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Research Article

Quantifying Uncertainty in HIV/AIDS Transmission: A Stochastic SEITA Model with Sensitivity Analysis

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Abstract: Human Immunodeficiency Virus/Acquired Immune Deficiency Syndrome (HIV/AIDS) remains a global health crisis characterized by significant inherent uncertainties in transmission and intervention effectiveness, which are poorly captured by deterministic models. This study aims to develop a stochastic Susceptible-Exposed-Infected-Treated-AIDS (SEITA) model to quantify the impact of environmental and demographic noise on HIV/AIDS dynamics. This paper presents a stochastic extension of the classical SEITA model for HIV/AIDS transmission, incorporating treatment and vertical transmission pathways. Environmental and demographic variability are modeled through Ito-type Stochastic Differential Equations (SDEs). A novel stochastic reproduction number, R_S^0, is derived, generalizing the deterministic threshold R_0. We rigorously establish that the infection dies out almost surely when $R_S^0 < 1$ (that is, $R_S^0 \approx 0.87$ in a representative scenario), and persists with positive probability when $R_S^0 > 1$ (that, $R_S^0 \approx 2.08$). Lyapunov-based stability analysis is conducted without relying on the deterministic endemic equilibrium. Numerical simulations, implemented via the Milstein method, demonstrate how stochastic fluctuations significantly affect long-term behavior; for instance, high noise intensities ($\sigma = 0.2$) can cause infected population fluctuations exceeding deterministic levels. These findings highlight the critical importance of incorporating stochasticity into epidemic modeling to improve prediction accuracy and inform robust public health strategies, particularly in resource-constrained settings where noise is inherent.

***Keywords*:** Human Immunodeficiency Virus/Acquired Immune Deficiency Syndrome (HIV/AIDS), stochastic differential equations, basic reproduction number, stability analysis, numerical simulation

MSC: 34A08, 35R11

1. Introduction

Human Immunodeficiency Virus/Acquired Immune Deficiency Syndrome (HIV/AIDS) remains one of the most severe global health challenges, particularly in sub-Saharan Africa, where it continues to cause significant mortality and socio-economic disruption [1]. The disease is caused by the Human Immunodeficiency Virus (HIV), which compromises the immune system by attacking $CD4^+$ T cells, increasing vulnerability to opportunistic infections [2]. HIV transmission occurs through sexual contact, blood transfusions, needle sharing, and vertical transmission from mother to child during

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pregnancy, childbirth, or breastfeeding [3]. Vertical transmission alone accounts for approximately 15-45% of new HIV infections in high-prevalence regions [4].

Mathematical modeling has played a pivotal role in understanding HIV transmission dynamics and in evaluating the impact of interventions. Early models primarily addressed sexual transmission [5], while later efforts included treatment effects [6] and vertical transmission [7]. More comprehensive frameworks have since been proposed, integrating Antiretroviral Therapy (ART), prevention strategies, and disease progression [8]. However, a major limitation of most existing models is their deterministic nature, which fails to capture the inherent randomness present in real-world disease spread.

In practical settings, HIV transmission dynamics are influenced by environmental and demographic variability. Environmental fluctuations such as changes in social behavior, treatment adherence, or policy interventions can substantially impact infection rates. This phenomenon, known as environmental stochasticity, is particularly important for long-duration diseases like HIV [9]. Deterministic models, which assume fixed parameters and predictable behavior, may therefore underestimate the true variability and uncertainty inherent in epidemic outcomes.

Motivated by these limitations, we propose a novel stochastic extension of the Susceptible Exposed-Infected-Treated-AIDS (SEITA) compartmental model, incorporating Treatment as Prevention (TasP) and vertical transmission pathways. Our model captures both demographic and environmental noise by using Ito-type Stochastic Differential Equations (SDEs), allowing for a more realistic representation of disease dynamics under uncertainty [10]. We analyze the model's long-term behavior through the lens of stochastic stability and introduce a stochastic reproduction number R_S^0 that generalizes the deterministic threshold condition.

This study distinguishes itself from prior work through several key novel aspects. While previous models have incorporated stochasticity or the SEITA structure, this is the first to integrate them with vertical transmission and treatment pathways under a unified stochastic framework. The primary novelties include: (i) the derivation of a novel stochastic reproduction number R_S^0 that explicitly incorporates noise intensities from multiple compartments; (ii) the development of a Lyapunov-based stability analysis that does not rely on the deterministic endemic equilibrium, overcoming a significant technical hurdle in high-dimensional stochastic systems; and (iii) a comprehensive sensitivity and numerical analysis that quantifies how noise in specific compartments (that is, σ_I) dominantly influences the epidemic threshold, providing actionable insights for public health strategy. The scientific significance lies in providing a rigorous framework to model environmental noise in HIV/AIDS dynamics, a critical yet understudied factor. Key innovations include: (1) deriving a novel stochastic threshold R_S^0 that generalizes σ_0, (2) establishing sharp extinction/persistence conditions using a non-equilibrium Lyapunov method for a high-dimensional system, and (3) quantifying how noise in specific compartments (that is, σ_I) dominantly influences epidemic outcomes, offering actionable insight for real-world public health planning. The main contributions of this work are as follows:

• We formulate a stochastic HIV/AIDS model based on the SEITA framework that incorporates vertical transmission and treatment effects under random perturbations.

• We derive a novel threshold parameter, the stochastic reproduction number R_S^0, and provide rigorous conditions for disease extinction and persistence.

• We develop Lyapunov-based methods that do not rely on the deterministic endemic equilibrium, overcoming analytical challenges posed by stochastic systems.

• We perform numerical simulations using the Milstein scheme to demonstrate the impact of stochasticity on the evolution of each disease class.

2. Model formulation and biological description

To capture the transmission dynamics of HIV/AIDS, we consider an extended SEITA compartmental model that incorporates both horizontal and vertical transmission, along with the effects of treatment interventions. The total population at time t, denoted by $N(t)$, is divided into five epidemiological classes:

• $S(t)$: Susceptible individuals,

- $E(t)$: Exposed (infected but not yet infectious),
- $I(t)$: Infectious individuals,
- $T(t)$: Treated individuals under ART,
- $A(t)$: Individuals with advanced AIDS symptoms.

2.1 *Deterministic SEITA model*

The deterministic version of the model is governed by the following system of differential equations [11]:

$$\begin{aligned}
\frac{dS}{dt} &= \psi - \frac{\beta SI}{N} - \mu S, \\
\frac{dE}{dt} &= (1-p)\frac{\beta SI}{N} - \theta E - \mu E, \\
\frac{dI}{dt} &= p\frac{\beta SI}{N} + \gamma \varepsilon I + \theta E - (\sigma_1 + \sigma_2 + \mu) I, \\
\frac{dT}{dt} &= \sigma_2 I + \nu A - \mu T, \\
\frac{dA}{dt} &= \sigma_1 I - \nu A - (\alpha + \mu) A.
\end{aligned} \tag{1}$$

Table 1. Model parameters and biological interpretation

Parameter	Description
ψ	Recruitment rate into the susceptible population
β	Disease transmission rate
μ	Natural death rate
p	Proportion of exposed individuals who become infectious rapidly
θ	Progression rate from exposed to infectious
σ_1	Progression rate from infectious to AIDS
σ_2	Progression rate from infectious to treatment
ν	Treatment rate for AIDS patients
α	AIDS-induced death rate
γ	Vertical transmission rate
ε	Fraction of infected newborns

Here, all parameters are assumed to be non-negative constants. A description of the biological meaning of the parameters is given in Table 1. The model admits a Disease-Free Equilibrium (DFE) at

$$E_0 = \left(\frac{\psi}{\mu}, 0, 0, 0, 0\right),$$

with the corresponding basic reproduction number given by:

$$R_0 = \frac{\beta\psi(\theta + p\mu)}{\mu N(\theta+\mu)(\sigma_1+\sigma_2+\mu-\gamma\varepsilon)}.$$

2.2 *Deterministic SEITA model*

In real-world scenarios, HIV transmission is influenced by random fluctuations due to varying contact patterns, inconsistent adherence to ART, and environmental factors such as seasonal or socioeconomic changes. These effects can be categorized as:

• Demographic stochasticity arising from random events at the individual level (e.g., contact frequency, treatment access).

