Epidemic propagation on adaptive coevolutionary networks with preferential local-world reconnecting strategy*

Song Yu-Rong(宋玉蓉)†, Jiang Guo-Ping(蒋国平), and Gong Yong-Wang(巩永旺)

College of Automation, Nanjing University of Posts and Telecommunications, Nanjing 210003, *China*

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In the propagation of an epidemic in a population, individuals adaptively adjust their behavior to avoid the risk of an epidemic. Differently from existing studies where new links are established randomly, a local link is established preferentially in this paper. We propose a new preferentially reconnecting edge strategy depending on spatial distance (PR-SD). For the PR-SD strategy, the new link is established at random with probability p and in a shortest distance with the probability $1-p$. We establish the epidemic model on an adaptive network using Cellular Automata, and demonstrate the effectiveness of the proposed model by numerical simulations. The results show that the smaller the value of parameter p, the more difficult the epidemic spread is. The PR-SD strategy breaks long-range links and establishes as many short-range links as possible, which causes the network efficiency to decrease quickly and the propagation of the epidemic is restrained effectively.

Keywords: adaptive networks, epidemic dynamics, network dynamics, cellular automata, local-world reconnecting mechanism

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1. Introduction

In complex networks, the influence of node dynamics on networks[1,2] and the formation of complex topologies[3,4] have been investigated for more than one decade. In many real networks, there are complicated mutual interactions and an influence between the behavior of nodes and the topology of the network. For instance, in the epidemic spreading in population, an individual is regarded as a node, and the social relationship between individuals as an edge of the network. The topology of the social network affects the epidemic spreading.[1,2,5–7] In scale-free networks, the absence of an epidemic threshold has been observed. Compared with the epidemic spreading over other networks (e.g. random networks, small-world networks or scale free networks), the spreading speed on the nearest neighbor coupled networks is slowest. On the other hand, when the epidemic is spreading in a population, people adapt their behavior to avoid contacting the infected individuals.[8,9] In Ref. [8], when susceptible individuals receive awareness from their infected neighbor nodes, they will take vaccination measures. Considering the effect of individual vigilance, in Ref. [9] it was thought that the effective spreading rate is dynamically adjusted with the time evolution at the vigilance period. The results of Refs. [8] and [9] show that individual awareness or vigilance can slow down the epidemic spreading speed effectively. In fact, a feedback loop between the state of the node and topology of the network is formed. This feedback loop can bring a complicated mutual interaction between a time varying network topology and the node dynamics. Networks with such a feedback loop are called adaptive co-evolutionary networks.[10]

Recently, adaptive networks have aroused the interest of many researchers and some important conclusions have been obtained.[10–15] In an adaptive network, the evolution of the topology depends on the dynamics of nodes, and the topological features have a strong influence on the dynamics of nodes. Gross *et al.*[11] and Shaw *et al.*[12] studied respectively the susceptible-infectedsusceptible (SIS) model and the susceptible-infected-recovered-susceptible (SIRS) model on an adaptive network, where the susceptible nodes can avoid contact with the infected nodes by rewiring their network connections. This means that new connections are established only with other susceptible or recovered nodes. More realistically, individuals may be not fully aware of their own and other's infection status. Risau-Gusman and Zanette[16,17] considered the case where the links between susceptible and infected individuals are broken by either individual, and then reconnected to a randomly chosen node of the network. The relevant results of the above research show that the introduction of rewiring affects both the network structure and the epidemic dynamics. However, the obtained results depend on the homogeneous networks in which susceptible-infective links are broken and are later reconnected at random. In Refs. [18] and [19] the networks with different initial structures and that randomly reconnect were considered, the results showed that

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†Corresponding author. E-mail: songyr@njupt.edu.cn

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the initial structure of topology plays an essential role in epidemic propagation at criticality. After a long enough evolution time, the network with any initial structure moves toward a topology similar to a ER network. Unlike previous related models[11,12,16–18] in which the probability of disconnecting links is constant, Lagorio *et al.*[19] considered that the probability is proportional to the degree of infected nodes and the preferential rewiring isolates the superspreaders more efficiently. Yoo *et al.*[20] considered the edge rewiring dynamics that is based on individual fitness, preserved the degree of each node, and took into account the individual adaptive behavior.

