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A STOCHASTIC SIRI EPIDEMIC MODEL WITH LÉVY NOISE

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Abstract. Some diseases such as herpes, bovine and human tuberculosis exhibit relapse in which the recovered individuals do not acquit permanent immunity but return to infectious class. Such diseases are modeled by SIRI models. In this paper, we establish the existence of a unique global positive solution for a stochastic epidemic model with relapse and jumps. We also investigate the dynamic properties of the solution around both disease-free and endemic equilibria points of the deterministic model. Furthermore, we present some numerical results to support the theoretical work.

1. **Introduction and model formulation.** Epidemiology is a quantitative discipline that relies on a working knowledge of probability and statistics. Mathematical modeling is one of the most important themes in epidemiology. It has been widely used to analyze the spread of infectious diseases [8, 10]. This kind of diseases can be modeled by SIR models which are useful for studying chickenpox, mumps, or rubella [2, 3, 30]. In mathematical models we often distinguish between two types: deterministic and stochastic. Deterministic models are those in which there is no element of chance or uncertainty. As such, they can be thought to account for the mean trend of a process only. Stochastic models, on the other hand, account not only for the mean trend but also for the variance structure around it. For fixed starting values, a deterministic model will always produce the same result whereas a stochastic model may produce many different outputs, depending on the actual values the random variables take. Many infectious diseases, including Hepatitis B virus (HBV) [28], Hepatitis C virus (HCV) [16], the majority of human tuberculosis [21], herpes virus, have recurrent episodes such that the diseases are hard to be radically cured. For instance, Herpes simplex virus type 2 (HSV-2) that is usually transmitted by close physical or sexual contact, can cause genital herpes [17]. The major morbidity of genital herpes arises from its frequent reactivation rate.

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In one study, within one year after diagnosis, 90% of patients with a documented first episode of genital HSV-2 infection experience at least 1 relapse [4]. There have been many mathematical models [23, 25, 34, 39, 41] committed to study the impacts from relapse on dynamics [6, 37]. Most recently, Liu et al. [25] obtained global stability for an SEIR epidemic model with age-dependent relapse and latency by using Volterra-type Lyapunov functions. An epidemic model with relapse, which incorporates bilinear incidence rate and constant total population, was formulated by Tudor [32]. This system was extended to include nonlinear incidence functions by Moreira and Wang [29]. Blower [6] 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 [34], certain threshold stability results being obtained by particularizing $P(t)$. See also van den Driessche et al. [33] for an analysis of a related SEIRI model. For other works see [26, 40, 13] and the references therein. A deterministic SIRI disease can be modeled as follows

$$\begin{aligned} \dot{S}(t) &= N - \mu S(t) - \beta S(t) I(t), \\ \dot{I}(t) &= \beta S(t) I(t) - (\mu + \gamma) I(t) + \delta R(t), \\ \dot{R}(t) &= \gamma I(t) - (\mu + \delta) R(t), \end{aligned} \tag{1.1}$$

where, $N, \beta, \mu, \delta, \gamma$ are all positive constants, $S(t)$ is the number of the individuals susceptible to the disease, $I(t)$ denotes the infected members and $R(t)$ represents the members who have recovered from the infection. In this model, the parameters have the following features: N is the total number of the susceptible, β is the disease transmission coefficient, μ represents the natural death rate, γ represents the rate of recovery from infection and δ is the rate of relapse.
The basic reproduction number [9, 11]

$$\mathcal{R}_0 = \frac{N\beta(\mu + \delta)}{\mu^2(\mu + \gamma + \delta)},$$

has a great importance in epidemiology since it is a threshold quantity which determines whether an epidemic occurs or the disease simply dies out. It can also be defined as the number of secondary infections caused by a single infective introduced into a population made up entirely of susceptible individuals over the course of the infection of this single infective. The deterministic model (1.1) has been discussed by Vargas-De-León in [36]. He showed the following behaviors of solutions according to the value of the threshold $\mathcal{R}_0$.
If $\mathcal{R}_0 \leq 1$, then the model (1.1) has only the disease-free equilibrium

$$E_0 = (\frac{N}{\mu}, 0, 0),$$

and moreover, it is globally asymptotically stable, this means that the endemic disease will not appear.
If $\mathcal{R}_0 > 1$, E_0 becomes unstable, and therefore there exists a globally asymptotically stable equilibrium

$$E^* = (S^*, I^*, R^*) = (\frac{N}{\mu\mathcal{R}_0}, \frac{\mu}{\beta}(\mathcal{R}_0 - 1), \frac{\mu\gamma}{\beta(\mu + \delta)}(\mathcal{R}_0 - 1)),$$

which means that the disease will be persistent ([15], [35]).
In the few past years, authors have been paying a great interest to epidemic models with a stochastic perturbation [14, 19, 24, 38, 46] and epidemic models with random perturbation [7]. Tornatore et al. [31] introduced a stochastic SIR epidemic model with and without distributed time delay, they investigated the stability of disease-free equilibrium. Gray et al. [12] considered a stochastic SIS epidemic model with a constant population size and studied the existence of a stationary distribution and the persistence. Zhao and Jiang [47, 48] were interested in a stochastic SIS model with vaccination, they presented a threshold value on the extinction and persistence for the model. Zhou et al. [49] investigated survival and stationary distribution of a stochastic SIR epidemic model. Lahrouz and Settati discussed necessary and sufficient conditions for extinction and persistence of a stochastic SIRS system [20] Lei and Yang [22] proposed a stochastic version of the SIRI epidemic model, they studied the dynamical behavior of the following stochastic model

$$\begin{aligned} dS(t) &= [N-\mu S(t)-\beta S(t)I(t)]dt+\sigma_1 S dW_1(t),\\ dI(t) &= [\beta S(t)I(t)-(\mu+\gamma)I(t)+\delta R(t)]dt+\sigma_2 I dW_2(t),\\ dR(t) &= [\gamma I(t)-(\mu+\delta)R(t)]dt+\sigma_3 R dW_3(t), \end{aligned} \tag{1.2}$$

where $W_i(t),\ i=1,2,3$ are independent Brownian motions and $\sigma_i^2,\ i=1,2,3$ are the corresponding intensities of stochastic perturbations. Since both E_0 and E^* do not represent equilibria points to the model (1.2), the authors showed that for any initial value $(S(0),I(0),R(0))^T\in\mathbb{R}^3_+$:
If $\mathcal{R}_0\leq 1$, the global positive solution fluctuates randomly around the disease-free equilibrium E^0,

