THRESHOLD DYNAMICS OF STOCHASTIC MODELS WITH TIME DELAYS: A CASE STUDY FOR YUNNAN, CHINA

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Abstract. In this paper, we provide an effective method for estimating the thresholds of the stochastic models with time delays by using of the nonnegative semimartingale convergence theorem. Firstly, we establish the stochastic delay differential equation models for two diseases, and obtain two thresholds of two diseases and the sufficient conditions for the persistence and extinction of two diseases. Then, numerical simulations for co-infection of HIV/AIDS and Gonorrhea in Yunnan Province, China, are carried out. Finally, we discuss some biological implications and focus on the impact of some key model parameters. One of the most interesting findings is that the stochastic fluctuation and time delays introduced into the deterministic models can suppress the outbreak of the diseases, which can provide some useful control strategies to regulate the dynamics of the diseases, and the numerical simulations verify this phenomenon.

1. **Introduction.** At present, in order to better study the transmission mechanism of infectious diseases, many researchers introduced noises into the deterministic models and studied the effect of noise on the dynamics of the established stochastic epidemic models. Stochastic models could be a more appropriate way of modeling epidemics in many circumstances [11, 24]. In particular, Liu et al. [11] established a deterministic model with non-linear incidence rate, and studied the global stability of the model by the basic reproduction number of the model. Then, based on the deterministic model, the stochastic model is established, the dynamics of the stochastic model is proved theoretically, and the properties of these two models are compared. The paper [24] shows that the stochastic models are able to consider randomness of infectious contacts occurring in the latent or infectious periods. The

combination of stochastic model and deterministic counterpart can make people understand the epidemic trend of infectious diseases more comprehensively, and make the established theory and prevention strategy more reliable. Many realistic stochastic epidemic models can be derived based on their corresponding deterministic counterparts. Britton [3] gave an excellent survey on stochastic differential equation (SDE) epidemic models which presented the exact and asymptotic properties of a simple stochastic epidemic model, and illustrated by studying effects of vaccination and inference procedures for important parameters such as the basic reproduction number and the critical vaccination coverage. Allen [1] provided a great introduction to the methods of derivation for various types of stochastic models including SDE epidemic models. There are different possible approaches including random effects in the models, both from biological and mathematical perspective [9]. The general stochastic differential equation SIRS model introduced in this paper adopts the modeling approach from Mao et al. [13], which has been pursued in [11, 24], and assume that the parameters involved in the model always fluctuate around some average value due to continuous fluctuation in the environment.

On the other hand, the spread of infectious diseases is not only related to the current state, but also to the historical state (see [25, 23]). Therefore, it is more practical to use stochastic differential equation model with time delay to describe the spread of infectious diseases. Recently, many scholars have studied the stochastic infectious disease model with time delay. In particular, Liu et al. [10] proposed and studied the stochastic SEIR epidemic model with infinite distribution delay, and obtained the sufficient conditions for the global asymptotic stability of the endemic equilibrium. Another part of the work is to introduce the disturbance of some parameter variables in the system, which can make the system have a disease-free equilibrium point and give the stability conditions of the disease-free equilibrium point. However, when the parameters of the delay infectious disease model are disturbed randomly, the stochastic model generally does not have disease-free equilibrium and endemic equilibrium. Tornatore et al. [19] proposed a stochastic model with latency and time delay for mosquito transmission, and proposed a strategy for controlling disease transmission. After that, Fan et al. [5] considered the persistence and extinction of a class of stochastic SIR epidemic model with generalized nonlinear incidence and transient immunity and time delay, and obtained the threshold of the model. On the basis of [19, 5], Berrhazi et al. [2] considered the stochastic SIR epidemic model with Beddington-DeAngelis incidence and delayed immune loss under Lévy noise disturbance, and studied its persistence, extinction and threshold problems. Considering the effect of vaccination and the incubation period of some diseases (such as tuberculosis, AIDS, measles) after infection, Liu et al. [12] discussed the stochastic SVEIR epidemic model with distribution delay. By constructing appropriate stochastic Lyapunov function, the existence, uniqueness and ergodicity of the positive distribution of the system were obtained.

Motivated by above mentioned papers, we introduce time delays and stochastic into the deterministic differential equations. This paper is organized as follows: In Section 2.1, we give the basic theory and related lemmas. In Section 2.2, we construct a class of stochastic differential equations models with time delays, and give the thresholds and dynamics of the models by using the theorems and lemmas of Section 2.1. In Section 3, we do a case study of the models in Section 2.2. In addition, we carry out some numerical simulations aiming to HIV/AIDS and

Gonorrhea transmission in Yunnan by using the models. In Section 4, we summarize the whole paper.

2. Modeling and theoretical analysis.

2.1. Basic theory and related lemmas.
Firstly, we introduce some lemmas and notations, which will be used in the following parts. Generally speaking, the d-dimensional stochastic differential equation can be expressed as follows:

$$\mathrm{d}X(t) = f(t, X(t))\mathrm{d}t + g(t, X(t))\mathrm{d}B(t), \tag{2.1.1}$$

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, g are locally Lipschitz with respect to the second variable. $B(t)$ is an m-dimensional standard Brownian motion defined on the above probability space. The differential operator L of system (2.1.1) is defined by

$$L = \frac{\partial}{\partial t} + \sum_{i=1}^{d} f_i(t)\frac{\partial}{\partial x_i} + \frac{1}{2}\sum_{i,j=1}^{d} [g^T(x,t)g(x,t)]_{ij}\frac{\partial^2}{\partial x_i \partial x_j}. \tag{2.1.2}$$

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

$$LV(x,t) = V_t(x,t) + V_x(x,t)f(x,t) + \frac{1}{2}trace[g^T(x,t)g(x,t)], \tag{2.1.3}$$

where $V_t(x,t) = \frac{\partial V}{\partial t}, V_x(x,t) = (\frac{\partial V}{\partial x_1}, \cdots, \frac{\partial V}{\partial x_d}), V_{xx} = (\frac{\partial^2 V}{\partial x_1 \partial x_j})_{d \times d}$. In view of Itô's formula, if $x(t) \in \mathbb{R}^d$, then $\mathrm{d}V(x,t) = LV(x,t)\mathrm{d}t + V_x(x,t)g(x,t)\mathrm{d}B(t)$.

Lemma 2.1. [27] *Let $A(t)$ and $U(t)$ be two continuous adapted increasing processes on $t \geqslant 0$ with $A(0) = U(0) = 0$ a.s. Suppose that $M(t)$ is a real-valued continuous local martingale with $M(0) = 0$ a.s. and $X(0)$ a nonnegative F_0-measurable random variable with $E(X(0)) < \infty$. Define $X(t) = X(0) + A(t) - U(t) + M(t)$ for all $t \geqslant 0$. If $X(t)$ is nonnegative, then $\lim_{t\to\infty} A(t) < \infty$ implies that $\lim_{t\to\infty} U(t) < \infty, \lim_{t\to\infty} X(t) < \infty$ and $-\infty < \lim_{t\to\infty} M(t) < \infty$ with probability one.*

Lemma 2.2. [27] *Let $M(t)$, $t \geqslant 0$, be a local martingale vanishing at time 0 and define*

$$\rho_M(t) := \int_o^t \frac{d[M,M](s)}{(1+s)^2}, t \geqslant 0, \tag{2.1.4}$$

where $[M,M](t)$ is Meyers angle bracket process. Then $\lim_{t\to\infty} \frac{M(t)}{t} = 0$ a.s. provided $\lim_{t\to\infty} \rho_M(t) < \infty$ a.s.

Lemma 2.3. [4] *Set $f \in \mathbb{C}[[0, \infty] \times \Omega, (0, \infty)]$, and $\langle x(t)\rangle = \frac{1}{t}\int_0^t x(s)ds$. Suppose that there exist positive constants λ_0, λ such that*

$$\log f(t) = \lambda t - \lambda_0 \int_0^t f(s)ds + F(t) \ a.s. \tag{2.1.5}$$

for all $t \geqslant 0$, where $F \in \mathbb{C}[[0, \infty] \times \Omega, (0, \infty)]$ and $\lim_{t\to\infty} \frac{F(t)}{t} = 0$ a.s. Then

$$\lim_{t\to\infty} \langle f(t)\rangle = \frac{\lambda}{\lambda_0} \ a.s. \tag{2.1.6}$$

In real world, a group of people will often be infected with a variety of diseases, especially two of them have the same or similar transmission routes. We discuss a class of models with two infectious diseases as follows.

