

## The spread of epidemic under voluntary vaccination with heterogeneous infection rates

Qingfang Chen, Hong Ding, Yixuan Yang, Jingrui Wang, Xing Jin\*

*School of Cyberspace, Hangzhou Dianzi University, Hangzhou 310018, China*

Received Day Month Year  
 Revised Day Month Year

Epidemics are usually spread widely and can cause plenty of losses to humans. In real society, vaccination is the principal method to suppress the spread of infectious diseases. The SIS (Susceptible-Infected-Susceptible) model is proposed, which considers that the presence of voluntary vaccination may affect the spread of the epidemic. In most studies to date, it has been considered that the infection rate of nodes in the SIS model is not heterogeneous. However, in reality, there exist differences in the neighbor network structure and the number of contact, which may affect the spread of infectious diseases in society. As a consequence, it can be reasonably assumed that the infection rate of the nodes is heterogeneous because of the number of contact among people. Here we propose an improved SIS model with heterogeneity in infection rates, which is proportional to the degree of nodes. By conducting simulations, it is illustrated that the almost all vaccinated nodes have high degrees, when the infection rate is positively correlated with the degree of the nodes. Based on the role of the vaccinated node in the network, it is reasonable to assume that the vaccinated node can be regarded as removing the network. Hence these nodes can divide network into many connected sub-graphs which make the epidemic fall more quickly, the heterogeneity of infection rates has a strong inhibitory effect on the epidemic transmission. On the opposite side, when the infection rate is negative related to the degree of the infection rate nodes, it is difficult for most nodes to meet the inoculation conditions, and the number of inoculations close to none.



*Keywords:* SIS model; Dynamical system; Heterogeneous infection rate; Individual vaccination.

PACS Nos.:

### 1. Introduction

Large-scale infectious diseases, which can be spread directly or indirectly from person to person, usually cause blocky losses to human societies. For example, the Black Death [1–3] caused about 25 million deaths in the 14th century, and especially now COVID-19 [4–7] outbreak has caused varying degrees of social panic. On March 11, 2020, the World Health Organization officially announced at the press conference that COVID-19 was listed as a global pandemic. As of June 1, 2020, approximately

\*Corresponding author. Email: jinxing@hdu.edu.cn(Xing Jin)

2 *Qingfang Chen & Hong Ding & Yixuan Yang & Jingrui Wang & Xing Jin*

6 million people worldwide have been diagnosed with infections, including approximate 400,000 deaths. The current number is still rising rapidly. Since the outbreak of the virus, many scientists have studied the spread of COVID-19. Fang et al. [8] gave theoretical results about epidemic threshold and influence of isolation factor. Cooper et al. [9] verified the effectiveness of proper restrictions and strong policies for disease control. This illustrates the importance of studying the spread of infectious diseases. As a result, how to control the spread of the epidemic more effectively becomes a key topic for many research scholars.

To curb the spread of the virus, vaccines have been invented by medical scientists, which has been widely recognized as the most effective way to suppress the spread of many epidemics. In addition to the therapeutic use of vaccines, many researchers have noted that different vaccination strategies can change the spread of epidemics. To study the impact of different immune strategies on epidemic transmission, research scholars proposed different immunization strategies based on different vaccination points in the selection network such as random immunity [10], target immunity [11] and acquaintance immunity [12]. Generally speaking, these aforementioned immunization strategies are based on compulsive vaccination. However, compulsive vaccination is not realistic for several reasons: religion, the cost of vaccination, and the side effects of vaccination. As a consequence, people will weigh their interests and decide whether to get vaccinated. Therefore, voluntary vaccinations has been proposed to explore the dynamical process in epidemics.

In the line of voluntary vaccinations, Zhang et al. [13] proposed the SIS model to simulate voluntary vaccination and found that the voluntary immunization strategy inhibited the spread of the epidemic in a scale-free network. On the basis of the SIS model, Xue et al. [14] studied the susceptible individuals to estimate the risk through direct neighbors and choose whether to vaccinate, which can effectively inhibit the spread of the epidemic. Lu et al. [15] considered that the vaccinated individuals were not infected and proposed the SIR (Susceptible-Infected-Removed) model, and this research showed that whether vaccinated or not, the consciousness of vaccination can effectively increase the threshold of disease transmission and slow the speed of disease transmission. On the basis of SIR model, Rao et al. [16] showed that not only vaccination can eradicate disease but also treatment rate even in presence of time delays would contain the disease. Besides these, some research scholars [17–19] also studied the role of individual mimicry behaviors and population structures in vaccination.

