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PERIODIC SOLUTION AND STATIONARY DISTRIBUTION OF A STOCHASTIC EPIDEMIC MODEL WITH TWO DIFFERENT EPIDEMICS AND DIFFERENT EPIDEMIOLOGICAL FRAMEWORKS

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Abstract. In epidemiology, more than one infectious disease can pose a risk to the host population. This area of study has attracted researchers in recent times. In this article, we have considered an epidemic model that incorporates two different transmission techniques, namely SIR and SIRS. The considered deterministic model has been perturbed stochastically at transmission rates. The analysis has been done for the resulting stochastic model. Firstly, we explore the existence of the positive T-periodic solution for the stochastic system. We determine that the non-autonomous periodic version of the system with white noise has a positive periodic solution using the Lyapunov function and Khasminskii theory. In addition, we analyze the positive recurrence of the system. The results obtained in this article give the idea that reducing the white noise in the stochastic model is critical for observing positive T-periodic solutions and positive recurrence. Finally, we present some examples and perform numerical simulations to validate the theoretical results established in this study.

1. Introduction

To understand how different diseases interact or spread in a community is tricky for the researchers studying diseases. When multiple diseases spread at the same time, it gets even more complicated. Therefore, it is evident to introduce some special types of modeling techniques to acknowledge how two diseases spread together in a community.

Deterministic models, characterized by their use of fixed parameters and precise equations, have been instrumental in laying the groundwork for our understanding of how diseases spread through populations. These models, based on differential equations, have provided valuable insights into disease dynamics, transmission rates, and the impact of interventions in controlling epidemics. For instance, in [8], a discrete time model has been considered by Hamer et al. They investigated the evolution of recurrent measles outbreaks. Kermack et al. [12] conducted a mathematical analysis of the dynamics of the epidemics within a homogeneous population, establishing a threshold condition for outbreaks and analyzing how an epidemic progresses over time. Their work established the foundation for compartmental models such as the SIR model, which divided the population into susceptible, infected and recovered categories. Further, they developed the threshold theory and constructed the traditional SIS epidemic model [13]. Nieto investigated a deterministic SIR framework enhanced with anti-infectious control strategies, such as vaccination and treatment, and demonstrated how these interventions could significantly alter the epidemic trajectory through numerical simulations and analytical insights [28]. For a long period, researchers have relied on the deterministic model to investigate infectious diseases. Several studies have been done to investigate the dynamical behavior; see [9, 17, 18, 19, 25].

While deterministic models offer a solid framework for studying disease spread, they often simplify the inherent randomness and variability present in real-world epidemics, leading to an increased interest in stochastic modeling approaches that better capture the complexities and uncertainties of disease transmission dynamics. Stochastic mathematical models of epidemics include

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uncertainty or unpredictability in the spread of infectious diseases. In the context of epidemiology, the use of stochastic models developed as a response to the limitations of deterministic models in capturing the inherent variability and random occurrences seen in actual disease outbreaks. The inclusion of randomness into models, which yielded a more realistic portrayal of the dynamics of disease spread, is what made the stochastic approach popular.

In the mid 19^{th} century, researchers started finding challenges dealing with random events and uncertainty in various domains, which is when stochastic modeling was first introduced. However, in the early to mid 20^{th} century, a significant increase in the widespread use and formalization of stochastic modeling, especially in the field of sciences, can be seen. Several contributions have been made by stochastic models. For instance, Liu et al. [20] studied an HBV infection model with stochastic perturbation incorporating logistic growth. They established the necessary condition for the disease's extinction and the presence of the ergodic stationary distribution. Bao et al. [3] conducted an analysis on stochastic SIRS model including interval parameters and established the definition for the stochastic basic reproduction number. Kuang et al. [16] investigated a stochastic SIRS epidemic model incorporating saturated incidence. They deduced that smaller white noise is required for the persistence of the epidemic and large noise will prevent the epidemic from spreading. Zinhi et al. [34] investigated a stochastic SIRS model and established conditions for the existence of stationary distributions and disease extinction. Similarly, Hening et al. [10] analyzed the long-term behavior of SIQRS models and provided rigorous stochastic stability results. For vector-borne and zoonotic diseases, Dutta et al. [6] explored the dynamics of the Nipah virus, focusing on equilibrium analysis, sensitivity, and uncertainty quantification. From a control-theoretic perspective, Russell and Cunniffe [31] highlighted a counterintuitive outcome where optimal control may hinder disease eradication in an environment with the stochastic domain. Liu et al. [21] analyzed a stochastic prey-predator model and determined the sufficient conditions for the extinction of the predator population. Therefore, in recent times, many studies have been done on stochastic epidemic models and researchers have demonstrated how environmental noise affects population model dynamics (see [11, 22, 7]).

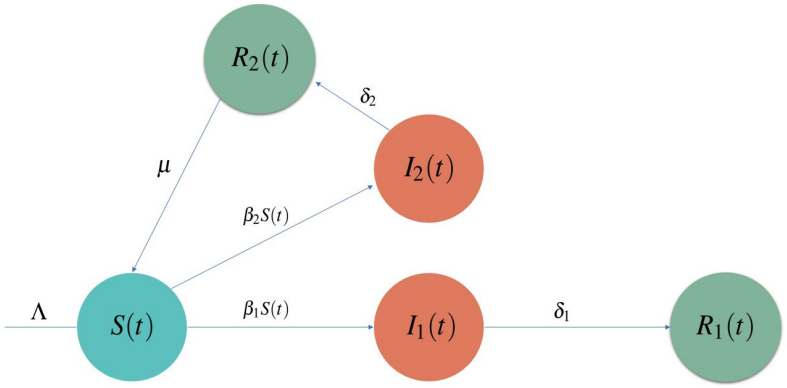

FIGURE 1. Population flow for the disease without interaction effect

In epidemiology, a single disease outbreak is the main focus of majority of the epidemic models, even though multiple diseases can generate an epidemic. There are instances when two major health issues occur simultaneously. It is same as dealing with two difficult situations together, which makes things even more difficult for everyone. Moreover, adding randomness to a two-pathogen model helps to better capture real-world disease dynamics. Random effects, like environmental or demographic changes, can affect how diseases spread, persist, or die out. These features cannot be fully explained by models with only one pathogen or by purely deterministic models. Several analysis has been done previously on double epidemics. Chang et al. [5] proposed a hypothesis for a twofold epidemic asymmetry and constructed an SIRS epidemic model with two

distinct non-linear saturation incidence rates. A double epidemic hypothesis-based delayed SIR disease transmission model has been adopted by Meng [24] and Meng et al. [26]. In [33, 27, 30], the authors have considered a stochastic epidemic model for SIS that includes double epidemic hypothesis with a saturated incidence rate.

Motivated by the above-mentioned works, in our study, we aim to more accurately reflect real population dynamics by integrating two different epidemiological frameworks: SIR and SIRS models. To illustrate this, we consider two infectious diseases, for example, measles, which is well-categorized by the SIR epidemic model due to the development of long-lasting immunity after infection, and influenza, which is often modeled using the SIRS framework because individuals can lose immunity over time and become susceptible again. These examples allow us to capture different aspects of infection and immunity dynamics within a single modeling framework. In Figure 1, we have used two distinct transmission techniques to illustrate the disease spread model. The deterministic model resulting from the transformation of the model dynamics depicted in Figure 1 is involving a system of first order differential equations that results from converting the model dynamics shown in Figure 1 into a mathematical model

$$\begin{aligned}
\frac{d\mathcal{S}(t)}{dt} &= \Lambda - \beta_1\mathcal{S}(t)\mathcal{I}_1(t) - \beta_2\mathcal{S}(t)\mathcal{I}_2(t) - \gamma\mathcal{S}(t) + \mu\mathcal{R}_2(t),\\
\frac{d\mathcal{I}_1(t)}{dt} &= \beta_1\mathcal{S}(t)\mathcal{I}_1(t) - \gamma_1\mathcal{I}_1(t) - \delta_1\mathcal{I}_1(t),\\
\frac{d\mathcal{I}_2(t)}{dt} &= \beta_2\mathcal{S}(t)\mathcal{I}_2(t) - \gamma_2\mathcal{I}_2(t) - \delta_2\mathcal{I}_2(t),\\
\frac{d\mathcal{R}_1(t)}{dt} &= \delta_1\mathcal{I}_1(t) - \gamma_3\mathcal{R}_1(t),\\
\frac{d\mathcal{R}_2(t)}{dt} &= \delta_2\mathcal{I}_2(t) - \gamma_4\mathcal{R}_2(t) - \mu\mathcal{R}_2(t).
\end{aligned} \tag{1.1}$$

Here the number of people who are susceptible to the disease at time t is denoted by $\mathcal{S}(t)$, and the number of people who are infected with the first and second diseases at time t are denoted by $\mathcal{I}_1(t)$ and $\mathcal{I}_2(t)$, respectively. $\mathcal{R}_1(t)$ and $\mathcal{R}_2(t)$ denote the number of individuals who have recovered from the infections with first and second disease at time t, respectively. It is important to observe that all of the parameters of the system (1.1) are positive. The birth and immigration rate of the population is denoted by Λ. The infection transmission coefficient rates by the first and second diseases are denoted by β_1 and β_2, respectively, while γ, γ_1, γ_2, γ_3, and γ_4 represent the mortality rates of susceptible individuals, individuals infected by the first disease, individuals infected by the second disease, individuals recovered from the first disease, and individuals recovered from the second disease, respectively. δ_1 and δ_2 are the recovery rates for first and second diseases respectively. Lastly, μ stands for the immunity loss ratio for the second disease.

Note that all the parameters Λ, β_1, β_2, γ, γ_1, γ_2, γ_3, γ_4, δ_1, δ_2 and μ are constants.

Furthermore, in terms of biology, it makes sense to assume that

$$\gamma \le \min\{\gamma_1, \gamma_2, \gamma_3, \gamma_4\}. \tag{1.2}$$

Remark 1.1. In classical models, the competitive exclusion principle states that two infections giving complete cross-immunity cannot co-exist. However, in our model, co-existence becomes possible because we combine the SIR and SIRS structures. In the SIRS framework, recovered individuals can lose immunity and become susceptible again, which helps both infections persist. This is further supported by the stability analysis of the deterministic system given in Section 3.