In fact, preferential attachment and local-world connectivity exist in many physical complex networks, showing the abundant properties of a scale-free and small-world. In this paper, we focus on the preferential local-world reconnecting mechanism, and study the co-evolutionary dynamics between an epidemic and network through a susceptible-infected-susceptible mode based on Cellular Automata (CA). The rest of this paper is organized as follows. In Section 2, we define the adaptive SIS model with a preferential local-world reconnecting strategy using the CA. In Section 3, aimed at the proposed reconnecting strategy, the interplay between epidemic propagation and network evolution is studied through a numerical simulation epidemic evolution. The problem of how the rewiring probability and initial topology influence the epidemic threshold is discussed. We also discuss the bi-stability issue using our model. We summarize the results in Section 4.

2. Epidemic model for adaptive networks with a preferential reconnection

2.1. CA model

We establish a SIS epidemic model on an adaptive network with the preferential mechanism using CA. The CA is defined as the 4-uplet:

$$CA = (N, Q, V, f). \tag{1}$$

Here, N is the cellular space, in which each cell can be in one of a finite number of distinct states at each discrete time. This cellular space may be of any dimension, and is of infinite extent. Q is the finite state set whose elements are all the possible states of the cells. V is the so-called neighborhood. The state of cell i at time t is denoted by $q_i(t)$ and the state of its neighborhood by $q_{V_i}(t-1)$, where V_i refers to the neighborhood of cell i. For the neighborhood of any cell, it can be a combination of the neighbors of a cell, which is a spatial vector including v cell(s). Finally, f denotes the local transition rules, which specifies how the dynamics run for the CA. Each cell transforms from its current state to a new state (at the next time) based on its current state and the states of its neighbors, according to the following transition rules:

$$q_i(t) = f(q_i(t-1), q_{V_i}(t-1)). \tag{2}$$

Considering an adaptive network with N nodes, a one-dimensional CA can be used to model the co-evolution between the etwork dynamic and epidemic dynamics. A cell of the CA is a node of the network. Generally, the topology of the network is denoted by the adjacency matrix A of the network that describes the neighborhood of these nodes. An adaptive network is a kind of dynamically changing network, so the topology is described by a varying-time matrix $A(t)$ in our model and matrix element $a_{ij}(t)$ describes the connected relationship between node i and j at time t. If an existing link connects between nodes i and j then $a_{ij}(t) = 1$, otherwise, $a_{ij}(t) = 0$. It is easy to obtain the neighborhood of CA from $A(t)$, and the neighborhood of node i at time t is denoted by $V_i(t) = \{a_{ij}(t) | a_{ij}(t) = 1, j = 1, 2, \ldots, N\}$.

2.2. Epidemic spreading dynamics

Nodes follow a SIS pattern in which each node can be susceptible (state 0), or infected (state 1). Let $Q = \{0, 1\}$, and denote $q_i(t) \in Q$ the state variable of node i at time t, namely

$$q_i(t) = \begin{cases} 1, & \text{node } i \text{ is infected at time } t, \\ 0, & \text{node } i \text{ is susceptible at time } t. \end{cases} \tag{3}$$

Initially, all nodes are in a susceptible state, with one node chosen randomly as an infected state. In each time unit t, susceptible node i contacts infected node j, then the node i becomes infected with probability β. So, the rate for a susceptible node ($q_i(t) = 0$) to become an infected state ($q_i(t+1) = 1$) is $1 - (1-\beta)^{m_i(t)}$ at the next time, where $m_i(t)$ is the number of infected neighbors the node i has at time t and

$$m_i(t) = \sum_{j=1}^{N} a_{ij}(t) q_j(t). \tag{4}$$

Meanwhile, an infected node may be cured with probability δ and becomes susceptible.

The density of infected nodes $\rho(t)$ can be obtained by

$$\rho(t) = \frac{1}{N} \sum_{i=1}^{N} q_i(t). \tag{5}$$

The transition rule of an epidemic dynamic can be described by

$$Q(t+1) = F(A(t), Q(t)). \tag{6}$$

2.3. Network dynamics

While the epidemic spreads, the network is also rewired adaptively. We divide the rewiring process into two steps. The first step is to disconnect the edge (DE) and the second step is to reconnect the edge (RE).

