Journal of Statistical Mechanics: Theory and Experiment
An IOP and SISSA journal

Epidemic spreading in scale-free networks with community structure

Wei Huang and Chunguang Li[1]

Centre for Nonlinear and Complex Systems, School of Electronic Engineering, University of Electronic Science and Technology of China, Chengdu 610054, People's Republic of China
E-mail: huangweixp@yahoo.com.cn and cgli@uestc.edu.cn

Received 23 November 2006
Accepted 20 December 2006
Published 17 January 2007

Online at stacks.iop.org/JSTAT/2007/P01014
doi:10.1088/1742-5468/2007/01/P01014

Abstract. Many real networks consist of communities: groups of nodes within which connections are dense, but among which connections are sparser. In this paper, based on the susceptible–infected (SI) model, epidemic prevalence is compared between two kinds of scale-free network, with and without community structure, which have the same degree distribution. It is found that networks with strong community structure are helpful for reducing the danger brought by epidemic prevalence in the network, which is shown by the comparison results of the prevalence scale, the variability of the prevalence scale and the average degree of new infected nodes at each time step. We also note that when facing networks with strong community structure, special attention should be paid to nodes that are close to the initial infected seeds.

Keywords: network dynamics, random graphs, networks

J. Stat. Mech. (2007) P01014

[1] Author to whom any correspondence should be addressed.

 1742-5468/07/P01014+13$30.00

Contents

1. Introduction

Studies on complex networks, including technological, social and biological networks of various kinds, have attracted significant attention in recent years [1, 2]. Scientists find that there exist various topological structures of networks in real life. Notably, it is found that many complex networks are scale-free [1]–[4], which means that the degree distributions of these networks follow a power-law form $P(k) \sim k^{-\gamma}$, where $P(k)$ is the probability that a node in the network is connected to k other nodes and γ is a positive real number determined by the given network. Another significant finding recently is that many real-life networks present *community structure* [9, 10]. That is, there are groups of nodes with many connections between their members and few connections to nodes outside the group. The community may be classmates, friends, co-workers, club members, etc.

Dynamical processes taking place in complex networks, such as virus spreading [5, 8], rumour propagation [6] and information propagation [7], are one of the most important subjects in this field. In the last few years there have been intensive studies of epidemic spreading in complex networks, especially in scale-free networks. In [8], the authors have shown that the growth of the epidemic prevalence is virtually instantaneous in scale-free networks characterized by diverging degree fluctuations, independently of the structure of the connectivity correlation functions characterizing the population network. There has also been some research revealing the influence of community structure upon epidemic spreading in complex networks. In [11], the authors proposed a growth model to create a scale-free network with a tunable strength (denoted by Q) of community structure and investigated the influence of the community structure upon the susceptible–infected–removed–susceptible (SIRS) epidemiological process. They pointed out that a transition occurs as Q changes from 1 to 0 for the global synchronization, and that the local synchronization behaves very differently. In [12], the authors found that, compared with the random network, the community network has a broader degree distribution, a smaller threshold of epidemic outbreak, and more prevalence to keep the outbreak endemic. However, there is little work revealing differences of virus spreading in scale-free networks with and without communities (hereafter we will call them SFcN and SFN for short) when both networks are assigned to the same degree distributions. In this study we shall present a study of epidemic spreading in SFcNs with different strengths

doi:10.1088/1742-5468/2007/01/P01014

J. Stat. Mech. (2007) P01014

of community structure and their corresponding SFNs, discovering that differences of virus prevalence effects between the two scale-free network kinds vary with the strengths of community structure of the SFcN. By revealing how community structure affects the infection propagation, our research can further shed more light on designing effective disease immunization measures in real-life networks.

In order to study the effects of community structure in epidemic spreading, we will focus on the standard susceptible–infected (SI) model in which all individuals are classified into two distinct states, namely susceptible (S) and infected (I). Each individual is represented by a node of the network and the links represent the connections along which infection may spread. We assume that the epidemic is propagated between susceptible and infected individuals at a rate of λ, and that once a susceptible individual is infected it remains in this state. The SI model on both the SFcN and the SFN is well adapted to characterize the differences of prevalence effects between SFcNs and SFNs, although our study can be easily extended to other epidemiologic models such as susceptible–infected–susceptible (SIS) and susceptible–infected–removed (SIR) models.

2. Network model

In our study, we adopt the evolving undirected network model proposed in [9], which exhibits community structure. The network model is based on the inner-community preferential attachment and inter-community preferential attachment mechanisms. The network model follows power-law degree distributions, which are observed in many real-life networks. Also the strength of community structure for this network model can be varied by adjusting a tunable parameter. The model assumes there is a total of M ($M \geq 2$) communities in the network. The model is defined by the following scheme.

