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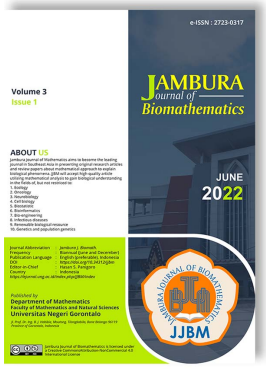

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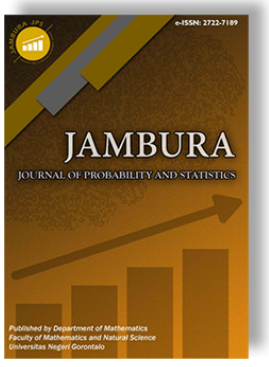

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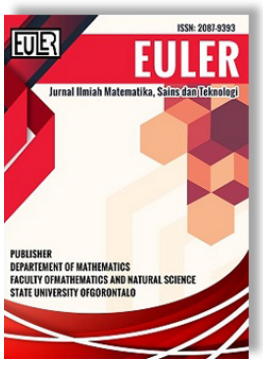

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Research Article

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Dynamical Properties of HIV/AIDS Model with Saturated Treatment

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ABSTRACT. *Mathematical models are crucial for developing control strategies, understanding disease transmission dynamics, and solving real-world problems. In this study, it applied a novel approach to the HIV/AIDS model. The saturated treatment was essentially used to model HIV/AIDS. A significant analysis of the HIV/AIDS epidemic model was presented, incorporating the new parameter. The mathematical analysis conducted on the model involved the examination of its boundedness, determination of its equilibria, calculation of the HIV/AIDS reproductive number, and assessment of the stability of these equilibria. The verification of the convergence analysis confirmed the effectiveness of the proposed scheme. The numerical results and simulations of the HIV/AIDS model are shown. Biologically, we have conducted investigations to determine the effect of several parameters on the dynamics of HIV/AIDS transmission.*

1. Introduction

Globally, infectious diseases are the cause of death in 25 percen of the fatalities. The spread of infectious diseases is affected by several factors, including media coverage, population migration, and changes in temperature [1]. The global health challenges posed by human immunodeficiency virus (HIV) and acquired immunodeficiency syndrome (AIDS) are immense. The HIV/AIDS pandemic, which was identified in the early 1980s, has profoundly affected global communities, with sub-Saharan Africa experiencing the most severe consequences. The global number of people living with HIV/AIDS reached approximately 37.7 million in 2023, with 1.5 million new cases reported annually [2]. Each year, millions of people die from human immunodeficiency virus (HIV), one of the deadliest and most contagious viruses in existence [3]. The public health crisis caused by HIV/AIDS has persisted worldwide. Despite significant advances in understanding and treating HIV / AIDS, it remains a major global health concern [4].

Mathematical models are valuable tools for developing public health strategies [5–12]. While the long-term accuracy of the mathematical model in predicting AIDS cases is questionable, a model based on disease transmission interactions could still help researchers to answer key questions. Several mathematical models have recently been developed to explain HIV/AIDS transmission patterns, such as [1, 3, 13–25] and the references mentioned therein. Watthanasirikosone and Modnak [15] offer a valuable framework for interventions and strategies to curb the spread of HIV/AIDS using SICA models for HIV transmission. Therefore, we recommend conducting this study.

Epidemiological models often incorporate saturation effects into treatment process descriptions. Some researchers have mathematically expressed saturation in their treatment. For example: Zhou and Chui [26] proposed the treatment function $T(I) = \frac{\sigma I}{k+I}$, where σ represents the maximum treatment rate, and k is the infection level at which the recovery rate is half its maximum $\left(T(k) = \frac{\sigma}{2}\right)$, thus indicating the speed of saturation. In 2020, Yadav and Srivastava considered a treatment function $T(I) = \frac{\sigma I}{1+rI}$, where σ is the treatment rate, and r is a constant related to the level of saturation. A larger r value implies stricter resource limitations. When $r = 0$, it indicates that the treatment is accessible to everyone [27]. Other forms of saturated treatments have been reported by Jana et al. [28] and Hu et al. [29]. Their work includes a treatment function, $T(I) = \frac{\sigma uI}{1+ruI}$, incorporating a control variable, u. The analysis showed that this function approaches zero for low values of I or u and approaches a finite limit as I becomes very large. The expression σ/r represents the maximum available therapeutic resources, whereas $1/(1 + ruI)$ represents the efficiency of these resources; both factors greatly influence the spread and control of disease.

Motivated by these various studies, we propose an epidemic model that considers saturated treatment for HIV/AIDS problems. This model was extended from the model by

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Watthanasirikosone and Modnak [15]. This article is structured as follows: Section 2 presents the model development for HIV/AIDS transmission. Section 3 covers positivity, boundedness, equilibria, HIV/AIDS reproductive number, and stability. Section 4 provides a numerical simulation to confirm the analytical results presented graphically. In Section 5, the conclusions are presented.

2. Model Formulation For HIV/AIDS

In this section a mathematical model for antiretroviral therapy and AIDS treatment is presented. The model divides the human population ($\mathcal{M}$) into four groups: susceptible individuals (S), HIV-infected individuals (I), AIDS patients (A), and those with controlled HIV/AIDS (C). The controlled class, designated by "C", includes people with HIV who are currently asymptomatic due to antiretroviral therapy (ART), even though the virus remains in their bloodstream. Moreover, knowledge of HIV status among class members leads to more careful behavior to prevent the spread of the disease. We posit the average birth and death rates for b and d, respectively. Let h_1 and h_3 denote the mortality rates of HIV and AIDS, respectively. Susceptible individuals acquire HIV through HIV, AIDS, or controlled individuals at rates α_1, α_2, and α_3, respectively. The HIV infection rate in susceptible individuals is α_1 for HIV-positive individuals, α_2 for AIDS patients, and α_3 for individuals under medical observation.

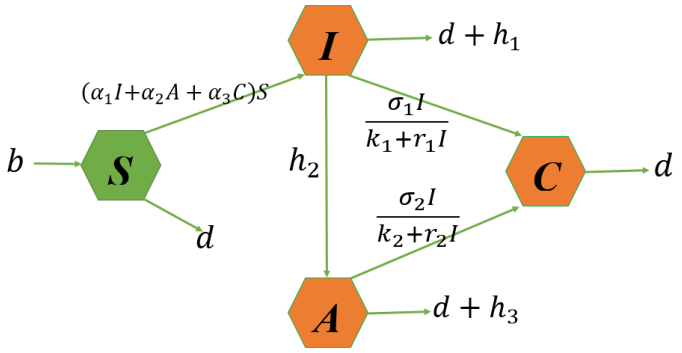

Figure 1. Scheme of HIV/AIDS transmission

Antiretroviral therapy in HIV/AIDS patients is important to optimally suppress the virus so that it can delay or prevent the development of chronic kidney disease [30]. However, in reality, diverse geographic structures have become challenges in healthcare services. Limited healthcare resources can lead to delays in healthcare services, including treatment for infected patients [31, 32]. Based on considerations of the geographical challenges in health services, we propose a mathematical form of HIV/AIDS treatment with different saturations from [26, 27] as below. For treatment on the HIV and AIDS patients, respectively

$$T_1(I) = \frac{\sigma_1 I}{k_1 + r_1 I} \quad \text{and} \quad T_2(I) = \frac{\sigma_2 I}{k_2 + r_2 I}$$

where, we assume treatment rates of σ_1 for HIV-infected individuals receiving ART, and σ_2 for AIDS patients receiving medical care. The treated patients were placed in the control group. In addition, individuals with HIV progress to AIDS at a rate of h_2. k_1, k_2 represent the half-saturation parameters and measure how soon saturation occurs. k_1 is the size of the virus in the HIV case at saturation 50 %, and k_2 is the size infected in the AIDS case at saturation 50 %. Next, r_1 indicates the significant impact of infection on HIV patients who are delayed for treatment. Next, r_2 indicates the significant impact of infection in AIDS patients who are delayed in treatment.

