# A Stochastic Differential Equation Model for HIV/AIDS Transmission Dynamics in Heterosexual Populations

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**Abstract**

A mathematical HIV/AIDS transmission model for heterosexual populations integrates environmental noise to analyze population variations. The model uses three distinct compartments for the population: susceptible $S(t)$, infected $I(t)$, and AIDS $A(t)$. The mathematical well-posedness of the model was confirmed through positivity and boundedness tests which led to the determination of both deterministic $R_0$ and stochastic $R_0^S$ reproduction numbers using the method of next generation matrix approach and the results showed how environmental noise influences transmission. The system demonstrates local asymptotic stability at the disease-free equilibrium when $R_0^S < 1$ with noise intensities significantly influencing the stability conditions and an endemic equilibrium occurs when $R_0^S > 1$ characterized by fluctuations around deterministic predictions. The simulation results demonstrate substantial fluctuations in disease prevalence when noise levels reach $\sigma_1 = 0.1$, $\sigma_2 = 0.15$ and $\sigma_3 = 0.1$. The model extended to a discrete-state continuous-time Markov chain framework, enabling analysis of transition probabilities and stationary distributions under noise effects. Numerical simulations using the Euler-Maruyama method demonstrated that noise introduces substantial variability in compartmental trajectories, with the infected population exhibiting particularly pronounced fluctuations due to its higher noise sensitivity. Multiple stochastic realizations revealed significant outcome diversity despite identical parameters, emphasizing the importance of incorporating environmental variability in HIV/AIDS disease forecasting. The Markov chain extension facilitated analysis of transition probabilities between susceptible, infected, and AIDS compartments, enabling policymakers to track disease progression dynamics. This approach informs long-term treatment strategies, particularly in ensuring sustainable antiretroviral therapy resource allocation as infected individuals advance to AIDS

Key words: HIV/AIDS Stochastic Model, Heterosexual Population

## 1 Introduction

The global HIV/AIDS pandemic remains one of the most significant public health challenges of our time, with profound implications for both developed and developing nations. Since its emergence, Acquired Immunodeficiency Syndrome (AIDS) caused by the Human Immunodeficiency Virus (HIV) has claimed millions of lives, weakening immune systems and leaving individuals

vulnerable to opportunistic infections and cancers (21). The disease primarily targets $CD4^+$ T lymphocytes, with clinical AIDS diagnosed when $CD4^+$ cell counts fall below $200mm^{-3}$, a critical threshold marking severe immunosuppression (13).

Mathematical modeling has become an indispensable tool in understanding HIV transmission dynamics and evaluating intervention strategies (1; 11). While deterministic models like the Susceptible-Infected-Recovered (SIR) framework have provided valuable insights (11), they often fail to capture the inherent stochasticity of disease transmission in real populations. This limitation becomes particularly apparent when modeling small populations or attempting to estimate outbreak probabilities and final epidemic sizes (5). Stochastic Differential Equation (SDE) models address these shortcomings by incorporating random fluctuations that better reflect biological and environmental variability (4).

Recent advances in stochastic modeling have revealed important phenomena not captured by deterministic approaches. Studies have demonstrated that environmental noise can both stabilize and destabilize disease dynamics, with even small perturbations significantly affecting long-term outcomes (9; 2). This understanding has led to the development of increasingly sophisticated models that account for heterogeneous transmission patterns (14), varying infectiousness stages (16), and population-specific risk behaviors (7).

This paper contributes to the growing body of stochastic HIV/AIDS modeling literature by developing a novel framework specifically tailored to heterosexual transmission networks, which account for approximately 80% of infections in many endemic regions (8). Our approach integrates differential equation methods with Markov chain techniques (22; 23) to capture both the continuous progression of infection and discrete transition probabilities between disease states. The model builds upon previous work by (6) and (18), while addressing several critical gaps in existing formulations.

This study ultimately aims to bridge the gap between deterministic HIV/AIDS models and real-world transmission dynamics by incorporating environmental stochasticity, providing a more robust framework for understanding disease spread and evaluating control strategies in heterosexual populations.

# 2 Model Description, Formulation and Analysis

## 2.1 Model Description and Formulation

The population under study $N(t)$ is sub divided into three compartments; Susceptible S(t), Infected class $I(t)$ and Aids class $A(t)$ where, $S(t)$ are individuals who are at risk of contracting HIV, Infective I(t) are individuals who are infected with HIV and can transmit the virus while AIDS A(t) compartment comprises of individuals who have progressed to AIDS and are no longer sexually active (assumed to be no longer transmitting the virus. The susceptible individuals are recruited at a rate $\Lambda$. Natural mortality rate is given by $\mu$. The probability of transmission of the virus is given by $\beta = \frac{\tau c}{N(t)}$, where $\tau$ is the contact rate of HIV transmission per sexual act and $c$ quantifies the mean sexual partnership rate between susceptible and infectious population members. The infected individuals then progress from HIV to AIDS at the rate $\delta$ and $\alpha$ is the rate at which individuals die as a result of AIDS. $\sigma_1$, $\sigma_2$, $\sigma_3$ are noise intensities representing random fluctuations and $dB_1(t)$, $dB_2(t)$, $dB_3(t)$ are standard brownian motions representing stochastic noise. The summary of the model parameters are shown in Table 1.

Table 1: **Model Descriptions**

| **Parameters** | **Model parameters descriptions** |
|---|---|
| $\Lambda$ | Natural recruitment rate to susceptible class |
| $\beta$ | Rate at which the virus is transmitted |
| $\tau$ | The rate at of HIV transmission per sexual act |
| $c$ | sexual partnership rate between susceptible and infectious |
| $\delta$ | Rate at which infected individuals transit from HIV to AIDS |
| $\alpha$ | the rate at which individuals die as a result of AIDS |
| $\sigma_i$ | Represents noise intensities random fluctuations |
| $\mu$ | Natural mortality rate |
| $dB_i(t)$ | Represent standard brownian motions representing stochastic noise |

The stochastic terms account for random fluctuations in the recruitment of susceptible individuals into the population, reflecting real-life scenarios such as migration. At a given time $t$, the total population is given by ;

$$N(t) = S(t) + I(t) + A(t) \tag{1}$$

This implies that the first derivative is given by;

$$dN(t) = [dS(t) + dI(t) + dA(t)]dt \tag{2}$$

where the system of stochastic differential equations (SDEs) described above is of the form:

$$\begin{aligned} dS(t) &= [\Lambda - \beta SI - \mu S]\, dt + \sigma_1 S\, dB_1(t), \\ dI(t) &= [\beta SI - (\mu + \delta)I]\, dt + \sigma_2 I\, dB_2(t), \\ dA(t) &= [\delta I - (\mu + \alpha)A]\, dt + \sigma_3 A\, dB_3(t), \end{aligned} \tag{3}$$

where $B_i$; $i = 1, 2, 3$ are the diffusion terms such that $B_i \sim N(0, 1)$. The description above can be described schematically by:

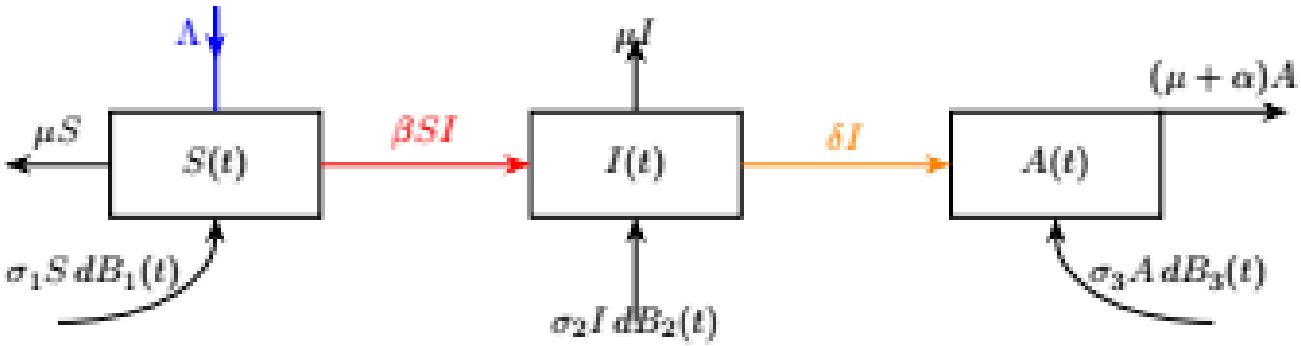


Figure 1: SIA Schematic diagram

# 3 Model Analysis

## 3.1 Stochastic Moment Analysis

This analysis presents a comprehensive stochastic framework for HIV/AIDS population dynamics, examining both mean behavior and variability through moment analysis. The derivation begins with exact moment equations obtained through Itô calculus, revealing how disease transmission, progression, and mortality shape population means and variances.

