Epidemic spreading on adaptive weighted networks based on SEIS model *†

Jiangtian Pan [1],Li Ding [1§],Yun Feng [1],Yunhan Huang [1]

Abstract

In this paper, we study the problem of epidemic spreading on an adaptive weighted network based on the SEIS model, in which the weight varies depending on the feedback of local infective information. The model is tested by discrete-time Monte-Carlo simulation based on the initial ER random networks. Specifically, we focus on the effect of the exposed period on epidemic spreading and weight adapting. In addition, the interactions among the infected rate, latency unrevealed rate and recovery rate in epidemic spreading are also considered. Besides, simulations of steady-state, dynamic process and threshold condition based on these three parameters are displayed. Furthermore, effects of different weight-adapting strategy on epidemic spreading are shown. We find that compared with constant weights in the network, adaptive weight can effectively contribute to inhibit the epidemic spreading.

Keywords Epidemic spreading; adaptive weighted network; SEIS model.

1 Introduction

Epidemic spreading in complex networks has attracted an increasing amount of attention during the past few decades. The majority of studies on epidemic spreading are based on SI, SIS, SIR models [1]-[10], in which S, I, R individually represents the susceptible, infected and recovery period. Recently, a new model

*This work is supported by National Natural Science Foundation of China under Grant 61403284.

†1. Department of Automation, Wuhan University, Wuhan, 430072, P. R. China. § Corresponding Author: Email: liding@whu.edu.cn (Li Ding), Tel: +86-27-68773985

named as SEIS has been raised. Compared with SIS model, there is a transition period between susceptible and infected period, which is named exposed period (labeled as 'E'). Individuals who are in exposed period are infectious although they fail to demonstrate any infected symptoms. Based on SEIS model, the majority of research are focused on global dynamics of the epidemic spreading. In [11] Yoshiaki and Yoichi studied global dynamical consistency between the continuous SEIS epidemic model and its discrete-time analogue. Xu et al. used Lyapunov function, LaSalle's invariance principle and comparison arguments to analyze the interaction between global dynamics and basic reproduction number in [12]. In [13], Liu et al. introduced the stochasticity into a deterministic model which had stated variables SEIS with varying population size. They concluded that the stochastic model possessed a unique global solution under building up a suitable Lyapunov function and using generalized Itô's formula. Moreover, some simple conditions on extinction and persistence in the mean of the disease with probability one were shown. When the noises were weak, there was a stationary distribution to the stochastic model. In [14] Bowong et al. proposed a method based on synchronization to identify the parameters and to estimate the underlying variables for the SEI model from actual data. They exploited the close link between mathematical modelling, structural identifiability analysis, synchronization, and parameter estimation to obtain biological insights into the system modelled.

Previous research listed above considers the dynamic behavior based on the global information, which means that individuals can receive infective information from the whole network. However, in large sums of networks, individuals can only be connected to a limited neighboring nodes. Hence, researching on the information from neighboring nodes has a more practical meaning. This kind of information, which is obtained from neighbors can be termed as 'local information'. In our previous work [15], we have considered epidemic spreading on an adaptive weighted network in which the topology varies according to the both global and local infected information based on the SIS model. In this paper, we extend the framework of [15] to SEIS model. Specifically, due to the exposed period, there will exist a difference of infected density between reality and what individuals receive. Hence, the adaptive weights are different from the values that are supposed to be.

In Figure 1, due to the exposed period, individuals cannot recognize whether neighbors are infectious or