2.2. **Modeling and analysis for double diseases.** In the paper [14], Meng and Zhao formulated a kind of epidemic model with two classes of epidemics as follows

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} = A - dS - \dfrac{\beta_1 S I_1}{1 + a_1 I_1} - \dfrac{\beta_2 S I_2}{1 + a_2 I_2} + r_1 I_1 + r_2 I_2, \\[2mm] \dfrac{\mathrm{d}I_1}{\mathrm{d}t} = \dfrac{\beta_1 S I_1}{1 + a_1 I_1} - (d + \alpha_1 + r_1)I_1, \\[2mm] \dfrac{\mathrm{d}I_2}{\mathrm{d}t} = \dfrac{\beta_2 S I_2}{1 + a_2 I_2} - (d + \alpha_2 + r_2)I_2, \end{cases} \tag{2.2.1}$$

where S denotes the number of the population susceptible to the diseases, I_1 and I_2 are the total population of the infective in terms of two diseases at time t, respectively. The recruitment to the susceptible population is to be considered as a constant A, β_1 and β_2 are the contact rates, d is natural mortality rate, α_1 and α_2 are the rates of diseases-related death, r_1 and r_2 are the treatment cure rates of two diseases, respectively. a_i is the parameter that measure the inhibitory effect for I_i. The incidence rate $\frac{\beta_i S I_i}{1+a_i I_i}(i = 1, 2)$ of susceptible individuals through their contacts with infectious. The two thresholds of the model are $\mathcal{R}_1 = \frac{\beta_1 A}{d(d+\alpha_1+r_1)}$ and $\mathcal{R}_2 = \frac{\beta_2 A}{d(d+\alpha_2+r_2)}$, and the four equilibria are $E_0 = \left(\frac{A}{d}, 0, 0\right)$, $E_1 = (S_1^*, I_1^*, 0)$, $E_2 = (S_2^*, 0, I_2^*)$ and $E^* = (S^*, I_1^{**}, I_2^{**})$, respectively. In addition, the dynamic behavior for the model (2.2.1) is described in the following theorem.

Theorem 2.4. [14] *For system (2.2.1), the following conclusions are true.*

(i) If $\mathcal{R}_1 < 1$ and $\mathcal{R}_2 < 1$, then both diseases go extinct and system (2.2.1) has a unique stable diseases-extinction equilibrium E_0.

(ii) If $\mathcal{R}_1 > 1$ and $\mathcal{R}_2 < 1$, then the disease I_2 goes extinct and system (2.2.1) has a unique stable equilibrium E_1.

(iii) If $\mathcal{R}_1 < 1$ and $\mathcal{R}_2 > 1$, then the disease I_1 goes extinct and system (2.2.1) has a unique stable equilibrium E_2.

(iv) If $\mathcal{R}_1 > 1$ and $\mathcal{R}_2 > 1$, then E^* is a unique stable equilibrium, which implies both diseases of system (2.2.1) are permanent.

Next, we study the stochastic model with time delays corresponding to model (2.2.1).

$$\begin{cases} \mathrm{d}S = \left[A - dS - \dfrac{\beta_1 S(t-\tau_1) I_1}{1 + a_1 I_1} - \dfrac{\beta_2 S(t-\tau_2) I_2}{1 + a_2 I_2} + r_1 I_1 + r_2 I_2\right]\mathrm{d}t + \sigma_3 S \mathrm{d}B_3(t), \\[2mm] \mathrm{d}I_1 = \left[\dfrac{\beta_1 S(t-\tau_1) I_1}{1 + a_1 I_1} - (d + \alpha_1 + r_1)I_1\right]\mathrm{d}t + \sigma_1 I_1 \mathrm{d}B_1(t), \\[2mm] \mathrm{d}I_2 = \left[\dfrac{\beta_2 S(t-\tau_2) I_2}{1 + a_2 I_2} - (d + \alpha_2 + r_2)I_2\right]\mathrm{d}t + \sigma_2 I_2 \mathrm{d}B_2(t), \end{cases}$$
$$\tag{2.2.2}$$

where $B_i(t)$ represents a standard Brownian motion with $B_i(0) = 0$ and $\sigma_i^2 > 0$ ($i = 1, 2, 3$) denotes the intensity of the white noise, τ_i denotes that S has become the susceptible population of I_i before τ_i. On the basis of model (2.2.2), we define two thresholds: $\mathcal{R}_1^* = \frac{1}{d+\alpha_1+r_1} \cdot \left(\frac{\beta_1 A}{d} - \frac{\sigma_1^2}{2}\right)$ and $\mathcal{R}_2^* = \frac{1}{d+\alpha_2+r_2} \cdot \left(\frac{\beta_2 A}{d} - \frac{\sigma_2^2}{2}\right)$, respectively.

Lemma 2.5. *For the solution* $(S(t), I_1(t), I_2(t))$ *of model (2.2.2) with any initial value* $(S(0), I_1(0), I_1(0)) \in \mathbb{R}_+^3$, *we have*

$$\limsup_{t \to \infty}(S(t) + I_1(t) + I_2(t)) < \infty \ a.s. \tag{2.2.3}$$

Moreover,

$$\lim_{t \to +\infty} \frac{1}{t} \int_0^t \sigma_1 I_1(\theta) \, dB_1(\theta) = 0, \quad \lim_{t \to +\infty} \frac{1}{t} \int_0^t \sigma_2 I_2(\theta) \, dB_2(\theta) = 0,$$

$$\lim_{t \to +\infty} \frac{1}{t} \int_0^t \sigma_3 S(\theta) \, dB_3(\theta) = 0, \quad \lim_{t \to +\infty} \frac{1}{t} \int_0^t \sigma_i \, dB_i(\theta) = 0, \ (i = 1, 2, 3) \ a.s. \tag{2.2.4}$$

Proof. From (2.2.2), we get

$$\mathrm{d}(S + I_1 + I_2) = [A - d(S + I_1 + I_2) - \alpha_1 I_1 - \alpha_2 I_2] \, \mathrm{d}t$$
$$+ \sigma_3 S \mathrm{d}B_3(t) + \sigma_1 I_1 \mathrm{d}B_1(t) + \sigma_2 I_2 \mathrm{d}B_2(t). \tag{2.2.5}$$

This equation has the solution

$$S(t) + I_1(t) + I_2(t) = \frac{A}{d} + \left[(S(0) + I_1(0) + I_2(0)) - \frac{A}{d} \right] e^{-dt}$$

$$- \alpha_1 \int_0^t e^{-d(t-s)} I_1(s) \mathrm{d}s - \alpha_2 \int_0^t e^{-d(t-s)} I_2(s) \mathrm{d}s + M(t) \tag{2.2.6}$$

$$\leq \frac{A}{d} + \left[(S(0) + I_1(0) + I_2(0)) - \frac{A}{d} \right] e^{-dt} + M(t),$$

where

$$M(t) = \sigma_3 \int_0^t e^{-d(t-s)} S(s) \mathrm{d}B_3(s) + \sigma_1 \int_0^t e^{-d(t-s)} I_1(s) \mathrm{d}B_1(s)$$

$$+ \sigma_2 \int_0^t e^{-d(t-s)} I_2(s) \mathrm{d}B_2(s)$$

is a continuous local martingale with $M(0) = 0$ a.s. Define

$$X(t) = X(0) + A(t) - U(t) + M(t), \tag{2.2.7}$$

with $X(0) = (S(0) + I_1(0) + I_2(0))$, $A(t) = \frac{A}{d}(1 - e^{-dt})$ and $U(t) = (S(0) + I_1(0) + I_2(0))(1 - e^{-dt})$ for all $t \geq 0$. Due to the stochastic comparison theorem, $S(t) + I_1(t) + I_2(t) \leq X(t)$ a.s. It is easy to check that $A(t)$ and $U(t)$ are continuous adapted increasing processes for $t \geq 0$ with $A(0) = U(t) = 0$. By using Lemma 2.1, we have $\lim_{t \to \infty} X(t) < \infty$ a.s. This completes the proof of (2.2.3).