### 3.1.1 Mean of Susceptible Compartment S(t)

Starting from the the first equation of the SDE system (3) given by:

$$dS = (\Lambda - \beta SI - \mu S)dt + \sigma_1 S dB_1(t) \tag{4}$$

Apply Itô's lemma (15) to $f(S) = S$:

$$df(S) = \frac{\partial f}{\partial S} dS + \frac{1}{2}\frac{\partial^2 f}{\partial S^2}(dS)^2 \tag{5}$$

Since $\frac{\partial^2 f}{\partial S^2} = 0$, we have:

$$E[dS] = E[(\Lambda - \beta SI - \mu S)dt] + E[\sigma_1 S dB_1(t)] \tag{6}$$

Noting $E[dB_1(t)] = 0$ and dividing by $dt$:

$$\frac{dm_S}{dt} = \Lambda - \beta E[SI] - \mu m_S \tag{7}$$

where $m_S = E[S(t)]$.

The susceptible population dynamics combine three components: a constant recruitment rate ($\Lambda$) independent of population size, a non-linear infection term ($-\beta E[SI]$) accounting for both mean interactions ($m_S m_I$) and disease spread correlations ($\mathrm{Cov}(S, I)$), and a linear mortality term ($-\mu m_S$) ensuring biological realism. The model balances inflow ($\Lambda$) against depletion from both infection (incorporating spatial/temporal correlations through covariance) and natural deaths ($\mu$), with their relative magnitudes determining the baseline susceptible population.

### 3.1.2 Variance of Susceptible Compartment S(t)

Using Itô's lemma on $f(S) = S^2$:

$$d(S^2) = 2SdS + (dS)^2 \tag{8}$$

Substituting $dS$ and taking expectations:

$$E[d(S^2)] = 2E[S(\Lambda - \beta SI - \mu S)]dt + \sigma_1^2 E[S^2]dt \tag{9}$$

$$= [2\Lambda m_S - 2\beta E[S^2 I] - 2\mu E[S^2] + \sigma_1^2 E[S^2]]dt \tag{10}$$

Since $V_S = E[S^2] - m_S^2$, we obtain:

$$\frac{dV_S}{dt} = \sigma_1^2(V_S + m_S^2) + 2\Lambda m_S - 2\beta E[S^2 I] - 2\mu(V_S + m_S^2) - 2m_S\frac{dm_S}{dt} \tag{11}$$

After simplification:

$$\frac{dV_S}{dt} = \sigma_1^2 m_S^2 - 2\mu V_S - 2\beta(E[S^2 I] - m_S E[SI]) \tag{12}$$

The variance dynamics of the susceptible population are governed by three key mechanisms: demographic stochasticity ($\sigma_1^2 m_S^2$), introducing quadratic noise scaling with population size; transmission effects ($-2\beta(E[S^2I] - m_S E[SI])$), capturing higher-order infection-driven variability through third-order moments and spatial clustering; and variance damping ($-2\mu V_S$), where mortality dissipates fluctuations at twice the mean rate. These components collectively determine how noise, nonlinear transmission processes, and natural population turnover shape the stochastic spread of disease. The system's full description requires moment closure due to its dependence on higher-order correlations.

### 3.1.3 Mean of Infected Compartment I(t)

From the SDE system (3):

$$dI = (\beta SI - (\mu + \delta)I)dt + \sigma_2 I dB_2(t) \tag{13}$$

Following similar steps yields:

$$\frac{dm_I}{dt} = \beta E[SI] - (\mu + \delta)m_I \tag{14}$$

The mean infected population dynamics are characterized by competing growth and loss processes.The growth term ($\beta E[SI]$) directly mirrors the corresponding loss term in the susceptible population equation, maintaining conservation of individuals in the transmission process. This term highlights how disease transmission depends crucially on the average population levels ($m_S m_I$).

The loss terms ($-(\mu + \delta)m_I$) combine two distinct removal processes from the infected population. The natural mortality rate ($\mu$) affects all population compartments equally, while the disease progression rate ($\delta$) specifically represents the transition from HIV infection to AIDS. The combined coefficient ($\mu + \delta$) determines the average duration of the infected state, with higher values leading to faster depletion of the infected population through either death or disease progression. The progression rate $\delta$ serves as a critical parameter linking the infected and AIDS compartments in the model's disease staging framework.

### 3.1.4 Variance of Infected Compartment I(t)

Similarly for $I^2$:

$$\frac{dV_I}{dt} = \sigma_2^2 m_I^2 - 2(\mu + \delta)V_I + 2\beta(E[SI^2] - m_I E[SI]) \tag{15}$$

The infected population's variance dynamics are shaped by competing effects: a positive feedback term ($2\beta(E[SI^2] - m_I E[SI]$) drives variability amplification through nonlinear transmission and higher-order correlations, while an enhanced damping term ($-2(\mu+\delta)V_I$) provides stronger variability suppression than in susceptible populations due to combined natural mortality ($\mu$) and AIDS progression ($\delta$). The third-order moment $E[SI^2]$ captures how spatial clustering affects transmission variability, with the multiplicative nature of infection potentially causing rapid variability growth during outbreaks. However, the quadratic variance dependence makes the damping effects twice as strong as for mean population dynamics, creating a balance between infection-driven variability amplification and disease progression/mortality-induced dissipation.

### 3.1.5 Mean of Aids Compartment A(t)

From the SDE system (3):

$$dA = (\delta I - (\mu + \alpha)A)dt + \sigma_3 A dB_3(t) \tag{16}$$

Similarly applying Ito's lemma (15); Yielding:

$$\frac{dm_A}{dt} = \delta m_I - (\mu + \alpha)m_A \tag{17}$$

The mean AIDS population dynamics balance inflow from disease progression ($\delta m_I$), where $\delta^{-1}$ represents the average pre-AIDS infection duration, against accelerated mortality ($-(\mu+\alpha)m_A$) that combines natural death ($\mu$) with AIDS-specific fatality ($\alpha \gg \mu$). The model captures the unidirectional flow from HIV to AIDS, with the large $\alpha$ term reflecting significantly reduced post-diagnosis survival times and establishing AIDS as the terminal disease stage in this progression framework. The relative magnitudes of $\delta$ and $(\mu + \alpha)$ determine the equilibrium size of the AIDS population.

#### 3.1.6 Variance of AIDS Compartment A(t)

For $A^2$:

$$\frac{dV_A}{dt} = \sigma_3^2 m_A^2 - 2(\mu + \alpha)V_A + 2\delta(E[IA] - m_A m_I) \tag{18}$$

The AIDS compartment's variance dynamics are characterized by two dominant effects. The coupling term ($2\delta(E[IA] - m_A m_I)$ facilitates variability transfer from the infected population, where the magnitude depends on the covariance structure between infected and AIDS individuals. This term ensures outbreak fluctuations propagate through disease progression stages. The strong damping term ($-2(\mu+\alpha)V_A$) reflects the AIDS compartment's rapid turnover, where the combined natural and disease-induced mortality ($\alpha \gg \mu$) quickly dissipates variability, making this compartment's fluctuations the most short-lived in the system.

