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Mathematical Modeling of HIV Investigating the Effect of Inconsistent Treatment with Saturated Incidence Function

O. A. Odebiyi[1*], Salahu W. O[2], J. K. Oladejo[3], O. O Olabisi[4]
Ladoke Akintola University of Technology, Ogbomoso, Nigeria
oaodebiyi@lautech.edu.ng; jkoladejo@lautech.edu.ng

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Abstract

The Human Immunodeficiency Virus (HIV) remains a significant global health challenge, with millions of people worldwide living with the virus. Despite advances in treatment and prevention, the disease continues to spread, underscoring the need for a deeper understanding of its transmission dynamics. This study presents a mathematical model of HIV transmission dynamics, incorporating a saturation term to capture the complex interactions among susceptible, infected, AIDS, and treated populations. The validity of the solution confirms that the model is well-defined and holds epidemiological significance. The basic reproduction number is obtained using the next-generation matrix approach. To assess the stability of the model, we conducted a thorough analysis of the local and global stability of both the disease-free and endemic equilibria. This analysis provides a comprehensive understanding of the model's behavior, illuminating the conditions necessary for the disease to persist or die out. A sensitivity analysis is conducted to identify key parameters influencing the model's behavior. Numerical simulations are then performed to further explore the dynamics of the system. Our results highlight the importance of targeted interventions to control the spread of the disease, thereby informing public health policy and intervention strategies.

Keywords: HIV, Mathematical Modeling, Inconsistent Treatment, Saturated Incidence

Introduction

The Human Immunodeficiency Virus (HIV) is a viral infection that attacks the body's immune system, specifically the CD4 cells (T cells), which help the immune system fight off infections. If left untreated, HIV can lead to Acquired Immunodeficiency Syndrome (AIDS), a condition where the immune system is severely damaged, making it difficult to fight off opportunistic infections and certain types of cancer (Mbabazi, 2016; Odebiyi & Oladejo, 2024).

Globally, the HIV/AIDS situation is dire, with approximately 39.9 million people living with HIV as of 2023. That year saw 1.3 million new infections and 630,000 AID-related death. Since the epidemic's start, 42.3 million lives have been lost to HIV/AIDS-related causes. The impact is disproportionately felt in Africa, where 65% of global HIV cases are found, putting a strain on health care systems, depleting the workforce, and increasing orphan numbers. The region's statistics are alarming, highlighting the need for sustained efforts to combat the spread of the disease [2]. In fact, worldwide, it is estimated that between 250,000 and 350,000 deaths were averted in 2005 as a result of increased treatment access (UNAIDS, 2005).

Research by Stoddart and Reyes (2006) indicates that HIV infection progresses through 2-6 stages before AIDS can be diagnosed based on clinical symptoms, with viral load, or CD4+ Tcell count. The Health of an HIV-positive individual depends on several key factors, including the health of their immune system, CD4+ Tcell count, viral load in the blood and exposure to diseases in their environment.

To address this public health issue, it's crucial to focus on prevention, diagnosis, screening, vaccination and treatment. This includes increasing access to antiretroviral therapy, promoting awareness and implementing targeted interventions to reduce new infections and improve quality of life for those living with HIV/AIDS (Oladejo & Oluyo, 2020; Ibrahim et al., 2021; Stoddart & Reyes, 2026; Odebiyi et al.,2024).

Inconsistent treatment of HIV can lead to severe consequences, including the development of drug-resistant strains, progression to AIDS, increased risk of opportunistic infections

and transmission, reduced quality of life, and increased healthcare costs, often resulting from non-adherence to medication, lack of access to healthcare, stigma and discrimination, drug resistance and viral mutations, economic constraints, and complexity of treatment regimens (Sr. Mary et al., 2024; Olusola et al., 2025; Cox et al., 2022).

Infectious disease modeling involves using different infection rates to understand how disease spread. Researchers have explored various approaches, including saturated and monotone formulations, to improve the accuracy of these models. One such approach is the saturated incidence rate, which takes into account the impact of inhibition on disease transmission, and is often represented mathematically as a function of the infection rate, susceptible population, infected population and inhibition coefficient.

The function of this term $\frac{\alpha_1 SI}{1+\omega S}$ is to model the rate at which new infections occur. The saturation effect, represented by the term $(1+\omega S)$, reduces or rather slow down the rate of new infections as the susceptible population increases.

Epidemiologically, saturation incidence marks a pivotal point in an epidemic's trajectory, where disease prevalence stabilizes. By examining these formulations, researchers can gain valuable insights into the spread of epidemics. This study aims to investigate the impact of inconsistent treatment on disease transmission, employing a saturation incidence function to capture the epidemic's dynamics. Specifically, the objective is to examine how changes in parameters such as infection rate, treatment adherence, saturation and others affect the behavior of the model.

This study is organized as follows: Section 2 explains the formulation of model, including the model's positivity and boundedness. Section 3 presents an in-depth mathematical examination of the model, encompassing key aspects such as the basic reproduction, stability analysis of the disease-free equilibrium, global asymptotic stability of both disease-free and endemic equilibria, and a sensitivity analysis of the basic reproduction number. Section 4 focuses on numerical simulations and discussions while Section 5 summarizes the findings.

Model Formulation

In this study, the susceptible and infectious epidemic model (SI) is presented. A population size of $N(t)$ was partitioned into 4 subclasses of individuals which are; susceptible population, Infected population, AIDS population, and treated population with sizes denoted by $S(t), I(t), A(t),$ and $T(t)$, respectively such that $N = S + I + A + T,$ as shown in figure 1 below:

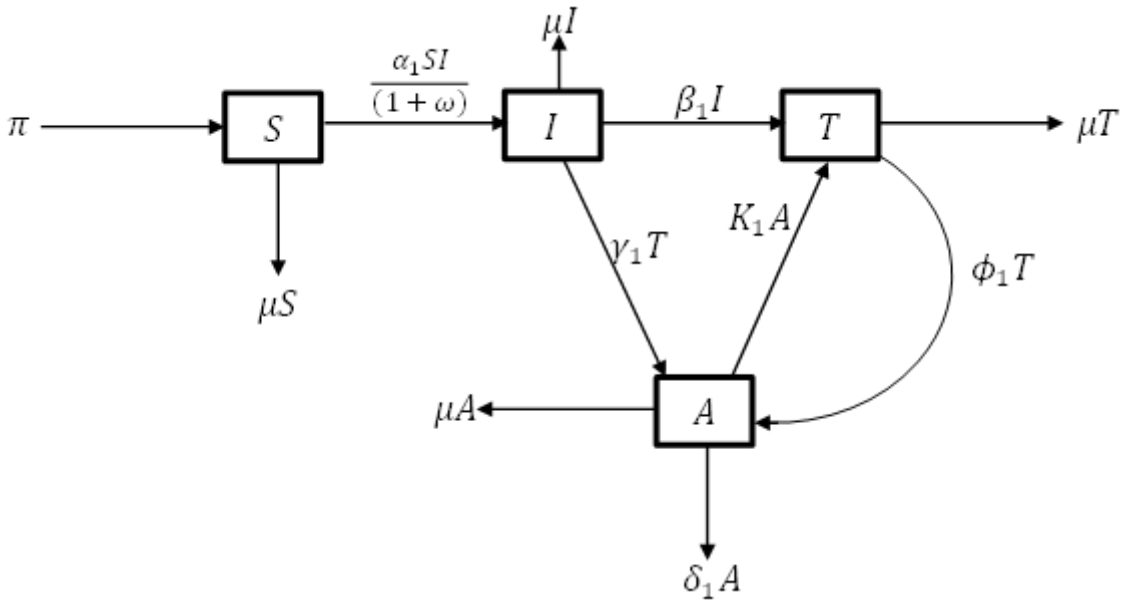

Figure 1: Model Flow chart for HIV spread dynamics

The model explores the dynamics of HIV transmission, incorporating a saturation term. The model is governed by a system of nonlinear differential equations, which capture the complex interactions between the compartments. The model consisting of four compartments: Susceptible, Infected, AIDS, and treated, with the susceptible population entering through recruitment by immigration at a constant rate (π). Newly infected individuals become infected at a rate (α_1) and progressed to the infected compartment. Infected individuals later progress to the AIDS compartment at a rate (ϕ_1) if left untreated. Infected individuals receive ART treatment at (β_1), while those in the AIDS compartment receive treatment at a rate of (k). AIDS-related mortality removes individuals from the AIDS compartment at a rate (σ_1). Natural death occurs in all compartment.

