

## MATHEMATICAL ASSESSMENT AND STABILITY ANALYSIS OF HIV/AIDS EPIDEMIC MODEL WITH VERTICAL TRANSMISSION AND TREATMENT

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### Abstract

*This study presents a mathematical assessment of the dynamics of HIV/AIDS epidemic with vertical transmission and treatment. The well-posedness is analyzed using the theories of positivity and boundedness. Using the next generation matrix approach, the model's basic reproduction number was obtained. The findings from the analysis also revealed that the model possesses two equilibrium points, the locally stable HIV/AIDS free equilibrium when the threshold parameter is less than unity and endemic equilibrium when  $R_0 > 1$ . The impact of the parameters associated to the basic reproduction number  $R_0$  is investigated using the normalized forward sensitivity index. Furthermore, the model is expanded to incorporate time-dependent antiretroviral treatment and the use of condom. The model's qualitative analysis is supported by numerical simulation.*

**Keywords:** *Mathematical model, HIV/AIDS, Vertical transmission, Treatment, Stability, Lyapunov Function, Optimal Control Theory*

### 1. INTRODUCTION

The emergence and reemergence of infectious diseases like monkeypox, tuberculosis, hepatitis and HIV/AIDS have always been a major public health concern most especially in developing and developed economies of the world (CDC, 2023). The relationship between the causative agent, environment and the host determine the likelihood of infectious diseases. The symptoms of Human Immunodeficiency Virus (HIV) vary depending on the stage of infection. During the initial stage, known as acute HIV infection, people may experience no symptoms or mild flu-like symptoms such as fever, headache, and sore throat within a few weeks of contracting the virus. As the infection progresses and the immune system become compromised, individuals may develop additional symptoms including swollen lymph nodes, weight loss, fever, diarrhea, and cough (WHO, 2023). There are two unique methods through which infectious illness might spread: the vertical and horizontal routes (Olaniyi et al., 2024). Horizontal transmission is the spread of infection from one host to another, typically through direct physical contact or ingestion or inhalation of infective materials while vertical transmission on the other hand is the transfer of infection from a mother to a child either through infected placenta or breastfeeding (Adepoju & Ibrahim, 2024; Adepoju & Olaniyi, 2021). According to World Health Organization estimates from 2021, there were 38.4 million people living with HIV and 650,000 HIV-related deaths worldwide (WHO, 2023; Olaniyi et al., 2024). Although there is currently no vaccine or specific treatment to stop the spread of HIV/AIDS, the dynamics of the disease can be stopped with effective

anti-retroviral medications (ARVs) (WHO, 2023; Omale & Aja, 2019). HIV/AIDS can be spread mainly by sharing contaminated sharp objects, transfusion of contaminated blood, and unprotected sexual activity (WHO, 2023; Cheneke et al., 2021). Another way that HIV can spread is through mother-to-child transmission, also known as vertical transmission. This mostly happens during pregnancy, childbirth, or nursing, and it could be sped up by other important variables such the mother's viral load and the type of birth that was had (Glass et al., 2020). By 2025, it is reported that about ninety five percent of persons living with HIV would be receiving life-saving antiretroviral therapy which would suppress their viral load and prevent HIV from spreading to others in the human population (WHO, 2023; Adepoju & Ibrahim, 2024).

An important tool used for studying and analyzing the dynamical spread of infectious disease in the human population is mathematical modelling (see for instance (Adepoju & Ibrahim, 2024; Bolaji et al., 2024; Olaniyi et al., 2018; Olaniyi & Chuma, 2023) and numerous models have been formulated to analyze the transmission dynamics of HIV/AIDS in order to curb its dynamical spread. Naresh et al. (2006) developed a non-linear mathematical model to study the spread of HIV in a variable-size population through both horizontal and vertical transmission. Their study demonstrated that controlling vertical transmission rate can significantly reduce disease spread and maintain infective and AIDS population equilibrium values at desired levels. Baryarama et al. (2006) developed an HIV/AIDS model that accounts for complacency among adults, where complacency is inversely related to the number of AIDS cases in a community. Kimbir et al. (2012) extended a one-sex mathematical model of HIV/AIDS transmission dynamics to investigate the impact of Counseling and Antiretroviral Therapy (ART) on disease control. Safiel et al. (2012) developed a nonlinear deterministic mathematical model to investigate the dynamics of HIV/AIDS with treatment and vertical transmission. Their result suggested that treatment measures (ARVs) and controlling vertical transmission rates can significantly reduce disease transmission. Kaymakamzade et al. (2018) developed and analyzed two epidemic models to assess the impact of awareness programs on HIV and AIDS transmission in Turkey while Omondi et al. (2018) emphasized the crucial role of public health in controlling HIV spread and mitigating its impact on populations. Similarly, Omale and Aja (2019) addressed the devastating impact of HIV/AIDS, affecting individuals of all ages and genders.

Furthermore, Gurmu et al. (2020) developed a nonlinear deterministic mathematical model to investigate HIV/AIDS transmission dynamics, incorporating a drug resistance compartment and Akinwumi et al. (2021) developed and analyzed a mathematical model to investigate the transmission dynamics of HIV/AIDS and the impact of early treatment on disease progression. According to Ayele et al. (2021), a comprehensive HIV/AIDS epidemic model was developed to capture essential compartments of aware and unaware susceptible individuals. Espitia et al. (2022) to investigate the impact of bisexual behavior on HIV transmission in a global community and Espitia et al. (2022) formulated an HIV/AIDS model capturing homosexual and heterosexual populations. Twagirumukiza and Singirankabo (2021) worked on the mathematical analysis of delayed HIV/AIDS model with treatment and vertical transmission. Olaniyi et al. (2024) developed a mathematical model to examine the dynamical spread of HIV/AIDS, focusing on the impact of vertical transmission and nonlinear treatment.

This research is primarily aimed at reducing vertical transmission of HIV/AIDS and the impact of treatment together with time dependent control measures on the human population. It is important to state clearly that in this study, the primary mode of transmission of HIV/AIDS is through heterosexual contact. Also, treated individuals are assumed to have a negligible viral transmission and due to carelessness, some treated individuals transitioned into full blown AIDS. The organization of the work is as follows: Section 2 presents the full description of model. The analysis of the model is carried out in Section 3, while in Section 4, the numerical simulations of the system are performed. Section 5 wraps up the work with concluding remarks.

