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Research article

Dynamics and stochastic sensitivity technique of a stochastic SIRS epidemic model

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Abstract: The article investigates a stochastic SIRS epidemic model by perturbing the natural death rate. A stochastic threshold R_s that determines the dynamics of the model is given. More concretely, if $R_s < 1$, then the disease will die out; if $R_s > 1$, then the model exhibits an ergodic stationary distribution. Furthermore, a Gaussian approximation of the stationary distribution is given using the stochastic sensitivity technique. For the visual description of the distribution, a confidence ellipsoid is presented by the visualization geometric method of confidence domains. The confidence ellipsoid is helpful for us to estimate the equilibrium region of the stochastic model. Moreover, several numerical analyses are presented to verify the results.

Keywords: SIRS model; stochastic threshold; ergodic stationary distribution; stochastic sensitivity technique; confidence ellipsoid
Mathematics Subject Classification: 92D30, 92D25, 60H10, 34D20, 37A30

1. Introduction

In the 21st century, the COVID-19 pandemic brought great disaster to all countries of the world [1]. The World Health Organization reported that nearly 77,907,303 people were infected with COVID-19, and there were 7,109,103 people deaths in the world until 11 January 2026 [2]. Since the contributions of Kermack and McKendrick [3], mathematical modeling has been instrumental in advancing both qualitative and quantitative insights into infectious disease dynamics, thus providing a critical foundation to formulate effective control methods. Many scholars have used mathematical models to study epidemics and have achieved significant results [4–13].

Many diseases have an increasing infectiousness at the beginning of the epidemic, which gradually declines due to external human interventions or people's fear of infectious diseases. In 2015, Cai et al [14]. considered the impact of psychological factors and intervention strategies in a SIRS model as follows:

$$\begin{cases} dS(t) &= [\Lambda - \mu S(t) - \frac{\beta I(t)}{f(I(t))}S(t) + \delta R(t)]dt - \frac{\sigma I(t)S(t)}{f(I(t))}dB(t), \\ dI(t) &= [\frac{\beta I(t)}{f(I(t))}S(t) - (\mu + \gamma + \epsilon)I(t)]dt + \frac{\sigma I(t)S(t)}{f(I(t))}dB(t), \\ dR(t) &= [\gamma I(t) - (\mu + \delta)R(t)]dt, \end{cases} \quad (1.1)$$

where $S(t)$ denotes susceptible people, $I(t)$ denotes the infectious population, $R(t)$ represents the population of recovered individuals. The function $f(I)$ meets the following conditions:

($\mathbf{C}_1$) $f(0) > 0$, $f'(I) > 0$, when $I > 0$;

($\mathbf{C}_2$) A constant $\theta > 0$ exists, s.t. $(\frac{I}{f(I)})' > 0$, when $I \in [0, \theta]$; $(\frac{I}{f(I)})' < 0$, when $I \in [\theta, \infty)$.

Epidemiologically, ($\mathbf{C}_1$) and ($\mathbf{C}_2$) characterize the impact of intervention strategies which are determined by an important parameter θ: when $I \in [0, \theta]$, the incidence rate is increasing; however, it reduces when $I \in [\theta, \infty)$. The authors in [14] obtained a reproduction number R_0^s. If $R_0^s < 1$, then the disease will extinct; alternatively, there is an endemic stationary distribution under mild extra conditions if $R_0^s > 1$. They have not investigated the Gaussian approximation of the stationary distribution. Moreover, the influence of stochastic factors on the natural death rate μ has not been investigated.

It is worth mentioning that natural death rate μ is not constant but stochastically fluctuates due to the stochastic environmental noise [15]. Furthermore, the sources of stochastic noise shocks experienced by populations with different health statuses may be independent or weakly correlated [16–18]. For instance, the primary source of stochastic shocks for susceptible individuals may be accidental events and behavioral risks; for infected individuals, it may be microscopic fluctuations in the pathogen-host interactions; and for recovered individuals, it may be long-term health sequelae and fluctuations in the immune status [19–21]. Hence, one lets $\xi_i(t), i = 1, 2, 3$, represent the sources of stochastic shocks of susceptible individuals, infected individuals, and recovered individuals, respectively.

Additionally, due to differences of the susceptible, infected, and recovered populations, their sensitivity to environmental stochastic fluctuations also varies. For example, the infected individuals may be more sensitive to fluctuations in medical resources due to the disease itself, while the recovered individuals may respond differently to additional health shocks due to changes in immunity [22, 23]. Thus, one will assign distinct noise sensitivity coefficients, namely σ_1, σ_2, and σ_3, to the three compartments to more accurately reflect the heterogeneity in how different populations are influenced by stochastic noises.

Let $\varepsilon \geq 0$ represent the overall level of the environmental noise fluctuation that affects the natural death rate μ. A larger ε indicates a highly unstable environment, such as underdeveloped healthcare systems, the government's policy intervention, or social unrest, which leads to intense mortality fluctuations. Conversely, a smaller ε reflects a relatively stable socio-environmental condition.

Naturally, some interesting questions arise: What is the impact of the noise sensitivity and the overall intensity of environmental noise fluctuations on the threshold of disease transmission; Is there a stationary distribution; and, how to visualize this stationary distribution?

Many scholars have used different types of stochastic differential equations(SDEs) to study infectious disease models. For example, Albano et al. used Itô's-type SDEs to study SIR models, with a focus on statistical inference [24]. Additionally, Jiang et al. used Itô's type SDEs to study the SIR model and found that disease transmission is related to noise [25]. Lanconelli et al. studied the SIR and SIS models using Stratonovich-type SDEs and made a noteworthy discovery: the intensity of noise described through Stratonovich calculus is not relevant for the extinction of the disease, [26–28]. To investigate the impact of the noise intensity, this paper attempts to study the SIR model using Itô's-type SDEs.

Motivated by the above discussions, one improves the natural death rate μ with stochastic process as follows:

$$\mu \rightarrow \mu + \varepsilon\sigma_i\xi_i(t), i = 1, 2, 3,$$

where $\xi_i(t)$ represents mutually Gaussian white noises, ε measures the overall intensity level of the environmental noises fluctuations, and σ_i measures the noises sensitivity of S, I, and R to environmental fluctuations. $\varepsilon\sigma_i$ measures the effective noise intensity of S, I, and R to environmental noises fluctuations. One introduces randomness into the deterministic model corresponding to Model (1.1) by perturbing μ by $\mu + \varepsilon\sigma_i\xi_i(t), (i = 1, 2, 3)$; then, one obtains the following stochastic SIRS model:

$$\begin{cases} dS(t) = [\Lambda - (\mu + \varepsilon\sigma_1\xi_1(t))S(t) - \frac{\beta I(t)}{f(I(t))}S(t) + \delta R(t)]dt, \\ dI(t) = [\frac{\beta I(t)}{f(I(t))}S(t) - (\mu + \varepsilon\sigma_2\xi_2(t) + \gamma + \epsilon)I(t)]dt, \\ dR(t) = [\gamma I(t) - (\mu + \varepsilon\sigma_3\xi_3(t) + \delta)R(t)]dt. \end{cases} \tag{1.2}$$

Let $B_i(t)$ be the standard one-dimensional independent Brownian motion [29]. Notice that the relations between the $\xi_i(t)$ and $B_i(t)$ are $dB_i(t) = \xi_i(t)dt, (i = 1, 2, 3)$. Then, Model (1.2) becomes the following model:

$$\begin{cases} dS(t) = [\Lambda - \mu S(t) - \frac{\beta I(t)}{f(I(t))}S(t) + \delta R(t)]dt - \varepsilon\sigma_1 S(t)dB_1(t), \\ dI(t) = [\frac{\beta I(t)}{f(I(t))}S(t) - (\mu + \gamma + \epsilon)I(t)]dt - \varepsilon\sigma_2 I(t)dB_2(t), \\ dR(t) = [\gamma I(t) - (\mu + \delta)R(t)]dt - \varepsilon\sigma_3 R(t)dB_3(t). \end{cases} \tag{1.3}$$

It is worth noting that Model (1.3) can be reduced to the traditional single-noise model if $\sigma_1 = \sigma_2 = \sigma_3$, and the three Brownian motions are completely positively correlated. Model (1.3) allows us to explore the deep mechanisms of stochastic environment structures on the disease dynamics by regulating the independence of noise. When $\varepsilon = 0$, Model (1.3) degenerates into the deterministic model with respect to the stochastic model (1.1).

The article is organized as follows: in Section 2, some preliminaries are presented; in Section 3, several theoretical results are obtained, including the existence of uniqueness for the global positive solution, disease extinction, stationary distribution, and stochastic sensitivity technique; in Section 4, the obtained results are validated by few numerical analyses; and Section 5, one gives a few conclusions.

2. Preliminaries

Denote $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t\geq 0}, P)$ as the complete probability space with filtration $\{\mathcal{F}_t\}_{t\geq 0}$ that satisfies the usual condition. Let $\mathbb{R}^3_+ := \{(X_1, X_2, X_3) : X_i > 0, i = 1, 2, 3\}$. $\langle x(t)\rangle = \frac{1}{t}\int_0^t x(r)dr$, $\langle\cdot,\cdot\rangle$ represents the Euclidean scalar product. s.t. represents such that. a.s. denotes almost surely. For convenience, one uses S, I, R, and N to replace $S(t)$, $I(t)$, $R(t)$, and $N(t)$, respectively. $E[X]$ represents the expectation of X.

Definition 2.1. *[29] Let $X(t) \in \mathbb{R}^3_+$ be the solution of following equation:*

$$dX(t) = f(X(t), t)dt + g(X(t), t)dB(t), \quad t \geq 0. \tag{2.1}$$

For Eq (2.1), one defines a operator $\mathcal{L}$ as follows:

$$\mathcal{L} = \frac{\partial}{\partial t} + \sum_{i=1}^{3} f_i(X,t)\frac{\partial}{\partial X_i} + \frac{1}{2}\sum_{i,j=1}^{3}\Big[g^\top(X,t)g(X,t)\Big]_{ij}\frac{\partial^2}{\partial X_i \partial X_j}.$$

By operating $\mathcal{L}$ on $V(X,t)$, one has the following:

$$\mathcal{L}V(X,t) = V_t(X,t) + V_X(X,t)f(X,t) + \frac{1}{2}trace\Big[g^\top(X,t)V_{XX}(X,t)g(X,t)\Big], \tag{2.2}$$

where

$$V_X = \Big(\frac{\partial V}{\partial X_1}, \frac{\partial V}{\partial X_2}, \frac{\partial V}{\partial X_3}\Big)^\top, \; V_{XX} = \Big(\frac{\partial^2 V}{\partial X_i \partial X_j}\Big)_{3\times 3}.$$

Then, one has Itô's formula as follows:

$$dV(X(t),t) = \mathcal{L}V(X(t),t)dt + V_X(X(t),t)g(X(t),t)dB(t).$$

Definition 2.2. *[29] Denote $M = \{M_t\}_{t\geq 0}$ as a local martingale. Then, the following two properties hold:*

$$(i) \qquad \lim_{t\to\infty}\langle M, M\rangle_t = \infty \Rightarrow \lim_{t\to\infty}\frac{M_t}{\langle M, M\rangle_t} = 0 \;\; a.s.$$

$$(ii) \qquad \lim_{t\to\infty}\frac{\langle M, M\rangle_t}{t} < \infty\,. \Rightarrow \lim_{t\to\infty}\frac{M_t}{t} = 0. \;\; a.s.$$

