Modelling Medical Resources for a Stochastic Epidemic Model With Vaccination Concerning High-Risk Infectious Diseases

Yuanmei Dong, Jin Yang*

First auther's e-mail: dym18855448705@163.com,*Corresponding auther's e-mail: seehom@126.com

Department of Mathematics, Chongqing Jiaotong University, Chongqing 400074, PR China

Abstract. In this paper, a stochastic epidemic model including the time since vaccination (vaccine-age) in the presence of high-risk infectious diseases is formulated and studied. The stationary distribution and ergodicity of the proposed model are investigated. The results suggest that with treatments triaged between high-risk and low-risk infectious diseases, reasonable allocations of medical resources can accelerate the extinction of diseases and, also, that large random fluctuations favor the extinction of the diseases.

Keywords: Stochastic epidemic model, Vaccination, Limited medical resources, Resource allocation

1 Introduction

In the early period after a new infectious disease has appeared, there are only a few infected people, so a hospital's resources are sufficient at this time. But rapid spread of the disease and fast increases in the number of cases leads to shortages of medical resources such as vaccines, medicines, and doctors in hospitals [1,2,3]. Meanwhile, healthcare workers also face pressure to triage patients with high-risk or low-risk infectious diseases, and these decisions played important roles in the prevention and control of infectious diseases. Therefore, limited medical resources and pressure regarding patient selection are two important factors in the spread of infectious diseases.

To study the mechanism of limited medical resources and patient selection pressure on disease control. Qin et al. proposed a non-smooth Filippov infectious disease model with threshold strategy to investigate how limited medical resources and patient selection pressure affect the outbreaks of high-risk R_0 and low-risk infectious diseases[4,5]. With limited medical resources, they found that choice pressure can help to prevent the spread of emerging infectious diseases, and it is also shown that lowering the threshold in a timely manner or increasing the maximum recovery rate was beneficial to the control of emerging infectious diseases.

The effect of vaccines can only be maintained for short periods for many infectious diseases, so that the impact of the time since vaccinations should be taken into consideration[6]. Let ς be the vaccine-age (i.e., the length of the period since the vaccination took place) and denote the number of the vaccinated population with vaccine-age ς at time t as $V(\varsigma, t)$, $\alpha(\varsigma)$ is the

PMBDA 2023, December 15-17, Nanjing, People's Republic of China

DOI 10.4108/eai.15-12-2023.2345394

rate at which the vaccine wanes at vaccine-age ς and $0 \leqslant \alpha(\varsigma) \leqslant 1$. Here, $\int_0^\infty \alpha(\varsigma)d\varsigma = \infty$, which guarantees that no one will be vaccinated when vaccine-age ς is close to infinity. In other words, when vaccine-age ς approaches infinity $\lim_{\varsigma \to \infty} V(\varsigma, t) = 0$, the number of vaccinated people tends to zero. For simplicity, every year the number of people with influenza is assumed to be a constant, i.e., $I_2 = k$, thus, the number of patients with high-risk disease is represented by $I(t)$. So model can be described as

$$\begin{cases} \dfrac{dS(t)}{dt} = \Lambda - (d+g)S(t) - \delta S(t)I(t) + \int_0^\infty \alpha(\varsigma)V(\varsigma,t)d\varsigma + m\mu I(t), \\ \dfrac{\partial V(\varsigma,t)}{\partial \varsigma} + \dfrac{\partial V(\varsigma,t)}{\partial t} = -(d+\alpha(\varsigma))V(\varsigma,t), \\ \dfrac{dI(t)}{dt} = \delta S(t)I(t) - (\mu + d)I(t) - \dfrac{c_1 I(t)}{1 + b_1 I(t) + rb_2 k}, \\ \dfrac{dR(t)}{dt} = (1-m)\mu I(t) + \dfrac{c_1 I(t)}{1 + b_1 I(t) + rb_2 k} - dR(t), \end{cases} \tag{1}$$

where the total population is divided into four classes: the susceptible populations $S(t)$, the vaccinated $V(t)$, the high-risk infectious diseases $I(t)$ and those removed $R(t)$. Λ, d, g, δ, μ, c_1, b_1, b_2 are respectively the population recruitment rate, the natural mortality rate, the ratio coefficient of vaccinated and susceptible patients, the effective contact rate of infected and susceptible patients, the recovery rate of infected patients, the maximum recovery rate of patients per unit time, the impact of medical resource limitation on the treatment of high-risk infectious diseases, and the impact of medical resource constraints on the treatment of low-risk infectious diseases. Assume that the infected people with temporary immunity become susceptible at rate m, while the infected people with permanent immunity become recovered at rate $1 - m$. For $r = 0$, indicates that high-risk infectious diseases are above the threshold, so medical workers should not only give priority to treat patients with high risk disease, but also allocate only limited medical resources to these patients. For $r \in (0, 1]$, both high-risk and low-risk infectious diseases are treated.

