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STOCHASTIC STABILITY AND LONG-TERM DYNAMICS OF AN HIV INFECTION MODEL WITH CD4+ T-CELL HOMEOSTASIS AND CTL IMMUNE RESPONSE

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Abstract. We examine a stochastic HIV infection model that includes CD4+ T-cell homeostasis and CTL-mediated immune response, represented by four compartments: uninfected CD4+ T cells, infected CD4+ T cells, free HIV virions, and CTL response. The model accounts for environmental fluctuations using multiplicative Itô noise. We establish the existence and uniqueness of the stochastic solution and derive Lyapunov estimates to show stochastic boundedness and long-term infection behavior. The impact of environmental noise on viral persistence and extinction thresholds is emphasized. Numerical simulations illustrate these theoretical findings, highlighting how stochastic perturbations affect CD4+ T-cell depletion, viral load, and CTL control, providing a robust framework for understanding HIV-immune system dynamics amidst environmental variability.

Keywords: homeostatic growth; immune response; global stability; deterministic HIV model; stochastic HIV models; infection cells; stationary distribution; extinction; stochastic differential equations; stochastic stability; stochastic Lyapunov function.

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1. INTRODUCTION

Human immunodeficiency virus (HIV) infection remains a major problem in mathematical and computational immunology because the interaction between viral replication, target-cell depletion, and host immune response produces complex temporal dynamics. In particular, the interaction among uninfected CD4+ T cells, infected CD4+ T cells, free viral particles, and cytotoxic T lymphocytes (CTLs) provides a fundamental framework for describing the progression of HIV infection and the ability of the immune system to control viral replication.

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CD4+ T cells play a central role in the immune response and are progressively depleted during HIV infection. Infected CD4+ T cells constitute an important source of viral production, whereas CTLs contribute to the elimination of infected cells and therefore represent one of the principal mechanisms of cellular immune control. Consequently, mathematical models incorporating both viral replication and CTL-mediated immune clearance can provide a useful framework for studying the interaction between HIV and the host immune system.

An additional biological mechanism that is relevant to the dynamics of CD4+ T cells is homeostatic proliferation. In the absence of severe infection, the CD4+ T-cell population is regulated around a characteristic physiological level. When the population decreases, compensatory proliferation can partially restore the population. To represent this mechanism, we incorporate a logistic-type homeostatic term

$$\rho B(t) \left(1 - \frac{B(t)}{K}\right),$$

where $B(t)$ denotes the concentration of uninfected CD4+ T cells, ρ is the maximum homeostatic proliferation rate, and K represents the carrying capacity associated with the healthy CD4+ T-cell population.

Human immunodeficiency virus (HIV) infection is a significant global public health concern, as the number of individuals living with HIV/AIDS worldwide exceeds 38 million [1]. In particular, the distinctive characteristic of HIV infection involves a progressive decline in CD4+T cells, leading to development of acquired immunodeficiency syndrome (AIDS), and consequently increasing susceptibility to opportunistic infections. The immune system plays an indispensable role in controlling HIV infection, and the interaction between the virus and the host cytotoxic T lymphocyte (CTL) immune response is a complex mathematical and dynamic process. Mathematical models have been widely used to provide a deeper understanding of the dynamics and to assess the impact of differential intervention strategies. However, many existing models of HIV infection neglect the immune response, particularly the cellular key role in controlling viral replication and delaying the progression to AIDS [2-6]. In this context, human immunodeficiency immune response mediated by CD8+CTLs, which nonetheless plays a virus (HIV) infection models that consider CD4+T cell homeostasis and the cytotoxic T lymphocyte dynamics have been established. Furthermore, the global stability of both the disease-free and endemic equilibrium points has been investigated. Under specific parametric conditions, it was demonstrated that the model exhibits backward and forward (trans critical) bifurcation. In addition, Hopf and Hopf bifurcations were identified. Moreover, a time lag was incorporated into the model to represent the delay between CD4+T cell infection and viral particle production, followed by stability and bifurcation analysis for the delay system. Comprehensive numerical experiments were also conducted to visualize the dynamical behavior of the HIV model and validate the analytical

findings [7-8]. More broadly, mathematical models of viral infections have played an important role in enhancing understanding of the dynamic of the viruses across different biological systems, including HIV, hepatitis T virus (HTV), and others. In particular, ordinary differential equations have been extensively used in epidemiology and virology to describe the dynamics of infectious diseases [9-21]. The basic and important research topics in this field includes the existence of the threshold values that distinguish between local and global stability of steady states, as well as the existence of periodic solutions, persistence, and extinction of disease. Furthermore, the author's previous research has made significant contributions to analyzing the properties of these systems from the perspective of stability and dynamics. Their work focused on system stability analysis, bifurcation analysis, and numerical simulation to evaluate system behavior under different scenarios. In [22], the author's developed a stochastic HIV model that captures the dynamics of infection by incorporating different epidemiological classes, encompassing susceptible individuals, asymptomatic HIV- positive individuals, and those exhibiting symptoms. Initially, the deterministic counterpart of the model was analyzed to establish the existence and stability of both disease-free and endemic equilibrium with in deterministic version of the model. Their analytical results confirmed that the basic reproduction number R_0 plays a fundamental role in determining the qualitative behavior of the system. Furthermore, the study extended the model by introducing environmental stochastic perturbations to better reflect real- world uncertainties. It was shown that stochastic effects can significantly influence the long –term dynamics of the disease. In particular, the results revealed the existence of critical thresholds under which the infection may become extinct. Interestingly even in situations where the deterministic model predicts persistence of the disease, stochastic fluctuations may lead to its eradication. From an epidemiological perspective, the inclusion of noise intensity was found to reduce oscillatory behavior in the system and decrease the average number of infected individuals. This highlights the important role of stochastic in shaping disease and suggests that deterministic models alone may not provide a complete picture of HIV progression. Motivated by these findings, the present study aims to further investigate HIV dynamics by incorporating both immune response mechanisms and stochastic effects within a unified modeling framework. In particular, we consider a dynamical model that includes homeostatic growth of CD4+T cells along cytotoxic immune response, and we extend it to stochastic differential equation system to account for environmental variability. The remainder of this work is organized as follows. In first section, we introduced fundamental definitions and basic concepts required for the analysis. In the subsequent section, we formulate the extend the existing model presented in [7] and analyze it's essential dynamical properties, including positive and boundedness of solutions, as well as the local and global stability of equilibrium points. Thereafter, we construct the corresponding stochastic model preliminary theoretical results. Finally, we perform numerical simulations system to illustrate the theoretical results.

Deterministic ordinary differential equation models have been extensively used to describe HIV infection dynamics. Such models can characterize viral replication, target-cell depletion, immune-mediated clearance, and the effects of therapeutic interventions. However, deterministic models implicitly assume that the biological parameters and population dynamics evolve without random environmental fluctuations.

This assumption may be restrictive in biological systems. Viral replication, cellular activation, immune responses, and host-environment interactions can be affected by intrinsic and extrinsic sources of variability. Consequently, stochastic differential equation models provide a natural extension of deterministic HIV models by incorporating environmental fluctuations directly into the population dynamics.

In the present study, we introduce multiplicative Itô noise into the four biological compartments. The multiplicative structure $\sigma_i X_i(t) dW_i(t)$ is particularly appropriate when the magnitude of environmental fluctuations depends on the size of the corresponding population.

Thus, stochasticity is introduced without changing the biological compartmental structure of the deterministic model.

Although stochastic HIV models have been investigated from different perspectives, several aspects of the long-term behavior of multi-compartment HIV models incorporating both CD4+ T-cell homeostasis and CTL-mediated immune clearance remain insufficiently characterized. In particular, a rigorous analysis should distinguish between local well-posedness, positivity, global existence, stochastic boundedness, and the asymptotic behavior of infection-related variables. Moreover, the effect of environmental noise on viral persistence and extinction cannot be inferred solely from deterministic threshold conditions.

The main objective of this work is to develop and rigorously analyze a stochastic HIV infection model incorporating CD4+ T-cell homeostasis and CTL-mediated immune response. The specific objectives are:

- to formulate a four-dimensional deterministic HIV model describing the interactions among uninfected CD4+ T cells, infected CD4+ T cells, free virus, and CTLs;
- to construct a stochastic counterpart by incorporating multiplicative environmental noise into the four biological compartments;
- to establish existence and uniqueness of the local stochastic solution;
- to prove positivity and forward invariance of the biological state space;
- to establish global existence and exclude finite-time explosion;
- to derive deterministic disease-free and endemic equilibria and determine the corresponding threshold quantity;
- to investigate stochastic extinction and persistence under appropriate conditions;

to examine numerically how environmental noise influences viral load, CD4+ T-cell dynamics, and CTL-mediated immune control.

The main contributions of this study are threefold. First, we provide a rigorous well-posedness analysis of the proposed stochastic HIV model, including positivity, global existence, and non-explosion. Second, we investigate threshold-dependent long-term behavior of the infection and clarify the role of environmental stochasticity in modifying deterministic HIV dynamics. Third, numerical experiments are used to complement the analytical results and illustrate the biological consequences of stochastic perturbations.

The remainder of the paper is organized as follows. Section 2 introduces the stochastic-analysis preliminaries required throughout the paper. Section 3 presents the deterministic and stochastic HIV models and discusses the biological meaning of their parameters. Section 4 establishes well-posedness, positivity, and global existence of the stochastic system. Section 5 investigates the stochastic long-term behavior, including extinction and persistence under suitable conditions. Section 6 presents numerical simulations supporting the analytical findings. Section 7 discusses the biological implications of the results, and concludes the paper.

2. PRELIMINARIES

In this section, we present some basic concepts related to stochastic differential equations and stochastic dynamical systems that are relevant to our study.

Definition 2.1 [22] Let $(\Omega, \mathcal{F}, \mathbb{P})$ be a complete probability space with a natural filtration $\{\mathcal{F}_t\}_{t \in [0, t]}$ satisfying the usual conditions. A standard one-dimensional brownian motion (or wiener process) $(B_t)_{t \geq 0}$ is a real valued continuous $\{\mathcal{F}_t\}_{t \in [0, t]}$ -adapted process with the following properties:

- (i) $\beta_0 = 0$ almost surely.
- (ii) For any $0 \leq s < t < \infty$, the increment $\beta_t - \beta_s$ is normally distributed with mean zero and variance $t - s$.
- (iii) The increments are independent of $\mathcal{F}_s$.

Definition 2.2 [22] Assume that $(\Omega, \mathcal{F}, \mathbb{P})$ is a complete probability space with natural filtration $\{\mathcal{F}_t\}_{t \geq 0}$ satisfying the usual conditions. We assume that, $\beta(t) = (\beta_1(t) \dots \beta_m(t))^T t > 0$ is an n -dimensional Brownian motion (wiener process) defined on the space. Let $0 \leq t_0 < \infty$ and $x_0 \in \mathcal{F}_{t_0}$ – a measurable $\mathbb{R}^d$ – random variable with $E|x_0|^2 < \infty$. Assume that $f: \mathbb{R}^d \times [t_0, T] \rightarrow \mathbb{R}^d$ and $g: \mathbb{R}^d \times [t_0, T] \rightarrow \mathbb{R}^{d \times m}$ are both Borel measurable. Consider the following d –dimensional stochastic differential equation of Itô type

$$d(X(t)) = f(t, X(t))dt + g(t, X(t))d\beta(t) \quad (2.0)$$

With initial value $X(0) = X_0$. By definition of stochastic differential equation, this equation is equivalent to the following stochastic differential equation:

$$X(t) = X(t_0) + \int_{t_0}^t f(s, X(s))ds + \int_{t_0}^t \sigma(X(s))d\beta(s).$$

Definition 2.3 [22] An $\mathbb{R}^d$ -valued stochastic process $\{X(t)_{t_0 < t < T}\}$ is called a solution of equation (2.0) if it has the following properties:

- (i) $\{X(t)\}$ is continuous and $\mathcal{F}_t$ -adapted;
- (ii) $f(t, X(t)) \in L([t_0, T], \mathbb{R}^d)$ and $g(s, X(s)) \in L^2([t_0, T], \mathbb{R}^{d \times m})$,
- (iii) Equation (2.0) holds for every $t \in [t_0, T]$ with probability 1.

Theorem 2.4 [22]: Assume that there exist two positive constant k and $\tilde{k}$ such that:

- (i) **(Lipschitz condition)** for all $x, y \in \mathbb{R}^k$, and all $t \in [t_0, T]$

$$|f(t, x) - f(t, y)|^2 \vee |g(t, x) - g(t, y)|^2 < \tilde{k}|x - y|^2,$$

- (ii) **(Linear growth condition)** For all $(x, t) \in \mathbb{R}^k \times [t_0, T]$

$$|f(t, x)|^2 \vee |g(t, x)|^2 < K(1 + |x|^2).$$

Then there exists a unique solution belong to $M^k([t_0, T]; \mathbb{R}^k)$.

Definition 2.5 [22]: (Stability of differential equation)

(i) The trivial solution of equation (2.0) is called stable if, for any $\varepsilon \in (0, 1)$, there exists $\delta_0 = \delta_0(\varepsilon, t_0) > 0$ with $\{|x(t, t_0, x_0)| < \varepsilon\}$ for all $t \geq t_0$ when $|x_0| < \delta$. Otherwise it is said to be unstable.

(ii) The trivial solution of equation (2.0) is asymptotically stable if it is stable and there exists $\delta_0 = \delta_0(t_0) > 0$ such that $\lim_{t \rightarrow \infty} x(t, t_0, x_0) = 0$, whenever $|x_0| < \delta$.

