Journal of Humanitarian and Applied Sciences - مجلة العلوم الإنسانية والتطبيقية
(رقم الإيداع المحلي - 90-2020)
كلية الآداب والعلوم قصر خيار - جامعة المرقب
JHAS
Volume 9 - Issue 17
المجلد 9 - العدد 17

Immunodeficiency Virus (HIV)

A Study of the Transition from the Deterministic Model to the Stochastic Model

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Received: 04/11/2025 | Accepted:19/11/2025

Abstract

Understanding the intricate dynamics that control the behavior of viruses like HIV requires the use of models that simulate the spread of epidemic diseases. The objective of this study is to examine how deterministic and stochastic elements affect the epidemic system's stability, which offers important insights into how to stop the virus's propagation and enhance treatment approaches. A deterministic model was created to explain the dynamics of the virus's propagation, emphasizing the system's stability at particular moments. EE and DFE were the two primary examples that were examined. Numerical results were obtained that demonstrated how the system behaved in various scenarios. To assess how random changes affect epidemiological dynamics, the deterministic model was then transformed into a stochastic model.

Keywords: Immunodeficiency virus model, AIDS/HIV, stochastic model, covariance matrix, diffusion matrix.

فيروس نقص المناعة (HIV): دراسة التحول من النموذج الحتمي إلى النموذج العشوائي

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ملخص البحث

إن فهم الديناميكيات المعقدة التي تتحكم في سلوك الفيروسات مثل فيروس نقص المناعة البشرية يتطلب استخدام نماذج تحاكي انتشار الأمراض الوبائية. الهدف من هذه الدراسة هو دراسة كيفية تأثير العناصر الحتمية والعشوائية على استقرار النظام الوبائي، والذي يقدم رؤى مهمة حول كيفية وقف انتشار الفيروس وتعزيز أساليب العلاج. تم إنشاء نموذج حتمي لشرح ديناميكيات انتشار الفيروس، مع التأكيد على استقرار النظام في لحظات معينة. وكان EE وDFE المثالين الأساسيين التي تم فحصها. تم الحصول على نتائج عددية توضح كيفية تصرف النظام في سيناريوهات مختلفة. ولتقييم مدى تأثير التغيرات العشوائية على الديناميكيات الوبائية، تم بعد ذلك تحويل النموذج الحتمي إلى نموذج عشوائي.

الكلمات المفتاحية: نموذج فيروس نقص المناعة البشرية، الإيدز/ HIV، النموذج العشوائي، مصفوفة التغاير، مصفوفة الانتشار.

Introduction

Human immunodeficiency virus (HIV) is a highly contagious virus that attacks the human body's immune system. HIV targets human immune system white blood cells (WBCs) and infects them. HIV destroys white blood cells, weakening the immune system, and creates millions of copies in the blood. Helper T cells or CD4+ cells are the names given to this subset of white blood cells. (Raza et al., 2020). In patients with acquired immunodeficiency syndrome (AIDS), the immune system is significantly compromised. HIV impairs the body's defenses against infections that a healthy person would typically be able to withstand. Immunodeficiency results from the progressive destruction or weakening of an infected person's immune cells over time. CD4+ T cell counts are frequently used to assess immune function. In most cases, AIDS occurs 10 to 15 years after HIV infection if treatment is not received. Sadly, there is a significant chance that people with AIDS may experience severe clinical symptoms. Since its discovery in the 1980s, HIV has spread around the world, impacting millions of individuals. Contact with certain body fluids, including blood, semen, vaginal secretions, and breast milk, is the main way that HIV is spread (Wu et al., 2024). Millions of individuals worldwide are losing their lives and means of subsistence due to the AIDS pandemic. The daily infection rate is estimated to be 15,000, and it is predicted to increase. In areas and nations with high rates of poverty, gender inequality, and inadequate public services, the situation is worse. Actually, in many regions of the world, the spread of HIV/AIDS at the start of the twenty-first century is an indication of inadequate development and a failure to establish more wealthy and equal communities (Collins, Rau, & Unies, 2000). HIV/AIDS is one of the most researched infectious illnesses and a global health concern. Numerous biological fields, including epidemiology, virology, immunology, and drug development, as well as non-biomedical fields, including the social sciences and humanities, study HIV/AIDS. The scientific consensus that explains the clinical manifestations of HIV/AIDS in terms of the virus's interactions with immune system cells, the behavior and demographics of immune system cells, and above all the virus's interactions with the host cells' biomolecular machinery is a solid foundation for all biomedical specialties involved in the disease (Fajardo-Ortiz et al., 2017). The World Health Organization reported that since its discovery in 1981, AIDS had killed over 25 million people as of January 2006. Highly active antiretroviral therapy (HAART) is a treatment for HIV infection. Many patients have found it to be beneficial in reducing viral replication, and it is a multidrug. However, because of viral changes brought on by drug pressure, prolonged usage of these medications results in treatment resistance. Consequently, because HIV has a very high rate of mutation, current information on drug resistance is essential for HIV treatment. For usage by virologists and physicians, this data must be manually retrieved by specialists from scholarly publications (Bui et al., 2010). Although there is currently no cure for AIDS, it can be controlled with proper medical care, such as antiretroviral

