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Stochastic analysis of an HIV model with various infection stages

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Abstract

In this study, we develop a stochastic model that captures the dynamics of HIV infection, encompassing susceptible individuals, asymptomatic HIV-positive individuals, and those exhibiting symptoms. Initially, we examine the existence and stability of both disease-free and endemic equilibria within the deterministic version of the model. Our analytical findings indicate that the basic reproduction number, $\mathcal{R}_0$, is a pivotal factor in determining the uniqueness and global stability of these equilibria. Furthermore, we explore the impact of environmental noise on the HIV disease model, identifying two critical thresholds, $\mathcal{R}_1^s$ and $\mathcal{R}_2^s$ (with $\mathcal{R}_2^s < \mathcal{R}_1^s$). If $\mathcal{R}_1^s$ is less than unity, the disease is likely to be eradicated; conversely, if $\mathcal{R}_2^s$ exceeds unity, the disease will persist, and a unique stationary distribution will emerge. Additionally, our numerical simulations reveal that when $\mathcal{R}_2^s < 1 < \mathcal{R}_1^s$, the disease may still face extinction. From an epidemiological viewpoint, our observations suggest that a decrease in environmental noise intensity results in a reduction of the oscillation amplitude in the disease dynamics. Conversely, an increase in noise intensity is associated with a lower mean of infectious individuals and a left-skewed distribution.

Keywords: Stochastic HIV disease model; Infection stages; Extinction; Stationary distribution; Probability density function

1 Introduction

Human Immunodeficiency Virus (HIV) causes Acquired Immunodeficiency Syndrome (AIDS) which is one of the infectious diseases that has been a threat to the public's health. Generally speaking, the disease is divided into four stages: acute infection stage (mainly characterized by symptoms of colds); asymptomatic infection period (without any symptoms, but with the virus continuing to multiply in the body); symptomatic infection period (symptoms related to AIDS begin to appear, showing persistent Lymphadenopathy, fever, weight loss, and so on); and a period typical of AIDS (the immune system is severely damaged, and various immune deficiencies, opportunistic infection, and malignant tumors occur). How it evolves, how long it takes, and the impact it has on the individual is determined by various factors such as physical quality, lifestyle, environment, and so on.

To better understand HIV/AIDS epidemiology, establishing mathematical models that conform to transmission characteristics based on its development stage and infection mode has been widely investigated. In [1], the authors divided the total clinical stages

into two phases, asymptomatic and symptomatic. Researchers [2] assessed the waiting time distributions for increasing phases of HIV/AIDS infection, and they studied the possibility of infection transmission between exposed and infected patients at various stages of illness. Naresh et al. [3] developed an HIV disease classification system with four subcategories—HIV-negative but susceptible, HIV-infected but unaware, HIV-positive, and HIV-infected but conscious—and they looked into the effect of sexual activity records on disease transmission. Huo et al. [4] established an HIV model that divided the population into five compartments: susceptible patients, HIV-positive individuals, those with full-blown AIDS but not receiving treatment, those being treated, and those who have changed their sexual habits to become immune to HIV infection in order to study the effect of treatment on the transmission dynamics of the HIV/AIDS epidemic model. For other HIV/AIDS models or another epidemic model, please see [5–10] and the references cited therein.

Nature is full of complexity and spontaneous processes, and the development of the population does not always obey purely deterministic rules. Population density never attains a fixed value over time but rather experiences oscillation around some average value [11]. Environmental noise is an inevitable factor that not only causes disturbance in the dynamics, but may also be beneficial by causing resonances and enhancing stability [12]. Noise can be classified into additive and multiplicative based on its source. Additive noise is not controlled by the system and can be directly introduced, while multiplicative noise is related to system parameters and variables. It should be noted that in systems with additive noise when the initial population density is very low, noise may result in negative solutions in stochastic systems, which is unrealistic. However, multiplicative noise ensures the nonnegativity of the solution. Moreover, the presence of multiplicative noise is characterized by the existence of an absorbing barrier at zero population density and the occurrence of anomalous fluctuations [13]. In biological systems, noise is often caused by environmental fluctuations and is typically considered multiplicative white noise. Continuous-time dynamics frequently focuses on white noise fluctuations and is modeled using a system of stochastic differential equations (SDEs). It has been theoretically and computationally documented [14–20] that environmental noise can impact the spread pattern of infection, and play an essential role in efforts to prevent or mitigate disease transmission. The stochasticity in the model is usually induced by assuming that the stochastic perturbations are directly proportional to each group in the population, which is a standard technique in stochastic population modeling (refer to [21–25] and the references therein). For example, in order to study a stochastic sex-structured HIV/AIDS epidemic model with effects of infective screening, Rathinasamy et al. [21] assumed that stochastic perturbations are of the white noise type and are directly proportional to susceptible males, susceptible females, infected males, infected females, and AIDS-class. Qi and Jiang [23] considered the impact of random environmental disturbance on the deterministic HIV/AIDS model with disease carrier screening and active seeking of treatment which divided the population into seven clusters including susceptible individuals, infectious and symptomatic primary HIV-infected individuals, asymptomatic and infectious disease carriers, and so on. By incorporating environmental noise into HIV/AIDS models, these authors captured the dynamic behavior of these systems, including the possible existence of a stationary distribution and the influence of white noise intensity on disease extinction and persistence. To summarize, introducing environmental noise in stochastic models is crucial for capturing

realistic system behavior, exploring emergent phenomena, quantifying uncertainty, and more.

However, stochastic HIV disease modeling with different infection stages has received little attention. Motivated by this, we mainly focus on how environmental fluctuations impact the dynamics of an HIV model with a nonmonotone incidence rate and obscure symptoms. This paper is organized as follows. Section 2 introduces some mathematical models used in this paper. Section 3 gives some preliminary results. Section 4 derives the stochastic extinction of the disease. An analysis of the stationary distribution is presented in Sect. 5. By solving the Fokker–Planck equation, the expression of the probability density function for the stationary distribution is obtained in Sect. 6. We display some numerical simulations in Sect. 7. In Sect. 8, we briefly conclude. Finally, the detailed proofs of our theoretical results are provided in the Appendix.

2 Model derivations

Following infection, HIV rapidly replicates in the body. Within a few days to weeks, some patients experience flu-like symptoms such as headaches, fever, sore throat, and a rash [26, 27]. However, at this stage, no special treatment is required and clinical symptoms will disappear on their own. Furthermore, considering that patients in a typical stage of AIDS are almost incapable of infecting others and therefore have no influence on the dynamics of the HIV infection, we only distinguish infected individuals into those who are going through the asymptomatic stage and those who are going through the symptomatic stage. Meanwhile, it is assumed that an infected individual in the symptomatic phase cannot spread the disease since he/she has been diagnosed and no longer has the ability to interact with other individuals in his/her everyday activities. The corresponding mathematical model is as follows:

$$\begin{cases} \dfrac{\mathrm{d}S(t)}{\mathrm{d}t} = \Lambda - \mu S(t) - \beta S(t) g(I_a(t)), \\ \dfrac{\mathrm{d}I_a(t)}{\mathrm{d}t} = \beta S(t) g(I_a(t)) - (\alpha + \mu) I_a(t), \\ \dfrac{\mathrm{d}I_s(t)}{\mathrm{d}t} = \alpha I_a(t) - (\mu + \gamma) I_s(t), \end{cases} \tag{2.1}$$

with

$$\mathbb{X} := \mathbb{R}^3_+ = \{(S(t), I_a(t), I_s(t)) : S(t) > 0,\ I_a(t) > 0,\ I_s(t) > 0\}, \tag{2.2}$$

where S is the susceptible group which could be infected with HIV, I_a is the infected population without obvious clinical symptoms, and I_s is the infected population with obvious clinical symptoms which is tested for HIV and diagnosed. All of the parameters $\Lambda, \mu, \beta, \alpha$, and γ are positive constants: Λ denotes the constant birth rate, μ is the natural mortality in the absence of HIV infection, β the power of infection that causes a new infection when a susceptible and an infective in class I_a come into contact, α represents the diagnosis rate of infections, and γ the disease-related mortality rate.

We are aware that incidence rates are critical in describing the interaction between susceptible and infected individuals within HIV/AIDS infection models, and that nonlinear incidence rates are more generic and realistic [28–30]. In [31], the authors proposed an

interactive expression $g(I_a)S$, where g is a nonlinear bounded map with saturation, and it allows for the introduction of certain "psychological" effects: given a large number of infectives, the infection forces $g(I_a)$ may drop as I_a grows, since, in the presence of a large number of infectives, the population may tend to limit the number of interactions per unit time. This incidence rate $g(I_a)S$ takes into account the behavioral changes and crowding impact of infective individuals and avoids the contact rate from becoming unbounded by selecting appropriate settings. Similar to [32], we adopt

$$g(I_a) = \frac{I_a}{1 + \delta I_a^2},$$

where $\frac{g(I_a)}{I_a} = \frac{1}{1+\delta I_a^2}$ refers to the psychological or inhibiting influence of susceptible individuals' behavioral changes when the number of infected individuals is relatively big and $\delta > 0$. Then the model (2.1) can be rewritten as follows:

$$\begin{cases} \dfrac{\mathrm{d}S(t)}{\mathrm{d}t} = \Lambda - \mu S(t) - \dfrac{\beta S(t) I_a(t)}{1 + \delta I_a^2(t)}, \\ \dfrac{\mathrm{d}I_a(t)}{\mathrm{d}t} = \dfrac{\beta S(t) I_a(t)}{1 + \delta I_a^2(t)} - (\alpha + \mu) I_a(t), \\ \dfrac{\mathrm{d}I_s(t)}{\mathrm{d}t} = \alpha I_a(t) - (\mu + \gamma) I_s(t). \end{cases} \tag{2.3}$$

Notably, variable I_s in model (2.3) does not appear directly in the first two equations, implying that the individuals in compartment I_s do not spread the disease. The model (2.3) can be simplified as follows when the equation for I_s is not taken into account:

$$\begin{cases} \dfrac{\mathrm{d}S(t)}{\mathrm{d}t} = \Lambda - \mu S(t) - \dfrac{\beta S(t) I_a(t)}{1 + \delta I_a^2(t)}, \\ \dfrac{\mathrm{d}I_a(t)}{\mathrm{d}t} = \dfrac{\beta S(t) I_a(t)}{1 + \delta I_a^2(t)} - (\alpha + \mu) I_a(t). \end{cases} \tag{2.4}$$

Due to the significant effect of environmental noise on the dynamics of HIV infection, we assume that stochastic perturbations are of the white noise type and directly proportional to S and I_a. For Δt small, it is appropriate to consider $X = (S, I_a)$ as a Markov process with the following specifications:

$$\mathbf{E}[S(t + \Delta t) - S(t) | X = x] \approx \left[\Lambda - \mu S(t) - \frac{\beta S(t) I_a(t)}{1 + \delta I_a^2(t)} \right] \Delta t,$$

$$\mathbf{E}[I_a(t + \Delta t) - I_a(t) | X = x] \approx \left[\frac{\beta S(t) I_a(t)}{1 + \delta I_a^2(t)} - (\alpha + \mu) I_a(t) \right] \Delta t,$$

and

$$\mathrm{Var}[S(t + \Delta t) - S(t) | X = x] \approx \sigma_1^2 S^2(t) \Delta t,$$

$$\mathrm{Var}[I_a(t + \Delta t) - I_a(t) | X = x] \approx \sigma_2^2 I_a^2(t) \Delta t.$$

Then the deterministic model (2.4) can be described in the following form:

$$\begin{cases} \mathrm{d}S(t) = \left(\Lambda - \mu S(t) - \dfrac{\beta S(t) I_a(t)}{1 + \delta I_a^2(t)}\right)\mathrm{d}t + \sigma_1 S(t)\mathrm{d}B(t), \\ \mathrm{d}I_a(t) = \left(\dfrac{\beta S(t) I_a(t)}{1 + \delta I_a^2(t)} - (\alpha + \mu) I_a(t)\right)\mathrm{d}t + \sigma_2 I_a(t)\mathrm{d}B(t), \end{cases} \tag{2.5}$$

and the terms $\sigma_1 S(t)\mathrm{d}B(t)$ and $\sigma_2 I_a(t)\mathrm{d}B(t)$ biologically reflect the contacts of the environment and individuals involved in the process. The Brownian motion $B(t)$ is described across the whole probability space $(\mathbb{R}_+^2, \mathbf{B}(\mathbb{R}_+^2), \{\mathcal{F}_t\}_{t\geq 0}, \mathbf{P})$ with $B(0) = 0$, where $\mathbf{B}(\mathbb{R}_+^2)$ denotes the Borel σ-algebra on $\mathbb{R}_+^2$, and a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ satisfies the usual conditions (i.e., it is right continuous and increasing while $\mathcal{F}_0$ contains all $\mathbf{P}$-null sets); $\sigma_i^2 > 0$ $(i = 1, 2)$ express the intensities of the white noise $B(t)$. Other parameters are the same as in the ODE model (2.3) and the SDE model's (2.5) state space is $\mathbb{X}$ as well.

