The Impacts of Testing Susceptible and Exposed Individuals on Emerging Infectious Diseases with Infectiousness in Incubation Period

Jiajun Ding¹, Liyan Gao^{2,3,*}, Qiuhui Pan^{2,4}, and Mingfeng He^{2,4}

¹ School of Foreign Languages, Dalian University of Technology, Dalian, China

² School of Mathematical Science, Dalian University of Technology, Dalian, China

³ School of Physics, Dalian University of Technology, Dalian, China

⁴ School of Innovation and Entrepreneurship, Dalian University of Technology, Dalian, China

Email: 1833262712@qq.com (J.J.D.); lygao@dlut.edu.cn (L.Y.G.); qhpan@dlut.edu.cn (Q.H.P.);

mfhe@dlut.edu.cn (M.F.H)

*Corresponding author

Abstract—On the premise that both susceptible and exposed individuals in incubation period are asymptomatic, in this paper, we establish epidemic models with compulsory testing or voluntary testing to study the impact of susceptible and exposed individuals' testing rate on the emerging infectious diseases. Results show that when compulsory testing is performed on individuals, sometimes the testing rate of susceptible and exposed individuals can be increased to prevent epidemic from spreading among the population. Sometimes both the testing rate of susceptible, exposed and symptomatic infected individuals need to reach a certain level, and sometimes it can be achieved by increasing the testing rate of symptomatic infected individuals. When individuals take voluntary testing, if individuals think the risk of epidemic is low, increasing the individual's trust in medical treatment is conducive to increasing the testing number of exposed individuals. However, if individuals think the risk is high, the change in medical trust can not affect the testing number of exposed individuals. Comparing these two models, it is interesting to note that the epidemic size corresponding to average testing rate of voluntary testing is almost the same as the epidemic size corresponding to the same testing rate of compulsory testing, reflecting the effectiveness of voluntary testing.

Keywords—susceptible and exposed individuals, testing behavior, emerging infectious diseases, epidemic model, game theory

I. INTRODUCTION

Emerging infectious diseases refer to infectious diseases that have emerged in the past 20 years, including infectious diseases caused by newly discovered pathogens or pathogens that infect new groups, as well as re-emerging infectious diseases [1]. Human beings lack awareness of emerging infectious diseases and natural immunity, which causes harm to human health and brings economic losses to society. Therefore, research

institutions have strengthened research on emerging infectious diseases. So far, major emerging infectious diseases that have occurred around the world include AIDS, Severe Acute Respiratory Syndrome (SARS), influenza A H1N1, Ebola virus, and Middle East Respiratory Syndrome (MERS).

Take COVID-19 as an example, exposed individuals do not show symptoms and are infectious, which makes epidemic prevention and control more difficult. The current research on such emerging infectious diseases with incubation period is mainly focused on the impact of some prevention and control measures such as closing the city and restricting personnel contact on the epidemic [2– 7]. Susceptible and exposed individuals can be confirmed by testing, and their testing behavior can be governmentdriven compulsory behavior or individual-driven voluntary behavior. Compulsory behavior is testing a fixed percentage of susceptible and exposed individuals. Voluntary behavior means that you need to bear the cost for testing, but you will not get timely treatment without testing. Therefore, susceptible and exposed individuals will make a comprehensive judgment to determine whether to test or not. The method of comprehensive judgment is to use the game model to calculate the average payoff and use the Fermi equation to give the judgment result. By studying the impact of compulsory testing and voluntary testing on emerging infectious diseases with infectiousness in the incubation period, this paper hopes to provide suggestions and theoretical support for the government to effectively prevent and control such infectious diseases.

II. LITERATURE REVIEW

The compartment model is a powerful mathematical framework for understanding the complex dynamics of epidemic. According to the disease state, the SIS model can be defined, that is, infected individuals who have recovered are at risk of becoming infected again [8]. Authors of Refs. [9–11] established the SIR model, which

Manuscript received December 26, 2023; revised February 7, 2024; accepted March 13, 2024; published September 24, 2024.

has been widely used in epidemiology. Based on this, various epidemic models have been proposed, such as SEIR [12, 13], SEIQR [14], and MSEIR [15] models.

As a tool for optimizing individual behavior, game theory has been applied in many fields such as biology to study the reasons why cooperative behavior appears in nature [16–21]. Recently, researchers have combined the game process with the spread of epidemic and discussed issues such as voluntary vaccination [22–27]. It takes time and money for vaccinators so that they are free from disease with a large likelihood. Self-interested individuals try to benefit from herd immunity while avoiding vaccination. Consequently, such hitchhiking leads herd immunity is inevitably disturbed [28–37]. The application of evolutionary game theory in vaccination research not only provides a mathematical means to explain epidemiological dynamics, but also explains the voluntary vaccination behavior of individuals [38–42]. Fu et al. [43] studied the role of individual imitation behavior and population structure in vaccination. Kabir and Tanimoto [44] explored how the spread of contagious diseases can be reduced with intermediate defense measures taken up with two strategy-update rules: individual based or strategy based.