## 2. MODEL FORMULATION

The total human population at time  $t$ , denoted by  $N(t)$ , is subdivided into five mutually exclusive compartments of susceptible human, denoted by  $S(t)$ , asymptotically infected human, denoted by  $I_a(t)$ , symptomatically infectious human, denoted by  $I_s(t)$ , treated human, denoted by  $T(t)$  and the full-blown AIDS human denoted by  $A(t)$ , respectively. Then, the total human population is obtained as:

$$N(t) = S(t) + I_a(t) + I_s(t) + T(t) + A(t) \quad (1)$$

The susceptible component of the population increases due to the coming in of new born babies into the population at a rate  $\pi$ . The population decreases due to effective contact with infectious and full-blown AIDS individuals, respectively at an incidence rate  $c_1\beta I_a$ ,  $c_2\beta I_s$  and  $c_3\beta A$ , where  $\beta$  is the effective contact rate and  $c_1$ ,  $c_2$  and  $c_3$  are the modification parameters due to the level of infection in an HIV infected and full-blown AIDS individuals. It is further reduced by vertical transmission and natural mortality at a rate  $a$  and  $\mu$ , respectively. Then, the rate of change of susceptible individuals is given by:

$$\frac{dS}{dt} = \pi - a\pi I_s - \beta(c_1 I_a + c_2 I_s + c_3 A)S - \mu S \quad (2)$$

The population of the asymptomatic infected individuals increases as a result of infection of individuals in the susceptible class at a rate  $\beta(c_1 I_a + c_2 I_s + c_3 A)$ . The class reduces due to the progression of latently infected individuals to active HIV/AIDS infection at a rate  $\sigma$  and full-blown AIDS individuals at a rate  $\varphi$ . The population is further decreased by natural death at a rate  $\mu$ . Then, the rate of change of asymptomatic individuals is given by:

$$\frac{dI_a}{dt} = \beta(c_1 I_a + c_2 I_s + c_3 A)S - (\sigma + \varphi + \mu)I_a \quad (3)$$

The population of infectious individuals increases based on the progression of exposed/latently infected individuals to active HIV infection at a rate  $\sigma$  and further increased by vertical transmission at a rate  $a\pi I_s$ . The population reduces due to treatment at a rate  $\gamma$  and progression to full blown AIDS at a rate  $\psi$ . It is further reduced due to disease induced death and natural mortality at a rate  $\delta$  and  $\mu$ . Then, the rate of change of infectious individuals is given by:

$$\frac{dI_s}{dt} = \sigma I_a - (\gamma + \xi + \delta + \mu)I_s \quad (4)$$

The population of treated individuals increases based on the progression of symptomatic individuals at a rate. The population is reduced by the progression of treated individuals to the full-blown AIDS at a rate  $\tau$ . The population is further reduced due to natural mortality and disease induced death at rates  $\mu$  and  $\delta$ . Then, the rate of change of treated individuals is given by:

$$\frac{dT}{dt} = \gamma I_s - (\tau + \mu + \delta)T \quad (5)$$

The full-blown AIDS compartment increases as a result of progression of asymptomatic individuals at a rate  $\phi$ , symptomatic individuals at a rate  $\psi$  and treated individuals at a rate  $\tau$ . The population is further reduced by natural mortality and disease induced death at rates  $\mu$  and  $\delta$ . Thus, the rate of change of AIDS individuals is given by:

$$\frac{dA}{dt} = \phi I_a + \xi I_s + \tau T - (\delta + \mu)A \quad (6)$$

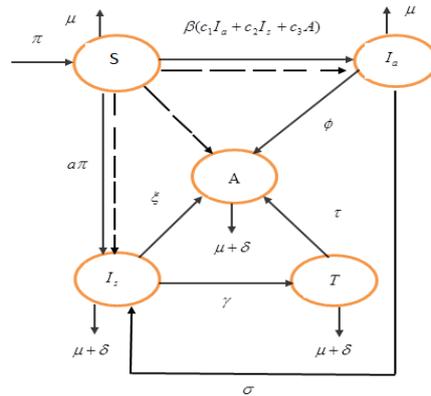
Thus, the mathematical model describing the transmission dynamics of HIV/AIDS is given as follows:

$$\left. \begin{aligned} \frac{ds}{dt} &= \pi - \beta S(c_1 I_a + c_2 I_s + c_3 A) - \mu S - a \pi I_s \\ \frac{dI_a}{dt} &= \beta S(c_1 I_a + c_2 I_s + c_3 A) - (\sigma + \phi + \mu)I_a \\ \frac{dI_s}{dt} &= \sigma I_a + a \pi I_s - (\gamma + \delta + \xi + \mu)I_s \\ \frac{dT}{dt} &= \gamma I_s - (\delta + \tau + \mu)T \\ \frac{dA}{dt} &= \tau T + \phi I_a + \xi I_s - (\delta + \mu)A \end{aligned} \right\} \quad (7)$$

The state variables (7) are subject to the initial conditions:  $S(t) > 0, I_a(t) \geq 0, I_s(t) \geq 0, T(t) \geq 0, A(t) \geq 0$

**Table 1. The Description of Variables of the HIV/AIDS Model (7)**

Variables	Description
S	Susceptible individual
$I_a$	Asymptomatic individual
$I_s$	Symptomatic individual
T	Treated individual
A	Full-blown AIDS individual



**Figure 1. The Schematic Diagram Describing the Dynamical Spread of HIV/AIDS**

**Table 2. The Description of Parameters of the HIV/AIDS Model (7)**

Parameters	Description
$\pi$	Recruitment rate into the population
$\beta$	Transmission rate
$\mu$	Natural mortality rate
$\delta$	Disease induced death rate
$\sigma$	Progression rate from asymptomatic to symptomatic class
$\alpha$	Vertical transmission rate
$\gamma$	Progression rate from symptomatic to treated class
$\phi$	Rate at which asymptomatic become full blown Aids
$\xi$	Rate at which symptomatic become full blown Aids
$\tau$	Rate at which treated become full blown Aids
$c_1, c_2, c_3$	Probability of disease transmission

### 3. ANALYSIS OF THE HIV/AIDS MODEL

#### 3.1. The Invariant region

The HIV/AIDS model (7) will be analyzed in a biologically feasible region defined as follows. This region can be shown to be positively invariant and attracting for all positive solutions of the HIV/AIDS model.

$$D = \{(S, I_a, I_s, T, A) \in \mathbb{R}_+^5 : S + I_a + I_s + T + A \leq \frac{\pi}{\mu}\}$$

**Theorem 1:** The region  $D \subset \mathbb{R}_+^5$  is positively-invariant and attracting with respect to the HIV/AIDS model with non-negative initial data.