3 Preliminary results

The premise to ensure that our work can be carried out smoothly is to understand the dynamics of the deterministic mode (2.3).

Without hesitation, it is necessary for us to provide a theorem on the positivity and boundedness of the solution to model (2.3).

Theorem 3.1 *All solutions* $(S(t), I_a(t), I_s(t))$ *of the model* (2.3) *with any initial value* $(S(0), I_a(0), I_s(0)) \in \mathbb{X}$ *remain positive for all* $t \geq 0$, *and are defined in the positive bounded invariant set*

$$\Gamma = \left\{(S, I_a, I_s) \in \mathbb{X} : S \geq 0,\ I_a \geq 0,\ I_s \geq 0,\ 0 < S + I_a + I_s \leq \frac{\Lambda}{\mu}\right\} \subset \mathbb{X}. \tag{3.1}$$

From now on, we mainly focus on the existence and stability of equilibria in the model (2.3). Defining the basic reproduction number

$$\mathcal{R}_0 = \frac{\beta S_0}{\alpha + \mu} = \frac{\beta \Lambda}{\mu(\alpha + \mu)}, \tag{3.2}$$

we can obtain the following theorems.

Theorem 3.2 *For the model* (2.3),

(*i*) *there is always the disease-free equilibrium* $E_0 = (S_0, 0, 0)$, *where* $S_0 = \frac{\Lambda}{\mu}$;

(*ii*) *if* $\mathcal{R}_0 > 1$, *there exists a unique endemic equilibrium* $E^* = (S^*, I_a^*, I_s^*)$ *with*

$$S^* = \frac{(\mu + \alpha)(1 + \delta I_a^{*2})}{\beta}, \quad I_a^* = \frac{\sqrt{\beta^2 - 4\mu^2\delta(1 - \mathcal{R}_0)} - \beta}{2\mu\delta}, \quad I_s^* = \frac{\alpha}{\mu + \gamma} I_a^*, \tag{3.3}$$

where I_a^* *is the positive root of the following equation*:

$$H(I_a) := p_2 I_a^2 + p_1 I_a + p_0 = 0,$$

with

$$p_2 = (\alpha + \mu)\delta\mu, \quad p_1 = \beta(\alpha + \mu), \quad p_0 = -\Lambda\beta + \mu(\alpha + \mu).$$

Remark 3.3 A simple computation reveals that

$$\frac{\mathrm{d}I_a}{\mathrm{d}\delta} = -\frac{\frac{\mathrm{d}p_2}{\mathrm{d}\delta}I_a^2 + \frac{\mathrm{d}p_1}{\mathrm{d}\delta}I_a + \frac{\mathrm{d}p_0}{\mathrm{d}\delta}}{2p_2I_a + p_1} = -\frac{\mu(\alpha+\mu)I_a^2}{2\delta\mu(\alpha+\mu)I_a + \beta(\alpha+\mu)} < 0.$$

This indicates that the number of infective individuals decreases due to the psychological or inhibitory effect of susceptible individuals' behavioral change.

Theorem 3.4 *If* $\mathcal{R}_0 < 1$, *the disease-free equilibrium* $E_0 = (\frac{\Lambda}{\mu}, 0, 0)$ *of the model* (2.3) *is globally asymptotically stable in the domain* Γ, *while if* $\mathcal{R}_0 > 1$, E_0 *is unstable*.

Theorem 3.5 *If* $\mathcal{R}_0 > 1$, *the model* (2.3) *admits a unique endemic equilibrium* $E^* = (S^*, I_a^*, I_s^*)$ *which is globally asymptotically stable in* Γ.

Remark 3.6 Theorems 3.4 and 3.5 show that $\mathcal{R}_0$ is critical for determining whether or not an endemic equilibrium exists for the model (2.3).

In addition, prior to initiating comprehensive research on the influence of environmental noise on the HIV infection dynamics, it is imperative to first provide an explanation regarding the existence and boundedness of solutions for the stochastic model (2.5). Please refer to the following theorems for specific details.

Theorem 3.7 *For any* $t \geq 0$, *the model* (2.5) *has a unique positive solution* $(S(t), I_a(t))$ *which will stay in* $\mathbb{X}$ *with probability one for any given initial value* $(S(0), I_a(0)) \in \mathbb{X}$.

We skip the proof of Theorem 3.7 because it is standard and comparable to that of Lemma 2.4 in [33].

Theorem 3.8 *For any initial value* $X_0 = (S(0), I_a(0)) \in \mathbb{X}$, *the solutions of the model* (2.5) *are stochastically ultimately bounded and permanent*.

Here, the Markov process $X(t) = (S(t), I_a(t))$ of our stochastic model (2.5) is first shown to be geometrically ergodic, implying that a stationary distribution exists.

Theorem 3.9 *The Markov process* $X(t) = (S(t), I_a(t))$ *of the model* (2.5), *with an initial value* $X_0 = (S(0), I_a(0)) \in \mathbb{X}$, *exhibits V-geometric ergodicity*.

For convenience, the proofs of the above results will be presented in the Appendix.

4 Disease stochastic extinction

In the context of infection models, we consistently assess the long-term possibility of disease eradication. The reproduction number is widely recognized as one of the most crucial variables in epidemiology. This section provides an expression of the stochastic reproduction number

$$\mathcal{R}_1^s := \frac{\beta\Lambda}{\mu\left(\alpha+\mu+\frac{\sigma_2^2}{2}\right)}, \tag{4.1}$$

and we then obtain the following theorem about the extinction of the disease for the model (2.5).

Theorem 4.1 *Assume that* $\mathcal{R}_1^s < 1$, *then the solution* $(S(t), I_a(t))$ *of the model* (2.5) *has the following property*:

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t S(s)\mathrm{d}s \le \frac{\Lambda}{\mu} \quad a.s., \qquad \limsup_{t\to\infty} \frac{\log I_a(t)}{t} < 0 \quad a.s.$$

In other words, the disease dies out with probability one.

Proof By applying the comparison theorem for stochastic differential equations [34] and the law of large numbers [35], we analyze the first equation of the model (2.5). Considering the auxiliary equation below, which includes a stochastic perturbation

$$\mathrm{d}\mathcal{S}(t) = (\Lambda - \mu\mathcal{S}(t))\mathrm{d}t + \sigma_1\mathcal{S}(t)\mathrm{d}B(t), \tag{4.2}$$

with the initial value $\mathcal{S}(0) = S(0) > 0$, and setting

$$\boldsymbol{g}(\mathcal{S}(t)) = \Lambda - \mu\mathcal{S}(t), \quad \boldsymbol{\sigma}(\mathcal{S}(t)) = \sigma_1\mathcal{S}(t), \ \mathcal{S}(t) \in (0,\infty),$$

we calculate that

$$\int_{c_0}^{\mathcal{S}} \frac{\boldsymbol{g}(u)}{\boldsymbol{\sigma}^2(u)}\mathrm{d}u = \int_{c_0}^{\mathcal{S}} \frac{1}{\sigma_1^2u^2}(\Lambda - \mu u)\mathrm{d}u = \frac{1}{\sigma_1^2}\Big(-\frac{\Lambda}{\mathcal{S}} - \mu\ln\mathcal{S}\Big) + C_1,$$

where c_0 is an arbitrary fixed positive constant and C_1 is a constant determined by the foregoing formula. It is evident that

$$\begin{aligned}\int_0^\infty \frac{1}{\boldsymbol{\sigma}^2(\mathcal{S})}\mathrm{e}^{2\int_{c_0}^{\mathcal{S}}\frac{\boldsymbol{g}(u)}{\boldsymbol{\sigma}^2(u)}\mathrm{d}u}\mathrm{d}\mathcal{S} &= \int_0^\infty \frac{1}{\sigma_1^2\mathcal{S}^2}\mathrm{e}^{2\left(\frac{1}{\sigma_1^2}(-\frac{\Lambda}{\mathcal{S}}-\mu\ln\mathcal{S})+C_1\right)}\mathrm{d}\mathcal{S}\\ &= \frac{\mathrm{e}^{2C_1}}{\sigma_1^2}\int_0^\infty \mathcal{S}^{-\frac{2\mu}{\sigma_1^2}-2}\mathrm{e}^{-\frac{2\Lambda}{\sigma_1^2\mathcal{S}}}\mathrm{d}\mathcal{S} < \infty,\end{aligned}$$

thus, we obtain that the model (4.2) is ergodic with the ergodic distribution

$$\boldsymbol{k}(\mathcal{S}) = \frac{\mathrm{e}^{2C_1}}{\sigma_1^2}\mathcal{S}^{-\frac{2\mu}{\sigma_1^2}-2}\mathrm{e}^{-\frac{2\Lambda}{\sigma_1^2\mathcal{S}}}, \ \mathcal{S} \in (0,\infty),$$

where $C_2 \triangleq \frac{\mathrm{e}^{2C_1}}{\sigma_1^2}$ is a constant such that $\int_0^\infty \boldsymbol{k}(\mathcal{S})\mathrm{d}\mathcal{S} = 1$. It then follows that

$$\lim_{t\to\infty}\frac{1}{t}\int_0^t \mathcal{S}(s)\mathrm{d}s = \int_0^\infty \mathcal{S}\boldsymbol{k}(\mathcal{S})\mathrm{d}\mathcal{S} \ \text{ a.s.} \tag{4.3}$$

and, from Theorem 3.1 of [36], we have $\int_0^\infty \mathcal{S}\boldsymbol{k}(\mathcal{S})\mathrm{d}\mathcal{S} = \frac{\Lambda}{\mu}$.

