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Different behaviors of epidemic spreading in scale-free networks with identical degree sequence

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Abstract

Recently, the study of dynamical behaviors of the susceptible-infected (SI) disease model in complex networks, especially in Barabási–Albert (BA) scale-free networks, has attracted much attention. Although some interesting phenomena have been observed, the formative reasons for those particular dynamical behaviors are still not well understood, despite the speculation that topological properties (for example the degree distribution) have a strong impact on epidemic spreading. In this paper, we study the evolution behaviors of epidemic spreading on a class of scale-free networks sharing identical degree sequence, and observe significantly different evolution behaviors in the whole family of networks. We show that the power-law degree distribution does not suffice to characterize the dynamical behaviors of disease diffusion on scale-free networks.

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(Some figures in this article are in colour only in the electronic version)

1. Introduction

In recent years, both the network structure and dynamics on complex networks have been studied intensively [1–4]. Concerning the former, scientists have presented a series of statistical complex topological characteristics such as the small-world phenomenon [5] and scale-free (SF) property [6] by investigating various kinds of real networks including the Internet [7], the World Wide Web [8], the scientific web [9], the protein interaction networks [10], and so on. Concerning the latter, most researchers have focused on the problem: how the properties of networks influence the dynamical processes taking place upon the networks [11, 12].

Amongst various typical dynamical processes built on complex networks, epidemic spreading has attracted more and more significant attention [13–28], and studying epidemic spreading is important for both the theoretical research and the real disease control.

A fundamental model for disease spreading is the susceptible-infected (SI) model. Among most previous studies of epidemic spreading for the SI model, the underlying networks are treated as scale-free, a remarkable property observed in many real networks [29]. These studies revealed several dynamical characteristics of epidemic spreading, such as the initial exponential growth of the prevalence measured by the density of infected nodes, the hierarchical propagation of the infection measured by the average degree of newly infected nodes and so forth [13, 14]. It is acknowledged that the behaviors of epidemic spreading in complex networks are influenced by the topological properties of networks; that is to say, the spreading forms in networks with different topological properties behave differently. For example, it has been shown that a degree distribution has a drastic effect on the propagation dynamics of epidemics [13, 14]. Then, a question naturally arises, what if the degree distribution itself can characterize the epidemics on networks? In other words, for networks sharing the same (power-law) degree distribution, will the dynamical processes of epidemic spreading exhibit the same behavior? As far as we know, it is still unknown whether the degree distribution, especially the power-law scaling, is sufficient to characterize the evolution behaviors of the dynamics on the scale-free networks.

In this paper, we adopt a growth model which can generate a class of scale-free networks with the same degree sequence controlled by a probability parameter q, and we apply the SI disease model to the networks for studying the influence of q on the dynamical process of epidemic spreading. We show that the parameter q has a strong impact on the evolution behaviors of the infection pervasion, namely different behaviors of epidemic spreading occur as q increases from 0 to 1. Since in the full range of $0 \leqslant q \leqslant 1$, the network family has the identical degree sequence and thus the same degree distribution, we can conclude that the power-law degree distribution is not sufficient to characterize disease spreading running on scale-free networks.

2. Network model and its properties

In this paper, we adopt the growth undirected model proposed in [30], which can generate a class of scale-free networks with identical degree sequence controlled by a probability parameter q. The model is constructed in an iterative way. Let G_t denote the networks after t iterations; then the rules of construction can be depicted as follows. Starting with two nodes connected by an edge, which is the initial networks G_0. After each iteration step $t \geqslant 1$, G_t is obtained from G_{t-1} by performing the operations: each existing edge in G_{t-1} is replaced either by a loop of edges on the top right corner of figure 1 with probability q, or by the loop on the bottom right corner of figure 1 with probability $1-q$. The growth process is repeated t times, with the graphs obtained in the limit $t \to \infty$. In figures 2 and 3, we show the growth process for two limiting cases of $q = 1$ and $q = 0$, respectively.