With the initial and boundary conditions as follows

$$S(0) = S_0, I(0) = I_0, R(0) = R_0, V(\varsigma, 0) = V_0(\varsigma), V(0, t) = gS(t), \tag{2}$$

where

$$S_0 + I_0 + R_0 + \int_0^\infty V_0(\varsigma)d\varsigma = N_0, \tag{3}$$

N_0 is a constant and denotes the total population size at the initial time. Integrating the second equation in model (1) along the characteristic line $t - \varsigma =$ constant, which implies that

$$V(\varsigma,t)=\begin{cases} gS(t-\varsigma)W_0(\varsigma), & t>\varsigma\geqslant 0,\\ V_0(\varsigma-t)\dfrac{W_0(\varsigma)}{W_0(\varsigma-t)}, & \varsigma\geqslant t>0, \end{cases} \tag{4}$$

where $W_0(\varsigma)=e^{-\int_0^{\varsigma}(d+\alpha(\tau))\mathrm{d}\tau}$.

For $t\geqslant 0$, so

$$\int_0^{\infty}\alpha(\varsigma)V(\varsigma,t)\mathrm{d}\varsigma=\int_0^{t}\alpha(\varsigma)gS(t-\varsigma)W_0(\varsigma)\mathrm{d}\varsigma+\int_t^{\infty}\alpha(\varsigma)V_0(\varsigma-t)\frac{W_0(\varsigma)}{W_0(\varsigma-t)}\mathrm{d}\varsigma\leqslant e^{-dt}\int_t^{\infty}V_0(\varsigma-t)\mathrm{d}\varsigma<e^{-dt}N_0, \tag{5}$$

where $\hat{\varsigma}=\varsigma-t$, because $e^{-dt}N_0\to 0$ as $t\to\infty$, then expression (2) becomes

$$\int_0^{\infty}\alpha(\varsigma)V(\varsigma,t)\mathrm{d}\varsigma=\int_0^{\infty}\alpha(\varsigma)gS(t-\varsigma)W_0(\varsigma)\mathrm{d}\varsigma. \tag{6}$$

Substituting equation (6) into the first equation of model (1)

$$\dot{S}(t)=\Lambda-(d+g)S(t)-\delta S(t)I(t)+\int_0^{\infty}W(\varsigma)S(t-\varsigma)\mathrm{d}\varsigma+m\mu I(t), \tag{7}$$

with

$$\Gamma(\varsigma)=g\alpha(\varsigma)W_0(\varsigma). \tag{8}$$

It is found that the recovered population $R(t)$ does not affect the dynamics of $S(t)$ and $I(t)$, for system (1) so we only need to consider the following equations,

$$\begin{cases} \dot{S}(t) &= \Lambda-(d+g)S(t)-\delta S(t)I(t)+\int_0^{\infty}W(\varsigma)S(t-\varsigma)\mathrm{d}\varsigma+m\mu I(t),\\ \dot{I}(t) &= \delta S(t)I(t)-(\mu+d)I(t)-\dfrac{c_1I(t)}{1+b_1I(t)+rb_2k}. \end{cases} \tag{9}$$

However, human behaviors are inevitably influenced by environmental factors. For example, humidity, wind direction and other factors can directly affect the contact rates between infected and susceptible populations [8]. It means that some parameters of the model (1) are no longer constants, but fluctuate within a certain range. Therefore, assume that parameters δ and d are disturbed by white noise, and the fluctuations are formulated by a stochastic process $\delta=\delta+\sigma_1\dot{B}_1(t)$, $-d=-d+\sigma_2\dot{B}_2(t)$, where σ_1 and σ_2 represent the intensity of the white noise. $B_1(t)$ and $B_2(t)$ are standard Brownian motions and defined on a complete probability space $\left(\Omega,\mathcal{Z},\{\mathcal{Z}_t\}_{t\geqslant 0},\mathbb{P}\right)$ with its filtration $\{\mathcal{Z}_t\}_{t\geqslant 0}$, where $\{\mathcal{Z}_t\}_{t\geqslant 0}$ is right continuous and $\mathcal{Z}_0$ contains all $\mathbb{P}$-null sets [7]. Then model (1) with white noise can be described by the following equations,

