DYNAMICS OF A STOCHASTIC SIR MODEL WITH BOTH HORIZONTAL AND VERTICAL TRANSMISSION*

Anqi Miao¹, Tongqian Zhang^{1,2,†}, Jian Zhang¹ and Chaoyang Wang¹

Abstract A stochastic mathematical model with both horizontal and vertical transmission is proposed to investigate the dynamical behavior of SIR disease. By employing theories of stochastic differential equation and inequality techniques, the threshold associating on extinction and persistence of infectious diseases is deduced for the case of the small noise. Our results show that the threshold completely depends on the stochastic perturbation and the basic reproductive number of the corresponding deterministic model. Moreover, we find that large noise is conducive to control the spread of diseases and the persistent disease in deterministic model may eliminate ultimately due to the effect of large noise. Finally, numerical simulations are performed to illustrate the theoretical results.

Keywords Stochastic SIR epidemic model, vertical transmission, extinction, persistence, threshold.

MSC(2010) 60H10, 92F99.

1. Introduction

Infectious diseases threaten human health and bring huge disaster to human beings. People have realized the importance of quantitative studies on the spread of infectious diseases to predict and control them. Mathematical models have been confirmed to be an effective and valuable approach to understand the dynamical behavior of infectious disease, then a large number of mathematical models have been constructed to investigate the dynamical behavior of propagation and evolution rule of infectious diseases [2]. A classical epidemic model known as SIR model was proposed and investigated firstly by Kermack and Mckendric in 1927 [12]. In

[†]the corresponding author. Email address:zhangtongqian@sdust.edu.cn (T. Zhang)

¹College of Mathematics and Systems Science, Shandong University of Science and Technology, Qingdao 266590, China

²State Key Laboratory of Mining Disaster Prevention and Control Co-founded by Shandong Province and the Ministry of Science and Technology, Shandong University of Science and Technology, Qingdao 266590, China

^{*}The authors were supported by Shandong Provincial Natural Science Foundation of China (No.ZR2015AQ001), the National Natural Science Foundation of China (No. 11371230), SDUST Research Fund (2014TDJH102), and Joint Innovative Center for Safe and Effective Mining Technology and Equipment of Coal Resources, Shandong Province of China, SDUST Innovation Fund for Graduate Students (SDKDYC180348).

SIR model, population is divided into three classes, the susceptible, the infectious and the recovered or removed. SIR models play a crucial role in studying the evolution of infectious diseases and have received widely attentions (see, for examples, [26,34,36,39-41,44]). SIR models mainly described the horizontal propagation and the evolution of diseases between members of the same population. However, researchers have shown that some diseases such as AIDS [28,31,35] and Hepatitis B or Hepatitis C [29,30], are transmitted horizontally, i.e., these kinds of diseases are directly transmitting from the mother to an embryo, fetus, or baby during pregnancy or childbirth. As it is important to study the dynamical properties emerge by the horizontal transmission, Lu et al. [22] proposed an SIR epidemic model incorporating both horizontal and vertical transmission as below:

$$\begin{cases} \frac{\mathrm{d}S(t)}{\mathrm{d}t} = -bS(t) - \beta S(t)I(t) + pdI(t) + b(S(t) + R(t)),\\ \frac{\mathrm{d}I(t)}{\mathrm{d}t} = \beta S(t)I(t) - dI(t) - \gamma I(t) + qdI(t),\\ \frac{\mathrm{d}R(t)}{\mathrm{d}t} = \gamma I(t) - bR(t), \end{cases}$$
(1.1)

where S(t), I(t) and R(t) represent the members of the susceptible, the infectious and the removed members from infection, respectively. b is the birth and death rate. β is the contact rate. The constant p(0 is the proportion of the offspring $of infective parents who are susceptible for the disease. <math>\gamma$ is the recovery rate of the infective individuals and p+q=1. Note that the total population size is normalized to one and the basic reproductive number of model (1.1) is defined as $R_0 = \beta/(pd+\gamma)$. The authors pointed out that if $R_0 < 1$, the infection-free equilibrium $P_0(1,0,0)$ is globally asymptotically stable while if $R_0 > 1$, the infection-free equilibrium P_0 is unstable, and the endemic equilibrium $P^*(S^*, I^*, R^*)$ is globally asymptotically stable.

