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Research Article

Dynamic Analysis of Extinction and Stationary Distribution of a Stochastic Dual-Strain SEIR Epidemic Model with Double Saturated Incidence Rates

S. Saravanan, C. Monica*

Department of Mathematics, Vellore Institute of Technology, Vellore, 632014, Tamilnadu, India
E-mail: monica.c@vit.ac.in

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Abstract: This study aims to enhance and extend the mathematical model of a dynamic stochastic dual-strain SEIR epidemic with a double-saturated incidence rate. The model is represented by a nonlinear system of differential equations that describe the dynamics of susceptible, exposed, infected and recovered individuals, with the exposed and infected compartments further divided into sub-classes for the first and second strains. We develop an innovative stochastic epidemic model where drug-sensitive and drug-resistant infected groups interact through mutation. The primary objective is to determine the existence and uniqueness of a positive global solution using a well-deservedly constructed Lyapunov function, enabling a deeper analysis of the system's complexities. This analytical framework reveals the interactions between disease transmission, treatment dynamics, and stochastic influences. A significant contribution to this work is defining the stochastic basic reproduction number $\mathcal{R}_0$ as a threshold for the progression of both strains. Under low noise conditions and $\mathcal{R}_0^* > 1$, the model predicts the emergence of an ergodic stationary distribution, offering insights into long-term disease trends. Conversely, in high-noise scenarios, $\widehat{\mathcal{R}}_0^* < 1$, the analysis explores the extinction and persistence of drug-sensitive and drug-resistant infections. Our analytical results are further confirmed by simulations of epidemics spreading across drug-sensitive and drug-sensitive populations. Based on our simulations and theoretical predictions, we find that they are closely aligned.

***Keywords*:** drug sensitive and resistance, extinction, Lyapunov function, stationary distribution, stochastic Susceptible Exposed Infected and Recovered (SEIR) epidemic model

MSC: 34N05, 60H10, 60H20

Abbreviation

$\mathcal{SEIR}$	Susceptible Exposed Infected and Recovered.
a.s.	almost surely.
SDE	Stochastic Differential Equation.
HBV	Hepatitis B Virus.
HIV	Human Immunodeficiency Virus.
SIRI	Susceptible Infected Recovered and Immune.

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1. Introduction

The foundational work of Kermack and McKendrick in 1927 [1] laid the groundwork for a significant portion of mathematical modeling within the field of epidemiology. Their pioneering efforts introduced a systematic approach to understanding the spread of infectious diseases through mathematical frameworks. A key aspect of their contribution is the Susceptible Infected Removed (SIR) model, an acronym for susceptible, infected, and recovered compartments. This model has proven to be highly effective in analyzing a wide range of infectious diseases by categorizing the population into those who are susceptible to infection, those who are currently infected, and those who have recovered and presumably gained immunity. However, the SIR model has its limitations. It does not account for the incubation period, the time between exposure to the pathogen and the onset of infectiousness. To address this gap, an additional compartment is introduced, leading to the development of the SEIR model. The SEIR model stands for susceptible, exposed, infected, and recovered. The 'exposed' compartment represents individuals who have been exposed to the pathogen but are not yet infectious. This modification allows for a more accurate representation of disease dynamics, especially for diseases with a significant incubation period. The SEIR model has become a critical tool in the study of infectious diseases. Numerous scholarly articles have employed this model to investigate various epidemics and pandemics. For instance, the SEIR framework has been instrumental in understanding the spread of COVID-19 [2, 3], providing insights into how the virus propagates through populations and helping to inform public health interventions. Similarly, it has been used to study other infectious diseases such as the HBV [4] and HIV [5], among others. By incorporating the exposed compartment, researchers can better predict the course of an epidemic and evaluate the potential impact of different control strategies.

The process of mutation has been observed in numerous infections, including influenza, dengue fever, COVID-19, HIV, and tuberculosis [6–10]. These mutations result in the emergence of multiple strains of a pathogen, which complicates the landscape of infectious disease dynamics. The presence of multiple strains necessitates the development of more sophisticated mathematical models. Models that incorporate two or more strains are particularly effective in examining and understanding the evolution of these strains within a single disease outbreak. As an illustration, one research examined the worldwide dynamics of the multi-strain SEIR epidemic model, focusing on its use in the COVID-19 pandemic [11]. This approach helps researchers to understand how different strains interact, spread, and impact public health measures. By considering multiple strains, the model provides a more comprehensive understanding of the disease dynamics and can better inform strategies for intervention and control. Furthermore, recent research has delved into the examination and optimal management of a dual and multi-strain SEIR infection model [12, 13]. These studies focus on developing strategies to control the spread of diseases with multiple strains, taking into account factors such as transmission rates, mutation rates, and the effectiveness of different intervention measures. This research highlights the importance of considering multiple strains in epidemiological modeling to effectively manage and mitigate the impact of infectious diseases.

Drugs are in increasing demand in both livestock and human medicine to treat and prevent diseases, as well as to maintain productivity in food animals [14, 15]. This widespread use has been linked to the development of drug resistance, which refers to the reduced effectiveness of a treatment in curing diseases. Consequently, drug resistance poses a significant threat to public health [16, 17]. Numerous studies have been conducted to understand and characterize the emergence and spread of drug resistance in various systems [18–20]. For instance, Pecerska et al. [21] proposed a method for quantifying the transmission fitness costs associated with multi-drug-resistant tuberculosis. Karmakar et al. [22] estimated tuberculosis drug resistance amplification rates in high-burden settings, shedding light on how resistance spreads in areas heavily affected by the disease. Friedman et al. [23] introduced a mathematical model that explores the evolution of two bacterial strains drug-resistant and non-drug-resistant, within a population of patients and healthcare workers in a hospital setting. This model provides insights into how drug resistance can proliferate in healthcare environments.

Additionally, Kitaro et al. [24] conducted a study using both modeling and bifurcation analysis to understand tuberculosis with a multi-drug-resistant compartment, incorporating chemoprophylaxis treatment functions. This approach helps identify critical points where interventions could be most effective. Abatih et al. [25] developed a modeling framework that includes a susceptible class, a class infected with drug-sensitive bacteria, and a class infected with drug-resistant bacteria among pigs, highlighting the importance of monitoring and managing drug resistance in livestock. Moreover, Yang et al. [26] constructed a mathematical model that includes optimal strategies for age-specific vaccination

and antiviral treatment against influenza dynamics. This model aims to identify the most effective ways to combat influenza outbreaks while considering the role of drug resistance.

This paper presents a dual-strain stochastic SEIR model that incorporates both drug-sensitive and drug-resistant populations. By introducing stochastic noise, we capture the random fluctuations that can affect the disease dynamics, especially in the context of mutation-driven transitions between drug-sensitive and drug-resistant strains. The main objectives of this study are to investigate the existence and uniqueness of an ergodic stationary distribution, determine the thresholds for disease persistence or extinction, and examine the influence of stochasticity on the spread of drug resistance. We also explore the interaction between the two strains and how their dynamics are influenced by both deterministic factors (e.g., transmission rates and mutation) and stochastic noise.

This paper proceeds as follows: Section 2 introduces a dual strain SEIR epidemic model (1) and (3), detailing both deterministic and stochastic components. Section 3 defines the notations, terms, and some preliminaries used in subsequent analyses. In Section 4, we establish the existence and uniqueness of an ergodic stationary distribution for drug-sensitive and drug-resistant infections by constructing a stochastic Lyapunov function that aligns with the solutions to the system (3). Section 5 outlines conditions under which infections may become extinct. Theoretical findings are supported by examples with numerical simulations provided in Section 6. Section 7 provides a concise discussion of the main results and outlines directions for future research. Elaborated in this section.

2. Model

Environmental fluctuations play a vital role in natural ecosystems, encompassing factors such as temperature variations, the immunological state of hosts, and incubation periods. Occasionally, even small fluctuations can suppress population explosions in dynamic systems. SDEs provide a more realistic representation of these influencing factors compared to deterministic systems [27], offering additional realism. Numerous scholars have incorporated stochastic perturbations into their models, considering both biological and mathematical perspectives [28–31]. However, to date, there has been limited exploration of the stochastic SIRI system with saturated incidence and relapse, particularly concerning drug resistance mutation. Therefore, we propose a dual-strain SEIR model to account for transmissions involving both drug-sensitive and drug-resistant strains, specifically addressing drug-resistance mutation as follows:

$$\begin{aligned}
\frac{d\mathcal{S}}{dt} &= \Theta - \frac{\beta_1 \mathcal{S}\mathcal{I}_1}{1+h_1\mathcal{I}_1} - \frac{\beta_2 \mathcal{S}\mathcal{I}_2}{1+h_1\mathcal{I}_2} - \rho\mathcal{S},\\
\frac{d\mathcal{E}_1}{dt} &= \frac{\beta_1 \mathcal{S}\mathcal{I}_1}{1+h_1\mathcal{I}_1} - (\eta+\rho)\mathcal{E}_1,\\
\frac{d\mathcal{E}_2}{dt} &= \frac{\beta_2 \mathcal{S}\mathcal{I}_2}{1+h_2\mathcal{I}_2} - (\delta+\rho)\mathcal{E}_2,\\
\frac{d\mathcal{I}_1}{dt} &= \eta\mathcal{E}_1 - (\xi+\rho)\mathcal{I}_1,\\
\frac{d\mathcal{I}_2}{dt} &= \delta\mathcal{E}_2 - (\gamma+\rho)\mathcal{I}_2,\\
\frac{d\mathcal{R}}{dt} &= \xi\mathcal{I}_1 + \gamma\mathcal{I}_2 - \rho\mathcal{R}.
\end{aligned} \tag{1}$$

In accordance with the non-negative terms. In Tables 1 and 2, detailed environmental illustrations are shown for the parameters.

Table 1. Model variables and description

Variables	Description
$\mathcal{S}(t)$	Susceptible population
$\mathcal{E}_1(t)$	Drug-sensitive individuals were exposed to strain-I
$\mathcal{E}_2(t)$	Drug-resistance individuals were exposed to strain-II
$\mathcal{I}_1(t)$	People infected with drug-sensitive strain-I
$\mathcal{I}_2(t)$	People infected with drug-sensitive strain-II
$\mathcal{R}(t)$	Recovered population

Table 2. Model parameters and description

Parameters	Description
Θ	Recruitment rate of birth (or) immigration
β_1	Transmission rate of drug-sensitive people
β_2	Transmission rate of drug-resistance people
h_1	Saturated factor that measures inhibitory effect in the strain-I
h_2	Saturated factor that measures inhibitory effect in the strain-II
ρ	Natural death rate
η	Strain-I latency rate of drug-sensitive
δ	Strain-II latency rate of drug- resistance
ξ	Recovery rate infective individuals of Strain-I
γ	Recovery rate infective individuals of Strain-II

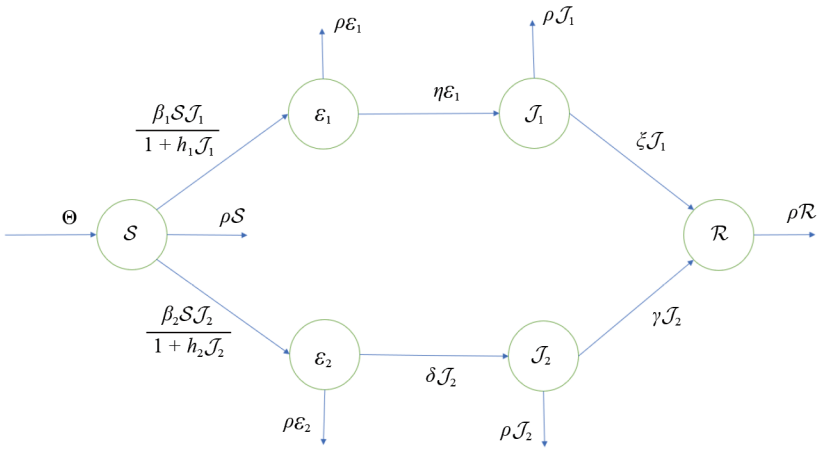


Figure 1. The detailed flowchart of deterministic $\mathcal{S}$, $\mathcal{E}_1$, $\mathcal{E}_2$, $\mathcal{I}_1$, $\mathcal{I}_2$, $\mathcal{R}$ epidemic model of system (1)

In this model, $\frac{1}{1+h\mathcal{I}}$ measures the inhibition effect resulting from the behavioral changes of the people who are affected by the disease when their numbers are crowded with other infected individuals, while $\mathcal{S}(t)\mathcal{I}(t)$ measures the infection forces. The flowchart illustrating the dynamics of the $\mathcal{S}$, $\mathcal{E}_1$, $\mathcal{E}_2$, $\mathcal{I}_1$, $\mathcal{I}_2$, $\mathcal{R}$ epidemic model is presented in Figure 1, depicting the transitions between the different compartments of susceptible, exposed, infectious, and recovered individuals. An invariant set of positive values defines the region Ξ of model (1) is,

$$\Xi = \left\{(\mathcal{S}, \mathcal{E}_1, \mathcal{E}_2, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}) \in \mathbb{R}_6^+ : 0 < \mathcal{S}+\mathcal{E}_1+\mathcal{E}_2+\mathcal{I}_1+\mathcal{I}_2+\mathcal{R} \leq \frac{\Theta}{\rho},\right.$$

$$\left.\mathcal{S} > 0, \mathcal{E}_1 > 0, \mathcal{E}_2 > 0, \mathcal{I}_1 > 0, \mathcal{I}_2 > 0, \mathcal{R} > 0\right\}.$$

Therefore, this is the basic reproduction number

$$\mathcal{R}_0 = \max\{\mathcal{R}_1, \mathcal{R}_2\}. \tag{2}$$

$$\mathcal{R}_1 = \frac{\Theta\beta_1\eta}{\rho(\eta+\rho)(\xi+\rho)}, \quad \mathcal{R}_2 = \frac{\Theta\beta_2\delta}{\rho(\delta+\rho)(\gamma+\rho)}.$$

Here, $\mathcal{R}_1$ denotes the reproduction number associated with strain-1, while $\mathcal{R}_2$ denotes the reproduction number associated with strain-2. These parameters quantify the potential for each strain to spread within the population. An individual infected with the virus is expected to cause an average of two secondary transmissions from that individual in a susceptible population. According to the threshold value $\mathcal{R}_0$, it displays the following behavior of the solution:

• According to model (1), if $\mathcal{R}_0 < 1$, there exists a unique disease-free equilibrium $\mathcal{E}_0 = \left(\mathcal{S}^0, \mathcal{E}_1^0, \mathcal{E}_2^0, \mathcal{I}_1^0, \mathcal{I}_2^0, \mathcal{R}^0\right) = \left(\frac{\Theta}{\rho}, 0, 0, 0, 0, 0\right)$, which is globally asymptotically stable.

• When $\mathcal{R}_0 > 1$ in addition to $\mathcal{E}_0$, model (1), contains a global asymptotically stable positive endemic equilibrium $\mathcal{E}^* = (\mathcal{S}^*, \mathcal{E}_1^*, \mathcal{E}_2^*, \mathcal{I}_1^*, \mathcal{I}_2^*, \mathcal{R}^*)$. The specific proof is similar to references [32, 33].

In this paper, our aim is to delve into the dynamics of a stochastic SEIR epidemic model featuring both drug-resistant and drug-sensitive strains. Our focus is on investigating and discussing the presence of a stationary distribution for the model's solutions. By exploring how these strains interact within the population dynamics, we aim to shed light on the epidemiological implications of drug resistance and contribute new insights to the field of infectious disease modeling.

The population dynamics of an ecosystem are inherently influenced by fluctuations in the environment. These fluctuations can significantly impact the behavior and interactions of species within the ecosystem. It has become evident that stochastic models, which account for randomness and variability, offer a more realistic portrayal of these dynamics compared to traditional deterministic models. In stochastic modeling, there are various approaches to incorporate these effects. For instance, some studies introduce noise into the transmission parameters of the model, reflecting uncertainties in how diseases or interactions spread among individuals. Others integrate stochasticity by considering noise that is proportional to each state variable, capturing fluctuations in population sizes or other ecological variables over time [34–37]. By adopting stochastic modeling techniques, researchers can better simulate and understand the complex dynamics of ecosystems. These methods allow for the exploration of how random events and fluctuations in environmental conditions influence population trends, species interactions, and ultimately, the stability of ecological systems. Thus, stochastic models play a crucial role in bridging the gap between theoretical models and real-world observations in ecology and epidemiology.

