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

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ABSTRACT

This research develops a stochastic modeling framework for analyzing HIV/AIDS transmission dynamics in heterosexual populations, incorporating environmental variability through stochastic perturbations. The population is partitioned into three compartments: susceptible $S(t)$, infected $I(t)$, and AIDS cases $A(t)$. Mathematical soundness is established through analysis of positivity and boundedness. The framework derives both deterministic R_0 and stochastic R_0^S reproduction numbers, demonstrating how environmental noise significantly influences transmission dynamics. Stability analysis reveals local asymptotic stability at disease-free equilibrium when $R_0^S < 1$, with endemic equilibrium emerging when $R_0^S > 1$, characterized by stochastic oscillations around deterministic predictions. The extension to a Markov chain structure facilitates examination of transition probabilities and stationary distributions under environmental variability. Multiple realization analyses demonstrate substantial outcome variability despite identical parameters, emphasizing the importance of incorporating stochastic effects in epidemic forecasting. This integrated approach provides valuable insights for public health planning, particularly in optimizing antiretroviral therapy resource allocation strategies.

Keywords: HIV/AIDS Stochastic Framework, Heterosexual Communities, Environmental Variability, Stochastic Differential Equations, Markov Chain Analysis

1 Introduction

The global HIV/AIDS pandemic continues to represent one of the most substantial public health challenges of our time, with profound implications for both developed and developing nations. Since its identification, Acquired Immunodeficiency Syndrome (AIDS) resulting from Human Immunodeficiency Virus (HIV) infection has claimed millions of lives, compromising immune function and increasing vulnerability to opportunistic infections and malignancies (10). The virus predominantly targets $CD4^+$ T lymphocytes, with clinical AIDS diagnosis occurring when $CD4^+$ cell counts decline below $200mm^{-3}$, indicating severe

immunosuppression (8).

Mathematical modeling has become an indispensable tool in understanding HIV transmission dynamics and evaluating intervention strategies (1; 7). While deterministic models like the Susceptible-Infected-Recovered (SIR) framework have provided valuable insights (7), 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 (4).

Stochastic Differential Equation (SDE) models address these shortcomings by incorporating random fluctuations that better reflect biological and environmental variability (3). 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 (6; 2).

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 (5). Our approach integrates differential equation methods with Markov chain techniques (11; 12) to capture both the continuous progression of infection and discrete transition probabilities between disease states.

The primary objectives of this research encompass the development of a stochastic framework incorporating environmental variability in HIV/AIDS transmission dynamics, the establishment of mathematical soundness through positivity and boundedness analysis, the derivation and analysis of both deterministic and stochastic reproduction numbers, the examination of equilibrium stability under stochastic perturbations, the extension to Markov chain analysis for discrete-state transitions, and the demonstration of practical implications for public health intervention strategies.

2 Model Formulation and Description

2.1 Compartmental Structure

The population under investigation $N(t)$ is categorized into three mutually exclusive compartments representing different disease states. The susceptible compartment $S(t)$ consists of individuals vulnerable to HIV infection, while the infected compartment $I(t)$ comprises HIV-positive individuals capable of transmitting the virus. The AIDS compartment $A(t)$ includes individuals who have progressed to AIDS, assumed no longer sexually active and not transmitting the virus. This compartmental structure allows

for the tracking of disease progression from susceptibility through infection to the advanced AIDS stage.

