A STOCHASTIC SIRI EPIDEMIC MODEL WITH RELAPSE AND MEDIA COVERAGE

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Abstract. This work is devoted to investigate the existence and uniqueness of a global positive solution for a stochastic epidemic model with relapse and media coverage. We also study the dynamical properties of the solution around both disease-free and endemic equilibria points of the deterministic model. Furthermore, we show the existence of a stationary distribution. Numerical simulations are presented to confirm the theoretical results.

1. Introduction. Infectious disease is a major public health problem in the World. Mathematical models have been widely used in providing quantitative predictions and evaluating interventions. Models in systems biology help to explain and understand many aspects of biological processes occurring at viral or population level. In mathematical epidemiology, the compartmental approach of Kermack and McKendrick (1927) uses the deterministic dynamics to make predictions of infectious diseases in a number of different biological contexts. Deterministic models have the advantage of simplicity, as they rely on the law of large numbers to longitudinally describe the average trends in a collection of molecules or viruses or other populations of interest. However, the dynamics of such collections are inherently stochastic.
phenomena and there are many reasons why one should use stochastic rather than deterministic modeling to capture their dynamics. Hence, the development of stochastic methods in the study of infectious diseases is of great significance to make predictions that can guide public policy.

In this work, we investigate stochastic SIRI models, which consist of systems with three compartments: susceptibles, infectives, and removed, labeled by $S, I$ and $R$. In such models, susceptibles become infectious, then are removed with temporary immunity and then become infectious again. This recurrence of disease is an important feature of some animal and human diseases, for example tuberculosis including human and bovine and herpes [4, 37]. In [32] an epidemic model with relapse, which incorporates bilinear incidence rate and constant total population, was formulated. This system was extended to include nonlinear incidence functions by Moreira and Wang [28]. Blower [4] developed a compartmental model for genital herpes, assuming standard incidence for the disease transmission and constant recruitment rate. A more general SIRI model, formulated as an integro-differential system with the fraction $P(t)$ of recovered individuals remaining in the recovered class, $t$ time units after the recovery, expressed in an abstract form, has been proposed and analyzed by van den Driessche and Zou [33], with certain threshold stability results being obtained by particularizing $P(t)$. See also van den Driessche et al. [34] for an analysis of a related SEIRI model. For other works see [11, 23, 38] and the references therein. A deterministic SIRI disease can be modeled as follows [35]:

$$\dot{S} = \mu - \beta SI - \mu S,$$
$$\dot{I} = \beta SI - (\gamma + \mu)I + \delta R,$$
$$\dot{R} = \gamma I - (\mu + \delta)R.$$

Recently, various mathematical models have been used to investigate the impact of the media coverage. Li and Cui [20], Tchuenche et al. [31], and Sun et al. [30] used deterministic models to investigate the effects of media coverage on the transmission dynamics, by means of the following incidence function

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

where $\beta_1$ is the contact rate before media alert; the term $\frac{\beta_2 I}{m + I}$ measures the effect of reduction of the contact rate when infectious individuals are reported in the media. Because the coverage report cannot prevent disease from spreading completely, we have $\beta_1 \geq \beta_2 > 0$. The half-saturation constant $m > 0$ the contact transmission. The function $\frac{I}{m + I}$ is a continuous bounded function that takes into account disease saturation or psychological effects. The deterministic SIRS epidemic model can be modelled as follows:

$$dS = \left[ \Lambda - \left( \beta_1 - \frac{\beta_2 I}{m + I} \right) SI - \mu S \right] dt,$$
$$dI = \left( \left( \beta_1 - \frac{\beta_2 I}{m + I} \right) SI - (\mu + \gamma)I + \delta R \right) dt,$$
$$dR = [\gamma I - (\mu + \delta)R] dt,$$

where, $\Lambda, \mu, \beta_1, \beta_2, m, \gamma, \delta$ are all positive constants, $S(t)$ is the numbers of the individuals susceptible to the disease, $I(t)$ are the infected members and $R(t)$ represents the members who have recovered from the infection. In this model, the parameters have the following features: $\Lambda$ is the total numbers of the susceptible, $\mu$
represents the natural death rate, \( \gamma \) represents the rate of recovery from infection and \( \delta \) is the rate of temporary immunity. The basic reproduction number \([11]\) for this model is given by

\[
R_0 = \frac{\beta_1 \Lambda}{\mu \left( \mu + \gamma - \frac{\gamma \delta}{\mu + \delta} \right)}.
\]

It is a threshold quantity which determines whether an epidemic occurs or the disease simply dies out. In the few past years, epidemic models with stochastic perturbation have attracted much attention (see e.g. \([12, 13, 18, 22, 40]\)). For stochastic models with white noise incorporating media coverage (see Cai et al. \([7]\)), where the effects of stochastic dynamics of an SIS model incorporating media coverage was investigated. Lui and Zheng \([25]\) investigated the stochastic disease dynamics of an SIS epidemic model on two patches incorporating media coverage. Zhao et al. \([40]\) studied the basic dynamical features of a stochastic SIR epidemic model incorporating media coverage. In the real world, epidemic dynamics is unavoidably perturbed by some kind of environmental noise. To reveal the effects of stochasticity, many authors have introduced randomness into epidemic models \([2, 12, 13, 18, 21, 22, 26, 36, 41]\). Amongst authors who studied SIRI epidemic model, we can cite \([19]\) for an epidemic model perturbed by white noise, \([39]\) for a nonautonomous periodic SIRI model, \([9]\) where authors discussed stability and instability for an epidemic model with relapse, and \([3]\) for a stochastic SIRI model driven by lévy noise. Then to make model \((1.1)\) more reasonable and realistic, we introduce a corresponding stochastic perturbation as follows:

\[
\begin{align*}
\frac{dS}{dt} &= \left[ \Lambda - \left( \beta_1 - \frac{\beta_2 I}{\mu + \delta} \right) SI - \mu S \right] dt + \sigma_1 S dB_1(t), \\
\frac{dI(t)}{dt} &= \left[ \left( \beta_1 - \frac{\beta_2 I}{\mu + \delta} \right) SI - \left( \mu + \gamma \right) I + \delta R \right] dt + \sigma_2 I dB_2(t), \\
\frac{dR(t)}{dt} &= \left[ \gamma I - \left( \mu + \delta \right) R \right] dt + \sigma_3 R dB_3(t),
\end{align*}
\]

(1.2)

where \(B_1(t), B_2(t), B_3(t)\) are independent Brownian motions, and \(\sigma_1, \sigma_2, \sigma_3\) are the intensities of the white noises. Notice that this model appears when we assume that the parameter \(\mu\) is affected by some stochastic perturbation in each equation, in other words, we replace \(\mu\) by \(\mu - \sigma_i \dot{B}_i(t)\) in each equation.

