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Research article

The impact of behavioral change on the epidemic under the benefit comparison

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Abstract: Human behavior has a major impact on the spread of the disease during an epidemic. At the same time, the spread of disease has an impact on human behavior. In this paper, we propose a coupled model of human behavior and disease transmission, take into account both individual-based risk assessment and neighbor-based replicator dynamics. The transmission threshold of epidemic disease and the stability of disease-free equilibrium point are analyzed. Some numerical simulations are carried out for the system. Three kinds of return matrices are considered and analyzed one by one. The simulation results show that the change of human behavior can effectively inhibit the spread of the disease, individual-based risk assessments had a stronger effect on disease suppression, but also more hitchhikers. This work contributes to the study of the relationship between human behavior and disease epidemics.

Keywords: behavioral change; epidemic spread; game theory; stability; hitchhiking

1. Introduction

The impact of personal behavior choices on epidemic transmission has been studied for a long time [1]. It is well known that human behavior, such as wearing face masks, vaccination, avoiding public crowded places and so on, has a significant impact on the spread of epidemics on complex networks [2]. Studying the dynamics of human behavior during epidemic transmission is of great significance in suppressing the spread of epidemics [3].

In the event of an outbreak, frequent hand washing, wearing a mask can reduce transmission rates, and vaccination can immunize people against the disease. The effects of these behaviors on disease are shown in the following ways in the model: Switches in infectious disease state (e.g. moving vaccinators to vaccinated state or immune state) [4–6]; changes in model parameters (infection rate is reduced by modeling social distance) [7–9]; changes in social contact structure (link-breaking or rewiring process is modeled in individual-based modelling) [10–12]. In this paper, we use an economic

objective function to simulate the dynamics of human behaviour.

Game exists in every aspect of our life, especially among human interactions. The application of game theory in the model of infectious diseases has been increasing in recent years [13]. Earlier studies used a non-iterative game model coupled with the SIR epidemic model to study the impact of population behavior on epidemics [14, 15]. This kind of models are limited to lifelong vaccination strategies. In recent years, many more practical models have been proposed, such as iterative game model, imitating dynamics, random games, etc [16–18]. In this paper, the iterative game model and simulation dynamics are used. In the game model, the quantification of payoffs is the key factor to solve the problem. There are two ways to quantify payoffs: Self-assessment and neighbor-based assessment. Self-assessment rely on the memory and personal perception of the disease [19, 20], whilst neighbor-based assessment puts individuals into an environment where personal decisions are influenced by the choices of their neighbours, where the neighbours are determined by the topology of the contact network [21]. Our model takes into account both approaches and analyses the differences in terms of the intensity of disease suppression and the number of hitchhikers.

There are some new models of human behavioral games, especially the behavior of vaccination. Recently, Kabir et al. [22] presented the two layer SIR/V-UA epidemic diffusion model, the results of the paper found that awareness can promote personal vaccination. In the same year, they analyzed behavioral incentives in the vaccination dilemma [23], simulations showed that vaccine characteristics are more important in controlling the treatment adoption than the cost of treatment itself. Kuga et al. [24] evaluate the performance of vaccination-subsidizing policies in the face of a seasonal epidemic, the results suggest that subsidies should be aimed at voluntary vaccinators while avoiding overspending. Arefin et al. [25] proposed the effect of vaccination behavior on the prevalence of specific strains during the simultaneous spread of two influenza-like disease strains. The results show that the efficacy of the vaccine against the new strain plays a key role in controlling the epidemic of the disease. In reference [26], the authors proposed a model couples an SIR model with selection of behaviors, it mainly analysis the time scales difference of behavior changes and epidemic transmission, not consider influence from surrounding neighbors. Inspired by the reference, we built a model that coupled human behavior with epidemic transmission, but the difference is that we consider both self-assessment and the influence of neighbors on individual behavior dynamics.

The main contributions of this article are: the coupling model of human behavior and epidemic disease is proposed, and it has practical implications; the individual-based risk assessment and neighbor-based replication dynamics are considered; The suppression of epidemic diseases and the situation of free riding under two kinds of payoff evaluation methods are analyzed.

The rest of this paper is organized as follows. In section 2, we introduce a system that combines epidemics and personal behavior on complex networks. In section 3, we estimate the epidemic threshold of epidemic spreading model and the stability of disease-free equilibrium. Some numerical simulations are given in section 4. Finally, the conclusions are summarized in section 5.