Now, we assume the environmental fluctuations are white noise type, which are directly proportional to $\mathcal{S}$, $\mathcal{E}_1$, $\mathcal{E}_2$, $\mathcal{I}_1$, $\mathcal{I}_2$, and $\mathcal{R}$ and influenced on $\frac{d\mathcal{S}}{dt}$, $\frac{d\mathcal{E}_1}{dt}$, $d\mathcal{E}_2 dt$, $\frac{d\mathcal{I}_1}{dt}$, $\frac{d\mathcal{I}_2}{dt}$, and $\frac{d\mathcal{R}}{dt}$ in model (1). The stochastic SEIR model is given by:

$$
\begin{aligned}
d\mathcal{S} &= \left[\Theta - \frac{\beta_1 \mathcal{S}\mathcal{I}_1}{1+h_1\mathcal{I}_1} - \frac{\beta_2 \mathcal{S}\mathcal{I}_2}{1+h_1\mathcal{I}_2} - \rho\mathcal{S}\right] dt + \rho_1 \mathcal{S} d\mathcal{B}_1(t),\\
d\mathcal{E}_1 &= \left[\frac{\beta_1 \mathcal{S}\mathcal{I}_1}{1+h_1\mathcal{I}_1} - (\eta+\rho)\mathcal{E}_1\right] dt + \rho_2 \mathcal{E}_1 d\mathcal{B}_2(t),\\
d\mathcal{E}_2 &= \left[\frac{\beta_2 \mathcal{S}\mathcal{I}_2}{1+h_2\mathcal{I}_2} - (\delta+\rho)\mathcal{E}_2\right] dt + \rho_3 \mathcal{E}_2 d\mathcal{B}_3(t),\\
d\mathcal{I}_1 &= [\eta\mathcal{E}_1 - (\xi+\rho)\mathcal{I}_1]\, dt + \rho_4 \mathcal{I}_1 d\mathcal{B}_4(t),\\
d\mathcal{I}_2 &= [\delta\mathcal{E}_2 - (\gamma+\rho)\mathcal{I}_2]\, dt + \rho_5 \mathcal{I}_2 d\mathcal{B}_5(t),\\
d\mathcal{R} &= [\xi\mathcal{I}_1 + \gamma\mathcal{I}_2 - \rho\mathcal{R}]\, dt + \rho_6 \mathcal{R} d\mathcal{B}_6(t),
\end{aligned}
\tag{3}
$$

where $\mathcal{B}_i$'s are standard one-dimensional independent Brownian motion, $\rho_i > 0$ are the intensity of the white noise, ($i = 1, 2, 3, 4, 5, 6$) that is specified on a complete probability area $(\Omega, \mathcal{F}, \mathcal{P})$ with $\{\mathcal{F}_t\}_{t\in\mathbb{R}^4_+}$ filtration fulfilling the normal requirements, $\mathcal{F}_0$ contains all $\mathcal{P}$-null sets, whereas $\{\mathcal{F}_t\}_{t\in\mathbb{R}^4_+}$ value is increasing and continuous [38]. In all cases, the coefficients are not negative, $\Theta > 0$. This study examines the incidence rates of double saturation, along with rates of drug resistance and drug sensitivity, in the context of disease mutation using the stochastic SEIRS epidemic model. It will be necessary to investigate a model's dynamical properties in order to determine whether it exhibits a stationary ergodic distribution.

3. Preliminaries

The analysis of the epidemic model's dynamical behavior hinges on assessing whether the solution remains globally positive throughout. In summary, the study conclusively establishes both the existence and uniqueness of a globally positive solution. To comprehensively explore the dynamical behavior of model (3), an initial thorough investigation into its static properties is imperative. The first step is to consider stochastic differential equations in d-dimensions

$$
d\mathcal{X} = f(\mathcal{X}(\mathfrak{t}), \mathfrak{t})dt + g(\mathcal{X}(\mathfrak{t}), \mathfrak{t})d\mathcal{B}(t) \quad \text{for} \quad \mathfrak{t} \geq \mathfrak{t}_0,
$$

with the initial value for $\mathcal{X}(0) = \mathcal{X}_0 \in \mathbb{R}^d$. The differential operator $\mathfrak{L}$ associated with the equation above can be defined as follows:

$$
\mathfrak{L} = \frac{\partial}{\partial t} + \sum_{\mathfrak{i}=1}^{d} f_{\mathfrak{i}}(\mathcal{X}, t)\frac{\partial}{\partial \mathcal{X}_{\mathfrak{i}}} + \frac{1}{2}\sum_{\mathfrak{i},\mathfrak{j}=1}^{d} \left[g^T(\mathcal{X}, t)g(\mathcal{X}, t)\right]_{\mathfrak{i}\mathfrak{j}} \frac{\partial^2}{\partial \mathcal{X}_{\mathfrak{i}}\partial \mathcal{X}_{\mathfrak{j}}}.
$$

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

$$\mathfrak{L}V(\mathcal{X}, t) = V_t(\mathcal{X}, t) + V_{\mathcal{X}}(\mathcal{X}, t)f(\mathcal{X}, t) + \frac{1}{2}\text{trace}\left[g^T(\mathcal{X}, t)V_{\mathcal{X}\mathcal{X}}(\mathcal{X}, t)g(\mathcal{X}, t)\right],$$

where $V_t = \dfrac{\partial V}{\partial t}$, $V_{\mathcal{X}} = \left(\dfrac{\partial V}{\partial \mathcal{X}_1}, \dfrac{\partial V}{\partial \mathcal{X}_2}, ..., \dfrac{\partial V}{\partial \mathcal{X}_d}\right)$, $V_{\mathcal{X}\mathcal{X}} = \left(\dfrac{\partial^2 V}{\partial \mathcal{X}_i \partial \mathcal{X}_j}\right)_{d\times d}$. Thus, by Itô's formula [39], if $\mathcal{X}(t) \in \mathbb{R}^d$, then

$$dV(\mathcal{X}(t), t) = \mathfrak{L}V(\mathcal{X}(t), t)dt + V_{\mathcal{X}}(\mathcal{X}(t), t)g(\mathcal{X}(t), t)d\mathcal{B}(t).$$

To establish the conclusions presented in Sections 4 and 5, it is imperative to introduce the following theorem and accompanying lemmas.

Lemma 1 [39] There is a bounded open domain $\theta_\varepsilon \subset \mathbb{R}^6_+$ with regular boundary Ω satisfying conditions $\mathfrak{H}_1$ and $\mathfrak{H}_2$, then $y(t)$ has a unique ergodic stationary distribution $k(\cdot)$ in any positive value among.

$\mathfrak{H}_1$: there exist a constant $\mathcal{M}$ and the following inequality is $\sum_{i,\,j=1}^{6} \mathfrak{a}_{ij}(\mathrm{x})\mathfrak{Z}_i\mathfrak{Z}_j \geq \mathcal{M}||\mathfrak{Z}||^2$.

$\mathfrak{H}_2$: some neighborhood θ_ε and a non-negative $\mathfrak{C}^2$-function follows $\mathcal{L}\mathcal{V} \leq -1$ for any $\mathbb{R}^6_+/\theta_\varepsilon$.

$$\Longrightarrow \mathcal{P}\left\{\lim_{\mathcal{T}\to+\infty}\frac{1}{\mathcal{T}}\int_0^{\mathcal{T}}\mathcal{F}(\mathrm{x}(t))dt = \int_{\mathbb{R}^d}\mathcal{F}(\mathrm{x})k(d\mathrm{x})\right\} = 1, \quad \forall \mathrm{x} \in \mathbb{R}^d,$$

where $\mathcal{F}(\cdot)$ is an integrable function with respect to k.

Lemma 2 For any initial value $(\mathcal{S}(0), \mathcal{E}_1(0), \mathcal{E}_2(0), \mathcal{I}_1(0), \mathcal{I}_2(0), \mathcal{R}(0))^T \in \mathbb{R}^6_+$, there has a unique positive solution $(\mathcal{S}(t), \mathcal{E}_1(t), \mathcal{E}_2(t), \mathcal{I}_1(t), \mathcal{I}_2(t), \mathcal{R}(t))^T$ of the model (3) on $t \geq 0$ almost surely (a.s).

Remark 1 The proof of Lemma 2 follows a similar approach as in [40–42], and is consistent with the methodology described in [43]. Therefore, the proof will not be provided here.

4. Ergodic stationary distribution

It is essential to analyze under what conditions a disease will persist and become prevalent within a population when studying epidemic dynamical models. For example, deterministic models often rely on proving that their endemic equilibrium acts as a global attractor or achieves global asymptotic stability. However, for system (3), the concept of an endemic equilibrium does not apply. This section endeavors to establish the existence of an ergodic stationary distribution, drawing on Has'minskii's theory [39], which provides insights into the enduring presence of the disease.

The following stochastic differential equation describes $\mathcal{X}(t)$ as a homogeneous Markov process in $\mathbb{R}^6_+$:

$$d\mathcal{X}(t) = f(\mathcal{X}(t))dt + \sum_{i=1}^{k} g_i(\mathcal{X})d\mathcal{B}_i(t).$$

Therefore, the diffusion matrix can be described as follows:

$$\mathcal{A}(\mathrm{x}) = (a_{(ij)}(\mathrm{x})),\ a_{ij}(\mathrm{x}) = \sum_{i=1}^{k} g_i^p(\mathrm{x})g_i^q(\mathrm{x}).$$

Theorem 1 If

$$\mathcal{R}_0^* = \max\{\mathcal{R}_1^*, \mathcal{R}_2^*\} > 1, \tag{4}$$

$$\mathcal{R}_1^* = \frac{\Theta\beta_1\eta}{\left(\rho+\frac{\rho_1^2}{2}\right)\left(\eta+\rho+\frac{\rho_2^2}{2}\right)\left(\xi+\rho+\frac{\rho_4^2}{2}\right)},\ \ \mathcal{R}_2^* = \frac{\Theta\beta_2\delta}{\left(\rho+\frac{\rho_1^2}{2}\right)\left(\delta+\rho+\frac{\rho_3^2}{2}\right)\left(\gamma+\rho+\frac{\rho_5^2}{2}\right)}$$

the model (3) has a unique ergodic stationary distribution $k(\cdot)$ in any positive value.

Proof. The investigation unfolds through two key steps. Initially, we rigorously verify the uniform elliptic condition $(\mathfrak{H}_1)$ to ensure its validity. Subsequently, we methodically construct a non-negative Lyapunov function that satisfies the condition $(\mathfrak{H}_2)$ as outlined in Lemma 1. These steps collectively form the foundation for advancing our theoretical understanding in this study.

Step 1

The diffusion matrices of the model (3) is

$$\mathcal{A} = \begin{pmatrix} \rho_1^2\mathcal{S}^2 & 0 & 0 & 0 & 0 & 0 \\ 0 & \rho_2^2\mathcal{E}_1^2 & 0 & 0 & 0 & \\ 0 & 0 & \rho_3^2\mathcal{E}_2^2 & 0 & 0 & 0 \\ 0 & 0 & 0 & \rho_4^2\mathcal{I}_1^2 & 0 & 0 \\ 0 & 0 & 0 & 0 & \rho_5^2\mathcal{I}_2^2 & 0 \\ 0 & 0 & 0 & 0 & 0 & \rho_6^2\mathcal{R}^2 \end{pmatrix}$$

Choose, $\mathcal{M} = \min_{(\mathcal{S},\,\mathcal{E}_1,\,\mathcal{E}_2,\,\mathcal{I}_1,\,\mathcal{I}_2,\,\mathcal{R})\in\bar{\theta}_\varepsilon\subset\mathbb{R}_+^6}\left\{\rho_1^2\mathcal{S}^2+\rho_2^2\mathcal{E}_1^2+\rho_3^2\mathcal{E}_2^2+\rho_4^2\mathcal{I}_1^2+\rho_5^2\mathcal{I}_2^2+\rho_6^2\mathcal{R}^2\right\}$.

$$\begin{aligned}\sum_{i,\,j=1}^{6}\mathfrak{a}_{i,\,j}(\mathcal{S},\,\mathcal{E}_1,\,\mathcal{E}_2,\,\mathcal{I}_1,\,\mathcal{I}_2,\,\mathcal{R})\mathfrak{Z}_i\mathfrak{Z}_j &= \rho_1^2\mathcal{S}^2\mathfrak{Z}_1^2+\rho_2^2\mathcal{E}_1^2\mathfrak{Z}_2^2+\rho_3^2\mathcal{E}_2^2\mathfrak{Z}_3^2+\rho_4^2\mathcal{I}_1^2\mathfrak{Z}_4^2 \\ &\quad+\rho_5^2\mathcal{I}_2^2\mathfrak{Z}_5^2+\rho_6^2\mathcal{R}^2\mathfrak{Z}_6^2 \\ &\geq \mathcal{M}|\mathfrak{Z}|^2,\end{aligned} \tag{5}$$

where, $(\mathcal{S},\,\mathcal{E}_1,\,\mathcal{E}_2,\,\mathcal{I}_1,\,\mathcal{I}_2,\,\mathcal{R})\in\bar{\theta}_\varepsilon$ and $\mathfrak{Z}=(\mathfrak{Z}_1,\,\mathfrak{Z}_2,\,\mathfrak{Z}_3,\,\mathfrak{Z}_4,\,\mathfrak{Z}_5,\,\mathfrak{Z}_6)\in\mathbb{R}_+^6$.

Since $\mathcal{A}$ is positive, Lemma 1 implies that condition $\mathfrak{H}_1$ is satisfied.

Step 2

Constructing a $\mathfrak{C}^2$-function $\mathcal{V}$: $\mathbb{R}_+^6\to\mathbb{R}$ as follows,

$$\begin{aligned}\mathcal{V}(\mathcal{S},\mathcal{E}_1,\mathcal{E}_2,\mathcal{I}_1,\mathcal{I}_2,\mathcal{R}) &= \mathcal{M}\,(\mathcal{S}+\mathcal{E}_1+\mathcal{E}_2+\mathcal{I}_1+\mathcal{I}_2+\mathcal{R}-\alpha_4\ln\mathcal{I}_1-\alpha_5\ln\mathcal{I}_2\\ &\quad -\alpha_2\ln\mathcal{E}_1-\alpha_3\ln\mathcal{E}_2-\alpha_1\ln\mathcal{S})\\ &\quad +\frac{1}{\vartheta+1}(\mathcal{S}+\mathcal{E}_1+\mathcal{E}_2+\mathcal{I}_1+\mathcal{I}_2+\mathcal{R})^{\vartheta+1}-\ln\mathcal{S}-\ln\mathcal{E}_1\\ &\quad -\ln\mathcal{E}_2-\ln\mathcal{I}_1-\ln\mathcal{I}_2-\ln\mathcal{R}+(\mathcal{S}+\mathcal{E}_1+\mathcal{E}_2+\mathcal{I}_1+\mathcal{I}_2+\mathcal{R})\\ &= \mathcal{M}\mathcal{V}_1+\mathcal{V}_2+\mathcal{V}_3+\mathcal{V}_4+\mathcal{V}_5+\mathcal{V}_6+\mathcal{V}_7+\mathcal{V}_8+\mathcal{V}_9,\end{aligned}$$

where θ is a constant, $0<\theta<\dfrac{2\rho}{\rho_1^2\vee\rho_2^2\vee\rho_3^2\vee\rho_4^2\vee\rho_5^2\vee\rho_6^2}$,

$$\mathcal{V}_1=\mathcal{S}+\mathcal{E}_1+\mathcal{E}_2+\mathcal{I}_1+\mathcal{I}_2+\mathcal{R}-\alpha_4\ln\mathcal{I}_1-\alpha_5\ln\mathcal{I}_2-\alpha_2\ln\mathcal{E}_1-\alpha_3\ln\mathcal{E}_2-\alpha_1\ln\mathcal{S},$$

$$\mathcal{V}_2=\frac{1}{\vartheta+1}(\mathcal{S}+\mathcal{E}_1+\mathcal{E}_2+\mathcal{I}_1+\mathcal{I}_2+\mathcal{R})^{\theta+1},\quad \mathcal{V}_3=-\ln\mathcal{S},\quad \mathcal{V}_4=-\ln\mathcal{E}_1,\quad \mathcal{V}_5=-\ln\mathcal{E}_2,$$

$$\mathcal{V}_6=-\ln\mathcal{I}_1,\quad \mathcal{V}_7=-\ln\mathcal{I}_2,\quad \mathcal{V}_8=-\ln\mathcal{R},\quad \mathcal{V}_9=\mathcal{S}+\mathcal{E}_1+\mathcal{E}_2+\mathcal{I}_1+\mathcal{I}_2+\mathcal{R},$$

$$\alpha_1=\frac{\Theta}{\rho+\dfrac{\rho_1^2}{2}},\quad \alpha_2=\frac{\Theta}{\eta+\rho+\dfrac{\rho_2^2}{2}},\quad \alpha_3=\frac{\Theta}{\delta+\rho+\dfrac{\rho_3^2}{2}},$$

$$\alpha_4=\frac{\Theta}{\xi+\rho+\dfrac{\rho_4^2}{2}},\quad \alpha_5=\frac{\Theta}{\gamma+\rho+\dfrac{\rho_5^2}{2}}.$$

Therefore, we consider $\mathcal{M}>0$ and make it large enough satisfies the condition,

$$-\mathcal{M}\Lambda+\mathcal{P}\le -2,$$

where, $\mathcal{P}$ is positive constant. There is an easy way to check that

$$\liminf_{i\to\infty(\mathcal{S},\mathcal{E}_1,\mathcal{E}_2,\mathcal{I}_1,\mathcal{I}_2,\mathcal{R})\in\mathbb{R}_+^6/\bar{\theta}_{\mathfrak{n}}}\Gamma(\mathcal{S},\mathcal{E}_1,\mathcal{E}_2,\mathcal{I}_1,\mathcal{I}_2,\mathcal{R})=+\infty.$$

Where, $\bar{\theta}_{\mathfrak{n}} = \left(\frac{1}{\mathfrak{n}}, \mathfrak{n}\right) \times \left(\frac{1}{\mathfrak{n}}, \mathfrak{n}\right) \times \left(\frac{1}{\mathfrak{n}}, \mathfrak{n}\right) \times \left(\frac{1}{\mathfrak{n}}, \mathfrak{n}\right) \times \left(\frac{1}{\mathfrak{n}}, \mathfrak{n}\right), \times \left(\frac{1}{\mathfrak{n}}, \mathfrak{n}\right)$. Moreover, $\Gamma(\mathcal{S}, \mathcal{E}_1, \mathcal{E}_2, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R})$ is a continuous function. Hence $\Gamma(\mathcal{S}, \mathcal{E}_1, \mathcal{E}_2, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R})$ must have a minimum point $\Gamma(\mathcal{S}^0, \mathcal{E}_1^0, \mathcal{E}_2^0, \mathcal{I}_1^0, \mathcal{I}_2^0, \mathcal{R}^0)$ in the interior of $\mathbb{R}_+^6$.