In the actual situation, the environmental conditions, health habits, and physical condition of individuals survival are different, and the infection rate varies from individual to individual. By considering the different probability of epidemic infection at different ages (such as youths, adults), Cai et al. [20] proposed the SVIR (Susceptible, Vaccinated, Infected, and Recovered populations) model to prove that the heterogeneity of infection rates can prevent or accelerate the spread of the epidemic. However, Cai did not consider that the impact of contacts on the disease. Inspired by these aspects, we want to discuss the impact of node infection rates on

disease transmission based on the number of contacts, which we aim to address in what follows.

According to the above considerations, in this paper, we propose an improved model based on voluntary vaccination, the heterogeneity of the infection rate is determined by the adjustable variable  $\tau$ , which indicates the degree of correlation between the node infection rate and its own degree. Then we discuss the possible effects of changes in the  $\tau$  value on the spread of epidemic in our model. Scale-free network is used for numerical simulations to study the effects of the heterogeneity of infection rates on the spread of epidemic. It is found that: (1) when  $\tau = -1$  (node infection rate is negative correlated with its own degree), the vaccination conditions of the edge nodes are harsh, the central nodes cannot meet the vaccination conditions, and most of them are unwilling to be vaccinated; (2) when  $\tau = 1$  (node infection rate is positively correlated with its own degree), the central node is more willing to be vaccinated, as a result, the average degree would decrease, thereby effectively limits the spread of the epidemic.

## 2. Model

We describe the spread of epidemic through the SIS epidemiological model. In the SIS model, the node status can be divided into susceptible (S) and infected (I).  $V_s$  and  $V_I$  are used to represent the susceptible nodes and the infected nodes, respectively. If node  $i \in V_S$  and node  $j \in V_I$  are bordered, it is assumed that the node  $i$  will be infected by node  $j$  with a probability  $\beta$ . Consequently, the overall probability  $\lambda_i$  that node  $i$  to be infected by its neighbors can be calculated as follows:

$$\lambda_i = 1 - (1 - \beta)^{f_i} \quad (1)$$

where  $f_i$  means the number of infected neighbors of node  $i$ . After being infected, each patient has a probability  $\mu$  to restore to a susceptible status at each time step. In this paper, we set  $\mu = 1$  to simplify the model without loss of generality [13].

In real life, the infection rate varies greatly between individuals due to different health habits, physical strengths and social conditions between individuals. In order to realistically simulate the spread of epidemic in the world, each node with heterogeneous infection rate has been considered in our model. Because the characteristic of nodes can only be represented by the degree of the node itself in our network structure, we will make the heterogeneous infection rate of the node correlated with the degree of the node itself. In order for the heterogeneous infection rate properly changes which is based on the standard infection rate (The standard infection rate can be understood as the average infection rate of the population, and we will use  $\beta$  to mean this), and we have the following requirements for the infection rate: (1) The trend of infection rate with degree should be influenced by average degree, to ensure the maximum infection rate and the minimum infection rate are appropri-



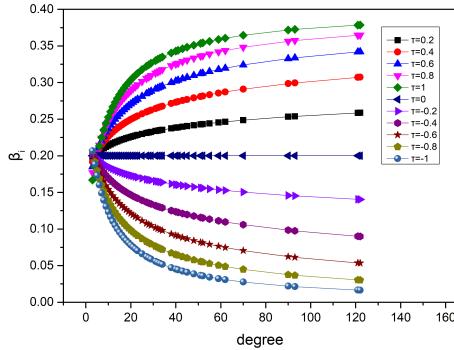


Fig. 1: the node's infection rate  $\beta_i$  for different degrees in the BA network defined above ( $\beta = 0.2$ ).

ate. (2) The curve of infection rate should be smooth and consistent with a certain distribution. Specifically, the node's infection rate can be re-defined as [21]:

$$\beta_i = \frac{2(k_i + 1)^\tau \beta}{\langle (k + 1)^\tau \rangle + (k_i + 1)^\tau} \quad (2)$$

when  $\langle (k + 1)^\tau \rangle = \sum_k (k + 1)^\tau p(k)$ ,  $p(k)$  is the degree distribution of each node with degree  $k$ , and  $k_i$  is node  $i$ 's degree. And  $\tau$  is introduced as the power exponent to adjust the infection rate. Because the  $\tau$  effect on the infection rate can be fully reflected in  $-1$  to  $1$ , hence  $\tau$  is arranged from  $-1$  to  $1$  in our model.

