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ANALYSIS AND OPTIMAL CONTROL OF HIV GROWTH MODEL IN THE BODY WITH ANTIRETROVIRAL THERAPY

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ABSTRACT

Human Immunodeficiency Virus (HIV) is a virus that affects the human immune system. HIV infection causes a decrease in the body's immunity because the virus attacks immune-building cells, especially T-CD4 cells. Currently, there is no treatment that can cure or eliminate HIV, but antiretroviral therapy can be done. This study discusses the growth model of HIV in the body that is given control in an effort to maximize healthy T-CD4 cells. In this model, the infection-free and infected equilibrium points are also discussed, and their stability is analyzed. Then the optimal control is solved using the Pontryagin Maximum Principle method and solved numerically using the fourth-order Runge Kutta method. Based on the analysis and simulation results, the system is asymptotically stable around the infection-free equilibrium point and unstable around the infected equilibrium point. Simulation results show that with the control of antiretroviral therapy, the T-CD4 cell population grows significantly, which can improve the quality of life of patients. And the growth of HIV in the body can be inhibited until it cannot reproduce itself.

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1. INTRODUCTION

Human Immunodeficiency Virus (HIV) is a virus that damages the human immune system. HIV infection causes a decrease in the body's immunity because the virus attacks immune-building cells, especially T-CD4 cells, thus damaging or disrupting their function. In more advanced stages, HIV infection can lead to AIDS, which is characterized by a sharp decline in immunity, making the body more susceptible to various other comorbidities and opportunistic infections [1]. HIV is categorized as a Lentivirus genus in the Retroviridae family in the Orthoretrovirinae subfamily [2].To this day, no treatment that can treat or eliminate HIV, but antiretroviral therapy is possible. The introduction of highly effective antiretroviral therapy (ART) has transformed the treatment of people living with human immunodeficiency virus (PLWH). Combination ART has multiple benefits for individuals and society, mediated by achieving viral suppression, improving health-related quality of life, and preventing HIV transmission [3],[4]. Various classes of antiretroviral drugs with different mechanisms have been discovered. ART generally uses two drugs from one class along with a third drug from another class. The anti-HIV drug classes include Nucleoside reverse transcriptase inhibitors (NRTIs) and non-nucleoside reverse transcriptase inhibitors (NNRTI), which inhibit viral RNA transcription by suppressing reverse transcriptase. Protease inhibitors (PIs) inhibit the viral protease enzyme, stopping viral maturation, while integrase inhibitors block viral DNA integration into T-CD4 cells [5].

The Ministry of Health reports that the number of HIV/AIDS cases in Indonesia is still relatively high. The Ministry of Health predicts that by September 2023, there will be more than 500,000 HIV cases recorded. Based on the number of estimated cases until September 2023, there were 515,455 people living with HIV (PLWH) in Indonesia. Of these, 454,723 people, or about 88 percent of them, have been infected with HIV or know their HIV status [6]. Based on this data, the number of housewives infected with HIV reached 35%. This figure is higher than HIV cases in other groups, such as husbands of sex workers and MSM (man sex with man) groups. Currently, HIV cases in children aged 1-14 years reach 14,150 cases. This number increases annually by about 700-1000 children with HIV [7].

Mathematical modeling is often used to explain biological phenomena such as the spread of disease. In this case, mathematical modeling is important to analyze HIV/AIDS infection. There have been many researches on mathematical modeling of the spread of HIV/AIDS. Zamzami et al. [8] have conducted mathematical modeling and stability analysis on the spread of HIV/AIDS with treatment. In this research, there are four compartments, such as the population of vulnerable individuals, the population of HIV-infected individuals, the population of AIDS-infected individuals, and the population of individuals undergoing treatment. Then Lamusu et al. [9] built a mathematical model of the spread of HIV/AIDS with three compartments i.e., Susceptible (S), Infected (I), and AIDS (A) applied to two cities. Rahayu et al. [10] built a mathematical model on the growth of the number of HIV viruses. In this research, there are three compartments such as healthy T-CD4 cells, infected T-CD4 cells and HIV.