Then we define a non-negative $\mathfrak{C}^2$-function, $d\mathcal{V}$: $\mathbb{R}_+^6 \to \mathbb{R}_+$ as follows,

$$d\mathcal{V} = (\mathcal{S}, \mathcal{E}_1, \mathcal{E}_2, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}) = \Gamma(\mathcal{S}, \mathcal{E}_1, \mathcal{E}_2, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}) - \Gamma(\mathcal{S}^0, \mathcal{E}_1^0, \mathcal{E}_2^0, \mathcal{I}_1^0, \mathcal{I}_2^0, \mathcal{R}^0).$$

As a result of applying the Itô's formula,

$$\begin{aligned}
\mathcal{L}\mathcal{V}_1 = & -\alpha_1\frac{\Theta}{\mathcal{S}} - \alpha_1\frac{\beta_1\mathcal{I}_1}{1+h_1\mathcal{I}_1} - \alpha_1\frac{\beta_2\mathcal{I}_2}{1+h_2\mathcal{I}_2} - \alpha_1\rho + \alpha_1\frac{\rho_1^2}{2} \\
& - \alpha_2\frac{\beta_1\mathcal{S}\mathcal{I}_1}{(1+h_1\mathcal{I}_1)\mathcal{E}_1} - \alpha_2(\eta+\rho) + \alpha_2\frac{\rho_2^2}{2} - \alpha_3\frac{\beta_2\mathcal{S}\mathcal{I}_2}{(1+h_2\mathcal{I}_2)\mathcal{E}_2} \\
& - \alpha_3(\delta+\rho) + \alpha_3\frac{\rho_3^2}{2} - \alpha_4\frac{\eta\mathcal{E}_1}{\mathcal{I}_1} - \alpha_4(\xi+\rho) + \alpha_4\frac{\rho_4^2}{2} \\
& - \alpha_5\frac{\delta\mathcal{E}_2}{\mathcal{I}_2} - \alpha_5(\gamma+\rho) + \alpha_5\frac{\rho_5^2}{2} \\
= & -\alpha_1\frac{\Theta}{\mathcal{S}} - \alpha_1\frac{\beta_1\mathcal{I}_1}{1+h_1\mathcal{I}_1} - \alpha_1\frac{\beta_2\mathcal{I}_2}{1+h_2\mathcal{I}_2} - \alpha_2\frac{\beta_1\mathcal{S}\mathcal{I}_1}{(1+h_1\mathcal{I}_1)\mathcal{E}_1} - \alpha_3\frac{\beta_2\mathcal{S}\mathcal{I}_2}{(1+h_2\mathcal{I}_2)\mathcal{E}_2} \\
& - \alpha_4\frac{\eta\mathcal{E}_1}{\mathcal{I}_1} - \alpha_5\frac{\delta\mathcal{E}_2}{\mathcal{I}_2} + \alpha_1\left(\rho+\frac{\rho_1^2}{2}\right) + \alpha_2\left(\eta+\rho+\frac{\rho_2^2}{2}\right) + \alpha_4\left(\xi+\rho+\frac{\rho_4^2}{2}\right) \\
& + \alpha_3\left(\delta+\rho+\frac{\rho_3^2}{2}\right) + \alpha_5\left(\gamma+\rho+\frac{\rho_5^2}{2}\right). \qquad (6)
\end{aligned}$$

Using inequalities $a+b \geq 2\sqrt{ab}$, $a, b > 0$.

$$\mathcal{L}\mathcal{V}_1 \leq -2\left[\alpha_1\alpha_2\alpha_4\frac{\Theta\beta_1\eta}{1+h_1\mathcal{I}_1}\right]^{\frac{1}{2}} - 2\left[\alpha_1\alpha_3\alpha_5\frac{\Theta\beta_2\delta}{1+h_2\mathcal{I}_2}\right]^{\frac{1}{2}} + \alpha_1\frac{\beta_1\mathcal{I}_1}{1+h_1\mathcal{I}_1}$$

$$+ \alpha_1\frac{\beta_2\mathcal{I}_2}{1+h_2\mathcal{I}_2} + \alpha_1\left(\rho+\frac{\rho_1^2}{2}\right) + \alpha_2\left(\eta+\rho+\frac{\rho_2^2}{2}\right)$$

$$+ \alpha_4\left(\xi+\rho+\frac{\rho_4^2}{2}\right) + \alpha_3\left(\delta+\rho+\frac{\rho_3^2}{2}\right) + \alpha_5\left(\gamma+\rho+\frac{\rho_5^2}{2}\right)$$

$$\leq -4\left[\alpha_1\alpha_2\alpha_4\frac{\Theta\beta_1\eta}{1+h_1\mathcal{I}_1}\cdot\alpha_1\alpha_3\alpha_5\frac{\Theta\beta_2\delta}{1+h_2\mathcal{I}_2}\right]^{1/4} + \alpha_1\frac{\beta_1\mathcal{I}_1}{1+h_1\mathcal{I}_1}$$

$$+ \alpha_1\frac{\beta_2\mathcal{I}_2}{1+h_2\mathcal{I}_2} + \alpha_1\left(\rho+\frac{\rho_1^2}{2}\right) + \alpha_2\left(\eta+\rho+\frac{\rho_2^2}{2}\right)$$

$$+ \alpha_4\left(\xi+\rho+\frac{\rho_4^2}{2}\right) + \alpha_3\left(\delta+\rho+\frac{\rho_3^2}{2}\right) + \alpha_5\left(\gamma+\rho+\frac{\rho_5^2}{2}\right)$$

$$\leq -4\Theta\left\{\max[\mathcal{R}_1^*, \mathcal{R}_2^*]^{1/4} - 1\right\} + \alpha_1\frac{\beta_1\mathcal{I}_1}{1+h_1\mathcal{I}_1} + \alpha_1\frac{\beta_2\mathcal{I}_2}{1+h_2\mathcal{I}_2}$$

$$= -\mathcal{Y} + \alpha_1\frac{\beta_1\mathcal{I}_1}{1+h_1\mathcal{I}_1} + \alpha_1\frac{\beta_2\mathcal{I}_2}{1+h_2\mathcal{I}_2}, \tag{7}$$

where, $\mathcal{Y} = 4\Theta\left\{\max[\mathcal{R}_1^*, \mathcal{R}_2^*]^{1/4} - 1\right\} > 0$.

Similarly,

$$
\begin{aligned}
\mathcal{L}\mathcal{V}_2 &= (\mathcal{S}+\mathcal{E}_1+\mathcal{E}_2+\mathcal{I}_1+\mathcal{I}_2+\mathcal{R})^{\vartheta}(\Theta-\rho\mathcal{S}-\rho\mathcal{E}_1-\rho\mathcal{E}_2-\rho\mathcal{I}_1-\rho\mathcal{I}_2-\rho\mathcal{R}) \\
&\quad+\frac{\vartheta}{2}(\mathcal{S}+\mathcal{E}_1+\mathcal{E}_2+\mathcal{I}_1+\mathcal{I}_2+\mathcal{R})^{\vartheta-1} \\
&\quad\times(\rho_1^2\mathcal{S}^2+\rho_2^2\mathcal{E}_2^2+\rho_3^2\mathcal{E}_2^2+\rho_4^2\mathcal{I}_1^2+\rho_5^2\mathcal{I}_2^2+\rho_6^2\mathcal{R}^2) \\
&\leq (\mathcal{S}+\mathcal{E}_1+\mathcal{E}_2+\mathcal{I}_1+\mathcal{I}_2+\mathcal{R})^{\vartheta}[\Theta-\rho(\mathcal{S}+\mathcal{E}_1+\mathcal{E}_2+\mathcal{I}_1+\mathcal{I}_2+\mathcal{R})] \\
&\quad+\frac{\vartheta}{2}(\mathcal{S}+\mathcal{E}_1+\mathcal{E}_2+\mathcal{I}_1+\mathcal{I}_2+\mathcal{R})^{\vartheta-1} \\
&\quad\times(\rho_1^2\mathcal{S}^2+\rho_2^2\mathcal{E}_2^2+\rho_3^2\mathcal{E}_2^2+\rho_4^2\mathcal{I}_1^2+\rho_5^2\mathcal{I}_2^2+\rho_6^2\mathcal{R}^2) \\
&\leq \Theta(\mathcal{S}+\mathcal{E}_1+\mathcal{E}_2+\mathcal{I}_1+\mathcal{I}_2+\mathcal{R})^{\vartheta}-\left[\rho-\frac{\vartheta}{2}\left(\rho_1^2\vee\rho_2^2\vee\rho_3^2\vee\rho_4^2\vee\rho_5^2\vee\rho_6^2\right)\right] \\
&\quad\times(\mathcal{S}+\mathcal{E}_1+\mathcal{E}_2+\mathcal{I}_1+\mathcal{I}_2+\mathcal{R})^{\vartheta+1} \\
&\leq \Theta-\frac{1}{2}\left[\rho-\frac{\vartheta}{2}\left(\rho_1^2\vee\rho_2^2\vee\rho_3^2\vee\rho_4^2\vee\rho_5^2\vee\rho_6^2\right)\right] \\
&\quad\times\left(\mathcal{S}^{\vartheta+1}+\mathcal{E}_1^{\vartheta+1}+\mathcal{E}_2^{\vartheta+1}+\mathcal{I}_1^{\vartheta+1}+\mathcal{I}_2^{\vartheta+1}+\mathcal{R}^{\vartheta+1}\right)
\end{aligned}
$$

$$
\mathcal{L}\mathcal{V}_2 \leq \Theta+\Lambda, \tag{8}
$$

where,

$$
\begin{aligned}
\Lambda = \sup_{(\mathcal{S},\,\mathcal{E}_1,\,\mathcal{E}_2,\,\mathcal{I}_1,\,\mathcal{I}_2,\,\mathcal{R})\in\mathbb{R}_+^6}\Big\{&\Theta(\mathcal{S}+\mathcal{E}_1+\mathcal{E}_2+\mathcal{I}_1+\mathcal{I}_2+\mathcal{R})^{\vartheta} \\
&-\frac{1}{2}\left[\rho-\frac{\vartheta}{2}\left(\rho_1^2\vee\rho_2^2\vee\rho_3^2\vee\rho_4^2\vee\rho_5^2\vee\rho_6^2\right)\right] \\
&\left(\mathcal{S}^{\vartheta+1}+\mathcal{E}_1^{\vartheta+1}+\mathcal{E}_2^{\vartheta+1}+\mathcal{I}_1^{\vartheta+1}+\mathcal{I}_2^{\vartheta+1}+\mathcal{R}^{\vartheta+1}\right)\Big\}.
\end{aligned}
$$

We can also get

$$\mathcal{L}\mathcal{V}_3 = -\frac{\Theta}{\mathcal{S}} + \frac{\beta_1 \mathcal{I}_1}{1+h_1\mathcal{I}_1} + \frac{\beta_2 \mathcal{I}_2}{1+h_2\mathcal{I}_2} + \rho + \frac{\rho_1^2}{2} \tag{9}$$

$$\mathcal{L}\mathcal{V}_4 = -\frac{\beta_1 \mathcal{S}\mathcal{I}_1}{(1+h_1\mathcal{I}_1)\mathcal{E}_1} + \eta + \rho + \frac{\rho_2^2}{2} \tag{10}$$

$$\mathcal{L}\mathcal{V}_5 = -\frac{\beta_2 \mathcal{S}\mathcal{I}_2}{(1+h_2\mathcal{I}_2)\mathcal{E}_2} + \delta + \rho + \frac{\rho_3^2}{2} \tag{11}$$

$$\mathcal{L}\mathcal{V}_6 = -\frac{\eta \mathcal{E}_1}{\mathcal{I}_1} + \xi + \rho + \frac{\rho_4^2}{2} \tag{12}$$

$$\mathcal{L}\mathcal{V}_7 = -\frac{\delta \mathcal{E}_2}{\mathcal{I}_2} + \gamma + \rho + \frac{\rho_5^2}{2} \tag{13}$$

$$\mathcal{L}\mathcal{V}_8 = -\frac{\xi \mathcal{I}_1}{\mathcal{R}} - \frac{\gamma \mathcal{I}_2}{\mathcal{R}} + \rho + \frac{\rho_4^2}{2} \tag{14}$$

$$\mathcal{L}\mathcal{V}_9 = \Theta - \rho\left(\mathcal{S} + \mathcal{E}_1 + \mathcal{E}_2 + \mathcal{I}_1 + \mathcal{I}_2 + \mathcal{R}\right) \tag{15}$$

We obtained

$$\begin{aligned}
\mathcal{L}\mathcal{V} = & -\mathcal{M}\Theta + \mathcal{M}\frac{\alpha_1\beta_1\mathcal{I}_1}{1+h_1\mathcal{I}_1} + \mathcal{M}\frac{\alpha_1\beta_2\mathcal{I}_2}{1+h_2\mathcal{I}_2} \\
& - \frac{1}{2}\left[\rho - \frac{\vartheta}{2}\left(\rho_1^2 \vee \rho_2^2 \vee \rho_3^2 \vee \rho_4^2 \vee \rho_5^2 \vee \rho_6^2\right)\right] \\
& \times \left(\mathcal{S}^{\vartheta+1} + \mathcal{E}_1^{\vartheta+1} + \mathcal{E}_2^{\vartheta+1} + \mathcal{I}_1^{\vartheta+1} + \mathcal{I}_2^{\vartheta+1} + \mathcal{R}^{\vartheta+1}\right) - \frac{\Theta}{\mathcal{S}} + \frac{\beta_1\mathcal{I}_1}{1+h_1\mathcal{I}_1} \\
& + \frac{\beta_2\mathcal{I}_2}{1+h_2\mathcal{I}_2} + 6\rho - \frac{\beta_1\mathcal{S}\mathcal{I}_1}{(1+h_1\mathcal{I}_1)\mathcal{E}_1} + \eta - \frac{\beta_2\mathcal{S}\mathcal{I}_2}{(1+h_2\mathcal{I}_2)\mathcal{E}_2} + \delta - \frac{\eta\mathcal{E}_1}{\mathcal{I}_1} + \xi \\
& - \frac{\delta\mathcal{E}_2}{\mathcal{I}_2} + \gamma - \frac{\xi\mathcal{I}_1}{\mathcal{R}} - \frac{\gamma\mathcal{I}_2}{\mathcal{R}} - \rho\left(\mathcal{S} + \mathcal{E}_1 + \mathcal{E}_2 + \mathcal{I}_1 + \mathcal{I}_2 + \mathcal{R}\right) \\
& + \left(\frac{\rho_1^2 + \rho_2^2 + \rho_3^2 + \rho_4^2 + \rho_5^2 + \rho_6^2}{2}\right)
\end{aligned}$$

$$
\begin{aligned}
&= -\mathcal{M}\Theta + \mathcal{M}\frac{\alpha_1\beta_1\mathcal{I}_1}{1+h_1\mathcal{I}_1} + \mathcal{M}\frac{\alpha_1\beta_2\mathcal{I}_2}{1+h_2\mathcal{I}_2} + \frac{\beta_1\mathcal{I}_1}{1+h_1\mathcal{I}_1} + \frac{\beta_2\mathcal{I}_2}{1+h_2\mathcal{I}_2} \\
&- \frac{\beta_1\mathcal{S}\mathcal{I}_1}{(1+h_1\mathcal{I}_1)\mathcal{E}_1} - \frac{\beta_2\mathcal{S}\mathcal{I}_2}{(1+h_2\mathcal{I}_2)\mathcal{E}_2} - \frac{\Theta}{S} - \frac{\eta\mathcal{E}_1}{\mathcal{I}_1} - \frac{\delta\mathcal{E}_2}{\mathcal{I}_2} - \frac{\xi\mathcal{I}_1}{\mathcal{R}} - \frac{\gamma\mathcal{I}_2}{\mathcal{R}} \\
&- \frac{1}{2}\left[\rho - \frac{\vartheta}{2}\left(\rho_1^2 \vee \rho_2^2 \vee \rho_3^2 \vee \rho_4^2 \vee \rho_5^2 \vee \rho_6^2\right)\right] \\
&\times \left(\mathcal{S}^{\vartheta+1} + \mathcal{E}_1^{\vartheta+1} + \mathcal{E}_2^{\vartheta+1} + \mathcal{I}_1^{\vartheta+1} + \mathcal{I}_2^{\vartheta+1} + \mathcal{R}^{\vartheta+1}\right) \\
&- \rho\left(\mathcal{S} + \mathcal{E}_1 + \mathcal{E}_2 + \mathcal{I}_1 + \mathcal{I}_2 + \mathcal{R}\right) + \Theta + \eta + \delta + \gamma + 6\rho \\
&+ \left(\frac{\rho_1^2 + \rho_2^2 + \rho_3^2 + \rho_4^2 + \rho_5^2 + \rho_6^2}{2}\right). \qquad (16)
\end{aligned}
$$