Step 1: Initialization. Start from a small number m_0 ($m_0 > 1$) of fully connected nodes in each community. Add a link between every two communities, so that there are altogether $M(M-1)/2$ inter-community links. The nodes to which inter-community links are connected are randomly chosen in each community.

Step 2: Growth. At each time step, a new node is added to a randomly selected community. The new node will be connected to m ($1 \leq m \leq m_0$) nodes in the same community through *inner-community links* (defined as the links that connect nodes in the same community) and with probability α connected to n ($1 \leq n \leq M$) nodes in other $M-1$ communities through *inter-community links* (defined as the links that connect nodes among different communities).

Step 3: Preferential attachments.

(a) Inner-community preferential attachment. When choosing nodes in the same community (we denote it as the jth community) to which the new node connects through inner-community links, we assume that the probability Π that a new node will be connected to node i in community j depends on the inner-degree s_ij (defined as the number of inner-links connected to node i) of that node, such that

$$\Pi(s_{ij}) = \frac{s_{ij}}{\sum_k s_{kj}}. \tag{1}$$

(b) Inter-community preferential attachment. When choosing the nodes in other communities to which the new node connects through inter-community links, we assume that the probability Π that a new node will be connected to node i in community k

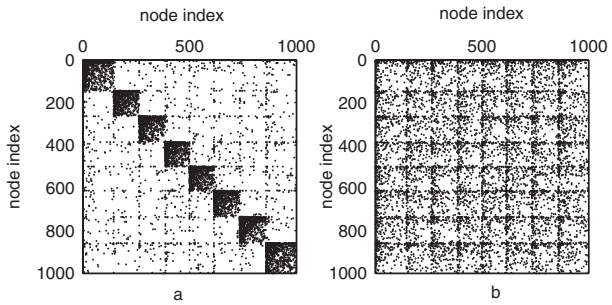

Figure 1. Sparsity pattern of the adjacent matrix for an SFcN (a) and an SFN (b) with the number of nodes $N = 1000$. The parameter α controlling the strength of community structure of the SFcN is set to be 0.05.

$(k \neq j)$ depends on the inter-degree l_{ik} (defined as the number of inter-links connected to the node), such that

$$\Pi(l_{ik}) = \frac{l_{ik}}{\sum_{m,n,n\neq j} l_{mn}}. \tag{2}$$

After a certain number of time steps, this scheme generates an SFcN with the number of nodes N. The motivation for adopting this network-evolving model is that there exist both inner-community and inter-community preferential attachment mechanisms in real-life networks [9].

In order to analyse the differences of prevalence effects brought by peer community structure consideration, an SFN, which has the same degree distribution as the corresponding SFcN, is generated according to the switching algorithm mentioned in [13]. Following the algorithm, we start from a given SFcN network and carry out a series of Monte Carlo switching steps whereby a pair of links (A–B, C–D) is selected at random and the ends are exchanged to give (A–D, C–B). The exchange is only performed if it generates no multiple links or self-links; otherwise it is not performed. To ensure that the new network obtained shows good mixing, we switch altogether $5M$ pairs (they are mutually independent) of links. Figure 1 shows the sparsity pattern of the adjacent matrix for an SFcN, as well as an SFN with the same degree distribution. We can observe that figure 1(a) (for the SFcN) presents distinct community structure while the dots on figure 1(b) (for the SFN) mix with each other uniformly, which means that the SFN we obtained using the switching algorithm from an SFcN shows no community structure. The average shortest path lengths L are also calculated for both scale-free network kinds. It is observed from figure 2 that the average shortest path lengths decay with the increasing of α for both network kinds. A more important point is that the average shortest path lengths of SFcNs are obviously larger than those of SFNs.

3. Measurements

In order to measure the strengths of community structure for complex networks, reference [14] proposed the following measurement:

$$Q = \sum_{i=1}^{M} (e_{ii} - a_i^2), \tag{3}$$

J. Stat. Mech. (2007) P01014

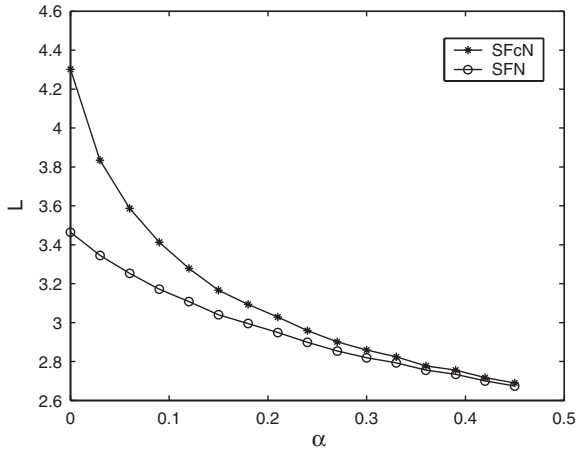

Figure 2. Average length of shortest path lengths versus the control parameter α for an SFcN and an SFN with the number of nodes $N = 500$. Asterisks and circles are results for SFcNs and SFNs respectively. Solid lines are guides for the eyes.

