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Stationary Distribution of Stochastic SEIRS Epidemic Model with Saturated Incidence Rate and Saturated Treatment Function

S. Saravanan*, *C. Monica*

Department of Mathematics, School of Advanced Sciences (SAS), Vellore Institute of Technology, Vellore, India.
*Corresponding Author.

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Abstract

This investigation aims to enhance and broaden the mathematical model that governs a dynamic stochastic SEIRS (Susceptible, Exposed, Infective, and Recovery) epidemic. This complex model integrates crucial components, including a saturated incidence rate and saturated treatment function, which are fundamental in molding epidemic dynamics. The objective is to explore the presence and uniqueness of a positive global solution through the application of a meticulously designed Lyapunov function, facilitating a more profound analysis of the intricacies of the systems. This analytical framework enables us to uncover the interactions among disease transmission, treatment dynamics, and stochastic influences. This theoretical framework assumes that treatment responses are directly related to incidence cases within the healthcare system as long as they remain within the system. A key aspect of our contribution lies in defining the stochastic basic reproduction number $\mathcal{R}_0$ as a critical threshold that determines the course of the epidemic. Under conditions characterized by low noise levels and $\mathcal{R}_0^{\delta} > 1$, it establishes the prerequisites for the appearance of an ergodic stationary distribution, offering insights into the potential long-term trends in disease dissemination. Conversely, in scenarios characterized by high noise intensity $\widetilde{\mathcal{R}_0^{\delta}} < 1$, our analysis sheds light on the inevitable eradication of the disease. To further enhance the theoretical underpinning, our research integrates extensive numerical simulations. These simulations not only confirm the validity of our theoretical findings but also provide a dynamic visualization of the implications of the model. The dual methodology of theoretical analysis and simulations contributes to a nuanced understanding of stochasticity and epidemic dynamics.

Keywords: Extinction, Lyapunov function, Mathematical modelling, Stationary distribution, Stochastic SEIRS epidemic model.

Introduction

Recent research has investigated different epidemic models for preventing and managing infectious diseases, including measles, tuberculosis, and the flu[1,2]. A mathematical model is an important tool for studying the development and effects of infectious diseases in epidemiology[3,4]. In addition to identifying disease trends, analyzing epidemiological studies, and making general predictions about diseases, researchers can also use mathematical models to analyze epidemiological data[5-7].

It is one of the prominent topics of population genetics research to comprehend how mutations and selection interact with each other. A better part of this field's research is devoted to deterministic models,

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while a second major part deals with stochastic models[8,9]. It is possible to formulate deterministic mutation-selection equations as discrete or continuous-time dynamical systems by using methods developed for dynamical systems. In stochastic mutation-selection models, as in the Moran and Wright-Fischer models[10,11], it is also possible to include fluctuations arising from random reproduction over very long-time scales. These fluctuations cannot be captured by deterministic dynamics.

An existing epidemiological model assumes, sometimes incorrectly, that one particular pathogen causes a pandemic. Regardless of this, they ignore the mutations that occur over time, which results in the emergence of different strains of the pathogen. The majority of mutations undertaken have no significant impact on the pathogen's bio-epidemiological behavior[12]. In some cases, pathogen mutations result in diseases that are more contagious, more deadly, and have a higher mortality rate[13]. Our understanding of the structure and mutation processes of pathogens will help us develop better medicines and vaccines. Therefore, it is necessary to incorporate the model in which the effect of pathogen mutations from epidemiological documentation based on tests on populations. This will enhance its accuracy. The spread of epidemics over time is impossible to capture using these models if the infectious pathogen is mutated. As an example, drug resistance to anticancer drugs is systems; tumor cells sensitive to toxic agents may probabilistically mutate into cells resistant to the drug's activity over time. This modeling involves the derivation of a probability distribution for mutant cells, which is an important aspect of it. In this scenario, populations undergo stochastic mutations, births, and deaths. It is helpful to model drug-resistance based on the probability distribution of antidrug tumor cells to transform more effective cancer treatment strategies[14].

The efficacy of treatment in halting the dissemination of various infectious diseases is widely acknowledged for its noteworthy success. It is assumed that the spread of infection is proportional to the number of individuals who are infected in classical epidemic models. However, in general, the rate of recovery is influenced by medical resources, including drugs, immunizations, hospital beds, isolation areas, and the effectiveness of the treatment. It is very important to adopt the most appropriate treatment method for a given disease since every country or community has limited resources for treating a particular disease. The following is a constant treatment introduced by Wang and Ruan[15] in a SIR model:

$$F(\mathcal{I}) = \begin{cases} \rho\mathcal{I}, & \mathcal{I} > 0 \\ 0, & \mathcal{I} = 0 \end{cases}. \qquad 1$$

It is denoted by ρ as a positive constant, and I as the number of infected individuals. This represented a constrained ability to treat. Furthermore, Wang[16] examined the following piecewise linear treatment functions:

$$F(I) = \begin{cases} \rho\mathcal{I}, & 0 \le \mathcal{I} \le \mathcal{I}_0 \\ l_0, & \mathcal{I} \ge 0 \end{cases}, \qquad 2$$

where $l_0 = \rho I_0, \rho$ and I_0 are positive constants. Aside from this, the treatment efficiency will be seriously affected by the delay in treating infected individuals. A saturated treatment function was also proposed by Zhang and Liu [17]:

$$T(\mathcal{I}) = \frac{\rho\mathcal{I}}{1+b\mathcal{I}}.$$

Where $\rho > 0, \quad b > 0, \quad '\rho'$is a cure rate and the parameter $'b'$ evaluates the impact of treatment in the infected delays. When the number of infected individuals is very low, this saturated treatment function produces near linear results, while for higher values of I, it approaches a fixed limit[18-20]. Additionally, this treatment function has a continuous and finite value for each feasible value of I. It has been extensively discussed in many literatures how to model epidemic dynamics using SIR or SIS models with different types of incidence rates and treatment functions[21-23]. The saturated treatment function and saturated incidence rate, however, have not been studied as much in the stochastic SEIRS epidemic models.

This study has serious concerns regarding a stochastic SEIRS epidemic model including the recovered compartment R(t) in S(t) along with saturated incidence rates and saturated treatment functions. This paper explores the nuances of virus evolution by analyzing and integrating an evolutionary epidemic model with stochastic SEIRS models. This integration captures the stochastic evolution of susceptible, exposed, infected, and

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recovered populations. Over time, individuals within this population engage in stochastic interactions and undergo state transitions. This perspective characterizes the infectious process as a white noise interaction, enabling easy recovery from infection through treatment. Additionally, those who recover from the disease undergo treatment for disease mutation and revert to the vulnerable stage upon recovery. In this study, our aim is to demonstrate the substantial effects of saturation treatment on a stochastic SEIRS epidemic model.

This paper continues in the following manner: The SEIRS treatment function epidemic model Eq.4 is described in Section 2 of the paper. The global positivity and uniqueness of solutions for the stochastic model Eq 4 with a positive initial value are established in Section 3. In Section 4, by constructing a stochastic Lyapunov function to fit the solutions of the system Eq 4, demonstrating the ergodic stationary distribution existence and its uniqueness. The condition for the extinction of the infections is constructed in Section 5. The theoretical results are based on examples and numerical calculations in section 6. A brief discussion and future work of the main findings are presented in Section 7.

Model

This paper introduces an exposed class to an epidemic model, aiming to elucidate the dynamic aspects of the epidemic and their implications. Within the SEIR paradigm, individuals highly susceptible to the disease transition into an exposed compartment upon contact with an infectious person. Notably, there is a subsequent non-infectious period post-exposure, during which the individual remains non-contagious until the incubation period concludes. Additionally, individuals who have recovered from the disease, having undergone treatment and gained immunity, are permanently protected against reinfection [24-26]. Consequently, the deterministic SEIRS epidemic model, featuring a saturated incidence rate and saturated treatment function, can be formulated as follows:

$$\frac{d\mathcal{S}}{dt} = \Theta - \frac{\beta\mathcal{S}\mathcal{I}}{1+\psi\mathcal{I}} - \mu\mathcal{S} + \eta\mathcal{R},$$
$$\frac{d\mathcal{E}}{dt} = \frac{(1-\nu)\beta\mathcal{S}\mathcal{I}}{1+\psi\mathcal{I}} - (\mu+\varepsilon)\mathcal{E},$$
$$\frac{d\mathcal{I}}{dt} = \varepsilon\mathcal{E} + \frac{\nu\beta\mathcal{S}\mathcal{I}}{1+\psi\mathcal{I}} - \frac{(1+\phi)\gamma\mathcal{I}}{(1+b\mathcal{I})} - (\mu+\delta)\mathcal{I}, \quad 3$$
$$\frac{d\mathcal{R}}{dt} = \frac{(1-\phi)\gamma\mathcal{I}}{1+b\mathcal{I}} - (\mu+\eta)\mathcal{R},$$

by the conditions of non-negative terms $(\mathcal{S}(0), \mathcal{E}(0), \mathcal{I}(0), \mathcal{R}(0)) \geq 0$. The detailed environmental illustrations of the parameters are given in Tables 1 and 2.

Table 1. Model Variables and Description

Variables	Description
$\boldsymbol{\mathcal{S}(t)}$	Susceptible population
$\boldsymbol{\mathcal{E}(t)}$	Exposed population
$\boldsymbol{\mathcal{I}(t)}$	Infected population
$\boldsymbol{\mathcal{R}(t)}$	Recovered population

Table 2. Model Parameters and Description

Variables	Description
$\boldsymbol{\Theta}$	Recruitment rate
$\boldsymbol{\beta}$	Transmission rate
$\boldsymbol{\varphi}$	Saturated factor that measures inhibitory effect
$\boldsymbol{\nu}$	Fast progression rate
$\boldsymbol{\varepsilon}$	Rate at which peoples become infectious
$\boldsymbol{\mu}$	Natural death rate
$\boldsymbol{\delta}$	Disease induced death rate
$\boldsymbol{\phi}$	Failure treatment rate
$\boldsymbol{\gamma}$	Recovery rate
$\boldsymbol{b}$	Treatment effect of infected delayed measured by saturated factor
$\boldsymbol{\eta}$	Loss of immunity

At time t, the population densities for susceptible, exposed, infected, and removed are plotted as $S(t)$,$E(t)$,$I(t)$, and $R(t)$. In the real world, abundant and unpredictable environmental noise hurts population models. The mathematical modelling of ecological systems is also limited by deterministic systems, regardless of environmental fluctuations. There is a lot of difficulty in fitting data to them perfectly [27]. Thus, stochastic models are receiving growing attention from researchers. Additionally, there have been discussions of various stochastic perturbations with population models [28,29]. A random fluctuation in the population dynamics model is inevitable in real life. An epidemic model of a stochastic SIRS epidemic was developed by Li et al.[30] to understand the mechanism of influenza A virus transmission. A study by Feng T et al.[31] demonstrated how environmental noise can change qualitative behaviors. Our paper considers a stochastic SEIRS epidemic model with a saturated

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incidence rate based on the above motivations[25] $\frac{\beta\mathcal{S}\mathcal{I}}{1+\varphi\mathcal{I}}$ and continuously differentiable treatment function $T(\mathcal{I}) = \frac{r\mathcal{I}}{1+b\mathcal{I}}$ produced by[32] incorporating the recovered compartment $\mathcal{R}(t)$ in $\mathcal{S}(t)$ with the saturation phenomenon of constrained medical resources. Where $\frac{1}{1+\varphi\mathcal{I}}$ measures the inhibition effect resulting from the behavioral changes of the affected people when their number of growths from the congested affected of the infected individuals, while $\beta\mathcal{S}(t)\mathcal{I}(t)$ measures the infection force of the disease. If establishing $\Upsilon = \{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}): \mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R} \leq \frac{\Theta}{\mu}, (\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}) \geq 0\}$. There is no problem verifying that region Υ is positively invariant concerning model Eq.3. The reproduction number is:

$$\mathcal{R}_0 = \frac{\beta\Theta(\varepsilon+\mu\nu)}{\mu(\varepsilon+\mu)((1-\phi)\gamma+\delta+\mu)}.$$

In a susceptible population, it is the average number of secondary transmissions of a single infected person. It displays the behavior of the solution according to the value of the threshold $\mathcal{R}_0$:

- In model Eq 3, if $\mathcal{R}_0 < 1$, there exists a unique disease-free equilibrium $\mathcal{E}_0 = \left(\frac{\Theta}{\mu}, 0,0,0\right)$, which is globally asymptotically stable.
- When $\mathcal{R}_0 > 1$ in addition to E_0, model Eq 3, contains a global asymptotically stable positive endemic equilibrium $\mathcal{E}^*(\mathcal{S}^*,\mathcal{E}^*,\mathcal{I}^*,\mathcal{R}^*)$.

