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GLOBAL STABILITY ANALYSIS OF AN AGE-STRUCTURED MATHEMATICAL MODEL OF HIV/AIDS TRANSMSSION DYNAMICS WITH CONTROL STATEGIES

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ABSTRACT

The global stability analysis of an age-structured model of HIV/AIDS transmission dynamics in Nigeria has been performed. The two age classes considered are people under 15 years old (immature susceptible) and people between 15 to 64 years (mature susceptible). Python software was used in the analysis. Data used was obtained from the Nigerian Centre for Disease Control. The basic reproduction number (R_0) calculated was 5.15. The global stability analysis carried out shows that with $R_0 \geq 1$, the disease is globally asymptotically unstable implying that the disease will spread without intervention. However, three control measures were applied to each of the two age classes and the effective reproduction number obtained was 0.41 which implies that the disease - free equilibrium is globally asymptotically stable. Hence, applying the three controls each to both children and adults populations was very necessary as this lead to a reduction of number of new infections among susceptible individuals. Therefore, urgent interventions are highly needed to alleviate the transmission dynamics of the disease.

Key words: Global stability, HIV/AIDS, Reproduction number, Control strategies; Age- structured

1 INTRODUCTION

There are a lot of infectious diseases in the world. They include human immunodeficiency virus (HIV), cholera and measles. We shall in this paper, look at the transmission mechanism of HIV as a communicable disease. HIV/AIDS is a chronic communicable disease caused by the Human Immunodeficiency Virus, which attacks white blood cells (CD4 T-lymphocytes) and causes the immune system to deteriorate. If not treated, it advances to Acquired Immunodeficiency Syndrome (AIDS), leaving the body vulnerable to life-threatening opportunistic infections and cancers (WHO, 2026). HIV/AIDS can be passed from person to person through avenues such as breast milk, blood , semen, vaginal and rectal fluids, and breast milk, The key routes are unprotected sexual contact, sharing contaminated needles or syringes, and from parent to child during pregnancy, birth, or breastfeeding. Safe contacts in which the disease cannot be transmitted through include hugging, kissing, sharing food, or insect bites. Nigeria has an adult HIV prevalence of about 1.3% to 1.4%, with roughly 1.9 million people living with the virus, making it one of the largest HIV burdens globally. Approximately 41.0 million people globally live with HIV. In 2025, 570,000 people died from AIDS-related illnesses. That is about one person dying every minute. Around 1.2 million new infections occurred including 94,000 children aged 0 - 14 years (UNAIDS, 2025). Recent international funding contractions have disrupted community-led prevention and testing frameworks worldwide (UNAIDS, 2026).

There are three stages of HIV/AIDS infections. Acute HIV infection which is the first stage of the virus and which shows symptoms like fever, rash and sore throat usually occurs 2 to 4 weeks after exposure. During this stage, the virus multiplies rapidly. The Chronic HIV infection (latent stage) is the second stage of the virus which remains active but reproduces at low levels. At this stage, symptoms may be absent for years while the immune system slowly erodes. The AIDS stage is the final and most severe stage. At this stage, the immune system is very weak, leading to dangerous opportunistic infections and illnesses like tuberculosis and severe pneumonia. It is marked by a CD4 count below 200 cells/mm^3 (WHO, 2026). Control measures for HIV/AIDS mitigation include antiretroviral treatment (ART), post exposure prophylaxis (Pep), pre - exposure prophylaxis (Prep), screening and education.

Several research works on mathematical models of HIV/AIDS transmission and control have been published. Some of them are reviewed and mentioned here. Juping et al (2020) carried out a work titled "Global stability for age-infection-structured human immunodeficiency virus model with heterogeneous transmission". Their mathematical analysis shows that the local and global dynamics are completely determined by the basic reproductive number R_0. If $R_0 < 1$, disease-free equilibrium is globally asymptotically stable. If $R_0 > 1$, the disease-free equilibrium is unstable and the unique endemic equilibrium is globally asymptotically stable. The proofs of global stability utilize Lyapunov functions. Besides, the numerical simulations were illustrated to support their theoretical results and sensitivity analysis of each parameter and their R_0 was obtained by the method of partial rank correlation coefficient (PRCC). Wang and Liu (2022) studied the dynamics of the Hybrid HIV/AIDS Model with age-structure, self-protection and media coverage. They explored the impacts of age structure, self-protection and media coverage on HIV/AIDS transmission using a hybrid model combining partial differential equations (PDEs) and ordinary differential equations (ODEs). Kifle and Wondinmu (2020) developed a non-linear deterministic dynamical system to investigate HIV/AIDS dynamics with age structure and different transmission modes in Ethiopia. Biswas and Haque (2020) carried out a research work titled understanding transmission dynamics of HIV/AIDS with sensitivity analysis and optimal control strategies. They proposed a comprehensive mathematical model to study HIV/AIDS transmission, focusing on males and females. Li and Wang (2019) analyzed an age-structured HIV infection model with cell-to-cell transmission. In their study, they investigated an age-structured HIV infection model with cell-to-cell transmission, highlighting the importance of age structure in HIV dynamics. Omondi et al (2018) developed and examined a model of HIV infection in Kenya's sex-structured population. Pre-exposure prophylaxis, or PrEP, were used, and the results of the model analysis indicate that this has had a good impact on limiting the spread of HIV. Bandara et al (2023) formulated and analyzed a mathematical model on HIV infection and nutrition. Their findings show a favorable correlation between HIV viral loads and the rate of viral generation.

2 MODEL STRUCTURE

We developed an age-structured model of HIV/AIDS transmission dynamics in Nigeria with control measures. The model consists of two age classes: children (less than 15 years) and adults (15-64 years). The total population at time t denoted by $N(t)$ is subdivided into twelve mutually exclusive compartments of Susceptible children ($S_C(t)$), Susceptible adult ($S_A(t)$), Exposed children ($E_C(t)$), Exposed adults ($E_A(t)$), Aware children infective ($I_{1C}(t)$), Aware adults infective ($I_{1A}(t)$), Unaware children infective ($I_{2C}(t)$), Unaware adults infective ($I_{2A}(t)$), Treated children ($T_C(t)$), Treated adult ($T_A(t)$), AIDS children ($A_C(t)$) and AIDS adults ($A_A(t)$) . Hence,

$$N(t) = S_C(t)+S_A(t)+E_C(t)+E_A(t)+I_{1C}(t)+I_{1A}(t)+I_{2C}(t)+I_{2A}(t)+T_C(t)+T_A(t)+A_C(t)+A_A(t).$$

Our aim was to determine the global stability of the model using control measures. Below is the diagram of the model without and with control measures and their equations.

