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# Global threshold dynamics of a stochastic epidemic model incorporating media coverage

Bin Yang<sup>1</sup>, Yongli Cai<sup>1</sup>, Kai Wang<sup>2</sup> and Weiming Wang<sup>1\*</sup>

\*Correspondence: weimingwang2003@163.com ¹ School of Mathematics Science, Huaiyin Normal University, Huaian, P.R. China Full list of author information is available at the end of the article

#### Abstract

In this paper, we investigate the global threshold dynamics of a stochastic SIS epidemic model incorporating media coverage. We give the basic reproduction number  $\mathcal{R}_0^s$  and establish a global threshold theorem by Feller's test: if  $\mathcal{R}_0^s \leq 1$ , the disease will die out a.s.; if  $\mathcal{R}_0^s > 1$ , the disease will persist a.s. In the case of  $\mathcal{R}_0^s > 1$ , we prove the existence, uniqueness, and global asymptotic stability of the invariant density of the Fokker–Planck equations associated with the stochastic model. Via numerical simulations, we find that the average extinction time decreases with the increase of noise intensity  $\sigma$ , and also find that the increasing  $\sigma$  will be beneficial to control the disease spread. Thus, in order to control the spread of the disease, we must increase the intensity of noise  $\sigma$ .

MSC: 92D30; 60H10; 93E15

**Keywords:** Basic reproduction number; Feller's test; Invariant density; Fokker–Planck equations

#### 1 Introduction

It is now widely believed that environmental variations have a critical influence on the spread of the disease [1-4], and stochastic noise plays an indispensable role in the transmission of diseases, especially in a small population. Therefore, it seems more practical to consider stochastic epidemic models [5-20].

In order to understand the role of media coverage towards the disease transmission dynamics in a random environment, based on the results in [21], Cai et al. [22] studied the following stochastic differential equations (SDE) SIS model with a standard incidence rate:

$$\begin{cases} dS(t) = (\Lambda - \mu S - (\beta_1 - \frac{\beta_2 I}{b+I}) \frac{SI}{S+I} + \gamma I) dt + \sigma_1 S dB_1(t), \\ dI(t) = ((\beta_1 - \frac{\beta_2 I}{b+I}) \frac{SI}{S+I} - (\mu + \gamma) I) dt + \sigma_2 I dB_2(t), \end{cases}$$
(1)

where S(t) and I(t) are the number of susceptible and infective individuals at time t, respectively.  $\beta_1 - \frac{\beta_2 I}{b+I}$  is the effective contact rate,  $\beta_1$  is the usual contact rate without considering the infective individuals,  $\beta_2$  is the maximum reduced contact rate due to the presence of the infected individuals.  $\sigma_i$  (i=1,2) is a real constant and  $\sigma_i^2$  is known as the intensity of



environmental fluctuations,  $B_i(t)$  (i = 1, 2) is the standard one-dimensional independent Wiener process defined over the complete probability space ( $\Omega$ ,  $\mathcal{F}$ , { $\mathcal{F}_t$ } $_{t>0}$ , Prob).

In [22], the authors defined  $R_0^s := \frac{\beta_1}{\mu + \gamma} - \frac{\sigma_2^2}{2(\mu + \gamma)}$  and obtained the following results by using the method of stochastic stability [23]:

(A1) If one of the following conditions

$$R_0^s < 1$$
 and  $\sigma_2^2 < 2\beta_1$ , or  $\sigma_2^2 \ge 2\beta_1$ ,

holds, then the disease will die out with probability one (Theorem 3.1, [22]).

(A2) If  $R_0^s > 1$ , then the disease will persist with probability one (Theorem 4.1, [22]).

Furthermore, based on the results in [22], by using the theory of Markov semigroup and asymptotic properties [9, 24], Guo et al. [25] studied the following SDE SIS model with bistable incidence rate:

$$\begin{cases} dS(t) = \left[ \Lambda - \mu S(t) - (\beta_1 - \frac{\beta_2 I(t)}{m + I(t)}) S(t) I(t) + \gamma I(t) \right] dt + \sigma S(t) dB(t), \\ dI(t) = \left[ (\beta_1 - \frac{\beta_2 I(t)}{m + I(t)}) S(t) I(t) - (\mu + \gamma) I(t) \right] dt + \sigma I(t) dB(t), \end{cases}$$
(2)

and obtained that:

(B1) If

$$R_0^s := \frac{\Lambda \beta_1}{\mu(\mu + \gamma)} - \frac{\sigma^2}{2(\mu + \gamma)} < 1,$$

then the disease will die out with probability one (Theorem 3.4, [25]).