The goal of this study is to investigate whether the stochastic $SEIRS$ epidemic model solution has an ergodic stationary distribution. In our approach, stochastic perturbations are incorporated. Keeping these facts in mind motivates us to keep working hard. It is assumed in this paper that stochastic perturbations are of the white noise type, where S, E, I, and R are directly proportional to each other, with $\frac{d\mathcal{S}}{dt}$, $\frac{d\mathcal{E}}{dt}$, $\frac{d\mathcal{I}}{dt}$, and $\frac{d\mathcal{R}}{dt}$ influencing the system Eq 3. Considering the above, proposing the following stochastic SEIRS epidemic model that integrates saturated treatment and contact rates:

$$d\mathcal{S} = \left[\Theta - \frac{\beta\mathcal{S}\mathcal{I}}{1+\psi\mathcal{I}} - \mu\mathcal{S} + \eta\mathcal{R}\right]dt + \varrho_1\mathcal{S}d\mathcal{B}_1(t),$$

$$d\mathcal{E} = \left[\frac{(1-\nu)\beta\mathcal{S}\mathcal{I}}{1+\psi\mathcal{I}} - (\mu+\varepsilon)\mathcal{E}\right]dt + \varrho_2\mathcal{E}d\mathcal{B}_2(t), \quad 4$$

$$d\mathcal{I} = \left[\varepsilon\mathcal{E} + \frac{\nu\beta\mathcal{S}\mathcal{I}}{1+\psi\mathcal{I}} - \frac{(1+\phi)\gamma\mathcal{I}}{(1+b\mathcal{I})} - (\mu+\delta)\mathcal{I}\right]dt + \varrho_3\mathcal{I}d\mathcal{B}_3(t),$$

$$d\mathcal{R} = \left[\frac{(1-\phi)\gamma\mathcal{I}}{1+b\mathcal{I}} - (\mu+\eta)\mathcal{R}\right]dt + \varrho_4\mathcal{R}d\mathcal{B}_4(t),$$

where $\mathcal{B}_i$'s are standard one- dimentional independent Brownian motion, $\varrho_i > 0$ is the intensity of the white noise, $(i = 1,2,3,4)$ that is specified on a complete probability area $(\Omega,\mathcal{F},P)$ with $\{\mathcal{F}\}_{t\in\mathbb{R}_+^4}$ filtration fulfilling the normal requirements, $\mathcal{F}_0$ contains all P-null sets, whereas $\{\mathcal{F}\}_{t\in\mathbb{R}_+^4}$ value is increasing and continuous [33]. In all cases, the coefficients are not negative, $\Theta > 0$. In this study, saturated treatment rates and contact rates are discussed with the stochastic $SEIRS$ epidemic model. To determine whether the model has a stationary ergodic distribution, the model's dynamical properties will be investigated.

Existence of a Unique Global Solution

The investigation into the dynamics of an epidemic model necessitates a comprehensive evaluation of the solution's global and positive aspects. In summary, the subsequent findings confirm the existence and uniqueness of a positive global solution. To delve into the dynamic behavior of model Eq 4, a preliminary analysis of its static features is indispensable for a thorough understanding. Firstly, considering stochastic differential equations in d-dimensions:

$d\mathcal{X} = f(\mathcal{X}(t),t)dt + g(\mathcal{X}(t),t)d\mathcal{B}(t)$ for $t \geq t_0$,

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

$$\mathcal{L} = \frac{\partial}{\partial t} + \sum_{i=1}^{d} f_i(\mathcal{X},t)\frac{\partial}{\partial\mathcal{X}} + \frac{1}{2}\sum_{i,j=1}^{d}[g^T(\mathcal{X},t)g(\mathcal{X},t)]_{ij}\frac{\partial^2}{\partial\mathcal{X}_i\partial\mathcal{X}_j}.$$

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

$$\mathcal{L}\mathcal{V}(\mathcal{X},t) = \mathcal{V}_t(\mathcal{X},t)f(\mathcal{X},t) + \frac{1}{2}trace[g^T(\mathcal{X},t)\mathcal{V}_{\mathcal{X}\mathcal{X}}(\mathcal{X},t)g(\mathcal{X},t)].$$

Where $\mathcal{V}_t = \frac{\partial\mathcal{V}}{\partial t}$, $\mathcal{V}_{\mathcal{X}} = \left(\frac{\partial\mathcal{V}}{\partial\mathcal{X}_1}, \frac{\partial\mathcal{V}}{\partial\mathcal{X}_2}, \ldots, \frac{\partial\mathcal{V}}{\partial\mathcal{X}_d},\right)$, $\mathcal{V}_{\mathcal{X}\mathcal{X}} = \left(\frac{\partial^2}{\partial\mathcal{X}_i\partial\mathcal{X}_j}\right)_{d\times d}$. Thus, by Ito's formula, if $\mathcal{X}_t \in \mathbb{R}^d$, then$d\mathcal{V}(\mathcal{X}(t),t) = \mathcal{L}\mathcal{V}(\mathcal{X}(t),t)dt + \mathcal{V}_{\mathcal{X}}(\mathcal{X}(t),t)g(\mathcal{X}(t),t)d\mathcal{B}(t)$.

There are several theories of stationary distributions that will be discussed in the next section (Hasminskii[34]).

Theorem 1: For any initial value $(\mathcal{S}(0),\mathcal{E}(0),\mathcal{I}(0),\mathcal{R}(0)) \in \mathbb{R}_+^4$, there is a unique

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solution $\mathcal{S}(t),\mathcal{E}(t),\mathcal{I}(t),\mathcal{R}(t))$ of the model Eq 3, on $t>0$ and the solution will remain in $\mathbb{R}_+^4$ with probability one.

Proof: It is known that for any initial value $\big(\mathcal{S}(0),\mathcal{E}(0),\mathcal{I}(0),\mathcal{R}(0)\big)$ the coefficients in the system Eq.3, satisfy the local Lipschitz condition, and that a unique local solution $\mathcal{S}(t),\mathcal{E}(t),\mathcal{I}(t),\mathcal{R}(t))$ can be found on $[0,\tau^*)$ *almost surely*, where τ^* is the explosion time [33]. The solution must be universally applicable. Only one thing needs to be proved: $\tau^*=+\infty$ almost certainly exists. Now, let $\ell_0>0$ be a sufficiently large number such that $\big(\mathcal{S}(0),\mathcal{E}(0),\mathcal{I}(0),\mathcal{R}(0)\big)$ lies inside the interval. For each integer $\left[\frac{1}{\ell_0},\ell_0\right]$ define the stopping time [33],

$$\tau_\ell=\inf\Big\{t\in[0,\tau^*):\mathcal{S}(t)\notin\left(\tfrac{1}{\ell},\ell\right)\ or\ \mathcal{E}(t)\notin\left(\tfrac{1}{\ell},\ell\right)\ or\ \mathcal{I}(t)\notin\left(\tfrac{1}{\ell},\ell\right)\ or\ \mathcal{R}(t)\notin\left(\tfrac{1}{\ell},\ell\right)\Big\},$$

a typical format is set to $\emptyset=+\infty$ (Under normal conditions, $\emptyset$ denotes the empty set). It is clear that τ_ℓ is rising in ℓ and $\tau_\infty<\tau^*$. Then $\tau_\ell=\lim_{\ell\to\infty}\tau_\ell$ makes sense, and $\tau_\ell<\tau^*$ a. s. An important step is to construct a Lyapunov function. Consider that $\tau_\infty<\infty$, then there are two constants $\mathcal{T}>0$ and $\epsilon\in(0,1)$ such that $\mathcal{P}\{\tau_\infty\le\mathcal{T}\}>\epsilon$. Therefore, there is an integer $\ell_1\ge\ell_0$ such that, $\mathcal{P}\{\tau_\infty<\mathcal{T}\}\ge\epsilon\ \forall\ \ell\ge\ell_1$. Thus, the term is defined by us a $\mathbb{C}^2$ function $\mathcal{V}:\mathbb{R}_+^4\to\mathbb{R}_+$ as follows:

$$\mathcal{V}(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})=\mathcal{S}-1-\ln\mathcal{S}+\mathcal{E}-1-\ln\mathcal{E}+\mathcal{I}-1-\ln\mathcal{I}+\mathcal{R}-1-\ln\mathcal{R}. \quad 5$$

Applying the Ito formula it will be,

$$d\mathcal{V}=p\mathcal{V}dt+\varrho_1(\mathcal{S}-1)d\mathcal{W}_1(t)+\varrho_2(\mathcal{E}-1)d\mathcal{W}_2(t)+\varrho_3(\mathcal{I}-1)d\mathcal{W}_3(t)+\varrho_4(\mathcal{R}-1)d\mathcal{W}_4(t). \quad 6$$

Therefore,

$$\mathcal{LV}=\left(1-\tfrac{1}{\mathcal{S}}\right)\left(\Theta-\tfrac{\beta\mathcal{S}\mathcal{I}}{1+\psi\mathcal{I}}-\mu\mathcal{S}+\eta\mathcal{R}\right)+\left(1-\tfrac{1}{\mathcal{E}}\right)\left(\tfrac{(1-\nu)\beta\mathcal{S}\mathcal{I}}{1+\psi\mathcal{I}}-(\mu+\varepsilon)\mathcal{E}\right)+\left(1-\tfrac{1}{\mathcal{I}}\right)\left(\varepsilon\mathcal{E}+\tfrac{\nu\beta\mathcal{S}\mathcal{I}}{1+\psi\mathcal{I}}-\tfrac{(1+\phi)\gamma\mathcal{I}}{(1+b\mathcal{I})}-(\mu+\delta)\mathcal{I}\right)+\left(1-\tfrac{1}{\mathcal{R}}\right)\left(\tfrac{(1-\phi)\gamma\mathcal{I}}{1+b\mathcal{I}}-(\mu+\eta)\mathcal{R}\right)$$

$$=\Theta+4\mu+\epsilon+\eta-\mu(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})-\frac{\Theta}{\mathcal{S}}-\frac{\eta\mathcal{R}}{\mathcal{S}}-\frac{\epsilon\mathcal{E}}{\mathcal{I}}-\frac{\beta\mathcal{S}\mathcal{I}}{1+\varphi\mathcal{I}}+\frac{\beta\mathcal{I}}{1+\varphi\mathcal{I}}+\frac{(1-\vartheta)\beta\mathcal{S}\mathcal{I}}{1+\varphi\mathcal{I}}-\frac{(1-\vartheta)\beta\mathcal{S}\mathcal{I}}{\mathcal{E}(1+\varphi\mathcal{I})}+\frac{(1-\phi)\gamma\mathcal{I}}{\mathcal{R}(1+b\mathcal{I})}-\frac{\vartheta\beta\mathcal{S}\mathcal{I}}{1+\varphi\mathcal{I}}-\frac{\vartheta\beta\mathcal{S}}{1+\varphi\mathcal{I}}+\frac{(1-\phi)\gamma}{\mathcal{R}(1+b\mathcal{I})}+\frac{\varrho_1^2+\varrho_2^2+\varrho_3^2+\varrho_4^2}{2}$$

$$\le\Theta+4\mu+\epsilon+\eta+\tfrac{\varrho_1^2+\varrho_2^2+\varrho_3^2+\varrho_4^2}{2}$$

$$\le\mathcal{K}_0,$$

where $\mathcal{K}_0$ is a suitable constant that is independent. The remainder of the proof of Theorem 1 is similar to Mao et al.[34] and it's neglected.