Define a bounded closed domain Π_ε:

$$
\begin{aligned}
\Pi_\varepsilon = \Big\{ &(\mathcal{S}, \mathcal{E}_1, \mathcal{E}_2, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}) \in \mathbb{R}_+^6 : \varepsilon_1 \le \mathcal{S} \le 1/\varepsilon_1, \varepsilon_2 \le \mathcal{E}_1 \le 1/\varepsilon_2, \\
&\varepsilon_3 \le \mathcal{E}_2 \le 1/\varepsilon_3, \varepsilon_4 \le \mathcal{I}_1 \le 1/\varepsilon_4, \varepsilon_5 \le \mathcal{I}_2 \le 1/\varepsilon_5, \varepsilon_6 \le \mathcal{R} \le 1/\varepsilon_6 \Big\},
\end{aligned}
$$

where $0 < \varepsilon < 1$ is sufficiently small constant. In the set Π_ε^c, making ε small enough such that it satisfies the following condition holds.

$$
-\frac{\Theta}{\varepsilon_1} + \mathcal{H} \le -1 \qquad (17)
$$

$$
-\mathcal{M}\Theta + \frac{\mathcal{M}c_1\beta_1\mathcal{I}_1}{h_1} + \mathcal{J} \le -1 \qquad (18)
$$

$$
-\mathcal{M}\Theta + \frac{\mathcal{M}c_2\beta_2\mathcal{I}_2}{h_2} + \mathcal{Q} \le -1 \qquad (19)
$$

$$
-2\left(\frac{\beta_1\rho}{\varepsilon_2}\right)^{1/2} + \mathcal{J} \le -1 \qquad (20)
$$

$$-2\left(\frac{\beta_2\rho}{\varepsilon_3}\right)^{1/2}+\mathfrak{Q}\leq -1 \tag{21}$$

$$-\frac{\xi}{\varepsilon_6}-\frac{\gamma}{\varepsilon_6}+\mathcal{H}\leq -1 \tag{22}$$

$$-\frac{1}{4}\left[\rho-\frac{\vartheta}{2}\left(\rho_1^2\vee\rho_2^2\vee\rho_3^2\vee\rho_4^2\vee\rho_5^2\vee\rho_6^2\right)\right]\frac{1}{\varepsilon_i^{\vartheta+1}}+\mathcal{H}\leq -1 \tag{23}$$

It is shown that $\mathcal{H}$, $\mathcal{J}$ and $\mathfrak{Q}$ are positive constants. Here $\varepsilon=(\varepsilon_1,...,\varepsilon_6)$ is sufficiently small constant. For convenience, $\mathbb{R}^6_+/\Pi_\varepsilon$ is divided into 12 domains:

$$\Pi_1=\left\{(\mathcal{S},\mathcal{E}_1,\mathcal{E}_2,\mathcal{I}_1,\mathcal{I}_2,\mathcal{R})\in\mathbb{R}^6_+\colon 0<\mathcal{S}<\varepsilon_1\right\}$$

$$\Pi_2=\left\{(\mathcal{S},\mathcal{E}_1,\mathcal{E}_2,\mathcal{I}_1,\mathcal{I}_2,\mathcal{R})\in\mathbb{R}^6_+\colon 0<\mathcal{I}_1<\varepsilon_4\right\}$$

$$\Pi_3=\left\{(\mathcal{S},\mathcal{E}_1,\mathcal{E}_2,\mathcal{I}_1,\mathcal{I}_2,\mathcal{R})\in\mathbb{R}^6_+\colon 0<\mathcal{I}_2<\varepsilon_5,\mathcal{S}\geq\varepsilon_1\right\}$$

$$\Pi_4=\left\{(\mathcal{S},\mathcal{E}_1,\mathcal{E}_2,\mathcal{I}_1,\mathcal{I}_2,\mathcal{R})\in\mathbb{R}^6_+\colon 0<\mathcal{E}_1<\varepsilon_2\right\}$$

$$\Pi_5=\left\{(\mathcal{S},\mathcal{E}_1,\mathcal{E}_2,\mathcal{I}_1,\mathcal{I}_2,\mathcal{R})\in\mathbb{R}^6_+\colon 0<\mathcal{E}_2<\varepsilon_3,\mathcal{S}\geq\varepsilon_1\right\}$$

$$\Pi_6=\left\{(\mathcal{S},\mathcal{E}_1,\mathcal{E}_2,\mathcal{I}_1,\mathcal{I}_2,\mathcal{R})\in\mathbb{R}^6_+\colon 0<\mathcal{R}<\varepsilon_6,\mathcal{I}_1\geq\varepsilon_4,\mathcal{I}_2\geq\varepsilon_5\right\}$$

$$\Pi_7=\left\{(\mathcal{S},\mathcal{E}_1,\mathcal{E}_2,\mathcal{I}_1,\mathcal{I}_2,\mathcal{R})\in\mathbb{R}^6_+\colon \mathcal{S}>\frac{1}{\varepsilon_1}\right\}$$

$$\Pi_8=\left\{(\mathcal{S},\mathcal{E}_1,\mathcal{E}_2,\mathcal{I}_1,\mathcal{I}_2,\mathcal{R})\in\mathbb{R}^6_+\colon \mathcal{E}_1>\frac{1}{\varepsilon_2}\right\}$$

$$\Pi_9=\left\{(\mathcal{S},\mathcal{E}_1,\mathcal{E}_2,\mathcal{I}_1,\mathcal{I}_2,\mathcal{R})\in\mathbb{R}^6_+\colon \mathcal{E}_2>\frac{1}{\varepsilon_3}\right\}$$

$$\Pi_{10}=\left\{(\mathcal{S},\mathcal{E}_1,\mathcal{E}_2,\mathcal{I}_1,\mathcal{I}_2,\mathcal{R})\in\mathbb{R}^6_+\colon \mathcal{I}_1>\frac{1}{\varepsilon_4}\right\}$$

$$\Pi_{11} = \left\{(\mathcal{S}, \mathcal{E}_1, \mathcal{E}_2, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}) \in \mathbb{R}^6_+ : \mathcal{I}_2 > \frac{1}{\varepsilon_5}\right\}$$

$$\Pi_{12} = \left\{(\mathcal{S}, \mathcal{E}_1, \mathcal{E}_2, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}) \in \mathbb{R}^6_+ : \mathcal{R} > \frac{1}{\varepsilon_6}\right\}.$$

Obviously, $\mathbb{R}^6_+ / \Pi_\varepsilon = \Pi_1 \cup \Pi_2 \cup \Pi_3 \cup ... \cup \Pi_{12}$. Then, we only need to verify that $\mathcal{LV}(\mathcal{S}, \mathcal{E}_1, \mathcal{E}_2, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}) \leq -1$ in the above 12 domains.

Case I: Consider that $(\mathcal{S}, \mathcal{E}_1, \mathcal{E}_2, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}) \in \Pi_1$, by (17), we get

$$\begin{aligned}
\mathcal{LV} = & -\frac{\Theta}{\mathcal{S}} + \frac{\mathcal{M}\alpha_1\beta_1\mathcal{I}_1}{h_1} + \frac{\mathcal{M}\alpha_2\beta_2\mathcal{I}_2}{h_2} - \frac{1}{2}\left[\rho - \frac{\vartheta}{2}\left(\rho_1^2 \vee \rho_2^2 \vee \rho_3^2 \vee \rho_4^2 \vee \rho_5^2 \vee \rho_6^2\right)\right] \\
& \times \left(\mathcal{S}^{\vartheta+1} + \mathcal{E}_1^{\vartheta+1} + \mathcal{E}_2^{\vartheta+1} + \mathcal{I}_1^{\vartheta+1} + \mathcal{I}_2^{\vartheta+1} + \mathcal{R}^{\vartheta+1}\right) + \Theta + \eta + \delta + \xi + \gamma + 4\rho \\
& + \left(\frac{\rho_1^2 + \rho_2^2 + \rho_3^2 + \rho_6^2}{2}\right) \\
= & -\frac{\Theta}{\mathcal{S}} + \mathcal{H} \leq -\frac{\Theta}{\varepsilon_1} + \mathcal{H} \\
\leq & -1
\end{aligned}$$

where,

$$\begin{aligned}
\mathcal{H} = & \sup_{(\mathcal{S}, \mathcal{E}_1, \mathcal{E}_2, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}) \in \mathbb{R}^6_+} \left\{\frac{\mathcal{M}\alpha_1\beta_1\mathcal{I}_1}{h_1} + \frac{\mathcal{M}\alpha_2\beta_2\mathcal{I}_2}{h_2}\right. \\
& -\frac{1}{2}\left[\rho - \frac{\vartheta}{2}\left(\rho_1^2 \vee \rho_2^2 \vee \rho_3^2 \vee \rho_4^2 \vee \rho_5^2 \vee \rho_6^2\right)\right] \\
& \times \left(\mathcal{S}^{\vartheta+1} + \mathcal{E}_1^{\vartheta+1} + \mathcal{E}_2^{\vartheta+1} + \mathcal{I}_1^{\vartheta+1} + \mathcal{I}_2^{\vartheta+1} + \mathcal{R}^{\vartheta+1}\right) + \Theta + \eta \\
& \left. + \delta + \xi + \gamma + 4\rho + \left(\frac{\rho_1^2 + \rho_2^2 + \rho_3^2 + \rho_6^2}{2}\right)\right\}
\end{aligned}$$

Case II: Consider that $(\mathcal{S}, \mathcal{E}_1, \mathcal{E}_2, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}) \in \Pi_2$, by (18), we obtain

$$\begin{aligned}
\mathcal{L}\mathcal{V} = & -\mathcal{M}\Theta + \frac{\mathcal{M}\alpha_1\beta_1\mathcal{I}_1}{1+h_1\mathcal{I}_1} - \frac{1}{2}\left[\rho - \frac{\vartheta}{2}\left(\rho_1^2 \vee \rho_2^2 \vee \rho_3^2 \vee \rho_4^2 \vee \rho_5^2 \vee \rho_6^2\right)\right] \\
& \times \left(\mathcal{S}^{\vartheta+1} + \mathcal{E}_1^{\vartheta+1} + \mathcal{E}_2^{\vartheta+1} + \mathcal{I}_1^{\vartheta+1} + \mathcal{I}_2^{\vartheta+1} + \mathcal{R}^{\vartheta+1}\right) + \Theta + \eta \\
& + \delta + \gamma + \xi + 6\rho + \left(\frac{\rho_1^2 + \rho_2^2 + \rho_3^2 + \rho_6^2}{2}\right) \\
= & -\mathcal{M}\Theta + \frac{\mathcal{M}\alpha_1\beta_1\mathcal{I}_1}{1+h_1\mathcal{I}_1} + \mathcal{J} \\
\leq & -1
\end{aligned}$$

where,

$$\begin{aligned}
\mathcal{J} = & \sup_{(\mathcal{S},\, \mathcal{E}_1,\, \mathcal{E}_2,\, \mathcal{I}_1,\, \mathcal{I}_2,\, \mathcal{R}) \in \mathbb{R}_+^6} \left\{ \frac{\mathcal{M}\alpha_1\beta_1\mathcal{I}_1}{h_1} - \frac{1}{2}\left[\rho - \frac{\vartheta}{2}\left(\rho_1^2 \vee \rho_2^2 \vee \rho_3^2 \vee \rho_4^2 \vee \rho_5^2 \vee \rho_6^2\right)\right] \right. \\
& \times \left(\mathcal{S}^{\vartheta+1} + \mathcal{E}_1^{\vartheta+1} + \mathcal{E}_2^{\vartheta+1} + \mathcal{I}_1^{\vartheta+1} + \mathcal{I}_2^{\vartheta+1} + \mathcal{R}^{\vartheta+1}\right) + \Theta + \eta \\
& \left. + \delta + \xi + \gamma + 4\rho + \left(\frac{\rho_1^2 + \rho_2^2 + \rho_3^2 + \rho_6^2}{2}\right) \right\}
\end{aligned}$$

Case III: Consider that $(\mathcal{S},\, \mathcal{E}_1,\, \mathcal{E}_2,\, \mathcal{I}_1,\, \mathcal{I}_2,\, \mathcal{R}) \in \Pi_3$, in view of (19), we achieve

$$\begin{aligned}
\mathcal{L}\mathcal{V} = & -\mathcal{M}\Theta + \frac{\mathcal{M}\alpha_2\beta_2\mathcal{I}_2}{1+h_2\mathcal{I}_2} - \frac{1}{2}\left[\rho - \frac{\vartheta}{2}\left(\rho_1^2 \vee \rho_2^2 \vee \rho_3^2 \vee \rho_4^2 \vee \rho_5^2 \vee \rho_6^2\right)\right] \\
& \times \left(\mathcal{S}^{\vartheta+1} + \mathcal{E}_1^{\vartheta+1} + \mathcal{E}_2^{\vartheta+1} + \mathcal{I}_1^{\vartheta+1} + \mathcal{I}_2^{\vartheta+1} + \mathcal{R}^{\vartheta+1}\right) + \Theta + \eta \\
& + \delta + \gamma + \xi + 4\rho + \left(\frac{\rho_1^2 + \rho_2^2 + \rho_3^2 + \rho_6^2}{2}\right) \\
= & -\mathcal{M}\Theta + \frac{\mathcal{M}\alpha_2\beta_2\mathcal{I}_2}{1+h_2\mathcal{I}_2} + \mathcal{Q} \\
\leq & -1
\end{aligned}$$

where,

$$\mathcal{Q} = \sup_{(\mathcal{S},\,\mathcal{E}_1,\,\mathcal{E}_2,\,\mathcal{I}_1,\,\mathcal{I}_2,\,\mathcal{R})\in\mathbb{R}^6_+} \left\{ \frac{\mathcal{M}\alpha_2\beta_2\mathcal{I}_2}{h_2} - \frac{1}{2}\left[\rho - \frac{\vartheta}{2}\left(\rho_1^2 \vee \rho_2^2 \vee \rho_3^2 \vee \rho_4^2 \vee \rho_5^2 \vee \rho_6^2\right)\right] \right.$$

$$\times \left(\mathcal{S}^{\vartheta+1} + \mathcal{E}_1^{\vartheta+1} + \mathcal{E}_2^{\vartheta+1} + \mathcal{I}_1^{\vartheta+1} + \mathcal{I}_2^{\vartheta+1} + \mathcal{R}^{\vartheta+1}\right) + \Theta + \eta$$

$$\left. + \delta + \xi + \gamma + 4\rho + \left(\frac{\rho_1^2 + \rho_2^2 + \rho_3^2 + \rho_6^2}{2}\right)\right\}$$