In most existing studies,[11,12,17,19] in the strategy of DE it is assumed that all edges connecting susceptible and infected nodes are broken at rate ω and then in the strategy of RE it

is assumed that the corresponding susceptible node is then reconnected to a randomly chosen non-infected node. Self-connections and multiple connections between pairs of nodes are avoided. For simplicity, we adopt a similar strategy of DE to the ones in Refs. [11], [12], [17], and [19] and focus on the mechanism of RE. A link connecting a susceptible node x with an infected node y (SI link) is broken with probability ω at time t. Namely, $P\{a_{xy}(t)=0, a_{yx}(t)=0\}=\omega$. For the sake of avoiding infection, the susceptible node x is regarded as the source node and establishes a new link with a destination node z in the RE step.

The nodes of possible candidates are denoted by the set of C. If a node x is fully aware of the infection status of other nodes, then the set C only includes those susceptible nodes which are not neighbors of node x. Let $\chi_{xi}(t)=a_{xi}(t)+q_i(t)$, $i=1,\ldots,N$ and $i\neq x$, then $C=\{i|\chi_{xi}(t)=0\}$. In another case, the presence of asymptotically-infected individuals causes node x to make an ambiguous judge, so the set C includes all nodes which are not neighbors of node x, namely $C=\{i|a_{xi}(t)=0\}$. Let $|C|$ be the number of elements in C. Obviously, $|C|\geq 1$ in general.

Now, we come to consider more realistic situations where the source node has a preference to choose a node with special properties as a destination node. For example, when an epidemic spreads in a population, individuals tend to stay home and avoid going away, which means that more new short-range links replace those broken links. Namely, the source node preferentially selects a nearby node as the destination node. So, we propose a new reconnecting edge strategy which is a preferential reconnection depending on the spatial distance between the two nodes (PR-SD). The source node preferentially selects the nearest neighbor node to connect.

In the PR-SD, the spatial distance between two nodes is emphasized. Similar to the constructing algorithm of the small world network given by Wastts and Strogatz,[4] a node is randomly chosen from set C as the destination node with probability p for maintaining a part of long-range edges, and a node of set C that has the shortest distance with source node x is selected as the destination node with probability $(1-p)$. The rules are given by

$$\begin{cases} P\{z=i, i\in C|\xi<p\}=\dfrac{1}{|C|}, \\ P\{z=i, i=\min(d_{xj}, j\in C)|\xi\geq p\}=1, \end{cases} \tag{7}$$

where d_{ij} denotes the spatial distance of the node pair (i,j), $\xi\in[0,1]$, and a new link between two nodes (x,z) is connected, i.e., $a_{xz}(t)=1$, and $a_{zx}(t)=1$.

The probability p is a key parameter in the PR-SD strategy. The following cases are considered here:

i) $p=0$, which denotes the nearest neighboring reconnection. Once an SI link is broken, only the nearest node among C with x can become the new neighborhood. In all new established links, the long-range links are avoided effectively;

ii) $p=1$, which means that the spatial distance between nodes is ignored and a new neighbor node is randomly chosen from set C, which is similar to the RE strategy in previous studies;[11,12,17,19]

iii) $0<p<1$, which means that possible long-range links can be established by the probability p in the co-evolutionary process. The process is similar to establishing WS networks.

The transition rule of the network dynamic can be described by

$$A(t+1)=G(A(t),Q(t+1)). \tag{8}$$

3. Numerical simulations and analysis

Previous studies have shown that the initial structure of the network plays an essential role in epidemic spreading. In this section, we will perform some extensive numerical simulations using the proposed PR-SD strategy for some typical initial network topologies, which include the nearest neighboring coupled network (NC), Watta–Strogatz (WS) small-world network, Erdos–Renyi (ER) random networks,[21] and Barabasi–Albert (BA) networks.[3]

3.1. Interplay between epidemic propagation and network evolution

The time evolution curves of an epidemic on adaptive networks are shown in Fig. 1. It can be seen that the probability p in the PR-SD strategy plays an essential role in epidemic propagation, and the initial structure of the network has an important effect on the spreading speed of an epidemic.

Regardless of any p value, the epidemic on the network with BA being the initial topology spreads more quickly than that on the networks with other initial topologies. In addition, when $p=0$, the epidemic propagation is constrained very much for any initial topology.

Furthermore, provided that the time of spreading evolution is long enough, the densities of infection for different initial networks approach the same steady state, which is different from the result in static networks where the speed and scale of propagation are both influenced by the network structure.