The asymptotic behavior of the equilibrium solution for a disease-free system

The various stability concepts have been explored in the context of disease-free equilibrium. Stochastically asymptotic stability, focusing on probabilities near equilibrium, elucidates the asymptotic trajectory of sample paths within a system, providing insight into almost sure behaviors. Assuming global stochastic asymptotic stability for E_0, one can employ a Lyapunov function to identify an equilibrium devoid of disease. Analyzing the stochastic model Eq 4, up to asymptotic equilibrium allows for a comprehensive examination of its asymptotic behavior.

Theorem 2: If $\mathcal{E}_0=\left(\frac{\Theta}{\mu},0,0,0\right)$ is a disease-free equilibrium of the stochastic model Eq 4, is globally asymptotically stable on $\mathcal{D}$. Then $\mathcal{R}_0\le1$.

Proof. Now define the function $\mathcal{C}^2$ is $\mathcal{V}:\mathbb{R}_+^4\to\mathbb{R}_+$ as follows,

$$\mathcal{V}(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})=\ln(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})^2+\ln\mathcal{I}.$$

The Lyapunov function generator $\mathcal{L}$ on $\mathcal{V}$ provides the following result,

$$\mathcal{V}(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})=\left(\Theta-\tfrac{\beta\mathcal{S}\mathcal{I}}{1+\psi\mathcal{I}}-\mu\mathcal{S}+\eta\mathcal{R}\right)\left(\tfrac{2}{(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})}\right)+\left(\tfrac{(1-\nu)\beta\mathcal{S}\mathcal{I}}{1+\psi\mathcal{I}}-(\mu+\varepsilon)\mathcal{E}\right)\left(\tfrac{2}{(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})}\right)+\left(\varepsilon\mathcal{E}+\tfrac{\nu\beta\mathcal{S}\mathcal{I}}{1+\psi\mathcal{I}}-\tfrac{(1+\phi)\gamma\mathcal{I}}{(1+b\mathcal{I})}-(\mu+\delta)\mathcal{I}\right)\left(\tfrac{2}{(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})}+\tfrac{1}{\mathcal{I}}\right)+\left(\tfrac{(1-\phi)\gamma\mathcal{I}}{1+b\mathcal{I}}-(\mu+\eta)\mathcal{R}\right)\left(\tfrac{2}{(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})}\right)$$

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$$+\frac{1}{2}\left(\frac{-2}{(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})^2}\right)\varrho_1^2\mathcal{S}^2 - \left(\frac{2}{2(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})^2}\right)\varrho_2^2\mathcal{E}^2 + \frac{1}{2}\left(\frac{-2}{(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})^2} - \frac{1}{\mathcal{I}^2}\right)\varrho_3^2\mathcal{I}^2 - \left(\frac{2}{2(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})^2}\right)\varrho_4^2\mathcal{R}^2.$$

In order to simplify that $\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R}\leq 1$, in our case,

$$\mathcal{LV} = -2\mu + \left(\frac{\varphi\beta\Theta}{\mu} - (1-\phi)\gamma - (\mu+\delta)\right) - \frac{1}{2}\sigma_3^2 - \frac{\sigma_1^2\mathcal{S}^2+\sigma_2^2\mathcal{E}^2+\sigma_3^2\mathcal{I}^2+\sigma_4^2\mathcal{R}^2}{(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})^2}.$$

Then $\mathcal{LV}(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})$ becomes a negative definite on $\mathcal{D}$ with a condition $\mathcal{R}_0\leq 1$. The disease will persist here, so there is only one condition for $\mathcal{R}_0$, and the other two are automatically satisfied. The disease-less equilibrium solution $\mathcal{E}_0=\left(\frac{\Theta}{\mu},0,0,0\right)$ of the stochastic model Eq 4, is globally asymptotically stable on $\mathcal{D}$.

Remark 1: The above Theorem 2 provides that the disease cases exist when $\mathcal{R}_0<1$. From the stability of the condition $\left((1-\phi)\gamma+\mu+\mathcal{S}\right)\geq\frac{\varphi\beta\Theta}{\mu}$, then the disease will disappear. Taking into account the $\mathcal{R}_0=\frac{\beta\Theta(\varepsilon+\mu\nu)}{\mu(\varepsilon+\mu)((1-\phi)\gamma+\delta+\mu)}<1$. Then $\mathcal{E}_0=\left(\frac{\Theta}{\mu},0,0,0\right)$ of the stochastic model Eq 4, is asymptotically stable in the large.

Remark 2: According to Theorem 2, the stochastic model Eq 4 will approach disease-less equilibrium if the white noise intensity is high enough. Since the intensity of white noise ϱ_i (for i $=1,2,3,4$) is small, the solutions of stochastic model Eq 4, will generally fluctuate around the diseases-less equilibrium of deterministic model Eq 3.

Existence of ergodic stationary distribution

and a function $f(.)$ with respect to a measure $\omega(.)$ is an integral function.

In the analysis of epidemic dynamical models, assessing the persistence and prevalence of a disease in a population holds paramount importance. Deterministic models often establish global attractor or global asymptotic stability by focusing on their endemic equilibrium. Notably, model Eq 4, lacks such equilibrium. This section introduces the concept of an ergodic stationary distribution, grounded in Has'minskii's theory [34], providing evidence for the persistence of the disease.

The following stochastic differential equation describes $\mathcal{X}(t)$ as a homogeneous Markov process in $\mathbb{R}_+^4$.

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

As a result, the diffusion matrix can be defined as follows:

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

Lemma 1: [35] There is a unique ergodic stationary distribution $\omega(.)$ for the Markov process $\mathcal{X}(t)$ if the domain $\mathbb{D}\subset\mathbb{R}_4^+$ has a regular boundary Γ^*, with the following properties:

$\mathcal{H}_1$: there is a positive number $\mathcal{M}$ such that

$$\sum_{i,j=1}^{4}a_{ij}(x)\,\xi_i\xi_j\geq\mathcal{M}|\xi|^2\ for\ x\in\mathbb{D},\xi_i\in\mathbb{R}_+^4\ (i=1,2,3,4).$$

$\mathcal{H}_2$: there exists a non-negative $\mathcal{C}^2$ function $\mathcal{V}(x)$and a positive constant $\mathcal{C}^*$ such that $\mathcal{VL}\leq\mathcal{C}^*$ for any $\mathbb{R}_+^4\backslash\mathbb{D}$. Then

$$\mathcal{P}_{\mathcal{X}}\left\{\lim_{\mathcal{T}\to\infty}\frac{1}{\mathcal{T}}\int_0^{\mathcal{T}}f(\mathcal{X}(t))\,dt=\int_{\mathbb{R}_+^4}f(x)\omega(dx)\right\}=1,$$

Define a parameter value $\mathcal{R}_0^{\mathcal{S}}$ corresponding to the basic reproduction number $\mathcal{R}_0$ of the system Eq 4, as follows:

$$\mathcal{R}_0^{\mathcal{S}}=\frac{\Theta\beta\vartheta\epsilon}{\left(\mu+\frac{\varrho_1^2}{2}\right)\left(\epsilon+\mu+\frac{\varrho_2^2}{2}\right)\left((1-\phi)\gamma+\mu+\delta+\frac{\varrho_3^2}{2}\right)}.$$

Theorem 3: Assume that $\mathcal{R}_0^{\mathcal{S}}>1$, then the model Eq.4, has a unique stationary distribution $\Gamma(.)$ and it has the ergodic property.

Proof: Defining the diffusion matrix for a system

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$$\mathcal{A}=\begin{pmatrix} \varrho_1^2\mathcal{S}^2 & 0 & 0 & 0 & 0\\ 0 & \varrho_2^2\mathcal{E}^2 & 0 & 0 & 0\\ 0 & 0 & \varrho_3^2\mathcal{I}^2 & 0 & 0\\ 0 & 0 & 0 & 0 & \varrho_4^2\mathcal{R}^2 \end{pmatrix}$$

There is a positive number

$$\mathcal{Z}=\min_{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})\in\mathcal{D}}\{\varrho_1^2\mathcal{S}^2,\varrho_2^2\mathcal{E}^2,\varrho_3^2\mathcal{I}^2,\varrho_4^2\mathcal{R}^2\},$$

such that

$$\sum_{i,j=1}^{4}a_{ij}\varsigma_i\varsigma_j=\varrho_1^2\mathcal{S}^2\varsigma_1^2+\varrho_2^2\mathcal{E}^2\varsigma_2^2+\varrho_3^2\mathcal{I}^2\varsigma_3^2+\varrho_4^2\mathcal{R}^2\varsigma_4^2\geq\mathcal{Z}\,|\varsigma|^2,(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})\in\mathcal{D}_\epsilon,\varsigma\in\mathbb{R}^4, \quad 7$$

which indicates that Lemma 1 $(\mathcal{H}_1)$ and Assumption 1(i) are satisfied.

Construct a $\mathcal{C}^2$- function $\mathcal{V}:\mathbb{R}_+^4\to\mathbb{R}$ in the following form

$$\begin{aligned} d\mathcal{V}=\mathcal{K}_0(&\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R}-\alpha_1\ln\mathcal{S}-\alpha_2\ln\mathcal{E}\\ &-\alpha_3\ln\mathcal{I})\\ &+\frac{1}{\theta+1}(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})^{\theta+1}\\ &-\ln\mathcal{S}\,ln-\mathcal{E}\\ &-\ln\mathcal{R}\;+(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})\\ &=\mathcal{K}_0\mathcal{V}_1+\mathcal{V}_2+\mathcal{V}_3+\mathcal{V}_4+\mathcal{V}_5+\mathcal{V}_6. \end{aligned}$$

Where θ is a constant satisfying

$0<\theta<\frac{2}{\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2}$,

$$\alpha_1=\frac{\Theta}{\mu+\frac{\varrho_1^2}{2}},\quad \alpha_2=\frac{\Theta}{\varepsilon+\mu+\frac{\varrho_2^2}{2}}$$

$$\alpha_3=\frac{\Theta}{(1-\phi)\gamma+\delta+\mu+\frac{\varrho_3^2}{2}}.$$

There is an easy way to check that

$$\lim_{i\to\infty,(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})\in\mathbb{R}_+^4\setminus\overline{U_n}}inf\,\Gamma(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})=+\infty,$$

where $\overline{U_k}=\left(\frac{1}{k},k\right)\times\left(\frac{1}{k},k\right)\times\left(\frac{1}{k},k\right)\times\left(\frac{1}{k},k\right)$.

Furthermore, $\Gamma(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})$ is a continuous function. Hence, $\Gamma(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})$ must have a minimum point $\big(\mathcal{S}(0),\mathcal{E}(0),\mathcal{I}(0),\mathcal{R}(0)\big)$ in the interior of $\mathbb{R}_+^4$. Our next step is to define non-negative $\mathcal{C}^2$- function $d\mathcal{V}:\mathbb{R}_+^4\to\mathbb{R}_+$ as follows;

$$d\mathcal{V}(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})=\Gamma(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})-\Gamma(\mathcal{S}_0,\mathcal{E}_0,\mathcal{I}_0,\mathcal{R}_0).$$