therapy (ART), which enhances life quality and health while lowering the risk of recurrence (Attaullah et al., 2023). Human immunodeficiency virus (HIV) has a vast economic impact on communities and countries worldwide, extending beyond the individual level. HIV's worldwide spread continues to be a serious public health concern, requiring ongoing efforts to comprehend and manage its dynamics. Using mathematical methods to model real-world issues has shown to be a useful tool for comprehending the intricate relationships within HIV transmission dynamics and for developing efficient control measures for HIV prevention (Khan et al., 2024). HIV is currently among the most fatal infectious diseases in the world (Shigdel, 2012). Clinical care and health education (medicine, dentistry, nursing, etc.) are still significantly impacted by HIV and AIDS (Galane, 2014). The World Health Organization estimates that adolescents and emerging adults between the ages of 15 and 24 account for half of all HIV infections worldwide. The unique problems that people between the ages of 18 and 25 encounter can increase their exposure to and vulnerability to HIV infection (Zefi, 2017). The use of mathematical models is essential when researching the dynamics of HIV/AIDS transmission (Raza et al., 2020). Over the past few decades, models that incorporate differential equations with delays have become used in a variety of technical and scientific domains. Furthermore, the model exhibits more stochastic features and becomes more realistic with the addition of stochastic elements (Khan et al., 2024). In this work, we characterize the dynamics of HIV infection using a novel mathematical model that incorporates a system of differential equations. Then we convert it to a random model in order to study the random fluctuations that may occur in the deterministic model.

In order to evaluate whether an initial HIV infection will progress to AIDS. The following system of equations is satisfied by their number densities (Allen, 2010), (Chou & Friedman, 2016).

$$\begin{cases} \frac{dT}{dt} = f_1(T, T^*, V) = A - \beta TV - \mu T. \\ \frac{dT^*}{dt} = f_2(T, T^*, V) = \beta TV - \mu^* T^*. \\ \frac{dV}{dt} = f_3(T, T^*, V) = \gamma \mu^* T^* - \kappa V. \end{cases} .$$

variable	Description
$T.$	CD4^+ T cells
$T^*.$	Infected CD4^+ T cells
$V.$	The HIV virus outside the T cells
$A.$	The normal T cell generation rate
$\beta.$	The percentage of healthy T lymphocytes infected by an exogenous virus.
$\mu.$	Death rate of T
$\mu^*.$	Death rate of $T^*.$
$\gamma.$	Amount of virus particles that emerge after one infected CD4^+T cell dies.
$\kappa.$	The virus death rate

Before creating the stochastic model for system, it is useful to study the stability of this system at certain points.

