A STOCHASTIC MODEL FOR THE SPREAD OF HIV/AIDS IN A HETEROSEXUAL POPULATION

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Title page

DECLARATION

This research thesis is my original work prepared with no other than the indicated sources and support and has not been presented elsewhere for a degree or any other award.

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CERTIFICATION

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DEDICATION

To my wife Roseline Khakayi Namusende

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ABSTRACT

Population dynamics and fluctuations exert strong effects on infectious disease transmission dynamics of infections such as HIV/AIDS. Existing stochastic HIV/AIDS models predominantly focused on homosexual populations and high-risk groups, largely neglecting the unique dynamics of heterosexual transmission. This oversight was particularly problematic given the distinct contact patterns that characterize heterosexual spread. In this study, a stochastic differential equation (SDE) model was derived to analyze HIV/AIDS transmission within a heterosexual population with incorporation of random perturbations to account for variability in the environment, migration, and other population uncertainties. The model partitions the population into susceptible $S(t)$, infected $I(t)$, and AIDS $A(t)$ compartments, with transmission governed by frequency-dependent contact rates. We established the model's mathematical well-posedness through positivity and boundedness proofs, and derived both deterministic R_0 and stochastic R_0^S reproduction numbers, demonstrating how environmental noise reduced transmission potential through the correction term $-\frac{\sigma_2^2}{2(\mu+\delta)}$. Itô's lemma was used to derive the mean and variance equations for each compartment. Stability analysis revealed that the disease-free equilibrium becomes locally asymptotically stable when $R_0^S < 1$, while an endemic equilibrium emerges when $R_0^S > 1$. Numerical simulations with noise intensities ($\sigma_1 = 0.1$, $\sigma_2 = 0.15$, $\sigma_3 = 0.1$) showed significant fluctuations in disease prevalence, particularly in the infected compartment. The study further developed a discrete-time Markov chain framework to analyze transition probabilities under noise effects, providing a comprehensive stochastic perspective on HIV/AIDS transmission dynamics that more accurately reflected real-world epidemic behavior compared to deterministic approaches.

ABBREVIATIONS AND ACRONYMS

ODE	:	Ordinary Differential Equation
BIC	:	Bayesian information criterion
MLE	:	Maximum Likelihood estimator
SDE	:	Stochastic Differential Equation
SIR	:	Susceptible-Infective-Recovered
SIS	:	Susceptible-Infective-Susceptible

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CHAPTER ONE

INTRODUCTION

1.1 Background of the study

Acquired Immunodeficiency Syndrome (AIDS) that induces Human Immunodeficiency Virus (HIV) has become a worldwide menace for both developed and developing nations. AIDS is a lethal disease that weakens the immune system of the body, leaving the patients susceptible to other life-taking opportunistic infections, neurological disorders, and rare cancers [3]. In the last decade, there have been comprehensive epidemiological investigations of HIV/AIDS, and it has been considered the worst plague of our era. Despite much uncertainty regarding HIV, scientists have gained invaluable knowledge of its transmission between populations [4].

HIV primarily infects some immune cells, the $CD4^+$ T lymphocytes. As a reaction to infection, the body's immune system produces both HIV-specific antibodies and cytotoxic T cells. It is seen by studies [15] that the antibody levels turn detectable between two to ten weeks after initial exposure. Healthy individuals without any HIV infection typically have a count of about $1000mm^{-3}$ of $CD4^+$ T cells. The clinical diagnosis of AIDS is established when this count drops to $200mm^{-3}$ or lower in HIV-infected patients[34]. The gradual loss of these key immune-regulating $CD4^+$ T cells results in the immunosuppression that is the hallmark of AIDS. The disease progression from HIV infection to AIDS, involving a considerable time lag, is therefore regulated by the body's capacity to replace virus-infected damaged cells [1].

HIV targets primarily certain immune cells, the $CD4^+$ T lymphocytes. As a response to infection, the body's immune system generates cytotoxic T cells as well as HIV-specific antibodies. It is seen from studies [15] that the levels of antibodies become

measurable ten to two weeks from the time of initial exposure. Uninfected healthy individuals typically have a count of approximately $1000mm^{-3}$ of $CD4^+$ T cells. The clinical diagnosis of AIDS is established if this count reduces to $200mm^{-3}$ or lower in HIV-infected individuals. The gradual loss of these crucial immune-regulating $CD4^+$ T cells results in the immunosuppression hallmark of AIDS. HIV infection process to AIDS with a considerable time lag is thus regulated by the capacity of the body to replace virus-infected damaged cells[13,18].

HIV is primarily a sexually transmitted disease – it is spread through common body fluid such as blood and semen. Transmission by sexual contact only is considered in this research. Perhaps one of the most vital aspects of HIV is the long latent period of 6 weeks to 8 weeks before AIDS [4]. In actual fact, there are some carriers who are disease positive but AIDS negative. During this period of infection the individual is infective and may infect others. This research assumes that as soon as an individual transitions from this infective state to AIDS, the individual ceases to be sexually active and is no longer able to infect others [18].

Stochastic Differential Equations (SDE's) models are also very close to ODE deterministic models except for including an additional stochastic term in their time derivatives of the compartments. SDE's have the advantage that, computationally, the simulation is nearly as fast as that of the equivalent deterministic ODE model. The use of deterministic epidemic models, e.g., the Susceptible - Infected - Recovered (SIR) model are impressive, but while the deterministic models have many advantages, it can be difficult (but not impossible) to incorporate realistic population networks into such models[31]. It is also difficult to incorporate realistic probability distributions for the time spent during the infectious period. This makes deterministic models unsuitable for the estimation of the probability of an outbreak, and the probability distributions of the final size of an epidemic in small populations. These call for the use of stochastic models

[5][33].

The path of the stochastic model gets nearer and nearer to the deterministic path and continues to shift randomly around the deterministic equilibrium for a very long time with very high probability. For instance in the simplest Susceptible-Infectious-Susceptible (SIS) model, the deterministic dynamic skeleton tells one a great deal about the behavior of the stochastic paths, and poses questions which are meaningful to the stochastic model: What is the behavior of the stochastic path of It as it oscillates near the deterministic equilibrium? Starting from the deterministic equilibrium, what is the form of the distribution of the time until the stochastic path is zero? The other properties which are peculiar to the stochastic epidemic models include: probability of an outbreak, quasi-stationary probability distribution, final size distribution of an epidemic and the expected duration of an epidemic.

1.2 Statement of the Problem

HIV/AIDS remains a major global health issue, with heterosexual transmission accounting for nearly 80% of infections in many high-prevalence areas. Existing stochastic HIV/AIDS models predominantly focused on homosexual populations and high-risk groups, largely neglecting the unique dynamics of heterosexual transmission. This oversight was particularly problematic given the distinct contact patterns that characterize heterosexual spread. The models have critical limitations that reduce their predictive accuracy and effectiveness in guiding interventions. The models also often ignore behavioral variations such as mobility patterns. The interplay between these behaviors and viral transmission remains inadequately addressed. In this study, a stochastic differential equation (SDE) model was derived to analyze HIV/AIDS transmission within a heterosexual population with incorporation of random perturbations to account for variability in the environment, migration, and other population uncertainties. The study

addresses these gaps by examining how heterosexual-specific contact patterns influence the stochastic component that is diffusion terms and noise terms on HIV/AIDS threshold R_0^S. This study further combines stochastic differential equations with Markov chain methods, tailored for heterosexual transmission, to provide both theoretical insights and practical simulation tools for public health strategies in affected regions.

1.3 Objectives

1.3.1 General Objective

To develop a stochastic model for the spread of HIV/AIDS in a heterosexual population using system of SDEs

1.3.2 Specific Objectives

(i) Develop a stochastic model of HIV/AIDS dynamics using a system of stochastic differential equations (SDEs).

(ii) To determine the mean and variance of the developed $SDEs$ population model.

(iii) Analyze the stability of the $SDEs$ model around its equilibrium points.

(iv) Perform numerical simulations of the stochastic differential equations (SDEs) model to investigate the impact of stochastic noise on HIV/AIDS disease spread in heterosexual population

1.4 Justification of the study

The development of a stochastic HIV/AIDS model for heterosexual populations is justified by critical limitations in existing deterministic approaches, which fail to capture the inherent randomness in disease transmission dynamics and predominantly focus on homosexual populations, thereby neglecting the unique contact patterns that characterize heterosexual spread. This research addresses these gaps by incorporating environmental noise, migration fluctuations, and behavioral variability through stochastic

differential equations and Markov chain methods, providing a more realistic framework that accounts for probability distributions of outbreak sizes, extinction probabilities, and the impact of random perturbations on disease persistence. The resulting model offers public health policymakers enhanced predictive tools for assessing intervention effectiveness under uncertainty, optimizing resource allocation, and designing robust control strategies that reflect the stochastic nature of real-world epidemic behavior in heterosexual networks.