**Proof:** Let the total population  $N(t) = S(t) + I_a(t) + I_s(t) + T(t) + A(t)$ , then adding all the equation of the system (7) gives the following results:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI_a}{dt} + \frac{dI_s}{dt} + \frac{dT}{dt} + \frac{dA}{dt} \tag{8}$$

Then the summation of the change of the total population yields

$$\frac{dN}{dt} = \pi - \mu N \quad (9)$$

so that,

$$\frac{dN}{dt} + \mu N \leq \pi \quad (10)$$

solving (10) by standard technique, it follows that

$$N(t) \leq N(0)e^{-\mu t} + \frac{\pi}{\mu}(1 - e^{-\mu t}) \quad (11)$$

If  $N(0) \leq \frac{\pi}{\mu}$ , then  $N(t) \leq \frac{\pi}{\mu}$  for all  $t > 0$ . Hence the feasible region  $D$  of the HIV/AIDS model is positively invariant. Furthermore, if  $N(0) \geq \frac{\pi}{\mu}$ , then the solution enters  $D$  in finite time or  $N(t)$  approaches  $\frac{\pi}{\mu}$  asymptotically as  $t \rightarrow \infty$ . Hence the region  $D$  attracts all the solutions in  $R_+^5$ .

Since the region is positively-invariant and attracting, then it is enough to investigate the dynamics of the mathematical model (7) in the feasible region. Hence, the HIV model (7) is mathematically and epidemiologically meaningful.

### 3.2. Positivity of Solutions

The mathematical model governing the dynamical spread of HIV/AIDS model (7) considers only the human population. Then, it is important that all its state variables and associated parameters are non-negative for all time,  $t$ . Hence, the following result holds for all the state variables in the mathematical model.

**Theorem 2** : Let the initial data for the HIV/AIDS mathematical model (8) be  $S(0) > 0$ ,  $I_a(0) > 0$ ,  $I_s(0) > 0$ ,  $T(0) > 0$  and  $A(0) > 0$ . Then, the solution of the HIV/AIDS mathematical model (7), remains non-negative for all  $t > 0$ .

**Proof** : From the first compartment of the system (7), it follows that

$$\frac{dS}{dt} = \pi - a\pi I_s - \beta(c_1 I_a + c_2 I_s + c_3 A)S - \mu S \quad (12)$$

this implies that

$$\frac{dS}{dt} + \beta(c_1 I_a + c_2 I_s + c_3 A + \mu)S > 0 \quad (13)$$

solving (13) by standard technique yields

$$S(t) > S(0) \exp \left[ - \int_0^t \beta (c_1 I_a(w) + c_2 I_s(w) + c_3 A(w)) dw + \mu t \right] > 0 \text{ for all } t > 0 \quad (14)$$

Following the same step, it can be shown that the remaining state variables  $I_a(t) > 0, I_s(t) > 0, T(t) > 0, A(t) > 0 \forall t > 0$ . Thus, all the solution of the HIV/AIDS model (7) remain positive for all non-negative initial conditions.

### 3.3. Equilibrium Points and Stability Analysis

#### 3.3.1. Disease-free Equilibrium

The disease-free equilibrium point is a stable position where the entire population has no infection. Then at steady state, the HIV/AIDS model (1) has a disease-free equilibrium point.

$$\mathcal{E}_0 = \left( \frac{\pi}{\mu}, 0, 0, 0, 0 \right) \quad (15)$$

#### 3.3.2. Basic Reproduction Number

The basic reproduction number, denoted  $R_0$ , is defined as the average number of new cases of secondary infections caused by an infectious individual in the population of susceptible. It is an important threshold under which the incidence of HIV/AIDS persists or dies out in the human population. The basic reproduction number  $R_0$  is calculated using the approach of Adepoju and Ibrahim (2024); Diekmann et al. (1990); Van den Driessche and Watmough (2002) where the infectious compartments of the system (1) are considered at  $E_0$ . The transmission matrix  $F$  and transition matrix  $V$  obtained at HIV/AIDS free equilibrium  $E_0$  are given as follows:

$$\mathcal{F} = \begin{pmatrix} \beta \frac{c_1 \pi}{\mu} & \beta \frac{c_2 \pi}{\mu} & 0 & \beta \frac{c_3 \pi}{\mu} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$\mathcal{V} = \begin{pmatrix} k_1 & 0 & 0 & 0 \\ -\sigma & k_2 - a\pi & 0 & 0 \\ 0 & -\gamma & k_3 & 0 \\ -\phi & -\xi & -\tau & k_4 \end{pmatrix}$$

$$k_1 = (\sigma + \mu + \phi), \quad k_2 = (\gamma + \delta + \mu + \xi), \quad k_3 = (\delta + \mu + \tau), \quad k_4 = (\delta + \mu)$$

Then the spectral radius of  $FV^{-1}$  is the dominant eigenvalue of  $|FV^{-1} - \lambda I|$  obtained as

$$\mathcal{R}_0 = \frac{\beta \left[ \pi(c_1 k_3 k_4 (k_2 - a\pi) + c_2 \sigma k_3 k_4 + c_3 \sigma (\gamma \tau + k_3 \xi) + c_3 \phi k_3 (k_2 - a\pi)) \right]}{k_1 k_3 k_4 \mu (k_2 - a\pi)} \quad (16)$$

Since the threshold parameter  $R_0$  is usually positive, then the inequality  $k_2 - a\pi > 0$  must hold.

**Theorem 3** : The HIV/AIDS free equilibrium point of the system (7) is locally asymptotically stable whenever the basic reproduction number  $R_0$  is less than unity.

