Traffic driven epidemic spreading in weighted networks with different routes

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Received 12 June 2014, www.tsi.lv

Abstract

How does traffic processes in weighted networks impact on the dynamics of epidemic spreading have attracted increasing attention. It is of great importance to reduce the epidemic spreading velocity and increase the critical epidemic threshold of those real world networks. In this paper, the traffic driven epidemic spreading behaviour in BBV weighted networks is investigated. Formulas to describe the infected density and the epidemic threshold of weighted networks are derived and validated by simulations. The infected density and the epidemic threshold are found to undergo a corresponding change when packets are forwarded through different routes according to the different tuneable parameter. By simulations, the optimal route is explored which is better to control the epidemic spreading.

Keywords: weighted network, BBV network, epidemic spreading, traffic flow

1 Introduction

Since the seminal study of small-world networks by Watts and Strogatz [1] and on scale-free networks by Barabási and Albert [2], the complex networks have attracted the dramatically increasing interest in the past few years. A great deal of real networks can be viewed as complex networks while nodes representing individuals and edges representing the relationships between them. The previous studies on networks have been principally focused on those unweighted networks, edges between nodes are either present or not, represented as binary states. However, lots of social, biological, and communication systems such as the mobile networks [3], the scientific collaboration networks [4], the cellular metabolism [5], the world-wide airport network [6] and the Internet [7] have presented that real networks are specified not only by their topology but also by the weight of the edges. Lots of models [8-15] have been presented to describe those weighted networks, among which the BBV model [12], take into account the coupled dynamical evolution of topology and weights, is the most widely used.

The past decade has witnessed lots of large-scale international epidemics among human, animal, and plant which caused an enormous amount of damage or loss. Those disease outbreaks in real systems can be viewed as epidemic spreading on complex networks while individuals are modelled as nodes and possible contacts between individuals are connected by edges. It is of great significance to inspect how to control the epidemic spreading taking place in complex networks. Many models have been proposed to investigate the feature of epidemic spreading such as SIS [16, 17], SIR [18], and SI [17, 19, 20] models. In these models, a node is classified in three states: susceptible (which will not infect others but may be infected), infected (which is infective) and recovered (which has recovered from the disease and has immunity). The propagation of epidemic from one node to another is assumed to be a reaction process, that is, an infected node can infect any of its neighbourhood nodes with a fixed probability v at each time step and the recovering rate of infected ones is ψ. Hence the effective spreading rate λ is defined as $\lambda=v/\psi$. Without lack of generality, ψ is set to 1, since it only affects at the definition of the time scale.

Recently, it was found that a susceptible node is more likely to be infected if it receives more packets from infected neighbours [21]. And in many real systems, propagation of the epidemic will not occur unless there is a packet interaction on the network that can physically transfer the epidemic from one node to another. The probability that the epidemic spreads from infected to susceptible nodes mainly depends on the traffic flow. A novel approach, which called traffic driven epidemic spreading is introduced to investigate the outcome of the epidemic spreading process driven by traffic flow [21-24]. However, these studies are focused on unweighted networks, the important issue of how the traffic-driven epidemic spreads in weighted networks has not been considered.

In this paper, we probe the traffic driven epidemic spreading behaviour in BBV weighted networks to obtain the formulas of the infected density and the epidemic

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threshold of weighted networks. While the packets are specified to transfer through the path based on the weight of the edges with a tuneable parameter α, the optimal route is explored which is better to control the epidemic spreading by simulations.

This paper is organized as follows. In section 2 we describe the models and the formulas, followed by the experimental evaluations on BBV weighted networks and real world network in section 3. The conclusions are given in section 4.

2 Models

2.1 NETWORK MODEL

In BBV weighted networks, the topological as well as weighted properties can be described by a weighted adjacency matrix $\boldsymbol{W}$, whose elements w_{ij} denote the weight of the edge between node i and j. The generation of BBV weighted networks is based on two coupled mechanisms:[6, 11]

(i) Growth. Starting from an initial number of N_0 nodes fully connected by edges with assigned weight w_0, a new node will be added at each time step. The newly added node is connected to m different previously existing nodes with equal weight w_0 for every edge and chooses preferentially nodes with large strength according to the probability $\prod_{n\to i} = s_i \Big/ \sum_l s_l$, where s_i is the node strength described as $s_i = \sum_j w_{ij}$.

