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**Mhammed Mediani**

# Mathematical analysis of a class of stochastic epidemic models

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مقدمة لنيل شهادة

**دكتوراه في الرياضيات**

التخصص: العمليات العشوائية والإحصاء

من إعداد

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# التحليل الرياضياتي لفئة من النماذج الوبائية العشوائية

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# Abstract

Over the past few decades, epidemics caused by highly pathogenic viruses such as the coronavirus, measles virus, and human immunodeficiency virus (HIV) have posed severe threats to public health. From a microscopic perspective, stochasticity plays a crucial role in viral replication, yet theoretical investigations of epidemic models incorporating biologically plausible stochastic effects remain limited. This thesis aims to address this gap by analyzing the evolutionary dynamics of stochastic epidemic models and assessing the impact of stochastic perturbations on viral pathogenesis and disease progression.

In this work, we develop and study stochastic models that incorporate viral production mechanisms while integrating the Ornstein-Uhlenbeck or Black-Karasinski process. Specifically, we assume that key epidemiological parameters-such as the conversion rate from an eclipse phase to an infectious state—evolve according to the Ornstein-Uhlenbeck process. Within a well-defined mathematical framework, we establish and extend non-standard analytical approaches to demonstrate fundamental properties of these stochastic models, including the existence and uniqueness of global solutions, the presence of stationary distributions, and the conditions under which disease extinction occurs. Furthermore, we show that introducing stochastic perturbations in this manner is both mathematically rigorous and biologically justified.To support our theoretical findings, numerical simulations are conducted at the end of each section. These simulations validate the analytical results and illustrate the accuracy of the derived conditions and threshold parameters.

**Keywords:** Stochastic epidemic model, Measles Epidemic Model, Stochastic Dynamics, Stochastic HIV-1 model,Ornstein–Uhlenbeck process, Black-Karasinski Process, Stationary distribution, Probability density, Extinction.

# Résumé (French-version)

Au cours des dernières décennies, les épidémies causées par des virus hautement pathogènes tels que le coronavirus, le virus de la rougeole et le virus de l'immunodéficience humaine (VIH) ont représenté de graves menaces pour la santé publique. D'un point de vue microscopique, la stochasticité joue un rôle crucial dans la réplication virale, mais les études théoriques sur les modèles épidémiologiques intégrant des effets stochastiques biologiquement plausibles restent limitées. Cette thèse vise à combler cette lacune en analysant la dynamique évolutive des modèles épidémiologiques stochastiques et en évaluant l'impact des perturbations stochastiques sur la pathogenèse virale et la progression des maladies.

Dans ce travail, nous développons et étudions des modèles stochastiques intégrant des mécanismes de production virale et incorporant les processus d'Ornstein-Uhlenbeck ou de Black-Karasinski. Plus précisément, nous supposons que certains paramètres épidémiologiques clés, tels que le taux de conversion des cellules en phase d'éclipse en cellules infectieuses, évoluent selon le processus d'Ornstein-Uhlenbeck. Dans un cadre mathématique bien défini, nous établissons et étendons des approches analytiques non conventionnelles pour démontrer des propriétés fondamentales de ces modèles stochastiques, notamment l'existence et l'unicité des solutions globales, l'existence d'une distribution stationnaire et les conditions d'extinction de la maladie. De plus, nous montrons que l'introduction de perturbations stochastiques de cette manière est à la fois mathématiquement rigoureuse et biologiquement justifiée.

Afin de soutenir nos résultats théoriques, des simulations numériques sont réalisées à la fin de chaque section. Ces simulations valident les analyses développées et illustrent la précision des conditions et des seuils obtenus.

**Mots-clés :** Modèle épidémique stochastique, Modèle épidémique de la rougeole, Dynamique stochastique, Modèle stochastique du VIH-1, Équation de Fokker-Planck, Processus d'Ornstein–Uhlenbeck, Processus de Black-Karasinski, Distribution stationnaire, Densité de probabilité, Extinction.

# الملخص (Arabic-version)

على مدار العقود القليلة الماضية، شكلت الأوبئة التي تسببها الفيروسات شديدة العدوى، مثل فيروس كورونا، وفيروس الحصبة، وفيروس نقص المناعة البشرية ، (HIV) تهديدًا خطيرًا للصحة العامة. ومن منظور مجهري، تلعب العشوائية دورًا حاسمًا في عملية تكاثر الفيروسات، ومع ذلك، لا تزال الدراسات النظرية حول النماذج الوبائية التي تتضمن تأثيرات عشوائية بيولوجية معقولة محدودة. تهدف هذه الأطروحة إلى سد هذه الفجوة من خلال تحليل الديناميكيات التطورية للنماذج الوبائية العشوائية ودراسة تأثير الاضطرابات العشوائية على تطور الفيروسات وتطور الأمراض بشكل عام.

في هذا العمل، نقوم بتطوير ودراسة نماذج عشوائية تدمج آليات إنتاج الفيروسات مع عمليات أورنشتاين-أولنبيك أو بلاك- كاراسينسكي. على وجه التحديد، نفترض أن بعض المعلمات الوبائية الأساسية، مثل معدل انتقال الخلايا من الطور الكامن إلى الطور المعدي، تتبع عملية أورنشتاين-أولنبيك. ضمن إطار رياضي محدد بدقة، نقوم بتطوير واعتماد مناهج تحليلية جديدة وغير تقليدية لإثبات الخصائص الأساسية لهذه النماذج العشوائية، بما في ذلك وجود وحيدة الحلول العالمية، ووجود توزيع ثابت، وتحديد الشروط اللازمة لانقراض المرض.

بالإضافة إلى ذلك، نوضح أن إدخال الاضطرابات العشوائية بهذه الطريقة له مبررات رياضية قوية وأساس بيولوجي واضح. لدعم النتائج النظرية، يتم إجراء محاكاة رقمية في نهاية كل قسم، حيث تؤكد هذه المحاكاة صحة التحليل النظري وتوضح دقة الشروط والمعايير المستخلصة.

**الكلمات المفتاحية:** نموذج وبائي عشوائي، نموذج وبائي للحصبة، ديناميكيات عشوائية، نموذج عشوائي لفيروس نقص المناعة البشرية-1، معادلة فوكَر-بلانك، عملية أورنشتاين-أولنبيك، عملية بلاك- كاراسينسكي، التوزيع الثابت، كثافة الاحتمال، الانقراض.

# Dedication

*This modest work is dedicated to:*

*My dear mother,*

*My dear father,*

*My dear wife,*

*My brothers and my sisters,*

*All my family and friends.*

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# Notations

- **a.s.**: almost surely, or $P$-almost surely, or with probability 1.
- $\mathbf{A := B}$: $A$ is defined by $B$ or $A$ is denoted by $B$.
- $\mathbf{A(x) \equiv B(x)}$: $A(x)$ and $B(x)$ are identically equal, i.e., $A(x) = B(x)$ for all $x$.
- $\emptyset$: the empty set.
- $A^c$: the complement of $A$ in $\Omega$, i.e., $A^c = \Omega - A$.
- $A \subset B$: $A \cap B^c = \emptyset$.
- $a \wedge b$: the minimum of $a$ and $b$.
- $a^+ = \max\{a, 0\}$.
- $a^- = \max\{-a, 0\}$.
- $a \vee b$: the maximum of $a$ and $b$.
- $f : A \to B$: the mapping $f$ from $A$ to $B$.
- $\mathbb{N}_0 = \{0, 1, 2, \dots\}$.
- $\mathbb{Q}$: the set of rational numbers.
- $\mathbb{Q}_+$: the set of nonnegative rational numbers.
- $\mathbb{R}^d$: the $d$-dimensional Euclidean space.
- $B_r = \{x \in \mathbb{R}^d \mid \|x\| < r\}$.
- $C([0, \infty); \mathbb{R}^d)$: the space of continuous functions from $[0, \infty)$ to $\mathbb{R}^d$.

- $D([0,\infty))$: the space of functions that are right-continuous and have left-limits.
- $\mathbb{R}_+$: the set of all nonnegative real numbers, i.e., $\mathbb{R}_+ = [0,\infty)$.
- $\mathbb{R}^d$: the $d$-dimensional Euclidean space.
- $\sigma(C)$: the $\sigma$-algebra generated by $C$.
- $\mathcal{B}^d$: the Borel $\sigma$-algebra on $\mathbb{R}^d$.
- $\mathbb{R}^{d\times m}$: the space of real $d\times m$-matrices.
- $\mathcal{B}^{d\times m}$: the Borel $\sigma$-algebra on $\mathbb{R}^{d\times m}$.
- $|x|$: the Euclidean norm of a vector $x$.
- $V_x = \left(\dfrac{\partial V}{\partial x_1},\ldots,\dfrac{\partial V}{\partial x_d}\right)$.
- $V_{xx} = \left(\dfrac{\partial^2 V}{\partial x_i \partial x_j}\right)_{d\times d}$.
- $L^p([a,b];\mathbb{R}^d)$: the family of Borel measurable functions $h:[a,b]\to\mathbb{R}^d$ such that $\int_a^b |h(t)|^p\,dt<\infty$.
- $L^p([a,b];\mathbb{R}^d)$: the family of $\mathbb{R}^d$-valued $\mathcal{F}_t$-adapted processes $\{f(t)\}_{a\le t\le b}$ such that $\int_a^b |f(t)|^p\,dt<\infty$ a.s.
- $M^p([a,b];\mathbb{R}^d)$: the family of processes $\{f(t)\}_{a\le t\le b}$ in $L^p([a,b];\mathbb{R}^d)$ such that $\mathbb{E}\int_a^b |f(t)|^p\,dt<\infty$.
- $L^p(\mathbb{R}_+;\mathbb{R}^d)$: the family of processes $\{f(t)\}_{t\ge 0}$ such that for every $T>0$, $\{f(t)\}_{0\le t\le T}\in L^p([0,T];\mathbb{R}^d)$.
- $M^p(\mathbb{R}_+;\mathbb{R}^d)$: the family of processes $\{f(t)\}_{t\ge 0}$ such that for every $T>0$, $\{f(t)\}_{0\le t\le T}\in M^p([0,T];\mathbb{R}^d)$.
- $\mathbb{1}_A(x) = \begin{cases} 1, & x\in A,\\ 0, & x\notin A.\end{cases}$
- $p(t;x,y) = \dfrac{1}{\sqrt{2\pi t}} e^{-\frac{(x-y)^2}{2t}}, \quad t>0, x,y\in\mathbb{R}$.

- $\mathcal{B}(U)$: the smallest $\sigma$-field containing all open sets of the topological space $U$.
- $\sim$: means "is distributed as" or "has the probability law of".
- $\mathcal{F}_t$: the $\sigma$-field of events determined by a process up to time $t$.
- $\mathcal{F}_T^+$: the $\sigma$-field of events determined immediately after the optional time $T$.
- $\tau_D = \inf\{t \geq 0 \mid X_t \notin D\}$: the first exit time of the Brownian motion from the set $D$.
- $\tau_b = \inf\{t \geq 0 \mid X_t = b\}$: the first hitting time of level $b$ by the one-dimensional Brownian motion.
- $\mathcal{F} \otimes \mathcal{G} := \sigma(A \times B; A \in \mathcal{F}, B \in \mathcal{G})$ : The product $\sigma$-field formed from the $\sigma$-fields $\mathcal{F}$ and $\mathcal{G}$.
- $I_d$ : The $(d \times d)$ identity matrix.
- $\mathbb{E}[X_t]$: the mathematical expectation (mean) of the random variable $X_t$.
- $\mathrm{Var}(X_t)$: the variance of $X_t$.
- $\mathrm{Cov}(X_s, X_t)$: the covariance between $X_s$ and $X_t$.
- $\mathbf{R\bar{H}}(l)$: the class of Hurwitz stable matrices.

Other notations will be explained where they first appear.

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# List of published and submitted papers

- **Mhammed Mediani**, Abdeldjalil Slama, Ahmed Boudaoui, and Thabet Abdeljawad. *Analysis of a stochastic SEIuIrR epidemic model incorporating the Ornstein-Uhlenbeck process.* Heliyon, vol. 10, no. 16, 2024. Published by Elsevier.
- **Mhammed Mediani**, Abdeldjalil Slama, Ahmed Boudaoui, Hebatallah J. Alsakaji, and Fathalla A. Rihan. A stochastic SEIR model with Holling type II treatment and Ornstein–Uhlenbeck process. *Kuwait Journal of Science*, 2025, Article 100525, Elsevier.
- **Mhammed Mediani**, Abdeldjalil Slama, and Ahmed Boudaoui, *Dynamics of a Stochastic HIV-1 Model with Immune Response and Black-Karasinski Process*, **Submitted paper (2025)**.
- **Mhammed Mediani**, Abdeldjalil Slama, and Ahmed Boudaoui, *Threshold Behavior and Stochastic Effects in a Network-Based SIR Model with Saturated Treatment*, **Submitted paper (2025)**.

# List of conferences

## International Conferences

1. **Mhammed Mediani, Abdeldjalil Slama, Ahmed Boudaoui**
   **Title:** Analyzing the Stationary Distribution and Extinction of a Stochastic Epidemiological Model for COVID-19 Incorporating the Ornstein-Uhlenbeck Process
   **Conference:** RAMA12, Adrar, Algeria, November 09-12, 2023.

2. **Mhammed Mediani, Abdeldjalil Slama, Ahmed Boudaoui**
   **Title:** Stochastic Dynamics and Threshold Analysis of a Network-Structured SIR Epidemic Model with Saturated Treatment
   **Conference:** The Second International Conference on Mathematical Sciences and Applications, University May 8, 1945 Guelma, Algeria, October 27–29, 2024.

## National Conferences

1. **Mhammed Mediani, Abdeldjalil Slama, Ahmed Boudaoui**
   **Title:** Dynamics of a Stochastic Measles Model: Examining Stationary Distribution and Extinction with Ornstein–Uhlenbeck Process
   **Conference:** First National Conference on Nonlinear Differential Equations and Their Applications (NCNDEA'2024), Oum El Bouaghi University, Algeria, February 10-11, 2024.

2. **Mhammed Mediani, Abdeldjalil Slama, Ahmed Boudaoui**
   **Title:** Analysis of a Stochastic HIV-1 Model with Cell-to-Cell Transmission and Black-Karasinski Process
   **Conference:** National Conference on Mathematical Modeling for Dynamic Systems (M2DS), Constantine, Algeria, June 26-27, 2024.

3. **Abdeldjalil Kadri,Ahmed Boudaoui, Mhammed Mediani**
   **Title:** Optimal Control Analysis for the COVID-19 Stochastic Epidemic Model with Vaccination
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4. **Mhammed Mediani, Abdeldjalil Slama, Ahmed Boudaoui**
   **Title:** Extinction Dynamics and Periodic Behavior in a Stochastic Epidemic Model with Impulsive Vaccination and Ornstein–Uhlenbeck Process
   **Conference:** National Conference on Mathematics and Applications (NCMA2025), University of Adrar, Algeria, November 04–05, 2025.

5. **Abdeldjalil Kadri,Ahmed Boudaoui, Mhammed Mediani**
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# General Introduction

Epidemiology, a branch of applied mathematics, focuses on the scientific study of the spread and control of infectious diseases. It plays a vital role in public health by informing policies aimed at preventing and managing disease outbreaks. Through systematic observation, rigorous analysis, and the use of mathematical modeling, epidemiologists have developed powerful tools to understand the dynamics of disease transmission within populations. Mathematical models, in particular, have proven invaluable for predicting infection rates, estimating healthcare system demands, and assessing the effectiveness of intervention strategies such as vaccination, quarantine, and social distancing [1, 2]. The COVID-19 pandemic, with its unprecedented global consequences, underscored the critical need for flexible and accurate models that extend beyond classical epidemiological approaches.

Historically, mathematical models in epidemiology have been deterministic, offering predictions based on fixed parameters and initial conditions. Models like the SIR (Susceptible-Infectious-Recovered) [3] and SEIR (Susceptible-Exposed-Infectious-Recovered) frameworks categorize populations into health compartments and simulate how individuals transition between these compartments over time [4, 5, 6]. These models, which provide insights into infection rates, recovery times, and the potential for herd immunity, have been instrumental in shaping strategies for disease containment. For example, deterministic models played a crucial role in predicting COVID-19 infection peaks and assessing the effects of public health interventions [7, 8, 9]. Their simplicity and interpretability have made deterministic models widely applicable and accessible.

However, real-world disease dynamics are influenced by numerous unpredictable factors beyond the reach of deterministic models. Variability in individual immunity, social behavior, and environmental conditions contribute to an inherent randomness that deterministic equations struggle to capture. For instance, individual

contact patterns may vary widely, super-spreader events can lead to sudden infection spikes, and new virus strains may emerge unexpectedly, altering transmission dynamics. Recognizing these complexities, researchers have developed stochastic epidemiological models that explicitly incorporate randomness, providing more realistic simulations of disease progression [10, 11, 12]. Stochastic models capture the variability and uncertainty intrinsic to infectious disease dynamics, allowing researchers to explore a range of possible outbreak scenarios. For COVID-19, these models were particularly valuable for understanding outbreak probabilities, the effectiveness of interventions under various conditions, and the impacts of unpredictable events like new variants [13]. Thus, stochastic modeling has become a cornerstone for developing flexible, adaptable public health responses that can accommodate uncertainties.

Recently, a growing body of research has focused on exploring the dynamic behavior of epidemiological models that incorporate stochastic perturbations. This interest stems from the recognition that stochastic modeling techniques can more accurately capture the complex and variable nature of infectious disease spread. To date, several methods have been developed to integrate stochastic perturbations into deterministic models. Among these, a particularly common approach assumes that certain model parameters follow an Itô process, specifically the Ornstein-Uhlenbeck (OU) process.

This approach allows for the modeling of parameters that are not static but instead subject to real-world fluctuations and uncertainties. Parameters may vary due to factors such as changes in testing capacities, shifts in public health policies, or differences in population behavior. By employing the OU process, these variations are modeled with a mean-reverting property, ensuring that while parameters may fluctuate, they tend to return to a central value over time. This property makes the OU process an effective tool for modeling random disturbances, offering a stable yet realistic framework for understanding the stochastic elements in epidemiological dynamics.

This thesis aims to advance the study of perturbative epidemiological models by introducing new methods, generalizations, and analytical techniques for predicting and controlling disease dynamics. By combining deterministic and stochastic approaches, this research seeks to build a structured, comprehensive framework that deepens our understanding of disease spread and informs public health strategy.

Organized into five chapters, the development of the mathematical and epidemiological framework starts in Chapter 1, which provides a detailed literature review tracing the development of epidemiological modeling. It examines two main areas: (1) Classical deterministic models, such as the SIR and SEIR frameworks, which have historically shaped disease modeling, and (2) Stochastic models, which incorporate randomness to address the unpredictable nature of disease spread. This chapter highlights how stochastic approaches evolved to capture real-world variability more accurately, underscoring their importance in modern epidemiology.

Chapter 2 lays the theoretical groundwork by presenting key definitions, essential mathematical results, and the analytical tools used throughout the work. The core findings are presented in Chapter 3, where we develop and analyze a new stochastic model for COVID-19 that integrates the Ornstein-Uhlenbeck process. This chapter begins by proving the global positivity and uniqueness of the model's solution. By constructing specific Lyapunov functions, we derive the condition $\mathcal{R}_0^s > 1$, which ensures the existence of a unique stationary distribution with ergodic properties. We also establish criteria for the exponential extinction of the disease. Numerical simulations validate these theoretical results, demonstrating the model's practical applicability.

In Chapter 4, we introduce a stochastic model for measles transmission. We establish the existence and uniqueness of a globally positive solution and use the Lyapunov method to demonstrate the existence of a stationary distribution under the threshold condition $\mathcal{R}_0^s > 1$, indicating the disease's persistence in biological terms. Sufficient conditions for disease extinction are also derived, providing a more comprehensive view of measles transmission dynamics. Numerical simulations further illustrate the impact of stochastic factors on disease spread.

Chapter 5 explores a stochastic model for HIV-1 that incorporates virus-to-cell and cell-to-cell transmission, as well as humoral immunity, within the Black-Karasinski process framework. This process is shown to be biologically relevant for HIV-1 modeling. We demonstrate the existence of a unique global solution and introduce two critical thresholds, $\mathcal{R}_0^s$ and $\mathcal{R}_0^{ex}$, which classify the possible dynamic behaviors of HIV-1. Our results indicate that (i) if $\mathcal{R}_0^s > 1$, the model has a stationary distribution $\Psi(.)$, revealing the system's long-term behavior, and (ii) if $\mathcal{R}_0^{ex} < 1$, infected cell and virus populations decline significantly, suggesting a foundation for HIV-1 control strategies. By solving the corresponding Fokker-Planck equation, we approximate

the probability density function of $\Psi(.)$ near a positive quasi-equilibrium, providing insights into the probabilistic distribution around equilibrium states. These theoretical findings are supported by numerical simulations.

Each chapter (Chapters 3 to 5) introduces novel analytical methods that extend existing studies in mathematical epidemiology, providing tools that are adaptable to a broader range of epidemiological models. Results from these chapters have been published or submitted to peer-reviewed journals, underscoring the scientific significance and practical relevance of this research. This thesis offers a comprehensive set of analytical tools and frameworks for understanding disease transmission in both deterministic and stochastic contexts, enhancing the precision and adaptability of epidemiological models.

Chapter 1

# Frameworks and Variants in Epidemiological Modeling: Deterministic and Stochastic Approaches

## 1.1 Overview of Compartmental Epidemiological Models

The historical development of compartmental models in epidemiology dates back to 1906 when W.H. Hamer introduced the concept that the spread of infection depended on the number of susceptible and infected individuals in a population [14]. His formulation, based on the "mass action law," provided a method for calculating new infection rates, which later became a cornerstone of compartmental modeling. Expanding on Hamer's ideas, Ross adapted the mass action principle to a continuous-time context, developing a model in 1911 that incorporated both mosquito and human populations [15, 16]. His work demonstrated that reducing the mosquito population below a critical threshold could effectively limit disease spread, a concept that later contributed to the development of the basic reproduction number, an essential measure in epidemiology.

Building on these early contributions, Kermack and McKendrick introduced their influential epidemic model between 1927 and 1933 through a series of three seminal papers [3, 17, 18]. Initially formulated as a simple model, it was later generalized to incorporate factors such as the "age of infection," representing the time elapsed since an individual became infected. Their work laid the foundation for understanding disease dynamics through compartmental models, providing a comprehensive framework that has since been extended and refined in numerous ways. Although Ross and Kermack-McKendrick did not explicitly define the threshold now known as the basic reproduction number ($R_0$), it was formally introduced by MacDonald in 1957 during his research on malaria [19]. Around the same time, N. Bailey's book, The Mathematical Theory of Infectious Diseases and Its Applications, synthesized existing knowledge and further enriched the field [20]. Subsequent contributions by Dietz in 1967 and Hethcote in 1976 expanded the theoretical landscape, leading to the development of key epidemiological models such as $SIS$, $SIR$, and $SIRS$, which continue to be widely used in research today [21, 22, 23].

As these models evolved, researchers introduced greater complexity to account for real-world epidemiological factors. In 1992, Lorca and Hethcote refined the $SIRS$ model by incorporating birth and death rates [24], while Hethcote also examined pertussis transmission using age-structured models [25]. Further advance-

ments included delays, additional compartments, and the integration of vaccination effects to capture epidemiological nuances more accurately [26]. Studies by Wen, Yang, and others explored critical aspects such as disease latency, immunity duration, and vaccination impact, leading to the development of extended models like $SAIRS$ and $MSEIR$, which considered asymptomatic carriers and maternal immunity in infants, respectively [27, 28, 29].

Hethcote's introduction of the $MSEIR$ model emphasized the significance of $R_0$ in determining disease dynamics. Specifically, when $R_0 \leq 1$, the disease-free state remains stable, whereas an $R_0 > 1$ leads to a stable endemic equilibrium [30]. This realization underscored the predictive power of $R_0$ in assessing long-term disease trends and equilibrium states within populations. Over the past century, compartmental models have undergone continuous refinement, adapting to new scientific insights and real-world challenges. From early theoretical assumptions to sophisticated, highly structured simulations, these models remain indispensable tools for understanding and controlling infectious diseases.

## 1.2 Deterministic Frameworks of Classical Models

In mathematical epidemiology, models categorize individuals based on their disease status, using several key compartments to represent different stages in the infection process:

- **Susceptible (S):** Individuals who have not contracted the disease but are at risk. They can become infected upon exposure to an infected person.
- **Exposed (E):** Individuals in the incubation phase who have been infected but are not yet able to transmit the disease to others. This stage represents those who harbor the infection but do not yet exhibit symptoms.
- **Infected (I):** Individuals actively carrying the disease and capable of spreading it to susceptible individuals. They are the primary drivers of transmission within the population.
- **Recovered or Removed (R):** Individuals who have either overcome the disease and gained immunity (either temporary or permanent) or have been isolated from the population. They no longer contribute to disease transmission.

These categories serve as the foundation for classical models, which describe the spread of infectious diseases through populations by tracking individuals as they transition between these compartments. Such models, including SIR, SEIR, and their variations, provide valuable insights into the dynamics of disease spread and the effectiveness of control measures.

### Susceptible-Infectious (SI) Model

The Susceptible-Infected (SI) model serves as one of the simplest frameworks in mathematical epidemiology, designed to capture the transmission dynamics of certain infectious diseases. In this model, the population is divided into two key compartments:

- **Susceptible (S):** Individuals who are vulnerable to the disease and may become infected upon contact with an infected individual.
- **Infected (I):** Individuals who have contracted the disease and remain infected indefinitely, with no possibility of recovery.

This model is particularly suitable for diseases where infection leads to a permanent state, without recovery, such as in the cases of rabies or HIV/AIDS [31, 32, 33]. The SI model's simplicity allows researchers to analyze the spread of such diseases over time by observing the transition of individuals from the susceptible to the infected compartment.

### Susceptible-Infectious-Susceptible (SIS) Model

The Susceptible-Infected-Susceptible (SIS) model builds upon the basic structure of the SI model but includes a key distinction: individuals who become infected have a probability of recovery and return to the susceptible state, rather than remaining permanently infected.

In this model, individuals transition between two main compartments:

- **Susceptible (S):** Individuals who have not contracted the disease and are at risk of infection.
- **Infected (I):** Individuals who are currently infected. Unlike the SI model, these individuals can recover and revert to the susceptible state, meaning they remain vulnerable to re-infection.

The SIS model is particularly well-suited for infectious diseases where immunity is temporary or non-existent, allowing individuals to be re-infected multiple times. This structure is applicable to diseases such as sexually transmitted infections (e.g., gonorrhea), parasitic infections like malaria, and certain bacterial infections including pertussis and tuberculosis [34, 35].

### Susceptible-Infectious-Recovered (SIR) Model

The Susceptible-Infected-Recovered (SIR) model is a foundational approach in epidemic modeling, dividing the population into three distinct stages:

- **Susceptible (S):** Individuals who have not yet contracted the disease but are at risk of infection.
- **Infected (I):** Individuals who have been exposed to and are currently carrying the disease, thereby capable of spreading it to susceptible individuals.
- **Recovered (R):** Individuals who have overcome the infection and gained immunity, either temporarily or permanently, thus no longer participating in disease transmission.

This model is particularly applicable to diseases where individuals gain immunity after infection, such as chickenpox, rubella, and polio. Its simplicity and clear compartmental transitions make the SIR model a widely used framework for studying the spread of infections that follow this immunity-gaining pattern [36, 37, 38, 39].

### Susceptible-Infectious-Recovered-Susceptible (SIRS) Model

The Susceptible-Infected-Recovered-Susceptible (SIRS) model extends the traditional SIR model by incorporating a mechanism for the loss of immunity over time. In this framework, individuals who have recovered from an infection gradually return to a susceptible state, allowing for the possibility of re-infection.

The compartments in this model are defined as follows:

- **Susceptible (S):** Individuals who are at risk of infection.
- **Infected (I):** Individuals who are currently infected and capable of spreading the disease.

- **Recovered (R):** Individuals who have recovered from infection and temporarily gained immunity, which will wane over time, making them susceptible again.

The SIRS model is particularly suited for analyzing diseases where immunity is not permanent, such as dengue fever, seasonal influenza, and respiratory syncytial virus. These infections are characterized by a cyclical pattern of susceptibility and immunity due to the gradual loss of immune protection [40, 41, 42].

#### Susceptible-Exposed-Infectious-Recovered (SEIR) Model

The Susceptible-Exposed-Infected-Recovered (SEIR) model expands upon the SIR framework by introducing an additional compartment to account for a latent period, during which individuals have contracted the infection but are not yet infectious.

This model consists of the following stages:

- **Susceptible (S):** Individuals who are at risk of infection.
- **Exposed (E):** Individuals who have been infected and are in an incubation phase. At this stage, they carry the disease but cannot yet transmit it to others.
- **Infected (I):** Individuals who have progressed to the infectious phase and are capable of spreading the disease.
- **Recovered (R):** Individuals who have overcome the infection and are now immune, either temporarily or permanently.

By adding the *Exposed* compartment, the SEIR model effectively incorporates a delay between initial infection and the onset of infectiousness, which is essential for modeling diseases with an incubation period, such as measles or Ebola. This structure allows for a more accurate representation of diseases where individuals do not become immediately infectious following exposure [43, 44, 45, 46, 47].

## 1.3 Extensions and Non-Classical Models

Non-classical epidemic models build upon the traditional SIR framework by introducing additional compartments to capture various complexities of disease transmission and immunity [48]. These additional classes help account for factors like

asymptomatic carriers, quarantine measures, vaccination, hospitalization, and maternally derived immunity. Below are descriptions of some commonly added compartments:

- **Asymptomatic (A):** This compartment represents individuals who are infected but show no symptoms. Despite being asymptomatic, they can still contribute to disease transmission.
- **Quarantined (Q):** Individuals in this compartment are infected but isolated from the general population, which limits their ability to spread the disease.
- **Vaccinated (V):** This group includes individuals who have received a vaccine and thus have acquired immunity, either partially or fully, against the infection.
- **Hospitalized (H):** Infected individuals experiencing severe symptoms may be hospitalized, where their chances of spreading the disease are reduced due to medical containment measures.
- **Maternally Derived Immunity (M):** This compartment covers newborns who possess temporary immunity received through maternal antibodies, providing protection during early life.

These additional compartments allow for the development of modified epidemic models that can address more specific characteristics of diseases and public health interventions. Such adaptations are essential for modeling diseases that do not fit within the classical framework, providing a nuanced approach to understanding transmission dynamics.

#### Susceptible-Asymptomatic-Infectious-Recovered-Susceptible (SAIRS) Model

The Susceptible-Asymptomatic-Infected-Recovered-Susceptible (SAIRS) model extends the basic SIRS framework by including an *Asymptomatic* compartment, which represents individuals who, although infected, do not exhibit any symptoms. Despite the lack of symptoms, asymptomatic individuals are capable of transmitting the disease, adding a layer of complexity to disease spread dynamics.

The compartments in the SAIRS model are as follows:

- **Susceptible (S):** Individuals who are at risk of infection.
- **Asymptomatic (A):** Infected individuals who show no symptoms but can still transmit the infection to others.
- **Infected (I):** Symptomatic individuals who are currently capable of spreading the disease.
- **Recovered (R):** Individuals who have recovered and gained temporary immunity, which eventually wanes, returning them to the susceptible state.

This model is especially useful for capturing the dynamics of diseases like Norovirus and COVID-19, where asymptomatic transmission plays a significant role in the spread of infection. By including an asymptomatic group, the SAIRS model provides a more realistic framework for understanding these diseases' transmission patterns [49, 50, 51, 52].

#### Susceptible-Infectious-Quarantined-Recovered (SIQR) Model

The Susceptible-Infected-Quarantined-Recovered (SIQR) model builds on the traditional SIR model by incorporating a *Quarantined* compartment. This addition represents infected individuals who are isolated from the susceptible population to reduce the spread of infection.

The compartments in the SIQR model are structured as follows:

- **Susceptible (S):** Individuals who are at risk of contracting the infection.
- **Infected (I):** Individuals who are actively infected and capable of transmitting the disease.
- **Quarantined (Q):** Infected individuals who have been isolated to prevent further transmission to the susceptible population.
- **Recovered (R):** Individuals who have recovered from the infection and are now immune.

By incorporating a quarantined group, the SIQR model provides a valuable framework for analyzing the effects of isolation measures on disease spread. This model is particularly relevant for managing and studying diseases where quarantine and

isolation are key control strategies, such as in the cases of COVID-19 and Ebola [53, 54, 55, 56].

### Susceptible-Vaccinated-Infectious-Recovered (SVIR) Model

The Susceptible-Vaccinated-Infected-Recovered (SVIR) model expands upon the traditional SIR framework by incorporating a *Vaccinated* compartment. This additional category represents individuals who have received immunization against the disease, thereby reducing their likelihood of infection depending on the effectiveness of the vaccine.

In this model, individuals are divided into the following compartments:

- **Susceptible (S):** Individuals who have not contracted the disease and are at risk of infection.
- **Vaccinated (V):** Those who have been vaccinated, thereby having a lower probability of becoming infected based on the vaccine's efficacy.
- **Infected (I):** Individuals who are currently infected and capable of spreading the disease.
- **Recovered (R):** Individuals who have recovered from the disease and have acquired immunity.

The SVIR model is particularly relevant for analyzing diseases where vaccination is a primary control measure, such as measles and influenza. It helps in evaluating the role of immunization in reducing susceptibility and, consequently, controlling the spread of infection [57, 58, 59, 60].

### Susceptible-Exposed-Infectious-Hospitalized-Recovered (SEIHR) Model

The Susceptible-Exposed-Infected-Hospitalised-Recovered (SEIHR) model extends the standard SEIR model by adding a *Hospitalised* compartment. This modification allows for a more detailed analysis of diseases where some infected individuals experience severe symptoms and require hospital care.

The SEIHR model includes the following compartments:

- **Susceptible (S):** Individuals who have not been exposed to the disease but are at risk of infection.

- **Exposed (E):** Individuals who have contracted the disease and are in the incubation period, during which they are not yet infectious.
- **Infected (I):** Individuals who are actively infected and capable of transmitting the disease.
- **Hospitalised (H):** Infected individuals with severe symptoms who have been admitted to a hospital, where their potential to spread the disease is reduced due to isolation.
- **Recovered (R):** Individuals who have recovered from the disease, acquiring immunity.

The addition of the hospitalised compartment in the SEIHR model enables the examination of hospitalisation's impact on disease transmission dynamics. It is particularly relevant for studying illnesses such as COVID-19, where hospitalisation plays a significant role in managing severe cases and reducing the spread of infection [61, 62, 63, 64].

#### Maternal-Susceptible-Exposed-Infectious-Recovered (MSEIR) Model

The Maternally Derived Immunity-Susceptible-Exposed-Infected-Recovered (MSEIR) model expands upon the SEIR framework by introducing a compartment for maternally derived immunity, enabling it to account for temporary immunity in newborns passed down from mothers.

This model includes the following compartments:

- **Maternally Derived Immunity (M):** Newborns who possess temporary immunity acquired through maternal antibodies, which is often conferred during pregnancy or through breastfeeding.
- **Susceptible (S):** Individuals without immunity who are at risk of infection once maternal immunity wanes.
- **Exposed (E):** Individuals who have been infected but are not yet infectious, representing the incubation period.
- **Infected (I):** Individuals who have contracted the disease and can transmit it to others.

- **Recovered (R):** Individuals who have recovered and gained immunity.

The MSEIR model is useful for studying diseases such as measles or chickenpox, where newborns initially receive immunity from maternal antibodies, which eventually diminishes over time, increasing susceptibility [30, 65].

## 1.4 Stochastic of Epidemiological Models

Deterministic models have long been valuable in epidemiology, providing insights into how diseases spread by simulating real-world conditions and outcomes. However, these models may lack precision in capturing the stochastic, or random, behaviors observed in real epidemics, particularly when small population sizes are involved. In these cases, the randomness inherent in disease transmission can cause significant deviations from predictions made by deterministic models. This makes stochastic models essential for a more nuanced understanding of disease dynamics.

### 1.4.1 Early Foundations of Stochastic Epidemic Modeling

The field of epidemic modeling saw foundational contributions in the 1920s and 1930s. Kermack and McKendrick's groundbreaking work established compartmental models that treated populations in distinct disease states, such as susceptible, infected, and recovered (SIR models) [3, 17, 18]. Their deterministic framework laid the groundwork for later stochastic adaptations. By analyzing disease transmission and recovery dynamics, their models provided an essential platform for the development of stochastic modeling approaches.

In 1928 and 1931, Reed and Frost, alongside Greenwood, introduced discrete-time stochastic models focusing on the generation-by-generation spread of infection [66]. Although Reed and Frost's model was not published immediately, it was shared through lectures in 1928 and later became a fundamental reference in stochastic epidemic theory. This model emphasized randomness in the transmission process, which is critical in smaller populations where disease outcomes can vary significantly from deterministic predictions.

### 1.4.2 Contributions of Key Figures in Stochastic Epidemic Theory

Building upon Kermack and McKendrick's models, Bartlett's work in the late 1940s highlighted the importance of incorporating randomness, especially in small populations where stochastic effects are pronounced. His stochastic extensions provided a deeper understanding of how random fluctuations in transmission rates could impact disease spread. Bartlett's approach advanced the field by showing that random variations in infection and recovery rates are central to accurate epidemic modeling [67, 68].

Kendall also contributed significantly to stochastic epidemic modeling. His studies on branching processes—a type of stochastic process where individuals can produce multiple 'offspring'—have direct applications in epidemiology. This approach is particularly relevant in modeling scenarios where a single infected individual may spread the disease to multiple others, capturing a more realistic view of disease transmission dynamics [69].

Bailey's seminal work in epidemic modeling provided a thorough overview of both deterministic and stochastic frameworks, as well as guidance on parameter estimation techniques. His work continues to be a key reference for researchers exploring both traditional and modern approaches to epidemic modeling [20].

Additional influential figures in stochastic epidemic modeling include Ball, Becker, and Barbour, who expanded the theoretical foundation and practical applications of these models. Each contributed unique perspectives and models that have been instrumental in understanding epidemic behaviors under stochastic influences [70, 71, 72, 73, 74, 75].

### 1.4.3 Modern Advances: Incorporating Environmental Noise into Stochastic Models

Recent advancements in stochastic epidemic modeling have incorporated various forms of noise—such as Gaussian, Lévy, and telegraph noise—into disease models. Environmental noise, especially white noise, represents small, continuous variations in transmission parameters due to external factors [76]. Recognizing the impact of these minor fluctuations is essential for constructing models that reflect realistic, variable conditions in disease spread.

### 1.4.4 Exploring the Impact of Stochastic Differential Equations in Modeling Uncertainty

Stochastic differential equations (SDEs) have become increasingly valuable tools, offering a flexible framework that allows for the incorporation of random environmental effects. This approach has improved the realism of epidemic models, as SDEs capture the impact of stochastic perturbations on disease dynamics more accurately than deterministic models [76, 77, 78].

Gray et al.[77], for example, analyzed a stochastic SIS model and explored the necessary conditions for disease extinction and persistence, correlating these states with perturbation levels and the basic reproduction number, $\mathcal{R}_0$ . Their findings revealed conditions under which a stationary distribution could exist and provided expressions for its mean and variance. Zhao and Jiang expanded on earlier work by integrating environmental white noise into an SIV model with vaccination [79], introducing a modified reproduction number $\tilde{\mathcal{R}}_0$, to examine extinction and persistence conditions under stochastic influences. Fatini et al.[80] studied a stochastic SIRI model with relapse, demonstrating the system's well-posedness and defining a stochastic threshold and stability conditions.

The evolution of stochastic epidemic modeling reflects the growing need for flexible, realistic models that capture both the predictable and unpredictable elements of disease transmission. By incorporating randomness at various levels-from individual interactions to environmental fluctuations-stochastic models offer a robust alternative to deterministic approaches, especially in capturing the complexities of disease spread in diverse population settings.

Chapter 2

# Preliminaries

In this brief section, we provide essential definitions and foundational concepts related to random processes, covering only the basics needed for the remainder of the thesis, without delving into the comprehensive analysis available in [81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93].

## 2.1 Events and Probability

In the study of uncertainty and randomness, events and probability serve as foundational concepts. Events represent outcomes or collections of outcomes from a random experiment, while probability provides a systematic way to quantify the likelihood of these events. Together, they form the basis of probability theory, enabling us to model and analyze phenomena governed by chance, from rolling dice to complex epidemiological dynamics. Before diving into formal definitions and properties, it is crucial to understand these building blocks that underpin all stochastic models and probabilistic reasoning.
The definitions, theories, and properties presented in this section are adapted from and based on the works referenced in [81].

**Definition 2.1.**
Let $\Omega$ be a non-empty set. A collection of subsets $\mathcal{F}$ is said to form a $\sigma$-field on $\Omega$ if the following properties hold:

1. $\mathcal{F}$ contains the empty set $\emptyset$.
2. If a set $A$ is in $\mathcal{F}$, then its complement $\Omega \setminus A$ is also included in $\mathcal{F}$.
3. For any sequence of sets $A_1, A_2, \ldots$ in $\mathcal{F}$, their union $\bigcup_{i=1}^{\infty} A_i$ is also an element of $\mathcal{F}$.

**Definition 2.2.**
Let $\mathcal{F}$ be a $\sigma$-algebra on the set $\Omega$. A probability measure $P$ is a mapping

$$P : \mathcal{F} \to [0,1]$$

that satisfies the following properties:

1. $P(\Omega) = 1$;

2. For any collection of pairwise disjoint sets $A_1, A_2, \ldots$ (where $A_i \cap A_j = \emptyset$ for $i \neq j$) in $\mathcal{F}$, the measure satisfies

$$P\left(\bigcup_{i=1}^{\infty} A_i\right) = \sum_{i=1}^{\infty} P(A_i). \tag{2.1}$$

The structure $(\Omega, \mathcal{F}, P)$ is referred to as a *probability space*. The elements of $\mathcal{F}$ are called *events*, and an event $A$ is said to happen *almost surely* (a.s.) if $P(A) = 1$.

## 2.2 Random Variables

A random variable is a fundamental concept in probability theory that bridges the gap between abstract outcomes of random experiments and real-valued quantities that we can analyze. Essentially, a random variable is a function that maps the outcomes of a probabilistic experiment to numerical values, allowing us to quantify uncertainty. By studying random variables, we can explore the statistical properties of random systems and model various phenomena, including those found in stochastic processes and epidemiological models.

The concepts, definitions, and theoretical foundations discussed in this section have been drawn and adapted from the references [81].

**Definition 2.3.**

Let $\mathcal{F}$ be a $\sigma$-algebra on the set $\Omega$. A function $\xi : \Omega \to \mathbb{R}$ is called $\mathcal{F}$-measurable if

$$\{\omega \in \Omega : \xi(\omega) \in B\} \in \mathcal{F} \tag{2.2}$$

for every Borel set $B \in \mathcal{B}(\mathbb{R})$. In the context of a probability space $(\Omega, \mathcal{F}, P)$, any such function $\xi$ is referred to as a *random variable*.

**Definition 2.4.**

A random variable $\xi : \Omega \to \mathbb{R}$ induces a probability measure on $\mathbb{R}$, given by

$$P_\xi(B) = P(\{\omega \in \Omega : \xi(\omega) \in B\}), \tag{2.3}$$

where $B \in \mathcal{B}(\mathbb{R})$ is a Borel set. This probability measure $P_\xi$ is called the *distribution* of $\xi$. The function $F_\xi : \mathbb{R} \to [0, 1]$, defined as

$$F_\xi(x) = P(\{\omega \in \Omega : \xi(\omega) \leq x\}), \tag{2.4}$$

is referred to as the *cumulative distribution function* (c.d.f.) of $\xi$.

**Proposition 2.1.**

*Suppose $\xi, \eta : \Omega \to \mathbb{R}$ are two integrable random variables that are independent. Then, they are* uncorrelated, *meaning*

$$E[\xi\eta] = E[\xi]E[\eta], \tag{2.5}$$

*provided that the random variable $\xi\eta$ is also integrable. Furthermore, if $\xi_1, \ldots, \xi_n : \Omega \to \mathbb{R}$ are independent integrable random variables, then*

$$E[\xi_1\xi_2 \ldots \xi_n] = E[\xi_1]E[\xi_2] \ldots E[\xi_n], \tag{2.6}$$

*under the assumption that the product $\xi_1\xi_2 \ldots \xi_n$ is also integrable.*

**Proposition 2.2.**

*A random variable $\xi$ is said to be* independent *of a $\sigma$-algebra $\mathcal{G}$ if the $\sigma$-algebras $\sigma(\xi)$ and $\mathcal{G}$ are independent. This concept extends naturally to a collection (finite or infinite) of random variables, $\sigma$-algebras, or a mixture of both. Specifically, such a collection is called* independent *if, for any finite selection of random variables $\xi_1, \ldots, \xi_m$ and $\sigma$-algebras $\mathcal{G}_1, \ldots, \mathcal{G}_n$ from this collection, the $\sigma$-algebras*

$$\sigma(\xi_1), \ldots, \sigma(\xi_m), \mathcal{G}_1, \ldots, \mathcal{G}_n$$

*are independent.*

**Definition 2.5.**

Let $\xi$ be an integrable random variable, and let $\eta$ be any random variable. The conditional expectation of $\xi$ given $\eta$, denoted by $E(\xi \mid \eta)$, is a random variable satisfying the following properties:

1. The random variable $E(\xi \mid \eta)$ is measurable with respect to the $\sigma$-algebra generated by $\eta$, denoted by $\sigma(\eta)$.

2. For every measurable set $A \in \sigma(\eta)$, the equality

$$\int_A E(\xi \mid \eta)\, dP = \int_A \xi\, dP \tag{2.7}$$

holds.

**Remark 2.1.**

The conditional probability of an event $A \in \mathcal{F}$ given $\eta$ can alternatively be described as

$$P(A \mid \eta) = E(\mathbb{1}_A \mid \eta), \tag{2.8}$$

where $\mathbb{1}_A$ is the indicator function of the event $A$.

**Proposition 2.3.**

*The conditional expectation satisfies the following fundamental properties:*

1. *For any real numbers $a, b$, and for any integrable random variables $\xi, \zeta$, we have*
$$E(a\xi + b\zeta \mid G) = aE(\xi \mid G) + bE(\zeta \mid G) \quad \textit{(linearity)}. \tag{2.9}$$
2. *For any integrable random variable $\xi$,*
$$E(E(\xi \mid G)) = E(\xi). \tag{2.10}$$
3. *If $\xi$ is $G$-measurable and $\xi\zeta$ is integrable, then*
$$E(\xi\zeta \mid G) = \xi E(\zeta \mid G) \quad \textit{(taking out known factors)}. \tag{2.11}$$
4. *If $\xi$ is independent of $G$, then*
$$E(\xi \mid G) = E(\xi) \quad \textit{(independence simplifies the expectation)}. \tag{2.12}$$
5. *For $\sigma$-fields $H \subseteq G$, the tower property holds:*
$$E(E(\xi \mid G) \mid H) = E(\xi \mid H). \tag{2.13}$$
6. *If $\xi \geq 0$, then*
$$E(\xi \mid G) \geq 0 \quad \textit{(positivity)}. \tag{2.14}$$

*Here, $\xi$ and $\zeta$ are random variables defined on a probability space $(\Omega, \mathcal{F}, P)$, and $G$ and $H$ are sub-$\sigma$-algebras of $\mathcal{F}$. All stated equalities and the inequality in (6) hold almost surely with respect to $P$.*

## 2.3 Gaussian Random Variables

Gaussian random variables, fundamental in probability theory, are characterized by their symmetric bell-shaped density function, determined by the mean and variance. These variables arise naturally in diverse contexts, such as measurement errors or biological systems. Their importance is underscored by the Central Limit Theorem, which explains their prevalence in modeling the sum of random influences.

In the context of this thesis, Gaussian random variables play a vital role in modeling the random disturbances within epidemiological systems, providing a mathematical foundation for analyzing variability in disease dynamics.
The definitions, properties, and theories outlined in this section are derived from and informed by the works cited in references [82].

**Definition 2.6.**

A real-valued random variable $X$ is called a **standard normal (or Gaussian)** variable if its probability distribution is characterized by the density function

$$p_X(x) = \frac{1}{\sqrt{2\pi}} \exp\left(-\frac{x^2}{2}\right), \tag{2.15}$$

where $x \in \mathbb{R}$.

**Proposition 2.4.**

*A real-valued random variable $Y$ is said to follow a Gaussian distribution with parameters $\mathcal{N}(m, \sigma^2)$, where $\sigma > 0$ and $m \in \mathbb{R}$, if any of the following three equivalent conditions are satisfied:*

*(i) $Y$ can be expressed as $Y = \sigma X + m$, where $X$ is a standard normal random variable (i.e., $X$ has a $\mathcal{N}(0,1)$ distribution);*

*(ii) The probability density function of $Y$ is given by*

$$p_Y(y) = \frac{1}{\sigma\sqrt{2\pi}} \exp\left(-\frac{(y-m)^2}{2\sigma^2}\right), \quad y \in \mathbb{R}; \tag{2.16}$$

*(iii) The characteristic function of $Y$ takes the form*

$$\mathbb{E}[e^{i\xi Y}] = \exp\left(im\xi - \frac{\sigma^2}{2}\xi^2\right), \quad \xi \in \mathbb{R}. \tag{2.17}$$

*Moreover, for such a $Y$, the expected value and variance are given by:*

$$\mathbb{E}[Y] = m, \quad var(Y) = \sigma^2. \tag{2.18}$$

**Definition 2.7 (Sum of independent normal variables).**

Let $Y$ be a random variable with a $\mathcal{N}(m, \sigma^2)$ distribution and $Y'$ a random variable with a $\mathcal{N}(m', \sigma'^2)$ distribution. If $Y$ and $Y'$ are independent, then the random variable $Y + Y'$ is distributed according to $\mathcal{N}(m + m', \sigma^2 + \sigma'^2)$.

**Proposition 2.5.**

*Let $(X_n)_{n\geq 1}$ be a sequence of real-valued random variables such that, for each $n > 1$, $X_n$ has a $\mathcal{N}(m_n, \sigma_n^2)$ distribution. Assume that $X_n$ converges to a random variable $X$ in the $L^2$ sense. Then:*

*(i) The random variable $X$ is distributed according to $\mathcal{N}(m, \sigma^2)$, where $m = \lim_{n\to\infty} m_n$ and $\sigma = \lim_{n\to\infty} \sigma_n$.*

*(ii) This convergence also extends to all $L^p$ spaces for $1 \leq p < \infty$.*

**Theorem 2.6.**

*(i) Let $\Sigma$ be a nonnegative, symmetric endomorphism of a finite-dimensional vector space $E$. Then, there exists a Gaussian random vector $X$ such that its covariance operator coincides with $l$.*

*(ii) Suppose $X$ is a centered Gaussian random vector, represented as $X = (X_1, \ldots, X_d)$ in a basis of $E$ where the covariance operator is diagonal. Let $X$ be expressed as $X = \sum_{j=1}^{d} \lambda_j e_j$, where $1 \leq j \leq d$, and the eigenvalues satisfy:*

$$\lambda_1 \geq \lambda_2 \geq \ldots \geq \lambda_d > 0 = \lambda_{r+1} = \ldots = \lambda_d.$$

*In this decomposition, $X = \sum_{j=1}^{d} Y_j e_j$, where $Y_j$ are independent centered Gaussian random variables, with variances $\mathrm{Var}(Y_j) = \lambda_j$. Consequently, the support of the probability distribution $p_X$ of $X$ is the subspace of $E$ spanned by $e_1, \ldots, e_r$. Additionally, $p_X$ is absolutely continuous with respect to the Lebesgue measure on $E$ if and only if $r = d$. In that case, the density of $X$ is given by:*

$$p_X(x) = \frac{1}{(2\pi)^{d/2}\sqrt{\det \Sigma}} \exp\left(-\frac{1}{2}\langle x, \Sigma^{-1} x\rangle\right), \tag{2.19}$$

*where $\gamma$ denotes the covariance operator.*

## 2.4 Stochastic Process

A stochastic process is a collection of random variables indexed by time or another parameter, which evolves over time according to probabilistic rules. In contrast to deterministic processes, where outcomes are predictable, a stochastic process incorporates randomness, making it suitable for modeling phenomena that evolve unpredictably, such as population dynamics in epidemiology or financial markets. The study of stochastic processes allows us to describe systems where the future state depends not only on the current state but also on inherent random fluctuations. These processes are central to the analysis of systems subject to uncertainty,

and they provide a framework for understanding a wide range of real-world situations.

The definitions, theories, and properties presented in this section are adapted from and based on the works referenced in [82, 83, 84, 85, 86].

**Definition 2.8.**

Let $T$ be a set, $(E, \mathcal{E})$ a measurable space. A stochastic process indexed by $T$, taking its values in $(E, \mathcal{E})$, is a family of measurable mappings $X_t, t \in T$, from a probability space $(\Omega, \mathcal{F}, P)$ into $(E, \mathcal{E})$. The space $(E, \mathcal{E})$ is called the state space.

The set $T$ may be thought of as "time". The most usual cases are $T = \mathbb{N}$ and $T = \mathbb{R}_+$, but they are by no means the only interesting ones. In this book, we consider deal mainly with the case $T = \mathbb{R}_+$. and $E$ will usually be $\mathbb{R}^d$ or a Borel subset of $\mathbb{R}^d$ and $\mathcal{E}$ the Borel $\sigma$-field on $E$.

For every $\omega \in \Omega$, the mapping $t \mapsto X_t(\omega)$ is a "curve" in $E$ which is referred to as a *trajectory* or a *path* of $X$. We may think of a path as a point chosen randomly in the space $\mathcal{F}(T, E)$ of all functions from $T$ into $E$, or, as we shall see later, in a reasonable subset of this space.

**Theorem 2.7 ([83]).**

*Given a probability measure $\mu$ on $\mathbb{R}$, there exist a probability space $(\Omega, \mathcal{F}, P)$ and a sequence of independent random variables $X_n$, defined on $\Omega$, such that $X_n(P) = \mu$ for every $n$.*

**Theorem 2.8 ([83]).**

*Let $\mu$ be a probability measure on $\mathbb{R}$. Then, there exists a probability space $(\Omega, \mathcal{F}, P)$ and a collection of independent random variables $(X_n)_{n\geq 1}$ defined on this space such that each $X_n$ has the distribution $\mu$, i.e., $X_n \sim \mu$ for all $n \geq 1$.*

**Proposition 2.9.**

*Let $H$ be a separable real Hilbert space. There exist a probability space $(\Omega, \mathcal{F}, P)$ and a family $X(h), h \in H$, of random variables on this space, such that*

1. *the map $h \mapsto X(h)$ is linear;*
2. *for each $h$, the r.v. $X(h)$ is gaussian centered and*

$$\mathbb{E}[X(h)^2] = \|h\|_H^2. \tag{2.20}$$

**Definition 2.9.**

A process $Y$ is said to be a modification of a process $X$ if, for all $t \geq 0$, the equality $X_t = Y_t$ holds almost surely, i.e.,

$$P(X_t = Y_t) = 1. \tag{2.21}$$

**Definition 2.10.**

The processes $X$ and $Y$ are said to have identical finite-dimensional distributions if, for every positive integer $n$, any sequence of times $0 \leq t_1 < t_2 < \cdots < t_n < \infty$, and every measurable set $A \in \mathcal{B}(\mathbb{R}^n)$, the following holds:

$$P[(X_{t_1}, \ldots, X_{t_n}) \in A] = P[(Y_{t_1}, \ldots, Y_{t_n}) \in A]. \tag{2.22}$$

**Definition 2.11.**

The processes $X$ and $Y$ are said to be indistinguishable if their sample paths coincide almost surely, meaning:

$$P[X_t = Y_t \text{ for all } t \geq 0] = 1. \tag{2.23}$$

**Definition 2.12.**

Let $X$ and $Y$ be stochastic processes defined on the probability spaces $(\Omega, \mathcal{F}, P)$ and $(\Omega', \mathcal{F}', P')$, respectively, with a common state space $(\mathbb{R}^d, \mathcal{B}(\mathbb{R}^d))$. The processes $X$ and $Y$ are said to share the same finite-dimensional distributions if, for every integer $n \geq 1$, any times $0 \leq t_1 < t_2 < \cdots < t_n < \infty$, and any measurable set $A \in \mathcal{B}(\mathbb{R}^n)$, the following equality holds:

$$P[(X_{t_1}, \ldots, X_{t_n}) \in A] = P'[(Y_{t_1}, \ldots, Y_{t_n}) \in A]. \tag{2.24}$$

### 2.4.1 Filtration and Martingales

A *filtration* is defined as a sequence of sub-$\sigma$-algebras $(\mathcal{F}_t)_{t \in T}$ that is increasing, meaning that if $s \leq t$, then $\mathcal{F}_s \subseteq \mathcal{F}_t$. The index set $T$ is usually either $\mathbb{N}$ or $\mathbb{R}_+$. The $\sigma$-algebra $\mathcal{F}_t$ corresponds to the information accessible at time $t$. A stochastic process $X = (X(t))_{t \in T}$ defined on the sample space $\Omega$ is termed *adapted* to the filtration $(\mathcal{F}_t)_{t \in T}$ if $X(t) : \Omega \to \mathbb{R}$ is $\mathcal{F}_t$-measurable for every $t$ in $T$. The *natural filtration* of a process $X = (X(t))_{t \in T}$ is specified by $\mathcal{F}_t = \sigma(X_s; s \leq t)$, which embodies the information gleaned from observing $X$ from time 0 to $t$. This filtration is denoted as $\mathcal{F}^X$. Therefore, any process $X$ is adapted to its own natural filtration $\mathcal{F}^X$.

