Mathematical Biosciences and Engineering

http://www.aimspress.com/journal/mbe

MBE, 19(10): 10618–10636.
DOI: 10.3934/mbe.2022496
Received: 28 May 2022
Revised: 19 July 2022
Accepted: 20 July 2022
Published: 26 July 2022

Research article

Dynamics of a stochastic SIRS epidemic model with standard incidence and vaccination

Tingting Xue[1,*], Xiaolin Fan[1] and Zhiguo Chang[2]

[1] School of Mathematics and Physics, Xinjiang Institute of Engineering, Urumqi, Xinjiang, China
[2] School of Safety Science, Xinjiang Institute of Engineering, Urumqi, Xinjiang, China

* **Correspondence:** Email: xuett@cumt.edu.cn.

Abstract: A stochastic SIRS epidemic model with vaccination is discussed. A new stochastic threshold R_0^s is determined. When the noise is very low ($R_0^s < 1$), the disease becomes extinct, and if $R_0^s > 1$, the disease persists. Furthermore, we show that the solution of the stochastic model oscillates around the endemic equilibrium point and the intensity of the fluctuation is proportional to the intensity of the white noise. Computer simulations are used to support our findings.

Keywords: epidemic model; stochastic; Brownian motion; extinction; persistence; numerical simulation

1. Introduction

An infectious disease is a disease that can spread widely from person to person or from person to animal, passing through various channels to another person or species [1]. The disease can usually be transmitted through direct contact with infected individuals, bodily fluids and faeces of infected persons, objects contaminated by infected persons, through air, water, food, contact, soil, vertical transmission (mother-to-child transmission), bodily fluids, fecal-oral transmission, etc [2–4]. With the high integration of the global economy and the increasingly frequent exchanges between countries, international tourism creates convenient conditions for the spread and prevalence of infectious diseases in the world [5, 6]. Whenever an infectious disease breaks out in any part of the world today, it can quickly spread to neighboring areas or spread to other parts of the world [7]. At the same time, due to the continuous deterioration of the environment and the abuse of antibiotics, the drug resistance and pathogenicity of some viruses are increasing, so that some patients with infectious diseases are re-infected due to the re-activation of pathogens after recovery, leading to the recurrence of the disease. These phenomena make it more and more difficult to control and prevent diseases. In recent years, many mathematicians have devoted themselves to the study of infectious diseases [8–10]. Accurate

research on the mechanism of disease outbreak, spread and epidemic, and targeted prevention and control measures can effectively reduce the harm caused by the disease.

The dynamic model of infectious disease, which divides the total population into different compartments according to different stages and states, is an important tool to study the spread and transmission mechanism of epidemic diseases. Kermack and McKendrick put forward the famous SIR model [11] and SIS model [12] in 1927 and 1932 respectively, and obtained the threshold theory for determining the prevalence of diseases. The basic regeneration number is used as the threshold to judge the extinction and persistence of infectious diseases, which lays a foundation for studying the dynamics of infectious diseases. Since then, many scholars have established various infectious disease models (such as SIS [13, 14], SIR [15, 16], SIRS [17, 18], SEIR [19, 20], SIQ [21], SIQS [22], etc.) to study the spread of the disease and put forward methods to control the disease [23–25]. Vaccination has become an important and commonly used strategy to eliminate infectious diseases, which can effectively reduce the infection of infectious diseases. Jin et al. [26] studied the following SIRS epidemic model with vaccination

$$\begin{cases} \dfrac{dS}{dt} = bN - \lambda\dfrac{SI}{N} + \theta I + eR - (\mu + p)S, \\ \dfrac{dI}{dt} = \lambda\dfrac{SI}{N} - (\mu + \varepsilon + c + \theta)I, \\ \dfrac{dR}{dt} = cI + pS - (\mu + e)R, \end{cases} \tag{1.1}$$

with $S(0) = S_0 \geq 0, I(0) = I_0 \geq 0, R(0) = R_0 \geq 0$, where S, I and R respectively represent the number of susceptible, infected and removed individuals at time t, $S + I + R = N$, represents the number of total population at time t, b is the natural birth rate, λ is the transmission rate of the disease, θ is the transfer rate from I to S, c represents the treatment rate, e represents the loss of immunity rate, μ represents the natural death rate, ε represents the disease-related death rate, p represents vaccination rate. From [26], the basic regeneration number of model (1.1) is as follows:

$$\mathrm{R}_0 = \frac{\lambda}{b + \varepsilon + c + \theta} \times \frac{b + e}{b + e + p}. \tag{1.2}$$

Model (1.1) has the following dynamical properties:

(i) If $\mathrm{R}_0 < 1$, then $E^0\left(\frac{b+e}{b+e+p}, 0, \frac{p}{b+e+p}\right)$ is globally asymptotically stable.

(ii) If $\mathrm{R}_0 > 1$, then E^0 is unstable and model (1.1) has a unique endemic equilibrium point E^* which is globally asymptotically stable.

However, the use of deterministic methods to study the transmission of infectious diseases has certain limitations. The spread of diseases is not only affected by biological properties such as virulence and drug resistance, social factors such as people's prevention and control strategies and media reports, but also by random factors such as environmental noise. In the real world, infectious disease models are always subjected to random interference from the external environment, which makes the parameters in the model (such as exposure rate, mortality rate, recovery rate, etc.) show random fluctuations. In addition, in the early stage of the spread of an infectious disease or when it is about to disappear, the number of infected people is small and the number of individuals involved is not very large, which leads to a large impact of random interference on the spread of the disease. At this time, it is not always

ideal to use deterministic infectious disease model to describe and predict the development process and infectious law of the disease, so it has more practical significance to study the dynamic properties of infectious disease model under the influence of random factors. In discussing the influence of environmental noise on infectious disease system, three kinds of noise are mainly studied: white noise, telegraph noise and Lévy noise. White noise refers to some subtle disturbances in the environment, such as changes in air humidity and temperature, which can be described by the formal derivative of Brown motion. At present, the most common method to introduce white noise is to perturb the parameters in the model. For example, Gray et al. [27] assumed that some random environmetnal factor is acting simultaneously on each individual in the population. In this case, a random ambient white noise perturbation of incidence causes β to change as a random variable $\tilde{\beta}$, that is, $\tilde{\beta}dt = \beta dt + \sigma dB(t)$. They established the following SIS epidemic model with environmental random disturbance

$$\begin{cases} dS = [\mu N - \beta S I + \gamma I - \mu S]\, dt - \sigma S I dB(t), \\ dI = [\beta S I - (\mu + \gamma) I]\, dt + \sigma S I dB(t), \end{cases}$$

where $\sigma^2 > 0$ is white noise intensity, $B(t)$ is a standard Brownian motion. The authors then proved that the model has a unique global positive solution and established conditions for the extinction and persistence of the disease. They found that the amount of white noise had a significant effect on the presence and extinction of the disease. The authors [28] studied the dynamics of a stochastic SIRS infectious disease model with saturated incidence. When the noise was low, the authors obtained a threshold for the stochastic system that determines the extinction and persistence of infectious diseases. They found that loud noise suppressed the spread of the disease. Article [29] studied a periodic stochastic SIVS epidemic model with nonlinear incidence and vaccination. By constructing a new random Lyapunov function and using a new technique, the threshold conditions for the existence of the random positive periodic solution and the disappearance of the disease were established. The authors [30] studied the asymptotic properties of a class of stochastic delayed SIR infectious disease models with temporary immunity. Sufficient conditions were established for the extinction and persistence of epidemics. A threshold between the persistence and extinction of the epidemic was obtained. Compared with the deterministic model, the influence threshold of white noise was smaller than that of deterministic system. The authors [31] analyzed a time-delay SIQR infectious disease model with mixed inoculation and elimination strategies under white noise disturbance. The existence and uniqueness of positive solutions were proved. A random threshold was established to study the extinction and persistence of random infectious diseases. Then the existence of stationary distribution of stochastic model with time delay was studied.

