



Extinction and Stationary Distribution of a Stochastic SIRS Epidemic Model Incorporating Media Coverage

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Authors' contributions

This work was carried out in collaboration between all authors. Author Modeste designed the study, analyzed the analyses of the study and all the revisions of the manuscript. Author Eric managed the analyses of the study, wrote the draft and final copies of the manuscript and writing numerical simulations. All authors read and approved the final manuscript.

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Abstract

In this paper, we include stochastic perturbation into SIRS epidemic model incorporating media coverage and study their dynamics. Our model is obtained by taking into account both for demographic stochasticity and environmental fluctuations on contact rate before alert media β_1 . First, we show that the model is biologically well-posed by proving the global existence, positivity and boundedness of solution. Then, sufficient conditions for the extinction of infectious disease is proved. We also established sufficient conditions for the existence of an ergodic stationary distribution to the model. Finally, the theoretical results are illustrated by numerical simulations; in addition we show that the media coverage can reduce the peak of infective individuals via numerical simulations.

Keywords: Stochastic SIRS model; media coverage; stationary distribution; extinction.

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1 Introduction

In Epidemiology, mathematical modeling is an important tool to understand and control the spread of infectious diseases. In 1927, Kermack and Mc Kendricks [1] establish the classical deterministic SIR (Susceptible-Infected-Removed) model. Since then, many authors have studied the SIR disease model. The SIR model is the simplest description for some infectious diseases of permanent or long immunity, such as chickenpox, smallpox, measles, etc. But we know that for some diseases, such as influenza and sexual diseases, recovered or removed individuals lose immunity and return to the susceptible class. In mathematical modeling, compartmental SIRS model is commonly used to describe the dynamics of these diseases. The deterministic SIRS model can be expressed by the following ordinary differential equations:

$$\begin{cases} \frac{dS_t}{dt} = \Lambda - \mu S_t - g(I_t)S_t + \eta R_t \\ \frac{dI_t}{dt} = g(I_t)S_t - (\mu + \alpha + \lambda)I_t \\ \frac{dR_t}{dt} = \lambda I_t - (\mu + \eta)R_t \end{cases} \quad (1.1)$$

The parameters in system (1.1) have the following biological meanings: Λ denotes the influx of individuals into susceptible class, μ represents the natural death rate of S, I, R compartments, λ denotes the recovery rate of the infective individuals, η stands for the rate at which recovered individuals lose immunity and return to the susceptible class and α denotes the death rate due to disease. These parameters are assumed to be nonnegative. Many authors have investigated the SIRS epidemic models [2, 3, 4] and references therein. Transmission of the infection is governed by the incidence rate $g(I)S$ and $g(I)$ is called the infectious force. When an infectious disease appears and starts to spread in a region, one of the immediate actions to take is to educate people about the preventive knowledge of the disease through mass media coverage [5]. Mass media (television, radio, newspapers, billboards and booklets) have been used as a way of delivering preventive health messages due to their potential influence on people's behavior [6]. For instance, during epidemic of human Ebola virus disease in West Africa (2014), media coverage played an important role in helping both the government authority make intervention strategies to contain the disease and people's response to the disease. These messages greatly reduced the contact number per unit time, and therefore decreased the incidence rate. Due to media coverage, changes in human behavior consequently lead to a reduction in the number of the effective contact rate per unit time of susceptible individuals [7, 8]. This demonstrated the importance of considering the infectious forces that include the adaptation of individuals to infection risks under media coverage. Many mathematical models has been formulated to describe the impact of media coverage on the transmission dynamics of infectious disease [9, 10, 11]. Those important and useful works on deterministic models provide a great insight effect on the epidemic model.

However, real biological systems will always be exposed to influences that are not completely understood or not explicitly feasible to model. Population growth in the natural world is inherently stochastic because of numerous unpredictable causes. Ignoring these phenomena in the modeling may affect the analysis of the studied biological systems. Let us note that for human disease, the nature of epidemic growth and spread is inherently random due to the unpredictability of person-to-person contacts [12], and population is subject to a continuous spectrum of disturbances. A way of modeling these elements is by including stochastic influences or noise into epidemic model. Rand and Wilson [13], partitioned this stochastic effects into three classes: demographic fluctuations or internal fluctuations arising from stochastic nature of population, randomness in the environment or external fluctuations and thereby in the parameters affecting the epidemic and measurement errors which are easier to handle as they are not involved in the dynamics. To be realistic, models of biological systems should take into account a change in the environment and the variability of characteristic of individuals. Hence, introduction of stochasticity into population dynamics of diseases can bring to light new insights. In recent years, epidemic models under environmental noise described by stochastic differential equation (SDE) have been studied by many researchers.

Consequently, many authors introduced stochastic perturbations into deterministic models to reveal the effects of environmental noise in biological system and epidemic model [14, 15, 16, 17]. Many realistic stochastic epidemic models come from their deterministic formulations [18, 19, 20]. The main focus of this article is to investigate the effect of external and internal fluctuations on the disease spreading incorporating media coverage. The rest of this paper is organized as follows. In section 2, we derive a stochastic differential SIRS model incorporating media coverage with necessary definitions and preliminaries that will be used in our analysis. The global existence, positivity and the boundedness of the solution are studied in section 3. In this section, sufficient conditions for the extinction and the existence of a unique stationary distribution are obtained. We end this section by numerical simulations to illustrate our theoretical results. In section 4, some conclusions are given.

2 Materials and Methods

Studying epidemic model, leads to two problems. The first one is: will the disease die out? The second problem is: will the disease persist in the population? This section presents the main results of our work. We used the theory of Has'minskii [21], to show that there is an ergodic stationary distribution for the solution of epidemic model (2.4), when the intensity of white noise is small: then the infectious disease described by system (2.4) will be persistent. To prove the extinction of the disease, we showed that I_t will tend to zero exponentially with probability one, by using stochastic calculus and Lyapunov function. In order to confirm our theoretical results, we provide numerical simulations using Milstein's method mentioned in [22]. Figure were developed using Matlab software. To estimate the probability density function of S_t, I_t and R_t , we used kernel density estimation (KDE).

2.1 Stochastic model derivation

Let S_t, I_t and R_t be the number of susceptible, infective and removed individuals at time t , respectively. In the absence of media coverage, in many epidemic models, the bilinear incidence rate $\beta S_t I_t$, are frequently used. The bilinear incidence rate is based on the law of mass action. In the presence of media coverage, social distancing mechanisms come into effect. The reporting by media is assumed to be an increasing function of the number of present infectious cases, as a consequence at the contact rate between susceptible and infectious individuals there is a decreasing function of the number of present infectious cases. Model (1.1) includes the adaption of individuals behavior under media coverage. Especially, Liu and Cui [9], Tchuenche et al.[23], and Sun et al.[10] incorporated a nonlinear function of the number of infective in their transmission term to investigate the effects of media coverage on the transmission dynamics:

$$g(I) = \left(\beta_1 - \frac{\beta_2 I}{b + I} \right) I,$$

where β_1 is the contact rate before media alert; the term $\frac{\beta_2 I}{b + I}$ measures the effect of reduction of the contact rate when infectious individuals are influenced by media alert. Because the coverage report cannot prevent disease from spreading completely we have $\beta_1 \geq \beta_2$. The half-saturation constant $b > 0$ reflects the impact of media coverage on the contact transmission. The function $\frac{I}{b + I}$ is a continuous bounded function which takes into account disease saturation or psychological effects [24]. It follows from above discussion, that SIRS epidemic model incorporating media coverage takes the following form

$$\begin{cases} \frac{dS_t}{dt} = \Lambda - \mu S_t - \left(\beta_1 - \frac{\beta_2 I_t}{b + I_t} \right) S_t I_t + \eta R_t \\ \frac{dI_t}{dt} = \left(\beta_1 - \frac{\beta_2 I_t}{b + I_t} \right) S_t I_t - (\mu + \alpha + \lambda) I_t \\ \frac{dR_t}{dt} = \lambda I_t - (\mu + \eta) R_t \end{cases} \quad (2.1)$$