**Definition 2.13.**

Given a probability space $(\Omega, \mathcal{F}, P)$, equipped with a filtration $(\mathcal{F}_t)_{t\geq 0}$, a real-valued stochastic process $M = (M(t); t \geq 0)$ is called an $(\mathcal{F}_t)$-*martingale*, or simply, a *martingale*, if it is adapted and integrable, and if

$$\mathbb{E}(M(t)|\mathcal{F}_s) = M(s) \quad \text{a.s., for all } s < t. \tag{2.25}$$

This is a function that remains constant on average in a conditional sense, having no tendency to increase or decrease. In particular,

$$\mathbb{E}[M(t)] = \mathbb{E}[M(0)]. \tag{2.26}$$

**Definition 2.14.**

A process $M = (M(t))_t$ is called a submartingale [resp. supermartingale] if it is adapted and integrable, and if

$$E(M(t) \mid \mathcal{F}_s) \geq M(s) \quad \text{a.s., for all } s < t, \tag{2.27}$$

[resp., if $E(M(t) \mid \mathcal{F}_s) \leq M(s)$ a.s.].

**Proposition 2.10.**

*If $M$ is a martingale and $\Phi$ is a convex function on $\mathbb{R}$ such that $N(t) = \Phi(M(t))$ is integrable, then $N = (N(t); t \leq T)$ is a submartingale.*

## 2.4.2 Stopping Time Theorem

To analyze random functions, it makes sense to introduce random times, as a stopping time represents a decision made using the information available up to that moment. Accordingly, we proceed with the following definition:

**Definition 2.15.**

A random variable $\tau : \Omega \to T \cup \{\infty\}$ is an $\mathcal{F}$-stopping time if

$$\{\tau \leq t\} \in \mathcal{F}_t, \quad \forall t \in T. \tag{2.28}$$

Here are some examples: A constant $\tau = t_0$ is a stopping time, as well as the minimum $\min(\tau_1, \tau_2)$ of two stopping times—and thus $\min(\tau_1, t_0)$—as well as $\inf_n \tau_n$ and $\sup_n \tau_n$ when the $\tau_n$ are stopping times. Finally, the hitting time of a closed set $F$ by a continuous process $X$ is

$$\tau_F = \inf\{t \geq 0 : X(t) \in F\}, \tag{2.29}$$

which is a stopping time when $X$ is $\mathcal{F}$-adapted. Indeed, by the continuity of trajectories,

$$\{\tau_F \leq t\} = \bigcup_{s\in[0,t]} \{X(s) \in F\} = [\bigcap_{k\geq 1} \bigcup_{s\in Q\cap[0,t]} \{\mathrm{dist}(X(s), F) \leq 1/k\}] \bigcup \{X(t) \in F\}. \quad (2.30)$$

which is an element of $\mathcal{F}_t$.

For $X$ a continuous random function and $\tau$ a stopping time that is almost surely finite, we consider the random variable

$$X(\tau) : \omega \to X(\tau(\omega), \omega).$$

In the case $T = \mathbb{N}$, it is easy to see that $X(\tau)$ is a random variable, obtained by a measurable pasting of random variables. In the case $T = \mathbb{R}_+$, it is less immediate: we can approximate $\tau$ by stopping times $\tau_n$ taking countable values,

$$\tau_n = \frac{k+1}{n} \quad \text{if} \quad \tau \in \left[\frac{k}{n}, \frac{k+1}{n}\right), \quad \tau_n = \infty \quad \text{if} \quad \tau = \infty, \quad (2.31)$$

and note that $X(\tau) = \lim_{n\to\infty} X(\tau_n)$.

Think of a martingale $M$ as the player's gain in a fair game, and a stopping time $\tau$ as the moment when the player decides to stop playing. Unless there is foresight of the future (or insider knowledge), the stopping strategy should not turn the fair game into a favorable or unfavorable one. This is what the following theorem asserts, called the stopping theorem or sampling theorem, where the fixed time $t$ in Definition 2.13 is replaced by a bounded stopping time:

**Theorem 2.11 ([82]).**
*Let $M = (M(t))_t$ be a continuous $\mathcal{F}$-martingale, and let $\tau$ be a bounded $\mathcal{F}$-stopping time with $0 \leq s \leq \tau \leq Cst < \infty$. Then,*

$$\mathbb{E}[M(\tau)|\mathcal{F}_s] = M(s). \quad (2.32)$$

*If $S$ is a continuous $\mathcal{F}$-supermartingale, then we have $\mathbb{E}[S(\tau)|\mathcal{F}_s] \leq S(s)$.*

Consequently, we obtain the very useful property:

**Properties 2.12.**

$$\mathbb{E}M(\tau) = \mathbb{E}M(0), \quad (2.33)$$

which implies that the expected gain remains unchanged under a valid stopping strategy, as indicated above.

Additionally, note that this result cannot hold without a condition on $\tau$. For instance, the hitting time $T_b$ of the point $b$,

$$T_b = \inf\{t \geq 0 : B(t) = b\},$$

is an almost surely finite stopping time (see the remark below), and $B(T_b) = b$. Therefore, $\mathbb{E}B(T_b) = b \neq \mathbb{E}B(0) = 0$.

The theorem does not apply here—$\tau$ is unbounded—but it does imply that

$$\mathbb{E}B(T_b \wedge n) = 0 \quad \text{for all } n, \tag{2.34}$$

or equivalently,

$$bP(T_b \leq n) + \mathbb{E}(B(n)\mathbf{1}_{\{T_b > n\}}) = 0. \tag{2.35}$$

**Corollary 2.13 (Stopping of Bounded Martingales).**
*Let $M$ be a continuous martingale, and let $\tau$ be a stopping time that is almost surely finite, such that*

$$|M(\tau \wedge n)| \leq K \quad \forall n \tag{2.36}$$

*for a constant $K < \infty$. Then,*

$$\mathbb{E}M(\tau) = \mathbb{E}M(0). \tag{2.37}$$

**Theorem 2.14 (Strong law of large numbers [86]).**
*Let $M = \{M_t\}_{t\geq 0}$ be a real-valued continuous local martingale vanishing at $t = 0$. Then*

$$\lim_{t\to\infty} (M,M)_t = \infty \quad a.s. \implies \lim_{t\to\infty} \frac{M_t}{(M,M)_t} = 0 \quad a.s. \tag{2.38}$$

*and also*

$$\lim_{t\to\infty} \frac{(M,M)_t}{t} < \infty \quad a.s. \implies \lim_{t\to\infty} \frac{M_t}{t} = 0 \quad a.s. \tag{2.39}$$

## 2.5 Brownian Motion

Brownian motion is a stochastic process that models the random movement of particles suspended in a fluid, which was first observed by botanist Robert Brown in 1827. It is a fundamental concept in probability theory and serves as a key model for continuous-time random processes. Brownian motion has several important properties: it starts at zero, has independent and normally distributed increments, and

exhibits continuous paths that are nowhere differentiable. This process is widely used to model phenomena in physics, finance, and various branches of mathematics, especially when the system evolves under uncertainty and randomness. In epidemiology, Brownian motion can be used to describe the random fluctuations in the spread of diseases or other dynamic processes.

The concepts, definitions, and theoretical foundations discussed in this section have been drawn and adapted from the references [82, 84].

**Definition 2.16.**

A *Brownian motion* (in $d$ dimensions, starting from 0) is a family $(B_t)_{t\in\mathbb{R}_+}$ of random variables taking values in $\mathbb{R}^d$, defined on a probability space $(\Omega, \mathcal{F}, P)$, that satisfies:

(P1) We have $B_0 = 0$ almost surely. Moreover, for any integer $p \geq 1$ and real numbers $0 = t_0 < t_1 < \cdots < t_p$, the random variables $B_{t_1} - B_{t_0}, B_{t_2} - B_{t_1}, \ldots, B_{t_p} - B_{t_{p-1}}$ are independent. Additionally, for each $j \in \{1, \ldots, p\}$, $B_{t_j} - B_{t_{j-1}}$ is a centered Gaussian vector with covariance $(t_j - t_{j-1})I_d$.

(P2) For every $\omega \in \Omega$, the function $t \mapsto B_t(\omega)$ is continuous.

**Theorem 2.15.**

*The Brownian motion exists. In other words, it is possible to construct on a suitable probability space a family $(B_t)_{t\in\mathbb{R}_+}$ of random variables that satisfies properties (P1) and (P2).*

### 2.5.1 Characteristics of Brownian Motion Sample Paths

In this section, we explore the characteristics of sample paths of Brownian motion (see Figure 2.1). Let $(B_t)_{t\geq 0}$ be a fixed Brownian motion. For each $t \geq 0$, we define

$$\mathcal{F}_t = \sigma(B_s : s \leq t),$$

where $\mathcal{F}_t$ is the sigma-algebra generated by $B_s$ for $s$ up to time $t$.

Observe that $\mathcal{F}_s \subseteq \mathcal{F}_t$ whenever $s \leq t$. Additionally, we define

$$\mathcal{F}_0^+ = \bigcap_{s>0} \mathcal{F}_s.$$

We proceed by introducing a fundamental 0-1 law.

**Theorem 2.16.**

*(Blumenthal's 0-1 Law) The $\sigma$-algebra $\mathcal{F}_0^+$ is trivial, meaning that $P(A) = 0$ or $1$ for every $A \in \mathcal{F}_0^+$.*

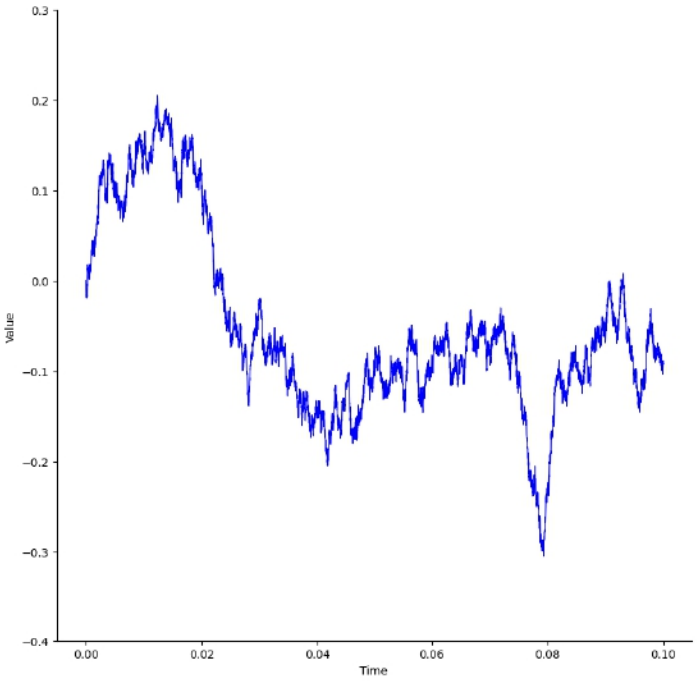



Figure 2.1: Sample Path Simulation of Brownian Motion Over the Interval $[0, 1]$.

**Proposition 2.17** ([82])**.**

- *Almost surely, for any* $\varepsilon > 0$,

$$\sup_{0 \le s \le \varepsilon} B_s > 0, \qquad \inf_{0 \le s \le \varepsilon} B_s < 0. \tag{2.40}$$

- *For each* $a \in \mathbb{R}$, *define* $T_a = \inf\{t \ge 0 : B_t = a\}$ *(where we take* $\inf \emptyset = \infty$ *by convention). Then,*

$$\text{almost surely}, \quad \forall a \in \mathbb{R}, \quad T_a < \infty. \tag{2.41}$$

*As a result, we have almost surely*

$$\limsup_{t \to \infty} B_t = +\infty, \qquad \liminf_{t \to \infty} B_t = -\infty. \tag{2.42}$$

**Corollary 2.18.**

*With probability one, the function* $t \mapsto B_t$ *is not monotonic on any non-trivial interval.*

**Corollary 2.19.**

*With probability one, the function* $t \mapsto B_t$ *has infinite variation over any non-trivial interval.*

## 2.6 Stochastic Integration

Stochastic integration is a mathematical framework that extends traditional integration to processes that involve randomness, such as stochastic processes. Unlike deterministic integrals, stochastic integrals are used to model systems where the integrand or the limits of integration are random, often described by processes like

Brownian motion. The most common form of stochastic integration is the Itô integral, which is designed to handle the irregular nature of stochastic processes, particularly their discontinuous or non-differentiable paths. Stochastic integration is a key tool in the analysis of dynamic systems under uncertainty, providing a rigorous way to model and solve problems in areas like finance, physics, and epidemiology, where randomness plays a crucial role in the evolution of systems over time.
The definitions, properties, and theories outlined in this section are derived from and informed by the works cited in references [87, 88].

### 2.6.1 Filtrations and Measurability

**Definition 2.17.**

Given a filtration $\mathcal{F} = (\mathcal{F}_t, t \geq 0)$ on $(\Omega, \mathcal{A}, P)$, a Brownian motion $B$ defined on $(\Omega, \mathcal{A}, P)$ is called an $\mathcal{F}$-Brownian motion if it is adapted to $\mathcal{F}$, and if $B(t) - B(s)$ is independent of $\mathcal{F}_s$ for $0 \leq s \leq t$.

**Definition 2.18.**

A real function $\varphi(t, \omega)$ on $\mathbb{R}^+ \times \Omega$ [respectively, $[0, T] \times \Omega$] is said to be progressively measurable if for all $t \in \mathbb{R}^+$ [respectively, $t \in [0, T]$], the mapping $(s, \omega) \mapsto \varphi(s, \omega)$ from $[0, t] \times \Omega$ to $\mathbb{R}$ is $\mathcal{B}([0, t]) \otimes \mathcal{F}_t$-measurable.

**Proposition 2.20.**

*Any continuous adapted stochastic function is progressively measurable.*

**Notation 2.21.**

*We denote by $M^2(\mathbb{R}^+)$ [respectively, $M^2([0, T])$] the space of progressively measurable random functions, where :*

$$\mathbb{E} \int_{\mathbb{R}_+} \varphi^2(t, \omega)\, dt < \infty \quad [resp., \mathbb{E} \int_0^T \varphi^2(t, \omega)\, dt < \infty], \quad \text{and } M^2 = \bigcap_T M^2[0, T].$$

### 2.6.2 Stochastic Integral in $M^2(\mathbb{R}^+)$

**Definition 2.19.**

The random variable $\int_{\mathbb{R}^+} \varphi\, dB \in L^2(\Omega, \mathcal{A}, P)$ is called the stochastic integral of $\varphi \in M^2(\mathbb{R}^+)$.

**Theorem 2.22.**

*For $\varphi, \psi$ in $M^2(\mathbb{R}^+)$, we have*

$$\begin{aligned} &\mathbb{E}\left(\int_{\mathbb{R}^+} \varphi(t)\, dB(t)\right) = 0, \quad \mathbb{E}\left(\int_{\mathbb{R}^+} \varphi(t)\, dB(t)\right)^2 = \mathbb{E}\int_{\mathbb{R}^+} \varphi(t)^2\, dt, \\ &\mathbb{E}\left(\int_{\mathbb{R}^+} \varphi(t)\, dB(t)\right)\left(\int_{\mathbb{R}^+} \psi(t)\, dB(t)\right) = \mathbb{E}\int_{\mathbb{R}^+} \varphi(t)\psi(t)\, dt. \end{aligned} \tag{2.43}$$

**Proposition 2.23.**

*The random function $t \mapsto \int_0^t \varphi\, dB$ is mean-square continuous at every point $t_0 \geq 0$, and it has a continuous version on $\mathbb{R}^+$.*

### 2.6.3 Itô's Formula and Itô Process

Any real-valued random function of the form

$$X(t) = X(0) + \int_0^t \varphi(s)\, dB(s) + \int_0^t \psi(s)\, ds \tag{2.44}$$

with $\varphi, \psi \in M^2$ and $X(0) \in L^2(0)$, is called an Itô stochastic process, and we denote its stochastic differential by $dX(s) = \varphi(s)\, dB(s) + \psi(s)\, ds$. Such a representation is unique. The same arguments show that Itô's formula applies to these processes and also to the complex case (by considering the real and imaginary parts separately). For $\Phi \in C^2$,

$$\Phi(X(t)) = \Phi(X(0)) + \int_0^t \Phi'(X(s))\varphi(s)\, dB(s) + \int_0^t \Phi'(X(s))\psi(s)\, ds + \int_0^t \frac{1}{2}\Phi''(X(s))\varphi(s)^2\, ds \tag{2.45}$$

Equation (2.45) can also be expressed in differential notation as

$$d\Phi(X(t)) = \Phi'(X(t))\, dX(t) + \frac{1}{2}\Phi''(X(t))\, d\langle X\rangle(t), \tag{2.46}$$

where we define the quadratic variation $\langle X\rangle$ of the Itô process $X$ by

$$\langle X\rangle(t) = \int_0^t \varphi(s)^2\, ds \tag{2.47}$$

which corresponds to Equation (2.45), the quadratic variation of the martingale part $\int_0^t \varphi(s)\, dB(s)$.

### 2.6.4 General Itô Integral and Formula

**Theorem 2.24.**

*If* $X(t) = X(0) + \int_0^t \varphi(s)\,dB(s) + \int_0^t \psi(s)\,ds$ *is an Itô process, then*

$$\Phi(X(t)) = \Phi(X(0)) + \int_0^t \Phi'(X(s))\varphi(s)\,dB(s) + \int_0^t \Phi'(X(s))\psi(s)\,ds + \int_0^t \frac{1}{2}\Phi''(X(s))\varphi(s)^2\,ds. \tag{2.48}$$

**Lemma 2.25.**

*The Itô formula for Brownian motion:*

$$\Phi(B(t)) = \Phi(B(0)) + \int_0^t \Phi'(B(s))dB(s) + \frac{1}{2}\int_0^t \Phi''(B(s))ds, \tag{2.49}$$

### 2.6.5 Vector Itô Formula

A family $B = (B_j)_{1\le j\le k}$ of independent Brownian motions is called a Brownian motion of dimension $k$. We further define the notion of an $\mathcal{F}$-Brownian motion by Definition 2.17, with $B \in \mathbb{R}^k$. If $\varphi_{i,j} \in M^2_{loc}$, $i \le d, j \le k$, we denote briefly $\varphi = (\varphi_{i,j})_{i,j} \in M^2_{loc}$, and we denote $\int_0^t \varphi(s)dB(s)$ as the process taking values in $\mathbb{R}^d$, whose $i$-th component is given by $\sum_{j=1}^k \int_0^t \varphi_{i,j}(s)dB_j(s)$. Itô's formula extends to the vector case without major difficulty, and we can also consider functions depending on time.

**Theorem 2.26.**

*Let* $\Phi(t,x)$ *be of class* $C^{1,2}$ *on* $\mathbb{R}^+ \times \mathbb{R}^d$, *and let* $\varphi, \psi \in M^2_{loc}$ *with* $\psi \in \mathbb{R}^d$ *and* $\varphi$ *a* $d \times k$ *matrix. For* $X(t) = X(0) + \int_0^t \varphi dB + \int_0^t \psi ds$, *we have*

$$\begin{aligned}\Phi(t, X_t) &= \Phi(0, X_0) + \int_0^t \frac{\partial}{\partial t}\Phi(s, X_s)\,ds + \sum_{i=1}^d \int_0^t \frac{\partial}{\partial x_i}\Phi(s, X_s)\psi_i(s)\,ds \\ &+ \sum_{i=1}^d \int_0^t \frac{\partial}{\partial x_i}\Phi(s, X_s)\sum_{j=1}^k \varphi_{i,j}(s)dB_j(s) \\ &+ \frac{1}{2}\int_0^t \sum_{i,j=1}^d \frac{\partial^2}{\partial x_i \partial x_j}\Phi(s, X_s)\sum_{j=1}^k \varphi_{i,j}(s)\varphi_{j,j}(s)\,ds.\end{aligned} \tag{2.50}$$

*In differential form, we have*

$$d\Phi(t, X_t) = \frac{\partial}{\partial t}\Phi dt + \nabla\Phi \cdot dX_t + \frac{1}{2}tr(\Phi''\varphi\varphi^*)dt, \tag{2.51}$$

*where* $\nabla$ *is the gradient and* $\Phi'' = \left(\frac{\partial^2}{\partial x_i \partial x_j}\Phi\right)_{i,j}$ *is the* $d \times d$ *matrix of second-order partial derivatives.*

## 2.7 Stochastic Differential Equations

Stochastic differential equations (SDEs) are differential equations that involve random processes, providing a way to model systems where the evolution over time is influenced by both deterministic forces and random fluctuations. These equations extend classical ordinary differential equations by incorporating terms that represent randomness, typically modeled by Brownian motion or other stochastic processes. SDEs are essential for describing a wide range of phenomena, from the behavior of financial markets to the spread of diseases in epidemiological models. Solving an SDE often requires specialized techniques, such as Itô's Lemma and stochastic integration, due to the presence of randomness. The solutions to these equations are themselves random processes, making SDEs a powerful tool for understanding complex systems driven by uncertainty.
The definitions, properties, and theories outlined in this section are derived from and informed by the works cited in references [89, 90, 91, 92, 94, 95].

**Definition 2.20.**
Let $\sigma : \mathbb{R} \to \mathbb{R}^+$ and $b : \mathbb{R} \to \mathbb{R}$ be given functions. Let $(B(t))_{t\geq 0}$ be a Brownian motion and $X_0$ be a random variable defined on the same probability space $(\Omega, \mathcal{A}, \mathbb{P})$, where $X_0$ and $B$ are independent.

A stochastic differential equation (SDE) is an equation of the form

$$dX(t) = b(X(t))\,dt + \sigma(X(t))\,dB(t), \quad X(0) = X_0, \tag{2.52}$$

where the function $b$ is called the *drift coefficient* and $\sigma$ is called the *diffusion coefficient*.

The solution of a stochastic differential equation is called a *diffusion process* (or simply a diffusion).

Let $\mathcal{F}_t$ denote the filtration generated by $(B(s))_{s\leq t}$ and $X_0$, completed with respect to the $\mathbb{P}$-null sets.

### 2.7.1 Existence and Uniqueness of Strong Solutions

We present the fundamental results ensuring the existence and uniqueness of strong solutions to stochastic differential equations under standard Lipschitz and growth conditions.

**Theorem 2.27 ([90]).**

*Suppose $X_0 \in L^2$, and $b$ and $\sigma$ are Lipschitz continuous, i.e.,*

$$|b(x) - b(y)| + |\sigma(x) - \sigma(y)| \leq K|x - y|. \tag{2.53}$$

*Then, the equation (2.52) has a unique solution $X \in M^2$.*

**Theorem 2.28 ([90]).**

*Suppose the coefficients $b$ and $\sigma$ are globally Lipschitz and grow at most linearly in the space variable,*

$$|b(t,x) - b(t,y)| + |\sigma(t,x) - \sigma(t,y)| \leq K|x - y|, \tag{2.54}$$

$$|b(t,x)|^2 + |\sigma(t,x)|^2 \leq K^2(1 + |x|^2), \tag{2.55}$$

*for $t \geq 0$, $x, y \in \mathbb{R}^d$, with $K$ being a finite constant. Then, for $X_0 \in L^2(\mathcal{F}_0)$, the equation (2.52) has a unique solution $X \in M^2$.*

**Corollary 2.29 (One-dimensional Stochastic Differential Equation Comparison Theorem).**

*Consider the following stochastic processes:*

1. *two continuous, $(F_t)$-adapted real-valued processes, $x_1(t,\omega)$ and $x_2(t,\omega)$;*
2. *a one-dimensional $(F_t)$-Brownian motion $B(t,\omega)$ such that $B(0) = 0$ almost surely;*
3. *two real-valued $(F_t)$-adapted, measurable processes $\beta_1(t,\omega)$ and $\beta_2(t,\omega)$.*

*Assume that the following conditions hold almost surely:*

$$\begin{aligned}
&(i) \quad \int_0^t \sigma(s, x_1(s))dB(s) + \int_0^t \beta_1(s)ds, \quad i = 1, 2,\\
&(ii) \quad x_1(0) \leq x_2(0),\\
&(iii) \quad \beta_1(t) \leq b_1(t, x_1(t)) \quad \text{for all } t \geq 0,\\
&(iiii) \quad \beta_2(t) \geq b_2(t, x_2(t)) \quad \text{for all } t \geq 0.
\end{aligned}$$

*Under these conditions, it follows with probability one that*

$$x_1(t) \leq x_2(t) \quad \text{for all } t \geq 0. \tag{2.56}$$

**Lemma 2.30 (Fatou's Lemma).**

*The classical form of Fatou's Lemma states that:*

$$int_{\Omega} \liminf_{n\to\infty} f_n \leq \liminf_{n\to\infty} \int_{\Omega} f_n, \tag{2.57}$$

*where $f_n$ is a sequence of nonnegative measurable functions on the measure space $(\Omega, \mathcal{F}, \mathbb{P})$, and it holds that $\int_{\Omega} \liminf_{n\to\infty} f_n < \infty$.*

### 2.7.2 The Fokker-Planck Equation

The Fokker–Planck equation is a partial differential equation that governs the time evolution of the probability density function associated with a stochastic process. When a dynamical system is described by a set of differential equations containing at least one stochastic component, the system's behavior—incorporating both deterministic and random influences—can be expressed as

$$dX_t = f(X_t, t)dt + \sigma(X_t, t)dB_t, \tag{2.58}$$

where $X_t$ represents the $n$-dimensional random state vector, $f$ denotes the deterministic force vector, and $B_t$ corresponds to an $m$-dimensional standard Brownian motion.

**Definition 2.21.**

The Fokker–Planck equation characterizes the temporal evolution of the probability density function of the stochastic variable $X_t$, particularly in systems influenced by both drift and diffusion effects. Originally formulated by Fokker and Planck to study Brownian motion, the equation takes the form

$$\frac{\partial p(x,t)}{\partial t} = -\sum_{i=1}^{n} \frac{\partial}{\partial x_i}\left(f_i(x,t)p(x,t)\right) + \sum_{i=1}^{n}\sum_{j=1}^{n} \frac{\partial^2}{\partial x_i \partial x_j}\Big(D_{ij}(x,t)p(x,t)\Big), \tag{2.59}$$

where $\mathbf{f} = [f_1, \ldots, f_n]^T$ represents the deterministic force vector, and $D = \frac{1}{2}\sigma\sigma^T$ is the diffusion tensor, with $\sigma$ being an $n \times n$ matrix that encapsulates the diffusion dynamics within the system.

### 2.7.3 Langevin Equation

Let $(\Omega, \mathcal{F}, P)$ be a probability space. First, let us give the definition of the Ornstein-Uhlenbeck process.

**Definition 2.22.**

We define the Langevin equation

$$\begin{cases} dX_t = (\phi - kX_t)\,dt + \sqrt{\beta}dB_t, & t \geq 0 \\ X_0 \sim \mu \end{cases} \tag{2.60}$$

where $(B_t)_{t\geq 0}$ is a standard Brownian motion and $\mu$ is a probability distribution with real values. The parameter $\phi$ is real, and the parameters $k$ and $\beta$ are positive real numbers. The variables $X_0$ and $(B_t)_{t\geq 0}$ are independent.

**Proposition 2.31.**

*The unique strong solution of the Langevin equation (2.60) starting from $X_0$ is given by the Ornstein-Uhlenbeck process, defined by*

$$X_t := X_0 e^{-kt} + \frac{\phi}{k}\left(1 - e^{-kt}\right) + \sqrt{\beta}\int_0^t e^{k(u-t)}\,dB_u, \qquad t \geq 0. \tag{2.61}$$

This process was introduced by Ornstein and Uhlenbeck in 1930 to describe the motion of a particle with friction . A trajectory of this process is shown in Figure 2.2.

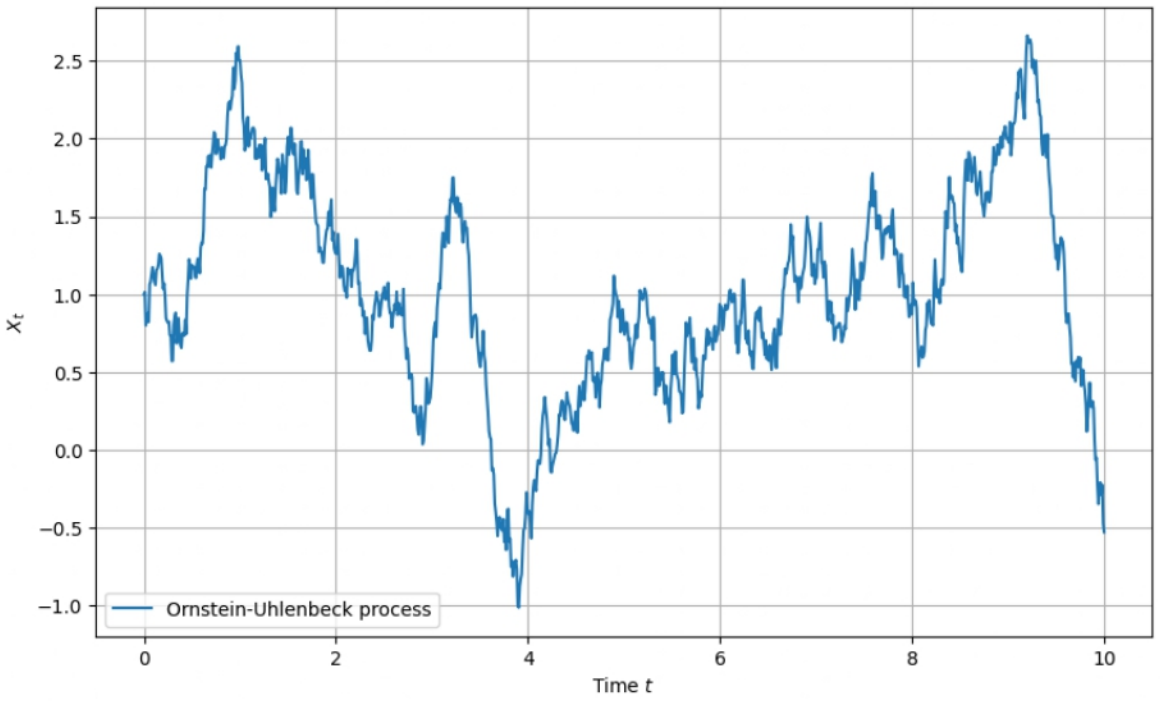



Figure 2.2: Trajectory of an Ornstein-Uhlenbeck Process with $\phi = 1$, $k = 1$, $\beta = 1$, $X_0 = 1$.

**Remark 2.2.**

The process can be represented as an affine transformation of a time-changed Brownian motion. For $t \in \mathbb{R}_+$, we have:

$$X_t = X_0 e^{-kt} + \frac{\phi}{k}\left(1 - e^{-kt}\right) + \sqrt{\frac{\beta}{2k}}\tilde{B}_{e^{2kt}-1}e^{-kt}, \tag{2.62}$$

where $(\tilde{B}_t)_{t\geq 0}$ is a standard Brownian motion.

**Proposition 2.32.**

*For every $t \in \mathbb{R}_+$, we have:*

$$\begin{aligned} \mathbb{E}[X_t] &= \frac{\phi}{k} + e^{-kt}\left(\mathbb{E}[X_0] - \frac{\phi}{k}\right), \\ Var(X_t) &= \frac{\beta}{2k} + \left(Var(X_0) - \frac{\beta}{2k}\right)e^{-2kt}, \\ Cov(X_s, X_t) &= \left[Var(X_0) + \frac{\beta}{2k}(e^{-2k(t\wedge s)} - 1)\right]e^{-k(t+s)}. \end{aligned} \tag{2.63}$$

**Remark 2.3.**

If $\mu = \frac{\phi}{k}$ and $\sigma^2 = \frac{\beta}{2k}$, then $(X_t)_{t\geq 0}$ is a stationary Gaussian process with the same expectation and variance.

**Remark 2.4.**

By a simple transformation, the model can be written as:

$$\begin{cases} d\tilde{X}_t = -\tilde{k}\tilde{X}_t dt + d\tilde{B}_t, & t \geq 0, \\ \tilde{X}_0 = X_0 - \frac{\phi}{k} \end{cases} \tag{2.64}$$

where $\tilde{X}_t := X_{\frac{t}{\beta}} - \frac{\phi}{k}, \tilde{k} := \frac{k}{\beta}$, and $\tilde{B}_t := \sqrt{\beta}B_{\frac{t}{\beta}}$ (which is still a Brownian motion).

## 2.8 Existence of a Stationary Distribution

The existence of a stationary distribution refers to the concept where a stochastic process, particularly a Markov process, reaches a state where its probability distribution no longer changes over time. In other words, once the system has reached this stationary distribution, the future behavior of the process becomes independent of its initial state, and the process remains in equilibrium. This property is crucial in many applications, such as the study of disease spread in epidemiology, where we want to understand the long-term behavior of a population. The existence

of a stationary distribution is often tied to specific conditions, such as irreducibility, aperiodicity, and positive recurrence of the underlying Markov chain. Proving the existence of a stationary distribution requires sophisticated mathematical techniques, including the use of Lyapunov functions and fixed-point theorems.
The concepts, definitions, and theoretical foundations discussed in this section have been drawn and adapted from the references [86, 93, 118].

### 2.8.1 Markov Property

Let $(\Omega, \mathcal{A}, \mathbb{P})$ be a probability space, $\mathcal{F} = (\mathcal{F}(t), t \geq 0)$ a filtration on this space, and $X = (X(t); t \geq 0)$ a process adapted to $\mathcal{F}$, which we will assume takes values in $\mathbb{R}^d$ (with $d \geq 1$) for simplicity.

**Definition 2.23.**
The random function $X$ is a Markov process (with respect to $\mathcal{F}$) if for all $t \geq 0$ and any Borel set $A \subset \mathbb{R}^d$,

$$P(X(t+s) \in A | \mathcal{F}_t) = P(X(t+s) \in A | X(t)). \tag{2.65}$$

This last equality is called the Markov property. It means that the law of $X(t+s)$ given $\mathcal{F}_t$ depends only on $X(t)$: the law of the future depends on the past only through the present. In other words, the future and the past are conditionally independent given the present state $X(t)$.

Consider a homogeneous Markov process in $\mathbb{R}^d$ represented by the stochastic differential equation:

$$dX(t) = f(X(t))dt + g(X(t))dB(t), \tag{2.66}$$

where the initial value $X(0) \in \mathbb{R}^d$, and $B(t)$ denotes a $d$-dimensional Brownian motion on a complete probability space $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t\geq 0}, \mathbb{P})$. The functions $f : \mathbb{R}^d \to \mathbb{R}^d$ and $g : \mathbb{R}^d \to \mathbb{R}^{d\times m}$ are assumed to be Borel measurable. The lemma below addresses conditions for the existence of a stationary distribution.

**Lemma 2.33 (Refer to Lemma A.4, page 16 in [93]).**
*Assume that there exists a bounded, closed domain $A \subset \mathbb{R}^d$ with a regular boundary $\Gamma$. For any initial condition $X(0) \in \mathbb{R}^d$, if*

$$\liminf_{t\to\infty} \frac{1}{t} \int_0^t \mathbb{P}(s, X(s), A)\, ds > 0 \quad \textit{almost surely}, \tag{2.67}$$

*where $\mathbb{P}(s, X(s), \cdot)$ represents the transition probability of $X(t)$, then system (A.2) has a solution with the Feller property, and it supports at least one stationary distribution $\varpi(\cdot)$ on $\mathbb{R}^d$.*

**Definition 2.24 (Feller Property).**
Let $\mathbb{E}$ be a locally compact, Hausdorff space (LCH space). A transition function $\{\mathcal{P}_t\}_{t\geq 0}$ is said to have the Feller property if, for every $f \in C_0(\mathbb{E})$,

- $\mathcal{P}_t f \in C_0(\mathbb{E})$,
- the map $t \mapsto \mathcal{P}_t f$ is continuous in the norm topology of $C_0(\mathbb{E})$,
- $\mathcal{P}_0 f = f$.

A Markov process $X$ with a Feller transition function is called a Feller process.

**Theorem 2.34 ([118] Perron–Frobenius theorem).**
*Let $B \in \mathbb{R}^{n\times n}$ be a non-negative and irreducible matrix. Then:*

- *The matrix $B$ has a positive real eigenvalue $\rho(B)$, called the spectral radius of $B$, such that $\rho(B)$ is a simple eigenvalue.*
- *There exists a strictly positive left eigenvector $\vartheta = (\vartheta_1, \ldots, \vartheta_n)$ such that*

$$\vartheta B = \rho(B)\, \vartheta.$$

- *The spectral radius $\rho(B)$ dominates all other eigenvalues in modulus, i.e.,*

$$|\lambda| \leq \rho(B) \quad \text{for all eigenvalues } \lambda \text{ of } B.$$

Chapter 3

# Stochastic Dynamics of an $SEI_uI_rR$ Epidemic Model Driven by an Ornstein–Uhlenbeck Process

## 3.1 Introduction

The COVID-19 pandemic, driven by the novel coronavirus, has underscored the critical role of mathematical modeling in understanding infectious disease dynamics, devising effective mitigation strategies, and forecasting future epidemiological trends[1, 2, 4, 5, 6, 96, 97, 98]. While early models predominantly employed deterministic frameworks to simulate the spread of the virus, the complexity of real-world interactions and the stochastic nature of human behavior highlighted the limitations of such approaches. Key factors such as individual variability in susceptibility, heterogeneous contact patterns, and the unpredictability of viral mutations necessitated a paradigm shift toward models that account for randomness.

This shift has catalyzed the integration of stochastic processes and probabilistic methodologies into COVID-19 modeling, enabling researchers to better capture the inherent uncertainties and unpredictability of epidemic progression. This chapter examines the interplay between deterministic and stochastic approaches in modeling the spread of COVID-19. Specifically, it addresses the mechanisms by which stochasticity is incorporated into epidemiological models, evaluates the advantages of these approaches in enhancing model robustness and uncertainty quantification, and discusses the computational and interpretive challenges they introduce. By exploring this synthesis of analytical methodologies, we aim to provide a nuanced perspective on the dynamic and multifaceted nature of COVID-19 transmission modeling[99, 100, 101, 102, 103]. Notably, Gatyeni et al. proposed the $SEI_uI_rR$ model, which serves as a foundation for the subsequent analysis presented in this work.

$$\begin{cases} \dfrac{dS(t)}{dt} = \Delta - \beta\left(v_1 I_u + v_2 I_r\right) S - \mu S, \\ \dfrac{dE(t)}{dt} = \beta\left(v_1 I_u + v_2 I_r\right) S - (\sigma + \mu) E, \\ \dfrac{dI_u(t)}{dt} = \sigma(1-\rho)E - \left(\mu + d_1 + \delta + \gamma_{I_u}\right) I_u, \\ \dfrac{dI_r(t)}{dt} = \sigma\rho E + \delta I_u - \left(\mu + d_2 + \gamma_{I_r}\right) I_r, \\ \dfrac{dR(t)}{dt} = \gamma_{I_u} I_u + \gamma_{I_r} I_r - \mu R, \end{cases} \tag{3.1}$$

where $S(t)$ denotes the susceptible population, $E(t)$ represents exposed individuals, $I_u(t)$ represents undetected infected individuals, $I_r(t)$ represents detected (reported) infected individuals, and $R(t)$ denotes the recovered individuals.

Table 3.1: Description of state parameters of the model (3.1) .

| Parameter | Description |
|---|---|
| $\Delta$ | Birth rate |
| $\mu$ | Natural death rate |
| $\beta$ | Disease transmission rate |
| $\rho$ | Proportion of $E$ which becomes undiagnosed |
| $v_1$ | Transmissibility relative to undetected people |
| $v_2$ | Transmissibility relative to detected people |
| $\delta$ | The transmission rate at which undetected people become detected |
| $d_1$ | Disease related death rate in $I_u$ compartment |
| $d_2$ | Disease related s death rate in $I_r$ compartment |
| $\sigma$ | Incubation rate Fitted |
| $\gamma_{I_u}$ | Recovery rate of people in $I_u$ compartment |
| $\gamma_{I_r}$ | Recovery rate of people in $I_r$ compartment |

Throughout this work, we denote the time-dependent state variables of the system by $S(t), E(t), I_u(t), I_r(t), R(t)$. For simplicity of notation, we sometimes write $(S, E, I_u, I_r, R)$ instead of $(S(t), E(t), I_u(t), I_r(t), R(t))$ when no ambiguity arises.

Assuming all parameters are constant and positive, their respective descriptions are provided in Table 3.1. It is feasible to achieve that system (3.1) possesses a positive invariant region [104, 105]:

$$\Omega = \left\{(S, E, I_u, I_r, R) \in \mathbb{R}^5_+, 0 \leq S + E + I_u + I_r + R \leq \frac{\Delta}{\mu}\right\}.$$

The expression for the basic reproduction number of system (3.1) is provided as follows:

$$\mathcal{R}_0 = \mathcal{R}_1 + \mathcal{R}_2 + \mathcal{R}_3,$$

where

$$\mathcal{R}_1 = \frac{\sigma\beta S_0 v_1(1-\rho)}{(\sigma+\mu)\left(\delta+\gamma_{I_u}+\mu+d_1\right)}, \quad \mathcal{R}_2 = \frac{\sigma\beta S_0 v_2\rho}{(\sigma+\mu)\left(\gamma_{I_r}+\mu+d_2\right)},$$
$$\mathcal{R}_3 = \frac{\sigma\beta S_0 v_2(1-\rho)\delta}{(\sigma+\mu)\left(\delta+\gamma_{I_u}+\mu+d_1\right)\left(\gamma_{I_r}+\mu+d_2\right)},$$

which plays a crucial role in determining the occurrence of the disease, in a situation where $S_0 = \frac{\Delta}{\mu}$.

The threshold dynamics of system (3.1) can be characterized as follows:

- When $\mathcal{R}_0 < 1$, the system (3.1) admits a disease-free equilibrium $E(S_0, 0, 0, 0, 0) = \left(\frac{\Delta}{\mu}, 0, 0, 0, 0\right)$, which is locally asymptotically stable within the region $\Omega$.
- Conversely, when $\mathcal{R}_0 \geq 1$, the system (3.1) possesses a globally asymptotically stable endemic equilibrium $X^* = (S^*, E^*, I_u^*, I_r^*, R^*)$ within the region $\Omega$.

Recent years have witnessed growing interest among researchers in exploring the dynamic behavior of epidemiological models under stochastic perturbations. The adoption of stochastic modeling techniques has been recognized as a more precise approach to capturing the complexities of infectious disease transmission [99, 106, 107].

To date, several methodologies have been developed to incorporate stochastic perturbations into deterministic frameworks. A widely employed approach assumes that system parameters evolve according to an Itô process, specifically the Ornstein-Uhlenbeck process [102, 108, 109, 110]. Liu et al. [111], for instance, proposed a stochastic logistic model that integrates diffusion and Ornstein-Uhlenbeck processes to account for randomness and fluctuations.

Building upon these insights, this study introduces stochastic perturbations to the transmission rate $\delta$ the rate at which undetected individuals become detected by employing the Ornstein-Uhlenbeck process. This choice reflects real-world scenarios where $\delta$ may fluctuate due to variations in testing capacity, public health interventions, or behavioral changes within the population. Modeling $\delta$ as a stochastic process enables the framework to capture such temporal variations, described as follows:

$$d\delta(t) = \rho_1[\bar{\delta} - \delta(t)]dt + \sigma_1 dB(t),$$

where $\bar{\delta}$ represents the long-term mean level of the transmission rate $\delta$, and $\rho_1$ denotes the speed of reversion. Here, $B(t)$ is a standard Brownian motion defined on a complete probability space $\left(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t\geq 0}, \mathbf{P}\right)$ with a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ [112]. The parameter $\sigma_1$ captures the intensity of $B(t)$.

To ensure the positivity of the stochastic model, which is critical in practical scenarios, a nonnegative formulation is adopted. Specifically, the variable $\max\{\delta(t), 0\}$ is employed in place of $\delta(t)$ as suggested by Liu et al. [101]. Consequently, the

resulting stochastic model is articulated as follows:

$$\begin{cases} dS(t) = [\Delta - \beta(v_1 I_u + v_2 I_r) S - \mu S]dt, \\ dE(t) = [\beta(v_1 I_u + v_2 I_r) S - (\sigma + \mu)E]dt, \\ dI_u(t) = [\sigma(1-\rho)E - \left(\mu + d_1 + \max\{\delta(t), 0\} + \gamma_{I_u}\right) I_u]dt, \\ dI_r(t) = [\sigma\rho E + \max\{\delta(t), 0\} I_u - \left(\mu + d_2 + \gamma_{I_r}\right) I_r]dt, \\ dR(t) = [\gamma_{I_u} I_u + \gamma_{I_r} I_r - \mu R]dt, \\ d\delta(t) = \rho_1[\bar{\delta} - \delta(t)]dt + \sigma_1 dB(t). \end{cases} \tag{3.2}$$

## 3.2 Existence and Uniqueness of the Global Solution

To analyze the properties of the proposed epidemic system, it is imperative to first confirm the existence and uniqueness of a global solution. This ensures the validity of the model for all time $t \geq 0$. The following theorem establishes these foundational properties for system (3.2) under any given initial conditions.

**Theorem 3.1.**
*For any initial condition* $(S(0), E(0), I_u(0), I_r(0), R(0), \delta(0))^T \in \mathbb{R}^5_+ \times \mathbb{R}$, *there exists a unique solution* $(S(t), E(t), I_u(t), I_r(t), R(t), \delta(t))^T$ *to system* (3.2) *defined for all* $t \geq 0$. *Furthermore, this solution remains in the domain* $\mathbb{R}^5_+ \times \mathbb{R}$ *almost surely (a.s.).*

**Proof.** Since that all the coefficients of system (3.2) satisfy the local Lipschitz conditions, there will exist a unique local solution $(S(t), E(t), I_u(t), I_r(t), R(t), \delta(t))^T$ on the interval $[0, \rho_e)$ for any initial value $(S(0), E(0), I_u(0), I_r(0), R(0), \delta(0))^T \in \mathbb{R}^5_+ \times \mathbb{R}$, where $\rho_e$ denotes the time of explosion [112]. So, to prove that this solution is global, it suffices to prove that: $\rho_e = \infty$ a.s. Letting $D_n = (1/n, n) \times (1/n, n) \times (1/n, n) \times (1/n, n) \times (1/n, n) \times (1/n, n)$, for any $(S(0), E(0), I_u(0), I_r(0), R(0), \delta(0))^T \in \mathbb{R}^5_+ \times \mathbb{R}$, one easily obtains a sufficientl ylarg einteger $j_0$ to satisfy $(S(0), E(0), I_u(0), I_r(0), R(0), \exp\delta(0)) \in D_{j_0}$. In this sense, for each integer $j \geq j_0$, a stopping time set $\rho_j$ is defined by [112]:

$$\begin{aligned} \rho_j = \inf\Bigg\{ & t \in (0, \rho_e) : \min\left\{S(t), E(t), I_u(t), I_r(t), R(t), e^{\delta(t)}\right\} \leq \frac{1}{j} \\ & \text{or} \quad \max\left\{S(t), E(t), I_u(t), I_r(t), R(t), e^{\delta(t)}\right\} \geq j\Bigg\}, \end{aligned}$$

where we assume that $\inf\emptyset = \infty$ (with $\emptyset$ representing the empty set)
and $\rho_\infty = \lim_{j\to\infty} \rho_j$, whence $\rho_\infty \leq \rho_e$ a.s.If $\rho_\infty = \infty$ a.s. is true, then $\rho_e = \infty$ a.s. and

$(S(t), E(t), I_u(t), I_r(t), R(t), \delta(t))^T \in \mathbb{R}^5_+ \times \mathbb{R}$ a.s. for all $t \geq 0$. So we must prove that $\rho_\infty = \infty$ a.s. To prove the latter, we use the proof backwards, assuming that it is false, It means there is a pair of constants $(\varepsilon, T_1) \in ((0,1), \mathbb{R}_+)$ such that :

$$\mathbf{P}\{\rho_\infty \leq T\} > \varepsilon.$$

Consequently, there exists an integer $j_1 \geq j_0$ such that

$$\mathbf{P}\Big\{\rho_j \leq T\Big\} := \mathbf{P}\Big\{\Pi_j\Big\} \geq \varepsilon \qquad \forall j \geq j_1. \tag{3.3}$$

Define the function $U : \mathbb{R}^5_+ \times \mathbb{R} \to \mathbb{R}_+$ by

$$U(S,E,I_u,I_r,R,\delta) = (S-1-\ln S)+(E-1-\ln E)+(I_u-1-\ln I_u)+(I_r-1-\ln I_r)+(R-1-\ln R)+\frac{\delta^2}{2}.$$

Applying the Itô's formula , we obtain

$$dU(S,E,I_u,I_r,R,\delta) = \mathcal{L}U(S,E,I_u,I_r,R,\delta)dt + \sigma_1\delta dB(t), \tag{3.4}$$

where $\mathcal{L}U : \mathbb{R}^5_+ \times \mathbb{R} \to \mathbb{R}_+$ is defined by

$$\begin{aligned}
\mathcal{L}U =& \Delta + 5\mu + \sigma + d_1 + d_2 + \gamma_{I_u} + \gamma_{I_r} + \frac{\sigma_1^2}{2} + \max\{\delta(t),0\} + \beta(v_1 I_u + v_2 I_r) \\
&- \mu S - \mu E - \mu I_u - d_1 I_u - \mu I_r - d_2 I_r - \mu R \\
&- \frac{\Delta}{S} - \frac{\beta(v_1 I_u + v_2 I_r)S}{E} - \frac{\sigma(1-\rho)E}{I_u} - \frac{\sigma\rho E}{I_r} - \frac{\max\{\delta(t),0\}I_u}{I_r} - \frac{\gamma_{I_u} I_u}{R} - \frac{\gamma_{I_r} I_r}{R} \\
&+ \rho_1\bar{\delta}\delta - \rho_1\delta^2 \\
\leq& \Delta + 5\mu + \sigma + d_1 + d_2 + \gamma_{I_u} + \gamma_{I_r} + \frac{\sigma_1^2}{2} + |\delta| + \beta(v_1 I_u + v_2 I_r) + \rho_1\bar{\delta}\delta - \rho_1\delta^2.
\end{aligned} \tag{3.5}$$

Based on model (3.2) we have

$$\begin{aligned}
\frac{d(S+E+I_u+I_r+R)}{dt} =& \Delta - \mu(S+E+I_u+I_r+R) - (d_1 I_u + d_2 I_r) \\
\leq& \Delta - \mu(S+E+I_u+I_r+R).
\end{aligned}$$

Consequently, this suggests that

$$\begin{aligned}
S(t)+E(t)+I_u(t)+I_r(t)+R(t) &\leq \begin{cases} S(0)+E(0)+I_u(0)+I_r(0)+R(0), \\ \text{if } S(0)+E(0)+I_u(0)+I_r(0)+R(0) \geq \dfrac{\Delta}{\mu}, \\ \dfrac{\Delta}{\mu}, \text{ if } S(0)+E(0)+I_u(0)+I_r(0)+R(0) < \dfrac{\Delta}{\mu} \end{cases} \\
&\leq \Upsilon_1,
\end{aligned} \tag{3.6}$$

where

$$\Upsilon_1 := \sup\left\{S(0)+E(0)+I_u(0)+I_r(0)+R(0), \frac{\Delta}{\mu}\right\}.$$

Substiiuting (3.6) into (3.5) tead to that

$$\mathcal{L}U \le \Delta + 5\mu + \sigma + d_1 + d_2 + \gamma_{I_u} + \gamma_{I_r} + \frac{\sigma_1^2}{2} + \beta(v_1+v_2)\Upsilon_1 + \sup_{\delta\in\mathbb{R}}\left\{-\rho_1\delta^2 + |\delta| + \rho_1\bar{\delta}\delta\right\} := \Upsilon_2. \tag{3.7}$$

In this case, $\Upsilon_2$ represente a positive constant which is independent of the variables $S, E, I_u, I_r, R$ and $\delta$.

Consequently, by inetegrating both sides of equation (3.4) from 0 to $\rho_j \wedge T$ for any $j \ge j_1$, and then taking the mathematical expectations leads to

$$\begin{aligned}
0 &\le EU\left(S\left(\rho_j\wedge T\right), E\left(\rho_j\wedge T\right), I_u\left(\rho_j\wedge T\right), I_r\left(\rho_j\wedge T\right), R\left(\rho_j\wedge T\right), \delta\left(\rho_j\wedge T\right)\right) \\
&= EU\left(S(0), E(0), I_u(0), I_r(0), R(0), \delta(0)\right) + E\int_0^{\rho_j\wedge T} \mathcal{L}U\left(S(\xi), E(\xi), I_u(\xi), I_r(\xi), R(\xi), \delta(\xi)\right) d\xi \\
&\le EU\left(S(0), E(0), I_u(0), I_r(0), R(0), \delta(0)\right) + \Upsilon_2 T.
\end{aligned} \tag{3.8}$$

Similarly, based on (3.3), it can be deduced that for all $\zeta \in \Pi_j$, at least one of the variables $S, E, I_u, I_r, R$ and $\delta$ must be either $\frac{1}{j}$ or $j$, ensuring that $U\left(S\left(\rho_j,\zeta\right), E\left(\rho_j,\zeta\right), I_u\left(\rho_j,\zeta\right), I_r\left(\rho_j,\zeta\right), R\left(\rho_j,\zeta\right), \delta\left(\rho_j,\zeta\right)\right)$ will not be less than

$$(j-1-\ln j)\wedge\frac{\ln^2 j}{2} \text{ or } \left(\frac{1}{j}-1+\ln j\right)\wedge\frac{\ln^2 j}{2}.$$

Based on (3.3) and (3.8), we have:

$$\begin{aligned}
&\mathbb{E}U\left(S(0), E(0), I_u(0), I_r(0), R(0), \delta(0)\right) + \Upsilon_2 T \\
&\ge \mathbb{E}U\left(S\left(\rho_j\wedge T\right), E\left(\rho_j\wedge T\right), I_u\left(\rho_j\wedge T\right), I_r\left(\rho_j\wedge T\right), R\left(\rho_j\wedge T\right), \delta\left(\rho_j\wedge T\right)\right) \\
&\ge \mathbb{E}\left[\mathbb{1}_{\Pi_j(\zeta)}U\left(S\left(\rho_j\wedge T\right), E\left(\rho_j\wedge T\right), I_u\left(\rho_j\wedge T\right), I_r\left(\rho_j\wedge T\right), R\left(\rho_j\wedge T\right), \delta\left(\rho_j\wedge T\right)\right)\right] \\
&\ge \mathbf{P}\left(\Pi_j(\zeta)\right)U\left(S\left(\rho_j,\zeta\right), E\left(\rho_j,\zeta\right), I_u\left(\rho_j,\zeta\right), I_r\left(\rho_j,\zeta\right), R\left(\rho_j,\zeta\right), \delta\left(\rho_j,\zeta\right)\right) \\
&\ge \varepsilon\left[(j-1-\ln j)\wedge\left(\frac{1}{j}-1+\ln j\right)\wedge\frac{\ln^2 j}{2}\right].
\end{aligned} \tag{3.9}$$

Considering the unlimited nature of j, as j approaches positive infinity, a contradiction appears.

$$+\infty < \mathbb{E}U\left(S(0), E(0), I_u(0), I_r(0), R(0), \delta(0)\right) + \Upsilon_2 T = +\infty.$$

That is to say $\rho_\infty = \infty$ a.s. The demonstration is finished. □

**Remark 3.1.**

Under the conditions outlined in the proof of Theorem 3.1, it can be concluded that if the initial population satisfies

$$S(0)+E(0)+I_u(0)+I_r(0)+R(0)<\frac{\Delta}{\mu},$$

then the set

$$\Sigma=\left\{(S,E,I_u,I_r,R,\delta)\in\mathbb{R}^5_+\times\mathbb{R}\;\middle|\;0\le S+E+I_u+I_r+R\le\frac{\Delta}{\mu}\right\}$$

is positively invariant under the dynamics of system (3.2).

