Applied Mathematics and Computation 329 (2018) 210–226

Contents lists available at ScienceDirect

Applied Mathematics and Computation

journal homepage: www.elsevier.com/locate/amc

Environmental variability in a stochastic epidemic model

Yongli Cai[a], Jianjun Jiao[b], Zhanji Gui[c], Yuting Liu[a,d], Weiming Wang[a,*]

[a] School of Mathematical Science, Huaiyin Normal University, Huaian 223300, PR China
[b] School of Mathematical and Statistics, Guizhou University of Finance and Economics, Guiyang 550025, PR China
[c] Software Department, Hainan College of Software Technology, Qionghai 571400, PR China
[d] College of Mathematics and Information Science, Wenzhou University, Wenzhou 325035, PR China

ARTICLE INFO

Keywords:
Environment fluctuations
Mean-reverting
Intensity of volatility
Speed of reversion
Stationary distribution

ABSTRACT

In this paper, we investigate the stochastic dynamics of a simple epidemic model incorporating the mean-reverting Ornstein–Uhlenbeck process analytically and numerically. We define two threshold parameters, the stochastic demographic reproduction number $\mathcal{R}_d^s$ and the stochastic basic reproduction number $\mathcal{R}_0^s$, to utilize in identifying the stochastic extinction and persistence of the disease. We find that the stochastic disease dynamics can be determined by the environment fluctuations which measured by the intensity of volatility and the speed of reversion: the larger intensity of volatility or the smaller speed of reversion can suppress the outbreak of the disease, the smaller intensity of volatility or the the higher speed of reversion can enhance the outbreak of the disease. Furthermore, via numerical simulations, we find that the stochastic model has an endemic stationary distribution which leads to the stochastic persistence of the disease. Our results show that mean-reverting process is a well-established way of introducing stochastic environmental noise into biologically realistic population dynamic models.

1. Introduction

Epidemiological models, which can be identified as the disease transmission in the populations, have been revealed as a powerful tool to analyze the spread and control the infectious diseases qualitatively and quantitatively, as the research results are helpful to predict the developing tendency of infectious diseases, to determine the key factors of the spread of infectious disease and to seek the optimum strategies of preventing and controlling the spread of infectious diseases [1–7].

In recent years, a number of epidemic models have been formulated to describe the impact of environmental noise on the dynamics of infectious diseases [8–15]. And in reality, if the environment is randomly varying, the population is subject to a continuous spectrum of disturbances [8]. The environmental fluctuations may involve the variations of factors such as climate, habitats, health habits, medical quality and so on, which may affect the natural birth rate, death rate and so on [16]. Especially, for human diseases, the nature of epidemic growth and spread is inherently random due to the unpredictability of person-to-person contacts [17] and population is subject to a continuous spectrum of disturbances [8]. Hence the variability and randomness of the environment are fed through to the state of the epidemic [18]. In the view of this point, stochastic differential equations (SDE) could be a more appropriate way of modeling disease spreading in many circumstances [10].

* Corresponding author.
E-mail addresses: caiyongli@hytc.edu.cn (Y. Cai), jiaojianjun05@126.com (J. Jiao), zhanjigui@sohu.com (Z. Gui), liuyuting62@163.com (Y. Liu), wangwm_math@hytc.edu.cn (W. Wang).

https://doi.org/10.1016/j.amc.2018.02.009
0096-3003/

In a stochastic differential equation (SDE) model of a dynamical biological system, environmental variability is often treated by modifying the parameters in the model. And there are two common approaches to modify parameters for a varying environment. The first one assumes that the parameters can be adequately modeled by linear functions of white noise [8,10,13,14,19–27], and the second approach assumes that the parameters satisfy mean-reverting stochastic processes [12,16,28–30]. Allen [16] investigated the relations between these two approaches and showed that the mean-reverting stochastic processes is a practical and biologically realistic way to incorporate the effects of environmental variability in the parameters.

Thanks the insight work of Allen [16], in this paper, we will focus on how environment fluctuations introduced in the natural death rate incorporating the mean-reverting processes affect the extinction and persistence of the disease dynamics. The rest of this article is organized as follows: In Section 2, we derive a deterministic epidemic model (or without noise) and its corresponding stochastic version (or with noise). In Section 3, we show the existence of the global solution. In Section 4, we give the stochastic dynamics in details. In Section 5, we provide some numerical simulations to support our findings. In the last section, Section 6, we provide a brief discussion and the summary of the main results.

2. Model derivations and preliminaries

2.1. The deterministic model

Motivated by Xiao and Chen [31,32], in this subsection, we firstly give the following assumptions to our model:

(H1) Let $S(t)$ and $I(t)$ be the density of the susceptible (S) and infectious (I), respectively, and assume that the total population N is split into a susceptible part S and an infected part I, i.e., $N(t) = S(t) + I(t)$.

(H2) Assume that the maximum per capita birth rate of uninfected hosts is a, the per capita density-dependent reduction in birth rate is c, obviously, $1/c$ is the carrying capacity, and the natural death rate is μ.

(H3) Assume that only susceptible S is capable of reproducing with logistic law, epidemiologically speaking, the infectious I is removed by death (say, its disease-induced death rate is constant α) before having the possibility of reproducing [33]. And assume that the infectious I still contributes with S to population growth toward the carrying capacity, and there is no recovery from the infectious I.

(H4) The disease transmission is assumed to be mass action βSI, where $\beta > 0$ is the transmission coefficient.

Based on the assumptions above, we can obtain the following epidemiological model:

$$\begin{cases} \dfrac{dS}{dt} = aS[1 - c(S+I)] - \beta SI - \mu S, \\ \dfrac{dI}{dt} = \beta SI - (\mu + \alpha)I, \end{cases} \tag{1}$$

It is worthy to note that model (1) is a modified case of Xiao's model [31,32]. Also, model (1) can be seen as a special case $f = 0$ of Ebert's model [34]. In the view of this point, model (1) can be used to investigate the dynamics of the parasites–hosts system.

In the context of epidemic models with variable population size, the view that the qualitative dynamics of such systems are controlled in specific ways by the demographic threshold $\mathcal{R}_d$, the basic reproduction number for the demographic process [35]. For model (1), the demographic reproductive number $\mathcal{R}_d$ is given by

$$\mathcal{R}_d = \frac{a}{\mu}. \tag{2}$$

On the other hand, the basic reproduction number $\mathcal{R}_0$ is often considered as the threshold quantity that determines when an infection can invade and persist in a new host population, which is defined as the expected number of secondary cases produced, in a completely susceptible population, by a typical infected individual during its entire period of infectiousness [36–38]. For model (1), the basic reproduction number is given by

$$\mathcal{R}_0 = \frac{\beta(a-\mu)}{ac(\alpha+\mu)} = \frac{\beta(\mathcal{R}_d - 1)}{c\mathcal{R}_d(\alpha+\mu)}. \tag{3}$$

Then we can obtain following properties of model (1). The proof of this theorem is similar to that in [34] and hence is omitted here.

Theorem 2.1.

(A) *If* $\mathcal{R}_d < 1$, *then for any given initial value* $(S_0, I_0) \in \mathbb{R}^2_+$, *the susceptible* $S(t)$ *and infectious* $I(t)$ *tend to* (0, 0).

(B) *If* $\mathcal{R}_d > 1$ *holds, then*

(B1) *if* $\mathcal{R}_0 \le 1$, *then the disease-free steady state* $\left(\dfrac{a-\mu}{ac}, 0\right) = \left(\dfrac{\mathcal{R}_d - 1}{c\mathcal{R}_d}, 0\right)$ *is globally asymptotically stable with respect to initial value* $(S_0, I_0) \in \mathbb{R}^2_+$.

(B2) *if* $\mathcal{R}_0 > 1$, *there exists a unique endemic state*

$$E^* = \left(\frac{\alpha+\mu}{\beta}, \frac{(a-\mu)\beta - ac(\alpha+\mu)}{\beta(ac+\beta)}\right) = \left(\frac{\mathcal{R}_d - 1}{c\mathcal{R}_d\mathcal{R}_0}, \frac{\mu(\mathcal{R}_d-1)^2(\mathcal{R}_0-1)}{c\mathcal{R}_d\mathcal{R}_0(\mathcal{R}_0(\alpha+\mu)+\mu(R_d-1))}\right),$$

which is global asymptotic stability with respect to initial value $(S_0, I_0) \in \mathbb{R}^2_+$.

2.2. The stochastic model

Notice that the parameter μ in model (1) represents the natural death rate, a measure of the number of deaths (in general, or due to a specific cause) in a particular population, scaled to the size of that population, per unit of time. And in the real situation, the natural death rate μ always fluctuate around an average value due to continuous fluctuations in the environment [14]. To model the effect of the randomly varying environment in μ, we may adopt two common approaches:

The firstly possible model for μ in a randomly-varying environment is a linear function of Gaussian white noise:

$$\mu(t) = \mu_e + \sigma \frac{dW(t)}{dt}, \tag{4}$$

where μ_e is a positive constant which measures the long-run mean level of the natural death rate $\mu(t)$, and $W(t)$ is a standard Wienner process, $\sigma > 0$ denotes the intensity of environmental forcing.