For convenience, we denote

$$M_1(t) = \sigma_1 \int_0^t I_1(s) \mathrm{d}B_1(s), M_2(t) = \sigma_2 \int_0^t I_2(s) \mathrm{d}B_2(s), M_3(t) = \sigma_3 \int_0^t S(s) \mathrm{d}B_3(s),$$

$$M_4(t) = \sigma_1 \int_0^t \mathrm{d}B_1(s), M_5(t) = \sigma_2 \int_0^t \mathrm{d}B_2(s), M_6(t) = \sigma_3 \int_0^t \mathrm{d}B_3(s). \tag{2.2.8}$$

Computing $[M_1, M_1](t) = \sigma_1^2 \int_0^t I_1^2(s) \mathrm{d}s$, by Lemma 2.2 and (2.2.3), we obtain

$$\lim_{t \to \infty} \rho_1(t) = \lim_{t \to \infty} \int_0^t \frac{\sigma_1^2 I_1^2(s) \mathrm{d}s}{(1+s)^2} \leq \sigma_1^2 \sup_{t \geq 0} \{I_1^2(t)\} < \infty. \tag{2.2.9}$$

Then, by Lemma 2.2, $\lim_{t \to \infty} \frac{1}{t} \int_0^t \sigma_1 I_1(s) \mathrm{d}B_1(s) = 0$. The left can be proved similarly. This completes the proof. $\qquad \square$

Lemma 2.6. *For the solution $(S(t), I_1(t), I_2(t))$ of model (2.2.2) with any initial value $(S(0), I_1(0), I_1(0)) \in \mathbb{R}_+^3$, we have*

$$\limsup_{t \to \infty} \langle S(t) + I_1(t) + I_2(t) \rangle < \frac{A}{d} \ a.s. \tag{2.2.10}$$

Proof. We set

$$M_a(t) = \int_0^t S(s) \mathrm{d}B_3(s), \ M_a^*(t) = \int_0^t e^{-d(t-s)} S(s) \mathrm{d}B_3(s),$$

$$M_b(t) = \int_0^t I_1(s) \mathrm{d}B_1(s), \ M_b^*(t) = \int_0^t e^{-d(t-s)} I_1(s) \mathrm{d}B_1(s), \tag{2.2.11}$$

$$M_c(t) = \int_0^t I_2(s) \mathrm{d}B_2(s), \ M_c^*(t) = \int_0^t e^{-d(t-s)} I_2(s) \mathrm{d}B_2(s).$$

By Lemma 2.5, we have

$$\lim_{t \to \infty} \frac{1}{t} M_a(t) = 0, \ \lim_{t \to \infty} \frac{1}{t} M_a^*(t) = 0,$$

$$\lim_{t \to \infty} \frac{1}{t} M_b(t) = 0, \ \lim_{t \to \infty} \frac{1}{t} M_b^*(t) = 0, \tag{2.2.12}$$

$$\lim_{t \to \infty} \frac{1}{t} M_c(t) = 0, \ \lim_{t \to \infty} \frac{1}{t} M_c^*(t) = 0 \text{ a.s.}$$

From (2.2.6), since

$$\begin{aligned}
\langle M(t) \rangle &= \frac{\sigma_3}{t} \int_0^t \int_0^s e^{-d(s-u)} S(u) \mathrm{d}B_3(u) \mathrm{d}s \\
&\quad + \frac{\sigma_1}{t} \int_0^t \int_0^s e^{-d(s-u)} I_1(u) \mathrm{d}B_1(u) \mathrm{d}s \\
&\quad + \frac{\sigma_2}{t} \int_0^t \int_0^s e^{-d(s-u)} I_2(u) \mathrm{d}B_2(u) \mathrm{d}s \\
&= \frac{\sigma_3}{t} \left(\int_0^t S(u) \mathrm{d}B_3(u) - \int_0^t e^{-d(t-u)} S(u) \mathrm{d}B_3(u) \right) \\
&\quad + \frac{\sigma_1}{t} \left(\int_0^t I_1(u) \mathrm{d}B_1(u) - \int_0^t e^{-d(t-u)} I_1(u) \mathrm{d}B_1(u) \right) \\
&\quad + \frac{\sigma_2}{t} \left(\int_0^t I_2(u) \mathrm{d}B_2(u) - \int_0^t e^{-d(t-u)} I_2(u) \mathrm{d}B_2(u) \right),
\end{aligned} \tag{2.2.13}$$

by (2.2.12), we obtain $\lim_{t \to \infty} \langle M(t) \rangle = 0$. From (2.2.6), we get

$$\begin{aligned}
&\lim_{t \to \infty} \frac{1}{t} \int_0^t \left[S(0) + I_1(0) + I_2(0) - \frac{A}{d} \right] e^{-ds} \mathrm{d}s \\
&= \lim_{t \to \infty} \frac{1}{dt} \left\{ \left[S(0) + I_1(0) + I_2(0) - \frac{A}{d} \right] (1 - e^{-dt}) \right\} = 0.
\end{aligned} \tag{2.2.14}$$

By (2.2.6), (2.2.13) and (2.2.14), we obtain

$$\limsup_{t \to \infty} \langle S(t) + I_1(t) + I_2(t) \rangle < \frac{A}{d} \text{ a.s.}$$

This completes the proof. □

Lemma 2.7. *For any given initial value $(S(0), I_1(0), I_1(0)) \in \mathbb{R}_+^3$, there exists a unique positive solution $(S(t), I_1(t), I_2(t))$ to system (2.2.2) on $t \geq 0$ and the solution will remain in $\mathbb{R}_+^3$ with probability one, that is to say, $(S(t), I_1(t), I_2(t)) \in \mathbb{R}_+^3$ for all $t \geq 0$ almost surely.*

Proof. Since the coefficients of system (2.2.2) are locally Lipschitz continuous, for any given initial value $(S(0), I_1(0), I_1(0)) \in \mathbb{R}_+^3$, there exists a unique local solution $(S(t), I_1(t), I_2(t))$ on $t \in [0, \tau_e)$, where τ_e denotes the explosion time (see [3]). To verify that this solution is global, we only need to prove $\tau_e = +\infty$ a.s.

Let $k_0 > 0$ be enough large such that each component of $(S(0), I_1(0), I_1(0))$ is no large than k_0. For each integer $k > k_0$, define the stopping time

$$\tau_k = \inf \{t \in [0, \tau_e) : S(t) \geq k, I_1(t) \geq k, I_2(t) \geq k\},$$

where throughout this paper we set $\inf \emptyset = +\infty$. Obviously, τ_k is increasing as $k \to \infty$. Set $\tau_\infty = \lim_{k \to \infty} \tau_k$, then we can get $\tau_\infty \leq \tau_e$ a.s.

Define a $\mathbb{C}^2$-function $V : \mathbb{R}_+^3 \to \mathbb{R}_+$ by

$$V(X) = S + I_1 + I_2.$$

By Itô's formula, we get

$$\begin{aligned}
\mathrm{d}V(X) &= [A - dS - dI_1 - dI_2 - \alpha_1 I_1 - \alpha_2 I_2]\,\mathrm{d}t \\
&\quad + \sigma_3 S \mathrm{d}B_3(t) + \sigma_1 I_1 \mathrm{d}B_1(t) + \sigma_2 I_2 \mathrm{d}B_2(t) \\
&\triangleq LV\mathrm{d}t + \sigma_3 S \mathrm{d}B_3(t) + \sigma_1 I_1 \mathrm{d}B_1(t) + \sigma_2 I_2 \mathrm{d}B_2(t),
\end{aligned}$$

where

$$LV = A - dS - dI_1 - dI_2 - \alpha_1 I_1 - \alpha_2 I_2 \leqslant A.$$

For any $k \geqslant k_0$, there exists $T > 0$ such that $\tau_k \in (0, T \wedge \tau_k]$. By the generalized Itô's formula, for any $t \in (0, T \wedge \tau_k]$, we have

$$\begin{aligned}
EV(X(T \wedge \tau_k)) &= EV(X(S(0), I_1(0), I_2(0))) + E \int_0^{T \wedge \tau_k} LV(X(s))\mathrm{d}s \\
&\leqslant EV(X(S(0), I_1(0), I_2(0))) + AT,
\end{aligned}$$

where E is the expectation of the function. Let $k \to \infty$, then $t \to \infty$, it follows that $\lim_{k \to \infty} P(\tau_k \leqslant T) = 0$, therefore $P(\tau_\infty \leqslant T) = 0$. Since $T > 0$ is arbitrary, it results in

$$P(\tau_\infty < \infty) = 0, \ P(\tau_\infty = \infty) = 1.$$

This completes the proof. $\qquad\qquad\square$

Theorem 2.8. *Let $(S(t), I_1(t), I_2(t))$ be the positive solution of model (2.2.2) with initial value $(S(0), I_1(0), I_2(0)) \in \mathbb{R}_+^3$. Then as $\mathcal{R}_i^* < 1$, two infectious diseases of model (2.2.2) go extinct almost surely, i.e. $\lim_{t \to \infty} I_i(t) = 0$, $i = 1, 2$. Moreover, $\lim_{t \to \infty} S(t) = \frac{A}{d}$ a.s.*