## 3.3 Existence of a Stationary Distribution

This section focuses on deriving sufficient conditions to establish the existence of a stationary distribution, which signifies the long-term persistence of the susceptible population, exposed individuals, undetected infected individuals, and detected infected individuals. To facilitate this, we first introduce the following key parameter:

$$\begin{aligned}\mathcal{R}_0^s=&\frac{S_0}{\sigma+\mu}\Bigg(\frac{\sigma\beta v_1(1-\rho)}{\bar{\delta}+\gamma_{I_u}+\mu+d_1}+\frac{\sigma\beta v_2\rho}{\gamma_{I_r}+\mu+d_2}\\&+\frac{\sigma\beta v_2(1-\rho)}{(\bar{\delta}+\gamma_{I_u}+\mu+d_1)(\gamma_{I_r}+\mu+d_2)}\left(\frac{1}{\sqrt{\pi}}\int_{-\frac{\delta\sqrt{\rho_1}}{\sigma_1}}^{\infty}\left(\frac{\sigma_1}{\sqrt{\rho_1}}y+\bar{\delta}\right)^{\frac{1}{4}}e^{-y^2}\,dy\right)^4\Bigg)\\&-\Bigg(\frac{\sigma\beta v_1(1-\rho)\Delta}{\mu(\mu+d_1+\bar{\delta}+\gamma_{I_u})^2}\\&+\frac{\sigma\beta v_2(1-\rho)\Delta\left(\frac{1}{\sqrt{\pi}}\int_{-\frac{\delta\sqrt{\rho_1}}{\sigma_1}}^{\infty}\left(\frac{\sigma_1}{\sqrt{\rho_1}}y+\bar{\delta}\right)^{\frac{1}{4}}e^{-y^2}\,dy\right)^4}{\mu(\mu+d_1+\bar{\delta}+\gamma_{I_u})^2(\mu+d_2+\gamma_{I_r})}\Bigg)\\&\times\frac{\sigma_1}{2\sqrt{\pi\rho_1}(\sigma+\mu)}.\end{aligned}\tag{3.10}$$

The parameter $\mathcal{R}_0^s$ serves as the stochastic reproduction number, encapsulating both the deterministic and stochastic dynamics of the system. The following theorem formalizes the conditions under which the system exhibits a stationary distribution.

**Theorem 3.2.**

*Suppose the initial values* $(S(0), E(0), I_u(0), I_r(0), R(0), \delta(0)) \in \mathbb{R}^5_+ \times \mathbb{R}$. *If* $\mathcal{R}_0^s > 1$, *then the solution* $(S(t), E(t), I_u(t), I_r(t), R(t), \delta(t))$ *of system* (3.2) *possesses a unique stationary distribution* $\Theta(.)$ *with the ergodic property.*

**Proof.** Establish the Lyapunov function in the following manner:

$$\bar{\mathcal{W}}(S, E, I_u, I_r, R, \delta) = \tilde{N}\mathcal{W}_0 + \mathcal{W}_1 + \mathcal{W}_2 + W_3,$$

where

$$\begin{aligned}
\mathcal{W}_0 &= -2\ln E - (a_1 + a_2 + a_3)\ln S - (a_4 + a_5)\ln I_u - (a_6 + a_7)\ln I_r \\
&+ (a_1 + a_2 + a_3)\beta R, \\
\mathcal{W}_1 &= -\ln S - \ln E - \ln I_u - \ln I_r, \\
\mathcal{W}_2 &= -\ln\left(\frac{\Delta}{\mu} - S - E - I_u - I_r - R\right), \mathcal{W}_3 = \frac{\delta^2}{2}.
\end{aligned}$$

The positive constants $a_1$, $a_2$, $a_3$, $a_4$, $a_5$,$a_6$ and $a_7$ will be determined later, while $\tilde{N}$ is a suitably large positive constant satisfying the given condition

$$-\tilde{N}(\sigma + \mu)(\mathcal{R}_0^s - 1) + \Upsilon_3 \leq -2 \tag{3.11}$$

and

$$\Upsilon_3 := \sup_{\delta \in \mathbb{R}} \left\{ -\frac{\rho_1}{2}\delta^2 + |\delta| + \rho_1 \bar{\delta}\delta \right\} + \frac{\beta\Delta(v_1 + v_2)}{\mu} + 6\mu + 2\sigma + d_1 + d_2 + \gamma_{I_u} + \gamma_{I_r} + \frac{\sigma_1^2}{2} < \infty. \tag{3.12}$$

Indeed, $\bar{\mathcal{W}}(S, E, I_u, I_r, R, \delta)$ exhibits continuity, conforming to

$$\liminf_{m \to \infty, (S,E,I_u,I_r,R,\delta) \in \Sigma \setminus D_m} \bar{\mathcal{W}}(S, E, I_u, I_r, R, \delta) = +\infty.$$

Consequently, a non-negative $C^2$-function $\mathcal{W}(S, E, I_u, I_r, R, \delta)$ is provided as follows

$$\mathcal{W}(S, E, I_u, I_r, R, \delta) = \bar{\mathcal{W}}(S, E, I_u, I_r, R, \delta) - \bar{\mathcal{W}}\left(S_0, E_0, I_{u_0}, I_{r_0}, R_0, \delta_0\right),$$

where $\left(S_0, E_0, I_{u_0}, I_{r_0}, R_0, \delta_0\right) \in \Sigma$ is the minimum point of $\bar{\mathcal{W}}(S, E, I_u, I_r, R, \delta)$.

By Applying Itô's formula to $\mathcal{W}_0$, we obtain

$$\begin{aligned}
\mathcal{L}\mathcal{W}_0 =& -(\frac{a_1+a_2+a_3}{S})(\Delta-\beta(v_1I_u+v_2I_r)S-\mu S)-\frac{2}{E}(\beta(v_1I_u+v_2I_r)S-(\sigma+\mu)E)\\
&-(\frac{a_4+a_5}{I_u})(\sigma(1-\rho)E-\left(\mu+d_1+\max\{\delta(t),0\}+\gamma_{I_u}\right)I_u)\\
&(\frac{a_6+a_7}{I_r})(\sigma\rho E+\max\{\delta(t),0\}I_u-\left(\mu+d_2+\gamma_{I_r}\right)I_r)\\
&-(a_1+a_2+a_3)\beta(\gamma_{I_u}I_u+\gamma_{I_r}I_r-\mu R)\\
\leq&(-\frac{a_1\Delta}{S}-\frac{a_4\sigma(1-\rho)E}{I_u}-\frac{\beta v_1I_uS}{E})+a_1\mu+a_4(\mu+d_1+\max\{\delta(t),0\}+\gamma_{I_u})\\
&+(-\frac{a_2\Delta}{S}-\frac{a_6\sigma\rho E}{I_r}-\frac{\beta v_2I_rS}{E})+a_2\mu+a_6(\mu+d_2+\gamma_{I_r})\\
&+(-\frac{a_3\Delta}{S}-\frac{a_5\sigma(1-\rho)E}{I_u}-\frac{a_7\max\{\delta,0\}I_u}{I_r}-\frac{\beta v_2I_rS}{E})\\
&+a_3\mu+a_5(\mu+d_1+\max\{\delta(t),0\}+\gamma_{I_u})+a_7(\mu+d_2+\gamma_{I_r})\\
&+(a_1+a_2+a_3)\beta(v_1I_u+v_2I_r)+(a_1+a_2+a_3)\beta(\gamma_{I_u}I_u+\gamma_{I_r}I_r)+2(\sigma+\mu)\\
\leq&-3\sqrt[3]{a_1a_4\sigma\beta v_1(1-\rho)\Delta}+a_1\mu+a_4(\mu+d_1+\bar{\delta}+\gamma_{I_u})\\
&-3\sqrt[3]{a_2a_6\sigma\beta v_2\rho\Delta}+a_2\mu+a_6(\mu+d_2+\gamma_{I_r})\\
&-4\sqrt[4]{a_3a_5a_7\sigma\beta v_2(1-\rho)\Delta\tilde{\delta}}+a_3\mu+a_5(\mu+d_1+\bar{\delta}+\gamma_{I_u})+a_7(\mu+d_2+\gamma_{I_r})\\
&+(a_4+a_5)A(t)\vee 0+2(\sigma+\mu)+(a_1+a_2+a_3)\beta(v_1I_u+v_2I_r)\\
&+(a_1+a_2+a_3)\beta(\gamma_{I_u}I_u+\gamma_{I_r}I_r),
\end{aligned}\tag{3.13}$$

where

$$\tilde{\delta}=\max\{\delta(t),0\},\qquad A(t)=\delta(t)-\bar{\delta}.$$

In the previous inequality, we have applied the arithmetic-geometric men inequality

$$\frac{b_1+b_2+\ldots+b_n}{n}\geq(b_1b_2\ldots b_n)^{\frac{1}{n}}\qquad \text{For any } b_n>0 \text{and } n\in\mathbb{N}.$$

Concerning the equationcorresponding to the sixth position in system (3.2), that is

$$d\delta(t)=\rho_1[\bar{\delta}-\delta(t)]dt+\sigma_1dB(t).$$

Based on the references [101, 102, 113, 114] it can be inferred that $\delta(t)$ exhibits the ergodic property, and it is expected to undergo weak convergence towards the invariant density

$$f(y)=\frac{\sqrt{\rho_1}}{\sqrt{\pi}\sigma_1}e^{-\frac{\rho_1(y-\bar{\delta})^2}{\sigma_1^2}},y\in\mathbb{R}.$$

Having incorporated the ergodic theorem [115], the aforementioned leads us to the following conclusion

$$\begin{aligned}\int_{-\infty}^{\infty}(y\vee 0)^{\frac{1}{4}}f(y)dy &= \int_{0}^{\infty}y^{\frac{1}{4}}f(y)dy\\ &= \int_{0}^{\infty}y^{\frac{1}{4}}\frac{\sqrt{\rho_1}}{\sqrt{\pi}\sigma_1}e^{-\frac{\rho_1(y-\bar{\delta})^2}{\sigma_1^2}}dy\\ &= \frac{1}{\sqrt{\pi}}\int_{-\frac{\bar{\delta}\sqrt{\rho_1}}{\sigma_1}}^{\infty}\left(\frac{\sigma_1}{\sqrt{\rho_1}}y+\bar{\delta}\right)^{\frac{1}{4}}e^{-y^2}dy.\end{aligned} \tag{3.14}$$

Likewise, in the case of the stochastic differential equation

$$dA(t)=\rho_1 A(t)dt+\sigma_1 dB(t).$$

The ergodic property of $A(t)$ and its eventual weak convergence to the invariant density can be readily derived

$$g(y)=\frac{\sqrt{\rho_1}}{\sqrt{\pi}\sigma_1}e^{-\frac{\rho_1 y^2}{\sigma_1^2}},y\in\mathbb{R}.$$

Drawing upon the ergodic theorem [115], we arriveat the following conclusion

$$\begin{aligned}\int_{-\infty}^{\infty}(y\vee 0)g(y)dy &= \int_{0}^{\infty}yg(y)dy\\ &= \int_{0}^{\infty}y\frac{\sqrt{\rho_1}}{\sqrt{\pi}\sigma_1}e^{-\frac{\rho_1 y^2}{\sigma_1^2}}dy\\ &= \frac{\sigma_1}{2\sqrt{\pi\rho_1}}.\end{aligned} \tag{3.15}$$

Substituting (3.14) and (3.15) into (3.13) leads to that

$$\begin{aligned}\mathcal{L}\mathcal{W}_0 \le & -3\sqrt[3]{a_1a_4\sigma\beta v_1(1-\rho)\Delta}+a_1\mu+a_4(\mu+d_1+\bar{\delta}+\gamma_{I_u})-3\sqrt[3]{a_2a_6\sigma\beta v_2\rho\Delta}+a_2\mu\\ &+a_6(\mu+d_2+\gamma_{I_r})-4\sqrt[4]{a_3a_5a_7\sigma\beta v_2(1-\rho)\Delta\widehat{\delta}}\\ &+(4\sqrt[4]{a_3a_5a_7\sigma\beta v_2(1-\rho)\Delta\widehat{\delta}}-4\sqrt[4]{a_3a_5a_7\sigma\beta v_2(1-\rho)\Delta\bar{\delta}})\\ &+a_3\mu+a_5(\mu+d_1+\bar{\delta}+\gamma_{I_u})+a_7(\mu+d_2+\gamma_{I_r})\\ &+2(\sigma+\mu)+(a_4+a_5)\frac{\sigma_1}{2\sqrt{\pi\rho_1}}+(a_4+a_5)(A(t)\vee 0-\int_{0}^{\infty}yg(y)dy)\\ &+(a_1+a_2+a_3)\beta(v_1I_u+v_2I_r)+(a_1+a_2+a_3)\beta(\gamma_{I_u}I_u+\gamma_{I_r}I_r),\end{aligned} \tag{3.16}$$

where

$$\widehat{\delta}=\left(\int_{-\infty}^{\infty}(y\vee 0)^{\frac{1}{4}}f(y)dy\right)^4=\left(\frac{1}{\sqrt{\pi}}\int_{-\frac{\delta\sqrt{\rho_1}}{\sigma_1}}^{\infty}\left(\frac{\sigma_1}{\sqrt{\rho_1}}y+\bar{\delta}\right)^{\frac{1}{4}}e^{-y^2}dy\right)^4.$$

Let

$$a_1\mu = a_4(\mu + d_1 + \bar{\delta} + \gamma_{I_u}) = \frac{\sigma\beta v_1(1-\rho)\Delta}{\mu(\mu + d_1 + \bar{\delta} + \gamma_{I_u})},$$
$$a_2\mu = a_6(\mu + d_2 + \gamma_{I_r}) = \frac{\sigma\beta v_2\rho\Delta}{\mu(\mu + d_2 + \gamma_{I_r})}$$
$$a_3\mu = a_5(\mu + d_1 + \bar{\delta} + \gamma_{I_u}) = a_7(\mu + d_2 + \gamma_{I_r}) = \frac{\sigma\beta v_2(1-\rho)\Delta\widehat{\delta}}{\mu(\mu + d_1 + \bar{\delta} + \gamma_{I_u})(\mu + d_2 + \gamma_{I_r})}.$$

Consequently, we acquire

$$a_1 = \frac{\sigma\beta v_1(1-\rho)\Delta}{\mu^2(\mu + d_1 + \bar{\delta} + \gamma_{I_u})}, a_4 = \frac{\sigma\beta v_1(1-\rho)\Delta}{\mu(\mu + d_1 + \bar{\delta} + \gamma_{I_u})^2}$$
$$a_2 = \frac{\sigma\beta v_2\rho\Delta}{\mu^2(\mu + d_2 + \gamma_{I_r})}, a_6 = \frac{\sigma\beta v_2\rho\Delta}{\mu(\mu + d_2 + \gamma_{I_r})^2}$$
$$a_3 = \frac{\sigma\beta v_2(1-\rho)\Delta\widehat{\delta}}{\mu^2(\mu + d_1 + \bar{\delta} + \gamma_{I_u})(\mu + d_2 + \gamma_{I_r})}, a_5 = \frac{\sigma\beta v_2(1-\rho)\Delta\widehat{\delta}}{\mu(\mu + d_1 + \bar{\delta} + \gamma_{I_u})^2(\mu + d_2 + \gamma_{I_r})},$$
$$a_7 = \frac{\sigma\beta v_2(1-\rho)\Delta\widehat{\delta}}{\mu(\mu + d_1 + \bar{\delta} + \gamma_{I_u})(\mu + d_2 + \gamma_{I_r})^2}.$$

Consequently, as a result

$$\begin{aligned}
\mathcal{L}\mathcal{W}_0 \leq & -\frac{\sigma\beta v_1(1-\rho)\Delta}{\mu(\mu + d_1 + \bar{\delta} + \gamma_{I_u})} - \frac{\sigma\beta v_2\rho\Delta}{\mu(\mu + d_2 + \gamma_{I_r})} - \frac{\sigma\beta v_2(1-\rho)\Delta\widehat{\delta}}{\mu(\mu + d_1 + \bar{\delta} + \gamma_{I_u})(\mu + d_2 + \gamma_{I_r})} \\
& + 2(\sigma + \mu) + (a_4 + a_5)\frac{\sigma_1}{2\sqrt{\pi\rho_1}} + 4\sqrt[4]{a_3a_5a_7\sigma\beta v_2(1-\rho)\Delta}(\sqrt[4]{\widehat{\delta}} - \sqrt[4]{\bar{\delta}}) \\
& + (a_4 + a_5)(A(t) \vee 0 - \int_0^\infty yg(y)dy) + (a_1 + a_2 + a_3)\beta(v_1I_u + v_2I_r) + (a_1 + a_2 \\
& + a_3)\beta(\gamma_{I_u}I_u + \gamma_{I_r}I_r) \\
\leq & -(\sigma + \mu)(\mathcal{R}_0^s - 1) + \sigma + \mu + (a_4 + a_5)(A(t) \vee 0 - \int_0^\infty yg(y)dy) \\
& + (a_1 + a_2 + a_3)\beta(v_1I_u + v_2I_r) + (a_1 + a_2 + a_3)\beta(\gamma_{I_u}I_u + \gamma_{I_r}I_r),
\end{aligned} \tag{3.17}$$

where

$$\mathcal{R}_0^s = \frac{S_0}{(\sigma + \mu)}\left(\frac{\sigma\beta v_1(1-\rho)}{\left(\bar{\delta} + \gamma_{I_u} + \mu + d_1\right)} + \frac{\sigma\beta v_2\rho}{\left(\gamma_{I_r} + \mu + d_2\right)} + \frac{\sigma\beta v_2(1-\rho)\widehat{\delta}}{\left(\bar{\delta} + \gamma_{I_u} + \mu + d_1\right)\left(\gamma_{I_r} + \mu + d_2\right)}\right)$$
$$- (a_1 + a_5)\frac{\sigma_1}{2\sqrt{\pi\rho_1}(\sigma + \mu)}.$$

Analogously, employing Itô's formula to $\mathcal{W}_1$, $\mathcal{W}_2$, and $\mathcal{W}_3$, respectively, yields the

following results

$$\begin{aligned}\mathcal{LW}_1 =& -\frac{\Delta}{S}+\beta(v_1I_u+v_2I_r)+\mu-\frac{\beta(v_1I_u+v_2I_r)S}{E}+\sigma+\mu\\ &-\frac{\sigma(1-\rho)E}{I_u}+(\mu+d_1+\max\{\delta(t),0\}+\gamma_{I_u})-\frac{\sigma\rho E}{I_r}-\frac{\max\{\delta(t),0\}I_u}{I_r}+(\mu+d_2+\gamma_{I_r})\\ \leq& -\frac{\Delta}{S}-\frac{\beta(v_1I_u+v_2I_r)S}{E}+\frac{\beta\Delta(v_1+v_2)}{\mu}+|\delta|+4\mu+\sigma+d_1+d_2+\gamma_{I_u}+\gamma_{I_r},\end{aligned} \tag{3.18}$$

$$\begin{aligned}\mathcal{LW}_2 =& \frac{1}{\frac{\Delta}{\mu}-S-E-I_u-I_r-R}(\Delta-\mu S-\mu I_u-\mu I_r-d_1I_u-d_2I_r-\mu R)\\ \leq& \mu-\frac{d_1I_u+d_2I_r}{\frac{\Delta}{\mu}-S-E-I_u-I_r-R}\end{aligned} \tag{3.19}$$

and

$$\mathcal{LW}_3=\rho_1\bar{\delta}\delta-\rho_1\delta^2+\frac{\sigma_1^2}{2}. \tag{3.20}$$

By (3.17),(3.18), (3.19) and (3.20), we get

$$\begin{aligned}\mathcal{LW} \leq& -\tilde{N}(\sigma+\mu)(\mathcal{R}_0^s-1)+4\sqrt[4]{a_3a_5a_7\sigma\beta v_2(1-\rho)\Delta}(\sqrt[4]{\delta}-\sqrt[4]{\tilde{\delta}})\\ &+\tilde{N}(a_1+a_2+a_3)\beta(v_1I_u+v_2I_r)+\tilde{N}(a_1+a_2+a_3)\beta(\gamma_{I_u}I_u+\gamma_{I_r}I_r)\\ &+\tilde{N}(a_4+a_5)(A(t)\vee 0-\int_0^\infty yg(y)dy)-\frac{\Delta}{S}-\frac{\beta(v_1I_u+v_2I_r)S}{E}\\ &-\frac{d_1I_u+d_2I_r}{\frac{\Delta}{\mu}-S-E-I_u-I_r-R}\\ &+\rho_1\bar{\delta}\delta-\frac{\rho_1}{2}\delta^2-\frac{\rho_1}{2}\delta^2+|\delta|+\frac{\beta\Delta(v_1+v_2)}{\mu}+6\mu+2\sigma+d_1+d_2+\gamma_{I_u}+\gamma_{I_r}+\frac{\sigma_1^2}{2}\\ :=&\mathbf{H}(S,E,I_u,I_r,R;\delta)+4\sqrt[4]{a_3a_5a_7\sigma\beta v_2(1-\rho)\Delta}(\sqrt[4]{\delta}-\sqrt[4]{\tilde{\delta}})+\tilde{N}(a_4+a_5)(A(t)\vee 0\\ &-\int_0^\infty yg(y)dy),\end{aligned} \tag{3.21}$$

where

$$\begin{aligned}&\mathbf{H}(S,E,I_u,I_r,R;\delta)=-\tilde{N}(\sigma+\mu)(\mathcal{R}_0^s-1)+\tilde{N}(a_1+a_2+a_3)\beta(v_1I_u+v_2I_r)\\ &+\tilde{N}(a_1+a_2+a_3)\beta(\gamma_{I_u}I_u+\gamma_{I_r}I_r)-\frac{\Delta}{S}-\frac{\beta(v_1I_u+v_2I_r)S}{E}-\frac{d_1I_u+d_2I_r}{\frac{\Delta}{\mu}-S-E-I_u-I_r-R}\\ &+\rho_1\bar{\delta}\delta-\frac{\rho_1}{2}\delta^2-\frac{\rho_1}{2}\delta^2+|\delta|+\frac{\beta\Delta(v_1+v_2)}{\mu}+6\mu+2\sigma+d_1+d_2+\gamma_{I_u}+\gamma_{I_r}+\frac{\sigma_1^2}{2}.\end{aligned} \tag{3.22}$$

Following that, we define a suitable bounded subset $\widehat{E}$ is described by

$$\widehat{E} = \left\{(S,E,I_u,I_r,R)^T \in \Sigma, S \geq \varepsilon, I_u \geq \varepsilon, I_r \geq \varepsilon, E \geq \varepsilon^3, S+E+I_u+I_r+R \leq \frac{\Delta}{\mu} - \varepsilon^2, |\delta| \leq \frac{1}{\varepsilon}\right\}.$$

Let $\varepsilon$ be a sufficiently small positive constant that fulfills the following conditions .

$$-\frac{\Delta}{\varepsilon} + \Upsilon_4 \leq -1 \tag{3.23}$$

$$\varepsilon \leq \frac{1}{\tilde{N}\beta(a_1+a_2+a_3)(v_1+v_2+\gamma_{I_u}+\gamma_{I_r})} \tag{3.24}$$

$$-\frac{\beta(v_1+v_2)}{\varepsilon} + \Upsilon_4 \leq -1 \tag{3.25}$$

$$-\frac{d_1+d_2}{\varepsilon} + \Upsilon_4 \leq -1 \tag{3.26}$$

$$-\frac{\rho_1}{2\varepsilon^2} + \Upsilon_4 \leq -1, \tag{3.27}$$

where

$$\begin{aligned}\Upsilon_4 := &\sup_{\delta\in\mathbb{R}}\left\{-\frac{\rho_1}{2}\delta^2 + |\delta| + \rho_1\bar{\delta}\delta\right\} + \tilde{N}(a_1+a_2+a_3)\beta(v_1I_u+v_2I_r)\\ &+\tilde{N}(a_1+a_2+a_3)\beta(\gamma_{I_u}I_u+\gamma_{I_r}I_r)\\ &+\frac{\beta\Delta(v_1+v_2)}{\mu} + 6\mu + 2\sigma + d_1 + d_2 + \gamma_{I_u} + \gamma_{I_r} + \frac{\sigma_1^2}{2},\end{aligned} \tag{3.28}$$

we can then partition the set $\Sigma\backslash\widehat{E}$ into the following five subsets $\widehat{E}_i^c, i = 1,2,3,4,5$, where

$$\widehat{E}_1^c = \left\{(S,E,I_u,I_r,R,\delta)^T \in \Sigma, S < \varepsilon\right\},$$
$$\widehat{E}_2^c = \left\{(S,E,I_u,I_r,R,\delta)^T \in \Sigma, I_u < \varepsilon, I_r < \varepsilon\right\},$$
$$\widehat{E}_3^c = \left\{(S,E,I_u,I_r,R,\delta)^T \in \Sigma, E < \varepsilon^3, I_u \geq \varepsilon, I_r \geq \varepsilon\right\},$$
$$\widehat{E}_4^c = \left\{(S,E,I_u,I_r,R,\delta)^T \in \Sigma, S+E+I_u+I_r+R > \frac{\Delta}{\mu} - \varepsilon^2, I_u \geq \varepsilon, I_r \geq \varepsilon\right\},$$
$$\widehat{E}_5^c = \left\{(S,E,I_u,I_r,R,\delta)^T \in \Sigma, |\delta| > \frac{1}{\varepsilon}\right\}.$$

Evidently, it is apparent that

$$\Sigma\backslash\widehat{E} = \bigcup_{i=1}^{5}\widehat{E}_i^c.$$

We shall now proceed to establish the proof that $\mathbf{H}(S,E,I_u,I_r,R;\delta) \leq -1$ on $\widehat{E}^c$.

Put differently, we must verify its fulfillment across the aforementioned five regions

- **Case 1.** For any $(S,E,I_u,I_r,R,\delta)^T \in \widehat{E}_1^c$, according to (3.22), we get

$$\begin{aligned}
\mathbf{H}(S,E,I_u,I_r,R,\delta) \leq & -\frac{\Delta}{S} + \tilde{N}(a_1+a_2+a_3)\beta(v_1 I_u + v_2 I_r) + \tilde{N}(a_1+a_2+a_3)\beta(\gamma_{I_u} I_u + \gamma_{I_r} I_r) \\
& + \rho_1 \bar{\delta}\delta - \frac{\rho_1}{2}\delta^2 + |\delta| + \frac{\beta\Delta(v_1+v_2)}{\mu} + 6\mu + 2\sigma + d_1 + d_2 + \gamma_{I_u} + \gamma_{I_r} + \frac{\sigma_1^2}{2} \\
\leq & -\frac{\Delta}{S} + \tilde{N}(a_1+a_2+a_3)\beta(v_1+v_2)\frac{\Delta}{\mu} + \tilde{N}(a_1+a_2+a_3)\beta(\gamma_{I_u}+\gamma_{I_r})\frac{\Delta}{\mu} \\
& + \rho_1 \bar{\delta}\delta - \frac{\rho_1}{2}\delta^2 + |\delta| + \frac{\beta\Delta(v_1+v_2)}{\mu} + 6\mu + 2\sigma + d_1 + d_2 + \gamma_{I_u} + \gamma_{I_r} + \frac{\sigma_1^2}{2} \\
\leq & -\frac{\Delta}{\varepsilon} + \Upsilon_4 \\
\leq & -1,
\end{aligned} \tag{3.29}$$

  which follows from (3.23) and (3.28)

- **Case 2.** For any $(S,E,I_u,I_r,R,\delta)^T \in \widehat{E}_2^c$, by (3.22), we obtain

$$\begin{aligned}
\mathbf{H}(S,E,I_u,I_r,R,\delta) \leq & -\tilde{N}(\sigma+\mu)(\mathcal{R}_0^s - 1) + \tilde{N}(a_1+a_2+a_3)\beta(v_1 I_u + v_2 I_r) \\
& + \tilde{N}(a_1+a_2+a_3)\beta(\gamma_{I_u} I_u + \gamma_{I_r} I_r) + \rho_1 \bar{\delta}\delta - \frac{\rho_1}{2}\delta^2 + |\delta| + \frac{\beta\Delta(v_1+v_2)}{\mu} \\
& + 6\mu + 2\sigma + d_1 + d_2 + \gamma_{I_u} + \gamma_{I_r} + \frac{\sigma_1^2}{2} \\
\leq & -\tilde{N}(\sigma+\mu)(\mathcal{R}_0^s - 1) + \Upsilon_3 + \tilde{N}\beta(a_1+a_2+a_3)(v_1+v_2+\gamma_{I_u}+\gamma_{I_r})\varepsilon \\
\leq & -1,
\end{aligned} \tag{3.30}$$

  which follows from (3.11) and (3.24) .

- **Case 3.** For any $(S,E,I_u,I_r,R,\delta)^T \in \widehat{E}_3^c$, from (3.22) it follows that

$$\begin{aligned}
\mathbf{H}(S,E,I_u,I_r,R,\delta) \leq & -\frac{\beta(v_1I_u+v_2I_r)S}{E}+\tilde{N}(a_1+a_2+a_3)\beta(v_1I_u+v_2I_r)\\
&+\tilde{N}(a_1+a_2+a_3)\beta(\gamma_{I_u}I_u+\gamma_{I_r}I_r)+\rho_1\bar{\delta}\delta-\frac{\rho_1}{2}\delta^2+|\delta|+\frac{\beta\Delta(v_1+v_2)}{\mu}\\
&+6\mu+2\sigma+d_1+d_2+\gamma_{I_u}+\gamma_{I_r}+\frac{\sigma_1^2}{2}\\
\leq & -\frac{\beta(v_1I_u+v_2I_r)S}{E}+\tilde{N}(a_1+a_2+a_3)\beta(v_1+v_2)\frac{\Delta}{\mu}\\
&+\tilde{N}(a_1+a_2+a_3)\beta(\gamma_{I_u}+\gamma_{I_r})\frac{\Delta}{\mu}+\rho_1\bar{\delta}\delta-\frac{\rho_1}{2}\delta^2+|\delta|+\frac{\beta\Delta(v_1+v_2)}{\mu}\\
&+6\mu+2\sigma+d_1+d_2+\gamma_{I_u}+\gamma_{I_r}+\frac{\sigma_1^2}{2}\\
\leq & -\frac{(v_1+v_2)\varepsilon^2}{\varepsilon^3}+\Upsilon_4\\
\leq & -\frac{(v_1+v_2)}{\varepsilon}+\Upsilon_4\\
\leq & -1,
\end{aligned} \tag{3.31}$$

  which follows from (3.25) .

- **Case 4.** For any $(S,E,I_u,I_r,R,\delta)^T \in \widehat{E}_4^c$, in view of (3.22), we obtain

$$\begin{aligned}
\mathbf{H}(S,E,I_u,I_r,R,\delta) \leq & -\frac{d_1I_u+d_2I_r}{\frac{\Delta}{\mu}-S-E-I_u-I_r-R}\\
&+\tilde{N}(a_1+a_2+a_3)\beta(v_1I_u+v_2I_r)+\tilde{N}(a_1+a_2+a_3)\beta(\gamma_{I_u}I_u+\gamma_{I_r}I_r)\\
&+\rho_1\bar{\delta}\delta-\frac{\rho_1}{2}\delta^2+|\delta|+\frac{\beta\Delta(v_1+v_2)}{\mu}+6\mu+2\sigma+d_1+d_2+\gamma_{I_u}+\gamma_{I_r}+\frac{\sigma_1^2}{2}\\
\leq & -\frac{d_1I_u+d_2I_r}{\frac{\Delta}{\mu}-S-E-I_u-I_r-R}\\
&+\tilde{N}(a_1+a_2+a_3)\beta(v_1+v_2)\frac{\Delta}{\mu}+\tilde{N}(a_1+a_2+a_3)\beta(\gamma_{I_u}+\gamma_{I_r})\frac{\Delta}{\mu}\\
&+\rho_1\bar{\delta}\delta-\frac{\rho_1}{2}\delta^2+|\delta|+\frac{\beta\Delta(v_1+v_2)}{\mu}+6\mu+2\sigma+d_1+d_2+\gamma_{I_u}+\gamma_{I_r}+\frac{\sigma_1^2}{2}\\
\leq & -\frac{(d_1+d_2)\varepsilon}{\varepsilon^2}+\Upsilon_4\\
\leq & -\frac{(d_1+d_2)}{\varepsilon}+\Upsilon_4\\
\leq & -1,
\end{aligned} \tag{3.32}$$

  which follows from (3.26) .

- **Case 5.** For any $(S,E,I_u,I_r,R,\delta)^T \in \widehat{E}_5^c$, in view of (3.22), we have

$$\begin{aligned}
\mathbf{H}(S,E,I_u,I_r,R,\delta) \leq & -\frac{\rho_1}{2}\delta^2 + \tilde{N}(a_1+a_2+a_3)\beta(v_1I_u+v_2I_r) + \tilde{N}(a_1+a_2+a_3)\beta(\gamma_{I_u}I_u+\gamma_{I_r}I_r) \\
& + \rho_1\bar{\delta}\delta - \frac{\rho_1}{2}\delta^2 + |\delta| + \frac{\beta\Delta(v_1+v_2)}{\mu} + 6\mu + 2\sigma + d_1 + d_2 + \gamma_{I_u} + \gamma_{I_r} + \frac{\sigma_1^2}{2} \\
\leq & -\frac{\rho_1}{2}\delta^2 + \tilde{N}(a_1+a_2+a_3)\beta(v_1+v_2)\frac{\Delta}{\mu} + \tilde{N}(a_1+a_2+a_3)\beta(\gamma_{I_u}+\gamma_{I_r})\frac{\Delta}{\mu} \\
& + \rho_1\bar{\delta}\delta - \frac{\rho_1}{2}\delta^2 + |\delta| + \frac{\beta\Delta(v_1+v_2)}{\mu} + 6\mu + 2\sigma + d_1 + d_2 + \gamma_{I_u} + \gamma_{I_r} + \frac{\sigma_1^2}{2} \\
\leq & -\frac{\rho_1}{2\varepsilon^2} + \Upsilon_4 \\
\leq & -1,
\end{aligned} \tag{3.33}$$

  which follows from (3.27).

Drawing from the evidence presented in inequalities (3.29), (3.30), (3.31), (3.32) and (3.33), a straightforward conclusion can be reached, establishing the existence of a sufficiently small $\varepsilon$ that satisfies The following condition

$$\mathbf{H}(S,E,I_u,I_r,R,\delta) \leq -1 \qquad \text{for any} (S,E,I_u,I_r,R,\delta)^T \in \widehat{E}^c. \tag{3.34}$$

So we have

$$\mathbf{H}(S,E,I_u,I_r,R,\delta) \leq \Upsilon_5 < \infty \qquad \text{for any} (S,E,I_u,I_r,R,\delta)^T \in \mathbb{R}_+^5 \times \mathbb{R}, \tag{3.35}$$

where

$$\begin{aligned}
\Upsilon_5 := \sup_{(S,E,I_u,I_r,R,\delta)\in\mathbb{R}_+^5\times\mathbb{R}} \Big\{ & -\tilde{N}(\sigma+\mu)(\mathcal{R}_0^s - 1) + \tilde{N}(a_1+a_2+a_3)\beta(v_1I_u+v_2I_r) \\
& + \tilde{N}(a_1+a_2+a_3)\beta(\gamma_{I_u}I_u+\gamma_{I_r}I_r) - \frac{\Delta}{S} - \frac{\beta(v_1I_u+v_2I_r)S}{E} - \frac{d_1I_u+d_2I_r}{\frac{\Delta}{\mu}-S-E-I_u-I_r-R} \\
& + \rho_1\bar{\delta}\delta\frac{\rho_1}{2}\delta^2 - \frac{\rho_1}{2}\delta^2 + \delta + \frac{\beta\Delta(v_1+v_2)}{\mu} + 6\mu + 2\sigma + d_1 + d_2 + \gamma_{I_u} + \gamma_{I_r} + \frac{\sigma_1^2}{2} \Big\}.
\end{aligned}$$

By integrating equation (3.21) over the interval $[0,t]$ for any initial values $(S(0),E(0),I_u(0),I_r(0),R(0),\delta(0)) \in \Sigma$ and subsequently calculating the mathematical

expectation, the following expression is obtained

$$
\begin{aligned}
0 \leq & \frac{\mathbb{E}\mathcal{W}(S(t), E(t), I_u(t), I_r(t), R(t), \delta(t))}{t} \\
= & \frac{\mathbb{E}\mathcal{W}(S(0), E(0), I_u(0), I_r(0), R(0), \delta(0))}{t} + \frac{1}{t}\int_0^t \mathbb{E}(\mathcal{L}\mathcal{W}(S(s), E(s), I_u(s), I_r(s), R(s), \delta(s)))ds \\
\leq & \frac{\mathbb{E}\mathcal{W}(S(0), E(0), I_u(0), I_r(0), R(0), \delta(0))}{t} + \frac{1}{t}\int_0^t \mathbb{E}(\mathbf{H}(S(s), E(s), I_u(s), I_r(s), R(s), \delta(s)))ds \\
& + 4\tilde{N}\sqrt[4]{a_3 a_5 a_7 \sigma\beta v_2(1-\rho)\Delta}\mathbb{E}\left[\int_{-\infty}^{\infty}(y \vee 0)^{\frac{1}{4}} f(y)dy - \frac{1}{t}\int_0^t (\delta(s) \vee 0)^{\frac{1}{4}} ds\right] \\
& + \tilde{N}\mathbb{E}\left[\frac{1}{t}\int_0^t (A(s) \vee 0)ds - \int_0^{\infty} y g(y)dy\right].
\end{aligned}
\tag{3.36}
$$

Utilizing the ergodic properties of both $\delta(t)$ and $A(t)$, along with the strong law of large numbers as demonstrated in [112], we derive the following

$$
\lim_{t\to\infty}\mathbb{E}\left[\int_{-\infty}^{\infty}(y \vee 0)^{\frac{1}{4}} f(y)dy - \frac{1}{t}\int_0^t (\delta(s) \vee 0)^{\frac{1}{4}} ds\right] = 0 \qquad a.s.,
$$

and

$$
\lim_{t\to\infty}\mathbb{E}\left[\frac{1}{t}\int_0^t (A(s) \vee 0)ds - \int_0^{\infty} y g(y)dy\right] = 0 \qquad a.s.
$$

Therefore, by applying the inferior limit to both sides of (3.36), we deduce the subsequent outcome

$$
\begin{aligned}
0 \leq & \liminf_{t\to\infty} \frac{\mathbb{E}\mathcal{W}(S(0), E(0), I_u(0), I_r(0), R(0), \delta(0))}{t} \\
& + \liminf_{t\to\infty} \frac{1}{t}\int_0^t \mathbb{E}(\mathbf{H}(S(s), E(s), I_u(s), I_r(s), R(s), \delta(s)))ds \\
= & \liminf_{t\to\infty} \frac{1}{t}\int_0^t \mathbb{E}(\mathbf{H}(S(s), E(s), I_u(s), I_r(s), R(s), \delta(s)))ds \qquad a.s.
\end{aligned}
\tag{3.37}
$$

Furthermore, in accordance with to (3.34) and (3.35), it follows that we obtain

$$\begin{aligned}
&\liminf_{t\to\infty}\frac{1}{t}\int_0^t \mathbb{E}(\mathbf{H}(S(s),E(s),I_u(s),I_r(s),R(s),\delta(s)))ds\\
=&\liminf_{t\to\infty}\frac{1}{t}\int_0^t \mathbb{E}(\mathbf{H}(S(s),E(s),I_u(s),I_r(s),R(s),\delta(s)))\mathbf{1}_{(S(s),E(s),I_u(s),I_r(s),R(s),\delta(s))^T\in\widehat{E}}ds\\
&+\liminf_{t\to\infty}\frac{1}{t}\int_0^t \mathbb{E}(\mathbf{H}(S(s),E(s),I_u(s),I_r(s),R(s),\delta(s)))\mathbb{1}_{(S(s),E(s),I_u(s),I_r(s),R(s),\delta(s))^T\in(\Sigma\setminus\widehat{E})}ds\\
\leq&\Upsilon_5\liminf_{t\to\infty}\frac{1}{t}\int_0^t \mathbb{1}_{(S(s),E(s),I_u(s),I_r(s),R(s),\delta(s))^T\in\widehat{E}}ds\\
&-\liminf_{t\to\infty}\frac{1}{t}\int_0^t \mathbb{1}_{(S(s),E(s),I_u(s),I_r(s),R(s),\delta(s))^T\in(\Sigma\setminus\widehat{E})}ds\\
\leq&-1+(\Upsilon_5+1)\liminf_{t\to\infty}\frac{1}{t}\int_0^t \mathbb{1}_{(S(s),E(s),I_u(s),I_r(s),R(s),\delta(s))^T\in\widehat{E}}ds.
\end{aligned} \tag{3.38}$$

Consequently, from (3.37) and (3.38) lead to the conclusion that

$$\liminf_{t\to\infty}\frac{1}{t}\int_0^t \mathbb{1}_{(S(s),E(s),I_u(s),I_r(s),R(s),\delta(s))^T\in\widehat{E}}ds \geq \frac{1}{\Upsilon_5+1}>0 \qquad a.s. \tag{3.39}$$

Considering the event probability definition and the application of Fatou's lemma [116, 101, 102]

$$\liminf_{t\to\infty}\frac{1}{t}\int_0^t \mathbf{P}(s,S(s),E(s),I_u(s),I_r(s),R(s),\delta(s),\widehat{E})ds \geq \frac{1}{\Upsilon_5+1}>0 \qquad a.s. \tag{3.40}$$

Where $\mathbf{P}(s,S(s),E(s),I_u(s),I_r(s),R(s),\delta(s),\widehat{E})$ is the transition probability of $(S(s),E(s),I_u(s),I_r(s),R(s),\delta(s))^T$ belonging to set $\widehat{E}$.Thus, we have fulfilled the conditions of Lemma 2.33 and thus the system (3.2) has at least one stationary distribution $\Theta(.)$ on $\mathbb{R}^5_+\times\mathbb{R}$ which has the Feller property. This completes the proof. □

## 3.4 Extinction of the Disease

In this section, we establish the sufficient conditions for the complete eradication of the disease. To do so, we introduce a key parameter that determines the extinction threshold:

$$\mathcal{R}_0^{ex}=\sqrt[3]{\frac{\mathcal{R}_3}{2}+\sqrt{\left(\frac{\mathcal{R}_3}{2}\right)^2+\left(\frac{\mathcal{R}_1+\mathcal{R}_2}{3}\right)^3}}+\sqrt[3]{\frac{\mathcal{R}_3}{2}-\sqrt{\left(\frac{\mathcal{R}_3}{2}\right)^2+\left(\frac{\mathcal{R}_1+\mathcal{R}_2}{3}\right)^3}}. \tag{3.41}$$

The parameter $\mathcal{R}_0^{ex}$ represents the stochastic extinction reproduction number, which encapsulates the dynamics leading to the potential eradication of the disease. The following theorem formalizes the conditions for extinction:

**Theorem 3.3.**

*Consider the solution* $(S(t), E(t), I_u(t), I_r(t), R(t), \delta(t))$ *to system* (3.2) *with initial values* $(S(0), E(0), I_u(0), I_r(0), R(0), \delta(0)) \in \mathbb{R}_+^5 \times \mathbb{R}$. *Assume that, from the previous established theorem, the stochastic process* $(S(0), E(0), I_u(0), I_r(0), R(0), \delta(0))$ *admits a unique stationary distribution* $\Theta(.,.)$ *and is ergodic. If* $\mathcal{R}_0^{ex} < 1$ *and*

$$\psi = \min\left\{\sigma+\mu, \mu+d_1+\bar{\delta}+\gamma_{I_u}, \mu+d_2+\gamma_{I_r}\right\}(\mathcal{R}_0^{ex}-1)$$
$$+\left(\frac{v_1(\mu+d_1+\bar{\delta}+\gamma_{I_u})(\mu+d_2+\gamma_{I_r})\mathcal{R}_0^{ex}}{\mathcal{R}_0^{ex}(\mu+d_2+\gamma_{I_r})S_0v_1+\bar{\delta}S_0v_2}+\frac{(\mu+d_2+\gamma_{I_r})\mathcal{R}_0^{ex}}{S_0}\right)\int_0^{\infty}\int_{-\infty}^{\infty}|l-s_0|\Theta(l,\delta)dld\delta$$
$$+\left(\frac{v_2(\mu+d_1\bar{\delta}+\gamma_{I_u})\mathcal{R}_0^{ex}}{v_1(\mu+d_2+\gamma_{I_r})\mathcal{R}_0^{ex}+\bar{\delta}v_2}+1\right)\frac{\sigma_1}{\sqrt{\pi\rho_1}} \quad \textit{is negative,}$$

*then*

$$\lim_{t\to\infty} E(t) = 0, \quad \lim_{t\to\infty} I_u(t) = 0, \quad \lim_{t\to\infty} I_r(t) = 0 \quad a.s.$$

*Specifically, the disease undergoes exponential extinction almost surely.*

**Proof.** Based on the first equation of system (3.2), we have:

$$dS(t) \le (\Delta - \mu S)dt.$$

Let the following auxiliary logistical equation be

$$dL(t) = (\Delta - \mu L(t))dt. \tag{3.42}$$

Assume that $L(t)$ represents the solution of (3.42) with the initial condition $L(0) = S(0) > 0$. Referring to the theorem shown in [117], we obtain $S(t) \le L(t)$, for any $t \ge 0$ a.s.

According to the Perron–Frobenius theorem [118], the matrix

$$B = \begin{pmatrix} 0 & \frac{\beta S_0 v_1}{\sigma+\mu} & \frac{\beta S_0 v_2}{\sigma+\mu} \\ \frac{\sigma(1-\rho)}{\mu+d_1+\bar{\delta}+\gamma_{I_u}} & 0 & 0 \\ \frac{\sigma\rho}{\mu+d_2+\gamma_{I_r}} & \frac{\bar{\delta}}{\mu+d_2+\gamma_{I_r}} & 0 \end{pmatrix}$$

is non-negative and irreducible. Hence, it possesses a positive left eigenvector $(\vartheta_1, \vartheta_2, \vartheta_3)$ associated with its spectral radius, which is given by the basic reproduction number $\mathcal{R}_0^{ex}$.

In particular, $\mathcal{R}_0^{ex}$ is the dominant eigenvalue of $B$ and satisfies

$$\mathcal{R}_0^{ex}(\vartheta_1, \vartheta_2, \vartheta_3) = (\vartheta_1, \vartheta_2, \vartheta_3)B.$$

Define a $C^2$-lyapunov function $\dot{G}(E, I_u, I_r)$ by

$$\dot{G}(E, I_u, I_r) = \alpha_1 E + \alpha_2 I_u + \alpha_3 I_r,$$

where

$$\alpha_1 = \frac{\vartheta_1}{\sigma + \mu}, \alpha_2 = \frac{\vartheta_2}{\mu + d_1 + \bar{\delta} + \gamma_{I_u}}, \alpha_3 = \frac{\vartheta_3}{\mu + d_2 + \gamma_{I_r}}.$$

Which inplies

$$\begin{aligned}
\mathcal{L}(\ln \dot{G}) =& \frac{1}{\dot{G}}[\alpha_1(\beta(v_1 I_u + v_2 I_r) S - (\sigma + \mu)E) + \alpha_2(\sigma(1-\rho)E - \left(\mu + d_1 + \tilde{\delta} + \gamma_{I_u}\right) I_u) \\
&+ \alpha_3(\sigma\rho E + \tilde{\delta} I_u - \left(\mu + d_2 + \gamma_{I_r}\right) I_r)] \\
=& \frac{1}{\dot{G}}[\alpha_1(\beta(v_1 I_u + v_2 I_r) S_0 - (\sigma + \mu)E) + \alpha_2(\sigma(1-\rho)E - \left(\mu + d_1 + \bar{\delta} + \gamma_{I_u}\right) I_u) \\
&+ \alpha_3(\sigma\rho E + \bar{\delta} I_u - \left(\mu + d_2 + \gamma_{I_r}\right) I_r)] + \frac{\alpha_1(\beta(v_1 I_u + v_2 I_r)(S - S_0)}{\dot{G}} \\
&+ \frac{\alpha_2(\bar{\delta} - \tilde{\delta}) I_u}{\dot{G}} + \frac{\alpha_3(\tilde{\delta} - \bar{\delta}) I_u}{\dot{G}} \\
\leq& \frac{1}{\dot{G}}[\frac{\vartheta_1}{\sigma + \mu}(\beta(v_1 I_u + v_2 I_r) S_0 - (\sigma + \mu)E) \\
&+ \frac{\vartheta_2}{\mu + d_1 + \bar{\delta} + \gamma_{I_u}}(\sigma(1-\rho)E - \left(\mu + d_1 + \bar{\delta} + \gamma_{I_u}\right) I_u) \\
&+ \frac{\vartheta_3}{\mu + d_2 + \gamma_{I_r}}(\sigma\rho E + \bar{\delta} I_u - \left(\mu + d_2 + \gamma_{I_r}\right) I_r)] \\
&+ \frac{\alpha_1(\beta(v_1 I_u + v_2 I_r)(L - S_0)}{\dot{G}} + \frac{\alpha_2|\tilde{\delta} - \bar{\delta}| I_u}{\dot{G}} + \frac{\alpha_3|\tilde{\delta} - \bar{\delta}| I_u}{\dot{G}} \\
\leq& \frac{1}{\dot{G}}(\vartheta_1, \vartheta_2, \vartheta_3)(B(E, I_u, I_r)^T - (E, I_u, I_r)^T) \\
&+ \frac{\alpha_1(\beta(v_1 I_u + v_2 I_r)(L - S_0)}{\dot{G}} + \frac{(\alpha_2 + \alpha_3)|\tilde{\delta} - \bar{\delta}| I_u}{\dot{G}} \\
\leq& \frac{1}{\dot{G}}(\mathcal{R}_0^{ex} - 1)(\vartheta_1 E + \vartheta_2 I_u + \vartheta_3 I_r) + \frac{\alpha_1\beta(\alpha_3 v_1 + \alpha_2 v_2)}{\alpha_2\alpha_3}|L - S_0| + \frac{(\alpha_2 + \alpha_3)}{\alpha_2}|\tilde{\delta} - \bar{\delta}| \\
\leq& \frac{1}{\dot{G}}(\mathcal{R}_0^{ex} - 1)(\alpha_1(\sigma + \mu)E + \alpha_2(\mu + d_1\bar{\delta} + \gamma_{I_u}) I_u + \alpha_3(\mu + d_2 + \gamma_{I_r}) I_r) \\
&+ \frac{\alpha_1\beta(\alpha_3 v_1 + \alpha_2 v_2)}{\alpha_2\alpha_3}|L - S_0| + \frac{(\alpha_2 + \alpha_3)}{\alpha_2}|\tilde{\delta} - \bar{\delta}| \\
\leq& \min\left\{\sigma + \mu, \mu + d_1 + \bar{\delta} + \gamma_{I_u}, \mu + d_2 + \gamma_{I_r}\right\}(\mathcal{R}_0^{ex} - 1) + \chi_1|L - S_0| + \chi_2|\tilde{\delta} - \bar{\delta}|,
\end{aligned} \tag{3.43}$$

where

$$\chi_1 := \frac{v_1(\mu+d_1+\bar{\delta}+\gamma_{I_u})(\mu+d_2+\gamma_{I_r})\mathcal{R}^{ex}}{\mathcal{R}^{ex}(\mu+d_2+\gamma_{I_r})S_0v_1+\bar{\delta}S_0v_2} + \frac{(\mu+d_2+\gamma_{I_r})\mathcal{R}_0^{ex}}{S_0}$$

$$\chi_2 = \frac{v_2(\mu+d_1\bar{\delta}+\gamma_{I_u})\mathcal{R}_0^{ex}}{v_1(\mu+d_2+\gamma_{I_r})\mathcal{R}_0^{ex}+\bar{\delta}v_2}+1.$$

Upon integrating both sides of (3.43) over the interval $[0,t]$ and subsequently dividing by t, it follows that:

$$\begin{aligned}\frac{\ln \mathcal{G}(E(t),I_u(t),I_r(t))}{t} \leq & \frac{\ln \mathcal{G}(E(0),I_u(0),I_r(0))}{t} \\ & + \min\left\{\sigma+\mu,\mu+d_1+\bar{\delta}+\gamma_{I_u},\mu+d_2+\gamma_{I_r}\right\}(\mathcal{R}_0^{ex}-1) \\ & + \chi_1\frac{1}{t}\int_0^t |L(s)-S_0|ds + \chi_2\frac{1}{t}\int_0^t |\tilde{\delta}(s)-\bar{\delta}|ds.\end{aligned} \tag{3.44}$$

Considering Theorem 3.2 along with the strong law of large numbers [101, 102, 112, 115], we can conclude that the process$(L(t),\delta(t))$ possesses a distinct stationary distribution $\Theta(.,.)$ and exhibits the property of ergodicity so

$$\lim_{t\to\infty}\frac{1}{t}\int_0^t |L(s)-S_0|ds = \int_0^\infty\int_{-\infty}^\infty |l-S_0|\Theta(l,\delta)dld\delta \tag{3.45}$$

and

$$\lim_{t\to\infty}\frac{1}{t}\int_0^t |\tilde{\delta}(s)-\bar{\delta}|ds = \int_{-\infty}^\infty |\tilde{\delta}(y)-\bar{\delta}|f(y)dy = \frac{\sigma_1}{\sqrt{\pi\rho_1}}. \tag{3.46}$$

Applying the upper limit to both sides of (3.44) and consolidating it with (3.45) and (3.46), we arrive at

$$\begin{aligned}\limsup_{t\to\infty}\frac{\ln \mathcal{G}(E(t),I_u(t),I_r(t))}{t} \leq & \min\left\{\sigma+\mu,\mu+d_1+\bar{\delta}+\gamma_{I_u},\mu+d_2+\gamma_{I_r}\right\}(\mathcal{R}_0^{ex}-1) \\ & + \chi_1\int_0^\infty\int_{-\infty}^\infty |l-S_0|\Theta(l,\delta)dld\delta + \chi_2\frac{\sigma_1}{\sqrt{\pi\rho_1}} := \psi \quad \textbf{a.s.}\end{aligned}$$

and if $\psi$ is negative, we can infer that

$\limsup_{t\to\infty}\frac{\ln E(t)}{t} < 0, \limsup_{t\to\infty}\frac{\ln I_u(t)}{t} < 0$ and $\limsup_{t\to\infty}\frac{\ln I_r(t)}{t} < 0$ **a.s.**

This entails that

$\lim_{t\to\infty} E(t) = 0, \lim_{t\to\infty} I_u(t) = 0$ and $\lim_{t\to\infty} I_r(t) = 0$ **a.s.**

This finalizes the theorem's proof. □

## 3.5 Numerical simulations

Using the advanced technique outlined by Milstein in reference [119], the discretized equation corresponding to system (3.2) can be derived

$$\begin{cases} S[j+1] = S[j] + (\Delta - \beta(v_1 I_u[j] + v_2 I_r[j])S[j] - \mu S[j])\Delta t, \\ E[j+1] = E[j] + (\beta(v_1 I_u[j] + v_2 I_r[j])S[j] - (\sigma+\mu)E[j])\Delta t, \\ I_u[j+1] = I_u[j] + (\sigma(1-\rho)E[j] - (\mu + d_1 + \max(\delta[j],0) + \gamma_{Iu})I_u[j])\Delta t, \\ I_r[j+1] = I_r[j] + (\sigma\rho E[j] + \max(\delta[j],0)I_u[j] - (\mu + d_2 + \gamma_{Ir})I_r[j])\Delta t, \\ R[j+1] = R[j] + (\gamma_{Iu} I_u[j] + \gamma_{Ir} I_r[j] - \mu R[j])\Delta t, \\ \delta[j+1] = \delta[j] + \rho_1\left(\bar{\delta} - \delta[j]\right)\Delta t + \sigma_1\sqrt{\Delta t}\iota_j, \end{cases} \tag{3.47}$$

in accordance with the jth iteration of Equation (3.47), denoted as $(S[j],,E[j],I_u[j],,I_r[j],R[j],\delta[j])$, where $\Delta t$ represents the positive time increment, $\iota_j$ signifies independent Gaussian random variables adhering to the $N(0,1)$ distribution for $j = 1,...,n$. Realistic parameter values are selected from established sources, and the biological parameters are comprehensively listed in Table 3.2.
In this section, our primary focus is on confirming the validity of the following two outcomes:

1. The condition for $\mathcal{R}_0^s > 1$ leads to the existence of a distinctive ergodic stationary distribution.

2. System (3.2) undergoes exponential extinction when $\mathcal{R}_0^{ex} < 1$ and $\psi < 0$.

Table 3.2: List of parameters of stochastic system (3.2) .

| Parameters | Values | Source |
|---|---|---|
| $\Delta$ | 0.1 | Assumed |
| $\mu$ | 0.0399 | Assumed |
| $\beta$ | 0.6594 | [120] |
| $\rho$ | 0.2929 | [120] |
| $v_1$ | 0.3958 | [105] |
| $v_2$ | 0.4941 | [105] |
| $\bar{\delta}$ | 0.1 | Estimated |
| $d_1$ | 0.0290 | [120] |
| $d_2$ | 0.4897 | [120] |
| $\sigma$ | 0.5732 | [105] |
| $\gamma_{I_u}$ | 0.0458 | [120] |
| $\gamma_{I_r}$ | 0.0806 | [120] |

**Example 3.1.**

Given the values $\beta = 0.6594, \rho_1 = 0.1$ and $\sigma_1 = 0.02$, a calculation yields

$$\begin{aligned}\mathcal{R}_0^s = \frac{S_0}{\sigma+\mu}\Bigg(&\frac{\sigma\beta v_1(1-\rho)}{\bar{\delta}+\gamma_{I_u}+\mu+d_1}+\frac{\sigma\beta v_2\rho}{\gamma_{I_r}+\mu+d_2}\\&+\frac{\sigma\beta v_2(1-\rho)}{(\bar{\delta}+\gamma_{I_u}+\mu+d_1)(\gamma_{I_r}+\mu+d_2)}\left(\frac{1}{\sqrt{\pi}}\int_{-\frac{\bar{\delta}\sqrt{\rho_1}}{\sigma_1}}^{\infty}\left(\frac{\sigma_1}{\sqrt{\rho_1}}y+\bar{\delta}\right)^{\frac{1}{4}}e^{-y^2}dy\right)^4\Bigg)\\&-\Bigg(\frac{\sigma\beta v_1(1-\rho)\Delta}{\mu(\mu+d_1+\bar{\delta}+\gamma_{I_u})^2}\\&+\frac{\sigma\beta v_2(1-\rho)\Delta\left(\frac{1}{\sqrt{\pi}}\int_{-\frac{\bar{\delta}\sqrt{\rho_1}}{\sigma_1}}^{\infty}\left(\frac{\sigma_1}{\sqrt{\rho_1}}y+\bar{\delta}\right)^{\frac{1}{4}}e^{-y^2}dy\right)^4}{\mu(\mu+d_1+\bar{\delta}+\gamma_{I_u})^2(\mu+d_2+\gamma_{I_r})}\Bigg)\\&\times\frac{\sigma_1}{2\sqrt{\pi\rho_1}(\sigma+\mu)}\\\approx\ &2.54697>1.\end{aligned}$$

According to Theorem 3.2, we deduce the existence of a singular stationary distribution $\Theta(.)$ with ergodic properties. This is illustrated in Figure 3.1 and Figure 3.2.

