

Dynamics of a Stochastic SIR Epidemic Model with Logistic Growth*

Yubo Liu¹, Jianli Li^{1,†} and Daipeng Kuang¹

Abstract In this paper, a stochastic SIR epidemic model with saturated treatment function, non-monotone incidence rate and logistic growth is studied. First, we prove that the stochastic model has a unique global positive solution. Next, by constructing a suitable Lyapunov function, we can show that there exists an ergodic stationary distribution in the random SIR model. Then, we show that a sufficient condition can make the disease tend to extinction. Finally, some numerical simulations are used to prove our analytical result.

Keywords Logistic growth, Saturated treatment, Stationary distribution and ergodicity, Non-monotone incidence, Extinction.

MSC(2010) 37A50, 37H10, 37N25.

1. Introduction

Infectious diseases are caused by pathogens and spread through air, water, food and other vectors. In 1927, the Black Death broke out in London. Through the study of the Black Death, the classical SIR model was developed by Kermack and Mckendrick [14]. Later, they proposed the threshold theory based on the study of this model, which laid foundation for researchers who study the epidemic model. From then on, many researchers had begun to analyze epidemics through mathematical models [6, 11, 21, 29]. In the study of infectious diseases, mathematical models have contributed to the prediction and control of infectious diseases (see [3, 38]).

In the dynamics of infectious diseases, the SIR model divides the total population into the following three categories: Susceptible, Infected and Removed. SIR epidemic model can be used to simulate the behavior of some infectious diseases, such as HIV/AIDS, tuberculosis (TB), measles and dengue [24]. In traditional studies, the authors usually assumed that the incidence of infectious diseases is bilinear $g(I)S = \beta IS$ [28]. However, in real life, when an infectious disease infects a significant number of people, bilinear incidence is not suitable for the study of this situation. Therefore, in order to deal with different situations, many researchers have further investigated the incidence of infectious diseases. Liu, Levin and Iwasa [25] proposed the general nonlinear incidence $g(I)S = \frac{KIP S}{1+aI^q}$, where $K, a, p, q > 0$. In

[†]The corresponding author.

Email address: ybliulyb@163.com (Y. Liu), ljianli18@163.com (J. Li),
KuangDP007@163.com (D. Kuang)

¹College of Mathematics and Statistics, Hunan Normal University, Changsha,
Hunan 410081, China

*The authors were supported by the National Natural Science Foundation of China of China (Grant No. 12071105) and the Key Projects of Science Research in Universities of Anhui Province (Grant No. KJ2021A1049).

1973, there was an outbreak of cholera in southern Italy. Capasso and Serio studied it, and found that a saturated incidence rate $g(I)S = \frac{KIS}{1+aI}$ was more suitable for studying the infectious disease of Bari, where $g(I) = \frac{KI}{1+aI}$ and KI represented the infectious ability of the disease [2, 18, 30, 44]. We make I large enough, and then $g(I)$ tends to a saturation level. In addition, many authors have explored more varied incidences [4, 13, 34, 37, 41, 42].

COVID-19 (Corona Virus Disease 2019) has higher transmissibility than SARS (severe acute respiratory syndrome) [37, 42]. In December 2019, there was an outbreak of COVID-19 in Wuhan, China. The Chinese government has taken effective control measures such as isolating the source of infection, quarantining citizens at home and preventing the gathering of people. The outbreak has been effectively controlled. Interventions have reduced economic losses in the long run. Therefore, the control measures of the government are necessary for the face of the rampant epidemic [7]. In order to simulate this phenomenon, Xiao and Ruan proposed the following incidence [36]

$$g(I) = \frac{KI}{1 + aI^2}. \quad (1.1)$$

Compared with the nonlinear incidence rate proposed by Capasso and Serio, this incidence rate can measure some psychological effects on the population. When there are many infected people in an area, the population often choose to reduce their exposure to the outside world, which leads to a decrease in the infection force of the disease [18, 44]. The specific description is shown in Figure 1 in [36].

Different from the exponential growth model, the logistic growth model focuses on the growth of population over time. At the same time, carrying capacity is also considered with limited resources. Therefore, we have found that studying the logistic growth model is more realistic [23, 43], and it is also reasonable to consider non-monotonic incidence to simulate government interference [17].

Treatment of infected individuals is indispensable for better epidemic control. In classical epidemic models, the authors often use $T(I) = \alpha I$ as a treatment function. It follows that the treatment function $T(I)$ is proportional to I . However, when the infected population is sufficiently large, many people cannot be properly treated due to limited social resources. Thus, Wang and Ruan [35] introduced a constant treatment function, and Wang [33] showed a segmented treatment function. In this paper, we will use a nonlinear treatment function as follows

$$T(I) = \frac{aI}{1 + bI}.$$

It has the advantage of describing the situation, in which the treatment rate will reach the saturation value $\frac{a}{b}$ due to the lack of medical resources and treatment experience [10]. Thus, we can know that the nonlinear treatment function is more reasonable.

According to the above analysis, Ghosh et al. [10] introduced an SIR epidemic model with logistic growth, saturated treatment function as

$$\begin{cases} dS = \left[rS \left(1 - \frac{S}{K} \right) - \frac{\beta SI}{1+aI^2} - \nu S \right] dt, \\ dI = \left[\frac{\beta SI}{1+aI^2} - (\rho + v + \gamma)I - \frac{\alpha u I}{1+buI} \right] dt, \\ dR = \left[\frac{\alpha u I}{1+buI} + \gamma I + \nu S - \delta R \right] dt. \end{cases} \quad (1.2)$$

In addition to being controlled by a special growth rate r , the susceptible are also determined by the carrying capacity K . δ, v are the transition rates from recovered to susceptible status and the mortality due to infectious diseases respectively. β, γ are the transmission rates of the infectious disease and the transition rate from susceptible to recovered respectively. a is saturation factor representing the effect of psychological factors due to the disease. ρ is the natural death rate. α, u, b, ν are the cure rates, the treatment control parameter, the delayed parameter of treatment and the vaccination rate of susceptible respectively. Compared with the model in [11, 29], our model takes into account the saturated treatment function and the non-monotonic incidence rate, which can be used to describe the government's control on the spread of infectious diseases and the limited social medical resources. We assume that all parameters of the above system are positive, and we can derive the basic reproduction number of model (1.2) by using the method of Ma and Zhou [26], $R_0 = \frac{\beta K(r-\nu)}{r(\rho+v+\gamma+\alpha u)}$. In this paper, we denote $\mathbb{R}_+^3 = \{(x_1, x_2, x_3) | x_i > 0, i = 1, 2, 3\}$.

When studying the spread of epidemics, researchers now consider the impact of environmental noise such as high temperature, freezing, drought, humidity and hurricanes. Besides, they show that the existence of random factors such that the development of infectious diseases can be interfered [8, 20, 22, 31, 32, 40]. The stochastic model can make up for the shortcomings of the deterministic model. Gard pointed out that the population dynamics is often disturbed by random perturbations [9], Cai et al. revealed that the outbreak of diseases can be suppressed by white noise [1, 39]. In this article, we will take environmental noise into account because of its impact on infectious diseases, and we can get the following system

$$\begin{cases} dS = \left[rS \left(1 - \frac{S}{K} \right) - \frac{\beta SI}{1+aI^2} - \nu S \right] dt + \sigma_1 S dB_1(t), \\ dI = \left[\frac{\beta SI}{1+aI^2} - (\rho + v + \gamma)I - \frac{\alpha u I}{1+buI} \right] dt + \sigma_2 I dB_2(t), \\ dR = \left[\frac{\alpha u I}{1+buI} + \gamma I + \nu S - \delta R \right] dt + \sigma_3 R dB_3(t), \end{cases} \quad (1.3)$$

where $B_1(t), B_2(t), B_3(t)$ are independent standard Brownian motions, and $\sigma_i > 0, (i = 1, 2, 3)$ denotes the intensity of the Gaussian environmental noise.

The innovation points of this paper are as follows.

- Stochastic model with logistic growth and non-monotonic incidence have no relevant studies. More importantly, due to the complexity of model (1.3), if the treatment rate of model (1.3) is linear, i.e., $b = 0$, we can obtain a threshold.

- Compared with the proof of Theorem 1 in [11], not only we are considering a more complex non-monotonic incidence, but also we can obtain that the system obeys a stationary distribution without considering the condition $\lambda_0 := \mu - \frac{1}{2}(\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2)$, which means that we can get the same conclusion after removing a condition.

- For the incidence $g(I) = \frac{KI}{1+aI^n} (n = 0, 1, 2)$, it is worth noting that we can also use our method to draw corresponding conclusions. Therefore, in this article, we promote the relevant studies.