2. Materials and methods

The scale-free network has serious heterogeneity and has a wide range of applications in life [27], such as: Power grids [28], world wide web [29], ecological networks [30], etc. we consider human behavior changes and epidemic transmission on scale-free networks. The target network has *N* nodes,

and its adjacency matrix is *A*. if node *i* and node j ($j \neq i$) are connected, then $a_{ij} = a_{ji} = 1$; otherwise $a_{ij} = a_{ji} = 0$. $k_i = \sum_{j=1}^{N} a_{ij}$ denotes the degree of node *i*. Assuming that the network is strongly connected, then matrix A is an irreducible matrix.

In this article, we mainly consider the impact of spontaneous human behavior changes such as wearing masks and hand washing on the spread of epidemics. At the same time, we consider the classic SIS model without immunity. The SIS model is a paradigmatic epidemic spreading model, in which each node can be in one of two states, either susceptible or infected. We assume that each infected node recovers at a unit rate. We consider that individuals may adopt two mutually exclusive behaviours, *s* ("safeguard") and *n* ("normal"). Specifically, we assume that individuals adopting behaviour *s* are able to reduce infection rate with respect to individuals adopting behaviours *n*. Thus, two infection rates λ_s and λ_n are considered for the two behaviours *s* and *n*. Naturally, we suppose that $\lambda_n > \lambda_s$. Let $S_i(t)$ denotes the probability that individual *i* is in susceptible state at time t, and $I_i(t)$ denotes the probability that individual *i* can be written as:

$$\begin{cases} \dot{S}_{i}(t) = -[\lambda_{s}x_{is}(t)S_{i}(t) + \lambda_{n}x_{in}(t)S_{i}(t)] \sum_{j=1}^{N} a_{ij}I_{j}(t) + I_{i}(t), \\ \dot{I}_{i}(t) = -I_{i}(t) + [\lambda_{s}x_{is}(t)S_{i}(t) + \lambda_{n}x_{in}(t)S_{i}(t)] \sum_{j=1}^{N} a_{ij}I_{j}(t). \end{cases}$$
(2.1)

 $x_{is}(t)$ is the probability that individual *i* chooses behaviour *s* at time t, $x_{in}(t)$ is the probability that individual *i* chooses behaviour *n* at time t. The specific content of x_{is} and x_{in} will be described later.

Considering human contact, we apply the replication equation in evolutionary game theory to analyze the dynamics of human behavior in the process of disease transmission. The quantification of individual's payoff mainly considers factors such as the cost of behavior, psychological fear, and risk of infection. The extent of psychological fear are different when contact with individual with behaviour s or behaviour n, also the risk of infection are different. The payoff matrix is as follows:

$$M = \begin{pmatrix} s & n \\ s & u_{11} & u_{12} \\ n & u_{21} & u_{22} \end{pmatrix}.$$

The first column of the matrix represents the two strategies of an individual, and the first row represents the two strategies of its neighbors. u_{11} denotes the payoff of individual who adopts behaviour *s* when he interact with the one who adopts the same behavior. u_{12} denotes the payoff of individual who adopts behaviour *s* when he interact with the one who adopts behavior *n*. u_{21} denotes the payoff of individual who adopts behaviour *s* when he interact with the one who adopts behavior *s*. u_{22} denotes the payoff of individual who adopts behaviour *n* when he interact with the one who adopts behavior *s*. u_{22} denotes the payoff of individual who adopts behaviour *n* when he interact with the person who adopts the same behavior.

We define that u_{is} is the expected payoff of individual *i* choosing behaviour *s*, $u_{is} = u_{11}p_{is} + u_{12}p_{in}$. We also define that u_{in} is the expected payoff of individual *i* choosing behaviour *n*, $u_{in} = u_{21}p_{is} + u_{22}p_{in}$. u_i is the expected payoff of individual *i* choosing behaviour *s* with probability $x_{is}(t)$, $u_i = u_{is}x_{is} + u_{in}x_{in}$. $p_{is} = \frac{1}{k_i} \sum_{i=1}^{N} a_{ij}x_{js}$ and $p_{in} = \frac{1}{k_i} \sum_{i=1}^{N} a_{ij}x_{jn}$, respectively represent the probability of neighbors with behavior *s* and neighbors with behavior *n* of node *i*, and $p_{is} + p_{in} = 1$. For the dynamics of human behavior, it is assumed that individuals interact randomly with their neighbors at a certain rate. Generally if the payoff of a pure strategy is greater than the expected payoff of mixed strategy adopted now, then the pure strategy is adopted with a higher probability. In this situation, the rate of change is assumed proportional to the difference in payoff. At the same time, it was assumed that the changes in strategies of individuals affected by their neighbors were proportional to the prevalence of disease in their neighbors. We assume that individuals are bounded rational. Based on the above assumptions, we can construct the behavior' replicator dynamics equation as:

$$\begin{cases} \dot{x}_{is}(t) = bx_{is}(t)(u_{is} - u_i)\frac{1}{k_i}\sum_{j=1}^{N} a_{ij}I_j(t), \\ \dot{x}_{in}(t) = bx_{in}(t)(u_{in} - u_i)\frac{1}{k_i}\sum_{j=1}^{N} a_{ij}I_j(t). \end{cases}$$
(2.2)

where i = 1, 2, ..., N, $x_{is}(t)$ is the probability that individual *i* chooses behaviour *s* at time t, $x_{in}(t)$ is the probability that individual *i* chooses behaviour *n* at time t, and $x_{is}(t) + x_{in}(t) = 1$. b > 0 is the proportionality constant. k_i denotes the degree of node *i*. The disease prevalence of neighbors is defined as $\frac{1}{k_i} \sum_{j=1}^{N} a_{ij}I_j(t)$.

The dynamics of behaviours is modelled as a selection dynamics. In addition to considering the interaction with neighbors, we also take into account the payoff ignoring the surrounding impact. For example, consider only the benefits of changing behavior or the benefits of being infected. We also assume that it's proportional to the probability that individual *i* is infected. The dynamic of behaviours becomes:

$$\begin{cases} \dot{x}_{is}(t) = ax_i(t)(1 - x_i(t))(f_s - f_n)I_i(t) + bx_i(t)(u_{is} - u_i)\frac{1}{k_i}\sum_{j=1}^N a_{ij}I_j(t), \\ \dot{x}_{in}(t) = ax_i(t)(1 - x_i(t))(f_n - f_s)I_i(t) + bx_i(t)(u_{in} - u_i)\frac{1}{k_i}\sum_{j=1}^N a_{ij}I_j(t). \end{cases}$$
(2.3)

 f_s is the payoff of behaviour s, and f_e is the payoff of behaviour e. a > 0 is the proportionality constant.

 $x_{is} + x_{in} = 1$, and $S_i + I_i = 1$. Therefore, the limit system of the complete dynamics of infection and behaviour is given by

$$\begin{cases} \dot{x}_{is}(t) = ax_{is}(t)(1 - x_{is}(t))(f_s - f_n)I_i(t) + bx_{is}(t)(u_{is} - u_i)\frac{1}{k_i}\sum_{j=1}^N a_{ij}I_j(t), \\ \dot{I}_i(t) = -I_i(t) + \left[\lambda_s(1 - I_i(t))x_{is}(t) + \lambda_n(1 - I_i(t))(1 - x_{is}(t))\right]\sum_{j=1}^N a_{ij}I_j(t). \end{cases}$$
(2.4)

In the model, human behavior affects the infection rate of an epidemic, and the spread of an epidemic affects individual behavior choices. When the epidemic dies out, human behavior will be unaffected by the epidemic. The key of this paper is to address the outbreak of epidemics under the influence of human behavior. Next we will analyze the stability of the equilibrium point in the epidemic model.

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3. Results

Let $\Omega = \{(\rho_1, \rho_2, ..., \rho_N) \in R^N_+ | 0 \le \rho_i \le 1, i = 1, 2, ..., N\}$. It's easy to prove that Ω is the positive invariant set and is the solution space for system (2.1). $E_{i0} = (x^*_{is}, 0)$ is the disease-free equilibrium of individual *i* in system (2.4). The disease-free equilibrium of system (2.4) is $E_0 = [E_{10}E_{20}...E_{N0}]$. Let us first prove the existence of the endemic equilibrium point.

Lemma 1 (Gerschgorin theorem [31]). Let $A = (a_{ij}) \in C^{n \times n}$ and let $r_i = \sum_{j=1, j \neq i}^n |a_{ij}|, i = 1, 2, ..., n$. Then, all the eigenvalues of A lie in the union of n closed discs $\bigcup_{i=1}^n \{z \in C : |z - a_{ii}| \le r_i\}$, where C is the set of complex number and $C^{n \times n}$ represents the complex matrices set of the order $n \times n$.

Theorem 1. If $\lambda_s > \frac{1}{\rho(A)}$, then the epidemic spreads will becomes endemic. $\rho(A)$ is the spectral radius of the adjacency matrix $A = (a_{ij})_{N \times N}$.