Optimal control is a science in mathematics that has been developed to find the best method of controlling dynamic systems [11]. The Pontryagin Maximum Principle is one of the optimal control methods for dynamic models, as shown by the following research [12],[13],[14],[15]. So in this research, the mathematical model of HIV growth was obtained from the mathematical model of Rahayu et al. [10], which was then developed with control in the form of a combination of antiretrovirals, i.e., Nucleoside Reverse Transcriptase Inhibitor (NRTI) and Protease Inhibitor (PI). And modify some parameters that are also taken from research by Dubey et al. [16].

2. RESEARCH METHODS

The research steps on the HIV growth model in the body with antiretroviral therapy (ART) are as follows:

1. Explain the model used in the HIV growth in the body.
2. From the model that has been obtained, the equilibrium point of the HIV growth model in the body is determined. The equilibrium point can be obtained by taking the first derivative equal to zero. Definition [17]:

Given a first-order differential equation $\dot{x}(t) = f\big(x(t)\big)$ with $x \in \mathbb{R}^n$, the solution with an initial state $x(0) = x_0$ is denoted by $x(t, x_0)$. The vector x that satisfies $f(\bar{x}) = 0$ is called an equilibrium point.

3. Analyzing the stability of the model around the equilibrium point. To analyze the equilibrium point at a certain condition, a nonlinear system of differential equations can be linearized using the Jacobian matrix.
   If given a function $f = (f_1, f_2, \dots, f_n)$in a system $\dot{x} = f(x)$. Then, the Jacobian matrix:
$$Jf(x^*) = \begin{bmatrix} \frac{\partial f_1}{\partial x_1}(x^*) & \frac{\partial f_1}{\partial x_2}(x^*) & \cdots & \frac{\partial f_1}{\partial x_n}(x^*) \\ \frac{\partial f_2}{\partial x_1}(x^*) & \frac{\partial f_2}{\partial x_2}(x^*) & \cdots & \frac{\partial f_2}{\partial x_n}(x^*) \\ \vdots & \vdots & \vdots & \vdots \\ \frac{\partial f_n}{\partial x_1}(x^*) & \frac{\partial f_n}{\partial x_2}(x^*) & \dots & \frac{\partial f_n}{\partial x_n}(x^*) \end{bmatrix}$$
   To make it easier to determine the eigenvalue of the characteristic equation, it can also be done using the Routh-Hurwitz criterion.
4. The model of HIV growth in the body is developed with control in the form of antiretroviral therapy, NRTI, and PI therapy.
5. Numerical simulation was carried out using the 4th-order Runge-Kutta method. After the simulation, the results were analyzed to determine the graph behavior of the HIV growth model in the body when before and after being given control.

3. RESULTS AND DISCUSSION

3.1 Mathematical Model

The model that will be discussed in this research has three variables: healthy T-CD4 cell population (H), infected T-CD4 cell population (I), and HIV population (V). Some assumptions used in the model of HIV growth in the body with antiretroviral therapy are as follows:

1. The spread of HIV occurs internally, i.e., in the human body.
2. There are no other viruses that attack other than HIV.
3. The growth of HIV in the body only affects the population of T-CD4 cells and HIV.
4. Control input is in the form of antiretroviral combination therapy, namely NRTI and PI.