where $a_i = \sum_{j=1}^{M} e_{ij}$ and e_{ij} is the fraction of links in the network that connect nodes in communities i and j. According to the network-evolving model in [9], when the number of nodes is large, we have

$$e_{ii} = \frac{m/M}{m + \alpha n} = \frac{m}{M(m + \alpha n)}, \tag{4}$$

$$a_i = e_{ii} + \sum_{j \neq i} e_{ij} = \frac{m/M}{m + \alpha n} + \frac{\alpha n/M}{m + \alpha n} + \frac{(\alpha n/(M-1))((M-1)/M)}{m + \alpha n} = \frac{m + 2\alpha n}{M(m + \alpha n)}. \tag{5}$$

Substituting equations (4) and (5) into (3), we obtain

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

Therefore we can fix m and n and adjust the values of α to get networks with various strengths of community structure. Figure 3 depicts the relationship between Q and α for SFcNs. The solid line is plotted according to equation (6) and asterisks are calculated on the SFcN model using the mathematical software *MATLAB*. We see that the two results coincide with each other quite well and that the strengths of community structure gradually weaken with the increasing of α. In addition, by using the algorithm mentioned in [14], we also calculated the values of the modularity Q for the corresponding SFNs[2], which are indicated by circles in figure 3. The value of Q always fluctuates around zero for SFNs, which gives a further evidence that a randomized SFN does not present community structure.

An important measurement for the virus prevalence effect is the epidemic variability mentioned in [15]. The variability plays an important role in the accuracy and the forecasting capabilities of numerical models and has thus to be quantified in order to assess

[2] Following [14], we only picked up the peak value of Q corresponding to a perfect identification of the communities.

J. Stat. Mech. (2007) P01014

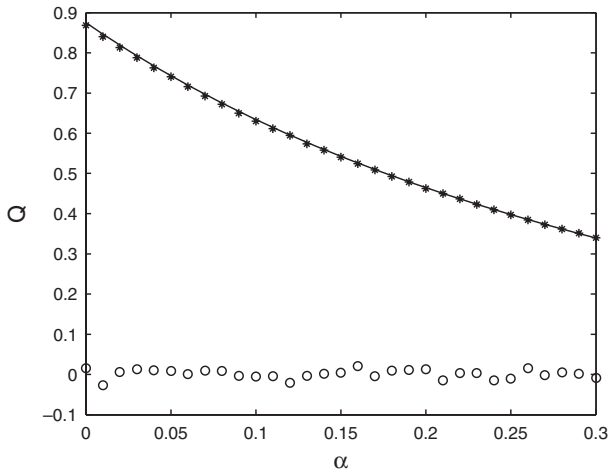

Figure 3. The relationship between the strength of community structure Q and the control parameter α. Asterisks are calculated on the SFcN model and the solid line is plotted according to equation (6) with $m = 4$, $n = 7$ and $M = 8$. They coincide with each other quite well. Circles are calculated for the corresponding SFNs using the algorithm mentioned in [14]. The network size N is set to be 2000.

the meaningfulness of simulations with respect to real outbreaks [16]. The variability of outbreaks as the relative variation of the prevalence (density of infected individuals $i(t)$) is given [15] by

$$\Delta[i(t)] = \frac{\sqrt{\langle i^2(t)\rangle - \langle i(t)\rangle^2}}{\langle i(t)\rangle}. \tag{7}$$

The variability $\Delta[i(t)]$ measures the predictability of the density of infected individuals $i(t)$ at each time step. Larger $\Delta[i(t)]$ means worse predictability while smaller $\Delta[i(t)]$ denotes a better one. Research on epidemic variability can enlighten people to make good predictions about future possible cases of virus prevalence in complex networks, which will facilitate people being able to adopt effective measures against possible disease prevalence. In the same way, we can define variabilities of other quantities, such as the average degree $d(t)$ of new infected nodes at each time step t.