Considering all the above assumptions made, the following system of ordinary differential equations of the proposed model is therefore formulated:

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - \frac{\alpha_1 SI}{1+\omega S} - \mu S \\
\frac{dI}{dt} &= \frac{\alpha_1 SI}{1+\omega S} - (\mu + \beta_1 + \gamma_1) I \\
\frac{dA}{dt} &= \gamma_1 I + \phi_1 T - (k_1 + \delta_1 + \mu) A \\
\frac{dT}{dt} &= \beta_1 I + K_1 A - (\mu + \phi_1) T
\end{aligned}\right\} \quad (1)$$

$$S(0) = S_0, I(0) = I_0, A(0) = A_0, T(0) = T_0 \quad (2)$$

where, $\frac{\beta I_1 S}{1+\omega S}$ is the incidence rate and ω is the saturation term to the susceptible.

Table 1: Model variables and parameters are defined as follows:

Parameters/ variables	**Description**
π	Recruitment rate
β_1	Rate of treatment after infection
γ_1	Progression rate from infected to AIDS
α_1	Infection rate
μ	Natural mortality rate unrelated to AIDS
δ_1	Death rate due to AIDS
ϕ_1	Progression rate from treated to AIDS
k_1	Progression rate from AIDS to treated
$S(t)$	Susceptible population at a given time (t).
$I(t)$	Infected population
$T(t)$	Treated population
$A(t)$	AIDS population

Positivity and boundedness of the model: In this section we shall show from model (1) that the state variables are non-negative and the solutions remain positive for all $t \geq 0$. Hence, the parameters in the model are assumed to be positive.

Theorem 1: Let the initial conditions or values of the state variables be such that $\{(S(0) \geq 0, I(0) \geq 0, A(0) \geq 0, T(0) \geq 0,) \in \Omega\}$, then the set $(S(t), I(t), A(t), T(t))$ is non-negative in Ω for all $t \geq 0$.

Proof: Considering the first equation in (1), are considered for the positivity of the state variables as follows, following the approach of (Oladejo & Oluyo, 2020 ; Olusola et al., 2025; Adeyemi & Oluyo, 2025).

$$\frac{dS}{dt} \geq -\left(\frac{\beta I_1}{1+\alpha S} + \mu\right)S$$

$$\frac{dS}{dt} \geq -\int\left(\frac{\beta I_1}{1+\alpha S} + \mu\right)S$$

Using variable separable

$$\frac{dS}{S} \geq -\int\left(\frac{\beta I_1}{1+\alpha S} + \mu\right)dt$$

$$\ln s \geq -\left(\frac{\beta I_1}{1+\alpha S} + \mu\right)t + C$$

$$S(t) \geq e^{-\left(\frac{\beta I_1}{1+\alpha S} + \mu\right)t}.e^{C_1}$$

$$S(t) = S_0 e^{-\left(\frac{\beta I_1}{1+\alpha S} + \mu\right)t}.$$

$$S(0) = S_0 \Rightarrow A_1 = S_0$$

Since $S(t) \geq 0$, for all $t > 0$ provided that $S_0 \geq 0$.

Hence, $S(t) \geq 0$

For the second compartment of (1)

$$\frac{dI}{dt} \geq \frac{\alpha_1 SI}{1+\omega S} - (\mu + \beta_1 + \gamma_1)I$$

$$\frac{dI}{dt} \geq -\int(\mu + \beta_1 + \gamma_1)I$$

Using variable separable

$$\frac{dI}{I} \geq -\int(\mu + \beta_1 + \gamma_1)dt$$

$$I(t) \geq e^{-(\mu+\beta_1+\gamma_1)t}.e^{C_1}$$

$$I(t) = I_0 e^{-(\mu+\beta_1+\gamma_1)t}.$$

$$I(0) = I_0 \Rightarrow A_1 = I_0$$

Since $I(t) \geq 0$, for all $t > 0$ provided that $I_0 \geq 0$.

Hence, $I(t) \geq 0$

It is possible to show using the same procedure for other state variables that:

$$I(t) \geq I(0)e^{-(\mu+\beta_1+\gamma_1))t} \geq 0, A(t) \geq A(0)e^{-(k_1+\sigma_1+\mu)t} \geq 0,$$
$$T(t) \geq T(0)e^{-(\mu+\phi_1)t} \geq 0,$$

This shows that all the solutions of equation (1) are positive for all $t \geq 0$. Therefore, the HIV/AIDS transmission model stated in (1) is both epidemiologically significant and numerically well posed in an attainable given region $\Omega \geq 0$

Theorem 2: Every solution in the region $\Omega = \left\{ \left(S(t), I(t), A(t), T(t) \in \Omega_+^4 : N(t) \leq \frac{\pi}{\mu} \right) \right\}$ is positively invariant with respect to the HIV/AIDS model (1) in the populations. The solutions for the system are contained and remain in the region Ω for all time $t \geq 0$.

Proof: Considering the equation of the model, and adding up all the derivatives with respect to time t, we obtained

$$\frac{dN(t)}{dt} = \pi + \mu(S + I + A + T) - \sigma_1 A$$

$$\frac{dN(t)}{dt} = \pi + \mu N$$

$$N \leq \frac{\pi}{\mu} + \left(N_0 - \frac{\pi}{\mu} \right) e^{-\mu t}$$

Where N_0 is the initial size of the population

Therefore,

$$\lim_{t \to \infty} N(t) \leq \frac{\pi}{\mu}$$

This result implies that HIV/AIDS model (1) has non-zero negative and bounded solution in the region Ω and all the solutions starting in Ω approach, enter or stay in Ω. Hence, it is sufficient to conclude that the model is epidemiologically well posed.

Mathematical Analysis of the Model

Disease free equilibrium point*:* This is a state where the disease is completely eliminated from the population, that is, there is no infected individuals, no disease transmission and population is entirely susceptible.

At the equilibrium,

$$\frac{dS}{dt}=\frac{dV}{dt}=\frac{dI_1}{dt}=\frac{dI_2}{dt}=\frac{dT}{dt}=\frac{dA}{dt}=0$$

$$\left.\begin{aligned}
\frac{dS}{dt}&=\pi-\frac{\alpha_1 SI}{1+\omega S}-\mu S\\
\frac{dI}{dt}&=\frac{\alpha_1 SI}{1+\omega S}-(\mu+\beta_1+\gamma_1)I\\
\frac{dA}{dt}&=\gamma_1 I+\phi_1 T-(k_1+\delta_1+\mu)A\\
\frac{dT}{dt}&=\beta_1 I+K_1 A-(\mu+\phi_1)T
\end{aligned}\right\} \qquad (3)$$

At the disease free equilibrium, we set $S\neq 0, I=A=T=0$ substituting these to equation (3) and solving gives the infection – free equilibrium as

$$E^0=\left(S^0,I^0,A^0,T^0\right)=\left(\frac{\pi}{\mu},0,0,0\right) \qquad (4)$$

Endemic equilibrium point*:* The endemic equilibrium is a steady- state solution where the disease persists in the population at a stable level. At endemic equilibrium, $S\neq 0, I\neq 0, A\neq 0, T\neq 0,$

