

Traffic Driven Epidemic Spreading in Weighted Homogeneous Networks

Zinan Chang^{1,2*} and Fei Shao^{1,2}

¹ Jiangsu Information Analysis Engineering Laboratory, Nanjing 211169, China

² School of Computer Engineering, Jinling Institute of Technology, Nanjing 211169, China

*changzn@jit.edu.cn

Abstract

To investigate the impact of weights on the traffic driven epidemic spreading in weighted homogeneous networks, an epidemic model in pseudo-random network with adjustable weights is presented. In the scenario that epidemic pathway is defined and driven by traffic flows, the epidemic spreading velocity of SI model in weighted homogeneous networks is obviously accelerated when the edge weight is different. And in traffic driven SIS model, the epidemic threshold is found to be proportional to the inverse of the average node betweenness. It is better to control the epidemic spreading when the edges have the same weight. Simulation results have confirmed the theoretical predictions.

Keywords: Traffic driven, epidemic spreading, weighted networks, SI model, SIS model

1. Introduction

Recently, a number of studies [1-3] have been conducted to explore the topological characteristics of the real-world networks. The real-world networks are proved to be classified into two distinct groups according to their structure properties. The first one is homogeneous complex networks, a typical example is the random graph model [1] where each node has roughly the same number of links. Recent studies indicate that many social, biological, and communication systems have a scale-free topological characteristic [3] and exhibit a power-law degree distribution. In these networks, the probability that a node has k connections follows the distribution $P(k) \sim k^{-\gamma}$ while the degrees of all the nodes in homogeneous networks are almost the same.

These studies on the topological characteristics shed light on the exploration of the dynamic process taking place on the network such as epidemic spreading. Some models have been proposed to control the epidemic spreading on the real-world networks where the nodes are classified in three states: susceptible (which will not infect others but may be infected), infected (which is infective) and recovered (which has recovered and will not be infected). The SI [4-6], SIS [7-9], and SIR [10-12] models are proposed based on the discrete states of the nodes. These models always assume that a susceptible node will be infected by an infected one during one time step with probability ν , and at the same time the infected node will be recovered with rate δ . Then the effective spreading rate λ is defined as $\lambda = \nu/\delta$. Without lack of generality, δ is often set to 1. In these previous models which assume that each node's infectivity is strictly equal to its degree, each infected node will contact every neighbor once within one time step. However, lots of networks are found that the nodes only interact with some of their neighbors at intervals. For instance, Internet connection at a given time depends on the specific traffic and routing protocols. And in many real world networks, the epidemic spreading will not occur unless there is at least one packet interaction in the network that can physically transfer the epidemic from one node to another. The epidemic spreading rate mainly depends on the

traffic flow. A novel approach called traffic driven epidemic spreading is introduced to investigate the epidemic spreading process [13-16].

The previous studies on networks have been principally focused on the unweighted network where edges between nodes are either present or not. However, lots of real world systems such as the scientific collaboration networks [17], the world-wide airport network [18], and the Internet [19] have proved to be specified not only by the topology but also by the edge weight. How the traffic-driven epidemic spreads in weighted homogeneous networks has not been considered. In this paper, we proposed a model of pseudo-random network with adjustable within-community edge weight. By changing the within-community edge weight in the same community, we investigate the epidemic spreading in weighted homogeneous networks with SI model and SIS model.

This paper is organized as follows. In Section 2 we describe the traffic driven epidemics spreading models, followed by the experimental simulations in Section 3. The conclusions are given in Section 4.

2. Models

We also employ the pseudo-random network model [20] to investigate the epidemic spreading since the average node degree is same as the fully random network [1]. These networks are comprised of n nodes which are divided into $mods$ communities with $n/mods$ nodes. Each node has average Z_{in} within community edges and Z_{out} between community edges. While Z_{in} is varied, the value of Z_{out} is chosen to keep the total average degree fixed which is set as $\langle k \rangle$. And we assign different weights to the different kinds of edges: between-community edges are given a fixed weight of w_{out} while within-community edges are given the weight w_{in} . We define $w = w_{in}/w_{out}$ to check the impact of weight on the traffic driven epidemic spreading.

The classical SIS model [7-9] does not take into account the possibility of nodes removal due to death or acquired immunization, and thus nodes run stochastically through the cycle susceptible \rightarrow infected \rightarrow susceptible. While investigating the dynamical behaviors in the very early stage of epidemic outbreaks, the SI [4-6] model is used where infected nodes remain always infective and spread the infection to susceptible neighbors. The SI model can be viewed as the limit of the SIS model when the recovering rate is 0.