The rest of this paper is organized as follows. In the next section we prove existence and uniqueness of a global positive solution. In Section 3 we study asymptotic behavior around the disease-free equilibrium of the deterministic model and we show that the solution of the stochastic model \((1.2)\) oscillates around the disease-free equilibrium of the deterministic model \((1.1)\) if \(R_0 \leq 1\). In Section 4 we show that when \(R_0 > 1\), the solution of stochastic model \((1.2)\) oscillates around the endemic equilibrium of deterministic model \((1.1)\). In Section 5 we prove that the solution of stochastic model \((1.2)\) has a stationary distribution and is ergodic. In the last section we study the persistence in mean. The presented results are illustrated and confirmed by numerical simulations.

Throughout this paper, we let \((\Omega, \mathcal{F}, \mathcal{P})\) be a complete probability space with a filtration \(\{\mathcal{F}_t\}_{t \geq 0}\) satisfying the usual conditions (i.e., it is increasing and right continuous while \(\mathcal{F}_0\) contains all \(\mathcal{P}\)-null sets). In addition, we let \(B_i(t), (i = 1, 2, 3)\), be defined on the above probability space. A general \(d\)-dimensional stochastic system is given by

\[
\frac{dX(t)}{dt} = F(t, X(t)) dt + G(t, X(t)) dB(t),
\]

(1.3)
where \( F(t, X(t)) \) is a function in \( \mathbb{R}^d \) defined in \([t_0, +\infty[ \times \mathbb{R}^d \), and \( G(t, X(t)) \) is a \( d \times m \) matrix, \( F \) and \( G \) are locally Lipschitz in \( X \) and \( B(t) \) is a \( d \)-dimensional Wiener process. Let \( S_h = \{ x \in \mathbb{R}^d : |x| < h \} \), where \(| | \) denote the euclidean norm. The differential operator \( L \) acts on a function \( V \in C^{1,2}(\mathbb{R}_+ \times S_h; \mathbb{R}_+) \) as follows
\[
LV(t, x) = V_t(t, x) + V_x(t, x)F(t, x) + \frac{1}{2} \text{trace} \left[ G^T(t, x)V_{xx}(t, x)G(x, t) \right].
\]

By Itô’s formula, if \( x(t) \in S_h \),
\[
dV(t, x(t)) = LV(t, x(t))dt + V_x(t, x(t))G(t, x(t))dB(t),
\]
where
\[
V_t = \frac{\partial V}{\partial t}, \quad V_x = \left( \frac{\partial V}{\partial x_1}, \frac{\partial V}{\partial x_2}, \ldots, \frac{\partial V}{\partial x_d} \right), \quad V_{xx} = \left( \frac{\partial^2 V}{\partial x_i \partial x_j} \right)_{2 \times 2}.
\]

2. Existence and uniqueness of the global positive solution. Assume that \( S(t), I(t) \) and \( R(t) \) in the model (1.2) are population densities of the susceptible, infected, and removed at time \( t \) respectively. In this section, using the Lyapunov analysis method ([27]) we shall show that the model (1.2) has a unique local positive solution. Then we show that this solution is global, and obtain the following results

**Theorem 2.1.** For any given initial value \((S(0), I(0), R(0)) \in \mathbb{R}_+^3\), the model (1.2) has a unique global solution \((S(t), I(t), R(t)) \in \mathbb{R}_+^3\) for all \( t \geq 0 \) almost surely.

**Proof.** Since the drift and the diffusion are locally Lipschitz, then for any given initial value \((S(0), I(0), R(0)) \in \mathbb{R}_+^3\), there is a unique local solution \((S(t), I(t), R(t))\) for \( t \in [0, \tau_e) \), where \( \tau_e \) is the explosion time (see [26, 27]). To show that this solution is global, we need to show that \( \tau_e = \infty \) a.s. At first, we prove that \( S(t), I(t), R(t) \) do not explode to infinity in a finite time. Let \( k_0 > 0 \) be sufficiently large so that \( S(0), I(0) \) and \( R(0) \) lie within the interval \([\frac{1}{k_0}, k_0]\). For each integer \( k \geq k_0 \), we define the stopping time:
\[
\tau_k = \inf \left\{ t \in [0, \tau_e) : S(t) \notin \left( \frac{1}{k}, k \right) \text{ or } I(t) \notin \left( \frac{1}{k}, k \right) \text{ or } R(t) \notin \left( \frac{1}{k}, k \right) \right\},
\]
where \( \tau_k \) is increasing as \( k \uparrow \infty \). Set \( \tau_\infty = \lim_{k \to \infty} \tau_k \), whence, \( \tau_\infty \leq \tau_e \) a.s. Showing that \( \tau_\infty = \infty \), means that \( \tau_e = \infty \) and \((S(t), I(t), R(t)) \in \mathbb{R}_+^3\) a.s. If this statement is false, then there exists a pair of constants \( T > 0 \) and \( \varepsilon \in (0, 1) \) such that \( \mathcal{P}\{\tau_\infty \leq T\} > \varepsilon \). Thus there is an integer \( k_1 \geq k_0 \) such that
\[
\mathcal{P}\{\tau_k \leq T\} \geq \varepsilon, \quad \forall k \geq k_1.
\]
Consider the \( C^2 \)-function \( V_1 : \mathbb{R}_+^3 \to \mathbb{R}_+ \) as follows
\[
V_1(S, I, R) = \left( S - a - a \log \frac{S}{a} \right) + (I - 1 - \log(I)) + (R - 1 - \log R).
\]
Let \( X(t) = (S(t), I(t), R(t)) \) then by (1.5) and using the expectation (see [14] and [27]), we obtain for all \( k \geq k_0 \)
\[
EV_1(X(T \wedge \tau_k)) = V_1(X(0)) + \mathbb{E} \int_0^{T \wedge \tau_k} LV_1(X(u \wedge \tau_k))du,
\]
where $L V_1$ is given by

$$LV_1 = \left(1 - \frac{\alpha}{S}\right) \left[\Lambda - \mu S - \left(\beta_1 - \frac{\beta_2 I}{m+I}\right) S I\right] + \frac{a \sigma^2}{2}$$
$$+ \left(1 - \frac{1}{T}\right) \left[\left(\beta_1 - \frac{\beta_2 I}{m+I}\right) S I - (\mu + \gamma) I + \delta R\right] + \frac{\sigma^2}{2}$$
$$+ \left(1 - \frac{1}{R}\right) [\gamma I - (\mu + \delta) R + \frac{\sigma^2}{2}]$$

$$= \Lambda - \mu S - \frac{a \Lambda}{S} + \alpha (\beta_1 - \frac{\beta_2 I}{m+I}) I + \frac{a \sigma^2}{2} - \left(\beta_1 - \frac{\beta_2 I}{m+I}\right) S$$
$$+ 2 \mu + \gamma - \mu I - \frac{\delta R}{T} + \frac{\sigma^2}{2} - \mu R - \frac{\gamma I}{R} + \frac{\sigma^2}{2}$$

$$\leq \Lambda + \alpha (\beta_1 I + \frac{a \sigma^2}{2}) + 2 \mu + \gamma - \mu I + \frac{\sigma^2}{2} + \frac{\sigma^2}{2},$$

choosing $a = \frac{\mu}{\beta_1}$, we obtain

$$LV_1 \leq \Lambda + \alpha (\beta_1 I + \frac{a \sigma^2}{2}) + 2 \mu + \gamma - \mu I + \frac{\sigma^2}{2} + \frac{\sigma^2}{2} = K_0 > 0. \quad (2.4)$$