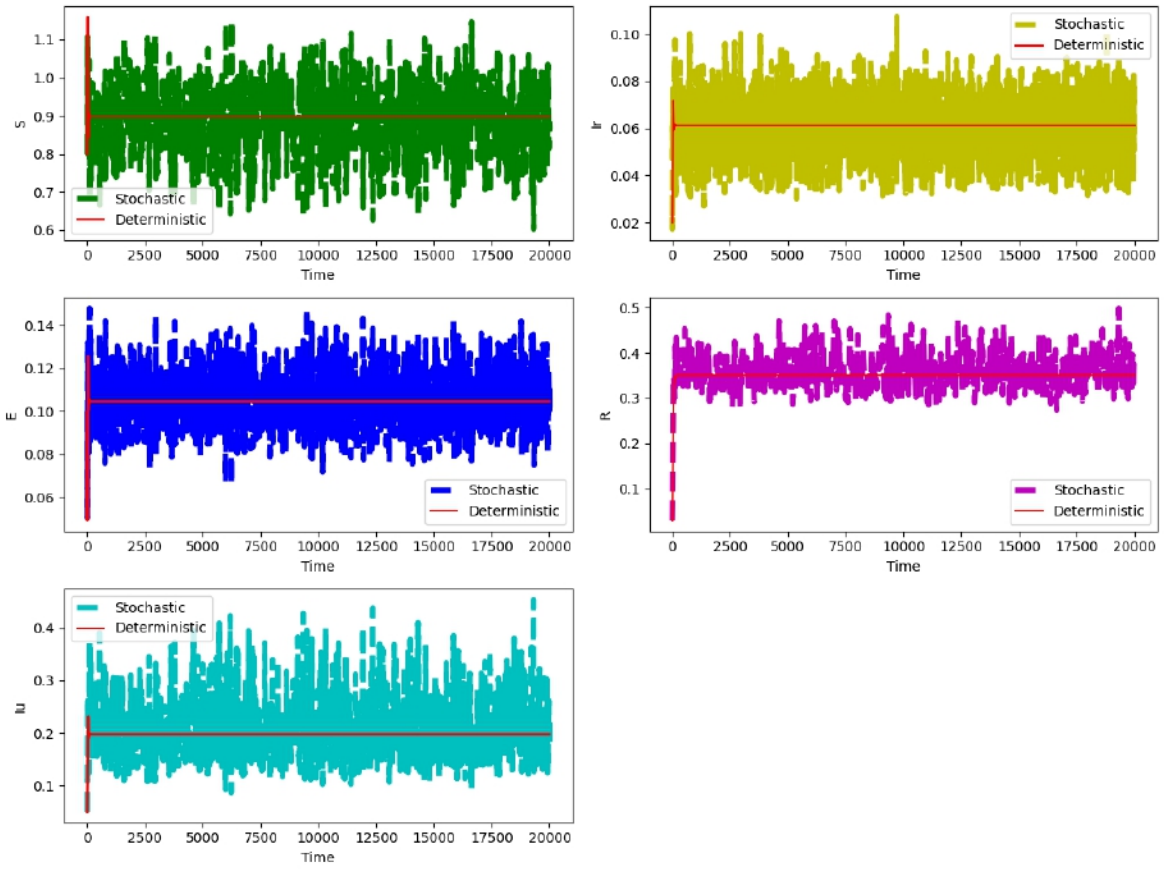



Figure 3.1: The illustration depicts the simulated evolution of the solution $(S(t), E(t), I_u(t), I_r(t), R(t))$ in systems (3.1) and (3.2) using the given parameter values $\beta = 0.6594, \rho_1 = 0.1$, and $\sigma_1 = 0.02$.

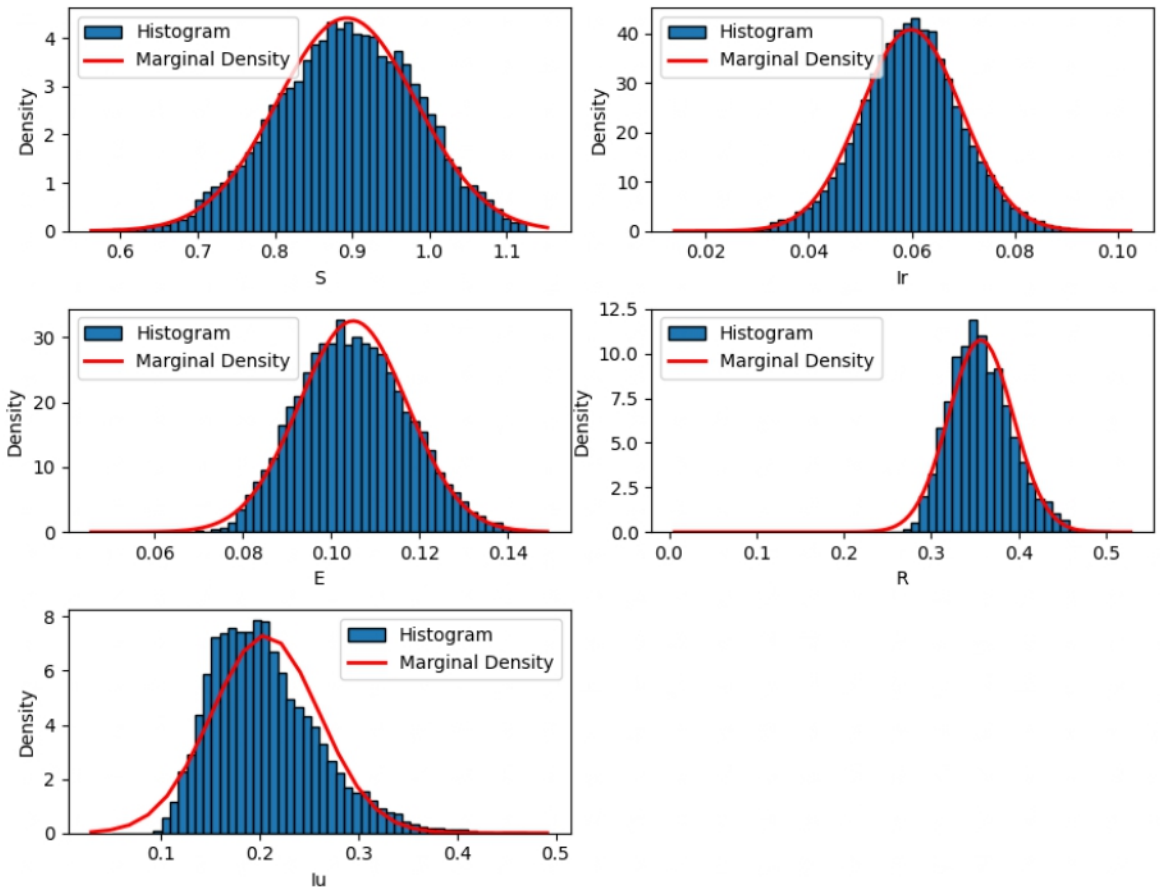



Figure 3.2: Frequency Histograms and Marginal Density Functions for Variables$S, E, I_u, I_r$, and $R$ within System (3.2).

**Example 3.2.**

Assume that $\beta = 0.2, \rho_1 = 4$ and $\sigma_1 = 0.08$, then we similarly compute that

$$\mathcal{R}_0^{ex} = \sqrt[3]{\frac{\mathcal{R}_3}{2} + \sqrt{(\frac{\mathcal{R}_3}{2})^2 + (\frac{\mathcal{R}_1 + \mathcal{R}_2}{3})^3}} + \sqrt[3]{\frac{\mathcal{R}_3}{2} - \sqrt{(\frac{\mathcal{R}_3}{2})^2 + (\frac{\mathcal{R}_1 + \mathcal{R}_2}{3})^3}} \approx 0.16 < 1$$

and

$$\begin{aligned}\psi =& \min\left\{\sigma + \mu, \mu + d_1 + \bar{\delta} + \gamma_{I_u}, \mu + d_2 + \gamma_{I_r}\right\}(\mathcal{R}_0^{ex} - 1) \\ &+ (\frac{v_1(\mu + d_1 + \bar{\delta} + \gamma_{I_u})(\mu + d_2 + \gamma_{I_r})\mathcal{R}^{ex}}{\mathcal{R}^{ex}(\mu + d_2 + \gamma_{I_r})S_0 v_1 + \bar{\delta} S_0 v_2} + \frac{(\mu + d_2 + \gamma_{I_r})\mathcal{R}_0^{ex}}{S_0}) \int_0^{\infty} \int_{-\infty}^{\infty} |l - S_0| \Theta(l, \delta) dl d\delta \\ &+ (\frac{v_2(\mu + d_1 \bar{\delta} + \gamma_{I_u})\mathcal{R}_0^{ex}}{v_1(\mu + d_2 + \gamma_{I_r})\mathcal{R}_0^{ex} + \bar{\delta} v_2} + 1)\frac{\sigma_1}{\sqrt{\pi \rho_1}} \approx -0.003.\end{aligned}$$

The implications of Theorem 3.3 readily demonstrate that the infected population will undergo exponential extinction, thereby guaranteeing the eradication of the disease outbreak. Figure 3.3 confirms this.

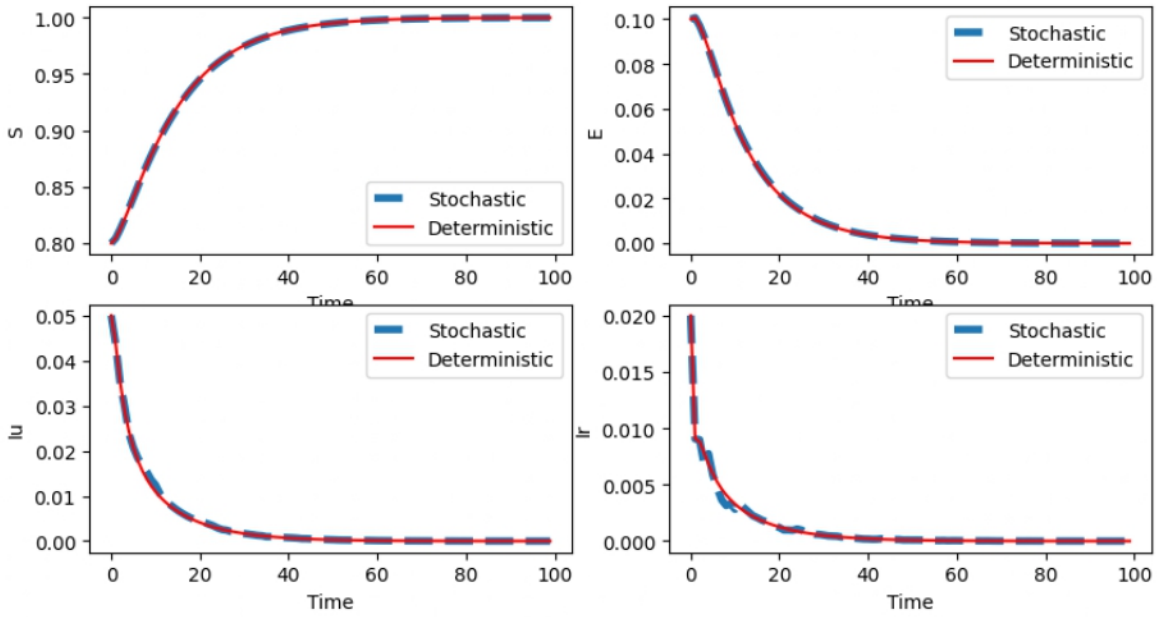



Figure 3.3: the simulations of the solution $(S(t), E(t), I_u(t), I_r(t), R(t))$ in deterministic system (3.1) and the stochastic system (3.2) with the parameter values $\beta = 0.2, \rho_1 = 4$, and $\sigma_1 = 0.08$.

## 3.6 Discussion

This study has delved into the development and analysis of a stochastic $SEI_uI_rR$ model for COVID-19, incorporating the Ornstein-Uhlenbeck process to capture

transmission rate dynamics. The findings have provided valuable insights into the intricate dynamics of the disease. Initially, the existence of a unique global solution to the stochastic model was rigorously established, ensuring its mathematical soundness and applicability.

Subsequently, stability conditions were examined using the Lyapunov stochastic function and Fatou's lemma, revealing strong stability of the disease dynamics in the presence of random perturbations. Furthermore, the conditions for complete disease eradication were explored through the comparison theory of one-dimensional stochastic differential equations and the theory of ergodicity, offering a theoretical foundation for potential disease control strategies.

This work highlights the importance of incorporating stochastic elements into mathematical modeling, emphasizing both the mathematical rigor and biological relevance of such approaches. Additionally, numerical simulations demonstrated the practical implications of the theoretical results, effectively bridging the gap between abstract modeling and real-world applications. These contributions not only enhance the mathematical understanding of COVID-19 dynamics but also provide potential strategies to mitigate the impacts of global health challenges.

Despite these advances, the current methodologies and the complexity of system (3.2) impose certain limitations, including the inability to establish a definitive disease eradication threshold. Future work will focus on extending this model to incorporate regime-switching dynamics, which may offer deeper insights into the stochastic behavior of the system. These challenges are deferred to forthcoming research endeavors, aiming to further refine the theoretical and practical

Chapter 4

# Stochastic SEIR Epidemic Dynamics with Holling Type II Treatment Under Ornstein–Uhlenbeck Perturbations

## 4.1 Introduction

The measles virus, a member of the Paramyxoviridae family, is the causative agent of a highly infectious respiratory disease. Symptoms typically include conjunctivitis, high fever, rhinitis, coughing, a maculopapular rash, and the hallmark Koplik's spots. While most cases resolve without complications within three weeks following an incubation period of 10 to 14 days, individuals with vitamin A deficiencies or malnutrition face a heightened risk of severe complications, including diarrhea, encephalitis, otitis media, and vision loss.

Despite the availability of an effective vaccine, measles remains a pressing global health challenge. It is a leading cause of morbidity and mortality among children under five years old. History has recorded significant outbreaks, such as the California epidemic from 1988 to 1990, which resulted in over 16,000 cases and more than 70 fatalities [121]. Similarly, Madagascar experienced a severe outbreak in 2018 that affected 50,000 people and caused 300 deaths, primarily among children [122]. In the same year, the World Health Organization (WHO) reported a staggering 140,000 deaths globally due to measles. While global vaccination initiatives have led to a 73% reduction in measles mortality between 2000 and 2018, the disease remains prevalent in many developing regions of Asia and Africa [123].

Epidemic models play a pivotal role in describing disease transmission dynamics and evaluating control measures. Over recent years, deterministic models have gained prominence for analyzing the complex behavior of measles and devising effective prevention and mitigation strategies. For instance, mathematical studies on pulse vaccination and its impact on controlling measles transmission have been explored in [124]. The importance of social distancing in curbing the spread of measles is highlighted in [123], while other deterministic models have assessed the influence of immunization and therapeutic interventions on measles dynamics [125, 126, 127, 128, 129]. Studies like [130] investigate the effects of quarantine and treatment strategies, with additional significant findings presented in [131].

The literature also features various case studies applying mathematical approaches to measles outbreaks. Notably, HW Berhe et al. [132] proposed an SEIR model incorporating optimal control strategies for managing measles epidemics within human

Table 4.1: Description of state parameters of the model (4.1) .

| Parameter | Description |
|---|---|
| $\Lambda$ | Recruitment rate of susceptible population by immigration or birth |
| $\beta$ | Effective contact rate of measles |
| $\theta$ | The rate of effective vaccination of susceptible humans |
| $\mu$ | Natural death rate of humans |
| $k_1$ | Extent of the effect of the infected being delayed for treatment |
| $k_2$ | Saturation constant which represents the effect of psychological and protection measure that protects the interaction of susceptible and infected humans |
| $\delta_1$ | rate of progress from exposed to infectious class |
| $\gamma$ | Natural recovery rate from measles |
| $\alpha$ | Recovery rate of measles infected class due to treatment |
| $\delta_2$ | Disease induced death rate of measles |

populations:

$$\begin{cases} \dfrac{dS(t)}{dt} = \Lambda - \dfrac{\beta S(t)I(t)}{1+k_1 I(t)} - (\mu+\theta)S(t), \\ \dfrac{dE(t)}{dt} = \dfrac{\beta S(t)I(t)}{1+k_1 I(t)} - (\delta_1+\mu)E(t), \\ \dfrac{dI(t)}{dt} = \delta_1 E(t) - \left(\mu+\delta_2+\gamma+\dfrac{\alpha}{1+k_2 I(t)}\right)I(t), \\ \dfrac{dR(t)}{dt} = \theta S(t) + (\gamma+\dfrac{\alpha}{1+k_2 I(t)})I(t) - \mu R(t), \end{cases} \tag{4.1}$$

At time $t$, the variables $S(t)$, $E(t)$, $I(t)$, and $R(t)$ denote the respective numbers of susceptible individuals, individuals who have been exposed to the disease without exhibiting clinical symptoms, asymptomatically infected individuals, currently infected individuals, and recovered individuals.

Throughout this work, we denote the time-dependent state variables of the system by $S(t), E(t), I(t), R(t)$. For simplicity of notation, we sometimes write $(S, E, I, R)$ instead of $(S(t), E(t), I(t), R(t))$ when no ambiguity arises.

All parameters are assumed to be positive constants, and their descriptions are given in Table 4.1. By employing the techniques outlined in the study by Ma et al. [133] and applying the generation matrix approach, it can be established that

system (4.1) possesses a positive invariant set[132]:

$$\mathcal{D} = \left\{(S,E,I,R) \in \mathbb{R}^4_+, 0 \leq S+E+I+R \leq \frac{\Lambda}{\mu}\right\},$$

and the formula for the basic reproduction number of system (4.1) is determined by

$$\mathcal{R}_0 = \frac{\beta\Lambda\delta_1}{(\mu+\theta)(\mu+\delta_1)(\mu+\delta_2+\gamma+\alpha)} = \frac{\beta S_0^{'}\delta_1}{(\mu+\delta_1)(\mu+\delta_2+\gamma+\alpha)},$$

where $S_0 = \frac{\Lambda}{\mu}, S_0^{'} = \frac{\Lambda}{\mu+\theta}$. In reference [132], researchers found that when $\mathcal{R}_0$ is less than 1, the system exhibits a locally asymptotically stable disease-free equilibrium $E_0 = (S_0, 0, 0, 0)$, while for $\mathcal{R}_0$ greater than 1, the model has a singular endemic equilibrium $E^* = (S^*, E^*, I^*, R^*)$ that is locally asymptotically stable.

To model the impact of environmental noise on the progression rate from the exposed to the infectious class ($\delta_1$) and the disease-induced mortality rate for measles ($\delta_2$), we assume that $\delta_1$ and $\delta_2$ are stochastic variables. These parameters exhibit inherent randomness and adhere to the following specified formulation [112]:

$$d\delta_1(t) = \rho_1[\bar{\delta_1} - \delta_1(t)]dt + \sigma_1 dB_1(t), \qquad d\delta_2(t) = \rho_2[\bar{\delta_2} - \delta_2(t)]dt + \sigma_2 dB_2(t).$$

In the context of the model, where $\bar{\delta_1}$ and $\bar{\delta_2}$ represent the long-time mean levels of infection rates, and $\rho_i (i = 1, 2)$ signify the speeds of reversion, the stochastic dynamics involve $B_i(t)$, which are independent standard Brownian motion parameters defined on a complete probability space $\left(\mathcal{D}, \mathcal{F}, \{\mathcal{F}_t\}_{t\geq 0}, \mathbf{P}\right)$ with a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ [112]. Additionally, $\sigma_i > 0 (i = 1, 2)$ represents the intensity of $B_i(t)$.

Respectively, it is evident that the Ornstein–Uhlenbeck processes $\delta_1$ and $\delta_2$ converge to limit distributions $\mathbb{N}(\bar{\delta_1}, \frac{\sigma_1^2}{2\rho_1})$ and $\mathbb{N}(\bar{\delta_2}, \frac{\sigma_2^2}{2\rho_2})$. In other words, the probability density functions of the limit distributions are expressed as [101, 113, 114, 134] : $\Upsilon_1(x) = \frac{\sqrt{\rho_1}}{\sqrt{\pi}\sigma_1} e^{\frac{-\rho_1(x-\bar{\delta_1})^2}{\sigma_1^2}}$ and $\Upsilon_2(x) = \frac{\sqrt{\rho_2}}{\sqrt{\pi}\sigma_2} e^{\frac{-\rho_2(x-\bar{\delta_2})^2}{\sigma_2^2}}$. In light of the principles outlined in the ergodic theorem [115], we acquire :

$$\begin{aligned}
\int_{-\infty}^{\infty} (x \vee 0)^{\frac{1}{3}} \Upsilon_i(x)dx &= \int_0^{\infty} y^{\frac{1}{3}} \Upsilon_i(x)dx \\
&= \int_0^{\infty} x^{\frac{1}{3}} \frac{\sqrt{\rho_i}}{\sqrt{\pi}\sigma_i} e^{-\frac{\rho_i(x-\bar{\delta_i})^2}{\sigma_i^2}} dx \\
&= \frac{1}{\sqrt{\pi}} \int_{-\frac{\bar{\delta_i}\sqrt{\rho_i}}{\sigma_i}}^{\infty} \left(\frac{\sigma_i}{\sqrt{\rho_i}} x + \bar{\delta_i}\right)^{\frac{1}{3}} e^{-x^2} dx (i = 1, 2).
\end{aligned} \tag{4.2}$$

We suppose that :

$$\widehat{\delta_i} = \left( \int_{-\infty}^{\infty} (x \vee 0)^{\frac{1}{3}} \Upsilon_i(x) dx \right)^3 \qquad (i = 1, 2).$$

In a parallel manner, considering the pair of stochastic differential equations :

$$dq(t) = \rho_1 q(t)dt + \sigma_1 dB_1(t), \qquad dr(t) = \rho_2 r(t)dt + \sigma_2 dB_1(t) (\textbf{with} : q(t) = \delta_1 - \bar{\delta_1}, r(t) = \delta_2 - \bar{\delta_2}).$$

It is straightforward to deduce that both $q(t)$ and $r(t)$ exhibit the ergodic property, leading to their weak convergence toward the invariant density

$$\bar{\Upsilon_1}(x) = \frac{\sqrt{\rho_1}}{\sqrt{\pi}\sigma_1} e^{-\frac{\rho_1 x^2}{\sigma_1^2}}, \bar{\Upsilon_2}(x) = \frac{\sqrt{\rho_2}}{\sqrt{\pi}\sigma_2} e^{-\frac{\rho_2 x^2}{\sigma_2^2}}, x \in \mathbb{R}.$$

Following the principles outlined in the ergodic theorem [115], we acquire :

$$\int_{-\infty}^{\infty} (x \vee 0) \bar{\Upsilon_1}(x) dx = \int_0^{\infty} x \Upsilon_1(x) dx = \int_0^{\infty} x \frac{\sqrt{\rho_1}}{\sqrt{\pi}\sigma_1} e^{-\frac{\rho_1 x^2}{\sigma_1^2}} dx = \frac{\sigma_1}{2\sqrt{\pi\rho_1}}, \tag{4.3}$$

and

$$\int_{-\infty}^{\infty} (x \vee 0) \bar{\Upsilon_2}(x) dx = \int_0^{\infty} x \Upsilon_2(x) dx = \int_0^{\infty} x \frac{\sqrt{\rho_2}}{\sqrt{\pi}\sigma_2} e^{-\frac{\rho_2 x^2}{\sigma_2^2}} dx = \frac{\sigma_2}{2\sqrt{\pi\rho_2}}. \tag{4.4}$$

In the customary approach, the stochastic model presumes that all parameters hold nonnegative values, allowing an investigation into the relevance of introducing positivity into the discourse. To assess this, the variable $\max\{\delta_1(t), 0\}$ is substituted for the variable $\delta_1$ and the variable $\max\{\delta_2(t), 0\}$ is substituted for the variable $\delta_2$ as outlined in reference [101, 135]. This modification leads to the derivation of the subsequent stochastic model in the following manner:

$$\begin{cases} dS(t) = [\Lambda - \dfrac{\beta SI}{1 + k_1 I} - (\mu + \theta)S]dt, \\ dE(t) = [\dfrac{\beta SI}{1 + k_1 I} - (\tilde{\delta_1}(t) + \mu)E]dt, \\ dI(t) = [\tilde{\delta_1}(t)E - \left(\mu + \tilde{\delta_2}(t) + \gamma + \dfrac{\alpha}{1 + k_2 I}\right) I]dt, \\ dR(t) = [\theta S + (\gamma + \dfrac{\alpha}{1 + k_2 I})I - \mu R]dt, \\ d\delta_1(t) = \rho_1[\bar{\delta_1} - \delta_1(t)]dt + \sigma_1 dB_1(t), \\ d\delta_2(t) = \rho_2[\bar{\delta_2} - \delta_2(t)]dt + \sigma_2 dB_2(t). \end{cases} \tag{4.5}$$

## 4.2 Existence and Uniqueness of the Global Solution

In the real-world scenario, it is essential for every subset of a model depicting an epidemic to maintain non-negativity for subsequent dynamic analysis. Consequently, the proof of the presence and singularity of a universally positive solution to the given system (4.5) becomes crucial in this context.

**Theorem 4.1.**
*For any initial value* $(S(0),E(0),I(0),R(0),\delta_1(0),\delta_2(0)) \in \mathbb{R}^4_+ \times \mathbb{R}^2$, *there is a unique solution* $(S(t),E(t),I(t),R(t),\delta_1(t),\delta_2(t))$ *to the system* (4.5) *on* $t \geq 0$ *and the solution will remain in* $\mathbb{R}^4_+ \times \mathbb{R}^2$ *almost surely (a.s.).*

**Proof.** We will employ the theories outlined in [112] to establish the existence and uniqueness of the global solution to system (4.5) for $t \geq 0$. Given that all coefficients of system (4.5) satisfy local Lipschitz conditions, for any initial value $(S(0),E(0),I(0),R(0),\delta_1(0),\delta_2(0)) \in \mathbb{R}^4_+ \times \mathbb{R}^2$, there exists a unique local solution $(S(t),E(t),I(t),R(t),\delta_1(t),\delta_2(t))$ on the interval $\left[0,\upsilon_\rho\right)$, where $\upsilon_\rho$ represents an explosion time [112]. To demonstrate the global nature of this solution, i.e., to verify $\upsilon_\rho = \infty$ almost surely (a.s.), we establish that for a sufficiently large integer $n_0 \geq 1$, $S(0),E(0),I(0),R(0),e^{\delta_1(0)}$, and $e^{\delta_2(0)}$ all lie within the interval $\left[\frac{1}{n_0},n_0\right)$. For each integer $n \geq n_0$, we define a stopping time as per [112] :

$$\upsilon_n = \inf\left\{t \in [0,\upsilon_\rho) : \min\left\{S(t),E(t),I(t),R(t),e^{\delta_1(t)},e^{\delta_2(t)}\right\} \leq \frac{1}{n} \right.$$
$$\left. \text{or} \quad \max\left\{S(t),E(t),I(t),R(t),e^{\delta_1(t)},e^{\delta_2(t)}\right\} \geq n\right\}.$$

Defining $\inf\emptyset$ as $\infty$ (with $\emptyset$ being the empty set), it is evident that $\upsilon_n$is monotonically increasing as s approaches infinity. Let $\upsilon_\infty$ be the limit of $\upsilon_n$. Consequently, we establish $\upsilon_\infty \leq \upsilon_\rho$ almost surely (a.s.). (i) In the scenario where $\upsilon_\infty = \infty$ a.s., it follows that $\upsilon_e = \infty$ a.s., implying that $(S(t),E(t),I(t),R(t),\delta_1(t),\delta_2(t)) \in \mathbb{R}^4_+ \times \mathbb{R}^2$ a.s. for all $t \geq 0$. (ii) If $\upsilon_\infty < \infty$, let's assume the existence of constants $t_1 > 0$ and $\varepsilon \in (0,1)$ such that $\mathbf{P}\{\upsilon_\infty \leq t_1\} > \varepsilon$. Thus, there exists an integer $n_1 \geq n_0$ such that :

$$\mathbf{P}\{\upsilon_\infty \leq t_1\} \geq \varepsilon \qquad \forall n \geq n_1. \tag{4.6}$$

Define a non-negative $C^2$–function $\mathcal{Q} : \mathbb{R}^4_+ \times \mathbb{R}^2 \to \mathbb{R}_+$ by :

$$\mathcal{Q}(S,E,I,R,\delta_1,\delta_2) = \Phi(S) + \Phi(E) + \Phi(I) + \Phi(R) + \frac{\delta_1^2}{2} + \frac{\delta_2^2}{2},$$

where $\Phi(z) = z - 1 - \ln z (\forall z > 0)$

Utilizing the Itô's formula , we obtain :

$$dQ(S,E,I,R,\delta_1,\delta_2) = \mathcal{L}Q(S,E,I,R,\delta_1,\delta_2)dt + (\sigma_1\delta_1 + \sigma_2\delta_2)dB(t), \tag{4.7}$$

where $\mathcal{L}Q$ is defined by :

$$\begin{aligned}
\mathcal{L}Q =& \Lambda + 4\mu + \theta + \gamma + \frac{\sigma_1^2}{2} + \frac{\sigma_2^2}{2} - \mu(S+E+I+R) \\
& \bar{\delta_1} + \bar{\delta_2} + \frac{\alpha}{1+k_1 I} - \frac{\Lambda}{S} + \frac{\beta I}{1+k_1 I} - \frac{\beta SI}{(1+k_1 I)E} - \frac{\bar{\delta_1} E}{I} \\
& - \frac{\theta S}{R} - \frac{\gamma I}{R} - \frac{\alpha I}{(1+k_2 I)R} - \bar{\delta_2} I + \rho_1 \bar{\delta_1}\delta_1 - \rho_1\delta_1^2 + \rho_2\bar{\delta_2}\delta_2 - \rho_2\delta_2^2 \\
\leq & \Lambda + 4\mu + \theta + \gamma + \frac{\sigma_1^2}{2} + \frac{\sigma_2^2}{2} + |\delta_1| + |\delta_2| + \frac{\alpha}{1+k_1 I} + \frac{\beta I}{1+k_1 I} \\
& + \rho_1\bar{\delta_1}\delta_1 - \rho_1\delta_1^2 + \rho_2\bar{\delta_2}\delta_2 - \rho_2\delta_2^2.
\end{aligned} \tag{4.8}$$

Moreover, we acquire

$$\begin{aligned}
d(S+E+I+R) =& [\Lambda - \mu(S+E+I+R) - \bar{\delta_2} I]dt \\
\leq & [\Lambda - \mu(S+E+I+R)]dt,
\end{aligned}$$

indicating that

$$\begin{aligned}
S(t)+E(t)+I(t)+R(t) \leq & \begin{cases} S(0)+E(0)+I(0)+R(0), \text{ if } S(0)+E(0)+I(0)+R(0) \geq \dfrac{\Lambda}{\mu}, \\ \dfrac{\Lambda}{\mu}, \text{ if } S(0)+E(0)+I(0)++R(0) < \dfrac{\Lambda}{\mu} \end{cases} \\
\leq & \varpi_1,
\end{aligned} \tag{4.9}$$

where

$$\varpi_1 := \sup\left\{S(0)+E(0)+I(0)+R(0), \frac{\Lambda}{\mu}\right\}.$$

Substituting expressions (4.9) into (4.8) yields the following :

$$\begin{aligned}
\mathcal{L}Q \leq & \Lambda + 4\mu + \theta + \gamma + \frac{\sigma_1^2}{2} + \frac{\sigma_2^2}{2} + \alpha + \frac{\beta\varpi_1}{1+k_1\varpi_1} \\
& + \sup_{\delta_1,\delta_2\in\mathbb{R}} \left\{|\delta_1| + |\delta_2| + \rho_1\bar{\delta_1}\delta_1 - \rho_1\delta_1^2 + \rho_2\bar{\delta_2}\delta_2 - \rho_2\delta_2^2\right\} \\
:= & \varpi_2.
\end{aligned} \tag{4.10}$$

Here, $\varpi_2$ is a constant that satisfies a condition independent of $S, E, I, R, \delta_1, \delta_2$ and $t$. The remaining steps of the proof closely follow those outlined in Theorem 3.1 and are consequently omitted. This concludes the proof of Theorem 4.1. □

**Remark 4.1.**

By examining the proof of Theorem 4.1, it is evident that if the initial conditions $S(0)+E(0)+I(0)+R(0)<\frac{\Lambda}{\mu}$, then the set

$$\mathbf{D}=\left\{(S,E,I,R,\delta_1,\delta_2)\in\mathbb{R}_+^4\times\mathbb{R}^2, 0\leq S+E+I+R\leq\frac{\Lambda}{\mu}\right\}$$

remains positively invariant for the system (4.5). Therefore, moving forward, we consistently assume that the initial values $(S(0),E(0),I(0),R(0),\delta_1(0),\delta_2(0))$ of the system (4.5) fall within the designated region **D**. This assumption implies that the unique global solution $(S(t),E(t),I(t),R(t),\delta_1(t),\delta_2(t))$ to the system (4.5) will also, with probability one, reside within the region **D**.

## 4.3 Existence of a stationary solution

In this section, should the initial values $(S(0),E(0),I(0),R(0),\delta_1(0),\delta_2(0))$ belong to $\mathbb{R}_+^4\times\mathbb{R}^2$, we will proceed to establish additional conditions ensuring the stationary nature of the solution. Put differently, the system (4.5) supports the existence of at least one stationary Markov process on $\mathbb{R}_+^4\times\mathbb{R}^2$, signifying the persistence of the disease in the long term. Prior to this analysis, we introduce a stochastic critical value $\mathcal{R}_0^s$, linked to $\mathcal{R}_0$ as follows:

$$\begin{aligned}\mathcal{R}_0^s=&\frac{\Lambda\beta(\frac{1}{\sqrt{\pi}}\int_{-\frac{\bar{\delta_1}\sqrt{\rho_1}}{\sigma_1}}^{\infty}\left(\frac{\sigma_1}{\sqrt{\rho_1}}x+\bar{\delta_1}\right)^{\frac{1}{3}}e^{-x^2}dx)^{\frac{1}{3}}}{(\mu+\theta)\left(\mu+\bar{\delta_1}\right)\left(\mu+\bar{\delta_2}+\gamma+\alpha\right)}-\frac{\sigma_1}{2\sqrt{\pi\rho_1}(\mu+\bar{\delta_1})}\\&-\frac{\Lambda\beta(\frac{1}{\sqrt{\pi}}\int_{-\frac{\bar{\delta_1}\sqrt{\rho_1}}{\sigma_1}}^{\infty}\left(\frac{\sigma_1}{\sqrt{\rho_1}}x+\bar{\delta_1}\right)^{\frac{1}{3}}e^{-x^2}dx)^{\frac{1}{3}}}{(\mu+\theta)(\mu+\bar{\delta_1}+\gamma+\alpha)^2}\frac{\sigma_2}{2\sqrt{\pi\rho_2}(\mu+\bar{\delta_1})}.\end{aligned}$$

**Theorem 4.2.**

*For any initial values* $(S(0),E(0),I(0),R(0),\delta_1(0),\delta_2(0))^T\in\mathbb{R}_+^4\times\mathbb{R}^2$, *if* $\mathcal{R}_0^s$ *is greater than* 1, *system* (4.5) *is guaranteed to possess at least one stationary solution* $(S(t),E(t),I(t),R(t),\delta_1(t),\delta_2(t))^T$ *on* $\mathbb{R}_+^4\times\mathbb{R}^2$.

**Proof.** As per Remark 4.1, it follows that $(S(t),E(t),I(t),R(t),\delta_1(t),\delta_2(t))^T$ belongs to **D** almost surely. Consequently, all references to $\mathbb{R}^d$ in Lemma 2.33 should be replaced with **D** for the system (4.5). The proof process is delineated into three

steps: the initial two steps involve constructing a nonnegative $C^2$–function $\mathcal{V}(S(t),E(t),I(t),R(t),\delta_1(t),\delta_2(t))$ and a compact set $\mathcal{W}\subset\mathbf{D}$, ensuring $\mathcal{L}\mathcal{V}\leq -1$ for all $(S,E,I,R,\delta_1,\delta_2)^T\in\mathbf{D}\setminus\mathcal{W}$. The final step entails establishing the existence of a stationary distribution for system (4.5) utilizing Theorem 2.2 from Du et al. [136].

- **Step 1 (Formation of a nonnegative $C^2$–function):** Define a $C^2$– function $\bar{\mathcal{V}}:\mathbf{D}\to\mathbb{R}$ as follows :

$$\bar{\mathcal{V}}(S,E,I,R,\delta_1,\delta_2)=\tilde{M}\mathcal{V}_1(S,E,I,R)+\mathcal{V}_2(S,E,I,R)+\mathcal{V}_3(S,E,I,R)+\mathcal{V}_4(\delta_1,\delta_2).$$

Here, $a_1$ and $a_2$ represent positive constants to be determined later, and $\bar{\mathcal{M}}$ is a suitably large positive constant that adheres to the specified condition:

$$-\tilde{M}(\bar{\delta_1}+\mu)(\mathcal{R}_0^s-1)+\varpi_4\leq -2 \tag{4.11}$$

and

$$\begin{aligned}\varpi_3:=&\sup_{\delta_1,\delta_2\in\mathbb{R}}\left\{-\frac{\rho_1}{2}\delta_1^2+|\delta_1|+\rho_1\bar{\delta_1}\delta_1-\frac{\rho_2}{2}\delta_2^2+|\delta_2|+\rho_2\bar{\delta_2}\delta_2\right\}+a_1\theta S_0\\&+\frac{a_1\alpha S_0\tilde{M}}{1+k_2S_0}+\frac{a_1\beta S_0}{(1+k_1S_0)}\\&+4\mu+\theta+\gamma+\alpha+\frac{\beta S_0}{1+k_1S_0}+\frac{\sigma_1^2}{2}+\frac{\sigma_2^2}{2}<\infty,\end{aligned} \tag{4.12}$$

$$\begin{aligned}\varpi_4:=&\sup_{\delta_1,\delta_2\in\mathbb{R}}\left\{-\frac{\rho_1}{2}\delta_1^2+|\delta_1|+\rho_1\bar{\delta_1}\delta_1\right.\\&\left.-\frac{\rho_2}{2}\delta_2^2+|\delta_2|+\rho_2\bar{\delta_2}\delta_2\right\}\\&+a_1\theta S_0+a_1\gamma S_0+\frac{a_1\alpha S_0\tilde{M}}{1+k_2S_0}\\&+\frac{a_1\beta S_0}{1+k_1S_0}+4\mu+\theta+\gamma+\alpha\\&+\frac{\beta S_0}{1+k_1S_0}+\frac{\sigma_1^2}{2}+\frac{\sigma_2^2}{2}<\infty.\end{aligned} \tag{4.13}$$

$$\varpi_5=\sup_{\delta_1\in\mathbb{R}}\{|\delta_1|\}<\infty,\varpi_6=\sup_{\delta_2\in\mathbb{R}}\{|\delta_2|\}<\infty$$

$$\mathcal{V}_1(S,E,I,R)=-\ln E-a_1\ln S-a_2\ln I-a_1R,$$

$$\mathcal{V}_2(S,E,I,R)=-\ln S-\ln E-\ln I,$$

$$\mathcal{V}_3(S,E,I,R)=-\ln\left(\frac{\Lambda}{\mu}-S-E-I-R\right),\mathcal{V}_4(\delta_1,\delta_2)=\frac{\delta_1^2}{2}+\frac{\delta_2^2}{2}.$$

Clearly, $\bar{V}(S(t),E(t),I(t),R(t),\delta_1(t),\delta_2(t))$ is a continuously differentiable function and attains its minimum at the point $(S^0,E^0,I^0,R^0,\delta_1^0,\delta_2^0)$; thus, we define a non-negative $C^2-$ function $\mathcal{V}:\mathbf{D}\to\bar{\mathbb{R}}$ as follows :

$$\mathcal{V}(S,E,I,R,\delta_1,\delta_2)=\bar{\mathcal{V}}(S,E,I,R,\delta_1,\delta_2)-\bar{\mathcal{V}}\left(S^0,E^0,I^0,R^0,\delta_1^1,\delta_2^0\right)^T.$$

Utilizing Itô's formula [112] for $V_1$, we obtain

$$\begin{aligned}
\mathcal{L}\mathcal{V}_1 =& -(\frac{a_1}{S})(\Lambda-\frac{\beta SI}{1+k_1I}-(\mu+\theta)S)-(\frac{1}{E})(\frac{\beta SI}{1+k_1I}-(\tilde{\delta_1}(t)+\mu)E)\\
&-(\frac{a_2}{I})(\tilde{\delta_1}(t)E-\left(\mu+\tilde{\delta_2}(t)+\gamma+\frac{\alpha}{1+k_2I}\right)I)\\
&+a_1(\theta S+(\gamma+\frac{\alpha}{1+k_2I})I-\mu R)\\
\leq&(-\frac{a_1\Lambda}{S}-\frac{\beta SI}{(1+k_1I)E}-\frac{a_2\tilde{\delta_1}E}{I})+a_1(\mu+\theta)+a_2(\mu+\bar{\delta_2}+\gamma+\alpha)\\
&+(\mu+\bar{\delta_1})+\frac{a_1\beta SI}{(1+k_1I)S}+\tilde{\delta_1}-\bar{\delta_1}+a_2\bar{\delta_2}-a_2\bar{\delta_2}\\
&+a_1\theta S+a_1\gamma I+\frac{a_1\alpha S_0}{1+k_2S_0}\\
\leq&-3\sqrt[3]{a_1a_2\Lambda\beta\tilde{\delta_1}}+a_1(\mu+\theta)+a_2(\mu+\bar{\delta_1}+\gamma+\alpha)+(\mu+\bar{\delta_1})+\frac{a_1\beta I}{(1+k_1I)}\\
&+q(t)\vee 0+a_2r(t)\vee 0+a_1\theta S+a_1\gamma I+\frac{a_1\alpha S_0}{1+k_2S_0}.
\end{aligned}\tag{4.14}$$

Replacing (4.2), (4.3) and (4.4) into (4.14) leads to that

$$\begin{aligned}
\mathcal{L}\mathcal{V}_1 \leq&-3\sqrt[3]{a_1a_2\Lambda\beta\widehat{\delta_1}}+(3\sqrt[3]{a_1a_2\Lambda\beta\widehat{\delta_1}}-3\sqrt[3]{a_1a_2\Lambda\beta\tilde{\delta_1}})+a_1(\mu+\theta)\\
&+a_2(\mu+\bar{\delta_1}+\gamma+\alpha)+(\mu+\bar{\delta_1})+\frac{\sigma_1}{2\sqrt{\pi\rho_1}}\\
&+(q(t)\vee 0-\int_0^\infty x\bar{\Upsilon}_1(x)dx)+a_2(r(t)\vee 0-\int_0^\infty x\bar{\Upsilon}_2(x)dx)\\
&+a_1\theta S+a_1\gamma I+\frac{a_1\alpha S_0}{1+k_2S_0}+\frac{a_1\beta I}{(1+k_1I)}+a_2\frac{\sigma_2}{2\sqrt{\pi\rho_2}}.
\end{aligned}\tag{4.15}$$

Let :

$$a_1=\frac{\Lambda\beta\widehat{\delta_1}}{(\mu+\theta)^2(\mu+\bar{\delta_1}+\gamma+\alpha)},\qquad a_2=\frac{\Lambda\beta\widehat{\delta_1}}{(\mu+\theta)(\mu+\bar{\delta_1}+\gamma+\alpha)^2}$$

and thus

$$\begin{aligned}\mathcal{L}\mathcal{V}_1 \leq & -\frac{\Lambda\beta\widehat{\delta_1}}{(\mu+\theta)(\mu+\bar{\delta_1}+\gamma+\alpha)}+(\mu+\bar{\delta_1})+\frac{\sigma_1}{2\sqrt{\pi\rho_1}}+a_2\frac{\sigma_2}{2\sqrt{\pi\rho_2}}\\ &+3\sqrt[3]{a_1a_2\Lambda\beta}(\sqrt[3]{\widehat{\delta_1}}-\sqrt[3]{\tilde{\delta_1}})+(q(t)\vee 0-\int_0^\infty x\bar{\Upsilon_1}(x)dx)+a_2(r(t)\vee 0\\ &-\int_0^\infty x\bar{\Upsilon_2}(x)dx)+a_1\theta S+a_1\gamma I+\frac{a_1\alpha S_0}{1+k_2S_0}+\frac{a_1\beta I}{(1+k_1I)}.\\ :=&-(\mu+\bar{\delta_1})(\mathcal{R}_0^s-1)+3\sqrt[3]{a_1a_2\Lambda\beta}(\sqrt[3]{\widehat{\delta_1}}-\sqrt[3]{\tilde{\delta_1}})+(q(t)\vee 0-\int_0^\infty x\Upsilon_1(x)dx)\\ &+a_2(r(t)\vee 0-\int_0^\infty x\Upsilon_2(x)dx)+a_1\theta S+a_1\gamma I+\frac{a_1\alpha S_0}{1+k_2S_0}+\frac{a_1\beta I}{(1+k_1I)}.\end{aligned} \tag{4.16}$$

Likewise, employing Itô's formula on $\mathcal{V}_2, \mathcal{V}_3$ and $\mathcal{V}_4$, respectively, yields the following.

$$\begin{aligned}\mathcal{L}\mathcal{V}_2 =&-\frac{\Lambda}{S}-\frac{\beta SI}{(1+k_1I)E}-\frac{\tilde{\delta_1}E}{I}+3\mu+\theta+\tilde{\delta_1}+\tilde{\delta_2}+\gamma+\frac{\alpha}{1+k_2I}+\frac{\beta I}{1+k_1I}\\ \leq&-\frac{\Lambda}{S}-\frac{\beta SI}{(1+k_1I)E}-\frac{\tilde{\delta_1}E}{I}+3\mu+\theta+|\delta_1|+|\delta_2|+\gamma+\alpha+\frac{\beta S_0}{1+k_1S_0}.\end{aligned} \tag{4.17}$$

$$\begin{aligned}\mathcal{L}\mathcal{V}_3 =&\frac{\Lambda+\mu(-S-E-I-R)-\tilde{\delta_2}I}{\frac{\Lambda}{\mu}-S-E-I-R}\\ \leq&\mu-\frac{\tilde{\delta_2}I}{\frac{\Lambda}{\mu}-S-E-I-R}\end{aligned} \tag{4.18}$$

and

$$\mathcal{L}\mathcal{V}_4=\rho_1\bar{\delta_1}\delta_1-\rho_1\delta_1^2+\frac{\sigma_1^2}{2}+\rho_2\bar{\delta_2}\delta_2-\rho_2\delta_2^2+\frac{\sigma_2^2}{2}. \tag{4.19}$$

By (4.16),(4.17), (4.18) and (4.19), we get:

$$\begin{aligned}
\mathcal{L}\mathcal{V} \leq & -\tilde{M}(\mu+\bar{\delta_1})(\mathcal{R}_0^s-1)+3\tilde{M}\sqrt[3]{a_1a_2\Lambda\beta}(\sqrt[3]{\widehat{\delta_1}}-\sqrt[3]{\bar{\delta_1}}) \\
& +\tilde{M}(q(t)\vee 0-\int_0^\infty x\Upsilon_1(x)dx) \\
& +a_2\tilde{M}(r(t)\vee 0-\int_0^\infty x\Upsilon_2(x)dx)+a_1\theta S+a_1\gamma I+\frac{a_1\alpha S_0\tilde{M}}{1+k_2S_0}+\frac{a_1\beta I}{(1+k_1I)} \\
& -\frac{\Lambda}{S}-\frac{\beta SI}{(1+k_1I)E}-\frac{\tilde{\delta_1}E}{I}+4\mu+\theta+|\delta_1|+|\delta_2|+\gamma+\alpha+\frac{\beta S_0}{1+k_1S_0} \\
& -\frac{\tilde{\delta_2}I}{\frac{\Lambda}{\mu}-S-E-I-R}+\rho_1\bar{\delta_1}\delta_1-\rho_1\delta_1^2+\frac{\sigma_1^2}{2}+\rho_2\bar{\delta_2}\delta_2-\rho_2\delta_2^2+\frac{\sigma_2^2}{2} \\
:=&\mathbf{Z}(S,E,I,R;\delta_1,\delta_2)+3\tilde{M}\sqrt[3]{a_1a_2\Lambda\beta}(\sqrt[3]{\widehat{\delta_1}}-\sqrt[3]{\bar{\delta_1}})+\tilde{M}(q(t)\vee 0 \\
& -\int_0^\infty x\bar{\Upsilon}_1(x)dx)+a_2\tilde{M}(r(t)\vee 0-\int_0^\infty x\bar{\Upsilon}_2(x)dx),
\end{aligned} \tag{4.20}$$

where :

$$\begin{aligned}
\mathbf{Z}(S,E,I,R;\delta_1,\delta_2)=&-\tilde{M}(\mu+\bar{\delta_1})(\mathcal{R}_0^s-1)++a_1\theta S+a_1\gamma I+\frac{a_1\alpha S_0\tilde{M}}{1+k_2S_0}+\frac{a_1\beta I}{(1+k_1I)} \\
& -\frac{\Lambda}{S}-\frac{\beta SI}{(1+k_1I)E}-\frac{\tilde{\delta_1}E}{I}+4\mu+\theta+|\delta_1|+|\delta_2|+\gamma+\alpha+\frac{\beta S_0}{1+k_1S_0} \\
& -\frac{\tilde{\delta_2}I}{\frac{\Lambda}{\mu}-S-E-I-R}+\rho_1\bar{\delta_1}\delta_1-\rho_1\delta_1^2+\frac{\sigma_1^2}{2}+\rho_2\bar{\delta_2}\delta_2-\rho_2\delta_2^2+\frac{\sigma_2^2}{2}.
\end{aligned} \tag{4.21}$$

- **Step 2 (Formation of a bounded set):** Define abounded closed set $\mathcal{W}_\epsilon$ by :

$$\mathcal{W}_\epsilon=\left\{(S,E,I,R,\delta_1,\delta_2)^T\in\mathbf{D}\;\middle|\;\begin{array}{l} S\geq\epsilon,\quad E\geq\epsilon^2,\quad I\geq\epsilon^3, \\ S+E+I+R\leq\dfrac{\Lambda}{\mu}-\epsilon^2, \\ |\delta_1|\leq\dfrac{1}{\epsilon},\quad |\delta_2|\leq\dfrac{1}{\epsilon}\end{array}\right\}.$$

where $\epsilon$ is a positive constant chosen to be small enough to meet the following conditions :

$$-\frac{\Lambda}{\epsilon}+\omega_4\leq -1 \tag{4.22}$$

$$-\omega_5\frac{1}{\epsilon}+a_1\gamma\epsilon<1 \tag{4.23}$$

$$-\frac{\beta S_0}{(1+k_1S_0)\epsilon}+\omega_4\leq -1 \tag{4.24}$$

$$-\varpi_6\frac{1}{\epsilon}+\varpi_4\leq -1 \tag{4.25}$$

$$-\frac{\rho_1}{2\epsilon^2}+\varpi_4\leq -1. \tag{4.26}$$

$$-\frac{\rho_2}{2\epsilon^2}+\varpi_4\leq -1. \tag{4.27}$$

We can partition the set $\mathbf{D}\setminus\mathcal{W}_\epsilon$ into the following seven subsets $\mathcal{W}^c_{\epsilon,i}$, where $i=1,...,6$, such that :

$$\mathcal{W}^c_{\epsilon,1}=\Big\{(S,E,I,R,\delta_1,\delta_2)^T\in\mathbf{D},S<\epsilon\Big\},$$

$$\mathcal{W}^c_{\epsilon,2}=\Big\{(S,E,I,R,\delta_1,\delta_2)^T\in\mathbf{D},I<\epsilon\Big\},$$

$$\mathcal{W}^c_{\epsilon,3}=\Big\{(S,E,I,R,\delta_1,\delta_2)^T\in\mathbf{D},E<\epsilon^2,S\geq\epsilon,\Big\},$$

$$\mathcal{W}^c_{\epsilon,4}=\left\{(S,E,I,R,\delta_1,\delta_2)^T\in\mathbf{D},S+E+I+R>\frac{\Lambda}{\mu}-\epsilon^2,I\geq\epsilon\right\},$$

$$\mathcal{W}^c_{\epsilon,5}=\left\{(S,E,I,R,\delta_1,\delta_2)^T\in\mathbf{D},|\delta_1|>\frac{1}{\epsilon}\right\},$$

$$\mathcal{W}^c_{\epsilon,6}=\left\{(S,E,I,R,\delta_1,\delta_2)^T\in\mathbf{D},|\delta_2|>\frac{1}{\epsilon}\right\}.$$

Hence we have :

– **Case 1 :** For any $(S,E,I,R,\delta_1,\delta_2)^T\in\mathcal{W}^c_{\epsilon,1}$, From (4.21) we have :

$$\begin{aligned}\mathbf{Z}(S,E,I,R;\delta_1,\delta_2)\leq&-\frac{\Lambda}{S}+a_1\theta S_0+a_1\gamma S_0+\frac{a_1\alpha S_0\tilde{M}}{1+k_2S_0}+\frac{a_1\beta S_0}{(1+k_1S_0)}\\&+4\mu+\theta+|\delta_1|+|\delta_2|+\gamma+\alpha+\frac{\beta S_0}{1+k_1S_0}\\&+\rho_1\bar{\delta_1}\delta_1-\frac{\rho_1}{2}\delta_1^2+\frac{\sigma_1^2}{2}+\rho_2\bar{\delta_2}\delta_2-\frac{\rho_2}{2}\delta_2^2+\frac{\sigma_2^2}{2}\\\leq&\frac{-\Lambda}{\epsilon}+\varpi_4\\\leq&-1,\end{aligned} \tag{4.28}$$

as a consequence of (4.22).