By directly integrating (4), the average natural death rate over an interval $[0, T]$ is equal to

$$\overline{\mu} = \frac{1}{T}\int_0^T \mu(t)dt = \mu_e + \sigma \frac{W(T)}{T} \sim \mathbb{N}\left(\mu_e, \sigma^2/T\right). \tag{5}$$

That is, the average per capita death rate $\overline{\mu}$ over an interval of length T has a variance which goes to infinity as $T \to 0$. And there comes a question of whether it is reasonable for the average value over a finite time period of the natural death rate $\mu(t)$ to become increasingly more variable as the time interval decreases.

The secondly possible model for μ in a randomly-varying environment, we introduce the classical mean-reverting process, the Ornstein–Uhlenbeck process which, has the form:

$$d\mu(t) = \theta\Big(\mu_e - \mu(t)\Big)dt + \xi dB(t), \tag{6}$$

where $B(t)$ is a standard Brownian motion, θ and ξ are all positive constants, θ the speed of reversion, ξ the intensity of volatility. And in the theory of Financial-Economics, this format of mean-reverting process was studied by Dixit and Pindyck [39] firstly, and is also known as Dixit and Pindyck model.

Through the stochastic integral format for the arithmetic Ornstein–Uhlenbeck process (6), we can obtain the following explicit form solution:

$$\mu(t) = \mu_e + (\mu_0 - \mu_e)e^{-\theta t} + \xi \int_0^t e^{-\theta(t-s)}dB(s), \tag{7}$$

where $\mu_0 := \mu(0)$.

Easy to know, over an interval $[0, T]$, the expected value of $\mu(t)$ is:

$$\mathbf{E}[\mu(t)] = \mu_e + (\mu_0 - \mu_e)e^{-\theta T}, \tag{8}$$

and the variance of $\mu(t)$ is:

$$\mathbf{Var}[\mu(t)] = \frac{\xi^2}{2\theta}\left(1 - e^{-2\theta T}\right). \tag{9}$$

Thus, as $T \to 0$, $\mathbf{E} \to \mu_0$ with $\mathbf{Var}[\mu(t)] \to 0$. Obviously, unlike when the natural death rate $\mu(t)$ is a linear function of white noise in (5), the variance goes to 0 rather than ∞ as $T \to 0$, this is a biologically reasonable assumption. Indeed this is a well-established way of introducing stochastic environmental noise into biologically realistic population dynamic models.

Based on the discussions above, in this paper, we will focus on the randomly-varying environment introduced in the natural death rate incorporating the mean-reverting stochastic processes.

Considering (6) again, from the results in (8) and (9), one can easy to know that the term $\xi \int_0^t e^{-\theta(t-s)}dB(s)$ follows the normal distribution $\mathbb{N}\left(0, \frac{\xi^2}{2\theta}\left(1 - e^{-2\theta t}\right)\right)$, it is almost everywhere equal to

$$\xi \int_0^t e^{-\theta(t-s)}dB(s) = \frac{\xi}{\sqrt{2\theta}}\sqrt{1 - e^{-2\theta t}}\,\frac{dB(t)}{dt} \quad a.e..$$

Thus, (7) can be almost surely written as follows:

$$\mu(t) = \mu_e + (\mu_0 - \mu_e)e^{-\theta t} + \sigma(t)\frac{dB(t)}{dt}, \tag{10}$$

where

$$\sigma(t) = \frac{\xi}{\sqrt{2\theta}}\sqrt{1 - e^{-2\theta t}}. \tag{11}$$

Combining model (1) with (6) together, we can derive the following stochastic differential equations:

$$\begin{cases} dS(t) = \Big[aS(t)\Big(1 - c(S(t) + I(t)\Big) - \beta S(t)I(t) - \mu(t)S(t)\Big]dt, \\ dI(t) = \Big[\beta S(t)I(t) - (\mu(t) + \alpha)I(t)\Big]dt, \\ d\mu(t) = \theta\Big(\mu_e - \mu(t)\Big)dt + \xi dB(t). \end{cases} \tag{12}$$

Substituting (10) into model (12), we can obtain the following stochastic model:

$$\begin{cases} dS = S\Big[a - \mu_e + (\mu_e - \mu_0)e^{-\theta t} - acS - (ac + \beta)I\Big]dt - \sigma(t)SdB(t), \\ dI = I\Big[\beta S - \mu_e - \alpha + (\mu_e - \mu_0)e^{-\theta t}\Big]dt - \sigma(t)IdB(t). \end{cases} \tag{13}$$

2.3. Preliminaries

Throughout this paper, let $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t\geq 0}, \mathbb{P})$ be a complete space with a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ satisfying the usual conditions, i.e., it is right continuous and $\mathcal{F}_0$ contains all $\mathbb{P}$-null sets.

In addition, we introduce the following useful Lemma which can be found in [11].

Lemma 2.2. *Let $f \in C[[0, \infty) \times \Omega, (0, \infty)]$.*

(i) If there exist positive constants λ_0 and λ such that

$$\ln f(t) \leq \lambda t - \lambda_0 \int_0^t f(s)ds + F(t), \quad a.s. \tag{14}$$

for all $t \geq 0$, where $F \in C[[0, \infty) \times \Omega, \mathbb{R}]$ and $\lim_{t\to\infty} \frac{F(t)}{t} = 0$ a.s., then

$$\limsup_{t\to\infty} \frac{1}{t} \int_0^t f(s)ds \leq \frac{\lambda}{\lambda_0} \quad a.s.$$

(ii) If there exist positive constants λ_0, λ such that

$$\ln f(t) \geq \lambda t - \lambda_0 \int_0^t f(s)ds + F(t) \quad a.s. \tag{15}$$

for all $t \geq 0$, where $F \in C[[0, \infty) \times \Omega, \mathbb{R}]$ and $\lim_{t\to\infty} \frac{F(t)}{t} = 0$ a.s., then

$$\liminf_{t\to\infty} \frac{1}{t} \int_0^t f(s)ds \geq \frac{\lambda}{\lambda_0} \quad a.s..$$

3. Existence and uniqueness of the global positive solution

Theorem 3.1. *For any initial value $(S(0), I(0)) \in \mathbb{R}^2_+$, there is a unique positive solution $(S(t), I(t))$ of the SDE model (13) on $t \geq 0$, and the solution will remain in $\mathbb{R}^2_+$ with probability one.*

Proof. We give the proof in the following two cases.

Case I: $\mu_0 < \mu_e$.

In order to prove the existence and uniqueness of $S(t)$, we only need prove the boundedness of $S(t)$. Let $z_1(t)$ be the solution of the following equation

$$dz_1 = z_1(a - \mu_0 - acz_1)dt - \sigma(t)z_1 dB(t). \tag{16}$$

Making use of comparison theorem for stochastic equations [11] gives $S(t) \leq z_1(t)$. At the same time, (16) has the explicit solution of the form:

$$z_1(t) = \frac{\exp\left\{\int_0^t \left[a - \mu_0 - \frac{\sigma^2(s)}{2}\right]ds - \int_0^t \sigma(s)dB(s)\right\}}{\frac{1}{z_1(0)} + ac\int_0^t \exp\left\{\int_0^s \left[a - \mu_0 - \frac{\sigma^2(\tau)}{2}\right]d\tau - \int_0^s \sigma(\tau)dB(\tau)\right\}ds}. \tag{17}$$

In view of (11), we have

$$z_1(t)=\frac{\exp\left\{\left(a-\mu_0-\frac{\xi^2}{4\theta}\right)t+\frac{\xi^2}{8\theta^2}(1-e^{-2\theta t})-\int_0^t\sigma(s)dB(s)\right\}}{\frac{1}{z_1(0)}+ac\int_0^t\exp\left\{\left(a-\mu_0-\frac{\xi^2}{4\theta}\right)s+\frac{\xi^2}{8\theta^2}(1-e^{-2\theta s})-\int_0^s\sigma(\tau)dB(\tau)\right\}ds}. \tag{18}$$

If $a<\mu_0+\frac{\xi^2}{4\theta}$, then it follows from (18) that

$$S(t)\le z_1(t)\le z_1(0)\exp\left\{-t\left[\left(\mu_0+\frac{\xi^2}{4\theta}-a\right)-\frac{\xi^2}{8\theta^2t}\left(1-e^{-2\theta t}\right)+\frac{1}{t}\int_0^t\sigma(t)dB(s)\right]\right\}.$$

Since $\lim_{t\to\infty}\frac{\xi^2}{8\theta^2t}(1-e^{-2\theta t})=0$, and according the strong law of large number of local martingale [40], we can get

$$\lim_{t\to\infty}\frac{1}{t}\int_0^t\sigma(s)dB(s)=0, \tag{19}$$

yields $\limsup_{t\to\infty}z_1(t)\le 0$ *a.s.*. Then we can obtain $\lim_{t\to\infty}S(t)=0$ *a.s.*. Hence,

$$\limsup_{t\to\infty}\frac{\ln S(t)}{t}\le 0\quad a.s.. \tag{20}$$

Set

$$F(s)=\frac{\xi^2}{8\theta^2}\left(e^{-2\theta t}-e^{-2\theta s}\right)+\int_0^t\sigma(s)dB(s)-\int_0^s\sigma(\tau)dB(\tau).$$