**Proof** : The Jacobian matrix of the system (7) obtained at  $E_0$  is given as

$$J|_{\varepsilon_0} = \begin{bmatrix} -\mu & -\frac{c_1\beta\pi}{\mu} & -a\pi - \frac{c_2\beta\pi}{\mu} & 0 & \frac{c_3\beta\pi}{\mu} \\ 0 & \frac{c_1\beta\pi}{\mu} - k_1 & \frac{c_2\beta\pi}{\mu} & 0 & \frac{c_3\beta\pi}{\mu} \\ 0 & \sigma & a\pi - k_2 & 0 & 0 \\ 0 & 0 & \gamma & -k_3 & 0 \\ 0 & \varphi & \xi & \tau & -k_4 \end{bmatrix} \quad (17)$$

Obviously, one of the eigenvalues of (17) is obtained as  $\lambda_1 = -\mu$  and the remaining are obtained from the polynomial given by

$$\lambda^4 + A_1\lambda^3 + A_2\lambda^2 + A_3\lambda + A_4 = 0 \quad (18)$$

where

$$A_1 = k_1 + k_2 + k_3 + k_4 - a\pi - \frac{c_1\beta\pi}{\mu} > 0$$

$$A_2 = (k_1(k_2 + k_3k_4 - a\pi) + k_2(k_3 + k_4) + k_3k_4 - a\pi(k_3 + k_4)) + \frac{c_1\beta\pi}{\mu}(a\pi - k_2 - k_3 - k_4) - \frac{c_2\beta\pi\varphi}{\mu} - \frac{c_3\beta\pi\varphi}{\mu} > 0$$

$$A_3 = (k_2k_3k_4 - k_3k_4a\pi + k_1(k_2(k_3 + k_4) + k_3k_4 - a\pi(k_3 + k_4))) + \frac{c_1\beta\pi}{\mu}(a\pi(k_3 + k_4) - k_2(k_3 + k_4) - k_3k_4) - \frac{c_3\beta\pi}{\mu}(\varphi(k_2 + k_3 - a\pi) + \sigma\xi + \frac{c_2\beta\pi}{\mu}(k_3 + k_4)\sigma) > 0$$

$$A_4 = (k_1k_3k_4\mu(k_2 - a\pi))(1 - R_0)$$

Clearly, the polynomial (18) is the solution of the Jacobian matrix (17). Then following Descarte's rule, all the coefficient of the polynomial are positive. It can be concluded that all the eigenvalues of the Jacobian matrix are negative, real and distinct. Hence, the disease-free equilibrium of the HIV/AIDS model (7) is locally asymptotically stable.

### 3.3.3. Global Stability of HIV/AIDS Free Equilibrium

**Theorem 4** : The HBV-free equilibrium denoted by  $E_0$  is globally asymptotically stable whenever  $R_0 < 1$ .

**Proof** : The proof is based on using comparison theorem as applied in Augusto and Gumel (2010); Erinle-Ibrahim et al. (2022) by considering the infectious compartments. The infected compartments of system (7) can be written in the form

$$\begin{pmatrix} \frac{dI_a}{dt} \\ \frac{dI_s}{dt} \\ \frac{dT}{dt} \\ \frac{dA}{dt} \end{pmatrix} = \begin{pmatrix} I_a \\ I_s \\ T \\ A \end{pmatrix} - \left(1 - \frac{S}{N}\right) F \begin{pmatrix} I_a \\ I_s \\ T \\ A \end{pmatrix} \quad (19)$$

where the matrices  $F$  and  $V$  are given in section (3.32) respectively. Since  $S(t) \leq N(t)$  for all  $t \geq 0$ , it follows from (19) that

$$\begin{pmatrix} \frac{dI_a}{dt} \\ \frac{dI_s}{dt} \\ \frac{dT}{dt} \\ \frac{dA}{dt} \end{pmatrix} \leq (F - V) \begin{pmatrix} I_a \\ I_s \\ T \\ A \end{pmatrix} \quad (20)$$

Now, using the fact that the eigenvalues of  $|(F - V) - \lambda I|$  all have negative real parts as obtained in (18), it follows that the differential inequality (20) is stable whenever  $R_0 < 1$ . Consequently,  $(I_a(t), I_s(t), T(t), A(t)) \rightarrow (0, 0, 0, 0)$  as  $t \rightarrow \infty$ . Thus by comparison theorem (Huo and Feng 2013), it follows that  $(I_a, I_s, T, A) \rightarrow (0, 0, 0, 0)$ . Then  $(S, I_a, I_s, T, A) \rightarrow \left(\frac{\pi}{\mu}, 0, 0, 0, 0\right)$  as  $t \rightarrow \infty$ . Hence the HIV/AIDS free equilibrium  $E_0$ , is globally asymptotically stable when  $R_0 < 1$ .

### 3.4. Endemic Equilibrium

The endemic equilibrium point is the state where there is presence of disease in the population (Adepoju & Ibrahim, 2024; Egonmwan & Okuonghae, 2019; Olaniyi et al., 2023). Let the endemic equilibrium point be denoted by  $E^*$  and  $\lambda^* = \beta(c_1 I_a + c_2 I_s + c_3 A)$  be the force of infection such that  $(S^*, I_a^*, I_s^*, T^*, A^*)$ . Then, at steady states

$$\frac{dS^*}{dt} = \frac{dI_a^*}{dt} = \frac{dI_s^*}{dt} = \frac{dT^*}{dt} = \frac{dA^*}{dt} = 0 \quad (21)$$

Solving system (7) simultaneously yields

$$\begin{aligned} S^* &= \frac{\pi k_1 (k_2 - a\pi)}{k_1 (k_2 - a\pi)(\lambda^* + \mu) + \pi a \sigma \lambda^*} \\ I_a^* &= \frac{\lambda^* \pi (k_2 - a\pi)}{k_1 (k_2 - a\pi)(\lambda^* + \mu) + \pi a \sigma \lambda^*} \\ I_s^* &= \frac{\lambda^* \pi \sigma}{k_1 (k_2 - a\pi)(\lambda^* + \mu) + \pi a \sigma \lambda^*} \\ T^* &= \frac{\gamma \pi \sigma \lambda^*}{k_3 (k_1 (k_2 - a\pi)(\lambda^* + \mu) + \pi a \sigma \lambda^*)} \\ A^* &= \frac{\tau \gamma \pi \sigma \lambda^* + k_3 \phi \lambda^* \pi (k_2 - a\pi) + k_3 \pi \sigma \lambda^* \xi}{k_3 k_4 (k_1 (k_2 - a\pi)(\lambda^* + \mu) + \pi a \sigma \lambda^*)} \end{aligned} \quad (22)$$

Substituting the values of  $I_a^*$ ,  $I_s^*$  and  $A^*$  into the force of infection yields

$$\lambda^* = \frac{k_1 k_3 k_4 \mu (k_2 - a\pi)}{k_3 k_4 (k_1 k_2 - a\pi (k_1 - \sigma))} (R_0 - 1) \quad (23)$$

Ultimately, there would be persistence of HIV/AIDS in the population whenever the basic reproduction number  $R_0$  is greater than unity.