Proof. Define a function $V = \ln I_i(t)$. By Itô's formula, we obtain

$$\mathrm{d}\ln I_i(t) = \left(\frac{\beta_i S(t - \tau_i)}{1 + a_i I_i} - (d + \alpha_i + r_i) - \frac{1}{2}\sigma_i^2\right)\mathrm{d}t + \sigma_i \mathrm{d}B_i(t). \qquad (2.2.15)$$

Integrating (2.2.15) from 0 to t gives

$$
\ln I_i(t) = \beta_i \int\limits_0^t \frac{S(r-\tau_i)}{1+a_iI_i}\mathrm{d}r - (d+\alpha_i+r_i)t - \frac{1}{2}\sigma_i^2 t + \sigma_i B_i(t) - \sigma_i B_i(0) + \ln I_i(0)
$$

$$
\leqslant \beta_i \int\limits_0^t S(r-\tau_i)\mathrm{d}r - (d+\alpha_i+r_i)t - \frac{1}{2}\sigma_i^2 t + \sigma_i B_i(t) - \sigma_i B_i(0) + \ln I_i(0)
$$

$$
= \beta_i \int\limits_0^t S(r)\mathrm{d}r + \beta_i \int\limits_{-\tau_i}^0 S(r)\mathrm{d}r - \beta_i \int\limits_{t-\tau_i}^t S(r)\mathrm{d}r - (d+\alpha_i+r_i)t
$$

$$
- \frac{1}{2}\sigma_i^2 t + +\sigma_i B_i(t) - \sigma_i B_i(0) + \ln I_i(0)
$$

$$
\triangleq \beta_i \int\limits_0^t S(r)\mathrm{d}r - (d+\alpha_i+r_i)t - \frac{1}{2}\sigma_i^2 t + P(t),
$$

$$(2.2.16)$$

where

$$
P(t) = \beta_i \int\limits_{-\tau_i}^0 S(r)\mathrm{d}r - \beta_i \int\limits_{t-\tau_i}^t S(r)\mathrm{d}r + \sigma_i B_i(t) - \sigma_i B_i(0) + \ln I_i(0).
$$

Dividing both sides of (2.2.16) by t, we have

$$
\frac{\ln I_i(t)}{t} \leqslant \beta_i \langle S \rangle - (d+\alpha_i+r_i) - \frac{1}{2}\sigma_i^2 + \frac{P(t)}{t}. \tag{2.2.17}
$$

By integrating the model (2.2.2) and dividing the two sides by t, we conclude that

$$
\begin{cases}
\dfrac{S(t)-S(0)}{t} = A - d\langle S \rangle - \dfrac{\beta_1}{t}\int\limits_0^t \dfrac{S(r-\tau_1)I_1}{1+a_1I_1}\mathrm{d}r - \dfrac{\beta_2}{t}\int\limits_0^t \dfrac{S(r-\tau_2)I_2}{1+a_2I_2}\mathrm{d}r \\
\qquad\qquad +r_1\langle I_1 \rangle + r_2\langle I_2 \rangle + \dfrac{\sigma_3}{t}\int\limits_0^t S(r)\mathrm{d}B_3(r), \\
\dfrac{I_i(t)-I_i(0)}{t} = \dfrac{\beta_i}{t}\int\limits_0^t \dfrac{S(r-\tau_i)I_i}{1+a_iI_i}\mathrm{d}r - (d+\alpha_i+r_i)\langle I_i \rangle + \dfrac{\sigma_i}{t}\int\limits_0^t I_i(r)\mathrm{d}B_i(r).
\end{cases}
$$

$$(2.2.18)$$

Calculating the sum of (2.2.18), we can assert that

$$
\frac{S(t)-S(0)}{t} + \frac{I_1(t)-I_1(0)}{t} + \frac{I_2(t)-I_2(0)}{t}
$$
$$
= A - d\langle S \rangle + r_1\langle I_1 \rangle + r_2\langle I_2 \rangle - (d+\alpha_1+r_1)\langle I_1 \rangle - (d+\alpha_2+r_2)\langle I_2 \rangle
$$
$$
+\frac{\sigma_3}{t}\int\limits_0^t S(r)\mathrm{d}B_3(r) + \frac{\sigma_1}{t}\int\limits_0^t I_1(r)\mathrm{d}B_1(r) + \frac{\sigma_2}{t}\int\limits_0^t I_2(r)\mathrm{d}B_2(r) \tag{2.2.19}
$$
$$
\triangleq A - d\langle S \rangle - (d+\alpha_1)\langle I_1 \rangle - (d+\alpha_2)\langle I_2 \rangle + \frac{W}{t},
$$

where

$$\frac{W}{t} = \frac{\sigma_3}{t} \int\limits_0^t S(r) \mathrm{d}B_3(r) + \frac{\sigma_1}{t} \int\limits_0^t I_1(r) \mathrm{d}B_1(r) + \frac{\sigma_2}{t} \int\limits_0^t I_2(r) \mathrm{d}B_2(r).$$

According to (2.2.19), we have

$$d\langle S \rangle = A - (d + \alpha_1) \langle I_1 \rangle - (d + \alpha_2) \langle I_2 \rangle + \frac{W}{t}$$
$$- \frac{S(t) - S(0)}{t} - \frac{I_1(t) - I_1(0)}{t} - \frac{I_2(t) - I_2(0)}{t}. \tag{2.2.20}$$

Putting (2.2.20) into (2.2.17), we obtain

$$\frac{\ln I_i(t)}{t} \leqslant \beta_i \left(\frac{A}{d} - \frac{(d+\alpha_1)}{d} \langle I_1 \rangle - \frac{(d+\alpha_2)}{d} \langle I_2 \rangle + \frac{W}{td} - \frac{S(t) - S(0)}{td} \right.$$
$$\left. - \frac{I_1(t) - I_1(0)}{td} - \frac{I_2(t) - I_2(0)}{td} \right) - (d + \alpha_i + r_i) - \frac{1}{2}\sigma_i^2 + \frac{P(t)}{t}. \tag{2.2.21}$$

By Lemma 2.5 and Lemma 2.6, we get

$$\lim_{t \to \infty} \frac{\ln I_i(t)}{t} \leqslant \beta_i \frac{A}{d} - (d + \alpha_i + r_i) - \frac{1}{2}\sigma_i^2$$
$$- \beta_i \frac{(d+\alpha_1)}{d} \lim_{t \to \infty} \langle I_1 \rangle - \beta_i \frac{(d+\alpha_2)}{d} \lim_{t \to \infty} \langle I_2 \rangle \tag{2.2.22}$$
$$\leqslant \beta_i \frac{A}{d} - (d + \alpha_i + r_i) - \frac{1}{2}\sigma_i^2.$$

That is, when $\mathcal{R}_i^* = \frac{1}{d+\alpha_i+r_i} \cdot \left(\frac{\beta_i A}{d} - \frac{\sigma_i^2}{2} \right) < 1$, there obviously holds $\limsup\limits_{t\to\infty} \frac{\ln I_i(t)}{t} < 0$, which implies $\lim\limits_{t\to\infty} I_i(t) = 0$, $\lim\limits_{t\to\infty} S(t) = \frac{A}{d} = S_0$. This completes the proof. $\square$

Theorem 2.9. *Let $(S(t), I_1(t), I_2(t))$ be any positive solution of model (2.2.2) with initial value $(S(0), I_1(0), I_2(0)) \in \mathbb{R}_+^3$, then we have the following results.*