Throughout this paper, let $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t \geq 0}, \mathbb{P})$ be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t \geq 0}$, and let $\{\mathcal{F}_t\}_{t \geq 0}$ satisfy the usual conditions (i.e., it is increasing and right continuous, while \mathcal{F}_0 contains all \mathbb{P} -null sets). Here, $B(t)$ is a d -dimensional standard Wiener process defined on this complete probability. Let $a \vee b = \max(a, b), a \wedge b = \min(a, b)$. Next, using the above definition, we can consider

the d -dimensional Itô's process, and it has the following form

$$dX(t) = \tilde{f}(X(t), t)dt + \tilde{g}(X(t), t)dB(t), \quad (1.4)$$

and the initial value of this equation is $X(0) = X_0 \in \mathbb{R}^d$, $\tilde{f}(t) \in \Psi^1(\mathbb{R}_+, \mathbb{R}^n)$, $\tilde{g}(t) \in \Psi^2(\mathbb{R}_+, \mathbb{R}^{n \times m})$ are measurable functions, $\Psi^1(\mathbb{R}_+, \mathbb{R}^n)$ is absolutely integrable function space, and $\Psi^2(\mathbb{R}_+, \mathbb{R}^{n \times m})$ is quadratic absolutely integrable function space. Denote the differential operator \mathcal{L} of (1.4) as

$$\mathcal{L} = \frac{\partial}{\partial t} + \sum_{i=1}^d \tilde{f}_i(X, t) \frac{\partial}{\partial X_i} + \frac{1}{2} \sum_{i,j=1}^d [\tilde{g}^T(X, t)\tilde{g}(X, t)]_{ij} \frac{\partial^2}{\partial X_i \partial X_j}.$$

Letting $V(X, t) \in C^{2,1}(\mathbb{R}^d \times [t_0, \infty); \mathbb{R}_+)$ and making \mathcal{L} act on a function $V(X, t)$, we obtain

$$\mathcal{L}V(X, t) = V_t(X, t) + V_X(X, t)\tilde{f}(X, t) + \frac{1}{2}\text{trace}[\tilde{g}^T(X, t)V_{XX}(X, t)\tilde{g}(X, t)],$$

where

$$V_t = \frac{\partial V}{\partial t}, V_X = \left(\frac{\partial V}{\partial X_1}, \dots, \frac{\partial V}{\partial X_d} \right), V_{XX} = \left(\frac{\partial^2}{\partial X_i \partial X_j} \right)_{d \times d}.$$

If $X(t) \in \mathbb{R}^d$, then the Itô's formula is given by

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

The rest of this article is organized as follows. In Section 2, we show the existence and uniqueness of the global positive solution of a stochastic system with white noise. In Section 3, we obtain a sufficient condition that the random system has an ergodic stationary distribution by constructing the Lyapunov function. In Section 4, we show that a sufficient condition can make the disease tend to extinction. In Section 5, we use some numerical simulations to summarize our results and make assumptions about our future research.

2. Existence and uniqueness of the positive solution

Studying the long-term behavior of system (1.3) requires us to know whether this model has a unique global positive solution. Obviously, $S(t), I(t), R(t)$ are non-negative. Next, we need to reveal the existence of a global positive solution (the solution will not explode in a finite time) for system (1.3). It is easy to derive the following theory.

Theorem 2.1. *For any initial condition $(S(0), I(0), R(0)) \in \mathbb{R}_+^3$, there is a unique solution $(S(t), I(t), R(t))$ of the stochastic system (1.3) for $t \geq 0$, and the solution will remain in \mathbb{R}_+^3 with probability one.*

Proof. Since the proof of this theorem is similar to that of Theorem 2.1 in [29], we omit it. \square

3. Existence of the stationary distribution

An aspect of studying epidemic models is to find what conditions make the disease persistent. For deterministic models, we often show the global attractiveness of the endemic equilibrium. For random models, we usually prove that the corresponding model has an ergodic stationary distribution, which implies that the disease will be persistent.

Letting $X(t)$ be a time-homogeneous Markov process in \mathbb{R}_+^l (denote l -dimensional Euclidean space), and $X(t)$ is described by the SDEs

$$dX(t) = b(X)dt + \sum_{r=1}^k f_r(X)dB_r(t),$$

and this diffusion matrix is defined as

$$A(x) = (a_{ij}(x)), a_{ij}(x) = \sum_{r=1}^k f_i^r(x)f_j^r(x).$$

There exists a bounded domain $D \subset \mathbb{R}_+^3$ with the regular boundary Γ , and D has the following properties.

(A1) For any $x \in D$, $\xi = (\xi_1, \xi_2, \xi_3) \in \mathbb{R}_+^3$, there is a positive constant \tilde{M} such that $\sum_{i,j=1}^3 a_{i,j} \xi_i \xi_j \geq \tilde{M} |\xi|^2$.

(A2) For any $x \in \mathbb{R}_+^3 \setminus D$, there exists a nonnegative C^2 -function $V(X, t)$ such that $\mathcal{L}V < 0$.

Lemma 3.1 ([15]). *If (A1) and (A2) hold, then the Markov process $X(t)$ has a stationary distribution $\mu(\cdot)$. Let $f(\cdot)$ be a integrable function with respect to the measure $\mu(\cdot)$. Then,*

$$\mathbb{P}^x \left\{ \lim_{T \rightarrow \infty} \frac{1}{T} \int_0^T f(X(t))dt = \int_{\mathbb{R}_+^3} f(x)\mu(dx) \right\} = 1,$$

for all $x \in \mathbb{R}_+^3$.

Lemma 3.2 (Lemma 3, [11]). *For any $\lambda > 1$, $a \geq 0$, $x \geq 0$, the next inequality holds: $x \leq ax^\lambda + a^{\frac{1}{1-\lambda}}$.*

Theorem 3.1. *If $R_0^s > 1$, assume the initial value $(S(0), I(0), R(0)) \in \mathbb{R}_+^3$. Then, the solution of system (1.3) admits a unique stationary distribution $\mu(\cdot)$, and it has the ergodic property, where*

$$R_0^s = \frac{\beta K(r - \nu - \frac{1}{2}\sigma_1^2)}{r(\rho + v + \gamma + \alpha u + \frac{1}{2}\sigma_2^2)}. \tag{3.1}$$

Proof. In order to reveal that system (1.3) has a unique stationary distribution, we need to verify both conditions (A1) and (A2). First, we prove condition (A1). The diffusion matrix of system (1.3) is expressed as

$$A = \begin{pmatrix} \sigma_1^2 S^2 & & \\ & \sigma_2^2 I^2 & \\ & & \sigma_3^2 R^2 \end{pmatrix}.$$

Let $\tilde{M} = \min_{(S,I,R) \in \tilde{D}_\alpha \subset \mathbb{R}_+^3} \{\sigma_1^2 S^2, \sigma_2^2 I^2, \sigma_3^2 R^2\}$, then

$$\begin{aligned} \sum_{i,j=1}^3 a_{i,j}(S, I, R) \xi_i \xi_j &= \begin{pmatrix} \sigma_1 S \xi_1 & & \\ & \sigma_2 I \xi_2 & \\ & & \sigma_3 R \xi_3 \end{pmatrix} \begin{pmatrix} \sigma_1 S \xi_1 \\ \sigma_2 I \xi_2 \\ \sigma_3 R \xi_3 \end{pmatrix} \\ &= (\sigma_1 S)^2 \xi_1^2 + (\sigma_2 I)^2 \xi_2^2 + (\sigma_3 R)^2 \xi_3^2 \geq \tilde{M} \|\xi\|^2. \end{aligned}$$

For any $(S, I, R) \in \tilde{D}_\alpha$, $\xi = (\xi_1, \xi_2, \xi_3) \in \mathbb{R}_+^3$, where

$$\tilde{D}_\alpha = \left[\frac{1}{\alpha}, \alpha\right] \times \left[\frac{1}{\alpha}, \alpha\right] \times \left[\frac{1}{\alpha}, \alpha\right],$$

α is sufficiently large constant.

Therefore, condition (A1) is satisfied.

Next, we need to construct a non-negative C^2 -function $V(S, I, R)$.

We construct a suitable C^2 -function $W(S, I, R)$ as

$$W(S, I, R) = MV_1 + V_2 + V_3, \quad (3.2)$$

$$V_1 = -\ln I - b_1 \ln S + b_2 I, \quad (3.3)$$

$$V_2 = -\ln R, \quad (3.4)$$

$$V_3 = \frac{1}{1+\theta} (S + I + R)^{1+\theta}, \quad (3.5)$$

where $b_1 = \frac{\beta K}{r}$, $b_2 = \frac{\beta^2 K}{r(\rho+v+\gamma)}$, $0 < \theta < \frac{(\rho+v)\wedge\delta}{3^{\theta/2}(\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2)}$.

For simplicity, we set

$$\xi = (R_0^s - 1) \left(\rho + v + \gamma + \alpha u + \frac{1}{2} \sigma_2^2 \right) > 0,$$

and let M be sufficiently large. Therefore,

$$-M\xi + \delta + \frac{1}{2} \sigma_3^2 + N \leq -2, \quad (3.6)$$

where N will be determined later.

In fact, $W(S, I, R)$ is a continuous function which follows

$$\liminf_{m \rightarrow \infty, (S,I,R) \in \mathbb{R}_+^3 \setminus U_m} W(S, I, R) = +\infty,$$

where $U_m = \left[\frac{1}{m}, m\right] \times \left[\frac{1}{m}, m\right] \times \left[\frac{1}{m}, m\right]$.

