Epidemic Spreading with Variant Infection Rates on Scale-Free Network

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Abstract. In this paper, we proposed a susceptible-infected model with variant infection rates because different individuals have different resistance to diseases in different periods of real epidemic events. We consider two cases: Case 1, we know every individual's infection rate to a kind of epidemic, satisfy a type of distribution. Case 2, assume all individuals have same initial infection rates, a susceptible individual's infection rate will be less than the initial rate if he is not infected after limited number of contacts with infected ones. For both two cases, at the time t_D, preventive and control measures bring into effects, every individual's infection rate would decrease. We implemented this models on scale-free networks, and found that the epidemic process before the time t_D in Case 1 is almost the same as that in Standard SI model if the infection rate in Standard SI model equals the mean infection rate in Case 1. Furthermore, using numerical simulation, we analysis the effects of the parameter t_D, and find the bimodal distribution of final infection rates. Finally, we conclude what we get in this paper and give our future direction.

Keywords: Epidemic spreading, Variant infection rate, Scale-free network.

1 Introduction

The previous works about epidemic spreading in scale-free networks present us with completely new epidemic propagation scenarios in which a highly heterogeneous structure will lead to the absence of any epidemic threshold [1, 2]. These works mainly concentrate on the susceptible-infected-susceptible SIS [3, 4], susceptible-infected-removed SIR [5, 6]. Another typical model, susceptible-infected SI [7-8] models, also become the focus of the study, because in many cases, this model is more suitable to describe the dynamical process, for example, In the process of broadcasting [8], each node can be in two discrete states, either received or unreceived. A node in the received state has received information and can forward it to others like the infected individual in the epidemic process, while a node in the unreceived state is similar to the susceptible one. Since the node in the received state generally will not

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lose information. What's more, this model is more appropriate than SIS and SIR models when investigating the dynamical behaviors in the very early stage of epidemic outbreaks when the effects of recovery and death can be ignored.

However, the common assumption in most of the aforementioned works [9–11], when a susceptible individual contacts with an infected individual, the susceptible individual will be infected at a constant infection rate λ in the whole epidemic process. But in real system, individuals may have different infection rates and the infection rates may even change in time due to efficient preventive and control measures. It has been pointed out that the proper formulation of the infection rate requires taking many more detailed factors into account [12].Under this consideration, they display that different infection rates lead to different threshold behaviors in the SF networks [12]. And then Ke Hu took into account the effect of density of infected neighbors around an individual in the definition of spreading rate, some interesting results were found [7].

In our paper, we thought the infection rate of a susceptible individual was determined by his own body mass and the preventive and control measures, this means special body mass might have a better resistance to a kind of epidemic, and efficient preventive and control measures would lead to lower infection rate for susceptible individuals (In our paper, the infection rate of a susceptible individual means the rate that this susceptible individual would be infected, maybe we use "infected rate" better, but there is no such as kind of usage). At the beginning of the epidemic, people know little about the epidemic, nothing preventive and control measures were taken, the infection rates should be different for different body mass. After a period of time t_D, people recognize the severity of epidemic and then more preventive and control measures will bring into effect, the infection rates should also be different. For example, the SARS happened in Hong Kong in 2003 [13], all Indian resided there are not infected, while children are more difficult to be tainted than adults, so are some special people. When people realized the severity of this epidemic, some corresponding measures were taken, the number of infected individuals in every day decreases rapidly. Another example is the spreading of Hepatitis B, when people know more about this epidemic. Although it can't be cured, some preventive and control measures are found, so many susceptible individuals will hardly be infected, we can say their infection rates tend to be zeros. Obviously, the infection rates are different for different people in different periods.

So a new SI models consider different infection rates for different people in different periods are more suitable to describe the epidemic process. In our paper, we will put forward two kinds of improvement models in terms of different considering on the variant infection rates.

2 Model

In the Standard SI network model [9–11], individuals can be in two discrete states, either susceptible or infected. A susceptible individual will be infected at rate λ when he contacts with an infected individual. The infected individual will be still infected in the whole process. The total population N is assumed to be constant. Thus, if $S(t)$ and $I(t)$ are the number of susceptible and infected individuals at time t, respectively, then $N = S(t) + I(t)$. In our model, we will consider individual's different infection rates, so we consider the two cases as follow:

Case 1: We know every individual's infection rates, satisfy a kind of distribution. The different infection rates are attributed to different body mass. At the time t_D, some preventive and control measures bring into effect, and then the infection rates will decrease. Assume the infection rate at the time t is λ_{before}, and then the infection rate at the time t+1will be $\lambda_{after}=f_1(t, \lambda_{before})$.