### 3.5. Global Stability of Endemic Equilibrium

The global asymptotic stability of the endemic equilibrium point is solved using the approach of Adepoju and Ibrahim (2024); Olaniyi et al. (2024)

Theorem 5 : The endemic equilibrium points  $E^*$  is globally asymptotically stable whenever  $R_0 > 1$

Proof : Consider the Lyapunov function  $G : D \in \mathbb{R}_+^5 \rightarrow \mathbb{R}_+$  defined by

$$G = \frac{1}{2}((S - S^*) + (I_a - I_a^*) + (I_s - I_s^*) + (T - T^*) + (A - A^*))^2 \quad (24)$$

Taking the time derivate of (24) gives

$$\begin{aligned} \dot{G} &= \{(S - S^*) + (I_a - I_a^*) + (I_s - I_s^*) + (T - T^*) + (A - A^*)\} \frac{d}{dt} (S + I_a + I_s + T + A) \\ &= \{(S - S^*) + (I_a - I_a^*) + (I_s - I_s^*) + (T - T^*) + (A - A^*)\} \\ &\times \{\pi - \mu(S + I_a + I_s + T + A) - \delta(I_s + T + A)\} \\ \dot{G} &\leq \{(S - S^*) + (I_a - I_a^*) + (I_s - I_s^*) + (T - T^*) + (A - A^*)\} \\ &\times \{\pi - \mu(S + I_a + I_s + T + A)\} \\ &= \mu\{(S - S^*) + (I_a - I_a^*) + (I_s - I_s^*) + (T - T^*) + (A - A^*)\} \\ &\times \{(S + I_a + I_s + T + A) - \frac{\pi}{\mu}\} \end{aligned} \quad (25)$$

Since  $N^* = \frac{\pi}{\mu}$ , then (25) becomes

$$\begin{aligned} \dot{G} &= \{(S - S^*) + (I_a - I_a^*) + (I_s - I_s^*) + (T - T^*) + (A - A^*)\} \\ &\times \{(S + I_a + I_s + T + A) - (S^* + I_a^* + I_s^* + T^* + A^*)\} \\ &= -\mu\{(S - S^*) + (I_a - I_a^*) + (I_s - I_s^*) + (T - T^*) + (A - A^*)\} \\ &\times \{(S - S^*) + (I_a - I_a^*) + (I_s - I_s^*) + (T - T^*) + (A - A^*)\} \\ &= -\mu\{(S - S^*) + (I_a - I_a^*) + (I_s - I_s^*) + (T - T^*) + (A - A^*)\}^2 \end{aligned} \quad (26)$$

Proportionately, the time derivative of the continuously differentiable function  $G$  is negative semi-definite. That is  $\dot{G} \leq 0$ . Then, the function  $G$  is a Lyapunov function.

Therefore,  $\dot{G} = 0$  provided  $S = S^*$ ,  $I_a = I_a^*$ ,  $I_s = I_s^*$ ,  $T = T^*$  and  $A = A^*$ . Then, by LaSalle's invariance principle (LaSalle 1976), the largest invariance set for which  $\dot{G} = 0$  is the singleton set  $\{E^*\}$ , which implies that the endemic equilibrium point of the HIV/AIDS model (7) is globally asymptotically stable.

### 3.6. Sensitivity Analysis

The changes occurring in the dynamics of HIV/AIDS are observed by computing the sensitivity indices of the basic reproduction number. The sensitivity analysis is important to discover how best the spread of HIV/AIDS can be minimized by studying the relative critical factors responsible for its transmission and prevalence with respect to the values of the parameters of the model. However, the normalized forward sensitivity index of a variable to a parameter is the ratio of the relative change in the variable to the relative change in the parameter (Chitnis et al., 2008; Rois et al., 2021). The normalized

forward sensitivity indices of the basic reproduction number  $R_0$ , relative to its parameter  $q$  is given by

$$\Gamma_q^{R_0} = \frac{\partial R_0}{\partial q} \times \frac{q}{R_0} \quad (27)$$

Using (27), the sensitivity indices of the parameters associated with basic reproduction number is calculated and the result is presented in Table 3.

**Table 3. The Parameter Values for Optimality System Simulations**

Parameter	Values	Sensitivity Index	Source
$\beta$	0.0003	+1.0000	Cai et al. (2009)
$\pi$	0.14	+1.0027	Yusuf and Benyah (2012)
$\mu$	0.5	-1.7124	Naresh et al. (2006)
$\gamma$	0.4	-0.0013	Naresh et al. (2006)
$\delta$	0.02	-0.0125	Cai et al. (2009)
$\phi$	0.743	-0.2723	Omale and Aja (2019)
$\sigma$	0.01	-0.0017	Cai et al. (2009)
$\xi$	0.09	-0.0001	Ibrahim et al. (2021)
$\alpha$	0.3	+0.0002	Cai et al. (2009)
$\tau$	1.05	+0.0003	Oyovwevotu (2021)
$c_1$	3	+0.6731	Safiel et al. (2012)
$c_2$	2	+0.3222	Safiel et al. (2012)
$c_3$	1	+0.0002	Safiel et al. (2012)

#### 4. ANALYSIS OF THE OPTIMAL CONTROL

Here, the HIV/AIDS model (7) is expanded to incorporate two optimal control functions  $u_i(t), i = 1, 2$  where  $u_1(t)$ , represents the control for Antiretroviral treatment and  $u_2(t)$ , represents the control of use of condom. The resulting non-autonomous system for the HIV/AIDS transmission is provided as follows:

$$\left. \begin{aligned} \frac{ds}{dt} &= \pi - (1 - u_1)\beta S(c_1 I_a + c_2 I_s + c_3 A) - (1 - u_2)a\pi I_s - \mu S \\ \frac{dI_a}{dt} &= (1 - u_1)\beta S(c_1 I_a + c_2 I_s + c_3 A) - (\sigma + \phi + \mu)I_a \\ \frac{dI_s}{dt} &= \sigma I_a - (\gamma + \delta + \xi + \mu)I_s + (1 - u_2)a\pi I_s \\ \frac{dT}{dt} &= \gamma I_s - (\delta + \tau + \mu)T \\ \frac{dA}{dt} &= \tau T + \phi I_a + \xi I_s - (\delta + \mu)A \end{aligned} \right\} \quad (28)$$