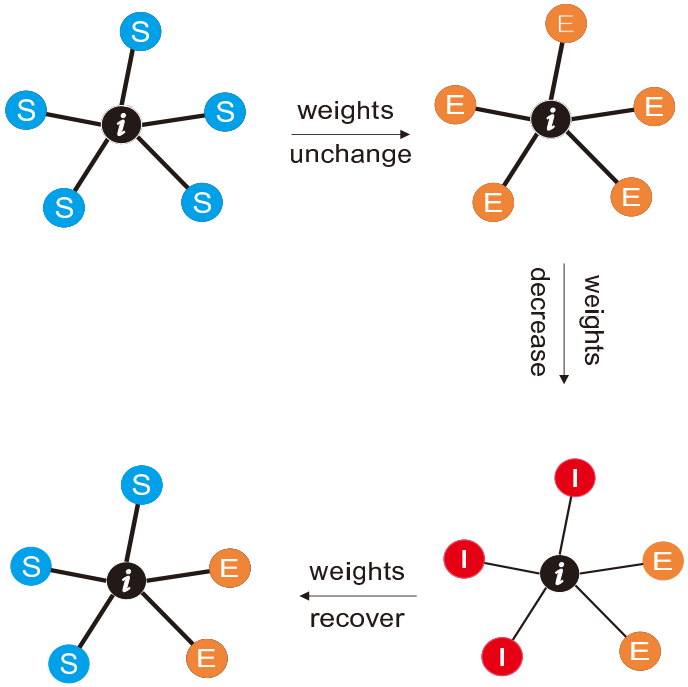


Figure 1: Edge weight updates according to the local infective information.

not. So individual i updates all the edge weights in each step. It can be found that when the density of local infected individuals increases the weights will decrease accordingly. However, when most of the infectious individuals are at exposed period, individual i has a large probability to be infected but still keeps large weight of edges to the neighboring individuals. Hence the prevention of individual i is weakened. This is quite different from our previous work [14].

The outline of this paper is as follows. In Section 2, we give the model description. Then the threshold condition is presented in Section 3. Section 4 includes the simulation results of steady-state value, dynamic propagation and threshold condition. Finally, the proposed algorithms about how weight varies according to the local infective information are shown in Section 5.

2 Model description

In this part, we specifically describe the discrete-time mathematical model based on the microscopic Markov chain approach (MMCA) [16]. In our model, each individual contacts with all his neighbors once in each step. We define $P_i^S(t)$, $P_i^E(t)$, $P_i^I(t)$ as the probability that individual i is susceptible, exposed and

infected at step t respectively. Hence, $P_i^S(t) + P_i^I(t) + P_i^E(t) = 1$. Meanwhile α is defined as the probability of staying in exposed period in the next step which is named as latency unrevealed rate. And β and γ are individually named as infected rate and recovery rate. The MMCA equation accordance with our model can be presented in (2.1) below,

$$\begin{cases} P_i^S(t+1) = P_i^S(t)q_i(t) + P_i^I(t)\gamma, \\ P_i^E(t+1) = \alpha P_i^E(t) + (1 - q_i(t))P_i^S(t), \\ P_i^I(t+1) = P_i^I(t)(1-\gamma) + (1-\alpha)P_i^E(t), \end{cases} \tag{2.1}$$

where $q_i(t)$ stands for the probability that individual i is not being infected at step t and

$$q_i(t) = \prod_{j=0}^{k_i}(1 - \beta(P_j^I(t) + P_j^E(t))\omega_{ij}(t)), \tag{2.2}$$

in which $\omega_{ij}(t)$ means the edge weight connecting from individual i to its neighboring individual j and can also be defined as follows according to different weights adapting strategies

$$\omega_{ij}(t) = 1 - \frac{n_i(t)}{k_i}, \qquad \text{(Strategy(a))}$$

$$\omega_{ij}(t) = 1 - (\frac{n_i(t)}{k_i})^2, \qquad \text{(Strategy(b))}$$

$$\omega_{ij}(t) = 1 - \sqrt{1 - (\frac{n_i(t)}{k_i} - 1)^2}. \qquad \text{(Strategy(c))}$$

(2.3)

Here k_i stands for the degree of individual i and n_i means the number of infected individuals in the neighborhood. Strategy (a) - (c) are shown in Figure 2.

(2.1) - (2.2) demonstrate the transitions among the probability of three states between step t and its next step $t+1$. It is worth noting that when α=0, those susceptible individuals who are infected in step t, will enter into the exposed period at step $t+1$ and immediately be revealed into the infected period at step $t+2$. In this way, the model is similar to the standard SIS model.