Thus, our model takes the following form (see Figure 1 for its diagram):

$$\begin{aligned}
\frac{dS}{dt} &= b - \alpha_1 IS - \alpha_2 AS - \alpha_3 CS - dS,\\
\frac{dI}{dt} &= \alpha_1 IS + \alpha_2 AS + \alpha_3 CS - dI - h_1 I - h_2 I - \frac{\sigma_1 I}{k_1 + r_1 I},\\
\frac{dA}{dt} &= h_2 I - dA - h_3 A - \frac{\sigma_2 A}{k_2 + r_2 A},\\
\frac{dC}{dt} &= \frac{\sigma_1 I}{k_1 + r_1 I} + \frac{\sigma_2 A}{k_2 + r_2 A} - dC,
\end{aligned} \tag{1}$$

and initial condition

$$S > 0, I \geq 0, A \geq 0, C \geq 0. \tag{2}$$

Table 1. The description of HIV/AIDS model parameter

Parameter	Description	Value	References
b	Birth rate	10	Assumption
d	Natural death rate	0.03	[33]
α_1	Transmission rate from HIV group	0.00045	[15]
α_2	Transmission rate from AIDS group	0.000025	[15]
α_3	Transmission rate from controlled group	0.00015	[15]
h_1	Rate of the disease-related death of I	0.1	[15, 34]
h_2	Rate progression to A for I	0.05	[15, 34]
h_3	Rate of the disease-related death of A	0.2	[15, 34]
σ_1	Rate of treatment for HIV patients	0.7	[15]
σ_2	Rate of treatment for AIDS patients	0.7	[15]
k_1	Speed of saturation for HIV group	0.1-0.7	Assumption
k_2	Speed of saturation for AIDS group	0.1-0.7	Assumption
r_1	Resource limitations for HIV patient to treatment	0.1-0.7	Assumption
r_2	Resource limitations for AIDS patient to treatment	0.1-0.7	Assumption

3. Analytical Results

3.1. Positivity and boundedness

Theorem 1. *Any solution (S, I, A, C) to system (1) subject to nonnegative initial conditions (2) remains positive for all $t > 0$.*

Proof. From HIV/AIDS model in system (1), we have

$$\begin{aligned}
\left.\frac{dS}{dt}\right|_{S=0} &= b > 0,\\
\left.\frac{dI}{dt}\right|_{I=0} &= \alpha_1 IS + \alpha_2 AS + \alpha_3 CS \geq 0,\\
\left.\frac{dA}{dt}\right|_{A=0} &= h_2 I \geq 0,
\end{aligned} \tag{3}$$

$$\left.\frac{dC}{dt}\right|_{C=0} = \frac{\sigma_1 I}{k_1 + r_1 I} + \frac{\sigma_2 A}{k_2 + r_2 A} \geq 0.$$

The preceding calculation demonstrates that all rates are nonnegative within the boundaries of the positive region $\mathbb{R}^4_+$. Thus, the solutions are confined to the positive region whenever the system's initial state is in the nonnegative region $\mathbb{R}^4_+$. □

Theorem 2. *The solutions of system (1) are bounded on the interval* $t \in [0, t_0]$.

Proof. Since we adding all equation in system (1), we get

$$\mathcal{M}(t) = S(t) + I(t) + A(t) + C(t). \tag{4}$$

Next, By substituting system (1) into the derivative results from eq. (4), we get

$$\begin{aligned}\frac{d\mathcal{M}}{dt} &= \frac{dS}{dt} + \frac{dI}{dt} + \frac{dA}{dt} + \frac{dC}{dt},\\ &= b - h_1 I - h_3 A - d\mathcal{M},\\ &\leq b - d\mathcal{M}.\end{aligned}$$

Thusly,

$$0 \leq \lim_{x\to\infty} \sup \mathcal{M}(t) \leq \frac{b}{d},$$

Consequently, all solutions pertaining to the system (1) are definitively bounded for all $t \in [0, t_0]$. Thus, the specified region is

$$\Omega = \left\{(S, I, A, C) \in \mathbb{R}^4_+ : 0 \leq \mathcal{M}(t) \leq \frac{b}{d}\right\}.$$

□

3.2. Equilibria and HIV/AIDS reproductive number

System (1) always had a HIV/AIDS-free equilibria which given by

$$\Sigma_0 = \left(\frac{b}{d}, 0, 0, 0,\right)$$

To determine reproductive number $\mathfrak{R}_0$, from system (1) we have the $\mathcal{F}$ transition and $\mathcal{V}$ transmission matrices, where

$$\mathcal{F} = \begin{pmatrix}\alpha_1 IS + \alpha_2 AS + \alpha_3 CS\\ 0\\ 0\end{pmatrix},$$

$$\mathcal{V} = \begin{pmatrix}(d + h_1 + h_2 + \frac{\sigma_1}{k_1 + r_1 I})I\\ (d + h_3)A + \frac{\sigma_2 A}{k_2 + r_2 A} - h_2 I\\ dC - \frac{\sigma_1 I}{k_1 + r_1 I} - \frac{\sigma_2 A}{k_2 + r_2 A}\end{pmatrix}.$$

Biologically, F is entrywise non-negative and V is a non-singular M-matrix, so V^{-1} is entrywise nonnegative. Define $F = \left[\frac{\partial \mathcal{F}_i(y_0)}{\partial y_j}\right]$ and $V = \left[\frac{\partial \mathcal{V}_i(y_0)}{\partial y_j}\right]$ for $i \leq 1, j \leq m$. In addition, $\frac{dy_i}{dt} = \mathcal{F}_i(y) - \mathcal{V}_i(y)$ for $i = 1,..., m$, where $y = (y_1, y_2, ..., y_n)^T$ is the number of individuals in each compartment [35]. Next, HIV/AIDS reproductive number is given by

$$F = \begin{pmatrix}\frac{\alpha_1 b}{d} & \frac{\alpha_2 b}{d} & \frac{\alpha_3 b}{d}\\ 0 & 0 & 0\\ 0 & 0 & 0\end{pmatrix},$$

$$V = \begin{pmatrix}d + h_1 + h_2 + \frac{\sigma_1}{k_1} & 0 & 0\\ -h_2 & d + h_3 + \frac{\sigma_2}{k_2} & 0\\ -\frac{\sigma_1}{k_1} & -\frac{\sigma_2}{k_2} & d\end{pmatrix}.$$

Further, the HIV/AIDS reproductive number is then determined as the spectral radius of FV^{-1}, which yields

$$\begin{aligned}\mathfrak{R}_0 = &\frac{k_1 k_2 \left(\alpha_1 b \left(d + h_3 + \frac{\sigma_2}{k_2}\right) + \alpha_2 b h_2\right)}{k_1 k_2 \left(d + h_1 + h_2 + \frac{\sigma_1}{k_1}\right)\left(d + h_3 + \frac{\sigma_2}{k_2}\right) d}\\ &+ \frac{\frac{\alpha_3 b}{d}\left(\sigma_2 h_2 k_1 + k_2 \sigma_1 \left(d + h_3 + \frac{\sigma_2}{k_2}\right)\right)}{k_1 k_2 \left(d + h_1 + h_2 + \frac{\sigma_1}{k_1}\right)\left(d + h_3 + \frac{\sigma_2}{k_2}\right) d}.\end{aligned}$$

Next, HIV/AIDS equilibria which given by

$$\Sigma_* = (S^*, I^*, A^*, C^*),$$

where

$$\begin{aligned}S^* =& \frac{b}{d + \alpha_1 I^* + \alpha_2 A^* + \alpha_3 C^*},\\ I^* =& \frac{A^*}{h_2}\left(d + h_3 + \frac{\sigma_2}{k_2 + r_2 A^*}\right),\\ A^* =& \frac{I^*}{\alpha_2 S^*}\left(d + h_1 + h_2 + \frac{\sigma_1}{k_1 + r_1 I^*}\right) - \frac{\alpha_1 I^* + \alpha_3 C^*}{\alpha_2},\\ C^* =& \frac{\sigma_1 I^*}{(k_1 + r_1 I^*)d} + \frac{\sigma_1 A^*}{(k_2 + r_2 A^*)d}.\end{aligned}$$

3.3. Stability of HIV/AIDS-free equilibria

Theorem 3. *The HIV/AIDS free equilibria,* Σ_0 *is locally asymptotically stable if* $\mathfrak{R}_0 < 1$.