Case IV: Consider that $(\mathcal{S},\,\mathcal{E}_1,\,\mathcal{E}_2,\,\mathcal{I}_1,\,\mathcal{I}_2,\,\mathcal{R}) \in \Pi_4$, considering (20), we receive

$$\mathcal{L}\mathcal{V} = -2\left(\frac{\beta_1\rho\mathcal{S}\mathcal{I}_1}{E_1}\right)^{1/2} + \frac{\mathcal{M}\alpha_1\beta_1\mathcal{I}_1}{1+h_1\mathcal{I}_1} - \frac{1}{2}\left[\rho - \frac{\vartheta}{2}\left(\rho_1^2 \vee \rho_2^2 \vee \rho_3^2 \vee \rho_4^2 \vee \rho_5^2 \vee \rho_6^2\right)\right]$$

$$\times \left(\mathcal{S}^{\vartheta+1} + \mathcal{E}_1^{\vartheta+1} + \mathcal{E}_2^{\vartheta+1} + \mathcal{I}_1^{\vartheta+1} + \mathcal{I}_2^{\vartheta+1} + \mathcal{R}^{\vartheta+1}\right) + \Theta + \eta$$

$$+ \delta + \gamma + \xi + 4\rho + \left(\frac{\rho_1^2 + \rho_2^2 + \rho_3^2 + \rho_6^2}{2}\right)$$

$$= -2\left(\frac{\beta_1\rho\mathcal{S}\mathcal{I}_1}{\mathcal{E}_1}\right)^{1/2} + \mathcal{J}$$

$$\leq -1$$

Case V: Consider that $(\mathcal{S},\,\mathcal{E}_1,\,\mathcal{E}_2,\,\mathcal{I}_1,\,\mathcal{I}_2,\,\mathcal{R}) \in \Pi_5$, in light of (21), we gather

$$\mathcal{L}\mathcal{V} = -2\left(\frac{\beta_2\rho\mathcal{S}\mathcal{I}_2}{\mathcal{E}_2}\right)^{1/2} + \frac{\mathcal{M}\alpha_2\beta_2\mathcal{I}_2}{1+h_2\mathcal{I}_2} - \frac{1}{2}\left[\rho - \frac{\vartheta}{2}\left(\rho_1^2 \vee \rho_2^2 \vee \rho_3^2 \vee \rho_4^2 \vee \rho_5^2 \vee \rho_6^2\right)\right]$$

$$\times \left(\mathcal{S}^{\vartheta+1} + \mathcal{E}_1^{\vartheta+1} + \mathcal{E}_2^{\vartheta+1} + \mathcal{I}_1^{\vartheta+1} + \mathcal{I}_2^{\vartheta+1} + \mathcal{R}^{\vartheta+1}\right) + \Theta + \eta$$

$$+ \delta + \gamma + \xi + 4\rho + \left(\frac{\rho_1^2 + \rho_2^2 + \rho_3^2 + \rho_6^2}{2}\right)$$

$$= -2\left(\frac{\beta_2\rho\mathcal{S}\mathcal{I}_2}{\mathcal{E}_2}\right)^{1/2} + \mathcal{Q}$$

$$\leq -1$$

Case VI: Consider that $(\mathcal{S},\, \mathcal{E}_1,\, \mathcal{E}_2,\, \mathcal{I}_1,\, \mathcal{I}_2,\, \mathcal{R}) \in \Pi_6$, because of (22), we get

$$\begin{aligned}
\mathcal{L}\mathcal{V} = & -\frac{\xi \mathcal{I}_1}{\mathcal{R}} - \frac{\gamma \mathcal{I}_2}{\mathcal{R}} + \frac{\mathcal{M}\alpha_1\beta_1\mathcal{I}_1}{1+h_1\mathcal{I}_1} + \frac{\mathcal{M}\alpha_2\beta_2\mathcal{I}_2}{1+h_2\mathcal{I}_2} \\
& -\frac{1}{2}\left[\rho - \frac{\vartheta}{2}\left(\rho_1^2 \vee \rho_2^2 \vee \rho_3^2 \vee \rho_4^2 \vee \rho_5^2 \vee \rho_6^2\right)\right] \\
& \times \left(\mathcal{S}^{\vartheta+1} + \mathcal{E}_1^{\vartheta+1} + \mathcal{E}_2^{\vartheta+1} + \mathcal{I}_1^{\vartheta+1} + \mathcal{I}_2^{\vartheta+1} + \mathcal{R}^{\vartheta+1}\right) + \Theta + \eta \\
& + \delta + \xi + \gamma + 4\rho + \left(\frac{\rho_1^2 + \rho_2^2 + \rho_3^2 + \rho_6^2}{2}\right) \\
= & -\frac{\xi \mathcal{I}_1}{\mathcal{R}} - \frac{\gamma \mathcal{I}_2}{\mathcal{R}} + \mathcal{H} \\
\leq & -1
\end{aligned}$$

Case VII: Consider that $(\mathcal{S},\, \mathcal{E}_1,\, \mathcal{E}_2,\, \mathcal{I}_1,\, \mathcal{I}_2,\, \mathcal{R}) \in \Pi_7$, we receive

$$\begin{aligned}
\mathcal{L}\mathcal{V} = & -\frac{1}{4}\left[\rho - \frac{\vartheta}{2}\left(\rho_1^2 \vee \rho_2^2 \vee \rho_3^2 \vee \rho_4^2 \vee \rho_5^2 \vee \rho_6^2\right)\right]\mathcal{S}^{\vartheta+1} \\
& -\frac{1}{4}\left[\rho - \frac{\vartheta}{2}\left(\rho_1^2 \vee \rho_2^2 \vee \rho_3^2 \vee \rho_4^2 \vee \rho_5^2 \vee \rho_6^2\right)\right]\mathcal{S}^{\vartheta+1} \\
& -\frac{1}{2}\left[\rho - \frac{\vartheta}{2}\left(\rho_1^2 \vee \rho_2^2 \vee \rho_3^2 \vee \rho_4^2 \vee \rho_5^2 \vee \rho_6^2\right)\right] \\
& \times \left(\mathcal{E}_1^{\vartheta+1} + \mathcal{E}_2^{\vartheta+1} + \mathcal{I}_1^{\vartheta+1} + \mathcal{I}_2^{\vartheta+1} + \mathcal{R}^{\vartheta+1}\right) + \frac{\mathcal{M}\alpha_1\beta_1\mathcal{I}_1}{1+h_1\mathcal{I}_1} + \frac{\mathcal{M}\alpha_2\beta_2\mathcal{I}_2}{1+h_2\mathcal{I}_2} \\
& + \Theta + \eta + \delta + \xi + \gamma + 4\rho + \left(\frac{\rho_1^2 + \rho_2^2 + \rho_3^2 + \rho_6^2}{2}\right) \\
\leq & -\frac{1}{4}\left[\rho - \frac{\vartheta}{2}\left(\rho_1^2 \vee \rho_2^2 \vee \rho_3^2 \vee \rho_4^2 \vee \rho_5^2 \vee \rho_6^2\right)\right]\mathcal{S}^{\vartheta+1} + \mathcal{H} \\
\leq & -\frac{1}{4}\left[\rho - \frac{\vartheta}{2}\left(\rho_1^2 \vee \rho_2^2 \vee \rho_3^2 \vee \rho_4^2 \vee \rho_5^2 \vee \rho_6^2\right)\right]\frac{1}{\varepsilon_1^{\vartheta+1}} + \mathcal{H}
\end{aligned}$$

Subsequently, providing under requirement (23) for $i = 1$. We get $\mathcal{L}\mathcal{V} \leq -1$ on Π_7.

Similarly, it follows from the equation (23) for $i = 2, \ldots, 6$, the same procedure can deduced for the compartments $\mathcal{E}_1, \mathcal{E}_2, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}$ on $\mathcal{LV} \leq -1$ and for Π_i, $i = 8, \ldots, 12$.

Based on the mentioned 12 cases above, it can be concluded that

$$\mathcal{LV}(\mathcal{S}, \mathcal{E}_1, \mathcal{E}_2, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}) \leq -1.$$

This confirms that condition $(\mathfrak{H}_2)$ stated in Lemma 1 has been fulfilled, indicating the existence of a stationary distribution for model (3) and establishing its ergodic nature. Thus, the proof is complete. □

Remark 2 Theorem 1, reveals that system (3) has a unique ergodic stationary distribution $k(\cdot)$, if

$$\mathcal{R}_0^* = \max \left\{ \mathcal{R}_1^* = \frac{\Theta\beta_1\eta}{\left(\rho + \frac{\rho_1^2}{2}\right)\left(\eta + \rho + \frac{\rho_2^2}{2}\right)\left(\xi + \rho + \frac{\rho_4^2}{2}\right)}, \right.$$

$$\left. \mathcal{R}_2^* = \frac{\Theta\beta_2\delta}{\left(\rho + \frac{\rho_1^2}{2}\right)\left(\delta + \rho + \frac{\rho_3^2}{2}\right)\left(\gamma + \rho + \frac{\rho_5^2}{2}\right)} \right\} > 1.$$

Note that the expression of $\mathcal{R}_0^*$ coincide with the threshold of $\mathcal{R}_0$ of the deterministic system (1), if the white noise is not taken into account. This shows that we generalize the results of deterministic system.

5. Extinction of the disease

This section focuses on examining the phenomenon of infection extinction, providing a distinct perspective on managing disease spread. Biologically, disease extinction denotes the eventual disappearance of diseases. The stochastic model (3) is instrumental in delineating the essential conditions required for disease extinction.

Lemma 3 Let $(\mathcal{S}(t), \mathcal{E}_1(t), \mathcal{E}_2(t), \mathcal{I}_1(t), \mathcal{I}_2(t), \mathcal{R}(t))$, be the solution of the model (3) with the any initial condition $(\mathcal{S}(0), \mathcal{E}_1(0), \mathcal{E}_2(0), \mathcal{I}_1(0), \mathcal{I}_2(0), \mathcal{R}(0)) \in \mathbb{R}_+^6$. Then

$$\lim_{t \to +\infty} \frac{1}{t} \left(\mathcal{S}(t) + \mathcal{E}_1(t) + \mathcal{E}_2(t) + \mathcal{I}_1(t) + \mathcal{I}_2(t) + \mathcal{R}(t)\right) = 0, \quad a.s. \tag{24}$$

Moreover, if $\rho > \frac{1}{t}\left(\rho_1^2 + \rho_2^2 + \rho_3^2 + \rho_4^2 + \rho_5^2 + \rho_6^2\right)$, then

$$\lim_{t\to\infty}\int_o^t \frac{\mathcal{S}(t)d\mathcal{B}_1(t)}{t}=0,\quad \lim_{t\to\infty}\int_o^t \frac{\mathcal{E}_1(t)d\mathcal{B}_2(t)}{t}=0,\quad a.s.$$

$$\lim_{t\to\infty}\int_o^t \frac{\mathcal{E}_2(t)d\mathcal{B}_3(t)}{t}=0,\quad \lim_{t\to\infty}\int_o^t \frac{\mathcal{I}_1(t)d\mathcal{B}_4(t)}{t}=0,\quad a.s. \tag{25}$$

$$\lim_{t\to\infty}\int_o^t \frac{\mathcal{I}_2(t)d\mathcal{B}_5(t)}{t}=0,\quad \lim_{t\to\infty}\int_o^t \frac{\mathcal{R}(t)d\mathcal{B}_6(t)}{t}=0,\quad a.s.$$

The proof of Lemma 3 could be accomplished by using the same method used by Zhao and Jiang [30] for Lemmas 2.1 and 2.2. Therefore, it can be skipped from this section.

Define the double threshold value:

$$\widehat{\mathcal{R}}_0^*=\max\left(\widehat{\mathcal{R}}_1^*,\widehat{\mathcal{R}}_2^*\right)<1, \tag{26}$$

where,

$$\widehat{\mathcal{R}}_1^*=\frac{\Theta\beta_1\eta}{\rho\left(\eta+\rho+\frac{\rho_2^2}{2}\right)\left(\xi+\rho+\frac{\rho_4^2}{2}\right)}$$

$$=\mathcal{R}_1-\frac{\Theta\beta_1\eta\rho_2^2\rho_4^2}{2\rho(\eta+\rho)(\xi+\rho)\left(\eta+\rho+\frac{\rho_2^2}{2}\right)\left(\xi+\rho+\frac{\rho_4^2}{2}\right)}.$$

$$\widehat{\mathcal{R}}_2^*=\frac{\Theta\beta_2\delta}{\rho\left(\delta+\rho+\frac{\rho_3^2}{2}\right)\left(\gamma+\rho+\frac{\rho_5^2}{2}\right)}$$

$$=\mathcal{R}_2-\frac{\Theta\beta_2\delta\rho_3^2\rho_5^2}{2\rho(\delta+\rho)(\gamma+\rho)\left(\delta+\rho+\frac{\rho_3^2}{2}\right)\left(\gamma+\rho+\frac{\rho_5^2}{2}\right)}.$$

Here, $\mathcal{R}_1$ and $\mathcal{R}_2$ are corresponding to the basic reproduction number for deterministic model (1).

Theorem 2 Assume that $\rho>\frac{1}{2}\left(\rho_1^2\vee\rho_2^2\vee\rho_3^2\vee\rho_4^2\vee\rho_5^2\vee\rho_6^2\right)$.

Let $(\mathcal{S}(t),\mathcal{E}_1(t),\mathcal{E}_2(t),\mathcal{I}_1(t),\mathcal{I}_2(t),\mathcal{R}(t))$ the solution of the model (3) with any initial condition $(\mathcal{S}(0),\mathcal{E}_1(0),\mathcal{E}_2(0),\mathcal{I}_1(0),\mathcal{I}_2(0),\mathcal{R}(0))\in\mathbb{R}_+^6$. If $\mathcal{R}_1^*<1$ and $\mathcal{R}_2^*<1$. Then $\mathcal{I}_1(t)$ and $\mathcal{I}_2(t)$, will go extinct almost surely.

$$\lim_{t\to\infty}\mathcal{I}_1(t)=0\quad a.s.\qquad \lim_{t\to\infty}\mathcal{I}_2(t)=0\quad a.s.$$

Meanwhile,

$$\lim_{t\to\infty} \mathcal{S}(t) = \frac{\Theta}{\rho}, \quad \lim_{t\to\infty} \mathcal{E}_1(t) = 0, \quad \lim_{t\to\infty} \mathcal{E}_2(t) = 0, \quad \lim_{t\to\infty} \mathcal{R}(t) = 0 \quad a.s. \tag{27}$$

Proof. (1) An equivalent $\mathcal{C}^2$-function to $\mathcal{P}_1(t)$ is constructed by,

$$\mathcal{P}_1(t) = \rho_2\mathcal{E}_1 + \rho_4\mathcal{I}_1.$$

Using Itô's formula to $\mathcal{P}_1(t)$ gives,

$$d(\ln\mathcal{P}_1) = \mathcal{L}(\ln\mathcal{P}_1)dt = \frac{1}{\mathcal{P}_1}\left\{\left[\frac{\beta_1\mathcal{S}(t)\mathcal{I}_1(t)}{1+h_1\mathcal{I}_1(t)} - (\eta+\rho)\mathcal{E}_1(t)\right]dt + \rho_2\mathcal{E}_1(t)d\mathcal{B}_2(t)\right.$$

$$\left.[\eta\mathcal{E}_1(t) - (\xi+\rho)\mathcal{I}_1(t)]\,dt + \rho_4\mathcal{I}_1(t)d\mathcal{B}_4(t)\right\}$$

$$d(\ln\mathcal{P}_1) = \frac{1}{\mathcal{P}_1}\left[\left(\beta_1\mathcal{S}(t) - (\eta+\rho) - \frac{\rho_2^2}{2}\right) + (\rho_2\mathcal{E}_1(t)d\mathcal{B}_2(t))\right.$$

$$\left.\left(\eta - (\xi+\rho) - \frac{\rho_4^2}{2}\right) + \rho_4\mathcal{I}_1(t)d\mathcal{B}_4(t)\right].$$

Similarly, integrating from 0 to t and then dividing by t on both sides given by,

$$\frac{\ln\mathcal{P}_1}{t} = \frac{1}{t\mathcal{P}_1}\int_0^t\left[\left(\beta_1\mathcal{S}(t) - (\eta+\rho) - \frac{\rho_2^2}{2}\right)\left(\eta - (\xi+\rho) - \frac{\rho_4^2}{2}\right)\right]dt + \frac{\mathcal{F}(t)}{t}$$

$$\leq \left(\beta_1\frac{\Theta}{\rho} - (\eta+\rho) - \frac{\rho_2^2}{2}\right)\left(\eta - (\xi+\rho) - \frac{\rho_4^2}{2}\right) + \frac{\mathcal{F}(t)}{t}$$

$$\leq \left(\eta+\rho+\frac{\rho_2^2}{2}\right)\left(\xi+\rho+\frac{\rho_4^2}{2}\right)(\mathcal{R}_1^* - 1) + \frac{\mathcal{F}(t)}{t}.$$

Where $\mathcal{F}(t) = \mathcal{M}_1(t) + \ln\mathcal{I}_1(t) + \ln\mathcal{E}_1(t)$ with $\mathcal{M}_1(t) = \int_0^t \rho_4^2\mathcal{I}_1(t)d\mathcal{B}_4(t) + \int_0^t \rho_2^2\mathcal{E}_1(t)d\mathcal{B}_2(t)$. The local continuous martingale. It can be observed that

$$\limsup_{t\to+\infty} \frac{<\mathcal{M}_1, \mathcal{M}_1>_t}{t} \leq \rho_2^2 + \rho_4^2\left(\frac{\Theta}{2}\right)^2 < +\infty$$

Then based on the strong law of large numbers shown in Lemma 3. It implies

$$\lim_{t\to+\infty}\frac{\mathcal{M}_1(t)}{t}=0.$$

Then one can obtained

$$\frac{\ln\mathcal{P}_1(t)}{t}\leq\frac{\beta_1\Theta}{\rho}-(\eta+\rho)(\xi+\rho)-\frac{\rho_2^2}{2}-\frac{\rho_4^2}{2}+\frac{\mathcal{F}(t)}{t}$$

$$\leq\left(\eta+\rho+\frac{\rho_2^2}{2}\right)\left(\xi+\rho+\frac{\rho_4^2}{2}\right)(\widehat{\mathcal{R}}_1^*-1)+\frac{\mathcal{F}(t)}{t}.$$

Provided that $\widehat{\mathcal{R}}_1^*<1$, taking the superior limit of both side leads to

$$\limsup_{t\to+\infty}\frac{\ln\mathcal{P}_1(t)}{t}\leq\left(\eta+\rho+\frac{\rho_2^2}{2}\right)\left(\xi+\rho+\frac{\rho_4^2}{2}\right)(\widehat{\mathcal{R}}_1^*-1)<0.$$

which implies,

$$\lim_{t\to\infty}\mathcal{P}_1(t)=0\quad\lim_{t\to\infty}\mathcal{E}_1(t)=0\quad\&\quad\lim_{t\to\infty}\mathcal{I}_1(t)=0$$