$$\begin{cases} \mathrm{d}S(t) = \left(\Lambda - (d+g)S(t) - \delta S(t)I(t) + \int_0^\infty W(\varsigma)S(t-\varsigma)d\varsigma + m\mu I(t)\right)\mathrm{d}t - \sigma_1 S(t)I(t)\mathrm{d}B_1(t), \\ \mathrm{d}I(t) = \left(\delta S(t)I(t) - (\mu+d)I(t) - \dfrac{c_1 I(t)}{1+h_1 I(t)+rh_2 k}\right)\mathrm{d}t + \sigma_1 S(t)I(t)\mathrm{d}B_1(t) + \sigma_2 I(t)\mathrm{d}B_2(t). \end{cases} \tag{10}$$

The paper is arranged as follows. It is easy to prove the existence and uniqueness of the global solution will first be investigated. I will not repeat myself in this article, the stationary distribution and ergodicity for the proposed system are investigated. Finally, numerical simulations are conducted to support the analytical results and biological implications are addressed.

2 Stationary distribution

To discuss the existence of model (10) traversing a stationary distribution, an useful lemma is introduced firstly. Let $Z(t)$ be a homogeneous Markov process in $\mathbb{R}^d$ ($\mathbb{R}^d$ is a d -dimensional Euclidean space), which can be described by the following Stochastic Differential Equation[9]

$$dZ(t) = b(Z)dt + \sum_{r=1}^{l} h_r(Z)dB_r(t),$$

with the diffusion matrix defined as follows

$$\tilde{A} = (a_{ij}(Z)), (a_{ij}(Z)) = \sum_{r=1}^{l} h_r^i(Z)h_r^j(Z).$$

lemma 2. Assume that there is a bounded domain $Q \subset \mathbb{R}^d$ with regular boundary, and Q satisfies [10]:

(1) there is with a positive constant F_0 such that

$$\sum_{i,j=1}^{d} a_{ij}\xi_i\xi_j \geq F_0 \mid \xi \mid^2, z \in Q, \xi \in \mathbb{R}^d;$$

(2) there is a nonnegative C^2 -function V such that $\mathcal{L}V$ is negative for any $\mathbb{R}^d / Q$. Then

$$\mathbb{P}_y\left\{\lim_{T\to\infty}\frac{1}{T}\int_0^T f(Z(t))dt = \int_{\mathbb{R}^d} f(z)\mu(dz)\right\} = 1,$$

for all $z \in \mathbb{R}^d$, where $f(.)$ is a function that is integrable with respect to the measure μ.

For convenience, let $\alpha \vee \beta$ be the maximum of α and β, and denote $h^u = \sup_{t\in[0,\infty)} h(t)$, $h^l = \inf_{t\in[0,\infty)} h(t)$. The following results can be obtained from Lemma 2.

Theorem 2: If there exists with a constant $K(K > \sigma_1\sigma_2)$ which leads λ to be positive, where

$$\lambda = \frac{\delta\Lambda}{d+g} - \frac{Kg}{d}N_0 - (d+g+1)\Lambda K - (\mu + d + \frac{\sigma_2^2}{2}) - \frac{c_1}{1+b_1N_0+rb_2k} - \frac{K\Lambda\sigma_1^2N_0^2}{2}, \quad \text{so}$$

for given initial value $(S(0), I(0)) \in \mathbb{R}_+^2$, system (10) allows for a stationary $\mu(.)$ which is ergodic.

Proof: The diffusion matrix of stochastic the system (10) is

$$\tilde{A}(S, I) = \begin{pmatrix} \sigma_1^2S^2I^2 & 0 \\ 0 & \sigma_2^2I^2 \end{pmatrix}.$$

Let $\bar{Q}$ be any bounded domain in $\mathbb{R}_+^2$, then there is a positive constant $F_0 = \min\{\sigma_1^2S^2I^2, \sigma_2^2I^2\}$ such that $\sum_{i,j=1}^{2} a_{ij}(S, I)\xi_i\xi_j = \sigma_1^2S^2I^2\xi_1^2 + \sigma_2^2I^2\xi_2^2 \geq F_0 \mid \xi \mid^2$, for all $(S, I) \in \bar{Q}$ and $\xi \in \mathbb{R}^2$.