1.5 Significance of the Study

In reality, the influx of people into a population is stochastic. The model presented has stochastic fluctuation in the migration of new susceptible into the susceptible class. The essence of this study is to introduce perturbations to make the model realistic in real life situation. Analysis of the stochastic model helps us to better understand how the disease spreads and improve our understanding on the major contributing factors in the spread of the disease.

1.6 Methods

In this study, the development and analysis of the model is done by the use of the following methods;

(i) Ordinary Differential Equations (ODEs)- These are differential equation in which the dependent variable (the unknown function and its derivative) are functions of one independent variable (the quantity upon which the dependent variable depends).

(ii) Monte Carlo Markov Chain approach has been used to estimate the probability distribution of of the model. A system is said to possess a Markovian property if;

$$P(X_{t+1} = K \mid X_0, \ldots, X_t) = P(X_{t+1}, K).$$

with probability one for all K and any $t \in \mathbb{N}$.

(iii) Perform numerical simulations of the stochastic differential equations (SDEs) model using the Sim.DiffProc package in R software.

CHAPTER TWO

LITERATURE REVIEW

2.1 Introduction

Since the discovery of HIV/AIDS, there has been an increase of mathematical models describing the dynamics of the disease. The study of mathematical Epidemiology i.e the analysis and building of mathematical models describing the control and spread of infectious diseases is of great concern in the areas of Mathematical Biology. Nowadays, the mathematical epidemic models are used routinely in the assessment of public health policies by health agencies [2],[5].

2.1.1 Stochastic Modeling in HIV/AIDS Epidemiology

Mathematical modeling of HIV/AIDS transmission has evolved significantly from deterministic to stochastic frameworks that better capture the inherent randomness in disease dynamics. [6] pioneered early stochastic differential equation (SDE) approaches to HIV modeling, demonstrating how environmental fluctuations affect transmission patterns. This work established the foundation for incorporating Brownian motion terms into epidemiological models, a methodology that our study extends to heterosexual populations.

Building on this foundation, [28] examined stochastic stability in SIR models with random perturbations, proving that endemic equilibria can be stochastically asymptotically stable under certain noise conditions. Their analysis of noise-induced stability transitions informs our approach to deriving the stochastic reproduction number R_0^S and its stability conditions.

Recent advances by [2] incorporated white noise effects into multi-group HIV models, showing how environmental variability reduces effective transmission rates. Their find-

ing that noise intensities σ_k quantitatively impact disease persistence aligns with our numerical simulations demonstrating compartment-specific fluctuations under varying noise parameters.

2.1.2 HIV Criminalization and Legal Frameworks

The legal context of HIV transmission is critically examined in [29], whose groundbreaking analysis of HIV criminalization and sex offender registration reveals how punitive laws persist despite scientific advances. Fulham's case study of Marcus Bennett illustrates how individuals face lifelong consequences for consensual sexual conduct without actual transmission, highlighting the disconnect between legal punishment and epidemiological reality.

This legal analysis is complemented by public health research from [35], which documents how HIV criminalization laws in over thirty states often ignore modern scientific understanding of transmission risks, particularly the undetectable=untransmittable (U=U) principle. Their findings that these laws discourage testing and treatment engagement reinforce the importance of our stochastic modeling approach in quantifying actual transmission risks.

[18] contributed significantly to understanding differential infectivity in HIV progression, developing models that account for variations in infectiousness across disease stages. While their DI model focused on chronic stage variations, our stochastic framework extends this by incorporating environmental noise affecting all disease compartments.

2.1.3 Methodological Advances in Stochastic Epidemiology

The mathematical foundation for our approach draws from [8], whose work on stochastic calculus with respect to semimartingales provides the theoretical basis for Itô's lemma applications in our moment analysis. Their rigorous treatment of stochastic integration enables our derivation of mean and variance equations for each compartment.

[11] advanced stability analysis for stochastic epidemic models, establishing conditions under which disease-free equilibria remain stable under perturbation. Their framework informs our stability proofs for both deterministic and stochastic reproduction numbers, particularly the critical threshold $R_0^S < 1$ for disease eradication.

Recent work by [20] incorporated parameter perturbations into HIV models, demonstrating how stochasticity in recruitment and transmission rates affects long-term disease behavior. Their findings support our inclusion of noise terms in the susceptible recruitment and infection processes.

2.1.4 Integration of Legal and Epidemiological Perspectives

The synthesis of legal analysis and mathematical modeling remains underdeveloped in existing literature. While [4] provides comprehensive legal critique and [6] offers sophisticated mathematical frameworks, few studies integrate these perspectives. Our research bridges this gap by developing stochastic models that can inform legal and policy discussions about HIV criminalization.

[16] established the virological basis for modern HIV treatment, demonstrating how antiretroviral therapy suppresses viral loads to undetectable levels. This scientific reality, which renders sexual transmission effectively impossible, fundamentally challenges the premises of many HIV criminalization laws yet remains inadequately incorporated into existing mathematical models.

[**?**] recently applied fractional order calculus to HIV/AIDS modeling, capturing memory effects in disease progression. While their approach differs from our SDE framework, their findings about complex temporal dynamics reinforce the need for sophisticated mathematical tools in HIV research.

Stochastic models offer distinct advantages by generating probability distributions of potential outcomes (e.g., infected cell counts at time t), whereas deterministic approaches yield single-point predictions. This makes stochastic outputs more biologically meaningful [11]. Epidemiologically, investigating stochastic system properties – includ-

ing global stability, stochastic permanence, and noise's impact on established results – becomes crucial for understanding disease dynamics.

Modern stochastic differential equation techniques have enabled researchers to incorporate noise into epidemic modeling, whether through system noise or parameter fluctuations. Notably, studies [15,16] demonstrate noise's dual role: while potentially destabilizing systems, it can also stabilize populations by preventing unbounded growth, proving even minimal noise can avert population explosions in dynamic systems.

The influence of stochasticity in epidemic modeling has been explored from multiple perspectives. [20] introduced stochastic effects via parameter perturbations in an HIV model, while [28] demonstrated the stochastic asymptotic stability of endemic equilibrium for a perturbed two-group SIR system. [11] compared the stability of disease-free equilibria in both deterministic and stochastic SIR frameworks. Together, these studies underscore how environmental noise significantly alters epidemic behavior, with stochastic models offering more realistic dynamics than their deterministic counterparts [11]. A simple stochastic epidemic model is developed by considering a Susceptible and infective class of individuals. The susceptible (S) individuals changes their classification after being in contact with infected (I) individuals at a rate β and total population N. Considering the process to being homogeneous, the probability of the infection during the time interval $[t, t + \Delta t]$ is given by;

$$P[(S_{t+\Delta t}, I_{t+\Delta t})] - (S_t, I_t) = \beta \frac{S_t I_t}{N} \Delta t + \epsilon \Delta t$$

with the complimentary probability

$$P[(S_{t+\Delta t}, I_{t+\Delta t}) - (S_t, I_t)] = 1 - \beta \frac{S_t I_t}{N} \Delta t + \epsilon \Delta t$$

where $\epsilon \Delta t$ is the error term.

These model analyses the stochastic processes that occur in individuals moving from susceptible compartment to that of infective compartment only [20].

Gyongy and Krylov in [6] developed the HIV transmission dynamics model representing the progression from HIV+ status to AIDS. They divided the population into categories of progressive infectious stages. The model translated the movements of the stages into various equations in deterministic case and state transition probabilities in the stochastic case. Their model is as shown below;

$$\begin{aligned} \dot{S}(t) &= Q_0 - \beta_m S - \mu S, \\ \dot{I}_1(t) &= \beta_m S - (\theta + \delta + \mu) I_1, \\ \dot{I}_2(t) &= \theta I_1 - (\pi + \delta + \mu) I_2, \\ \dot{A}(t) &= \delta I_1 + \delta I_2 + \pi I_2 - (\alpha + \mu) A, \end{aligned} \tag{2.1}$$

where β_m is the probability of getting infected after being in contact with an infected individual. The control $u_1 \in [0, 1]$ is the successful use of condom by susceptibles while protecting themselves. The parameter θ measures the rate at which unaware infectives are detected by screening to become aware infectives, π measures the progression rate on already aware individuals to the AIDS class, the parameter δ represents the progression rate from untreated HIV infection to AIDS for both infectious classes, while α denotes the mortality rate attributable to AIDS-related complications. Analysis of (2.1) shows that there exists a stable disease free equilibrium and endemic equilibrium. Although the model captures the condition for effective control. This led [6] to the development of stochastic differential equations (SDE) models.

