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GLOBAL DYNAMICS OF DETERMINISTIC AND STOCHASTIC SIRS EPIDEMIC MODELS*

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Abstract In this paper, we analyze the dynamic behavior of Heesterbeek et al. [12] obtained saturating contact rate applied to SIRS epidemic model. We define two threshold values, the deterministic basic reproduction number R_0 and the stochastic basic reproduction number R_0^s, by comparing the value with one to determine the persistence and extinction of the disease. For deterministic model, if $R_0 < 1$, we show that the disease-free equilibrium is globally asymptotically stable; while if $R_0 > 1$, the system admits a unique endemic equilibrium which is locally asymptotically stable. For stochastic model, we also establish the threshold value R_0^s for disease persistence and extinction. Finally, some numerical simulations are presented to illustrate our theoretical results. Our results prove that large stochastic perturbation will lead to the extinction of diseases with probability one, revealing the significant influence of stochastic perturbation on diseases and the importance of incorporating stochastic perturbation into deterministic model.

Keywords Epidemic, stochastic, stationary solution, extinction, noise.

1. Introduction

Infectious diseases kill millions of people every year. For example, more than 20 million people died of plague in India at the beginning of the 20th century, and the black death killed many more Europeans in 1346 [32]. Therefore, how to control the spread of disease is one of the most important hot topics, which is necessary to understand and analyze the transmission mechanism of infectious diseases. Mathematical model is an important tool to analyze the spread and control of infectious diseases [1, 13, 38]. Nowadays, more and more mathematicians are working on the dynamics of infectious disease models [9, 10, 19, 30, 33, 36, 37, 39, 42, 43]. For example, Lan et al. [19] considered a stochastic SIQR epidemic model with saturating incidence. They obtained the existence of a unique stable stationary distribution using the Markov semigroup theory and pointed out that environmental noise plays a positive role in controlling disease. The classical SIR model is the Kermack and Mckendrick model, and the total population is divided into three compartments Susceptible(S), Infective(I), and Removed(R). In many infectious diseases, such as pertussis, dysentery, influenza, and malaria, recovered individuals are once again

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susceptible (temporarily immune). Therefore, many scholars proposed the SIRS epidemic models [2, 15, 20, 23, 29]. Particularly, Wang et al. [2] considered the following general SIRS infectious disease model:

$$\begin{cases} \dfrac{dS(t)}{dt} = b - g(S,I) - dS(t) + \delta R(t), \\ \dfrac{dI(t)}{dt} = g(S,I) - (\mu + \alpha + d)I(t), \\ \dfrac{dR(t)}{dt} = \mu I(t) - (d+\delta)R(t), \end{cases} \tag{1.1}$$

where $S(t)$, $I(t)$, $R(t)$ denotes the numbers of susceptible individuals, infective individuals and removed individuals, respectively. Parameters b, d, δ, α and μ are all positive constants. b represents the recruitment rate of the population, d denotes the natural death rate of the population, δ is the rate at which removed individuals loss immunity and return to the susceptible class, α stands for the diseased death rate, μ is the recovery rate of infective individuals. The transmission function $g(S,I)$ plays an important role in epidemiological models. Up to now, many experts have studied many kinds of incidences of epidemic models [5, 8, 12, 26, 28]. Such as, the bilinear incidence rate $g(S,I) = \beta SI$, the standard incidence rate $g(S,I) = \frac{\beta SI}{N}$ and so on. Especially, Heesterbeek et al. [12] proposed a saturating contact rate $g(S,I) = \frac{\beta SI}{h(N)}$, where $\frac{N}{h(N)}$ is the "unknown" probability for an individual to take part in a contact; One of the reasonable requirements for $h(N)$ is that it's a non-decreasing function of N. N represents the total population size and β is the probability per unit time of transmitting the infection between two individuals taking part in a contact. Finally, they got this expression $h(N) = \frac{1+fN+\sqrt{1+2fN}}{f}$. This saturating incidence rate is more realistic than the bilinear rate and the standard rate, as it takes into account the crowding effect and behavioral change of the infective individuals and prevents the unboundedness of the contact rate occurring by choosing suitable parameters. Furthermore, combining $g(S,I) = \frac{\beta SI}{h(N)}$ into model (1.1), we get the following model:

$$\begin{cases} \dfrac{dS(t)}{dt} = b - \dfrac{\beta S(t)I(t)}{h(N)} - dS(t) + \delta R(t), \\ \dfrac{dI(t)}{dt} = \dfrac{\beta S(t)I(t)}{h(N)} - (\mu + \alpha + d)I(t), \\ \dfrac{dR(t)}{dt} = \mu I(t) - (d+\delta)R(t), \end{cases} \tag{1.2}$$

where $N(t) = S(t) + I(t) + R(t)$, other parameters have the same meaning as the system (1.1). One disadvantage of deterministic models is that they ignore the effect of environmental noise on disease transmission. Numerous studies have shown that environmental noise may affect the spread and control measures of diseases. For instance, in temperate regions, influenza A outbreaks occur in winter. At the same time, the obtained result shown that relative humidity also affects disease transmission and survival [17]. Therefore, the stochastic model is more rational and realistic than the deterministic model. Recently, many researchers [3, 4, 16, 21, 24, 25, 27, 35, 40, 41] have introduced stochastic environmental perturbations into deterministic models. Liu et al. [25] studied a stochastic SEIR epidemic model with standard incidence. They established sufficient conditions for extinction of the

disease. And they also showed the existence of the stationary distribution of system, which implies the disease is persistent. Their results improve the previous result to a greater extent. Cao et al. [4] assumed that the disease transmission coefficient β is subject to the environmental white noise. They concluded that as long as the white noise is large enough, the disease will die out almost surely. Motivated by these works, we assume that the environmental fluctuation affects the rate of transmission per contact. Hence, in this paper, we formulate the fluctuation in the environment by a stochastic process $\beta + \sigma\dot{B}(t)$ instead of a constant β. Whereupon, we obtain the following stochastic model:

$$\begin{cases} dS(t) = [b - \dfrac{\beta S(t)I(t)}{h(N)} - dS(t) + \delta R(t)]dt - \dfrac{\sigma S(t)I(t)}{h(N)}dB(t), \\ dI(t) = [\dfrac{\beta S(t)I(t)}{h(N)} - (\mu + \alpha + d)I(t)]dt + \dfrac{\sigma S(t)I(t)}{h(N)}dB(t), \\ dR(t) = [\mu I(t) - (d + \delta)R(t)]dt, \end{cases} \tag{1.3}$$

where $B(t)$ represents standard Brownian motion, σ^2 denotes the intensity of white noise.

Throughout this paper, unless otherwise specified, let $(\Omega, \mathscr{F}, \{\mathscr{F}_t\}_{t\geq 0}, P)$ denote a complete probability space with a filtration $\{\mathscr{F}_t\}_{t\geq 0}$ satisfying the usual conditions (i.e. it is right continuous and $\mathscr{F}_0$ contains all P-null sets). Moreover, for the sake of convenience, we define the following symbols. Let $\mathbb{R}_+ = [0, \infty), \mathbb{R}^n_+ = \{x = (x_1, ..., x_n) \in \mathbb{R}^n : x_i > 0, i = 1, ..., n\}$; C^2 denotes the class of functions in $\mathbb{R}^n$ which are twice continuously differentiable; *a.s.* means almost surely; *i.e.* means that is to say.

This paper mainly discusses the following problems:

- For model (1.2), how to define the basic reproduction number ?
- For model (1.3), under what conditions the disease will become extinct? and under what conditions the disease will stochastic persistent?
- What factors will play an important role in the extinction of infectious diseases?

This paper is organized as follows. In section 2, we show that the disease-free equilibrium is globally asymptotically stable if the basic reproduction number $R_0 < 1$; and if $R_0 > 1$, model (1.2) admits a unique endemic equilibrium which is locally asymptotically stable. In Section 3, we also define a stochastic basic reproduction number R_0^s. If $R_0^s < 1$ and under additional conditions, the disease will extinct. If $R_0^s > 1$, the model (1.3) has a stationary distribution which implies that the disease is persistent. Finally, we introduce several numerical simulations to illustrate the main results and the paper ends with some discussion in section 4.

