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DYNAMICAL BEHAVIOR OF A STOCHASTIC SIR EPIDEMIC MODEL WITH GENERAL INCIDENCE FUNCTION AND IMMIGRATION: CASE OF COVID-19

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Abstract. In this study, a novel stochastic model for coronavirus disease 2019 (COVID-19) transmission is formulated with the presence of immigration, vaccination and general incidence function. The environment variability in this work is characterized by Gaussian white noise. We prove the existence, uniqueness and positivity of the solution of the model and investigate the stochastic ultimate boundedness. Sufficient conditions are presented for the extinction of the disease according to the values of the threshold parameter R_0^S that represents the basic reproduction number of our stochastic model. Moreover, we prove that the number of infected individuals is always persistent in the mean. Also, the sensitivity analysis is used to discover parameters that have impact on the threshold parameter R_0^S. Some numerical experiments are also presented to illustrate the theoretical results.

Keywords: stochastic model; COVID-19; immigration; vaccination; white noise; extinction; persistence.

2020 AMS Subject Classification: 60H10, 60H40, 60J65, 92D25, 92D30.

1. INTRODUCTION

In Epidemiology, mathematical modeling is an essential tool in studying and analyzing the spread of infectious diseases and it is considered as an effective way to forecast the outbreak

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of an epidemic. Understanding the transmission of diseases in communities and countries is extremely important to identify factors that are responsible for their existence.

Recently, many authors have proposed and studied different types of deterministic epidemic models. For instance, Semlali et al [1] studied the global stability of ordinary differential equations (ODEs) model with general incidence rate, taking into account the effects of immigration and vaccination. They divided the total population into three classes $S(t)$, $I(t)$ and $R(t)$ that represent susceptible, infected and recovered individuals at time t, respectively. More precisely, the dynamics of the three classes was governed by the following system of ODEs

$$\left\{\begin{array}{ll} \frac{dS}{dt} & = A+b-\mu S-f(S,I)I-\nu S, \\ \frac{dI}{dt} & = c+f(S,I)I-(\mu+\gamma+r)I, \\ \frac{dR}{dt} & = rI+\nu S-\mu R, \end{array}\right. \tag{1}$$

where the susceptible individuals are recruited at a rate A and become infected by effective contact with infected individuals at rate $f(S,I)I$. The natural death rate in all classes is denoted by μ, while γ is the death rate due to COVID-19. The parameter ν is the rate of vaccination and r denotes the recovery rate of the infected individuals. Finally, b and c represent the immigrant to susceptible and the immigrant to infected, respectively.

Most real world problems are not deterministic and our real life is full of randomness and stochasticity and is influenced by environmental fluctuations. May [2] revealed that some main parameters of the epidemic model, such as birth rates, death rates, recruited rates and disease spread rates, are affected by environmental noise to some extent. Therefore, incorporating stochastic effects into the model can bring more advantages and gives us a more realistic way of modeling epidemic models compared to their deterministic corresponding models [2, 3].

There are different possible approaches that result in different effects on epidemic dynamical systems to include random perturbations in the models [4, 5, 6, 7, 8]. Particularly, approaches observed most often, such as parameters disturbance, ambient noise which is proportional to the variables [9, 10].

Brownian motion is the primary choice for simulating random motion and noise in modeling continuous-time systems. This choice is based on their good statistical characteristics. For

example, Brownian motion has finite moments of all orders and there are powerful analytical tools that can solve the Brownian motion problem.

In order to better simulate the impact of environmental noise during disease transmission, we consider a random disturbance depending on the variables of state S, I and R. Thus, the stochastic version of the deterministic system (1) is given by the following system of stochastic differential equations (SDEs)

$$\begin{cases} dS(t) &= [A+b-\mu S(t)-f(S(t),I(t))I(t)-\nu S(t)]dt+\sigma_1 S(t)dB_1(t), \\ dI(t) &= [c+f(S(t),I(t))I(t)-(\mu+\gamma+r)I(t)]dt+\sigma_2 I(t)dB_2(t), \\ dR(t) &= [rI(t)+\nu S(t)-\mu R(t)]dt+\sigma_3 R(t)dB_3(t), \end{cases} \tag{2}$$

where $B_i(t)$ are independent standard Brownian motions with $B_i(0)=0$, defined on a complete probability space $(\Omega,F,\mathbb{P})$ with a filtration $\{F_t\}_{t\geq 0}$ satisfying the usual conditions (i.e., it is increasing and right continuous while F_0 contains all $\mathbb{P}$-null sets) and σ_i denote the intensities of perturbations, i=1, 2, 3.

It is important to note that the model presented by system (2) generalizes several special cases. For instance, we obtain the model in [9, 11] when $b=0$, $c=0$, $\nu=0$ and $f(S,I)=\frac{\beta S}{1+kI}$. Also, we get the model of Jiang et al. [12] when $b=0$, $c=0$, $\nu=0$ and $f(S,I)=\beta S$. Furthermore, the model introduced in [13] is a particular case of system (2), it suffices to take $b=0$, $c=0$, $\nu=0$, $f(S,I)=\beta S$ and $B_1(t)=B_2(t)=B_3(t)$ for all $t\geq 0$.

Obviously, the first two equations of system (2) do not depend on the variable R, model (2) can be rewrite by following system

$$\begin{cases} dS(t) &= [A+b-\mu S(t)-f(S(t),I(t))I(t)-\nu S(t)]dt+\sigma_1 S(t)dB_1(t), \\ dI(t) &= [c+f(S(t),I(t))I(t)-(\mu+\gamma+r)I(t)]dt+\sigma_2 I(t)dB_2(t). \end{cases} \tag{3}$$

Moreover and according to [1], we assume that the general incidence function f is continuously differentiable in the interior of $\mathbb{R}^2_+$ and satisfies the following conditions

(H_1) $f(0,I)=0$, for all $I\geq 0$.

(H_2) $\frac{\partial f}{\partial S}(S,I)>0$, for all $S>0$ and $I\geq 0$.

(H_3) $\frac{\partial f}{\partial I}(S,I)\leq 0$, for all $S\geq 0$ and $I\geq 0$.

Furthermore, we assume that

(H_4) $f(S,I) \leq \delta S$, for some real constant $\delta > 0$ and all $S > 0$.

The rest of the present paper is organized as follows. In the next section, the well posedness of the stochastic model (3) is proved by showing the existence, uniqueness and positivity of the solution. Also, the stochastic ultimate boundedness of the solution is established. In Section 3, we show the extinction of the disease in terms of the threshold parameter R_0^S. In Section 4, the sensitivity analysis is used to discover parameters that have impact on the threshold value R_0^S. In Section 5, the persistence in the mean is investigated. We give some numerical simulations to illustrate our main results in Section 6. The paper ends with a brief discussion and conclusion.

2. Existence, Uniqueness of the Global Positive Solution and Stochastic Ultimate Boundedness

Since the solution of the stochastic model (3) has biological significance, it should be positive. Furthermore, to study the dynamical behavior of the system (3), 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 model (3) is global positive and bounded.

Theorem 2.1. *For any given initial condition $(S(0),I(0)) \in \mathbb{R}^2_+$, there is a unique positive solution $(S(t),I(t))$ of model (3) for all $t \geq 0$ and the solution will remain in $\mathbb{R}^2_+$ with probability one. That is $(S(t),I(t)) \in \mathbb{R}^2_+$ for all $t \geq 0$ almost surly (a.s.).*

Proof. Since the coefficients of system (3) are locally lipschitz continuous, then from [14], for any initial condition $(S(0),I(0)) \in \mathbb{R}^2_+$, there is a unique local solution $(S(t),I(t))$ on $t \in [0,t_e)$, where t_e is the explosion time. To show that this solution is global, we need to prove $t_e = +\infty$ almost surely.