Substituting (2.4) in (2.3) yields

$$0 \leq \mathbb{E}[V_1(X(\tau_k \wedge T))] \leq V_1(X(0)) + K_0 T. \quad (2.5)$$

Let $\Omega_k = \{\tau_k \leq T\}$. By (2.1) we have $\mathbb{P}(\Omega_k) \geq \varepsilon$, for $k \geq k_1$. On the other hand, from (2.2), we have $V_1(X(T \wedge \tau_k)) \geq 0$, thus

$$\mathbb{E}[V_1(X(T \wedge \tau_k))] = \mathbb{E}[1_{\Omega_k} V_1(X(T \wedge \tau_k))] + \mathbb{E}\left[1_{\Omega^c_k} V_1(X(T \wedge \tau_k))\right]$$
$$\geq \mathbb{E}[1_{\Omega_k} V_1(X(\tau_k))], \quad (2.6)$$

where $1_A$ is the characteristic function of a Borel-set $A$. For every $w \in \Omega_k$, some component of $X(\tau_k, w) = ((S(\tau_k, w), I(\tau_k, w), R(\tau_k, w))$ is equal either $1/k$ or $k$, so

$$V_1(X(\tau_k, w)) \geq A(k), \quad (2.7)$$

where

$$A(k) = \min(f(1, k), f(1, 1/k), f(\mu/\beta_1, k), f(\mu/\beta_1, 1/k), f(\mu/\beta_2, k), f(\mu/\beta_2, 1/k))$$

and $f(a, x) = x - a - a \log \left(\frac{x}{a}\right)$. Combining (2.5)-(2.7) yields

$$V_1(X(0)) + K_0 T \geq \mathbb{E}[1_{\Omega_k} V_1(X(T \wedge \tau_k))]$$
$$\geq A(k) \mathbb{P}(\Omega_k)$$
$$\geq A(k) \varepsilon.$$

By taking limit when $k$ tends to $\infty$ we obtain

$$\infty > V_1(X(0)) + K_0 T = \infty$$

which is a contradiction, so we must have $\tau_\varepsilon = \infty$ a.s. Consequently $S(t)$, $I(t)$ and $R(t)$ are positive and the solution to (1.2) is global. The proof is complete. \qed
3. The dynamic property around the disease-free equilibrium. In this section we are interested in the behavior of the global positive solution \((S(t), I(t), R(t))\) around the disease-free equilibrium \(E_0\).

**Theorem 3.1.** If \(\mathcal{R}_0 \leq 1\) and the following condition is satisfied

\[
\sigma_1^2 < \mu, \quad \sigma_2^2 < 2\mu, \quad \sigma_3^2 < 2\mu,
\]

then for any given initial condition \((S(0), I(0), R(0)) \in \mathbb{R}_+^3\), the solution of model \((1.2)\) has the property

\[
\limsup_{\tau \to +\infty} \frac{1}{t} \int_0^t \left[ (S(u) - \frac{\Lambda}{\mu})^2 + I^2(u) + R^2(u) \right] du \leq \frac{K_1}{m},
\]

where \(m = \min \left[ 2(\mu - \sigma_1^2), (2\mu - \sigma_2^2), (2\mu - \sigma_3^2) \right] \) and \(K_1 = 2 \left( 1 + \frac{2\mu}{\delta} \right) \left( \frac{\Lambda}{\mu} \right)^2 \)

**Proof.** Define the \(C^2\) function

\[
V_2(S, I, R) = \vartheta_1 + x\vartheta_2 + y\vartheta_3,
\]

with

\[
\vartheta_1 = \left( S - \frac{\Lambda}{\mu} + I + R \right)^2, \quad \vartheta_2 = \left( S - \frac{\Lambda}{\mu} + I \right)^2, \quad \vartheta_3 = I + zR,
\]

where \(x, y\) and \(z\) are positive real constants to be chosen in the following. Using Itô formula, we obtain

\[
\begin{align*}
\frac{d\vartheta_1}{\sigma_1} &= L\vartheta_1 dt + 2\left( S - \frac{\Lambda}{\mu} + I + R \right) \left[ \sigma_1 S dB_1(t) + \sigma_2 I dB_2(t) + \sigma_3 R dB_3(t) \right], \\
\frac{d\vartheta_2}{\sigma_2} &= L\vartheta_2 dt + 2\left( S - \frac{\Lambda}{\mu} + I \right) \left[ \sigma_1 S dB_1(t) + \sigma_2 I dB_2(t) \right], \\
\frac{d\vartheta_3}{\sigma_3} &= L\vartheta_3 dt + \sigma_2 I dB_2 + z\sigma_3 R dB_3, \\
\frac{dV_2}{\sigma_3} &= d\vartheta_1 + x d\vartheta_2 + y d\vartheta_3.
\end{align*}
\]

We have,

\[
L\vartheta_1 = 2 \left[ \left( S - \frac{\Lambda}{\mu} \right)^2 + I + R \right] \left[ -\mu \left( S - \frac{\Lambda}{\mu} \right) - \mu I - \mu R \right] + (\sigma_1 S)^2 + (\sigma_2 I)^2 + (\sigma_3 R)^2
\]

\[
= -2\mu \left( S - \frac{\Lambda}{\mu} \right)^2 - (2\mu - \sigma_2^2) I^2 - (2\mu - \sigma_3^2) R^2 - 4\mu I \left( S - \frac{\Lambda}{\mu} \right)
\]

\[
-4\mu R \left( S - \frac{\Lambda}{\mu} \right) - 4\mu IR + \sigma_1^2 \left[ \left( S - \frac{\Lambda}{\mu} \right) + \frac{\Lambda}{\mu} \right]^2,
\]

beside

\[
L\vartheta_2 = 2 \left[ \left( S - \frac{\Lambda}{\mu} \right)^2 + I \right] \left[ -\mu \left( S - \frac{\Lambda}{\mu} \right) - (\gamma + \mu) I + \delta R \right] + (\sigma_1 S)^2 + (\sigma_2 I)^2
\]

\[
= -2\mu \left( S - \frac{\Lambda}{\mu} \right)^2 - \left[ 2(\mu + \gamma) - \sigma_2^2 \right] I^2 - 2(\gamma + 2\mu) I \left( S - \frac{\Lambda}{\mu} \right)
\]
\[ +2\delta R \left( S - \frac{\Lambda}{\mu} \right) + 2\delta IR + \sigma_1^2 S^2 \]
\[ \leq -2\mu \left( S - \frac{\Lambda}{\mu} \right)^2 - (2\mu - \sigma_2^2) I^2 - 2(\gamma + 2\mu) I \left( S - \frac{\Lambda}{\mu} \right) \]
\[ +2\delta R \left( S - \frac{\Lambda}{\mu} \right) + 2\delta IR + \sigma_1^2 \left( S - \frac{\Lambda}{\mu} \right)^2. \]