Proof. Consider the Jacobian matrix at Σ_0 then

$$J(\Sigma_0) = \begin{pmatrix}-d & -\alpha_1 S_0 & -\alpha_2 S_0 & -\alpha_3 S_0\\ 0 & \eta_{21} & \alpha_2 S_0 & \alpha_3 S_0\\ 0 & h_2 & -\eta_{31} & 0\\ 0 & \sigma_1 & \sigma_2 & -d\end{pmatrix}, \tag{5}$$

where

$$\begin{aligned}\eta_{21} &= \alpha_1 S_0 - (d + h_1 + h_2 + \frac{\sigma_1}{k_1}),\\ \eta_{31} &= (d + h_3 + \frac{\sigma_2}{k_2}).\end{aligned}$$

From the matrix $J(\Sigma_0)$ in eq. (5), we get a characteristic equation

$$K(\lambda) = \lambda^4 + b_1\lambda^3 + b_2\lambda^2 + b_3\lambda + b_4, \tag{6}$$

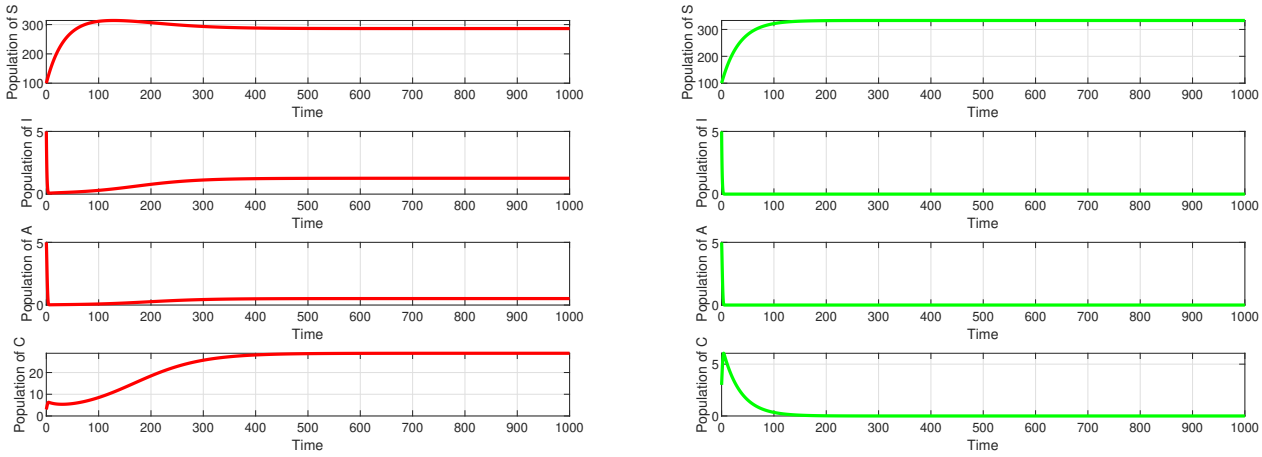

Figure 2. (a) Dynamics of HIV/AIDS transmission when $\Re_0 > 1$. (b) Dynamics of HIV/AIDS transmission when $\Re_0 < 1$.

where

$$
\begin{aligned}
b_1 &= 2d + \eta_{31} - \eta_{21},\\
b_2 &= d^2 + 2d\eta_{31} - (S_0\alpha_2 h_2 + S_0\alpha_3\sigma_1 + 2d\eta_{21} + \eta_{21}\eta_{31}),\\
b_3 &= d^2\eta_{31} - (2d\eta_{21}\eta_{31} + d^2\eta_{21} + S_0\alpha_3 h_2\sigma_2 + S_0\alpha_3\eta_{31}\sigma_1\\
&\quad + S_0 d\alpha_3\sigma_1 + 2S_0 d\alpha_2 h_2),\\
b_4 &= (d\alpha_2 h_2 + \alpha_3\eta_{31}\sigma_1 + \alpha_3 h_2\sigma_2)(-S_0 d) - d^2\eta_{21}\eta_{31}.
\end{aligned}
$$

From $K(\lambda)$ in eq. (6), we deduce $\mathrm{Re}(\lambda_i), i = 1, 2, 3, 4$. Therefore, the HIV / AIDS-free equilibria are locally asymptotically stable, given their existence. The numerical representation of the analytical results is visible in eq. (14). Based on these results, we observe that using the parameter values presented in Table 1 consistently, except $\alpha_1 = 0.00000011$, $\alpha_2 = 0.00000021$, $\alpha_3 = 0.000000015$ produces negative real parts of the eigenvalues. □

Theorem 4. *The HIV/AIDS free equilibria Σ_0 of the system (1) are globally asymptotically stable for $\Re_0 < 1$.*

Proof. Define the Lyapunov function

$$V_0 = \omega_1 I + \omega_2 A + \omega_3 C, \tag{7}$$

where

$$
\begin{aligned}
\omega_1 &= d\sigma_1 k_2\left(d + h_3 + \frac{\sigma_2}{k_2}\right),\\
\omega_2 &= d\sigma_2 k_1\left(d + h_1 + h_2 + \frac{\sigma_1}{k_1}\right),\\
\omega_3 &= k_1 k_2\left(\alpha_1 b\left(d + h_3 + \frac{\sigma_2}{k_2}\right) + \alpha_2 b h_2\right)\\
&\quad + \frac{\alpha_3 b}{d}\left(\sigma_2 h_2 k_1 + k_2\sigma_1\left(d + h_3 + \frac{\sigma_2}{k_2}\right)\right).
\end{aligned}
$$