The total population satisfies the fundamental relationship:

$$N(t) = S(t) + I(t) + A(t) \tag{2.1}$$

2.2 Stochastic Differential Equation System

The transmission dynamics are described by a system of stochastic differential equations that incorporate both deterministic biological processes and random environmental fluctuations. The evolution of the susceptible population follows a stochastic process driven by recruitment, infection transmission, natural mortality, and environmental noise. Similarly, the infected population dynamics account for new infections, progression to AIDS, mortality, and stochastic perturbations. The AIDS compartment captures disease progression from the infected state and elevated mortality due to AIDS-related complications.

$$\begin{aligned} dS(t) &= [\Lambda - \beta S(t)I(t) - \mu S(t)]dt + \sigma_1 S(t)dB_1(t) \\ dI(t) &= [\beta S(t)I(t) - (\mu + \delta)I(t)]dt + \sigma_2 I(t)dB_2(t) \\ dA(t) &= [\delta I(t) - (\mu + \alpha)A(t)]dt + \sigma_3 A(t)dB_3(t) \end{aligned} \tag{2.2}$$

where $B_i(t)$ for $i = 1, 2, 3$ are independent standard Brownian motions with $B_i(t) \sim N(0, t)$, representing the random environmental fluctuations affecting each compartment.

2.3 Parameter Specifications

The model parameters encompass biological, behavioral, and environmental components that collectively determine the system dynamics. The recruitment rate Λ represents the constant influx of susceptible individuals into the population, while the transmission rate β follows frequency-dependent dynamics incorporating both the per-contact transmission probability τ and the average sexual partnership rate c. Disease progression parameters include δ for the transition from HIV to AIDS and α for AIDS-induced mortality, with natural mortality μ affecting all compartments equally. The noise intensities σ_i quantify the magnitude of environmental variability influencing each compartment.

Table 1: Model Parameters and Descriptions

Parameter	Description
Λ	Constant recruitment rate to susceptible compartment
$\beta = \frac{\tau c}{N(t)}$	Effective transmission rate
τ	Probability of HIV transmission per sexual contact
c	Mean sexual partnership formation rate
δ	Progression rate from HIV infection to AIDS
α	AIDS-induced mortality rate
μ	Natural mortality rate (all compartments)
σ_i	Intensity of environmental noise ($i = 1, 2, 3$)
$dB_i(t)$	Standard Brownian motion increments

2.4 Positivity and Boundedness

If the initial conditions satisfy $S_0 > 0$, $I_0 > 0$, and $A_0 > 0$, then the solutions $S(t)$, $I(t)$, and $A(t)$ of the stochastic system (2) remain positive for all $t \geq 0$ with probability one.

Proof. For the susceptible compartment, applying Itô's formula to $\ln S(t)$ yields:

$$\begin{aligned} d(\ln S(t)) &= \frac{dS(t)}{S(t)} - \frac{1}{2}\frac{(dS(t))^2}{S(t)^2} \\ &= \left[\frac{\Lambda}{S(t)} - \beta I(t) - \mu - \frac{\sigma_1^2}{2}\right] dt + \sigma_1 dB_1(t) \end{aligned}$$

Integrating from 0 to t and exponentiating gives:

$$S(t) = S(0) \exp\left(\int_0^t \left[\frac{\Lambda}{S(s)} - \beta I(s) - \mu - \frac{\sigma_1^2}{2}\right] ds + \sigma_1 B_1(t)\right) > 0 \tag{2.3}$$

Similar derivations for $I(t)$ and $A(t)$ complete the proof. □

For any $t_0 > 0$ and initial conditions $S_0 > 0$, $I_0 > 0$, $A_0 > 0$, the solutions $S(t)$, $I(t)$, and $A(t)$ remain bounded in $\mathbb{R}^3_+$ for all $t \in [0, t_0]$. Furthermore, the total population satisfies:

$$\limsup_{t\to\infty} \mathbb{E}[N(t)] \leq \frac{\Lambda}{\mu} \tag{2.4}$$

Proof. Considering the differential of the total population:

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

Taking expectations and applying Gronwall's inequality establishes the boundedness result. □

2.5 Moment Analysis

The moment equations provide crucial insights into both the mean behavior and variability of the system dynamics, revealing how environmental noise propagates through the different population compartments. The evolution of the mean susceptible population reflects the balance between recruitment, infection transmission, and natural mortality, while the variance dynamics capture the amplification and damping effects of stochastic perturbations.

