

Optimal Control Analysis of HIV/AIDS Model with PrEP Strategy and Behavioral Changes

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ABSTRACT Combining Pre-Exposure Prophylaxis (PrEP) with behavioral strategies has become vital in curbing HIV/AIDS transmission, offering a pathway to reduce infections and allocate health resources more effectively. In this study, we developed a six-compartment mathematical model to analyze the spread and control of HIV/AIDS. Through rigorous analysis, we calculated the basic reproduction number (R_0) to assess transmission potential and evaluated the stability of the disease-free equilibrium state. To identify optimal strategies for reducing HIV/AIDS prevalence, we designed an optimal control framework using Pontryagin's Minimum Principle. This approach allowed us to evaluate the effectiveness of prevention tactics such as public health education, condom use, screening, and treatment both individually and in combination. Our findings highlight that pairing community awareness campaigns with increased condom usage among adults, alongside proactive screening and treatment programs, significantly reduces new infections and overall prevalence. Numerical simulations further demonstrated how these measures lower infection rates, aligning with our theoretical predictions. These insights emphasize the importance of integrated, multi-pronged interventions to guide public health policies and resource distribution in the fight against HIV/AIDS.

KEYWORDS: Optimal control, Stability, Equilibrium, Basic reproduction number.

I. INTRODUCTION

HIV originated from a virus found within chimpanzees' population in West Africa in the decade of the 1930s and was first transmitted to humans by blood exposure during hunting. Over the years, the virus feasted across Africa, and eventually to various regions of the world [1]. Today, HIV remains a significant global public health challenge, claimed approximately 40.4 lives. The virus continues to spread in every country, with some regions even seeing an increase in new infections. At the close of 2022, about 39 million people were alive with HIV, with two-thirds of them around 25.6 million located in African Region. That same year, around 630,000 individuals pass away from HIV-related causes, and 1.3 million individuals were newly diagnosed with the virus. Organizations like WHO, the Global Fund, and UNAIDS are working together on global strategies to tackle HIV, all aimed at achieving the Sustainable Development Goal of ending the epidemic by 2030. Encouragingly, among those living with HIV, about 86% recognized their status, 76% were getting antiretroviral therapy, and 71% had curbed viral loads. [1], [2]. Unfortunately, there is currently no cure for HIV, and managing the epidemic is still a work in progress [3]. However, effective prevention, diagnosis, treatment, and care options, including the use of PrEP and antiretroviral therapy (ART), have made a significant difference in slowing the infection's progression. Without treatment, HIV can advance to an extreme stage known as acquired immunodeficiency syndrome (AIDS).

Thanks to medical advances, HIV is now considered a controllable chronic health state, allowing many individuals active with HIV to lead hale and healthy lives. The infection advances through several stages: (i) Primary Stage (Asymptomatic Stage): During this initial phase, the virus is present in the blood but cannot be detected by standard medical tests.; (ii) Asymptomatic Stage: This stage is symptom-free but can be diagnosed through a medical test.; (iii) Symptomatic Stage: Here, individuals may begin to experience symptoms such as fatigue, weight loss, and severe dehydration; and (iv) AIDS Stage: This advanced phase makes treatment more challenging and can lead to death without immediate care. HIV is transmitted in several ways, including unsafe sexual practices, exposure to infected blood, and mother-to-child spread during childbirth or breastfeeding. However, adopting safe practices—often summarized as the ABC approach (Abstain, Be faithful, and Use Condoms)—can significantly reduce the risk of transmission. Sadly, people living with HIV often face higher rates of other infectious diseases and significant barriers to accessing preventive measures and care. Active interventions and healthcare service area are essential to tackling the community health issues surrounding HIV. Unfortunately, proscribing drug use often traps people in a cycle of confinement, which can worsen health hazards for HIV, tuberculosis (TB), and other ailments—particularly amid susceptible populations like individuals who give a jab (drugs) (PWID). The landscape of Pre-Exposure Prophylaxis (PrEP) has undergone significant changes since the original brief was published, [4,5] particularly in terms of its availability and variety, with over 75% of usage occurring in the African region. PrEP is a daily pill taken by HIV-negative persons to prevent HIV infection; its main job is to stop HIV from replicating in the body. Medications like tenofovir/emtricitabine are effective at inhibiting this replication. PrEP is highly active up to 99% in preventing HIV infection when used constantly [6]. It provides protection even if other prevention means, such as condoms, flop or are not used, and it significantly decreases transmission hazard, especially for high-risk groups like sexually energetic individuals and jab drug users. PrEP comes in the form of oral pills or injectables and is recommended by the World Health Organization (WHO) as a protective measure for those who might be exposed to HIV [4, 5].

In recent years, optimal control analysis of HIV/AIDS models has been an active area of research, aiming to identify the most effective control strategies to mitigate the spread of the disease. Several studies have incorporated various factors, including the variable flow of infectious immigrants [7-13], Pre-Exposure Prophylaxis (PrEP) [14-19], and behavioural change [20-22], to better represent the complex dynamics of HIV transmission. One of the studies on optimal control of HIV/AIDS models was conducted by [23], which considered a basic model with a single control variable representing treatment. The authors used Pontryagin's Maximum Principle to achieve the optimal control policy, which was found to be a bang-bang control. Later, [22] their work incorporated Treatment and behavioural change into the model. The optimal control strategy involves a combination of treatment and behavioural change. The impact of immigration on HIV transmission has also been studied in the context of optimal control. [20] developed a model that accounts for the variable flow of infectives and immigrants, and found that the optimal control policy involves a combination of behavioural change, screening and treatment. Similarly, behavioural change has been recognized as a crucial factor in HIV transmission, and several Studies have incorporated it into optimal control models. [24] developed a model that accounts for behavioral change through education and awareness campaigns, and found that the optimal control policy involves a combination of education and treatment. Similarly, [21] considered a model with behavioural change, intake of a balanced nutritional diet and treatment, and found that the optimal control strategy involves a combination of triple control to ensure eradication. [6], proposed a six-compartmental mathematical model that combines PrEP as a control measure to regulate the spread of HIV/AIDS in a population. The result highlights the position of targeted interventions in realizing a disease-free environment. They recommended based on their findings, daily pre-exposure Prophylaxis (PrEP) for prone populations.