$$\lim_{t\to+\infty}\sup\frac{1}{t}\mathbb{E}\int_0^t[m_1(S(r)-\frac{N}{\mu})^2+m_2I(r)^2+m_3R(r)^2]dr\leq\delta_1,$$

for some positive constants m_1,m_2,m_3 and δ_1.
If $\mathcal{R}_0>1$, the global positive solution fluctuates randomly around the endemic equilibrium E^*,

$$\lim_{t\to+\infty}\sup\frac{1}{t}\mathbb{E}\int_0^t[n_1(S(r)-S^*)^2+n_2(I(r)-I^*)^2+n_3(R(r)-R^*)^2]dr\leq\delta_2,$$

for some positive constants n_1,n_2,n_3 and δ_2.
Problem (1.2) is a stochastic model driven by white noise only, therefore its solution is continuous. But when encountered with massive diseases like avian influenza, such a disturbance may break the continuity of the solution. Thus the importance of the Lévy noise in the study of the dynamical behavior of the model is of great significance to the prevention and the control of the disease.
In recent works on dynamics of solution to a stochastic model driven by a Lévy noise, one can see [42, 43, 44, 45]. In [42], authors studied a stochastic SIRS model driven by Lévy noise, and investigated the dynamics of the model around the disease-free and endemic equilibria. However SIRI models present some difficulties compared to SIR or SIRS models, since the equation on I cannot be written in the form $\dot{I}(t)=I(t)h(.)$ [34]. The aim of the present work is to study SIRI model, using Lévy noise perturbation to extend the work of [22], which is developed only for a system with a standard Brownian motion as noise disturbance as we described above.
In this paper we are interested in extending the models (1.1) and (1.2) to the

following one, which is driven by Lévy noise,

$$\begin{aligned} dS(t) &= [N-\mu S(t)-\beta S(t)I(t)]dt+\sigma_1 S(t)dW_1(t)+\int_Y q_1(y)S(t-)\tilde{N}(dt,dy),\\ dI(t) &= [(\beta S(t)+\mu+\gamma)I(t)+\delta R(t)]dt+\sigma_2 I(t)dW_2(t)+\int_Y q_2(y)I(t-)\tilde{N}(dt,dy),\\ dR(t) &= [\gamma I(t)-(\mu+\delta)R(t)]dt+\sigma_3 R(t)dW_3(t)+\int_Y q_3(y)R(t-)\tilde{N}(dt,dy). \end{aligned} \tag{1.3}$$

On the foundation of the models (1.1) and (1.2), system (1.3) has been linked to a stochastic perturbation, in which $W_i(t)$ is a standard Brownian motion defined on a complete probability space $(\Omega,\mathcal{F},\mathbb{P})$ with the filtration $(\mathcal{F}_t)_{t\geq 0}$, satisfying the usual conditions, $X(t-)$ is the left limit of $X(t)$, $N(dt,dy)$ is a Poisson counting measure with the stationary compensator $\nu(dy)dt$, $\tilde{N}(dt,dy)=N(dt,dy)-\nu(dy)dt$ and ν is defined on a measurable subset Y of $[0,\infty)$ with $\nu(Y)<\infty$ and $\sigma_i\geq 0$ represent the intensity of $W_i(t)$, $q_i(y)>-1,\ i=1,2,3$. Our study will be as follows: in the second section, we investigate the existence and uniqueness of the global positive solution to model (1.3). The third section is devoted to studying the behavior of solutions to the system (1.3) around the disease-free equilibrium E_0. In the fourth section we study the behavior of solutions around the endemic equilibrium E^* and, in the final part, we present our numerical results supported by real scenarios.

2. Global positive solutions of the system (1.3).

In this section using the same argument as in [43], we will establish the existence of a unique global positive solution for our stochastic epidemic model with relapse and jumps. In what follows, we shall impose two standard assumptions, **(H1)** and **(H2)**, which are essential to prove the existence and uniqueness of a global positive solution of (1.3).

(H1) For each $A>0$ there exists $L_A>0$ such that
$\int_Y |Z_i(x_1,y)-Z_i(x_2,y)|^2\nu(dy)\leq L_A|x_1-x_2|^2$ $i=1,2,3$, with $|x_1|\vee|x_2|\leq A$, where

$$\begin{aligned} Z_1(x,y) &= q_1(y)x \ \ for \ \ x=S(t-),\\ Z_2(x,y) &= q_2(y)x \ \ for \ \ x=I(t-),\\ Z_3(x,y) &= q_3(y)x \ \ for \ \ x=R(t-). \end{aligned}$$

(H2) $|\log(1+q_i(y))|\leq M$ for $q_i(y)>-1$, $i=1,2,3$, where M is a positive constant.

The next theorem ensures the existence and uniqueness of a global positive solution.

Theorem 2.1. *For any given initial value $(S(0),I(0),R(0))\in\mathbb{R}^3_+$, model (1.3) has a unique global solution $(S(t),I(t),R(t))\in\mathbb{R}^3_+$ for all $t\geq 0$ a.s.*

Proof. By **(H1)** and the fact that the drift and the diffusion are locally Lipschitz, 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 τ_e is the explosion time. 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 $m_0>0$ be sufficiently large so that $S(0),I(0),R(0)$ lie within the interval $[\frac{1}{m_0},m_0]$. For each integer $m\geq m_0$, we define the stopping time:

$$\tau_m=\inf\{t\in[0,\tau_e)/\ S(t)\notin(\frac{1}{m},m)\ \ or\ \ I(t)\notin(\frac{1}{m},m)\ \ or\ \ R(t)\notin(\frac{1}{m},m)\},$$

which is increasing as $m \uparrow \infty$. Set $\tau_\infty = \lim_{m\to\infty} \tau_m$, whence, $\tau_\infty \leq \tau_e$ a.s. If we can show that $\tau_\infty = \infty$ is true, then $\tau_e = \infty$ and $(S(t), I(t), R(t)) \in \mathbb{R}^3_+$ a.s. If this statement is false, then there exist two constants $T > 0$ and $0 < \varepsilon < 1$ such that $\mathbb{P}(\tau_\infty \leq T) \geq \varepsilon$. We consider the following function

$$G(S(t), I(t), R(t)) = (S - c - c\log\frac{S}{c}) + (I - 1 - \log I) + (R - 1 - \log R),$$