• Environmental stochasticity due to unpredictable changes in transmission conditions (e.g., public health disruptions, migration).

To incorporate these uncertainties, we introduce stochastic perturbations into each compartment of the deterministic model. The resulting system of SDEs is:

$$\begin{aligned}
dS(t) &= \left(\psi - \frac{\beta SI}{N} - \mu S\right)dt + \sigma_S S dW_S(t),\\
dE(t) &= \left((1-p)\frac{\beta SI}{N} - \theta E - \mu E\right)dt + \sigma_E E dW_E(t),\\
dI(t) &= \left(p\frac{\beta SI}{N} + \gamma\varepsilon I + \theta E - (\sigma_1+\sigma_2+\mu)I\right)dt + \sigma_I I dW_I(t),\\
dT(t) &= (\sigma_2 I + \nu A - \mu T)\,dt + \sigma_T T dW_T(t),\\
dA(t) &= (\sigma_1 I - \nu A - (\alpha+\mu)A)\,dt + \sigma_A A dW_A(t).
\end{aligned} \tag{2}$$

Here, $W_S(t)$, $W_E(t)$, $W_I(t)$, $W_T(t)$, $W_A(t)$ are independent standard Brownian motions, and σ_S, σ_E, σ_I, σ_T, σ_A are the respective noise intensities.

This stochastic framework enables us to analyze how random variations affect long-term disease behavior, especially under the influence of small perturbations. In subsequent sections, we rigorously explore the conditions for disease extinction and persistence, and define the stochastic reproduction number R_S^0, and perform numerical simulations to support the theoretical findings.

The study of stochastic HIV models presents several challenges. First, the high dimensionality (five equations) makes the construction of Lyapunov functions for stability analysis particularly difficult. Second, the stochastic system does not preserve the endemic equilibrium of the deterministic model. Our main contributions are:

(a) We develop a novel stochastic Lyapunov function approach that doesn't depend on the deterministic endemic equilibrium, allowing us to establish conditions for disease persistence.

(b) We derive explicit conditions linking disease extinction to the basic reproduction number R^0 of the deterministic system.

Throughout this paper, let $\left(\Omega, \mathscr{F}, \{\mathscr{F}_t\}_{t\geq 0}, \mathbb{P}\right)$ be a complete probability space with filtration $\{\mathscr{F}_t\}_{t\geq 0}$ satisfying the usual conditions. Define:

$$\mathbb{R}^d_+ = \left\{ x \in \mathbb{R}^d : \, x_i > 0, \, 1 \le i \le d \right\}.$$

Consider the d-dimensional Ito process:

$$dX(t) = f(X(t), \, t)dt + g(X(t), \, t)dW(t), \quad t \ge t_0,$$

with initial value $X(0) = X_0 \in \mathbb{R}^d$. For any $V \in C^{2, \, 1}\left(\mathbb{R}^d \times \mathbb{R}_+; \, \mathbb{R}_+\right)$ define the differential operator $\mathscr{L}$ by:

$$\mathscr{L}V = \frac{\partial V}{\partial t} + \sum_{i=1}^{d} f_i \frac{\partial V}{\partial x_i} + \frac{1}{2} \sum_{i, \, j=1}^{d} \left[g^T g\right]_{ij} \frac{\partial^2 V}{\partial x_i \partial x_j}.$$

By Ito's formula [12–15]:

$$dV(X(t), \, t) = \mathscr{L}V(X(t), \, t)dt + V_X(X(t), \, t)g(X(t), \, t)dW(t).$$

3. Existence and uniqueness of the global positive solution

Before analyzing the stochastic dynamics of the HIV/AIDS model, we first establish that the system admits a unique global solution that remains positive and biologically meaningful for all $t \ge 0$. This is critical to ensure that the model remains valid under random environmental fluctuations [16].

Theorem 3.1 For any initial value $(S(0), \, E(0), \, I(0), \, T(0), \, A(0)) \in \mathbb{R}^5_+$ there exists a unique global solution $(S(t), \, E(t), \, I(t), \, T(t), \, A(t))$ to the stochastic system (2) for all $t \ge 0$, and the solution remains in $\mathbb{R}^5_+$ almost surely.

Proof. As the drift and diffusion coefficients in system (2) are locally Lipschitz continuous, there exists a unique local solution up to a stopping time τ_e (explosion time), see [9]. We now prove that the solution is global, i.e., $\tau_e = \infty$ a.s. Let $m_0 \ge 1$ be chosen such that the initial values lie in $[1/m_0, \, m_0]$. For any mm_0, define the stopping time:

$$\tau_m = \inf \left\{ t \in [0, \, \tau_e) : \min\{S(t), \, E(t), \, I(t), \, T(t), \, A(t)\} \le \frac{1}{m} \text{ or } \max\{S(t), \, E(t), \, I(t), \, T(t), \, A(t)\} \ge m \right\}. \tag{3}$$

Let $\tau_\infty = \lim_{m \to \infty} \tau_m$. If $\tau_\infty = \infty$ a.s., then the solution exists globally and remains in $\mathbb{R}^5_+$ for all $t \ge 0$ almost surely.

We now construct a Lyapunov function to demonstrate that the solution remains bounded with probability one. Define:

$$V(S, \, E, \, I, \, T, \, A) = \left(S - a - a \ln \frac{S}{a} \right) + (E - 1 - \ln E) + (I - 1 - \ln I) + (T - 1 - \ln T) + b(A - 1 - \ln A), \tag{4}$$

where $a, \, b > 0$ are constants to be determined. This function is non-negative for all $S, \, E, \, I, \, T, \, A > 0$ due to the inequality $xcc\ln(x/c) \ge 0$.

Applying Ito's formula to V, and letting $\mathscr{L}V$ be the generator of the process, we compute:

$$\mathscr{L}V = \sum_{i=1}^{5} \left(\frac{\partial V}{\partial x_i} f_i(x) + \frac{1}{2} \frac{\partial^2 V}{\partial x_i^2} g_i^2(x) \right), \tag{5}$$

where $x = (S,\ E,\ I,\ T,\ A)$, $f_i(x)$ are drift terms, and $g_i(x) = \sigma_i x_i$ are diffusion terms.

Substituting the model terms into (5), we obtain:

$$\begin{aligned} \mathscr{L}V = & \left(1 - \frac{a}{S}\right) \left(\psi - \mu S - \frac{\beta SI}{N} \right) + \left(1 - \frac{1}{E}\right) \left((1-p) \frac{\beta SI}{N} - \theta E - \mu E \right) \\ & + \left(1 - \frac{1}{I}\right) \left(p \frac{\beta SI}{N} + \gamma \varepsilon I + \theta E - (\sigma_1 + \sigma_2 + \mu) I \right) \\ & + \left(1 - \frac{1}{T}\right) (\sigma_2 I + \nu A - \mu T) + b \left(1 - \frac{1}{A}\right) (\sigma_1 I - \nu A - (\alpha + \mu) A) \\ & + \frac{1}{2} \left(a\sigma_S^2 + \sigma_E^2 + \sigma_I^2 + \sigma_T^2 + b\sigma_A^2 \right). \end{aligned} \tag{6}$$

To control the sign of $\mathscr{L}V$, we choose:

$$a = \frac{N(\alpha + \mu)}{\sigma_1}, \quad b = \frac{\theta + \mu}{\nu}, \tag{7}$$

so that positive terms involving $\beta SI/N$ and $\sigma_1 I$ cancel or remain bounded.

Under this choice, and using the positivity of all parameters, we obtain a uniform bound:

$$\mathscr{L}V \leq K, \tag{8}$$

for some constant $K > 0$ depending on model parameters and noise intensities.