We consider the following stopping time t^* by

$$t^* = inf\{t \in [0,t_e) : S(t) \leq 0 \text{ or } I(t) \leq 0\}, \text{ with } \inf\emptyset = +\infty.$$

It's clear that $t^* \leq t_e$, so if we prove that $t^* = +\infty$ (a.s.), then $t_e = +\infty$ (a.s.) which means that $(S(t),I(t))$ will remain in $\mathbb{R}^2_+$ (a.s) for all $t \geq 0$.

Assume that $t^* < +\infty$, then there exists a $T > 0$ such that $\mathbb{P}(t^* < T) > 0$.

Define the C^2-function V: $\mathbb{R}^2_+ \to \mathbb{R}$ by

$$V(S,I) = lnS + lnI.$$

By Itô's formula, for all $t \in [0,t^*)$ and almost all $\omega \in \{t^* < T\}$ we obtain

$$\begin{aligned} lnS(t) + lnI(t) - lnS(0) - lnI(0) &= \int_0^t \left[\frac{A+b}{S(s)} - (\mu+\nu) - \frac{f(S(s),I(s))I(s)}{S(s)} \right. \\ &\quad \left. -\frac{1}{2}\sigma_1^2\right] ds + \int_0^t \left[\frac{c}{I(s)} + f(S(s),I(s)) \right. \\ &\quad \left. -(\mu+\gamma+r) - \frac{1}{2}\sigma_2^2\right] ds + \sigma_1 B_1(t) + \sigma_2 B_2(t) \\ &\geq \int_0^t \left[-(2\mu+\nu+\gamma+r) - \frac{f(S(s),I(s))I(s)}{S(s)} \right. \\ &\quad \left. -\frac{1}{2}(\sigma_1^2+\sigma_2^2)\right] ds + \sigma_1 B_1(t) + \sigma_2 B_2(t). \end{aligned}$$

According to (H_4), we have

$$\begin{aligned} lnS(t) + lnI(t) - lnS(0) - lnI(0) &\geq \int_0^t \big[-(2\mu+\nu+\gamma+r) - \delta I(s) \\ &\quad -\frac{1}{2}(\sigma_1^2+\sigma_2^2)\big] ds + \sigma_1 B_1(t) + \sigma_2 B_2(t). \end{aligned}$$

From the definition of t^*, it follows that the solution of system (3) is positive on $[0,t^*)$ for almost all $\omega \in \{t^* < T\}$ and $S(t^*) = 0$ or $I(t^*) = 0$.

Therefore, $\lim\limits_{t\to t^*}(lnS(t) + lnI(t)) = -\infty$. Letting $t \mapsto t^*$ in the inequality above, we get

$$-\infty \geq -[2\mu+\nu+\gamma+r+\frac{1}{2}(\sigma_1^2+\sigma_2^2)]t^* - \delta\int_0^{t^*} I(s)ds + \sigma_1 B_1(t^*) + \sigma_2 B_2(t^*) > -\infty,$$

that contradicts our assumption. Thus, $t^* = t_e = +\infty$ (a.s.). This completes the proof. ■

Theorem 2.1 shows that the solution of model (3) will remain in $\mathbb{R}^2_+$ and one need know how the solution varies in $\mathbb{R}^2_+$ in more details. In population dynamic systems, the properties of positivity and non-explosion are important but are often not sufficient. It is necessary to discuss other properties of the solution of system (3). From a biological point of view, the property of stochastically ultimate boundedness is more desirable than the nonexplosion property.

First, we give the definition of the stochastic ultimate boundedness of the solution, then an essential theorem follows directly.

Definition 1. *The solution $X(t) = (S(t),I(t))$ of system (3) is said to be stochastically ultimately bounded if, for any $\varepsilon \in (0,1)$, there is a positive constant α such that for any initial value*

$X(0) \in \mathbb{R}^2_+$, the solution $X(t)$ of model (3) verifies the property

$$\limsup_{t\to\infty} \mathbb{P}(\|X\| > \alpha) \leq \varepsilon.$$

Theorem 2.2. *For any initial value $X(0) = (S(0), I(0)) \in \mathbb{R}^2_+$, system (3) is stochastically ultimately bounded.*

Proof. Let $m_0 > 0$ be sufficiently large such that every component of $X(0)$ is contained within the interval $(\frac{1}{m_0}, m_0)$. For each integer $m \geq m_0$, define the stopping time

$$t_m = inf\Big\{t \geq 0 : S(t) \notin (\tfrac{1}{m}, m) \text{ or } I(t) \notin (\tfrac{1}{m}, m)\Big\},$$

which by Theorem 2.1 has the properties that $t_m \mapsto +\infty$ almost surely as $m \mapsto +\infty$. We have

$$\begin{aligned} d(S(t)+I(t)) &= [A+b+c-(\mu+\nu)S(t)-(\mu+\gamma+r)I(t)]dt \\ &\quad +\sigma_1 S(t)dB_1(t)+\sigma_2 I(t)dB_2(t) \\ &= [A+b+c-\mu(S(t)+I(t))-\nu S(t)-(\gamma+r)I(t)]dt \\ &\quad +\sigma_1 S(t)dB_1(t)+\sigma_2 I(t)dB_2(t). \end{aligned}$$

On the other hand, by applying Itô's formula to $e^{\mu t}(S(t)+I(t))$ we get

$$\begin{aligned} d\Big[e^{\mu t}(S(t)+I(t))\Big] &= e^{\mu t}\Big[\mu\big(S(t)+I(t)\big)dt + d(S(t)+I(t))\Big] \\ &= e^{\mu t}\Big[\Big(A+b+c-\nu S(t)-(\gamma+r)I(t)\Big)dt+\sigma_1 S(t)dB_1(t) \\ &\quad +\sigma_2 I(t)dB_2(t)\Big] \\ &= \Big[(A+b+c)e^{\mu t}-\nu S(t)e^{\mu t}-(\gamma+r)I(t)e^{\mu t}\Big]dt+e^{\mu t}\sigma_1 S(t)dB_1(t) \\ &\quad +e^{\mu t}\sigma_2 I(t)dB_2(t) \\ &\leq \Big((A+b+c)e^{\mu t}\Big)dt+e^{\mu t}\sigma_1 S(t)dB_1(t)+e^{\mu t}\sigma_2 I(t)dB_2(t). \end{aligned}$$

By integrating this inequality and taking expectations on both sides, we obtain

$$\begin{aligned} \mathbb{E}\Big[e^{\mu(t\wedge t_m)}\Big(S(t\wedge t_m)+I(t\wedge t_m)\Big)-(S(0)+I(0))\Big] &\leq \mathbb{E}\Big[\int_0^{t\wedge t_m}\big(A+b+c\big)e^{\mu s}ds\Big] \\ &\leq \frac{A+b+c}{\mu}\big(e^{\mu t}-1\big). \end{aligned}$$

Let $m \to +\infty$, then

$$e^{\mu t}\mathbb{E}\big[(S(t)+I(t))\big]-(S(0)+I(0)) \leq \tfrac{A+b+c}{\mu}\big(e^{\mu t}-1\big).$$