2. Dynamics of the deterministic model

From system (1.2), we can obtain that the total population $\dot{N} = b - dN - \alpha I$. In this way, we get $\Gamma = \{(S, I, R) \mid S \geq 0, I \geq 0, R \geq 0, \frac{b}{d+\alpha} \leq S + I + R \leq \frac{b}{d}\}$ is a positive invariant set of system (1.2). By simple calculation, it's easy to get that the system (1.2) has two equilibrium states: the disease-free equilibrium state $E_0 = (S_0, 0, 0) = (\frac{b}{d}, 0, 0)$ which exists for all parameter values. Define the basic reproduction number $R_0 = \frac{\beta b}{d(\mu+d+\alpha)h(\frac{b}{d})}$.

Next, we will calculate the endemic equilibrium. From model (1.2), the coordinates of an equilibrium $E^* = (S^*, I^*, R^*)$ satisfy

$$\begin{cases} b - \dfrac{\beta S^* I^*}{h(N^*)} - dS^* + \delta R^* = 0, \\ \dfrac{\beta S^* I^*}{h(N^*)} - (\mu + \alpha + d) I^* = 0, \\ \mu I^* - (d + \delta) R^* = 0, \end{cases} \tag{2.1}$$

from the second equation we can obtain that

$$S^* = \frac{d + \mu + \alpha}{\beta} h(N^*). \tag{2.2}$$

Similarly, from the third equation we have

$$I^* = \frac{d + \delta}{\mu} R^*. \tag{2.3}$$

Substituting (2.2) and (2.3) into the first equation in (2.1) gives

$$R^* = \frac{\mu[b - \frac{d(d+\mu+\alpha)}{\beta} h(N^*)]}{(d + \mu + \alpha)(d + \delta) - \mu\delta} = \frac{\frac{d(d+\mu+\alpha)}{\beta} h(N^*) \mu[\frac{\beta b}{d(d+\mu+\alpha)h(N^*)} - 1]}{(d + \mu + \alpha)(d + \delta) - \mu\delta}.$$

One can see that, if $R_0 > 1$, system (1.2) has a unique endemic equilibrium $E^* = (S^*, I^*, R^*)$ which is locally asymptotic stable.

Theorem 2.1. *If $R_0 < 1$, the disease-free equilibrium $E_0 = (\frac{b}{d}, 0, 0)$ is globally asymptotically stable; while if $R_0 > 1$, E_0 is unstable, and system (1.2) has a unique endemic equilibrium $E^* = (S^*, I^*, R^*)$ which is locally asymptotically stable.*

Proof. When $R_0 < 1$, we will show that $\lim\limits_{t\to\infty} I(t) = 0$. From the second equation of model (1.2) that

$$\begin{aligned} \frac{dI}{dt} =& (\frac{\beta S}{h(N)} - (\mu + d + \alpha)) I \\ \leq& (\frac{\beta b}{h(\frac{b}{d}) d} - (\mu + d + \alpha)) I \\ =& (\mu + d + \alpha)(R_0 - 1) I. \end{aligned}$$

The linear comparison system

$$\frac{d\tilde{I}}{dt} = (\mu + d + \alpha)(R_0 - 1)\tilde{I}, \ \tilde{I}(0) = I(0).$$

Obviously, $\tilde{I} = e^{(d+\mu+\alpha)(R_0-1)t}$. Consequently, $\lim\limits_{t\to\infty} \tilde{I}(t) = 0$. By the comparison principle, we have $\lim\limits_{t\to\infty} I(t) = 0$. Therefore, for any small $\epsilon > 0$, there exists $T > 0$, such that for all $t \geq T$, $I(t) \leq \epsilon$. From the first equation of model (1.2), we have

$$b - dN - \alpha\epsilon \leq \frac{dN}{dt} \leq b - dN.$$

For the arbitrariness of ϵ, it is easy to get $\lim\limits_{t\to\infty} N(t) = \frac{b}{d}$. Hence, the disease-free equilibrium E_0 is globally asymptotically stable, if $R_0 < 1$.

When $R_0 > 1$, the Jacobian matrix of model (1.2) at E_0 is

$$J(E_0) = \begin{pmatrix} -d & -\frac{\beta b}{dh(\frac{b}{d})} & \delta \\ 0 & \frac{\beta b}{dh(\frac{b}{d})} - (\mu + d + \alpha) & 0 \\ 0 & \mu & -(d+\delta) \end{pmatrix},$$

which obeys the following characteristic equation:

$$(\lambda + d)[\lambda - (\frac{\beta b}{dh(\frac{b}{d})} - (\mu + d + \alpha))](\lambda + \delta + d) = 0.$$

Therefore, $\lambda_1 = -d < 0$, $\lambda_2 = (\mu+d+\alpha)(\frac{\beta b}{dh(\frac{b}{d})(\mu+d+\alpha)}-1) > 0$ and $\lambda_3 = -\delta-d < 0$.

Whereupon, E_0 is unstable. Then, we will discuss the stability of endemic equilibrium.

The stability of the endemic equilibrium $E^* = (S^*, I^*, R^*)$ of system (1.2) is determined by the Jacobian matrix

$$J(E^*) = \begin{pmatrix} -d - \frac{\beta I^*}{h(N^*)} & -\frac{\beta S^*}{h(N^*)} & \delta \\ \frac{\beta I^*}{h(N^*)} & -(\mu + \alpha + d) & 0 \\ 0 & \mu & -(d+\delta) \end{pmatrix},$$

we get the following characteristic equation:

$$\lambda^3 + B_1\lambda^2 + B_2\lambda + B_3 = 0,$$

where $B_1 = 3d + \mu + \delta + \alpha + M$, $B_2 = (d + \mu + \alpha)(d + \delta) + d(d + \mu + \alpha)(d + \delta) + (d + M)(d + \delta)$, $B_3 = d(d + \mu + \alpha)(d + \delta) + \mu\delta M$, $M = \frac{(d+\delta)k}{\mu h(N^*)R^*}$.

For $R_0 > 1$, we have $B_j > 0$ $(j = 1, 2, 3)$. Furthermore, by simple calculation, we have

$$H_1 = B_1 > 0,$$

$$H_2 = \begin{vmatrix} B_1 & B_3 \\ 1 & B_2 \end{vmatrix} = B_1B_2 - B_3 > 0,$$

$$H_3 = \begin{vmatrix} B_1 & B_3 & 0 \\ 1 & B_2 & 0 \\ 0 & B_1 & B_3 \end{vmatrix} = B_3(B_1B_2 - B_3) > 0.$$

Hence the Routh-Hurwitz conditions are satisfied. Therefore, the endemic equilibrium E^* is local asymptotically stable, if $R_0 > 1$. This completes the proof. □

Remark 2.1. Theorems 2.1 reveals that the basic reproduction number R_0 is a threshold value of model (1.2) determining whether the disease is extinct or not. That is to say, if $R_0 < 1$, the disease is extinct, while if $R_0 > 1$, the disease is persistent.

3. Dynamics of stochastic model

In this section, we mainly study the dynamic behavior of the model (1.3). To study the dynamics of infectious disease models, the main concerns are how to control outbreaks of infectious diseases and under what conditions they are persistent. And can we still get a threshold for disease persistence or extinction?

3.1. Existence and uniqueness of the global positive solution

The following result is related to the existence and uniqueness of the global positive solution, which is a prerequisite for researching the long term behavior of model (1.3).