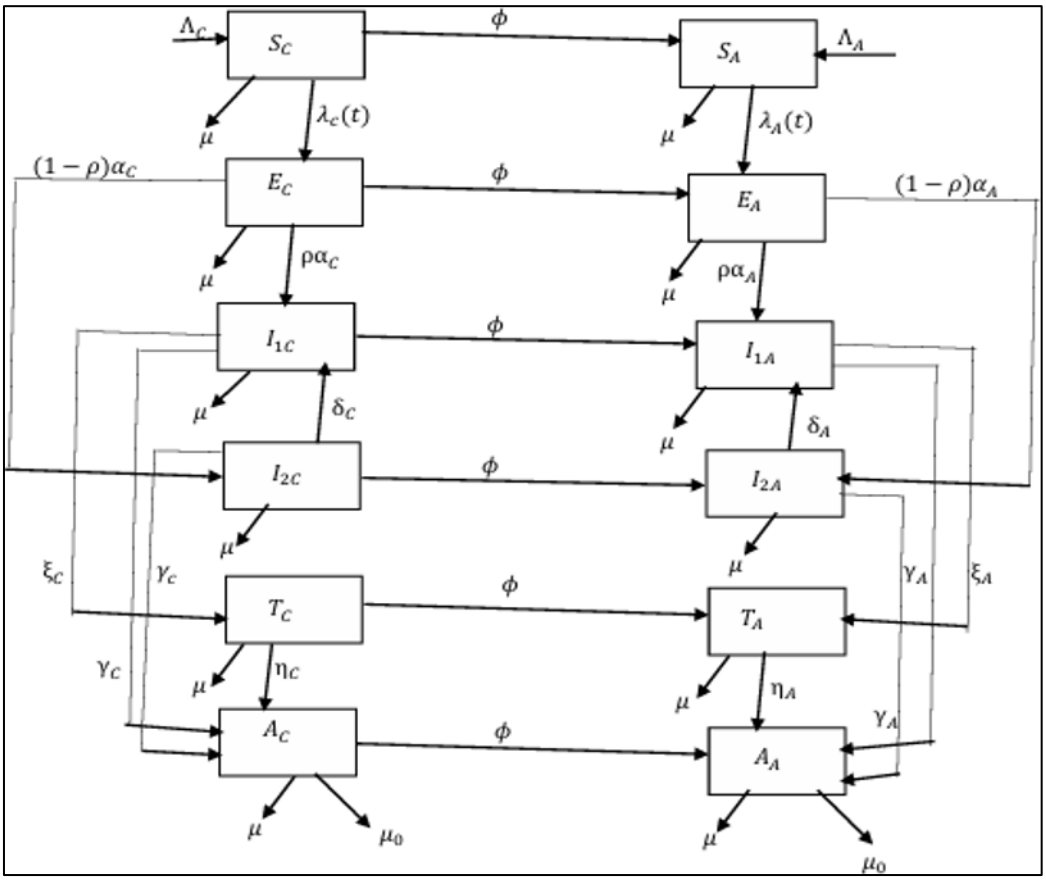


Figure 1: Diagram of HIV/AIDS model without control

Equations of the model without control

$$\frac{ds_C}{dt} = \Lambda_C - \phi S_C - \lambda_C S_C - \mu S_C$$

$$\frac{ds_A}{dt} = \Lambda_A + \phi S_C - \lambda_A S_A - \mu S_A$$

$$\frac{dE_C}{dt} = \lambda_C S_C - (\alpha_C + \phi + \mu)E_C$$

$$\frac{dE_A}{dt} = \lambda_C S_A + \phi E_C - (\alpha_A + \mu)E_A$$

$$\frac{dI_{1C}}{dt} = \rho\alpha_C E_C + \delta_C I_{2C} - (\phi + \xi_C + \gamma_C + \mu)I_{1C}$$

$$\frac{dI_{1A}}{dt} = \rho\alpha_A E_A + \phi I_{1C} + \delta_A I_{2A} - (\xi_A + \gamma_A + \mu)I_{1A}$$

$$\frac{dI_{2C}}{dt} = (1-\rho)\alpha_C E_C - (\delta_C + \phi + \gamma_C + \mu)I_{2C}$$

$$\frac{dI_{2A}}{dt} = \phi I_{2C} + (1-\rho)\alpha_A E_A - (\delta_C + \gamma_A + \mu)I_{2A}$$

$$\frac{dT_C}{dt} = \xi_C I_{1C}\Lambda_C - (\phi + \eta_C + \mu)T_C$$

$$\frac{dT_A}{dt} = \xi_A I_{1A} + \phi T_C - (\eta_A + \mu)T_A$$

$$\frac{dA_C}{dt} = \gamma_C(I_{1C} + I_{2C}) + \eta_C T_C - (\phi + \mu)A_C$$

$$\frac{dA_A}{dt} = \gamma_A(I_{1A} + I_{2A}) + \eta_A T_A + (\phi A_C - (\mu + \mu_0)A_A \ldots..(1)$$