Therefore,

$$\mathbb{E}\big[(S(t)+I(t))\big] \leq (S(0)+I(0))e^{-\mu t} + \tfrac{A+b+c}{\mu}\big(1-e^{-\mu t}\big).$$

Consequently,

$$\limsup_{t\to\infty} \mathbb{E}\big[(S(t)+I(t))\big] \leq \tfrac{A+b+c}{\mu}.$$

Thus,

$$\limsup_{t\to\infty} \mathbb{E}\|X\| \leq \tfrac{A+b+c}{\mu}, \quad \text{where} \quad \|X\| = \sqrt{S^2+I^2} \leq S+I.$$

Then for any given $0<\varepsilon<1$, let $\alpha = \frac{A+b+c}{\mu\varepsilon}$. By virtue of Markov's inequality, we get

$$\limsup_{t\to\infty} \mathbb{P}(\|X\| > \alpha) \leq \limsup_{t\to\infty} \tfrac{\mathbb{E}\|X\|}{\alpha} \leq \tfrac{A+b+c}{\mu} \times \tfrac{1}{\alpha} = \varepsilon.$$

This completes the proof. ■

Consider the feasible region for the corresponding deterministic system of the stochastic model (3), see [1].

$$\Gamma = \Big\{(S,I) \in \mathbb{R}^2_+ : S+I \leq \tfrac{A+b+c}{\mu}\Big\},$$

and define the set $\Gamma^* \subset \Gamma$ by

$$\Gamma^* = \Big\{(S,I) \in \mathbb{R}^2_+ : S+I \leq \frac{A+b}{\mu+\nu}\Big\}. \tag{4}$$

In the following, we will prove that Γ^* is almost surely positively invariant with respect to the stochastic model (3).

Theorem 2.3. *The region Γ^* is almost surely positive invariant of the stochastic model.*

Proof. Let $(S(0),I(0)) \in \Gamma^*$ and $k_0 > 0$ be sufficiently large such that each component of $(S(0),I(0))$ is contained within the interval $(\frac{1}{k_0}, \frac{A+b}{\mu+\nu}]$. Define, for each integer $k \geq k_0$, the stopping times

$$\tau_k = inf\Big\{t>0 : (S(t),I(t)) \in \Gamma^* \; and \; (S(t),I(t)) \notin \Big(\tfrac{1}{k}, \tfrac{A+b}{\mu+\nu}\Big]^2\Big\}$$

and

$$\tau = inf\big\{t>0 : (S(t),I(t)) \notin \Gamma^*\big\}.$$

It suffices to prove that $\mathbb{P}(\tau = +\infty) = 1$, that is, $\mathbb{P}(\tau < t) = 0$ for all $t > 0$.

Obviously, $(\tau < t) \subset (\tau_k < t)$, then $\mathbb{P}(\tau < t) \leq \mathbb{P}(\tau_k < t)$. So, we only need to show that $\limsup_{k\to\infty} \mathbb{P}(\tau_k < t) = 0$.

For this, we consider the C^2-function $\mathscr{U} : \mathbb{R}_+^2 \to \mathbb{R}_+$ defined by

$$\mathscr{U}(S,I) = \frac{1}{S} + \frac{1}{I}.$$

For all $t \geq 0$ and $0 \leq s \leq t \wedge \tau_k$, using Itô's formula, we obtain

$$d\mathscr{U}(S(s),I(s)) = L\mathscr{U}(S(s),I(s))ds - \frac{\sigma_1}{S(s)}dB_1(s) - \frac{\sigma_2}{I(s)}dB_2(s),$$

where

$$\begin{aligned} L\mathscr{U}(S(s),I(s)) &= \left[-\frac{A+b}{S^2(s)} + \frac{\mu+\nu}{S(s)} + \frac{f(S(s),I(s))I(s)}{S^2(s)} + \frac{\sigma_1^2}{S(s)}\right] \\ &\quad + \left[-\frac{c}{I^2(s)} - \frac{f(S(s),I(s))}{I(s)} + \frac{\mu+\gamma+r}{I(s)} + \frac{\sigma_2^2}{I(s)}\right]. \end{aligned}$$

Then

$$\begin{aligned} d\mathscr{U}(S(s),I(s)) &\leq \left(\mu+\nu+\frac{f(S(s),I(s))I(s)}{S(s)} + \sigma_1^2\right)\frac{ds}{S(s)} + \left(\mu+\gamma+r+\sigma_2^2\right)\frac{ds}{I(s)} \\ &\quad - \frac{\sigma_1}{S(s)}dB_1(s) - \frac{\sigma_2}{I(s)}dB_2(s). \end{aligned}$$

According to (H_4) and (4), we get

$$\begin{aligned} d\mathscr{U}(S(s),I(s)) &\leq \left(\mu+\nu+\frac{\delta(A+b)}{\mu+\nu} + \sigma_1^2\right)\frac{ds}{S(s)} + \left(\mu+\gamma+r+\sigma_2^2\right)\frac{ds}{I(s)} \\ &\quad - \frac{\sigma_1}{S(s)}dB_1(s) - \frac{\sigma_2}{I(s)}dB_2(s). \end{aligned}$$

Therefore,

$$d\mathscr{U}(S(s),I(s)) \leq N\mathscr{U}(S(s),I(s))ds - \frac{\sigma_1}{S(s)}dB_1(s) - \frac{\sigma_2}{I(s)}dB_2(s), \tag{5}$$

where

$$N = max\left\{\mu+\nu+\frac{\delta(A+b)}{\mu+\nu} + \sigma_1^2, \mu+\gamma+r+\sigma_2^2\right\}.$$

By integrating, taking the expectation on both sides of (5) and applying Fubini's theorem, we obtain

$$\mathbb{E}[\mathscr{U}(S(s),I(s))] \leq \mathscr{U}(S(0),I(0)) + N\int_0^s \mathbb{E}[\mathscr{U}(S(u),I(u))]du.$$

From Gronwall Lemma, we have for all $0 \leq s \leq t \wedge \tau_k$,

$$\mathbb{E}[\mathscr{U}(S(s),I(s))] \leq \mathscr{U}(S(0),I(0))e^{Ns}.$$

Hence,

$$\mathbb{E}[\mathscr{U}(S(t\wedge\tau_k),I(t\wedge\tau_k))] \leq \mathscr{U}(S(0),I(0))e^{N(t\wedge\tau_k)} \leq \mathscr{U}(S(0),I(0))e^{Nt}, \forall t \geq 0. \tag{6}$$

Since $\mathscr{U}(S(t\wedge\tau_k),I(t\wedge\tau_k))>0$ and some component of $(S(\tau_k),I(\tau_k))$ is less than or equal to $\frac{1}{k}$, then $\mathscr{U}(S(\tau_k),I(\tau_k))\geq k$, which implies that

$$\mathbb{E}[\mathscr{U}(S(t\wedge\tau_k),I(t\wedge\tau_k))]\geq\mathbb{E}[\mathscr{U}(S(\tau_k),I(\tau_k))\chi_{\{\tau_k<t\}}]\geq k\mathbb{P}(\tau_k<t), \tag{7}$$

where $\chi_{\{\tau_k<t\}}$ is the indicator function of $\{\tau_k<t\}$.