(iii) The trivial solution of equation (2.0) is called stochastically stable if, for any $\varepsilon \in (0, 1)$, there exists $\delta_0 = \delta_0(\varepsilon, t_0) > 0$ and $r > 0$ such that $\mathbb{P}\{|x(t, t_0, x_0)| < r : t > t_0\} > 1 - \varepsilon$, when $|x_0| < \delta$.

Otherwise it is said to be unstable.

v) The trivial solution of equation (2.0) is called stochastically asymptotically stable if for any $\varepsilon \in (0, 1)$ there exists $\delta_0 = \delta_0(\varepsilon, t_0) > 0$ with $\mathbb{P}\{\lim_{t \rightarrow \infty} x(t, t_0, x_0) = 0\} \geq 1 - \varepsilon$, when $|x_0| < \delta$.

(vi) The trivial solution of equation (2.0) is called stochastically asymptotically stable in the large if it is stochastically stable and moreover, for all $x_0 \in \mathbb{R}^d$

$$\mathbb{P}\{\lim_{t \rightarrow \infty} x(t, t_0, x_0) = 0\} = 1.$$

(vii) The trivial solution of equation (2.1) is called globally asymptotically stable any $\varepsilon > 0$ there exists $\delta > 0$ such that $\{|x(t) - x^*| < \varepsilon : \forall t > 0\}$, when $|x(0) - x^*| < \delta$ where x^* is equilibrium point.

Definition 2.6 [22]: Let $C^{2,1}(\mathbb{R}^d \times \mathbb{R}_+; \mathbb{R})$ denote the family of all real-valued functions $V(x, t)$ defined on $\mathbb{R}^d \times \mathbb{R}_+$ such that are twice continuously differentiable in X and once in t . If $V(x, t) \in C^{2,1}(\mathbb{R}^d \times \mathbb{R}_+; \mathbb{R})$, we define the Lyapunov operator by

$$\mathcal{L} := \frac{\partial}{\partial t} + \sum_{i=1}^d f_i(x, t) \frac{\partial}{\partial x_i} + \frac{1}{2} \sum_{i,j} [g(x, t) g^T(x, t)]_{ij} \frac{\partial^2}{\partial x_i \partial x_j},$$

$$\text{where, } V_t = \frac{\partial v}{\partial t}, V_x = \left(\frac{\partial v}{\partial x_1}, \dots, \frac{\partial v}{\partial x_d} \right), V_{xx} = \left(\frac{\partial^2 v}{\partial x_i \partial x_j} \right)_{d \times d} = \begin{bmatrix} \frac{\partial^2 v}{\partial x_1 \partial x_1} & \dots & \frac{\partial^2 v}{\partial x_1 \partial x_d} \\ \vdots & \dots & \vdots \\ \frac{\partial^2 v}{\partial x_d \partial x_1} & \dots & \frac{\partial^2 v}{\partial x_d \partial x_d} \end{bmatrix}$$

so,

$$\mathcal{L}[V] = \left[V_t + f(x, t) V_x + 1/2 \text{trace} \left(g^T(t) \left(\frac{\partial^2 v}{\partial x^2} \right) g(t) \right) \right] dt.$$

Definition (2.7)[22]: A stochastic process $X(t) = (B(t), C(t), D(t), E(t))$ is said to be ultimately bounded in mean if there exists a positive constant $M > 0$ and at time $T > 0$ such that: $\mathbb{E}|X(t)| \leq M, \forall t \geq T$. Equivalently, $\limsup_{t \rightarrow \infty} \mathbb{E}[|X(t)|] \leq M$.

For the analysis, it is convenient to introduce

$$X(t) = (B(t), C(t), D(t), E(t)) \in \mathbb{R}_+^4.$$

We write the stochastic HIV model as

$$dX(t) = F(X(t))dt + G(X(t))dW(t). \quad (2.1)$$

The drift vector is

$$F(x) = (F_1(x), F_2(x), F_3(x), F_4(x))^T,$$

where, $x = (b, c, d, e)^T$, $F_1(x) = \Lambda + \rho b(1 - \frac{b}{K}) - kbd - d_B b$, $F_2(x) = kbd - d_c c - d_x e c$, $F_3(x) = pc - d_d d$, and $F_4(x) = qc - d_e e$.

The diffusion matrix is diagonal:

$$G(x) = G = \begin{pmatrix} \sigma_1 b & 0 & 0 & 0 \\ 0 & \sigma_2 c & 0 & 0 \\ 0 & 0 & \sigma_3 d & 0 \\ 0 & 0 & 0 & \sigma_4 e \end{pmatrix}. \quad (2.2)$$

This formulation will be used throughout the analytical part of the paper.

We now give the precise notion of solution.

Definition 2.8 (Strong solution)[22]: Let $X_0 \in \mathbb{R}_+^4$ be $\mathcal{F}_0$ -measurable. An adapted continuous process

$$X(t) = (B(t), C(t), D(t), E(t))$$

is called a strong solution of (2.1) on an interval $[0, \tau)$, where τ is a stopping time, if

$$X(t) = X_0 + \int_0^t F(X(s)) ds + \int_0^t G(X(s)) dW(s)$$

for every $0 \leq t < \tau$, almost surely. In component form, this means, for example,

$$B(t) = B_0 + \int_0^t F_1(X(s)) ds + \int_0^t \sigma_1 B(s) dW_1(s),$$

and similarly for C, D, E .

The following property is fundamental for our first existence theorem.

Definition 2.9 (Local Lipschitz continuity)[22] A function $\mathbf{H}: \mathbb{R}^4 \rightarrow \mathbb{R}^m$ is said to be **locally Lipschitz** if, for every $\mathbf{R} > \mathbf{0}$, there exists a constant $\mathbf{L}_R > \mathbf{0}$ such that

$$\|H(x) - H(y)\| \leq L_R \|x - y\|$$

whenever $\|x\| \leq R, \|y\| \leq R$.

Lemma 2.10 (Local Lipschitz Property of the HIV Coefficients) For the stochastic HIV model (2.1), the drift coefficient $\mathbf{F}: \mathbb{R}^4 \rightarrow \mathbb{R}^4$ and the diffusion coefficient $\mathbf{G}: \mathbb{R}^4 \rightarrow \mathbb{R}^4 \times \mathbb{R}^4$ are locally Lipschitz continuous.

Proof Each component of $\mathbf{F}$ is a polynomial in $(\mathbf{b}, \mathbf{c}, \mathbf{d}, \mathbf{e})$. For example,

$$F_1(b, c, d, e) = \Lambda + \rho b - \frac{\rho}{K} b^2 - kbd - d_B b.$$

The only nonlinear terms are b^2 and bd . For x, y in a bounded set, say

$$\|x\|, \|y\| \leq R,$$

we have

$$|b_x^2 - b_y^2| = |b_x - b_y| |b_x + b_y| \leq 2R |b_x - b_y|.$$

Similarly, $|b_x d_x - b_y d_y|$ can be written as

$$b_x d_x - b_y d_y = d_x (b_x - b_y) + b_y (d_x - d_y).$$

Hence

$$|b_x d_x - b_y d_y| \leq R |b_x - b_y| + R |d_x - d_y|.$$

Therefore F_1 is Lipschitz on every bounded subset. The same argument applies to

$$F_2(b, c, d, e) = kbd - d_c c - d_x ec,$$

because the nonlinear terms are bd and ec . The remaining two components,

$$F_3(b, c, d, e) = pc - d_d d$$

and

$$F_4(b, c, d, e) = qc - d_e e,$$

are linear. Therefore F is locally Lipschitz. For G , every nonzero entry is a linear function:

$$\sigma_1 b, \sigma_2 c, \sigma_3 d, \sigma_4 e.$$

Consequently,

$$\|G(x) - G(y)\| \leq C\|x - y\|$$

for some constant $C > 0$. Thus G is globally Lipschitz, and therefore locally Lipschitz. Hence F and G are locally Lipschitz.

Because the coefficients are only locally Lipschitz rather than globally Lipschitz, the first existence result initially gives a local solution. We therefore need to define its maximal lifetime.

Definition 2.11 (Explosion time)[22] The explosion time of the maximal local solution $\mathbf{X}(t)$ of (2.1) is given by

$$\tau_e = \lim_{n \rightarrow \infty} \tau_n, \quad (2.3)$$

where

$$\tau_n = \inf\{t \geq 0: \|\mathbf{X}(t)\| \geq n\}. \quad (2.4)$$

By convention, $\inf \emptyset = \infty$. If $\tau_e < \infty$, the solution leaves every bounded subset of $\mathbb{R}^4$ in finite time.

Let $V \in C^2(\mathbb{R}_+^4; \mathbb{R})$. For $x = (b, c, d, e)$, the infinitesimal generator associated with (2.1) is

$$LV(x) = \sum_{i=1}^4 F_i(x) \frac{\partial V}{\partial x_i} + \frac{1}{2} (GG^T)_{ij}(x) \frac{\partial^2 V}{\partial x_i \partial x_j}. \quad (2.5)$$

Since $G(x)$ is diagonal,

$$GG^T = \begin{pmatrix} \sigma_1^2 b^2 & 0 & 0 & 0 \\ 0 & \sigma_2^2 c^2 & 0 & 0 \\ 0 & 0 & \sigma_3^2 d^2 & 0 \\ 0 & 0 & 0 & \sigma_4^2 e^2 \end{pmatrix}$$

Therefore

$$\begin{aligned} LV &= F_1 V_b + F_2 V_c + F_3 V_d + F_4 V_e \\ &+ \frac{1}{2} \sigma_1^2 b^2 V_{bb} + \frac{1}{2} \sigma_2^2 c^2 V_{cc} \\ &+ \frac{1}{2} \sigma_3^2 d^2 V_{dd} + \frac{1}{2} \sigma_4^2 e^2 V_{ee}. \end{aligned} \quad (2.6)$$

This operator will be the principal analytical tool in Sections 4–6.

Theorem 2.12 [22] (Itô's Formula) Let $\mathbf{X}(t)$ satisfy $d\mathbf{X}(t) = \mathbf{F}(\mathbf{X}(t))dt + \mathbf{G}(\mathbf{X}(t))d\mathbf{W}(t)$, and let $V \in C^2(\mathbb{R}_+^4; \mathbb{R})$. Then

$$dV(\mathbf{X}(t)) = LV(\mathbf{X}(t))dt + \nabla V(\mathbf{X}(t))^T \mathbf{G}(\mathbf{X}(t))d\mathbf{W}(t). \quad (2.7)$$

Equivalently,

$$V(X(t)) = V(X_0) + \int_0^t LV(X(s)) ds + \int_0^t \nabla V(X(t))^T G(X(t)) dW(s). \quad (2.8)$$

The final stochastic integral is a local martingale whenever the standard square-integrability conditions hold.

Definition 2.13 A function $V: \Omega \times \mathbb{R}_+^4 \rightarrow [0, \infty)$ is called a stochastic radially unbounded Lyapunov function if $V(\omega, \cdot) \in \mathcal{C}^2(\mathbb{R}_+^4)$ and $V(\omega, x) \rightarrow \infty$ as $\|x\| \rightarrow \infty$ almost surely.

For the present model, a particularly useful choice is

$$V_1(B, C, D, E) = B + C + D + E. \quad (2.9)$$

This function will be used to establish global existence and boundedness.

For higher-order moment estimates we will later use

$$V_p(B, C, D, E) = 1 + B^p + C^p + D^p + E^p, p \geq 2. \quad (2.10)$$

Definition 2.14(Biological state space): The natural state space of the stochastic HIV model is

$$\mathcal{X} = \mathbb{R}_+^4. \quad (2.11)$$

Its interior is

$$\mathcal{X}^\circ = \mathbb{R}_+^4. \quad (2.12)$$

The boundary $\partial\mathcal{X} = \mathcal{X} \setminus \mathcal{X}^\circ$ contains states in which at least one biological compartment is zero.

The desired invariance property is

$$X_0 \in \mathcal{X} \implies X(t) \in \mathcal{X}, \forall t \geq 0, \text{a.s.} \quad (2.13)$$

This will be proved rigorously in Section 4.

Definition 2.15 (Almost sure extinction) We say that HIV infection becomes extinct almost surely if

$$\lim_{t \rightarrow \infty} [C(t) + D(t)] = 0 \text{ a.s.} \quad (2.14)$$

A **stronger exponential extinction** property is

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \ln(C(t) + D(t)) < 0 \text{ a.s.} \quad (2.15)$$

Whenever (2.15) holds, infection converges to zero exponentially fast.

We must also distinguish persistence from mere non-extinction.

Definition 2.16 (Stochastic persistence) The infection is said to be stochastically persistent if, for every sufficiently small $\varepsilon > 0$, there exists $\delta > 0$ such that

$$\liminf_{t \rightarrow \infty} \mathbb{P}\{C(t) + D(t) > \delta\} \geq 1 - \varepsilon. \quad (2.16)$$

This is the appropriate notion to use later if we prove persistence in probability.

3. MATHEMATICAL MODEL FORMULATIONS

In this section, we adopt an existing deterministic HIV model and develop its stochastic counterpart by incorporating environmental fluctuations and provide the biological interpretation of the model variables and parameters.