The basic reproduction number of system (2.1) is $\mathcal{R}^0 = \frac{\Lambda\beta_1}{\mu(\mu + \alpha + \lambda)}$. It is a threshold quantity which determines the extinction and persistence of the epidemic. Model (2.1) has always a disease-free equilibrium $E^0 = \left(\frac{\Lambda}{\mu}, 0, 0\right)$ and an endemic equilibrium $E^* = (S^*, I^*, R^*)$ if $\mathcal{R}^0 > 1$. The disease-free equilibrium is globally asymptotically stable if $\mathcal{R}^0 \leq 1$, that is the disease dies out. The endemic equilibrium is globally asymptotically stable if $\mathcal{R}^0 > 1$, in the set Γ , where $\Gamma = \{(x_1, x_2, x_3) \in \mathbb{R}^3 / x_1 \geq 0, x_2 \geq 0, x_3 \geq 0, 0 < x_1 + x_2 + x_3 \leq \frac{\Lambda}{\mu}\}$. This means that the disease persists in the population. These results of model (2.1) were studied in [9]. There are mainly four types of approaches for applying modeling techniques of stochastic differential equation (SDE) to introduce environmental noises into biological systems. In the first modeling procedure, an SDE model is obtained as approximation to the continuous time Markov chain [25]. The second procedure is the technique of parameter perturbation. Due to external fluctuations, which account for other sources of stochasticity such as environmental fluctuations due to changes in meteorological conditions, variability within and between individuals and other factors, parameters involved in epidemic models are not absolute constants, and they may fluctuate around some average value. In [26, 27], authors demonstrated that one more system parameter(s) can be perturbed stochastically with white noise term to derive environmentally perturbed system. In particular, the transmission of influenza is sensitive to random meteorological factors such as absolute humidity, temperature and precipitation. Random fluctuations in temperature or humidity will have an impact upon the fluctuations in contact rate β . To incorporate the effect of environmental random fluctuations on the transmission dynamics of influenza A in human population based on mathematical model, Keeling and Rohani [28] introduce an additive stochastic perturbation in the contact rate β . In this case, β changes to a random variable $\tilde{\beta}$ with average value β and variance σ^2 . More precisely each infected individual makes

$$\tilde{\beta}dt = \beta dt + \sigma dB_t$$

potentially infectious contacts with individuals in $[t, t+dt]$. Thus the number of potentially infectious contacts that a single infected individual makes with another individual in $[t, t+dt]$ is normally distributed with average βdt and variance $\sigma^2 dt$. In [29, 30, 31, 17], the situation of parameter perturbation is considered. In [29] Weiming et al., assumed that the contact rate β_1 in model (2.1) is subject to environmental noise, that is $\beta_1 dt \rightarrow \beta_1 dt + \sigma dB_t$. Then system (2.1), becomes Itô SDE

$$\begin{cases} dS_t = \left(\Lambda - \mu S_t - \left(\beta_1 - \frac{\beta_2 I_t}{b+I_t} \right) S_t I_t + \eta R_t \right) dt - \sigma S_t I_t dB_t \\ dI_t = \left(\left(\beta_1 - \frac{\beta_2 I_t}{b+I_t} \right) S_t I_t - (\mu + \alpha + \lambda) I_t \right) dt + \sigma S_t I_t dB_t \\ dR_t = (\lambda I_t - (\mu + \eta) R_t) dt \end{cases} \quad (2.2)$$

where B_t is a brownian motion and σ is the intensity of environmental white noise. In [29], the authors proved how environmental fluctuations of the contact coefficient affect the extinction of the disease. Such a form of stochasticity neglects the individual nature of the population. The third approach to include random perturbation in a biological model is considered in [14]: here, the authors formulate the stochastic model by introducing the multiplicative noise terms into the growth equations of susceptible, infective and removed populations. This approach assumed: that stochastic environmental factor acts simultaneously on each individual in the population and that stochastic perturbation is of a white noise type which is directly proportional to the population size. Mathematically speaking, this approach is based on the assumption that the noise is uniform over the state and over time [32]. Following this approach, Yan. Z et al. [33], investigate the effects of environment fluctuations on the disease's dynamics through studying the stochastic dynamics of an SIRS model incorporating media coverage. They got the following system of stochastic differential equation

$$\begin{cases} dS_t = \left(A - dS_t - \left(\beta_1 - \frac{\beta_2 I_t}{\alpha_1 + I_t} \right) \frac{S_t I_t}{1 + \alpha_2 I_t} + f R_t \right) dt + \sigma_1 S_t dB_t^1 \\ dI_t = \left(\left(\beta_1 - \frac{\beta_2 I_t}{\alpha_1 + I_t} \right) \frac{S_t I_t}{1 + \alpha_2 I_t} - (d + e) I_t - \gamma I_t \right) dt + \sigma_2 I_t dB_t^2 \\ dR_t = (\gamma I_t - (f + d) R_t) dt + \sigma_3 R_t dB_t^3 \end{cases} \quad (2.3)$$

where $B_t^i, i = 1, 2, 3$ are independent Brownian motion with $B_0^i = 0, i = 1, 2, 3$ and $\sigma_i, i = 1, 2, 3$ is real constants and known as the intensity of environmental fluctuations. Their authors proved that system (2.3) has a stationary distribution under certain parametric restrictions. The stochastic model (2.3) takes care of the fact that intrinsic growth rates of susceptible, infective and removed individuals are randomly fluctuating due to demographic stochasticity. This formulation takes into account random immigration and emigration or death and birth or individual characteristic of constituents of system (susceptible, infective and removed). The last one, is white noise stochastic perturbations around the positive endemic equilibrium of epidemic models . For example, in [18] Liu investigated an SIRS epidemic model incorporating media coverage with random perturbation. He assumed that stochastic perturbation was of white type noise, which was directly proportional to distance S_t, I_t, R_t from values of S^*, I^*, R^* . By this way, model (2.1) takes the following form:

$$\begin{cases} dS_t = \left(\Lambda - \mu S_t - \left(\beta_1 - \frac{\beta_2 I_t}{b + I_t} \right) S_t I_t + \eta R_t \right) dt + \sigma_1 (S_t - S^*) dB_t^1 \\ dI_t = \left(\left(\beta_1 - \frac{\beta_2 I_t}{b + I_t} \right) S_t I_t - (\mu + \alpha + \lambda) I_t \right) dt + \sigma_2 (I_t - I^*) dB_t^2 \\ dR_t = (\lambda I_t - (\mu + \eta) R_t) dt + \sigma_3 (R_t - R^*) dB_t^3 \end{cases}$$

where $B_t^i, i = 1, 2, 3$ are independent Brownian motion with $B_0^i = 0, i = 1, 2, 3$ and $\sigma_i, i = 1, 2, 3$ is real constants and known as the intensity of environmental fluctuations. In this paper, Liu proves that the endemic equilibrium of the stochastic model is asymptotically stable in the large. Generally the epidemic models described by stochastic differential equations are obtained either by technique of parameter perturbation or by the method introduced in [14]. In this work we will consider a stochastic version of model (2.1) by combining second and third approaches and study their dynamics. One advantage of this approach is that it naturally accounts both for demographic stochasticity and environmental fluctuations on contact rate. That is, the stochastic perturbation is assumed to be of a white noise type which is directly proportional to the number of susceptible, infectious and removed, and $\beta_1 dt$ in model (2.1) is replaced by $\widetilde{\beta}_1 dt = \beta_1 dt + \sigma_4 dB_t^4$. Then we obtain the following stochastic SIRS epidemic model incorporating media coverage:

$$\begin{cases} dS_t = \left(\Lambda - \mu S_t - \left(\beta_1 - \frac{\beta_2 I_t}{b + I_t} \right) S_t I_t + \eta R_t \right) dt + \sigma_1 S_t dB_t^1 \\ \quad - \sigma_4 S_t I_t dB_t^4 \\ dI_t = \left(\left(\beta_1 - \frac{\beta_2 I_t}{b + I_t} \right) S_t I_t - (\mu + \alpha + \lambda) I_t \right) dt + \sigma_2 I_t dB_t^2 \\ \quad + \sigma_4 S_t I_t dB_t^4 \\ dR_t = (\lambda I_t - (\mu + \eta) R_t) dt + \sigma_3 R_t dB_t^3 \end{cases} \quad (2.4)$$

where B_t^i are independent Brownian motion with $B_0^i = 0$ and σ_i denotes the intensity of the white noise, $i = 1, 2, 3, 4$. We notice that, the existence of stationary distribution of model (2.4) seems rare. To fill the gap, this paper is devoted to study the existence of a stationary distribution of model (2.4).

