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The spread of epidemic under voluntary vaccination with heterogeneous infection rates

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Epidemics usually spread widely and can cause a great deal of loss to humans. In the real world, vaccination is the principal method for suppressing the spread of infectious diseases. The Susceptible-Infected-Susceptible (SIS) model suggests that voluntary vaccination may affect the spread of an epidemic. Most studies to date have argued that the infection rates of nodes in the SIS model are not heterogeneous. However, in reality, there exist differences in the neighbor network structure and the number of contacts, 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 amount of contact among people. Here, we propose an improved SIS model with heterogeneity in infection rates, proportional to the degree of nodes. By conducting simulations, we illustrate that almost all vaccinated nodes have high degrees when the infection rate is positively correlated with the degree of a node. These vaccinated nodes can divide the whole network into many connected sub-graphs, which significantly slows down the propagation of an epidemic; the heterogeneity of infection rates has a strong inhibitory effect on epidemic transmission. On the other hand, when the infection rate is negatively related to the degrees of the infection rate nodes, it is difficult for most nodes to meet the inoculation conditions, and the number of inoculations is close to zero.

Keywords: SIS model; dynamical system; heterogeneous infection rate; individual vaccination.

1. Introduction

Large-scale infectious diseases, which can 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 the current COVID-19 outbreak has caused varying degrees of social panic. On March 11, 2020, the World Health Organization officially announced at a press conference that COVID-19[4–7] was a global pandemic. As of June 1, 2020, approximately 6 million people worldwide have been diagnosed with infections, including approximately

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400 000 who have died. 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] provided theoretical results about epidemic thresholds and the influence of the 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 more effectively control the spread of an epidemic has become a key topic for many research scholars.

To curb the spread of the virus, medical scientists have invented vaccines, which have 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 approaches to immunization on epidemic transmission, research scholars have proposed a range of such 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 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 have been proposed to explore the dynamic process underlying epidemics.

In the area of voluntary vaccinations, Zhang *et al.*[13] proposed the Susceptible-Infected-Susceptible (SIS) model to simulate voluntary vaccination and found that the voluntary immunization strategy inhibited the spread of an epidemic in a scale-free network. On the basis of the SIS model, Xu *et al.*[14] studied 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] took into consideration that the vaccinated individuals were not infected and proposed the Susceptible-Infected-Removed (SIR) model; this research showed that whether vaccinated or not, the consciousness of vaccination can effectively increase the threshold and slow the speed of disease transmission. On the basis of the SIR model, Rao *et al.*[16] showed that not only can vaccination eradicate disease but the treatment rate, even with time delays, can also contain the disease. Besides these studies, some research scholars[17–19] have also studied the role of individual mimicry behavior and population structures in vaccination.

In an actual situation, the environmental conditions, health habits and physical conditions affecting 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 Susceptible, Vaccinated, Infected and Recovered populations (SVIR) model to prove that the heterogeneity of infection rates can prevent or accelerate the spread of an epidemic. However, Cai *et al.* did not consider 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.

Based on the above considerations, in this paper, we propose an improved model based on voluntary vaccination, the heterogeneity of the infection rate being determined by the adjustable variable τ, 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 τ value on the spread of an epidemic in our model. A scale-free network is used for numerical simulations to study the effects of the heterogeneity of infection rates on the spread of an epidemic. We find that (1) when $\tau = -1$ (the node infection rate is negatively 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 $\tau = 1$ (the node infection rate is positively correlated with its own degree), the central node is more willing to be vaccinated, and as a result, the average degree decreases, thereby effectively limiting the spread of the epidemic.