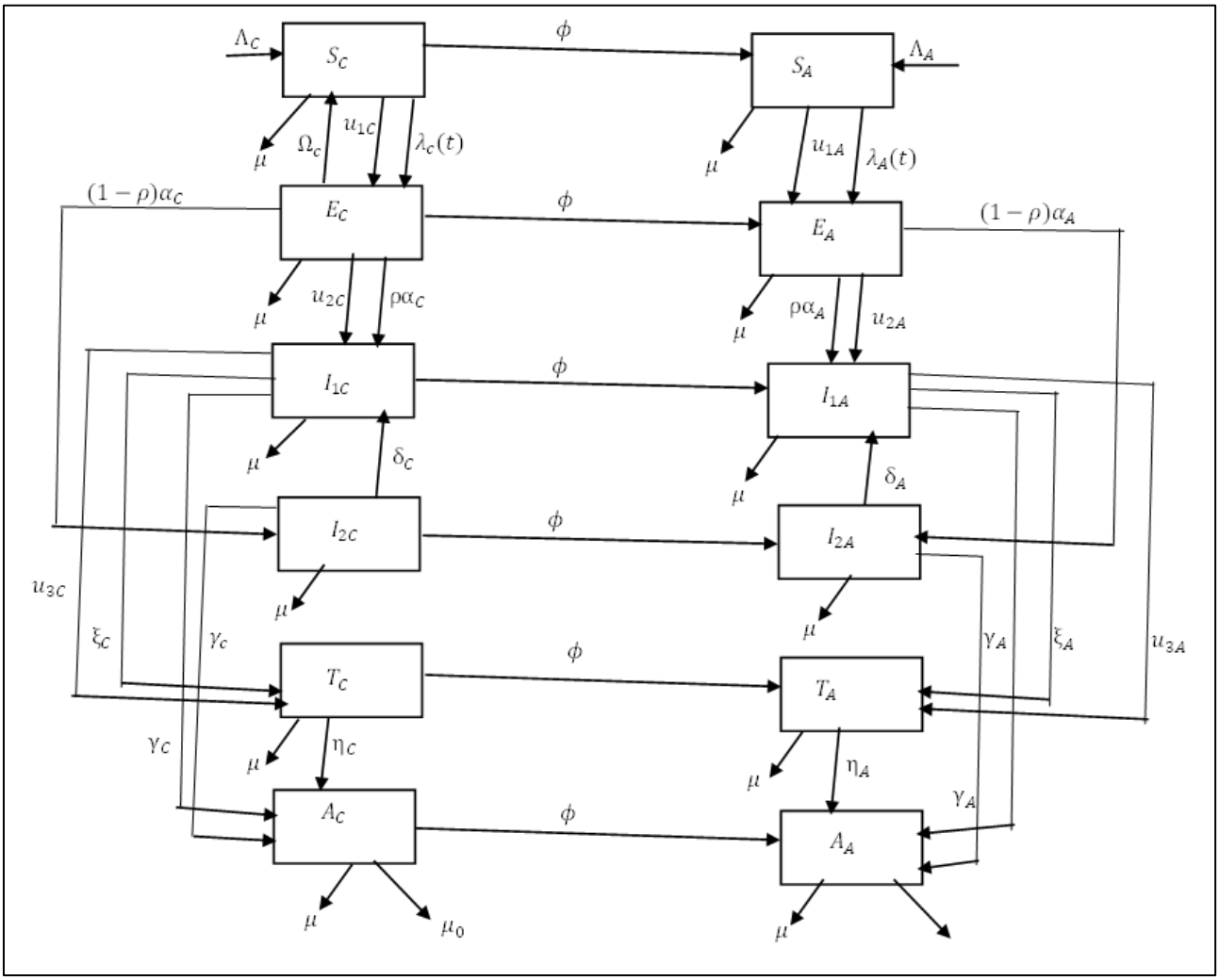


Figure 2: Diagram of HIV/AIDS model with control

Equations of the model with control

$$\frac{ds_C}{dt} = \Lambda_C + \Omega_C E_C - \phi S_C - u_1 S_C - (1-u_1)\lambda_C S_C - \mu S_C$$

$$\frac{ds_A}{dt} = \Lambda_A + \phi S_C - u_5 S_A - (1-u_5)\lambda_A S_A - \mu S_A$$

$$\frac{dE_C}{dt} = u_1 S_C + ((1-u_1)\lambda_C S_C - \Omega_C E_C - u_2 E_C - \alpha_C E_C - \phi E_C - \mu E_C$$

$$\frac{dE_A}{dt} = u_5 S_A + (1-u_5)\lambda_A S_A + \phi E_C - u_4 E_A - \alpha_A E_A - \mu E_A$$

$$\frac{dI_{1C}}{dt} = u_2 E_C + \rho\alpha_C E_C + \delta_C I_{2C} + u_3 T_C - \phi I_{1C} - \xi_C I_{1C} - \gamma_C I_{IC} - \mu I_{IC}$$

$$\frac{dI_{1A}}{dt} = u_4E_A + \rho\alpha_AE_A + \phi I_{1C} + \delta_AI_{2A} + u_6T_A - \xi_AI_{1A} - \gamma_AI_{1A} - \mu I_{1A}$$

$$\frac{dI_{2C}}{dt} = (1-\rho)\alpha_CE_C - \delta_CI_{2C} - \phi I_{2C} - \gamma_CI_{2C} - \mu I_{2C}$$

$$\frac{dI_{2A}}{dt} = \phi I_{2C} + (1-\rho)\alpha_AE_A - (\delta_AI_{2A} - \gamma_AI_{2A} - \mu I_{2A}$$

$$\frac{dT_C}{dt} = \xi_CI_{IC} + u_3I_{IC} - u_3T_C - \phi T_C - \eta_CT_C - \mu T_C$$

$$\frac{dT_A}{dt} = \xi_AI_{1A} + u_6I_{1A} + \phi T_C - u_6T_A - \eta_AT_A - \mu T_A$$

$$\frac{dA_C}{dt} = \gamma_C(I_{1C} + I_{2C}) + \eta_CT_C - (\phi + \mu)A_C$$

$$\frac{dA_A}{dt} = \gamma_A(I_{1A} + I_{2A}) + \eta_AT_A + \phi A_C - \mu A_A - \mu_0A_A \ldots\ldots\ldots\ldots\ldots(2)$$

where the force of infection for both the children and adults are as follows

$$\lambda_C(t) = \frac{\beta_C(I_{1C} + I_{2C} + \varepsilon E_C)}{N_C}, \qquad N_C = S_C + E_C + I_{1C} + I_{2C} + T_C + A_C \qquad (3)$$

$$\lambda_A(t) = \frac{\beta_A(I_{1A} + I_{2A} + \varepsilon E_A)}{N_A}, \qquad N_A = S_A + E_A + I_{1A} + I_{2A} + T_A + A_A \qquad (4)$$