At present, few scholars analyze the influence of individual testing behavior on emerging infectious diseases from the perspective of game theory. In order to study this problem, this paper establishes the compulsory testing model and the voluntary testing game model respectively, and compares the results of epidemic transmission based on the two models.

III. MATERIALS AND METHODS

A. Epidemic Model of Compulsory Testing

Consider the emerging infectious diseases with infectiousness in the incubation period, such as COVID-19. The individuals in a population can be classified into susceptible (S) , exposed (I_A) , symptomatic infected individuals (I_S) , hospitalized (H) , recovered (R) , and death (D) . Exposed individuals in the incubation period will not show symptoms, but can spread the virus. Susceptible will be infected with probability β in contact with the exposed individuals or the symptomatic infected individuals. Exposed individuals are less infectious than symptomatic infected individuals, let η be the decreasing proportion of infectivity in the incubation period [5]. The incidence rate of exposed individuals is α , and then become symptomatic infected individuals. Both the susceptible and exposed individuals have no symptoms. It is impossible to distinguish the epidemiological status of these individuals without medical means. Furthermore, the exposed individuals can infect the susceptible, it is particularly important to test susceptible and exposed individuals, and to isolate those who are infectious. Since the test behavior of susceptible individuals have no effect on the spread of infectious diseases, there is no need to reflect the test process of susceptible individuals. Compulsory testing is a government-driven behavior, a fixed rate of susceptible and exposed individuals will be tested, and those who are diagnosed as infected must be hospitalized for isolation. Suppose the testing rate of susceptible and exposed individuals is m , and the testing rate of symptomatic infected individuals is u . The self-healing rate of symptomatic infected individuals is r_{I_s} and the mortality rate is d_{I_s} . The recovery rate of hospitalized is r_H and the mortality rate is d_H . Use the compartment model to describe the spread of infectious diseases. The transformation relationship of populations is shown in Fig. 1.

Fig. 1. Diagram of population transformation.

Based on the above transformation diagram, the differential equation model can be established as follows:

$$
\dot{S} = -\beta S(\eta I_A + I_S),
$$
\n
$$
\dot{I}_A = \beta S(\eta I_A + I_S) - mI_A - \alpha I_A,
$$
\n
$$
\dot{I}_S = \alpha I_A - uI_S - (r_{I_S} + d_{I_S})I_S,
$$
\n
$$
\dot{H} = mI_A + uI_S - (r_H + d_H)H,
$$
\n
$$
\dot{R} = r_{I_S}I_S + r_HH,
$$
\n
$$
\dot{D} = d_{I_S}I_S + d_HH.
$$
\n(1)

Initial values are $S(0) = N_0 = 8 \times 10^7$, $I_A(0) = 1$, $I_s(0) = 0$, $H(0) = 0$, $R(0) = 0$, $D(0) = 0$.

B. Epidemic Model of Voluntary Testing

When the testing behavior of susceptible and exposed individuals is individual-driven voluntary behavior, individuals make decisions without knowing whether they are infected or not. It often depends on certain psychological factors, such as testing cost, the necessity of timely treatment, and so on. In the following, we will use evolutionary game theory to describe this problem.

Since neither the susceptible nor the exposed individuals exhibit symptoms, they can choose test (C) or not test (D) . Individuals who choose strategy C need to bear the testing cost $c \mid c \in [0,1]$). The susceptible who chose strategy D have no loss. The exposed individuals who adopt strategy C need to pay the testing cost, and meanwhile receive the benefit r from the treatment opportunity, which is called the cure benefit. The exposed individuals who adopt strategy D will face

the harm of aggravated illness and cause damage to the health, which is represented by the risk cost d . The corresponding payoff structure are listed in Table I. Assuming that the testing cost is less than the benefit of treatment opportunities, and less than the risk of aggravating the disease.