3.1. DETERMINISTIC MODEL FORMULATION

HIV targets CD4+ T cells upon entering the body, leading to a decline in their population. Uninfected CD4+ T cells send signals to activate CTLs to eliminate infected cells and prevent viral spread. To maintain the level of CD4+ T cells, homeostatic proliferation allows the replenishment of CD4+ T cells [25, 26]. Mathematical modeling is critical for understanding these interactions, predicting HIV progression, evaluating treatment strategies and guiding the development of immunotherapies. Here, we construct a mathematical model that depicts the dynamics of the spread of HIV infection with

CD4+T and CTLs cells based on the following assumptions:

(A1) $B(t)$ and $C(t)$ are the concentrations of uninfected and infected CD4+ T cells at any time $t > 0$, respectively.

(A2) $D(t)$ and $E(t)$ are the concentrations of free virus and CTLs cells at any time $t > 0$, respectively.

(A3) The CD4+ T cells are assumed to be growing with the constant growth rate Λ [27].

(A4) To model the homeostatic production of CD4+ T cells in response to the presence of a virus and the subsequent decrease in healthy CD4+ T cells, which can initiate throughout the infection, we use $\rho B(t) \left(1 - \frac{B(t)}{K}\right)$, K is the carrying capacity of healthy cells.

The biological meaning:

-If K decreases, reproduction is activated.

-If K approaches the normal level, growth gradually stops.

(A5) k is the rate at which CD4+ T cells get infected as they come in contact with the virus.

(A6) d_x the clearance rate of infected CD4+T cells due to CTL immune response

(A7) p and q are the production rates of free virus and CTLs cells, respectively.

(A8) d_B , d_D , d_C and d_E are the natural death rates of uninfected CD4+T cells, infected CD4+T cells, free virus and CTLs cells, respectively.

In view of the above said assumptions, we propose the following HIV infection model

$$\begin{aligned} \frac{dB}{dt} &= \Lambda + \rho B(t) \left(1 - \frac{B(t)}{K}\right) - kD(t)B(t) - d_B B(t), \\ \frac{dC(t)}{dt} &= kB(t)D(t) - d_C C(t) - d_x E(t)C(t), \end{aligned} \quad (3.1)$$

$$\frac{dD(t)}{dt} = pC(t) - d_D D(t),$$

$$\frac{dE(t)}{dt} = qC(t) - d_E E(t).$$

With initial value $B(0) = B_0 > 0$, $C(0) = 0$, $D(0) = D_0 \geq 0$, $E(0) = E_0 \geq 0$. All parameters in the model are considered to have non-negative values, and their meanings are proved in summarized in Table 1.

The initial represent the moment when then HIV enters a body that has a normal number CD4+T cells, no infected cells yet, an initial viral load, and an immune response that may be present or not yet activated, so the infected dynamics start from realistic and non-negative biology state.

Table 1. Values of parameters.

References	Values	Parameters and Variables	Description
_____	_____	B	Uninfected CD4+T cells
_____	_____	C	Infected CD4+ T cells
_____	_____	D	Free HIV virus
_____	_____	E	CTL immune response
Assumed	$10 \times day^{-1}$	Λ	Natural inflow rate of uninfected CD4+B cell
Assumed	$1.6126 \times 10^{-4} mm^3 day^{-1}$	k	Infection rate of uninfected CD4+ T cell with virus
[17]	$0.1 day^{-1}$	d_B	Death rate of uninfected CD4+ T cell
[17]	$0.01 day^{-1}$	p	Maximum homeostatic growth rate
Assumed	$2.4 day^{-1}$	d_D	Death rate of free virus
[7]	$0.3 day^{-1}$	d_C	Death rate of infected CD4+T cells
[7]	$0.01 day^{-1}$	d_E	Death rate of CTL immune response
Assumed	$0.01 day^{-1}$	d_x	Clearance rate of infected cells by CTL immune response
Assumed	$30 day^{-1}$	p	Rate of virus production
[18]	$300 day^{-1}$	G	Homeostatic half velocity
[18]	$0.8 day^{-1}$	q	Constant growth rate of CTLs immune response

3.1.1. DISEASE-FREE EQUILIBRIUM

We now derive the disease-free equilibrium rigorously. At a disease-free state, there are no infected cells, no free virus, and consequently no activated CTL response generated by infection. Therefore,

$$C^* = 0, D^* = 0, E^* = 0.$$

The healthy-cell component B^* must satisfy

$$0 = \Lambda + \rho B^* \left(1 - \frac{B^*}{K}\right) - d_B B^*.$$

Expanding,

$$0 = \Lambda + \rho B^* - \frac{\rho}{K} (B^*)^2 - d_B B^*.$$

Hence

$$\frac{\rho}{K} (B^*)^2 + (d_B - \rho) B^* - \Lambda = 0. \quad (3.2)$$

Assuming $\rho > 0$, multiplication by K gives

$$\rho (B^*)^2 + K(d_B - \rho) B^* - K\Lambda = 0.$$

Applying the quadratic formula,

$$B^* = \frac{-K(d_B - \rho) \pm \sqrt{K^2(d_B - \rho)^2 + 4\rho K\Lambda}}{2\rho}.$$

Thus

$$B^* = \frac{K(\rho - d_B) \pm \sqrt{K^2(\rho - d_B)^2 + 4\rho K\Lambda}}{2\rho}.$$

Because

$$K^2(\rho - d_B)^2 + 4\rho K\Lambda > |K(\rho - d_B)|,$$

the negative branch is strictly negative. Hence only the positive branch is biologically admissible. We therefore obtain

$$B_0^* = \frac{K(\rho - d_B) + \sqrt{K^2(\rho - d_B)^2 + 4\rho K\Lambda}}{2\rho}. \quad (3.3)$$

Consequently, the unique biologically meaningful disease-free equilibrium is

$$\mathcal{E}_0 = (B_0^*, 0, 0, 0). \quad (3.4)$$

The quantity B_0^* is the healthy CD4+ T-cell carrying equilibrium in the absence of HIV infection. It is important that B_0^* is not simply $d_B \Lambda$. That expression would arise if homeostatic proliferation were absent. Here, because the model contains $\rho B(1 - \frac{B}{K})$, the disease-free population is determined by the nonlinear balance equation (3.2). This distinction is important when calculating R_0 .

3.1.2. LINEARIZATION AROUND THE DISEASE-FREE EQUILIBRIUM

To derive the basic reproduction number, we identify the infected compartments. The infection-related variables are C, D . The variable E is not included in the infected subsystem at the linear level because $-d_x EC$ is a product of two quantities that vanish at the disease-free equilibrium. Indeed, around $\mathcal{E}_0 = (B_0^*, 0, 0, 0)$, the term EC is of second order. Therefore the linearized infected subsystem is

$$\frac{dC}{dt} = kB_0^*D - d_C C, \quad \frac{dD}{dt} = pC - d_D D. \quad (3.5)$$

This two-dimensional subsystem is the correct starting point for the next-generation method.

We now derive R_0 systematically. Let $x_I = (C, D)$ denote the infected subsystem. Following the next-generation approach, write

$$\frac{dx_I}{dt} = F(x_I) - V(x_I),$$

where F contains the terms associated with the appearance of newly infected biological units, while V contains transfers and removals. For the present model, we choose $F = \begin{pmatrix} kB_0^*D \\ 0 \end{pmatrix}$, because new infected CD4+ T cells are generated through viral infection. The transition/removal vector is

$$V = \begin{pmatrix} d_C C \\ d_D D - pC \end{pmatrix}.$$

Thus the Jacobian matrices evaluated at the disease-free equilibrium are

$$F = DF(\mathcal{E}_0) = \begin{pmatrix} 0 & kB_0^* \\ 0 & 0 \end{pmatrix}, \quad (3.6)$$

and

$$V = DV(\mathcal{E}_0) = \begin{pmatrix} d_C & 0 \\ -p & d_D \end{pmatrix}. \quad (3.7)$$

Since $\det V = d_C d_D > 0$, the matrix V is invertible. Its inverse is

$$V^{-1} = \begin{pmatrix} \frac{1}{d_C} & 0 \\ \frac{p}{d_C d_D} & \frac{1}{d_D} \end{pmatrix}. \quad (3.8)$$

The **next-generation matrix** is defined by

$$\mathcal{K} = FV^{-1}. \quad (3.9)$$

Therefore,

$$\mathcal{K} = \begin{pmatrix} 0 & kB_0^* \\ 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{d_C} & 0 \\ \frac{p}{d_C d_D} & \frac{1}{d_D} \end{pmatrix} = \begin{pmatrix} \frac{kB_0^* p}{d_C d_D} & \frac{kB_0^*}{d_D} \\ 0 & 0 \end{pmatrix}.$$

Hence

$$\mathcal{K} = \begin{pmatrix} \frac{kB_0^*p}{d_C d_D} & \frac{kB_0^*}{d_D} \\ 0 & 0 \end{pmatrix}. \quad (3.10)$$

Because $\mathcal{K}$ is upper triangular, its eigenvalues are $\lambda_1 = \frac{kB_0^*p}{d_C d_D}$, $\lambda_2 = 0$. Consequently, $\rho(\mathcal{K}) = \frac{kB_0^*p}{d_C d_D}$.

Therefore, we define

$$R_0 = \rho(\mathcal{K}) = \frac{kB_0^*p}{d_C d_D}. \quad (3.11)$$

Substituting (3.3),

$$R_0 = \rho(\mathcal{K}) = \frac{kp \ K(\rho - dB) \pm \sqrt{K^2(\rho - dB)^2 + 4\rho K\Lambda}}{2\rho}. \quad (3.12)$$

This is the correct deterministic basic reproduction number for the present model under the chosen next-generation decomposition.

The expression $R_0 = \frac{kB_0^*p}{d_C d_D}$ has a natural interpretation. The factor $\frac{1}{d_C}$ represents the characteristic lifetime of an infected CD4+ T cell. During that period, infected cells produce virus at rate p . Each viral particle has an average lifetime $\frac{1}{d_D}$. The infection rate is controlled by kB_0^* .

Therefore the combination $\frac{kB_0^*p}{d_C d_D}$ measures the effective number of newly infected CD4+ T cells generated by one infection cycle near the disease-free state. Importantly, CTL-mediated clearance does not appear explicitly in R_0 . This is not an omission. At the disease-free equilibrium, $C^* = E^* = 0$, and therefore $d_x EC$ is a nonlinear term of order two. Hence CTL-mediated removal affects the endemic dynamics and nonlinear persistence behavior, but it does not contribute to the first-order invasion reproduction number around the disease-free equilibrium. This is an important biological and mathematical observation.

The next-generation matrix gives the natural invasion threshold $R_0 = 1$. Thus the deterministic model has the standard threshold interpretation: $R_0 < 1$. Corresponds to the regime in which HIV cannot invade the disease-free state at the linearized level. Conversely, $R_0 > 1$ corresponds to the regime in which the infection has positive linear growth near the disease-free equilibrium.

However, we should be careful in the paper: $R_0 < 1$ by itself should not yet be stated as a global extinction theorem.

To establish global asymptotic stability of the disease-free equilibrium, we still need an appropriate stability argument.

Likewise, $R_0 > 1$ does not automatically prove stochastic persistence. These will be separate theorems.

Since $R_0 = \frac{kB_0^*p}{d_C d_D}$, we can define the critical infection rate

$$k_c = \frac{d_C d_D}{B_0^* p}. \quad (3.13)$$

Then $R_0 < 1 \Leftrightarrow k < k_c$, while $R_0 > 1 \Leftrightarrow k > k_c$. This will be useful in the numerical section because we can vary k while keeping the other biological parameters fixed.

There is an important consequence of the above derivation. The parameter d_x does not occur in (3.11). Therefore we should not claim that increasing d_x changes the basic invasion threshold R_0 .

Instead: d_x controls nonlinear immune clearance after infection is established.

Whereas R_0 controls initial invasion of the disease-free state. This distinction gives the paper a much stronger biological interpretation.

We now have the deterministic threshold $R_0 = \frac{kB_0^*p}{d_C d_D}$. The stochastic model is

$$\begin{aligned} dB &= F_1(X)dt + \sigma_1 B dW_1, \\ dC &= F_2(X)dt + \sigma_2 C dW_2, \\ dD &= F_3(X)dt + \sigma_3 D dW_3, \\ dE &= F_4(X)dt + \sigma_4 E dW_4. \end{aligned}$$

Near the disease-free state, the infected subsystem becomes approximately

$$dC = (kB_0^*D - d_C C)dt + \sigma_2 C dW_2, \quad dD = (pC - d_D D)dt + \sigma_3 D dW_3. \quad (3.14)$$

3.2. STOCHASTIC MODEL FORMULATION

We extend the deterministic HI Model by adding white noise terms. The stochastic HIV model is driven by introducing white noise terms $\sigma_i d\beta_i(t)$ to the state variable $B(t)$, $C(t)$, $D(t)$ and $E(t)$. Here $\beta_i(t)$, $i = 1, 2, 3$, are independent standard Brownian motion, and σ_i represent the intensities of environmental stochastic effects. This leads to a stochastic differential equation system in the Ito sense. Thus, the system can be written as follows:

$$\begin{aligned} dB(t) &= \left[\Lambda + \rho B(t) \left(1 - \frac{B(t)}{K} \right) - kD(t)B(t) - d_B B(t) \right] dt + \sigma_1 B(t) d\beta_1(t), \\ dC(t) &= [kB(t)D(t) - d_C C(t) - d_x E(t)C(t)]dt + \sigma_2 C(t) d\beta_2(t), \\ dD(t) &= [pC(t) - d_D D(t)]dt + \sigma_3 D(t) d\beta_3(t), \\ dE(t) &= [qC(t) - d_E E(t)]dt + \sigma_4 E(t) d\beta_4(t). \end{aligned} \quad (3.15)$$

The multiplicative noise terms represent environmental fluctuations whose magnitude scales with the corresponding biological population.