Let V_j denote the number of nodes newly added into the networks at step j, and let E_t denote the total number of edges present at step t. In view of the construction rules, it is not difficult to find the relation $E_t = 4E_{t-1} = 4^t$ $(t \geqslant 0)$. On the other hand, each existing edge at the present step $t-1$ will generate two new nodes at the next step t; therefore, we have $V_t = 2E_{t-1} = 2 \times 4_{t-1}$ $(t \geqslant 1)$. Then the number of total nodes N_t present at step t is

$$N_t = \sum_{j=0}^{t} V_j = \frac{2}{3}(2 + 4^t). \tag{1}$$

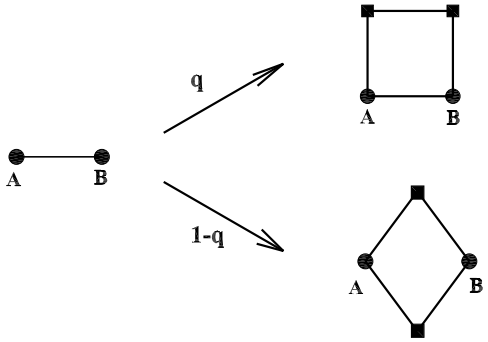

Figure 1. Iterative construction methods of the networks. Each link is replaced by either of the loops on the right-hand side of arrows with a certain probability, where black squares represent new vertices.

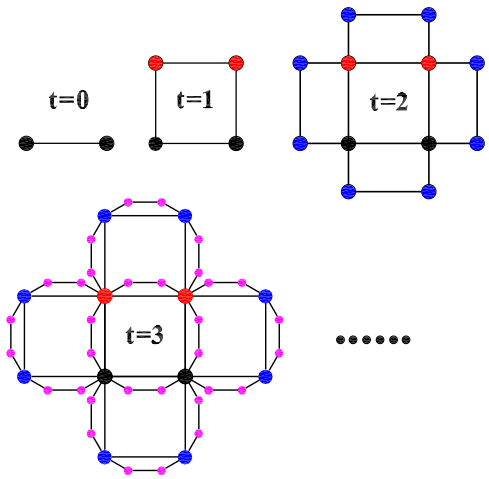

Figure 2. Illustration of the first four evolution steps of the network growth process for the particular case $q = 1$.

Obviously, one can obtain that the average degree after step t is

$$\langle k \rangle = \frac{2E_t}{N_t} = \frac{3 \times 4^t}{2 + 4^t}, \tag{2}$$

which approaches 3 in the limit of large t, independent of the parameter q.

By construction, the degree of node i at step t, represented as $k_i(t)$, behaves as $k_i(t) = 2k_i(t-1) = 2^{t+1-t_i}$, where $t_i (t_i \geqslant 1)$ denotes the time step when node i was added into the networks. For the nodes with $t_i = 0$, namely the two nodes in G_0, we have $k_i(t) = 2^t$. One can find that the degree of node i increases by a factor 2 at each time step. Thus, the degree spectrum of the networks is discrete. In G_t, all possible degrees of nodes are 2, $2^2, 2^3, \ldots, 2^{t-1}, 2^t$; and the number of nodes with degree $k = 2^{t+1-m}$ is $n_k = V_m = 2 \times 4^{m-1}$, which is also the number of nodes added into the networks at step m. Hence, all the networks G_t have the same degree sequence (thus the same degree distribution) in the full range of q.

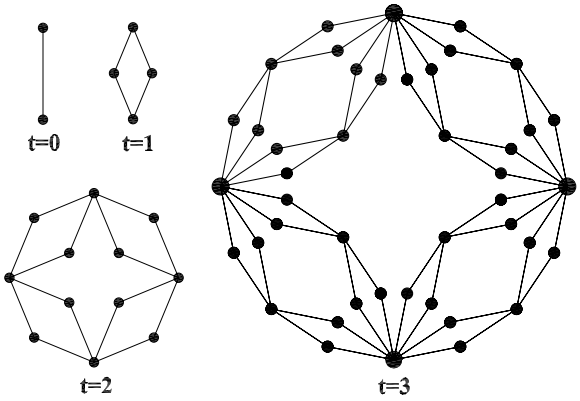

Figure 3. Sketch of the iteration process of the network for the particular case $q = 0$.

Since the degree spectrum of the networks is not continuous, it follows that the cumulative degree distribution [11] is given by

$$P_{\text{cum}}(k) = \frac{\Sigma_{k' \geqslant k} n_{k'}}{N_t} = \frac{\sum_{i=1}^{m} 2 \times 4^{i-1} + 2}{\frac{2}{3}(2 + 4^t)} = \frac{3 - 4^{-m}}{k^2 + 2 \times 4^{-m}}, \tag{3}$$

which leads to $P_{\text{cum}}(k) \sim k^{-2}$ for large m. Thus, the degree distribution $P(k)$ of the networks follows the same power-law form $P(k) \sim k^{-3}$, which is independent of q. Note that the same degree exponent has been obtained in the famous Barabási–Albert (BA) scale-free networks [6].

