

Epidemic spreading in weighted scale-free networks with community structure

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Epidemic spreading in weighted scale-free networks with community structure

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Abstract. Many empirical studies reveal that the weights and community structure are ubiquitous in various natural and artificial networks. In this paper, based on the SI disease model, we investigate the epidemic spreading in weighted scale-free networks with community structure. Two exponents, α and β , are introduced to weight the internal edges and external edges, respectively; and a tunable probability parameter q is also introduced to adjust the strength of community structure. We find the external weighting exponent β plays a much more important role in slackening the epidemic spreading and reducing the danger brought by the epidemic than the internal weighting exponent α . Moreover, a novel result we find is that the strong community structure is no longer helpful for slackening the danger brought by the epidemic in the weighted cases. In addition, we show the hierarchical dynamics of the epidemic spreading in the weighted scale-free networks with communities which is also displayed in the famous BA scale-free networks.

Keywords: network dynamics, random graphs, networks, diffusion

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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 www [8], the scientific web [9], the protein 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]. As one of the 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 real disease control.

In most previous studies of epidemic spreading, the underlying networks are only treated as scale-free networks of which scaling behavior is showed by many real networks [29]. But some other characteristics in real networks have not been considered or have been considered less sufficiently. For example, many real-world networks show community structure, especially in social networks [30]–[34]. That is to say, there are groups of nodes with a high density of edges within them and a lower density of edges between groups. Members in the same community or group can be regarded as classmates, friends, confreres, countrymen, etc. Recently, there has also been some research to investigate the influence of community structure upon epidemic spreading in scale-free networks. In [27], the authors investigated the global and local synchronization induced by the SIRS epidemic dynamics. They found a small strength (denoted by Q) of community structure induces better global synchronization and a phase transition occurs as Q changes from 1 to 0, whereas the effect of Q behaves differently for the local synchronization. In [28], the authors found the networks with strong community structure (namely, larger Q) are helpful for epidemic prevention.

On the other hand, apart from the topological properties it is significant to realize that many real-world networks are weighted [36]–[38]. Indeed, the weight (or the strength) is one of the most important indications in lots of real networks, for example, in social networks it can represent the intimacy between individuals; in the internet the weight can

imply the knowledge of its traffic flow or the bandwidths of routers [4]; in the world-wide airport networks it can evaluate the importance of an airport [2, 36], and so on [37, 38]. In particular, for epidemic spreading, the weight can indicate the extent of the frequency of the contacting of two nodes in scale-free networks: the larger the weight is, the more intensively the two nodes communicate, while, at the same time, the more possible it is a susceptible individual will be infected through the edge where the transmission rate is larger.

However, there are few studies to combine weights and community well in scale-free networks for epidemic spreading. Indeed, the presence of community structure will make the weighting patterns diverse. For example, we know that, in the networks with communities, there are two types among all edges, which are internal edges and external edges. In general, the individuals (nodes) which have internal edges show they may be members of the same organizations (communities), but members of different organizations for the individuals with external edges. Therefore, the interaction within the communities will be different in some respects such as strength or frequency. That means, for a weighted network with communities, the weighting patterns on the edges should be different according to their types.

In this paper, we study the epidemic spreading in the weighted scale-free networks with community structure, and the disease model we use is the SI model in which the individuals are classified into two types, i.e., susceptible and infected. Firstly, we adopt a model which can generate a weighted scale-free network with community structure. In our model the modularity Q is controlled by a probability parameter q , and two weighting exponents α and β are introduced to affect the weighting pattern of the internal edges and external edges, respectively. Based on the network, a more realistic transmission rate (also called the infection rate) integrating α and β is presented. Therefore, the epidemic is under the charge of three parameters, namely q , α and β , as time goes on. We show that, in the weighted cases, the networks with stronger community structure are no longer necessarily helpful in weakening the spreading of the disease. And, for a fixed strength of community structure, a larger β has a more obvious effect upon slackening the spreading of the disease.

2. Network model

In this section, we adopt a growth model to create a scale-free network that can exhibit community structure, of which the strength Q is adjusted by a probability parameter q , and then we propose a weighting pattern to make the networks weighted. The model algorithm is defined as follows.

- (1) Initially, start from a total of M communities in the whole network denoted by $C_1, C_2, \dots, C_{M-1}, C_M$ and there are a small number (m_0) of fully connected vertices in each community. In order to keep the whole network connected, we add a link between each two communities by connecting two vertices randomly chosen in each two communities, respectively.
- (2) At each time step, a new vertex is added into the network by becoming a member of a randomly chosen community. This new vertex will link to m ($< m_0$) different vertices in the same chosen community, and link to n vertices in other $M - 1$ communities

with probability q . Moreover, the first m internal edges in the same community C_h is chosen according to the preferential attachment mechanism, which means the probability that the new vertex will connect to the vertex i in the community C_h is $\prod(k_i) = k_i / \sum_{j \in C_h} k_j$. Then, for each one of the n external edges with probability q , choose a community C_s randomly among the remaining $M - 1$ communities and use the above preferential attachment mechanism to connect the new vertex to one vertex in C_s .

- (3) Repeat steps (2) until the vertices in the network amount to N and the evolution will be stopped when $t = N - m_0 * M$.

Then, we make the present network weighted by some weighting patterns. Among varieties of weighting patterns in complex networks, making use of the nodes' degrees to express the weights of edges is very important, namely the weight between two connected nodes i and j with degree k_i and k_j may be represented as a function of their degrees [36]–[38], i.e. $w_{ij} = w_0(k_i k_j)^\theta$, where the basic parameter w_0 and the weighting exponent θ depend on the particular complex networks (e.g. in the *E. coli* metabolic network $\theta = 0.5$; in the US airport network (USAN) $\theta = 0.8$ [40]; in the scientist collaboration networks (SCN) $\theta = 0$ [36]). It is noteworthy that the weight w_{ij} belongs to an edge between nodes i and j . Similarly, a single node can also be measured by weights: the strength s_i of node i (with degree k_i) is obtained by summing the weights of the links that are connected to it, i.e. $s_i = \sum_{j \in NS(i)} w_0(k_i k_j)^\theta$, where $NS(i)$ denotes the neighboring nodes' set of node i . The details of the weighting exponent θ will be discussed in section 3.

The degree distribution resulting from the above model can be conveniently obtained by using several approaches in the limiting of large network size, which is given as follows.

One can separate the degree k_i of node i into two components, i.e. k_i^{in} and k_i^{out} , which count the neighbors of node i in the same community and other communities, respectively. Then, $k_i = k_i^{\text{in}} + k_i^{\text{out}}$. Using the mean-field theory [39], one can obtain

$$\frac{\partial k_i^{\text{in}}(t)}{\partial t} = \frac{m}{M} \frac{k_i}{(2mt/M) + (qnt/M) + ((M-1)/M)qnt(1/(M-1))} = \frac{m(k_i^{\text{in}} + k_i^{\text{out}})}{2(m+qn)t}, \quad (1)$$

$$\begin{aligned} \frac{\partial k_i^{\text{out}}(t)}{\partial t} &= \frac{M-1}{M} \frac{qn}{M-1} \frac{k_i}{(2mt/M) + (qnt/M) + ((M-1)/M)qnt(1/(M-1))} \\ &= \frac{qn(k_i^{\text{in}} + k_i^{\text{out}})}{2(m+qn)t}. \end{aligned} \quad (2)$$

Consequently, $k_i^{\text{in}} = m(t/t_i)^{0.5}$, $k_i^{\text{out}} = qn(t/t_i)^{0.5}$, where t_i means node i is added into the network at time t_i and $k_i = (m+qn)(t/t_i)^{0.5}$. The probability of a node with degree $k_i(t) < k$ can be written as

$$P(k_i(t) < k) = P\left(t_i > \left(\frac{m+qn}{k}\right)^2 t\right) = 1 - \left(\frac{m+qn}{k}\right)^2 \frac{t}{t+m_0}. \quad (3)$$

Thus, we have

$$P(k) = \frac{\partial P(k_i(t) < k)}{\partial k} \simeq 2(m+qn)^2 k^{-3}, \quad (4)$$

which obviously shows scale-free behavior (of degree distribution).