In this paper, inspired by the above literature, random white noise disturbance is introduced to establish a deterministic SIRS model (1.1) corresponding to SIRS infectious disease model with random disturbance, the specific form is as follows:

$$\begin{cases} dS = [bN - \lambda \dfrac{SI}{N} + \theta I + eR - (\mu + p) S]dt - \sigma \dfrac{SI}{N} dB(t), \\ dI = [\lambda \dfrac{SI}{N} - (\mu + \varepsilon + c + \theta) I]dt + \sigma \dfrac{SI}{N} dB(t), \\ dR = [cI + pS - (\mu + e) R]dt. \end{cases} \tag{1.3}$$

According to (1.3), the equation of the total population N is as follows:

$$dN = \left[(b-\mu)N - \varepsilon I\right]dt. \tag{1.4}$$

Let $x = \frac{S}{N}$, $y = \frac{I}{N}$, $z = \frac{R}{N}$, then model (1.3) becomes the following system

$$\begin{cases} dx = \left[b - (b+p)x + ez + \theta y - (\lambda - \varepsilon)xy\right]dt - \sigma xy dB(t), \\ dy = \left[-(b+\varepsilon+c+\theta)y + \lambda xy + \varepsilon y^2\right]dt + \sigma xy dB(t), \\ dz = \left[-(b+e)z + cy + px + \varepsilon yz\right]dt. \end{cases} \tag{1.5}$$

On account of the relation $x + y + z = 1$, we can simplify the model (1.5) to discuss the following problem

$$\begin{cases} dy = \left[-(b+\varepsilon+c+\theta-\lambda)y - \lambda zy + (\varepsilon-\lambda)y^2\right]dt + \sigma(1-y-z)y dB(t), \\ dz = \left[p - (b+e+p)z + (c-p)y + \varepsilon yz\right]dt, \end{cases} \tag{1.6}$$

with $(y(0), z(0)) \in \mathbb{R}^2_+$ and $y(0) + z(0) < 1$.

The specific research work of this paper is as follows: Section 2 proves that model (1.5) has a unique global positive solution. Section 3 deduces the conditions leading to death from disease. Section 4 gives the conditions for the persistence of the disease. In Section 5, we deduce that the solution of model (1.6) vibrates around the endemic equilibrium point and that the intensity of the fluctuation is proportional to the magnitude of the white noise. The key to solve this problem is to choose an appropriate Lyapunov function. Section 6 gives a brief conclusion. This paper verifies the rationality of relevant theorems through several examples. In addition, numerical simulations are used to support our results.

2. Existence and uniqueness of positive solution

Here we give the existence and uniqueness theorem of global positive solution for model (1.5). Feasibility region

$$\Gamma^* = \{(x, y, z) : x > 0, y > 0, z > 0, x + y + z = 1\}$$

is the positive invariant set of model (1.5) with probability one.

Theorem 1. *For any given* $(x(0), y(0), z(0)) \in \Gamma^*$, *model (1.5) has a unique positive solution* $(x(t), y(t), z(t))$ $(\forall t \geq 0)$. *The solution is still in* Γ^* *with probability one, i.e.,* $(x(t), y(t), z(t)) \in \Gamma^*$ $(\forall t \geq 0)$ *almost surely (briefly a.s.).*

Proof Obviously, model (1.5) satisfies the local Lipschitz condition. Therefore, for any given $(x(0), y(0), z(0)) \in \mathbb{R}^3_+$, model (1.5) has a unique local solution $(x(t), y(t), z(t))$ on $t \in [0, \tau_e)$, where τ_e is the explosion time. The following proves that $\tau_e = \infty$ a.s. Let $k_0 > 1$ be large enough so that $(x(0), y(0), z(0)) \in \left[\frac{1}{k_0}, 1\right]^3$. For each integer $k \geq k_0$, the stopping time is defined by

$$\tau_k = \inf\left\{t \in [0, \tau_e) : \min\{x(t), y(t), z(t)\} \leq \frac{1}{k}\right\},$$

where $\inf \phi = \infty$. By the above definition, τ_k is increasing as $k \to \infty$. Let $\tau_\infty = \lim_{k\to\infty} \tau_k$, thus $\tau_\infty \le \tau_e$ a.s. If it can be proved that $\tau_\infty = \infty$ a.s., then $\tau_e = \infty$ a.s. The following proves that $\tau_\infty = \infty$ a.s. By contradiction, there exist $T > 0$, $\epsilon \in (0, 1)$, making $P\{\tau_\infty \le T\} > \epsilon$. Hence, there is an integer $k_1 \ge k_0$ such that

$$P\{\tau_k \le T\} \ge \epsilon, \ \forall k \ge k_1. \tag{2.1}$$

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

$$V(x, y, z) = \ln xyz.$$

Apply Itô formula [32] to obtain the following formula

$$dV(x, y, z) = LVdt + \sigma(x - y)\, dB(t), \tag{2.2}$$

where

$$\begin{aligned} LV =& \frac{1}{x}\left[b - (b + p)\, x + ez + \theta y - (\lambda - \varepsilon)\, xy\right] \\ &+ \frac{1}{y}\left[-(b + \varepsilon + c + \theta)\, y + \lambda xy + \varepsilon y^2\right] \\ &+ \frac{1}{z}\left[-(b + e)\, z + cy + px + \varepsilon yz\right] - \frac{1}{2}\sigma^2\left(x^2 + y^2\right) \\ \ge& -(3b + p + e + \varepsilon + c + \theta) + (3\varepsilon - \lambda)\, y + \lambda x - \frac{1}{2}\sigma^2\left(x^2 + y^2\right) \\ \triangleq& h(x, y, z). \end{aligned}$$

Because h is continuous and $x + y + z = 1$, there exists $H < 0$, so that $h(x, y, z) \ge H$ for $(x, y, z) \in \Gamma^*$. So, for $\forall k \ge k_1$, we get

$$\begin{aligned} &EV(x(\tau_k \wedge T), y(\tau_k \wedge T), z(\tau_k \wedge T)) - V(x(0), y(0), z(0)) \\ &\ge E\int_0^{\tau_k \wedge T} LV(x(s), y(s), z(s))ds \ge HT > -\infty. \end{aligned} \tag{2.3}$$

Let $\Omega_k = \{\tau_k \le T\}$ for $k \ge k_1$. Set I_{Ω_k} be an index function of Ω_k, then $P(\Omega_k) \ge \epsilon$. On the other side, we have

$$\begin{aligned} &EV(x(\tau_k \wedge T), y(\tau_k \wedge T), z(\tau_k \wedge T)) \\ \le& E[\ln x(\tau_k \wedge T)] \le E[I \ln x(\tau_k, \omega)] \le \epsilon \ln \frac{1}{k}. \end{aligned} \tag{2.4}$$

Let $k \to \infty$, (2.3) and (2.4) produce a contradiction $-\infty > HT > -\infty$. Hence, $\tau_\infty = \infty$ a.s.

3. Extinction of the disease

Here we present sufficient conditions for disease extinction in model (1.6).

Theorem 2. *Let* $(y(t), z(t))$ *be the solution of model (1.6) with* $(y(0), z(0)) \in \mathbb{R}^2_+$ *and* $y(0) + z(0) < 1$.