Hyman and Jia [18] proposed the Differential infectivity (DI) model accounting for the infectiousness differences and correlation between individuals during the stages and the viral loads with the rates of developing AIDS. The assumptions of the model included that the susceptible population was homogeneous thus neglecting variations in susceptibility, risk behavior as well as other factors associated with the dynamics of HIV spread. They studied a small population a subset of a high risk larger population. The population was then divided as susceptible individuals S, the HIV infection population

I, which was subdivided into n subgroups, $I_1, I_2, ..., I_n$, and the group of AIDS patients A. Their model was as shown below;

$$\begin{aligned}
\frac{dS}{dt} &= \mu S^0 - \mu S - \sum_{j=1}^{n} \beta_j I_j S, \\
\frac{dI_k}{dt} &= p_k \sum_{j=1}^{n} \beta_j I_j S - (\mu + \gamma_k) I_k, k = 1, 2, ..., n, \\
\frac{dA}{dt} &= \sum_{k=1}^{n} \gamma_k I_k - \delta A. \qquad (2.2)
\end{aligned}$$

Where S^0 presents a constant steady state of the susceptible population S, when no virus is in the population. μ is the rate of inflow and outflow, which maintains the equilibrium S^0. p_k is the probability of an individual enters subgroup k, when he is infected, where $\sum_k = 1^n p_k = 1$. γ_k is the rate of leaving the high-risk population because of behavior changes that are induced by either HIV-related illnesses or a positive HIV test. Lastly, δ is the die rate of A which satisfies $\delta \geq \mu$.

They assumed the rate of infection, depended upon the transmission probability per partner β_k of individuals in subgroup k, the proportion of individuals in the subgroup I_k, and the number of contacts of an individual per unit of time, $r(N)$, which was supposed $r(N) = N$.

Analysis of the model accounts for the differences in infectiousness between individuals during the chronic stages, and the correlation between viral loads and rates of developing AIDS. They considered the recruitment of the individuals into the susceptible class as being constant and in reality it is considered to be stochastic in nature. Hence, the need of development of a stochastic model that captures the constant recruitment rate of susceptibles in a heterosexual population.

Model (2.2) was improved by [2] by taking the account the effect of randomly fluctuating environment in system (2.1) and incorporating white noise effect. They obtained the

following stochastic equation;

$$\begin{aligned} dS &= (\mu S^0 - \mu S - \sum_{j=1}^{n} \beta_j I_j S)dt + \sigma_1 S dB_1(t), \\ dI_k &= [p_k \sum_{j=1}^{n} \beta_j I_j S - (\mu + \gamma_k) I_k]dt + \sigma_{k+1} I_k dB_{k+1}(t), k = 1, 2, \ldots, n, \\ dA &= (\sum_{k=1}^{n} \gamma_j I_j - \delta A)dt + \sigma_{n+2} A dB_{n+2}(t). \end{aligned} \tag{2.3}$$

where $B_k(t), k = 1, 2, ..., n + 2$, are also independent standard brownian motions and $\sigma_k > 0, k = 1, 2, \ldots, n + 2$, represent the intensities of $B_k(t), k = 1, 2, \ldots, n + 2$, respectively.

Analysis of equation (2.3) shows that the disease free equilibrium is stochastically asymptotically stable whenever $R_0 < 1$, while the endemic equilibrium is stochastically asymptotically stable whenever $R_0 > 1$.

The existing literature reveals several critical gaps that our research addresses:

(i) Limited integration of stochastic methods with heterosexual-specific transmission dynamics

(ii) Inadequate mathematical representation of how environmental variability affects legal risk assessments

(iii) Sparse analysis of how noise-induced fluctuations in disease prevalence might inform proportionality in criminal sentencing

(iv) Few studies connecting quantitative transmission risks with legal frameworks of intent and culpability

Our stochastic modeling approach addresses these gaps by providing quantitative tools to assess actual transmission risks, which can inform more principled legal approaches

to HIV criminalization. By deriving both deterministic R_0 and stochastic R_0^S reproduction numbers, we offer frameworks that better reflect the complex, variable nature of real-world HIV transmission dynamics.

The integration of Markov chain methods with SDE approaches further enables analysis of both continuous dynamics and discrete state transitions, providing comprehensive tools for understanding HIV spread in heterosexual populations under uncertainty. This methodological innovation supports the call by [4] for legal reforms grounded in scientific reality rather than outdated fears and misconceptions about HIV transmission.

CHAPTER THREE

MODEL FORMULATION AND ANALYSIS

3.1 Introduction

In this chapter SDEs HIV/AIDS model has been developed to investigate the dynamics of HIV/AIDs in heterosexual population. The model illustrates the transition of individuals between various compartment in a heterosexual population. The probability distribution of the formulated model was estimated using a Markov Chain Monte Carlo (MCMC) approach.

3.2 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 Λ. Natural mortality rate is given by μ. The probability of transmission of the virus is given by $\beta = \frac{\tau c}{N(t)}$, where τ 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 δ and α is the rate at which individuals die as a result of AIDS. σ_1, σ_2, σ_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 3.1.

Table 3.1: **Model descriptions**

Parameters	**Model parameters descriptions**
Λ	Natural recruitment rate to susceptible class
β	Rate at which the virus is transmitted
τ	The rate at of HIV transmission per sexual act.
c	sexual partnership rate between susceptible and infectious
δ	Rate at which infected individuals transit from HIV to AIDS
α	the rate at which individuals die as a result of AIDS.
σ_i	Represents noise intensities random fluctuations
μ	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 ;

$$N(t) = S(t) + I(t) + A(t) \tag{3.1}$$

This implies that the first derivative is given by;

$$dN(t) = [dS(t) + dI(t) + dA(t)]dt \tag{3.2}$$

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

$$\begin{aligned} dS(t) &= [\Lambda - \beta SI - \mu S]\, dt + \sigma_1 S\, dB_1(t), \\ dI(t) &= [\beta SI - (\mu + \delta)I]\, dt + \sigma_2 I\, dB_2(t), \\ dA(t) &= [\delta I - (\mu + \alpha)A]\, dt + \sigma_3 A\, dB_3(t), \end{aligned} \tag{3.3}$$

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:

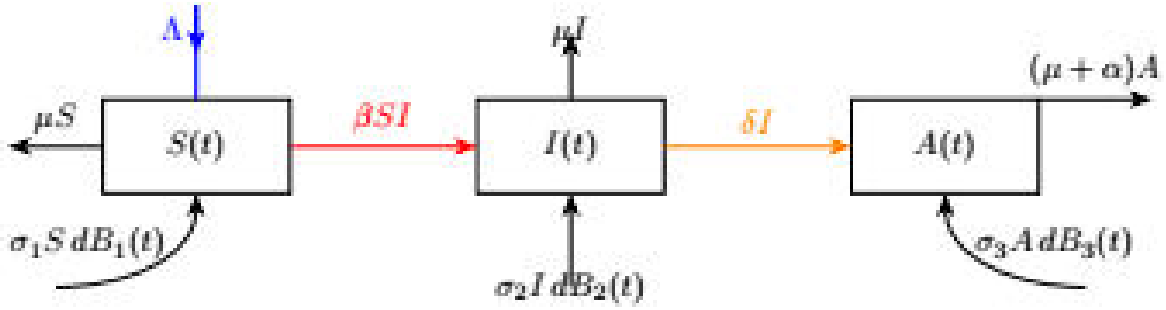

Figure 3.1: SIA Schematic diagram

The formulation of the model (3.1) was developed based on the following assumptions:

(i) HIV/AIDS only spread through heterosexual contact by assuming a single transmission route (heterosexual) to avoid the complexity of modeling multiple transmission pathways such as intravenous drug use, mother-to-child, or men who have sex with men.

(ii) Individuals who have progressed to AIDS can not transmit the virus since they are sexually inactive. By excluding AIDS-driven transmission avoids additional nonlinear terms in the equations, making stability and analysis more feasible.

3.3 Model Analysis

3.3.1 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.

Mean of Susceptible Compartment S(t)

Starting from the the first equation of the SDE system (3.3) given by:

$$dS = (\Lambda - \beta SI - \mu S)dt + \sigma_1 S dB_1(t) \tag{3.4}$$

Apply Itô's lemma [19] to $f(S) = S$:

$$df(S) = \frac{\partial f}{\partial S} dS + \frac{1}{2} \frac{\partial^2 f}{\partial S^2} (dS)^2 \tag{3.5}$$

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

$$E[dS] = E[(\Lambda - \beta SI - \mu S)dt] + E[\sigma_1 S dB_1(t)] \tag{3.6}$$

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

$$\frac{dm_S}{dt} = \Lambda - \beta E[SI] - \mu m_S \tag{3.7}$$

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

The mean susceptible population dynamics are governed by three key components. The recruitment term (Λ) represents a constant inflow of new susceptible individuals into the population, modeling either birth processes or migration. Unlike density-dependent growth models, this term remains independent of the current population size, reflecting an open population system where new susceptibles enter at a fixed rate regardless of existing numbers.

The infection term ($-\beta E[SI]$) captures the non-linear nature of HIV transmission dynamics. This crucial component involves the expectation of susceptible-infected interactions ($E[SI]$), which decomposes into $m_S m_I + \text{Cov}(S, I)$, revealing that disease transmission depends not only on mean population levels but also on the synchronization between susceptible and infected populations through their covariance. The covariance term accounts for important spatial and temporal correlations in disease spread that would be neglected in simple mean-field approximations.