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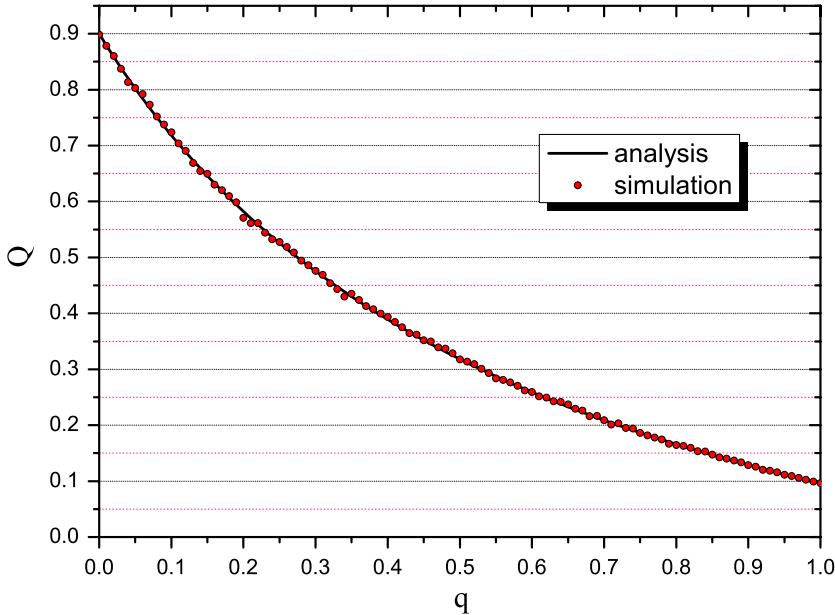


Figure 1. The modularity Q versus the control parameter q with $m_0 = 5$, $m = 4$, $n = 7$ and $M = 10$. The solid line is from equation (6) and the red circles are from the simulation. The total number of nodes N is 10^4 .

Another important measurement from the present model is the strength of community structure (also called modularity), which is proposed by Newman *et al* [34] (and modified by Kashtan *et al* [35]). Modularity Q can be defined as follows:

$$Q = \sum_{i=1}^M (e_{ii} - a_i^2), \quad (5)$$

where $a_i = \sum_{j=1}^M e_{ij}$ and e_{ij} denotes the ratio of the number of edges between community i and j to the total number of edges in the network. In terms of the present model, for large N , one can find that $e_{ii} = m/M(m + qn)$, $a_i = (m + 2qn)/M(m + qn)$. Thus, we have

$$Q = \frac{m}{m + qn} - \frac{1}{M} \left(\frac{m + 2qn}{m + qn} \right)^2. \quad (6)$$

Therefore, for the fixed values of m , n and M , one can adjust the values of q to get networks with various strengths of community structure. In figure 1, we show the relationship between the modularity Q and a full range of q . The solid line is from equation (6) and the red circles are from the simulation. As figure 1 shows, the modularity Q decreases as q changes from zero to one, namely the strength of community structure gradually weakens with the increasing of q . One can see that the simulation result coincides with our analytical result much better.

3. Epidemic model

In this paper, we adopt the susceptible–infected (SI) disease model [13, 14] and study epidemic spreading on the weighted network with communities, trying to understand how

the weights and community structure affect the dynamical process of disease spreading. As referred to in section 2, the edge between two nodes i and j is made weighted according to $w_{ij} = w_0(k_i k_j)^\theta$. Generally speaking, θ should be characterized by the edges' types which are internal edges and external edges in the present network. Thus, we introduce two weighting exponents: α for the internal edges and β for the external edges. That means if the two connected nodes are in the same community the weight on the edge is $w_{ij} = w_0(k_i k_j)^\alpha$, else the weight is $w_{ij} = w_0(k_i k_j)^\beta$. Here, according to most real networks, we set $\alpha, \beta > 0$ and $w_0 = 1$ without loss of generality. Similarly, the strength of node i should consist of s_i^{in} and s_i^{out} , which denote the sum of weights of all neighboring internal edges connected to node i and the sum of weights of all neighboring external edges connected to the node, respectively, namely we have that

$$s_k = s_k^{\text{in}} + s_k^{\text{out}}, \quad (7)$$

$$s_k^{\text{in}} = \sum_{j \in \text{INS}(i)} w_{ij} = \sum_{j \in \text{INS}(i)} w_0(k_i k_j)^\alpha, \quad (8)$$

$$s_k^{\text{out}} = \sum_{j \in \text{ENS}(i)} w_{ij} = \sum_{j \in \text{ENS}(i)} w_0(k_i k_j)^\beta, \quad (9)$$

where $\text{INS}(i)$ denotes the internal neighboring nodes' set of node i , i.e. the nodes in $\text{INS}(i)$ are in the same community with node i . $\text{ENS}(i)$ denotes the external neighboring nodes' set of node i which means the nodes in $\text{ENS}(i)$ are in the different communities with node i .

Here, the transmission rate on the edge from the infected node i to the susceptible node j will be redistributed by the proportion that the edge's weight accounts for, that is to say λ_{ij} can be defined as follows: when the edge is internal, we have

$$\lambda_{ij} = \lambda k_i^{\text{in}} \frac{w_{ij}}{s_i^{\text{in}}}, \quad (10)$$

and else, when the edge is external, we have

$$\lambda_{ij} = \lambda k_i^{\text{out}} \frac{w_{ij}}{s_i^{\text{out}}}. \quad (11)$$

From this we know the greater proportion of s_k^{in} (or s_k^{out}) that the weight w_{ij} of an internal (or external) edge accounts for, the more probability the disease transmits through the edge. For our modified SI model, the epidemic evolves by the following rules: initially, 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 (node) i acquires the infection at the transmission rate λ_{ji} in one contact with any neighboring infected individual j . Therefore, the total probability that the susceptible node i with degree k_i will be infected at time step t is given by $1 - \prod_{j \in \text{NIS}(i,t)} (1 - \lambda_{ji})$, where $\text{NIS}(i,t)$ denotes the neighboring infected nodes' set of i at time step t . Notice that, if we set $\alpha = \beta = 0$, then the transmission rate (on each edge) will be a constant λ , which is according to the standard SI model [13].

For a better understanding of prevalence induced by the epidemic spreading, we define the quantity $i_k(t) = I_k/N_k$ to be the density of infected nodes with degree k , where $I_k(t)$ and N_k represent the number of infected nodes and total nodes with degree k , respectively.