TABLE I. PAYOFF STRUCTURE

Strategy	Payoff
$C_{\rm S}$	$-c$
C_I	$-c+r$
D_S	0
${}^D\!I_A$	$-d$

Here, we only consider the voluntary behavior of susceptible and exposed individuals, and the symptomatic infected individuals still maintain compulsory behavior. Susceptible and exposed individuals cannot distinguish their epidemiological identity. They can obtain the cumulative testing number of susceptible and exposed individuals C_{S+I_A} and the cumulative diagnosed number of exposed individuals C_{I_A} . From this, susceptible and exposed individuals can estimate the average payoff of strategy C and D , respectively.

$$
\Pi_C = (1 - \frac{C_{I_A}}{C_{S+I_A}})(-c) + \frac{C_{I_A}}{C_{S+I_A}}(r-c)
$$
 (2)

$$
\Pi_{D} = -d \frac{C_{I_{A}}}{C_{S+I_{A}}} \tag{3}
$$

Susceptible and exposed individuals determine the behavior according to the payoff, assuming that the testing rate of susceptible and exposed individuals is:

$$
m = \begin{cases} \frac{1}{1 + \exp(\Pi_D - \Pi_C)/K}, & I(t) \neq 0, \\ 0, & I(t) = 0. \end{cases}
$$
 (4)

where $K > 0$.

IV. RESULT AND DISCUSSION

A. Result of Compulsory Testing

Eq. (1) has two disease-free equilibrium points. One corresponding to $P_0^1 = (S_0^1, 0, 0, 0) = (N_0, 0, 0, 0)$, epidemic will not break out. Another corresponding to case in which the epidemic has ended, which is recorded as $P_0^2 = (S_0^2, 0, 0, 0)$ 0 $P_0^2 = (S_0^2, 0, 0, 0).$

We use the next generation matrix method to calculate the basic reproduction number of model (1). \mathcal{F} represents the matrix of emerging infected diseases, ν represents the transition matrix between the epidemic equations. It can be obtained from model (1):

$$
\mathcal{F} = \begin{bmatrix} \beta S(\eta I_A + I_S) \\ 0 \\ 0 \\ 0 \end{bmatrix}, \mathcal{V} = \begin{bmatrix} mI_A + \alpha I_A \\ uI_S + (r_I_A + d_I)^I_S - \alpha I_A \\ \beta S(\beta I_A + I_S) \\ \beta S(\beta I_A + I_S) \\ (r_H + d_H)H - mI_A - uI_S \end{bmatrix}.
$$

The Jacobian matrix of $\mathcal F$ and $\mathcal V$ at the disease-free equilibrium point are:

$$
F = \begin{bmatrix} \beta S_0 \eta & \beta S_0 \\ 0 & 0 \end{bmatrix}, V = \begin{bmatrix} m + \alpha & 0 \\ -\alpha & u + r_{I_s} + d_{I_s} \end{bmatrix}.
$$

Then the next generation matrix is:

$$
F V^{-1} = \begin{bmatrix} \frac{\beta S_0 \eta (u + r_{I_s} + d_{I_s}) + \beta S_0 \alpha}{(m + \alpha)(u + r_{I_s} + d_{I_s})} & \frac{\beta S_0}{u + r_{I_s} + d_{I_s}} \\ 0 & 0 \end{bmatrix}.
$$

According to the literature [45], the maximum spectral radius is known, that is, the basic reproduction number is:

$$
R_0 = \rho(FV^{-1}) = \frac{\beta S_0 \eta(u + r_{I_s} + d_{I_s}) + \beta S_0 \alpha}{(m + \alpha)(u + r_{I_s} + d_{I_s})}.
$$

Take the parameters of model (1) as shown in Table II.

TABLE II. THE PARAMETERS OF MODEL (1)

Parameter	Define	Value
β	infection rate	$[2 \times 10^{-9}, 5 \times 10^{-9}]$
η	decreasing proportion of infectivity	[0,1]
α	incidence rate	[0,1]
\boldsymbol{m}	testing rate of susceptible and exposed individuals	[0,1]
\boldsymbol{u}	testing rate of symptomatic infected individuals	[0,1]
r_{I_S}	self-healing rate of symptomatic infected individuals	0.027
dI_S	mortality rate of symptomatic infected individuals	0.003
r_H	recovery rate of hospitalized	0.049
d_H	mortality rate of hospitalized	0.001

The effect of the testing rate of susceptible and exposed individuals m and the testing rate of symptomatic infected individuals u on the basic reproduction number is shown in Fig. 2.

Fig. 2. The effect of testing rate of susceptible and exposed individuals m and the testing rate of symptomatic infected individuals u on the basic reproduction number.