(2) Another form of equivalent $\mathcal{C}^2$-function $\mathcal{P}_2(t)$ is contracted by

$$\mathcal{P}_2(t)=\mathcal{E}_2\rho_3+\mathcal{I}_2\rho_5.$$

Using Ito's formula to $\mathcal{P}_2(t)$ gives,

$$d(\ln\mathcal{P}_2)=\mathcal{L}(\ln\mathcal{P}_2)dt=\frac{1}{\mathcal{P}_2}\left\{\left[\frac{\beta_2\mathcal{S}(t)\mathcal{I}_2(t)}{1+h_2\mathcal{I}_2(t)}-(\delta+\rho)\mathcal{E}_2(t)\right]dt+\rho_3\mathcal{E}_2(t)d\mathcal{B}_3(t)\right.$$

$$\left.[\delta\mathcal{E}_2(t)-(\gamma+\rho)\mathcal{I}_2(t)]\,dt+\rho_5\mathcal{I}_2(t)d\mathcal{B}_5(t)\right\}$$

$$d(\ln\mathcal{P}_2)=\frac{1}{\mathcal{P}_2}\left[\left(\beta_2\mathcal{S}(t)-(\delta+\rho)-\frac{\rho_3^2}{2}\right)+(\rho_3\mathcal{E}_2(t)d\mathcal{B}_3(t))\right.$$

$$\left.\left(\delta-(\gamma+\rho)-\frac{\rho_5^2}{2}\right)+\rho_5\mathcal{I}_2(t)d\mathcal{B}_5(t)\right].$$

Similarly, integrating from 0 to t and then dividing by t on both sides given by,

$$\frac{\ln \mathcal{P}_2}{t} = \frac{1}{t\mathcal{P}_2}\int_0^t \left[\left(\beta_2 \mathcal{S}(t) - (\delta+\rho) - \frac{\rho_3^2}{2}\right)\left(\delta - (\gamma+\rho) - \frac{\rho_5^2}{2}\right)\right] dt + \frac{\mathcal{G}(t)}{t}$$

$$\leq \left(\beta_2 \frac{\Theta}{\rho} - (\delta+\rho) - \frac{\rho_3^2}{2}\right)\left(\delta - (\gamma+\rho) - \frac{\rho_5^2}{2}\right) + \frac{\mathcal{G}(t)}{t}$$

$$\leq \left(\delta+\rho+\frac{\rho_3^2}{2}\right)\left(\gamma+\rho+\frac{\rho_5^2}{2}\right)(\mathcal{R}_2^* - 1) + \frac{\mathcal{G}(t)}{t}.$$

Where $\mathcal{G}(t) = \mathcal{M}_2(t) + \ln \mathcal{E}_2(t) + \ln \mathcal{I}_2(t)$. Here $\mathcal{M}_2(t) = \int_0^t (\rho_3 \mathcal{E}_2 + \rho_5 \mathcal{I}_2)(t) d\mathcal{B}_3(t) d\mathcal{B}_5(t)$ is the local martingale. Similarly,

$$\lim_{t\to+\infty} \frac{\mathcal{M}_2(t)}{t} = 0.$$

Since,

$$\limsup_{t\to+\infty} \frac{< \mathcal{M}_2, \mathcal{M}_2 >_t}{t} < \rho_3^2 \rho_5^2 \left(\frac{\Theta}{\rho}\right)^2 < +\infty.$$

Let $\varepsilon \to 0$ and then take the superior limit of both side, it leads to

$$\limsup_{t\to+\infty} \frac{\ln(\mathcal{P}_2(t))}{t} \leq \left(\delta+\rho+\frac{\rho_3^2}{2}\right)\left(\gamma+\rho+\frac{\rho_5^2}{2}\right)(\widehat{\mathcal{R}}_2^* - 1) < 0.$$

which implies that,

$$\lim_{t\to+\infty} \mathcal{P}_2(t) = 0, \quad (ie), \quad \lim_{t\to+\infty} \mathcal{E}_2(t) = 0 \quad \& \quad \lim_{t\to+\infty} \mathcal{I}_2(t) = 0$$

Together the condition (1) & (2) it denotes,

$$\lim_{t\to+\infty} \mathcal{S}(t) = \frac{\Theta}{\rho} \quad \& \quad \lim_{t\to+\infty} \mathcal{R}(t) = 0. \quad a.s.$$

Therefore the proof is completed. □

Remark 3 Theorem 2 illustrate that if $\widehat{\mathcal{R}}_1^* < 1$, & $\widehat{\mathcal{R}}_2^* < 1$. The disease will go to extinct. Note that when $\widehat{\mathcal{R}}_0^* < 1$, we notice that the larger the intensity of white noise are the easier the extinction of the disease. Thus we can control the outbreak of the disease by adjusting the intensity of environment noises.

6. Numerical simulations

Thorough investigations have been conducted into both the extinction and persistence dynamics of diseases. To substantiate the effectiveness of the simulation regarding drug resistance and drug sensitivity, numerical simulations will be performed using the Milstein scheme [44, 45]. Below is the discretization equation for model (3):

$$\mathcal{S}^{l+1} = \mathcal{S}^l + \left[\Theta - \frac{\beta_1 \mathcal{S}^l \mathcal{I}_1^l}{1+h_1\mathcal{I}_1^l} - \frac{\beta_2 \mathcal{S}^l \mathcal{I}_2^l}{1+h_2\mathcal{I}_2^l} - \rho \mathcal{S}^l\right]\Delta t,$$

$$+\rho_1 \mathcal{S}^l \sqrt{\Delta t \xi_{1,l}} + \frac{\rho_1^2}{2}\mathcal{S}^l(\xi_{1,l}^2 - 1)\Delta t,$$

$$\mathcal{E}_1^{l+1} = \mathcal{E}_1^l + \left[\frac{\beta_1 \mathcal{S}^l \mathcal{I}_1^l}{1+h_1\mathcal{I}_1^l} - (\eta+\rho)\mathcal{E}_1^l\right]\Delta t,$$

$$+\rho_2 \mathcal{E}_1^l \sqrt{\Delta t \xi_{2,l}} + \frac{\rho_2^2}{2}\mathcal{E}_1^l(\xi_{2,l}^2 - 1)\Delta t,$$

$$\mathcal{E}_2^{l+1} = \mathcal{E}_2^l + \left[\frac{\beta_2 \mathcal{S}^l \mathcal{I}_2^l}{1+h_2\mathcal{I}_2^l} - (\delta+\rho)\mathcal{E}_2^l\right]\Delta t,$$

$$+\rho_3 \mathcal{E}_2^l \sqrt{\Delta t \xi_{3,l}} + \frac{\rho_3^2}{2}\mathcal{E}_2^l(\xi_{3,l}^2 - 1)\Delta t,$$

$$\mathcal{I}_1^{l+1} = \mathcal{I}_1^l + \left[\eta \mathcal{E}_1^l - (\xi+\rho)\mathcal{I}_1^l\right]\Delta t,$$

$$+\rho_4 \mathcal{I}_1^l \sqrt{\Delta t \xi_{4,l}} + \frac{\rho_4^2}{2}\mathcal{I}_1^l(\xi_{4,l}^2 - 1)\Delta t,$$

$$\mathcal{I}_2^{l+1} = \mathcal{I}_2^l + \left[\delta \mathcal{E}_2^l - (\xi+\rho)\mathcal{I}_2^l\right]\Delta t,$$

$$+\rho_5 \mathcal{I}_2^l \sqrt{\Delta t \xi_{5,l}} + \frac{\rho_5^2}{2}\mathcal{I}_2^l(\xi_{5,l}^2 - 1)\Delta t,$$

$$\mathcal{R}^{l+1} = \mathcal{R}^l + \left[\xi I_1^l + \gamma I_2^l \rho \mathcal{R}^l\right]\Delta t,$$

$$+\rho_6 \mathcal{R}^l \sqrt{\Delta t \xi_{6,l}} + \frac{\rho_6^2}{2}\mathcal{R}^l(\xi_{6,l}^2 - 1)\Delta t,$$

where the time increment $\Delta t > 0$, and ξ_i^2 is a the Gaussian random variable ($i = 0, 1, 2, ..., n$). In Table 3, we present the parameter values that validate our theoretical finding by providing examples.

Table 3. The parameters used in the simulation of model (3)

Parameters	$\mathbb{E}_1$	$\mathbb{E}_2$	Source
Θ	4.00	3.00	presumed
β_1	0.50	0.06	presumed
β_2	0.60	0.09	presumed
h_1	0.05	0.0.5	presumed
h_2	0.07	0.07	presumed
η	0.60	0.70	presumed
δ	0.50	0.70	presumed
ξ	0.20	0.50	presumed
γ	0.5	0.5	presumed
ρ	0.30	0.30	presumed
$\mathcal{S}(0)$	0.40	0.40	presumed
$\mathcal{E}_1(0)$	0.30	0.30	presumed
$\mathcal{E}_2(0)$	0.30	0.30	presumed
$\mathcal{I}_1(0)$	0.4	0.4	presumed
$\mathcal{I}_2(0)$	0.20	0.40	presumed
$\mathcal{R}(0)$	0.50	0.50	presumed
Δt	0.01	0.01	presumed

Example 1 Assume that the parameters for environmental white noise are as follows:

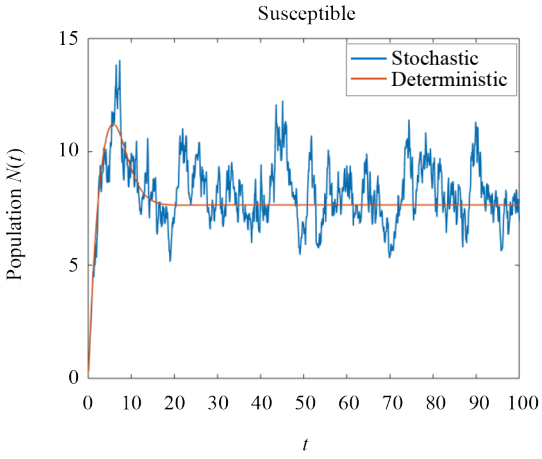
$\rho_i = 0.2, \forall\, i = 1$ to 6 [6]. Furthermore, Table 3 ($\mathbb{E}_1$) shows the parameter values in relation to the biological feasibility results. Then

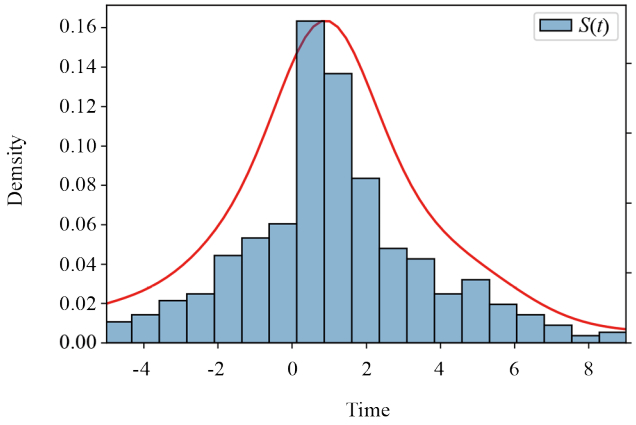
$$\mathcal{R}_0^* = \max\left\{\mathcal{R}_1^* = \frac{\Theta\beta_1\eta}{\left(\rho+\frac{\rho_1^2}{2}\right)\left(\eta+\rho+\frac{\rho_2^2}{2}\right)\left(\xi+\rho+\frac{\rho_4^2}{2}\right)},\right.$$

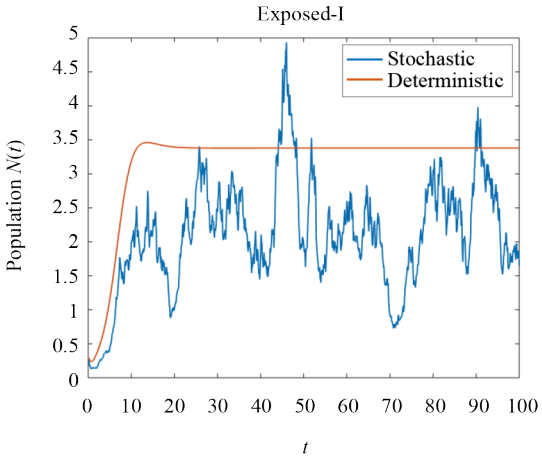
$$\left.\mathcal{R}_2^* = \frac{\Theta\beta_2\eta}{\left(\rho+\frac{\rho_1^2}{2}\right)\left(\delta+\rho+\frac{\rho_3^2}{2}\right)\left(\gamma+\rho+\frac{\rho_5^2}{2}\right)}\right\}$$

$$= \max\{\mathcal{R}_1^* = 2.3803, \quad \mathcal{R}_2^* = 1.8480\} > 1,$$

If the parameter condition specified in Theorem 1 holds true, it signifies that the stochastic model (3) exhibits ergodic characteristics and possesses a unique stationary distribution $k(\cdot)$. Figures 2 and 3 depict the solutions of model (3) displaying both upward and downward trends within a narrow vicinity. Notably, Figure 2 visually confirms the existence of a stationary distribution through the density functions presented on the right-hand side.


Susceptible
Stochastic
Deterministic
Population $N(t)$
t


$S(t)$
Demsity
Time


Exposed-I
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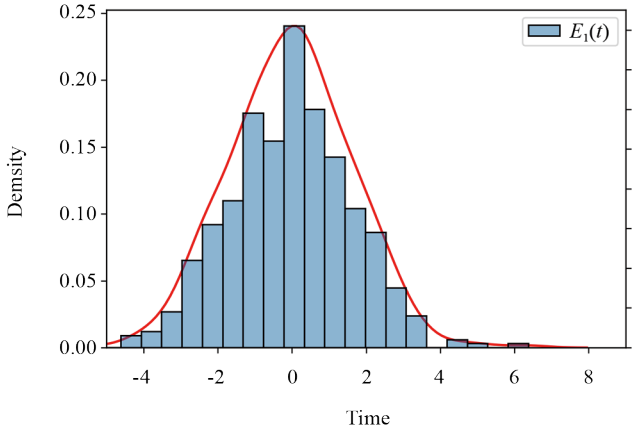
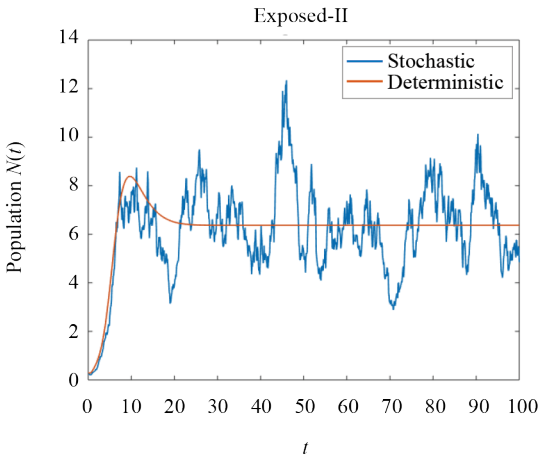
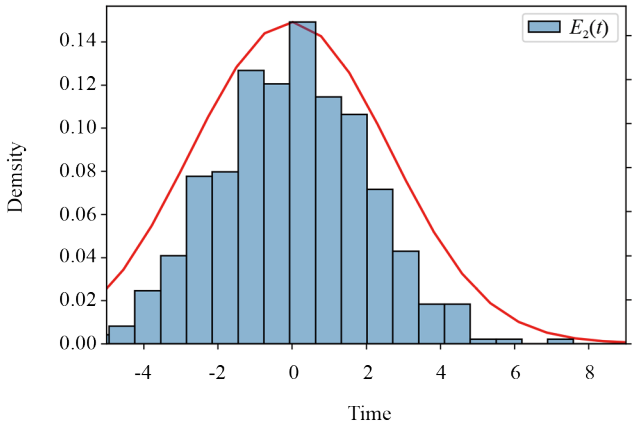
Population $N(t)$
t


