Research Article

Stability and Threshold of a Stochastic SIRS Epidemic Model with Vertical Transmission and Transfer from Infectious to Susceptible Individuals

Driss Kiouach and Yassine Sabbar

MSTI Team, High School of Technology, Ibn Zohr University, Agadir, Morocco

Correspondence should be addressed to Driss Kiouach; d.kiouach@uiz.ac.ma

Received 22 December 2017; Revised 14 March 2018; Accepted 25 March 2018; Published 6 May 2018

Academic Editor: Zhengqiu Zhang

Copyright © 2018 Driss Kiouach and Yassine Sabbar. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

The aim of this paper is to generalize the nonlinear incidence rate of a stochastic SIRS (susceptible-infected-recovered-susceptible) epidemic model. Our basic model was enriched with the hypotheses of vertical transmission and transfer from infected to susceptible individuals, to approach the reality. Our analysis showed that the model is well-posed. Under some conditions imposed on the intensity of the white noise perturbation, the global stability of the system is proven. Furthermore, the threshold of our model which determines the extinction and persistence of the disease is established. Numerical examples are realized to prove the rigor of our theoretical results.

1. Introduction

Since the earlier works of Kermack and McKendrick (see, e.g., [1, 2]), mathematical models have proven their adequacy to describe and analyze the propagation and control of infectious diseases such as Diphtheria, Polio, Tuberculosis, Cholera, and Toxoplasmosis. Various epidemic models of population dynamics have been proposed (see, e.g., [3–6]). Generally, the contact between individuals leads to the dissemination of a disease. The vertical transmission is also possible, which means the direct transmission of a disease from the mother to a fetus during pregnancy. This hypothesis was not taken into consideration in the basic SIRS epidemic model. To be more realistic, our model considers this new hypothesis. In addition, for some bacterial agent infections, recovery cannot produce immunity for a long time. Infected individuals may recover after some treatments and therapies and then go back directly to the susceptible compartment [7]. Considering those two new hypotheses, we obtain the following SIRS epidemic model:

\[
\begin{align*}
\dot{S}(t) &= A + b (S(t) + R(t)) - \beta SI - \mu S(t) + pbI(t) + \gamma_1 I(t) + kR(t), \\
\dot{I}(t) &= \beta SI + qbI(t) - (\mu + \gamma_1 + \gamma_2 + a) I(t), \\
\dot{R}(t) &= \gamma_2 I(t) - (\mu + k) R(t),
\end{align*}
\]

(1)

where \( S(t), I(t), \) and \( R(t) \) denote the numbers of susceptible, infected, and recovered individuals at time \( t \), respectively. \( A \) is the recruitment rate of susceptible corresponding to immigration. \( b \) and \( \mu \) are the birth rate and natural death rate, respectively. It is assumed that \( \mu > b \) [8]. \( \gamma_1 \) is the transfer rate from the infected class to the susceptible class and \( \gamma_2 \) is the transfer rate from the infected class to the recovered class. \( k \) is the rate of individuals recovering and returning to \( S \) from \( R \). \( a \) is the disease-related death rate. \( q \) is the vertical transmission coefficient, with \( 0 < q < 1 \) and \( p = 1 - q \). \( \beta = \bar{p}c \) represents the transmission rate, which is the product of the contact rate \( c \) and the probability of transmission per contact \( \bar{p} \). The parameters involved in system (1) are all positive constants. According to the theory in [7, 8], the basic reproduction number of system (1) is \( R_0 = \frac{\beta A}{(\mu - b)(\mu + \gamma_1 + \gamma_2 + a - qb)} \). If \( R_0 \leq 1 \), the deterministic model (1) has only the disease-free equilibrium \( P^0(A/(\mu - b), 0, 0) \), which is globally asymptotically stable. If \( R_0 > 1 \), \( P^0 \) becomes unstable and there exists an endemic equilibrium \( P^*(S^*, I^*, R^*) \) such that
\[ I^* = \frac{(R_0 - 1) (\mu + k) (\mu - b) (\mu + \gamma_1 + \gamma_2 + a - qb)}{\beta \gamma_2 (\mu - b) + [\beta (\mu - b) + \beta + a (\mu - b) (\mu + \gamma_1 + \gamma_2 + a - qb)] (\mu + k)}, \]

\[ S^* = \frac{(\mu + \gamma_1 + \gamma_2 + a - qb) I^*}{\beta}, \]

\[ R^* = \frac{\gamma_2 I^*}{\mu + k}. \]

The incidence rate is the number of new infected situations per population in a given time phase. In many previous epidemic models, the bilinear incidence rate is frequently used (see, e.g., [9–11]). However, there exist many forms of nonlinear incidence rate and each form presents some advantages as the following examples:

1. \( f(S, I) = (\beta - (\beta_1 I / (V + I)) S I, (\beta > \beta_1, V > 0)) [12]. \)
2. \( f(S, I) = \beta S I / (1 + k I^p), (k > 0, 0 \leq p \leq 1) [13]. \)
3. \( f(S, I) = \beta S I / (1 + k_1 I + k_2 S), (k_1 > 0, k_2 \geq 0) [14]. \)
4. \( f(S, I) = \beta S^u I / (1 + S^v), (u \geq v) [15]. \)