If $a\ge\mu_0+\frac{\xi^2}{4\theta}$, then applying mean value theorem for integral to (18) gives

$$\begin{aligned}z_1(t)&\le\frac{1}{ac\int_0^t\exp\left\{\left(a-\mu_0-\frac{\xi^2}{4\theta}\right)(s-t)+F(s)\right\}ds}\\&\le\frac{1}{ac\exp\{F(\zeta)\}\int_0^t\exp\left\{\left(a-\mu_0-\frac{\xi^2}{4\theta}\right)(s-t)\right\}ds}\\&\le\frac{\left(a-\mu_0-\xi^2/4\theta\right)}{ac}\cdot\frac{1}{\exp\{F(\zeta)\}\left\{1-\exp\left[-\left(a-\mu_0-\xi^2/4\theta\right)t\right]\right\}},\end{aligned}$$

where $\varsigma\in[0,t]$. Considering (19) again leads to that $\limsup_{t\to\infty}\frac{\ln z_1(t)}{t}\le 0$ *a.s.*, thus

$$\limsup_{t\to\infty}\frac{\ln S(t)}{t}\le 0\quad a.s..$$

That is to say, for any $\varepsilon\in(0,1)$, there is a positive constant $K(\varepsilon)$, such that

$$\mathbb{P}(0<S(t)\le K)\ge 1-\varepsilon,\quad \text{for } t\ge 0. \tag{21}$$

Next, we consider the infectious $I(t)$. It follows from (21) that $I(t)$ satisfies

$$dI\le I[K\beta-\alpha]dt-\sigma(t)IdB(t),\quad \text{for } t\ge 0.$$

and

$$dI\ge -I[\mu_e+\alpha]dt-\sigma(t)IdB(t),\quad \text{for } t\ge 0.$$

Also by the comparison theorem of stochastic equations, we obtain

$$\underline{I}(t)\le I(t)\le\bar{I}(t),\quad a.s., \tag{22}$$

where

$$\underline{I}(t) = I(0)\exp\left\{\left(K\beta - \alpha - \frac{\xi^2}{4\theta}\right)t + \frac{\xi^2}{8\theta^2}(1-e^{-2\theta t}) - \int_0^t \sigma(s)dB(s)\right\},$$

$$\bar{I}(t) = I(0)\exp\left\{\left(-\mu_e - \alpha - \frac{\xi^2}{4\theta}\right)t + \frac{\xi^2}{8\theta^2}(1-e^{-2\theta t}) - \int_0^t \sigma(s)dB(s)\right\}.$$

From the representation of solutions $\underline{I}(t), \bar{I}(t)$, we can easily get the existence and uniqueness of $I(t)$ for $t\in[0,\infty)$.

Case II: $\mu_0 > \mu_e$.

Suppose that $z_2(t)$ is the solution of equation:

$$dz_2 = z_2(a - \mu_e - acz_2)dt - \sigma(t)z_2 dB(t), \tag{23}$$

in all the cases of $a < \mu_e + \frac{\xi^2}{4\theta}$ and $a \geq \mu_e + \frac{\xi^2}{4\theta}$, similar to the method above, we can obtain

$$\limsup_{t\to\infty} \frac{\ln S(t)}{t} \leq 0.$$

Similarly, $I(t)$ satisfies

$$dI \leq I[K\beta - \mu_e - \alpha]dt - \sigma(t)IdB(t), \quad \text{for } t \geq 0,$$

and

$$dI \geq -I[\mu_e + \alpha + \mu_0]dt - \sigma(t)IdB(t), \quad \text{for } t \geq 0.$$

Then we can also get the existence and uniqueness of $I(t)$ for $t\in[0,\infty)$, which is the desired assertion. □

4. Stochastic dynamics of the disease

In this section, we will focus on the stochastic extinction and persistence of the disease of the SDE model (13). First of all, we introduce two threshold parameters for the SDE model (13), one is $\mathcal{R}_d^s$, and the other is $\mathcal{R}_0^s$.

$$\mathcal{R}_d^s := \frac{a}{\mu_e + \xi^2/4\theta}. \tag{24}$$

$$\mathcal{R}_0^s := \frac{\beta\left(a - \mu_e - \xi^2/4\theta\right)}{ac\left(\alpha + \mu_e + \xi^2/4\theta\right)} = \frac{\beta\left(\mathcal{R}_d^s - 1\right)}{c\left(\alpha\mathcal{R}_d^s + a\right)}. \tag{25}$$

4.1. Stochastic extinction of the whole hosts population

In this subsection, we will give the results of stochastic extinction of the whole population N.

Theorem 4.1. *If $\mathcal{R}_d^s < 1$, for any given initial value $(S(0), I(0)) \in \mathbb{R}_+^2$, the solution $(S(t), I(t))$ of the SDE model (13) obeys*

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

namely, $S(t)$ and $I(t)$ will go to extinction a.s. In other words, the whole population $N(t)$ dies out with probability one.

Proof. For model (13), applying Itô's formula to $\ln S(t)$ and $\ln I(t)$ and integrating both sides from 0 to t, we obtain

$$\ln S(t) = \ln S(0) + \left(\mu_e + \xi^2/4\theta\right)\left(\mathcal{R}_d^s - 1\right)t - ac\int_0^t S(s)ds - (ac+\beta)\int_0^t I(s)ds + tf_1(t) - \int_0^t \sigma(s)dB(s), \tag{26}$$

and

$$\ln I(t) = \ln I(0) - \left(\mu_e + \frac{\xi^2}{4\theta} + \alpha\right)t + \beta\int_0^t S(s)ds + tf_1(t) - \int_0^t \sigma(s)dB(s), \tag{27}$$

where

$$f_1(t) = \frac{\xi^2}{8\theta^2 t}(1-e^{-2\theta t}) - \frac{\mu_0 - \mu_e}{t\theta}(1-e^{-\theta t}). \tag{28}$$

By virtue of (26), we have

$$\frac{1}{t}\ln S(t) \leq \frac{\ln S(0)}{t} + \left(\mu_e + \xi^2/4\theta\right)\left(\mathcal{R}_d^s - 1\right) + f_1(t) - \frac{1}{t}\int_0^t \sigma(s)dB(s). \tag{29}$$

Obviously, $\lim_{t\to\infty} f_1(t) = 0$. Noting that $\int_0^t \sigma(s)dB(s) = \frac{\xi}{\sqrt{2\theta}}\int_0^t \sqrt{1-e^{-2\theta t}}dB(s)$ is a local martingale, it follows from the strong law of large number of local martingale [40] that

$$\lim_{t\to\infty}\frac{1}{t}\int_0^t \sigma(s)dB(s) = 0, \quad a.s.$$

Taking upper limit on both sides of (29) and making use of $\mathcal{R}_d^s < 1$ yields

$$\limsup_{t\to\infty}\frac{\ln S(t)}{t} \le \left(\mu_e + \xi^2/4\theta\right)\left(\mathcal{R}_d^s - 1\right) < 0, \quad a.s.$$

which implies

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

Hence, for an arbitrary positive constant ε_1, there exists a constant $T_1 = T_1(w)$ and a set Ω_{ε_1} such that $P(\Omega_{\varepsilon_1}) \ge 1-\varepsilon_1$ and $S(t,\omega) \le \varepsilon_1$ for $t \ge T_1, w \in \Omega_{\varepsilon_1}$.

In the view of (27), for $t \ge T_1$, we get

$$\frac{1}{t}\ln I(t,\omega) \le \ln I(0,\omega) - \left(\mu_e + \frac{\xi^2}{4\theta} + \alpha\right) + \beta\varepsilon_1 + f_1(t) - \frac{1}{t}\int_0^t \sigma(s)dB(s), \tag{30}$$

where $f_1(t)$ is defined in (28). Taking upper limit on both sides of (30) and with the arbitrary of ε_1 yields

$$\limsup_{t\to\infty}\frac{1}{t}\ln I(t,\omega) \le -\left(\mu_e + \frac{\xi^2}{4\theta} + \alpha\right) < 0, \tag{31}$$

which implies $\lim_{t\to\infty} I(t) = 0$. The proof is completed. □

Theorem 4.1 gives the results in the case of $\mathcal{R}_d^s < 1$. Next, we will focus on the stochastic dynamics of model (13) in the case of $\mathcal{R}_d^s > 1$.

