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Nonlinear stochastic cholera epidemic model under the influence of noise

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ABSTRACT

Epidemic cholera is the term for acute diarrhea brought on by the pathogen's abundance within the human body. A mathematical model for the epidemic cholera is created by analyzing when an individual becomes ill and exhibits signs following exposure to the pathogen concentration. The model is first developed from a deterministic viewpoint and then converted into a model containing stochastic differential equations. Besides offering a biological explanation for the stochastic system, we prove that the corresponding deterministic model has potential equilibria. As such, we introduce stability theorems. The research shows that there is a unique global solution for the proposed stochastic model. Necessary conditions are defined by using the Lyapunov function theory, which ensures that the model remains stable in the average for $\mathbb{R}_0^s > 1$. When $\mathbb{R}^s < 1$, our evidence suggests that the illness is probably gone from the population. To strengthen the validity of the acquired analytical results, graphical solutions were created. This work provides a solid theoretical framework for a thorough comprehension of a range of chronic communicable diseases. In addition, we will provide a method for developing Lyapunov functions that may be used to analyze the stationary distributions of models with nonlinear random disruptions.

Keywords: Environmental Noise; Threshold; Extinction ; Numerical Simulation

1 Introduction

Cholera is caused by intestinal infections caused by the marine animal pathogen Vibrio cholerae. Roughly 200 serogroups make up the bacterium Vibrio cholerae, but only O139 and O1—the two that cause cholera infections—are capable of doing so [1, 2]. Before entering the mucous membrane that covers the intestine and shields its epithelial cells, these bacteria show that they can survive and travel through the acidic environment of the stomach [1, 3]. The small intestine's endothelial cells secrete more water and electrolytes when the bacteria produce enterotoxins in the colonized gut [1]. John Snow showed in 1854 that eating or drinking tainted food or water might start cholera epidemics [4]. Other means of transmission do, however, exist. For example, contact with sick people may contribute to the virus spreading throughout the susceptible individuals. If these people are at a higher risk of getting the illness, they might infect other members of the family who cook or use shared water containers [4]. A person can spread the virus whether or not they show symptoms, and symptoms can appear anytime from a few hours to five days after infection. On the other hand, the first two to three days are often when symptoms start to appear [5]. Severe cramping in the legs, vomiting, and watery diarrhea are the most common symptoms of this illness. Patients with infection must receive treatment

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quickly because they run the danger of dehydration, acidosis, and circulatory collapse if therapy is delayed. This illness has a 12- to 24-hour mortality risk in severe patients [4,6]. Following their recovery, patients may be immune to the illness for three to ten years, according to research. Nevertheless, research also suggests that this immunity can wait a few weeks or months later [4,7]. Diarrheal infections remain the primary cause of mortality for infants and children, in part because of the difficulty in accessing sanitary facilities and clean water in undeveloped and emerging nations [8]. Furthermore, as mentioned by Sun et al. [9], this illness has a weak monitoring system and represents a significant hazard to human civilization due to its high death and morbidity rates. Therefore, it is essential to examine mathematical models that clarify cholera transmission pathways in order to understand the disease's progress and devise control measures.

Various mathematical frameworks have been studied in order to comprehend the cholera transmission patterns; these are reviewed in [1,4,6–9] and the cited literature. In [7], a Susceptible-Infectious-Recovered (SIR) model was presented, which included two types of bacterial concentrations: less-infectious and antibiotic-resistant. Asymptomatic and symptomatic subgroups are also separated within the infected category. Using numerical simulations, optimal control theory, and sensitivity analysis, the authors investigated the cost-effectiveness of several management approaches in two communities where the illness is thought to be endemic. A class for the vibrio bacterial abundance in the environment was included in the SIR-type model that Wang and Modnak [10] examined. Three preventative actions are included in the model: medical care, water cleanliness, and vaccinations. The authors gave the control parameters constant values, and they analyzed the equilibrium points' stability. They conducted an additional analysis of a more thorough cholera model with time-dependent controls by applying Pontryagin's Maximum Principle. This study gave the necessary optimality requirements and proved that the optimal control issue had a solution. The authors included a number of control methods in their study [6], such as treatment, vaccination, isolation, and public health awareness campaigns. Additionally, the model included the concentration of microorganisms as a separate compartment. They contrasted the fundamental reproduction number and the combined threshold parameter with the threshold values connected to treatment, vaccination, and education in order to evaluate the possible advantages for the community. The Lyapunov functional approach was employed to do a stability study of the fixed points.

It is highly advised to apply mathematical modeling techniques to investigate the dynamics of epidemic propagation and develop control measures [11–15]. These models provide a compromise between the robustness of data links and biological scenarios by depicting the infection's natural course. The models produced so far provide an explanation for various aspects of cholera dynamics. While most models take a deterministic approach, epidemiologists researching the dynamics and control of the cholera outbreak are very interested in environmental sounds [23]. Complicating matters are unpredictable elements like social interactions or other characteristics of the population, such as the start and spread of epidemics. That being said, an epidemic's present and future states can be significantly influenced by the variety and unpredictability of its surroundings.

It is important to highlight that there is a strong correlation between the spread and persistence of bacteria and changes in the surrounding environment. Stochastic components are intrinsic to the dynamics of an infection, both with respect to parameters and states. Stochasticity is acknowledged by epidemiology as a key component of epidemic modeling. Despite their inherent randomness, the disturbances included in the model should show positive auto-correlation. Additionally, these fluctuations can be analytically derived from the related problem by the use of the probability density function [24–26]. Modeling epidemics generally uses two main approaches: deterministic modeling and stochastic modeling. As the stochastic approach can offer a greater degree of realism than deterministic models, and hence this approach is preferred when modeling biological systems [27–30]. Stochastic differential equations (SDEs) can be utilized to determine a distribution of predicted outcomes, such as the number of infected individuals over time t. Moreover, a stochastic model produces distinct outcomes after repeated simulations, providing more insightful information than determinis-

tic models. To describe the dynamics of cholera infection, a number of deterministic models, such as those shown in [31,32], have been developed.

In this work, we developed a stochastic epidemic system to clarify the dynamics of cholera transmission, with a particular emphasis on the disease's long-term dynamics in the setting of migrating communities that are vulnerable to contamination with pathogens. There are six distinct groups that make up the population as a whole, which includes both humans and microbes. These sections are designated $\mathcal{S}(t)$, $\mathcal{I}(t)$, $\mathcal{R}(t)$, and $\mathcal{B}(t)$, which stands for the susceptible, infected, isolated, and recovered human subjects as well as the bacterial pathogen, in that order. These groupings are related by mathematical formulas that take into account the features of the disease and incorporate the associated noises from the surroundings. In particular, we factor in the time interval that elapses between an infection and a person's beginning of cholera symptoms.

The remainder of the manuscript is organized as follows. In Section 2, we put forth a model that governs the dynamics of cholera. We give enough information in Section 3 to show that there is a single positive global solution. The study of the infection's persistence and eradication is covered in detail in Sections 4 and 5, respectively. We verify the theoretical results with numerical experiments and graphical displays in Section 6. Section 7 provides a summary of the research and recommendations for more studies.

2 Models formulation

The moments at which a person catches the disease and the instant at which they show symptoms following exposure to the environment will be incorporated into a mathematical model to explain the dynamics of the epidemic cholera. Thus, we will create a model that incorporates the Susceptible-Infectious-Recovered ($\mathcal{SIR}$) type of model and takes into consideration the pathogen's concentration or population density while explaining the dynamics of cholera. The whole human population $N(t)$ is stratified at any time $t \geq 0$ into three sub-groups, $\mathcal{S}(t)$, $\mathcal{I}(t)$ and recovered $\mathcal{R}(t)$, which indicate the sizes of susceptible, infected, and recovered individuals, respectively. We denotes the bacterium concentration in the water or food by the notion $\mathcal{B}(t)$. The population has a constant inflow that is reflected within the term ξ. We consider a uniform death rate $\mu > 0$ and it happens within all the compartments. We assumed two different types of interactions: β_1 shows the interaction of human population with the environment and β_2 is the interaction among human. The vibro bacteria are dying at the constant rate δ. Beside these, we have imposed the following assumption on the model:

A_1 : Each parameter of the system is a positive real number and are nonnegative.

A_2 : The constant, represented by c, represents the average number of encounters in a unit of time.

A_3 : Every person in the population as a whole and the pathogen in the surroundings have an equal chance of moving into a different class. Stated differently, the distribution of exponential types determines the moving probability between the compartments. In an exponential distribution, the inverse of that parameter can be used to compute the expected average time spent in a class.

A_4 : It is assumed that neither the population's size nor its demographic composition will change over time and that no new people will ever permanently join or depart the population. This suggests that the disease is only spreading within the confines of the population and that the dynamics of the epidemic are unaffected by outside forces.

A_5 : Recovered people are immune to cholera since they do not need medical attention. It is believed that no one who has recovered from cholera passes away. Given this, one may reasonably conclude that the natural mortality rate for both susceptible and recovered people is μ. This illustrates the notion that the only people who can die from cholera are those who are ill and untreated or those who are receiving care.

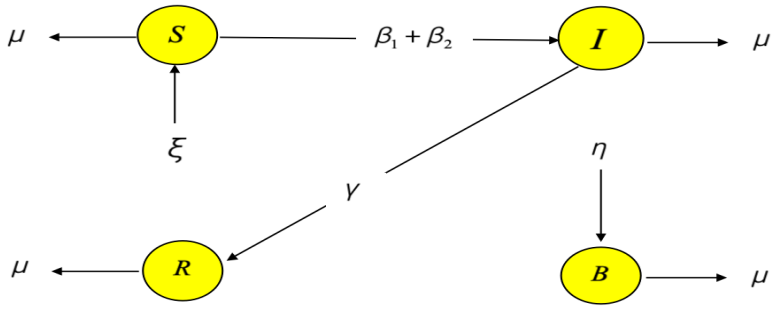


Figure 1: Flow chart of the proposed model (2.1).