There have been many mathematical models [16–21] committed to studying the impacts of nonlinear transmission on the propagation of a disease. The aim of our work is to generalize the results found by those authors. According to the reality, we shall consider our model with the following general functional response:

\[ f(S, I) = \frac{\beta S I}{1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI}, \]

where \( \alpha_1, \alpha_2, \alpha_3 \geq 0. \) It is necessary to mention that \( 1 + \alpha_3 S + \alpha_2 I + \alpha_3 SI \) is a general form which represents mutual interference between \( S \) and \( I. \) In particular cases,

1. when \( \alpha_1 = \alpha_2 = \alpha_3 = 0, \) \( f(S, I) \) becomes a bilinear mass-action function response (namely, type I Holling functional response) [22];
2. when \( \alpha_2 = \alpha_3 = 0, \) \( f(S, I) \) becomes a saturated incidence rate (namely, Holling type II functional response) [20];
3. when \( \alpha_3 = 0, \) \( f(S, I) \) becomes a Beddington-DeAngelis functional response (namely, modified type II Holling functional response) [23];
4. when \( \alpha_3 = \alpha_1 \alpha_2, \) \( f(S, I) \) becomes a Crowley-Martín functional response introduced in [24].

By all what we have introduced, we consider the following system:

\[ \dot{S}(t) = A + b (S(t) + R(t)) - \frac{\beta S(t) I(t)}{\psi(S, I)} - \mu S(t) + pb I(t) + \gamma_1 I(t) + k R(t), \]

\[ \dot{I}(t) = \frac{\beta S(t) I(t)}{\psi(S, I)} + q b I(t) - (\mu + \gamma_1 + \gamma_2 + a) I(t), \]

\[ \dot{R}(t) = \gamma_2 I(t) - (\mu + k) R(t), \]

where \( \psi(S, I) = 1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI. \) The basic reproduction number of system (4) can be presented as follows:

\[ R_0 = \frac{\beta A}{[(\mu - b) + \alpha_1 A] (\mu + \gamma_1 + \gamma_2 + a - qb)}. \]

The deterministic model constructed above can be improved by taking into account the unpredictable biological condition. In the real world, biological phenomena are often affected by the environmental noise, and the nature of epidemic growth is inherently random due to the unpredictability of person-person contacts in the case of horizontal transmission, or in the other case of vertical transmission (mother-fetus). That is to say, the parameter \( \beta \) involved in system (4) is not absolute constant and may fluctuate around some average values. Both from a biological and mathematical perspectives, the main of our study is to investigate how the stochastic character of human transmission affects the spread of a disease through studying the dynamical behavior of our stochastic model. We assume that the contact rate \( \beta \) is perturbed by Gaussian white noise, which is presented by \( \beta + \sigma B(t), \) where \( B(t) \) is a standard Brownian motion with intensity \( \sigma > 0. \) Then we obtain the following SIRS epidemic model with perturbation stochastic and general functional response:

\[ dS(t) = \left[ A + b (S(t) + R(t)) - \frac{\beta S(t) I(t)}{\psi(S, I)} - \mu S(t) + pb I(t) + \gamma_1 I(t) + k R(t) \right] dt \]

\[ + \sigma S(t) I(t) \psi(S, I) dB(t), \]

\[ dI(t) = \left[ \frac{\beta S(t) I(t)}{\psi(S, I)} + q b I(t) - (\mu + \gamma_1 + \gamma_2 + a) I(t) \right] dt + \sigma S(t) I(t) \psi(S, I) dB(t), \]

\[ dR(t) = \left[ \gamma_2 I(t) - (\mu + k) R(t) \right] dt. \]
Many authors have been introduced random effects into population systems by different techniques (see, e.g., [25–34]). Furthermore, in the study of the dynamical behavior of the epidemic models, we are interested in two situations. One is when the disease goes to extinction, the other is when the disease prevails. Thus many authors have studied this interesting topic. For example, Li and Jiang [35] investigated the threshold of SIR epidemic model with stochastic perturbation. Zhao and Jiang [36] investigated the threshold of a stochastic SIRS epidemic model with saturated incidence [37]. Then, they considered the threshold of a stochastic SIS epidemic model with standard incidence and imperfect vaccine. In case that the disease goes to extinction, they showed that the disease-free equilibrium is almost surely stable by using the nonnegative semimartingale convergence theorem. In this work, we consider a stochastic SIRS epidemic model with general incidence rate (3). This generalization is the main difficulty to be overcome in establishing the threshold of SIRS epidemic model (6), which has never been examined in the previously studies. In addition, we prove the global stability of SIRS epidemic model (4) with stochastic perturbations, which has not been proven in the previous papers.

This paper is organized as follows. In Section 2, we present some preliminaries which will be used in our following analysis. In Section 3, we show that there is a unique global positive solution of system (6). In Section 4, we give the conditions for the moment exponential stability of the equilibrium $P^0$ of stochastic system (6). In Sections 5 and 6, we establish sufficient conditions for persistence and extinction of disease, respectively. In Section 7, we present some numerical simulations to illustrate our main results. The paper ends with a brief discussion.

2. Preliminaries

Throughout this paper, we let $(\Omega, \mathcal{F}, \mathbb{P})$ be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t \geq 0}$ satisfying the usual conditions (i.e., $\{\mathcal{F}_t\}_{t \geq 0}$ is increasing and right continuous while $\mathcal{F}_0$ contains all $\mathbb{P}$-null sets). $\mathbb{B}(t)$ is defined on this complete probability space. We also let $\mathbb{R}_+^d = \{x \in \mathbb{R}^d : x_i > 0, \ 1 \leq i \leq d\}$.

