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Research article

Analysis of a stochastic epidemic model for cholera disease based on probability density function with standard incidence rate

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Abstract: Acute diarrhea caused by consuming unclean water or food is known as the epidemic cholera. A model for the epidemic cholera is formulated by considering the instants at which a person contracts the disease and the instant at which the individual exhibits symptoms after consuming the poisoned food and water. Initially, the model is formulated from the deterministic point of view, and then it is converted to a system of stochastic differential equations. In addition to the biological interpretation of the stochastic model, we proved the existence of the possible equilibria of the associated deterministic model, and accordingly, stability theorems are presented. It is demonstrated that the proposed stochastic model has a unique global solution, and adequate criteria are constructed by using the Lyapunov function theory, which guarantees that the system has persistence in the mean whenever $\mathbf{R_s^0} > 1$. For the case of $R_s < 1$, we proved that the disease will tend to be eliminated from the community. Some graphical solutions were produced in order to better validate the analytical results that were acquired. This research can offer a solid theoretical foundation for comprehensive knowledge of other chronic communicable diseases. Additionally, our approach seeks to offer a technique for creating Lyapunov functions that may be utilized to investigate the stationary distributions of models with non-linear stochastic perturbations.

Keywords: probability density function; environmental noise; threshold; persistence; extinction
Mathematics Subject Classification: 60E05, 60J65, 60K37

1. Introduction

Found in marine animals, the bacterium Vibrio cholerae infects the bowel, thus causing cholera. This bacteria has about 200 serogroups, although only two of them (O139 and O1) causes cholera

infection [1, 2]. They manage to get through and survive the stomach's gastric acid barrier before penetrating the mucous lining that covers the epithelial cells of the intestine [1, 3]. Once they have colonized the intestine, they release enterotoxins which cause the small intestine's endothelium cells to secrete more electrolytes and water [1]. John Snow demonstrated in 1854 that cholera outbreaks can be brought on by consuming infected water or food [4]. However, there are additional means of dissemination as well; for example, contact with infectious people can potentially cause infection in the vulnerable population. If these persons have a higher chance of contracting the disease, they can spread it to household members who prepare meals or use water from containers, for example; for details, [4]. A person may be infectious without or with symptoms, which may appear anywhere between a few hours and five days after getting the infection. However, the symptoms of the infection usually start within first 2–3 days [5]. The most common symptoms related to this disease are copious leg cramps, and vomiting, watery diarrhea. It is very crucial that the infected people should receive treatment as quickly as possible because if they are not treated, they can become dehydrated, develop acidosis and experience circulatory collapse. In the worst cases, this disease has a 12 to 24 hour death risk [4, 6]. According to some research and testing, a person who has recovered may be immune to the sickness for three to ten years. However, new studies indicate that the immunity can be lost after a few weeks or months [4, 7]. Due to the difficulty in getting sanitary facilities and clean water in the underdeveloped and developing nations, diarrheal diseases are the leading cause of infant and child deaths [8]. Furthermore, according to Sun et al. [9], this disease has created a serious hazard to human society, causing a significant amount of mortality and morbidity, and it has a poor system of surveillance. Therefore, it is crucial to examine the mathematical models describing the transmission mechanism of cholera in order to understand how the disease spreads and how one can control its spread.

Numerous mathematical models have been investigated to explain how cholera spreads, including [1, 4, 6–9] and the references listed therein. An SIR (susceptible-infectious-recovered) type of model was put forth in [7], and takes into account two classes of hyperinection and less-infectious bacterial concentrations. Further, the infected class is subdivided into two compartments, namely, asymptomatic and symptomatic groups. Using the techniques of sensitivity analysis, optimum control theory and numerical simulations, the authors analyzed the cost-effectiveness of a variety of controlling techniques for the two populations where the disease is assumed to be endemic. An SIR type model with a class for the vibrio bacterial concentration in the environment was taken into consideration by Wang and Modnak [10]. Three preventative interventions are included in the model: immunisation, medical care, and water sanitation. The authors provided constant values to the control parameters and the stability analysis of the equilibrium points was performed. On the basis of Pontryagin's Maximum Principle, they also analyzed a more comprehensive cholera model with time-dependent controls, demonstrating the existence of a solution to the optimal control problem and obtaining the essential optimality conditions. As control measures, the authors of [6] included a public health awareness campaign, immunization, isolation and therapy. This work also considered the bacterial concentration into the model as a separate compartment. To evaluate the potential community benefits of these strategies, the basic reproduction number was compared with the education, vaccination, and treatment induced threshold quantities and with the combined threshold parameter. The stability analysis of the fixed points was examined by using the Lyapunov functional technique.

It is strongly advised to utilize the mathematical modeling methods in order to examine the

ways in which an epidemic spreads and how to control it [11–15]. Due to their representation of the natural history of the infection, such models can strike a compromise between, biological scenario and the robustness of their data connections. A wide spectrum of cholera dynamics have been highlighted by models that have been developed thus far. The majority of models are from a deterministic point of view: however, the noises associated with the environment is always of significant interest to epidemiologists while investigating the dynamics and control of the epidemic cholera [16]. Interpersonal interactions or other aspects of the population are unpredictable, for example, the epidemic onset and its propagation. As a result, the environment's diversity and the unpredictability of the nature will have a high impact on epidemic's present and future status.

It is worthy to mention that the persistence and distribution of the bacteria are strongly related with the corresponding change in the environment. The dynamics of an infection is inherently stochastic both in terms of parameters and states, so epidemiology considers stochasticity to be a key element in epidemic modeling. The disturbances being included in the model should be positively autocorrelated, although these are naturally random. Additionally, from the associated problem, one can easily analytically obtain these fluctuations by utilizing the probability density function [17–19]. Generally, deterministic and stochastic modeling are the two main types of techniques for modeling epidemics. When modeling biological systems, models consisting of stochastic differential equations (SDEs) are given preference over deterministic models because they provide a higher level of reality [20–23]. To establish a distribution of expected outcomes, such as the number of infected people over time t, we might employ SDEs. Additionally, when simulated numerous times, a stochastic model produces different results that are more valuable than deterministic models. To characterize the dynamics of the cholera infection, a number of deterministic models have been put forth, such as [24, 25].

In the present study, we developed a stochastic epidemic model to explain the dynamics of cholera transmission, particularly its long-term behavior, in a situation of migrating populations where bacterial contamination may occur. The entire population (of both humans and bacteria) is divided into six groups in a disjoint manner. These compartments are $\mathbb{S}(t)$, $\mathbb{I}(t)$, $\mathbb{Q}(t)$, $\mathbb{R}(t)$, $\mathbb{B}_1(t)$ and $\mathbb{B}_2(t)$ respectively standing for the human being susceptible, infected, quarantine and, recovered and concentration of vibro-cholera in water and food. According to the features of the sickness and after taking into account the environmental noises, these groups are related to one another through mathematical equations. More specifically, we take into considerations the interval between the infection and the onset of cholera symptoms in an individual.

The structure of the remaining manuscript is as follows. We suggest a model for governing the dynamics of cholera in Section 2. Section 3 provides the equilibria of the model, and the threshold parameter is calculated therein. In Section 4, we provided sufficient details showing the existence of a unique positive global solution. The permanence and elimination of the infection are investigated in Sections 5 and 6. In Section 7 numerical simulations find the probability density function for the proposed model. In Section 8, theoretical findings are numerically tested and graphically displayed. Section 9 of the study includes a summary as well as suggestions for future investigations.

2. Models formulation

In this section, we will formulate a mathematical model for describing the dynamics of cholera by including the instants at which a person contracts the disease and instant at which he/she exhibits symptoms after consuming contaminated water or food. As a result, we will consider a model describing the dynamics of cholera which incorporates the Susceptible-Infectious-Quarantined-Recovered ($\mathbb{SIQR}$) classes and also takes two additional classes of cholera bacterium densities. At any time $t \geq 0$, we have stratified the entire human population $\mathbb{N}(t)$ into four sub-groups: $\mathbb{S}(t)$, $\mathbb{I}(t)$, $\mathbb{Q}(t)$ and recovered $\mathbb{R}(t)$ which respectively shows the sizes of the susceptible, infectious, quarantined and recovered individuals. Similarly, the bacteria population is divided into two compartments: $\mathbb{B}_1(t)$, and $\mathbb{B}_2(t)$, that respectively stand for the concentrations of cholera bacteria in water and food. To formulate the model, we considered the constant inflow of individuals into the susceptible class at a rate Π. The natural death rate is assumed to be $\mu > 0$, and it is uniform for each compartment. The vulnerable population can contract the cholera bacterium from both of the sources at rates $\beta_1\mathbb{B}_1(t)$ and $\beta_2\mathbb{B}_2(t)$, respectively. Here, the constants β_1 and β_2 are positive, and physically these parameters show the transmission rates of cholera bacteria via infected water and food. It is also taken into account that a recovered person can lose the immunity and could gain susceptibility at a constant rate ω. A fraction of the infected people will move to the quarantine class, and this movement is assumed constantly. They are segregated and receiving appropriate medication at rate δ during this time. Due to treatment or self-resistance to the disease, the quarantined people will recover with a rate ε. The individuals in infected and quarantined compartments will experience disease-induced mortality which are respectively α_1 and α_2. Each infected person increases the concentration of cholera germs in the water and food at the rates η_1 and η_2, respectively. Further, the biology of the problem suggests that both the concentrations of the bacteria could decrease at a constant rate d and hence be included into the model. In addition, we impose the following assumptions on the model:

A_1 : All of the parameters $\mu, \Lambda, \kappa, \beta, \eta_1, \eta_2$ and d are positive real numbers, and $\delta, \omega, \varepsilon, \alpha_1, \alpha_2$ are nonnegative.

A_2 : The mean contact in a unit time is constant, and it is denoted by c.

A_3 : Every person/entity in the entire populations has an equal chance of moving into another class and the same is the case for water and food cholera bacteria population classes $\mathbb{B}_1$ and $\mathbb{B}_2$. In other words, the exponential distribution determines the probability distribution of movements among the compartments, and the projected average time spent in a class can be calculated by taking into account the inverse of that parameter in an exponential distribution.

A_4 : It is assumed that the size and demographic structure of the population remains constant over time, and there are no new individuals entering or leaving the population permanently. This means that the spread of the disease is contained within the boundaries of the population, and there is no external influence on the epidemic dynamics.

A_5 : The recovered people require no treatment, and consequently, such people are immune to cholera. We suppose that no recoverable persons died as a result of cholera. In this context, it is reasonable to assume that both recovered and susceptible persons die at the natural mortality rate

μ. This reflects that only untreated infectious individuals without symptoms or who are receiving treatment can die from cholera.