4. Results for prevalence effects on an SFcN and its randomized counterpart SFN

In reality, the community network will be dynamic where new people are added (born) into the communities and old people are removed (die) from the communities. Compared with the period between birth and death, epidemic spreading is a fast process and hence can be considered as a spreading on a static network. In the present work, we have performed Monte Carlo (MC) simulations of the model with synchronous updating on a series of static networks following the rule of the SI model. First let us see a typical example with the parameter $\alpha = 0.01$. In this case the SFcN shows strong community structure (the value of Q is around 0.845). Simulations were implemented averaging over 30 different network configurations, and for each configuration we took 500 different initial conditions with exactly one randomly chosen node infected. We find that virus

J. Stat. Mech. (2007) P01014

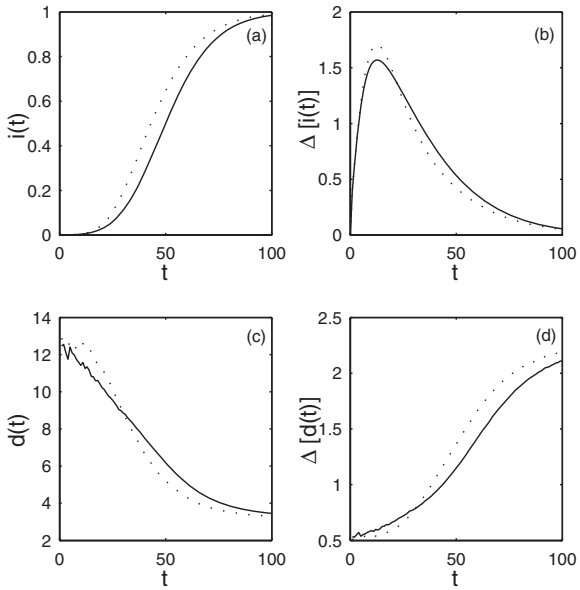

Figure 4. Epidemic spreading on an SFcN and an SFN with the number of nodes $N = 2000$, the control parameter $\alpha = 0.01$ and the infection rate $\lambda = 0.03$. Solid and dotted lines represent cases for the SFcN and the SFN respectively. The four panels represent: (a) time evolution of fraction of infected individuals $i(t)$, (b) variability of $i(t)$, (c) average degree of new infected nodes $d(t)$ and (d) variability of $d(t)$ for both network kinds.

prevalence on the SFN is heavier than that on the corresponding SFcN, as is shown in figure 4(a). One of the reasons might be the discrepancy of average shortest path lengths between the two scale-free network kinds. Another reason attributed to the different behaviour is that a community would tend to 'keep' the epidemics inside itself until at least one node in another community is infected. To confirm this, we have investigated the average time $t_{\rm esc}$ that it takes for the epidemics to escape a community and infect another in an SFcN, as is shown in figure 5(a). It can be seen that $t_{\rm esc}$ monotonically decays with the increasing of α, which indicates that when network presents stronger community structure, it takes more time for an epidemic to enter into a new community from the community in which the initial node is infected in the beginning. Figure 5(b) shows the average fraction of infected individuals $i_{\rm esc}$ for both SFcNs and their randomized counterpart SFNs when the Monte Carlo iteration time steps have just reached $t_{\rm esc}$. We find that $i_{\rm esc}$ for the SFcN is obviously smaller than that for the SFN, which can well explain the observed fact that virus prevalence on the SFN is heavier than that on the SFcN.

Figure 4(b) displays the time evolution of $\Delta[i(t)]$ for outbreaks starting from one randomly selected infected seed. This figure shows that the peak value of $\Delta[i(t)]$ for outbreaks on the SFN is larger than on the SFcN, implying that communities can play a role in weakening the epidemic variability. However, at the later stage it is the reverse case, i.e. the later stage of epidemic spreading tends to be more variable on the SFcN. This result leads people to investigate more thoroughly the discrepancy of epidemic prevalence between SFcNs and SFNs.

J. Stat. Mech. (2007) P01014

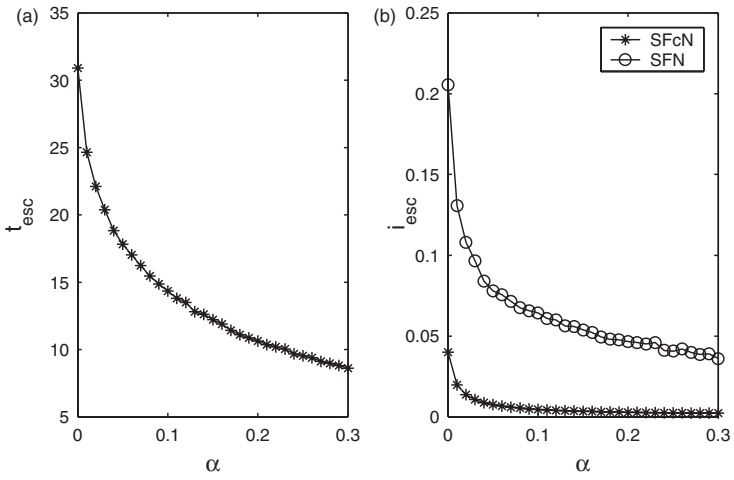

Figure 5. Plots for: (a) the average time $t_{\rm esc}$ that it takes for an epidemic to enter into a new community from the community in which the initial node is infected in the beginning (for the SFcN only); (b) the fraction of infected individuals $i_{\rm esc}$ for the SFcN and SFN at the time when epidemic spreads into a new community. The network size N and infection rate λ are set to be 2000 and 0.03 respectively.