$$\beta TV - \mu^* T^* \Longrightarrow \mu^* T^* = \beta TV.$$

$$\gamma \mu^* T^* - \kappa V = 0.$$

$$\gamma \beta TV - \kappa V = 0.$$

$$(\gamma \beta T - \kappa)V = 0.$$

$$V = 0 \; or \; \gamma \beta T - \kappa = 0 \; \Rightarrow T = \frac{\kappa}{\gamma \beta}.$$

$$A - \beta TV - \mu T = 0.$$

$$A - \beta \left(\frac{\kappa}{\gamma \beta}\right) V - \mu \left(\frac{\kappa}{\gamma \beta}\right) = 0.$$

$$\frac{\kappa V}{\gamma} = A - \frac{\mu \kappa}{\gamma \beta}.$$

$$V = \frac{A\gamma}{\kappa} - \frac{\mu}{\beta}.$$

If $V = 0, T^* = 0$ and $A - \beta TV - \mu T = 0 \Rightarrow T = \frac{A}{\mu}$.

hence the DFE will be $(\frac{A}{\mu}, 0, 0)$.

The Jacobian matrix of the system is

$$\begin{pmatrix} -\beta V - \mu & 0 & -\beta T \\ \beta V & -\mu^* & \beta T \\ 0 & \gamma \mu^* & -\kappa \end{pmatrix}.$$

The Jacobian matrix of the system at DFE

$$\begin{pmatrix} -\mu & 0 & -\beta \frac{A}{\mu} \\ 0 & -\mu^* & \beta \frac{A}{\mu} \\ 0 & \gamma \mu^* & -\kappa \end{pmatrix}.$$

Then $|J - \lambda I| = 0$.

$$\left| \begin{pmatrix} -\mu & 0 & -\beta \frac{A}{\mu} \\ 0 & -\mu^* & \beta \frac{A}{\mu} \\ 0 & \gamma \mu^* & -\kappa \end{pmatrix} - \lambda \begin{pmatrix} 1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{pmatrix} \right| = 0.$$

$$\left|\begin{pmatrix} -\mu & 0 & -\frac{\beta A}{\mu} \\ 0 & -\mu^* & \frac{\beta A}{\mu} \\ 0 & \gamma\mu^* & -\kappa \end{pmatrix} - \begin{pmatrix} \lambda & 0 & 0 \\ 0 & \lambda & 0 \\ 0 & 0 & \lambda \end{pmatrix}\right| = 0.$$

$$\begin{vmatrix} -\mu-\lambda & 0 & -\frac{\beta A}{\mu} \\ 0 & -\mu^*-\lambda & \frac{\beta A}{\mu} \\ 0 & \gamma\mu^* & -\kappa-\lambda \end{vmatrix} = 0.$$

The characteristic polynomial is

$$(-\mu-\lambda)\left[(-\mu^*-\lambda)(-\kappa-\lambda) - \left(\beta\frac{A}{\mu}\right)(\gamma\mu^*)\right] = 0.$$

Then

$$(-\mu-\lambda) = 0 \Rightarrow \lambda = -\mu < 0.$$

Or

$$\left[(-\mu^*-\lambda)(-\kappa-\lambda) - \left(\beta\frac{A}{\mu}\right)(\gamma\mu^*)\right] = 0.$$

$$\lambda^2 + (\mu^*+\kappa)\lambda + \kappa\mu^* - \frac{\beta A\gamma\mu^*}{\mu} = 0.$$

Using Routh-Hurwitz criteria where:

$$a_1 = (\mu^*+\kappa) > 0.$$

$$a_2 = \kappa\mu^* - \frac{\beta A\gamma\mu^*}{\mu} > 0.$$

If

$$\kappa\mu^* - \frac{\beta A\gamma\mu^*}{\mu} \Longrightarrow \kappa\mu^* > \frac{\beta A\gamma\mu^*}{\mu} \Longrightarrow \kappa > \frac{\beta A\gamma}{\mu} \Longrightarrow \frac{\kappa}{\gamma} > \frac{\beta A}{\mu}.$$