It is now well known that stochastic noise is widely present in biological systems [5, 17, 18, 23, 33, 38, 43] and stochastic noise factors play an important role in transmission of infectious diseases, because it can provide an additional degree of realism in comparison to their deterministic counterparts. Therefore, many scholars have studied the effect of stochasticity on epidemic models [1,3,25,27], different stochastic perturbation approaches have been introduced into epidemic models and excellent results have been obtained. The authors of the articles [6, 10, 15] have considered epidemic models including the environment noise and have analyzed the dynamical behavior by using method of time Markov chain. The transmission coefficient perturbation in epidemic models induced by environment white noise has been investigated [9, 16, 21, 32, 42], in which the stochastic perturbation of system can be offset by summation. In the research articles [7, 14], the authors have extensively studied the environmental noise which was proportional to the variables. Stochastic epidemic models with a complex type of noises described by the combination of parameter perturbation and proportion of the variables were investigated in [8, 20]. In the research articles [37, 45], the authors have paid their attention on the epidemic models with Lévy jump noise, and in the articles [4, 19], the authors focused on stochastic perturbation around the positive equilibria of deterministic models.

In this paper, motivated by the above works, we introduce transmission coefficient perturbation with white noises into model (1.1) i.e., we replace β by $\beta + \sigma dB(t)$,

where B(t) is a standard Brownian motion with intensity $\sigma^2 > 0$. Then the resultant model turns into the following form:

$$\begin{cases} dS(t) = (-bS(t) - \beta S(t)I(t) + pdI(t) + b(S(t) + R(t)))dt - \sigma S(t)I(t)dB(t), \\ dI(t) = (\beta S(t)I(t) - dI(t) - \gamma I(t) + qdI(t))dt + \sigma S(t)I(t)dB(t), \\ dR(t) = (\gamma I(t) - bR(t))dt. \end{cases}$$
(1.2)

This paper is organized as follows. In Section 2, we will give some notations, definitions and lemmas which will be used to obtain our main results. In addition, the conditions leading to the extinction of the infectious disease will be given in Section 3. In Section 4, we will deduce the condition for the disease being persistent. In Section 5, we will give a brief conclusion to summary of the paper and also give some numerical simulations to illustrate the theoretical results.

2. Preliminaries

Throughout this paper, for convenience let $(\Omega, \mathcal{F}, \{\mathcal{F}\}_{t\geq 0}, \mathcal{P})$ be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ satisfying the usual conditions such as increasing and right continuous and \mathcal{F}_0 contains all \mathcal{P} -null sets. Further, B(t) represents a scalar Brownian motion defined on the complete probability space Ω and $R^3_+ = \{x_i > 0, i = 1, 2, 3\}$. For a continuous function f, we define $\langle f(t) \rangle = \frac{1}{t} \int_0^t f(\tau) d\tau$, then we have

Definition 2.1. For model (1.2),

- (i) the diseases I(t) is said to be extinctive if $\lim_{t \to +\infty} I(t) = 0$;
- (ii) the diseases I(t) is said to be permanent in mean if there exist a positive constant λ such that $\liminf_{t \to +\infty} \langle I(t) \rangle \ge \lambda$.

The following lemmas indicate the global existence, non-negativity and invariance of unique solution of model (1.2).

Lemma 2.1. For any initial value $(S_0, I_0, R_0) \in R^3_+$, there exists a unique solution (S(t), I(t), R(t)) to model (1.2) on $t \ge 0$, and the solution remains in R^3_+ with probability one, namely, $(S(t), I(t), R(t)) \in R^3_+$ for all $t \ge 0$ a.s.

Proof. Firstly, we know that, for any initial values $(S_0, I_0, R_0) \in \mathbb{R}^3_+$, because the coefficients of model (1.2) are locally Lipschitz continuous, then there exists a unique local solution on $[0, \tau_{\epsilon})$ where τ_{ϵ} is the explosion time. To prove that this solution is global, we need to show $\tau_{\epsilon} = \infty$ a.s. To do it, let $\epsilon_0 > 0$ such that $S_0 > \epsilon_0, I_0 > \epsilon_0, R_0 > \epsilon_0$. For any positive ϵ satisfying $\epsilon \leq \epsilon_0$, define the stopping time τ_{ϵ} by

$$\tau_{\epsilon} = \inf\{t \in [0, \tau_{\epsilon}) : S(t) \le \epsilon \text{ or } I(t) \le \epsilon\},\$$

with the traditional setting $\inf \emptyset = \infty$, where \emptyset denotes the empty set. Clearly, τ_{ϵ} is increasing as $\epsilon \to 0$. Set $\tau_0 = \lim_{\epsilon \to 0}$, then $\tau_0 \leq \tau_{\epsilon}$ a.s, hence we need only to prove $\tau_0 = \infty$ a.s. Otherwise, then there exists a pair of constants T > 0 and $\delta \in (0,1)$ such that $P\{\tau_0 \leq T\} > \delta$. Hence, there exists a positive constant $\epsilon_1 \leq \epsilon_0$ such that $P\{\tau_0 \leq T\} > \delta$ for any positive $\epsilon \leq \epsilon_1$.