$E_1(t)$
Demsity
Time


Exposed-II
Stochastic
Deterministic
Population $N(t)$
t


$E_2(t)$
Demsity
Time

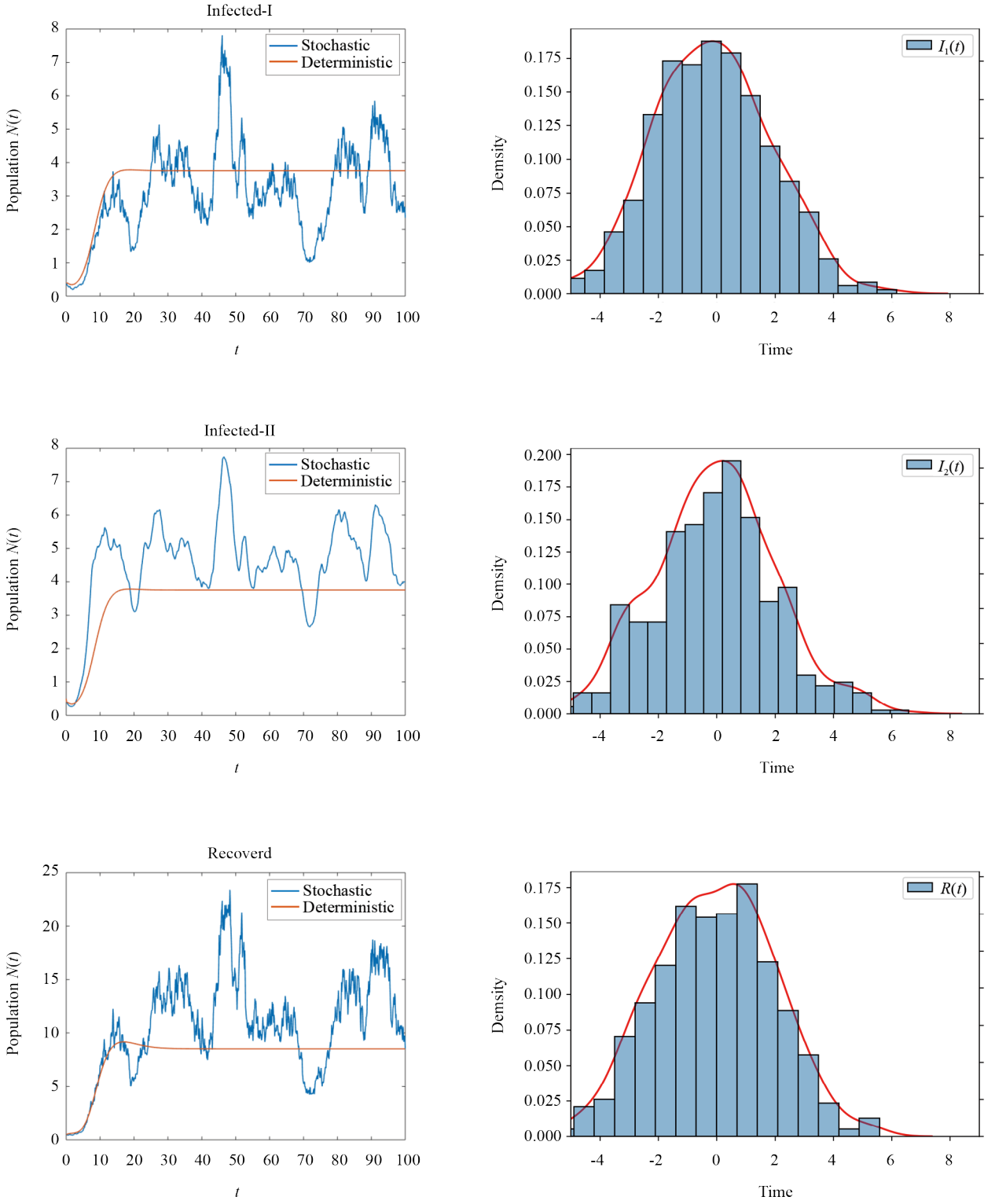


Figure 2. This diagram consists of a time sequence of stochastic persistence and stationary distribution of diseases based on the model (3) for both $\mathcal{R}_0^* > 1$ in (4) & $\widehat{\mathcal{R}}_0^* > 1$ in (26). On the right side of the column, the probability density function for $\mathcal{S}(t)$, $\mathcal{E}_1(t)$, $\mathcal{E}_2(t)$, $\mathcal{I}_1(t)$, $\mathcal{I}_2(t)$, and $\mathcal{R}(t)$ is shown in the form of a histogram

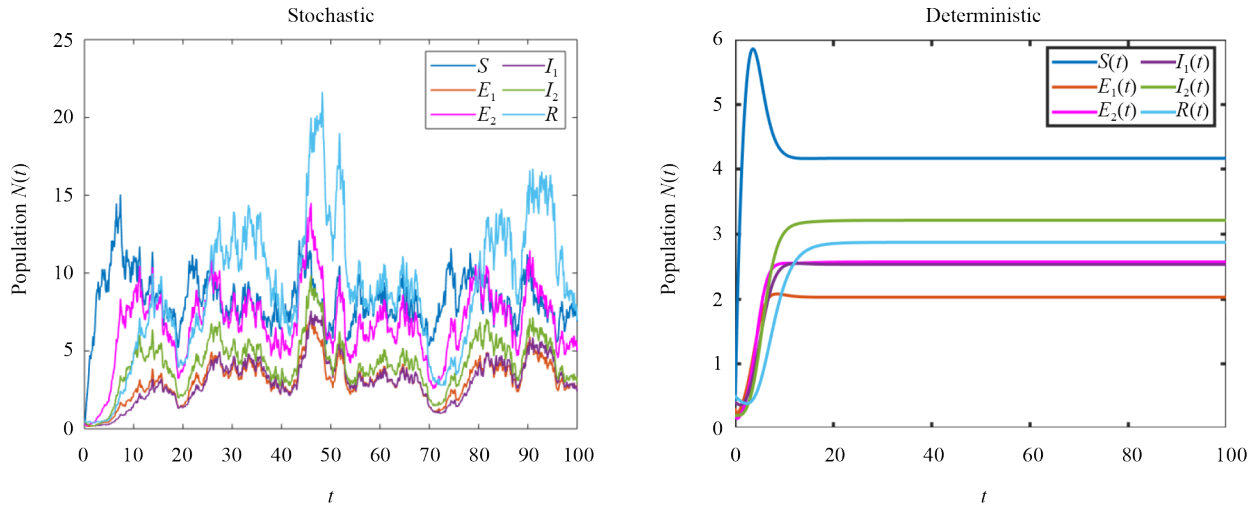


Figure 3. Comparison of solutions on $\mathcal{S}(t)$, $\mathcal{E}_1(t)$, $\mathcal{E}_2(t)$, $\mathcal{I}_1(t)$, $\mathcal{I}_2(t)$, and $\mathcal{R}(t)$: for each class of the Deterministic and Stochastic system with $\mathcal{R}_0^*$ greater than 1 in (4)

Example 2 Assume that the parameters for environmental white noise are as follows:

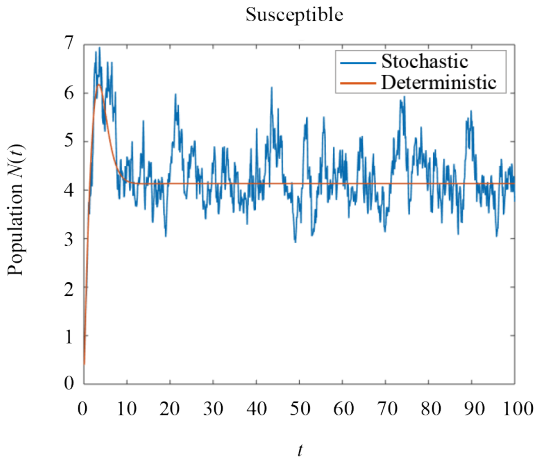
$\rho_i = 0.2, \forall\, i = 1$ to 6 [6]. Furthermore, Table 3 ($\mathbb{E}_2$) shows the parameter values in relation to the biological feasibility results. Then

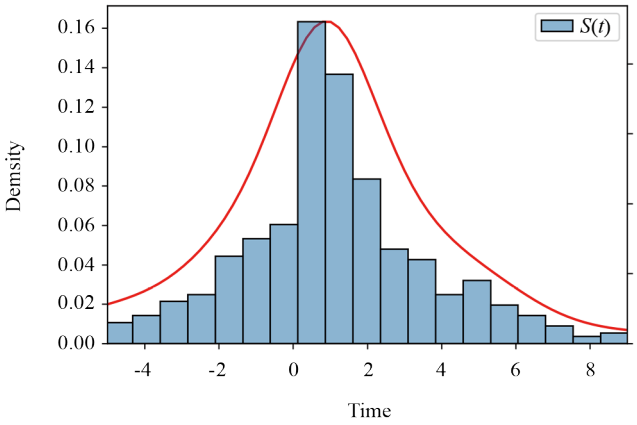
$$\widehat{\mathcal{R}}_0^* = \max\left\{\widehat{\mathcal{R}}_1^* = \mathcal{R}_1 - \frac{\Theta\beta_1\eta\rho_2^2\rho_4^2}{2\rho(\eta+\rho)(\xi+\rho)\left(\eta+\rho+\frac{\rho_2^2}{2}\right)\left(\xi+\rho+\frac{\rho_4^2}{2}\right)}\right.$$

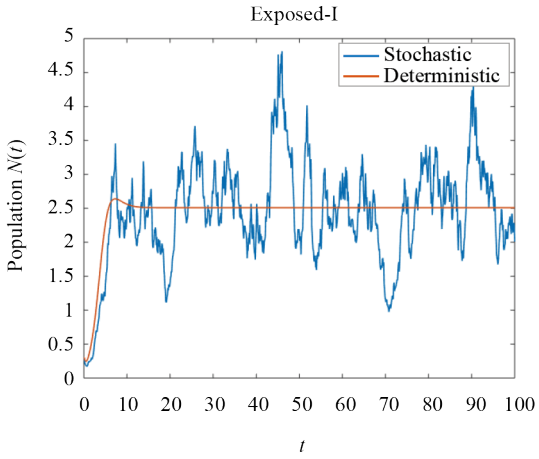
$$\left.\widehat{\mathcal{R}}_2^* = \mathcal{R}_2 - \frac{\Theta\beta_2\delta\rho_3^2\rho_5^2}{2\rho(\delta+\rho)(\gamma+\rho)\left(\delta+\rho+\frac{\rho_3^2}{2}\right)\left(\gamma+\rho+\frac{\rho_5^2}{2}\right)}\right\}.$$

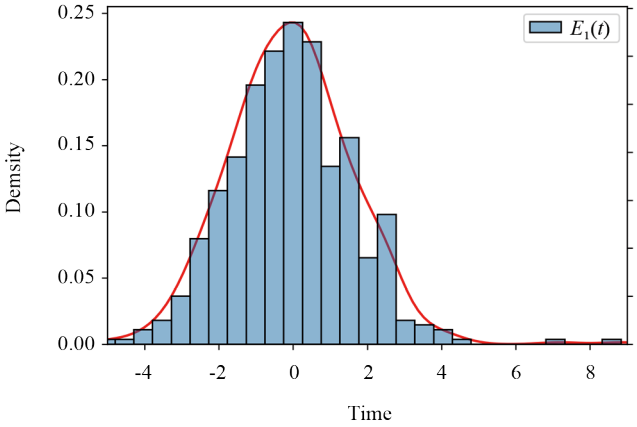
$$\widehat{\mathcal{R}}_0^* = \max\left(\widehat{\mathcal{R}}_1^* = 0.9226, \widehat{\mathcal{R}}_2^* = 0.9600\right) < 1.$$

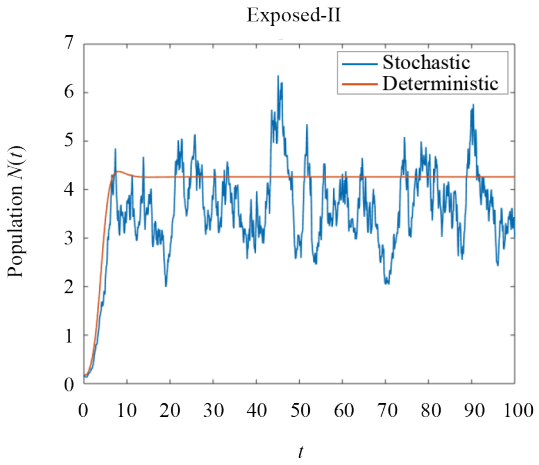
According to Theorem 2, if all parameter conditions are satisfied, exposed and infected individuals will almost certainly go extinct. This conclusion is supported by Figure 4. Numerical simulations with specific values such as $\rho_i = 0.2, \forall\, i = 1$ to 6, depicted in Figures 4 and 5, indicate a high likelihood of infectious individuals becoming extinct under large stochastic parameter values. Figures 4 and 5 below demonstrate that when white noise values are large, infectious diseases tend to go extinct. This suggests that stochastic fluctuations can effectively suppress disease outbreaks, whereas smaller noise values may result in persistent infectious diseases. Furthermore, Figures 2 and 3 illustrate that under suitable conditions, the stochastic model (3) exhibits an ergodic stationary distribution. Thus, there is consistent alignment between the theoretical findings of Theorems 1 and 2 and the outcomes observed in numerical simulations.


Susceptible
Stochastic
Deterministic
Population $N(t)$
t


$S(t)$
Demsity
Time


Exposed-I
Stochastic
Deterministic
Population $N(t)$
t


$E_1(t)$
Demsity
Time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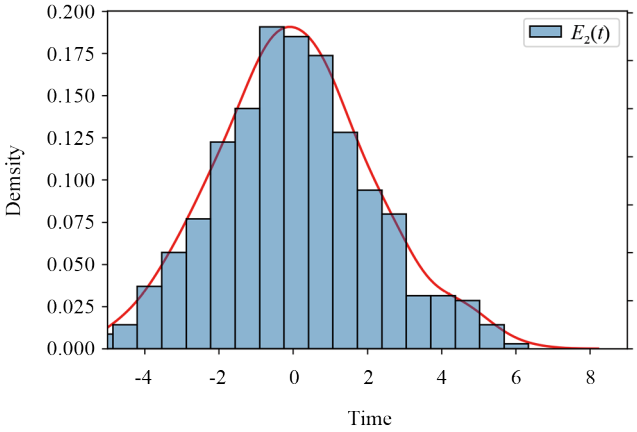
$E_2(t)$
Demsity
Time

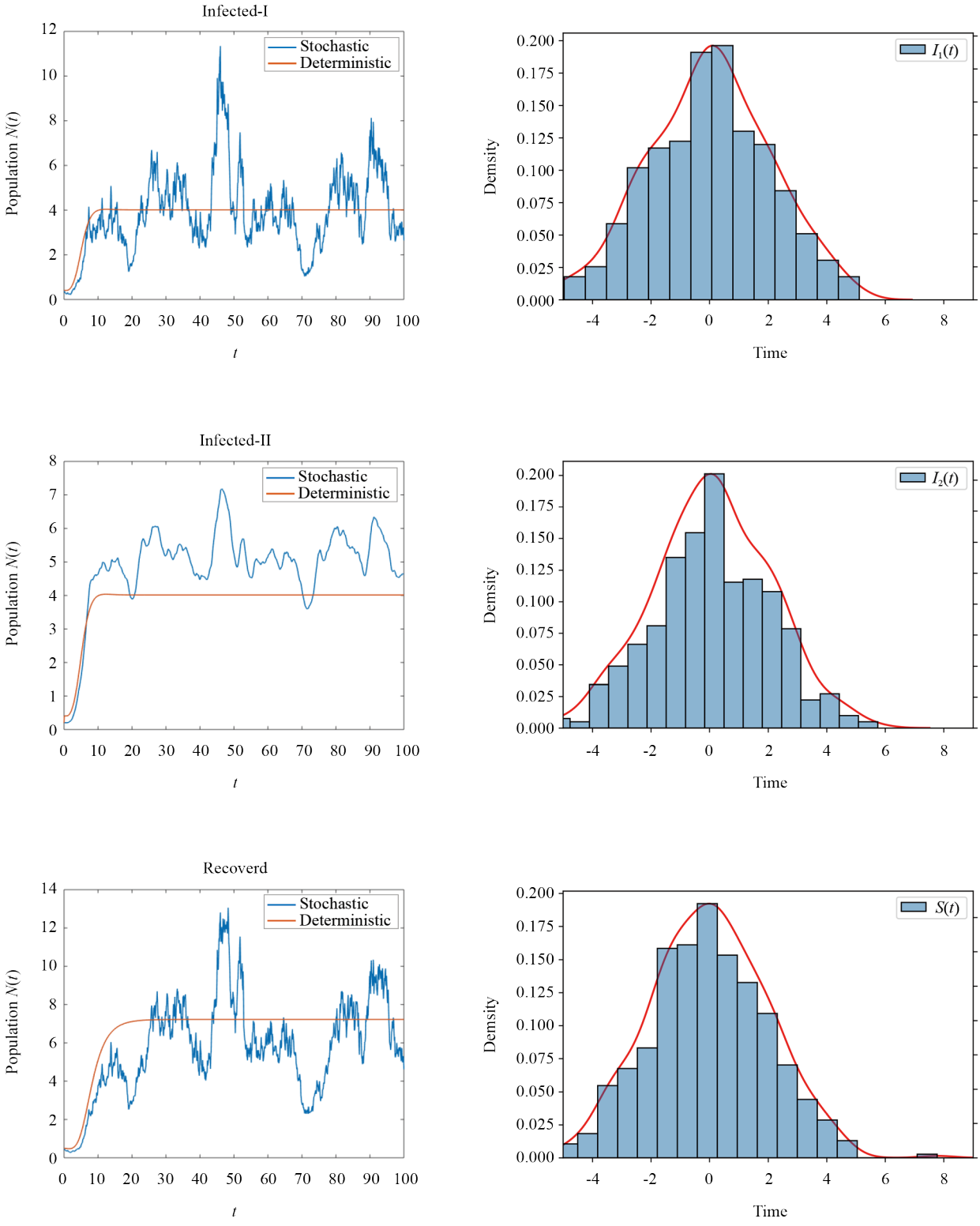

Figure 4. This time sequence diagram illustrates how disease extinction occurs in model (3) for both $\mathcal{R}_0^* < 1$ in (4) & $\widehat{\mathcal{R}}_0^* < 1$ in (26). On the right side of the column, the probability density function for $\mathcal{S}(t)$, $\mathcal{E}_1(t)$, $\mathcal{E}_2(t)$, $\mathcal{I}_1(t)$, $\mathcal{I}_2(t)$, and $\mathcal{R}(t)$ is shown in the form of a histogram