4.2. Stochastic disease-free and endemic dynamics

Definition 4.2 [20]**.** Model (13) is said to be weak persistent in the mean if

$$\limsup_{t\to\infty}\frac{1}{t}\int_0^t S(s)ds > 0, \quad \limsup_{t\to\infty}\frac{1}{t}\int_0^t I(s)ds > 0.$$

Theorem 4.3. *If $\mathcal{R}_d^s > 1$, then*

(i) *if $\mathcal{R}_0^s < 1$, then for any given initial value $(S(0), I(0)) \in R_+^2$, the solution $(S(t), I(t))$ of the SDE model (13) obeys*

$$\lim_{t\to\infty}\frac{1}{t}\int_0^t S(s)ds = \frac{\mathcal{R}_d^s - 1}{c\mathcal{R}_d^s}, \quad \lim_{t\to\infty} I(t) = 0 \quad a.s.$$

namely, $S(t)$ will be persist and $I(t)$ go to extinction a.s. In other words, the disease dies out with probability one.

(ii) *if $\mathcal{R}_0^s > 1$, then the solution $(S(t), I(t))$ of model (13) has the following properties:*

$$0 < \limsup_{t\to\infty}\frac{1}{t}\int_0^t S(s)ds \le \frac{\mathcal{R}_d^s - 1}{c\mathcal{R}_d^s\mathcal{R}_0^s} \quad a.s. \tag{32}$$

and

$$0 < \frac{\left(\mu_e + \xi^2/4\theta\right)\left(\mathcal{R}_0^s - 1\right)\left(\mathcal{R}_d^s - 1\right)}{\mathcal{R}_0^s(ac+\beta)} \le \liminf_{t\to\infty}\frac{1}{t}\int_0^t I(s)ds \le \limsup_{t\to\infty}\frac{1}{t}\int_0^t I(s)ds \quad a.s. \tag{33}$$

That is, model (13) is weak persistence in the mean (i.e.,) with probability one.

Proof. (i) If $\mathcal{R}_d^s > 1$, it then follows (26) that

$$\ln S(t) \quad \le \left(\mu_e + \xi^2/4\theta)(\mathcal{R}_d^s - 1)\right)t - ac\int_0^t S(s)ds + \ln S(0) + t f_1(t) - \int_0^t \sigma(s)dB(s). \tag{34}$$

According to Lemma 2.2, we have

$$\limsup_{t\to\infty}\frac{1}{t}\int_0^t S(s)ds \le \frac{\left(\mu_e + \xi^2/4\theta\right)\left(\mathcal{R}_d^s - 1\right)}{ac} = \frac{\mathcal{R}_d^s - 1}{c\mathcal{R}_d^s} > 0, \tag{35}$$

which implies that, for any $\varepsilon > 0$, there is a $T_2 = T_2(w) > 0$ such that

$$\frac{1}{t}\int_0^t S(s,\omega)ds \le \frac{\mathcal{R}_d^s - 1}{c\mathcal{R}_d^s} + \varepsilon, \quad \text{for } t \ge T_2. \tag{36}$$

From (27), for $t \ge T_2(w)$, we obtain

$$\begin{aligned}\frac{\ln I(t,\omega)}{t} &= \frac{\ln I(0,\omega)}{t} - \left(\mu_e + \xi^2/4\theta + \alpha\right) + \frac{\beta}{t}\int_0^t S(s,\omega)ds + f_1(t) - \frac{1}{t}\int_0^t \sigma(s)dB(s)\\ &\le -\left(\mu_e + \xi^2/4\theta + \alpha\right) + \beta\left(\frac{\mathcal{R}_d^s - 1}{c\mathcal{R}_d^s} + \varepsilon\right) + \frac{\ln I(0,\omega)}{t} + f_1(t) - \frac{1}{t}\int_0^t \sigma(s)dB(s)\\ &= \left(\mu_e + \xi^2/4\theta + \alpha\right)(\mathcal{R}_0^s - 1) + \beta\varepsilon + \frac{\ln I(0,\omega)}{t} + f_1(t) - \frac{1}{t}\int_0^t \sigma(s)dB(s).\end{aligned} \tag{37}$$

Letting $t \to \infty$, if $\mathcal{R}_0^s < 1$, and with the arbitrariness of ε, we can get

$$\limsup_{t\to\infty} \frac{1}{t}\ln I(t,\omega) \le \left(\mu_e + \xi^2/4\theta + \alpha\right)(\mathcal{R}_0^s - 1) < 0, \tag{38}$$

which implies $\lim_{t\to\infty} I(t) = 0$ a.s. Hence, for an arbitrary positive constant ε, there exists a constant $\tilde{T} = \tilde{T}(\omega)$ and a set Ω_ε such that $\mathbb{P}(\Omega_\varepsilon) \ge 1 - \varepsilon$ and $I(t,\omega) \le \varepsilon$ for $t \ge \tilde{T}$. Then it follows from (26) that

$$\ln S(t,\omega) \ge \ln S(0,\omega) + \left(\mu_e + \xi^2/4\theta\right)\left(\mathcal{R}_d^s - 1\right)t - ac\int_0^t S(s)ds - \varepsilon(ac+\beta)t + tf_1(t) - \int_0^t \sigma(s)dB(s). \tag{39}$$

It follows from Lemma 2.2 that

$$\liminf_{t\to\infty} \frac{1}{t}\int_0^t S(s,\omega)ds \ge \frac{\mathcal{R}_d^s - 1}{c\mathcal{R}_d^s} > 0 \quad a.s \tag{40}$$

which, together with (35), implies that $\lim_{t\to\infty} \frac{1}{t}\int_0^t S(s)ds = \frac{\mathcal{R}_d^s - 1}{c\mathcal{R}_d^s}$ a.s. This ends the proof of (i).

(ii) It follows from (26) that

$$\int_0^t S(s)ds = \frac{\left(\mu_e + \xi^2/4\theta\right)\left(\mathcal{R}_d^s - 1\right)}{ac}t - \frac{ac+\beta}{ac}\int_0^t I(s)ds + \varphi_1(t), \tag{41}$$

where

$$\varphi_1(t) = \frac{1}{ac}\left(\ln S(0) - \ln S(t) + tf_1(t) - \int_0^t \sigma(s)dB(s)\right).$$

In the view of (27) and (41), we can get

$$\ln I(t) = \left(\mu_e + \xi^2/4\theta + \alpha\right)(\mathcal{R}_0^s - 1)t - \frac{\beta(ac+\beta)}{ac}\int_0^t I(s)ds + \varphi_2(t), \tag{42}$$

where $\varphi_2(t) = \ln I(0) + \beta\varphi_1(t) + tf_1(t) - \int_0^t \sigma(s)dB(s)$. By the strong law of large numbers for martingales [40], we have

$$\lim_{t\to\infty}\left(f_1(t) - \frac{1}{t}\int_0^t \sigma(s)dB(s)\right) = 0.$$

Noting that $\limsup_{t\to\infty} \frac{1}{t}\ln\frac{S(t)}{S(0)} \le 0$, it follows from Lemma 2.2 that

$$\liminf_{t\to\infty} \frac{1}{t}\int_0^t I(s)ds \ge \frac{(\mu_e + \xi^2/4\theta)(\mathcal{R}_0^s - 1)(\mathcal{R}_d^s - 1)}{\mathcal{R}_0^s(ac+\beta)} := I_*,$$

which implies that, for any arbitrary $\eta > 0 (\eta < I_*)$, there is a $T(\omega)$ such that

$$\frac{1}{t}\int_0^t I(s,\omega)ds \ge I_* - \eta, \quad t \ge T(\omega), \tag{43}$$

then, it follows from (26) and (43) that

$$\begin{aligned}\ln S(t,\omega) \le \ln S(0) &+ \left(\mu_e + \frac{\xi^2}{4\theta}\right)(\mathcal{R}_d^s - 1)t - (ac+\beta)(I_* - \eta)t\\ &-ac\int_0^t S(s)ds + tf_1(t) - \int_0^t \sigma(s)dB(s).\end{aligned} \tag{44}$$

By Lemma 2.2 again, with η arbitrary, we get

$$\limsup_{t\to\infty}\frac{1}{t}\int_0^t S(s)ds \le \frac{\mathcal{R}_d^s-1}{c\mathcal{R}_d^s\mathcal{R}_0^s} \quad a.s.$$

and $\dfrac{\mathcal{R}_d^s-1}{c\mathcal{R}_d^s\mathcal{R}_0^s} > 0$.

We claim that $\limsup_{t\to\infty}\frac{1}{t}\int_0^t S(s)ds > 0$. In fact, for any $\omega \in \left\{\limsup_{t\to\infty}\frac{1}{t}\int_0^t S(s,\omega)ds = 0\right\}$, we have from (27)

$$\limsup_{t\to\infty}\frac{\ln I(t,\omega)}{t} \le -\left(\mu_e+\frac{\xi^2}{4\theta}+\alpha\right) < 0.$$

That is to say, $\lim_{t\to\infty} I(t,\omega) = 0$, which contradicts with (33). This finishes the proof of (ii). □

4.3. Two threshold quantities: $\mathcal{R}_d^s$ and $\mathcal{R}_0^s$

Combining with the use of $\mathcal{R}_d^s$ in Theorems 4.1 and 4.3, we can conclude that $\mathcal{R}_d^s$ can be seen as a threshold parameter to govern whether the whole population extinct or not: if $\mathcal{R}_d^s < 1$, the whole population $N(t)$ will die out a.s; while if $\mathcal{R}_d^s > 1$, $N(t)$ will persist with probability one. Obviously, this is the same use as the demographic reproduction number $\mathcal{R}_d$. In the view of this point, we can call $\mathcal{R}_d^s$ as the stochastic demographic reproduction number for the SDE model (13).