Theorem 3.1. *For any initial value* $(S(0), I(0), R(0)) \in \mathbb{R}^3_+$*, system (1.3) has a unique global positive solution* $(S(t), I(t), R(t))$ *for* $t \geq 0$*. Furthermore, the solution will remain in* $\mathbb{R}^3_+$ *with probability one, namely* $(S(t), I(t), R(t)) \in \mathbb{R}^3_+$ *for all* $t \geq 0$ *almost surely.*

Proof. Note that the coefficients of our model (1.3) are locally Lipschitz continuous and for any given initial value $(S(0), I(0), R(0)) \in \mathbb{R}^3_+$, there exists a unique local solution $(S(t), I(t), R(t))$ on $t \in [0, \tau_e)$, where τ_e is the explosion time [31]. Therefore, the unique local solution to model (1.3) is positive by Itõ's formula. In order to demonstrate that this solution is global, it's sufficient to prove that $\tau_e = \infty$ a.s.. Let $k_0 \geq 0$ be a sufficiently large constant for every component of $(S(0), I(0), R(0))$ all lying within the interval $[\frac{1}{k_0}, k_0] \times [\frac{1}{k_0}, k_0] \times [\frac{1}{k_0}, k_0]$. For each integer $k \geq k_0$, we define the stopping time as follows:

$$\tau_k = \inf\{t \in [0, \tau_e) : \min\{S(t), I(t), R(t)\} \leq \frac{1}{k} \quad \text{or} \quad \max\{S(t), I(t), R(t)\} \geq k\}.$$

Throughout this paper, we set $\inf \emptyset = \infty$(as usual $\emptyset$ denotes the empty set). Clearly, τ_k is increasing as $k \to \infty$. Set $\tau_\infty = \lim\limits_{k\to\infty} \tau_k$, hence $\tau_\infty \leq \tau_e$ a.s.. If we can show that $\tau_\infty = \infty$ a.s., then $\tau_e = \infty$ a.s. In other words, to complete the proof, we only need to show that $\tau_\infty = \infty$ a.s.. If this statement is false, there is a pair of constants $T > 0$ and $\varepsilon \in (0, 1)$ such that $P\{\tau_k \leq T\} \geq \varepsilon$ for any $k \geq k_0$.

We define a C^2-function $V : \mathbb{R}^3_+ \to \mathbb{R}_+$ as follows:

$$V(S, I, R) = S - \ln S - 1 + I - \ln I - 1 + R - \ln R - 1,$$

the nonnegativity of this function can be obtained from

$$x - \ln x - 1 \geq 0, \quad x > 0.$$

Applying Itô's formula yields

$$dV = LVdt + \frac{\sigma(I - S)}{h(N)} dB(t),$$

and

$$\begin{aligned}\mathcal{L}V =& b-dS-\frac{\beta SI}{h(N)}+\delta R-\frac{b}{S}+d+\frac{\beta I}{h(N)}-\frac{\delta}{S}R+\frac{\sigma^2I^2}{2h^2(N)}+\frac{\beta SI}{h(N)}-(d+\mu+\alpha)I\\ &-\frac{\beta S}{h(N)}+d+\mu+\alpha+\frac{\sigma^2S^2}{2h^2(N)}+\mu I-(d+\delta)R-\frac{\mu I}{R}+d+\delta\\ \leq& 3d+b+\mu+\alpha+\delta+\frac{\beta I}{h(N)}+\frac{\sigma^2N^2}{2h^2(N)}\\ \leq& 3d+b+\mu+\alpha+\delta+\beta+\frac{\sigma^2}{2}\\ :=& K,\end{aligned}$$

where K is a positive number, the remainder of the proof follows that in Li et al [22], here, we omit it. The proof is complete. □

Remark 3.1. From theorem 3.1, we know that for any initial value $(S(0), I(0), R(0))\in \mathbb{R}^3_+$, system (1.3) has a unique positive solution $(S(t), I(t), R(t)) \in \mathbb{R}^3_+$ with probability one. From system (1.3), it is straightforward to get $\dot{N} = b - dN - \alpha I$, then, the region $\Gamma = \{(S, I, R) \in \mathbb{R}^3_+ : \frac{b}{d+\alpha} \leq N \leq \frac{b}{d}\}$ also is a positively invariant set of system (1.3). In the following, we always assume that the initial value $(S(0), I(0), R(0)) \in \Gamma$.

3.2. Extinction of disease

Obtaining sufficient conditions for the extinction of infectious diseases can provide us with effective measures, so it is important to study it. In this subsection, we shall establish sufficient conditions for extinction of the disease in the stochastic model (1.3).

Theorem 3.2. *Let $(S(t), I(t), R(t))$ be a solution of model (1.3) with initial value $(S(0), I(0), R(0)) \in \Gamma$. Assume one of the following conditions is satisfied*
(i) $\sigma^2 > \max\{\frac{\beta^2}{2(\mu+d+\alpha)}, \frac{d\beta h(\frac{b}{d})}{b}\}$;
(ii) $\sigma^2 < \frac{d\beta h(\frac{b}{d})}{b}$ *and* $R_0^s = \frac{\beta b}{d(\mu+d+\alpha+\frac{\sigma^2b^2}{2h^2(\frac{b}{d})d^2})h(\frac{b}{d})} < 1$.
Then, the disease $I(t)$ goes to extinction exponentially with probability one, i.e.,

$$\lim_{t\to\infty} I(t) = 0 \quad a.s..$$

Proof. For model (1.3), applying Itô's formula, one can obtain that

$$\begin{aligned} d\ln I &= [\frac{\beta S}{h(N)} - (d+\mu+\alpha) - \frac{\sigma^2S^2}{2h^2(N)}]dt + \frac{\sigma S}{h(N)}dB(t)\\ &= \Psi(\frac{S}{h(N)})dt + \frac{\sigma S}{h(N)}dB(t),\end{aligned} \tag{3.1}$$

where

$$\begin{aligned}\Psi(\frac{S}{h(N)}) =&\frac{\beta S}{h(N)} - (d+\mu+\alpha) - \frac{\sigma^2 S^2}{2h^2(N)}\\ =&-\frac{\sigma^2}{2}(\frac{S}{h(N)} - \frac{\beta}{\sigma^2})^2 + \frac{\beta^2}{2\sigma^2} - (d+\mu+\alpha)\\ \leq&\frac{\beta^2}{2\sigma^2} - (d+\mu+\alpha).\end{aligned}$$

Then $d\ln I \leq (\frac{\beta^2}{2\sigma^2} - (d+\mu+\alpha))dt + \frac{\sigma S}{h(N)}dB(t)$. Integrating both sides from 0 to t, dividing by t and taking the limit, since the strong law of large numbers for local martingales, we derive that $\lim\limits_{t\to\infty}\frac{1}{t}\int_0^t \frac{\sigma S(s)}{h(N(s))}dB(s) = 0$ almost surely and

$$\limsup_{t\to\infty}\frac{\ln I(t)}{t} \leq \frac{\beta^2}{2\sigma^2} - (d+\mu+\alpha) < 0 \quad a.s.. \tag{3.2}$$

Next, we consider the case that $\frac{\beta h(\frac{b}{d})}{\sigma^2} > \frac{b}{d}$, that is $\sigma^2 < \frac{d\beta h(\frac{b}{d})}{b}$. Noting that $\Psi(x)$ is monotone increasing for $x \in (0, \frac{\beta}{\sigma^2}]$.

From (3.1), we have

$$\begin{aligned}d\ln I =& [-\frac{\sigma^2}{2}(\frac{b}{dh(\frac{b}{d})} - \frac{\beta}{\sigma^2})^2 + \frac{\beta^2}{2\sigma^2} - (d+\mu+\alpha)]dt + \frac{\sigma S}{h(N)}dB(t)\\ \leq& [(d+\mu+\alpha+\frac{\sigma^2 b^2}{2h^2(\frac{b}{d})d^2})(R_0^s - 1)]dt + \frac{\sigma S}{h(N)}dB(t).\end{aligned}$$

Similarly, one can see that

$$\limsup_{t\to\infty}\frac{\ln I(t)}{t} \leq (d+\mu+\alpha+\frac{\sigma^2 b^2}{2h^2(\frac{b}{d})d^2})(R_0^s - 1) < 0 \quad a.s.. \tag{3.3}$$

From (3.2) and (3.3), we have $\lim\limits_{t\to\infty} I(t) = 0 \quad a.s.$. In other words, the disease $I(t)$ dies out with probability one. This completes the proof. □

Remark 3.2. From theorem 3.2, one can see that diseases go extinct in two ways. One is the environmental disturbance is large enough; The other is the threshold R_0^s is less than one, plus additional conditions, for small intensity noise case. This reveals that environmental disturbance has a great impact on infectious diseases, and the threshold R_0^s is also the key value to determine whether infectious diseases break out or not.

3.3. Existence of stationary solution

In this subsection, we investigate the existence of the solution to model (1.3) which is a stationary Markov process, which implies that the disease is persistent.