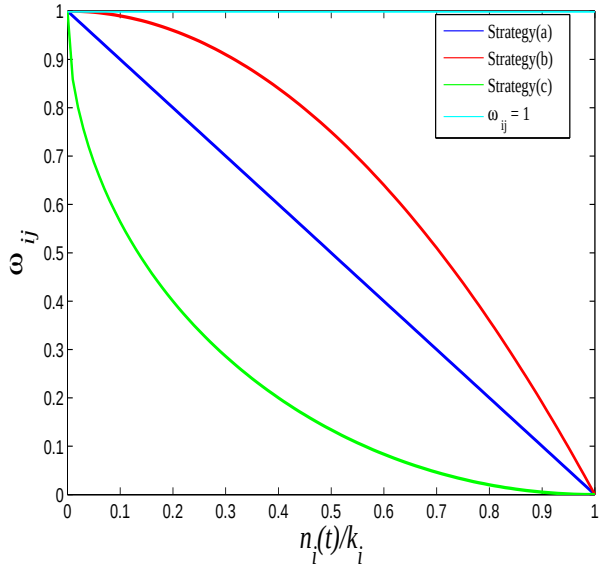


Figure 2: The interactions between weight and local infectious density under different algorithms.

It is shown in (2.3) that $\omega_{ij}(t)$ is related to the density of infected individual among the neighboring individuals at step t, and this weight adapting is shown in Figure 1. There are usually three weight adapting choices for individuals. Firstly, weight varies linearly according to the infected density among neighboring individuals, which is presented as Strategy(a). Secondly, the larger neighboring infected density is, the faster the weight decreases accordance with Strategy(b). Thirdly, in order to minimize the probability of being infected, the weights of edges are decreased significantly when the disease has not been spread wide (Strategy(c)), which can be understand as early prevention. Specially, if weight ω_{ij}=1 constantly, the model will degenerate to standard SEIS model.

3 Monte-Carlo Simulation and analysis

In this section, we generate a Monte-Carlo simulation of our proposed model based on Erdös – Rényi(ER) Random network [17]. All the results exhibited below are all based on the Mote-Carlo simulation which averages 10 iterations. The network is constructed with 500 individuals and is built as follows.

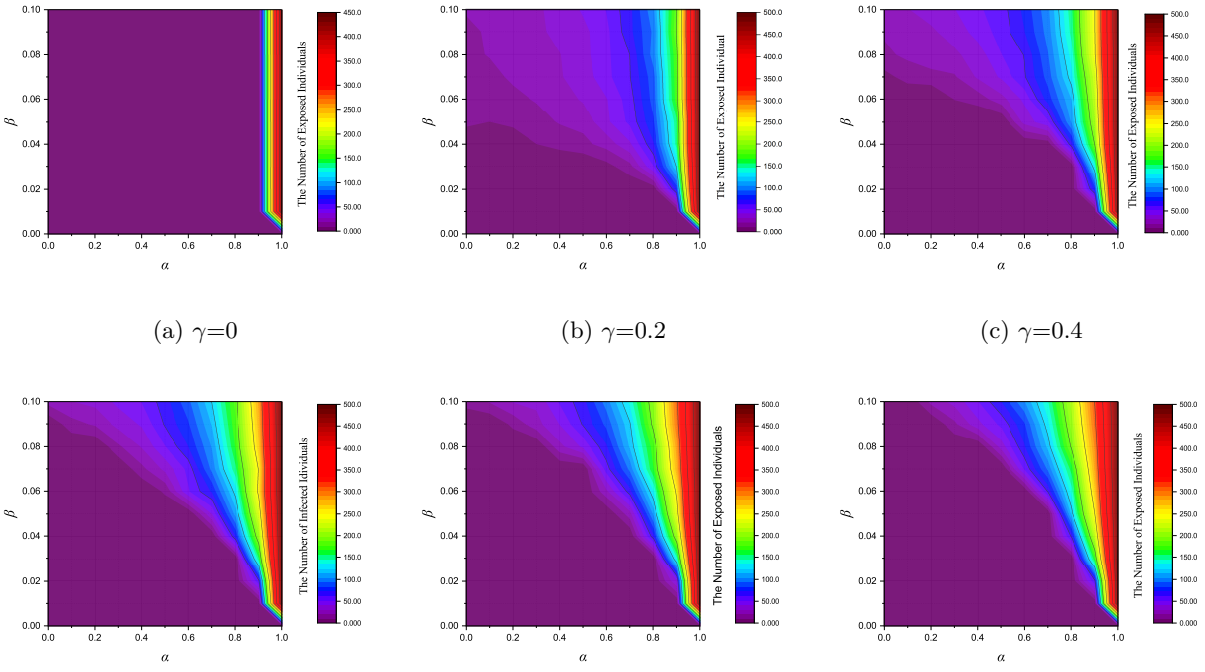
* The number of individuals in the network is set to be N=500.