Using the inequality \((a + b)^2 \leq 2a^2 + 2b^2\) for all \(a, b \in \mathbb{R}\) we obtain
\[ L\vartheta_1 \leq -2(\mu - \sigma_1^2) \left( S - \frac{\Lambda}{\mu} \right)^2 - (2\mu - \sigma_2^2) I^2 - \left( 2\mu - \sigma_1^2 \right)^2 I \]
\[ -2(\gamma + 2\mu) I \left( S - \frac{\Lambda}{\mu} \right) + 2\delta R \left( S - \frac{\Lambda}{\mu} \right) + 2\delta IR + 2 \left( \frac{\Lambda\sigma_1}{\mu} \right)^2, \] (3.3)

and
\[ L\vartheta_2 \leq -2(\mu - \sigma_1^2) \left( S - \frac{\Lambda}{\mu} \right)^2 - (2\mu - \sigma_2^2) I^2 \]
\[ -2(\gamma + 2\mu) I \left( S - \frac{\Lambda}{\mu} \right) + 2\delta R \left( S - \frac{\Lambda}{\mu} \right) + 2\delta IR + 2 \left( \frac{\Lambda\sigma_1}{\mu} \right)^2. \] (3.4)

However
\[ L\vartheta_3 = \left( \beta_1 - \frac{\beta_2 I}{m + I} \right) SI - (\mu + \gamma) I + \delta R + z[\gamma I - (\mu + \delta) R]. \]

Since \(S, I > 0\),
\[ L\vartheta_3 \leq \beta_1 SI + [\gamma z - (\mu + \gamma)] I + [\delta - z(\mu + \delta)] R \]
\[ = \beta_1 I \left( S - \frac{\Lambda}{\mu} \right) + \left[ \frac{\Lambda\beta_1}{\mu} - (\gamma + \mu) + \gamma z \right] I + [\delta - z(\mu + \gamma)] R. \]

Since \(R_0 \leq 1\) we can choose \(z\) such that
\[ \frac{\delta\gamma}{\mu + \delta} \leq \gamma z \leq (\gamma + \mu) - \frac{\Lambda\beta_1}{\mu} = \frac{\delta\gamma}{\mu + \delta} + \frac{\mu(\mu + \delta + \gamma)}{\mu + \delta}(1 - R_0) \]

Using the fact that \(I, R > 0\),
\[ L\vartheta_3 \leq \beta_1 I \left( S - \frac{\Lambda}{\mu} \right). \] (3.5)

By choosing \(x = \frac{2\mu}{\delta}\) and \(\beta_1 y = 4\mu + x(2\mu + \gamma)\), then combining Eq (3.3)-(3.5), we obtain
\[ LV_2 \leq -2(\mu - \sigma_1^2) \left( 1 + \frac{2\mu}{\delta} \right) \left( S - \frac{\Lambda}{\mu} \right)^2 - (2\mu - \sigma_2^2) \left( 1 + \frac{2\mu}{\delta} \right) I^2 \]
\[ - (2\mu - \sigma_3^2) R^2 + 2 \left( 1 + \frac{2\mu}{\delta} \right) \left( \frac{\Lambda\sigma_1}{\mu} \right)^2. \] (3.6)
Substituting \( m = \min \left[ 2(\mu - \sigma_1^2) \left(1 + \frac{2\mu}{\delta}\right), (2\mu - \sigma_2^2) \left(1 + \frac{2\mu}{\delta}\right), (2\mu - \sigma_3^2) \right] \) into \( dV_2 \),

\[
dV_2 \leq -m \left[ \left( S - \frac{\Lambda}{\mu} \right)^2 + I^2 + R^2 \right] + 2 \left( 1 + \frac{2\mu}{\delta} \right) \left( \frac{\Lambda \sigma_1}{\mu} \right)^2 \\
+ 2\sigma_1 \left[ 2 \left( \frac{\Lambda}{\mu} - S \right) + 2I + R \right] SdB_1(t) \\
+ 2\sigma_2 \left[ 2 \left( S - \frac{\Lambda}{\mu} \right) + 2I + R \right] IdB_2(t) + \sigma_3 \left[ \left( S - \frac{\Lambda}{\mu} \right) + I + R \right] RdB_3(t).
\]

By integration we deduce that

\[
0 \leq V_2(t) \leq V_2(0) - m \int_0^t \left[ \left( S(u) - \frac{\Lambda}{\mu} \right)^2 + I(u)^2 + R(u)^2 \right] du + M(t) \\
+ 2 \left( 1 + \frac{2\mu}{\delta} \right) \left( \frac{\Lambda \sigma_1}{\mu} \right)^2 t, \tag{3.7}
\]

where \( M(t) \) is a martingale defined by

\[
M(t) = 2\sigma_1 \int_0^t \left[ S - \frac{\Lambda}{\mu} + I + R + x \left( S - \frac{\Lambda}{\mu} + I \right) \right] SdB_1(t) \\
+ 2\sigma_2 \int_0^t \left[ S - \frac{\Lambda}{\mu} + I + R + x \left( S - \frac{\Lambda}{\mu} + I \right) + y \right] IdB_2(t) \\
+ \sigma_3 \int_0^t \left[ 2 \left( S - \frac{\Lambda}{\mu} + I + R \right) + yz \right] RdB_3(t).
\]

Using Expectation, dividing both sides of (3.7) by \( t \) and letting \( t \to \infty \), it follows that

\[
\lim_{t \to \infty} \frac{1}{t} \mathbb{E} \int_0^t \left[ \left( S - \frac{\Lambda}{\mu} \right)^2 + I^2 + R^2 \right] ds \leq \frac{2}{m} \left( 1 + \frac{2\mu}{\delta} \right) \left( \frac{\Lambda \sigma_1}{\mu} \right)^2 \text{ a.s.} \tag{3.8}
\]

The proof is therefore complete. \( \square \)

**Remark 1.**

1. Note that, in general, the disease-free state is not an equilibrium of the stochastic model. Consequently, we mainly estimate the average oscillation around this equilibrium to exhibit whether the disease will die out. In a biological interpretation, one expects any solution to be close to the deterministic equilibrium most of the time if the stochastic effects are small.