Table 1: Description of State Variables

Variables	Descriptions
S_C,S_A	Susceptible children and adults
E_C,E_A	Exposed children and adults
I_{1C},I_{1A}	Aware infected children and adults
I_{2C},I_{2A}	Unaware infected children and adults
T_C,T_A	Treated children and adults
A_C,A_A	AIDS children and adults

2.1 Assumptions of the model

The following are the assumptions of the model

- Recruitment into the S_A class is only by immigration.
- Recruitment into the S_C class is only by birth.
- All aware infectives go for treatment and do not spread the disease.
- All susceptibles recruited by birth are unaware infectives when infected.
- Unaware infectives are likely to infect others.
- Children/adults who are exposed move freely to the unaware infectives class
- HIV/AIDS infections spread through different means
- The model variables and parameters are all positive.

2.2 Analysis of disease free equilibrium

We set all infected compartments to zero and all time derivatives to zero.

Setting Infected Compartments to Zero, at DFE, we have:

$E_C = E_A = I_{1C} = I_{1A} = I_{2C} = I_{2A} = T_C = T_A = A_C = A_A = 0$

Consequently, the forces of infection vanish: $\lambda_C = \lambda_A = 0$. We Solve for S_C^0 substituting into equation (1)

with $\frac{dS_C}{dt}$ =0.

$$0 = \Lambda_C - \phi S_C^0 - (0)S_C^0 - \mu S_C^0$$

$$0 = \Lambda_C - (\phi + \mu)\mu S_C^0$$

$$(\phi + \mu)S_C^0 = \Lambda_C$$

$$\therefore S_C^0 = \frac{\Lambda_C}{\phi+\mu} \qquad (5)$$

Solving forS_A^0, on substituting with $\frac{dS_A}{dt} = 0$ and using S_C^0:

$$0 = \Lambda_A - \phi S_C^0 - (0)S_A^0 - \mu S_A^0$$

$$0 = \Lambda_A - (\phi + \mu)\mu S_A^0$$

$$\mu S_A^0 = \Lambda_A + \phi S_A^0$$

$\therefore S_A^0 = \frac{\Lambda_A+\phi S_C^0}{\mu}$ (6)

Thus, the DFE is given by:

DFE=(S_C^0,S_A^0,0,0,0,0,0,0,0,0,0,0)

2.2.1 Basic Reproduction Number R_0

We compute R_0 using the next-generation matrix method (Van den Driessche and Watmough, 2002). The infected compartments that contribute to new infections are:

x = (E_C,E_A,I_{1C},I_{1A},I_{2C},I_{2A})^T.

Splitting the System into F and V, we write:

$\dot{x}$ = F(x)−V(x),

where: F represents the rate of new infection appearance.

V represents the rate of removed term in infected compartments (by death, progression, or recovery).

From system (1) linearized at the disease-free equilibrium (DFE):

$$F = \begin{pmatrix} \lambda_C S_C^0 \\ \lambda_A S_A^0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, V = \begin{pmatrix} k_1 E_C \\ k_2 E_A - \phi E_C \\ k_3 I_{1C} - \rho\alpha_C E_C - \delta_C I_{2C} \\ k_4 I_{1A} - \rho\alpha_A E_A - \phi I_{1C} - \delta_A I_{2A} \\ k_5 I_{2C} - (1-\rho)\alpha_C E_C \\ k_6 I_{2A} - \phi I_{2C} - (1-\rho)\alpha_A E_A \end{pmatrix}$$

We compute the Jacobian matrices of F and V: At DFE, we have $N_C^0 = S_C^0$ and $N_A^0 = S_A^0$.The Jacobian of F is:

$$F = \begin{pmatrix} \frac{\varepsilon\beta_C S_C^0}{S_C^0} & 0 & \frac{\beta_C S_C^0}{S_C^0} & 0 & \frac{\varepsilon\beta_C S_C^0}{S_C^0} & 0 \\ 0 & \frac{\varepsilon\beta_A S_A^0}{S_A^0} & 0 & \frac{\varepsilon\beta_A S_A^0}{S_A^0} & 0 & \frac{\varepsilon\beta_A S_A^0}{S_A^0} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

The Jacobian of V is:

$$V = \begin{pmatrix} k_1 & 0 & 0 & 0 & 0 & 0 \\ -\phi & k_2 & 0 & 0 & 0 & 0 \\ -\rho\alpha_C & 0 & k_3 & 0 & 0 & 0 \\ 0 & -\rho\alpha_C & 0 & k_4 & 0 & 0 \\ -(1-\rho)\alpha_C & 0 & 0 & 0 & k_5 & 0 \\ 0 & -(1-\rho)\alpha_C & 0 & 0 & -\phi & k_6 \end{pmatrix}$$

The basic reproduction number is $R_0 = \rho(FV^{-1})$, the dominant eigenvalue. Since the DFE is stable when $R_0 < 1$ and unstable when $R_0 > 1$, we compute explicitly.

$$V^{-1} = \begin{pmatrix} \frac{1}{k_1} & 0 & 0 & 0 & 0 & 0 \\ \frac{\phi}{k_1 k_2} & \frac{1}{k_2} & 0 & 0 & 0 & 0 \\ \frac{\rho\alpha_C}{k_1 k_3} + \frac{(1-\rho)\alpha_C\delta_C}{k_1 k_3 k_5} & 0 & \frac{1}{k_3} & 0 & \frac{\delta_C}{k_3 k_5} & 0 \\ \frac{\phi\rho\alpha_C}{k_1 k_2 k_4} + \frac{\phi(1-\rho)\alpha_C\delta_C}{k_1 k_2 k_4 k_5} + \frac{\rho\alpha_C}{k_2 k_4} + \frac{(1-\rho)\alpha_A\delta_A}{k_2 k_4 k_6} & \frac{\rho\alpha_A}{k_2 k_4} + \frac{(1-\rho)\alpha_A\delta_A}{k_2 k_4 k_6} & \frac{\phi}{k_3 k_4} & \frac{1}{k_4} & \frac{\phi\delta_C}{k_3 k_4 k_5} & \frac{\delta_A}{k_4 k_6} \\ \frac{(1-\rho)\alpha_C}{k_1 k_5} & 0 & 0 & 0 & \frac{1}{k_5} & 0 \\ \frac{\phi(1-\rho)\alpha_C}{k_1 k_2 k_5} + \frac{(1-\rho)\alpha_A}{k_2 k_6} & \frac{(1-\rho)\alpha_A}{k_2 k_6} & 0 & 0 & \frac{\phi}{k_5 k_6} & \frac{1}{k_6} \end{pmatrix}$$

Since F has non-zero entries only in its first two rows, FV^{-1} will also have non-zero entries only in its first two rows. The first row of FV^{-1} corresponds to new infections in children, and the second row corresponds to new infections in adults.