The traffic-driven epidemic spreading model can be described as below:

- 1) All the nodes can create packets with addresses of destination, receive packets from other nodes, and route packets to their destinations;
- 2) At each time step, an information packet is generated at every node with probability β , with randomly chosen sources and destinations; and all the packets are forwarded one step toward their destinations through the weighted shortest path [17, 21] at the same time (Each node has unbounded packet delivery capability for simplicity);
- 3) A packet, upon reaching its destination, is removed from the system;
- 4) A susceptible node has a probability λ of becoming infected every time it receives a packet from an infected neighbor. (In SIS model, the probability λ is the effective spreading rate while the recovering rate is fixed to 1.)

Since weights in most weighted networks are not the cost of them but the operationalizations of tie strength, the edge weight need to be reversed before directly applying Dijkstra's algorithm [22] to identify the weighted shortest paths in these networks. The distance between two nodes is just the inverse of the absolute weight of edge linked the two nodes. Moreover, we can also take advantage of the proposed weighted shortest path to extend weighted betweenness b_{wi} to estimate the possible packets passing through a node i . The weighted betweenness of node i , b_{wi} , is defined as

$$b_{wi} = \sum_{s,t} \frac{\sigma(s,i,t)}{\sigma(s,t)} \quad (1)$$

$\sigma(s,i,t)$ is the number of weighted shortest paths between nodes s and t that pass through node i and $\sigma(s,t)$ is the total number of weighted shortest paths between node s and t and the sum is over all pairs s, t of all distinct nodes. This may be normalized by dividing through the number of pairs of nodes not including i , which is $(n-1)*(n-2)/2$.

In the traffic driven epidemic spreading model, we can obtain the density of infected nodes $i(t)$ using mean-field theory as follows:

$$\frac{di(t)}{dt} = \lambda * \beta * n * \langle b_w \rangle * i(t) * (1-i(t)) \quad (2)$$

Equation (3) states that the average density of newly infected nodes is proportional to the spreading rate λ , the density of susceptible nodes that may become infected, $1-i(t)$, the probability of a packet passing through a link pointing to an infected node, $i(t)$, the total number of packets, $\beta*n$, and the fraction of packet passing through node which is equal to the average betweenness, $\langle b_w \rangle$. Equation (2) can be solved with the initial condition $i(t)_{t=0}=i_0$,

$$i(t) = \frac{i_0}{i_0 + e^{-\lambda*\beta*n*\langle b_w \rangle*t} - i_0 * e^{-\lambda*\beta*n*\langle b_w \rangle*t}} \quad (3)$$

In early stage, the density of infected nodes is very small, we can get that

$$i(t) \approx i_0 * e^{\lambda*\beta*n*\langle b_w \rangle*t} \quad (4)$$

The above calculations imply that the density of infected nodes is proportional to the spreading rate λ and the packet generation rate β in a given network. And the greater average betweenness is, the higher the density of infected nodes is. In other words, the epidemic spreading in a certain network is obviously accelerated when the average betweenness is greater.

Finally, we extend it to traffic driven SIS model. The only thing is to add the decaying item, which is proportional to the product of the curing rate γ (we set it to 1 for simplicity) and the average density of infected nodes $i(t)$. So in SIS model, the Equation is

$$\frac{di(t)}{dt} = -i(t) + \lambda * \beta * n * \langle b_w \rangle * i(t) * (1-i(t)) \quad (5)$$