Case 2: We don't affirm individual's infection rates in advance, but if a individual is still susceptible after m_1 contacts with infected ones, we can think he has relatively better resistance, corresponding to lower infection rate. Assume the infection rate of a susceptible individual is λ_{before} at the time t, after m_1 contacts with infected individuals, if he is still susceptible, then the infection rate will be $\lambda_{after}=f_2(m_1, t, \lambda_{before})$. At the time t_D, people know the severity of the epidemic and some preventive and control measures bring into effect, then the infection rate will be influenced by both body mass and preventive and control measures. Assume the infection rate at the time t is λ_{before}, after m_2 contacts with infected individuals, if he is still susceptible, then the infection rate will be $\lambda_{after}=f_3(m_2, t, \lambda_{before})$. In this case, the infection rates change in dependence of the time.

From the papers [14][15], we know that human body mass satisfies the normal distribution, and then we can use the normal distribution to reflect individual's different infection rates of different body mass. So we can adopt normal distribution in Case 1. What's more, before the time time t_D in Case 2, assume the normcdf denotes the cumulative normal distribution function(cdf), p = normcdf(λ, u, δ) returns the cdf of the normal distribution with mean u and standard deviation δ; while norminv denotes the inverse of the normal cumulative distribution function, λ=norminv(p, u, δ) returns the inverse cdf for the normal distribution with mean u and standard deviation δ, evaluated at the values in p. so the function f_1 can be expressed as $\lambda_{after}=f_2(t, m_1, \lambda_{before})=$ norminv(normcdf(λ_{before} , u, δ)-$k_1*m_1/(1/\lambda_{before})$, u, δ)= norminv(normcdf(λ_{before} , u, δ)-$k_1*m_1*\lambda_{before}$, u, δ), here, $k_1*m_1*\lambda_{before}$ describe the probability that his infection rate is less than λ_{before} , $k_1<1$ is a gain parameter . In our experiment, set $k_1=0.1$, $m_1=10$,u=δ=0.01;

After the time t_D, some measures will bring into effects, both body mass and preventive and control measures will affect the infection rates. So the function f_3 should be different from f_2. From the SARS happened in Hongkong in 2003, when people and government took some measures to prevent the epidemic, the infection rate decreased, as can be seen in Fig. 1. The curves can be well approximated using the function λ=a*exp(-b*t), where b(b<1) is a gain parameter. In our paper, we will use this type of exponential function to reflect the decrease of individual's infection rates due to preventive and control measures, so the function f_3 can be described as $\lambda_{after}=f_3(m_2, t, \lambda_{before})= \exp(-m_2*k_2) *\lambda_{before,}$ $k_2<1$ is a gain parameter. In our simulation, set m2=6; $k_2=0.2/6$; we can also use this type of function in Case 1, so the function f_1 in our experiment will be described as $\lambda_{after}=f_1(t, \lambda_{before})= 0.05* \lambda_{before,}$

Next, we will discuss our above models from the theory, and then the simulation results will testify what we get and give us some new understanding about the dynamic process.

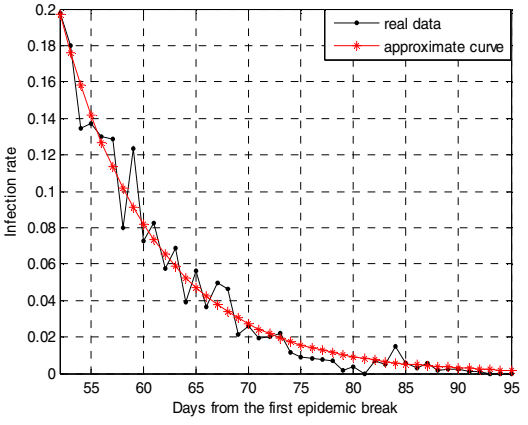

Fig. 1. The infection rate decreasing in dependence of the days about the SARS happened in Hongkong in 2003, The star-marker lines represent the approximation by the function λ=a*exp(-b*t), the best fit parameter are a=60, b=0.11

3 Theory Analysis

In this section, we try to analysis the differences between standard SI model with constant infection rates and Case 1 with variant infection rates for different body mass before the time t_D.