,

Define a C^2 function $V: \mathbb{R}^3_+ \to \overline{\mathbb{R}}_+$ by

 $V(S(t), I(t), R(t)) = -\ln S - \ln I - \ln R.$

Obviously, V is positive definite. Using Itô's formula, we obtain

$$\mathrm{d}V = LV\mathrm{d}t + \sigma(I - S)\mathrm{d}B,$$

where

$$LV = \beta I - pd\frac{I}{S} - b\frac{R}{S} - \beta S + d + \gamma - qd - \gamma \frac{I}{R} + b + \frac{1}{2}\sigma^{2}(S^{2} + I^{2}).$$

Then, we have

Ì

$$LV \leq \beta + b + pd + \gamma + \frac{1}{2}\sigma^2(S^2 + I^2) \leq \beta + b + pd + \gamma + \sigma^2 := C.$$

Thus,

$$\mathrm{d}V \le C\mathrm{d}t + \sigma(I - S)\mathrm{d}B.$$

Integrating both sides from 0 to $\tau_e \wedge T$, and then taking expectations, yields

$$EV(S(\tau_{\epsilon} \wedge T), I(\tau_{\epsilon} \wedge T)) \leq V(S_0, I_0, R_0) + CT.$$

Set $\Omega_{\epsilon} = \{\tau_e \leq T\}$ for any positive $\epsilon \leq \epsilon_1$ and then $P(\Omega_{\epsilon}) > \delta$. Note that for every $\omega \in \Omega_{\epsilon}$, there is at least one of $S(\tau_{\epsilon}, \omega), I(\tau_{\epsilon}, \omega), R(\tau_{\epsilon}, \omega)$ equals ϵ , then

$$V(S(\tau_{\epsilon}), I(\tau_{\epsilon}), R(\tau_{\epsilon}, \omega)) \ge -\ln \epsilon.$$

Consequently,

$$V(S_0, I_0, R_0) + CT \ge E[I_{\Omega_{\epsilon}}V(S(\tau_{\epsilon} \wedge T), I(\tau_{\epsilon} \wedge T)R(\tau_{\epsilon} \wedge T))]$$

= $P(\Omega_{\epsilon})V(S(\tau_{\epsilon}), I(\tau_{\epsilon}), R(\tau_{\epsilon}))$
> $-\delta \ln \epsilon,$

where $I_{\Omega_{\epsilon}}$ is the indicator function of Ω_{ϵ} . Letting $\epsilon \to 0$ leads to the contradiction $\infty > V(S_0, I_0, R_0) + CT = \infty$. Therefore, we must have $\tau_0 = \infty$ a.s.

The proof of Lemma 2.1 is completed.

Remark 2.1. By using the methods from Ji et al. [11], we can also prove, for any initial value $(S_0, I_0, R_0) \in \overline{R}^3_+$, there exists a unique solution (S(t), I(t), R(t)) to model (1.2) on $t \ge 0$, and the solution remains in \overline{R}^3_+ with probability one, namely, $(S(t), I(t), R(t)) \in \overline{R}^3_+$ for all $t \ge 0$ a.s.

Remark 2.2. By Lemma 2.1 and Remark 2.1, we can obtain the region

$$\Gamma = \{ (S(t), I(t), R(t)) \in \mathbb{R}^3_+ : S(t) + I(t) + R(t) \le 1 \}$$

which is a positively invariant set, then through the paper, we always let the initial value $(S_0, I_0, R_0) \in \Gamma$.

Lemma 2.2. Let (S(t), I(t), R(t)) be a solution of model (1.2) with initial value $(S_0, I_0, R_0) \in \Gamma$. Then

$$\lim_{t \to +\infty} \frac{1}{t} \int_0^t \sigma S(\tau) dB(\tau) = 0.$$

Proof. Let $Z(t) = \int_0^t \sigma S(\tau) dB(\tau)$ and $\theta > 2$. By the Burkholder-Davis-Gundy inequality in [24] and Lemma 2.1, we have

$$E\left[\sup_{0\leq\tau\leq t}|Z(\tau)|^{\theta}\right]\leq C_{\theta}E\left[\int_{0}^{t}\sigma^{2}S^{2}(\tau)\mathrm{d}\tau\right]^{\frac{\theta}{2}}\leq C_{\theta}t^{\frac{\theta}{2}}E\left[\sup_{0\leq\tau\leq t}\sigma^{\theta}S^{\theta}(\tau)\right]\leq C_{\theta}\sigma^{\theta}t^{\frac{\theta}{2}},$$

where C_{θ} . Then, for any $0 < \varepsilon < \frac{\theta}{2} - 1$, by Doob's martingale inequality [24],