### 3.2 Positivity of Solutions of the Model

Since model (3) describes human population, it should be well posed. Thus in this section, positivity and boundedness solutions of model (3) are discussed.

**Theorem 3.1.** *If the initial conditions satisfy $S_0 > 0$, $I_0 > 0$, $A_0 > 0$, then the solutions $S_t$, $I_t$, $A_t$ remain positive for all $t \geq 0$ since human population cannot grow negatively.*

*Proof.* For susceptible compartment $S(t)$, the SDE for $S(t)$ is:

$$dS(t) = [\Lambda - \beta S(t)I(t) - \mu S(t)]\, dt + \sigma_1 S(t) dB_1(t) \tag{19}$$

This can be rewritten as:

$$\frac{dS(t)}{S(t)} = \left[\frac{\Lambda}{S(t)} - \beta I(t) - \mu\right] dt + \sigma_1 dB_1(t) \tag{20}$$

Consider the transformation $Y_t = \ln X_t^i$ for each component $X_t^i$ of the system and applying Itō's formula:

$$d(\ln X_t^i) = \left(\frac{f_i(X_t)}{X_t^i} - \frac{g_i^2(X_t)}{2(X_t^i)^2}\right) dt + \frac{g_i(X_t)}{X_t^i} dW_t \tag{21}$$

to $\ln S(t)$ gives:

$$d(\ln S(t)) = \frac{dS(t)}{S(t)} - \frac{1}{2}\frac{(dS(t))^2}{S(t)^2} \tag{22}$$

Substituting and simplifying:

$$d(\ln S(t)) = \left[\frac{\Lambda}{S(t)} - \beta I(t) - \mu - \frac{\sigma_1^2}{2}\right] dt + \sigma_1 dB_1(t) \tag{23}$$

Integrating from 0 to $t$:

$$\ln S(t) - \ln S(0) = \int_0^t \left[ \frac{\Lambda}{S(s)} - \beta I(s) - \mu - \frac{\sigma_1^2}{2} \right] ds + \sigma_1 B_1(t) \tag{24}$$

Taking exponential of both sides:

$$S(t) = S(0) \exp\left( \int_0^t \left[ \frac{\Lambda}{S(s)} - \beta I(s) - \mu - \frac{\sigma_1^2}{2} \right] ds + \sigma_1 B_1(t) \right) > 0 \tag{25}$$

Since $S(0) > 0$ and the exponential function is always positive, $S(t) > 0$ for all $t > 0$.

In a similar manner for infected compartment $I(t)$ and $A(t)$ yields:

$$I(t) = I(0) \exp\left( \int_0^t \left[ \beta S(s) - (\mu + \delta) - \frac{\sigma_2^2}{2} \right] ds + \sigma_2 B_2(t) \right) > 0 \tag{26}$$

and

$$A(t) = A(0) \exp\left( \int_0^t \left[ \frac{\delta I(s)}{A(s)} - (\mu + \alpha) - \frac{\sigma_3^2}{2} \right] ds + \sigma_3 B_3(t) \right) > 0 \tag{27}$$

□

The analysis demonstrates that the solutions $S(t)$, $I(t)$, and $A(t)$ of the stochastic model (3) remain strictly positive for all time $t \geq 0$ when initialized with positive conditions $S_0 > 0$, $I_0 > 0$, and $A_0 > 0$. This is proven by expressing each compartment in its logarithmic form via Itô's formula and showing that their solutions are exponentials of finite stochastic integrals, which guarantees positivity.

### 3.3 Boundedness of Solutions of the Model

**Proposition 3.2.** *Let $t_0 > 0$. If $S_0 > 0$, $I_0 > 0$ and $A_0 > 0$ then $S(t)$, $I(t)$ and $A(t)$ will each remain bounded in $\in R^3$ for all $t \in [0, t_0]$.*

*Proof.* The differential of $N(t)$ is:

$$dN(t) = dS(t) + dI(t) + dA(t) \tag{28}$$

Substituting the model equations (3.1) in (3.15) we obtain:

$$dN(t) = [\Lambda - \mu N(t) - \alpha A(t)]\, dt + \sigma_1 S(t) dB_1(t) + \sigma_2 I(t) dB_2(t) + \sigma_3 A(t) dB_3(t) \tag{29}$$

We analyze the boundedness of a stochastic epidemic model, taking expectations to measure fluctuations around the mean yields:

$$\frac{dE[N(t)]}{dt} = \Lambda - \mu E[N(t)] - \alpha E[A(t)] \leq \Lambda - \mu E[N(t)] \tag{30}$$

This is a differential inequality of the form:

$$\frac{dE[N(t)]}{dt} + \mu E[N(t)] \leq \Lambda \tag{31}$$

Multiplying both sides by the integrating factor $e^{\mu t}$:

$$\frac{d}{dt}\left( E[N(t)] e^{\mu t} \right) \leq \Lambda e^{\mu t} \tag{32}$$

Integrating both sides from 0 to $t$:

$$E[N(t)]e^{\mu t} - N(0) \leq \frac{\Lambda}{\mu}(e^{\mu t} - 1) \tag{33}$$

Rearranging:

$$E[N(t)] \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}) \leq \max\left(N(0), \frac{\Lambda}{\mu}\right) \tag{34}$$

As $t \to \infty$, we have:

$$\lim_{t\to\infty} E[N(t)] \leq \frac{\Lambda}{\mu} \tag{35}$$

The mathematical analysis demonstrates that the total population size $N(t) = S(t)+I(t)+A(t)$ remains bounded above by $\frac{\Lambda}{\mu}$ in expectation, where $\Lambda$ represents the constant recruitment rate and $\mu$ denotes the natural mortality rate. This fundamental upper bound has important implications for all population compartments: the susceptible ($S(t)$), infected ($I(t)$), and AIDS ($A(t)$) subpopulations are each constrained by this limit.

This implies that the population growth is bounded by the disease-free case and the bound is consistent with the disease-free equilibrium where $A(t) = 0$.

□

# 4 Determination of the Basic Reproduction Number

The basic reproduction number $R_0$ is the number of secondary infections resulting from the introduction of an infective individual into a population of susceptible individuals.

*Proof.* First, we identify the disease-free equilibrium (DFE) by setting the infected compartments to zero and solving the steady-state equations:

$$\text{DFE} = (S^0, I^0, A^0) = \left(\frac{\Lambda}{\mu}, 0, 0\right) \tag{36}$$

This is obtained by solving (37):

$$\begin{cases} \Lambda - \beta S^0 I^0 - \mu S^0 = 0 & \Rightarrow S^0 = \dfrac{\Lambda}{\mu} \\ \beta S^0 I^0 - (\mu + \delta) I^0 = 0 & \Rightarrow I^0 = 0 \\ \delta I^0 - (\mu + \alpha) A^0 = 0 & \Rightarrow A^0 = 0 \end{cases} \tag{37}$$

Using the method of next generation matrix method to compute $R_0$. The infected compartments are $I$ and $A$, but since $A$ individuals do not transmit the virus, we only consider $I$.

The new infection terms $\mathcal{F}$ and transition terms $\mathcal{V}$ are extracted from the infected compartment equation:

$$\frac{dI}{dt} = \underbrace{\beta SI}_{\mathcal{F}} - \underbrace{(\mu + \delta)I}_{\mathcal{V}} \tag{38}$$

Thus:

$$\mathcal{F} \quad = \beta SI \tag{39}$$

$$\mathcal{V} \quad = (\mu + \delta)I \tag{40}$$

By linearization system (3) at DFE, we compute the Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ with respect to $I$ and evaluate at the DFE:

$$F = \left.\frac{\partial \mathcal{F}}{\partial I}\right|_{\text{DFE}} = \beta S^0 = \beta \frac{\Lambda}{\mu} \tag{41}$$

$$V = \left.\frac{\partial \mathcal{V}}{\partial I}\right|_{\text{DFE}} = \mu + \delta \tag{42}$$

The basic reproduction number is the spectral radius of the next generation matrix $FV^{-1}$. Since both $F$ and $V$ are scalars in this case we have:

$$R_0 = \frac{F}{V} = \frac{\beta \frac{\Lambda}{\mu}}{\mu + \delta} \tag{43}$$

Substituting $\beta = \frac{\tau c}{N}$ in (43) and noting that at DFE, $N = S^0 = \frac{\Lambda}{\mu}$ yields:

$$R_0 = \frac{\tau c \frac{\Lambda}{\mu} \frac{1}{\frac{\Lambda}{\mu}}}{\mu + \delta} = \frac{\tau c}{\mu + \delta} \tag{44}$$

The basic reproduction number $R_0$ can be interpreted as: $\tau c$ - The product of transmission probability $\tau$ and contact rate $c$, representing the expected number of new infections per unit time from one infected individual in a fully susceptible population. $\frac{1}{\mu+\delta}$ - The average duration of infectiousness, accounting for both natural mortality $\mu$ and progression to AIDS - $\delta$.