For simplify, we write $X(t) = (B(t), C(t), D(t), E(t))^T$. Hence

$$dX(t) = F(X(t))dt + G(X(t))dW(t). \quad (3.16)$$

4. BIOLOGICAL ACCEPTABLE OF THE STOCHASTIC MODEL

In this section, we will show whether the solution of equation (3.2) are realistic or not, for all the parameters adopted in the model. In order to make sure that the solutions are positive and bounded, we will use the principle of random comparison, so we have:

Theorem 4.1 (Existence, Uniqueness and Positivity of the Local Solution) The solutions of (3.2) are positive and bounded.

Proof Consider the first equation of (3.2).

$$\begin{aligned} dB &= \left[\Lambda + \rho B(t) \left(1 - \frac{B(t)}{K} \right) - kD(t)B(t) - d_B B(t) \right] dt + \sigma_1 B(t) d\beta_1(t) \\ &\leq \left[\Lambda + \rho B \left(1 - \frac{B}{K} \right) \right] dt + \sigma_1 B d\beta_1(t). \end{aligned}$$

Now, consider the new SDE

$$dB^* = \left[\Lambda + \rho B^* \left(1 - \frac{B^*}{K} \right) \right] dt + \sigma_1 B^* d\beta_1(t).$$

Note that it is not a Geometric Brownian Motion equation due to the presence of the logistic term and the constant Λ , and therefore it does not have a closed-form solution in the usual sense. Therefore, the correct mathematical solution is to use appropriate approximations. If $\frac{B}{K} \approx 0$, then the new SDE becomes

$$dB^* = [\Lambda + \rho B^*] dt + \sigma_1 B^* d\beta_1(t)$$

and this is a linear equation in Itô, which can be solved using the integrating factor method. Then the explicit solution is given by

$$B^* = \Phi_1(t) \{ B^*(0) + \Lambda \int_0^t \Phi_1(\tau)^{-1} d\tau \},$$

where $\Phi_1(t) = \exp \left[\left(\rho - \frac{\sigma_1^2}{2} \right) t + \sigma_1 \beta_1(t) \right]$.

This represents a good approximation in the early stages of the infection.

Then

$$B(t) \leq \exp \left[\left(\rho - \frac{\sigma_1^2}{2} \right) t + \sigma_1 \beta_1(t) \right] + \Lambda \int_0^t \exp \left[- \left(\rho - \frac{\sigma_1^2}{2} \right) t - \sigma_1 \beta_1(t) \right] d\tau.$$

We claim that

$$\lim_{t \rightarrow \infty} \sup B(t) \leq \frac{\Lambda}{\frac{\sigma_1^2}{2} - \rho}.$$

For, suppose that $a = \rho - \frac{\sigma_1^2}{2}$, with $a < 0$. We have

$$B(t) \leq X(t)(B(0) + \Lambda I(t)),$$

where $X(t) = e^{at + \sigma_1 \beta_1(t)}$, and $I(t) = \int_0^t X^{-1}(\tau) d\tau$. From the "Law of the Iterated Logarithm"

we know that $\frac{\beta_1(t)}{t} \rightarrow 0$ a.s. So,

$$\frac{\log X(t)}{t} = a + \sigma_1 \frac{\beta_1(t)}{t} \rightarrow a \text{ a.s.}$$

Since $a < 0$, then $X(t) \rightarrow 0$ a.s. To compute $X(t)I(t)$. Write

$$I(t) = \int_0^t e^{-a\tau - \sigma_1 \beta_1(\tau)} d\tau.$$

Then

$$X(t)I(t) = \int_0^t e^{a(t-\tau) + \sigma_1(\beta_1(t) - \beta_1(\tau))} d\tau.$$

Set $s = t - \tau$, we get

$$X(t)I(t) = \int_0^t e^{as + \sigma_1(\beta_1(t) - \beta_1(t-s))} ds.$$

Since $W(t) - W(t-s) \sim N(0, s)$, and $a < 0$, then the factor e^{as} suppresses the random growth exponentially. And by using the ergodic theorem for the exponential Brownian motion, we obtain

$$\lim_{t \rightarrow \infty} X(t)I(t) = -\frac{1}{a} \quad a.s.$$

Hence

$$\limsup_{t \rightarrow \infty} B(t) \leq \Lambda \left(-\frac{1}{a} \right),$$

that is

$$\limsup_{t \rightarrow \infty} B(t) \leq \frac{\Lambda}{\frac{\sigma_1^2}{2} - \rho} := \lambda_1 = B_{max}$$

Equation in the right hand side models a process which naturally falls back to it's equilibrium level of Λ .

$$\frac{dC(t)}{dt} = kB(t)D(t) - d_C C(t) - d_x E(t)C(t) + \sigma_2(t)C(t)d\beta_2(t)$$

$$\leq kB(t)D(t) - d_C C(t) + \sigma_2(t)C(t)d\beta_2(t),$$

By using $B(t) \leq \frac{\Lambda}{d_B} = \lambda_1$, and $D \leq D_{max}$, then

$$dC(t) \leq (k\lambda_1 D_{max} - d_C C(t))dt + \sigma_2(t)C(t)dB(t)$$

$$\leq (k\lambda_1\lambda_3 - d_C C(t))dt + \sigma_2(t)C(t)d\beta_2(t)$$

$$\leq (\eta - d_C C(t))dt + \sigma_2(t)C(t)d\beta_2(t),$$

where $\eta = k\lambda_1\lambda_3$. Now consider the SDE:

$$dC^* = (\eta - d_C C^*(t))dt + \sigma_2(t)C^*(t)d\beta_2(t) \text{ is provided by:}$$

$$C^*(t) = \Phi_2(t)\{I^* + \eta \int_0^t \Phi_2(\tau)^{-1} d\tau\},$$

where

$$\Phi_2(t) = \exp\left\{-\left(d_C + \frac{\sigma_2^2}{2}(t)\right)t + \sigma_2\beta_2(t)\right\}.$$

Then

$$C(t) \leq C^*(0) \exp\left\{-\left(d_C + \frac{\sigma_2^2}{2}\right)t + \sigma_2\beta_2(t)\right\}$$

$$+ \eta \int_0^t \exp\left\{(d_C + \frac{\sigma_2^2}{2})(t - \tau) - \sigma_2(\beta_2(t) - \beta_2(\tau))\right\} d\tau.$$

Hence

$$\lim_{t \rightarrow \infty} \sup C(t) \leq \frac{\eta}{d_C} =: \lambda_2 = C_{max}.$$

Similarly, the SDE

$$dD = (pC(t) - d_D D(t))dt + \sigma_3(t)D(t)d\beta_3(t)$$

$$\leq (p\lambda_2 - d_D D(t))dt + \sigma_3 D(t)d\beta_3(t).$$

Consider the SDE

$$dD^* = (p\lambda_2 - d_D D^*(t))dt + \sigma_3 D^*(t)d\beta_3(t).$$

Hence

$$\lim_{t \rightarrow \infty} \sup D(t) \leq \frac{p\lambda_2}{d_D} =: \lambda_3$$

Finally,

$$dE(t) = (qC(t) - d_E E(t))dt + \sigma_4(t)E(t)d\beta_4(t),$$

By using λ_2

$$dE(t) \leq (q\lambda_2 - d_E E(t))dt + \sigma_4(t)E(t)d\beta_4(t).$$

Now solve the SDE

$$dE^* = (q\lambda_2 - d_E E^*(t))dt + \sigma_4(t)E^*(t)d\beta_4(t),$$

$$E^*(t) = \Phi_4(t)\{E^*(0) + \int_0^t \Phi_4^{-1}(\tau)d\tau\},$$

$$\text{where } \Phi_4 = \exp\left\{-\left(d_E + \frac{\sigma_4^2}{2}(t)\right)t + \sigma_4\beta_4(t)\right\}$$

Hence,

$$E(t) \leq E^*(0) \exp \left\{ - \left(d_E + \frac{\sigma_4^2}{2}(t) \right) t + \sigma_4 \beta_4(t) \right\} \\ + q\lambda_2 \int_0^t \exp \left\{ - \left(d_E + \frac{\sigma_4^2}{2}(t) \right) (t - \tau) - \sigma_4(\beta_4(t) - \beta_4(\tau)) \right\} d\tau.$$

Consequently,

$$\lim_{t \rightarrow \infty} \sup E(t) \leq \frac{q\lambda_2}{(d_E)} =: \lambda_4.$$

So, we have:

$$\Delta = \{(B, C, D, E) \in \mathbb{R}^4 : B \leq \lambda_1, C \leq \lambda_2, D \leq \lambda_3, E \leq \lambda_4\},$$

where

$$\lambda_1 := \frac{\Lambda}{\frac{\sigma_1^2}{2} - \rho}, \lambda_2 := \frac{\eta}{d_C}, \lambda_3 := \frac{p\lambda_2}{d_D}, \text{ and } \frac{q\lambda_2}{d_E} =: \lambda_4.$$

We conclude from the above discussion that the domain region Δ is positive, and this indicates that the model in equation (3.2) is called biologically acceptable.

Biological interpretation:

The existence and uniqueness result ensures that the stochastic HIV model is well-posed and produces biologically meaningful trajectories that remain valid for all time under random environmental fluctuations.

Corollary 4.2 For the first equation of (3.2) we have two important cases:

(i) If $\rho < \frac{\sigma_1^2}{2}$, then $\lim_{t \rightarrow \infty} \sup B(t) \leq \frac{\Lambda}{\frac{\sigma_1^2}{2} - \rho}$. That is, the large noise prevents the explosion of

$B(t)$, and the solution becomes almost certainly limited.

(ii) If $\Lambda = 0$, then $\lim_{t \rightarrow \infty} B(t) = 0$ a.s. And it is the classical result of the stochastic logistic equation without an external source.

Theorem (4.3) For any initial value $X_0 = (B(0), C(0), D(0), E(0)) \in \mathbb{R}_+^4$, the solution of model (3.2) stochastically ultimately bounded permanent Here, the Markov process $(B(t), C(t), D(t), E(t)) \in \mathbb{R}_+^4$ of our stochastic model (3.2) is first shown to be geometrically ergodic, implying that exist stationary distribution exist.

Proof Let $N(t) = B(t) + C(t)$ and define the Lyapunov function

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

Applying Ito's formula, we obtain

$$dV(t) = dN(t) - \frac{1}{N^2} dN(t) + \frac{1}{N^3} (dN(t))^2.$$

By substituting the stochastic system (3.2) in to above equation and simplifying yields

$$dV(t) = \left(1 - \frac{1}{N^2}\right) \left[\Lambda + \rho B(t) \left(1 - \frac{B(t)}{K}\right) - kD(t)B(t) - d_B B(t) + kD(t)B(t) - d_C C(t) - d_x E(t)C(t) \right] dt + \left(1 - \frac{1}{N^2}\right) (\sigma_1 B d\beta + \sigma_2 C d\beta) + \frac{1}{N^3} (\sigma_1^2 B^2 + \sigma_2^2 C^2) dt.$$

Let $\mathcal{L}$ denote the generator of the stochastic system. Then

$$\begin{aligned} \mathcal{L}V &= \left(1 - \frac{1}{N^2}\right) \left[\Lambda + \rho B(t) \left(1 - \frac{B(t)}{K}\right) - d_B B(t) - d_C C(t) - d_x E(t)C(t) \right] \\ &\quad + \frac{1}{N^3} (\sigma_1^2 B^2 + \sigma_2^2 C^2) \end{aligned}$$

Using the inequality $B(t) \leq N(t)$ and $C(t) \leq N$ we obtain

$$\mathcal{L}V \leq K_1 - K_2 V(x, t) \text{ such that } K_1, K_2 > 0 \quad (4.1)$$

where the model (2.3) is uniformly elliptic, thus there exists a jointly continuous function $p: (0, \infty) \times X \times X \rightarrow (0, \infty)$ such that $P_t(X_0, Z)$ remains strictly positive for all (t, X_0, Z) , and for every measure set A ,

$$P_t(X_0, Z) = \int_A P_t(X_0, Z) dZ.$$

For $w > 0$, then if $\inf\{P_t(X_0, Z): X_0, Z \in X, |X_0|, |Z| \leq w\} \geq \hat{C}$ (a constant $\hat{C} = \hat{C}(t, w) > 0$).

For any set A ,

$$P_t(X_0, Z) = \int_A P_t(X_0, Z) dZ \geq \hat{C} \text{Leb}(A, B_w(0)) = \hat{C} \text{Leb}(B_w(0)) P(A).$$

Provides the minorization condition, where Leb denotes the Lebesgue measure and

$$\mathbb{P}(A) = \text{Leb}(A \cap B_w(0)) / \text{Leb}(B_w(0)).$$

This end the proof.

Biological interpretation:

It means HIV persists at controlled, bounded levels inside the host despite random fluctuations.

Theorem (4.4) The Markov process $(B(t), C(t), D(t), E(t)) \in \mathbb{R}^4$ of stochastic model (3.2) with initial value $X_0 = (B(0), C(0), D(0), E(0)) \in \mathbb{R}_+^4$, exhibits V -geometrically ergodicity.