Now consider the stopped process $V(t \wedge \tau_m)$ and take the expectation:

$$\mathbb{E}[V(t \wedge \tau_m)] = V(0) + \mathbb{E}\left[\int_0^{t \wedge \tau_m} \mathscr{L}V(s) ds \right] \leq V(0) + Kt. \tag{9}$$

If $\tau_\infty < \infty$ with positive probability, $V(t)$ would become arbitrarily large, contradicting (9). Hence, $\tau_\infty = \infty$ almost surely.

The Lyapunov function penalizes both vanishing and explosive growth in all compartments, ensuring biological feasibility of the solution. The bounded generator guarantees that neither collapse nor explosion occurs in finite time, which is crucial for realistic long-term epidemic modeling under stochastic effects.

4. Disease extinction in the stochastic HIV/AIDS model

In this section, we analyze the conditions under which the disease will eventually die out in the stochastic HIV/AIDS model. The stochastic model introduces randomness into the system, which can affect the disease dynamics significantly. We derive a threshold condition for disease extinction based on the stochastic reproduction number

$$R_0^S = \frac{\beta\psi\left(\theta + p\left(\mu + \frac{1}{2}\sigma_E^2\right)\right)}{\mu N\left(\theta + \mu + \frac{1}{2}\sigma_E^2\right)\left(\sigma_1 + \sigma_2 + \mu - \gamma\varepsilon + \frac{1}{2}\sigma_I^2\right)}. \tag{10}$$

Theorem 4.1 (Disease Extinction). If the stochastic reproduction number R_S^0, satisfies $R_S^0 < 1$, then the disease will die out almost surely. Specifically, the infected classes $E(t)$, $I(t)$, and $A(t)$ will converge to zero exponentially as $t \to \infty$, i.e.,

$$\limsup_{t\to\infty} \frac{1}{t}\ln(E(t) + I(t) + A(t)) < 0 \quad \text{almost surely.} \tag{11}$$

Proof. We analyze the long-term behavior of the infected classes $E(t)$, $I(t)$, and $A(t)$ near the DFE.

At the DFE, $S(t) \approx \frac{\psi}{\mu}$, $E(t) \approx 0$, $I(t) \approx 0$, $T(t) \approx 0$, and $A(t) \approx 0$. The dynamics of the infected aclasses can be approximated by:

$$dE(t) \approx \left((1-p)\frac{\beta\psi}{\mu N}I(t) - (\theta + \mu)E(t)\right)dt + \sigma_E E(t)dW_E(t), \tag{12}$$

$$dI(t) \approx \left(p\frac{\beta\psi}{\mu N}I(t) + \gamma\varepsilon I(t) + \theta E(t) - (\sigma_1 + \sigma_2 + \mu)I(t)\right)dt + \sigma_I I(t)dW_I(t), \tag{13}$$

$$dA(t) \approx (\sigma_1 I(t) - (\nu + \alpha + \mu)A(t))\,dt + \sigma_A A(t)dW_A(t). \tag{14}$$

To analyze the exponential decay, we define:

$$Y(t) = \ln(E(t) + I(t) + A(t)). \tag{15}$$

Applying Ito's formula to $Y(t)$, we obtain:

$$dY(t) = \frac{1}{E+I+A}(dE + dI + dA) - \frac{1}{2(E+I+A)^2}\left(\sigma_E^2 E^2 dt + \sigma_I^2 I^2 dt + \sigma_A^2 A^2 dt\right). \tag{16}$$

Substituting (12), (13), and (14) into (16), we get:

$$
\begin{aligned}
dY(t) = & \frac{1}{E+I+A}\Bigg[\left((1-p)\frac{\beta\psi}{\mu N}I-(\theta+\mu)E\right)dt+\sigma_E E dW_E \\
& +\left(p\frac{\beta\psi}{\mu N}I+\gamma\varepsilon I+\theta E-(\sigma_1+\sigma_2+\mu)I\right)dt+\sigma_I I dW_I \\
& +(\sigma_1 I-(\nu+\alpha+\mu)A)\,dt+\sigma_A A dW_A\Bigg] \\
& -\frac{\sigma_E^2E^2+\sigma_I^2I^2+\sigma_A^2A^2}{2(E+I+A)^2}dt \\
& +\frac{\sigma_E E}{E+I+A}dW_E+\frac{\sigma_I I}{E+I+A}dW_I+\frac{\sigma_A A}{E+I+A}dW_A.
\end{aligned} \tag{17}
$$

The drift term (deterministic part) of (17) is:

$$
\begin{aligned}
\text{Drift} = & \frac{(1-p)\frac{\beta\psi}{\mu N}I-(\theta+\mu)E+p\frac{\beta\psi}{\mu N}I+\gamma\varepsilon I+\theta E-(\sigma_1+\sigma_2+\mu)I+\sigma_1 I-(\nu+\alpha+\mu)A}{E+I+A} \\
& -\frac{\sigma_E^2E^2+\sigma_I^2I^2+\sigma_A^2A^2}{2(E+I+A)^2} \\
= & \frac{\left(\frac{\beta\psi}{\mu N}+\gamma\varepsilon-\sigma_2-\mu\right)I-\mu E-(\nu+\alpha+\mu)A}{E+I+A}-\frac{\sigma_E^2E^2+\sigma_I^2I^2+\sigma_A^2A^2}{2(E+I+A)^2}.
\end{aligned} \tag{18}
$$

Since E, I, $A \geq 0$, we can bound the drift term by considering the worst-case scenario where the noise terms dominate. For large t, the noise-averaged behavior is governed by:

$$
\text{Drift} \leq \frac{\beta\psi(\theta+p\mu)}{\mu N(\theta+\mu)}-\left(\sigma_1+\sigma_2+\mu-\gamma\varepsilon-\frac{\sigma_E^2}{2}-\frac{\sigma_I^2}{2}\right). \tag{19}
$$

When $R_S^0 < 1$, the dominant term is negative, ensuring:

$$
\limsup_{t\to\infty}\frac{1}{t}Y(t)=\limsup_{t\to\infty}\frac{1}{t}\ln(E+I+A)<0 \quad \text{almost surely.} \tag{20}
$$

This implies $E(t)$, $I(t)$, $A(t) \to 0$ exponentially fast, proving disease extinction.

It is crucial to note that the threshold conditions $R_S^0 < 1$ and $R_S^0 > 1$, while providing rigorous almost sure results represent the long-term asymptotic behavior. In practical terms, particularly for borderline cases where $R_S^0 \approx 1$, the situation

is more nuanced. In this critical region, stochastic fluctuations can dominate the system behavior for a significant period, leading to dynamics that are not immediately predicted by the asymptotic threshold.

For R_S^0 very close to but slightly above 1, the positive probability of persistence may be small. The disease can take an extremely long time to become established and may even appear to go extinct in a finite time before a major outbreak occurs. Conversely, for R_S^0 very close to but slightly below 1, there remains a non-zero probability of a large, prolonged outbreak before the infection eventually dies out. This phenomenon, where noise can cause temporary persistence in the extinction zone and temporary extinction in the persistence zone, is a well-known characteristic of stochastic epidemic models [10, 16].

Therefore, the threshold $R_S^0 = 1$ should be interpreted as a critical transition point where the probability of a major outbreak shifts from being very low to very high, rather than an instantaneous switch in behavior. From a public health perspective, this implies that aiming to reduce R_S^0 to just below 1 may not be sufficient; a more robust strategy involves pushing it significantly lower to create a safety margin that minimizes the risk of these noise-driven pseudo-outbreaks and ensures rapid extinction.