Letting $\mathcal{S}(t)$ be the solution of (4.2) with the initial value $\mathcal{S}(0) = S(0) > 0$, by the comparison theorem for stochastic differential equations [34], we get

$$S(t) \le \mathcal{S}(t) \quad \text{for any } \ t \ge 0 \ \text{ a.s.} \tag{4.4}$$

Thus we have

$$\lim_{t\to\infty}\frac{1}{t}\int_0^t S(s)\mathrm{d}s \le \lim_{t\to\infty}\frac{1}{t}\int_0^t \mathcal{S}(s)\mathrm{d}s = \frac{\Lambda}{\mu}\ \text{a.s.} \tag{4.5}$$

Applying Itô's formula yields

$$\mathrm{d}\log I_a(t) = \left(\frac{\beta S(t)}{1+\delta I_a^2(t)} - (\alpha+\mu) - \frac{\sigma_2^2}{2}\right)\mathrm{d}t + \sigma_2 \mathrm{d}B(t). \tag{4.6}$$

Hence, from Eq. (4.6), we derive the following equation by integrating both sides from 0 to t:

$$\begin{aligned}\log I_a(t) &= \log I_a(0) + \int_0^t \left(\frac{\beta S(s)}{1+\delta I_a^2(s)} - (\alpha+\mu) - \frac{\sigma_2^2}{2}\right)\mathrm{d}s + \int_0^t \sigma_2 \mathrm{d}B(s)\\ &\le \log I_a(0) + \int_0^t \left(\beta S(s) - (\alpha+\mu) - \frac{\sigma_2^2}{2}\right)\mathrm{d}s + \int_0^t \sigma_2 \mathrm{d}B(s).\end{aligned} \tag{4.7}$$

Setting $\phi(t) := \int_0^t \sigma_2 \mathrm{d}B(s)$, we have

$$\frac{\langle \phi(t), \phi(t)\rangle}{t} = \frac{1}{t}\int_0^t \sigma_2^2 \mathrm{d}s = \sigma_2^2 < +\infty.$$

Hence, using the law of large numbers for martingales [11], we obtain $\limsup_{t\to\infty} \frac{\phi(t)}{t} = 0$ a.s.

It follows from (4.5) that dividing by t on both sides of (4.7) and letting $t \to \infty$, we derive

$$\begin{aligned}\limsup_{t\to\infty}\frac{\log I_a(t)}{t} &\le \frac{\beta\Lambda}{\mu} - \left(\alpha+\mu+\frac{\sigma_2^2}{2}\right)\\ &= \left(\alpha+\mu+\frac{\sigma_2^2}{2}\right)(\mathcal{R}_1^s - 1).\end{aligned} \tag{4.8}$$

Therefore, under the condition $\mathcal{R}_1^s < 1$, one may claim that

$$\limsup_{t\to\infty}\frac{\log I_a(t)}{t} < 0 \quad \text{a.s.}$$ □

Remark 4.2 Theorem 4.1 provides sufficient conditions for the disease to be eradicated in the long run. Due to the fact that $\mathcal{R}_1^s < \mathcal{R}_0$, we are pleasantly surprised to discover a scenario where $\mathcal{R}_0 > 1$ but $\mathcal{R}_1^s < 1$ such that the asymptomatic infected population $I_a(t)$ of the SDE model (2.5) becomes extinct exponentially, which is quite different from the result of Theorem 3.5 for the deterministic model (2.3). This tells us that the impact of environmental noises on disease dynamics must not be ignored.

5 Existence of ergodic stationary distribution

The other major concern in epidemiology is the long-term persistence of the disease. In Theorem 3.9, we conclude that the Markov process of the SDE model (2.5), denoted as $X(t)$, possesses a stationary distribution for the initial value $X_0 = (S(0), I_a(0)) \in \mathbb{X}$. We will analyze the endemic stationary distribution of $X(t)$ in the model (2.5).

Khasminskii [37] presented the following famous conclusion about the existence and uniqueness of the stationary distribution of the stochastic process $X(t)$.

Lemma 5.1 ([37]) *Suppose there exists a smooth boundary* $\partial\Pi$ *that encloses a bounded open domain* $\Pi \subset \mathbb{R}^2_+$, *which exhibits the following properties*:

(i) *In the domain* Π *and some neighborhood, the minimum eigenvalue of the diffusion matrix* $J(X)$ *is distant from zero*;

(ii) *If* $X \in \mathbb{R}^2_+ \setminus \Pi$, *the mean time* τ *for a path originating from* X *to reach the set* Π *is finite, and* $\sup_{X\in\mathbb{V}} \mathbf{E}\tau < \infty$ *holds for every compact subset* $\mathbb{V} \subset \mathbb{R}^2_+$.

Then the Markov process $X(t)$ *starting from* $X_0 \in \mathbb{R}^2_+$ *possesses a unique stationary distribution* $\boldsymbol{\pi}(\cdot)$. *Furthermore, if* $f(\cdot)$ *is a function that can be integrated with respect to the measure* $\boldsymbol{\pi}$, *then*

$$\mathbf{P}\left\{\lim_{t\to\infty}\frac{1}{T}\int_0^T f(X(t))\mathrm{d}t = \int_{\mathbb{R}^2_+} f(Y)\boldsymbol{\pi}(\mathrm{d}Y)\right\} = 1.$$

Denote

$$\mathcal{R}_2^s = \frac{\beta\Lambda}{\left(\mu+\frac{\sigma_1^2}{2}\right)\left(\alpha+\mu+\frac{\sigma_2^2}{2}\right)},$$

then we obtain the following result.

Theorem 5.2 *If* $\mathcal{R}_2^s > 1$, *then for the model* (2.5) *with an initial value* $X_0 \in \mathbb{X}$, *there is a unique stationary distribution with the ergodicity property*

$$\mathbf{P}\left\{\lim_{T\to\infty}\frac{1}{T}\int_0^T f(X_i(t))\mathrm{d}t = \int_\Omega Y_i\boldsymbol{\pi}(\mathrm{d}Y_1,\mathrm{d}Y_2)\right\} = 1.$$

Proof Since $\mathcal{L}$ is uniformly elliptic in $\mathbb{X}$, (i) of Lemma 5.1 holds. To verify (ii), it is sufficient to demonstrate that there are some neighborhood Π and a nonnegative C^2-function V such that $\mathcal{L}V$ is negative definite for $X \in \mathbb{X} \setminus \Pi$.

Define the function

$$V(S,I_a) = MV_1 + V_2 + V_3,$$

where

$$V_1 = -\frac{\beta\Lambda}{\left(\mu+\frac{\sigma_1^2}{2}\right)^2}\ln S - \ln I_a,\quad V_2 = \frac{1}{m+1}(S+I_a)^{m+1},\quad V_3 = -\ln S.$$

Here, $M > 0$ is a sufficiently large constant satisfying

$$-M\left(\alpha+\mu+\tfrac{\sigma_2^2}{2}\right)(\mathcal{R}_2^s-1) + \Upsilon_2 \le -2,$$

where

$$\Upsilon_2 = \sup_{(S,I_a)\in\mathbb{R}^2_+}\left\{\Lambda(S+I_a)^m - \frac{\mu}{2}S^{m+1} - \frac{\alpha+\mu}{2}I_a^{m+1} + \mu + \frac{\sigma_1^2}{2}\right\}.$$

It is easy to check that

$$\liminf_{n\to\infty,\,(S,I_a)\in\mathbb{R}^2_+\setminus U_n} V(S,I_a) = +\infty,$$

where $U_n = (\frac{1}{n}, n) \times (\frac{1}{n}, n)$. Furthermore, $V(S,I_a)$ is a continuous function. Hence, (S,I_a) must have a minimum point $(\tilde{S}_0, \tilde{I}_{a0})$ in the interior of $\mathbb{R}^2_+$. Then we define a nonnegative function $\widetilde{V} : \mathbb{R}^2_+ \to \mathbb{R}$ as follows:

$$\widetilde{V}(S,I_a) = V(S,I_a) - V(\tilde{S}_0, \tilde{I}_{a0}).$$

A simple calculation yields that

$$\mathcal{L}(-\ln S) = -\frac{\Lambda}{S} + \mu + \frac{\beta I_a}{1+\delta I_a^2} + \frac{\sigma_1^2}{2} \tag{5.1}$$

and

$$\mathcal{L}(-\ln I_a) = -\frac{\beta S}{1+\delta I_a^2} + \left(\alpha + \mu + \frac{\sigma_2^2}{2}\right). \tag{5.2}$$

Set $\kappa = \dfrac{\Lambda}{\mu + \frac{\sigma_1^2}{2}}$, then it follows from (5.2) that

$$\begin{aligned}
\mathcal{L}(-\ln I_a) &\le -\beta\kappa\left(\frac{S}{\kappa(1+\delta I_a^2)} - 1\right) - \beta\kappa + \left(\alpha + \mu + \frac{\sigma_2^2}{2}\right) \\
&\le -\beta\kappa + \beta\kappa\left(1 - \frac{S}{\kappa(1+\delta I_a^2)}\right) + \left(\alpha + \mu + \frac{\sigma_2^2}{2}\right) \\
&\le -\beta\kappa + \beta\kappa\ln(1+\delta I_a^2) + \beta\kappa\ln\frac{\kappa}{S} + \left(\alpha + \mu + \frac{\sigma_2^2}{2}\right) \\
&\le -2\beta\kappa + \beta\kappa\delta I_a^2 + \frac{\beta\kappa^2}{S} + \left(\alpha + \mu + \frac{\sigma_2^2}{2}\right).
\end{aligned}$$

Hence, we have

$$\begin{aligned}
\mathcal{L}V_1 &\le \frac{\beta\Lambda}{\left(\mu + \frac{\sigma_1^2}{2}\right)^2}\left(-\frac{\Lambda}{S} + \mu + \frac{\beta I_a}{1+\delta I_a^2} + \frac{\sigma_1^2}{2}\right) - \frac{2\beta\Lambda}{\mu + \frac{\sigma_1^2}{2}} + \frac{\beta\delta\Lambda I_a^2}{\mu + \frac{\sigma_1^2}{2}} \\
&\quad + \frac{\beta\Lambda^2}{\left(\mu + \frac{\sigma_1^2}{2}\right)^2 S} + \left(\alpha + \mu + \frac{\sigma_2^2}{2}\right) \\
&= -\left(\alpha + \mu + \frac{\sigma_2^2}{2}\right)(\mathcal{R}_2^s - 1) + \frac{\beta^2\Lambda I_a}{\left(\mu + \frac{\sigma_1^2}{2}\right)^2(1+\delta I_a)^2} + \frac{\beta\delta\Lambda I_a^2}{\mu + \frac{\sigma_1^2}{2}}.
\end{aligned}$$

By the Itô's formula, we get

$$\begin{aligned}
\mathcal{L}V_2 &= (S+I_a)^m(\Lambda - \mu S - (\alpha+\mu)I_a) + \frac{m(S+I_a)^{m-1}}{2}\left(\sigma_1^2 S^2 + \sigma_2^2 I_a^2\right) \\
&\le \Lambda(S+I_a)^m - \mu S^{m+1} - (\alpha+\mu)I_a^{m+1} + \frac{m\sigma_1^2}{2}S^{m+1} + \frac{m\sigma_2^2}{2}I_a^{m+1}.
\end{aligned}$$

Choosing $m = \min\left\{1, \frac{\mu}{\sigma_1^2} \wedge \frac{\alpha+\mu}{\sigma_2^2}\right\}$, we have

$$\mathcal{L}V_2 \leq \Lambda(S+I_a)^m - \frac{\mu}{2}S^{m+1} - \frac{\alpha+\mu}{2}I_a^{m+1}.$$

