ANALYSIS OF A STOCHASTIC RECOVERY-RELAPSE EPIDEMIC MODEL WITH PERIODIC PARAMETERS AND MEDIA COVERAGE*

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Abstract This paper addresses, motivated by mathematical work on infectious disease models, the impacts of environmental noise and media coverage on the dynamics of recovery-relapse infectious diseases. A susceptibleinfectious-recovered-infectious model is formulated with both vertical transmission and horizontal transmission. The existence and uniqueness of the positive global solution is studied by constructing suitable Lyapunov-type function. Then, the existence of positive periodic solutions is verified by applying Khasminskii's theory. The existence of positive periodic solutions indicates the continued survival of the diseases. Besides, sufficient conditions for the extinction of the diseases are obtained. Numerical simulations then demonstrate the dynamics of the solutions. The paper extends the results of the corresponding deterministic system.

Keywords Stochastic epidemic model, media coverage, periodic solution, vertical transmission, recovery-relapse.

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1. Introduction

The last decades have witnessed a number of major outbreaks of infectious diseases, including dengue fever, tuberculosis, H7N9 virus, and most recently, Zika virus. Worldwide more than 3.9 billion people in 128 countries are at risk of infection with dengue viruses [2]. In 2016, 10.4 million people fell ill with tuberculosis, and 1.7 million died from the disease (WHO,2016). Infectious diseases are responsible for a quarter of all deaths in the world each year, so diminishing their impact is a global priority.

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In recent decades, a substantial number of mathematical modelling approaches have been applied to model different types of infectious diseases [7, 15, 19, 20, 24, 28, 33, 34, 41, 42]. It is now widely recognised that the classical SIS and SIR compartmental structures are two fundamental frameworks in modelling the spread of infectious diseases. Using these fundamental frameworks, a number of researchers have investigated the spread rule of infectious diseases including smallpox, influenza, and measles [13, 18]. However, for certain infectious diseases such as herpes simplex virus, hepatitis, and tuberculosis, individuals who have been recovered may relapse with reactivation of latent infection and revert to the infective class. To this end, Tudor [37] developed a SILI compartmental structure for this type of infectious diseases. He summarised the stability characteristics of the equilibrium points. More recently, Driessche and Zou [38] proposed an integrodifferential equation to model the general relapse phenomenon in infectious diseases, who identified the basic reproduction number for the model and established the threshold property of the basic reproduction number.

Most of the work mentioned above only takes into account the horizontal transmission of diseases, ignoring the possible importance of vertical transmission (transmission directly from the mother to an embryo, fetus, or baby during pregnancy or childbirth). However, infectious diseases including tuberculosis, herpes simplex virus, and HIV can be passed from mother to child [26,35]. Therefore many scholars have studied the effect of mother-to-child transmission on epidemic models and many good results have been obtained, see [4,21] and the references therein.

Another essential aspect in disease modelling is the incidence rate, which is considered to play a vital role in ensuring that the model can give a reasonable qualitative description of the disease dynamics [11,17,23]. In most classical disease transmission models, the incidence rate is assumed to be bilinear incidence given by βSI (where β is the transmission rate, S is the susceptibles, I is the infected) [1]. However, with the progress of information technology, media coverage (e.g., television, radio, and newspaper) is becoming more and more important in the prevention and control of infectious diseases [25, 36]. When the media coverage is intervened, the contact rate may reduce if people know about the transmission way from media and then reduce their contact with others.

Keeping all the above work in mind, we divide the total population of infectious diseases with relapse into three classes, namely the uninfected susceptibles (S), the infected who are in an infectious virus-shedding state (I) and the infected who are in a non-infectious state (L). Then we obtain the following SILI model with vertical transmission and media coverage:

$$\begin{pmatrix}
\frac{dS}{dt} = \mu - [\beta_1 - \beta_2 f(I)]SI - \mu S - \mu(1 - \alpha)I, \\
\frac{dI}{dt} = [\beta_1 - \beta_2 f(I)]SI - (\alpha \mu + \gamma)I + \lambda L, \\
\frac{dL}{dt} = \gamma I - (\mu + \lambda)L,
\end{cases}$$
(1.1)

where μ is the recruitment rate of individuals, which we suppose equal to the natural mortality rate of individuals, α is the rate of horizontal transmission and $1 - \alpha$ is the rate of vertical transmission. γ is the recovery rate of I and λ is the relapse rate. β_1 is the usual contact rate without considering the infective individuals and β_2 ($< \beta_1$) is the maximum reduced contact rate due to the presence of the infected individuals. The function f(I) satisfies f(0) = 0, $f'(I) \ge 0$ and $\lim_{I_t\to\infty} f(I) = 1$ (see Ref. [6] for more biological meanings). The transfer diagram of the SILI system is shown in Figure 1.



Figure 1. Transfer diagram of the SILI system (1.1)

One drawback of the deterministic system (1.1) is that it ignores the effects of environmental factors on the transmission of diseases. Environmental factors have a significant role in shaping the dynamics of infectious diseases [9,14,29,31,39]. Britton and Traoré [3] indicated that environmental factors could be significant during the period immediately after the introduction of the infection into a population. Dalal et al. [8] stated that stochastic differential equations could be used to study the spread of infectious diseases where environmental factors took place.

From a mathematical point of view, there are many ways to consider environmental factors into the study of infectious disease dynamics. For one thing, parameters such as effective contact rates that affect the dynamics of disease transmission are subject to small random perturbations (e.g., rainstorms and droughts), which are often described by white noise [10, 12]. For another, seasonal changes in environmental factors such as temperature and rainfall may lead to the periodic variation of parameters affecting the spread of diseases. In mathematics, these seasonal changes can be described using periodic functions [32, 40]. In this paper, we study an epidemic model that includes both small random perturbations and seasonal factors explicitly.