Proof. We calculate the steady-state probability of infection for each node i from (2.4), which is determined by the following nonlinear equation:

$$I_i(t) = [\lambda_s(1 - I_i(t))x_{is}(t) + \lambda_n(1 - I_i(t))(1 - x_{is}(t))] \sum_{j=1}^N a_{ij}I_j(t).$$

Let $\lambda_i = \lambda_s x_{is} + \lambda_n x_{in}$ and $\Theta_i = \sum_{j=1}^N a_{ij} I_j(t)$, bring these two equations into the above equation, then we obtain a self-consistency equation and expressed as *f*:

$$\Theta_i = \sum_{j=1}^N a_{ij} \frac{\lambda_i \Theta_i}{1 + \lambda_i \Theta_i} \equiv f(\Theta_i).$$

Obviously, $\Theta_i = 0$ is always a solution of this equation. Note that:

$$f'(\Theta_i) = \sum_{j=1}^N a_{ij} \frac{\lambda}{(1+\lambda\Theta_i)^2} > 0,$$
$$f''(\Theta_i) = \sum_{j=1}^N a_{ij} \frac{-2\lambda_i^2}{(1+\lambda_i\Theta_i)^3} < 0.$$

Therefore, it's a concave function. According to the properties of concave functions, a nontrivial solution of individual *i* exists only if

$$\frac{df(\Theta_i)}{d\Theta_i}|_{\Theta_i=0} = \sum_{j=1}^N a_{ij} \frac{\lambda_i}{(1+\lambda_i\Theta_i)^2}|_{\Theta_i=0} = \sum_{j=1}^N a_{ij}\lambda_i > 1.$$

Then, we have

$$\lambda_i > 1 / \sum_{j=1}^N a_{ij}.$$

It is easy to know that $\min\{\lambda_i\} = \lambda_s$, and according to the Gerschgorin theorem, we can conclude that there is *i* satisfying $1/\rho(A) > 1/\sum_{j=1}^{N} a_{ij}$. According to the above conditions, we can conclude that

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if $\lambda_s > 1/\rho(A)$, there must be *i* satisfying $\lambda_i > 1/\sum_{j=1}^N a_{ij}$. system (2.4) will have a unique positive equilibrium

equilibrium.

The proof is therefore completed.

Let $\dot{x}_{is}(t) = f_i(t)$, $\dot{f}_i(t) = g_i(t)$. Next we will prove the stability of the disease-free equilibrium.

Theorem 2. The disease-free equilibrium E_0 is locally stable if $\lambda_n < \frac{1}{\rho(A)}$ and E_0 is unstable if $\lambda_s > \frac{1}{\rho(A)}$.

Proof: The jacobian matrix of system (2.4) at E_0 is

$$J(E_0) = \begin{pmatrix} o & * \\ o & A_1 \end{pmatrix},\tag{3.1}$$

where A_1 is an $n \times n$ matrix whose elements are represented as:

$$(A_1)_{ij} = \begin{cases} -1, & i = j, \\ [\lambda_s x_{is} + \lambda_n (1 - x_{is})] a_{ij}, & i \neq j. \end{cases}$$
(3.2)

Obviously, $A_1 = -E + \Lambda A$, where

$$\Lambda = diag\{\lambda_s x_{1s} + \lambda_n(1-x_{1s}), \lambda_s x_{2s} + \lambda_n(1-x_{2s}), \dots, \lambda_s x_{Ns} + \lambda_n(1-x_{Ns})\}.$$

Suppose the eigenvalues of *A* are arranged in order as: $\lambda_1 \leq \lambda_2 \leq \cdots \leq \lambda_N = \rho(A)$. the maximum eigenvalue of $A_1, \rho(A_1)$, conforms to the following inequality:

$$\rho(A_1) \le \max\{(\lambda_s x_{is} + \lambda_n (1 - x_{is}))\rho(A) - 1\} = \lambda_n \rho(A) - 1.$$

If $\rho(A) \leq 0$, then $\rho(A_1) < 0$. we can draw that all eigenvalues of $J(E_0)$ are non-positive and eigenvalues with a zero real part correspond only to single elementary factors and hence, the disease-free equilibrium point is locally asymptotically stable. If $\rho(A) > 0$, and $\lambda_n < \frac{1}{\rho(A)}$, then $\rho(A_1) < 0$. Similarly, the disease-free equilibrium point is locally asymptotically asymptotically stable. If $\lambda_s > \frac{1}{\rho(A)}$, then $\rho(A_1) > 0$ and hence, the disease-free equilibrium point is unstable.

Set the left eigenvector of *A* corresponding to $\rho(A)$ as $\omega = (\omega_1, \omega_2, ..., \omega_N)$. since *A* is irreducible, then $\omega_i > 0$ [32]. We will analyze the global stability of the disease-free equilibrium.

Theorem 3. The disease-free equilibrium E_0 is globally stable if $\lambda_n < \frac{1}{\rho(A)}$.