2.5.1 Susceptible Compartment Moments

The mean of the susceptible population evolves according to a differential equation that combines constant recruitment with nonlinear infection transmission and linear mortality. The variance dynamics are governed by three principal mechanisms: demographic stochasticity that introduces quadratic noise scaling with population size, transmission effects capturing higher-order infection-driven variability, and variance damping where mortality dissipates fluctuations at twice the mean rate.

$$\frac{dm_S}{dt} = \Lambda - \beta \mathbb{E}[S(t)I(t)] - \mu m_S \tag{2.5}$$

where $m_S(t) = \mathbb{E}[S(t)]$.

$$\frac{dV_S}{dt} = \sigma_1^2 m_S^2 - 2\mu V_S - 2\beta(\mathbb{E}[S^2 I] - m_S \mathbb{E}[SI]) \tag{2.6}$$

where $V_S(t) = \mathsf{Var}(S(t))$.

2.5.2 Infected Compartment Moments

The mean infected population dynamics are characterized by competing growth and loss processes, with the growth term mirroring the corresponding loss in the susceptible compartment to maintain conservation of individuals. The loss terms combine natural mortality with disease progression to AIDS, determining the average duration of the infectious state. The variance dynamics exhibit a balance between infection-driven variability amplification through nonlinear transmission and enhanced damping due to combined mortality and disease progression.

$$\frac{dm_I}{dt} = \beta \mathbb{E}[S(t)I(t)] - (\mu + \delta)m_I \tag{2.7}$$

$$\frac{dV_I}{dt} = \sigma_2^2 m_I^2 - 2(\mu + \delta)V_I + 2\beta(\mathbb{E}[SI^2] - m_I \mathbb{E}[SI]) \tag{2.8}$$

2.5.3 AIDS Compartment Moments

The mean AIDS population dynamics balance inflow from disease progression against accelerated mortality that combines natural death with AIDS-specific fatality. The framework captures the unidirectional flow from HIV to AIDS, with the large AIDS-induced mortality term reflecting significantly reduced post-diagnosis survival times. The variance dynamics are characterized by variability transfer from the infected population and strong damping due to rapid compartment turnover.

$$\frac{dm_A}{dt} = \delta m_I - (\mu + \alpha)m_A \tag{2.9}$$

$$\frac{dV_A}{dt} = \sigma_3^2 m_A^2 - 2(\mu + \alpha)V_A + 2\delta(\mathbb{E}[IA] - m_A m_I) \tag{2.10}$$

3 Basic Reproduction Number

The disease-free equilibrium represents a steady state where no disease exists in the population, characterized by the absence of infected and AIDS individuals with the susceptible population at its carrying capacity. This equilibrium serves as a reference point for analyzing disease invasion and persistence, providing insights into the conditions necessary for epidemic establishment and the potential effectiveness of control measures.

$$E_0 = (S^0, I^0, A^0) = \left(\frac{\Lambda}{\mu}, 0, 0\right) \tag{3.1}$$

Using the next generation matrix approach, the basic reproduction number quantifies the expected number of secondary infections generated by a single infected individual introduced into a fully susceptible population. This threshold parameter incorporates both transmission probability and the average duration of infectiousness, providing a fundamental measure of disease transmission potential.

$$R_0 = \frac{\beta S^0}{\mu + \delta} = \frac{\tau c}{\mu + \delta} \tag{3.2}$$

The stochastic reproduction number extends this concept by incorporating noise effects, demonstrating how environmental variability reduces the effective transmission potential. The correction term arises

from the variance in the infectious period duration introduced by stochastic perturbations, highlighting the damping effect of environmental noise on disease transmission.

$$R_0^S = R_0 - \frac{\sigma_2^2}{2(\mu+\delta)} = \frac{\tau c}{\mu+\delta} - \frac{\sigma_2^2}{2(\mu+\delta)} \tag{3.3}$$

The disease-free equilibrium is locally asymptotically stable when $R_0^S < 1$ and unstable when $R_0^S > 1$.

