Dynamic spreading behavior of homogeneous and heterogeneous networks*

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Abstract The detailed investigation of the dynamic epidemic spreading on homogeneous and heterogeneous networks was carried out. After the analysis of the basic epidemic models, the susceptible-infected-susceptible (SIS) model on homogenous and heterogeneous networks is established, and the dynamical evolution of the density of the infected individuals in these two different kinds of networks is analyzed theoretically. It indicates that heterogeneous networks are easier to propagate for the epidemics and the leading spreading behavior is dictated by the exponential increasing in the initial outbreaks. Large-scale simulations display that the infection is much faster on heterogeneous networks than that on homogeneous ones. It means that the network topology can have a significant effect on the epidemics taking place on complex networks. Some containment strategies of epidemic outbreaks are presented according to the theoretical analyses and numerical simulations.

Keywords: complex networks, dynamic epidemic spreading, SIS model, homogeneous networks, heterogeneous networks, containment strategies.

The accurate modeling of the epidemics is the first step to understand the impact of the infectious diseases and develop the effective control strategies. Mathematical analysis and dynamics[1,2] are often used to model the disease propagation. Kermack and McKendrick firstly published the relevant results and proposed the famous susceptible-infected-removed (SIR)[3] model in 1926, and then susceptible-infected-susceptible (SIS) model was presented in 1932[4]. Here individuals always lie in several discrete states, such as susceptible (S), infective (I) and removed (R) etc. At the same time every one has the equal probability to contact with others, i.e. homogeneous mixing hypothesis. However, many dynamic characteristics[1,2] are not considered in the classical SIR/SIS models, such as individual age, social structure, migration and geographical patch structure. In order to increase the accuracy and relevance of epidemic models, we must consider these factors and the connectivity pattern of the network of contacts among individuals along which the disease can be transmitted. They may be the critical features in the epidemic spreading[5]. Recently many studies have observed that the heterogeneity of the contact network can induce noticeable effects on the spreading behavior and the deployment of immunization resources.

Many social, biological and technological systems can be properly studied by the theory of complex networks[6—10]. The small-world[11] effect and scale-free (SF)[12] property are recently found to be typical characteristics in these real-world networks. Especially for SF networks, its degree distribution is characterized by the power-law behavior $P(k) \sim k^{-\alpha-2}$, with an exponent $0<\alpha\leqslant 1$. SF property means that a large number of vertices have a few links, but statistically exists a few vertices with very large degree, i.e. the so-called "hubs" or "super-spreaders". Pastor- Satorras et al.[13,14] observed that whatever the spreading rate in SF networks the infection would be widespread, eventually leading to the absence of any epidemic threshold below which the infection cannot initiate a major outbreak. This new scenario may radically change a lot of drawn results and stimulate many relevant researches[15—20] because the epidemic threshold is fundamental to the classical epidemiology. In addition it is of importance to the sexual contact network[9] and Internet[10] since they appear to

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be scale free. It also implies that sexually transmitted diseases and computer virus can be more easily disseminated, and it requires us to investigate thoroughly their spreading dynamics. To control the epidemic outbreaks, it is necessary for us to study the dynamical evolution of epidemic spreading. Barthelemey et al.[21,22] studied the dynamical properties of susceptible-infective (SI) model taking place on complex networks and found that the growth of infected individuals is governed by an exponential growth with a timescale τ proportional to the ratio between the first and second moment of the network's degree distribution, i.e. $\tau \sim \langle k \rangle / \langle k^2 \rangle$. In this study we extended this result and focused on the dynamical spreading behavior of SIS epidemic model on complex networks and found out that Barthelemey's result is only a special case of our conclusions.

In the following sections we will describe a detailed investigation of epidemic spreading on homogeneous and heterogeneous complex networks. At first we will introduce the basic theory of epidemic infection and the traditional threshold theory, successively the dynamic evolution of SIS epidemic spreading on homogeneous and heterogeneous networks studied by the analytical method and computer simulation. At last we will present some containment strategies to control the fast epidemic spreading on complex networks and give our conclusions.