In general, consider the n-dimensional stochastic differential equation

$$dx(t) = f(x(t))dt + \sum_{i=1}^{n} g_s(x(t))dB_s(t) \quad t \geq t_0, \tag{3.4}$$

with the initial value $x(t_0) = x_0 \in \mathbb{R}^n, B(t)$ denotes the $n-$dimensional standard Brownian motion defined on a complete probability space $(\Omega, \mathscr{F}, \{\mathscr{F}_t\}_{t\geq 0}, P)$.

By the definition of stochastic differential equation, Eq. (3.4) is equivalent to the following stochastic integral equation

$$x(t) = x_0 + \int_{t_0}^{t} f(x(s))ds + \sum_{s=1}^{n} \int_{t_0}^{t} g_s(x(t))dB_s(t) \quad t \geq t_0. \tag{3.5}$$

Lemma 3.1. *Suppose that the vectors $f(x), g_1(x), ..., g_n(x)(t \geq t_0, x \in \mathbb{R}^n)$ are continuous functions of x, and independent, such that for some constant M the following conditions hold in U_G for every $G > 0$:*

$$(i) \quad |f(x) - f(y)| + \sum_{s=1}^{n} |g_s(x) - g_s(y)| \leq M|x - y|; \tag{3.6}$$

$$(ii) \quad |f(x)| + \sum_{s=1}^{n} |g_s(x)| \leq M(1 + |x|); \tag{3.7}$$

and there exists a function $V(x) \in C^2$ in $\mathbb{R}^n$ with the properties

$$V(x) \geq 0, \quad \text{and} \quad \sup_{|x|>G} -M_G \to -\infty \quad \text{as} \quad G \to \infty. \tag{3.8}$$

Suppose further that the process $X^x(t)$ is regular for at least one $x \in \mathbb{R}^n$. Then there exists a solution of (3.5) which is a stationary Markov process.

Remark 3.3. Condition (3.8) can be replaced by the weaker condition that $\mathcal{L}V \leq -1$ outside some compact set (see [11, Chapter 4]).

Theorem 3.3. *Let $(S(t), I(t), R(t))$ be the solution of system (1.3) with any initial value $(S(t), I(t), R(t)) \in \Gamma$. Assume that $R_0^s > 1$, then the solution $(S(t), I(t), R(t))$ is a stationary solution of $\mathbb{R}^3_+$.*

Proof. Define a C^2-function $G : \Gamma \to \mathbb{R}$ by

$$\begin{aligned} G(S, I, R) &= g(-l_1 \ln S - \ln I - l_2 h^2(N)) - (\ln S + \ln R) - \ln(\frac{b}{d} - N) - \ln(N - \frac{b}{d+\alpha}) \\ &:= gV_1 + V_2 + V_3 + V_4, \end{aligned}$$

where $V_1 = -l_1 \ln S - \ln I - l_2 h^2(N)$, $V_2 = -\ln S - \ln R$, $V_3 = -\ln(\frac{b}{d} - N)$, $V_4 = -\ln(N - \frac{b}{d+\alpha})$, $l_1 = \frac{d+\mu+\alpha+\frac{\sigma^2 b^2}{2h^2(\frac{b}{d})d^2}}{d}$, $l_2 = \frac{d+\mu+\alpha+\frac{\sigma^2 b^2}{2h^2(\frac{b}{d})d^2}}{2bh(\frac{b}{d})}$ and $g > 0$ satisfies the following condition

$$-3g(d+\mu+\alpha+\frac{\sigma^2 b^2}{2h^2(\frac{b}{d})d^2})(\sqrt[3]{R_0^s}-1)+4d+\delta+\alpha+\frac{\beta b}{h(\frac{b}{d})d}+\frac{\sigma^2 b^2}{2h^2(\frac{b}{d})d^2} \leq -2. \tag{3.9}$$

Obviously, $G(S, I, R)$ is a continue function of (S, I, R). Thus there exists a unique minimum value point $(S^*(k), I^*(k), R^*(k))$ of $G(S, I, R)$ in Γ and $S^*(k) > 0, I^*(k) > 0, R^*(k) > 0$. Then we define a nonnegative C^2-function $V(S, I, R) : \Gamma \to \mathbb{R}_+$ as follows:

$$V(S, I, R) = G(S, I, R) - G(S^*(k), I^*(k), R^*(k)). \tag{3.10}$$

By using Itô's formula to V_1 yields

$$\mathcal{L}V_1 = l_1(-\frac{b}{S} + d + \frac{\beta I}{h(N)} - \frac{\delta R}{S} + \frac{\sigma^2 I^2}{2h^2(N)}) - \frac{\beta S}{h(N)} + d + \mu + \alpha + \frac{\sigma^2 S^2}{2h^2(N)}$$

$$
\begin{aligned}
&- 2l_2(1+\frac{1}{\sqrt{1+2fN}})(b-dN-\alpha I)h(N)\\
\leq &- l_1\frac{b}{S}-\frac{\beta S}{h(N)}-2l_2bh(N)+{}_1d+2l_2bh(\frac{b}{d})+d+\mu+\alpha+\frac{\sigma^2b^2}{2h^2(\frac{b}{d})d^2}\\
&+(l_1\frac{\beta}{h(\frac{b}{d})}+2l_2\alpha h(N)(1+\frac{1}{\sqrt{1+2fN}}))I+l_1\frac{\sigma^2}{2h^2(\frac{b}{d})}I^2\\
\leq &- 3\sqrt[3]{2l_1l_2b^2\beta}+3(d+\mu+\alpha+\frac{\sigma^2b^2}{2h^2(\frac{b}{d})d^2})+(l_1\frac{\beta}{h(\frac{b}{d})}+4l_2\alpha h(\frac{b}{d}))I+l_1\frac{\sigma^2}{2h^2(\frac{b}{d})}I^2\\
= &- 3(d+\mu+\alpha+\frac{\sigma^2b^2}{2h^2(\frac{b}{d})d^2})(\sqrt[3]{R_0^s}-1)+(l_1\frac{\beta}{h(\frac{b}{d})}+4l_2\alpha h(\frac{b}{d}))I+l_1\frac{\sigma^2}{2h^2(\frac{b}{d})}I^2,
\end{aligned}
\tag{3.11}
$$

likewise

$$
\begin{aligned}
\mathcal{L}V_2 = &- \frac{b}{S}+d+\frac{\beta I}{h(N)}-\frac{\delta R}{S}+\frac{\sigma^2I^2}{2h^2(N)}-\frac{\mu I}{R}+d+\delta\\
\leq &- \frac{b}{S}-\frac{\mu I}{R}+2d+\delta+\frac{\beta b}{h(\frac{b}{d})d}+\frac{\sigma^2b^2}{2h^2(\frac{b}{d})d^2},
\end{aligned}
\tag{3.12}
$$

similarly

$$
\mathcal{L}V_3=\frac{b-dN-\alpha I}{\frac{b}{d}-N}=d-\frac{\alpha I}{\frac{b}{d}-N},
\tag{3.13}
$$

and

$$
\begin{aligned}
\mathcal{L}V_4 &= \frac{dN-b+\alpha I}{N-\frac{b}{d+\alpha}}\\
&\leq \frac{(d+\alpha)N-\alpha S}{N-\frac{b}{d+\alpha}}\\
&= d+\alpha-\frac{\alpha S}{N-\frac{b}{d+\alpha}},
\end{aligned}
\tag{3.14}
$$

combining (3.9), (3.11), (3.12), (3.13) and (3.14), we have

$$
\begin{aligned}
\mathcal{L}V \leq &- 3g(d+\mu+\alpha+\frac{\sigma^2b^2}{2h^2(\frac{b}{d})d^2})(\sqrt[3]{R_0^s}-1)+g(l_1\frac{\beta}{h(\frac{b}{d})}+4l_2\alpha h(\frac{b}{d}))I+gl_1\frac{\sigma^2}{2h^2(\frac{b}{d})}I^2\\
&- \frac{b}{S}-\frac{\mu I}{R}+4d+\delta+\alpha+\frac{\beta b}{h(\frac{b}{d})d}+\frac{\sigma^2b^2}{2h^2(\frac{b}{d})d^2}-\frac{\alpha I}{\frac{b}{d}-N}-\frac{\alpha S}{N-\frac{b}{d+\alpha}}\\
\leq &- 2+g(l_1\frac{\beta}{h(\frac{b}{d})}+4l_2\alpha h(\frac{b}{d}))I+gl_1\frac{\sigma^2}{2h^2(\frac{b}{d})}I^2-\frac{b}{S}-\frac{\mu I}{R}-\frac{\alpha I}{\frac{b}{d}-N}-\frac{\alpha S}{N-\frac{b}{d+\alpha}}.
\end{aligned}
$$