2.2 Preliminaries

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

all \mathbb{P} - null sets), on which are defined all random variables. For a d - dimensional stochastic differential equation

$$dx(t) = f(x(t), t)dt + g(x(t), t)dB(t) \text{ for } t \geq t_0 \tag{2.5}$$

with initial value $x(t_0) = x_0 \in \mathbb{R}^d$, where $B(t)$ denotes d -dimensional standard Brownian motion. The differential operator L of (2.5) is defined [34] by,

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

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

$$LV(x, t) = V_t(x, t) + V_x(x, t)f(x, t) + \frac{1}{2}\text{trace} [g^T(x, t)V_{xx}(x, t)g(x, t)],$$

where: $V_t = \frac{\partial V}{\partial t}$, $V_x = \left(\frac{\partial V}{\partial x_1}, \dots, \frac{\partial V}{\partial x_d} \right)$ and $V_{xx} = \left(\frac{\partial^2 V}{\partial x_i \partial x_j} \right)_{d \times d}$. By virtue of Itô's formula,

$$dV(x(t), t) = LV(x(t), t)dt + V_x(x(t), t)g(x(t), t)dB(t).$$

Here, $C^{2,1}(\mathbb{R}^d \times [t_0, \infty]; \mathbb{R}_+)$ 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 . For technical and biological reasons, we assume that at the initial state, each compartment of our model is not empty; we also consider a closed set Γ^+ as follows

$$\Gamma^+ = \{(x_1, x_2, x_3) \in \mathbb{R}_+^3 / 0 < x_1 + x_2 + x_3 \leq \frac{\Lambda}{\mu}\},$$

with

$$\mathbb{R}_+^3 = \{(x_1, x_2, x_3) \in \mathbb{R}^3 / x_i > 0, \quad i = 1, 2, 3\}.$$

In order to verify the existence of a stationary distribution, we shall introduce a well known result from Has'minskii [21].

Definition 2.1 ([21]). Let $P^x(t, \cdot)$ denote the probability measure induced by $X_t = (S_t, I_t, R_t)$ with initial value x ; that is, $P^x(t, B) = \mathbb{P}(X_t \in B \mid X_0 = x)$, for any borel set $B \subset \mathbb{R}_+^3$. If there is probability measure $P^\infty(\cdot)$ on the measurable space $(\mathbb{R}_+^3; \mathfrak{B}(\mathbb{R}_+^3))$ such that

$$\lim_{t \rightarrow \infty} P^x(t, B) = P^\infty(B) \quad \text{for all } x \in \mathbb{R}_+^3,$$

we then say that model (2.4) has a stationary distribution $P^\infty(\cdot)$.

Let X_t be a regular time-homogeneous Markov process in \mathbb{R}_+^n described by the following stochastic differential equation:

$$dX_t = b(X_t)dt + \sum_{r=1}^k f_r(X_t)dB_t^r,$$

and the diffusion matrix is defined as follows:

$$A(X) = (a_{ij}(x)), \quad a_{ij}(X) = \sum_{r=1}^k f_r^i(X)f_r^j(X).$$

We have the following lemma, which will be useful to prove the theorem related to the stationary distribution for SDE (2.4).

Lemma 2.1 ([21]). *The Markov process $(X_t)_{t \geq 0}$ has a unique stationary distribution $\mu(\cdot)$, if there is a bounded $U \subset \mathbb{R}^n$ with regular boundary such that its closure $\bar{U} \subset \mathbb{R}^n$, having the following properties:*

(i) *In the open domain U and some neighborhood thereof, the smallest eigenvalue of the diffusion matrix $A(X)$ is bounded away from zero.*

(ii) *If $x \in \mathbb{R}^n \setminus U$, the mean time τ at which a path issuing from x reaches the set U is finite, and $\sup_{x \in K} E^x \tau < \infty$ for every compact subset $K \subset \mathbb{R}^n$. Moreover, if $f(\cdot)$ is a function integrable with respect to the measure μ , then*

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

for all $x \in \mathbb{R}^n$.

3 Results and Discussion

3.1 Global existence and positivity

As the solution of SDE (2.4) has biological significance, it should be positive. Moreover, to investigate the dynamical behavior of system (2.4), it is necessary to prove that the solution has a global existence. Our main goal in this section, is to prove that the solution of system (2.4) is global, positive and bounded.

Theorem 3.1. *For any given initial condition $(S_0, I_0, R_0) \in \Gamma^+$, there is a unique solution (S_t, I_t, R_t) to system (2.4) on $t \geq 0$ and the solution will remain in Γ^+ with probability one.*

Proof. Since the coefficients of (2.4) are locally Lipschitz continuous, for any given initial value $(S_0, I_0, R_0) \in \Gamma^+$, there is a unique local solution (S_t, I_t, R_t) on $t \in [0, \tau_e)$, where τ_e is the explosion time [34]. To verify that this solution is global, we only need to prove that $\tau_e = +\infty$ a.s. Firstly, we show that $(S_t, I_t, R_t) \in \mathbb{R}_+^3$ a.s for all $t \in [0, \tau_e)$. Let us consider the stopping time τ^+ , defined by:

$$\tau^+ = \inf \{t \in [0, \tau_e) / S_t \leq 0 \text{ or } I_t \leq 0 \text{ or } R_t \leq 0\} \wedge \tau_e, \text{ with } \inf \emptyset = \infty.$$

We need to prove that $\tau^+ = \tau_e$ a.s. Clearly, $\tau^+ \leq \tau_e$. Obviously, for all $t \in [0, \tau^+)$, $S_t > 0, I_t > 0$ and $R_t > 0$. In view of Itô's formula, for all $t \in [0, \tau^+)$, we obtain:

$$\begin{aligned} \ln(S_t I_t R_t) - \ln(S_0 I_0 R_0) &= \int_0^t \left[\frac{\Lambda}{S_u} - \mu - \left(\beta_1 - \frac{\beta_2 I_u}{b + I_u}\right) I_u + \frac{\eta R_u}{S_u} - \frac{1}{2} \sigma_1^2 - \frac{1}{2} \sigma_4^2 I_u^2 \right] du \\ &+ \int_0^t \left[\left(\beta_1 - \frac{\beta_2 I_u}{b + I_u}\right) S_u - (\mu + \alpha + \lambda) - \frac{1}{2} \sigma_2^2 - \frac{1}{2} \sigma_4^2 S_u^2 \right] du \\ &+ \int_0^t \left[\frac{\lambda I_u}{R_u} - (\mu + \eta) - \frac{1}{2} \sigma_3^2 \right] du \\ &+ \int_0^t [\sigma_1 dB_u^1 + \sigma_2 dB_u^2 + \sigma_3 dB_u^3 + \sigma_4 (S_u - I_u) dB_u^4]. \end{aligned}$$

Since, $\beta_1 \geq \beta_2 \geq 0$ we have:

$$\begin{aligned} \ln(S_t I_t R_t) - \ln(S_0 I_0 R_0) &\geq \int_0^{t \wedge \tau^+} K(S_u, I_u, R_u) du \\ &+ \int_0^{t \wedge \tau^+} [\sigma_1 dB_u^1 + \sigma_2 dB_u^2 + \sigma_3 dB_u^3 + \sigma_4 (S_u - I_u) dB_u^4] = H(t), \quad (3.1) \end{aligned}$$

where

$$K(S_t, I_t, R_t) = -(3\mu + \alpha + \lambda + \eta) - \beta_1 I_t - \frac{\sigma_1^2 + \sigma_2^2 + \sigma_3^2}{2} - \frac{\sigma_4^2(I_t^2 + S_t^2)}{2}.$$