Thus,

$$\begin{aligned}\mathcal{L}V \leq & -M\left(\alpha+\mu+\frac{\sigma_2^2}{2}\right)(\mathcal{R}_2^s-1) + \left(\frac{M\beta^2\Lambda}{\left(\mu+\frac{\sigma_1^2}{2}\right)^2}+\beta\right)I_a + \frac{M\beta\delta\Lambda I_a^2}{\mu+\frac{\sigma_1^2}{2}} \\ & +\Lambda(S+I_a)^m - \frac{\mu}{2}S^{m+1} - \frac{\alpha+\mu}{2}I_a^{m+1} - \frac{\Lambda}{S} + \mu + \frac{\sigma_1^2}{2}\end{aligned}$$

Next, we construct the bounded closed set

$$\Pi := \left\{(S, I_a) : \epsilon \leq S \leq \frac{1}{\epsilon}, \epsilon \leq I_a \leq \frac{1}{\epsilon}\right\},$$

where ϵ denotes a sufficiently small constant. We take ϵ based on the following conditions:

$$\begin{aligned}& -\frac{\Lambda}{\epsilon} + \Upsilon_1 \leq -1, \\ & -M\left(\alpha+\mu+\frac{\sigma_2^2}{2}\right)(\mathcal{R}_2^s-1) + \left(\frac{M\beta^2\Lambda}{\left(\mu+\frac{\sigma_1^2}{2}\right)^2}+\beta\right)\epsilon + \frac{M\beta\delta\Lambda\epsilon^2}{\mu+\frac{\sigma_1^2}{2}} + \Upsilon_2 \leq -1, \\ & -\frac{\mu}{2\epsilon^{m+1}} + \Upsilon_1 \leq -1, \\ & -\frac{\alpha+\mu}{2\epsilon^{m+1}} + \Upsilon_1 \leq -1,\end{aligned}$$

where

$$\begin{aligned}\Upsilon_1 = \sup_{(S,I_a)\in\mathbb{R}_+^2}\Bigg\{ & \left(\frac{M\beta^2\Lambda}{\left(\mu+\frac{\sigma_1^2}{2}\right)^2}+\beta\right)I_a + \frac{M\beta\delta\Lambda I_a^2}{\mu+\frac{\sigma_1^2}{2}} + \Lambda(S+I_a)^m - \frac{\mu}{4}S^{m+1} \\ & -\frac{\alpha+\mu}{4}I_a^{m+1} + \mu + \frac{\sigma_1^2}{2}\Bigg\} < \infty.\end{aligned}$$

For the sake of convenience, we partition $\mathbb{R}_+^2 \setminus \Pi$ into four domains,

$$\begin{aligned}\Pi_1 &= \{(S, I_a) \in \mathbb{R}_+^2, 0 < S < \epsilon\}, \\ \Pi_2 &= \{(S, I_a) \in \mathbb{R}_+^2, 0 < I_a < \epsilon\}, \\ \Pi_3 &= \left\{(S, I_a) \in \mathbb{R}_+^2, S \geq \frac{1}{\epsilon}\right\}, \\ \Pi_4 &= \left\{(S, I_a) \in \mathbb{R}_+^2, I_a \geq \frac{1}{\epsilon}\right\}.\end{aligned}$$

We will prove that $\mathcal{L}\widetilde{V}(S, I_a) \leq -1$ on $\mathbb{R}_+^2 \setminus \Pi$, which is equivalent to showing it on the above four domains.

Case 1: If $(S, I_a) \in \Pi_1$, one obtains

$$\mathcal{L}\widetilde{V} \leq -\frac{\Lambda}{S} + \Upsilon_1 \leq -\frac{\Lambda}{\epsilon} + \Upsilon_1 \leq -1.$$

Case 2: If $(S, I_a) \in \Pi_2$, one obtains

$$\mathcal{L}\widetilde{V} \leq -M\left(\alpha + \mu + \frac{\sigma_2^2}{2}\right)(\mathcal{R}_2^s - 1) + \left(\frac{M\beta^2\Lambda}{\left(\mu + \frac{\sigma_1^2}{2}\right)^2} + \beta\right)\epsilon + \frac{M\beta\delta\Lambda\epsilon^2}{\mu + \frac{\sigma_1^2}{2}} + \Upsilon_2 \leq -1.$$

Case 3: If $(S, I_a) \in \Pi_3$, one obtains

$$\mathcal{L}\widetilde{V} \leq -\frac{\mu}{2}S^{m+1} + \Upsilon_1 \leq -\frac{\mu}{2\epsilon^{m+1}} + \Upsilon_1 \leq -1.$$

Case 4: If $(S, I_a) \in \Pi_4$, one obtains

$$\mathcal{L}\widetilde{V} \leq -\frac{\alpha + \mu}{2}I_a^{m+1} + \Upsilon_1 \leq -\frac{\alpha + \mu}{2\epsilon^{m+1}} + \Upsilon_1 \leq -1.$$

Hence we get $\mathcal{L}\widetilde{V}(S, I_a) < 0$ for $(S, I_a) \in \mathbb{R}_+^2 \setminus \Pi$. Consequently, the model (2.5) has an ergodic stationary distribution $\boldsymbol{\pi}(\cdot)$.

For $C > 0$, the ergodicity property states that

$$\lim_{T\to\infty} \frac{1}{T}\int_0^T (X_i(t) \wedge C)\mathrm{d}t = \int_{\mathbb{X}} (Y_i \wedge C)\boldsymbol{\pi}(\mathrm{d}Y_1, \mathrm{d}Y_2) \quad \text{a.s.} \tag{5.3}$$

In view of the dominated convergence theorem and $\mathbf{E}(X_i(t)) < C_1$ $(i = 1, 2)$ (see Theorem 3.8), we get

$$\mathbf{E}\Big(\lim_{T\to\infty} \frac{1}{T}\int_0^T (X_i(t) \wedge C)\mathrm{d}t\Big) = \lim_{T\to\infty} \frac{1}{T}\int_0^T \mathbf{E}(X_i(t) \wedge C)\mathrm{d}t \leq C_1$$

and

$$\int_{\mathbb{X}} (Y_i \wedge C)\boldsymbol{\pi}(\mathrm{d}Y_1, \mathrm{d}Y_2) \leq C_1.$$

Letting $C \to \infty$ results in $\int_{\mathbb{X}} Y_i\boldsymbol{\pi}(\mathrm{d}Y_1, \mathrm{d}Y_2) \leq C_1$. Therefore, with respect to the measure $\boldsymbol{\pi}(\cdot)$, the function $f(X) = X$ is integrable. □

Remark 5.3 Theorem 5.2 shows that the solution of the SDE model (2.5) can have an asymptotically stationary distribution, suggesting stochastic stability. Furthermore, Theorem 5.2 means that if the theorem's conditions are satisfied, the solution of the model (2.5) oscillates around the endemic equilibrium E^* of the ODE model (2.3), and the model exhibits ergodicity wherein the positive solution converges towards a unique stationary distribution. This demonstrates the disease's persistence under certain conditions.

6 Probability density function analysis

According to the aforementioned study, the global solution (S, I_a) of the model (2.5) has a stationary distribution. Now, we calculate the distribution's explicit local probability density function when $\mathcal{R}_2^s > 1$.

Letting $(x_1, x_2)^T = (\ln S, \ln I_a)^T$, we use Itô's formula to obtain

$$\begin{cases} \mathrm{d}x_1 = \left(\Lambda \mathrm{e}^{-x_1} - \mu - \dfrac{\beta \mathrm{e}^{x_2}}{1 + \delta \mathrm{e}^{2x_2}} - \dfrac{\sigma_1^2}{2}\right) \mathrm{d}t + \sigma_1 \mathrm{d}B(t), \\ \mathrm{d}x_2 = \left(\dfrac{\beta \mathrm{e}^{x_1}}{1 + \delta \mathrm{e}^{2x_2}} - (\alpha + \mu) - \dfrac{\sigma_2^2}{2}\right) \mathrm{d}t + \sigma_2 \mathrm{d}B(t). \end{cases} \tag{6.1}$$

Then, we can know that the model (6.1) has a quasistable equilibrium $E^+ = (S^+, I_a^+) := (\mathrm{e}^{x_1^+}, \mathrm{e}^{x_2^+}) \in \mathbb{R}_+^2$, where

$$S^+ = \frac{(\alpha + \mu + \frac{\sigma_2^2}{2})(1 + \delta I_a^{+2})}{\beta}, \quad I_a^+ = \frac{\sqrt{\beta^2 + 4\delta(\mu + \frac{\sigma_1^2}{2})^2(\mathcal{R}_2^s - 1)} - \beta}{2\delta(\mu + \frac{\sigma_1^2}{2})}. \tag{6.2}$$

In light of the foregoing, let $(y_1, y_2)^T = (x_1 - x_1^+, x_2 - x_2^+)$, where $x_1^+ = \ln S^+$, $x_2^+ = \ln I_a^+$, then the corresponding linearized model (6.1) takes the form

$$\begin{cases} \mathrm{d}y_1 = (-d_{11} y_1 - d_{12} y_2)\mathrm{d}t + \sigma_1 \mathrm{d}B(t), \\ \mathrm{d}y_2 = (d_{21} y_1 - d_{22} y_2)\mathrm{d}t + \sigma_2 \mathrm{d}B(t), \end{cases} \tag{6.3}$$

where

$$\begin{aligned} &d_{11} = \Lambda \mathrm{e}^{-x_1^+} > 0, \quad d_{12} = \frac{\beta \mathrm{e}^{x_2^+}(1 - \delta \mathrm{e}^{2x_2^+})}{(1 + \delta \mathrm{e}^{2x_2^+})^2}, \\ &d_{21} = \frac{\beta \mathrm{e}^{x_1^+}}{1 + \delta \mathrm{e}^{2x_2^+}} > 0, \quad d_{22} = \frac{2\beta\delta \mathrm{e}^{x_1^+} \mathrm{e}^{2x_2^+}}{(1 + \delta \mathrm{e}^{2x_2^+})^2} > 0. \end{aligned} \tag{6.4}$$

Then, the local probability density function for the model (2.5) is described in the following theorem.