Therefore, a non-negative C^2 -function $V(S, I, R)$ is defined by

$$V(S, I, R) = W(S, I, R) - W(S^0, I^0, R^0),$$

where $(S^0, I^0, R^0) \in \mathbb{R}_+^3$ is the minimum point of $W(S, I, R)$.

By Itô's formula, we obtain

$$\mathcal{L}V_1 = -\frac{1}{I} \left[\frac{\beta SI}{1+aI^2} - (\rho+v+r)I - \frac{\alpha u I}{1+buI} \right] + \frac{1}{2} \sigma_2^2$$

$$\begin{aligned}
& -\frac{b_1}{S} \left[rS \left(1 - \frac{S}{K} \right) - \frac{\beta SI}{1 + aI^2} - \nu S \right] + \frac{b_1}{2} \sigma_1^2 \\
& + b_2 \left[\frac{\beta SI}{1 + aI^2} - (\rho + v + \gamma)I - \frac{\alpha u I}{1 + buI} \right] \\
& = -\frac{\beta S}{1 + aI^2} + \left[\rho + v + \gamma + \frac{1}{2} \sigma_2^2 \right] + \frac{\alpha u}{1 + buI} \\
& - b_1 \left[r - \nu - \frac{1}{2} \sigma_1^2 \right] + \frac{b_1 r S}{K} + \frac{b_1 \beta I}{1 + aI^2} \\
& + \frac{b_2 \beta SI}{1 + aI^2} - b_2 (\rho + v + \gamma)I - \frac{b_2 \alpha u I}{1 + buI}.
\end{aligned}$$

From $-\frac{\beta S}{1+aI^2} = -\beta S + \frac{a\beta SI^2}{1+aI^2}$, we have

$$\begin{aligned}
\mathcal{L}V_1 = & - \left[\beta - \frac{b_1 r}{K} \right] S + \left[\rho + v + \gamma + \frac{1}{2} \sigma_2^2 + \frac{\alpha u}{1 + buI} \right] - b_1 \left[r - \nu - \frac{1}{2} \sigma_1^2 \right] \\
& + \frac{b_1 \beta I}{1 + aI^2} + \frac{a\beta SI^2}{1 + aI^2} + \frac{b_2 \beta SI}{1 + aI^2} - b_2 (\rho + v + \gamma)I - \frac{b_2 \alpha u I}{1 + buI}.
\end{aligned}$$

By $b_1 = \frac{\beta K}{r}$, $b_2 = \frac{\beta^2 K}{r(\rho+v+\gamma)}$, we get

$$\begin{aligned}
\mathcal{L}V_1 \leq & \left[\rho + v + \gamma + \alpha u + \frac{1}{2} \sigma_2^2 \right] - \frac{\beta K (r - \nu - \frac{1}{2} \sigma_1^2)}{r} \\
& + \frac{\beta^2 K I}{r} + \frac{a\beta SI^2}{1 + aI^2} + \frac{\beta^3 K S I}{r(\rho + v + \gamma)(1 + aI^2)} - \frac{\beta^2 K I}{r} \\
& = - (R_0^s - 1) \left[\rho + v + \gamma + \alpha u + \frac{1}{2} \sigma_2^2 \right] \\
& + \frac{a\beta SI^2}{1 + aI^2} + \frac{\beta^3 K S I}{r(\rho + v + \gamma)(1 + aI^2)}. \tag{3.7}
\end{aligned}$$

Moreover, using $a^2 + b^2 \geq 2ab$, we obtain

$$\begin{aligned}
\mathcal{L}V_1 \leq & - (R_0^s - 1) \left[\rho + v + \gamma + \alpha u + \frac{1}{2} \sigma_2^2 \right] + \frac{\sqrt{a}\beta}{2} SI + \frac{\beta^3 K}{r(\rho + v + \gamma)} SI \\
& = - (R_0^s - 1) \left[\rho + v + \gamma + \alpha u + \frac{1}{2} \sigma_2^2 \right] + \left[\frac{\sqrt{a}\beta}{2} + \frac{\beta^3 K}{r(\rho + v + \gamma)} \right] SI. \tag{3.8}
\end{aligned}$$

Similarly, one has

$$\begin{aligned}
\mathcal{L}V_2 = & -\frac{\alpha u I}{R(1 + buI)} - \frac{\gamma I}{R} - \frac{\nu S}{R} + \left(\delta + \frac{1}{2} \sigma_3^2 \right) \\
& \leq -\frac{\gamma I}{R} + \left(\delta + \frac{1}{2} \sigma_3^2 \right). \tag{3.9}
\end{aligned}$$

From $a^2 + b^2 + c^2 \leq (a + b + c)^2$, $-(a + b + c)^\theta a \leq -a^{\theta+1}$, similarly, we derive

$$\begin{aligned}
\mathcal{L}V_3 = & (S + I + R)^\theta \left[rS \left(1 - \frac{S}{K} \right) - (\rho + v)I - \delta R \right] \\
& + \frac{\theta}{2} (S + I + R)^{\theta-1} (\sigma_1^2 S^2 + \sigma_2^2 I^2 + \sigma_3^2 R^2)
\end{aligned}$$

$$\begin{aligned}
&\leq (S + I + R)^\theta \left[rS \left(1 - \frac{S}{K}\right) - (\rho + v)I - \delta R \right] \\
&\quad + \frac{\theta}{2} (S + I + R)^{\theta+1} (\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2) \\
&\leq rS(S + I + R)^\theta - \frac{r}{K} S^{\theta+2} - (\rho + v)I^{\theta+1} \\
&\quad - \delta R^{\theta+1} + \frac{\theta}{2} (S + I + R)^{\theta+1} (\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2). \tag{3.10}
\end{aligned}$$

Using $|\sum_{i=1}^K a_i|^n \leq K^{n-1} \sum_{i=1}^K |a_i|^n$, for $\forall n \geq 1$, we obtain

$$(S + I + R)^{1+\theta} \leq 3^\theta (S^{\theta+1} + I^{\theta+1} + R^{\theta+1})$$

and

$$\begin{aligned}
\mathcal{L}V_3 &\leq rS(S + I + R)^\theta - \frac{r}{K} S^{\theta+2} - (\rho + v)I^{\theta+1} - \delta R^{\theta+1} \\
&\quad + \frac{3^\theta \theta}{2} (S^{\theta+1} + I^{\theta+1} + R^{\theta+1}) (\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2) \\
&\leq rS(S + I + R)^\theta - \frac{r}{2K} S^{\theta+2} - \frac{\delta}{2} R^{\theta+1} - \frac{r}{2K} S^{\theta+2} - \frac{\delta}{4} R^{\theta+1} \\
&\quad + \frac{3^\theta \theta}{2} S^{\theta+1} (\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2) - \left[\frac{\rho + v}{4} - \frac{3^\theta \theta}{2} (\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2) \right] I^{\theta+1} \\
&\quad - \left[\frac{\delta}{4} - \frac{3^\theta \theta}{2} (\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2) \right] R^{\theta+1} - \frac{\rho + v}{4} I^{\theta+1} - \frac{\rho + v}{2} I^{\theta+1} \\
&\leq rS(S + I + R)^\theta - \frac{r}{2K} S^{\theta+2} - \frac{\rho + v}{2} I^{\theta+1} - \frac{\delta}{2} R^{\theta+1} - \frac{r}{2K} S^{\theta+2} \\
&\quad + \frac{3^\theta \theta}{2} S^{\theta+1} (\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2) - \frac{\rho + v}{4} I^{\theta+1} - \frac{\delta}{4} R^{\theta+1}.
\end{aligned}$$

Then,

$$\mathcal{L}V_3 \leq -\frac{r}{2K} S^{\theta+2} - \frac{\rho + v}{2} I^{\theta+1} - \frac{\delta}{2} R^{\theta+1} + N, \tag{3.11}$$

where N is defined as

$$\begin{aligned}
N &= \sup_{(S, I, R) \in \mathbb{R}_+^3} \left\{ rS(S + I + R)^\theta - \frac{r}{2K} S^{\theta+2} - \frac{\delta}{4} R^{\theta+1} \right. \\
&\quad \left. + \frac{3^\theta \theta}{2} S^{\theta+1} (\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2) - \frac{\rho + v}{4} I^{\theta+1} \right\} < \infty.
\end{aligned}$$