The system (28) is analyzed using the Pontryagin's maximum principle (Adepoju & Ibrahim, 2024; Olaniyi & Chuma, 2023; Oyovwevotu, 2021; Pontryagin, 2018) with the

goal of minimizing the infectious population while minimizing the cost of implementing the control. This can only be determined using the objective functional given as

$$J = \int_0^T (E_1 I_a + E_2 I_s + E_3 T + E_4 A + \frac{1}{2} \sum_{i=1}^2 B_i u_i^2) dt \quad (29)$$

where  $T$  is the final time for the implementation of the controls and  $B_i, i=1,2$  are positive weight constants. The cost control for antiretroviral treatment is given as  $\frac{1}{2} B_1 u_1^2$  while the cost control for the use of condom is given by  $\frac{1}{2} B_2 u_2^2$ . The quadratic cost functional is used to show the non-linearity of each of the control as can be seen in existing literature (Adepoju & Ibrahim, 2024; Olaniyi et al., 2020; Olaniyi & Chuma, 2023; Tasman et al., 2022). The optimal control double is required to minimize the cost functional (29) subject to the state system (28) and the minimization problem is given by

$$J(u_1^*, u_2^*) = J(u^*) = \min J(u_1, u_2) : (u_1, u_2) \in U \quad (30)$$

where  $U = \{u_i(t) : 0 \leq u_i \leq 1, i = 1, 2 \text{ and } t \in [0, T]\}$  is Lebesgue measurable.

#### 4.1. Characterization of Optimal Control

The Pontryagin's maximum principle (Pontryagin, 2018) is used to minimize the problem (29) subject to the state system (28) and can be converted into auxiliary minimization problem of Hamiltonian with respect to the control variables  $u_1^*$  and  $u_2^*$ . The Hamiltonian of the control problem is given as

$$\begin{aligned} H = & E_1 I_a + E_2 I_s + E_3 T + E_4 A + \frac{1}{2} \sum_{i=1}^2 B_i U_i^2 \\ & + \lambda_S (\pi - (1 - u_1(t)) \beta S (c_1 I_a + c_2 I_s + c_3 A) - (1 - u_2(t)) a \pi I_s - \mu S) \\ & + \lambda_{I_a} ((1 - u_1(t)) \beta S (c_1 I_a + c_2 I_s + c_3 A) - (\mu + \sigma + \phi) I_a) \\ & + \lambda_{I_s} (\sigma I_a - (\gamma + \delta + \mu + \xi) I_s + (1 - u_2(t)) a \pi I_s) \\ & + \lambda_T (\gamma I_s - (\delta + \mu + \tau) T) \\ & + \lambda_A (\tau T + \phi I_a + \xi I_s - (\delta + \mu) A) \end{aligned} \quad (31)$$

where  $\lambda_S, \lambda_{I_a}, \lambda_{I_s}, \lambda_T, \lambda_A$  are the co-state variables associated with each of the state variable respectively. The existence of the co-state variables and the characterization of the control variables are shown in the following

**Theorem 6** : Given the optimal control double  $(u_1^*, u_2^*)$  with the solution of the corresponding state system (28) satisfying (29), then there exist adjoint variables  $\lambda_S, \lambda_{I_a}, \lambda_{I_s}, \lambda_T$  and  $\lambda_A$  satisfying the co-state system given by

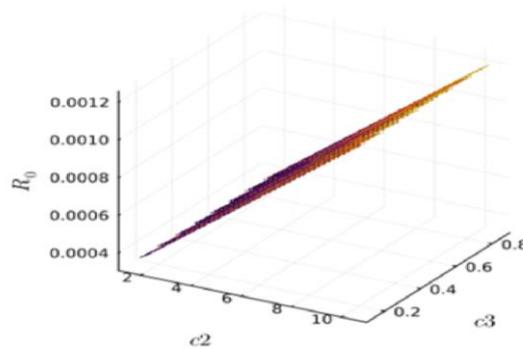
$$\begin{aligned}
 \frac{d\lambda_s}{dt} &= (1-u_1)\beta(c_1I_a + c_2I_s + c_3A)(\lambda_s - \lambda_{i_a}) + \mu\lambda_s \\
 \frac{d\lambda_{i_a}}{dt} &= (1-u_1)\beta C_1 S(\lambda_s - \lambda_{i_a}) + \sigma(\lambda_{i_a} - \lambda_s) + \phi(\lambda_{i_a} - \lambda_A) + \mu\lambda_{i_a} - E_1 \\
 \frac{d\lambda_{i_s}}{dt} &= (1-u_1)\beta C_2 S(\lambda_s - \lambda_{i_s}) + (1-u_2)a\pi(\lambda_s - \lambda_{i_s}) + \gamma(\lambda_{i_s} - \lambda_A) + \xi(\lambda_{i_s} - \lambda_A) + \lambda_{i_s}(\mu + \delta) - E_2 \\
 \frac{d\lambda_T}{dt} &= \tau(\lambda_T - \lambda_A) + \lambda_T(\delta + \mu) - E_3 \\
 \frac{d\lambda_A}{dt} &= (1-u_1)\beta C_3 S(\lambda_s - \lambda_{i_a}) + \lambda_A(\delta + \mu) - E_4
 \end{aligned} \tag{32}$$

with the transversality conditions

$$\lambda_i(Tf) = 0 \quad i = 1,2 \tag{33}$$

and optimal control characterizations

$$\begin{aligned}
 u_1^* &= \min \left\{ \max \left\{ 0, \frac{\beta S(E_1 I_a + E_2 I_s + E_3 A)(\lambda_{i_a} - \lambda_s)}{B_1} \right\}, 1 \right\} \\
 u_2^* &= \min \left\{ \max \left\{ 0, \frac{a\pi I_s(\lambda_s - \lambda_{i_s})}{B_2} \right\}, 1 \right\}
 \end{aligned} \tag{34}$$



**Figure 2. Effect of the Transmission Probabilities  $C_1$  And  $C_2$  on the Basic Reproduction Number Indicating Prevalence of HIV/AIDS in the Human Population**

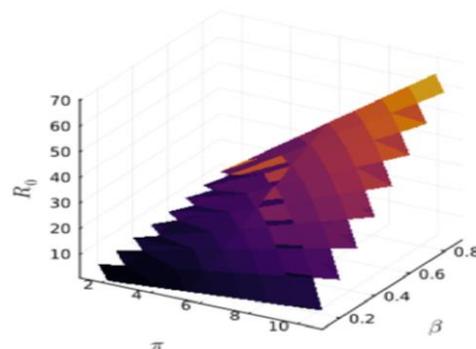
#### 4.2. Optimality System Simulation

The optimality system is obtained by coupling the state system (28), adjoint system (31) and optimal control characterization together. Their corresponding initial and transversality conditions are solved using the forward-backward fourth order Runge-Kutta method (Lenhart & Workman, 2007). The values of the parameters employed are listed in Table 3 so that  $R_0 = 0.0002988$ . The impacts of combining both controls are examined in this study. The initial condition for the state variables is  $S = 400, I_a = 100, I_s$