The mathematical model that results from the above-mentioned suppositions is as follows:

$$\begin{aligned}
\frac{d\mathbb{S}}{dt} &= \Pi - \frac{\beta_1 \mathbb{B}_1(t)\mathbb{S}(t)}{\mathbb{N}(t)} - \frac{\beta_2 \mathbb{B}_2(t)\mathbb{S}(t)}{\mathbb{N}(t)} - \mu\mathbb{S}(t) + \omega\mathbb{R}(t),\\
\frac{d\mathbb{I}}{dt} &= \frac{\beta_1 \mathbb{B}_1(t)\mathbb{S}(t)}{\mathbb{N}(t)} + \frac{\beta_2 \mathbb{B}_2(t)\mathbb{S}(t)}{\mathbb{N}(t)} - (\alpha_1 + \mu + \delta)\mathbb{I}(t),\\
\frac{d\mathbb{Q}}{dt} &= \delta\mathbb{I}(t) - (\alpha_2 + \mu + \varepsilon)\mathbb{Q}(t),\\
\frac{d\mathbb{R}}{dt} &= \varepsilon\mathbb{Q}(t) - (\omega + \mu)\mathbb{R}(t),\\
\frac{d\mathbb{B}_1}{dt} &= \eta_1\mathbb{I}(t) - d\mathbb{B}_1(t),\\
\frac{d\mathbb{B}_2}{dt} &= \eta_2\mathbb{I}(t) - d\mathbb{B}_2(t).
\end{aligned} \tag{2.1}$$

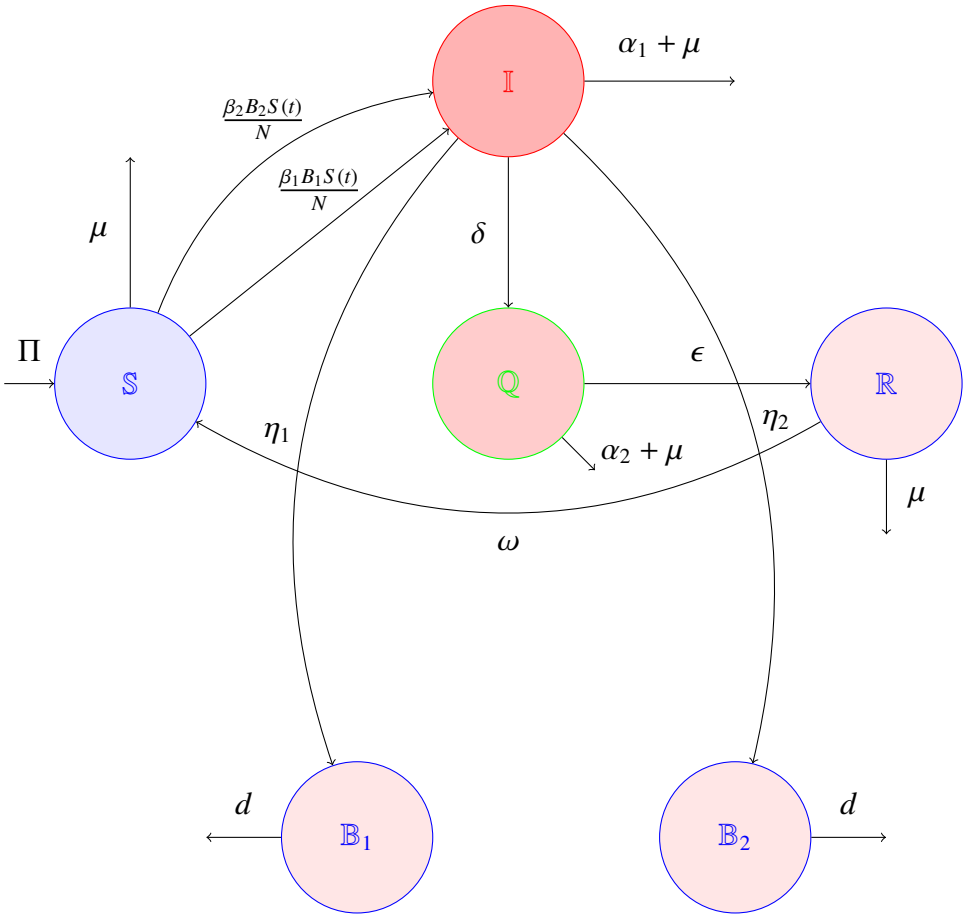


Figure 1. A complete description of the flowcharts of cholera dynamics governed by model (2.1).

In order to consider the stochastic fluctuations in system (2.1), we will take into account the functions $\mathbb{W}_i(t)$ for $i = 1, \cdots, 6$ with $\mathbb{W}_i(0) = 0$ in the respective classes. Biological interpretations of these functions are the inclusion of the fluctuations due to the environment and are called the Brownian motions. The intensity associated with each noise is described by γ_i for $i = 1, \cdots, 6$. By considering these fluctuations, the proposed stochastic model becomes

$$\begin{aligned}
d\mathbb{S} &= \left[\Pi - \frac{\beta_1\mathbb{B}_1(t)\mathbb{S}(t)}{\mathbb{N}(t)} - \frac{\beta_2\mathbb{B}_2(t)\mathbb{S}(t)}{\mathbb{N}(t)} - \mu\mathbb{S}(t) + \omega\mathbb{R}(t)\right]dt + \gamma_1\mathbb{S}(t)d\mathbb{W}_1(t),\\
d\mathbb{I} &= \left[\frac{\beta_1\mathbb{B}_1(t)\mathbb{S}(t)}{\mathbb{N}(t)} + \frac{\beta_2\mathbb{B}_2(t)\mathbb{S}(t)}{\mathbb{N}(t)} - (\alpha_1 + \mu + \delta)\mathbb{I}(t)\right]dt + \gamma_2\mathbb{I}(t)d\mathbb{W}_2(t),\\
d\mathbb{Q} &= \left[\delta\mathbb{I}(t) - (\alpha_2 + \mu + \varepsilon)\mathbb{Q}(t)\right]dt + \gamma_3\mathbb{Q}(t)d\mathbb{W}_3(t),\\
d\mathbb{R} &= \left[\varepsilon\mathbb{Q}(t) - (\mu + \omega)\mathbb{R}(t)\right]dt + \gamma_4\mathbb{R}(t)d\mathbb{W}_4(t),\\
d\mathbb{B}_1 &= \left[- d\mathbb{B}_1(t) + \eta_1\mathbb{I}(t)\right]dt + \gamma_5\mathbb{B}_1(t)d\mathbb{W}_5(t),\\
d\mathbb{B}_2 &= \left[\eta_2\mathbb{I}(t) - d\mathbb{B}_2(t)\right]dt + \gamma_6\mathbb{B}_2(t)d\mathbb{W}_6(t).
\end{aligned} \tag{2.2}$$

In this work, we intend to use model (2.2) and find possible answers to the questions listed below:

Q_1 : Is random noise affecting the dynamic behavior of the epidemic cholera?

Q_2 : Does polluted water plays a significant role in the spread of the disease?

Q_3 : Does food tainted with the Vibrio-cholerae effect the dynamics of the underlying disease?

Q_4 : What standard is used to determine whether the model has undergone extinction?

Q_5 : What standard is used to determine whether the infection is persistent?

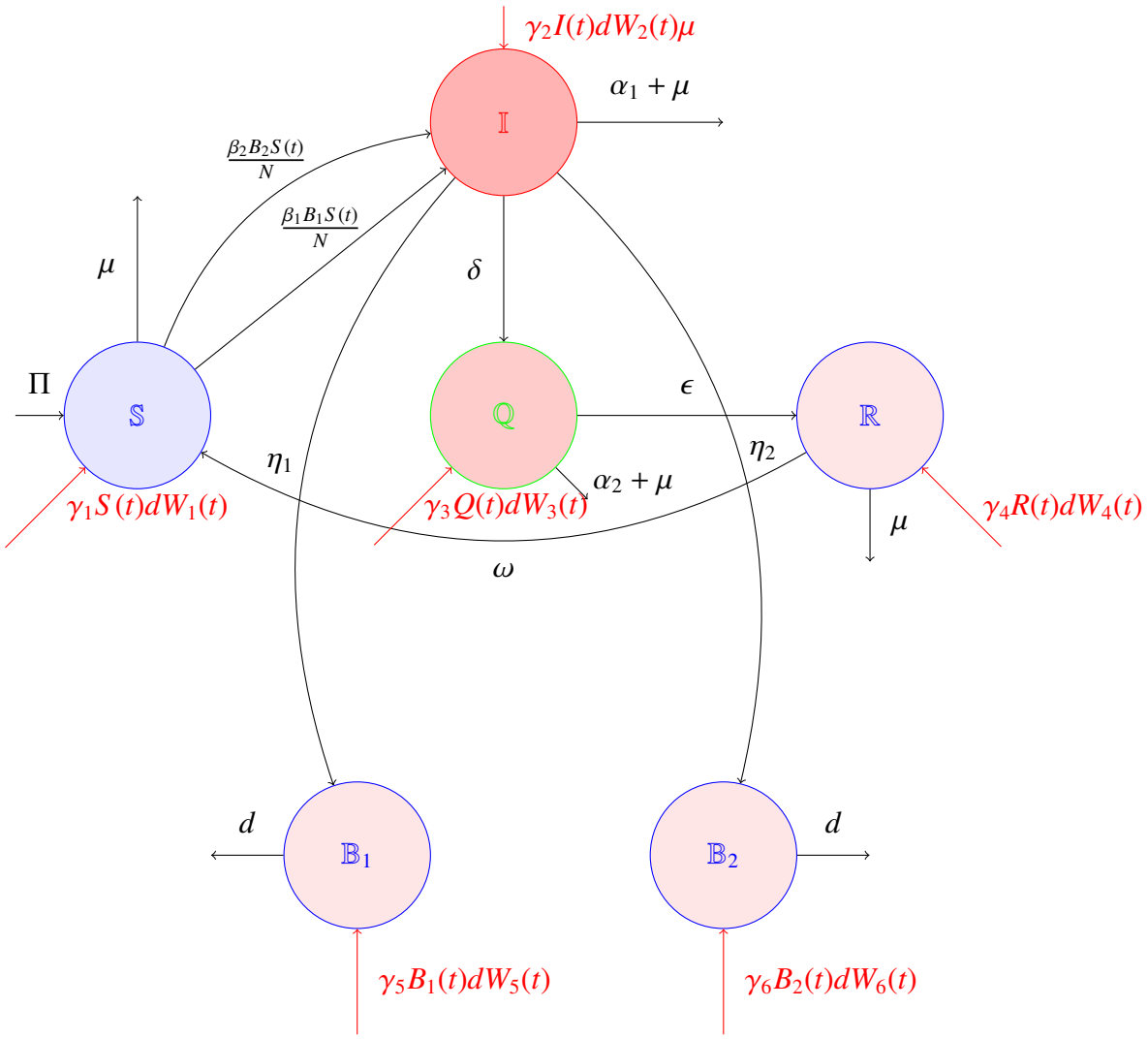


Figure 2. The detailed flowcharts of cholera disease transmission of system (2.2).

3. Deterministic model analysis

First, we show that the model (2.1) makes sense from a biological perspective since, under non-negative initial conditions, the solutions of (2.1) are non-negative. Second, we provide the formulations for the equilibrium points where disease is absent and where it is endemic, as well as the expression for the threshold parameter. The model is then linearized, which enables us to derive several significant findings that are required for the local study of the steady states. In the continuation, we will take into

account the notations below:

$$\begin{aligned} a_1 &= \delta + \alpha_1 + \mu, \\ a_2 &= \varepsilon + \alpha_2 + \mu, \\ a_3 &= \omega + \mu, \\ \rho_1 &= \Pi\eta_1 a_2 a_3 + d(a_1 a_2 a_3 - \delta\varepsilon), \\ \rho_2 &= \Pi\eta_2 a_2 a_3 + d(a_1 a_2 a_3 - \delta\varepsilon), \\ \bar{D} &= a_1 a_2 a_3 \mu + \beta(a_1 a_2 a_3 - \delta\varepsilon\omega), \\ A &= a_1 a_2 a_3, \\ \tilde{A} &= a_1 a_2 a_3 - \delta\varepsilon\omega. \end{aligned} \tag{3.1}$$

3.1. Equilibrium points and the basic reproduction number

A process described by a system of ordinary differential equation (ODEs) is said to be in the equilibrium state if there is no change in the system with respect to the independent variable. Since, we are dealing with a dynamical system in mathematical epidemiology, the solution of the model which is time independent is said to be the equilibrium state of the system. Upon solving the above problem, we will obtain the equilibria of the problem. Hence, by following the studies [11,22,26], a disease-free equilibrium (DFE) for the proposed $\mathbb{SIQRB}$ Cholera model can be written in the form of

$$\begin{aligned} \Pi - \frac{\beta_1 \mathbb{B}_1(t)\mathbb{S}(t)}{\mathbb{N}(t)} - \frac{\beta_2 \mathbb{B}_2(t)\mathbb{S}(t)}{\mathbb{N}(t)} - \mu\mathbb{S}(t) + \omega\mathbb{R}(t) &= 0, \\ \frac{\beta_1 \mathbb{B}_1(t)\mathbb{S}(t)}{\mathbb{N}(t)} + \frac{\beta_2 \mathbb{B}_2(t)\mathbb{S}(t)}{\mathbb{N}(t)} - (\alpha_1 + \mu + \delta)\mathbb{I}(t) &= 0, \\ \delta\mathbb{I}(t) - (\alpha_2 + \mu + \varepsilon)\mathbb{Q}(t) &= 0, \\ \varepsilon\mathbb{Q}(t) - (\omega + \mu)\mathbb{R}(t) &= 0, \\ \eta_1\mathbb{I}(t) - d\mathbb{B}_1(t) &= 0, \\ \eta_2\mathbb{I}(t) - d\mathbb{B}_2(t) &= 0. \end{aligned} \tag{3.2}$$