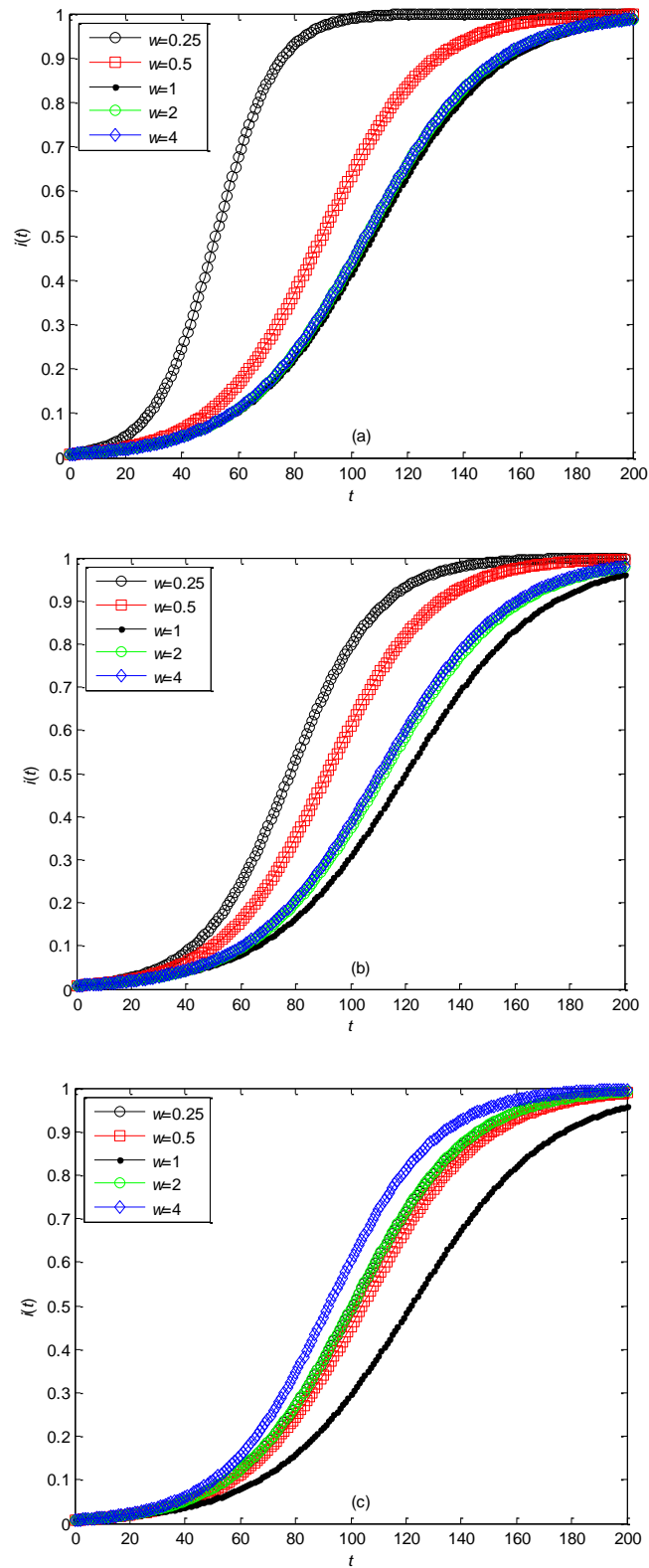
The ultimate purpose is the general prediction of a nonzero epidemic threshold, λ_c , which is the simple criterion for finding optimal immunization strategies. If the value of λ is above the threshold, the epidemic spread and become persistent. On the contrary, below it, the epidemics die out and endemic states are impossible. After imposing the stationary condition of (5), $di(t)/dt=0$, we obtain the equation of the epidemic threshold:

$$\lambda_c = \frac{1}{\beta * n * \langle b_w \rangle} \quad (6)$$

3. Simulations and Analysis

At first, we check the impact of within-community edge weight w on the average density of infected nodes using pseudo-random networks with $n = 128$ nodes which are

divided into $mods=4$ communities with 32 nodes in each community. The average degree $\langle k \rangle$ is set to 16. Simulation results are shown in Figure 1.



**Figure 1. Average Density of Infected Nodes $i(t)$ Versus Time t . $\lambda=0.02$ $\beta=1$
(a). $Z_{in}=12$ (b). $Z_{in}=8$ (c). $Z_{in}=4$**

From Figure 1, we can obtain that the edge weight have great influence on the traffic driven epidemic spreading. No matter the weight homogeneous network have pronounce community structure or not, the variation of edge weight will result in the acceleration of the traffic driven epidemic spreading. In each figure, when $w=1$, which means the edges have the same weight, the average density of infected nodes is less than the other situations. The epidemic spreading velocity, which can be measured by the average density of infected nodes in different time, is the lowest when the edges have the same weight.

Then we change the spreading rate λ and the packet generated rate β separately to check the impact of the spreading rate and the traffic flow on epidemic spreading. Simulation results are shown in Figure 2.