(ii) Weight dynamics. The weight of each newly added edge is initially set to a given value w_0 which is often set to 1 for simplicity. But the adding of edge connecting to node i will result in increasing the weight of the other edges linked to node i which is proportional to the edge weights. If the total increase is δ (we will focus on the simplest form: $\delta_i=\delta$), we can get

$$w_{ij} = w_{ij} + \Delta w_{ij} = w_{ij} + \delta * \frac{w_{ij}}{s_i}. \tag{1}$$

This will yield the strength increase of node i as:

$$s_i = s_i + \delta + w_0. \tag{2}$$

The degree distribution of BBV network $P(k) \propto k^{-\gamma_k}$ and the strength distribution $P(s) \propto s^{-\gamma_s}$ yield scale-free properties with the same exponent [6, 11, 12, 14]:

$$\gamma_k = \gamma_s = \frac{4\delta+3}{2\delta+1} = 2 + \frac{1}{2\delta+1}. \tag{3}$$

2.2 TRAFFIC MODEL

Our traffic model can be described as follows:

1) All the nodes can create packets with addresses of destination, receive packets from other nodes, and forward packets to their destinations.

2) At each time step t, an information packet is generated at every node with probability β with randomly chosen sources and destinations.

3) At each time step t, each node forwards all packets in its queue one step according to the specified route at the same time. In our model, each node has unbounded packet delivery capability for simplicity which means traffic congestion will not occur.

4) A packet, upon reaching its destination, is removed from the system.

Denote $P_{i\to j}$ as the path between node i and j, which pass through the nodes sequence $x_0(=i), x_1, x_2, \cdots, x_{n-1}, x_n(=j)$, we define

$$F(P_{i\to j}, \alpha) = \sum_{i=0}^{n-1} {w_{ij}}^{\alpha}. \tag{4}$$

In our routing strategy, we specify the route between i and j as the one makes $F(P_{i\to j}, \alpha)$ minimum under a tunable parameter α. When α is 0, the specified route is the same as the traditional the shortest path route [25].

2.3 EPIDEMIC MODEL

While investigating the dynamical behaviours in the very early stage of epidemic outbreaks, this case corresponds to the simplified SI [20] model, for which infected nodes remain always infective and spread the infection to susceptible neighbours with spreading rate λ.

With the average density of infected nodes of degree k defined as $i_k(t)$, in BBV weighted networks we have

$$\frac{di_k(t)}{dt} = \lambda * \beta * n * b_k * \left(1 - i_k(t)\right) * \Theta(t). \tag{5}$$

The right-hand side takes into account the probability that a node with k neighbours belongs to the susceptible class represented by $(1-i_k(t))$ and gets the infection via packets travelling from infected nodes. The latter process is determined by the spreading probability λ, the number of packets that a node of degree k receives at each time step $\beta*n*b_k$ (n is the node number and b_k is the so-called algorithmic betweenness [26]), and the probability $\Theta(t)$ that a packet travels through a link pointing to an infected node.

Assume that the network is uncorrelated, $\Theta(t)$ takes the form

$$\Theta(t) = \frac{\sum_k b_k P(k) i_k(t)}{\sum_k b_k P(k)} = \frac{\sum_k b_k P(k) i_k(t)}{< b >}. \tag{6}$$

When the epidemic begins spreading, the density of infected nodes $i_k(t)$ is very small. With the initial condition $i(t)_{t=0} = i_0$, we gain

$$i(t) = i_0\left(\frac{<b>^2}{<b^2>}(e^{\frac{t}{\tau}} - 1) + 1\right). \quad (7)$$

And the epidemic outbreaks time scale of the BBV networks is

$$\tau = <b> /(\lambda * \beta * n * <b^2>). \quad (8)$$

The classical SIR [18] model, which is often used for these in which the infected nodes will get recovered and will not return to the susceptible state again, and thus nodes run stochastically through the cycle susceptible → infected → recovered. It is generally used to study epidemics leading to endemic states with a stationary average density of infected nodes. With the effective spreading rate is defined as λ (which means the recovering rate ψ=1), we can obtain

$$\begin{cases} \dfrac{ds_k(t)}{dt} = -\lambda * \beta * n * b_k * s_k(t) * \Theta(t) \\ \dfrac{di_k(t)}{dt} = -i_k(t) + \lambda * \beta * n * b_k * s_k(t) * \Theta(t) \\ \dfrac{dr_k(t)}{dt} = i_k(t) \end{cases}. \quad (9)$$

In the second row of formula (9), $-i_k(t)$ means the infected nodes are recovered with ψ=1 and $s_k(t)$ means the average density of susceptible nodes of degree k which is replaced by $(1-i_k(t))$ in formula (5). $r_k(t)$ means the average density of recovered nodes of degree k. The other symbols in formula (9) have the same meaning as in formula (5).