Next, we will construct a compact subset D_ϵ such that condition (A2) holds. Define the bounded closed set

$$D_\epsilon = \left\{ (S, I, R) \in \mathbb{R}_+^3 : \epsilon^3 \leq S \leq \frac{1}{\epsilon^3}, \epsilon \leq I \leq \frac{1}{\epsilon}, \epsilon^2 \leq R \leq \frac{1}{\epsilon^2} \right\},$$

where $\epsilon > 0$ satisfies the following conditions

$$\epsilon \leq \frac{r}{2MK}, \tag{3.12}$$

$$-2 + M\beta\epsilon + \frac{M\beta^3 K\epsilon}{2\sqrt{ar}(\rho + v + \gamma)} \leq -1, \quad (3.13)$$

$$-2 + \left[\frac{\sqrt{a}\beta}{2} + \frac{\beta^3 K}{r(\rho + v + \gamma)} \right]^{\frac{1}{2+\theta}} M\epsilon \leq -1, \quad (3.14)$$

$$-2 - \frac{\gamma}{\epsilon} + L_1 \leq -1, \quad (3.15)$$

$$-2 - \frac{r}{4K\epsilon^{3\theta+6}} + L_1 \leq -1, \quad (3.16)$$

$$-2 - \left(\frac{\rho + v}{4\epsilon^{\theta+1}} \right) + L_1 \leq -1, \quad (3.17)$$

$$-2 - \left(\frac{\delta}{4\epsilon^{2\theta+2}} \right) + L_1 \leq -1, \quad (3.18)$$

where

$$L_1 = \sup_{(S,I,R) \in \mathbb{R}_+^3} \left\{ \frac{M\beta^3 KS}{2\sqrt{ar}(\rho + v + \gamma)} + M\beta S - \frac{r}{4K} S^{\theta+2} - \frac{\rho + v}{4} I^{\theta+1} - \frac{\delta}{4} R^{\theta+1} \right\} < \infty.$$

We can divide $\mathbb{R}_+^3 \setminus D_\epsilon$ into six domains,

$$\begin{aligned} D_1 &= \{(S, I, R) \in \mathbb{R}_+^3 : 0 \leq S \leq \epsilon^3\}, D_2 = \{(S, I, R) \in \mathbb{R}_+^3 : 0 \leq I \leq \epsilon\}, \\ D_3 &= \{(S, I, R) \in \mathbb{R}_+^3 : 0 \leq R \leq \epsilon^2, I \geq \epsilon\}, D_4 = \left\{ (S, I, R) \in \mathbb{R}_+^3 : S \geq \frac{1}{\epsilon^3} \right\}, \\ D_5 &= \left\{ (S, I, R) \in \mathbb{R}_+^3 : I \geq \frac{1}{\epsilon} \right\}, D_6 = \left\{ (S, I, R) \in \mathbb{R}_+^3 : R \geq \frac{1}{\epsilon^2} \right\}. \end{aligned}$$

Clearly,

$$D_\epsilon^c = D_1 \cup D_2 \cup D_3 \cup D_4 \cup D_5 \cup D_6.$$

Next, we will show

$$\mathcal{L}V \leq -1 \quad \text{on } D_\epsilon^c.$$

From the above findings, we only need to prove that $\mathcal{L}V \leq -1$ holds in $D_i (i = 1, 2, 3, 4, 5, 6)$.

$$\begin{aligned} \mathcal{L}V(S, I, R) &= M\mathcal{L}V_1 + \mathcal{L}V_2 + \mathcal{L}V_3 \\ &\leq -M(R_0^s - 1) \left[\rho + v + \gamma + \alpha u + \frac{1}{2}\sigma_2^2 \right] + \frac{M\beta^3 KSI}{r(\rho + v + \gamma)(1 + aI^2)} \\ &\quad + \frac{Ma\beta SI^2}{1 + aI^2} - \frac{\gamma I}{R} + \delta + \frac{1}{2}\sigma_3^2 - \frac{r}{2K} S^{\theta+2} - \frac{\rho + v}{2} I^{\theta+1} - \frac{\delta}{2} R^{\theta+1} + N \\ &= -M\xi + \frac{Ma\beta SI^2}{1 + aI^2} + \frac{M\beta^3 KSI}{r(\rho + v + \gamma)(1 + aI^2)} - \frac{\gamma I}{R} + \delta \\ &\quad + \frac{1}{2}\sigma_3^2 - \frac{r}{2K} S^{\theta+2} - \frac{\rho + v}{2} I^{\theta+1} - \frac{\delta}{2} R^{\theta+1} + N. \end{aligned}$$

Case 1. If $(S, I, R) \in D_1$,

$$\begin{aligned} \mathcal{L}V &\leq -M\xi + \frac{Ma\beta SI^2}{1 + aI^2} + \frac{M\beta^3 KSI}{r(\rho + v + \gamma)(1 + aI^2)} - \frac{\gamma I}{R} \\ &\quad + \delta + \frac{1}{2}\sigma_3^2 - \frac{r}{2K} S^{\theta+2} - \frac{\rho + v}{2} I^{\theta+1} - \frac{\delta}{2} R^{\theta+1} + N \end{aligned}$$

$$\begin{aligned} &\leq -2 + M\beta S + \frac{M\beta^3 K S}{2\sqrt{ar}(\rho + v + \gamma)} \\ &\leq -2 + M\beta\epsilon + \frac{M\beta^3 K\epsilon}{2\sqrt{ar}(\rho + v + \gamma)}. \end{aligned}$$

By using (3.13), we have

$$\mathcal{L}V \leq -1.$$

Case 2. If $(S, I, R) \in D_2$, following Lemma 3.2, we can obtain

$$\begin{aligned} \mathcal{L}V &\leq -M\xi + \frac{M\sqrt{a}\beta}{2}SI + \frac{M\beta^3 KSI}{r(\rho + v + \gamma)} - \frac{\gamma I}{R} + \delta \\ &\quad + \frac{1}{2}\sigma_3^2 - \frac{r}{2K}S^{\theta+2} - \frac{\rho + v}{2}I^{\theta+1} - \frac{\delta}{2}R^{\theta+1} + N \\ &\leq -2 + \left[\frac{\sqrt{a}\beta}{2} + \frac{\beta^3 K}{r(\rho + v + \gamma)} \right] MSI - \frac{r}{2K}S^{\theta+2} \\ &\leq -2 + \left[\frac{\sqrt{a}\beta}{2} + \frac{\beta^3 K}{r(\rho + v + \gamma)} \right] MSe - \frac{r}{2K}S^{\theta+2} \\ &\leq -2 + \left[\frac{\sqrt{a}\beta}{2} + \frac{\beta^3 K}{r(\rho + v + \gamma)} \right] M\epsilon \left[\frac{1}{\frac{\sqrt{a}\beta}{2} + \frac{\beta^3 K}{r(\rho + v + \gamma)}} S^{\theta+2} \right. \\ &\quad \left. + \left(\frac{\sqrt{a}\beta}{2} + \frac{\beta^3 K}{r(\rho + v + \gamma)} \right)^{\frac{1}{1+\theta}} \right] - \frac{r}{2K}S^{\theta+2} \\ &\leq -2 + \left[\frac{\sqrt{a}\beta}{2} + \frac{\beta^3 K}{r(\rho + v + \gamma)} \right]^{\frac{2+\theta}{1+\theta}} M\epsilon + M\epsilon S^{\theta+2} - \frac{r}{2K}S^{\theta+2} \\ &= -2 + \left[\frac{\sqrt{a}\beta}{2} + \frac{\beta^3 K}{r(\rho + v + \gamma)} \right]^{\frac{2+\theta}{1+\theta}} M\epsilon. \end{aligned}$$

Together with (3.14), we can easily show

$$\mathcal{L}V \leq -1.$$

Case 3. If $(S, I, R) \in D_3$,

$$\begin{aligned} \mathcal{L}V &\leq -M\xi + \frac{Ma\beta SI^2}{1 + aI^2} + \frac{M\beta^3 KSI}{r(\rho + v + \gamma)(1 + aI^2)} - \frac{\gamma I}{R} \\ &\quad + \delta + \frac{1}{2}\sigma_3^2 - \frac{r}{2K}S^{\theta+2} - \frac{\rho + v}{2}I^{\theta+1} - \frac{\delta}{2}R^{\theta+1} + N \\ &\leq -2 - \frac{\gamma I}{R} - \frac{r}{4K}S^{\theta+2} - \frac{\rho + v}{4}I^{\theta+1} - \frac{\delta}{4}R^{\theta+1} + L_1 \\ &\leq -2 + -\frac{\gamma I}{R} + L_1 \\ &\leq -2 - \frac{\gamma}{\epsilon} + L_1. \end{aligned} \tag{3.20}$$

By (3.15), we have

$$\mathcal{L}V \leq -1.$$

Case 4. If $(S, I, R) \in D_4$,

$$\begin{aligned} \mathcal{L}V &\leq -M\xi + \frac{Ma\beta SI^2}{1+aI^2} + \frac{M\beta^3 KSI}{r(\rho+v+\gamma)(1+aI^2)} - \frac{\gamma I}{R} \\ &\quad + \delta + \frac{1}{2}\sigma_3^2 - \frac{r}{2K}S^{\theta+2} - \frac{\rho+v}{2}I^{\theta+1} - \frac{\delta}{2}R^{\theta+1} + N \\ &\leq -2 - \frac{\gamma I}{R} - \frac{r}{4K}S^{\theta+2} - \frac{\rho+v}{4}I^{\theta+1} - \frac{\delta}{4}R^{\theta+1} + L_1 \\ &\leq -2 - \frac{r}{4K\epsilon^{3\theta+6}} + L_1. \end{aligned} \quad (3.21)$$