where c is a positive constant to be determined below. Then by Itô's formula we have

$$\begin{aligned} dG(S(t), I(t)R(t)) &= LGdt + \sigma_1(S-c)dW_1 + \sigma_2(I-1)dW_2 + \sigma_3(R-1)dW_3 \\ &\quad + \int_Y [q_1(y)S - c\log(1+q_1(y))]\tilde{N}(dt, dy) \\ &\quad + \int_Y [q_2(y)I - \log(1+q_2(y))] \\ &\quad + \int_Y [q_3(y)R - \log(1+q_3(y))]\tilde{N}(dt, dy), \end{aligned} \tag{2.1}$$

where

$$\begin{aligned} LG &= (N + 2\mu + \gamma + \delta + c\mu) - (\mu + \beta)S + (c\beta - \mu)I - \mu R - N\frac{c}{S} \\ &\quad -\delta\frac{R}{I} - \gamma\frac{I}{R} + \frac{c\sigma_1^2}{2} + \frac{\sigma_2^2}{2} + \frac{\sigma_3^2}{2} \\ &\quad + \int_Y [cq_1(y) - c\log(1+q_1(y)) + q_2(y) - \log(1+q_2(y))]\nu(dy) \\ &\quad + \int_Y [q_3(y) - \log(1+q_3(y))]\nu(dy). \end{aligned} \tag{2.2}$$

Choosing $c = \frac{\mu}{\beta}$, taking into account that $x - \log(1+x) \geq 0$, $\forall x > -1$, and using **(H2)** we obtain

$$LG \leq N + 2\mu + \gamma + \delta + c\mu + \frac{c\sigma_1^2}{2} + \frac{\sigma_2^2}{2} + \frac{\sigma_3^2}{2} + 3K' = K,$$

where

$$\begin{aligned} K' &= \max\{\int_Y [cq_1(y) - c\log(1+q_1(y))]\nu(dy), \int_Y [q_2(y) - \log(1+q_2(y))]\nu(dy), \\ &\quad \int_Y [q_3(y) - \log(1+q_3(y))]\nu(dy)\}. \end{aligned}$$

Integrating both sides of (2.1) between 0 and $\tau_m \wedge T$ and taking expectation,

$$0 \leq \mathbb{E}[G(S(\tau_m \wedge T), I(\tau_m \wedge T), R(\tau_m \wedge T))] \leq G(S(0), I(0), R(0)) + KT.$$

Define for each $h > 0$, $V(h) := \inf\{G(x_i), x_i \geq h \text{ or } x_i \leq \frac{1}{h}, i = 1, 2, 3\}$,
where $x_1 = S$, $x_2 = I$, $x_3 = R$. We have $\lim_{h\to\infty} V(h) = \infty$.
Therefore,

$$\begin{aligned} G(S(0), I(0), R(0)) + KT &\geq \mathbb{E}[\mathbb{1}_{\{\tau_m \leq T\}} G(S(\tau_m, \omega), I(\tau_m, \omega), R(\tau_m, \omega))] \\ &\geq \varepsilon V(m). \end{aligned}$$

Letting $m \to \infty$, leads to $\infty > G(S(0), I(0), R(0)) + KT = \infty$ and this is a contradiction. Hence the model has a unique global solution $(S(t), I(t), R(t)) \in \mathbb{R}^3_+$ a.s. □

3. **The dynamical properties around the disease-free equilibrium.** In this section we are interested in the behavior of the global positive solutions $(S(t), I(t), R(t))$ around the disease-free equilibrium E_0. The study of the dynamical properties of the solution is investigated in the following theorem. Let

$$\begin{aligned} l_1 &= \frac{2\mu(\mu+\delta)}{\mu+\gamma+\delta} - 2\sigma_1^2 - 6\int_Y q_1^2(y)\nu(dy), \\ l_2 &= \frac{2\mu(\beta-\mu)}{\beta} - \sigma_2^2 - 3\int_Y q_2^2\nu(dy), \\ l_3 &= 2\mu(\frac{\mu+\delta}{\gamma} - \frac{\mu}{\beta}) - (1+\frac{2\mu}{\gamma})\sigma_3^2 - (3+\frac{2\mu}{\gamma})\int_Y q_3^2(y)\nu(dy), \\ M_1 &= \frac{8\delta^2}{\beta} + [2\sigma_1^2 + 6\int_Y q_1^2(y)\nu(dy)](\frac{N}{\mu})^2. \end{aligned}$$

Theorem 3.1. *If $\mathcal{R}_0 \leq 1$ and*

$$\begin{aligned} \frac{\mu(\mu+\delta)}{\mu+\gamma+\delta} &> \sigma_1^2 + 3\int_Y q_1^2(y)\nu(dy), \\ \frac{2\mu(\beta-\mu)}{\beta} &> \sigma_2^2 + 3\int_Y q_2^2(y)\nu(dy), \\ 2\mu(\frac{\mu+\delta}{\gamma} - \frac{\mu}{\beta}) &> (1+\frac{2\mu}{\gamma})\sigma_3^2 + (3+\frac{2\mu}{\gamma})\int_Y q_3^2(y)\nu(dy), \end{aligned}$$

hold true, then for any initial condition $(S(0), I(0), R(0)) \in \mathbb{R}^3_+$ we have

$$\lim_{\tau\to+\infty}\sup\frac{1}{\tau}\mathbb{E}\{\int_0^\tau ((S(t)-\frac{N}{\mu})^2 + I^2(t) + R^2(t))dt\} \leq \frac{M_1}{k},$$

where $k = \min\{l_1, l_2, l_3\}$.