This study [25] uses mathematical modeling to evaluate how effective Pre-Exposure Prophylaxis (PrEP) is in averting HIV transmission and controlling its spread at different phases of infection. The results highlight

the significance of targeted interventions in creating a disease-free environment. The research also pinpointed key factors that significantly influence the dynamics of HIV, while noting that increasing these factors could lead to a higher basic reproduction number (R_0). The authors strongly recommend the daily use of PrEP for those at higher risk of HIV infection, including vaccinated individuals. This research aims to address critical gaps regarding the impact of public sensitization, condom usage, and screening of new arrivals, as well as the efficacy of antiretroviral drugs, the combination of PrEP treatment, and behavioral changes on disease incidence and optimal control variables. Additionally, it will examine the influence of HIV-infected immigrants on HIV/AIDS transmission. Building on [25]'s work about PrEP's role in achieving disease-free stability, this study will explore how these control strategies affect HIV/AIDS dynamics in the context of immigration of HIV-infected individuals. In Section II we have the epidemic model with control and the methodologies adopted. Section III presents numerical simulations to examine optimality, followed by a detailed discussion of the results. The paper concludes with a summary of the findings in Section IV.

II. MATERIALS AND METHODS

A. The HIV/AIDS Epidemic Model with Controls

In this section, we will explore the optimal control approaches within the model to determine the most effective approach for reducing the number of people infected with HIV/AIDS. Our goal is to identify the

optimal values $u^* = (u_1^*, u_2^*, u_3^*, u_4^*)$ of the controls $u = (u_1, u_2, u_3, u_4)$ and the intervention time interval $[0, t_f]$ and also minimize the objective function. The control $u_1(t)$ represents the efforts to prevent HIV/AIDS infection through public sensitization and condom usage, while $u_2(t)$ represents the decision to seek a PrEP strategy. The control to increase the screening/treatment of infected individuals is $u_3(t)$. the efficacy of anti-retroviral, on the treatment of HIV/AIDS infected individuals $u_4(t)$.

The model denoted the total human population at time t , by $N(t)$, stratified into six classes namely; the susceptible class, $S(t)$; the class of susceptible individuals on the PrEP strategy, $P(t)$; the asymptomatic infected individuals in the primary stage, $I_a(t)$; the symptomatic infected individuals in the pre-AIDS stage, $I_s(t)$; the infected individuals who are receiving treatment, $T(t)$; the infected individuals with full-blown AIDS, $A(t)$. The total population per unit time is $N(t)$. We consider the following system of six nonlinear differential equations:

$$\left. \begin{aligned}
 \frac{dS}{dt} &= \pi + (1 - \varepsilon(1 - u_3))m + \omega P - \lambda(1 - u_1)S - (\theta u_2 + \mu)S \\
 \frac{dP}{dt} &= \theta u_2 S - (\omega + \mu)P \\
 \frac{dI_a}{dt} &= \lambda(1 - u_1)S + \varepsilon m(1 - u_3) - (\phi + \mu)I_a \\
 \frac{dI_s}{dt} &= \phi I_a - (\tau u_4 + \mu)I_s \\
 \frac{dT}{dt} &= \gamma \tau u_4 I_s + \sigma A - (\kappa + \mu)T \\
 \frac{dA}{dt} &= (1 - \gamma)\tau u_4 I_s + \kappa T - (\sigma + \delta + \mu)A
 \end{aligned} \right\} \quad (1)$$

$$\lambda = \left(\frac{\beta_1 I_a}{1 + \eta_1 I_a} + \frac{\beta_2 I_s}{1 + \eta_2 I_s} + \frac{\beta_3 T}{1 + \eta_3 T} \right)$$

Table 1: The following notations are used in the course of this work

Parameters	Interpretation	Value	Reference
π	the recruitment rate	500	Assumed
m	the immigrant population	500	Assumed
ε	infected immigrant	0.05	[6]
μ	Natural mortality rate	0.0084-0.01243	[26]
δ	disease-induced death rate	0.952	Assumed
$\beta_1, \beta_2, \beta_3$	contact rate of I_a, I_s and T respectively	0.18, 0.062, 0.062	Assumed
η_1, η_2, η_3	inhibitory effect of I_a, I_s and T respectively	0.1, 0.05, 0.08	Assumed
c	number of sexual partners	3	[6]
ω	progression from Prep to Susceptible	0.45	Assumed
κ	fraction of T that moves to A	0.08	[6]
τ	the rate of movement in the pre-AIDS class	0.05	[6]
γ	fraction of I_a that moves to A	0.46	Assumed
ϕ	progression of I_a to I_s	0.06	Assumed
θ	fraction of S using Prep	0.43	Assumed
σ	fraction of A that moves to T	0.45	Assumed

B. Model Assumptions

The model we developed is based on the following assumptions:

- Individuals infected with HIV without AIDS symptoms I_a and I_s , those who do not take ART treatment progress to AIDS class A.
- All individuals involved in this study are assumed to have the same natural death rate.
- HIV/AIDS is transmitted through sexual intercourse or infection from infected needles and infected blood.
- The population we are studying is diverse and changes over time.
- There is an inflow of new members π , who are susceptible and m , immigrant into the population per time.
- Only individuals who are HIV-positive and exhibit symptoms of AIDS, referred to as A experience deaths due to AIDS at a rate of δ .

The total population size at time t represented as $N(t)$ can be determined from the model described

above.

$$N(t) = S(t) + P(t) + I_a(t) + I_s(t) + T(t) + A(t) \quad (2)$$

The objective function outlines the optimization problem we are focusing on, specifically aimed at identifying resourceful control strategies. Our goal is to minimize the number of susceptible and infected individuals while keeping costs as low as possible over a defined period, from 0 to t_f . The function is given by

$$F = (u_1, u_2, u_3, u_4) \\ = \int_0^{t_f} \left[A_1 S + A_2 P + A_3 I_a + A_4 I_s + A_5 A + A_6 T + \frac{1}{2} (B_1 u_1^2 + B_2 u_2^2 + B_3 u_3^2 + B_4 u_4^2) \right] dt \quad (3)$$