Assume that $\mathbb{P}(\{\tau^+ < \tau_e\}) > 0$. By continuity of the solution of system (2.4), on event $\{\tau^+ < \tau_e\}$, we have

$$S_{\tau^+} I_{\tau^+} R_{\tau^+} = 0.$$

So,

$$\lim_{t \rightarrow \tau^+} \ln(S_t I_t R_t) = -\infty. \quad (3.2)$$

Combining (3.1) and (3.2), we have on $\{\tau^+ < \tau_e\}$ that $-\infty \geq H(\tau^+)$. Therefore,

$$\{\tau^+ < \tau_e\} \subset \{-\infty \geq H(\tau^+)\}.$$

Since, $H(\tau^+)$ is finite on event $\{\tau^+ < \tau_e\}$, we have a contradiction. Hence, $\mathbb{P}(\{\tau^+ < \tau_e\}) = 0$ and $\tau^+ = \tau_e$ a.s.

Now, we prove that $\tau_e = +\infty$ a.s. Let $m_0 > 0$ be sufficiently large so that $S_0 + I_0 + R_0 \in (\frac{1}{m_0}, \frac{\Lambda}{\mu}]$. Define, for each integer $m \geq m_0$, the stopping time

$$\tau_m = \inf\{t \in [0, \tau_e) / S_t + I_t + R_t \notin (\frac{1}{m}, \frac{\Lambda}{\mu}]\}, \text{ with } \inf \emptyset = \infty.$$

Clearly, τ_m is increasing as $m \uparrow \infty$ a.s. Set $\tau_\infty = \lim_{m \rightarrow \infty} \tau_m$. We consider the function V defined for any vector $x = (x_1, x_2, x_3) \in \mathbb{R}_+^3$ by

$$V(x) = \frac{1}{x_1} + \frac{1}{x_2} + \frac{1}{x_3}.$$

By virtue of Itô's formula, for all $t \geq 0$ and $s \in [0, t \wedge \tau_m]$ we get

$$\begin{aligned} dV(S_s, I_s, R_s) &= \left[-\frac{\Lambda}{S_s^2} + \frac{\mu}{S_s} + (\beta_1 - \frac{\beta_2 I_s}{b + I_s}) \frac{I_s}{S_s} - \eta \frac{R_s}{S_s^2} + \frac{\sigma_1^2}{S_s} + \frac{\sigma_4^2 I_s^2}{S_s} \right] ds \\ &+ \left[(\mu + \alpha + \lambda) - (\beta_1 - \frac{\beta_2 I_s}{b + I_s}) \frac{S_s}{I_s} + \frac{\sigma_2^2}{I_s} + \frac{\sigma_4^2 S_s^2}{I_s} \right] ds \\ &+ \left[\frac{(\mu + \eta)}{R_s} - \lambda \frac{I_s}{R_s^2} + \frac{\sigma_3^2}{R_s} \right] ds \\ &- \frac{\sigma_1}{S_s} dB_s^1 - \frac{\sigma_2}{I_s} dB_s^2 - \frac{\sigma_3}{R_s} dB_s^3 + \frac{\sigma_4(I_s^2 - S_s^2)}{I_s S_s} dB_s^4. \end{aligned}$$

Integrate the above equality from 0 to s on both sides yields

$$\begin{aligned} V(S_s, I_s, R_s) - V(S_0, I_0, R_0) &= \int_0^s \left[-\frac{\Lambda}{S_u^2} + \frac{\mu}{S_u} + (\beta_1 - \frac{\beta_2 I_u}{b + I_u}) \frac{I_u}{S_u} - \eta \frac{R_u}{S_u^2} + \frac{\sigma_1^2}{S_u} + \frac{\sigma_4^2 I_u^2}{S_u} \right] du \\ &+ \int_0^s \left[(\mu + \alpha + \lambda) - (\beta_1 - \frac{\beta_2 I_u}{b + I_u}) \frac{S_u}{I_u} + \frac{\sigma_2^2}{I_u} + \frac{\sigma_4^2 S_u^2}{I_u} \right] du \\ &+ \int_0^s \left[\frac{(\mu + \eta)}{R_u} - \lambda \frac{I_u}{R_u^2} + \frac{\sigma_3^2}{R_u} \right] du \\ &+ \int_0^s \left[-\frac{\sigma_1}{S_u} dB_u^1 - \frac{\sigma_2}{I_u} dB_u^2 - \frac{\sigma_3}{R_u} dB_u^3 + \frac{\sigma_4(I_u^2 - S_u^2)}{I_u S_u} dB_u^4 \right]. \end{aligned}$$

Since for any $s \in [0, t \wedge \tau_m]$, we have $(S_s, I_s, R_s) \in (\frac{1}{m}, \frac{\Lambda}{\mu}]^3$ a.s. For any $m \geq m_0$, in view of

$\beta_1 \geq \beta_2 \geq 0$, we have:

$$\begin{aligned} V(S_s, I_s, R_s) - V(S_0, I_0, R_0) &\leq \int_0^s [\mu + (\beta_1 + \beta_2)I_u + \sigma_1^2 + \sigma_4^2 I_u^2] \frac{du}{S_u} \\ &+ \int_0^s [(\mu + \alpha + \lambda) + \sigma_2^2 + \sigma_4^2 S_u^2] \frac{du}{I_u} \\ &+ \int_0^s [(\mu + \eta) + \sigma_3^2] \frac{du}{R_u} \\ &+ \int_0^s \left[-\frac{\sigma_1}{S_u} dB_u^1 - \frac{\sigma_2}{I_u} dB_u^2 - \frac{\sigma_3}{R_u} dB_u^3 + \frac{\sigma_4(I_u^2 - S_u^2)}{I_u S_u} dB_u^4 \right]. \end{aligned}$$

Hence,

$$\begin{aligned} V(S_s, I_s, R_s) - V(S_0, I_0, R_0) &\leq C \int_0^s V(S_u, I_u, R_u) du \\ &+ \int_0^s \left[-\frac{\sigma_1}{S_u} dB_u^1 - \frac{\sigma_2}{I_u} dB_u^2 - \frac{\sigma_3}{R_u} dB_u^3 + \frac{\sigma_4(I_u^2 - S_u^2)}{I_u S_u} dB_u^4 \right] \end{aligned} \quad (3.3)$$

where

$$C = \max \left\{ \mu + (\beta_1 + \beta_2) \frac{\Lambda}{\mu} + \sigma_1^2 + \sigma_4^2 \frac{\Lambda^2}{\mu^2}, \mu + \alpha + \lambda + \sigma_2^2 + \sigma_4^2 \frac{\Lambda^2}{\mu^2}, \mu + \eta + \sigma_3^2 \right\}.$$

Taking the expectation on both sides of (3.3) and applying Fubini's theorem, we have:

$$\mathbb{E}V(S_s, I_s, R_s) \leq V(S_0, I_0, R_0) + C \int_0^s \mathbb{E}V(S_u, I_u, R_u) du.$$

According to Gronwall's Lemma, we deduce that

$$\forall s \in [0, t \wedge \tau_m], \quad \mathbb{E}V(S_s, I_s, R_s) \leq V(S_0, I_0, R_0) \exp(Cs).$$

Thus, for all $t \geq 0$

$$\mathbb{E}V(S_{t \wedge \tau_m}, I_{t \wedge \tau_m}, R_{t \wedge \tau_m}) \leq V(S_0, I_0, R_0) \exp(C(t \wedge \tau_m)) \leq V(S_0, I_0, R_0) \exp(Ct). \quad (3.4)$$