It is assumed that environmental noise primarily perturbs infection transmission rates β_1 and β_2 by $\beta_1 + \sigma_1\dot{B}_1(t)$ and $\beta_2 + \sigma_2\dot{B}_2(t)$, where $\dot{B}_i(t)$ for $i = 1, 2$ are standard Brownian motions satisfying $B_i(0) = 0$ and are mutually independent. The term $\dot{B}_i(t)$ denotes the formal time derivative of $B_i(t)$, representing white noise, for $i = 1, 2$. Furthermore, the parameters $\sigma_{ij}^2 > 0$ represent the intensities of the respective white noise, $i, j = 1, 2$. Using these perturbations, we

derive the subsequent stochastic model that corresponds to model (1.1):

$$\begin{aligned}
d\mathcal{S}(t) &= \big[\Lambda - \beta_1\mathcal{S}(t)\mathcal{I}_1(t) - \beta_2\mathcal{S}(t)\mathcal{I}_2(t) - \gamma\mathcal{S}(t) + \mu\mathcal{R}_2(t)\big]dt - \sigma_1\mathcal{S}(t)\mathcal{I}_1(t)dB_1(t)\\
&\quad - \sigma_2\mathcal{S}(t)\mathcal{I}_2(t)dB_2(t),\\
d\mathcal{I}_1(t) &= [\beta_1\mathcal{S}(t)\mathcal{I}_1(t) - \gamma_1\mathcal{I}_1(t) - \delta_1\mathcal{I}_1(t)]dt + \sigma_1\mathcal{S}(t)\mathcal{I}_1(t)dB_1(t),\\
d\mathcal{I}_2(t) &= [\beta_2\mathcal{S}(t)\mathcal{I}_2(t) - \gamma_2\mathcal{I}_2(t) - \delta_2\mathcal{I}_2(t)]dt + \sigma_2\mathcal{S}(t)\mathcal{I}_2(t)dB_2(t),\\
d\mathcal{R}_1(t) &= [\delta_1\mathcal{I}_1(t) - \gamma_3\mathcal{R}_1(t)]dt,\\
d\mathcal{R}_2(t) &= [\delta_2\mathcal{I}_2(t) - \gamma_4\mathcal{R}_2(t) - \mu\mathcal{R}_2(t)]dt.
\end{aligned} \tag{1.3}$$

Note that the stochastic perturbations are incorporated via multiplicative noise terms of the form $\beta_j + \sigma_j\dot{B}_j(t)$, where β_j for $j = 1, 2$ remains a fixed, strictly positive parameter. This ensures that the transmission rate remains biologically meaningful (non-negative) at all times.

To our knowledge, no study has been conducted regarding the existence of periodic solution and stationary distribution of the stochastic system (1.3). Although, the existence and uniqueness of solutions of the stochastic system (1.3) has been validated in [32]. Our goal in this article is to study the existence of positive T-periodic solutions and the stationary distribution of the solution to the system (1.3).

The remaining sections of the article are organized as follows. In Section 2, we present some preliminaries required to establish our results. In Section 3, we perform a dynamical analysis of the deterministic system, including the stability analysis of equilibria. Section 4 and Section 5 are devoted to the main results of this article, where we establish sufficient conditions for the existence of positive T-periodic solutions and positive recurrence for the stochastic system (1.3). Numerical simulations are provided in Section 6 to validate the theoretical findings. Finally, conclusions are drawn in Section 7.

2. Preliminaries

It is obvious to observe that the study on epidemic over any population starts when there is an epidemic outbreak happens and hence

$$(\mathcal{S}(0), \mathcal{I}_1(0), \mathcal{I}_2(0), \mathcal{R}_1(0), \mathcal{R}_2(0)) \in \Delta, \tag{2.1}$$

which guarantees that the population is positive. For the definition of Δ, we refer to the following remark:

Remark 2.1 ([4])**.** We denote the total population as $N(t)$. Thus $N(t)$ is given as $N(t) = \mathcal{S}(t) + \mathcal{I}_1(t) + \mathcal{I}_2(t) + \mathcal{R}_1(t) + \mathcal{R}_2(t)$.

We may infer from the system (1.3) that

$$\frac{dN(t)}{dt} = [\Lambda - \gamma\mathcal{S}(t) - \gamma_1\mathcal{I}_1(t) - \gamma_2\mathcal{I}_2(t) - \gamma_3\mathcal{R}_1(t) - \gamma_4\mathcal{R}_2(t)].$$

Applying inequality (1.2), we obtain

$$\frac{dN(t)}{dt} \le [\Lambda - \gamma N(t)].$$

Therefore,

$$N(t) \le \Big(N(0) - \frac{\Lambda}{\gamma}\Big)e^{-\gamma t} + \frac{\Lambda}{\gamma} \le \max\Big(N(0), \frac{\Lambda}{\gamma}\Big) := \mathbf{M}. \tag{2.2}$$

where $\mathbf{M}$ is a positive constant.

Consider a set

$$\Delta = \big\{\big((\mathcal{S}(t), \mathcal{I}_1(t), \mathcal{I}_2(t), \mathcal{R}_1(t), \mathcal{R}_2(t)\big) \in \mathbb{R}^+_5 : \mathcal{S}(t) + \mathcal{I}_1(t) + \mathcal{I}_2(t) + \mathcal{R}_1(t) + \mathcal{R}_2(t) \le \frac{\Lambda}{\gamma}\big\}. \tag{2.3}$$

Thus, the bound $N(t) \le \frac{\Lambda}{\gamma}$ is verified by the fact that $(\mathcal{S}(0), \mathcal{I}_1(0), \mathcal{I}_2(0), \mathcal{R}_1(0), \mathcal{R}_2(0)) \in \Delta$.

Hence, the total population $N(t)$ is bounded.

Notation

(1) Throughout this article, $\langle X\rangle_T$ represents the time average of the process $X(t)$ over the interval $[0,T]$. Formally

$$\langle X\rangle_T = \frac{1}{T}\int_0^T X(t)dt.$$

(2) In this paper, "a.s." or "*a.s.*" refers to almost surely, indicating that the specified property holds with probability one.
(3) For any two real numbers c and d, the symbol $c\wedge d$ represents their minimum, while $c\vee d$ represents their maximum.

Definition 2.2 ([14])**.** Let $t_1, t_2, \dots, t_n$ be any arbitrary finite sequence and $X(t)$ be a stochastic process. If $X(t_1+h), X(t_2+h), \dots, X(t_n+h)$, the joint distribution of random variables is independent of h, where $h = mT$ for $(m = \pm1, \pm2, \dots)$, then $X(t)$ is regarded periodic with period T.

Lemma 2.3 ([14])**.** *Consider the stochastic differential equation defined as follows:*

$$dy(t) = f(t, y(t))dt + \sigma(t, y(t))dB(t). \tag{2.4}$$

The functions $f(t,y(t))$ and $\sigma(t,y(t))$ defined in (2.4) *are periodic in t with periods T and $y\in\mathbb{R}^n$. Let us assume that* (2.4) *possesses a unique global solution. Suppose that $U(t,y(t))\in\mathbb{C}^2$ is a function that is periodic in t with period T as well as holds the properties mentioned below:*

(i)

$$\inf_{|y|>R} U(t, y(t)) \to \infty \quad as\ R\to\infty, \tag{2.5}$$

(ii) *outside some compact set, we have*

$$LU(t, y(t)) \le -1, \tag{2.6}$$

where L is the operator

$$L = \frac{\partial}{\partial t} + \sum_{i=1}^{n} f_i(t,y)\frac{\partial}{\partial y_i} + \frac{1}{2}\sum_{i,j=1^n} c_{i,j}(t,y)\frac{\partial^2}{\partial y_i y_j}.$$

Then, system (2.4) *possesses a T-periodic solution.*

We now present some useful information about Markov chains and stochastic differential equations. Let $Q = \{1, 2, \dots, N\}$ represent the state space and $\zeta(t)$ be the continuous-time Markov chain that switches between the N regimes. In [23], Mao and Yuan proposed the following description of the Markov process $(y(t), \zeta(t)) \in \mathbb{R}^n_+ \times Q$:

$$\begin{aligned} dy(t) &= P(y(t),\zeta(t))dt + \sigma(y(t),\zeta(t))dB(t),\\ y(0) &= y_0 \in \mathbb{R}^n_+, \zeta(0) = \bar{u} \in Q, \end{aligned}$$

where $P(y(t),\zeta(t)) : \mathbb{R}^n \times Q \to \mathbb{R}^n, \sigma(x(t),\zeta(t)) : \mathbb{R}^n \times Q \to \mathbb{R}^{n\times d}$.

Furthermore, we define a linear operator L for a function $V(x,k)\in C^2(\mathbb{R}^n)\times Q$, by

$$\begin{aligned} LV(y,l) &= \sum_{i=1}^{n} b_i(y,l)\frac{\partial V(y,l)}{\partial y_i} + \sum_{i,j=1}^{n}\frac{c_{ij}(y,l)}{2}\frac{\partial^2 V(y,l)}{\partial y_i y_j} + \sum_{i=1}^{n} q_{ki}(x)V(x,i), c(y,l)\\ &= \sigma(y,l)\sigma^T(y,l). \end{aligned}$$

Definition 2.4. Given a set D and τ_D is the first reach time of D for the process X_t^x. If $P(\tau_D < \infty) = 1$ for each $x\notin D$, the Markov process X_t^x with $X_0 = x$ is recurrent on D. Moreover, τ_D is defined as follows

$$\tau_D = inf\{t > 0, X_t^x \in D\}.$$

Furthermore, if $\mathbb{E}(\tau_D) < \infty$ for every $x\notin D$, the process X_t^x is called positive recurrent on D.

3. Dynamical analysis of the deterministic system

This section explores the existence and uniqueness of a global positive solution by employing the Picard-Lindelöf theorem and related results of classical theory of ordinary differential equations [29]. Additionally, we establish conditions that ensure both local and global asymptotic stability of equilibrium points, using Lyapunov's direct method.