So from the assumptions mentioned above, a compartment diagram of the HIV growth model in the body is obtained as follows [10]:

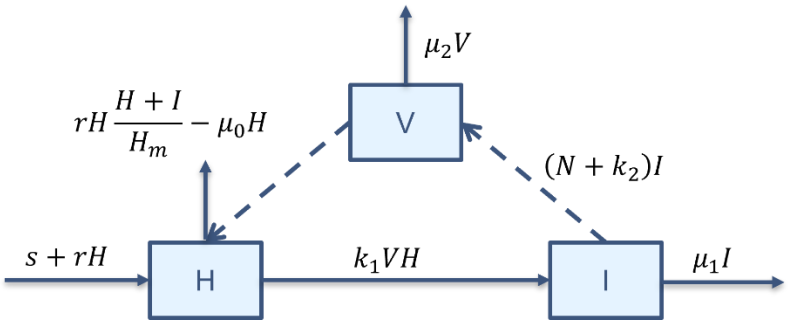


Figure 1. Model Diagram of the HIV Growth in the Body

Figure 1 illustrates that the inbound arrow signifies an increase in the population of variables H, I, and V. Conversely, the outgoing arrow indicates a decrease in the population size of these variables. The dashed line from I to V indicates that the population of V will increase without decreasing the population of I.

Similarly, the dashed line from V to H indicates that V attacks H without reducing its population. Based on **Figure 1**, we obtain the following mathematical model in **Equation (1)**:

$$\begin{aligned}\frac{dH}{dt} &= s + rH\left(1 - \frac{H+I}{H_m}\right) - \mu_0 H - k_1 VH \\ \frac{dI}{dt} &= k_1 VH - \mu_1 I \\ \frac{dV}{dt} &= (N + k_2)I - \mu_2 V\end{aligned} \tag{1}$$

The parameters used are as follows:

s : The production rate of healthy T-CD4 cells
μ_0 : The natural death rate of T-CD4 cells
r : The growth rate of T-CD4 cells
H_m : The maximal capacity of T-CD4 cells
k_1 : The rate of infected T-CD4 cells
μ_1 : The death rate of infected T-CD4 cells
N : Source of virus
k_2 : The rate of HIV inside T-CD4 cells
μ_2 : HIV mortality rate

3.2 Equilibrium Point

The equilibrium point is obtained if the following conditions are satisfied,

$$\frac{dH}{dt} = \frac{dI}{dt} = \frac{dV}{dt} = 0$$

The equilibrium point of the HIV growth model in the body has two equilibrium points, which are the infection-free equilibrium point and the infected equilibrium point.

3.2.1. Infection-Free Equilibrium Point

The infection-free equilibrium point is the condition when the body has not been infected, i.e., the condition when $I = 0$ and $V = 0$. Then, the infection-free equilibrium point can be written with $E_0 = (H_0, 0,0)$, where H_0 is given by:

$$E_0 = \left(\frac{H_m}{2r}\left[(r-\mu_0) + \sqrt{\frac{4sr}{H_m} + (r-\mu_0)^2}\right], 0,0\right)$$

3.2.2. Infection Equilibrium Point

The infected equilibrium point is the equilibrium point where T-CD4 cells become infected. So that the infected equilibrium point can be written as $E_1 = (H_1, I_1, V_1)$, with

$$H_1 = \frac{\mu_2\mu_1}{k_1k_2}$$

$$I_1 = \frac{r\mu_1\mu_2H_mk_1k_2 - \mu_0\mu_1\mu_2H_mk_1k_2 + sH_mk_1^2k_2^2 - r\mu_1^2\mu_2^2}{H_mk_1^2k_2^2\mu_1}$$

$$V_1 = \frac{r\mu_1\mu_2H_mk_1k_2 - \mu_0\mu_1\mu_2H_mk_1k_2 + sH_mk_1^2k_2^2 - r\mu_1^2\mu_2^2}{H_mk_1^2k_2\mu_1\mu_2}$$