The further explanation can be obtained from the analysis of statistical properties of the networks. Based on the PR-SD strategy, we analyze the following properties of the networks: clustering coefficient (CC), average path length (APL), network efficient (NE), and the second moment of mean degree ($\langle k^2\rangle/\langle k\rangle$). The results are shown in Table 1.

Table 1 exhibits the significant changes of network statistical properties at $t=0$ and $t=50$ for different values of p and different initial network topologies. The following phenomena can be observed from the table.

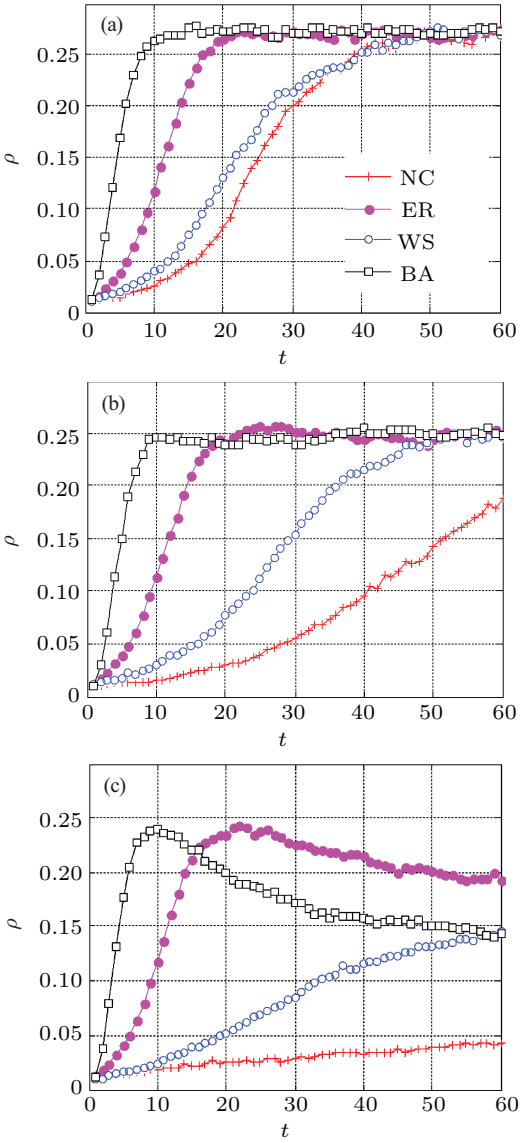


Fig. 1. (color online) Time evolutions of epidemic propagation on adaptive networks for PR-SD strategy in the cases where the values of p are 1 (a), 0.15 (b), and 0 (c), and other parameters are $\beta = 0.4$, $\delta = 0.8$, $\omega = 0.5$, $I(0) = 2$, and $N = 500$.

For the PR-SD strategy, the key parameter p has a significant effect on the evolution of network topology. If we take $p = 1$, the statistical properties of networks with different initial topologies, such as NC, ER, WS or BA, will tend to be those of an ER network. Namely, the original properties of networks are not preserved in the epidemic process and the degree distribution eventually approaches a Poisson-like distribution and the CC becomes small and APL short regardless of the initial topology. If we take $p = 0.15$, the statistical properties of networks with different initial topologies will tend to be those of a small-world network with dense clustering structures, a short average path length and Poisson distribution of node degrees. If we take $p = 0$, the APL and CC of networks increase quickly and the NE decreases, which results in the constraint of epidemic propagation as shown in Fig. 1(c), and the statistical properties of networks with different initial topologies will tend to be those of the nearest neighbor coupled (NC) networks with a long APL, high CC, and low NE.

For any case, the value of $\langle k^2 \rangle / \langle k \rangle$ is close to the value of $\langle k \rangle$ after evolution, which means that the final evolution networks tend to be homogeneous ones.

In the following, some necessary theoretical analysis is provided to further prove the results of Fig. 1 and Table 1.

Firstly, the change of degree distribution is explained. At time t, the status of node i with degree $k_i(t)$ is infected with the probability $\rho_i(t)$ and is susceptible with the probability $1 - \rho_i(t)$. For simplicity, we can assume that the infected density of the neighborhood of node i is $\rho(t)$. So, the number of SI links of node i can be described by

$$l_{\mathrm{SI}} = \rho_i(t) k_i(t) (1 - \rho(t)). \tag{9}$$

Table 1. Comparison among network statistical properties using PS-SD ($N = 500$).