Proof Let $N(t) = B(t) + C(t)$ and $V(t) = N(t) + \frac{1}{N(t)}$. Using Itos formula and (4.1),

$$\mathbb{E}e^{K_2 t} V(t) = \mathbb{E}V(0) + \mathbb{E} \left(\int_0^t e^{K_2 s} (K_2 V(s) + \mathcal{L}V(s)) ds \right)$$

$$\leq \mathbb{E}V(0) + K_1 \mathbb{E} \left(\int_0^t e^{K_2 s} ds \right)$$

$$= \mathbb{E}V(0) + \frac{K_1}{K_2} (e^{K_2 t} - 1).$$

It follows that

$$\mathbb{E}V(t) = \mathbb{E}V(0)e^{-K_2 t} + \frac{K_1}{K_2} (1 - e^{-K_2 t}) := \hat{C}.$$

We choose a big a big enough constant ς such that $\frac{\hat{c}}{\varsigma} < 1$ and apply Chebyshev's inequality to obtain

$$\mathbb{P}\left\{N(t) + \frac{1}{N(t)} > \varsigma\right\} \leq \frac{1}{\varsigma} \mathbb{E}\left\{N(t) + \frac{1}{N(t)}\right\} \leq \frac{\hat{c}}{\varsigma} := \varepsilon.$$

This implies

$$1 - \varepsilon \leq \mathbb{P}\left\{N(t) + \frac{1}{N(t)} \leq \varsigma\right\} \leq \mathbb{P}\left\{\frac{1}{\varsigma} \leq N \leq \varsigma\right\}.$$

Nothing $N^2 \leq 2|X|^2 \leq 2N^2$, we have

$$\mathbb{P}\left\{\frac{1}{\sqrt{2\varsigma}} \leq \frac{N}{\sqrt{2}} \leq |X| \leq \varsigma\right\} \geq 1 - \varepsilon.$$

According to the definition of stochastic ultimate boundedness and stochastic permanence the stochastic model (3.2) is stochastically bounded and permanent.

Biological interpretation:

The populations remain biologically controlled and do not grow without bound despite random fluctuations.

Theorem (4.5) Consider the stochastic HIV model (3.2) with initial value $X_0 = (B_0, C_0, D_0, E_0) \in \mathbb{R}_+^4$ and $t \in [0, \tau_e)$ almost surely, where τ_e the explosion time. Then system (4.1) admits a unique maximal strong local solution.

Proof

Step 1. Local existence and uniqueness. Write the system in the vector form. Put

$$X = (B(t), C(t), D(t), E(t)) \in \mathbb{R}_+^4$$

Define $F: \mathbb{R}^4 \rightarrow \mathbb{R}^4$ by $F(X) = (F_1(X), F_2(X), F_3(X), F_4(X))$, where

$$F_1(X) = \Lambda + \rho B \left(1 - \frac{B}{K}\right) - kBD - d_B B, \quad F_2(X) = kBD - d_C C - d_x EC, \quad F_3(X) = pC - d_D D, \text{ and } F_4(X) = qC - d_E E.$$

As for the diffusion matrix, it is

$$G(X) = \begin{pmatrix} \sigma_1 B & 0 & 0 & 0 \\ 0 & \sigma_2 C & 0 & 0 \\ 0 & 0 & \sigma_3 D & 0 \\ 0 & 0 & 0 & \sigma_4 E \end{pmatrix}.$$

So,

$$dX(t) = F(X(t))dt + G(X(t))dW(t),$$

where, $W(t) = (\beta_1(t), \beta_2(t), \beta_3(t), \beta_4(t))$.

Now we notice that all the components of G are polynomials in (B, C, D, E) . Therefore, F and G are locally Lipschitz on $\mathbb{R}^4$, meaning that for every $R > 0$, there exists a constant $L_R > 0$ such that if

if $\|X\| < R$, $\|Y\| < R$, then $\|F(X) - F(Y)\| + \|G(X) - G(Y)\| \leq L_R \|X - Y\|$.

Therefore, from the standard existence and uniqueness theorem for stochastic differential equations, there exists a unique strong solution $X(t)$ on the interval $[0, \tau_e)$. This proves the existence and uniqueness of the local solution.

Step 2. Positivity of $\mathbf{B}(t)$. We $\mathbf{B}(0) = \mathbf{B}_0 > \mathbf{0}$. We define the time to reach zero by

$$\tau_B = \inf\{t > 0: B(t) = 0\}.$$

We want to prove that $\mathbb{P}\{\tau_B < \tau_e\} = 0$. On the interval $0 \leq t < \tau_B$, we have $B(t) > 0$. Therefore, we can define $Y_1(t) = \ln B(t)$. By applying Itô's formula to the function $f(x) = \ln x$, we have

$$f'(x) = \frac{1}{x}, f''(x) = -\frac{1}{x^2}.$$

Hence

$$d \ln B(t) = \frac{1}{B(t)} dB(t) - \frac{1}{2} \frac{1}{B^2(t)} (dB(t))^2. \quad (4.2)$$

Since

$$dB(t) = F_1(X(t))dt + \sigma_1 B(t)d\beta_1(t),$$

Then from (4.2),

$$(dB(t))^2 = \sigma_1 B^2(t)dt.$$

So

$$d \ln B(t) = [B(t)\Lambda + \rho - \frac{\rho}{K}B(t) - kD(t) - d_B - \frac{1}{2}\sigma_1^2]dt + \sigma_1 d\beta_1(t). \quad (4.3)$$

Note that $B(t) = e^{Y_1(t)}$. As long as $Y_1(t)$ remains bounded over any finite time interval before the explosion time, then $B(t) = e^{Y_1(t)} > 0$. Thus, $B(t)$ cannot reach zero in a finite time. So $B(t) > 0, 0 \leq t < \tau_e$, a.s.

Step 3. Positivity of $\mathbf{C}, \mathbf{D}, \mathbf{E}$. We will use the same method for each variable.

The variable $\mathbf{C}(t)$. Assume that $\mathbf{C}_0 > \mathbf{0}$. Set

$$Y_2(t) = \ln C(t).$$

By applying Itô, we obtain

$$d \ln C(t) = [C(t)kB(t)D(t) - d_c - d_x E(t) - \frac{1}{2}\sigma_2^2]dt + \sigma_2 d\beta_2(t). \quad (4.4)$$

so, $C(t) = e^{Y_2(t)}$. Then $C(t) > 0$ for every $t < \tau_e$ a.s.

The variable $D(t)$. If $D_0 > 0$, set $Y_3(t) = \ln D(t)$. By applying Itô, we obtain

$$d \ln D(t) = [D(t)pC(t) - d_D - \frac{1}{2}\sigma_3^2]dt + \sigma_3 d\beta_3(t). \quad (4.5)$$

Then $D(t) = e^{Y_3(t)} > 0$. So $D(t) > 0, 0 \leq t < \tau_e$, a.s.

The variable $E(t)$. If $E_0 > 0$. Set $Y_4(t) = \ln E(t)$. By applying Itô, we obtain

$$d \ln E(t) = [E(t)qC(t) - dE - \frac{1}{2}\sigma_4^2]dt + \sigma_4 d\beta_4(t). \quad (4.6)$$

Consequently, $E(t) = e^{Y_4(t)} > 0$. Hence $E(t) > 0, 0 \leq t < \tau_e$, a.s.

Step 4. Nonnegativity when the initial value is zero. Here we must be very precise, because the HIV model we adopted allows for $\mathbf{C}_0 = \mathbf{0}, \mathbf{D}_0 = \mathbf{0}, \mathbf{E}_0 = \mathbf{0}$. Therefore, $\ln \mathbf{C}$, $\ln \mathbf{D}$, or $\ln \mathbf{E}$ should not be used at zero. Instead, we use boundary structure on the boundary limits. When $\mathbf{C} = \mathbf{0}$, we have

$$F_2(B, 0, D, E) = kBD.$$

Since $B, D \geq 0$, then $F(B, 0, D, E) \geq 0$. At the same time $\sigma_2 C = 0$ when $C = 0$. So there is no random force pushing C into the negative region, and the drift at the boundary is directed inward. Therefore $C(t) \geq 0$.

When $D = 0$, we have

$$F_3(B, C, 0, E) = pC \geq 0.$$

At the same time $\sigma_3 D = 0$. Hence $D(t) \geq 0$. When $E = 0$, we have

$$F_4(B, C, D, 0) = qC \geq 0.$$

Also $\sigma_4 E = 0$. So, $E(t) \geq 0$. When $B = 0$, we have

$$F_1(0, C, D, E) = \Lambda.$$

Assume that $\Lambda > 0$. The drift at the boundary $B = 0$ moves directly inward. Also, $\sigma_1 B = 0$ at $B = 0$. So B cannot cross the boundary into the negative region. Then $B(t) \geq 0$. Since $B_0 > 0$ and $\Lambda > 0$. We obtained the strongest result in the previous step: $B(t) > 0$.

The final result is the sum of the previous results. If it is

$$B_0 > 0, C_0 > 0, D_0 > 0, E_0 > 0,$$

then

$$(B(t), C(t), D(t), E(t)) \in \mathbb{R}^4$$

for all $0 \leq t < \tau_e$ a.s. As for whether it is $C_0, D_0, E_0 \geq 0$, then

$$(B(t), C(t), D(t), E(t)) \in \mathbb{R}_+^4$$

for all $0 \leq t < \tau_e$. a.s. And thus, the set

$$\mathbb{R}_+^4 = \{(B, C, D, E) : B, C, D, E \geq 0\}.$$

forward invariant with respect to the stochastic system (4.1). And this completes the proof. $\square$

Theorem 4.6 (Global Existence and Non-Explosion) Consider the stochastic HIV system (3.2). Assume the hypotheses of Theorem 4.5 hold. Then the unique local positive solution $\mathbf{X}(t) = (B(t), C(t), D(t), E(t))$ exists globally, i.e., $\tau_e = \infty$ a.s. Consequently, $(t) \in \mathbb{R}_+^4, t \geq 0$, a.s. Moreover, for every finite $T > 0$,

$$\mathbb{E}[\sup_{0 \leq t \leq T} (B(t) + C(t) + D(t) + E(t))] < \infty.$$

Proof We will prove the result in several steps.

Step 1. Choosing Lyapunov function. Consider

$$V(B, C, D, E) = B + C + D + E. \quad (4.8)$$

This function is very suitable for the biological model because it represents, up to a scaling constant, the total size of the viral cellular population. Because all the variables are non-negative, $V(X) \geq 0$. As well as $V(X) \rightarrow \infty$ when $\|X\| \rightarrow \infty$.

So, V is a radially unbounded Lyapunov function.

Step 2. Applying Itô's formula.

Since

$$V(B, C, D, E) = B + C + D + E,$$

then

$$V_B = V_C = V_D = V_E = 1,$$

and all second derivatives equal zero. Therefore, there are no second-order Itô limits. Then

$$dV(t) = dB(t) + dC(t) + dD(t) + dE(t).$$

With compensation from the system, we obtain

$$\begin{aligned} dV(t) = & [\Lambda + \rho B - \frac{\rho}{K} B^2 - kBD - d_B B + kBD - d_C C - d_x EC + pC - d_D D + qC - d_E E] dt \\ & + \sigma_1 B d\beta_1 + \sigma_2 C d\beta_2 + \sigma_3 D d\beta_3 + \sigma_4 E d\beta_4. \end{aligned}$$

Here an important feature of the model appears:

$$-kBD + kBD = 0.$$

That is, the transfer of healthy cells to the infected cells does not directly change the total number. Therefore

$$\begin{aligned} dV(t) = & [\Lambda + \rho B - \frac{\rho}{K} B^2 - d_B B + (p - dC + q)C - d_D D - d_x EC - d_E E] dt \\ & + \sum_{i=1}^4 \sigma_i X_i(t) d\beta_i(t), \end{aligned}$$

where $X_1 = B, X_2 = C, X_3 = D, X_4 = E$.

Step 3. Estimation of the drift limit

We use first $-\frac{\rho}{K}B^2 \leq 0$, also $-d_x EC \leq 0$. So

$$LV \leq \Lambda + \rho B - d_B B + (p - dC + q)C - d_D D - d_E E.$$

Since

$$B \leq V, C \leq V, D \leq V, E \leq V,$$

There exists a positive constant c_0 such that

$$LV \leq c_0(1 + V). \quad (4.9)$$

For example, one can choose

$$c_0 = \max\{\Lambda, \rho + d_B, p + q + d_C, d_D, d_E\}.$$

then

$$LV \leq c_0(1 + V).$$

Step 4. Expectation estimation

We take the expectation from the previous equation. And since the random integrals have a mean of zero,

$$\mathbb{E} \int_0^t \sigma_i X_i(t) d\beta_i(s) = 0,$$

we get

$$\mathbb{E}V(t) \leq V(0) + c_0 t + c_0 \int_0^t \mathbb{E}V(s) ds.$$

Using Grönwall's inequality,

$$\mathbb{E}V(t) \leq (V(0) + c_0 t)e^{c_0 t}. \quad (4.10)$$

Consequently,

$$\mathbb{E}V(t) < \infty, \text{ for every } t < \infty.$$

Step 5. Exclusion of the explosion

Define the stop time.

$$\tau_n = \inf\{t \geq 0: V(X(t)) \geq n\}.$$

If an explosion occurs in a finite time, it must $\tau_n \rightarrow \tau_e < \infty$. Take $T > 0$. From the definition of τ_n .

$$V(X(\tau_n \wedge T)) \geq n$$

on the event $\{\tau_n \leq T\}$. So

$$n\mathbb{P}\{\tau_n \leq T\} \leq \mathbb{E}V(X(\tau_n \wedge T)).$$

And using the previous estimate, we obtain a constant $C_T < \infty$ such that

$$\mathbb{P}\{\tau_n \leq T\} \leq \frac{C_T}{n}. \text{ Then } \lim_{n \rightarrow \infty} \mathbb{P}\{\tau_n \leq T\} = 0. \text{ Since}$$

$$\{\tau_e \leq T\} \subseteq \bigcap_{n=1}^{\infty} \{\tau_n \leq T\},$$

then $\mathbb{P}\{\tau_e \leq T\} = 0$. Since $T > 0$ is arbitrary, $\mathbb{P}\{\tau_e \leq \infty\} = 0$. Hence $\tau_e = \infty$ a.s. this prove the existences of the global solution.