(B2) If

$$R_0^s > 1$$
 and  $\sigma^2 < 2\mu \min\{1, A\}$ 

hold, then the stochastic process (S(t), I(t)) has a unique stationary distribution (Theorem 3.7, [25]).

It is well known that epidemic threshold theorem holds for most deterministic compartmental epidemic models by the basic reproduction number  $R_0$  [26]: if  $R_0 < 1$ , there is a disease-free equilibrium which is globally asymptotically stable; if  $R_0 > 1$ , there exists an endemic equilibrium which is globally asymptotically stable. However, in [A1] for SDE model (1), there is an extra condition  $\sigma_2^2 < 2\beta_1$ , and in [B2] for SDE model (2), there is an extra condition  $\sigma^2 < 2\mu \min\{1,A\}$ .

There naturally comes a question: Is there any global threshold theorem for a stochastic epidemic model (e.g., SDE model (1) or (2)) incorporating media coverage?

Thanks to the insightful work of Xu [10], in this paper, we will focus on the global threshold dynamics for the following SDE epidemic model incorporating media coverage:

$$\begin{cases} dS(t) = (\mu N - \mu S - (\beta_1 - \frac{\beta_2 I}{b+I}) \frac{SI}{S+I} + \gamma I) dt - \frac{\sigma SI}{S+I} dB(t), \\ dI(t) = ((\beta_1 - \frac{\beta_2 I}{b+I}) \frac{SI}{S+I} - (\mu + \gamma) I) dt + \frac{\sigma SI}{S+I} dB(t). \end{cases}$$
(3)

Since S(t) + I(t) = N, which is a constant, SDE model (3) can degenerate into the following one-dimensional model:

$$dI(t) = IF(I) dt + \sigma I \left(1 - \frac{I}{N}\right) dB(t), \tag{4}$$

where

$$F(I) := \left(\beta_1 - \frac{\beta_2 I}{b+I}\right) \left(1 - \frac{I}{N}\right) - (\mu + \gamma). \tag{5}$$

Particularly, if  $\sigma = 0$  in (4), i.e., without environmental noise, we can obtain

$$dI(t) = IF(I) dt. (6)$$

For simplicity, we call model (6) a deterministic model corresponding to SDE model (4).

One goal of this paper is to establish a global threshold theorem for SDE model (4). We will prove that the basic reproduction number  $\mathcal{R}_0^s$  can be used to govern the stochastic dynamics of SDE model (4).

The other goal is to further study the invariant density of process I(t). Many long-term asymptotic properties of dynamical systems or random dynamical systems can be described in terms of invariant measure [27] and the density function with respect to Lebesgue measure of the marginals of an invariant measure that can be called an invariant density [28]. If invariant density is  $\mathcal{L}^1$  on a set  $\Omega$ , it satisfies the Fokker–Planck equations (FPE) in the interior of  $\Omega$  [29]. Hence, we will investigate the FPE associated with (4) and solve the invariant density.

This paper is organized as follows. In Sect. 2, we present the global stochastic threshold theorem. In Sect. 3, in the case of disease persistence, we derive the existence, uniqueness, global stability, and an explicit formula of an invariant density of the Fokker–Planck equation associated with (4). In Sect. 4, we give some numerical examples to show the complicated stochastic dynamics of the model. And in the last section, Sect. 5, we provide a brief discussion and the summary of our main results.

#### 2 Stochastic threshold theorem

In this section, we will focus on the stochastic threshold theorem for model (4). First of all, we state the global existence of the uniqueness and boundedness of the positive solution of model (4).

**Theorem 2.1** For any given initial value  $I_0 \in (0, N)$ , SDE (4) has a unique global positive solution  $I(t) \in (0, N)$  for all  $t \ge 0$  with probability one, namely

$$\mathbb{P}\{I(t) \in (0,N) : \forall t \geq 0\} = 1.$$

The proof of Theorem 2.1 is similar to that in [22] or [25]. So we omit it here.

Next, similar to [22, 25] and [8], we define the basic reproduction number  $\mathcal{R}_0^s$  for SDE model (4) as follows:

$$\mathcal{R}_0^s := \frac{\beta_1}{\mu + \gamma} - \frac{\sigma^2}{2(\mu + \gamma)} = R_0 - \frac{\sigma^2}{2(\mu + \gamma)},\tag{7}$$

where  $R_0 := \frac{\beta_1}{\mu + \gamma}$  is the basic reproduction number of the deterministic model (6).

#### Theorem 2.2

(i) If  $\mathcal{R}_0^s \leq 1$ , for any given initial value  $I_0 \in (0, N)$ ,

$$\mathbb{P}\Big\{\lim_{t\to\infty}I(t)=0\Big\}=1.$$

Namely, the disease will go extinct with probability one.

