

Research Article Analysis of a Stochastic SIR Model with Vaccination and Nonlinear Incidence Rate

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Received 1 March 2019; Accepted 1 August 2019; Published 21 August 2019

Academic Editor: Elena Kaikina

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We expand an SIR epidemic model with vertical and nonlinear incidence rates from a deterministic frame to a stochastic one. The existence of a positive global analytical solution of the proposed stochastic model is shown, and conditions for the extinction and persistence of the disease are established. The presented results are demonstrated by numerical simulations.

1. Introduction

Mathematical models play an important role in the analysis and control of infectious diseases, and thus effective measures can be taken to reduce its transmission as much as possible. The study of mathematical models in epidemiology has received much attention from many scientists, and some novel results are obtained [1–4]. Recently, stochastic analysis has widely been applied in mathematical modeling in biology [5–9].

For compartmental mathematical models, the total population is divided into three classes, namely, susceptible population S(t), infected population I(t), and recover population R(t). For more details, see [10]. For some diseases, such as AIDS, rubella, varicella, hepatitis B, hepatitis, syphilis, and mumps, it is one of the main transmission modes that infected mothers infect their unborn or newborn offsprings, called vertical transmission [11]. Meng and Chen [12] proposed an SIR epidemic model with vaccination and vertical transmission mode as follows:

$$\begin{cases} \frac{dS}{dt} = -\beta SI - bS + (1 - m)pdI + b(1 - m)(S + R), \\ \frac{dI}{dt} = \beta SI - (pd + r)I, \\ \frac{dR}{dt} = rI - bR + dmpI + mb(S + R), \end{cases}$$
(1)

where *b* is the mortality rate in the susceptible and the recovered individuals and *d* is the mortality rate in the infective individuals. The constants *p* and *q* (p + q = 1) are vertical transmission rates, namely, *p* and *q* are, respectively, the proportion of the offspring of infective parents that are susceptible individuals and the rest are born infected. The arrival of newborns constitutes a recruitment rate of b(S + R) into the susceptible individuals and q dI into the infectious individuals. $m(0 \le m \le 1)$ is the successful vaccination proportion to the newborn from *S* and *R*, *r* is the recover rate in the infective individuals into recovered individuals, and β is the contact rate. System (1) has a basic reproduction number R_0 defined by

$$R_0 = \frac{(1-m)\beta}{pd+r}.$$
 (2)

On the contrary, environmental fluctuations have great influence on all aspects of real life. The aim of this work is to study the effect of these environmental fluctuations on the transmission rate β . We assume that the stochastic perturbations are of white noise type, that is, $\beta \longrightarrow \beta + \sigma B(t)$, where B(t) is a Brownian motion and σ is the intensity. Then, the stochastic version corresponding to the deterministic model (1) with general incidence rate is as follows:

$$\begin{cases} dS = \left(-\frac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI} - bS + (1-m)pdI + b(1-m)(S+R)\right)dt - \frac{\sigma SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI}dB(t), \\ dI = \left(\frac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI} - (pd+r)I\right)dt + \frac{\sigma SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI}dB(t), \\ dR = (rI - bR + dmpI + mb(S+R))dt, \end{cases}$$
(3)

where B(t) is the independent standard Brownian motions defined on a complete probability space $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t\geq 0}, P)$ with a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ satisfying the usual conditions (i.e., it is increasing and right continuous while \mathcal{F}_0 contains all Pnull sets).

 $g(S, I) = \beta SI/(1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI)$ is the incidence rate, where $\alpha_1, \alpha_2, \alpha_3 \ge 0$. In addition, the incidence rate is the number of new infected situations by population in a determined time period. To model the disease transmission process, several authors employ the following bilinear incidence rate βSI , where β is a positive constant [13]. Yet, there exist many forms of nonlinear incidence rate and every form presents some advantages [14–16]. It is very important to note that $g(S, I) = \beta SI/(1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI)$ is a general form which represents mutual interference between *S* and *I*. in particular cases:

- (1) If $\alpha_1 = \alpha_2 = \alpha_3 = 0$, g(S, I) becomes a bilinear incidence rate
- (2) If $\alpha_1 = \alpha_2 = 0$ or $\alpha_1 = \alpha_3 = 0$, g(S, I) becomes a saturated incidence rate [17]
- (3) If α₃ = 0, g(S, I) becomes a Beddington-DeAngelis functional response [18]
- (4) If $\alpha_3 = \alpha_1 \alpha_2$, g(S, I) becomes the Crowley-Martin functional response presented in [19]

Next, we consider the *d*-dimensional stochastic system:

$$dx(t) = f(x(t), t)dt + g(x(t), t)dB(t),$$
 (4)

where f(x,t) is a function in IR^d defined in $[t_0, +\infty)$ and g(x,t) is a $d \times m$ matrix, and f and g are locally Lipschitz functions in x. $\{B(t)\}_{t\geq 0}$ is a d-dimensional standard Wiener process defined on the above probability space.