3.1. Existence and uniqueness of the positive solution.

This section focuses on analyzing the positivity and boundedness of solutions for the deterministic system (1.1) under the given initial condition

$$(\mathcal{S}(0),\mathcal{I}_1(0),\mathcal{I}_2(0),\mathcal{R}_1(0),\mathcal{R}_2(0)) \in \Xi. \tag{3.1}$$

Since system (1.1) has locally Lipschitz continuous coefficients, it guarantees the existence of a unique maximal local solution $(\mathcal{S}(t),\mathcal{I}_1(t),\mathcal{I}_2(t),\mathcal{R}_1(t),\mathcal{R}_2(t))$ for all $t \in [0,\tau)$, where τ denotes the explosion time. Also, we ensure that the initial condition (3.1) holds.

To establish the global existence and positivity of the solution, we now present the following theorem, which ensures that the solution $(\mathcal{S}(t),\mathcal{I}_1(t),\mathcal{I}_2(t),\mathcal{R}_1(t),\mathcal{R}_2(t))$ remains well defined for all $t \geq 0$ without breaking down in a finite time.

Theorem 3.1. *For the initial condition* (3.1)*, the solution of the system* (1.1) *exists globally and remains positive for all* $t \geq 0$*.*

Proof. Consider $(\mathcal{S}(t),\mathcal{I}_1(t),\mathcal{I}_2(t),\mathcal{R}_1(t),\mathcal{R}_2(t))$ as a solution of the system (1.1) under the initial condition (3.1). We first establish the positivity of $\mathcal{S}(t)$.

Suppose, for contradiction, that there exists a first time t_1 such that $S(t_1) = 0$. From the first equation of system (1.1), we have

$$\frac{d\mathcal{S}(t)}{dt}\Big|_{t=t_1} = \Lambda > 0.$$

This indicates that $\mathcal{S}(t)$ becomes negative for some $t \in (t_1 - h, t_1)$, where $h > 0$ is a sufficiently small real number, which contradicts the assumption that $\mathcal{S}(t) > 0$ for all $t \in [0, t_1)$. Hence, we conclude that $\mathcal{S}(t) > 0$ for all $t \in [0,\tau)$.

Next, we analyze the positivity of $\mathcal{I}_1(t)$ and $\mathcal{I}_2(t)$. From the second and third equations of the system (1.1), it follows that

$$\mathcal{I}_j(t) = \mathcal{I}_j(0) exp\Big(\int_0^t \big[\beta_1\mathcal{S}(v) - (\gamma_1 + \delta_1)\big]dv\Big), \quad j = 1, 2.$$

Since the exponential function is always positive and $\mathcal{I}_j(0) > 0$ from the initial condition (3.1), it follows that $\mathcal{I}_j(t) > 0$ for all $t \in [0,\tau)$ and $j = 1, 2$.

Lastly, we verify the positivity of $\mathcal{R}(t)$. From the fourth and fifth equations of system (1.1), we obtain

$$\begin{aligned} d\mathcal{R}_1(t) &\geq -\gamma_3\mathcal{R}_1(t) \quad \text{for all } t \in [0,\tau),\\ d\mathcal{R}_2(t) &\geq (\gamma_4 + \mu)\mathcal{R}_1(t) \quad \text{for all } t \in [0,\tau), \end{aligned}$$

which can further be deduced as

$$\begin{aligned} \mathcal{R}_1(t) &\geq \mathcal{R}_1(0)e^{-\gamma_3 t} \quad \text{for all } t \in [0,\tau),\\ \mathcal{R}_2(t) &\geq \mathcal{R}_2(0)e^{(-\gamma_4+\mu)t} \quad \text{for all } t \in [0,\tau). \end{aligned}$$

Since $\mathcal{R}_1(0) > 0$ and $\mathcal{R}_2(0) > 0$ from (3.1), we conclude that $\mathcal{R}_j(t) > 0$ for all $t \in [0,\tau)$ and $j = 1, 2$.

As the positivity of the solution $(\mathcal{S}(t),\mathcal{I}_1(t),\mathcal{I}_2(t),\mathcal{R}_1(t),\mathcal{R}_2(t))$ has been established, it remains to show that the solution is globally defined. The boundedness of the solution, as established by inequality (2.2), implies that it cannot blow up in finite time, which confirms that $\tau = \infty$. Therefore, the solution $(\mathcal{S}(t),\mathcal{I}_1(t),\mathcal{I}_2(t),\mathcal{R}_1(t),\mathcal{R}_2(t))$ remains positive and exists for all $t \geq 0$. This completes the proof. □

3.2. **Equilibrium analysis and local stability of equilibrium.** To begin our equilibrium analysis, we first determine the equilibrium points of the system (1.1). By solving the system for steady-state conditions, we obtain the following equilibrium points:

$$\begin{aligned}
E_0^* &= \Big(\frac{\Lambda}{\gamma}, 0, 0, 0, 0\Big),\\
E_1^* &= \Big(\frac{\gamma_1+\delta_1}{\beta_1}, \frac{-\gamma\gamma_1-\gamma\delta_1+\beta_1\Lambda}{\beta(\gamma_1+\delta_1)}, 0, \frac{-\delta_1(\gamma\gamma_1+\gamma\delta_1-\beta_1\Lambda)}{\beta_1\gamma_3(\gamma_1+\delta_1)}, 0\Big)\\
&= \Big(\frac{\gamma_1+\delta_1}{\beta_1}, \frac{\gamma(\mathfrak{R}_1-1)}{\beta}, 0, \frac{\delta_1\gamma(\mathfrak{R}_1-1)}{\beta_1\gamma_3}, 0\Big),\\
E_2^* &= \Big(\frac{\gamma_2+\delta_2}{\beta_2}, 0, \frac{-(\gamma\gamma_2+\gamma\delta_2-\beta_2\Lambda)(\gamma_4+\mu)}{\beta_2(\gamma_2\gamma_4+\gamma_4\delta_2+\gamma_2\mu)}, 0, \frac{-\delta_2(\gamma\gamma_2+\gamma\delta_2-\beta_2\Lambda)}{\beta_2(\gamma_2\gamma_4+\gamma_4\delta_2+\gamma_2\mu)}\Big)\\
&= \Big(\frac{\gamma_2+\delta_2}{\beta_2}, 0, \frac{\gamma(\gamma_2+\delta_2)(\gamma_4+\mu)(\mathfrak{R}_2-1)}{\beta_2(\gamma_2\gamma_4+\gamma_4\delta_2+\gamma_2\mu)}, 0, \frac{\gamma\delta_2(\gamma_2+\delta_2)(\mathfrak{R}_2-1)}{\beta_2(\gamma_2\gamma_4+\gamma_4\delta_2+\gamma_2\mu)}\Big),
\end{aligned}$$

where $\mathfrak{R}_1 = \frac{\beta_1\Lambda}{\gamma(\gamma_1+\delta_1)}$ and $\mathfrak{R}_2 = \frac{\beta_2\Lambda}{\gamma(\gamma_2+\delta_2)}$. Using the threshold parameters $\mathfrak{R}_1$ and $\mathfrak{R}_2$, we can determine the local stability of the equilibria.

Remark 3.2. We now define the invasion reproduction numbers for the system (1.1) corresponding to each disease as follows

$$\begin{aligned}
\mathcal{R}_2^{inv} &= \frac{\beta_2 S^*}{\gamma_2+\delta_2}; \quad S^* = \frac{\gamma_1+\delta_1}{\beta_1},\\
\mathcal{R}_1^{inv} &= \frac{\beta_1 S^{**}}{\gamma_1+\delta_1}; \quad S^* = \frac{\gamma_2+\delta_2}{\beta_2}.
\end{aligned}$$

Here, S^* and S^{**} represent the susceptible population at the equilibria E_1^* and E_2^*, respectively. Biologically, $\mathcal{R}_2^{inv}$ measures the average number of secondary infections caused by an individual of second disease when introduced into a population where first disease is already endemic (and similarly for $\mathcal{R}_1^{inv}$).

If $\mathcal{R}_2^{inv} < 1$, second disease cannot invade, similarly $\mathcal{R}_1^{inv} < 1$ prevents invasion by first disease.

We summarize the stability results in the following theorem.

Theorem 3.3. *Consider system* (1.1)*, the local stability of its equilibrium points is characterized as follows:*

(1) *The disease-free equilibrium* $E_0^* = \Big(\frac{\Lambda}{\gamma}, 0, 0, 0, 0\Big)$ *is locally asymptotically stable if* $\mathfrak{R}_1 < 1$ *and* $\mathfrak{R}_2 < 1$.
(2) *The equilibrium* E_1^* *is locally asymptotically stable if* $\mathfrak{R}_1 > 1$ *and* $\mathcal{R}_2^{inv} < 1$.
(3) *The equilibrium* E_2^* *is locally asymptotically stable if* $\mathfrak{R}_2 > 1$ *and* $\mathcal{R}_1^{inv} < 1$.

Proof. We will present the proof of the theorem sequentially.

(1) To analyze the local stability of system (1.1) at the disease-free equilibrium $E_0^* = \Big(\frac{\Lambda}{\gamma}, 0, 0, 0, 0\Big)$, we compute the corresponding Jacobian matrix

$$\mathcal{J}(E_0^*) = \begin{bmatrix}
-\gamma & -\beta_1\frac{\Lambda}{\gamma} & -\beta_2\frac{\Lambda}{\gamma} & 0 & \mu\\
0 & \beta_1\frac{\Lambda}{\gamma} - (\gamma_1+\delta_1) & 0 & 0 & 0\\
0 & 0 & \beta_2\frac{\Lambda}{\gamma} - (\gamma_2+\delta_2) & 0 & 0\\
0 & \delta_1 & 0 & -\gamma_3 & 0\\
0 & 0 & \delta_2 & 0 & -(\gamma_4+\mu)
\end{bmatrix}.$$

The eigenvalues of $\mathcal{J}(E_0^*)$ are:

$$\mathcal{E}_1 = -\gamma, \quad \mathcal{E}_2 = \beta_1\frac{\Lambda}{\gamma} - (\gamma_1+\delta_1), \quad \mathcal{E}_3 = -\gamma_3, \quad \mathcal{E}_4 = \beta_2\frac{\Lambda}{\gamma} - (\gamma_2+\delta_2), \quad \mathcal{E}_5 = -(\gamma_4+\mu). \tag{3.2}$$

.

From (3.2), we observe that $\mathcal{E}_1 < 0$, $\mathcal{E}_3 < 0$ and $\mathcal{E}_5 < 0$. Furthermore, since $\mathfrak{R}_1 < 1$ and $\mathfrak{R}_2 < 1$, it follows that $\mathcal{E}_2 < 0$ and $\mathcal{E}_4 < 0$. Therefore, since all the eigenvalues possess negative real parts, the equilibrium point $E_0^* = \left(\frac{\Lambda}{\gamma}, 0, 0, 0, 0\right)$ is asymptotically stable.