3.3 Stability Analysis

After obtaining the equilibrium point of the HIV growth model in the body, the next step is to analyze the stability of the system. Based on **Equations (1)**, it can be seen that the mathematical model of HIV growth in the body is a nonlinear system of differential equations, so to get stability, linearization will be carried out using the Jacobi matrix. The Jacobian matrix is a linear approximation of the nonlinear system of the model. **Equation (1)** can be expressed as a function of the variables $H, I,$ and V, so that the equation can be expressed generally as follows:

$$\begin{aligned}\frac{dH}{dt} &= s + rH\left(1 - \frac{H+I}{H_m}\right) - \mu_0 H - k_1 VH = f_1(H,I,V)\\ \frac{dI}{dt} &= k_1 VH - \mu_1 I = f_2(H,I,V)\\ \frac{dV}{dt} &= (N+k_2)I - \mu_2 V = f_3(H,I,V)\end{aligned} \tag{2}$$

The Jacobian matrix of **Equation (2)** is as follows:

$$J = \begin{bmatrix} \frac{\partial f_1}{\partial H} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial V} \\ \frac{\partial f_2}{\partial H} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial V} \\ \frac{\partial f_3}{\partial H} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial V} \end{bmatrix} = \begin{bmatrix} -j_{11} & -\frac{rH}{Hm} & -k_1 H \\ k_1 V & -\mu_1 & k_1 H \\ 0 & N+k_2 & -\mu_2 \end{bmatrix}$$

where $j_{11} = -r\left(1 - \frac{2H}{H_m}\right) + \mu_0 + k_1 V$

3.3.1. Infection-Free Equilibrium Point Stability

To determine the stability of the infection-free equilibrium point E_0, then substitute the value of the infection-free equilibrium point $E_0 = \left(\frac{H_m}{2r}\left[(r-\mu_0) + \sqrt{\frac{4sr}{H_m} + (r-\mu_0)^2}\right], 0, 0\right)$, in the Jacobi Matrix so obtained:

$$J(E_0) = \begin{bmatrix} -j_{11} & -\frac{rH_0}{Hm} & -k_1 H_0 \\ 0 & -\mu_1 & k_1 H_0 \\ 0 & N+k_2 & -\mu_2 \end{bmatrix}$$

where $j_{11} = -r\left(1 - \frac{2H_0}{H_m}\right) + \mu_0$

The characteristic equation of the Jacobian matrix can be found by using $|\lambda I - J(E_0)| = 0$, so obtained:

$$(\lambda - j_{11})(\lambda^2 + b\lambda + c) = 0$$

where $b = \mu_1 + \mu_2$ and $c = \mu_1\mu_2 - Nk_1H_0 - k_2k_1H_0$

So that the eigenvalues of the matrix $J(E_0)$ are obtained $\lambda_1 = -j_{11}$ and $\lambda_{2,3}$ is found using the ABC formula

$$\lambda_{2,3} = \frac{-(\mu_1+\mu_2) \pm \sqrt{(\mu_1+\mu_2)^2 - 4(\mu_1\mu_2 - Nk_1H_0 - k_2k_1H_0)}}{2}$$

Since the parameter values are positive, it is obtained that $\lambda_1 < 0$ and $\Re(\lambda_2), \Re(\lambda_3) < 0$,, it can be concluded that the infection-free equilibrium point is asymptotically stable.

3.3.2. Infection Equilibrium Point Stability

To determine the stability of the infection-free equilibrium point (E_0), then substitute the value of the infection-free equilibrium point $E_1 = (H_1, I_1, V_1)$ in the Jacobi Matrix so that it is obtained:

$$J(E_1) = \begin{bmatrix} -j_{11} & -\frac{rH_0}{Hm} & -k_1H_1 \\ k_1V_1 & -\mu_1 & k_1H_1 \\ 0 & N+k_2 & -\mu_2 \end{bmatrix}$$

where $j_{11} = -r\left(1 - \frac{2H}{H_m}\right) + \mu_0 + k_1V$

The characteristic equation of the Jacobian matrix can be found by using $|\lambda I - J(E_0)| = 0$, so the following equation is obtained:

$$(\lambda + j_{11})[(\lambda + \mu_1)(\lambda + \mu_2) - (N + k_2)(k_1H_1)] = 0$$
$$A_0\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0 \quad (3)$$