Now, define a non-negative C^2-function V and find a closed set $Q \in \mathbb{R}_+^2$ such that $\sup_{(S,I)\in\mathbb{R}_+^2\setminus Q} \mathcal{L}V(S, I) < -R < 0$, with R being a positive constant. Choosing a constant $\theta \in (0, 1)$ small enough which satisfies (i): $\omega - \frac{\theta}{2}(\sigma_1^2 \vee \sigma^2) > 0$.

Then select a large enough positive constant F such that (ii): $\Phi^u - F\lambda \leq -2$, where the explicit expression of $\Phi(S, I)$ will be derived in the following. Thus, define a C^2-function $H = FV_1 + V_2 + V_3$, where

$$V_1 = K\left(S - \Lambda - \Lambda\ln\frac{S}{\Lambda}\right) - \ln I - \frac{\delta}{d+g}(S+I), V_2 = \frac{1}{\theta+1}(S+I)^{\theta+1}, \quad V_3 = -\ln S.$$

It is shown that $H(S, I)$ gets the minimum value at the unique point (S_*, I_*). Therefore, a nonnegative C^2-Lyapunov function V can be defined as follows,

$$V = FV_1 + V_2 + V_3 - H(S_*, I_*). \tag{11}$$

Calculating $\mathcal{L}V_1$ and it is found that $\mathcal{L}V_1$ consists of the following three parts,

$$\mathcal{L}(-\ln I) = (\mu + d) + \frac{c_1}{1+b_1I(t)+rb_2k} - \delta S(t) + \frac{\sigma_1^2S^2(t)}{2} + \frac{\sigma_2^2}{2} + \sigma_1\sigma_2S(t), \tag{12}$$

$$\mathcal{L}(S+I) = \Lambda - (d+g)S(t) + \int_0^\infty W(\varsigma)S(t-\varsigma)d\varsigma + m\mu I(t) - \frac{c_1I(t)}{1+b_1I(t)+rb_2k} - (\mu+d)I(t), \tag{13}$$

$$\mathcal{L}\left(S-\Lambda-\Lambda\ln\frac{S}{\Lambda}\right)=\left(1-\frac{\Lambda}{S}\right)\left(\Lambda-(d+g)S(t)-\delta S(t)I(t)+\int W(\varsigma)S(t-\varsigma)d\varsigma+m\mu I(t)\right)+\frac{\Lambda\sigma_1^2I^2(t)}{2}. \tag{14}$$

Thus,

$$\begin{aligned}\mathcal{L}V_1 &= K(1-\frac{\Lambda}{S})\left(\Lambda-(d+g)S(t)-\delta S(t)I(t)+\int_0^\infty W(\varsigma)S(t-\varsigma)d\varsigma+m\mu I(t)\right)\\ &+\frac{K\Lambda\sigma_1^2I(t)^2}{2}+(\mu+d+\frac{\sigma_2^2}{2})+\frac{c_1}{1+b_1I(t)+rb_2k}-\delta S(t)+\frac{\sigma_1^2S^2(t)}{2}+\sigma_1\sigma_2S(t)\\ &-\frac{\delta}{g+g}\left(\Lambda-(d+g)S(t)+\int_0^\infty W(\varsigma)S(t-\varsigma)d\varsigma+m\mu I(t)-(\mu+d)I(t)-\frac{c_1I(t)}{1+b_1I(t)+rb_2k}\right)\\ &\leqslant-\lambda+f(S)+\left(m\mu K+\delta K\Lambda+\frac{\delta(\mu+d)}{d+g}+\frac{\delta}{d+g}\cdot\frac{c_1}{1+b_1(N_0-\varepsilon)+rb_2k}\right)I(t),\end{aligned} \tag{15}$$

where

$$f(S)=-\frac{(K-\sigma_1\sigma_2)(S-\Lambda)^2}{S}. \tag{16}$$

Furthermore,

$$f'(S)=-\frac{(K-\sigma_1\sigma_2)(S^2-\Lambda^2)}{S^2}\text{ and }f''(S)=-\frac{2(K-\sigma_1\sigma_2)\Lambda^2}{S^3}<0.$$