Definition 2.3. *[30] Denote U as a variable that depends on the parameter r; then, the sensitivity index of U corresponding to r is defined by the following:*

$$\Upsilon^U_p := \frac{\partial U}{\partial r} \times \frac{r}{U}. \tag{2.3}$$

Lemma 2.1. *Let $(S(t), I(t), R(t))$ be the solution for the stochastic model (1.3); then, the following properties hold:*

$$\lim_{t\to\infty}\frac{S(t)+I(t)+R(t)}{t} = 0, \;\; \lim_{t\to\infty}\frac{S(t)}{t} = \lim_{t\to\infty}\frac{I(t)}{t} = \lim_{t\to\infty}\frac{R(t)}{t} = 0.$$

Lemma 2.2. *Let $(S(t), I(t), R(t))$ be the solution of Model (1.3); if $\mu > \frac{\varepsilon^2(\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2)}{2}$, then the following properties hold*

$$\lim_{t\to\infty}\frac{1}{t}\int_0^t S(s)dB_1(s) = 0, \;\; \lim_{t\to\infty}\frac{1}{t}\int_0^t I(s)dB_2(s) = 0, \;\; \lim_{t\to\infty}\frac{1}{t}\int_0^t R(s)dB_3(s) = 0.$$

Remark 2.1. *The proof of Lemma 2.1 and Lemma2.2 are given in the Appendix.*

Lemma 2.3. *[31] There is a domain $D \subset \mathbb{R}^d$ which is regular, its boundary Γ is regular, such that the closure $\overline{D} \in \mathbb{R}^d$, and the properties hold as follows:*

(i) There is a constant $C > 0$, s.t. $\sum_{i,j=1}^d a_{ij}(x)\zeta_i\zeta_j \geq C \mid \zeta \mid^2$, for $\zeta \in \mathbb{R}^d, x \in D$.

(ii) There is a C^2−function $V > 0$ s.t. $\mathcal{L}V < 0$, for $\forall X \in \mathbb{R}^d \setminus D$. Consequently, $X(t)$, there exists a unique stationary distribution $\pi(\cdot)$, and

$$\mathrm{P}\Big\{\lim_{T\to\infty}\frac{1}{T}\int_0^T f(X(t))dt = \int_{\mathbb{R}^3_+} f(x)\pi(dx)\Big\} = 1.$$

3. Theoretical results

3.1. Existence uniqueness of global positive solution

Theorem 3.1. *Model* (1.3) *exists with a unique positive solution* $X(t) = (S(t), I(t), R(t))$*, which is global and stays in* $\mathbb{R}^3_+$ *with a probability of 1.*

Proof Because coefficients for Model (1.3) are local Lipschitz, then for $\forall X(0) \in \mathbb{R}^3_+$, the model has a local solution $X(t)$ and is unique, $0 \le t < \tau_e$; here, τ_e represents the explosion time. Next, one demonstrates $X(t)$ as a global solution (i.e, $\tau_e = \infty$). Denote $n_0 > 0$ as big enough s.t. $S(0)$, $I(0)$, and $R(0)$ belong to $[\frac{1}{n_0}, n_0]$. For $n > n_0$, one defines the stop time as follows:

$$\tau_n = \inf\left\{t \in [0, \tau_e] : \min\{S(t), I(t), R(t)\} \le \frac{1}{n} \text{ or } \max\{S(t), I(t), R(t)\} \ge n\right\}.$$

Let $\inf \emptyset = \infty$. Obviously, τ_n monotonously increases when $n \to \infty$. Denotes $\tau_\infty = \lim_{n\to\infty} \tau_n$; it follows that $\tau_\infty \le \tau_e$ a.s.

Now, one only needs manifest $\tau_\infty = \infty$ a.s. When $\tau_\infty \ne \infty$, two numbers $T \in (0, \infty)$ and $\kappa \in (0, 1)$ exist s.t. $P\{\tau_\infty \le T\} \ge \kappa$. Then, one has an integer $n_1 \ge n_0$; the follows inequality holds:

$$P\{\tau_n \le T\} \ge \kappa,\ n \ge n_1. \tag{3.1}$$

Construct a Liapunov function as follows:

$$V(S, I, R) = (S - a - a\ln\frac{S}{a}) + (I - 1 - \ln I) + (R - 1 - \ln R).$$

From Itô's formula, one has

$$\begin{aligned}
\mathrm{d}V &= \left(1 - \frac{a}{S}\right)\left[(\Lambda - \mu S - \frac{\beta I}{f(I)}S + \delta R)\mathrm{d}t - \varepsilon\sigma_1 S\,\mathrm{d}B_1(t)\right] \\
&\quad + \left(1 - \frac{1}{I}\right)\left[(\frac{\beta I}{f(I)}S - (\mu + \gamma + \epsilon)I)\mathrm{d}t - \varepsilon\sigma_2 I\mathrm{d}B_2(t)\right] \\
&\quad + \left(1 - \frac{1}{R}\right)[(\gamma I - (\mu + \delta)R)\mathrm{d}t - \varepsilon\sigma_3 R\mathrm{d}B_3(t)] + \varepsilon^2\frac{a\sigma_1^2 + \sigma_2^2 + \sigma_3^2}{2}\mathrm{d}t \\
&:= \mathcal{L}V\mathrm{d}t - \varepsilon\sigma_1(S - a)\mathrm{d}B_1(t) - \varepsilon\sigma_2(I - 1)\mathrm{d}B_2(t) - \varepsilon\sigma_3(R - 1)\mathrm{d}B_3(t),
\end{aligned}$$

where

$$\begin{aligned}
\mathcal{L}V &= \Lambda - \mu(S + I + R) - \epsilon I - \frac{a\Lambda}{S} + a\mu + \frac{a\beta I}{f(I)} - \frac{a\delta R}{S} - \frac{\beta S}{f(I)} \\
&\quad + \mu + \gamma + \epsilon - \frac{\gamma I}{R} + \mu + \delta + \varepsilon^2\frac{a\sigma_1^2 + \sigma_2^2 + \sigma_3^2}{2} \\
&\le \Lambda + a\mu + (\frac{a\beta}{f(0)} - \epsilon)I + 2\mu + \gamma + \epsilon + \delta + \varepsilon^2\frac{a\sigma_1^2 + \sigma_2^2 + \sigma_3^2}{2}.
\end{aligned}$$

Choose $a = \frac{\epsilon f(0)}{\beta}$; then, one has the following:

$$\mathcal{L}V \le \Lambda + (a + 2)\mu + \gamma + \epsilon + \delta + \varepsilon^2\frac{a\sigma_1^2 + \sigma_2^2 + \sigma_3^2}{2} := K_1,$$

The rest similar to what is described in [14].

3.2. Disease extinction

Theorem 3.2. *Assume* $\mu > \frac{\varepsilon^2(\sigma_1^2\vee\sigma_2^2\vee\sigma_3^2)}{2}$. $(S(t), I(t), R(t))$ *represents the solution of Model* (1.3) *with* $X(0) \in \mathbb{R}_+^3$. *When* $R_s < 1$, *then*

$$\lim_{t\to\infty}\frac{\ln I(t)}{t} \le (\mu+\gamma+\epsilon)(R_s-1) < 0, \quad \lim_{t\to\infty}\langle S(t)\rangle = \frac{\Lambda}{\mu}, \quad \lim_{t\to\infty}\langle R(t)\rangle = 0, \quad a.s,$$

where

$$R_s = \frac{\Lambda\beta}{\mu f(0)(\mu+\gamma+\epsilon)} - \frac{\varepsilon^2\sigma_2^2}{2(\mu+\gamma+\epsilon)}. \tag{3.2}$$

Proof From the first two equations of (1.3), one has the following:

$$\mathrm{d}(S(t)+I(t)) = \Lambda - \mu S(t) + \delta R(t) - (\mu+\gamma+\epsilon)I(t) - \varepsilon\sigma_1 S(t)\mathrm{d}B_1(t) - \varepsilon\sigma_2 I(t)\mathrm{d}B_2(t). \tag{3.3}$$

Integrate Eq (3.3); then, one has the following:

$$\begin{aligned}\frac{1}{t}(S(t)-S(0)) + \frac{1}{t}(I(t)-I(0)) \;&=\; \Lambda - \mu\langle S(t)\rangle + \delta\langle R(t)\rangle - (\mu+\gamma+\epsilon)\langle I(t)\rangle \\ &\quad -\frac{1}{t}\int_0^t \varepsilon\sigma_1 S(s)\mathrm{d}B_1(s) - \frac{1}{t}\int_0^t \varepsilon\sigma_2 I(s)\mathrm{d}B_2(s). \end{aligned}\tag{3.4}$$

From Model (1.3), one gets the following:

$$\frac{1}{t}(R(t)-R(0)) = \gamma\langle I(t)\rangle - (\mu+\delta)\langle R(t)\rangle - \frac{1}{t}\int_0^t \varepsilon\sigma_3 R(s)\mathrm{d}B_3(s). \tag{3.5}$$

Combining Eqs (3.4) and (3.5), one obtains the following:

$$\langle S(t)\rangle = \frac{\Lambda}{\mu} - \frac{[(\mu+\delta)(\mu+\gamma+\epsilon) - \delta\gamma]}{\mu(\mu+\delta)}\langle I(t)\rangle - \varphi(t), \tag{3.6}$$

where

$$\begin{aligned}\varphi(t) \;=\; &\frac{1}{\mu}\left(\frac{S(t)-S(0)}{t} + \frac{I(t)-I(0)}{t} - \frac{1}{t}\int_0^t \varepsilon\sigma_1 S(s)\mathrm{d}B_1(s) - \frac{1}{t}\int_0^t \varepsilon\sigma_2 I(s)\mathrm{d}B_2(s)\right) \\ &+\frac{\delta}{\mu(\mu+\delta)}\left(\frac{(R(t)-R(0))}{t} - \frac{1}{t}\int_0^t \varepsilon\sigma_3 R(s)\mathrm{d}B_3(s)\right).\end{aligned}$$

From Lemmas 2.1 and 2.2,

$$\lim_{t\to\infty}\varphi(t) = 0. \tag{3.7}$$

Using Itô's formula for $\ln I(t)$, one has the following:

$$\mathrm{d}\ln I(t) \;=\; \left[\frac{\beta S(t)}{f(I(t))} - (\mu+\gamma+\epsilon) - \frac{\varepsilon^2\sigma_2^2}{2}\right]\mathrm{d}t - \varepsilon\sigma_2\mathrm{d}B_2(t)$$

$$\leq \quad \left[\frac{\beta S(t)}{f(0)} - (\mu+\gamma+\epsilon) - \frac{\varepsilon^2\sigma_2^2}{2}\right]\mathrm{d}t - \varepsilon\sigma_2 \mathrm{d}B_2(t). \tag{3.8}$$