2. If \( R_0 \leq 1 \) and under the conditions of Theorem 3.1 we conclude that the solution fluctuates around the disease-free equilibrium. Furthermore if \( \sigma_1 = 0 \) then the disease-free equilibrium \( E_0 = (\Lambda/\mu, 0, 0) \) is an equilibrium of stochastic model (1.2). In this case, (3.6) becomes

\[
LV_2 \leq -2(\mu - \sigma_1^2) \left( 1 + \frac{2\mu}{\delta} \right) \left( S - \frac{\Lambda}{\mu} \right)^2 - (2\mu - \sigma_2^2) \left( 1 + \frac{2\mu}{\delta} \right) I^2 - (2\mu - \sigma_3^2) R^2.
\]

Then under assumption \( \sigma_1 = 0 \) and using the Lyapunov method, we deduce the following result

If \( R_0 \leq 1, \sigma_2^2 < 2\mu \) and \( \sigma_3^2 < 2\mu \) then the disease-free equilibrium \( E_0 = (\Lambda/\mu, 0, 0) \) of system (1.2) is globally asymptotically stable, namely the disease will extinct with probability 1.
3. If \( \sigma_1 = \sigma_2 = \sigma_3 = 0 \) then Equation (3.6) becomes

\[
LV_2 \leq -2\mu \left( 1 + \frac{2\mu}{\delta} \right) \left( S - \frac{\Lambda}{\mu} \right)^2 - 2\mu \left( 1 + \frac{2\mu}{\delta} \right) I^2 - 2\mu R^2.
\]

By Lyapunov’s method, the disease-free equilibrium \( E_0 \) of the deterministic model (1.1) is globally asymptotically stable, if \( R_0 \leq 1 \).

4. The dynamic property around the endemic equilibrium. In this section we assume that \( R_0 > 1 \). Let us first prove that there is only one endemic equilibrium. Indeed, if we let the left-hand side of each differential equation of system (1.1) be zero, the endemic \((S^*, I^*, R^*)\) satisfies \( S^* > 0, I^* > 0, R^* > 0 \) and

\[
\begin{align*}
\Lambda - \mu S^* - \left( \beta_1 - \frac{\beta_2 I^*}{m + I^*} \right) S^* I^* &= 0, \\
\left( \beta_1 - \frac{\beta_2 I^*}{m + I^*} \right) S^* I^* - (\mu + \gamma) I^* + \delta R^* &= 0,
\end{align*}
\]

(4.1)

This implies that

\[
S^* = \frac{\gamma + \mu - \frac{\delta \gamma}{\mu + \delta}}{\beta_1 - \frac{\beta_2}{m + I^*}}, \quad R^* = \frac{\gamma I^*}{\mu + \delta}.
\]

Substituting the above expressions into the first equation of system (4.1), we obtain

\[
AI^*^2 + BI^* + C = 0,
\]

where

\[
\begin{align*}
A &= -\mu(\beta_1 - \beta_2) \left( 1 + \frac{\gamma}{\mu + \delta} \right), \\
B &= \Lambda(\beta_1 - \beta_2) - \mu(\mu + m\beta_1) \left( 1 + \frac{\gamma}{\mu + \delta} \right), \\
C &= m\mu^2(R_0 - 1) \left( 1 + \frac{\gamma}{\mu + \delta} \right).
\end{align*}
\]

Since \( R_0 > 1 \) and \( \beta_1 \geq \beta_2 \) we have \( A \leq 0 \) and \( C > 0 \), hence system (1.1) has a unique endemic equilibrium \( E^* = (S^*, I^*, R^*) \). Then, before studying the behavior of the global positive solution \((S(t), I(t), R(t))\) of the system (1.2) around the endemic equilibrium \( E^* \), we define \( K_2 \) and \( m_2 \) as follows

\[
\begin{align*}
m_2 &= \min_{1 \leq i \leq 3} (\mu - \sigma_i^2), \\
K_2 &= \frac{(\frac{2\mu}{\delta} + 1)(\sigma_1^2(S^*)^2 + \sigma_2^2(I^*)^2) + (\sigma_3 R^*)^2 + \frac{2\sigma_3^2 (2\mu + \gamma) + 2\mu}{\delta_1 - \frac{\sigma_2^2}{m + I^*}} \left( \frac{\sigma_2^2 I^* + \sigma_2^2 (R^*)^2}{2\delta I^*} \right)}{\beta_1 - \frac{\beta_2}{m + I^*}}.
\end{align*}
\]

(4.2)

**Theorem 4.1.** Assume \( R_0 > 1 \) and

\[
\sigma_i^2 < \mu \quad i = 1, 2, 3.
\]

Then, for any given initial condition \((S(0), I(0), R(0)) \in \mathbb{R}_+^3\), the solution \((S(t), I(t), R(t))\) satisfies

\[
\limsup_{t \to \infty} \frac{1}{t} \mathbb{E} \left[ \int_0^t \left( (S(u) - S^*)^2 + (I(u) - I^*)^2 + (R(u) - R^*)^2 \right) du \right] \leq \frac{K_2}{m_2}.
\]
Proof. We consider the following function:

\[ V_1(t, R(t)) = W_1(t, I(t), R(t)) + qW_2(t, I(t), R(t)) \]

where \( W_1 \) and \( W_2 \) are two functions defined as follows

\[
W_1(t, I(t), R(t)) = \frac{1}{2} (S - S^* + I - I^* + R - R^*)^2 + \frac{p}{2} (S - S^* + I - I^*)^2
\]

\[
W_2(t, I(t), R(t)) = \left( I - I^* - I^* \log \frac{I}{I^*} \right) + r \left( R - R^* - R^* \log \frac{R}{R^*} \right),
\]

where \( p, q \) and \( r \) are three positive constants to be determined below. Using Itô's formula,

\[
dW_1 = LW_1 dt + (S - S^* + I - I^* + R - R^*)[\sigma_1 S dB_1 + \sigma_2 I dB_2 + \sigma_3 R dB_3] + p(S - S^* + I - I^*)[\sigma_1 S dB_1 + \sigma_2 I dB_2] \]

\[
dW_2 = LW_2 dt + \sigma_2(I - I^*) dB_2 + r\sigma_3(R - R^*) dB_3,
\]

\[
dV_2 = dW_1 + qdW_2. \tag{4.4}
\]

We have

\[
LW_1 = (S - S^* + I - I^* + R - R^*)(\lambda - \mu S - \mu I - \mu R)
\]

\[
+ p(S - S^* + I - I^*)(\lambda - \mu S - (\mu + \gamma) I + \delta R) + \frac{p + 1}{2} (\sigma_1^2 S^2 + \sigma_2^2 I^2)
\]

\[
+ \frac{\sigma_3^2 R^2}{2}
\]

\[
= (S - S^* + I - I^* + R - R^*)[-\mu(S - S^*) - \mu(I - I^*) - \mu(R - R^*)]
\]

\[
+ p(S - S^* + I - I^*)[-\mu(S - S^*) + (\mu + \gamma)(I - I^*) + \delta(R - R^*)]
\]

\[
+ \frac{p + 1}{2} (\sigma_1^2 S^2 + \sigma_2^2 I^2) + \frac{\sigma_3^2 R^2}{2}
\]

\[
= -\mu(1 + p)(S - S^*)^2 - (\mu + p(\mu + \gamma))(I - I^*)^2 - \mu(R - R^*)^2
\]

\[
- (p(2\mu + \gamma) + 2\mu)(S - S^*)(I - I^*)
\]

\[
+ (p\delta - 2\mu)(S - S^*)(R - R^*) + (p\delta - 2\mu)(I - I^*)(R - R^*)
\]

\[
+ \frac{p + 1}{2} (\sigma_1^2 S^2 + \sigma_2^2 I^2) + \frac{\sigma_3^2 R^2}{2}, \tag{4.5}
\]

beside

\[
LW_2 = (I - I^*) \left[ \left( \beta_1 - \frac{\beta_2 I}{m + I} \right) S - (\mu + \gamma) S + \delta \frac{R}{I} \right] + \frac{\sigma_2^2 I^*}{2}
\]

\[
+ r \left( 1 - \frac{R^*}{R} \right) (\gamma I - (\mu + \delta) R) + r \frac{\sigma_3^2 R^*}{2}
\]