Since $V(S_{t \wedge \tau_m}, I_{t \wedge \tau_m}, R_{t \wedge \tau_m}) > 0$, we have

$$\begin{aligned} \mathbb{E}V(S_{t \wedge \tau_m}, I_{t \wedge \tau_m}, R_{t \wedge \tau_m}) &= \mathbb{E}[V(S_{t \wedge \tau_m}, I_{t \wedge \tau_m}, R_{t \wedge \tau_m}) \mathbf{1}_{(\tau_m \leq t)}] \\ &+ \mathbb{E}[V(S_{t \wedge \tau_m}, I_{t \wedge \tau_m}, R_{t \wedge \tau_m}) \mathbf{1}_{(\tau_m > t)}] \\ &\geq \mathbb{E}[V(S_{\tau_m}, I_{\tau_m}, R_{\tau_m}) \mathbf{1}_{(\tau_m \leq t)}], \end{aligned} \quad (3.5)$$

where $\mathbf{1}_A$ is characteristic function of A . Since each components of $(S_{\tau_m}, I_{\tau_m}, R_{\tau_m})$ are smaller than $\frac{1}{m}$, in view of (3.5) we deduce

$$\mathbb{E}V(S_{t \wedge \tau_m}, I_{t \wedge \tau_m}, R_{t \wedge \tau_m}) \geq 3m \mathbb{P}(\tau_m \leq t). \quad (3.6)$$

From the inequalities (3.4) and (3.6), we get for all $t \geq 0$

$$\mathbb{P}(\tau_m \leq t) \leq \frac{V(S_0, I_0, R_0) \exp(Ct)}{3m}.$$

By letting $m \rightarrow \infty$, we obtain for any $t \geq 0$, $\mathbb{P}(\tau_\infty \leq t) = 0$. Hence, $\mathbb{P}(\tau_\infty = \infty) = 1$. Now, since $\tau_e \geq \tau_\infty$ a.s, then $\tau_e = \infty$ a.s. \square

3.2 Extinction of the disease

Extinction is one of the most basic questions studied in the populations dynamics. Here, it means that the disease will disappear. The following theorem gives a condition for the extinction of a disease expressed in terms of intensities of noise and system parameters. Let us introduce

$$\widehat{\mathcal{R}}^0 = \frac{\mu(\beta_1 + \beta_2)^2}{2\sigma_4^2\Lambda\beta_1} \mathcal{R}^0 - \frac{\sigma_2^2}{2(\mu + \alpha + \lambda)}.$$

Theorem 3.2. *Let (S_t, I_t, R_t) be the solution of system (2.4) with initial condition in Γ^+ . If $\widehat{\mathcal{R}}^0 < 1$, then*

$$\limsup_{t \rightarrow +\infty} \frac{\ln I_t}{t} \leq (\mu + \alpha + \lambda)(\widehat{\mathcal{R}}^0 - 1) < 0 \text{ a.s.}$$

Proof. It follows from Itô's formula that, for all $t \geq 0$

$$\begin{aligned} \ln I_t &= \ln I_0 + \int_0^t \left[(\beta_1 - \frac{\beta_2 I_u}{b + I_u}) S_u - (\mu + \alpha + \lambda) - \frac{1}{2} \sigma_2^2 - \frac{\sigma_4^2 S_u^2}{2} \right] du \\ &\quad + \sigma_2 B_t^2 + M_t \\ &\leq \ln I_0 + \int_0^t \left[-\frac{\sigma_4^2 S_u^2}{2} + (\beta_1 + \beta_2) S_u - (\mu + \alpha + \lambda) - \frac{1}{2} \sigma_2^2 \right] du \\ &\quad + \sigma_2 B_t^2 + M_t, \end{aligned} \tag{3.7}$$

where $M_t = \int_0^t \sigma_4 S_u dB_u^4$. $(M_t)_{t \geq 0}$ is a continuous local martingale whose quadratic variation is $\langle M, M \rangle_t = \sigma_4^2 \int_0^t S_u^2 du$. By virtue of Doob's martingale inequality[35], for any positive numbers ν , T and θ we have

$$\mathbb{P} \left\{ \sup_{0 \leq t \leq T} \left[M_t - \frac{\nu}{2} \langle M, M \rangle_t \right] > \theta \right\} \leq \exp(-\nu\theta). \tag{3.8}$$

Choose now $\nu = c(0 < c < 1)$, $T = k$ and $\theta = \frac{2}{c} \ln k$, for every integer $k \geq 0$. According to (3.8) we obtain

$$\mathbb{P} \left\{ \sup_{0 \leq t \leq k} \left[M_t - \frac{c}{2} \langle M, M \rangle_t \right] > \frac{2}{c} \ln k \right\} \leq \frac{1}{k^2}.$$

By applying Borel-Cantelli's Lemma [34] leads to that for almost all $\omega \in \Omega$, there is a integer k_0 such as

$$\sup_{0 \leq t \leq k} \left[M_t - \frac{c}{2} \langle M, M \rangle_t \right] \leq \frac{2}{c} \ln k, \quad k \geq k_0.$$

That is, for all $0 \leq t \leq k$, $k \geq k_0$

$$M_t \leq \frac{1}{2} c \sigma_4^2 \int_0^t S_u^2 du + \frac{2}{c} \ln k, \text{ a.s.} \tag{3.9}$$

Substituting (3.9) in (3.7), for all $0 \leq t \leq k$, $k \geq k_0$, we obtain

$$\begin{aligned} \ln I_t &\leq \ln I_0 + \int_0^t \left[-\frac{1}{2} (1 - c) \sigma_4^2 S_u^2 + (\beta_1 + \beta_2) S_u - (\mu + \alpha + \lambda) - \frac{1}{2} \sigma_2^2 \right] du \\ &\quad + \sigma_2 B_t^2 + \frac{2}{c} \ln k, \text{ a.s.} \end{aligned}$$

Since

$$-\frac{1}{2} (1 - c) \sigma_4^2 S_u^2 + (\beta_1 + \beta_2) S_u - (\mu + \alpha + \lambda) - \frac{1}{2} \sigma_2^2 \leq \frac{(\beta_1 + \beta_2)^2}{2(1 - c) \sigma_4^2} - (\mu + \alpha + \lambda) - \frac{1}{2} \sigma_2^2,$$

we have

$$\ln I_t \leq \ln I_0 + \left[\frac{(\beta_1 + \beta_2)^2}{2(1-c)\sigma_4^2} - (\mu + \alpha + \lambda) - \frac{1}{2}\sigma_2^2 \right] t + \sigma_2 B_t^2 + \frac{2}{c} \ln k.$$

Thus, for $k - 1 \leq t \leq k$, we obtain

$$\frac{\ln I_t}{t} \leq \frac{\ln I_0}{t} + \left[\frac{(\beta_1 + \beta_2)^2}{2(1-c)\sigma_4^2} - (\mu + \alpha + \lambda) - \frac{1}{2}\sigma_2^2 \right] + \sigma_2 \frac{B_t^2}{t} + \frac{2}{c} \frac{\ln k}{k-1}.$$

Now, letting $k \rightarrow +\infty$, so $t \rightarrow +\infty$ and applying the strong law of large numbers to the Brownian motion [34] we have,

$$\limsup_{t \rightarrow +\infty} \frac{\ln I_t}{t} \leq \frac{(\beta_1 + \beta_2)^2}{2(1-c)\sigma_4^2} - (\mu + \alpha + \lambda) - \frac{1}{2}\sigma_2^2 \text{ a.s.}$$

Finally, by sending $c \rightarrow 0$, in view of $\widehat{\mathcal{R}}^0 < 1$, we obtain

$$\limsup_{t \rightarrow +\infty} \frac{\ln I_t}{t} \leq (\mu + \alpha + \lambda)(\widehat{\mathcal{R}}^0 - 1) < 0 \text{ a.s.}$$

□

3.3 Existence of the stationary distribution

We now establish the main Theorem of this section.