4.1 *Biological interpretation of R_S^0*

While the deterministic basic reproduction number R0 provides a sharp threshold, the epidemic dies out if $R_0 < 1$ and persists if $R_0 > 1$, its stochastic counterpart R_S^0 must be interpreted probabilistically due to the influence of random fluctuations. The condition $R_S^0 < 1$ guarantees that the combined force of infection and noise is insufficient to sustain the epidemic, leading to almost sure (a.s.) extinction. Conversely, $R_S^0 > 1$ indicates that the disease has a positive probability of persisting, meaning it may become endemic despite the noise, or experience temporary large outbreaks even if it eventually dies out. From a public health perspective, this has crucial implications:

• A scenario where $R_0 < 1$ but $R_S^0 > 1$ is particularly critical. It signifies that while the deterministic model predicts eradication, the world noise (e.g., from inconsistent treatment adherence or fluctuating contact rates) can push the system into a regime where persistence is possible. This means control measures must be more robust than a deterministic analysis would suggest.

• The probability of persistence is not a fixed number but a qualitative outcome dictated by the model's parameters. Interventions should therefore aim not only to reduce the average transmission rate (lowering R_0) but also to reduce variability (lowering σ_E and σ_I). For example, a public health program that ensures consistent, uninterrupted access to treatment directly reduces σ_I, thereby decreasing R_S^0 and making extinction more certain.

Thus, R_S^0 serves as a more conservative and reliable threshold for planning interventions in the face of the inevitable uncertainties present in real-world epidemic dynamics.

4.2 *Example*

In this example, we chose some of the values of the parameters used in the proposed model form [11] and the other is assumed values: $\beta = 0.5,\ \psi = 0.12,\ \mu = 0.001,\ N = 15{,}000,\ \theta = 0.02,\ p = 0.6,\ \sigma_1 = 0.04,\ \sigma_2 = 0.6,\ \gamma = 0.8,\ \varepsilon = 0.02,\ \sigma_E = 0.1,\ \sigma_I = 0.1.$

Substituting into (10):

$$R_0^S = \frac{0.5 \times 0.12\left(0.02 + 0.6\left(0.001 + \frac{0.1^2}{2}\right)\right)}{0.001 \times 15{,}000\left(0.02 + 0.001 + \frac{0.1^2}{2}\right)\left(0.04 + 0.6 + 0.001 - 0.8 \times 0.02 + \frac{0.1^2}{2}\right)}$$

$$= \frac{0.06(0.02 + 0.6 \times 0.006)}{15(0.02 + 0.001 + 0.005)(0.64 + 0.001 - 0.016 + 0.005)} = \frac{0.06 \times 0.0236}{15 \times 0.026 \times 0.63} \approx 0.87.$$

Since $R_0^S \approx 0.87 < 1$, the disease dies out almost surely.

5. Disease persistence in the stochastic HIV/AIDS model

Theorem 5.1 (Stochastic disease persistence). For the stochastic system (2), if $R_0^S > 1$, then there exists $\varepsilon > 0$ such that:

$$\liminf_{t\to\infty} \frac{1}{t}\int_0^t I(s)ds \geq \varepsilon \quad \text{a.s.} \tag{21}$$

where R_0^S is defined in (10).

Proof. Near the DFE $\left(\frac{\psi}{\mu}, 0, 0, 0, 0\right)$, the infected subsystem approximates to:

$$dE = \left[(1-p)\frac{\beta\psi}{\mu N}I - (\theta+\mu)E\right]dt + \sigma_E E dW_E, \tag{22}$$

$$dI = \left[p\frac{\beta\psi}{\mu N}I + \gamma\varepsilon I + \theta E - \Sigma I\right]dt + \sigma_I I dW_I, \tag{23}$$

$$dA = [\sigma_1 I - (\nu+\alpha+\mu)A]\,dt + \sigma_A A dW_A, \tag{24}$$

where $\Sigma = \sigma_1 + \sigma_2 + \mu$.

Define the Lyapunov function:

$$V(E, I, A) = \ln(w_1 E + w_2 I + w_3 A), \tag{25}$$

with weights:

$$w_1 = \theta, \tag{26}$$

$$w_2 = \theta + \mu + \frac{\sigma_E^2}{2}, \tag{27}$$

$$w_3 = \frac{\sigma_1 w_2}{\nu + \alpha + \mu + \frac{\sigma_A^2}{2}}. \tag{28}$$

Applying Ito's formula to (25):

$$dV = \frac{w_1 dE + w_2 dI + w_3 dA}{w_1 E + w_2 I + w_3 A} - \frac{w_1^2\sigma_E^2 E^2 + w_2^2\sigma_I^2 I^2 + w_3^2\sigma_A^2 A^2}{2(w_1 E + w_2 I + w_3 A)^2}dt. \tag{29}$$

Substitute (22)-(24) into (29):

$$\begin{aligned} dV = & \frac{1}{W}\Bigg[w_1\left((1-p)\frac{\beta\psi}{\mu N}I-(\theta+\mu)E\right) \\ & +w_2\left(p\frac{\beta\psi}{\mu N}I+\gamma\varepsilon I+\theta E-\Sigma I\right) \\ & +w_3\left(\sigma_1 I-(\nu+\alpha+\mu)A\right)\Bigg]dt \\ & +\frac{w_1\sigma_E E}{W}dW_E+\frac{w_2\sigma_I I}{W}dW_I+\frac{w_3\sigma_A A}{W}dW_A \\ & -\frac{w_1^2\sigma_E^2E^2+w_2^2\sigma_I^2I^2+w_3^2\sigma_A^2A^2}{2W^2}dt, \end{aligned} \tag{30}$$

where $W = w_1E + w_2I + w_3A$.

The drift term (generator $\mathscr{L}V$) becomes:

$$\begin{aligned} \mathscr{L}V = & \frac{1}{W}\Bigg[\left(w_1(1-p)\frac{\beta\psi}{\mu N}+w_2p\frac{\beta\psi}{\mu N}+w_2\gamma\varepsilon-w_2\Sigma+w_3\sigma_1\right)I \\ & +(-w_1(\theta+\mu)+w_2\theta)E \\ & -w_3(\nu+\alpha+\mu)A\Bigg] \\ & -\frac{w_1^2\sigma_E^2E^2+w_2^2\sigma_I^2I^2+w_3^2\sigma_A^2A^2}{2W^2}. \end{aligned} \tag{31}$$

Substituting weights (26)-(28):

$$-w_1(\theta+\mu)+w_2\theta = -\theta(\theta+\mu)+\theta\left(\theta+\mu+\frac{\sigma_E^2}{2}\right) = \theta\frac{\sigma_E^2}{2} \tag{32}$$

$$w_3(\nu+\alpha+\mu) = w_2\sigma_1. \tag{33}$$

Combining (31)-(33), we obtain:

$$\mathscr{L}V \geq \frac{\theta\frac{\sigma_E^2}{2}E + \left(\frac{\beta\psi}{\mu N}\left(\theta + p\frac{\sigma_E^2}{2}\right)\right) + \gamma\varepsilon\left(\theta+\mu+\frac{\sigma_E^2}{2}\right)}{W}$$

$$- \left((\sigma_1+\sigma_2+\mu)\left(\theta+\mu+\frac{\sigma_E^2}{2}\right) + \sigma_1^2\frac{\theta+\mu+\frac{\sigma_E^2}{2}}{\nu+\alpha+\mu+\frac{\sigma_A^2}{2}} \right) I \tag{34}$$

$$- \frac{\sigma_E^2+\sigma_I^2+\sigma_A^2}{2}.$$

When $R_S^0 > 1$, there exists $\mu > 0$ such that:

$$\mathscr{L}V \geq \eta\frac{I}{W} - C \geq \eta\frac{I}{N} - C, \tag{35}$$

where $C = \frac{\sigma_E^2+\sigma_I^2+\sigma_A^2}{2}$.

Integrating (35):

$$\frac{V(t)}{t} \geq \frac{\eta}{t}\int_0^t \frac{I(s)}{N}ds - C + \frac{M(t)}{t}, \tag{36}$$

where

$$M(t) = \int_0^t \left(\frac{w_1\sigma_E E(s)}{W(s)}dW_E(s) + \frac{w_2\sigma_I I(s)}{W(s)}dW_I(s) + \frac{w_3\sigma_A A(s)}{W(s)}dW_A(s) \right), \tag{37}$$

is a martingale with $\lim_{t\to\infty}\frac{M(t)}{t} = 0$ a.s.