2. Model

We describe the spread of an epidemic using the SIS epidemiological model. In the SIS model, node status can be divided into susceptible (S) and infected (I). V_S and V_I represent the susceptible nodes and the infected nodes, respectively. If node $i \in V_S$ and node $j \in V_I$ border one another, it is assumed that node i will be infected by node j, with a probability β. Consequently, the overall probability λ_i that node i will be infected by its neighbors can be calculated as follows:

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

where f_i means the number of infected neighbors of node i. After being infected, each patient has a probability μ to be restored 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, infection rates vary greatly between individuals due to different health habits, physical strengths and social conditions. In order to realistically simulate the spread of an epidemic in the world, each node with a heterogeneous infection rate has been considered in our model. Because the characteristics 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 correlate with the degree of the node itself. In order to properly define the heterogeneous infection rate based on the standard infection rate β (β can be understood as the average infection rate of the population), the infection rate must meet the following requirements: (1) The infection rate can be controlled by a parameter τ to produce positive and negative correlation results, and the results of two different correlations are better to be symmetric, because the different correlations only express an opposite change in the direction (increase or decrease) of infection rates. (2) Many phenomena fit into

the power-law distribution in real life.[13] Hence, the heterogeneous infection rate can be more actual, if it follows the power-law distribution. (3) In the real world, when the heterogeneous infection rate is positively correlated with the degree, the infection rate of an individual is usually higher (respectively, lower) than the average infection rate of the network, if that individual's degree is more (respectively, less) than the average degree. Hence, the average degree of the network may be introduced to regulate the heterogeneous infection rate. Specifically, the node's infection rate can be re-defined as follows[21]:

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

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. τ is introduced as the power exponent, to adjust the infection rate. Because the effect of τ on the infection rate can be fully reflected within the range -1 to 1, τ is arranged from -1 to 1 in our model.

With the change in the value of τ, 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) Conversely, 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 having a better chance of being vaccinated and medical conditions.

Before experimentally studying the heterogeneous infection rate β_i, we need to discuss social network structures, which determine the degree distribution of each node. More recent 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 γ is the degree exponent of the scale-free Barabsi Albert (BA) network. Hence, we will choose the BA network to simulate a real world network in this paper. Specifically, we set the BA network to have $N = 2000$ and an average degree $\langle k \rangle = 6$. The change relation between degree and β_i under different τ values is shown in Fig. 1.

Figure 1 provides the following three findings:

(1) When $\tau = 0$, the infection rate of each node is equal to β. This 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 the node itself. The positive correlation also increases with the increase of τ.
(3) When $\tau < 0$, the infection rate of each node is negatively correlated with the degree of the node itself. Not surprisingly, the negative correlation increases with the decrease of τ. The change relation between β_i and the degree of a node conforms to the characteristics of the power-law distribution.

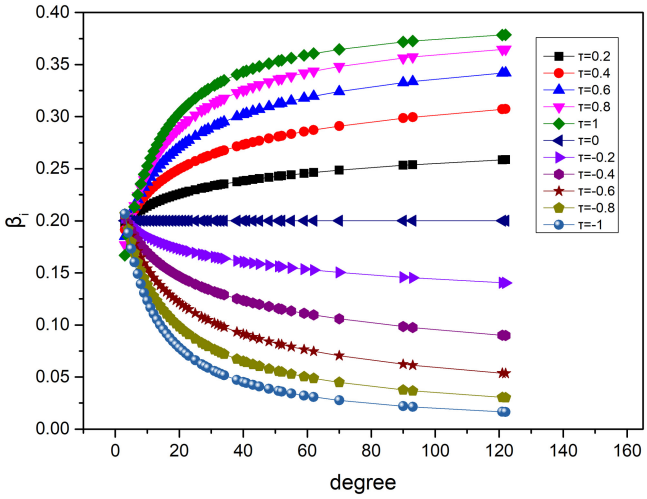


Fig. 1. (Color online) The node's infection rate β_i for different degrees in the BA network defined above ($\beta = 0.2$).