The derivative of V_0 with respect to t is

$$
\begin{aligned}
\frac{dV_0}{dt} &= \omega_1\frac{dI}{dt} + \omega_2\frac{dA}{dt} + \omega_3\frac{dC}{dt}\\
&= \omega_1\left(\alpha_1 IS + \alpha_2 AS + \alpha_3 CS - dI - h_1 I - h_2 I\right.\\
&\quad \left. - \frac{\sigma_1 I}{k_1 + r_1 I}\right) + \omega_2\left(h_2 I - dA - h_3 A - \frac{\sigma_2 A}{k_2 + r_2 A}\right)\\
&\quad + \omega_3\left(\frac{\sigma_1 I}{k_1 + r_1 I} + \frac{\sigma_2 A}{k_2 + r_2 A} - dC\right)\\
&= \omega_3\left(\frac{\sigma_1 I}{k_1 + r_1 I} + \frac{\sigma_2 A}{k_2 + r_2 A}\right) - \omega_1\left(d + h_1 + h_2\right.\\
&\quad \left. + \frac{\sigma_1}{k_1 + r_1 I}\right) I - \omega_2\left(d + h_3 + \frac{\sigma_2}{k_2 + r_2 A}\right) A\\
&= \omega_3\left(\frac{\sigma_1 I}{k_1 + r_1 I}\right) - \omega_1\left(d + h_1 + h_2 + \frac{\sigma_1}{k_1 + r_1 I}\right) I\\
&\quad + \omega_3\left(\frac{\sigma_2 A}{k_2 + r_2 A}\right) - \omega_2\left(d + h_3 + \frac{\sigma_2}{k_2 + r_2 A}\right) A\\
&\leq \left(\omega_3\frac{\sigma_1}{k_1} - \omega_1\left(d + h_1 + h_2 + \frac{\sigma_1}{k_1}\right)\right) I + \left(\omega_3\frac{\sigma_2}{k_2}\right.\\
&\quad \left. - \omega_2\left(d + h_3 + \frac{\sigma_2}{k_2}\right)\right) A
\end{aligned}
\tag{8}
$$

Substituting ω_1, ω_2, and ω_3 in eq. (7) into eq. (8), we get

$$\frac{dV_0}{dt} \leq (\Re_0 - 1)(I + A).$$

Clearly, $\frac{dV_0}{dt} < 0$ for $\Re_0 < 1$. This led to the HIV/AIDS-free point (Σ_0) becoming globally asymptotically stable. □

3.4. Stability of HIV/AIDS equilibria

Theorem 5. *The HIV/AIDS equilibria, Σ_* is locally asymptotically stable if $\Re_0 > 1$.*

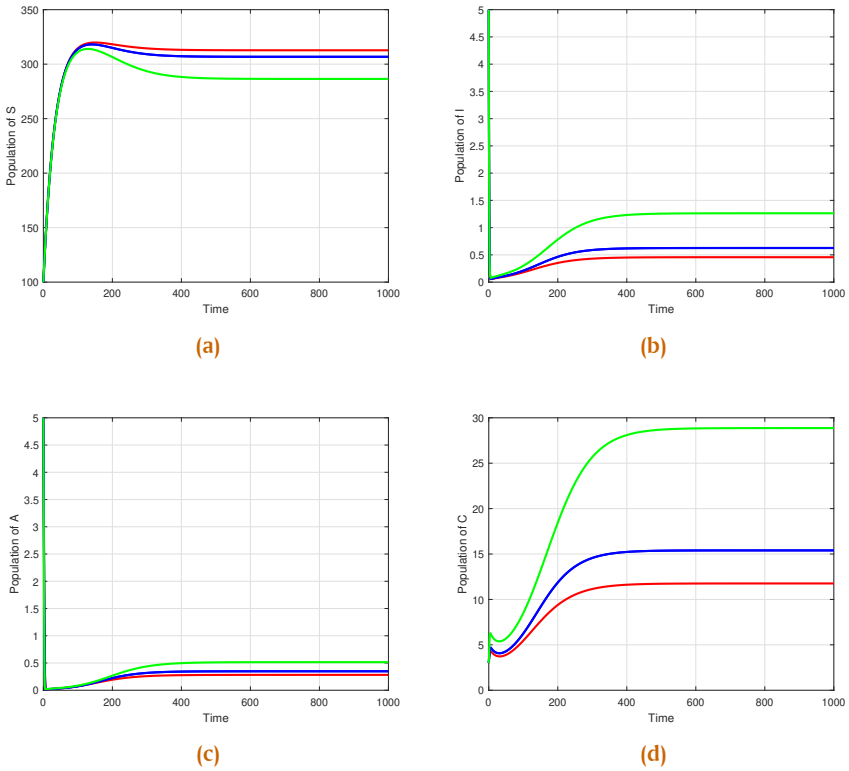

Figure 3. Simulation of the speed of saturation for HIV parameter (k_1) with respect to time (t) for each compartments (a) S, (b) I, (c) A, and (d) C. The red, blue, and green lines represent $k_1 = 0.7$, $k_1 = 0.4$, and $k_1 = 0.1$, respectively

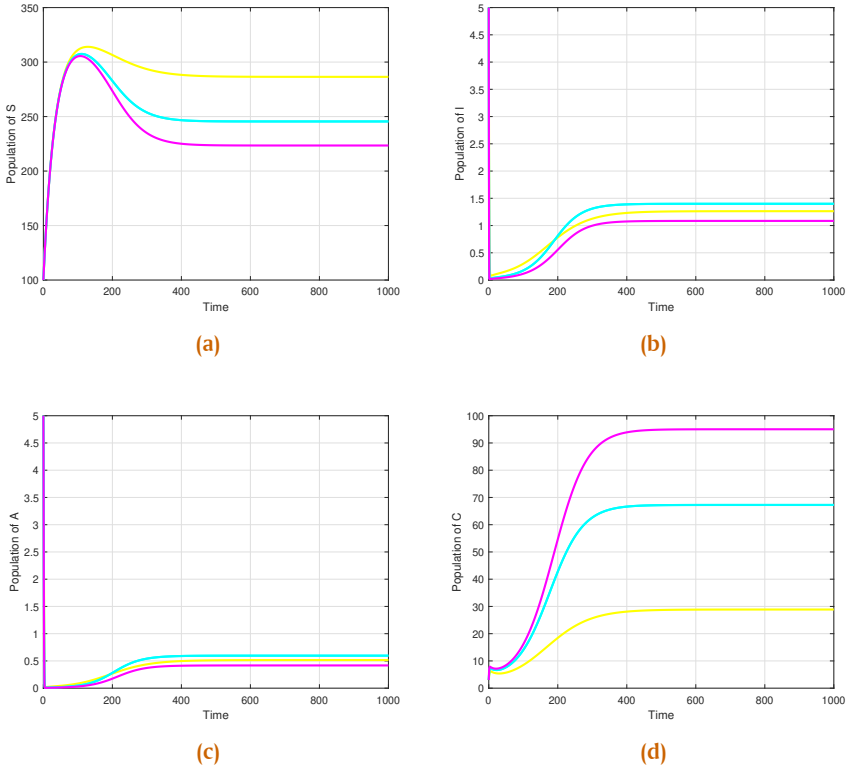

Figure 4. Simulation of the effects of unawareness parameter (k_2) with respect to time (t) for each compartments (a) S, (b) I, (c) A, and (d) C. The magenta, cyan, and yellow lines represent $k_2 = 0.7$, $k_2 = 0.4$, and $k_2 = 0.1$, respectively.