From Theorem 4.3, we can know that: in the case of $\mathcal{R}_d^s > 1$, if $\mathcal{R}_0^s < 1$, the disease $I(t)$ will extinct a.s; while if $\mathcal{R}_0^s > 1$, $I(t)$ will weak persist with probability one. That is to say, $\mathcal{R}_0^s$ can be used to determines when an infection can invade and persist in a new host population. Actually, this is the same epidemiological meaning as the basic reproduction number $\mathcal{R}_0$. Hence, we can call $\mathcal{R}_0^s$ as the stochastic basic reproduction number for the SDE model (13).

Remark 4.4. (i) The expressions of $\mathcal{R}_d^s$ in (24) suggests that

(i-1) $\mathcal{R}_d^s < \mathcal{R}_d$. Then we can easily find an example when $\mathcal{R}_d > 1$ but $\mathcal{R}_d^s < 1$ such that the whole population goes to extinct a.s. This implies that large environment fluctuations can induce the whole population $N(t)$ dies out with probability one.

(i-2) $\mathcal{R}_d^s$ is decreasing with respect to the intensity of volatility ξ, and increasing with respect to the speed of reversion θ.

(ii) In Theorem 4.1, the condition $\mathcal{R}_d^s < 1$ implies that

$$\xi > 2\sqrt{\theta(a-\mu_e)} := \xi^* \quad \text{or} \quad \theta < \frac{\xi^2}{4(a-\mu_e)} := \theta_*.$$

In both cases above, $S(t)$ and $I(t)$ will go to extinction with probability one. Together with the fact $\mathcal{R}_d^s < 1 < \mathcal{R}_d$, we can conclude that the large intensity of volatility ξ or the small speed of reversion θ may induce the extinction of the whole population $N(t)$.

Remark 4.5. (i) The expressions of $\mathcal{R}_0^s$ in (25) suggests that

(i-1) $\mathcal{R}_0^s < \mathcal{R}_0$. And we can easily find an example when $\mathcal{R}_0 > 1$ but $\mathcal{R}_0^s < 1$ such that the infectious $I(t)$ goes to extinct a.s. This implies that large environment fluctuations can suppress the outbreak of disease.

(i-2) $\mathcal{R}_0^s$ is decreasing with respect to the intensity of volatility ξ, and increasing with respect to the speed of reversion θ.

(ii) In Theorem 4.3,

(ii-1) $\mathcal{R}_0^s < 1$ implies that

$$\xi_* := \frac{2\sqrt{\theta(ac+\beta)[\beta(a-\mu_e)-ac(\alpha+\mu_e)]}}{ac+\beta} < \xi < 2\sqrt{\theta(a-\mu_e)} =: \xi^*$$

or

$$\theta_* =: \frac{\xi^2}{4(a-\mu_e)} < \theta < \frac{\xi^2}{4(\beta(a-\mu_e)-ac(\alpha+\mu_e))} =: \theta^*.$$

In both cases above, only the infectious $I(t)$ will die out with probability one. Considering the fact $\mathcal{R}_0^s < 1 < \mathcal{R}_0$, we can conclude that the large intensity of volatility ξ or the small speed of reversion θ may induce the extinction of the infectious $I(t)$.

(ii-2) $\mathcal{R}_0^s > 1$ imply that

$$\xi < \xi_* = \frac{2\sqrt{\theta(ac+\beta)[\beta(a-\mu_e)-ac(\alpha+\mu_e)]}}{ac+\beta}$$

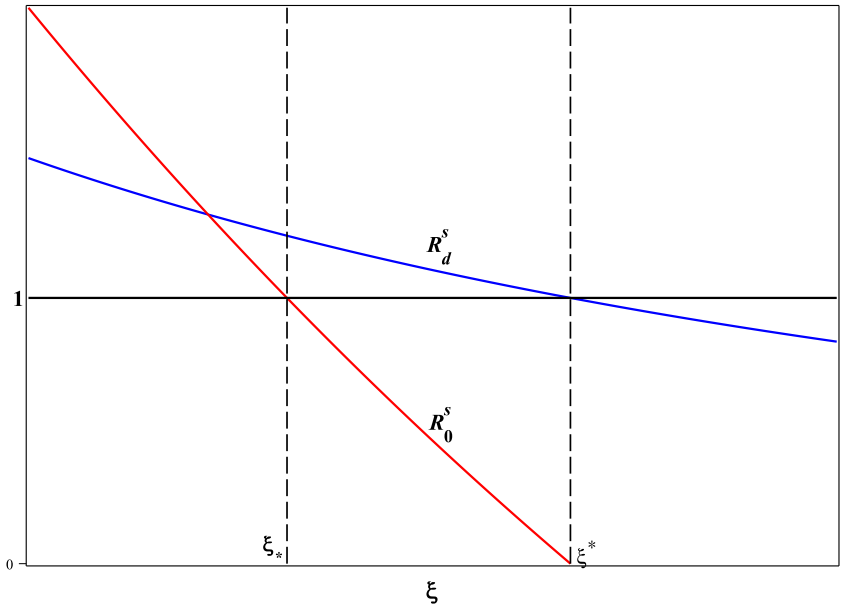


Fig. 1. The relations between $\mathcal{R}_0^s, \mathcal{R}_d^s$ with the intensity of volatility ξ.

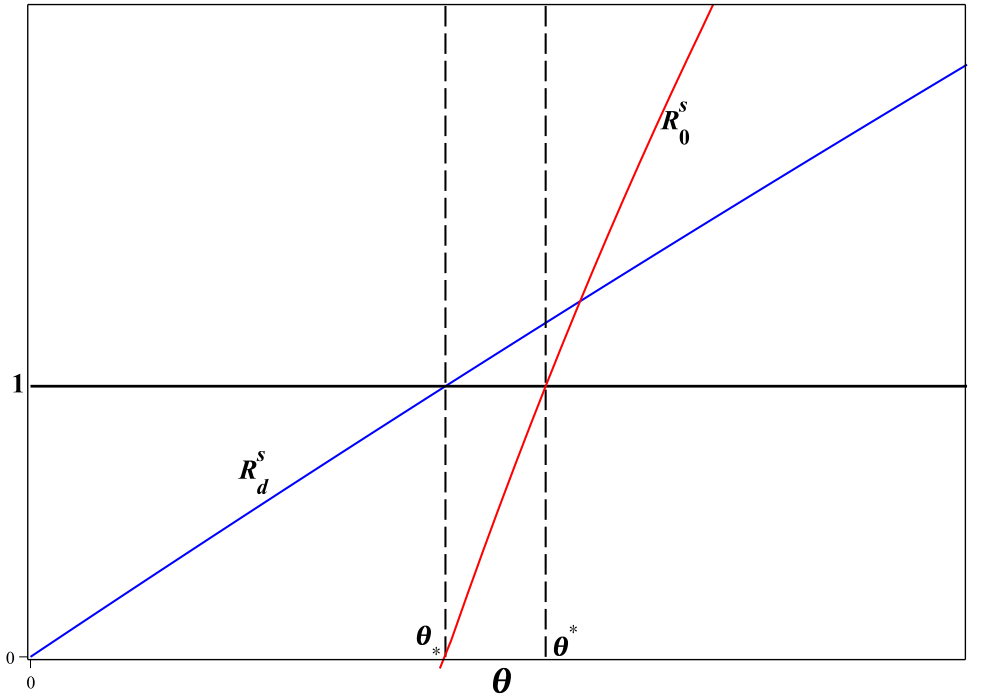


Fig. 2. The relations between $\mathcal{R}_0^s, \mathcal{R}_d^s$ with the speed of reversion θ.

or

$$\theta > \theta^* = \frac{\xi^2}{4(\beta(a-\mu_e) - ac(\alpha+\mu_e))}.$$

This means that the small intensity of volatility ξ or large speed of reversion θ can enhance the outbreak of the disease.

For the sake of learning Theorems 4.1 and 4.3 further, we show the relations between $\mathcal{R}_0^s, \mathcal{R}_d^s$ with ξ and θ in Figs. 1 and 2, respectively. From Fig. 1, we can see that when $\xi < \xi_*$, $\mathcal{R}_d^s > 1$ and $\mathcal{R}_0^s > 1$, the infectious $I(t)$ will spread a.s. and endemic dynamics occurs; when $\xi_* < \xi < \xi^*$, $\mathcal{R}_d^s > 1$ and $\mathcal{R}_0^s < 1$, the infectious $I(t)$ will die out a.s; while $\xi > \xi^*$, $\mathcal{R}_d^s < 1$, the whole

population $N(t)$ will extinct a.s. In Fig. 2, we can see that when $\theta < \theta_*$, $\mathcal{R}_d^s < 1$, the whole population $N(t)$ will extinct a.s.; when $\theta_* < \theta < \theta^*$, $\mathcal{R}_d^s > 1$ and $\mathcal{R}_0^s < 1$, the infectious $I(t)$ will die out a.s.; while $\theta > \theta^*$, $\mathcal{R}_d^s > 1$ and $\mathcal{R}_0^s > 1$, the infectious $I(t)$ will spread a.s. and endemic dynamics occurs.