The network is represented by a connected graph G(V,E), where V is the set of n individuals and E the set of relations between them. The state of the system, at time t, is described by a vector $X(t)=[x_1(t),x_2(t)...,x_n(t)]$, where $x_i(t)=1$ if the individual i is infected and $x_i(t)= 0$,otherwise. Denote by $A=(a_{ij})_{i,j=1....n}$ the adjacent matrix of the graph structure on the set of individuals. $a_{ij}=1$ if there is a link between node i and j, and $a_{ij}=0$, otherwise. Denote $y_i(t)$ as at time t the number of the ith individual's infected neighbors, then at time t, the ith individual can be infected at the rate $P(y_i(t))<1$(k is a limited number),if we generate a uniform distributed random data ξ between 0 and 1,if $\xi<P(y_i(t))$,we can say this individual will be infected, if $\xi>P(y_i(t))$,he will still be susceptible. If we use the function Ceil(x) to return the minimum integer not less than x, and then the ith individual's state can be expressed as $Ceil(P(y_i(t)-\xi))$. Because $0<P(y_i(t))<1$, $0=<\xi<=1$,and then $-1<P(y_i(t))-\xi<1$, the value of $Ceil(P(y_i(t))-\xi)$ is 0 or 1. So the epidemic process of the Standard SI model can be described as follow.

$$[y_1(t), y_2(t),\cdots y_n(t)]=[x_1(t), x_2(t),\cdots x_n(t)]A \quad (1)$$

$$[z_1(t+1),\cdots z_n(t+1)]=Ceil([P(y_1(t)),\cdots P(y_n(t))]-rand(1,n)) \quad (2)$$

$$X(t+1)=Z(t+1)\vee X(t) \quad (3)$$

In the equation (2), the rand(1,n) denote a row vector, it has n independent random element all between 0 and 1, $(z_i(t))_{i=1.....n}$ denotes that at time t, the ith individual's state, but it contains the followed case: the ith individual who is infected at time t-1

may become susceptible at time t because $P(y_i(t))< \xi$. The operation $\vee$ denotes the "or" operation, if one of the two data is 1, and then the result is 1, if both two data are 0, and then the result is 0, so the equation (3) can ensure the infected individuals at time t-1 must be infected at time t. We can see the equations (1), (2) and (3) can describe the SI model equally.

For Case 1, equation (2) should be changed into equation (4) as follow:

$$[z_1(t+1),\cdots z_n(t+1)]=Ceil([P_1(y_1(t)),\cdots P_n(y_n(t))]-rand(1,n)) \tag{4}$$

Here $P_i(y_i(t))=1-(1-\lambda_i)^{y_i(t)}$, $\lambda_i(i=1,2\ldots.n)$ $(0<\lambda_i<1)$satisfies a kind of distribution. Equation (4) is a random equation, so we can consider the expectation E(Z(t)), and then the equation (4) can be changed into equation (5).

$$E([z_1(t+1)\cdots z_n(t+1)])=E(Ceil([P_1(y_1(t))\cdots P_n(y_n(t))]-rand(1,n))) \tag{5}$$

First, we give a theorem.

Theorem 1. If A and B are independent random variables, B satisfies the uniform [0, 1] distribution, and the variable $A \in [0,1)$, then E(Ceil(A-B)) =E(A).

Proof. (1) random variable A is continuous random variable.

Assume that the density function of random variable A is $f(a), a\in[0,1)$, the density function of B is $f(b)=1, b \in [0,1]$, so the joint density function of A and B is f(a, b)=f(a)*f(b), $a\in [0,1)$, $b\in [0,1]$, then,

$$Ceil(A-B)=\begin{cases}0 & A \le B\\ 1 & A > B\end{cases}$$

$$E(Ceil(A-B))=1*P(A>B)=\int_0^1\int_0^a f(a)f(b)dadb=\int_0^1 af(a)da=E(A)$$

(2) random variable A is discrete random variable .