1 Basic theory of SIR/SIS epidemic spreading

1.1 SIR model

In SIR[3] model the whole population is divided into three classes: susceptible, infective and removed. The susceptible ones do not have the disease but can catch it if they keep in contact with the infective ones. The infective ones have caught the disease and can pass it on. The removed ones have recovered from the disease and acquired permanent immunity so that they can never get it or pass it on. The fractions s, i and r of individuals in three states S, I and R are governed by the following differential equations:

$$\frac{ds}{dt} = -\beta is, \quad \frac{di}{dt} = \beta is - \gamma i, \quad \frac{dr}{dt} = \gamma i, \tag{1}$$

where any susceptible individual has a uniform probability β per unit time to catch the disease, infective individuals recover and become immune at a fixed rate γ. In SIR model, we suppose that the total population keeps constant, namely, s, i and r must satisfy the normalization condition $s + i + r = 1$, so we only consider the first two equations. We can define the ratio of $\frac{s(0)}{\rho}$ as the basic reproductive number R_0, i.e. $R_0 = \frac{s(0)}{\rho}$, where $\rho = \frac{\gamma}{\beta}$ and $s(0)$ is the initial density of susceptible nodes. R_0 means that an infected one will infect the average number of susceptible ones during the initial stage. When $R_0 > 1$ the disease will be epidemic, $R_0 < 1$ the disease will not spread in the wide range, so $R_0 = 1$ is the critical threshold of the SIR model. It is shown in Fig. 1.

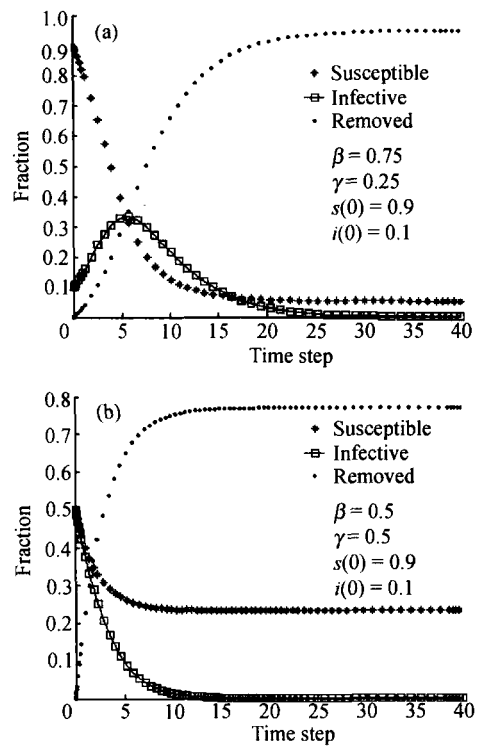

Fig. 1. Evolution of densities of susceptible, infective and removed ones in SIR model with different R_0. (a) $R_0 = 2.7 > 1$ the disease is epidemic; (b) $R_0 = 0.9 < 1$ the disease is not epidemic.

1.2 SIS model

However not all diseases can confer the immunity on their survivors. Some diseases can be cured by medicine, or unluckily be infected again by a patient. Tuberculosis and gonorrhea are two much-studied examples[2]. Computer viruses can also fall into this category and they can be cured by anti-virus software, but so far the computer do not have the permanent virus-checking program to avoid the subsequent attacks from the same kind of virus. Such diseases can

be described by SIS[4] model. SIS[4] model assumes that the individual can exist only in two discrete states: susceptible and infective. Its dynamics can be represented by the following differential systems:

$$\frac{ds}{dt} = -\beta is + \gamma i, \quad \frac{di}{dt} = \beta is - \gamma i. \qquad (2)$$

It is similar to SIR model, s and i can be normalized, i.e. $s + i = 1$. Correspondingly, we can define the basic reproductive number R_0 of SIS model, $R_0 = \frac{1}{\rho} = \frac{\beta}{\gamma}$, where $\rho = \frac{\gamma}{\beta}$. When $R_0 > 1$ the disease is epidemic and leads to the endemic state; when $R_0 < 1$ the epidemic will disappear in the end, so $R_0 = 1$ is also the threshold of the SIS model. It is demonstrated in Fig.2.