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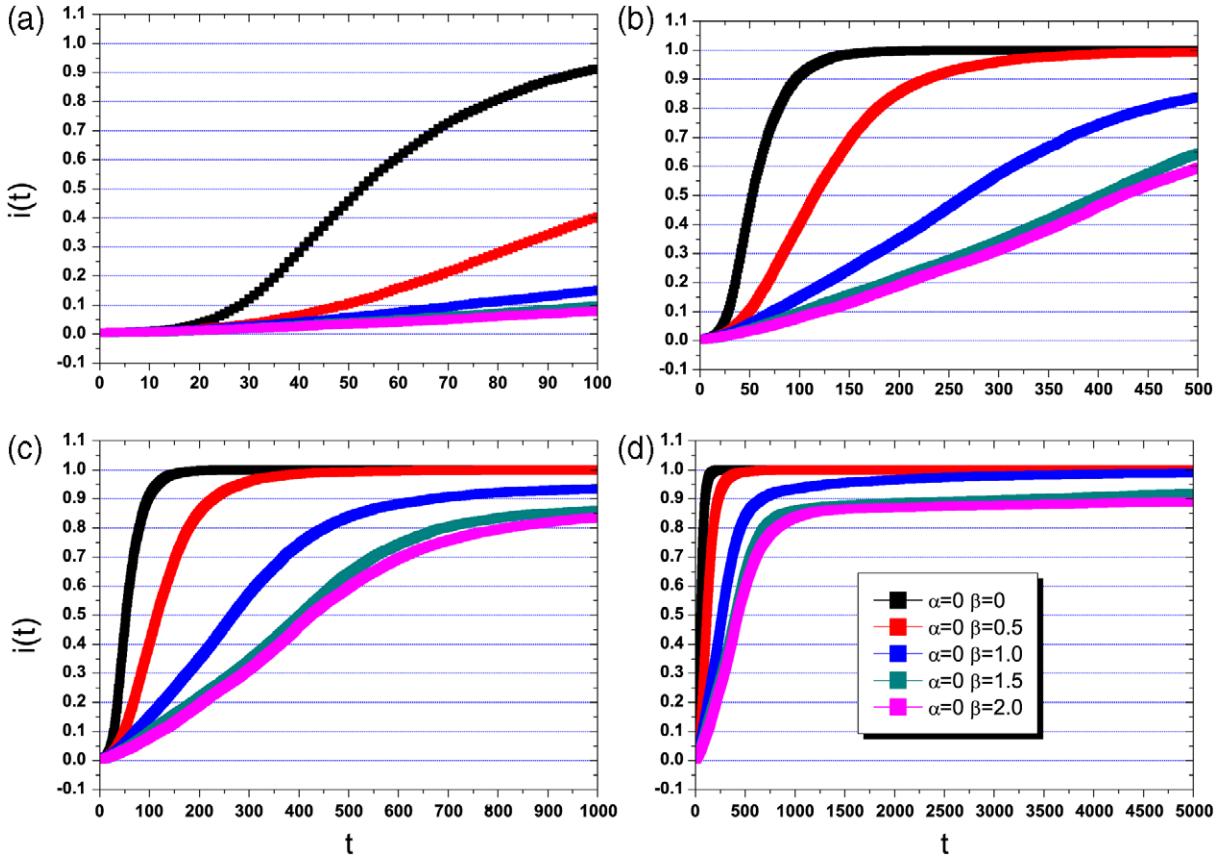


Figure 2. The time evolution of the fraction of infected nodes $i(t)$ with different values of β , namely $\beta = 0, 0.5, 1.0, 1.5$ and 2.0 . The other related parameters are $\lambda = 0.01$, $q = 0.1$ ($Q \simeq 0.72$) and $\alpha = 0$. (a)–(d) have different timescales for the purpose of better visibility.

Similarly, we have $s_k(t) = S_k/N_k$. 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. $i(t) = \sum_k P(k)i_k(t)$.

4. Simulation results and analysis

In this section, we apply the modified SI model described in the above section to the proposed network. For the initial conditions of disease diffusion, we select one node and assume it is infected; then the disease will spread throughout the network till all the nodes are infected (i.e. $\lim_{t \rightarrow \infty} i(t) = 1$). In our simulations, we have $m_0 = 5$, $m = 4$, $n = 7$, $M = 10$ and $N = 10^4$ without loss of generality, since the strength of community structure can be conveniently adjusted by the tunable parameter q . Therefore, the dynamical behaviors of disease diffusion are under the control of the modularity parameter q and the weighting exponents α and β .

First of all, we investigate the time behavior (also called temporal behavior) of the density of infected nodes (i.e. the prevalence $i(t)$) in the given networks with strong community structure ($Q \simeq 0.72$), as shown in figures 2–5. The simulations are averaged over 500 independent realizations.

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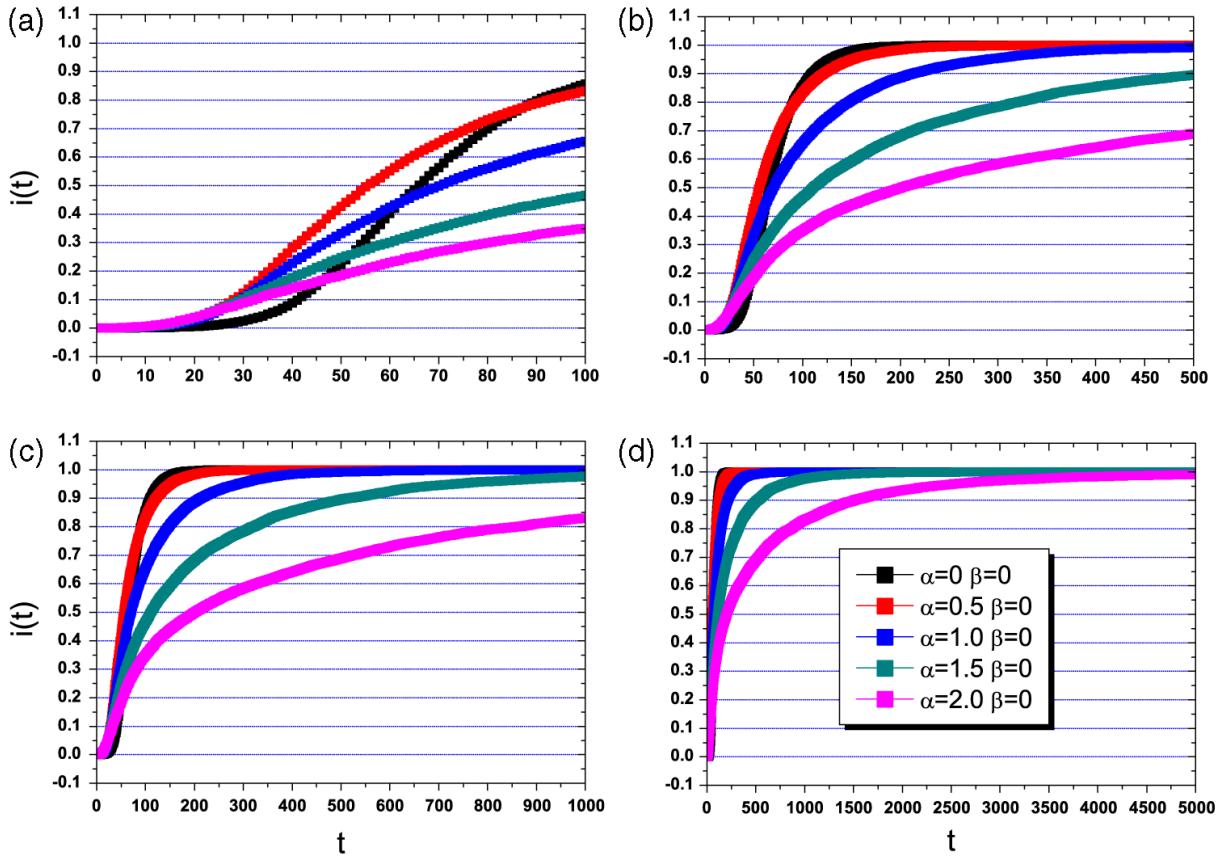


Figure 3. The time evolution of the fraction of infected nodes $i(t)$ with different values of α , namely $\alpha = 0, 0.5, 1.0, 1.5$ and 2.0 . The other related parameters are $\lambda = 0.01$, $q = 0.1(Q \simeq 0.72)$ and $\beta = 0$. (a)–(d) have different timescales for the purpose of better visibility.