Next, we discuss another property—clustering coefficient—of the networks. By definition, the clustering coefficient [5] of a node i with degree k_i is given by $C_i = 2e_i/[k_i(k_i - 1)]$, where e_i is the number of existing triangles attached to node i, and $k_i(k_i - 1)/2$ is the total number of possible triangles including i. The clustering coefficient of the whole network is the average over all individual $C_i's$. By construction, it is easy to find that there are no triangles in G_t, so the clustering of every node and its average value in G_t are both zero, which does not depend on the parameter q.

A third important quantity of a network is the average path length (APL). Let l_{ij} be the shortest path length from node i to j; then the average path length l_t of G_t is defined by the mean of l_{ij} over all pairs of nodes in the network:

$$l_t = \frac{L_t}{N_t(N_t - 1)/2}, \tag{4}$$

where

$$L_t = \sum_{i \in G_t, j \in G_t, i \neq j} l_{ij} \tag{5}$$

denotes the sum of the shortest path length between two nodes over all pairs.

For the special case of $q = 1$, the networks are reduced to the (1,3)-flower introduced in [31]. For this limiting case, the APL shows a logarithmic scaling with network order [30], which implies that the network exhibits a small-world behavior. For $q = 0$, the network is exactly the hierarchical lattices that was proposed by Berker and Ostlund [32], and its APL increases as a square power of the network order [33, 34], indicating that the network exhibits a 'large-world' behavior of typical node–node distances. For the two limiting cases of $q = 1$ and $q = 0$, both networks belong to a deterministic growing type of networks, which have

received much attention from the scientific communities and have proved to be a useful tool [35–39]. For $0 < q < 1$, the APLs show that there is a crossover between small world and large world [30]. This behavior is similar to that in the Watts–Strogatz (WS) model [5].

As discussed above, the networks exhibit many interesting properties: they have the same degree sequence independent of the parameter q; they are scale-free and non-clustered; and they have a crossover between large world and small world. Hence, it is worthwhile to investigate the dynamical processes taking place on the networks to find the different behaviors compared with the other networks, such as the BA network. In what follows, we study epidemic spreading, which is one of the most intensively studied issues in physics.

3. The SI disease model on the networks

3.1. Introduction to the SI disease model

Here we study the SI disease model [13, 14] on the studied networks, in order to find the dynamical behaviors that show how diseases diffuse on the networks. To this end, we first introduce the classic SI model. In the SI model, all individuals (nodes) can be divided into two classes depending on their states: susceptible (healthy) and infected. Hereafter the notation t will be used to express the time of disease spreading, different from the meaning (iterations of networks) in section 2. Then, the epidemic evolves by the following rules: initially ($t = 0$), there are a number of I_0-infected nodes and any infected node can pass the disease to its susceptible neighbors. Then, at each time step, a susceptible individual acquires the infection at the transmission rate λ in one contact with any neighboring infected individual. Therefore, the total probability that a susceptible node i with degree k_i becomes infected at time step t is given by $1 - (1 - \lambda)^{\theta(i,t)}$, where $\theta(i, t)$ denotes the number of infected neighboring nodes of the susceptible node i at time step t.

To take into account the heterogeneity induced by the presence of nodes with different degrees, we define the quantity $i_k(t) = I_k(t)/M_k$ to be the density of infected nodes with degree k, where $I_k(t)$ and M_k represent the number of infected nodes and total nodes with degree k, respectively. Similarly, we have $s_k(t) = S_k(t)/M_k$. Obviously, $i_k(t) + s_k(t) = 1$. The global quantities such as the (average) epidemic prevalence are therefore expressed by an average over the various degree classes, i.e. $i(t) = \sum_k P(k) i_k(t) = I(t)/N$, where $I(t)$ denotes the number of infected nodes in the whole network at time step t and N stands for the network size (the total number of nodes). Thus, one can see that the prevalence $i(t)$ means the density of infect nodes in the whole network at time step t.