Proof. Let $m(t) = S(t) - \frac{N}{\mu}$; $n(t) = I(t)$; $p(t) = R(t)$, then model (1.3) becomes

$$\begin{aligned} dm(t) &= [-\mu m(t) - \beta m(t)n(t) - \beta\frac{N}{\mu}n(t)]dt + \sigma_1(m(t)+\frac{N}{\mu})dW_1(t) \\ &\quad + \int_Y q_1(y)(m(t-)+\frac{N}{\mu})\tilde{N}(dt,dy), \\ dn(t) &= [\beta m(t)n(t) - (\mu+\gamma-\beta\frac{N}{\mu})n(t) + \delta p(t)]dt + \sigma_2 n(t)dW_2(t), \\ &\quad + \int_Y q_2(y)n(t-)\tilde{N}(dt,dy) \\ dp(t) &= [\gamma n(t) - (\mu+\delta)p(t)]dt + \sigma_3 p(t)dW_3(t) + \int_Y q_3(y)p(t-)\tilde{N}(dt,dy). \end{aligned} \tag{3.1}$$

We consider the following function

$$G(m(t), n(t), p(t)) = (m(t)+n(t)+p(t))^2 + am(t) + bp^2(t),$$

where a, b are two positive constants to be determined below.
Then by Itô's formula we obtain

$$\begin{aligned} dG(m(t),n(t),p(t)) &= LGdt + 2(m+n+p)[\sigma_1(m+\frac{N}{\mu})dW_1 + \sigma_2 dW_2 + \sigma_3 dW_3] \\ &\quad + a\sigma_2 n dW_2 + 2b\sigma_3 p^2 dW_3 \\ &\quad + 2(m+n+p)\int_Y [q_1(y)(m+\frac{N}{\mu}) + q_2(y)n + q_3(y)p]\tilde{N}(dt,dy) \\ &\quad + a\int_Y q_2(y)n\tilde{N}(dt,dy) + 2b\int_Y q_3(y)p^2\tilde{N}(dt,dy) \\ &\quad + \int_Y [(q_1(y)(m+\frac{N}{\mu}) + q_2(y)n + q_3(y)p)^2 + b(q_3(y)p)^2]\tilde{N}(dt,dy), \end{aligned} \tag{3.2}$$

where

$$\begin{aligned} LG &= -2(m+n+p)(-\mu m - \mu n - \mu p) + a[\beta mn - (\mu+\gamma-\frac{N}{\mu})n + \delta p] \\ &\quad + 2bp(\gamma n - (\mu+\delta)p) + \sigma_1^2(m+\frac{N}{\mu})^2 + \sigma_2^2 n^2 + \sigma_3^2(1+b)p^2 \\ &\quad + \int_Y [(q_1(y)(m+\frac{N}{\mu}) + q_2(y)n + q_3(y)p)^2 + b(q_3(y)p)^2]\nu(dy) \\ &= -2\mu m^2 - 2\mu n^2 - [2\mu + 2b(\gamma+\mu)]p^2 + (a\beta - 4\mu)mn + (2b\gamma - 4\mu)np \\ &\quad - 4\mu mp - a(\mu+\gamma-\frac{\beta N}{\mu})n + a\delta p + \sigma_1^2(m+\frac{N}{\mu})^2 + \sigma_2^2 n^2 \\ &\quad + \sigma_3^2(1+b)p^2 + \int_Y [(q_1(y)(m+\frac{N}{\mu}) + q_2(y)n + q_3(y)p)^2 + b(q_3(y)p)^2]\nu(dy) \\ &\le -2\mu m^2 - 2\mu n^2 - [2\mu + 2b(\gamma+\mu)]p^2 + (a\beta - 4\mu)mn + (2b\gamma - 4\mu)np \\ &\quad - 4\mu mp - a(1-\mathcal{R}_0)\frac{\mu(\mu+\gamma+\delta)}{\mu+\delta}n + a\delta n + a\delta p + \sigma_1^2(m+\frac{N}{\mu})^2 + \sigma_2^2 n^2 + \sigma_3^2(1+b)p^2 \\ &\quad + \int_Y [(q_1(y)(m+\frac{N}{\mu}) + q_2(y)n + q_3(y)p)^2 + b(q_3(y)p)^2]\nu(dy). \end{aligned}$$

Choosing $a = \frac{4\mu}{\beta}$, $b = \frac{2\mu}{\gamma}$ and noticing that $-a(1-\mathcal{R}_0)\frac{\mu(\mu+\gamma+\delta)}{\mu+\delta}n \le 0$ because $\mathcal{R}_0 \le 1$, we derive

$$\begin{aligned} LG &\le -2\mu m^2 - 2\mu n^2 - [2\mu + \frac{4\mu}{\gamma}(\gamma+\mu)]p^2 - 4\mu mp \\ &\quad + a\delta n + a\delta p + \sigma_1^2(m+\frac{N}{\mu})^2 + \sigma_2^2 n^2 \\ &\quad + \sigma_3^2(1+b)p^2 + \int_Y [(q_1(y)(m+\frac{N}{\mu}) + q_2(y)n + q_3(y)p)^2 + b(q_3(y)p)^2]\nu(dy). \end{aligned}$$

Using the inequalities $2ab \leq a^2 + b^2$ and $2ab \leq \frac{a^2}{\varepsilon} + \varepsilon^2 b^2$ with $\varepsilon = \frac{\mu+\gamma+\delta}{\gamma}$ and $(a+b+c)^2 \leq 3a^2 + 3b^2 + 3c^2$ we obtain

$$\begin{aligned} LG \quad \leq \quad & -[\frac{2\mu(\mu+\delta)}{\mu+\gamma+\delta} - 2\sigma_1^2 - 6\int_Y q_1^2(y)\nu(dy)]m^2 \\ & -[\frac{2\mu(\beta-\mu)}{\beta} - \sigma_2^2 - 3\int_Y q_2^2\nu(dy)]n^2 \\ & -[2\mu(\frac{\mu+\delta}{\gamma} - \frac{\mu}{\beta}) - (1+\frac{2\mu}{\gamma})\sigma_3^2 + (3+\frac{2\mu}{\gamma})\int_Y q_3^2(y)\nu(dy)]p^2 \\ & +\frac{8\delta^2}{\beta} + [2\sigma_1^2 + 6\int_Y q_1^2(y)\nu(dy)](\frac{N}{\mu})^2. \end{aligned}$$

Then

$$LG \leq -l_1 m^2 - l_2 n^2 - l_3 p^2 + M_1.$$

Integrating both sides of (3.2) between 0 and τ and taking expectation,

$$\begin{aligned} 0 \leq \mathbb{E}(G(m(\tau), n(\tau), p(\tau))) \quad \leq \quad & G(m(0), n(0), p(0)) + M_1\tau \\ & +\mathbb{E}\{\int_0^\tau (-l_1(S(t) - \frac{N}{\mu})^2 - l_2 I^2(t) - l_3 R^2(t))dt\}. \end{aligned}$$

Let $k = \min\{l_1, l_2, l_3\}$, then

$$\mathbb{E}\{\int_0^\tau ((S(t) - \frac{N}{\mu})^2 + I^2(t) + R^2(t))dt\} \leq \frac{G(m(0), n(0), p(0)}{k} + \frac{M_1}{k}\tau.$$