Assume that the value of random variable A is $a_1, a_2\ldots\ldots a_n$, $0\le a_1<a_2<\cdots<a_n<1$ and the corresponding probability is $p_1, p_{2,}\cdots p_n$, $0<p_i\le 1, \forall i=1,2\cdots n$

$$E(Ceil(A-B))=P(A>B)$$
$$=\sum_{i=1}^{n} p_i P(B<a_i)=\sum_{i=1}^{n} p_i a_i=E(A)$$

So the theory is right no matter the type of the random variable A. For small λ, $P_i(y_i(t))=1-(1-\lambda_i)^{y_i(t)}=\lambda_i y_i(t)$, and then, we can infer:

$$E(Ceil([P_1(y_1(t)),\cdots P_n(y_n(t))]-rand(1,n)))$$
$$=E([P_1(y_1(t)),\cdots P_n(y_n(t))])\approx E([\lambda_1 y_1(t),\cdots \lambda_n y_n(t)])$$

$$E(Ceil([P(y_1(t)),\cdots P(y_n(t))]-rand(1,n)))$$
$$=E([P(y_1(t)),\cdots P(y_n(t))])\approx E([\lambda\ y_1(t),\cdots \lambda\ y_n(t)])$$

We know that λ_i and $Y_i(t)$ are independent random variables, so we can infer that:

$$E([\lambda_1 y_1(t), \cdots \lambda_n y_n(t)]) = \lambda E([y_1(t), \cdots y_n(t)])$$

So for small λ, if the mean of random variable λ in Case 1 equals the λ in standard SI model, the equation (2) and (4) can be thought equally. So the epidemic processes before the time t_D in Case 1 should be almost the same with that in Standard SI model.

4 Simulation Analysis

To demonstrate the differences of our improvement models on the spreading processes, we perform extensive numerical simulations on the Baraba′si and Albert (BA) networks[5], the network size N=1000, and in our paper, we consider three kinds of cases: Standard SI model, Case 1 and Case 2. In standard SI model, assume the constant infection rate λ=0.01; In Case 1, we adopt the normal distribution with mean u=0.01 and standard deviation δ=0.01. Next, we will discuss our models from the several aspects as follow.

(1) Epidemic spreading processes

Barthélemy et al. [9,10] studied the SI model in Barabási-Albert (BA) scale-free networks, and found that the density of infected nodes, denoted by i(t), grows approximately in the exponential form, $i(t)= e^{ct}$, where the time scale c is proportional to the ratio between the second and the first moments of the degree distribution, $c\sim\langle k^2\rangle/\langle k\rangle$.

From the theory analysis in last section, the epidemic processes of Standard SI model and Case 1 before the t_D should be almost the same when the infection rate in Standard SI model equals the mean infection rate in Case 1.The similar result can be seen in fig.2 by means of simulation. We set t_D=100, and run our simulation for about 100 times, the average of all values was the final result. From the graph, the almost

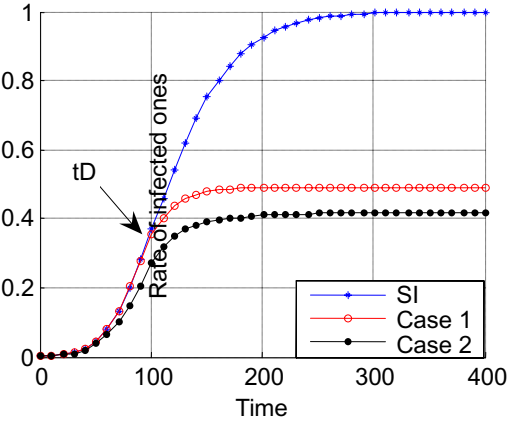

Fig. 2. Average rate of infected individuals in dependence of the simulation time for SI model, Case 1, Case 2. The infection rate in Standard SI model, mean infection rate in Case 1 and initial infection rate in Case 2 are all 0.01, t_D=100, other parameters settings are introduced in section 2. We run our simulation for about 100 times, the average value as the result.

same epidemic processes were shown before the time $t_{D,}$ but after the preventive and control measures bring into effects, the process in Case 1 become slow, and reach the steady state quickly, some people would not be infected finally, this can explain why few epidemics can cause all individuals being infected.

However, in Case 2, the initial infection rates are all assumed to be 0.01, before the time t_D, some individuals were thought to have better resistance if they were still susceptible after a certain number of contacts with infected ones. These individuals have lower infection rates, and then mean infection rate decreased gradually before the time t_D, So the epidemic process for Case 2 is slower than that for Case 1, as can be seen in fig.2.

From the epidemic process, we know that: On the one hand, if we use the constant infection rate to study the statistical characters of epidemic processes instead of individual's random infection rate satisfying a kind of distribution whose mean value equal the constant infection rate in Standard SI model, we can also get the similar results. On the other hand, preventing and control measures should be taken as soon as possible, so the parameter t_D is important in our present model.