where,

$A_0 = 1$

$A_1 = j_{11} + \mu_1 + \mu_2$

$A_2 = j_{11}\mu_1 + j_{11}\mu_2 + \mu_1\mu_2 - H_1k_1N - H_1k_1k_2$

$A_3 = j_{11}\mu_1\mu_2 - j_{11}H_1k_1k_2 - j_{11}H_1k_1N$

To simplify the calculation in determining the eigenvalue of the characteristic equation, the Routh Hurwitz criterion is used to determine the stability properties of the system around the equilibrium point of infection, as follows:

$$A_0\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0$$

λ^3	A_0	A_2
λ^2	A_1	A_3
λ^1	b_1	0
λ	c_1	0

where $b_1 = \frac{A_1A_2 - A_3}{A_1}$ and $c_1 = \frac{b_1A_3 - A_10}{b_1} = A_3$. **Equation (3)** will have negative characteristic roots if and only if A_1, b_1, c_1 are positive, which means $A_1, A_2, A_3 > 0$ and $A_1A_2 > A_3$. So, the model of HIV growth in the body around the equilibrium point of infection will be asymptotically stable if $A_1, A_2, A_3 > 0$ and $A_1A_2 > A_3$. However, if the condition is taken that $A_1 < 0$, , then the model of HIV growth in the body around the equilibrium point of infection will be unstable.

3.4 Optimal Control Solution

In this research, optimal control aims to maximize the healthyT-CD4 cells at the cost of implementing minimum θ_0 and θ_1 controls. The objective function of the HIV growth model in the body with antiretroviral therapy is as follows:

$$J(\theta_0, \theta_1) = \max \int_{t_0}^{t_f} \left[a_1H - \frac{1}{2}\left(b_1\theta_0^2(t) + b_2\theta_1^2(t)\right)\right] dt$$

where

a_1 : population weight of healthy T-CD4 cells

b_1 : cost weight on NRTI therapy control (θ_0)

b_2 : cost weight on PI therapy control (θ_1)

With the following constraints:

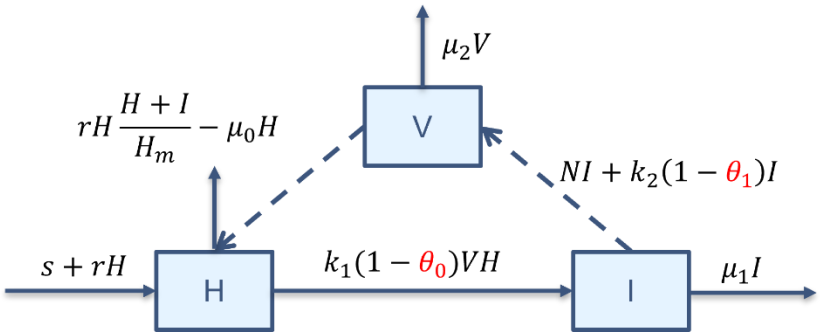


Figure 2. Model Diagram of HIV Growth in the Body with Antiretroviral Therapy Control

Based on the compartment diagram in **Figure 2**, a mathematical model of the HIV growth in the body with controls can be formulated as follows:

$$\frac{dH}{dt} = s + rH\left(1 - \frac{H+I}{H_m}\right) - \mu_0 H - k_1(1-\theta_0)VH$$

$$\frac{dI}{dt} = k_1(1-\theta_0)VH - \mu_1 I$$

$$\frac{dV}{dt} = NI + k_2(1-\theta_1)I - \mu_2 V$$

and

$$0 \le \theta_0(t) \le 1, \qquad 0 \le \theta_1(t) \le 1,$$

The steps used in solving the optimal control of the HIV growth model in the body using the Pontryagin Maximum Principle method are as follows **[18]**:

1. Obtain the Pontryagin function form (Hamiltonian)

$$\mathcal{H} = a_1 H - \frac{1}{2}(b_1\theta_0^2 + b_2\theta_1^2) + \lambda_1\left(s + rH\left(1 - \frac{H+I}{H_m}\right) - \mu_0 H - k_1(1-\theta_0)VH\right) + \lambda_2(k_1(1-\theta_0)VH - \mu_1 I) + \lambda_3(NI + k_2(1-\theta_1)I - \mu_2 V) \quad (4)$$

2. Determine the optimal condition of the Hamiltonian function ($\mathcal{H}$) on the control θ
   The optimal control equation can be obtained by deriving **Equation (4)** to each of the control variables θ_0 and θ_1.
   - Equation ($\mathcal{H}$) is derived to θ_0, obtained as follows,
   $$\theta_0^* = \frac{(\lambda_1 - \lambda_2)k_1 VH}{b_1}$$
   - Equation ($\mathcal{H}$) is derived to θ_1, obtained as follows,
   $$\theta_1^* = \frac{\lambda_3 k_2 I}{b_2}$$
3. Get the optimal $\mathcal{H}^*$ value
   Substituting the optimal θ_0^* dan θ_1^* results into the Hamiltonian form, thus obtained:
   $$\mathcal{H} = a_1 H - \frac{1}{2}(b_1\theta_0^2 + b_2\theta_1^2) + \lambda_1\left(s + rH\left(1 - \frac{H+I}{H_m}\right) - \mu_0 H - k_1(1-\theta_0^*)VH\right) + \lambda_2(k_1(1-\theta_0^*)VH - \mu_1 I) + \lambda_3(NI + k_2(1-\theta_1^*)I - \mu_2 V)$$
4. Get state and costate equations
   - State equation: $\dot{\boldsymbol{x}}^*(t) = +\left(\frac{\partial \mathcal{H}}{\partial \boldsymbol{\lambda}}\right)_*$

   Obtained by:

$$\dot{H}^*(t) = +\left(\frac{\partial \mathcal{H}}{\partial \lambda_1}\right)_* = s + rH\left(1 - \frac{H+I}{H_m}\right) - \mu_0 H - k_1(1-\theta_0^*)VH$$

$$\dot{I}^*(t) = +\left(\frac{\partial \mathcal{H}}{\partial \lambda_2}\right)_* = k_1(1-\theta_0^*)VH - \mu_1 I \quad (5)$$

$$V^*(t) = +\left(\frac{\partial \mathcal{H}}{\partial \lambda_3}\right)_* = NI + k_2(1-\theta_1^*)I - \mu_2 V$$

- Costate equation: $\dot{\boldsymbol{\lambda}}^*(t) = -\left(\frac{\partial \mathcal{H}}{\partial \boldsymbol{x}}\right)_*$

Obatined by:

$$\dot{\lambda}_1^*(t) = -\left(\frac{\partial \mathcal{H}}{\partial H}\right)_* = -\left[a_1 + \lambda_1\left(r\left(1 - \frac{2H+I}{H_m}\right) - \mu_0 - k_1(1-\theta_0^*)V\right) + \lambda_2(k_1(1-\theta_0^*)V)\right]$$

$$\dot{\lambda}_2^*(t) = -\left(\frac{\partial \mathcal{H}}{\partial I}\right)_* = -[\lambda_2(-\mu_1) + \lambda_3(N + k_2(1-\theta_1^*)] \quad (6)$$

$$\dot{\lambda}_3^*(t) = -\left(\frac{\partial \mathcal{H}}{\partial V}\right)_* = -[\lambda_1(-k_1(1-\theta_0^*)H) + \lambda_2(k_1(1-\theta_0^*)H) - \lambda_3\mu_2]$$