Hence, we conclude that

$$\lim_{\tau\to+\infty} \sup \frac{1}{\tau}\mathbb{E}\{\int_0^\tau ((S(t) - \frac{N}{\mu})^2 + I^2(t) + R^2(t))dt\} \leq \frac{M_1}{k}.$$

□

Remark 1. From the last theorem we see that if $\mathcal{R}_0 \leq 1$, the global positive solutions of (1.3) fluctuate randomly around the disease-free equilibrium. The intensity of those fluctuations is directly related to the values of $q_i(y)$ and σ_i, $i = 1, 2, 3$, which state that the smaller the values of $q_i(y)$ and σ_i are, the nearer the solution is from E_0. From an epidemiological point of view, the epidemic will tend to die out, when the intensity of the stochastic perturbation is small enough. In other words, small stochastic disturbance can lead to extinction of the disease.

4. **The dynamical properties around the endemic equilibrium.** Now we study the behavior of the global positive solution $(S(t), I(t), R(t))$ of the system

(1.3) around the endemic equilibrium E^*. Let

$$\begin{aligned}
l_1 &= \frac{\mu\delta}{\mu+\gamma+\delta} - \sigma_1^2 - 3\int_Y q_1^2(y)\nu(dy),\\
l_2 &= \frac{2\mu+\gamma+\delta}{\mu+\gamma+\delta} - \sigma_2^2 - 3\int_Y q_2^2(y)\nu(dy),\\
l_3 &= \frac{\mu(\mu+\delta)}{\gamma} - (1+\frac{2\mu}{\gamma})\sigma_3^2 - (3+\frac{2\mu}{\gamma})\int_Y q_3^2(y)\nu(dy),\\
M_2 &= (\mu+\gamma+\delta)(I^*)^2 + \frac{\mu(\mu+\gamma+\delta)}{\gamma}(S^*)^2 + \frac{\mu(\mu+\gamma+\delta)}{\delta}(\mu+\delta)^2\\
&\quad +\sigma_1^2(S^*)^2 + \sigma_2^2(I^*)^2 + \sigma_1^3(1+\frac{2\mu}{\gamma})(R^*)^2\\
&\quad +3\int_Y [q_1^2(y)(S^*)^2 + 3q_2^2(y)(I^*)^2 + q_3^2(y)(3+\frac{2\mu}{\gamma})(R^*)^2]\nu(dy).
\end{aligned}$$

Theorem 4.1. *Let $(S(t), I(t), R(t)) \in \mathbb{R}^3_+$ be the solution of the stochastic model (1.3) with initial value $(S(0), I(0), R(0)) \in \mathbb{R}^3_+$. If $\mathcal{R}_0 > 1$ and the following conditions*

$$\frac{\mu\delta}{\mu+\gamma+\delta} > \sigma_1^2 + 3\int_Y q_1^2(y)\nu(dy),$$

$$\frac{2\mu+\gamma+\delta}{\mu+\gamma+\delta} > \sigma_2^2 + 3\int_Y q_2^2(y)\nu(dy),$$

$$\frac{\mu(\mu+\delta)}{\gamma} > (1+\frac{2\mu}{\gamma})\sigma_3^2 + (3+\frac{2\mu}{\gamma})\int_Y q_3^2(y)\nu(dy),$$

are fulfilled, then

$$\lim_{\tau\to\infty}\sup\frac{1}{\tau}\mathbb{E}[\int_0^\tau \{(S-S^*)^2 + (I-I^*)^2 + (R-R^*)^2\}dt] \le \frac{M_2}{k},$$

where (S^*, I^*, R^*) is the endemic equilibrium and $k = \min\{l_1, l_2, l_3\}$.

Proof. We consider the following function

$$G(S(t), I(t), R(t)) = \frac{1}{2}(S-S^*+I-I^*+R-R^*)^2 + u(I-I^*) + \frac{1}{2}v(R-R^*)^2,$$

where u and v are two positive constants to be determined below.
Using Itô's formula,

$$\begin{aligned}
dG &= LGdt + (S-S^*+I-I^*+R-R^*)[\sigma_1 SdW_1 + \sigma_2 IdW_2 + \sigma_3 RdW_3]\\
&\quad +u\sigma_2 IdW_2 + \sigma_3 v(R-R^*)RdW_3 + \int_Y [\frac{1}{2}(q_1(y)S + q_2(y)I + q_3(y)R)^2]\tilde{N}(dt,dy)\\
&\quad + \int_Y [\frac{1}{2}v(q_3(y)R)^2 + uq_2 I + v(R-R^*)q_3(y)R]\tilde{N}(dt,dy)\\
&\quad +(S-S^*+I-I^*+R-R^*)\int_Y [(q_1(y)S + q_2(y)I + q_3(y)R)]\tilde{N}(dt,dy),
\end{aligned} \tag{4.1}$$

where

$$\begin{aligned} LG &= (S-S^*+I-I^*+R-R^*)[N-\beta SI-\mu S+\beta SI-(\mu+\gamma)I+\delta R \\ &\quad +\gamma I-(\mu+\delta)R]+v(R-R^*)[\gamma I-(\mu+\delta)R]+\sigma_1^2S^2+\sigma_2^2I^2 \\ &\quad +(1+v)\sigma_3^2R^2+\int_Y[\frac{1}{2}(q_1(y)S+q_2(y)I+q_3(y)R)^2]\nu(dy) \\ &\quad +\int_Y[+uq_2(y)I+\frac{1}{2}v(q_3(y)R)^2]\nu(dy). \end{aligned} \tag{4.2}$$

Using the fact that

$$\begin{aligned} N &= \mu S^*+\beta S^*I^*, \\ \beta S^*I^* &= (\mu+\gamma)I^*-\delta R^*, \\ \gamma I^* &= (\mu+\delta)R^*, \end{aligned}$$

we deduce

$$\begin{aligned} LG &= -\mu(S-S^*)^2-\mu(I-I^*)^2-[\mu+(\mu+\delta)v](R-R^*)^2 \\ &\quad -2\mu(S-S^*)(R-R^*)+(u\beta-2\mu)(S-S^*)(I-I^*)+(I-I^*)(R-R^*)(\gamma v-2\mu) \\ &\quad +u\beta(S-S^*)I^*+u\beta(I-I^*)S^*-u(\mu+\gamma)(I-I^*) \\ &\quad +\sigma_1^2S^2+\sigma_2^2I^2+(1+v)\sigma_3^2R^2 \\ &\quad +\int_Y[\frac{1}{2}(q_1(y)S+q_2(y)I+q_3(y)R)^2]\nu(dy)+\int_Y[\frac{1}{2}v(q_3(y)R)^2]\nu(dy). \end{aligned} \tag{4.3}$$