We approach modeling the control efforts by using a linear combination of different strategies $u_i^*(t)$ ($i = 1, 2, \dots, 4$). The constants A_i ($i = 1, 2, \dots, 6$) and B_i ($i = 1, 2, \dots, 4$) are weight constants that help balance each term in the integrand, ensuring that no single term dominates. The terms $B_1 u^2$, $B_2 u^2$, $B_3 u^2$ and $B_4 u^2$ represent the costs associated with public sensitization condom usage, the establishment of Prep strategy, Screening of new arrivals and the cost effect of anti-retroviral drugs, respectively. The problem is to find optimal functions $(u_1^*, u_2^*, u_3^*, u_4^*)$ such that

$$F(u_1^*, u_2^*, u_3^*, u_4^*) = \min F(u_1, u_2, u_3, u_4), (u_1, u_2, u_3, u_4) \in U \quad (4)$$

where the control set is defined as $U = (u_1, u_2, u_3, u_4) | u_i(t)$

Is Lebesgue measurable on $[0, 1]$,

$$0 \leq u_1(t), u_2(t), u_3(t), u_4(t) \leq 1, t \in [0, t_f],$$

subject to the system described in (1) along with appropriate initial conditions. To tackle this optimal control problem, we will use the Pontryagin's Minimum Principle [27]. Before we derive the optimality, system and demonstrate the presence of an optimal control for the system, we first examine the model using constant controls.

C. Analysis of the Model with Constant Controls

Disease free Equilibrium

In this section, we will identify the steady-state solutions, assess their stability, and calculate the basic reproductive number of the system (1). Hence, HIV/AIDS-free equilibrium, which is given by:

$$E^0 = \left\{ \frac{K_2 (\pi + m + m\varepsilon(1 - u_3))}{K_1 K_2 - \omega \theta u_2}, \frac{\theta u_2 (\pi + m + m\varepsilon(1 - u_3))}{K_1 K_2 - \omega \theta u_2}, 0, 0, 0, 0 \right\} \quad (5)$$

where $K_1 = \theta u_2 + \mu$, $K_2 = \omega + \mu$.

Basic Reproduction Number

The basic reproduction number, denoted by R_0 , using the Next Generation Matrix, associated with the system (1), is given by

$$R_0 = \frac{(1 - u_1) ((\beta_1 \tau u_4 + \beta_1 \mu + \phi \beta_2) K_6 + \beta_3 K_5 \tau u_4 \phi) K_2 (m\varepsilon(1 - u_3) - K_3)}{K_4 K_6 (\tau u_4 + \mu) (\omega \theta u_2 - (\theta u_2 + \mu))} \quad (6)$$

where $K_2 = \omega + \mu$, $K_3 = \pi + m$, $K_4 = \phi + \mu$, $K_5 = \delta \gamma + \gamma \mu + \sigma$, $K_6 = \delta \kappa + \delta \mu + \kappa \mu + \mu^2 + \mu \sigma$.

The threshold R_0 known as the basic reproduction number, signifies the average number of secondary

infections that occur when one infected individual interacts with a fully susceptible population. The controls in our system can significantly impact R_0 . To understand how u_1 , u_2 , u_3 , and u_4 , affect R_0 , a simple computation reveals their effects.

$$\begin{aligned}\frac{\partial R_0}{\partial u_1} &= -\frac{(m\varepsilon(1-u_3)-K_3)((\beta_1\tau u_4+\beta_1\mu+\phi\beta_2)K_6+\beta_3K_5\tau u_4\phi)K_2}{K_4K_6(\tau u_4+\mu)(\omega\theta u_2-(\theta u_2+\mu))} \\ \frac{\partial R_0}{\partial u_2} &= -\frac{(1-u_1)(m\varepsilon(1-u_3)-K_3)((\beta_1\tau u_4+\beta_1\mu+\phi\beta_2)K_6+\beta_3K_5\tau u_4\phi)K_2(\omega\theta-K_2\theta)}{K_4K_6(\tau u_4+\mu)(\omega\theta u_2-(\theta u_2+\mu))^2} \\ \frac{\partial R_0}{\partial u_3} &= -\frac{(1-u_1)(\varepsilon(1-u_3))((\beta_1\tau u_4+\beta_1\mu+\phi\beta_2)K_6+\beta_3K_5\tau u_4\phi)K_2}{K_4K_6(\tau u_4+\mu)(\omega\theta u_2-(\theta u_2+\mu))} \\ \frac{\partial R_0}{\partial u_4} &= -\frac{(1-u_1)(m\varepsilon(1-u_3)-K_3)K_2\tau\phi(K_6\beta_2-\beta_3K_5\mu)}{K_4K_6(\tau u_4+\mu)^2(\theta u_2(\omega-K_2)-K_2\mu)}\end{aligned}\quad (7)$$

Thus:

$$\frac{\partial R_0}{\partial u_1} < 0, \frac{\partial R_0}{\partial u_2} < 0, \frac{\partial R_0}{\partial u_3} < 0, \text{ and } \frac{\partial R_0}{\partial u_4} < 0.$$

From this analysis, we observed that the more effort put into public sensitization as the control function u_1 of the susceptible, seeking of PrEP strategy u_2 in P, Screening of new arrivals in susceptible, and the efficacy of the anti-retroviral drugs in treatment all reduces R_0 . This approach has the potential to be a valuable control strategy and will be further examined using numerical simulations.

Stability Analysis via Disease free equilibrium

The variational matrix Q_0 in relation to E_0 is given by

$$Q_0 = \begin{pmatrix} -K_1 & \omega & 0 & 0 & 0 & 0 \\ \theta u_2 & -K_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & -K_3 & 0 & 0 & 0 \\ 0 & 0 & \phi u_4 & -K_4 & 0 & 0 \\ 0 & 0 & 0 & \gamma\tau u_4 & -K_5 & \sigma \\ 0 & 0 & 0 & (1-\gamma)\tau u_4 & \kappa & -K_6 \end{pmatrix} \quad (8)$$

where $K_1 = \theta u_2 + \mu$, $K_2 = \omega + \mu$, $K_3 = \phi + \mu$, $K_4 = \tau u_4 + \mu$, $K_5 = \kappa + \mu$, $K_6 = \sigma + \delta + \mu$.