(2) Effect of the parameter t_D

As can be seen in fig.2, the parameter t_D has a large impact on the epidemic process, so in this section, we will discuss its effects from two aspects. First, we analysis the effects on the epidemic process. The number of increased infected ones in dependence of the time can be seen in fig.3, we fix the parameter t_D=100, and run our simulation for about 100 times, the average value as our results. From the graph, we know that the spreading speed in Case 1 is similar to that in the standard SI model until the time t_D, after some efficient measure taken, the number of increased infected ones at every simulation time decrease sharply. So is that in Case 2. The maximum of increased infected ones in Case 2 is less than other models because of the less mean infection rates.

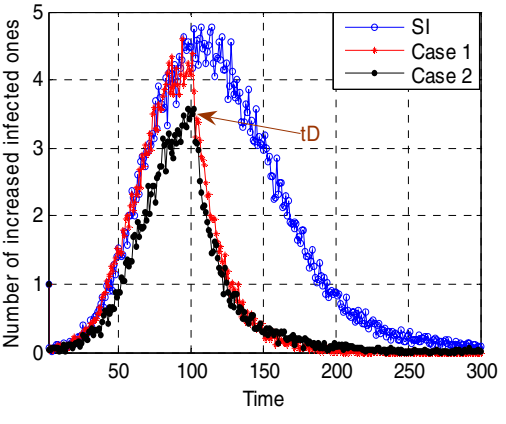

Fig. 3. Average rate of increased infected individuals in dependence of the simulation time for SI model, Case 1, Case 2. The infection rate in Standard SI model, mean infection rate in Case 1 and initial infection rate in Case 2 are all 0.01, t_D=100, other parameters settings are introduced in section 2. We run our simulation for about 100 times, the average value as the result.

Second, we study the steady infected rates for different parameter t_D, which were shown in the fig.4, We run our simulation for about 100 times, the average rates of final infected ones as results. From the graph, we can see with the increase of t_D, more people will be infected. And the rates increased at a exponential form when the parameter t_D is less than 150, so it's better to adopt the preventive and control measures as soon as possible. Another interesting phenomenon is the rates of final infected ones in Case 2 is larger than that in Case 1 when the parameter t_D is small, this is because, the parameter settings cause the preventive and control measures in Case 2 less effective than that in Case 1, but when t_D is large enough, the process in Case 2 before the time t_D is slower than that in Case 1, although the preventive and control measures in Case 2 is less effective, the rate of final infected ones are still lower than that in Case 1.

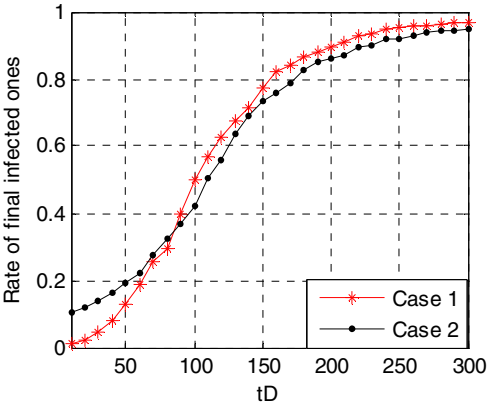

Fig. 4. Average rate of final infected individuals in dependence of the parameter tD for Case 1, Case 2. The mean infection rate in Case 1 and initial infection rate in Case 2 are both 0.01, other parameters settings are introduced in section 2. We run our simulation for about 100 times, the average value as the result.

(3) Distribution of final infection rates

In Case 1, the infection rates are assumed to be normal distribution, but after the time t_D, preventive and control measures are taken, so susceptible individual's infection rates will decrease, how's the final infection rates? That's what we should study in this section. By means of simulation, the distribution of the final infection rates can be seen in fig.5, most of individuals has almost zero infection rates, that means many people won't be infected in the final steady state, as can also be seen in fig.2, because after a period of time, people gain more information about this epidemic, and some efficient measures will bring into effects, finally the infection rates will tend to be zeros.

However, in Case 2, we can see obvious bimodal distribution, this difference can be explained as follow: at the initial time of epidemic break, few knowledge about the epidemic, people pay no attention, and then many people will be infected, while people realize the severity of this epidemic, many people will take many efficient measures, and a large portion of people will not be infected in the end.