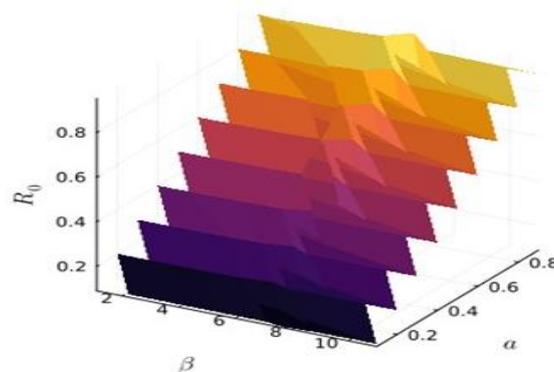
$= 50$ ,  $T = 20$ , and  $A = 30$ . The weight constants of the objective functional are given as  $E_1 = 1$ ,  $E_2 = 1$ ,  $E_3 = 1$ ,  $E_4 = 1$ ,  $B_1 = 1$  and  $B_2 = 1$

The effect of the transmission probability of disease per contact by an asymptomatic individual  $c_1$  and the transmission probability of disease per contact by a symptomatic individual  $c_2$  on the basic reproduction number of the HIV/AIDS model is demonstrated in Figure 2. It is observed that both have corresponding increase on the basic reproduction number and will increase the prevalence of HIV/AIDS in the population. In Figure 3, increase in the value of recruitment rate  $\pi$  and transmission rate  $\beta$ , resulted into a complementary increase on the basic reproduction number. As shown Figure 4, increase in the value of vertical transmission rate  $a$  and transmission rate  $\beta$ , resulted into a proportionate increase on the threshold parameter. Figure 5 depicts the convergence of the symptomatic infected individuals to the disease-free equilibrium point while Figure 6 shows the convergence of the asymptomatic infected individuals to a unique endemic equilibrium point regardless of the initial sizes of their population.

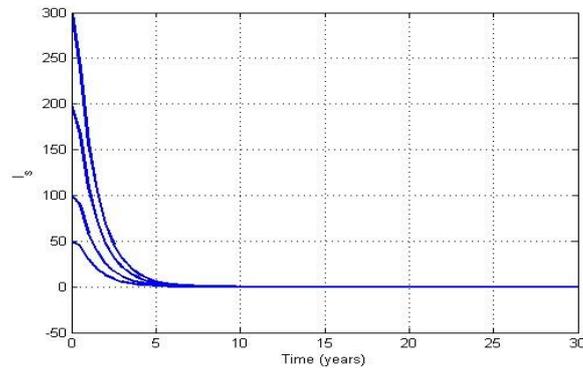
Figure 3 shows the effect of transmission rate on the asymptomatic class. At  $\beta = 0.001$ , it is observed that there is little or no transmission. At  $\beta = 0.002$ , a slight increase in transmission is observed and when  $\beta = 0.005$ , an increase in transmission is observed. This might be due to carelessness on the part of the individual involved and may lead to prevalence of HIV/AIDS in the population. The behavior of asymptomatic individuals.



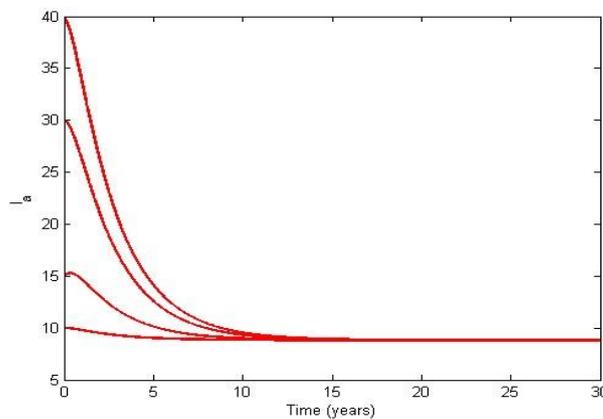
**Figure 3. Effect of  $\beta$  and  $\pi$  on the Basic Reproduction Number  $R_0$  Indicating High Rate of the Dynamical Spread of HIV/AIDS in the Population**



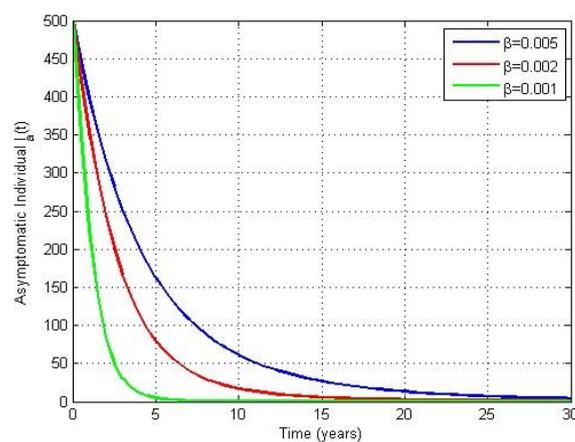
**Figure 4. Effect of  $\beta$  and  $a$  on the Basic Reproduction Number  $R_0$  Indicating Prevalence of HIV/AIDS in the Population**



**Figure 5. Global Stability of Symptomatic Individuals Around HIV/AIDS-Free Equilibrium Indicating Zero Infection Regardless of the Initial Size of the Population**



**Figure 6. Global Stability of Asymptomatic Individuals Around Endemic Equilibrium Depicting the Presence of Disease in the Population**



**Figure 7. Effect of Varying Values of Transmission Rate on the Asymptomatic Compartment**

When antiretroviral treatment  $u_1(t)$  is administered is shown in Figure 8. It is observed that when  $u_1(t)$  is maximally maintained, the viral load of the asymptomatic individuals reduces and will curb dynamical spread of HIV/AIDS in the population. The impact of the use of condom  $u_2(t)$  by the full-blown AIDS individuals is shown in Figure 9. It is deduced that when the use of condom is strictly followed as prescribed, there would be reduction in transmission. The impact of the control combination on the symptomatic individuals for antiretroviral treatment and the use of condom is depicted in Figure 10. It is evident that when both  $u_1(t)$  and  $u_2(t)$  are taken into consideration, the population of symptomatic individuals is considerably smaller. This indicates that dynamical spread of HIV/AIDS in the population can be controlled. The influence of control combination of antiretroviral treatment and the use of condom on the population of AIDS individuals is depicted in Figure 10. It can be deduced that the population of AIDS individuals reduced drastically in the presence of both controls and this will help in minimizing the spread of the disease. It is noteworthy to state that the results in the optimality system are different from the results obtained in the already existing studies.