In general, we consider the $d$-dimensional stochastic differential equation:

$$dx(t) = f(x(t), t) \, dt + g(x(t), t) \, dB(t) \quad \forall t \geq t_0,$$  \hspace{1cm} (7)

with initial value $x(0) = x_0 \in \mathbb{R}_+^d$. $B(t)$ denotes $n$-dimensional standard Brownian motion defined on the complete probability space $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t \geq 0}, \mathbb{P})$. $C^{2,1}(\mathbb{R}^d \times [t_0, \infty); \mathbb{R}_+)$ is the family of all nonnegative functions $V(x, t)$ defined on $\mathbb{R}_+^d \times [t_0, \infty)$ such that they are continuously twice differentiable in $x$ and once in $t$. The differential operator $L$ of (7) is defined by the following [40]:

$$L = \frac{\partial}{\partial t} + \sum_{i=1}^{d} f_i(x(t)) \frac{\partial}{\partial x_i} + \frac{1}{2} \sum_{i,j=1}^{d} \left[ g^r(x(t)) g(x(t)) \right]_{ij} \frac{\partial^2}{\partial x_i \partial x_j}.$$  \hspace{1cm} (8)

If $L$ acts on a function $V \in C^{2,1}(\mathbb{R}^d \times [t_0, \infty); \mathbb{R}_+)$, then

$$LV(x(t), t) = V_t(x(t), t) + V_x(x(t), t) f(x(t), t)$$

$$+ \frac{1}{2} \text{trace} \left[ g^r(x(t)) V_{xx}(x(t), t) g(x(t), t) \right].$$  \hspace{1cm} (9)

By Itô’s formula, if $x(t) \in \mathbb{R}^d$, we have

$$dV(x(t), t) = LV(x(t), t) \, dt$$

$$+ V_x(x(t), t) g(x(t), t) \, dB(t).$$  \hspace{1cm} (10)

Next, we shall present the definition of $h$th moment exponential stability (see [41]).

Definition 1. The equilibrium $x = 0$ of the system (7) is said to be $h$th moment exponentially stable if there is a pair of positive constants $C_1$ and $C_2$ such that, for all $x_0 \in \mathbb{R}^d$,

$$E\left( |x(x_0, t)|^h \right) \leq C_1 |x_0|^h e^{-C_2 t} \quad \text{on } t \geq 0.$$  \hspace{1cm} (11)

3. Existence and Uniqueness of the Positive Solution

Since $S(t), I(t),$ and $R(t)$ represent the number of the susceptible, the infected, and the recovered individuals at time $t$, respectively, they should be nonnegative. So, the first step of our study is to prove that system (6) has a unique global positive solution. We define a bounded set $\Delta$ as follows:

$$\Delta := \left\{ x = (x_1, x_2, x_3) : x_1 > 0, \ x_2 > 0, \ x_3 > 0, \ x_4 \right\}$$

$$+ x_2 + x_3 < \frac{A}{\mu - b} \quad \text{a.s.}$$  \hspace{1cm} (12)

Theorem 2. For any initial value $(S(0), I(0), R(0)) \in \Delta$, there exists a unique positive solution $(S(t), I(t), R(t))$ of system (6) on $t \geq 0$, and the solution will remain in $\mathbb{R}_+^3$ with probability one. That is to say, the solution $(S(t), I(t), R(t)) \in \mathbb{R}_+^3$ for all $t \geq 0$ almost surely.

Proof. Let $(S(0), I(0), R(0)) \in \Delta$ and $N(t) = S(t) + I(t) + R(t)$. It is easy to check that

$$dN(t) = [A - (\mu - b) N(t) - al] \, dt.$$  \hspace{1cm} (13)

Then, if $(S(s), I(s), R(s)) \in \mathbb{R}_+^3$ for all $0 \leq s \leq t$ a.s. we get

$$dN(s) < [A - (\mu - b) N(s)] \, ds \quad \text{a.s.}$$  \hspace{1cm} (14)
Now, by integration we obtain
\[ N(s) = \frac{A}{\mu - b} \left( N(0) - \frac{A}{\mu - b} \right) e^{-\mu s} \quad \forall s \in [0, t] \text{ a.s.} \] (15)
Then \( N(s) < A/\mu - b \), and
\[ S(s), I(s), R(s) \in [0, \frac{A}{\mu - b}] \quad \forall s \in [0, t] \text{ a.s.} \] (16)
Since the coefficients of system (6) are locally Lipschitz continuous, then for any initial value \((S(0), I(0), R(0)) \in \mathbb{R}_+^3\), there is a unique local solution \((S(t), I(t), R(t))\) on \([0, \tau_e]\), where \(\tau_e\) is the explosion time. To show that the solution is global, we only need to prove that \(\tau_e = \infty \) a.s.

