinctly illustrated at $t = 200$ in Fig. 4. From bottom to top the cure rate γ is set to be 0.025, 0.005, 0.001, and 0 respectively, while the infection rate β is fixed to be 0.005.

4.2. Simulation of epidemic spreading in random and SF networks

Since the degree distribution of LW network displays the transition from the exponential to power-law distribution, we will compare the SIS spreading behavior of LW model with that of Erdös and Rényi (ER) [19] random network and BA [7] SF model.

In Fig. 5, we illustrate the dynamical evolution of the fraction of infected vertices (infection density) in ER and BA model with the same SIS parameters as that in Fig. 3. Obviously, when $M = m$ (e.g. $M = m = 4$), LW model displays the exponential distribution and its spreading behavior is similar to that of ER model. While $M \gg m$ (e.g. $M = 40$ and $m = 4$), LW model approaches the BA SF networks, and correspondingly the epidemic behavior is approximately identical to that of BA model. All these results can be easily acquired by comparing Figs. 3 and 5.

In Fig. 6, we give the dynamical evolution of infection density in ER and BA model with the same SIS parameters as that in Fig. 4. In parallel, when $M = m = 4$, we can see that Fig. 4(a) can be approximated to Fig. 6(a). While $M = 40$ and $m = 4$, Fig. 4(b) is almost identical to Fig. 6(b).

From Figs. 3–6, the simulation parameters of LW network are set to be $N = 10{,}000$, $m = 4$, and $m_0 = 4$. The numerical results are averaged over 10 independent network implementation and 20 SIS dynamics. And all these numerical results are consistent with the theoretical analyses in the previous section.

4.3. The epidemic threshold of the LW network

Pastor-Satorras et al. [12] observed that whatever the spreading rate be in BA network the infection would be widespread, eventually leading to the absence of any epidemic threshold below which the infection cannot initiate a major outbreak. Moreno et al. [14] further point out that the epidemic threshold of SIS or SIR (susceptible–infective–removed) model in complex network (such as SW and SF networks) is proportional to $\frac{\langle k \rangle}{\langle k^2 \rangle}$, which tends to 0 for the BA SF model in the thermodynamic limit. Now that the heterogeneity $\frac{\langle k^2 \rangle}{\langle k \rangle}$ of LW networks increases with the local-world size M, the epidemic threshold of LW networks will decrease with M, which is clearly seen from

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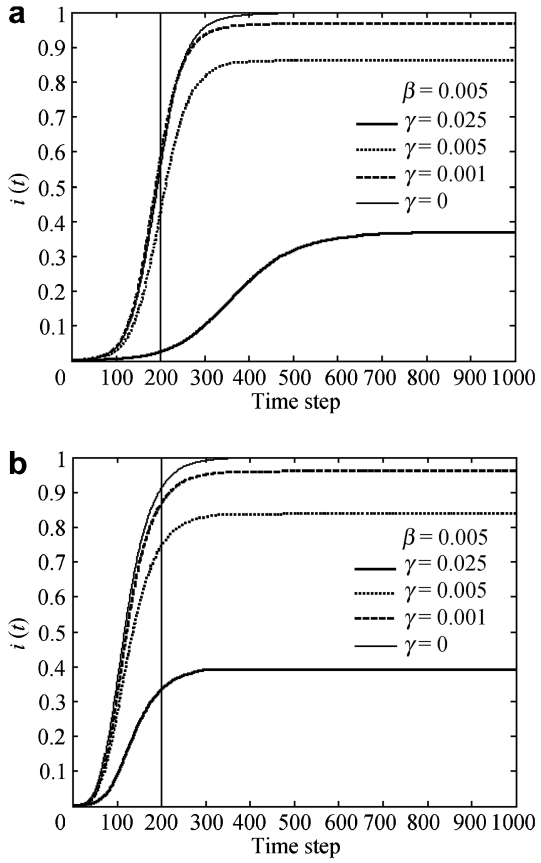

Fig. 6. Effect of various cure rates on dynamic spreading processes of ER (a) and BA (b) network under the same infection rate.

Fig. 7. In the simulation, LW network parameters are preset to be the same as those of Fig. 2.

4.4. Control of the epidemic spreading in complex networks

According to the above-mentioned theoretical analyses and numerical simulations, we should take effective strategies to control the infectious disease or computer virus in complex networks. At first we can see that SIS dynamics is almost instantaneous during the early stage of the epidemic outbreaks, and so we should take immediate control measures as soon as possible. Second, we should try to improve the health-care to increase the cure rate and decrease the infection rate. At last we can constrain the individuals' contact ranges, namely, to decrease the local world (M) of the newly added vertices. That is to say, we can modify the topology of the contact network, such as quarantining the infected patients or computers with virus, to slow down the dynamical contagion process and control the further spreading of the disease.

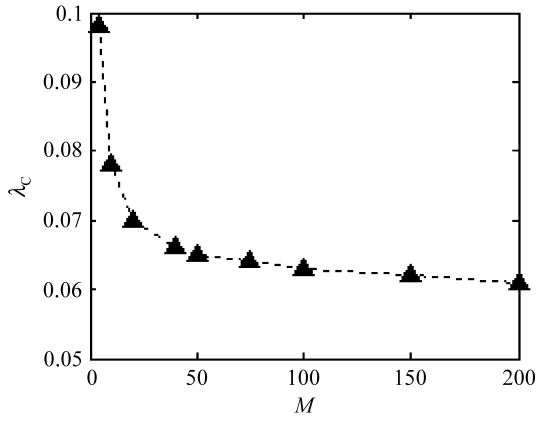

Fig. 7. Effect of M on the epidemic threshold of LW network.

5. Conclusions

In this paper we have studied the epidemic spreading behavior in complex networks with local preferential attachment. Theoretically, the dynamical evolution of infected individuals is correlated with the infection rate, the cure rate and the heterogeneity of LW networks ($\frac{\langle k^2\rangle}{\langle k\rangle}$), which is determined by the local-world size M. Large-scale simulations are carried out to verify that the local-world size M determines the transitional behaviors of epidemic spreading in the LW network. In addition, M also dominates the epidemic threshold of the LW network.

The results indicate that the epidemic outbreaks are almost instantaneous and so it is necessary for us to find and cure the infective ones as soon as possible. Improving the health-care and quarantining the infected individuals is also of high importance to control the diffusion of infectious diseases for highly heterogeneous networks.

The current analyses and simulations provide an intuitive description of the spreading phenomena in complex networks, and it will help us further understand various real-world propagation mechanisms and dissemination behaviors. Furthermore, it will be very important and meaningful for us to assign the time step to be the specific time (such as an hour, a day, and a week), and further study is needed in the future.

Acknowledgements

This work was partly supported by the National Natural Science Foundation of China (Grant Nos. 60774088 and 60574036), the Research Fund for the Doctoral Program of China (Grant No. 20050055013), the Program for New Century Excellent Talents in University of China (NCET) and the Science and Technology Research Key Project of Education Ministry of China (Grant No.107024).

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