Considering the following bounded closed set

$$
D_\varepsilon=\{(S,I,R)\in\Gamma:\varepsilon\leq S\leq\frac{b}{d},\varepsilon\leq I\leq\frac{b}{d},\varepsilon^2\leq R\leq\frac{b}{d},\frac{b}{d+\alpha}+\varepsilon^2\leq N\leq\frac{b}{d}-\varepsilon^2\},
$$

where $\varepsilon>0$ is a sufficiently small number. In the set $\Gamma\setminus D_\varepsilon$, we can choose ε sufficiently small such that the following conditions hold

$$
-2+g(l_1\frac{\beta}{h(\frac{b}{d})}+4l_2\alpha h(\frac{b}{d}))\frac{b}{d}+gl_1\frac{\sigma^2}{2h^2(\frac{b}{d})}\frac{b^2}{d^2}-\frac{b}{\varepsilon}<-1,
\tag{3.15}
$$

$$-2+g(l_1\frac{\beta}{h(\frac{b}{d})}+4l_2\alpha h(\frac{b}{d}))\varepsilon+gl_1\frac{\sigma^2}{2h^2(\frac{b}{d})}\varepsilon^2<-1, \tag{3.16}$$

$$-2+g(l_1\frac{\beta}{h(\frac{b}{d})}+4l_2\alpha h(\frac{b}{d}))\frac{b}{d}+gl_1\frac{\sigma^2}{2h^2(\frac{b}{d})}\frac{b^2}{d^2}-\frac{\mu}{\varepsilon}<-1, \tag{3.17}$$

$$-2+g(l_1\frac{\beta}{h(\frac{b}{d})}+4l_2\alpha h(\frac{b}{d}))\frac{b}{d}+gl_1\frac{\sigma^2}{2h^2(\frac{b}{d})}\frac{b^2}{d^2}-\frac{\alpha}{\varepsilon}<-1. \tag{3.18}$$

For convenience, we divide $\Gamma\setminus D_\varepsilon$ into five domains

$$\begin{aligned}
&D_1=\{(S,I,R)\in\Gamma:0<S<\varepsilon\},\quad B_2=\{(S,I,R)\in\Gamma:0<I<\varepsilon\},\\
&D_3=\{(S,I,R)\in\Gamma:\varepsilon\le I,0<R\le\varepsilon^2\},\\
&D_4=\{(S,I,R)\in\Gamma:\varepsilon\le I<\frac{b}{d},\frac{b}{d}-\varepsilon^2\le N<\frac{b}{d}\},\\
&D_5=\{(S,I,R,V)\in\Gamma:\varepsilon\le I<\frac{b}{d},\frac{b}{d+\alpha}-\varepsilon^2\le N<\frac{b}{d+\alpha}+\varepsilon^2\}.
\end{aligned}$$

Then, $\Gamma\setminus D_\varepsilon=D_1\cup D_2\cup D_3\cup D_4\cup D_5$.
Case 1. if $(S,I,R)\in D_1$, we have

$$\begin{aligned}
\mathcal{L}V&<-2+g(l_1\frac{\beta}{h(\frac{b}{d})}+4l_2\alpha h(\frac{b}{d}))I+gl_1\frac{\sigma^2}{2h^2(\frac{b}{d})}I^2-\frac{b}{S}\\
&<-2+g(l_1\frac{\beta}{h(\frac{b}{d})}+4l_2\alpha h(\frac{b}{d}))\frac{b}{d}+gl_1\frac{\sigma^2}{2h^2(\frac{b}{d})}\frac{b^2}{d^2}-\frac{b}{\varepsilon}\\
&<-1,
\end{aligned}$$

which follows from (3.15), we arrive at $\mathcal{L}V<-1$ for all $(S,I,R)\in D_1$.
Case 2. if $(S,I,R)\in D_2$, we get

$$\begin{aligned}
\mathcal{L}V&<-2+g(l_1\frac{\beta}{h(\frac{b}{d})}+4l_2\alpha h(\frac{b}{d}))I+gl_1\frac{\sigma^2}{2h^2(\frac{b}{d})}I^2\\
&<-2+g(l_1\frac{\beta}{h(\frac{b}{d})}+4l_2\alpha h(\frac{b}{d}))\varepsilon+gl_1\frac{\sigma^2}{2h^2(\frac{b}{d})}\varepsilon^2\\
&<-1,
\end{aligned}$$

which follows from (3.16), we have $\mathcal{L}V<-1$ for all $(S,I,R)\in D_2$.
Case 3. if $(S,I,R)\in D_3$, we obtain

$$\begin{aligned}
\mathcal{L}V&<-2+g(l_1\frac{\beta}{h(\frac{b}{d})}+4l_2\alpha h(\frac{b}{d}))I+gl_1\frac{\sigma^2}{2h^2(\frac{b}{d})}I^2-\frac{\mu I}{R}\\
&<-2+g(l_1\frac{\beta}{h(\frac{b}{d})}+4l_2\alpha h(\frac{b}{d}))\frac{b}{d}+gl_1\frac{\sigma^2}{2h^2(\frac{b}{d})}\frac{b^2}{d^2}-\frac{\mu}{\varepsilon}\\
&<-1,
\end{aligned}$$

which follows from (3.17), one can see that $\mathcal{L}V<-1$ for all $(S,I,R)\in D_3$.
Case 4. if $(S,I,R)\in D_4$, we gain

$$\mathcal{L}V<-2+g(l_1\frac{\beta}{h(\frac{b}{d})}+4l_2\alpha h(\frac{b}{d}))I+gl_1\frac{\sigma^2}{2h^2(\frac{b}{d})}I^2-\frac{\alpha I}{\frac{b}{d}-N}$$

$$
\begin{aligned}
&< -2 + g(l_1 \frac{\beta}{h(\frac{b}{d})} + 4l_2\alpha h(\frac{b}{d}))\frac{b}{d} + gl_1 \frac{\sigma^2}{2h^2(\frac{b}{d})}\frac{b^2}{d^2} - \frac{\alpha}{\varepsilon} \\
&< -1,
\end{aligned}
$$

which follows from (3.18), thus $\mathcal{L}V < -1$ for all $(S, I, R) \in D_4$.
Case 5. if $(S, I, R) \in D_5$, we obtain

$$
\begin{aligned}
\mathcal{L}V &< -2 + g(l_1 \frac{\beta}{h(\frac{b}{d})} + 4l_2\alpha h(\frac{b}{d}))I + gl_1 \frac{\sigma^2}{2h^2(\frac{b}{d})}I^2 - \frac{\alpha S}{N - \frac{b}{d+\alpha}} \\
&< -2 + g(l_1 \frac{\beta}{h(\frac{b}{d})} + 4l_2\alpha h(\frac{b}{d}))\frac{b}{d} + gl_1 \frac{\sigma^2}{2h^2(\frac{b}{d})}\frac{b^2}{d^2} - \frac{\alpha}{\varepsilon} \\
&< -1,
\end{aligned}
$$

which follows from (3.18), hence $\mathcal{L}V < -1$ for all $(S, I, R) \in D_5$.

From above, we can obtain that

$$
\mathcal{L}V(S, I, R) \leq -1 \quad \text{for all } (S, I, R) \in \Gamma \setminus D_\varepsilon.
$$

According to Lemma 3.1, we can obtain that the solution of model (1.3) is a stationary Markov process. This completes the proof. □

Remark 3.4. By comparing $R_0 = \frac{\beta b}{d(\mu+d+\alpha)h(\frac{b}{d})}$ and $R_0^s = \frac{\beta b}{d(\mu+d+\alpha+\frac{\sigma^2 b^2}{2h^2(\frac{b}{d})d^2})h(\frac{b}{d})}$, one can see that R_0^s is less than R_0, if $\sigma \neq 0$. It is shown that the results of the stochastic model (1.3) are better than those of the deterministic model (1.2). That's one of the important reasons why we incorporate stochastic perturbations in deterministic model.