Denote by $\mathscr{C}^{2,1}(IR^d \times [t_0, +\infty); IR_+)$ the family of all nonnegative functions U(x, t) defined on $IR^d \times [t_0, +\infty)$ such that they are continuously twice differentiable in x and once in t. The differential operator \mathscr{L} [16] associated with (4) is defined by

$$\mathcal{L} = \frac{\partial}{\partial t} + \sum_{i=1}^{d} f_i(x, t) \cdot \frac{\partial}{\partial x_i} + \frac{1}{2} \sum_{i,j=1}^{d} \left[g^T(x, t) g(x, t) \right]_{ij} \cdot \frac{\partial}{\partial x_i \partial x_j}.$$
(5)

If the differential operator \mathscr{L} acts on a function $U \in \mathscr{C}^{2,1}(IR^d \times [t_0, +\infty); IR_+)$, then

$$\mathcal{L}U = U_t(x,t) + U_x(x,t)f(x,t) + \frac{1}{2}\mathrm{Trac}[g(x,t)^T U_{xx}(x,t)g(x,t)],$$
(6)

where $U_t(x,t) = \partial U/\partial t$, $U_x(x,t) = (\partial U/\partial x_1, \dots, \partial U/\partial x_d)$, and $U_{xx}(x,t) = (\partial^2 U/\partial x_i \partial x_i)$.

The organization of this paper is as follows. In Section 2, we show the existence of unique positive global solution to the given SDE system. Extinction and persistence in mean results are explored in Section 3 and Section 4, respectively. In Section 5, the analytical results are illustrated with the support of numerical examples. Finally, we close the paper with conclusion and future directions.

2. Existence and Uniqueness of the Nonnegative Solution

As we are dealing with the population model, the positive solution of the model is of our interest. The coefficients of (3) are locally Lipschitz continuous and do not satisfy the linear growth condition, so the solution of (3) may explode at a finite time. The following theorem shows that the solution is positive and will not explode at a finite time.

We define a subset Δ of IR^3 as follows:

$$\Delta = \left\{ (x, y, z) \in IR_+^3 : x + y + z = 1 \right\}.$$
(7)

Theorem 1. For any given initial value $X_0 = (S(0), I(0), R(0)) \in \Delta$, there is a unique positive solution X(t) = (S(t), I(t), R(t)) of (3) on $t \ge 0$, and the solution will remain in Δ with probability 1, namely, $(S(t), I(t), R(t)) \in \Delta$ for all $t \ge 0$ almost surely (briefly a.s.).

Proof 1. Since the coefficients of system (3) are locally Lipschitz continuous, for any initial value $(S(0), I(0), R(0)) \in \Delta$, there is a unique local solution on $[0, \tau_e)$, where τ_e is the explosion time. To show this solution is global, we need to show that $\tau_e = \infty$ a.s. For this, we define the stopping time τ by

$$\tau = \inf \{ t \in [0, \tau_e) : S(t) \le 0, \text{ or } I(t) \le 0, \text{ or } R(t) \le 0 \},$$
(8)

with the traditional setting $\inf \emptyset = \infty$ (as usual \emptyset denotes the empty set). We have $\tau \leq \tau_e$. If we can show that $\tau = \infty$ a.s., then $\tau_e = \infty$ a.s. and $(S(t), I(t), R(t)) \in \Delta$ for all $t \geq 0$. In other words, to complete the proof, all we only need to show is that $\tau = \infty$. Assume that this statement is false, then there exists a T > 0 such that $P(\tau < T) > 0$. Define a \mathscr{C}^2 function *U*, by the expression

$$U(S, I, R) = -\ln(SIR).$$
(9)

Using Itô's formula, we get

$$dU(X(t)) = \frac{-1}{S} \left[-\frac{\beta SI}{f(S,I)} - bS + (1-m)pdI + b(1-m)(S+R) \right] dt$$

$$-\frac{1}{I} \left[\frac{\beta SI}{f(S,I)} - (pd+r)I \right] dt - \frac{1}{R} \left[rI - bR + dmpI + mb(S+R) \right] dt$$

$$+ \left[\frac{1}{2} \left(\frac{\sigma I}{f(S,I)} \right)^2 + \frac{1}{2} \left(\frac{\sigma S}{f(S,I)} \right)^2 \right] dt + \left[\frac{\sigma I}{f(S,I)} - \frac{\sigma S}{f(S,I)} \right] dB(t).$$
 (10)