5. Numerical simulations and dynamics comparisons

In this section, we give some numerical results to show complex disease dynamic outcomes of the SDE model (13) by using the Milstein's method mentioned in Higham [41]. In this way, the numerical scheme for the Milstein's method applied to the stochastic model (13) under considerations is given by

$$\begin{cases} S_{k+1} = S_k + S_k\Big[a - \mu_e + (\mu_e - \mu_0)e^{-\theta(k\Delta t)} - acS_k - (ac+\beta)I_k\Big]\Delta t \\ \qquad -\dfrac{\xi}{\sqrt{2\theta}}(1 - e^{-\theta(k\Delta t)})S(k)\eta_k - \dfrac{\xi^2(1-e^{-\theta(k\Delta t)})^2}{4\theta}S(k)^2(\eta_k^2 - 1)\Delta t, \\ I_{k+1} = I_k + I_k\Big[\beta S_k - \mu_e - \alpha + (\mu_e - \mu_0)e^{-\theta(k\Delta t)}\Big]\Delta t \\ \qquad -\dfrac{\xi}{\sqrt{2\theta}}(1 - e^{-\theta(k\Delta t)})I(k)\eta_k - \dfrac{\xi^2(1-e^{-\theta(k\Delta t)})^2}{4\theta}I(k)^2(\eta_k^2 - 1)\Delta t, \end{cases}$$

where $\eta_k (k = 1, 2, \ldots, n)$ are independent Gaussian random variables $\mathbf{N}(0, 1)$.

Motivated by [34,42], the parameter values are chosen as follows (See, Table 1 for details). $a = 0.4$/d (maximum birth rate in the absen ce of competition), $\alpha = 0.15$/d (disease induced excess mortality rate), $\beta = 0.025$/d/host, $c = 0.1$ and hence the carrying capacity is 10. The long-run mean level of the natural death rate $\mu_e = 0.01$/d (life span in the absence of parasite 100 d), and the initial value of the natural death rate μ_0 is assumed as 0.001/d.

The main goal of this section is to further investigate the answers to the following three questions as we proposed in the introduction:

1. Compare different dynamic outcomes of the deterministic model (1) v.s. its stochastic version (13).
2. The stationary distributions of the solutions of stochastic model (13).

5.1. Stochasticity suppresses the disease outbreak

In this subsection, we give numerical results to show different dynamic outcomes of the deterministic model (1) v.s. its stochastic version (13) with the same set of parameter values. As an example, the initial values are assumed as $(S(0), I(0)) = (9, 1) \in \mathbb{R}_+^2$.

1. For the deterministic model (1), $\mathcal{R}_0 = 1.5234 > 1$, $\mathcal{R}_d = 40 > 1$. Consequently, according to Corollary 2.1, model (1) has a disease-free equilibrium $E_0 = (9.75, 0)$ which is a saddle point, and an endemic equilibrium point $E^* = (6.4, 2.0615)$ which is globally stable (See, Fig. 3).
2. For the corresponding stochastic model (13), we will focus on the role of noise strength on the resulting dynamics. We start our numerical simulation with environmental forcing intensity $\xi = 0.25 > \xi^* = 0.2487$. In this case, $\mathcal{R}_d^s = 6.4430 > 1$, $\mathcal{R}_0^s = 0.9588 < 1$, thus, from Theorem 4.3, we can conclude that the disease will die out in mean with probability one. From and the numerical simulation results in Fig. 4, we can see that, after some initial transients the solution $I(t)$ of the SDE model (13) goes to extinct, while $S(t)$ fluctuate and persist almost surely. To see the disease dynamics of (13) when $\mathcal{R}_0^s > 1$, we choose three different values of $\xi^* > \xi = 0.01, 0.05$ and 0.2, respectively. The corresponding values of $\mathcal{R}_d^s = 39.6694$, 33.1034 and 9.2308, and $\mathcal{R}_0^s = 1.5223$, 1.4958 and 1.1530. According to Theorem 4.3, the infectious $I(t)$ is persist, which is confirmed by the numerical results shown in Fig. 5, and one can see that the evolution of a single stochastic path resulted from the SDE model (13) with the deterministic dynamics from the model (1) for $S(t)$ and $I(t)$ with $\xi = 0.01$ (Fig. 5(a)), 0.05 (Fig. 5(b)), 0.2 (Fig. 5(c)), respectively. As shown, the stochastic paths fluctuated slightly around the deterministic dynamics in all these cases and no extinction was observed.

5.2. Stationary distributions

For the sake of learning the effects of the noise on the disease dynamics, we have repeated the simulation 10000 times keeping all parameters fixed and never observing any extinction scenario up to $t = 600$. This is confirmed by the histograms in Figs. 6 and 7, showing the stationary distributions of $S(t)$ and $I(t)$ at $t = 600$ for the stochastic model (13). And the numerical method for them can be found in [10].

1. *Disease-free stationary distribution:* The unique disease-free stationary distribution occurs when $\mathcal{R}_0^S < 1$ where $S(t)$ is persist and $I(t)$ goes to extinction exponentially a.s. To illustrate the disease-free stationary distribution, we provide numerical results in Fig. 6 whose parameters are the same as those in Fig. 4.

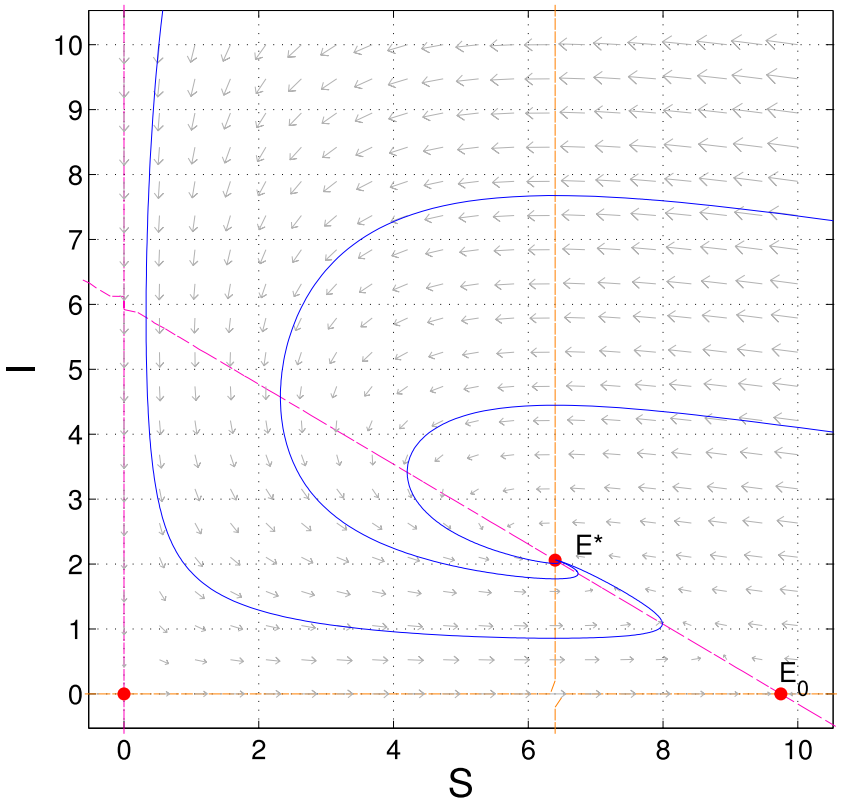


Fig. 3. The phase portrait of model (1). The endemic equilibrium point $E^* = (6.4, 2.0615)$ is globally asymptotically stable, the disease-free equilibrium $E_0 = (9.75, 0)$ and the extinction state (0, 0) are saddle points.