In addition, we also investigated the average degree of new infected nodes $d(t)$ at each time step t. Both monotonically decreasing curves plotted in figure 4(c) show that superspreaders (i.e. large-degree nodes) are usually infected at the early stage of an outbreak. It is well known that the infection of large-degree nodes at the early stage of an outbreak can lead to much heavier virus prevalence in the network, as compared to small-degree nodes, which can be called a *large-degree effect*. This is also the reason that usually large-degree nodes are first vaccinated when people try to prevent large-scale spreading of diseases in the network. This strategy has proved to be efficient [17]. From figure 4(c) we can find that the average degree of new infected nodes in an SFcN is smaller than that in an SFN at the early stage of an outbreak. The fact implies that the SFcN, which weakens the large-degree effect, leaves people more time to protect large-degree nodes. Figure 4(d) shows how $\Delta[d(t)]$ (the variability of average degree of new infected nodes at each time step t) evolves with time steps for both network kinds. One might think that at the early stage of epidemic spreading, an SFcN possesses a lower overall variability of $d(t)$ because the nodes are restricted to several communities; however, this is not the case. An SFcN possesses a larger $\Delta[d(t)]$ at the early stage of epidemic spreading, while at the later stage it is the reverse case.

In figure 6 we give the systematic plots for epidemic prevalence on SFcNs with different strengths of community structure and their randomized counterparts SFNs; this consists of four colour contour maps. The network parameters are the same as those in figure 4. The four panels show the difference of $i(t)$, $\Delta[i(t)]$, $d(t)$, $\Delta[d(t)]$ between the SFcN and the SFN respectively, namely

$$\delta(i(t)) = i_{\rm SFcN}(t) - i_{\rm SFN}(t), \tag{8}$$

$$\delta(\Delta[i(t)]) = \Delta[i_{\rm SFcN}(t)] - \Delta[i_{\rm SFN}(t)], \tag{9}$$

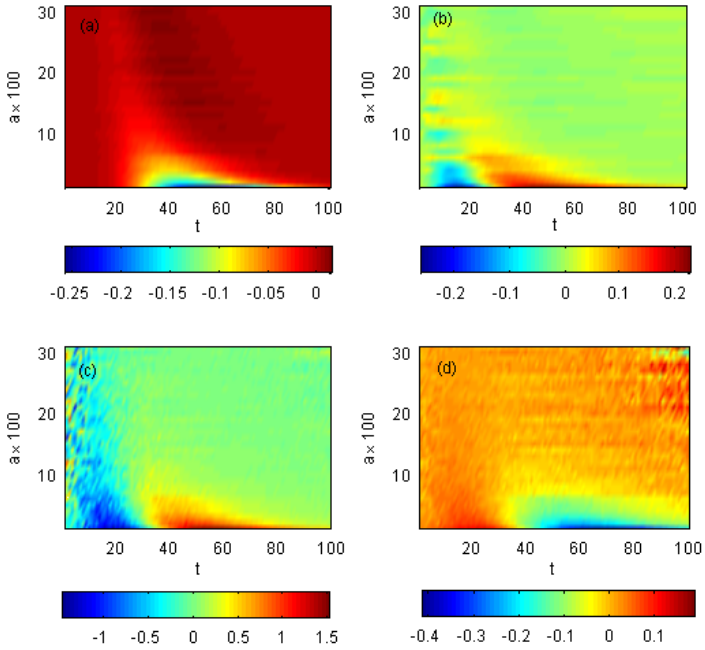

Figure 6. Plots for prevalence effects in SFcNs and SFNs with the number of nodes $N = 2000$, the infection rate $\lambda = 0.03$ and the control parameter α ranging from 0 to 0.3. Initially only one node is randomly selected and assumed to be infected. (a) Difference of fractions of infected individuals $\delta(i(t))$ versus time steps. (b) Variability of $\delta(i(t))$. (c) Difference of average degree of new infected nodes $\delta(d(t))$ versus time steps. (d) Variability of $\delta(d(t))$.

$$\delta(d(t)) = d_{\mathrm{SFcN}}(t) - d_{\mathrm{SFN}}(t), \tag{10}$$

$$\delta(\Delta[d(t)]) = \Delta[d_{\mathrm{SFcN}}(t)] - \Delta[d_{\mathrm{SFN}}(t)]. \tag{11}$$

As can be predicted, prevalence effects of an SFcN are gradually closer to that of an SFN when the strength of community structure becomes weaker and weaker. Except for the case with strong community structure (for the SFcN) we qualitatively analysed just now, an interesting fact shown in figure 6(a) is worth noticing. The dark red region, which represents values of $\delta(i(t))$ slightly larger than zero, means that virus prevalence on the SFcN may be slightly heavier than on the SFN at some stages of virus spreading. This fact might be attributed to the effect of high density of intra-community links, which can aggravate virus spreading in the network at some stages of epidemic spreading, although this effect is slight. This minor point needs to be noticed when we take specific measures to ward off heavy virus spreading in the network. Similarly, from figures 6(b)–(d), we find that as the community strength of an SFcN weakens, $\Delta[i(t)]$, $d(t)$, $\Delta[d(t)]$ behave less differently for an SFcN and its randomized counterpart SFN.