After some basic mathematical calculation we can easily get the following DFE points for the proposed (2.1). Hence, the equilibrium state of the proposed deterministic model is of the form

$$\mathbb{E}^0 = \left(\mathbb{S}^0, \mathbb{I}^0, \mathbb{Q}^0, \mathbb{R}^0, \mathbb{B}_1^0, \mathbb{B}_2^0\right) = \left(\frac{\Pi}{\mu}, 0, 0, 0, 0, 0\right). \tag{3.3}$$

Now, consider that $\mathbb{F}_i(t)$ is the rate of appearance of new infections in the compartment associated with index i, $\mathbb{V}_i^+(t)$ is the rate of transfer of individuals into the compartment associated with index i by all other means, and $\mathbb{V}_i^-(t)$ is the rate of transfer of individuals out of the compartment associated with index i. In this way, the matrices $\mathbb{F}(t)$, $\mathbb{V}^+(t)$ and $\mathbb{V}^-(t)$, associated with model (2.1), are given by

$$\mathbb{F}(t) = \begin{bmatrix} 0 \\ \frac{\beta_1 \mathbb{B}_1(t)\mathbb{S}(t)}{\mathbb{N}(t)} + \frac{\beta_2 \mathbb{B}_2(t)\mathbb{S}(t)}{\mathbb{N}(t)} \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \quad \mathbb{V}^+(t) = \begin{bmatrix} \Pi + \omega\mathbb{R}(t) \\ 0 \\ \delta\mathbb{I}(t) \\ \varepsilon\mathbb{Q}(t) \\ \eta_1\mathbb{I}(t) \\ \eta_2\mathbb{I}(t) \end{bmatrix},$$

and

$$\mathbb{V}^{-}(t) = \begin{bmatrix} \frac{\beta_1 \mathbb{B}_1(t)\mathbb{S}(t)}{\mathbb{N}(t)} + \frac{\beta_2 \mathbb{B}_2(t)\mathbb{S}(t)}{\mathbb{N}(t)} + \mu\mathbb{S}(t) \\ a_1\mathbb{I}(t) \\ a_2\mathbb{Q}(t) \\ a_3\mathbb{R}(t) \\ d\mathbb{B}_1(t) \\ d\mathbb{B}_2(t) \end{bmatrix}.$$

Therefore, considering $\mathbb{V}(t) = \mathbb{V}^{-}(t) - \mathbb{V}^{+}(t)$, the Jacobian matrices of $\mathbb{F}(t)$ and of $\mathbb{V}(t)$ are, respectively, and then put the disease-free equilibrium values of $\mathbb{E}^0$ in $\mathbb{F}(t)$ and of $\mathbb{V}(t)$, given by

$$F_{\mathbb{E}^0} == \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_1 \mathbb{B}_1}{\mathbb{N}} + \frac{\beta_2 \mathbb{B}_2}{\mathbb{N}} & 0 & 0 & 0 & \frac{\beta_1 \mathbb{S}^0}{\mathbb{N}} & \frac{\beta_2 \mathbb{S}^0}{\mathbb{N}} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

and

$$V_{\mathbb{E}^0} = \begin{bmatrix} \frac{\beta_1 \mathbb{B}_1}{\mathbb{N}(t)} + \frac{\beta_2 \mathbb{B}_2}{\mathbb{N}(t)} + \mu & 0 & 0 & -\omega & \frac{\beta_1 \mathbb{S}^0}{\mathbb{N}} & \frac{\beta_2 \mathbb{S}^0}{\mathbb{N}(t)} \\ 0 & a_1 & 0 & 0 & 0 & 0 \\ 0 & -\delta & a_2 & 0 & 0 & 0 \\ 0 & 0 & -\varepsilon & a_3 & 0 & 0 \\ 0 & -\eta_1 & 0 & 0 & d & 0 \\ 0 & -\eta_2 & 0 & 0 & 0 & d \end{bmatrix}.$$

The basic reproduction number of model (2.1) is then given by

$$R_0 = \rho\left(F_{\mathbb{E}^0} V_{\mathbb{E}^0}^{-1}\right).$$

The following expression defines the basic reproduction number for the proposed problem

$$R_0 = R_1 + R_2, \tag{3.4}$$

where

$$R_1 = \frac{\beta_1 \Pi \eta_1}{\mu d(\delta + \alpha_1 + \mu)}, \qquad R_2 = \frac{\beta_2 \Pi \eta_2}{\mu d(\delta + \alpha_1 + \mu)}. \tag{3.5}$$

Moreover, when $R_0 > 1$, then by the use of Eqs (3.1) and (3.4) we determine that there an endemic equilibrium given by

$$E^* = (\mathbb{S}^*, \mathbb{I}^*, \mathbb{Q}^*, \mathbb{R}^*, \mathbb{B}_1^*, \mathbb{B}_2^*), \tag{3.6}$$

where

$$\begin{aligned}
\mathbb{S}^* &= \frac{a_1\rho}{\eta_1\eta_2\bar{D}},\\
\mathbb{I}^* &= \frac{(\beta_1+\beta_2)\Pi a_2a_3\,(R_0-1)}{R_0\bar{D}},\\
\mathbb{Q}^* &= \frac{(\beta_1+\beta_2)\Pi a_3\delta\,(R_0-1)}{R_0\bar{D}},\\
\mathbb{R}^* &= \frac{(\beta_1+\beta_2)\Pi\delta\varepsilon\,(R_0-1)}{R_0\bar{D}},\\
\mathbb{B}_1^* &= \frac{(\beta_1+\beta_2)\Pi\eta_1a_2a_3\,(R_0-1)}{R_0\bar{D}d},\\
\mathbb{B}_2^* &= \frac{(\beta_1+\beta_2)\Pi\eta_2a_2a_3\,(R_0-1)}{R_0\bar{D}d}.
\end{aligned}$$

4. Stochastic model analysis

In this part of the study, the stochastic system is studied for extinction and ergodic stationary distribution, and the existence and uniqueness of solution, are all investigated.

4.1. Positive global solution of the model

While studying the dynamic behaviors of a stochastic system, it is the first crucial issue is to check the model for the existence of a global solution. Further, the nature of the long term behavior and positivity of the solution are of major significance for a model that describes population dynamics. This part of the work mainly deals with the existence of a unique non-negative global solution to the stochastic model (2.2). It is necessary that the coefficients of the model's equation must be locally Lipschitz and must satisfy the condition of linear growth in order to prove that a stochastic model has a unique global solution.

Theorem 1. *A solution* $(\mathbb{S}(t),\mathbb{I}(t),\mathbb{Q}(t),\mathbb{R}(t),\mathbb{B}_1(t),\mathbb{B}_2(t))$ *of model (*2.2*) on* $t\geq 0$ *for any initial value* $(\mathbb{S}(0),\mathbb{I}(0),\mathbb{Q}(0),\mathbb{R}(0),\mathbb{B}_1(0),\mathbb{B}_2(0))\in\mathbb{R}^6_+$*, and the solution will remain in* $\mathbb{R}^6_+$ *with probability one, i.e., for all* $t\geq 0$*, namely,* $(\mathbb{S}(t),\mathbb{I}(t),\mathbb{Q}(t),\mathbb{R}(t),\mathbb{B}_1(t),\mathbb{B}_2(t))\in\mathbb{R}^6_+$.

Proof: For the non-negative initial values of the state variables, one can easily notice that the coefficients of the model are locally Lipschitz. This assures that for any time t, the proposed problem has a local unique solution in the interval $[0,\tau_e)$. The term τ_e stands for the explosion time, and for more details, readers are suggested to visit [27,28]. It is sufficient to show that $\tau_e=\infty$ for proving that actually such solution is global. To prove this, let us consider a large enough positive real number k_0 in such a way that each solution of the problem lies within the interval $[\frac{1}{k_0},k_0]$. Further, for $k\geq k_0$ let

$$\begin{aligned}
\tau_k = inf\{t\in[0,\tau_e) : \frac{1}{k}\geq \min\{\mathbb{S}(t),\mathbb{I}(t),\mathbb{Q}(t),\mathbb{R}(t),\mathbb{B}_1(t),\mathbb{B}_2(t)\},\ \text{or}\\
k\leq \max\{\mathbb{S}(t),\mathbb{I}(t),\mathbb{Q}(t),\mathbb{R}(t),\mathbb{B}(t),\mathbb{B}_1(t),\mathbb{B}_2(t)\}.
\end{aligned}\tag{4.1}$$

Throughout this paper, $\inf \phi = \infty$ whenever ϕ denotes an empty set. According to the definition, τ_k is increasing as $k \to \infty$. Thus, by assuming the limit of τ_k to be τ_∞, $\tau_\infty \leq \tau_e$ almost surely (a.s.). In other word, we need to show that $\tau_\infty = \infty$ a.s. If this assertion is false, then there exists a pair of constants $T > 0$ and $\epsilon \in (0, 1)$ such that

$$\epsilon < P\{\tau_\infty \leq T\}. \tag{4.2}$$

Thus, for an integer $k_0 \leq k_1$, we have

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

Let's define a Lyapunov function of the following form to move forward:

$$\begin{aligned} \mathbb{V} = &(\mathbb{S} - c_1 - c_1 \log \frac{\mathbb{S}}{c_1}) + (\mathbb{I} - \log \mathbb{I} - 1) + (\mathbb{Q} - \log \mathbb{Q} - 1) + (\mathbb{R} - \log \mathbb{R} - 1) \\ &+ (\mathbb{B}_1 - \log \mathbb{B}_1 - 1) + (\mathbb{B}_2 - \log \mathbb{B}_2 - 1), \end{aligned} \tag{4.3}$$

here the parameter c_1 will be determined at later stages. Making use of Itoˆs formula, we obtain

$$\begin{aligned} d\mathbb{V}(\mathbb{S}, \mathbb{I}, \mathbb{Q}, \mathbb{R}, \mathbb{B}_1, B_2) = &L\mathbb{V}(\mathbb{S}, \mathbb{I}, \mathbb{Q}, \mathbb{R}, \mathbb{B}_1, \mathbb{B}_2)dt + \gamma_1(\mathbb{S} - c_1)d\mathbb{W}_1(t) + \gamma_2(\mathbb{I} - 1)d\mathbb{W}_2(t) \\ &+ \gamma_3(\mathbb{Q} - 1)d\mathbb{W}_3(t) + \gamma_3(\mathbb{R} - 1)d\mathbb{W}_4(t) + \gamma_5(\mathbb{B}_1 - 1)d\mathbb{W}_5(t) + \gamma_6(\mathbb{B}_2 - 1)d\mathbb{W}_6(t). \end{aligned} \tag{4.4}$$

Here, the $L\mathbb{V}$ operator is from the space $\mathbb{R}^6_+$ to $\mathbb{R}_+$ and defined by