Following from (3.16), we have

$$\mathcal{L}V \leq -1.$$

Case 5. If $(S, I, R) \in D_5$,

$$\begin{aligned} \mathcal{L}V &\leq -M\xi + \frac{Ma\beta SI^2}{1+aI^2} + \frac{M\beta^3 KSI}{r(\rho+v+\gamma)(1+aI^2)} - \frac{\gamma I}{R} \\ &\quad + \delta + \frac{1}{2}\sigma_3^2 - \frac{r}{2K}S^{\theta+2} - \frac{\rho+v}{2}I^{\theta+1} - \frac{\delta}{2}R^{\theta+1} + N \\ &\leq -2 - \frac{\gamma I}{R} - \frac{r}{4K}S^{\theta+2} - \frac{\rho+v}{4}I^{\theta+1} - \frac{\delta}{4}R^{\theta+1} + L_1 \\ &\leq -2 - \frac{\rho+v}{4\epsilon^{\theta+1}} + L_1. \end{aligned} \quad (3.22)$$

Together with (3.17), one gets

$$\mathcal{L}V \leq -1.$$

Case 6. If $(S, I, R) \in D_6$,

$$\begin{aligned} \mathcal{L}V &\leq -M\xi + \frac{Ma\beta SI^2}{1+aI^2} + \frac{M\beta^3 KSI}{r(\rho+v+\gamma)(1+aI^2)} - \frac{\gamma I}{R} \\ &\quad + \delta + \frac{1}{2}\sigma_3^2 - \frac{r}{2K}S^{\theta+2} - \frac{\rho+v}{2}I^{\theta+1} - \frac{\delta}{2}R^{\theta+1} + N \\ &\leq -2 - \frac{\gamma I}{R} - \frac{r}{4K}S^{\theta+2} - \frac{\rho+v}{4}I^{\theta+1} - \frac{\delta}{4}R^{\theta+1} + L_1 \\ &\leq -2 - \frac{\delta}{4\epsilon^{2\theta+2}} + L_1, \end{aligned} \quad (3.23)$$

and from (3.18), we derive $\mathcal{L}V \leq -1$.

The proof is complete. \square

4. Extinction of the disease

In our study of epidemic models, we are mainly concerned with the conditions that can make the disease tend to extinct. In this section, we will reveal that the disease will be extinct under some conditions and show the state of the susceptible individuals and recovered individuals after disease has been cleared.

Lemma 4.1 ([27]). *Letting $M = \{M_t\}_{t \geq 0}$ be a real-valued continuous local martingale vanishing at $t = 0$, we have*

$$\lim_{t \rightarrow \infty} \langle M, M \rangle_t = \infty \quad a.s. \quad \Rightarrow \quad \lim_{t \rightarrow \infty} \frac{M_t}{\langle M, M \rangle_t} = 0 \quad a.s.$$

and

$$\limsup_{t \rightarrow \infty} \frac{\langle M, M \rangle_t}{t} < \infty \quad a.s. \quad \Rightarrow \quad \lim_{t \rightarrow \infty} \frac{M_t}{t} = 0 \quad a.s.$$

Lemma 4.2. For any initial value $(S(0), I(0), R(0)) \in \mathbb{R}_+^3$, letting $(S(t), I(t), R(t))$ be the solution of stochastic system (1.3), we obtain

$$\begin{aligned} \limsup_{t \rightarrow \infty} (S(t) + I(t) + R(t)) &< \infty, & \limsup_{t \rightarrow \infty} \frac{\ln S(t)}{t} &= 0 \quad a.s., \\ \lim_{t \rightarrow \infty} \frac{S(t)}{t} &= 0, & \lim_{t \rightarrow \infty} \frac{I(t)}{t} &= 0, & \lim_{t \rightarrow \infty} \frac{R(t)}{t} &= 0 \quad a.s., \\ \lim_{t \rightarrow \infty} \frac{\int_0^t \sigma_1 S dB_1(s)}{t} &= 0, & \lim_{t \rightarrow \infty} \frac{\int_0^t \sigma_2 I dB_2(s)}{t} &= 0, & \lim_{t \rightarrow \infty} \frac{\int_0^t \sigma_3 R dB_3(s)}{t} &= 0 \quad a.s. \end{aligned}$$

Proof. (i) According to system (1.3) and $\tilde{N}(t) = S(t) + I(t) + R(t)$, we can get

$$\begin{aligned} \mathcal{L}\tilde{N} &= rS \left(1 - \frac{S}{K}\right) - (\rho + v + \gamma)I - \delta R + rS - rS \\ &= 2rS - \frac{rS^2}{K} - (\rho + v + \gamma)I - \delta R - rS \\ &\leq Kr - \left[\sqrt{\frac{r}{K}}S - \sqrt{Kr}\right]^2 - [(\rho + v + \gamma) \wedge \delta \wedge r]\tilde{N} \\ &\leq Kr - [(\rho + v + \gamma) \wedge \delta \wedge r]\tilde{N}. \end{aligned}$$

Then,

$$d\tilde{N} \leq (Kr - [(\rho + v + \gamma) \wedge \delta \wedge r]\tilde{N})dt + \sigma_1 S dB_1(t) + \sigma_2 I dB_2(t) + \sigma_3 R dB_3(t).$$

By Theorem 4.1 in [5], we know

$$\limsup_{t \rightarrow \infty} (S(t) + I(t) + R(t)) < \infty \quad a.s.$$

In addition, we can easily get

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

(ii) If $\lim_{t \rightarrow \infty} S(t) = 0$, using Itô's formula, we get

$$\begin{aligned} d(\ln I(t)) &= \left[\frac{1}{I} \left(\frac{\beta SI}{1 + aI^2} - (\rho + v + r)I - \frac{\alpha u I}{1 + buI} \right) - \frac{1}{2} \sigma_2^2 \right] dt + \sigma_2 dB_2(t) \\ &\leq \left[\frac{\beta S}{1 + aI^2} - (\rho + v + r + \frac{1}{2} \sigma_2^2) \right] dt + \sigma_2 dB_2(t). \end{aligned} \quad (4.1)$$

Integrating (4.1) from 0 to t , we have

$$\begin{aligned} \frac{\ln I(t) - \ln I(0)}{t} &\leq \frac{1}{t} \int_0^t \frac{\beta S}{1 + aI^2} - (\rho + v + \gamma + \frac{1}{2} \sigma_2^2) ds + \frac{1}{t} \int_0^t \sigma_2 dB_2(s) \\ &\leq \frac{1}{t} \int_0^t \beta S ds - (\rho + v + \gamma + \frac{1}{2} \sigma_2^2) + \frac{M_2(t)}{t}, \end{aligned} \quad (4.2)$$

where $M_2(t) = \int_0^t \sigma_2 dB_2(s)$. Using the strong law of large numbers [19], we have

$$\lim_{t \rightarrow \infty} \frac{M_2(t)}{t} = \lim_{t \rightarrow \infty} \frac{\int_0^t \sigma_2 dB_2(s)}{t} = 0. \quad (4.3)$$

From $\lim_{t \rightarrow \infty} S(t) = 0$, we get

$$\limsup_{t \rightarrow \infty} \frac{\ln I(t)}{t} < -(\rho + v + \gamma + \frac{1}{2}\sigma_2^2) < 0.$$

Therefore, we can get

$$\lim_{t \rightarrow \infty} I(t) = 0. \quad (4.3)$$

Applying Itô's formula again, we get

$$\begin{aligned} d(S + I + R) = & \left[rS \left(1 - \frac{S}{K} \right) - (\rho + v)I - \delta R \right] \\ & + \sigma_1 S dB_1(t) + \sigma_2 I dB_2(t) + \sigma_3 R dB_3(t). \end{aligned} \quad (4.4)$$

Setting $M_3(t) = \int_0^t S(s) dB_1(s)$, we have

$$\langle M_3, M_3 \rangle_t = \int_0^t S^2(s) ds \leq \left[\sup_{t \geq 0} S^2(t) \right] t,$$

By using Lemma 4.1 (see, for details, [5]), from $\limsup_{t \rightarrow \infty} (S(t) + I(t) + R(t)) < \infty$, we can get

$$\lim_{t \rightarrow \infty} \frac{\int_0^t \sigma_1 S(s) dB_1(s)}{t} = 0. \quad (4.5)$$

Similarly, we also get

$$\lim_{t \rightarrow \infty} \frac{\int_0^t \sigma_2 I(s) dB_2(s)}{t} = 0, \quad \lim_{t \rightarrow \infty} \frac{\int_0^t \sigma_3 R(s) dB_3(s)}{t} = 0. \quad (4.6)$$