With the change of  $\tau$  value, the correlation between the node infection rate and its own degree will also change. Normally, the meaning of different correlations in reality can be understood as follows: (1) If the infection rate is positively correlated with the degree of the node, individuals who live in central cities are more likely to be infected, because they have a lot of contact with other individuals; (2) Oppositely, the infection rate can also be negatively correlated with the degree of the node, which can be explained as individuals who live in central cities have a better chance of vaccinating and medical conditions.

Before experimentally studying the heterogeneous infection rate  $\beta_i$ , we need to discuss social networks' structures which determine the degree distribution of each node. More recently, research has illustrated that the degree distributions of many social networks are not Poissonian, but follow a power law [22]:  $P(k) \sim k^{-\gamma}$  where  $\gamma$  is the degree exponent of the scale-free BA (Barabási Albert) network. Hence, we will choose the BA network to simulate the real world network in this paper. Specifically, it is set that the BA network has  $N = 2000$  and the average degree  $\langle k \rangle = 6$ . And the change relation between degree and  $\beta_i$  under different  $\tau$  values is shown in Fig. 1.

By observing Fig. 1, we can get the following three findings:

- 1) When  $\tau = 0$ , the infection rate of each node is equal to  $\beta$ . It means that everyone is equally likely to be infected and there is no individual difference.
- 2) When  $\tau > 0$ , the infection rate of each node is positively correlated with the degree of node itself. The positive correlation also increases with the increase of  $\tau$ .
- 3) When  $\tau < 0$ , the infection rate of each node is negatively correlated with the degree of node itself. Not surprisingly, the negative correlation increases with the decrease of  $\tau$ . The change relation between  $\beta_i$  and the degree of nodes conform to the characteristics of the power law distribution.

Consequently, the overall infection rate of the node  $i$  can be re-defined by substituting  $\beta_i$  into Eq. (1):

$$\lambda_i = 1 - (1 - \beta_i)^{f_i} \quad (3)$$

Next, we will discuss the process of rational individuals making a decision to vaccinate in terms of the heterogeneous infection rates. Generally, rational individuals who always seek personal interests will attempt to minimize their own costs of vaccination. To mimic the behavior of rational individuals in taking vaccinations, firstly, we define the cost function [13, 23] of each strategic behavior, and then employ game theory to model the decision-making process of each rational individual. Specifically, the expense of the node  $i$ 's behaviors can be described by the cost of vaccination ( $M_v$ ) and the cost of non-vaccination ( $M_{ni}$ ):

$$\begin{cases} M_{ni} = c_1 \lambda_i \\ M_v = c_2 \end{cases} \quad (4)$$

where  $c_1$  is the parameter related to the loss of been infected and  $c_2$  is the parameter related to the cost of vaccination ( $c_1 > 0, c_2 > 0$ ). In this proposed model, the behavior of a rational individual is determined by the relationship between  $M_{ni}$  and  $M_v$ : individual will vaccinate when  $M_{ni} > M_v$  is satisfied, otherwise, individual will not vaccinate. As same as susceptible nodes and infected nodes, we use  $V_D$  to denote the vaccinated nodes. If the node  $i \in V_D$ , it cannot be infected by others any more. Due to various vaccines have a different period of validity, to simplify our model, the validity of various vaccines discussed are assumed to be permanent in this paper [13].

Without loss of generality, the epidemic propagation can be modeled as a discrete time step process. Firstly, we randomly choose some infected nodes to initialize the epidemic propagation. Next, the following four procedures are used to illustrate the process of the epidemic propagation in one time step:

6 *Qingfang Chen & Hong Ding & Yixuan Yang & Jingrui Wang & Xing Jin*

- (1) Based on the expense, synchronously update whether all the node  $i \in V_S$  wants to be vaccinated.
- (2) Put the node that wants to be vaccinated into  $V_D$ .
- (3) Synchronously update whether all the node  $i \in V_S$  to be infected.
- (4) The infected node recovers to the susceptible status, that is, put the node  $i \in V_I$  into  $V_S$ .