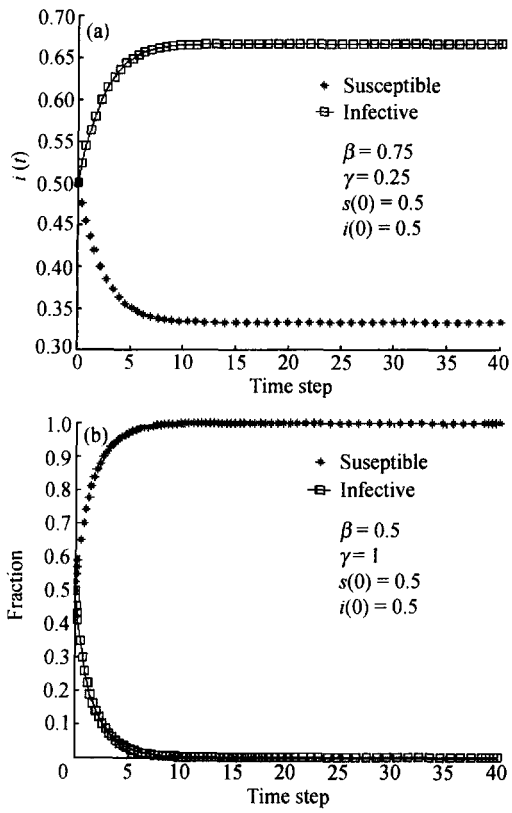

Fig. 2. Evolution of densities of susceptible, infective ones in SIS model with different R_0. (a) $R_0 = 3 > 1$ the disease is epidemic; (b) $R_0 = 0.5 < 1$ the disease is not epidemic.

2 Epidemic spreading on complex networks

The classic epidemic models display the typical critical phenomenon and there exists a nontrivial epidemic threshold below which the infection will die out in the end, and above which the infection will be epidemic. But these models do not take into account the effect of the topology of the contact network on the epidemic spreading. Recently the rapid advances in complex network theory revamp again the study of the epidemic spreading law[13—20] and it seems to be some remarkable results in this field. For homogeneous networks including Erdos and Renyi[23] (ER) random network and Watts and Strogatz[11] (WS) small-world network, there still exists a critical threshold. But it seems that the epidemic threshold[13,14] is absent for the scale-free network created by Barabasi and Albert (BA)[12] algorithm. This striking result for BA network may change radically many known epidemic conclusions.

The smaller threshold, even the absence of epidemic threshold, contributes to a general understanding of epidemic spreading on heterogeneous networks, but this does not provide a large amount of information about the time pattern of the dynamical processes and how effectively the spreading occurs. It also raises some new questions on how to protect the network and find the optimal strategies for the deployment of immunization resources. So it is necessary for us to investigate the temporal evolution of epidemic outbreaks on complex networks. In this section we will focus on the dynamic properties of SIS model on complex networks and find that the epidemic evolution on SI model[21,22] is a special case of the dynamic evolution of our SIS model.

2.1 Dynamic evolution of SIS model on homogeneous networks

ER random graphs and WS small world networks have highly peaked degree distribution and most nodes have approximately the same degree. So the homogeneous mixing hypothesis[1,2] holds for these two kinds of networks and we can take the mean-field analysis for SIS model on such these networks.

Similarly $s(t)$ and $i(t)$ are the densities of susceptible and infective individuals respectively at time t, and they must always satisfy the normal condition: $s(t) + i(t) = 1$. So the differential equation (2) is simplied as

$$\frac{di(t)}{dt} = \beta\langle k\rangle i(t)[1 - i(t)] - \gamma i(t). \qquad (3)$$

The creation item on the right side of Eq. (3) which is proportional to the spreading rate β and the average degree $\langle k\rangle$, the density of susceptible individuals $[1 - i(t)]$ and the density of infected individuals $i(t)$, denotes the growth rate of infected individuals.

The decaying item, which is proportional to the curing rate γ and the density of infected individuals $i(t)$, represents the decreasing rate of infected ones.

We can define $\lambda = \beta/\gamma$ to be the effective spreading rate and $\lambda_C = \frac{1}{\langle k\rangle}$ to be the spreading threshold of homogeneous networks[13,14]. Here we discuss the solution of Eq. (3) under the super-critical ($\lambda > \lambda_C$) and sub-critical ($\lambda < \lambda_C$) case respectively.

(i) $\lambda > \lambda_C$. The solution of Eq. (3) is easily conquered at this time, denoted as

$$i(t) = \frac{i_0 \exp(t/\tau_H)}{1 + i_0 \frac{\beta\langle k\rangle}{\beta\langle k\rangle - \gamma}[\exp(t/\tau_H) - 1]}, \quad (4)$$

where $\tau_H = \frac{1}{\beta\langle k\rangle - \gamma}$ is the time-scale of the infection growth and i_0 is the initial density of infected individuals. In the initial phase the leading behavior is dominated by $i(t) \approx i_0 \exp(t/\tau_H)$, i.e. an exponential growth. Especially, if the infected individuals cannot be cured during the initial stage of the epidemics, we can set $\gamma = 0$, and it will reduce to SI model. Thus $\tau_H' = \frac{1}{\beta\langle k\rangle}$ and $i(t) = \frac{i_0 \exp(t/\tau_H')}{1 + i_0[\exp(t/\tau_H') - 1]}$ are totally consistent with the result of SI model on homogeneous networks in Refs. [21,22].