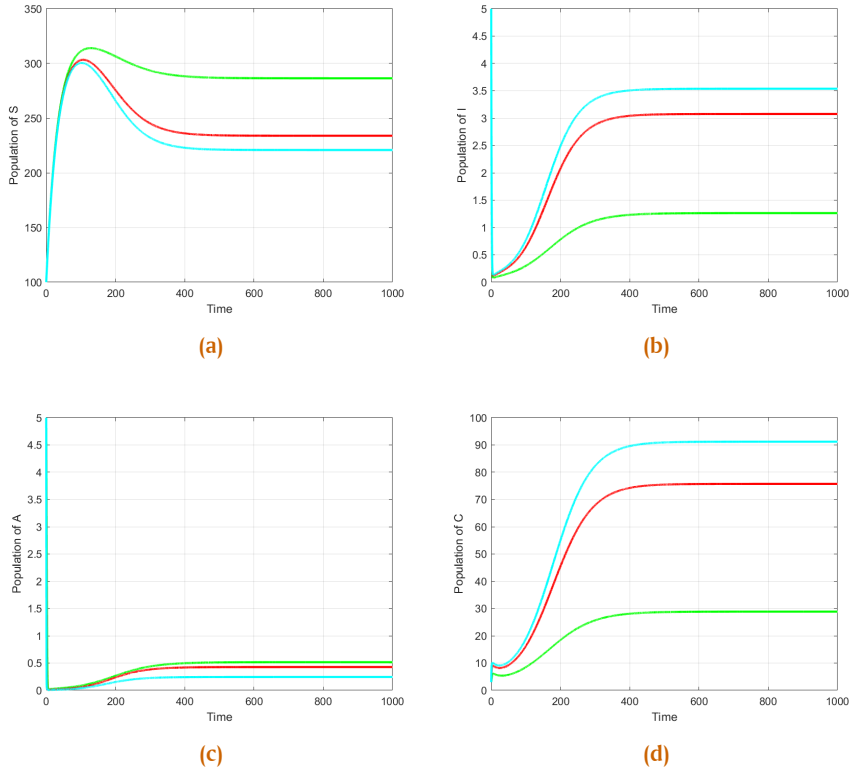

Figure 5. Simulation of the effects of unawareness parameter (r_1) with respect to time (t) for each compartments (a) S, (b) I, (c) A, and (d) C. The cyan, red, and green lines represent $r_1 = 0.7$, $r_1 = 0.4$, and $r_1 = 0.1$, respectively.

Proof. Consider the Jacobian matrix at Σ_* then

$$J(\Sigma_*) = \begin{pmatrix} -\phi_{11} & -\alpha_1 S^* & -\alpha_2 S^* & -\alpha_3 S^* \\ \phi_{21} & \phi_{22} & \alpha_2 S^* & \alpha_3 S^* \\ 0 & h_2 & -\phi_{31} & 0 \\ 0 & \phi_{41} & \phi_{42} & -d \end{pmatrix}, \tag{9}$$

where

$$\begin{aligned} \phi_{11} &= (d + \alpha_1 I^* + \alpha_2 A^* + \alpha_3 C^*), \\ \phi_{21} &= \alpha_1 I^* + \alpha_2 A^* + \alpha_3 C^*, \\ \phi_{22} &= \alpha_1 S^* - (d + h_1 + h_2) - \frac{\sigma_1}{(k_1 + r_1 I)^2}, \\ \phi_{31} &= (d + h_3) + \frac{\sigma_2 k_2}{k_2 + r_2 A}, \\ \phi_{41} &= \frac{\sigma_1 k_1}{k_1 + r_1 I^*}, \\ \phi_{42} &= \frac{\sigma_2 k_2}{k_2 + r_2 A^*}. \end{aligned}$$

From the matrix $J(\Sigma_*)$ in eq. (9), we get a characteristic equation

$$L(\lambda) = \lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4, \tag{10}$$

where

$$\begin{aligned} a_1 &= d + \phi_{31} + \phi_{11} - \phi_{22}, \\ a_2 &= \alpha_1\phi_{21}S^* + d\phi_{11} + d\phi_{31} + \phi_{11}\phi_{31} - (S^*\alpha_2 h_2 + S^*\alpha_3\phi_{41} \\ &\quad + d\phi_{22} + \phi_{11}\phi_{22} + \phi_{22}\phi_{31}), \\ a_3 &= S^*\alpha_2 h_2\phi_{21} + S^* d\alpha_1\phi_{21} + S^*\alpha_1\phi_{21}\phi_{31} + S^*\alpha_3\phi_{21}\phi_{41} \\ &\quad + d\phi_{11}\phi_{31} - (S^* d\alpha_2 h_2 + S^*\alpha_2 h_2\phi_{11} + S^*\alpha_3\phi_{11}\phi_{41} \\ &\quad + S^*\alpha_3\phi_{31}\phi_{41} + S^*\alpha_3 h_2\phi_{42}) - (d\phi_{22}\phi_{31} + d\phi_{11}\phi_{22} \\ &\quad + \phi_{11}\phi_{22}\phi_{31}), \\ a_4 &= S^* d\alpha_2 h_2\phi_{21} + S^*\alpha_3 h_2\phi_{21}\phi_{42} + S^*\alpha_3\phi_{21}\phi_{31}\phi_{41} \\ &\quad + S^* d\alpha_1\phi_{21}\phi_{31} - (d\phi_{11}\phi_{22}\phi_{31} + S^* d\alpha_2 h_2\phi_{11} \\ &\quad + S^*\alpha_3 h_2\phi_{11}\phi_{42} + S^*\alpha_3\phi_{11}\phi_{31}\phi_{41}). \end{aligned}$$

From $L(\lambda)$ in eq. (10), we deduce $\mathtt{Re}(\lambda_i), i = 1, 2, 3, 4$. Therefore, the HIV / AIDS equilibria are locally asymptotically stable, given their existence. The numerical representation of the analytical results is visible in eq. (13). Based on these results, we observe that using the parameter values presented in Table 1 consistently yields negative real parts of the eigenvalues. □

Theorem 6. *If $\mathfrak{R}_0 > 1$, HIV/AIDS equilibria Σ_* exist and are globally asymptotically stable, whereas when $\mathfrak{R}_0 < 1$, Σ_* is unstable.*

Proof. The global stability of system (1) is under analysis at the point Σ_*, provided that the conditions for the existence of Σ_* are satisfied, and that $\mathfrak{R}_0 > 1$. The definition and derivation of Lyapunov function V_* are as follows:

$$V_* = \frac{1}{2}\left[(S - S^*) + (I - I^*) + (A - A^*) + (C - C^*)\right]^2. \tag{11}$$

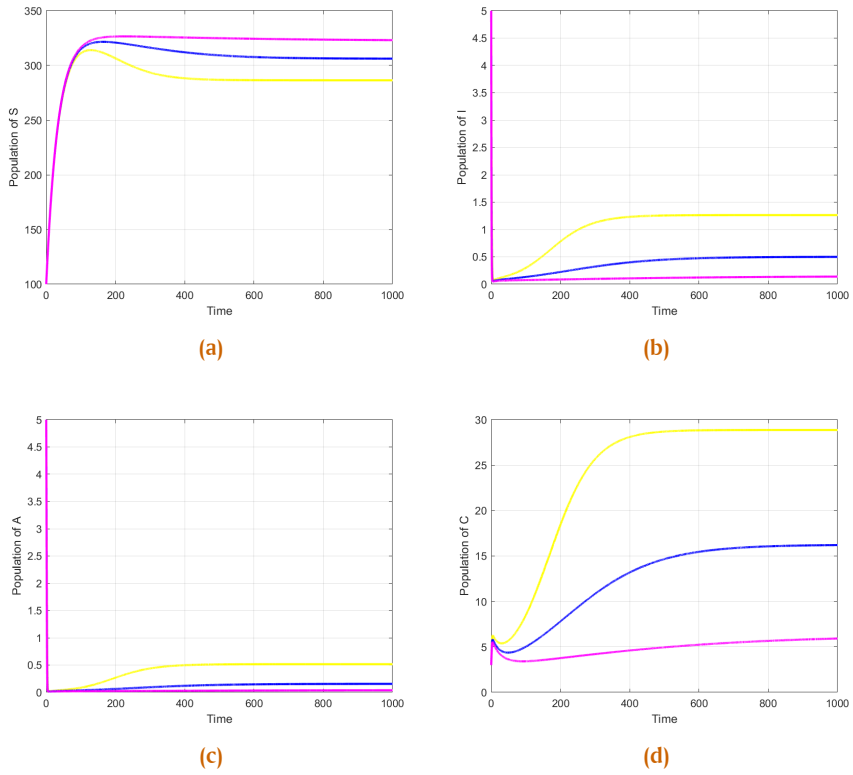

Figure 6. Simulation of the effects of unawareness parameter (r_2) with respect to time (t) for each compartments (a) S, (b) I, (c) A, and (f) C. The magenta, blue, and yellow lines represent $r_2 = 0.7$, $r_2 = 0.4$, and $r_2 = 0.1$, respectively.