Theorem 6.1 *Assuming that $\mathcal{R}_2^s > 1$, for any initial value $(S(0), I_a(0)) \in \mathbb{R}_+^2$, the stationary distribution of the model (2.5) around $E^+ = (S^+, I_a^+)$ has a unique log-normal density function $\mathcal{P}(S, I_a)$, which is defined as*

$$\mathcal{P}(S, I_a) = (2\pi)^{-1} |\Sigma|^{-\frac{1}{2}} \mathrm{e}^{-\frac{1}{2}\left(\ln \frac{S}{S^+}, \ln \frac{I_a}{I_a^+}\right) \Sigma^{-1} \left(\ln \frac{S}{S^+}, \ln \frac{I_a}{I_a^+}\right)^T}, \tag{6.5}$$

where $\Sigma = \varrho_1^2 \mathcal{Z}_1^{-1} \Sigma_{10} (\mathcal{Z}_1^{-1})^T + \varrho_2^2 \mathcal{Z}_2^{-1} \Sigma_{20} (\mathcal{Z}_2^{-1})^T$ *is a positive definite matrix with* $\varrho_1 = d_{21}\sigma_1$, $\varrho_2 = \sigma_2$,

$$\Sigma_{10} = \begin{pmatrix} \frac{1}{2(d_{11}+d_{22})} & 0 \\ 0 & \frac{1}{2(d_{11}+d_{22})(d_{11}d_{22}+d_{12}d_{21})} \end{pmatrix},$$

$$\Sigma_{20} = \begin{pmatrix} \frac{d_{11}d_{22}+d_{12}d_{21}+d_{22}^2}{2(d_{11}+d_{22})(d_{11}d_{22}+d_{12}d_{21})} & -\frac{d_{12}d_{22}}{2(d_{11}+d_{22})(d_{11}d_{22}+d_{12}d_{21})} \\ -\frac{d_{12}d_{22}}{2(d_{11}+d_{22})(d_{11}d_{22}+d_{12}d_{21})} & \frac{d_{12}^2}{2(d_{11}+d_{22})(d_{11}d_{22}+d_{12}d_{21})} \end{pmatrix},$$

and

$$\mathcal{Z}_1 = \begin{pmatrix} d_{21} & -d_{22} \\ 0 & 1 \end{pmatrix}, \quad \mathcal{Z}_2 = \begin{pmatrix} -\frac{d_{21}}{d_{12}} & 0 \\ -\frac{d_{21}}{d_{12}d_{22}} & 1 \end{pmatrix}.$$

Proof For simplicity, let $\mathcal{Y} = (y_1, y_2)^T$, $\hat{G} = \operatorname{diag}(\sigma_1, \sigma_2)$, and

$$A = \begin{pmatrix} -d_{11} & -d_{12} \\ d_{21} & -d_{22} \end{pmatrix}.$$

We then rewrite the model (6.3) as $\mathrm{d}\mathcal{Y} = A\mathcal{Y}\mathrm{d}t + \hat{G}\mathrm{d}B(t)$. The density function $\mathcal{P}(\mathcal{Y})$ around $E^+ = (S^+, I_a^+)$, according to the theory in [38], fulfills the Fokker–Planck equation as follows:

$$-\sum_{i=1}^{2} \frac{\sigma_i^2}{2} \frac{\partial^2}{\partial y_i^2} \mathcal{P} + \frac{\partial}{\partial y_1}\big[(-d_{11}y_1 - d_{12}y_2)\mathcal{P}\big] + \frac{\partial}{\partial y_2}\big[(d_{21}y_1 - d_{22}y_2)\mathcal{P}\big] = 0, \tag{6.6}$$

where $\mathcal{P}(\mathcal{Y}) = c\mathrm{e}^{-\frac{1}{2}\mathcal{Y}\mathcal{W}\mathcal{Y}^T}$, $\int_{\mathbb{R}_+^2} \mathcal{P}(\mathcal{Y})\mathrm{d}\mathcal{Y} = 1$ ensures $c > 0$, and $\mathcal{W}$ is a real symmetric matrix [39]. Then $\mathcal{W}$ obeys the algebraic equation $\mathcal{W}\hat{G}^2\mathcal{W} + A^T\mathcal{W} + \mathcal{W}A = 0$. Letting $\Sigma = \mathcal{W}^{-1}$, the following is an equivalent equation:

$$\hat{G}^2 + A\Sigma + \Sigma A^T = 0. \tag{6.7}$$

Using the concept of finite independent superposition [40], Eq. (6.7) is identical to putting two equations together:

$$\hat{G}_i^2 + A\Sigma_i + \Sigma_i A^T = 0, \quad i = 1, 2,$$

where $\hat{G}_1 = \operatorname{diag}(\sigma_1, 0)$, $\hat{G}_2 = \operatorname{diag}(0, \sigma_2)$, $\Sigma = \Sigma_1 + \Sigma_2$, and $\hat{G}^2 = \hat{G}_1^2 + \hat{G}_2^2$. The characteristic polynomial of A is

$$\mathcal{F}_A(\lambda) = \lambda^2 + (d_{11} + d_{22})\lambda + d_{11}d_{22} + d_{12}d_{21} \tag{6.8}$$

and, if $d_{11}d_{22} + d_{12}d_{21} > 0$, then all the real-parts of the eigenvalues are negative. To obtain the specific expression for Σ of Eq. (6.7), two procedures must be completed.

First, for the equation

$$\hat{G}_1^2 + A\Sigma_1 + \Sigma_1 A^T = 0, \tag{6.9}$$

where $\hat{G}_1 = \mathrm{diag}(\sigma_1, 0)$, we can calculate a matrix

$$\mathcal{B}_1 = \begin{pmatrix} -d_{11} - d_{22} & -d_{11}d_{22} - d_{12}d_{21} \\ 1 & 0 \end{pmatrix} = \mathcal{Z}_1 A \mathcal{Z}_1^{-1}, \tag{6.10}$$

where $\mathcal{Z}_1 = \begin{pmatrix} d_{21} & -d_{22} \\ 0 & 1 \end{pmatrix}$. Equation (6.9) is changed to the following equation:

$$\mathcal{Z}_1 \hat{G}_1^2 \mathcal{Z}_1^T + \mathcal{B}_1 \mathcal{Z}_1 \Sigma_1 \mathcal{Z}_1^T + \mathcal{Z}_1 \Sigma_1 \mathcal{Z}_1^T \mathcal{B}_1^T = 0,$$

which is equivalent to

$$G_0^2 + \mathcal{B}_1 \Sigma_{10} + \Sigma_{10} \mathcal{B}_1^T = 0,$$

where $G_0 = \mathrm{diag}(1, 0)$, $\Sigma_{10} = \varrho_1^{-2} \mathcal{Z}_1 \Sigma_1 \mathcal{Z}_1^T$, $\varrho_1 = d_{21}\sigma_1$, and we can derive

$$\Sigma_{10} = \begin{pmatrix} \frac{1}{2(d_{11}+d_{22})} & 0 \\ 0 & \frac{1}{2(d_{11}+d_{22})(d_{11}d_{22}+d_{12}d_{21})} \end{pmatrix},$$

which is positive definite. Thus, we obtain that $\Sigma_1 = \varrho_1^2 \mathcal{Z}_1^{-1} \Sigma_{10} (\mathcal{Z}_1^{-1})^T$ is positive definite.

Second, consider the algebraic equation

$$\hat{G}_2^2 + A\Sigma_2 + \Sigma_2 A^T = 0, \tag{6.11}$$

where $\hat{G}_2 = \mathrm{diag}(0, \sigma_2)$. Similarly, we can get the following matrix:

$$\mathcal{B}_2 = \begin{pmatrix} -d_{11} & d_{21} \\ -d_{12} & -d_{22} \end{pmatrix} = \mathcal{Z}_2 A \mathcal{Z}_2^{-1}, \tag{6.12}$$

where $\mathcal{Z}_2 = \begin{pmatrix} -\frac{d_{21}}{d_{12}} & 0 \\ -\frac{d_{21}}{d_{12}d_{22}} & 1 \end{pmatrix}$. Equation (6.11) can then be changed to the following equation:

$$\mathcal{Z}_2 \hat{G}_2^2 \mathcal{Z}_2^T + \mathcal{B}_2 \mathcal{Z}_2 \Sigma_2 \mathcal{Z}_2^T + \mathcal{Z}_2 \Sigma_2 \mathcal{Z}_2^T \mathcal{B}_2^T = 0,$$

which is equivalent to

$$G_0^2 + \mathcal{B}_2 \Sigma_{20} + \Sigma_{20} \mathcal{B}_2^T = 0,$$

where

$$\Sigma_{20} = \begin{pmatrix} \frac{d_{11}d_{22}+d_{12}d_{21}+d_{22}^2}{2(d_{11}+d_{22})(d_{11}d_{22}+d_{12}d_{21})} & -\frac{d_{12}d_{22}}{2(d_{11}+d_{22})(d_{11}d_{22}+d_{12}d_{21})} \\ -\frac{d_{12}d_{22}}{2(d_{11}+d_{22})(d_{11}d_{22}+d_{12}d_{21})} & \frac{d_{12}^2}{2(d_{11}+d_{22})(d_{11}d_{22}+d_{12}d_{21})} \end{pmatrix} = \varrho_2^{-2} \mathcal{Z}_2 \Sigma_2 \mathcal{Z}_2^T,$$

and $\varrho_2 = \sigma_2$, so we also have the positive definite matrix $\Sigma_2 = \varrho_2^2 \mathcal{Z}_2^{-1} \Sigma_{20} (\mathcal{Z}_2^{-1})^T$.

Thereupon, because $\Sigma = \Sigma_1 + \Sigma_2$ in Eq. (6.7) is positive definite, a locally and nearly normal probability density function $\mathcal{P}(S, I_a)$ exists around the point E^+. □

Remark 6.2 Theorems 5.2 and 6.1 show that if $\mathcal{R}_2^s > 1$, the unique ergodic stationary distribution of the model (2.5) admits the corresponding probability density function.

7 Simulation results

Several numerical simulations are performed to validate dynamical findings for the model (2.5). We employ Milstein's higher order method [41] to simulate and investigate the impact of environmental fluctuations on disease spreading by studying the dynamics of the stochastic HIV model in asymptomatic HIV-infected and symptomatic HIV-infected individuals. The corresponding biological parameters and initial value of the model (2.5) are shown in Table 1, and we fix the following parameters:

$$\Lambda = 5,\ \alpha = 4,\ \delta = 2,\ \gamma = 0.2,\ \mu = 0.1,\ \beta = 0.1. \tag{7.1}$$

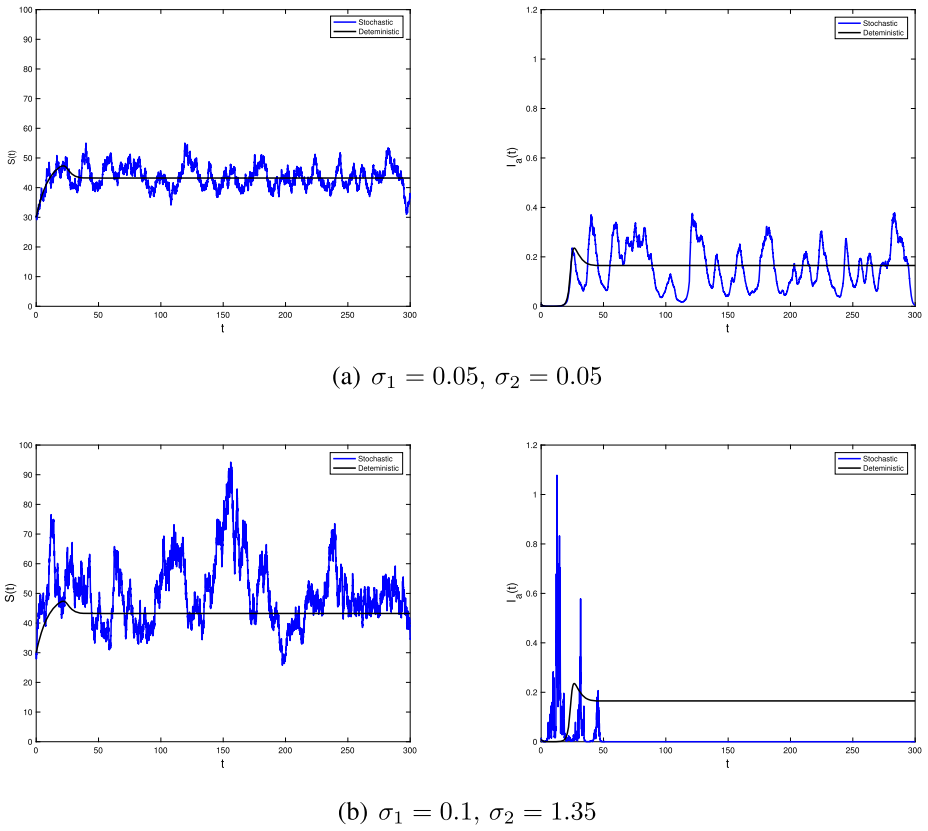
Then we obtain $\mathcal{R}_0 = \frac{\beta\Lambda}{\mu(\alpha+\mu)} = 1.2195 > 1$ and the endemic equilibrium $E^* = (43.2335, 0.165)$ which is globally asymptotically stable. Furthermore, there is an unstable disease-free equilibrium $E_0 = (\frac{\Lambda}{\mu}, 0) = (50, 0)$ in the model (2.5).