Proof: Define the Lyapunov function as following:

$$V(t) = \sum_{i=1}^{N} \omega_i I_i.$$

Since $\omega_i > 0$ for i = 1, 2, ..., N. The derivative of V(t) with respect to t along the solution of the system (2.4) is given by

$$\begin{aligned} \frac{dV(t)}{dt} &= \sum_{i=1}^{N} \omega_i \dot{I}_i(t) \leq \sum_{i=1}^{N} \omega_i (-I_i(t) + \lambda_n \sum_{j=1}^{N} a_{ij} I_j(t)) \\ &= \lambda_n \omega A I - \omega I = \lambda_n \rho(A) \omega I - \omega I = (\lambda_n \rho(A) - 1) \omega I. \end{aligned}$$

If $\lambda_n \rho(A) < 1$, then $\frac{dV(t)}{dt} \le 0$ and $\frac{dV(t)}{dt} = 0$ if and only if I = 0. By LaSalle's Invariance Principle, E_0 is globally stable.

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4. Numerical simulations

This section uses simulation to characterize how human behavior affects epidemics. First, the dynamics of the epidemic model is simulated without considering the game. Then the epidemic spread is also simulated when the epidemic model is coupled with human behavior. By comparing the spread of epidemics in these two models, we can discover the impact of human behavior on epidemics. In order to understand the impact of the two considerations on the disease in order to provide the government with a better way of guidance, we simulated the impact of the two parameters on the disease. In order to understand the situation of human free-riding during the spread of disease, and thus understand the psychological role of humans in the process of disease, we also simulated the impact of two parameters on the free-riding situation. Finally, through simulation, the rationality of theoretical analysis is verified and more effective measures to prevent disease outbreaks is found.

Using growth mechanism and preferential attachment mechanism to generate a BA scale-free network, where the network has 100 nodes. We first randomly generate a network of 5 nodes, and then increase the nodes one by one. According to the preferential attachment rule, the newly generated node has priority to link with the node with greater degree. Five edges are added for each additional node. The adjacency matrix A is determined. According to theory 1, the transmission threshold of epidemic disease on scale-free network was calculated to be 0.0715.

Let the initial epidemic probability be 0.025. According to the classic SIS model, $\dot{I}_i = -I_i + \lambda(1 - I_i) \sum_{j=1}^{N} a_{ij}I_j(t)$, Figure 1 and Figure 2 are time series diagrams of infections diseases when infection rate λ is 0.06 and 0.08, respectively.



Figure 1. Time Series of classical SIS($\lambda = 0.06$).

We randomly selected three individuals and plotted their time series. To be more intuitive, we have also drawn a general time series diagram. In Figure 1, one can see that whether for an individual or for the population, the epidemic will eventually die out.

Figure 2 shows the time series diagram with the infection rate of 0.08. By comparing Figure 1 with Figure 2, it can be seen that if $\lambda > 0.0715$, epidemic will spread; if $\lambda < 0.0715$, the epidemic will die out.

Compared with the classical SIS transmission model, the epidemic transmission model coupled with human behavior game is more realistic. Different epidemics differ in the degree of infectivity,



Figure 2. Time Series of classical SIS($\lambda = 0.08$).

harmfulness, and difficulty in defense, so the corresponding benefit matrix varies. The payoff matrix is divided into four. The first kind: $u_{11} > u_{21}$, $u_{12} < u_{22}$. In this kind of payoff matrix, both collaborators or both betrayers are the best choice. This situation rarely occurs in the context of this article, so it is not considered in this article. The second kind: $u_{11} < u_{21}$, $u_{12} < u_{22}$. In this kind of payoff matrix, strategy *s* is batter choice than strategy *n* under any circumstances. That is to say, defensive strategy is not necessary during the epidemic or the cost of treatment is higher than the harm of the disease, we represent this payoff matrix in terms of M2. The third kind: $u_{11} < u_{21}$, $u_{12} > u_{22}$. In this kind of payoff matrix, it takes into account that one's neighbors' defense strategies will reduce his infection rate, so he will no longer has a defense strategy, and that is the case of free-rider, we will represent this payoff matrix in terms of M3. The fourth kind: $u_{11} > u_{21}$, $u_{12} > u_{22}$. It indicate that the defense strategy is low consumption and high yield, we will represent this revenue matrix in terms of M4.

In order to compare the epidemic transmission with the classical SIS model, the same infection rate was obtained for behavior n in the following simulation. Based on previous models, it's easy to know that when the disease dies out, human behavior will not change any more, but its state x_i won't necessarily go to 0 or 1. Let's first consider the case where the infection rate of behavioral n is 0.06, and the infection rate of behaviour s is 0.05. Without loss of generality, let a = 0.5, b = 0.5. We will analyze the effects of a and b on the disease later.