(2) The Jacobian matrix corresponding to the equilibrium point $E_1^* = \left(\frac{\gamma_1+\delta_1}{\beta_1}, \frac{\gamma(\mathfrak{R}_1-1)}{\beta}, 0, \frac{\delta_1\gamma(\mathfrak{R}_1-1)}{\beta_1\gamma_3}, 0\right)$ of the system (1.1) is expressed as

$$\mathcal{J}(E_1^*) = \begin{bmatrix} -\gamma(\mathfrak{R}_1-1)-\gamma & -(\gamma_1+\delta_1) & 0 & 0 & \mu \\ \gamma(\mathfrak{R}_1-1) & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_2(\gamma_1+\delta_1)}{\beta_1} - (\gamma_2+\delta_2) & 0 & 0 \\ 0 & \delta_1 & 0 & -\gamma_3 & 0 \\ 0 & 0 & \delta_2 & 0 & -(\gamma_4+\mu) \end{bmatrix}.$$

The eigenvalues of $J(E_1^*)$ are:

$$\mathcal{E}_1 = -(\gamma_4+\mu) < 0,$$
$$\mathcal{E}_2 = \frac{\beta_2(\gamma_1+\delta_1)}{\beta_1} - (\gamma_2+\delta_2) = (\gamma_2+\delta_2)(\mathcal{R}_2^{inv} - 1) < 0,$$
$$\mathcal{E}_3 = -\gamma_3 < 0,$$

and the remaining two eigenvalues are the roots of the quadratic equation

$$\mathcal{E}^2 + (\gamma(\mathfrak{R}_1-1)+\gamma)\,\mathcal{E} + \gamma(\gamma_1+\delta_1)(\mathfrak{R}_1-1) = 0.$$

By the Routh-Hurvitz criteria [15] for quadratic equations, both roots have negative real parts if and only if the coefficients of the above quadratic equation satisfy $\gamma(\mathfrak{R}_1-1) > 0$ and $\gamma(\gamma_1+\delta_1)(\mathfrak{R}_1-1) > 0$. Since $\gamma, \gamma_1, \delta_1 > 0$, these conditions hold if and only if $\mathfrak{R}_1 > 1$.

Thus, all eigenvalues have negative real parts if $\mathfrak{R}_1 > 1$. Therefore, the equilibrium E_1^* is asymptotically stable.

(3) This case is similar to the previous one, so the analysis is omitted. This completes the proof. □

3.3. **Global stability of disease-free equilibrium E_0^*.** After establishing the local stability of the disease-free equilibrium E_0^*, we now aim to establish its global stability. We begin with the following theorem.

Theorem 3.4. *Under the conditions $\mathfrak{R}_1 < 1$ and $\mathfrak{R}_2 < 1$, the disease-free equilibrium E_0^* of system (1.1) is globally asymptotically stable.*

Proof. We use Lyapunov's direct method to prove the result. Consider the following positive definite Lyapunov function,

$$V^* = \left(\mathcal{S} - \frac{\Lambda}{\gamma} + \mathcal{I}_1 + \mathcal{I}_2\right)^2 + a_1{\mathcal{I}_1}^2 + a_2{\mathcal{I}_2}^2 + {\mathcal{R}_1}^2 + {\mathcal{R}_2}^2,$$

where a_i's $(i = 1, 2)$ are positive constants to be chosen later.

Differentiating V^* along the solutions of system (1.1), we obtain

$$\begin{aligned}
\frac{dV^*}{dt} &= 2\Big(\mathcal{S} - \frac{\Lambda}{\gamma} + \mathcal{I}_1 + \mathcal{I}_2\Big)\Big(\Lambda - \gamma\mathcal{S} + \mu\mathcal{R}_2 - (\gamma_1+\delta_1)\mathcal{I}_1 - (\gamma_2+\delta_2)\mathcal{I}_2\Big) \\
&\quad + 2a_1\mathcal{I}_1\Big(\beta_1\mathcal{S}\mathcal{I}_1 - (\gamma_1+\delta_1)\mathcal{I}_1\Big) + 2a_2\mathcal{I}_2\Big(\beta_1\mathcal{S}\mathcal{I}_2 - (\gamma_2+\delta_2)\mathcal{I}_2\Big) \\
&\quad + 2\mathcal{R}_1(\delta_1\mathcal{I}_1 - \gamma_3\mathcal{R}_1) + 2\mathcal{R}_2(\delta_2\mathcal{I}_2 - \gamma_4\mathcal{R}_2 - \mu\mathcal{R}_2) \\
&= -2\gamma\Big(S - \frac{\Lambda}{\gamma}\Big)^2 - 2\big((\gamma_1+\delta_1)(a_1+1)\big){\mathcal{I}_1}^2 - 2\big((\gamma_2+\delta_2)(a_2+1)\big){\mathcal{I}_2}^2 \\
&\quad - 2\gamma_3{\mathcal{R}_1}^2 - 2(\gamma_4+\mu){\mathcal{R}_2}^2 + 2\mu\Big(\mathcal{S} - \frac{\Lambda}{\gamma}\Big)\mathcal{R}_2 - 2(\gamma_1+\delta_1)\Big(\mathcal{S} - \frac{\Lambda}{\gamma}\Big)\mathcal{I}_1 \\
&\quad - 2(\gamma_2+\delta_2)\Big(\mathcal{S} - \frac{\Lambda}{\gamma}\Big)\mathcal{I}_2 - 2\gamma\Big(\mathcal{S} - \frac{\Lambda}{\gamma}\Big)\mathcal{I}_1 + 2\mu\mathcal{I}_1\mathcal{R}_2 - 2(\gamma_2+\delta_2)\mathcal{I}_1\mathcal{I}_2
\end{aligned}$$

$$
\begin{aligned}
&+ 2\gamma\Big(\frac{\Lambda}{\gamma} - \mathcal{S}\Big) + 2\mu\mathcal{I}_2\mathcal{R}_2 - 2(\gamma_1+\delta_1)\mathcal{I}_1\mathcal{I}_2 + 2a_1\beta_1\mathcal{S}{\mathcal{I}_1}^2 + 2a_2\beta_2\mathcal{S}{\mathcal{I}_2}^2 \\
&+ 2\delta_1\mathcal{I}_1\mathcal{R}_1 + 2\delta_2\mathcal{I}_2\mathcal{R}_2 \\
&\le -2\gamma\Big(S - \frac{\Lambda}{\gamma}\Big)^2 - 2(\gamma_1+\delta_1){\mathcal{I}_1}^2 - 2(\gamma_2+\delta_2){\mathcal{I}_2}^2 - 2\gamma_3{\mathcal{R}_1}^2 - 2(\gamma_4+\mu){\mathcal{R}_2}^2 \\
&+ 2\mu\Big(\mathcal{S} - \frac{\Lambda}{\gamma}\Big)\mathcal{R}_2 + 2(\gamma+\gamma_1+\delta_1)\Big(\frac{\Lambda}{\gamma} - \mathcal{S}\Big)\mathcal{I}_1 + 2(\gamma+\gamma_2+\delta_2)\Big(\frac{\Lambda}{\gamma} - \mathcal{S}\Big)\mathcal{I}_2 \\
&+ 2\mu\mathcal{I}_1\mathcal{R}_2 + 2a_1\Big(\frac{\beta_1\Lambda}{\gamma} - (\gamma_1+\delta_1)\Big){\mathcal{I}_1}^2 + 2a_2\Big(\frac{\beta_2\Lambda}{\gamma} - (\gamma_2+\delta_2)\Big){\mathcal{I}_2}^2 \\
&+ 2\delta_1\mathcal{I}_1\mathcal{R}_1 + 2(\mu+\delta_2)\mathcal{I}_2\mathcal{R}_2.
\end{aligned}
$$

Using Young's inequality for the following expressions

$$
\begin{aligned}
2\Big(\frac{\Lambda}{\gamma} - \mathcal{S}\Big)\mathcal{I}_1 &\le c_1\Big(\frac{\Lambda}{\gamma} - \mathcal{S}\Big)^2 + \frac{1}{c_1}\mathcal{I}_1^2, \\
2\Big(\frac{\Lambda}{\gamma} - \mathcal{S}\Big)\mathcal{I}_2 &\le c_1\Big(\frac{\Lambda}{\gamma} - \mathcal{S}\Big)^2 + \frac{1}{c_1}\mathcal{I}_2^2, \\
2\Big(\mathcal{S} - \frac{\Lambda}{\gamma}\Big)\mathcal{R}_2 &\le c_1\Big(\mathcal{S} - \frac{\Lambda}{\gamma}\Big)^2 + \frac{1}{c_1}\mathcal{R}_2^2, \\
2\mathcal{I}_1\mathcal{R}_1 &\le \frac{1}{c_2}{\mathcal{I}_1}^2 + c_2{\mathcal{R}_1}^2, \\
2\mathcal{I}_1\mathcal{R}_2 &\le \frac{1}{c_3}{\mathcal{I}_1}^2 + c_3{\mathcal{R}_2}^2, \\
2\mathcal{I}_2\mathcal{R}_2 &\le \frac{1}{c_3}{\mathcal{I}_2}^2 + c_3{\mathcal{R}_2}^2,
\end{aligned}
$$

where

$$
c_1 = \frac{\gamma}{\gamma_1+\gamma_2+\gamma_3+\gamma_4+\delta_1+\delta_2}, \quad c_2 = \frac{\gamma_1+\delta_1}{\gamma_2+\gamma_3+\gamma_4},
$$

and c_3 is a positive constant.