By imposing $di_k(t)/dt=0$ and $s_k(t) + i_k(t) + r_k(t) = 1$, we get

$$i_k(t) = \frac{\lambda * \beta * n * b_k * \Theta}{1 + \lambda * \beta * n * b_k * \Theta}. \quad (10)$$

Substituting formula (6) into formula (10), we get

$$\Theta = \frac{\sum_k \dfrac{b_k^{\,2} * P(k) * \lambda * \beta * n * \Theta}{1 + b_k * \lambda * \beta * n * \Theta}}{\sum_k b_k * P(k)}. \quad (11)$$

The value $\Theta = 0$ is always a solution. In order to have a non-zero solution, the condition must be filled.

$$\frac{\dfrac{d}{d\Theta}\left(\sum_k \dfrac{b_k^{\,2} * P(k) * \lambda * \beta * n * \Theta}{1 + b_k * \lambda * \beta * n * \Theta}\right)}{\sum_k b_k * P(k)}\Big|_{\Theta=0} \geq 1. \quad (12)$$

Therefore,

$$\frac{\dfrac{d}{d\Theta}\left(\sum_k \dfrac{b_k^{\,2} * P(k) * \lambda * \beta * n * \Theta}{1 + b_k * \lambda * \beta * n * \Theta}\right)}{<b>}\Big|_{\Theta=0} \geq 1, \quad (13)$$

$$\frac{1}{<b>} * \sum_k b_k^{\,2} * P(k) * \lambda * \beta * n \geq 1, \quad (14)$$

$$\frac{1}{<b>} * <b^2> * \lambda * \beta * n \geq 1. \quad (15)$$

We can obtain the epidemic threshold of traffic driven SIR epidemic model in BBV weighted networks:

$$\lambda_c = \frac{<b>}{<b^2> * \beta * n}. \quad (16)$$

3 Simulations and analysis

In figure 1, we plot the infected density $i(t)$ versus time t with different parameter α in a BBV weighted network with n=1000, δ=6, m=6 and ω_0=1. (For every network, 20 instances are generated and for each instance, we run 20 simulations. The results are the average over all the simulations.)

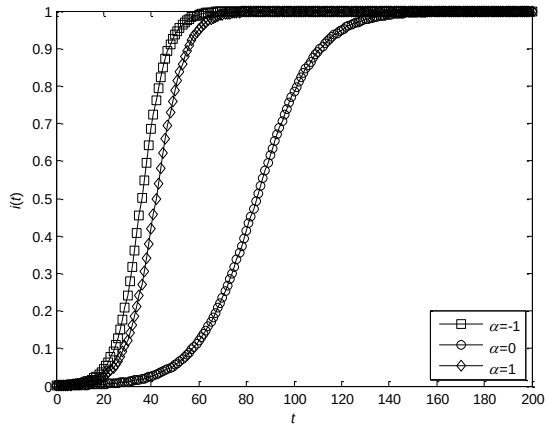

FIGURE 1 $i(t)$ VS t. BBV network with λ=0.01, β=1, n=1000, δ=6, m=6 and ω_0=1

From figure 1, we can discover that the infected density $i(t)$ varies with the tuneable parameter α. Figure 1 shows that when the parameter t is the same, the infected density $i(t)$ is the smallest in the case that the packets are forwarded through the route path with tuneable parameter

α=0, which means the velocity of epidemic spreading is the lowest.

Then we check the impact of the spreading rate λ and the packet generation rate β on the infected density. Simulation results are shown in figure 2.