By (6) and (7), we get that for all $t\geq 0$

$$\mathbb{P}(\tau_k<t)\leq\frac{\mathscr{U}(S(0),I(0))e^{Nt}}{k}.$$

Thus,
$$\limsup_{k\to\infty}\mathbb{P}(\tau_k<t)=0.$$

This completes the proof. ■

3. EXTINCTION OF DISEASE

Among the main concerns of epidemiology is how to regulate the dynamics of the disease in order to eradicate it in the long term. For this reason, we investigate the conditions for the extinction of the disease.

Our stochastic SIR epidemic model describes the dynamics of a communicable disease into a population with positive flow of infective c. In this case, the infection cannot be eliminated from the population and persists in the mean. That means, as people migrate, the disease will persist as long as there are undiagnosed infections. $c=0$ will be an ideal case before migration is controlled. In the following, we will show by Theorem 3.1 the conditions for the extinction of the disease.

Also, model (3) can be rewrite by following system

$$\begin{cases} dS(t) &= [A+b-\mu S(t)-f(S(t),I(t))I(t)-\nu S(t)]dt+\sigma_1 S(t)dB_1(t),\\ dI(t) &= [f(S(t),I(t))I(t)-(\mu+\gamma+r)I(t)]dt+\sigma_2 I(t)dB_2(t). \end{cases} \tag{8}$$

For the corresponding deterministic system of (8), we can use the results presented by Semlali et al. [1]. It is easy to get the basic reproduction number of disease that is given as follows

$$R_0=\frac{f(\frac{A+b}{\mu+\nu},0)}{\mu+\gamma+r}. \tag{9}$$

Similarly, we define the following threshold of our stochastic SIR epidemic model (8) by

$$R_0^S=R_0-\frac{\sigma_2^2}{2(\mu+\gamma+r)}. \tag{10}$$

Remark 1. *In absence of noise, we have* $R_0^S = R_0$.

Theorem 3.1. *Let* $(S(t),I(t))$ *be the solution of the system (8) with any initial value* $(S(0),I(0)) \in \Gamma^*$. *Then*

$$\limsup_{t\to\infty} \frac{lnI(t)}{t} \leq (\mu+\gamma+r)(R_0^S-1) \ \ (a.s.).$$

Moreover, if $R_0^S < 1$, *then* $\lim_{t\to\infty}\langle I\rangle_t = 0$ *(a.s.) and* $\lim_{t\to\infty}\langle S\rangle_t = \frac{A+b}{\mu+\nu}$ *(a.s.). In other words, the disease dies out with probability one.*

Proof. Applying Itô's formula to $S(t)+I(t)$, we obtain

$$\begin{aligned} d(S(t)+I(t)) &= [A+b-(\mu+\nu)S(t)-(\mu+\gamma+r)I(t)]dt \\ &\quad +\sigma_1 S(t)dB_1(t)+\sigma_2 I(t)dB_2(t). \end{aligned}$$

Then

$$\begin{aligned} \frac{S(t)+I(t)}{t}-\frac{S(0)+I(0)}{t} &= A+b-(\mu+\nu)\langle S\rangle_t-(\mu+\gamma+r)\langle I\rangle_t \\ &\quad +\frac{\sigma_1}{t}\int_0^t S(s)dB_1(s)+\frac{\sigma_2}{t}\int_0^t I(s)dB_2(s). \end{aligned}$$

Therefore,

$$\begin{aligned} (\mu+\nu)\langle S\rangle_t &= A+b-(\mu+\gamma+r)\langle I\rangle_t-\frac{S(t)+I(t)}{t}+\frac{S(0)+I(0)}{t} \\ &\quad +\frac{\sigma_1}{t}\int_0^t S(s)dB_1(s)+\frac{\sigma_2}{t}\int_0^t I(s)dB_2(s). \end{aligned}$$

Thus,

$$\langle S\rangle_t = \frac{A+b}{\mu+\nu}-\frac{\mu+\gamma+r}{\mu+\nu}\langle I\rangle_t - G(t), \tag{11}$$

where

$$G(t) = \frac{S(t)+I(t)}{(\mu+\nu)t}-\frac{S(0)+I(0)}{(\mu+\nu)t}-\frac{\sigma_1}{(\mu+\nu)t}\int_0^t S(s)dB_1(s)-\frac{\sigma_2}{(\mu+\nu)t}\int_0^t I(s)dB_2(s).$$

Since $(S(t),I(t)) \in \Gamma^*$, we have

$$\lim_{t\to\infty} G(t) = 0 \ (a.s.). \tag{12}$$

Now, we apply Itô's formula to $lnI(t)$, we get

$$dlnI(t) = [f(S(t),I(t))-(\mu+\gamma+r+\tfrac{1}{2}\sigma_2^2)]dt+\sigma_2 dB_2(t).$$

Hence,

$$\frac{lnI(t)}{t} = f(S(t),I(t)) - (\mu+\gamma+r+\frac{1}{2}\sigma_2^2) + \frac{\sigma_2 B_2(t)}{t} + \frac{lnI(0)}{t}.$$

Since $S \leq \frac{A+b}{\mu+\nu}$ and according to (H_2) and (H_3), we get

$$\frac{lnI(t)}{t} \leq f\left(\frac{A+b}{\mu+\nu},0\right) - (\mu+\gamma+r+\frac{1}{2}\sigma_2^2) + \frac{\sigma_2 B_2(t)}{t} + \frac{lnI(0)}{t}.$$

Consequently,

$$\frac{lnI(t)}{t} \leq (\mu+\gamma+r)(R_0 - \frac{\sigma_2^2}{2(\mu+\gamma+r)} - 1) + \frac{\sigma_2 B_2(t)}{t} + \frac{lnI(0)}{t}.$$

Thus,

$$\frac{lnI(t)}{t} \leq (\mu+\gamma+r)(R_0^S - 1) + \frac{\sigma_2 B_2(t)}{t} + \frac{lnI(0)}{t}.$$

By the large number theorem for martingales, we get

$$\lim_{t\to\infty} \frac{B_2(t)}{t} = 0 \ (a.s.). \tag{13}$$

Since $R_0^S < 1$, then

$$\limsup_{t\to\infty} \frac{lnI(t)}{t} \leq (\mu+\gamma+r)(R_0^S - 1) < 0 \ (a.s.). \tag{14}$$

This implies

$$\lim_{t\to\infty} I(t) = 0 \ (a.s.). \tag{15}$$

From (11), we have

$$\lim_{t\to\infty} \langle S\rangle_t = \frac{A+b}{\mu+\nu} - \frac{\mu+\gamma+r}{\mu+\nu} \lim_{t\to\infty} \langle I\rangle_t - \lim_{t\to\infty} G(t).$$

Taking into account (12) and (15), we get

$$\lim_{t\to\infty} \langle S\rangle_t = \frac{A+b}{\mu+\nu} \ (a.s.). \tag{16}$$

The proof is therefore complete. ■

4. SENSITIVITY ANALYSIS

As in the previous section, we assume that there is no flow of infected immigrants, this implies that $c = 0$. In the following, we will perform the sensitivity analysis.

The sensitivity analysis is essential to determine the best way to reduce the effect of disease. It indicates the influence and the impact of each parameter on the disease and discover parameters that have a high impact on the stochastic reproduction number R_0^S.

The sensitivity of a variable with respect to model parameters is usually measured by sensitivity

index. When the variable is a differentiable function of the parameter, the sensitivity index can be defined using partial derivatives.