The mortality term ($-\mu m_S$) introduces exponential decay representing natural deaths in the population. This linear term acts as a stabilizing factor that prevents unbounded

growth of the susceptible population, ensuring the model remains biologically realistic. The mortality rate μ serves as a balancing force against the recruitment term, with their relative magnitudes determining the baseline susceptible population level in the absence of disease.

Variance of Susceptible Compartment S(t)

Using Itô's lemma on $f(S) = S^2$:

$$d(S^2) = 2SdS + (dS)^2 \tag{3.8}$$

Substituting dS and taking expectations:

$$E[d(S^2)] = 2E[S(\Lambda - \beta SI - \mu S)]dt + \sigma_1^2 E[S^2]dt \tag{3.9}$$

$$= [2\Lambda m_S - 2\beta E[S^2 I] - 2\mu E[S^2] + \sigma_1^2 E[S^2]]dt \tag{3.10}$$

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

$$\frac{dV_S}{dt} = \sigma_1^2(V_S + m_S^2) + 2\Lambda m_S - 2\beta E[S^2 I] - 2\mu(V_S + m_S^2) - 2m_S\frac{dm_S}{dt} \tag{3.11}$$

After simplification:

$$\frac{dV_S}{dt} = \sigma_1^2 m_S^2 - 2\mu V_S - 2\beta(E[S^2 I] - m_S E[SI]) \tag{3.12}$$

The variance dynamics of the susceptible population reveal three fundamental aspects of stochastic disease spread. The noise term ($\sigma_1^2 m_S^2$) captures demographic stochasticity, showing that random fluctuations scale quadratically with the mean susceptible population. This nonlinear relationship implies that while larger populations experience greater absolute variability in susceptibility numbers, the relative variability (coefficient of variation) actually decreases with population size.

The transmission terms ($-2\beta(E[S^2 I] - m_S E[SI])$) encode the complex stochastic interactions between susceptible and infected populations. These terms generate variability through the infection process, with the third-order moments ($E[S^2 I]$) reflecting how

disease transmission depends on both the mean densities and the spatial clustering of susceptible and infected individuals. The mathematical structure necessitates moment closure approximations, as the full dynamics depend on higher-order correlations in the system.

The damping term ($-2\mu V_S$) provides a stabilizing mechanism, showing how natural mortality consistently reduces population variability over time. This term acts as a "variance sink" that opposes the noise-driven fluctuations, with the mortality rate μ determining the characteristic timescale for variance dissipation. The factor of 2 appears because variance depends on squared deviations, making the damping effect twice as strong as for the mean population.

Mean of Infected Compartment I(t)

From the SDE system (3.3):

$$dI = (\beta SI - (\mu + \delta)I)dt + \sigma_2 I dB_2(t) \tag{3.13}$$

Following similar steps as used in (3.4) to (3.7) yields:

$$\frac{dm_I}{dt} = \beta E[SI] - (\mu + \delta)m_I \tag{3.14}$$

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 (μ) affects all population compartments equally, while the disease progression rate (δ) 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 δ serves as a critical parameter linking the infected and AIDS compartments in the model's disease staging framework.

Variance of Infected Compartment I(t)

Similarly for I^2:

$$\frac{dV_I}{dt} = \sigma_2^2 m_I^2 - 2(\mu + \delta)V_I + 2\beta(E[SI^2] - m_I E[SI]) \tag{3.15}$$

The variance dynamics of the infected population exhibit competing stochastic effects that shape outbreak patterns. The positive feedback term $(2\beta(E[SI^2] - m_I E[SI]))$ demonstrates how the infection process intrinsically amplifies variability through nonlinear transmission dynamics. This amplification mechanism arises from the multiplicative nature of disease spread, where simultaneous fluctuations in both susceptible and infected populations can reinforce each other, potentially leading to explosive growth in variability during emerging outbreaks. The term's structure reveals that this variability amplification depends on higher-order correlations between susceptible and infected individuals, captured through the third-order moment $E[SI^2]$, which quantifies how spatial clustering or heterogeneity in contact patterns affects transmission variability.

The enhanced damping term $(-2(\mu + \delta)V_I)$ provides a counterbalancing force that is markedly stronger than the corresponding term in the susceptible population. The additional δ term reflects how progression to AIDS actively removes individuals from the infected compartment, serving as an extra source of variability reduction beyond natural mortality. This dual damping effect means that while the infected population may experience rapid variability growth during transmission events, the combined forces of natural mortality and disease progression work to constrain this variability more effectively than in other compartments. The factor of 2 again emerges from the quadratic

nature of variance, indicating that these damping processes operate more strongly on variability than on mean population levels.

Mean of Aids Compartment A(t)

From the SDE system (3.3):

$$dA = (\delta I - (\mu + \alpha)A)dt + \sigma_3 A dB_3(t) \tag{3.16}$$

Similarly applying Ito's lemma [19]; Yielding:

$$\frac{dm_A}{dt} = \delta m_I - (\mu + \alpha)m_A \tag{3.17}$$

The mean AIDS population dynamics are governed by two opposing processes. The inflow term (δm_I) establishes a direct epidemiological coupling with the infected compartment, representing the progression rate from HIV infection to AIDS. This term reflects the clinical reality that individuals in the infected compartment will eventually develop AIDS at rate δ, creating a unidirectional flow through the disease stages. The progression rate δ determines the average time spent in the HIV-positive stage before developing AIDS, with $1/\delta$ representing the mean duration of the pre-AIDS infection period.

The mortality term ($-(\mu + \alpha)m_A$) combines two distinct exit pathways from the AIDS population. While μ represents baseline mortality affecting all compartments equally, the AIDS-specific mortality rate α typically dominates ($\alpha \gg \mu$), reflecting the significantly higher fatality rate in this advanced disease stage. The combined coefficient $(\mu + \alpha)$ determines the rapidity of population turnover in the AIDS compartment, with the large magnitude of α explaining the characteristically short survival time post-AIDS diagnosis in untreated cases. This term captures the terminal nature of the AIDS stage in the model's disease progression framework.

Variance of AIDS Compartment A(t)

For A^2:

$$\frac{dV_A}{dt} = \sigma_3^2 m_A^2 - 2(\mu + \alpha)V_A + 2\delta(E[IA] - m_A m_I) \tag{3.18}$$

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.

3.3.2 Positivity of Solutions of the Model

Since model (3.1) describes human population, it should be well posed. Thus in this section, positivity and boundedness solutions of model (3.1) are discussed.

Theorem 3.3.1. *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 $t \geq 0$.*

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

$$dS(t) = [\Lambda - \beta S(t)I(t) - \mu S(t)]\, dt + \sigma_1 S(t) dB_1(t) \tag{3.19}$$

This can be rewritten as:

$$\frac{dS(t)}{S(t)} = \left[\frac{\Lambda}{S(t)} - \beta I(t) - \mu\right] dt + \sigma_1 dB_1(t) \tag{3.20}$$

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

$$d(\ln X_t^i) = \left(\frac{f_i(X_t)}{X_t^i} - \frac{g_i^2(X_t)}{2(X_t^i)^2}\right) dt + \frac{g_i(X_t)}{X_t^i} dW_t \tag{3.21}$$

to $\ln S(t)$ gives:

$$d(\ln S(t)) = \frac{dS(t)}{S(t)} - \frac{1}{2}\frac{(dS(t))^2}{S(t)^2} \tag{3.22}$$

Substituting and simplifying:

$$d(\ln S(t)) = \left[\frac{\Lambda}{S(t)} - \beta I(t) - \mu - \frac{\sigma_1^2}{2}\right] dt + \sigma_1 dB_1(t) \tag{3.23}$$

Integrating from 0 to t:

$$\ln S(t) - \ln S(0) = \int_0^t \left[\frac{\Lambda}{S(s)} - \beta I(s) - \mu - \frac{\sigma_1^2}{2}\right] ds + \sigma_1 B_1(t) \tag{3.24}$$

Exponentiating both sides:

$$S(t) = S(0)\exp\left(\int_0^t \left[\frac{\Lambda}{S(s)} - \beta I(s) - \mu - \frac{\sigma_1^2}{2}\right] ds + \sigma_1 B_1(t)\right) > 0 \tag{3.25}$$

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

For infected compartment $I(t)$: The SDE for $I(t)$ is:

$$dI(t) = [\beta S(t)I(t) - (\mu + \delta)I(t)]\, dt + \sigma_2 I(t) dB_2(t) \tag{3.26}$$

Applying Itô's formula to $\ln I(t)$:

$$d(\ln I(t)) = \left[\beta S(t) - (\mu + \delta) - \frac{\sigma_2^2}{2}\right] dt + \sigma_2 dB_2(t) \tag{3.27}$$

Integrating and exponentiating:

$$I(t) = I(0)\exp\left(\int_0^t \left[\beta S(s) - (\mu + \delta) - \frac{\sigma_2^2}{2}\right] ds + \sigma_2 B_2(t)\right) > 0 \tag{3.28}$$

Also the AIDS compartment $(A(t))$:

The SDE for $A(t)$ is:

$$dA(t) = [\delta I(t) - (\mu + \alpha)A(t)]\, dt + \sigma_3 A(t) dB_3(t) \tag{3.29}$$

Applying the same method:

$$d(\ln A(t)) = \left[\frac{\delta I(t)}{A(t)} - (\mu + \alpha) - \frac{\sigma_3^2}{2}\right] dt + \sigma_3 dB_3(t) \tag{3.30}$$

Integrating and exponentiating:

$$A(t) = A(0)\exp\left(\int_0^t \left[\frac{\delta I(s)}{A(s)} - (\mu + \alpha) - \frac{\sigma_3^2}{2}\right] ds + \sigma_3 B_3(t)\right) > 0 \tag{3.31}$$

□

The analysis demonstrates that the solutions $S(t)$, $I(t)$, and $A(t)$ of the stochastic model (3.1) 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.