* The average degree of the network is predicted as $< k > \approx 5$ as in reference [9].

* In each step, every pair of nodes in the network is connected based on the probability of p, which is determined by $p =< k > /(N-1)$.

* The initial weight of each pair of connected nodes are set to be 1 while that of unconnected pair of nodes are set to be 0.

Firstly, the number of infected and exposed individuals in steady-state under different α, β and γ are proposed in Figure 3 and Figure 4. α and γ are set in the range from 0 to 1 and β is set in the range from 0 to 0.1. We randomly choose 50 individuals who are staying in infected period when t=0 as initial infected individuals. Meanwhile, other individuals are set to stay in susceptible period as in reference [18-19].

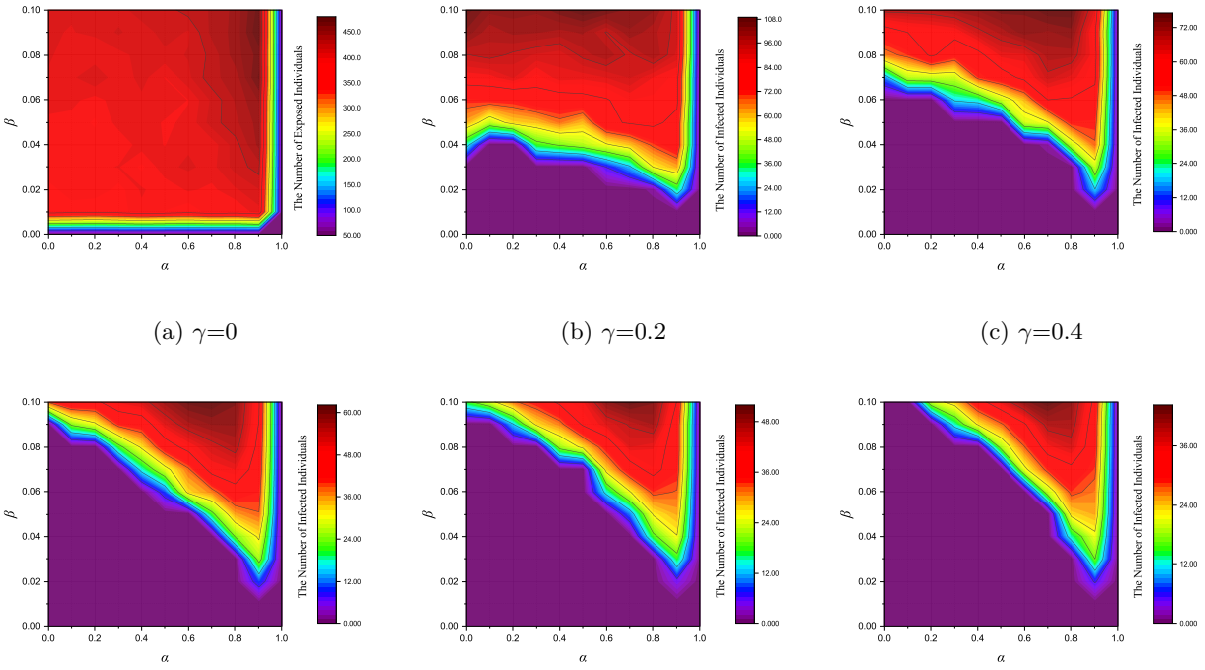


(a) γ=0 (b) γ=0.2 (c) γ=0.4

(d) γ=0.6 (e) γ=0.8 (f) γ=1.0

Figure 3: The number of exposed individuals in steady-state under different recovery rate

In Figure 3(a)-(f), it can be found that when β is close to 0, disease cannot break out under any α and γ. It is because that low infected rate inhibits disease breaking out in the network. Secondly, the threshold value of β increases with the increasing γ under any α. Specially when α=0, the model is similar to the