Choosing $u=\frac{2\mu}{\beta}$ $v=\frac{2\mu}{\gamma}$, we obtain

$$\begin{aligned} LG &\leq -\mu(S-S^*)^2-\mu(I-I^*)^2-[\mu+(\mu+\delta)\frac{2\mu}{\gamma}](R-R^*)^2 \\ &\quad -2\mu(S-S^*)(R-R^*)+2\mu(S-S^*)I^*+2\mu(I-I^*)S^*-2\mu(\mu+\delta)(I-I^*) \\ &\quad +\frac{1}{2}\sigma_1^2S^2+\frac{1}{2}\sigma_2^2I^2+\frac{1}{2}(1+v)\sigma_3^2R^2 \\ &\quad +\int_Y\frac{1}{2}(q_1(y)S+q_2(y)I+q_3(y)R)^2+\frac{1}{2}v(q_3(y)R)^2\nu(dy). \end{aligned}$$

Using $2ab\leq\frac{a^2}{\varepsilon}+\varepsilon b^2$, where $\varepsilon=\frac{\mu+\gamma+\delta}{\gamma}$, we get

$$\begin{aligned} LG &\leq -(\mu-\frac{\gamma\mu}{\mu+\gamma+\delta})(S-S^*)^2-\mu(I-I^*)^2-[\mu+\frac{2\mu(\mu+\delta)}{\gamma}-\frac{\mu(\mu+\gamma+\delta)}{\gamma}](R-R^*)^2 \\ &\quad +2\mu(S-S^*)I^*+2\mu(I-I^*)S^*-2\mu(\mu+\delta)(I-I^*)+\frac{1}{2}\sigma_1^2S^2+\frac{1}{2}\sigma_2^2I^2 \\ &\quad +\frac{1}{2}(1+v)\sigma_3^2R^2+\int_Y\frac{1}{2}[(q_1(y)S+q_2(y)I+q_3(y)R)^2+v(q_3(y)R)^2]\nu(dy). \end{aligned}$$

Using the inequalities $2ab \leq \frac{a^2}{\varepsilon} + \varepsilon b^2$ and $2ab \leq \frac{a^2}{\varepsilon'} + \varepsilon' b^2$, with $\varepsilon = \frac{\gamma}{\mu+\gamma+\delta}$, $\varepsilon' = \frac{\delta}{\mu+\gamma+\delta}$ and $\varepsilon'' = \frac{\mu}{\mu+\gamma+\delta}$ we obtain

$$\begin{aligned} LG \leq & -(\frac{\mu\delta}{\mu+\gamma+\delta})(S-S^*)^2 - (\frac{2\mu+\gamma+\delta}{\mu+\gamma+\delta}(I-I^*)^2 \\ & -(\frac{\mu(\mu+\delta)}{\gamma})(R-R^*)^2 \\ & +(\mu+\gamma+\delta)(I^*)^2 + \frac{\mu(\mu+\gamma+\delta)}{\gamma}(S^*)^2 + \frac{\mu(\mu+\gamma+\delta)}{\delta}(\mu+\delta)^2 \\ & +\frac{1}{2}\sigma_1^2 S^2 + \frac{1}{2}\sigma_2^2 I^2 + \frac{1}{2}(1+v)\sigma_3^2 R^2 \\ & +\int_Y \frac{1}{2}[(q_1(y)S + q_2(y)I + q_3(y)R)^2 + v(q_3(y)R)^2]\nu(dy). \end{aligned}$$

Taking into account now that $2ab \leq a^2 + b^2$ and $(a+b+c)^2 \leq 3(a^2+b^2+c^2)$,

$$\begin{aligned} LG \leq & -[\frac{\mu\delta}{\mu+\gamma+\delta} - \sigma_1^2 - 3\int_Y q_1^2(y)\nu(dy)](S-S^*)^2 \\ & -[\frac{2\mu+\gamma+\delta}{\mu+\gamma+\delta} - \sigma_2^2 - 3\int_Y q_2^2(y)\nu(dy)](I-I^*)^2 \\ & -[\frac{\mu(\mu+\delta)}{\gamma} - (1+\frac{2\mu}{\gamma})\sigma_3^2 - (3+\frac{2\mu}{\gamma})\int_Y q_3^2(y)\nu(dy)](R-R^*)^2 \\ & +(\mu+\gamma+\delta)(I^*)^2 + \frac{\mu(\mu+\gamma+\delta)}{\gamma}(S^*)^2 + \frac{\mu(\mu+\gamma+\delta)}{\delta}(\mu+\delta)^2 \\ & +\sigma_1^2(S^*)^2 + \sigma_2^2(I^*)^2 + \sigma_1^3(1+\frac{2\mu}{\gamma})(R^*)^2 \\ & +3\int_Y [q_1^2(y)(S^*)^2 + 3q_2^2(y)(I^*)^2 + q_3^2(y)(3+\frac{2\mu}{\gamma})(R^*)^2]\nu(dy). \end{aligned}$$

Integrating both sides of (4.1) between 0 and τ and taking expectation,

$$\begin{aligned} 0 \leq \mathbb{E}[G(S(\tau),I(\tau),R(\tau))] \leq & \; G(S(0),I(0),R(0)) + \tau M_2 \\ & +\mathbb{E}[\int_0^\tau \{-l_1(S-S^*)^2 - l_2(I-I^*)^2 - l_3(R-R^*)^2\}dt]. \end{aligned}$$

Therefore

$$\mathbb{E}[\int_0^\tau \{l_1(S-S^*)^2 + l_2(I-I^*)^2 + l_3(R-R^*)^2\}dt] \leq G(S(0),I(0),R(0)) + \tau M_2.$$

Let $k = \min\{l_1, l_2, l_3\}$. Then

$$\mathbb{E}[\int_0^\tau \{(S-S^*)^2 + (I-I^*)^2 + (R-R^*)^2\}dt] \leq \frac{G(S(0),I(0),R(0))}{k} + \frac{\tau M_2}{k}.$$

Hence, we finally have

$$\lim_{\tau\to\infty} \sup \frac{1}{\tau}\mathbb{E}[\int_0^\tau \{(S-S^*)^2 + (I-I^*)^2 + (R-R^*)^2\}dt] \leq \frac{M_2}{k}.$$

□

Remark 2. From the previous theorem we conclude that if $\mathcal{R}_0 > 1$, the solutions of (1.3) fluctuate around the endemic equilibrium. The intensity of those fluctuations is strongly related to the values of $q_i(y)$ and σ_i $(i = 1, 2, 3)$, which means that the lower the values of $q_i(y)$ and σ_i $(i = 1, 2, 3)$ are, the nearer the solution is from E^*.