To derive the stochastic $R_0^S$, we use the approach from (5), where noise reduces the effective reproduction number.

The Itô drift-diffusion process for $I(t)$ is:

$$dI = [\beta SI - (\mu + \delta)I]\, dt + \sigma_2 I\, dB_2(t).$$

The noise term introduces an additional variance correction. The adjusted reproduction number becomes:

$$R_0^S = R_0 - \frac{\sigma_2^2}{2(\mu + \delta)}.$$

The final expression for $R_0^S$ substituting $R_0$ is given by:

$$R_0^S = \frac{\beta \Lambda}{\mu(\mu + \delta)} - \frac{\sigma_2^2}{2(\mu + \delta)}.$$

This implies that if $R_0^S < 1$, the disease dies out and if $R_0^S > 1$, the disease may persist. The term $\frac{\sigma_2^2}{2(\mu+\delta)}$ quantifies how noise reduces $R_0$.

□

# 5 Existence of Stochastic Disease-Free Equilibrium (DFE)

The Disease-Free Equilibrium (DFE) is a steady state solution of the system where there is no disease present in the population. For our model, this means that:

$$I(t) = 0 \quad \text{and} \quad A(t) = 0 \quad \text{for all} \quad t \geq 0 \tag{45}$$

At DFE, $I(t) = 0$ and $A(t) = 0$. The susceptible equation becomes:

$$dS(t) = [\Lambda - \mu S(t)]\, dt + \sigma_1 S(t)\, dB_1(t).$$

Given $I(t) = 0$ and $A(t) = 0$, the mean of $S(t)$ at equilibrium is:

$$E[S] = \frac{\Lambda}{\mu}.$$

The variance is:

$$\text{Var}(S) = \frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu - \sigma_1^2)}.$$

Thus, the disease-free equilibrium (DFE) is a random variable:

$$S^0 \sim \left(\frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu - \sigma_1^2)}\right), \quad I^0 = 0, \quad A^0 = 0.$$

Therefore the Stochastic $DFE$ is given by;

$$DFE = \left(\frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu - \sigma_1^2)}, \quad 0, \quad 0\right)$$

### 5.1 Stochastic Stability Analysis at DFE

**Theorem 5.1.** *For any time $t \geq 0$, the stochastic DFE=*

$$S^0 \sim \left(\frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu - \sigma_1^2)}\right), \quad I^0 = 0, \quad A^0 = 0.$$

*of model (3) is locally asymptotically stable whenever $R_0^S < 1$ and unstable whenever $R_0^S > 1$.*

*Proof.* For the stochastic system, we use the method of Lyapunov functions to analyze stability. The stochastic system linearized at $E_0$ is:

$$\begin{aligned}
dX_1 &= \left(-\mu X_1 - \beta\frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu - \sigma_1^2)}X_2\right)dt + \sigma_1\frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu - \sigma_1^2)}dB_1(t) \\
dX_2 &= \left(\beta\frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu - \sigma_1^2)} - \mu - \delta\right)X_2 dt + \sigma_2 \cdot 0 \cdot dB_2(t) \\
dX_3 &= (\delta X_2 - (\mu + \alpha)X_3)\,dt + \sigma_3 \cdot 0 \cdot dB_3(t)
\end{aligned} \tag{46}$$

where $X = (S - S^0, I - I^0, A - A^0)$.
Considering the quadratic Lyapunov function (3):

$$V(X) = \frac{1}{2}\left(X_1^2 + X_2^2 + X_3^2\right) \tag{47}$$

Applying Itô's formula (6):

$$dV = \mathcal{L}V dt + \sigma_1\frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu - \sigma_1^2)}X_1 dB_1(t) \tag{48}$$

where $\mathcal{L}V$ is the diffusion operator:

$$\begin{aligned}
\mathcal{L}V = {} & X_1(-\mu X_1 - \beta\frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu - \sigma_1^2)}X_2) + X_2(\beta\frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu - \sigma_1^2)} - \mu - \delta)X_2 + X_3(\delta X_2 - (\mu + \alpha)X_3) \\
& + \frac{1}{2}\sigma_1^2\left(\frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu - \sigma_1^2)}\right)^2
\end{aligned}$$

For stability, we need $\mathcal{L}V$ negative definite. The dominant terms are:

$$\mathcal{L}V \leq -\mu X_1^2 + (\beta \frac{\Lambda^2 \sigma_1^2}{\mu^2(2\mu - \sigma_1^2)} - \mu - \delta) X_2^2 - (\mu + \alpha) X_3^2 + \text{higher order terms} + \text{noise term} \quad (49)$$

The condition $R_0^S < 1$ makes the $X_2^2$ coefficient negative.

The stochastic DFE is:

$$\text{DFE}^S = \left( \frac{\Lambda^2 \sigma_1^2}{\mu^2(2\mu - \sigma_1^2)}, 0, 0 \right),$$

and it is stochastically stable if $R_0^S < 1$, and Unstable if $R_0^S > 1$. This implies that if $\sigma_1 > 0$, the stochastic DFE is not a fixed point but a stationary distribution centered at $(S_0, 0, 0)$.

The stability analysis reveals that the disease-free equilibrium (DFE) is stable when $R_0^S < 1$ in both deterministic and stochastic frameworks, though the stochastic case imposes the additional requirement of sufficiently small noise intensity ($\sigma_1$ below a threshold). While the deterministic model guarantees local asymptotic stability of $E_0$ when $R_0^S < 1$, the stochastic version demonstrates more restrictive stability conditions due to system fluctuations. In both frameworks, the DFE becomes unstable when $R_0^S > 1$, indicating potential disease endemicity. This highlights how stochastic effects introduce additional constraints beyond the deterministic stability threshold. □

# 6 Stochastic Endemic Equilibrium Analysis

**Theorem 6.1.** *For any time $t \geq 0$, the EE of the stochastic model (3.1) is locally asymptotically stable whenever $R_0^S > 1$, otherwise unstable.*

*Proof.* By linearized System at EE, let $(x, y, z) = (S - S^*, I - I^*, A - A^*)$. The linearized system is:

$$\begin{cases} dx = [-\beta(I^* x + S^* y) - \mu x]dt + \sigma_1 S^* dB_1(t) \\ dy = [\beta(I^* x + S^* y) - (\mu + \delta) y]dt + \sigma_2 I^* dB_2(t) \\ dz = [\delta y - (\mu + \alpha) z]dt + \sigma_3 A^* dB_3(t) \end{cases} \quad (50)$$

Consider the quadratic Lyapunov function:

$$V(x, y, z) = \frac{1}{2}\left(ax^2 + by^2 + cz^2\right) \quad (51)$$

where $a, b, c > 0$ are constants to be determined.
Applying Itô's formula:

$$dV = \mathcal{L}V dt + \text{noise terms} \quad (52)$$

where the diffusion operator is:

$$\mathcal{L}V = ax[-\beta(I^* x + S^* y) - \mu x] + by[\beta(I^* x + S^* y) - (\mu + \delta) y]$$
$$+ cz[\delta y - (\mu + \alpha) z] + \frac{1}{2}(a\sigma_1^2 S^{*2} + b\sigma_2^2 I^{*2} + c\sigma_3^2 A^{*2})$$

To ensure $\mathcal{L}V$ is negative definite, we choose $a = b$ to cancel cross terms, select $c$ such that remaining terms are negative such that the noise intensities must satisfy:

$$\sigma_1^2 < \frac{2\mu}{S^*}, \quad \sigma_2^2 < \frac{2(\mu + \delta)}{I^*}, \quad \sigma_3^2 < \frac{2(\mu + \alpha)}{A^*} \quad (53)$$

This implies that the endemic equilibrium $E^*$ is locally asymptotically stable in the deterministic case when $R_0 > 1$, stochastically stable when $R_0^S > 1$ and noise intensities are sufficiently small and the stability conditions are more restrictive in the stochastic case.