For $\mathfrak{R}_1 \le 1$ and $\mathfrak{R}_2 \le 1$, it is possible to choose constants a_1 and a_2 such that

$$
\begin{aligned}
a_1\Big(\frac{\beta_1\Lambda}{\gamma} - (\gamma_1+\delta_1)\Big) + \frac{1}{c_1}(\gamma+\gamma_1+\delta_1) + \frac{1}{c_2}\delta_1 + \frac{1}{c_3}\mu = 0, \\
a_2\Big(\frac{\beta_2\Lambda}{\gamma} - (\gamma_2+\delta_2)\Big) + \frac{1}{c_1}(\gamma_1+\gamma_2+\delta_2) + \frac{1}{c_3}(\mu+\delta_2) = 0.
\end{aligned}
$$

Thus, the derivative of the Lyapunov function V^* satisfies the inequality

$$
\frac{dV^*}{dt} \le -\gamma\Big(S - \frac{\Lambda}{\gamma}\Big)^2 - (\gamma_1+\delta_1){\mathcal{I}_1}^2 - (\gamma_2+\delta_2){\mathcal{I}_2}^2 - \gamma_3{\mathcal{R}_1}^2 - (\gamma_4+\mu){\mathcal{R}_2}^2.
$$

Therefore, V^* is negative definite, implying that the disease-free equilibrium E_0^* of system (1.1) is globally asymptotically stable, which concludes the proof of the theorem. □

4. Existence of positive T-periodic solutions

Periodicity is a very important property for epidemic models to plan and respond to public health outbreak. It may help us to predict the severity and timing of the outbreak. In this section, we establish the existence of periodic solution for our model (1.3) under certain conditions.

Theorem 4.1. *Assume that with respect to the strong kernel, the stochastic system* (1.3) *possesses a solution* $(\mathcal{S}(t), \mathcal{I}_1(t), \mathcal{I}_2(t), \mathcal{R}_1(t), \mathcal{R}_2(t))$. *Then, the system* (1.3) *possesses a positive* T*-periodic solution if*

$$
G = \frac{\langle 2\Lambda(t)\beta_1\rangle_T^{1/2}}{\langle \gamma_1+\gamma_2+\delta_1+\delta_2+\gamma+(\sigma_1^2+\sigma_2^2)\rangle_T} > 1.
$$

Proof. Let us define a $\mathcal{C}^2$-function

$$V_1 = -ln\mathcal{S} - ln\mathcal{I}_1 - ln\mathcal{I}_2 + \kappa(t).$$

Using Ito's formula, we have

$$\begin{aligned}
LV_1 &= \frac{-1}{\mathcal{S}}\big(\Lambda - \beta_1\mathcal{S}(t)\mathcal{I}_1(t) - \beta_2\mathcal{S}(t)\mathcal{I}_2(t) - \gamma\mathcal{S}(t) + \mu\mathcal{R}_2(t)\big) + \frac{1}{2}\sigma_1^2\mathcal{I}_1^2(t) + \frac{1}{2}\sigma_2^2\mathcal{I}_2^2(t)\\
&\quad - \frac{1}{\mathcal{I}_1}\big(\beta_1\mathcal{S}(t)\mathcal{I}_1(t) - \gamma_1\mathcal{I}_1(t) - \delta_1\mathcal{I}_1(t)\big) + \frac{1}{2}\sigma_1^2\mathcal{I}_1^2(t) - \frac{1}{\mathcal{I}_2}\big(\beta_2\mathcal{S}(t)\mathcal{I}_2(t) - \gamma_2\mathcal{I}_2(t) - \delta_2\mathcal{I}_2(t)\big)\\
&\quad + \frac{1}{2}\sigma_2^2\mathcal{I}_2^2(t) + \dot{\kappa}(t)\\
&= \sigma_1^2\mathcal{I}_1^2(t) + \sigma_2^2\mathcal{I}_2^2(t) + \gamma_1 + \gamma_2 + \delta_1 + \delta_2 + \gamma - \frac{\Lambda}{\mathcal{S}} + \beta_1\mathcal{I}_1 + \beta_2\mathcal{I}_2 - \mu\frac{\mathcal{R}_2}{\mathcal{S}} - \beta_1\mathcal{S} - \beta_2\mathcal{S} + \dot{\kappa}(t)\\
&\le (\sigma_1^2 \vee \sigma_2^2) + \gamma_1 + \gamma_2 + \delta_1 + \delta_2 + \gamma - (2\Lambda\beta_1)^{1/2} - (2\mu\beta_2\mathcal{R}_2)^{1/2} + \beta_1\mathcal{I}_1 + \beta_2\mathcal{I}_2 + \dot{\kappa}(t).
\end{aligned}$$

Let us assume that

$$\begin{aligned}
\dot{\kappa}(t) &= \langle(\sigma_1^2 \vee \sigma_2^2) + \gamma_1 + \gamma_2 + \delta_1 + \delta_2 + \gamma\rangle_T - 2\beta_1^{1/2}\langle\Lambda^{1/2}\rangle_T\\
&\quad - (\sigma_1^2 \vee \sigma_2^2) + \gamma_1 + \gamma_2 + \delta_1 + \delta_2 + \gamma + (2\Lambda\beta_1)^{1/2}.
\end{aligned}$$

Using the above assumption, we obtain

$$\begin{aligned}
LV_1 &\le \langle(\sigma_1^2 \vee \sigma_2^2) + \gamma_1 + \gamma_2 + \delta_1 + \delta_2 + \gamma\rangle_T - 2\beta_1^{1/2}\langle\Lambda^{1/2}\rangle_T - (2\mu\beta_2\mathcal{R}_2)^{1/2} + \beta_1\mathcal{I}_1 + \beta_2\mathcal{I}_2\\
&\le -\langle(\sigma_1^2 \vee \sigma_2^2) + \gamma_1 + \gamma_2 + \delta_1 + \delta_2 + \gamma\rangle_T(H-1) + \beta_1\mathcal{I}_1 + \beta_2\mathcal{I}_2 - (2\mu\beta_2\mathcal{R}_2)^{1/2} \qquad (4.1)\\
&\le -A(H-1) + \beta_1\mathcal{I}_1 + \beta_2\mathcal{I}_2 - (2\mu\beta_2\mathcal{R}_2)^{1/2},
\end{aligned}$$

where $A = \inf\{(\sigma_1^2 \vee \sigma_2^2) + \gamma_1 + \gamma_2 + \delta_1 + \delta_2 + \gamma\}$. Let us now define

$$V_2 = -\ln\mathcal{S}, \quad V_3 = -\ln\mathcal{R}_1 - \ln\mathcal{R}_2, \quad V_4 = -\ln\mathcal{I}_1 - \ln\mathcal{I}_2,$$

$$V_5 = \frac{(\mathcal{S} + \mathcal{I}_1 + \mathcal{I}_2 + \mathcal{R}_1 + \mathcal{R}_2)^{\theta+1}}{\theta+1}, \quad 0 < \theta < 1.$$

Supposing $\mathcal{N} = \mathcal{S} + \mathcal{I}_1 + \mathcal{I}_2 + \mathcal{R}_1 + \mathcal{R}_2$, thus

$$V_5 = \frac{\mathcal{N}^{\theta+1}}{\theta+1}, \quad 0 < \theta < 1.$$

Using the similar computation as above, we obtain

$$LV_2 \le \gamma + \frac{1}{2}(\sigma_1^2 \vee \sigma_2^2) - \frac{\Lambda}{\mathcal{S}} - \mu\frac{\mathcal{R}_2}{\mathcal{S}}.$$

By denoting $B = \sup\{\gamma + \frac{1}{2}(\sigma_1^2 \vee \sigma_2^2)\}$, we obtain

$$LV_2 \le B - \frac{\Lambda}{\mathcal{S}} - \mu\frac{\mathcal{R}_2}{\mathcal{S}}. \qquad (4.2)$$

Similarly, we have

$$LV_3 \le C - \delta_1\frac{\mathcal{I}_1}{\mathcal{R}_1} - \delta_2\frac{\mathcal{I}_2}{\mathcal{R}_2}, \qquad (4.3)$$

where $C = \sup\{\gamma_3 + \gamma_4 + \mu\}$. Moreover, we have

$$LV_4 \le D - (\eta_1 - \eta_2)\mathcal{S}, \qquad (4.4)$$

where $D = \sup\{\gamma_1 + \gamma_2 + \delta_1 + \delta_2 + \frac{1}{2}(\sigma_1^2 \vee \sigma_2^2)\}$. Applying Ito's formula on V_5, we obtain

$$LV_5 = \mathcal{N}^\theta(\Lambda - \gamma\mathcal{S} - \gamma_1\mathcal{I}_1 - \gamma_2\mathcal{I}_2 - \gamma_3\mathcal{R}_1 - \gamma_4\mathcal{R}_2) + \frac{1}{2}\theta\mathcal{N}^{\theta-1}(2\sigma_1^2\mathcal{S}^2\mathcal{I}_1^2 + 2\sigma_2^2\mathcal{S}^2\mathcal{I}_2^2).$$

Suppose $\alpha = min\{\gamma, \gamma_1, \gamma_2, \gamma_3, \gamma_4\}$. Then

$$\begin{aligned}
LV_5 &\le \mathcal{N}^\theta\big(\Lambda - \alpha\mathcal{N}\big) + \theta\mathcal{N}^{\theta-1}(\sigma_1^2 \vee \sigma_2^2)\\
&\le -\Big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\Big)\Big(\mathcal{S}^{\theta+1} + \mathcal{I}_1^{\theta+1} + \mathcal{I}_2^{\theta+1} + \mathcal{R}_1^{\theta+1} + \mathcal{R}_2^{\theta+1}\Big) + \Lambda\mathcal{N}^\theta,
\end{aligned}$$

where $\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2) > 0$ i.e. $0 < \theta < \frac{2\alpha}{\sigma_1^2 \vee \sigma_2^2}$ and

$$E = \sup\Big\{ -\frac{1}{2}\Big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\Big)\Big(\mathcal{S}^{\theta+1} + \mathcal{I}_1^{\theta+1} + \mathcal{I}_2^{\theta+1} + \mathcal{R}_1^{\theta+1} + \mathcal{R}_2^{\theta+1}\Big)\Big\} + \beta(\mathcal{S} + \mathcal{I}_1 + \mathcal{I}_2 + \mathcal{R}_1 + \mathcal{R}_2)^\theta.$$

After some computations, we obtain

$$LV_5 \le -\frac{1}{2}\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big)\Big(\mathcal{S}^{\theta+1} + \mathcal{I}_1^{\theta+1} + \mathcal{I}_2^{\theta+1} + \mathcal{R}_1^{\theta+1} + \mathcal{R}_2^{\theta+1}\Big) + E. \tag{4.5}$$

We now consider a $\mathcal{C}^2$-function, which is defined as $V : [0,\infty) \times \mathbb{R}_+^5 \to \mathbb{R}$ by

$$V(t, \mathcal{S}, \mathcal{I}_1, \mathcal{I}_1, , \mathcal{R}_1, \mathcal{R}_2) = MV_1 + V_2 + V_3 + V_4 + V_5,$$

where the positive constant M satisfies $-MA(H-1) + B + C + D + E \le -2$.