Since $f(S, I) \ge 1$, and $S \le 1$, $I \le 1$, we get,

$$dU(X(t)) \le H dt + \frac{\sigma(I-S)}{f(S,I)} dB(t), \tag{11}$$

where $H = \beta + 2b + pd + r + \sigma^2$. Then, we have

$$U(X(t)) \le U(X_0) + \int_0^t H \, ds + \int_0^t \frac{\sigma(I(s) - S(s))}{f(S(s), I(s))} \, dB(s).$$
(12)

Note that some components of $X(\tau)$ equal 0; thus, $\lim_{t \to \tau} U(X(t)) = +\infty$.

Letting $t \longrightarrow \tau$ in (12), we obtain

$$+\infty \le U(X_0) + \int_0^\tau H\,ds + \int_0^\tau \frac{\sigma(I(s) - S(s))}{f(S(s), I(s))} dB(s) < +\infty,$$
(13)

which contradicts our assumption. Then, $\tau = \infty$ a.s. This completes the proof of theorem.

3. Extinction

In the following, we give a condition for the extinction of the disease. Let

$$R_{s} = \frac{R_{0}}{(1-m)} - \frac{\sigma^{2}}{2(1+\alpha_{1}(1-m))^{2}(pd+r)}.$$
 (14)

Theorem 2. Let (S(t), I(t), R(t)) be the solution of system (3) with initial value $(S(0), I(0), R(0)) \in \Delta$. Assume that

(i)
$$\sigma^2 > \beta^2 / (2(pd + r))$$
 or
(ii) $R < 1$ and $\sigma^2 < \beta$

(ii)
$$R_s < 1$$
 and $\sigma^2 < \beta$

Then,

 $\limsup_{t \longrightarrow +\infty} \ln \frac{I(t)}{t} \le \frac{\beta^2}{2\sigma^2} - (pd + r) < 0 \quad \text{a.s. if } (i) \text{ holds,}$

$$\limsup_{t \to +\infty} \ln \frac{I(t)}{t} \le \left(\tilde{R}_s - 1\right) (pd + r) < 0 \text{ a.s. if } (ii) \text{ holds.}$$
(15)

Namely, I(t) tends to zero exponentially a.s., i.e., the disease dies out with probability 1.

Proof 2. Applying Itô's formula to system (3) leads to

$$d\ln I(t) = \left[\frac{\beta S}{f(S,I)} - (pd+r) - \frac{\sigma^2 S^2}{2f^2(S,I)}\right] dt$$

$$+ \frac{\sigma S}{f(S,I)} dB(t).$$
(16)

Integrating both sides of (16) from 0 to t, we get

$$\ln I(t) = -\frac{\sigma^2}{2} \int_0^t \left(\frac{S}{f(S,I)} - \frac{\beta}{\sigma^2}\right)^2 du + \frac{\beta^2}{2\sigma^2}t - (pd+r)t + M(t) + \ln I(0) \leq \left(\frac{\beta^2}{2\sigma^2} - (pd+r)\right)t + M(t) + \ln I(0),$$
(17)

where $M(t) = \int_0^t (\sigma S(u)/f(S(u), I(u))) dB(u)$, which is a local continuous martingale, and M(0) = 0. Moreover, its quadratic variation is

$$\langle M, M \rangle_t = \int_0^t \left(\frac{\sigma S(u)}{f(S(u), I(u))} \right)^2 du \le \sigma^2 t \quad \text{a.s.}$$
(18)

By the large number theorem for martingales [21], we obtain

$$\lim_{t \to +\infty} \frac{M(t)}{t} = 0 \quad \text{a.s.}$$
(19)

If condition (i) is satisfied, dividing by t and taking the limit superior of both sides of (17), we get

$$\limsup_{t \longrightarrow +\infty} \frac{\ln I(t)}{t} \le \frac{\beta^2}{2\sigma^2} - (pd + r) < 0 \quad \text{a.s.}$$
(20)