This kind of phenomenon also exists in Case 2, but as the infection rates were known aforehand, and they are distributed inside a range. So the bimodal phenomenon is not obvious in Case 1, but we still can see a little peak near the 0.01.

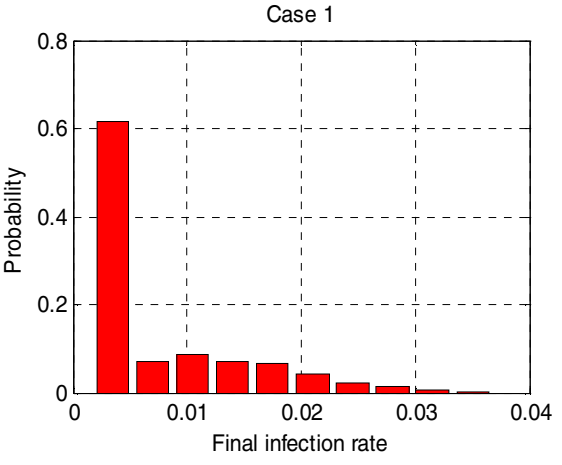

Fig. 5. Distribution of individual's final infection rates for different m in Case 2

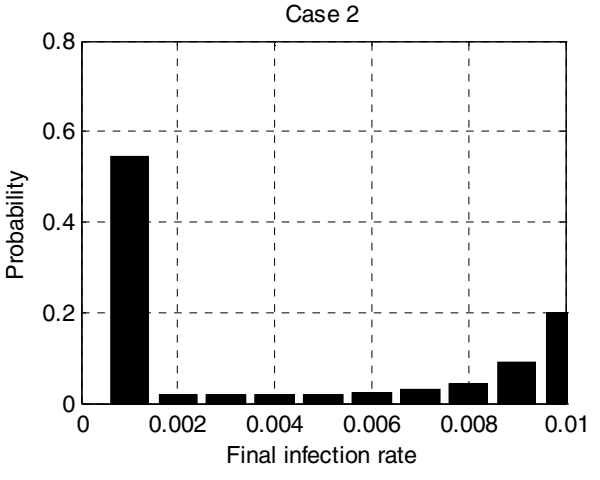

Fig. 6. Distribution of individual's final infection rates for Case 1, Case 2. The mean infection rate in Case 1 and initial infection rate in Case 2 are both 0.01, t_D=100, other parameters settings are introduced in section 2. We run our simulation for about 100 times, the average value as the result.

5 Conclusion and Discussion

Almost all the previous studies about the epidemic spreading models in scale-free networks essentially assume that the infection rates for all individuals are the same.

In real epidemic events, individual's infection rate are dominant by his resistance to diseases and preventive and control measures, special body mass might have better resistance to a kind of epidemic, and better preventive and control measures bring lower probability to be infected. To simulate the epidemic process, it's essential to consider the individual's different infection rate. In our paper, we proposed two kinds of SI models with variant infection rates. In Case 1, we know every individual's infection rate, satisfying a kind of distribution. We use several random equations to describe the epidemic processes equally, and from the theory, we know that if the mean infection rate equals the infection rate in standard SI model, the epidemic processes before the time t_D in two models will be almost the same, the simulations testify the results. In Case 2, we don't know every body's resistance to the diseases, considering that a susceptible individual will have a lower infection rate if he is not infected after limited number of contacts with infected individuals. The epidemic process is relatively slow because of the decreased mean infection rates.

Moreover, we know that the parameter t_D has a large impact on the epidemic process, after the time t_D, the epidemic process become slow, and the number of increased infected ones at every simulation time decrease sharply, while the mean infection rates in both Case 1 and Case 2 will decrease, finally many people won't be infected, corresponding to almost zero infection rate, this can explain why most people will hardly be infected in most of epidemic break. What's more, from the distribution of final infected rates, we know that the distribution in Case 2 is bimodal, correspond to a real life to a society in which a relative large portion of people will be infected because of less resistance against the epidemic while another large portion of people will hardly be infected in the end due to efficient preventive and control measures.

We put forward a SI model with variant infection rates to describe the epidemic process, give us some new understanding about epidemic process. But some parameter settings are artificial, such as m_1, m_2, k_1, k_2, and so on; this parameters may be different for different epidemic events, so it should be more appropriate to combine the real epidemic break to affirm these parameters. Whether the thought on other models and networks can get some useful results is worthy of further study.

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