(ii) If  $\mathcal{R}_0^s > 1$ , for any given initial value  $I_0 \in (0, N)$ ,

$$\mathbb{P}\left\{\sup_{0 \le t < \infty} I(t) = N\right\} = \mathbb{P}\left\{\inf_{0 \le t < \infty} I(t) = 0\right\} = 1.$$

In particular, the process I(t) is recurrent: for every  $\theta \in (0, N)$ , we have

$$\mathbb{P}\big\{I(t)=\theta:\exists t\in[0,\infty)\big\}=1.$$

Namely, the disease will persist with probability one.

Proof Define

$$Y(t) = \log \frac{I(t)}{N - I(t)},$$

then

$$I(t) = \frac{Ne^{Y(t)}}{1 + e^{Y(t)}},\tag{8}$$

hence

$$\lim_{Y \to -\infty} I(t) = 0, \qquad \lim_{Y \to +\infty} I(t) = N. \tag{9}$$

Using Itô's formula for Y(t), we have

$$dY(t) = \left(\frac{NF(I)}{N-I} + \frac{1}{2}\left(\frac{1}{(N-I)^2} - \frac{1}{I^2}\right)\sigma^2(N-I)^2I^2\right)dt + \sigma dB(t)$$

$$= \left(\beta_1 - (\mu + \gamma) - \frac{\sigma^2}{2} - \frac{\beta_2 I}{b+I} - \frac{(\mu + \gamma)I}{N-I} + \frac{\sigma^2}{N}I\right)dt + \sigma dB(t). \tag{10}$$

Substituting (8) into (10), we obtain

$$dY(t) = (\mu + \gamma) \left( \mathcal{R}_0^s - 1 - \frac{\beta_2 N e^Y}{(\mu + \gamma)(b + b e^Y + N e^Y)} - e^Y + \frac{\sigma^2 e^Y}{(\mu + \gamma)(1 + e^Y)} \right) dt + \sigma dB(t).$$
(11)

Following [30, 31], the scale function defined for (11) is given by

$$\psi(x) = \int_0^x \phi(\xi) \, d\xi,$$

with

$$\begin{split} \phi(\xi) &= \exp\left\{-\frac{2(\mu + \gamma)}{\sigma^2} \int_0^{\xi} \left(\mathcal{R}_0^s - 1 - \frac{\beta_2 N e^s}{(\mu + \gamma)(b + b e^s + N e^s)} - e^s \right. \\ &+ \frac{\sigma^2 e^s}{(\mu + \gamma)(1 + e^s)}\right) ds \right\} \\ &= \exp\left\{-\frac{2(\mu + \gamma)}{\sigma^2} \left(\mathcal{R}_0^s - 1\right) \xi + \frac{2(\mu + \gamma)}{\sigma^2} \left(e^{\xi} - 1\right) + 2\log 2 - 2\log\left(1 + e^{\xi}\right) \right. \\ &+ \frac{2\beta_2 N}{\sigma^2 (b + N)} \left(\log\left(b + (b + N)e^{\xi}\right) - \log(2b + N)\right) \right\}. \end{split}$$

It is easy to see that  $\psi(\infty) = \infty$ . While if  $\mathcal{R}_0^s > 1$ , then  $\psi(-\infty) = -\int_0^\infty \phi(-\xi) \, d\xi = -\infty$ . On the other hand, if  $\xi > 0$  and  $\mathcal{R}_0^s \le 1$ , then

$$\phi(-\xi) = \exp\left\{\frac{2(\mu + \gamma)}{\sigma^2} \left(\mathcal{R}_0^s - 1\right) \xi + \frac{2(\mu + \gamma)}{\sigma^2} \left(e^{-\xi} - 1\right) + 2\log 2 - 2\log\left(1 + e^{-\xi}\right) + \frac{2\beta_2 N}{\sigma^2 (b + N)} \left(\log\left(b + (b + N)e^{-\xi}\right) - \log(2b + N)\right)\right\} < 4.$$

Hence, if  $\mathcal{R}_0^s \le 1$ ,  $\psi(-\infty) = -\int_0^\infty \phi(-\xi) \, d\xi \ge -4 \int_0^\infty \, d\xi = -\infty$ .