In Fig. 2, there is a curve that divides the $u - m$ space into two parts, namely $R_0 > 1$ (Area I) and $R_0 < 1$ (Area II). Epidemic will break out, if (m, u) is in Area I. The combination of (m, u) in Area II will prevent epidemic from breaking out. The specific values correspond to various emerging infectious diseases. Fig. 2(a) shows that when $u < 0.029$, no matter what the value of *m* is, epidemic can break out. When $u > 0.529$, no matter what the value of m is, the epidemic will not break out. With the increase of m , the critical value of u that enables epidemic break out gradually decreases. In other words, in order to prevent the spread of the disease, increasing the testing rate of symptomatic infected individuals can tolerate a smaller testing rate of susceptible and exposed individuals. Considering that the number of symptomatic infected individuals is much smaller than the total number of susceptible and exposed individuals, increasing the testing rate of symptomatic infected individuals is an effective measure to prevent the spread of the epidemic. Fig. 2(b) shows that when $m > 0.898$, no matter what the value of u is, the epidemic cannot spread. When $m < 0.057$, no matter what the value of u is, epidemic can spread. This reveals that even if all symptomatic infected individuals are tested, if susceptible and exposed individuals are negatively tested, there is no guarantee that the epidemic can be controlled. Fig. 2(c) shows that when $m < 0.047$, no matter what the value of u is, epidemic can break out. When $u < 0.065$, no matter what the value of *m* is, epidemic can break out. This means that under this set of parameters, even if all the susceptible and exposed individuals are tested, if the symptomatic infected individuals are negatively tested, the epidemic cannot be controlled. Fig. 2(d) shows that when $u > 0.77$, no matter what the value of m is, no epidemic will be spread. When $m > 0.505$, no matter what the value of u is, no epidemic will be spread. This indicates that when the testing rate of symptomatic infected individuals is large, even if susceptible and exposed individuals are not tested, the epidemic can be controlled. Similarly, when the testing rate of susceptible and exposed individuals is large, even if symptomatic infected individuals are not tested, it can also control the epidemic.

The incidence rate of exposed individuals, the testing rate of susceptible and exposed individuals will directly affect the number of exposed individuals, thus affecting the transmission process of infectious disease. Therefore, in the following, the influence of the incidence rate α , the testing rate of susceptible and exposed individuals m on the basic regeneration number is studied. The results are shown in Fig. 3.

Fig. 3. When $(\beta, \eta) = (3.3 \times 10^{-9}, 0.5)$, the effect of incidence rate of exposed individuals α , the testing rate of susceptible and exposed individuals m on the basic reproduction number.

In Fig. 3, there is also a curve that divides the $\alpha - m$ space into two parts, namely $R_0 > 1$ and $R_0 < 1$. When $\alpha > 0.66$, no matter what the value of *m* is, the epidemic cannot break out. The higher the incidence of exposed individuals, the shorter the transition time from exposed individuals to symptomatic infected individuals. The short incubation period makes it possible for individuals to be detected and isolated in time after infection, thus reducing the epidemic size. It can be seen that emerging infectious diseases with a long incubation period are difficult to control, while emerging infectious diseases with a short incubation period are easy to control.

When $R_0 > 1$, its value will affect the epidemic's final size. Let $\beta = 3.3 \times 10^{-9}$, $\eta = 0.5$, $\alpha = 0.25$, $u = 0.3$, $m \in \{0.0099, 0.0265, 0.05, 0.0697\}$, time courses of the epidemic size ($N_0 - S$) is shown in Fig. 4.

Fig. 4. Time courses of the epidemic size, when $u=0.3$, $m{\in} \{0.0099,0.0265,0.05,0.0697\}$.

From Fig. 4, we can find that the greater the testing rate of susceptible and exposed individuals, the smaller the value of R_0 , the slower the spread of epidemic. And the time for the epidemic size to reach stable value is later, the epidemic final size is smaller. Therefore, increasing the testing rate of susceptible and exposed individuals can delay the peak period of epidemic and reduce the

epidemic size, but it will prolong the duration of the epidemic.

By testing susceptible and exposed individuals, exposed individuals hidden in social environments can be screened out. The cumulative testing number of exposed individuals is recorded as C_{I_A} . We show the C_{I_A} versus the testing rate of susceptible and exposed individuals under different values of u in Fig. 5.

Fig. 5. When $u \in \{0.25, 0.3, 0.35, 0.4\}$, C_{I_A} versus the testing rate of susceptible and exposed individuals.

It can be seen from Fig. 5 that for each given value of u , there is a testing rate m_u^* that can achieve the maximum cumulative testing number of exposed individuals. When $m < m_u^*$, it can be considered insufficient testing, so that there are more exposed individuals in the population. We can screen out more exposed individuals by increasing the testing rate. When $m > m_u^*$, it can be considered over-testing. At this time, the testing rate of exposed individuals is high, but the proportion of exposed individuals in the population is small. Therefore, by increasing the testing rate, no more exposed individuals will be screened out. It can also be seen from Fig. 5 that as the value of u increases, the value of m_u^* decreases. We can say that when the testing rate of symptomatic infected individuals is high, the testing rate of susceptible and exposed individuals can be reduced to screen out the maximum number of exposed individuals.