– **Case 2 :** For any $(S,E,I,R,\delta_1,\delta_2)^T \in \mathcal{W}^c_{\epsilon,2}$, From (4.21) we obtain :

$$\begin{aligned}
\mathbf{Z}(S,E,I,R;\delta_1,\delta_2) \leq & -\tilde{M}(\mu+\bar{\delta_1})(\mathcal{R}_0^s-1)++a_1\theta S+a_1\gamma I+\frac{a_1\alpha S_0\tilde{M}}{1+k_2S_0}+\frac{a_1\beta I}{(1+k_1I)}\\
& -\frac{\beta SI}{(1+k_1I)E}-\frac{\bar{\delta_1}E}{I}+4\mu+\theta+|\delta_1|+|\delta_2|+\gamma+\alpha+\frac{\beta S_0}{1+k_1S_0}\\
& +\rho_1\bar{\delta_1}\delta_1-\frac{\rho_1}{2}\delta_1^2+\frac{\sigma_1^2}{2}+\rho_2\bar{\delta_2}\delta_2-\frac{\rho_2}{2}\delta_2^2+\frac{\sigma_2^2}{2}\\
\leq & -\tilde{M}(\mu+\bar{\delta_1})(\mathcal{R}_0^s-1)++a_1\theta S_0+a_1\gamma\epsilon-\frac{\varpi_5S_0}{\epsilon}+\frac{a_1\alpha S_0\tilde{M}}{1+k_2S_0}\\
& +\frac{a_1\beta S_0}{(1+k_1S_0)}+4\mu+\theta+|\delta_1|+|\delta_2|+\gamma+\alpha+\frac{\beta S_0}{1+k_1S_0}\\
& +\rho_1\bar{\delta_1}\delta_1-\frac{\rho_1}{2}\delta_1^2+\frac{\sigma_1^2}{2}+\rho_2\bar{\delta_2}\delta_2-\frac{\rho_2}{2}\delta_2^2+\frac{\sigma_2^2}{2}.\\
\leq & -\tilde{M}(\mu+\bar{\delta_1})(\mathcal{R}_0^s-1)+a_1\gamma\epsilon-\frac{\varpi_5S_0}{\epsilon}+\varpi_3\\
\leq & -1,
\end{aligned} \tag{4.29}$$

as a consequence of (4.11) and (4.23).

– **Case 3 :** For any $(S,E,I,R,\delta_1,\delta_2)^T \in \mathcal{W}^c_{\epsilon,3}$, according to (4.21), we get :

$$\begin{aligned}
\mathbf{Z}(S,E,I,R;\delta_1,\delta_2) \leq & -\frac{\beta SI}{(1+k_1I)E}+a_1\theta S_0+a_1\gamma S_0+\frac{a_1\alpha S_0\tilde{M}}{1+k_2S_0}+\frac{a_1\beta S_0}{(1+k_1S_0)}\\
& +4\mu+\theta+|\delta_1|+|\delta_2|+\gamma+\alpha+\frac{\beta S_0}{1+k_1S_0}\\
& +\rho_1\bar{\delta_1}\delta_1-\frac{\rho_1}{2}\delta_1^2+\frac{\sigma_1^2}{2}+\rho_2\bar{\delta_2}\delta_2-\frac{\rho_2}{2}\delta_2^2+\frac{\sigma_2^2}{2}.\\
\leq & \frac{-\beta S_0}{(1+k_1S_0)\epsilon}+\varpi_4\\
\leq & -1,
\end{aligned} \tag{4.30}$$

as a consequence of (4.24).

– **Case 4 :** For any $(S,E,I,R,\delta_1,\delta_2)^T \in \mathcal{W}^c_{\epsilon,4}$, by (4.21), we have :

$$\begin{aligned}\mathbf{Z}(S,E,I,R;\delta_1,\delta_2) \leq & -\frac{\bar{\delta_2} I}{\frac{\Lambda}{\mu}-S-E-I-R} + a_1\theta S_0 + a_1\gamma S_0 + \frac{a_1\alpha S_0\tilde{M}}{1+k_2S_0} \\ & + \frac{a_1\beta S_0}{(1+k_1S_0)} + 4\mu + \theta + |\delta_1| + |\delta_2| + \gamma + \alpha + \frac{\beta S_0}{1+k_1S_0} \\ & + \rho_1\bar{\delta_1}\delta_1 - \frac{\rho_1}{2}\delta_1^2 + \frac{\sigma_1^2}{2} + \rho_2\bar{\delta_2}\delta_2 - \frac{\rho_2}{2}\delta_2^2 + \frac{\sigma_2^2}{2}. \\ \leq & \frac{-\omega_6}{\epsilon} + \omega_4 \\ \leq & -1,\end{aligned} \tag{4.31}$$

as a consequence of (4.25).

– **Case 5 :** For any $(S,E,I,R,\delta_1,\delta_2)^T \in \mathcal{W}^c_{\epsilon,5}$, From (4.21), it follows that :

$$\begin{aligned}\mathbf{Z}(S,E,I,R;\delta_1,\delta_2) \leq & \frac{\rho_1}{2} + a_1\theta S_0 + a_1\gamma I + \frac{a_1\alpha S_0\tilde{M}}{1+k_2S_0} + \frac{a_1\beta S_0}{(1+k_1S_0)} \\ & + 4\mu + \theta + |\delta_1| + |\delta_2| + \gamma + \alpha + \frac{\beta S_0}{1+k_1S_0} \\ & + \rho_1\bar{\delta_1}\delta_1 - \frac{\rho_1}{2}\delta_1^2 + \frac{\sigma_1^2}{2} + \rho_2\bar{\delta_2}\delta_2 - \frac{\rho_2}{2}\delta_2^2 + \frac{\sigma_2^2}{2}. \\ \leq & \frac{-\rho_1}{2\epsilon^2} + \omega_4 \\ \leq & -1,\end{aligned} \tag{4.32}$$

as a consequence of (4.26).

– **Case 6 :** For any $(S,E,I,R,\delta_1,\delta_2)^T \in \mathcal{W}^c_{\epsilon,6}$, From (4.21), it follows that :

$$\begin{aligned}\mathbf{Z}(S,E,I,R;\delta_1,\delta_2) \leq & \frac{\rho_2}{2} + a_1\theta S_0 + a_1\gamma I + \frac{a_1\alpha S_0\tilde{M}}{1+k_2S_0} + \frac{a_1\beta S_0}{(1+k_1S_0)} \\ & + 4\mu + \theta + |\delta_1| + |\delta_2| + \gamma + \alpha + \frac{\beta S_0}{1+k_1S_0} \\ & + \rho_1\bar{\delta_1}\delta_1 - \frac{\rho_1}{2}\delta_1^2 + \frac{\sigma_1^2}{2} + \rho_2\bar{\delta_2}\delta_2 - \frac{\rho_2}{2}\delta_2^2 + \frac{\sigma_2^2}{2}. \\ \leq & \frac{-\rho_1}{2\epsilon^2} + \omega_4 \\ \leq & -1,\end{aligned} \tag{4.33}$$

as a consequence of (4.27).

Drawing from (4.28), (4.29), (4.30), (4.31), (4.32), and (4.33), it is straightforward to deduce the existence of a sufficiently small $\epsilon$, such that :

$$\mathbf{Z}(S,E,I,R;\delta_1,\delta_2) \leq -1 \qquad \text{for any} (S,E,I,R;\delta_1,\delta_2)^T \in \mathbf{D}\setminus\mathcal{W}_\epsilon. \tag{4.34}$$

Let :

$$\begin{aligned}\varpi_7 := \sup_{(S,E,I,R,\delta_1,\delta_2)\in\mathbb{R}^4_+\times\mathbb{R}^2} \Bigg\{ & -\tilde{M}(\mu+\bar{\delta_1})(\mathcal{R}_0^s-1)++a_1\theta S+a_1\gamma I+\frac{a_1\alpha S_0\tilde{M}}{1+k_2S_0}+\frac{a_1\beta I}{(1+k_1I)} \\ & -\frac{\Lambda}{S}-\frac{\beta SI}{(1+k_1I)E}-\frac{\tilde{\delta_1}E}{I}+4\mu+\theta+|\delta_1|+|\delta_2|+\gamma+\alpha+\frac{\beta S_0}{1+k_1S_0} \\ & -\frac{\tilde{\delta_2}I}{\frac{\Lambda}{\mu}-S-E-I-R}+\rho_1\bar{\delta_1}\delta_1-\rho_1\delta_1^2+\frac{\sigma_1^2}{2}+\rho_2\bar{\delta_2}\delta_2-\rho_2\delta_2^2+\frac{\sigma_2^2}{2}\Bigg\}.\end{aligned}$$

Then :

$$\mathbf{Z}(S,E,I,R,\delta_1,\delta_2)\leq\varpi_7<\infty \qquad \text{for any}(S,E,I,R,\delta_1,\delta_2)^T\in\mathbb{R}^4_+\times\mathbb{R}^2. \tag{4.35}$$

- **Step 3 (Existence of a stationary distribution):**
For any initial condition $(S(0),E(0),I(0),R(0),\delta_1(0),\delta_2(0))^T\in\mathbf{D}$ integrating the expression (4.20) from 0 to $t$ and subsequently calculating the mathematical expectation we obtain :

$$\begin{aligned}0\leq & \frac{\mathbb{E}\mathcal{V}(S(t),E(t),I(t),R(t),\delta_1(t),\delta_2(0))}{t} \\ & =\frac{\mathbb{E}\mathcal{V}(S(0),E(0),I(0),R(0),\delta_1(0),\delta_2(0))}{t} \\ & +\frac{1}{t}\int_0^t\mathbb{E}(\mathcal{L}\mathcal{V}(S(s),E(s),I(t),R(s),\delta_1(s),\delta_2(s)))ds \\ & \leq\frac{\mathbb{E}\mathcal{V}(S(0),E(0),I(0),R(0),\delta_1(0),\delta_1(0))}{t} \\ & +\frac{1}{t}\int_0^t\mathbb{E}(\mathbf{Z}(S(s),E(s),I(t),R(s),\delta_1(s),\delta_2(s)))ds \\ & +3\tilde{M}\sqrt[3]{a_1a_2\Lambda\beta}\mathbb{E}\left[\int_{-\infty}^{\infty}(x\vee0)^{\frac{1}{3}}\Upsilon_1(x)dx-\frac{1}{t}\int_0^t(\delta_1(s)\vee0)^{\frac{1}{3}}ds\right] \\ & +\tilde{M}\mathbb{E}\left[\frac{1}{t}\int_0^t(q(s)\vee0)ds-\int_0^{\infty}x\bar{\Upsilon}_1(x)dx\right] \\ & +a_2\tilde{M}\mathbb{E}\left[\frac{1}{t}\int_0^t(r(s)\vee0)ds-\int_0^{\infty}x\bar{\Upsilon}_2(x)dx\right].\end{aligned} \tag{4.36}$$

Through the ergodic properties of $\delta_1$, $\delta_2$, $q(t)$ and $r(t)$, coupled with the strong law of large numbers [112], we have :

$$\lim_{t\to\infty}\mathbb{E}\left[\int_{-\infty}^{\infty}(x\vee0)^{\frac{1}{3}}\Upsilon_1(x)dx-\frac{1}{t}\int_0^t(\delta_1(s)\vee0)^{\frac{1}{3}}ds\right]=0 \qquad a.s. \tag{4.37}$$

and

$$\lim_{t\to\infty}\mathbb{E}\left[\frac{1}{t}\int_0^t (q(s)\vee 0)ds-\int_0^\infty x\bar{\Upsilon}_1(x)dx\right]=0 \qquad a.s. \tag{4.38}$$

and

$$\lim_{t\to\infty}\mathbb{E}\left[\frac{1}{t}\int_0^t (r(s)\vee 0)ds-\int_0^\infty x\bar{\Upsilon}_2(x)dx\right]=0 \qquad a.s. \tag{4.39}$$

By applying the lower limit to both sides of (4.36) and consolidating it with (4.37), (4.38) and (4.39), we arrive at :

$$\begin{aligned}0\leq&\liminf_{t\to\infty}\frac{\mathbb{E}\mathcal{V}(S(0),E(0),I(0),R(0),\delta_1(0),\delta_1(0))}{t}\\&+\liminf_{t\to\infty}\frac{1}{t}\int_0^t\mathbb{E}(\mathbf{Z}(S(s),E(s),I(t),R(s),\delta_1(s),\delta_2(s)))ds\\=&\liminf_{t\to\infty}\frac{1}{t}\int_0^t\mathbb{E}(\mathbf{Z}(S(s),E(s),I(t),R(s),\delta_1(s),\delta_2(s)))ds \qquad a.s.\end{aligned} \tag{4.40}$$

Furthermore, in accordance with (4.34) and (4.35), we obtain :

$$\begin{aligned}&\liminf_{t\to\infty}\frac{1}{t}\int_0^t\mathbb{E}(\mathbf{Z}(S(s),E(s),I(t),R(s),\delta_1(s),\delta_2(s)))ds\\=&\liminf_{t\to\infty}\frac{1}{t}\int_0^t\mathbb{E}(\mathbf{Z}(S(s),E(s),I(t),R(s),\delta_1(s),\delta_2(s)))\mathbb{1}_{(S(s),E(s),I(s),R(s),\delta_1(s),\delta_2(s))^T\in\mathcal{W}_\varepsilon}ds\\&+\liminf_{t\to\infty}\frac{1}{t}\int_0^t\mathbb{E}(\mathbf{Z}(S(s),E(s),I(s),R(s),\delta_1(s),\delta_2(s)))\mathbb{1}_{(S(s),E(s),I(s),R(s),\delta_1(s),\delta_2(s))^T\in(\mathbf{D}\setminus\mathcal{W}_\varepsilon)}ds\\\leq&\varpi_7\liminf_{t\to\infty}\frac{1}{t}\int_0^t\mathbb{1}_{(S(s),E(s),I(s),R(s),\delta_1(s),\delta_2(s))^T\in\mathcal{W}_\varepsilon}ds\\&-\liminf_{t\to\infty}\frac{1}{t}\int_0^t\mathbb{1}_{(S(s),E(s),I(s),R(s),\delta_1(s),\delta_2(s))^T\in(\mathbf{D}\setminus\mathcal{W}_\varepsilon)}ds\\\leq&-1+(\varpi_7+1)\liminf_{t\to\infty}\frac{1}{t}\int_0^t\mathbb{1}_{(S(s),E(s),I(s),R(s),\delta_1(s),\delta_2(s))^T\in\mathcal{W}_\varepsilon}ds.\end{aligned} \tag{4.41}$$

By considering (4.40) and (4.41), we deduce :

$$\liminf_{t\to\infty}\frac{1}{t}\int_0^t\mathbb{1}_{(S(s),E(s),I(s),R(s),\delta_1(s),\delta_2(s))^T\in\mathcal{W}_\varepsilon}ds\geq\frac{1}{\varpi_7+1}>0 \qquad a.s. \tag{4.42}$$

Based on the definition of event probability and the Fatou lemma , (4.42) can be expressed equivalently in the following form :

$$\liminf_{t\to\infty}\frac{1}{t}\int_0^t\mathbf{P}(s,(S(s),E(s),I(s),R(s),\delta_1(s),\delta_2(s)),\mathcal{W}_\varepsilon)ds\geq\frac{1}{\varpi_7+1}>0 \qquad a.s. \tag{4.43}$$

In the expression $\mathbf{P}(t,(S(t),E(t),I(t),R(t),\delta_1(t),\delta_2(t)),\mathcal{W}_\varepsilon)$, $\mathbf{P}$ represents the transition probability of the state vector $(S(t),E(t),I(t),R(t),\delta_1(t),\delta_2(t))$ belonging to the set $\mathcal{W}_\varepsilon$. Consequently, by invoking Lemma 2.33, it follows that the system (4.5) possesses at least one stationary distribution, denoted as $\Psi(.)$, defined on the space $\mathbb{R}^4_+ \times \mathbb{R}^2$, and this distribution exhibits the Feller property. With these considerations, the proof is concluded. □

## 4.4 Extinction

In this section, our primary focus is on exploring the conditions that are sufficient for the eradication of the disease.

**Theorem 4.3.**

*For any initial condition* $(S(0),E(0),I(0),R(0),\delta_1(0),\delta_2(0)) \in \mathbb{R}^4_+ \times \mathbb{R}^2$, *the solution* $(S(t),E(t),I(t),R(t),\delta_1(t),\delta_2 t))$ *of system* (4.5) *exhibits the following characteristic :*

$$\limsup_{t\to\infty} \frac{\ln(\frac{\Theta_1}{\bar{\delta_1}+\mu}E + \frac{\Theta_2}{\mu+\bar{\delta_2}+\gamma+\alpha}I)}{t} \leq \min\left\{\bar{\delta_1}+\mu, \mu+\bar{\delta_2}+\gamma+\alpha\right\}(\mathcal{R}_0^{ex}-1) \qquad \textbf{a.s.},$$

*where :* $\Theta_1 = \dfrac{\bar{\delta_1}}{\sqrt{\mathcal{R}_0}(\mu+\bar{\delta_2}+\gamma+\alpha)}, \Theta_2 = 1$ *and*

$$\mathcal{R}_0^{ex} = \sqrt{\mathcal{R}_0} + (1+\frac{(\mu+\theta)(\mu+\bar{\delta_1})}{\bar{\delta_1}\mu})\frac{\sigma_1}{\sqrt{\pi\rho_1}\min\left\{\bar{\delta_1}+\mu, \mu+\bar{\delta_2}+\gamma+\alpha\right\}} + \frac{\sigma_2}{\sqrt{\pi\rho_2}\min\left\{\bar{\delta_1}+\mu, \mu+\bar{\delta_2}+\gamma+\alpha\right\}}.$$

*In particular, when* $\mathcal{R}_0^{ex}$ *is less than* 1, *then :* $\lim_{t\to\infty} E(t) = 0, \lim_{t\to\infty} I(t) = 0$ **a.s.**

**Proof.** According to Theorem 1.4 in reference [137], it can be deduced that there exists a left eigenvector $(\Theta_1,\Theta_2)$ for matrix $\mathsf{B} = \begin{pmatrix} 0 & \frac{\beta S_0}{\bar{\delta_1}+\mu} \\ \frac{\bar{\delta_1}}{\mu+\bar{\delta_2}+\gamma+\alpha} & 0 \end{pmatrix}$ corresponding to $\sqrt{\mathcal{R}_0}$ i.e.,$\sqrt{\mathcal{R}_0}(\Theta_1,\Theta_2) = (\Theta_1,\Theta_2)\mathsf{B}$. Taking $\Theta_1 = \dfrac{\bar{\delta_1}}{\sqrt{\mathcal{R}_0}(\mu+\bar{\delta_2}+\gamma+\alpha)}, \Theta_2 = 1,$

and denoting $\digamma(E,I) = \frac{\Theta_1}{\bar{\delta_1}+\mu}E + \frac{\Theta_2}{\mu+\bar{\delta_2}+\gamma+\alpha}I$, we obtain

$$\begin{aligned}
\mathcal{L}(\ln \mathcal{F}) =& \frac{1}{\mathcal{F}}\Bigg[\frac{\Theta_1}{\bar{\delta_1}+\mu}\Big(\frac{\beta SI}{1+k_1 I} - (\bar{\delta_1}(t)+\mu)E\Big) \\
&+ \frac{\Theta_2}{\mu+\bar{\delta_2}+\gamma+\alpha}\Big(\bar{\delta_1}(t)E - \Big(\mu+\bar{\delta_2}(t)+\gamma+\frac{\alpha}{1+k_2 I}\Big)I\Big)\Bigg] \\
\leq& \frac{1}{\mathcal{F}}\Bigg[\frac{\Theta_1}{\bar{\delta_1}+\mu}\Big(\beta S_0' I - (\bar{\delta_1}+\mu)E\Big) \\
&+ \frac{\Theta_2}{\mu+\bar{\delta_2}+\gamma+\alpha}\Big(\bar{\delta_1}E - (\mu+\bar{\delta_2}+\gamma+\alpha)I\Big)\Bigg] \\
&+ \frac{\Theta_1}{\bar{\delta_1}+\mu}\frac{|\bar{\delta_1}-\bar{\delta_1}|E}{\mathcal{F}} + \frac{\Theta_2}{\mu+\bar{\delta_2}+\gamma+\alpha}\frac{|\bar{\delta_1}-\bar{\delta_1}|}{\mathcal{F}} \\
&+ \frac{\Theta_2}{\mu+\bar{\delta_2}+\gamma+\alpha}\frac{|\bar{\delta_2}-\bar{\delta_2}|}{\mathcal{F}} \\
\leq& \frac{1}{\mathcal{F}}(\Theta_1,\Theta_2)\Big(\mathsf{B}(E,I)^T - (E,I)^T\Big) \\
&+ \frac{\Theta_1}{\bar{\delta_1}+\mu}\frac{|\delta_1-\bar{\delta_1}|E}{\mathcal{F}} + \frac{\Theta_2}{\mu+\bar{\delta_2}+\gamma+\alpha}\frac{|\delta_2-\bar{\delta_2}|E}{\mathcal{F}} \\
&+ \frac{\Theta_2}{\mu+\bar{\delta_2}+\gamma+\alpha}\frac{|\delta_2-\bar{\delta_2}|I}{\mathcal{F}} \\
\leq& \min\{\bar{\delta_1}+\mu,\ \mu+\bar{\delta_2}+\gamma+\alpha\}(\sqrt{\mathcal{R}_0}-1) \\
&+ \left(1+\frac{\beta S_0'}{\sqrt{\mathcal{R}_0}(\mu+\bar{\delta_2}+\gamma+\alpha)}\right)|\delta_1-\bar{\delta_1}| + |\delta_2-\bar{\delta_2}|.
\end{aligned} \tag{4.44}$$

Moreover, based on the principles of ergodicity from reference [102], both $\delta_1$ and $\delta_2$ are expected to undergo weak convergence toward the normal distributions $N(\bar{\delta_1}, \frac{\sigma_1^2}{2\rho_1})$ and $N(\bar{\delta_2}, \frac{\sigma_2^2}{2\rho_2})$, respectively, in the following manner:

$$\Upsilon_1(x) = \frac{\sqrt{\rho_1}}{\sqrt{\pi}\sigma_1}e^{-\frac{\rho_1(y-\bar{\delta_1})^2}{\sigma_1^2}}, x\in\mathbb{R}, \qquad \Upsilon_2(x) = \frac{\sqrt{\rho_2}}{\sqrt{\pi}\sigma_2}e^{-\frac{\rho_2(y-\bar{\delta_2})^2}{\sigma_2^2}}, x\in\mathbb{R}.$$

Subsequently, we obtain :

$$\lim_{t\to\infty}\frac{1}{t}\int_0^t |\delta_1(s)-\bar{\delta_1}|ds = \int_{-\infty}^{\infty}|\delta_1(x)-\bar{\delta_1}|\Upsilon_1(x)dx = \frac{\sigma_1}{\sqrt{\pi\rho_1}} \tag{4.45}$$

and

$$\lim_{t\to\infty}\frac{1}{t}\int_0^t |\delta_2(s)-\bar{\delta_2}|ds = \int_{-\infty}^{\infty}|\delta_2(x)-\bar{\delta_2}|\Upsilon_2(x)dx = \frac{\sigma_2}{\sqrt{\pi\rho_2}}. \tag{4.46}$$

By integrating (4.44) over the interval $[0,t]$, dividing by $t$, and taking the limit as $t$ approaches infinity on both sides, and subsequently combining with (4.45) and (4.46), we derive the following result :

$$\begin{aligned}
\limsup_{t\to\infty}\frac{\ln \digamma(E(t),I(t),)}{t} &\leq \min\left\{\bar{\delta_1}+\mu,\mu+\bar{\delta_2}+\gamma+\alpha\right\}(\sqrt{\mathcal{R}_0}-1)\\
&\quad+(1+\frac{\beta S_0^{'}}{\sqrt{\mathcal{R}_0}(\mu+\bar{\delta_2}+\gamma+\alpha)})\frac{1}{t}\int_0^t|\delta_1(s)-\bar{\delta_1}|ds\\
&\quad+\frac{1}{t}\int_0^t|\bar{\delta_2}(s)-\bar{\delta_2}|ds\\
&\leq \min\left\{\bar{\delta_1}+\mu,\mu+\bar{\delta_2}+\gamma+\alpha\right\}(\sqrt{\mathcal{R}_0}-1)\\
&\quad+(1+\frac{\beta S_0^{'}}{\sqrt{\mathcal{R}_0}(\mu+\bar{\delta_2}+\gamma+\alpha)})\frac{\sigma_1}{\sqrt{\pi\rho_1}}+\frac{\sigma_2}{\sqrt{\pi\rho_2}}\\
&\leq \min\left\{\bar{\delta_1}+\mu,\mu+\bar{\delta_2}+\gamma+\alpha\right\}(\mathcal{R}_0^{ex}-1)\qquad \textbf{a.s.}
\end{aligned}$$

Hence, when $\mathcal{R}_0^{ex}$ is less than 1, the disease will lead to extinction. This concludes the proof. □

## 4.5 Computational simulations

Employing the widely recognized Milstein [119] high-order numerical technique, we will present two practical instances along with numerical simulations to validate the analytical findings mentioned earlier in this section; the discretization equation for system (4.5) is articulated as follows :

$$\begin{cases}
S^{\mathbf{k}+1} = S^{\mathbf{k}}+[\Lambda-\dfrac{\beta S^{\mathbf{k}}I^{\mathbf{k}}}{1+k_1I^{\mathbf{k}}}-(\mu+\theta)S^{\mathbf{k}}]\Delta t,\\
E^{\mathbf{k}+1} = E^{\mathbf{k}}+[\dfrac{\beta S^{\mathbf{k}}I^{\mathbf{k}}}{1+k_1I^{\mathbf{k}}}-(\tilde{\delta_1}^{\mathbf{k}}+\mu)E^{\mathbf{k}}]\Delta t,\\
I^{\mathbf{k}+1} = I^{\mathbf{k}}+[\tilde{\delta_1}^{\mathbf{k}}E^{\mathbf{k}}-\left(\mu+\tilde{\delta_2}^{\mathbf{k}}+\gamma+\dfrac{\alpha}{1+k_2I^{\mathbf{k}}}\right)I^{\mathbf{k}}]\Delta t,\\
R^{\mathbf{k}+1} = R^{\mathbf{k}}+[\theta S^{\mathbf{k}}+(\gamma+\dfrac{\alpha}{1+k_2I^{\mathbf{k}}})I^{\mathbf{k}}-\mu R^{\mathbf{k}}]\Delta t,\\
\delta_1^{\mathbf{k}+1} = \delta_1^{\mathbf{k}}+\rho_1[\bar{\delta_1}-\delta_1^{\mathbf{k}}(t)]\Delta t+\sigma_1\sqrt{\Delta t}\xi_{1,\mathbf{k}},\\
\delta_2^{\mathbf{k}+1} = \delta_2^{\mathbf{k}}+\rho_2[\bar{\delta_2}-\delta_2^{\mathbf{k}}]\Delta t+\sigma_2\sqrt{\Delta t}\xi_{2,\mathbf{k}}.
\end{cases}\tag{4.47}$$

Here, considering a positive time increment $\Delta t>0$, and $\xi_{i,\mathbf{k}}$(with $i=1,2$) representing two independent Gaussian random variables following the distribution $\mathcal{N}(0,1)$ for $\mathbf{k}=1,...,n$ , we select realistic parameter values from established references,

Table 4.2: List of parameters of stochastic system (4.5) .

| Parameters | Values | Source |
|---|---|---|
| $\Lambda$ | 2.5 | Assumed |
| $\mu$ | 0.0875 | [138] |
| $\bar{\delta_1}$ | 0.125 | [139] |
| $k_1$ | 0.1 | [132] |
| $k_2$ | 1.25 | [132] |
| $\alpha$ | 0.01 | Estimated |
| $\bar{\delta_2}$ | 0.2 | Estimated |
| $\theta$ | 0.002999 | Estimated |

presenting all biological parameter values in Table 4.2. Subsequently, through numerical simulations, our primary focus revolves around validating two key aspects:

- Examining the stationary distribution of system (4.5) under minimal stochastic noise.
- Assessing the impact of environmental noise on the disease extinction of system (4.5).

**Example 4.1.**

To numerically establish the presence of a stationary distribution, we choose the parameters as follows: $(\sigma_1, \sigma_2) = (0.01999; 0.01999), (\rho_1, \rho_2) = (0.1; 0.1), \gamma = 0.02, \beta = 0.002$ and the remaining parameter values are outlined in Table 4.2. Through direct computation, the results indicate that:

$$\mathcal{R}_0^s = \frac{\Lambda\beta(\frac{1}{\sqrt{\pi}}\int_{-\frac{\bar{\delta_1}\sqrt{\rho_1}}{\sigma_1}}^{\infty}\left(\frac{\sigma_1}{\sqrt{\rho_1}}x+\bar{\delta_1}\right)^{\frac{1}{3}}e^{-x^2}dx)^{\frac{1}{3}}}{(\mu+\theta)\left(\mu+\bar{\delta_1}\right)\left(\mu+\bar{\delta_2}+\gamma+\alpha\right)} - \frac{\sigma_1}{2\sqrt{\pi\rho_1}(\mu+\bar{\delta_1})}$$
$$- \frac{\Lambda\beta(\frac{1}{\sqrt{\pi}}\int_{-\frac{\bar{\delta_1}\sqrt{\rho_1}}{\sigma_1}}^{\infty}\left(\frac{\sigma_1}{\sqrt{\rho_1}}x+\bar{\delta_1}\right)^{\frac{1}{3}}e^{-x^2}dx)^{\frac{1}{3}}}{(\mu+\theta)(\mu+\bar{\delta_1}+\gamma+\alpha)^2}\frac{\sigma_2}{2\sqrt{\pi\rho_2}(\mu+\bar{\delta_1})} \approx 10.37 > 1.$$

Consequently, meeting the criteria stipulated in Theorem 4.2, we can conclude that system (4.5) possesses a stationary distribution denoted by $\Psi(.)$, signifying the sus-

tained and predominant existence of individuals exposed to measles and those infected with measles with absolute certainty. This is illustrated in Figure 4.1 and Figure 4.2.

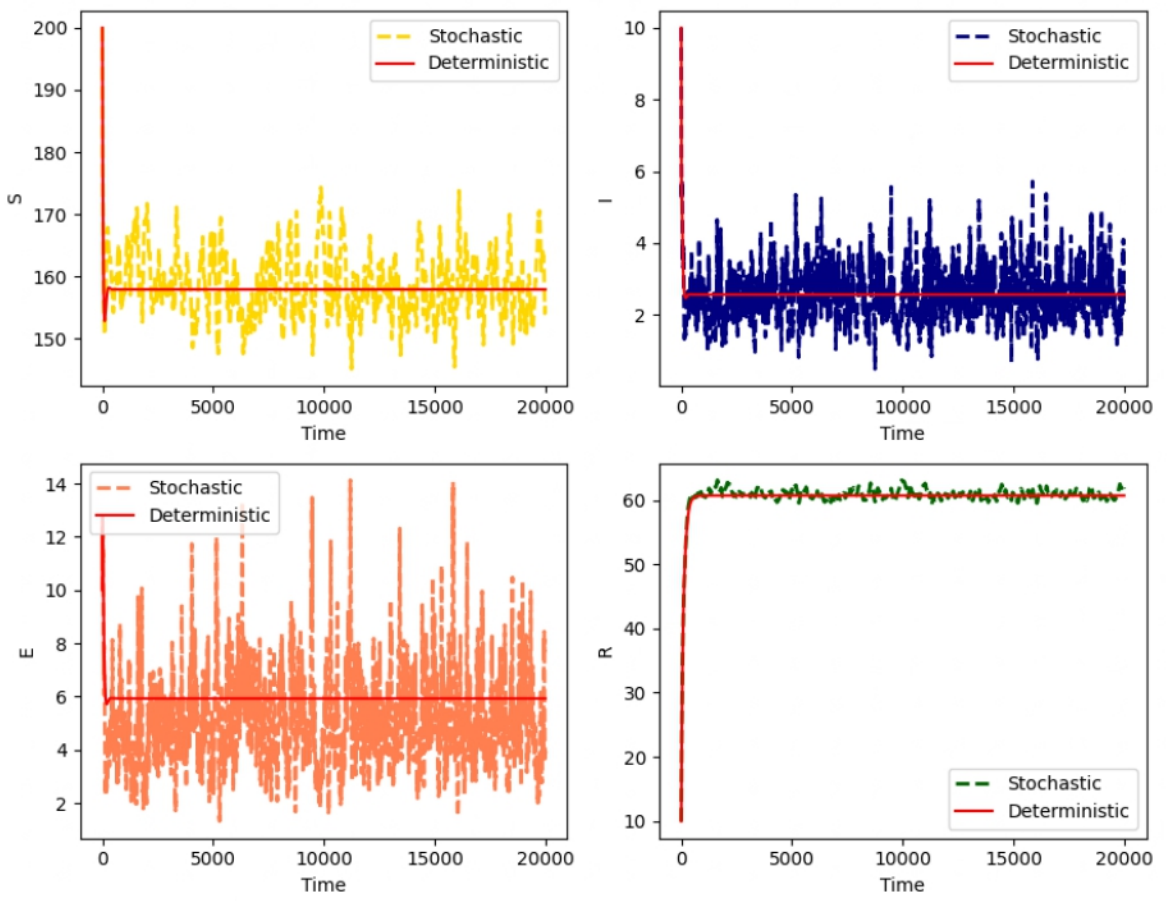



Figure 4.1: The illustration depicts the simulated evolution of the solution $(S(t), I(t), E(t), R(t))$ in systems (4.1) and (4.5) using the given parameter values $(\sigma_1, \sigma_2) = (0.01999; 0.01999), (\rho_1, \rho_2) = (0.1; 0.1), \gamma = 0.02, \beta = 0.002$.

**Example 4.2.**

In order to affirm the assertion that a high reversion rate or low volatility intensity can induce the extinction of the Measles disease, we opt for the parameter values $(\sigma_1, \sigma_2) = (0.001999; 0.001999), (\rho_1, \rho_2) = (0.3; 0.3), \gamma = 0.3, \beta = 0.0000002$, with the remaining parameters detailed in Table 4.2. Subsequently, we perform the neces-

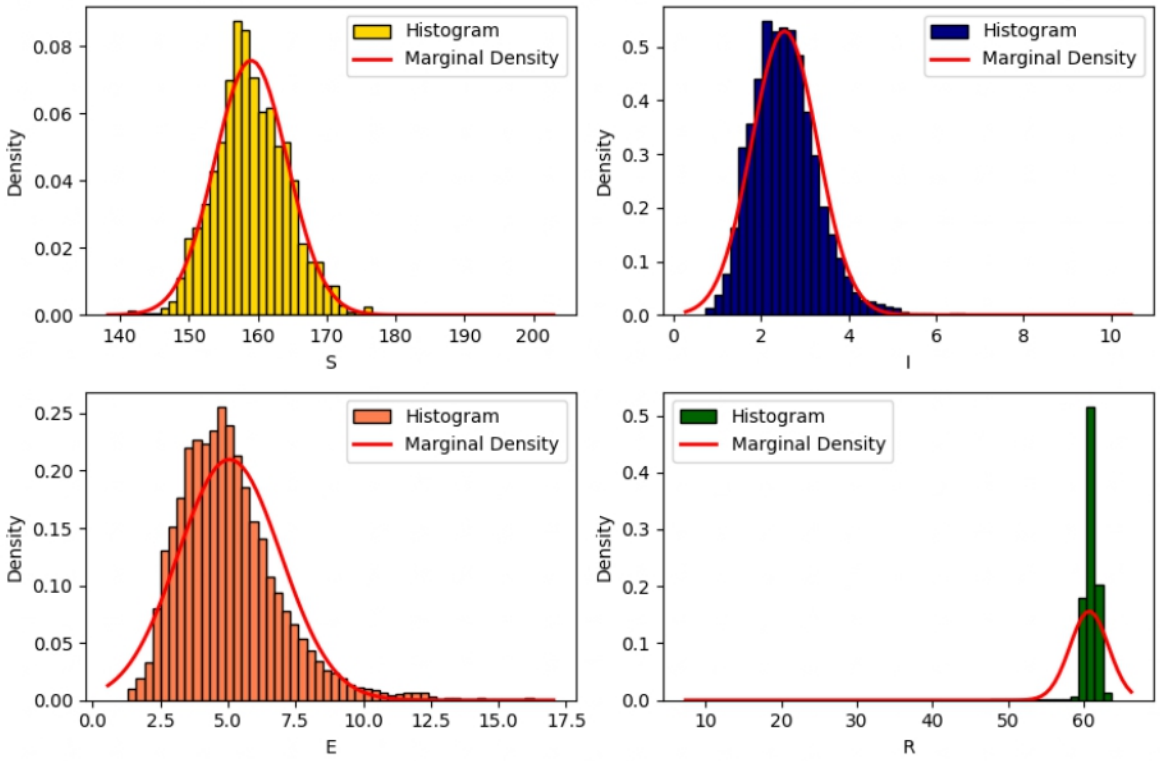



Figure 4.2: Frequency Histograms and Marginal Density Functions for Variables $S, E, I$, and $R$ within System (4.5).

sary computations to establish that:

$$\begin{aligned}\mathcal{R}_0^X =& \sqrt{\mathcal{R}_0} + (1 + \frac{(\mu+\theta)(\mu+\bar{\delta_1})}{\bar{\delta_1}\mu}) \frac{\sigma_1}{\sqrt{\pi\rho_1}\min\left\{\bar{\delta_1}+\mu, \mu+\bar{\delta_2}+\gamma+\alpha\right\}} \\ &+ \frac{\sigma_2}{\sqrt{\pi\rho_2}\min\left\{\bar{\delta_1}+\mu, \mu+\bar{\delta_2}+\gamma+\alpha\right\}} \\ \approx& 0.024 \\ <& 1,\end{aligned}$$

as per Theorem 4.3, it becomes apparent that the Measles disease is poised to undergo exponential extinction almost surely. This assertion is corroborated by the visual representation in Figure 4.3.

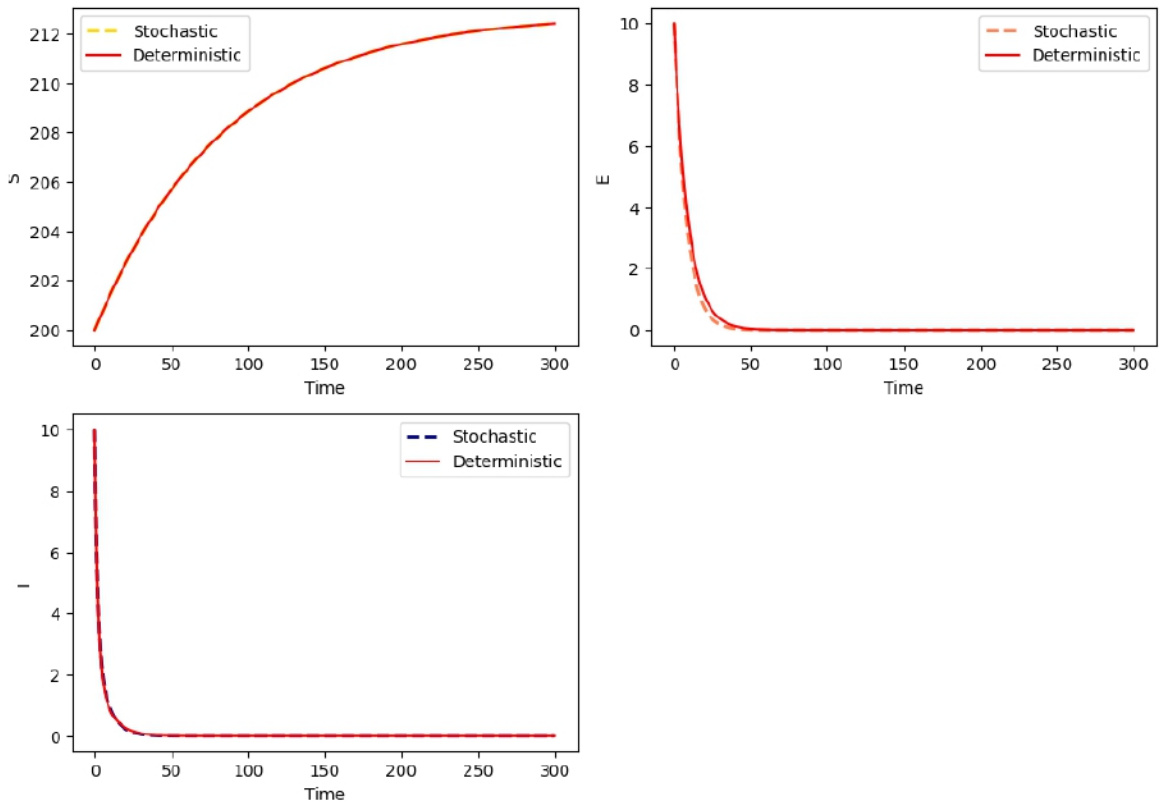



Figure 4.3: The solutions for both the deterministic system (4.1) and the stochastic counterpart (4.5) are explored under the specified parameter values $(\sigma_1, \sigma_2) = (0.001999; 0.001999), (\rho_1, \rho_2) = (0.3; 0.3), \gamma = 0.3, \beta = 0.0000002$ .

## 4.6 Discussion

This work examines a stochastic model for measles transmission dynamics, enriched by the integration of the Ornstein–Uhlenbeck process. The study begins by rigorously demonstrating that the stochastic system (4.5) possesses a unique global positive solution, laying a solid theoretical foundation. Following this, we explore the existence of ergodic stationary distributions for the random system (4.5). This is achieved through the construction of a sequence of carefully designed Lyapunov functions, highlighting the role of the Ornstein–Uhlenbeck process in establishing such distributions. Additionally, sufficient conditions for disease extinction are derived, offering a theoretical basis for strategies aimed at preventing and controlling the spread of infectious diseases.

To reinforce these theoretical insights, numerical simulations are conducted, providing empirical validation of the findings presented. Despite these advancements, the study is not without limitations. The investigation remains confined to the formulation and analysis of specific stochastic systems, leaving key questions unanswered. For instance, future research should evaluate the applicability of alternative random perturbations in addressing similar epidemiological problems, thus broadening the scope and robustness of this approach.

Chapter 5

# Threshold Dynamics of a Stochastic HIV-1 Model with Dual Transmission Pathways, Immune Response and Black-Karasinski Process

## 5.1 Introduction

The persistent challenge of Acquired Immunodeficiency Syndrome (AIDS) continues to endanger global public health and societal stability. According to the Global AIDS Update 2021, an estimated 37.7 million people worldwide were living with Human Immunodeficiency Virus (HIV) in 2020 [140]. HIV specifically targets lymphocytes, particularly T-helper cells, thereby impairing the immune system and reducing its ability to combat infections. This compromised immunity leaves individuals susceptible to numerous opportunistic diseases [141, 142]. T-helper cells are vital for adaptive immune responses, as they secrete differentiation factors essential for the functioning of other immune system components [143, 144].

To curb the impact and spread of HIV/AIDS, public health interventions have employed strategies such as media-driven campaigns aimed at educating individuals and promoting behavioral changes to reduce risk exposure [145]. In parallel, mathematical modeling has become a critical tool in studying HIV dynamics, particularly in understanding viral replication and transmission processes [146]. Early models primarily analyzed interactions among healthy cells, infected cells, and free virus particles, focusing on virus-to-cell transmission pathways [147]. However, researchers have since discovered that HIV can also propagate via direct contact between infected and uninfected cells [148].

Emerging evidence suggests that cell-to-cell transmission is more efficient than virus-to-cell transmission, as the bloodstream presents additional challenges compared to tissue environments. Consequently, mathematical models of HIV infection now often incorporate both transmission mechanisms to provide a more comprehensive understanding of the disease's dynamics. Over time, ordinary differential equation models that integrate these dual pathways have gained traction, offering valuable insights into the complexities of HIV/AIDS [149, 150, 151]. Notably, Jiazhe Lin et al. [152] proposed an HIV model that closely aligns with the framework described below

$$\begin{cases} \dfrac{dx(t)}{dt} = \Lambda - dx(t) - \beta_1 x(t)v(t) - \beta_2 x(t)y(t), \\ \dfrac{dy(t)}{dt} = \beta_1 x(t)v(t) + \beta_2 x(t)y(t) - ay(t) - \alpha y(t), \\ \dfrac{dv(t)}{dt} = ky(t) - uv(t) - pv(t)z(t), \\ \dfrac{dz(t)}{dt} = cv(t)z(t) - bz(t) - \beta z(t). \end{cases} \tag{5.1}$$

This dynamic framework models the progression of infection by examining the interplay between various components. The variable $x(t)$ represents the population of uninfected cells, which are produced at a rate $\Lambda$ and experience natural death at a rate $d$. Infected cells are represented by $y(t)$, while $v(t)$ denotes the viral load, and $z(t)$ accounts for $B$-cell populations.

Throughout this work, we denote the time-dependent state variables of the system by $x(t), y(t), v(t), z(t)$. For simplicity of notation, we sometimes write $(x, y, v, z)$ instead of $(x(t), y(t), v(t), z(t))$ when no ambiguity arises.

The model includes two distinct infection pathways: virus-to-cell transmission with rate $\beta_1$ and cell-to-cell transmission with rate $\beta_2$.

Death rates are incorporated for different populations, with $a$, $u$, and $b$ representing the mortality of infected cells, viruses, and $B$-cells, respectively. Disease-induced mortality is modeled through parameters $\alpha$ for infected cells and $\beta$ for $B$-cells. The parameter $k$ represents the production of free virus particles by each infected cell. Interactions involving $B$-cells are expressed through terms such as $pv(t)z(t)$, describing viral elimination by $B$-cells, and $cv(t)z(t)$, which captures the stimulation of $B$-cell production in response to antigen presence.

By employing the methodology developed by Ma et al. [133] and utilizing the generation matrix technique, it can be established that system (5.1) possesses a positively invariant set.

$$\Omega = \left\{ (x, y, v, z) | x + y < \frac{\Lambda}{\mu_1}, v + \frac{p}{c} z < \frac{k\Lambda}{\mu_2} \right\}, \mu_1 = \min\{d, a\}, \mu_2 = \min\{d, a\} \min\{u, b\},$$

the basic reproduction number for system (5.1) is calculated as

$$\mathcal{R}_0 = \frac{(\beta_1 k + \beta_2 u) x_0}{u(a + \alpha)},$$

which serves as a decisive factor in determining the occurrence of the disease,where $x_0 = \frac{\Lambda}{d}$.
Moreover, by integrating the global stability theory of equilibrium [153, 154], system (5.1) exhibits the ensuing threshold dynamics :

- When $\mathcal{R}_0 < 1$, system (5.1) attains a disease-free equilibrium denoted as $E_0(x_0, 0, 0, 0) = (\frac{\Lambda}{d}, 0, 0, 0)$, which exhibits local asymptotic stability within the region $\Omega$.

- In the case where $\mathcal{R}_0$ is greater than 1, alongside the disease-free equilibrium $E_0$, there exists a distinct endemic equilibrium denoted as $E^*(x^*, y^*, v^*, z^*)$, with $x^*, y^*, v^*$, and $z^*$ being greater than 0, which demonstrates local asymptotic stability within the interior of the domain $\Omega$.

In contrast, the formulation of system (5.1) assumes a stable and deterministic environment. To extend this framework and account for environmental variability, we reformulate system (5.1) as a stochastic system, following the approach outlined in the previous section. To model the impact of environmental noise on the transition rate $\beta_1$ and the virus transmission rate $\beta_2$, these parameters are treated as stochastic variables. Their behavior is governed by random fluctuations, consistent with the formulation presented in [112]

$$d\beta_j(t) = \rho_j[\bar{\beta}_j - \beta_j(t)]dt + \sigma_j dB_j(t), (j = 1, 2), \tag{5.2}$$

where $\bar{\beta_1}$ and $\bar{\beta_2}$ represent positive constants indicating the long-term average level of $\beta_1$ and $\beta_2$; $\rho_1, \rho_2, \sigma_1$, and $\sigma_2$ are positive constants denoting the rate of reversion and the intensity of volatility, respectively; $B_i(t)$, $(i = 1, 2)$ signifies a standard Brownian motion defined within a complete probability space $\left(\Pi, \mathcal{F}, \{\mathcal{F}_t\}_{t\geq 0}, \mathbf{P}\right)$ with a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ adhering to standard conditions [112].

While the Ornstein–Uhlenbeck process provides a more nuanced representation of environmental noise compared to linear perturbations, it does not inherently ensure the non-negativity of $\beta_1$ and $\beta_2$. This limitation arises from their assumed distributions, $\beta_1 \rightsquigarrow \mathbb{N}(E(\beta_1(t)), \mathbf{Var}(\beta_1(t)))$ and $\beta_2 \rightsquigarrow \mathbb{N}(E(\beta_2(t)), \mathbf{Var}(\beta_2(t)))$, which permit negative values. Inspired by the framework proposed by Allen [108], we hypothesize that $\beta_1$ and $\beta_2$ follow a Black–Karasinski process [155].

In this model, the natural logarithms of $\beta_1(t)$ and $\beta_2(t)$ are governed by an Ornstein–Uhlenbeck process, as described by their respective stochastic differential equations. This transformation ensures that $\beta_1(t)$ and $\beta_2(t)$ remain strictly positive, aligning with their biological interpretation in the model. The use of the Black–Karasinski framework not only addresses the non-negativity issue but also provides a robust method for modeling the dynamic influence of environmental noise on these critical parameters

$$d\ln\beta_j(t) = \rho_j[\ln\bar{\beta}_j - \ln\beta_j(t)]dt + \sigma_j dB_j(t), \qquad (j = 1, 2), \tag{5.3}$$

where $\rho_j, B_j(t)$ and $\sigma_j$ $(j = 1, 2)$ remain consistent with those defined in (5.2). It is evident that $\beta_1(t) > 0$ and $\beta_2 > 0$ are prerequisites.

Consequently, by substituting $\beta_j$ in system (5.1) with $\beta_j(t)$ as defined in (5.3) (where $j = 1, 2$), the stochastic counterpart of (5.1) is formulated as follows

$$\begin{cases} dx(t) = [\Lambda - dx(t) - \beta_1(t)x(t)v(t) - \beta_2(t)x(t)y(t)]dt, \\ dy(t) = [\beta_1(t)x(t)v(t) + \beta_2(t)x(t)y(t) - ay(t) - \alpha y(t)]dt, \\ dv(t) = [ky(t) - uv(t) - pv(t)z(t)]dt, \\ dz(t) = [cv(t)z(t) - bz(t) - \beta z(t)]dt, \\ d(\ln \beta_1(t)) = \rho_1[\ln \bar{\beta_1} - \ln \beta_1(t)]dt + \sigma_1 dB_1(t), \\ d(\ln \beta_2(t)) = \rho_2[\ln \bar{\beta_2} - \ln \beta_2(t)]dt + \sigma_2 dB_2(t). \end{cases} \tag{5.4}$$

For ease of representation, let $\varphi(t) = \ln \dfrac{\beta_1(t)}{\bar{\beta_1}}$ and $\Phi(t) = \ln \dfrac{\beta_2(t)}{\bar{\beta_2}}$, making system (5.4) equivalently expressed as

$$\begin{cases} dx(t) = [\Lambda - dx(t) - \bar{\beta_1} e^{\varphi} x(t)v(t) - \bar{\beta_2} e^{\Phi} x(t)y(t)]dt, \\ dy(t) = [\bar{\beta_1} e^{\varphi} x(t)v(t) + \bar{\beta_2} e^{\Phi} x(t)y(t) - ay(t) - \alpha y(t)]dt, \\ dv(t) = [ky(t) - uv(t) - pv(t)z(t)]dt, \\ dz(t) = [cv(t)z(t) - bz(t) - \beta z(t)]dt, \\ d\varphi(t) = -\rho_1 \varphi(t)dt + \sigma_1 dB_1(t), \\ d\Phi(t) = -\rho_2 \Phi(t)dt + \sigma_2 dB_2(t). \end{cases} \tag{5.5}$$

## 5.2 Existence and Uniqueness of the Global Solution

Within the realm of biology, a pivotal inquiry revolves around determining if the solution is both globally applicable and positive. This section establishes the existence and uniqueness of the global positive solution for system (5.5).