$$\mathbb{P}\left\{\omega: \sup_{k\delta \le t \le (k+1)\delta} |Z(t)|^{\theta} > (k\delta)^{1+\varepsilon+\frac{\theta}{2}}\right\} \le \frac{E\left(|Z((k+1)\delta)|^{\theta}\right)}{(k\delta)^{1+\varepsilon+\frac{\theta}{2}}} \le \frac{C_{\theta}\sigma^{\theta}[(k+1)\delta]^{\frac{\theta}{2}}}{(k\delta)^{1+\varepsilon+\frac{\theta}{2}}} \le \frac{2^{\frac{\theta}{2}}C_{\theta}\sigma^{\theta}}{(k\delta)^{1+\varepsilon}}$$

holds. Thus by Borel-Cantelli lemma in [24], for almost all $\omega \in \Omega$, we get that

$$\sup_{k\delta \le t \le (k+1)\delta} |Z(t)|^{\theta} \le (k\delta)^{1+\varepsilon+\frac{\theta}{2}}$$

holds for all but finitely many k. Thus, there exists a positive $k_0(\omega)$, for almost all $\omega \in \Omega$ and $k \geq k_0(\omega)$, such that $\sup_{k\delta \leq t \leq (k+1)\delta} |Z(t)|^{\theta} \leq (k\delta)^{1+\varepsilon+\frac{\theta}{2}}$. Hence, if $k \geq k_0(\omega)$ and $k\delta \leq t \leq (k+1)\delta$, then for almost all $\omega \in \Omega$,

$$\frac{\ln |Z(t)|^{\theta}}{\ln t} \le \frac{\left(1 + \varepsilon + \frac{\theta}{2}\right)\ln(k\delta)}{\ln(k\delta)} = 1 + \varepsilon + \frac{\theta}{2}.$$

Hence, we have

$$|Z(t)| \le t^{\frac{1}{2} + \frac{1+\varepsilon}{\theta}}$$

Then, for the above ε , there exists a constant $T(\omega)$ and a set Ω_{ϵ} , such that $\mathbb{P}(\Omega_{\epsilon}) \geq 1 - \epsilon$ and for $t \geq T(\omega), \omega \in \Omega_{\epsilon}$,

$$0 \leq \liminf_{t \to +\infty} \frac{|Z(t)|}{t} \leq \limsup_{t \to +\infty} \frac{|Z(t)|}{t} \leq \limsup_{t \to +\infty} t^{\frac{1+\varepsilon}{\theta} - \frac{1}{2}} = 0.$$

Then we have

$$\lim_{t \to +\infty} \frac{|Z(t)|}{t} = 0,$$

i.e.

$$\lim_{t \to +\infty} \frac{Z(t)}{t} = \lim_{t \to +\infty} \frac{1}{t} \int_0^t \sigma S(\tau) \mathrm{d}B(\tau) = 0.$$

This completes the proof of Lemma 2.2.

3. Extinction

In this section, we deduce the condition which is crucially important to the disease to be died out. Let us consider

$$R_0^* = rac{eta}{pd+\gamma} - rac{\sigma^2}{2(pd+\gamma)} = R_0 - rac{\sigma^2}{2(pd+\gamma)},$$

and the we have the results mentioned in the following theorem

Theorem 3.1. If $\sigma^2 > \max\{\beta, \frac{\beta^2}{2(pd+\gamma)}\}$ or $\sigma^2 \leq \beta$ and $R_0^* < 1$, then the infectious disease of model (1.2) goes to extinction almost surely. Moreover,

$$\lim_{t \to +\infty} R(t) = 0, \lim_{t \to +\infty} S(t) = 1,$$

a.s.

Proof. Let (S(t), I(t), R(t)) be a solution of system (1.2) with initial value $(S_0, I_0, R_0) \in \Gamma$. Applying Itô's formula to the second equation of model (1.2) leads to

$$d\ln I(t) = \left(\beta S(t) - (pd + \gamma) - \frac{\sigma^2}{2}S^2(t)\right)dt + \sigma S(t)dB(t).$$
(3.1)

Integrating both sides of (3.1) from 0 to t gives

$$\ln I(t) = \int_0^t \left(\beta S(\tau) - \frac{\sigma^2}{2} S^2(\tau)\right) d\tau - (pd + \gamma)t + M(t) + \ln I(0), \quad (3.2)$$

where $M(t) = \int_0^t \sigma S(\tau) dB(\tau)$ and M(t) is the local continuous martingale with M(0) = 0. Next we have two cases to be discussed, depending on $\sigma^2 > \beta$.