Hence the DFE is stable if

$$\frac{\beta A}{\mu} < \frac{\kappa}{\gamma}.$$

On the other hand, if

$$\frac{\beta A}{\mu} > \frac{\kappa}{\gamma}.$$

The DFE is unstable and there exists another equilibrium point EE with

$$\bar{T}^* = \frac{\kappa\bar{V}}{\mu^*\gamma}.$$

The equilibrium point is

$$\left(\frac{\kappa}{\gamma\beta}, \frac{\kappa\bar{V}}{\mu^*\gamma}, \frac{A\gamma}{\kappa} - \frac{\mu}{\beta}\right).$$

The Jacobian matrix is

$$\begin{pmatrix} -\frac{\beta A\gamma}{\kappa} & 0 & -\frac{\kappa}{\gamma} \\ \frac{\beta A\gamma}{\kappa} - \mu & -\mu^* & \frac{\kappa}{\gamma} \\ 0 & \gamma\mu^* & -\kappa \end{pmatrix}.$$

The characteristic polynomial is

$$\left(-\frac{\beta A\gamma}{\kappa} - \lambda\right)\left[(-\mu^* - \lambda)(-\kappa - \lambda) - \left(\frac{\kappa}{\gamma}\right)(\gamma\mu^*)\right] - \frac{\kappa}{\gamma}\left[\left(\frac{\beta A\gamma}{\kappa} - \mu\right)(\gamma\mu^*)\right] = 0.$$

$$\left(-\frac{\beta A\gamma}{\kappa} - \lambda\right)[\lambda^2 + (\kappa + \mu^*)\lambda - \beta A\gamma\mu^* + \kappa\mu^*\mu] = 0.$$

Then

$$-\frac{\beta A\gamma}{\kappa} - \lambda = 0 \Longrightarrow \lambda = -\frac{\beta A\gamma}{\kappa} < 0.$$

Or

$$\lambda^2 + (\kappa + \mu^*)\lambda - \beta A\gamma\mu^* + \kappa\mu^*\mu = 0.$$

By Routh-Hurwitz EE is stable if

$$a_1 = \kappa + \mu^* > 0.$$

$$a_2 = -(\beta A\gamma\mu^* - \kappa\mu^*\mu) > 0\,.$$

$$\beta A\gamma > \kappa\mu.$$

That is if

$$\frac{\beta A}{\mu} > \frac{\kappa}{\gamma}.$$

Hence

$$R_0 = \frac{\beta A\gamma}{\kappa\mu}.$$

It is clear that if $R_0 < 1$, DFE is stable on the other hand if $R_0 > 1$ DFE is unstable and EE is stable.

Numerical Representation:

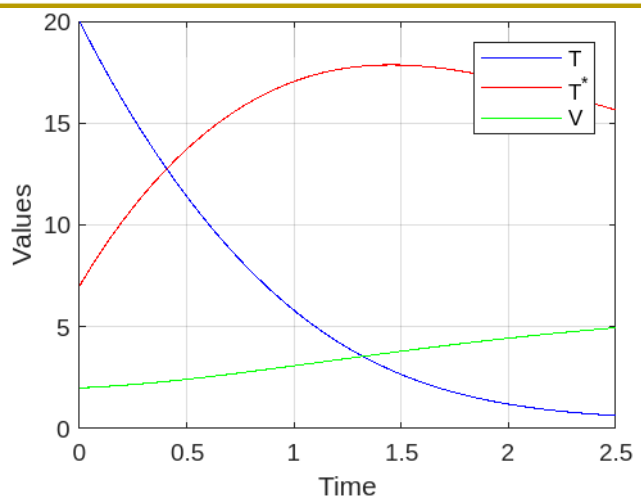

Figure 1. HIV; $A = 1, \beta = 1, \mu = 0.4, \mu^* = 0.5, \gamma = 1, \kappa = 1.$

The graph shows the deterioration of healthy CD4+ T cells, which start at 20 and decrease towards zero, indicating a weak immune system, while infected CD4+ T cells start with a H between 2 and 10, which increases and then fluctuates, which indicates the development of the infection, and V (viral load). It starts at less than five and increases slowly, which indicates the spread of the virus, but at a lower rate. Thus, the model shows instability in the system, where immune cells deteriorate and infections increase.