3.3.3 Boundedness of Solutions of the Model

Proposition 3.3.1. *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 positive and bounded in $\in \mathbb{R}^3$ for all $t \in [0, t_0]$.*

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

$$dN(t) = dS(t) + dI(t) + dA(t) \tag{3.32}$$

Substituting the model equations (3.3) in (3.2) we obtain:

$$dN(t) = [\Lambda - \mu N(t) - \alpha A(t)]\, dt + \sigma_1 S(t) dB_1(t) + \sigma_2 I(t) dB_2(t) + \sigma_3 A(t) dB_3(t) \tag{3.33}$$

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

$$\frac{d\mathbb{E}[N(t)]}{dt} = \Lambda - \mu \mathbb{E}[N(t)] - \alpha \mathbb{E}[A(t)] \leq \Lambda - \mu \mathbb{E}[N(t)] \tag{3.34}$$

This is a differential inequality of the form:

$$\frac{d\mathbb{E}[N(t)]}{dt} + \mu \mathbb{E}[N(t)] \leq \Lambda \tag{3.35}$$

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

$$\frac{d}{dt}\left(\mathbb{E}[N(t)] e^{\mu t}\right) \leq \Lambda e^{\mu t} \tag{3.36}$$

Integrating both sides from 0 to t:

$$\mathbb{E}[N(t)] e^{\mu t} - N(0) \leq \frac{\Lambda}{\mu}(e^{\mu t} - 1) \tag{3.37}$$

Rearranging:

$$\mathbb{E}[N(t)] \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}) \leq \max\left(N(0), \frac{\Lambda}{\mu}\right) \tag{3.38}$$

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

$$\lim_{t\to\infty} \mathbb{E}[N(t)] \leq \frac{\Lambda}{\mu} \tag{3.39}$$

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 Λ represents the constant recruitment rate and μ 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 $A(t) = 0$.

□

3.4 Determination of the Basic Reproduction Number

Definition 1. *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.*

Theorem 3.4.1. *Consider a compartmental epidemiological model with n infected compartments. Let $F_i(x)$ be the rate of appearance of new infections in compartment i, and $V_i(x) = V_i^-(x) - V_i^+(x)$ where $V_i^+(x)$ is the rate of transfer into compartment i by all other means and $V_i^-(x)$ is the rate of transfer out of compartment i.*
Define the $n \times n$ matrices:

$$F = \left[\frac{\partial F_i}{\partial x_j}(x_0)\right] \quad \textit{and} \quad V = \left[\frac{\partial V_i}{\partial x_j}(x_0)\right]$$

where x_0 is the disease-free equilibrium.

Then the basic reproduction number R_0 is given by the spectral radius of the next generation matrix:

$$R_0 = \rho(FV^{-1})$$

where $\rho(J)$ denotes the spectral radius of matrix J.

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

$$\text{DFE} = (S^0, I^0, A^0) = \left(\frac{\Lambda}{\mu}, 0, 0\right) \tag{3.40}$$

This is obtained by solving:

$$\begin{cases} \Lambda - \beta S^0 I^0 - \mu S^0 = 0 & \Rightarrow S^0 = \dfrac{\Lambda}{\mu} \\ \beta S^0 I^0 - (\mu + \delta) I^0 = 0 & \Rightarrow I^0 = 0 \\ \delta I^0 - (\mu + \alpha) A^0 = 0 & \Rightarrow A^0 = 0 \end{cases} \tag{3.41}$$

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 (by assumption iii), we only consider I.

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

$$\frac{dI}{dt} = \underbrace{\beta S I}_{\mathcal{F}} - \underbrace{(\mu + \delta) I}_{\mathcal{V}} \tag{3.42}$$

Thus:

$$\mathcal{F} \quad = \beta S I \tag{3.43}$$

$$\mathcal{V} \quad = (\mu + \delta) I \tag{3.44}$$

By linearization system (3.1) at DFE, we compute the Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ with respect to I and evaluate at the DFE:

$$F = \left.\frac{\partial \mathcal{F}}{\partial I}\right|_{\text{DFE}} = \beta S^0 = \beta \frac{\Lambda}{\mu} \tag{3.45}$$

$$V = \left.\frac{\partial \mathcal{V}}{\partial I}\right|_{\text{DFE}} = \mu + \delta \tag{3.46}$$

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:

$$R_0 = \frac{F}{V} = \frac{\beta \frac{\Lambda}{\mu}}{\mu + \delta} \tag{3.47}$$

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

$$R_0 = \frac{\tau c \frac{\Lambda}{\mu} \frac{1}{\frac{\Lambda}{\mu}}}{\mu + \delta} = \frac{\tau c}{\mu + \delta} \tag{3.48}$$

The basic reproduction number R_0 can be interpreted as: τc - The product of transmission probability τ 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 μ and progression to AIDS - δ.

To derive the stochastic R_0^S, we use the approach from [28], 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.

□

3.5 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.

$$I(t) = 0 \quad \text{and} \quad A(t) = 0 \quad \text{for all} \quad t \geq 0 \tag{3.49}$$

The Disease-Free Equilibrium (DFE) is obtained by setting the infected compartments to zero ($I = 0$, $A = 0$).

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

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

3.5.1 Equilibrium Distribution of $S(t)$

The mean of $S(t)$ at equilibrium is:

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

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;

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

3.5.2 Stochastic Stability Analysis at DFE

Theorem 3.5.1. *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 (3.1) 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:

$$\begin{aligned} dX_1 &= \left(-\mu X_1 - \beta \frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu-\sigma_1^2)} X_2\right) dt + \sigma_1 \frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu-\sigma_1^2)} dB_1(t) \\ dX_2 &= \left(\beta \frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu-\sigma_1^2)} - \mu - \delta\right) X_2 dt + \sigma_2 \cdot 0 \cdot dB_2(t) \\ dX_3 &= (\delta X_2 - (\mu+\alpha)X_3)\, dt + \sigma_3 \cdot 0 \cdot dB_3(t) \end{aligned} \tag{3.50}$$

where $X = (S - S^0, I - I^0, A - A^0)$.

Considering the quadratic Lyapunov function [5]:

$$V(X) = \frac{1}{2}\left(X_1^2 + X_2^2 + X_3^2\right) \tag{3.51}$$

Applying Itô's formula [8]:

$$dV = \mathcal{L}V\, dt + \sigma_1 \frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu-\sigma_1^2)} X_1 dB_1(t) \tag{3.52}$$

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:

$$\mathcal{L}V \leq -\mu X_1^2 + (\beta \frac{\Lambda^2\sigma_1^2}{\mu^2(2\mu-\sigma_1^2)} - \mu - \delta)X_2^2 - (\mu+\alpha)X_3^2 + \text{higher order terms} + \text{noise term} \tag{3.53}$$

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

The stochastic DFE is:

$$\text{DFE}^S = E_0 = \left(\frac{\Lambda^2 \sigma_1^2}{\mu^2(2\mu - \sigma_1^2)}, 0, 0 \right),$$

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 yields several important insights about the disease-free equilibrium (DFE). When the basic reproduction number satisfies $R_0^S < 1$, the DFE demonstrates stability in both deterministic and stochastic frameworks. However, the stochastic stability requires an additional condition that the noise intensity σ_1 remains below a certain threshold. This reveals that the stability conditions are inherently more restrictive in the stochastic case compared to its deterministic counterpart, as fluctuations introduce additional constraints on the system's behavior.

The analysis implies that the deterministic model, E_0 is locally asymptotically stable when $R_0^S < 1$. For the stochastic model, stability requires both $R_0^S < 1$ and sufficiently small noise intensities. Finally, when R_0^S exceeds 1, the equilibrium becomes unstable in both cases, indicating that the disease may become endemic in the population. □

3.6 Stochastic Endemic Equilibrium Analysis

Theorem 3.6.1. *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 (3.3) at EE, let $(x, y, z) = (S - S^*, I - I^*, A - A^*)$. The linearized system is:

$$\begin{cases} dx = \big[-\beta(I^*x + S^*y) - \mu x \big] dt + \sigma_1 S^* dB_1(t) \\ dy = \big[\beta(I^*x + S^*y) - (\mu + \delta)y \big] dt + \sigma_2 I^* dB_2(t) \\ dz = \big[\delta y - (\mu + \alpha)z \big] dt + \sigma_3 A^* dB_3(t) \end{cases} \tag{3.54}$$

Consider the quadratic Lyapunov function:

$$V(x, y, z) = \frac{1}{2} \left(ax^2 + by^2 + cz^2 \right) \tag{3.55}$$

where $a, b, c > 0$ are constants to be determined.