In Fig. 1(a), if we choose the environmental forcing intensities $(\sigma_1, \sigma_2) = (0.05, 0.05)$, then simple calculations show that $\mathcal{R}_1^s = \frac{\Lambda\beta}{\mu(\alpha+\mu+\frac{\sigma_2^2}{2})} = 1.2191 > 1$ and $\mathcal{R}_2^s = \frac{\Lambda\beta}{(\mu+\frac{\sigma_1^2}{2})(\alpha+\mu+\frac{\sigma_2^2}{2})} = 1.2041 > 1$. Based on Theorem 5.2, it can be inferred that the disease exhibits persistence. The profiles displayed on the right of Fig. 1(a) demonstrate that the trajectory of $I_a(t)$ fluctuates in proximity to the deterministic steady state value of $I_a^* = 0.165$. Increasing (σ_1, σ_2) to $(0.1, 1.35)$, direct calculations yield $\mathcal{R}_2^s = 0.9502 < \mathcal{R}_1^s = 0.9978 < 1 < \mathcal{R}_0 = 1.2195$. From Theorem 4.1, the infectious variable $I_a(t)$ in the model (2.5) is almost certain to disappear. Figure 1(b) indicates that large enough environmental noises cause the disease to die out.

In Fig. 2, we adopt $(\sigma_1, \sigma_2) = (0.05, 0.05)$ and $(0.05, 0.1)$, respectively, and then $\mathcal{R}_1^s$ and $\mathcal{R}_2^s$ are larger than 1. For the model (2.5), Figs. 2(a) and 2(b) depict histograms of the probability density function for $S(t)$ and $I_a(t)$. At the epidemic equilibrium $E^* = (43.2335, 0.165)$ of the deterministic model (2.4), the oscillations exhibit a symmetric distribution. Furthermore, these fluctuations are represented in the stationary distributions of the model.

Table 1 Definitions of variables and parameters in model (2.5)

Parameters	Description	Value	Resource
Λ	A constant rate of recruitment population	≥ 0.5	[21, 42]
β	Transmission rate of the susceptible individuals	[0.005, 1]	[21, 36]
μ	Natural mortality rate of the population	[0.05, 1]	[21]
δ	Psychological or inhibitory effects	[0.001, 4]	[16, 32]
α	Diagnosis rate of infected population	[0.01, 10]	[7]
γ	Disease mortality of the symptomatic infected individuals	[0.1, 1]	[1]
$(S(0), I_a(0))$	The initial value of the model (2.5)	(30, 20)	Assumed
σ_1, σ_2	The intensities of the noise	Variables	Estimated



(a) $\sigma_1 = 0.05,\ \sigma_2 = 0.05$

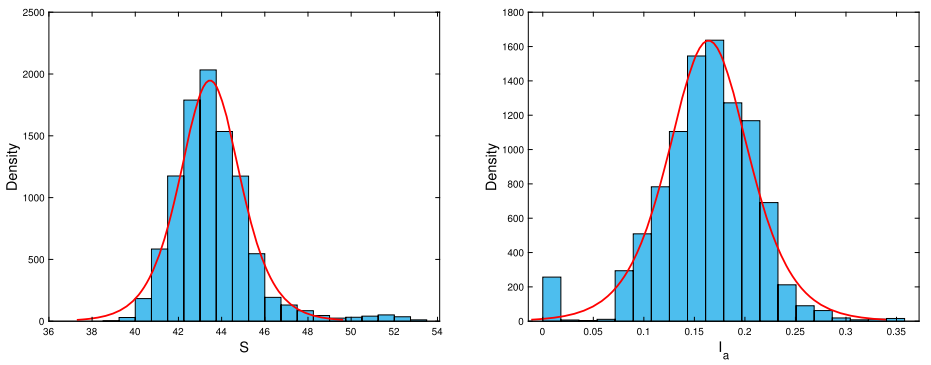
(b) $\sigma_1 = 0.1,\ \sigma_2 = 1.35$

Figure 1 The paths of $S(t)$ and $I_a(t)$ for the model (2.5) with different noise intensities (σ_1, σ_2) and other parameters taken as in (7.1)

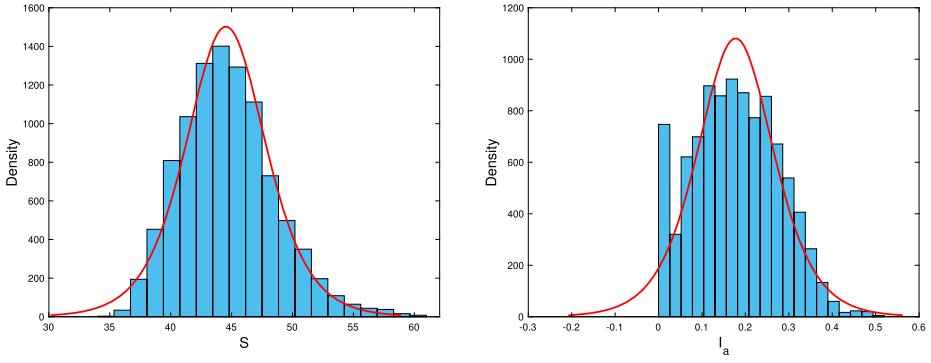
In Fig. 3, we take $(\sigma_1, \sigma_2) = (0.05, 0.05)$, $(0.05, 0.1)$, and $(0.1, 0.1)$, respectively, and the valuess of $\mathcal{R}_1^s$ and $\mathcal{R}_2^s$ are all larger than 1. From Theorem 5.2, we see that $I_a(t)$ is almost certainly a persistent disease. Figures 3(a) and 3(b) depict how $S(t)$ and $I_a(t)$ mean values get smaller and smaller as the intensity of random disturbance increases, while the $I_a(t)$ distribution has a higher negative skew.

Moreover, we also display how the graph of the probability density function for individuals from S and I_a changes with respect to the psychological or inhibitory effect δ. Figures 4(a) and 4(b) illustrate graphs of $\mathcal{U}_1(S, \delta)$ and $\mathcal{U}_2(I_a, \delta)$ derived using noise intensities $\sigma_1 = 0.05$, $\sigma_2 = 0.05$, and the other parameters taken as in (7.1), respectively. It is obvious that a high psychological or inhibitory rate δ might reduce disease transmission.

Regrettably, Theorems 4.1 and 5.2 do not provide any information regarding the disease dynamics of the stochastic model (2.5) when $\mathcal{R}_2^s < 1 < \mathcal{R}_1^s$. In Fig. 5, we present a schematic of the SDE model's (2.5) stochastic dynamics of extinction and endemic. In other words, the disease is extinct if the parameters are in domain I; otherwise, the disease is persistent if the parameters are in domain II, and the model has an ergodic stationary distribution. In Fig. 6, when then parameters locate in domain III, we choose $(\sigma_1, \sigma_2) = (0.22, 0.05)$, $(0.25, 0.05)$, $(0.3, 0.1)$, and in the case of $\mathcal{R}_2^s < 1 < \mathcal{R}_1^s$, we concentrate on the impact of σ_1 and σ_2 on the disease dynamics for the model (2.5). They show that the infectious population $I_a(t)$ will become extinct as soon as possible.

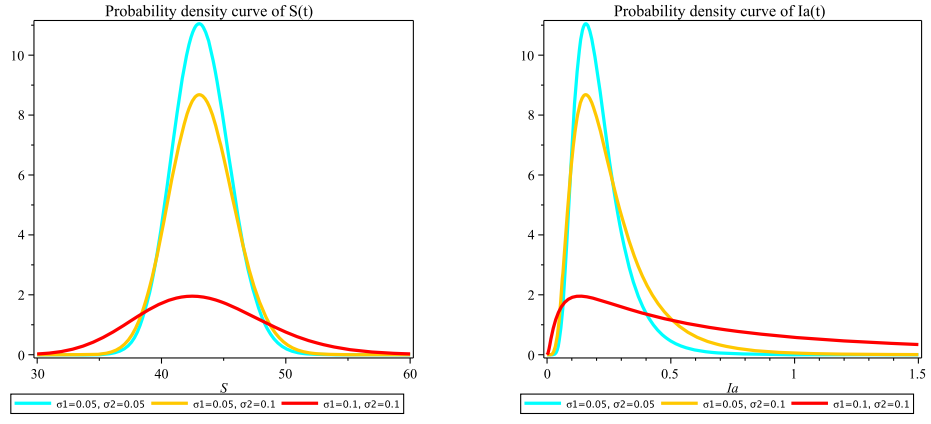


(a) $\sigma_1 = 0.05,\ \sigma_2 = 0.05$



(b) $\sigma_1 = 0.05,\ \sigma_2 = 0.1$

Figure 2 Probability density function histogram for $S(t)$ and $I_a(t)$ at $t = 1000$ for the model (2.5) with two different values of σ_1 and σ_2: (a) $(\sigma_1, \sigma_2) = (0.05, 0.05)$ and (b) $(\sigma_1, \sigma_2) = (0.05, 0.1)$, the probability density functions of $S(t)$ and $I_a(t)$ are represented by the red smoothed curves, keeping other parameters the same as in (7.1). The number of simulations for the frequency histogram fitting density curves of $S(t)$ and $I_a(t)$ of (2.5) is 1000000, and the run time of our code is about 5.386 seconds



(a) Probability density curve of S (b) Probability density curve of I_a

Figure 3 The probability density functions of S and I_a for the model (2.5) with $(\sigma_1, \sigma_2) = (0.05, 0.05)$, $(0.05, 0.1)$, and $(0.1, 0.1)$, respectively. The other parameters are taken as in (7.1)

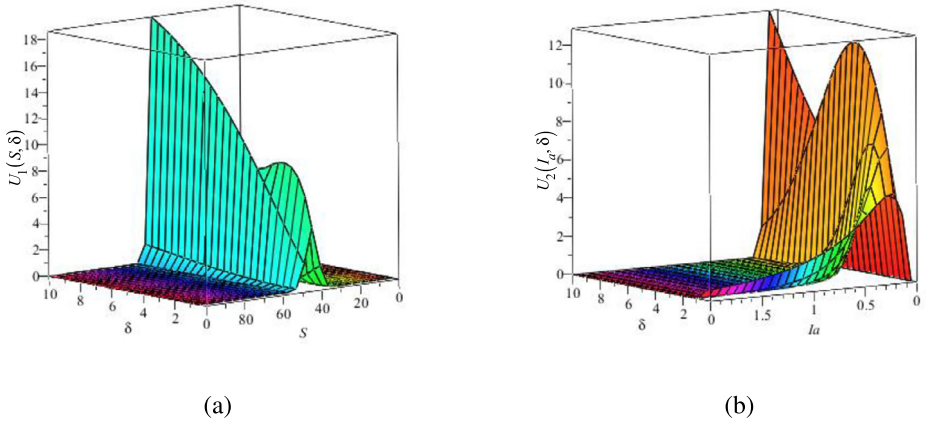
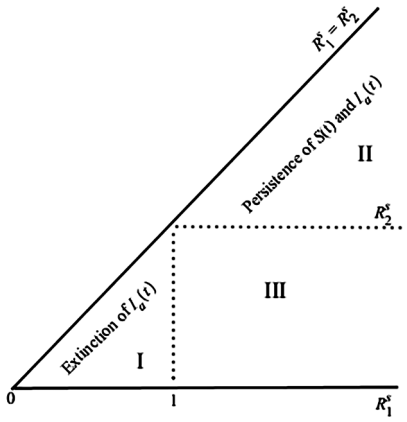
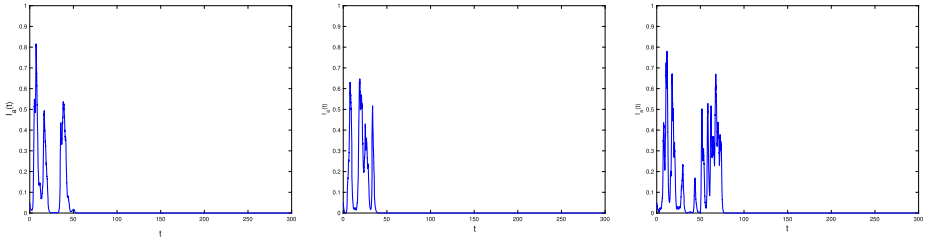