Initial topology	Statistical property	$t = 0$	$t = 50$ ($p = 1$)	$t = 50$ ($p = 0.15$)	$t = 50$ ($p = 0$)
NC	CC	0.6	0.013	0.5115	0.58
	APL	42.08	3.69	6.277	39.26
	NE	0.06	0.295	0.185	0.063
	$\langle k^2 \rangle / \langle k \rangle$	6	7.368	6.52	6.12
ER	CC	0.014	0.018	0.329	0.49
	APL	3.67	3.68	4.57	28.195
	NE	0.293	0.295	0.242	0.078
	$\langle k^2 \rangle / \langle k \rangle$	6.86	7.344	6.82	6.65
WS	CC	0.43	0.011	0.34	0.52
	APL	5.27	3.66	4.60	11.34
	NE	0.213	0.30	0.24	0.125
	$\langle k^2 \rangle / \langle k \rangle$	6.11	7.455	6.78	6.65
BA	CC	0.078	0.012	0.336	0.5
	APL	3.08	3.65	4.61	18.66
	NE	0.345	0.295	0.24	0.095
	$\langle k^2 \rangle / \langle k \rangle$	16.37	7.237	6.87	7.16

Obviously, a hub node has higher $k_i(t)$ and $\rho_i(t)$ than other nodes. According to Eq. (9), the number of SI links for the hub is more than those of other nodes, meaning that more edges connecting the hub node are broken in the process of rewiring edges. With the co-evolutionary mechanism, these nodes with a high degree lose more edges, causing the degree distribution to tend to be homogeneous.

Secondly, the changes of APL for networks are considered. According to the reconnecting rules (7), the PR-SD strategy makes the rewiring process for a network structure similar to the establishing process for a WS network. In Ref. [22], the complete solution for APL has been drawn by

$$\mathrm{APL}(p) = \frac{N}{\langle k\rangle} f(N\langle k\rangle p), \tag{10}$$

where

$$f(x) = \frac{4}{\sqrt{x^2+4x}} \tanh^{-1} \frac{x}{\sqrt{x^2+4x}}. \tag{11}$$

Let $N = 500$, $p = 0.15$, and $\langle k\rangle = 6$, the theoretical value of APL from Eq. (10) is obtained to be 4.51, which coincides with the simulation result in Table 1.

3.2. Epidemic threshold

We also study the epidemic threshold under different initial networks based on the PR-SD strategy. Each datum point is obtained by averaging over 100 independent realizations. In our simulations, initial topologies are chosen as the 4 identical typical networks, $\omega = 0.5$, λ varies from 0 to 1 in steps of 0.0125, and 100 runs are performed for each λ. Figure 2 shows the numerical results for the density of infection in the steady state with respect to the spreading rate $\lambda(=\beta/\delta)$ for different values of probability p and initial topologies based on the PR-SD strategy. As shown in Fig. 2(a) for the case of $p = 0$, the initial topologies of networks influence the epidemic. Figures 2(b) and 2(c) display the 2 cases for $p = 0.15$ and $p = 1$, showing similar spreading properties but small differences in epidemic threshold between different initial topologies of networks.

Furthermore, two steady states, such as a disease state and an endemic state, can occur in our simulations. The region of bi-stability can be observed from Fig. 3. If the epidemic dies out in any time of the 100 runs for any value of λ, the disease-free state is considered to be stable. We define the rate of the disease-free state as a ratio of the number of disease-free states to 100 runs, which is shown in Fig. 3. The regions of bi-stability are similar for different values of probability p and almost all are located in $\lambda \in (0.2, 0.7)$. For different initial topologies of networks, there are a wider region of bi-stability and a lower rate of disease-free state in the BA network than those in NC, ER, and WS networks. For any case, the rate of disease-free state decreases exponentially as λ increases.