(i) If $\sigma^2 > \max\left\{\frac{(\lambda-\varepsilon)^2}{2(b+c+\theta)}, \lambda - \varepsilon\right\}$, *then*

$$\limsup_{t\to\infty} \frac{\ln y(t)}{t} \le -(b + c + \theta) + \frac{(\lambda - \varepsilon)^2}{2\sigma^2} < 0 \quad \text{a.s.} \tag{3.1}$$

(ii) If $\lambda - \varepsilon < 0$*, then*

$$\limsup_{t\to\infty} \frac{\ln y(t)}{t} \le -(b + c + \theta) < 0 \quad \text{a.s.} \tag{3.2}$$

(iii) If $R_0^s < 1$ *and* $\frac{2(b+c+\theta+\varepsilon)p}{b+e} < \sigma^2 \le \lambda - \varepsilon$*, then*

$$\limsup_{t\to\infty} \frac{\ln y(t)}{t} \le \frac{(b + c + \theta + \varepsilon)(b + e + p)}{b + e}(R_0^s - 1) < 0 \quad \text{a.s.} \tag{3.3}$$

where

$$R_0^s = \frac{\left(2\lambda - \sigma^2\right)(b + e) + 2(b + c + \theta + \varepsilon)p}{2(b + c + \theta + \varepsilon)(b + e + p)} = \mathrm{R}_0 - \frac{\sigma^2(b + e) - 2(b + c + \theta + \varepsilon)p}{2(b + c + \theta + \varepsilon)(b + e + p)}. \tag{3.4}$$

$$\mathrm{R}_0 = \frac{\lambda}{b + \varepsilon + c + \theta} \times \frac{b + e}{b + e + p}.$$

That is, $y(t)$ *tends to zero exponentially* a.s.*, i.e., the probability of extinction is one.*

Proof Define a C-function $V : \mathbb{R}_+ \to \mathbb{R}$ by $V = \ln y$. Apply Itô formula [32] to obtain the following formula

$$\begin{aligned}
dV =& d(\ln y) = \frac{\partial V}{\partial y}dy + \frac{1}{2}\frac{\partial^2 V}{\partial y^2}dydy \\
=& \frac{1}{y}\left\{\left[-(b + \varepsilon + c + \theta - \lambda)y - \lambda zy + (\varepsilon - \lambda)y^2\right]dt\right. \\
&\left. + \sigma(1 - y - z)ydB(t)\right\} + \frac{1}{2}\left(-\frac{1}{y^2}\right)\sigma^2(1 - y - z)^2y^2dt \\
=& \left[-(b + \varepsilon + c + \theta) + \lambda(1 - y - z) + \varepsilon y\right. \\
&\left. - \frac{1}{2}\sigma^2(1 - y - z)^2\right]dt + \sigma(1 - y - z)dB(t) \\
=& \left[-(b + \varepsilon + c + \theta) + (\lambda - \varepsilon)(1 - y - z) + \varepsilon(1 - z)\right. \\
&\left. - \frac{1}{2}\sigma^2(1 - y - z)^2\right]dt + \sigma(1 - y - z)dB(t) \\
=& \left[-(b + c + \theta) + (\lambda - \varepsilon)(1 - y - z) - \varepsilon z\right. \\
&\left. - \frac{1}{2}\sigma^2(1 - y - z)^2\right]dt + \sigma(1 - y - z)dB(t) \\
\le& \left[-(b + c + \theta) + (\lambda - \varepsilon)(1 - y - z)\right. \\
&\left. - \frac{1}{2}\sigma^2(1 - y - z)^2\right]dt + \sigma(1 - y - z)dB(t) \\
=& \left[-(b + c + \theta) + (\lambda - \varepsilon)x - \frac{1}{2}\sigma^2x^2\right]dt + \sigma(1 - y - z)dB(t) \\
=& f(x)dt + \sigma(1 - y - z)dB(t),
\end{aligned} \tag{3.5}$$

where $f:(0,1)\to\mathbb{R}$ is defined as follows:

$$\begin{aligned} f(x) &= -(b+c+\theta)+(\lambda-\varepsilon)x-\frac{1}{2}\sigma^2x^2 \\ &= -\frac{1}{2}\sigma^2\left(x-\frac{\lambda-\varepsilon}{\sigma^2}\right)^2-(b+c+\theta)+\frac{(\lambda-\varepsilon)^2}{2\sigma^2}, \end{aligned} \tag{3.6}$$

where $x=1-y-z\in(0,1)$. Integrating this from 0 to t and dividing by t on (3.5) of both sides, we have

$$\frac{\ln y(t)}{t}\le\frac{\ln y(0)}{t}+\frac{1}{t}\int_0^t f(x(s))\,ds+\frac{1}{t}\int_0^t\sigma(1-y(s)-z(s))\,dB(s). \tag{3.7}$$

Case 1.

$$\begin{aligned} f(x) &= -\frac{1}{2}\sigma^2\left(x-\frac{\lambda-\varepsilon}{\sigma^2}\right)^2-(b+c+\theta)+\frac{(\lambda-\varepsilon)^2}{2\sigma^2} \\ &\le -(b+c+\theta)+\frac{(\lambda-\varepsilon)^2}{2\sigma^2}, \end{aligned} \tag{3.8}$$

which is negative by the condition $\sigma^2>\max\left\{\frac{(\lambda-\varepsilon)^2}{2(b+c+\theta)},\lambda-\varepsilon\right\}$. By (3.7), (3.8), we have

$$\frac{\ln y(t)}{t}\le\frac{\ln y(0)}{t}+\left[-(b+c+\theta)+\frac{(\lambda-\varepsilon)^2}{2\sigma^2}\right]+\frac{M_1(t)}{t}, \tag{3.9}$$

where $M_1(t):=\sigma\int_0^t(1-y-z)\,dB(s)$. According to Martingale's large number theorem,

$$\lim_{t\to\infty}\frac{M_1(t)}{t}=0\quad\text{a.s.} \tag{3.10}$$

Taking the superior limit on both sides of (3.9), we can get

$$\limsup_{t\to\infty}\frac{\ln y(t)}{t}\le-(b+c+\theta)+\frac{(\lambda-\varepsilon)^2}{2\sigma^2}<0\quad\text{a.s.} \tag{3.11}$$

Case 2. If $\lambda-\varepsilon<0$, according to (3.6), one has $f(x)\le f(0)=-(b+c+\theta)$. By (3.7), we have

$$\frac{\ln y(t)}{t}\le\frac{\ln y(0)}{t}-(b+c+\theta)+\frac{M_1(t)}{t}.$$

Similar to Case 1, we can get

$$\limsup_{t\to\infty}\frac{\ln y(t)}{t}\le-(b+c+\theta)<0\quad\text{a.s.} \tag{3.12}$$

Case 3. From (3.8) and $\sigma^2\le\lambda-\varepsilon$, we get $x^*=\frac{\lambda-\varepsilon}{\sigma^2}\ge1$. Then by taking the maximum value of $f(x)$, we can get

$$\begin{aligned} f_M &= f(1)=\lambda-(b+c+\theta+\varepsilon)-\frac{1}{2}\sigma^2 \\ &= \frac{(b+c+\theta+\varepsilon)(b+e+p)}{b+e}(R_0^s-1). \end{aligned} \tag{3.13}$$

By (3.7), (3.13), one has

$$\frac{\ln y(t)}{t} \leq \frac{\ln y(0)}{t} + \frac{(b+c+\theta+\varepsilon)(b+e+p)}{b+e}(R_0^s - 1) + \frac{M_1(t)}{t}.$$

If $R_0^s < 1$, then

$$\limsup_{t\to\infty} \frac{\ln y(t)}{t} \leq \frac{(b+c+\theta+\varepsilon)(b+e+p)}{b+e}(R_0^s - 1) < 0 \quad \text{a.s.} \tag{3.14}$$

Cases 1–3, Eqs (3.11), (3.12) and (3.14) mean $\lim_{t\to\infty} y(t) = 0$ a.s.

It is useful to observe that in the classical deterministic model (when $\sigma = 0$ in model (1.6)), $y(t)$ tends to 0 if and only if $\mathrm{R}_0 < 1$, while in the stochastic model (1.6), $y(t)$ tends to 0 if and only if $R_0^s = \mathrm{R}_0 - \frac{\sigma^2(b+e)-2(b+c+\theta+\varepsilon)p}{2(b+c+\theta+\varepsilon)(b+e+p)} < 1$ and $\frac{2(b+c+\theta+\varepsilon)p}{b+e} < \sigma^2 \leq \lambda - \varepsilon$. In other words, the conditions for $y(t)$ to become extinct in the stochastic model are weaker than in the classical deterministic model. The following example illustrates this result more explicitly.

Example 1. This paper assumes that the time unit is one day and the population size is one million. For model (1.6), the parameters are selected as follows:

$$b = 0.2,\ \lambda = 0.6,\ \varepsilon = 0.1,\ e = 0.14,\ p = 0.06,\ c = 0.15,\ \theta = 0.05,\ \sigma = 0.65.$$

After calculation, we have $R_0^s = 0.811 < 1$ and

$$0.17647 = \frac{2(b+c+\theta+\varepsilon)p}{b+e} < \sigma^2 = 0.4225 \leq \lambda - \varepsilon = 0.5.$$

According to Case (iii) of Theorem 2, the solution $(y(t), z(t))$ of (1.6) satisfies the following inequality:

$$\limsup_{t\to\infty} \frac{\ln y(t)}{t} \leq \frac{(b+c+\theta+\varepsilon)(b+e+p)}{b+e}(R_0^s - 1) \doteq -0.11125 < 0 \quad \text{a.s.}$$

That is, $y(t)$ will tend to zero exponentially with probability one. This shows that the disease is extinct.