If the condition (ii) is satisfied, note that

$$\frac{\beta S}{1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI} = \frac{\beta}{1 + \alpha_1} - \frac{\beta(1 - S)}{(1 + \alpha_1)(1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI)} - \frac{\beta \alpha_2 I}{(1 + \alpha_1)(1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI)} - \frac{\beta \alpha_3 SI}{(1 + \alpha_1)(1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI)} \le \frac{\beta}{1 + \alpha_1} \le \frac{\beta}{1 + \alpha_1(1 - m)}.$$
(21)

It follows that

$$\frac{\ln I(t)}{t} = \int_{0}^{t} \left(\frac{\beta S}{f(S,I)} - (pd+r) - \frac{\sigma^{2}S^{2}}{2f^{2}(S,I)}\right) du + \frac{M(t)}{t} + \frac{\ln I(0)}{t}$$

$$\leq \frac{\beta}{1 + \alpha_{1}(1-m)} - (pd+r) - \frac{\sigma^{2}}{2(1 + \alpha_{1}(1-m))^{2}} + \frac{M(t)}{t} + \frac{\ln I(0)}{t}$$

$$= (pd+r) \left[\frac{\beta}{(1 + \alpha_{1}(1-m))(pd+r)} - \frac{\sigma^{2}}{2(1 + \alpha_{1}(1-m))^{2}(pd+r)} - 1\right]$$

$$+ \frac{M(t)}{t} + \frac{\ln I(0)}{t}$$

$$= (pd+r)[R_{s} - 1] + \frac{M(t)}{t} + \frac{\ln I(0)}{t}.$$
(22)

Taking the superior limit of both sides of (22), we obtain

$$\limsup_{t \longrightarrow +\infty} \frac{\ln I(t)}{t} \le (pd+r) [R_s - 1] < 0 \quad \text{a.s.},$$
(23)

which implies that $\lim_{t \to +\infty} I(t) = 0$.

Remark 1. From Theorem 1, we can get that the disease will die out if $R_s < 1$, and the white noise is not large such that $\sigma^2 < \beta$, while if the white noise is large enough such that condition (i) is satisfied, then the infectious disease of system (3) goes to extinction almost surely.

4. Persistence

Here, we investigate the condition for the persistence of the disease. The basic reproduction number R_0 (see [20]), is the threshold between disease extinction and persistence, with extinction for $R_0 \le 1$ and persistence for $R_0 > 1$ in the deterministic model. In the stochastic model, we define the threshold of persistence for disease as

$$R_{s}^{*} = (1-m)R_{0} - \frac{\sigma^{2}}{2(1+\alpha_{1}(1-m))^{2}(1-m)(pd+r)}.$$
(24)

Definition 1. System (3) is said to be persistent in the mean if

$$\liminf_{t \to \infty} \frac{1}{t} \int_0^t I(u) du > 0 \quad \text{a.s.}$$
(25)

We define

$$\langle x(t) \rangle = \frac{1}{t} \int_0^t x(u) du.$$
 (26)

Theorem 3. If $R_s^* > 1$, then the solution (S(t), I(t), R(t)) of system (3) with initial initial value $(S(0), I(0), R(0)) \in \Delta$ is persistent in mean. Moreover, we have

$$\liminf_{t \to \infty} \langle I(t) \rangle \ge \frac{(1 + \alpha_1 (1 - m))(pd + r)}{\chi} \left[R_s^* - 1 \right] \quad \text{a.s.,}$$
(27)

where $\chi = \beta (1 - m) (((r + dmp)/b) (1 - m) + ((\alpha_2 + \alpha_3) (pd + r)/\beta (1 - m))).$

International Journal of Differential Equations

Proof 3. We have

$$\langle S(t) \rangle + \langle I(t) \rangle + \langle R(t) \rangle = 1.$$
 (28)

Integrating from 0 to t and dividing by t > 0 the third equation of system (3), we get

$$\frac{R(t) - R(0)}{t} = mb\langle S(t) \rangle + (r + dmp)\langle I(t) \rangle$$

$$-b(1 - m)\langle R(t) \rangle.$$
(29)