Let \(e_0 > 0\) such that \(S(0), I(0), R(0) > e_0\). For each integer \(\epsilon \leq e_0\), we define the following stopping times:
\[ \tau_e = \inf \{ t \in [0, \tau_e] : S(t) \leq \epsilon \text{ or } I(t) \leq \epsilon \text{ or } R(t) \leq \epsilon \}, \]
\[ \tau = \lim_{\epsilon \to 0} \tau_e \]
\[ = \inf \{ t \in [0, \tau_e] : S(t) \leq 0 \text{ or } I(t) \leq 0 \text{ or } R(t) \leq 0 \}. \] (17)
Consider the \(\mathbb{C}^2\)-function \(V_1\) defined for \(X = (S, I, R) \in \mathbb{R}_+^3\) by
\[ V_1(X) = \ln \left( \frac{(\mu - b)S}{A} \right) - \ln \left( \frac{(\mu - b)I}{A} \right) - \ln \left( \frac{(\mu - b)R}{A} \right). \] (18)
Making use Itô's formula to \(V_1\), we obtain for all \(t \geq 0\) and \(s \in [0, t \wedge \tau_e]\)
\[ dv_1(X(s)) = -\left( \frac{\beta I(s)}{\psi(S(s), I(s))} - \frac{(\beta b + \gamma_1)I(s)}{S(s)} + kR(s) \right) + \sigma^2 \frac{I^2(s)}{2\psi^2(S(s), I(s))} ds + \left( -\frac{\beta S(s)}{\psi(S(s), I(s))} + (\mu + \gamma_1 + \gamma_2 + a + qb^2) + \frac{\sigma^2 S^2(s)}{2\psi^2(S(s), I(s))} \right) ds \]
\[ + \left( \mu + k - \frac{\gamma_2 I(s)}{R(s)} \right) d\]s
\[ + \left( \sigma (I(s) - S(s)) / \psi(S(s), I(s)) \right) dB(s) \leq \frac{3\mu + \gamma_1 + \gamma_2 + a + k}{\psi(S(s), I(s))} \]
\[ + \frac{\beta I(s)}{\psi(S(s), I(s))} + \frac{\sigma^2 I^2(s)}{2\psi^2(S(s), I(s))} \]
\[ + \frac{\sigma^2 S^2(s)}{2\psi^2(S(s), I(s))} ds + \sigma (I(s) - S(s)) / \psi(S(s), I(s)) dB(s). \] (19)
For all \(s \in [0, t \wedge \tau_e]\), we have
\[ \frac{I(s)}{\psi(S(s), I(s))} \leq \frac{A}{(\mu - b) + \alpha_1 A}, \]
\[ \frac{S(s)}{\psi(S(s), I(s))} \leq \frac{A}{(\mu - b) + \alpha_1 A}. \] (20)
Therefore
\[ dV_1(X(s)) \leq M + \sigma (I(s) - S(s)) / \psi(S(s), I(s)) dB(s) \text{ a.s.,} \] (21)
where \(M = 3\mu + \gamma_1 + \gamma_2 + a + k + \beta A((\mu - b) + \alpha_1 A) + \sigma^2 A^2/((\mu - b) + \alpha_1 A)^2\). Integrating both sides of (21) from 0 to \(t \wedge \tau_e\), and after taking the expectation on both sides, we obtain that
\[ EV_1(X(t \wedge \tau_e)) \leq V_1(X(0)) + ME(t \wedge \tau_e) \]
\[ \leq V_1(X(0)) + Mt. \]
Since \(V_1(X(t \wedge \tau_e)) > 0\), then
\[ \mathbb{E} [V_1(X(t \wedge \tau_e))] \geq \mathbb{E} [V_1(X(t \wedge \tau_e))] \mathbb{1}_{[t \leq \tau_e]} \]
\[ \geq \mathbb{E} [V_1(X(t \wedge \tau_e))] \mathbb{1}_{[t \leq \tau_e]} \]
\[ \geq -\left( \frac{\mu - b}{A} \right) \mathbb{P} (\tau_e \leq t). \] (23)
For \(\tau_e\), there is some component of \(X(\tau_e)\) equal to \(\epsilon\). Therefore \(V_1(X(\tau_e)) \geq -\ln ((\mu - b)e/A)\).

Thus
\[ \mathbb{E} [V_1(X(t \wedge \tau_e))] \geq \mathbb{E} [V_1(X(t \wedge \tau_e))] \mathbb{1}_{[t \leq \tau_e]} \]
\[ \geq -\ln \left( \frac{\mu - b}{A} \right) \mathbb{P} (\tau_e \leq t). \] (24)
Hence, from (24) we conclude
\[ \mathbb{P} (\tau_e \leq t) \leq -\frac{V_1(X(0)) + Mt}{\ln ((\mu - b)e/A)}. \] (25)
Extending \(\epsilon\) to 0, we obtain for all \(t > 0\), \(\mathbb{P}(\tau \leq t) = 0\). Hence \(\mathbb{P}(\tau = \infty) = 1\). Thus, \(\tau = \tau_e = \infty\) a.s. which completes the proof of the theorem.

From Theorem 2 and (16) we can conclude the following corollary.

**Corollary 3.** The set \(\Delta\) is almost surely positively invariant; that is, if \((S(0), I(0), R(0)) \in \Delta\), then \(\mathbb{P}((S(t), I(t), R(t)) \in \Delta) = 1\) for all \(t \geq 0\).

**4. Moment Exponential Stability**

In order to obtain the conditions of moment exponential stability, we will use the following theorem (for the proof of this theorem we refer the reader to [41]).
Theorem 4. Suppose there exists a function $V(t, x) \in C^{1,2}(\mathbb{R} \times \mathbb{R}^n)$ satisfying the following inequalities:

$$K_1 |x|^h \leq V(t, x) \leq K_2 |x|^h,$$

$$LV(t, x) \leq -K_3 |x|^h, K_i > 0, h > 0.$$  \hspace{1cm} (26)

Then the equilibrium of the system (7) is $h$th moment exponentially stable. When $h = 2$, it is usually said to be exponentially stable in mean square and the equilibrium $x = 0$ is globally asymptotically stable.

From Young's inequality, we have the following inequalities.