Solving equation (3) simultaneously, we have the endemic equilibrium points of the HIV model (3) designated by $x^*=\left(S^*,I^*,A^*,T^*\right)$ is obtained as:

$$\left.\begin{aligned} s^* &= \frac{\pi}{\left(\lambda^* + \mu\right)} \\ I^* &= \frac{\lambda^* \pi}{Z_1\left(\lambda^* + \mu\right)} \\ T^* &= \frac{\beta_1 \lambda^* \pi Z_2 + k_1 \gamma_1 \lambda^* \pi}{Z_1 Z_2 Z_3 - \phi_1 Z_2\left(\lambda^* + \mu\right)} \\ A^* &= \frac{\gamma_1 \lambda^* \pi\left(Z_2 Z_3 - k_1 \phi_1\right) + \phi_1 \beta_1 \lambda^* \pi Z_1 Z_2 + \phi_1 k_1 \gamma_1 \lambda^* \pi Z_1}{Z_1 Z_2\left(\lambda^* + \mu\right)\left(Z_1 Z_2 - k_1 \phi_1\right)} \end{aligned}\right\} \quad (5)$$

Where,

$$Z_1 = \left(\mu + \beta_1 + \gamma_1\right), Z_2 = \left(k_1 + \delta_1 + \mu\right), Z_3 = \left(\mu + \phi_1\right), \quad \lambda^* = \frac{\alpha_1 I}{1 + \omega S} \quad (6)$$

Basic Reproduction Number (R_0)

The next generation matrix is constructed by examining novel infection routes and inter-compartmental transmission dynamics. Utilizing the methodology outlined by [10].

Let M be the next generation matrix. It comprises of two parts F and V^{-1} where

$$F = \left[\frac{\partial f_1(x_0)}{\partial x_j}\right], \qquad V = \left[\frac{\partial V_1(x_0)}{\partial x_j}\right]$$

The term f_i and v_i are computed as

$$f_i = \begin{bmatrix} \frac{\alpha_1 SI}{1 + \omega S} \\ 0 \\ 0 \end{bmatrix}, v_i = \begin{bmatrix} -\left(\mu + \beta_1 + \gamma_1\right) I \\ \gamma_1 I + \phi_1 T - \left(k_1 + \delta_1 + \mu\right) A \\ \beta_1 I + k_1 A - \left(\mu + \phi_1\right) T \end{bmatrix}$$

$$, \; Df(E_0) = \left[\frac{\partial f_1(E_0)}{\partial x_j}\right], \; DV = \left[\frac{\partial V_1}{\partial x_j}\right]$$

$$F_i = \begin{bmatrix} \frac{\alpha_1 S}{1 + \omega S} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, V_i = \begin{bmatrix} \left(\mu + \beta_1 + \gamma_1\right) & 0 & 0 \\ -\gamma_1 & \left(k_1 + \delta_1 + \mu\right) & -\phi_1 \\ -\beta_1 & -k_1 & \left(\mu + \phi_1\right) \end{bmatrix} \quad (7)$$

The Jacobian matrices of F_i and V_i at the disease free equilibrium point, $x_0 = \left(\frac{\pi}{\mu},0,0,0\right)$ are:

$$DF(x_0) = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right] = \begin{bmatrix} \frac{\alpha_1 S_0}{1+\omega S_0} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \Rightarrow \begin{bmatrix} \frac{\alpha_1 \pi}{\mu\left(1+\frac{\omega\pi}{\mu}\right)} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

$$V^{-1} = \begin{bmatrix} \frac{1}{(\mu+\beta_1+\gamma_1)} & 0 & 0 \\ \frac{\mu\gamma_1+\beta_1\phi_1}{(\mu+\beta_1+\gamma_1)(\mu^2+\mu k_1+\mu\delta_1-k_1\phi_1)} & \frac{\mu}{(\mu^2+\mu k_1+\mu\delta_1-k_1\phi_1)} & \frac{\phi_1}{(\mu^2+\mu k_1+\mu\delta_1-k_1\phi_1)} \\ \frac{\mu\beta_1+k_1\beta_1+\beta_1\delta_1+\gamma_1 k_1}{(\mu+\beta_1+\gamma_1)(\mu^2+\mu k_1+\mu\delta_1-k_1\phi_1)} & \frac{k_1}{(\mu^2+\mu k_1+\mu\delta_1-k_1\phi_1)} & \frac{k_1+\delta_1+\mu}{(\mu^2+\mu k_1+\mu\delta_1-k_1\phi_1)} \end{bmatrix}$$

(8)

$$FV^{-1} = \begin{bmatrix} \frac{\alpha_1\pi}{\mu\left(1+\frac{\omega\pi}{\mu}\right)(\mu+\beta_1+\gamma_1)} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

(9)

The basic reproduction number, which is the dominant Eigen-value of the product FV^{-1}, is therefore obtained as:

$$R_0 = \frac{\pi\alpha_1}{\pi\mu\omega + \pi\omega\beta_1 + \pi\omega\gamma_1 + \mu^2 + \mu\beta_1 + \mu\gamma_1}$$

(10)

Stability analysis of the Disease free equilibrium

Theorem 4*:* The disease-free state is locally asymptotically stable if the basic reproduction number $R_0 < 1$ and unstable if otherwise.

Proof:

We evaluate the Jacobian matrix of the model at the disease free equilibrium

$$x_0 = \left(\frac{\pi}{\mu}, 0, 0, 0\right)$$

$$J = \begin{bmatrix} -\mu & \frac{\alpha_1 S_0}{1+\omega S_0} & 0 & 0 \\ 0 & \frac{\alpha_1 S_0}{1+\omega S_0} - (\mu+\beta_1+\gamma_1) & 0 & 0 \\ 0 & \gamma_1 & -(k_1+\delta_1+\mu) & \phi_1 \\ 0 & \beta_1 & k_1 & -(\mu+\phi_1) \end{bmatrix} \tag{11}$$

$$J(E_0) \Rightarrow \begin{bmatrix} -\mu & \frac{\alpha_1 \pi}{\mu\left(1+\frac{\omega\pi}{\mu}\right)} & 0 & 0 \\ 0 & \frac{\alpha_1 \pi}{\mu\left(1+\frac{\omega\pi}{\mu}\right)} - (\mu+\beta_1+\gamma_1) & 0 & 0 \\ 0 & \gamma_1 & -(k_1+\delta_1+\mu) & \phi_1 \\ 0 & \beta_1 & k_1 & -(\mu+\phi_1) \end{bmatrix} \Rightarrow$$

$$\begin{bmatrix} -\mu & \frac{\alpha_1 \pi}{\mu+\omega\pi} & 0 & 0 \\ 0 & \frac{\alpha_1 \pi}{\mu+\omega\pi} - (\mu+\beta_1+\gamma_1) & 0 & 0 \\ 0 & \gamma_1 & -(k_1+\delta_1+\mu) & \phi_1 \\ 0 & \beta_1 & k_1 & -(\mu+\phi_1) \end{bmatrix} \tag{12}$$

$$|A - \lambda I| = 0 \Rightarrow$$

$$\begin{bmatrix} -\mu & \frac{\alpha_1 \pi}{\mu+\omega\pi} & 0 & 0 \\ 0 & \frac{\alpha_1 \pi}{\mu+\omega\pi} - (\mu+\beta_1+\gamma_1) & 0 & 0 \\ 0 & \gamma_1 & -(k_1+\delta_1+\mu) & \phi_1 \\ 0 & \beta_1 & k_1 & -(\mu+\phi_1) \end{bmatrix} - \begin{bmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{bmatrix} = 0$$

$$\Rightarrow \begin{vmatrix} -\mu-\lambda & \dfrac{\alpha_1\pi}{\mu+\omega\pi} & 0 & 0 \\ 0 & \left(\dfrac{\alpha_1\pi}{\mu+\omega\pi}-(\mu+\beta_1+\gamma_1)\right)-\lambda & 0 & 0 \\ 0 & \gamma_1 & -(k_1+\delta_1+\mu)-\lambda & \phi_1 \\ 0 & \beta_1 & k_1 & -(\mu+\phi_1)-\lambda \end{vmatrix} = 0 \qquad (13)$$

The eigenvalues obtained are:

$$\begin{vmatrix} -\mu \\ -\mu-\frac{1}{2}\delta_1-\frac{1}{2}k_1-\frac{1}{2}\phi_1+\frac{1}{2}\sqrt{4\phi_1k_1+\delta_1^2+2\delta_1k_1-2\delta_1\phi_1+k_1^2-2k_1\phi_1+\phi_1^2} \\ -\mu-\frac{1}{2}\delta_1-\frac{1}{2}k_1-\frac{1}{2}\phi_1-\frac{1}{2}\sqrt{4\phi_1k_1+\delta_1^2+2\delta_1k_1-2\delta_1\phi_1+k_1^2-2k_1\phi_1+\phi_1^2} \\ -\dfrac{\pi\mu\omega+\pi\omega\beta_1+\pi\omega\gamma_1-\pi\alpha_1+\mu^2+\mu\beta_1+\mu\gamma_1}{\pi\omega+\mu} \end{vmatrix} \qquad (14)$$

All the eigenvalues in equation (14) are negative, except the second one. Now the condition for stability of the the disease free equilibrium is that all the eigenvalues must be negative.