Biologically when the basic reproduction number exceeds 1 and noise and diffusion terms are not too large, the HIV/AIDS disease will persist at stable endemic levels.

# 7 Analysis of HIV/AIDS Discrete Time MC Model

In this section, we develop a stochastic framework to analyze the dynamics of HIV/AIDS transmission. Traditional deterministic models provide valuable insights into disease behavior, but they fail to capture the inherent randomness in transmission processes and disease progression. We therefore construct a discrete-state continuous-time Markov chain model that accounts for these stochastic effects and analyze the transition probabilities, probability of evolution and stationary distribution analysis with respect to noise effect and hence simulate the effects of noise in the dynamics of HIV/AIDS disease.

**Definition 7.1.** *The Markov chain is defined on the discrete state space:*

$$\mathcal{S} = \{(s, i, a) \in Z_+^3 \,|\, s + i + a \leq N_{max}\}$$

*where:*

*(i)* $s$ = *number of susceptible individuals*

*(ii)* $i$ = *number of infected individuals*

*(iii)* $a$ = *number of AIDS cases*

*(iv)* $N_{max}$ = *maximum population capacity*

**Definition 7.2.** *We consider discrete time steps:*

$$t \in \{0, \Delta t, 2\Delta t, \ldots\}$$

*where $\Delta t$ is chosen sufficiently small to ensure only one transition can occur per time step.*

## 7.1 Transition Probability Analysis with Noise Effects

In this section, we compute the transition probabilities of the stochastic HIV/AIDS model, incorporating noise terms to quantify the likelihood of each event in a small time interval. This is essential for simulating disease dynamics using stochastic simulation algorithms, particularly in the context of the stochastic reproduction number $R_0^S$.

**Theorem 7.3.** *For $\Delta t \to 0$, the transition probabilities are:*

$$p_{(s,i,a)\to(s',i',a')} = \begin{cases} \Lambda\Delta t + o(\Delta t) & \textit{(Recruitment)} \\ \frac{\tau csi}{s+i+a}\Delta t + \sigma_1 si\, dB_1(t) + o(\Delta t) & \textit{(Infection with noise)} \\ \delta i\Delta t + \sigma_2 i\, dB_2(t) + o(\Delta t) & \textit{(Progression with noise)} \\ \mu s\Delta t + o(\Delta t) & \textit{(S death)} \\ \mu i\Delta t + \sigma_3 i\, dB_3(t) + o(\Delta t) & \textit{(I death with noise)} \\ (\mu+\alpha)a\Delta t + \sigma_4 a\, dB_4(t) + o(\Delta t) & \textit{(A death with noise)} \\ 1 - R_{tot}\Delta t + o(\Delta t) & \textit{(No change)} \end{cases} \tag{54}$$

*where:*

$$R_{tot} = \Lambda + \frac{\tau csi}{s+i+a} + \delta i + \mu(s+i) + (\mu+\alpha)a \tag{55}$$

*and $\sigma_k\, dB_k(t)$ represent noise terms affecting infection, progression, and death rates.*

*Proof.* Incorporating noise in transition probabilities

(i) The deterministic infection rate is $\frac{\tau csi}{s+i+a}$, but variability introduces a noise term $\sigma_1 si\, dB_1(t)$:

$$P_{\text{infection}} = \left(\frac{\tau csi}{s+i+a}\right)\Delta t + \sigma_1 si\, \Delta B_1(t) + o(\Delta t) \tag{56}$$

Here, $\Delta B_1(t) \sim \mathcal{N}(0, \Delta t)$ represents Wiener process increments.

(ii) The progression rate $\delta i$ is perturbed by $\sigma_2 i\, dB_2(t)$:

$$P_{\text{progression}} = \delta i \Delta t + \sigma_2 i\, \Delta B_2(t) + o(\Delta t) \tag{57}$$

This noise term modifies the expected transition time to AIDS, affecting $R_0^S$ and mortality terms with noise include; natural death in $I$: $\mu i \Delta t + \sigma_3 i\, \Delta B_3(t)$ and AIDS-related death: $(\mu+\alpha)a\Delta t + \sigma_4 a\, \Delta B_4(t)$

The probability of no state change is adjusted by the total transition rate $R_{tot}$, now including noise-induced variance:

$$P_{\text{no change}} = 1 - \left[R_{tot}\Delta t + \sum_k \sigma_k(\text{noise contributions})\right] + o(\Delta t) \tag{58}$$

The stochastic term $\sigma_1 si\, dB_1(t)$ introduces variability in disease transmission dynamics, effectively reducing the infection rate through random fluctuations in contact patterns. This phenomenon directly relates to the stochastic reproduction number $R_0^S$, given by:

$$R_0^S = \frac{\tau c}{\mu+\delta} - \frac{\sigma_2^2}{2(\mu+\delta)}, \tag{59}$$

The stochastic analysis reveals how noise terms significantly modify disease dynamics through multiple pathways. The progression noise $\sigma_2^2$ appears quadratically in $R_0^S$, demonstrating its damping effect by introducing variability in the infectious period duration. Similarly, mortality noise terms $\sigma_3$ (infected) and $\sigma_4$ (AIDS) alter disease burden by stochastically modifying the duration of infectious and terminal stages. These fluctuations create complex interactions: when $R_0^S < 1$, noise can accelerate extinction through increased transmission variance, while for $R_0^S > 1$, it may sustain endemic conditions or generate unpredictable outbreak patterns through nonlinear stochastic interactions.

Key insights emerge from incorporating environmental noise terms $\sigma_k dB_k(t)$. First, they provide a crucial connection between deterministic $R_0$ and stochastic $R_0^S$ by modifying transition probabilities. Second, these terms collectively adjust both individual transition rates and overall system dynamics, mathematically justifying the variance correction in $R_0^S$. While maintaining the core frequency-dependent transmission structure ($\tau csi/N$), the noise terms enhance biological realism by capturing inherent randomness in human contact patterns and behavioral fluctuations characteristic of real epidemics. □

## 7.2 Probability of Evolution of Solutions with Stochastic Effects

This section analyzes the probabilistic evolution of our HIV/AIDS model under noise, connecting the system's state transitions to the stochastic reproduction number $R_0^S$. The inclusion of noise terms provides a complete characterization of how random fluctuations influence disease dynamics.