Integrate Eq (3.8) and combine Eq (3.6); thus,

$$\begin{aligned}
\frac{\ln I(t)}{t} &\leq \frac{\beta\langle S(t)\rangle}{f(0)} - (\mu+\gamma+\epsilon) - \frac{\varepsilon^2\sigma_2^2}{2} - \frac{1}{t}\int_0^t \varepsilon\sigma_2 \mathrm{d}B_2(s) + \frac{\ln I(0)}{t} \\
&= \frac{\beta\Lambda}{\mu f(0)} - (\mu+\gamma+\epsilon) - \frac{\varepsilon^2\sigma_2^2}{2} - \frac{\beta[(\mu+\delta)(\mu+\gamma+\epsilon)-\delta\gamma]}{\mu(\mu+\delta)f(0)}\langle I\rangle \\
&\quad -\frac{\beta\varphi(t)}{f(0)} - \frac{1}{t}\int_0^t \varepsilon\sigma_2 \mathrm{d}B_2(s) + \frac{\ln I(0)}{t} \\
&\leq \frac{\beta\Lambda}{\mu f(0)} - (\mu+\gamma+\epsilon) - \frac{\varepsilon^2\sigma_2^2}{2} - \frac{\beta\varphi(t)}{f(0)} - \frac{1}{t}\int_0^t \varepsilon\sigma_2 \mathrm{d}B_2(s) + \frac{\ln I(0)}{t}.
\end{aligned}$$

Apply Definition 2.2; then, one gets $\lim_{t\to\infty}\frac{1}{t}\int_0^t \varepsilon\sigma_2 \mathrm{d}B_2(s) = 0$. Furthermore, one can obtain the following:

$$\lim_{t\to\infty}\frac{\ln I(t)}{t} \leq \frac{\beta\Lambda}{\mu f(0)} - (\mu+\gamma+\epsilon) - \frac{\varepsilon^2\sigma_2^2}{2} = (\mu+\gamma+\epsilon)(R_s-1) < 0.$$

It means that

$$\lim_{t\to\infty} I(t) = 0. \tag{3.9}$$

Combining with Eqs (3.6), (3.7), and (3.9), one gets the following:

$$\begin{aligned}
\lim_{t\to\infty}\langle S(t)\rangle &= \lim_{t\to\infty}\left[\frac{\Lambda}{\mu} - \frac{[(\mu+\delta)(\mu+\gamma+\epsilon)-\delta\gamma]}{\mu(\mu+\delta)}\langle I(t)\rangle - \varphi(t)\right] \\
&= \frac{\Lambda}{\mu} - \frac{[(\mu+\delta)(\mu+\gamma+\epsilon)-\delta\gamma]}{\mu(\mu+\delta)}\lim_{t\to\infty}\langle I(t)\rangle - \lim_{t\to\infty}\varphi(t) = \frac{\Lambda}{\mu}.
\end{aligned}$$

Form Model (1.3), one has the following:

$$\langle R(t)\rangle = \frac{\gamma}{\mu+\delta}\langle I(t)\rangle - \frac{\int_0^t \varepsilon\sigma_3 R(s)\mathrm{d}B_3(s)}{(\mu+\delta)t} - \frac{R(t)-R(0)}{(\mu+\delta)t}. \tag{3.10}$$

From Eq (3.10), and applying the Lemmas 2.1, and 2.2, one gets the following:

$$\lim_{t\to\infty}\langle R(t)\rangle = 0.$$

Remark 3.1. *Compare the stochastic threshold* $R_s = \frac{\Lambda\beta}{\mu f(0)(\mu+\gamma+\epsilon)} - \frac{\varepsilon^2\sigma_2^2}{2(\mu+\gamma+\epsilon)}$ *of Model* (1.3) *with the threshold* $R_0 = \frac{\Lambda\beta}{\mu f(0)(\mu+\gamma+\epsilon)}$ *of the deterministic model with respect to Model* (1.3)*; then, one has* $R_s = R_0 - \frac{\varepsilon^2\sigma_2^2}{2(\mu+\gamma+\epsilon)}$. *When* $R_0 > 1$, *the disease is persistent. Moreover, a large noise intensity may lead to* $R_s < 1$, *which means the disease is in extinction.*

Remark 3.2. *From the proof process of Theorem 3.2, we found that when considering a correlated Brownian motion, that is, $dB_i dB_j = \rho_{ij} dt$, then the threshold R_s remains unchanged.*

Remark 3.3. *One can study the impact of the overall level of environmental noises fluctuations and the sensitivity on the stochastic threshold of disease transmission. Now, one studies the sensitivity indexs of the ε and σ_2 of R_s by applying Definition 2.3. By Equation (2.3), one has the following:*

$$\Upsilon_{\varepsilon}^{R_s} := \frac{\partial R_s}{\partial \varepsilon} \times \frac{\varepsilon}{R_s} = -\frac{\varepsilon^2\sigma_2^2}{(\mu+\gamma+\epsilon)R_s}, \Upsilon_{\sigma_2}^{R_s} := \frac{\partial R_s}{\partial \sigma_2} \times \frac{\sigma_2}{R_s} = -\frac{\varepsilon^2\sigma_2^2}{(\mu+\gamma+\epsilon)R_s}.$$

Then, one gets $\Upsilon_{\varepsilon}^{R_s} = \Upsilon_{\sigma_2}^{R_s}$, which means that the overall intensity of environmental fluctuations and the noise sensitivity of infected persons are equally important in influencing the threshold of disease transmission.

Mathematically speaking, the dependence of R_s on ε and σ_2 is entirely captured by the product term $\varepsilon^2\sigma_2^2$. This indicates that the overall fluctuation level must act through the sensitivity of infected individuals to exert its effect. In a highly unstable environment (large ε), if infected individuals are insensitive to it (small σ_2), then the impact may be negligible. Conversely, in an apparently stable environment, exceptionally sensitive infected individuals may still trigger disease extinction. This reveals a profound coupling relationship between environmental fluctuations and the population response capacity.

3.3. Stationary distribution and stochastic sensitivity technique

Theorem 3.3. *When $R_s > 1$ and*

$$\Psi < \min\left\{\frac{[\mu(1+\vartheta_1)]^2 S^{*2}}{\mu(1+\vartheta_1)-\varepsilon^2\sigma_1^2}, \frac{[\mu+\epsilon+\vartheta_1(\mu+\gamma+\epsilon)]^2 I^{*2}}{\mu+\epsilon+\vartheta_1(\mu+\gamma+\epsilon)-\varepsilon^2\sigma_2^2}, \frac{[\mu+\vartheta_3(\mu+\delta)]^2 R^{*2}}{\mu+\vartheta_3(\mu+\delta)-\varepsilon^2\sigma_3^2}\right\},$$

where

$$\begin{aligned} \Psi &= \frac{\vartheta_2\varepsilon^2\sigma_2^2 I^*}{2} + \frac{\mu(1+\vartheta_1)\varepsilon^2\sigma_1^2 S^{*2}}{\mu(1+\vartheta_1)-\varepsilon^2\sigma_1^2} + \frac{[\mu+\epsilon+\vartheta_1(\mu+\gamma+\epsilon)]\varepsilon^2\sigma_2^2 I^{*2}}{\mu+\epsilon+\vartheta_1(\mu+\gamma+\epsilon)-\varepsilon^2\sigma_2^2} \\ &\quad + \frac{[\mu+\vartheta_3(\mu+\delta)]\varepsilon^2\sigma_3^2 R^{*2}}{\mu+\vartheta_3(\mu+\delta)-\varepsilon^2\sigma_3^2}, \\ \vartheta_1 &= \frac{2\mu}{\delta},\ \vartheta_2 = \frac{1}{\beta f(0)} f(I^*)[\delta(2\mu+\epsilon)+2\mu(2\mu+\gamma+\epsilon)],\ \vartheta_3 = \frac{\epsilon}{\gamma}, \end{aligned} \tag{3.11}$$

then Model (1.3) *undergoes a stationary distribution $\pi(\cdot)$; here, (S^*, I^*, R^*) represents an endemic equilibrium of the deterministic model that corresponds to Model* (1.3)*.*

Proof First, one calculates the diffusion matrix of Model (1.3) as follows:

$$\mathbf{A}(X) = \varepsilon^2 \begin{pmatrix} \sigma_1^2 S^2 & 0 & 0 \\ 0 & \sigma_2^2 I^2 & 0 \\ 0 & 0 & \sigma_3^2 R^2 \end{pmatrix}.$$

Then, there is a constant $C = \varepsilon^2 \min\left\{\sigma_1^2 S^2,\ \sigma_2^2 I^2,\ \sigma_3^2 R^2\right\}$, s.t. every $(S,\ I,\ R,) \in \overline{D} \subset \mathbb{R}_+^3$ and $\zeta \in \mathbb{R}_+^3 \setminus \{(\zeta_1, \zeta_2, \zeta_3) \in \mathbb{R}_+^3 : \zeta_1 = \zeta_2 = \zeta_3\}$. One has the following:

$$\begin{aligned}\sum_{i,j=1}^{3} a_{i,j}(x)\zeta_i\zeta_j &= \varepsilon^2[\sigma_1^2 S^2 \zeta_1^2 + \sigma_2^2 I^2 \zeta_2^2 + \sigma_3^2 R^2 \zeta_3^2] \\ &\geq \varepsilon^2 \min\left\{\sigma_1^2 S^2,\ \sigma_2^2 I^2,\ \sigma_3^2 R^2\right\} |\zeta|^2 = C|\zeta|^2.\end{aligned}$$

Hence, Condition (i) of Lemma 2.3 is satisfied.

Next, one verifies Condition (ii) of Lemma 2.3. Notice that $R_s = R_0 - \frac{\varepsilon^2\sigma_2^2}{2(\mu+\gamma+\epsilon)}$; when $R_s > 1$, one has $R_0 > 1$. Then, the deterministic model with respect to Model (1.3) has a equilibrium $(S^*, I^*, R^*) := E^* = (E_1^*, E_2^*, E_3^*)$, which is stable, here, S^*, I^*, and R^* satisfy the following equations:

$$\begin{aligned}&\Lambda = \mu S^* + \frac{\beta I^*}{f(I^*)}S^* - \delta R^*, \frac{\beta I^*}{f(I^*)}S^* = (\mu + \gamma + \epsilon)I^*, \gamma I^* = (\mu + \delta)R^*, S^* = \frac{1}{\beta} f(I^*)(\mu + \gamma + \epsilon), \\ &\Lambda - \frac{\mu}{\beta} f(I^*)(\mu + \gamma + \epsilon) - (\mu + \gamma + \epsilon)I^* + \frac{\delta\gamma I^*}{\mu + \delta} = 0, R^* = \frac{\gamma I^*}{\mu + \delta}.\end{aligned}$$

One defines the following function:

$$\begin{aligned}V(S, I, R) &= \frac{1}{2}(S - S^* + I - I^* + R - R^*)^2 + \frac{\vartheta_1}{2}(S - S^* + I - I^*)^2 \\ &\quad +\vartheta_2\left(I - I^* - I^* \ln\left(\frac{I}{I^*}\right)\right) + \frac{\vartheta_3}{2}(R - R^*)^2 \\ &:= V_1 + \vartheta_1 V_2 + \vartheta_2 V_3 + \vartheta_3 V_4.\end{aligned}$$