(ii) $\lambda < \lambda_c$. We can solve Eq. (3) and obtain

$$\begin{aligned} i(t) &= i_0 \exp[(\beta\langle k\rangle - \gamma)t] \\ &= i_0 \exp(-t/\tau_H), \end{aligned} \quad (5)$$

where the time-scale $\tau_H = \frac{\lambda\lambda_c}{\beta(\lambda_c - \lambda)}$.

So we can see that the infection will increase with time and lead to an endemic state when $\lambda > \lambda_C$, while the infection will exponentially die out if $\lambda < \lambda_C$. And this result is shown in Fig. 3.

Apparently the evolution of infected nodes is dominated by the initial infection density i_0 and time-scale τ_H of the exponential behavior. In order to control the epidemic outbreak, we should take measures to decrease i_0 and increase τ_H. We usually quarantine the infected nodes to decrease i_0 and the average number of contacts $\langle k\rangle$ to prevent the spread of the contagious disease. It is also easy to understand why we try to improve the health-cares to increase the cure rate γ and decrease the infection rate β to increase τ_H for controlling the epidemic outbreak.

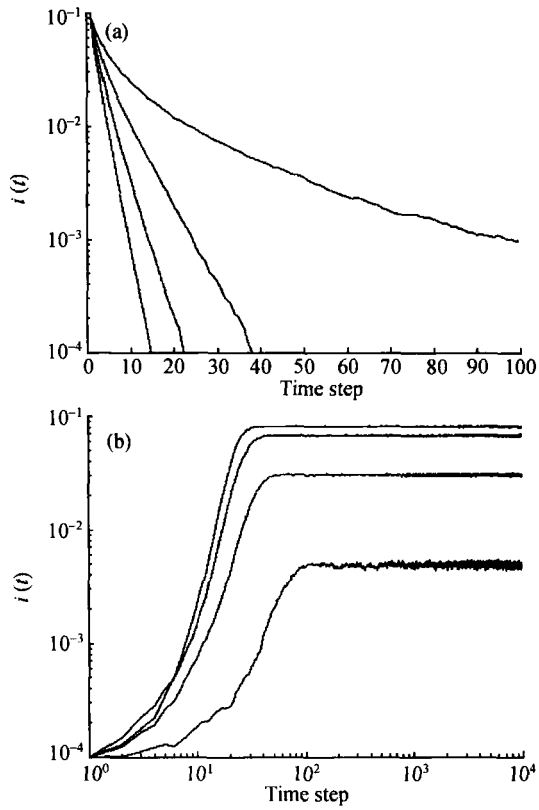

Fig. 3. Time evolution of SIS epidemic spreading over WS network. (a) $\lambda < \lambda_C$. From top to bottom $\lambda = 0.16, 0.14, 0.12, 0.1$ respectively; (b) $\lambda > \lambda_C$. From top to bottom $\lambda = 0.24, 0.22, 0.2, 0.18$ respectively.

2.2 Dynamic evolution of SIS model on heterogeneous networks

The above analyses and discussions are valid for homogeneous networks. However many real-world networks[6–8] are highly heterogeneous and the degree displays the power-law distribution, in which the degree of vertices is fluctuating greatly and the average degree is not any more a meaningful characterization of the network properties. In order to take this into account, it is necessary for us to write down the infection dynamics equation for various $i_k(t)$. In SIS model the equation reads as

$$\frac{di_k(t)}{dt} = \beta k[1 - i_k(t)]\Theta_k(t) - \gamma i_k(t), \quad (6)$$

where the creation term on the right side of Eq. (6) is proportional to the spreading rate β, degree k, the probability $[1 - i_k(t)]$ that a vertex with degree k is not infected and the probability $\Theta_k(t)$ that the neighbor of any node with degree k is infected; the decaying item is proportional to the cured rate γ and the density of infected individuals $i_k(t)$.