**Theorem 5.1.**

*For every prescribed initial value* $(x(0), y(0), v(0), z(0), \varphi(0), \Phi(0))^T \in \mathbb{R}^4_+ \times \mathbb{R}^2$, *model* (5.5) *exhibits a unique global positive solution* $(x(t), y(t), v(t), z(t), \varphi(t), \Phi(t))$ *for all* $t \geq 0$ *with almost certain probability, meaning* $(x(t), y(t), v(t), z(t), \varphi(t), \Phi(t))^T \in \mathbb{R}^4_+ \times \mathbb{R}^2$ *holds for all* $t \geq 0$ *almost surely.*

**Proof.** Note that the coefficients of the given system exhibit local Lipschitz continuity. Following the work of Mao [112], it can be inferred that the system (5.5) possesses a unique local solution $(x(t), y(t), v(t), z(t), \varphi(t), \Phi(t))$ over the interval $t \in$

$[0,\Omega_e)$, where $\Omega_e$ represents an explosion time. Consider the set $\mathbb{F}_j := (-j,j)\times(-j,j)\times(-j,j)\times(-j,j)\times(-j,j)\times(-j,j)$, with $j>0$. It is evident that, for any initial condition $(x(0),y(0),v(0),z(0),\varphi(0),\Phi(0))^T \in \mathbb{R}^4_+\times\mathbb{R}^2$, there exists a sufficiently large integer $m_0 \geq 1$ such that $(\ln x(t),\ln y(t),\ln v(t),\ln z(t),\varphi(t),\Phi(t)) \in \mathbb{F}_m$, where $\mathbb{F}_m$ is defined for each integer $m \geq m_0$. In this scenario, a stopping time $\Pi_m$ is defined for $m \geq m_0$

$$\Pi_m = \inf\{t \in [0,\Omega_e) : (\ln x(t),\ln y(t),\ln v(t),\ln z(t),\varphi(t),\Phi(t)) \notin \mathbb{F}_m\}.$$

It is evident that $\Pi_m$ is a monotonically increasing function over the interval $[m_0,\infty)$. Defining $\Pi_\infty$ as the limit $\Pi_m$ approaches infinity, we leverage fundamental results, such as $\inf\phi = \infty$ and $\Pi_\infty \leq \Omega_e$ almost surely, to establish the equivalence of the first part of Theorem 5.1 with the assertion $\Pi_\infty = \infty$ almost surely. To demonstrate this, we employ a proof by contradiction. Assume $\Pi_\infty < \infty$ almost surely; then, there exist constants $\bar{T}>0$, $\varepsilon \in (0,1)$, and a positive integer $m_1$, such that :

$$\mathbf{P}\left\{\Pi_m \leq \bar{T}\right\} \geq \varepsilon \qquad\qquad \forall m \geq m_1. \tag{5.6}$$

From a certain perspective, considering any $t \leq \Pi_m$, we formulate the following nonnegative $C^2-$ Lyapunov function $\mathbb{U}(x,y,v,z,\varphi,\Phi)$:

$$\mathbb{U}(x,y,v,z,\varphi,\Phi) = (x-1-\ln x)+(y-1-\ln y)+(v-1-\ln v)+\frac{p}{c}(z-1-\ln z)+e^{\varphi}-\varphi-1+e^{\Phi}-\Phi-1$$

Applying the Itô's formula, we derive

$$\begin{aligned}
\mathcal{L}\mathbb{U} =& \Lambda - dx - ay - \alpha y + ky - uv - pvz + pvz - \frac{pb}{c}z - \frac{p\beta}{c}z - \frac{\Lambda}{x} \\
&+ d + \bar{\beta}_1 e^{\varphi} v + \bar{\beta}_2 e^{\Phi} y - \frac{\bar{\beta}_1 e^{\varphi} xv}{y} - \bar{\beta}_2 e^{\Phi} x + a + \alpha - \frac{ky}{v} + u + pz - pv + \frac{pb}{c} + \frac{p\beta}{c} \\
&- \rho_1\varphi(e^{\varphi}-1) + \frac{\sigma_1^2}{2}e^{\varphi} - \rho_2\Phi(e^{\Phi}-1) + \frac{\sigma_2^2}{2}e^{\Phi} \\
\leq& \Lambda + d + a + u + \alpha + \frac{pb}{c} + ky + \bar{\beta}_1 e^{\varphi} v + \bar{\beta}_2 e^{\Phi} y + pz + \frac{p\beta}{c} \\
&- \rho_1\varphi(e^{\varphi}-1) + \frac{\sigma_1^2}{2}e^{\varphi} - \rho_2\Phi(e^{\Phi}-1) + \frac{\sigma_2^2}{2}e^{\Phi}.
\end{aligned} \tag{5.7}$$

Furthermore,we obtain

$$\begin{aligned}
d(x+y) =& [\Lambda - dx - ay - \alpha y]dt \\
\leq& [\Lambda - dx - ay]dt \\
\leq& [\Lambda - \min\{d,a\}(x+y)]dt.
\end{aligned}$$

This suggests that

$$x(t)+y(t)\leq\begin{cases} x(0)+y(0),\ \text{if } x(0)+y(0)\geq\dfrac{\Lambda}{\mu_1} \\ \dfrac{\Lambda}{\mu_1},\ \text{if } x(0)+y(0)<\dfrac{\Lambda}{\mu_1}\end{cases}\leq\Delta_1, \tag{5.8}$$

where

$$\mu_1=\min\{d,a\},\Delta_1:=\max\left\{x(0)+y(0),\frac{\Lambda}{\mu_1}\right\},$$

and

$$\begin{aligned} d(v+\frac{p}{c}z)=&[ky-uv-\frac{bp}{c}z-\frac{\beta p}{c}z]dt\\ \leq&[ky-uv-\frac{bp}{c}z]dt\\ \leq&[ky-\min\{u,b\}(v+\frac{p}{c}z)]dt, \end{aligned}$$

so

$$v(t)+\frac{p}{c}z(t)\leq\begin{cases} v(0)+\dfrac{p}{c}z(0),\ \text{if } v(0)+\dfrac{p}{c}z(0)\geq\dfrac{k\Lambda}{\mu_2} \\ \dfrac{k\Lambda}{\mu_2},\ \text{if } v(0)+\dfrac{p}{c}z(0)<\dfrac{\Lambda}{\mu_2}\end{cases}\leq\Delta_2, \tag{5.9}$$

where

$$\mu_2=\min\{d,a\}\min\{u,b\},\Delta_2:=\max\left\{v(0)+\frac{p}{c}z(0),\frac{k\Lambda}{\mu_2}\right\},$$

It's worthwhile to note that

$$\lim_{|\varphi|\to\infty}(-\rho_1\varphi(e^{\varphi}-1)+M_1e^{\varphi})=-\infty,\forall M_1>0,\ \lim_{|\Phi|\to\infty}(-\rho_2\Phi(e^{\varphi}-1)+M_2e^{\Phi})=-\infty,\forall M_2>0.$$

We determine

$$\Delta_3=\sup_{\varphi\in\mathbb{R}}\left\{(\frac{\sigma_1^2}{2}+\bar{\beta_1}\Delta_2)e^{\varphi}-\rho_1\varphi(e^{\varphi}-1)\right\}<\infty, \tag{5.10}$$

$$\Delta_4=\sup_{\Phi\in\mathbb{R}}\left\{(\frac{\sigma_2^2}{2}+\bar{\beta_2}\Delta_1)e^{\Phi}-\rho_2\Phi(e^{\Phi}-1)\right\}<\infty. \tag{5.11}$$

Substituting (5.8), (5.9), (5.10) and (5.11) into (5.7) leads to that

$$\mathcal{L}\mathbb{U}=\leq\ \ \Lambda+d+a+u+\alpha+\frac{pb}{c}+\frac{p\beta}{c}+k\Delta_1+p\Delta_2+\Delta_3+\Delta_4:=\Delta_5.$$

In this context, let $\Delta_5$ be a positive constant unaffected by the variables $x,y,v,z,\varphi$, and $\Phi$. The remaining steps of the proof closely resemble those in Theorem 3.1 , and we, therefore, exclude them for brevity. This concludes the proof. □

Define the set $\Xi$ as follows

$$\Xi=\left\{(x,y,v,z,\varphi,\Phi)\in\mathbb{R}_+^4\times\mathbb{R}^2,x+y<\frac{\Lambda}{\mu_1},v+\frac{p}{c}z<\frac{k\Lambda}{\mu_2}\right\}.$$

**Remark 5.1.**

Throughout the remainder of this paper, we assume that $(x(0), y(0), v(0), z(0), \varphi(0), \Phi(0)) \in \Xi$. By Theorem 5.1, the solution $(x(t), y(t), v(t), z(t), \varphi(t), \Phi(t))$ will remain in $\Xi$ a.s.

## 5.3 Stationary distribution

In this section, we will establish sufficient conditions for the existence of a stationary distribution, which plays an important role in the analysis of disease persistence. Define

$$\mathcal{R}_0^s = \frac{\Lambda(\bar{\beta}_1 k e^{\frac{\sigma_1^2}{12\rho_1}} + \bar{\beta}_2 u e^{\frac{\sigma_2^2}{8\rho_2}})}{(a+\alpha)ud}.$$

**Theorem 5.2.**

*If $\mathcal{R}_0^s > 1$, system (5.5) has at least one stationary distribution $\Psi(.)$ on $\mathbb{R}_+^4 \times \mathbb{R}^2$.*

**Proof.** As per Remark 5.1, $(x(t), y(t), v(t), z(t), \varphi(t), \Phi(t))$ belongs to $\Xi$ almost surely. Consequently, all the descriptions of $\mathbb{R}^d$ in Lemma 2.33 need to be adjusted to $\Xi$ for system (5.5). The proof of Theorem 5.2 is partitioned into the subsequent three steps.

- **Step 1. (Construction of a non-negative $C^2$–function):** An important Lyapunov function $\mathcal{V}(x, y, v, z, \varphi, \Phi)$ is defined by

$$\begin{aligned}\mathcal{V}_0(x, y, v, z, \varphi, \Phi) = \bar{\mathcal{S}}(&-\ln y - \ln z - (\omega_1 + \omega_2)\ln x - \omega_3 \ln v + \frac{\omega_3 p}{b} z + \frac{\omega_3 c}{b} v) \\ &- \ln x - \ln y - \ln v - \ln(\mu_1 - x - y) - \ln(\mu_2 - v - \frac{p}{c} z) \\ &+ (e^{\varphi} - \varphi - 1) + (e^{\Phi} - \Phi - 1),\end{aligned}$$

  where $\omega_1, \omega_2, \omega_3$ represent positive constants to be determined subsequently, and let $\bar{\mathcal{S}}$ be a positive constant sufficiently large to satisfy the condition to be specified later in (5.29). Let

$$\begin{aligned}\mathcal{V}_1(x, y, v, z) &= -\ln y - \ln z - (\omega_1 + \omega_2)\ln x - \omega_3 \ln v + \frac{\omega_3 p}{b} z + \frac{\omega_3 c}{b} v \\ \mathcal{V}_2(x, y, v) &= -\ln x - \ln y - \ln v \\ \mathcal{V}_2(x, y, v, z) &= -\ln(\frac{\Lambda}{\mu_1} - x - y) - \ln(\frac{k\Lambda}{\mu_2} - v - \frac{p}{c} z) \\ \mathcal{V}_3(\varphi, \Phi) &= (e^{\varphi} - \varphi - 1) + (e^{\Phi} - \Phi - 1).\end{aligned}$$

Note that

$$\liminf_{(x(t),y(t),v(t),z(t),\varphi(t),\Phi(t))\to\partial\Xi} \mathcal{V}_0(x(t),y(t),v(t),z(t),\varphi(t),\Phi(t)) = \infty.$$

Here, $\partial\Xi$ represents the boundary of $\Xi$. Consequently, there exists a point $(x_0,y_0,v_0,z_0,\varphi_0,\Phi_0)\in\Xi\backslash\partial\Xi$ such that $\mathcal{V}_0(x,y,v,z,\varphi,\Phi)$ will be minimized. Subsequently, a suitable nonnegative $C^2$–Lyapunov function $\mathcal{V}(x(t),y(t),v(t),z(t),\varphi(t),\Phi(t))$ can be formulated by

$$\begin{aligned}\mathcal{V}(x(t),y(t),v(t),z(t),\varphi(t),\Phi(t)) =& \mathcal{V}_0(x(t),y(t),v(t),z(t),\varphi(t),\Phi(t))\\ &- \mathcal{V}_0(x_0,y_0,v_0,z_0,\varphi_0,\Phi_0).\end{aligned}$$

Applying Itô's formula [112] to $\mathcal{V}_1$ , we have

$$\begin{aligned}\mathcal{L}\mathcal{V}_1 =& -\frac{\bar{\omega}_1\Lambda}{x}+\bar{\omega}_1 d+\bar{\omega}_1\bar{\beta}_1 e^{\varphi}v+\bar{\omega}_1\bar{\beta}_2 e^{\Phi}y\\ &-\frac{\bar{\omega}_2\Lambda}{x}+\bar{\omega}_2 d+\bar{\omega}_2\bar{\beta}_1 e^{\varphi}v+\bar{\omega}_2\bar{\beta}_2 e^{\Phi}y\\ &-\frac{\bar{\beta}_1 e^{\varphi}xv}{y}-\bar{\beta}_2 e^{\Phi}x+a+\alpha\\ &-\frac{\bar{\omega}_3 ky}{v}+\bar{\omega}_3 u+\bar{\omega}_3 pz-cv+b+\beta\\ &+\frac{\bar{\omega}_3 cky}{b}-\frac{\bar{\omega}_3 cuv}{b}-\frac{\bar{\omega}_3 cpvz}{b}\\ &+\frac{\bar{\omega}_3 pcvz}{b}-\bar{\omega}_3 pz-\frac{\bar{\omega}_3 p\beta z}{b}\\ \leq&(-\frac{\bar{\omega}_1\Lambda}{x}-\frac{\bar{\beta}_1 e^{\varphi}xv}{y}-\frac{\bar{\omega}_3 ky}{v})+\bar{\omega}_1 d+\bar{\omega}_3 u\\ &(-\frac{\bar{\omega}_2\Lambda}{x}-\bar{\beta}_2 e^{\Phi}x)+\bar{\omega}_2 d+a+\alpha+b+\beta\\ &+\frac{\bar{\omega}_3 ck}{b}y+(\bar{\omega}_1+\bar{\omega}_2)\bar{\beta}_1 e^{\varphi}v+(\bar{\omega}_1+\bar{\omega}_2)\bar{\beta}_2 e^{\Phi}y\\ \leq&-3\sqrt[3]{\bar{\omega}_1\bar{\omega}_3\Lambda\bar{\beta}_1 k}e^{\frac{\varphi}{3}}+\bar{\omega}_1 d+\bar{\omega}_3 u-2\sqrt[2]{\bar{\omega}_2\Lambda\bar{\beta}_2}e^{\frac{\Phi}{2}}+\bar{\omega}_2 d+a+\alpha+b+\beta\\ &+\frac{\bar{\omega}_3 ck}{b}y+(\bar{\omega}_1+\bar{\omega}_2)\bar{\beta}_1 e^{\varphi}v+(\bar{\omega}_1+\bar{\omega}_2)\bar{\beta}_2 e^{\Phi}y.\end{aligned} \tag{5.12}$$

Concerning the equations fifth and sixth within the framework of system (5.5), namely

$$d\varphi(t) = -\rho_1\varphi(t)dt+\sigma_1 dB_1(t),\qquad d\Phi(t) = -\rho_2\Phi(t)dt+\sigma_2 dB_2(t).$$

Referring to the literature [113, 114, 156], it can be readily established that both $\varphi(t)$ and $\Phi(t)$ exhibit ergodic characteristics, leading to their eventual

weak convergence towards the invariant densities

$$\pi_i(g) = \frac{\sqrt{\rho_i}}{\sqrt{\pi}\sigma_i} e^{-\frac{\rho_i g^2}{\sigma_i^2}}, (g \in \mathbb{R}; i = 1,2),$$

respectively. Namely $\mathbb{N}(0, \frac{\sigma_1^2}{2\rho_1})$ and $\mathbb{N}(0, \frac{\sigma_2^2}{2\rho_2})$ Therefore, combining the ergodic theorem [115], we obtain

$$\int_{\mathbb{R}} e^{e\tau} \pi_i(\tau) d\tau = \frac{1}{\sqrt{\pi}} \int_{\mathbb{R}} e^{-\tau^2 + \frac{e^2\sigma_i^2}{4\rho_i}} d\tau = e^{\frac{e^2\sigma_i^2}{4\rho_i}}, \text{for any} \quad e > 0, (i = 1,2), \quad \text{a.s.} \tag{5.13}$$

Simplifying matters, we assign values $O_0 = \frac{\mu_2(a+\alpha)(\mathcal{R}_0^s - 1)}{4(\bar{\omega}_1 + \bar{\omega}_2)\bar{\beta}_1 k\Lambda e^{\frac{\sigma_1^2}{\rho_1}}}$, $\bar{O}_0 = \frac{\mu_1(a+\alpha)(\mathcal{R}_0^s - 1)}{4(\bar{\omega}_1 + \bar{\omega}_2)\bar{\beta}_2\Lambda e^{\frac{\sigma_2^2}{\rho_2}}}$, $a_0 = \frac{\bar{\beta}_1(\bar{\omega}_1 + \bar{\omega}_2)}{4O_0}$ and $\bar{a_0} = \frac{\bar{\beta}_2(\bar{\omega}_1 + \bar{\omega}_2)}{4\bar{O}_0}$; considering $(x(t), y(t), v(t), z(t), \varphi(t), \Phi(t)) \in \Xi$ for all $t \geq 0$ it can be asserted that

$$e^{\varphi(t)} v(t) \leq (\frac{1}{4O_0} + O_0 e^{2\varphi(t)}) v(t) \leq \frac{1}{4O_0} v(t) + O_0 \frac{k\Lambda}{\mu_2} e^{2\varphi(t)}$$

$$e^{\Phi(t)} y(t) \leq (\frac{1}{4\bar{O}_0} + \bar{O}_0 e^{2\Phi(t)}) y(t) \leq \frac{1}{4\bar{O}_0} y(t) + \bar{O}_0 \frac{\Lambda}{\mu_1} e^{2\Phi(t)}$$

and let

$$\bar{\omega}_1 = \frac{\Lambda\bar{\beta}_1 k e^{\frac{\sigma_1^2}{12\rho_1}}}{ud^2}, \bar{\omega}_2 = \frac{\Lambda\bar{\beta}_1 e^{\frac{\sigma_2^2}{8\rho_2}}}{d^2}, \bar{\omega}_3 = \frac{\Lambda\bar{\beta}_1 k e^{\frac{\sigma_1^2}{12\rho_1}}}{u^2 d}.$$

Combining (5.12) and (5.13) we get

$$\begin{aligned}
\mathcal{L}\mathcal{V}_1 \leq & -\frac{(a+\alpha)}{2}(\mathcal{R}_0^s - 1) - 3\sqrt[3]{\bar{\omega}_1\bar{\omega}_3\Lambda\bar{\beta}_1 k}(e^{\frac{\varphi}{3}} - e^{\frac{\sigma_1^2}{36\rho_1}}) - 2\sqrt[2]{\bar{\omega}_2\Lambda\bar{\beta}_2}(e^{\frac{\Phi}{2}} - e^{\frac{\sigma_2^2}{16\rho_2}}) \\
& + b + \beta + (\frac{\bar{\omega}_3 ck}{b} + \bar{a_0}) y + a_0 \frac{k\Lambda}{\mu_2} + (\bar{\omega}_1 \\
& + \bar{\omega}_2)\bar{\beta}_1 \frac{k\Lambda}{\mu_2} O_0(e^{2\varphi} - e^{\frac{\sigma_1^2}{\rho_1}}) + (\bar{\omega}_1 + \bar{\omega}_2)\bar{\beta}_2 \frac{\Lambda}{\mu_1} \bar{O}_0(e^{2\Phi} - e^{\frac{\sigma_2^2}{\rho_2}}) \\
\leq & -\frac{(a+\alpha)}{2}(\mathcal{R}_0^s - 1) + \mathcal{W}_1(\varphi, \Phi) + \mathcal{W}_2(y),
\end{aligned} \tag{5.14}$$

where

$$\begin{aligned}
\mathcal{W}_1(\varphi,\Phi) &= -3\sqrt[3]{\bar{\omega}_1\bar{\omega}_3\Lambda\bar{\beta}_1 k}(e^{\frac{\varphi}{3}} - e^{\frac{\sigma_1^2}{36\rho_1}}) - 2\sqrt[2]{\bar{\omega}_2\Lambda\bar{\beta}_2}(e^{\frac{\Phi}{2}} - e^{\frac{\sigma_2^2}{16\rho_2}}) \\
&+ (\bar{\omega}_1+\bar{\omega}_2)\bar{\beta}_1\frac{k\Lambda}{\mu_2}O_0(e^{2\varphi} - e^{\frac{\sigma_1^2}{\rho_1}}) + (\bar{\omega}_1+\bar{\omega}_2)\bar{\beta}_2\frac{\Lambda}{\mu_1}\bar{O}_0(e^{2\Phi} - e^{\frac{\sigma_2^2}{\rho_2}}) \\
\mathcal{W}_2(y) &= (\frac{\bar{\omega}_3 ck}{b} + \bar{a_0})y + b + \beta + a_0\frac{k\Lambda}{\mu_2}.
\end{aligned}$$

In the same vein, utilizing Itô's formula on $\mathcal{V}_2, \mathcal{V}_3$ and $\mathcal{V}_4$, respectively, results in

$$\begin{aligned}
\mathcal{L}\mathcal{V}_2 =& -\frac{\Lambda}{x} - \frac{\bar{\beta}_1 e^{\varphi} xv}{y} - \frac{ky}{v} + d + a + \alpha + u \\
&+ pz - \bar{\beta}_2 e^{\Phi} x + \bar{\beta}_1 e^{\varphi} v + \bar{\beta}_2 e^{\Phi} y \\
\leq& -\frac{\Lambda}{x} - \frac{ky}{v} + d + a + \alpha + u \\
&+ \frac{c\Lambda k}{\mu_2} + \bar{\beta}_1\frac{k\Lambda}{\mu_2}e^{\varphi} + \bar{\beta}_2\frac{\Lambda}{\mu_1}e^{\Phi},
\end{aligned} \tag{5.15}$$

$$\begin{aligned}
\mathcal{L}\mathcal{V}_3 =& \frac{\Lambda - dx - ay - \alpha y}{\bar{\mu_1} - x - y} + \frac{ky - uv - \frac{bp}{c}z - \frac{\beta p}{c}z}{\bar{\mu_2} - v - \frac{p}{c}z} \\
\leq& \frac{-\alpha y}{\bar{\mu_1} - x - y} + \frac{-\frac{\beta p}{c}z}{\bar{\mu_2} - v - \frac{p}{c}z} + \bar{\mu_1} + \bar{\mu_2},
\end{aligned} \tag{5.16}$$

where $\bar{\mu_1} = \frac{\Lambda}{\mu_1}, \bar{\mu_2} = \frac{k\Lambda}{\mu_2}$, and

$$\mathcal{L}\mathcal{V}_4 = -\rho_1\varphi(e^{\varphi} - 1) + \frac{\sigma_1^2}{2}e^{\varphi} - \rho_2\Phi(e^{\Phi} - 1) + \frac{\sigma_2^2}{2}e^{\Phi}. \tag{5.17}$$

By (5.14), (5.15), (5.16) and (5.17), we get

$$\begin{aligned}
\mathcal{L}\mathcal{V} \leq& -\frac{\bar{S}(a+\alpha)}{2}(\mathcal{R}_0^s - 1) + \bar{S}\mathcal{W}_1(\varphi,\Phi) + \bar{S}\mathcal{W}_2(y) \\
&- \frac{\Lambda}{x} - \frac{ky}{v} - \frac{\alpha y}{\mu_1 - x - y} - \frac{\frac{\beta p}{c}z}{\mu_2 - v - \frac{p}{c}z} \\
&+ d + a + \alpha + u + \bar{\mu_1} + \bar{\mu_2} + \frac{c\Lambda k}{\mu_2} + (\frac{\sigma_1^2}{2} + \bar{\beta}_1\frac{k\Lambda}{\mu_2})e^{\varphi} + (\frac{\sigma_2^2}{2} + \bar{\beta}_2\frac{\Lambda}{\mu_1})e^{\Phi} \\
&- \rho_1\varphi(e^{\varphi} - 1) - \rho_2\Phi(e^{\Phi} - 1).
\end{aligned} \tag{5.18}$$

Considering

$$\Delta_6 = \sup_{\varphi\in\mathbb{R}}\left\{(\frac{\sigma_1^2}{2} + \frac{\bar{\beta}_1 k\Lambda}{\mu_2})e^{\varphi} - \frac{\rho_1}{2}\varphi(e^{\varphi} - 1)\right\} < \infty,$$

$$\Delta_7 = \sup_{\Phi\in\mathbb{R}}\left\{(\frac{\sigma_2^2}{2}+\frac{\bar{\beta_2}\Lambda}{\mu_1})e^{\Phi}-\frac{\rho_2}{2}\Phi(e^{\Phi}-1)\right\}<\infty,$$

it follows that

$$\mathcal{L}\mathcal{V}\leq \Psi(x,y,v,z,\varphi,\Phi)+\bar{\mathcal{S}}\mathcal{W}_1(\varphi,\Phi), \tag{5.19}$$

where

$$\begin{aligned}\Psi(x,y,v,z,\varphi,\Phi) &= -\frac{\bar{\mathcal{S}}(a+\alpha)}{2}(\mathcal{R}_0^s-1)+\bar{\mathcal{S}}\mathcal{W}_2(y)\\ &-\frac{\Lambda}{x}-\frac{ky}{v}-\frac{\alpha y}{\mu_1-x-y}-\frac{\frac{\beta p}{c}z}{\mu_2-v-\frac{p}{c}z}\\ &+d+a+\alpha+u+b+\beta+\bar{\mu_1}+\bar{\mu_2}+\Delta_6+\Delta_7+\frac{c\Lambda k}{\mu_2}-\frac{\rho_1}{2}\varphi(e^{\varphi}-1)-\frac{\rho_2}{2}\Phi(e^{\Phi}-1).\end{aligned} \tag{5.20}$$

- **Step 2. (Construction of a compact set):** Define a bounded closed set $\mathbb{H}$ by

$$\begin{aligned}\mathbb{H}_{\epsilon} := \Big\{&(x,y,v,z,\varphi,\Phi)^T\in\Xi : x\geq\epsilon, y\geq\epsilon, v\geq\epsilon^2, x+y\leq\bar{\mu_1}-\epsilon^2, v+\frac{p}{c}z\leq\bar{\mu_2}-\epsilon^2,\\ &|\varphi|\leq\frac{1}{\epsilon},|\Phi|\leq\frac{1}{\epsilon}\Big\}.\end{aligned}$$

Here, consider a positive constant $\epsilon$ that is sufficiently small, meeting the following set of inequalities

$$-\frac{\Lambda}{\epsilon}+\Delta_9\leq -1, \tag{5.21}$$

$$\epsilon<\frac{b}{\bar{\mathcal{S}}(\bar{\omega}_3 ck+b\bar{a_0})}, \tag{5.22}$$

$$-\frac{k}{\epsilon}+\Delta_9\leq -1, \tag{5.23}$$

$$-\frac{\alpha}{\epsilon}+\Delta_9\leq -1, \tag{5.24}$$

$$-\frac{\beta}{\epsilon}+\Delta_9\leq -1, \tag{5.25}$$

$$\Delta_9-\frac{\rho_1+\rho_2}{\epsilon}(1-e^{\frac{-1}{\epsilon}})\leq -1. \tag{5.26}$$

Our emphasis is placed on the sets listed below

$$\begin{aligned}
&\mathbb{H}^c_{\epsilon,1} = \Big\{(x,y,v,z,\varphi,\Phi)^T \in \Xi, x < \epsilon\Big\}, \mathbb{H}^c_{\epsilon,2} = \Big\{(x,y,v,z,\varphi,\Phi)^T \in \Xi, y < \epsilon\Big\},\\
&\mathbb{H}^c_{\epsilon,3} = \Big\{(x,y,v,z,\varphi,\Phi)^T \in \Xi, v < \epsilon^2, y \geq \epsilon\Big\},\\
&\mathbb{H}^c_{\epsilon,4} = \Big\{(x,y,v,z,\varphi,\Phi)^T \in \Xi, x+y < \bar{\mu_1} - \epsilon^2, y \geq \epsilon\Big\},\\
&\mathbb{H}^c_{\epsilon,5} = \Big\{(x,y,v,z,\varphi,\Phi)^T \in \Xi, v + \frac{p}{c}z < \bar{\mu_2} - \epsilon^2, z \geq \epsilon\Big\},\\
&\mathbb{H}^c_{\epsilon,6} = \Big\{(x,y,v,z,\varphi,\Phi)^T \in \Xi, |\varphi| > \frac{1}{\epsilon}, |\Phi| > \frac{1}{\epsilon}\Big\}.
\end{aligned}$$

Clearly, $\Xi \backslash \mathbb{H}_\epsilon = \bigcup_{i=1}^{6} \mathbb{H}^c_{\epsilon,i^*}$. Subsequently, we will verify that $\dot{\Psi}(x,y,v,z,\varphi,\Phi) < -1$ holds on $\mathbb{H}_\epsilon$, necessitating confirmation across the six aforementioned regions.

– **Case 1 :** For any $(x,y,v,z,\varphi,\Phi)^T \in \mathbb{H}^c_{\epsilon,1}$, in view of (5.20), we have

$$\begin{aligned}
\dot{\Psi}(x,y,v,z,\varphi,\Phi) \leq & -\frac{\Lambda}{x} + \bar{\mathcal{S}}\mathcal{W}_2(y) + d + a + \alpha + u \\
& + b + \beta + \bar{\mu_1} + \bar{\mu_2} + \Delta_6 + \Delta_7 + \frac{c\Lambda k}{\mu_2} \\
\leq & -\frac{\Lambda}{\epsilon} + \Delta_9 \\
\leq & -1,
\end{aligned} \tag{5.27}$$

which follows from (5.21) and
$\Delta_9 = \bar{\mathcal{S}}[(\frac{\bar{\omega}_3 ck}{b} + \bar{a_0})\frac{\Lambda}{\mu_1} + b + \beta + a_0\frac{k\Lambda}{\mu_2}] + d + a + \alpha + u + b + \beta + \bar{\mu_1} + \bar{\mu_2} + \Delta_6 + \Delta_7 + \frac{c\Lambda k}{\mu_2} < \infty$ .

– **Case 2 :** For any $(x,y,v,z,\varphi,\Phi)^T \in \mathbb{H}^c_{\epsilon,2}$, according to (5.20), we obtain

$$\begin{aligned}
\dot{\Psi}(x,y,v,z,\varphi,\Phi) \leq & -\frac{\bar{\mathcal{S}}(a+\alpha)}{2}(\mathcal{R}_0^s - 1) + \bar{\mathcal{S}}\mathcal{W}_2(y) \\
& + d + a + \alpha + u + b + \beta + \bar{\mu_1} + \bar{\mu_2} + \Delta_6 + \Delta_7 + \frac{c\Lambda k}{\mu_2} \\
\leq & -\frac{\bar{\mathcal{S}}(a+\alpha)}{2}(\mathcal{R}_0^s - 1) + \bar{\mathcal{S}}(\frac{\bar{\omega}_3 ck}{b} + \bar{a_0})\epsilon + \Delta_8 \\
\leq & -1,
\end{aligned} \tag{5.28}$$

as a consequence of (5.22) and
$\Delta_8 = \bar{\mathcal{S}}(b + \beta + \frac{a_0 k\Lambda}{\mu_2}) + d + a + \alpha + u + b + \beta + \bar{\mu_1} + \bar{\mu_2} + \Delta_6 + \Delta_7 + \frac{c\Lambda k}{\mu_2} < \infty,$

$$-\frac{\bar{\mathcal{S}}(a+\alpha)}{2}(\mathcal{R}_0^s - 1) + \Delta_8 \leq -2. \tag{5.29}$$

– **Case 3 :** For any $(x, y, v, z, \varphi, \Phi)^T \in \mathbb{H}^c_{\epsilon,3}$, from (5.20), we get

$$\begin{aligned}\dot{\mathcal{U}}(x, y, v, z, \varphi, \Phi) \leq & -\frac{ky}{v} + \bar{\mathcal{S}}\mathcal{W}_2(y) + d + a + \alpha + u \\ & + b + \beta + \bar{\mu_1} + \bar{\mu_2} + \Delta_6 + \Delta_7 + \frac{c\Lambda k}{\mu_2} \\ \leq & -\frac{k\epsilon}{\epsilon^2} + \Delta_9 \\ \leq & -\frac{k}{\epsilon} + \Delta_9 \\ \leq & -1,\end{aligned} \tag{5.30}$$

as a result of (5.23).

– **Case 4 :** For any $(x, y, v, z, \varphi, \Phi)^T \in \mathbb{H}^c_{\epsilon,4}$, according to (5.20), we have

$$\begin{aligned}\dot{\mathcal{U}}(x, y, v, z, \varphi, \Phi) \leq & -\frac{\alpha y}{\mu_1 - x - y} + \bar{\mathcal{S}}\mathcal{W}_2(y) + d + a + \alpha + u + b + \beta + \bar{\mu_1} + \bar{\mu_2} \\ & + \Delta_6 + \Delta_7 + \frac{c\Lambda k}{\mu_2} \\ \leq & -\frac{\alpha\epsilon}{\epsilon^2} + \Delta_9 \\ \leq & -\frac{\alpha}{\epsilon} + \Delta_9 \\ \leq & -1,\end{aligned} \tag{5.31}$$

which follows from (5.24).

– **Case 5 :** For any $(x, y, v, z, \varphi, \Phi)^T \in \mathbb{H}^c_{\epsilon,5}$, by (5.20), we obtain

$$\begin{aligned}\dot{\mathcal{U}}(x, y, v, z, \varphi, \Phi) \leq & -\frac{\frac{\beta p}{c} z}{\mu_2 - v - \frac{p}{c} z} + \bar{\mathcal{S}}\mathcal{W}_2(y) + d + a + \alpha + u + b + \beta + \bar{\mu_1} + \bar{\mu_2} \\ & + \Delta_6 + \Delta_7 + \frac{c\Lambda k}{\mu_2} \\ \leq & -\frac{\beta\epsilon}{\epsilon^2} + \Delta_9 \\ \leq & -\frac{\beta}{\epsilon} + \Delta_9 \\ \leq & -1,\end{aligned} \tag{5.32}$$

as a consequence of (5.25).

– **Case 6 :** For any $(x,y,v,z,\varphi,\Phi)^T \in \mathbb{H}^c_{\epsilon,6}$, from (5.20), it follows that

$$\begin{aligned}\mathcal{Y}(x,y,v,z,\varphi,\Phi) \leq & -\frac{\rho_1}{2}\varphi(e^{\varphi}-1)-\frac{\rho_2}{2}\Phi(e^{\Phi}-1)+\bar{\mathcal{S}}\mathcal{W}_2(y)+d+a+\alpha+\\ & u+b+\beta+\bar{\mu_1}+\bar{\mu_2}+\Delta_6+\Delta_7+\frac{c\Lambda k}{\mu_2}\\ \leq & \Delta_9-\frac{\rho_1+\rho_2}{\epsilon}(1-e^{\frac{-1}{\epsilon}})\\ \leq & -1,\end{aligned} \tag{5.33}$$

as a result of (5.26).

Considering (5.27), (5.28), (5.30), (5.31), (5.32) and (5.33), it is evident that we can readily deduce the existence of a sufficiently small $\epsilon$

$$\mathcal{Y}(x,y,v,z,\varphi,\Phi) \leq -1 \qquad \text{for any} (x,y,v,z,\varphi,\Phi)^T \in \Xi \setminus \mathbb{H}_{\epsilon}. \tag{5.34}$$

Let

$$\begin{aligned}\Delta_{10} := \sup_{(x,y,v,z,\varphi,\Phi)^T\in\mathbb{R}^4_+\times\mathbb{R}^2} \Bigg\{ & -\frac{\bar{\mathcal{S}}(a+\alpha)}{2}(\mathcal{R}_0^s-1)+\bar{\mathcal{S}}\mathcal{W}_2(y)-\frac{\Lambda}{x}-\frac{ky}{v}-\frac{\alpha y}{\mu_1-x-y}\\ & -\frac{\frac{\beta p}{c}z}{\mu_2-v-\frac{p}{c}z}+d+a+\alpha+u+b+\beta+\bar{\mu_1}+\bar{\mu_2}+\Delta_6+\Delta_7+\frac{c\Lambda k}{\mu_2}\\ & -\frac{\rho_1}{2}\varphi(e^{\varphi}-1)-\frac{\rho_2}{2}\Phi(e^{\Phi}-1)\Bigg\}.\end{aligned}$$

Then :

$$\mathcal{Y}(x,y,v,z,\varphi,\Phi) \leq \Delta_{10} < \infty \qquad \text{for any} \quad (x,y,v,z,\varphi,\Phi)^T \in \mathbb{R}^4_+ \times \mathbb{R}^2. \tag{5.35}$$

- **Step 3. (Existence):** For any initial condition $(x(0),y(0),v(0),z(0),\varphi(0),\Phi(0)) \in \Xi$, integrating both sides of equation (5.19) from 0 to $t$ and subsequently com-

puting the mathematical expectation results in

$$\begin{aligned}0 \leq & \frac{\mathbb{E}\mathcal{V}(x(t), y(t), v(t), z(t), \varphi(t), \Phi(t))}{t} \\ & = \frac{\mathbb{E}\mathcal{V}(x(0), y(0), v(0), z(0), \varphi(0), \Phi(0))}{t} \\ & + \frac{1}{t}\int_0^t \mathbb{E}(\mathcal{L}\mathcal{V}(x(s), y(s), v(s), z(s), \varphi(s), \Phi(s)))ds \\ & \leq \frac{\mathbb{E}\mathcal{V}(x(0), y(0), v(0), z(0), \varphi(0), \Phi(0))}{t} \\ & + \frac{1}{t}\int_0^t \mathbb{E}(\dot{\mathcal{V}}(x(s), y(s), v(s), z(s), \varphi(s), \Phi(s)))ds \\ & + \bar{\mathcal{S}}\mathbb{E}(\frac{1}{t}\int_0^t \mathcal{W}_1(\varphi(s), \Phi(s)))ds.\end{aligned} \tag{5.36}$$

To a certain extent, employing (5.34) and (5.35), one obtains

$$\begin{aligned}& \frac{1}{t}\int_0^t \mathbb{E}(\dot{\mathcal{V}}(x(s), y(s), v(s), z(s), \varphi(s), \Phi(s)))ds \\ =& \frac{1}{t}\int_0^t \mathbb{E}(\dot{\mathcal{V}}(x(s), y(s), v(s), z(s), \varphi(s), \Phi(s)))\mathbb{1}_{(x(s),y(s),v(s),z(s),\varphi(s),\Phi(s))^T \in \mathbb{H}_\epsilon} ds \\ & + \frac{1}{t}\int_0^t \mathbb{E}(\dot{\mathcal{V}}(x(s), y(s), v(s), z(s), \varphi(s), \Phi(s)))\mathbb{1}_{(x(s),y(s),v(s),z(s),\varphi(s),\Phi(s))^T \in (\Xi \setminus \mathbb{H}_\epsilon)} ds \\ \leq & \frac{\Delta_{10}}{t}\int_0^t \mathbb{1}_{(x(s),y(s),v(s),z(s),\varphi(s),\Phi(s))^T \in \mathbb{H}_\epsilon} ds - \frac{1}{t}\int_0^t \mathbb{1}_{(x(s),y(s),v(s),z(s),\varphi(s),\Phi(s))^T \in (\Xi \setminus \mathbb{H}_\epsilon)} ds \\ \leq & -1 + \frac{\Delta_{10}+1}{t}\int_0^t \mathbb{1}_{(x(s),y(s),v(s),z(s),\varphi(s),\Phi(s))^T \in \mathbb{H}_\epsilon} ds.\end{aligned} \tag{5.37}$$

Combining (5.36) and (5.37) yields

$$\begin{aligned}\frac{\Delta_{10}+1}{t}\int_0^t \mathbb{1}_{(x(s),y(s),v(s),z(s),\varphi(s),\Phi(s))^T \in \mathbb{H}_\epsilon} ds \geq & 1 - \frac{\mathbb{E}\mathcal{V}(x(0), y(0), v(0), z(0), \varphi(0), \Phi(0))}{t} \\ & - \bar{\mathcal{S}}\mathbb{E}(\frac{1}{t}\int_0^t \mathcal{W}_1(\varphi(s), \Phi(s)))ds.\end{aligned} \tag{5.38}$$

From a different perspective, relying on (5.13), the strong law of large numbers [112], and the ergodicity of $\Upsilon_i(t)$, $(i = 1, 2, \Upsilon_1(t) = \varphi(t)$ and $\Upsilon_2(t) = \Phi(t))$, we obtain

$$\begin{aligned}\lim_{t\to\infty}\mathbb{E}\left[\frac{1}{t}\int_0^t \left(e^{\theta\Upsilon_i(s)} - e^{\frac{\theta^2\sigma_i^2}{4\rho_i}}\right)ds\right] &= \mathbb{E}\left(\lim_{t\to\infty}\frac{1}{t}\int_0^t e^{\theta\Upsilon_i(s)}ds\right) - e^{\frac{\theta^2\sigma_i^2}{4\rho_i}} \\ &= \int_{\mathbb{R}} e^{\theta\tau}\pi_i(\tau)d\tau - e^{\frac{\theta^2\sigma_i^2}{4\rho_i}} = 0, \quad \text{a.s. } \forall\theta > 0.\end{aligned}$$

This, coupled with the formulations of $\mathcal{W}_1(\varphi(s),\Phi(s))$, implies

$$\lim_{t\to\infty}\mathbb{E}(\frac{1}{t}\int_0^t \mathcal{W}_1(\varphi(s),\Phi(s)))ds=0, \quad \text{a.s.} \tag{5.39}$$

By taking the lower limit on both sides of (5.38) and consolidating with (5.39), one arrives at:

$$\liminf_{t\to\infty}\frac{1}{t}\int_0^t \mathbb{1}_{(x(s),y(s),v(s),z(s),\varphi(s),\Phi(s))^T\in\mathbb{H}_\epsilon}ds\geq\frac{1}{\Delta_{10}+1}>0 \qquad a.s. \tag{5.40}$$

Considering the definition of event probability and invoking Fatou's lemma [136], the expression (5.40) is tantamount to

$$\liminf_{t\to\infty}\frac{1}{t}\int_0^t \mathbf{P}(s,(x(s),y(s),v(s),z(s),\varphi(s),\Phi(s)),\mathbb{H}_\epsilon)ds\geq\frac{1}{\Delta_{10}+1}>0 \qquad a.s. \tag{5.41}$$

In the context of $\mathbf{P}(t,(x(t),y(t),v(t),z(t),\varphi(t),\Phi(t)),\mathbb{H}_\epsilon)$ denoting the transition probability for $(t,(x(t),y(t),v(t),z(t),\varphi(t),\Phi(t))^T$ within set $\mathbb{H}_\epsilon$, Lemma 2.33 establishes the existence of at least one stationary distribution $\Psi(.)$ on $\mathbb{R}^4_+\times\mathbb{R}^2$ for the system defined by system (5.5). This distribution possesses the Feller property, thereby concluding the proof. □

## 5.4 Probability density function

In this section, we will derive the explicit formula for the probability density function of the distribution $\Psi(.)$. Mathematically, obtaining the probability density for system (5.5) is a more detailed and specific task compared to finding the stationary distribution. We begin by presenting equivalent transformations of system (5.5).

### 5.4.1 Equivalent reformulations of system (5.5)

Firstly, we define a quasi-chronic infection equilibrium $E^+=(x^+,y^+,v^+,z^+,\varphi^+,\Phi^+)^T$ subject to stochastic influences through the following equations

$$\begin{cases}\Lambda-dx^+-\bar{\beta}_1e^{\varphi^+}x^+v^+-\bar{\beta}_2e^{\Phi^+}x^+y^+=0,\\ d\bar{\beta}_1e^{\varphi^+}x^+v^++\bar{\beta}_2e^{\Phi^+}x^+y^+-ay^+-\alpha y^+=0,\\ ky^+-uv^+-pv^+z^+=0,\\ cv^+z^+-bz^+-\beta z^+=0,\\ -\rho_1\varphi^+=0,\\ -\rho_2\Phi^+=0.\end{cases} \tag{5.42}$$

By solving Equation (5.42), we find that if $\mathcal{R}_0^s > 1$, then

$$x^+ = x^*, y^+ = y^*, v^+ = v^*, z^+ = z^*, \varphi^+ = \Phi^+ = 0,$$

where $x^*, y^*, v^*$ and $z^*$ are the same as in Section 5.1.
Let $(x_1, x_2, x_3, x_4)^T = (x-x^+, y-y^+, v-v^+, z-z^+)^T$ According to Itô's integral and system (5.5), the corresponding linearized system of (5.5) around $E^+$ is given by

$$\begin{cases} dx_1 = (-a_{11}x_1 - a_{12}x_2 - a_{13}x_3 - a_{15}x_5 - a_{16}x_6)dt, \\ dx_2 = (a_{21}x_1 - a_{22}x_2 + a_{23}x_3 + a_{25}x_5 + a_{26}x_6)dt, \\ dx_3 = (a_{32}x_2 - a_{33}x_3 - a_{34}x_4)dt, \\ dx_4 = (a_{43}x_3 - a_{44}x_4)dt, \\ dx_5 = -\rho_1 x_5 dt + \sigma_1 dB_1(t), \\ dx_6 = -\rho_2 x_6 dt + \sigma_2 dB_2(t), \end{cases} \tag{5.43}$$

where

$$a_{11} = d + \bar{\beta}_1 v^+ + \bar{\beta}_2 y^+ > 0, a_{12} = \bar{\beta}_2 x^+ > 0,$$
$$a_{13} = \bar{\beta}_1 x^+ > 0, a_{15} = \bar{\beta}_1 x^+ v^+ > 0, a_{16} = \bar{\beta}_2 x^+ y^+ > 0,$$
$$a_{21} = \bar{\beta}_1 v^+ > 0, a_{22} = a + \alpha - \bar{\beta}_2 x^+ = \frac{\bar{\beta}_1 x^+ v^+}{y^+} > 0,$$
$$a_{23} = \bar{\beta}_1 x^+ > 0, a_{25} = \bar{\beta}_1 x^+ v^+ > 0, a_{26} = \bar{\beta}_2 x^+ y^+ > 0,$$
$$a_{32} = k > 0, a_{33} = u + pz^+ > 0, a_{34} = pv^+ > 0, a_{43} = cz^+ > 0, a_{44} = b + \beta - cv^+ = 0.$$

### 5.4.2 Probability density of stationary distribution $\Psi(.)$

**Theorem 5.3.**

*If $R_0^s > 1$, the stationary distribution $\Psi(.)$ around $E^+$ approximately has a normal density function, which is defined by*

$$\mathcal{J}(x, y, v, z, \varphi, \phi) = (2\pi)^{-3}|\Sigma|^{-1/2} \exp\left\{-\frac{1}{2}(x-x^+, y-y^+, v-v^+, z-z^+, \varphi-\varphi^+, \phi-\phi^+)\Sigma^{-1}\right.$$
$$\left.(x-x^+, y-y^+, v-v^+, z-z^+, \varphi-\varphi^+, \phi-\phi^+)^T\right\},$$

*where $\Sigma$ is a positive definite matrix, and the specific form of $\Sigma$ is given as follows*

1. *If $P_2 = 0$, then*

$$\Sigma = \bar{\bar{\Omega}}_1^2 (Q_{11}Q_{10}Q_9Q_3Q_2Q_1)^{-1} \bar{\bar{\Sigma}}_{01} [(Q_{11}Q_{10}Q_9Q_3Q_2Q_1)^{-1}]^T$$
$$+ \bar{\bar{\Omega}}_2^2 (Q_{22}Q_{21}Q_{20}Q_{14}Q_{13}Q_{12})^{-1} \bar{\bar{\Sigma}}_{02} [(Q_{22}Q_{21}Q_{20}Q_{14}Q_{13}Q_{12})^{-1}]^T.$$

2. *If* $P_4 \neq 0$, *then*

$$\Sigma = \Omega_1^2 (Q_6 Q_5 Q_4 Q_3 Q_2 Q_1)^{-1} \Sigma_{01} [(Q_6 Q_5 Q_4 Q_3 Q_2 Q_1)^{-1}]^T + \Omega_2^2 (Q_{17} Q_{16} Q_{15} Q_{14} Q_{13} Q_{12})^{-1} \Sigma_{02} [(Q_{17} Q_{16} Q_{15} Q_{14} Q_{13} Q_{12})^{-1}]^T.$$

3. *If* $P_4 = 0$, *then*

$$\Sigma = \bar{\Omega_1}^2 (Q_8 Q_7 Q_4 Q_3 Q_2 Q_1)^{-1} \bar{\Sigma_{01}} [(Q_8 Q_7 Q_4 Q_3 Q_2 Q_1)^{-1}]^T + \bar{\Omega_2}^2 (Q_{19} Q_{18} Q_9 Q_8 Q_7 Q_6)^{-1} \bar{\Sigma_{02}} [(Q_{19} Q_{18} Q_9 Q_8 Q_7 Q_6)^{-1}]^T,$$

*where* $P_i (i = 1, ..., 7)$ *and* $P_1 \neq 0; \Omega_i, \bar{\Omega_i}, \bar{\bar{\Omega}}_i (i = 1, 2); \vartheta_i, \bar{\vartheta}_i, \bar{\bar{\vartheta}}_i, \chi_i, \bar{\chi}_i, \bar{\bar{\chi}}_i (i = 1, ..., 5)$ *and* $o_i, \bar{o_1} (i = 1, ..., 4)$ *are shown in* (5.49), (5.50), (5.51), (5.52), (5.53), (5.55), (5.57), (5.58), (5.61), (5.63) *and* (5.65), *respectively.* $Q_j$, $\Sigma_{0i}$, $\bar{\Sigma_{0i}}$ *and* $\bar{\bar{\Sigma}}_{0i}$ *will be given in the following proof,* $\forall i = 1, 2 \; ; j = 1, ..., 22$.

**Lemma 5.4 ( [93], Definition A.1, page 17).**

*If the matrix* $A$ *is a* $l$*-dimensional square matrix whose characteristic polynomial is defined as* $\varphi_\lambda(A) = \lambda^l + a_1 \lambda^{l-1} + a_2 \lambda^{l-2} + \cdots + a_{l-1} \lambda + a_l$,

$$A^{(k)} = \begin{vmatrix} a_1 & a_3 & a_5 & \cdots & a_{2k-1} \\ 1 & a_2 & a_4 & \cdots & a_{2k-2} \\ 0 & a_1 & a_3 & \cdots & a_{2k-3} \\ \vdots & \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & 0 & \cdots & a_k \end{vmatrix},$$

$k = 1, 2, ..., l$ *then matrix* $A$ *has all negative real-part eigenvalues. In this case, we call* $A \in R\bar{H}(l)$.

**Lemma 5.5 ( [157], Lemma 2.4, page 08).**

*Consider the five-dimensional real algebraic equation*

$$H_0^2 + A_0 \Sigma_0 + \Sigma_0 A_0^T = 0,$$

*where* $\Sigma_0$ *is a symmetric matrix,* $H_0 = diag(1, 0, 0, 0, 0)$ *and*

$$A_0 = \begin{pmatrix} -g_1 & -g_2 & -g_3 & -g_4 & -g_5 \\ 1 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 \end{pmatrix}.$$

*If* $A_0 \in \mathbf{R\bar{H}}(5)$, *or its equivalent*

$$M_1 = g_1 > 0, M_2 = g_1 g_2 - g_3 > 0, M_4 = g_1(g_2 g_3 - g_1 g_4) - (g_1^2 - g_1 g_5) > 0,$$
$$M_6 = (g_1 g_2 - g_3)(g_3 g_4 - g_5) - (g_1 g_4 - g_5)^2 > 0, \text{ and } g_3 > 0, g_4 > 0,$$

*then*

$$\Sigma_0 = \begin{bmatrix} \theta_{11} & 0 & -\theta_{22} & 0 & \theta_{33} \\ 0 & \theta_{22} & 0 & -\theta_{33} & 0 \\ -\theta_{22} & 0 & \theta_{33} & 0 & -\theta_{44} \\ 0 & -\theta_{33} & 0 & \theta_{44} & 0 \\ \theta_{13} & 0 & -\theta_{44} & 0 & \theta_{55} \end{bmatrix}$$

*is a positive definite matrix, where*

$$\theta_{11} = \frac{g_2(g_3 g_4 - g_2 g_5) - g_4(g_1 g_4 - g_5)}{2M_4}, \theta_{22} = \frac{g_3 g_4 - g_2 g_5}{2M_4},$$
$$\theta_{33} = \frac{g_1 g_4 - g_5}{2M_4}, \theta_{44} = \frac{g_1 g_4 - g_5}{2M_4}, \theta_{55} = \frac{M_3}{2M_4}.$$

**Lemma 5.6 ( [158], Lemma 4.1, page 18).**
*Let* $A_0\Sigma_0 + H_0^2 + \Sigma_0 A_0^T = 0$, *be an algebraic equation where* $H_0 = diag(1,0,0,0)$ *and* $\Sigma_0$ *is a symmetric matrix real, and the standard matrix*

$$A_0 = \begin{pmatrix} -\bar{g}_1 & -\bar{g}_2 & -\bar{g}_3 & -\bar{g}_4 \\ 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \end{pmatrix}.$$

*If* $\bar{g}_1 > 0, \bar{g}_3 > 0, \bar{g}_4 > 0$ *and* $\bar{g}_1\bar{g}_2\bar{g}_3 - \bar{g}_3^2 - \bar{g}_1^2\bar{g}_4 > 0$, *then* $\Sigma_0$ *is a positive definite matrix, where*

$$\Sigma_0 = \begin{pmatrix} \frac{g_2 g_3 - g_1 g_4}{2(g_1 g_2 g_3 - g_3^2 - g_1^2 g_4)} & 0 & -\frac{g_3}{2(g_1 g_2 g_3 - g_3^2 - g_1^2 g_4)} & 0 \\ 0 & \frac{\bar{g}_3}{2(g_1 g_2 g_3 - g_3^2 - g_1^2 g_4)} & 0 & -\frac{\bar{g}_1}{2(g_1 g_2 g_3 - g_3^2 - g_1^2 g_4)} \\ -\frac{g_3}{2(g_1 g_2 g_3 - g_3^2 - g_1^2 g_4)} & 0 & \frac{g_1}{2(g_1 g_2 g_3 - g_3^2 - g_1^2 g_4)} & 0 \\ 0 & -\frac{\tau_1}{2(\tau_1 \tau_2 \tau_3 - g_3^2 - \tau_1^2 g_4)} & 0 & \frac{g_1 g_2 - g_3}{2(g_1 g_2 g_3 - g_3^2 - g_1^2 g_4)} \end{pmatrix}.$$

**Proof.** To simplify matters, let $X = (x_1, x_2, x_3, x_4, x_5, x_6)^T, B(t) = (0,0,0,0,B_1(t), B_2(t))^T$,

$$A = \begin{pmatrix} -a_{11} & -a_{12} & -a_{13} & 0 & -a_{15} & -a_{16} \\ a_{21} & -a_{22} & a_{23} & 0 & a_{25} & a_{26} \\ 0 & 0 & a_{43} & -a_{44} & -0 & 0 \\ 0 & 0 & 0 & 0 & -\rho_1 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\rho_2 \end{pmatrix}, \quad H = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \sigma_1 & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma_2 \end{pmatrix},$$

with these notations, system (5.43) can be rewritten in the following equivalent form

$$d\Upsilon(t) = A\Upsilon(t)dt + HdB(t). \tag{5.44}$$

According to the theory of continuous Markov processes [159], system (5.44) has a unique probability density $\mathcal{J}(x_1, x_2, x_3, x_4, x_5, x_6)$, which is governed by the following five-dimensional Fokker-Planck equation

$$\frac{\partial}{\partial t}\mathcal{J}(\Upsilon(t), t) + \frac{\partial}{\partial \Upsilon(t)}[A\Upsilon(t)\mathcal{J}(\Upsilon(t), t)] - \frac{\sigma_1^2}{2}\frac{\partial^2}{\partial x_5^2}\mathcal{J}(X(t), t) - \frac{\sigma_2^2}{2}\frac{\partial^2}{\partial x_6^2}\mathcal{J}(\Upsilon(t), t) = 0. \tag{5.45}$$

Next, we will derive the precise expression for the probability density by solving Equation (5.45). Note that $\frac{\partial}{\partial t}\mathcal{J}(\Upsilon(t), t) = 0$ in the stationary case. Because the diffusion matrix $H$is constant, the probability density $\mathcal{J}(\Upsilon)$ can be expressed as a normal distribution, as demonstrated in Roozen's study [160], namely

$$\mathcal{J}(\Upsilon) = s\exp\left(-\frac{1}{2}\Upsilon^T D\Upsilon\right),$$

where $D$ is a real symmetric matrix and $s$ is a positive constant that meets the normalization condition $\int_{\mathbb{R}^6} \mathcal{J}(\Upsilon)d\Upsilon = 1$.
Inserting these results into Equation (5.45), we get that the constant $s = (2\pi)^{-3}|\Sigma|^{-\frac{1}{2}}$ and $D$ satisfies the subsequent algebraic equation

$$DH^2D + DA + A^TD = 0. \tag{5.46}$$

If $D$ is positive definite and therefore invertible, let $D^{-1} = \Sigma$. Under this condition, the algebraic equation (5.46) can be reformulated as follows

$$H^2 + A\Sigma + \Sigma A^T = 0 \tag{5.47}$$

Taking into account the finite independent superposition principle [159],and so

$$H_i^2 + A\Sigma_i + \Sigma_i A^T = 0, \qquad i = 1,2,$$

where $H_1^2 = diag(0,0,0,0,\sigma_1^2,0)$, $H_2^2 = diag(0,0,0,0,0,\sigma_1^2)$, and the symmetric matrices $\Sigma_i$ $(i = 1,2)$ are their solutions, respectively. It is easy to see that $\sigma^2 = \sigma_1^2 + \sigma_2^2$ and $H^2 = H_1^2 + H_2^2$. We are now ready to determine the specific form of $\sigma$ and verify its positive definiteness, which we will accomplish in two steps.