4. Numerical simulations and conclusion

In this section, we shall adopt the Milstein's Higher Order Method mentioned in [14] to verify our theoretical results. The corresponding discretization equations are

$$
\begin{cases}
S_{k+1} = S_k + (b - \dfrac{\beta S_k I_k}{h(N_k)} - dS_k + \delta R_k)\Delta t - \dfrac{\sigma S_k I_k}{h(N_k)}\sqrt{\Delta t}\xi_k - \dfrac{\sigma^2 S_k I_k}{2h(N_k)}(\xi_k^2 - 1)\Delta t, \\
I_{k+1} = I_k + (\dfrac{\beta S_k I_k}{h(N_k)} - (\mu_k + \alpha_k + d_k)I_k)\Delta t + \dfrac{\sigma S_k I_k}{h(N_k)}\sqrt{\Delta t}\xi_k + \dfrac{\sigma^2 S_k I_k}{2h(N_k)}(\xi_k^2 - 1)\Delta t, \\
R_{k+1} = R_k + (\mu_k I_k - (d_k + \delta_k)R_k)\Delta t,
\end{cases}
\tag{4.1}
$$

where the time increment $\Delta t > 0$, $\xi_k (k = 1, 2, ..., n)$ are independent Gaussian random variables which follow the standard normal distribution $N(0, 1)$.

To verify our main theoretical results, we assume that the parameters are as follows

$$
b = 0.8, \beta = 0.8, d = 0.1, \delta = 0.3, \mu = 0.1, \alpha = 0.1, f = 0.5. \tag{4.2}
$$

First, we focus on the stationary distribution of stochastic model (1.3). The other parameters remain unchanged. Let $\sigma = 0.1$, we have $R_0^s \approx 1.328 > 1$, $R_0 \approx 1.333 > 1$. Thereupon, system (1.2) has a unique endemic equilibrium E^* which is locally asymptotically stable and system (1.3) has a stationary distribution

which implies that the disease $I(t)$ is stochastic persistent (see Figure 1). In addition, the system (1.3) exhibits oscillation around the endemic equilibrium of the corresponding deterministic system (see Figure 2).

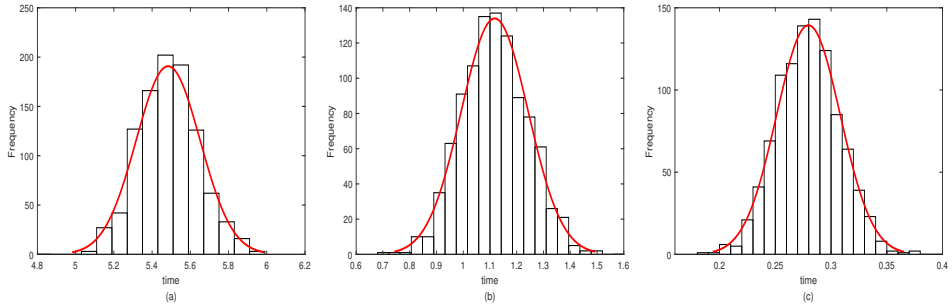

Figure 1. (a), (b) and (c) are frequency histograms of susceptible, infected and removed, respectively with $\sigma = 0.1$ and initial values $(S(0), I(0), R(0)) = (3.5, 2, 0.5)$.

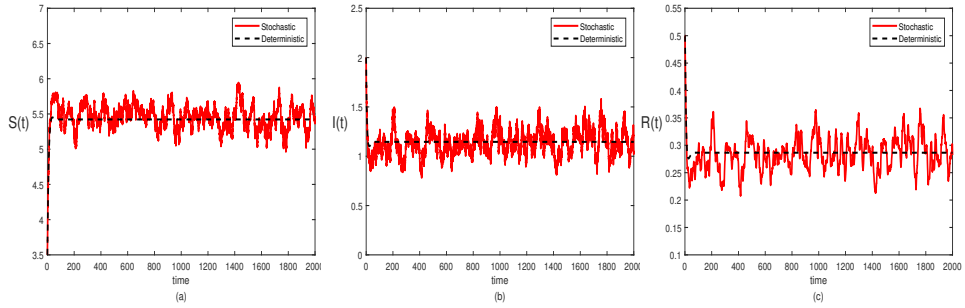

Figure 2. (a), (b) and (c) are the sample paths of susceptible, infected and removed, respectively with $\sigma = 0.1$ and initial values $(S(0), I(0), R(0)) = (3.5, 2, 0.5)$.

Then, we want to test the effect of white noise on individuals. Hence, we performs the following set of numerical simulations. In case of (4.2), we increase the noise intensity σ to 0.25. After computation of R_0 and R_0^s, we have $R_0 \approx 1.333 > 1$ and $R_0^s \approx 1.299 > 1$. Similarly, the solution of model (1.2) is locally asymptotically stable and model (1.3) has a stationary distribution (see Figure 3 and Figure 4). By comparing Figure 1 and Figure 3, one can see that the distribution of S, I and R becomes more widespread as σ increases. At the same time, from Figure 2 and Figure 4, we can also see that the solution of the model (1.3) oscillates over a larger range, if σ goes up.

Last but not the least, we want to test whether the conditions required by our stochastic model are actually weaker than those required by the deterministic model. To achieve this, we choose $\sigma = 0.9$. It is easy to get $\sigma^2 = 0.81 < \frac{d\beta h(\frac{b}{d})}{b} = 1.6$ and $R_0^s \approx 0.997 < 1 < R_0 = 1.333$. According to the theorem we proved above, we know that system (1.2) has a unique endemic equilibrium E^* which is locally asymptotically stable. Meanwhile, for stochastic model (1.3), one can see that the disease $I(t)$ is extinct, which shows that stochastic perturbations are beneficial to control outbreaks of disease (see Figure 5).

In the real world, population system is inevitably affected by various environ-

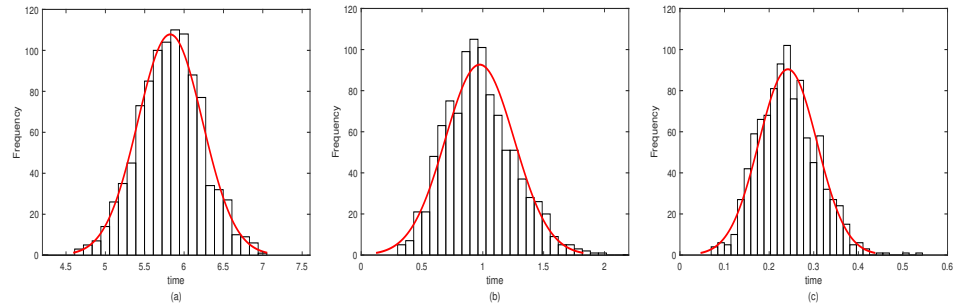

Figure 3. (a), (b) and (c) are frequency histograms of susceptible, infected and removed, respectively with $\sigma = 0.15$ and initial values $(S(0), I(0), R(0)) = (3.5, 2, 0.5)$.