Biological interpretation:

This result shows the despite stochastic perturbations, the HIV system remains stable and bounded, with persistent virus-immune interaction converging to a long-term stochastically behavior.

Theorem (4.8) The model (3.2) has unique global positive solution $(B(t), C(t), D(t), E(t))$ for $t \in [0, \tau_e)$

and for every initial value $(B_0, C_0, D_0, E_0) \in \mathbb{R}_+^4$. Moreover, the solution will remain in $\mathbb{R}_+^4$ almost surely.

Proof In order to proof the existence of the global solution, it is sufficient to show that the explosion time $\tau_e = \infty$ almost surely. If $K_0 \in \mathbb{Z}_+$ is a sufficient large such that (B_0, C_0, D_0, E_0) lies in the closed ball $B(K_0) \subseteq \mathbb{R}_+^4$. For $K \geq K_0$, we take and describe the stop- time as

$$\tau_k = \inf\{t \in [0, \tau_e): B \notin \left(\frac{1}{K}, K\right) \text{ or } E \notin \left(\frac{1}{K}, K\right)\}.$$

Assume that $\inf \emptyset = \infty$ (as usual, $\emptyset = \text{null set}$). According to the description of stopping time, we can know that $k \rightarrow \infty$. Let $\lim_{k \rightarrow \infty} \tau_k = \tau_\infty$, then, $\tau_\infty < \tau_e$ almost surely. If $\mathbb{P}\{\tau_\infty = \infty\} = 1$, then $\mathbb{P}\{\tau_e = \infty\} = 1$. If this declaration false, i.e., $\tau_\infty \neq \infty$, then $T > 0$ and $\varepsilon \in (0, 1)$. And they satisfy $\mathbb{P}\{\tau_\infty \leq T\} > \varepsilon$. Thus there exists an integer $k_1 \geq k_0$, so that $\mathbb{P}\{\tau_1 \leq T\} > \varepsilon$. Define C^2 - function as the function continues second derivatives. So we construct a C^2 - function $V: \mathbb{R}_+^4 \rightarrow \mathbb{R}_+ \cup \{\infty\}$ as follows. Let

$$V(B, C, D, E) = \sum_{X \in (B, C, D, E)} (X(t) - 1 - \ln X(t))$$

$$V(B, C, D, E) = (B(t) - 1 - \ln B(t)) + (C(t) - 1 - \ln C(t))$$

$$+ (D(t) - 1 - \ln D(t)) + (E(t) - 1 - \ln E(t)).$$

The partial derivative for V for all variables

$$\frac{\partial V}{\partial B} = 1 - \frac{1}{B}, \frac{\partial V}{\partial C} = 1 - \frac{1}{C}, \frac{\partial V}{\partial D} = 1 - \frac{1}{D}, \frac{\partial V}{\partial E} = 1 - \frac{1}{E}.$$

So

$$dV = \sum_{X \in (B, C, D, E)} \left(1 - \frac{1}{X}\right) dX + \frac{1}{2} \sum_{X \in (B, C, D, E)} \frac{1}{X^2} (\sigma_X X)^2 dt,$$

and

$$dX = f_X dt + \sigma_X X d\beta_X.$$

Then

$$\begin{aligned} dV &= \sum_{X \in (B, C, D, E)} \left(1 - \frac{1}{X}\right) dX + \frac{1}{2} \sum_{X \in (B, C, D, E)} \frac{1}{X^2} (\sigma_X X)^2 dt \\ dV &= \sum_X \left(1 - \frac{1}{X}\right) f_X dX + \frac{1}{2} \sum_X (\sigma_X)^2 dt + \sum_X \left(1 - \frac{1}{X}\right) \sigma_X X d\beta_X \\ dV &= \frac{\partial V}{\partial B} dB + \frac{\partial V}{\partial C} dC + \frac{\partial V}{\partial D} dD + \frac{\partial V}{\partial E} dE + \frac{1}{2} (\sigma_1^2 + \sigma_2^2 + \sigma_3^2 + \sigma_4^2) = \\ &= \left[\left(1 - \frac{1}{B}\right) \left(\Lambda + \rho B(t) \left(1 - \frac{B(t)}{K}\right) - kD(t)B(t) - d_B B(t) \right) + \left(1 - \frac{1}{C}\right) (PC(t) - d_V D(t)) + \right. \\ &\quad \left. \left(1 - \frac{1}{D}\right) (PC(t) - d_V D(t)) + \left(1 - \frac{1}{E}\right) (qC(t) - d_E E(t)) + \frac{1}{2} (\sigma_1^2 + \sigma_2^2 + \sigma_3^2 + \sigma_4^2) \right] dt \\ &\quad + \left(1 - \frac{1}{B}\right) B \sigma_1 d\beta_1 + \left(1 - \frac{1}{C}\right) C \sigma_2 d\beta_2 + \left(1 - \frac{1}{D}\right) D \sigma_3 d\beta_3 + \left(1 - \frac{1}{E}\right) E \sigma_4 d\beta_4. \\ dV &= f(B, C, D, E) dt + g(B, C, D, E) d\beta \end{aligned}$$

where,

$$\begin{aligned} f(B, C, D, E) &= \left(1 - \frac{1}{B}\right) \left(\Lambda + \rho B(t) \left(1 - \frac{B(t)}{K}\right) - kD(t)B(t) - d_B B(t) \right) \\ &\quad + \left(1 - \frac{1}{C}\right) (PC(t) - d_V D(t)) + \left(1 - \frac{1}{D}\right) (PC(t) - d_V D(t)) \\ &\quad + \left(1 - \frac{1}{E}\right) (qC(t) - d_E E(t)) + \frac{1}{2} (\sigma_1^2 + \sigma_2^2 + \sigma_3^2 + \sigma_4^2) \end{aligned}$$

and

$$\begin{aligned} g(B, C, D, E) &= \left(1 - \frac{1}{B}\right) B \sigma_1 d\beta_1 + \left(1 - \frac{1}{C}\right) C \sigma_2 d\beta_2 + \left(1 - \frac{1}{D}\right) D \sigma_3 d\beta_3 \\ &\quad + \left(1 - \frac{1}{E}\right) E \sigma_4 d\beta_4 \end{aligned}$$

Then, There is $M > 0$, so $\|f(B, C, D, E)\| \leq M$, for all $(B, C, D, E) \in \mathbb{R}_+^4$. It follows that

$$\int_0^{\tau_m \wedge t} dV(B, C, D, E) \leq \int_0^{\tau_m \wedge t} M dt + \int_0^{\tau_m \wedge t} g(B, C, D, E) d\beta$$

Where $\{\tau_K \wedge T\} = \min\{\tau_K, T\}$. Consider the aforementioned inequality Exceptions, yield

$$= \mathbb{E}V(B(\tau_K \wedge t), C(\tau_K \wedge t), D(\tau_K \wedge t), E(\tau_K \wedge t)) \leq V(B(0), C(0), D(0), E(0)) + MT.$$

Note that no one less than one of $(B(\tau_K \wedge t), C(\tau_K \wedge t), D(\tau_K \wedge t))$ and $E(\tau_K \wedge t)$ lies in $(K, \frac{1}{K})$ for every $w \in \Omega_k$, consequently,

$$V(B(\tau_K \wedge t), C(\tau_K \wedge t), D(\tau_K \wedge t), E(\tau_K \wedge t)) \geq (k - 1 - \ln k) \Lambda \left(\frac{1}{k} - 1 - \ln \frac{1}{k} \right)$$

Hence, from 27

$$\begin{aligned} & \mathbb{E}V(B(\tau_k \wedge t), C(\tau_k \wedge t), D(\tau_k \wedge t), E(\tau_k \wedge t)) \\ & \geq \mathbb{E}I_{\Omega_k}(k - 1 - \ln k) \wedge \left(\frac{1}{k} - 1 - \ln \frac{1}{k}\right) \\ & \geq \varepsilon(k - 1 - \ln k) \wedge \left(\frac{1}{k} - 1 - \ln \frac{1}{k}\right). \end{aligned}$$

Here I_{Ω_k} the characteristic function of Ω_k . Take $k \rightarrow \infty$ implies to a contradiction:

$$\infty > V(B(0), C(0), D(0), E(0)) + MT = \infty.$$

Therefore, $\mathbb{P}\{\tau_\infty = \infty\} = 1$. Thus,

$$\mathbb{P}\{(B(t), C(t), D(t), E(t)) \in \mathbb{R}_+^4 : t \geq 0\} = 1.$$

Biological interpretation:

The results show that the stochastic HIV model remains biologically valid with unique global positive solution, preserving positivity under random environmental fluctuations.

5. DISEASE STOCHASTIC EXTINCTIONS

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

$$R_1^s = \frac{kp\Lambda}{d_B \left(d_C + \frac{\sigma_2^2}{2}\right) \left(d_D + \frac{\sigma_3^2}{2}\right)}$$

If $R_1^s < 1$ implies to almost surly extension.

Theorem (5.1) Assume that $R_1^s < 1$, then the solution $(B(t), C(t), D(t), E(t))$ of the model (3.2) has the following property:

$$\begin{aligned} & \lim_{t \rightarrow \infty} \sup \frac{1}{t} \int_0^t B(s) ds \leq \frac{\Lambda}{\mu} \quad , \quad \lim_{t \rightarrow \infty} \sup \frac{\log C(t)}{t} < \infty \quad , \quad \lim_{t \rightarrow \infty} \sup \frac{\log D(t)}{t} < 0 \quad \text{and} \\ & \lim_{t \rightarrow \infty} \sup \frac{\log E(t)}{t} < 0. \end{aligned}$$

The theorem demonstrates that infection in that four compartment HIV model dies out with probability 1, as both infected cells and viral load gradually decay to zero, while susceptible cells remain within their natural bounds. This reflects the body ability to completely control the infection and maintain long-term immunological stability, even under stochastic fluctuations.

Proof Consider the first equation of (3.2)

$$dB = \left(\Lambda + \rho B(t) \left(1 - \frac{B(t)}{K} \right) - kD(t)B(t) - d_B B(t) \right) dt + \sigma_1 B(t) d\beta_1(t) \quad (5.1)$$

with the initial value $(0) = B_0 > 0$, and setting with

$$dB \leq (\Lambda - d_B B(t)) dt + \sigma_1 B(t) d\beta_1(t),$$

$f(B(t)) = \Lambda - d_B B(t)$ and $(B(t)) = \sigma_1 B(t)$, $B(t) \in (0, \infty)$.

We calculate that

$$\int_{a_0}^{B(t)} \frac{g(u)}{\sigma^2(u)} du = \int_{a_0}^{B(t)} \frac{1}{\sigma_1^2 u^2} (\Lambda - d_B u) du = \frac{1}{\sigma_1^2} \left(-\frac{\Lambda}{B} - d_B \ln B(t) \right) + C_1,$$

$$\int_{a_0}^{B(t)} \frac{f(u)}{g^2(u)} du = \int_{a_0}^{B(t)} \frac{1}{\sigma_1^2 u^2} (\Lambda - d_u u) du = \frac{1}{\sigma_1^2} \left(-\frac{\Lambda}{B} - d_B \ln B(t) \right) + C_1$$

where a_0 is an arbitrary fixed positive constant and C_1 is constant determined by the foregoing formula. It is evident that

$$\begin{aligned} \int_0^\infty \frac{1}{g^2(B)} e^{\int_{a_0}^B \frac{f(u)}{g^2(u)} du} dB &= \int_0^\infty \frac{1}{\sigma_1^2 B^2} e^{2\left(\frac{1}{\sigma_1^2}\left(-\frac{\Lambda}{B} - d_B \ln B(t)\right) + C_1\right)} dB \\ &= \frac{e^{2C_1}}{\sigma_1^2} \int_0^\infty B^{-\frac{2d_B}{\sigma_1^2} - 2} e^{-\frac{2\Lambda}{\sigma_1^2 B}} dB < \infty, \end{aligned}$$

thus, we obtain that the model (5.1) is ergodic with the ergodic distribution

$$K(B) = \frac{e^{2C_1}}{\sigma_1^2} B^{-\frac{2d_B}{\sigma_1^2} - 2} e^{-\frac{2\Lambda}{\sigma_1^2 B}} dB, B(t) \in (0, \infty),$$

Where $C_2 \triangleq \frac{e^{2C_1}}{\sigma_1^2}$ is a constant such that $\int_0^\infty BK(B)dB = 1$. It then follows that

$$\int_0^\infty BK(B) dB = \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t B(s) ds,$$

almost surly and from Theorem (3.1) of [28] we have $\int_0^\infty BK(B)dB = \frac{\Lambda}{B}$.