By the operating $\mathcal{L}$ of (2.2), one has the following:

$$\begin{aligned}\mathcal{L}V_1 &= (S - S^* + I - I^* + R - R^*)[\Lambda - \mu S - (\mu + \epsilon)I - \mu R] + \frac{\varepsilon^2\sigma_1^2 S^2}{2} + \frac{\varepsilon^2\sigma_2^2 I^2}{2} + \frac{\varepsilon^2\sigma_3^2 R^2}{2} \\ &= -\mu(S - S^*)^2 - (\mu + \epsilon)(I - I^*)^2 - \mu(R - R^*)^2 \\ &\quad -2\mu(S - S^*)(R - R^*) - (2\mu + \epsilon)(I - I^*)(R - R^*) \\ &\quad -(2\mu + \epsilon)(S - S^*)(I - I^*) + \frac{\varepsilon^2\sigma_1^2 S^2}{2} + \frac{\varepsilon^2\sigma_2^2 I^2}{2} + \frac{\varepsilon^2\sigma_3^2 R^2}{2}, \\ \mathcal{L}V_2 &= (S - S^* + I - I^*)[\Lambda - \mu S - (\mu + \gamma + \epsilon)I + \delta R] + \frac{\varepsilon^2\sigma_1^2 S^2}{2} + \frac{\varepsilon^2\sigma_2^2 I^2}{2} \\ &= -\mu(S - S^*)^2 - (\mu + \gamma + \epsilon)(I - I^*)^2 - (2\mu + \gamma + \delta)(S - S^*)(I - I^*) \\ &\quad +\delta(S - S^*)(R - R^*) + \delta(I - I^*)(R - R^*) + \frac{\varepsilon^2\sigma_1^2 S^2}{2} + \frac{\varepsilon^2\sigma_2^2 I^2}{2}, \\ \mathcal{L}V_3 &= (I - I^*)[\frac{\beta S}{f(I)} - (\mu + \gamma + \epsilon)] + \frac{\varepsilon^2\sigma_2^2 I^*}{2} \\ &= -\frac{\beta S}{f(I)f(I^*)}[f(I) - f(I^*)](I - I^*) + \frac{\varepsilon^2\sigma_2^2 I^*}{2} + \frac{\beta}{f(I^*)}(S - S^*)(I - I^*) \\ &\leq \frac{\beta}{f(I^*)}(S - S^*)(I - I^*) + \frac{\varepsilon^2\sigma_2^2 I^*}{2},\end{aligned}$$

$$\begin{aligned}\mathcal{L}V_4 &= (R-R^*)(\gamma I-(\mu+\delta)R)+\frac{\varepsilon^2\sigma_3^2R^2}{2}\\ &= \gamma(I-I^*)(R-R^*)+\frac{\varepsilon^2\sigma_3^2R^2}{2}-(\mu+\delta)(R-R^*)^2.\end{aligned}$$

It follows that

$$\begin{aligned}\mathcal{L}V &= \mathcal{L}V_1+\vartheta_1\mathcal{L}V_2+\vartheta_2\mathcal{L}V_3+\vartheta_3\mathcal{L}V_4\\ &\le -\mu(1+\vartheta_1)(S-S^*)^2-[\mu+\epsilon+\vartheta_1(\mu+\gamma+\epsilon)](I-I^*)^2\\ &\quad -[\mu+\vartheta_3(\mu+\delta)](R-R^*)^2+\varepsilon^2\sigma_1^2S^2+\varepsilon^2\sigma_2^2I^2+\varepsilon^2\sigma_3^2R^2+\frac{\varepsilon^2\sigma_2^2I^*}{2}\\ &= -[\mu(1+\vartheta_1)-\varepsilon^2\sigma_1^2]\left[S-\frac{\mu(1+\vartheta_1)S^*}{\mu(1+\vartheta_1)-\varepsilon^2\sigma_1^2}\right]^2+\frac{\mu(1+\vartheta_1)\varepsilon^2\sigma_1^2S^{*2}}{\mu(1+\vartheta_1)-\varepsilon^2\sigma_1^2}\\ &\quad -[\mu+\epsilon+\vartheta_1(\mu+\gamma+\epsilon)-\varepsilon^2\sigma_2^2]\left[I-\frac{(\mu+\epsilon+\vartheta_1(\mu+\gamma+\epsilon))I^*}{\mu+\epsilon+\vartheta_1(\mu+\gamma+\epsilon)-\varepsilon^2\sigma_2^2}\right]^2\\ &\quad -[\mu+\vartheta_3(\mu+\delta)-\varepsilon^2\sigma_3^2]\left[R-\frac{(\mu+\vartheta_3(\mu+\delta))R^*}{\mu+\vartheta_3(\mu+\delta)-\varepsilon^2\sigma_3^2}\right]^2+\frac{\vartheta_2\varepsilon^2\sigma_2^2I^*}{2}\\ &\quad +\frac{[\mu+\vartheta_3(\mu+\delta)]\varepsilon^2\sigma_3^2R^{*2}}{\mu+\vartheta_3(\mu+\delta)-\varepsilon^2\sigma_3^2}+\frac{(\mu+\epsilon+\vartheta_1(\mu+\gamma+\epsilon))\varepsilon^2\sigma_2^2I^{*2}}{\mu+\epsilon+\vartheta_1(\mu+\gamma+\epsilon)-\varepsilon^2\sigma_2^2}\\ &:= -a_1(S-\varpi_1S^*)^2-a_2(I-\varpi_2I^*)^2-a_3(R-\varpi_3R^*)^2+\Psi.\end{aligned}$$

Then, one gets $\mathcal{L}V \le -a_1(S-\varpi_1S^*)^2-a_2(I-\varpi_2I^*)^2-a_3(R-\varpi_3R^*)^2+\Psi$.
At the condition of Theorem 3.3, one has the following:

$$0<\Psi<\min\left\{a_1\varpi_1^2S^{*2},a_2\varpi_2^2I^{*2},a_3\varpi_3^2R^{*2}\right\}.$$

It leads to the following ellipsoid:

$$a_1(S-\varpi_1S^*)^2+a_2(I-\varpi_2I^*)^2+a_3(R-\varpi_3R^*)^2=\Psi,$$

which completely falls inside $\mathbb{R}_+^3$. Therefore, there exists a neighborhood D, such that $\overline{D}\in\mathbb{R}_+^3$, where $\overline{D}$ is a closed set of D. Then, $\mathcal{L}V(X)<0, X\in\mathbb{R}_+^3\setminus D$. Then, Condition (*ii*) for the Lemma 2.3 is satisfied.

Remark 3.4. *Drift dominates in the diffusion outside a compact set. The condition for the existence of a stationary distribution is sufficient, but this condition is relatively complex and may be limited in practice. However, for some given parameters, we can quickly verify whether this condition is met through a numerical simulation to achieve the purpose of the application.*

Remark 3.5. *When the Brownian motions are correlated, that is,* $dB_idB_j=\rho_{ij}dt$*, the ergodicity analysis becomes more involved. The key difference is that the diffusion matrix (or the covariance matrix of the noise) becomes non-diagonal:* $\mathbf{A}(X)=[\rho_{ij}\sigma_i\sigma_jX_iX_j]_{i,j=1,2,3}$*; this matrix must be positive definite for the process to be non-degenerate, which imposes additional constraints on the correlation coefficients* ρ_{ij}*.*

Theorem 3.4. *Denote $X(t,\varepsilon)$ as a solution of the stochastic model (1.3). When $R_s > 1$, then the model (1.3) has a Gaussian approximation $\rho(X,\varepsilon)$ for the stationary distribution around E^*:*

$$\rho(X,\varepsilon) = \frac{\exp\left(-\frac{\left\langle X-E^*, W^{-1}(X-E^*)\right\rangle}{2\varepsilon^2}\right)}{\sqrt{(2\pi\varepsilon^2)^3 \det W}}. \tag{3.12}$$

and a confidence ellipsoid

$$\frac{\beta_1^2}{\mu_1} + \frac{\beta_2^2}{\mu_2} + \frac{\beta_3^2}{\mu_3} = \varepsilon^2 k(P), \tag{3.13}$$

where W represents the stochastic sensitivity matrix for E^*, and $k(P)$ represents the inverse function for the fiducial probability $P(k) = erf\left(\sqrt{\frac{k}{2}}\right) - \sqrt{\frac{2k}{\pi}}e^{-\frac{k}{2}}$. β_i, μ_i $(i = 1, 2, 3)$ and $k(P)$ will be given later.

Proof Let $X = (x_1, x_2, x_3)^\top := (S, I, R)^\top$; Model (1.3) can be written as follows:

$$\dot{X} = f(X) + \varepsilon g(X)\xi(t);$$

here, $f(X)$ represents a 3-dimensional function, $g(X)$ is a 3×3 matrix valued function, $\xi(t)$ is a 3-dimensional white Gaussian noise, and ε measures the overall volatility level of the external stochastic environment. From Theorem 2.1 of [14], E^* is stable and the noise is non-degenerate. The asymptotics

$$z(t) = \lim_{\varepsilon\to 0} \frac{X(t,\varepsilon) - E^*}{\varepsilon},$$

for deviations of $X(t,\varepsilon)$ from E^* satisfies the following linear stochastic model:

$$\dot{z}(t) = Fz + G\xi(t), F = \frac{\partial f}{\partial X}(E^*), G = g(E^*). \tag{3.14}$$

Additionally, $z(t)$ has the second moments $\mathrm{E}\left[z(t)z(t)^\top\right] := M(t)$, which satisfies the following equation:

$$\dot{M} = FW + WF^\top + GG^\top. \tag{3.15}$$

Since E_* is stable, it leads to $W = \lim_{t\to\infty} M(t) := W$, which exists. Additionally, W mets the following matrix equation:

$$FW + WF^\top + GG^\top = 0. \tag{3.16}$$

Furthermore, we get a covariance

$$D(\varepsilon) = \lim_{t\to\infty} \mathrm{E}[(X(t,\varepsilon) - E^*)(X(t,\varepsilon) - E^*)^\top],$$

of $X(t,\varepsilon)$ near E^* in steady regime: $D(\varepsilon) \approx \varepsilon^2 W$. From [32], one can obtain the Gaussian approximation $\rho(X,\varepsilon)$ in (3.12) for the stationary distribution. To visualize this stationary distribution, one constructs a confidence ellipsoid as follows:

$$\frac{\beta_1^2}{\mu_1} + \frac{\beta_2^2}{\mu_2} + \frac{\beta_3^2}{\mu_3} = \varepsilon^2 k(P); \tag{3.17}$$

here, $\beta_1 = \langle X-E^*, v_1\rangle, \beta_2 = \langle X-E^*, v_2\rangle, \beta_3 = \langle X-E^*, v_3\rangle, \mu_1, \mu_2, \mu_3$ are eigenvalues, v_1, v_2, v_3 represent normalized eigenvectors of W, and $k(P)$ denotes inverse function of

$$P(k) = \mathrm{erf}\Big(\sqrt{\frac{k}{2}}\Big) - \sqrt{\frac{2k}{\pi}}e^{-\frac{k}{2}}. \tag{3.18}$$

Remark 3.6. *The size of the confidence ellipsoid (3.17) is determined by both the eigenvalues μ_i and the noise intensity ε. The larger the eigenvalue, the weaker the model's ability to resist stochastic disturbances in that direction, and the wider the range of the stochastic fluctuations. The shape and orientation of an ellipsoid are determined by the eigenvector v_i. Each eigenvector defines a principal axis direction of the ellipsoid, and its corresponding eigenvalue μ_i determines the half length of the axis in that direction (proportional to $\varepsilon\sqrt{\mu_i}$). This indicates that the degree of diffusion of the stochastic states in a specific direction reveals the most vulnerable direction of the model's dynamics.*

Remark 3.7. *This approximation is usually only effective when the noise is small and the stationary distribution is tightly concentrated near the equilibrium. When the model experiences extinction caused by big noise, the confidence ellipsoid may not be applicable.*

Remark 3.8. *When all three noises are, $dB_i(t)dB_j(t) = \rho_{ij}dt$, the covariance matrix W mets the following matrix equation:*

$$FW + WF^\top + GG^\top + Q = 0, \quad Q_{ij} = \rho_{ij}\sigma_i\sigma_j E_i^* E_j^*, i, j = 1, 2, 3, \tag{3.19}$$

which makes the orientation of the confidence ellipsoid dependent on both the deterministic dynamics and the noise correlation structure.