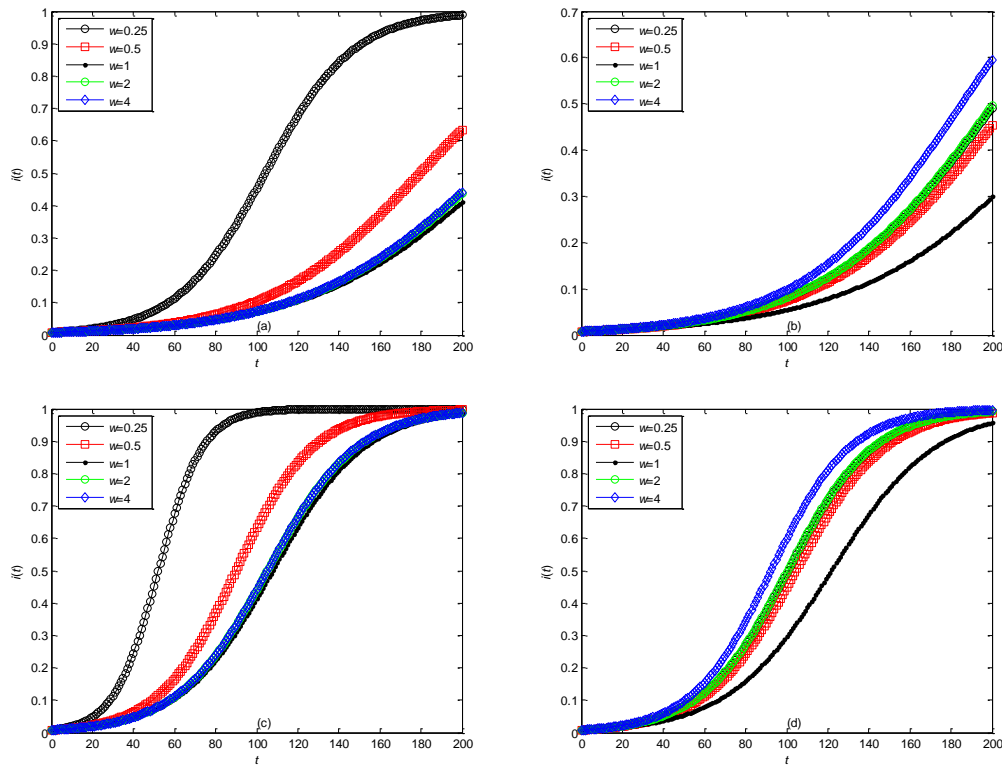


Figure 2. Average Density of Infected Nodes $i(t)$ versus Time t . (a). $Z_{in}=12$ $\lambda=0.01$ $\beta=1$ (b). $Z_{in}=4$ $\lambda=0.01$ $\beta=1$ (c). $Z_{in}=12$ $\lambda=0.01$ $\beta=2$ (d). $Z_{in}=4$ $\lambda=0.01$ $\beta=2$

Figure 2 also proves that when the edges have the same weight, the epidemic spreading velocity is the lowest. By comparing figure 2(a) with Figure 1(a) and Figure 2(b) with Figure 1(c), we can obtain that when the spreading rate is decreased, the epidemic spreading velocity is also decreased. By comparing Figure 2(c) with Figure 1(a) and Figure 2(d) with Figure 1(c), we can find that when the product of the spreading rate λ and the packet generated rate β is the same, the epidemic spreading velocity is nearly the same. It means our theoretical analysis is correct.

Then we check the epidemic threshold of traffic driven SIS model. We choose 25% nodes randomly and set these nodes to be infected. Then calculate the average density of infected nodes at different time.