Applying Itô's formula:

$$dV = \mathcal{L}V\,dt + \text{noise terms} \tag{3.56}$$

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:

$$\sigma_1^2 < \frac{2\mu}{S^*}, \quad \sigma_2^2 < \frac{2(\mu + \delta)}{I^*}, \quad \sigma_3^2 < \frac{2(\mu + \alpha)}{A^*} \tag{3.57}$$

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 disease will persist at stable endemic levels.

3.7 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 2. *The Markov chain is defined on the discrete state space:*

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

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 3. *We consider discrete time steps:*

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

where Δt *is chosen sufficiently small to ensure only one transition can occur per time step.*

Proof. The state space $\mathcal{S} = \{(s, i, a) \in \mathbb{Z}_+^3 \,|\, s + i + a \leq N_{max}\}$ is well-defined as a discrete Markov chain state space for the following reasons:
Since $\mathbb{Z}_+^3$ represents all triples of non-negative integers, and N_{max} is finite, the state space $\mathcal{S}$ is finite and countable. Specifically:

$$|\mathcal{S}| = \sum_{n=0}^{N_{max}} \binom{n+2}{2} = \binom{N_{max}+3}{3} < \infty$$

This follows from the stars and bars combinatorial formula for the number of non-negative integer solutions to $s + i + a \leq N_{max}$.
Each state (s, i, a) corresponds to a physically realizable population configuration:

(i) $s, i, a \geq 0$ ensures non-negative population counts

(ii) $s + i + a \leq N_{max}$ imposes a realistic carrying capacity

(iii) The compartments maintain their epidemiological meaning throughout transitions

The state space captures all relevant information for future evolution since; the future dynamics depend only on current compartment sizes, no historical information beyond current counts is needed and all possible epidemiological states are represented
Thus, $\mathcal{S}$ satisfies all requirements for a Markov chain state space. □

Proof. The condition $\Delta t < \frac{1}{\max_{(s,i,a)\in\mathcal{S}} Q(s,i,a)}$ ensures the discrete-time approximation validity for the continuous-time Markov chain.
Let $Q(s,i,a)$ be the total transition rate from state (s,i,a). From the transition structure, we have:

$$Q(s,i,a) = \Lambda + \beta si + \mu s + \mu i + \delta i + (\mu + \alpha)a$$

Since $\mathcal{S}$ is finite and all parameters are finite, there exists:

$$Q_{max} = \max_{(s,i,a)\in\mathcal{S}} Q(s,i,a) < \infty$$

Specifically, the maximum occurs when all compartments are at their maximum possible values subject to $s + i + a \leq N_{max}$.
For a continuous-time Markov chain, the probability of more than one transition in time interval Δt is $o(\Delta t)$. By choosing:

$$\Delta t < \frac{1}{Q_{max}}$$

we ensure that for any state (s,i,a):

$$\mathbb{P}(\text{more than one transition in } \Delta t) \leq 1 - e^{-Q(s,i,a)\Delta t} - Q(s,i,a)\Delta t e^{-Q(s,i,a)\Delta t}$$

Using the Taylor expansion $e^{-x} = 1 - x + \frac{x^2}{2} - \cdots$, we get:

$$\mathbb{P}(\text{more than one transition}) \leq \frac{(Q(s,i,a)\Delta t)^2}{2} + O((Q(s,i,a)\Delta t)^3)$$

Since $Q(s,i,a)\Delta t < Q_{max}\Delta t < 1$, this probability is bounded by $O(\Delta t^2)$.
The discrete-time transition probabilities approximate the continuous-time probabilities with error $O(\Delta t^2)$:

$$\mathbb{P}(\text{transition } j \text{ in } \Delta t) = r_j \Delta t + O(\Delta t^2)$$

where r_j are the individual transition rates.

This discretization is consistent with the tau-leaping approximation of the exact Gillespie algorithm, providing a computable discrete-time Markov chain that approximates the continuous-time process. □

The discrete-time process preserves the Markov property of the underlying continuous-time process.

Let $\mathcal{F}_t$ be the filtration generated by the process up to time t. For the discrete-time process at times $t_k = k\Delta t$, we need to show:

$$\mathbb{P}(\mathbf{X}(t_{k+1}) = \mathbf{y} \,|\, \mathcal{F}_{t_k}) = \mathbb{P}(\mathbf{X}(t_{k+1}) = \mathbf{y} \,|\, \mathbf{X}(t_k))$$

The original continuous-time process satisfies:

$$\mathbb{P}(\mathbf{X}(t+s) = \mathbf{y} \,|\, \mathcal{F}_t) = \mathbb{P}(\mathbf{X}(t+s) = \mathbf{y} \,|\, \mathbf{X}(t))$$

For the sampled process at discrete times t_k, the Markov property is inherited because:

$$\mathbb{P}(\mathbf{X}(t_{k+1}) = \mathbf{y} \,|\, \mathcal{F}_{t_k}) = \mathbb{P}(\mathbf{X}(t_k + \Delta t) = \mathbf{y} \,|\, \mathcal{F}_{t_k}) = \mathbb{P}(\mathbf{X}(t_k + \Delta t) = \mathbf{y} \,|\, \mathbf{X}(t_k))$$

The discrete-time transition probabilities are determined solely by the current state:

$$P_{\Delta t}(\mathbf{x}, \mathbf{y}) = \mathbb{P}(\mathbf{X}(t + \Delta t) = \mathbf{y} \,|\, \mathbf{X}(t) = \mathbf{x})$$

and depend only on $\mathbf{x}$, not on the history before time t.

Therefore, the discrete-time process $\{\mathbf{X}(t_k)\}_{k=0}^{\infty}$ is a Markov chain. □

The developed stochastic framework provides a rigorous mathematical foundation for analyzing HIV/AIDS dynamics under demographic stochasticity. Key conclusions include; the state space $\mathcal{S}$ is properly defined as a finite, discrete set capturing all epidemiologically relevant configurations, ensuring the Markov property is well-defined and preserved, the time discretization condition $\Delta t < 1/Q_{max}$ guarantees that the discrete-time approximation faithfully represents the continuous-time process while enabling practical computation and simulation, the framework correctly captures demographic stochasticity—the inherent randomness in individual-level events like infection

and progression—which is distinct from the environmental fluctuations modeled by SDE approaches, the discrete-state Markov chain approach maintains consistency with both the underlying continuous-time process and the deterministic model in the large population limit, while providing additional insights into small population effects and extinction probabilities and the finite state space enables computation of key epidemiological quantities such as stationary distributions, hitting probabilities, and mean first passage times, which are crucial for understanding long-term disease behavior and control strategies. This framework bridges the gap between deterministic models and individual-based simulations, providing a powerful tool for understanding the role of chance events in disease dynamics and for designing effective intervention strategies in the face of uncertainty.

3.7.1 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 3.7.1. *Theorem (Transition Probabilities with Noise) For $\Delta t \to 0$, the transition probabilities are:*

$$p_{(s,i,a)\to(s',i',a')} = \begin{cases} \Lambda\Delta t + o(\Delta t) & \textit{(Recruitment)} \\ \frac{\tau csi}{s+i+a}\Delta t + \sigma_1 si\, dB_1(t) + o(\Delta t) & \textit{(Infection with noise)} \\ \delta i\Delta t + \sigma_2 i\, dB_2(t) + o(\Delta t) & \textit{(Progression with noise)} \\ \mu s\Delta t + o(\Delta t) & \textit{(S death)} \\ \mu i\Delta t + \sigma_3 i\, dB_3(t) + o(\Delta t) & \textit{(I death with noise)} \\ (\mu+\alpha)a\Delta t + \sigma_4 a\, dB_4(t) + o(\Delta t) & \textit{(A death with noise)} \\ 1 - R_{tot}\Delta t + o(\Delta t) & \textit{(No change)} \end{cases} \tag{3.58}$$

where:

$$R_{tot} = \Lambda + \frac{\tau csi}{s+i+a} + \delta i + \mu(s+i) + (\mu+\alpha)a \tag{3.59}$$

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 $\sigma_1 si\, dB_1(t)$:

$$P_{\text{infection}} = \left(\frac{\tau csi}{s+i+a}\right)\Delta t + \sigma_1 si\,\Delta B_1(t) + o(\Delta t) \tag{3.60}$$

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

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

$$P_{\text{progression}} = \delta i \Delta t + \sigma_2 i\,\Delta B_2(t) + o(\Delta t) \tag{3.61}$$

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:

$$P_{\text{no change}} = 1 - \left[R_{tot}\Delta t + \sum_k \sigma_k(\text{noise contributions})\right] + o(\Delta t) \tag{3.62}$$

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:

$$R_0^S = \frac{\tau c}{\mu+\delta} - \frac{\sigma_2^2}{2(\mu+\delta)}, \tag{3.63}$$

Where the progression noise term σ_2 appears quadratically, demonstrating its significant damping effect on the basic reproduction number. The variability introduced by σ_2 in the progression rate δi affects the duration of the infectious period, as individuals transition to AIDS at stochastic rates rather than deterministic intervals, thereby modifying the disease's transmission potential.