The following is the mathematical model that results from the aforementioned assumptions:

$$\begin{aligned}
\mathcal{S}^*(t) &= \xi - \frac{\beta_1 \mathcal{B}(t)\mathcal{S}(t)}{\mathcal{N}(t)} - \frac{\beta_2 \mathcal{I}(t)\mathcal{S}(t)}{\mathcal{N}(t)} - \mu \mathcal{S}(t), \\
\mathcal{I}^*(t) &= \frac{\beta_1 \mathcal{B}(t)\mathcal{S}(t)}{\mathcal{N}(t)} + \frac{\beta_2 \mathcal{I}(t)\mathcal{S}(t)}{\mathcal{N}(t)} - (\mu + \gamma)\mathcal{I}(t), \\
\mathcal{R}^*(t) &= \gamma \mathcal{I}(t) - \mu \mathcal{R}(t), \\
\mathcal{B}^*(t) &= \eta \mathcal{I}(t) - \delta \mathcal{B}(t).
\end{aligned} \tag{2.1}$$

The state transition diagram of the proposed model $\mathcal{SIRB}$ is demonstrated in Figure 1. We can easily determine the following disease-free equilibrium (DFE) points for the suggested model (2.1) using a few simple mathematical computations. As a result, the following describes the equilibrium state of the suggested deterministic model:

$$\mathcal{E}^0 = (\mathcal{S}^0, \mathcal{I}^0, \mathcal{R}^0, \mathcal{B}^0,) = \left(\frac{\xi}{\mu}, 0, 0, 0, 0, 0 \right). \tag{2.2}$$

We compute the basic reproduction number, denoted by $\mathbb{R}_0$, of the model. The associated next-generation matrix to the model is given by

$$\mathcal{F} = \begin{bmatrix} \beta_2 & \beta_1 \\ 0 & 0 \end{bmatrix} \text{ and } \mathcal{V} = \begin{bmatrix} \cdot\gamma + \mu & 0 \\ -\eta & \delta \end{bmatrix}$$

The spectral radius of $\mathcal{F}^{-1}$, i.e., the threshold quantity is given by

$$\mathbb{R}_0 = \rho\left(\mathcal{F}\mathcal{V}^{-1}\right) = \frac{1}{\gamma + \mu}\left(\beta_2 + \beta_1 \frac{\eta}{\delta}\right). \tag{2.3}$$

Based on the framework of [11], the disease-free equilibrium is locally asymptotically stable when $\mathbb{R}_0 < 1$ and unstable when $\mathbb{R}_0 > 1$. Moreover, when $\mathbb{R}_0 > 1$, then there an endemic equilibrium given by

$$E^* = (\mathcal{S}^*, \mathcal{I}^*, \mathcal{Q}^*, \mathcal{R}^*, \mathcal{B}^*), \tag{2.4}$$

The functions $\mathcal{Z}_i(t)$ for $i = 1, \cdots, 4$ with $\mathcal{Z}_i(0) = 0$ in the corresponding classes will be taken into consideration in order to analyze the stochastic fluctuations in system (2.1).

We shall include the functions $\mathcal{Z}_i(t)$ for $i = 1, \cdots, 4$ with starting conditions $\mathcal{Z}_i(0) = 0$ inside their respective classes in order to account for stochastic fluctuations in system (2.1). These functions

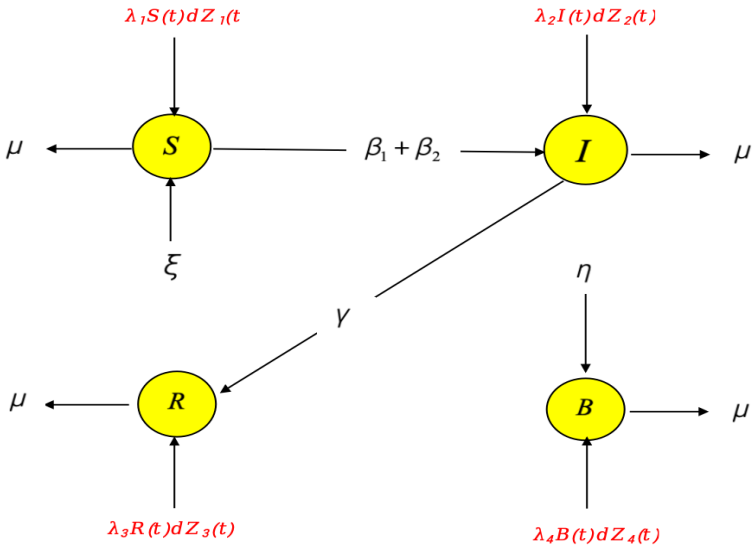


Figure 2: Flow chart of the proposed stochastic model (2.5).

are interpreted biologically by including environmental variations, which are referred to as Brownian movements. For $i = 1, \cdots, 4$, the intensity corresponding to each noise is denoted by λ_i. When these variations are taken into account, the suggested stochastic model becomes

$$\begin{aligned} d\mathcal{S} &= \left[\xi - \frac{\beta_1 \mathcal{B}(t)\mathcal{S}(t)}{\mathcal{N}(t)} - \frac{\beta_2 \mathcal{I}(t)\mathcal{S}(t)}{\mathcal{N}(t)} - \mu \mathcal{S}(t)\right] dt + \lambda_1 \mathcal{S}(t) d\mathcal{Z}_1(t), \\ d\mathcal{I} &= \left[\frac{\beta_1 \mathcal{B}(t)\mathcal{S}(t)}{\mathcal{N}(t)} + \frac{\beta_2 \mathcal{I}(t)\mathcal{S}(t)}{\mathcal{N}(t)} - (\mu + \gamma)\mathcal{I}(t)\right] dt + \lambda_2 \mathcal{I}(t) d\mathcal{Z}_2(t), \\ d\mathcal{R} &= \left[\gamma \mathcal{I}(t) - \mu \mathcal{R}(t)\right] dt + \lambda_3 \mathcal{R}(t) d\mathcal{Z}_3(t), \\ d\mathcal{B} &= \left[\eta \mathcal{I}(t) - \delta \mathcal{B}(t)\right] dt + \lambda_4 \mathcal{B}(t) d\mathcal{Z}_4(t). \end{aligned} \tag{2.5}$$

The state transition diagram of the proposed stochastic model $\mathcal{SIRB}$ is demonstrated in Figure 2. Our goal in this study is to use model (2.5) and look for answers to the following questions:

Q_1 : Does random noise affect how cholera epidemics behave dynamically?

Q_2 : Does the disease's ability to spread be significantly influenced by contaminated water?

Q_3 : Does eating food tainted with Vibrio cholerae alter the course of the underlying illness?

Q_4 : Which of the following criteria is applied to determine if the model has undergone extinction?

Q_5 : Which of the following criteria is used to determine the stationary distribution?

3 Positive global solution of the model

Demonstrating the existence of a non-local solution in the permitted region is a prerequisite for exploring the dynamic behaviors of the system. This can be achieved, for example, by confirming that the associated parameters of the system (2.5) satisfy the growth and Lipschitz criteria, which are required to ensure that the model has a non-negative solution. Prior to delving into these characteristics, let us assert the following crucial assertion and subsequently demonstrate its proof.

Theorem 3.1. *If the initial values of the dependent variables are positive than the stochastic model has a global solution* $(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})(t) \in \mathbb{R}^4_+$ *for system (2.5) for all* $0 \leq t$ *almost surely (a.s).*

Proof: It is easy to see that the model's coefficients are locally Lipschitz for the non-negative initial values of the state variables. This guarantees that the presented issue has a local unique solution in the interval $[0,\tau_e)$ for any time t. The explosion time is denoted by the symbol τ_e. Readers are referred to [34, 35] for further information. Demonstrating that $\tau_e = \infty$ suffices to establish the fact that this type of solution is, in fact, global. In order to demonstrate this, let us take a sufficiently big positive real integer k_0 such that every solution to the issue falls inside the interval $[\frac{1}{k_0}, k_0]$. Moreover, let $k \geq k_0$ and define

$$\begin{aligned}\tau_k = inf\{t \in [0,\tau_e) : \frac{1}{k} \geq \min\{\mathcal{S}(t),\mathcal{I}(t),\mathcal{R}(t),\mathcal{B}(t)\}, \text{ or}\\ k \leq \max\{\mathcal{S}(t),\mathcal{I}(t),\mathcal{R}(t),\mathcal{B}(t)\}.\end{aligned} \tag{3.1}$$

In this work, an empty set is denoted as ϕ if $\inf\phi = \infty$. The definition states that τ_k increases as $k \to \infty$. Assuming that τ_∞ is the limit of τ_k, $\tau_\infty \leq \tau_e$ nearly inevitably (a,s.). Stated otherwise, we must demonstrate that $\tau_\infty = \infty$ a.s. If this claim is untrue, then there are two constants such that $T > 0$ and $\epsilon \in (0,1)$ related to each other as follows

$$\epsilon < P\{T \geq \tau_\infty\}. \tag{3.2}$$

Therefore, for the value $k_0 \leq k_1$, we get

$$P\{\tau_k \leq T\} \geq \epsilon, \ \forall\, k \geq k_1.$$

To proceed, let's establish a Lyapunov function of the subsequent form:

$$\begin{aligned}\mathcal{V} = (\mathcal{S} - c_1 log\frac{\mathcal{S}}{c_1} - c_1) + (-(\log\mathcal{I}+1)+\mathcal{I}) + (\mathcal{R} - log\mathcal{R} - 1)\\ + (-1 - log\mathcal{B} + \mathcal{B}),\end{aligned} \tag{3.3}$$

and later on, the value of c_1 (a constant) will be ascertained. Using Itô's formula, we get at:

$$\begin{aligned}d\mathcal{V} = L\mathcal{V}dt + \lambda_1(\mathcal{S}-c_1)d\mathcal{Z}_1(t) + \lambda_2(\mathcal{I}-1)d\mathcal{Z}_2(t)\\ + \lambda_3(\mathcal{R}-1)d\mathcal{Z}_3(t) + \lambda_4(\mathcal{B}-1)d\mathcal{Z}_4(t),\end{aligned} \tag{3.4}$$

where $\mathcal{V} = \mathcal{V}(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})$. From this function, we can define the $L\mathcal{V}$ operator from $\mathbb{R}^4_+$ to $\mathbb{R}_+$ that has the following form

$$\begin{aligned}L\mathcal{V} &= \left(1-\frac{c_1}{\mathcal{S}}\right)\left(\xi - \frac{\beta_1\mathcal{B}\mathcal{S}}{\mathcal{N}} - \frac{\beta_2\mathcal{I}\mathcal{S}}{\mathcal{N}} - \mu\mathcal{S}\right) + \frac{c_1}{2}{\lambda_1}^2\\ &+ \left(1-\frac{1}{\mathcal{I}}\right)\left(\frac{\beta_1\mathcal{B}\mathcal{S}}{\mathcal{N}} + \frac{\beta_2\mathcal{I}\mathcal{S}}{\mathcal{N}} - (\mu+\gamma)\mathcal{I}\right) + \frac{1}{2}{\lambda_2}^2\\ &+ \left(1-\frac{1}{\mathcal{R}}\right)\left(\gamma\mathcal{I} - \mu\mathcal{R}\right) + \frac{1}{2}{\lambda_3}^2 + \left(1-\frac{1}{\mathcal{B}}\right)\left(\eta\mathcal{I} - \delta\mathcal{B}\right) + \frac{1}{2}{\lambda_4}^2\\ &= \xi - \frac{\beta_1\mathcal{B}\mathcal{S}}{\mathcal{N}} - \frac{\beta_2\mathcal{I}\mathcal{S}}{\mathcal{N}} - \mu\mathcal{S} - c_1\xi + \frac{c_1\beta_1\mathcal{B}}{\mathcal{N}} + \frac{c_1\beta_2\mathcal{I}}{\mathcal{N}} + c_1\mu + \frac{c_1}{2}{\lambda_1}^2\\ &+ \frac{\beta_1\mathcal{B}\mathcal{S}}{\mathcal{N}} + \frac{\beta_2\mathcal{I}\mathcal{S}}{\mathcal{N}} - (\mu+\gamma)\mathcal{I} - \frac{\beta_1\mathcal{B}\mathcal{S}}{\mathcal{I}\mathcal{N}} - \frac{\beta_2\mathcal{S}}{\mathcal{N}} + (\mu+\gamma) + \frac{1}{2}{\lambda_2}^2\\ &+ \gamma\mathcal{I} - \mu\mathcal{R} - \frac{\gamma\mathcal{I}}{\mathcal{R}} + \mu + \eta\mathcal{I} - \delta\mathcal{B} - \frac{\eta\mathcal{I}}{\mathcal{B}} + \delta + \frac{1}{2}{\lambda_3}^2 + \frac{1}{2}{\lambda_4}^2.\end{aligned} \tag{3.5}$$