Note that $V(t, \mathcal{S}, \mathcal{I}_1, \mathcal{I}_1, , \mathcal{R}_1, \mathcal{R}_2) \to \infty$ as $|(\mathcal{S}, \mathcal{I}_1, \mathcal{I}_1, , \mathcal{R}_1, \mathcal{R}_2)| \to \infty$, ensuring that condition (i) of Lemma 2.3 is satisfied. Now, we are required to prove condition (i) of Lemma 2.3.

Equations (4.1), (4.2), (4.3), (4.4) and (4.5) when combined yield

$$\begin{aligned}
LV &\le -MA(H-1) + M\beta_1\mathcal{I}_1 + M\beta_2\mathcal{I}_2 - M(2\mu\beta_2\mathcal{R}_2)^{1/2} + B - \frac{\Lambda}{S} - \mu\frac{\mathcal{R}_2}{\mathcal{S}} + C - \delta_1\frac{\mathcal{I}_1}{\mathcal{R}_1} - \delta_2\frac{\mathcal{I}_2}{\mathcal{R}_2} \\
&\quad + D - (\eta_1 - \eta_2)\mathcal{S} - \frac{1}{2}\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big)\Big(\mathcal{S}^{\theta+1} + \mathcal{I}_1^{\theta+1} + \mathcal{I}_2^{\theta+1} + \mathcal{R}_1^{\theta+1} + \mathcal{R}_2^{\theta+1}\Big) + E \\
&\le -2 + M\beta_1\mathcal{I}_1 + M\beta_2\mathcal{I}_2 - M(2\mu\beta_2\mathcal{R}_2)^{1/2} - \frac{\Lambda}{\mathcal{S}} - \mu\frac{\mathcal{R}_2}{\mathcal{S}} - \delta_1\frac{\mathcal{I}_1}{\mathcal{R}_1} - \delta_2\frac{\mathcal{I}_2}{\mathcal{R}_2} - (\eta_1 - \eta_2)\mathcal{S} \\
&\quad - \frac{1}{2}\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big)\Big(\mathcal{S}^{\theta+1} + \mathcal{I}_1^{\theta+1} + \mathcal{I}_2^{\theta+1} + \mathcal{R}_1^{\theta+1} + \mathcal{R}_2^{\theta+1}\Big).
\end{aligned} \tag{4.6}$$

WE define a closed and bounded set

$$\begin{aligned}
\Theta = \Big\{(\mathcal{S}, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}_1, \mathcal{R}_2) \in \mathbb{R}_+^5 : \epsilon_1 < \mathcal{S} < \frac{1}{\epsilon_1}, \epsilon_2 < \mathcal{I}_1 < \frac{1}{\epsilon_2}, \epsilon_3 < \mathcal{I}_2 < \frac{1}{\epsilon_3}, \\
\epsilon_4 < \mathcal{R}_1 < \frac{1}{\epsilon_4}, \epsilon_5 < \mathcal{R}_2 < \frac{1}{\epsilon_5}\Big\},
\end{aligned}$$

where $0 < \epsilon_1, \epsilon_2, \epsilon_3, \epsilon_4, \epsilon_5 < 1$ are sufficiently small real numbers. Clearly, Θ is a closed and bounded subset of $\mathbb{R}_+^5$, hence compact. Furthermore, $\epsilon_1, \epsilon_2, \epsilon_3, \epsilon_4$ and ϵ_5 satisfy the following condition on the set $\mathbb{R}_+^5 \backslash \Theta$:

$$\begin{aligned}
0 < \epsilon_1 &\le \min\Big\{\Big[\frac{\frac{1}{2}\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big) - (\beta_1 + \beta_2)}{O+1}\Big]^{\frac{1}{\theta+1}}, \frac{\Lambda}{O+1}\Big\}, \\
0 < \epsilon_2 &\le \min\Big\{\Big[\frac{\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2}{4(O+1)}\Big]^{1/\theta+1}, \frac{1}{M\beta_1}\Big\}, \\
0 < \epsilon_3 &\le \min\Big\{\Big[\frac{\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)}{4(O+1)}\Big]^{1/\theta+1}, \frac{1}{M\beta_2}\Big\}, \\
0 < \epsilon_4 &\le \min\Big\{\Big[\frac{\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)}{2(O+1)}\Big]^{1/\theta+1}, \frac{\delta_1\epsilon_2}{O+1}\Big\}, \\
0 < \epsilon_5 &\le \min\Big\{\Big[\frac{1}{2}\frac{\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big) - \frac{\mu}{\epsilon_1}}{O+1}\Big]^{1/\theta+1}, \frac{\delta_2\epsilon_3}{O+1}\Big\},
\end{aligned}$$

where

$$O = -2 + M\beta_1\mathcal{I}_1 + M\beta_2\mathcal{I}_2 - M(2\mu\beta_2\mathcal{R}_2)^{1/2} - \frac{1}{4}\Big(\alpha - \frac{\theta}{2}\big(\sigma_1^2 \vee \sigma_2^2\big)\Big)\Big(\mathcal{I}_1^{\theta+1} + \mathcal{I}_2^{\theta+1}\Big).$$

To facilitate the further discussion, we divide $\Theta^c = R_+^5 \backslash \Theta$ into ten domains $\Theta^i; i = 1, 2, 3 \dots 10$ such that $(\mathcal{S}, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}_1, \mathcal{R}_2) \in \Theta^i$ for every $i = 1, 2, 3 \dots 10$ and

$$\begin{aligned}
\Theta^1 = \Big\{\mathcal{S} > \frac{1}{\epsilon_1}\Big\}, \quad \Theta^2 = \Big\{\mathcal{I}_1 > \frac{1}{\epsilon_2}\Big\}, \\
\Theta^3 = \Big\{\mathcal{I}_2 > \frac{1}{\epsilon_3}\Big\}, \quad \Theta^4 = \Big\{\mathcal{R}_1 > \frac{1}{\epsilon_4}\Big\},
\end{aligned}$$

$$\Theta^5 = \Big\{0 < \mathcal{S} < \epsilon_1\Big\}, \quad \Theta^6 = \Big\{0 < \mathcal{S} < \epsilon_1, \mathcal{R}_2 > \frac{1}{\epsilon_5}\Big\},$$
$$\Theta^7 = \Big\{0 < \mathcal{I}_1 < \epsilon_2\Big\}, \quad \Theta^8 = \Big\{0 < \mathcal{I}_2 < \epsilon_3\Big\},$$
$$\Theta^9 = \Big\{\epsilon_1 \le \mathcal{S} \le \frac{1}{\epsilon_1}, \epsilon_2 \le \mathcal{I}_1 \le \frac{1}{\epsilon_2}, \epsilon_3 \le \mathcal{I}_2 \le \frac{1}{\epsilon_3}, 0 < \mathcal{R}_1 < \epsilon_4\Big\},$$
$$\Theta^{10} = \Big\{\epsilon_1 \le \mathcal{S} \le \frac{1}{\epsilon_1}, \epsilon_2 \le \mathcal{I}_1 \le \frac{1}{\epsilon_2}, \epsilon_3 \le \mathcal{I}_2 \le \frac{1}{\epsilon_3}, \epsilon_4 \le \mathcal{R}_1 \le \frac{1}{\epsilon_4}, 0 < \mathcal{R}_2 < \epsilon_5\Big\}.$$

It can be observed here that $\Theta^c = \bigcup_{i=1}^{10} \Theta^i$. Further, we prove that $LV \le -1$ for any $(t, \mathcal{S}, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}_1, \mathcal{R}_2) \in \mathbb{R}_+ \times \Theta^c$, i.e. we have to prove that $LV \le -1$ on all the ten domains mentioned above. We discuss it in the following cases:

Case 1. For any $(t, \mathcal{S}, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}_1, \mathcal{R}_2) \in \mathbb{R}_+ \times \Theta^1$,

$$\begin{aligned} LV &\le O - \frac{1}{2}\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big)\mathcal{S}^{\theta+1} - (\beta_1 + \beta_2)\mathcal{S} \\ &\le O - \frac{1}{2}\frac{\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big)}{\epsilon_1^{\theta+1}} - \frac{(\beta_1 + \beta_2)}{\epsilon_1}. \end{aligned}$$

Since $0 < \epsilon_1 < 1$, we have $-\frac{1}{\epsilon_1} \le -\frac{1}{\epsilon_1^\theta}$; $\theta > 0$. Thus, we obtain

$$LV \le O - \frac{1}{2}\frac{\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big)}{\epsilon_1^{\theta+1}} - \frac{(\beta_1 + \beta_2)}{\epsilon_1^{\theta+1}} \le -1.$$

Case 2. For any $(t, \mathcal{S}, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}_1, \mathcal{R}_2) \in \mathbb{R}_+ \times \Theta^2$, we have

$$LV \le O - \frac{1}{4}\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big)\mathcal{I}_1^{\theta+1} \le O - \frac{1}{4}\frac{\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big)}{\epsilon_2^{\theta+1}} \le -1.$$

Case 3. For any $(t, \mathcal{S}, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}_1, \mathcal{R}_2) \in \mathbb{R}_+ \times \Theta^3$, we obtain

$$LV \le O - \frac{1}{4}\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big)\mathcal{I}_2^{\theta+1} \le O - \frac{1}{4}\frac{\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big)}{\epsilon_3^{\theta+1}} \le -1.$$

Case 4. For any $(t, \mathcal{S}, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}_1, \mathcal{R}_2) \in \mathbb{R}_+ \times \Theta^4$, we have

$$LV \le O - \frac{1}{4}\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big)\mathcal{R}_1^{\theta+1} \le O - \frac{1}{4}\frac{\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big)}{\epsilon_4^{\theta+1}} \le -1.$$

Case 5. For any $(t, \mathcal{S}, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}_1, \mathcal{R}_2) \in \mathbb{R}_+ \times \Theta^6$, we obtain

$$LV \le O - \frac{\Lambda}{\mathcal{S}} < O - \frac{\Lambda}{\epsilon_1} < -1.$$