Letting $B(t)$ be the solution of (5.1) with the initial value $B(0) = B_0 > 0$, by comparison theorem for stochastic differential equations [34].Applying Itô's formula yields

$$\begin{aligned} d \ln C &= \left(\frac{kB(t)D(t)}{C(t)} - d_C - d_x E(t) - \frac{1}{2} \sigma_2^2 \right) dt - \sigma_2(t) d\beta_2(t) \\ &\leq kB(t)D(t) - d_C - d_x E(t) - \frac{1}{2} \sigma_2^2 dt + \sigma_2(t) d\beta_2(t) \end{aligned} \quad (5.2)$$

Hence from equation (5.2), we derive the following equation by integrating both side from 0 tot

$$\begin{aligned} \ln C(t) &= \ln C(0) + \int_0^t \frac{kB(\tau)D(\tau)}{C(\tau)} - d_C - d_x E(\tau) - \frac{1}{2} \sigma_2^2 d\tau + \sigma_2(\tau) d\beta_2(\tau) \\ &\leq \ln C(0) + \int_0^t \left(kB(\tau)D(\tau) - d_C - d_x E(\tau) - \frac{1}{2} \sigma_2^2 \right) d\tau + \int_0^t \sigma_2(\tau) d\beta_2(\tau) \end{aligned} \quad (5.3)$$

Setting $\phi(t) := \int_0^t \sigma_2 d\beta_2(\tau)$, we have

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

Hence, using the law of large numbers for martingales, we obtain $\limsup_{t \rightarrow \infty} \frac{\phi(t)}{t} = 0$ a.s. It follows from (5.2) the dividing by t on both sides of (5.3) and letting $t \rightarrow \infty$, we derive

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \ln C(t) \leq kD \frac{\Lambda}{B} - \left((d_C + d_x E + \frac{1}{2} \sigma_2^2) \right) < \limsup_{t \rightarrow \infty} \frac{1}{t} \ln C(t) \leq k \frac{\Lambda}{B} - \left(d_C + \frac{1}{2} \sigma_2^2 \right) < 0. \text{ a.s.}$$

Hence from equation (5.2), we derive the following equation by integrating both side from 0 tot

$$\begin{aligned} \lim_{t \rightarrow \infty} \frac{\ln C(t)}{t} &\leq \frac{1}{t} \int_0^t \left(kB(\tau)V(\tau) - d_C - d_x E(\tau) - \frac{1}{2} \sigma_2^2 \right) d\tau + \frac{1}{t} \int_0^t \sigma_2(\tau) d\beta_2(\tau) d\tau \\ &\limsup_{t \rightarrow \infty} \frac{1}{t} \ln C(t) \\ &\leq \limsup_{t \rightarrow \infty} \frac{1}{t} \left(\int_0^t \left(kB(t)V(t) - d_C C(t) - d_x E(t)C(t) - \frac{1}{2} \sigma_2^2 \right) dt + \int_0^t \sigma_2(\tau) d\beta_2(\tau) d\tau \right) \\ &\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t \left(kB(t)V(t) - d_C C(t) - d_x E(t)C(t) - \frac{1}{2} \sigma_2^2 \right) dt \\ &\leq kD \frac{\Lambda}{d_B} - (d_C + d_x E(t)C(t) + \frac{1}{2} \sigma_2^2) \\ &< k \frac{\Lambda}{d_B} - (d_C C(t) + d_x E(t)C(t) + \frac{1}{2} \sigma_2^2) < 0 \end{aligned}$$

Likewise,

$$d \ln D = \left(\frac{pC(t)}{D(t)} - d_D - \frac{1}{2} \sigma_3^2 \right) dt + \sigma_3(t) d\beta_3(t) \leq \left(pC(t) - d_D - \frac{1}{2} \sigma_3^2 \right) dt + \sigma_3(t) d\beta_3(t)$$

Therefore, setting $\varphi_2(t) := \int_0^t \sigma_3^2(\tau) d\tau$, we have

$$\langle \varphi_2(t), \varphi_2(t) \rangle = \frac{1}{t} \int_0^t \sigma_3^2 ds = \sigma_3^2 < \infty.$$

Hence, using the law of large numbers for martingales, we obtain $\limsup_{t \rightarrow \infty} \frac{\varphi_2(t)}{t} = 0$ a.s. It follows from (5.2) the dividing by t on both sides of (5.3) and letting $t \rightarrow \infty$, we derive

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \ln D(t) \leq p \left(k \frac{\Lambda}{B} - \left((d_C + \frac{1}{2} \sigma_2^2) + (d_D + \frac{1}{2} \sigma_3^2) \right) \right) < 0. \text{ a.s.}$$

Let $\varphi_2(t) = \ln E(t)$. By using Itô's formula

$$d \ln E(t) = \left(\frac{qC(t)}{E(t)} - d_E - \frac{1}{2} \sigma_4^2 \right) dt + \sigma_4(t) d\beta_4(t). \quad (5.4)$$

Hence from equation (5.4), we derive the following equation by integrating both side from 0 tot

$$\begin{aligned} \ln E(t) &= \ln E(0) + \int_0^t \left(\frac{qC(\tau)}{E(\tau)} - d_E - \frac{1}{2} \sigma_4^2 \right) d\tau + \int_0^t \sigma_4(\tau) d\beta_4(\tau) \\ &\leq \ln E(0) + \int_0^t \left(qC(\tau) - d_E - \frac{1}{2} \sigma_4^2 \right) d\tau + \int_0^t \sigma_4(\tau) d\beta_4(\tau). \end{aligned} \quad (5.3)$$

Setting $\varphi_4(t) := \int_0^t \sigma_4^2(\tau) d\tau$, we have

$$\langle \varphi_4(t), \varphi_4(t) \rangle = \frac{1}{t} \int_0^t \sigma_4^2 ds = \sigma_4^2 < \infty.$$

Hence, using the law of large numbers for martingales, we obtain $\limsup_{t \rightarrow \infty} \frac{\varphi_4(t)}{t} = 0$ a.s. It follows from (5.4) the dividing by t on both sides of (5.4) and letting $t \rightarrow \infty$, we derive

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \log E(t) \leq \left(qC(t) - (d_E + \frac{1}{2} \sigma_4^2) \right) < 0. \text{ a.s.}$$

Remark (5.2) Theorem (5.1) provides sufficient conditions for disease to be eradicated in the long run. Due to the fact that $R_1^s < R_0$, we are pleasantly surprised to discover a scenario where $R_0 > 1$ but $R_1^s < 1$ such that the asymptotically infected population $C(t)$ of the SDE model (3.2) become extension, which is quite differential from the result of Theorem (5.1) for deterministic model (3.1). This tells us that the impact of environmental noise on disease dynamics must not be ignored.

6. NUMERICAL SIMULATION

The stochastic HIV model is driven by introducing white noise terms $\sigma_i d\beta_i(t)$ to the state variable $B(t)$, $C(t)$, $D(t)$ and $E(t)$. Here $\beta_i(t), i = 1, 2, 3$, are independent standard Brownian motion, and σ_i represent the intensities of environmental stochastic effects. This leads to a stochastic differential equation system in the Ito sense. Thus, the system can be written as follows:

$$B_{n+1} = B_n + \Lambda + \rho B_n(t) \left(1 - \frac{B_n(t)}{K} \right) - k B_n D_n - d_B B_n + \sigma_1 B_n \sqrt{\Delta t} \xi_{n,1} + \frac{\sigma_1^2}{2} (\xi_{n,1}^2 - 1) \Delta t B_n,$$

$$C_{n+1} = C_n + k B_n D_n - d_C C_n - d_x E_n C_n + \sigma_2 C_n \sqrt{\Delta t} \xi_{n,2} + \frac{\sigma_2^2}{2} (\xi_{n,2}^2 - 1) \Delta t C_n,$$

$$D_{n+1} = D_n + p C_n - d_D D_n + \sigma_3 D_n \sqrt{\Delta t} \xi_{n,3} + \frac{\sigma_3^2}{2} (\xi_{n,3}^2 - 1) \Delta t D_n,$$

$$E_{n+1} = E_n + q C_n - d_E E_n + \sigma_4 E_n \sqrt{\Delta t} \xi_{n,4} + \frac{\sigma_4^2}{2} (\xi_{n,4}^2 - 1) \Delta t E_n.$$

We will explain how to choose parameter values. We will choose values close to the known clinical data for the early HIV infection stage. The proposed initial values:

Baseline Scenario:

$$B(0) = 1000 \text{ cell/mm}^3, C(0) = 0, D(0) = 10 \text{ copies/mm}^3, E(0) = 5 \text{ cell/mm}^3$$

Interpretation:

- A healthy person has approximately 800–1200 CD4+ cells/mm³.
- At the moment the virus enters, there may not be any infected cells yet.
- The initial viral load is small.
- CTL response is still weak.

Now choosing noise coefficients, in published HIV models, the values are often used:

$$0.01 \leq \sigma_i \leq 0.30$$

to avoid losing positivity.

Numerical methodology: The Milstein scheme was used for the random case with multiplicative noise for each variable $X_{n+1} = X_n + f(X_n)\Delta t + \sigma X_n \sqrt{\Delta t}$:

$$\xi_n + \frac{1}{2} \sigma^2 X_n (\xi_{n-1}^2 - 1) \Delta t.$$

Then, non-negativity was imposed after each step because the variables represent vital concentrations.

Important notice: The shapes use normalization for each variable between 0 and 1 so that the differences in scales do not hide any of the curves.

Definition of the five cases:

The case	Initial values	The intensity of the noise $\sigma_1, \sigma_2, \sigma_3, \sigma_4$	Biological modification
Case 1: Weak environmental fluctuation	$B_0 = 1000, C_0 = 0, D_0 = 10, E_0 = 1$	0.02, 0.02, 0.02, 0.02	Essential
Case 2: Moderate random noise	$B_0 = 1000, C_0 = 0, D_0 = 10, E_0 = 1$	0.06, 0.06, 0.06, 0.06	Essential
Case 3: High primary viral load	$B_0 = 1000, C_0 = 0, D_0 = 60, E_0 = 1$	0.05, 0.05, 0.07, 0.05	Essential
Case 4: Strong CTL response	$B_0 = 1000, C_0 = 0, D_0 = 20, E_0 = 20$	0.04, 0.04, 0.05, 0.04	$d_x = 0.025, q = 1.2$
Case 5: Weak immune removal	$B_0 = 1000, C_0 = 0, D_0 = 20, E_0 = 0.5$	0.06, 0.08, 0.08, 0.06	$d_x = 0.003, q = 0.4$

These values are balanced, numerically stable, and usually provide realistic curves for both deterministic and stochastic models.

How do we choose K ? In healthy adults: 800–1200 CD4 cells/mm³, so I suggest: $K = 1200$.

Case 1: Weak environmental fluctuation

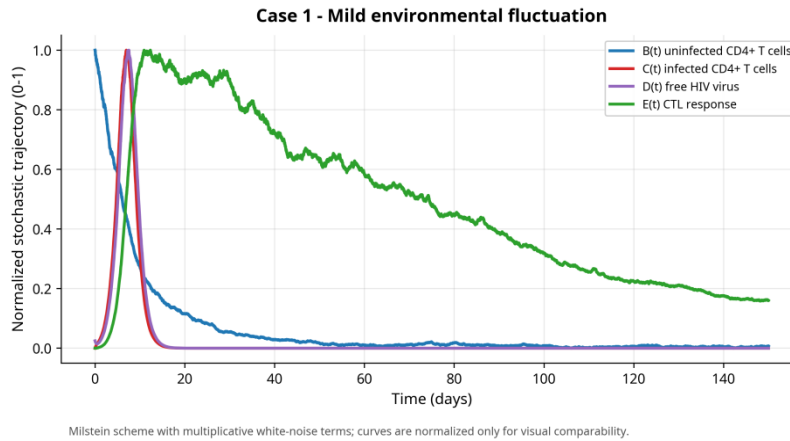


Figure 1. Four colored curves in a single figure for the random case: B, C, D, E.

Biological interpretation: There is a limited drop in healthy cells B with a short viral peak; then the immune response E gradually grows, reducing the infected cells C and the virus D to a low level.

Raw numerical indicators: Minimum $B \approx 90.76$, Maximum $C \approx 33.17$, Maximum $D \approx 403.81$, Maximum $E \approx 129.63$.

Case 2: Moderate random noise

Biological interpretation: Noise increases the roughness of pathways around the general trend; biologically, this represents daily variations in exposure and environment, with immune control remaining possible after a transient viral spike.

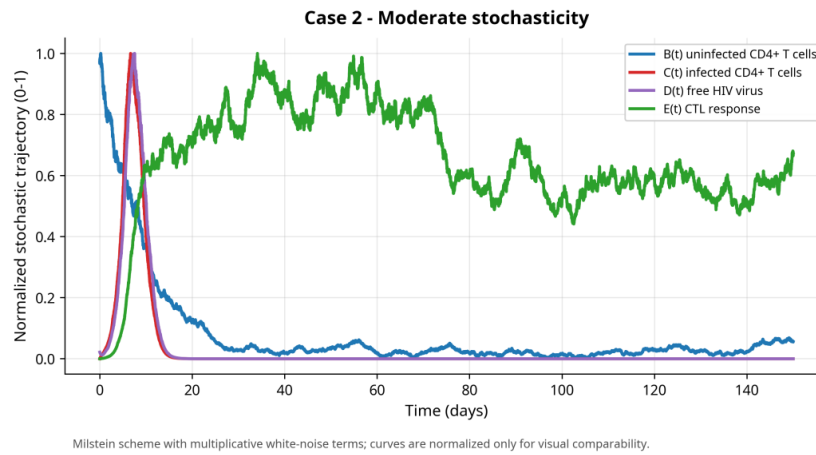


Figure 2. Four colored curves in a single figure for the random case: B , C , D , E .