In figure 2, we set $\alpha = 0$ and plot the time evolution of prevalence with various β . In this case, there are no weights on the internal edges and therefore the transmission rate on these internal edges will not be redistributed by the weights, namely the transmission rate is λ for each internal edge. We find that the external weighting exponent β has a strong impact upon the disease spreading, namely the larger the exponent β is, the more slowly the prevalence $i(t)$ grows. This result is valid for different stages of the prevalence growth including initial outbreak, accelerated growth and decelerated growth, as shown in figures 2(a)–(d).

In figure 3, we set $\beta = 0$ and plot the time evolution of prevalence with various α . In this case, the transmission rate is λ on each external edge, but redistributed by the weights on each internal edge. As figure 3(a) displays, for the weighted cases (i.e. $\alpha > 0$), the smaller the exponent α is, the more quickly the prevalence $i(t)$ grows, and this result is valid for different stages of the prevalence growth. While in the unweighted case (i.e. $\alpha = 0$), the prevalence $i(t)$ grows much slowly at the initial stage by comparing the weighted case, however, since the prevalence $i(t)$ in the unweighted case has a longer accelerated growth than that in the weighted cases. Thus, at a certain time, the prevalence

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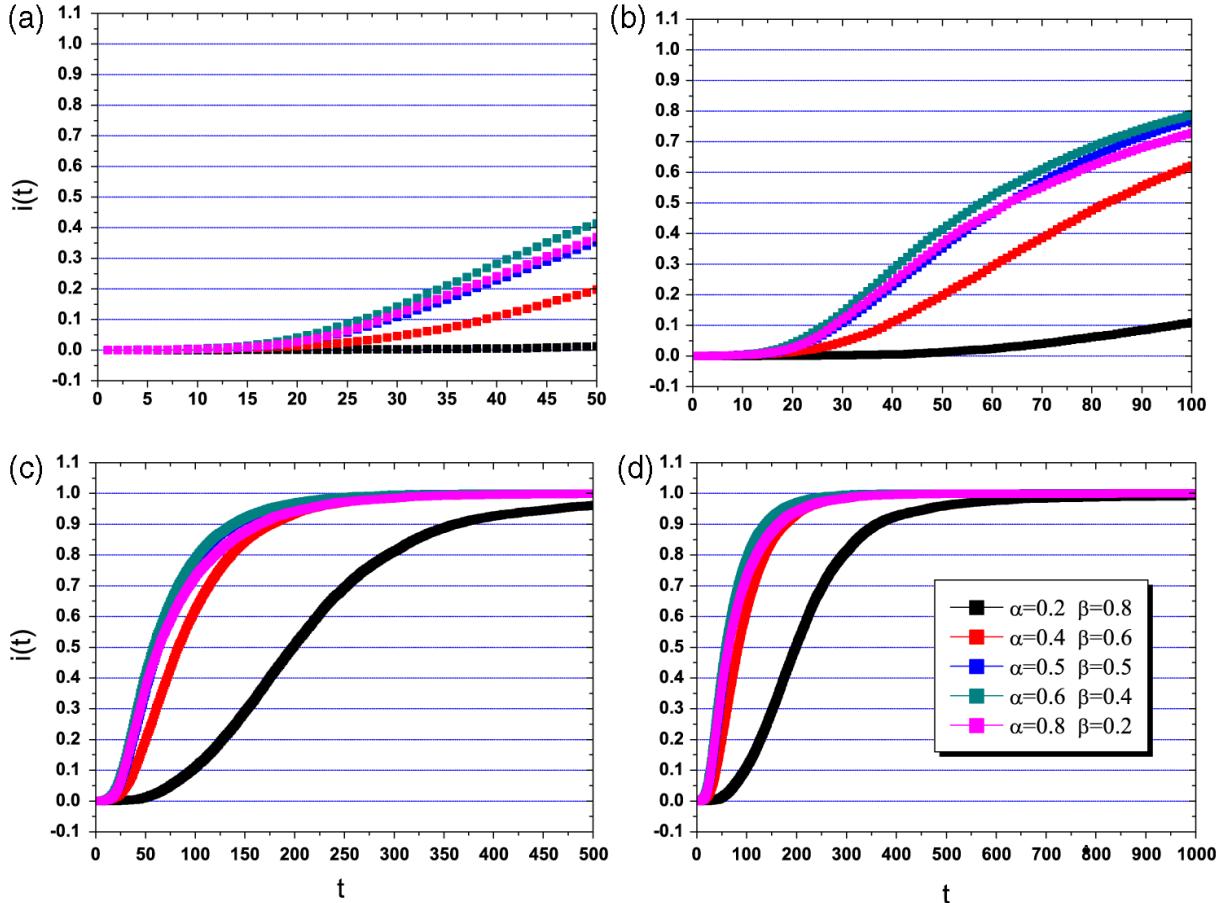


Figure 4. The time evolution of the fraction of infected nodes $i(t)$ with different configurations of α and β for a fixed sum $\alpha + \beta = 1$, namely $\alpha = 0.2, 0.4, 0.5, 0.6$ and 0.8 and accordingly $\beta = 0.8, 0.6, 0.5, 0.4$ and 0.2 . The other related parameters are $\lambda = 0.01$ and $q = 0.1$ ($Q \approx 0.72$). (a)–(d) have different timescales for the purpose of better visibility.

$i(t)$ for $\alpha = 0$ will go beyond the prevalence for $\alpha > 0$ (see figures 3(a) and (b)). And in the long term, the prevalence $i(t)$ in the unweighted case grows quicker than that in the weighted cases (see figures 3(c) and (d)).

From figures 2 and 3, one can obtain that the weights can reduce the prevalence induced by the epidemic, especially at the decelerated growth stage, and both the larger internal weighting exponent α or external weighting exponent β can have a full effect on delaying the epidemic spreading, which provides precious time for people to develop and dispose the control policies (such as throttling, immunization, etc) in advance of a large-scale prevalence $i(t)$.