As a result of applying the Ito formula, that gives

$$\begin{aligned} \mathfrak{L}\mathcal{V}_1=\Theta&-\frac{\beta\mathcal{S}\mathcal{I}}{1+\varphi\mathcal{I}}-\mu\mathcal{S}+\eta\mathcal{R}+\frac{(1-\vartheta)\beta\mathcal{S}\mathcal{I}}{(1+\varphi\mathcal{I})\mathcal{E}}\\ &-(\varepsilon+\mu)-\frac{\varepsilon\mathcal{E}}{\mathcal{I}}-\frac{\vartheta\beta\mathcal{S}\mathcal{I}}{1+\varphi\mathcal{I}}\\ &-\frac{(1-\phi)\gamma}{1+b\mathcal{I}}-(\mu+\delta)-\alpha_1\frac{\Theta}{\mathcal{S}}\\ &-\alpha_1\frac{\beta\mathcal{I}}{1+\varphi\mathcal{I}}+\alpha_1\mu+\alpha_1\frac{\eta\mathcal{R}}{\mathcal{S}}\\ &+\alpha_1\frac{\varrho_1^2}{2}-\alpha_2\frac{(1-\vartheta)\beta\mathcal{S}\mathcal{I}}{(1+\varphi\mathcal{I})\mathcal{E}}\\ &-\alpha_2(\varepsilon+\mu)-\alpha_3\frac{\varepsilon\mathcal{E}}{\mathcal{I}}-\alpha_3\frac{\vartheta\beta\mathcal{S}}{1+\varphi\mathcal{I}}\\ &-\alpha_3\frac{(1-\phi)\gamma}{1+b\mathcal{I}}-\alpha_3(\mu+\delta)\\ &+\alpha_3\frac{\varrho_2^2}{2}+\alpha_3\frac{\varrho_3^2}{2}\\ =\Theta&-\alpha_1\frac{\Theta}{\mathcal{S}}-\alpha_2\frac{(1-\vartheta)\beta\mathcal{S}\mathcal{I}}{(1+\varphi\mathcal{I})\mathcal{E}}-\alpha_3\frac{\varepsilon\mathcal{E}}{\mathcal{I}}-\alpha_3\frac{\vartheta\beta\mathcal{S}}{1+\varphi\mathcal{I}}\\ &+\alpha_1\left(\mu+\frac{\varrho_1^2}{2}\right)\\ &+\alpha_2\left(\varepsilon+\mu+\frac{\varrho_2^2}{2}\right)\\ &+\alpha_3\left((1-\phi)\gamma+\delta+\mu\right.\\ &\left.+\frac{\varrho_3^2}{2}\right). \end{aligned} \quad 8$$

Using inequality $a+b\geq2\sqrt{ab},\quad a,b>0,$

$$\begin{aligned} \mathfrak{L}V_1=&-2\left(\alpha_2\alpha_3\frac{\varepsilon(1-\vartheta)\beta\mathcal{S}}{1+\varphi\mathcal{I}}\right)^{\frac{1}{2}}\\ &-2\left(\alpha_1\alpha_3\frac{\vartheta\beta\Theta}{1+\varphi\mathcal{I}}\right)^2+\Theta\\ &+\alpha_1\frac{\beta\mathcal{I}}{1+\varphi\mathcal{I}}+\alpha_3\frac{(1-\phi)\gamma}{1+b\mathcal{I}}\\ &+\alpha_1\left(\mu+\frac{\varrho_1^2}{2}\right)+\alpha_2\left(\varepsilon+\mu+\frac{\varrho_2^2}{2}\right)\\ &+\alpha_3\left((1-\phi)\gamma+\delta+\mu+\frac{\varrho_3^2}{2}\right) \end{aligned}$$

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$$\leq -4\left(\frac{\alpha_1\alpha_2\alpha_3(1-\vartheta)\beta\Theta\varepsilon}{1+\varphi\mathcal{I}}\right)^{\frac{1}{4}}+\Theta+\alpha_1\frac{\beta\mathcal{I}}{1+\varphi\mathcal{I}}+\alpha_3\frac{(1-\phi)\gamma}{1+b\mathcal{I}}+\alpha_1\left(\mu+\frac{\varrho_1^2}{2}\right)+\alpha_2\left(\varepsilon+\mu+\frac{\varrho_2^2}{2}\right)+\alpha_3\left((1-\phi)\gamma+\delta+\mu+\frac{\varrho_3^2}{2}\right)$$

$$=-4\left(\frac{\Theta^4\vartheta\beta\varepsilon\mu}{\left(\mu+\frac{\varrho_1^2}{2}\right)\left(\varepsilon+\mu+\frac{\varrho_2^2}{2}\right)\left((1-\phi)\gamma+\delta+\mu+\frac{\varrho_3^2}{2}\right)\wedge(1+\varphi\mathcal{I})}\right)^{\frac{1}{4}}+4\Theta+\alpha_1\frac{\beta\mathcal{I}}{1+\varphi\mathcal{I}}+\alpha_3\frac{(1-\phi)\gamma}{1+b\mathcal{I}}$$

$$=-4\Theta\left\{\left[\frac{\vartheta\beta\varepsilon\mu}{\left(\mu+\frac{\varrho_1^2}{2}\right)\left(\varepsilon+\mu+\frac{\varrho_2^2}{2}\right)\left((1-\phi)\gamma+\delta+\mu+\frac{\varrho_3^2}{2}\right)\wedge(1+\varphi\mathcal{I})}\right]^{\frac{1}{4}}-1\right\}+\alpha_1\frac{\beta\mathcal{I}}{1+\varphi\mathcal{I}}+\alpha_3\frac{(1-\phi)\gamma}{1+b\mathcal{I}}$$

$$=-\vartheta+\alpha_1\frac{\beta\mathcal{I}}{1+\varphi\mathcal{I}}+\alpha_3\frac{(1-\phi)\gamma}{1+b\mathcal{I}}, \qquad 9$$

where,

$$\vartheta=4\Theta\left\{\left[\frac{\vartheta\beta\varepsilon\mu}{\left(\mu+\frac{\varrho_1^2}{2}\right)\left(\varepsilon+\mu+\frac{\varrho_2^2}{2}\right)\left((1-\phi)\gamma+\delta+\mu+\frac{\varrho_3^2}{2}\right)\wedge(1+\varphi\mathcal{I})}\right]^{\frac{1}{4}}-1\right\}>0.$$

Similarly

$$\mathfrak{L}V_2=(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})^{\nu}(\Theta-\mu\mathcal{S}-\mu\mathcal{E}-(\mu-\vartheta)\mathcal{I}-\mu\mathcal{R})+\frac{\nu}{2}(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})^{\nu-1}\times(\varrho_1^2\mathcal{S}^2+\varrho_2^2\mathcal{E}^2+\varrho_3^2\mathcal{I}^2+\varrho_4^2\mathcal{R}^2)$$

$$\leq(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})^{\nu}[\Theta-\mu(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})]+\frac{\nu}{2}(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})^{\nu+1}\times(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)$$

$$=\Theta(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})^{\nu}-\left[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\right](\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})^{\nu+1}$$

$$\leq\Theta-\frac{1}{2}\left[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\right](\mathcal{S}^{\nu+1}+\mathcal{E}^{\nu+1}+\mathcal{I}^{\nu+1}+\mathcal{R}^{\nu+1}). \qquad 10$$

Where

$$\Xi=\sup_{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})\in R_+^4}\left\{\Theta(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})^{\nu}-\frac{1}{2}\left[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\right](\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})^{\nu+1}\right\},$$

there is also the possibility of getting

$$\mathfrak{L}V_3=-\frac{\Theta}{\mathcal{S}}+\frac{\beta\mathcal{I}}{1+\varphi\mathcal{I}}+\mu+\frac{\eta\mathcal{R}}{\mathcal{S}}+\frac{\varrho_1^2}{2}, \qquad 11$$

$$\mathfrak{L}V_4=-\frac{(1-\vartheta)\beta\mathcal{S}\mathcal{I}}{\mathcal{E}(1+\varphi\mathcal{I})}+\mu+\varepsilon+\frac{\varrho_2^2}{2}, \qquad 12$$

$$\mathfrak{L}V_5=-\frac{(1-\phi)\gamma\mathcal{I}}{(1+b\mathcal{I})\mathcal{R}}+\mu+\delta+\eta+\frac{\varrho_4^2}{2}, \qquad 13$$

$$\mathfrak{L}V_6=\Theta-\mu(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R}), \qquad 14$$

our findings

$$\mathfrak{L}V=-PM+\frac{P\alpha_1\beta\mathcal{I}}{1+\varphi\mathcal{I}}+\frac{P\alpha_3(1-\phi)\gamma}{1+b\mathcal{I}}-\frac{1}{2}\left[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\right]\times(\mathcal{S}^{\nu+1}+\mathcal{E}^{\nu+1}+\mathcal{I}^{\nu+1}+\mathcal{R}^{\nu+1})-\frac{\Theta}{\mathcal{S}}+\frac{\beta\mathcal{I}}{1+\varphi\mathcal{I}}+\mu+\frac{\eta\mathcal{R}}{\mathcal{S}}-\frac{(1-\vartheta)\beta\mathcal{S}\mathcal{I}}{\mathcal{E}(1+\varphi\mathcal{I})}+\mu+\varepsilon-\frac{(1-\phi)\gamma\mathcal{I}}{(1+b\mathcal{I})\mathcal{R}}+\mu+\delta+\eta+\Theta-\mu(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})+\frac{\varrho_1^2}{2}+\frac{\varrho_2^2}{2}+\frac{\varrho_4^2}{2}.$$

$$\mathfrak{L}V=-PM+\frac{P\alpha_1\beta\mathcal{I}}{1+\varphi\mathcal{I}}+\frac{P\alpha_3(1-\phi)\gamma}{1+b\mathcal{I}}-\frac{1}{2}\left[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\right]\times(\mathcal{S}^{\nu+1}+\mathcal{E}^{\nu+1}+\mathcal{I}^{\nu+1}+\mathcal{R}^{\nu+1})-\frac{(1-\vartheta)\beta\mathcal{S}\mathcal{I}}{\mathcal{E}(1+\varphi\mathcal{I})}-\frac{(1-\phi)\gamma\mathcal{I}}{(1+b\mathcal{I})\mathcal{R}}-\frac{\Theta}{\mathcal{S}}+\frac{\beta\mathcal{I}}{1+\varphi\mathcal{I}}+\frac{\eta\mathcal{R}}{\mathcal{S}}-\mu(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R})+\Theta+\delta+\eta+\varepsilon+3\mu+\frac{\varrho_1^2+\varrho_2^2+\varrho_4^2}{2}. \qquad 15$$

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In order to construct a closed domain $\mathfrak{D}_\epsilon$ with a bounded boundary, our steps are as follows:

$$\mathfrak{D}_\epsilon = \Big[(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}) \in \mathbb{R}^4_+ : \varepsilon_1 < \mathcal{S} < \frac{1}{\varepsilon_1}, \varepsilon_2 < \mathcal{E} < \frac{1}{\varepsilon_2}, \varepsilon_1 < \mathcal{S} < \frac{1}{\varepsilon_1}, \varepsilon_2 < \mathcal{S} < \frac{1}{\varepsilon_2}\Big],$$

where $0 < \varepsilon_1, \varepsilon_2 < 1$ is a sufficiently small constant. In the set $\mathbb{R}^4_+ \setminus \mathfrak{D}_\epsilon$, it is possible to choose ϵ small enough to meet the following conditions.

$$-\frac{\Theta}{\varepsilon_1} + H \le -1, \quad 16$$

$$-P\left(M - \frac{\alpha_1\beta\varepsilon_2}{\varphi} - \frac{\alpha_3(1-\phi)\gamma}{b}\right) + G \le -1, \quad 17$$

$$-2\left(\frac{\alpha_1 P\varepsilon_1\mu\beta}{\varphi}\right)^{\frac{1}{2}} + T \le -1, \quad 18$$

$$-\frac{(1-\phi)\gamma}{b\varepsilon_2} + \frac{P\alpha_1\beta}{\varphi} + F \le -1, \quad 19$$

$$-\frac{1}{4}\left[\mu - \frac{\vartheta}{2}(\varrho_1^2 \vee \varrho_1^2 \vee \varrho_2^2 \vee \varrho_3^2 \vee \varrho_4^2)\right]\frac{1}{\varepsilon_1^{\vartheta+1}} + J \le -1, \quad 20$$

$$-\frac{1}{4}\left[\mu - \frac{\vartheta}{2}(\varrho_1^2 \vee \varrho_1^2 \vee \varrho_2^2 \vee \varrho_3^2 \vee \varrho_4^2)\right]\frac{1}{\varepsilon_1^{2(\vartheta+1)}} + W \le -1, \quad 21$$

$$-\frac{1}{4}\left[\mu - \frac{\vartheta}{2}(\varrho_1^2 \vee \varrho_1^2 \vee \varrho_2^2 \vee \varrho_3^2 \vee \varrho_4^2)\right]\frac{1}{\varepsilon_2^{\vartheta+1}} + X \le -1, \quad 22$$

$$-\frac{1}{4}\left[\mu - \frac{\vartheta}{2}(\varrho_1^2 \vee \varrho_1^2 \vee \varrho_2^2 \vee \varrho_3^2 \vee \varrho_4^2)\right]\frac{1}{\varepsilon_2^{2(\vartheta+1)}} + Y \le -1, \quad 23$$

Here H, G, T, F, J, W, X, and Y are the positive constants that are given explicitly in the expression Eq 16 to Eq 23. For our convenience. It is possible to divide $\mathbb{R}^4_+ \setminus \mathcal{D}_\epsilon$ into eight domains.