This simply implies that:

$$-\mu-\frac{1}{2}\delta_1-\frac{1}{2}k_1-\frac{1}{2}\phi_1+\frac{1}{2}\sqrt{k_1^2+\left(4\phi_1^2+2\delta_1-2\phi_1\right)k_1+(\phi_1-\delta_1)^2}<0 \qquad (15)$$

The equation can further be simplified to satisfy the conditions for the local stability of DFE as:

$$\frac{1}{2}\sqrt{k_1^2+\left(4\phi_1^2+2\delta_1-2\phi_1\right)k_1+(\phi_1-\delta_1)^2}<\mu+\frac{1}{2}\delta_1+\frac{1}{2}k_1+\frac{1}{2}\phi_1$$

$$\sqrt{k_1^2+\left(4\phi_1^2+2\delta_1-2\phi_1\right)k_1+(\phi_1-\delta_1)^2}<2\mu+\delta_1+k_1+\phi_1 \qquad (16)$$

Hence the DFE is locally asymptotically stable since all the $\lambda's$ are negative.

Global Asymptotic Stability of the Disease free Equilibrium

Theorem 6: The disease free equilibrium point of the model is globally asymptotically stable whenever the basic reproduction number is less than unity.

Proof: Following the approach used by (Adeyemi & Oluyo, 2023), the global asymptotic stability of the HIV model (1) is investigated. Re-writing the HIV model (1) in a compact form as follows

$$\begin{aligned} \frac{dX}{dt} &= F(X,\ Z), \\ \frac{dZ}{dt} &= G(X,\ Z),\ G(X,\ 0) = 0, \end{aligned} \tag{17}$$

Where X is the uninfected class of the HIV model and Z is the infected classes of the model i.e. $X = S \in R_+^2$ and $Z = (I_1, I_2, T, A) \in R_+^4$. Also, let the disease-free equilibrium point of the HIV model be denoted by $\varepsilon_0 = (X^*, 0)$. Then, the following properties must be satisfied

H_1 : For $\frac{dX}{dt} = F(X, 0)$, X^* is globally asymptotically stable

H_2 : $G(X, Z) = AZ - \hat{G}(X,\ Z) \geq 0$,

Where $A = \partial G / \partial Z$, which is an M-matrix evaluated at $(X^*, 0)$ with non-negative off diagonal entries.

Theorem 7: The disease-free $\varepsilon_0 = (X^*, 0)$ of the HIV model (1) is globally asymptotically stable if the properties H_1 and H_2 are satisfied.

Proof: $F(X,\ Z)$ and $G(X,\ Z)$ are obtained from the HIV model (1) as

$$S(0) = S_0, V(0) = V_0, I_1(0) = I_{1_0}, I_2(0) = I_{2_0}, A(0) = A_0$$

$$F(X,\ Z) = \left(\pi - \frac{\alpha_1 SI}{1 + \omega S} \grave{} - \mu S \right) \tag{18}$$

$$G(X,\ Z) = \begin{bmatrix} \frac{\alpha_1 SI}{1 + \omega S} - (\mu + \beta_1 + \gamma_1) I \\ \gamma_1 I + \phi_1 T - (k_1 + \delta_1 + \mu) A \\ \beta_1 I + K_1 A - (\mu + \phi_1) T \end{bmatrix} \tag{19}$$

Such that,

$$F(X,\ 0) = (\pi - \mu S)$$

$$\frac{dX}{dt} = f(X,0)$$

$$\frac{dS}{dt} = \pi - (\mu + \alpha)S \qquad (20)$$

Simplifying Equation (17) gives

$$S(t) = \frac{\pi}{\mu} + \left(S(0) - \frac{\pi}{\mu} \right) \ell^{-(\mu+\alpha)t} \qquad (21)$$

Irrespective of the initial sizes of the variables as $t \to \infty$, then, $S(t) \to \frac{\pi}{\mu}$. Therefore, the DFE $(X^*, 0)$ is globally asymptotically stable satisfying property H_1.

Now, to establish the second property H_2, recall that

$$G(X, Z) = \begin{bmatrix} \frac{\alpha_1 SI}{1+\omega S} - (\mu + \beta_1 + \gamma_1)I \\ \gamma_1 I + \phi_1 T - (k_1 + \delta_1 + \mu)A \\ \beta_1 I + K_1 A - (\mu + \phi_1)T \end{bmatrix} \qquad (22)$$

An M-matrix whose off diagonal entries are non-negative is obtained as

$$A = \frac{\partial G}{\partial Z} = \begin{pmatrix} \frac{\alpha_1}{1+\omega S} S^* - (\mu + \beta_1 + \gamma_1) & 0 & 0 \\ \gamma_1 & -(k_1 + \delta_1 + \mu) & \phi_1 \\ \beta_1 & k_1 & (\mu + \phi_1) \end{pmatrix} \qquad (23)$$

Where $S^* = \frac{\pi}{\mu}$. Then, from

$$\hat{G}(X, Z) = AZ - G(X, Z),$$

$$\hat{G}(X, Z) = \begin{pmatrix} \frac{\alpha_1}{1+\omega S}(S^* - S) \\ 0 \\ 0 \\ 0 \end{pmatrix} \qquad (24)$$

Since $0 \le S \le \frac{\pi}{\mu}$, it is obvious that $\hat{G}_1(X,Y) \ge 0$, $\hat{G}_2(X,Y) = 0, \hat{G}_3(X,Y) = 0$, $\hat{G}_4(X,Y) = 0$

Hence, property H_2 is satisfied. Therefore, the disease-free equilibrium of the HIV model (1) is globally asymptotically stable.

Global asymptotic stability of endemic equilibrium

Theorem 5: The global asymptotic stability of the HIV model (1) around the endemic equilibrium point is globally asymptotically stable in the region D whenever the basic reproduction number is greater than one.

Proof: Consider a quadratic Lyapunov function $L: D \in R_+^4 \to R_+$ defined by

$$L = \frac{1}{2}\left\{\left(S - S^*\right) + \left(I - I^*\right) + \left(A - A^*\right) + \left(T - T^*\right)\right\}^2 \tag{25}$$

The time derivative of the Lyapunov function (11) is given by

$$\frac{dL}{dt} = \left\{\left(S - S^*\right) + \left(I - I^*\right) + \left(A - A^*\right) + \left(T - T^*\right)\right\}\frac{d}{dt}\left(S + I + A + T\right)$$

$$\frac{dL}{dt} = \left\{\left(S - S^*\right) + \left(I - I^*\right) + \left(A - A^*\right) + \left(T - T^*\right)\right\}\left\{\pi - \mu\left(S + I + A + T\right)\right\}$$

$$= -\mu\left\{\left(S - S^*\right) + \left(I - I^*\right) + \left(A - A^*\right) + \left(T - T^*\right)\right\}\left\{\left(S + I + A + T\right) - \frac{\pi}{\mu}\right\} \tag{26}$$