Definition 2. *The normalized forward sensitivity index of R_0^S that depends differentiably on a parameter p is defined by*

$$\rho_p^{R_0^S} = \frac{\partial R_0^S}{\partial p}\frac{p}{R_0^S}. \tag{17}$$

We perform the sensitivity analysis using (17) with parameters A, b, σ_2, ν, γ, r and μ, we get

$$\rho_A^{R_0^S} = \frac{A}{R_0^S}\frac{\partial R_0^S}{\partial A} = \frac{2A\frac{\partial f}{\partial S}(\frac{A+b}{\mu+\nu},0)}{(\mu+\nu)\left(2f(\frac{A+b}{\mu+\nu},0)-\sigma_2^2\right)} > 0,$$

$$\rho_b^{R_0^S} = \frac{b}{R_0^S}\frac{\partial R_0^S}{\partial b} = \frac{2b\frac{\partial f}{\partial S}(\frac{A+b}{\mu+\nu},0)}{(\mu+\nu)\left(2f(\frac{A+b}{\mu+\nu},0)-\sigma_2^2\right)} > 0,$$

$$\rho_{\sigma_2}^{R_0^S} = \frac{\sigma_2}{R_0^S}\frac{\partial R_0^S}{\partial \sigma_2} = \frac{-2\sigma_2^2}{2f(\frac{A+b}{\mu+\nu},0)-\sigma_2^2} < 0,$$

$$\rho_\nu^{R_0^S} = \frac{\nu}{R_0^S}\frac{\partial R_0^S}{\partial \nu} = \frac{-2\nu(A+b)\frac{\partial f}{\partial S}(\frac{A+b}{\mu+\nu},0)}{(\mu+\nu)^2\left(2f(\frac{A+b}{\mu+\nu},0)-\sigma_2^2\right)} < 0,$$

$$\rho_\gamma^{R_0^S} = \frac{\gamma}{R_0^S}\frac{\partial R_0^S}{\partial \gamma} = \frac{-\gamma}{\mu+\gamma+r} < 0,$$

$$\rho_r^{R_0^S} = \frac{r}{R_0^S}\frac{\partial R_0^S}{\partial r} = \frac{-r}{\mu+\gamma+r} < 0,$$

$$\rho_\mu^{R_0^S} = \frac{\mu}{R_0^S}\frac{\partial R_0^S}{\partial \mu} = -\mu\left[\frac{1}{\mu+\gamma+r} + \frac{2(A+b)\frac{\partial f}{\partial S}(\frac{A+b}{\mu+\nu},0)}{(\mu+\nu)^2\left(2f(\frac{A+b}{\mu+\nu},0)-\sigma_2^2\right)}\right] < 0.$$

Equations above show that the parameters A and b are proportional to the stochastic reproduction number R_0^S. Consequently, an increase or decrease in these parameters will increase or decrease the stochastic reproduction number R_0^S. However, the parameters σ_2, ν, γ, r, μ are inversely proportional to R_0^S. So, an increase in these parameters will decrease R_0^S, while a decrease in these parameters will increase R_0^S.

5. PERSISTENCE OF DISEASE

In this section, we will investigate the persistence in the mean of the system (3). First we recall the definition of the persistence in the mean, then we give two lemmas that we will use to prove Theorem 5.1.

Definition 3. *The variable I in system (3) is said to be persistent in the mean, if*

$$\liminf_{t\to\infty}\langle I\rangle_t > 0 \ \ (a.s.), \ \ where \ \ \ \langle I\rangle_t = \tfrac{1}{t}\int_0^t I(s)ds.$$

Lemma 1. *Let $(S(t),I(t))$ be the solution of system (3) with any initial value $(S(0),I(0))\in\mathbb{R}^2_+$, we have*

$$\lim_{t\to\infty}\tfrac{S(t)+I(t)}{t} = 0 \quad (a.s.).$$

Moreover, we have

$$\lim_{t\to\infty}\tfrac{S(t)}{t} = 0 \quad (a.s.) \quad and \quad \lim_{t\to\infty}\tfrac{I(t)}{t} = 0 \quad (a.s.).$$

Proof. Let $u(t) = S(t)+I(t)$ and define $w(u) = (1+u)^\theta$, where θ is a positive real constant to be chosen in the following. Using Itô's formula, we get

$$dw(u(t)) = Lw(u(t))dt + \theta(1+u(t))^{\theta-1}[\sigma_1 S(t)dB_1(t) + \sigma_2 I(t)dB_2(t)],$$

where

$$\begin{aligned}
L(w(u)) &= \theta(1+u)^{\theta-1}\big[A+b+c-\mu u-\nu S-(\gamma+r)I\big] \\
&\quad +\frac{\theta(\theta-1)}{2}(1+u)^{\theta-2}(\sigma_1^2S^2+\sigma_2^2I^2) \\
&= \theta(1+u)^{\theta-2}\Big[(1+u)(A+b+c-\mu u-\nu S-(\gamma+r)I) \\
&\quad +\frac{(\theta-1)}{2}(\sigma_1^2S^2+\sigma_2^2I^2)\Big] \\
&\le \theta(1+u)^{\theta-2}\Big[(1+u)(A+b+c-\mu u)+\frac{(\theta-1)}{2}(\sigma_1^2S^2+\sigma_2^2I^2)\Big] \\
&\le \theta(1+u)^{\theta-2}\Big[(1+u)(A+b+c-\mu u)+(\frac{\theta-1}{2}\vee 0)(\sigma_1^2\vee\sigma_2^2)u^2\Big] \\
&= \theta(1+u)^{\theta-2}\Big[-\big[\mu-(\frac{\theta-1}{2}\vee 0)(\sigma_1^2\vee\sigma_2^2)\big]u^2+(A+b+c-\mu)u \\
&\quad +A+b+c\Big].
\end{aligned}$$

We choose $\theta > 0$ such that

$$\mu - (\tfrac{\theta-1}{2} \vee 0)(\sigma_1^2 \vee \sigma_2^2) := \lambda > 0.$$

Then

$$Lw(u) \leq \theta(1+u)^{\theta-2}[-\lambda u^2 + (A+b+c-\mu)u + A + b + c].$$

Therefore,

$$\begin{aligned} dw(u(t)) &\leq \theta(1+u(t))^{\theta-2}\big[-\lambda u^2(t) + (A+b+c-\mu)u(t) + A + b + c\big]dt \\ &\quad + \theta(1+u(t))^{\theta-1}\big[\sigma_1 S(t)dB_1(t) + \sigma_2 I(t)dB_2(t)\big]. \end{aligned}$$

For $0 < k < \theta\lambda$, we get

$$d[e^{kt}w(u(t))] = L[e^{kt}w(u(t))]dt + \theta e^{kt}(1+u(t))^{\theta-1}[\sigma_1 S(t)dB_1(t) + \sigma_2 I(t)dB_2].$$

Thus,

$$\mathbb{E}[e^{kt}w(u(t))] = w(u(0)) + \mathbb{E}\left[\int_0^t L(e^{ks}w(u(s)))ds\right], \tag{18}$$

where

$$\begin{aligned} L[e^{kt}w(u(t))] &= ke^{kt}w(u(t)) + e^{kt}L(w(u(t))) \\ &\leq \theta e^{kt}(1+u(t))^{\theta-2}\left[\frac{k}{\theta}(1+u(t))^2 - \lambda u^2(t) + (A+b+c-\mu)u(t)\right. \\ &\quad \left. + A + b + c\right] \\ &= \theta e^{kt}(1+u(t))^{\theta-2}\left[-\left(\lambda - \frac{k}{\theta}\right)u^2(t) + \left(A+b+c-\mu+\frac{2k}{\theta}\right)u(t)\right. \\ &\quad \left. + A + b + c + \frac{k}{\theta}\right] \\ &\leq \theta e^{kt}M, \end{aligned}$$

where

$$M := \sup_{u\in\mathbb{R}_+}\left\{(1+u)^{\theta-2}\Big[-\left(\lambda - \tfrac{k}{\theta}\right)u^2 + \left(A+b+c-\mu+\tfrac{2k}{\theta}\right)u + A + b + c + \tfrac{k}{\theta}\Big]\right\}.$$