It follows from [31, Propositions 5.22] and [10, Lemma A.2] that we end the proof. □

#### 3 The properties of invariant density

In this section, in the case of  $\mathcal{R}_0^s > 1$ , we will focus on the properties of the invariant density of the process I(t) of SDE model (4). Instead of studying SDE model (4) directly, we investigate the associated Fokker–Planck equation (FPE) with (4), which is given by

$$\frac{\partial p(t,x)}{\partial t} = -\frac{\partial}{\partial x} \left\{ x F(x) p(t,x) \right\} + \frac{1}{2} \sigma^2 \frac{\partial^2}{\partial x^2} \left( x^2 \left( 1 - \frac{x}{N} \right)^2 p(t,x) \right). \tag{12}$$

From (12), we denote by  $\{\mathbb{P}(t)\}_{t\geq 0}$  the Markov semigroup. And the following theorem gives the existence, uniqueness, and asymptotic stability of an invariant density of the Markov semigroup  $\{\mathbb{P}(t)\}$ .

**Theorem 3.1** If  $\mathcal{R}_0^s > 1$ , then there exists a unique invariant probability measure  $v_{\sigma}^s$  for (12) which has the density  $p_{\sigma}^s$  with respect to the Lebesgue measure. Moreover,

(i) the process I(t) has the ergodic properties, i.e., for any  $v_{\sigma}^{s}$ -integrable function G,

$$\mathbb{P}_{I_0}\left(\lim_{t\to\infty}\frac{1}{t}\int_0^t G(I_\tau)\,d\tau = \int_0^K G(y)v_\sigma^s(dy)\right) = 1$$

*for all*  $I_0 \in (0, N)$ ;

(ii) the invariant density  $p_{\sigma}^{s}$  is globally asymptotically stable in the sense that

$$\lim_{t\to\infty}\int_0^K \left|\mathbb{P}(t)g(x)-p_\sigma^s(x)\right|dx=0,\quad\forall g\in L^1_+(0,N),$$

and

$$L^{1}_{+}\big((0,N)\big) := \left\{ w \in L^{1}(\mathcal{R}) : \int_{0}^{N} w(x) \, dx = 1, w(x) = 0 \text{ for } x \ge N \text{ or } x \le 0, \right.$$

$$and \ w(x) \ge 0 \text{ for } x \in \mathcal{R} \right\}.$$

(iii) the unique invariant density  $p_{\sigma}^{s}$  is given by

$$p_{\sigma}^{s}(x) := CN^{3 - \frac{2\beta_{2}N}{\sigma^{2}(b+N)}} \frac{x^{c_{0}(\mathcal{R}_{0}^{s}-1)-1}(x+b)^{-\frac{2\beta_{2}N}{\sigma^{2}(b+N)}}}{(N-x)^{c_{0}(\mathcal{R}_{0}^{s}-1)+3 - \frac{2\beta_{2}N}{\sigma^{2}(b+N)}}} e^{-\frac{c_{0}x}{N-x}}$$
(13)

with

$$C^{-1} = \int_0^\infty \left( b + (b+N)e^{\xi} \right)^{-\frac{2N\beta_2}{\sigma^2(b+N)}} \left( e^{\xi} + 1 \right)^2 e^{c_0(\mathcal{R}_0^s - 1)\xi - c_0 e^{\xi}} d\xi, \tag{14}$$

and  $c_0 = \frac{2(\mu + \gamma)}{\sigma^2}$ .

*Proof* First of all, we need to study the following FPE associated with SDE (11):

$$\frac{\partial u(t,\xi)}{\partial t} = -\frac{\partial}{\partial \xi} \left\{ (\mu + \gamma) \left( \mathcal{R}_0^s - 1 - \frac{\beta_2 N e^{\xi}}{(\mu + \gamma)(b + b e^{\xi} + N e^{\xi})} \right) - e^{\xi} + \frac{\sigma^2 e^{\xi}}{(\mu + \gamma)(1 + e^{\xi})} \right\} + \frac{1}{2} \sigma^2 \frac{\partial^2 u(t,\xi)}{\partial \xi^2}.$$
(15)

We next prove assertions (i) and (ii). Since Y(t) of (11) is conservative and non-degenerate (i.e.,  $\frac{1}{2}\sigma^2 > 0$ ), by [32, p. 153], there exists a unique classical fundamental solution to (15). Thus it follows from [33, p. 365 and p. 368] that there exists a generalized solution  $u(t, \xi) \in L^1_+(\mathcal{R})$  for all t > 0,  $\xi \in \mathcal{R}$  provided that the initial density  $u_0 \in L^1_+(\mathcal{R})$ .

Motivated by [10], we define a Lyapunov function V:

$$V(\xi) = e^{-\alpha \xi} + \xi^2,$$

where  $\alpha = \frac{\mu + \gamma}{\sigma^2} (\mathcal{R}_0^s - 1) > 0$ . Hence by [33, Theorem 11.9.1], we have the uniqueness and global asymptotic stability of the invariant density  $u_\sigma^s$ . From [10, Theorem A.7], we have assertions (i) and (ii).