For uncorrelated networks, the probability that an edge departing from a vertex of degree k arrives at a vertex of degree k' is independent of the degree k of the initial vertex. In this situation, the probability that each edge of a susceptible one pointing to an infected vertex of degree k' is proportional to the fraction of edges emanated from those infected vertexes. Moreover each infected vertex can either be one of the initial seeds (infected at $t = 0$) or be infected at $t > 0$. In the latter case, at least one of the edges of the infected vertex is pointing to another infected one, from which the infection has been transmitted. The term Θ_k is equal for various degree k and therefore is equal to the sum of these two terms, thus we can calculate

$$\Theta(t) = \Theta_k(t) = \frac{\sum_{k'} k'P(k')i_{k'}(0)}{\langle k\rangle} + \frac{\sum_{k'} (k'-1)P(k')[i_{k'}(t) - i_{k'}(0)]}{\langle k\rangle}, \tag{7}$$

where $\langle k\rangle$ denotes the average degree and becomes the proper normalization factor dictated by the total number of edges.

Differentiating Eq. (7) the following equation is acquired:

$$\frac{\mathrm{d}}{\mathrm{d}t}\Theta(t) = \frac{\sum_{k'} (k'-1)P(k')\frac{\mathrm{d}}{\mathrm{d}t}i_{k'}(t)}{\langle k\rangle}. \tag{8}$$

Neglecting terms of order $O(i^2)$ of Eq. (6) and simplifying the result, we obtain

$$\frac{\mathrm{d}i_k(t)}{\mathrm{d}t} \approx (\beta k - \gamma)\Theta(t). \tag{9}$$

Then inserting Eq. (9) into Eq. (8) and we get

$$\frac{\mathrm{d}\Theta(t)}{\mathrm{d}t} \approx \frac{\sum_{k'} (k'-1)P(k')(\beta k' - \gamma)\Theta(t)}{\langle k\rangle} = \frac{\beta\langle k^2\rangle - (\beta+\gamma)\langle k\rangle + \gamma}{\langle k\rangle}\Theta(t). \tag{10}$$

Combining Eq. (9) with Eq. (10), and then conquering them in the case of the uniform initial condition $i_k(t=0) = i_0$, we acquire the analytical solutions of Eq. (6)

$$i_k(t) \approx i_0\left[\frac{(\beta k - \gamma)\langle k\rangle}{\beta\langle k^2\rangle - (\beta+\gamma)\langle k\rangle + \gamma}(e^{\frac{t}{\tau_H}} - 1) + 1\right]. \tag{11}$$

Thus the total average infection density is

$$i(t) = \sum_k P(k)i_k(t) \approx i_0\left[\frac{\beta\langle k\rangle^2 - \gamma\langle k\rangle}{\beta\langle k^2\rangle - (\beta+\gamma)\langle k\rangle + \gamma}(e^{\frac{t}{\tau_H}} - 1) + 1\right], \tag{12}$$

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

Obviously the time-scale of an epidemic outbreak is related to the second moment of the degree distribution. In the networks with a very heterogeneous connectivity pattern, $\langle k^2\rangle$ is very large and τ_H is very small, so it indicates a very fast spread of the infection. For scale-free networks with the degree exponent between 2 and 3, we have $\langle k^2\rangle \to \infty$ with the network size $N \to \infty$, thus $\tau_H \to 0$. Therefore in uncorrelated scale-free networks we face a virtually instantaneous rise of the epidemic incidence.

Remarks:

(i) In homogeneous networks, such as an ER random graph with Poisson degree distribution, where $\langle k^2\rangle = \langle k\rangle^2 + \langle k\rangle$, we can recover the result

$$\tau_H = \frac{\langle k\rangle}{\beta\langle k\rangle^2 - \gamma\langle k\rangle + \gamma} = \frac{1}{\beta\langle k\rangle - \gamma + \frac{\gamma}{\langle k\rangle}}. \tag{13}$$

For $\gamma < 1$ and $\langle k\rangle \gg 1$, thus

$$\tau_H \approx \frac{1}{\beta\langle k\rangle - \gamma}, \tag{14}$$

which coincidently agrees with the result in Section 2.1.

(ii) If the infected individuals cannot be cured during the initial stage of the epidemics, we can set $\gamma = 0$ and obtain

$$\tau_H = \frac{\langle k\rangle}{\beta\langle k^2\rangle - (\beta+\gamma)\langle k\rangle + \gamma} = \frac{\langle k\rangle}{\beta(\langle k^2\rangle - \langle k\rangle)}, \tag{15}$$

$$i(t) = i_0\left[\frac{\langle k\rangle^2}{\langle k^2\rangle - \langle k\rangle}(e^{\frac{t}{\tau_H}} - 1) + 1\right], \tag{16}$$

which are completely the same as the results of SI model on heterogeneous networks[21,22].