In general, depending on the case in question, the parameters affected by random noise may be different. In this paper, we assume that the contact coefficients β_i are affected by the stochastic noise. We replace the contact coefficients β_i with

$$\beta_i + \sigma_i \dot{B}_{i,t}, \ i = 1, 2,$$

where $B_{i,t}$ are standard Brownian motions with intensities σ_i . To consider the influence of periodic factors on the dynamics of infectious diseases, we assume that the coefficients $\mu, \beta_i, \sigma_i, \alpha, \gamma, \lambda$ are positive and continuous *T*-periodic functions. These are standard techniques in stochastic and periodic modelling [5, 30]. With these specifics, we reach:

$$\begin{cases} dS_t = [\mu_t - (\beta_{1,t} - \beta_{2,t}f(I_t))S_tI_t - \mu_tS_t - \mu_t(1 - \alpha_t)I_t]dt \\ -\sigma_{1,t}S_tI_tdB_{1,t} + \sigma_{2,t}f(I_t)S_tI_tdB_{2,t}, \\ dI_t = [(\beta_{1,t} - \beta_{2,t}f(I_t))S_tI_t - (\alpha_t\mu_t + \gamma_t)I_t + \lambda_tL_t]dt \\ +\sigma_{1,t}S_tI_tdB_{1,t} - \sigma_{2,t}f(I_t)S_tI_tdB_{2,t}, \\ dL_t = [\gamma_tI_t - (\mu_t + \lambda_t)L_t]dt. \end{cases}$$
(1.2)

The primary goal of this paper is to analyse the dynamics of a stochastic nonautonomous SILI system with vertical transmission and media coverage. The rest of the paper is organized as follows. In Section 2, we present some primary results on stochastic differential equations. In Section 3, we perform an analysis of the existence and uniqueness of the positive global solution. In Section 4, we verify the existence of a positive periodic solution to the model by using Khasminskii's theory. In Section 5, we obtain sufficient conditions for the extinction of the disease. In Section 6, some numerical simulations are carried out to illustrate the theoretical results. Finally, some concluding remarks are presented.

2. Preliminaries

Throughout this paper, let $(\Omega, \mathcal{F}, \mathbb{P})$ be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ satisfying the usual conditions. U is a given open set in the l-dimensional Euclidean space \mathbb{R}^l . $L^p([0,T];\mathbb{R}^d)$ is the family of \mathbb{R}^d -valued \mathcal{F}_t adapted processes $\{x(t)\}_{a\leq t\leq b}$ such that $\int_a^b |x(t)|^p dt < \infty$ almost surely (a.s.), and $L^p(\mathbb{R}_+;\mathbb{R}^d)$ denotes the family of processes $\{x(t)\}_{0\leq t}$ such that for every T > 0, $\{x(t)\}_{0\leq t\leq T} \in L^p([0,T];\mathbb{R}^d)$. $I = \{t: 0 \leq t < \infty\}$ and $E = I \times \mathbb{R}^l$. \mathcal{C}^2 is the family functions on E which are twice continuously differentiable with respect to $x \in \mathbb{R}^l$ and continuously differentiable with respect to $t \in I$. Let f_t be an integrable function on I, we denote $\langle f \rangle_t = \frac{1}{t} \int_0^t f(s) ds$, $\check{f} = \sup_{t \in I} f(t)$ and $\hat{f} = \inf_{t \in I} f(t)$.

Definition 2.1 ([22]). A stochastic process $x(t, \omega)$ is said to be periodic with period T if its finite dimensional distributions are periodic with period T, i.e., for any positive integer m and for any finite sequence of numbers t_1, \ldots, t_m , the joint distributions of the random variables $x(t_{1+kT}, \omega), \ldots, x(t_{m+kT}, \omega)$ are independent of $k, k = \pm 1, \pm 2, \ldots$

Consider the following equation:

$$X(t) = X(0) + \int_{t_0}^t b(s, X(s)) ds + \sum_{r=1}^k \int_{t_0}^t \sigma_r(s, X(s)) dB_r(s),$$
(2.1)

where the vectors b(s, x), $\sigma_1(s, x)$,..., $\sigma_k(s, x)$ $s \in [t_0, T]$, $x \in \mathbb{R}^l$ are continuous functions of (s, x) such that for some constant B the following conditions hold in the entire domain of definition:

$$|b(s,x) - b(s,y)| + \sum_{r=1}^{k} |\sigma_r(s,x) - \sigma_r(s,y)| \le B|x-y|,$$

$$|b(s,x)| + \sum_{r=1}^{k} |\sigma_r(s,x)| \le B(1+|x|).$$
(2.2)

Lemma 2.1 ([22]). Suppose that the coefficients of (2.1) are T-periodic in t and satisfy (2.2) in every cylinder $I \times U$, and suppose further that there exists a non-negative function $\mathcal{V} \in C^2$ in E which T-periodic in t, and satisfies the following conditions:

- (i) $\inf_{|x|>\mathbf{R}} \mathcal{V}(x,t) \to \infty \quad as \quad \mathbf{R} \to \infty;$
- (ii) $\mathcal{LV}(x,t) \leq -1$ outside some compact set,

where $a_{ij} = \sum_{r=1}^{k} \sigma_r^i(x,t) \sigma_r^j(x,t)$ and the operator \mathcal{L} is given by

$$\mathcal{L} = \frac{\partial}{\partial t} + \sum_{i=1}^{l} b_i(x,t) \frac{\partial}{\partial x_i} + \frac{1}{2} \sum_{i,j=1}^{l} a_{ij}(x,t) \frac{\partial^2}{\partial x_i \partial x_j}.$$

Then there exists a solution of (2.1) which is a T-periodic Markov process.