Specifically, the pseudo-code of the epidemic propagation is presented in Algorithm 1.

---

**Algorithm 1** Simulate the epidemic propagation

---

```

1: Initialize a BA network and build the  $V_I$  set by randomly selecting five infected
   nodes. #Initialize network structure and  $V_I$ 
2:  $I[n] = \emptyset$  and  $S[n] = \emptyset$  and  $D[n] = \emptyset$ 
3: Set  $V'_I = V_I$  # $V'_I$  is used as the temporary set to obtain the set of new infected
   nodes in each time step
4: for each time step  $t \in [0, n]$  do
5:   for each node  $i \in V_S$  do
6:     Calculate  $\lambda_i$  based on Eq. (2) and calculate  $M_{ni}$  based on Eq. (4)
7:     if  $M_{ni} > M_v$  then
8:       Set  $V_D = V_D \cup i$  and  $V_S = V_S \setminus i$ 
9:       continue
10:    else
11:      Random a number  $P$  between 0 and 1
12:      if  $P < \lambda_i$  then #It means the node  $i$  is infected
13:        Set  $V'_I = V'_I \cup i$  and  $V_S = V_S \setminus i$ 
14:      end if
15:    end if
16:  end for
17:  #The recovery process of infected nodes
18:  for each node  $j \in V_I$  do
19:    Set  $V'_I = V'_I \setminus j$  and  $V_S = V_S \cup j$ 
20:  end for
21:   $V_I = V'_I$  #Update the set of infected nodes and network states
22:   $I[t] = |V_I|$  and  $S[t] = |V_S|$  and  $D[t] = |V_D|$  #Record the number of nodes in
   different states at time step  $t$ 
23: end for
24: return  $I[n]$ ,  $S[n]$  and  $D[n]$  #Get three arrays that represent the spread of
   epidemic

```

---

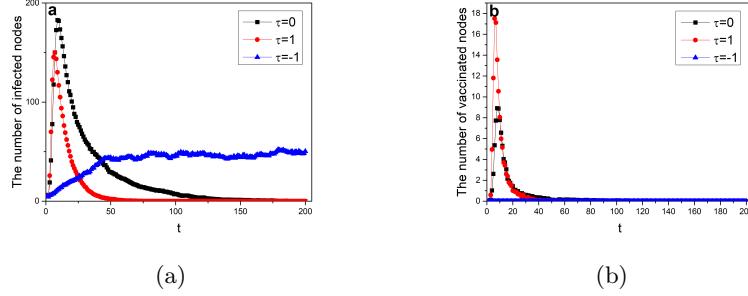


Fig. 2: The spread of epidemics in BA network with the different settings of  $\tau$ : (a) the number of infected individuals without voluntary vaccination versus time, (b) the number of vaccinations versus time.

### 3. Result

Firstly, we study the impact of  $\tau$  on the spread of epidemics in the BA network. Specifically, we tune  $\tau$  in the range of  $\{-1, 0, 1\}$ . Three values  $\{-1, 0, 1\}$  are respectively expressed as: the infection rate has a positive, no, and negative correlation with the degree of the node. And, all the results is averaged over 50 instances.

The parameters that have been used in experiments are exhibited in Table 1. In order for the epidemic to spread at a proper speed, we set the infection rate  $\beta$  to be 0.2 [13]. To simplify the model without loss of generality, the recovery rate  $\mu$  is set to be 1 [13]. In most cases, it costs less to prevent disease than to cure disease, hence the cost  $c_2$  is smaller than the cost  $c_1$  which is normally set as 1 [13]. In order to be consistent,  $c_2$  is fixed to be 0.7 as an instance. We will give an explicit statement when the value of a parameter is modified.