In figures 4 and 5, we explore the relation between the prevalence and the different ratios of α to β (i.e. different α and β conditions). In these cases, both the internal edges and external edges are weighted (i.e. $\alpha \neq 0$ and $\beta \neq 0$). As shown in figures 4 and 5, the curves appear to have no apparent orderliness under the different conditions of α and β . However, if we classify these different conditions, there still is some useful information

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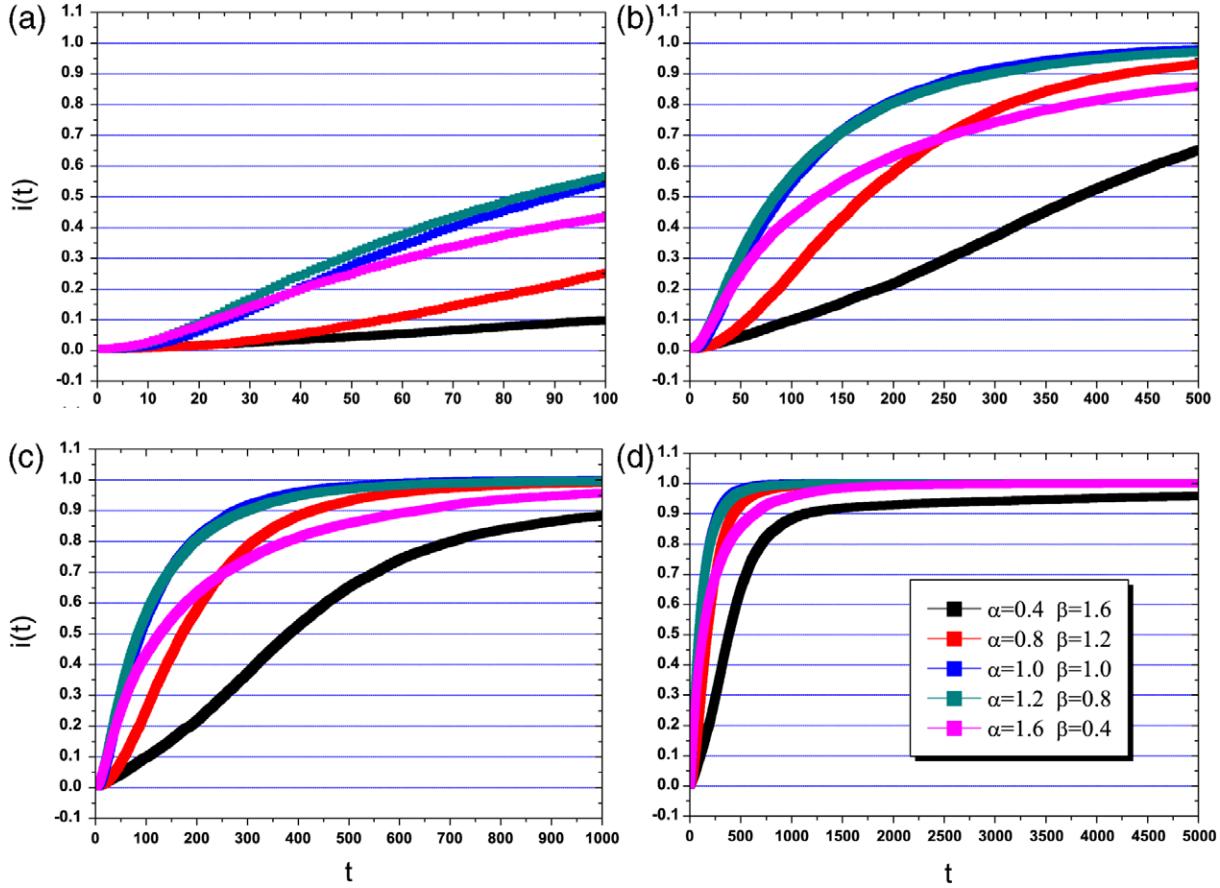


Figure 5. The time evolution of the fraction of infected nodes $i(t)$ with different configurations of α and β for a fixed sum $\alpha + \beta = 2$, namely $\alpha = 0.4, 0.8, 1.0, 1.2$ and 1.6 and accordingly $\beta = 1.6, 1.2, 1.0, 0.8$ and 0.4 . The other related parameters are $\lambda = 0.01$ and $q = 0.1$ ($Q \simeq 0.72$). (a)–(d) have different timescales for the purpose of better visibility.

which should be noticed. In figure 4, for the curves of $(\alpha/\beta) > 1$, the prevalence $i(t)$ behaves similarly at the initial stage; but as time goes on, it seems to be that the larger the ratio α/β is, the more slowly the prevalence $i(t)$ grows. Whereas for the cases of $(\alpha/\beta) \leq 1$, the curves show clearly that the prevalence grows much more slowly on decreasing α/β . These results also can be verified in figure 5, namely if $(\alpha/\beta) > 1$, the prevalence $i(t)$ grows slowly with increasing α/β ; else if $(\alpha/\beta) \leq 1$, the prevalence $i(t)$ grows much more slowly with decreasing α/β .

It is noted that, for figures 4 and 5, we fix the sum of α and β , i.e. the pairs of α and β satisfy $\alpha + \beta = 1$ and $\alpha + \beta = 2$ in figure 4 and figure 5, respectively. We find the sum also has an effect on the prevalence $i(t)$, namely for the same ratio α/β , the prevalence grows slowly with the sum increasing. In addition, from figures 4 and 5, one can see the parameter β is more sensitive for the prevalence $i(t)$, that is to say the external weighting exponent β has a stronger impact on slackening the epidemic spreading than the internal weighting exponent α .

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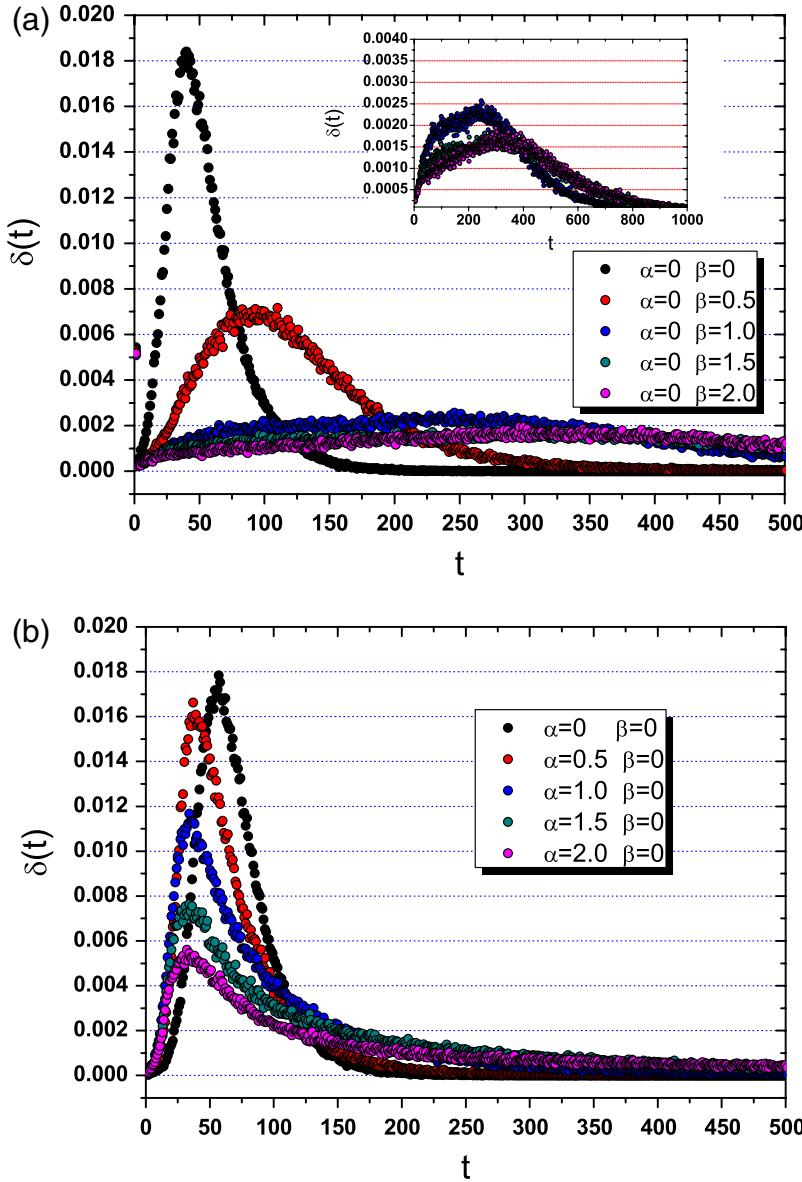


Figure 6. (a) Spreading velocity versus time with parameters $\alpha = 0$ and different values of β . The inset shows the spreading velocity in the cases of $\beta = 1.0, 1.5$ and 2.0 with a large timescale for better visibility. (b) Spreading velocity versus time with parameters $\beta = 0$ and different values of α . The other related parameters in both (a) and (b) are $\lambda = 0.01$ and $q = 0.1$ ($Q \simeq 0.72$).