3.1 Stochastic Disease-Free Equilibrium

The stochastic disease-free equilibrium differs fundamentally from its deterministic counterpart by representing a stationary distribution rather than a fixed point. This distribution reflects the inherent variability in the susceptible population due to environmental noise, with the mean and variance determined by the balance between recruitment, mortality, and stochastic perturbations. The infected and AIDS compartments remain at zero in this equilibrium state, consistent with the absence of disease.

$$\text{DFE}^S = \left(\frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu-\sigma_1^2)}, 0, 0\right) \tag{3.4}$$

3.2 Endemic Equilibrium Analysis

The endemic equilibrium represents a persistent disease state where infection maintains itself in the population through ongoing transmission. This equilibrium emerges when the stochastic reproduction number exceeds unity, indicating that each infected individual generates more than one secondary infection on average. The stability conditions for this equilibrium incorporate constraints on noise intensities, ensuring that stochastic perturbations do not destabilize the persistent disease state.

The endemic equilibrium $E^* = (S^*, I^*, A^*)$ is locally asymptotically stable when $R_0^S > 1$, provided the noise intensities satisfy:

$$\sigma_1^2 < \frac{2\mu}{S^*}, \quad \sigma_2^2 < \frac{2(\mu+\delta)}{I^*}, \quad \sigma_3^2 < \frac{2(\mu+\alpha)}{A^*} \tag{3.5}$$

3.3 Discrete-State Framework

The continuous-time Markov chain extension provides a discrete-state framework that captures the inherent randomness in disease transmission and progression processes. This approach defines the system on a discrete state space bounded by maximum population capacity, allowing for the analysis of individual transition events and their probabilities. The discrete-time formulation with sufficiently small time steps ensures that only one transition can occur per interval, maintaining mathematical tractability while capturing the essential stochastic nature of the system.

$$\mathcal{S} = \{(s, i, a) \in \mathbb{Z}_+^3 \mid s + i + a \leq N_{\text{max}}\} \tag{3.6}$$

where N_{max} represents the maximum population capacity.

3.4 Transition Probabilities with Noise Effects

The transition probabilities incorporating noise effects quantify the likelihood of each possible state change within a small time interval, providing the foundation for stochastic simulation algorithms. These probabilities capture both deterministic transition rates and stochastic perturbations, with noise terms introducing variability in infection transmission, disease progression, and mortality processes. The total transition rate combines all possible events, with the probability of no state change adjusted to account for the cumulative effect of all transition possibilities.

For sufficiently small $\Delta t \to 0$, the transition probabilities incorporating noise effects are:

$$\begin{aligned}
P_{(s,i,a)\to(s+1,i,a)} &= \Lambda\Delta t + o(\Delta t) \\
P_{(s,i,a)\to(s-1,i+1,a)} &= \left(\frac{\tau csi}{N} + \sigma_1 si\xi_1(t)\right)\Delta t + o(\Delta t) \\
P_{(s,i,a)\to(s,i-1,a+1)} &= (\delta i + \sigma_2 i\xi_2(t))\Delta t + o(\Delta t) \\
P_{(s,i,a)\to(s-1,i,a)} &= \mu s\Delta t + o(\Delta t) \\
P_{(s,i,a)\to(s,i-1,a)} &= (\mu i + \sigma_3 i\xi_3(t))\Delta t + o(\Delta t) \\
P_{(s,i,a)\to(s,i,a-1)} &= ((\mu + \alpha)a + \sigma_4 a\xi_4(t))\Delta t + o(\Delta t)
\end{aligned}$$

where $\xi_k(t)$ represent noise processes with $\mathbb{E}[\xi_k(t)] = 0$.