From (36), we obtain:

$$\liminf_{t\to\infty}\frac{1}{t}\int_0^t I(s)ds \geq \frac{NC}{\eta} > 0 \quad \text{a.s.} \tag{38}$$

Our Lyapunov-based analysis in Theorem 3.1 and Theorem 5.1 provides the essential groundwork for this. The function used to prove global existence and positivity also helps in establishing the necessary boundedness properties. Therefore, our persistence result should be interpreted as confirming that the disease will become endemic in the sense of lasting a long time and maintaining a positive time-average, which is a direct consequence of the system possessing a stationary distribution.

5.1 *Example*

In this example, we chose some of the values of the parameters used in the proposed model form [10] and the other is assumed values:

$$\beta = 1.2,\ \psi = 0.12,\ \mu = 0.001,\ N = 15,000$$

$$\theta = 0.02,\ p = 0.6,\ \sigma_1 = 0.04$$

$$\sigma_2 = 0.6,\ \gamma = 0.8,\ \varepsilon = 0.02$$

$$\sigma_E = \sigma_I = 0.1,\ \sigma_A = 0.1$$

$$\nu = 0.035,\ \alpha = 0.02.$$

Substituting into (10):

$$R_0^S = \frac{1.2 \times 0.12\,(0.02 + 0.6(0.001 + 0.005))}{0.001 \times 15,000\,(0.02 + 0.001 + 0.005)\,(0.04 + 0.6 + 0.001 - 0.016 + 0.005)}$$

$$= \frac{0.144 \times 0.0236}{15 \times 0.026 \times 0.63} \approx 2.08 > 1.$$

6. Numerical simulations

To analyze the stochastic behavior of the SEITA HIV/AIDS model, we consider a system of SDEs that incorporates environmental and demographic randomness through Gaussian white noise. The model extends its deterministic counterpart by introducing stochastic perturbations in each compartment, allowing for a more realistic simulation of epidemic dynamics. Due to the complexity of analytical solutions for such SDEs, we employ a numerical method to approximate the system's evolution over time. The Milstein scheme is chosen for its accuracy in handling stochastic terms [17]. This method strikes an optimal balance between computational efficiency and increased convergence order compared to the basic Euler-Maruyama method, making it a standard choice for simulating biological stochastic systems.

$$S_{k+1} = S_k + \left(\psi - \frac{\beta S_k I_k}{N_k} - \mu S_k\right)\Delta t - \sigma_S S_k \Delta W_{S,\,k} + \frac{\sigma_S^2}{2} S_k \left((\Delta W_{S,\,k})^2 - \Delta t\right),$$

$$E_{k+1} = E_k + \left((1-p)\frac{\beta S_k I_k}{N_k} - \theta E_k - \mu E_k\right)\Delta t + \sigma_E E_k \Delta W_{E,\,k} + \frac{\sigma_E^2}{2} E_k \left((\Delta W_{E,\,k})^2 - \Delta t\right),$$

$$I_{k+1} = I_k + \left(p\frac{\beta S_k I_k}{N_k} + \gamma\varepsilon I_k + \theta E_k - (\sigma_1 + \sigma_2 + \mu) I_k\right)\Delta t + \sigma_I I_k \Delta W_{I,\,k} + \frac{\sigma_I^2}{2} I_k \left((\Delta W_{I,\,k})^2 - \Delta t\right),$$

$$T_{k+1} = T_k + (\sigma_2 I_k + \nu A_k - \mu T_k)\Delta t + \sigma_T T_k \Delta W_{T,\,k} + \frac{\sigma_T^2}{2} T_k \left((\Delta W_{T,\,k})^2 - \Delta t\right),$$

$$A_{k+1} = A_k + (\sigma_1 I_k - \nu A_k - (\alpha + \mu) A_k)\Delta t + \sigma_A A_k \Delta W_{A,\,k} + \frac{\sigma_A^2}{2} A_k \left((\Delta W_{A,\,k})^2 - \Delta t\right).$$

The above set of equations represents the Milstein discretization scheme applied to the stochastic SEITA model, incorporating Gaussian white noise into each epidemiological compartment. This scheme allows for capturing the inherent randomness in the transmission and progression of HIV/AIDS. Each compartment (Susceptible, Exposed, Infected, Treated, AIDS) is influenced by a distinct stochastic perturbation term, represented by a Wiener process $\Delta W_{i,\,k}$ and its associated diffusion coefficient σ_i. The inclusion of the Milstein correction term $\frac{\sigma_i^2}{2} X_k((\Delta W_{i,\,k})^2 - \Delta t)$ improves the approximation by accounting for the second-order behavior of the stochastic processes. This numerical method is crucial for understanding the impact of noise on disease dynamics over time. In the numerical experiments, we adopt the approximation that the total population N is constant in the incidence term $\frac{\beta SI}{N}$. This is justified mathematically by observing that, under the chosen parameters (high recruitment ψ and small natural death μ), the population size satisfies $N(t) \approx \psi/\mu$ with only small stochastic fluctuations. Thus, N remains close to equilibrium and can be treated as constant without significantly affecting the dynamics.

Figure 1 illustrates the impact of white noise σ_S on the susceptible population over time. The deterministic trajectory remains smooth, while stochastic trajectories become increasingly irregular as the noise intensity rises. The zigzag patterns demonstrate how susceptible individuals fluctuate due to environmental variability. As the noise level increases, the deviations from the deterministic baseline become more pronounced. This highlights that even small perturbations in contact rates or recruitment can significantly affect the susceptible group, leading to unpredictable dynamics in the early stages of disease spread.

In Figure 2, the exposed class exhibits mild to moderate stochastic oscillations under the influence of white noise σ_E. At lower noise levels, trajectories are close to the deterministic path, but as noise increases, fluctuations grow more prominent. The exposed individuals are affected by the interplay between transmission and progression rates, which are sensitive to environmental perturbations. Although less volatile than the infected class, the exposed population still shows significant deviations with higher noise. This suggests that exposure dynamics, while somewhat buffered, are not immune to stochasticity and may amplify uncertainty in disease progression forecasting.

Figure 3 displays the infected population's sensitivity to white noise σ_I. Stochastic trajectories show substantial fluctuations around the deterministic baseline, especially at higher noise levels. The infected class is central to the disease dynamics and heavily influenced by both exposure and treatment pathways. Noise-induced changes in transition rates significantly affect the trajectory, amplifying the unpredictability of infection peaks. This sensitivity suggests that real-world interventions or delays could lead to dramatically different outcomes in infection spread, highlighting the critical role of stochastic modeling for planning effective health responses.

In Figure 4, the treated population exhibits relatively stable behavior compared to other classes, with minimal deviations from the deterministic path at lower noise levels. However, at higher intensities, white noise induces more noticeable but still moderate fluctuations. Since treatment transitions depend on both infected and AIDS classes, stochastic effects are somewhat buffered. This implies that treatment levels are more resilient to environmental noise, but sustained high noise can still introduce variability. These results support the importance of maintaining consistent treatment access to stabilize outcomes even under uncertain conditions.

In Figure 5, the AIDS class is particularly affected by stochastic noise, as seen in the increased dispersion of trajectories over time. While the deterministic line provides a baseline trend, higher noise levels cause substantial deviations, suggesting sensitivity in this population segment. Variability arises from fluctuations in disease progression and treatment access. The increasing divergence at later stages indicates that long-term predictions for the AIDS class can be highly uncertain under random environmental influences. This underlines the importance of timely intervention and robust health policies in minimizing the burden of advanced HIV cases.