To provide deeper insights into HIV dynamics, we will turn to the stochastic model.

The stochastic model for system:

Rewrite the model so that it is free of recovered people and deaths

$$\begin{cases} \frac{dT}{dt} = -\beta TV. \\ \frac{dT^*}{dt} = \beta TV. \\ \frac{dV}{dt} = -\kappa V. \end{cases} .$$

1- Probabilities associated with changes in the HIV model

Table. Probabilities associated with changes in the HIVmodel

Changes,Δx_i	**Probability,p_i**
$(-1,1,0)^{\mathrm{tr}}.$	$\beta TV\ \Delta t.$
$(0,0,-1)^{\mathrm{tr}}.$	$\kappa V\ \Delta t.$

2- The expectation $E(\Delta x) = \sum_{i=1}^{2} p_i \Delta x_i$ is 3×1 matrix, the expectation can be expressed as follows.

$$E(\Delta x) = \sum_{i=1}^{2} p_i \Delta x_i = p_1 \Delta x_1 + p_2 \Delta x_2 \,,$$

$$E(\Delta x) = \sum_{i=1}^{2} p_i \Delta x_i = \beta TV \begin{pmatrix} -1 \\ 1 \\ 0 \end{pmatrix} + \kappa V \begin{pmatrix} 0 \\ 0 \\ -1 \end{pmatrix}.$$

$$E(\Delta x) = \begin{pmatrix} -\beta TV \\ \beta TV \\ 0 \end{pmatrix} + \begin{pmatrix} 0 \\ 0 \\ -\kappa V \end{pmatrix}.$$

$$E(\Delta x) = \begin{pmatrix} -\beta TV \\ \beta TV \\ -\kappa V \end{pmatrix} \Delta t.$$

3- The covariance matrix can be expressed as follows.

$$E(\Delta x(\Delta x)^T) = \sum_{i=1}^{2} p_i \Delta x_i \, (\Delta x_i)^T.$$

$$= p_1 \Delta x_1 (\Delta x_1)^T + p_2 \Delta x_2 (\Delta x_2)^T.$$

$$E(\Delta x(\Delta x)^T) = \begin{pmatrix} -\beta TV \\ \beta TV \\ 0 \end{pmatrix} (-1 \;\; 1 \;\; 0) + \begin{pmatrix} 0 \\ 0 \\ -\kappa V \end{pmatrix} (0 \;\; 0 \;\; -1).$$

$$E(\Delta x(\Delta x)^T) = \begin{pmatrix} \beta TV & -\beta TV & 0 \\ -\beta TV & \beta TV & 0 \\ 0 & 0 & 0 \end{pmatrix} + \begin{pmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & \kappa V \end{pmatrix}.$$

$$E(\Delta x(\Delta x)^T) = \begin{pmatrix} \beta TV & -\beta TV & 0 \\ -\beta TV & \beta TV & 0 \\ 0 & 0 & \kappa V \end{pmatrix} \Delta t.$$

$$E(\Delta x(\Delta x)^T) = \begin{pmatrix} \beta TV & -\beta TV & 0 \\ -\beta TV & \beta TV & 0 \\ 0 & 0 & \kappa V \end{pmatrix} \Delta t = V \Delta t \,.$$