Therefore, the eigenvalues of the characteristic equation of $J(E^0)$, determined using Maple 2020 are;

$$\begin{aligned}
\lambda_1 &= -\frac{K_5}{2} - \frac{K_6}{2} + \frac{\sqrt{K_5^2 - 2K_6K_5 + K_6^2 + 4\kappa\sigma}}{2} \\
\lambda_2 &= -\frac{K_5}{2} - \frac{K_6}{2} - \frac{\sqrt{K_5^2 - 2K_6K_5 + K_6^2 + 4\kappa\sigma}}{2} \\
\lambda_3 &= -K_3, \quad \lambda_4 = -K_4 \\
\lambda_5 &= -\frac{K_2}{2} - \frac{K_1}{2} + \frac{\sqrt{K_1^2 - 2K_2K_1 + K_2^2 + 4\theta u_2\omega}}{2} \\
\lambda_6 &= -\frac{K_2}{2} - \frac{K_1}{2} - \frac{\sqrt{K_1^2 - 2K_2K_1 + K_2^2 + 4\theta u_2\omega}}{2}
\end{aligned} \tag{9}$$

Therefore, all the eigenvalues of the characteristic equation $J(E^0)$ are real and negative if $R_0 < 1$. We conclude that the HIV/AIDS-free equilibrium is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. The endemic equilibrium is given by $E^* = (S^*, P^*, I_a^*, I_s^*, A^*, T^*)$ the following:

$$\begin{aligned}
S^* &= \frac{K_2(mu_3\varepsilon - m\varepsilon + \pi + m)}{-\lambda u_1 K_2 - \theta \omega u_2 + K_1 K_2 + \lambda K_2} \\
P^* &= \frac{\theta u_2(mu_3\varepsilon - m\varepsilon + \pi + m)}{-\lambda u_1 K_2 - \theta \omega u_2 + K_1 K_2 + \lambda K_2} \\
I_a^* &= \frac{((-1+u_1)(m+\pi)\lambda + m\varepsilon K_1(-1+u_3))K_2 - m\varepsilon \omega \theta u_2(-1+u_3)}{K_3(((1+u_1)\lambda - K_1)K_2 + \theta \omega u_2)} \\
I_s^* &= \frac{\phi u_4(m\omega \theta u_2 u_3 \varepsilon - K_1 K_2 m u_3 \varepsilon - m\omega \theta u_2 \varepsilon + K_1 K_2 m \varepsilon - K_2 \pi \lambda u_1 - K_2 m \lambda u_1 + K_2 \pi \lambda + K_2 \lambda m)}{K_4 K_3(-\lambda u_1 K_2 - \theta \omega u_2 + K_1 K_2 + \lambda K_2)} \\
A^* &= \frac{\phi \tau (((-m-\pi)\lambda - K_1 m \varepsilon)K_2 + m\omega \theta \varepsilon)((-\gamma+1)K_5 + \gamma \kappa)}{K_4 K_3((-\lambda - K_1)K_2 + \theta \omega)(K_6 K_5 - \kappa \sigma)} \\
T^* &= \frac{((-\gamma+1)\sigma + K_6 \gamma)\phi (((-m-\pi)\lambda - m\varepsilon K_1)K_2 + m\omega \theta \varepsilon)\tau}{K_4 K_3((-\lambda - K_1)K_2 + \theta \omega)(K_6 K_5 - \kappa \sigma)}
\end{aligned} \tag{10}$$

D. Characterization of the Optimal Control

In this section, we will first demonstrate that an optimal control exists for system (1).

Theorem 1. There exist optimal controls $(u_1^*(t), u_2^*(t), u_3^*(t), u_4^*(t))$ and equivalent solutions, $S^*, P^*, I_a^*, I_s^*, A^*, T^*$ that minimize $F(u_1, u_2, u_3, u_4)$ over U .

Proof. The integrand of the objective functional F specified by (3) is a convex function of (u_1, u_2, u_3, u_4) and state system (1) satisfies the Lipschitz property to the state variables since state solutions are bounded. To find an optimal solution,

we have to determine the Lagrangian and Hamiltonian for the problem (1) & (3). The Lagrangian for this control problem can be expressed as follows:

$$L = A_1 S + A_2 P + A_3 I_a + A_4 I_s + A_5 A + A_6 T + \frac{1}{2} (B_1 u_1^2 + B_2 u_2^2 + B_3 u_3^2 + B_4 u_4^2) \quad (11)$$

We should obtain the minimal value of the Lagrangian. Here, the Hamiltonian H for the control problem,

where $\lambda_i, i = 1, 2, 3, 4, 5, 6$ are the adjoint variables, is given by

$$\begin{aligned} H = & A_1 S + A_2 P + A_3 I_a + A_4 I_s + A_5 A + A_6 T + \frac{1}{2} (B_1 u_1^2 + B_2 u_2^2 + B_3 u_3^2 + B_4 u_4^2) + \lambda_2 (\theta u_2 S - (\omega + \mu) P) \\ & + \lambda_1 (\pi + (1 - \varepsilon (1 - u_3)) m + \omega P - \lambda (1 - u_1) S - (\theta u_2 + \mu) S) + \lambda_3 (\lambda (1 - u_1) S + \varepsilon m (1 - u_3) - (\phi + \mu) I_a) \\ & + \lambda_4 (\phi I_a - (\tau u_4 + \mu) I_s) + \lambda_5 (\gamma \tau u_4 I_s + \sigma A - (\kappa + \mu) T) + \lambda_6 ((1 - \gamma) \tau u_4 I_s + \kappa T - (\sigma + \delta + \mu) A) \end{aligned} \quad (12)$$

We will now outline the necessary conditions that the optimal control functions and their corresponding states need to meet, using Pontryagin's Minimum Principle [27].