Therefore, from (18) we have

$$\mathbb{E}[e^{kt}(1+u(t))^{\theta}] \leq (1+u(0))^{\theta} + \tfrac{\theta M}{k}e^{kt}.$$

Hence,

$$\limsup_{t\to\infty}\mathbb{E}[(1+u(t))^{\theta}] \leq \frac{\theta M}{k} := M_0 \quad \text{(a.s.)},$$

which together with the continuity of u implies that there exists a constant $H > 0$ such that

$$\mathbb{E}[(1+u(t))^{\theta}] \leq H, \, for all \, t \geq 0. \tag{19}$$

With (19), we can proceed as in [3] to complete the proof. ■

Lemma 2. *For any initial value $(S(0),I(0)) \in \mathbb{R}^2_+$ the solution $(S(t),I(t))$ of system (3) verifies*

$$\lim_{t\to\infty}\frac{\int_0^t S(s)dB_1(s)}{t} = 0 \ \ (a.s.) \quad and \quad \lim_{t\to\infty}\frac{\int_0^t I(s)dB_2(s)}{t} = 0 \ \ (a.s.).$$

Proof. We proceed as in Lemma 2.2 of [3] ■

Theorem 5.1. *For any initial value $(S(0),I(0)) \in \mathbb{R}^2_+$, the variable I of model (3) is persistent in the mean (a.s.). Moreover, we have*

$$\liminf_{t\to\infty}\langle I\rangle_t \geq \frac{c}{\mu+\gamma+r}.$$

Proof. We have

$$dI = [c + f(S,I)I - (\mu+\gamma+r)I]dt + \sigma_2 I dB_2(t).$$

Then

$$\frac{I(t)-I(0)}{t} = c + \langle f(S,I)I\rangle_t - (\mu+\gamma+r)\langle I\rangle_t + \frac{\sigma_2}{t}\int_0^t I(s)dB_2(s).$$

Therefore,

$$\liminf_{t\to\infty}\frac{I(t)-I(0)}{t} \geq c - (\mu+\gamma+r)\liminf_{t\to\infty}\langle I\rangle_t + \liminf_{t\to\infty}\frac{\sigma_2}{t}\int_0^t I(s)dB_2(s).$$

According to Lemma 1 and Lemma 2, we get

$$0 \geq c - (\mu+\gamma+r)\liminf_{t\to\infty}\langle I\rangle_t.$$

Consequently,

$$\liminf_{t\to\infty}\langle I\rangle_t \geq \frac{c}{\mu+\gamma+r}.$$

This completes the proof. ■

6. NUMERICAL SIMULATIONS

In this section, some numerical simulations are given to illustrate the obtained theoretical results. Throughout the following numerical simulations, we choose $f(S,I) = \frac{\beta S}{1+\alpha_1 S+\alpha_2 I}$, where β is the rate of infection, α_1 and α_2 measuring the effects of saturation. The corresponding

discretization system of model (3) is given as follows

$$\begin{cases} S_{k+1} &= S_k + \left[A + b - \mu S_k - \frac{\beta S_k I_k}{1+\alpha_1 S_k + \alpha_2 I_k} - \nu S_k\right]\Delta t + \sigma_1 S_k \sqrt{\Delta t}\,\xi_k \\ & \quad + \frac{1}{2}\sigma_1^2 S_k(\xi_k^2 - 1)\Delta t, \\ I_{k+1} &= I_k + \left[c + \frac{\beta S_k I_k}{1+\alpha_1 S_k + \alpha_2 I_k} - (\mu + \gamma + r) I_k\right]\Delta t + \sigma_2 I_k \sqrt{\Delta t}\,\xi_k \\ & \quad + \frac{1}{2}\sigma_2^2 I_k(\xi_k^2 - 1)\Delta t, \end{cases}$$

where ξ_k (k = 1,2, ...) are independent Gaussian random variables which follow standard normal distribution $N(0,1)$.

Firstly, taking $A = 5$, $b = 1$, $c = 0$, $\beta = 0.01$, $\gamma = 0.01$, $\mu = 0.01$, $\nu = 0.01$, $r = 0.01$, $\alpha_1 = 0.1$, $\alpha_2 = 0.1$, $\sigma_1 = 0.03$ and $\sigma_2 = 0.03$. By computing, we get $R_0 = 3.2258 > 1$ and $R_0^S = 3.2108 > 1$. Figure 1 is an illustration of the trajectories of $S(t)$ and $I(t)$ using the parameters cited before, it supports the theoretical results seen in Section 3.

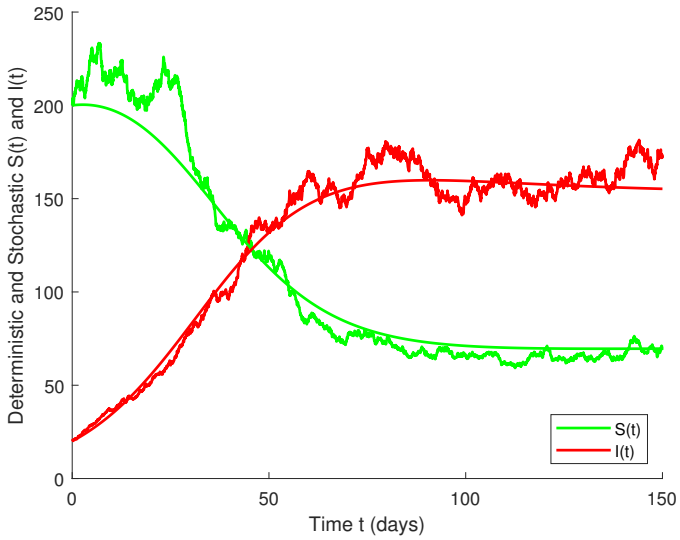

FIGURE 1. Simulations of paths of $S(t)$ and $I(t)$ for the stochastic system and the corresponding deterministic system when $c = 0$.

Secondly, we take $c = 0.4$ and the same other parameters as above. In this case, we have $c > 0$, then according to Theorem 5.1, the disease will persist almost surely and the infection cannot be eliminated from the population as illustrated in Figure 2. The performed simulation

for the disease support our theoretical result where we assumed that the persistence will hold in the population.

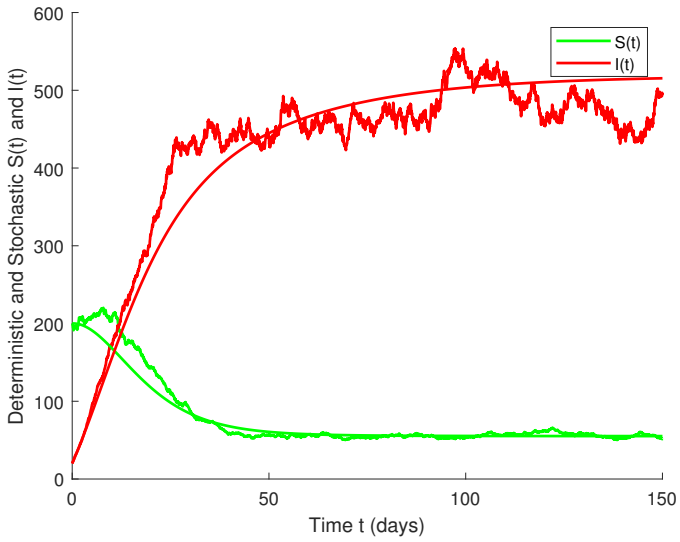

FIGURE 2. Simulations of paths of $S(t)$ and $I(t)$ for the stochastic system and the corresponding deterministic system when $c = 0.4$.