Theorem 3.3. Assume that $\mathcal{R}^0 > 1$. Let $E^* = (S^*, I^*, R^*)$ be the endemic equilibrium of system (2.1). If the following conditions hold $0 < \delta < \min\{m_1 S^{*2}, m_2 I^{*2}, m_3 R^{*2}\}$, where

$$\begin{aligned} m_1 &= (\varpi_3 + \varpi_4)\mu - (\varpi_3 + \varpi_4)\sigma_1^2 - \varpi_2 I^* \sigma_4^2 \\ m_2 &= \varpi_3(\mu + \alpha + \lambda) + \varpi_4(\mu + \alpha) - (\varpi_3 + \varpi_4)\sigma_2^2 \\ m_3 &= \varpi_4\mu + \varpi_1(\mu + \eta) - (\varpi_1 + \varpi_4)\sigma_3^2 \\ \delta &= (\varpi_3 + \varpi_4)S^{*2}\sigma_1^2 + (\varpi_3 + \varpi_4 + \frac{\varpi_2}{2})I^{*2}\sigma_2^2 + (\varpi_1 + \varpi_4)R^{*2}\sigma_3^2 \\ &\quad + \varpi_2 S^{*2} I^* \sigma_4^2 \\ \varpi_1 &= \alpha\eta(\beta_1 b + I^*(\beta_1 - \beta_2)) \\ \varpi_2 &= (2\lambda\mu(2\mu + \alpha + \lambda) + \eta\lambda(2\mu + \alpha))(b + I^*) \\ \varpi_3 &= 2\lambda\mu(\beta_1 b + I^*(\beta_1 - \beta_2)) \\ \varpi_4 &= \eta\lambda(\beta_1 b + I^*(\beta_1 - \beta_2)) \end{aligned} \tag{3.10}$$

then for any initial value in $(S_0, I_0, R_0) \in \Gamma^+$, system (2.4) has unique stationary distribution $\mu(\cdot)$ and solution (S_t, I_t, R_t) have ergodic property.

Proof. Since $\mathcal{R}^0 > 1$, there is a positive endemic equilibrium $E^* = (S^*, I^*, R^*)$ of system (2.1). Let us consider the functions $\Theta_1, \Theta_2, \Theta_3, \Theta_4$ defined for any vector $x = (x_1, x_2, x_3) \in \mathbb{R}_+^3$ by

$$\begin{aligned} \Theta_1(x_1, x_2, x_3) &= \frac{(x_3 - R^*)^2}{2}, \quad \Theta_2(x_1, x_2, x_3) = x_2 - I^* - I^* \log \left(\frac{x_2}{I^*} \right), \\ \Theta_3(x_1, x_2, x_3) &= \frac{(x_1 - S^* + x_2 - I^*)^2}{2} \\ \Theta_4(x_1, x_2, x_3) &= \frac{(x_1 - S^* + x_2 - I^* + x_3 - R^*)^2}{2}. \end{aligned}$$

We will show that model (2.4) admits a stationary distribution by considering the Lyapunov function

$$\Theta = \varpi_1 \Theta_1 + \varpi_2 \Theta_2 + \varpi_3 \Theta_3 + \varpi_4 \Theta_4, \tag{3.11}$$

where $\varpi_1, \varpi_2, \varpi_3$ and ϖ_4 are defined in (3.10). The non-negativity of Θ can be derived from $u - 1 - \log u \geq 0, \forall u > 0$. An application of the differential operator L to Θ_1 yields for any $t \geq 0$

$$L\Theta_1(S_t, I_t, R_t) = (R_t - R^*)(\lambda I_t - (\mu + \eta)R_t) + \frac{1}{2}\sigma_3^2 R_t^2.$$

At the equilibrium state E^* we have $\lambda I^* - (\mu + \eta)R^* = 0$, hence

$$L\Theta_1(S_t, I_t, R_t) = -(\mu + \eta)(R_t - R^*)^2 + \lambda(I_t - I^*)(R_t - R^*) + \frac{1}{2}\sigma_3^2(R_t - R^* + R^*)^2.$$

Using the inequality $\frac{(a+b)^2}{2} \leq a^2 + b^2$, we derive

$$L\Theta_1(S_t, I_t, R_t) \leq -(\mu + \eta - \sigma_3^2)(R_t - R^*)^2 + \lambda(I_t - I^*)(R_t - R^*) + \sigma_3^2 R^{*2}. \quad (3.12)$$

Next, we calculate $L\Theta_2$

$$L\Theta_2(S_t, I_t, R_t) = \left(1 - \frac{I^*}{I_t}\right) \left((\beta_1 - \frac{\beta_2 I_t}{b + I_t}) S_t I_t - (\mu + \alpha + \lambda) I_t \right) + \frac{I^*}{2} (\sigma_2^2 + \sigma_4^2 S_t^2). \quad (3.13)$$

At the equilibrium state E^* , we remark that

$$\mu + \alpha + \lambda = (\beta_1 - \frac{\beta_2 I^*}{b + I^*}) S^*. \quad (3.14)$$

Substituting (3.14) into (3.13) and using $\frac{(a+b)^2}{2} \leq a^2 + b^2$, we get

$$L\Theta_2(S_t, I_t, R_t) \leq (S_t - S^*)(I_t - I^*) \frac{\beta_1 b + I^*(\beta_1 - \beta_2)}{b + I^*} + \frac{I^* \sigma_2^2}{2} + I^* \sigma_4^2 (S_t - S^*)^2 + I^* \sigma_4^2 S^{*2}. \quad (3.15)$$

We, now calculate $L\Theta_3$

$$L\Theta_3(S_t, I_t, R_t) = (S_t - S^* + I_t - I^*)(\Lambda - \mu S_t - (\mu + \alpha + \lambda)I_t + \eta R_t) + \frac{1}{2}\sigma_1^2 S_t^2 + \frac{1}{2}\sigma_2^2 I_t^2.$$

Using $\Lambda = \mu S^* + (\mu + \alpha + \lambda)I^* - \eta R^*$ and $\frac{(a+b)^2}{2} \leq a^2 + b^2$ we have

$$L\Theta_3(S_t, I_t, R_t) \leq -(\mu - \sigma_1^2)(S_t - S^*)^2 - (\mu + \alpha + \lambda - \sigma_2^2)(I_t - I^*)^2 - (2\mu + \alpha + \lambda)(S_t - S^*)(I_t - I^*) + \eta(S_t - S^*)(R_t - R^*) + \eta(I_t - I^*)(R_t - R^*) + \sigma_1^2 S^{*2} + \sigma_2^2 I^{*2}. \quad (3.16)$$

At last, similarly for the function $L\Theta_4$, we obtain

$$L\Theta_4(S_t, I_t, R_t) \leq -(\mu - \sigma_1^2)(S_t - S^*)^2 - (\mu + \alpha - \sigma_2^2)(I_t - I^*)^2 - (\mu - \sigma_3^2)(R_t - R^*)^2 - (2\mu + \alpha)(S_t - S^*)(I_t - I^*) - 2\mu(S_t - S^*)(R_t - R^*) - (2\mu + \alpha)(I_t - I^*)(R_t - R^*) + \sigma_1^2 S^{*2} + \sigma_2^2 I^{*2} + \sigma_3^2 R^{*2}. \quad (3.17)$$