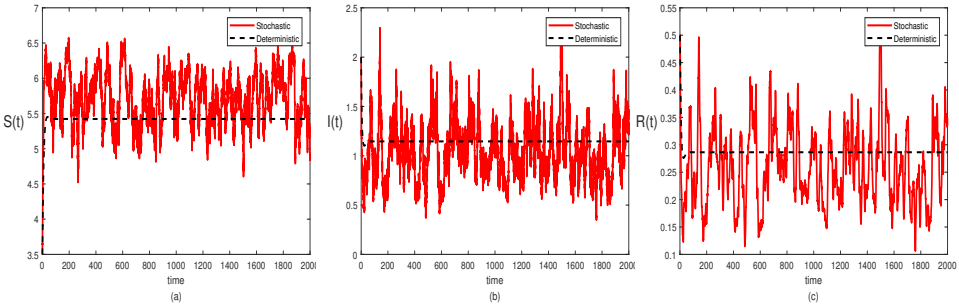

Figure 4. (a), (b) and (c) are the sample paths of susceptible, infected and removed, respectively with $\sigma = 0.15$ and initial values $(S(0), I(0), R(0)) = (3.5, 2, 0.5)$.

mental noises. Therefore, we consider the dynamics behavior of stochastic SIRS epidemic model. We assume that the stochastic perturbation is a white noise type which perturbs the disease transmission coefficient β. For deterministic system (1.2), we define a basic reproduction number $R_0 = \frac{\beta b}{d(\mu+d+\alpha)h(\frac{b}{d})}$, the disease-free equilibrium is globally asymptotically stable if $R_0 < 1$; while if $R_0 > 1$, system (1.2) admits a unique endemic equilibrium which is locally asymptotically stable. For stochastic system (1.3), we also obtain a basic reproduction number $R_0^s = \frac{\beta b}{d(\mu+d+\alpha+\frac{\sigma^2 b^2}{2h^2(\frac{b}{d})d^2})h(\frac{b}{d})}$. Epidemiologically, we draw the following conclusions:

Environmental noise can inhibit the outbreak of infectious diseases: From (ii) of theorem 3.2, one can see that if $R_0^s < 1$ plus the additional conditions, the disease will go to extinct with probability one; Theorem 2.1 indicates that the deterministic model (1.2) has a unique endemic equilibrium E^* which is locally asymptotically stable if the basic reproduction number $R_0 > 1$. Furthermore, from remark 3.4, we know $R_0^s < R_0$. If we choose suitable parameters such that $R_0^s < 1 < R_0$, then the deterministic model (1.2) has an endemic equilibrium while the stochastic model (1.3) has disease extinction with probability one (see Figure 5).

The extent of environmental noise impact: From (i) of theorem 3.2, If white noise is large enough, the disease will extinct exponentially. However, for smaller environmental noise conditions, additional condition $R_0^s < 1$ need to be added to ensure that the extinction of disease. In this case, the environmental noise is negligible. That is to say, when the environmental noise is large, the stochastic model

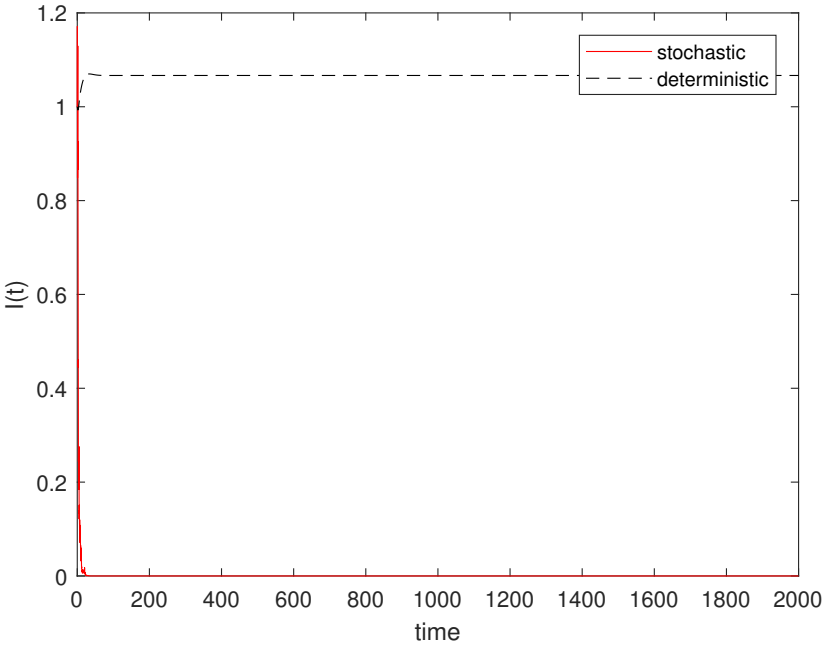

Figure 5. That is the sample paths of infected with $\sigma = 0.9$ and initial values $(S(0), I(0), R(0)) = (3.5, 2, 0.5)$.

is more suitable for describing epidemic models than the deterministic model. Theorem 2.1 reveals system (1.2) has a unique endemic equilibrium E^* which is locally asymptotically stable if $R_0 > 1$. Under this condition, when the noise intensity σ is small enough to imply that $R_0^s = R_0 - \frac{\sigma^2 b^3 \beta}{2h^3(\frac{b}{d})d^3(\mu+d+\alpha)(\mu+d+\alpha+\frac{\sigma^2 b^2}{2h^2(\frac{b}{d})d^2})} > 1$, from theorem 3.3, environmental noise forces the solution of model (1.3) to oscillate around the endemic equilibrium state (see Figure 2 and Figure 4). Furthermore, from Figure 2 and Figure 4, we can see that when the noise intensity σ is increased, the amplitude fluctuation of the solution of the model (1.3) becomes larger.

In addition, we want to refine our results further. From Theorems 3.2 and Theorems 3.3, we need to explore what happens if $\frac{d\beta h(\frac{b}{d})}{b} < \sigma^2 < \frac{\beta^2}{2(\mu+d+\alpha)}$ and $R_0^s =< 1$? Hence, we add an additional set of numerical simulations: $b = 0.8,\ \beta = 0.6,\ d = 0.01,\ \delta = 0.3,\ \mu = 0.08,\ \alpha = 0.1,\ f = 0.5,\ \sigma = 0.99$. We can directly calculate $\frac{d\beta h(\frac{b}{d})}{b} = 0.75 < \sigma^2 \approx 0.941 < \frac{\beta^2}{2(\mu+d+\alpha)} \approx 0.947$ and $R_0^s \approx 0.978 < 1$. From Figure 6, one can see that the infectious disease $I(t)$ go extinct exponentially almost surely. For system (1.2), we also can get $R_0 \approx 2.526 > 1$. Hence, system (1.2) has a unique endemic equilibrium E^* which implies that the infectious disease $I(t)$ is persistent.

From above, one can see that it is necessary to take environmental noise into account when studying the dynamics of infectious disease models. Obviously, environmental noise plays an important role in the spread and control of diseases, which provides us with some useful control strategies for controlling infectious diseases.

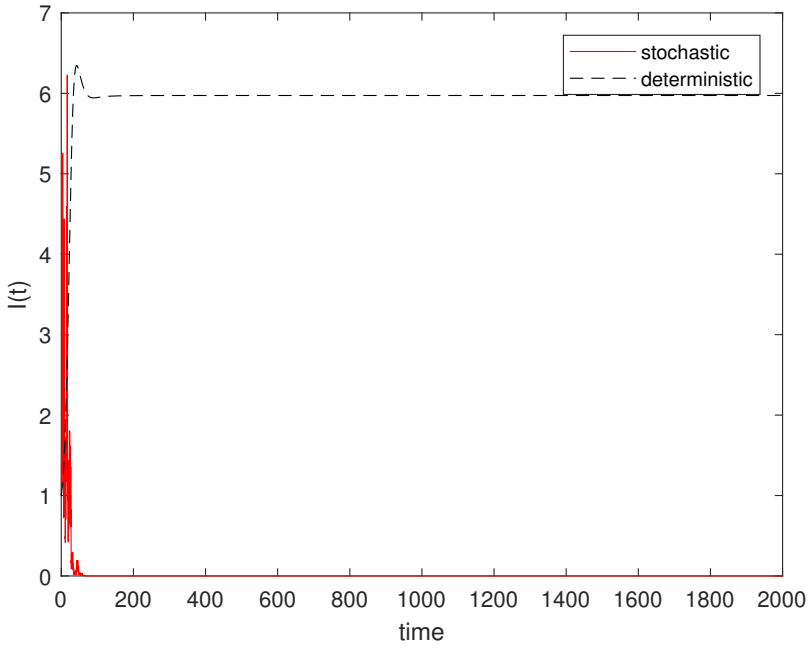

Figure 6. That is the sample paths of infected with $\sigma = 0.97$ and initial values $(S(0), I(0), R(0)) = (3.5, 2, 0.5)$.

And we know it is beneficial to the control of outbreaks of infectious disease.

There are some interesting topics to explore in the future, we know that there are many types of environment noise, in addition to white noise, we can also incorporate telegraph noise [6, 18]. Furthermore, we can consider more complex systems, such as considering delayed [34], impulsive [7] perturbations and so on. We leave that work for the future.