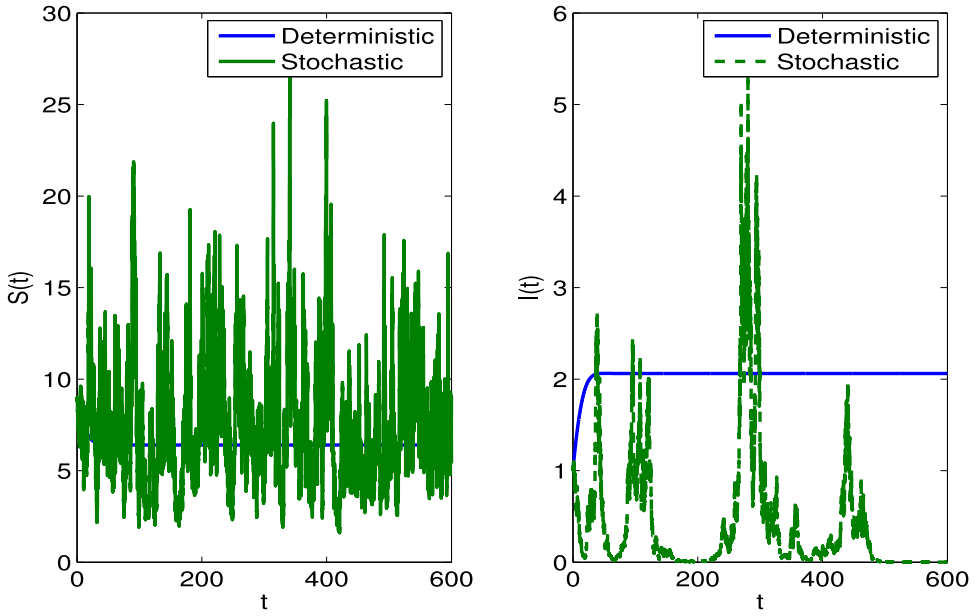
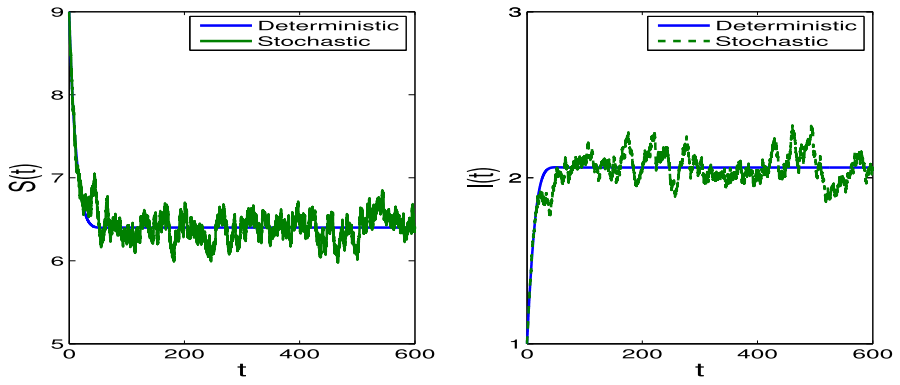
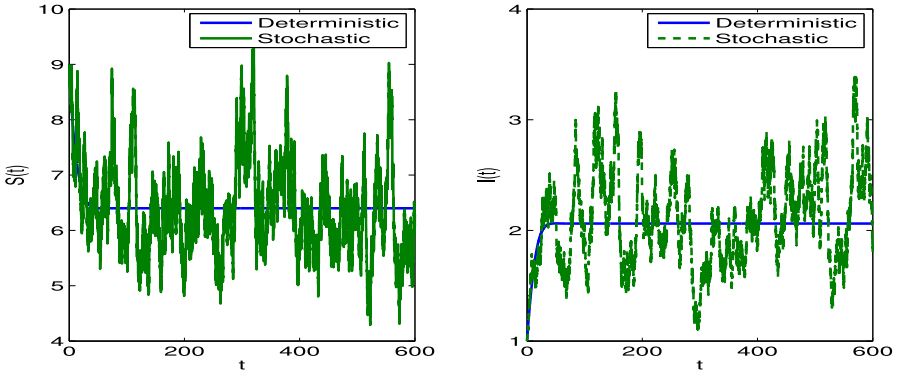


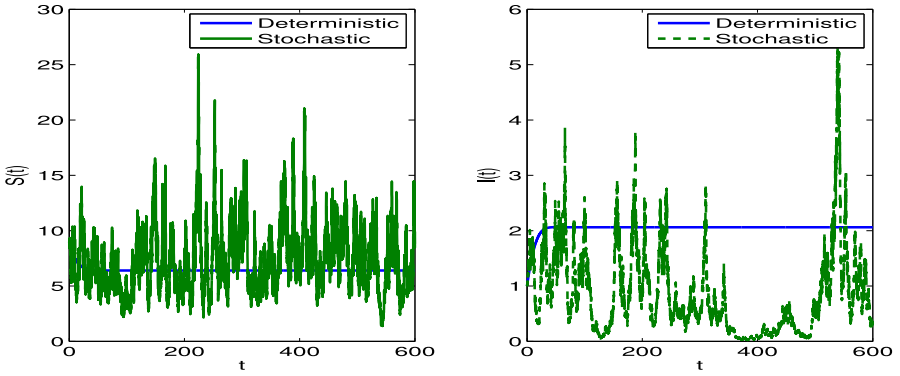
Fig. 4. The evolution of a single path of $S(t)$ and $I(t)$ for Model (13) and its corresponding deterministic Model (1) with $\xi = 0.25$, and all other parameters are taken in Table 1. The initial value of all solution is (9, 1). The time unit is day.



(a) $\xi = 0.01$



(b) $\xi = 0.05$



(c) $\xi = 0.2$

Fig. 5. The evolution of a single path of $S(t)$ and $I(t)$ for the SDE model (13) and its corresponding deterministic model (1) and all other parameters are taken in Table 1. The initial value of all solution is (9, 1). The time unit is day.

2. *Endemic stationary distribution:* The endemic stationary distribution occurs when $\mathcal{R}_0^S > 1$. We take the parameters as in Fig. 5 and show the numerical results in Fig. 7. One can see that, when $\xi = 0.01$, the distribution appears closer to a normal distribution (see Fig. 7(a)), but as ξ increases to 0.2, the distribution is positively skewed (see Fig. 7(c)). We can conclude that for lower ξ (e.g., $\xi = 0.01$), the amplitude of fluctuation is slight and the oscillations are more symmetrically distributed; while for higher ξ (e.g., $\xi = 0.2$), the amplitude of fluctuation is remarkable and the distribution of the solution is skewed.

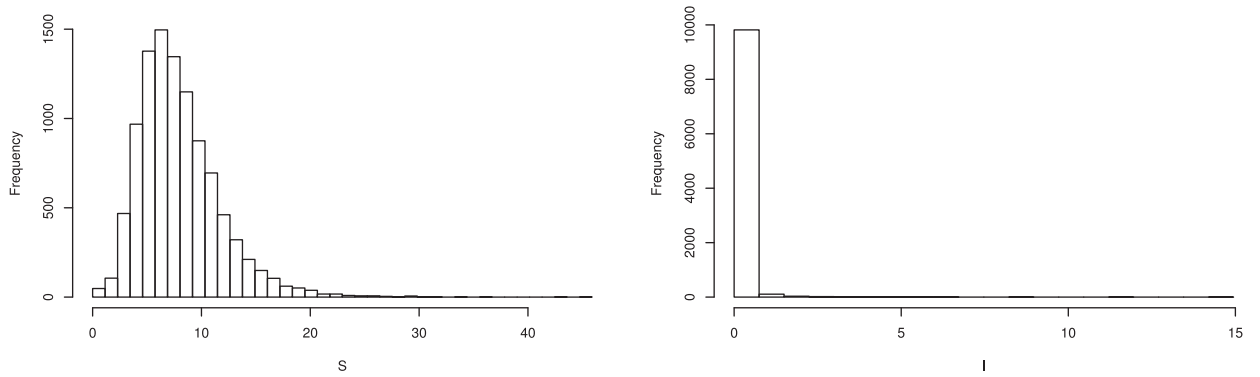


Fig. 6. The histograms of the values of the path $S(t)$ and $I(t)$ for the SDE model (13) based on 10, 000 stochastic simulations, and the parameters are taken as the same as in Fig. 4.

Table 1
Parameter values in numerical simulations for model (13).

Parameter	Biological meaning	Value range	Sources
a	The maximum birth rate of uninfected hosts	0.4/d	[34,42]
c	The density-dependent reduction in birth rate	0.1/d	Estimated
α	The disease-induced death rate	0–0.2/d	[34,42]
β	Transmission coefficient	0.01–0.1/d/host	[34]
μ_e	The long-run mean level of the natural death rate	0–0.02/d	[34]
μ_0	The initial value of the natural death rate	0.001	Estimated
θ	The speed of reversion	0.3	[12]
ξ	The intensity of volatility	0–1	Estimated

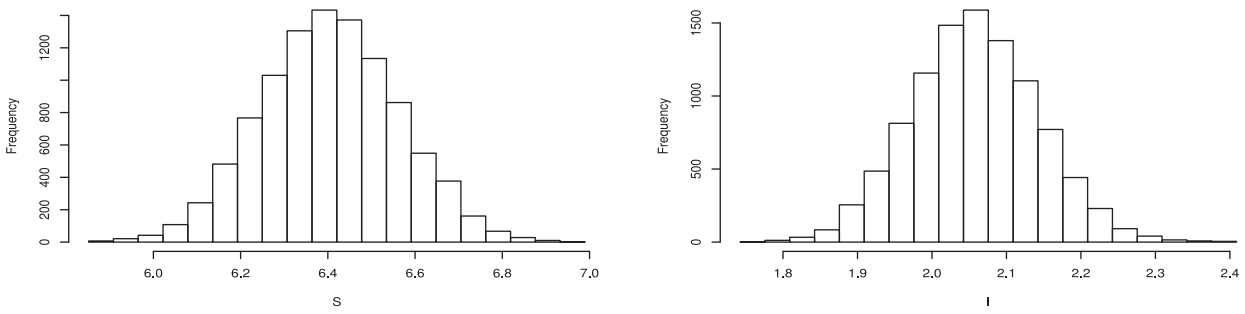
6. Conclusions and discussions

Among the various ways of constructing a stochastic model systems for a given deterministic system, in this paper, we propose a stochastic version of a simple epidemic model incorporating mean-reverting Ornstein–Uhlenbeck process. The mean-reverting processes possess several important feature that better characterize environmental variability in biological systems than does a linear function of white noise. Most importantly, mean-reverting processes over a linear function of Gaussian white noise are continuity, nonnegativity, practicality, possession of asymptotic distributions and so on. In our model, we suppose that stochastic environmental factor acts simultaneously on each individual in the host populations, and assume that the stochastic perturbation is a white noise type that is influenced on the natural death rate μ. This is a well-established way of introducing stochastic environmental noise into biologically realistic population dynamic models. The value of our study lies in two aspects:

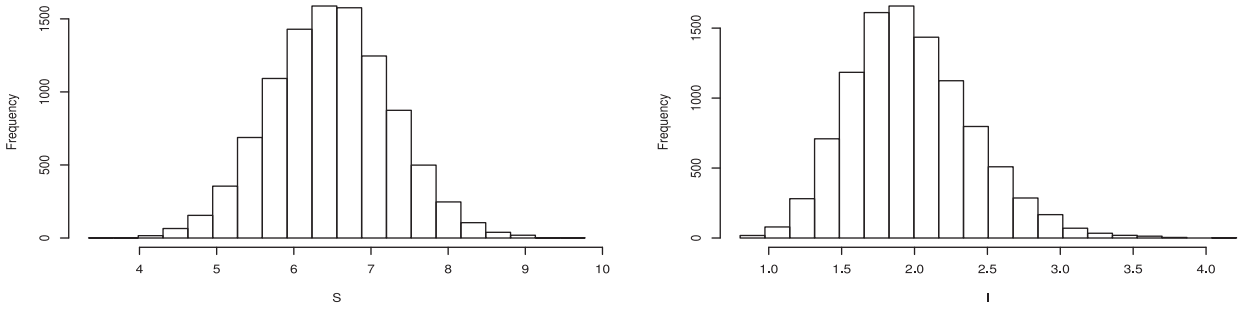
Mathematically, we have introduced two threshold parameters, the stochastic demographic reproduction number $\mathcal{R}_d^s$ and the stochastic basic reproduction number $\mathcal{R}_0^s$, which can be used to utilize in identifying the stochastic extinction and persistence for the SDE model (13). More precisely, if $\mathcal{R}_d^s < 1$, the whole population will go to extinct with probability one (c.f., Theorem 4.1); while if $\mathcal{R}_d^s > 1$, the SDE model (13) exhibits rich and complex dynamics: if $\mathcal{R}_0^s < 1$, the disease will extinction a.s. (c.f., Theorem 4.3(i)), while if $\mathcal{R}_0^s > 1$, the disease will persist a.s. (c.f., Theorem 4.3(ii)).

Epidemiologically, we partially provide the effects of the environment fluctuations (measured by the intensity of volatility ξ or the speed of reversion θ) on the disease spreading to the SDE model (13). We summarize our main findings as follows:

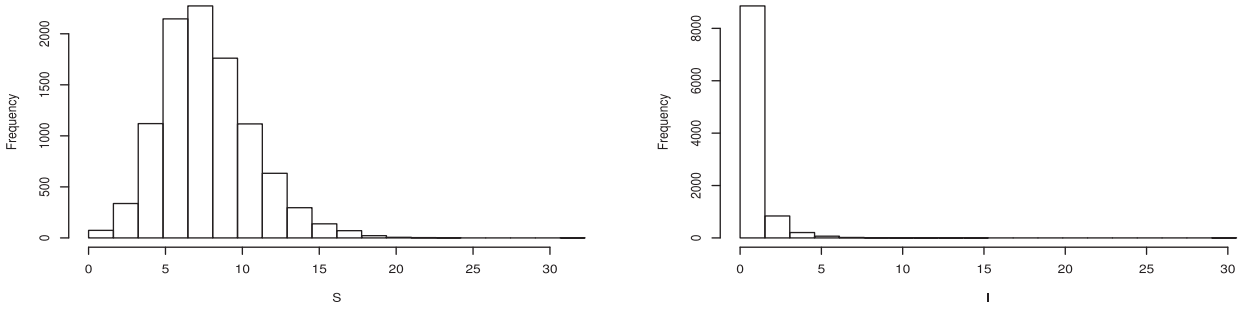
1. *The environment fluctuations can suppress the disease outbreak*: In the case of $\mathcal{R}_d^s > 1$, Theorem 4.3(i) indicates that the extinction of disease in the stochastic model (13) occurs if the basic reproduction number $\mathcal{R}_0^S < 1$. Corollary 2.1 shows that the deterministic model (1) admits a unique endemic equilibrium E^* which is globally asymptotically stable if its basic reproduction number $\mathcal{R}_0 > 1$. From Remark 4.5, we know that $\mathcal{R}_0^S < \mathcal{R}_0$, and hence there may exist a fact that $\mathcal{R}_0^s < 1 < \mathcal{R}_0^s$. This is the case when the deterministic model (1) has an endemic (see Fig. 3) while the stochastic model (13) has disease extinction with probability one (see Fig. 4). This implies that larger the intensity of volatility ξ or the smaller speed of reversion θ can suppress the outbreak of the disease (see Theorem 4.3(i), Remark 4.5(ii-1) and Fig. 4). In contrast, that the smaller intensity of volatility ξ or the the higher speed of reversion θ can enhance the outbreak of the disease (see Theorem 4.3(ii), Remark 4.5(ii-2) and Fig. 7). In one word, large environment fluctuations in I-class can suppress the outbreak of disease.
2. *The stationary distributions governed by $\mathcal{R}_0^S$:* As suggested by numerical results in Figs. 6 and 7, the stochastic model (13) has two kinds of stationary distributions depending on the value of $\mathcal{R}_0^s$: If $\mathcal{R}_0^s < 1$, it has disease–free stationary



(a) $\xi = 0.01$



(b) $\xi = 0.05$



(c) $\xi = 0.2$

Fig. 7. The histograms of the values of the path $S(t)$ and $I(t)$ for the SDE model (13) based on 10,000 stochastic simulations, and all other parameters are taken in Table 1. The initial value of all solutions is (9, 1).

distribution (see Fig. 6) which means that the disease will die out with probability one; while if $\mathcal{R}_0^s > 1$ it has endemic stationary distribution (c.f., Fig. 7). The latter leads to the stochastically persistence of the disease.

In addition, by simple computations, we can easy to know that the SDE model (13) has no disease-free equilibrium point, which is used to define the basic reproduction number. In (24) and (25), we define two threshold parameters $\mathcal{R}_d^s$ and $\mathcal{R}_0^s$, respectively. And in Remarks 4.5 and 4.4, by the epidemiological meaning, we call these two parameters as stochastic demographic reproduction number and the stochastic basic reproduction number, respectively. Actually, these two parameters are

obtained from the proofs of Theorems 4.1 and 4.3. We do think there may be methods to computer these two parameters directly, and this is desirable in future studies.

Furthermore, as introduced in Section 1, our model (1) can be seen as a special case $f = 0$ of Ebert's model [34]:

$$\begin{cases} \dfrac{dS}{dt} = a(S + fI)[1 - c(S + I)] - \beta SI - \mu S, \\ \dfrac{dI}{dt} = \beta SI - (\mu + \alpha)I, \end{cases} \tag{45}$$

where f is the relative fecundity of an infected host I. Epidemiologically, our model (1) can be seen in the special case of that the parasite induced host birth rate reduction as high as 100% (naturally, it is reported in many horizontally transmitted parasites, the parasite induced host birth rate reduction can be as high as 90% [34]), which is to say $f = 0$. The deterministic model (45), resembles a simple SI type model, predicts the existence of a globally attractive positive steady state. And the numerical simulations of the stochastic model corresponding to (45) indicates that extinction of host is a likely outcome in some parameter regions.

Our results can be seen as supplements with that in [34]. On one hand, we give mathematical analysis in details of the deterministic model (1) and its stochastic version (13), and show these two models exhibit complex dynamics (c.f., Theorems 4.1 and 4.3). On the other hand, our numerical results in Section 5 with ecologically meaningful parameters which cited from [34,42] can be used to explain the observed rich outcomes (such as extinction of host, parasite-free and persistence) dependent on parameter values. And the stochastic version of model (45) will be interesting and desirable in our future studies.

Acknowledgments

The authors would like to thank the anonymous referees for very helpful suggestions and comments which led to improvement of our. original manuscript. This research was supported by the National Science Foundation of China (61672013, 11601179, 61772017 & 11761019) and the Natural Science Foundation of the Jiangsu Higher Education Institutions of China (16KJB110003), Huaian Key Laboratory for Infectious Diseases Control and Prevention (HAP201704).

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