B. Result of Voluntary Testing

Let $K = 0.1$, $C_{S+I_A}(0) = 1$, $C_{I_A}(0) = 0$, Fig. 6 shows the time courses of various compartments and testing rate $m(t)$ when $\beta = 3.3 \times 10^{-9}$, $\eta = 0.5$, $\alpha = 0.25$, $r = 5$, $d = 10$, $c = 0.5$, $u = 0.3$.

It can be seen from Fig. $6(a)$ that the susceptible individuals gradually decrease with time, and finally reach a stable state. In Fig. 6(b), there are peaks in the number of I_A , I_S , and H . The peak times of these three groups are relatively close. We assume that the epidemic ends when there are no exposed and symptomatic infected individuals in the population. Therefore, under this set of parameters, the epidemic lasted for 676 days. Fig. 6(c) shows that the number of

recovered and deaths individuals gradually increase to a stable state as the epidemic spreads. In Fig. 6(d), the testing rate $m(t)$ of susceptible and exposed individuals goes up from 0.0067 and then drops after reaching its maximum value. When the epidemic is over, stop testing, $m(t) = 0$. Therefore, we can calculate the average testing rate of susceptible and exposed individuals during the transmission cycle of epidemic is $\overline{m} = 0.0099$. It can be seen that even if the testing cost is very low, the testing rate of susceptible and exposed individuals is always within a very low value range.

Fig. 6. When $c=0.5$, $u=0.3$, the evolution of various compartments and the testing rate of susceptible and exposed individuals.

To study the effects of testing cost on the epidemic, we examine the changes of $m(t)$ and $N_0 - S(t)$ in Fig. 7.

Fig. 7. When $u=0.3$, the evolution of testing rate $m(t)$ and epidemic size $N_0 - S(t)$.

The numerical results in Fig. 7(a) show that the lower the testing cost, the higher the testing rate of susceptible and exposed individuals, and the longer the duration of the epidemic. Fig. 7(b) shows that the smaller the testing cost, the smaller the epidemic size. The effect of adjusting testing rate can be achieved by adjusting the value of c . By comparing Fig. 7(b) and Fig. 4, it is interesting to find that the epidemic size corresponding to the average testing rate of voluntary testing is almost the same as the epidemic size corresponding to the same testing rate of compulsory testing. Epidemic can be controlled through voluntary testing.

When $u \in \{0.25, 0.3, 0.35, 0.4\}$, the evolution of the cumulative testing number of exposed individuals C_{I_A} with respect to testing cost c is shown in Fig. 8.

Fig. 8. The evolution of the cumulative testing number of exposed individuals C_{I_A} with respect to testing cost c.

As can be seen from Fig. 8, when the testing cost is small, the testing rate of susceptible and exposed individuals is high, so that the epidemic cannot spread. With the increases of c , the testing rate decreases, so the epidemic can spread in the system. Exposed individuals can be tested within this parameter range, so the cumulative testing number of exposed individuals increases. When $c = c_u^*$, the maximum number of exposed individuals can be tested. However, when $c > c_u[*]$, the situation changes. Although the fraction of exposed individuals in the system is large, the testing rate is too small because of the high testing cost. And that's what makes the cumulative testing number of exposed individuals go down. In addition, it can be seen that the value of c_u^* increases with the increases of u .

In the following, the results of the testing rate $m(t)$ are shown in Fig. 9.

Fig. 9. The results of the testing rate $m(t)$ when $\beta = 3.3 \times 10^{-9}$, $\eta = 0.5$, $\alpha = 0.25$, $c = 0.5$, $u = 0.3$.

The numerical examples presented in Fig. 9 indicate that in the early stage of the epidemic, neither the cure benefit nor the risk cost has an impact on the testing rate of susceptible and exposed individuals. When the epidemic is in a phase of rampant spread, cure benefit, and risk cost will have effects. The greater the cure benefit and risk cost, the higher the testing rate of susceptible and exposed individuals. In other words, the optimistic expectation of being cured and a high risk awareness of the epidemic will increase the testing rate of voluntary testing and put the prevention and control of the epidemic in a proactive state. High cure benefit is a manifestation of trust in medical treatment. The high risk cost reflects the risk awareness of emerging infectious diseases.

When $r \in \{2,4,6,8\}$, the value of C_{I_A} corresponding to the change of d , as shown in Fig. 10.

Fig. 10. The value of C_{I_A} corresponding to the change of d.