Lemma 5. Let $h \geq 2$ and $e, x, y > 0$. Then

$$x^{h-1} y \leq \frac{(h-1)e}{h} x^h + \frac{1}{he^{h-1}} y^h,$$

$$x^{h-2} y^2 \leq \frac{(h-2)e}{h} x^h + \frac{1}{he^{(h-2)/2}} y^h.$$ \hspace{1cm} (28)

From Theorem 4, we get the sufficient conditions of the moment exponential stability, which are given by the following theorem.

Theorem 6. Let $h \geq 2$. If the conditions $R_0 < 1$ and

$$\sigma^2 < \frac{2}{h-1} \frac{A}{(\mu - b) + \alpha_i A} \left[ (\mu + \gamma_1 + \gamma_2 + a - qb) - \frac{\beta A}{(\mu - b) + \alpha_i A} \right]$$  \hspace{1cm} (29)

hold, the disease-free equilibrium $P^0$ of system (6) is $h$th moment exponentially stable in $\Delta$.

Proof. Let $h \geq 2$. Considering the following Lyapunov function,

$$V_2 = \lambda_1 \left( \frac{A}{\mu - b} - S \right)^h + \frac{1}{h} I^h + \lambda_2 R^h,$$ \hspace{1cm} (30)

where $\lambda_i, i = 1, 2$ are real positive constants to be determined later. It is easy to check that inequalities (26) are true. Then we compute

$$LV_2 = -h\lambda_1 \left( \frac{A}{\mu - b} - S \right)^h \left( \mu - b \right) + h\lambda_1 \left( \frac{A}{\mu - b} - S \right)^{h-1} \frac{\beta IS}{\psi(S, I)} - \frac{\lambda_1 (p\theta + \gamma_1) \left( \frac{A}{\mu - b} - S \right)^{h-1} I}{(h-2) + \alpha_i A}$$ \hspace{1cm} (31)

In $\Delta$, we have

$$LV_2 \leq -h\lambda_1 \left( \frac{A}{\mu - b} - S \right)^h \left( \mu - b \right) + \left[ \frac{\beta S}{\psi(S, I)} - (\mu + \gamma_1 + \gamma_2 + a - qb) \right] I^h$$

$$+ h\lambda_2 \frac{\beta A}{(\mu - b) + \alpha_i A} \frac{\left( \frac{A}{\mu - b} - S \right)^{h-1} \sigma^2 S^2 I^2}{\psi^2(S, I)} + \frac{(h-1) \sigma^2 IS^2}{2 \psi^2(S, I)}.$$ \hspace{1cm} (32)

By using Lemma 5, we get

$$I \left( \frac{A}{\mu - b} - S \right)^{h-1} \leq \frac{(h-1)e}{h} \left( \frac{A}{\mu - b} - S \right)^h + \frac{1}{he^{h-1}} I^h,$$ \hspace{1cm} (33)

$$I^2 \left( \frac{A}{\mu - b} - S \right)^{h-2} \leq \frac{(h-2)e}{h} \left( \frac{A}{\mu - b} - S \right)^h + \frac{1}{he^{(h-2)/2}} I^h,$$ \hspace{1cm} (34)

$$IR^{h-1} \leq \frac{(h-1)e}{h} R^h + \frac{1}{he^{h-1}} I^h.$$ \hspace{1cm} (35)

Then

$$LV_2 \leq -h\lambda_1 \left( \frac{A}{\mu - b} - S \right)^h \left( \mu - b \right) + \left[ \frac{\beta S}{\psi(S, I)} - (\mu + \gamma_1 + \gamma_2 + a - qb) \right] I^h$$

$$+ h\lambda_2 \frac{\beta A}{(\mu - b) + \alpha_i A} \frac{\left( \frac{A}{\mu - b} - S \right)^{h-1} \sigma^2 S^2 I^2}{\psi^2(S, I)} + \frac{(h-1) \sigma^2 IS^2}{2 \psi^2(S, I)}.$$ \hspace{1cm} (36)
\[- \left[ \left( \mu + \gamma_1 + \gamma_2 + a - qb - \frac{\beta A}{(\mu - b) + \alpha_1 A} \right) \right. \]
\[- \frac{(h - 1)}{2} \frac{A^2 \sigma^2}{(\mu - b) + \alpha_1 A} e^{-(h-1)/h} \]
\[+ \left. \left( \frac{\beta A}{(\mu - b) + \alpha_1 A} \right) e^{-1/h} \right) \]
\[\left. + \left( \frac{(h - 1) \sigma^2 A^2}{(\mu - b) + \alpha_1 A} \right) e^{(2-h)/h} \lambda_1 + \gamma_2 e^{1/h} \lambda_2 \right] I^h \]
\[- \left[ h (\mu + k) - (h - 1) \gamma_2 e \right] \lambda_2 R^h. \] (34)

We chose \( \epsilon \) to be sufficiently small such that the coefficients of \( (A/(\mu - b) - S)^h \) and \( R^h \) are negative, and as \( (\mu + \gamma_1 + \gamma_2 + a - qb - \beta A/((\mu - b) + \alpha_1 A))/((\mu - b) + \alpha_1 A)^2 > 0 \), we can choose \( \lambda_1 \) and \( \lambda_2 \) being positive such that the coefficient of \( I^h \) is negative. This ends the proof.

Under the Theorems 4 and 6, we have in the case \( h = 2 \) the following corollary.