**Theorem 7.4.** *The equation governing system evolution with noise is:*

$$\begin{aligned}\frac{d}{dt}p_{s,i,a}(t) = &\Lambda p_{s-1,i,a}(t)\\ &+\left(\frac{\tau c(s+1)(i-1)}{N}+\sigma_1(s+1)(i-1)\xi_1(t)\right)p_{s+1,i-1,a}(t)\\ &+\left(\delta(i+1)+\sigma_2(i+1)\xi_2(t)\right)p_{s,i+1,a-1}(t)\\ &+\mu(s+1)p_{s+1,i,a}(t)\\ &+\left(\mu(i+1)+\sigma_3(i+1)\xi_3(t)\right)p_{s,i+1,a}(t)\\ &+\left((\mu+\alpha)(a+1)+\sigma_4(a+1)\xi_4(t)\right)p_{s,i,a+1}(t)\\ &-\left(R_{tot}+\sum_{k=1}^{4}\sigma_k\xi_k(t)\right)p_{s,i,a}(t)\end{aligned}$$

*where $\xi_k(t)$ represent noise processes and $\sigma_k$ their intensities.*

*Proof.* The discrete-time evolution with noise terms becomes:

$$\begin{aligned}p_{s,i,a}(t+\Delta t) = &\Lambda\Delta t p_{s-1,i,a}(t)\\ &+\left(\frac{\tau c(s+1)(i-1)}{N}+\sigma_1\Delta B_1\right)\Delta t p_{s+1,i-1,a}(t)\\ &+\left(\delta(i+1)+\sigma_2\Delta B_2\right)\Delta t p_{s,i+1,a-1}(t)\\ &+(\text{other terms})\\ &+\left(1-\left(R_{tot}+\sum\sigma_k\Delta B_k\right)\Delta t\right)p_{s,i,a}(t)+o(\Delta t)\end{aligned}$$

Taking $\Delta t\to 0$ yields the continuous-time equation where $\Delta B_k/\Delta t\to\xi_k(t)$. □

The stochastic reproduction number $R_0^S$ captures how environmental noise modifies disease transmission dynamics through several mechanisms. The infection noise ($\sigma_1$) reduces effective transmission rates by introducing variability in contact patterns, which is reflected in the correction term $-\frac{1}{2(\mu+\delta)}\sum_{k=1}^{4}\sigma_k^2$ of the equation:

$$R_0^S = \frac{\tau c}{\mu+\delta}-\frac{1}{2(\mu+\delta)}\sum_{k=1}^{4}\sigma_k^2. \tag{60}$$

The stochastic model reveals how noise terms fundamentally alter disease dynamics. Progression noise ($\sigma_2$) modifies the infectious period distribution by introducing variability in the $\delta$ transition rate, while mortality noises ($\sigma_3$, $\sigma_4$) affect disease stage durations and transmission potential. Below the epidemic threshold ($R_0^S<1$), noise accelerates disease extinction by increasing transmission variance, whereas above threshold ($R_0^S>1$), it creates metastable endemic states with prevalence fluctuations around deterministic equilibria. The covariance structure between noise processes $\xi_k(t)$ critically influences outbreak variability, particularly when transmission and progression noises are positively correlated.

Key mathematical insights emerge from the noise structure: (1) multiplicative noise preserves solution non-negativity, maintaining probabilistic interpretation of states $p_{s,i,a}(t)$; (2) the quadratic

$\sigma_2^2$ correction in $R_0^S$ originates from Itô-to-Stratonovich conversion in the continuous-time limit; and (3) while keeping core frequency-dependent transmission ($\tau csi/N$), the model effectively adjusts rates through noise interactions. This framework rigorously connects individual stochastic transitions to population-level thresholds, providing a complete stochastic description of HIV/AIDS disease progression.

# 8 Stochastic Simulation Algorithm with Noise Effects

The total transition rate incorporating noise terms becomes:

$$R_{tot}^S = \underbrace{\Lambda}_{\text{recruitment}} + \underbrace{\left(\frac{\tau cSI}{N} + \sigma_1 SI\xi_1(t)\right)}_{\text{noisy infection}} + \underbrace{(\delta I + \sigma_2 I\xi_2(t))}_{\text{noisy progression}}$$
$$+ \underbrace{\mu S + (\mu I + \sigma_3 I\xi_3(t)) + ((\mu+\alpha)A + \sigma_4 A\xi_4(t))}_{\text{noisy deaths}}$$

**Theorem 8.1.** *The waiting time distribution becomes:*

$$P(\tau > t) = E\left[e^{-\int_0^t R_{tot}^S(s)ds}\right]$$

*with density:*

$$f_\tau^S(t) = E\left[R_{tot}^S(t)e^{-\int_0^t R_{tot}^S(s)ds}\right]$$

*Proof.* For small $\Delta t$, the survival probability modifies to:

$$P_0(\Delta t) = 1 - R_{tot}^S\Delta t + \frac{1}{2}\sum_{k=1}^4 \sigma_k^2\Delta t + o(\Delta t)$$

The Itô correction term $\frac{1}{2}\sum\sigma_k^2$ emerges from:

$$\lim_{n\to\infty}\prod_{j=1}^n\left(1 - R_{tot}^S(t_j)\frac{t}{n} + \frac{1}{2}\sum\sigma_k^2\frac{t}{n}\right) = E\left[e^{-\int_0^t R_{tot}^S(s)ds}\right]$$

□

The modified transition probabilities reflect the adjusted reproduction number:

$$R_0^S = \frac{\tau c}{\mu+\delta} - \frac{1}{2(\mu+\delta)}\sum_{k=1}^4\sigma_k^2 \tag{61}$$

The event selection probabilities become:

$$P^S(\text{select } k) = \frac{r_k^S}{R_{tot}^S}$$

where:

$$r_1^S = \Lambda$$
$$r_2^S = \frac{\tau cSI}{N} + \sigma_1 SI\xi_1(t)$$
$$r_3^S = \delta I + \sigma_2 I\xi_2(t)$$
$$r_4^S = \mu S + (\mu I + \sigma_3 I\xi_3(t)) + ((\mu+\alpha)A + \sigma_4 A\xi_4(t))$$

The incorporation of noise terms in the stochastic HIV/AIDS model yields several important epidemiological consequences. First, the infection probability conditional on any event occurring becomes $P^S(\text{infection}|\text{event}) = (\tau cSI/N + \sigma_1 SI\xi_1(t))/R_{tot}^S$, where the additional term $\sigma_1 SI\xi_1(t)$ captures variability in transmission. This noise component increases the variance in infection events, leading to more unpredictable outbreak patterns compared to deterministic models.

When the stochastic reproduction number satisfies $R_0^S < 1$, the modified transition rates accelerate the system's convergence to the disease-free state. The noise terms effectively enhance the probability of extinction by introducing additional pathways for the infection to die out. Conversely, in the endemic case where $R_0^S > 1$, the noise induces persistent oscillations in disease prevalence around the deterministic equilibrium. These fluctuations, driven by the $\sigma_k \xi_k(t)$ terms, better reflect the observed variability in real-world HIV/AIDS dynamics, where perfect stability is never achieved due to constantly changing social and environmental factors.

## 9 Stationary Distribution Analysis with Noise Effects

Incorporating noise in transition rates we modify each transition rate with additive noise terms:

$$\begin{aligned}
\text{Infection rate} &\to \frac{\tau csi}{N} + \sigma_1 si\xi_1(t) \\
\text{Progression rate} &\to \delta i + \sigma_2 i\xi_2(t) \\
\text{Death rates} &\to \begin{cases} \mu i + \sigma_3 i\xi_3(t) & \text{(Infected)} \\ (\mu+\alpha)a + \sigma_4 a\xi_4(t) & \text{(AIDS)} \end{cases}
\end{aligned}$$

where $\xi_k(t)$ are white noise processes with $E[\xi_k(t)] = 0$ and $E[\xi_k(t)\xi_l(t')] = \delta_{kl}\delta_{(t-t')}$.
Deriving the noise-modified Equation. The probability evolution now follows:

$$\frac{d}{dt}p_{s,i,a}(t) = \sum_{k=1}^{4} \left[\mathcal{L}_k + \sigma_k \mathcal{N}_k\right] p_{s,i,a}(t) \tag{62}$$

where $\mathcal{L}_k$ are deterministic Liouville operators and $\mathcal{N}_k$ are noise operators. At equilibrium:

$$0 = \left(\sum_{k=1}^{4} \mathcal{L}_k + \frac{1}{2}\sum_{k=1}^{4} \sigma_k^2 \mathcal{N}_k^2\right) \pi_{s,i,a}^S \tag{63}$$

Global balance equation with noise. The stationary distribution $\pi_{s,i,a}^S$ satisfies:

$$\begin{aligned}
&\pi_{s,i,a}^S \left[\Lambda + \frac{\tau csi}{N} + \delta i + \mu(s+i) + (\mu+\alpha)a + \frac{1}{2}\left(\sigma_1^2 s^2 i^2 + \sigma_2^2 i^2 + \sigma_3^2 i^2 + \sigma_4^2 a^2\right)\right] \\
&= (\text{Inflow terms with corresponding noise corrections})
\end{aligned}$$