3.2. Simulation results and analysis

We continue to report the simulation results of the SI disease model on the networks under consideration, and give our analysis. In our simulations, for the initial conditions of disease diffusion, we randomly select one node and assume that it is infected, then the disease will spread throughout the network till all the nodes are infected (i.e. $\lim_{t\to\infty} i(t) = 1$). Simulations were performed for networks G_7 with order $N = N_7 = 10\,924$, averaging over 500 independent realizations of the dynamics. We will show that different behaviors can be observed for epidemic spreading on the family of scale-free networks with the same degree sequence.

In figures 4(a) and (b), we plot the temporal behaviors of the prevalence $i(t)$ for two limiting cases of $q = 0$ and $q = 1$, respectively. It is observed that the prevalence $i(t)$ in the case of $q = 0$ grows much more slowly than that in the case of $q = 1$, which means that the

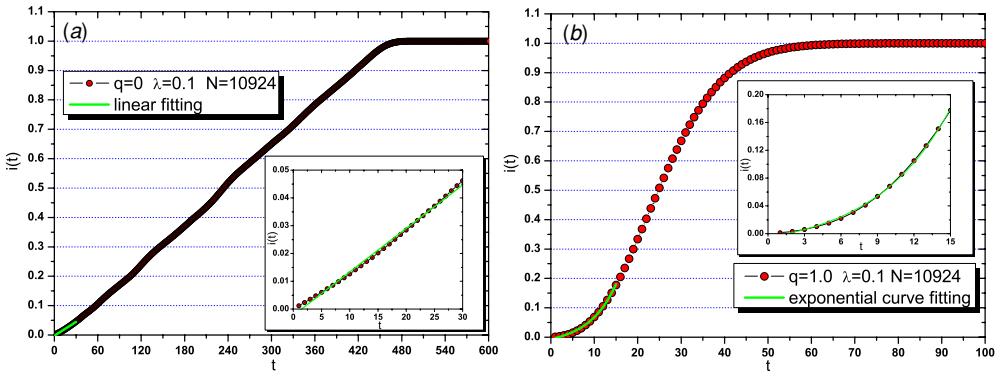

Figure 4. The time evolution of the prevalence $i(t)$ with $\lambda = 0.1$ and $N = 10\,924$. (a) and (b) correspond respectively, to the cases $q = 0$ and $q = 0$. The insets of (a) and (b) show that in the early times, $i(t)$ grows approximatively with time linearly and exponentially, respectively.

network for $q = 0$ cases can resist the epidemic much better than the network corresponding to the case of $q = 1$. In the case of $q = 0$, the prevalence $i(t)$ seems to have a linear growth at the initial stage of the disease diffusion (see the inset of figure 4(a)). While for the case of $q = 1$, it seems to be an exponential growth of $i(t)$ in the early times (see the inset of figure 4(b)) that is also shown on the BA networks [13], and by the exponential curve fitting, the initial prevalence grows as $i(t) \simeq a + b \times e^{t/c}$, where three fitting parameters are $a = -0.0239 \pm 0.0031$, $b = 0.0185 \pm 0.0019$ and $c = 6.2459 \pm 0.2383$. The difference of initial growth might be attributed to the structures of the two special networks: in the network generated by $q = 1$, the nodes' neighbors could be with various degrees, and hub nodes are connected to each other. This structure is similar to that of the BA networks. Whereas for the network in the case of $q = 0$, the neighbors of nodes with degrees larger than 2 are restricted to the nodes with the lowest degree 2. That is to say all the nodes with degrees greater than 2 are surrounded by the nodes with degree 2. In fact, $q = 0$ and $q = 1$ are two extreme cases. For other cases of $0 < q < 1$, the evolution behaviors of the disease dynamics are also obviously different. In figure 5, we report the time behaviors of $i(t)$ for the general q (i.e. $0 < q < 1$), which displays that $i(t)$ is the increasing function of q: the larger the parameter q is, the more quickly the prevalence $i(t)$ grows.