**Corollary 7.** If the conditions \( R_0 < 1 \) and \( \sigma^2 < 2(A/((\mu - b) + \alpha_1 A))^2[(\mu + \gamma_1 + \gamma_2 + a - qb - \beta A/((\mu - b) + \alpha_1 A)) - (\mu - b + a)]^2 > 0 \), we can choose \( \lambda_1 \) and \( \lambda_2 \) being positive such that the coefficient of \( I^2 \) is negative. This ends the proof.

**Remark 8.** Biologically, the global stability of free disease equilibrium may be interpreted as the inescapable fate of the epidemic extinction, regardless of its initial situation.

5. Persistence

In this section, our main concern is to determine the conditions for the spread and persistence of an infectious disease. Given that the value of the deterministic threshold \( R_0 \) characterizes the dynamical behaviors of system (4) and guarantees persistence or extinction of the disease. Similarly, we define the threshold of our stochastic SIRS epidemic model (6) as follows:

\[ R_s^0 = R_0 \left[ 1 - \frac{\sigma^2 A}{2 \psi((\mu - b) + \alpha_1 A)} \right]. \] (35)

To avoid any ambiguity, we define \( \langle x(t) \rangle = (1/t) \int_0^t x(s) d\sigma \).

**Theorem 9.** If \( R_s^0 > 1 \), then the solution \( (S(t), I(t), R(t)) \) of system (6) with initial value \( (S(0), I(0), R(0)) \in \Delta \) is persistent in the mean. Moreover, we have

\[ \liminf_{t \to \infty} \langle I(t) \rangle \geq I_*, > 0, \]
\[ \liminf_{t \to \infty} \langle R(t) \rangle \geq \frac{\gamma_2 I_*}{(\mu + k)} > 0, \]
\[ \liminf_{t \to \infty} \left\langle \frac{A}{\mu - b} - S(t) \right\rangle \]
\[ \geq \frac{[((\mu + \gamma_1 + \gamma_2 + a) + (\mu - b) + \gamma_2) I_*}{(\mu - b) (\mu + k)} > 0, \] (36)

where

\[ I_* = \frac{(R_0^0 - 1)(\mu + r_2 + \delta + \gamma) \beta^{-1} (\mu - b) (\mu + k) ((\mu - b) + \alpha_1 A)}{(k + b) (\mu - b + a) + \beta A (\mu - b) + A (\mu + k) (\alpha_2 (\mu - b) + \alpha_3 A) > 0. \] (37)

The following result is needed for the proof of Theorem 9 (see [35]).

**Lemma 10.** Let \( h \in \mathbb{C}([0, \infty) \times \Omega, (0, \infty)) \) and \( H \in \mathbb{C}([0, \infty) \times \Omega, \mathbb{R}) \). If there exist positive constants \( \lambda_0 \) and \( \lambda \) such that

\[ \ln h(t) \geq \lambda_0 t - \lambda \int_0^t h(s) ds + H(t) \] a.s. \] (38)

for all \( t \geq 0 \), and \( \lim_{t \to \infty} (H(t)/t) = 0 \) a.s., then

\[ \liminf_{t \to \infty} \frac{(h(t))}{(h(t))} \geq \frac{\lambda_0}{\lambda} \] a.s. \] (39)

**Proof of Theorem 9.** We consider a function \( V_3 \) defined by

\[ V_3 = \omega_1 (S + I + R) + \omega_2 S + \ln I, \] (40)

Furthermore, the \( h \)th moment exponential stability means that the number of infected individuals tends to the state of extinction exponentially fast.
Since $(S(t), I(t), R(t)) \in \Delta$, we get
\[-BSI \psi(S, I) \geq -\frac{BAI}{\mu - b} \]
\[\beta S \psi(S, I) = \frac{BA}{(\mu - b) + \alpha_1 A} \]
\[-\beta (\mu - b) \frac{(A/(\mu - b) - S)}{((\mu - b) + \alpha_1 A)} \psi(S, I) \]
\[-\beta \alpha_2 A I \psi(S, I) \]
\[-\beta \alpha_3 A SI \psi(S, I) \]
\[\geq \beta A \frac{(\mu - b) - \alpha_1 A}{\mu - b} \]
\[-\beta (\mu - b) \frac{(A - \mu - S)}{(\mu - b) + \alpha_1 A} \psi(S, I) \]
\[-\beta A \frac{(\mu - b) + \alpha_1 A}{\alpha_2 + \alpha_3 (\mu - b)} I. \]

Inject those two inequalities in expression of $dV_3$, we get
\[dV_3 \geq \omega_1 \left[ (A/\mu - S) - \frac{\beta A}{(\mu - b) + \alpha_1 A} \right] \]
\[\omega_2 \left[ A - (\mu - b) S - \frac{BAI}{\mu - b} \right] \]
\[(b + k) R \right] dt - \omega_2 \frac{\sigma SI}{\psi(S, I)} dB \]
\[-\omega_2 \frac{\beta A}{(\mu - b) + \alpha_1 A} \left[ (A - \mu - S) \right] \]
\[-\frac{\beta A}{(\mu - b) + \alpha_1 A} \left( A - \frac{\mu - b}{\alpha_2 + \alpha_3 (\mu - b)} \right) I. \]