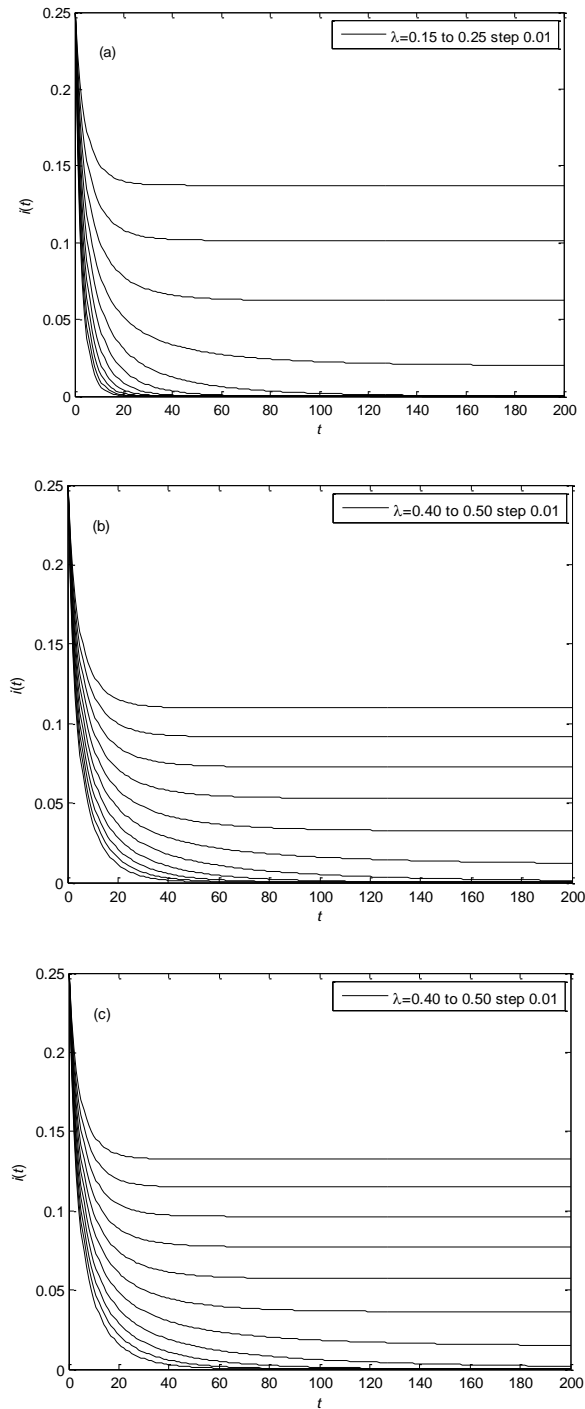


Figure 3. $i(t)$ versus t . The Results Denote the Case of Different Spreading Rate λ (from bottom to top) as Labeled in the Figures. $Z_{in}=12$ $\beta=1$ (a). $w=0.25$ (b). $w=1$ (c). $w=4$

Figure 3 exhibit the epidemic threshold λ_c of the different weight homogeneous networks. In Figure 3(a), when the spreading rate λ is lower than 0.21, the infected nodes disappear in the end state. And while it is up to 0.22, the infections can proliferate on the network. It is in good agreement with analytical finding of the formula (6), $\lambda_c=0.2157$. And the predication of formula (6) for $w=1$ and $w=4$ is 0.4451 and 0.4337 consequently.

One can see clearly from Figure 3(b) and 3(c) that the simulation results agree very well with the analytic results.

Then we change the with-in community edge number Z_{in} to check its impact on the epidemic threshold λ_c . Simulation results are shown in Figure 4 and Figure 5.