Let choose $c_1 = \frac{\delta}{\beta_1}$, such that $c_1\beta_1 - \delta = 0$. As $\mathcal{R} + \mathcal{I} + \mathcal{S} \leq 1$, we have

$$L\mathcal{V} \leq \xi + c_1\mu + \beta_2 + 2\mu + \gamma + \delta + \frac{c_1}{2}{\lambda_1}^2 + \frac{1}{2}{\lambda_2}^2 + \frac{1}{2}{\lambda_3}^2 + \frac{1}{2}{\lambda_4}^2 = K. \tag{3.6}$$

Subsequently, the proof structure is much the same as that of [35] for Theorem 2.1. The theorem's proof is therefore finished by eliminating the pointless stages.

4 Extinction

Considering situations where an infectious illness would vanish from the community or go extinct is essential to understanding the dynamics of the disease. The stochastic model (2.5) solution will approach zero with probability one by changing the sizes of the noises, as we shall show in this section. Let's consider the following relation:

$$\langle \P(t) \rangle = \frac{1}{t}\int_0^t \P(s)ds.$$

Lemma 4.1. *(Strong Law) [34,35] Assume that $\mathcal{M} = \{\mathcal{M}\}_{0\leq t}$ is a real-valued and continuous along with the property of local martingale that approaches zero as t approaches 0. Then*

$$\begin{aligned} &\lim_{t\to\infty} \langle \mathcal{W}, \mathcal{W} \rangle_t = \infty, \ \Rightarrow \ \lim_{t\to\infty} \frac{\mathcal{W}_t}{\langle \mathcal{W}, \mathcal{W} \rangle_t} = 0, \ a.s. \\ &\limsup_{t\to\infty} \frac{\langle \mathcal{W}, \mathcal{W} \rangle_t}{t} < 0, \ \Rightarrow \ \lim_{t\to\infty} \frac{\mathcal{W}_t}{t} = 0, \ a.s. \end{aligned} \tag{4.1}$$

Lemma 4.2. *[34] Let $(\mathcal{S}, \mathcal{I}, \mathcal{R}, \mathcal{B})$ be the solution of system (2.5) with initial value $(\mathcal{S}(0), \mathcal{I}(0), \mathcal{R}(0), \mathcal{B}(0)) \in \mathbb{R}^6_+$. Then*

$$\limsup_{t\to\infty} \frac{\ln \mathcal{S}(t)}{t} = 0, \ \limsup_{t\to\infty} \frac{\ln \mathcal{I}(t)}{t} = 0, \ \limsup_{t\to\infty} \frac{\ln \mathcal{R}(t)}{t} = 0, \ \limsup_{t\to\infty} \frac{\ln \mathcal{B}(t)}{t} = 0, \ a.s. \tag{4.2}$$

Furthermore, if $\mu > \frac{\lambda_1^2 \vee \lambda_2^2 \vee \lambda_3^2}{2}$, and $\delta > \frac{\lambda_4^2}{2}$ and then

$$\begin{aligned} \lim_{t\to\infty} \frac{\int_0^t \mathcal{S}(s)d\mathcal{Z}_1(s)}{t} &= 0, \\ \lim_{t\to\infty} \frac{\int_0^t \mathcal{I}(u)d\mathcal{Z}_2(u)}{t} &= 0, \\ \lim_{t\to\infty} \frac{\int_0^t \mathcal{R}(s)d\mathcal{Z}_3(s)}{t} &= 0, \\ \lim_{t\to\infty} \frac{\int_0^t \mathcal{B}(s)d\mathcal{Z}_4(s)}{t} &= 0, \ a.s. \end{aligned} \tag{4.3}$$

Then, the solution of system (2.5)

$$\begin{aligned} \limsup_{t\to\infty} \mathcal{S}(t) &= \frac{\xi}{\mu}, \\ \limsup_{t\to\infty} \mathcal{I}(t) &= 0, \\ \limsup_{t\to\infty} \mathcal{R}(t) &= 0, \\ \limsup_{t\to\infty} \mathcal{B}(t) &= 0, \ \text{a.s.} \end{aligned} \tag{4.4}$$

One may use the evidence of Lemmas 2.1 and 2.2 in [35] to support Lemma 4.2. As a result, the lemma's proof is simple and is left out.

It is first necessary to specify the threshold parameter in order to explain the extinction theory of system (2.5).

$$\mathbb{R}_s = \frac{\beta_1 \eta}{\delta\left(\mu + \gamma + \frac{\lambda_2^2}{2}\right)}.$$

Theorem 4.3. *Let us examine the starting data* $(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})(0)$ *and correspond to this,* $(\mathcal{S},\mathcal{I},\mathcal{Q},\mathcal{R},\mathcal{B})(0) \in \mathbb{R}_+^4$ *represents a single solution of system (2.5). Then, the subsequent relations apply to such solution if* $\mathbb{R}_s < 1$*:*

$$\begin{aligned}
&\lim_{t\to\infty} \langle \mathcal{S}(t) \rangle = \frac{\Pi}{\mu}, \ \ a.s., \\
&\lim_{t\to\infty} \langle \mathcal{I}(t) \rangle = 0, \ \ a.s., \\
&\lim_{t\to\infty} \langle \mathcal{R}(t) \rangle = 0, \ \ a.s., \\
&\lim_{t\to\infty} \langle \mathcal{B}(t) \rangle = 0, \ \ a.s.,
\end{aligned} \tag{4.5}$$

This implies that the infection within the community will certainly be eradicated in the long run.

Proof: Through the integration of system (2.5), we obtained the subsequent set of formulas:

$$\begin{aligned}
\frac{(\mathcal{S}(t)-\mathcal{S}(0))}{t} &= \xi - \frac{\beta_1\langle \mathcal{B}\mathcal{S}\rangle}{\langle \mathcal{N}\rangle} - \frac{\beta_2\langle \mathcal{I}\mathcal{S}\rangle}{\langle \mathcal{N}\rangle} - \mu\langle \mathcal{S}\rangle + \frac{\lambda_1 \int_0^t \mathcal{S}(r)d\mathcal{Z}_1(r)}{t}, \\
\frac{(\mathcal{I}(t)-\mathcal{I}(0))}{t} &= \frac{\beta_1\langle \mathcal{B}\mathcal{S}\rangle}{\langle \mathcal{N}\rangle} - \frac{\beta_2\langle \mathcal{I}\mathcal{S}\rangle}{\langle \mathcal{N}\rangle} - (\mu+\gamma)\langle \mathcal{I}\rangle + \frac{\lambda_2 \int_0^t \mathcal{I}(r)\mathcal{Z}_2(r)}{t}, \\
\frac{(\mathcal{R}(t)-\mathcal{R}(0))}{t} &= \gamma\langle \mathcal{I}\rangle - \mu\langle \mathcal{R}\rangle + \frac{\lambda_3 \int_0^t \mathcal{R}(r)d\mathcal{Z}_3(r)}{t}, \\
\frac{(\mathcal{B}(t)-\mathcal{B}(0))}{t} &= \eta\langle \mathcal{I}\rangle - \delta\langle \mathcal{B}\rangle + \frac{\lambda_4 \int_0^t \mathcal{B}(r)d\mathcal{Z}_4(r)}{t}.
\end{aligned} \tag{4.6}$$

Taking into account the system mentioned above's second-to-last relation, we have:

$$\begin{aligned}
\langle \mathcal{B}\rangle &= \frac{\eta}{\delta}\langle \mathcal{I}\rangle - \frac{1}{\delta}\left(\frac{\mathcal{B}(t)-\mathcal{B}(0)}{t}\right) + \frac{\lambda_4}{\delta}\left(\frac{\int_0^t \mathcal{B}(r)d\mathcal{Z}_4(r)}{t}\right), \\
&= \frac{\eta}{\delta}\langle \mathcal{I}\rangle + \mathcal{M}_1(t),
\end{aligned} \tag{4.7}$$

where

$$\mathcal{M}_1(t) = -\frac{1}{\delta}\left(\frac{\mathcal{B}(t)-\mathcal{B}(0)}{t}\right) + \frac{\lambda_4}{\delta}\left(\frac{\int_0^t \mathcal{B}(r)d\mathcal{Z}_4(r)}{t}\right). \tag{4.8}$$

Using the It'ao formula directly on the second relation in system (2.5) produces the following results:

$$\begin{aligned}
d\log\mathcal{I} &= \left[\frac{\beta_1\mathcal{B}\mathcal{S}}{\mathcal{I}\mathcal{N}} + \frac{\beta_2\mathcal{S}}{\mathcal{N}} - (\mu+\gamma) - \frac{\lambda_2^2}{2}\right]dt + \lambda_2 d\mathcal{Z}_2(t) \\
&\leq \left[\frac{\beta_1\mathcal{B}}{\mathcal{I}} + \beta_2 - (\mu+\gamma+\frac{\lambda_2^2}{2})\right]dt + \lambda_2 d\mathcal{Z}_2(t).
\end{aligned} \tag{4.9}$$

If Eq. (4.9) is integrated from 0 to t and then divided by t, the resulting relation may be obtained with great ease:

$$\frac{\log \mathcal{I} - \log \mathcal{I}(0)}{t} \leq \left[\frac{\beta_1 \langle \mathcal{B} \rangle}{\langle \mathcal{I} \rangle} + \beta_2 - (\mu + \gamma + \frac{\lambda_2^2}{2})\right] + \frac{\lambda_2 d\mathcal{Z}_2(t)}{t}. \tag{4.10}$$