We now prove assertion (iii). The stationary FPE associated with SDE (11) is given by

$$\frac{1}{2}\sigma^{2}\frac{\partial^{2}u_{\sigma}^{s}(\xi)}{\partial\xi^{2}} = \frac{\partial}{\partial\xi}\left\{ (\mu + \gamma)\left(\mathcal{R}_{0}^{s} - 1 - \frac{\beta_{2}Ne^{\xi}}{(\mu + \gamma)(b + be^{\xi} + Ne^{\xi})}\right) - e^{\xi} + \frac{\sigma^{2}e^{\xi}}{(\mu + \gamma)(1 + e^{\xi})}\right\} u_{\sigma}^{s}(\xi).$$
(16)

Rewrite system (16) in the following simpler form:

$$\frac{dy(\xi)}{d\xi} - a(\xi)y(\xi) = -c,\tag{17}$$

where  $y(\xi) = u_{\sigma}^{s}(\xi)$ , c is a constant and

$$a(\xi) = \frac{2(\mu + \gamma)}{\sigma^2} \left( \mathcal{R}_0^s - 1 - \frac{\beta_2 N e^{\xi}}{(\mu + \gamma)(b + b e^{\xi} + N e^{\xi})} - e^{\xi} + \frac{\sigma^2 e^{\xi}}{(\mu + \gamma)(1 + e^{\xi})} \right)$$
$$= c_0 \left( \mathcal{R}_0^s - 1 - \frac{2\beta_2 N e^{\xi}}{c_0 \sigma^2 (b + b e^{\xi} + N e^{\xi})} - e^{\xi} + \frac{2e^{\xi}}{c_0 (1 + e^{\xi})} \right).$$

The solution of (17) is

$$y(\xi) = u_{\sigma}^{s}(\xi) = A(\xi) \left( K - c \int_{1}^{\xi} \frac{1}{A(z)} dz \right)$$

with

$$A(\xi) = (b + (b + N)e^{\xi})^{-\frac{2N\beta_2}{\sigma^2(b+N)}} (e^{\xi} + 1)^2 e^{c_0(\mathcal{R}_0^s - 1)\xi - c_0e^{\xi}},$$

where  $c_0 = \frac{2(\mu + \gamma)}{\sigma^2}$ .

It follows from the conditions  $u^s_{\sigma}(\xi) \geq 0$ ,  $\int_0^\infty u^s_{\sigma}(\xi) d\xi = 1$  that c = 0 and

$$K^{-1} = \int_0^\infty \left( b + (b+N)e^{\xi} \right)^{-\frac{2N\beta_2}{\sigma^2(b+N)}} \left( e^{\xi} + 1 \right)^2 e^{c_0(\mathcal{R}_0^s - 1)\xi - c_0 e^{\xi}} d\xi.$$

Simple computations reveal that

$$\begin{split} K^{-1} &< b^{-\frac{2N\beta_2}{\sigma^2(b+N)}} \left( c_0^{-(c_0(\mathcal{R}_0^s-1)} \Gamma\left(c_0 \big(\mathcal{R}_0^s-1\big) + 2\right) + 2 c_0^{-(c_0(\mathcal{R}_0^s-1)+1)} \right. \\ & \cdot \Gamma\left(c_0 \big(\mathcal{R}_0^s-1\big) + 1\right) + c_0^{-c_0(\mathcal{R}_0^s-1)} \Gamma\left(c_0 \big(\mathcal{R}_0^s-1\big)\right) \right) \\ &= b^{-\frac{2N\beta_2}{\sigma^2(b+N)}} c_0^{-c_0(\mathcal{R}_0^s-1)} \big(\mathcal{R}_0^{s\,2} + c_0^{-1} \big(\mathcal{R}_0^s-1\big)\big) \Gamma\left(c_0 \big(\mathcal{R}_0^s-1\big)\right). \end{split}$$

Note that  $\Gamma(c_0(\mathcal{R}_0^s - 1)) > 0$  for  $\mathcal{R}_0^s > 1$ , and thus C defined in (14) is finite. We can conclude that  $u_\sigma^s$  is an invariant which has the form

$$u_{\sigma}^{s}(\xi) := C(b + (b + N)e^{\xi})^{-\frac{2N\beta_{2}}{\sigma^{2}(b+N)}} (e^{\xi} + 1)^{2} e^{c_{0}(\mathcal{R}_{0}^{s} - 1)\xi - c_{0}e^{\xi}}.$$
 (18)

In the same way as in the proof of [10, Theorem A.6], we have

$$p_{\sigma}^{s}(x) = \frac{N}{x(N-x)} u_{\sigma}^{s} \left(\log \frac{x}{N-x}\right).$$

This, together with (18), implies assertion (iii).