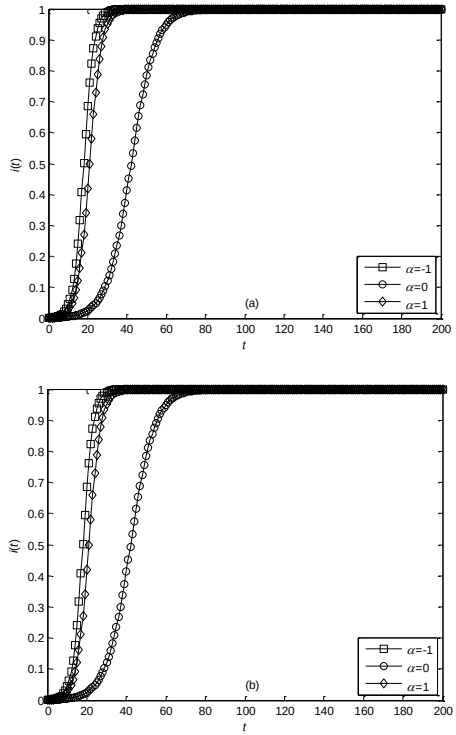

FIGURE 2 $i(t)$ VS t. BBV network with n=1000, δ=6, m=6 and ω_0=1. (a) λ=0.02, β=1 (b) λ=0.01, β=2

As is shown in figure 2(a), when the spreading rate λ is doubled, the velocity of epidemic spreading is increased correspondingly which means the spreading velocity is apparently related to the characteristics of epidemic. In figure 2(b), the packet generation rate β is doubled which means there are more packets and more traffic flow in the network, the spreading velocity is greater consequently. Compare figure 2(a) with figure 2(b), we can obtain that in a given network, when the product of the spreading rate and the packet generation rate is fixed, the velocity of epidemic spreading is almost constant. The accuracy of the formula (7) is proved to be correct.

Then we check the impact of the BBV parameter δ on the velocity of epidemic spreading. We set δ=3 and 12 to obtain different simulation results in figure 3.

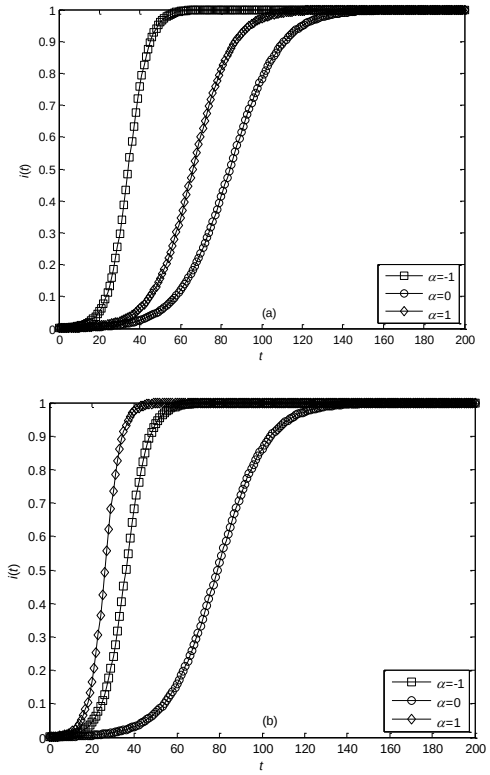

FIGURE 3 $i(t)$ VS t. BBV network with λ=0.01, β=1, n=1000, m=6 and ω_0=1. (a) δ=3 (b) δ=12

From figure 3(a) and 3(b), we can figure that even though the BBV parameter δ is changed, the velocity of epidemic spreading with α=0 is the slowest. And the BBV parameter δ will have a great influence on the velocity of epidemic spreading.

Then we check the influence of the newly added edge number m and the total node number n. Simulation results are shown in figure 4.

From figure 4, we can come to the conclusion that both the newly added edge number m and the total node number n have a certain effect on the velocity of epidemic spreading. And the velocity of epidemic spreading with α=0 is also the slowest.