## Step 1

Let

$$H_1^2 + A\Sigma_1 + \Sigma_1 A^T = 0. \tag{5.48}$$

Define $A_1 = (Q_3Q_2Q_1)A(Q_3Q_2Q_1)^{-1}$, where

$$Q_1 = \begin{pmatrix} 0 & 0 & 0 & 0 & 1 & 0 \\ 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}, Q_2 = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 1 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}, Q_3 = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & \frac{a_{32}}{P_1} & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}.$$

Then it is straightforward to obtain that

$$A_1 = \begin{pmatrix} -\rho_1 & 0 & 0 & 0 & 0 & 0 \\ -a_{15} & \iota_1 & \iota_2 & -a_{13} & 0 & -a_{16} \\ 0 & P_1 & \iota_3 & 0 & 0 & 0 \\ 0 & 0 & P_2 & -a_{33} & -a_{34} & 0 \\ 0 & 0 & P_3 & a_{34} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\rho_2 \end{pmatrix},$$

where

$$P_1 = a_{21} + a_{12} + a_{22} - a_{11} \neq 0, P_2 = \frac{a_{32}(-a_{12} - a_{22})}{P_1} + \frac{a_{33}a_{32}}{P_1} + a_{32}, P_3 = \iota_5 = \frac{-a_{34}a_{32}}{P_1},$$
$$\iota_1 = a_{12} - a_{11}, \iota_2 = -a_{12} + \frac{a_{13}a_{32}}{P_1}, \iota_3 = -a_{12} - a_{22}. \tag{5.49}$$

Based on the value of $P_2$, which is

**Case 1**

If $P_2 \neq 0$, Let $A_2 = Q_4 A_1 Q_4^{-1}$, where $Q_4$ has the following form

$$Q_4 = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & \frac{P_3}{P_2} & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}.$$

Then it is easy to obtain that

$$A_2 = \begin{pmatrix} -\rho_1 & 0 & 0 & 0 & 0 & 0 \\ -a_{15} & \iota_1 & \iota_2 & -a_{13} & 0 & -a_{16} \\ 0 & P_1 & \iota_3 & 0 & 0 & 0 \\ 0 & 0 & P_2 & \iota_4 & -a_{34} & 0 \\ 0 & 0 & 0 & P_4 & G & 0 \\ 0 & 0 & 0 & 0 & 0 & -\rho_2 \end{pmatrix},$$

where

$$P_4 = \frac{-a_{33}P_3}{P_2} + \frac{a_{34}P_3^2}{P_2^2} + a_{34}, \quad G = \frac{-a_{34}P_3}{P_2}. \tag{5.50}$$

Depending on the value of $P_4$, we examine the following two conditions

**Case 1-1**

If $P_4 \neq 0$, Let $A_3 = Q_5 A_2 Q_5^{-1}$, where $Q_5$ is given by

$$Q_5 = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & P_1P_2P_4 & P_1P_4(-a_{12}-a_{22}-a_{33}) & P_6 & P_7 & 0 \\ 0 & 0 & P_2P_4 & -a_{33}P_4 & P_5 & 0 \\ 0 & 0 & 0 & P_4 & G & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix},$$

where

$$P_5 = \frac{a_{34}a_{33}P_3}{P_2} - a_{34}^2, P_6 = P_4 a_{33}^2, \quad P_7 = P_4 a_{33} a_{34} - \frac{a_{34}P_3P_5}{P_2}. \tag{5.51}$$

Then

$$A_3 = \begin{pmatrix} -\rho_1 & 0 & 0 & 0 & 0 & 0 \\ -a_{15}P_1P_2P_4 & -o_1 & -o_2 & -o_3 & -o_4 & -a_{16}P_1P_3P_4 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\rho_2 \end{pmatrix},$$

where

$$\begin{aligned} &o_1 = -(\iota_1 + \iota_3 + \iota_4 + \iota_5), o_2 = P_4a_{34} - P_1\iota_1 + \iota_1\iota_3 + \iota_1\iota_4 + \iota_1\iota_5 + \iota_3\iota_4 + \iota_3\iota_5 + \iota_4\iota_5, \\ &o_3 = P_1\iota_1\iota_4 - \iota_1\iota_3\iota_4 - \iota_1\iota_3\iota_5 - \iota_1\iota_4\iota_5 - \iota_1\iota_4\iota_5 - P_4a_{34}\iota_1 - P_4a_{34}\iota_5 + P_1\iota_1\iota_5 - P_1P_2a_{13}, \\ &o_4 = \iota_1\iota_3\iota_4\iota_5 - P_1\iota_1\iota_4\iota_5 - P_1P_4\iota_2a_{34} + P_4a_{34}\iota_1\iota_5 + P_1P_2a_{13}\iota_5. \end{aligned} \tag{5.52}$$

Additionally, let $A_4 = Q_6A_3Q_6^{-1}$, where $Q_6$ is given by

$$Q_6 = \begin{pmatrix} -a_{15}P_1P_2P_4 & -o_1 & -o_2 & -o_3 & -o_4 & -a_{16}P_1P_2P_4 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}.$$

Through straightforward calculations, we obtain

$$A_4 = \begin{pmatrix} -\vartheta_1 & -\vartheta_2 & -\vartheta_3 & -\vartheta_4 & -\vartheta_5 & -a_{16}P_1P_2P_4(\rho_2 - \rho_1) \\ 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\rho_2 \end{pmatrix},$$

where

$$\vartheta_1 = \rho_1 + o_1, \vartheta_2 = o_1\rho_1 + o_2, \vartheta_3 = o_2\rho_1 + o_3, \vartheta_4 = o_3\rho_1 + o_4, \vartheta_5 = o_4\rho_1. \tag{5.53}$$

Moreover, Equation (5.48) can be reformulated into the following form

$$\begin{aligned} &(Q_6Q_5Q_4Q_3Q_2Q_1)H_1^2(Q_6Q_5Q_4Q_3Q_2Q_1)^T + A_4(Q_6Q_5Q_4Q_3Q_2Q_1)\Sigma_1(Q_6Q_5Q_4Q_3Q_2Q_1)^T \\ &+ (Q_6Q_5Q_4Q_3Q_2Q_1)\Sigma_1(Q_6Q_5Q_4Q_3Q_2Q_1)^TA_4^T = 0, \end{aligned}$$

that is,

$$H_0^2 + A_4\Sigma_{01} + \Sigma_{01}A_4^T = 0, \tag{5.54}$$

where $\Sigma_{01} = \Omega_1^{-2}(Q_6Q_5Q_4Q_3Q_2Q_1)\Sigma_1(Q_6Q_5Q_4Q_3Q_2Q_1)^T$ and $\Omega_1 = -a_{15}P_1P_2P_4\sigma_1$. Based on Lemma 5.5, we can conclude that $\Sigma_{01}$ is given by the following form

$$\Sigma_{01} = \begin{pmatrix} \Theta_{11} & 0 & -\Theta_{22} & 0 & \Theta_{33} & 0 \\ 0 & \Theta_{22} & 0 & -\Theta_{33} & 0 & 0 \\ -\Theta_{22} & 0 & \Theta_{33} & 0 & -\Theta_{44} & 0 \\ 0 & -\Theta_{33} & 0 & \Theta_{44} & 0 & 0 \\ \Theta_{33} & 0 & -\Theta_{44} & 0 & \Theta_{55} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

where

$$\Theta_{11} = \frac{\vartheta_2(\vartheta_3\vartheta_4 - \vartheta_2\vartheta_5) - \vartheta_4(\vartheta_1\vartheta_4 - \vartheta_5)}{2[(\vartheta_1\vartheta_2 - \vartheta_3)(\vartheta_3\vartheta_4 - \vartheta_2\vartheta_5) - (\vartheta_1\vartheta_4 - \vartheta_5)^2]},$$
$$\Theta_{22} = \frac{\vartheta_3\vartheta_4 - \vartheta_2\vartheta_5}{2[(\vartheta_1\vartheta_2 - \vartheta_3)(\vartheta_3\vartheta_4 - \vartheta_2\vartheta_5) - (\vartheta_1\vartheta_4 - \vartheta_5)^2]},$$
$$\Theta_{33} = \frac{\vartheta_1\vartheta_4 - \vartheta_5}{2[(\vartheta_1\vartheta_2 - \vartheta_3)(\vartheta_3\vartheta_4 - \vartheta_2\vartheta_5) - (\vartheta_1\vartheta_4 - \vartheta_5)^2]},$$
$$\Theta_{44} = \frac{\vartheta_1\vartheta_2 - \vartheta_3}{2[(\vartheta_1\vartheta_2 - \vartheta_3)(\vartheta_3\vartheta_4 - \vartheta_2\vartheta_5) - (\vartheta_1\vartheta_4 - \vartheta_5)^2]},$$
$$\Theta_{55} = \frac{\vartheta_1(\vartheta_2\vartheta_3 - \vartheta_1\vartheta_4) - (\vartheta_3^2 - \vartheta_1\vartheta_5)}{2[(\vartheta_1\vartheta_2 - \vartheta_3)(\vartheta_3\vartheta_4 - \vartheta_2\vartheta_5) - (\vartheta_1\vartheta_4 - \vartheta_5)^2]}.$$

Then $\Sigma_1 = \Omega_1^2(Q_6Q_5Q_4Q_3Q_2Q_1)^{-1}\Sigma_{01}[(Q_6Q_5Q_4Q_3Q_2Q_1)^{-1}]^T$ and the leading principal submatrix of order five of $\Sigma_{01}$ is positive definite as demonstrated below

$$\Sigma_{01}^{(05)} = \begin{pmatrix} \Theta_{11} & 0 & -\Theta_{22} & 0 & \Theta_{33} \\ 0 & \Theta_{22} & 0 & -\Theta_{33} & 0 \\ -\Theta_{22} & 0 & \Theta_{33} & 0 & -\Theta_{44} \\ 0 & -\Theta_{33} & 0 & \Theta_{44} & 0 \\ \Theta_{33} & 0 & -\Theta_{44} & 0 & \Theta_{55} \end{pmatrix}.$$

**Case 1-2**

If $P_4 = 0$, Let $A_5 = (Q_8Q_7)A_2(Q_8Q_7)^{-1}$, where the standardized transformation matrices $Q_7$ and $Q_8$ are defined as follows

$$Q_7 = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & P_1P_2 & P_2(-a_{22}-a_{12}-a_{33}+\frac{a_{34}P_3}{P_2}) & (-a_{33}+\frac{a_{34}P_3}{P_2})^2 & a_{34}a_{33} & 0 \\ 0 & 0 & P_2 & -a_{33}+\frac{a_{34}P_3}{P_2} & -a_{34} & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}$$

and

$$Q_8 = \begin{pmatrix} -a_{15}P_1P_2 & -o_1 & -o_2 & -o_3 & -o_4 & -a_{16}P_1P_2 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}.$$

Direct calculation gives that

$$A_5 = \begin{pmatrix} -\bar{\vartheta}_1 & -\bar{\vartheta}_2 & -\bar{\vartheta}_3 & -\bar{\vartheta}_4 & -\bar{\vartheta}_5 & -a_{16}P_1P_2(\rho_2-\rho_1) \\ 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & G & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\rho_2 \end{pmatrix},$$

where

$$\bar{\vartheta}_1 = \rho_1 + o_1, \bar{\vartheta}_2 = o_1\rho_1 + o_2, \bar{\vartheta}_3 = o_2\rho_1 + o_3, \bar{\vartheta}_4 = o_3\rho_1, \bar{\vartheta}_5 = o_4\rho_1 - o_4G \quad with \quad (P_4 = 0). \tag{5.55}$$

Likewise, Equation (5.54) can be reformulated into the following equivalent expression

$$(Q_8Q_7Q_4Q_3Q_2Q_1)H_1^2(Q_8Q_7Q_4Q_3Q_2Q_1)^T + A_5(Q_8Q_7Q_4Q_3Q_2Q_1)\Sigma_1(Q_8Q_7Q_4Q_3Q_2Q_1)^T$$
$$+(Q_8Q_7Q_4Q_3Q_2Q_1)\Sigma_1(Q_8Q_7Q_4Q_3Q_2Q_1)^TA_5^T = 0.$$

**i.e.**,

$$H_0^2 + A_5\bar{\Sigma_{01}} + \bar{\Sigma_{01}}A_5^T = 0, \tag{5.56}$$

where $\bar{\Sigma_{01}} = \bar{\Omega_1}^{-2}(Q_8Q_7Q_4Q_3Q_2Q_1)\Sigma_1(Q_8Q_7Q_4Q_3Q_2Q_1)^T$ and $\bar{\Omega_1} = -a_{15}P_1P_2\sigma_1$. Referring to Lemma 5.6, we obtain that $\bar{\Sigma_{01}}$ takes the following form

$$\bar{\Sigma_{01}} = \begin{pmatrix} \bar{\Theta_{11}} & 0 & -\bar{\Theta_{22}} & 0 & 0 & 0 \\ 0 & \bar{\Theta_{22}} & 0 & -\bar{\Theta_{33}} & 0 & 0 \\ -\bar{\Theta_{22}} & 0 & \bar{\Theta_{33}} & 0 & 0 & 0 \\ 0 & -\bar{\Theta_{33}} & 0 & \bar{\Theta_{44}} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

where

$$\bar{\Theta_{11}} = \frac{\rho_1^2(o_1o_2 - o_3) + o_2(\rho_1o_2 + o_3)}{2(\rho_1^3 + \rho_1^2o_1 + \rho_1o_2 + o_3)(o_1o_2 - o_3)}, \bar{\Theta}_{22} = \frac{\rho_1o_2 + o_3}{2(\rho_1^3 + \rho_1^2o_1 + \rho_1o_2 + o_3)(o_1o_2 - o_3)},$$
$$\bar{\Theta_{33}} = \frac{\rho_1 + o_1}{2(\rho_1^3 + \rho_1^2o_1 + \rho_1o_2 + o_3)(o_1o_2 - o_3)}, \bar{\Theta_{44}} = \frac{\rho_1o_1(\rho_1 + o_1) + o_1o_2 - o_3}{2(\rho_1^3 + \rho_1^2o_1 + \rho_1o_2 + o_3)(o_1o_2 - o_3)}.$$

Then $\Sigma_1 = \bar{\Omega_1}^2(Q_8Q_7Q_4Q_3Q_2Q_1)^{-1}\bar{\Sigma_{01}}[(Q_8Q_7Q_4Q_3Q_2Q_1)^{-1}]^T$ and the fourth-order submatrix of $\bar{\Sigma_{01}}$ is positive definite as shown below

$$\bar{\Sigma_{01}}^{(04)} = \begin{pmatrix} \bar{\Theta_{11}} & 0 & -\bar{\Theta_{22}} & 0 \\ 0 & \bar{\Theta_{22}} & 0 & -\bar{\Theta_{33}} \\ -\bar{\Theta_{22}} & 0 & \bar{\Theta_{33}} & 0 \\ 0 & -\bar{\Theta_{33}} & 0 & \bar{\Theta_{44}} \end{pmatrix}.$$

**Case 2**

If $P_2 = 0$, Let $A_6 = Q_9A_1Q_9^{-1}$, where $Q_9$ has the following form

$$Q_9 = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix},$$

then we have

$$A_6 = \begin{pmatrix} -\rho_1 & 0 & 0 & 0 & 0 & 0 \\ -a_{15} & \iota_1 & \iota_2 & 0 & -a_{13} & -a_{16} \\ 0 & P_1 & \iota_3 & 0 & 0 & 0 \\ 0 & 0 & P_3 & 0 & a_{34} & 0 \\ 0 & 0 & 0 & -a_{34} & -a_{33} & 0 \\ 0 & 0 & 0 & 0 & 0 & -\rho_2 \end{pmatrix}.$$

Next, define $A_7 = (Q_{11}Q_{10})A_6(Q_{11}Q_{10})^{-1}$, where the standardized transformation matrices $Q_{10}$ and $Q_{11}$ are defined as follows

$$Q_{10} = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & -a_{34}P_1P_3 & a_{34}P_3(a_{33}-\iota_3) & -a_{33}(a_{33}^2-a_{34}^2) & a_{33}(2a_{34}^2-a_{33}^2) & 0 \\ 0 & 0 & -a_{34}P_3 & a_{33}P_3 & a_{33}^2-a_{34}^2 & 0 \\ 0 & 0 & 0 & -a_{34} & -a_{33} & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}$$

and

$$Q_{11} = \begin{pmatrix} a_{15}a_{34}P_1P_3 & -\bar{o_1} & -\bar{o_2} & -\bar{o_3} & -\bar{o_4} & a_{16}a_{34}P_1P_3 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix},$$

where

$$\begin{aligned} &\bar{o_1} = -(\iota_1+\iota_3+a_{33}), \bar{o_2} = \iota_1\iota_3 - \iota_1a_{33} - \iota_3a_{33} + a_{34}^2 - P_1\iota_2, \\ &\bar{o_3} = \iota_1\iota_3a_{33} - a_{34}^2\iota_1 - a_{34}^2\iota_3, \bar{o_4} = a_{34}^2\iota_1\iota_3. \end{aligned} \tag{5.57}$$

It can be readily shown that

$$A_7 = \begin{pmatrix} -\bar{\bar{\vartheta}}_1 & -\bar{\bar{\vartheta}}_2 & -\bar{\bar{\vartheta}}_3 & -\bar{\bar{\vartheta}}_4 & -\bar{\bar{\vartheta}}_5 & \iota_2(\rho_1-\rho_2) \\ 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\rho_2 \end{pmatrix},$$

where

$$\begin{aligned} &\bar{\bar{\vartheta}}_1 = \rho_1 + \bar{o_1}, \bar{\bar{\vartheta}}_2 = \bar{o_1}\rho_1 + \bar{o_2}, \bar{\bar{\vartheta}}_3 = \bar{o_2}\rho_1 + \bar{o_3}, \\ &\bar{\bar{\vartheta}}_4 = \bar{o_3}\rho_1 + \bar{o_4}, \bar{\bar{\vartheta}}_5 = \bar{o_4}\rho_1. \end{aligned} \tag{5.58}$$

Additionally, Equation (5.48) can be equivalently rewritten in the following form

$$\begin{aligned}&(Q_{11}Q_{10}Q_9Q_3Q_2Q_1)H_1^2(Q_{11}Q_{10}Q_9Q_3Q_2Q_1)^T\\&+A_7(Q_{11}Q_{10}Q_9Q_3Q_2Q_1)\Sigma_1(Q_{11}Q_{10}Q_9Q_3Q_2Q_1)^T\\&+(Q_{11}Q_{10}Q_9Q_3Q_2Q_1)\Sigma_1(Q_{11}Q_{10}Q_9Q_3Q_2Q_1)^TA_7^T=0.\end{aligned}$$

The preceding equation can be reformulated as the following equivalent expression

$$H_0^2+A_7\overline{\overline{\Sigma}}_{01}+\overline{\overline{\Sigma}}_{01}A_7^T=0, \tag{5.59}$$

where $\overline{\overline{\Sigma}}_{01}=\bar{\bar{\Omega}}_1^{-2}(Q_{11}Q_{10}Q_9Q_3Q_2Q_1)\Sigma_1(Q_{11}Q_{10}Q_9Q_3Q_2Q_1)^T$ and $\bar{\bar{\Omega}}_1=a_{15}a_{34}P_1P_3\sigma_1$. By applying Lemma 5.5, we derive that $\overline{\overline{\Sigma}}_{01}$ assumes the following form

$$\overline{\overline{\Sigma}}_{01}=\begin{pmatrix}\overline{\overline{\Theta}}_{11}&0&-\overline{\overline{\Theta}}_{22}&0&\overline{\overline{\Theta}}_{33}&0\\0&\overline{\overline{\Theta}}_{22}&0&-\overline{\overline{\Theta}}_{33}&0&0\\-\overline{\overline{\Theta}}_{22}&0&\overline{\overline{\Theta}}_{33}&0&-\overline{\overline{\Theta}}_{44}&0\\0&-\overline{\overline{\Theta}}_{33}&0&\overline{\overline{\Theta}}_{44}&0&0\\\bar{\Theta}_{33}&0&-\overline{\overline{\Theta}}_{44}&0&\overline{\overline{\Theta}}_{55}&0\\0&0&0&0&0&0\end{pmatrix},$$

where

$$\begin{aligned}\overline{\overline{\Theta}}_{11}&=\frac{\bar{\bar{\vartheta}}_2(\bar{\bar{\vartheta}}_3\bar{\bar{\vartheta}}_4-\bar{\bar{\vartheta}}_2\bar{\bar{\vartheta}}_5-\bar{\bar{\vartheta}}_4(\bar{\bar{\vartheta}}_1\bar{\bar{\vartheta}}_4-\bar{\bar{\vartheta}}_5))}{2[(\bar{\bar{\vartheta}}_1\bar{\bar{\vartheta}}_2-\bar{\bar{\vartheta}}_3)(\bar{\bar{\vartheta}}_3\bar{\bar{\vartheta}}_4-\bar{\bar{\vartheta}}_2\bar{\bar{\vartheta}}_5)-(\bar{\bar{\vartheta}}_1\bar{\bar{\vartheta}}_4-\bar{\bar{\vartheta}}_5)^2]},\\\overline{\overline{\Theta}}_{22}&=\frac{\bar{\bar{\vartheta}}_3\bar{\bar{\vartheta}}_4-\bar{\bar{\vartheta}}_2\bar{\bar{\vartheta}}_5}{2[(\bar{\bar{\vartheta}}_1\bar{\bar{\vartheta}}_2-\bar{\bar{\vartheta}}_3)(\bar{\bar{\vartheta}}_3\bar{\bar{\vartheta}}_4-\bar{\bar{\vartheta}}_2\bar{\bar{\vartheta}}_5)-(\bar{\bar{\vartheta}}_1\bar{\bar{\vartheta}}_4-\bar{\bar{\vartheta}}_5)^2]},\\\overline{\overline{\Theta}}_{33}&=\frac{\bar{\bar{\vartheta}}_1\bar{\bar{\vartheta}}_4-\bar{\bar{\vartheta}}_5}{2[(\bar{\bar{\vartheta}}_1\bar{\bar{\vartheta}}_2-\bar{\bar{\vartheta}}_3)(\bar{\bar{\vartheta}}_3\bar{\bar{\vartheta}}_4-\bar{\bar{\vartheta}}_2\bar{\bar{\vartheta}}_5)-(\bar{\bar{\vartheta}}_1\bar{\bar{\vartheta}}_4-\bar{\bar{\vartheta}}_5)^2]},\\\overline{\overline{\Theta}}_{44}&=\frac{\bar{\bar{\vartheta}}_1\bar{\bar{\vartheta}}_2-\bar{\bar{\vartheta}}_3}{2[(\bar{\bar{\vartheta}}_1\bar{\bar{\vartheta}}_2-\bar{\bar{\vartheta}}_3)(\bar{\bar{\vartheta}}_3\bar{\bar{\vartheta}}_4-\bar{\bar{\vartheta}}_2\bar{\bar{\vartheta}}_5)-(\bar{\bar{\vartheta}}_1\bar{\bar{\vartheta}}_4-\bar{\bar{\vartheta}}_5)^2]},\\\overline{\overline{\Theta}}_{55}&=\frac{\bar{\bar{\vartheta}}_1(\bar{\bar{\vartheta}}_2\bar{\bar{\vartheta}}_3-\bar{\bar{\vartheta}}_1\bar{\bar{\vartheta}}_4)-(\bar{\bar{\vartheta}}_3^2-\bar{\bar{\vartheta}}_1\bar{\bar{\vartheta}}_5)}{2[(\bar{\bar{\vartheta}}_1\bar{\bar{\vartheta}}_2-\bar{\bar{\vartheta}}_3)(\bar{\bar{\vartheta}}_3\bar{\bar{\vartheta}}_4-\bar{\bar{\vartheta}}_2\bar{\bar{\vartheta}}_5)-(\bar{\bar{\vartheta}}_1\bar{\bar{\vartheta}}_4-\bar{\bar{\vartheta}}_5)^2]}.\end{aligned}$$

Then $\Sigma_1=\bar{\bar{\Omega}}_1^2(Q_{11}Q_{10}Q_9Q_3Q_2Q_1)^{-1}\overline{\overline{\Sigma}}_{01}[(Q_{11}Q_{10}Q_9Q_3Q_2Q_1)^{-1}]^T$ and the leading principal submatrix of order five of $\overline{\overline{\Sigma}}_{01}$ is positive definite as demonstrated below

$$\overline{\overline{\Sigma}}_{01}^{\mp05)}=\begin{pmatrix}\overline{\overline{\Theta}}_{11}&0&-\overline{\overline{\Theta}}_{22}&0&\overline{\overline{\Theta}}_{33}\\0&\overline{\overline{\Theta}}_{22}&0&-\overline{\overline{\Theta}}_{33}&0\\-\overline{\overline{\Theta}}_{22}&0&\overline{\overline{\Theta}}_{33}&0&-\overline{\overline{\Theta}}_{44}\\0&-\overline{\overline{\Theta}}_{33}&0&\overline{\overline{\Theta}}_{44}&0\\\bar{\Theta}_{33}&0&-\overline{\overline{\Theta}}_{44}&0&\overline{\overline{\Theta}}_{55}\end{pmatrix}.$$

## Step 2

Consider the algebraic equation

$$H_2^2 + A\Sigma_2 + \Sigma_2 A^T = 0. \tag{5.60}$$

Following the procedure outlined in Step 1, we define $A_8 = (Q_{14}Q_{13}Q_{12})A(Q_{14}Q_{13}Q_{12})^{-1}$, where

$$Q_{12} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 1 \\ 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \end{pmatrix}, Q_{13} = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 1 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}, Q_{14} = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & \frac{a_{32}}{P_1} & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}.$$

Then we compute that

$$A_8 = \begin{pmatrix} -\rho_2 & 0 & 0 & 0 & 0 & 0 \\ -a_{16} & \iota_1 & \iota_2 & -a_{13} & 0 & -a_{15} \\ 0 & P_1 & \iota_3 & 0 & 0 & 0 \\ 0 & 0 & P_2 & -a_{33} & -a_{34} & 0 \\ 0 & 0 & P_3 & a_{34} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\rho_1 \end{pmatrix}.$$

Given the value of $P_2$, we will examine the following two cases:

### Case 1

If $P_2 \neq 0$, Let $A_9 = Q_{15}A_8Q_{15}^{-1}$, where

$$Q_{15} = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & \frac{P_3}{P_2} & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}.$$

Next, we calculate that

$$A_9 = \begin{pmatrix} -\rho_2 & 0 & 0 & 0 & 0 & 0 \\ -a_{16} & \iota_1 & \iota_2 & -a_{13} & 0 & -a_{15} \\ 0 & P_1 & \iota_3 & 0 & 0 & 0 \\ 0 & 0 & P_2 & \iota_4 & -a_{34} & 0 \\ 0 & 0 & 0 & P_4 & G & 0 \\ 0 & 0 & 0 & 0 & 0 & -\rho_1 \end{pmatrix},$$

and so

**Case 1-1**

If $P_4 \neq 0$, Let $A_{10} = (Q_{17}Q_{16})A_9(Q_{17}Q_{16})^{-1}$, where the standardized transformation matrices $Q_{16}$ and $Q_{17}$ are defined as follows

$$Q_{16} = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & P_1P_2P_4 & P_1P_4(-a_{12}-a_{22}-a_{33}) & P_6 & P_7 & 0 \\ 0 & 0 & P_2P_4 & -a_{33}P_4 & P_5 & 0 \\ 0 & 0 & 0 & P_4 & G & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}$$

and

$$Q_{17} = \begin{pmatrix} -a_{16}P_1P_2P_4 & -o_1 & -o_2 & -o_3 & -o_4 & -a_{15}P_1P_2P_4 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}.$$

Then it becomes clear that

$$A_{10} = \begin{pmatrix} -\chi_1 & -\chi_2 & -\chi_3 & -\chi_4 & -\chi_5 & -a_{15}P_1P_2P_4(\rho_1-\rho_2) \\ 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\rho_1 \end{pmatrix},$$

where

$$\chi_1 = \rho_2 + o_1, \chi_2 = o_1\rho_2 + o_2, \chi_3 = o_2\rho_2 + o_3, \chi_4 = o_3\rho_2 + o_4, \chi_5 = o_4\rho_2. \tag{5.61}$$

Thus, Equation (5.60) can be transformed into the following form

$$\begin{aligned}&(Q_{17}Q_{16}Q_{15}Q_{14}Q_{13}Q_{12})H_2^2(Q_{17}Q_{16}Q_{15}Q_{14}Q_{13}Q_{12})^T\\&+A_{10}(Q_{17}Q_{16}Q_{15}Q_{14}Q_{13}Q_{12})\Sigma_2(Q_{17}Q_{16}Q_{15}Q_{14}Q_{13}Q_{12})^T\\&+(Q_{17}Q_{16}Q_{15}Q_{14}Q_{13}Q_{12})\Sigma_2(Q_{17}Q_{16}Q_{15}Q_{14}Q_{13}Q_{12})^TA_{10}^T=0,\end{aligned}$$

that is

$$H_0^2+A_{10}\Sigma_{02}+\Sigma_{02}A_{10}^T=0, \tag{5.62}$$

where $\Sigma_{02}=\Omega_2^{-2}(Q_{17}Q_{16}Q_{15}Q_{14}Q_{13}Q_{12})\Sigma_2(Q_{17}Q_{16}Q_{15}Q_{14}Q_{13}Q_{12})^T$ and $\Omega_2=-a_{16}P_1P_2P_4\sigma_2$. By applying Lemma 5.5, we derive that $\Sigma_{02}$ assumes the following form

$$\Sigma_{02}=\begin{pmatrix}\Xi_{11}&0&-\Xi_{22}&0&\Xi_{33}&0\\0&\Xi_{22}&0&-\Xi_{33}&0&0\\-\Xi_{22}&0&\Xi_{33}&0&-\Xi_{44}&0\\0&-\Xi_{33}&0&\Xi_{44}&0&0\\\Xi_{33}&0&-\Xi_{44}&0&\Xi_{55}&0\\0&0&0&0&0&0\end{pmatrix},$$

where

$$\begin{aligned}\Xi_{11}&=\frac{\chi_2(\chi_3\chi_4-\chi_2\chi_5)-\chi_4(\chi_1\chi_4-\chi_5)}{2[(\chi_1\chi_2-\chi_3)(\chi_3\chi_4-\chi_2\chi_5)-(\chi_1\chi_4-\chi_5)^2]},\\\Xi_{22}&=\frac{\chi_3\chi_4-\chi_2\chi_5}{2[(\chi_1\chi_2-\chi_3)(\chi_3\chi_4-\chi_2\chi_5)-(\chi_1\chi_4-\chi_5)^2]}\\,\Xi_{33}&=\frac{\chi_1\chi_4-\chi_5}{2[(\chi_1\chi_2-\chi_3)(\chi_3\chi_4-\chi_2\chi_5)-(\chi_1\chi_4-\chi_5)^2]},\\\Xi_{44}&=\frac{\chi_1\chi_2-\chi_3}{2[(\chi_1\chi_2-\chi_3)(\chi_3\chi_4-\chi_2\chi_5)-(\chi_1\chi_4-\chi_5)^2]},\\\Xi_{55}&=\frac{\chi_1(\chi_2\chi_3-\chi_1\chi_4)-(\chi_3^2-\chi_1\chi_5)}{2[(\chi_1\chi_2-\chi_3)(\chi_3\chi_4-\chi_2\chi_5)-(\chi_1\chi_4-\chi_5)^2]}.\end{aligned}$$

Then $\Sigma_2=\Omega_2^2(Q_{17}Q_{16}Q_{15}Q_{14}Q_{13}Q_{12})^{-1}\Sigma_{02}[(Q_{17}Q_{16}Q_{15}Q_{14}Q_{13}Q_{12})^{-1}]^T$ and the leading principal submatrix of order five of $\Sigma_{02}$ is positive definite as demonstrated below

$$\Sigma_{02}^{(05)}=\begin{pmatrix}\Xi_{11}&0&-\Xi_{22}&0&\Xi_{33}\\0&\Xi_{22}&0&-\Xi_{33}&0\\-\Theta_{22}&0&\Theta_{33}&0&-\Theta_{44}\\0&-\Xi_{33}&0&\Xi_{44}&0\\\Xi_{33}&0&-\Xi_{44}&0&\Xi_{55}\end{pmatrix}.$$

**Case 1-2**

If $P_4 = 0$, Let $A_{11} = (Q_{19}Q_{18})A_9(Q_{19}Q_{18})^{-1}$, where the standardized transformation matrices $Q_{18}$ and $Q_{19}$ are defined as follows

$$Q_{18} = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & P_1P_2 & P_2(-a_{22} - a_{12} - a_{33} + \frac{a_{34}P_3}{P_2}) & (-a_{33} + \frac{a_{34}P_3}{P_2})^2 & a_{34}a_{33} & 0 \\ 0 & 0 & P_2 & -a_{33} + \frac{a_{34}P_3}{P_2} & -a_{34} & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}$$

and

$$Q_{19} = \begin{pmatrix} -a_{16}P_1P_2 & -\bar{o}_1 & -\bar{o}_2 & -\bar{o}_3 & -\bar{o}_4 & -a_{15}P_1P_2 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}.$$

Direct calculation gives that

$$A_{11} = \begin{pmatrix} -\bar{\chi_1} & -\bar{\chi_2} & -\bar{\chi_3} & -\bar{\chi_4} & -\bar{\chi_5} & -a_{15}P_1P_2(\rho_1 - \rho_2) \\ 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & G & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\rho_1 \end{pmatrix},$$

where

$$\bar{\chi_1} = \rho_2 + o_1, \bar{\chi_2} = o_1\rho_2 + o_2, \bar{\chi_3} = o_2\rho_2 + o_3, \bar{\chi_4} = o_3\rho_2, \bar{\chi_5} = o_4\rho_2 - o_4G \quad with \quad (P_4 = 0). \tag{5.63}$$

Likewise, Equation (5.54) can be reformulated into the following equivalent expression

$$\begin{aligned} &(Q_{19}Q_{18}Q_9Q_8Q_7Q_6)H_2^2(Q_{19}Q_{18}Q_9Q_8Q_7Q_6)^T \\ &+A_{11}(Q_{19}Q_{18}Q_9Q_8Q_7Q_6)\Sigma_2(Q_{19}Q_{18}Q_9Q_8Q_7Q_6)^T \\ &+(Q_{19}Q_{18}Q_9Q_8Q_7Q_6)\Sigma_2(Q_{19}Q_{18}Q_9Q_8Q_7Q_6)^TA_{11}^T = 0. \end{aligned}$$

**i.e.**,

$$H_0^2 + A_{11}\bar{\Sigma_{02}} + \bar{\Sigma_{02}}A_{11}^T = 0, \tag{5.64}$$

where $\bar{\Sigma_{02}} = \bar{\Omega_2}^{-2}(Q_{19}Q_{18}Q_9Q_8Q_7Q_6)\Sigma_2(Q_{19}Q_{18}Q_9Q_8Q_7Q_6)^T$ and $\bar{\Omega_2} = -a_{15}P_1P_2\sigma_2$. Based on Lemma 5.6, we can conclude that $\bar{\Sigma_{02}}$ is given by the following form

$$\bar{\Sigma_{02}} = \begin{pmatrix} \bar{\Xi_{11}} & 0 & -\bar{\Xi_{22}} & 0 & 0 & 0 \\ 0 & \bar{\Xi_{22}} & 0 & -\bar{\Xi_{33}} & 0 & 0 \\ -\bar{\Xi_{22}} & 0 & \bar{\Xi_{33}} & 0 & 0 & 0 \\ 0 & -\bar{\Xi_{33}} & 0 & \bar{\Xi_{44}} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

where

$$\bar{\Theta_{11}} = \frac{\rho_1^2(o_1o_2 - o_3) + o_2(\rho_1o_2 + o_3)}{2(\rho_1^3 + \rho_1^2o_1 + \rho_1o_2 + o_3)(o_1o_2 - o_3)}, \bar{\Theta}_{22} = \frac{\rho_1o_2 + o_3}{2(\rho_1^3 + \rho_1^2o_1 + \rho_1o_2 + o_3)(o_1o_2 - o_3)},$$
$$\bar{\Theta_{33}} = \frac{\rho_1 + o_1}{2(\rho_1^3 + \rho_1^2o_1 + \rho_1o_2 + o_3)(o_1o_2 - o_3)}, \bar{\Theta_{44}} = \frac{\rho_1o_1(\rho_1 + o_1) + o_1o_2 - o_3}{2(\rho_1^3 + \rho_1^2o_1 + \rho_1o_2 + o_3)(o_1o_2 - o_3)}.$$

Then $\Sigma_2 = \bar{\Omega_2}^2(Q_{19}Q_{18}Q_9Q_8Q_7Q_6)^{-1}\bar{\Sigma_{02}}[(Q_{19}Q_{18}Q_9Q_8Q_7Q_6)^{-1}]^T$ and the fourth-order submatrix of $\bar{\Sigma_{02}}$ is positive definite as shown below

$$\bar{\Sigma_{02}}^{(04)} = \begin{pmatrix} \bar{\Xi_{11}} & 0 & -\bar{\Xi_{22}} & 0 \\ 0 & \bar{\Xi_{22}} & 0 & -\bar{\Xi_{33}} \\ -\bar{\Xi_{22}} & 0 & \bar{\Xi_{33}} & 0 \\ 0 & -\bar{\Xi_{33}} & 0 & \bar{\Xi_{44}} \end{pmatrix}.$$

**Case 2**

If $P_2 = 0$, Let $A_{12} = (Q_{22}Q_{21}Q_{20})A_8(Q_{22}Q_{21}Q_{20})^{-1}$, where the standardized transformation matrices $Q_{20}$, $Q_{20}$, and $Q_{20}$ are given by

$$Q_{20} = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix},$$

$$Q_{21} = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & -a_{34}P_1P_3 & a_{34}P_3(a_{33}-\iota_3) & -a_{33}(a_{33}^2-a_{34}^2) & a_{33}(2a_{34}^2-a_{33}^2) & 0 \\ 0 & 0 & -a_{34}P_3 & a_{33}P_3 & a_{33}^2-a_{34}^2 & 0 \\ 0 & 0 & 0 & -a_{34} & -a_{33} & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}$$

and

$$Q_{22} = \begin{pmatrix} a_{16}a_{34}P_1P_3 & -\bar{o_1} & -\bar{o_2} & -\bar{o_3} & -\bar{o_4} & a_{15}a_{34}P_1P_3 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}.$$

Then

$$A_{12} = \begin{pmatrix} -\bar{\bar{\chi}}_1 & -\bar{\bar{\chi}}_2 & -\bar{\bar{\chi}}_3 & -\bar{\bar{\chi}}_4 & -\bar{\bar{\chi}}_5 & \iota_2(\rho_2-\rho_1) \\ 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\rho_1 \end{pmatrix},$$

where

$$\bar{\bar{\chi}}_1 = \rho_2 + \bar{o_1}, \bar{\bar{\chi}}_2 = \bar{o_1}\rho_2 + \bar{o_2}, \bar{\bar{\chi}}_3 = \bar{o_2}\rho_2 + \bar{o_3}, \bar{\bar{\chi}}_3 = \bar{o_3}\rho_2 + \bar{o_4}, \bar{\bar{\chi}}_4 = \bar{o_4}\rho_2. \tag{5.65}$$

Additionally, Equation (5.60) can be equivalently rewritten in the following form

$$\begin{aligned} &(Q_{22}Q_{21}Q_{20}Q_{14}Q_{13}Q_{12})H_2^2(Q_{22}Q_{21}Q_{20}Q_{14}Q_{13}Q_{12})^T \\ &+ A_{12}(Q_{22}Q_{21}Q_{20}Q_{14}Q_{13}Q_{12})\Sigma_2(Q_{22}Q_{21}Q_{20}Q_{14}Q_{13}Q_{12})^T \\ &+ (Q_{22}Q_{21}Q_{20}Q_{14}Q_{13}Q_{12})\Sigma_2(Q_{22}Q_{21}Q_{20}Q_{14}Q_{13}Q_{12})^T A_{12}^T = 0. \end{aligned}$$

which implies that

$$H_0^2 + A_{12}\bar{\bar{\Sigma}}_{02} + \bar{\bar{\Sigma}}_{02}A_{12}^T = 0, \tag{5.66}$$

where $\bar{\bar{\Sigma}}_{02} = \bar{\bar{\Omega}}_2^{-2}(Q_{22}Q_{21}Q_{20}Q_{14}Q_{13}Q_{12})\Sigma_2(Q_{22}Q_{21}Q_{20}Q_{14}Q_{13}Q_{12})^T$
and $\bar{\bar{\Omega}}_2 = a_{16}a_{34}P_1P_3\sigma_2$. Referring to Lemma 5.5, we obtain that $\bar{\bar{\Sigma}}_{02}$ takes the fol-

lowing form:

$$\Sigma_{02}^{=}=\begin{pmatrix} \Xi_{11}^{=} & 0 & -\Xi_{22}^{=} & 0 & \Xi_{33}^{=} & 0 \\ 0 & \Xi_{22}^{=} & 0 & -\Xi_{33}^{=} & 0 & 0 \\ -\Xi_{22}^{=} & 0 & \Xi_{33}^{=} & 0 & -\Xi_{44}^{=} & 0 \\ 0 & -\Xi_{33}^{=} & 0 & \Xi_{44}^{=} & 0 & 0 \\ \Xi_{33}^{-} & 0 & -\Xi_{44}^{=} & 0 & \Xi_{55}^{=} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

where

$$\begin{aligned}
\Xi_{11}^{=} &= \frac{\bar{\bar{\chi}}_2(\bar{\bar{\chi}}_3\bar{\bar{\chi}}_4-\bar{\bar{\chi}}_2\bar{\bar{\chi}}_5)-\bar{\bar{\chi}}_4(\bar{\bar{\chi}}_1\bar{\bar{\chi}}_4-\bar{\bar{\chi}}_5)}{2[(\bar{\bar{\chi}}_1\bar{\bar{\chi}}_2-\bar{\bar{\chi}}_3)(\bar{\bar{\chi}}_3\bar{\bar{\chi}}_4-\bar{\bar{\chi}}_2\bar{\bar{\chi}}_5)-(\bar{\bar{\chi}}_1\bar{\bar{\chi}}_4-\bar{\bar{\chi}}_5)^2]},\\
\Xi_{22}^{=} &= \frac{\bar{\bar{\chi}}_3\bar{\bar{\chi}}_4-\bar{\bar{\chi}}_2\bar{\bar{\chi}}_5}{2[(\bar{\bar{\chi}}_1\bar{\bar{\chi}}_2-\bar{\bar{\chi}}_3)(\bar{\bar{\chi}}_3\bar{\bar{\chi}}_4-\bar{\bar{\chi}}_2\bar{\bar{\chi}}_5)-(\bar{\bar{\chi}}_1\bar{\bar{\chi}}_4-\bar{\bar{\chi}}_5)^2]},\\
\Xi_{33}^{=} &= \frac{\bar{\bar{\chi}}_1\bar{\bar{\chi}}_4-\bar{\bar{\chi}}_5}{2[(\bar{\bar{\chi}}_1\bar{\bar{\chi}}_2-\bar{\bar{\chi}}_3)(\bar{\bar{\chi}}_3\bar{\bar{\chi}}_4-\bar{\bar{\chi}}_2\bar{\bar{\chi}}_5)-(\bar{\bar{\chi}}_1\bar{\bar{\chi}}_4-\bar{\bar{\chi}}_5)^2]},\\
\Xi_{44}^{=} &= \frac{\bar{\bar{\chi}}_1\bar{\bar{\chi}}_2-\bar{\bar{\chi}}_3}{2[(\bar{\bar{\chi}}_1\bar{\bar{\chi}}_2-\bar{\bar{\chi}}_3)(\bar{\bar{\chi}}_3\bar{\bar{\chi}}_4-\bar{\bar{\chi}}_2\bar{\bar{\chi}}_5)-(\bar{\bar{\chi}}_1\bar{\bar{\chi}}_4-\bar{\bar{\chi}}_5)^2]},\\
\Xi_{55}^{=} &= \frac{\bar{\bar{\chi}}_1(\bar{\bar{\chi}}_2\bar{\bar{\chi}}_3-\bar{\bar{\chi}}_1\bar{\bar{\chi}}_4)-(\bar{\bar{\chi}}_3^2-\bar{\bar{\chi}}_1\bar{\bar{\chi}}_5)}{2[(\bar{\bar{\chi}}_1\bar{\bar{\chi}}_2-\bar{\bar{\chi}}_3)(\bar{\bar{\chi}}_3\bar{\bar{\chi}}_4-\bar{\bar{\chi}}_2\bar{\bar{\chi}}_5)-(\bar{\bar{\chi}}_1\bar{\bar{\chi}}_4-\bar{\bar{\chi}}_5)^2]}.
\end{aligned}$$

Then $\Sigma_2=\bar{\bar{\Omega}}_2^2(Q_{22}Q_{21}Q_{20}Q_{14}Q_{13}Q_{12})^{-1}\Sigma_{02}^{=}[(Q_{22}Q_{21}Q_{20}Q_{14}Q_{13}Q_{12})^{-1}]^T$ and the leading principal submatrix of order five of $\Sigma_{02}^{=}$ is positive definite as demonstrated below

$$\Sigma_{02}^{\bar{\bar{\tau}}(05)}=\begin{pmatrix} \Xi_{11}^{=} & 0 & -\Xi_{22}^{=} & 0 & \Xi_{33}^{=} \\ 0 & \Xi_{22}^{=} & 0 & -\Xi_{33}^{=} & 0 \\ -\Xi_{22}^{=} & 0 & \Xi_{33}^{=} & 0 & -\Xi_{44}^{=} \\ 0 & -\Xi_{33}^{=} & 0 & \Xi_{44}^{=} & 0 \\ \Xi_{33}^{-} & 0 & -\Xi_{44}^{=} & 0 & \Xi_{55}^{=} \end{pmatrix}.$$

Then $\Sigma=\Sigma_1+\Sigma_2$ is positive define.Thus, the expression for a normal density function around the quasi-endemic equilibrium of model (5.5) is derived by

$$\mathcal{J}(x,y,z,v,\varphi,\phi)=(2\pi)^{-3}|\Sigma|^{-1/2}e^{-\frac{1}{2}(x-x^+,y-y^+,v-v^+,z-z^+,\varphi-\varphi^+,\phi-\phi^+)\Sigma^{-1}(x-x^+,y-y^+,v-v^+,z-z^+,\varphi-\varphi^+,\phi-\phi^+)^T}.$$

This completes the proof. □

## 5.5 Extinction

This section focuses on analyzing the conditions sufficient for the long-term extinction of the disease within system (5.5) by utilizing the concept of the basic repro-

duction number $\mathcal{R}_0$. Initially, a critical threshold $\mathcal{R}_0^{ex}$ is defined to delineate the conditions for disease extinction

$$\mathcal{R}_0^{ex} = \mathcal{R}_0 + \frac{\mathcal{R}_0 u d}{\min_{1\le i\le 2}\left\{\frac{\bar{\beta}_i}{\zeta_i}\right\}\mu_1}(e^{\frac{\sigma_1^2}{\rho_1}} - 2e^{\frac{\sigma_1^2}{4\rho_1}} + 1)^{\frac{1}{2}} + \frac{\bar{\beta}_2\Lambda}{\min_{1\le i\le 2}\left\{\frac{\bar{\beta}_i}{\zeta_i}\right\}\mu_1}(e^{\frac{\sigma_2^2}{\rho_2}} - 2e^{\frac{\sigma_2^2}{4\rho_2}} + 1)^{\frac{1}{2}},$$

where $\zeta_1 = \dfrac{\bar{\beta}_1}{u}, \zeta_2 = \dfrac{\bar{\beta}_1 k + \bar{\beta}_2 u}{u(a+\alpha)}$.

**Theorem 5.7.**

*If $\mathcal{R}_0^{ex} < 1$, then HIV–1 within system (5.5) with $x(0) < x_0$ will experience exponential decay with a probability of 1. To be more precise*

$$\limsup_{t\to\infty} \frac{\ln(\frac{\mathcal{R}_0}{x_0}y + \frac{\bar{\beta}_1}{u}v)}{t} \le \min_{1\le i\le 2}\left\{\frac{\bar{\beta}_i}{\zeta_i}\right\}(\mathcal{R}_0^{ex} - 1) < 0 \quad a.s. \tag{5.67}$$

**Proof.** Employing Itô's formula on the natural logarithm of $\mathsf{F}(t)$, one obtains

$$\begin{aligned}\frac{d\ln\mathsf{F}(t)}{dt} &= \frac{1}{\mathsf{F}}\left\{\zeta_1[(\bar{\beta}_1 e^{\varphi}v + \bar{\beta}_2 e^{\Phi}y)x - (a+\alpha)y] + \zeta_2[ky - (u+pz)v]\right\}\\ &\le \frac{1}{\mathsf{F}}\Big\{\zeta_1(\bar{\beta}_1 v + \bar{\beta}_2 y)x - [\zeta_1(a+\alpha) - \zeta_2 k]y - [\zeta_2 u]v\\ &\quad + \zeta_1[(\bar{\beta}_1 e^{\varphi}v + \bar{\beta}_2 e^{\Phi}y)x - \zeta_1[(\bar{\beta}_1 v + \bar{\beta}_2 y)x\Big\}.\end{aligned} \tag{5.68}$$

Employing the formulas for $\zeta_i$ (where $i = 1, 2$ ), it is straightforward to verify that they fulfill the given equalities

$$\begin{cases}\zeta_1(a+\alpha) - \zeta_2 k = \bar{\beta}_2,\\ \zeta_2 u = \bar{\beta}_1.\end{cases} \tag{5.69}$$

Using the first equation of system (5.5), we find that $dx(t) \le [x_0 - x(t)]dt$. Considering $x(0) < x_0$, it follows that $x(t) < x_0$ for any $t \ge 0$. If $\mathcal{R}_0^{ex} < 1$, we obtain $\mathcal{R}_0 < 1$. Consolidating equations (5.68) and (5.69) leads to

$$\begin{aligned}\frac{d\ln\mathsf{F}(t)}{dt} &\le \frac{1}{\mathsf{F}}\left[(\frac{\mathcal{R}_0 x}{x_0} - 1)(\bar{\beta}_1 v + \bar{\beta}_2 y)\right] + \frac{\zeta_1\frac{\bar{\beta}_1\Lambda}{\mu_1}v(e^{\varphi} - 1) + \zeta_1\frac{\bar{\beta}_2\Lambda}{\mu_1}y(e^{\Phi} - 1)}{\mathsf{F}}\\ &\le \frac{(\bar{\beta}_1 v + \bar{\beta}_2 y)(\mathcal{R}_0 - 1)}{\zeta_1 y + \zeta_2 v} + \frac{\zeta_1\frac{\bar{\beta}_1\Lambda}{\mu_1}v|e^{\varphi} - 1| + \zeta_1\frac{\bar{\beta}_2\Lambda}{\mu_1}y|e^{\Phi} - 1|}{\zeta_1 y + \zeta_2 v}\\ &\le \min_{1\le i\le 2}\left\{\frac{\bar{\beta}_i}{\zeta_i}\right\}(\mathcal{R}_0 - 1) + \frac{\mathcal{R}_0 u d}{\mu_1}|e^{\varphi} - 1| + \frac{\bar{\beta}_2\Lambda}{\mu_1}|e^{\Phi} - 1|,\end{aligned} \tag{5.70}$$

thus

$$\frac{\ln \digamma(t)}{t} - \frac{\ln \digamma(0)}{t} \leq \min_{1\leq i\leq 2}\left\{\frac{\bar{\beta}_i}{\zeta_i}\right\}(\mathcal{R}_0 - 1) + \frac{\mathcal{R}_0 u d}{\mu_1}\frac{1}{t}\int_0^t |e^{\varphi(s)} - 1|ds + \frac{\bar{\beta}_2\Lambda}{\mu_1}\frac{1}{t}\int_0^t |e^{\Phi(s)} - 1|ds, \tag{5.71}$$

by applying the ergodicity of $\varphi(t)$ and $\Phi(t)$ and considering the superior limit on both sides of equation (5.71), we derive the result

$$\begin{aligned}\limsup_{t\to\infty}\frac{\ln \digamma(t)}{t} &\leq \min_{1\leq i\leq 2}\left\{\frac{\bar{\beta}_i}{\zeta_i}\right\}(\mathcal{R}_0 - 1) + \frac{\mathcal{R}_0 u d}{\mu_1}\lim_{t\to\infty}\frac{1}{t}\int_0^t |e^{\varphi(s)} - 1|ds \\ &\quad + \frac{\bar{\beta}_2\Lambda}{\mu_1}\lim_{t\to\infty}\frac{1}{t}\int_0^t |e^{\Phi(s)} - 1|ds \\ &= \min_{1\leq i\leq 2}\left\{\frac{\bar{\beta}_i}{\zeta_i}\right\}(\mathcal{R}_0 - 1) + \frac{\mathcal{R}_0 u d}{\mu_1}\int_{\mathbb{R}}\left|e^{\tau} - 1\right|\pi_1(\tau)d\tau + \frac{\bar{\beta}_2\Lambda}{\mu_1}\int_{\mathbb{R}}\left|e^{\tau} - 1\right|\pi_2(\tau)d\tau.\end{aligned} \tag{5.72}$$

Utilizing equation (3.14) in conjunction with Hölder's inequality yields the following result

$$\int_{\mathbb{R}}\left|e^{\tau} - 1\right|\pi_i(\tau)d\tau \leq \left(\int_{\mathbb{R}}\left|e^{\tau} - 1\right|^2\pi_i(\tau)d\tau\right)^{\frac{1}{2}} = \left(e^{\frac{\sigma_i^2}{\rho_i}} - 2e^{\frac{\sigma_i^2}{4\rho_i}} + 1\right)^{\frac{1}{2}}. \tag{5.73}$$

Consequently, based on (5.72) and (5.73)

$$\begin{aligned}\limsup_{t\to\infty}\frac{\ln \digamma(t)}{t} &\leq \min_{1\leq i\leq 2}\left\{\frac{\bar{\beta}_i}{\zeta_i}\right\}(\mathcal{R}_0 - 1) + \frac{\mathcal{R}_0 u d}{\mu_1}\left(e^{\frac{\sigma_1^2}{\rho_1}} - 2e^{\frac{\sigma_1^2}{4\rho_1}} + 1\right)^{\frac{1}{2}} + \frac{\bar{\beta}_2\Lambda}{\mu_1}\left(e^{\frac{\sigma_2^2}{\rho_2}} - 2e^{\frac{\sigma_2^2}{4\rho_2}} + 1\right)^{\frac{1}{2}} \\ &= \min_{1\leq i\leq 2}\left\{\frac{\bar{\beta}_i}{\zeta_i}\right\}(\mathcal{R}_0^{ex} - 1).\end{aligned} \tag{5.74}$$

If $\mathcal{R}_0^{ex}$ is less than 1, we consequently have $\lim_{t\to\infty}\digamma(t) = 0$ almost surely, resulting in $\lim_{t\to\infty} y(t) = 0$ and $\lim_{t\to\infty} v(t) = 0$, a.s. Thus, the proof is concluded. □

## 5.6 Computational simulations

To validate our aforementioned theoretical findings through numerous numerical simulations, we must initially convert system (5.5) into a discrete representation. Employing the well-established Milstein's higher-order method [119], we derive the

discretized equation for system (5.5) as follows:

$$\begin{cases} x^{j+1} = x^j + [\Lambda - dx^j - \bar{\beta}_1 e^{\varphi^j} x^j v^j - \bar{\beta}_2 e^{\Phi^j} x^j y^j]\Delta t, \\ y^{j+1} = y^j + [\bar{\beta}_1 e^{\varphi^j} x^j v^j + \bar{\beta}_2 e^{\Phi^j} x^j y^j - a y^j - \alpha y^j]\Delta t, \\ v^{j+1} = v^j + [k y^j - u v^j - p v^j z^j]\Delta t, \\ z^{j+1} = z^j + [c v^j z^j - b z^j - \beta z^j]\Delta t, \\ \varphi^{j+1} = \varphi^j - \rho_1 \varphi^j \Delta t + \sigma_1 \sqrt{\Delta t}\varepsilon_1^j, \\ \Phi^{j+1} = \Phi^j - \rho_2 \Phi^j \Delta t + \sigma_2 \sqrt{\Delta t}\varepsilon_2^j. \end{cases} \tag{5.75}$$

Here, the time step is denoted by $\Delta t > 0$. $\varepsilon_i^j$, represents two independent random variables following the Gaussian distribution $N(0,1)$ for $i = 1,2$; $j = 1,2,...,n$. Furthermore, $(x^j, y^j, v^j, z^j, \varphi^j, \Phi^j)$ signifies the respective values of the jth iteration of the discretization equation mentioned above.

We select realistic parameter values sourced from published references and list all biological parameter values in Table 5.1. Subsequently, through numerical simulations, our primary focus is on verifying three key aspects:

1. The existence of a stationary distribution for system (5.5) under the condition $\mathcal{R}_0^x > 1$.
2. The existence of the probability density.
3. Assessing the impact of significant reversion speed or low volatility intensity on the quantities of infected cells and free viruses.