Since $N^* \le \frac{\pi}{\mu}$, then, the following result is obtained

$$\frac{dL}{dt} \le -\mu\left\{\left(S - S^*\right) + \left(I - I^*\right) + \left(A - A^*\right) + \left(T - T^*\right)\right\}\left\{\begin{matrix}\left(S + I + A + T\right) - \\ \left(S^* + I^* + T^*\right) \\ + A^*\end{matrix}\right\}$$

$$= -\mu\left\{\left(S - S^*\right) + \left(I - I^*\right) + \left(A - A^*\right) + \left(T - T^*\right)\right\} \times \left\{\left(S - S^*\right) + \left(I - I^*\right) + \left(A - A^*\right) + \left(T - T^*\right)\right\}$$

$$= -\mu\left\{\left(S - S^*\right) + \left(I - I^*\right) + \left(A - A^*\right) + \left(T - T^*\right)\right\}^2 \tag{27}$$

Since the time derivative of the continuously differentiable function G is negative semi-definite i.e., $\frac{dL}{dt} \le 0$, then, the function L is a Lyapunov function. Therefore, $\frac{dL}{dt} = 0$ provided that $S = S^*$, $I = I^*$, $A = A^*$, and, $T = T^*$. Then, by LaSalle, the largest invariance set

for which $\frac{dL}{dt} = 0$ is the singleton set $\{\varepsilon^*\}$, which implies that the endemic equilibrium point of the HIV/AIDS model with saturated incidence rate in (1) is globally asymptotically stable.

Sensitivity Analysis of the basic Reproduction number

Sensitivity analysis is crucial for evaluating the stability and accuracy of mathematical models. This process helps researchers pinpoint the most influential factors driving disease transmission and assess the potential impact of interventions. It investigates how changes in certain epidemiological features of the model affect the transmission dynamics of the HIV/AIDS model (1).

The normalized forward sensitivity index of a variable to a parameter is a ratio of the relative change in the variable to the relative change in the parameter. When a variable is differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivatives.

Adapting the methodology introduced by Chitnis et al., (2008), the normalized forward sensitivity index of a variable "k" that depends differentiable on a parameter "e" is defined as:

$$X_e^k := \frac{\partial k}{\partial e} * \frac{e}{k} \tag{28}$$

Following the approach used in (Olaniyi & Obaniyi, 2014; Habibah & Rois, 2024), the sensitivity indices of the parameters relative to the basic reproduction number (R_0), (10) is established. Therefore, the normalized forward sensitivity indices of the basic reproduction number (R_0) associated with parameters, ρ, is calculated as

$$X_\rho^{R_0} := \frac{\partial R_0}{\partial \rho} * \frac{\rho}{R_0} \tag{29}$$

As we have an explicit formula for R_0 in equation (10), and we derive an analytical expression for the sensitivity of R_0, as $X_\rho^{R_0} := \frac{\partial R_0}{\partial \rho} * \frac{\rho}{R_0}$ with respect to each of the parameters involved in R_0 as computed in table 2 below:

Table 2: Sensitivity Result

Parameter	Value	Sensitivity Index	Source
π	0.06	+0.178082	Ayele et al., (2021)
α_1	0.0009	+1.00000	Sarah et al., (2011)
μ	0.0013	-0.17938	Omondi & Luboobi, (2019)
γ_1	0.999925	-0.99869	Sr. Mary et al., (2024)
ω	0.1	-0.82192	Odebiyi & Oladejo, (2024)
β_1	0.000075	-0.000749	Omondi & Luboobi, (2019)
δ_1	0.00128		Omondi & Luboobi, (2019)

Interpretation of Sensitivity Indices*:* The sensitivity indices provide valuable insights into the relationships between model parameters and outcomes. A high sensitivity index for a particular parameter indicates that small changes to that parameter can significantly impact the model's behavior. Conversely, a low sensitivity index suggests that the model is relatively insensitive to changes in that parameter. Positive sensitivity indices reveal parameters that increase the outcome, while negative indices identify parameters that decrease the outcome. By analyzing the sensitivity indices, researchers can identify key parameters driving model behavior, prioritize parameters for further investigation and also targeted interventions to optimize outcomes.

The sensitivity indices of the basic reproduction number R_0, relative to its parameters are summarized in Table 2. It can be seen that there are parameters with positive and negative sensitivity indices. Parameters representing recruitment rate (π), and Infection rate (α_1), are parameters with positive indices while parameters such as $\beta_1, \gamma_1, \mu,$ and ω are parameters with negative sensitivity indices accordingly. It is worth noting that unhindered

increase in the values of parameters with positive sensitivity indices will lead to a corresponding increase in the value of the associated basic reproduction number.

Numerical Simulations and Discussion

A computational simulation of the model was conducted using Maple 18 Mathematical software to gain deeper insights into the dynamic transmission patterns of HIV/AIDS. This simulation facilitated an examination of how various parameters influence the basic reproduction number, providing a clearer understanding of their role in shaping the epidemic's trajectory.