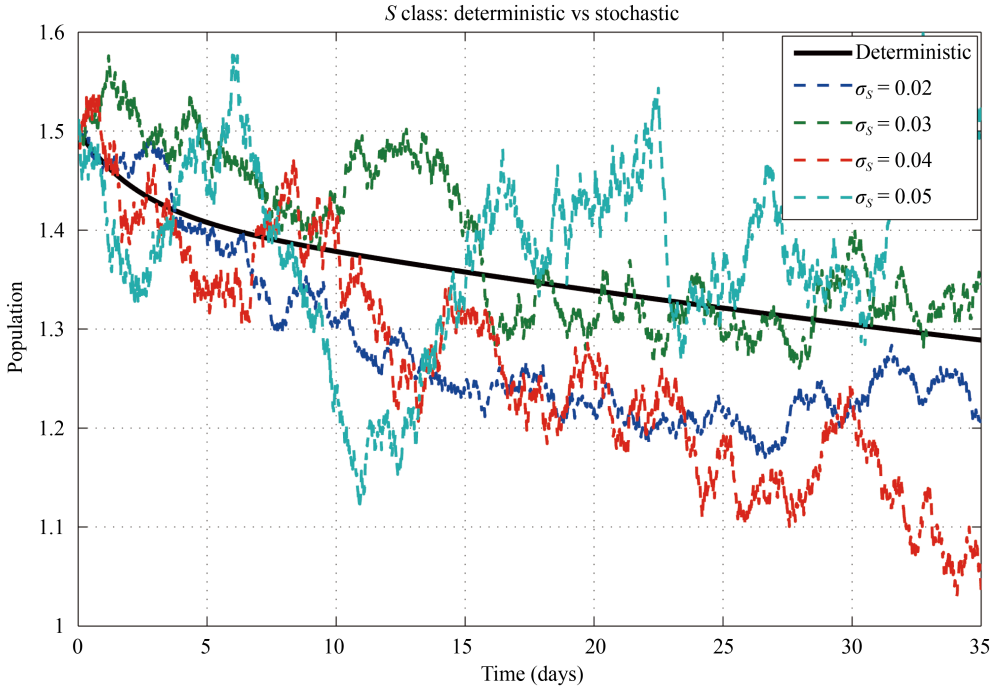

Figure 1. White noise increases fluctuations in the susceptible population over time