Combining (3.12),(3.15)-(3.16)-(3.17) and multiplying appropriately by coefficients $\varpi_1, \varpi_2, \varpi_3, \varpi_4$ of (3.11) determined in (3.10), we eliminate the product $(I_t - I^*)(R_t - R^*), (S_t - S^*)(R_t - R^*)$ and $(I_t - I^*)(S_t - S^*)$ from $L\Theta$. Consequently,

$$L\Theta(S_t, I_t, R_t) \leq -m_1(S_t - S^*)^2 - m_2(I_t - I^*)^2 - m_3(R_t - R^*)^2 + \delta, \quad (3.18)$$

where m_1, m_2, m_3 and δ are defined in (3.10). Since $0 < \delta < \min\{m_1 S^{*2}, m_2 I^{*2}, m_3 R^{*2}\}$, then the ellipsoid

$$m_1(S_t - S^*)^2 + m_2(I_t - I^*)^2 + m_3(R_t - R^*)^2 = \delta$$

lies entirely in \mathbb{R}_+^3 . We can take U to be any neighborhood of the ellipsoid such as $\bar{U} \subset \mathbb{R}_+^3$. So for $(S, I, R) \in \mathbb{R}_+^3 \setminus U$, $L\Theta(S, I, R) < -C$ (C is a positive constant). Hence, with the reference to Zhu et al.[36] condition (ii) in Lemma 2.1 is satisfied. Besides, there is $M = \min\{\sigma_1 S^2, \sigma_2 I^2, \sigma_3 R^2\} > 0$, such as

$$\sum_{i,j=1}^3 a_{ij}(S, I, R)\xi_i\xi_j = \sigma_1^2 S^2 \xi_1^2 + \sigma_2^2 I^2 \xi_2^2 + \sigma_3^2 R^2 \xi_3^2 + \sigma_4^2 S^2 I^2 (\xi_1 - \xi_2)^2 \geq M\|\xi\|^2,$$

for all $(S, I, R) \in \bar{U}$ and $\xi \in \mathbb{R}_+^3$. Thus, the diffusion matrix associated to (2.4) is uniformly elliptic in U . Then, by Rayleigh's principle in [37], condition (i) in Lemma 2.1 is also satisfied. Therefore, we can conclude that stochastic system (2.4) has a stationary distribution $\mu(\cdot)$ and it is ergodic. \square

3.4 Numerical simulations and discussions

Here we always choose initial value as $(S_0, I_0, R_0) = (15, 5, 1)$.

3.4.1 Extinction and impact of media coverage

In Figure 1 and Figure 2, we choose parameters as follows

$$\begin{aligned} \Lambda &= 35, \beta_1 = 0.02, \beta_2 = 0.018, \eta = 0.02, \\ \mu &= 0.5, \alpha = 0.6, \lambda = 0.08, b = 30. \end{aligned} \tag{3.19}$$

We have $\mathcal{R}^0 = 1.186 > 1$. In Figure 1, we choose $\sigma_1 = 0.01, \sigma_2 = 0.8, \sigma_3 = 0.03, \sigma_4 = 0.04$. Then, $\widehat{\mathcal{R}}^0 = 0.1112 < 1$. We can therefore conclude by Theorem 3.2, that for any initial value $(S_0, I_0, R_0) \in \Gamma^+$, I_t obeys

$$\limsup_{t \rightarrow +\infty} \frac{\ln I_t}{t} \leq -1.049 \text{ a.s.}$$

So, I_t will tend to zero exponentially with probability one. Which is a phenomenon different from its corresponding deterministic model (2.1)(see Figure 1). To see the disease dynamics of model (2.4), in Figure 2 we choose $\sigma_1 = 0.01, \sigma_2 = 0.08, \sigma_3 = 0.03, \sigma_4 = 0.004$. Then $\widehat{\mathcal{R}}^0 = 38.2375 > 1$. In this case, our simulations suggests that I_t is stochastically persistent (see Figure 2). By Theorem 3.2, we can know, if $\widehat{\mathcal{R}}^0 < 1$, then the infectious disease of system (2.4) goes to extinction almost surely. In other words, when the white noise is large enough such that $\sigma_4^2(2(\mu + \alpha + \lambda) + \sigma_2^2) > (\beta_1 + \beta_2)^2$ is satisfied, the disease we also die. So, large noise can lead to the extinction of disease. Figure 1 and Figure 2 suggests that $\widehat{\mathcal{R}}^0$ is the threshold associated with the extinction of infectious disease of system (2.4). To illustrate the impact of media on the spread of an epidemic, in case of endemic situation, we consider a variation of the parameter of the media coverage $\beta_2 = 0, 0.011, 0.018$ with parameters as follows

$$\begin{aligned} \Lambda &= 35, \beta_1 = 0.028, \eta = 0.02, \mu = 0.5, \alpha = 0.6, \lambda = 0.08, b = 30, \\ \sigma_1 &= 0.01, \sigma_2 = 0.08, \sigma_3 = 0.03, \sigma_4 = 0.004. \end{aligned} \tag{3.20}$$

We remark that increasing the media coverage parameter, decreases magnitude of infected individuals (Figure 3). This states that the media coverage can reduce the propagation of the disease among the population.

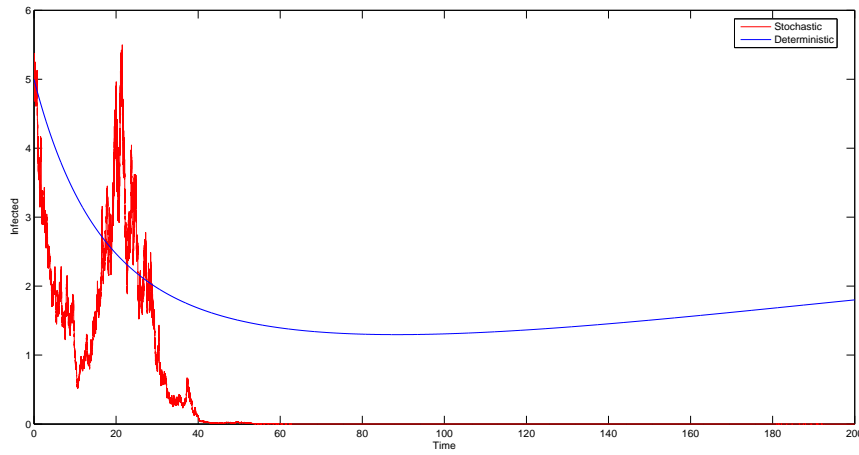


Fig. 1. Trajectories of I_t for model (2.4) and model (2.1). Parameters are taken as (3.19). $\sigma_1 = 0.01, \sigma_2 = 0.8, \sigma_3 = 0.03, \sigma_4 = 0.04$ and $\widehat{\mathcal{R}}^0 = 0.1112 < 1$.

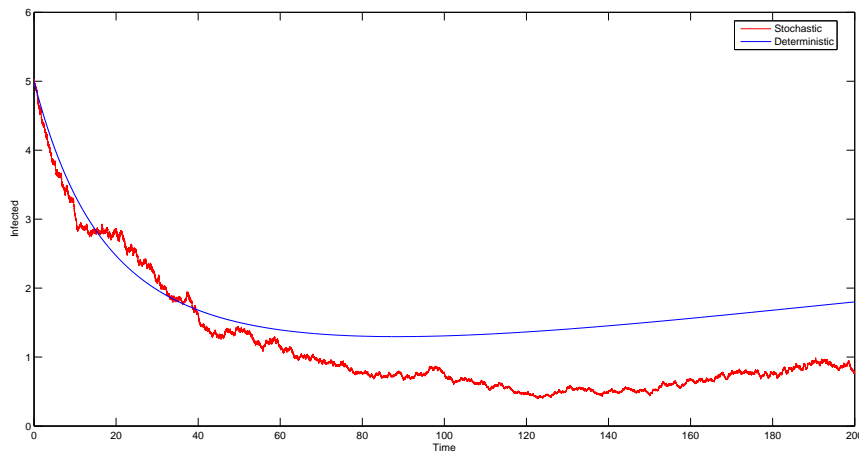


Fig. 2. Trajectories of I_t for model (2.4) and model (2.1). Parameters are taken as (3.19). $\sigma_1 = 0.01, \sigma_2 = 0.08, \sigma_3 = 0.03, \sigma_4 = 0.004$ and $\widehat{\mathcal{R}}^0 = 38.2375 > 1$.