To more deeply understand the temporal behaviors of the epidemic spreading, we study spreading velocity which can be written as [13]

$$\delta(t) = \frac{di(t)}{dt} \approx i(t) - i(t-1). \quad (12)$$

Obviously, as figures 6 and 7 display, the spreading velocity goes up to a peak at a certain time step t where the prevalence $i(t)$ has the fastest growth rate, then the spreading

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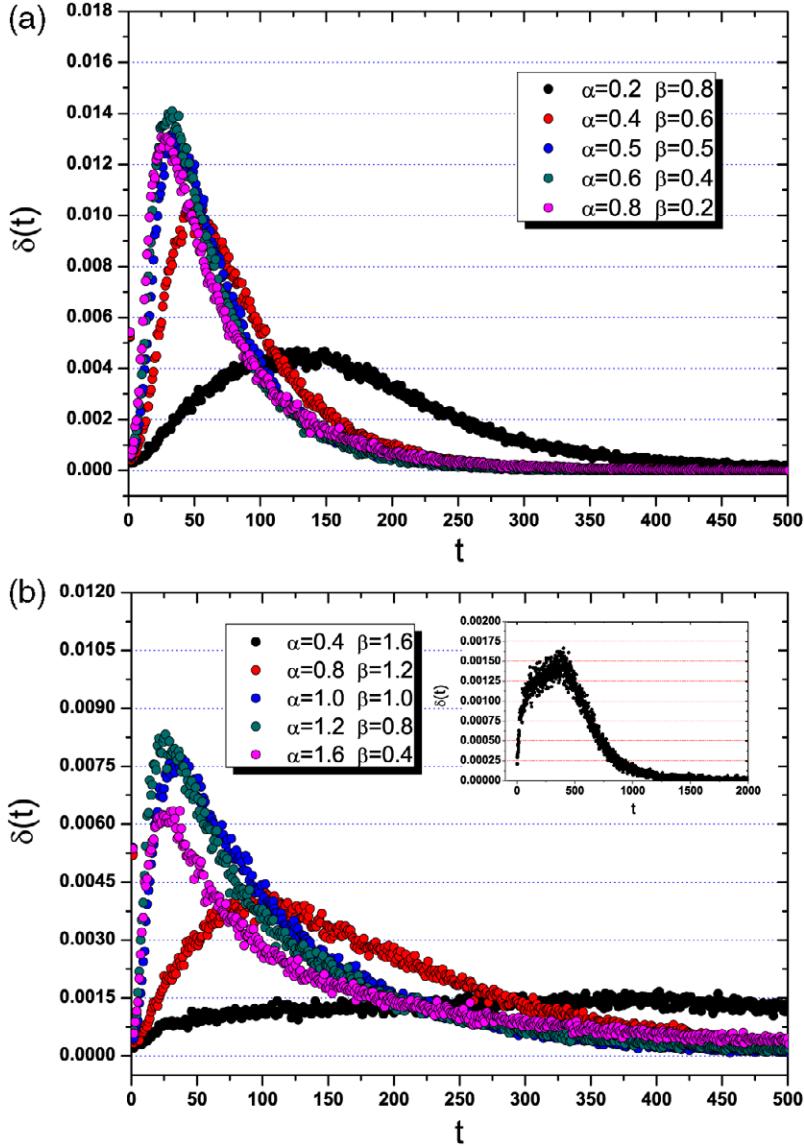


Figure 7. Spreading velocity versus time with different values of α and β for $\alpha + \beta = 1$ in (a) and $\alpha + \beta = 2$ in (b), respectively. The other related parameters are $\lambda = 0.01$ and $q = 0.1$ ($Q \simeq 0.72$). The inset in (b) shows the spreading velocity in the case of $\alpha = 0.4$ and $\beta = 1.6$ with a large timescale for better visibility.

velocity reduces to zero. That is to say, the prevalence $i(t)$ undergoes an accelerated growth before the time step t , and then undergoes a decelerated growth after the time step t . This time step t also is referred as the ‘peak’ time, which means a transition from the small incidence of the epidemic to a macroscopic outbreak.

Concretely, in figure 6(a) where we have $\alpha = 0$ for all the curves, it is observed that the larger the exponent β is, the more slowly the spreading velocity arrives at the peak time, and the spreading velocity at the peak time (i.e. the fastest growth rate) also decreases on increasing β . These features are consistent with the results obtained from figure 2. In figure 6(b) where we have $\beta = 0$ for all the curves, one can see the value

of spreading velocity at the peak time decreases as α increases. The peak time also is brought forward as α increases, which means the prevalence with larger α might have a shorter accelerated growth. Of course, a smaller peak time also makes the growth of $i(t)$ arrive at the decelerated growth stage more quickly as shown in figure 3.

In figures 7(a) and (b), we have $\alpha + \beta = 1$ and $\alpha + \beta = 2$, respectively, and these figures might help us to explore the effect of the ratio α/β on the spreading velocity. We find that $\delta(t)$ increases and arrives at the peak time more quickly for $(\alpha/\beta) > 1$ than the one for $(\alpha/\beta) < 1$. Also it is noted that there is no distinct change for the ratios with $(\alpha/\beta) > 1$, but the diversification of spreading velocity induced by different ratios arises in the case of $(\alpha/\beta) < 1$. Namely, a smaller ratio of α to β might give rise to a larger peak time and a much smaller value of fastest growth rate at the peak time (see the case of $\alpha = 0.4$ and $\beta = 1.6$). In addition, the sum of α and β can also influence the fastest growth rate. As shown in figure 7, for the same ratio, the larger the sum is, the much smaller the fastest growth rate at the peak time is. From figures 7(a) and (b), one can obviously see the external weighting exponent β is much more sensitive than the internal weighting exponent α in the present epidemic system, and for the systems with the smaller values of α/β , the diseases will diffuse with difficulty.

In the above investigations, we have studied the influence of the internal weighting exponent α and external weighting exponent β on the prevalence given by a network with strong community structure ($Q \approx 0.72$). Now we study the influence of different strengths of community structure on the prevalence $i(t)$, in other words, we study the effect of the probability parameter q on the prevalence $i(t)$. As referred to in section 2, the relation between the parameter q and the modularity Q is that the larger the parameter q is, the smaller the modularity Q is.