If we substitute relation (4.7) in Eq. (4.10), we have:

$$\begin{aligned} \frac{\log \mathcal{I}(t)}{t} &\leq \left[\frac{\beta_1(\frac{\eta}{\delta}\langle \mathcal{I} \rangle + \mathcal{M}_1(t))}{\langle \mathcal{I} \rangle} + \beta_2 - (\mu + \gamma + \frac{\lambda_2^2}{2})\right] + \frac{\log \mathcal{I}(0)}{t} + \frac{\delta_2 d\mathcal{Z}_2(t)}{t} \\ &\leq \left[\frac{\frac{\beta_1 \eta}{\delta}\langle \mathcal{I} \rangle}{\langle \mathcal{I} \rangle} + \beta_2 - (\mu + \gamma + \frac{\lambda_2^2}{2})\right] + \frac{\beta_1 \mathcal{M}_1(t)}{\langle \mathcal{I} \rangle} + \frac{\log \mathcal{I}(0)}{t} + \frac{\lambda_2 d\mathcal{Z}_2(t)}{t} \\ &= \left[\frac{\beta_1 \eta}{\delta} + \beta_2 - (\mu + \gamma + \frac{\lambda_2^2}{2})\right] + \frac{\beta_1 \mathcal{M}_1(t)}{\langle \mathcal{I} \rangle} + \frac{\log \mathcal{I}(0)}{t} + \frac{\lambda_2 d\mathcal{Z}_2(t)}{t}. \end{aligned} \tag{4.11}$$

Further, $\mathcal{M}_i(t) = \frac{\gamma_i}{t}\int_0^t g_i d\mathcal{W}_i(t)$ for $i = 1, 2, \cdots 4.$, $g_1 = \mathcal{S}, g_2 = \mathcal{I}, g_3 = \mathcal{R}, g_4 = \mathcal{B}$ are the continuous local-martingale function and its value is 0 at $t = 0$. By letting $t \to \infty$ and using Lemma 4.2, we have

$$\lim_{t\to\infty} \sup \frac{1}{t}\mathcal{M}_i(t) = 0. \tag{4.12}$$

Following similar approach, we can easily obtain the result $\lim_{t\to\infty} \sup \mathcal{M}_1(t) = 0$. Further, by choosing $\mathbb{R}_s < 1$, Eq (4.11) leads to

$$\lim_{t\to\infty} \sup \frac{\log \mathcal{I}(t)}{t} \leq \left(\mu + \gamma + \frac{\lambda_2^2}{2}\right)\left(\mathbb{R}_s - 1\right) < 0, \text{ a.s.} \tag{4.13}$$

Due to relationship (4.13), we get

$$\lim_{t\to\infty} \langle \mathcal{I} \rangle = 0, \text{ a.s.} \tag{4.14}$$

Equations (4.7) employ relation (4.14) and the following facts: $\lim_{t\to\infty} \sup \mathcal{M}_1(t) = 0$, we obtain

$$\lim_{t\to\infty} \langle \mathcal{B} \rangle = 0, \text{ a.s,} \tag{4.15}$$

Similarly, we can obtain:

$$\lim_{t\to\infty} \langle \mathcal{R}(t) \rangle = 0, \text{ a.s.} \tag{4.16}$$

Lastly, we shall consider system's initial equation (4.6). We obtain by calculating the integral from 0 to t, dividing the result by t, and applying the relations (4.15) and (4.16)

$$\lim_{t\to\infty} \langle \mathcal{S} \rangle = \frac{\xi}{\mu}, \text{ a.s.} \tag{4.17}$$

This concludes the proof.

5 Ergodic stationary distribution

SDEs models like system (2.5) do not have an endemic fixed point like deterministic models do. As such, it is unable to evaluate the stability of the condition, and more precisely the endemic equilibria (EE), in which the illness continues to exist. Scholars have instead chosen to explore the idea of uniqueness and the presence of stationary distributions as a substitute approach to study the persistence of epidemics within the stochastic framework. This clearly shows that the epidemics are still present. For

future reference, we shall remember a finding by Hasminskii [36]. Before moving further, let us define the following process:

$$d\P(t) = \sum_{r=1}^{d} g_r(t, \P(t)) d\mathcal{B}_r(t). \tag{5.1}$$

The diffusion matrix is

$$\Pi(x) = (\Upsilon_{ij}(x)), \quad \Upsilon_{ij}(x) = \sum_{r=1}^{d} g_r^i(x) g_r^j(x).$$

Lemma 5.1. *Assume that* $\mathbf{U} \in \mathbb{R}^d$ *represents an open bounded domain with a regular border of* Γ. *The domain* $\mathbf{U}$ *has the following characteristics:*

1. *The lowest eigenvalue of the matrix* $\mathbf{A}(\mathbf{t})$ *is constrained away from zero in the vicinity of* $\mathbf{U}$ *and inside its domain.*

2. *The average time* τ *needed to travel the path from* $\mathbf{x}$ *to* $\mathbf{U}$ *is not infinite if* $\mathbf{x}$ *is a member of the space* $\mathbb{R}^d \backslash \mathbf{U}$. *Furthermore, the number* $\sup_{x \in K} E^x \tau$ *is finite for any compact subset* $K \subset \mathbb{R}^n$. *Furthermore, if* $f(\cdot)$ *is an integrable function concerning the measure* $\cdot$.

Then, the Markov process $\mathcal{F}(t)$ *admits a unique ergodic stationary distribution* $\pi(\cdot)$, *and*

$$\mathbb{P}\left\{\lim_{T\to\infty} \frac{1}{T} \int_0^T f(X(t))dt = \int_{\mathbb{R}^d} f(x)\pi(dx)\right\} = 1, \text{ for all } x \in \mathbb{R}^d.$$

Define the parameter

$$\mathbb{R}_0^s = \frac{\xi\beta_1\eta\gamma}{\left(\mu + \frac{\lambda_1^2}{2}\right)\left(\mu + \gamma + \frac{\lambda_2^2}{2}\right)\left(\mu + \frac{\lambda_3^2}{2}\right)\left(\delta + \frac{\lambda_4^2}{2}\right)}. \tag{5.2}$$

Theorem 5.2. *Let* $1 < \mathbb{R}_0^s$ *and* $\mu - \frac{\lambda_1^2 \vee \lambda_2^2 \vee \lambda_3^2 \vee \lambda_4^2}{2} > 0$, *then for* $(\mathcal{S}, \mathcal{I}, \mathcal{R}, \mathcal{B})(0) \in \mathbb{R}_+^4$, *(2.5) has a unique distribution* π.

Proof. First, we must confirm that Lemma 5.1's conditions (1) and (2) are true. In order to derive result (1), take into account the diffusion matrix:

$$\Upsilon = \begin{pmatrix} \lambda_1^2\mathcal{S}^2 & 0 & 0 & 0 \\ 0 & \lambda_2^2\mathcal{I}^2 & 0 & 0 \\ 0 & 0 & \lambda_3^2\mathcal{R}^2 & 0 \\ 0 & 0 & 0 & \lambda_4^2\mathcal{B}^2 \end{pmatrix}.$$

A proof is given for result (1) of Lemma 5.1, which states that the matrix Υ is positive-definite on any compact subset of $\mathbb{R}_+^4$.

In addition to the above, our main objective is to derive property (2). Let C^2-operator $\mathcal{V} : \mathbb{R}_+^4 \to \mathbb{R}$ as given:

$$\begin{aligned} \mathcal{V}(\mathcal{S}, \mathcal{I}, \mathcal{R}, \mathcal{B}) = & \left(- \ln \mathcal{S} - c_1 \ln \mathcal{I} - c_2 \ln \mathcal{R} - c_3 \ln \mathcal{B} + \xi \int_0^t I(s)ds \right) \\ & - \ln \mathcal{S} + \zeta \int_t^t \mathcal{I}(s)ds - \ln \mathcal{R} - \ln \mathcal{B} + \frac{1}{\rho+1}(\mathcal{S} + \mathcal{I} + \mathcal{R} + \mathcal{B})^{\rho+1} \\ & = \mathcal{V}_1 + \mathcal{V}_2 + \mathcal{V}_3 + \mathcal{V}_4 + \mathcal{V}_5, \end{aligned} \tag{5.3}$$

where c_1, c_2 and c_2 are positive constant, will define later. Note that $\mathcal{V}(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})$ is not only defines on each point, but also goes to $+\infty$ as $(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})$ goes to the limit of $\mathbb{R}^4_+$ and $||(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})|| \to \infty$.

Thus, we have a tiny point in the domain of $\mathbb{R}^4_+$, denoted as $(\mathcal{S}(0),\mathcal{I}(0),\mathcal{R}(0),\mathcal{B}(0))$. A C^2- operator $\widetilde{V} : \mathbb{R}^4_+ \to \mathbb{R}_+$ is also taken as follows:

$$\begin{aligned}\widetilde{V}(S,\mathcal{I},\mathcal{R},\mathcal{B}) = &\bigg(- c_1 \ln \mathcal{S} - c_2 \ln \mathcal{I} - c_3 \ln \mathcal{R} - c_4 \ln \mathcal{B} + \xi \int_0^t I(s)ds \bigg) - \ln \mathcal{S} \\ &- \ln \mathcal{S} + \zeta \int_t^t \mathcal{I}(s)ds - \ln \mathcal{R} - \ln \mathcal{B} + \frac{1}{\rho+1}(\mathcal{S}+\mathcal{I}+\mathcal{R}+\mathcal{B})^{\rho+1} \\ &- \mathcal{V}(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})(0) \\ &:= \sum_{i=1}^{5} \mathcal{V}_i - \mathcal{V}(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})(0),\end{aligned} \tag{5.4}$$

here c_4 is positive constant and will define later, also $(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B}) \in (\frac{1}{n},n) \times (\frac{1}{n},n) \times (\frac{1}{n},n) \times (\frac{1}{n},n)$ and $n > 1$ is a so larger integer,

$$\begin{aligned}\mathcal{V}_1 &= -c_1 \ln \mathcal{S} - c_2 \ln \mathcal{I} - c_3 \ln \mathcal{R} - c_4 \ln \mathcal{B} + \xi \int_0^t \mathcal{I}(s)ds, \\ \mathcal{V}_2 &= - \ln \mathcal{S} + \zeta \int_t^t \mathcal{I}(s)ds, \\ \mathcal{V}_3 &= - \ln \mathcal{I}, \\ \mathcal{V}_4 &= - \ln \mathcal{R}, \\ \mathcal{V}_5 &= \frac{1}{\rho+1}(\mathcal{S}+\mathcal{I}+\mathcal{R}+\mathcal{B})^{\rho+1},\end{aligned} \tag{5.5}$$