4 – Formulate the stochastic system as

$$dX(t) = f(X(t), t)dt + h(X(t), t)dW(t).$$

Where

$$dX(t) = \begin{bmatrix} dT_t \\ dT_t^* \\ dV_t \end{bmatrix}, f(X(t), t) = \left[\frac{E(\Delta X)}{\Delta t}\right], h(X(t), t) = \sqrt{V} \text{ and } dW(t) = \begin{bmatrix} dW_1(t) \\ dW_2(t) \\ dW_3(t) \end{bmatrix}.$$

$$\begin{pmatrix} dT_t \\ dT_t^* \\ dV_t \end{pmatrix} = \begin{pmatrix} -\beta TV \\ \beta TV \\ -\kappa V \end{pmatrix} dt + \begin{pmatrix} \sqrt{\beta TV} & -\sqrt{\beta TV} & 0 \\ -\sqrt{\beta TV} & \sqrt{\beta TV} & 0 \\ 0 & 0 & \sqrt{\kappa V} \end{pmatrix} \cdot \begin{pmatrix} dW_1(t) \\ dW_2(t) \\ dW_3(t) \end{pmatrix}.$$

$$\begin{cases} dT_t = (-\beta T_t V_t)dt + \sqrt{\beta T_t V_t}\, dW_1(t) - \sqrt{\beta T_t V_t} dW_2(t)\,. \\ dT^*{}_t = (\beta T_t V_t)dt - \sqrt{\beta T_t V_t}\, dW_1(t) + \sqrt{\beta T_t V_t} dW_2(t). \quad . \\ dV_t = (-\kappa V_t)dt + \sqrt{\kappa V_t} dW_3(t). \end{cases}$$

5- Numerical Representation:

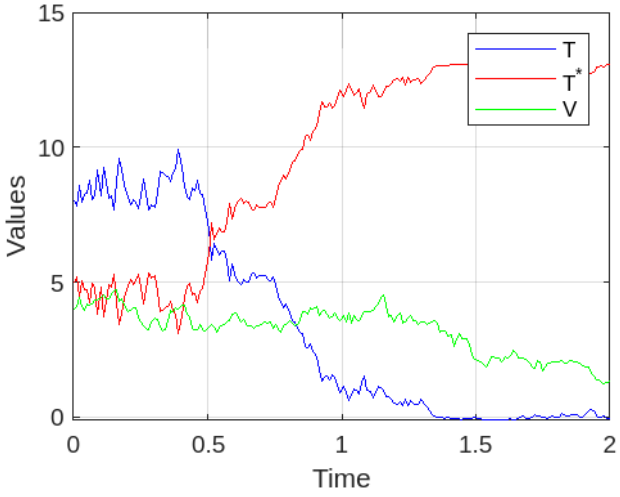

Figure2 .HIV; $\beta = 0.5, \kappa = 1.$

The equivalent system for the former system.

6- The diffusion matrix G of dimension 3×2 is:

$$G = \begin{pmatrix} -\sqrt{\beta TV} & 0 \\ \sqrt{\beta TV} & 0 \\ 0 & -\sqrt{\kappa V} \end{pmatrix}.$$

$$dX(t) = f(X(t), t)dt + g(X(t), t)dW(t).$$

Where

$$dX(t) = \begin{bmatrix} dT_t \\ dT_t^* \\ dV_t \end{bmatrix}, f(X(t), t) = \left[\frac{E(\Delta X)}{\Delta t}\right], g(X(t), t) = G \text{ and } dW(t) = \begin{bmatrix} dW_1(t) \\ dW_2(t) \end{bmatrix}.$$

Thus, the system takes the following form:

$$\begin{pmatrix} dT_t \\ dT_t^* \\ dV_t \end{pmatrix} = \begin{pmatrix} -\beta TV \\ \beta TV \\ -\kappa V \end{pmatrix} dt + \begin{pmatrix} -\sqrt{\beta TV} & 0 \\ \sqrt{\beta TV} & 0 \\ 0 & -\sqrt{\kappa V} \end{pmatrix} \cdot \begin{pmatrix} dW_1(t) \\ dW_2(t) \end{pmatrix}.$$

$$\begin{cases} dT_t = (-\beta TV\,)dt - \sqrt{\beta TV}\,dW_1(t). \\ dT_t^* = (\beta TV)dt + \sqrt{\beta TV}\,dW_1(t). \\ dV_t = (-\kappa V)dt - \sqrt{\kappa V}\,dW_2(t). \end{cases} .$$