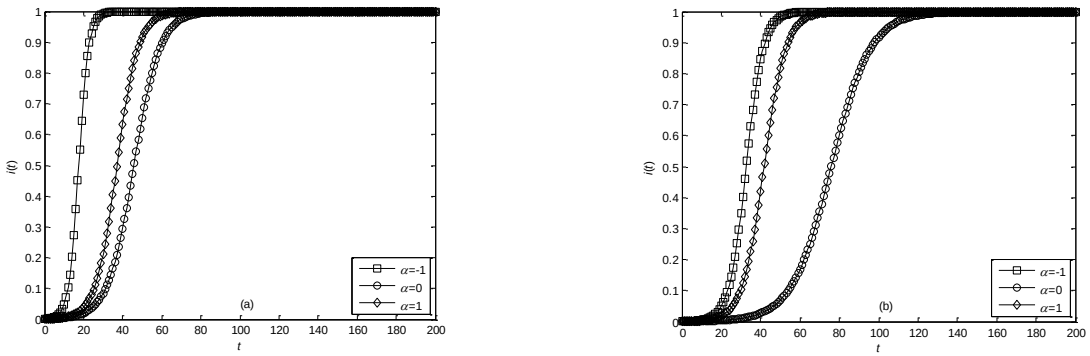

FIGURE 4 $i(t)$ VS t. BBV network with λ=0.01, β=1, δ=6 and ω_0=1. (a) n=1000, m=3 (b) n=2000, m=6

Then we extend to the SIR model. 20% of nodes are infected in the initial status and the spreading rate is increased step by step to check the infected density $i(t)$ of the endemic state.

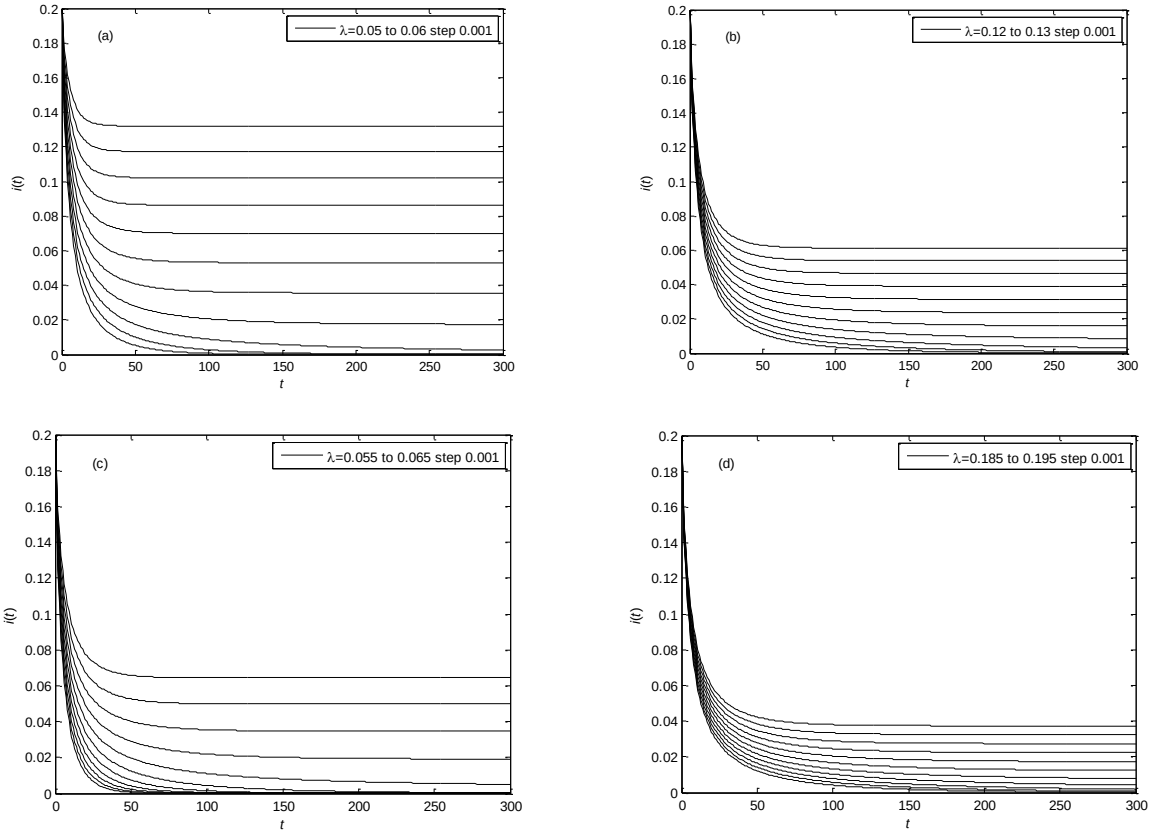

FIGURE 5 $i(t)$ VS t. BBV network with β=1, n=1000, δ=6, m=6 and ω_0=1. The results denote different spreading rate λ (from bottom to top) as labelled in the figures. (a) α=-1 (b) α=0 (c) α=1(d) α=0.2

Figure 5(a), 5(b), 5(c) and 5(d) exhibit the epidemic threshold λ_c of a BBV weighted network under differ route paths. In figure 5(a), when the spreading rate λ is lower than 0.052, the infected nodes disappear gradually. And while it is up to 0.053, the infections can proliferate on the network. It is in good agreement with analytical finding of the formula (16), λ_c=0.0521.