4. Numerical simulations

In the following, one gives several numerical analyses to verify the theoretical results by the Milstein method in Higham [33].

One chooses $f(I) = 1 + \alpha I^2$ [34], which meets the assumed Conditions ($\mathbf{C}_1$) and ($\mathbf{C}_2$). Then the stochastic model (1.3) becomes the following:

$$\begin{cases} \mathrm{d}S(t) = \Big[\Lambda - \mu S(t) - \frac{\beta I(t)}{1+\alpha I^2(t)}S(t) + \delta R(t)\Big]\mathrm{d}t - \varepsilon\sigma_1 S(t)\mathrm{d}B_1(t), \\ \mathrm{d}I(t) = \Big[\frac{\beta I(t)}{1+\alpha I^2(t)}S(t) - (\mu+\gamma+\epsilon)I(t)\Big]\mathrm{d}t - \varepsilon\sigma_2 I(t)\mathrm{d}B_2(t), \\ \mathrm{d}R(t) = [\gamma I(t) - (\mu+\delta)R(t)]\mathrm{d}t - \varepsilon\sigma_3 R(t)\mathrm{d}B_3(t). \end{cases} \tag{4.1}$$

One takes the initial value $X(0) = (0.9, 0.1, 0) \in \mathbb{R}_+^3$ and the parameters

$$\begin{aligned} \Lambda &= 1, \mu = 0.2, \beta = 0.1, \epsilon = 0.05, \gamma = 0.1, \delta = 0.25, \\ \alpha &= 0.001, \sigma_1 = 0.6, \sigma_2 = 0.8, \sigma_3 = 0.7. \end{aligned} \tag{4.2}$$

For the next numerical simulation, we will use the Milstein method in Higham [33]. Then, Model (4.1) can be rewritten as the following discretization equations:

$$\begin{cases} S_{k+1} = S_k + \Big[\Lambda - \mu S_k - \frac{\beta I_k}{1+\alpha I_k^2}S_k + \delta R_k\Big]\triangle t - \varepsilon\sigma_1 S_k\sqrt{\triangle t}\xi_{1,k} - \frac{\varepsilon^2\sigma_1^2}{2}S_k(\xi_{1,k}^2 - 1)\triangle t, \\ I_{k+1} = I_k + \Big[\frac{\beta I_k}{1+\alpha I_k^2}S_k - (\mu+\gamma+\epsilon)I_k\Big]\triangle t - \varepsilon\sigma_2 I_k\sqrt{\triangle t}\xi_{2,k} - \frac{\varepsilon^2\sigma_2^2}{2}I_k(\xi_{2,k}^2 - 1)\triangle t, \\ R_{k+1} = R_k + [\gamma I_k - (\mu+\delta)R_k]\triangle t - \varepsilon\sigma_3 R_k\sqrt{\triangle t}\xi_{3,k} - \frac{\varepsilon^2\sigma_3^2}{2}R_k(\xi_{3,k}^2 - 1)\triangle t. \end{cases} \tag{4.3}$$

where $\xi_{1,k}, \xi_{2,k}$ and $\xi_{3,k}, k = 1, 2, \cdots, n$ are independent Gaussian random variables $N(0, 1)$, and the step size is $\Delta t = 0.01$.

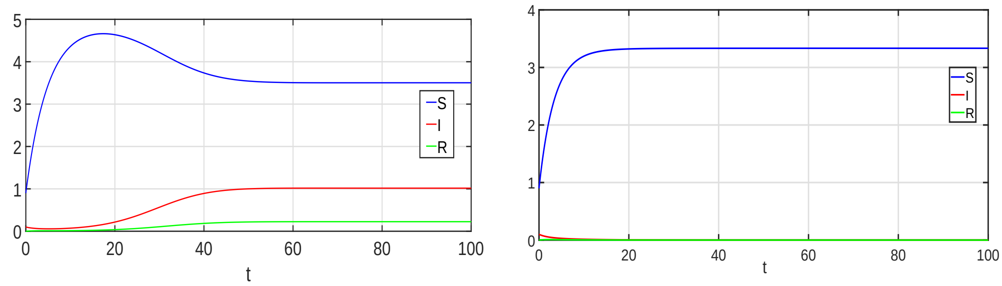
4.1. Extinction and stationary distribution

Now, we discuss the influence of the natural death rate μ. We choose parameters in (4.2); when $R_0 = 1.4286 > 1$, the equilibrium

$$E^* = (S^*, I^*, R^*) = (3.50362, 1.01641, 0.225869)$$

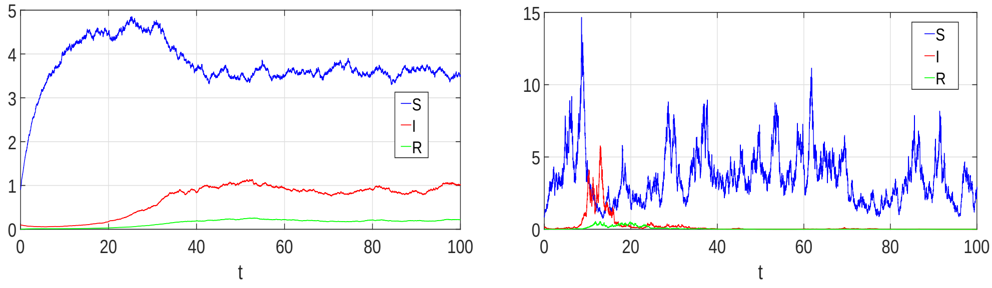
is stable, which means the disease is persistent (Figure 1(a)). When μ increases from $\mu = 0.2$ to $\mu = 0.3$ and other parameters in (4.2), has $R_0 = 0.8571 < 1$, it leads equilibrium $E_0 = (\frac{\Lambda}{\mu}, 0, 0) = (3.33, 0, 0)$ to be stable, that is, S tends to $\frac{\Lambda}{\mu} = 3.33$, I goes to 0, and R tends to 0 as t increases. This implies that the disease dies out (Figure 1(b)). From Figure 1, one knows that a high μ can lead to disease extinction.

Next, we discuss the stationary distribution and extinction of Model (4.1). One chooses the overall intensity of environmental fluctuations $\varepsilon = 0.05$. From (3.2) and (3.11), one can easy calculate $R_s = 1.4263 > 1$ and $\Psi = 0.0155 < \min\{6.3942, 0.8385, 0.0217\} = 0.0217$. By Theorem 3.3, Model (4.1) exists as stationary distribution, which means that disease is persistent (Figure 2(a)). One increases the overall noise intensity from $\varepsilon = 0.05$ to $\varepsilon = 0.75$; from (3.2), calculate that $R_s = 0.9143 < 1$ and $\frac{\varepsilon^2\sigma_2^2}{2} = 0.18 < \mu = 0.2$. By Theorem 3.2, S tends to $\frac{\Lambda}{\mu} = 5$, I tends to 0, R tends to 0 as t increases, and to disease extinction (Figure 2(b)). From Figure 2, we know that the high noise intensity can lead to disease extinction. From Figure 1, and Figure 2, one knows that even if a deterministic model predicts that a disease will spread, it may actually disappear due to stochastic factors in a noisy environment.



(a) (b)

Figure 1. Impact of the natural death rate on the disease dynamics. (a) For $\mu = 0.2 (R_0 > 1)$, the disease persists at an endemic equilibrium. (b) For $\mu = 0.3 (R_0 < 1)$, the disease dies out, leaving only the susceptible population. Other parameters are taken as (4.2).



(a) (b)

Figure 2. Impact of the overall intensity of environmental fluctuations ε on disease dynamics. (a) For $\varepsilon = 0.05(R_s > 1)$, the model exhibits a stationary distribution. (b) For $\varepsilon = 0.75(R_s < 1)$, the disease dies out, leaving only the susceptible population. Other parameters are taken as (4.2).

4.2. Stochastic sensitivity

Now, one presents a noise-induced transition from persistence to when it dies out. From Eq (3.16) and the parameters in (4.2), one has the following stochastic sensitivity matrix:

$$W = \begin{bmatrix} 10.28450 & -3.20838 & -0.916167 \\ -3.20838 & 6.67913 & 1.27549 \\ -0.916167 & 1.27549 & 0.311217 \end{bmatrix}.$$

Then, the eigenvalues of matrix W are as follows:

$$\mu_1 = 12.334, \mu_2 = 4.88552, \mu_3 = 0.0553449. \tag{4.4}$$

It follows that the normalized eigenvectors of matrix W are

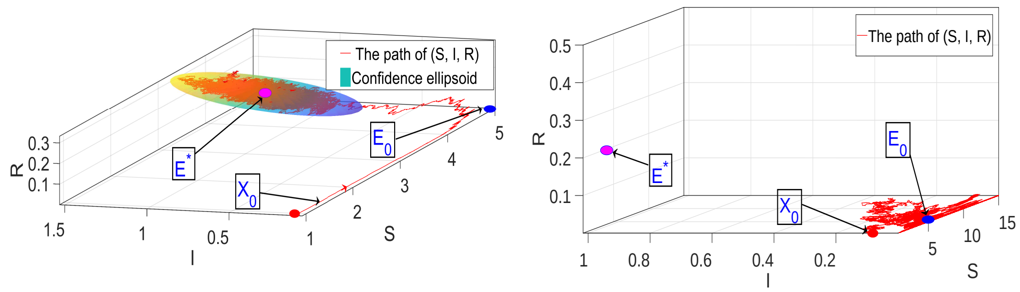
$$\begin{aligned} v_1 &= (0.851815, -0.510141, -0.119031)^\top, \\ v_2 &= (0.522748, 0.842483, 0.130217)^\top, \\ v_3 &= (0.0338526, -0.173144, 0.984314)^\top, \end{aligned} \tag{4.5}$$

respectively. When $P(k) = 0.998$, by Eq (3.18), one has $k(P) = 14.7955$; then, one has the confidence ellipsoid in the following:

$$\frac{\langle X - E^*, v_1\rangle^2}{\mu_1} + \frac{\langle X - E^*, v_2\rangle^2}{\mu_2} + \frac{\langle X - E^*, v_3\rangle^2}{\mu_3} = 14.7955\varepsilon^2, \tag{4.6}$$

where μ_1, μ_2, and μ_3 are given in (4.4), and v_1, v_2, and v_3 are given in (4.5). Since μ_1 is the largest eigenvalue, the confidence ellipsoid stretches along the direction of the eigenvector v_1 on the longest axis. This means that the stochastic sensitivity is highest in the direction of v_1, and even small noises can cause significant deviations in the model state in this direction. The longest axis along the direction of v_1 is roughly an increase in S accompanied by a decrease in I and R, or vice versa.