The above experiments are based on the supposition that at the beginning of an outbreak there exists only one infected seed in the network. We may ask whether the number and the distribution of initial infect seeds in the network can affect the differences

J. Stat. Mech. (2007) P01014

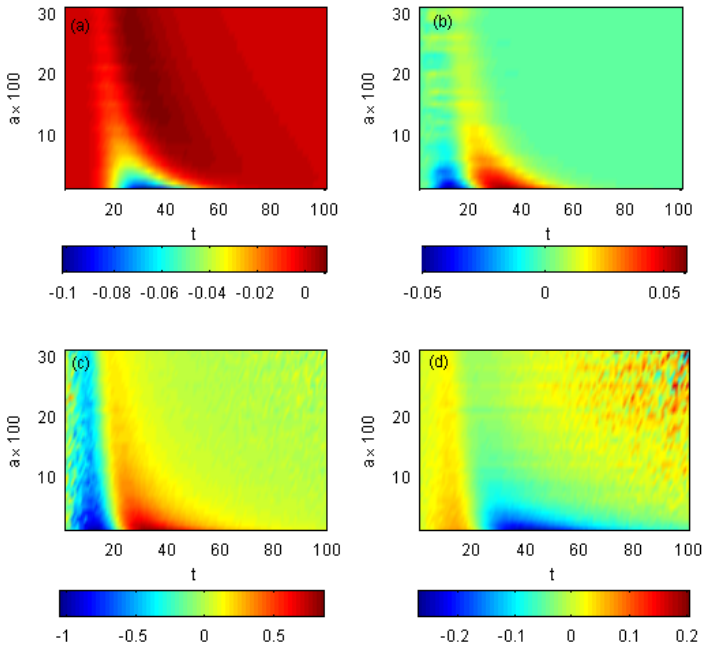

Figure 7. Plots for prevalence effects in SFcNs and SFNs with the number of nodes $N = 2000$, the infection rate $\lambda = 0.03$ and the control parameter α ranging from 0 to 0.3. For the SFcN, initially there is only one infected seed (randomly selected) in each community. The same nodes are infected initially in the SFN. (a) Difference of fractions of infected individuals $\delta(i(t))$ versus time steps. (b) Variability of $\delta(i(t))$. (c) Difference of average degree of new infected nodes $\delta(d(t))$ versus time steps. (d) Variability of $\delta(d(t))$.

of epidemic prevalence between SFcNs and SFNs. Some people might guess that if each community in a network is randomly given an infected seed, virus prevalence is much stronger in the SFcN (with strong community structure) than in the corresponding SFN with the same degree distribution. They argue that each community in the network possesses an infected seed and that groups of nodes in a single community have higher link density, thus provoking epidemic prevalence in the complete network. However, our simulation results revealed that it is not the case, as can be seen from the systematic plots in figure 7. The parameters are the same as those in figure 6. We find that the prevalence phenomena are much the same as in the case of only one infected seed existing in the network in the beginning. This fact further assures people that networks with community structure can weaken the prevalence of diseases, even though each community has infected individuals before the outbreak of diseases.

Another important parameter which affects the infection time of a node is its distance to the seed L as measured by the number of hops of the shortest path [18]. The spatial component is not taken into account here for both network kinds, and the distance between two nodes is given by the smallest number of hops d to go from one node to another. In figure 8(a), we show the relationship between the average infection time $\langle t_{\rm inf} \rangle$ (i.e. the time a node is infected) and L for both scale-free network kinds. Each group of points (the lines are only guides for the eyes) are averaged over 1000 realizations in one network