Table 1: Parameters setting

| Parament | Commentate  | Value |
|----------|---|-------|
| $\beta$  | Infection rate  | 0.2   |
| $\mu$    | The recovery probability of restoring to a susceptible status | 1     |
| $c_1$    | The loss of been infected                                     | 1     |
| $c_2$    | The cost of vaccination                                       | 0.7   |

As shown in Fig. 2, it is found that: (1) When  $\tau = -1$ , almost no node chooses to vaccinate, and the number of infected individuals remains at a small value along with the time step; (2) When  $\tau = 1$ , the maximum number of infected individuals is smaller than the maximum of infected individuals under the case of the non-heterogeneity infection rate ( $\tau = 0$ ), and the disease seems subsiding faster. As

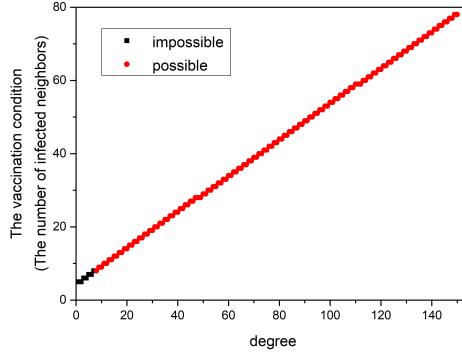


Fig. 3: Vaccination conditions of nodes with different degrees in the BA network.

shown below, we will discuss and explain these two mentioned findings.

### 3.1. When $\tau$ is set to be -1, almost no one is vaccinated in the BA network

According to the proposed model with heterogeneous infection rates, the influence of individual's willingness to vaccinate is determined by the relationship among the parameters  $c_1$ ,  $c_2$ , and  $\lambda_i$ .

Based on the expense of each behavior, we can determine the vaccination conditions of each node. According to Eq. (4), it is illustrated that the node  $i$  will take the vaccination behavior if the following equation is satisfied:

$$\begin{aligned}
 M_{ni} &> M_v \\
 c_1 \lambda_i &> c_2 \\
 1 - (1 - \beta)^{f_i} &> \frac{c_2}{c_1} \\
 f_i &\geq \lfloor \log_{1-\beta_i} \frac{c_1 - c_2}{c_1} \rfloor + 1
 \end{aligned} \tag{5}$$

Accordingly, we can find out the necessary condition for each node to vaccinate in the BA network: the number of infected neighbors of each node  $f_i$  should be greater than a threshold.

As shown in Fig. 3, by conducting simulations in the BA network, it is found that the nodes with low degrees ( $k \leq 7$ ) are possible to vaccinate. Moreover, the vaccination condition of nodes with high degrees ( $k > 7$ ) is also impossible to reach.

We can use Eq. (5) to explain the above finding. As can be seen from Fig. 1, it is gotten that  $\beta_i$  is negatively correlated with the degree of node when  $\tau = -1$ , therefore,  $1 - \beta_i$  increases with the increase of the degree of node  $i$ . By the properties of the logarithm function, because  $\frac{c_1 - c_2}{c_1} = 0.3 < 1$ , the vaccination condition  $f_i$

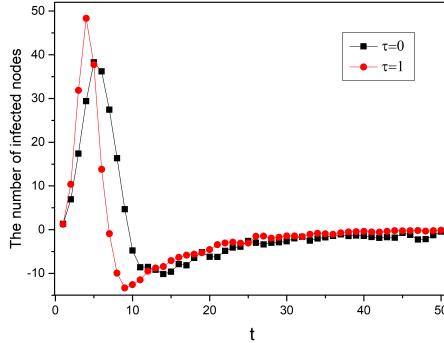


Fig. 4: The number of infected individuals between adjacent time steps.

becomes bigger as the degrees of the nodes increase, until the vaccination condition becomes impossible. Specifically, it is found that the vaccination condition of the node is greater than the degree of the node itself when the node with high degrees ( $k > 7$ ).

Overall, from the description above, we can know that: when  $\tau$  is set to be -1, the nodes with small degrees ( $k \leq 7$ ) cannot reach the vaccinating condition and the vaccination condition of nodes with big degrees ( $k > 7$ ) is also impossible. Hence almost no node to vaccinate in the BA network, i.e., the finding 1 is proved.

### 3.2. When $\tau$ is set to 1, the regression of epidemic in the BA network is accelerated

As seen from Fig. 2, it is directly observed that when  $\tau = 1$ , the epidemic declines earlier than the case when  $\tau = 0$ . In order to confirm this mentioned observation, we plot the difference of infected individuals between adjacent time steps with respect to  $\tau = 0$  or  $\tau = 1$ . Due to the difference number of infected individuals almost converges to 0 at time step 50 when  $\tau$  is fixed as 1. Therefore, the first 50 time steps are selected to plot, and the result is shown in Fig. 4.