Theorem 2. Given an optimal control pair $(u_1^*, u_2^*, u_3^*, u_4^*)$ and a solution $(S^*, P^*, I_a^*, I_s^*, A^*, T^*)$ of the corresponding state system (1), there exists adjoint variables $\lambda_i, i = 1, 2, 3, 4, 5, 6$ satisfying

$$\left. \begin{aligned} \frac{d\lambda_1(t)}{dt} &= -A_1 - \lambda_1 (-\lambda (1 - u_1) - K_1) - \lambda_2 \theta u_2 - \lambda_3 \lambda (1 - u_1) \\ \frac{d\lambda_2(t)}{dt} &= \lambda_2 K_2 - \lambda_1 \omega - A_2 \\ \frac{d\lambda_3(t)}{dt} &= \lambda_3 K_3 - \lambda_4 \phi - A_3 \\ \frac{d\lambda_4(t)}{dt} &= -A_4 + \lambda_4 K_4 - \lambda_5 \gamma \tau u_4 - \lambda_6 (-\gamma + 1) \tau u_4 \\ \frac{d\lambda_5(t)}{dt} &= \lambda_6 K_6 - \lambda_5 \sigma - A_5 \\ \frac{d\lambda_6(t)}{dt} &= \lambda_5 K_5 - \lambda_6 \kappa - A_6 \end{aligned} \right\} \quad (13)$$

and the transversality conditions

$$\lambda_1(t_f) = \lambda_2(t_f) = \lambda_3(t_f) = \lambda_4(t_f) = \lambda_5(t_f) = \lambda_6(t_f) = 0 \quad (14)$$

as the terminal conditions at the final time t_f and the optimal control variables $(u_1^*, u_2^*, u_3^*, u_4^*)$ are defined as;

$$u_1^* = \max \left\{ 0, \min \left(\frac{S(\lambda_3 - \lambda_1) \lambda}{B_1}, 1 \right) \right\} \quad (15)$$

$$u_2^* = \max \left\{ 0, \min \left(\frac{S(\lambda_1 - \lambda_2)\theta}{B_2}, 1 \right) \right\} \quad (16)$$

$$u_3^* = \max \left\{ 0, \min \left(\frac{\varepsilon m(\lambda_3 - \lambda_1)}{B_3}, 1 \right) \right\} \quad (17)$$

$$u_4^* = \max \left\{ 0, \min \left(\frac{(\gamma(\lambda_6 - \lambda_5) - \lambda_6 + \lambda_4)I_s\tau}{B_4}, 1 \right) \right\} \quad (18)$$

Proof. To obtain the transversality conditions and the adjoint equations, we utilize the Hamiltonian (12). The adjoint system outcomes from Pontryagin's Minimum Principle (13) from

$$\begin{aligned} \frac{d\lambda_1(t)}{dt} &= -\frac{\partial H}{\partial S}, \quad \frac{d\lambda_2(t)}{dt} = -\frac{\partial H}{\partial P}, \quad \frac{d\lambda_3(t)}{dt} = -\frac{\partial H}{\partial I_a}, \\ \frac{d\lambda_4(t)}{dt} &= -\frac{\partial H}{\partial I_s}, \quad \frac{d\lambda_5(t)}{dt} = -\frac{\partial H}{\partial A}, \quad \frac{d\lambda_6(t)}{dt} = -\frac{\partial H}{\partial T}. \end{aligned} \quad (19)$$

with $\lambda_i(t_f) = 0$, $i = 1, 2, 3, 4, 5, 6$. To obtain the characterization of the control given by (15)-(18), solving the equations,

$$\frac{\partial H}{\partial u_1} = 0, \quad \frac{\partial H}{\partial u_2} = 0, \quad \frac{\partial H}{\partial u_3} = 0, \quad \frac{\partial H}{\partial u_4} = 0. \quad (20)$$

within the interior of the control set and using the property of the control space U , we can determine the chosen characterization (15), (16), (17) and (18). Here we can call the equations (15), (16), (17) and (18) for $(u_1^*, u_2^*, u_3^*, u_4^*)$ the characterization of the optimal control. To determine the optimal control and state, we solve the optimality system, which includes the state system (1), the adjoint system (13), and the relevant initial conditions at $t=0$. We also take into account the boundary conditions (14) and the characterization of the optimal control outlined in (15), (16), (17), and (18). To address the optimality system, we utilize the transversality and initial conditions along with the characterization of the optimal control $(u_1^*, u_2^*, u_3^*, u_4^*)$ given by (15), (16), (17) and (18). Furthermore, the second derivative of the Lagrangian concerning u_1 , u_2 , u_3 and u_4 , respectively, is positive, which demonstrate that the optimal problem is minimum at controls u_1^* , u_2^* , u_3^* , and u_4^* . Therefore, the resulting optimality system derived from the state system alongside the adjoint system, the optimal control, and the transversality conditions, will be numerically solved.

III. RESULTS AND DISCUSSIONS

In this section, we showcase some numerical solutions for Model (1) by examining how different parameter and control values affect the outcomes. We'll use the initial values $S(0) = 1000$, $P(0) = 300$, $I_a(0) = 0$, $I_s(0) = 0$, $A(0) = 0$ and $T(0) = 0$. The parameters used in our analysis are listed in Table 1. You can find graphical representations of the model in the Figures below.

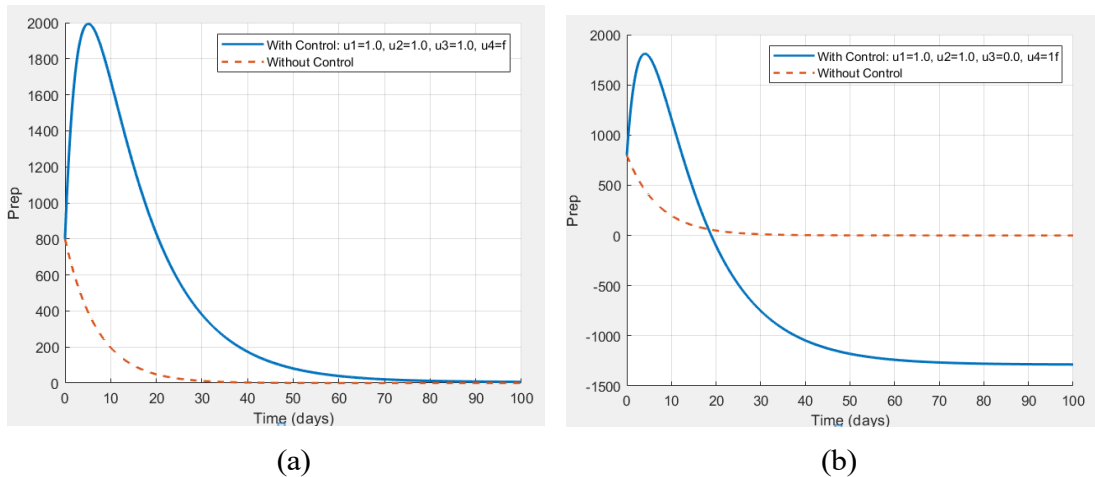


Figure 1: Plots of the Dynamics of Prep Strategies

In Figure 1, we found that adherence to PrEP strategies among individuals peaked during the first five days when there was no infection present. This adherence then declined to zero over 70 days without the immigration of infected individuals into the susceptible population. In contrast, Figure 1(b) shows that with the introduction of infected individuals, the decline in adherence lasted for only 19 days. This suggests that people don't view adherence to PrEP strategies as a long-term solution.