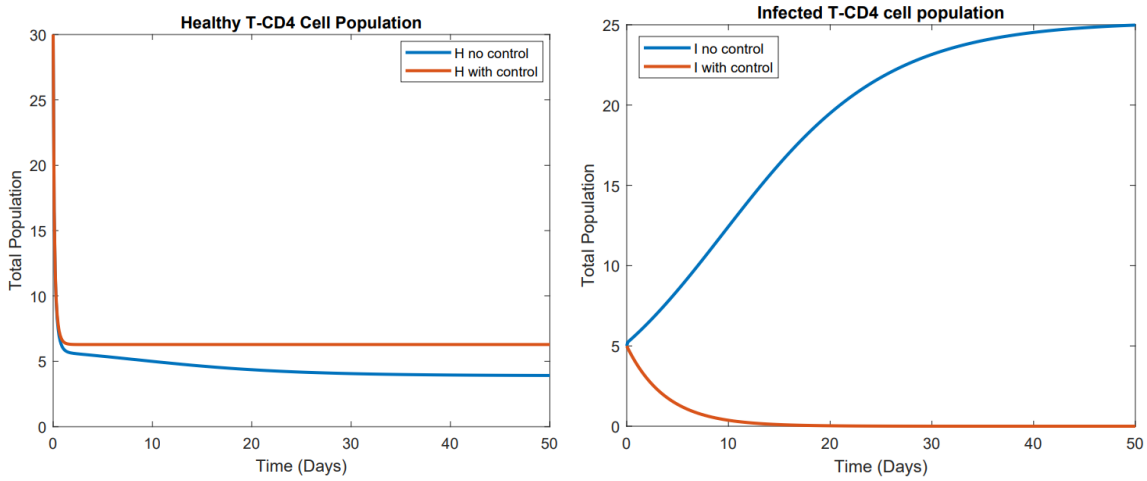
3.5 Analysis and Simulation Results

In this discussion, the initial values given are $H(0) = 30, I(0) = 3, V(0) = 5$ and the parameter values given in **Table 1**. From the discussion of stability analysis for the infection-free equilibrium point by inputting parameter values, $\lambda_1, \lambda_2, \lambda_3 < 0$, is obtained, the system around the infection-free equilibrium point is asymptotically stable. As for the infected equilibrium point, the Routh-Hurwitz table that needs to be considered is the component in the first column, namely A_0, A_1, b_1, c_1 and after submitting the parameter values, the values of A_0, A_1, b_1, c_1 are positive, so it can be concluded that the system around the infected equilibrium point is asymptotically stable. Then discussed the results of numerical simulations on the model equation of HIV growth in the body with antiretroviral therapy. Numerical simulations were carried out using the 4th-order Runge-Kutta method by inputting parameter values into the equation and comparing changes in population numbers before and after being given.

Table 1. Parameter Value of HIV Growth Model in the Body with Antiretroviral Therapy

Parameter	Description	Values	Source
s	The production rate of healthy T-CD4 cells	$10/mm^3$ hari	[10]
μ_0	The natural death rate of T-CD4 cells	0.02/hari	[10]
r	The growth rate of T-CD4 cells	0.3/hari	[16]
H_m	The maximal capacity of T-CD4 cells	$1000/mm^3$	[10]
k_1	The rate of infected T-CD4 cells	$0.002/mm^3$ hari	[16]
μ_1	The death rate of infected T-CD4 cells	0.26/hari	[10]
N	Source of virus	100	Assumed
k_2	The rate of HIV inside T-CD4 cells	$0.0024/mm^3$ hari	Assumed
μ_2	The death rate of HIV	3/hari	[16]