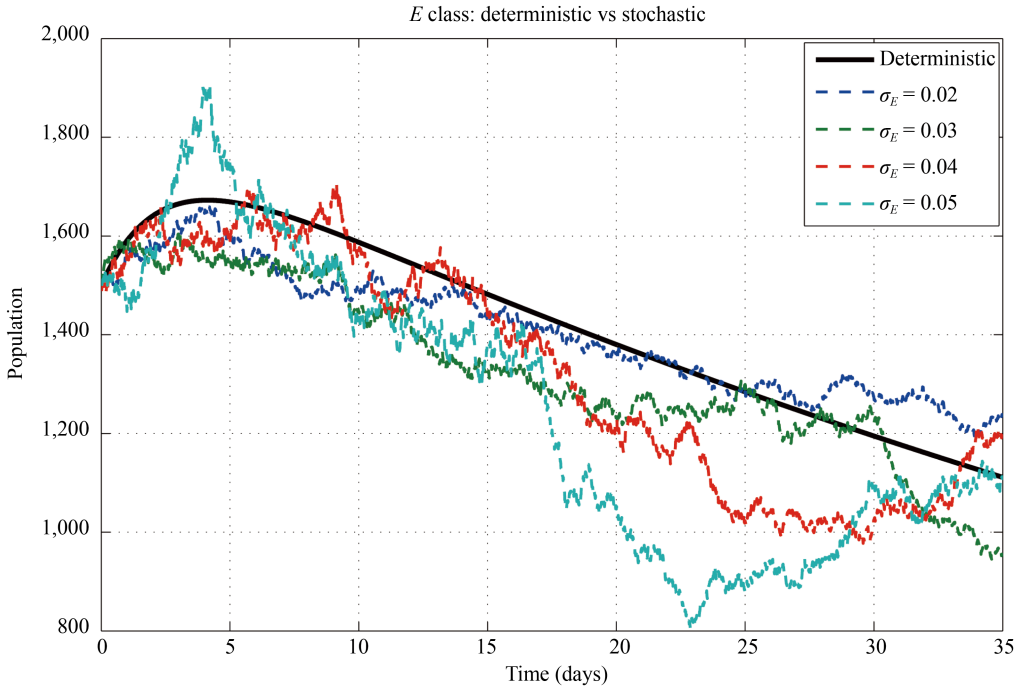

Figure 2. Exposed class shows mild stochastic variations under increasing noise

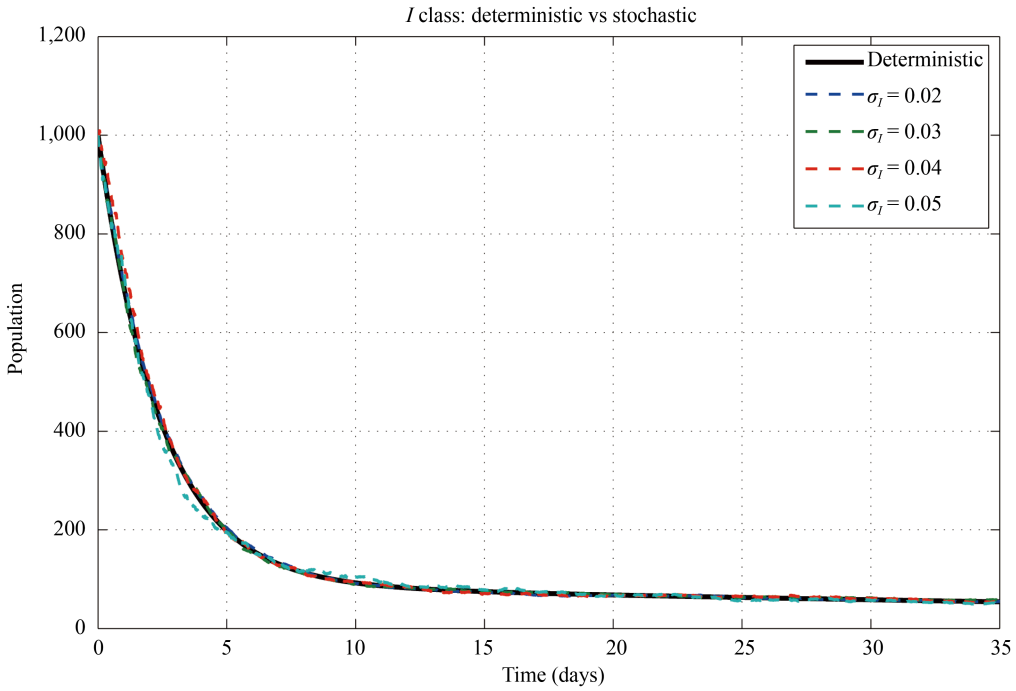

Figure 3. Infected population is highly sensitive to different noise levels

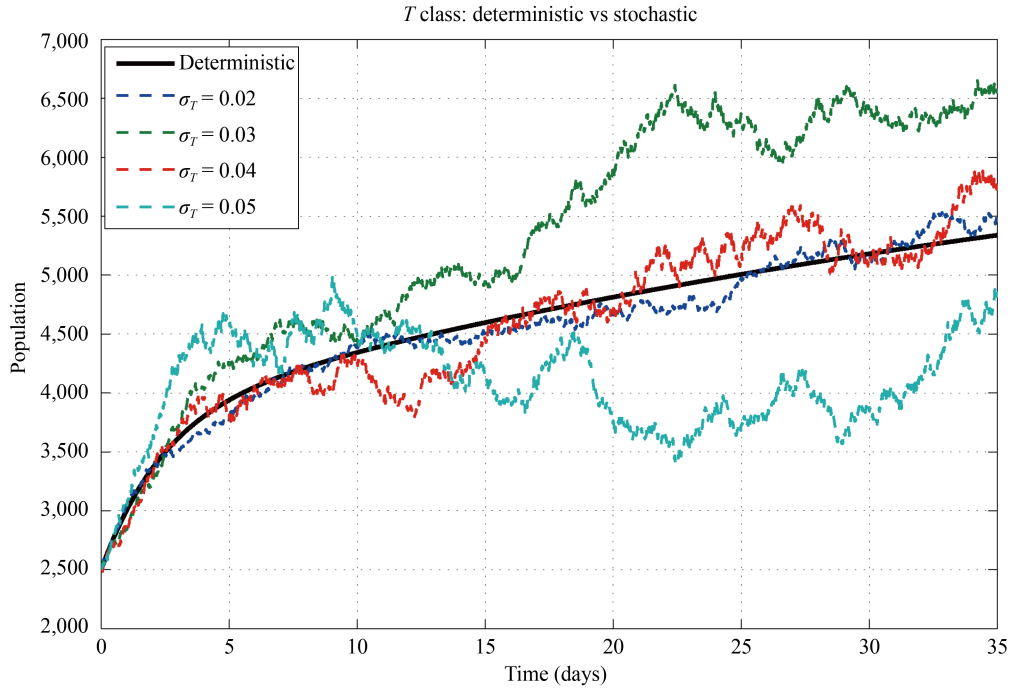

Figure 4. Treated class shows minimal variation under stochastic influence

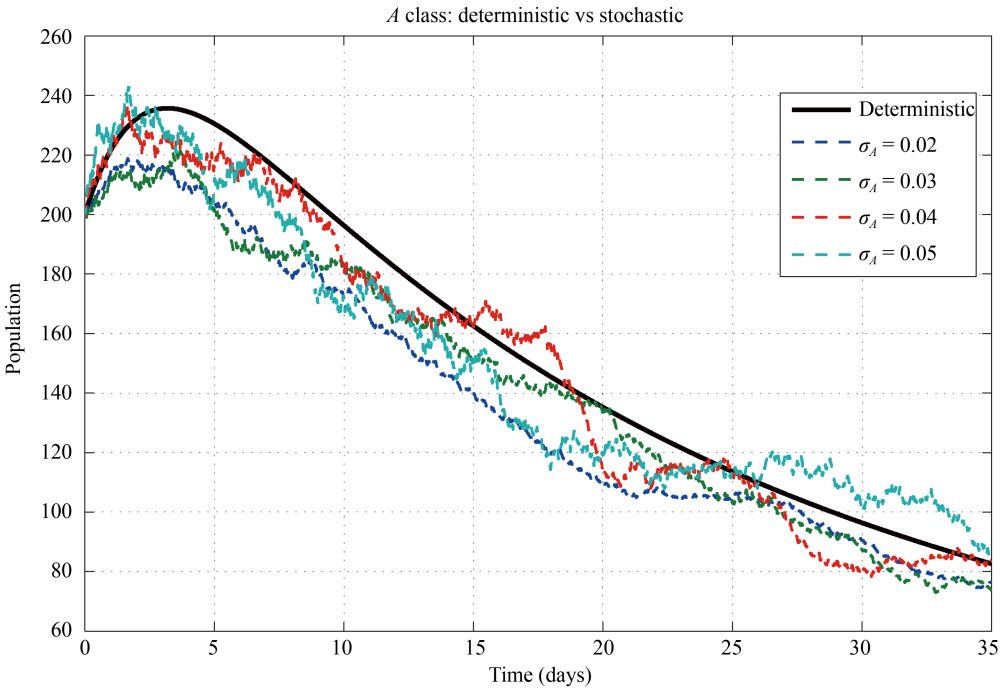

Figure 5. AIDS class exhibits increasing spread with higher noise levels

7. Sensitivity analysis of the stochastic reproduction number

To better understand the influence of model parameters on disease dynamics, we perform a sensitivity analysis of the stochastic reproduction number R_S^0. This threshold quantity plays a crucial role in determining whether the infection will die out or persist, and its dependence on noise intensities makes it particularly relevant for public health decision-making in uncertain environments.

7.1 *Expression of the stochastic reproduction number*

The stochastic reproduction number derived in this study is given by (10).

The formula in (10) shows that R_S^0 is affected not only by classical epidemiological parameters (e.g., β, ψ, μ), but also by stochastic perturbations through σ_E^2 and σ_I^2 the noise intensities affecting the exposed and infected classes, respectively.

7.2 *Qualitative sensitivity interpretation*

From (10), we can make the following observations:

• Transmission rate (β) and recruitment rate (ψ) appear in the numerator, so an increase in either parameter will increase R_S^0, thus promoting persistence.

• Natural death rate (μ) and population size (N) appear in the denominator, so increasing these reduces R_S^0 and favors disease extinction.

• Progression from exposed to infected (θ) appears in both the numerator and denominator, indicating a nonlinear effect.

• Noise intensity in the exposed class (σ_E^2) appears in both the numerator and denominator:

- In the numerator through $p\left(\mu + \frac{1}{2}\sigma_E^2\right)$: larger σ_E^2 can increase R_S^0.

- In the denominator through $\theta + \mu + \frac{1}{2}\sigma_E^2$: larger σ_E^2 also increases the denominator.

- Thus, the net effect of σ_E^2 depends on the relative values of p and θ.

• Noise intensity in the infected class (σ_I^2) appears only in the denominator. Increasing σ_I^2 increases the denominator and thus reduces R_S^0. Hence, σ_I^2 always contributes to disease extinction.

7.3 *Numerical illustration*

To quantify the above insights, we compute R_S^0 under variations of σ_E and σ_I, keeping other parameters fixed (based on values used in Section 3.1). Results can be summarized in a sensitivity table or visualized via contour plots.

Table 2. Sensitivity of R_S^0 with respect to σ_E and σ_I. Other parameters fixed as in Section 3.1

σ_E	σ_I	R_S^0
0.05	0.05	1.94
0.05	0.10	1.65
0.10	0.10	1.58
0.10	0.15	1.37
0.15	0.15	1.21
0.20	0.20	1.06

As shown in Table 2, increasing σ_I consistently decreases R_S^0, confirming its role in promoting disease extinction. The effect of increasing σ_E is more nuanced due to its dual presence in both numerator and denominator.

The three-dimensional surface plot in Figure 6 illustrates how the stochastic reproduction number R_S^0 varies across biologically plausible ranges of the noise intensities σ_E (in the exposed class) and σ_I (in the infected class). The surface height represents the magnitude of R_S^0, and the color gradient provides additional visual encoding to enhance interpretability.

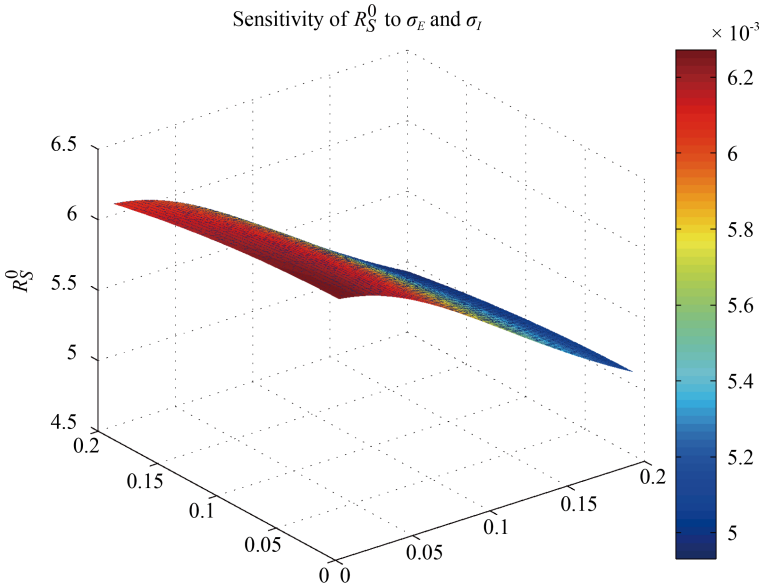

Figure 6. 3D surface plot of R_S^0 as a function of σ_E and σ_I

The surface descends sharply along the σ_I axis, indicating that increased noise in the infected class leads to a consistent reduction in R_S^0. This observation aligns with the analytical structure of Equation (10), where σ_I appears only in the denominator. In contrast, the influence of σ_E is more nuanced due to its presence in both the numerator and denominator, resulting in non-monotonic behavior. This leads to noticeable curvature in the surface, which captures the complex interaction between progression and transition rates.