For the corresponding deterministic model, one has $\mathrm{R}_0 = 1.02 > 1$. So (y^*, z^*) is globally asymptotically stable in $\Gamma_0 = \Gamma - \left\{\left(\frac{b+e}{b+e+p}, 0, \frac{p}{b+e+p}\right)\right\}$, where $\Gamma = \{x \geq 0, y \geq 0, z \geq 0, x+y+z = 1\}$, and the disease persists. Using the Euler-Maruyama (EM) method [33], we give the simulations shown in Figure 1 to support our results.

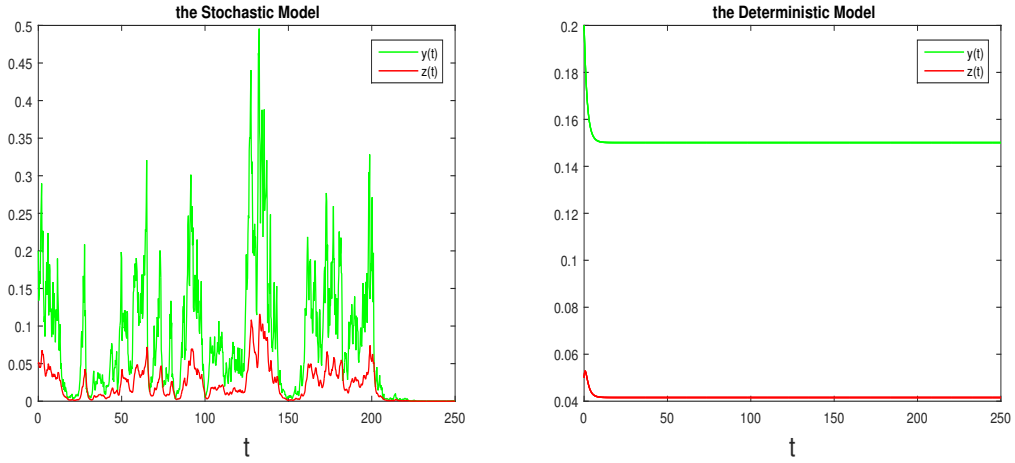

Figure 1. Computer simulation of the path $y(t)$, $z(t)$ for model (1.6) and its corresponding deterministic model, using the Euler-Maruyama (EM) method [33] with step size $\Delta t = 0.25$ and initial value $y(0) = 0.2$, $z(0) = 0.05$.

Example 2. The values of the parameters except $\sigma = 0.8$ are the same as those in Example 1. It is easy to verify that the system parameters obey the condition of Case (i) of Theorem 2, as

$$\sigma^2 = 0.64 > \max\left\{\frac{(\lambda-\varepsilon)^2}{2(b+c+\theta)}, \lambda-\varepsilon\right\} = 0.5.$$

So, by Case (i) of Theorem 2, we have

$$\limsup_{t\to\infty} \frac{\ln y(t)}{t} \le -(b+c+\theta) + \frac{(\lambda-\varepsilon)^2}{2\sigma^2} \doteq -0.2047 < 0 \quad \text{a.s.}$$

That is, $y(t)$ will tend to zero exponentially with probability one. The computer simulation shown in Figure 2 clearly supports this result, showing the extinction of the disease.

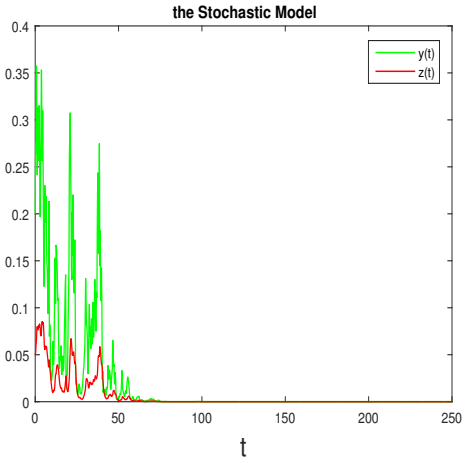

Figure 2. Computer simulation of the path $y(t)$, $z(t)$ for model (6), using the EM method with step size $\Delta t = 0.25$ and initial value $y(0) = 0.2$, $z(0) = 0.05$.

4. Persistence in the mean of the disease

Definition 1. *If the following conditions are true*

$$\liminf_{t\to\infty} \frac{\int_0^t y(s)\,ds}{t} > 0, \quad \liminf_{t\to\infty} \frac{\int_0^t z(s)\,ds}{t} > 0 \quad \text{a.s.}$$

Then model (1.6) is persistent.

For convenience, we define $\langle x(t)\rangle$ as follows:

$$\langle x(t)\rangle = \frac{\int_0^t x(s)\,ds}{t}.$$

Theorem 3. *For* $\forall (y(0), z(0)) \in \mathbb{R}^2_+$, $y(0) + z(0) < 1$, *if* $R_0^s > 1$, *then the solution* $(y(t), z(t))$ *of model (1.6) satisfies*

$$\liminf_{t\to\infty} \langle y(t)\rangle \geq y^*, \quad \liminf_{t\to\infty} \langle z(t)\rangle \geq \frac{c-p}{b+e+p} y^* + \frac{p}{b+e+p} \quad \text{a.s.}$$

where

$$y^* = \frac{b+e+p+c+\varepsilon}{2\varepsilon} + \frac{-p\lambda - (b+\theta)\varepsilon - (b+e+p)\sqrt{\Delta'}}{2\varepsilon(\lambda-\varepsilon)} < 1$$

and

$$\begin{aligned}\Delta' =& \left\{(\lambda-\varepsilon) + \frac{(c-p)\lambda}{b+e+p} + \frac{\varepsilon[\lambda-(b+\varepsilon+c+\theta)]}{b+e+p}\right\}^2 \\ & - \frac{4\varepsilon(\lambda-\varepsilon)}{b+e+p}\left[\lambda - \left(b+\varepsilon+c+\theta+\frac{\sigma^2}{2}+\frac{\lambda p}{b+e+p}\right)\right].\end{aligned}$$

Proof Integrating both sides of model (1.6) from 0 to t and dividing by t, we get

$$\begin{cases} \dfrac{y(t)-y(0)}{t} = -(b+\varepsilon+c+\theta-\lambda)\langle y(t)\rangle - \lambda\langle z(t)y(t)\rangle \\ \qquad\qquad - (\lambda-\varepsilon)\left\langle y(t)^2\right\rangle + \dfrac{\sigma}{t}\displaystyle\int_0^t (1-y-z)\,y dB(s), \\ \dfrac{z(t)-z(0)}{t} = p - (b+e+p)\langle z(t)\rangle + (c-p)\langle y(t)\rangle + \varepsilon\langle y(t)z(t)\rangle. \end{cases} \tag{4.1}$$

By (4.1), one has

$$\begin{aligned} & \varepsilon\cdot\frac{y(t)-y(0)}{t} + \lambda\cdot\frac{z(t)-z(0)}{t} \\ =& \{(c-p)\lambda + \varepsilon[\lambda-(b+\varepsilon+c+\theta)]\}\langle y(t)\rangle \\ & + \varepsilon(\varepsilon-\lambda)\left\langle y(t)^2\right\rangle + \frac{\varepsilon\sigma}{t}\int_0^t (1-y-z)\,y dB(s) \\ & + p\lambda - (b+e+p)\lambda\langle z(t)\rangle. \end{aligned}$$