In figure 8, we plot the prevalence $i(t)$ with different strengths of community structure for the unweighted case ($\alpha = 0, \beta = 0$) which also corresponds to the standard SI model. Notice that figure 8 is a color contour map, i.e. the Y coordinate stands for the various q values from 0 to 0.5, the X coordinate stands for the time step t and the corresponding values of $i(t)$ are shown by the different color coding. In figure 8, one can see that the smaller the parameter q is, the slowly the prevalence $i(t)$ grows. That is to say the strong community structure (namely a smaller value of q) is prone to reduce the growth of the prevalence $i(t)$, which is consistent with the result obtained from [28]. However, is the result necessarily valid for the weighted cases?

To answer this question, in figure 9, we plot the prevalence $i(t)$ with different strengths of community structure for the weighted cases and the figure consists of nine color contour maps which represent different configurations of α and β , as figures 9(a)–(i) display. We find the influence of q behaves very diversely with different α and β . It is noted that the slackening effect on the prevalence growth contributed by the strong community structure is still valid for the cases of $\beta = 0$ as figure 9(d) ($\alpha = 1.0, \beta = 0$) and figure 9(g) ($\alpha = 2.0, \beta = 0$) display. But, if we weight the external edges by increasing β , the strong community structure no longer reduces the danger brought by the epidemic spreading in the long term. For example, in figure 9(f) where we have $\alpha = 1$ and $\beta = 2$, despite at the initial stage the prevalence $i(t)$ in the networks with smaller q (i.e. larger Q) grows more slowly, however, as time goes on, it is obviously shown that the prevalence $i(t)$ in those networks with strong community structure grows much quickly than those with weak community structure. And for figure 9(f), one can see that the larger the parameter q is,

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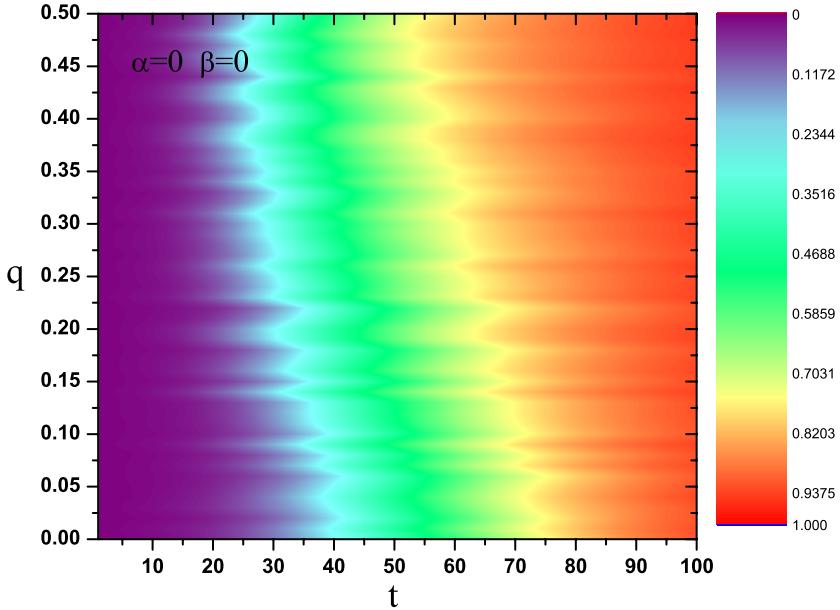


Figure 8. The time evolution of the fraction of infected nodes $i(t)$ with the probability parameter q ranging from 0 to 0.5 (i.e. Q from 0.898 56 to 0.317 649) in the unweighted case (i.e. $\alpha = 0$, $\beta = 0$) which corresponds to the standard SI model. The basic transmission rate is $\lambda = 0.01$.

the better the network resists the disease diffusion. That is to say, the influence of strong community structure in the unweighted case is no longer valid for the weighted case, actually the strong community structure has a reverse effect on the prevalence $i(t)$ compared with that in the unweighted case. The reason, we believe, can be attributed to the numbers of the external edges in the networks. As we know, the network with a larger q shows the weak community structure, i.e. the network has less internal edges and more external edges. From the previous analysis we obtain that the external weighting exponent β has a strong effect on reducing the disease diffusion. Therefore the prevalence $i(t)$ grows more slowly in the networks with more external edges. Furthermore, one can see figure 9(c) as a typical illustration. In figure 9(c), we have $\alpha = 0$ and $\beta = 2.0$, which means only external edges are weighted. In this case, the prevalence $i(t)$ in the networks with weak community structure grows much more slowly than that with strong community structure. Figure 9(c) presents a striking contrast to figures 9(a), (d) and (g) where no edges are weighted or only internal edges are weighted. In addition, from figures 9(e), (h) and (i), one can also find that the strong community structure unnecessarily might help to reduce the disease diffusion in the networks.

Another precise characterization of the epidemic spreading can be achieved by studying the average degree of the newly infected individuals at time step t [13, 14], which can be written as

$$\langle k_{\text{inf}}(t) \rangle = \frac{\sum_k k I_k(t) - \sum_k k I_k(t-1)}{I(t) - I(t-1)}, \quad (13)$$

where $I_k(t)$ stands for the number of infected nodes with degree k at time step t .

In figures 10(a)–(d), we plot the temporal behavior of $\langle k_{\text{inf}}(t) \rangle$ for the different α and β conditions on our weighted scale-free networks with communities. As displayed

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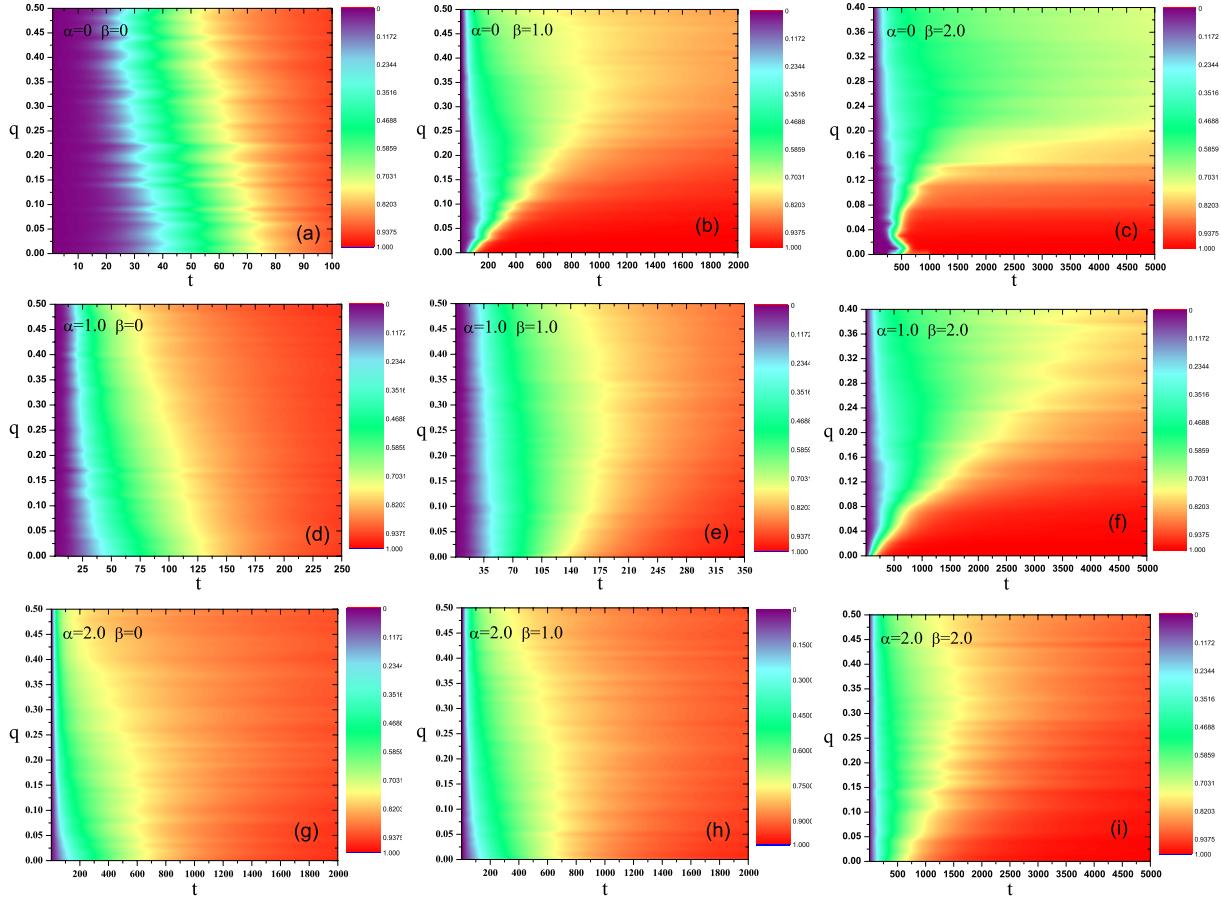