(iii) Again the evolution of infected nodes is also dominated by the initial infection density i_0 and time-scale τ_H in the very heterogeneous networks. In order to control epidemic outbreaks, we should try to decrease i_0 and increase τ_H. So it is necessary for us to

lower the initial infection density i_0, cut off the connection with other nodes and improve the cure probability to contain the epidemic proliferation.

3 Numerical simulation

We have carried out large-scale numerical simulations of SIS model on both homogeneous and heterogeneous complex networks to verify the theoretical predictions made in the previous section. The choice of the network models was dictated by the request of generating an uncorrelated network. We select WS[11] small world model as a representative of homogeneous networks, and the heterogeneous networks are generated with BA algorithm[12]. Simultanously we use an agent-based modeling strategy where at each time step the SIS dynamics is applied to each vertex by considering the actual state of the vertex and its neighbors.

Fig.3 shows the time evolution of SIS epidemic spreading over the WS network under the sub-critical phase ($\lambda < \lambda_c$) and super-critical phase ($\lambda > \lambda_c$) respectively. In the simulation the network size $N = 10000$, rewired probability $p = 1.0$ and the number of nearest neighbors $K = 6$. Obviously, in Fig.3 (a) the linear behavior in the semi-log scale is taken on when $\lambda < \lambda_c = \frac{1}{6}$ and the infection will die out quickly, while in Fig. 3 (b) the infection will increase into a steady state when $\lambda > \lambda_c$, i.e. an endemic state.

Fig.4 illustrates the time evolution of SIS epidemic spreading on BA network under the cure rate $\gamma = 0$, 0.0001, 0.001 and 0.01 respectively and constant infection rate $\beta = 0.001$. Fig. 5 shows the time evolution of SIS epidemic spreading on WS network under the same condition as Fig.4.

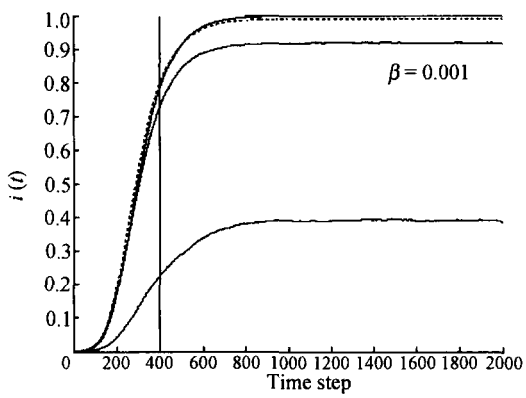

Fig. 4. Time evolution of SIS epidemic spreading on BA network. From top to bottom $\gamma = 0$, 0.0001, 0.001 and 0.01.

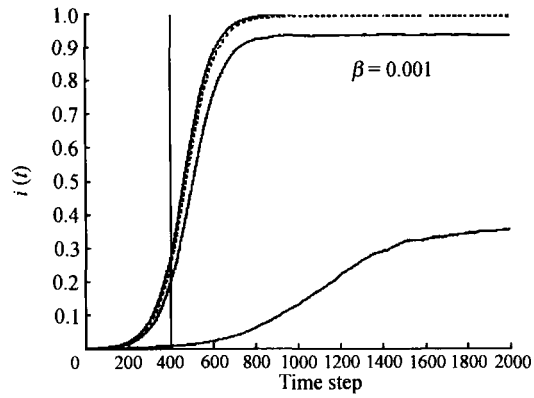

Fig. 5. Time evolution of SIS epidemic spreading on WS network. From top to bottom $\gamma = 0$, 0.0001, 0.001 and 0.01.

Fig. 6 plots the time evolution of SIS epidemic spreading on BA network under various infection rates $\beta = 0.0001$, 0.001, 0.01 and constant cure rate $\gamma = 0.001$. Fig. 7 shows the time evolution of SIS epidemic spreading on WS network under the same condition as Fig. 6. For WS network the spreading is always in the super-critical phase, that is to say, $\lambda > \lambda_c$.

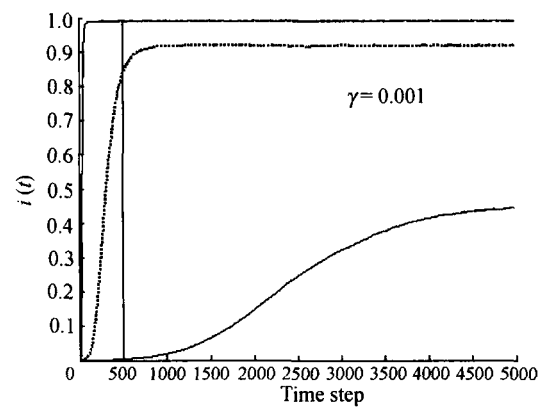

Fig. 6. Time evolution of SIS epidemic spreading on BA network. From top to bottom $\beta = 0.01$, 0.001 and 0.0001.