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

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

Next, we will discuss the process of rational individuals deciding whether to vaccinate in terms of the heterogeneous infection rates. Generally, rational individuals who always seek to fulfill personal interests will attempt to minimize their own costs of vaccination. To mimic the behavior of rational individuals in deciding about vaccinations, we first 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 nonvaccination (M_{ni}):

$$\begin{cases} M_{ni} = c_1\lambda_i, \\ M_v = c_2, \end{cases} \tag{4}$$

where c_1 is the parameter related to the loss from 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: an individual will vaccinate when $M_{ni} > M_v$ is satisfied; otherwise, the individual will not vaccinate. As with susceptible nodes and infected nodes, we use V_D to denote the vaccinated nodes. If the node $i \in V_D$, it can no longer be infected by others. Due to various vaccines having a different period of validity, to simplify our model, we assume the validity of the various vaccines discussed in this paper to be permanent.[13]

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

(1) Based on the expense, synchronously update whether all the nodes $i \in V_S$ 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.

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

Algorithm 1. Simulate the epidemic propagation

Initialize a BA network and build the V_I set by randomly selecting five infected nodes. #Initialize network structure and V_I
$I[n] = \emptyset$ and $S[n] = \emptyset$ and $D[n] = \emptyset$
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
for each time step $t \in [0, n]$ **do**
    for each node $i \in V_S$ **do**
        Calculate λ_i based on Eq. (2) and calculate M_{ni} based on Eq. (4)
        if $M_{ni} > M_v$ **then**
            Set $V_D = V_D \cup i$ and $V_S = V_S \setminus i$
            continue
        else
            Random a number P between 0 and 1
            if $P < \lambda_i$ **then** #It means the node i is infected
                Set $V_I^{'} = V_I^{'} \cup i$ and $V_S = V_S \setminus i$
            end if
        end if
    end for
    #The recovery process of infected nodes
    for each node $j \in V_I$ **do**
        Set $V_I^{'} = V_I^{'} \setminus j$ and $V_S = V_S \cup j$
    end for
    $V_I = V_I^{'}$ #Update the set of infected nodes and network states
    $I[t] = |V_I|$ and $S[t] = |V_S|$ and $D[t] = |V_D|$ #Record the number of nodes in different states at time step t
end for
return $I[n]$, $S[n]$ and $D[n]$ #Get three arrays that represent the spread of epidemic

Table 1. Parameters setting.

Parameter	Commentate	Value
β	Infection rate	0.2
μ	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

3. Result

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

The parameters used in experiments are exhibited in Table 1. In order for the epidemic to spread at a proper speed, we set the infection rate β to 0.2.[13] To simplify the model without a loss of generality, we set the recovery rate μ to 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 to 1.[13] In order to be consistent, c_2 is fixed at 0.7 as an instance. We will explicitly state when the value of a parameter is modified.

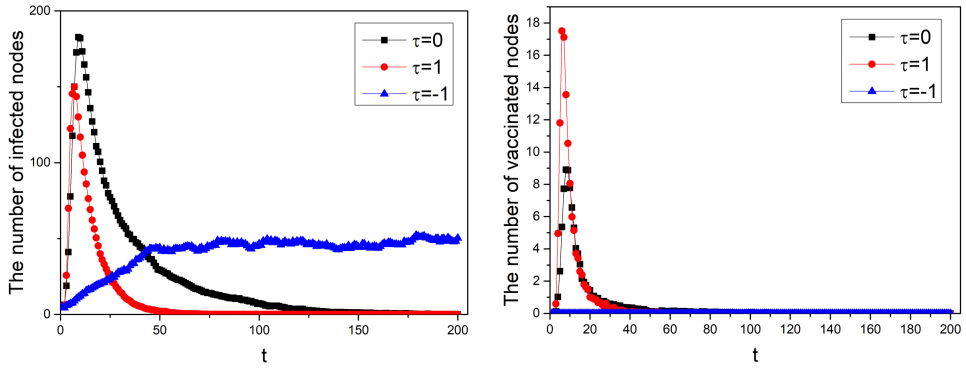
As shown in Fig. 2, (1) when $\tau = -1$, almost no node chooses to vaccinate, and the number of infected individuals remains at a small value along with the time step; (2) when $\tau = 1$, the maximum number of infected individuals is smaller than the maximum number of infected individuals under the case of the nonheterogeneous infection rate ($\tau = 0$), and the disease seems to subside faster. We discuss and explain these two findings below.