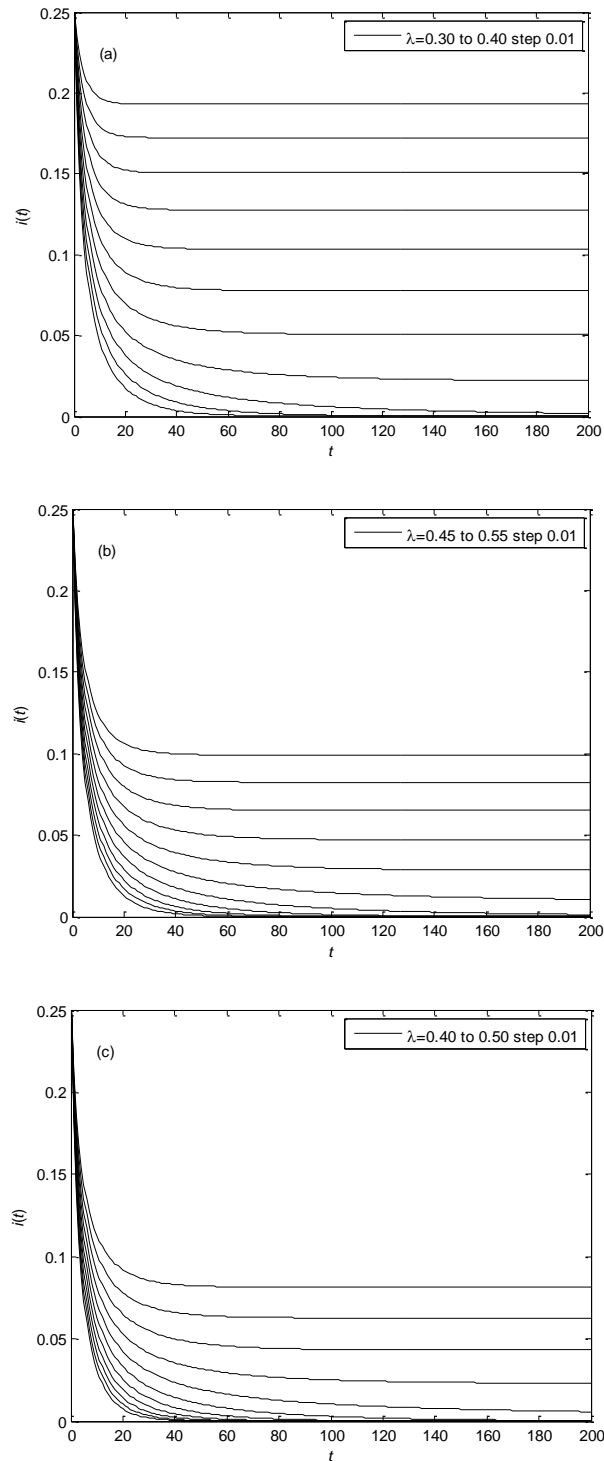


Figure 4. $i(t)$ versus t . The Results Denote the Case of Different Spreading Rate λ (from bottom to top) as Labeled in the Figures. $Z_{in}=8$ $\beta=1$ (a). $w=0.25$ (b). $w=1$ (c). $w=4$

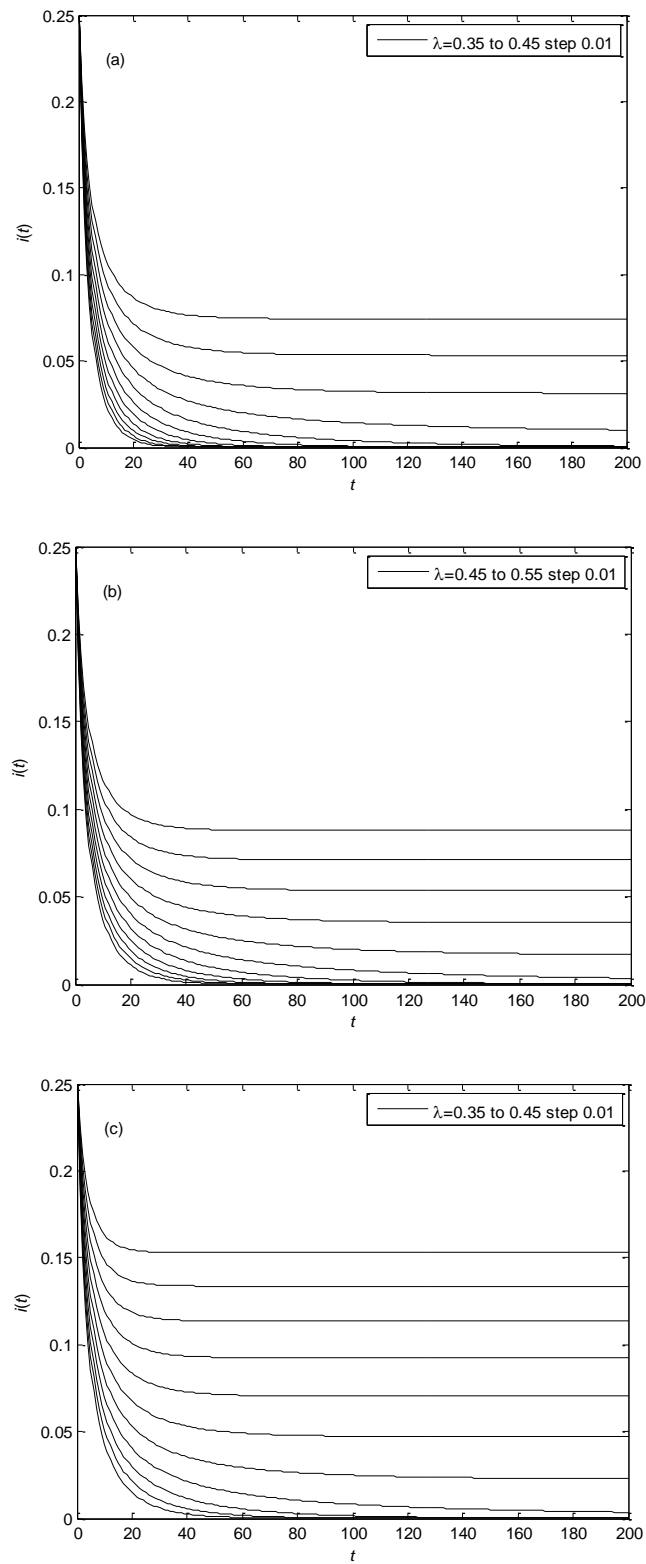


Figure 5. $i(t)$ versus t . The Results Denote the Case of Different Spreading Rate λ (from bottom to top) as Labeled in the Figures. $Z_{in}=4$ $\beta=1$ (a). $w=0.25$ (b). $w=1$ (c). $w=4$

When the with-in community edge number Z_{in} is 8, the analytic results of formula (6) are 0.3228, 0.4955, and 0.4593 with $w=0.25, 1,$ and 4 . When Z_{in} is 4, the analytic results are 0.4165, 0.5016, and 0.3812. The simulation results shown in Figure 4 and Figure 5 also agree well with the theoretical analysis.