To understand the disease diffusion in more depth, we study the spreading velocity of the infection, which can be written as [13]

$$\delta(t) = \frac{\mathrm{d}i(t)}{\mathrm{d}t} \approx i(t) - i(t-1). \tag{6}$$

As shown in figure 6, the different evolution forms of the spreading velocity exist in the special cases of $q = 0$ and $q = 1$. For the case of $q = 1$ (see figure 6(b)), the spreading velocity goes up to a peak at a certain time step, denoted as t', where the prevalence $i(t')$ has the fastest growth rate, then the spreading velocity descends till zero. Before the time step t', the prevalence undergoes an accelerated growth, while after the time step t', the prevalence undergoes a decelerated growth, as figure 4(b) displays. This time step t' is also referred as the 'peak' time, which means an existence of transition from the small incidence of the epidemic to a macroscopic outbreak. However, for $q = 0$, the case is completely different. In figure 6(a), we report the spreading velocity for the case of $q = 0$. It is obvious that the curve shows several different peaks, which means the infection pervades the networks irregularly. The

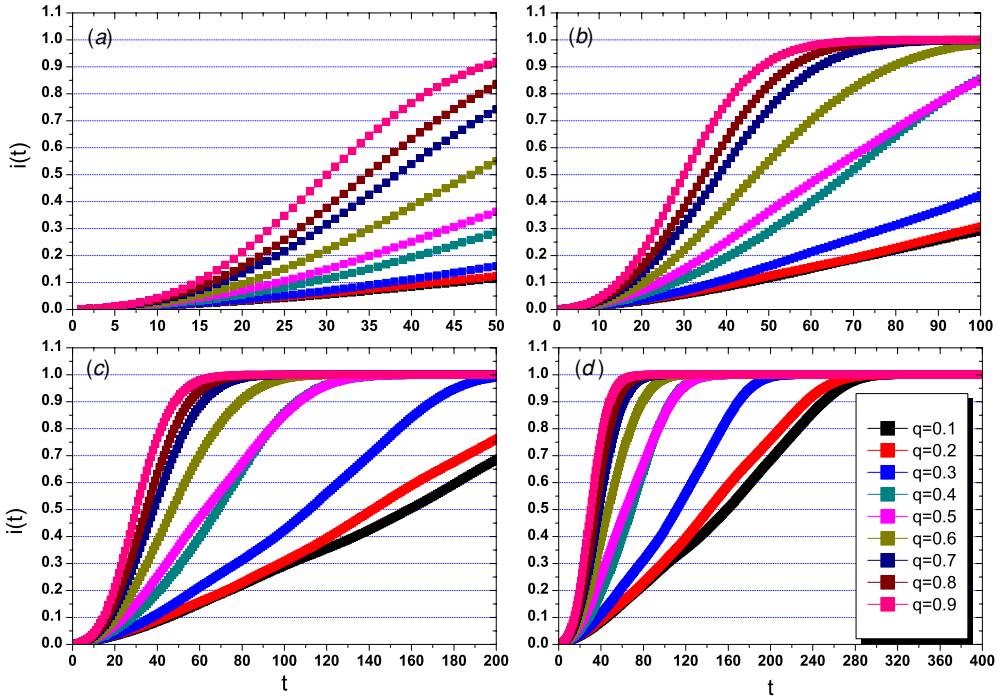

Figure 5. The time evolution of the prevalence $i(t)$ on the networks with general q (i.e. $q = 0.1, 0.2, \ldots, 0.9$). The other related parameters are $\lambda = 0.1$ and $N = 10924$. (*a*)–(*d*) have different time scales for the purpose of better visibility.

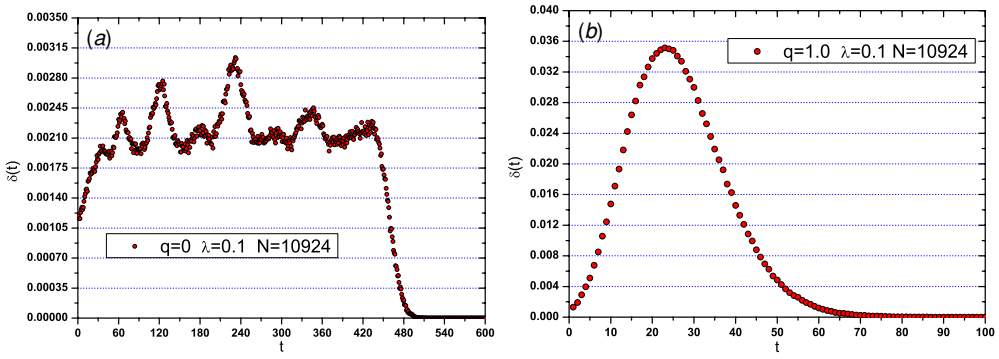

Figure 6. Spreading velocity for two networks of cases $q = 0$ and $q = 1$.

observed multi-peaks can account for the reasons why the growth curve of $i(t)$, shown in figure 4(*a*), does not behave as smoothly as that in figure 4(*b*). Moreover, we can see that the local peaks in figure 6(*a*) have different peak values, which implies that there are several local fastest growth rates for the $q = 0$ case that is in contrast to the behavior of the $q = 1$ case, where only one global fastest growth rate exists.