In Fig. 10, under different cure benefits, the cumulative testing number of exposed individuals increases with the risk cost, and then stabilizes at a higher level. When the risk cost is low, the higher the cure benefit, the more the cumulative testing number of exposed individuals. When the risk cost is higher, the value of the cure benefit does not affect the cumulative testing number of exposed individuals. Therefore, we can say that when individual's risk awareness of emerging infectious diseases is in a low range, the trust in medical treatment is high, which is conducive to increasing the testing number of susceptible and exposed individuals and testing more exposed individuals. However, when the risk awareness of emerging infectious disease is in a high range, the change of the trust in medical treatment does not affect the testing number of exposed individuals.

V. CONCLUSION

For emerging infectious diseases with infectiousness in incubation period, it is an effective way to screen out exposed individuals in time. The testing behavior of susceptible and exposed individuals may be governmentdriven compulsory behavior and test a fixed percentage of susceptible and exposed individuals. In this paper, we propose the epidemic model of compulsory testing. Results show that corresponding to different characteristics of emerging infectious diseases, the value range of the testing rate of symptomatic infected individuals, and the value range of the testing rate of susceptible and exposed individuals that enables infectious diseases to be controlled are different. In order to prevent the spread of the epidemic, sometimes the testing rate of susceptible and exposed individuals can be increased to prevent the epidemic from spreading among the population. Sometimes both the testing rate of symptomatic infected individuals, susceptible and exposed individuals need to reach a certain level, and sometimes it can be achieved by increasing the testing rate of symptomatic infected individuals. The common feature is that, in order to keep the epidemic from spreading, increasing the testing rate of symptomatic infected individuals can tolerate lower testing rate of susceptible and exposed individuals. The study found that increasing the testing rate of susceptible and exposed

individuals could delay the peak of the epidemic and reduce the epidemic size, but prolong the duration of the epidemic. For each testing rate of symptomatic infected individuals, there is a testing rate of susceptible and exposed that can test the maximum number of exposed individuals.

The testing behavior of susceptible and exposed individuals can also be individual-driven voluntary behavior. This article assumes that susceptible and exposed individuals will determine the testing intention based on the comprehensive judgment of some factors such as the testing cost, the possibility of being cured, and so on. Calculate the testing rate according to the payoff. Based on this rate, the epidemic model of voluntary testing is established. Results show that reducing the testing cost can improve the testing rate of susceptible and exposed individuals, reduce the transmission speed of the epidemic, and reduce the epidemic size. In addition, for each given testing rate of symptomatic infected individuals, there is a testing cost that can test the maximum number of exposed individuals. When the risk awareness is low, it is beneficial to increase the testing number of exposed individuals by increasing the trust in medical treatment. However, when the risk awareness is high, the change of the individual's trust in medical treatment does not affect the testing number of exposed individuals.

Comparing these two models, we find that the epidemic size of compulsory testing is comparable to the voluntary testing. It shows that voluntary testing is an effective measure to curb the spread of epidemic.

This article focuses on the testing behavior of susceptible and exposed individuals. Currently, compulsory testing and voluntary testing are considered separately. In actual epidemic prevention and control work, it is often more reasonable for close contacts to take compulsory testing measure, while take voluntary testing measure for non-close contacts. This will be studied in the subsequent work.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

AUTHOR CONTRIBUTIONS

Jiajun Ding conducted the research; Liyan Gao wrote the paper; Qiuhui Pan and Mingfeng He refined the ideas; all authors had approved the final version.

FUNDING

This research was funded by Postdoctoral Fellowship Program of CPSF, grant number GZC20230367.