$\mathfrak{D}_1 = \{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}) \in \mathbb{R}^4_+ : 0 < \mathcal{S} < \varepsilon_1\}$,
$\mathfrak{D}_2 = \{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}) \in \mathbb{R}^4_+ : 0 < \mathcal{I} < \varepsilon_1, \mathcal{S} \ge \varepsilon_1\}$,
$\mathfrak{D}_3 = \{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}) \in \mathbb{R}^4_+ : \mathcal{S} \ge \varepsilon_1 \mathcal{I} \ge \varepsilon_1, 0 < \mathcal{E} < \varepsilon_2\}$,
$\mathfrak{D}_4 = \{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}) \in \mathbb{R}^4_+ : 0 < \mathcal{R} < \varepsilon_2, \mathcal{I} \ge \varepsilon_1\}$,
$\mathfrak{D}_5 = \left\{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}) \in \mathbb{R}^4_+ : \mathcal{S} > \frac{1}{\varepsilon_1}\right\}$,
$\mathfrak{D}_6 = \left\{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}) \in \mathbb{R}^4_+ : \mathcal{I} > \frac{1}{\varepsilon_1}\right\}$,
$\mathfrak{D}_7 = \left\{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}) \in \mathbb{R}^4_+ : \mathcal{E} > \frac{1}{\varepsilon_2}\right\}$,
$\mathfrak{D}_8 = \left\{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}) \in \mathbb{R}^4_+ : \mathcal{R} > \frac{1}{\varepsilon_2}\right\}$.

Apparently, $\mathbb{R}^4_+ \setminus \mathcal{D}_\epsilon = \mathfrak{D}_1 \cup \mathfrak{D}_2 \cup \ldots \cup \mathfrak{D}_8$. Our next step will be to demonstrate that $\mathfrak{L}V(S, E, I, R) \le -1$ for any $\mathbb{R}^4_+ \setminus \mathcal{D}_\epsilon$.

Thus, it must be proven in each of the eight domains listed above.

Case-1: Suppose that $(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}) \in \mathfrak{D}_1$, according to Eq 16, here are the results

$$\begin{aligned}\mathfrak{L}V = &-\frac{\Theta}{\mathcal{S}} + \frac{P\alpha_1\beta\mathcal{I}}{1+\varphi I} \\ &-\frac{1}{2}\left(\mu - \frac{\vartheta}{2}(\varrho_1^2 \vee \varrho_2^2 \vee \varrho_3^2 \vee \varrho_4^2)\right) \\ &\times (\mathcal{S}^{\nu+1} + \mathcal{E}^{\nu+1} + \mathcal{I}^{\nu+1} + \mathcal{R}^{\nu+1}) \\ &+ \Theta + 3\mu + \eta + \varepsilon + \delta \\ &+ \frac{\varrho_1^2 + \varrho_2^2 + \varrho_4^2}{2} \\ \le &-\frac{\Theta}{\mathcal{S}} + H \le -\frac{\Theta}{\varepsilon_1} + H \le -1, \end{aligned} \quad 24$$

where,

$$\begin{aligned}&\mathcal{H} \\ &= \sup_{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}) \in R^4_+} \Bigg\{\frac{P\alpha_1\beta\mathcal{I}}{1+\varphi\mathcal{I}} \\ &-\frac{1}{2}\left(\mu - \frac{\vartheta}{2}(\varrho_1^2 \vee \varrho_2^2 \vee \varrho_3^2 \vee \varrho_4^2)\right)(\mathcal{S}^{\nu+1} + \mathcal{E}^{\nu+1} \\ &+ \mathcal{I}^{\nu+1} + \mathcal{R}^{\nu+1}) + \Theta + 3\mu + \eta + \varepsilon + \delta \\ &+ \frac{\varrho_1^2 + \varrho_2^2 + \varrho_4^2}{2}\Bigg\} \\ &< \infty. \end{aligned} \quad 25$$

Case-2: Suppose that $(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}) \in \mathfrak{D}_2$, in view of Eq 18, the result is

$$\begin{aligned}\mathfrak{L}V \le &-PM + \frac{P\alpha_1\beta\mathcal{S}\mathcal{I}}{(1+\varphi\mathcal{I})} + \frac{P\alpha_3(1-\phi)\gamma}{(1+b\mathcal{I})} \\ &-\frac{1}{2}\left[\mu - \frac{\nu}{2}(\varrho_1^2 \vee \varrho_2^2 \vee \varrho_3^2 \vee \varrho_4^2)\right] \\ &\times (\mathcal{S}^{\nu+1} + \mathcal{E}^{\nu+1} + \mathcal{I}^{\nu+1} + \mathcal{R}^{\nu+1}) \\ &+ \Theta + \delta + \eta + \varepsilon + 3\mu \\ &+ \frac{\varrho_1^2 + \varrho_2^2 + \varrho_4^2}{2} \\ \le &-PM + \frac{P\alpha_1\beta\varepsilon_2}{\varphi} + \frac{P\alpha_3(1-\phi)\gamma}{b} + G \\ \le &-P\left(M - \frac{\alpha_1\beta\varepsilon_2}{\varphi} + \frac{\alpha_3(1-\phi)\gamma}{b}\right) + G \le -1. \end{aligned} \quad 26$$

Case-3: Suppose that $(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}) \in \mathfrak{D}_3$, by Eq 18, getting

$$\begin{aligned}&\mathfrak{L}V \le -2\left(\frac{P\alpha_1\beta\mu\mathcal{S}\mathcal{I}}{\varphi\mathcal{E}}\right)^{\frac{1}{2}} + \frac{(1-\vartheta)\beta\mathcal{S}\mathcal{I}}{1+\varphi\mathcal{I}} + \frac{\beta\mathcal{I}}{\mathcal{E}(1+\varphi\mathcal{I})} + \Theta + \\ &3\mu + \eta + \varepsilon + \delta + \frac{\varrho_1^2+\varrho_2^2+\varrho_4^2}{2} \\ &\le -2\left(\frac{P\alpha_1\beta\mu\varepsilon_1\varepsilon_2}{\varphi\varepsilon_2}\right)^{\frac{1}{2}} + \frac{(1-\vartheta)\beta\varepsilon_1\varepsilon_2}{1+\varphi\mathcal{I}} + \frac{\beta\varepsilon_1}{\varepsilon_2(1+\varphi\mathcal{I})} + \\ &\Theta + 3\mu + \eta + \varepsilon + \delta + \frac{\varrho_1^2+\varrho_2^2+\varrho_4^2}{2} \\ &\le -2\left(\frac{P\alpha_1\beta\mu\varepsilon_1}{\varphi}\right)^{\frac{1}{2}} + T \le -1. \end{aligned} \quad 27$$

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Case-4: Suppose that $(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})\in\mathfrak{D}_4$, by Eq.19, getting

$$\mathfrak{L}V_{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})} \le -\frac{(1-\phi)\gamma}{\mathcal{R}(1+b\mathcal{I})}+\frac{P\alpha_1\beta\mathcal{I}}{\mathcal{R}(1+\varphi\mathcal{I})}+\frac{\eta}{\mathcal{S}}+3\mu+\delta+\Theta+B+\varepsilon+\beta+\frac{\varrho_1^2+\varrho_2^2+\varrho_4^2}{2} \quad -\frac{1}{2}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]\times(\mathcal{S}^{\nu+1}+\mathcal{E}^{\nu+1}+\mathcal{I}^{\nu+1}+\mathcal{R}^{\nu+1})$$
$$\le -\frac{(1-\phi)\gamma}{\mathcal{R}b}+\frac{P\alpha_1\beta\mathcal{I}}{\mathcal{R}\varphi}+F$$
$$\le -\frac{(1-\phi)\gamma}{\varepsilon_2 b}+\frac{P\alpha_1\beta\varepsilon_2}{\varepsilon_2\varphi}+F$$
$$\le -\frac{(1-\phi)\gamma}{\varepsilon_2 b}+\frac{P\alpha_1\beta}{\varphi}+F\,. \qquad 28$$

Case-5: Suppose that $(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})\in\mathfrak{D}_5$, by Eq 20, getting

$$\mathfrak{L}V_{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})} \le -\frac{1}{4}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]\mathcal{S}^{\nu+1}-\frac{1}{4}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]\mathcal{S}^{\nu+1}-\frac{1}{2}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big](\mathcal{E}^{\nu+1}+\mathcal{I}^{\nu+1}+\mathcal{R}^{\nu+1})+\frac{P\alpha_1\beta\varepsilon_1}{\varphi}+3\mu+\delta+\Theta+B+\eta+\varepsilon+\beta+\frac{\varrho_1^2+\varrho_2^2+\varrho_4^2}{2}$$
$$\le -\frac{1}{4}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]\mathcal{S}^{\nu+1}+J$$
$$\le -\frac{1}{4}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]\frac{1}{\varepsilon_1^{\nu+1}}+J\le -1.$$
29

Case-6: Suppose that $(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})\in\mathfrak{D}_6$, by Eq.21, getting

$$\mathfrak{L}V_{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})} \le -\frac{1}{4}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]\mathcal{I}^{\nu+1}-\frac{1}{4}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]\mathcal{I}^{\nu+1}-\frac{1}{2}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big](\mathcal{S}^{\nu+1}+\mathcal{E}^{\nu+1}+\mathcal{R}^{\nu+1})+\frac{P\alpha_1\beta\mathcal{I}}{1+\varphi}+\frac{\beta}{\varphi}+3\mu+\delta+\Theta+B+\eta+\varepsilon+\frac{\varrho_1^2+\varrho_2^2+\varrho_4^2}{2}$$
$$\le -\frac{1}{4}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]\mathcal{I}^{\nu+1}+W$$
$$\le -\frac{1}{4}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]\frac{1}{\varepsilon_1^{\nu+1}}+W$$
$$\le -1\,. \qquad 30$$

Case-7: Suppose that $(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})\in\mathfrak{D}_7$, by Eq.22, the result is

$$\mathfrak{L}V_{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})} \le -\frac{1}{4}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]\mathcal{E}^{\nu+1}-\frac{1}{4}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]\mathcal{E}^{\nu+1}-\frac{1}{2}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big](\mathcal{S}^{\nu+1}+\mathcal{I}^{\nu+1}+\mathcal{R}^{\nu+1})+\frac{P\alpha_1\beta\mathcal{I}}{(1+\varphi)\mathcal{E}}+3\mu+\delta+\Theta+B+\eta+\varepsilon+\beta+\frac{\varrho_1^2+\varrho_2^2+\varrho_4^2}{2}$$
$$\le -\frac{1}{4}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]\mathcal{E}^{\nu+1}+X$$
$$\le -\frac{1}{4}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]\frac{1}{\varepsilon_2^{\nu+1}}+X$$
$$\le -1\,. \qquad 31$$

Case-8: Suppose that $(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})\in\mathfrak{D}_8$, by Eq 23, the result is

$$\mathfrak{L}V_{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})} \le -\frac{1}{4}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]\mathcal{R}^{\nu+1}-\frac{1}{4}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]\mathcal{R}^{\nu+1}-\frac{1}{2}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big](\mathcal{S}^{\nu+1}+\mathcal{E}^{\nu+1}+\mathcal{I}^{\nu+1})+\frac{P\alpha_1\beta\varepsilon_1}{\varphi}+3\mu+\delta+\Theta+B+\eta+\varepsilon+\beta+\frac{\varrho_1^2+\varrho_2^2+\varrho_4^2}{2}$$
$$\le -\frac{1}{4}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]R^{\nu+1}+Y$$
$$\le -\frac{1}{4}\Big[\mu-\frac{\nu}{2}(\varrho_1^2\vee\varrho_2^2\vee\varrho_3^2\vee\varrho_4^2)\Big]\frac{1}{\varepsilon_2^{\nu+1}}+Y\le -1\,.$$
32

Thus, from the above eight cases, our conclusion is as follows:

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$\mathfrak{L}V_{(\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R})} \leq -1 \quad forall \quad (\mathcal{S},\mathcal{E},\mathcal{I},\mathcal{R}) \in \mathbb{R}_+^4$,
where ε is sufficiently small. In this case, assumption 1(ii) is satisfied.
Then, according to Lemma 1 $(\mathcal{H}_2)$, model Eq 4, has a unique stationary distribution and has an ergodic property. The proof is completed.