After calculation, we can get

$$\begin{aligned}\langle z(t)\rangle =&\left\{\frac{c-p}{b+e+p}+\frac{\varepsilon\left[\lambda-(b+\varepsilon+c+\theta)\right]}{(b+e+p)\lambda}\right\}\langle y(t)\rangle\\&-\frac{\varepsilon(\lambda-\varepsilon)}{(b+e+p)\lambda}\left\langle y(t)^2\right\rangle+\frac{p}{b+e+p}-\varphi(t),\end{aligned}\tag{4.2}$$

where

$$\varphi(t)=\frac{\varepsilon(y(t)-y(0))}{(b+e+p)\lambda t}+\frac{\lambda(z(t)-z(0))}{(b+e+p)\lambda t}-\frac{\varepsilon\sigma\int_0^t(1-y-z)\,ydB(s)}{(b+e+p)\lambda t}.$$

Considering that $0<x(t),y(t),z(t)<1$ and $x+y+z=1$, according to Martingale's large number theorem, we can obtain

$$\lim_{t\to\infty}\frac{y(t)}{t}=0,\ \lim_{t\to\infty}\frac{z(t)}{t}=0,\ \lim_{t\to\infty}\frac{\int_0^t x(s)\,y(s)\,dB(s)}{t}=0\quad\text{a.s.}$$

Then, it is obvious that

$$\lim_{t\to\infty}\varphi(t)=0\quad\text{a.s.}\tag{4.3}$$

According to Itô formula, one has

$$\begin{aligned}d(\ln y)=&\left[-(b+\varepsilon+c+\theta)+\lambda(1-y-z)+\varepsilon y\right.\\&\left.-\frac{\sigma^2(1-y-z)^2}{2}\right]dt+\sigma(1-y-z)\,dB(t).\end{aligned}\tag{4.4}$$

Integrating both sides of Eq (4.4) from 0 to t and dividing by t, we get

$$\begin{aligned}\frac{\ln y(t)-\ln y(0)}{t}=&\lambda-(b+\varepsilon+c+\theta)-(\lambda-\varepsilon)\langle y(t)\rangle\\&-\lambda\langle z(t)\rangle-\frac{\sigma^2\left\langle(1-y(t)-z(t))^2\right\rangle}{2}+\frac{M_1(t)}{t}\\\geq&\lambda-(b+\varepsilon+c+\theta)-(\lambda-\varepsilon)\langle y(t)\rangle-\lambda\langle z(t)\rangle-\frac{\sigma^2}{2}+\frac{M_1(t)}{t}.\end{aligned}\tag{4.5}$$

By (4.2) and Schwarz inequality, one has

$$\begin{aligned}\frac{\ln y(t)-\ln y(0)}{t}\geq&\left[\lambda-\left(b+\varepsilon+c+\theta+\frac{\sigma^2}{2}+\frac{\lambda p}{b+e+p}\right)\right]\\&-\pi_0\langle y(t)\rangle+\frac{\varepsilon(\lambda-\varepsilon)}{b+e+p}\langle y(t)\rangle^2+\lambda\varphi(t)+\frac{M_1(t)}{t},\end{aligned}\tag{4.6}$$

where $\pi_0=(\lambda-\varepsilon)+\frac{(c-p)\lambda+\varepsilon[\lambda-(b+\varepsilon+c+\theta)]}{b+e+p}$. Thus,

$$\frac{\varepsilon(\lambda-\varepsilon)}{b+e+p}\langle y(t)\rangle^2-\pi_0\langle y(t)\rangle+\left[\lambda-\left(b+\varepsilon+c+\theta+\frac{\sigma^2}{2}+\frac{\lambda p}{b+e+p}\right)\right]\leq\Phi(t),$$

where $\Phi(t) = \frac{\ln y(t) - \ln y(0)}{t} - \lambda\varphi(t) - \frac{M_1(t)}{t}$. By Eqs (3.10) and (4.3), combined with $-\infty < \ln y(t) < 0$ $(0 < y(t) < 1)$, we have

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

Then for arbitrary $\forall 0 < \epsilon < 1$, there exists a set Ω_ϵ such that $P(\Omega_\epsilon) \geq 1 - \epsilon$ and a random variable $T = T(\omega) > 0$ such that for any $\omega \in \Omega_\epsilon$ and any $t \geq T(\omega)$, then $\Phi(t) \leq \epsilon$. Hence, we get

$$\frac{\varepsilon(\lambda-\varepsilon)}{b+e+p}\langle y(t)\rangle^2 - \pi_0\langle y(t)\rangle + \left[\lambda - \left(b+\varepsilon+c+\theta+\frac{\sigma^2}{2}+\frac{\lambda p}{b+e+p}\right) - \epsilon\right] \leq 0. \tag{4.7}$$

For the sake of discussion, let's write (4.7) as $\pi_1\langle y(t)\rangle^2 + \pi_2\langle y(t)\rangle + \pi_3 \leq 0$, where $\pi_1 = \frac{\varepsilon(\lambda-\varepsilon)}{b+e+p}$, $\pi_2 = -\pi_0$, $\pi_3 = \lambda - \left(b+\varepsilon+c+\theta+\frac{\sigma^2}{2}+\frac{\lambda p}{b+e+p}\right) - \epsilon$. If $R_0^s > 1$, then

$$\begin{aligned}
\Delta =& \pi_2^2 - 4\pi_1\pi_3 \\
=& \pi_0^2 - \frac{4\varepsilon(\lambda-\varepsilon)}{b+e+p}\left[\lambda - \epsilon\right. \\
& \left. - \left(b+\varepsilon+c+\theta+\frac{\sigma^2}{2}+\frac{\lambda p}{b+e+p}\right)\right] \\
=& \left\{(\lambda-\varepsilon) + \frac{(c-p)\lambda - \varepsilon[\lambda-(b+\varepsilon+c+\theta)]}{b+e+p}\right\}^2 \\
& + \frac{4(c-p)\varepsilon\lambda[\lambda-(b+\varepsilon+c+\theta)]}{(b+e+p)^2} \\
& + \frac{(\lambda-\varepsilon)\varepsilon\left[\frac{4\lambda p}{b+e+p} + 2\sigma^2 + 4\epsilon\right]}{b+e+p} > 0.
\end{aligned} \tag{4.8}$$

If $\epsilon < \lambda - \left(b+\varepsilon+c+\theta+\frac{\sigma^2}{2}+\frac{\lambda p}{b+e+p}\right)$, then by (4.7), one has

$$y_1^\epsilon \leq \langle y(t)\rangle \leq y_2^\epsilon, \quad t \geq T(\omega),\ \omega \in \Omega_\epsilon, \tag{4.9}$$

where

$$y_1^\epsilon = \frac{(\lambda-\varepsilon)(b+e+p) + \{(c-p)\lambda + \varepsilon[\lambda-(b+\varepsilon+c+\theta)]\} - (b+e+p)\sqrt{\Delta}}{2(\lambda-\varepsilon)\varepsilon},$$

$$y_2^\epsilon = \frac{(\lambda-\varepsilon)(b+e+p) + \{(c-p)\lambda + \varepsilon[\lambda-(b+\varepsilon+c+\theta)]\} + (b+e+p)\sqrt{\Delta}}{2(\lambda-\varepsilon)\varepsilon}.$$

The following will prove that

$$0 < y_1^\epsilon < \frac{\lambda-(b+\varepsilon+c+\theta)}{\lambda-\varepsilon} < 1. \tag{4.10}$$

From (4.8), we can get

$$\lambda - \varepsilon + \frac{(c-p)\lambda - \varepsilon[\lambda-(b+\varepsilon+c+\theta)]}{b+e+p} < \sqrt{\Delta}.$$

By calculation, we can get

$$\begin{aligned}&(\lambda-\varepsilon)(b+e+p)+(c-p)\lambda+\varepsilon[\lambda-(b+\varepsilon+c+\theta)]-(b+e+p)\sqrt{\Delta}\\<&2\varepsilon[\lambda-(b+\varepsilon+c+\theta)],\end{aligned}$$

thus

$$y_1^{\epsilon}<\frac{2\varepsilon[\lambda-(b+\varepsilon+c+\theta)]}{2\varepsilon(\lambda-\varepsilon)}=\frac{\lambda-(b+\varepsilon+c+\theta)}{\lambda-\varepsilon}<1.$$