Integrating both sides of (4.4) and taking the limit on both sides of (4.4), we get

$$\begin{aligned} & \lim_{t \rightarrow \infty} \frac{S(t) + I(t) + R(t)}{t} \\ = & \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t \left[rS \left(1 - \frac{S}{K} \right) - (\rho + v)I - \delta R \right] ds \\ & + \lim_{t \rightarrow \infty} \left[\frac{\int_0^t \sigma_1 S(s) dB_1(s)}{t} + \frac{\int_0^t \sigma_2 I(s) dB_2(s)}{t} + \frac{\int_0^t \sigma_3 R(s) dB_3(s)}{t} \right] \\ \leq & \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t \left[rS \left(1 - \frac{S}{K} \right) - \delta R \right] ds. \end{aligned} \quad (4.7)$$

From $S(t), I(t)$ will go to zero exponentially with probability one, and we can get

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t \delta R ds \leq - \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t \frac{rS^2}{K} ds \leq 0. \quad (4.8)$$

Since the solution $R(t)$ will remain in \mathbb{R}_+^3 with probability one, then

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t R(s) ds = 0. \quad (4.9)$$

For biological reality, we know $\tilde{N}(t) \rightarrow 0$. From (4.3) and (4.9), we know that we must ensure $S(t) \rightarrow 0$. Combining with $\limsup_{t \rightarrow \infty} (S(t) + I(t) + R(t)) < \infty$ *a.s.*, we get

$$\limsup_{t \rightarrow \infty} \frac{\ln S(t)}{t} = 0 \quad \textit{a.s.}$$

□

Theorem 4.1. *For any initial value $(S(0), I(0), R(0)) \in \mathbb{R}_+^3$, let $(S(t), I(t), R(t))$ be the solution of stochastic system (1.3).*

If $R_0^h = \frac{\beta K(r - \nu - \frac{1}{2}\sigma_1^2)}{r(\rho + v + \gamma + \frac{1}{2}\sigma_2^2)} < 1$, then

$$\limsup_{t \rightarrow \infty} \frac{\ln I(t)}{t} < (R_0^h - 1)(\rho + v + \gamma + \frac{1}{2}\sigma_2^2) < 0.$$

That is, the disease will be extinct exponentially. Moreover, we can get

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t S ds = \frac{K(r - \nu - \frac{1}{2}\sigma_1^2)}{r}.$$

Proof. In order to prove the extinction of the disease, we need to prove the following property of $S(t)$. By using Itô's formula, we get

$$d(\ln S(t)) = \left[r - \frac{rS}{K} - \frac{\beta I}{1 + aI^2} - \nu - \frac{\sigma_1^2}{2} \right] dt + \sigma_1 dB_1(t). \quad (4.10)$$

Integrating (4.10) from 0 to t , we have

$$\begin{aligned} \frac{\ln S(t) - \ln S(0)}{t} &= r - \nu - \frac{\sigma_1^2}{2} - \frac{1}{t} \int_0^t \frac{rS}{K} ds - \frac{1}{t} \int_0^t \frac{\beta I}{1 + aI^2} ds + \frac{1}{t} \int_0^t \sigma_1 dB_1(s) \\ &\leq r - \nu - \frac{\sigma_1^2}{2} - \frac{1}{t} \int_0^t \frac{rS}{K} ds + \frac{M_1(t)}{t}, \end{aligned} \quad (4.11)$$

where $M_1(t) = \int_0^t \sigma_1 dB_1(s)$. Using the strong law of large numbers [19], we have

$$\lim_{t \rightarrow \infty} \frac{M_1(t)}{t} = \frac{\int_0^t \sigma_1 dB_1(s)}{t} = 0.$$

Thus, we can obtain

$$\limsup_{t \rightarrow \infty} \frac{\ln S(t) - \ln S(0)}{t} \leq r - \nu - \frac{\sigma_1^2}{2} - \limsup_{t \rightarrow \infty} \frac{1}{t} \int_0^t \frac{rS}{K} ds. \quad (4.12)$$

Then, from Lemma 4.1, we get

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \int_0^t S ds \leq \frac{K(r - \nu - \frac{1}{2}\sigma_1^2)}{r}. \quad (4.13)$$

Next, we can derive extinction of this disease. By using Itô's formula, we get

$$d(\ln I(t)) = \left[\frac{\beta S}{1 + aI^2} - (\rho + v + r + \frac{1}{2}\sigma_2^2) - \frac{\alpha u}{1 + buI} \right] dt + \sigma_2 dB_2(t). \quad (4.14)$$

Integrating (4.14) from 0 to t , we have

$$\begin{aligned} & \frac{\ln I(t) - \ln I(0)}{t} \\ &= \frac{1}{t} \int_0^t \frac{\beta S}{1 + aI^2} ds - (\rho + v + r + \frac{1}{2}\sigma_2^2) - \frac{1}{t} \int_0^t \frac{\alpha u}{1 + buI} ds + \frac{1}{t} \int_0^t \sigma_2 dB_2(s) \\ &\leq \frac{1}{t} \int_0^t \frac{\beta S}{1 + aI^2} ds - (\rho + v + r + \frac{1}{2}\sigma_2^2) + \frac{M_2(t)}{t}. \end{aligned}$$

Then, we can get

$$\frac{\ln I(t) - \ln I(0)}{t} \leq \frac{1}{t} \int_0^t \beta S ds - (\rho + v + r + \frac{1}{2}\sigma_2^2) + \frac{M_2(t)}{t}, \quad (4.15)$$

Taking the superior limit for (4.15), and from (4.3), (4.13) and $R_0^h < 1$, we can get

$$\begin{aligned} & \limsup_{t \rightarrow \infty} \frac{\ln I(t)}{t} \\ &\leq \frac{\beta K(r - \nu - \frac{1}{2}\sigma_1^2)}{r} - (\rho + v + r + \frac{1}{2}\sigma_2^2) \\ &\leq (R_0^h - 1)(\rho + v + r + \frac{1}{2}\sigma_2^2) < 0, \end{aligned}$$

which indicates $\lim_{t \rightarrow \infty} I(t) = 0$. Moreover, using Itô's formula and integrating $d(\ln S(t))$, we obtain

$$\frac{\ln S(t) - \ln S(0)}{t} = r - \nu - \frac{\sigma_1^2}{2} - \frac{1}{t} \int_0^t \frac{rS}{K} ds - \frac{1}{t} \int_0^t \frac{\beta I}{1 + aI^2} ds + \frac{1}{t} \int_0^t \sigma_1 dB_1(s).$$

Therefore,

$$\lim_{t \rightarrow \infty} \frac{\ln S(t) - \ln S(0)}{t} = r - \nu - \frac{\sigma_1^2}{2} - \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t \frac{rS}{K} ds + \lim_{t \rightarrow \infty} \frac{M_1(t)}{t}.$$

Then, we can get

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t S ds = \frac{K(r - \nu - \frac{1}{2}\sigma_1^2)}{r}.$$

This complete the proof. \square

5. Numerical simulation and Conclusion

In this part, we use the results of numerical simulations to prove our findings in the previous section and summarize our results. Using the method developed in [12],

the following discretization equation of stochastic system (1.3) will be obtained

$$\begin{cases} S_{k+1} = S_k + \left[rS_k \left(1 - \frac{S_k}{K}\right) - \frac{\beta S_k I_k}{1+aI_k^2} - \nu S_k \right] \Delta t + \frac{\sigma_1^2}{2} S_k (\xi_k^2 - 1) \Delta t + \sigma_1 S_k \sqrt{\Delta t} \xi_k, \\ I_{k+1} = I_k + \left[\frac{\beta S_k I_k}{1+aI_k^2} - (\rho + v + \gamma) I_k - \frac{\alpha u I_k}{1+buI_k} \right] \Delta t + \frac{\sigma_2^2}{2} I_k (\eta_k^2 - 1) \Delta t + \sigma_2 I_k \sqrt{\Delta t} \eta_k, \\ R_{k+1} = R_k + \left[\frac{\alpha u I_k}{1+buI_k} + \gamma I_k + \nu S_k - \delta R_k \right] \Delta t + \frac{\sigma_3^2}{2} R_k (\zeta_k^2 - 1) \Delta t + \sigma_3 R_k \sqrt{\Delta t} \zeta_k, \end{cases}$$

where the time increment $\Delta t > 0$, ξ_k, η_k, ζ_k are the random variables which obey the Gaussian distribution $N(0, 1)$ for $k = 1, 2, 3, \dots, n$, and they are independent. $\sigma_i^2 > 0$, ($i = 1, 2, 3$) are the intensity of environmental noise.

Table 5-1. List of parameters

Parameters	Definition	Value	Source
r	The intrinsic rate of the susceptible	2.5	[10]
K	The carrying capacity	100	[10]
β	The disease transmission rate	0.1	[10]
ρ	Natural death rate of the population	0.2	[10]
γ	Removal rate of infectious cases	0.7	[10]
v	The death rate due to disease	0.3	[10]
δ	The immune loss rate	0.2	[10]
a	The saturation factor	0.5	[10]
α	Cure rate	0.4	[10]
b	Delayed parameter of treatment	0.05	[10]
ν	Vaccination rate of susceptible	0.2	[10]
u	The treatment control parameter	0.4	[10]

Example 1. In order to ensure the authenticity of the data, the data we cite come from [10] and the stochastic fluctuation $(\sigma_1, \sigma_2, \sigma_3) = (0.01, 0.01, 0.01)$. In this case, $R_0^s = 6.7643 > 1$, which satisfies the condition of Theorem 3.1. As a result, system (1.3) has a ergodic stationary distribution such that the disease is persistent in a long term. Figure 1 confirms this fact.