If $\sigma^2 > \beta$, one can easily see from (3.2) that

$$\ln I(t) \le \left(\frac{\beta^2}{2\sigma^2} - (pd + \gamma)\right)t + M(t) + \ln I(0). \tag{3.3}$$

Dividing both sides of (3.3) by t(>0), we have

$$\frac{\ln I(t)}{t} \le -\left(pd + \gamma - \frac{\beta^2}{2\sigma^2}\right) + \frac{M(t)}{t} + \frac{\ln I(0)}{t}.$$
(3.4)

By Lemma 2.2, we obtain $\lim_{t\to+\infty} \frac{M(t)}{t} = 0$, then taking the limit superior on both sides of (3.4) it leads to

$$\limsup_{t \to +\infty} \frac{\ln I(t)}{t} \le -\left(pd + \gamma - \frac{\beta^2}{2\sigma^2}\right) < 0,$$

when $\sigma^2 > \frac{\beta^2}{2(pd+\gamma)}$, which implies that $\lim_{t\to+\infty} I(t) = 0$. If $\sigma^2 \leq \beta$, we can similarly have

$$\ln I(t) \le \left(\beta - (pd + \gamma) - \frac{\sigma^2}{2}\right)t + M(t) + \ln I(0).$$
(3.5)

Dividing both sides of (3.5) by t(>0), we have

$$\frac{\ln I(t)}{t} \le (pd+\gamma) \left[\frac{\beta}{pd+\gamma} - \frac{\sigma^2}{2(pd+\gamma)} - 1\right] + \frac{M(t)}{t} + \frac{\ln I(0)}{t}.$$
 (3.6)

We take the superior limit on both sides of (3.6) and one has that

$$\limsup_{t \to +\infty} \frac{\ln I(t)}{t} \le (pd + \gamma)(R_0^* - 1).$$

Then when $R_0^* < 1$, we obtain

$$\limsup_{t \to +\infty} \frac{\ln I(t)}{t} < 0$$

which implies that $\lim_{t\to+\infty} I(t) = 0$.

Next, we prove that $\lim_{t\to+\infty} R(t) = 0$ and $\lim_{t\to+\infty} S(t) = 1$, a.s. Since $\lim_{t\to+\infty} I(t) = 0$, then, the third equation of model (1.2) lead to $\lim_{t\to+\infty} R(t) = 0$, notice that S(t) + I(t) + R(t) = 1, thus, we have $\lim_{t\to+\infty} S(t) = 1$. This completes the proof of Theorem 3.1.

Remark 3.1. Theorem 3.1 shows that when $\sigma^2 > \max\{\beta, \beta^2/2(pd + \gamma)\}\)$, the infectious disease of model (1.2) goes to extinction almost surely, namely, large white noise stochastic disturbance is conducive to control infectious disease.

Remark 3.2. Note that $R_0^* = R_0 - \sigma^2/2(pd+\gamma)$. Obviously, $R_0 < 1$ leads to $R_0^* < 1$, while the other side is not true. This implies that the condition for I(t) going to extinction in the deterministic model is stronger than its stochastic counterpart due to the effect of the white noise disturbance.

4. Persistence in mean

Theorem 4.1. If $R_0^* > 1$, then the infectious disease I is permanent in mean, moreover, I satisfies

$$\liminf_{t \to +\infty} \langle I(t) \rangle \ge \frac{pd + \gamma}{\beta \left(1 + \frac{\gamma}{b}\right)} (R_0^* - 1),$$

a.s.

Proof. Integrating from 0 to t and dividing by t(> 0) on both sides of the third equation of model (1.2) yields

$$\frac{R(t) - R(0)}{t} = \gamma \langle I(t) \rangle - b \langle R(t) \rangle \triangleq \Theta(t).$$

Notice that $\langle S(t) \rangle + \langle I(t) \rangle + \langle R(t) \rangle = 1$, then one can get

$$\langle S(t) \rangle = 1 + \frac{\Theta(t)}{b} - \left(1 + \frac{\gamma}{b}\right) \langle I(t) \rangle$$

Applying Itô's formula, it has that

$$d(\ln I(t)) = \left[\beta S(t) - (pd + \gamma) - \frac{\sigma^2}{2}S^2(t)\right] dt + \sigma S(t) dB(t)$$
$$\geq \left[\beta S(t) - (pd + \gamma) - \frac{\sigma^2}{2}\right] dt + \sigma S(t) dB(t).$$
(4.1)

Integrating from 0 to t and dividing by t(> 0) on both sides of (4.1) yields

$$\frac{\ln I(t) - \ln I(0)}{t} \ge \beta \langle S(t) \rangle - \left[(pd + \gamma) + \frac{\sigma^2}{2} \right] + \frac{M(t)}{t},$$
$$= \beta \left(1 + \frac{\Theta(t)}{b} - \left(1 + \frac{\gamma}{b} \right) \langle I(t) \rangle \right)$$
$$- \left[pd + \gamma + \frac{\sigma^2}{2} \right] + \frac{M(t)}{t}.$$
(4.2)