The noise terms modify the effective reproduction number given by:

$$R_0^S = \underbrace{\frac{\tau c}{\mu+\delta}}_{\text{Deterministic}} - \underbrace{\frac{\sigma_1^2 + \sigma_2^2 + \sigma_3^2}{2(\mu+\delta)}}_{\text{Noise correction}} \tag{64}$$

By the use of the Fokker-Planck approximation, the variance terms appear as:

$$\frac{d}{dt}p = \underbrace{-\nabla \cdot (\mathbf{v}p)}_{\text{Drift}} + \underbrace{\frac{1}{2}\sum_{k=1}^{4} \sigma_k^2 \frac{\partial^2}{\partial x_k^2} p}_{\text{Diffusion}} \tag{65}$$

where the diffusion terms generate the quadratic noise corrections in $R_0^S$. □

For reversible transitions:

$$\pi_{s,i,a}^S \left( \frac{\tau csi}{N} + \sigma_1^2 s^2 i^2 \right) = \pi_{s-1,i+1,a}^S \left( \mu(i+1) + \sigma_3^2 (i+1)^2 \right)$$
$$\pi_{s,i,a}^S \left( \delta i + \sigma_2^2 i^2 \right) = \pi_{s,i-1,a+1}^S \left( (\mu+\alpha)(a+1) + \sigma_4^2 (a+1)^2 \right)$$

Noise-induced extinction occurs when $R_0^S < 1$, as the additional variance terms accelerate convergence to the disease-free state $\pi_{s,0,0}^S$. This indicates that in environments with lower reproduction numbers, noise effects drive the system towards the extinction of the disease. In contrast, endemic fluctuations are observed when $R_0^S > 1$, where the stationary distribution develops a peak at the endemic equilibrium level, represented by:

$$I^* = \frac{\Lambda}{\mu}\left(1 - \frac{1}{R_0^S}\right) \pm \sqrt{\frac{\sum \sigma_k^2}{2(\mu+\delta)}} \tag{66}$$

This expression characterizes the endemic state, incorporating both the mean transmission rate and the variability introduced by the noise terms. In the context of intervention design, control measures must account for both the mean transmission rate and the noise-induced variance. The effective intervention $\tau_{\text{eff}}$ is given by:

$$\tau_{\text{eff}} = \tau - \frac{N}{2c}\sum_{k=1}^{4} \sigma_k^2 \tag{67}$$

which adjusts the intervention parameter to account for stochastic fluctuations.
The implementation of the model requires discretizing the state space $(S, I, A)$, followed by solving the noise-modified balance equations iteratively. Once the system is solved, various statistical moments can be computed. The expected value of the infected population is given by:

$$E[I] = \sum i \pi_{s,i,a}^S$$
$$\text{Var}(I) = \sum (i - E[I])^2 \pi_{s,i,a}^S + \sum \sigma_k^2 E[I^2]$$

These moments allow for a deeper understanding of the system's behavior under the influence of stochastic noise.
The noise-aware stationary distribution analysis reveals several important insights. It provides a quantitative relationship between environmental variability and disease persistence, enabling the prediction of disease behavior under fluctuating conditions. Additionally, it defines the precise conditions for epidemic fade-out, specifically when $R_0^S < 1$, indicating the threshold below which disease extinction is likely. Finally, it enhances the design of interventions by accounting for the stochastic effects of noise, leading to more accurate thresholds for effective control measures.

## 10 Numerical Simulation

The stochastic HIV/AIDS model was simulated using the Euler-Maruyama method with varying noise intensities ($\sigma_1$, $\sigma_2$, $\sigma_3$) for the susceptible, infected, and AIDS compartments respectively. The simulation results reveal complex dynamics influenced by stochastic factors. The total population $N(t)$ exhibits fluctuations around a generally stable equilibrium, with the amplitude of these fluctuations increasing proportionally with noise intensity. This suggests that while the system maintains an overall balance, random environmental factors can cause significant temporary deviations from equilibrium. These results are as shown in Figure 2 and Figure 3.

## 11 Parameter Values

Table 2: Parameter values used in numerical simulations

| **Parameter** | **Description** | **Value** | **Source** |
|---|---|---|---|
| $\Lambda$ | Recruitment rate | Calculated from equilibrium | (8) |
| $\beta$ | Transmission rate | $\tau c/N$ | (13) |
| $\tau$ | Transmission probability per contact | 0.5 | (4) |
| $c$ | Mean sexual partnership rate | 3.0 | (7) |
| $\mu$ | Natural mortality rate | 0.02 | (24) |
| $\delta$ | HIV to AIDS progression rate | 0.1 | (8) |
| $\alpha$ | AIDS-related mortality rate | 0.3 | (24) |
| $\sigma_1$ | Susceptible noise intensity | 0.1 | (5) |
| $\sigma_2$ | Infected noise intensity | 0.15 | (2) |
| $\sigma_3$ | AIDS noise intensity | 0.1 | (4) |
| $S(0)$ | Initial susceptible population | 100 | Assumed |
| $I(0)$ | Initial infected population | 5 | Assumed |
| $A(0)$ | Initial AIDS population | 0 | Assumed |

## 12 Simulation Results of HIV/AIDS Model with Noise

From Figure 2, the susceptible population graph shows the temporal evolution of individuals at risk of HIV infection. Initially starting at 100 individuals, the population exhibits stochastic fluctuations (with noise intensity $\sigma_1 = 0.1$) around a generally decreasing trend. This decline reflects both natural mortality ($\mu = 0.02$) and infection transmission to the HIV-positive compartment. The noise introduces variability in the susceptible population size, with the magnitude of fluctuations proportional to the population size itself. The stabilization pattern suggests the system may be approaching a stochastic equilibrium where recruitment $\Lambda$ balances losses from infection and natural mortality.