As referred above, what figure 6(*a*) shows is an average result. If we consider any one individual run case, the spreading velocity curve might be different from the average one in some aspects such as the time when the local maxima appear, the number of the local

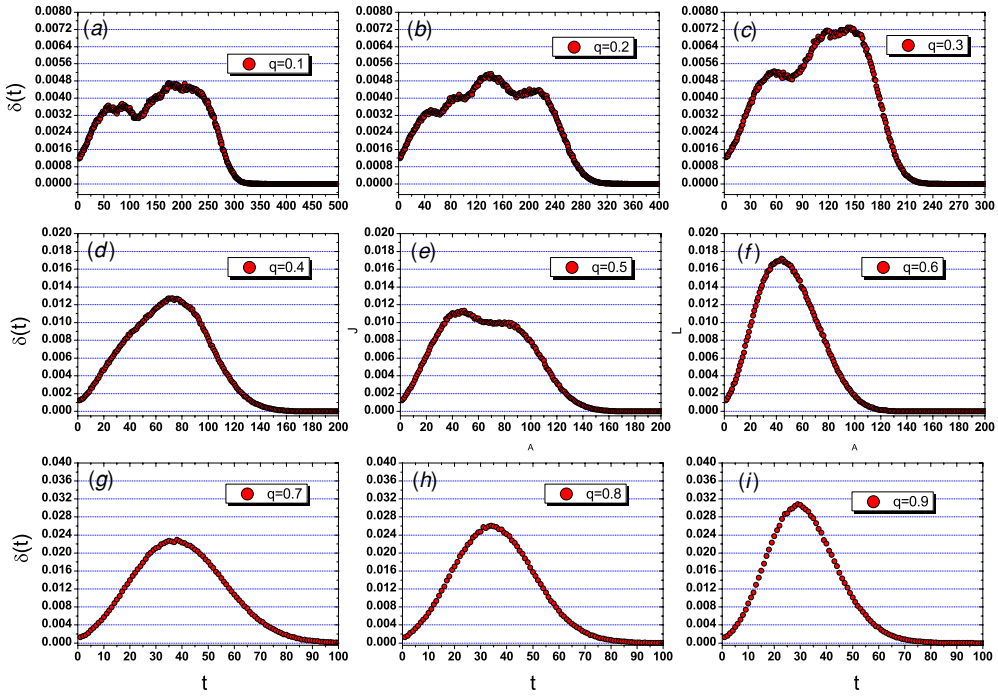

Figure 7. Spreading velocity versus time with $\lambda = 0.1$ and $N = 10\,924$. (*a*)–(*i*): on the networks with $q = 0.1$ to $q = 0.9$, respectively.

maxima, and so on. The reason, we believe, is mainly attributed by the initial seeds of the dynamics, i.e. the initial infected nodes. For two independent realizations of the dynamics, if they have the same initial seeds, their diffusion processes approximate to the same. In contrast, when the initial seeds are different, the epidemic diffusion might be quite different, and some measurements may also differ. That is to say the results from one single run are unstable. Therefore, we adopt the average method to weaken the effect of the initial seeds and other random factors. Those results averaged over many independent runs can help us to make a general analysis on the epidemic dynamics. Moreover, it should be mentioned that a similar average method has been commonly used in the previous studies of epidemic spreading.

To further study the spreading velocity for general q, in figure 7, we depict the behavior of spreading velocity for the networks corresponding to $0 < q < 1$. From figure 7, one can observe that the evolution behavior of spreading velocity transforms from a pattern with multi-peaks to a single peak as q increases from 0 to 1. After further analysis on figure 7, we find that the spreading velocity varies with q as follows: the larger the parameter q is, the more quickly the infection pervades the networks.