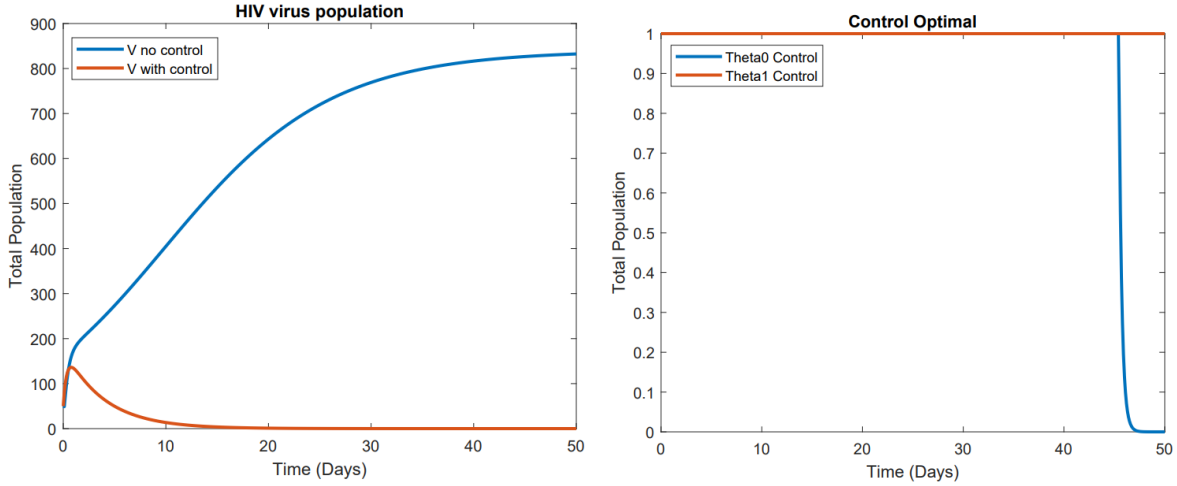
The following are the results of the numerical simulation comparison of the HIV growth model in the body with and without control:



(a) **(b)**

Figure 3. Comparison Graph of Changes in the Number of (a) Healthy T-CD4 Cell Population (b) Infected T-CD4 Cell Population

Figure 3 (a) shows that there is a difference in the healthy T-CD4 cell population before and after control. The total population of T-CD4 cells before control continued to decrease and then stabilized at 3,8. However, after the control, the T-CD4 cell population increased steadily until the 50th day at 7,6. This can happen because of the control in the form of NRTI therapy, which prevents the viral reverse transcriptase enzyme from copying RNA into DNA. So that without DNA, HIV and AIDS cannot reproduce themselves. In **Figure 3**, (b) it is obtained that the population of infected T-CD4 cells, after being given control, decreased when compared to before control. The population of infected T-CD4 cells, after being controlled, decreased to 0,0000068. However, if no control was given, then the population of infected T-CD4 cells increased to 29,5. This happened because of the control in the form of NRTI therapy.



(a) **(b)**

Figure 4. (a) Comparison Graph of Changes in the Number of HIV, (b) Graph of Changes in the Treatment of θ_0 and θ_1 Controls

Figure 4 (a) shows that there is a difference in the HIV before and after control. Before the control, the HIV population continued to increase to 984 on day 50. While after being given control, the HIV population decreased to 0,002. This shows that PI therapy can reduce the number of HIV that infect more healthy cells. **Figure 4** (b) shows the amount of control θ_0 and θ_1. The control θ_0 reaches a maximum of 1 until the 45th day. Then it decreases until it reaches a minimum of 0 on day 50. This shows that given the control, the population of healthy T-CD4 cells decreases because the virus is no longer detected in the viral load test for HIV. While in the control θ_2 until day 50 reaches a maximum of 1. This happens so that the HIV in the body cannot reproduce itself.

4. CONCLUSIONS

A mathematical model for the growth of HIV in the body has been developed with a control in the form of a combination of antiretroviral therapy, i.e., NRTI and PI. Based on the stability analysis of the equilibrium point, it is known that the disease-free equilibrium point is asymptotically stable. As for the infected equilibrium point, it can be concluded that the system around the infected equilibrium point is asymptotically stable. Then, numerical simulations were carried out using the 4th-order Runge Kutta method. The results of numerical simulations show that with the control of antiretroviral therapy, the population of T-CD4 cells grows significantly, which can improve the quality of life of patients. And the growth of HIV in the body can be inhibited so that it cannot reproduce itself.

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