When the overall level of the environmental fluctuation is $\varepsilon = 0.05$, by Theorem 3.3, there exists a stationary distribution in Model (4.1) there exists a stationary distribution. Meanwhile, from (4.6), one can obtain a confidence ellipsoid. From Figure 3 (a), we know that the path of (S, I, R) of the stochastic model (4.1) randomly oscillates near the equilibrium E^* in the first octant. The confidence ellipsoid is helpful for us to estimate the equilibrium region of the stochastic model. The confidence ellipsoid provides an expected range of fluctuations for the monitoring model. If the observed (S, I, R) data points frequently fall outside the confidence ellipsoid, then this may indicate that the model parameters need to be adjusted (e.g., the actual noise intensity is greater than assumed) or new factors that have not been considered by the model (such as super spreaders, virus mutations, etc.) may arise, which requires the initiation of warnings and investigations. The confidence ellipsoid provides a quantitative benchmark to evaluate the effectiveness of the control measures. If an intervention measure (such as increasing vaccination rates) is implemented and the observed confidence ellipsoid significantly shrinks, especially in the fluctuation direction of infected individuals, then this provides strong statistical evidence for the effectiveness of the measure.



(a) (b)

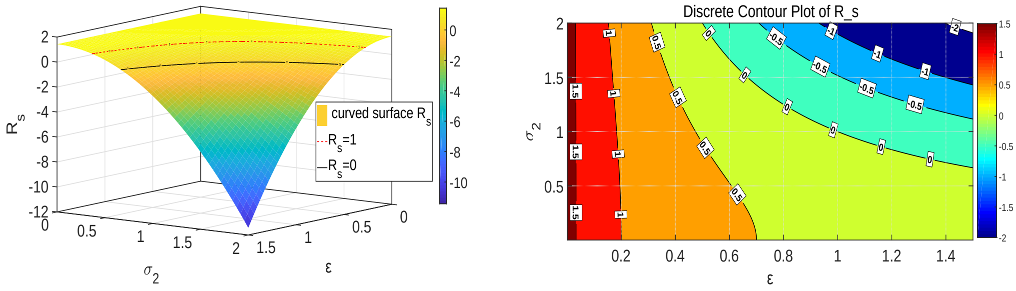
Figure 3. (a) The path of (S, I, R) and the confidence ellipsoid of Model (4.1) with $\varepsilon = 0.05$; (b) The path of (S, I, R) with $\varepsilon = 0.75$. Other parameters are taken as (4.2).

Furthermore, we used the Milstein method in Higham [33] with a design step size of $\Delta t = 0.01$ and a total time $t = T = 1000$ without seed simulation. We simulated 20,000 trajectories $(S(t), I(t), R(t))$ and collected the final states $(S(T), I(T), R(T))$ to obtain the scattered points contained within the ellipsoid (Figure 5(a)). Additionally, we quantified the relationship between covariance error and ε (Figure 5(b)). We drew marginal density plots for S, I, and R, separately (Figures 6 and 7). Additionally, the calculated proportion of points which fall into the ellipsoid is 0.9963, which is very close to the confidence probability $P = 0.998$, thus indicating that the local approximation is reliable.

Next, we increase the overall level of the environmental fluctuation from $\varepsilon = 0.05$ to $\varepsilon = 0.75$, one has $R_s = 0.9806 < 1$. By Theorem 3.2, the path of (S, I, R) will almost surely tend to the equilibrium $E_0 = (\frac{\Lambda}{\mu}, 0, 0) = (5, 0, 0)$. This transformation is called a noise-induced transition of the stochastic model (4.1) from the attraction $\{(S, I, R)|(S, I, R) \in \mathbb{R}^3_+\}$ of equilibrium E^* to the attraction $\{(S, I, R)|S > 0, I = R = 0\}$ of equilibrium E_0 (Figure 3 (b)).

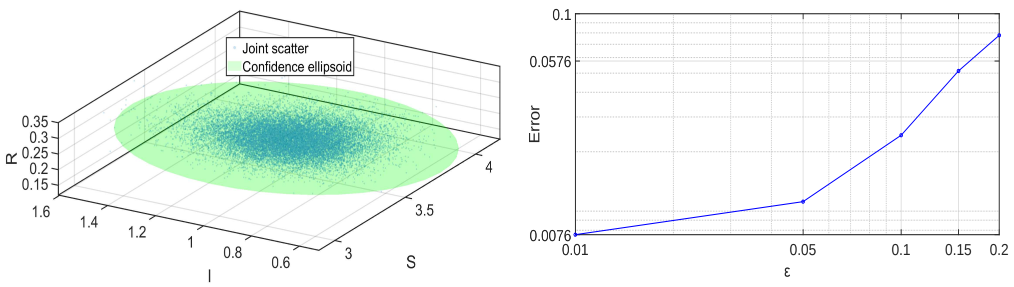
4.3. The influence of σ_2 and ε

To investigate the impact of the noise sensitivity σ_2 and the overall intensity ε of environmental fluctuations on the stochastic critical value R_s of disease transmission, one uses the parameters are taken as (4.2) to plot the curved surface and discrete contour of R_s with varying ε and σ_2, separately. From Figure 4(a) and (b), one can see that the larger ε and σ_2, the smaller R_s. This implies that the larger overall intensity ε of the environmental fluctuations and the noise sensitivity σ_2 can suppress the disease outbreak in a noisy environment.



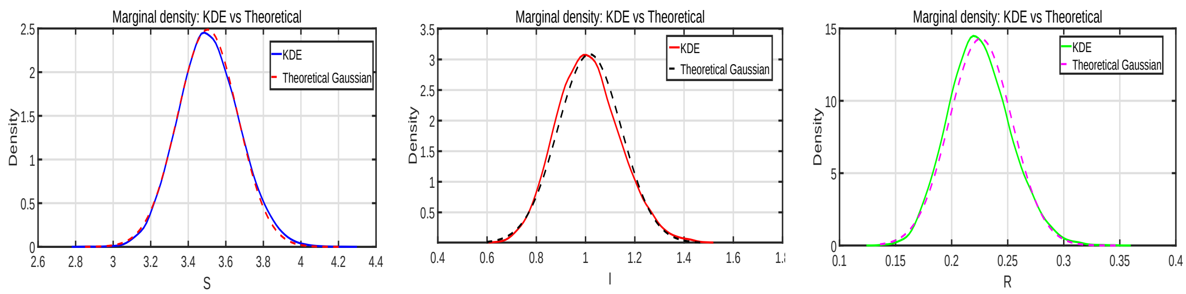
(a) (b)

Figure 4. Impact of the overall intensity of the environmental fluctuations ε and the noise sensitivity σ_2 on disease dynamics. (a) Curved surface of R_s with varying ε and σ_2; (b) Discrete contour plot of R_s with varying ε and σ_2. Other parameters are taken as (4.2).



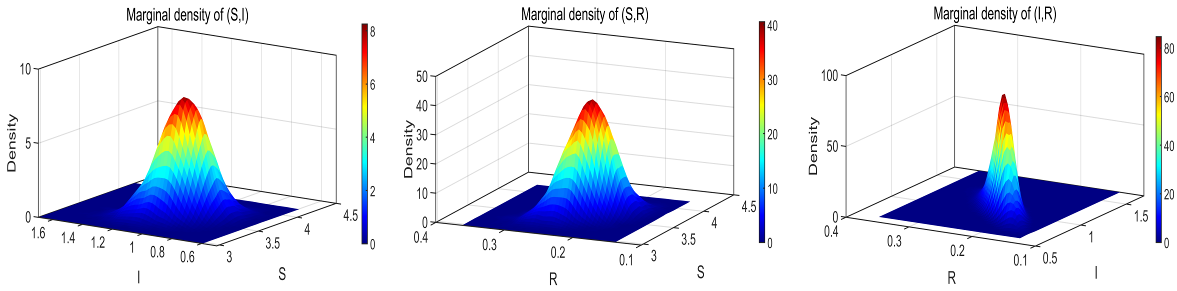
(a) (b)

Figure 5. (a) The joint scatter with a confidence ellipsoid overlay of the stochastic model (4.1) from 20,000 trajectories at time $t = 1000$ with $\varepsilon = 0.05$; (b) The covariance error vs ε. Other parameters are taken as (4.2).



(a) (b) (c)

Figure 6. Marginal density: Kernel Density Estimation (KDE) vs theoretical Guassian for $S(t)$, $I(t)$, and $R(t)$ of the stochastic model (4.1) from 20,000 trajectories at time $t = 1000$ with $\varepsilon = 0.05$. Other parameters are taken as (4.2).



(a) (b) (c)

Figure 7. The 3D graph of the joint two-dimensional densities of the stochastic model (4.1) from 20,000 trajectories at time $t = 1000$ with $\varepsilon = 0.05$. Other parameters are taken as (4.2).

5. Conclusions

This paper focused on the influence of the noise sensitivity and the overall intensity of environmental noise on the threshold for disease transmission, and studied a stochastic SIRS model by perturbing the natural death rate. A stochastic threshold R_s which determined the extinction or persistence of the disease was obtained.

More concretely, if $R_s < 1$, then disease will die out. If $R_s > 1$, then the model undergoes a stationary distribution with a mild condition, which means that the disease is persistent. Furthermore, a Gaussian approximation of the stationary distribution was given by the stochastic sensitivity technique. For a visual description of the distribution, a confidence ellipsoid was presented by the visualization geometric method of the confidence domains. The confidence ellipsoid can be used to estimate the equilibrium region of the stochastic model. In biological mathematics, the confidence ellipsoid describes the typical wave patterns and amplitudes of a system around an equilibrium point under noise driving. In terms of epidemiological applications, a risk map helps us move from binary judgments of whether a disease will spread to refined risk assessments of the extent and the probability of disease fluctuations, thus providing support for more scientific and robust public health decisions.

In a noisy environment, even if a deterministic model predicts that a disease will spread, then it may actually disappear due to stochastic factors. Our results indicate that the overall intensity of environmental fluctuations and the noise sensitivity of infected individuals are equally important in influencing the disease transmission threshold. Moreover, the numerical results support the obtained theoretical results.

Authors contributions

Jiabing Huang: Writing-original draft, software, methodology; Jierui Du: Supervision, funding acquisition; Haiye Liang: Editing, formal analysis. All authors have read and approved the final version of the manuscript for publication.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors declare there is no conflicts of interest.