(a) γ=0 (b) γ=0.2 (c) γ=0.4

(d) γ=0.6 (e) γ=0.8 (f) γ=1.0

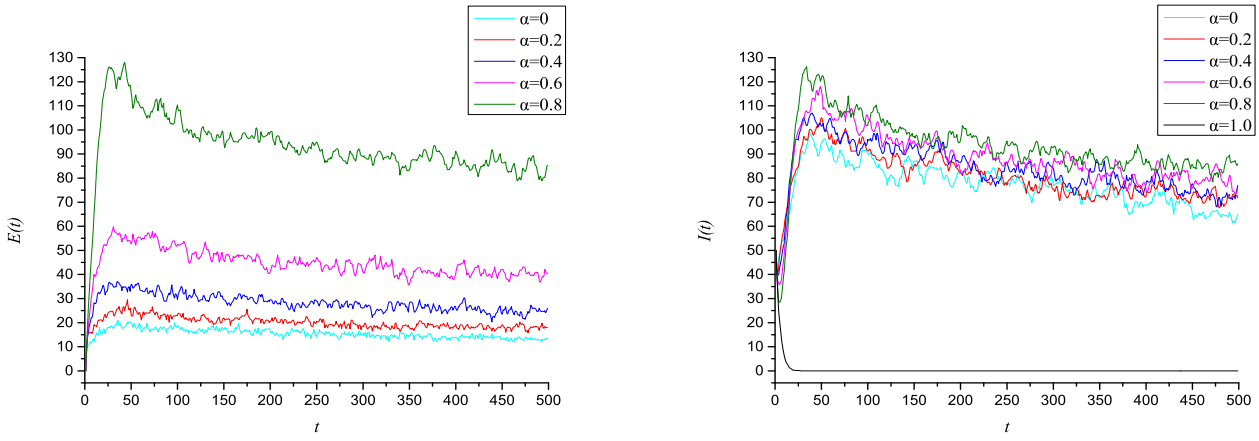
Figure 4: The number of infected individuals in steady-state under different recovery rate

SIS model, which can be explained that infected rate plays a positive effect while the recovery rate plays a negative effect on the epidemic spreading. In addition, with the increase of α and β, the number of exposed individuals increases significantly. Besides, both β and α increase the possibility of individuals staying at the exposed period. Hence, the exposed period is meaningful to this epidemic spreading model. Specially, when α=1, this model will degenerate to SI model. Meanwhile, nearly all individuals are staying at the exposed period when β>0. Finally, with the increase of γ, the area of disease cannot break out in the figure is enlarged.

In Figure 4(a) - (f), under the condition of α=1 or β=0, only those 50 initial infected individuals stay at infected period in steady-state when $\gamma = 0$. Meanwhile no individual stays at infected period when γ>0.Specially, in Figure 4(a), when γ=0, over 400 individuals among 500 will stay at infected period, unless β=0 or α=1. Generally, in Figure 4(b)-(f), the thresholds are the same as those in the exposed period which are shown in Figure 3(b)-(f). In addition, when γ=0.2, t he stable value does not have any obvious tendency with the α varying from 0 to 1 and with the constant β . This means that the increasing α can not

stimulate the epidemic spreading obviously when only a little individuals are exposed. However, there exists a tendency that the number of individuals, staying at the infected period in steady-state, increase when α is small and decrease when α is large. Besides, with the increasing recovery rate, the tendency mentioned becomes gradually obvious. And there is an inflection point between α=0.6 and α=0.8, where the number of infected individuals reaches the maximum. This is because the majority of exposed individuals cannot be revealed to infected ones, which will decrease the number of infected individuals when α is larger than the inflection point.

As a supplement, we exhibit the dynamic process of the epidemic spreading based on our model. In order to exhibit an obvious dynamic process, infected rate β is set as 0.05 and recovery rate γ is set as 0.2.