3.1. *When τ is -1, almost no one is vaccinated in the BA network*

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

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

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



(a) (b)

Fig. 2. (Color online) The spread of epidemics in BA network with the different settings of τ: (a) the number of infected individuals without voluntary vaccination vs time, (b) the number of vaccinations vs time.

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 particular threshold.

As shown in Fig. 3, by conducting simulations in the BA network, we find that it is possible to vaccinate the nodes with low degrees ($k \leq 7$); however, it is impossible to reach the vaccination condition for nodes with high degrees ($k > 7$).

We can use Eq. (5) to explain the above finding. As can be seen from Fig. 1, β_i is negatively correlated with the degree of the node when $\tau = -1$; therefore, $1 - \beta_i$ increases with the increase of the degree of node i. By the properties of the logarithm

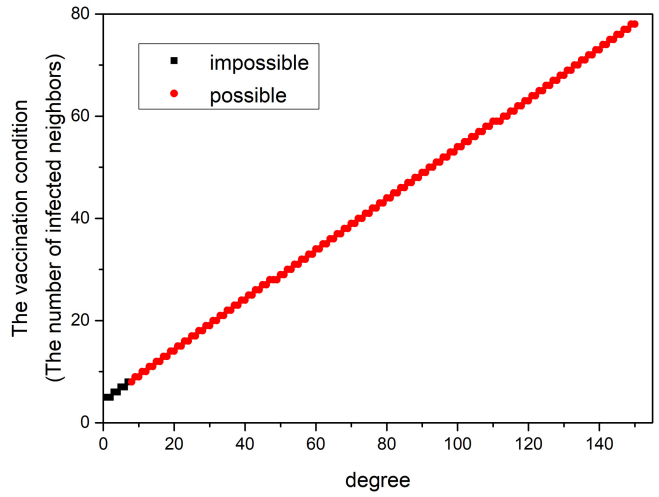


Fig. 3. (Color online) Vaccination conditions of nodes with different degrees in the BA network.

function, because $\frac{c_1-c_2}{c_1} = 0.3 < 1$, the value of the vaccination condition f_i increases as the degrees of the nodes increase, until the vaccination condition becomes impossible. Specifically, note that the vaccination condition of the node is greater than the degree of the node itself when the node has a high degree ($k > 7$).

Overall, from the description above, we know that when τ is -1, the nodes with low degrees ($k \leq 7$) cannot reach the vaccination condition and the vaccination condition of nodes with high degrees ($k > 7$) is also impossible. Hence, almost no node will vaccinate in the BA network, i.e. finding 1 is proved.

3.2. *When τ is 1, the regression of the epidemic in the BA network accelerates*

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

As can be seen from Fig. 4, when $\tau = 1$, the number of infected nodes begins to decline at the 7th time step, which is earlier than in the case without heterogeneity (i.e. $\tau = 0$). Moreover, when the number of nodes without heterogeneity begins to decline, the decline of patients with heterogeneity has already reached its peak, which 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 as the trend of $\tau = 0$. Analyzing Fig. 4 confirms that the epidemic will subside faster when $\tau = 1$.

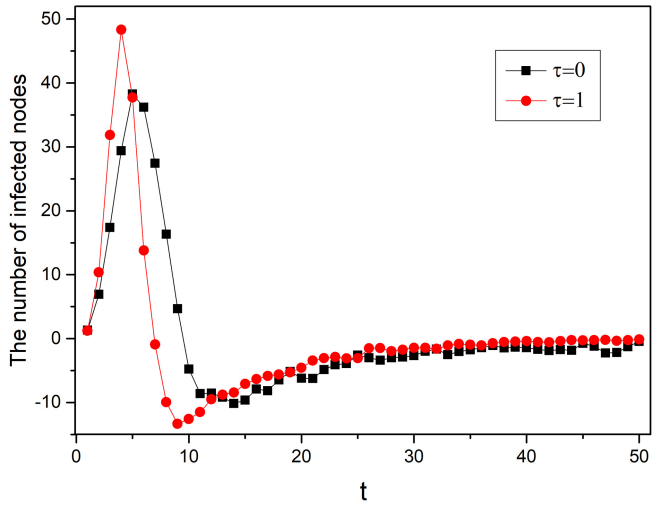


Fig. 4. (Color online) The number of infected individuals between adjacent time steps.