References

1. D. Wernli, L. Böttcher, F. Vanackere, Y. Kaspiarovich, M. Masood, N. Levrat, Understanding and governing global systemic crises in the 21st century: A complexity perspective, *Glob. Policy*, **14** (2023), 207–228. https://doi.org/10.1111/1758-5899.13192
2. *Coronavirus disease (COVID-19) pandemic*, World Health Organization (WHO), 2025. Available from: https://www.who.int/emergencies/diseases/novel-coronavirus-2019.
3. W. O. Kermack, A. G. McKendrick, Contributions to the mathematical theory of epidemics-I, *Proc. R. Soc. Lond. Ser. A*, **115** (1927), 701–721. https://doi.org/10.1007/bf02464423
4. Q. Yang, J. Huang, Dynamics analysis of an 11-dimensional multiscale COVID-19 model with interval parameters, *Int. J. Bifurcation Chaos*, **33** (2023), 2350140. https://doi.org/10.1142/S0218127423501407
5. Q. Yang, J. Huang, A stochastic multi-scale COVID-19 model with interval parameters, *J. Appl. Anal. Comput.*, **14** (2024), 515–542. https://doi.org/10.11948/20230298
6. J. Jiao, Z. Liu, S. Cai, Dynamics of an SEIR model with infectivity in incubation period and homestead-isolation on the susceptible, *Appl. Math. Lett.*, **107** (2020), 106442. https://doi.org/10.1016/j.aml.2020.106442
7. Y. Li, Z. Wei, Dynamics and optimal control of a stochastic coronavirus (COVID-19) epidemic model with diffusion, *Nonlin. Dyn.*, **109** (2022), 91–120. https://doi.org/10.1007/s11071-021-06998-9
8. Y. Wei, Q. Yang, G. Li, Dynamics of the stochastically perturbed Heroin epidemic model under non-degenerate noises, *Physica A*, **526** (2019), 120914. https://doi.org/10.1016/j.physa.2019.04.150
9. D. Li, S. Liu, J. Cui, Threshold dynamics and ergodicity of an SIRS epidemic model with Markovian switching, *J. Differ. Equ.*, **263** (2017), 8873–8915. https://doi.org/10.1016/j.jde.2017.08.066
10. S. Tang, J. Liang, Y. Xiao, A. Robert. Cheke, Sliding bifurcations of filippov two stage pest control models with economic thresholds, *SIAM J. Appl. Math.*, **72** (2012), 1061–1080. https://doi.org/10.1137/110847020
11. Y. Wei, J. Zhan, J. Guo, Asymptotic behaviors of a heroin epidemic model with nonlinear incidence rate influenced by stochastic perturbations, *J. Appl. Anal. Comput.*, **14** (2024), 1060–1077. https://doi.org/10.11948/20230323

12. P. Zhu, Y. Wei, The dynamics of a stochastic SEI model with standard incidence and infectivity in incubation period, *AIMS Math.*, **7** (2022), 18218–18238. https://doi.org/10.3934/math.20221002

13. Q. Liu, Q. Chen, Analysis of the deterministic and stochastic SIRS epidemic models with nonlinear incidence, *Physica A*, **428** (2015), 140–153. https://doi.org/10.1016/j.physa.2015.01.075

14. Y. Cai, Y. Kang, M. Banerjee, W. Wang, A stochastic SIRS epidemic model with infectious force under intervention strategies, *J. Differ. Equ.*, **259** (2015), 7463–7502. https://doi.org/10.1016/j.jde.2015.08.024

15. Y. Xu, X. Sun, H. Hu, Extinction and stationary distribution of a stochastic SIQR epidemic model with demographics and non-monotone incidence rate on scale-free networks, *J. Appl. Math. Comput.*, **68** (2022), 3367–3395. https://doi.org/10.1007/s12190-021-01645-3

16. Y. Cai, Y. Kang, W. Wang, A stochastic SIRS epidemic model with nonlinear incidence rate, *Appl. Math. Comput.*, **305** (2017), 221–240. https://doi.org/10.1016/j.amc.2017.02.003

17. A. Lahrouz, L. Omari, Extinction and stationary distribution of a stochastic SIRS epidemic model, *Statist. Probab. Lett.*, **83** (2013), 960–968. https://doi.org/10.1016/j.spl.2012.12.021

18. M. J. Keeling, P. Rohani, *Modeling infectious diseases in humans and animals*, Princeton: Princeton University Press, 2008. http://dx.doi.org/10.1086/591197

19. S. Funk, M. Salathé, V. A. Jansen, Modelling the influence of human behaviour on the spread of infectious, *J. R. Soc. Interface*, **50** (2010), 1247–1256. https://doi.org/10.1098/rsif.2010.0142

20. A. L. Lloyd, The dependence of viral parameter estimates on the assumed viral life cycle: limitations of studies of viral load data, *Proc. R. Soc. Lond. B. Biol. Sci.*, **268** (2001), 847–854. https://doi.org/10.1098/rspb.2000.1572

21. H. E. Davis, G. S. Assaf, L. McCorkell, H. Wei, R. J. Lowa, Y. Re'em, et al., Characterizing long COVID in an international cohort: 7 months of symptoms and their impact, *EClinicalMedicine*, **38** (2021), 101019. https://doi.org/10.1016/j.eclinm.2021.101019

22. A. W. Peters, K. S. Chawla, Z. A. Turnbull, Transforming ORs into ICUs, *N. Engl. J. Med.*, **382** (2020), e52. https://doi.org/10.1056/NEJMc2010853

23. P. Brodin, M. M. Davis, COVID-19 and long-term health consequences, *Nat. Med.*, **28** (2022), 2406–2408. https://doi.org/0.1001/jama.2020.19719

24. G. Albano, V. Giorno, G. Prez-Romero, F. de Asis Torres-Ruiz, Inference on a stochastic SIR model including growth curves, *Comput. Stat. Data Anal.*, **212** (2025), 108231. https://doi.org/10.1016/j.csda.2025.108231

25. C. Ji, D. Jiang, Threshold behaviour of a stochastic SIR model. *Appl. Math. Model.,* **38** (2014), 5067–5079. https://doi.org/10.1016/j.apm.2014.03.037

26. E. Bernardi, E, A. Lanconelli, A note about the invariance of the basic reproduction number for stochastically perturbed SIS models, *Stud. Appl. Math.*, **148** (2022), 1543–1562. https://doi.org/10.1111/sapm.12483

27. A. Lanconelli, M. Mori, Itôvs Stratonovich stochastic SIR models, *Appl. Math Lett.*, **134** (2022), 108368. https://doi.org/10.1016/j.aml.2022.108368

28. A. Lanconelli, B. T. Percin, On a new method for the stochastic perturbation of the disease transmission coefficient in SIS models, *Appl. Math Comput.*, **413** (2022), 126600. https://doi.org/10.1016/j.amc.2021.126600

29. X. Mao, *Stochastic differential equations and applications*, 2 Eds., Chichester: Woodhead Publishing, 2008. https://doi.org/10.1533/9780857099402

30. N. Chitnis, J. M. Hyman, J. M. Cushing, Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model, *Bull. Math. Biol.*, **70** (2008), 1272–1296. https://doi.org/10.1007/s11538-008-9299-0

31. R. Z. Has'miniskii, *Stochastic stability of differential equations*, 2 Eds., Berlin: Springer, 1980. http://dx.doi.org/10.1007/978-3-642-23280-0

32. I. Bashkirtseva, L. Ryashko, T. Ryazanova, Stochastic sensitivity technique in a persistence analysis of randomly forced population systems with multiple trophic levels, *Math. Biosci.*, **293** (2017), 38–45. https://doi.org/10.1007/s11538-008-9299-0

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

34. D. Xiao, S. Ruan, Global analysis of an epidemic model with nonmonotone incidence rate, *Math. Biosci.*, **208** (2007), 419–429. https://doi.org/10.1016/j.mbs.2006.09.025

35. Y. Zhao, D. Jiang, The threshold of a stochastic SIS epidemic model with vaccination, *Appl. Math. Comput.*, **243** (2014), 718–727. https://doi.org/10.1016/j.amc.2014.05.124

Appendix

(I). (Proof of Lemma 2.1): Denote $N = S + I + R$, and define function

$$\Phi(N) = (1 + N)^c, \tag{A.1}$$

here $c > 0$ represents a constant. Using Itô's formula, one obtains

$$\mathrm{d}\Phi(N) = \mathcal{L}\Phi(N)\mathrm{d}t + c(1 + N)^{c-1}\psi_1(t),$$

where

$$\begin{aligned} \mathcal{L}\Phi(N) &= c(1 + N)^{c-1}[\Lambda - \mu S - (\mu + \epsilon)I - \mu R] + \frac{c(c-1)\varepsilon^2}{2}(\sigma_1^2 S^2 + \sigma_2^2 I^2 + \sigma_3^2 R^2)(1 + N)^{c-2}, \\ \psi_1(t) &= -\varepsilon\sigma_1 S(t)\mathrm{d}B_1(t) - \varepsilon\sigma_2 I(t)\mathrm{d}B_2(t) - \varepsilon\sigma_3 R(t)\mathrm{d}B_3(t). \end{aligned}$$

Let $\psi_2(t) = \varepsilon^2[\sigma_1^2 S^2(t) + \sigma_2^2 I^2(t) + \sigma_3^2 R^2(t)]$, one has

$$\begin{aligned} \mathcal{L}\Phi(N) &= c(1 + N)^{c-2}\left\{(1 + N)[\Lambda - \mu S - (\mu + \epsilon)I - \mu R] + \frac{c-1}{2}\psi_2(t)\right\} \\ &\leq c(1 + N)^{c-2}\left\{(1 + N)(\Lambda - \mu N) + \frac{c-1}{2}\psi_2(t)\right\} \end{aligned}$$

$$
\begin{aligned}
&\leq c(1+N)^{c-2}\left\{(1+N)(\Lambda-\mu N)+\varepsilon^2(\frac{c-1}{2}\vee 0)(\sigma_1^2\vee\sigma_2^2\vee\sigma_3^2)N^2\right\}\\
&= c(1+N)^{c-2}\left\{-\left[\mu-\varepsilon^2(\frac{c-1}{2}\vee 0)(\sigma_1^2\vee\sigma_2^2\vee\sigma_3^2)\right]N^2\right.\\
&\quad\left.+(\Lambda-\mu)N+\Lambda\right\}.
\end{aligned}
$$

Selecte $c>0$ such that

$$\mu-\varepsilon^2(\frac{c-1}{2}\vee 0)(\sigma_1^2\vee\sigma_2^2\vee\sigma_3^2):=\Upsilon>0.$$

Then, one gets

$$\mathcal{L}\Phi(N)\leq c\{-\Upsilon N^2+(\Lambda-\mu)N+\Lambda\}(1+N)^{c-2}.$$

hence

$$\mathrm{d}\Phi(N)\leq c\{-\Upsilon N^2+(\Lambda-\mu)N+\Lambda\}(1+N(t))^{c-2}\mathrm{d}t+c(1+N)^{c-1}\psi_1(t). \tag{A.2}$$

For $0<k<c\Upsilon$, one has

$$\mathrm{d}[e^{kt}\Phi(N(t))]=\mathcal{L}[e^{kt}\Phi(N(t))]\mathrm{d}t+e^{kt}c(1+N(t))^{c-1}\psi_1(t).$$