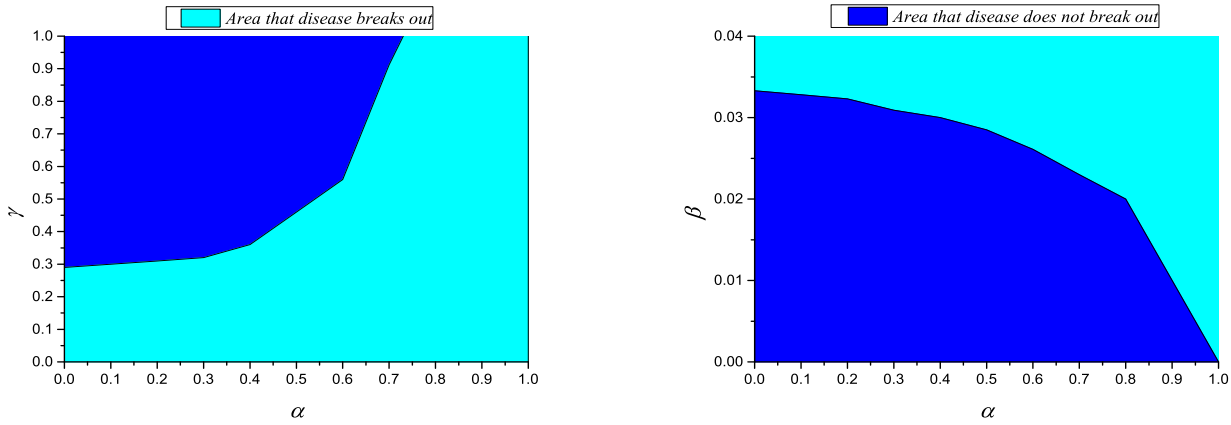
(a) Dynamic process of exposed individuals

(b) Dynamic process of infected individuals

Figure 5: Dynamic process of epidemic spreading under different α.

In Figure 5, we find that with the increasing α, the number of exposed individuals is monotonously increased while for infected individuals, the increasing α has only slight effect on the epidemic spreading. Specially, when α=1, the number of infected nodes is 0 and the number of exposed nodes approximates to the sum of individuals in the whole network (not shown in Figure 5).

Finally, thresholds of epidemic spreading with α, β, γ are shown in Figure 6. Under different values of α, β and different values of α, γ the condition that can just ensure the epidemic breaking out are proposed in Figure 6.



(a) Spreading threshold under different recovery rates and latency unrevealed rate

(b) Spreading threshold under different infected rates and latency unrevealed rate

Figure 6: The epidemic spreading threshold under different α and β

In Figure 6(a), we set infected rate β as 0.05 and recovery rate γ from 0.1 to 1 to guarantee that epidemic can spread under the latency unrevealed rate α ranging f rom 0 to 1. According to Figure 6(a), we can conclude that when α increases from 0 to 1, its variation affects slightly on the epidemic spreading when latency unrevealed rate α<0.4, while its contribution increases when α>0.4. Specifically, there is a critical point around $\alpha = 0.6$. When α>0.6, γ increases obviously. In addition, when α is large enough, even each infected individual getting recovered under the probability of 1, the epidemic spreading cannot be inhibited. This also validates the assumption that when α is large, it contributes largely to the epidemic spreading.

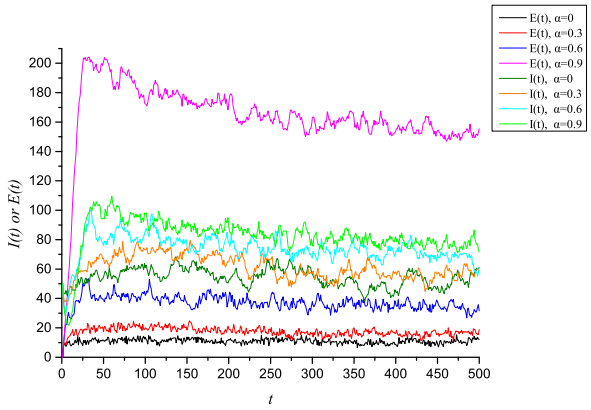
In Figure 6(b), the relationship between the infected rate β and latency unrevealed rate α in threshold condition is proposed. We set α from 0 to 1 and β from 0 to 0.1. Such small values are set in order to guarantee that the threshold can vary in an obvious tendency with the γ=0.2. We consider the coefficient between the infected rate β and the latency unrevealed rate α separately and find out that with increasing α, the infected rate β drops down rapidly. Particularly, when α=1, the disease will always breaks out, regardless how small the infected rate β is. This can also be explained by what we stated above.

Combing Figure 6(a) and Figure 6(b), we can find out that with the increasing α, the promotion of epidemic spreading is enlarged. Especially, when α is large enough, the spreading cannot be inhibited under any recovery rate with β=0.05. Furthermore, when α=1, the disease will break out even under the condition that $\beta \to 0_+$ or γ=1. It is considered that all individuals can only stay at the exposed period and the transition among S, E, I stops when α=1.