Thirdly, let $A = 5$, $b = 1$, $c = 0$, $\beta = 0.02$, $\gamma = 0.05$, $\mu = 0.1$, $\nu = 0.01$, $r = 0.025$, $\alpha_1 = 0.02$, $\alpha_2 = 0.5$, $\sigma_1 = 0.1$ and $\sigma_2 = 0.05$. In this case, we have $R_0^S = 2.9742 > 1$. Therefore, from Theorem 3.1 it follows that system is persistent in the mean, which means that the disease persist in the population, see Figure 3.

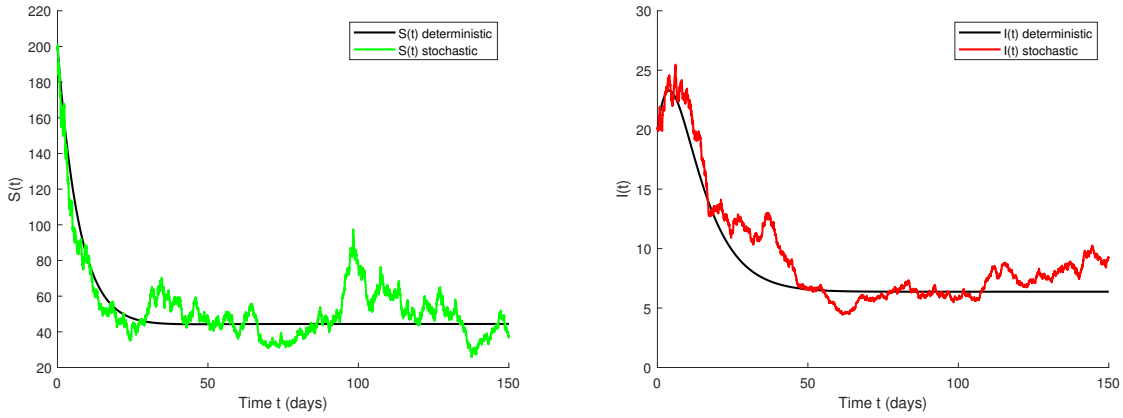

FIGURE 3. Dynamics of system (8) when $c = 0$ and $R_0^S = 2.9742 > 1$.

Fourthly, we take $A = 5$, $b = 1$, $c = 0$, $\beta = 0.01$, $\gamma = 0.05$, $\mu = 0.02$, $\nu = 0.01$, $r = 0.1$, $\alpha_1 = 0.05$, $\alpha_2 = 0.05$, $\sigma_1 = 0.3$ and $\sigma_2 = 0.2$. By computing, we have $R_0^S = 0.9519 < 1$. Hence, according to Theorem 3.1, if $R_0^S < 1$, for the positive solution $(S(t), I(t))$ of the system (8) the disease will extinct almost surely. Figure 4 clearly supports the theoretical result.

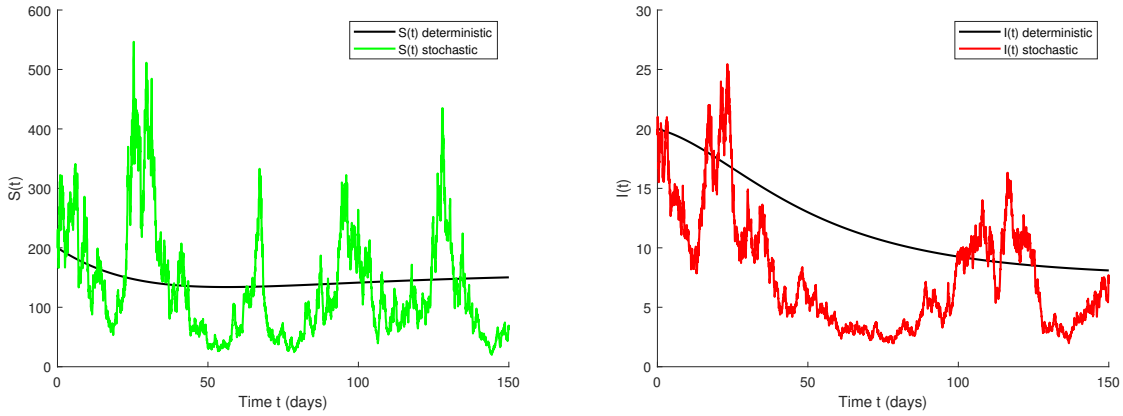

FIGURE 4. Dynamics of system (8) when $c = 0$ and $R_0^S = 0.9519 < 1$.

Next, let $A = 5$, $b = 1$, $c = 0$, $\beta = 0.02$, $\gamma = 0.1$, $\mu = 0.01$, $\nu = 0.05$, $r = 0.1$, $\alpha_1 = 0.07$, $\alpha_2 = 0.05$, $\sigma_1 = 0.1$ and $\sigma_2 = 0.5$. By calculation, we have $R_0 = 1.1905 > 1$ and $R_0^S = 0.5952 < 1$. Therefore, we can deduce that the disease dies out when σ_2 is sufficiently large as illustrated in Figure 5.

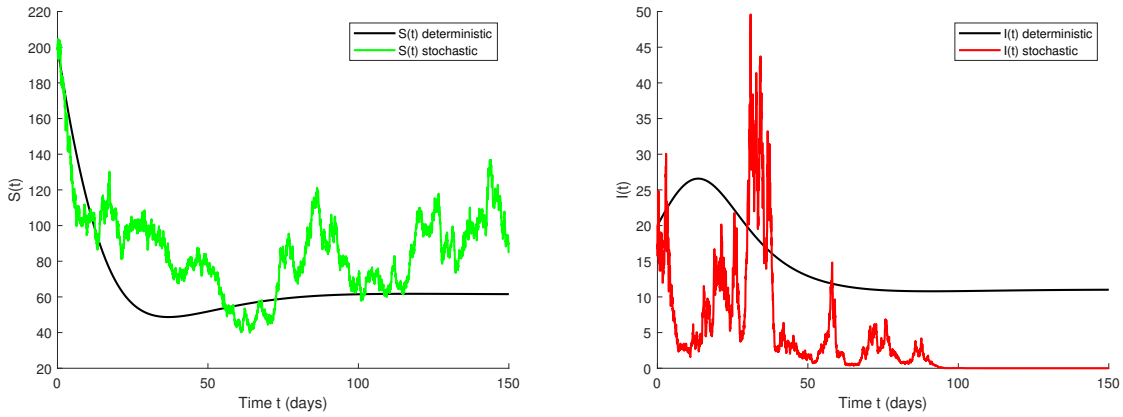

FIGURE 5. Dynamics of system (8) when $c = 0$, $R_0 = 1.1905 > 1$ and $R_0^S = 0.5952 < 1$.