From (28), one can get

$$\langle S(t) \rangle = -\left(\frac{r+dmp}{b} + 1 - m\right) \langle I(t) \rangle + (1-m) + \frac{\varphi(t)}{b},$$
(30)

where $\varphi(t) = (R(t) - R(0))/t$. From Itô's formula and (3), we obtain

$$\begin{aligned} d((1 + \alpha_{1}(1 - m))\ln I(t) + (\alpha_{2} + \alpha_{3})I) \\ &= \left[\frac{(1 + \alpha_{1}(1 - m))\beta S}{(1 + \alpha_{1}S + \alpha_{2}I + \alpha_{3}SI} - (1 + \alpha_{1}(1 - m))(pd + r) - \frac{\sigma^{2}(1 + \alpha_{1}(1 - m))S^{2}}{2(1 + \alpha_{1}S + \alpha_{2}I + \alpha_{3}SI)^{2}} \right] dt \\ &+ (\alpha_{2} + \alpha_{3}) \left[\frac{\beta SI}{1 + \alpha_{1}S + \alpha_{2}I + \alpha_{3}SI} - (pd + r)I \right] \\ &+ \frac{(1 + \alpha_{1}(1 - m))\sigma S}{1 + \alpha_{1}S + \alpha_{2}I + \alpha_{3}SI} dB + \frac{(\alpha_{2} + \alpha_{3})\sigma SI}{1 + \alpha_{1}S + \alpha_{2}I + \alpha_{3}SI} dB \\ &\geq \left[\frac{(1 + \alpha_{1}(1 - m))\beta S}{1 + \alpha_{1} + \alpha_{2}I + \alpha_{3}I} - (1 + \alpha_{1}(1 - m))(pd + r) - \frac{\sigma^{2}(1 + \alpha_{1}(1 - m))S^{2}}{2(1 + \alpha_{1}S + \alpha_{2}I + \alpha_{3}SI)^{2}} \right] dt, \\ (\alpha_{2} + \alpha_{3}) \left[\frac{\beta SI}{1 + \alpha_{1} + \alpha_{2}I + \alpha_{3}I} - (pd + r)I \right] + \frac{(1 + \alpha_{1}(1 - m))\sigma S}{1 + \alpha_{1} + \alpha_{2}I + \alpha_{3}I} dB + \frac{(\alpha_{2} + \alpha_{3})\sigma SI}{1 + \alpha_{1} + \alpha_{2}I + \alpha_{3}I} dB \\ &\geq \left[\beta(1 - m)S - (1 + \alpha_{1}(1 - m))(pd + r) - (\alpha_{2} + \alpha_{3})(pd + r)I - \frac{\sigma^{2}}{2(1 + \alpha_{1}(1 - m))} \right] dt + \sigma(1 - m)SdB. \end{aligned}$$

Integrating from 0 to *t* and dividing by t > 0 on both sides of yields

$$\frac{(1+\alpha_{1}(1-m))\ln I(t) - (1+\alpha_{1}(1-m))\ln I(0)}{t} + \frac{(\alpha_{2}+\alpha_{3})I - (\alpha_{2}+\alpha_{3})I(0)}{t}$$

$$\geq \left[\beta(1-m)\langle S\rangle - (1+\alpha_{1}(1-m))(pd+r) - (\alpha_{2}+\alpha_{3})(pd+r)\langle I\rangle - \frac{\sigma^{2}}{2(1+\alpha_{1}(1-m))}\right]dt + \frac{M(t)}{t}$$

$$\geq \beta(1-m)\left[-\left(\frac{r+dmp}{b}+1-m\right)\langle I(t)\rangle + (1-m) + \frac{\varphi(t)}{b}\right] - (\alpha_{2}+\alpha_{3})(pd+r)\langle I\rangle$$

$$-\frac{\sigma^{2}}{2(1+\alpha_{1}(1-m))} + \frac{M(t)}{t} - (1+\alpha_{1}(1-m))(pd+r) - (\alpha_{2}+\alpha_{3})(pd+r)$$

$$= -\beta(1-m)\left(\frac{r+dmp}{b}+1-m + \frac{(\alpha_{2}+\alpha_{3})(pd+r)}{\beta(1-m)}\right)\langle I(t)\rangle + \beta(1-m)^{2}$$

$$+\frac{\beta(1-m)\varphi(t)}{b} - (1+\alpha_{1}(1-m))(pd+r) - \frac{\sigma^{2}}{2(1+\alpha_{1}(1-m))} + \frac{M(t)}{t}.$$
(32)

We can rewrite the inequality (32) as

$$\langle I(t) \rangle \geq \frac{1}{\chi} \left[\beta (1-m)^2 - \left(1 + (1-m)\alpha_1\right)(pd+r) - \frac{\sigma^2}{2\left(1+\alpha_1(1-m)\right)} \right] + \frac{1}{\chi} \left[\frac{\beta (1-m)\varphi(t)}{b} + \frac{M(t)}{t} - \frac{(1+\alpha_1(1-m))\ln I(t) - (1+\alpha_1(1-m))\ln I(0)}{t} - \frac{(\alpha_2 + \alpha_3)I(t) - (\alpha_2 + \alpha_3)I(0)}{t} \right],$$

$$(33)$$

where $\chi = \beta (1 - m)((r + dmp/b) - 1 + m + ((\alpha_2 + \alpha_3)(pd + r)/\beta(1 - m)))$. We can see that $R(t) \le 1$ and $I(t) \le 1$. Thus, one has $\lim_{t \longrightarrow +\infty} R(t)/t = 0$, $\lim_{t \longrightarrow +\infty} I(t)/t = 0$, and