Case 6. For any $(t, \mathcal{S}, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}_1, \mathcal{R}_2) \in \mathbb{R}_+ \times \Theta^5$, we obtain

$$\begin{aligned} LV &\le O - \frac{1}{4}\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big)\mathcal{R}_2^{\theta+1} - \mu\frac{\mathcal{R}_2}{\mathcal{S}} \\ &\le O - \frac{1}{4}\frac{\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big)}{\epsilon_5^{\theta+1}} - \frac{\mu}{\epsilon_1\epsilon_5}. \end{aligned}$$

Since $0 < \epsilon_5 < 1$, we have $-\frac{1}{\epsilon_5} \le -\frac{1}{\epsilon_5^\theta}$; $\theta > 0$. Thus, we obtain

$$LV \le O - \frac{1}{4}\frac{\big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\big)}{\epsilon_5^{\theta+1}} - \frac{\mu}{\epsilon_1\epsilon_5^{\theta+1}} \le -1.$$

Case 7. For any $(t,\mathcal{S},\mathcal{I}_1,\mathcal{I}_2,\mathcal{R}_1,\mathcal{R}_2)\in\mathbb{R}_+\times\Theta^7$, we have

$$LV\le -2+M\beta_1\mathcal{I}_1\le -2+M\beta_1\epsilon_2<-1.$$

Case 8. For any $(t,\mathcal{S},\mathcal{I}_1,\mathcal{I}_2,\mathcal{R}_1,\mathcal{R}_2)\in\mathbb{R}_+\times\Theta^8$, we obtain

$$LV\le -2+M\beta_2\mathcal{I}_2\le -2+M\beta_2\epsilon_3<-1.$$

Case 9. For any $(t,\mathcal{S},\mathcal{I}_1,\mathcal{I}_2,\mathcal{R}_1,\mathcal{R}_2)\in\mathbb{R}_+\times\Theta^9$, we obtain

$$LV\le O-\delta_1\frac{\mathcal{I}_1}{\mathcal{R}_1}\le O-\delta_1\frac{\epsilon_2}{\epsilon_4}<-1.$$

Case 10. For any $(t,\mathcal{S},\mathcal{I}_1,\mathcal{I}_2,\mathcal{R}_1,\mathcal{R}_2)\in\mathbb{R}_+\times\Theta^{10}$, we have

$$LV\le O-\delta_2\frac{\mathcal{I}_2}{\mathcal{R}_2}\le O-\delta_2\frac{\epsilon_3}{\epsilon_5}<-1.$$

Thus, from the cases mentioned above, we can conclude that

$$LV\le -1\quad\forall\quad(t,\mathcal{S},\mathcal{I}_1,\mathcal{I}_2,\mathcal{R}_1,\mathcal{R}_2)\in\mathbb{R}_+\times\Theta^c,$$

where $\Theta\in\mathbb{R}^5_+$ is a closed set, i.e., $LV\le -1$ outside a compact set $\mathbb{R}_+\times\Theta$. Therefore, the condition (2.6) of Lemma 2.3 holds. Hence, system (1.3) possesses a positive T-periodic solution according to Lemma 2.3. □

5. Positive recurrence of the system

Positive recurrence is an essential property to study the long term behavior and persistence of the epidemics. A theorem which is required to show that the system (1.3) is positive recurrent is stated below:

Theorem 5.1. *Consider $(\mathcal{S},\mathcal{I}_1,\mathcal{I}_2,\mathcal{R}_1,\mathcal{R}_2)$ as a solution of system (1.3). Suppose that*

$$H=\frac{2\sqrt{\beta_1\Lambda}}{(\sigma_1^2\vee\sigma_2^2)+\gamma_1+\gamma_2+\delta_1+\delta_2+\gamma}>1.$$

Then, the solution $(\mathcal{S},\mathcal{I}_1,\mathcal{I}_2,\mathcal{R}_1,\mathcal{R}_2)$ is positive recurrent with respect to the domain

$$D_\nu=\Big\{(\mathcal{S},\mathcal{I}_1,\mathcal{I}_2,\mathcal{R}_1,\mathcal{R}_2)\in\mathbb{R}^5_+:\nu_1\le S\le\frac{1}{\nu_1},\nu_2\le\mathcal{I}_1\le\frac{1}{\nu_2},\nu_3\le\mathcal{I}_2\le\frac{1}{\nu_3},$$
$$\nu_4\le\mathcal{R}_1\le\frac{1}{\nu_4},\nu_5\le\mathcal{R}_2\le\frac{1}{\nu_5}\Big\},$$

where $\nu_i,\{i=1,2,3,4,5\}$ are sufficiently small real numbers.

Proof. Let $(X_1,X_2,X_3,X_4,X_5)=(\mathcal{S},\mathcal{I}_1,\mathcal{I}_2,\mathcal{R}_1,\mathcal{R}_2)$. Define

$$\overline{V_1}=-\ln\mathcal{S}-\ln\mathcal{I}_1-\ln\mathcal{I}_2.$$

By applying Itô's formula to $\overline{V_1}$, we obtain

$$L\overline{V_1}=-\frac{1}{\mathcal{S}}\big(\Lambda-\beta_1\mathcal{S}\mathcal{I}_1-\beta_2\mathcal{S}\mathcal{I}_2-\gamma\mathcal{S}+\mu\mathcal{R}_2\big)-\frac{1}{\mathcal{I}_1}\big(\beta_1\mathcal{S}\mathcal{I}_1-\gamma_1\mathcal{I}_1-\delta_1\mathcal{I}_1\big)$$
$$-\frac{1}{\mathcal{I}_2}\big(\beta_2\mathcal{S}\mathcal{I}_2-\gamma_2\mathcal{I}_2-\delta_2\mathcal{I}_2\big)+\frac{1}{2}\big(\sigma_1^2+\sigma_2^2\big).$$

Simplifying gives

$$L\overline{V_1}\le(\sigma_1^2\vee\sigma_2^2)+\gamma_1+\gamma_2+\delta_1+\delta_2+\gamma-(2\Lambda\beta_1)^{1/2}-(2\mu\beta_2\mathcal{R}_2)^{1/2}+\beta_1\mathcal{I}_1+\beta_2\mathcal{I}_2.$$

Hence,

$$L\overline{V_1}\le -A'(H-1)+\beta_1\mathcal{I}_1+\beta_2\mathcal{I}_2-(2\mu\beta_2\mathcal{R}_2)^{1/2},\tag{5.1}$$

where

$$A'=(\sigma_1^2\vee\sigma_2^2)+\gamma_1+\gamma_2+\delta_1+\delta_2+\gamma.$$

Next, we define

$$\overline{V_2} = -\ln \mathcal{S}, \quad \overline{V_3} = -\ln \mathcal{R}_1 - \ln \mathcal{R}_2, \quad \overline{V_4} = -\ln \mathcal{I}_1 - \ln \mathcal{I}_2,$$
$$\overline{V_5} = \frac{1}{\theta+1}(\mathcal{S} + \mathcal{I}_1 + \mathcal{I}_2 + \mathcal{R}_1 + \mathcal{R}_2)^{\theta+1},$$

where $0 < \theta < 1$. For $\overline{V_2}$,

$$L\overline{V_2} \le B' - \frac{\Lambda}{\mathcal{S}} - \mu\frac{\mathcal{R}_2}{\mathcal{S}}, \tag{5.2}$$

where $B' = \gamma + \frac{1}{2}(\sigma_1^2 \vee \sigma_2^2)$. Similarly,

$$L\overline{V_3} \le C' - \delta_1\frac{\mathcal{I}_1}{\mathcal{R}_1} - \delta_2\frac{\mathcal{I}_2}{\mathcal{R}_2}, \tag{5.3}$$

where $C' = \gamma_3 + \gamma_4 + \mu$, and

$$L\overline{V_4} \le D' - (\eta_1 - \eta_2)\mathcal{S}, \tag{5.4}$$

where $D' = \gamma_1 + \gamma_2 + \delta_1 + \delta_2 + \frac{1}{2}(\sigma_1^2 \vee \sigma_2^2)$.

Applying Itô's formula to $\overline{V_5}$, we have

$$L\overline{V_5} = \mathcal{N}^\theta\big(\Lambda - \alpha\mathcal{N}\big) + \frac{\theta}{2}\mathcal{N}^{\theta-1}(\sigma_1^2 \vee \sigma_2^2),$$

where $\mathcal{N} = \mathcal{S} + \mathcal{I}_1 + \mathcal{I}_2 + \mathcal{R}_1 + \mathcal{R}_2$ and $\alpha = \min\{\gamma, \gamma_1, \gamma_2, \gamma_3, \gamma_4\}$. Thus,

$$L\overline{V_5} \le -\frac{1}{2}\Big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\Big)\sum_i X_i^{\theta+1} + E', \tag{5.5}$$

for some constant E'.

Now, we consider the $\mathcal{C}^2$ function

$$\overline{V} = M\overline{V_1} + \overline{V_2} + \overline{V_3} + \overline{V_4} + \overline{V_5},$$

where $M > 0$ is chosen such that

$$-MA'(H-1) + B' + C' + D' + E' \le -2.$$

Combining (5.1)-(5.5), we obtain

$$L\overline{V} \le -2 + M\beta_1\mathcal{I}_1 + M\beta_2\mathcal{I}_2 - M(2\mu\beta_2\mathcal{R}_2)^{1/2} - \frac{\Lambda}{\mathcal{S}} - \mu\frac{\mathcal{R}_2}{\mathcal{S}} - \delta_1\frac{\mathcal{I}_1}{\mathcal{R}_1} - \delta_2\frac{\mathcal{I}_2}{\mathcal{R}_2} - (\eta_1 - \eta_2)\mathcal{S}$$
$$- \frac{1}{2}\Big(\alpha - \frac{\theta}{2}(\sigma_1^2 \vee \sigma_2^2)\Big)\sum_i X_i^{\theta+1}.$$

Following the same argument as in Theorem 4.1, we conclude that

$$L\overline{V} \le -1, \quad \text{for } (\mathcal{S}, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}_1, \mathcal{R}_2) \in \mathbb{R}^5_+ \setminus D_\nu.$$

Hence, the expected return time satisfies

$$\mathbb{E}(\tau_{D_\nu}) \le \overline{V}(\mathcal{S}(0), \mathcal{I}_1(0), \mathcal{I}_2(0), \mathcal{R}_1(0), \mathcal{R}_2(0)) < \infty.$$

Therefore, the solution $(\mathcal{S}, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}_1, \mathcal{R}_2)$ of (1.3) is positive recurrent in the domain $\mathbb{R}^5_+ \setminus D_\nu$. Hence, by Definition 2.4, the assertion is proved. □

Remark 5.2. We discuss a few special cases by restricting σ_1 and σ_2. The following are the situations:

Case 1: When $\sigma_1 \neq 0$ and $\sigma_2 = 0$. This represents that only the transmission rate for the first disease has been perturbed stochastically. In this case, although the transmission rate for the second disease has not been perturbed, changes in the dynamics of the first disease will indirectly influence the second disease. For instance, the behavior or interactions of individuals afflicted with the first disease (e.g., isolation, enhanced precautions) may change and impact the dynamics of the second disease's transmission. Over time, the stochastic perturbation in the first disease transmission can accumulate and lead to population level changes. These changes may have an indirect effect on susceptible, infected or recovered population from both the diseases. This impacts the overall epidemic dynamics and changes the course of the second disease.