As can be seen from Fig. 4, when  $\tau = 1$ , the number of infected nodes begins to decline at the 7<sup>th</sup> time step which is earlier than the case of without heterogeneity (i.e.,  $\tau = 0$ ). Moreover, when the number of nodes without heterogeneity begins to decline, the decline of patients with heterogeneity has reached its peak and its peak is still higher than that of  $\tau = 0$ . The decline trend of  $\tau = 1$  begins to decrease after the peak period, but it also remains almost the same with the trend of  $\tau = 0$ . After analyzing Fig. 4, it is confirmed that the epidemic will subside faster when  $\tau = 1$ .

Before discussing the reasons for the second finding, we need to discuss what factors influence the epidemic propagation at first. In our model, the propagation of epidemic can be expressed as the transmission between nodes' statuses according to: (1) the vaccinated nodes and (2) the infection rate  $\beta_i$ . With the aim to identify

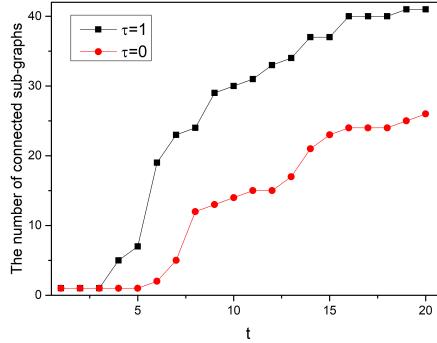


Fig. 5: The number of connected sub-graphs in a network

the reasons for the decline of infected nodes, the effect of these two factors should be thoroughly studied. Firstly, let's discuss how the ~~vaccinated nodes affect the propagation of epidemic~~. Since the vaccinated node cannot be infected by its neighbors, we can assume that the vaccinated node does not play any role in the epidemic transmission, although it still exists in the network. Hence we can make the following assumptions: ~~it is considered that~~ the vaccinated node and the edges connected to it are removed from the network in term of the epidemic transmission. As a result, the network can be divided into connected sub-graphs. Next, we discuss the effect of the connected sub-graphs on the epidemic transmission.



As seen from Fig. 5, it is observed that the number of connected sub-graphs tends to stabilize after the 15<sup>th</sup> time step in the transmission of the disease. We can find that the number of connected sub-graphs generated in the network when  $\tau = 1$  is more than that generated when  $\tau = 0$ . Combining with Eq. (5), this aforementioned finding can be explained as: the infection rate of the nodes is positively correlated with its own degree when  $\tau = 1$ , hence the nodes with high degrees have higher infection rates than  $\tau = 0$ , thereby improving the probability of the nodes with high degrees to vaccinate.

Subsequently, let's move on to the effects of connected sub-graphs on the epidemic transmission. Due to the fact that, nodes are more likely to establish a connection with the node of high degree when they join the BA network. Therefore, the connected sub-graphs formed in the network almost contain few nodes after the nodes with high degrees are vaccinated, which will effectively limit the epidemic transition from nodes to nodes. Overall, it is illustrated that the increase in the number of connected sub-graphs can indeed inhibit the spread of diseases in the network.

Based on the above analysis, most connected sub-graphs formed by the vaccinated nodes removed from the BA network are graphs with a few nodes, in which the epidemic is impossible to spread constantly. Therefore, in order to investigate

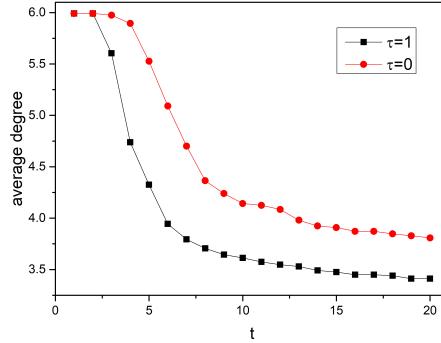


Fig. 6: The average degree of the connected sub-graph with the maximum number of nodes

the spread of the epidemic in the BA network, we should pay our attention to the sub-graph with the most nodes. Next, we will discuss the spread of epidemic in terms of the average degree of this connected sub-graph.