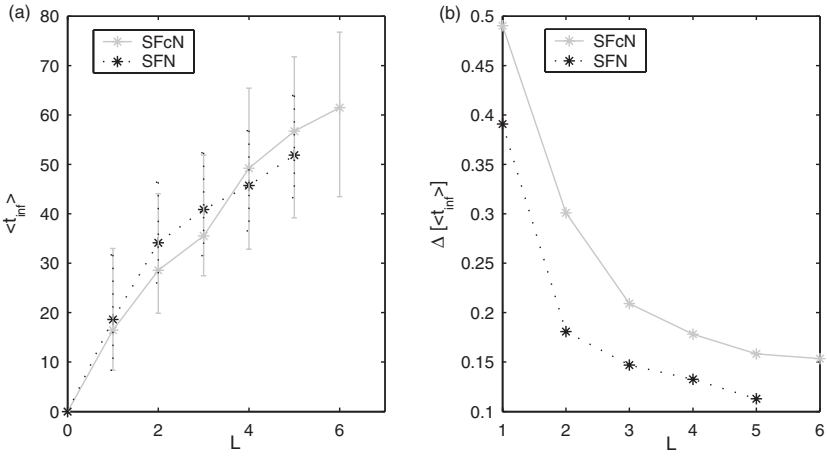

Figure 8. (a) Average infection time $\langle t_{\rm inf}\rangle$ and (b) variability of average infection time $\Delta[\langle t_{\rm inf}\rangle]$ as a function of distance L from the initial infected seed ($N = 1000$, $\alpha = 0.01$, averaged over 1000 outbreaks which start at exactly the same seed of degree $k_0 = 3$). The infection rate λ is set to be 0.03.

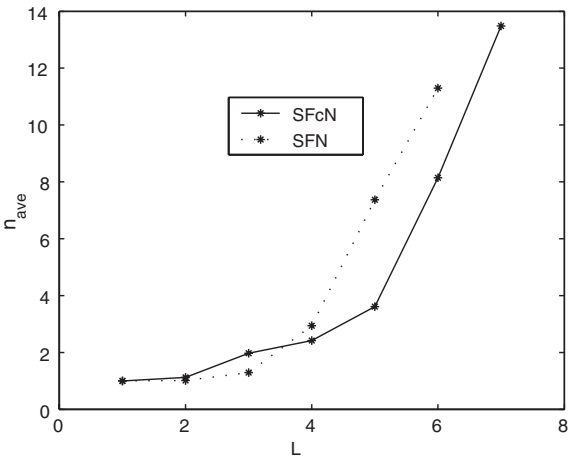

Figure 9. Average number of shortest paths between a randomly selected node with degree $k_0 = 3$ and a node at distance L. ($N = 1000$, $\alpha = 0.01$, averaged over 1000 random selections of an initial node with degree $k_0 = 3$.)

and each realization starts from a single infected seed with degree $k_0 = 3$. Both curves increase monotonically with the distance L, which means that nodes closer to the infected seed are infected first. It can also be observed on this panel that the infection time, $\langle t_{\rm inf}\rangle$, is larger for the SFN than for the SFcN for nodes at small distances from initial infected seed. Contrarily, the infection time is larger for the SFcN for nodes at larger distances. The reason can be attributed to the difference in the numbers of shortest paths in these networks, as was pointed out by [15]. More paths to go from one node to another in a small number of hops can indeed enhance virus spreading in networks. The average number of shortest paths $n_{\rm ave}$ between a randomly selected node and a node with distance L is enumerated in figure 9. Results are computed over 1000 random selections

J. Stat. Mech. (2007) P01014

of an initial node with degree $k_0 = 3$ on networks with $N = 1000$ nodes in order to get an accurate picture. The figure exhibits that for nodes at small distances (such as $d = 2$ and 3) from the initial infected node, n_{ave} are larger for the SFcN than for the SFN, while for nodes at greater distances it is the reverse case, which confirms the fact observed in figure 8(a). This result indicates that healthy individuals close to infected ones should be kept much closer watch on if the whole network exhibits community structure since in the case with communities they are more inclined to be infected, as compared to the case without communities.

The relationships between variability of infection time $\Delta[\langle t_{\text{inf}}\rangle]$ and the distance L from the initial infected seed are reported in figure 8(b) for both scale-free network kinds. The panel shows that the nodes in both networks show high values of $\Delta[\langle t_{\text{inf}}\rangle]$ when they are closer to the initial infected seed. We can also observe that the simulation points corresponding to spreading on an SFcN lie above the ones corresponding to spreading on an SFN, especially when $d = 1$ or 2.

5. Conclusion

In this paper, we have qualitatively studied the effects of community structure on epidemic spreading in scale-free networks, in which a parameter α controls the influence on the strength of community structure in the network and therefore on the epidemic dynamics. By comparing epidemic dynamics between scale-free networks with and without community structure, we concluded that networks with strong community structure are helpful for reducing the danger brought by epidemic prevalence in the network. The main results can be summarized as follows.

(i) We found that when the network exhibits strong community structure, i.e. with many intra-community links and few inter-community links, the epidemic prevalence is lighter, even though the initial infected seeds distribute in different communities far and wide.

(ii) Strong community structure induces a lower peak value of variability of the epidemic prevalence scale and therefore improves the predictability of the prevalence scale in the network.