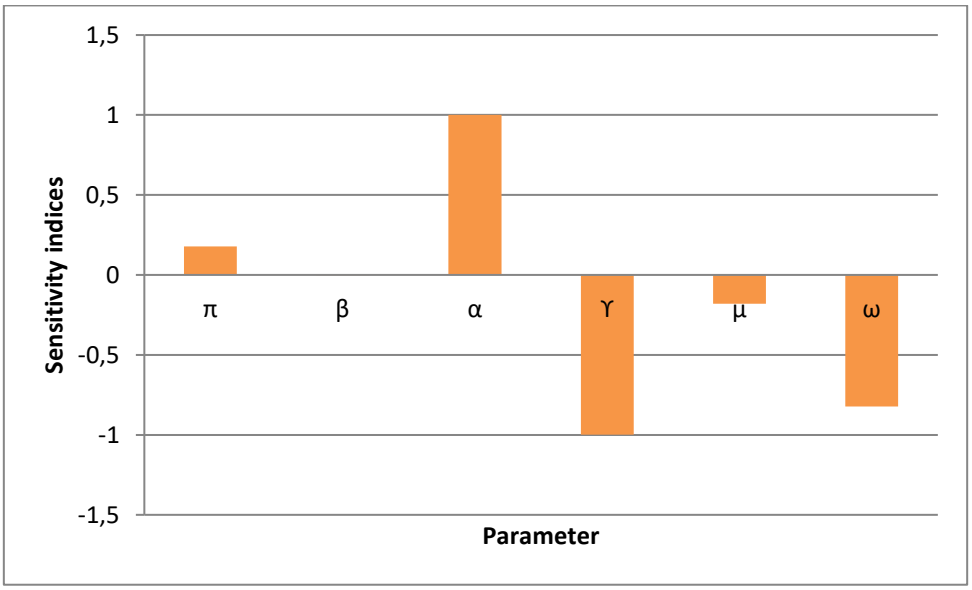

Fig 1: Graphical Representation of the Sensitivity indices of R_0

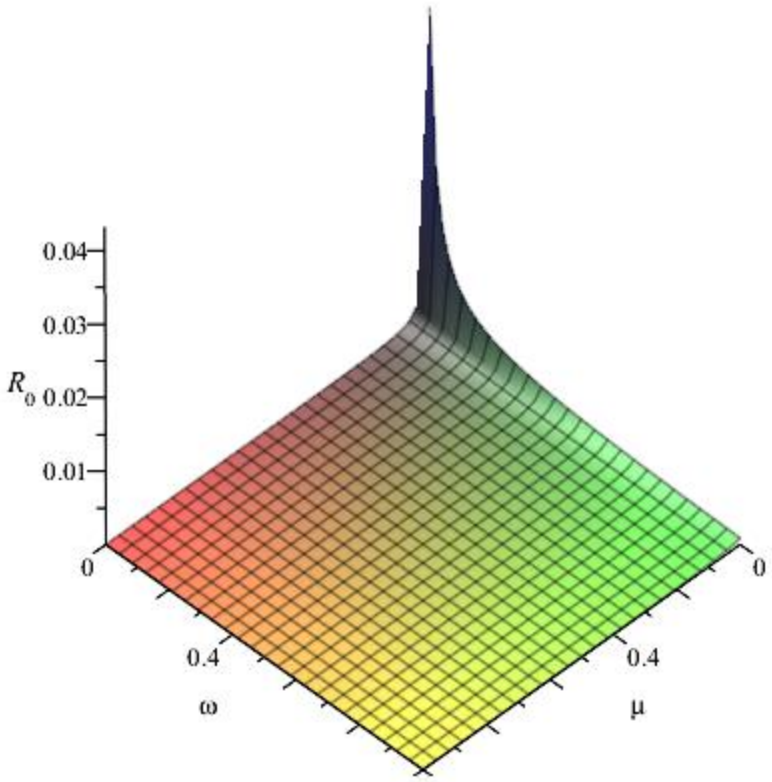

Fig. 2: Sensitivity of the basic reproduction number to the parameters π and ω

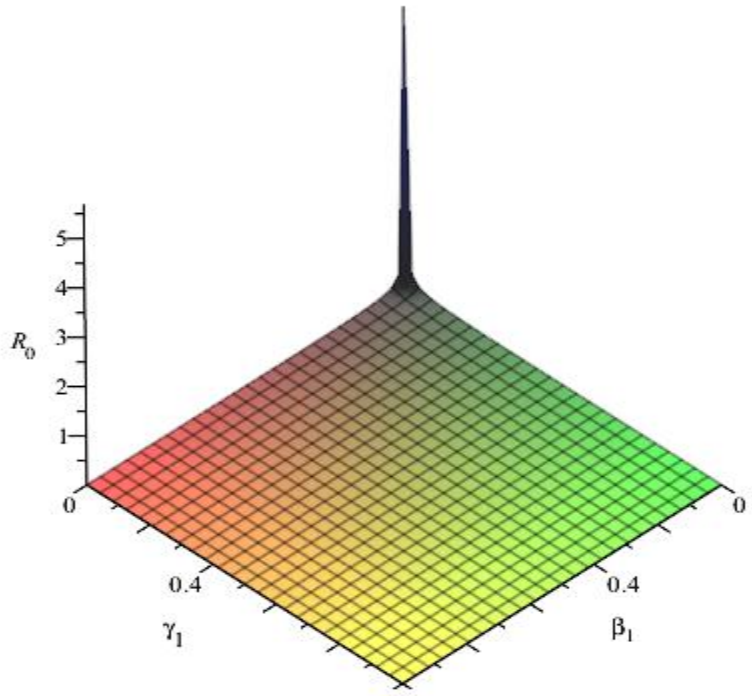

Fig. 3: Sensitivity of the basic reproduction number to the parameters γ_1 and β_1

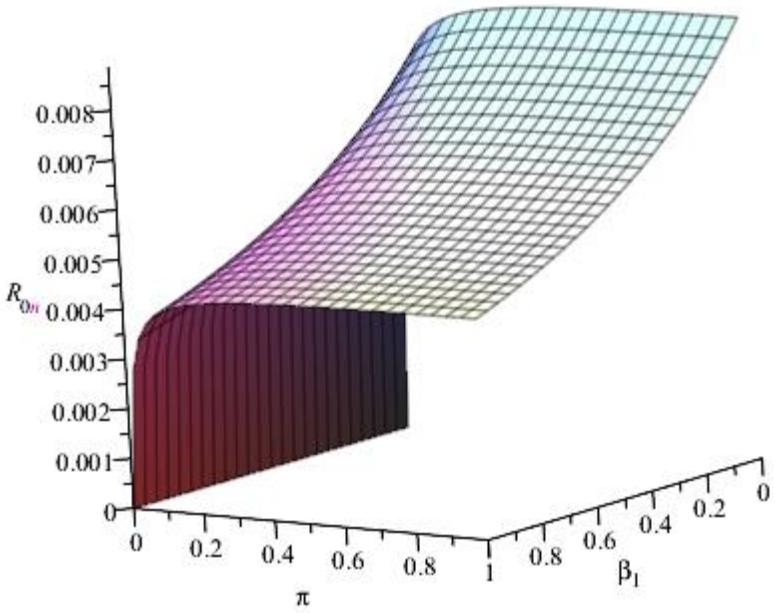

Fig. 4: Sensitivity of the basic reproduction number to the parameters π and θ

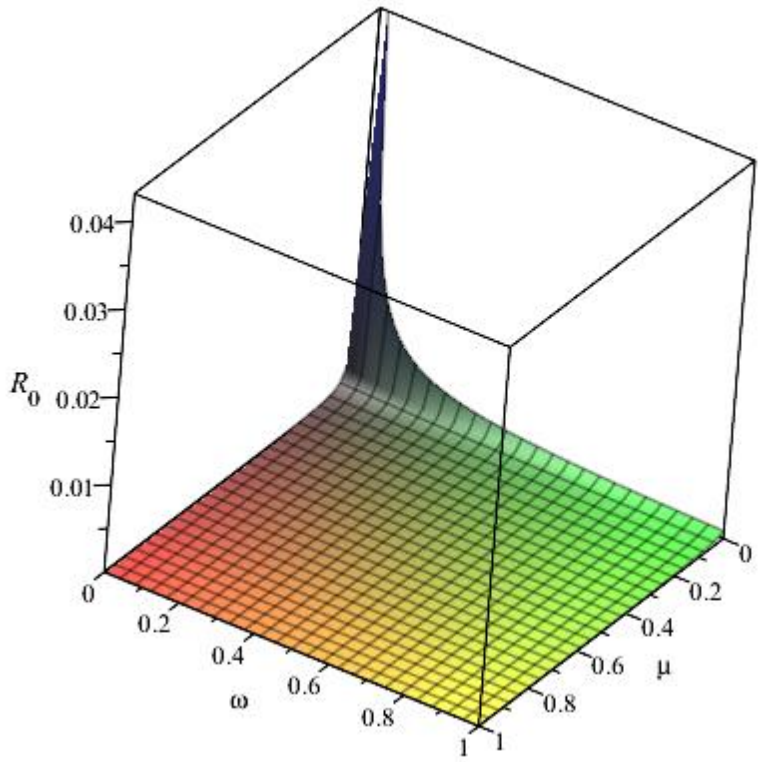

Fig. 5: Sensitivity of the basic reproduction number to the parameters ω and μ

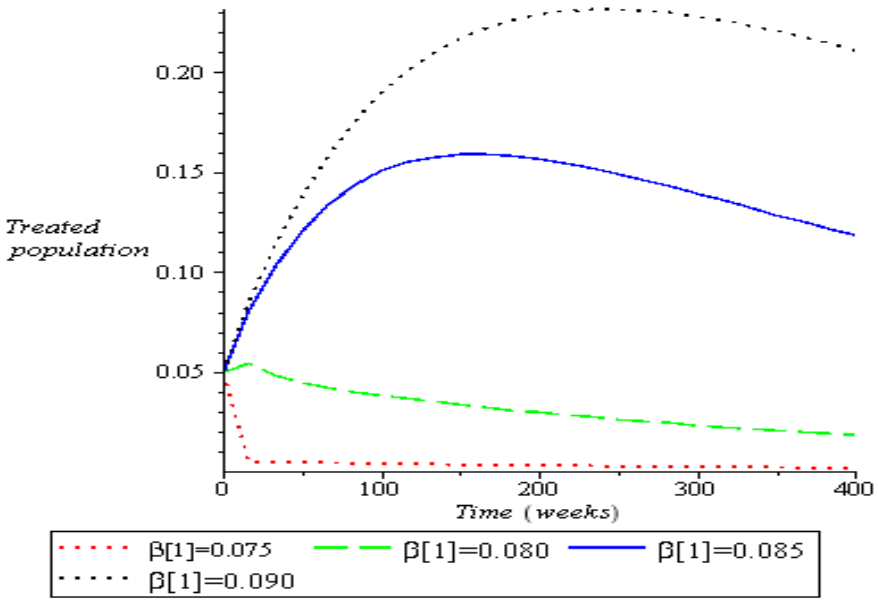

Fig 6: graph of treated infective population against time t, showcasing various rate of treatment after infection , (β),