From an epidemiological point view, the disease will tend to be persistent when the intensity of the stochastic perturbation is small enough. In other words, small stochastic disturbance can lead to persistence of the disease.

5. **Numerical results.** Numerical solutions of stochastic differential equations [1, 18] are very important in the study of real examples of epidemics. In this section we present simulations corresponding to the theoretical results proved in the previous sections. Around the disease-free equilibria we will consider the following $\sigma_1 = 0.04,\ \sigma_2 = 0.1,\ \sigma_3 = 0.2$ and $q_i(y) = \frac{-k_i y}{1+y^2},\ y = 0.5$, where $k_1 = 0.1,\ k_2 = 0.2,\ k_3 = 0.3$. Around the endemic equilibria we will choose $\sigma_1 = 0.04,\ \sigma_2 = 0.03, \sigma_3 = 0.02$ and $q_i(y) = \frac{-k_i y}{1+y^2},\ y = 0.5$, where $k_1 = 0.1,\ k_2 = 0.2,\ k_3 = 0.3$.

Example 5.1. This example represents a simulation for transmission dynamics and elimination potential of zoonotic tuberculosis in Morocco [27]. The average lifespan of the Moroccan cattle is 6 years which yields to a death rate of $\mu = 0.167$ per year. From the data on cattle population using least squares the birth rate was estimated 0.177 per year. The cattle to cattle transmission rate of bovine tuberculosis was estimated from the endemic prevalence in cattle to $\beta = 0.249$. We choose $\delta = 0.1$ and $\gamma = 0.2$ and get $\mathcal{R}_0 = 0.903 < 1$. Then, thanks to Theorem 3.1, solutions of (1.3) fluctuate around the disease-free equilibrium. Figure 1 is an illustration of the trajectories of the solutions to models (1.1) and (1.3) using the parameters cited above.

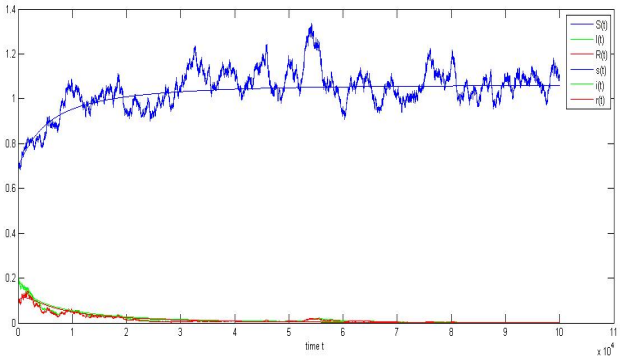

FIGURE 1. Trajectories of the solutions to the systems (1.1) and (1.3) for Moroccan zoonotic tuberculosis with $\mathcal{R}_0 \leq 1$.

Example 5.2. This example is motivated by bovine tuberculosis in a cattle herd, with time unit of one year [5]. We consider the following parameters $N = \mu = 0.1$, $\gamma = \delta = 0.5$ (equal periods of infection and recovery before relapse) $\beta = 0.6$ to obtain $\mathcal{R}_0 = 3.27$. Since $\mathcal{R}_0 > 1$, it follows from Theorem 4.1 that solutions of (1.3) fluctuate around the endemic equilibrium. Figure 2 shows trajectories of the solutions to (1.1) and (1.3) using these parameters.

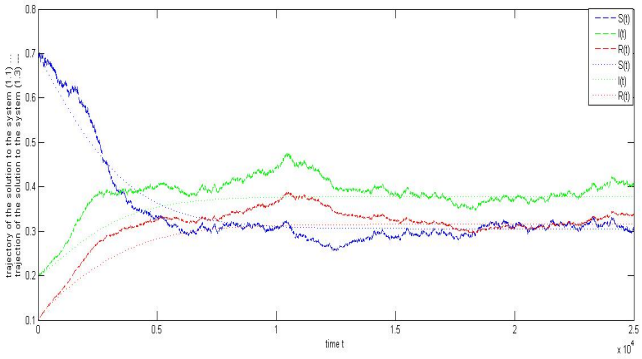

FIGURE 2. Trajectories of the solutions to the systems (1.1) and (1.3) for bovine tuberculosis [5] with $\mathcal{R}_0 > 1$, $N = \mu = 0.1$, $\beta = 0.6$ *and* $\gamma = \delta = 0.5$.

To get insight on the appropriate intervention strategies to prevent and control the spread of the disease, we show next the influence of some parameters on the dynamical behaviors of the stochastic model using sensitivity analysis.

Example 5.3. To determine how changes in relapse rate affect the spread of the disease, we examine the sensitivity index of the basic reproduction number, $\mathcal{R}_0$ with respect to the relapse parameter δ. The relapse sensitivity parameter is given by

$$\frac{\partial \mathcal{R}_0}{\partial \delta}\frac{\delta}{\mathcal{R}_0} = \frac{\gamma\delta}{(\mu+\delta)(\mu+\gamma+\delta)},$$

which has a positive sign. This means that an increase in the value of the relapse parameter will lead to an increase in $\mathcal{R}_0$ and asymptotically results into persistence of the disease in the population. The following three figures represent a variation of the relapse parameter. Let $N = 0.4$, $\mu = 0.3$, $\beta = 0.8$ and $\gamma = 0.2$. From Figure 3, we remark that increasing the relapse rate increases the magnitude of infected individuals.