Remark 2.1. It follows from (1.2) that the total population satisfies

$$\frac{d(S_t + I_t + L_t)}{dt} = \mu_t - \mu_t (S_t + I_t + L_t), \ t \in (0, \infty)$$

We can easily check that

$$\Gamma_1 = \{(S, I, L) \in \mathbb{R}^3_+ : S + I + L = 1\}$$
 a.s.

is positively invariant of the system (1.2) given $S_0 + I_0 + L_0 = 1$. Therefore, we consider only the following system:

$$\begin{cases} dI_t = [(\beta_{1,t} - \beta_{2,t}f(I_t))(1 - I_t - L_t)I_t - (\alpha_t\mu_t + \gamma_t)I_t + \lambda_tL_t]dt \\ + \sigma_{1,t}(1 - I_t - L_t)I_t dB_{1,t} - \sigma_{2,t}f(I_t)(1 - I_t - L_t)I_t dB_{2,t}, \\ dL_t = [\gamma_tI_t - (\mu_t + \lambda_t)L_t]dt, \end{cases}$$
(2.3)

where $1 - I_t - L_t = S_t$. It follows that

$$\Gamma_2 = \{ (I, L) \in \mathbb{R}^2_+ : 0 \le I + L < 1 \} \quad a.s$$

is positively invariant of the system (2.3). In the following, we assume that (I_t, L_t) is the solution of model (2.3) with initial value $(I_0, L_0) \in \Gamma_2$.

3. Existence and uniqueness of the positive solution

For the epidemic dynamical system (2.3), variables I_t and L_t should remain nonnegative for $t \ge 0$. We first prove that this is actually the case and furthermore, the positive solution is unique.

Theorem 3.1. For any given initial value $(I_0, L_0) \in \mathbb{R}^2_+$, there is a unique solution (I_t, L_t) of the system (2.3) on $t \ge 0$, and the solution (I_t, L_t) will remain in \mathbb{R}^2_+ with probability one.

Proof. Since the coefficients are locally Lipschitz continuous, the system (2.3) has a unique local solution (I_t, L_t) on $t \in [0, \tau_e)$, where τ_e is the explosion time. Now we need to verify that $\tau_e = \infty$ a.s. Let $k_0 > 0$ be sufficiently large such that I_0 and L_0 all lie within the interval $[\frac{1}{k_0}, k_0]$. For any $k \ge k_0$ $(k \in \mathbb{N}_+)$, define the stopping time

$$\tau_k = \inf \{t \in [0, \tau_e] : \min\{I_t, L_t\} \le 1/k \text{ or } \max\{I_t, L_t\} \ge k\}.$$

Clearly, τ_k is increasing as $k \to \infty$. Set $\tau_{\infty} = \lim_{k \to \infty} \tau_k$ then we have $\tau_{\infty} \leq \tau_e \ a.s.$ Now we need to verify $\tau_{\infty} = \infty \ a.s.$ If this statement is false, there exist two constants T > 0 and $\epsilon \in (0, 1)$ such that $\mathbb{P}\{\tau_{\infty} \leq T\} > \epsilon$. Hence, there exists an integer $k_1 \geq k_0$ such that

$$\mathbb{P}\{\tau_k \le T\} > \epsilon, \quad k \ge k_1. \tag{3.1}$$

Define a \mathcal{C}^2 -function $\mathcal{V}(I_t, L_t)$ by

$$\mathcal{V}(I_t, L_t) = I_t - 1 - \ln I_t + L_t - 1 - \ln L_t.$$

Using Itô formula we obtain that

$$d\mathcal{V}(I_t, L_t) = \mathcal{L}\mathcal{V}(I_t, L_t)dt + \sigma_{1,t}(1 - I_t - L_t)(I_t - 1)dB_{1,t} - \sigma_{2,t}f(I_t)(1 - I_t - L_t)(I_t - 1)dB_{2,t},$$
(3.2)

where

$$\begin{split} \mathcal{LV}(I_t, L_t) &= \left(1 - \frac{1}{I_t}\right) \left[(\beta_{1,t} - \beta_{2,t} f(I_t))(1 - I_t - L_t)I_t - (\alpha_t \mu_t + \gamma_t)I_t + \lambda_t L_t \right] \\ &+ \frac{1}{2} \sigma_{1,t}^2 (1 - I_t - L_t)^2 + \frac{1}{2} \sigma_{2,t}^2 f^2 (I_t)(1 - I_t - L_t)^2 \\ &+ \left(1 - \frac{1}{L_t}\right) \left[\gamma_t I_t - (\mu_t + \lambda_t)L_t \right] \\ &\leq \check{\beta}_1 + \check{\alpha} \check{\mu} + \check{\mu} + 2\check{\lambda} + 2\check{\gamma} + \frac{1}{2} \check{\sigma}_1^2 + \frac{1}{2} \check{\sigma}_2^2 := M. \end{split}$$

Integrating (3.2) from 0 to $\tau_k \wedge T$ and taking expectation on both sides we have that

$$\mathbb{E}\mathcal{V}(I_{\tau_k\wedge T}, L_{\tau_k\wedge T}) \le \mathcal{V}(I_0, L_0) + MT.$$
(3.3)

Set $\Omega_k = \{\tau_k \leq T\}$ for $k \geq k_1$, it then follows from (3.1) that $\mathbb{P}(\Omega_k) \geq \epsilon$. Note that for every $\omega \in \Omega_k$, there exists at least one of $I_{\tau_k}(\omega), L_{\tau_k}(\omega)$ equals either k or $\frac{1}{k}$. As a result, we have

$$\mathcal{V}(I_{\tau_k}(\omega), L_{\tau_k}(\omega)) \ge (k - 1 - \ln k) \wedge \left(\frac{1}{k} - 1 - \ln \frac{1}{k}\right).$$
(3.4)

It follows from (3.3) and (3.4) that

$$\begin{aligned} \mathcal{V}(I_0, L_0) + MT \geq & \mathbb{E}[1\Omega_k(\omega)V(I_{\tau_k}, L_{\tau_k})] \\ \geq & \epsilon \left[(k-1-\ln k) \wedge \left(\frac{1}{k} - 1 - \ln \frac{1}{k}\right) \right], \end{aligned}$$

where $1\Omega_k$ is the indicator function of Ω_k . Letting $k \to \infty$ leads to the contradiction that $\infty > \infty$. So we have $\tau_{\infty} = \infty$ a.s. This completes the proof.