It is clear that $f'(S)_{S=\Lambda}=0.$

So

$$f(S)\leqslant f(\Lambda)=0. \tag{17}$$

Therefore,

$$\mathcal{L}V_1\leqslant-\lambda+\left(m\mu K+\delta K\Lambda+\frac{\delta(\mu+d)}{d+g}+\frac{\delta}{d+g}\cdot\frac{c_1}{1+b_1(N_0-\varepsilon)+rb_2k}\right)I(t). \tag{18}$$

Similarly,

$$\begin{aligned}\mathcal{L}V_2 &= (S+I)^\theta(\Lambda-(d+g)S+\int_0^\infty W(\varsigma)S(t-\varsigma)d\varsigma+m\mu I-(\mu+d)I-\frac{c_1I}{1+b_1I+rb_2k})\\ &+\frac{\theta}{2}(S+I)^{\theta-1}\left(\sigma_1^2S^2I^2+\sigma_1^2S^2I^2+2\sigma_1\sigma_2SI^2+\sigma_2I^2\right)\\ &\leq\left(\Lambda+\frac{g}{d}N_0+m\mu N_0\right)(S+I)^\theta-w(S+I)^{\theta+1}+\frac{\theta}{2}(S+I)^{\theta-1}\left(\sigma_1^2\vee\sigma^2\right)(S+I)^2\\ &=\left(\Lambda+\frac{g}{d}N_0+m\mu N_0\right)(S+I)^\theta-\left[w-\frac{\theta}{2}\left(\sigma_1^2\vee\sigma^2\right)\right](S+I)^{\theta+1},\end{aligned} \tag{19}$$

where

$$\sigma=\sigma_1+\sigma_2\text{ and }\omega=\min\{(d+g),(u+d)\}.$$

Moreover,

$$\begin{aligned}\mathcal{L}\mathcal{V}_3 &= -\frac{\Lambda}{S} + (d+g) + \delta I(t) - \frac{1}{S}\int_0^\infty W(\varsigma)S(t-\varsigma)d\varsigma - \frac{m\mu I}{S} + \frac{\sigma_1^2 I^2(t)}{2} \\ &\leqslant -\frac{\Lambda}{S} + (d+g) + \frac{\sigma_1^2 N_0^2}{2} + \delta I(t).\end{aligned} \tag{20}$$

Substituting (18), (19) and (20) into (11), then $\mathcal{L}\mathcal{V} \leq \Phi(S, I) + \Psi(I)$, where

$$\begin{cases}\Phi(S, I) = \left(\Lambda + \dfrac{g}{d}N_0 + m\mu N_0\right)(S+I)^\theta - \left[\omega - \dfrac{\theta}{2}\left(\sigma_1^2 \vee \sigma^2\right)\right](S+I)^{\theta+1} - \dfrac{\Lambda}{S} + (d+g) + \dfrac{\sigma_1^2 N_0^2}{2}, \\ \Psi(I) = M\left[-\lambda + \left(m\mu k + \delta\Lambda k + \dfrac{\delta(u+d)}{d+g} + \dfrac{\delta}{d+g}\dfrac{c_1}{1+b_1\left(N_0-\varepsilon\right)+rb_2 k}\right)I(t)\right] + \delta I(t).\end{cases} \tag{21}$$

In view of (i), we observe that

(1) $\Phi(+\infty, I) + \Psi^u \to -\infty, \quad a.s. \quad S \to +\infty,$

(2) $\Phi(S, +\infty) + \Psi(+\infty) \to -\infty, \quad a.s. \quad I \to +\infty,$

(3) $\Phi\left(0^+, I\right) + \Psi^u \to -\infty, \quad a.s. \quad S \to 0^+.$

The above three cases are caused $\mathcal{L}V < -1$, respectively. According to condition the (ii), we have

(4) $\Phi\left(S, 0^+\right) + \Psi\left(0^+\right) \to \Phi^u - M\lambda \leq -2, \quad a.s. \quad I \to 0^+.$

Thus, take ζ small enough and let $Q = \left[\eta, \dfrac{1}{\eta}\right] \times \left[\eta, \dfrac{1}{\eta}\right]$, then it follows from case 4 that we have

$$\mathcal{L}\mathcal{V} < -1, \quad (S, I) \in \mathbb{R}_+^2 \setminus Q.$$

The proof is completed.