Then
\[dV_3 \geq \left[ (\omega_1 + \omega_2) (\mu - b) - \frac{\beta (\mu - b)}{(\mu - b) + \alpha_1 A} \right] \]
\[\left[ \beta A \frac{(A - \mu - S)}{(\mu - b) + \alpha_1 A} \right] I dt \]
\[+ \left[ - (\mu + \gamma_1 + \gamma_2 + a - qb) + \frac{\beta A}{(\mu - b) + \alpha_1 A} \right] I dt \]
\[-\frac{\sigma^2 A^2}{2((\mu - b) + \alpha_1 A)^2} \right] dt - (\omega_2 I - 1) \]
\[-\frac{\beta (b + k)}{(\mu + k)((\mu - b) + \alpha_1 A)} \]
\[\omega_1 = \frac{\beta (b + k)}{(\mu + k)((\mu - b) + \alpha_1 A)}, \]
\[\omega_2 = \frac{\beta (\mu - b)}{(\mu + k)((\mu - b) + \alpha_1 A)}. \]

By integration, we get
\[V_3 (X(t)) \geq - \frac{(k + b)(\mu - b)(\mu - b + a + \alpha A)(\alpha_2 + \alpha_3 (\mu - b))}{\beta^{-1}(\mu - b)(\mu - b) + \alpha_1 A} \times \int_0^t I(s) ds + (R_0^s - 1) \]
\[\left[ (\mu + \gamma_1 + \gamma_2 + a - qb) t + V_3 (X(0)) \right] - \int_0^t (\omega_2 I(s) - 1) \frac{\sigma S(s)}{\psi(S(s), I(s))} dB(s). \]

Hence
\[\ln I(t) \geq - \frac{(k + b)(\mu - b)(\mu - b + a) + \beta A(\mu - b) + A(\mu + k)(\alpha_2 (\mu - b) + \alpha_3 A)}{\beta^{-1}(\mu - b)(\mu + k) + \alpha_1 A} \times \int_0^t I(s) ds + (R_0^s - 1) \]
\[\left[ (\mu + \gamma_1 + \gamma_2 + a - qb) t + G(t) \right], \]
where
\[
G(t) = V_3(X(0)) - (\omega_1 + \omega_2)S(t) - \omega_1 I(t) - \omega_1 R(t) - \int_0^t (\omega_2 I(s) - 1) \frac{\sigma S(s)}{\psi(S(s), I(s))} dB(s).
\]

Thus, the strong law of large number for martingales implies that \( \lim_{t \to \infty} (G(t)/t) = 0 \) a.s.

By using Lemma 10, we have
\[
\lim_{t \to \infty} \langle R(t) \rangle \geq \frac{\gamma_1 I^*}{(\mu + k)}.
\]

Next, the third equation of system (6) gives
\[
\frac{R(t) - R(0)}{t} = \gamma_2 \langle I(t) \rangle + (\mu + k) \langle R(t) \rangle.
\]

Then
\[
\lim_{t \to \infty} \langle R(t) \rangle \geq \frac{\gamma_1 I^*}{(\mu + k)}.
\]

Finally, it follow from system (6) that
\[
d(S + I + R) = \left[ (\mu - b) \left( \frac{A}{\mu - b} - S \right) - (\mu - b + a) I - (\mu - b) R \right] dt.
\]

Then
\[
\lim_{t \to \infty} \langle S(t) \rangle \geq \frac{((\mu - b + a)(\mu + k) + (\mu - b) \gamma_2) I^*}{(\mu - b)(\mu + k)}.
\]

6. Extinction

In this section, we investigate the conditions for the extinction of the disease.

Theorem 11. Let \((S(t), I(t), R(t))\) be the solution of system (6) with initial value \((S(0), I(0), R(0))\) in \(\Delta\).

Assume that
\[
(a) \sigma^2 > \frac{\beta^2}{2(\mu + \gamma_1 + \gamma_2 + a - qb)},
\]

or \((b) R_0' < 1,
\]

or \((b) R_0' < 1,
\]

where \(M(t) = \int_0^t (\beta S(s)/\psi(S(s), I(s))) dB(s)\). By the large number theorem for martingale, we have
\[
\lim_{t \to \infty} \frac{M(t)}{t} = 0 \text{ a.s.}
\]

If the condition (a) is satisfied, (58) becomes
\[
\frac{\ln I(t)}{t} = \frac{1}{t} \int_0^t \left( \frac{\beta S(s)}{\psi(S(s), I(s))} - \frac{(\mu + \gamma_1 + \gamma_2 + a - qb)}{2} \right) ds + \frac{\ln I(0)}{t} + \frac{M(t)}{t}.
\]
Taking the limit superior of both sides, we obtain the desired assertion (55).

If the condition (b) is satisfied, then
\[
\frac{\ln I(t)}{t} \leq \left[ \frac{\beta A}{(\mu - b) + \alpha_1 A} - \left( \mu + \gamma_1 + \gamma_2 + a - qb \right) - \frac{\sigma^2}{2} \left( \frac{A^2}{(\mu - b) + \alpha_1 A} \right)^2 \right] + \frac{\ln I(0)}{t} + \frac{M(t)}{t} \leq R_0^t.
\]

Taking the limit superior of both sides, we obtain the desired assertion (56). This finishes the proof.

**Remark 12.** From Theorem 11, we concluded that when the noise is sufficiently small, the value of \( R_0^t \) which is below 1 will lead to the extinction of the disease. Therefore, we consider the value \( R_0^t \) as the threshold of stochastic system (6).

**Remark 13.** If the condition (a) holds, it is easy to prove that \( R_0^t < 1 \); and if the condition (b) holds, we directly have \( R_0^t < 1 \). Therefore an interesting open problem is whether we can establish the extinction of the disease only when \( R_0^t < 1 \).

### 7. Numerical Simulations

In this section, we present the numerical simulations to support the above theoretical results, illustrating extinction and persistence in mean of the disease. We mainly use Milstein’s higher-order method to discrete the system (6). Moreover, we numerically simulate the solution of a corresponding deterministic system for comparison.