4. Existence of the positive periodic solution

Theorem 4.1. The system (2.3) admits a nontrivial positive T-periodic solution if

$$\mathscr{R}_{1} = \frac{\langle \beta_{1} - \beta_{2} \rangle_{T}}{\left\langle \alpha \mu + \gamma - \frac{\lambda \gamma}{\mu + \lambda} + \frac{1}{2} \sigma_{1}^{2} + \frac{1}{2} \sigma_{2}^{2} \right\rangle_{T}} > 1,$$

which indicates that the diseases will persist.

Proof. Since the coefficients of the system (2.3) are continuous bounded positive T-periodic functions, the coefficients satisfy (2.2). To prove Theorem 4.1, it suffices to find a C^2 -function \mathcal{V} and a closed set $U \in \Gamma_2$ such that the conditions (i) and (ii) in Lemma 2.1 are hold.

Define a \mathcal{C}^2 -function $\mathcal{V}: [0,\infty) \times \mathbb{R}^2 \to \mathbb{R}$ by

$$\begin{aligned} \mathcal{V} &= M(-\ln I_t - m_1 \ln L_t + m_2 L_t + \omega_t) - \ln(1 - I_t - L_t) - \ln L_t \\ &=: M(\mathcal{V}_1 + \omega_t) + \mathcal{V}_2, \end{aligned}$$

where $m_1 = \frac{\langle \lambda \gamma \rangle_T}{\langle \mu + \lambda \rangle_T^2}$, $m_2 = \frac{\check{\beta}_1}{\hat{\mu} + \check{\lambda}}$, M is a positive constant and ω_t is a T-periodic function which will be defined later. It is easy to verify that

$$\liminf_{k \to \infty, (I,L) \in \Gamma_2 \setminus U_k} \mathcal{V}(t,I,L) = \infty,$$

where $U_k = \{(I,L) : 1/k < I + L < 1 - 1/k\}$. Hence $\mathcal{V}(t,I,L)$ is T-periodic in t and satisfies (i) in Lemma 2.1.

Applying Itô formula to \mathcal{V}_1 , we have that

$$\begin{aligned} \mathcal{LV}_{1} &= -\left(\beta_{1,t} - \beta_{2,t}f(I_{t})\right)(1 - I_{t} - L_{t}) + \left(\alpha_{t}\mu_{t} + \gamma_{t}\right) - \lambda_{t}\frac{L_{t}}{I_{t}} - m_{1}\gamma_{t}\frac{I_{t}}{L_{t}} \\ &+ \frac{1}{2}\sigma_{1,t}^{2}(1 - I_{t} - L_{t})^{2} + \frac{1}{2}\sigma_{2,t}^{2}f^{2}(I_{t})(1 - I_{t} - L_{t})^{2} + m_{1}(\mu_{t} + \lambda_{t}) \\ &+ m_{2}[\gamma_{t}I_{t} - (\mu_{t} + \lambda_{t})L_{t}] \\ &\leq -\lambda_{t}\frac{L_{t}}{I_{t}} - m_{1}\gamma_{t}\frac{I_{t}}{L_{t}} - \beta_{1,t} + \beta_{2,t} + \alpha_{t}\mu_{t} + \gamma_{t} + \frac{1}{2}\sigma_{1,t}^{2} + \frac{1}{2}\sigma_{2,t}^{2} + m_{1}(\mu_{t} + \lambda_{t}) \\ &+ (\beta_{1,t} + m_{2}\gamma_{t})I_{t} + [\beta_{1,t} - m_{2}(\mu_{t} + \lambda_{t})]L_{t} \\ &\leq -2\sqrt{m_{1}\lambda_{t}\gamma_{t}} - \beta_{1,t} + \beta_{2,t} + \alpha_{t}\mu_{t} + \gamma_{t} + \frac{1}{2}\sigma_{1,t}^{2} + \frac{1}{2}\sigma_{2,t}^{2} \\ &+ m_{1}(\mu_{t} + \lambda_{t}) + (\check{\beta}_{1} + m_{2}\check{\gamma})I_{t} \\ &=:\psi_{t} + (\check{\beta}_{1} + m_{2}\check{\gamma})I_{t}. \end{aligned}$$

$$(4.1)$$

Define a T-periodic function ω_t satisfying

$$\omega_t' = \langle \psi \rangle_T - \psi_t, \ \omega_0 = 0.$$

It then follows from (4.1) that

$$\mathcal{L}(\mathcal{V}_1 + \omega_t) \leq \langle \psi \rangle_T + (\check{\beta}_1 + m_2 \check{\gamma}) I_t = - \langle \phi \rangle_T \left(\mathscr{R}_1 - 1 \right) + (\check{\beta}_1 + m_2 \check{\gamma}) I_t,$$
(4.2)

where

$$\phi_t = \alpha_t \mu_t + \gamma_t - \frac{\lambda_t \gamma_t}{\mu_t + \lambda_t} + \frac{1}{2} \sigma_{1,t}^2 + \frac{1}{2} \sigma_{2,t}^2.$$