$$\begin{aligned} L\mathbb{V} = &\left(1 - \frac{c_1}{\mathbb{S}}\right)\left(\Pi - \frac{\beta_1 \mathbb{B}_1 \mathbb{S}}{\mathbb{N}} - \frac{\beta_2 \mathbb{B}_2 \mathbb{S}}{\mathbb{N}} + \omega \mathbb{R} - \mu \mathbb{S}\right) + \frac{c_1}{2}{\gamma_1}^2 \\ &+ \left(1 - \frac{1}{\mathbb{I}}\right)\left(\frac{\beta_1 \mathbb{B}_1 \mathbb{S}}{\mathbb{N}} + \frac{\beta_2 \mathbb{B}_2 \mathbb{S}}{\mathbb{N}} - (\alpha_1 + \mu + \delta)\mathbb{I}\right) + \frac{1}{2}{\gamma_2}^2 \\ &+ \left(1 - \frac{1}{\mathbb{Q}}\right)\left(\delta \mathbb{I} - (\alpha_2 + \mu + \varepsilon)\mathbb{Q}\right) + \frac{1}{2}{\gamma_3}^2 + \left(1 - \frac{1}{\mathbb{R}}\right)\left(\varepsilon \mathbb{Q} - (\mu + \omega)\mathbb{R}\right) + \frac{1}{2}{\gamma_4}^2 \\ &+ \left(1 - \frac{1}{\mathbb{B}_1}\right)\left(\eta_1 \mathbb{I} - d\mathbb{B}_1\right) + \left(1 - \frac{1}{\mathbb{B}_2}\right)\left(\eta_2 \mathbb{I} - d\mathbb{B}_2\right) \\ &+ \frac{1}{2}{\gamma_5}^2 + \frac{1}{2}{\gamma_6}^2 \\ = &\Pi - \frac{\beta_1 \mathbb{B}_1 \mathbb{S}}{\mathbb{N}} - \frac{\beta_2 \mathbb{B}_2 \mathbb{S}}{\mathbb{N}} + \omega \mathbb{R} - \mu \mathbb{S} - \frac{c_1 \Pi}{\mathbb{S}} + \frac{c_1 \beta_1 \mathbb{B}_1}{\mathbb{N}} + \frac{c_1 \beta_2 \mathbb{B}_2}{\mathbb{N}} - \frac{c_1 \omega \mathbb{R}}{\mathbb{S}} + c_1 \mu \\ &+ \frac{\beta_1 \mathbb{B}_1 \mathbb{S}}{\mathbb{N}} + \frac{\beta_2 \mathbb{B}_2 \mathbb{S}}{\mathbb{N}} - (\alpha_1 + \mu + \delta)\mathbb{I} \\ &- \frac{\beta_1 \mathbb{B}_1 \mathbb{S}}{\mathbb{N}\mathbb{I}} - \frac{\beta_2 \mathbb{B}_2 \mathbb{S}}{\mathbb{N}\mathbb{I}} + (\alpha_1 + \mu + \delta) + \delta \mathbb{I} - (\alpha_2 + \mu + \varepsilon)\mathbb{Q} - \frac{\delta \mathbb{I}}{\mathbb{Q}} + (\alpha_2 + \mu + \varepsilon) \\ &+ \varepsilon \mathbb{Q} - (\mu + \omega)\mathbb{R} - \frac{\varepsilon \mathbb{Q}}{\mathbb{R}} + (\mu + \omega) + \eta_1 \mathbb{I} - d\mathbb{B}_1 - \frac{\eta_1 \mathbb{I}}{\mathbb{B}_1} + d + \eta_2 \mathbb{I} - d\mathbb{B}_2 - \frac{\eta_2 \mathbb{I}}{\mathbb{B}_2} + d \\ &+ \frac{c_1{\gamma_1}^2 + {\gamma_2}^2 + {\gamma_3}^2 + {\gamma_4}^2 + {\gamma_5}^2 + {\gamma_6}^2}{2} \\ \leq &\Pi + c_1 \mu + c_1 \beta_1 \mathbb{B}_1 + c_1 \beta_2 \mathbb{B}_2 + (\alpha_1 + \mu + \delta) + (\alpha_2 + \mu + \varepsilon) + (\mu + \omega) + \eta_1 \mathbb{I} + \eta_2 \mathbb{I} + 2d \\ &- d(\mathbb{B}_1 + \mathbb{B}_2) + \frac{c_1{\gamma_1}^2 + {\gamma_2}^2 + {\gamma_3}^2 + {\gamma_4}^2 + {\gamma_5}^2 + {\gamma_6}^2}{2}. \end{aligned} \tag{4.5}$$

Let $\beta = Max\{\beta_1, \beta_2\}$, and then choose $c_1 = \frac{d}{\beta}$, such that $c_1\beta - d = 0$. Furthermore $\mathbb{S} + \mathbb{I} + \mathbb{Q} + \mathbb{R} \leq 1$, and thus,

$$\begin{aligned} L\mathbb{V} \leq \Pi + c_1\mu + \beta + \alpha_1 + 3\mu + \delta + \alpha_2 + \varepsilon + \omega + \eta_1 + \eta_2 + 2d \\ + \frac{c_1{\gamma_1}^2 + {\gamma_2}^2 + {\gamma_3}^2 + {\gamma_4}^2 + {\gamma_5}^2 + {\gamma_6}^2}{2} = K. \end{aligned} \tag{4.6}$$

The rest of the proof is much like the proof of Theorem 2.1 in [23]. Thus, by omitting the trivial steps, the proof of the theorem is completed.

5. Extinction

It is crucial to consider the scenarios in which an infectious disease will become extinct or disappear from the population while predicting its dynamics. In this part, we will demonstrate how the solution of stochastic model (2.2) will approach zero with probability one if we change the values of the white noises. Let

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

Lemma 1. *(Strong Law) [29, 30] Let $\mathbb{M} = \{\mathbb{M}\}_{0\leq t}$ be continuous and real valued along with local martingale which vanishs as $t \to 0$. Then,*

$$\begin{aligned} &\lim_{t\to\infty} \langle \mathbb{M}, \mathbb{M} \rangle_t = \infty, \ \Rightarrow \ \lim_{t\to\infty} \frac{\mathbb{M}_t}{\langle \mathbb{M}, \mathbb{M} \rangle_t} = 0, \ a.s. \\ &\limsup_{t\to\infty} \frac{\langle \mathbb{M}, \mathbb{M} \rangle_t}{t} < 0, \ \Rightarrow \ \lim_{t\to\infty} \frac{\mathbb{M}_t}{t} = 0, \ a.s. \end{aligned} \tag{5.1}$$

Lemma 2. *[23, 29] Let $(\mathbb{S}, \mathbb{I}, \mathbb{Q}, \mathbb{R}, \mathbb{B}_1, \mathbb{B}_2)$ be the solution of system (2.2) with initial value $(\mathbb{S}(0), \mathbb{I}(0), \mathbb{Q}(0), \mathbb{R}(0), \mathbb{B}_1(0), \mathbb{B}_2(0)) \in \mathbb{R}^6_+$. Then,*

$$\begin{aligned} &\limsup_{t\to\infty} \frac{\ln \mathbb{S}(t)}{t} = 0, \quad \limsup_{t\to\infty} \frac{\ln \mathbb{I}(t)}{t} = 0, \quad \limsup_{t\to\infty} \frac{\ln \mathbb{Q}(t)}{t} = 0, \\ &\limsup_{t\to\infty} \frac{\ln \mathbb{R}(t)}{t} = 0, \quad \limsup_{t\to\infty} \frac{\ln \mathbb{B}_1(t)}{t} = 0, \quad \limsup_{t\to\infty} \frac{\ln \mathbb{B}_2(t)}{t} = 0, \ a.s. \end{aligned} \tag{5.2}$$

Furthermore, if $\mu > \frac{\gamma_1^2 \vee \gamma_2^2 \vee \gamma_3^2 \vee \gamma_4^2}{2}$, and $d > \frac{\gamma_5^2 \vee \gamma_6^2}{2}$ and then

$$\begin{aligned} &\lim_{t\to\infty} \frac{\int_0^t \mathbb{S}(s)d\mathbb{W}_1(s)}{t} = 0, \quad \lim_{t\to\infty} \frac{\int_0^t \mathbb{I}(u)d\mathbb{W}_2(u)}{t} = 0, \quad \lim_{t\to\infty} \frac{\int_0^t \mathbb{Q}(u)d\mathbb{W}_3(u)}{t} = 0, \\ &\lim_{t\to\infty} \frac{\int_0^t \mathbb{R}(s)d\mathbb{W}_4(s)}{t} = 0, \quad \lim_{t\to\infty} \frac{\int_0^t \mathbb{B}_1(s)d\mathbb{W}_5(s)}{t} = 0, \quad \lim_{t\to\infty} \frac{\int_0^t \mathbb{B}_2(s)d\mathbb{W}_6(s)}{t} = 0, \ a.s. \end{aligned} \tag{5.3}$$

Then, the solution of system (2.2)

$$\begin{aligned}
\limsup_{t\to\infty} \mathbb{S}(t) &= \frac{\Pi}{\mu},\\
\limsup_{t\to\infty} \mathbb{I}(t) &= 0,\\
\limsup_{t\to\infty} \mathbb{Q}(t) &= 0,\\
\limsup_{t\to\infty} \mathbb{R}(t) &= 0,\\
\limsup_{t\to\infty} \mathbb{B}_1(t) &= 0,\\
\limsup_{t\to\infty} \mathbb{B}_2(t) &= 0, \quad \text{a.s.}
\end{aligned} \tag{5.4}$$

For proving Lemma 2, one can take into consideration the proof of lemma 2.1 and 2.2 in [29]. Consequently, the proof of the Lemma is straight forward and hence is omitted.

To discuss the extinction theory of system (2.2), let us define the threshold parameter

$$R_s = \frac{\beta_1\eta_1 + \beta_1\eta_2}{d\left(\alpha_1 + \mu + \delta + \frac{\gamma_2^2}{2}\right)}.$$

Theorem 2. *Assume that* $(\mathbb{S}(t), \mathbb{I}(t), \mathbb{Q}(t), \mathbb{R}(t), \mathbb{B}_1(t), \mathbb{B}_2(t))$ *correspond to initial data*

$(\mathbb{S}(0), \mathbb{I}(0), \mathbb{Q}(0), \mathbb{R}(0), \mathbb{B}_1(0), \mathbb{B}_2(0)) \in \mathbb{R}_+^6$ be a solution of model (2.2). Further, if $R_s < 1$, then such solution of system (2.2) satisfies the following relations:

$$\begin{aligned}
\lim_{t\to\infty} \langle \mathbb{S}(t) \rangle &= \frac{\Pi}{\mu}, \quad \text{a.s.},\\
\lim_{t\to\infty} \langle \mathbb{I}(t) \rangle &= 0, \quad \text{a.s.},\\
\lim_{t\to\infty} \langle \mathbb{Q}(t) \rangle &= 0, \quad \text{a.s.},\\
\lim_{t\to\infty} \langle \mathbb{R}(t) \rangle &= 0, \quad \text{a.s.},\\
\lim_{t\to\infty} \langle \mathbb{B}_1(t) \rangle &= 0, \quad \text{a.s.},\\
\lim_{t\to\infty} \langle \mathbb{B}_2(t) \rangle &= 0, \quad \text{a.s..}
\end{aligned} \tag{5.5}$$

This means that the infection from the community will be eradicated certainly in the long run.

Proof: By integrating system (2.2), we obtained the following set of equations:

$$\begin{aligned}
\frac{1}{t}(\mathbb{S}(t)-\mathbb{S}(0)) &= \Pi - \frac{\beta_1\langle\mathbb{B}_1\rangle\langle\mathbb{S}\rangle}{\langle\mathbb{N}\rangle} - \frac{\beta_2\langle\mathbb{B}_2\rangle\langle\mathbb{S}\rangle}{\langle\mathbb{N}\rangle} + \omega\langle\mathbb{R}\rangle - \mu\langle\mathbb{S}\rangle + \frac{\gamma_1\int_0^t \mathbb{S}(r)d\mathbb{W}_1(r)}{t},\\
\frac{1}{t}(\mathbb{I}(t)-\mathbb{I}(0)) &= \frac{\beta_1\langle\mathbb{B}_1\rangle\langle\mathbb{S}\rangle}{\langle\mathbb{N}\rangle} + \frac{\beta_2\langle\mathbb{B}_2\rangle\langle\mathbb{S}\rangle}{\langle\mathbb{N}\rangle} - (\alpha_1+\mu+\delta)\langle\mathbb{I}\rangle + \frac{\gamma_2\int_0^t \mathbb{I}(r)\mathbb{W}_2(r)}{t},\\
\frac{1}{t}(\mathbb{Q}(t)-\mathbb{Q}(0)) &= \delta\langle\mathbb{I}\rangle - (\alpha_2+\mu+\varepsilon)\langle\mathbb{Q}\rangle + \frac{\gamma_3\int_0^t \mathbb{Q}(r)d\mathbb{W}_3(r)}{t},\\
\frac{1}{t}(\mathbb{R}(t)-\mathbb{R}(0)) &= \varepsilon\langle\mathbb{Q}\rangle - (\mu+\omega)\langle\mathbb{R}\rangle + \frac{\gamma_4\int_0^t \mathbb{R}(r)d\mathbb{W}_4(r)}{t},\\
\frac{1}{t}(\mathbb{B}_1(t)-\mathbb{B}_1(0)) &= \eta_1\langle\mathbb{I}\rangle - d\langle\mathbb{B}_1\rangle + \frac{\gamma_5\int_0^t \mathbb{B}_1(r)d\mathbb{W}_5(r)}{t},\\
\frac{1}{t}(\mathbb{B}_1(t)-\mathbb{B}_2(0)) &= \eta_2\langle\mathbb{I}\rangle - d\langle\mathbb{B}_2\rangle + \frac{\gamma_6\int_0^t \mathbb{B}_2(r)d\mathbb{W}_6(r)}{t}.
\end{aligned} \tag{5.6}$$