From (4.2), we obtain

$$\langle I(t)\rangle \ge \frac{1}{\beta\left(1+\frac{\gamma}{b}\right)} \left[\beta\left(1+\frac{\Theta(t)}{b}\right) - pd - \gamma - \frac{\sigma^2}{2} - \frac{\ln I(t) - \ln I(0)}{t} + \frac{M(t)}{t}\right].$$
(4.3)

Since $R(t), I(t) \leq 1$, then one has that $\lim_{t \to +\infty} \frac{R(t)}{t} = 0$, $\lim_{t \to +\infty} \frac{\ln I(t)}{t} = 0$ and $\lim_{t \to +\infty} \Theta(t) = 0$. Notice that $\lim_{t \to +\infty} \frac{M(t)}{t} = 0$, by taking the inferior limit of both sides of (4.3), we have

$$\liminf_{t \to +\infty} \langle I(t) \rangle \ge \frac{1}{\beta \left(1 + \frac{\gamma}{b}\right)} \left[\beta - pd - \gamma - \frac{\sigma^2}{2} \right] = \frac{pd + \gamma}{\beta \left(1 + \frac{\gamma}{b}\right)} (R_0^* - 1).$$

This finishes the proof of Theorem 4.1.

Remark 4.1. Theorems 3.1 and 4.1 show that when the white noise is not large, R_0^* is the threshold associated with the extinction of infectious disease. Moreover, the condition for the disease to go to extinction or persistence depend on the intensity of white noise disturbances strongly. In addition to that small white noise disturbances will be beneficial to long-term prevalence of the disease, conversely, large white noise disturbances may cause the epidemic disease to be died out.

5. Conclusion and numerical simulation

In this paper, a stochastic SIR model with both horizontal and vertical transmission is proposed and investigated. The threshold dynamics are explored when the stochastic noise is small. Our results show that the threshold completely depends on the stochastic perturbation and the basic reproductive number of the corresponding deterministic model. Moreover, we find there exists significant difference between the threshold of deterministic and that of the stochastic model due to the effect of stochastic noise, and large noise is conducive to control the spread of diseases.

In the following, by employing the Euler Maruyama (EM) method [13,24], we make some numerical simulations to illustrate the extinction and persistence of the diseases in stochastic system and corresponding deterministic system for comparison.

For numerical simulations, we set parameters as $b = 0.008, \beta = 0.186, p = 0.04, d = 0.032, \gamma = 0.113$ in model (1.1). A simple computation shows that $R_0 = 1.6276 > 1$. Then model (1.1) has a unique stable positive equilibrium $P^*(0.6144, 0.0255, 0.3601)$, which implies that the disease of model (1.1) is permanent. (see Fig. 1)

Next, we consider the effect of stochastic white noise. Let $\sigma = 0.4$, obviously, $\sigma^2 > \max\{\beta, \beta^2/2(pd+\gamma)\}$, by Theorem 3.1, the disease dies out under a large white noise disturbance (see Fig. 2). If we change σ to 0.38, obviously, $\sigma^2 < \beta^2/2(pd+\gamma)$ and $R_0^* = 0.9791 < 1$, then by Theorem 3.1, the disease dies out (see Fig. 3). If we change σ to 0.1, obviously, $R_0^* = 1.3 > 1$, by Theorem 4.1, the disease is persistent (see Fig. 4). Fig 5 show the solution of the stochastic system oscillate around the positive equilibrium of the deterministic system.



Figure 1. Time series for S(t), I(t) and R(t) of the deterministic system with b = 0.008, $\beta = 0.186$, p = 0.04, d = 0.032, $\gamma = 0.113$, where $R_0 = 1.6276 > 1$.



Figure 2. Comparison of the deterministic system and stochastic system with $b = 0.008, \beta = 0.186, p = 0.04, d = 0.032, \gamma = 0.113, \sigma = 0.4$, where $R_0 = 1.6276 > 1$.



Figure 3. Comparison of the deterministic system and stochastic system, where $b = 0.008, \beta = 0.186, p = 0.04, d = 0.032, \gamma = 0.113, \sigma = 0.38$, where $R_0 = 1.6276 > 1, R_0^* = 0.9791 < 1$.



Figure 4. Comparison of the deterministic system and stochastic system, where $b = 0.008, \beta = 0.186, p = 0.04, d = 0.032, \gamma = 0.113, \sigma = 0.1$, where $R_0 = 1.6276 > 1, R_0^* = 1.5838 > 1$.



(a) 3D Phase diagram for S(t), I(t) and R(t) (b) 3D Phase diagram for S(t), I(t) and R(t) of the deterministic system.

Figure 5. Comparison of the deterministic system and stochastic system, where $b = 0.008, \beta = 0.186, p = 0.04, d = 0.032, \gamma = 0.113, \sigma = 0.1$, where $R_0 = 1.6276 > 1, R_0^* = 1.5838 > 1$.