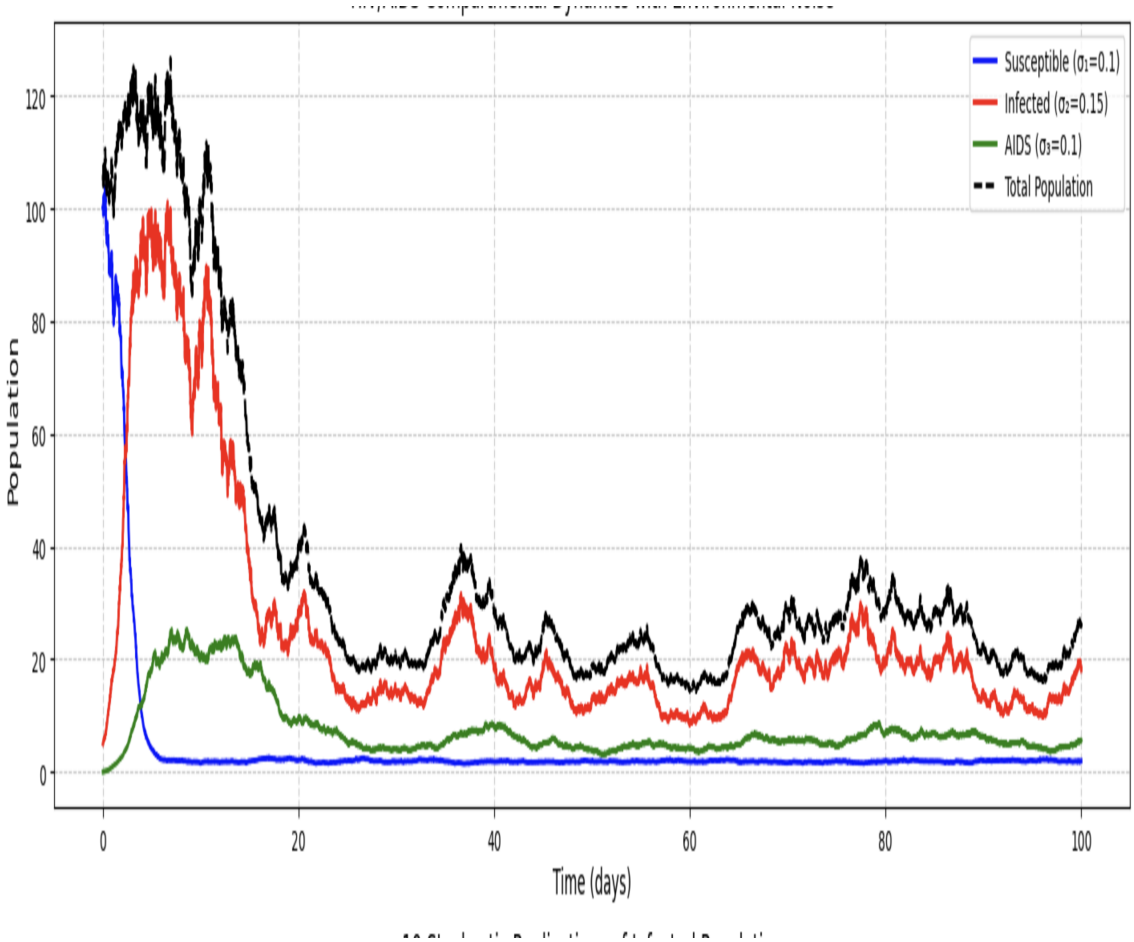


Figure 2: HIV/AIDS Compartmental Dynamics with Noise

The infected population plot displays more pronounced variability (noise intensity $\sigma_2 = 0.15$) compared to the susceptible group. Starting from an initial 5 infected individuals, the population grows rapidly due to new infections (transmission rate $\beta$ dependent on contact rate $c = 3.0$ and transmission probability $\tau = 0.5$), then stabilizes with significant stochastic oscillations. The higher noise intensity in this compartment amplifies the random fluctuations. The eventual stabilization reflects a balance between new infections and losses due to progression to AIDS ($\delta = 0.1$) and natural mortality.

The AIDS compartment shows a gradual increase from zero initial cases, with moderate noise ($\sigma_3 = 0.1$). The growth rate is determined by the progression rate from HIV to AIDS ($\delta = 0.1$), while the decline is influenced by the elevated mortality rate ($\alpha = 0.3$) specific to AIDS patients. The fluctuations are less dramatic than in the infected population but still noticeable, demonstrating how noise variability affects disease progression. The delayed peak compared to the infected population reflects the time lag in disease progression as seen in Figure 2.

Figure 3 is a graph that shows the multiple simulation plot that demonstrates the variability in model outcomes due to stochasticity. Each colored trajectory represents a different realization of the infected population dynamics under identical parameters but different noise sequences. The spread of outcomes highlights the importance of considering multiple realizations in stochastic modeling. While all runs follow similar qualitative patterns that is, initial growth followed by stabilization, the specific timing and magnitude of peaks vary considerably. This underscores how random environmental factors can lead to diverse epidemic trajectories even with identical initial conditions and transmission parameters. The stochastic HIV/AIDS model analysis

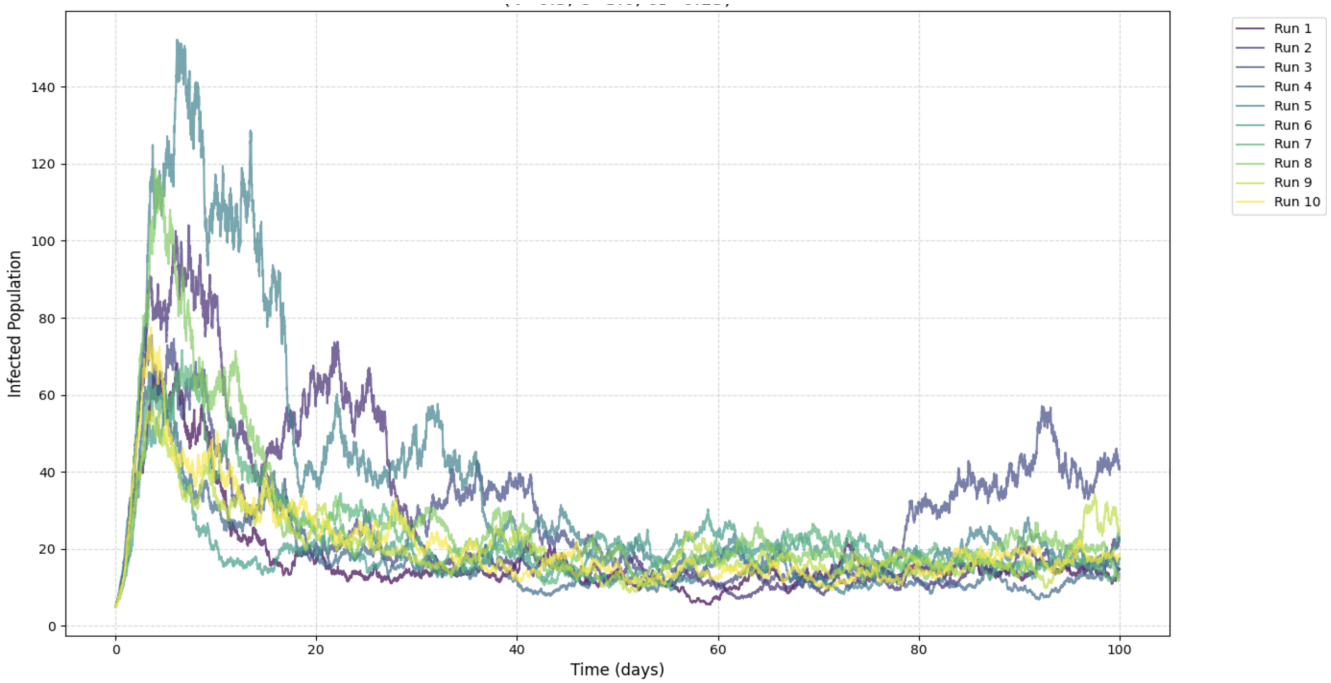


Figure 3: Multiple Stochastic Realizations

demonstrates how environmental variability ($\sigma_1 = 0.1$, $\sigma_2 = 0.15$, $\sigma_3 = 0.1$) influences disease dynamics across all compartments, with the infected population showing particularly pronounced fluctuations due to its higher noise intensity. While deterministic trends emerge from the underlying biological processes that is, transmission rate $\beta$, progression rate $\delta$, and mortality parameters, the multiple realizations reveal substantial outcome variability under identical conditions. The system exhibits stable stochastic behavior where recruitment balances disease-related and natural mortality, though compartment-specific noise creates distinct fluctuation patterns.

## 13 Conclusion

The stochastic HIV/AIDS model developed in this study provides significant insights into disease dynamics under noise variability. Key findings demonstrate that noise intensities substantially influence disease persistence, with the infected compartment showing the most pronounced fluctuations due to its higher noise parameter ($\sigma_2 = 0.15$). The derived stochastic reproduction number $R_0^S = \frac{\tau c}{\mu+\delta} - \frac{\sigma_2^2}{2(\mu+\delta)}$ quantitatively captures how environmental noise reduces transmission potential, offering a more realistic threshold parameter than its deterministic counterpart. Stability analysis confirms that noise modifies the conditions for disease eradication, requiring more stringent control measures when $R_0^S$ approaches 1 from below. Multiple stochastic realizations reveal substantial outcome variability despite identical parameters, emphasizing the importance of considering noises fluctuations in epidemic forecasting. The model's extension to a Markov chain framework successfully captures discrete-state transitions with noise effects, providing a versatile tool for analyzing both short-term outbreak dynamics and long-term endemic behavior. These results collectively demonstrate that conventional deterministic models may significantly underestimate the complexity of HIV/AIDS transmission dynamics in real populations.

## 14 Conflict of Interest

The authors declare that they have no any form of conflict of interest.

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