Since \( \mu + \delta = \gamma \frac{I^*}{R^*} \),

\[
LW_2 = (I - I^*) \left[ \left( \beta_1 - \frac{\beta_2 I}{m + I} \right) S - \left( \beta_1 - \frac{\beta_2 I^*}{m + I^*} \right) S^* + \delta \left( \frac{R}{I} - \frac{R^*}{I^*} \right) \right]
\]

\[
+ \frac{\sigma_2^2 I^* \gamma}{2} \left( 1 - \frac{R^*}{R} \right) (I - I^*) + r \frac{\sigma_3^2 R^*}{2}
\]
\[ LW_2 = -S(I - I^*)(\frac{\beta_2 I}{m + I} - \frac{\beta_2 I^*}{m + I^*}) + (I - I^*)\left(\frac{\beta_1 I}{m + I} - \frac{\beta_1 I^*}{m + I^*}\right)(S - S^*) + \frac{\sigma_2^2}{2} I^* r I^* \left(\frac{R + I - R I^*}{R R^*} + 1\right) + \delta R^* \left(\frac{R}{R^*} - \frac{R}{R^*} R I^* + 1\right) + r \frac{\sigma_3^2}{2} R^* \] (4.6)

Choosing \( p, q, r \) such that
\[ p = \frac{2\mu}{\delta}, \quad r \gamma I^* = \delta R^* \quad \text{and} \quad q = \frac{p(2\mu + \gamma) + 2\mu}{\beta_1 - \frac{\beta_2 I^*}{m + I^*}}. \] (4.7)

Moreover noticing that both \( I - I^* \) and \( \frac{\beta_2 I}{m + I} - \frac{\beta_2 I^*}{m + I^*} \) have the same sign. Then substituting (4.5) and (4.6) in (4.4) we deduce

\[ LV_3 \leq -\mu(p + 1)(S - S^*)^2 - (\mu + p(\mu + \gamma))(I - I^*)^2 - \mu(R - R^*)^2 + \frac{\sigma_2^2}{2} R^2 + \delta R^* \left(2 - \frac{RI^*}{IR^*} - \frac{RI^*}{IR^*}\right) + \frac{p + 1}{2}(\sigma_1^2 S^2 + \sigma_2^2 I^2) + q \left(\frac{\sigma_2^2}{2} I^* + r \frac{\sigma_3^2}{2} R^*\right) \]
\[ = -\mu(p + 1)(S - S^*)^2 - (\mu + p(\mu + \gamma))(I - I^*)^2 - \mu(R - R^*)^2 + \frac{\sigma_2^2}{2} R^2 - \delta R^* \left(\sqrt{\frac{RI^*}{IR^*}} - \sqrt{\frac{RI^*}{IR^*}}\right)^2 + \frac{p + 1}{2}(\sigma_1^2 S^2 + \sigma_2^2 I^2) + q \left(\frac{\sigma_2^2}{2} I^* + r \frac{\sigma_3^2}{2} R^*\right) \]
\[ \leq -\mu(p + 1)(S - S^*)^2 - (\mu + p(\mu + \gamma))(I - I^*)^2 - \mu(R - R^*)^2 + \frac{p + 1}{2}(\sigma_1^2 S^2 + \sigma_2^2 I^2) + \frac{\sigma_2^2}{2} R^2 + q \left(\frac{\sigma_2^2}{2} I^* + r \frac{\sigma_3^2}{2} R^*\right). \]

Using the inequalities \( a^2 \leq 2(a - b)^2 + 2b^2 \), we obtain

\[ LV_3 \leq -\mu(p + 1)(S - S^*)^2 - (\mu + p(\mu + \gamma))(I - I^*)^2 - (\mu - \sigma_1^2)(S - S^*)^2 - (\mu - \sigma_2^2)(I - I^*)^2 \]
\[ - (\mu - \sigma_3^2)(R - R^*)^2 + (p + 1)(\sigma_1^2(S^*)^2 + \sigma_2^2(I^*)^2 + \sigma_3^2(R^*)^2) + q \left(\frac{\sigma_2^2}{2} I^* + r \frac{\sigma_3^2}{2} R^*\right) \]
\[ \leq -(\mu - \sigma_1^2)(S - S^*)^2 - (\mu - \sigma_2^2)(I - I^*)^2 - (\mu - \sigma_3^2)(R - R^*)^2 \]
\[ + (p + 1)(\sigma_1^2(S^*)^2 + \sigma_2^2(I^*)^2 + \sigma_3^2(R^*)^2) + q \left(\frac{\sigma_2^2}{2} I^* + r \frac{\sigma_3^2}{2} R^*\right) \]. (4.8)

Substituting (4.8) in (4.4), then integrating both sides of (4.4) between 0 and \( t \) and taking expectation we get

\[ 0 \leq E[V_3(S(t), I(t), R(t))] \leq V_3(S(0), I(0), R(0)) + tK_2 \]
\[ + E \left[ \int_0^t \left( -\mu - \sigma_1^2 \right) (S(u) - S^*)^2 - \mu - \sigma_2^2)(I(u) - I^*)^2 - \mu - \sigma_3^2)(R(u) - R^*)^2 \right] du. \]
perturbations is small enough. Moreover in a biological view, the disease will persist when the intensity of stochastic solution of system (1.2) will fluctuate around the deterministic endemic equilibrium. From Theorem 4.1 we can conclude that if 

\[
\lim_{t \to \infty} \frac{1}{t} \mathbb{E} \left[ \int_0^t ((S(u) - S^*)^2 + (I(u) - I^*)^2 + (R(u) - R^*)^2) du \right] \leq \frac{V_3(S(0), I(0), R(0))}{m_2} + \frac{K_2}{m_2} t. 
\]

Hence

\[
\lim_{t \to \infty} \sup \frac{1}{t} \mathbb{E} \left[ \int_0^t ((S(u) - S^*)^2 + (I(u) - I^*)^2 + (R(u) - R^*)^2) du \right] \leq \frac{K_2}{m_2}.
\]

**Remark 2.** From Theorem 4.1 we can conclude that if \( R_0 > 1 \) and \( |\sigma| \) is small then solution of system (1.2) will fluctuate around the deterministic endemic equilibrium. Moreover in a biological view, the disease will persist when the intensity of stochastic perturbations is small enough.