Mortality dynamics are similarly influenced by noise terms: σ_3 introduces variability in the death rate of infected individuals (I), which can either shorten or lengthen the infectious period, while σ_4 affects AIDS-related mortality, altering the terminal stage duration and consequently the overall disease burden. These stochastic effects have crucial implications for disease control strategies.

When $R_0^S < 1$, noise can accelerate disease extinction by increasing variance in transmission events, while for $R_0^S > 1$, the same noise may either sustain endemic conditions or lead to unpredictable outbreak patterns due to the interaction between stochastic forcing and nonlinear transmission dynamics.

Several key observations emerge from this analysis. First, the explicit incorporation of environmental noise through terms $\sigma_k dB_k(t)$ in the transition probabilities provides a critical bridge between deterministic R_0 and its stochastic counterpart R_0^S. Second, these noise terms collectively modify both individual transition rates and the system's overall dynamics, mathematically justifying the variance correction term in R_0^S. Finally, while the frequency-dependent transmission mechanism $\left(\frac{\tau csi}{N}\right)$ remains fundamental to the model structure, the added noise terms enhance biological realism by accounting for the inherent randomness in human contact patterns and behavioral fluctuations that characterize real-world epidemic spread. □

3.7.2 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 3.7.2 (Stochastic Probability of Evolution)**.** *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 σ_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 (σ_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:

$$R_0^S = \frac{\tau c}{\mu+\delta} - \frac{1}{2(\mu+\delta)}\sum_{k=1}^{4}\sigma_k^2. \tag{3.64}$$

The progression noise (σ_2) alters the distribution of infectious periods by introducing stochasticity in the transition rate δ from infected to AIDS stages, effectively modifying the mean duration $(\mu + \delta)^{-1}$ of infectiousness. Mortality noises (σ_3 and σ_4) create variability in both the infected and AIDS stage durations, which impacts the time-integrated transmission potential through changes in the expected time spent in each disease state.

When $R_0^S < 1$, noise accelerates the system's convergence to disease-free equilibrium by increasing the variance in transmission events, effectively pushing probability mass toward extinction states ($\sigma_k = 0$). In the endemic case $R_0^S > 1$, the noise terms σ_k generate metastable disease persistence characterized by prevalence fluctuations around the deterministic equilibrium. The covariance structure between different noise processes $\xi_k(t)$ governs the statistical properties of outbreak sizes, with positive correlations between transmission and progression noises potentially amplifying outbreak variability.

Several important mathematical properties emerge from this analysis. The multiplicative form of noise terms in transition probabilities guarantees solution non-negativity, preserving the probabilistic interpretation of $p_{s,i,a}(t)$. The quadratic dependence on σ_2 in the R_0^S correction term arises naturally from the Itô-to-Stratonovich conversion when taking the continuous-time limit of the underlying stochastic process. While the frequency-dependent transmission mechanism $\tau csi/N$ maintains its basic form, the effective transmission rates become noise-adjusted through the interaction between deterministic parameters and stochastic terms. This provides a rigorous connection between individual-level stochastic transitions and population-level disease thresholds.

3.8 Stochastic Analysis with Noise Effects

The total transition rate incorporating noise terms becomes:

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

Theorem 3.8.1 (Noisy Inter-event Times)**.** *The waiting time distribution becomes:*

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

with density:

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

Proof. For small Δt, 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:

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

□

The modified transition probabilities reflect the adjusted reproduction number:

$$R_0^S = \frac{\tau c}{\mu + \delta} - \frac{1}{2(\mu+\delta)}\sum_{k=1}^4 \sigma_k^2 \tag{3.65}$$

The event selection probabilities become:

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

where:

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

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 $P^S(\text{infection}|\text{event}) = (\tau cSI/N + \sigma_1 SI\xi_1(t))/R^S_{tot}$, 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 $R^S_0 < 1$, 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 $R^S_0 > 1$, 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.

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

$$\frac{d}{dt}p_{s,i,a}(t) = \sum_{k=1}^{4} \left[\mathcal{L}_k + \sigma_k \mathcal{N}_k\right] p_{s,i,a}(t) \tag{3.66}$$

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

$$0 = \left(\sum_{k=1}^{4} \mathcal{L}_k + \frac{1}{2}\sum_{k=1}^{4} \sigma_k^2 \mathcal{N}_k^2\right) \pi^S_{s,i,a} \tag{3.67}$$

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

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

The noise terms modify the effective reproduction number given by:

$$R_0^S = \underbrace{\frac{\tau c}{\mu+\delta}}_{\text{Deterministic}} \underbrace{- \frac{\sigma_1^2 + \sigma_2^2 + \sigma_3^2}{2(\mu+\delta)}}_{\text{Noise correction}} \tag{3.68}$$

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

$$\frac{d}{dt}p = \underbrace{-\nabla\cdot(\mathbf{v}p)}_{\text{Drift}} + \underbrace{\frac{1}{2}\sum_{k=1}^{4}\sigma_k^2 \frac{\partial^2}{\partial x_k^2}p}_{\text{Diffusion}} \tag{3.69}$$

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

For reversible transitions:

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

Noise-induced extinction occurs when $R_0^S < 1$, as the additional variance terms accelerate convergence to the disease-free state $\pi^S_{s,0,0}$. 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:

$$I^* = \frac{\Lambda}{\mu}\left(1 - \frac{1}{R_0^S}\right) \pm \sqrt{\frac{\sum \sigma_k^2}{2(\mu + \delta)}} \tag{3.70}$$

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 τ_{eff} is given by:

$$\tau_{\text{eff}} = \tau - \frac{N}{2c}\sum_{k=1}^{4} \sigma_k^2 \tag{3.71}$$

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:

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

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.

CHAPTER FOUR

SIMULATION RESULTS

The stochastic HIV/AIDS model was simulated using the Euler-Maruyama method with varying noise intensities (σ_1, σ_2, σ_3) for the susceptible, infected, and AIDS compartments respectively. The simulation results (4.1) 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 4.1 and Figure 4.2.

Table 4.1: Parameter values used in numerical simulations

Parameter	**Description**	**Value**	**Source**
Λ	Recruitment rate	Calculated from equilibrium	[29]
β	Transmission rate	$\tau c/N$	[16]
τ	Transmission probability per contact	0.5	[6]
c	Mean sexual partnership rate	3.0	[9]
μ	Natural mortality rate	0.02	[36]
δ	HIV to AIDS progression rate	0.1	[29]
α	AIDS-related mortality rate	0.3	[36]
σ_1	Susceptible noise intensity	0.1	[28]
σ_2	Infected noise intensity	0.15	[2]
σ_3	AIDS noise intensity	0.1	[6]
$S(0)$	Initial susceptible population	100	Assumed
$I(0)$	Initial infected population	5	Assumed
$A(0)$	Initial AIDS population	0	Assumed

4.1 Simulation Results of HIV/AIDS Model with Noise

From Figure 4.1, 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 de-

creasing 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 Λ balances losses from infection and natural mortality.

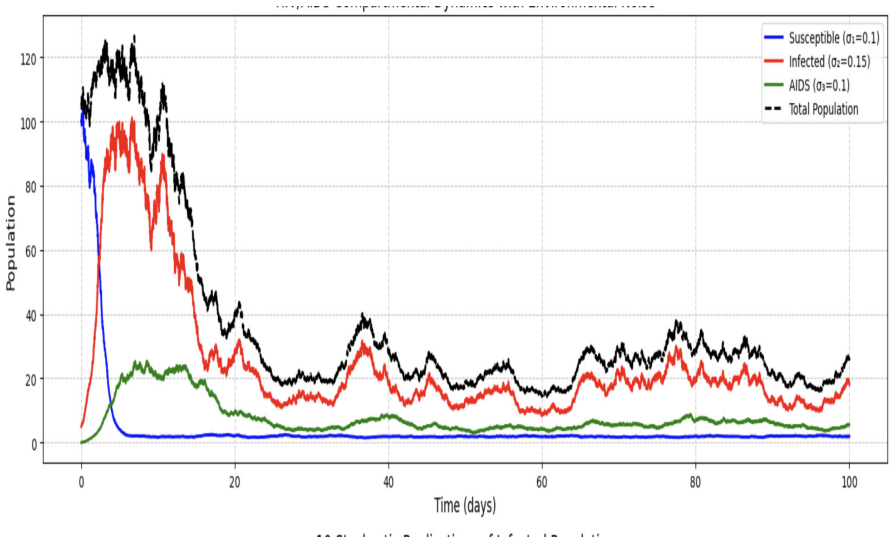

Figure 4.1: HIV/AIDS Compartmental Dynamics with Noise

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 β 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 4.1.