By taking into consideration the second last relation from the above system, we have

$$\begin{aligned}
\langle\mathbb{B}_1\rangle &= \frac{\eta_1}{d}\langle\mathbb{I}\rangle - \frac{1}{d}\Big(\frac{\mathbb{B}_1(t)-\mathbb{B}_1(0)}{t}\Big) + \frac{\gamma_5}{d}\Big(\frac{\int_0^t \mathbb{B}_1(r)d\mathbb{W}_5(r)}{t}\Big),\\
&= \frac{\eta_1}{d}\langle\mathbb{I}\rangle + \mathbb{M}_1(t),
\end{aligned} \tag{5.7}$$

where

$$\mathbb{M}_1(t) = -\frac{1}{d}\Big(\frac{\mathbb{B}_1(t)-\mathbb{B}_1(0)}{t}\Big) + \frac{\gamma_5}{d}\Big(\frac{\int_0^t \mathbb{B}_1(r)d\mathbb{W}_5(r)}{t}\Big). \tag{5.8}$$

In the same way, the last relation in Eq (5.6) yields

$$\begin{aligned}
\langle\mathbb{B}_2\rangle &= \frac{\eta_2}{d}\langle\mathbb{I}\rangle - \Big(\frac{\mathbb{B}_2(t)-\mathbb{B}_2(0)}{t}\Big)\frac{1}{d} + \frac{\gamma_6}{d}\Big(\frac{\int_0^t \mathbb{B}_2(r)d\mathbb{W}_6(r)}{t}\Big),\\
&= \frac{\eta_2}{d}\langle\mathbb{I}\rangle + \mathbb{M}_2(t),
\end{aligned} \tag{5.9}$$

where

$$\mathbb{M}_2(t) = -\frac{1}{d}\Big(\frac{\mathbb{B}_2(t)-\mathbb{B}_2(0)}{t}\Big) + \frac{\gamma_6}{d}\Big(\frac{\int_0^t \mathbb{B}_2(r)d\mathbb{W}_6(r)}{t}\Big). \tag{5.10}$$

Directly applying the Itô formula to the second relation in system (2.2) gives us

$$\begin{aligned}
d\log\mathbb{I} &= \Big[\frac{\beta_1\mathbb{B}_1\mathbb{S}}{\mathbb{I}\mathbb{N}} + \frac{\beta_2\mathbb{B}_2\mathbb{S}}{\mathbb{I}\mathbb{N}} - (\alpha_1+\mu+\delta) - \frac{\gamma_2^2}{2}\Big]dt + \gamma_2 d\mathbb{W}_2(t),\\
&\leq \Big[\frac{\beta_1\mathbb{B}_1}{\mathbb{I}} + \frac{\beta_2\mathbb{B}_2}{\mathbb{I}} - (\alpha_1+\mu+\delta+\frac{\gamma_2^2}{2})\Big]dt + \gamma_2 d\mathbb{W}_2(t).
\end{aligned} \tag{5.11}$$

One can obtain the following relation very easily by integrating Eq (5.11) from 0 to t and then dividing the same by t:

$$\frac{\log\mathbb{I}-\log\mathbb{I}(0)}{t} \leq \Big[\frac{\beta_1\langle\mathbb{B}_1\rangle}{\langle\mathbb{I}\rangle} + \frac{\beta_1\langle\mathbb{B}_2\rangle}{\langle\mathbb{I}\rangle} - (\alpha_1+\mu+\delta+\frac{\gamma_2^2}{2})\Big] + \frac{\gamma_2 d\mathbb{W}_2(t)}{t}. \tag{5.12}$$

If we substitute relations (5.7) and (5.9) in Eq (5.12), we get the following:

$$\begin{aligned}\frac{\log\mathbb{I}(t)}{t} &\leq \left[\frac{\beta_1(\frac{\eta_1}{d}\langle\mathbb{I}\rangle + \mathbb{M}_1(t))}{\langle\mathbb{I}\rangle} + \frac{\beta_1(\frac{\eta_2}{d}\langle\mathbb{I}\rangle + \mathbb{M}_2(t))}{\langle\mathbb{I}\rangle} - (\alpha_1 + \mu + \delta + \frac{\gamma_2^2}{2})\right] + \frac{\log\mathbb{I}(0)}{t} + \frac{\gamma_2 d\mathbb{W}_2(t)}{t} \\ &\leq \left[\frac{\frac{\beta_1\eta_1}{d}\langle\mathbb{I}\rangle}{\langle\mathbb{I}\rangle} + \frac{\frac{\beta_1\eta_2}{d}\langle\mathbb{I}\rangle}{\langle\mathbb{I}\rangle} - (\alpha_1 + \mu + \delta + \frac{\gamma_2^2}{2})\right] + \frac{\beta_1\mathbb{M}_1(t)}{\langle\mathbb{I}\rangle} + \frac{\beta_1\mathbb{M}_2(t)}{\langle\mathbb{I}\rangle} + \frac{\log\mathbb{I}(0)}{t} + \frac{\gamma_2 d\mathbb{W}_2(t)}{t} \quad (5.13) \\ &= \left[\frac{\beta_1\eta_1}{d} + \frac{\beta_1\eta_2}{d} - (\alpha_1 + \mu + \delta + \frac{\gamma_2^2}{2})\right] + \frac{\beta_1\mathbb{M}_1(t)}{\langle\mathbb{I}\rangle} + \frac{\beta_1\mathbb{M}_2(t)}{\langle\mathbb{I}\rangle} + \frac{\log\mathbb{I}(0)}{t} + \frac{\gamma_2 d\mathbb{W}_2(t)}{t}.\end{aligned}$$

Further, $\mathbb{M}_i(t) = \frac{\gamma_i}{t}\int_0^t g_i d\mathbb{W}_i(t)$ for $i = 1, 2, \cdots 6$. $g_1 = \mathbb{S}, g_2 = \mathbb{I}, g_3 = \mathbb{Q}, g_4 = \mathbb{R}, g_5 = \mathbb{B}_1, g_6 = \mathbb{B}_2$ are the continuous local martingale functions and equals 0 at $t = 0$. If we let $t \to \infty$ and use Lemma 2, we get

$$\lim_{t\to\infty}\sup\frac{1}{t}\mathbb{M}_i(t) = 0. \quad (5.14)$$

Utilizing a similar argument, we can derive very easily that $\lim_{t\to\infty}\sup\mathbb{M}_1(t) = 0$ and $\lim_{t\to\infty}\sup\mathbb{M}_2(t) = 0$.

Under the assumption of $R_s < 1$, Eq (5.13) becomes

$$\lim_{t\to\infty}\sup\frac{\log\mathbb{I}(t)}{t} \leq \left(\alpha_1 + \mu + \delta + \frac{\gamma_2^2}{2}\right)\left(R_s - 1\right) < 0, \quad \text{a.s.} \quad (5.15)$$

As a consequence of relation (5.15), we have

$$\lim_{t\to\infty}\langle\mathbb{I}\rangle = 0, \quad \text{a.s.} \quad (5.16)$$

Utilizing relation (5.16) in Eqs (5.7) and (5.9) as well as using the facts $\lim_{t\to\infty}\sup\mathbb{M}_1(t) = 0$ and $\lim_{t\to\infty}\sup\mathbb{M}_2(t) = 0$, we get

$$\lim_{t\to\infty}\langle\mathbb{B}_1\rangle = 0, \quad \text{a.s,} \quad (5.17)$$

and

$$\lim_{t\to\infty}\langle\mathbb{B}_2\rangle = 0, \quad \text{a.s.} \quad (5.18)$$

Now for the 3rd equation of system (5.6), we have

$$\langle\mathbb{Q}\rangle = \frac{\delta\langle\mathbb{I}\rangle}{(\alpha_2 + \mu + \varepsilon)} - \frac{\mathbb{Q}(t) - \mathbb{Q}(0))}{t((\alpha_2 + \mu + \varepsilon))} + \frac{1}{(\alpha_2 + \mu + \varepsilon)}\left(\frac{\gamma_3\int_0^t \mathbb{Q}(r)d\mathbb{W}_3(r)}{t}\right). \quad (5.19)$$

Utilizing relation (5.16) in Eq (5.19) as well as using the facts $\lim_{t\to\infty}\sup\mathbb{M}_3(t) = 0$, we get

$$\lim_{t\to\infty}\langle\mathbb{Q}\rangle = 0, \quad \text{a.s.} \quad (5.20)$$

In a similar way, we can get

$$\lim_{t\to\infty}\langle\mathbb{R}(t)\rangle = 0, \quad \text{a.s.} \quad (5.21)$$

Finally, we will take into account the first equation of system (5.6). By taking the integral from 0 to t, dividing the result by t and using relations (5.17), (5.18) and (5.21), we obtain

$$\begin{aligned}
&\frac{\mathbb{S}(t)-\mathbb{S}(0)}{t} = \Pi - \frac{\beta_1\langle\mathbb{B}_1\rangle\langle\mathbb{S}\rangle}{\langle\mathbb{N}\rangle} - \frac{\beta_2\langle\mathbb{B}_2\rangle\langle\mathbb{S}\rangle}{\langle\mathbb{N}\rangle} + \omega\langle\mathbb{R}\rangle - \mu\langle\mathbb{S}\rangle + \frac{\gamma_1\int_0^t \mathbb{S}(r)d\mathbb{W}_1(r)}{t},\\
&\langle\mathbb{S}\rangle = \frac{\Pi}{d} - \frac{1}{d}\Big[\frac{\beta_1\langle\mathbb{B}_1\rangle\langle\mathbb{S}\rangle}{\langle\mathbb{N}\rangle} - \frac{\beta_2\langle\mathbb{B}_2\rangle\langle\mathbb{S}\rangle}{\langle\mathbb{N}\rangle} + \omega\langle\mathbb{R}\rangle + \frac{\gamma_1\int_0^t \mathbb{S}(r)d\mathbb{W}_1(r)}{t}\Big].
\end{aligned} \tag{5.22}$$

This gives us

$$\lim_{t\to\infty}\langle\mathbb{S}\rangle = \frac{\Pi}{\mu} \text{ a.s.} \tag{5.23}$$

This completes the proof.

6. Persistence of the proposed system (2.2)

Now, we have to provide a condition for the persistence of Eq (2.2).

Definition 1. [30] The considered system (2.2) is persistent, if

$$\liminf_{t\to\infty}\frac{1}{t}\int_0^t(\mathbb{B}_1+\mathbb{B}_2)(r)dr > 0 \text{ a.s.} \tag{6.1}$$

Theorem 3. *If* $\mathbf{R_0^s} = \frac{\beta_1\beta_2\eta_1\eta_2}{(\alpha_1+\mu+\delta+\frac{\gamma_2{}^2}{2})(d+\frac{\gamma_5{}^2}{2})(d+\frac{\gamma_6{}^2}{2})}$ *then at any condition of initial values* $(\mathbb{S}(0),\mathbb{I}(0),\mathbb{Q}(0),\mathbb{R}(0),\mathbb{B}_1(0),\mathbb{B}_2(0)) \in R_+^6$, the disease $\mathbb{B}_1(t)+\mathbb{B}_2(t)$ has the following:

$$\liminf_{t\to\infty}\langle\mathbb{B}_1(t)+\mathbb{B}_2(t)\rangle \geq \frac{4(\mu+\frac{\gamma_1{}^2}{2})\Big[\sqrt[4]{(\frac{\beta_1\beta_2\eta_1\eta_2}{(\alpha_1+\mu+\delta+\frac{\gamma_2{}^2}{2})(d+\frac{\gamma_5{}^2}{2})(d+\frac{\gamma_6{}^2}{2})}-1}\Big]}{\beta}, \text{ a.s.} \tag{6.2}$$

Then we can say the disease will prevail if $\mathbf{R_0^s} > 1$.