Taking \( |\sigma| = 0 \) then Equation (4.8) becomes

\[
LV_3 \leq -\mu(S - S^*)^2 - \mu(I - I^*)^2 - \mu(R - R^*)^2. 
\]

By Lyapunov analysis, we have

**Corollary 1.** If \( R_0 > 1 \), the deterministic system (1.1) has a unique endemic equilibrium \( E^*(S^*, I^*, R^*) \) and it is globally asymptotically stable.

5. **Stationary distribution and ergodicity.** For the epidemic dynamical system (1.2), we are also interested in analyzing and proving some sufficient conditions ensuring that the disease persists and prevails in the host population. Since system (1.2) has no endemic equilibrium, we cannot show the persistence of the disease by proving the stability of the positive equilibrium as for the deterministic system. In this section using the theory of Has’minskii [14] we show that there is a stationary distribution of system. For the completeness of the paper, in this section, we list some theories about stationary distribution (see [14]).

Let \( X(t) \) be a homogeneous Markov process in \( E_d \) (denotes \( d \)-dimensional Euclidean space), described by the following stochastic differential equation.

\[
dX(t) = G(X(t))dt + \sum_{k=1}^{k=n} H_k(X(t))dB_k(t) \tag{5.1}
\]

The diffusion matrix is \( A(x) = (a_{ij}) \), \( a_{ij} = \sum_{k=1}^{k=n} H^i_k(x)H^j_k(x) \).

**Definition 5.1.** [14] Let \( \mathcal{P}(t, X, .) \) be the probability measure induced by \( X(t) = (S(t), I(t), R(t)) \) with initial value \( (S(0), I(0), R(0)) = x \). That is, \( \mathcal{P}(t, X, A) = \mathcal{P}(X(t) \in A|X(0) = x) \), for any Borel set \( A \subset \mathbb{R}^3_+ \). If there exists a probability measure \( \mu(.) \) such that \( \lim_{t \to \infty} \mathcal{P}(t, X, A) = \mu(A) \) for all \( X \in \mathbb{R}^3_+ \), then we say that SDE. (5.1) has a stationary distribution \( \mu(.) \).

**Lemma 5.2** [14]. Assume that there exists a bounded domain \( \mathcal{U} \subset E_d \) with regular boundary, which has the following properties:

\( (H_1) \) In the domain \( \mathcal{U} \) and some neighbourhood thereof, the smallest eigenvalue of the diffusion matrix \( A(X) \) is bounded away from zero.

\( (H_2) \) If \( x \in E_d \setminus \mathcal{U} \), the mean time \( \tau \) at which a path issuing from \( x \) reaches the set \( \mathcal{U} \) is finite, and \( \sup_{x \in K} \mathbb{E}_x(\tau) < \infty \) for every compact subset \( K \subset E_d \setminus \mathcal{U} \).
Then the Markov process $X(t)$ has a stationary distribution $\mu(\cdot)$. Moreover if $f(\cdot)$ is a integrable function with respect to the measure $\mu$, then

$$\mathcal{P} \left( \lim_{T \to \infty} \frac{1}{T} \int_0^T f(X(t))dt = \int_{E_{\delta}} f(x)\mu(dx) \right) = 1 \tag{5.2}$$

**Remark 3.** The proof of Theorem 2.1 shows that there exists a function $V$ such that $LV \leq K$, and define $\tilde{V} = V + K$, then $L\tilde{V} \leq \tilde{V}$, and

$$V_k = \inf_{(x,y,z) \in \mathbb{R}^3_+ \setminus D_k} \tilde{V} \to \infty \text{ a.s. } k \to \infty,$$

where $D_k = (1/k,k) \times (1/k,k) \times (1/k,k)$. Hence by \cite[Theorem 3.5, p 75]{14}, the solution $(S(t), I(t), R(t))$ is a homogeneous Markov process in $\mathbb{R}^3_+$. Then to show the existence of stationary distribution for system (1.2) we use \cite[Remark 2.1]{36}

**Remark 4 ([36]).** (i) To validate assumption $(H_2)$ of Lemma 5.2 (see \cite{41}), it is sufficient to show that there is some neighbour $\tilde{U}$ and non negative $C^2$ function $V$ such that there exists a positive constant $C$ such that,

$$LV(X) < -C \text{ for all } X \in \mathbb{R}^3_+ \setminus \tilde{U},$$

(ii) To verify $(H_1)$ of Lemma 5.2 (see \cite[Chapter 3]{10}, and Rayleigh’s principle in \cite[Chapter 6]{29}) it is sufficient to prove that there is a positive number $M$ such that for any bounded domain $D \subset \mathbb{R}^3_+$ we have

$$\sum_{i,j=1}^3 a_{ij}(x)\xi_i\xi_j \geq M||\xi||^2, \ x \in \bar{D}, \ \xi \in \mathbb{R}^3_+.$$

Using the same notation as seen previously in (4.2) we have the following result.

**Theorem 5.3.** If

$$0 < K_2 < \min((\mu - \sigma_1^2)(S^*)^2,(\mu - \sigma_2^2)(I^*)^2,(\mu - \sigma_3^2)(R^*)^2),$$

then for any given initial condition $(S(0), I(0), R(0)) \in \mathbb{R}^3_+$, system (1.2) has a stationary distribution and the solutions have ergodic property (5.2).

**Proof.** We use the nonnegative $C^2$-function $V_3(S, I, R)$ as in Theorem 2.1. Hence, it follows from (4.8) that

$$LV_3 \leq -((\mu - \sigma_1^2)^2(S - S^*)^2 - (\mu - \sigma_2^2)(I - I^*)^2 - (\mu - \sigma_3^2)(R - R^*)^2) + K_2.$$

Note that the ellipsoid

$$(\mu - \sigma_1^2)^2(S - S^*)^2 + (\mu - \sigma_2^2)(I - I^*)^2 + (\mu - \sigma_3^2)(R - R^*)^2 = K_2,$$

lies entirely in $\mathbb{R}^3_+$. We can take $\tilde{U}$ to be any neighborhood of the ellipsoid such that $\tilde{U} \subset \mathbb{R}^3_+$. From (4.8), we deduce that $LV_3(x) < 0$ for any $x \in \mathbb{R}^3_+ \setminus \tilde{U}$, which implies that condition $(H_2)$ in Lemma 5.2 is satisfied.