Example 2. If we decrease the infection rate from β to 0.01, and increase the random disturbance $(\sigma_1, \sigma_2, \sigma_3) = (0.2, 0.2, 0.2)$, and other parameters are the same as Table 5-1. Obviously, we can calculate $R_0^h = 0.75 < 1$, and this is consistent with the condition of Theorem 4.2, which implies that the disease will disappear, when the random perturbation is relatively large. In addition, we can get $\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t S ds = 91.2$, and Figure 2 illustrates this.

In this paper, we study the dynamical behaviors of a stochastic SIR epidemic model with logistic growth, non-monotone incidence rate and saturated treatment function. For deterministic model, Ghosh et al. [10] have derived the basic reproduction number $R_0 = \frac{\beta K(r-\nu)}{r(\rho+v+\gamma+\alpha u)}$. If $R_0 < 1$, system (1.2) has a disease-free equilibrium E_0 . If $R_0 > 1$, model (1.2) has an endemic equilibrium E^* .

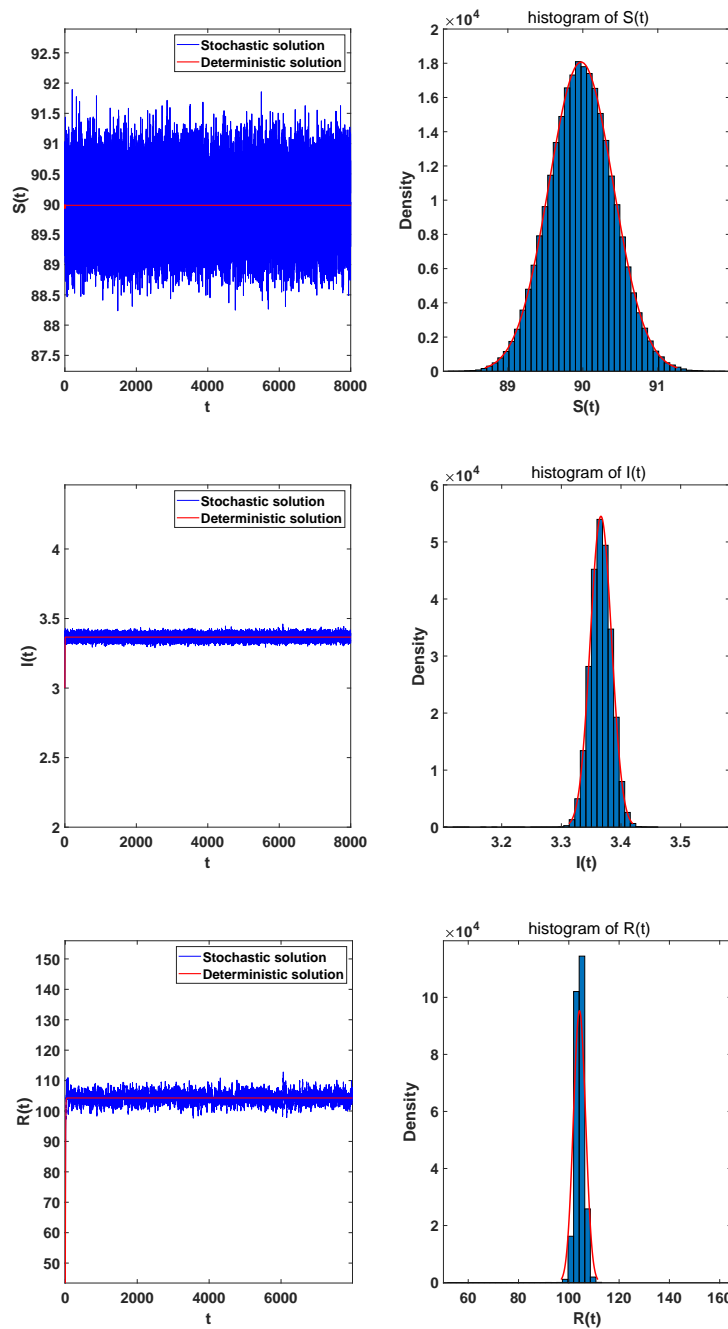


Figure 1. Set the noise intensity $(\sigma_1, \sigma_2, \sigma_3) = (0.01, 0.01, 0.01)$. In the left column, the solution of the stochastic model (1.3) is described as a blue curve, and the solution of the deterministic model (1.2) is described as a red curve. In the right figure, the distribution of the solution $(S(t), I(t), R(t))$ in system (1.3) is described.

For our stochastic model (1.3), we can get the following results.

- If $R_0^s = \frac{\beta K(r - \nu - \frac{1}{2}\sigma_1^2)}{r(\rho + \nu + \gamma + \alpha u + \frac{1}{2}\sigma_2^2)} > 1$, an ergodic stationary distribution exists in the

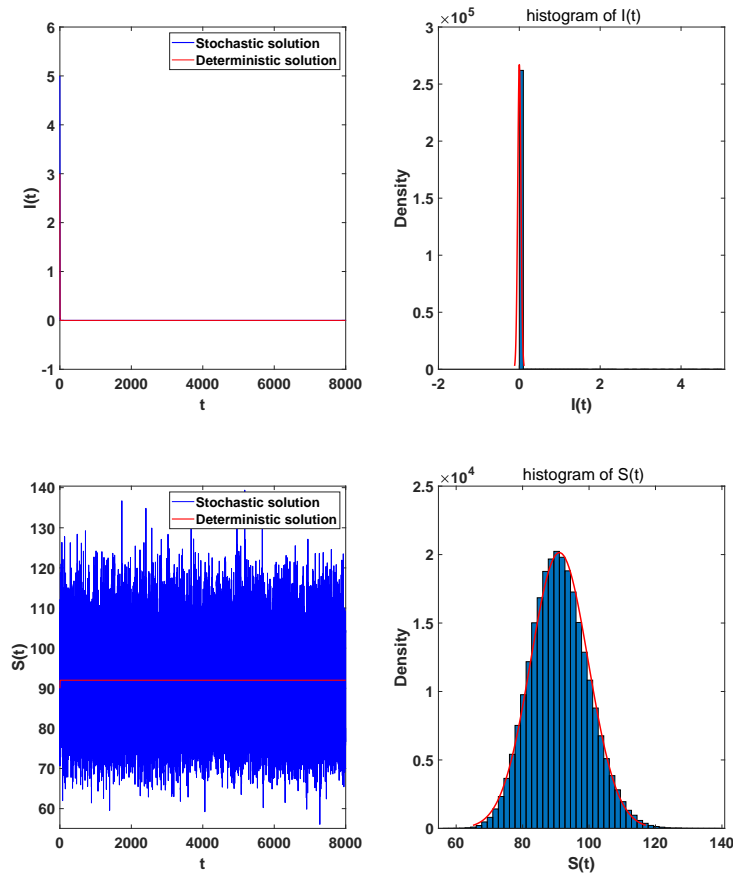


Figure 2. $\beta = 0.01$, the noise intensities $(\sigma_1, \sigma_2, \sigma_3) = (0.2, 0.2, 0.2)$, and other parameters are the same as Table 5-1. The solution of the stochastic model (1.3) is described as a blue curve, and the solution of the deterministic model (1.2) is described as a red curve. In the right figure, the distribution of the solution $I(t)$ in system (1.3) is described.

random SIR model.

- If $R_0^h = \frac{\beta K(r - \nu - \frac{1}{2}\sigma_1^2)}{r(\rho + \nu + \gamma + \frac{1}{2}\sigma_2^2)} < 1$, the disease will tend to extinction exponentially.

We can also compare the expressions for R_0^s and the basic reproduction number of the deterministic system R_0 . Obviously, when we ignore the environmental noise, we show $R_0^s = R_0$, which implies that the stochastic model is an extension of the corresponding deterministic model. Meanwhile, suppose that we consider the linear treatment function $T(I) = \Lambda I$, where Λ is cure rate. Then, $R_0^s = R_0^h$ is a threshold of stochastic model. Further, if we ignore the environmental noise, we have $R_0 = R_0^s = R_0^h$.

The following topics deserve further discussion. We know that environmental noise interference is an essential part of the ecosystem, and the existence of random factors such that the development of an infectious disease can be suppressed. Therefore, we consider the stochastic model with white noise. However, white noise is a continuous stochastic perturbation, and some discontinuous perturbations such that the color and Lévy noises can be further investigated, and the effect of the impulsive can also be considered. At the same time, we can also try to find the

probability density function by solving the Fokker-Planck equation of the stochastic model (1.3). We leave the above topics for our future work.

Acknowledgements

The authors thank the two anonymous reviewers and the editors for their careful reading and helpful comments, which have significantly contributed to improving the quality of our manuscript.