Thus,

$$\mathrm{E}[e^{kt}\Phi(N(t))]=\Phi(N(0))+\mathrm{E}\int_0^t\mathcal{L}[e^{ks}\Phi(N(s))]\mathrm{d}s, \tag{A.3}$$

where

$$
\begin{aligned}
\mathcal{L}[e^{kt}\Phi(N)] &= ke^{kt}\Phi(N)+e^{kt}\mathcal{L}\Phi(N)\\
&\leq ce^{kt}(1+N)^{c-2}\left\{\frac{k}{c}(1+N)^2-\Upsilon N^2+(\Lambda-\mu)N+\Lambda\right\}\\
&= c\left[-(\Upsilon-\frac{k}{c})N^2+(\Lambda-\mu+\frac{2k}{c})N+(\Lambda+\frac{k}{c})\right]e^{kt}(1+N)^{c-2}\\
&\leq ce^{kt}H_1,
\end{aligned}
$$

here,

$$H_1:=\sup_{N\in\mathbb{R}_+}(1+N)^{c-2}[-(\lambda-\frac{k}{c})N^2+(\Lambda-\mu+\frac{2k}{c})N+(\Lambda+\frac{k}{c})]+1.$$

Then, from (A.3), one has

$$\mathrm{E}[e^{kt}(1+N(t))^c]\leq(1+N(0))^c+\frac{cH_1}{k}e^{kt}.$$

It follows that

$$\limsup_{t\to\infty}\mathrm{E}[(1+N(t))^c]\leq\frac{cH_1}{k}.\ a.s.$$

Combining the continuity of $\Phi(t)$, existence a constant $M_0 > 0$, has

$$\mathrm{E}[(1+N(t))^c] \le M_0, t \ge 0. \tag{A.4}$$

Form (A.2), for any $\eta > 0$ small enough, one has

$$\mathrm{E}\left[\sup_{k\eta\le t\le(k+1)\eta}(1+N(t))^c\right] \le \mathrm{E}[(1+N(k\eta))^c] + M_1 + M_2 \le M_0 + M_1 + M_2,$$

where

$$\begin{aligned} M_1 &= \mathrm{E}\left[\sup_{k\eta\le t\le(k+1)\eta}\left|\int_{k\eta}^t [-\Upsilon N^2(s) + (\Lambda-\mu)N(s) + \Lambda]c(1+N(s))^{c-2}\mathrm{d}s\right|\right] \\ &\le c_1\mathrm{E}\left[\sup_{k\eta\le t\le(k+1)\eta}\left|\int_{k\eta}^t (1+N(s))^c\mathrm{d}s\right|\right] \le c_1\mathrm{E}\left[\int_{k\eta}^{(k+1)\eta}(1+N(s))^c\mathrm{d}s\right] \\ &\le c_1\eta\mathrm{E}\left[\sup_{k\eta\le t\le(k+1)\eta}(1+N(t))^c\right]. \\ M_2 &= \mathrm{E}\left[\sup_{k\eta\le t\le(k+1)\eta}\left|\int_{k\eta}^t c(1+N(s))^{c-1}\psi_1(s)\right|\right] \\ &\le \sqrt{32}\mathrm{E}\left[\int_{k\eta}^{(k+1)\eta} c^2(1+N(s))^{2(c-1)}\psi_2(s)\mathrm{d}s\right]^{\frac{1}{2}} \\ &\le \sqrt{32}c\varepsilon(\sigma_1^2\vee\sigma_2^2\vee\sigma_3^2)^{\frac{1}{2}}\eta^{\frac{1}{2}}\mathrm{E}\left[\sup_{k\eta\le t\le(k+1)\eta}(1+N)^c\right]. \end{aligned}$$

Apply Burkholder-Davis-Gundy inequality [29, 35], one obtains,

$$\mathrm{E}\left[\sup_{k\eta\le t\le(k+1)\eta}(1+N)^c\right] \le \left[c_1\eta + \sqrt{32}c\varepsilon(\sigma_1^2\vee\sigma_2^2\vee\sigma_3^2)^{\frac{1}{2}}\eta^{\frac{1}{2}}\right]\mathrm{E}\left[\sup_{k\eta\le t\le(k+1)\eta}(1+N(t))^c\right] + \mathrm{E}\left[(1+N(k\eta))^c\right].$$

Choose $\eta > 0$, such that $c_1\eta + \sqrt{32}c\varepsilon(\sigma_1^2\vee\sigma_2^2\vee\sigma_3^2)^{\frac{1}{2}}\eta^{\frac{1}{2}} \le \frac{1}{2}$. Then, one has

$$\mathrm{E}\left[\sup_{k\eta\le t\le(k+1)\eta}(1+N)^c\right] \le 2\mathrm{E}\left[(1+N(k\eta))^c\right] \le 2M_0,$$

Let $\epsilon_\Phi > 0$. Using Chebyshev inequality, one gets

$$\mathrm{P}\left\{\sup_{k\eta\le t\le(k+1)\eta}(1+N(t))^c > (k\eta)^{1+\epsilon_\Phi}\right\} \le \frac{\mathrm{E}\left[\sup_{k\eta\le t\le(k+1)\eta}(1+N(t))^c\right]}{(k\eta)^{1+\epsilon_\Phi}}\frac{2M_0}{(k\eta)^{1+\epsilon_\Phi}}, k = 1, 2, \cdots.$$

By Borel-Cantelli lemma [29], one has

$$\sup_{k\eta\le t\le(k+1)\eta}(1+N(t))^c \le (k\eta)^{1+\epsilon_\Phi}, a.s. \tag{A.5}$$

Then, exist a $k_1(\omega)$, s.t. inequality (A.5) holds for $\omega\in\Omega$, $k\ge k_1$, a.s.
Hence, when $k \ge k_1$ and $k\eta \le t \le (k+1)\eta$, one obtains

$$\frac{\log(1+N)^c}{\log t} \le \frac{(1+\epsilon_N)\log(k\eta)}{\log(k\eta)} = 1 + \epsilon_N.$$

Then,

$$\limsup_{t\to\infty}\frac{\log(1+N)^c}{\log t}\le 1+\epsilon_\Phi.$$

Let $\epsilon_\Phi\to 0$, one has

$$\limsup_{t\to\infty}\frac{\log(1+N)^c}{\log t}\le 1.$$

For $1<c<1+2\mu/[\varepsilon^2(\sigma_1^2\vee\sigma_2^2\vee\sigma_3^2)]$, one gets $\mu>(c-1)\varepsilon^2(\sigma_1^2\vee\sigma_2^2\vee\sigma_3^2)/2$, yields,

$$\limsup_{t\to\infty}\frac{\log N(t)}{\log t}\le\limsup_{t\to\infty}\frac{\log(1+N(t))}{\log t}\le\frac{1}{c}\ a.s.$$

Then, for $0<\iota_1<1-\frac{1}{c}$, has constant $T_1=T_1(\omega)$ and set Ω_{ι_1} s.t. $\mathrm{P}\{\Omega_\iota\}\ge 1-\iota_1$. Further, for $t\ge T_1,\omega\in\Omega_\iota$, one has

$$\log N(t)\le(\frac{1}{c}+\iota_1)\log t.$$

Thus,

$$\limsup_{t\to\infty}\frac{N(t)}{t}\le\limsup_{t\to\infty}\frac{t^{\frac{1}{c}+\iota_1}}{t}=0,$$

combining the positivity of solution, one has

$$\lim_{t\to\infty}\frac{N}{t}=\lim_{t\to\infty}\frac{S+I+R}{t}=0. \tag{A.6}$$

From (A.6), one obtains

$$\lim_{t\to\infty}\frac{S(t)}{t}=\lim_{t\to\infty}\frac{I(t)}{t}=\lim_{t\to\infty}\frac{R(t)}{t}=0.$$

(II). (Proof of Lemma 2.2):

Denote $Z_1=\int_0^t S(s)\mathrm{d}B_1(s)$, $Z_2=\int_0^t I(s)\mathrm{d}B_2(s)$, $Z_3=\int_0^t R(s)\mathrm{d}B_3(s)$, $2<c<1+\frac{2\mu}{\left[\varepsilon^2(\sigma_1^2\vee\sigma_2^2\vee\sigma_3^2)\right]}$. From Burkhold-Davis-Gundy inequality [29] and (A.4), has a number H_c, s.t.

$$\begin{aligned}\mathrm{E}\left[\sup_{0\le s\le t}|X(s)|^c\right] &\le H_c\mathrm{E}\left[\int_0^t S^2(s)\mathrm{d}s\right]^{\frac{c}{2}}\le H_c t^{\frac{c}{2}}\mathrm{E}\left[\sup_{0\le s\le t}S^2(s)\right]^{\frac{c}{2}}\\ &= H_c t^{\frac{c}{2}}\mathrm{E}\left[\sup_{0\le s\le t}S^c(s)\right]^{\frac{c}{2}}\le 2M_0H_c t^{\frac{c}{2}}.\end{aligned}$$

Let ϵ_{Z_1} is any constant, apply the Doob's inequality [29], one obtains

$$\mathrm{P}\left\{\omega:\sup_{k\eta\le t\le(k+1)\eta}|X(t)|^c>(k\eta)^{1+\epsilon_{Z_1}+\frac{c}{2}}\right\}\ \le\ \frac{\mathrm{E}[|X((k+1)\eta)|^c]}{(k\eta)^{1+\epsilon_{Z_1}+\frac{c}{2}}}\le\frac{2^{1+\frac{c}{2}}M_0H_c}{(k\eta)^{1+\epsilon_{Z_1}}}.$$

Using Borel-Cantelli lemma [29], we have

$$\sup_{k\eta\le t\le(k+1)\eta} |Z_1(t)|^c \le (k\eta)^{1+\epsilon_{Z_1}+\frac{c}{2}}, a.s. \tag{A.7}$$

Then there has a $k_2(\omega) > 0$, such that inequality $(A.7)$ holds, when $k \ge k_2$. Thus, when $k \ge k_2$ and $k\eta \le t \le (k+1)\eta$, yields

$$\frac{\log|X(t)|^c}{\log t} \le \frac{(1+\epsilon_{Z_1}+\frac{c}{2})\log(k\eta)}{\log(k\eta)} = 1+\epsilon_{Z_1}+\frac{c}{2}.$$

Hence,

$$\limsup_{t\to\infty} \frac{\log|Z_1(t)|^c}{\log t} \le \frac{1+\epsilon_X+\frac{c}{2}}{c}.$$

let $\epsilon_{Z_1} \to 0$, then

$$\limsup_{t\to\infty} \frac{\log|Z_1(t)|^c}{\log t} \le \frac{1}{2}+\frac{1}{c}.$$

Thus, for any $0 < \iota_2 < \frac{1}{2}-\frac{1}{c}$, has a constant $T_2 = T_2(\omega) > 0$ and set Ω_{ι_2}, s.t.

$$\mathrm{P}\{\Omega_\eta\} \ge 1-\iota_2,\ t \ge T_2, \omega \in \Omega_{\iota_2}.$$

Then,

$$\log|Z_1(t)| \le (1/2+1/c+\iota_2)\log t,$$

and

$$\limsup_{t\to\infty} \frac{Z_1(t)}{t} \le \limsup_{t\to\infty} \frac{t^{1/2+1/c+\iota_2}}{t} = 0.$$

Since

$$\liminf_{t\to\infty} \frac{|Z_1(t)|}{t} \ge 0,$$

it leads to $\lim_{t\to\infty}\frac{|Z_1(t)|}{t} = 0$. Hence, $\lim_{t\to\infty}\frac{Z_1(t)}{t} = 0$. Similarly, we obtain $\lim_{t\to\infty}\frac{Z_2(t)}{t} = \lim_{t\to\infty}\frac{Z_3(t)}{t} = 0$ a.s.