 $\lim_{t \to +\infty} \varphi(t) = 0$. Taking the inferior limit of both sides of (33) yields

$$\liminf_{t \to +\infty} \langle I(t) \rangle \geq \frac{1}{\chi} \left[\beta (1-m)^2 - (1+\alpha_1(1-m))(pd+r) - \frac{\sigma^2}{(2(1+\alpha_1(1-m))))} \right]$$

$$\geq \frac{(1+\alpha_1(1-m))(pd+r)}{\chi} \left[\frac{\beta (1-m)^2}{(1+\alpha_1(1-m))(pd+r)} - \frac{\sigma^2}{2(1+\alpha_1(1-m))^2(pd+r)} - 1 \right]$$
(34)
$$= \frac{(1+\alpha_1(1-m))(pd+r)}{\chi} [R_s^* - 1].$$

 \Box

This completes the proof of theorem.

5. Numerical Simulations

In order to illustrate our theoretical results, we give some numerical simulations. The values of m, σ , and β will be varied over the different examples.

Example 1. We choose the parameters in system (3) as follows:

m = 0.9, $\beta = 0.6,$ p = 0.1, b = 0.2, d = 0.4, r = 0.2, $\sigma = 0,$ $\alpha_1 = 0.6,$ $\alpha_2 = 0.1,$ $\alpha_3 = 0.1.$ (35)

By calculation, we have $R_0 = 0.4902$; in this case, the disease dies out as shown in Figure 1(a). By choosing m = 0.3, we obtain $R_0 = 1.635$, and we deduce that the disease persists in the population. Figure 1(b) illustrates this result.

Example 2. We choose the parameters in system (3) as follows:

$$m = 0.2,$$

 $\beta = 0.8,$
 $p = 0.5,$
 $b = 0.2,$
 $d = 0.4,$
 $r = 0.2,$
 $\sigma = 0.9,$
 $\alpha_1 = 0.6,$
 $\alpha_2 = 0.1,$
 $\alpha_3 = 0.1.$
(36)

A simple computation shows that

$$R_{s} = 0.9965 < 1,$$

$$R_{0} = 1.0811,$$

$$\sigma^{2} = 0.6400 > 0.1280 = \frac{\beta^{2}}{2(pd + r)}.$$
(37)

It follows that the condition of Theorem 2 is satisfied. We conclude that the disease dies out; Figure 2 illustrates this result.



FIGURE 1: Simulations of the path S(t), I(t), and R(t) for the corresponding deterministic system (1).



FIGURE 2: Simulations of the path (a) *S*(*t*), (b) *I*(*t*), and (c) *R*(*t*) for the deterministic system (1) and the corresponding stochastic system (3).



FIGURE 3: Simulations of the path (a) *S*(*t*), (b) *I*(*t*), and (c) *R*(*t*) for the deterministic system (1) and the corresponding stochastic system (3).

Example 3. On the contrary, we choose m = 0.05 and $\sigma = 0.1$ by simple calculation, and it can be found that $R_s^* = 1.2102 > 1$ which implies that the disease persists (Figure 3).

6. Conclusion

This article discusses a stochastic SIR epidemic model with vertical transmission and vaccination and nonlinear incidence rate. We have shown that when the noise is so small such that $\sigma^2 < \beta$, the extinction of the disease can be determined by the value of R_s , i.e., if $R_s < 1$, the disease dies out. Moreover, the disease dies out when the white noise is large enough such that $\sigma^2 > (\beta^2/2 (pd + r))$.

The persistence of the disease is determined by R_s^* , i.e., if $R_s^* > 1$, the disease persists. We presented some numerical simulations to illustrate the obtained analytical results. To go further in this study, we can give a new dimension to the stochastic SIR epidemic model (3) by introducing a different type of noise which is the random telegraph noise (RTN), modeled by the Markov chain. We will investigate this case in our future works.

Data Availability

The data used to support the findings of this study are available from the corresponding author upon request.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this paper.

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