$\rho > 1$, fulfilling $\mu - \frac{\rho}{2}(\lambda_1^2 \vee \lambda_2^2 \vee \lambda_3^2 \vee \lambda_4^2) > 0$,

$$\begin{aligned}\mathcal{A} = \sup_{(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})\in\mathbb{R}^4_+} \bigg(&- \frac{1}{4}\bigg[\mu - \frac{\rho}{2}(\lambda_1^2 \vee \lambda_2^2 \vee \lambda_3^2 \vee \lambda_4^2)\bigg] I^{\rho+1} \\ &2\mu + \mathcal{N} + \hat{\mathcal{B}} + \frac{\lambda_1^2}{2} + \frac{\lambda_2^2}{2} + \frac{\lambda_3^2}{2}\bigg).\end{aligned} \tag{5.6}$$

and

$$\begin{aligned}\hat{\mathcal{B}} = \sup_{(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})\in\mathbb{R}^4_+} &\bigg\{ A(\mathcal{S}+\mathcal{I}+\mathcal{R}+\mathcal{B})^{\rho} - \frac{1}{2}\bigg[\mu - \frac{\rho}{2}(\lambda_1^2 \vee \lambda_2^2 \vee \lambda_3^2 \vee \lambda_4^2)\bigg] \\ &\times (\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{Q})^{\rho+1}\bigg\} < \infty.\end{aligned} \tag{5.7}$$

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

$$
\begin{aligned}
L\mathcal{V}_1 &= -\frac{c_1\xi}{\mathcal{S}} + \frac{c_1\beta_1\mathcal{B}}{\mathcal{N}} + \frac{c_1\beta_2\mathcal{I}}{\mathcal{N}} + c_1\mu + \frac{c_1\lambda_1^2}{2} - \frac{c_2\beta_1\mathcal{B}\mathcal{S}}{\mathcal{I}\mathcal{N}} - \frac{c_2\beta_2\mathcal{S}}{\mathcal{N}} + c_2(\mu+\gamma) + \frac{c_2\lambda_2^2}{2} \\
&- \frac{c_3\gamma\mathcal{I}}{\mathcal{R}} + c_3\mu + \frac{c_3\lambda_1^2}{2} - \frac{c_4\eta\mathcal{I}}{\mathcal{B}} + \frac{c_4\lambda_1^2}{2} + c_4\delta + \xi\mathcal{I}(0) - \xi\mathcal{I}(t) \\
&\leq -4\sqrt[4]{\frac{c_1\xi}{\mathcal{S}} \times \frac{c_2\beta_1\mathcal{B}\mathcal{S}}{\mathcal{I}\mathcal{N}} \times \frac{c_3\gamma\mathcal{I}}{\mathcal{R}} \times \frac{c_4\eta\mathcal{I}}{\mathcal{B}}} + c_1\left(\mu + \frac{\lambda_1^2}{2}\right) + c_2\left(\mu+\gamma+\frac{\lambda_2^2}{2}\right) \\
&+ c_3\left(\mu + \frac{\lambda_3^2}{2}\right) + c_4\left(\delta + \frac{\lambda_4^2}{2}\right) + \xi - \xi\mathcal{I} + \frac{c_1\beta_1\mathcal{B}}{\mathcal{N}} + \frac{c_1\beta_2\mathcal{I}}{\mathcal{N}} \\
&\leq -4\sqrt[4]{\xi\beta_1\eta\gamma c_1c_2c_3c_4} + \xi + c_1\left(\mu + \frac{\lambda_1^2}{2}\right) + c_2\left(\mu+\gamma+\frac{\lambda_2^2}{2}\right) \\
&+ c_3\left(\mu + \frac{\lambda_3^2}{2}\right) + c_4\left(\delta + \frac{\lambda_4^2}{2}\right) - \xi\mathcal{I} + \frac{c_1\beta_1\mathcal{B}}{\mathcal{N}} + \frac{c_1\beta_2\mathcal{I}}{\mathcal{N}} \\
&\leq -4\xi\sqrt[4]{\frac{\xi\beta_1\eta\gamma}{\left(\mu+\frac{\lambda_1^2}{2}\right)\left(\mu+\gamma+\frac{\lambda_2^2}{2}\right)\left(\mu+\frac{\lambda_3^2}{2}\right)\left(\delta+\frac{\lambda_4^2}{2}\right)}} - 1 - \xi\mathcal{I} + \frac{c_1\beta_1\mathcal{B}}{\mathcal{N}} + \frac{c_1\beta_2\mathcal{I}}{\mathcal{N}} \\
&\leq -4\xi(\sqrt[4]{\mathbb{R}_0^s} - 1) - \xi\mathcal{I} + \frac{c_1\beta_1\mathcal{B}}{\mathcal{N}} + \frac{c_1\beta_2\mathcal{I}}{\mathcal{N}}.
\end{aligned} \tag{5.8}
$$

Let

$$
\begin{aligned}
\xi &= c_1\left(\mu + \frac{\lambda_1^2}{2}\right), \\
\xi &= c_2\left(\mu+\gamma+\frac{\lambda_2^2}{2}\right), \\
\xi &= c_3\left(\mu + \frac{\lambda_3^2}{2}\right), \\
\xi &= c_4\left(\delta + \frac{\lambda_4^2}{2}\right).
\end{aligned} \tag{5.9}
$$

Similarly, we can get

$$
L\mathcal{V}_2 = -\frac{\xi}{\mathcal{S}} + \frac{\beta_1\mathcal{B}}{\mathcal{N}} + \frac{\beta_2\mathcal{I}}{\mathcal{N}}\frac{c_1\lambda_1^2}{2} + \mu - \xi\mathcal{I}(t) + \xi\mathcal{I}(0). \tag{5.10}
$$

$$
L\mathcal{V}_3 = -\frac{\beta_1\mathcal{B}\mathcal{S}}{\mathcal{I}\mathcal{N}} - \frac{\beta_2\mathcal{S}}{\mathcal{N}} + (\mu+\gamma)\frac{c_1\lambda_3^2}{2}. \tag{5.11}
$$

$$
L\mathcal{V}_4 = -\frac{\gamma\mathcal{I}}{\mathcal{R}} + \mu\frac{c_1\lambda_2^2}{2}. \tag{5.12}
$$

$$
\begin{aligned}
L\mathcal{V}_5 &= (\mathcal{S}+\mathcal{I}+\mathcal{R}+\mathcal{B})^{\rho}[\xi-\mu(\mathcal{S}+\mathcal{I}+\mathcal{R}+\mathcal{B}))\mathcal{I}]+\frac{\rho}{2}(\mathcal{S}+\mathcal{I}+\mathcal{R}+\mathcal{B})^{\rho-1}\\
&\times(\lambda_1^2\mathcal{S}^2\vee\lambda_2^2\mathcal{I}^2\vee\lambda_3^2\mathcal{R}^2\vee\lambda_4^2\mathcal{B}^2)\\
&\leq(\mathcal{S}+\mathcal{I}+\mathcal{R}+\mathcal{B})^{\rho}[\mu\mathcal{N}-\mu(\mathcal{S}+\mathcal{I}+\mathcal{R}+\mathcal{B})]+\frac{\rho}{2}(\mathcal{S}+\mathcal{I}+\mathcal{R}+\mathcal{B})^{\rho+1}(\lambda_1^2\vee\lambda_2^2\vee\lambda_3^2\vee\lambda_4^2)\\
&\leq\xi(\mathcal{S}+\mathcal{I}+\mathcal{R}+\mathcal{B})^{\rho}-\mathcal{S}+\mathcal{I}+\mathcal{R}+\mathcal{B})^{\rho+1}\left[\mu-\frac{\rho}{2}(\lambda_1^2\vee\lambda_2^2\vee\lambda_3^2\vee\lambda_4^2)\right]\\
&\leq\hat{\mathcal{B}}-\frac{1}{2}\left[\mu-\frac{\rho}{2}(\lambda_1^2\vee\lambda_2^2\vee\lambda_3^2\vee\lambda_4^2)\right](\mathcal{S}+\mathcal{I}+\mathcal{R}+\mathcal{B})^{\rho+1}\\
&\leq\hat{\mathcal{B}}-\frac{1}{2}\left[\mu-\frac{\rho}{2}(\lambda_1^2\vee\lambda_2^2\vee\lambda_3^2\vee\lambda_4^2)\right](\mathcal{S}^{\rho+1}+\mathcal{I}^{\rho+1}+\mathcal{R}^{\rho+1}+\mathcal{B}^{\rho+1}).
\end{aligned}
\tag{5.13}
$$

$\hat{\mathcal{B}}$ is given in Eq. (5.7). From Eq. (5.8)–Eq. (5.13), we follows

$$
\begin{aligned}
L\widetilde{\mathcal{V}} \leq{}& -4\xi(\sqrt[4]{\mathbb{R}_0^s}-1)-\xi\mathcal{I}+\frac{c_1\beta_1\mathcal{B}}{\mathcal{N}}+\frac{c_1\beta_2\mathcal{I}}{\mathcal{N}}-\frac{\xi}{\mathcal{S}}+\frac{\beta_1\mathcal{B}}{\mathcal{N}}+\frac{\beta_2\mathcal{I}}{\mathcal{N}}\frac{c_1\lambda_1^2}{2}+\mu-\xi\mathcal{I}(t)+\xi\mathcal{I}(0)\\
&-\frac{\beta_1\mathcal{B}\mathcal{S}}{\mathcal{I}\mathcal{N}}-\frac{\beta_2\mathcal{S}}{\mathcal{N}}+(\mu+\gamma)\frac{c_1\lambda_3^2}{2}-\frac{\gamma\mathcal{I}}{\mathcal{R}}+\mu\frac{c_1\lambda_2^2}{2}\\
&+\hat{\mathcal{B}}-\frac{1}{2}\left[\mu-\frac{\rho}{2}(\lambda_1^2\vee\lambda_2^2\vee\lambda_3^2\vee\lambda_4^2)\right](\mathcal{S}^{\rho+1}+\mathcal{I}^{\rho+1}+\mathcal{R}^{\rho+1}+\mathcal{B}^{\rho+1}).
\end{aligned}
\tag{5.14}
$$

For $\zeta>0$, define a bounded closed set

$$
\mathbb{D}=\left\{(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})\in\mathbb{R}_+^4:\zeta\leq\mathcal{S}\leq\frac{1}{\zeta},\zeta\leq\mathcal{I}\leq\frac{1}{\zeta},\zeta^2\leq\mathcal{R}\leq\frac{1}{\zeta^2},\zeta^3\leq\mathcal{B}\leq\frac{1}{\zeta^3}\right\}.
$$