When we model human behavior, the benefits fall into two categories: individual-based risk assessment and neighbor-based replicator dynamics. In both cases, the size of the benefits should be uniform, so we assume that in the second kind of payoff matrix, we suppose that when people ignore the influence of their neighbors, the payoff of behavior n is greater than the payoff of behavior s. In the third and fourth kind of payoff matrix, we suppose that the payoff of behavior s is greater than the payoff of behavior n in the same situation.

In Figure 3, the three lines of each subgraph represent the time series of infectious disease dynamics of three selected individuals. Each subgraph corresponds to a kind of payoff matrix. Over time, the epidemic of each subgraph will eventually disappear. Figure 3 shows that from an individual's point of view, at this rate of transmission, infectious diseases eventually become extinct. The disappearance of some individual diseases does not mean the disappearance of all individual diseases. Next, we need to know if infectious diseases will go extinct in the whole population.



Figure 3. Individuals epidemic time series coupled with human behavior($\lambda_n = 0.06$).



Figure 4. Population epidemic time series coupled with human behavior($\lambda_n = 0.06$).

Figure 4 shows the prevalence of an epidemic by averaging the prevalence for all individual. On average, epidemics will eventually disappear. In other words, epidemics of all individuals will eventually become extinct. According to Figure 3 and Figure 4, when the transmission rate of an epidemic is less than the transmission threshold, the epidemic will eventually become extinct.

Next, we consider the case where the infection rate of behavior n is 0.08 and that of behavior s is 0.07. We perform the following numerical simulation:



Figure 5. Individuals epidemic time series coupled with human behavior($\lambda_n = 0.08$).

Figure 5 shows the infectious disease time series of the same three individuals as before in the case of three return matrices. In Figure 5, epidemic diseases can be effectively suppressed in the case of the fourth payoff matrix. By comparing 5a with 5b, it can be seen that 5a and 5b has different inhibitory effect on disease transmission, but which one is stronger will be analyzed by the average prevalence of all people.



Figure 6. Population epidemic time series coupled with human behavior($\lambda_n = 0.08$).

The comparison between 6a and 6b shows that the second payoff matrix has a stronger inhibitory effect on disease transmission than the third payoff matrix. As can be seen from Figures 6a and 6b, an epidemic still prevails when human behavior is taken into account. However, as can be seen from Figure 6c, the epidemic may be extinct when the defensive behavior is the best choice in any case.

In the personal behavior selection model, this paper considers individual-based and neighbor-based payoff assessments. a and b respectively indicate their weight in the behavior selection process. We set a + b = 1. In order to study the effectiveness of these two patterns in suppressing diseases, we did the following simulation.



Figure 7. Changes in the prevalence of epidemics with increasing *a*.

The abscissa of Figure 7 is the value of *a*, The ordinate represents the prevalence of the stable state of the disease corresponding to the value *a*. Figure 7 shows that as *a* increases, the steady state of the disease will decrease, which Shows that individual-based risk assessments have a stronger inhibitory effect on disease. Therefore, when developing strategies for disease control, countries should publicize more about the harmfulness of diseases and the benefits of prevention strategies.

Some people take into account that others' protective actions reduce the possibility of infection and therefore do not take protective actions, which is called hitchhiking. The following is a simulation of hitchhiking when *a* changes.

The simulation results show that when *a* increases, the free-riding situation becomes more serious. In other words, during the spread of the disease, when people ignore the influence of people around



Figure 8. Hitchhiking situation with increasing *a*.

them, the number of free-riders will gradually increase.

Through numerical simulation, we can conclude that epidemic models coupled with human behavior are more realistic. Through numerical simulation, we also know that the user's defensive behavior can well inhibit the spread of disease. During the period of disease transmission, countries can adopt strategies to reduce the consumption of defense strategies, thus curbing the spread of disease.

This article focuses on the classical SIS models that couple human behavior. Through simulation, we first verified the conclusion of the theoretical analysis. When the infection rate is greater than the epidemic threshold, the epidemic will prevail, and when the infection rate is less than the epidemic threshold, the epidemic will not prevail. When considering human behavior, we found that when the epidemic's infection rate is greater than the epidemic threshold, the epidemic may be suppressed due to human spontaneous protection measures. This article divides the incentives of human behavior into two types: individual-based risk assessment and neighbor-based replication dynamics. Through simulation, it is found that when the proportion of the former is greater than the latter, the effect of suppressing the spread of epidemics will be more obvious, but the number of free riders will gradually increase. Through analysis, we found that controlling people's benefits can effectively inhibit the spread of disease.