7- Numerical Representation:

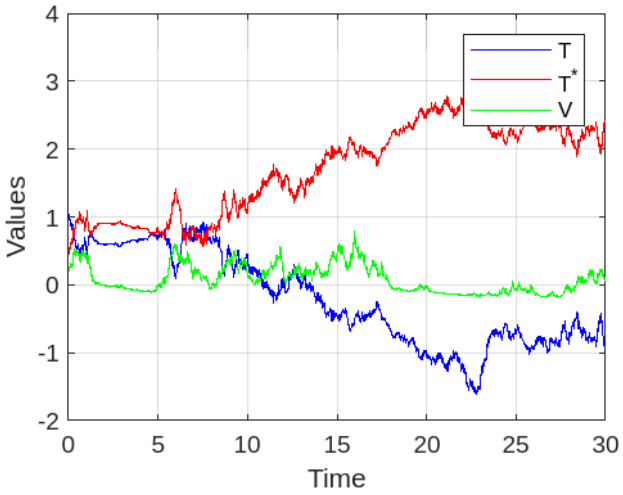

Figure 3. HIV; $\beta = 0.5, \kappa = 0.2.$

The drawing reflects the deterioration of healthy CD4+ T cells with fluctuations towards zero, while infected CD4+ T cells increase despite the fluctuations, which indicates the development of the infection, and (viral load) V fluctuates downwards, which indicates the spread of the infection with relative stability. Thus, the random model reflects the effect of stressors unexpected on the system, which makes the results more complex and realistic.

Conclusion

A mathematical model of the HIV virus was created in order to identify fixed points, such as the disease-free point, and to gain a thorough understanding of the virus's population dynamics. Using stability analysis and eigenvalue computation, it was discovered that some points were unstable and others were stable, reflecting the system's varying responses under particular circumstances. The deterministic model's numerical solution also demonstrated how several factors could impact the virus's stability. It was discovered that random factors significantly influence epidemiological dynamics when the model was transformed to a random form. These findings inform preventative and treatment plans and advance scientific knowledge of the virus's difficulties, which creates new avenues for further investigation.

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Appendix

1. Code of HIV (Deterministic model):

```
% Parameters
A = 1;         % Parameter A
beta = 1;      % Parameter beta
mu = 0.4;        % Parameter mu
mu_star = 0.5;  % Parameter mu*
gamma = 1;     % Parameter gamma
kappa = 1;     % Parameter kappa
```

```
% Initial conditions
T0 = 20;       % Initial condition for T
T_star0 = 7;  % Initial condition for T*
V0 = 2;       % Initial condition for V
initial_conditions = [T0, T_star0, V0];  % Combine initial conditions

% Time span
tspan = [0, 2.5];  % Time span for the simulation

% Solve the system using ode45
[t, X] = ode45(@ode_system, tspan, initial_conditions);

% Extract results
T_values = X(:, 1);
T_star_values = X(:, 2);
V_values = X(:, 3);

% Plot results
figure;
plot(t, T_values, '-b', 'DisplayName', 'T');
hold on;
plot(t, T_star_values, '-r', 'DisplayName', 'T^*');
plot(t, V_values, '-g', 'DisplayName', 'V');
xlabel('Time');
ylabel('Values');
legend;
grid on;
hold off;

% Define the system of ODEs as a sub-function
function dXdt = ode_system(~, X)
    % Parameters (nested inside function to ensure they are in scope)
    A = 1.0;        % Parameter A
    beta = 0.5;     % Parameter beta
    mu = 0.1;       % Parameter mu
    mu_star = 0.3;  % Parameter mu*
    gamma = 0.4;    % Parameter gamma
    kappa = 0.2;    % Parameter kappa

    % State variables
    T = X(1);
    T_star = X(2);
    V = X(3);

    % Differential equations
    dTdt = A - beta * T * V - mu * T;
    dT_stardt = beta * T * V - mu_star * T_star;
    dVdt = gamma * mu_star * T_star - kappa * V;

    % Return derivatives
    dXdt = [dTdt; dT_stardt; dVdt];
end
```