(i) If $\mathcal{R}_1^* > 1$ and $\mathcal{R}_2^* < 1$, then the disease I_2 goes extinct and the disease I_1 is permanent on average. Moreover, I_1 satisfies

$$\liminf_{t \to \infty} \langle I_1(t) \rangle = \frac{1}{a_1 + \frac{\beta_1(d+\alpha_1)}{d(d+\alpha_1+r_1)}} (\mathcal{R}_1^* - 1) > 0.$$

(ii) If $\mathcal{R}_2^* > 1$ and $\mathcal{R}_1^* < 1$, then the disease I_1 goes extinct and the disease I_2 is permanent on average. Moreover, I_2 satisfies

$$\liminf_{t \to \infty} \langle I_2(t) \rangle = \frac{1}{a_2 + \frac{\beta_2(d+\alpha_2)}{d(d+\alpha_2+r_2)}} (\mathcal{R}_2^* - 1) > 0.$$

(iii) If $\mathcal{R}_1^* > 1$ and $\mathcal{R}_2^* > 1$, then the two infections diseases I_1 and I_2 are permanent on average. Moreover, I_1 and I_2 satisfy

$$\liminf_{t \to \infty} \langle a_2 I_1(t) + a_1 I_2(t) \rangle \geqslant \frac{1}{\max\{m_1, m_2\}} \{a_2(\mathcal{R}_1^* - 1) + a_1(\mathcal{R}_2^* - 1)\} > 0,$$

where

$$m_1 \triangleq a_1(d + \alpha_1) + \frac{\beta_1}{d}(d + \alpha_1) + \frac{a_1 \beta_2(d + \alpha_1)}{a_2 d} + a_1 r_1,$$

$$m_2 \triangleq a_2(d + \alpha_2) + \frac{\beta_2}{d}(d + \alpha_2) + \frac{a_2 \beta_1(d + \alpha_2)}{a_1 d} + a_2 r_2.$$

Proof. Firstly, we prove (i). From model (2.2.2), we obtain

$$
\begin{aligned}
\mathrm{d}S &= \left[A - dS - \frac{\beta_1 S(t-\tau_1) I_1}{1+a_1 I_1} - \frac{\beta_2 S(t-\tau_2) I_2}{1+a_2 I_2} + r_1 I_1 + r_2 I_2 \right] \mathrm{d}t + \sigma_3 S \mathrm{d}B_3(t) \\
&= \left[A - dS - \frac{\beta_1}{a_1} S(t-\tau_1) + \frac{1}{a_1} \frac{\beta_1 S(t-\tau_1)}{1+a_1 I_1} - \frac{\beta_2 S(t-\tau_2) I_2}{1+a_2 I_2} + r_1 I_1 + r_2 I_2 \right] \mathrm{d}t \\
&\quad + \sigma_3 S \mathrm{d}B_3(t).
\end{aligned}
$$

$$(2.2.23)$$

Integrating (2.2.23) and dividing the two sides by t, we may assert that $\lim_{t\to\infty} I_2(t) = 0$ as $\mathcal{R}_2^* < 1$. From Theorem 2.8, we obtain

$$
\begin{aligned}
\frac{S(t) - S(0)}{t} &= A - d \langle S \rangle - \left[\frac{\beta_1}{a_1 t} \int_0^t S(r)\mathrm{d}r + \frac{\beta_1}{a_1 t} \int_{-\tau_1}^0 S(r)\mathrm{d}r - \frac{\beta_1}{a_1 t} \int_{t-\tau_1}^t S(r)\mathrm{d}r \right] \\
&\quad + \frac{1}{a_1 t} \int_0^t \frac{\beta_1 S(r-\tau_1)}{1+a_1 I_1} \mathrm{d}r - \frac{1}{t} \int_0^t \frac{\beta_2 S(r-\tau_2) I_2}{1+a_2 I_2} \mathrm{d}r \\
&\quad + r_1 \langle I_1 \rangle + r_2 \langle I_2 \rangle + \frac{\sigma_3}{t} \int_0^t S(r)\mathrm{d}B_3(r) \\
&= A - d \langle S \rangle - \frac{\beta_1}{a_1 t} \int_0^t S(r)\mathrm{d}r + \frac{1}{a_1 t} \int_0^t \frac{\beta_1 S(r-\tau_1)}{1+a_1 I_1} \mathrm{d}r + r_1 \langle I_1 \rangle + r_2 \langle I_2 \rangle \\
&\quad - \frac{1}{t} \int_0^t \frac{\beta_2 S(r-\tau_2) I_2}{1+a_2 I_2} \mathrm{d}r - \frac{\beta_1}{a_1 t} \int_{-\tau_1}^0 S(r)\mathrm{d}r + \frac{\beta_1}{a_1 t} \int_{t-\tau_1}^t S(r)\mathrm{d}r \\
&\triangleq A - \left(d + \frac{\beta_1}{a_1} \right) \langle S \rangle + \frac{1}{a_1 t} \int_0^t \frac{\beta_1 S(r-\tau_1)}{1+a_1 I_1} \mathrm{d}r + r_1 \langle I_1 \rangle + \frac{Q}{t},
\end{aligned}
$$

$$(2.2.24)$$

where

$$
\frac{Q}{t} = -\frac{1}{t} \int_0^t \frac{\beta_2 S(r-\tau_2) I_2}{1+a_2 I_2} \mathrm{d}r - \frac{\beta_1}{a_1 t} \int_{-\tau_1}^0 S(r)\mathrm{d}r + \frac{\beta_1}{a_1 t} \int_{t-\tau_1}^t S(r)\mathrm{d}r.
$$

By (2.2.24), we deduce

$$
A - \left(d + \frac{\beta_1}{a_1} \right) \langle S \rangle + \frac{1}{a_1 t} \int_0^t \frac{\beta_1 S(r-\tau_1)}{1+a_1 I_1} \mathrm{d}r + r_1 \langle I_1 \rangle = \frac{S(t) - S(0)}{t} - \frac{Q}{t} \triangleq \frac{\Phi}{t}.
$$

$$(2.2.25)$$

From model (2.2.2), we also obtain

$$
A - d \langle S \rangle - (d + \alpha_1) \langle I_1 \rangle - (d + \alpha_2) \langle I_2 \rangle \triangleq \frac{\Theta}{t}, \tag{2.2.26}
$$

where

$$
\frac{\Theta}{t} = \frac{S(t) - S(0) + I_1(t) - I_1(0) + I_2(t) - I_2(0) - M_1(t) - M_2(t) - M_3(t)}{t}.
$$

Define function $V = \ln I_1(t)$. By using Itô's formula and combining with (2.2.17), we obtain

$$\frac{\ln I_1(t)}{t} = \frac{1}{t} \int_0^t \frac{\beta_1 S(r - \tau_1)}{1 + a_1 I_1} \mathrm{d}r - (d + \alpha_1 + r_1) - \frac{1}{2}\sigma_1^2 + \frac{P(t)}{t}. \qquad (2.2.27)$$

Putting (2.2.25) and (2.2.26) into (2.2.27), we obtain

$$\begin{aligned}
\frac{\ln I_1(t)}{t} &= a_1 \left[-A + \left(d + \frac{\beta_1}{a_1} \right) \langle S \rangle - r_1 \langle I_1 \rangle \right] - (d + \alpha_1 + r_1) - \frac{1}{2}\sigma_1^2 + \frac{P(t)}{t} + \frac{a_1 \Phi}{t} \\
&= -a_1 A + (a_1 d + \beta_1) \langle S \rangle - a_1 r_1 \langle I_1 \rangle - (d + \alpha_1 + r_1) - \frac{1}{2}\sigma_1^2 + \frac{P(t)}{t} + \frac{a_1 \Phi}{t} \\
&= -a_1 A + (a_1 d + \beta_1) \left[\frac{A}{d} - \frac{(d + \alpha_1)}{d} \langle I_1 \rangle - \frac{(d + \alpha_2)}{d} \langle I_2 \rangle - \frac{\Theta}{dt} \right] \\
&\quad - a_1 r_1 \langle I_1 \rangle - (d + \alpha_1 + r_1) - \frac{1}{2}\sigma_1^2 + \frac{P(t)}{t} + \frac{a_1 \Phi}{t} \\
&= \left[\frac{A\beta_1}{d} - (d + \alpha_1 + r_1) - \frac{1}{2}\sigma_1^2 \right] - \left[a_1 d + a_1 \alpha_1 + \frac{\beta_1(d + \alpha_1)}{d} + a_1 r_1 \right] \langle I_1 \rangle \\
&\quad - (a_1 d + \beta_1) \frac{(d + \alpha_2)}{d} \langle I_2 \rangle - (a_1 d + \beta_1) \frac{\Theta}{dt} + \frac{P(t)}{t} + \frac{a_1 \Phi}{t}.
\end{aligned}$$
$$(2.2.28)$$