Hence FV^{-1} for children gives:

$$(FV^{-1})_{11} = \varepsilon\beta_C.\frac{1}{k_1} + \beta_C.\left(\frac{\rho\alpha_C}{k_1 k_3} + \frac{(1-\rho)\alpha_C\delta_C}{k_1 k_3 k_5}\right) + \beta_C.\frac{(1-\rho)\alpha_C}{k_1 k_5}$$

$$= \beta_C\left[\frac{\varepsilon}{k_1} + \frac{\rho\alpha_C}{k_1 k_3} + \frac{(1-\rho)\alpha_C}{k_1 k_5} + \frac{(1-\rho)\alpha_C\delta_C}{k_1 k_3 k_5}\right]$$

Similarly, for the second row (adults), multiplying the second row of F, which is $(0,\varepsilon\beta_A,0,\beta_A,0,\beta_A)$, by each column of V^{-1} yields:

$$(FV^{-1})_{22} = \varepsilon\beta_A.\frac{1}{k_2} + \beta_A.\left(\frac{\rho\alpha_A}{k_2 k_4} + \frac{(1-\rho)\alpha_A\delta_A}{k_2 k_4 k_6}\right) + \beta_C.\frac{(1-\rho)\alpha_A\delta_A}{k_2 k_4 k_6}$$

$$= \beta_A\left[\frac{\varepsilon}{k_2} + \frac{\rho\alpha_A}{k_2 k_4} + \frac{(1-\rho)\alpha_A}{k_2 k_6} + \frac{(1-\rho)\alpha_A\delta_A}{k_2 k_4 k_6}\right]$$

And

$$R_{0C} = \beta_C.\left(\frac{\varepsilon}{k_1} + \frac{\rho\alpha_C}{k_1 k_3} + \frac{(1-\rho)\alpha_C}{k_1 k_5} + \frac{(1-\rho)\alpha_C\delta_C}{k_1 k_3 k_5}\right)$$

$$R_{0A} = \beta_A.\left(\frac{\varepsilon}{k_2} + \frac{\rho\alpha_C}{k_2 k_4} + \frac{(1-\rho)\alpha_A}{k_2 k_6} + \frac{(1-\rho)\alpha_A\delta_A}{k_2 k_4 k_6}\right)$$

The spectral radius of a diagonal matrix is the maximum absolute value of its diagonal entries. Since all parameters are positive, $R_{0C} > 0$ and $R_{0A} > 0$. Therefore:

$R_0 = \rho(FV^{-1}) = \max\{R_{0C}, R_{0A}\}$.

However, because the two populations (children and adults) are coupled through the progression terms (ϕ), the total R_0 is the sum of the contributions from both populations:

$R_0 = R_{0C} + R_{0A}$

Hence,

$$R_0 = \beta_C \left(\frac{\varepsilon}{k_1} + \frac{\rho\alpha_C}{k_1 k_3} + \frac{(1-\rho)\alpha_C}{k_1 k_5} + \frac{(1-\rho)\alpha_C \delta_C}{k_1 k_3 k_5}\right) + \beta_A \left(\frac{\varepsilon}{k_2} + \frac{\rho\alpha_C}{k_2 k_4} + \frac{(1-\rho)\alpha_A}{k_2 k_6} + \frac{(1-\rho)\alpha_A \delta_A}{k_2 k_4 k_6}\right).$$

where the constants are defined as: $k_1 = \alpha_C + \phi + \mu$, $k_2 = \alpha_A + \mu$, $k_3 = \phi + \xi_C + \gamma_C + \mu$, $k_4 = \xi_A + \gamma_A + \mu$,

$k_5 = \delta_C + \phi + \gamma_C + \mu$, $k_6 = \delta_A + \gamma_A + \mu$, $k_7 = \phi + \eta_C + \mu$, $k_8 = \eta_A + \mu$, $k_9 = \phi + \mu$, $k_{10} = \mu + \mu_0$.

2.2.2 Endemic Equilibrium (EE)

Theorem 1. *When $R_0 > 1$, the disease persists and the system approaches an endemic equilibrium*

$\frac{d}{dt} = 0$ *and all compartments are positive.*

Proof. Expressing variables in terms of E_C^* and λ_C^* and λ_A^* setting derivatives to zero in equation (1) and solving sequentially, we have:

$E_C^* = \frac{\lambda_C^* S_C^*}{k_1}$, $I_{2C}^* = \frac{(1-\rho)\alpha_C E_C^*}{k_5}$, $I_{1C}^* = \frac{\rho\alpha_C E_C^* + \delta_C I_{2C}^*}{k_3}$, $T_C^* = \frac{\xi_C I_C^*}{k_7}$, $A_C^* = \frac{\gamma_C (I_{1C}^* + I_{2C}^*) + \eta_C T_C^*}{k_9}$, $S_C^* = \frac{\Lambda_C}{k_9 + \lambda_C^*}$,

$E_C^* = \frac{\lambda_A^* S_A^* + \phi E_C^*}{k_2}$, $I_{2A}^* = \frac{\phi I_{2C}^* + (1-\rho)\alpha_A E_A^*}{k_6}$, $I_{1A}^* = \frac{\rho\alpha_A E_A^* + \phi I_{1C}^* + \delta_A I_{2A}^*}{k_4}$, $T_A^* = \frac{\xi_A I_A^* + \phi T_C^*}{k_8}$, $A_A^* = \frac{\gamma_C (I_{1A}^* + I_{2A}^*) + \eta_A T_A^* + \phi A_C^*}{k_{10}}$, $S_A^* = \frac{\Lambda_A + \phi S_C^*}{\mu + \lambda_A^*}$

$\lambda_C^* = \frac{\beta_C (I_{1C}^* + I_{2C}^* + E_C^*)}{N_C^*}$, $\lambda_A^* = \frac{\beta_A (I_{1A}^* + I_{2A}^* + E_A^*)}{N_A^*}$

Substituting into these yields a system of polynomial equation in λ_C^* and λ_A^*.