Therefore, (4.10) is true. By (4.9), we have $\liminf_{t\to\infty}\langle y(t)\rangle\geq y_1^{\epsilon}$ a.s. Letting $\epsilon\to 0$, one has

$$\liminf_{t\to\infty}\langle y(t)\rangle\geq\frac{(b+e+p)\left(\pi_0-\sqrt{\Delta'}\right)}{2\varepsilon(\lambda-\varepsilon)}:=y^{*}\text{ a.s.},$$

where

$$\Delta':=\pi_0^2-\frac{4\varepsilon(\lambda-\varepsilon)}{b+e+p}\left[\lambda-\left(b+\varepsilon+c+\theta+\frac{\sigma^2}{2}+\frac{\lambda p}{b+e+p}\right)\right]$$

and

$$0<y^{*}\leq\frac{\lambda-(b+\varepsilon+c+\theta)}{\lambda-\varepsilon}<1.$$

Last, from the last equation of (4.1), we have

$$\begin{aligned}\frac{z(t)-z(0)}{t}&=p-(b+e+p)\langle z(t)\rangle+(c-p)\langle y(t)\rangle+\varepsilon\langle y(t)z(t)\rangle\\&\geq p-(b+e+p)\langle z(t)\rangle+(c-p)\langle y(t)\rangle,\end{aligned}$$

so

$$\langle z(t)\rangle\geq\frac{(c-p)\langle y(t)\rangle}{b+e+p}+\frac{p}{b+e+p}-\frac{z(t)-z(0)}{t(b+e+p)}.$$

Hence,

$$\liminf_{t\to\infty}\langle z(t)\rangle\geq\frac{c-p}{b+e+p}y^{*}+\frac{p}{b+e+p}\quad\text{a.s.}$$

Remark 1. *According to Theorems 2 and 3, when the noise is small enough, if $R_0^s>1$ or $R_0^s<1$, the disease will persist or disappear. Hence, we consider R_0^s as the threshold for model (1.6).*

Example 3. The values of the parameters except $\sigma=0.1$ are the same as those in Example 1. In this case, a simple calculation yields

$$R_0^s=\frac{\left(2\lambda-\sigma^2\right)(b+e)+2(b+c+\theta+\varepsilon)p}{2(b+c+\theta+\varepsilon)(b+e+p)}=1.1615>1.$$

Thus for $\forall(y(0),z(0))\in(0,1)\times(0,1)$, by Theorem 3, we have

$$\liminf_{t\to\infty}\langle y(t)\rangle\geq y^{*}\doteq 0.1514,\quad\liminf_{t\to\infty}\langle z(t)\rangle\geq\frac{c-p}{b+e+p}y^{*}+\frac{p}{b+e+p}\doteq 0.0423\quad\text{a.s.}$$

In other words, the disease persists. The computer simulations shown in Figure 3 support our results. They confirm the persistence of the disease.

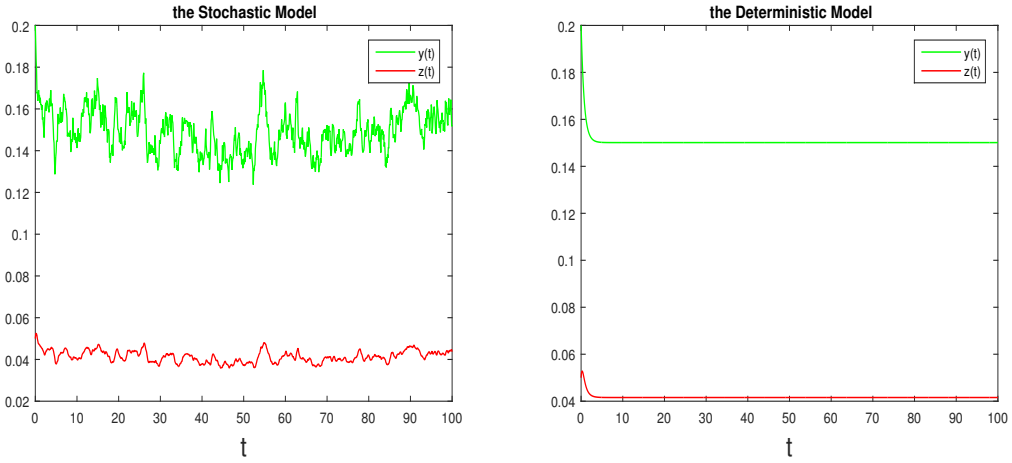

Figure 3. Computer simulation of the path $y(t)$, $z(t)$ for model (1.6) and its corresponding deterministic model, using the EM method with step size $\Delta t = 0.1$ and initial value $y(0) = 0.2$, $z(0) = 0.05$.

5. Asymptotic behavior on endemic equilibrium

This section discusses the impact of stochastic fluctuations of environment on endemic equilibrium $E^*(y^*, z^*)$ of corresponding deterministic systems.

Theorem 4. *If* $\mathrm{R}_0 > 1$, $\varepsilon^2 < \frac{4[p+(c-p)y^*](c-p)(\lambda-\varepsilon)}{\lambda z^*}$, *then for* $\forall\,(y(0), z(0)) \in \mathbb{R}^2_+$, $y(0) + z(0) < 1$, *the solution* $(y(t), z(t))$ *of model (1.6) satisfies the following properties:*

$$\limsup_{t\to\infty} \frac{\int_0^t \left[\left(\lambda - \varepsilon - \frac{\varepsilon\lambda\rho}{2(c-p)}\right)(y(\tau) - y^*)^2 + \frac{\lambda}{c-p}\left(\frac{p+(c-p)y^*}{z^*} - \frac{\varepsilon}{2\rho}\right)(z(\tau) - z^*)^2\right] d\tau}{t} \le \frac{\sigma^2 y^*}{2} \text{ a.s.} \tag{5.1}$$

where the positive constant ρ *satisfies* $\frac{\varepsilon z^*}{2[p+(c-p)y^*]} < \rho < \frac{2(c-p)(\lambda-\varepsilon)}{\varepsilon\lambda}$.

Proof Because $E^*(y^*, z^*)$ is the endemic equilibrium point of the deterministic model corresponding to model (1.6), one has

$$b + \varepsilon + c + \theta - \lambda = -\lambda z^* + (\varepsilon - \lambda) y^*, \tag{5.2}$$

$$p + (c - p) y^* + \varepsilon y^* z^* = (b + e + p) z^*. \tag{5.3}$$

The C^2-function $V : (0, 1) \times (0, 1) \to \mathbb{R}_+$ is defined as follows:

$$V(y, z) = \left(y - y^* - y^* \ln \frac{y}{y^*}\right) + \frac{\lambda}{c - p} \frac{(z - z^*)^2}{2} := V_1 + \frac{\lambda}{c - p} V_2, \tag{5.4}$$

where $V_1 = y - y^* - y^* \ln \frac{y}{y^*}$, $V_2 = \frac{(z-z^*)^2}{2}$. It is easy to see that the function V is non-negative. Let the

operator L be the generating operator of model (1.6). By (5.2), we have

$$\begin{aligned} LV_1 &= \left[-(b+\varepsilon+c+\theta-\lambda)-\lambda z+(\varepsilon-\lambda)y\right](y-y^*)+\frac{1}{2}\sigma^2(1-y-z)^2y^* \\ &= \left[\lambda(z^*-z)-(\varepsilon-\lambda)(y^*-y)\right](y-y^*)+\frac{1}{2}\sigma^2(1-y-z)^2y^* \\ &= -\lambda(y-y^*)(z-z^*)-(\lambda-\varepsilon)(y-y^*)^2+\frac{1}{2}\sigma^2(1-y-z)^2y^* \\ &\le -\lambda(y-y^*)(z-z^*)-(\lambda-\varepsilon)(y-y^*)^2+\frac{1}{2}\sigma^2y^*. \end{aligned} \tag{5.5}$$

By (5.3), one has

$$\begin{aligned} LV_2 &= (z-z^*)\left[p-(b+e+p)z+(c-p)y+\varepsilon yz\right] \\ &= (z-z^*)\left\{(c-p+\varepsilon z)(y-y^*)+\left[\varepsilon y^*-(b+e+p)\right](z-z^*)\right\} \\ &= (z-z^*)\left\{(c-p+\varepsilon z)(y-y^*)-\left[\frac{p}{z^*}+(c-p)\frac{y^*}{z^*}\right](z-z^*)\right\} \\ &= (c-p+\varepsilon z)(y-y^*)(z-z^*)-\left[\frac{p}{z^*}+(c-p)\frac{y^*}{z^*}\right](z-z^*)^2. \end{aligned} \tag{5.6}$$