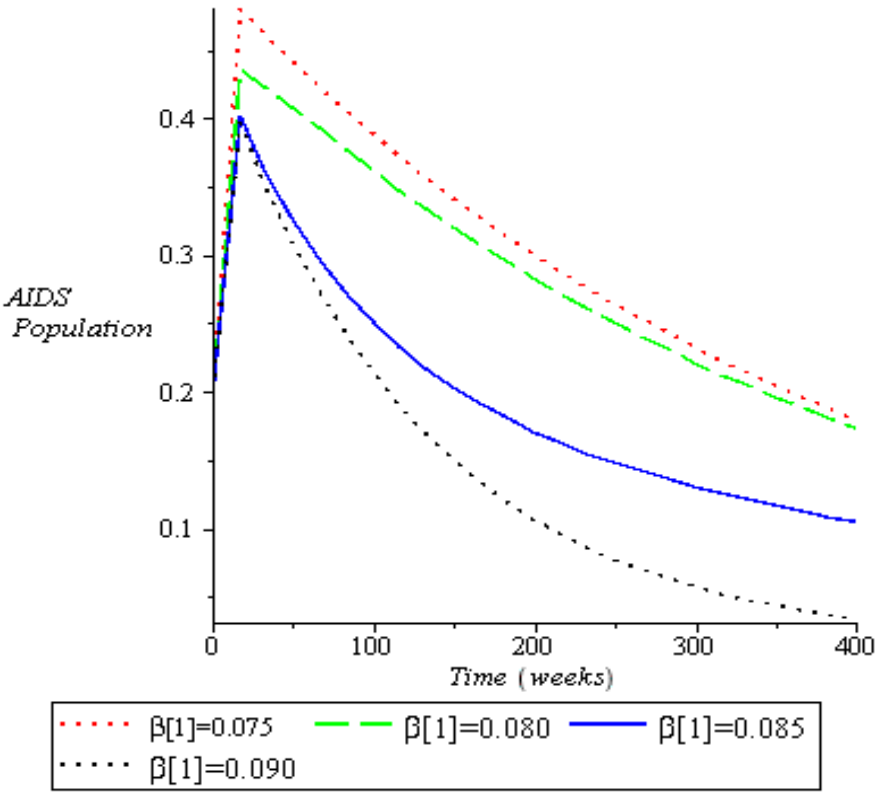

Fig 7: graph of treated infective population against time t, showcasing various rate of treatment after infection , (β),

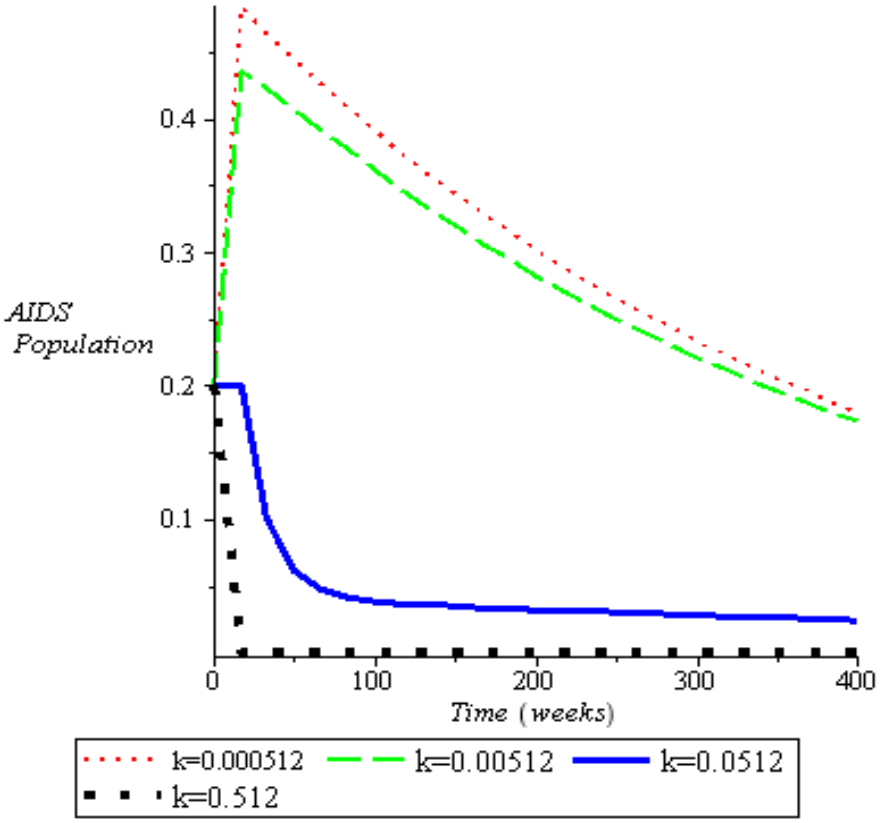

Fig 8: graph of treated infective population against time t, showcasing various rate of (k_1).

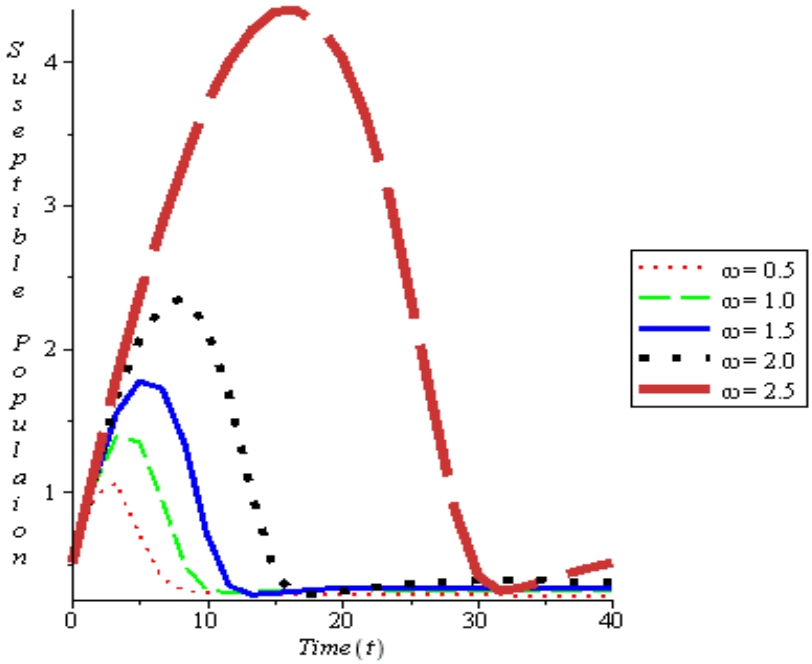

Fig 9: graph of Susceptible population against time t, showcasing various rates of saturation (ω).

Discussion

Fig 1 illustrates the graphical representation of the basic reproduction in form of a bar chart. The most sensitive parameter is the infection rate.

Fig 2 illustrates the relationship between recruitment rate of susceptible (π) and saturation term (ω) on the basic reproduction number, Notably, as the recruitment rate increase, the basic reproduction number increase. Conversely, increase in saturation rate, enhances a decrease in the basic reproduction number. Fig 3 illustrates the relationship between progression rate and the basic reproduction number. The simulation results demonstrates that as the progression rate from infected population to AIDS, (γ_1) increases, the basic reproduction decreases and also, an increase in β, leads to a decrease in the basic reproduction number. The decrease in R_0 indicates that the number of secondary infections generated by an infected individual is reduced, which can help control the outbreak. Fig 4 reveals the effect of recruitment rate (π) and treatment rate (β_1). As the recruitment rate increases, the basic reproduction number also increases, indicating a potential growth in the spread of the disease. In contrast, an increase in the rate of treatment after infection leads to a decrease in R_0, suggesting that treatment is effective in reducing the spread of the disease. Fig 5 also illustrates that an increase in saturation term and natural death rate unrelated to AIDS leads to an increase in the basic reproduction number.