5. Discussion and conclusions

The co-evolution dynamics of disease and human behavior has always been a research hotspot. Game theory is an important tool for the study of human behavior. There are four types of strategy updates for most of their research: individual-based risk assessment, strategy-based risk assessment, direct commitment and modified replicator dynamics. These four strategy update methods consider imitation between people. Optimization is another method to study human behavior. The optimization method considers the maximization of its own benefits. There are many studies on these two methods, but few studies combine these two methods. For example, if we buy goods, we will not only shop around, but also consider its cost performance. Therefore, I think the research combining these two methods is more realistic.

In the previous models, the authors believe that individuals randomly choose neighbors and interact with them. In this article, I consider that everyone will have a certain understanding of the behavior of neighbors, especially in rural areas. Therefore, in the behavioral dynamics, I consider that individuals will compare the payoff with the average payoff of its neighbors and made a strategic choice. In this paper, the payoff matrix is classified according to the different diseases and regions. We study the spread of infectious diseases by analyzing different benefit models. The results suggest that human behavior can suppress disease outbreaks.

In previous articles, many people used vaccine coverage as a key measure of disease transmission. The simulation results in this paper show that with the increase of *a*, the spread of the disease is getting smaller and smaller, but there are more and more people hitchhiking. Our goal is to effectively control the spread of disease, so vaccine coverage cannot be used as the only key factor to measure the spread of disease.

In this article, we build a model that couples human behavior and epidemic spread. In the human behavior dynamics model, we considered individual-based risk assessment and neighbor-based replicator dynamics. We divide human strategy choices into two categories, s and n, and the payoff matrix into three categories. On scale-free networks, we analyzed the impact of human behavior on the spread of epidemics. Simulation results show that human behavior helps to suppress the spread of epidemics, and the government should deliberately guide human behavior.

The role of human behavior in suppressing disease epidemics is essential and has been studied by many scholars. How should the state intervene to influence people's behavior so as to achieve the best effect of curbing the disease is an urgent problem to be solved. At present, most researches focus on the innovation of methods, but few on the specific feasible strategies. For example, when an epidemic strikes, there is no specific plan for how much vaccine a country should distribute, how many immunization stations it should set up, who it should vaccinate, and so on. The research direction in the future may be more closely related to the reality, so we need to consider more practical issues when building the model

Because people have different levels of education and different psychological qualities, their cognition and response to epidemics will also be very different. In future work, we will consider classifying people according to their psychological endurance, etc., and defining different benefit matrices to study the impact of human behavior on disease transmission.

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Conflict of interest

The authors declare there is no conflict of interest in this paper.

References

- 1. F. Verelst, L. Willem, P. Beutels, Behavioural change models for infectious disease transmission: A systematic review (2010–2015), *J. R. Soc. Interface*, **13** (2016), 20160820.
- 2. J. T. F. Lau, X. L. Yang, H. Y. Tsui, E. Pang, SARS related preventive and risk behaviours practised by Hong Kong-mainland China cross border travellers during the outbreak of the SARS epidemic in Hong Kong, *J. Epidemiol. Community Health*, **58** (2004), 988–996.
- 3. J. T. Vietri, M. Li, A. P. Galvani, G. B. Chapman, Vaccinating to help ourselves and others, *Med. Decis. Making*, **32** (2012), 447–458.
- 4. S. Abdelmalek, S. Bendoukha, Global asymptotic stability of a diffusive SVIR epidemic model with immigration of individuals, *Electron. J. Differ. Equations*, **284** (2016), 1–14.
- 5. C. R. Cai, Z. X. Wu, J. Y. Guan, Behavior of susceptible-vaccinated-infected-recovered epidemics with diversity in the infection rate of individuals, *Phys. Rev. E*, **88** (2013), 062805.
- 6. C. Deng, H. J. Gao, Stability of SVIR system with random perturbations, *Inter. J. Biomath.*, **5** (2012).
- 7. T. C. Reluga, C. T. Bergstrom, Game theory of social distancing in response to an epidemic, *PLoS Comput. Biol.*, **6** (2010).
- 8. E. P. Fenichel, C. Castillo-Chavez, M. G. Ceddia, G. Chowell, P. A. G. Parra, G. J. Hickling, et al., Adaptive human behavior in epidemiological models, *Proc. Natl. Acad. Sci.*, **108** (2011), 6306–6311.
- 9. E. P. Fenichel, X. X. Wang, The mchanism and phenomena of adaptive human behavior during an epidemic and the role of information, in *Modeling the Interplay Between Human Behavior and the Spread of Infectious Diseases*, Springer, (2013), 153–168.
- 10. P. C. Zhu, Q. Zhi, Y. M. Guo, Z. Wang, Analysis of epidemic spreading process in adaptive networks, *IEEE Trans. Circuits Syst. II: Express Briefs*, **66** (2018), 1252–1256.
- 11. Y. Z. Zhou, Y. J. Xia, Epidemic spreading on weighted adaptive networks, *Phys. A*, **399** (2014), 16–23.
- 12. T. Li, X. D. Liu, J. Wu, C. Wan, Z. H. Guan, Y. M. Wang, An epidemic spreading model on adaptive scale-free networks with feedback mechanism, *Physica A*, **450** (2016), 649–656.
- 13. S. L. Chang, M. Piraveenan, P. Pattison, M. Prokopenko, Game theoretic modelling of infectious disease dynamics and intervention methods: A review, *J. Biol. Dyn.*, **14** (2019), 57–89.
- 14. H. Zhang, F. Fu, W. Zhang, B. Wang, Rational behavior is a double-edged sword when considering voluntary vaccination, *Phys. A*, **391** (2012), 4807–4815.
- 15. Y. Zhang, The impact of other-regarding tendencies on the spatial vaccination game, *Chaos Solitons Fractals*, **56** (2013), 209–215.
- 16. Q. Li, M. C. Li, L. Lv, C. Guo, K. Lu, A new prediction model of infectious diseases with vaccination strategies based on evolutionary game theory, *Chaos Solitons Fractals*, **104** (2017), 51–60.
- 17. K. Kuga, J. Tanimoto, Which is more effective for suppressing an infectious disease: Imperfect vaccination or defense against contagion?, *J. Stat. Mech. Theory Exp.*, **2018** (2018), 023407.