By (5.4)–(5.6) and Young's inequality, we obtain

$$\begin{aligned} LV =& LV_1+\frac{\lambda}{c-p}LV_2 \\ \le& -(\lambda-\varepsilon)(y-y^*)^2+\frac{\varepsilon\lambda z(y-y^*)(z-z^*)}{c-p} \\ &-\frac{\lambda\left[p+(c-p)y^*\right]}{(c-p)z^*}(z-z^*)^2+\frac{1}{2}\sigma^2y^* \\ \le& -(\lambda-\varepsilon)(y-y^*)^2+\frac{\varepsilon\lambda|y-y^*||z-z^*|}{c-p} \\ &-\frac{\lambda\left[p+(c-p)y^*\right]}{(c-p)z^*}(z-z^*)^2+\frac{1}{2}\sigma^2y^* \\ \le& -\left(\lambda-\varepsilon-\frac{\varepsilon\lambda\rho}{2(c-p)}\right)(y-y^*)^2-\frac{\lambda}{c-p}\times \\ &\left[\frac{p+(c-p)y^*}{z^*}-\frac{\varepsilon}{2\rho}\right](z-z^*)^2+\frac{1}{2}\sigma^2y^* := H, \end{aligned} \tag{5.7}$$

where ρ is defined in Theorem 4. If $\varepsilon^2 < \frac{4\left[p+(c-p)y^*\right](c-p)(\lambda-\varepsilon)}{\lambda z^*}$, then $\lambda-\varepsilon-\frac{\varepsilon\lambda\rho}{2(c-p)}>0$, $\frac{p+(c-p)y^*}{z^*}-\frac{\varepsilon}{2\rho}>0$. Thus,

$$dV \le (1.6)dt+\sigma(1-y-z)(y-y^*)\,dB(t).$$

Integrating from 0 to t of the above inequality, we can obtain

$$V(t)-V(0) \le \int_0^t H(\tau)\,d\tau+\int_0^t \sigma(1-y-z)(y-y^*)\,dB(\tau). \tag{5.8}$$

Let $M_2(t) := \int_0^t \sigma(1-y-z)(y-y^*)\,dB(\tau)$. According to Martingale's large number theorem, we can get $\lim\limits_{t\to\infty} \frac{M_2(t)}{t} = 0$ a.s., this together with (5.8) shows

$$\limsup_{t\to\infty} \frac{\int_0^t H(\tau)\,d\tau}{t} \geq 0 \text{ a.s.}$$

Hence,

$$\limsup_{t\to\infty} \frac{\int_0^t \left[\left(\lambda-\varepsilon-\frac{\varepsilon\lambda\rho}{2(c-p)}\right)(y(\tau)-y^*)^2 + \frac{\lambda}{c-p}\left(\frac{p+(c-p)y^*}{z^*}-\frac{\varepsilon}{2\rho}\right)(z(\tau)-z^*)^2\right]d\tau}{t} \leq \frac{\sigma^2 y^*}{2} \text{ a.s.}$$

Example 4. The values of the parameters except σ are the same as those in Example 1. In this case, by a simple calculation, we can get $R_0 = 1.02 > 1$. By Theorem 4, we get that the difference between disturbance solutions $(y(t), z(t))$ of model (1.6) and $E^*(y^*, z^*)$ is only related to white noise level. By using the EM method, we show that computer simulations support our results. As expected, the solution oscillates around the endemic equilibrium E^* for a long time (see Figure 4). Specifically, the following images use the same parameters, but the intensity of white noise σ is different, that is, the first image uses $\sigma = 0.05$, the second image uses $\sigma = 0.01$. We observe that as the white noise becomes weaker, the fluctuation around E^* becomes smaller, which supports the result of Theorem 4.

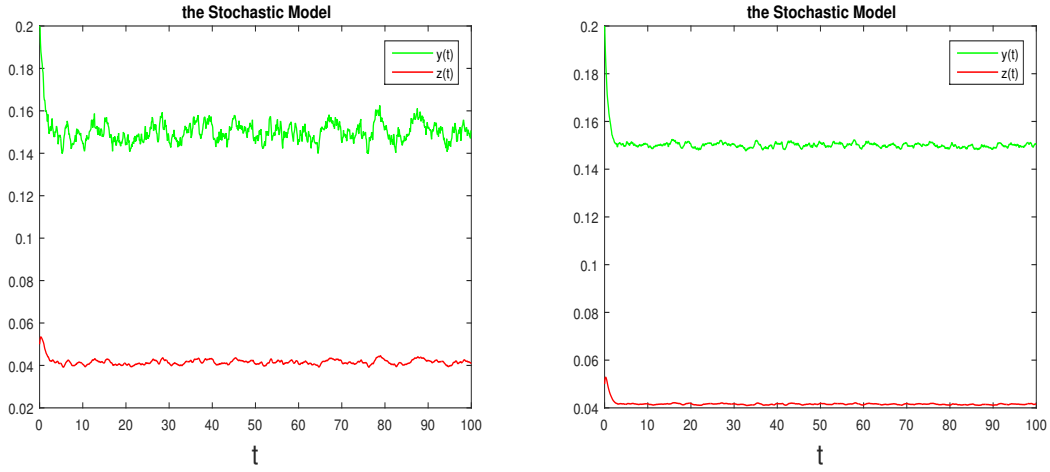

Figure 4. Computer simulation of the path $y(t)$, $z(t)$ for model (1.6). The graphs use different white noise, the left graph with $\sigma = 0.05$ and the right grapg with $\sigma = 0.01$. Using the EM method with step size $\Delta t = 0.1$ and initial value $y(0) = 0.2$, $z(0) = 0.05$.

6. Discussion and conclusions

The objective of this study is to investigate the transmission dynamics of a stochastic epidemic model with vaccination. A new stochastic threshold R_0^s is determined. By using stochastic Lyapunov function theory, the existence and uniqueness of global positive solutions of the model are proved (see Theorem 1). Next, the extinction of disease (see Theorem 2) and persistence conditions (see Theorem

3) are established. When the noise is very low ($R_0^s < 1$), the disease becomes extinct, and if $R_0^s > 1$, the disease persists. We also show that if the conditions of Theorem 4 are true, the solution of the model oscillates around the endemic equilibrium point of the deterministic system and the intensity of the fluctuation is proportional to the intensity of the white noise. In this paper, several examples are used to verify the rationality of the relevant theorems, and numerical simulations are used to support our research results. The results show that the extinction and persistence of the disease depended on the intensity of white noise, i.e., the higher the intensity of white noise, the higher the extinction rate of the disease; the lower the intensity of white noise, the more persistent the disease. Our study shows that stochastic epidemic models based on virus dynamics are more realistic. This theory can provide a solid foundation for the study of similar diseases and has a wide range of applications in the biomedical field. For example, a stochastic delayed infectious disease model can be considered to study the effect of incubation periods on disease dynamics. In addition, our proposed theory can also be used to study other infectious diseases, such as HIV, COIVD-19, tuberculosis and so on.

Acknowledgments

This research is funded by the Natural Science Foundation of Xinjiang Uygur Autonomous Region (Grant No. 2021D01B35), Natural Science Foundation of Colleges and Universities in Xinjiang Uygur Autonomous Region (Grant No. XJEDU2021Y048) and Doctoral Initiation Fund of Xinjiang Institute of Engineering (Grant No. 2020xgy012302).

Conflict of interest

The authors declare there is no conflict of interest.