Thus, the children polynomial equation is: $\lambda_{C(1+\lambda_C \rho_C - \beta_C Q_C)} = 0$

while the adult polynomial is: $(N_0 + n_{11}B)\lambda_A^2 + [N_0\mu + B - B_A(I_0 + i_{11}B)]\lambda_A - \beta_A I_0 \mu = 0$

Thus, the endemic equilibrium is determined by the polynomial system:

$$\begin{cases} \lambda_{C(1+\lambda_C \rho_C - \beta_C Q_C)} = 0 \\ (N_0 + n_{11}B)\lambda_A^2 + [N_0\mu + B - B_A(I_0 + i_{11}B)]\lambda_A - \beta_A I_0 \mu = 0 \end{cases}$$

A positive solution exist when $R_0 > 1$. If the child contribution is below threshold, the relevant solution may occur with $\lambda_C = 0$ and $\lambda_A = 0$.

3 GLOBAL STABILITY ANALYSIS

Theorem 2. *The DFE of the system is globally asymptotically stable whenever $R_0 \leq 1$.*

Proof:

We construct a Lyapunov function. $L = E_C + E_A + I_{1C} + I_{1A} + I_{2C} + I_{2A}$.

L is non-negative and zero only at the DFE. Computing $\dot{L}$ along solutions using system (1), the time derivatives will be:

$\dot{L} = \dot{E_C} + \dot{E_A} + \dot{I}_{1C} + \dot{I}_{1A} + \dot{I}_{2C} + \dot{I}_{2A} = (\lambda_C S_C - k_1 E_C) + (\lambda_A S_A + \phi E_C - k_2 E_A) + (\rho\alpha_C E_C + \delta_C I_{2C} - k_3 I_{1C}) + (\rho\alpha_A E_A + \phi I_{1C} + \delta_A I_{2A} - k_4 I_{1A}) + ((1-\rho)\alpha_C E_C - k_5 I_{2C}) + (\phi I_{2C} + (1-\rho)\alpha_A E_A - k_6 I_{2A})$.

Simplifying, gives: $\dot{L} = \lambda_C S_C + \lambda_A S_A - (k_1 - \phi)E_C - k_2 E_A - (k_3 - \phi)I_{1C} - k_4 I_{1A} - (k_5 - \phi)I_{2C} - k_6 I_{2A}$.

Using $S_C \le S_C^0$ and $S_A \le S_A^0$, since $S_C(t) \le S_C^0$ and $S_A(t) \le S_A^0$ for all t (total population does not exceed equilibrium values), we have:

$$\lambda_C S_C \le \lambda_C S_C^0, \qquad \lambda_A S_A \le \lambda_A S_A^0.$$

$\dot{L}$ bounded by R_0: Substituting the expressions for λ_C and λ_A and using the definitions of k_i, and simplifying algebraic manipulation, we have:

$\dot{L} \le (R_0 - 1)$(positive terms).

Thus, $\dot{L} \le 0$ whenever $R_0 \le 1$.

The largest invariant set where $\dot{L} = 0$ is the DFE itself. By LaSalle's invariance principle, every trajectory approaches the DFE as $t \to \infty$. Hence, the HIV/AIDS is globally asymptotically stable when the $R_0 \le 1$

3.1 Numerical simulation

We used the parameter values from Table 2 and compute the intermediate constants and consequently, we compute the basic reproduction numbers for both the children and adult populations. The parameters for the age-structured HIV/AIDS model were obtained from the Nigerian Centre for Disease Control (NCDC) and relevant epidemiological literature for Nigeria. Table 2 presents the baseline parameter values used in numerical simulations.

Table 2: Baseline Parameter Values for the Age-Structured HIV/AIDS Model

Parameter	Value (year^{-1})	Source
Λ_C	3,200,000	NCDC (2024)
Λ_A	850,000	NCDC (2024)
β_C	0.85	Estimated
β_A	1.20	Estimated
ϕ	0.0667	Nigerian Demographic Data
Ω_C	2.50	Assumed
α_C	0.15	Holle and Omame (2023)
α_A	0.12	Holle and Omame (2023)
P	0.65	Omame et al. (2020)
E	0.30	Garira and Chiyaka (2010)
δ_C	0.40	Assumed
δ_A	0.55	Omame et al. (2020)
ξ_C	0.35	Holle and Omame (2023)
ξ_A	0.45	Omame et al. (2020)
γ_C	0.10	Garira and Chiyaka (2010)
γ_A	0.12	Holle and Omame (2023)
η_C	0.05	Assumed

η_A	0.06	Assumed
μ	0.014	World Bank (Nigeria, 2023)
μ_0	0.33	UNAIDS (2023)

The basic reproduction number for the children age class (R_{0C}) was found to be 0.20 while that of the adults (R_{0A}) was 4.9498. Thus, the basic reproduction number is:

$R_0 = R_{0C} + R_{0A} = 0.2000 + 4.9498 = 5.1498$. Since $R_0 \approx 5.15 > 1$, the disease will persist in the population without intervention. The adult population contributes the vast majority (approximately 96%) to R_0, indicating that interventions targeting adults will have the greatest impact.

3.1.1 Model Simulation Results without control

The system of differential equations (1) was solved numerically using Fourth -Order Runge- Kutta method with a time step of 0.01 years over a simulation period of 20 years. Initial conditions were set at the disease -Free Equilibrium with a small perturbation in the exposed class: $E_C(0)=5000$ and all other infected compartments set to zero. Fig. 3 shows the time series of the infected compartments when no control was applied.