Before discussing the reasons for the second finding, we need to examine what factors influence epidemic propagation at first. In our model, the propagation of an epidemic can be expressed as the transmission between node statuses according to (1) the vaccinated nodes and (2) the infection rate β_i. To identify the reasons for a decline in infected nodes, the effects of these two factors should be thoroughly studied. Since the vaccinated node cannot be infected by its neighbors, we can see that the vaccinated node can insulate epidemic transmission. Hence, we can make the following assumption: although the network is not actually changed, the vaccinated node and the edges connected to it can be regarded as being removed from the network. 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 in Fig. 5, the number of connected sub-graphs tends to stabilize after the 15th time step in the transmission of the disease. We find that the number of connected sub-graphs generated in the network when $\tau = 1$ is higher than that generated when $\tau = 0$. Combined with Eq. (5), this can be explained as follows: the infection rate of a node is positively correlated with its own degree when $\tau = 1$; hence, the nodes with high degrees have higher infection rates than when $\tau = 0$, thereby improving the probability of the nodes with high degrees vaccinating.

Subsequently, we examine the effects of connected sub-graphs on epidemic transmission. As nodes are more likely to establish a connection with a node of high degree when they join the BA network, the connected sub-graphs formed in the network almost always contain few nodes after the nodes with high degrees are vaccinated, which will effectively limit epidemic transition from node to node. Overall, an increase in the number of connected sub-graphs can indeed inhibit the spread of diseases in the network.

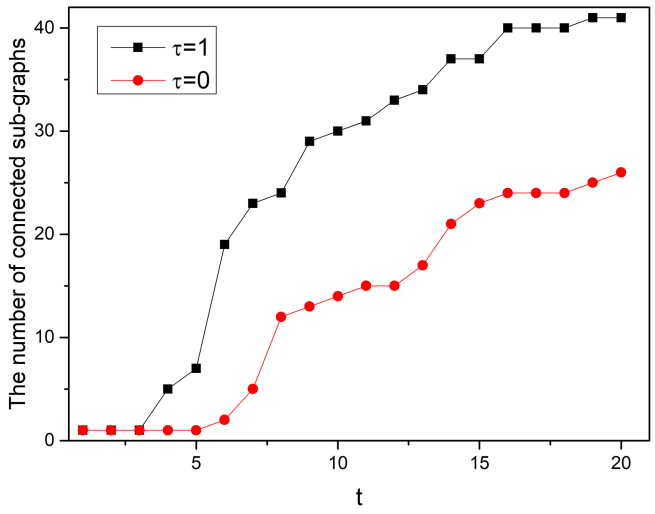


Fig. 5. (Color online) The number of connected sub-graphs in a network.

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 it is impossible for the epidemic to spread constantly. Therefore, in order to investigate the spread of the epidemic in the BA network, we should pay attention to the sub-graph with the most nodes. Next, we will discuss the spread of the epidemic in terms of the average degree of this connected sub-graph.

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

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

After discussing the influence of the vaccinated nodes on disease transmission, we will further analyze the role of β_i. If β_i is discussed independently, because it represents node i's probability of being infected by an infected neighbor, we cannot show how the disease spreads by analyzing β_i. Instead, we analyze $\sum \lambda_i$ at each time step to determine the impact of β_i on the epidemic transmission.