References

1. J. Gu, Z. Zhou, Y. Wang, Editorial: evolutionary mechanisms of infectious diseases, *Front. Microbiol.*, **12** (2021), 667561. https://doi.org/10.3389/fmicb.2021.667561
2. W. Garira, M. C. Mafunda, From individual health to community health: towards multiscale modeling of directly transmitted infectious disease systems, *J. Biol. Syst.*, **27** (2019), 131–166. https://doi.org/10.1142/S0218339019500074
3. T. Sawakami, K. Karako, P. P. Song, W. Sugiura, N. Kokudo, Infectious disease activity during the COVID-19 epidemic in Japan: lessons learned from prevention and control measures comment, *Biosci. Trends*, **15** (2020), 257–261. https://doi.org/10.5582/bst.2021.01269
4. Y. Takeuchi, T. Sasaki, T. Kajiwara, Construction of Lyapunov functions for some models of infectious diseases in vivo: from simple models to complex models, *Math. Biosci. Eng.*, **12** (2015), 117–133. https://doi.org/10.3934/mbe.2015.12.117
5. J. R. Giles, E. zu Erbach-Schoenberg, A. J. Tatem, L. Gardner, O. N. Bjornstad, C. J. E. Metcalf, et al., The duration of travel impacts the spatial dynamics of infectious diseases, *P. Natl. Acad. Sci. U.S.A.*, **117** (2000), 22572–22579. https://doi.org/10.1073/pnas.1922663117
6. J. Rossello, M. Santana-Gallego, W. Awan, Infectious disease risk and international tourism demand, *Health Policy Plann.*, **32** (2017), 538–548. https://doi.org/10.1093/heapol/czw177

7. K. E. Jones, N. G. Patel, M. A. Levy, A. Storeygard, D. Balk, J. L. Gittleman, et al., Global trends in emerging infectious diseases, *Nature*, **451** (2008), 990–993. https://doi.org/10.1038/nature06536
8. S. Hussain, E. N. Madi, H. Khan, S. Etemad, S. Rezapour, T. Sitthiwirattham, et al., Investigation of the stochastic modeling of COVID-19 with environmental noise from the analytical and numerical point of view, *Mathematics*, **9** (2022), 23. https://doi.org/10.3390/math9233122
9. A. Din, Y. J. Li, Q. Liu, Viral dynamics and control of hepatitis B virus (HBV) using an epidemic model, *Alexandria Eng. J.*, **59** (2020), 667–679. https://doi.org/10.1016/j.aej.2020.01.034
10. S. Kumar, A. Ahmadian, R. Kumar, D. Kumar, J. Singh, D. Baleanu, et al., An efficient numerical method for fractional SIR epidemic model of infectious disease by using Bernstein wavelets, *Mathematics*, **8** (2020), 558. https://doi.org/10.3390/math8040558
11. W. O. Kermack, A. G. McKendrick, A contribution to the mathematical theory of epidemics, *Proc. R. Soc. London*, **115** (1927), 700–721. https://doi.org/10.1098/rspa.1927.0118
12. W. O. Kermack, A. G. A. McKendrick, Contributions to the mathematical theory of epidemics–II. the problem of endemicity, *Bull. Math. Biol.*, **53** (1991), 57–87. https://doi.org/10.1007/BF02464424
13. P. van den Driessche, J. Watmough, A simple SIS epidemic model with a backward bifurcation, *J. Math. Biol.*, **40** (2000), 525–540. https://doi.org/10.1007/s002850000032
14. X. Z. Meng, S. N. Zhao, T. Feng, T. H. Zhang, Dynamics of a novel nonlinear stochastic SIS epidemic model with double epidemic hypothesis, *J. Math. Anal. Appl.*, **433** (2016), 227–242.https://doi.org/10.1016/j.jmaa.2015.07.056
15. C. C. McCluskey, Complete global stability for an SIR epidemic model with delay-Distributed or discrete, *Nonlinear Anal.: Real World Appl.*, **11** (2010), 55–59. https://doi.org/10.1016/j.nonrwa.2008.10.014
16. I. Cooper, A. Mondal, C. G. Antonopoulos, A SIR model assumption for the spread of COVID-19 in different communities, *Chaos, Solitons Fractals*, **139** (2020), 110057. https://doi.org/10.1016/j.chaos.2020.110057
17. Y. A. Zhao, D. Q. Jiang, X. R. Mao, A. Gray, The threshold of a stochastic SIRS epidemic model in a population with varying size, *Discrete Contin. Dyn. Syst. -B*, **20** (2015), 1277–1295. https://doi.org/10.3934/dcdsb.2015.20.1277
18. Y. l. Cai, Y. Kang, M. Banerjee, W. M. Wang, A stochastic SIRS epidemic model with infectious force under intervention strategies, *J. Differ. Equations*, **259** (2015), 7463–7502. https://doi.org/10.1016/j.jde.2015.08.024
19. Z. F. Yang, Z. Q. Zeng, K. Wang, Modified SEIR and AI prediction of the epidemics trend of COVID-19 in China under public health interventions, *J. Thorac. Dis.*, **13** (2020), 165–174. https://doi.org/10.21037/jtd.2020.02.64
20. J. M. Carcione, J. E. Santos, C. Bagaini, J. Ba, A Simulation of a COVID-19 epidemic based on a deterministic SEIR model, *Front. Public Health*, **8** (2020), 1–13. https://doi.org/10.3389/fpubh.2020.00230

21. S. Ruschel, T. Pereira, S. Yanchuk, L. S. Young, An SIQ delay differential equations model for disease control via isolation, *J. Math. Biol.*, **79** (2019), 249–279. https://doi.org/10.1007/s00285-019-01356-1

22. X. B. Zhang, X. H. Zhang, The threshold of a deterministic and a stochastic SIQS epidemic model with varying total population size, *Appl. Math. Model.*, **10** (2021), 749–767. https://doi.org/10.1016/j.apm.2020.09.050

23. Y. L. Huizer, C. M. Swaan, K. C. Leitmeyer, A. Timen, Usefulness and applicability of infectious disease control measures in air travel: a review, *Travel Med. Infect. Dis.*, **13** (2015), 19–30. https://doi.org/10.1016/j.tmaid.2014.11.008

24. A. Din, Y. J. Li, T. Khan, G. Zaman, Mathematical analysis of spread and control of the novel corona virus (COVID-19) in China, *Chaos, Solitons Fractals*, **141** (2021), 110286. https://doi.org/10.1016/j.chaos.2020.110286

25. S. R. Gani, S. V. Halawar, Optimal control for the spread of infectious disease: the role of awareness programs by media and antiviral treatment, *Optim. Control. Appl. Methods*, **39** (2018), 1407–1430. https://doi.org/10.1002/oca.2418

26. Z. Jin, The SIR epidemical models with continuous and impulsive vaccinations, *J. North China Inst. Technol.*, **24** (2003), 235–243.

27. A. Gray, D. Greenhalgh, L. Hu, X. Mao, J. Pan, A stochastic differential equation SIS epidemic model, *SIAM J. Appl. Math.*, **71** (2011), 876–902. https://doi.org/10.1137/10081856X

28. Y. N. Zhao, D. Q. Jiang, The threshold of a stochastic SIRS epidemic model with saturated incidence, *Appl. Math. Lett.*, **34** (2014), 90–93. https://doi.org/10.1016/j.aml.2013.11.002

29. B. Y. Wen, Z. D. Teng, Z. M. Li, The threshold of a periodic stochastic SIVS epidemic model with nonlinear incidence, *Phys. A*, **508** (2018), 532–549. https://doi.org/10.1016/j.physa.2018.05.056

30. Q. Liu, Q. M. Chen, D. Q. Jiang, The threshold of a stochastic delayed SIR epidemic model with temporary immunity, *Phys. A*, **450** (2016), 115–125. https://doi.org/10.1016/j.physa.2015.12.056

31. M. El Fatini, R. Pettersson, I. Sekkak, R. Taki, A stochastic analysis for a triple delayed SIQR epidemic model with vaccination and elimination strategies, *J. Appl. Math. Comput.*, **64** (2020), 781–805. https://doi.org/10.1007/s12190-020-01380-1

32. X. Mao, *Stochastic Differential Equations and Applications*, 2nd edition, Chichester: Horwood Publishing Limited, 2008.

33. D. J. Higham, An algorithmic introduction to numerical simulation of stochastic differential equations, *SIAM Rev.*, **43** (2001), 525–546. https://doi.org/10.1137/S0036144500378302