Raw numerical indicators: Minimum $B \approx 77.83$, Maximum $C \approx 37.85$, Maximum $D \approx 462.69$, Maximum $E \approx 205.25$.

Case 3: High primary viral load

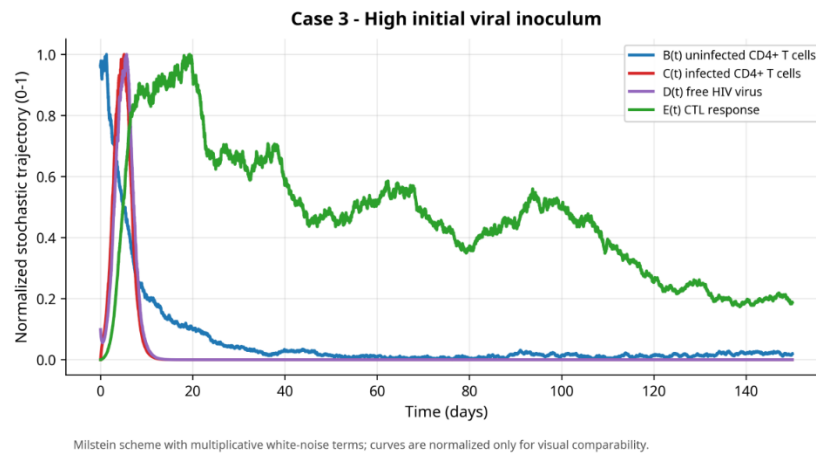


Figure 3. Four colored curves in a single shape for the random case: B , C , D , E .

Biological interpolation: The large initial viral load produces an earlier wave that is more severe than D and C , so B drops faster. Later, CTLs are stimulated, causing E to rise and suppress the infection.

Raw numerical indicators: Minimum $B \approx 84.93$, Maximum $C \approx 47.79$, Maximum $D \approx 607.29$, Maximum $E \approx 182.44$.

Case 4: Strong CTL response

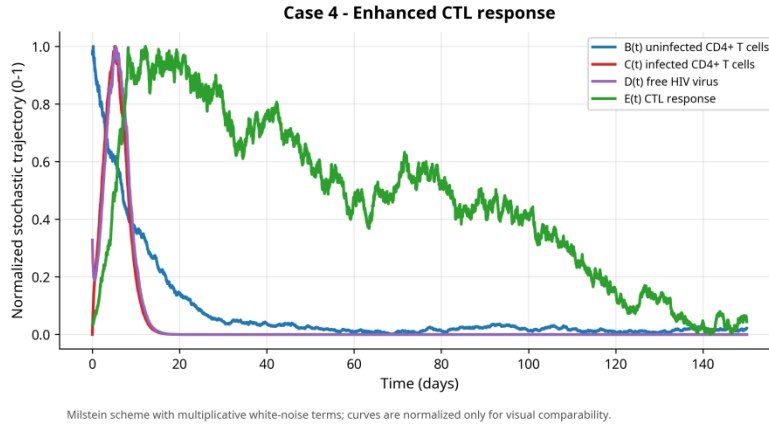


Figure 4. Four colored curves in a single shape for the random case: B, C, D, E .

Biological explanation: The initial increase in E and the increased removal efficiency of d_x accelerate the elimination of C ; therefore, the peak of D is shorter and weaker, and B recovers to a relatively higher level.

Raw numerical indicators: Minimum $B \approx 83.11$, Maximum $C \approx 4.99$, Maximum $D \approx 61.02$, Maximum $E \approx 56.12$.

Case 5: Weak immune removal

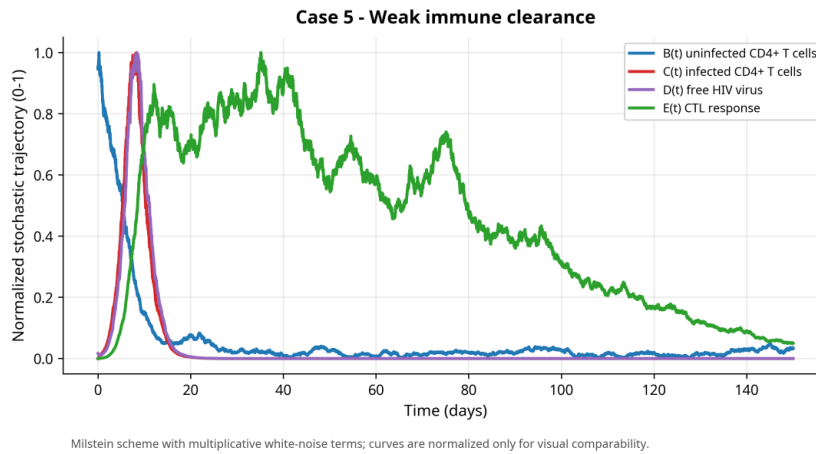


Figure 5. Four colored curves in a single shape for the random case: B, C, D, E .

Biological explanation: The reduced production/effectiveness of CTLs allows C and D to persist longer and with greater fluctuations; this explains the more pronounced decrease in B and the delayed control of the infection.

Raw numerical indicators: Minimum $B \approx 72.88$, Maximum $C \approx 98.05$, Maximum $D \approx 1211.75$, Maximum $E \approx 252.05$.

7. DISCUSSION AND CONCLUSIONS

The deterministic model describes HIV dynamics through a system of ordinary differential equations in which the evolution of all compartments is fully determined by the initial conditions and parameter values. The system exhibits predictable behavior, and its long-term dynamics are governed by equilibrium points determined mainly by threshold parameter R_0 . The stochastic model (3.2) is constructed as an extension of deterministic system by introducing multiplicative white noise via independent Brownian motions. This modification is directly incorporated into the original model structure to represent environmental and biological fluctuations. Consequently, the system dynamic becomes random, and the qualitative behavior is characterized in terms of stochastic stability, mean-square properties, and probabilistic long-term outcomes rather than deterministic equilibria. Thus, the stochastic formulation represents a perturbed version of the deterministic model, where random effects modify the trajectory behavior without changing the underlying biological structure of the system.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

REFERENCES

- [1] World Health Organization, HIV and AIDS, <https://www.who.int/news-room/fact-sheets/detail/hiv-aids>.
- [2] P.K. Srivastava, P. Chandra, Modeling the Dynamics of HIV and $CD4^+$ T Cells During Primary Infection, *Nonlinear Anal. Real World Appl.* 11 (2010), 612–618. <https://doi.org/10.1016/j.nonrwa.2008.10.037>.
- [3] E.A. Hernandez-Vargas, R.H. Middleton, Modeling the Three Stages in HIV Infection, *J. Theor. Biol.* 320 (2013), 33–40. <https://doi.org/10.1016/j.jtbi.2012.11.028>.
- [4] Y. Dong, W. Ma, Global Properties for a Class of Latent HIV Infection Dynamics Model with CTL Immune Response, *Int. J. Wavelets Multiresolution Inf. Process.* 10 (2012), 1250045. <https://doi.org/10.1142/s0219691312500452>.
- [5] M.A. Nowak, R.M. May, *Virus Dynamics: Mathematical Principles of Immunology and Virology*, Oxford University Press, Oxford, 2000. <https://doi.org/10.1093/oso/9780198504184.001.0001>.
- [6] B.D. Hassard, N.D. Kazarinoff, Y.-H. Wan, *Theory and Applications of Hopf Bifurcation*, London Math. Soc. Lecture Note Ser. 41, Cambridge University Press, Cambridge, 1981.

- [7] S. Prakash, A.K. Umrao, P.K. Srivastava, Dynamical Model of HIV Infection with Homeostatic Growth of $CD4^+$ T Cells and Immune Response, *Int. J. Biomath.* 18 (2025), 2450026.
<https://doi.org/10.1142/s1793524524500268>.
- [8] P.K. Srivastava, P. Chandra, Hopf Bifurcation and Periodic Solutions in a Dynamical Model for HIV and Immune Response, *Differ. Equ. Dyn. Syst.* 16 (2008), 77–100. <https://doi.org/10.1007/s12591-008-0006-2>.
- [9] S. Freeman, *Biological Science*, 3rd ed., Benjamin Cummings, 2007.
- [10] Q. Li, F. Cong, T. Liu, Y. Zhou, Stationary Distribution of a Stochastic HIV Model with Two Infective Stages, *Phys. A* 554 (2020), 124686. <https://doi.org/10.1016/j.physa.2020.124686>.
- [11] I.M. Longini, W.S. Clark, M. Haber, R. Horsburgh, The Stages of HIV Infection: Waiting Times and Infection Transmission Probabilities, in: C. Castillo-Chavez, (ed) *Mathematical and Statistical Approaches to AIDS Epidemiology*, Lecture Notes in Biomathematics, Springer, Berlin, Heidelberg, pp. 111–137, (1989).
https://doi.org/10.1007/978-3-642-93454-4_5.
- [12] R. Naresh, A. Tripathi, D. Sharma, A Nonlinear HIV/AIDS Model with Contact Tracing, *Appl. Math. Comput.* 217 (2011), 9575–9591. <https://doi.org/10.1016/j.amc.2011.04.033>.
- [13] H.-F. Huo, R. Chen, X.-Y. Wang, Modelling and Stability of HIV/AIDS Epidemic Model with Treatment, *Appl. Math. Model.* 40 (2016), 6550–6559. <https://doi.org/10.1016/j.apm.2016.01.054>.
- [14] C.C. McCluskey, A Model of HIV/AIDS with Staged Progression and Amelioration, *Math. Biosci.* 181 (2003), 1–16. [https://doi.org/10.1016/s0025-5564\(02\)00149-9](https://doi.org/10.1016/s0025-5564(02)00149-9).
- [15] J.M. Hyman, J. Li, The Reproductive Number for an HIV Model with Differential Infectivity and Staged Progression, *Linear Algebra Appl.* 398 (2005), 101–116. <https://doi.org/10.1016/j.laa.2004.07.017>.
- [16] A.J. Marks, D. Pillay, A.R. McLean, The Effect of Intrinsic Stochasticity on Transmitted HIV Drug Resistance Patterns, *J. Theor. Biol.* 262 (2010), 1–13. <https://doi.org/10.1016/j.jtbi.2009.09.017>.
- [17] L. Cai, S. Guo, S. Wang, Analysis of an Extended HIV/AIDS Epidemic Model with Treatment, *Appl. Math. Comput.* 236 (2014), 621–627. <https://doi.org/10.1016/j.amc.2014.02.078>.
- [18] F. Rao, P.S. Mandal, Y. Kang, Complicated Endemics of an SIRS Model with a Generalized Incidence under Preventive Vaccination and Treatment Controls, *Appl. Math. Model.* 67 (2019), 38–61.
<https://doi.org/10.1016/j.apm.2018.10.016>.
- [19] Z. Shi, D. Jiang, N. Shi, T. Hayat, A. Alsaedi, The Impact of Nonlinear Perturbation to the Dynamics of HIV Model, *Math. Methods Appl. Sci.* 45 (2022), 2542–2562. <https://doi.org/10.1002/mma.7939>.
- [20] M. Mahrouf, E.M. Lotfi, M. Maziane, K. Hattaf, N. Yousfi, A Stochastic Viral Infection Model with General Functional Response, *Nonlinear Anal. Differ. Equ.* 4 (2016), 435–445. <https://doi.org/10.12988/nade.2016.6642>.
- [21] F. Rao, Y. Tan, X. Lian, Stochastic Analysis of an HIV Model with Various Infection Stages, *Adv. Contin. Discrete Models* 2025 (2025), 38. <https://doi.org/10.1186/s13662-025-03908-w>.
- [22] X. Mao, *Stochastic Differential Equations and Applications*, 2nd ed., Horwood Publishing, Chichester, 2007.
- [23] X. Wang, A Simple Proof of Descartes's Rule of Signs, *Am. Math. Mon.* 111 (2004), 525–526.
<https://doi.org/10.1080/00029890.2004.11920108>.
- [24] I. Chueshov, *Monotone Random Systems Theory and Applications*, Springer Berlin Heidelberg, 2002.
<https://doi.org/10.1007/b83277>.
- [25] M.E. Moreno-Fernandez, P. Presicce, C.A. Chougnet, Homeostasis and Function of Regulatory T Cells in HIV/SIV Infection, *J. Virol.* 86 (2012), 10262–10269. <https://doi.org/10.1128/jvi.00993-12>.

- [26] C.L. Mackall, F.T. Hakim, R.E. Gress, Restoration of T-cell Homeostasis after T-cell Depletion, *Semin. Immunol.* 9 (1997), 339–346. <https://doi.org/10.1006/smim.1997.0091>.
- [27] S. Bonhoeffer, R.M. May, G.M. Shaw, M.A. Nowak, Virus Dynamics and Drug Therapy, *Proc. Natl. Acad. Sci. U.S.A.* 94 (1997), 6971–6976. <https://doi.org/10.1073/pnas.94.13.6971>.
- [28] N. Ikeda, S. Watanabe, A Comparison Theorem for Solutions of Stochastic Differential Equations and Its Applications, *Osaka J. Math.* 14 (1977), 619–633.