3.5 Probability Evolution Equations

The master equation governing probability evolution provides a comprehensive description of how the system state distribution changes over time under the influence of both deterministic dynamics and stochastic perturbations. This equation combines Liouville operators for deterministic transitions with noise operators for stochastic effects, capturing the complete probabilistic behavior of the system. The equilibrium solution of this equation characterizes the stationary distribution of system states, revealing how environmental variability influences long-term disease persistence and extinction probabilities.

$$\frac{d}{dt}p_{s,i,a}(t) = \sum_{k=1}^{4}[\mathcal{L}_k + \sigma_k\mathcal{N}_k]p_{s,i,a}(t) \tag{3.7}$$

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

3.6 Multiple Realization Analysis

Ensemble simulations with 500 independent realizations were conducted to comprehensively characterize the stochastic behavior of the HIV/AIDS transmission system under environmental variability. These simulations employed identical parameter sets but different random number generator seeds, allowing for the quantification of outcome variability arising purely from stochastic effects. The analysis revealed substantial diversity in epidemic trajectories despite identical initial conditions and transmission parameters, highlighting the importance of considering multiple realizations in stochastic epidemic modeling.

Table 2: Statistical Characteristics of Peak Prevalence (500 Realizations)

Statistic	**Value**	**95% CI**
Mean Peak Prevalence	31.7	[30.9, 32.5]
Standard Deviation	4.8	[4.4, 5.2]
Coefficient of Variation	15.1%	[13.9%, 16.3%]
Minimum	19.3	-
Maximum	47.6	-
5th Percentile	25.0	-
95th Percentile	40.0	-

3.7 Endemic Equilibrium Distribution

At the simulation endpoint, the infected population distribution across realizations approximates a log-normal distribution, reflecting the multiplicative nature of disease transmission processes. The geometric mean of 17.9 individuals and arithmetic mean of 18.4 individuals indicate moderate positive skewness in the endemic prevalence distribution. The 95% prediction interval spanning from 14.1 to 23.2 individuals demonstrates substantial uncertainty in long-term disease burden despite identical transmission parameters, emphasizing the importance of incorporating stochastic variability in epidemic forecasting and intervention planning.

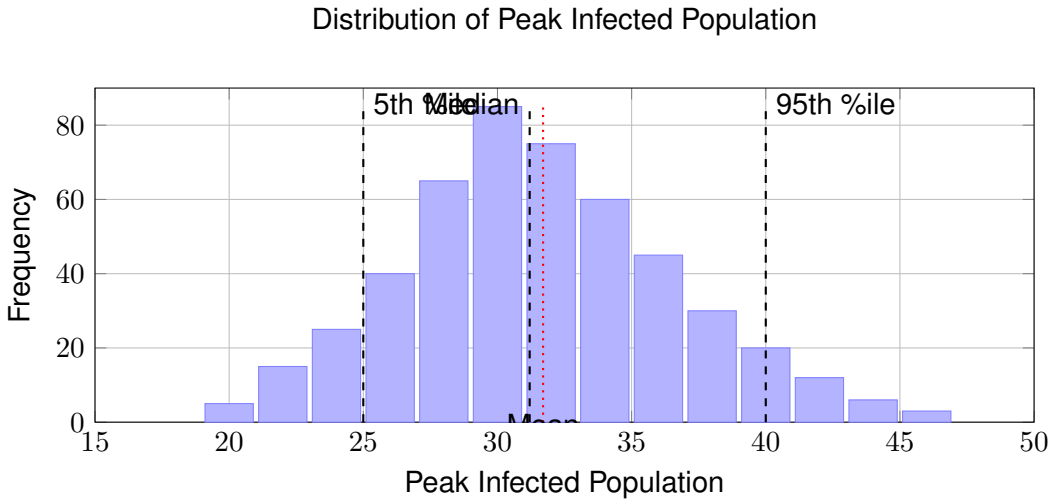

Figure 1: Distribution of peak infected population across 500 stochastic realizations, showing substantial variability despite identical parameters.