#### 4 Numerical results via disease dynamics

In this section, we give some numerical results to show complex disease dynamic outcomes of SDE model (4) by using Milstein's method [34], and the numerical scheme for model (4) under consideration is given by

$$\begin{split} I_{k+1} &= I_k + I_k \left[ \left( \beta_1 - \frac{\beta_2 I_k}{b + I_k} \right) (1 - I_k/N) - (\mu + \gamma) \right] \Delta t + \sigma I_k (1 - I_k/N) \eta_k \sqrt{\Delta t} \\ &\quad + \frac{\sigma^2}{2} \left( I_k (1 - I_k/N) \right)^2 \left( \eta_k^2 - 1 \right) \Delta t, \end{split}$$

where  $\eta_k$  (k = 1, 2, ..., n) are independent Gaussian random variables N(0, 1),  $\Delta t$  is the time step size.

From (7), we note that  $\mathcal{R}_0^s = R_0 - \frac{\sigma^2}{2(\mu + \gamma)} < R_0$ , if  $R_0 < 1$ , then  $\mathcal{R}_0^s < 1$ . We can know that if  $R_0 < 1$ , I(t) goes to extinction for the deterministic model (4) (see [21]); and from Theorem 2.1, I(t) almost surely tends to zero exponentially with probability one for the stochastic model (4). Therefore we only consider the case of  $R_0 > 1$ .

Following [22, 25], the choice for the following parameters remains unaltered:

$$\beta_1 = 0.15$$
,  $\beta_2 = 0.1$ ,  $\mu = 0.05$ ,  $\gamma = 0.02$ ,  $b = 10$ ,  $N = 1000$  (19)

and the initial value is  $I_0 = 10$ . In this case,  $R_0 = 2.143 > 1$ , and the deterministic model (6) has an unstable disease-free equilibrium  $E_0 = 0$  and a unique globally stable endemic equilibrium  $E^* = 34.4494$ .

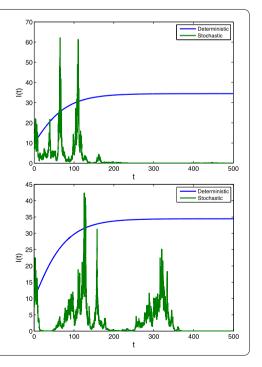
Next we will focus on the role of noise intensity  $\sigma$  on the resulting dynamics for SDE model (4).

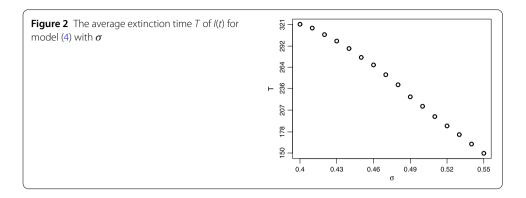
#### 4.1 Stochastic disease-free dynamics

First of all, we adopt  $\sigma = 0.405$ , in this case,  $\mathcal{R}_0^s = 0.97125 < 1$ . From Theorem 2.1(i), we know that the disease I(t) will go extinct with probability one.

In Fig. 1, we show the stochastic disease dynamics of the evolution of the single path of I(t) obtained from two different numerical simulations run with the same parameters. One can see that I(t) is strongly oscillatory at the beginning and finally dies out with probability one. Easy to know that, in these cases above, I(t) tends to the disease-free equilibrium  $E_0 = 0$  of the deterministic model (6) almost surely at last. It should be noted that, for model (6),  $R_0 = 2.143 > 1$ ,  $E_0 = 0$  is unstable. Hence we can conclude that, in this case, environmental noise can make unstable  $E_0$  to a stable one.

**Figure 1** The evolution of a single path of l(t) for model (4) with  $\sigma = 0.405$  and its corresponding deterministic model (6), and all other parameters are taken in (19). The initial value is  $l_0 = 10$ . The time unit is day





Furthermore, we repeat 10,000 simulations with the same parameters as in Fig. 1, we can calculate the average extinction time for I(t), and the results are shown in Fig. 2. For example, when  $\sigma = 0.405$ , the average extinction time for I(t); when  $\sigma = 0.45$ , it is 278.3943. We can conclude that the average extinction time decreases with the increase of noise intensity  $\sigma$ .