Remark 3:
If $\mathcal{R}_0^{\mathcal{S}} = \frac{\Theta\beta\vartheta\epsilon}{\left(\mu+\frac{\varrho_1^2}{2}\right)\left(\epsilon+\mu+\frac{\varrho_2^2}{2}\right)\left((1-\phi)\gamma+\mu+\delta+\frac{\varrho_3^2}{2}\right)} > 1$, then the system Eq.4, has a unique ergodic stationary distribution $\Gamma(.)$, according to Theorem 3. If the intensity of white noises is excluded from the analysis, the expression of $\mathcal{R}_0^{\mathcal{S}}$ corresponds to the threshold $\mathcal{R}_0$ of the deterministic system Eq.3. In particular, when $\varrho_i = 0, i = 1,2,3,4$. It shows that our results generalize those of the deterministic system.

Extinction of the disease infection
In this section, a different perspective on the spreading of disease can be gained by considering the extinction of infection. In a biological sense, the extinction of diseases indicates that they will eventually disappear. The stochastic model Eq 4, establishes the required conditions for disease extinction.

Lemma 2: Let $\big(\mathcal{S}(t),\mathcal{E}(t),\mathcal{I}(t),\mathcal{R}(t)\big)$ be the solution of system Eq.3, with any initial value $\big(\mathcal{S}(0),\mathcal{E}(0),\mathcal{I}(0),\mathcal{R}(0)\big) \in \mathbb{R}_+^4$. Then
$\lim_{t\to\infty} \frac{\mathcal{S}(t)}{t} = 0, \quad \lim_{t\to\infty}\left(\frac{\mathcal{E}(t),\mathcal{I}(t),\mathcal{R}(t)}{t}\right) = 0$ almost surely, 33
furthermore, if $\mu > \frac{\varrho_1^2 \vee \varrho_2^2 \vee \varrho_3^2 \vee \varrho_4^2}{2}$, then
$\lim_{t\to\infty} \int_0^t \frac{\mathcal{S}(s)d\mathfrak{B}_1(s)}{t} = 0, \quad \lim_{t\to\infty} \int_0^t \frac{\mathcal{E}(s)d\mathfrak{B}_2(s)}{t} = 0,$
$\lim_{t\to\infty} \int_0^t \frac{\mathcal{I}(s)d\mathfrak{B}_3(s)}{t} = 0, \lim_{t\to\infty} \int_0^t \frac{\mathcal{R}(s)d\mathfrak{B}_4(s)}{t} = 0,$
almost surely. 34
Since the proof of Lemma 2 is identical to that of Lemma 2.1 and Lemma 2.2 of Zhao and Jiang [36], it is therefore omitted.
Let us define a parameter
$$\widetilde{\mathcal{R}_0^{\mathcal{S}}} = \frac{\beta\vartheta(\varepsilon+\mu)}{(\varepsilon+\mu)^2\left((1-\phi)\gamma+\delta+\mu+\frac{\varrho_3^2}{2}\right)\wedge\left(\frac{\gamma^2\varrho_2^2}{2}\right)}.$$

Theorem 4: If $\widetilde{\mathcal{R}_0^{\mathcal{S}}} < 1$, then the disease will eventually be eradicated from the system Eq.4 and also satisfies the following condition

$$\lim_{t\to\infty} < \mathcal{S}(t) \geq \frac{\Theta}{\mu},$$
$$\lim_{t\to\infty} < \mathcal{E}(t) > = 0,$$
$$\lim_{t\to\infty} < \mathcal{R}(t) > = 0{,}35$$
and
$$lim\ sup_{t\to\infty} \frac{1}{t}\ln\left[\frac{(1-\phi)\gamma\mathcal{I}}{1+b\mathcal{I}} + (\mu+\varepsilon+\delta)\mathcal{R}(t)\right] \leq \vartheta\beta - \frac{1}{2(\varepsilon+\mu)^2}\left[(\varepsilon+\mu)^2\left((1-\phi)\gamma+\delta+\mu+\frac{\varrho_3^2}{2}\right)\wedge\left(\frac{\gamma^2\varrho_2^2}{2}\right)\right] < 0 \quad almost\ surely. \quad 36$$
Proof, The sum of the four variables in model Eq.4, yields
$$d(\mathcal{S}+\mathcal{E}+\mathcal{I}+\mathcal{R}) = [\mu - \vartheta\beta(\mathcal{S}(t)+\mathcal{E}(t)+\mathcal{I}(t)+\mathcal{R}(t))]dt + \varrho_{1\mathcal{S}}d\mathfrak{B}_1(t) + \varrho_2\mathcal{E}d\mathfrak{B}_2(t) + \varrho_{3\mathcal{I}}d\mathfrak{B}_3(t) + \varrho_{4\mathcal{R}}d\mathfrak{B}_4(t). \quad 37$$
This results in
$$\frac{\mathcal{S}(t)-\mathcal{S}(0)}{t} + \frac{\mathcal{E}(t)-\mathcal{E}(0)}{t} + \frac{\mathcal{I}(t)-\mathcal{I}(0)}{t} + \frac{\mathcal{R}(t)-\mathcal{R}(0)}{t} = \mathcal{A} - \mu < \mathcal{S}(t) > \mu < \mathcal{E}(t) > \mu < \mathcal{I}(t) > \mu < \mathcal{R}(t) > + \varrho_1\int_0^t \frac{\mathcal{S}(s)d\mathfrak{B}_1(s)}{t} + \varrho_2\int_0^t \frac{\mathcal{E}(s)d\mathfrak{B}_2(s)}{t} + \varrho_3\int_0^t \frac{\mathcal{I}(s)d\mathfrak{B}_3(s)}{t} + \varrho_4\int_0^t \frac{\mathcal{R}(s)d\mathfrak{B}_4(s)}{t}. \quad 38$$
let us take, $\varphi = \frac{(1-\phi)\gamma\mathcal{I}(t)}{1+b\mathcal{I}} + (\mu+\varepsilon+\delta)\mathcal{E}(t)$.
Our results from applying Ito's formula are
$$d\ln\varphi(t) = \left\{\frac{\beta\vartheta\mathcal{S}\mathcal{I} - \mu(\varepsilon+\mu)\big((1-\phi)\gamma+\mu+\delta\big)\mathcal{I}(t)}{\big((1-\vartheta)+\varepsilon\big)\mathcal{E}(t)+(\varepsilon+\delta+\mu)\mathcal{I}(t)} - \frac{\varepsilon^2\varrho_2^2\mathcal{E}^2(t)\Big((1-\phi)\gamma+(\varepsilon+\mu)^2\varrho_3^2I^2(t)\Big)}{2\big[\big((1-\vartheta)+\varepsilon\big)\mathcal{E}(t)+(\varepsilon+\mu+\delta)\mathcal{I}(t)\big]}\right\} + \frac{(1-\vartheta)\varepsilon\varrho_{2\mathcal{E}}(t)}{\big((1-\vartheta)+\varepsilon\big)\mathcal{E}(t)+(\varepsilon+\mu+\delta)\mathcal{I}(t)}d\mathfrak{B}_2(t) + \frac{(1-\phi)\gamma\varepsilon\varrho_{3\mathcal{I}}(t)}{\big((1-\vartheta)+\varepsilon\big)\mathcal{E}(t)+(\varepsilon+\mu+\delta)\mathcal{I}(t)}d\mathfrak{B}_3(t)$$
$$\leq \vartheta\beta - \frac{1}{2(\varepsilon+\mu)^2}\left[(\varepsilon+\mu)^2\left((1-\phi)\gamma+\mu+\delta+\frac{\varrho_3^2}{2}\right)\wedge\frac{\gamma^2\varrho_2^2}{2}\right] + \frac{(1-\vartheta)\varepsilon\varrho_{2\mathcal{E}}(t)}{\big((1-\vartheta)+\varepsilon\big)\mathcal{E}(t)+(\varepsilon+\mu+\delta)\mathcal{I}(t)}d\mathfrak{B}_2(t) + \frac{(1-\phi)\gamma\varepsilon\varrho_{3\mathcal{I}}(t)}{\big((1-\vartheta)+\varepsilon\big)\mathcal{E}(t)+(\varepsilon+\mu+\delta)\mathcal{I}(t)}d\mathfrak{B}_3(t). \quad 39$$
Integrating both sides of Eq 36 from 0 to t and together with Eq 34 and Eq 35, in order to obtain

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$$lim\ sup_{t\to\infty}\langle \mathcal{S}(t)+\mathcal{E}(t)+\mathcal{I}(t)+\mathcal{R}(t)\rangle = 1 \quad almost\ surely. \qquad 40$$

Integrating the both sides of Eq 39 from 0 tot, together with Eq 40, and noting that $\widetilde{R_0^{\mathcal{S}}} < 1$, one can get that

$$lim\ sup_{t\to\infty}\frac{\ln\varphi(t)}{t} \le \vartheta\beta - \frac{1}{2(\varepsilon+\mu)^2}\left[(\varepsilon+\mu)^2\left((1-\phi)\gamma+\mu+\delta+\frac{\varrho_3^2}{2}\right)\wedge\frac{\gamma^2\varrho_2^2}{2}\right] \qquad 41$$

which implies that

$$\lim_{t\to\infty}\mathcal{E}(t)=0, \lim_{t\to\infty}\mathcal{I}(t)=0, \quad and \quad \lim_{t\to\infty}\mathcal{R}(t)=0. \qquad 42$$

The implications of Lemma 2, and Eq.41 are as follows:

$$\lim_{t\to\infty}\langle \mathcal{S}(t)\rangle = \frac{\Theta}{\mu} = 1 \quad almost\ surely.$$

The proof has been completed.

Remark 4:

According to Theorem 4, if $\widetilde{\mathcal{R}_0^{\mathcal{S}}} = \frac{\beta\vartheta(\varepsilon+\mu)}{(\varepsilon+\mu)^2\left((1-\phi)\gamma+\delta+\mu+\frac{\varrho_3^2}{2}\right)\wedge\left(\frac{\gamma^2\varrho_2^2}{2}\right)} < 1$, the disease will eventually disappear. Take note of the expression for $\widetilde{\mathcal{R}_0^{\mathcal{S}}}$. It seems that the higher the white noise intensity, the easier it is to eradicate the disease. Therefore, adjusting the intensity of environmental noises can reduce disease outbreaks.

Results and Discussion

Numerical Simulation

The extinction and persistence of diseases have been investigated to the best of our ability. Some numerical simulations will be performed using the Milstein scheme[36] To demonstrate the effectiveness of our simulation. The discretization equation for model Eq 4, is as follows:

$$\mathcal{S}_{\ell+1} = \mathcal{S}_\ell + \left[\Theta - \frac{\beta\mathcal{S}_\ell\mathcal{I}_\ell}{1+\varphi\mathcal{I}_\ell} - \mu\mathcal{S}_\ell + \eta\mathcal{R}_\ell\right]\Delta t + \varrho_1\mathcal{S}_\ell\sqrt{\Delta t\xi_{1,l}} + \frac{\varrho_1^2}{2}\mathcal{S}_\ell\left(\xi_{1,l}^2-1\right)\Delta t,$$

$$\mathcal{E}_{\ell+1} = \mathcal{E}_\ell + \left[\frac{(1-\vartheta)\beta\mathcal{S}_\ell\mathcal{I}_\ell}{1+\varphi\mathcal{I}_\ell} - (\varepsilon+\mu)\mathcal{E}_\ell\right]\Delta t + \varrho_2\mathcal{E}_\ell\sqrt{\Delta t\xi_{2,l}} + \frac{\varrho_2^2}{2}\mathcal{E}_\ell\left(\xi_{2,l}^2-1\right)\Delta t\,,$$

$$\mathcal{I}_{\ell+1} = \mathcal{I}_\ell + \left[\frac{\vartheta\beta\mathcal{S}_\ell\mathcal{I}_\ell}{1+\varphi\mathcal{I}_\ell} + \varepsilon\mathcal{E}_\ell - \frac{(1-\phi)\gamma\mathcal{I}_\ell}{1+b\mathcal{I}_\ell} - (\mu+\delta)\mathcal{I}_\ell\right]\Delta t + \varrho_3\mathcal{I}_\ell\sqrt{\Delta t\xi_{3,l}} + \frac{\varrho_3^2}{2}\mathcal{I}_\ell\left(\xi_{3,l}^2-1\right)\Delta t\,,$$

$$\mathcal{R}_{\ell+1} = \mathcal{R}_\ell + \left[\frac{(1-\phi)\gamma\mathcal{I}_\ell}{1+b\mathcal{I}_\ell} - (\mu+\eta)\mathcal{R}_\ell\right]\Delta t + \varrho_4\mathcal{R}_\ell\sqrt{\Delta t\xi_{4,l}} + \frac{\varrho_4^2}{2}\mathcal{R}_\ell\left(\xi_{4,l}^2-1\right)\Delta t\,,$$

where the time increment $\Delta t > 0$, and ξ_i^2 is the Gaussian random variable $(i = 0,1,2,\dots,n)$.