3 Numerical investigations

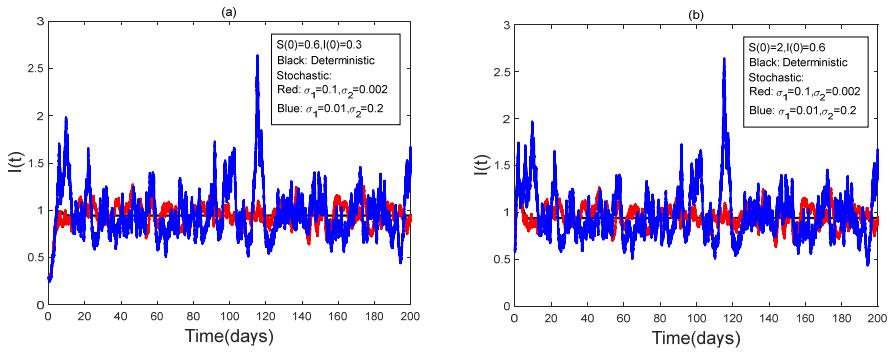

Fig 1. Stationary distribution of deterministic model and stochastic model. (a) We set initial values as $(S(0), I(0)) = (0.6, 0.3)$. (b) We set initial values as $(S(0), I(0)) = (2, 0.6)$. All other parameters were fixed as: $\Lambda = 0.98, \delta = 0.995, m = 0.3, \mu = 0.06,$ $g = 0.8, d = 0.27, c_1 = 0.7, b_1 = 0.3, b_2 = 0.9, r = 1, k = 1.$

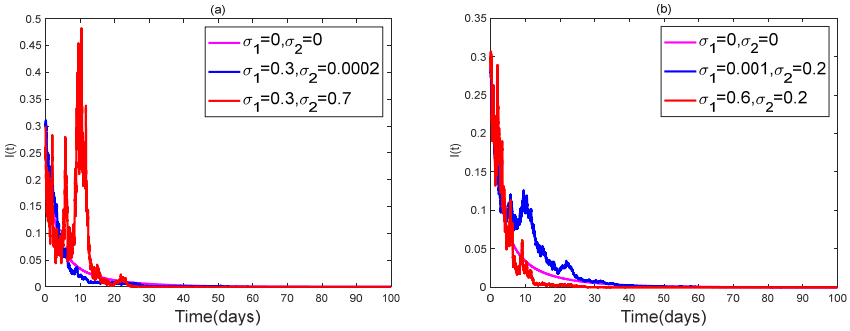

Fig 2. Effects of white noise on the extinction of infectious diseases. All baseline parameter values were fixed as : $\Lambda = 0.5, \delta = 0.8, m = 0.3, \mu = 0.2, g = 0.8, d = 0.27, c_1 = 0.7,$ $b_1 = 0.1, b_2 = 1.2, r = 1, k = 1.$

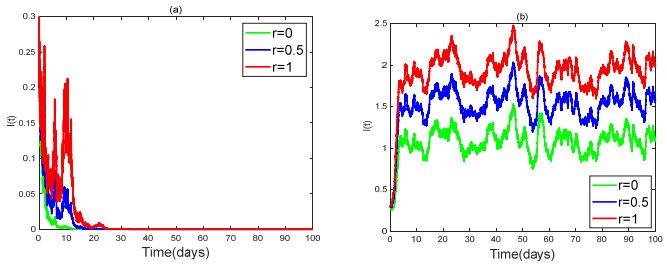

Fig 3. These plots show the sensitivity of r to disease extinction and disease persistence. (a) $\Lambda = 0.5, \delta = 0.8, \mu = 0.2, \sigma_1 = 0.3, \sigma_2 = 0.2, d = 0.3, b_1 = 0.1, b_2 = 1.2$; (b) $\Lambda = 0.98, \delta = 0.995, \mu = 0.06, \sigma_1 = 0.1, \sigma_2 = 0.02, d = 0.05, b_1 = 0.3, b_2 = 0.9$. All baseline parameter values were fixed as: $m = 0.3, g = 0.8, c_1 = 0.7, r = 1, k = 1$.