On the other hand, system (1.2) can be written as follows

$$d\begin{pmatrix} S \\ I \\ R \end{pmatrix} = \begin{pmatrix} \Lambda - \left( \frac{\beta_1}{m+I} \frac{\beta_2}{m+I} \right) S I - \mu S + \delta R \\ \left( \frac{\beta_1}{m+I} \frac{\beta_2}{m+I} \right) S I - (\gamma + \mu) I \\ \gamma I - (\mu + \delta) R \end{pmatrix} dt + \begin{pmatrix} \sigma_1 S \\ 0 \\ 0 \end{pmatrix} dB_1(t)$$

$$+ \begin{pmatrix} 0 \\ \sigma_2 I \\ 0 \end{pmatrix} dB_2(t) + \begin{pmatrix} 0 \\ 0 \\ \sigma_3 R \end{pmatrix} dB_3(t),$$
then the diffusion matrix is $A(X) = \text{diag}(\sigma_1^2 S^2, \sigma_2^2 I^2, \sigma_3^2 R^2)$.

Besides for all $(S, I, R) \in \mathcal{U}$, $\xi \in \mathbb{R}^3_+$, there is

$$M = \min(\sigma_1^2 S^2, \sigma_2^2 I^2, \sigma_3^2 R^2) > 0$$

such that

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

Then, by Rayleigh’s principle [29, p.342], condition (H1) in Lemma 5.2 is also verified. Therefore the stochastic model (1.2) has a stationary distribution $\mu(\cdot)$ and it is ergodic.

6. Numerical simulations.

**Example 6.1. Zoonotic tuberculosis in Morocco** Zoonotic diseases play a very important role among human communicable diseases. In a review of more than 1400 pathogens known to infect humans, it was found that more than 61% where zoonotic [1]. Zoonotic diseases often serve as a starting point of many pathogens that jump the species barrier and became effectively transmissible human to human. This example represents a simulation for transmission dynamic and elimination potential of zoonotic tuberculosis in Morocco [1]. The average lifespan of the Moroccan cattle is 6 years which yields to a death rate of $\mu = 0.167$. From the data cattle population using least squares the birth rate was estimated to 0.177. The cattle to cattle transmission rate of bovine tuberculosis is of the order $\beta = 0.249$, was estimated from the endemic prevalence in cattle. we choose $\delta = \beta_2 = 0.1$, $\gamma = 0.2$ and $m = 1$, $\sigma_1 = 0.02$, $\sigma_2 = 0.1$, $\sigma_3 = 0.2$, So, $R_0 = 0.89035$. Figure 1 is an illustration of the trajectories of the solutions to the models (1.1) and (1.2) using the parameters cited before. In this example, using the improved Milstein method [15] with step size $\Delta = 10^{-3}$, we give the numerical solution to system (1.2), with the given initial positive value $S(0) = 0.7; I(0) = 0.12; R(0) = 0.18$ and parameters cited before. Hence, according to Theorem 3.1, all positive solutions of the system (1.2) always fluctuate around the curves of system (1.1). Figure 1 clearly support this result.

**Example 6.2. Herpes simplex virus type 2** Herpes simplex virus type 2 is a human disease transmitted by close physical or sexual contact. The virus usually infects the genital tract or oral mucosa. An individual with herpes remains infected for life and the virus reactivate regularly producing a relapse period of infectiousness. Herpes simplex virus type 2 (HSV-2) is the most prevalent sexually transmitted pathogen worldwide. There is a considerable biological and epidemiological evidence that HSV-2 infection increases the risk of acquiring HIV infection and may also the risk of transmitting HIV [4]. For herpes an SIRI model is appropriate. In this example we are interested in presenting a simulation to illustrate the case of (HSV-2). For this we consider the same parameters presented by Blower [4]. Let $\Lambda = 0.1$, $\mu = 0.05$, $\beta_1 = 0.2$, $\beta_2 = 0.15$, $m = 1$ and $\gamma = \delta = 0.2857$. In this example, choose initial values $S(0) = 0.7; I(0) = 0.12; R(0) = 0.1$ and time step $\Delta = 0.001$, $\sigma_1 = 0.02$, $\sigma_2 = 0.01$, $\sigma_3 = 0.02$ So, $R_0 = 4.3219 > 1$ which implies that an
infected individual introduced into an entirely susceptible population will produce an average more than one infected in the next generation. Hence, according to Theorem 4.1, the solution of system (1.2) fluctuates around the solution of system (1.1). Fig 2 clearly support this result.

**Example 6.3.** In this example, using the parameter values \( \Lambda = 0.726, \mu = 0.3; \beta_1 = 0.28, \beta_2 = 0.01; \gamma = 0.2, \delta = 0.26, \ m = 1; \sigma_1 = 0.05; \sigma_2 = 0.01; \sigma_3 = 0.003 \) and initial values \( S(0) = 0.7, \ I(0) = 0.12, \ R(0) = 0.18 \). Hence, by computation we obtain \((\mu - \sigma_1^2)(S^*)^2 = 0.6479, (\mu - \sigma_2^2)(I^*)^2 = 0.1452, (\mu - \sigma_3^2)(R^*)^2 = 0.0185, \) and \( K_2 = 0.0185 \), so \( K_2 - m_2 = -4.3144 \times 10^{-5} \). Then from Theorem 5.3, the system (1.2) has a stationary distribution. This situation is illustrated by Figure 3 and Figure 4. Here the step size is \( \Delta t = 0.1 \). In Figure 3 and Figure 4 we see the density function at \( t = 9000 \) (see Figure 3) and \( t = 9500 \) (see Figure 4) are very close to each other that can be considered as a good estimation of the stationary distribution of system (1.2).

**Example 6.4. The impact of media coverage** To illustrate the impact of media on the spread of an epidemic, we consider a variation of the parameter of the media coverage \( \beta_2 = 0.01, 0.1, \) and 0.15 with \( \Lambda = 0.8, \mu = 0.25, \beta_1 = 0.18, \gamma = 0.2, \ m = 1; \sigma_1 = 0.01; \sigma_2 = 0.04; \sigma_3 = 0.3 \) and initial values \( S(0) = 0.5, \ I(0) = 0.4 \) and \( R(0) = 0.1 \). We remark that increasing the media coverage parameter, decreases magnitude of infected individuals (Figure 4). This states that the media coverage can reduce the propagation of the disease among the population.

**Conclusion.** In this work we have established the existence and the uniqueness of a global positive solution for a stochastic SIRI epidemic model and studied its the dynamical behavior. The analysis of the stochastic system shows the dynamical
Figure 2. Trajectories of stochastic and deterministic systems with the parameters values given in previous Example 2

Figure 3. The kernel density function estimations of $S(t)$, $I(t)$ and $R(t)$ of stochastic system (1.2) at time $t = 9000$, based on 10000 stochastic simulation with the parameters values given in Example 3
behavior around disease-free and endemic equilibria. This implies, in particular, that when the intensity of the stochastic perturbation is small enough, the epidemic will tend to die out provided that the reproduction number does not exceed a critical level. However, small stochastic disturbance can lead to persistence of the disease, when the reproduction number is larger than a critical level. Numerical results support the conclusions of our theoretical studies. Furthermore, simulations show that the role of media is crucial for reducing incidence. Increasing the media
coverage rate reduces the basic reproduction number, and hence the magnitude of the infectious individuals in the population. 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.

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