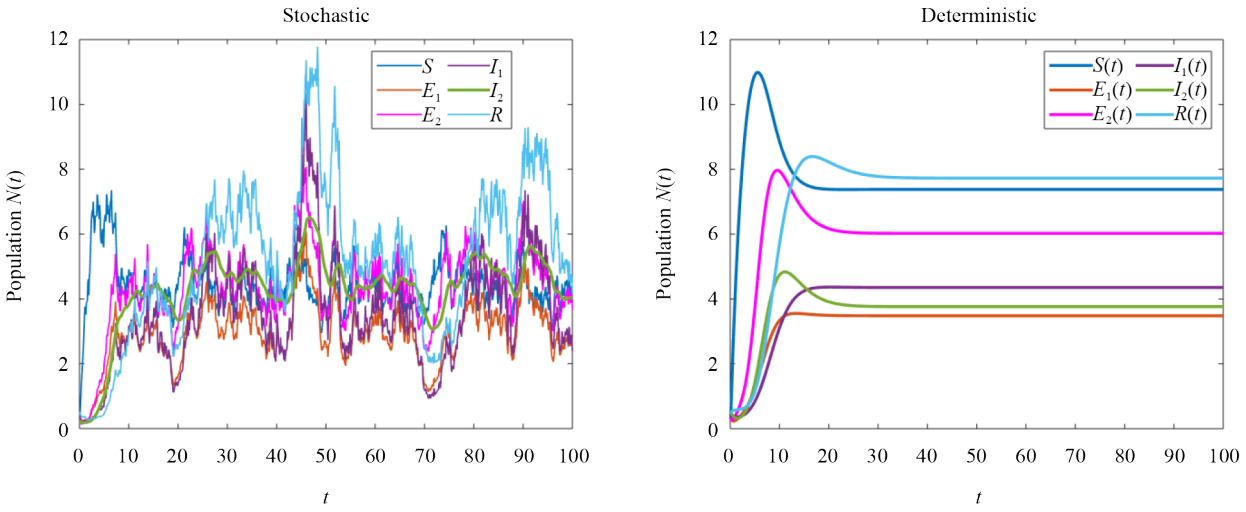

Figure 5. Comparison of solutions on $\mathcal{S}(t)$, $\mathcal{E}_1(t)$, $\mathcal{E}_2(t)$, $\mathcal{I}_1(t)$, $\mathcal{I}_2(t)$, and $\mathcal{R}(t)$: for each class of the Deterministic and Stochastic system with $\widehat{\mathcal{R}}_0^*$ less than 1 in (26)

7. Conclusion

In this paper, we explored a dual-strain stochastic SEIR epidemic model using two general incidence functions, structured as SEIR with six compartments: susceptible, two stages of exposed, two stages of infected, and removed individuals. Our focus extended to investigating a stochastic drug-resistant model encompassing both drug-sensitive and drug-resistant states. We derived the basic reproduction number $\mathcal{R}_0$ in (2) relative to the first and second strains and incorporated post-treatment mutation from drug-sensitive to drug-resistant states of the deterministic model (1). By establishing thresholds $\mathcal{R}_0^* > 1$ in (4) and $\widehat{\mathcal{R}}_0^* < 1$ in (26) for drug-sensitive and drug-resistant groups, we formulated conditions for extinction and persistence through stochastic Lyapunov functions. Additionally, we determined the ergodic stationary distribution $k(\cdot)$. Our analysis also led to the formulation of an ergodic stationary distribution, providing insights into the equilibrium state of the disease over time. We found that increased mutation rates driven by higher amplification rates facilitated the transition of drug-sensitive individuals to the drug-resistant state, eventually leading to an equilibrium between the two strains. This suggests that drug resistance, under certain conditions, may become a dominant feature of the epidemic. This study provides critical insights into the complex interactions between drug-sensitive and drug-resistant strains within a stochastic context, emphasizing the importance of considering random fluctuations in the dynamics of disease spread. Given the limited research on drug resistance in both human and animal populations, this model offers a valuable framework for understanding how drug-resistant strains evolve and persist in real-world settings. The insights generated from this work could inform public health strategies aimed at controlling the spread of multi-strain diseases, especially in contexts where drug resistance poses significant challenges. Future studies should delve into systems featuring delayed mutation post-treatment, exploring their dynamics and thresholds for disease persistence or extinction. These considerations open avenues for further research into how different noise structures impact epidemic outcomes.

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Conflict of interest

The author declare that they have no conflict of interest regarding the publication of this manuscript.

References

[1] Kermack WO, McKendrick AG. A contribution to the mathematical theory of epidemics. *Proceedings of the Royal Society of London A*. 1927; 115(772): 700-721. Available from: https://doi.org/10.1098/rspa.1927.0118.

[2] Omame A, Abbas M, Din A. Global asymptotic stability, extinction and ergodic stationary distribution in a stochastic model for dual variants of SARS-CoV-2. *Mathematics and Computers in Simulation*. 2023; 204: 302-336. Available from: https://doi.org/10.1016/j.matcom.2022.08.012.

[3] Dolbeault J, Turinici G. Heterogeneous social interactions and the COVID-19 lockdown outcome in a multi-group SEIR model. *Mathematical Modelling of Natural Phenomena*. 2020; 15: 36. Available from: https://doi.org/10.1051/mmnp/2020025.

[4] Din A, Li Y, Omame A. A stochastic stability analysis of an HBV-COVID-19 co-infection model in resource limitation settings. *Waves in Random and Complex Media*. 2022; 2022: 1-33. Available from: https://doi.org/10.1080/17455030.2022.2147598.

[5] Guseva V, Doktorova N, Krivorotko O, Otpushchennikova O, Parolina L, Vasilyeva I, et al. Building a seir-model for predicting the HIV/tuberculosis coinfection epidemic for russian territories with low TB burden. *International Journal of Infectious Diseases*. 2023; 134: S4-S5. Available from: https://doi.org/10.1016/j.ijid.2023.05.028.

[6] Liu Y. Extinction, persistence and density function analysis of a stochastic two-strain disease model with drug resistance mutation. *Applied Mathematics and Computation*. 2022; 433: 127393. Available from: https://doi.org/10.1016/j.amc.2022.127393.

[7] Omame A, Abbas M, Baleanu D. A stochastic model to assess the epidemiological impact of vaccine booster doses on COVID-19 and viral hepatitis *b* co-dynamics with real data. *CMES-Computer Modeling in Engineering & Sciences*. 2023; 138: 2973-3012. Available from: https://doi.org/10.32604/cmes.2023.029681.

[8] Taye MA. Host viral load during triple coinfection of SARS-CoV-2, influenza virus, and syncytial virus. *Contemporary Mathematics*. 2023; 4(3): 392-410. Available from: https://doi.org/10.37256/cm.4320232500.

[9] Moya ED, Pietrus A, Oliva SM. A mathematical model for the study of effectiveness in therapy in Tuberculosis taking into account associated diseases. *Contemporary Mathematics*. 2021; 2(1): 77-102. Available from: https://doi.org/10.37256/cm.212021694.

[10] Zhao X, Dong L. Dynamical behaviors of a stochastic HIV/AIDS epidemic model with treatment. *Mathematical Methods in the Applied Sciences*. 2024; 47(5): 3690-3704. Available from: https://doi.org/10.1002/mma.9188.

[11] Khyar O, Allali K. Global dynamics of a multi-strain SEIR epidemic model with general incidence rates: application to COVID-19 pandemic. *Nonlinear Dynamics*. 2020; 102(1): 489-509. Available from: https://doi.org/10.1007/s11071-020-05929-4.

[12] Otunuga OM. Analysis of multi-strain infection of vaccinated and recovered population through epidemic model: Application to COVID-19. *Plos One*. 2022; 17(7): e0271446. Available from: https://doi.org/10.1371/journal.pone.0271446.

[13] Saha P, Mondal B, Ghosh U. Global dynamics and optimal control of a two-strain epidemic model with non-monotone incidence and saturated treatment. *Iranian Journal of Science*. 2023; 47(5): 1575-1591. Available from: https://doi.org/10.1007/s40995-023-01511-w.

[14] Lee HS, Thakur KK, Bui VN, Pham TL, Bui AN, Dao TD, et al. A stochastic simulation model of African swine fever transmission in domestic pig farms in the Red River Delta region in Vietnam. *Transboundary and Emerging Diseases*. 2021; 68(3): 1384-1391. Available from: https://doi.org/10.1111/tbed.13802.

[15] Vidovic N, Vidovic S. Antimicrobial resistance and food animals: Influence of livestock environment on the emergence and dissemination of antimicrobial resistance. *Antibiotics*. 2020; 9(2): 52. Available from: https://doi.org/10.3390/antibiotics9020052.

[16] Olabode D, Rong L, Wang X. Stochastic investigation of HIV infection and the emergence of drug resistance. *Mathematical Biosciences and Engineering*. 2021; 19(2): 1174-1194. Available from: https://doi.org/10.3934/mbe.2022054.

[17] Hajri Y, Assiri TA, Amine S, Ahmad S, De la Sen M, et al. A stochastic co-infection model for HIV-1 and HIV-2 epidemic incorporating drug resistance and dual saturated incidence rates. *Alexandria Engineering Journal*. 2023; 84: 24-36. Available from: https://doi.org/10.1016/j.aej.2023.10.053.

[18] Wang Y, Cao J, Xue C, Li L. Mathematical analysis of epidemic models with treatment in heterogeneous networks. *Bulletin of Mathematical Biology*. 2023; 85(2): 11. Available from: https://doi.org/10.1007/s11538-022-01116-1.

[19] Bentaleb D, Harroudi S, Amine S, Allali K. Analysis and optimal control of a multistrain SEIR epidemic model with saturated incidence rate and treatment. *Differential Equations and Dynamical Systems*. 2023; 31(4): 907-923. Available from: https://doi.org/10.1007/s12591-020-00544-6.

[20] Saha P, Mondal B, Ghosh U. Global dynamics and optimal control of a two-strain epidemic model with non-monotone incidence and saturated treatment. *Iranian Journal of Science*. 2023; 47(5): 1575-1591. Available from: https://doi.org/10.1007/s40995-023-01511-w.

[21] Pečerska J, Kühnert D, Meehan CJ, Coscollá M, De Jong BC, Gagneux S, et al. Quantifying transmission fitness costs of multi-drug resistant tuberculosis. *Epidemics*. 2021; 36: 100471. Available from: https://doi.org/10.1016/j.epidem.2021.100471.

[22] Karmakar M, Ragonnet R, Ascher DB, Trauer JM, Denholm JT. Estimating tuberculosis drug resistance amplification rates in high-burden settings. *BMC Infectious Diseases*. 2022; 22(1): 82. Available from: https://doi.org/10.1186/s12879-022-07067-1.

[23] Friedman A, Ziyadi N, Boushaba K. A model of drug resistance with infection by health care workers. *Mathematical Biosciences & Engineering*. 2010; 7(4): 779-792. Available from: https://doi.org/10.3934/mbe.2010.7.779.

[24] Kitaro DB, Bole BK, Rao KP. Modeling and bifurcation analysis of tuberculosis with the multidrug-resistant compartment incorporating chemoprophylaxis treatment. *Frontiers in Applied Mathematics and Statistics*. 2023; 9: 1264201. Available from: https://doi.org/10.3389/fams.2023.1264201.

[25] Abatih EN, Alban L, Ersbøll AK, Wong DMLF. Impact of antimicrobial usage on the transmission dynamics of antimicrobial resistant bacteria among pigs. *Journal of Theoretical Biology*. 2009; 256(4): 561-573. Available from: https://doi.org/10.1016/j.jtbi.2008.10.017.

[26] Yang J, Yang L, Jin Z. Optimal strategies of the age-specific vaccination and antiviral treatment against influenza. *Chaos, Solitons & Fractals*. 2023; 168: 113199. Available from: https://doi-org/10.1016/j.chaos.2023.113199.

[27] Liu Q, Jiang D. Stationary distribution of a stochastic SIS epidemic model with double diseases and the Beddington-DeAngelis incidence. *Chaos: An Interdisciplinary Journal of Nonlinear Science*. 2017; 27(8): 083126. Available from: https://doi.org/10.1063/1.4986838.

[28] Rajasekar SP, Pitchaimani M. Ergodic stationary distribution and extinction of a stochastic SIRS epidemic model with logistic growth and nonlinear incidence. *Applied Mathematics and Computation*. 2020; 377: 125143. Available from: https://doi-org/10.1016/j.amc.2020.125143.

[29] Zhang XB, Zheng L. Complex dynamics of a stochastic SIR epidemic model with vertical transmission and varying total population size. *Journal of Nonlinear Science*. 2023; 33(6): 108. Available from: https://doi.org/10.1007/s00332-023-09960-8.

[30] Zhao Y, Jiang D. The threshold of a stochastic SIS epidemic model with vaccination. *Applied Mathematics and Computation*. 2014; 243: 718-727. Available from: https://doi.org/10.1016/j.amc.2014.05.124.

[31] Niu L, Chen Q, Teng Z. Threshold dynamics and probability density function of a stochastic multi-strain coinfection model with amplification and vaccination. *Qualitative Theory of Dynamical Systems*. 2024; 23(2): 94. Available from: https://doi.org/10.1007/s12346-024-00957-6.

[32] Khajanchi S, Bera S, Roy TK. Mathematical analysis of the global dynamics of a HTLV-I infection model, considering the role of cytotoxic T-lymphocytes. *Mathematics and Computers in Simulation*. 2021; 180: 354-378. Available from: https://doi.org/10.1016/j.matcom.2020.09.009.

[33] Bera S, Khajanchi S, Roy TK. Stability analysis of fuzzy HTLV-I infection model: a dynamic approach. *Journal of Applied Mathematics and Computing*. 2023; 69(1): 171-199. Available from: https://doi.org/10.1007/s12190-022-01741-y.

[34] Liu Q, Jiang D, Hayat T, Ahmad B. Analysis of a delayed vaccinated SIR epidemic model with temporary immunity and Lévy jumps. *Nonlinear Analysis: Hybrid Systems*. 2018; 27: 29-43. Available from: https://doi-org/10.1016/j.nahs.2017.08.002.

[35] Zhou B, Han B, Jiang D. Ergodic property, extinction and density function of a stochastic SIR epidemic model with nonlinear incidence and general stochastic perturbations. *Chaos, Solitons & Fractals*. 2021; 152: 111338. Available from: https://doi-org/10.1016/j.chaos.2021.111338.

[36] Bernard S, Piétrus A, Valmont K. Asymptotic behavior of a stochastic e-rumor model with Lévy jump. *Contemporary Mathematics*. 2020; 1(2): 149-169. Available from: https://doi.org/10.37256/cm.122020177.

[37] Zhao Y, Jiang D. The threshold of a stochastic SIS epidemic model with vaccination. *Applied Mathematics and Computation*. 2014; 243: 718-727. Available from: https://doi.org/10.1016/j.amc.2014.05.124.

[38] Mao X. 3-linear stochastic differential equations. In: *Stochastic Differential Equations and Applications*. 2nd ed. Cambridge: Woodhead Publishing; 2011. p.91-106.

[39] Khasminskii R. *Stochastic Stability of Differential Equations*. Berlin, Heidelberg: Springer Science & Business Media; 2011. Available from: https://doi.org/10.1007/978-3-642-23280-0.

[40] Zhang G, Li Z, Din A. A stochastic SIQR epidemic model with Lévy jumps and three-time delays. *Applied Mathematics and Computation*. 2022; 431: 127329. Available from: https://doi.org/10.1016/j.amc.2022.127329.

[41] Zhang S, Yuan S, Zhang T. A predator-prey model with different response functions to juvenile and adult prey in deterministic and stochastic environments. *Applied Mathematics and Computation*. 2022; 413: 126598. Available from: https://doi.org/10.1016/j.amc.2021.126598.

[42] Pitchaimani M, Brasanna DM. Stochastic dynamical probes in a triple delayed SICR model with general incidence rate and immunization strategies. *Chaos, Solitons & Fractals*. 2021; 143: 110540. Available from: https://doi.org/10.1016/j.chaos.2020.110540.

[43] Samantaa G, Bera SPP. Analysis of a Chlamydia epidemic model with pulse vaccination strategy in a random environment. *Nonlinear Analysis: Modelling and Control*. 2018; 23(4): 457-474. Available from: https://doi.org/10.15388/NA.2018.4.1.

[44] Higham DJ. An algorithmic introduction to numerical simulation of stochastic differential equations. *SIAM Review*. 2001; 43(3): 525-546. Available from: https://doi.org/10.1137/S0036144500378302.

[45] Kloeden PE, Platen E, Kloeden PE, Platen E. Strong taylor approximations. In: *Numerical Solution of Stochastic Differential Equations*. Berlin, Heidelberg: Springer; 1992. p.339-371. Available from: https://doi.org/10.1007/978-3-662-12616-5_10.