The parameter values are given in the following Table 3, which validates our theoretical finding by examples.

Table 3. The parameters used in the simulation of model Eq 4.

Parameters	$\mathbb{E}_1$	$\mathbb{E}_2$	Source
Θ	5.00	4.50	presumed
β	0.70	0.70	ref [37]
φ	0.50	0.45	presumed
ϑ	0.70	0.70	presumed
ϵ	0.50	0.55	presumed
η	0.50	0.50	ref [37]
γ	0.50	0.50	ref [38]
δ	0.70	0.50	presumed
ϕ	0.40	0.35	presumed
μ	0.30	0.30	ref [39]
b	0.75	0.50	presumed
$\mathcal{S}(0)\mathcal{E}(0)$	0.50	0.50	presumed
$\mathcal{I}(0)$	0.30	0.30	presumed
$\mathcal{R}(0)$	0.40	0.40	presumed
	0.60	0.60	presumed
Δt	0.01	0.01	ref [37]

Example 1: Assume that the environmental white noise parameters are $(\varrho_1,\varrho_2,\varrho_3,\varrho_4) = 0.2$. Furthermore, Table 3, $\mathbb{E}_1$ shows the parameter values in relation to the biological feasibility results. Then

$$\mathcal{R}_0^{\mathcal{S}} = \frac{\Theta\beta\varepsilon\vartheta}{\left(\mu+\frac{\varrho_1^2}{2}\right)\left(\mu+\varepsilon+\frac{\varrho_2^2}{2}\right)\left((1-\phi)\gamma+\mu+\delta+\frac{\varrho_3^2}{2}\right)} = 3.5367 > 1,$$

satisfies the parameter requirement in Theorem 3, it may be decided that the stochastic model Eq.4,

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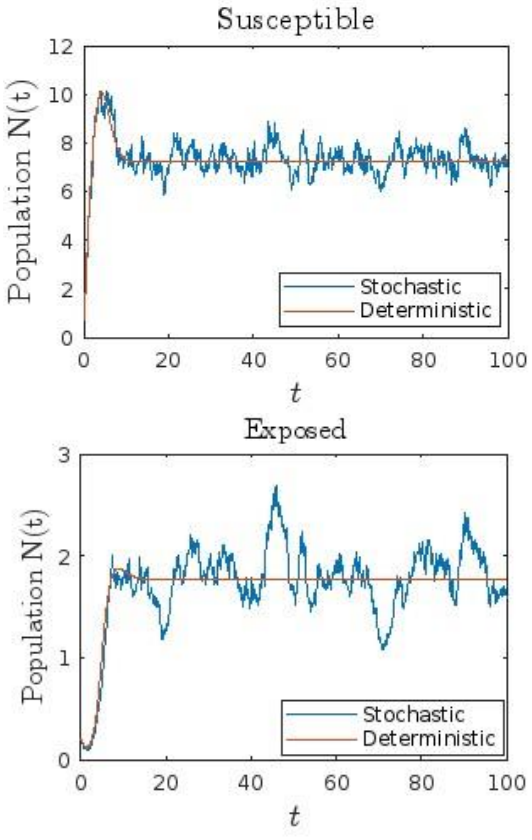
occupies ergodic attributes and a unique stationary distribution $\Pi(.)$. It is evident from Figs 1 and 3 that the solution of the model Eq 4 alternates between descending and ascending within a small area. It is evident from Fig 1, that there exists a stationary distribution from the density functions shown on the right-hand side.

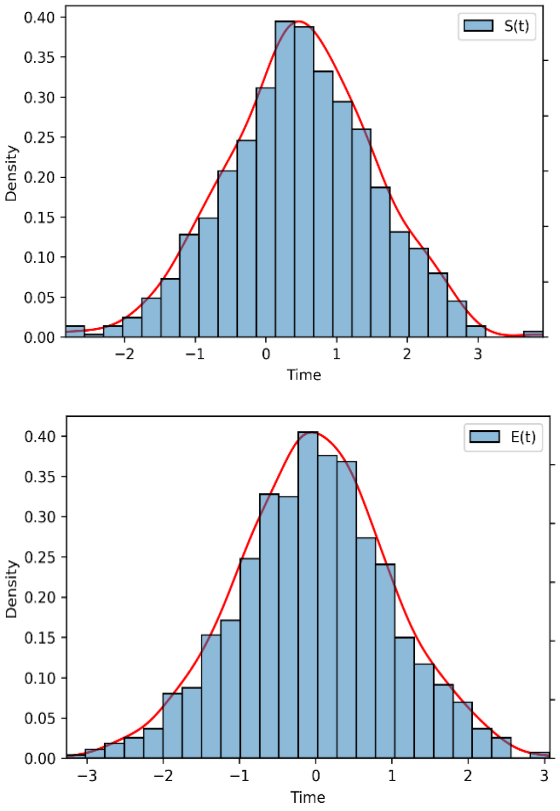
Example 2: Assume that the environmental white noise parameters are $(\varrho_1, \varrho_2, \varrho_3, \varrho_4) = 0.2$. Furthermore, Table 3, $(\mathbb{E}_2)$ shows the parameter values in relation to the biological feasibility results. Then

$$\widetilde{\mathcal{R}_0^{\delta}} = \frac{\beta\vartheta(\varepsilon+\mu)}{(\varepsilon+\mu)^2\left((1-\phi)\gamma+\mu+\delta+\frac{\varrho_3^2}{2}\right)\wedge\left(\frac{\gamma^2\varrho_2^2}{2}\right)} = 0.6556 < 1.$$

According to Theorem 4, exposed individuals and infected individuals will go extinct almost certainly if all parameter conditions are met. The conclusion of Theorem 4, is validated by Fig. 2. The numerical values of $\varrho_1 = \varrho_2 = \varrho_3 = \varrho_4 = 0.2$ shown in Figs 1 and 4 indicate the possibility of infective individuals going extinct under a set of large stochastic parameter values.

The below Figs 2 and 4 below demonstrate that when the white noise value is large, infectious diseases can go extinct. This implies that stochastic fluctuations can suppress disease outbreaks, but small values can lead to persistent infectious diseases. Further, Figs 1 and 3 illustrate that under appropriate conditions, the stochastic model Eq 4, has an ergodic stationary distribution. Consequently, there is full consistency between the theoretical outcomes of Theorems 3 and 4 and the numerical simulation examples.





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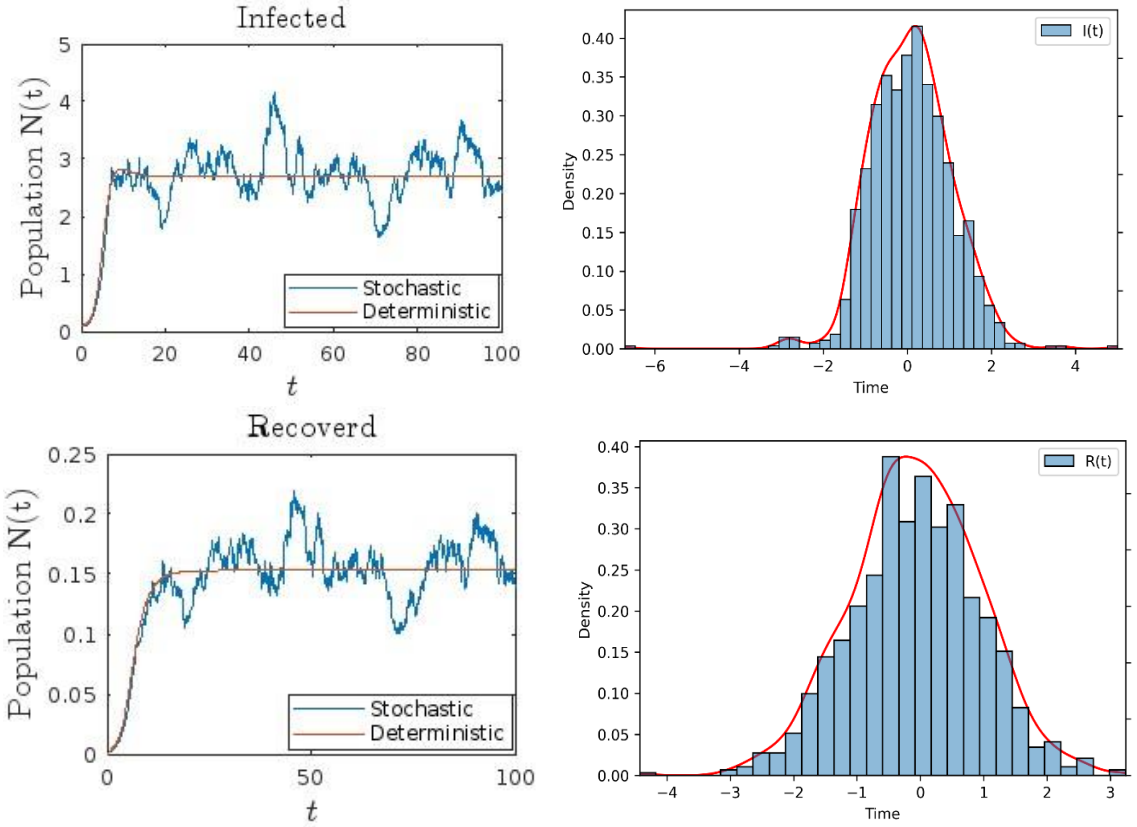
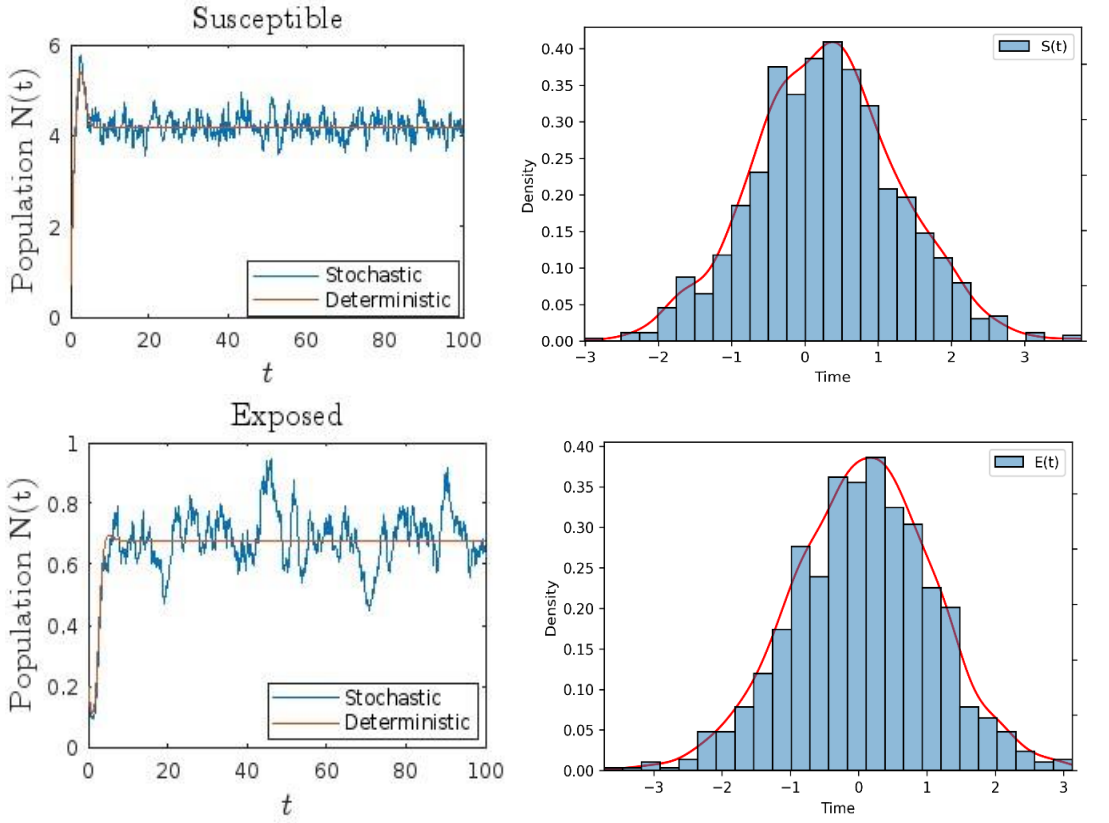


Figure 1. This diagram consists of a time sequence of stochastic persistence and stationary distribution of diseases based on the model Eq.4, for both R_0^S and $\widetilde{R}_0^S > 1$. On the right side of the column, the probability density function for $\mathcal{S}(t), \mathcal{E}(t), \mathcal{I}(t)$ and $\mathcal{R}(t)$ is shown in the form of a histogram.