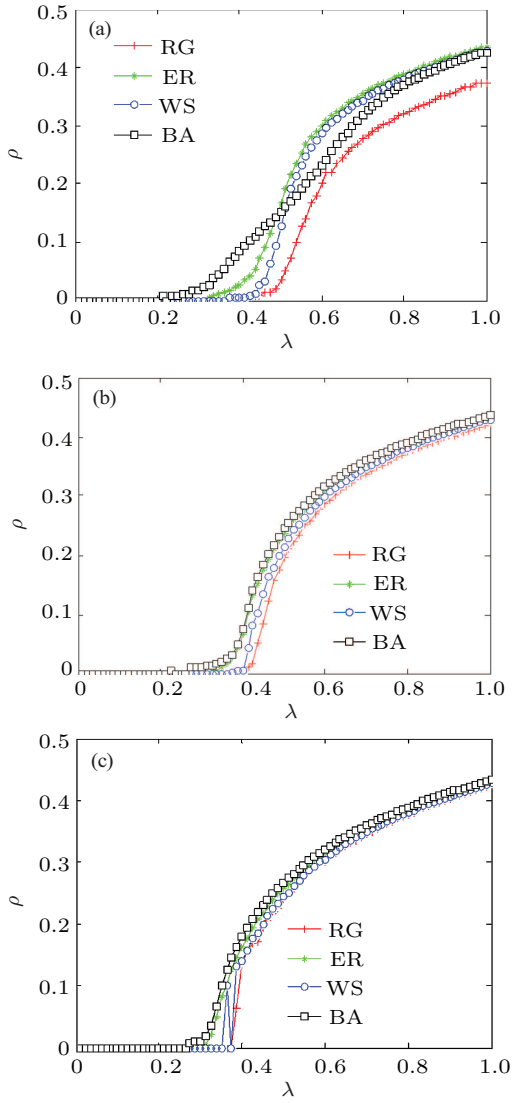


Fig. 2. (color online) Variations of infected density ρ with spreading rate λ in the cases where the values of probability p are 0 (a), 0.15 (b), and 1 (c), and the other relevant parameters are $N = 500$, $\langle k\rangle = 6$, $\omega = 0.5$, and $\delta = 0.8$.

The variations of density of infection ρ with probability of rewriting ω and the results of simulation are shown in Fig. 4.

The density ρ value decreases with the increase of ω, and the epidemic dies out as ω increases to a critical value denoted by ω_c. Namely, when $\omega \geq \omega_c$, only a small number of individuals get infected and the epidemic quickly dies out, which is a disease-free state.

Figure 4(a) shows the variations of ρ with ω for different values of probability p on the networks with ER or BA being initial topologies. Figure 4(b) shows the variations of ρ with ω for different initial topologies of networks and $p = 0.15$. The two figures show that the different values of p and different initial topologies of networks have small effects on ω_c. The larger the value of ω, the faster the network structure changes and the less effect on the propagation of the epidemic the initial topology of the network has. The smaller the value of p,

the smaller the density is, which indicates that avoiding long-range links can effectively restrain the propagation of an epidemic. Figure 4(c) shows the variations of ρ with ω for different values of β, indicating that β has a strong effect on the critical threshold ω_c, and the increase of the value of ω_c with β.

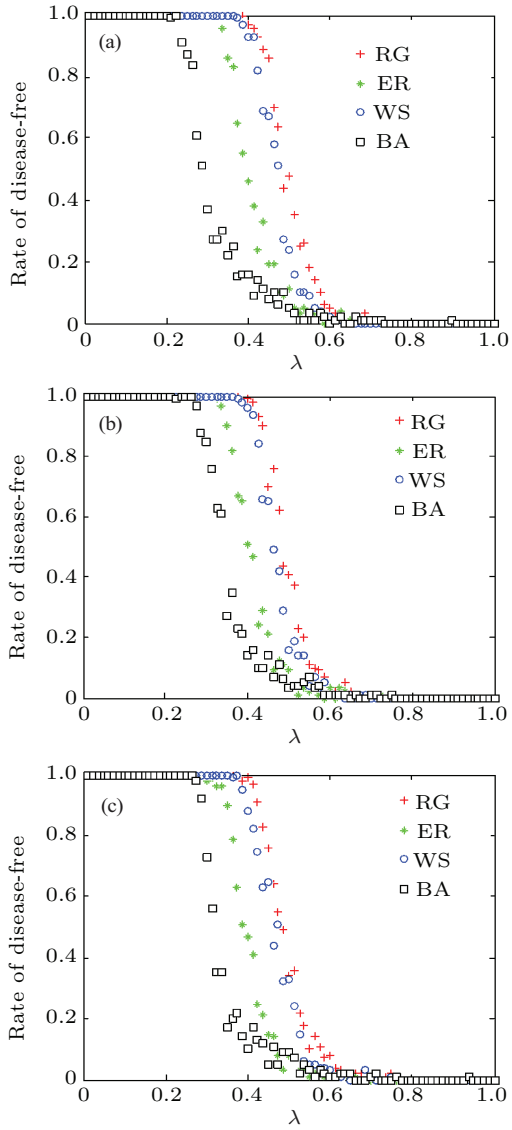
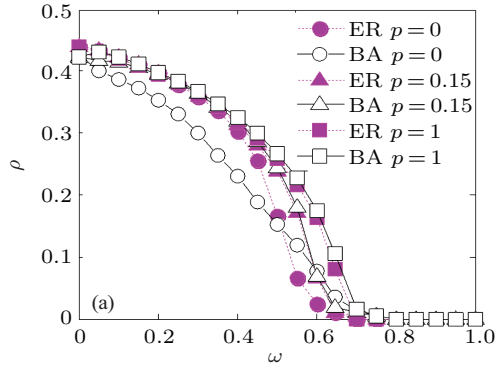