Similarly, we have

$$\mathcal{LV}_{2} = -\left[\frac{\mu_{t}}{1 - I_{t} - L_{t}} - (\beta_{1,t} - \beta_{2,t}f(I_{t}))I_{t} - \mu_{t} - \mu_{t}(1 - \alpha_{t})\frac{I_{t}}{1 - I_{t} - L_{t}}\right] + \frac{1}{2}\sigma_{1,t}^{2}I_{t}^{2} + \frac{1}{2}\sigma_{2,t}^{2}f^{2}(I_{t})I_{t}^{2} - \frac{\gamma_{t}I_{t}}{L_{t}} + \mu_{t} + \lambda_{t}$$

$$\leq -\frac{\hat{\mu}(1 - I_{t})}{1 - I_{t} - L_{t}} - \frac{\hat{\mu}\hat{\alpha}I_{t}}{1 - I_{t} - L_{t}} - \frac{\hat{\gamma}I_{t}}{L_{t}} + C,$$
(4.3)

where $C = \check{\beta}_1 + 2\check{\mu} + \check{\lambda} + \frac{1}{2}\check{\sigma}_1^2 + \frac{1}{2}\check{\sigma}_2^2$. In view of (4.2) and (4.3), one obtains that

$$\mathcal{LV} \leq -M \langle \phi \rangle_T \left(\mathscr{R}_1 - 1 \right) + M (\check{\beta}_1 + m_2 \check{\gamma}) I_t - \frac{\hat{\mu}(1 - I)}{1 - I_t - L_t} - \frac{\hat{\mu} \hat{\alpha} I_t}{1 - I_t - L_t} - \frac{\hat{\gamma} I_t}{L_t} + C.$$

$$(4.4)$$

Since $\varepsilon > 0$ is taken arbitrarily, we can choose M > 0 sufficiently large such that

$$M(\check{\beta}_1 + m_2\check{\gamma})\varepsilon - M \langle \phi \rangle_T (\mathscr{R}_1 - 1) + C < -1$$
$$M(\check{\beta}_1 + m_2\check{\gamma}) - \frac{\hat{\gamma}}{\varepsilon} + C < -1,$$
$$M(\check{\beta}_1 + m_2\check{\gamma}) - \frac{\hat{\mu}\min\{1, \hat{\alpha}\}}{\varepsilon} + C < -1.$$

Define the compact subset U as

$$U = \left\{ (I, L) \in \Gamma_2 : \varepsilon \leq I, \varepsilon^2 \leq L, I + L \leq 1 - \varepsilon \right\}.$$

Let us divide the complementary set of U into the following three parts:

$$U^c = \bigcup_{i=1}^3 U_i^c,$$

where $(I_t, L_t) \in \Gamma_2$ in every part of U and

$$\begin{split} U_1^c &= \left\{ (I,L) \in \Gamma_2 : \ 0 < I < \varepsilon \right\}, \\ U_2^c &= \left\{ (I,L) \in \Gamma_2 : \ \varepsilon \leq I < 1, \ 0 < L < \varepsilon^2 \right\}, \\ U_3^c &= \left\{ (I,L) \in \Gamma_2 : \ 1 - \varepsilon < I + L \right\}. \end{split}$$

In the following, we will analyze the range of differential operators $\mathcal{LV}(I_t, L_t)$ on each domain.

Case 1: $(I, L) \in U_1^c$. In view of (4.4), we have

$$\mathcal{LV} \leq -M \langle \phi \rangle_T (\mathscr{R}_1 - 1) + M(\check{\beta}_1 + m_2 \check{\gamma}) I_t + C$$

$$\leq -M \langle \phi \rangle_T (\mathscr{R}_1 - 1) + M(\check{\beta}_1 + m_2 \check{\gamma}) \varepsilon + C.$$
(4.5)

Case 2: $(I, L) \in U_2^c$. By using (4.4) one obtains that

$$\mathcal{LV} \leq M(\check{\beta}_1 + m_2\check{\gamma})I_t - \frac{\hat{\gamma}I_t}{L_t} + C$$

$$\leq M(\check{\beta}_1 + m_2\check{\gamma}) - \frac{\hat{\gamma}}{\varepsilon} + C.$$
(4.6)

Case 3: $(I, L) \in U_3^c$. It follows from (4.4) that we have

$$\mathcal{LV} \leq M(\check{\beta}_{1} + m_{2}\check{\gamma})I_{t} - \frac{\hat{\mu}(1 - I_{t})}{1 - I_{t} - L_{t}} - \frac{\hat{\mu}\hat{\alpha}I_{t}}{1 - I_{t} - L_{t}} - \frac{\hat{\gamma}I_{t}}{L_{t}} + C$$

$$\leq M(\check{\beta}_{1} + m_{2}\check{\gamma}) - \frac{\hat{\mu}(1 - I_{t})}{\varepsilon} - \frac{\hat{\mu}\hat{\alpha}I_{t}}{\varepsilon} + C$$

$$\leq M(\check{\beta}_{1} + m_{2}\check{\gamma}) - \frac{\hat{\mu}\min\{1, \hat{\alpha}\}}{\varepsilon} + C.$$

$$(4.7)$$

In view of (4.5)-(4.7), one obtains that

$$\mathcal{LV} \leq \begin{cases} -M \langle \phi \rangle_T \left(\mathscr{R}_1 - 1 \right) + M(\check{\beta}_1 + m_2 \check{\gamma}) \varepsilon + C < -1 \\ \text{as} \quad I_t \to 0^+, \\ M(\check{\beta}_1 + m_2 \check{\gamma}) - \frac{\hat{\gamma}}{\varepsilon} + C < -1 \\ \text{as} \quad I_t > 0, \ L_t \to 0^+, \\ M(\check{\beta}_1 + m_2 \check{\gamma}) - \frac{\hat{\mu} \min\{1, \hat{\alpha}\}}{\varepsilon} + C < -1 \\ \text{as} \quad I_t + L_t \to 1^-. \end{cases}$$

Therefore,

$$\mathcal{LV} < -1$$
 for all $(I_t, L_t) \in \Gamma_2 \setminus U$.

By Lemma 2.1, we obtain the required assertion.

Remark 4.1. By the form of \mathscr{R}_1 , we know that \mathscr{R}_1 is positively correlated with both the value of noise and the rate of horizontal transmission, indicating that:(i) environmental noise is conducive to the control of diseases; (ii) the reduction in vertical transmission rate helps to the control of diseases. Besides, \mathscr{R}_1 is negatively correlated with β_2 , indicating that media coverage also inhibits the diseases.