REFERENCES

- [1] J. Lederberg, R. E. Shope, and S. C. Oaks, Emerging Infection: Microbial Threats to Health in the United States, Washington (DC): National Academy Press, 1992.
- [2] Q. Y. Lin, S. Zhao, D. Z. Gao, et al., "A conceptual model for the coronavirus disease 2019 (COVID-19) outbreak in Wuhan, China with individual reaction and governmental action," International Journal of Infectious Diseases, vol. 93, pp. 211–216, 2020.
- [3] H. Joel, A. Sam, G. Amy, et al., "Feasibility of controlling COVID-19 outbreaks by isolation of cases and contacts," The Lancet Global Health, vol. 8, pp. e488–e496, 2020.
- [4] B. Tang, F. Xia, S. Y. Tang, et al., "The effectiveness of quarantine and isolation determine the trend of the COVID-19 epidemics in the final phase of the current outbreak in China," International Journal of Infectious Diseases, vol. 96, pp. 636–647, 2020.
- [5] Z. F. Yang, Z. Q. Zeng, K. Wang, et al., "Modified SEIR and AI prediction of the epidemics trend of COVID-19 in China under public health interventions," Journal of Thoracic Disease, vol. 12, pp. 165–174, 2020.
- [6] K. Prem, Y. Liu, T. W. Russell, et al., "The effect of control strategies to reduce social mixing on outcomes of the COVID-19 epidemic in Wuhan, China: A modelling study," The Lancet Public Health, vol. 5, pp. $e261 - e270$, 2020 .
- [7] C. R. Wells, P. Sah, S. M. Moghadas, et al., "Impact of international travel and border control measures on the global spread of the novel 2019 coronavirus outbreak," Proceedings of the National Academy of Sciences of the United States of America, vol. 117, pp. 7504–7509, 2020.
- M. Zhu, Z. G. Lin, and Q. Y. Zhang, "Coexistence of a crossdiffusive dengue fever model in a heterogeneous environment,' Computers and Mathematics with Applications, vol. 75, pp. 1004– 1015, 2018.
- [9] W. O. Kermack and A. G. McKendrick, "A contribution to the mathematical theory of epidemics," Proceedings of the Royal Society of London, vol. 115, pp. 700–721, 1927.
- [10] Z. Y. He, A. Abbes, H. Jahanshahi, et al., "Fractional-order discrete-time SIR epidemic model with vaccination: chaos and complexity," Mathematics, vol. 10, p. 165, 2022.
- [11] M. Bohner, G. Stamov, I. Stamova, et al., "On h-manifolds stability for impulsive delayed SIR epidemic models," Applied Mathematical Modelling, vol. 118, pp. 853–862, 2023.
- [12] P. E. Lekone and B. F. Finkenstadt, "Statistical inference in a stochastic epidemic SEIR model with control intervention: Ebola as a case study," Biometrics, vol. 62, pp. 1170–1177, 2006.
- [13] P. Diaz, P. Constantine, K. Kalmbach, et al., "A modified SEIR model for the spread of Ebola in Western Africa and metrics for resource allocation," Applied Mathematics and Computation, vol. 324, pp. 141–155, 2018.
- [14] H. Kang, K. Liu, and X. Fu, "Dynamics of an epidemic model with quarantine on scale-free networks," Physics Letters A, vol. 381, pp. 3945–3951, 2017.
- [15] H. A. Asfour and M. Ibrahim, "On the differential fractional transformation method of MSEIR epidemic model," International Journal of Computer Applications, vol. 113, pp. 10–16, 2015.
- [16] H. Ito and J. Tanimoto, "Scaling the phase-planes of social dilemma strengths shows game-class changes in the five rules governing the evolution of cooperation," Royal Society Open Science, vol. 5, 181085, 2018.
- [17] H. Ito and J. Tanimoto, "Dynamic utility: The sixth reciprocity mechanism for the evolution of cooperation," Royal Society Open Science, vol. 7, 200891, 2020.
- [18] G. Greenwood and D. Ashlock, "A comparison of the Moran process and replicator equations for evolving social dilemma game strategies," Biosystems, vol. 202, 104352, 2021.
- [19] Z. Y. Shi, W. Wei, M. Perc, et al., "Coupling group selection and network reciprocity in social dilemmas through multilayer networks," *Applied Mathematics and Computation*, vol. 418, 126835, 2022.
- [20] Y. L. Zhang, "Structured heterogeneity of network in evolutionary social dilemmas under assortative linking and learning," Physica D: Nonlinear Phenomena, vol. 454, 133846, 2023.
- [21] H. W. Lee, C. Cleveland, and A. Szolnoki, "Restoring spatial cooperation with myopic agents in a three-strategy social dilemma," Applied Mathematics and Computation, vol. 458, 128226, 2023.
- [22] K. Kuga and J. Tanimoto, "Which is more effective for suppressing an infectious disease: Imperfect vaccination or defense against contagion," Journal of Statistical Mechanics: Theory and Experiment, vol. 2018, 023407, 2018.
- [23] B. Y. Shi, G. L. Liu, H. J. Qiu, et al., "Exploring voluntary vaccination with bounded rationality through reinforcement learning," Physica A: Statistical Mechanics and Its Applications, vol. 515, pp. 171–182, 2019.