References

- [1] Y. Cai, Y. Kang, M. Banerjee and W. Wang, *A stochastic SIRS epidemic model with infectious force under intervention strategies*, Journal of Differential Equations, 2015, 259(12), 7463–7502.
- [2] V. Capasso and G. Serio, *A generalization of the Kermack-McKendrick deterministic epidemic model*, Mathematical Biosciences, 1978, 42(1–2), 43–61.
- [3] X. Chen, J. Cao, J. H. Park and J. Qiu, *Stability analysis and estimation of domain of attraction for the endemic equilibrium of an SEIQ epidemic model*, Nonlinear Dynamics, 2017, 87, 975–985.
- [4] Y. Chen and W. Zhao, *Dynamical analysis of a stochastic SIRS epidemic model with saturating contact rate*, Mathematical Biosciences and Engineering, 2020, 17(5), 5925–5943.
- [5] A. Din, A. Khan and D. Baleanu, *Stationary distribution and extinction of stochastic coronavirus (COVID-19) epidemic model*, Chaos, Solitons & Fractals, 2020, 139, Article ID 110036, 10 pages.
- [6] A. Dobay, G. E. C. Gall, D. J. Rankin and H. C. Bagheri, *Renaissance model of an epidemic with quarantine*, Journal of Theoretical Biology, 2013, 317, 348–358.
- [7] T. Duan, H. Jiang, X. Deng, et al., *Government Intervention, Risk Perception, and the Adoption of Protective Action Recommendations: Evidence from the COVID-19 Prevention and Control Experience of China*, International Journal of Environmental Research and Public Health, 2020, 17(10), 3387, 17 pages.
- [8] T. Feng, Z. Qiu and Y. Song, *Global analysis of a vector-host epidemic model in stochastic environments*, Journal of the Franklin Institute, 2019, 356(5), 2885–2900.
- [9] T. C. Gard, *Persistence in stochastic food web models*, Bulletin of Mathematical Biology, 1984, 46(3), 357–370.
- [10] J. K. Ghosh, P. Majumdar and U. Ghosh, *Qualitative analysis and optimal control of an SIR model with logistic growth, non-monotonic incidence and saturated treatment*, Mathematical Modelling of Natural Phenomena, 2021, 16(13), 26 pages.
- [11] B. Han, D. Jiang, B. Zhou, et al., *Stationary distribution and probability density function of a stochastic SIRS epidemic model with saturation incidence rate and logistic growth*, Chaos, Solitons & Fractals, 2020, 142, Article ID 110519, 15 pages.

- [12] D. J. Higham, *An Algorithmic Introduction to Numerical Simulation of Stochastic Differential Equations*, SIAM Review, 2001, 43(3), 525–546.
- [13] L. Huo and Y. Dong, *Analyzing the dynamics of a stochastic rumor propagation model incorporating media coverage*, Mathematical Methods in the Applied Sciences, 2020, 43(11), 6903–6920.
- [14] W. O. Kermack and A. G. McKendrick, *A contribution to the mathematical theory of epidemics*, Proceedings of the Royal Society A–Mathematical, Physical and Engineering Sciences, 1927, 115(772), 700–721.
- [15] R. Khasminskii, *Stochastic Stability of Differential Equations (Second Edition)*, Springer, Berlin/Heidelberg, 2012.
- [16] D. Kuang, Q. Yin and J. Li, *Stationary Distribution and Extinction of Stochastic HTLV-I Infection Model with CTL Immune Response under Regime Switching*, Journal of Nonlinear Modeling and Analysis, 2020, 2(4), 585–600.
- [17] G. Lan, Z. Lin, C. Wei and S. Zhang, *A stochastic SIRS epidemic model with non-monotone incidence rate under regime-switching*, Journal of the Franklin Institute, 2019, 356(16), 9844–9866.
- [18] G. M. Leung, T. H. Lam, L. M. Ho, et al., *The impact of community psychological responses on outbreak control for severe acute respiratory syndrome in Hong Kong*, Journal of Epidemiology & Community Health, 2003, 57(11), 857–863.
- [19] R. Sh. Lipster, *A strong law of large numbers for local martingales*, Stochastics, 1980, 3(1–4), 217–228.
- [20] Q. Liu and Q. Chen, *A note on the stationary distribution of a three-species food web stochastic model with generalist predator*, Applied Mathematics Letters, 2021, 114, Article ID 106929, 6 pages.
- [21] Q. Liu and D. Jiang, *Stationary distribution and extinction of a stochastic SIR model with nonlinear perturbation*, Applied Mathematics Letters, 2017, 73, 8–15.
- [22] Q. Liu and D. Jiang, *Dynamical behavior of a stochastic multigroup SIR epidemic model*, Physica A: Statistical Mechanics and its Applications, 2019, 526, Article ID 120975, 19 pages.
- [23] Q. Liu and D. Jiang, *Dynamics of a multigroup SIRS epidemic model with random perturbations and varying total population size*, Communications on Pure and Applied Analysis, 2020, 19(2), 1089–1110.
- [24] Q. Liu and D. Jiang, *Stationary distribution of a stochastic cholera model with imperfect vaccination*, Physica A: Statistical Mechanics and its Applications, 2020, 550, 124031, 16 pages.
- [25] W. Liu, S. A. Levin and Y. Iwasa, *Influence of nonlinear incidence rates upon the behavior of SIRS epidemiological models*, Journal of Mathematical Biology, 1986, 23(2), 187–204.
- [26] Z. Ma, Y. Zhou and J. Wu, *Modeling And Dynamic of Infectious Disease*, Higher Education Press, Beijing, 2009.
- [27] X. Mao, *Stochastic Differential Equations and Applications (Second Edition)*, Horwood, Chichester, 2007.

- [28] Y. Pang, Y. Han and W. Li, *The threshold of a stochastic SIQS epidemic model*, Advances In Difference Equations, 2014, 320, 15 pages.
- [29] S. P. Rajasekar and M. Pitchaimani, *Ergodic stationary distribution and extinction of a stochastic SIRS epidemic model with logistic growth and nonlinear incidence*, Applied Mathematics and Computation, 2020, 377, Article 125143, 15 pages.
- [30] S. Ruan and W. Wang, *Dynamical behavior of an epidemic model with a nonlinear incidence rate*, Journal of Differential Equations, 2003, 188(1), 135–163.
- [31] B. Sounvoravong and S. Guo, *Dynamics of a Diffusive SIR Epidemic Model with Time Delay*, Journal of Nonlinear Modeling and Analysis, 2019, 1(3), 319–334.
- [32] F. Tao and Z. Qiu, *Global analysis of a stochastic TB model with vaccination and treatment*, Discrete and Continuous Dynamical Systems, 2019, 24(6), 2923–2939.
- [33] W. Wang, *Backward bifurcation of an epidemic model with treatment*, Mathematical Biosciences, 2006, 201(1–2), 58–71.
- [34] H. Wang, N. Huang and D. O’Regan, *Dynamics of a stochastic SIQR epidemic model with saturated incidence rate*, Filomat, 2018, 32(15), 5239–5253.
- [35] W. Wang and S. Ruan, *Bifurcation in an epidemic model with constant removal rate of the infectives*, Journal of Mathematical Analysis and Applications, 2004, 291(2), 775–793.
- [36] D. Xiao and S. Ruan, *Global analysis of an epidemic model with nonmonotone incidence rate*, Mathematical Biosciences, 2007, 208(2), 419–429.
- [37] J. Xiao, J. Hu, G. He, et al., *The time-varying transmission dynamics of COVID-19 and synchronous public health interventions in China*, International Journal of Infectious Diseases, 2021, 103, 617–623.
- [38] Y. Xue and T. Li, *Stability and Hopf Bifurcation for a Delayed SIR Epidemic Model with Logistic Growth*, Abstract and Applied Analysis, 2013, Article ID 916130, 11 pages.
- [39] R. Yan and S. Sun, *Stochastic characteristics of a chemostat model with variable yield*, Physica A: Statistical Mechanics and its Applications, 2020, 537, Article ID 122681, 13 pages.
- [40] B. Yang, Y. Cai, K. Wang and W. Wang, *Global threshold dynamics of a stochastic epidemic model incorporating media coverage*, Advances in Difference Equations, 2018, 462, 14 pages.
- [41] X. Zhang and R. Liu, *The stationary distribution of a stochastic SIQS epidemic model with varying total population size*, Applied Mathematics Letters, 2021, 116, 106974, 6 pages.
- [42] X. Zhang and X. Zhang, *The threshold of a deterministic and a stochastic SIQS epidemic model with varying total population size*, Applied Mathematical Modelling, 2021, 91, 749–767.
- [43] Y. Zhang, K. Fan, S. Gao, et al., *Ergodic stationary distribution of a stochastic SIRS epidemic model incorporating media coverage and saturated incidence rate*, Physica A: Statistical Mechanics and its Applications, 2019, 514, 671–685.

-
- [44] X. Zou, Z. Xu, B. Wang, et al., *Study on the COVID-19 infection status, prevention and control strategies among people entering Shenzhen*, BMC Public Health, 2021, 21, 511, 11 pages.