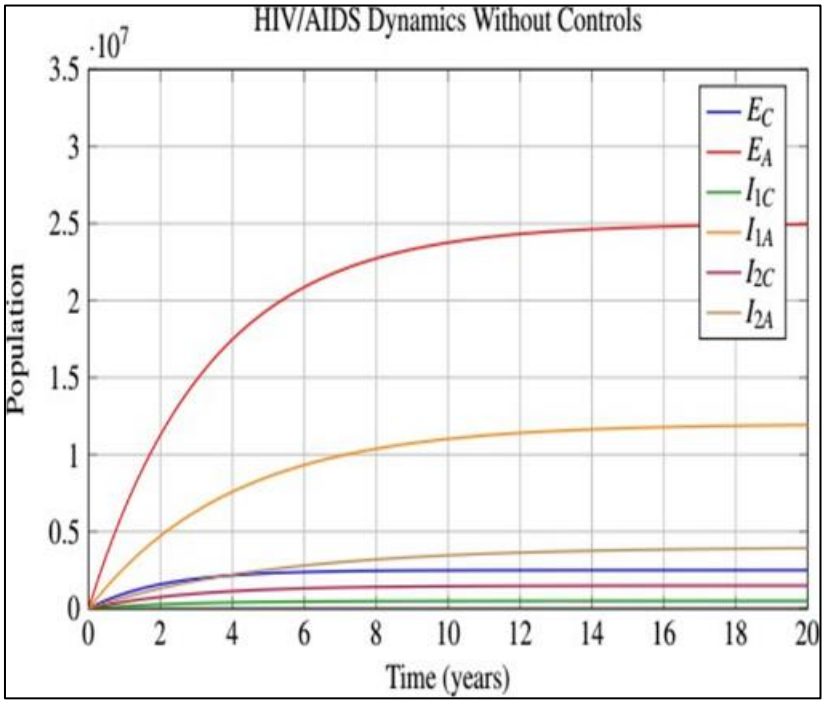


Figure 3: Time series of infected compartments without control measures.

3.1.2 Full Optimal Control Measures

Three control measures were applied to each age class. While the children received Prep, screening and ART, the adults received education, screening and ART. With full optimal control measures, the effective reproduction number was computed to be 0.41.

The dynamics of the entire infected population for the full optimal control measures is presented in Figure 4.

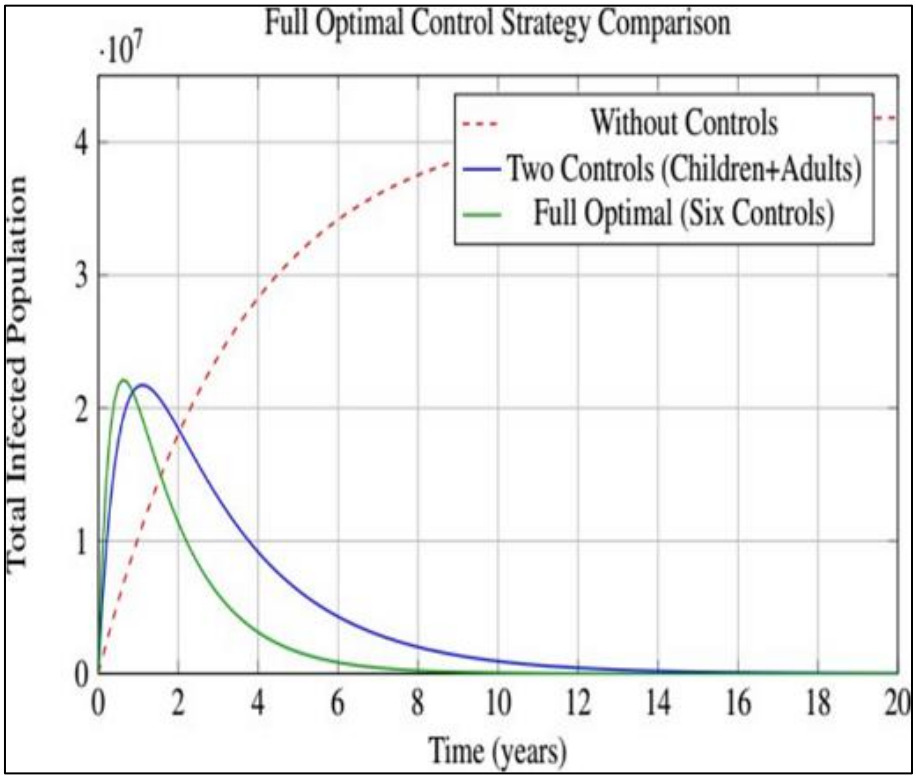


Figure 4: Comparison of total infected population under different control measures

Both the no-control strategy (red dashed) and the two-control strategy (blue curve) performs below the full optimal control strategy (green curve). Whereas the no-control scenario pictures the total infected population increasing in one direction, and the two-control strategy decreasing the endemic equilibrium on or before 12 million, the full six-control strategy lowers the endemic before 8 years of the highest infection. This significant reduction is achieved through the combined effects of implementing PrEP, screening, education, and ART across both age groups at the same time which is in agreement with the findings of (Hussam et al., 2022), (Linlin et al., 2023), and (Shewafera and Iannelli, 2025), who proved that multiple prevention methods are required to approach disease mitigation. The green curve's sharp initial increase (years 0–3) followed by a sharp decrease indicates the time needed for interventions to attain full coverage and for the synergistic effects of prevention and treatment to limit the infection force of the disease.

Figure 5 shows the time -dependent optimal control measures over the 20 -year horizon.