Proof: Let define

$$H_1 = -ln\mathbb{S} - K_1 ln\mathbb{I} - K_2 ln\mathbb{B}_1 - K_3\mathbb{B}_2, \tag{6.3}$$

where K_1, K_2, and K_3 are constants and we will find later. Applying Itô formula, we have

$$dH_1 = \mathcal{L}H_1 dt - \gamma_1 d\mathbb{W}_1(t) - K_1\gamma_2 d\mathbb{W}_2(t) - K_2\gamma_5 d\mathbb{W}_5(t) - K_3\gamma_6 d\mathbb{W}_6(t) \tag{6.4}$$

$$\begin{aligned}
\mathcal{L}H_1 &= -\frac{\Pi}{\mathbb{S}} + \frac{\beta_1\mathbb{B}_1}{\mathbb{N}} + \frac{\beta_2\mathbb{B}_2}{\mathbb{N}} - \frac{\omega\mathbb{R}}{\mathbb{S}} + \mu + \frac{\gamma_1{}^2}{2} - \frac{K_1\beta_1\mathbb{B}_1\mathbb{S}}{\mathbb{N}\mathbb{I}} - \frac{K_1\beta_2\mathbb{B}_2\mathbb{S}}{\mathbb{N}\mathbb{I}} + K_1(\alpha_1+\mu+\delta)\\
&\quad + \frac{K_1\gamma_2{}^2}{2} - \frac{K_2\eta_1\mathbb{I}}{\mathbb{B}_1} + K_2 d - \frac{K_3\eta_2\mathbb{I}}{\mathbb{B}_2} + K_3 d + \frac{K_2\gamma_5{}^2}{2} + \frac{K_3\gamma_6{}^2}{2}\\
&\leq \beta_1\mathbb{B}_1 + \beta_2\mathbb{B}_2 + (\mu+\frac{\gamma_1{}^2}{2}) - \frac{K_1\beta_1\mathbb{B}_1}{\mathbb{I}} - \frac{K_1\beta_2\mathbb{B}_2}{\mathbb{I}} + K_1(\alpha_1+\mu+\delta) + \frac{K_1\gamma_2{}^2}{2}\\
&\quad - \frac{K_2\eta_1\mathbb{I}}{\mathbb{B}_1} + K_2 d - \frac{K_3\eta_2\mathbb{I}}{\mathbb{B}_2} + K_3 d + \frac{K_2\gamma_5{}^2}{2} + \frac{K_3\gamma_6{}^2}{2}.
\end{aligned} \tag{6.5}$$

Let

$$K_1(\alpha_1+\mu+\delta+\frac{{\gamma_2}^2}{2})=(\mu+\frac{{\gamma_1}^2}{2}),\quad K_2(d+\frac{{\gamma_5}^2}{2})=(\mu+\frac{{\gamma_1}^2}{2}),\quad K_3(d+\frac{{\gamma_6}^2}{2})=(\mu+\frac{{\gamma_1}^2}{2}). \tag{6.6}$$

$$\begin{aligned}\mathcal{L}H_1 &\leq -4\sqrt[4]{(\frac{K_1\beta_1\mathbb{B}_1}{\mathbb{I}})(\frac{K_1\beta_2\mathbb{B}_2}{\mathbb{I}})(\frac{K_2\eta_1\mathbb{I}}{\mathbb{B}_1})(\frac{K_3\eta_2\mathbb{I}}{\mathbb{B}_2})}+4(\mu+\frac{{\gamma_1}^2}{2})+\beta_1\mathbb{B}_1+\beta_2\mathbb{B}_2,\\ &=-4\sqrt[4]{\frac{(\mu+\frac{{\gamma_1}^2}{2})^4\beta_1\beta_2\eta_1\eta_2}{(\alpha_1+\mu+\delta+\frac{{\gamma_2}^2}{2})(d+\frac{{\gamma_5}^2}{2})(d+\frac{{\gamma_6}^2}{2})}}+4(\mu+\frac{{\gamma_1}^2}{2})+\beta_1\mathbb{B}_1+\beta_2\mathbb{B}_2,\\ &=-4(\mu+\frac{{\gamma_1}^2}{2})\Big[\sqrt[4]{\frac{\beta_1\beta_2\eta_1\eta_2}{(\alpha_1+\mu+\delta+\frac{{\gamma_2}^2}{2})(d+\frac{{\gamma_5}^2}{2})(d+\frac{{\gamma_6}^2}{2})}}-1\Big]+\beta(\mathbb{B}_1+\mathbb{B}_2).\end{aligned} \tag{6.7}$$

$\beta = Max\{\beta_1,\beta_2\}$. Substituting Eq (6.7) into Eq (6.4), and then integrating both sides of Eq (6.4), we have

$$\begin{aligned}&\frac{H_1(\mathbb{S}(t),\mathbb{I}(t),\mathbb{B}_1(t),\mathbb{B}_2(t))-H_1(\mathbb{S}(0),\mathbb{I}(0),\mathbb{B}_1(0),\mathbb{B}_2(0))}{t}\\ &\leq -4(\mu+\frac{{\gamma_1}^2}{2})\Big[\sqrt[4]{\frac{\beta_1\beta_2\eta_1\eta_2}{(\alpha_1+\mu+\delta+\frac{{\gamma_2}^2}{2})(d+\frac{{\gamma_5}^2}{2})(d+\frac{{\gamma_6}^2}{2})}}-1\Big]+\beta\langle\mathbb{B}_1+\mathbb{B}_2\rangle\\ &-\frac{\gamma_1 d\mathbb{W}_1(t)}{t}-\frac{K_1\gamma_2 d\mathbb{W}_2(t)}{t}-\frac{K_2\gamma_5 d\mathbb{W}_5(t)}{t}-\frac{K_3\gamma_6 d\mathbb{W}_6(t)}{t}.\\ &\leq -4(\mu+\frac{{\gamma_1}^2}{2})\Big[\sqrt[4]{\frac{\beta_1\beta_2\eta_1\eta_2}{(\alpha_1+\mu+\delta+\frac{{\gamma_2}^2}{2})(d+\frac{\gamma_5^2}{2})(d+\frac{{\gamma_6}^2}{2})}}-1\Big]+\beta\langle\mathbb{B}_1+\mathbb{B}_2\rangle+\Psi(t),\end{aligned} \tag{6.8}$$

where $\Psi(t)=-\frac{\gamma_1 d\mathbb{W}_1(t)}{t}-\frac{K_1\gamma_2 d\mathbb{W}_2(t)}{t}-\frac{K_2\gamma_5 d\mathbb{W}_5(t)}{t}-\frac{K_3\gamma_6 d\mathbb{W}_6(t)}{t}$. From the strong law as stated in Lemma 1, we arrive at

$$\lim_{t\to\infty}\Psi(t)=0. \tag{6.9}$$

From Eq (6.8), we have

$$\begin{aligned}\langle\mathbb{B}_1+\mathbb{B}_2\rangle \geq\ &\frac{4(\mu+\frac{{\gamma_1}^2}{2})\Big[\sqrt[4]{\frac{\beta_1\beta_2\eta_1\eta_2}{(\alpha_1+\mu+\delta+\frac{{\gamma_2}^2}{2})(d+\frac{{\gamma_5}^2}{2})(d+\frac{{\gamma_6}^2}{2})}}-1\Big]}{\beta}-\frac{1}{\beta}\Psi(t)\\ &+\frac{1}{\beta}\Big(\frac{H_1(\mathbb{S}(t),\mathbb{I}(t),\mathbb{B}_1(t),\mathbb{B}_2(t))-H_1(\mathbb{S}(0),\mathbb{I}(0),\mathbb{B}_1(0),\mathbb{B}_2(0))}{t}\Big).\end{aligned} \tag{6.10}$$

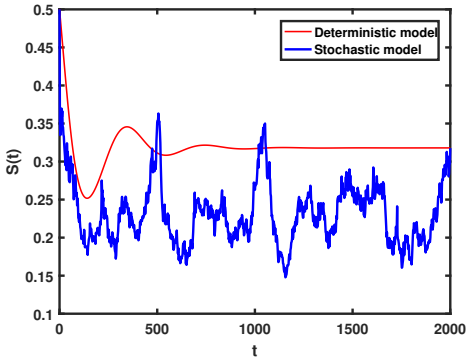
According to Lemma 2 and Eq (6.9), the limit superior of Eq (6.10), we have

$$\liminf_{t\to\infty}\ \langle\mathbb{B}_1+\mathbb{B}_2\rangle \geq \frac{4(\mu+\frac{{\gamma_1}^2}{2})\Big[\sqrt[4]{\mathbf{R_0^S}}-1\Big]}{\beta},\ \text{ a.s.} \tag{6.11}$$

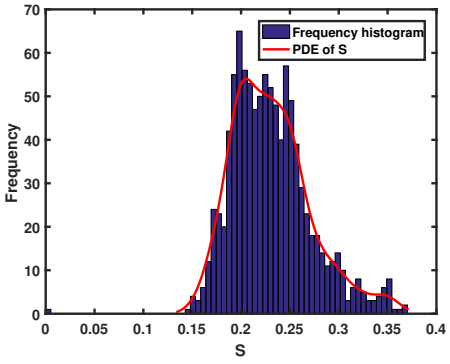
This finishes the proof of Theorem 3.

7. Simulations of the probability density function of system (2.2)

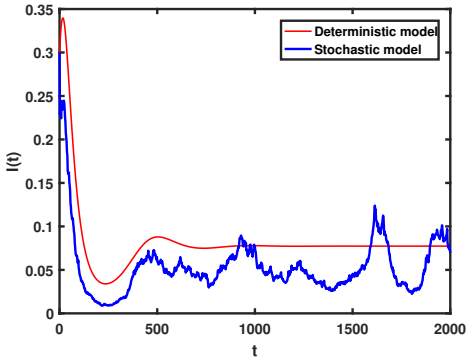
A probability density function (PDF) is a mathematical function that describes the probability distribution of a continuous random variable. It is a function that takes a real-valued number as input and returns the likelihood of that number being observed as the output.



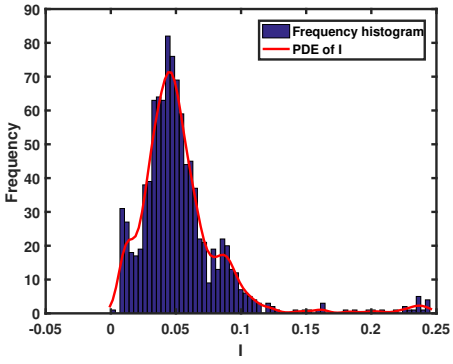
(a) $\mathbb{S}(t)$



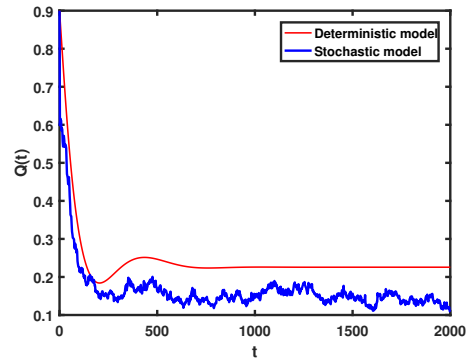
(b) Frequency histogram and probability density curve of $\mathbb{S}(t)$



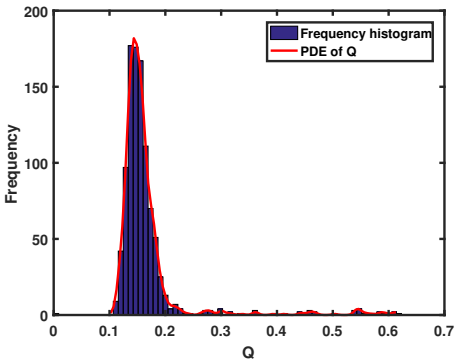
(c) $\mathbb{I}(t)$



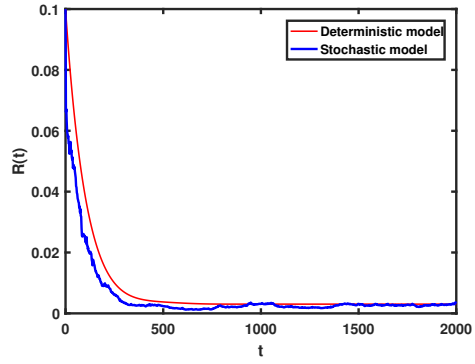
(d) Frequency histogram and probability density curve of $\mathbb{I}(t)$