Figure 9. The time evolution of the fraction of infected nodes $i(t)$ with the probability parameter q ranging from 0 to 0.5 (i.e. Q from 0.898 56 to 0.317 649) in the weighted cases. The basic transmission rate is $\lambda = 0.01$. (a)–(i): the different weighted cases with different α and β conditions. Color coding is identical to figure 8.

in these figures, all the curves show the same hierarchical dynamics which also appeared in the famous Barabási-Albert scale-free networks [13]. That is to say, the infection pervades the networks in a progressive cascade from higher degree classes to lower degree classes. Therefore, the probable process of epidemic spreading on the weighted networks with community structure is: after the hubs are very quickly infected, spreading is going always towards the smaller degree nodes, namely the nodes with the lowest degree are the last infected.

5. Conclusion

To sum up, in this paper, we have qualitatively investigated the epidemic spreading in weighted scale-free networks with community structure, where two exponents α and β are introduced to weight the internal edges and external edges, respectively. The strength of community structure is also adjusted by a probability parameter q . We have

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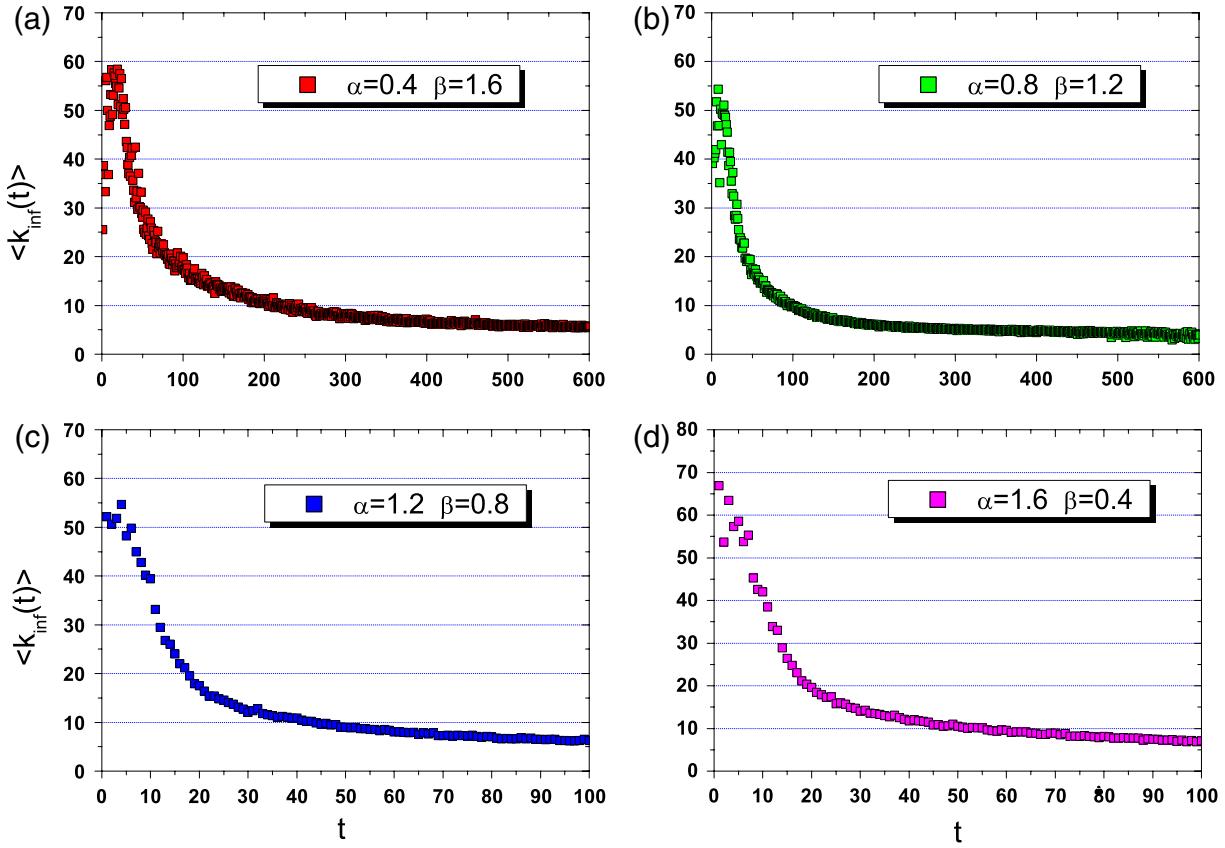


Figure 10. Temporal behavior of the average degree of the newly infected nodes with the different values of α and β . The other related parameters are $\lambda = 0.01$ and $q = 0.1$.

studied the temporal behavior and the spreading velocity of the prevalence $i(t)$ with different configurations of α and β . We find the infection pervades the networks with communities slower in the weighted case than that in the unweighted case (corresponding to $\alpha = 0, \beta = 0$). The external weighting exponent β plays a much more important role in slackening the epidemic spreading and reducing the danger brought by the epidemic than the internal weighting exponent α . That is to say, increasing β can efficiently reduce the epidemic diffusion by decreasing the spreading velocity and increasing the peak time.

Furthermore, we have also explored how the strength of community structure affects the epidemic spreading in the weighted cases. The result we obtain is not the same as that in [28], where the strong community structure (i.e. a smaller q) can be helpful in reducing the danger brought by the epidemic prevalence. From figure 9, we find the strong community structure no longer makes the disease spread more difficult in the weighted cases, especially in the cases with a larger β .

In addition, we have also studied the propagation of the infection through different degree classes in the weighted networks with communities. The hierarchical propagation of epidemic spreading in the present networks is typically exhibited as that in Barabási-Albert networks, i.e. the infection pervades the networks from the higher degree classes to the lower degree classes.

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In a way, epidemic spreading can be managed as a reaction-diffusion process [41]–[43], which also has a very close relation with information retrieval, influence spreading and so on. Actually the efficient diffusion or inefficient diffusion has its merits in special natural and artificial networks. Our work, we believe, not only provides deeper insights into the dynamical behaviors of epidemic spreading, but also might deliver some useful information or new insights into the corresponding control strategies.

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