According to Lemma 2.3, Lemma 2.5 and Lemma 2.6, when $\mathcal{R}_1^* > 1$ and $\mathcal{R}_2^* < 1$, we have

$$\lim_{t \to \infty} \langle I_1 \rangle = \frac{\lambda}{\lambda_0} = \frac{\frac{A\beta_1}{d} - (d + \alpha_1 + r_1) - \frac{1}{2}\sigma_1^2}{a_1 d + a_1 \alpha_1 + \frac{\beta_1(d + \alpha_1)}{d} + a_1 r_1} = \frac{1}{a_1 + \frac{\beta_1(d + \alpha_1)}{d(d + \alpha_1 + r_1)}} (\mathcal{R}_1^* - 1) > 0.$$

This completes the proof of (i).

(ii) The proof of (ii) is similar to that of (i).

(iii) From model (2.2.2), we get

$$\begin{aligned}
\mathrm{d}S &= \left[A - dS - \frac{\beta_1 S(t - \tau_1) I_1}{1 + a_1 I_1} - \frac{\beta_2 S(t - \tau_2) I_2}{1 + a_2 I_2} + r_1 I_1 + r_2 I_2 \right] \mathrm{d}t + \sigma_3 S \mathrm{d}B_3(t) \\
&= \left[A - dS - \frac{\beta_1}{a_1} S(t - \tau_1) + \frac{1}{a_1} \frac{\beta_1 S(t - \tau_1)}{1 + a_1 I_1} - \frac{\beta_2}{a_2} S(t - \tau_2) \right. \\
&\quad \left. + \frac{1}{a_2} \frac{\beta_2 S(t - \tau_2)}{1 + a_2 I_2} + r_1 I_1 + r_2 I_2 \right] \mathrm{d}t + \sigma_3 S \mathrm{d}B_3(t).
\end{aligned}$$
$$(2.2.29)$$

Similar to (2.2.23)-(2.2.25), integrating (2.2.29) yields

$$\begin{aligned}
A &- \left(d + \frac{\beta_1}{a_1} + \frac{\beta_2}{a_2} \right) \langle S \rangle + \frac{1}{a_1 t} \int_0^t \frac{\beta_1 S(r - \tau_1)}{1 + a_1 I_1} \mathrm{d}r \\
&+ \frac{1}{a_2 t} \int_0^t \frac{\beta_2 S(r - \tau_2)}{1 + a_2 I_2} \mathrm{d}r + r_1 \langle I_1 \rangle + r_2 \langle I_2 \rangle \\
&= \frac{S(t) - S(0)}{t} - \frac{Q}{t} \triangleq \frac{\Psi}{t}.
\end{aligned}$$
$$(2.2.30)$$

From model (2.2.2), we also get

$$A - d \langle S \rangle - (d + \alpha_1) \langle I_1 \rangle - (d + \alpha_2) \langle I_2 \rangle \triangleq \frac{\Theta}{t}, \qquad (2.2.31)$$

where

$$\frac{\Theta}{t} = \frac{S(t) - S(0) + I_1(t) - I_1(0) + I_2(t) - I_2(0) - M_1(t) - M_2(t) - M_3(t)}{t}.$$

Define the function $V = \ln(a_2 I_1(t) + a_1 I_2(t))$. Similar to (i), using Itô's formula, we get

$$\frac{\ln(a_2 I_1(t) + a_1 I_2(t))}{t}$$
$$= \frac{a_2}{t} \int_0^t \frac{\beta_1 S(r - \tau_1)}{1 + a_1 I_1} dr + \frac{a_1}{t} \int_0^t \frac{\beta_2 S(r - \tau_2)}{1 + a_2 I_2} dr - a_2(d + \alpha_1 + r_1) \qquad (2.2.32)$$
$$- a_1(d + \alpha_2 + r_2) - \frac{1}{2} a_2 \sigma_1^2 - \frac{1}{2} a_1 \sigma_2^2 + \frac{a_2 M_1(t)}{t} + \frac{a_1 M_2(t)}{t}.$$

Substituting (2.2.30) and (2.2.31) into (2.2.32), we get

$$\frac{\ln(a_2 I_1(t) + a_1 I_2(t))}{t}$$
$$= -a_1 a_2 A + a_1 a_2 \left(d + \frac{\beta_1}{a_1} + \frac{\beta_2}{a_2} \right) \langle S \rangle - a_1 a_2 r_1 \langle I_1 \rangle - a_1 a_2 r_2 \langle I_2 \rangle + a_1 a_2 \frac{\Psi}{t}$$
$$- a_2(d + \alpha_1 + r_1) - a_1(d + \alpha_2 + r_2) - \frac{1}{2} a_2 \sigma_1^2 - \frac{1}{2} a_1 \sigma_2^2 + \frac{a_2 M_1(t)}{t} + \frac{a_1 M_2(t)}{t}$$
$$\triangleq -a_1 a_2 A - a_2(d + \alpha_1 + r_1) - a_1(d + \alpha_2 + r_2) - \frac{1}{2} a_2 \sigma_1^2 - \frac{1}{2} a_1 \sigma_2^2$$
$$+ (a_1 a_2 d + a_2 \beta_1 + a_1 \beta_2) \langle S \rangle - a_1 a_2 r_1 \langle I_1 \rangle - a_1 a_2 r_2 \langle I_2 \rangle + \frac{\Upsilon}{t}$$
$$= -a_1 a_2 A - a_2(d + \alpha_1 + r_1) - a_1(d + \alpha_2 + r_2) - \frac{1}{2} a_2 \sigma_1^2 - \frac{1}{2} a_1 \sigma_2^2$$
$$+ (a_1 a_2 d + a_2 \beta_1 + a_1 \beta_2) \left[\frac{A}{d} - \frac{(d + \alpha_1)}{d} \langle I_1 \rangle - \frac{(d + \alpha_2)}{d} \langle I_2 \rangle - \frac{\Theta}{dt} \right]$$
$$- a_1 a_2 r_1 \langle I_1 \rangle - a_1 a_2 r_2 \langle I_2 \rangle + \frac{\Upsilon}{t}$$
$$= \left[\frac{A}{d} (a_2 \beta_1 + a_1 \beta_2) - a_2(d + \alpha_1 + r_1) - a_1(d + \alpha_2 + r_2) - \frac{1}{2} a_2 \sigma_1^2 - \frac{1}{2} a_1 \sigma_2^2 \right]$$
$$- \left[(a_1 a_2 d + a_2 \beta_1 + a_1 \beta_2) \frac{(d + \alpha_1)}{d} + a_1 a_2 r_1 \right] \langle I_1 \rangle$$
$$- \left[(a_1 a_2 d + a_2 \beta_1 + a_1 \beta_2) \frac{(d + \alpha_2)}{d} + a_1 a_2 r_2 \right] \langle I_2 \rangle$$
$$- (a_1 a_2 d + a_2 \beta_1 + a_1 \beta_2) \frac{\Theta}{dt} + \frac{\Upsilon}{t}.$$
$$(2.2.33)$$

Then we obtain

$$\frac{\ln(a_2 I_1(t) + a_1 I_2(t))}{t}$$

$$\geqslant \left[\frac{A}{d} (a_2 \beta_1 + a_1 \beta_2) - a_2(d + \alpha_1 + r_1) - a_1(d + \alpha_2 + r_2) - \frac{1}{2} a_2 \sigma_1^2 - \frac{1}{2} a_1 \sigma_2^2 \right]$$

$$- \max\{m_1, m_2\} \left[a_2 \langle I_1 \rangle + a_1 \langle I_2 \rangle \right] - (a_1 a_2 d + a_2 \beta_1 + a_1 \beta_2) \frac{\Theta}{dt} + \frac{\Upsilon}{t},$$

$$(2.2.34)$$

where

$$m_1 \triangleq a_1(d + \alpha_1) + \frac{\beta_1}{d}(d + \alpha_1) + \frac{a_1 \beta_2 (d + \alpha_1)}{a_2 d} + a_1 r_1,$$

$$m_2 \triangleq a_2(d + \alpha_2) + \frac{\beta_2}{d}(d + \alpha_2) + \frac{a_2 \beta_1 (d + \alpha_2)}{a_1 d} + a_2 r_2.$$