- [24] M. Alam, K. M. Kabir, and J. Tanimoto, "Based on mathematical epidemiology and evolutionary game theory, which is more effective: quarantine or isolation policy," Journal of Statistical Mechanics: Theory and Experiment, vol. 2020, 033502, 2020.
- [25] M. Arefin, K. M. Kabir, and J. Tanimoto, "A mean-field vaccination game scheme to analyze the effect of a single vaccination strategy on a two-strain epidemic spreading," Journal of Statistical Mechanics: Theory and Experiment, vol. 2020, 033501, 2020.
- [26] I. Nishimura, M. Arefin, Y. Tatsukawa, et al., "Social dilemma analysis on vaccination game accounting for the effect of immunity waning," Chaos, Solitons & Fractals, vol. 171, 113426, 2023.
- [27] J. R. Wang, H. Z. Zhang, X. Jin, et al., "Subsidy policy with punishment mechanism can promote voluntary vaccination behaviors in structured populations," Chaos, Solitons & Fractals, vol. 174, 113836, 2023.
- [28] E. Fukuda, S. Kokubo, J. Tanimoto, et al., "Risk assessment for infectious disease and its impact on voluntary vaccination behavior in social networks," Chaos, Solitons & Fractals, vol. 68, pp. 1–9, 2014.
- [29] G. Fukuda and J. Tanimoto, "Effects of stubborn decision-makers" on vaccination and disease propagation in social networks," International Journal of Automation and Logistics, vol. 2, pp. 78– 92, 2016.
- [30] K. M. Kabir and J. Tanimoto, "Modelling and analysing the coexistence of dual dilemmas in the proactive vaccination game and retroactive treatment game in epidemic viral dynamics," Proceedings of the Royal Society A, vol. 475, pp. 1-20, 2019.
- [31] K. M. Kabir, M. Jusup, and J. Tanimoto, "Behavioral incentives in a vaccination-dilemma setting with optional treatment," Physical Review E, vol. 100, 062402, 2019.
- [32] M. R. Arefin, M. Tanaka, K. M. Kabir, et al., "Interplay between cost and effectiveness in influenza vaccination uptake: vaccination game approach," Proceedings of the Royal Society A, vol. 475, pp. 1–19, 2019.
- [33] K. M. Kabir and J. Tanimoto, "Dynamical behaviors for vaccination can suppress infectious disease – A game theoretical approach," Chaos, Solitons & Fractals, vol. 123, pp. 229–239, 2019.
- [34] M. S. Islam, J. I. Ira, K. M. Kabir, et al., "Effect of lockdown and isolation to suppress the COVID-19 in Bangladesh: An epidemic compartments model," Journal of Applied Mathematics and Computing, vol. 4, pp. 83–93, 2020.
- [35] T. C. Bauch, "Imitation dynamics predict vaccinating behavior," Proceedings of the Royal Society B-Biological Sciences, vol. 272, pp. 1669–1675, 2005.
- [36] \overline{X} . Y. Meng, S. J. Han, L. L. Wu, et al., "How quarantine and social-distancing policy can suppress the outbreak of novel coronavirus in developing or under poverty level countries: a mathematical and statistical analysis," Reliability Engineering & System Safety, vol. 219, 108256, 2022.
- [37] K. M. Kabir, K. Kuga, and J. Tanimoto, "Effect of information spreading to suppress the disease contagion on the epidemic vaccination game," Chaos, Solitons & Fractals, vol. 19, pp. 180-187, 2019.
- [38] Y. T. Wei, Y. S. Lin, and B. Wu, "Vaccination dilemma on an evolving social network," Journal of Theoretical Biology, vol. 483, pp. 1–32, 2019.
- [39] \overline{O} , O. Wang, C. P. Du, Y. N. Geng, et al., "Historical payoff can not overcome the vaccination dilemma on Barabási-Albert scalefree networks," Chaos, Solitons & Fractals, vol. 130, 109453, 2020.
- [40] J. W. Wang, J. L. He, F. Y. Yu, et al., "Realistic decision-making process with memory and adaptability in evolutionary vaccination game," Chaos, Solitons & Fractals, vol. 132, 109582, 2020.
- [41] M. Chinazzi, J. T. Davis, M. Ajelli, et al., "The effect of travel restrictions on the spread of the 2019 novel coronavirus (COVID-19) outbreak," Science, vol. 368, p. 395, 2020.
- [42] U. G. Moritz, C. H. Yang, G. Bernardo, et al., "The effect of human mobility and control measures on the COVID-19 epidemic in China," Science, vol. 368, pp. 493–497, 2020.
- [43] F. Fu, D. I. Rosenbloom, L. Wang, et al., "Imitation dynamics of vaccination behavior on social networks," Proceedings of the Royal Society B-Biological Sciences, vol. 278, pp. 42–49, 2011.
- [44] K. M. Kabir and J. Tanimoto, "Vaccination strategies in a twolayer SIR/V-UA epidemic model with costly information and buzz effect," Communications in Nonlinear Science and Numerical Simulation, vol. 76, pp. 92–108, 2019.
- [45] P. Dreessche and J. Watmough, "Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission," Mathematical Biosciences, vol. 180, pp. 29–48, 2002.

Copyright © 2024 by the authors. This is an open access article distributed under the Creative Commons Attribution License (CC BY-NC-ND 4.0), which permits use, distribution and reproduction in any medium, provided that the article is properly cited, the use is noncommercial and no modifications or adaptations are made.