**Example 1.** For the deterministic model (4) and its stochastic version (6), the parameters are taken as follows:
\[
A = 0.4, \\
\beta = 0.2, \\
\mu = 0.3, \\
\gamma_1 = 0.01, \\
\gamma_2 = 0.03, \\
b = 0.2, \\
q = 0.1, \\
a = 0.1, \\
k = 0.2, \\
\alpha_1 = 0.05, \\
\alpha_2 = 0.02, \\
\alpha_3 = 0.01.
\]

For system (4), we get \( R_0 = 1.5873 > 1 \); thus it admits a unique endemic equilibrium \( P^* \) which is globally stable for any initial values \( (S(0), I(0), R(0)) \).

For the corresponding stochastic model (6), we choose \( \sigma = 0.5 \); then we have
\[
\beta^2 \left( \frac{2}{(\mu + \gamma_1 + \gamma_2 + a - qb)} - \frac{\sigma^2}{2} \right) = -0.2024 < 0.
\]

Then condition (a) of Theorem 11 is satisfied. We conclude that, for any initial value, \( I(t) \) obeys
\[
\limsup_{t \to \infty} \frac{1}{t} \ln \frac{I(t)}{I(0)} \leq \frac{\beta^2}{2\sigma^2} - (\mu + \gamma_1 + \gamma_2 + a - qb) = -0.34 < 0 \text{ a.s.}
\]

That is, \( I(t) \) will tend to zero exponentially with probability one (see Figure 1(a)).

**Example 2.** We choose the parameter values of our stochastic system (6) as follows:
\[
A = 0.2, \\
\beta = 0.52, \\
\mu = 0.3, \\
\gamma_1 = 0.01, \\
\gamma_2 = 0.03, \\
b = 0.2, \\
q = 0.01, \\
a = 0.1, \\
k = 0.2, \\
\alpha_1 = 0.05, \\
\alpha_2 = 0.02, \\
\alpha_3 = 0.01. \\
\sigma = 0.1.
\]
In this case, we have
\[
R_0^\prime = \frac{\beta A}{[(\mu - b) + \alpha_1 A]} \left( 1 - \frac{\sigma^2 A}{2\beta [(\mu - b) + \alpha_1 A]} \right) = 0.9210 < 1,
\]
(66)
\[
\sigma^2 - \frac{\beta [(\mu - b) + \alpha_1 A] - 0.25 < 0.
\]
Then, condition (b) of Theorem 11 is satisfied. We conclude that, for any initial value, \(I(t)\) obeys
\[
\limsup_{t \to \infty} \frac{1}{t} \ln \frac{I(t)}{I(0)} \leq (R_0^\prime - 1) (\mu + \gamma_1 + \gamma_2 + a - q b)
\]
(67)
\[
= -0.015 < 0 \ a.s.
\]
That is, \(I(t)\) will tend to zero exponentially with probability one (see Figure 1(b)).

Example 3. We choose the parameter values of our stochastic system (4) as follows:
\[
A = 0.2,
\]
\[
\beta = 0.8,
\]
\[
\mu = 0.3
\]
\[
\gamma_1 = 0.1,
\]
\[
\gamma_2 = 0.5,
\]
\[
b = 0.2,
\]
\[
q = 0.01,
\]
\[
a = 0.1,
\]
\[
k = 0.2,
\]
\[
\alpha_1 = 0.05,
\]
\[
\alpha_2 = 0.02,
\]
\[
\alpha_3 = 0.01,
\]
\[
\sigma = 0.1.
\]
(68)
In this case, we have
\[
R_0^\prime = \frac{\beta A}{[(\mu - b) + \alpha_1 A]} \left( 1 - \frac{\sigma^2 A}{2\beta [(\mu - b) + \alpha_1 A]} \right) = 1.4409 > 1.
\]
(69)
Then, according to Theorem 9, the solution of a stochastic system (6) is persistent in mean (see Figures 2 and 3).

8. Conclusion and Future Directions

In this paper, we considered a general stochastic SIRS epidemic model with vertical transmission and transfer from infectious to susceptible. Firstly, we proved that the solution of the stochastic system (6) is positive and bounded. Then, we obtained sufficient conditions for the stochastic stability of disease-free equilibrium by using a suitable Lyapunov function and other techniques of stochastic analysis. Furthermore, we showed that the disease persists when the basic
reproduction number $R_0^s > 1$. Finally, we given tow sufficient conditions for extinction of disease with probability 1.

Besides the white noise perturbation, epidemic models may be perturbed by telegraph noise (or burst noise) which can lead to switch the system from an environmental regime to another [42]. The telegraph noise can be illustrated as a switching between two or more subregimes of different environments [43]. Those switching regimes are often memoryless and the waiting time for the next switching follows the exponential distribution [44]. As a matter of fact, the effect of the telegraph noise on the population dynamics has received great attention recently. For example, Li et al. [45] investigated the threshold dynamics and ergodicity of an SIRS epidemic model with Markovian switching. Liu and Zhu [46] studied the stability and ergodicity of a budworm growth model with random perturbations. They showed that both white noises and regime-switching can change the stability of the model greatly. Liu et al. [47] established an interesting results on the dynamics of a stochastic regime-switching predator-prey model with harvesting and distributed delays. We seek in our future works to see the impact of the telegraph noise on the dynamics of complicated population systems.

Conflicts of Interest

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

References