(iii) Strong community structure weakens the *large-degree effect*, which can lead to a drastic danger to networks. This alleviates the frightening cases that more large-degree nodes are infected at the early stages of an outbreak, which could easily result in a large-scale epidemic spreading in the network.

Besides, the topological distance to the initial infected seed is also an important parameter in the epidemic spreading pattern. Nodes at short distances from the initial infected seed are infected at the early stage and have large variabilities of infection time. When networks exhibit strong community structure, it is worth special notice that nodes with small distances from initial infected seeds have shorter infection times in the network with strong community structure, which might be because of the discrepancy in the number of shortest paths between scale-free networks with and without communities. This point definitely needs to be considered when facing networks with strong community structure.

doi:10.1088/1742-5468/2007/01/P01014

J. Stat. Mech. (2007) P01014

This research may be also helpful in studying some other dynamic processes, such as information exchange and rumour propagation, in complex networks with communities.

Acknowledgments

This work was supported by the National Natural Science Foundation of China (NSFC) under Grants 60502009, the Fok Ying Tung Education Foundation under Grant 101064, the Program for New Century Excellent Talents in University, and the Youth Science and Technology Foundation of UESTC under grant L08010201JX04011.

References

[1] Albert R and Barabasi A-L, 2002 *Rev. Mod. Phys.* **74** 47
Dorogovtsev S N and Mendes J F F, 2002 *Adv. Phys.* **51** 1079
Newman M E J, 2003 *SIAM Rev.* **45** 167
[2] Barabasi A-L and Albert R, 1999 *Science* **286** 509
[3] Faloutsos M, Faloutsos P and Faloutsos C, 1999 *ACM SIGCOMM 99, Comput. Commun. Rev.* **29** 251
[4] Albert R, Jeong H and Barabasi A-L, 1999 *Nature* **401** 130
[5] Pastor-Satorras R and Vespignani A, 2001 *Phys. Rev. Lett.* **86** 3200
Pastor-Satorras R and Vespignani A, 2001 *Phys. Rev.* E **63** 066117
Pastor-Satorras R and Vespignani A, 2002 *Phys. Rev.* E **65** 035108
Barthelemy M, Barrat A, Pastor-Satorras R and Vespignani A, 2004 *Phys. Rev. Lett.* **92** 178701
[6] Liu Z, Lai Y and Ye N, 2003 *Phys. Rev.* E **67** 031911
Zanette D H, 2001 *Phys. Rev.* E **64** 050901(R)
Zanette D H, 2002 *Phys. Rev.* E **65** 041908
[7] Arenas A, Diaz-Guilera A and Guimera R, 2001 *Phys. Rev. Lett.* **86** 3196
Liebovitch L S and Schwartz I B, 2003 *Phys. Rev.* E **68** 017101
[8] Barthelemy M, Barrat A, Pastor-Satorras R and Vespignani A, 2005 *J. Theor. Biol.* **235** 275
[9] Li C and Maini P K, 2005 *J. Phys. A: Math. Gen.* **38** 9741
[10] Girvan M and Newman M E J, 2002 *Proc. Nat. Acad. Sci.* **99** 7821
Newman M E J, 2006 *Proc. Nat. Acad. Sci.* **103** 8577
Gronlund A and Holme P, 2004 *Phys. Rev.* E **70** 036108
Noh J D, Jeong H-C, Ahn Y-Y and Jeong H, 2004 *Preprint* cond-mat/0412149
Boguna M, Pastor-Satorras R, Diaz-Guilera A and Arenas A, 2004 *Phys. Rev.* E **70** 056122
Kimura M, Saito K and Ueda N, 2004 *Neural Networks* **17** 975
Li C and Chen G, 2006 *Physica* A **370** 869
[11] Yan G, Fu Z, Ren J and Wang W, 2006 *Preprint* physics/0602137
[12] Liu Z and Hu B, 2005 *Europhys. Lett.* **72** 315
[13] Newman M E, 2002 *Phys. Rev. Lett.* **89** 208701
Milo R, Kashtan N, Itzkovitz S, Newman M E J and Alon U, 2003 *Preprint* cond-mat/0312028
[14] Newman M E J and Girvan M, 2004 *Phys. Rev.* E **69** 026113
[15] Crépey P, Alvarez F P and Barthélemy M, 2006 *Phys. Rev.* E **73** 046131
[16] Colizza V, Barrat A, Barthélemy M and Vespignani A, 2006 *Proc. Nat. Acad. Sci.* **103** 2015
[17] Cohen R, Havlin S and ben Avraham D, 2003 *Phys. Rev. Lett.* **91** 247901
Holme P, 2004 *Europhys. Lett.* **68** 908
[18] Loecher M and Kadtke J, 2005 unpublished

J. Stat. Mech. (2007) P01014