Figure 4 Graphs of the probability density function: (a) $\mathcal{U}_1(S, \delta)$ and (b) $\mathcal{U}_2(I_a, \delta)$ with $\sigma_1 = 0.05$, $\sigma_2 = 0.05$, and the other parameters taken as in (7.1)

Figure 5 Schematic of the SDE model's (2.5) stochastic dynamics of extinction and endemic





(a) $\sigma_1 = 0.22$, $\sigma_2 = 0.05$ (b) $\sigma_1 = 0.25$, $\sigma_2 = 0.05$ (c) $\sigma_1 = 0.3$, $\sigma_2 = 0.1$

Figure 6 The path of $I_a(t)$ for the model (2.5), with the other parameters set as in (7.1)

8 Conclusion

A stochastic model of the HIV infection including asymptomatic and symptomatic infected individuals is presented in this paper. We assume the environmental fluctuations are of the white noise variety and disrupt two populations, S and I_a. On the basis of this, we investigate if the stochastic model (2.5) is stable and how environmental fluctuations affect it to understand how the HIV disease might spread in the long run.

Based on the mathematical analysis, the stochastic model (2.5) provides two random equilibria: one with endemic disease and the other without the disease. We then derive two threshold parameters, $\mathcal{R}_1^s = \frac{\beta\Lambda}{\mu(\alpha+\mu+\frac{\sigma_2^2}{2})}$ and $\mathcal{R}_2^s = \frac{\beta\Lambda}{(\mu+\frac{\sigma_1^2}{2})(\alpha+\mu+\frac{\sigma_2^2}{2})}$, from the proofs of disease elimination in Theorem 4.1 and persistence in Theorem 5.2. That is, depending on whether the values of $\mathcal{R}_1^s$ and $\mathcal{R}_2^s$ are less or larger than one, the disease will either become extinct or continue to exist. If $\mathcal{R}_1^s < 1$, the disease of the model (2.5) will go extinct in the long term (see Theorem 4.1); while if $\mathcal{R}_2^s > 1$, the disease will persist, and the model (2.5) exhibits an ergodic stationary distribution (see Theorem 5.2). However, for the case of $\mathcal{R}_2^s < 1 < \mathcal{R}_1^s$ of the model (2.5), we give some numerical examples to find that when $\mathcal{R}_1^s$ or both $\mathcal{R}_1^s$ and $\mathcal{R}_2^s$ are large, the random fluctuations have the potential to dampen disease outbreaks. Moreover, by solving the Fokker–Planck equation, we derive a log-normal density function $\mathcal{P}(S, I_a)$ of the model (2.5), see Theorem 6.1.

The primary goal of this study is to examine the influence of environmental noise on disease transmission dynamics within a model comprising susceptible and infected populations. Our findings indicate that the population trajectories do not settle at their equilibrium levels but instead fluctuate around these values. Notably, the results suggest that stronger stochastic disturbances are required to accelerate the control of disease transmission. Furthermore, the analysis reveals that increasing the intensity of environmental noise over time can play a crucial role in effectively eradicating disease transmission. These insights contribute to a deeper understanding of the interplay between deterministic and stochastic models, shedding light on the complexities of disease dynamics under varying levels of environmental variability.

Our model is built based on several simplifying assumptions, including homogeneous interactions within the population, fixed parameter values, and a simplified representation of HIV infection stages. While these assumptions facilitate analytical tractability and streamline the modeling process, they do not fully capture the complex and heterogeneous nature of HIV transmission and progression in real-world contexts. Additionally, the model does not adequately account for external and environmental factors such as socioeconomic conditions, behavioral diversity, and disparities in healthcare access. These factors are critical drivers of HIV dynamics, and their omission may limit the model's relevance and applicability to practical scenarios. To address these limitations, future research will focus on developing a more comprehensive model that incorporates coinfections (e.g., tuberculosis), diverse treatment strategies, and variations in healthcare accessibility. By expanding the scope of the model, we aim to improve its ability to reflect real-world complexities. Furthermore, integrating data-driven methodologies to parameterize stochastic elements will enhance the model's realism and predictive power, making it a more effective tool for informing HIV prevention and control efforts.

Appendix

Proof of Lemma 3.1 It follows from the first equation of the model (2.3) that

$$\frac{\mathrm{d}S(t)}{\mathrm{d}t} = \Lambda - \mu S(t) - \frac{\beta S(t) I_a(t)}{1 + \delta I_a^2(t)} \geq -\mu S(t) - \frac{\beta S(t) I_a(t)}{1 + \delta I_a^2(t)}, \tag{A.1}$$

thus

$$\frac{\mathrm{d}S(t)}{\mathrm{d}t} + \left(\mu + \frac{\beta I_a(t)}{1+\delta I_a^2(t)}\right) S(t) \geq 0. \tag{A.2}$$

Letting $f(t) = \mu + \frac{\beta I_a(t)}{1+\delta I_a^2(t)}$ and multiplying both sides of the latter inequality by $\mathrm{e}^{\int_0^t f(s)\mathrm{d}s}$, we obtain

$$\mathrm{e}^{\int_0^t f(s)\mathrm{d}s}\frac{\mathrm{d}S(t)}{\mathrm{d}t} + f(t)\mathrm{e}^{\int_0^t f(s)\mathrm{d}s} S(t) \geq 0 \tag{A.3}$$

and thus derive

$$\frac{\mathrm{d}}{\mathrm{d}t}\left(S(t)\mathrm{e}^{\int_0^t f(s)\mathrm{d}s}\right) \geq 0. \tag{A.4}$$

Integrating this inequality from 0 to t gives

$$\int_0^t \frac{\mathrm{d}}{\mathrm{d}s}\left(S(t)\mathrm{e}^{\int_0^t \left(\mu+\frac{\beta I_a(s)}{1+\delta I_a^2(s)}\right)\mathrm{d}s}\right)\mathrm{d}s \geq 0, \tag{A.5}$$

hence

$$S(t) \geq S(0)\mathrm{e}^{\int_0^t \left(\mu+\frac{\beta I_a(s)}{1+\delta I_a^2(s)}\right)\mathrm{d}s}, \tag{A.6}$$

so that $S(t) > 0$.

Similarly, we can prove that $I_a(t) \geq 0$ and $I_s(t) \geq 0$.

Summing up the three equations of the model (2.3) and letting $N(t) = S(t) + I_a(t) + I_s(t)$, we obtain

$$\Lambda - (\mu+\gamma)N(t) \leq \frac{\mathrm{d}N(t)}{\mathrm{d}t} = \Lambda - \mu N(t) - \gamma I_s \leq \Lambda - \mu N(t). \tag{A.7}$$

By integration, we derive

$$\frac{\Lambda}{\mu+\gamma} + \left(N(0) - \frac{\Lambda}{\mu+\gamma}\right)\mathrm{e}^{-(\mu+\gamma)t} \leq N(t) \leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu}\right)\mathrm{e}^{-\mu t}.$$

Thus, if we take the limit as $t \to \infty$, then $\frac{\Lambda}{\mu+\gamma} \leq \liminf_{t\to\infty} N(t) \leq \limsup_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu}$. This implies that for the model (2.3), the region Γ is a positively invariant set. □

Proof of Lemma 3.2 Setting the right-hand side of the model (2.3) equal to zero, we have

$$\begin{cases} \Lambda - \mu S(t) - \dfrac{\beta S(t) I_a(t)}{1+\delta I_a^2(t)} = 0, \\ \dfrac{\beta S(t) I_a(t)}{1+\delta I_a^2(t)} - (\alpha+\mu) I_a(t) = 0, \\ \alpha I_a(t) - (\mu+\gamma) I_s(t) = 0. \end{cases}$$

Obviously, there exists a disease-free equilibrium $E_0 = (S_0, 0, 0) = (\frac{\Lambda}{\mu}, 0)$ and direct calculations show that the basic reproduction number is $\mathcal{R}_0 = \frac{\beta\Lambda}{\mu(\alpha+\mu)}$.

We get the endemic equilibrium $E^* = (S^*, I_a^*, I_s^*)$ if $I_a(t) \neq 0$, where

$$S^* = \frac{(\mu+\alpha)(1+\delta I_a^{*2})}{\beta},\ I_a^* = \frac{\sqrt{\beta^2 - 4\mu^2\delta(1-\mathcal{R}_0)} - \beta}{2\mu\delta},\ I_s^* = \frac{\alpha}{\mu+\gamma} I_a^*.$$

Hence, if $\mathcal{R}_0 > 1$, then $E^* = \left(S^*, I_a^*, I_s^*\right)$ exists and $E_0 = (\frac{\Lambda}{\mu}, 0, 0)$ always exists. □

Proof of Theorem 3.4 Define the Lyapunov function

$$V(S, I_a, I_s) = \frac{1}{2}\left(S - \frac{\Lambda}{\mu}\right)^2 + a_1 I_a + a_2 I_s, \tag{A.8}$$

where positive constants a_1 and a_2 will be decided later. Then the derivative of V along the solution of the model (2.3) is provided by

$$\begin{aligned}
\frac{\mathrm{d}V}{\mathrm{d}t} &= \left(S - \frac{\Lambda}{\mu}\right)\frac{\mathrm{d}S}{\mathrm{d}t} + a_1\frac{\mathrm{d}I_a}{\mathrm{d}t} + a_2\frac{\mathrm{d}I_s}{\mathrm{d}t} \\
&= -\mu\left(S - \frac{\Lambda}{\mu}\right)^2 - \left(S - \frac{\Lambda}{\mu}\right)\left(\frac{\beta I_a}{1+\delta I_a^2}\left(S - \frac{\Lambda}{\mu}\right) + \frac{\Lambda\beta I_a}{\mu(1+\delta I_a^2)}\right) \\
&\quad + \frac{a_1\beta S I_a}{1+\delta I_a^2} - (a_1(\alpha+\mu) - a_2\alpha)I_a - a_2(\mu+\gamma)I_s \\
&\leq -\left(\mu + \frac{\beta I_a}{1+\delta I_a^2}\right)\left(S - \frac{\Lambda}{\mu}\right)^2 - \frac{a_1\mu(\alpha+\mu)(1-\mathcal{R}_0) - a_2\mu\alpha}{\mu(1+\delta I_a^2)} I_a - a_2(\mu+\gamma)I_s.
\end{aligned} \tag{A.9}$$

Note that S, I_a, and I_s are nonnegative. If $\mathcal{R}_0 < 1$ and $\frac{a_1}{a_2} \geq \frac{\alpha}{(\alpha+\mu)(1-\mathcal{R}_0)}$, the right-hand side of (A.9) contains only nonpositive terms, i.e., $\frac{\mathrm{d}V}{\mathrm{d}t} \leq 0$, and if $S = \frac{\Lambda}{\mu}$, $I_a = 0$, $I_s = 0$, then $\frac{\mathrm{d}V}{\mathrm{d}t} = 0$. Hence, the singleton $\{E_0\}$ is the largest invariant set in $\left\{(S, I_a, I_s) : \frac{\mathrm{d}V}{\mathrm{d}t} = 0\right\}$. Therefore, E_0 is globally asymptotically stable if $\mathcal{R}_0 < 1$.