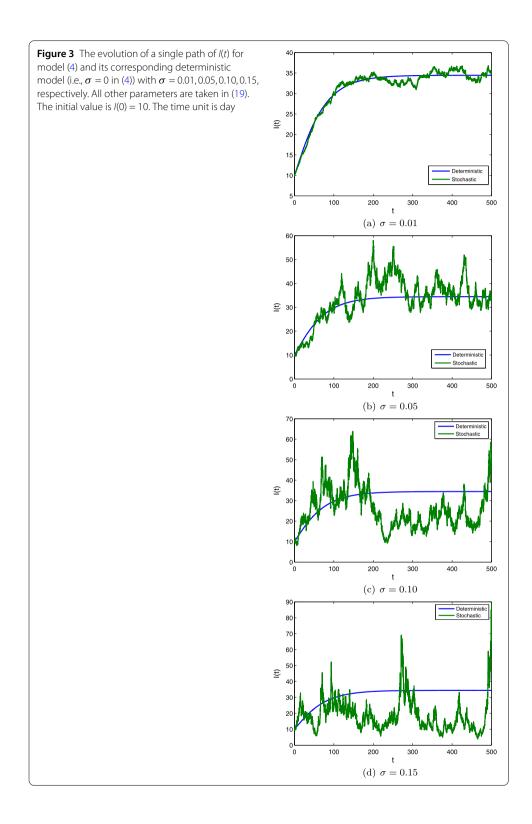
#### 4.2 Stochastic endemic dynamics

In this subsection, we will focus on the stochastic endemic dynamics of (4) in the case of  $\mathcal{R}_0^s > 1$ . For this reason, we choose  $\sigma = 0.01, 0.05, 0.10, 0.15$  implies that  $\mathcal{R}_0^s = 2.142, 2.125, 2.071, 1.982$ , respectively. From Theorem 2.1(ii), we can conclude that the disease will persist almost surely. In Fig. 3, we show the single path of I(t) for model (4) and its corresponding deterministic model (6) with  $\sigma = 0.01, 0.05, 0.10, 0.15, 0.25, 0.35$ , respectively, and we can see that the solutions I(t) of SDE model (4) fluctuate around the endemic equilibrium  $E^* = 34.4494$  of the deterministic model (6), respectively. In addition, we can find that the bigger noise intensity  $\sigma$ , the stronger oscillatory I(t).

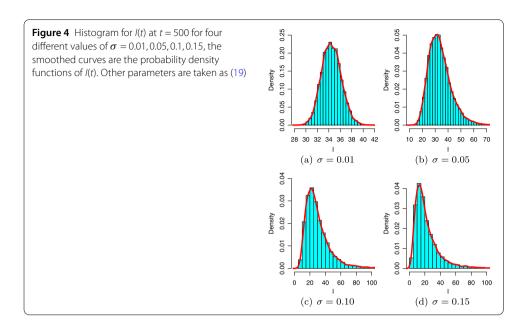
For the sake of learning the effects of the intensity of noise  $\sigma$  on the stochastic disease dynamics of SDE model (4), we have repeated the simulation 10,000 times, keeping the same parameters as in Fig. 3 and never observing any extinction scenario up to t = 500. These results are respectively confirmed by the histograms and the probability density functions in Fig. 4, showing the stationary distributions of I(t) at t = 500 for model (4). And the numerical method for them can be found in [9].

From Fig. 4, one can see that the solution to SDE model (4) in the persistent case also suggests for lower  $\sigma$  (e.g.,  $\sigma$  = 0.01 and 0.05), the amplitude of fluctuation is slightly and the oscillations to be more symmetrically distributed (cf. Figs. 4(a) and 4(b)), and the fluctuations are reflected at the stationary distributions. While for higher  $\sigma$  (e.g.,  $\sigma$  = 0.1 and 0.15), the amplitude of fluctuation is remarkable and the distributions of the solutions are skewed (cf. Figs. 4(c) and 4(d)) and the fluctuations are also reflected at the stationary distributions.

Furthermore, in the case of  $\sigma$  = 0.01 and 0.05, the distribution of I(t) appears closer to a normal distribution (see Figs. 4(a) and 4(b)). Simple computations show that, when  $\sigma$  = 0.01, the distribution of I(t) closely obeys the normal distribution  $N(34.4577, 1.6995^2)$ , and the values less than one standard deviation away from the mean account (32.7582, 36.1572) for 67.94% of the set; while two standard deviations from the mean account (31.1530, 37.7624) for 94.92%; and three standard deviations account (30.0517, 38.8637) for 98.99% (see Fig. 5(a)). Simple calculations show that the skewness (i.e., the measure of the asymmetry of the probability distribution of a real-valued random variable about its mean) in



this case is 0.1662598, which is a positive skew. And in the case of  $\sigma$  = 0.05, the distribution of I(t) closely obeys N(33.7969, 8.8664<sup>2</sup>), and the values less than three standard deviations away from the mean account (8.0924, 59.5015) for 98.75% (see Fig. 5(b)), the positive skewness is 1.045055.