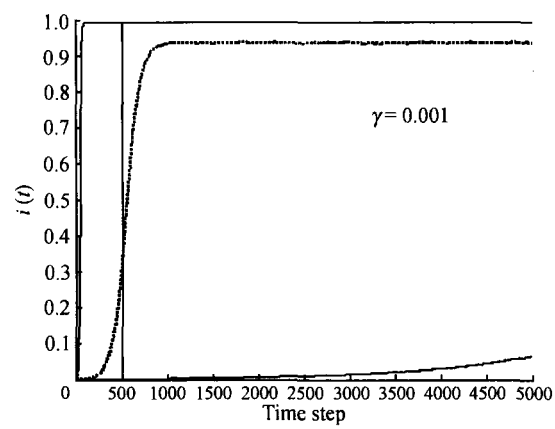

Fig. 7. Time evolution of SIS epidemic spreading on WS network. From top to bottom $\beta = 0.01$, 0.001 and 0.0001.

From Fig.4 to Fig.7 the running parameters are the network size $N = 10000$, the average degree $\langle k \rangle = 16$ for WS ($K = 8$) and BA ($m = 8$) network. We typically average our results for 10 network realizations and up to 20 SIS dynamics for each network realization. Obviously the infection prevalence is lowered with the increase of the cure rate under the same infection rate. On the contrary the higher the infection rate, the larger the prevalence will be. To control the disease propagation, we should improve the health-cares to enhance the cure rate and reduce the infection rate.

Fig.8 reports the effect of m on the spreading in BA model, and m is the number of edges added with every new node, which largely affects the first and second moments of BA network. Generally m increases the second moments more quickly than the first one. So the time-scale of evolution is decreased with m and the infection will be much faster. Similar results can be obtained in WS small-world network and is displayed in Fig.9. In the simulation the network size $N = 10000$, $\beta = 0.001$ and $\gamma = 0.001$. All these simulations agree with the theoretical analysis in Section 2.1 and Section 2.2. It also indicates that we should immediately quarantine the infected nodes to decrease the initial infective density and the number of connections with susceptible individuals (K or m) in the initial outbreaks, especially for the heterogeneous networks.

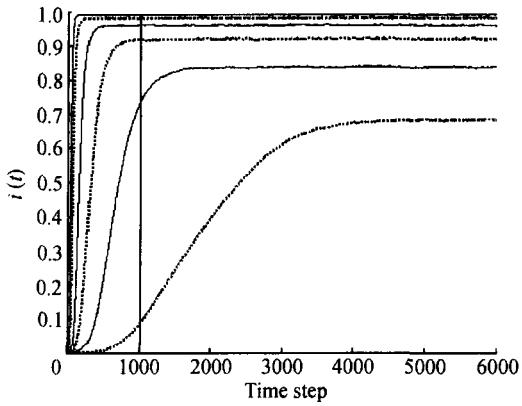

Fig. 8. Effect of m on the time evolution of SIS epidemic spreading in BA network. From bottom to top $m = 2, 4, 8, 16, 32, 64$.

In addition the density of infected individuals under the same condition is larger on BA networks than that on WS networks at some given time, which is easily seen in the initial stage at $t = 400$ in Figs.4 and 5, $t = 500$ in Figs.6 and 7 and $t = 1000$ in Figs. 8 and 9. That is to say, the disease or virus is easier to be propagated in heterogeneous networks, it will also lead to larger difficulty for us to control the epidemic spreading on such these networks at the beginning of the outbreak, such as computer virus and the sexually transmitted diseases etc. We should take immediate measures before these virus or diseases are epidemic.

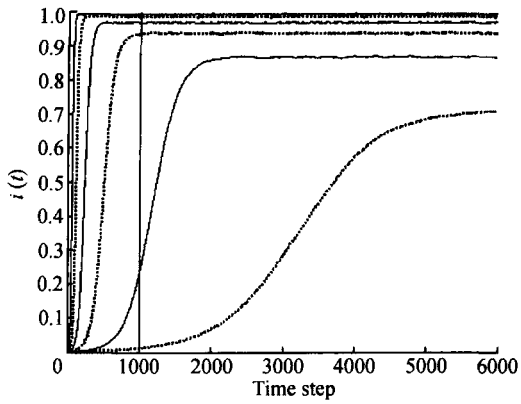

Fig. 9. Effect of K on the time evolution of SIS epidemic spreading in WS network. From bottom to top $K = 2, 4, 8, 16, 32, 64$.