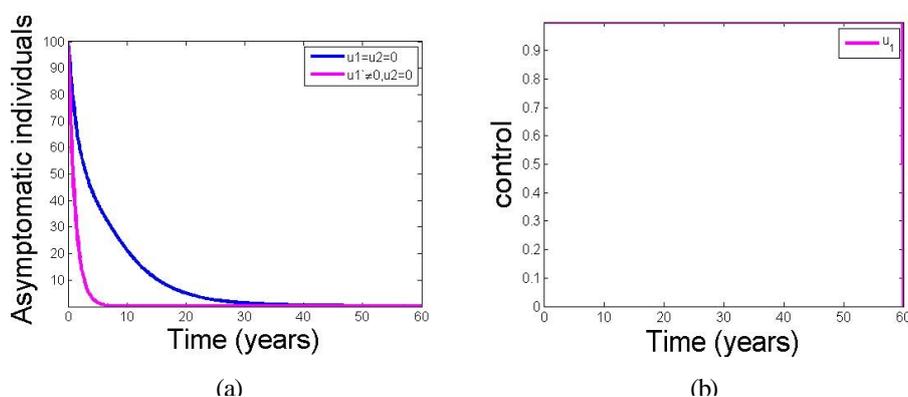


Figure 8. Trajectory of the Administration of Antiretroviral Treatment on the Asymptomatic Individuals

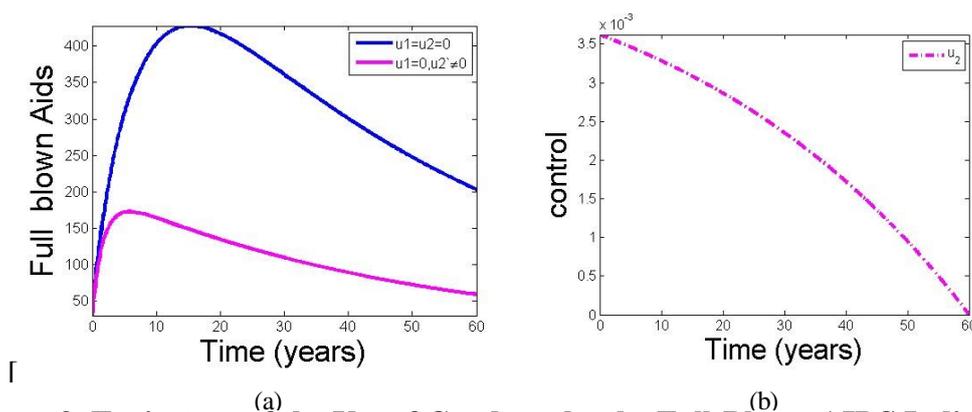
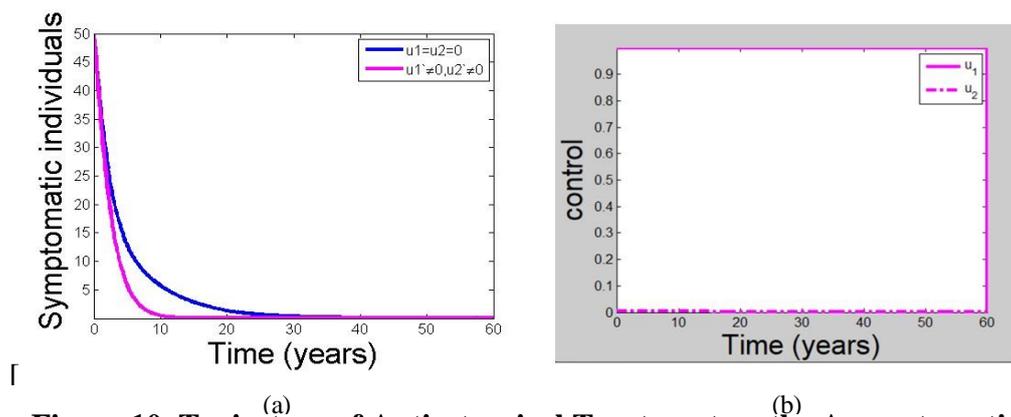


Figure 9. Trajectory of the Use of Condoms by the Full-Blown AIDS Individuals Depicting Reduction in the Transmission of HIV/AIDS in the Population



(a) (b)  
**Figure 10. Trajectory of Antiretroviral Treatment on the Asymptomatic Individuals Showing Reduction in the Viral Load**

## 5. CONCLUSION

In this study, a mathematical model was developed and analyzed to investigate the effect of vertical transmission and treatment of HIV/AIDS in human population using a system of ordinary differential equations. The human population is categorized into five mutually exclusive compartments of Susceptible, Asymptomatic, Symptomatic, Treated and full-blown AIDS individuals respectively. The well-posedness of the model was investigated using the theory of positivity and boundedness of solution. The disease-free equilibrium was obtained analytically and the basic reproduction number was calculated using the next generation matrix method. Using the linearized Jacobian matrix and Comparison test methods, the disease-free equilibrium point was shown to be locally and globally asymptotically stable when  $R_0 < 1$ . This implies that when the basic reproduction number is less than unity, HIV/AIDS can be controlled in the human population. This is in agreement with earlier results of existing literature. See for instance (Adepoju & Ibrahim, 2024; Ayele et al., 2021; Espitia et al., 2022). Using a quadratic Lyapunov function, the global asymptotic stability of the endemic equilibrium point was established. Furthermore, the influence of the parameters of the threshold parameter  $R_0$  was investigated using the normalized forward sensitivity index and it was shown that parameters with positive indices will increase  $R_0$  while those with negative indices will reduce it.

The impact of the optimal control double for antiretroviral treatment and the use of condom were examined using Pontryagin's maximum principle and it was deduced that time dependent control measures helped reduce the dynamical spread of HIV/AIDS in the human population. Antiretroviral treatment and the use of condom should be optimized and encouraged for infected individuals, as part of a comprehensive optimal control strategy to prevent transmission. Moreover, efforts should be made by policy makers and healthcare practitioners to increase the values of parameters with negative sensitivity indices and decrease the value of parameters with positive sensitivity indices to mitigate the spread of HIV/AIDS transmission dynamics in the population. The values of the parameter used in this study are picked from the already existing studies on HIV/AIDS.

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