Within the subregion $\mathbb{R}_+^4\setminus D$, the following conditions hold:

$$-\frac{\xi}{\zeta}+\mathcal{G}\leq-1, \tag{5.15}$$

$$-\xi+\mathcal{G}\leq-1, \tag{5.16}$$

$$-\xi+\zeta(1+c_3)+\mathcal{A}\leq-1, \tag{5.17}$$

$$-\frac{\gamma}{\zeta}+\mathcal{G}\leq-1, \tag{5.18}$$

$$-\frac{1}{\zeta}+\mathcal{G}\leq-1, \tag{5.19}$$

$$-\frac{1}{4}\left[\mu-\frac{\rho}{2}(\lambda_1^2\vee\lambda_2^2\vee\lambda_3^2\vee\lambda_4^2)\right]\frac{1}{\zeta^{\rho+1}}+\mathcal{G}\leq-1, \tag{5.20}$$

$$-\frac{1}{4}\left[\mu-\frac{\rho}{2}(\lambda_1^2\vee\lambda_2^2\vee\lambda_3^2\vee\lambda_4^2)\right]\frac{1}{\zeta^{2(\rho+1)}}+\mathcal{G}\leq-1, \tag{5.21}$$

$$-\frac{1}{4}\left[\mu-\frac{\rho}{2}(\lambda_1^2\vee\lambda_2^2\vee\lambda_3^2\vee\lambda_4^2)\right]\frac{1}{\zeta^{3(\rho+1)}}+\mathcal{G}\leq-1. \tag{5.22}$$

Where

$$
\mathcal{G}=\sup_{(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})\in\mathbb{R}_+^4}\left\{c_1\zeta I-\frac{1}{4}\left[\mu-\frac{\rho}{2}(\lambda_1^2\vee\lambda_2^2\vee\lambda_3^2\vee\lambda_4^2)\right]I^{\rho+1}\right]+3\mu+\gamma+\hat{\mathcal{B}}+\frac{\lambda_2^2}{2}+\frac{\lambda_1^2}{2}+\frac{\lambda_4^2}{2}\right\}. \tag{5.23}
$$

We need to show that $L\widetilde{V} \leq -1$ for any $(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}) \in \mathbb{R}^4_+ \setminus \mathbb{D}$, and $\mathbb{R}^4_+ \setminus \mathbb{D} = [\bigcup_{i=1}^{8} \mathbb{D}_i]$, where

$$\begin{aligned}
\mathbb{D}_1 &= \left\{(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B}) \in \mathbb{R}^4_+; 0 < \mathcal{S} < \zeta\right\},\\
\mathbb{D}_2 &= \left\{(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B}) \in \mathbb{R}^4_+; 0 < \mathcal{I} < \zeta\right\},\\
\mathbb{D}_3 &= \left\{(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B}) \in \mathbb{R}^4_+; 0 < \mathcal{R} < \zeta^2, \mathcal{E} \geq \xi\right\},\\
\mathbb{D}_4 &= \left\{(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B}) \in \mathbb{R}^4_+; 0 < \mathcal{B} < \zeta^3, \mathcal{I} \geq \xi^2\right\},\\
\mathbb{D}_5 &= \left\{(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B}) \in \mathbb{R}^4_+; \mathcal{S} > \frac{1}{\zeta}\right\},\\
\mathbb{D}_6 &= \left\{(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B}) \in \mathbb{R}^4_+; \mathcal{I} > \frac{1}{\zeta}\right\},\\
\mathbb{D}_7 &= \left\{(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B}) \in \mathbb{R}^4_+; \mathcal{R} > \frac{1}{\zeta^2}\right\},\\
\mathbb{D}_8 &= \left\{(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B}) \in \mathbb{R}^4_+; \mathcal{B} > \frac{1}{\zeta^3}\right\}.
\end{aligned} \tag{5.24}$$

Case 1. If $(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B}) \in \mathbb{D}_1$, then by Eq. (5.14), we get

$$\begin{aligned}
L\widetilde{V} &\leq -4\xi(\sqrt[4]{\mathbb{R}_0^s} - 1) - \xi\mathcal{I} + \frac{c_1\beta_1\mathcal{B}}{\mathcal{N}} + \frac{c_1\beta_2\mathcal{I}}{\mathcal{N}} - \frac{\xi}{\mathcal{S}} + \frac{\beta_1\mathcal{B}}{\mathcal{N}} + \frac{\beta_2\mathcal{I}}{\mathcal{N}}\frac{c_1\lambda_1^2}{2} + \mu - \xi\mathcal{I}(t) + \xi\mathcal{I}(0)\\
&\quad - \frac{\beta_1\mathcal{B}\mathcal{S}}{\mathcal{I}\mathcal{N}} - \frac{\beta_2\mathcal{S}}{\mathcal{N}} + (\mu+\gamma)\frac{c_1\lambda_3^2}{2} - \frac{\gamma\mathcal{I}}{\mathcal{R}} + \mu\frac{c_1\lambda_2^2}{2}\\
&\quad + \hat{\mathcal{B}} - \frac{1}{2}\left[\mu - \frac{\rho}{2}(\lambda_1^2 \vee \lambda_2^2 \vee \lambda_3^2 \vee \lambda_4^2)\right](\mathcal{S}^{\rho+1} + \mathcal{I}^{\rho+1} + \mathcal{R}^{\rho+1} + \mathcal{B}^{\rho+1})\\
&\leq -4\xi(\sqrt[4]{\mathbb{R}_0^s} - 1) - \xi\mathcal{I} + \frac{c_1\beta_1\mathcal{B}}{\mathcal{N}} + \frac{c_1\beta_2\mathcal{I}}{\mathcal{N}} + \frac{\beta_1\mathcal{B}}{\mathcal{N}} + \frac{\beta_2\mathcal{I}}{\mathcal{N}}\frac{c_1\lambda_1^2}{2} + \mu - \xi\mathcal{I}(t) + \xi\mathcal{I}(0)\\
&\quad + (\mu+\gamma)\frac{c_1\lambda_3^2}{2} + \mu\frac{c_1\lambda_2^2}{2} + \hat{\mathcal{B}} - \frac{1}{2}\left[\mu - \frac{\rho}{2}(\lambda_1^2 \vee \lambda_2^2 \vee \lambda_3^2 \vee \lambda_4^2)\right](\mathcal{S}^{\rho+1} + \mathcal{I}^{\rho+1} + \mathcal{R}^{\rho+1} + \mathcal{B}^{\rho+1}) - \frac{\xi}{\zeta}.
\end{aligned} \tag{5.25}$$

We can choose a sufficiently small $\zeta > 0$, by inequality (5.15), we obtain $L\widetilde{V} \leq -1$ for all $(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B}) \in \mathbb{D}_1$.

Making use of inequalities (5.16), (5.17) and (5.18), and being similar to the proof of **Case 1**, we make the conclusion that $\widetilde{V} \leq -1$ for all $(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B}) \in \mathbb{D}_2$, $(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B}) \in \mathbb{D}_3$ and $(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B}) \in \mathbb{D}_4$. **Case 2.** If $(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B}) \in \mathbb{D}_5$, then by Eq.(5.14), we get

$$
\begin{aligned}
L\widetilde{V} &\leq -4\xi(\sqrt[4]{\mathbb{R}_0^s}-1)-\xi\mathcal{I}+\frac{c_1\beta_1\mathcal{B}}{\mathcal{N}}+\frac{c_1\beta_2\mathcal{I}}{\mathcal{N}}-\frac{\xi}{\mathcal{S}}+\frac{\beta_1\mathcal{B}}{\mathcal{N}}+\frac{\beta_2\mathcal{I}}{\mathcal{N}}\frac{c_1\lambda_1^2}{2}+\mu-\xi\mathcal{I}(t)+\xi\mathcal{I}(0)\\
&-\frac{\beta_1\mathcal{B}\mathcal{S}}{\mathcal{I}\mathcal{N}}-\frac{\beta_2\mathcal{S}}{\mathcal{N}}+(\mu+\gamma)\frac{c_1\lambda_3^2}{2}-\frac{\gamma\mathcal{I}}{\mathcal{R}}+\mu\frac{c_1\lambda_2^2}{2}\\
&+\hat{\mathcal{B}}-\frac{1}{2}\left[\mu-\frac{\rho}{2}(\lambda_1^2\vee\lambda_2^2\vee\lambda_3^2\vee\lambda_4^2)\right](\mathcal{S}^{\rho+1}+\mathcal{I}^{\rho+1}+\mathcal{R}^{\rho+1}+\mathcal{B}^{\rho+1})\\
&\leq -4\xi(\sqrt[4]{\mathbb{R}_0^s}-1)-\xi\mathcal{I}+\frac{c_1\beta_1\mathcal{B}}{\mathcal{N}}+\frac{c_1\beta_2\mathcal{I}}{\mathcal{N}}+\frac{\beta_1\mathcal{B}}{\mathcal{N}}+\frac{\beta_2\mathcal{I}}{\mathcal{N}}\frac{c_1\lambda_1^2}{2}+\mu-\xi\mathcal{I}(t)+\xi\mathcal{I}(0)\\
&+(\mu+\gamma)\frac{c_1\lambda_3^2}{2}+\mu\frac{c_1\lambda_2^2}{2}+\hat{\mathcal{B}}-\frac{1}{2}\left[\mu-\frac{\rho}{2}(\lambda_1^2\vee\lambda_2^2\vee\lambda_3^2\vee\lambda_4^2)\right](\mathcal{S}^{\rho+1}+\mathcal{I}^{\rho+1}+\mathcal{R}^{\rho+1}+\mathcal{B}^{\rho+1})-\frac{\beta_2\zeta}{\zeta^2}.
\end{aligned}
\tag{5.26}
$$

We can choose a sufficiently small $\zeta^2>0$, according to inequality (5.19), we achieve that $L\widetilde{V}\leq -1$ for all $(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})\in\mathbb{D}_5$.
Moreover, as the proof of Case 2 is comparable to it, and when combined with (5.20), (5.21), and (5.22), the result $L\widetilde{V}\leq -1$ may be achieved for all $(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})\in\mathbb{D}_6$, $(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})\in\mathbb{D}_7$ and $(\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})\in\mathbb{D}_8$. Based on the conversation above, it is evident that

$$L\widetilde{V}<-\mathbf{W}<0 \text{ for all } (\mathcal{S},\mathcal{I},\mathcal{R},\mathcal{B})\in\mathbf{R}_+^4\setminus\mathbb{D}.$$

which illustrates the condition (2) of Lemma 5.1 is satisfied, and model (2.5) has a unique stationary distribution and the ergodicity holds. The proof is completed. ∎