The Jacobian matrix of the model (2.3) evaluated at E_0 is as follows:

$$J(E_0) = \begin{pmatrix} -\mu & -\beta S_0 & 0 \\ 0 & \beta S_0 - (\alpha+\mu) & 0 \\ 0 & \alpha & -(\mu+\gamma) \end{pmatrix}, \tag{A.10}$$

which has three eigenvalues $\lambda_1 = -\mu$, $\lambda_2 = \beta S_0 - (\alpha+\mu) = (\alpha+\mu)(\mathcal{R}_0 - 1)$, and $\lambda_3 = -(\mu+\gamma)$. Thus, when $\mathcal{R}_0 > 1$, E_0 becomes unstable. □

Proof of Theorem 3.5 By adding all the equations of the model (2.3), we discover that the whole population $N(t) = S(t) + I_a(t) + I_s(t)$ satisfies

$$\frac{\mathrm{d}N}{\mathrm{d}t} = \Lambda - \mu N - \gamma I_s. \tag{A.11}$$

Then the model (2.3) is equivalent to the following:

$$\begin{cases} \dfrac{\mathrm{d}N}{\mathrm{d}t} = \Lambda - \mu N - \gamma I_s, \\ \dfrac{\mathrm{d}I_a}{\mathrm{d}t} = \dfrac{\beta I_a}{1+\delta I_a^2}(N - I_a - I_s) - (\alpha+\mu) I_a, \\ \dfrac{\mathrm{d}I_s}{\mathrm{d}t} = \alpha I_a - (\mu+\gamma) I_s, \end{cases} \tag{A.12}$$

and $N^* = S^* + I_a^* + I_s^*$. Hence, if we can show the global stability of (N^*, I_a^*, I_s^*), then the conclusion of the theorem will be confirmed.

Consider the following function:

$$V(N, I_a, I_s) = \frac{1}{2}(N - N^*)^2 + c_1\left(I_a - I_a^* - I_a^* \log\frac{I_a}{I_a^*}\right) + \frac{1}{2}c_2(I_s - I_s^*)^2, \tag{A.13}$$

where the positive constants c_1 and c_2 will be found subsequently, and

$$\begin{aligned} \frac{\mathrm{d}V}{\mathrm{d}t} &= (N - N^*)\frac{\mathrm{d}N}{\mathrm{d}t} + c_1\frac{I_a - I_a^*}{I_a}\frac{\mathrm{d}I_a}{\mathrm{d}t} + c_2(I_s - I_s^*)\frac{\mathrm{d}I_s}{\mathrm{d}t} \\ &= -\mu(N - N^*)^2 - \gamma(N - N^*)(I_s - I_s^*) + c_2\alpha(I_s - I_s^*)(I_a - \frac{I_a^* I_s}{I_s^*}) \\ &\quad + c_1(I_a - I_a^*)\left(\frac{\beta(N - I_a - I_s)}{1+\delta I_a^2} - \frac{\beta(N^* - I_a^* - I_s^*)}{1+\delta I_a^{*2}}\right) \\ &= -\mu(N - N^*)^2 - \Big(\frac{d_1\beta}{1+\delta I_a^2} + \frac{c_1\beta\delta(N^* - I_a^* - I_s^*)(I_a + I_a^*)}{(1+\delta I_a^2)(1+\delta I_a^{*2})}\Big)(I_a - I_a^*)^2 \\ &\quad - \frac{c_2\alpha I_a^*}{I_s^*}(I_s - I_s^*)^2 + \frac{c_1\beta}{1+\delta I_a^2}(N - N^*)(I_a - I_a^*) - \gamma(N - N^*)(I_s - I_s^*) \\ &\quad - (\frac{c_1\beta}{1+\delta I_a^2} - c_2\alpha)(I_a - I_a^*)(I_s - I_s^*). \end{aligned} \tag{A.14}$$

Letting $\frac{c_1\beta}{1+\delta I_a^2} = c_2\alpha$ and $U = (N - N^*, I_a - I_a^*, I_s - I_s^*)^{\mathrm{T}}$ yields

$$\frac{\mathrm{d}V}{\mathrm{d}t} = -U^{\mathrm{T}}\begin{pmatrix} \mu & -\frac{c_2\alpha}{2} & \frac{\gamma}{2} \\ -\frac{c_2\alpha}{2} & c_2\alpha\big(1 + \frac{\delta(N^* - I_a^* - I_s^*)(I_a + I_a^*)}{1+\delta I_a{}^{*2}}\big) & 0 \\ \frac{\gamma}{2} & 0 & \frac{c_2\alpha I_a^*}{I_s^*} \end{pmatrix} U \triangleq -U^{\mathrm{T}}AU. \tag{A.15}$$

Choosing $c_2 < \min\left\{\frac{4\mu}{\alpha}, \frac{4\mu\delta(N^* - I_a^* - I_s)I_a^*}{\beta(1+\delta I_a^{*2})}\right\}$, it immediately follows from the positive-definiteness of the matrix A that $\frac{\mathrm{d}V}{\mathrm{d}t} < 0$. We find that (N^*, I_a^*, I_s^*) is globally asymptotically stable [43]. As a result, $E^* = (S^*, I_a^*, I_s^*)$ is globally asymptotically stable. □

Proof of Theorem 3.8 Let $N(t) = S(t) + I_a(t)$ and

$$V(t) = N(t) + \frac{1}{N(t)}.$$

Using Itô's formula and (A.17), we have

$$\begin{aligned}\mathbf{E}(\mathrm{e}^{\mu t}V(t)) &= \mathbf{E}(V(0)) + \mathbf{E}\Big(\int_0^t \mathrm{e}^{\mu s}(\mu V(s) + \mathcal{L}V(s))\mathrm{d}s\Big)\\ &\le \mathbf{E}(V(0)) + C\mathbf{E}\Big(\int_0^t \mathrm{e}^{\mu s}\mathrm{d}s\Big)\\ &= \mathbf{E}(V(0)) + \frac{C}{\mu}(\mathrm{e}^{\mu t} - 1).\end{aligned}$$

It follows that

$$\begin{aligned}\mathbf{E}(V(t)) &\le \mathrm{e}^{-\mu t}\mathbf{E}(V(0)) + \frac{C}{\mu}(1 - \mathrm{e}^{-\mu t})\\ &\le \mathbf{E}(V(0)) + \frac{C}{\mu} := \widetilde{C}.\end{aligned}$$

We choose a big enough constant ζ such that $\frac{\widetilde{C}}{\zeta} < 1$ and apply Chebyshev's inequality to obtain

$$\mathbf{P}\Big\{N + \frac{1}{N} > \zeta\Big\} \le \frac{1}{\zeta}\mathbf{E}\Big(N + \frac{1}{N}\Big) \le \frac{\widetilde{C}}{\zeta} := \varepsilon.$$

This implies

$$1 - \varepsilon \le \mathbf{P}\Big\{N + \frac{1}{N} \le \zeta\Big\} \le \mathbf{P}\Big\{\frac{1}{\zeta} \le N \le \zeta\Big\}.$$

Noting that $N^2 \le 2|X|^2 \le 2N^2$, we have

$$\mathbf{P}\Big\{\frac{1}{\sqrt{2}\zeta} \le \frac{N}{\sqrt{2}} \le |X| \le N \le \zeta\Big\} \ge 1 - \varepsilon.$$

According to the definitions of stochastic ultimate boundedness and stochastic permanence [44, 45], the SDE model (2.5) is stochastically bounded and permanent. □

Proof of Theorem 3.9 Letting $N(t) = S(t) + I_a(t)$ and

$$V(X(t)) = N(t) + \frac{1}{N(t)} \tag{A.16}$$

for $X(t) \in \mathbb{X}$, we then have $V(X(t)) \to \infty$ as the norm $|X| = \sqrt{X_1^2 + X_2^2} \to \infty$. Applying Itô's formula, we derive

$$\begin{aligned}\mathcal{L}V(X(t)) &= \Lambda - \mu N - \alpha I_a - \frac{\Lambda - \mu N - \alpha I_a}{N^2} + \frac{\sigma_1^2 S^2 + \sigma_2^2 I_a^2}{N^3}\\ &\le \Lambda - \mu(N + \frac{1}{N}) + \frac{2\mu}{N} - \frac{\Lambda}{N^2} + \frac{\alpha I_a}{N^2} + \frac{\sigma_1^2 S^2 + \sigma_2^2 I_a^2}{N^3}\\ &\le \Lambda - \frac{\Lambda}{N^2} + \frac{2\mu + \alpha + \sigma_1^2 + \sigma_2^2}{N} - \mu(N + \frac{1}{N})\\ &\le C - \mu V(X),\end{aligned} \tag{A.17}$$

where $C = \frac{4\Lambda^2+(2\mu+\alpha+\sigma_1^2+\sigma_2^2)^2}{4\Lambda}$.

The model (2.5) is uniformly elliptic, thus there exists a jointly continuous function $p : (0,\infty)\times\mathbb{X}\times\mathbb{X}\to(0,\infty)$ such that $p_t(X_0,Z)$ remains strictly positive for all (t,X_0,Z), and for every measure set $\mathbb{A}$ [46],

$$\mathbf{P}_t(X_0,\mathbb{A}) = \int_{\mathbb{A}} p_t(X_0,Z)\mathrm{d}Z. \tag{A.18}$$

For $\omega>0$, then $\inf\{p_t(X_0,Z) : X_0,Z\in\mathbb{X},|X_0|,|Z|\le\omega\}\ge\widehat{C}$ (a constant $\widehat{C}=\widehat{C}(\omega,t)>0$). For any set $\mathbb{A}$,

$$\mathbf{P}_t(X,\mathbb{A}) = \int_{\mathbb{A}} p_t(X_0,Z)\mathrm{d}Z \ge \widehat{C}\,\mathrm{Leb}(\mathbb{A}\cap\mathcal{B}_\omega(0)) = \widehat{C}\,\mathrm{Leb}(\mathcal{B}_\omega(0))\boldsymbol{p}(\mathbb{A})$$

provides the minorization condition, where Leb denotes the Lebesgue measure and $\boldsymbol{p}(\mathbb{A}) = \mathrm{Leb}(\mathbb{A}\cap\mathcal{B}_\omega(0))/\mathrm{Leb}(\mathcal{B}_\omega(0))$. This ends the proof. □

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FR: writing–original draft, visualization, software, formal analysis, reviewing, and editing. YT: formal analysis, visualization. XL: visualization, reviewing, and editing. All authors read and approved the final manuscript.

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Data availability
There are no data associated with the manuscript.

Declarations

Competing interests
The authors declare no competing interests.

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