References

[1] Y. Cai and W. Wang, *Fish-hook bifurcation branch in a spatial heterogeneous epidemic model with cross-diffusion*, Nonlinear Anal. RWA., 2016, 30, 99–125.

[2] Y. Cai, Y. Kang and W. Wang, *A stochastic SIRS epidemic model with nonlinear incidence rate*, Appl. Math. Comput., 2017, 305, 221–240.

[3] S. Cai, Y. Cai and X. Mao, *A stochastic differential equation SIS epidemic model with two independent Brownian motion*, J. Math. Anal. Appl., 2019, 472, 1536–1550.

[4] B. Cao, M. Shan, Q. Zhang and W. Wang, *A stochastic SIS epidemic model with vaccination*, Physica A., 2017, 486, 127–143.

[5] Z. Chang, X. Meng and X. Lu, *Analysis of a novel stochastic SIRS epidemic model with two different saturated incidence rates*, Physical A., 2017, 472, 103–116.

[6] Z. Chen, R. Zhang, J. Li and S. Zhang, C. Wei, *A stochastic nutrient-phytoplankton model with viral infection and Markov switching*, Chaos Soliton Fract., 2020, 140, 110109.

[7] Y. Deng and M. liu, *Analysis of a stochastic tumor-immune model with regime switching and impulsive perturbations*, Appl. Math. Model., 2020, 78, 482–504.

[8] K. Fan, Y. Zhang, S. Gao and X. Wei, *A class of stochastic delayed SIR epidemic models with generalized nonlinear incidence rate and temporary immunity*, Physica A., 2017, 481, 198–208.

[9] T. Feng and Z. Qiu, *Global dynamics of deterministic and stochastic epidemic systems with nonmonotone incidence rate*, Int. J. Biomath., DOI: 10.1016/j.physa.2019.01.014.

[10] W. Guo, Y. Cai, Q. Zhang and W. Wang, *Stochastic persistence and stationary distribution in an SIS epidemic model with media coverage*, Physical A., 2018, 492, 2220–2236.

[11] R. Has'minskii, *Stochastic Stability of Differential Equations, Sijthoff and Noordhoff*, Alphen aan den Rijn, The Netherlands, 1980.

[12] J. Heesterbeek and J. Metz, *The saturating contact rate in marriage and epidemic models*, J. Math. Biol., 1993, 31, 529–539.

[13] H. W. Hethcote, *The mathematics of infectious diseases*, SIAM Rev., 2000, 42, 599–653.

[14] D. Higham, *Analgorithmic introduction to numerical simulation of stochastic differential equations*, SIAM review., 2001, 43, 525–546.

[15] Z. Hu, Z. Teng and H. Jiang, *Stability analysis in a class of discrete SIRS epidemic models*, Nonlinear. Anal-Real., 2012, 13, 2017–2033.

[16] G. Hu, M. Liu and K. Wang, *The asymptotic behaviours of an epidemic model with two correlated stochastic perturbations*, Appl. Math. Comput., 2012, 218, 10520–10532.

[17] S. Jeffrey and K. Melvin, *Absolute humidity modulates influenza survival, transmission, and seasonality*, Proc. Natl. Acad. Sci., USA, 2009, 106, 3243–3248.

[18] W. Ji, Z. Wang and G. Hu, *Stationary distribution of a stochastic hybrid phytoplankton model with allelopathy*, Adv. Differ. Equ-Ny., 2020, 2020(1), 632.

[19] G. Lan, Z. Chen, C. Wei and S. Zhang, *Stationary distribution of a stochastic SIQR epidemic model with saturated incidence and degenerate diffusion*, Physica A., 2018, 511, 61–77.

[20] A. Lahrouz, L. Omari and D. Kiouach, *Global analysis of a deterministic and stochastic nonlinear SIRS epidemic model*, Nonlinear Anal-Model., 2011, 16, 59–76.

[21] F. Li and X. Meng, *Analysis and numerical simulations of a stochastic SEIQR epidemic system with quarantine-adjusted incidence and imperfect vaccination*, Comput. Math. Method. M., 2018, DOI: 10.1155/2018/7873902.

[22] Z. Li, Y. Mu and H. Xiang, *Mean persistence and extinction for a novel stochastic turbidostat model*, Nonlinear Dynam., 2019, 97(1), 185–202.

[23] Q. Liu and Q. Chen, *Analysis of the deterministic and stochastic SIRS epidemic models with nonlinear incidence*, Physica A., 2015, 428, 140–153.

[24] J. Liu and F. Wei, *Dynamics of stochastic SEIS epidemic model with varying population size*, Physica A., 2016, 464, 241–250.

[25] Q. Liu, D. Jiang, N. Shi and T. Hayat, *Stationary distribution and extinction of a stochastic SEIR epidemic model with standard incidence*, Physica A., 2017, 476, 58–69.

[26] Q. Liu, D. Jiang, T. Hayat and A. Alsaedi, *Stationary distribution and extinction of a stochastic dengue epidemic model*, J. Franklin. I., 2018, 355, 8891–8914.

[27] Q. Liu, D. Jiang, T. Hayat and A. Alsaedi, *Dynamics of a stochastic multigroup SIQR epidemic model with standard incidence rates*, J. Franklin. I., 2019, 356, 2960–2993.

[28] S. Liu, L. Zhang, X. Zhang and A. li, *Dynamics of a stochastic heroin epidemic model with bilinear incidence and varying population size*, Int. J. Biomath., 2019, 12(1), 1950005.

[29] M. Lu, J. Huang, S. Ruan and P. Yu, *Bifurcation analysis of an SIRS epidemic model with a generalized nonmonotone and saturated incidence rate*, J. Differ. Equations., 2019, 267, 1859–1898.

[30] Z. Ma and J. Li, *Dynamical modeling and analysis of epidemics*, International Association of Geodesy Symposia, 2009.

[31] X. Mao, *Stochastic Differential Equations and Applications*, Horwood, New York, 1997.

[32] P. Mead, *Epidemics of plague past, present, and future*, Lancet Infect. Dis., 2019, 19, 459–460.

[33] X. Meng, S. Zhao, T. Feng and T. Zhang, *Dynamics of a novel nonlinear stochastic SIS epidemic model with double epidemic hypothesis*, J. Math. Anal. Appl., 2016, 433, 227–242.

[34] E. Mohamed, P. Roger, S. Idriss, S. Idriss and T. Regragui, *A stochastic analysis for a triple delayed SIQR epidemic model with vaccination and elimination strategies*, J. Appl. Math. Comput., 2020, 64, 781–805.

[35] H. Qi, L. Liu and X. Meng, *Dynamics of a Nonautonomous Stochastic SIS Epidemic Model with Double Epidemic Hypothesis*, Complexity, 2017, DOI: 10.1155/2017/4861391.

[36] H. Qi, S. Zhang and X. Meng, *Periodic solution and ergodic stationary distribution of two stochastic SIQS epidemic systems*, Physica A., 2018, 508, 223–241.

[37] R. Shi, X. Jiang and L. Chen, *The effect of impulsive vaccination on an SIR epidemic model*, Appl. Math. Comput., 2009, 212, 305–311.

[38] W. Wang, Y. Cai, Z. Ding and Z. Gui, *A stochastic differential equation SIS epidemic model incorporating Ornstein-Uhlenbeck process*, Physica A., 2018, 509, 921–936.

[39] C. Wei and L. Chen, *A delayed epidemic model with pulse vaccination*, Discrete Dyn. Nat. Soc., DOI: 10.1155/2008/746951.

[40] Y. Zhao and D. Jiang, *The threshold of a stochastic SIS epidemic model with vaccination*, Appl. Math. Comput., 2014, 243, 718–727.

[41] Y. Zhou, W. Zhang and S. Yuan, *Survival and stationary distribution of a SIR epidemic model with stochastic perturbations*, Appl. Math. Comput., 2014, 244, 118–131.

[42] M. Zhou, H. Xiang and Z. Li, *Optimal control strategies for a reaction-diffusion epidemic system*, Nonlinear Anal-real., 2019, 46(2019), 446–464.

[43] G. Zhu, G. Chen, H. Zhang and X. Fu, *Propagation dynamics of an epidemic model with infective media connecting two separated networks of populations*, Commun. Nonlinear Sci., 2015, 20, 240–249.