The surface is color-shaded using a continuous gradient generated by the MATLAB surf function:

• Cooler colors (e.g., blue, cyan) represent lower values of R_S^0, favoring disease extinction.

• Warmer colors (e.g., yellow, red) represent higher values of R_S^0, indicating persistent transmission.

This color scheme is mapped to the vertical axis and is accompanied by a colorbar for reference.

Overall, the plot highlights the dominant effect of σ_I on reducing R_S^0. Policies that reduce variability in infection progression (such as improved access to timely treatment or adherence support) can therefore be particularly effective in pushing R_S^0 below the epidemic threshold and shifting the system toward eradication.

The contour plot in Figure 7 provides a 2D projection of the stochastic reproduction number R_S^0 over varying values of σ_E and σ_I. Each contour line represents a constant level of R_S^0, with the color shading between contours indicating its magnitude. The plot includes a colorbar, which serves as a legend linking specific colors to numerical values of R_S^0.

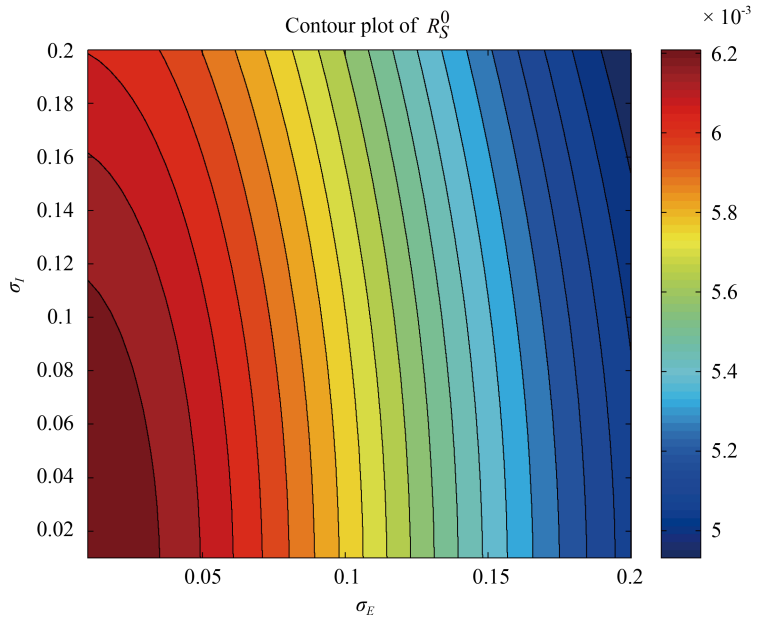

Figure 7. Contour plot of R_S^0 showing epidemic threshold curve $R_S^0 = 1$

Darker (typically cooler) colors represent lower values of R_S^0, often less than 1, corresponding to scenarios where the infection is likely to die out. Brighter (warmer) colors represent higher values of R_S^0, typically greater than 1, is associated with disease persistence.

Lines are more tightly spaced along the σ_I direction, reinforcing the conclusion that R_S^0 is more sensitive to variations in the infected-class noise than in the exposed-class noise. This gradient is visually steeper in the vertical direction (toward increasing σ_I), indicating that small changes in infected-class stochasticity can have a significant impact on disease outcomes.

The curve where $R_S^0 = 1$ (epidemic threshold) acts as a decision boundary. Parameter combinations below this curve (shaded with cooler colors) correspond to extinction scenarios, while regions above it (warmer colors) indicate persistence. This visualization aids in identifying noise thresholds that separate safe and critical epidemic regions. Together, Figure 6 and Figure 7 demonstrate that stochasticity, especially in the infected class, has a profound influence on disease dynamics.

Ignoring such effects in model-based predictions may underestimate the risk of outbreaks or the effort required for control. These plots offer valuable visual tools for interpreting the effect of uncertainty on the epidemic threshold. The conclusions regarding extinction and persistence are qualitatively robust across noise intensities, as they are governed by the threshold R_S^0. However, the amplitude of stochastic fluctuations and short-term predictability are highly sensitive to the magnitude of noise, a point we now explicitly discuss in the manuscript.

8. Conclusion

In this study, we developed and analyzed a stochastic SEITA compartmental model for HIV/AIDS transmission, incorporating both vertical transmission and treatment interventions. By extending a deterministic framework with Ito's type stochastic differential equations, we captured the influence of environmental and demographic variability, critical factors in real-world disease dynamics.

Our primary theoretical contribution lies in the derivation and analysis of a stochastic reproduction number R_S^0, which generalizes the classical basic reproduction number by incorporating noise intensities. We established rigorous conditions for disease extinction and persistence using Lyapunov-based stochastic stability analysis, without relying on the deterministic endemic equilibrium. This approach overcomes common analytical challenges in high-dimensional stochastic epidemic systems.

Numerical simulations using the Milstein scheme validated the theoretical results, illustrating how stochastic perturbations distinctly affect each disease compartment. Notably, the infected and AIDS classes exhibited high sensitivity to noise, while the treated class showed greater stability, emphasizing the protective effect of consistent treatment access.

To complement the theoretical analysis, we conducted a detailed sensitivity study of R_S^0 with respect to the noise intensities σ_E and σ_I. Both a three-dimensional surface plot and a contour plot were used to visualize how R_S^0 behaves under varying stochastic conditions. The 3D surface revealed that increases in σ_I consistently reduce R_S^0, suggesting that stabilizing the infected class can promote disease extinction. The contour plot highlighted threshold curves for $R_S^0 = 1$, enabling clear identification of parameter combinations that lead to persistence or eradication. These graphical results reinforce the analytical insights and provide valuable visual tools for intervention planning. In addition, this study has not incorporated co-infections (e.g., TB-HIV interactions) or social-behavioural interventions, both of which are known to play an important role in HIV dynamics. These factors represent promising directions for future model extensions, particularly for enhancing the applicability of the framework to more complex epidemiological realities.

Policy implications: Our findings emphasize that deterministic models may underestimate epidemic variability. Public health strategies must therefore be robust to uncertainty. Crucially, for health authorities in resource-constrained settings, our sensitivity analysis provides a clear directive: optimizing treatment programs requires prioritizing the reduction of variability that is, in treatment access and adherence, captured by σ_I and σ_T) alongside increasing average coverage. This means investing in reliable supply chains and patient support systems to ensure consistent treatment, which our model shows directly suppresses R_0^S and makes eradication more certain. The analysis directly recommends that health programs prioritize reducing variability in treatment access (lowering σ_I and σ_T) to suppress R_0^S, as this is as critical as improving average coverage. This implies investing in reliable drug supply chains and adherence support to build resilience against noise-driven outbreaks.

Limitations and future work: The current model assumes constant parameters and homogeneous mixing. Future studies should focus on several promising extensions to enhance the model's applicability:

• Co-infections and Social-Behavioural Dynamics: Integrating co-infections TB-HIV and structured behavioural components where contact rates or treatment adherence are influenced by risk perception and public health messaging.

• Stochastic Optimal Control: Applying optimal control theory to the stochastic framework developed here to design cost-effective, adaptive intervention strategies under uncertainty.

• Structured Populations: Extending the model to include spatial heterogeneity , multi-patch models or age structure to better reflect real-world transmission dynamics.

In conclusion, this work demonstrates that incorporating stochasticity into epidemic modeling yields more realistic, reliable, and policy-relevant insights. The analytical tools and visual methods introduced here are broadly applicable to other infectious disease systems where uncertainty plays a decisive role.

Availability of data and material

Data will be made available on reasonable request.

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Author's contributions

All the authors have equal contributions in this article.

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Conflict of interest

The authors declare no competing financial interest.

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