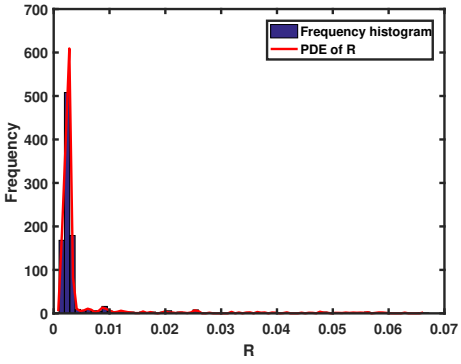
(e) $\mathbb{Q}(t)$



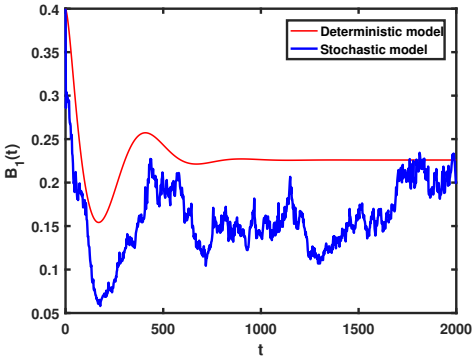
(f) Frequency histogram and probability density curve of $\mathbb{Q}(t)$



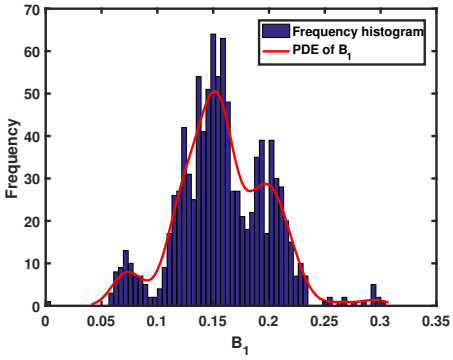
(g) $\mathbb{R}(t)$



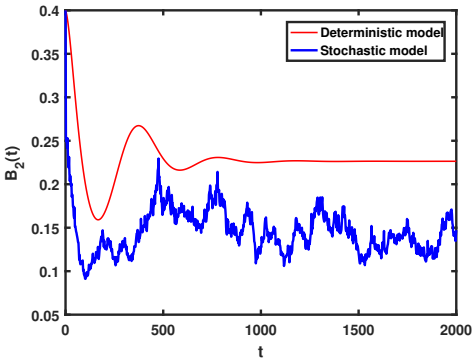
(h) Frequency histogram and probability density curve of $\mathbb{R}(t)$



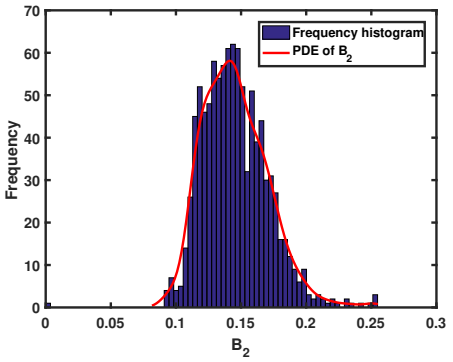
(i) $\mathbb{B}_1(t)$



(j) Frequency histogram and probability density curve of $\mathbb{B}_1(t)$



(k) $\mathbb{B}_2(t)$



(l) Frequency histogram and probability density curve of $\mathbb{B}_2(t)$

Figure 3. Solution profiles for the various compartments of model (2.2) and associated ODEs model.

Numerical investigation of the epidemiological impact of saturation effect on the various components of the model is presented in Figure 3. For numerical simulation of the compartments we chose the parameter and the initial value $\Pi = 0.9, \beta_1 = 0.007, \beta_2 = 0.003, \omega = 0.05, \delta = 0.05, \alpha_1 = 0.01, \mu = 0.003, \alpha_2 = 0.04, \eta_1 = 0.01, \eta_2 = 0.05, d = 0.03$, and $\gamma_1 = 0.50, \gamma_2 = 0.60, \gamma_3 = 0.50, \gamma_4 =$

$0.35, \gamma_5 = 0.41, \gamma_6 = 0.55$ and $d = 0.05$. and $\mathbb{S}(0) = 0.50, \mathbb{I}(0) = 0.30, \mathbb{Q}(0) = 0.90, \mathbb{R}(0) = 0.10, \mathbb{B}_1(0) = 0.40, \mathbb{B}_2(0) = 0.40$, and rest value we taken from Figure 5. It is observed that the saturation effect greatly impacted the different compartments.

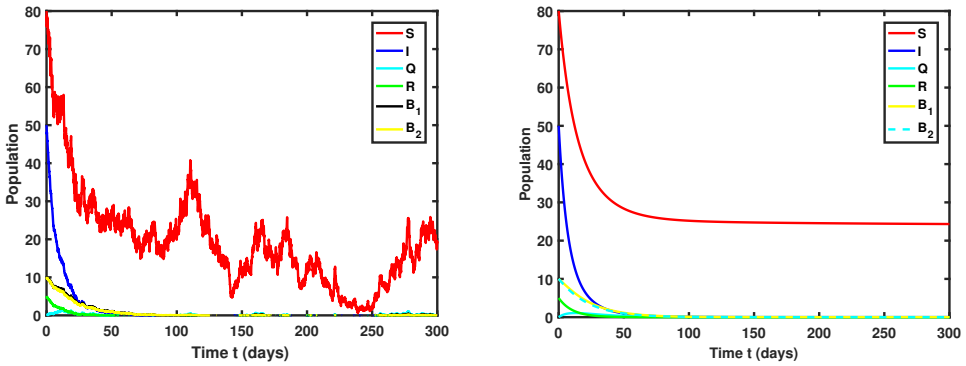
8. Numerical scheme and simulations

To quantitatively validate the theoretical conclusions associated with system (2.2), it is crucial to estimate suitable values of the parameters. To do so, the beginning population sizes of both humans and bacteria are assumed, and further, two sets of the values of the parameters are considered. The desired time interval for both scenarios is $[0, 300]$. Exercising the higher-ordered Milstein method, we have the following algorithm for obtaining numerical solution of the proposed stochastic model:

$$\begin{aligned}
\mathbb{S}_{i+1} &= \mathbb{S}_i + \left[\Pi - \frac{\beta_1 \mathbb{B}_{1,i}\mathbb{S}_i}{\mathbb{N}_i} - \frac{\beta_2 \mathbb{B}_{2,i}\mathbb{S}_i}{\mathbb{N}_i} + \omega\mathbb{R}_i - \mu\mathbb{S}_i\right] \triangle t + \gamma_1 \mathbb{S}_i \sqrt{\triangle t}\varsigma_{1,i} + \frac{\gamma_1^2}{2}\mathbb{S}_i(\varsigma_{1,i}^2 - 1) \triangle t,\\
\mathbb{I}_{i+1} &= \mathbb{I}_i + \left[\frac{\beta_1 \mathbb{B}_{1,i}\mathbb{S}_I}{\mathbb{N}_i} + \frac{\beta_2 \mathbb{B}_{2,i}\mathbb{S}_i}{\mathbb{N}_i} - (\alpha_1 + \mu + \delta)\mathbb{I}_i\right] \triangle t + \gamma_2 \mathbb{I}_i \sqrt{\triangle t}\varsigma_{2,i} + \frac{\gamma_2^2}{2}\mathbb{I}_i(\varsigma_{2,i}^2 - 1) \triangle t,\\
\mathbb{Q}_{i+1} &= \mathbb{Q}_i + \left[\delta\mathbb{I}_i - (\alpha_2 + \mu + \varepsilon)\mathbb{Q}_i\right] \triangle t + \gamma_3 \mathbb{Q}_i \sqrt{\triangle t}\varsigma_{3,i} + \frac{\gamma_3^2}{2}\mathbb{Q}_i(\varsigma_{3,i}^2 - 1) \triangle t,\\
\mathbb{R}_{i+1} &= \mathbb{R}_i + \left[\varepsilon\mathbb{Q}_i - (\mu + \omega)\mathbb{R}_i\right] \triangle t + \gamma_4 \mathbb{R}_i \sqrt{\triangle t}\varsigma_{4,i} + \frac{\gamma_4^2}{2}\mathbb{R}_i(\varsigma_{4,i}^2 - 1) \triangle t,\\
\mathbb{B}_{1,i+1} &= \mathbb{B}_{1_i} + \left[\eta_1\mathbb{I}_i - d\mathbb{B}_{1,i}\right] \triangle t + \gamma_5 \mathbb{B}_{1,i} \sqrt{\triangle t}\varsigma_{5,i} + \frac{\gamma_5^2}{2}\mathbb{B}_{1,i}(\varsigma_{5,i}^2 - 1) \triangle t,\\
\mathbb{B}_{2,i+1} &= \mathbb{B}_{2_i} + \left[\eta_2\mathbb{I}_i - d\mathbb{B}_{2,i}\right] \triangle t + \gamma_6 \mathbb{B}_{2,i} \sqrt{\triangle t}\varsigma_{6,i} + \frac{\gamma_6^2}{2}\mathbb{B}_{2,i}(\varsigma_{6,i}^2 - 1) \triangle t,
\end{aligned} \tag{8.1}$$

where $\varsigma_{i,j}(i = 1, \cdots, 6)$ denotes the standard Gaussian variables, having distribution $N(0, 1)$, and Δt is the time-step. The intensities of the noises satisfy the condition $\gamma_i > 0, (i = 1, \cdots, 6)$.

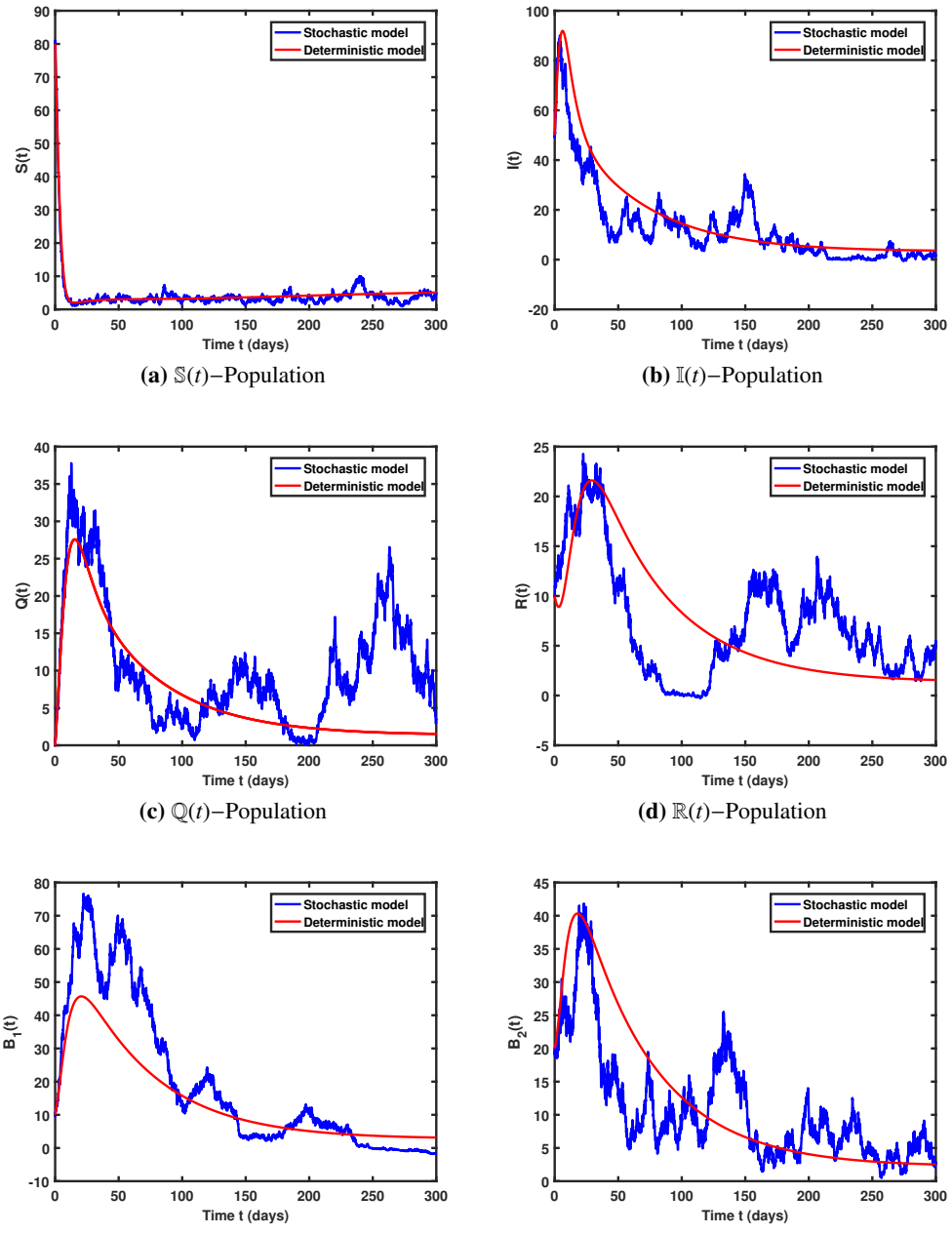
The initial conditions used for the simulations were assumed as follows: $\mathbb{S}(0) = 80, \mathbb{I}(0) = 50, \mathbb{Q}(0) = 0, \mathbb{R}(0) = 10, \mathbb{B}_1(0) = 10, \mathbb{B}_2(0) = 10$. Unless, otherwise stated, the values of parameters $\Pi = 0.12, \beta_1 = 0.002, \beta_2 = 0.003, \omega = 0.09, \mu = 0.005, \delta = 0.005, \alpha_1 = 0.2, \alpha_2 = 0.05, \eta_1 = 0.004, \eta_2 = 0.002$ and $d = 0.05$. Noise intensities were assumed as $\gamma_1 = 0.55, \gamma_2 = 0.20, \gamma_3 = 0.25, \gamma_4 = 0.13, \gamma_5 = 0.15, \gamma_6 = 0.10$. In Figure 4, simulations of the deterministic system (2.1) and perturbed system (2.2) are presented, when the associated stochastic reproduction numbers are given by $R_s < 1$. These figures depict both concentrations of the Vibrio choleae approaching zero like the trajectory of an exponential function with probability one, and this further shows the numerical verification of Theorem 2. The figures further suggest that both the stochastic and deterministic systems are in close agreement. Further, trajectories of both systems approach the DFE as time evolves.