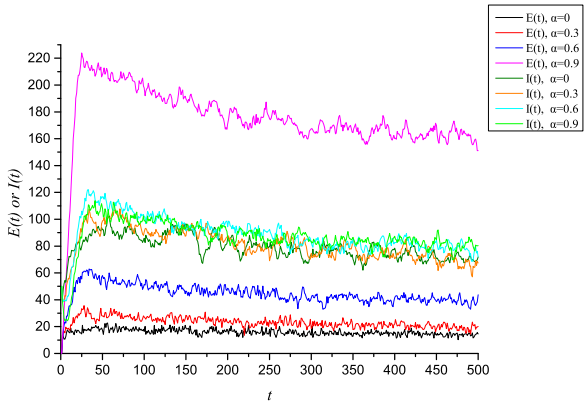
4 Strategies of weight adopting

Apparently, the simulation results above are all based on the weight adapting strategy, which the numerical relationship can be described as $\omega_{ij}(t) = 1 - k_i/n_i(t)$. However, it is unreasonable to keep the rate of variation in constant, regardless of the infected density. Hence, Strategy(b) and Strategy(c) are proposed based on different rate of weight variation. Hence, in this part, we focus on the effect of different weight adapting Strategies on epidemic spreading. Besides, in this part we set the infected rate β=0.05, recovery rate γ=0.2 and latency unrevealed rate α from 0 to 1 as reasonable ranges for the three variables .

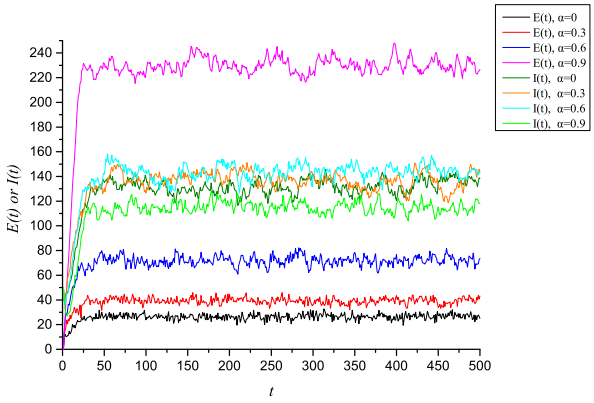
In Figure 7, it is obvious that Strategy(a)-(c) can inhibit epidemic spreading more obviously compared with $\omega_{ij}(t) = 1$, which verifies that the the control of weight adapting strategy is valid. By analyzing Strategy(a)-(c), it is found that (a) and (b) contribute almost the same to the epidemic spreading while the Strategy(c) dramatically decreases the number of infectious individuals and inhibits the spreading. Besides, there are some other details need to be noticed. Specifically, in all these three strategies, the number of infected period all varies slightly around 80 with a large α. Under such condition, our strategies have little effect on the infected state. In other words, when the number of exposed individuals is large and that of infected individuals is small, our adaptive weights have little effect on epidemic spreading. In addition, under Strategy(c), the value of infected individuals and exposed individuals at steady-state are all 0 when α=0 and α=0.3. In such condition, Strategy(c) inhibit the epidemic spreading in both SIS and SEIS model significantly compared with the other two strategies.



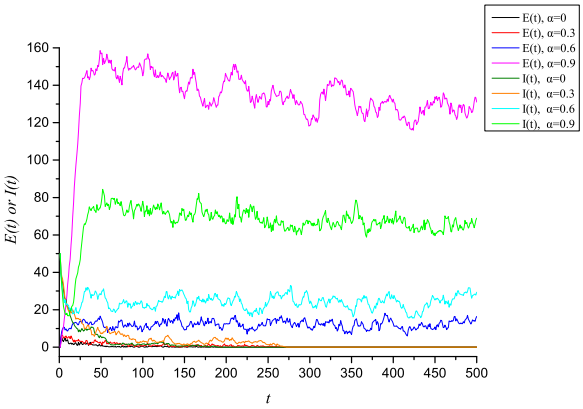
(a) $\omega_{ij}(t)$ =1



(b) algorithm(a)



(c) algorithm(b)



(d) algorithm(c)

Figure 7: The dynamic process of epidemic spreading under different weight adapting.