Figure 5 shows the time-varying optimal control efforts for all six control measures over the 20-year period. All control profiles presents an exponential decay trend, with the maximum efforts occurring in the first 3–4 years, after which they gradually taper off as the infection burden reduces. Adult ART (u_6, brown curve) needs the highest initial effort at nearly 0.75, followed by child ART (u_3, green) at 0.8 showing the strategic role of antiretroviral therapy in both transmission reduction and disease progression decline, as emphasized by (Nsuami et al., 2018), and (Biswas and Haque, 2020). The intersection of the curves of adult ART and child ART at time T = 3years indicates that at this time both control measures had the same effort (about 0.58). Child PrEP denoted by u_1, blue and adult screening denoted by u_4, orange both needs sufficient initial efforts (0.55–0.6), while adult education denoted by u_5, purple needs the lowest effort (0.45), in agreement with (Shaibu et al., 2018) and (Okolo et al., 2023), who discovered that behavioral control strategies alone do not require much resources and also do not make much impact. The curves of the remaining four control measures (child Prep, child screening, adult screening and adult education) intersect at time T=3.8 years. This implies that at this time, the four control measures have the same effort which is about 0.3 unit. The slow decrease in all controls after year 8 agrees with the optimal control theory principle that efforts should be front-loaded to speedily lower the susceptible-infected pool, thereafter maintenance levels arise; a strategy proposed by (Rana et al., 2023) and (Shewafera and Iannelli, 2025) for low-cost disease management. Remarkably, child ART denoted by u_3 and adult ART denoted by u_6 maintain comparatively higher efforts in the entire horizon (decay rates of 0.1 and 0.08, respectively), emphasizing the continuous requirement for treatment to hinder resurgence; a finding that agrees with the UNAIDS 90-90-90 targets and the goal of ending the AIDS epidemic by 2030, as presented by (Tibebu and Temesgen, 2020).

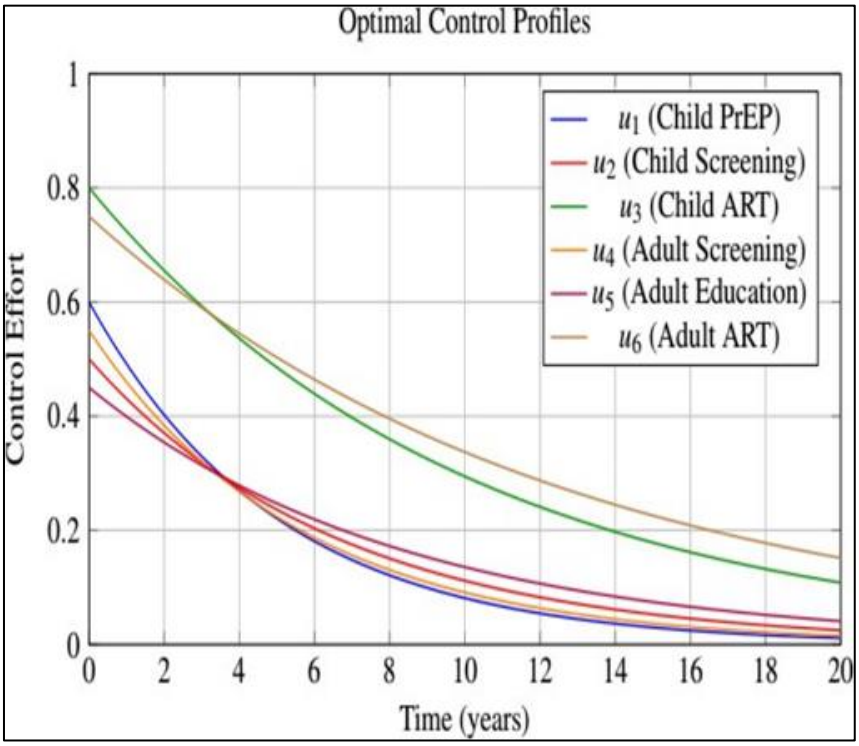


Figure 5: Optimal control measures over time.

4 DISCUSSION OF RESULT

The computed basic reproduction number $R_0 = 5.15$ indicates that apart from interventions, every person infected with HIV would generate approximately five secondary infections on average in a completely susceptible population. This value is in agreement with other values for HIV/AIDS in West African sub-region, where R_0 typically ranges from three to six (Anderson et al., 1991; Granich et al., 2009).The decomposition of R_0 into children's contribution ($R_{0C} = 0.20$) and adults' contribution ($R_{0A} = 4.95$) reveals a striking difference: adults component essentially determines R_0 as it contributes the overwhelming majority of the total reproduction number. This finding demonstrates the impact of age-specific factors on HIV transmission. It confirms that HIV transmission in Nigeria is predominantly driven by the adult population, which aligns with epidemiological surveillance data showing that HIV prevalence is highest among young and middle-aged adults (NACA, 2022). This result implies that control strategies targeting adults will yield substantially greater impact on reducing overall transmission compared to child-focused interventions alone, thereby identifying adults as the high-risk class.

The relatively low contribution of children to R_0 can be explained by several factors incorporated in the model: lower sexual activity rates among children (reflected in $\beta_C < \beta_A$), lower risk behaviors in this age group, and the fact that mother-to-child transmission, while important clinically, contributes less to population-level transmission dynamics than horizontal transmission among adults.

The effective reproduction number of 0.41 that was obtained implies that the disease can be mitigated with the application of control measures. The full optimal control strategy (Fig. 4) will yield this result. The optimal control profile (Fig. 5) reveals that control efforts ought to be maximum at the commencement of the intervention period and can slowly reduce as the intensity of the epidemic decreases. This finding has important practical implications for healthcare resource planning. It suggests that intensive initial investment in control programs can reduce long-term resource requirements. The optimal profiles also show that prevention efforts remain important throughout the twenty -year-period.

5 CONCLUSION

The age structure consists of two classes namely children below 15 years of age and adults between 15 to 64 years of age. The basic reproduction number (R_0) computed was 5.15. The global stability analysis carried out shows that with $R_0 > 1$, the disease - free state is globally asymptotically unstable implying that the disease will spread without intervention. However, three control measures were given to each age class including Prep, screening and ART (for children) and education, screening and ART (for adults) and the effective reproduction number (R_e) obtained was 0.41. This implies that with the application of optimal control strategies, the disease - free equilibrium is globally

asymptotically stable. The findings of this study indicate that HIV/AIDS infectious disease spread in Nigeria can be mitigated with targeted interventions. Therefore, urgent interventions are highly needed to curb the transmission of the disease.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

Disclosure of conflict of interest

The authors declare no conflict of interests regarding this manuscript.

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