(a) Stochastic System (2.2) **(b)** Derminstaic System (2.1)

Figure 4. Simulations of the deterministic model (2.1) and stochastic model (2.2) models when the associated stochastic reproduction numbers are less than one.

Biologically, Theorem 3 shows the persistence of the disease in view of system (2.2). To support the conclusion of the theorem numerically, we assumed: $\Pi = 0.12, \beta_1 = 0.008, \beta_2 = 0.007, \omega = 0.07, \delta = 0.05, \alpha_1 = 0.01, \mu = 0.005, \alpha_2 = 0.05, \eta_1 = 0.05, \eta_2 = 0.04, d = 0.06$, and $\gamma_1 = 0.60, \gamma_2 = 0.65, \gamma_3 = 0.50, \gamma_4 = 0.35, \gamma_5 = 0.41, \gamma_6 = 0.55$, and the initial conditions were the same as Figure 4, It has been noted that the disease will continue to spread throughout the population for low white noise levels. This result is further illustrated by Figure 5, wherein the trajectories of both the states $\mathbb{B}_1$ and $\mathbb{B}_2$ are non-zero, i.e., there must be some concentration of the bacteria. Ultimately, it supports the statement of Theorem 3. Further, it could be noticed from the behavior of the solution of the stochastic system that the curves oscillates around the endemic fixed point of the associated deterministic model (2.1). Under the condition of $\mathbf{R_0^s} > 1$, Figure 5 shows graphically the solution of both the systems. Here again, the concentrations of the bacteria (i.e., $\mathbb{B}_1$ and $\mathbb{B}_2$) are non-zero for all times t. This validates Theorem 3's implications for the deterministic model (2.1). When the related reproduction number for the stochastic system (2.2) is more than unity, the corresponding solution varies about the endemic equilibrium. Thus, in such cases, sound policies must developed which provide strong preventative measures against the various variants of the bacteria in order to control the spread of different strains and their concentrations within the population.



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

(c) $\mathbb{Q}(t)-$Population **(d)** $\mathbb{R}(t)-$Population

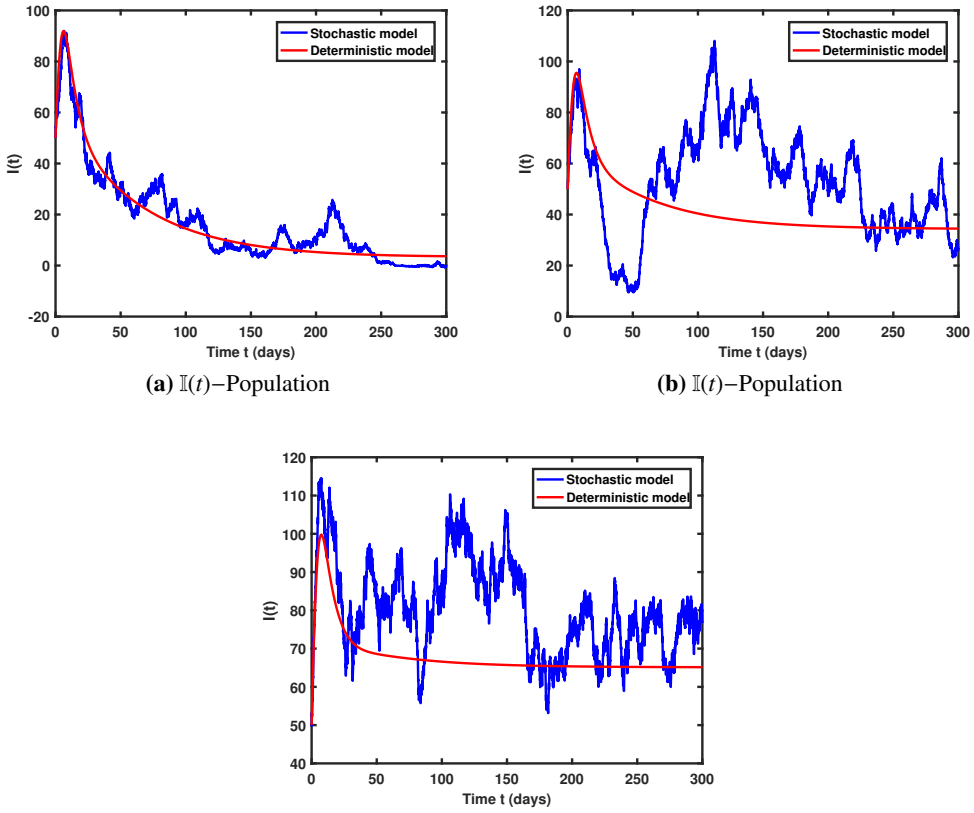
(e) $\mathbb{B}_1(t)-$Population **(f)** $\mathbb{B}_2(t)-$Population

Figure 5. Solution profiles for the various compartments of the deterministic model (2.1) and stochastic model (2.2).

8.1. The impact of white noise on $\mathbb{I}$-class

The impact on the class $\mathbb{I}$ of the intensity of the white noises corresponding to system (2.2) is shown in Figure 6. These figures suggest that increasing the values of γ_i for $i = 1, \cdots, 6$ leads towards the extinction of the disease. This means, the size of the infected class approaches zero as we increase the values of the intensity of the noises. Further, this indicates that for small values of the intensities, the infected compartment oscillates around the endemic steady state $\mathbb{I}^\star$, which confirms the result of

Theorem 3. Nonetheless, when the white noise terms are large enough, the corresponding solution $\mathbb{I}$, does not oscillate in the vicinity of the EE. This demonstrates that continuous efforts to increase stochastic disruptions through mass recovery of susceptible individuals, as well as effective treatment and care of the affected persons, could significantly lower the spread and circulation of the Cholera virus in the population.



(a) $\mathbb{I}(t)-$Population **(b)** $\mathbb{I}(t)-$Population

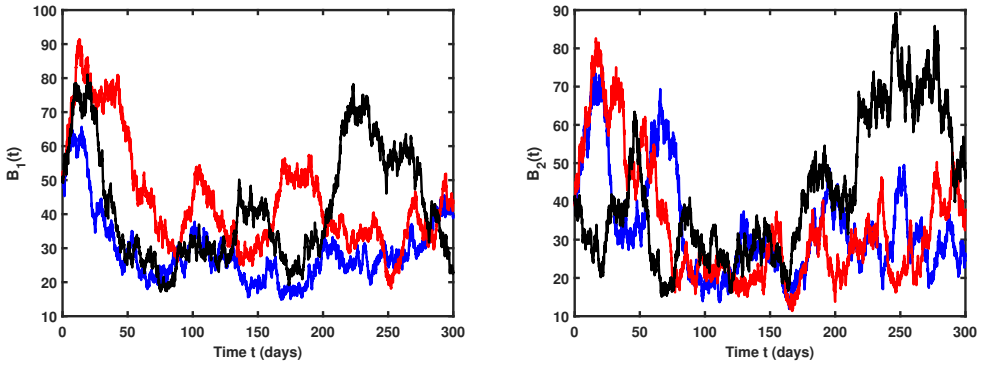
(c) $\mathbb{I}(t)-$Population

Figure 6. Simulations of $\mathbb{I}(t)$ based on the stochastic and deterministic systems, showing the effect of the intensities on the class $\mathbb{I}$, when $\Pi = 1.12, \beta_1 = 0.008, \beta_2 = 0.007, \omega = 0.07, \delta = 0.05, \alpha_1 = 0.01, \mu = 0.005, \alpha_2 = 0.05, \eta_1 = 0.05, \eta_2 = 0.04, d = 0.06$, and $\gamma_1 = 0.50, \gamma_2 = 0.66, \gamma_3 = 0.75, \gamma_4 = 0.35, \gamma_5 = 0.45, \gamma_6 = 0.50$, such that, $\mathbf{R_0^s} > 1$.

8.2. The impact of β_1 and β_2 on $\mathbb{B}_1(t)$ and $\mathbb{B}_1(t)$

Let us consider the values of the parameters as $(\beta_1, \beta_2) = (0.08, 0.06)$ and initial condition $(\mathbb{B}_1(0), \mathbb{B}_2(0)) = (50, 40)$ with different stochastic noises $(\gamma_1, \gamma_2, \gamma_3, \gamma_4, \gamma_5, \gamma_6) = (0.45, 0.15, 0.68, 0.25, 0.65, 0.45)$. The remaining values of the parameter are the same as were taken in the simulation of Figure 5. The concentrations of the Vibrio cholerae both in food and water, and the mean infected relative cure of model (2.2) along with the curves (obtained from simulating the deterministic model) are graphically depicted in Figure 7. By increasing the values of the transmission coefficients, the corresponding concentrations of the bacteria increase, and vice verse. Consequently, the size of the infected class increases and decreases. This suggests that by limiting the value of β_1 and

β_2, we can control and prevent Cholera in the long run. The disease can be eradicated if the stochastic disturbances are assumed to be sufficiently high and the contact terms are reduced.



(a) $\mathbb{B}_1(t)-$individuals **(b)** $\mathbb{B}_2(t)-$individuals

Figure 7. The plot shows the impact of β_1 and β_2 on the dynamics of $\mathbb{B}_1(t)$ and $\mathbb{B}_2(t)$ based on stochastic system (2.2).

9. Concluding remarks and future directions

The present work was about an acute diarrhea that is caused by consuming unclean water or food, and this infection is commonly known as cholera. Initially, the model for cholera infection is formulated by including the instants at which a person contracts the disease and the instant at which the individual exhibits the symptoms soon after consuming the polluted food and water. First of all, we developed the model by using the deterministic approach, and then it was converted to a system of stochastic differential equations. In addition to the biological interpretation of the stochastic model, we proved the existence of the possible steady states of the associated deterministic model and accordingly, the stability theorems are presented therein. It is shown that the proposed stochastic model has the unique global solution, and adequate criteria are achieved by using the Lyapunov function theory which guarantees that the model has an ergodic stationary distribution for $\mathbf{R_s^0} > 1$. If $R_s < 1$, we proved that the disease will tend be eliminate from of the community. We provided some graphical solutions to better validate the analytical results that were acquired. The result of this research can offer a solid theoretical foundation for a comprehensive knowledge of other chronic communicable diseases like Cholera. Moreover, our approach seeks to offer a technique for creating Lyapunov functions that may be utilized in investigating the stationary distribution of the models having stochastic perturbations of non-linear type.

The findings of this research further suggest that in contrast to the spreading of Cholera from human to human, studying the transmission of the disease via water and food is much more beneficial, as it decreases the chance of cross contamination. Nevertheless, researchers have noted that in order to significantly reduce the risk, all three of these aspects must be addressed together. In future research, the authors hope to incorporate other disease-related characteristics into the model, such as age and spatial impacts. It is also intended to incorporate different response functions into the study in the near future.

Use of AI tools declaration

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

Acknowledgments

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Conflict of interest

The authors declare that there are no conflicts of interest.

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