The time derivative of the function V_* in eq. (11), considering system (1), yields

$$\frac{dV_*}{dt} = [(S - S^*) + (I - I^*) + (A - A^*) + (C - C^*)]\,\frac{d\mathcal{M}}{dt}.$$

This is because $[S + I + A + C] = \frac{b}{d}$ and $\frac{d\mathcal{M}}{dt} = [b - d\mathcal{M}]$. It follows that

$$\frac{dV_*}{dt} = -\frac{1}{d}[b - d\mathcal{M}]^2. \tag{12}$$

$\frac{dV_*}{dt} < 0$ is strictly a Lyapunov function, as presented in eq. (12). Consequently, the HIV/AIDS equilibrium point Σ_* exhibits global asymptotic stability for $\mathfrak{R}_0 > 1$ within region Ω (see Theorem 2). From eq. (12), $\frac{dL_*}{dt} = 0$ if and only if we set $S = S^*, I = I^*, A = A^*$, and $C = C^*$, and $\frac{dV_*}{dt}$ converges in the positive regions Ω as $t \to \infty$. □

4. Numerical Results

This section presents a numerical simulation to illustrate the behavior of an HIV/AIDS model that incorporates saturation treatment. This is expected to support the preceding analysis. In the context of the numerical examples, we assume certain hypothetical values for the parameters, as shown in Table 1. Using the parameter values in Table 1, we obtain $\mathfrak{R}_0 = 1.657020423 > 1$, where

$$\lambda_1 = -1.652548, \lambda_2 = -0.205124, \lambda_3 = -7.120269, \quad (13)$$
$$\lambda_4 = -0.299999.$$

The existence and stability of endemic equilibrium points are presented here. The population dynamics for each compartment is shown in Figure 2a. Subsequently, apply the parameter values specified in Table 1, excluding only $\alpha_1 = 0.00000011$, $\alpha_2 = 0.00000021$, and $\alpha_3 = 0.000000015$. We obtain $\mathfrak{R}_0 = 0.000168786 < 1$, which shows the stability of the HIV-free points, where

$$\lambda_1 = -0.300000, \lambda_2 = -7.179903, \lambda_3 = -7.230065, \quad (14)$$
$$\lambda_4 = -0.299951.$$

Figure 2b shows the dynamics of the population in each compartment.

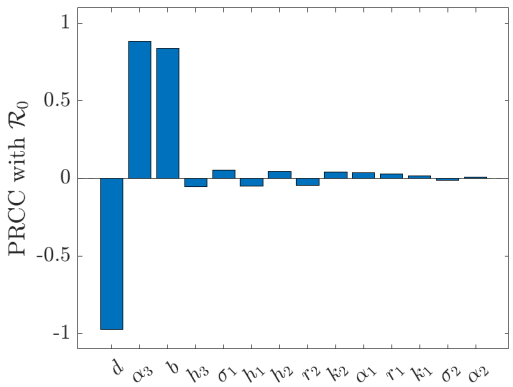

Figure 7. Partial rank correlation coefficient (PRCC) between model parameters and $\mathfrak{R}_0$ for the HIV/AIDS model.

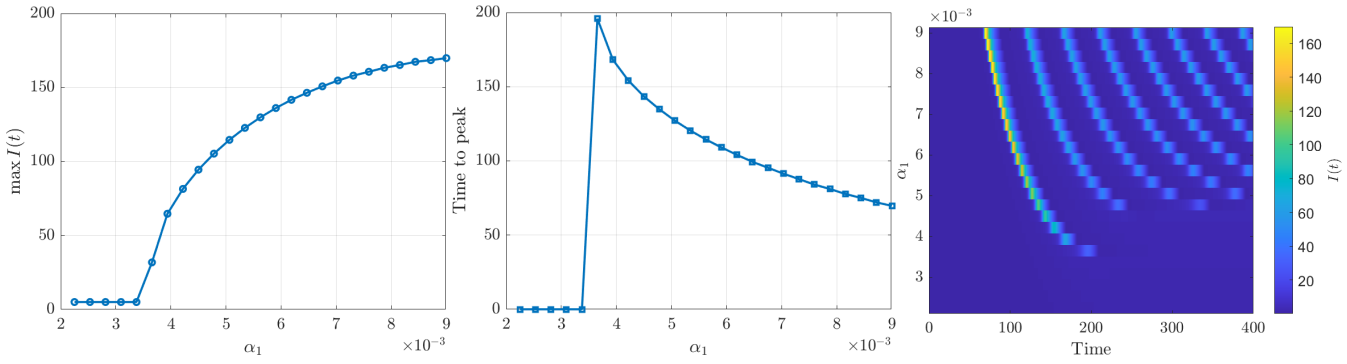

Figure 8. Impact of the transmission-related parameter α_1 on the infection dynamics. Panel (a) shows the influence of α_1 on the maximum number of infected individuals, max $I(t)$. Panel (b) shows the influence of α_1 on the time required to reach the peak. Panel (c) provides a heatmap of the temporal dynamics of $I(t)$ across different α_1. For these simulations, we use $\alpha_1 = 0.0045$.

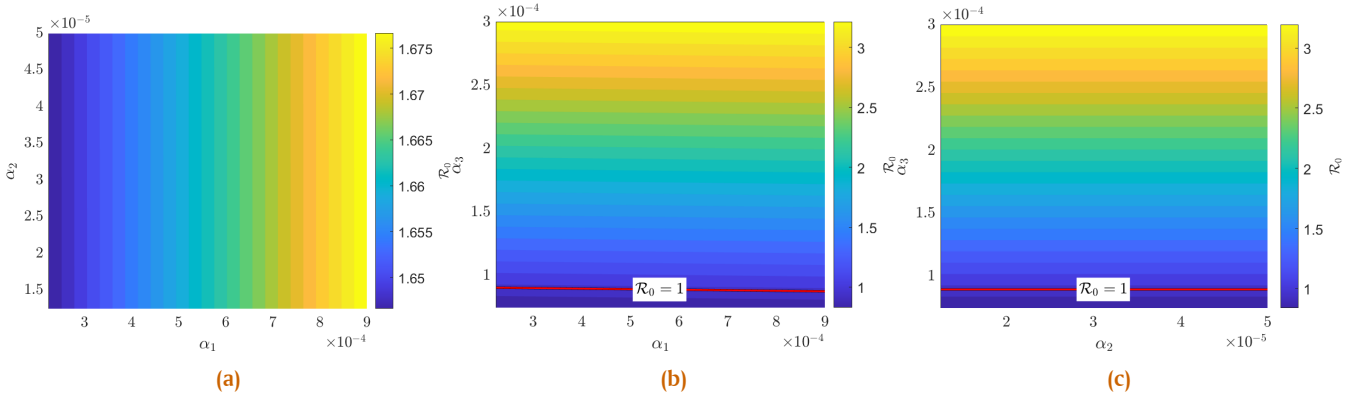

Figure 9. Contour plots of the basic reproduction number $\mathfrak{R}_0$ under pairwise variations of the transmission parameters α_1, α_2, and α_3. The red curves in (b) and (c) indicate the threshold $\mathfrak{R}_0 = 1$.