By Lemma 2.3, Lemma 2.5 and Lemma 2.6, we take the limit on both sides of (2.2.34) to get

$$\liminf_{t \to \infty} \langle a_2 I_1(t) + a_1 I_2(t) \rangle$$

$$\geqslant \frac{\lambda}{\lambda_0} = \frac{\frac{A}{d}(a_2 \beta_1 + a_1 \beta_2) - a_2(d + \alpha_1 + r_1) - a_1(d + \alpha_2 + r_2) - \frac{1}{2}a_2 \sigma_1^2 - \frac{1}{2}a_1 \sigma_2^2}{\max\{m_1, m_2\}}$$

$$= \frac{1}{\max\{m_1, m_2\}} \left\{ a_2(\mathcal{R}_1^* - 1) + a_1(\mathcal{R}_2^* - 1) \right\} > 0.$$

This completes the proof. $\qquad\square$

From Theorem 2.8 and Theorem 2.9, we can claim that the thresholds $\mathcal{R}_i^* (i = 1, 2)$ of the model (2.3.2) can describe the persistence and extinction of two diseases. In other words, if $\mathcal{R}_1^* < 1$ and $\mathcal{R}_2^* < 1$, then the two infections diseases I_1 and I_2 of model (2.3.2) go extinct; if $\mathcal{R}_1^* > 1$ and $\mathcal{R}_2^* < 1$, then the disease I_2 goes extinct and the disease I_1 is permanent on average; if $\mathcal{R}_2^* > 1$ and $\mathcal{R}_1^* < 1$, then the disease I_1 goes extinct and the disease I_2 is permanent on average; if $\mathcal{R}_1^* > 1$ and $\mathcal{R}_2^* > 1$, then the two infections diseases I_1 and I_2 are permanent on average.

3. **A case study for Yunnan in China.** In this section, basing on the model (2.2.1) and (2.2.2), we do a case study for HIV/AIDS and Gonorrhea in Yunnan Province, China. The transmission routes of these two kinds of diseases are close to those of the main infected population. The main transmission modes are as follows: sexual transmission, blood transmission, mother to child transmission. According to the report data [26]: The cumulative number HIV positives reported at the end of September 2018 was 850,000, including 260,000 recorded deaths in China, and the estimated number living with HIV/AIDS was 36.9 million around the world. At present, there are many papers about the spread of HIV/AIDS. The basic mathematical models of HIV/AIDS in-host has been developed to describe interactions between immune system and viruses [21]. In [7] and [16], a class of HIV/AIDS model with time delay and a class of HIV/AIDS model with age structure are studied respectively. In [15], a cell-to-cell transmission model of HIV/AIDS is studied. There are few literatures about Gonorrhea, the way of transmission is similar to AIDS, and its harm is not as serious as AIDS. Gonorrhea is a purulent inflammatory disease of genitourinary system caused by Neisseria gonorrhoeae. It is also because the transmission route and AIDS are similar, so it is necessary to study the co-infection model of these two infectious diseases. Yunnan is located in southwest

TABLE 1. Cumulative total of reported HIV/AIDS cases and the number of Gonorrhea infections increased annually from 2007 to 2016 in Yunnan Province, China (see [26, 18])

Year	2007	2008	2009	2010	2011
HIV/AIDS	57325	64460	71852	78613	85999
Gonorrhea	2358	2230	1818	1819	1720
Year	2012	2013	2014	2015	2016
HIV/AIDS	92666	98555	104903	111351	117817
Gonorrhea	1893	1643	2104	3028	4098

TABLE 2. Parameters and numerical values chosen for the simulation

Parameters	Definition	Value	Source
A	Recruitment rate for the susceptible population	92136	Estimated
d	Natural mortality rate	0.0149	[6]
α_1	Death rate for HIV/AIDS	0.7114	[26]
α_2	Death rate for Gonorrhea	0.3	Estimated
r_1	Cure rate for HIV/AIDS	0.79	Estimated
r_2	Cure rate for Gonorrhea	0.99994	Estimated
β_1	Infection rate for HIV/AIDS	0.9	Estimated
β_2	Infection rate for Gonorrhea	0.25	Estimated
a_1	Inhibition rate of HIV/AIDS on transmission	0.9	Estimated
a_2	Inhibition rate of Gonorrhea on transmission	1	Estimated
τ_1	Incubation period of AIDS	8 year	[26]
τ_2	Incubation period of Gonorrhea	0	[18]
$S(0)$	Initial value of susceptible population	80000	Estimated
$I_1(0)$	Initial value of HIV/AIDS patients	57325	[26]
$I_2(0)$	Initial value of Gonorrhea patients	12358	Estimated

of China, bordering the countries of Myanmar, Laos and Vietnam. According to the sixth national census in 2011 [17], there are 45,596,000 people in Yunnan. From the cumulative number of HIV/AIDS infections in Yunnan Province in 2007 [26], combining with the number of newly increased infections and deaths of HIV/AIDS from 2007 to 2016 [18], the cumulative number of HIV/AIDS infections in Yunnan Province from 2007 to 2016 is obtained (see Table 1). In addition, the number of Gonorrhea infections increased annually from 2007 to 2016 in Yunnan Province [18] is shown in Table 1.

Using Eviews 7.0, we will test the stationarity of the data for the number of people infected with HIV/AIDS and Gonorrhea from 2007 to 2016 in Yunnan Province, respectively. The autocorrelation and partial correlation coefficients of the test results show that the data series is stable and the statistics are good. Next, the parameters of the model (2.2.2) are further determined for fitting. The specific idea is that part of the parameters are based on the known literatures and some biological values, and then, based on the data in Table 1 and the parameters obtained, the

least square method is used to fit the model to estimate the remaining parameters. For the natural mortality of people in Yunnan Province, we choose $d = \frac{1}{67} = 0.0149$ where 67 is the average life of people in Yunnan [6]. For HIV/AIDS and Gonorrhea patients' death rate $\alpha_i (i = 1, 2)$ in Yunnan, we know that $1 - e^{-\alpha_i t}$ is the death probability of HIV/AIDS and Gonorrhea patients. We can take $\alpha_1 = 0.7114$ (see [7]) and detailed parameters values are shown in Table 2. We take 2007 as the initial time $t = 0$, according to the cumulative number of HIV/AIDS infections in Yunnan Province in 2007 [26], so we take $I_1(0) = 57325$, and the other two initial values $S(0)$ and $I_2(0)$ are obtained by estimation, as shown in Table 2. The number of susceptible persons, the population infected with HIV/AIDS and the population infected with Gonorrhea are obtained by numerical fitting using the parameters of Table 2, as shown in Fig.1, including the comparison of fitting data and statistical data.

When the parameters in Table 2 are substituted into the two thresholds of the model (2.2.1), they are both greater than unity, that is, both diseases are persistent. To illustrate the significance of model (2.2.2) in disease control, we first describe the dependence of each parameter in thresholds $\mathcal{R}_i^* (i = 1, 2)$ of model (2.2.2), namely the partial rank correlation coefficients (PRCCs). As shown in Fig.2. Considering the objective conditions of medical equipment and human life span, combining with the results of PRCC, we can adopt four ways to control the two diseases: (1) improving the recovery rate of diseases r_i; (2) reducing the infections rate of infectious diseases β_i; (3) reducing the number of imports to susceptible persons A and (4) using big noises $\sigma_i (i = 1, 2)$.