5 Conclusion

In this paper, a novel adaptive weighted network model is proposed. Firstly, we describe our model in a mathematical approach. Secondly, the Monte-Carlo simulation of our proposed model based on ER Random network is conducted. Finally, different strategies for weights adapting are proposed and the inhibiting effects of those algorithms on epidemic spreading are compared.

It is found that when the recovery rate γ is small, its increase has a positive effect on individuals staying in infected period. Meanwhile its increase has a negative effect on that when the latency unrevealed rate α is large. However, the larger the α is, the more obviously it contributes to epidemic spreading. Moreover, in the threshold condition, α affects both γ and β obviously when it is large. Especially, when α is approximate to 1, the disease can spread out under any γ and β except 0. Finally, we find out that Strategy(c) inhibits epidemic spreading effectively.

References

[1] Barabási A L, Albert R, Jeong H. Mean-field theory for scale-free random networks[J]. Physica A: Statistical Mechanics and its Applications, 1999, 272(1): 173-187.

[2] Mhatre V, Rosenberg C. Homogeneous vs heterogeneous clustered sensor networks: a comparative study[C]//Communications, 2004 IEEE International Conference on. IEEE, 2004, 6: 3646-3651.

[3] R.Pastor-Satorras, A.Vespignani, Phys.Rev.Lett 86(2001) 3200.

[4] R.Olinky, L.stone, Phys.Rev.E 70(2004) 030902.

[5] Haifeng Zhang, Xinchu Fu, Nonlinear Analysis 70(2009) 3273-3278.

[6] Jiancong Chen, Huiling Zhang, Zhihong Guan, Tao Li, Phys.A 391(2012) 1848-1854.

[7] Null N. The SEIS model, or, the contact process with a latent stage[J]. Medical Journal of Australia, 1968, 1(4):133-5.

[8] Wu Q, Zhang F. Threshold conditions for SIS epidemic models on edge-weighted networks[J]. Physica A: Statistical Mechanics and its Applications, 2016, 453: 77-83.

[9] Huang Y, Ding L, Feng Y. A novel epidemic spreading model with decreasing infection rate based on infection times[J]. Physica A: Statistical Mechanics and its Applications, 2016, 444: 1041-1048.

[10] Sanders J, Noble B, Van Gorder R A, et al. Mobility matrix evolution for an SIS epidemic patch model[J]. Physica A: Statistical Mechanics and its Applications, 2012, 391(24): 6256-6267.

[11] Muroya Y, Enatsu Y. A discrete-time analogue preserving the global stability of a continuous SEIS epidemic model[J]. Journal of Difference Equations and Applications, 2013, 19(9): 1463-1482.

[12] Xu R. Global dynamics of an SEIS epidemiological model with time delay describing a latent period[J]. Mathematics and Computers in Simulation, 2012, 85: 90-102.

[13] Liu J, Wei F. Dynamics of stochastic SEIS epidemic model with varying population size[J]. Physica A: Statistical Mechanics and its Applications, 2016, 464: 241-250.

[14] Bowong S, Kurths J. Parameter estimation based synchronization for an epidemic model with application to tuberculosis in Cameroon[J]. Physics Letters A, 2010, 374(44): 4496-4505.

[15] Feng Y, Ding L, Huang Y H, et al. Epidemic spreading on weighted networks with adaptive topology based on infective information[J]. Physica A: Statistical Mechanics and its Applications, 2016.

[16] Arenas A, Borge-Holthoefer J, Meloni S, et al. Discrete-time Markov chain approach to contact-based disease spreading in complex networks[J]. EPL (Europhysics Letters), 2010, 89(3): 38009.

[17] P.Erdös, A.Rényi, Publ.Math.Debrecen 6(1959) 290.

[18] Li K, Yu H, Zeng Z, et al. Epidemic transmission on random mobile network with diverse infection periods[J]. Communications in Nonlinear Science and Numerical Simulation, 2015, 22(1): 596-604.

[19] Zhang J, Sun J. Stability analysis of an SIS epidemic model with feedback mechanism on networks[J]. Physica A: Statistical Mechanics and its Applications, 2014, 394: 24-32.