At last, we take $A = 5$, $b = 1$, $c = 0$, $\beta = 0.02$, $\gamma = 0.1$, $\mu = 0.1$, $\nu = 0.02$, $r = 0.1$, $\alpha_1 = 0.01$, $\alpha_2 = 0.5$. By computing, we have $R_0 = 2.2222 > 1$. From the explicit expression of R_0^S in (10), we see that R_0^S is a decreasing function of the second intensity of perturbations σ_2, which is demonstrated in Figure 6.

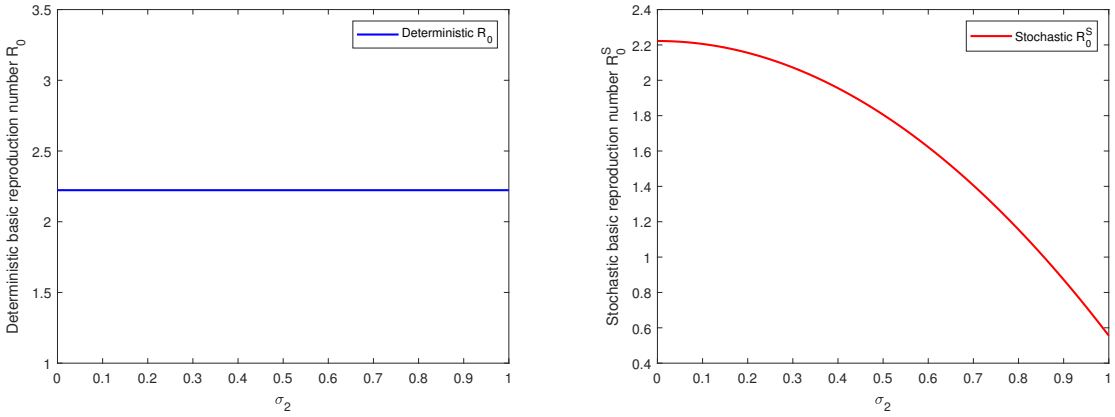

FIGURE 6. Plot of the basic reproduction number as a function of σ_2.

7. DISCUSSION AND CONCLUSION

In this work, we have studied a stochastic SIR epidemic model of COVID-19 transmission in the presence of immigration, vaccination and general incidence function by introducing random perturbations of white noise type directly proportional to the components of the solution. First, we have proved the existence and uniqueness of the global positive solution and investigated the stochastic ultimate boundedness.

In absence of infected immigrants $(c = 0)$, sufficient conditions have been presented for the extinction of the disease according to the values of R_0^S which is smaller than the basic reproduction number R_0 of the corresponding deterministic system which shows that the stochastic approach is more realistic than the deterministic approach. In other words, white noises can change the behavior of the model and force the extinction of the disease.

When $c > 0$, we have shown that the infection cannot be eliminated from the population and persists in the mean. i.e, as people migrate, the disease persists as long as there are undiagnosed infections. In this case, it is impossible to have a disease-free equilibrium but the model has a unique endemic steady state.

On the other hand, we have performed the sensitivity analysis with parameters A, b, σ_2, ν, γ, r and μ and we have indicated the influence and the impact of each parameter on the disease and discovered parameters that have a high impact on the stochastic reproduction number R_0^S.

It is known that COVID-19 can also spread indirectly through contact with an environment contaminated by viral particles of SARS-COV-2. Therefore, taking into account environmental contamination as in [15] and studying the memory effect on the dynamics of COVID-19 by means of the Hattaf fractional operators introduced in [16, 17], will be main goal of our future works.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

REFERENCES

[1] M. Semlali, K. Hattaf, M.E. El Kettani, Modeling and analysis of the dynamics of COVID-19 transmission in presence of immigration and vaccination, Commun. Math. Biol. Neurosci. 2022 (2022), 6. https://doi.org/10.28919/cmbn/7270.

[2] R. May, Stability and complexity in model ecosystems, Princeton University Press, Princeton, (1973).

[3] Y. Zhao, D. Jiang, The threshold of a stochastic SIS epidemic model with vaccination, Appl. Math. Comput. 243 (2014), 718–727. https://doi.org/10.1016/j.amc.2014.05.124.

[4] M. Mahrouf, J. Adnani, N. Yousfi, Stability analysis of a stochastic delayed SIR epidemic model with general incidence rate, Appl. Anal. 97 (2018), 2113–2121. https://doi.org/10.1080/00036811.2017.1359555.

[5] E. N'zi, M. N'zi, Extinction and stationary distribution of a stochastic SIRS epidemic model incorporating media coverage, J. Adv. Math. Computer Sci. 34 (2019), 1–19. https://doi.org/10.9734/jamcs/2019/v34i3-430205.

[6] R. Rifhat, Z. Teng, C. Wang, Extinction and persistence of a stochastic SIRV epidemic model with nonlinear incidence rate, Adv. Differ. Equ. 2021 (2021), 200. https://doi.org/10.1186/s13662-021-03347-3.

[7] Q. Liu, D. Jiang, Stationary distribution and extinction of a stochastic SIR model with nonlinear perturbation, Appl. Math. Lett. 73 (2017), 8–15. https://doi.org/10.1016/j.aml.2017.04.021.

[8] N.H. Du, N.N. Nhu, Permanence and extinction for the stochastic SIR epidemic model, J. Differ. Equ. 269 (2020), 9619–9652. https://doi.org/10.1016/j.jde.2020.06.049.

[9] Q. Yang, D. Jiang, N. Shi, et al. The ergodicity and extinction of stochastically perturbed SIR and SEIR epidemic models with saturated incidence, J. Math. Anal. Appl. 388 (2012), 248–271. https://doi.org/10.1016/j.jmaa.2011.11.072.

[10] K. Hattaf, M. Mahrouf, J. Adnani, et al. Qualitative analysis of a stochastic epidemic model with specific functional response and temporary immunity, Physica A: Stat. Mech. Appl. 490 (2018), 591–600. https://doi.org/10.1016/j.physa.2017.08.043.

[11] N. Gunasekaran, R. Vadivel, G. Zhai, et al. Finite-time stability analysis and control of stochastic SIR epidemic model: A study of COVID-19, Biomed. Signal Process. Control. 86 (2023), 105123. https://doi.org/10.1016/j.bspc.2023.105123.

[12] D. Jiang, J. Yu, C. Ji, et al. Asymptotic behavior of global positive solution to a stochastic SIR model, Math. Computer Model. 54 (2011), 221–232. https://doi.org/10.1016/j.mcm.2011.02.004.

[13] Y. Lin, D. Jiang, P. Xia, Long-time behavior of a stochastic SIR model, Appl. Math. Comput. 236 (2014), 1–9. https://doi.org/10.1016/j.amc.2014.03.035.

[14] X. Mao, Stochastic differential equations and applications, Horwood, Chichester, (1997).

[15] K. Hattaf, A.A. Mohsen, J. Harraq, et al. Modeling the dynamics of COVID-19 with carrier effect and environmental contamination, Int. J. Model. Simul. Sci. Comput. 12 (2021), 2150048. https://doi.org/10.1142/s1793962321500483.

[16] K. Hattaf, A new mixed fractional derivative with applications in computational biology, Computation. 12 (2024), 7. https://doi.org/10.3390/computation12010007.

[17] K. Hattaf, A new class of generalized fractal and fractal-fractional derivatives with non-singular kernels, Fractal Fract. 7 (2023), 395. https://doi.org/10.3390/fractalfract7050395.