According to the results of PRCC, we take the following values: A = 400, r_1 = 0.75, r_2 = 0.2, d = 0.0149, β_1 = 0.00007, β_2 = 0.00002, α_1 = 0.7114, α_2 = 0.1, a_1 = 0.0001, a_2 = 0.0001, σ_1 = 0.95, σ_2 = 0.9, σ_3 = 0.2, $\tau_1 = \tau_2 = 0.5$. It follows that $\mathcal{R}_1 = 1.2729 > 1 > \mathcal{R}_1^* = 0.9672$, $\mathcal{R}_2 = 1.7050 > 1 > \mathcal{R}_2^* = 0.4189$. Under these conditions, the two diseases of the deterministic model (2.2.1) will be persistent, whereas the two diseases described by the model (2.2.2) will be extinct (see Fig.3 and Fig.4).
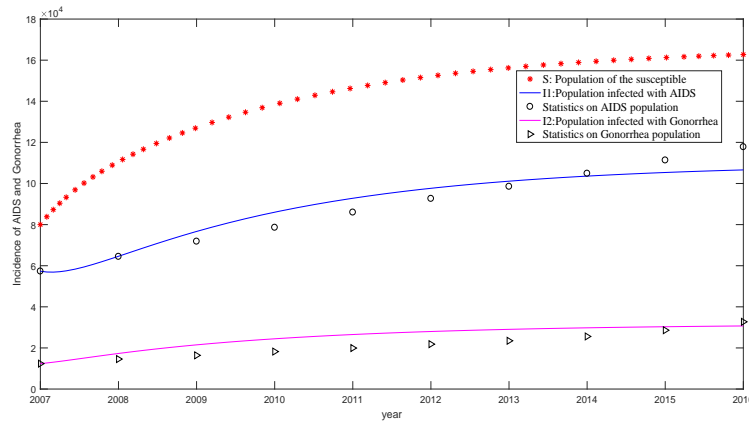

FIGURE 1. The model (2.2.2) is simulated by the parameters values in Table 2, and compared with the HIV/AIDS and Gonorrhea data in Yunnan Province from 2007 to 2016.
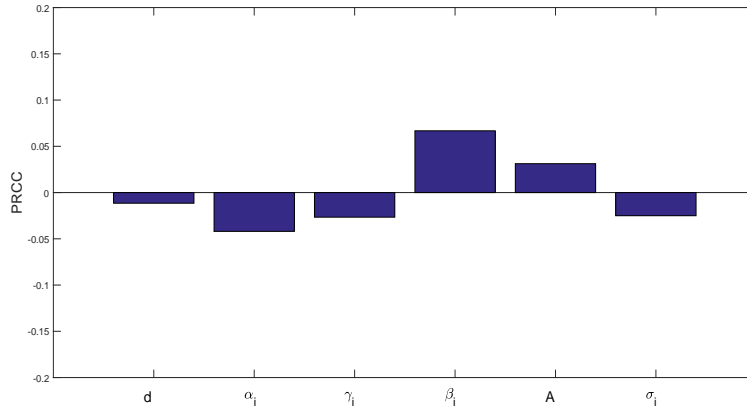
FIGURE 2. Partial rank correlation coefficients(PRCCs) results for the dependence of $\mathcal{R}_i^*$ on each parameter.
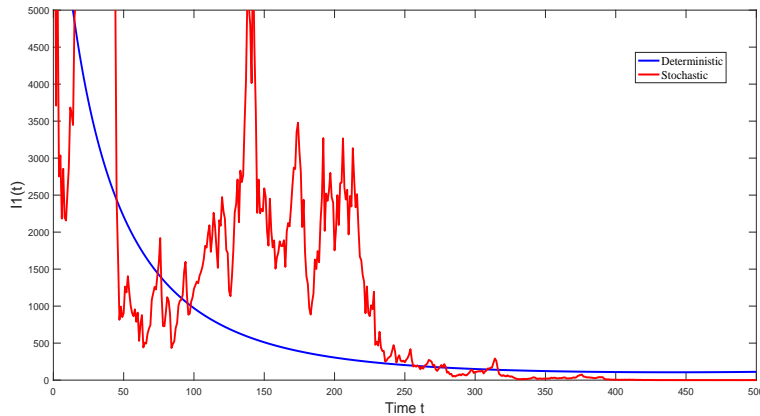

FIGURE 3. When $\mathcal{R}_1 = 1.2729 > 1 > \mathcal{R}_1^* = 0.9672$, model (2.2.1) describes HIV/AIDS infection I_1 will be persistent, but stochastic differential equation with time delay model (2.2.2) describes HIV/AIDS infection I_1 will be extinct.

4. **Discussions.** The fluctuation of natural environment will bring variability to biological system [20]. And environmental changes have a vital impact on the development of epidemics. Variability of temperature and rainfall may cause significant fluctuations in the dynamics of pathogenic fungi [22, 8]. In terms of human disease, the nature of epidemic spread and growth is inherently random due to the unpredictability of person-to-person contacts [11, 24]. Therefore, the variability and randomness of the environment are introduced into the epidemic model [20]. In general, the threshold of the model is a very important quantity for theoretical analysis of differential equation models describing infectious diseases. That is to say,
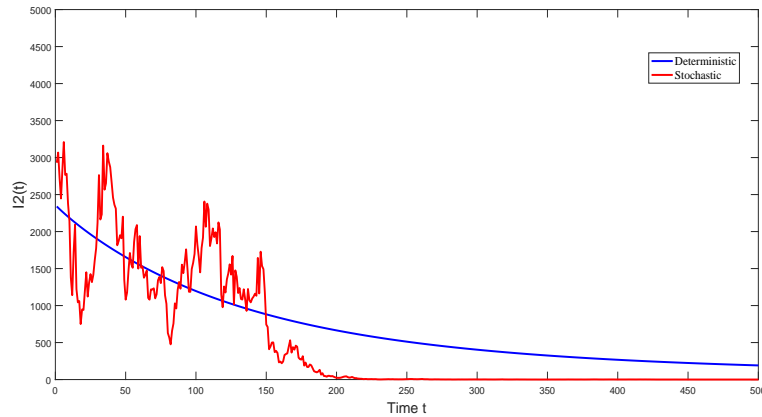
FIGURE 4. When $\mathcal{R}_2 = 1.7050 > 1 > \mathcal{R}_2^* = 0.4189$, model (2.2.1) describes Gonorrhea infection I_2 will be persistent, but stochastic differential equation with time delay model (2.2.2) describes Gonorrhea infection I_2 will be extinct.

the relationship between the threshold and 1 is used to analyze whether the disease is extinct or not. Therefore, in this paper, we discuss two classes of differential equations models. Specifically in each class of models, considering the introduction of randomness and time-delays, the change of infection rate, the existence of immunity loss and so on, then we theoretically analyze the thresholds changes in each class. We obtain the sufficient conditions for the extinction and persistence of diseases. In addition, we do a case study of (2.2.1) and (2.2.2), and we also carry out some numerical simulations aiming to HIV/AIDS and Gonorrhea transmission in Yunnan by using the models.

Through the case study, the results of numerical simulation and theoretical analysis are consistent, i.e., when $\mathcal{R}_i^* > 1$ $(i = 1, 2)$, the diseases will be persistent; when $\mathcal{R}_i^* < 1$ $(i = 1, 2)$, the diseases will be extinct. By comparing the thresholds of model (2.2.1) and model (2.2.2), and combining with the numerical simulations results, its are found that there are always $\mathcal{R}_i > \mathcal{R}_i^*$ $(i = 1, 2)$. Therefore, we are mostly concerned about the situations $\mathcal{R}_i > 1 > \mathcal{R}_i^*$ $(i = 1, 2)$, which are the significances of stochastic differential equations in controlling infectious diseases. Finally, we discuss some biological implications and focus on the impact of some key model parameters, the strategies of controlling HIV/AIDS and Gonorrhea are given, i.e., controlling the spread of infectious diseases by improving the recovery rate of diseases r_i, reducing the infections rate of infectious diseases β_i, declining the number of imports to susceptible people A and using big noises $\sigma_i (i = 1, 2)$.

Another possible and important extension for future work is that there is a class of stochastic model and its corresponding deterministic model, and the threshold of the stochastic model is larger than that of the deterministic model. That is, when the threshold of the stochastic model is greater than 1 and then greater than the threshold of the deterministic model, the disease of the deterministic model will be extinct, but the disease of the stochastic model will be persistent. This is the risk of introducing random noises into the deterministic models for the infectious

diseases models, but for the population models, this can maintain the growth of the population, so it is very meaningful to study this problem. All the aforementioned possible extensions are interesting, biologically important but yet mathematically challenging, and we have to leave them for future research projects.

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