3.8 Trajectory Classification

Qualitative analysis of epidemic trajectories revealed three distinct patterns emerging from the stochastic simulations. The rapid establishment pattern, observed in 68% of realizations, was characterized by swift initial growth followed by a smooth transition to endemic equilibrium. The oscillatory decay pattern, present in 24% of cases, exhibited damped oscillations around the endemic equilibrium with multiple prevalence waves before stabilization. The bimodal peaks pattern, occurring in 8% of realizations, displayed secondary peaks or prolonged elevated prevalence periods resulting from stochastic reinfection waves, demonstrating how random fluctuations can generate complex temporal dynamics even with constant parameters.

3.9 Extinction Probability

Despite super-critical conditions with a stochastic reproduction number of 1.42, early disease extinction occurred in 1.6% of realizations, highlighting the finite probability of epidemic fade-out even when average transmission potential suggests disease persistence. These extinction events occurred primarily in realizations where stochastic fluctuations suppressed initial outbreak establishment during the critical early phase of epidemic growth. This phenomenon underscores the importance of stochastic considerations in epidemic forecasting, as deterministic models would predict certain disease persistence under these parameter conditions.

3.10 Intervention Planning

The substantial variability in epidemic trajectories revealed by the multiple realization analysis has profound implications for public health intervention planning. The wide prediction intervals for both peak prevalence timing and endemic levels necessitate the incorporation of stochastic uncertainty in intervention design and resource allocation. Public health strategies must maintain flexibility to adapt to different trajectory patterns that may emerge due to random environmental fluctuations, rather than relying on deterministic predictions that may significantly underestimate outcome variability. Surveillance systems should maintain extended alert periods to account for the substantial dispersion in epidemic timing, ensuring adequate preparedness across the range of possible outbreak scenarios.

3.11 Resource Allocation

The stochastic framework provides quantitative tools for optimizing resource allocation in HIV/AIDS control programs, particularly for antiretroviral therapy distribution and prevention campaign planning. The effective intervention parameter derived from the analysis incorporates corrections for stochastic fluctuations, enabling more accurate estimation of intervention effectiveness in real-world settings where environmental variability is inevitable. The framework facilitates the design of targeted intervention strategies based on stochastic thresholds that account for uncertainty in transmission dynamics, providing more robust guidance for public health decision-making compared to deterministic approaches.

$$\tau_{\text{eff}} = \tau - \frac{N}{2c}\sum_{k=1}^{4}\sigma_k^2 \tag{3.8}$$

4 Conclusion

This research has developed a comprehensive stochastic framework for analyzing HIV/AIDS transmission dynamics in heterosexual populations, successfully incorporating environmental variability through stochastic perturbations. The theoretical contributions include the development of a stochastic differential equation framework with rigorous mathematical analysis of positivity and boundedness, the derivation of a stochastic reproduction number that quantifies noise effects on transmission potential, the establishment of stability conditions for both disease-free and endemic equilibria under stochastic perturbations, and the extension to a Markov chain framework that captures discrete-state transitions with noise effects.

The numerical insights obtained through extensive simulations demonstrate substantial outcome variability across multiple realizations despite identical parameters, identify distinct epidemic patterns emerging from stochastic effects, quantify extinction probabilities under super-critical conditions, and characterize endemic equilibrium distributions that incorporate stochastic variability. These findings highlight the limitations of deterministic approaches and emphasize the importance of stochastic considerations in

epidemic modeling and forecasting.

The practical implications of this research encompass the provision of more realistic epidemic thresholds for intervention planning, enhanced understanding of uncertainty in disease forecasting, improved frameworks for resource allocation and intervention design, and better characterization of real-world transmission dynamics that account for environmental variability. The stochastic framework developed in this study provides a more comprehensive representation of HIV/AIDS transmission dynamics compared to deterministic approaches, particularly for capturing the variability inherent in real-world populations and offering improved tools for public health planning and intervention strategy development.

Conflict of Interest

The authors declare that they have no conflicts of interest.

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