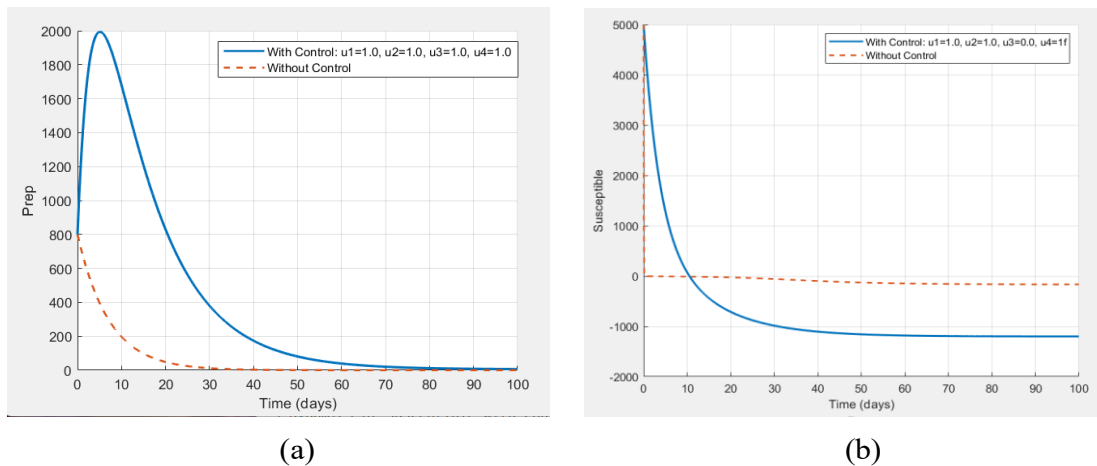


Figure 2: Plots of the Dynamics of Prep Strategies and Susceptible Class

Figure 2 demonstrates the dynamics of both adherences to PrEP strategies and the susceptible population, comparing scenarios with and without control measures.

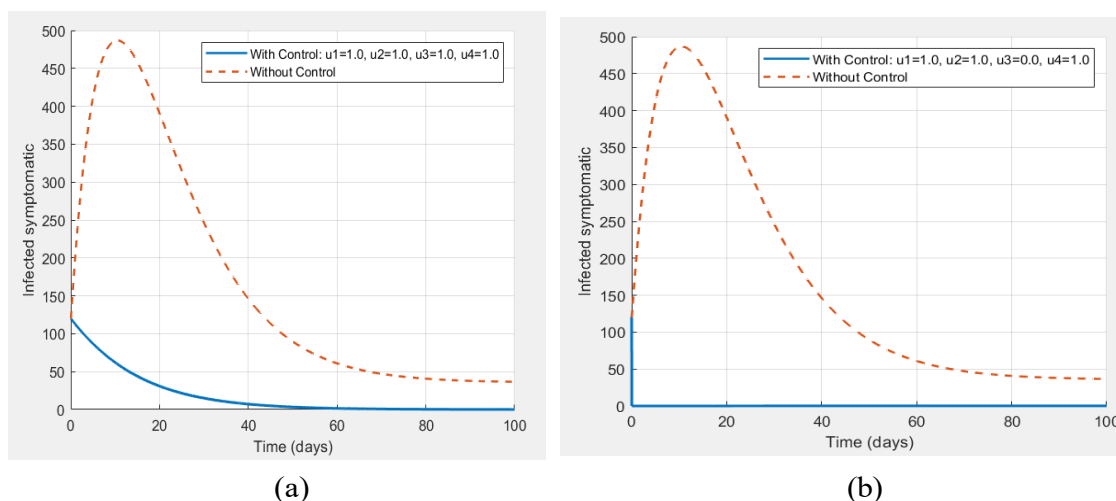


Figure 3: Dynamics of Symptomatic Infected Individuals

The figure above shows a significant rise in the number of symptomatic infected individuals initially, but over time, this number trends down toward zero. In contrast, when control measures are implemented, the number of infections decreases more effectively toward zero. This clearly highlights the importance of these control measures in managing infections.

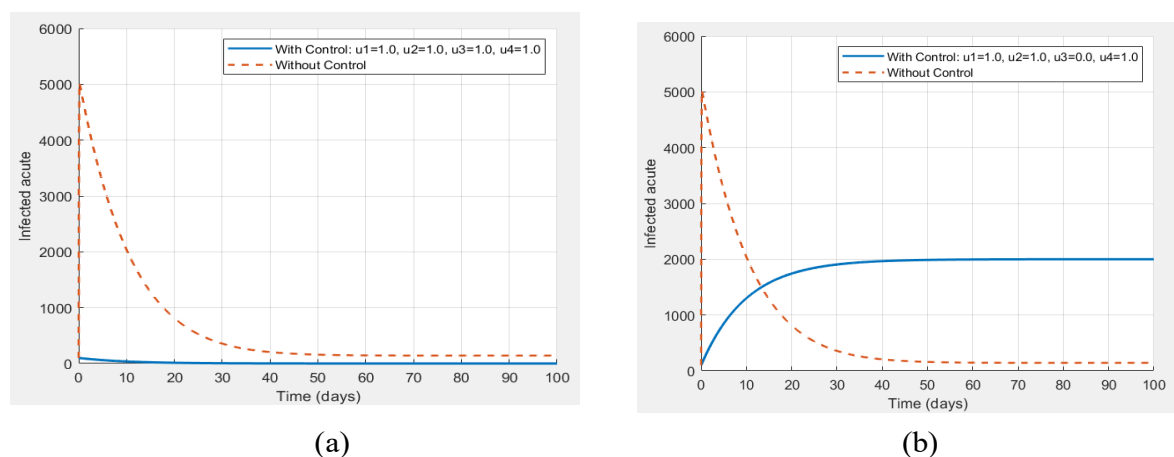


Figure 4: Dynamics of Acutely Infected Individuals

Figure 4 shows some interesting trends regarding acute infections. Initially, we see a sharp increase in the number of infected individuals during the first ten days without any control measures in place. After this period, however, the number of infections starts to decline. In Figure 4a, when we look at the situation without immigration of infected individuals and set the variable $u_1=1$, the infection levels are not significant. But in Figure 4b, once control measures are implemented while also allowing for the immigration of infected individuals, the number of infections spikes to 2,000 and then levels off. This clearly highlights the important role that the immigration of infected individuals plays in the dynamics of HIV/AIDS.