References

- L. J. S. Allen, An Introduction to Stochastic Processes with Applications to Biology, Second Edition, CRC Press, 2015.
- [2] R. Anderson and R. May, Infectious Diseases in Humans: Dynamics and Control, Oxford University Press, Oxford, 1992.
- [3] N. Bacaër, On the stochastic SIS epidemic model in a periodic environment, Journal of Mathematical Biology, 2014, 71(2), 491–511.
- [4] E. Beretta, V. Kolmanovskii and L. Shaikhet, Stability of epidemic model with time delays influenced by stochastic perturbations, Mathematics and Computers in Simulation, 1998, 45(3-4), 269–277.
- [5] F. Bian, W. Zhao, Y. Song and R. Yue, Dynamical analysis of a class of prey-predator model with Beddington-Deangelis functional response, stochastic perturbation, and impulsive toxicant input, Complexity, 2017, 2017, Article ID 3742197.
- [6] Y. Cai, Y. Kang, M. Banerjee and W. Wang, A stochastic SIRS epidemic model with infectious force under intervention strategies, Journal of Differential Equations, 2015, 259(12), 7463–7502.
- [7] N. T. Dieu, D. H. Nguyen, N. H. Du and G. Yin, *Classification of asymp*totic behavior in a stochastic SIR model, SIAM Journal on Applied Dynamical Systems, 2016, 15(2), 1062–1084.
- [8] N. H. Du and N. N. Nhu, Permanence and extinction of certain stochastic SIR models perturbed by a complex type of noises, Applied Mathematics Letters, 2017, 64, 223–230.
- [9] T. Feng, X. Meng, L. Liu and S. Gao, Application of inequalities technique to dynamics analysis of a stochastic eco-epidemiology model, Journal of Inequalities and Applications, 2016, 2016(1), 327.

- [10] A. Gray, D. Greenhalgh, X. Mao and J. Pan, The SIS epidemic model with markovian switching, Journal of Mathematical Analysis and Applications, 2012, 394(2), 496–516.
- [11] C. Ji, D. Jiang and N. Shi, The behavior of an SIR epidemic model with stochastic perturbation, Stochastic Analysis and Applications, 2012, 30(5), 755–773.
- [12] W. O. Kermack and A. G. McKendrick, A contribution to the mathematical theory of epidemics, Proceedings of the Royal Society of London A: Mathematical, Physical and Engineering Sciences, 1927, 115(772), 700–721.
- [13] P. E. Kloeden and E. Platen, Higher-order implicit strong numerical schemes for stochastic differential equations, Journal of Statistical Physics, 1992, 66(1), 283–314.
- [14] A. Lahrouz, A. Settati and A. Akharif, Effects of stochastic perturbation on the SIS epidemic system, Journal of Mathematical Biology, 2017, 74(1), 469–498.
- [15] X. Leng, T. Feng and X. Meng, Stochastic inequalities and applications to dynamics analysis of a novel SIVS epidemic model with jumps, Journal of Inequalities and Applications, 2017, 2017(1), 138.
- [16] Y. Lin and D. Jiang, Long-time behaviour of a perturbed SIR model by white noise, Discrete and Continuous Dynamical Systems-Series B, 2013, 18(7), 1873–1887.
- [17] G. Liu, X. Wang, X. Meng and S. Gao, Extinction and persistence in mean of a novel delay impulsive stochastic infected predator-prey system with jumps, Complexity, 2017, 2017, Article ID 1950970.
- [18] L. Liu and X. Meng, Optimal harvesting control and dynamics of two-species stochastic model with delays, Advances in Difference Equations, 2017, 2017(1), 18.
- [19] M. Liu, C. Bai and K. Wang, Asymptotic stability of a two-group stochastic SEIR model with infinite delays, Communications in Nonlinear Science and Numerical Simulation, 2014, 19(10), 3444–3453.
- [20] Q. Liu and Q. Chen, Analysis of the deterministic and stochastic SIRS epidemic models with nonlinear incidence, Physica A: Statistical Mechanics and its Applications, 2015, 428, 140–153.
- [21] Q. Lu, Stability of SIRS system with random perturbations, Physica A: Statistical Mechanics and its Applications, 2009, 388(18), 3677–3686.
- [22] Z. Lu, X. Chi and L. Chen, The effect of constant and pulse vaccination on SIR epidemic model with horizontal and vertical transmission, Mathematical and Computer Modelling, 2002, 36(9), 1039–1057.
- [23] X. Lv, L. Wang and X. Meng, Global analysis of a new nonlinear stochastic differential competition system with impulsive effect, Advances in Difference Equations, 2017, 2017(1), 296.
- [24] X. Mao, Stochastic Differential Equations and Applications. 2nd Edition, Horwood Publishing, Chichester, UK, 2007.
- [25] A. Miao, X. Wang, T. Zhang et al., Dynamical analysis of a stochastic SIS epidemic model with nonlinear incidence rate and double epidemic hypothesis, Advances in Difference Equations, 2017, 2017(1), 226.