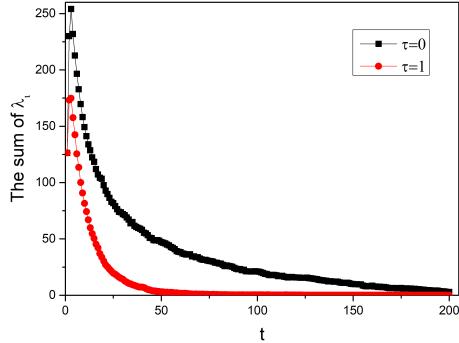
As seen from Fig. 6, it is observed that the average degree declines faster when  $\tau = 1$ . And the average degree with respect to  $\tau = 1$  becomes 3.5 which is lower than the case of  $\tau = 0$ , when the network structure tends to be stable. Because of the lower average degree, the network is more effectively curb the spread of epidemic than  $\tau = 0$ .

The reason for the decrease in average degree can be explained as that there are more central nodes to take vaccination. When  $\tau = 1$ , the infection rate  $\beta_i$  is proportional to node's degree, hence, in the course of epidemic transmission, central nodes with higher degrees are easier to vaccinate than  $\tau = 0$ . Moreover, according to Eq. (5), the condition of vaccination requires  $f_i$  to be 7 when  $\tau = 0$ . But the vaccination conditions of nodes would decrease significantly when  $\tau = 1$  as shown in Table 2, thereby, the central nodes are easier to choose vaccination when  $\tau = 1$ , so as to make the average degree be lower than  $\tau = 0$ .

Table 2: The condition of vaccination  $f_i$  when  $\tau = 1$

| The degree $k$ of node | The condition of vaccination $f_i$ |
|------------------------|------------------------------------|
| $1 \leq k \leq 31$     | 6                                  |
| $32 \leq k \leq 111$   | 4                                  |

After discussing the influence of **vaccinated nodes** on disease transmission, we will further analyze the role of  $\beta_i$ . If  $\beta_i$  is discussed independently, due to  $\beta_i$  repre-

Fig. 7:  $\sum \lambda_i$  in each time step

sents node  $i$ 's probability of being infected by an infected neighbor, we cannot show how the disease spreads by analyzing  $\beta_i$ . Therefore, We analyze  $\sum \lambda_i$  at each time step to determine the impact of  $\beta_i$  on the epidemic transmission.

As seen from Fig. 7,  $\sum \lambda_i$  is smaller when  $\tau = 1$  at each time step. It means that the epidemic transmission trend is weaker when  $\tau = 1$ . Next, we explain the reason for this finding. From Fig.1, we can get that  $\beta_i$  is greater than 0.2 only when the degree of node  $i$  is 1. These nodes are more likely to establish a connection with the node of high degree when they join the BA network. Since the nodes of high degree are almost all vaccinated when  $\tau = 1$ , the nodes of  $k = 1$  cannot be infected, hence the actual  $\beta_i$  of nodes involved in epidemic transmission is smaller. Therefore,  $\beta_i$  inhibits the epidemic transmission when  $\tau = 1$ .

Overall, from the above discussion, we can know that: when  $\tau = 1$ , the number of connected sub-graphs increases and the average degree of the connected sub-graph with the most nodes decreases during the dynamic of the epidemic, which simultaneously inhibit the spread of the epidemic. And the actual  $\beta_i$  of nodes involved in epidemic transmission is smaller when  $\tau = 1$ . Hence, the finding 2 is proved.

#### 4. Conclusion and discussion

This paper considers the differences in individual vaccination intentions and introduces heterogeneity of infection rates in the SIS model. We perform numerical simulations using a scale-free network and used different  $\tau$  values to study the differences in the spread of the epidemic in BA networks. Specifically, different values of  $\tau$  change the correlation between the infection rate of node and its degree.