5. Extinction of the disease

Theorem 5.1. For any initial value $(I_0, L_0) \in \mathbb{R}^2_+$, if

$$\mathscr{R}_2 = \frac{\check{\beta}_1 + \frac{1}{2}\check{\sigma}_1^2 + \frac{1}{2}\check{\sigma}_2^2}{\hat{\alpha}\hat{\mu} + \hat{\gamma} - \frac{\check{\lambda}\check{\gamma}}{\hat{\mu} + \hat{\lambda}}} < 1 \quad and \quad \hat{\alpha}\hat{\mu} + \hat{\gamma} > \frac{\check{\lambda}\check{\gamma}}{\hat{\mu} + \hat{\lambda}},$$

then the diseases in the stochastic system (2.3) will die out with probability one, *i.e.*,

$$\mathbb{P}\left\{\lim_{t\to\infty}I_t=0\right\}=1,\ \mathbb{P}\left\{\lim_{t\to\infty}L_t=0\right\}=1.$$

Proof. Let us define

$$\mathcal{V}(I_t, L_t) = \frac{1}{2}(I_t + \theta_1 L_t)^2 + \frac{1}{2}\theta_2 L_t^2,$$

where $\theta_1 = \frac{\check{\lambda}}{\hat{\mu} + \check{\lambda}}$, $\theta_2 = \frac{\theta_1(\hat{\alpha}\hat{\mu} + \hat{\gamma} - \theta_1\check{\gamma} - \check{\beta}_1)}{\check{\gamma}}$ are positive constants. By using Itô formula, we have that

$$\begin{split} \mathcal{LV}(I_t, L_t) = & (I_t + \theta_1 L_t) [(\beta_{1,t} - \beta_{2,t} f(I_t))(1 - I_t - L_t) I_t - (\alpha_t \mu_t + \gamma_t - \theta_1 \gamma_t) I_t \\ & + \lambda_t L_t - \theta_1 (\mu_t + \lambda_t) L_t] + \theta_2 [\gamma_t I_t - (\mu_t + \lambda_t) L_t] L_t \\ & + \frac{1}{2} \sigma_{1,t}^2 (1 - I_t - L_t)^2 I_t^2 + \frac{1}{2} \sigma_{2,t}^2 f^2 (I_t) (1 - I_t - L_t)^2 I_t^2 \\ \leq & (I_t + \theta_1 L_t) [\beta_{1,t} - \beta_{2,t} f(I_t) - (\alpha_t \mu_t + \gamma_t) \\ & + \theta_1 \gamma_t] I_t + (I_t + \theta_1 L_t) [\lambda_t - \theta_1 (\mu_t + \lambda_t)] L_t \\ & + \theta_2 [\gamma_t I_t - (\mu_t + \lambda_t) L_t] L_t + \frac{1}{2} \sigma_{1,t}^2 I_t^2 + \frac{1}{2} \sigma_{2,t}^2 I_t^2 \\ \leq & (I_t + \theta_1 L_t) [\check{\beta}_1 - (\hat{\alpha} \hat{\mu} + \hat{\gamma}) + \theta_1 \check{\gamma}] I_t + \theta_2 \check{\gamma} I_t L_t - \theta_2 (\hat{\mu} + \hat{\lambda}) L_t^2 \\ & + \frac{1}{2} \check{\sigma}_1^2 I_t^2 + \frac{1}{2} \check{\sigma}_2^2 I_t^2 \\ = & \left[\check{\beta}_1 - (\hat{\alpha} \hat{\mu} + \hat{\gamma}) + \theta_1 \check{\gamma} + \frac{1}{2} \check{\sigma}_1^2 + \frac{1}{2} \check{\sigma}_2^2 \right] I_t^2 - \theta_2 (\hat{\mu} + \hat{\lambda}) L_t^2 \\ & + [\theta_2 \check{\gamma} - \theta_1 (\hat{\alpha} \hat{\mu} + \hat{\gamma} - \check{\beta}_1 - \theta_1 \check{\gamma})] I_t L_t \\ = & (\hat{\alpha} \hat{\mu} + \hat{\gamma} - \theta_1 \check{\gamma}) (\mathscr{R}_2 - 1) I_t^2 - \theta_2 (\hat{\mu} + \hat{\lambda}) L_t^2 \\ < 0. \end{split}$$

It follows from Mao (Theorem 2.4, [27]) that the diseases will die out. This completes the proof. $\hfill \Box$

Remark 5.1. By Theorem 5.1, the diseases will die out if $\mathscr{R}_2 < 1$. It is noteworthy that, in the absence of environmental factors, the expression of \mathscr{R}_2 coincides with the threshold of the deterministic system (1.1), indicating that we generalized the results of the corresponding deterministic system (1.1).

6. Numerical Examples

In this section, we use the Milstein's Higher Order Method from [16] to show the dynamics of the system (2.3). All simulations are carried out with ©Matlab2016a. The system (2.3) can be rewritten as the following discrete system:

$$\begin{split} X_{k+1} = & X_k + h(X_k)\Delta t + g_1(X_k)\xi_k\sqrt{\Delta t} + g_2(X_k)\xi_k\sqrt{\Delta t} \\ & + \frac{1}{2}\sqrt{\Delta t}(\xi_k^2 - 1)(g_1(X_k + \sqrt{\Delta t}g_1(X_k)) - g_1(X_k)) \\ & + \frac{1}{2}\sqrt{\Delta t}(\xi_k^2 - 1)(g_2(X_k + \sqrt{\Delta t}g_2(X_k)) - g_2(X_k)), \end{split}$$

where $\Delta t = 0.001$ and $\xi_k, k = 1, 2, \cdots$ obey the Gaussian distribution N(0, 1). $X_k = (I_k, L_k)', x = (x_1, x_2)' \in \mathbb{R}^2_+$ and the vector-valued functions $h, g_1, g_2 : \mathbb{R}^2_+ \to \mathbb{R}^2$ are given by

$$h(x) = \begin{bmatrix} (\beta_1 - \beta_2 f(x_1))(1 - x_1 - x_2)x_1 - (\alpha \mu + \gamma)x_1 + \lambda x_2 \\ \gamma x_1 - (\mu + \lambda)x_2 \end{bmatrix},$$
$$g_1(x) = \begin{bmatrix} \sigma_1(1 - x_1 - x_2)x_1 \\ 0 \end{bmatrix},$$
$$g_2(x) = \begin{bmatrix} \sigma_2 f(x_1)(1 - x_1 - x_1)x_1 \\ 0 \end{bmatrix}.$$

To study the effects of environmental noise on the dynamics of the diseases, we start the numerical simulations with $f(I) = \frac{I}{1+I}$ and initial value $(I_0, L_0) = (0.1, 0.1)$. The simulations of the corresponding deterministic system are also studied as a comparison.