- [26] A. Miao, J. Zhang, T. Zhang and B. G. S. Pradeep, *Threshold dynamics of a stochastic SIR model with vertical transmission and vaccination*, Computational and Mathematical Methods in Medicine, 2017, 2017, Article ID 4820183.
- [27] H. Qi, L. Liu and X. Meng, Dynamics of a non-autonomous stochastic SIS epidemic model with double epidemic hypothesis, Complexity, 2017, 2017, Article ID 4861391.
- [28] W. Wang and W. Ma, A diffusive HIV infection model with nonlocal delayed transmission, Applied Mathematics Letters, 2018, 75, 96–101.
- [29] W. Wang and W. Ma, Hepatitis C virus infection is blocked by HMGB1: A new nonlocal and time-delayed reaction-diffusion model, Applied Mathematics and Computation, 2018, 320, 633–653.
- [30] W. Wang and W. Ma, Travelling wave solutions for a nonlocal dispersal HIV infection dynamical model, Journal of Mathematical Analysis and Applications, 2018, 457(1), 868–889.
- [31] W. Wang and T. Zhang, Caspase-1-mediated pyroptosis of the predominance for driving CD4⁺ T cells death: a nonlocal spatial mathematical model, Bulletin of Mathematical Biology, 2018, 80(3), 540–582.
- [32] C. Xu, Global threshold dynamics of a stochastic differential equation SIS model, Journal of Mathematical Analysis and Applications, 2017, 447(2), 736– 757.
- [33] S. Zhang, X. Meng, T. Feng and T. Zhang, Dynamics analysis and numerical simulations of a stochastic non-autonomous predator-prey system with impulsive effects, Nonlinear Analysis: Hybrid Systems, 2017, 26, 19–37.
- [34] T. Zhang, X. Meng and T. Zhang, Global dynamics of a virus dynamical model with cell-to-cell transmission and cure rate, Computational and Mathematical Methods in Medicine, 2015, 2015, Article ID 758362.
- [35] T. Zhang, X. Meng and T. Zhang, Global analysis for a delayed SIV model with direct and environmental transmissions, Journal of Applied Analysis & Computation, 2016, 6(2), 479–491.
- [36] T. Zhang, X. Meng, T. Zhang and Y. Song, Global dynamics for a new highdimensional SIR model with distributed delay, Applied Mathematics and Computation, 2012, 218(24), 11806–11819.
- [37] X. Zhang, D. Jiang, T. Hayat and B. Ahmad, Dynamics of a stochastic SIS model with double epidemic diseases driven by lévy jumps, Physica A: Statistical Mechanics and its Applications, 2017, 471, 767–777.
- [38] D. Zhao and S. Yuan, Break-even concentration and periodic behavior of a stochastic chemostat model with seasonal fluctuation, Communications in Nonlinear Science and Numerical Simulation, 2017, 46, 62–73.
- [39] J. Zhao, L. Wang and Z. Han, Stability analysis of two new SIRS models with two viruses, International Journal of Computer Mathematics, 2017, DOI: 10.1080/00207160.2017.1364369.
- [40] W. Zhao, J. Li and X. Meng, Dynamical analysis of SIR epidemic model with nonlinear pulse vaccination and lifelong immunity, Discrete Dynamics in Nature and Society, 2015, 2015, Article ID 848623.

- [41] W. Zhao, T. Zhang, Z. Chang et al., Dynamical analysis of SIR epidemic models with distributed delay, Journal of Applied Mathematics, 2013, 2013, Article ID 154387.
- [42] Y. Zhao, Y. Lin, D. Jiang et al., Stationary distribution of stochastic SIRS epidemic model with standard incidence, Discrete and Continuous Dynamical Systems-Series B, 2016, 21(7), 2363–2378.
- [43] Y. Zhao, S. Yuan and T. Zhang, Stochastic periodic solution of a nonautonomous toxic-producing phytoplankton allelopathy model with environmental fluctuation, Communications in Nonlinear Science and Numerical Simulation, 2017, 44, 266–276.
- [44] J. Zhou, Y. Yang and T. Zhang, Global stability of a discrete multigroup SIR model with nonlinear incidence rate, Mathematical Methods in the Applied Sciences, 2017, 40(14), 5370–5379.
- [45] Y. Zhou, S. Yuan and D. Zhao, Threshold behavior of a stochastic SIS model with jumps, Applied Mathematics and Computation, 2016, 275, 255–267.