By analyzing the experimental results, we get the following two findings : (1) Almost no one is vaccinated in the BA network when  $\tau = -1$ . By analyzing the vaccination condition, it is found that the condition of nodes with small degrees ( $k \leq 7$ ) is very harsh: all neighbors need to be infected. And the vaccination condition of nodes with high degrees ( $k > 7$ ) is impossible to reach. Therefore, the vaccination

condition is not conducive to the vaccination for nodes of all degrees when  $\tau = -1$ ; (2) The regression of epidemic in the BA network is accelerated when  $\tau = 1$ . We can get that the network forms more connected sub-graphs during the dynamic of the epidemic. And only one of these connected sub-graphs is capable of spreading the epidemic. We use  $\beta_i$  and the average degree to analyze it, and found that the probability of epidemic spread on this connected sub-graph is lower than non-infectious rate heterogeneity.

Overall, it can be seen that when the heterogeneity of the infection rate is positively correlated with the degree of node itself, it has a better influence on inhibiting the spread of the epidemic. In the real society, the individuals who are exposed to the epidemic, such as doctors and service workers, are more likely to be vaccinated and will have an inhibitory effect on the spread of the epidemic.

However, due to the complexity of human behavior and social behavior, there is a lot of work that needs to be studied in depth. This paper assumes that the anti-infective and resilience capabilities of each node are the same, however in reality, due to differences among individuals, some individuals may have low anti-infective ability and low resilience, and they can change the spread of the epidemic. In the real society, individual's vaccination is also affected by the media, etc. When individuals are delayed due to the news, the periodic outbreaks that may be caused by vaccination at different times are waiting for further study.

### Acknowledgements

This work was partially supported by Zhejiang Province Natural Science Foundation (No. LY20F030012).

### References

1. D. Herlihy, *The Black Death and the transformation of the West* (Harvard University Press, 1997).
2. P. Ziegler, *The black death* (Faber & Faber, 2013).
3. R. Horrox, *The Black Death* (Manchester University Press, Manchester, England, 2013).
4. V. Surveillances, *China CDC Weekly* **2**, p. 113 (2020).
5. H. A. Rothan and S. N. Byrareddy, *J. Autoimmun.* , p. 102433 (2020).
6. A. J. Kucharski, T. W. Russell, C. Diamond, Y. Liu *et al.*, *The lancet infectious diseases* (2020).
7. X. He, E. H. Lau, P. Wu, X. Deng *et al.*, *Nat. Med.* **26**, p. 672 (2020).
8. L. Feng, Q. Zhao and C. Zhou, *Chaos Solitons Fractals* **139**, p. 110016 (2020).
9. I. Cooper, A. Mondal and C. G. Antonopoulos, *Chaos Solitons Fractals* **139**, p. 110057 (2020).
10. C. Wang, J. C. Knight and M. C. Elder, On computer viral infection and the effect of immunization, in *Proceedings 16th Annual Computer Security Applications Conference (ACSAC'00)*, 2000.

## 14 REFERENCES

11. R. Pastor Satorras and A. Vespignani, *Phys. Rev. E* **65**, p. 036104 (2002).
12. R. Cohen, S. Havlin and D. ben Avraham, *Phys. Rev. Lett.* **91**, p. 247901 (2003).
13. H. Zhang, J. Zhang, C. Zhou, M. Small and B. Wang, *New J. Phys.* **12**, p. 023015 (2010).
14. S. Xue, F. Ruan, C. Yin, H. Zhang and B. Wang, *International Journal of Modern Physics C* **21**, p. 1197 (2010). 
15. Y. Lu, G. Jiang and Y. Song, *Chin. Phys. B* **21**, p. 100207 (2012).
16. P. R. S. Rao and M. N. Kumar, *Chaos Solitons Fractals* **75**, p. 34 (2015).
17. F. Fu, D. I. Rosenbloom, L. Wang and M. A. Nowak, *Proc. R. Soc. B-Biol. Sci.* **278**, p. 42 (2011).
18. H. Zhang, F. Fu, W. Zhang and B. Wang, *Physica A* **391**, p. 4807 (2012).
19. Y. Zhang, *Chaos Solitons Fractals* **56**, p. 209 (2013).
20. C. Cai, Z. Wu and J. Guan, *Phys. Rev. E* **88**, p. 062805 (2013).
21. J. X. Yang, *EPL (Europhysics Letters)* **124**, p. 58004 (2019).
22. A. L. Barabási and R. Albert, *Science* **286**, p. 509 (1999).
23. C. T. Bauch, *Proc. R. Soc. B-Biol. Sci.* **272**, p. 1669 (2005).