3.4.2 Stationary distribution

In Figure 4 and Figure 5, we choose parameters as follows

$$\begin{aligned} \Lambda &= 20, \beta_1 = 0.1, \beta_2 = 0.08, \eta = 3.8, \\ \mu &= 0.5, \alpha = 0.6, \lambda = 0.08, b = 30 \end{aligned} \tag{3.21}$$

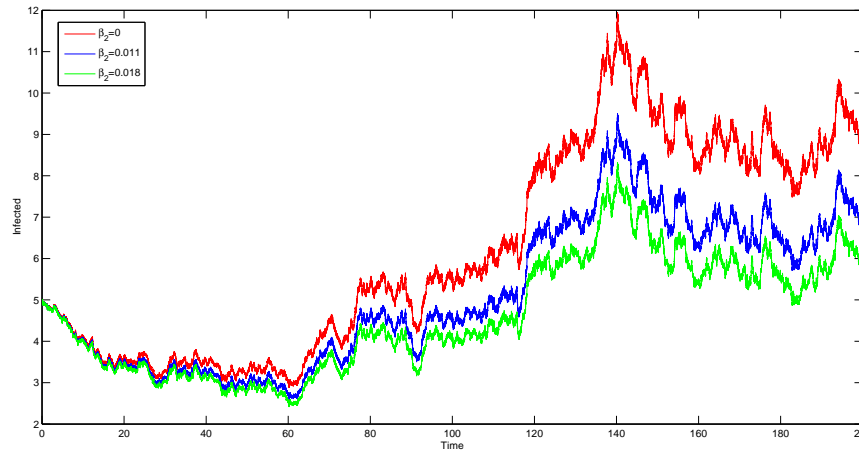


Fig. 3. Trajectories of I_t , with different values of β_2 , for model (2.4). Parameters are taken as (3.20.)

and $\sigma_1 = 0.001, \sigma_2 = 0.01, \sigma_3 = 0.003, \sigma_4 = 0.004$, so that

$$\begin{aligned} \mathcal{R}^0 &= 3.890 > 1, & E^* &= (15.087, 11.229, 0.209), & m_1 S^{*2} &= 139.881, \\ m_2 I^{*2} &= 174.392, & m_3 R^{*2} &= 1.401, & \delta &= 1.224. \end{aligned}$$

The conditions in Theorem 3.2 are satisfied. Accordingly, there is a stationary distribution for model (2.4). This situation is illustrated by Figure 4 and Figure 5, where the red lines represent the kernel density functions of S_t, I_t and R_t . Figure 4 and Figure 5 shows histograms of the approximate stationary distribution of the susceptible, infective and recovery classes. From histograms, we can see that the values of S_t, I_t and R_t are distributed normally around the average values 15.087, 11.229, 0.209 respectively. We see the density function at $t = 400$ (see Figure 4) and $t = 500$ (see Figure 5) based on 5000 stochastic simulation are very close to each other that can be considered as a good estimation of the stationary distribution of system (2.4). Theorem 3.2 gives the possibility that an asymptotically stationary distribution exists for the solution of model (2.4), which in turn implies the stability in a stochastic sense. Furthermore, Theorem 3.2 suggests that if the condition of the theorem is satisfied, then the stochastic model (2.4) oscillates around the endemic equilibrium E^* of the deterministic model (2.1). This indicates the persistence of the disease a.s. under certain conditions.

The results obtained in this study (extinction and stationary distribution) is in the line with the work of [33], where authors consider only multiplicative noise terms in their models. Our method to prove the extinction and persistence of the disease described by the stochastic model is different from that proposed by some authors. In particular [15], authors study the asymptotic behavior of the stochastic model solution around the disease-free equilibrium and the endemic equilibrium of deterministic model to determine whether the disease disappears or persists in the population.

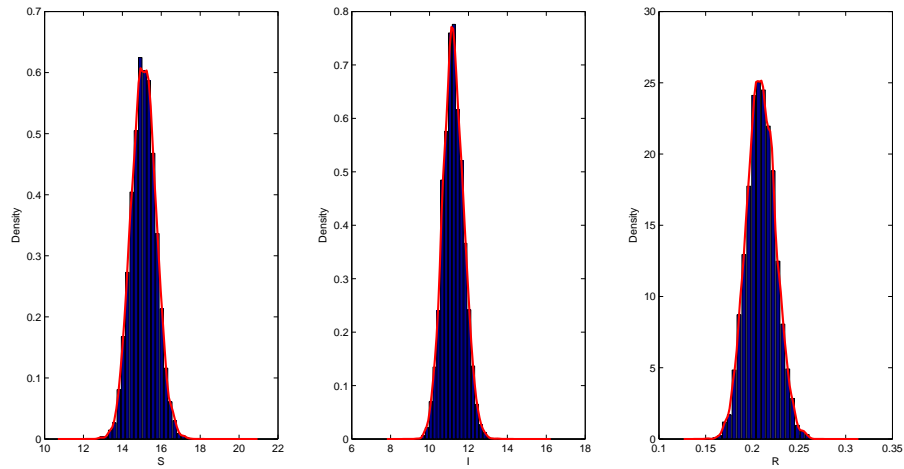


Fig. 4. Histogram and the kernel density function estimations of S_t, I_t and R_t of system (2.4) at time $t=400$. Parameters are taken as (3.21).

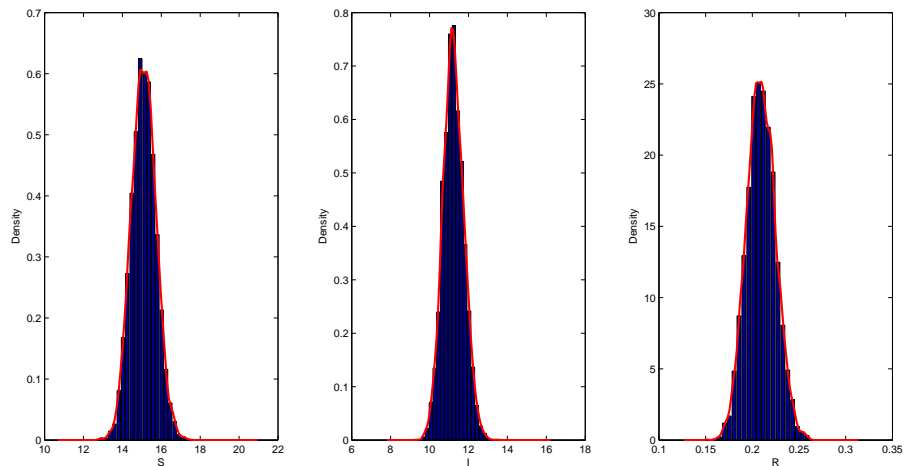


Fig. 5. Histogram and the kernel density function estimations of S_t, I_t and R_t of system (2.4) at time $t=500$. Parameters are taken as (3.21).

4 Conclusion

In this paper, we studied a new stochastic SIRS model that takes into account a reduction of interpersonal contacts as a result of media coverage about the disease. We proposed a stochastic version of the SIRS epidemic model incorporating media coverage by introducing the multiplicative noise terms into the growth equations of susceptible, infective, removed populations and noise introduced in contact rate. Firstly, we have proved the global existence, positivity and boundedness

of solution. Then we investigated the asymptotic behavior of the model. Theorem 3.2 demonstrates that if the noise intensity is sufficiently large so that $\sigma_4^2(2(\mu + \alpha + \lambda) + \sigma_2^2) > (\beta_1 + \beta_2)^2$ then the number of infective individuals I_t tends asymptotically to zero exponentially almost surely (see Figure 1). We also proved that if the intensity of the white noise is sufficiently small and $\mathcal{R}^0 > 1$ then there is a unique stationary distribution to stochastic system (see Theorem 3.3 and Figure 4, Figure 5). Finally, We give an illustration of our analytical results by numerical simulation. The value of our study lies on two aspects. Mathematically, we used stochastic Lyapunov functions to give, sufficient conditions for the extinction and the existence of a stationary distribution using the theory of Has'minskii. Epidemiologically, we find that random fluctuations can suppress disease outbreak, which can provide us with some useful control strategies to regulate disease dynamics. Furthermore, simulations show that the role of media is crucial for reducing incidence. Increasing the media coverage rate reduces the magnitude of the infectious individuals in the population (see Figure 3). We conclude that efforts should be made, by means of massive media coverage, in order to prevent the disease to spread widely in the population: especially for emergent diseases like Chikungunya, Ebola....

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Competing Interests

Authors have declared that no competing interests exist.

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