4.1. Effects of k_1 and k_2

The subsequent subsection provides an analysis of the effect of parameter k_1 resulting from speed saturation within the HIV group. We choose $k_1 = 0.1, 0.4, 0.7$ and consistently use the values in Table 1. Increased speed saturation in the HIV group led to a reduction in the population size of the I, A, C groups be reduced. This change is due to the level of infection at which the control treatment is half its saturation, such that the susceptible individuals S are increased. The details of this behavior are presented in Figure 3. Next, We choose $k_1 = 0.1, 0.4, 0.7$. Increased speed saturation in the HIV group led to a reduction in the population size of the I, A groups. However, the number of treatment controls is expected to increase. For the detailed dynamics, see Figure 4. Thus, the speed saturation parameters for HIV / AIDS play a role in controlling the impact of infection. This shows that geographic range has a significant impact on HIV/AIDS treatment. Patients in urban areas with well-funded healthcare systems may have more extensive access to cutting-edge treatments than those in rural or underserved regions, where healthcare infrastructure may be less robust.

4.2. Effect of parameter r_1 and r_2

To study the implications of implementing resource limitations in the system (1), we investigate the effects of the parameters r_1 and r_2. Selected value of $r_1 = 0.1, 0.4,$ and 0.7. Increased the value of parameter r_1 causing the peak number of HIV individuals to increase. A decrease in the number of AIDS patients resulted in more individuals being treated. Changes in the value of parameter r_1 affect the population of each class; S, I, A, and C are presented in Figure 5. Next, we selected value of $r_2 = 0.1, 0.4,$ and 0.7. Increased the value of parameter r_2 also caused the peak of HIV, AIDS, and Treatment individuals to decrease. For the detailed dynamics, see Figure 6. These results indicate that a policy designed to elevate treatment intensity is capable of reducing mortality rates for HIV/AIDS. This shows that limited health resources significantly influence the spread of HIV/AIDS. Limited health resources have an impact on the increasing number of patients, so improvements to various resources to maximize health services are very necessary.

4.3. Sensitivity analysis via PRCC

Partial rank correlation coefficient (PRCC) analysis is a global sensitivity method that measures the monotonic influence of parameters on a model outcome, while accounting for the effects of other parameters. Using Latin Hypercube Sampling (LHS) with $N = 1000$ parameter sets drawn from plausible ranges around the baseline values in Table 1, the closed-form $\mathfrak{R}_0$ was computed for each sample and PRCC values were

obtained via rank regression residuals. The results in Figure 7 show that the natural mortality rate d has the strongest negative effect on $\mathfrak{R}_0$, whereas the recruitment rate b and the transmission rate from controlled individuals α_3 exert the largest positive effects. Other parameters, including disease-induced mortalities, treatment rates, half-saturation constants, and resource-limitation terms, have comparatively minor influences, suggesting that interventions reducing α_3 and b are most effective in lowering $\mathfrak{R}_0$.

4.4. *Impact of α_1 on epidemic dynamics*

Figure 8 illustrates the influence of the transmission-related parameter α_1 on epidemic dynamics. As shown in panel (a), when α_1 is below a critical threshold, the maximum number of infected individuals remains low; however, once this threshold is exceeded, max $I(t)$ increases rapidly, indicating the onset of a major outbreak. Panel (b) reveals that the time to peak prevalence is longest near the threshold value but decreases monotonically as α_1 increases, implying that stronger transmission not only raises the epidemic magnitude but also accelerates its progression. The heat map in panel (c) further confirms these observations by showing that higher α_1 values lead to earlier and more intense outbreaks, with infection levels reaching substantially larger peaks over time. These results highlight the pivotal role of α_1 in determining the severity and timing of an epidemic.

4.5. *Contour and sensitivity analysis of $\mathfrak{R}_0$*

The contour plots in Figure 9 demonstrate how pairwise changes in the transmission parameters affect the basic reproduction number, $\mathfrak{R}_0$. In panel (a), varying α_1 and α_2 results in only a slight increase in $\mathfrak{R}_0$, with the effect more pronounced along the α_1 direction, indicating its greater influence. Panels (b) and (c) show that increasing α_3 significantly increases $\mathfrak{R}_0$, driving it above the epidemic threshold, whereas sufficiently small values of α_3 can reduce $\mathfrak{R}_0$ below unity. The red curves identify the critical parameter regions where $\mathfrak{R}_0 = 1$, separating the disease-free regime from the persistence regime and emphasizing the dominant role of α_3 in determining the outbreak potential.

5. Conclusion

We are developing a mathematical model. It describes how HIV/AIDS spreads. The model includes saturated treatment. The nonlinear mathematical model validates prior modeling investigations found within the existing literature. The fundamental properties of the model are established, next, the equilibria and basic reproductive number $\mathfrak{R}_0$ are computed. The results of the numerical analysis corroborate the theoretical results presented previously. Time series plots with respect to treatment suggest that early intervention increases the number of HIV-managed individuals. This model has been designed using saturated treatment. The existence of delayed treatment due to limited health resources has had a significant impact on the spread of HIV/AIDS. The problem in question becomes complex due to the geographical range that hinders the mobilization process in health services. Thus, adequate allocation of health resources to address the spread of HIV/AIDS is highly desirable for maximum antiretroviral treatment for infected individuals.

Author Contributions. **Beay, L. K.**: Conceptualization, Methodology, Writing–Review & Editing, Supervision, Project Administration. **Ilwaru, V. Y. I.**: Supervision, Methodology, Validation, Resources. **Ansori, M. F.**: Conceptualization, Methodology, Validation, Resources. **Bakarbessy, L.**: Supervision, Methodology, Validation, Resources. **Dahoklory, M.**: Supervision, Methodology, Validation, Resources. **Saija, M.**: Conceptualization, Validation, Writing–Review & Editing, Project Administration.

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Data availability. All the data presented are hypothetical and cited from prior publications.

References

[1] X. Zhai *et al.*, "Dynamics of an HIV/AIDS transmission model with protection awareness and fluctuations," *Chaos, Solitons & Fractals*, vol. 169, p. 113224, 2023. DOI:10.1016/j.chaos.2023.113224

[2] C. Elendu *et al.*, "Shaping sustainable paths for HIV/AIDS funding: a review and reminder," *Annals of Medicine & Surgery*, vol. 87, no. 3, pp. 1415–1445, 2025. DOI:10.1097/MS9.0000000000002976

[3] M. Farman *et al.*, "Numerical and quantitative analysis of HIV/AIDS model with modified Atangana-Baleanu in Caputo sense derivative," *Alexandria Engineering Journal*, vol. 66, pp. 31–42, 2023. DOI:10.1016/j.aej.2022.11.034

[4] E. U. Alum *et al.*, "Toward a cure – Advancing HIV/AIDs treatment modalities beyond antiretroviral therapy: A Review," *Medicine*, vol. 103, no. 27, p. e38768, 2024. DOI:10.1097/MD.0000000000038768

[5] N. Anggriani *et al.*, "A mathematical model for a disease outbreak considering waning-immunity class with nonlinear incidence and recovery rates," *Journal of Biosafety and Biosecurity*, vol. 6, no. 3, pp. 170–180, 2024. DOI:10.1016/j.jobb.2024.05.005

[6] M. Z. Ndii *et al.*, "Estimating the time reproduction number in Kupang City Indonesia, 2016–2020, and assessing the effects of vaccination and different wolbachia strains on dengue transmission dynamics," *Mathematics*, vol. 10, no. 12, p. 2075, 2022. DOI:10.3390/math10122075

[7] S. T. Tresna, N. Anggriani, and A. K. Supriatna, "Study of mathematical modeling for plant disease transmission: a systematic literature review during 2012–2022," *Jambura Journal of Biomathematics (JJBM)*, vol. 4, no. 1, pp. 1–14 2023. DOI:10.34312/jjbm.v4i1.18443