4 Conclusions

In this study we investigated mainly the dynamic behavior of SIS epidemic spreading on complex networks. And the approximate results can be easily extended to SIR epidemic model. We found that the topology of the contact network has an obvious effect on the epidemic spreading behavior. We have focused on the temporal behavior of SIS model on complex networks and it is shown that the leading behavior is dictated by the exponential increasing in the supercritical condition. In addition the disease or virus is more easily infected and the epidemic outbreak is almost instantaneous in the heterogeneous networks. However in the homogeneous networks the disease or virus will disseminate relatively slowly. It is very important for the epidemic control and deployment of immunization resources in the heterogeneous networks. So we should immediately take effective containment strategies to prevent the spreading of diseases in the initial outbreaks.

References

1 Murray J. D. Mathematical Biology. Berlin: Springer Verlag, 1993.
2 Anderson R. M. and May R. M. Infectious Diseases in Humans. Oxford: Oxford University Press, 1992.
3 Kermack W. O. and McKendrick A. G. Contributions to the mathematical theory of epidemics. Proc. Roy. Soc., 1927, A115: 700—721.

4 Kermack W. O. and McKendrick A. G. Contributions to the mathematical theory of epidemics. Proc. Roy. Soc., 1932, A138: 55—83.
5 Eubank S., Guclu H. and Kumar A. et al. Modelling disease outbreaks in realistic urban social networks. Nature, 2004, 429: 180—184.
6 Albert R. and Barabási A. L. Statistical mechanics of complex networks. Rev. Mod. Phys., 2002, 74: 47—97.
7 Dorogovtse S. N. and Mendes J. F. F. Evolution of networks. Advances in Physics, 2002, 51: 1079—1187.
8 Newman M. E. J. The structure and function of complex networks. SIAM Review, 2003, 45: 167—256.
9 Liljeros F., Edling C. R. and Amaral L. A. N. et al. The web of human sexual contacts. Nature, 2001, 411: 907—908.
10 Faloutsos M., Faloutsos P. and Faloutsos C. On power-law relationships of the Internet topology. Comput. Commun. Rev., 1999, 29: 251—263.
11 Watts D. J. and Strogatz S. H. Collective dynamics of small world networks. Nature, 1998, 393: 440—442.
12 Barabási A. L. and Albert R. Emergence of scaling in random networks. Science, 1999, 286: 509—512.
13 Pastor-Satorras R. and Vespignani A. Epidemic spreading in scale-free networks. Phys. Rev. Lett., 2001, 86: 3200—3203.
14 Pastor-Satorras R. and Vespignani A. Epidemic dynamics and endemic states in complex networks. Phys. Rev. (E), 2001, 63: 066117.
15 Zhou T., Fu Z. Q. and Wang B. H. Epidemic dynamics on complex networks. Progress in Natural Science, 2006, 16(5): 452—457.
16 Newman M. E. J. Spread of epidemic disease on networks. Phys. Rev. E, 2002, 66: 016128.
17 Moreno Y., Pastor-Satorras R. and Vespignani A. Epidemic outbreaks in complex heterogeneous networks. Eur. Phys. J. (B), 2002, 26: 521—529.
18 Lloyd A. L. and May R. M. How viruses spread among computers and people. Science, 2001, 292: 1316—1317.
19 Dezso Z. and Barabasi A. L. Halting viruses in scale-free networks. Phys. Rev. (E), 2002, 65: 055103.
20 Li Y., Liu Y. and Shan X. M. Dynamic properties of epidemic spreading on finite complex networks. Chinese Physics, 2005, 14: 2153—2157.
21 Barthelemy M., Barrat A. and Pastor-Satorras R. et al. Velocity and hierarchical spread of epidemic outbreaks in scale-free networks. Phys. Rev. Lett., 2004, 92: 178701.
22 Barthelemy M., Barrat A. and Pastor-Satorras R. et al. Dynamical patterns of epidemic outbreaks in complex heterogeneous networks. J. Theor. Biol., 2005, 235: 275—288.
23 Erdös P. and Rényi A. On random graphs. Publ. Math., 1959, 6: 290—297.