Fig 6 and 7 shows the effect of treatment rates on infected and AIDS population. The model simulations show that as the rate at which infected individuals seek treatment increases, the treatment population grows. This suggests that improving access to treatment can lead to more people receiving care. Additionally, as the rate at which AIDS population receive treatment increases, the AIDS population decreases as seen in Fig 4.7. This indicates that effective treatment can help reduce the number of individuals with advanced HIV disease. This result is in perfect agreement to that of (Odebiyi & Oladejo, 2024; Sr. Mary et al., 2024; Olusola et al., 2025).

Fig 8 shows the effect of progression rate from treatment to AIDS. The simulations show that as the rate of progression from treatment to AIDS increases, the AIDS population decreases. This suggests that effective treatment is able to slow or reverse disease progression, leading to fewer individuals developing advanced HIV disease (AIDS). This

findings highlights the importance of timely and effective treatment in managing HIV and preventing disease progression. This result also aligns with the findings of Sr. Mary et al., 2024; Olusola et al., 2025).

Fig 9 shows the effect of saturation on the susceptible population. The graph reveals an increasing trend, indicating that as the saturation term increases, more individuals remain susceptible due to reduced transmission rates. This however can be attributed to various factors such as increased awareness, improved interventions, precautionary measures taken by susceptible population or behavior change which limit the spread of the disease. As a result, the susceptible population grows, reflecting the effectiveness of these control measures. This findings agrees with that of Olaniyi (2018).

Conclussion

This study presents a comprehensive mathematical modeling framework to investigate the transmission dynamics of HIV. By formulating and analyzing a system of ordinary differential equations, we gained insights into the behavior of the disease, including the basic reproduction number and stability of disease-free equilibrium (DFE) and the endemic equilibrium was examined, providing a deeper understanding of the long-term dynamics of the disease. A sensitivity analysis was conducted to assess the impact of key parameters on the basic reproduction number, providing valuable information on the factors driving the spread of HIV. Numerical simulations were performed, and the results were visualized through graphs, revealing important trends and patterns. The findings suggest that increasing the rate at which infected individuals seek treatment leads to a rise in the population receiving treatment, while a higher rate of treatment among AIDS patients results in a decline in the AIDS population. These results have significant implications for the development of effective strategies to control the spread of HIV, highlighting the importance of timely treatment and targeted interventions. The study's outcomes can inform public health policy and guide the allocation of resources to combat the HIV epidemic. Future research should focus on identifying best optimal control strategies to mitigate disease transmission and also validate the model using real-world data which is crucial for translating theoretical findings into practical public health applications.

References

Adeyemi, MO.,& Oluyo, T.O. (2023). Mathematical modelling for the Control of Fly Borne Mastitis Disease in Cattle. *Frontirrs in Applied Mathematics and Statistics,* vol 9, 1-19. https://doi.org/10.3389/fams.2023.1171157

Ayele, T.K., Goufo, E.F.D., Mugisha, S. (2021). Mathematical Modeling of HIV/AIDS with Optimal Control: A case Study in Ethiopia. *Results in Physics*, 26, Article ID: 104263. https://doi.org/10.1016/j.rinp.2021.104263

Chitnis, N, Cushing, J. M, Hyman, M. (2008). Determining important parameters in the spread of Malaria Through the sensitivity analysis of Mathematical model. *Bulleting of Mathematical Biology*, 70:1272-1296. https://doi.org/10.1137/050638941

Cox N.S., Wu.L, Wittenaver, R.,Clark S. (2024). Impact of HIV self-testing of oral pre-exposure prophylaxis scale-up on drug resistance and HIV outcomes in Kenya; a modeling study. *The Lancet*, l 11(3), 167-175.

Habibah, U, Rois, M.A. (2024). Significant Parameters in the HIV/AIDS Transmission and Control Optmal Problem. *Jordan Journal of Mathematics and Statistics* (JJMS), 17(1), 23-43. https://www.who.int/news-room/fact-sheets/details/hiv-aids.

Ibrahim I.A., Daniel E.E, DanHausa A.A., Adamu M.U., Shawalu, C.J., &Yusuf A. (2021). Mathematical Modelling of Dynamics of HIV Transmission Depicting the Importance of Counselling and Treatment. *J. Appl. Sci. Environ. Manage,* 25 (6), 893-903. https://www.ajol.info/index.php/jasem

LaSalle J.P. The stability of dynamical systems, regional conference series in Applied Mathematics (*SIAM)*, Philadelphia. 1976

Mathematical Modeling on Assessing the Impact of Screening on HIV/AIDS Transmission Dynamics. *J.Appl. Sci. Environ. Manage.* 28 (8) 2255-2261 DOI: **https://dx.doi.org/10.4314/jasem.v28i8.1**

Mbabazi, D. (2016). Mathematical Modeling of the spread of HIV/AIDS by Markov Chain Process. *American Journal of Applied Mathematics*, 4(5), 235-246.

Odebiyi, O.A, Oladejo, J.K, Elijah, E.O, Olajide, O.A, Taiwo, A.A, Taiwo, A.J (2024). Mathematical Modeling on Assessing the Impact of Screening on HIV/AIDS Transmission Dynamics. J. Appl. Sci. Environ. Manage. 28(8), 2347-2357.

Odebiyi, O.A, & Oladejo, J.K. (2024). Stability Analysis of An HIV/AIDS Model with Saturated Incidence. International Journal of Mathematical Analysis and Modelling, 7(1), 75-98.

Oladejo, J.K & Oluyo T.O. (2020) Effects of PrEP on HIV/AIDS dynamics with immigration of infectives. *IJMA,* 5(2), 36-54. https://tnsmb.org/journal/index.php/article/view/54.

Olaniyi S. (2018). Dynamics of Zika Virus Model with Nonlinear Incidence and Optimal Control Strategies. *Applied Mathematics and Information Sciences*, 12(5),969-982.

Olaniyi, S. & Obaniyi, O.S. (2014). Qualitative Analysis of Malaria Dynamics with Nonlinear Incidence Function. *Applied Mathematical Sciences*, 8(78), 3889-3904.

Olusola A.O, J.K. Oladejo, Salahu W.O.,A.A. Taiwo, O.W. Ayanrinola (2025). Stability and Sensitivity Analysis of HIV/AIDS Model with Saturated Incidence Rate.

Transpublika International Research in Exact Sciences. Vol. 4 no. 2. https://doi.org/10.55047/tires.v4i2.1650

Omondi, E.O., R.W. and Luboobi, L/S. (2019). A Mathematical Modelling Study of HIV Infection in Two Heterosexual Age groups in Kenya. I*nfectious Disease Modelling*, 4,83-98. https://doi/10.1016/j.idm.2019.04.003

Onsongo, W.M., Mwini, E.D., Nyanaro, B.N. and Osman, S. (2022). Stability Analysis and Modelling the Dynamics of Psittacosis in Human and Poultry Populations. *Communication in Mathematical Biology and Neuroscience, 2022, 1-30.* 10.28919/cmbn/6357

Sarah, Al-sheikh., Farida, M., & Muna, A.,(2011). Stability Analysis of an HIV/AIDS Epidemic Model with Screening. *Inter.Math. Forum.* 6(66): 3251-3273.

Sr. Mary N.Y, Virginia M.K, Isaac, O.O (2024). Mathematical Modeling of HIV Investigating the Effect of Inconsistent Treatment. *Journal of Applied Mathematics and Physics*, 2024, 12: 1063-1078. **https://www.scrip.org/journal/jamp**

Stoddart, C.A & Reyes. R.A.(2006). Models of HIV-1 disease: A Review Status, Drug Discovery Today Disease models 3(1):113-119.

Van den Driessche, P, Watmough, J (2002). Reproduction numbers and Sub-threshold Endemic equilibra for compartment models of disease transmission. *Mathematical biosciences.* 180(1-2), 29-48.

World Health Organization (WHO), (2005). Interim clinical staging of HIV/AIDS and HIV/AIDS case definitions for Surveillance: African region. NLM Unique ID 101255175

World Health Organization (WHO), (2024). Fact Sheets on HIV/AIDS.