- 18. K. M. A. Kabir, K. Kuga, J. Tanimoto, Effect of information spreading to suppress the disease contagion on the epidemic vaccination game, *Chaos Solitons Fractals*, **119** (2019), 180–187.
- 19. C. T. Bauch, D. J. D. Earn, Vaccination and the theory of games, *Proc. Natl. Acad. Sci.*, **101** (2004), 13391–13394.
- 20. T. Dominic, R. Mary, V. H. A. Jan, W. J. Edmunds, R. Vivancos, A. Bukasa, et al., The effect of measles on health-related quality of life: A patient-based survey, *PLoS ONE*, **9** (2014).
- 21. X. Feng, B. Wu, L. Wang, Voluntary vaccination dilemma with evolving psychological perceptions, *J. Theor. Biol.*, **439** (2018), 65–75.
- K. M. A. Kabir, J. Tanimoto, Evolutionary vaccination game approach in metapopulation migration model with information spreading on different graphs, *Chaos Solitons Fractals*, 120 (2019), 41–55.
- 23. K. M. A. Kabir, M. Jusup, J. Tanimoto, Behavioral incentives in a vaccination-dilemma setting with optional treatment, *Phys. Rev. E*, **100** (2019), 062402.
- K. Kuga, J. Tanimoto, M. Jusup, To vaccinate or not to vaccinate: A comprehensive study of vaccination-subsidizing policies with multi-agent simulations and mean-field modeling, *J. Theor. Biol.*, 469 (2019), 107–126.
- 25. M. R. Arefin, K. M. A. Kabir1, J. Tanimoto. A mean-field vaccination game scheme to analyze the effect of a single vaccination strategy on a two-strain epidemic spreading, *J. Stat. Mech. Theory Exp.*, **2020** (2020), 033501.
- 26. P. Poletti, B. Caprile, M. Ajelli, A. Pugliese, S. Merlera, Spontaneous behavioural changes in response to epidemics, *J. Theor. Biol.*, **260** (2009), 31–40.
- 27. P. S. Romualdo, V. Alessandro, Epidemic spreading in scale-free networks, *Phys. Rev. Lett.*, **86** (2001), 3200–3203.
- 28. A. B. M. Nasiruzzaman, M. N. Akter, M. A. Mahmud, H. R. Pota, Exploration of power flow distribution to reveal scale-free characteristics in power grids, 2017 IEEE Power & Energy Society General Meeting, 2017. Available from: https://ieeexplore.ieee.org/abstract/document/8273917.
- 29. A. L. Barabasi, R. Albert, H. Jeong, Scale-free characteristics of random networks: the topology of the world-wide web, *Physica A*, **281** (2000), 69–77.
- 30. K. Dharshana, P. Mahendra, Emergence of scale-free characteristics in socio-ecological systems with bounded rationality, *Sci. Rep.*, **5** (2015), 10448.
- 31. P. Yang, Z. P. Xu, J. Feng, X. Fu, Feedback pinning control of collective behaviors aroused by epidemic spread on complex networks, *Chaos*, **29** (2019).
- 32. K. Li, Z. Ma, Z. Jia, M. Small, X Fu, Interplay between collective behavior and spreading dynamics on complex networks, *Chaos*, **29** (2012), 043113



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