Finally, we investigate the detail relationship between the epidemic threshold λ_c and the with-in community weight w by increasing w gradually.

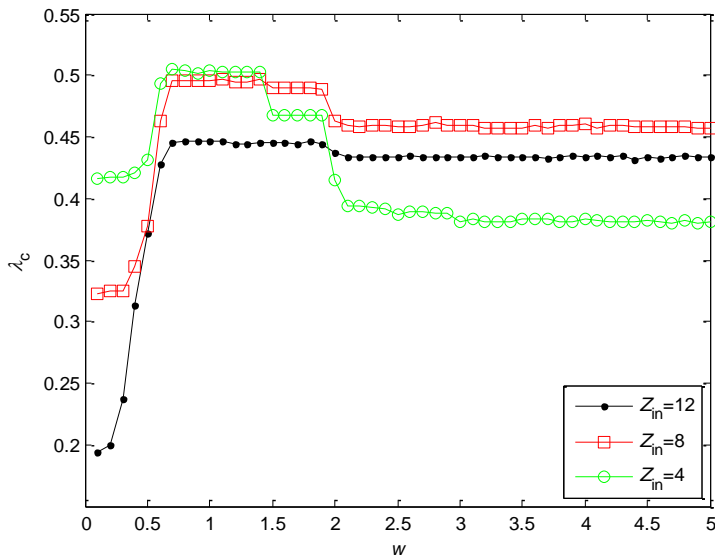


Figure 6. The Epidemic Threshold λ_c Versus the with-in Community Weight w . $\beta=1$

From Figure 6, we can notice that the epidemic threshold λ_c reaches its peak when the with-in community weight w is around 1. When $w \ll 1$, the epidemic threshold λ_c is very small. And when $w > 2$, the epidemic threshold λ_c is almost the same. It also proves that when the edges have the same weight, it is better to control the epidemic spreading.

Then we check the impact of the packet generated rate β on the epidemic threshold.

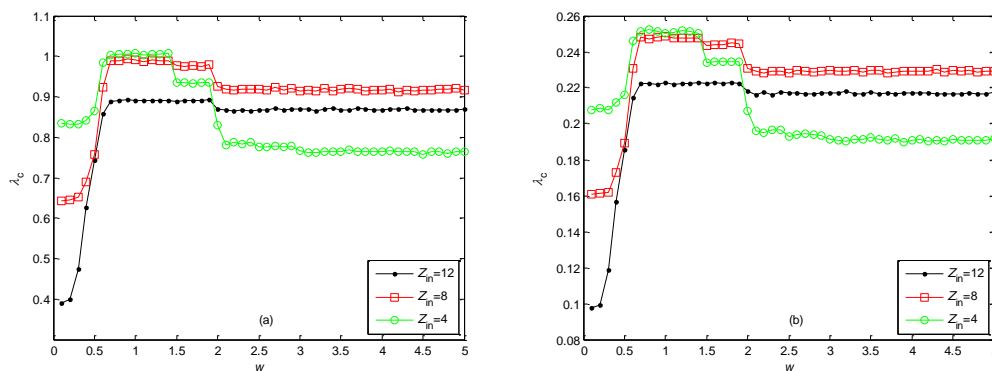


Figure 7. The Epidemic Threshold λ_c versus the with-in Community weight w . (a). $\beta=0.5$ (b). $\beta=2$

Figure 7 shows that changing the packet generated rate β will only affect the absolute value of the epidemic threshold. The enhancement of the packet generated rate will result in more packets in the network which will increase the epidemic threshold of traffic driven SIS model.

4. Conclusions

Aiming at controlling the traffic driven epidemic spreading in weight homogeneous networks, the epidemic spreading velocity of SI model and the epidemic threshold of SIS model are discussed in this paper. By changing the with-in community edge weight, we check the average density of infected nodes of SI model in different with-in community edge number to find when the edges have the same weight, the epidemic spreading velocity is the lowest. Additionally, the analytical results of the epidemic threshold of SIS model are obtained, which agree well with the numerical simulations. The results display that the epidemic threshold is proportional to the inverse of the product of the average weighted betweenness, the node number, and the packet generated rate in weighted homogeneous networks. It is of practical importance to develop appropriate immunization strategies to control the epidemic spreading.