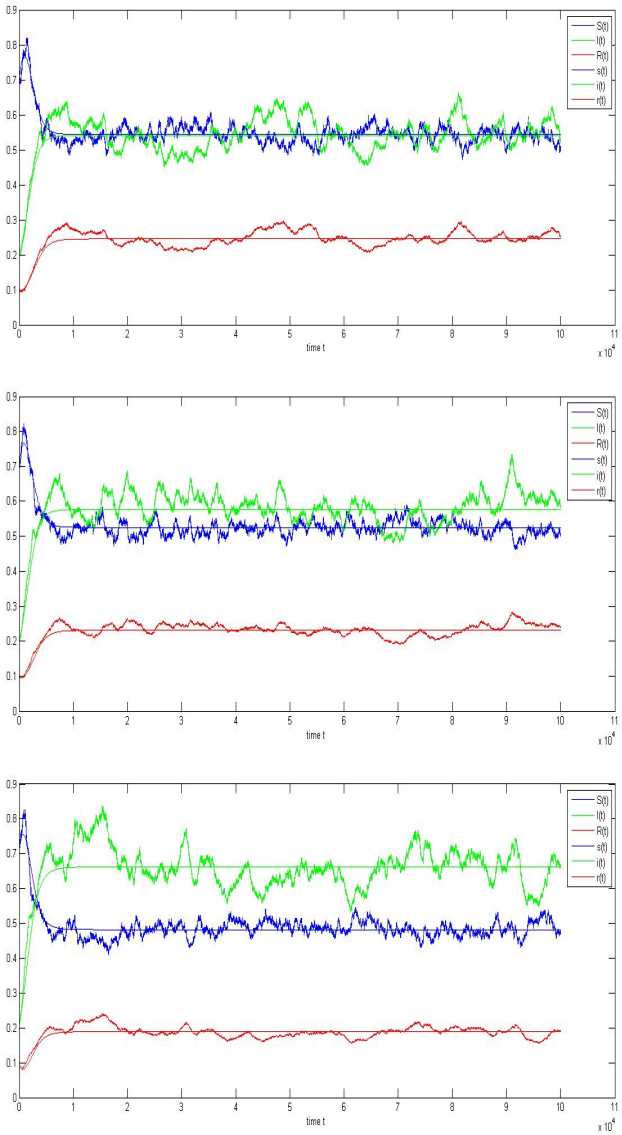

FIGURE 3. Trajectories of the solutions to the systems (1.1) and (1.3) with various relapse rate δ: $0.14, 0.2, 0.4$.

Example 5.4. The recovery sensitivity parameter is given by

$$\frac{\partial \mathcal{R}_0}{\partial \gamma}\frac{\gamma}{\mathcal{R}_0} = -\frac{\gamma}{\mu+\gamma+\delta}.$$

The negative sign of the sensitivity index shows that an increase in the value of the recovery parameter γ will lead to a decrease in $\mathcal{R}_0$ and asymptotically results into extinction of the disease in the population. The next figures illustrate a variation of the recovery rate, here with $N = 0.4$, $\mu = 0.3$, $\beta = 0.8$ and $\delta = 0.1$. From Figure

4, we remark that increasing the recovery rate, decreases the magnitude of infected individuals.

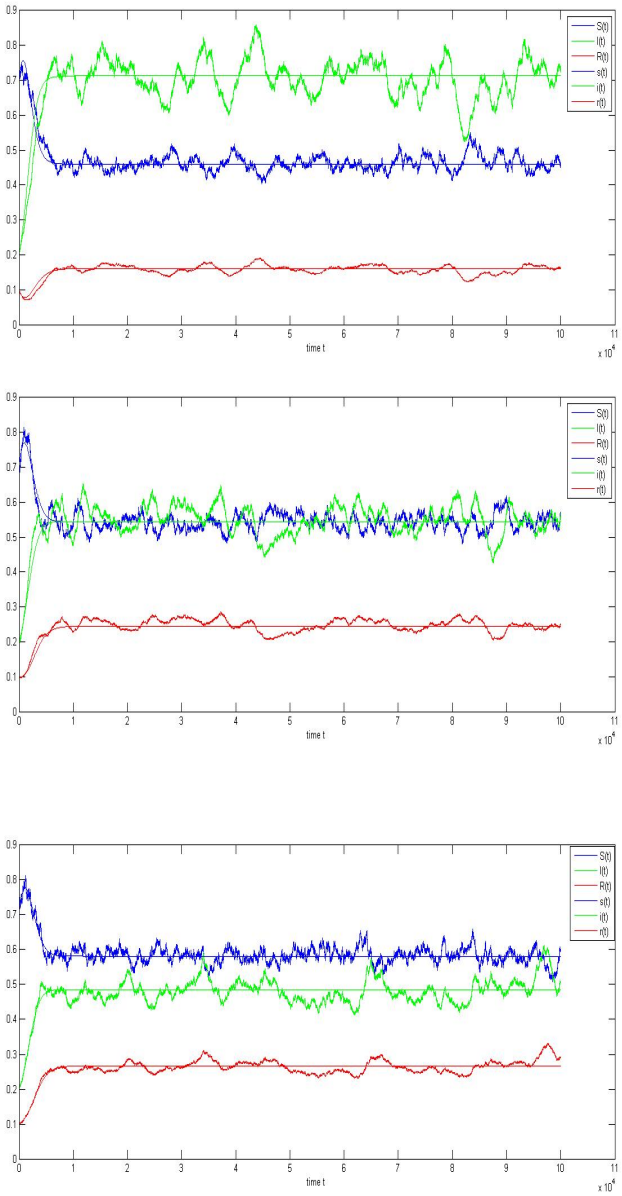

FIGURE 4. Trajectories of the solutions to the systems (1.1) and (1.3) with a various recovery rate γ: $0.09, 0.18, 0.22$

Conclusion. The jumps play a significant role in evolution of many real dynamical processes, including the case of epidemic spreading when encountered with massive diseases like avian influenza. In this work we have established the existence and the uniqueness of a global positive solution for a stochastic SIRI epidemic model driven by Lévy noise and studied its dynamical behavior. Using Lyapunov techniques we showed that the solution fluctuates around the equilibria under suitable conditions. Furthermore, we have illustrated from numerical results the changing effect of the relapse rate and recovery rate on the size of infectious individuals. Increasing the value of the relapse rate increases the basic reproduction number and, consequently, the magnitude of the infected individuals in the population. Per contra, increasing the value of the recovery rate decreases the basic reproduction number and also the magnitude of the infected individuals. In this way, it is pertinent to conclude that efforts should be encouraged in order to achieve a disease-free population. In a future coming work we will focus on stochastic model with vaccination.

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