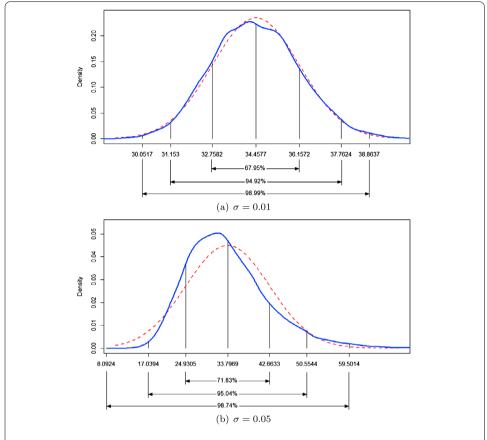


In order to understand the effect of the intensity of noise  $\sigma$  on the skewness of the distribution of I(t), we show the four probability density functions with  $\sigma = 0.01, 0.05, 0.1$ , and 0.15 in Fig. 6. Easy to see that, as  $\sigma$  increases, the means of I(t) become smaller and smaller, and the positive skewness of the distributions of I(t) becomes bigger and bigger.

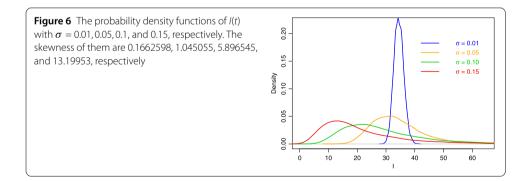
#### 5 Concluding remarks

In this paper, we investigate the global threshold dynamics of the SDE SIS epidemic model (4) incorporating media coverage. After defining the basic reproduction number  $\mathcal{R}_0^s$ , we establish a stochastic threshold theorem by using Feller's test for explosions of solutions to one-dimensional SDE model (4) (see Theorem 2.2). It is worthy to note that in the proof of this main result, we employ Feller's test [10, 30, 31]. This is very different from the previous results by constructing Lyapunov function to prove the threshold theorem (see, [8, 22, 25]). And in the case of  $\mathcal{R}_0^s$ , i.e., the disease persists with probability one, by studying the FPE associated with SDE model (4), we prove the existence, uniqueness, and global asymptotic stability of the invariant density of the FPE (see Theorem 3.1), which can be useful for us to understand the profile of the distribution of the process I(t).

Via numerical simulations, in the case of  $\mathcal{R}_0^s \leq 1$ , we find that the average extinction time decreases with the increase of noise intensity  $\sigma$ . And in the case of  $\mathcal{R}_0^s > 1$ , we find that the solutions I(t) of SDE model (4) fluctuate around the endemic equilibrium  $E^* = 34.4494$  of the deterministic model (6), and finally the distribution of I(t) seems like a normal stationary distribution. More precisely, for lower  $\sigma$  (e.g.,  $\sigma = 0.01$  and 0.05), the distribution of I(t) appears closer to a normal distribution (see Figs. 5(a) and 5(b)); while for higher  $\sigma$  (e.g.,  $\sigma = 0.1$  and 0.15), the amplitude of fluctuation is remarkable and the distribution of the solutions is positively skewed (cf. Figs. 4(c), 4(d), and 6). Obviously, variance increases with the increase of noise intensity  $\sigma$ , this is the main cause of positive skew distribution (see Figs. 5, 6 and also Figs. 4(c) and 4(d)). In this sense, we can claim that bigger noise  $\sigma$  will be beneficial to make I(t) stay away from the endemic equilibrium  $E^*$  of the deterministic model (6). In other words, increasing  $\sigma$  will be beneficial to control



**Figure 5** Probability density function for I(t) at t = 500 with  $\sigma = 0.01$  (**a**),  $\sigma = 0.05$  (**b**), the dotted red curves are the normal distributions **N**(34.4577, 1.6995<sup>2</sup>) (**a**) and **N**(33.7969, 8.8664<sup>2</sup>) (**b**), respectively. Other parameters are taken as (19)



the disease spread. Thus, in order to control the spread of the disease, we must increase the intensity of noise  $\sigma$ .

On the other hand, in Theorem 2.2, we give the global threshold dynamics by Feller's test for the explosions of solutions to one-dimensional SDE model (4). Unfortunately, this method cannot be used to study a two-dimensional SDE model (e.g., (1) or (2)). And the global threshold dynamics of a high-dimensional SDE model, e.g., (1) or (2), is desirable in the future study.

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#### **Competing interests**

The authors declare that they have no competing interests.

#### Authors' contributions

All authors contributed equally to the writing of this paper. All authors read and approved the final manuscript.

#### **Author details**

<sup>1</sup> School of Mathematics Science, Huaiyin Normal University, Huaian, P.R. China. <sup>2</sup> Department of Medical Engineering and Technology, Xinjiang Medical University, Urumqi, P.R. China.

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