As seen from Fig. 7, $\sum \lambda_i$ is smaller at each time step when $\tau = 1$. This means that the epidemic transmission trend is weaker when $\tau = 1$. This is because, as seen in

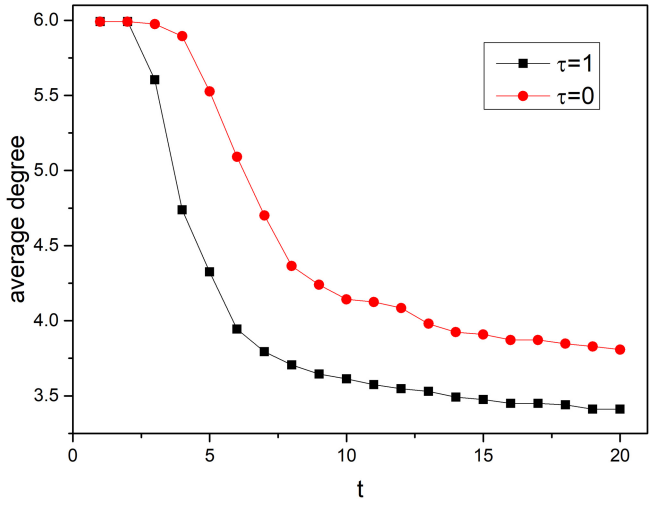


Fig. 6. (Color online) The average degree of the connected sub-graph with the maximum number of nodes.

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

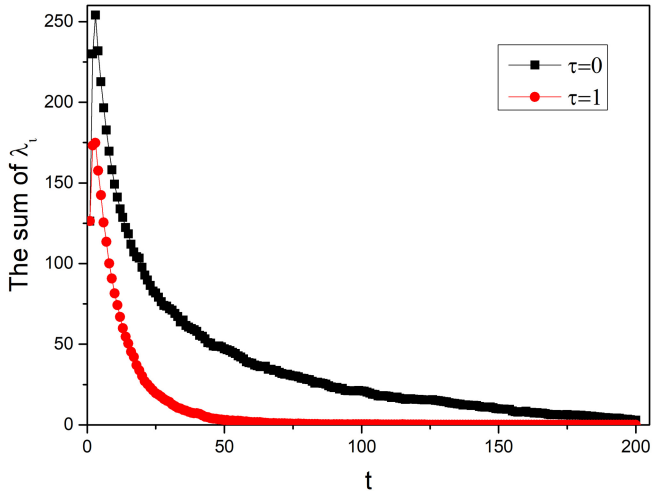


Fig. 7. (Color online) $\sum \lambda_i$ in each time step.

Fig. 1, β_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 a node with a high degree when they join the BA network. Since the nodes with high degrees are almost all vaccinated when $\tau = 1$, the nodes of $k = 1$ cannot be infected; hence, the actual β_i of nodes involved in epidemic transmission is smaller. Therefore, β_i inhibits epidemic transmission when $\tau = 1$.

Overall, from the above discussion, we 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 inhibits the spread thereof. Moreover, the actual β_i of nodes involved in epidemic transmission is smaller when $\tau = 1$. Hence, finding 2 is proved.

4. Conclusion and Discussion

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

By analyzing the experimental results, we derive 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. Also, it is impossible to reach the vaccination condition for nodes with high degrees ($k > 7$). Therefore, the vaccination condition is not conducive to the vaccination for nodes of all degrees when $\tau = -1$; (2) The regression of the epidemic in the BA network accelerates when $\tau = 1$. We find 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 β_i and the average degree to analyze it, and find that the probability of the epidemic spreading on this connected sub-graph is lower than the noninfectious rate heterogeneity.

Overall, when the heterogeneity of the infection rate is positively correlated with the degree of the node itself, it does more to inhibit the spread of the epidemic. In the real world, individuals who are exposed to an 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 an epidemic. In the real world, an individual's vaccination is also affected by the media and other factors. When individuals delay vaccinations due to the news, periodic outbreaks may be caused by vaccinations taking place at different times, something that awaits further study.

Acknowledgments

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