Fig. 3. (color online) Curves of rate of disease-free versus spreading rate λ in the cases where the values of probability p are 0 (a), 0.15 (b), and 1 (c), and other relevant parameters are $N = 500$, $\langle k \rangle = 6$, $\omega = 0.5$, and $\delta = 0.8$.



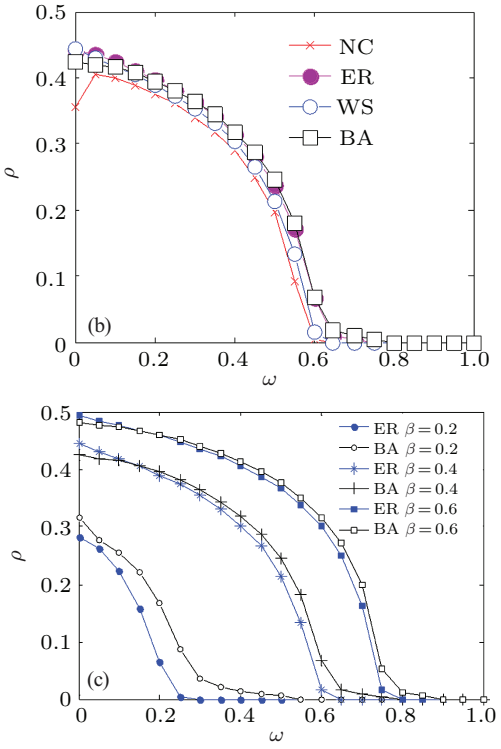


Fig. 4. (color online) Variations of ρ with the probability of rewriting ω for PR-SD strategy in the cases of (a) different values of p and $\beta = 0.4$, (b) different initial topologies, $p = 0.15$, and $\beta = 0.4$, and (c) different values of β, and $p = 0.15$.

4. Conclusions

We introduced and studied preferential reconnection strategies for the epidemic on adaptive networks. The states of nodes are changed according to SIS dynamics, while the network evolves according to an avoiding infection mechanism, in which a susceptible individual breaks a link with an infected individual with probability ω and establishes a new link with other susceptible peer according to PR-SD strategy where the new link is established at random with probability p and in the shortest distance with the probability $1 - p$.

We demonstrate our strategy by numerical simulations. The probability p is a key parameter. The smaller the value of p, the more difficult the epidemic spread is. This is because the PR-SD strategy breaks long-range links and establishes as many as possible short-range links, which causes the network efficiency to decrease quickly, and the propagation of epidemic is restrained effectively. The rewiring link leads to the phenomenon of bi-stability where a disease state and a disease-free state can co-exist. Furthermore, the rate of the disease-free state decreases exponentially as λ increases. The results also show that the size of the epidemic can be largely reduced by increasing the probability of rewiring and that the propagation can eventually be stopped by using a high enough value of ω. Furthermore, the higher the value of ω, the faster the network structure changes and the less effect on the propagation of epidemic the initial topology of the network has. In

other word, for differential initial topologies of networks, provided that these networks adopt the PR-SD strategy with same parameters, the networks approach similar statistic properties after a long time evolution.

In a word, unlike the reconnecting randomly strategy, the PR-SD strategy reduces the long-range links and the efficiency of network, which efficiently constrains the propagation of the epidemic.

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