Figure 4.2 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 stochas-

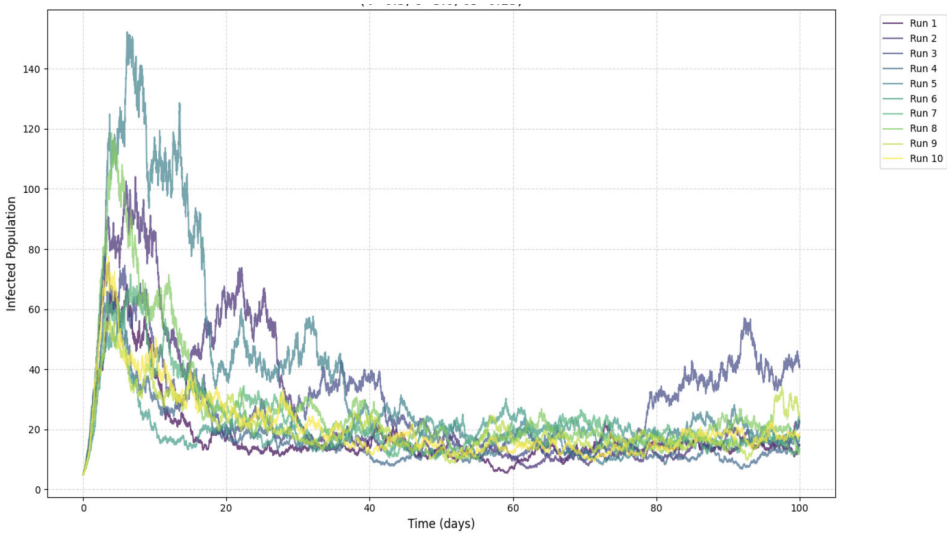

Figure 4.2: Multiple Stochastic Realizations

tic HIV/AIDS model analysis 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 β, progression rate δ, 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.

CHAPTER FIVE

CONCLUSIONS AND RECOMENDATIONS

5.1 Conclusion

This study successfully developed a comprehensive stochastic population model for HIV/AIDS transmission in heterosexual populations, achieving all specified objectives through the formulation of a stochastic differential equation system that incorporates environmental noise and demographic fluctuations. The analysis demonstrated that the model solutions remain positive and bounded, preserving biological realism, while 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 compared to deterministic approaches. Stability analysis revealed that the disease-free equilibrium becomes locally asymptotically stable when $R_0^S < 1$, with numerical simulations confirming significant fluctuations in disease prevalence, particularly in the infected compartment, highlighting the critical importance of accounting for stochasticity in epidemic forecasting and intervention planning for heterosexual populations.

5.2 Recommendations

Based on the study findings, we recommend that public health agencies incorporate stochastic modeling approaches into HIV/AIDS policy development, particularly by utilizing the noise-adjusted reproduction number R_0^S for more accurate threshold assessments and accounting for the pronounced variability in infected population dynamics when planning healthcare resources and intervention strategies. Future research should focus on estimating noise intensity parameters from empirical surveillance data, extending the framework to include age-structured mixing patterns and spatial heterogeneity in heterosexual networks, and developing parameter estimation methods that specifically capture the covariance structures between different noise processes to enhance the

model's predictive accuracy for real-world public health applications.

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Appendix A: R Simulation Code

This appendix contains the essential R code used for simulating the stochastic HIV/AIDS model.

A.1 Core Simulation Functions

Listing FIVE.1: Core Model Implementation

```
# Load required packages
library(Sim.DiffProc); library(ggplot2); library(dplyr)

# Model parameters (Table 4.1)
parameters <- list(
  Lambda = 2.0, tau = 0.5, c = 3.0, mu = 0.02,
  delta = 0.1, alpha = 0.3, sigma1 = 0.1, sigma2 = 0.15, sigma3 = 0.1
)

initial_state <- c(S = 100, I = 5, A = 0)

# Main simulation function
run_multiple_simulations <- function(n_simulations = 10) {
  simulations <- list()
  for (i in 1:n_simulations) {
    sim_result <- snssde1d(
      N = 1000, M = 1, t0 = 0, T = 100, Dt = 0.1,
      x0 = initial_state,
      drift = expression(
        Lambda - (tau * c / (S + I + A)) * S * I - mu * S,
        (tau * c / (S + I + A)) * S * I - (mu + delta) * I,
        delta * I - (mu + alpha) * A
      ),
      diffusion = expression(sigma1 * S, sigma2 * I, sigma3 * A),
      method = "euler", type = "ito"
    )
    simulations[[i]] <- as.data.frame(sim_result$X)
    simulations[[i]]$time <- seq(0, 100, 0.1)
    simulations[[i]]$simulation <- i
  }
  return(simulations)
}

# Calculate reproduction numbers
calculate_R0 <- function(parameters) {
  with(parameters, {
```

```
    R0_det <- (tau * c) / (mu + delta)
    R0_stoch <- R0_det - (sigma2^2) / (2 * (mu + delta))
    return(c(R0_deterministic = R0_det, R0_stochastic = R0_stoch))
  })
}
```

A.2 Analysis and Visualization

Listing FIVE.2: Data Analysis and Visualization

```
# Run simulations and calculate R0
set.seed(123)
multiple_sims <- run_multiple_simulations(10)
R0_values <- calculate_R0(parameters)

# Summary statistics
all_simulations <- do.call(rbind, multiple_sims)
summary_stats <- all_simulations %>%
  group_by(time) %>%
  summarise(
    S_mean = mean(S), S_sd = sd(S),
    I_mean = mean(I), I_sd = sd(I),
    A_mean = mean(A), A_sd = sd(A)
  )

# Plot mean dynamics with confidence intervals
plot_mean_dynamics <- ggplot(summary_stats, aes(x = time)) +
  geom_line(aes(y = S_mean, color = "Susceptible"), size = 1) +
  geom_ribbon(aes(ymin = S_mean - S_sd, ymax = S_mean + S_sd,
                  fill = "Susceptible"), alpha = 0.2) +
  geom_line(aes(y = I_mean, color = "Infected"), size = 1) +
  geom_ribbon(aes(ymin = I_mean - I_sd, ymax = I_mean + I_sd,
                  fill = "Infected"), alpha = 0.2) +
  geom_line(aes(y = A_mean, color = "AIDS"), size = 1) +
  geom_ribbon(aes(ymin = A_mean - A_sd, ymax = A_mean + A_sd,
                  fill = "AIDS"), alpha = 0.2) +
  labs(title = "HIV/AIDS Compartmental Dynamics with Noise",
       x = "Time", y = "Population Size") +
  theme_minimal()

# Multiple realizations plot
plot_multiple_realizations <- ggplot(all_simulations, aes(x = time, y =
  geom_line(aes(group = simulation), alpha = 0.7, size = 0.5) +
  labs(title = "Multiple Stochastic Realizations",
       x = "Time", y = "Infected Population") +
  theme_minimal() + theme(legend.position = "none")
```

A.3 Sensitivity and Equilibrium Analysis

Listing FIVE.3: Sensitivity and Equilibrium Analysis

```
# Sensitivity analysis
sensitivity_analysis <- function() {
  sigma_values <- seq(0.05, 0.25, by = 0.05)
  results <- data.frame()
  for (sigma_val in sigma_values) {
    mod_params <- parameters; mod_params$sigma2 <- sigma_val
    set.seed(123); sims <- run_multiple_simulations(5)
    final_infected <- sapply(sims, function(sim) tail(sim$I, 1))
    results <- rbind(results, data.frame(
      sigma = sigma_val, mean_final_I = mean(final_infected),
      sd_final_I = sd(final_infected),
      R0_stochastic = calculate_R0(mod_params)["R0_stochastic"]
    ))
  }
  return(results)
}

# Equilibrium analysis
calculate_equilibria <- function(parameters) {
  with(parameters, {
    DFE <- c(S_DFE = Lambda/mu, I_DFE = 0, A_DFE = 0)
    if (R0_values["R0_deterministic"] > 1) {
      S_EE <- (mu + delta) / (tau * c / (Lambda/mu))
      I_EE <- (Lambda - mu * S_EE) / (mu + delta)
      A_EE <- (delta * I_EE) / (mu + alpha)
      EE <- c(S_EE = S_EE, I_EE = I_EE, A_EE = A_EE)
    } else EE <- c(S_EE = NA, I_EE = NA, A_EE = NA)
    return(list(DFE = DFE, EE = EE))
  })
}

# Execute analyses
sensitivity_results <- sensitivity_analysis()
equilibria <- calculate_equilibria(parameters)

# Print key results
cat("R0 Deterministic:", round(R0_values["R0_deterministic"], 3), "\n")
cat("R0 Stochastic:", round(R0_values["R0_stochastic"], 3), "\n")
print("Disease-Free Equilibrium:"); print(equilibria$DFE)
```