All baseline parameter values can be found in references[4,5]. From Fig 1, the initial values are fixed as $(S(0), I(0)) = (0.6, 0.3)$ and $(S(0), I(0)) = (2, 0.6)$ by simple calculation, we have $K = 0.01 > \sigma_1\sigma_2, \lambda = 0.193 > 0,$

$$(\mathrm{i}): \omega - \frac{\theta}{2}(\sigma_1^2 \vee \sigma^2) \approx 0.330 > 0; (\mathrm{ii}): \Phi^u - M\lambda \approx -4.115 \leq -2.$$

Then all conditions of Theorem 2 are satisfied, which implies that system (2) allows for a stationary $\mu(.)$ which is ergodic.

It is noted that infectious diseases are influenced by many factors, such as environmental noise. From Fig 2(a), keep σ_1 and increasing σ_2, then the speed of disease elimination will be accelerated. Fix σ_2 and increasing σ_1 will also accelerate the extinction of the diseases Fig 2(b). It can be found that the greater the noise intensity, the more conducive it is to the extinction of infectious diseases. According to Fig 3(a), with the smaller of the resource allocations coefficient, resources will be prioritized to patients with high-risk infectious diseases, and then the disease will become extinct faster. When the disease is persistent, the scale of the outbreak will become smaller as decreases Fig 3(b).

4 Conclusion

In this paper, the existence and uniqueness of the ergodic stationary distribution are discussed by employing a novel combination of Lyapunov functions. Numerical studies are also performed to support our results. Compared to the previous results [4,5], the highlights are listed as follows: (1) the proposed model not only considers the effects of white noise, but also the vaccine-age are taken into account; (2) the ergodic stationary distribution of the model is also discussed; (3) random perturbations and medical resources are very critical to control the outbreaks of the diseases.

There are many interesting questions deserving future investigation. In this paper, for simplicity, the average year of infection with low-risk diseases is assumed to be a constant. But if we consider both high-risk and low-risk infectious diseases, then how to propose the stochastic model with high-risk and low-risk infectious diseases? How do limited medical resources, patient selection pressure and white noise affect the global dynamics of the proposed system? What is the feasible treatment for the prevention and control of infectious diseases? We leave these questions for future work.

Acknowledgements: This work was supported by the National Natural Science Foundation of China under Grants 11961024 (Y. Tan), 11801047 (J. Yang), and by Team Building Project for Graduate Tutors in Chongqing JDDSTD201802 (J. Yang), and by Joint Training Base Construction Project for Graduate Students in Chongqing JDLHPYJD2021016, and by the Program of Chongqing Municipal Education Commission KJQN201900707 (Z. Liu), and by the Natural Science Foundation of Chongqing under Grant cstc2019jcyj-msxmX0755 (Z. Liu), and by Group Building Scientific Innovation Project for universities in Chongqing CXQT21021.

Reference

[1] P. Nansheng, M. Qichen, Resource allocation in robust scheduling, J. Oper. Res. Soc. 74 (2023) 125-142.

[2] H. Zhao, L. Wang, S.M. Oliva, et al., Modelling and dynamics analysis of Zika transmission with limited medical resources, Bull. Math. Biol. 82 (2020) 1-50.

[3] D. He, X. Wang, D. Gao, et al., Modelling the 2016–2017 Yemen cholera outbreak with the impact of limited medical resources, J. Theor. Biol. 451 (2018) 80-85.

[4] W. Qin, S. Tang, The selection pressures induced non-smooth infectious disease model and bifurcation analysis, Chaos Soliton Fract. 69 (2014) 160-171.

[5] W. Qin, S. Tang, C. Xiang, et al., Effects of limited medical resource on a Filippov infectious disease model induced by selection pressure, Appl. Math. Comput. 283 (2016) 339-354.

[6] C. Cohet, R. van der Most, V. Bauchau, et al., Safety of AS03-adjuvanted influenza vaccines: a review of the evidence, Vaccine, 37 (2019) 3006-3021.

[7] X. Ran, L. Hu, L.F. Nie, et al., Effects of stochastic perturbation and vaccinated age on a vector-borne epidemic model with saturation incidence rate, Appl. Math. Comput. 394 (2021) 125798.

[8] H. Mori, Transport, collective motion, and Brownian motion, Prog. Theor. Phys. 33 (1965) 423-455.

[9] R. Qesmi, A. Hammoumi, A stochastic delay model of HIV pathogenesis with reactivation of latent reservoirs, Chaos Soliton Fract. 132 (2020) 109594.

[10] B. Tang, X. Wang, Q. Li, et al., Estimation of the transmission risk of the 2019-nCoV and its implication for public health interventions, J. Clin. Med. 9 (2020) 462.