Finally, we investigate another quantity $\langle k_{\rm inf}(t)\rangle$ characterizing the epidemic spreading, which is defined as the average degree of the newly infected individuals at time step t [13, 14]. By definition, this quantity reads

$$\langle k_{\rm inf}(t)\rangle = \frac{\sum_k kI_k(t) - \sum_k kI_k(t-1)}{I(t) - I(t-1)}, \tag{7}$$

where $I_k(t)$ stands for the number of the infected nodes with degree k at time step t. Anomalous to other quantities addressed above, we firstly investigate the two limiting cases of $q = 0$ and

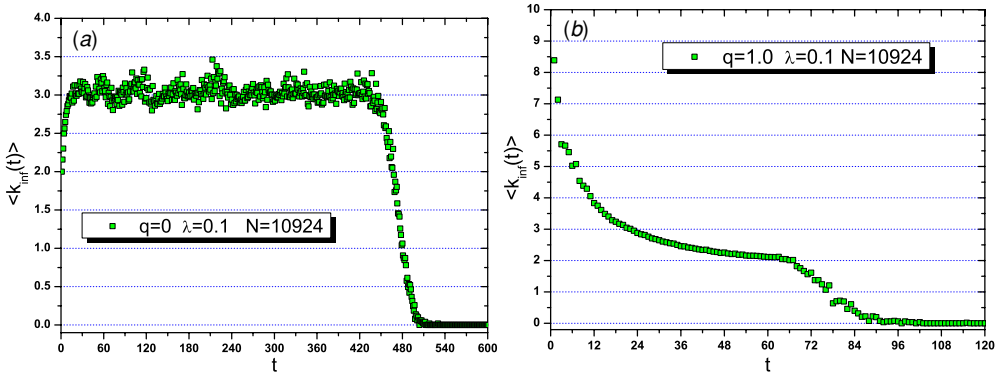

Figure 8. Temporal behavior of the average degree of the newly infected nodes for two particular cases of $q = 0$ and $q = 1$.

$q = 1$. In figure 8(a), we plot the evolution behavior of $\langle k_{\rm inf}(t)\rangle$ for the case of $q = 0$. The curve shows a plateau which is almost independent of time. After this plateau stage, $\langle k_{\rm inf}(t)\rangle$ decreases sharply at the end of epidemic spreading. As a whole, it is observed that a similar cascade effect exists in the infection time pattern, which is exactly shown on the BA network in [14]. But two significant distinct aspects should be denoted. Firstly, in figure 8(a), the asymptotic value of $\langle k_{\rm inf}(t)\rangle$ at the plateau stage is stabilized at around three, which is the average degree $\langle k\rangle$ of the present networks; while in [14], the corresponding asymptotic value is $\langle k^2\rangle/\langle k\rangle$ for the BA networks and this value is much larger than the average degree $\langle k\rangle$ of the BA networks. Secondly, in figure 8(a), the curve has a much longer duration at the plateau stage; while for the BA networks, this plateau stage exists when $t < \tau$, where τ is the time scale of the infection growth defined in [14] and τ is a quite small value when the networks order $N \to \infty$. These two characteristics suggest that the infection should pervade the network of the $q = 0$ case randomly rather than selectively in most time of the epidemic spreading, i.e. an apparent hierarchical propagation of the infection pervasion is absent in the case of $q = 0$. This may be understood based on the following heuristic arguments. According to the construction of the network, those nodes with degree larger than 2 are protected by the nodes with the lowest degree 2. Thus, before infecting nodes with high degree, the disease should firstly attack the lowest degree nodes successfully. Since 'large' nodes are not linked to each other, even if some hub nodes are infected, the infection cannot pervade other hub nodes and the whole network easily. In fact, at most times of the epidemic spreading, the disease infects the nodes with a degree of 2.

However, as shown in figure 8(b), for the case of $q = 1$, the curve for the average degree of the newly infected individuals at every time step shows an apparent hierarchical behavior: the infection pervades the entire network in a progressive cascade from high-degree nodes to low-degree nodes. During the epidemic spreading, the hubs are firstly infected in a very short time, then the spreading always goes toward the smaller degree nodes, namely the nodes with lowest degree are infected lastly. It should be noted that, although the epidemic propagation shows a precise hierarchical dynamics for the case of $q = 1$, it does not exhibit any short-term plateau stage as shown in the BA networks [14]. The reasons for this difference deserve a further indepth study, but we believe that the presence of the plateau stage in the BA networks is probably induced by the rescaled time t/τ used in [14].