Firstly, we choose: $\mu = 1+0.1 \sin(t)$, $\beta_1 = 0.2+0.1 \sin(t)$, $\beta_2 = 0.1+0.05 \sin(t)$, $\alpha = 0.6+0.1 \sin(t)$, $\gamma = 0.2+0.1 \sin(t)$, $\lambda = 0.1+0.02 \sin(t)$, $\sigma_1 = 0.2+0.1 \sin(t)$, $\sigma_2 = 0.2+0.1 \sin(t)$. It follows that $\Re_2 = 0.95 < 1$. By Theorem 5.1, the trivial solution (0,0) of the system (2.3) is stochastically asymptotically stable in the large. The numerical simulations in Figure 2 support these results clearly.

Secondly, we choose parameters: $\mu = 1 + 0.1 \sin(t)$, $\beta_1 = 0.6 + 0.1 \sin(t)$, $\beta_2 = 0.1 + 0.05 \sin(t)$, $\alpha = 0.2 + 0.1 \sin(t)$, $\gamma = 0.2 + 0.1 \sin(t)$, $\lambda = 0.1 + 0.02 \sin(t)$, $\sigma_1 = 0.1 + 0.05 \sin(t)$, $\sigma_2 = 0.1 + 0.05 \sin(t)$. Straightforward calculation yields that $\Re_1 = 2.91 > 1$. Figure 3 shows that the system (2.3) admits a nontrivial positive periodic solution. To proceed, we decrease the value of noise to $\sigma_1 = 0.03 + 0.01 \sin(t)$, $\sigma_2 = 0.03 + 0.01 \sin(t)$, the corresponding simulations are shown in Figure 4. By Figures 3 and 4, the solution of the system (2.3) fluctuates around the solution of the value of noise.



Figure 2. Sample path and phase portrait of the system (2.3) and its corresponding deterministic system



Figure 3. Sample path and phase portrait of the system (2.3) and its corresponding deterministic system



Figure 4. Sample path and phase portrait of the system (2.3) and its corresponding deterministic system

To analyze the effects of vertical transmission on the dynamics of diseases, we increase the value of horizontal transmission rate to $\alpha_1 = 0.4 + 0.1 \sin(t)$. The simulations are shown in Figure 5. The results show that the level of the diseases is decreased with the increase of α .



Figure 5. Sample path and phase portrait of the system (2.3) and its corresponding deterministic system

7. Concluding Remarks

In this paper, we present a non-autonomous stochastic model that helps to study the dynamics of diseases with relapse such as tuberculosis, herpes simplex virus and HIV. The model takes into account the effects of vertical transmission and media coverage on the spread of diseases. By using the Lyapunov method and Khasminskii theorem, we verify the existence of a nontrivial positive periodic solution, indicating that the disease will persist. Besides, we carry out the sufficient conditions for the stochastic asymptotic stability of the trivial solution. The results show that both stochastic noise and media coverage contribute to the control of the diseases. Also, the decrease of the vertical transmission rate plays a role in the control of the diseases. The paper extends the results of the corresponding deterministic system.

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References

- M. Alexander and S. Moghadas, Periodicity in an epidemic model with a generalized non-linear incidence, Mathematical Biosciences, 2004, 189(1), 75–96.
- [2] O. J. Brady, P. W. Gething, S. Bhatt et al., Refining the global spatial limits of dengue virus transmission by evidence-based consensus, PLoS neglected tropical diseases, 2012, 6(8), e1760.
- [3] T. Britton and A. Traoré, A stochastic vector-borne epidemic model: Quasistationarity and extinction, Mathematical Biosciences, 2017, 289, 89–95.
- [4] Y. Cai, Y. Kang, M. Banerjee and W. Wang, Complex dynamics of a hostparasite model with both horizontal and vertical transmissions in a spatial heterogeneous environment, Nonlinear Analysis: Real World Applications, 2018, 40, 444–465.
- [5] Z. Chang, X. Meng and T. Zhang, A new way of investigating the asymptotic behaviour of a stochastic sis system with multiplicative noise, Applied Mathematics Letters, 2019, 87, 80–86.
- [6] J. Cui, X. Tao and H. Zhu, An sis infection model incorporating media coverage, Rocky Mountain Journal of Mathematics, 2008, 38(2008), 1323–1334.
- [7] R. Cui and Y. Lou, A spatial sis model in advective heterogeneous environments, Journal of Differential Equations, 2016, 261(6), 3305–3343.
- [8] N. Dalal, D. Greenhalgh and X. Mao, A stochastic model for internal hiv dynamics, Journal of Mathematical Analysis and Applications, 2008, 341(2), 1084–1101.
- [9] T. Feng, X. Meng, L. Liu and S. Gao, Application of inequalities technique to dynamics analysis of a stochastic eco-epidemiology model, Journal of Inequalities and Applications, 2016, 2016(1), 327.
- [10] T. Feng and Z. Qiu, Global analysis of a stochastic tb model with vaccination and treatment, Discrete and Continuous Dynamical Systems-Series B, 2019, 24(6), 2923–2939.