And the predication of formula (16) for α=0, α=1 and α=0.2 is 0.1220, 0.0608 and 0.1877 consequently. One can see clearly from figure 5(b), 5(c) and 5(d) that the simulation results also agree very well with the analytic results.

To explore the exact optimal value of the tuneable parameter α, which can produce the largest epidemic threshold, we enlarge the tuneable parameter α step by step to achieve the corresponding epidemic threshold.

Figure 6 demonstrates that the epidemic threshold varies with the tuneable parameter, that is to say, the different route path. Moreover, the epidemic threshold reaches the peak when α is around 0.2. It means it is the most effective way to restrain the traffic driven epidemic spreading when the packets are forwarded through the route path specified by α=0.2.

To find the influence of the packet generation rate β on the epidemic threshold, we change β from 1 to 2 to get the simulation result as presented in figure 7.

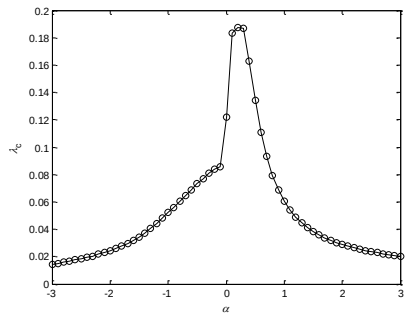

FIGURE 6 λ_c VS α. BBV network with β=1, n=1000, δ=6, m=6 and ω_0=1

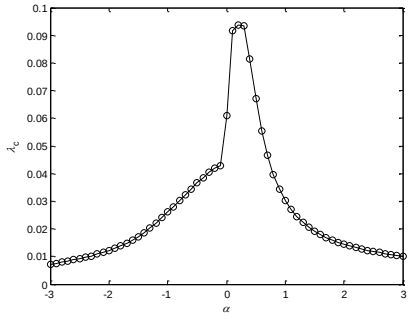

FIGURE 7 λ_c VS α. BBV network with β=2, n=1000, δ=6, m=6 and ω_0=1

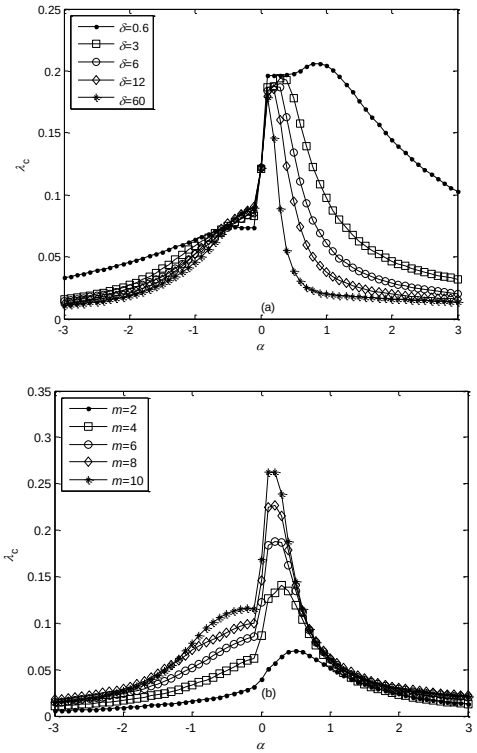

FIGURE 8 λ_c VS α. BBV network with β=1, n=1000 and ω_0=1. (a) m=6 (b) δ=6

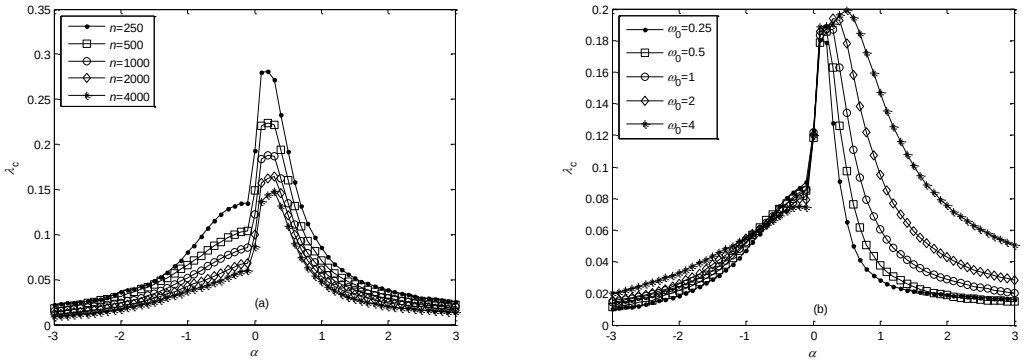

FIGURE 9 λ_c VS α. BBV network with β=1, δ=6 and m=6. (a) ω_0=1 (b) n=1000

When the packet generation rate is doubled, the epidemic threshold decreases by almost half which also agree well with the formula (16). The epidemic threshold also reaches the maximum value when α is around 0.2.

Then we investigate how the BBV parameter δ, the newly added edge number m, the total node number n and the assigned weight w_0 affect the epidemic threshold. The simulation results are depicted in figure 8 and figure 9.

As shown in figure 8(a), the highest epidemic threshold is achieved at different tuneable parameter α because of different parameter δ. The smaller the parameter δ is, the flatter the curve of the epidemic threshold is. Actually, when δ is set to 0, it is an unweighted network where the epidemic threshold is fixed no matter how the tuneable parameter α changes. From formula (3) we can discover that both the degree distribution and the strength distribution of BBV network depend on the parameter δ. When the parameter δ is increased, the distributions become broader which result the maximum value of the epidemic threshold is obtained at different tuneable parameter α.