Case 2: When $\sigma_1 = 0$ and $\sigma_2 \neq 0$. This represents that only the transmission rate for the second disease has been perturbed stochastically. This case is similar to case 1. Thus, perturbing only the second disease transmission can still have an indirect and complex effects on the overall epidemic dynamics.

Case 3: When $\sigma_1 = 0$ and $\sigma_2 = 0$. This represents the deterministic case, which is system (1.1).

Remark 5.3. We have constructed similar Lyapunov functional for proving both the existence of T-periodic solution and stationary distribution. The function has been chosen to satisfy the specific requirements of both the results. Although, different Lyapunov functional can be constructed for each problem. This choice was made in order to maintain uniformity and clarity, which provides the reader a unified framework for study.

6. Numerical simulations

This section includes discussion for numerical simulation results of the proposed stochastic models corresponding to the theoretical results proved in the previous sections.

Example 6.1. We have chosen the following parameter values in the system (1.3): $\Lambda(t) = 0.1$, $\beta_1 = 0.1$, $\beta_2 = 0.1$, $\gamma = 0.02$, $\gamma_1 = 0.02$, $\gamma_2 = 0.02$, $\gamma_3 = 0.1$, $\gamma_4 = 0.1$, $\delta_1 = 0.02$, $\delta_2 = 0.02$, $\sigma_1 = 0.1$ and $\sigma_2 = 0.1$. Further, we compute the condition

$$G = \frac{(2\Lambda\beta_1)^{\frac{1}{2}}}{\left(\gamma_1 + \gamma_2 + \delta_1 + \delta_2 + \gamma + (\sigma_1^2 + \sigma_2^2)\right)} = 1.1785 > 1.$$

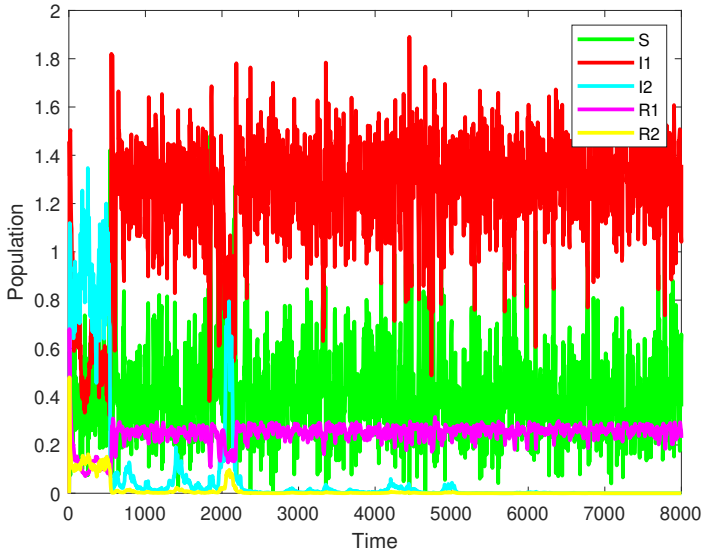

Figure 2. Trajectories of the stochastic system (1.3) obtained by the parameter values given in Example 6.1 and showing the existence of a T-periodic solution. The colored curves represent stochastic sample paths. Their oscillatory behavior around a stable mean over long time intervals suggests the presence of a nontrivial T-periodic solution in the presence of randomness.

To illustrate the existence of a positive T-periodic solution, we simulate the stochastic system over a long time horizon. As shown in Figure 2, all compartments exhibit bounded fluctuations and indicate recurrent oscillatory behavior, which suggests a form of periodicity in the presence of stochastic perturbations. In particular, the infected compartment $\mathcal{I}_1$ exhibits significant periodic oscillations, while the susceptible and recovered classes demonstrate bounded variations that align

with this periodicity. These results visually support our theoretical result based on Lyapunov function techniques and Khasminskii's theory.

The purpose of this example is to confirm the existence of a positive T-periodic solution in accordance with Theorem 4.1, rather than to assess long-term coexistence or extinction of either infection.

Example 6.2. In this example, we choose the following parameter values for system (1.3): $\Lambda = 0.07$, $\beta_1 = 0.1$, $\sigma_1^2 = 0.02$, $\sigma_2^2 = 0.04$, so $\sigma_1^2 \vee \sigma_2^2 = 0.04$, $\gamma_1 = 0.02$, $\gamma_2 = 0.02$, $\delta_1 = 0.02$, $\delta_2 = 0.02$, $\gamma = 0.02$. We compute

$$H = \frac{2\sqrt{\beta_1 \Lambda}}{(\sigma_1^2 \vee \sigma_2^2) + \gamma_1 + \gamma_2 + \delta_1 + \delta_2 + \gamma} = 1.19523 > 1.$$

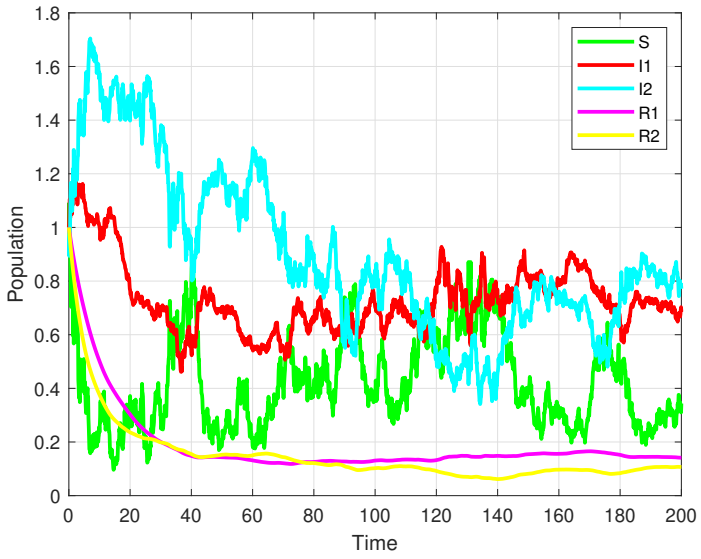

FIGURE 3. Sample trajectories of the stochastic system (1.3) under parameter values given in Example 6.2 and satisfying the positive recurrence condition given in Theorem 5.1. The compartments S, $\mathcal{I}_1$, $\mathcal{I}_2$, $\mathcal{R}_1$, and $\mathcal{R}_2$ are observed to fluctuate within bounded regions over a long period which indicates that the stochastic system admits a stationary distribution.

Figure 3 illustrates the behavior of the stochastic system when the parameter values (given in Example 6.2) fulfill the threshold condition for positive recurrence, as discussed in 5.1. The trajectories of all compartments remain within a stable range over time, which indicates the existence of a unique stationary distribution. It is important to note that, the infected class $\mathcal{I}_2$ stabilizes around a relatively higher mean compared to other classes, while $\mathcal{I}_1$ and the recovered classes fluctuate mildly around their mean values.

7. Discussions and concluding remarks

This article introduces a stochastic epidemic model that incorporates two different epidemiological frameworks, SIR and SIRS each governed by different nonlinear incidence rates. The randomness in disease transmission, an important aspect in real-world epidemics, is modeled by introducing stochastic perturbations in the infection rates. Specifically, in the system (1.1), the deterministic transmission rates $\beta_j dt$ are replaced by $\beta_j dt + \sigma_j dB_j(t)$ for $j = 1, 2$ to account for environmental fluctuations.

Our primary objective was to explore the long-term behavior of this model (1.3) under stochastic effects. We established sufficient conditions for the existence of a nontrivial positive T-periodic

solution, using Lyapunov functions and Khasminskii's method for stochastic periodic systems. We further showed the existence of a unique stationary distribution under an appropriate threshold condition, signifying long-term disease persistence under randomness.

Compared to previous studies, this work deals with the additional complexity arising from having two pathogens with different reinfection dynamics (SIRS and SIR), and multiplicative noise terms affecting transmission. Proving the existence of stationary distribution and periodic solutions under such a setting is significantly more involved due to the nonlinearity and the stochastic interactions between the compartments.

This model offers valuable insights into the dynamics of two interacting infectious diseases; however, it does have its limitations. For instance, we have assumed that stochasticity only enters through the transmission rates, while recovery and mortality processes are kept deterministic. The analysis is also restricted to specific noise structures (multiplicative Brownian motions), and future work could explore more general Lévy noise or correlated perturbations.

A potential extension of this work can involve the development of digital twins, which serve as real-time, data-integrated virtual representations of epidemic processes. Given that the present model accounts for two distinct epidemic dynamics, a digital twin approach could enhance its applicability by allowing real-time updates, scenario testing, and adaptive intervention strategies. The concept and mathematical foundation for such digital twins using Stieltjes differential equations, as proposed by Area et al. [1, 2], offer a novel perspective for extending the current theoretical basis toward real-time and adaptive disease modeling.

From an epidemiological perspective, our model captures the interaction between co-circulating pathogens in a fluctuating environment. However, for direct applicability to specific diseases, further refinement is required, including parameter estimation from data and possibly incorporating age structure, spatial effects, or vaccination strategies.

In summary, this work contributes to the mathematical understanding of stochastic epidemic models involving multiple transmission routes. It lays the foundation for more realistic multi-pathogen models and highlights the analytical challenges and richness of such systems.

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