**Example 5.1.**

To numerically establish the existence of a stationary distribution, we set $u = 0.3, k = 0.9$, and $(\rho_1, \rho_2, \sigma_1, \sigma_2) = (0.1, 0.1, 0.2, 0.4)$, and the remaining parameter values are detailed in Table 5.1. Through direct computation, we find that

$$\mathcal{R}_0^s = \frac{\Lambda(\bar{\beta}_1 k e^{\frac{\sigma_1^2}{12\rho_1}} + \bar{\beta}_2 u e^{\frac{\sigma_2^2}{8\rho_2}})}{(a+\alpha)ud} \approx 9.543 > 1.$$

Consequently, satisfying the conditions of **??**, we conclude that system (5.5) possesses a stationary distribution $\Psi(.)$, indicating the sustained prevalence of infected cells and free viruses with certainty. This is illustrated in Figure 5.1 and Figure 5.2.

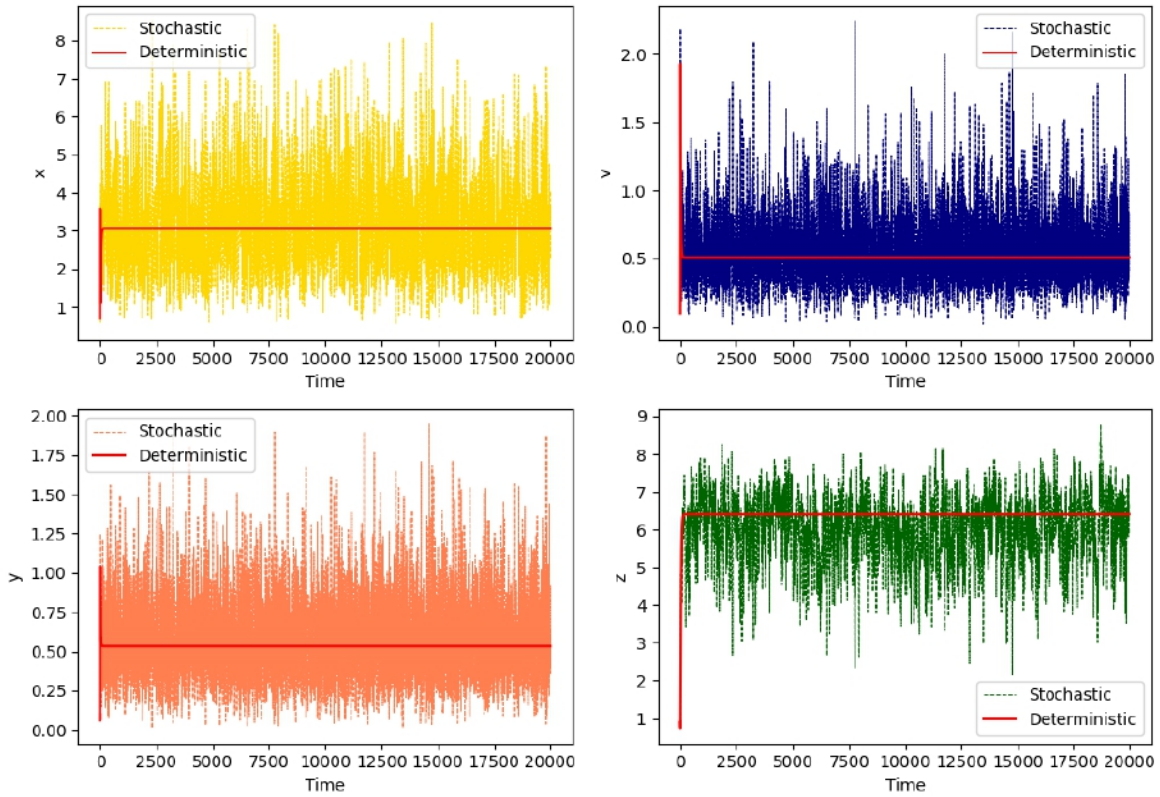



Figure 5.1: Depiction of Trajectories for Uninfected Target Cells $x$, Infected Cells $y$, Populations of Free Viruses $v$, and $B$ Cells $z$ for Both the Stochastic System (5.5) and the Corresponding Deterministic System (5.1), Utilizing Parameter Values $u = 0.3, k = 0.9$, and $(\rho_1, \rho_2, \sigma_1, \sigma_2) = (0.1, 0.1, 0.2, 0.4)$.

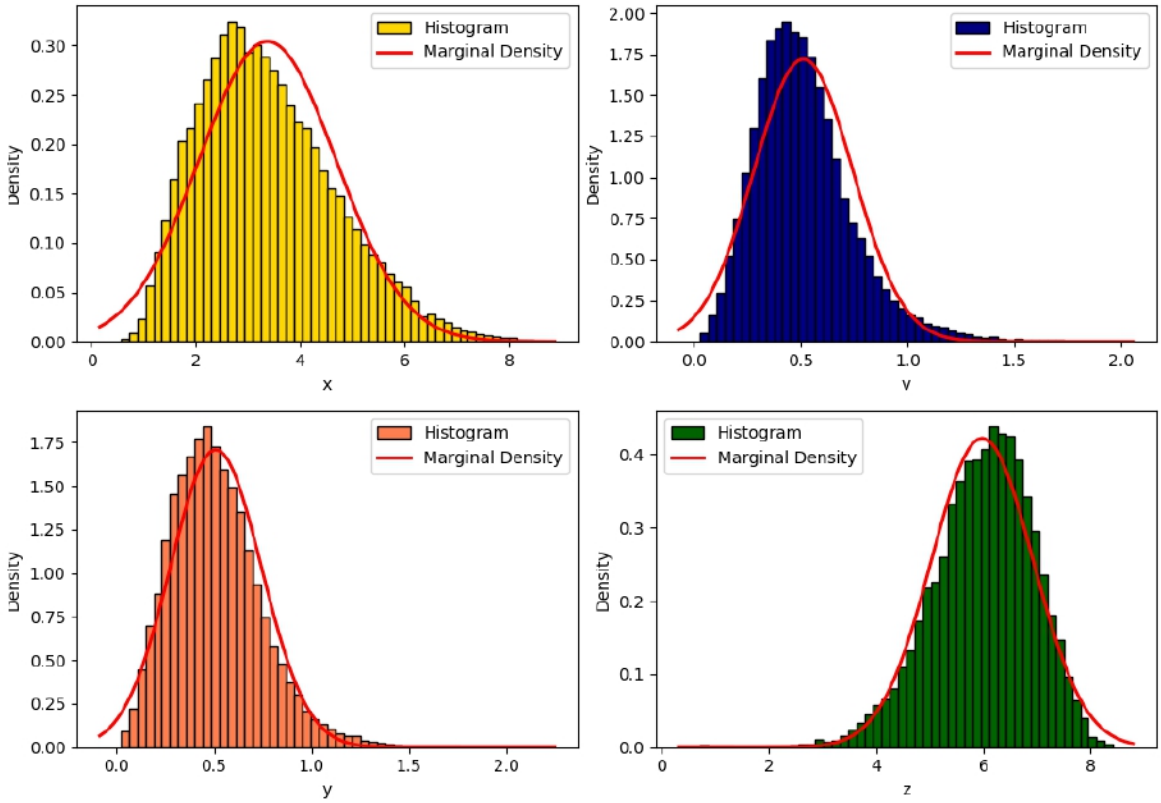



Figure 5.2: Frequency Histograms and Marginal Density Functions for Variables$x, y, v$, and $z$ within System (5.5).

Table 5.1: List of parameter values of system (5.5) .

| Parameters | Values | Source |
|---|---|---|
| $\Lambda$ | 1 | [161] |
| $d$ | 0.1 | Assumed |
| $\bar{\beta_1}$ | 0.4 | [161] |
| $\bar{\beta_2}$ | $4.7\times10^{-7}$ | [152] |
| $a$ | 0.5 | [161] |
| $p$ | 0.06 | [152] |
| $b$ | 0.05 | [161] |
| $c$ | 0.1 | [161] |
| $\beta$ | $10^{-3}$ | Assumed |
| $\alpha$ | 0.8 | Assumed |

**Example 5.2.**

To confirm the presence of the probability density around the quasi-chronic infection equilibrium, we set $u = 0.3, k = 0.9$, and $(\rho_1, \rho_2, \sigma_1, \sigma_2) = (0.1, 0.1, 0.2, 0.4)$, and the other parameters are listed in Table 5.1. Thus, we have $E^+ = (x^+, y^+, v^+, z^+, \varphi^+, \Phi^+)^T = (7.14244, 0.5392, 0.10002, 71.065)$ and

$$\mathcal{R}_0^s = \frac{\Lambda(\bar{\beta_1} ke^{\frac{\sigma_1^2}{12\rho_1}} + \bar{\beta_2} ue^{\frac{\sigma_2^2}{8\rho_2}})}{(a+\alpha)ud} \approx 9.543 > 1.$$

Therefore, the conditions of Theorem 5.3 are satisfied, indicating that system (5.5) possesses a Gaussian probability density near the quasi-chronic infection equilibrium $E^+$ . Furthermore, we find $P_4 = 0.06538640289179085 > 0$. Consequently, by the second case of Theorem 5.3, the specific form of the covariance matrix $\Sigma$,

$$\Sigma = \begin{pmatrix} 3633.1828 & -3790.6157 & 0.1997 & -0.0465 & -3.8278 & -33.9239 \\ -3790.6157 & 3954.8711 & -0.2084 & 0.0424 & 3.9978 & 35.4337 \\ 0.1997 & -0.2084 & 0.0000 & 0.0023 & -0.0012 & -0.0111 \\ -0.0465 & 0.0424 & 0.0023 & -0.5992 & -0.0091 & -0.0813 \\ -3.8278 & 3.9978 & -0.0012 & -0.0091 & 0.4039 & 0.0000 \\ -33.9239 & 35.4337 & -0.0111 & -0.0813 & 0.0000 & 0.4517 \end{pmatrix}$$

, and the marginal density functions of $x(t)$, $y(t)$, $v(t)$ and $z(t)$ can be obtained as

follows

$$\dot{L}_1(x) \sim \mathcal{N}(7.14244, 3633.1828), \dot{L}_2(y) \sim \mathcal{N}(0.5392, 3954.8711),$$
$$\dot{L}_3(v) \sim \mathcal{N}(0.10002, 0.0000), \dot{L}_4(z) \sim \mathcal{N}(71.065, -0.5992).$$

Figure 5.3 illustrates this.

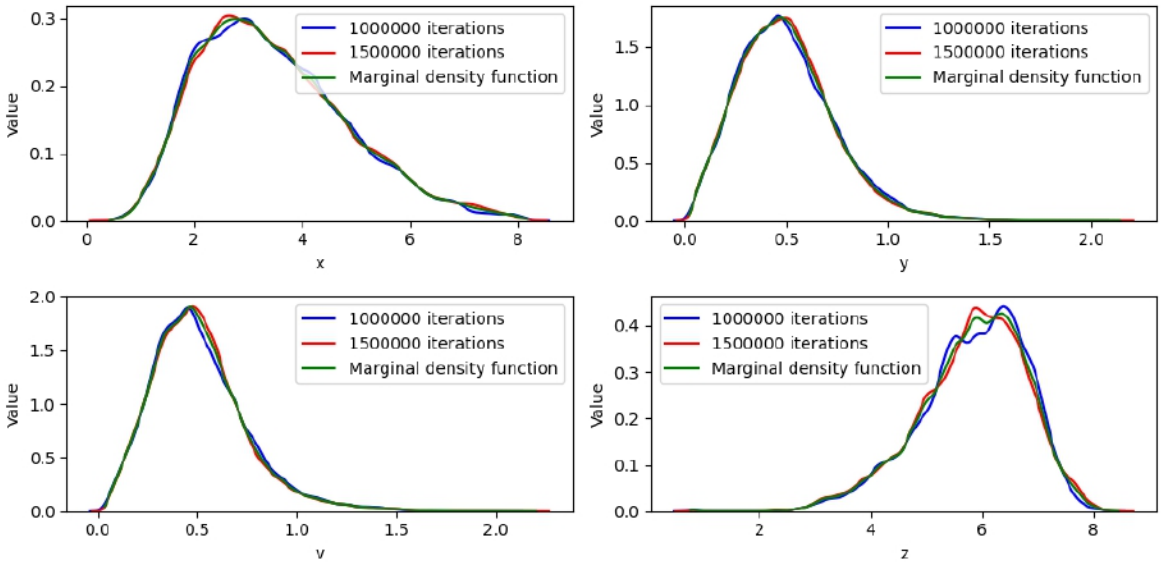



Figure 5.3: Numerical simulations for the frequency histogram fitting density curves of $x, y, v$ and $z$ of system (5.5) with 100,000 and 1,500,000 iteration points, respectively, and the marginal probability densities of $x, y, v$ and $z$ of system (5.5). All parameter values are the same as in Figure 5.1.

**Example 5.3.**

In order to confirm whether rapid reversion rates or low volatility levels can result in the extinction of infected cells and viruses, we chose specific parameter values, including $u = 0.000043, k = 1$, and $(\rho_1, \rho_2, \sigma_1, \sigma_2) = (1.9, 1.9, 0.1, 0.2)$, while keeping the remaining parameters consistent with those listed in Table 5.1. Subsequently, our computations revealed the outcomes

$$\begin{aligned}\mathcal{R}_0^{ex} =& \mathcal{R}_0 \\ &+ \frac{\mathcal{R}_0 ud}{\min_{1\le i\le 2}\left\{\frac{\bar{\beta}_i}{\zeta_i}\right\}\mu_1}\left(e^{\frac{\sigma_1^2}{\rho_1}} - 2e^{\frac{\sigma_1^2}{4\rho_1}} + 1\right)^{\frac{1}{2}} \\ &+ \frac{\bar{\beta}_2 \Lambda}{\min_{1\le i\le 2}\left\{\frac{\bar{\beta}_i}{\zeta_i}\right\}\mu_1}\left(e^{\frac{\sigma_2^2}{\rho_2}} - 2e^{\frac{\sigma_2^2}{4\rho_2}} + 1\right)^{\frac{1}{2}} \\ &\approx 0.0023 < 1.\end{aligned}$$

According to Theorem 5.7, it becomes evident that the infected cells and viruses will experience exponential extinction. This assertion is further supported by the findings depicted in Figure 5.4.

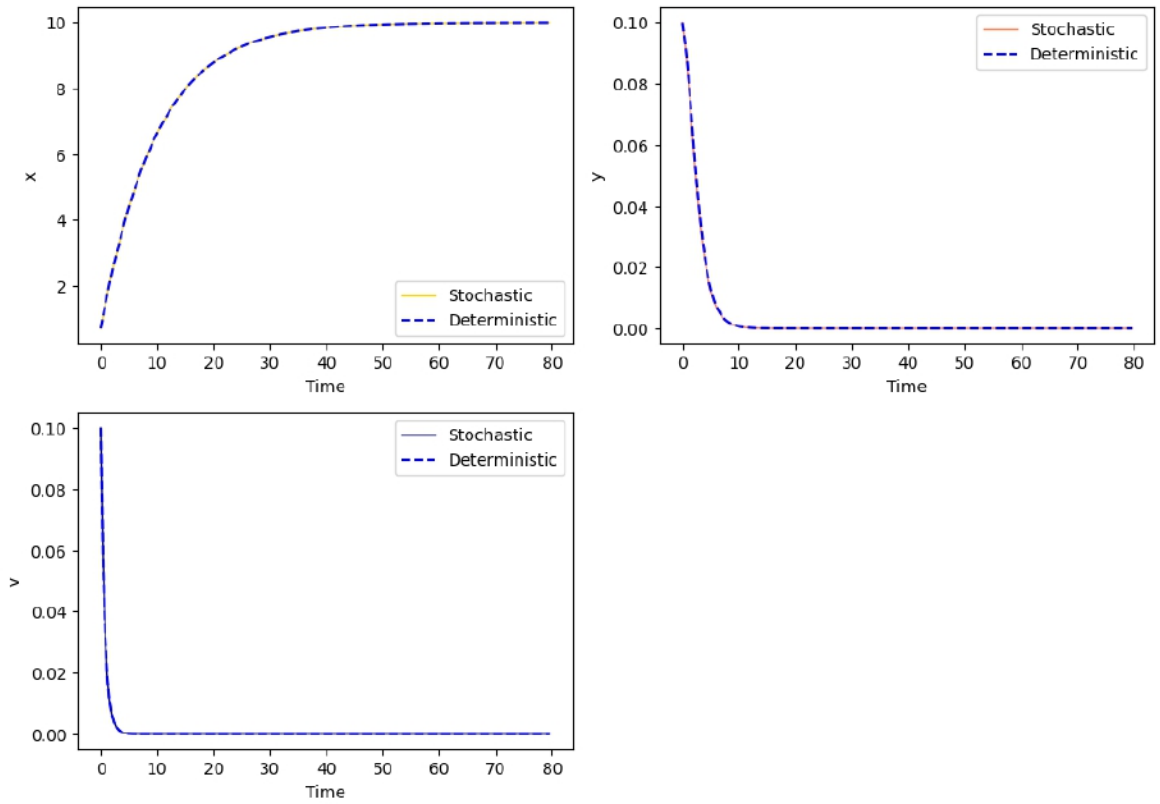



Figure 5.4: The solutions of the deterministic system (5.1) and the stochastic system (5.5) with the parameter values $u = 0.000043, k = 1$, and $(\rho_1, \rho_2, \sigma_1, \sigma_2) = (1.9, 1.9, 0.1, 0.2)$.

## 5.7 Discussion

This research focuses on a stochastic model of HIV-1 that incorporates virus-to-cell and cell-to-cell transmission pathways, alongside humoral immune responses and the Black-Karasinski process. This integrated approach provides a robust framework for analyzing within-host infection dynamics. The study begins by confirming the existence of a unique global solution for the system (5.5), which establishes a critical foundation for understanding the model's dynamic behavior. Through the application of a stochastic Lyapunov function, we derive sufficient conditions for the existence of a stationary distribution, offering insights into the system's long-term behavior. Furthermore, under these conditions, we determine the explicit probability density function that describes the distribution of system states around the quasi-chronic infection equilibrium.

Mathematically, the stationary distribution reflects weak stochastic stability, while the probability density function adds a nuanced perspective on the system's dynamics. Biologically, these findings indicate the sustained coexistence and interaction of all relevant cell populations and free viruses. Additionally, the study identifies conditions under which infected cells and free viruses can be eradicated, providing a theoretical foundation for designing effective disease control strategies. Notably, our results underscore the importance of parameters such as reversion speed and volatility intensity in regulating infection dynamics, with direct implications for disease management strategies.

To complement the theoretical results, numerical simulations illustrate the influence of environmental noise on population stability, offering novel perspectives for controlling HIV infections. Despite these contributions, further exploration is necessary to expand the model's applicability. Extending the Black-Karasinski process to other parameters within the system could improve its realism and alignment with empirical observations. Moreover, examining the impact of alternative stochastic disturbances, such as nonlinear noise or colored perturbations, may reveal additional complexities in infection dynamics. Collaborative studies with experimental researchers and clinicians, coupled with rigorous empirical validation, are essential to enhance the translational relevance of our findings, bridging the gap between theoretical insights and practical applications.

# Conclusion and Perspectives

This thesis has presented a comprehensive exploration of prominent stochastic epidemic models, aiming to address several open questions and challenges in the field through the development of innovative analytical frameworks. By introducing and leveraging novel methodologies distinct from those commonly employed in the literature, the research bridges critical gaps and contributes to a deeper understanding of stochastic epidemiological dynamics. This conclusion encapsulates the major achievements of the thesis, situating them within the broader context of mathematical epidemiology, while also identifying potential pathways for future research.

In Chapters 3 and 4, the stochastic dynamics of the coronavirus and measles epidemic models were analyzed with the incorporation of the Ornstein-Uhlenbeck (OU) process. Through rigorous analysis, it was demonstrated that the stochastic systems admit unique, positive, global solutions, ensuring the well-posedness of these models. By constructing carefully chosen Lyapunov functions, two critical thresholds were established:

- When $\mathcal{R}_0^s > 1$, the existence of a stationary distribution with ergodic properties is guaranteed, signifying the persistence of the disease over time.
- Conversely, when $\mathcal{R}_0^{ex} < 1$, exponential extinction of the infectious population occurs, reflecting effective disease control.

These theoretical insights were further corroborated through extensive numerical simulations, which provided a vivid representation of the impact of stochastic perturbations on epidemic dynamics.

Chapter 5 extended this analysis by solving the corresponding Fokker-Planck equation to derive an approximate expression for the probability density function

near a quasi-positive equilibrium. This result enriches the understanding of the stochastic system's behavior in the vicinity of equilibrium states and underscores the robustness of the proposed analytical techniques.

## The Role of the Ornstein-Uhlenbeck Process

A pivotal innovation in this thesis is the integration of the Ornstein-Uhlenbeck process into epidemic modeling. Its mean-reverting property ensures that transition rates stabilize around biologically plausible mean values, offering a more realistic depiction of random perturbations than traditional Brownian motion. Specifically:

- In Chapter 3, the OU process was employed to introduce stochastic perturbations to the transmission rate ($\delta$), modeling uncertainties in detecting infectious individuals.
- In Chapter 4, the OU process captured environmental noise influencing the progression rate from the exposed to infectious class ($\delta_1$) and the disease-induced mortality rate for measles ($\delta_2$).

This approach highlights the utility of the OU process in reconciling randomness with stability, making it a versatile tool for stochastic modeling in epidemiology.

## Refinement of Hypotheses and Assumptions

Building upon the insights of previous chapters, Chapter 6 proposed an alternative framework by incorporating the Black-Karasinski process. This process, which models the logarithm of parameters such as the recovery rate ($\beta_1$), addresses limitations of the OU process, particularly its occasional failure to ensure non-negativity. By integrating the Black-Karasinski process, the research achieves a more biologically consistent representation of key epidemic parameters while expanding the applicability of stochastic modeling techniques in epidemiology.

## Methodological Contributions

The analytical approaches developed in this thesis, including Lyapunov-based techniques, solutions to the Fokker-Planck equation, and the integration of advanced

stochastic processes, provide a robust toolkit for analyzing complex stochastic systems. These methodologies not only offer theoretical insights but also enhance the practical applicability of epidemic models in predicting disease dynamics and evaluating control strategies.

## Future Directions

The findings of this thesis pave the way for several exciting research directions:

1. **Incorporation of Lévy Processes:** Extending the stochastic framework to include Lévy processes would allow the modeling of jumps and heavy-tailed distributions, capturing rare but impactful epidemiological events.

2. **Machine Learning Integration:** Combining stochastic analysis with machine learning techniques could improve predictive accuracy and optimize intervention strategies.

3. **Network-Based Epidemic Models:** Future work could explore stochastic models on networks, considering contact structures and spatial heterogeneity.

4. **Time-Dependent Perturbations:** Investigating the impact of temporally varying stochastic perturbations could provide deeper insights into seasonal or periodic disease dynamics.

5. **Adaptive Immunity Dynamics:** Integrating stochastic processes with models of adaptive immune responses offers a promising avenue for understanding long-term disease-host interactions.

In summary, this thesis has contributed significantly to the mathematical analysis of random epidemic models, offering new perspectives on persistence, extinction, and stochastic stability. By employing advanced stochastic processes such as the Ornstein-Uhlenbeck and Black-Karasinski processes, the research provides a versatile and rigorous framework for studying epidemic dynamics under uncertainty. The methodologies and insights developed here not only address pressing challenges in stochastic epidemiology but also lay a strong foundation for future interdisciplinary research.

# Bibliography


[1] Geoff P Garnett. An introduction to mathematical models in sexually transmitted disease epidemiology. *Sexually transmitted infections*, 78(1):7–12, 2002.

[2] James M Hyman and E Ann Stanley. Using mathematical models to understand the aids epidemic. *Mathematical Biosciences*, 90(1-2):415–473, 1988.

[3] William Ogilvy Kermack and Anderson G McKendrick. A contribution to the mathematical theory of epidemics. *Proceedings of the royal society of london. Series A, Containing papers of a mathematical and physical character*, 115(772): 700–721, 1927.

[4] Dumitru Baleanu, Fahimeh Akhavan Ghassabzade, Juan J Nieto, and Amin Jajarmi. On a new and generalized fractional model for a real cholera outbreak. *Alexandria Engineering Journal*, 61(11):9175–9186, 2022.

[5] Hai-Feng Huo, Rui Chen, and Xun-Yang Wang. Modelling and stability of hiv/aids epidemic model with treatment. *Applied Mathematical Modelling*, 40 (13-14):6550–6559, 2016.

[6] Noura Yousfi, Khalid Hattaf, and Abdessamad Tridane. Modeling the adaptive immune response in hbv infection. *Journal of mathematical biology*, 63: 933–957, 2011.

[7] Youming Guo and Tingting Li. Modeling the competitive transmission of the omicron strain and delta strain of covid-19. *Journal of Mathematical Analysis and Applications*, 526(2):127283, 2023.

[8] Muhammad Altaf Khan and Abdon Atangana. Modeling the dynamics of novel coronavirus (2019-ncov) with fractional derivative. *Alexandria Engineering Journal*, 59(4):2379–2389, 2020.

[9] Faïçal Ndaïrou, Iván Area, Juan J Nieto, and Delfim FM Torres. Mathematical modeling of covid-19 transmission dynamics with a case study of wuhan. *Chaos, Solitons & Fractals*, 135:109846, 2020.

[10] Rukhsar Ikram, Amir Khan, Mostafa Zahri, Anwar Saeed, Mehmet Yavuz, and Poom Kumam. Extinction and stationary distribution of a stochastic covid-19 epidemic model with time-delay. *Computers in Biology and Medicine*, 141: 105115, 2022.

[11] Qun Liu, Daqing Jiang, Tasawar Hayat, and Bashir Ahmad. Stationary distribution and extinction of a stochastic predator–prey model with additional food and nonlinear perturbation. *Applied Mathematics and Computation*, 320: 226–239, 2018.

[12] Baoquan Zhou, Bingtao Han, Daqing Jiang, Tasawar Hayat, and Ahmed Alsaedi. Stationary distribution, extinction and probability density function of a stochastic vegetation–water model in arid ecosystems. *Journal of Nonlinear Science*, 32(3):30, 2022.

[13] Yu-Ming Chu, Zain Ul Abadin Zafar, Mustafa Inc, Shumaila Javeed, and Abu Safyan Ali. Numerical modeling of a novel stochastic coronavirus. *Fractals*, 30(08):2240211, 2022.

[14] W.H. Hamer. *The Milroy Lectures on Epidemic Diseases in England: The Evidence of Variability and of Persistency of Type ; Delivered Before the Royal College of Physicians of London, March 1st, 6th, and 8th, 1906*. Milroy lectures. Bedford Press, 1906.

[15] Shigui Ruan, Dongmei Xiao, and John C Beier. On the delayed ross–macdonald model for malaria transmission. *Bulletin of mathematical biology*, 70(4):1098–1114, 2008.

[16] David L Smith, Katherine E Battle, Simon I Hay, Christopher M Barker, Thomas W Scott, and F Ellis McKenzie. Ross, macdonald, and a theory for the

dynamics and control of mosquito-transmitted pathogens. *PLoS pathogens*, 8 (4):e1002588, 2012.

[17] William Ogilvy Kermack and Anderson G McKendrick. Contributions to the mathematical theory of epidemics. ii.—the problem of endemicity. *Proceedings of the Royal Society of London. Series A, containing papers of a mathematical and physical character*, 138(834):55–83, 1932.

[18] William Ogilvy Kermack and Anderson G McKendrick. Contributions to the mathematical theory of epidemics. iii.—further studies of the problem of endemicity. *Proceedings of the Royal Society of London. Series A, Containing papers of a mathematical and physical character*, 141(843):94–122, 1933.

[19] George Macdonald. *The Epidemiology and Control of Malaria*. Oxford University Press, London, United Kingdom, 1957.

[20] Bailey N.T.J. *The Mathematical Theory of Infectious Diseases and Its Applications*. Hafner Press, 1975. ISBN 9780028407401.

[21] Klaus Dietz. Epidemics and rumours: A survey. *Journal of the Royal Statistical Society: Series A (General)*, 130(4):505–528, 1967.

[22] Herbert W Hethcote. Qualitative analyses of communicable disease models. *Mathematical biosciences*, 28(3-4):335–356, 1976.

[23] Herbert W Hethcote. Three basic epidemiological models. In *Applied mathematical ecology*, pages 119–144. Springer, 1989.

[24] Jaime Mena-Lorcat and Herbert W Hethcote. Dynamic models of infectious diseases as regulators of population sizes. *Journal of mathematical biology*, 30 (7):693–716, 1992.

[25] Herbert W Hethcote. An age-structured model for pertussis transmission. *Mathematical biosciences*, 145(2):89–136, 1997.

[26] Kenneth L Cooke and P Van Den Driessche. Analysis of an seirs epidemic model with two delays. *Journal of mathematical biology*, 35:240–260, 1996.

[27] Yuliya N Kyrychko and Konstantin B Blyuss. Global properties of a delayed sir model with temporary immunity and nonlinear incidence rate. *Nonlinear analysis: real world applications*, 6(3):495–507, 2005.

[28] Michael L Taylor and Thomas W Carr. An sir epidemic model with partial temporary immunity modeled with delay. *Journal of Mathematical Biology*, 59: 841–880, 2009.

[29] Luosheng Wen and Xiaofan Yang. Global stability of a delayed sirs model with temporary immunity. *Chaos, Solitons & Fractals*, 38(1):221–226, 2008.

[30] Herbert W Hethcote. The mathematics of infectious diseases. *SIAM review*, 42(4):599–653, 2000.

[31] Lijuan Chen and Jitao Sun. Global stability of an si epidemic model with feedback controls. *Applied Mathematics Letters*, 28:53–55, 2014.

[32] PRASENJIT Das, DEBASIS Mukherjee, and Ying-Hen Hsieh. An si epidemic model with saturation incidence: discrete and stochastic version. *International Journal of Nonlinear Analysis and Applications*, pages 1–9, 2011.

[33] Bing Liu, Ying Duan, and Shi Luan. Dynamics of an si epidemic model with external effects in a polluted environment. *Nonlinear Analysis: Real World Applications*, 13(1):27–38, 2012.

[34] Alberto d'Onofrio. A note on the global behaviour of the network-based sis epidemic model. *Nonlinear Analysis: Real World Applications*, 9(4):1567–1572, 2008.

[35] Pauline van den Driessche and James Watmough. A simple sis epidemic model with a backward bifurcation. *Journal of Mathematical Biology*, 40(6): 525–540, 2000.

[36] Edoardo Beretta and Yasuhiro Takeuchi. Global stability of an sir epidemic model with time delays. *Journal of mathematical biology*, 33(3):250–260, 1995.

[37] Hongbin Guo, Michael Y Li, and Zhisheng Shuai. Global stability of the endemic equilibrium of multigroup sir epidemic models. *Canadian applied mathematics quarterly*, 14(3):259–284, 2006.

[38] Xia Ma, Yicang Zhou, and Hui Cao. Global stability of the endemic equilibrium of a discrete sir epidemic model. *Advances in Difference Equations*, 2013: 1–19, 2013.

[39] C Connell McCluskey. Global stability for an sir epidemic model with delay and nonlinear incidence. *Nonlinear Analysis: Real World Applications*, 11(4): 3106–3109, 2010.

[40] Marcos A Capistrán, Miguel A Moreles, and Bruno Lara. Parameter estimation of some epidemic models. the case of recurrent epidemics caused by respiratory syncytial virus. *Bulletin of mathematical biology*, 71:1890–1901, 2009.

[41] Brian J Coburn, Bradley G Wagner, and Sally Blower. Modeling influenza epidemics and pandemics: insights into the future of swine flu (h1n1). *BMC medicine*, 7:1–8, 2009.

[42] Stefan Edlund, James Kaufman, Justin Lessler, Judith Douglas, Michal Bromberg, Zalman Kaufman, Ravit Bassal, Gabriel Chodick, Rachel Marom, Varda Shalev, et al. Comparing three basic models for seasonal influenza. *Epidemics*, 3(3-4):135–142, 2011.

[43] Andrei Korobeinikov. Lyapunov functions and global properties for seir and seis epidemic models. *Mathematical medicine and biology: a journal of the IMA*, 21(2):75–83, 2004.

[44] Andrei Korobeinikov. Global properties of sir and seir epidemic models with multiple parallel infectious stages. *Bulletin of mathematical biology*, 71:75–83, 2009.

[45] Michael Y Li, Hal L Smith, and Liancheng Wang. Global dynamics of an seir epidemic model with vertical transmission. *SIAM Journal on Applied Mathematics*, 62(1):58–69, 2001.

[46] Chengjun Sun, Yiping Lin, and Shoupeng Tang. Global stability for an special seir epidemic model with nonlinear incidence rates. *Chaos, Solitons & Fractals*, 33(1):290–297, 2007.

[47] Juan Zhang and Zhien Ma. Global dynamics of an seir epidemic model with saturating contact rate. *Mathematical Biosciences*, 185(1):15–32, 2003.

[48] Egbert de Boer. Classical and non-classical models of the cochlea. *The Journal of the Acoustical Society of America*, 101(4):2148–2150, 1997.

[49] Asif Ahmed Essak and Brahim Boukanjime. Global stability of an sairs epidemic model with vaccinations, transient immunity and treatment. *Nonlinear Analysis: Real World Applications*, 73:103887, 2023.

[50] Stefania Ottaviano. Threshold dynamics of sairs epidemic model with semi-markov switching. *Mathematical Methods in the Applied Sciences*, 46(9):10289–10310, 2023.

[51] Stefania Ottaviano, Mattia Sensi, and Sara Sottile. Global stability of sairs epidemic models. *Nonlinear Analysis: Real World Applications*, 65:103501, 2022.

[52] Stefania Ottaviano, Mattia Sensi, and Sara Sottile. Global stability of multi-group sairs epidemic models. *Mathematical Methods in the Applied Sciences*, 46(13):14045–14071, 2023.

[53] Qun Liu, Daqing Jiang, and Ningzhong Shi. Threshold behavior in a stochastic siqr epidemic model with standard incidence and regime switching. *Applied Mathematics and Computation*, 316:310–325, 2018.

[54] S Suganya and V Parthiban. Optimal control analysis of fractional order delayed siqr model for covid-19. *The European Physical Journal Special Topics*, pages 1–13, 2024.

[55] Li Tiantian and Xue Yakui. Global stability analysis of a delayed seiqr epidemic model with quarantine and latent. *Applied Mathematics*, 2013, 2013.

[56] Kai Wang, Hongjie Fan, and Yanling Zhu. Dynamics and application of a generalized siqr epidemic model with vaccination and treatment. *Applied Mathematical Modelling*, 120:382–399, 2023.

[57] Abdulwasea Alkhazzan, Jungang Wang, Yufeng Nie, Hasib Khan, and Jehad Alzabut. A novel svir epidemic model with jumps for understanding the dynamics of the spread of dual diseases. *Chaos: An Interdisciplinary Journal of Nonlinear Science*, 34(9), 2024.

[58] Saiful Islam. Equilibriums and stability of svir epidemic model. *International Journal of Humanities, Arts, Medicine and Sciences*, 3:1–10, 2015.

[59] Xianning Liu, Yasuhiro Takeuchi, and Shingo Iwami. Svir epidemic models with vaccination strategies. *Journal of Theoretical biology*, 253(1):1–11, 2008.

[60] Amer M Salman, Mohd Hafiz Mohd, and Ahmed Muhammad. A novel approach to investigate the stability analysis and the dynamics of reaction–diffusion svir epidemic model. *Communications in Nonlinear Science and Numerical Simulation*, 126:107517, 2023.

[61] Xiaomin Lan, Guangmin Chen, Ruiyang Zhou, Kuicheng Zheng, Shaojian Cai, Fengying Wei, Zhen Jin, and Xuerong Mao. An seihr model with age group and social contact for analysis of fuzhou covid-19 large wave. *Infectious Disease Modelling*, 9(3):728–743, 2024.

[62] Ruiwu Niu, Eric WM Wong, Yin-Chi Chan, Michael Antonie Van Wyk, and Guanrong Chen. Modeling the covid-19 pandemic using an seihr model with human migration. *Ieee Access*, 8:195503–195514, 2020.

[63] Yanjin Wang, Pei Wang, Shudao Zhang, and Hao Pan. Uncertainty modeling of a modified seir epidemic model for covid-19. *Biology*, 11(8):1157, 2022.

[64] Yuanyuan Yu, Hui Wan, and Huaiping Zhu. The threshold value of the number of hospital beds in a seihr epidemic model. *Discrete and Continuous Dynamical Systems-B*, 28(2):1436–1470, 2023.

[65] Hisashi Inaba. Age-structured homogeneous epidemic systems with application to the mseir epidemic model. *Journal of mathematical biology*, 54:101–146, 2007.

[66] D.J. Daley and J. Gani. *Epidemic Modelling: An Introduction*. Cambridge Studies in Mathematical Biology. Cambridge University Press, 2001. ISBN 9780521014670.

[67] MS Bartlett. Some evolutionary stochastic processes. *Journal of the Royal Statistical Society. Series B (Methodological)*, 11(2):211–229, 1949.

[68] Maurice Stevenson Bartlett. *An introduction to stochastic processes*. University Press Cambridge, 1966.

[69] David G Kendall. Branching processes since 1873. *Journal of the London Mathematical Society*, 1(1):385–406, 1966.

[70] Frank Ball. Deterministic and stochastic epidemics with several kinds of susceptibles. *Advances in applied probability*, 17(1):1–22, 1985.

[71] Frank Ball and Peter Donnelly. Strong approximations for epidemic models. *Stochastic processes and their applications*, 55(1):1–21, 1995.

[72] Frank Ball, David Sirl, and Pieter Trapman. Analysis of a stochastic sir epidemic on a random network incorporating household structure. *Mathematical Biosciences*, 224(2):53–73, 2010.

[73] Andrew D Barbour. The duration of the closed stochastic epidemic. *Biometrika*, 62(2):477–482, 1975.

[74] Niels G Becker. On a general stochastic epidemic model. *Theoretical Population Biology*, 11(1):23–36, 1977.

[75] Norman C Severo. Generalizations of some stochastic epidemic models. *Mathematical Biosciences*, 4(3-4):395–402, 1969.

[76] Tom Britton. Stochastic epidemic models: a survey. *Mathematical biosciences*, 225(1):24–35, 2010.

[77] Alison Gray, David Greenhalgh, Liangjian Hu, Xuerong Mao, and Jiafeng Pan. A stochastic differential equation sis epidemic model. *SIAM Journal on Applied Mathematics*, 71(3):876–902, 2011.

[78] Jean-C Pedjeu and Gangaram S Ladde. Stochastic fractional differential equations: Modeling, method and analysis. *Chaos, Solitons & Fractals*, 45(3):279–293, 2012.

[79] Yanan Zhao and Daqing Jiang. The threshold of a stochastic sirs epidemic model with saturated incidence. *Applied Mathematics Letters*, 34:90–93, 2014.

[80] Mohamed El Fatini, Aadil Lahrouz, Roger Pettersson, Adel Settati, and Regragui Taki. Stochastic stability and instability of an epidemic model with relapse. *Applied Mathematics and computation*, 316:326–341, 2018.

[81] Z. Brzezniak and T. Zastawniak. *Basic Stochastic Processes: A Course Through Exercises*. Springer Undergraduate Mathematics Series. Springer London, 2012. ISBN 9781447105336.

[82] J.F.L. Gall. *Brownian Motion, Martingales, and Stochastic Calculus*. Graduate Texts in Mathematics. Springer International Publishing, 2016. ISBN 9783319310893.

[83] D. Revuz and M. Yor. *Continuous Martingales and Brownian Motion*. Die Grundlehren der mathematischen Wissenschaften in Einzeldarstellungen. Springer Berlin Heidelberg, 1991. ISBN 9783540521679.

[84] I. Karatzas and S. Shreve. *Brownian Motion and Stochastic Calculus*. Graduate Texts in Mathematics (113) (Book 113). Springer New York, 1991. ISBN 9780387976556.

[85] P.A. Meyer. *Martingales and Stochastic Integrals*. Number 1 in Lecture notes in mathematics. Springer-Verlag, 1972. ISBN 9783540059837.

[86] X. Mao. *Stochastic Differential Equations and Applications*. Woodhead Publishing, 2007. ISBN 9780857099402.

[87] H.H. Kuo. *Introduction to Stochastic Integration*. Universitext. Springer New York, 2006. ISBN 9780387310572.

[88] F. Comets and T. Meyre. *Calcul stochastique et modèles de diffusions - 2e éd.* Mathématiques appliquées pour le Master/SMAI. Dunod, 2015. ISBN 9782100739578.

[89] Carlos Mejia. The ornstein-uhlenbeck process. an introduction for commodity modelling with some extensions. an example with the london cocoa net spot instantaneous convenience yield (el proceso ornstein-uhlenbeck. una introducción para el modelaje en commodities con algunas extensiones. un ejemplo con la tasa neta, spot e instantanea de conveniencia asociada al cocoa futures de londres). 2017.

[90] E. Allen. *Modeling with Itô Stochastic Differential Equations*. Mathematical Modelling: Theory and Applications. Springer Netherlands, 2007. ISBN 9781402059537.

[91] N. Ikeda and S. Watanabe. *Stochastic Differential Equations and Diffusion Processes*. North-Holland mathematical library. North-Holland Publishing Company, 1981. ISBN 9780444861726.

[92] W. Rudin. *Real and Complex Analysis*. Mathematics series. McGraw-Hill, 1987. ISBN 9780071002769.

[93] Ying Yang, Jingwen Zhang, Kaiyuan Wang, and Guofang Zhang. Stationary distribution, density function and extinction of a stochastic siqr epidemic model with ornstein–uhlenbeck process. *Chaos, Solitons & Fractals*, 184:114972, 2024.

[94] Hannes Risken and Hannes Risken. *Fokker-planck equation*. Springer, 1996.

[95] C. Gardiner. *Stochastic Methods: A Handbook for the Natural and Social Sciences*. Springer Series in Synergetics. Springer Berlin Heidelberg, 2010. ISBN 9783642089626.

[96] Constantinos I Siettos and Lucia Russo. Mathematical modeling of infectious disease dynamics. *Virulence*, 4(4):295–306, 2013.

[97] Herbert W Virgin IV and Samuel H Speckt. Unraveling immunity to $\gamma$-herpesviruses: a new model for understanding the role of immunity in chronic virus infection. *Current opinion in immunology*, 11(4):371–379, 1999.

[98] Fan Bai, Krystin ES Huff, and Linda JS Allen. The effect of delay in viral production in within-host models during early infection. *Journal of biological dynamics*, 13(sup1):47–73, 2019.

[99] Bingtao Han, Daqing Jiang, Baoquan Zhou, Tasawar Hayat, and Ahmed Alsaedi. Stationary distribution and probability density function of a stochastic sirsi epidemic model with saturation incidence rate and logistic growth. *Chaos, Solitons & Fractals*, 142:110519, 2021.

[100] Aziz Laaribi, Brahim Boukanjime, Mohamed El Khalifi, Driss Bouggar, and Mohamed El Fatini. A generalized stochastic sirs epidemic model incorporating mean-reverting ornstein–uhlenbeck process. *Physica A: Statistical Mechanics and its Applications*, 615:128609, 2023.

[101] Qun Liu. Stationary distribution and extinction of a stochastic hliv model with viral production and ornstein–uhlenbeck process. *Communications in Nonlinear Science and Numerical Simulation*, 119:107111, 2023.

[102] Baoquan Zhou, Daqing Jiang, Bingtao Han, and Tasawar Hayat. Threshold dynamics and density function of a stochastic epidemic model with media coverage and mean-reverting ornstein–uhlenbeck process. *Mathematics and Computers in Simulation*, 196:15–44, 2022.

[103] Yaxin Zhou and Daqing Jiang. Dynamical behavior of a stochastic siqr epidemic model with ornstein–uhlenbeck process and standard incidence rate after dimensionality reduction. *Communications in Nonlinear Science and Numerical Simulation*, 116:106878, 2023.

[104] Yacine El hadj Moussa, Ahmed Boudaoui, Saif Ullah, Khursheed Muzammil, and Muhammad Bilal Riaz. Application of fractional optimal control theory for the mitigating of novel coronavirus in algeria. *Results in physics*, 39: 105651, 2022.

[105] SP Gatyeni, CW Chukwu, Faraimunashe Chirove, F Nyabadza, et al. Application of optimal control to the dynamics of covid-19 disease in south africa. *Scientific African*, 16:e01268, 2022.

[106] Ghulam Hussain, Amir Khan, Mostafa Zahri, and Gul Zaman. Ergodic stationary distribution of stochastic epidemic model for hbv with double saturated incidence rates and vaccination. *Chaos, Solitons & Fractals*, 160:112195, 2022.

[107] Qun Liu and Daqing Jiang. Stationary distribution and extinction of a stochastic sir model with nonlinear perturbation. *Applied Mathematics Letters*, 73:8–15, 2017.

[108] Edward Allen. Environmental variability and mean-reverting processes. *Discrete Contin. Dyn. Syst. Ser. B*, 21(7):2073–2089, 2016.

[109] Buyu Wen, Bing Liu, and Qianqian Cui. Analysis of a stochastic sib cholera model with saturation recovery rate and ornstein-uhlenbeck process. *Mathematical Biosciences and Engineering*, 20(7):11644–11655, 2023.

[110] Baoquan Zhou, Daqing Jiang, and Tasawar Hayat. Analysis of a stochastic population model with mean-reverting ornstein–uhlenbeck process and allee effects. *Communications in Nonlinear Science and Numerical Simulation*, 111: 106450, 2022.

[111] Qun Liu and Daqing Jiang. Analysis of a stochastic logistic model with diffusion and ornstein–uhlenbeck process. *Journal of Mathematical Physics*, 63(5), 2022.

[112] Xuerong Mao. *Stochastic Differential Equations and Their Applications*. Horwood Publishing, Chichester, 1997.

[113] Yongli Cai, Jianjun Jiao, Zhanji Gui, Yuting Liu, and Weiming Wang. Environmental variability in a stochastic epidemic model. *Applied Mathematics and Computation*, 329:210–226, 2018.

[114] Xiaofeng Zhang and Rong Yuan. A stochastic chemostat model with mean-reverting ornstein-uhlenbeck process and monod-haldane response function. *Applied Mathematics and Computation*, 394:125833, 2021.

[115] Anatoliy Yu. Kutoyants. *Statistical Inference for Ergodic Diffusion Processes*. Springer, London, 2003.

[116] Shyan-Shiou Chen, Chang-Yuan Cheng, and Yasuhiro Takeuchi. Stability analysis in delayed within-host viral dynamics with both viral and cellular infections. *Journal of Mathematical Analysis and Applications*, 442(2):642–672, 2016.

[117] Nobuyuki Ikeda and Shinzo Watanabe. *Stochastic Differential Equations and Diffusion Processes*. North-Holland Publishing Company, New York, 1981.

[118] Odo Diekmann and Johan Andre Peter Heesterbeek. *Mathematical epidemiology of infectious diseases: model building, analysis and interpretation*, volume 5. John Wiley & Sons, 2000.

[119] Desmond J Higham. An algorithmic introduction to numerical simulation of stochastic differential equations. *SIAM review*, 43(3):525–546, 2001.

[120] Saif Ullah and Muhammad Altaf Khan. Modeling the impact of non-pharmaceutical interventions on the dynamics of novel coronavirus with optimal control analysis with a case study. *Chaos, Solitons & Fractals*, 139: 110075, 2020.

[121] Muhammad Suleman Rana, Muhammad Usman, Muhammad Masroor Alam, Aamer Ikram, Muhammad Salman, and Massab Umair. The world's largest measles-rubella vaccination campaign in pakistan: time to invest in routine immunization. *Human Vaccines & Immunotherapeutics*, 18(5):2080444, 2022.

[122] F Ashraf and MO Ahmad. Nonstandard finite difference scheme for control of measles epidemiology. *Int. J. Adv. Appl. Sci*, 6(3):79–85, 2019.

[123] S Okyere-Siabouh and IA Adetunde. Mathematical model for the study of measles in cape coast metropolis. *International Journal of Modern Biology and Medicine*, 4(2):110–133, 2013.

[124] Siwaphorn Kanchanarat, Kadkanok Nudee, Settapat Chinviriyasit, and Wirawan Chinviriyasit. Mathematical analysis of pulse vaccination in controlling the dynamics of measles transmission. *Infectious Disease Modelling*, 8 (4):964–979, 2023.

[125] Md Abdul Kuddus, M Mohiuddin, and Azizur Rahman. Mathematical analysis of a measles transmission dynamics model in bangladesh with double dose vaccination. *Scientific reports*, 11(1):16571, 2021.

[126] Oluwatayo Michael Ogunmiloro, Amos Sesan Idowu, Temitope Olu Ogunlade, and Roselyn Opeyemi Akindutire. On the mathematical modeling of measles disease dynamics with encephalitis and relapse under the atangana–baleanu–caputo fractional operator and real measles data of nigeria. *International Journal of Applied and Computational Mathematics*, 7(5):185, 2021.

[127] Allison Portnoy, Yuli Lily Hsieh, Kaja Abbas, Petra Klepac, Heather Santos, Logan Brenzel, Mark Jit, and Matthew Ferrari. Differential health impact of intervention programs for time-varying disease risk: a measles vaccination modeling study. *BMC medicine*, 20(1):1–29, 2022.

[128] Mingwang Shen, Xiang Sun, Yanni Xiao, Yuanbao Liu, Congyue Wang, Zhiguo Wang, Libin Rong, and Zhihang Peng. The impact of supplementary immunization activities on measles transmission dynamics and implications for measles elimination goals: A mathematical modelling study. *Journal of Theoretical Biology*, 551:111242, 2022.

[129] Ratchada Viriyapong and Witchaya Ridbamroong. Global stability analysis and optimal control of measles model with vaccination and treatment. *Journal of Applied Mathematics and Computing*, 62:207–237, 2020.

[130] Ricardo Almeida and Sania Qureshi. A fractional measles model having monotonic real statistical data for constant transmission rate of the disease. *Fractal and Fractional*, 3(4):53, 2019.

[131] SM Garba, MA Safi, and S Usaini. Mathematical model for assessing the impact of vaccination and treatment on measles transmission dynamics. *Mathematical Methods in the Applied Sciences*, 40(18):6371–6388, 2017.

[132] Hailay Weldegiorgis Berhe and Oluwole Daniel Makinde. Computational modelling and optimal control of measles epidemic in human population. *Biosystems*, 190:104102, 2020.

[133] Zhien Ma, Yicang Zhou, and Jianhong Wu. *Modeling and dynamics of infectious diseases*, volume 11. World Scientific, 2009.

[134] Baoquan Zhou, Daqing Jiang, Bingtao Han, and Tasawar Hayat. Threshold dynamics and density function of a stochastic epidemic model with media coverage and mean-reverting ornstein–uhlenbeck process. *Mathematics and Computers in Simulation*, 196:15–44, 2022.

[135] Mhammed Mediani, Abdeldjalil Slama, Ahmed Boudaoui, and Thabet Abdeljawad. Analysis of a stochastic seiuirr epidemic model incorporating the ornstein-uhlenbeck process. *Heliyon*, 10(16), 2024.

[136] Nguyen Huu Du, Dang Hai Nguyen, and G George Yin. Conditions for permanence and ergodicity of certain stochastic predator–prey models. *Journal of Applied Probability*, 53(1):187–202, 2016.

[137] Abraham Berman and Robert J Plemmons. *Nonnegative matrices in the mathematical sciences*. SIAM, 1994.

[138] Getachew Teshome Tilahun, Seleshi Demie, and Alemayehu Eyob. Stochastic model of measles transmission dynamics with double dose vaccination. *Infectious Disease Modelling*, 5:478–494, 2020.

[139] Stephen Edward, KE Raymond, KT Gabriel, Felician Nestory, MG Godfrey, and MP Arbogast. A mathematical model for control and elimination of the transmission dynamics of measles. *Applied and Computational Mathematics*, 4 (6):396–408, 2015.

[140] The Joint United Nations Programme on HIV/AIDS. Confronting inequalities: Global aids update 2021, 2021. Available at: https://www.unaids.org/en/resources/documents/2021/global-aids-update-2021.

[141] Shalini Duggal, Tulsi Das Chugh, and Ashish Kumar Duggal. Hiv and malnutrition: effects on immune system. *Journal of Immunology Research*, 2012 (1):784740, 2012.

[142] Andrew S Edelman and Susan Zolla-Pazner. Aids: a syndrome of immune dysregulation, dysfunction, and deficiency. *The FASEB journal*, 3(1):22–30, 1989.

[143] Jinfang Zhu and William E Paul. Heterogeneity and plasticity of t helper cells. *Cell research*, 20(1):4–12, 2010.

[144] Xiaoliang Zhu and Jinfang Zhu. Cd4 t helper cell subsets and related human immunological disorders. *International journal of molecular sciences*, 21(21): 8011, 2020.

[145] Mesfin Awoke Bekalu and Steven Eggermont. Media use and hiv/aids knowledge: a knowledge gap perspective. *Health promotion international*, 29(4): 739–750, 2014.

[146] Onoja Matthew Akpa and Benjamin Agboola Oyejola. Modeling the transmission dynamics of hiv/aids epidemics: an introduction and a review. *The Journal of Infection in Developing Countries*, 4(10):597–608, 2010.

[147] Martin Nowak and Robert M May. *Virus dynamics: mathematical principles of immunology and virology: mathematical principles of immunology and virology*. Oxford University Press, UK, 2000.

[148] Jerome Feldmann and Olivier Schwartz. Hiv-1 virological synapse: live imaging of transmission. *Viruses*, 2(8):1666, 2010.

[149] Noura H AlShamrani, Reham H Halawani, Wafa Shammakh, and Ahmed M Elaiw. Global properties of hiv-1 dynamics models with ctl immune impairment and latent cell-to-cell spread. *Mathematics*, 11(17):3743, 2023.

[150] Jie Li, Xia Wang, and Yuming Chen. Analysis of an age-structured hiv infection model with cell-to-cell transmission. *The European Physical Journal Plus*, 139(1):78, 2024.

[151] Hui Miao and Meiyan Jiao. Threshold dynamics of an hiv-1 model with both virus-to-cell and cell-to-cell transmissions, immune responses, and three delays. *International Journal of Nonlinear Sciences and Numerical Simulation*, 24 (2):437–466, 2023.

[152] Jiazhe Lin, Rui Xu, and Xiaohong Tian. Threshold dynamics of an hiv-1 virus model with both virus-to-cell and cell-to-cell transmissions, intracellular delay, and humoral immunity. *Applied mathematics and computation*, 315:516–530, 2017.

[153] Ning Bai, Chenwei Song, and Rui Xu. Mathematical analysis and application of a cholera transmission model with waning vaccine-induced immunity. *Nonlinear Analysis: Real World Applications*, 58:103232, 2021.

[154] Xiaohong Tian, Rui Xu, and Jiazhe Lin. Mathematical analysis of a cholera infection model with vaccination strategy. *Applied Mathematics and Computation*, 361:517–535, 2019.

[155] Fischer Black and Piotr Karasinski. Bond and option pricing when short rates are lognormal. *Financial Analysts Journal*, 47(4):52–59, 1991.

[156] Yuguo Lin, Daqing Jiang, and Peiyan Xia. Long-time behavior of a stochastic sir model. *Applied Mathematics and Computation*, 236:1–9, 2014.

[157] Xinhong Zhang, Tan Su, and Daqing Jiang. Dynamics of a stochastic sveir epidemic model incorporating general incidence rate and ornstein–uhlenbeck process. *Journal of Nonlinear Science*, 33(5):76, 2023.

[158] Qun Liu. Analysis of a stochastic hiv model with cell-to-cell transmission and ornstein–uhlenbeck process. *Journal of Mathematical Physics*, 64(1), 2023.

[159] C.W. Gardiner. *Handbook of Stochastic Methods for Physics, Chemistry and the Natural Sciences*. Springer, Berlin, 1983.

[160] H Roozen. An asymptotic solution to a two-dimensional exit problem arising in population dynamics. *SIAM Journal on Applied Mathematics*, 49(6):1793–1810, 1989.

[161] Akiko Murase, Toru Sasaki, and Tsuyoshi Kajiwara. Stability analysis of pathogen-immune interaction dynamics. *Journal of Mathematical Biology*, 51: 247–267, 2005.