From figure 8(b) and figure 9(a), we can notice that the newly added edge number m and the total node number n have a little effect on the impact of tuneable parameter α on the epidemic threshold. They only affect the absolute value of the epidemic threshold. As shown in figure 9(b), the assigned weight w_0 also affect the tuneable parameter α to obtain the highest epidemic threshold. As presented in formula (2), when w_0 is considerably high, the minor variation of the parameter δ may be passed over. In other words, all edges have almost the same weight. The smaller the parameter δ is, the flatter the curve of the epidemic threshold is. Actually, when δ is set to 0, it is an unweighted network where the epidemic threshold is fixed no matter how the tuneable parameter α changes.

Finally, we choose the scientific collaboration network [27] which has a giant component of 5835 nodes to check whether our conclusions are tenable in real world network. Simulation result is shown in figure 10.

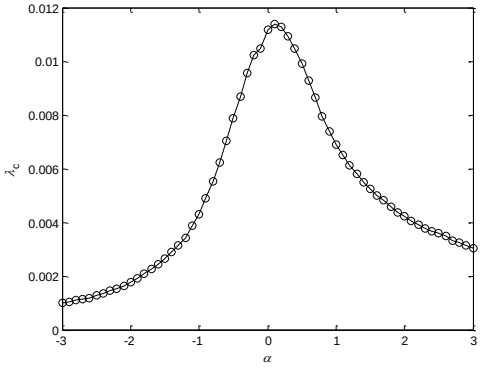

FIGURE 10 λ_c VS α. Real world network

From figure 10 we can discover that the epidemic threshold also varies with the tuneable parameter in the scientific collaboration network which has the maximum epidemic threshold when the tuneable parameter α is near 0.2. It means our conclusions also work well in the real world network.

4 Conclusions

Considering the traffic driven epidemic spreading behaviour in BBV weighted networks, this paper has deduced the formulas to describe the infected density and the epidemic threshold of BBV weighted networks. The infected density and the epidemic threshold of BBV weighted networks are found to be proportional to the ratio between the first and the second moments of the node betweenness distribution as well as the scale free networks. The validity of the presented formulas is demonstrated by simulations. The infected density and the epidemic threshold vary accordingly when packets are forwarded through different routes according to the tuneable parameter. In most cases, the epidemic threshold reaches the maximum when the optimal value of the tuneable parameter is 0.2. It is worth mentioning that in some weighted networks the optimal values fluctuate around the mentioned value. At last, we use the scientific collaboration network to show the validity of our conclusions on real world networks.

Acknowledgements

This work was partially supported by the National Natural Science Foundation of China (Grant No. 61373136), the Natural Science Foundation of Jiangsu Province, China (Grant No. BK2012082) and sponsored by Qing Lan Project.

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Publications: 3 papers