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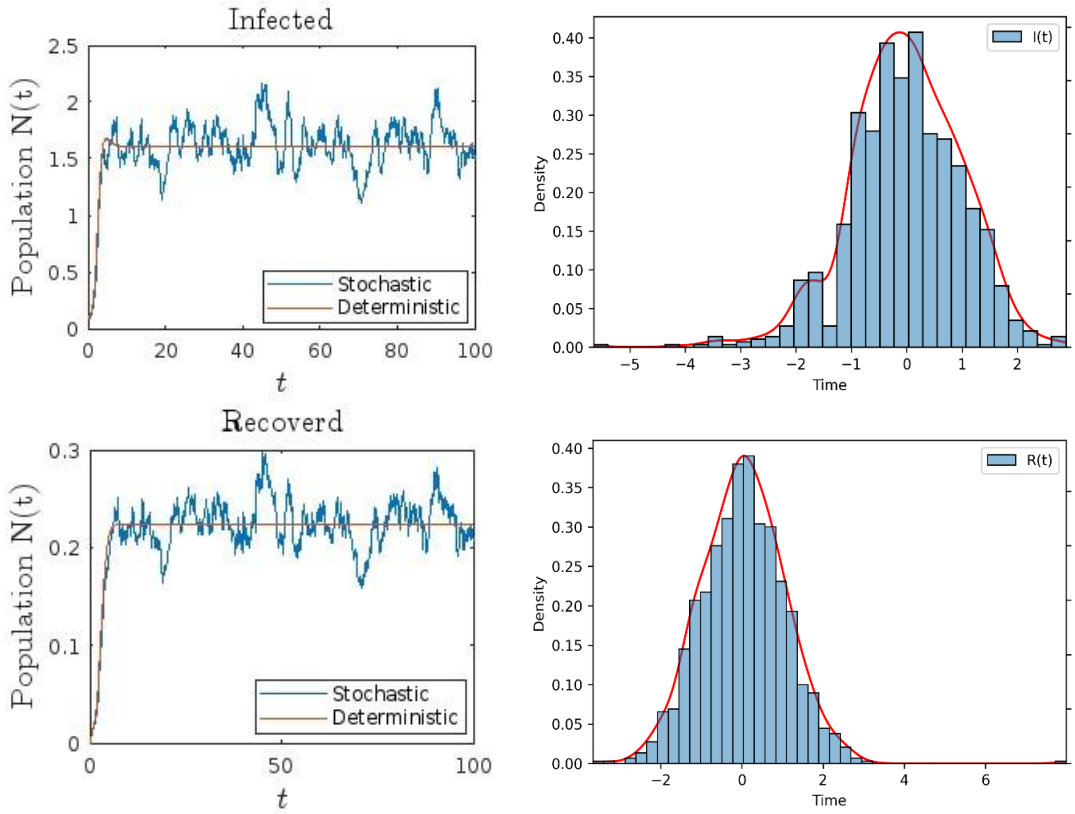

Figure 2. This time sequence diagram illustrates how disease extinction occurs in model Eq.4, for both R_0^S and $\widetilde{R}_0^S < 1$. On the right side of the column, the probability density function for $\mathcal{S}(t), \mathcal{E}(t), \mathcal{I}(t)$ and $\mathcal{R}(t)$ is shown in the form of a histogram.

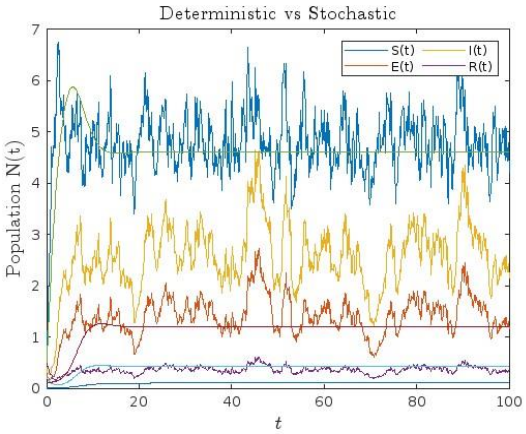

Figure 3. Comparison of solutions on $\mathcal{S}(t), \mathcal{E}(t), \mathcal{I}(t)$ and $\mathcal{R}(t)$ for each class of the Deterministic vs Stochastic system with R_0^S greater than 1.

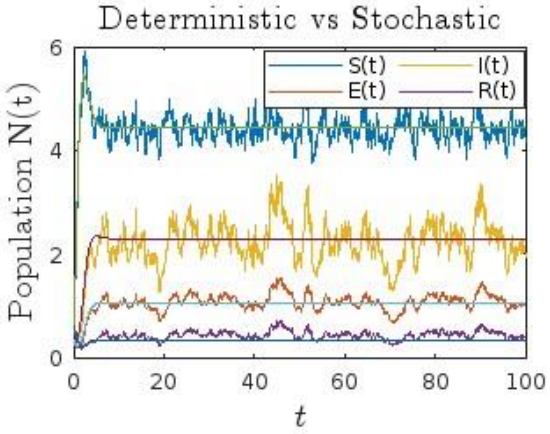

Figure 4. Comparison of solutions on $\mathcal{S}(t), \mathcal{E}(t), \mathcal{I}(t)$ and $\mathcal{R}(t)$ for each class of the Deterministic vs Stochastic system with $\widetilde{R}_0^S$ less than 1.

Conclusion

This paper explores the effect of treatment on disease transmission by considering the stochastic $\mathcal{SEIRS}$ epidemic model with saturated incidence and treatment function. In addition, there is a bi-linear incidence rate $h(\mathcal{I}) = \beta\mathcal{SI}$ as well as a saturation incidence rate $h(\mathcal{I}) = \frac{\beta\mathcal{SI}}{1+\varphi\mathcal{I}}$. An analysis of the global behavior of the model is presented in this paper, along with an estimation of the basic reproduction number $\mathcal{R}_0$. As a starting point, our initial findings

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indicated that $(\mathcal{S}(0), \mathcal{E}(0), \mathcal{I}(0), \mathcal{R}(0)) \in \mathbb{R}^4_+$ could be satisfied by a unique global positive solution. Furthermore, this research provides the necessary condition $\widetilde{\mathcal{R}_0^S} < 1$ for the disease to vanish. Our study also determines whether the stochastic Lyapunov function method is effective in determining whether $\mathcal{R}_0^S > 1$ exists for stationary distributions for positive solutions. Further, this study introduced white noise into our model Eq.4, to explore the dynamics of an autonomous stochastic epidemic disease mutation model. In the forthcoming research, our investigation will delve into Levy jumps and Markov Switching, as well as an epidemic model incorporating disease mutation. The importance of Theorem 4 becomes apparent in the presence of the condition $\widetilde{\mathcal{R}_0^S} < 1$, where the susceptible population predominates, resulting in the eradication of the infected recovering population. Through an analysis of the case $\widetilde{\mathcal{R}_0^S} > 1$ in Theorem 4, the objective of the study is to reveal the characteristics of stochastic systems described in Eq.4. Subsequent investigations will concentrate on these particular scenarios. Ultimately, our results were confirmed through numerical simulations, highlighting the beneficial effect of enhancing disease resistance in the realm of disease prevention and management.

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Authors' Declaration

- Conflicts of Interest: None.
- We hereby confirm that all the Figures and Tables in the manuscript are ours. Furthermore, any Figures and images, that are not ours, have been included with the necessary permission for re-publication, which is attached to the manuscript.
- No animal studies are present in the manuscript.
- No human studies are present in the manuscript.
- Ethical Clearance: The project was approved by the local ethical committee at Vellore Institute of Technology, Vellore, India.

Authors' Contribution Statement

S.S., conceptualization of the idea, development of the model framework, analysis, simulation, and writing. C.M., development of the model framework, supervision, validation, and submission process. All authors have read and agreed to the published version of the manuscript.

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التوزيع الثابت للنموذج الوبائي العشوائي لـ SEIRS مع معدل الإصابة المشبع ووظيفة العلاج المشبع

إس سارافانان، سي. مونيكا

قسم الرياضيات، كلية العلوم المتقدمة(SAS) ، معهد فيلور للتكنولوجيا، فيلور، الهند

الخلاصة

يهدف هذا البحث إلى تعزيز وتوسيع النموذج الرياضي الذي يحكم وباء SEIRS العشوائي الديناميكي (المعرض والمكشوف والمعدي والمعافى). يدمج هذا النموذج المعقد مكونات حاسمة، بما في ذلك معدل الإصابة المشبع و دالة العلاج المشبعة، والتي تعتبر أساسية في تشكيل ديناميكيات الوباء. الهدف هو استكشاف وجود و وحدانية الحل العالمي الإيجابي من خلال تطبيق وظيفة Lyapunov المصممة بدقة، مما يسهل إجراء تحليل أكثر عمقا لتعقيدات الأنظمة. يمكّننا هذا الإطار التحليلي من الكشف عن التفاعلات بين انتقال المرض وديناميكيات العلاج والتأثيرات العشوائية. ضمن هذا الإطار النظري، يفترض أن الاستجابة للعلاج تتناسب طرديا مع عدد الأفراد المصابين طالما بقيت حالات الإصابة داخل نظام الرعاية الصحية. يكمن أحد الجوانب الرئيسية لمساهمتنا في تحديد رقم التكاثر الأساسي العشوائي R_0 باعتباره عتبة حرجة تحدد مسار الوباء. في ظل الظروف التي تتميز بمستويات ضوضاء منخفضة و $R_0^S > 1$، فإنه يحدد المتطلبات الأساسية لظهور توزيع ثابت مريح، ويقدم نظرة ثاقبة للاتجاهات المحتملة طويلة المدى في انتشار المرض. وعلى العكس من ذلك، في السيناريوهات التي تتميز بكثافة الضوضاء العالية $\widetilde{\mathcal{R}_0^S} < 1$، يلقي تحليلنا الضوء على الاستئصال الحتمي للمرض. ولزيادة تعزيز الأسس النظرية، يدمج بحثنا عمليات محاكاة رقمية واسعة النطاق. لا تؤكد عمليات المحاكاة هذه صحة النتائج النظرية التي توصلنا إليها فحسب، بل توفر أيضًا تصورًا ديناميكيًا لآثار النموذج. تساهم هذه المنهجية المزدوجة، التي تجمع بين الفحص النظري الدقيق وعمليات المحاكاة العملية، في فهم دقيق لدور العشوائية في تشكيل ديناميكيات الوباء.

الكلمات المفتاحية: الانقراض، دالة ليابونوف، النمذجة الرياضية، التوزيع الثابت، نموذج الوباء العشوائي SEIRS..