[8] H. S. Panigoro and E. Rahmi, "Global stability of a fractional-order logistic growth model with infectious disease," *Jambura Journal of Biomathematics (JJBM)*, vol. 1, no. 2, pp. 49–56, 2020. DOI:10.34312/jjbm.v1i2.8135

[9] R. N. Apdo and R. N. Paluga, "Qualitative analysis of a mathematical model of COVID–19 with intervention strategies in the Philippines," *Jambura Journal of Biomathematics (JJBM)*, vol. 4, no. 1, pp. 46–54, 2023. DOI:10.34312/jjbm.v4i1.18990

[10] L. K. Beay *et al.*, " Modeling of abstinence behavior on the electoral lists with awareness campaigns and argumentative schemes," *Communication in Biomathematical Sciences*, vol. 7, no. 2, pp. 219–231, 2024. DOI:10.5614/cbms.2024.7.2.4

[11] L. K. Beay and M. Saija, "Dynamics of a stage–structure Rosenzweig–MacArthur model with linear harvesting in prey and cannibalism in predator," *Jambura Journal of Biomathematics (JJBM)*, vol. 2, no. 1, pp. 42–50, 2021. DOI:10.34312/jjbm.v2i1.10470

[12] L. K. Beay, "Modelling the effects of treatment and quarantine on measles," *AIP Conference Proceedings*, vol. 1937, no. 1, p. 020004, 2018. DOI:10.1063/1.5026076

[13] M. Marsudi *et al.*, "Global stability and optimal control of an HIV/AIDS epidemic model with behavioral change and treatment," *Engineering Letters*,

vol. 29, no. 2, pp. 575–591, 2021.
[14] T. K. Ayele, E. F. D. Goufo, and S. Mugisha, "Mathematical modeling of HIV/AIDS with optimal control: A case study in Ethiopia," *Results in Physics*, vol. 26, p. 104263, 2021. DOI:10.1016/j.rinp.2021.104263
[15] R. Wattanasirikosone and C. Modnak, "Analysing transmission dynamics of HIV/AIDS with optimal control strategy and its controlled state," *Journal of Biological Dynamics*, vol. 16, no. 1, pp. 499–527, 2022. DOI:10.1080/17513758.2022.2096934
[16] O. O. Apenteng *et al.*, "Analysing the impact of migration on HIV/AIDS cases using epidemiological modelling to guide policy makers," *Infectious Disease Modelling*, vol. 7, no. 1, pp. 252–261, 2022. DOI:10.1016/j.idm.2022.01.002
[17] C. C. Espitia *et al.*, "Mathematical model of HIV/AIDS considering sexual preferences under antiretroviral therapy, a case study in San Juan de Pasto, Colombia," *Journal of Computational Biology*, vol. 29, no. 5, pp. 483–493, 2022. DOI:10.1089/cmb.2021.0323
[18] S. W. Teklu and K. P. Rao, "HIV/AIDS-pneumonia codynamics model analysis with vaccination and treatment," *Computational and Mathematical Methods in Medicine*, vol. 2022, pp. 1–20, 2022. DOI:10.1155/2022/3105734
[19] Z. Chazuka, E. Mudimu, and D. Mathebula, "Stability and bifurcation analysis of an HIV model with pre–exposure prophylaxis and treatment interventions," *Scientific African*, vol. 23, p. e01979, 2024. DOI:10.1016/j.sciaf.2023.e01979
[20] S. Kar, D. K. Maiti, and A. P. Maiti, "HIV/AIDS control owing to local and global awareness, diagnosis, treatment tactics and control theory: fractional order modeling ," *Nonlinear Dynamics*, vol. 112, pp. 22391–22420, 2024. DOI:10.1007/s11071-024-10193-x
[21] H. J. Hamad, S. H. A. Khoshnaw, and M. Shahzad, " Model analysis for an HIV infectious disease using elasticity and sensitivity techniques," *AIMS Bioengineering*, vol. 11, no. 3, pp. 281–300, 2024. DOI:10.3934/bioeng.2024015
[22] S. W. Teklu *et al.*, "Analyses of an age structure HIV/AIDS compartmental model with optimal control theory," *Scientific Reports*, vol. 15, p. 5491, 2025. DOI:10.1038/s41598-024-82467-8
[23] Q. Dong, Y. Wang, and D. Jiang, "Dynamic analysis of an HIV model with CTL immune response and logarithmic Ornstein–Uhlenbeck process," *Chaos, Solitons & Fractals*, vol. 191, p. 115789, 2025. DOI:10.1016/j.chaos.2024.115789
[24] L. Huang *et al.*, " Transmission dynamics and stability analysis of fractional HIV and AIDS epidemic model with antiretroviral therapy ," *Scientific Reports*, vol. 15, p. 18212, 2025. DOI:10.1038/s41598-025-01355-x
[25] A. Abidemi *et al.*, " Insights into HIV/AIDS transmission dynamics and control in Indonesia–a mathematical modelling study ," *Partial Differential Equations in Applied Mathematics*, vol. 14, p. 101185, 2025. DOI:10.1016/j.padiff.2025.101185
[26] X. Zhou and J. Cui, "Analysis of stability and bifurcation for an SEIR epidemic model with saturated recovery rate," *Communications in Nonlinear Science and Numerical Simulation*, vol. 16, no. 11, pp. 4438–4450, 2011. DOI:10.1016/j.cnsns.2011.03.026
[27] A. Yadav and P. K. Srivastava, " The impact of information and saturated treatment with time delay in an infectious disease model," *Journal of Applied Mathematics and Computing*, vol. 66, pp. 277–305, 2021. DOI:10.1007/s12190-020-01436-2
[28] S. Jana, S. K. Nandi, and T. K. Kar, " Complex dynamics of an SIR epidemic model with saturated incidence rate and treatment," *Acta Biotheoretica*, vol. 64, pp. 65–84, 2016. DOI:10.1007/s10441-015-9273-9
[29] Y. Hu, H. Wang, and S. Jiang, "Analysis and optimal control of a two–strain SEIR epidemic model with saturated treatment rate," *Mathematics*, vol. 12, no. 19, p. 3026, 2024. DOI:10.3390/math12193026
[30] M. D. Abate *et al.*, "Incidence, progression and predictors of chronic kidney disease among adult HIV/AIDS patients on antiretroviral treatment in comprehensive specialised hospitals in the Amhara Region, Ethiopia, 2022: a multi–centre retrospective follow–up study," *BMJ Open*, vol. 15, no. 7, p. e090345, 2025. DOI:10.1136/bmjopen-2024-090345
[31] A. Malakapalli, "The structural challenges of geographic barriers to healthcare ," *Healers and Patients in North Carolina*, 2024.
[32] B. Hofmann, "Ethical issues with geographical variations in the provision of health care services ," *BMC Medical Ethics*, vol. 23, p. 127, 2022. DOI:10.1186/s12910-022-00869-7
[33] M. Khan *et al.*, " A novel fractal fractional mathematical model for HIV/ AIDS transmission stability and sensitivity with numerical analysis," *Scientific Reports*, vol. 15, p. 9291, 2025. DOI:10.1038/s41598-025-93436-0
[34] R. Naresh, A. Tripathi, and D. Sharma, " A nonlinear HIV/AIDS model with contact tracing," *Applied Mathematics and Computation*, vol. 217, no. 23, pp. 9575–9591, 2011. DOI:10.1016/j.amc.2011.04.033
[35] P. van den Driessche, " Reproduction numbers of infectious disease models ," *Infectious Disease Modelling*, vol. 2, no. 3, pp. 288–303, 2017. DOI:10.1016/j.idm.2017.06.002