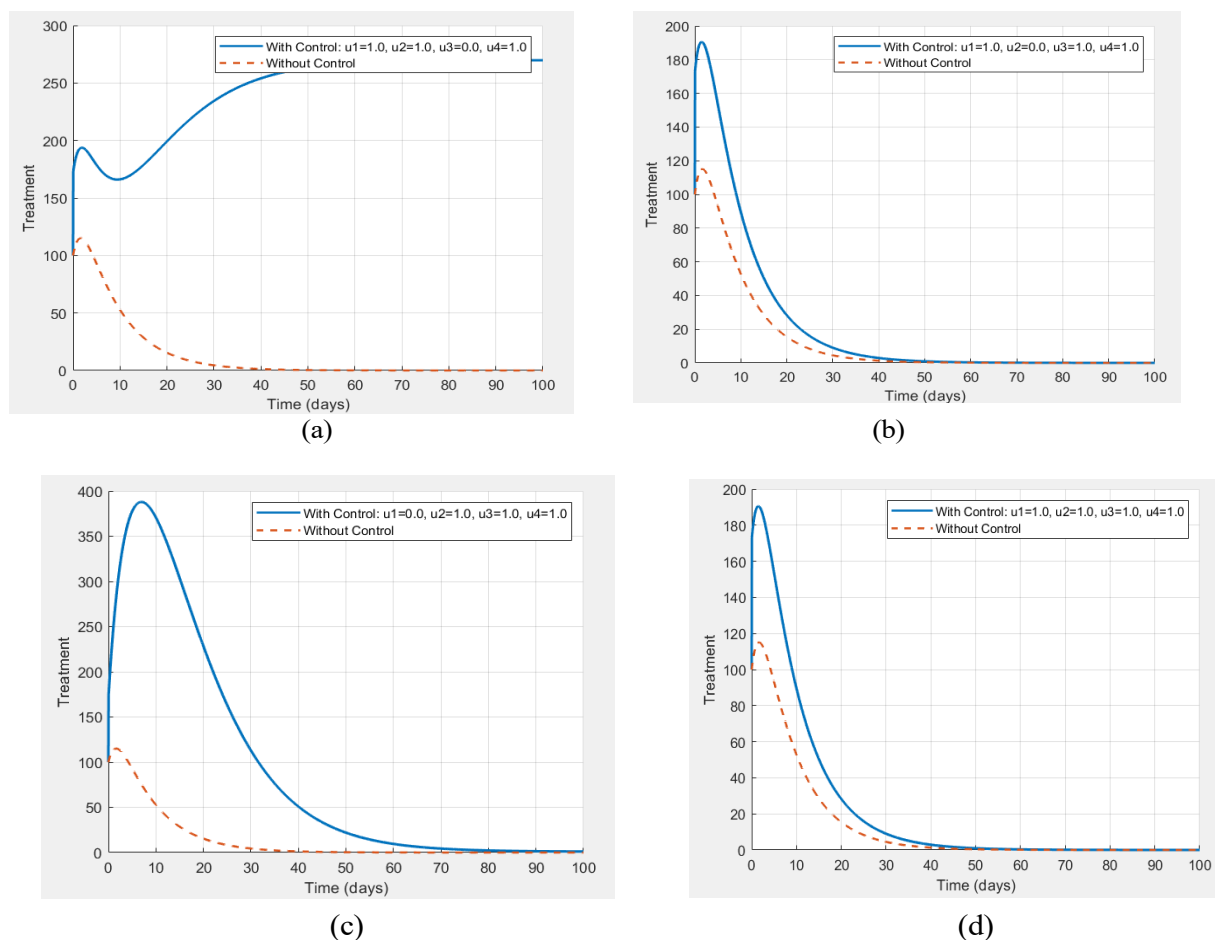


Figure 5: The dynamics of Treatment Individuals

Finally, Figure 5 highlights that the treatment class behavior improves with control measures and worsens in their absence. The data suggest that adopting PrEP strategies is not a long-term measure with the trend depicted in Figure 1. However, the four strategies— u_1 , u_2 , u_3 , and u_4 —can lead to a significant reduction in both the prevalence and incidence of the disease. Effective intervention strategies should focus on decreasing the infectiousness of people living with HIV while preventing transmission among susceptible individuals. Consequently, addressing both the susceptible and infected populations, including immigrants, can result in fewer new infections and a substantial decline in overall infection prevalence.

IV. CONCLUSION

In this study, a compartmental model was developed and examined to investigate the effect of PrEP and other control measures adopted in the dynamics of HIV/AIDs with in-flow of infective immigrants incorporating treatment with the saturated incidence. The equilibria point, the basic reproduction number and the stability at disease-free equilibrium were obtained. Also, an optimal control problem was proposed to determine the optimal approach that minimizes the number of HIV infections, this includes four control strategies; control $u_1(t)$ represents public sensitization and condom usage, and $u_2(t)$ represents the decision-making or adherence to Prep strategy. The control to increase the screening/treatment of infected individuals is $u_3(t)$. Also, the efficacy of the anti-retroviral drug in treatment $u_4(t)$. Using Pontryagin's minimum principle the optimality system was characterized and solved numerically. It was observed that the optimal technique of averting further incidence of HIV/AIDs is to prioritise public sensitization on

condom usage in adults, screening and treatment of infected individuals are robust control measures. However, according to this study, adherence to PrEP strategies is not a long-term measure for the control of HIV/AIDS in Africa.