- [11] T. Feng and Z. Qiu, Global dynamics of deterministic and stochastic epidemic systems with nonmonotone incidence rate, International Journal of Biomathematics, 2018, 11(8), 1850101.
- [12] T. Feng, Z. Qiu, X. Meng and L. Rong, Analysis of a stochastic hiv-1 infection model with degenerate diffusion, Applied Mathematics and Computation, 2019, 348, 437–455.
- [13] N. M. Ferguson, M. J. Keeling, W. J. Edmunds et al., *Planning for smallpox outbreaks*, Nature, 2003, 425(6959), 681–685.
- [14] A. Gray, D. Greenhalgh, L. Hu et al., A stochastic differential equation sis epidemic model, SIAM Journal on Applied Mathematics, 2011, 71(3), 876–902.
- [15] N. Hernandez-Ceron, Z. Feng and C. Castillo-Chavez, Discrete epidemic models with arbitrary stage distributions and applications to disease control, Bulletin of Mathematical Biology, 2013, 75(10), 1716–1746.
- [16] D. J. Higham., An algorithmic introduction to numerical simulation of stochastic differential equations, SIAM Review, 2001, 43(3), 525–546.
- [17] Z. Hu, W. Ma and S. Ruan, Analysis of sir epidemic models with nonlinear incidence rate and treatment, Mathematical Biosciences, 2012, 238(1), 12–20.
- [18] S. Iwami, Y. Takeuchi and X. Liu, Avian-human influenza epidemic model, Mathematical Biosciences, 2007, 207(1), 1–25.
- [19] J. Jiang and Z. Qiu, The complete classification for dynamics in a ninedimensional west nile virus model, SIAM Journal on Applied Mathematics, 2009, 69(5), 1205–1227.
- [20] J. Jiang, Z. Qiu, J. Wu and H. Zhu, Threshold conditions for west nile virus outbreaks, Bulletin of Mathematical Biology, 2009, 71(3), 627–647.
- [21] Y. Kang and C. Castillo-Chavez, Dynamics of si models with both horizontal and vertical transmissions as well as allee effects, Mathematical Biosciences, 2014, 248, 97–116.
- [22] R. Khasminskii, Stochastic stability of differential equations, 66, Springer Science and Business Media, 2011.
- [23] A. Korobeinikov, Global properties of infectious disease models with nonlinear incidence, Bulletin of Mathematical Biology, 2007, 69(6), 1871–1886.
- [24] M. Li, Z. Jin, G. Sun and J. Zhang, Modeling direct and indirect disease transmission using multi-group model, Journal of Mathematical Analysis and Applications, 2017, 446(2), 1292–1309.
- [25] Y. Li and J. Cui, The effect of constant and pulse vaccination on sis epidemic models incorporating media coverage, Communications in Nonlinear Science and Numerical Simulation, 2009, 14(5), 2353–2365.
- [26] H. Mantina, C. Kankasa, W. Klaskala et al., Vertical transmission of kaposi's sarcoma-associated herpesvirus, International Journal of Cancer, 2001, 94(5), 749–752.
- [27] X. Mao, Stochastic differential equations and applications, Elsevier, 2007.
- [28] P. Marcati and M. A. Pozio, Global asymptotic stability for a vector disease model with spatial spread, Journal of Mathematical Biology, 1980, 9(2), 179– 187.

- [29] X. Meng, F. Li and S. Gao, Global analysis and numerical simulations of a novel stochastic eco-epidemiological model with time delay, Applied Mathematics and Computation, 2018, 339, 701–726.
- [30] X. Meng, L. Wang and T. Zhang, Global dynamics analysis of a nonlinear impulsive stochastic chemostat system in a polluted environment, Journal of Applied Analysis and Computation, 2016, 6(3), 865–875.
- [31] A. Miao, T. Zhang, J. Zhang and C. Wang, Dynamics of a stochastic sir model with both horizontal and vertical transmission, Journal of Applied Analysis and Computation, 2018, 8(4), 1108–1121.
- [32] Y. Nakata and T. Kuniya, Global dynamics of a class of seirs epidemic models in a periodic environment, Journal of Mathematical Analysis and Applications, 2010, 363(1), 230–237.
- [33] Z. Qiu, M. Y. Li and Z. Shen, Global dynamics of an infinite dimensional epidemic model with nonlocal state structures, Journal of Differential Equations, 2018, 265(10), 5262–5296.
- [34] Y. Song, A. Miao, T. Zhang et al., Extinction and persistence of a stochastic sirs epidemic model with saturated incidence rate and transfer from infectious to susceptible, Advances in Difference Equations, 2018, 2018(1), 293.
- [35] J. Starke, Tuberculosis. an old disease but a new threat to the mother, fetus, and neonate, Clinics in perinatology, 1997, 24(1), 107–127.
- [36] J. M. Tchuenche, N. Dube, C. P. Bhunu et al., The impact of media coverage on the transmission dynamics of human influenza, BMC Public Health, 2011, 11(1), S5.
- [37] D. Tudor, A deterministic model for herpes infections in human and animal populations, Siam Review, 1990, 32(1), 136–139.
- [38] P. van den Driessche and X. Zou, Modeling diseases with latency and relapse, Mathematical Biosciences and Engineering, 2007, 4(2), 205–219.
- [39] F. Zhang and X. Zhao, A periodic epidemic model in a patchy environment, Journal of Mathematical Analysis and Applications, 2007, 325(1), 496–516.
- [40] S. Zhang, X. Meng, T. Feng and T. Zhang, Dynamics analysis and numerical simulations of a stochastic non-autonomous predator-prey system with impulsive effects, Nonlinear Analysis: Hybrid Systems, 2017, 26, 19–37.
- [41] W. Zhang, X. Meng and Y. Dong, Periodic solution and ergodic stationary distribution of stochastic siri epidemic systems with nonlinear perturbations, Journal of Systems Science and Complexity, 2019.
- [42] K. Zhou, M. Han and Q. Wang, Traveling wave solutions for a delayed diffusive sir epidemic model with nonlinear incidence rate and external supplies, Mathematical Methods in the Applied Sciences, 2017, 40(7), 2772–2783.