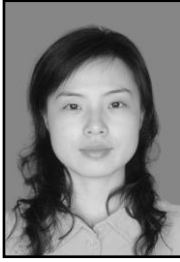
Acknowledgements

This work was partially supported by the National Natural Science Foundation of China (Grant No. 61373136, 61375121), the Natural Science Foundation of Jiangsu Province, China (Grant No. BK2012082), the Research Foundation of Jinling Institute of Technology (Grant No. JIT-B-201406) and sponsored by Qing Lan Project. The author also gratefully acknowledges the helpful comments and suggestions of the reviewers, which have improved the presentation.

References

- [1] P. Erdős, and A. Rényi, "Publications of the Mathematical Institute of the Hungarian Academy of Sciences", vol. 5 (1960).
- [2] D. J. Watts and S. H. Strogatz, "Nature", (1998), pp. 393, 6684.
- [3] A. L. Barabási, and R. Albert, Science. (1999), pp. 286, 543.
- [4] M. Barthélemy, A. Barrat, R. Pastor-Satorras, and A. Vespignani, Phys. Rev. Lett., vol. 92, no. 17, (2004).
- [5] M. Barthelemy, A. Barrat, R. Pastor-Satorras, and A. Vespignani, "Journal of Theoretical Biology", vol. 235, no. 2, (2005).
- [6] T. Zhou, J. G. Liu, W. J. Bai, G. R. Chen, and B. H. Wang, Phys. Rev. E. vol. 74, no. 5, (2006).
- [7] R. Pastor-Satorras, and A. Vespignani, Phys. Rev. Lett., vol. 86, no. 14, (2001).
- [8] R. Pastor-Satorras, and A. Vespignani, Phys. Rev. E. vol. 63, no. 6, (2001).
- [9] H. W. Hethcote, SIAM REVIEW. vol. 42, no. 4, (2000).
- [10] R. M. May, and A. L. Lloyd, Phys. Rev. E. vol. 64, no. 6, (2001).
- [11] Y. Moreno, R. Pastor-Satorras, and A. Vespignani, "European Physical Journal B", vol. 26, no. 4, (2002).
- [12] N. T. J. Bailey, "The mathematical theory of infectious diseases and its applications", Hafner Press, New York (1975)
- [13] S. Meloni, A. Arenas, and Y. Moreno, PNAS. vol. 106, no. 40, (2009).
- [14] Y. Q. Wang, and G. P. Jiang, ACTA PHYSICA SINICA, vol. 60, (2011).
- [15] H. X. Yang, W. X. Wang, and Y. C. Lai, CHAOS, vol. 22, (2012).
- [16] H. X. Yang, W. X. Wang, Y. C. Lai, and B. H. Wang, "Europhysics Letters", vol. 98, no. 6, (2012),.
- [17] M. E. J. Newman, Phys. Rev. E. vol. 64, no. 1, (2001).
- [18] A. Barrat, M. Barthelemy, R. Pastor-Satorras, and A. Vespignani, PNAS, vol. 101, pp. 11, (2004).
- [19] R. Pastor-Satorras, and A. Vespignani, "Evolution and structure of the Internet: A statistical physics approach", Cambridge University Press, Cambridge (2007).
- [20] M. E. J. Newman, and M. Girvan, Phys. Rev. E., vol. 69, no. 2, (2004).
- [21] U. Brandes, "Journal of Mathematical Sociology", vol. 25, no. 2, (2001).
- [22] E. W. Dijkstra, "Numerische mathematik", vol. 1, no. 1, (1959).

Authors



Zinan Chang, She received the Bachelor Degree in Computational Mathematics from Nanjing Normal University in 2000, Master degree in Computational Mathematics from Nanjing Normal University in 2006. From 2006 to now, she is a lecture at the School of Computer Engineering, Jinling Institute of Technology and working for Jiangsu Information Analysis Engineering Laboratory. Her current research interests include information security and complex dynamical networks.



Fei Shao, He received the Bachelor Degree in Computer Application from Jiangsu University of Science and Technology in 1998, Master degree in Computer Application Technology from Nanjing University of Technology in 2003, and Ph. D Degree in Information Security from Nanjing University of Posts & Telecommunications in 2013. From 2010 to now, he is an associate professor at the School of Computer Engineering, Jinling Institute of Technology and working for Jiangsu Information Analysis Engineering Laboratory. His current research interests include information security and complex dynamical networks.