6 Numerical scheme and simulations

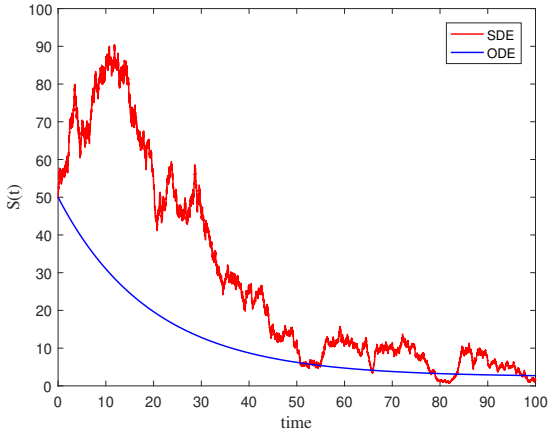
It is essential to determine appropriate parameter values in order to empirically validate the theoretical results related to system (2.5). For this purpose, two sets of parameter values are taken into consideration in addition to the initial population numbers of bacteria and humans. For all instances, $[0,100]$ is the required time interval. We possess the subsequent strategy to acquire the numerical solution of the suggested stochastic model by utilizing the higher-ordered Milstein approach:

$$
\begin{aligned}
\mathcal{S}_{i+1}&=\mathcal{S}_i+\left[\frac{\beta_1\mathcal{B}_i\mathcal{S}_i}{\mathcal{N}_i}-\frac{\beta_2\mathcal{I}_i\mathcal{S}_i}{\mathcal{N}_i}-\mu\mathcal{S}_i\right]\triangle t+\lambda_1\mathcal{S}_i\sqrt{\triangle t}\varsigma_{1,i}+\frac{\lambda_1^2}{2}\mathcal{S}_i(\varsigma_{1,i}^2-1)\triangle t,\\
\mathcal{I}_{i+1}&=\mathcal{I}_i+\left[\frac{\beta_1\mathcal{B}_i\mathcal{S}_i}{\mathcal{N}_i}+\frac{\beta_2\mathcal{I}_i\mathcal{S}_i}{\mathcal{N}_i}-(\mu+\gamma)\mathcal{I}_i\right]\triangle t+\lambda_2\mathcal{I}_i\sqrt{\triangle t}\varsigma_{2,i}+\frac{\lambda_2^2}{2}\mathcal{I}_i(\varsigma_{2,i}^2-1)\triangle t,\\
\mathcal{R}_{i+1}&=\mathcal{R}_i+\left[\gamma\mathcal{I}_i-\mu\mathcal{R}_i\right]\triangle t+\lambda_3\mathcal{R}_i\sqrt{\triangle t}\varsigma_{3,i}+\frac{\lambda_3^2}{2}\mathcal{R}_i(\varsigma_{3,i}^2-1)\triangle t,\\
\mathcal{B}_{i+1}&=\mathcal{B}_i+\left[[\eta\mathcal{I}_i-\delta\mathcal{B}_i\right]\triangle t+\lambda_4\mathcal{B}_{1,i}\sqrt{\triangle t}\varsigma_{4,i}+\frac{\lambda_4^2}{2}\mathcal{B}_{1,i}(\varsigma_{4,i}^2-1)\triangle t,
\end{aligned}
\tag{6.1}
$$

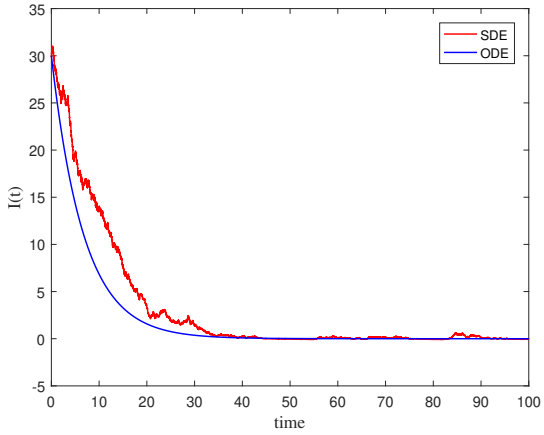
here $\varsigma_{i,j}(i=1,\cdots,4)$ represents the fundamental Gaussian stochastic variables, which have a distribution of $\mathcal{N}(0,1)$. The term Δt represents the constant time-step. For $i=1,2,3,4$, the white noise intensities are represented by the concepts $\lambda_i>0$. 1 contains the parameters, noise intensities, and beginning conditions that were used in the simulations. The deterministic system (2.1) and the perturbed system (2.5) are simulated and shown in Figure 3, taking into account the associated stochastic reproduction numbers, which are $\mathbb{R}^s<1$. As the concentrations of Vibrio cholerae go closer to zero, these graphs show what looks like the path of an exponential function with a probability of one. Theorem 4.3 is therefore numerically confirmed. Furthermore, the findings imply that the deterministic and stochastic systems agree closely. Furthermore, the systems' paths get closer to the DFE with time.

Parameter	Value	Parameter	Value
ξ	0.50	μ	0.03
β_1	0.04	β_2	0.05
γ	0.02	η	0.02
δ	0.01	λ_1	0.15
λ_2	0.20	λ_3	0.40
λ_4	0.35	$\mathcal{S}(0)$	0.50
$\mathcal{I}(0)$	0.30	$\mathcal{R}(0)$	0.05
$\mathcal{B}(0)$	0.20		

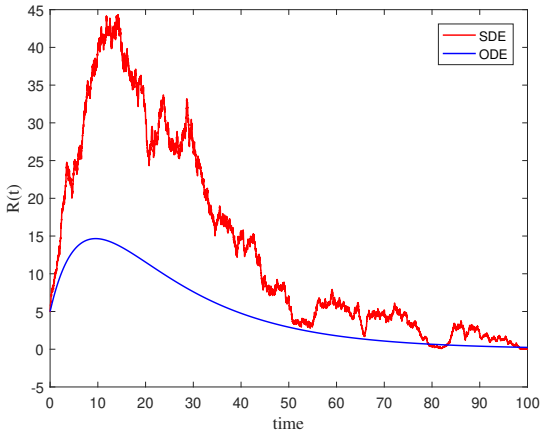
Table 1: Values of the parametric used in simulating models (2.1) and (2.5). All of the parameter values were estimated.



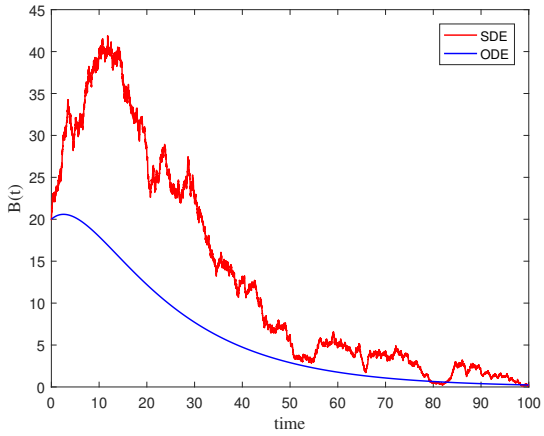
(a) $\mathcal{S}(t)-$Population



(b) $\mathcal{I}(t)-$Population



(c) $\mathcal{R}(t)-$Population



(d) $\mathcal{B}(t)-$Population

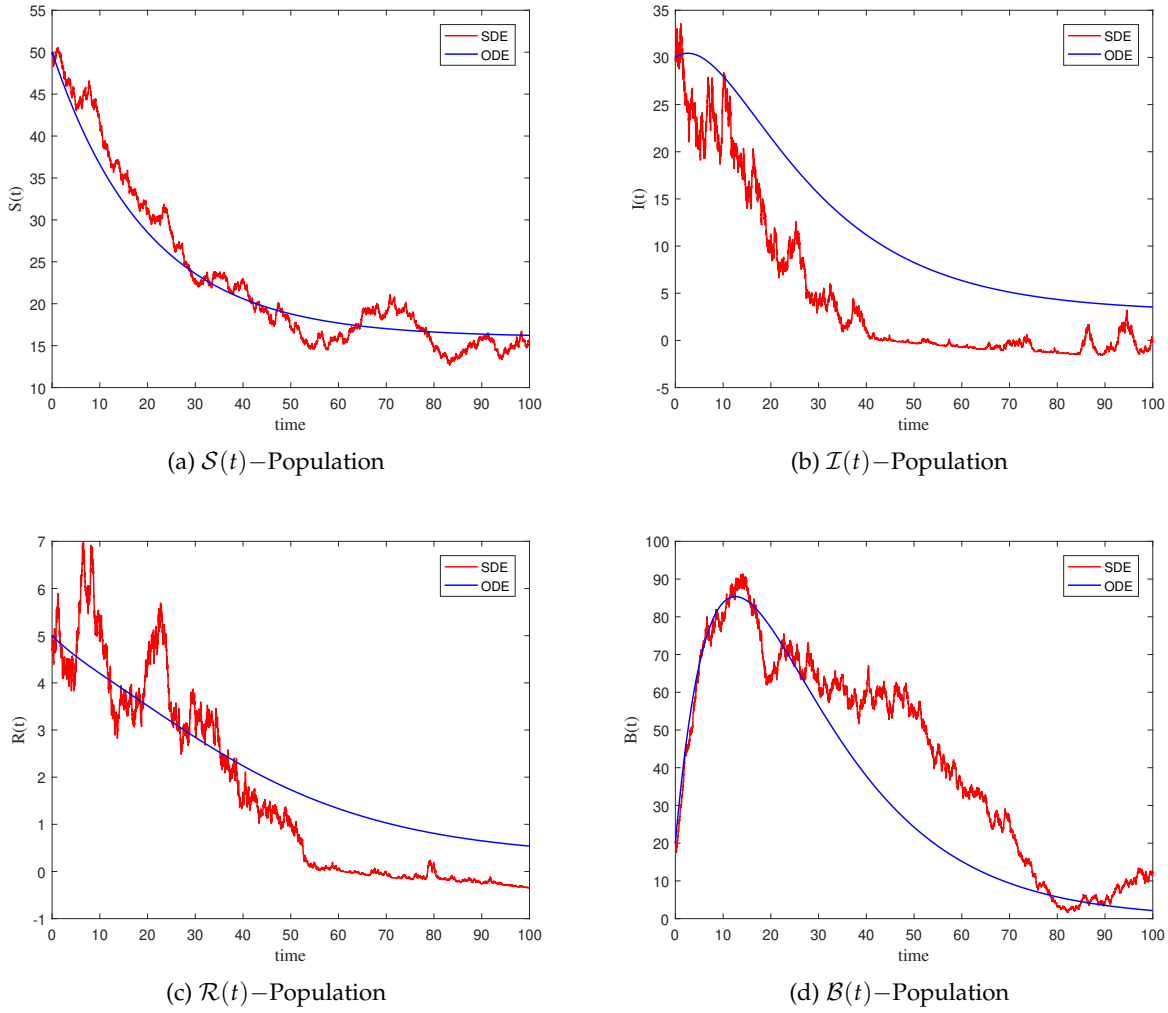
Figure 3: In scenarios where the corresponding stochastic reproduction numbers are smaller than one, simulations for the deterministic model (2.1) and the stochastic model (2.5) were runs.