For other cases of $0 < q < 1$, the average degree of the newly infected individuals at every time step is also dependent on the parameter q. In figure 9, we describe the simulation

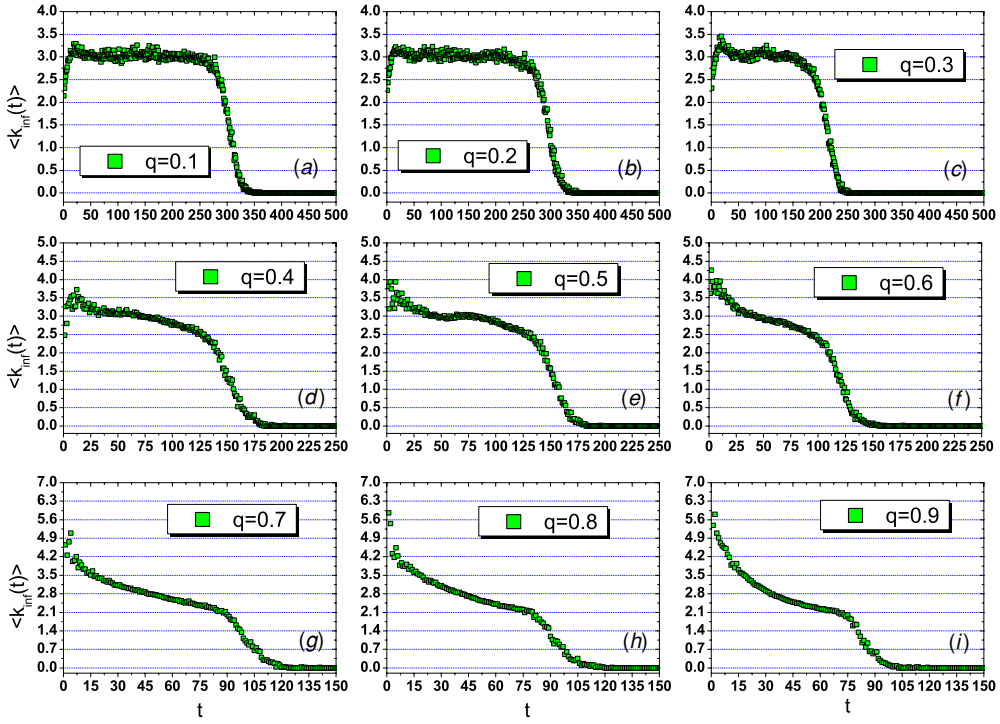

Figure 9. Temporal behavior of the average degree of the newly infected nodes for networks with order $N = 10\,924$ and different q that belongs to the interval $(0, 1)$. In all simulations, we set $\lambda = 0.1$.

results for networks with various q. It is shown that different infection modes occur as the parameter q increases from 0 to 1. Moreover, the larger the parameter q is, the more obvious the hierarchical propagation is.

4. Conclusion and discussion

To summarize, in this paper, we have studied the epidemic spreading in a class of scale-free networks with identical degree sequence, which is dominated by the tunable parameter q. We found that propagation dynamics is closely related to the parameter q: the larger the parameter q is, the more quickly the prevalence $i(t)$ grows. This means that in the network family, the networks with smaller q can relatively efficiently resist the epidemics. Particularly, we found that when the parameter q increases from 0 to 1, the evolution behavior of spreading velocity shows a crossover from a form with multiple peaks to a form with a single peak. In addition, we have also studied the infection time pattern for different q, and found the presence of the hierarchical propagation of the epidemics in networks with larger q, which is also previously observed in the BA networks.

Thus, the scale-free network family exhibits quite different evolution behaviors of disease diffusion, in spite of the fact that the whole family of networks have the same degree sequence and thus the same degree distribution. This signals that the power-law degree distribution alone does not suffice to characterize the evolution behaviors of epidemic spreading occurring on scale-free networks. Therefore, one should be careful when making a general statement about the propagation dynamics of epidemics on scale-free networks. We believe that our

work might shed some light into the understanding of epidemic spreading on networks. It is also expected that our results will be helpful in designing scale-free networks resistant to disease spread.

Acknowledgments

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