REFERENCES

- [1] Canadian Foundation for AIDS Research (CANFAR). History of HIV/AIDS [Internet]. 2023. Available from: <http://canfar.com>. Accessed 2 Jul 2024.
- [2] World Health Organization (WHO). HIV and AIDS: Key facts [Internet]. 2023. Available from: <https://www.who.int/newsroom/fact-sheets/detail/hiv-aids>. Accessed 2 Jul 2024.
- [3] Udoo I. J. M., Kimbir R. A., Odekunle R. M. (2015). Existence and uniqueness of solution of an HIV/AIDS model considering counseling, vaccination, and antiretroviral therapy (ART). *Mathematical Theory and Modeling*;5(11).
- [4] World Health Organization (WHO). WHO expands its recommendation on the use of oral PrEP for HIV prevention [Internet]. 2015. Available from: <https://www.who.int/news-room/recommendation-on-the-use-of-oral-PrEP/detail/hiv-aids>. Accessed 2 Jul 2024.
- [5] World Health Organization (WHO). Policy brief on oral pre-exposure prophylaxis of HIV infection (PrEP) [Internet]. 2015. Available from: <http://www.who.int/hiv/prep/policy-brief-prep-2015/en/>
- [6] Oladejo J. K., and Oluyo T. O. (2022). Effects of PrEP on the HIV/AIDS dynamics with of immigration of infectives. *International Journal of Mathematical Analysis and Modeling*. 5(2):264-283.
- [7] Chinwendu E. and Martin, (2015). A Mathematical Modelling to study the impact of Irresponsible Infective Immigrants and vertical Transmission on the Dynamics of HIV/AIDS. *J. Math. Comp. Sci.* vol. (1): 72-92.
- [8] Dabbo M. I. and Seidu B. (2013). Modelling the Combine Effect of careless Susceptible and infective immigrants on the dynamics of HIV/AIDS, *Journal of public Health & epidemiology*. Vol 3(1), 362-369
- [9] Issa S. and Massawe S. (2011). Modelling the effect of screening on the spread of HIV infection in a population with variable flow of of infective immigrants. *Scientific Research and Essays*. Vol 20. 4397-4405.
- [10] Lekan A. E., Momoh A. A., Tahir A. and Modibbo U. M. (2015). “On the Stability of Endemic Equilibrium State of HIV/AIDS Model with Irresponsible Infective Immigrants”. *Pacific Journal of Science and Technology*. 16(2):225-233.
- [11] Mastahun M. and Abdurahman X. (2017). Optimal Control of an HIV/AIDS epidemic model with infective immigration and behavioural change. *App .math*, vol 8, 87-105. <http://www.scirp.org/journal/am>
- [12] Shim Eunha (2004). An Epidemic Model with Immigration of Infectives and Vaccination. Published Master’s Thesis, *University of British Columbia, Vancouver*, BC Canada.
- [13] Mohammed, I. D. and Baba S.. (2012). Modelling the Effect of Irresponsible Infective Immigrants on the Transmission Dynamics of HIV/AIDS. *Advances in Applied Mathematical Biosciences*. ISSN 2248-9983 Volume 3, Number 1, pp. 31-40 International Research Publication House <http://www.irphouse.com>.
- [14] Kim S. B., Yoon M., Ku N. S., Kim M. H., Song J. E., (2014). Mathematical modeling of HIV prevention measures including pre-exposure prophylaxis on HIV incidence in South Korea. *PLoS ONE* 9, no. 3, e90080, 1–9. <http://doi.org/10.1371/journal.pone.0090080>
- [15] Abbas U. L., Anderson R. M., Mellors J. W. (2007). Potential impact of antiretroviral chemoprophylaxis on HIV-1 transmission in resource-limited settings. *PloS one*. 2007 Sep 19;2(9):e875.
- [16] Silva C. J. and Torres, D. F. M. (2017). Modeling and Optimal Control of HIV/AIDS Prevention through PrEP. *Ar Xiv:1703.06446v2 [math.oc]* <http://dx.doi.org/10.3934/dcdss.2018008>.
- [17] Afassinou K., Chirove F., Govinder K. S. (2017). Pre-exposure prophylaxis and antiretroviral

- treatment interventions with drug resistance. *Mathematical-Bioscience*. 285:92-101. Doi;10.1016/j.mbs.2017.01.005
- [18] Drabo E. F., Hay J. W., Vardavas R., Wagner Z. R., Sood N ., (2016). A cost-effectiveness analysis of pre-exposure prophylaxis for the prevention of HIV among Los Angeles County men who have sex with men. *Clinical Infectious Diseases*. 1;63(11):1495-504. doi:10.1093/cid/ciw578.
- [19] Moya E. M., Rodrigues D. S., Pietrus A., Severo A. M. (2022). A mathematical model for HIV/AIDS under preexposure and post-exposure prophylaxis. *Biomath*.2022;11(2):2208319
- [20] Mastahun, M. and Abdurahman, X. (2017). Optimal Control of an HIV/AIDS epidemic model with infective immigration and behavioural change. *App.math*, vol 8, 87-105. <http://www.scirp.org/journal/am>
- [21] Musa R., Robert W., and Nabendra P., (2012). Optimal control and sensitivity Analysis of an HIV/AIDS-Resistant Model with Behavioral Change. *Springer Nature*. 69(4) 543- 589. doi;10.1007/s10441-021-09421-3
- [22] Valesco and Hsieh, Y. H. (1994). Modelling the effect of Treatment and behaviour in the dynamics of HIV change in behavior and sexual industry: Its implications for spread of HIV/AIDS, *Bull. Math. Biol.* 66 143 166
- [23] Akudibillah, Gordon & Pandey, Abhishek & Medlock, (2019). Optimal control for HIV treatment. *Mathematical Biosciences and Engineering*. 16. 373-396. Doi:10.3934/mbe.2019018
- [24] 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*, Volume 26, 104263, ISSN 2211-3797. <https://doi.org/10.1016/j.rinp.2021.104263>
- [25] 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. *Journal of Applied Sciences and Environmental Management*. 2024;28(8):2347-2357.
- [26] World Health Organization (WHO). World Health Organization Data [Internet] 2025. Available from: <https://www.who.int/data/gho/data/countries/country-details/GHO/nigeria?hl=en-GB>. Accessed 27/5/2025
- [27] L.S. Pontragin, R.V. Gamkrelize, V.G. Boltyanskii, and E.F. Mishchenko, (1962). *The Mathematical Theory of Optimal Processes*, Wiley, New York, 1962.