Theorem 5.2 illustrates the disease's biological persistence in the context of system (2.5). We used

the noise intensities and parameter values from 2 to numerically validate the theorem's conclusion. It has been noted that the illness is still spreading among people, particularly in environments with low white noise levels. The trajectories of states $\mathcal{B}_1$ and $\mathcal{B}_2$ are non-zero in Figure 4, supporting this discovery by showing that there is some bacterial concentration present. This result is consistent with the claim in Theorem 5.2. Further investigations of the perturbed model solution's behavior reveals that the curves fluctuate around the EE point of the related deterministic model (2.1). Graphically depicted in Figure 4 under the condition $\mathbb{R}_0^s > 1$, the solutions of both systems consistently reveal non-zero bacterial concentrations, represented by $\mathcal{B}_1$ and $\mathcal{B}_2$, at every time t. This observation reinforces the implications of Theorem 5.2 for the deterministic model (2.1). Specifically, when $\mathbb{R}_0^s$ of model (2.5) exceeds unity, the corresponding solution exhibits oscillations around the endemic equilibrium. To effectively control the spread of diverse bacterial strains and the associated densities in the population under such conditions, it is crucial to design robust policies implementing strong preventive measures against various bacterial variations. According to Theorem 5.2, system (2.5) appears to possess an ergodic stationary distribution. Figure 5 confirm this.

Parameter	Value	Parameter	Value
ξ	1.30	μ	0.30
β_1	0.70	β_2	0.40
γ	0.40	η	0.20
δ	0.10	λ_1	0.35
λ_2	0.25	λ_3	0.40
λ_4	0.35	$\mathcal{S}(0)$	0.50
$\mathcal{I}(0)$	0.30	$\mathcal{R}(0)$	0.05
$\mathcal{B}(0)$	0.20		

Table 2: Values of the parametric used in simulating models (2.1) and (2.5) under the condition of $\mathbb{R}_0^s > 1$. All of the parameter values were estimated.



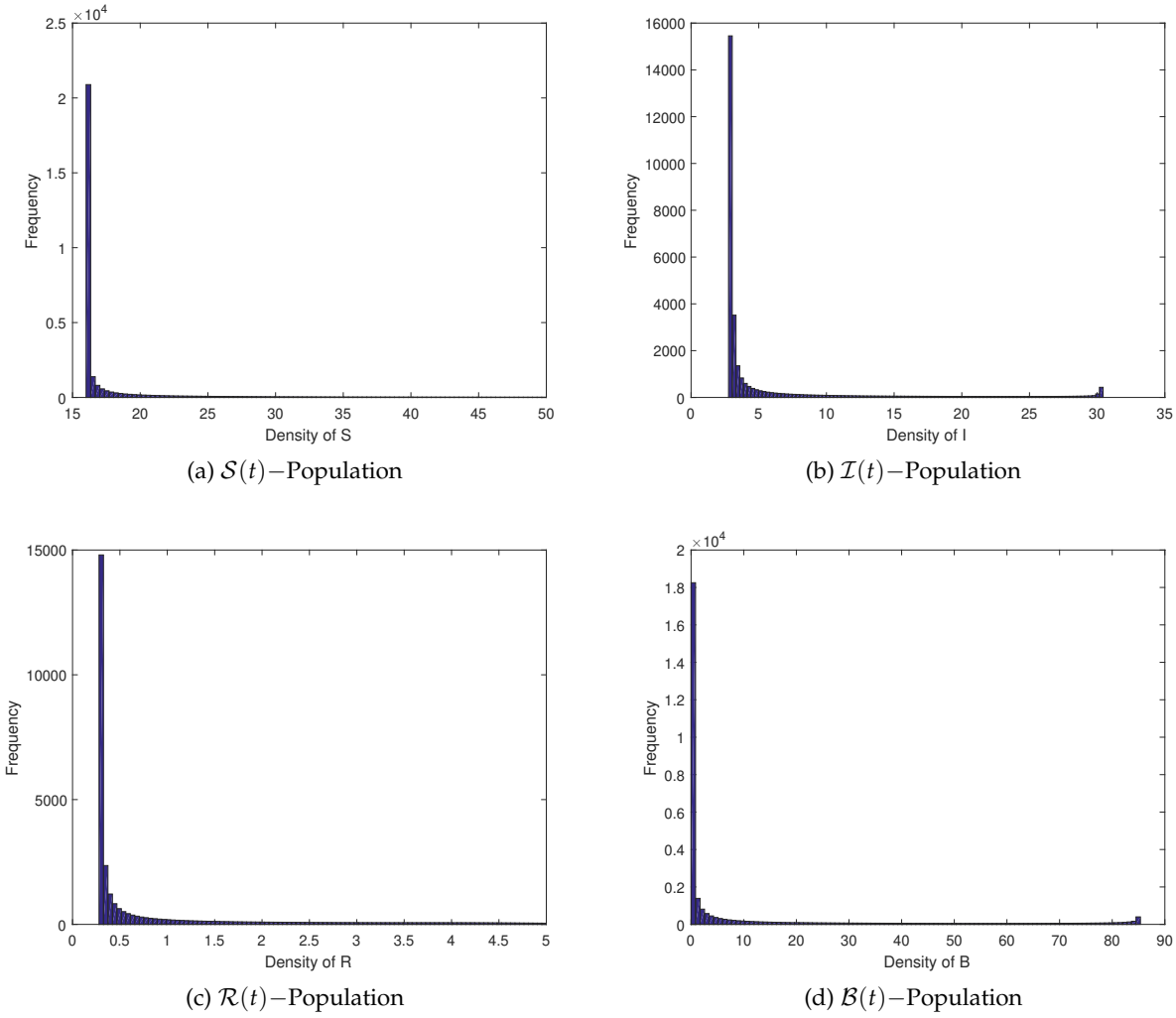
(a) $\mathcal{S}(t)-$Population

(b) $\mathcal{I}(t)-$Population

(c) $\mathcal{R}(t)-$Population

(d) $\mathcal{B}(t)-$Population

Figure 4: Profiles of the solutions for the different compartments of the deterministic model (2.1) and the stochastic model (2.5).



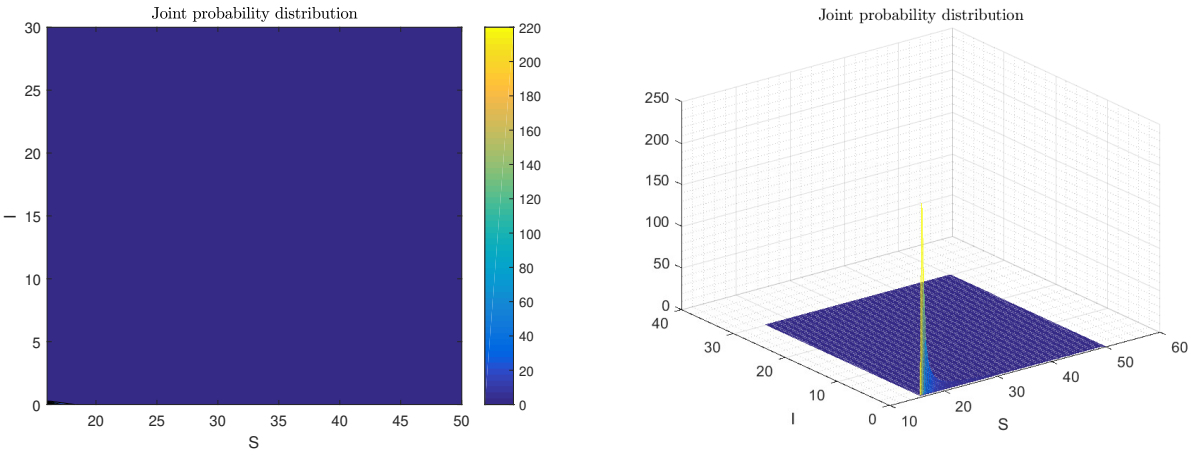
(a) $\mathcal{S}(t)-$Population (b) $\mathcal{I}(t)-$Population

(c) $\mathcal{R}(t)-$Population (d) $\mathcal{B}(t)-$Population

Figure 5: Ergodic stationary distribution of model (2.5).

6.1 Impact of noise on the shape of the probability density function

In the previous two parts of this section, we investigated the dynamical bifurcation system of the proposed system through numerical simulations, which is principally caused a switch in the sign of the threshold $\mathbb{R}_0^s$. Now, we will scout the long-rung phenomenological bifurcation (LRP-bifurcation), which mainly depends on the abrupt variation in the shape of the stationary probability density function of our model (See Figure 6).



(a) $\mathcal{S}(t)-$ and $\mathcal{I}(t)-$Populations

(b) $\mathcal{S}(t)-$ and $\mathcal{I}(t)-$Populations

Figure 6: (First part) The right panel of the figure displays the joint two-dimensional densities of individuals $\mathcal{S}$, and $\mathcal{I}$, from system (2.5) at $t = 2000$, the number of simulation experiments conducted for drawing the frequency histograms is $N = 50.000$, and corresponding to the data taken from Figure 4. Different colors represent varying density sizes. The left panel illustrates a 3D graph depicting the collective two-dimensional densities of $\mathcal{S}$, and $\mathcal{I}$.

7 Concluding remarks and future directions

To sum up, the beginning of sickness and visible symptoms following exposure to pathogen concentrations have been used to mimic epidemic cholera, which is characterized by acute diarrhea brought on by the pathogen's development. A system of stochastic differential equations is eventually derived from the model's original deterministic foundation. We also build possible equilibria of the related deterministic model and offer stability theorems, in addition to providing a biological justification for the stochastic model. Our findings demonstrate that there is a single global solution for the suggested stochastic model. We provide adequate conditions guaranteeing the mean stability of the system for $\mathbb{R}_0^s > 1$, using Lyapunov function theory. On the other hand, our results indicate that the disease has most likely been eradicated from the population when $\mathbb{R}^s < 1$. We provide graphical solutions in order to further bolster the validity of our analytical findings. A strong theoretical basis for an all-encompassing comprehension of the different chronic communicable illnesses is established by this study. We also introduce a construction technique for Lyapunov functions that may be used for the investigation of stationary distributions in models with nonlinear random disturbances.

Finding understanding how the illness spreads through food and water is particularly beneficial since it may lower the danger of cross-contamination, in contrast to the way that cholera is transmitted from person to person. The three factors of foodborne, waterborne, and human-to-human transmission must all be addressed in order to significantly reduce the danger, according to experts. The authors want to incorporate age and geographic effects, among other disease-related variables, into the model in future studies. Additionally, it is intended to include other reaction functionalities in future research.

Author contributions

Qura tul Ain: Methodology, Software, Formal Analysis, Investigation, Data Curation, Writing – Original Draft Preparation, Writing – Review & Editing.

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Conflict of interests

This work does not have any potential conflicts of interest.

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Declaration Statement of Generative AI

The author declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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