2. Code of HIV (Stochastic model):

```
% Parameters
beta = 0.5;     % Parameter beta
kappa = 1;    % Parameter kappa
```

```
% Initial conditions
T0 = 8;       % Initial condition for T
T_star0 = 5;  % Initial condition for T*
V0 = 4;       % Initial condition for V
initial_conditions = [T0, T_star0, V0];  % Combine initial conditions

% Time span
Tmax = 2;     % Maximum time
dt = 0.01;      % Time step
N = Tmax / dt;  % Number of steps
time = linspace(0, Tmax, N); % Time vector

% Preallocate arrays for results
T = zeros(1, N);
T_star = zeros(1, N);
V = zeros(1, N);

% Initial values
T(1) = T0;
T_star(1) = T_star0;
V(1) = V0;

% Simulate SDEs using Euler-Maruyama method
for i = 2:N
    dW1 = sqrt(dt) * randn;
    dW2 = sqrt(dt) * randn;
    dW3 = sqrt(dt) * randn;

    T(i) = T(i-1) + (-beta * T(i-1) * V(i-1)) * dt + sqrt(beta * T(i-1) *
V(i-1)) * (dW1 - dW2);
    T_star(i) = T_star(i-1) + (beta * T(i-1) * V(i-1)) * dt - sqrt(beta *
T(i-1) * V(i-1)) * (dW1 - dW2);
    V(i) = V(i-1) + (-kappa * V(i-1)) * dt + sqrt(kappa * V(i-1)) * dW3;
end

% Plot results
figure;
plot(time, T, '-b', 'DisplayName', 'T');
hold on;
plot(time, T_star, '-r', 'DisplayName', 'T^*');
plot(time, V, '-g', 'DisplayName', 'V');
xlabel('Time');
ylabel('Values');
legend;
grid on;
hold off;
```

3. Code of HIV (equevelant Stochastic model):

```
% Parameters
beta = 0.5;     % Parameter beta
kappa = 0.2;    % Parameter kappa

% Initial conditions
T0 = 1.0;       % Initial condition for T
T_star0 = 0.5;  % Initial condition for T*
V0 = 0.2;       % Initial condition for V
initial_conditions = [T0, T_star0, V0];  % Combine initial conditions
```

```
% Time span
Tmax = 30;     % Maximum time
dt = 0.01;      % Time step
N = Tmax / dt;  % Number of steps
time = linspace(0, Tmax, N); % Time vector

% Preallocate arrays for results
T = zeros(1, N);
T_star = zeros(1, N);
V = zeros(1, N);

% Initial values
T(1) = T0;
T_star(1) = T_star0;
V(1) = V0;

% Simulate SDEs using Euler-Maruyama method
for i = 2:N
    dW1 = sqrt(dt) * randn;
    dW2 = sqrt(dt) * randn;

    T(i) = T(i-1) + (-beta * T(i-1) * V(i-1)) * dt - sqrt(beta * T(i-1) *
V(i-1)) * dW1;
    T_star(i) = T_star(i-1) + (beta * T(i-1) * V(i-1)) * dt + sqrt(beta *
T(i-1) * V(i-1)) * dW1;
    V(i) = V(i-1) + (-kappa * V(i-1)) * dt - sqrt(kappa * V(i-1)) * dW2;
end

% Plot results
figure;
plot(time, T, '-b', 'DisplayName', 'T');
hold on;
plot(time, T_star, '-r', 'DisplayName', 'T^*');
plot(time, V, '-g', 'DisplayName', 'V');
xlabel('Time');
ylabel('Values');
legend;
grid on;
hold off;
```