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Understanding Transmission Dynamics of HIV/AIDS with Sensitivity Analysis and Optimal Control Strategies

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Abstract. In this article, we propose a comprehensive mathematical model that investigates the dynamics of HIV/AIDS transmission, concentrating on two distinct groups: males and females. Within this theoretical framework, seven classes of individuals are considered for each gender. We compute the basic reproduction number R_0, which is crucial to determine the risk of disease transmission among these populations. Furthermore, Routh-Hurwitz criterion is used in stability analysis of disease-free and endemic equilibrium points. The stability analysis of the model provides that at the disease-free equilibrium point the system is locally asymptotically stable when $R_0 < 1$, and if $R_0 > 1$ the system is locally asymptotically unstable at the endemic equilibrium point. Additionally, the sensitivity of both reproduction number and the state variables with the key parameters are explored in order to revealing the most influential parameters that affect the disease transmission. This study develops an optimal control framework incorporating prevention, screening, and treatment interventions to combat HIV/AIDS transmission in gender-stratified populations. Using fourth-order Runge-Kutta methods implemented in MATLAB, we numerically solve the control system and simulate population dynamics under various intervention scenarios. Our results demonstrate that the combined implementation of all three interventions yields superior infection reduction compared to isolated measures, with significant decreases in prevalence rates observed for both genders. These findings provide quantitative evidence for public health decision-making, offering both a validated mathematical tool for epidemic modeling and practical insights for optimizing resource allocation in HIV/AIDS control programs. The framework advances current methodologies by systematically evaluating intervention effectiveness while accounting for gender-specific transmission dynamics.

2020 Mathematics Subject Classifications: 92C42, 34C60, 92D25, 92D30, 37N25

Key Words and Phrases: HIV/AIDS model, Stability, Sensitivity, Optimal Control, Numerical Simulation

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1. Introduction

In 1981, for the first time acquired immunodeficiency syndrome (AIDS) was initially recognized as a new infectious disease within the community of homosexual in Los Angeles and San Francisco [1, 2]. Over the past forty years, the human immunodeficiency virus (HIV) has caused the deaths of numerous individuals, including men, women, and children worldwide, for instance in 2003 each day there were 15000 new infected individuals and 8000 died with HIV in the world [1]. The World Health Organization (WHO) reports that 71.3-112.8 million people have contracted HIV, and that 35.7-51.1 million people have lost their lives to the disease since the virus's inception. There were 39.9 million HIV-positive individuals worldwide by the end of 2023 and in the year 2023 the number of people who died of HIV related illness is 630000 globally [3]. Worldwide, it is estimated that $0.6-0.7\%$ of individuals aged 15 to 49 are infected with HIV, however the impact of the pandemic significantly differs among countries and regions. The World Health Organization reports that the most severely affected is the African Region continues with 3.4% of adults living with HIV which is almost two-thirds of all HIV-positive people globally.

There are several ways in which HIV transmitted from an infected to a victim and the most common ones are vertical transmission from mother to child, homosexual contact, blood transfusions, and shared drug injection needles [4–6]. Understanding HIV infection illness and researching the epidemiological dynamics of HIV are made possible in large part by mathematical modeling. During the years 1986-1988 a series of researches on the role of mathematical modelling on HIV transmission and the transmission dynamics of AIDS were published (see for example [7–10]). Researchers interested in HIV dynamics and transmission including both applied mathematicians and biologists.

Through the last four decades a huge number of researches on the development of the mathematical models of the disease and dynamics behavior, stability and sensitivity analysis of the model, and the role of basic reproduction number R_0 in controlling the spread of AIDS have been published. Some researchers have developed and examined the stability of equilibrium points within HIV/AIDS models. Contingent upon whether the reproduction number R_0 exceeds or falls below unity, they conducted an analysis on the stability and examined at both endemic and disease-free equilibrium points [6, 11, 12].

A mathematical model of HIV was created by Doyle et al. [13] to simulate how the virus spreads through sexual contact in a heterosexual population and the stability at the equilibrium points are carried out. They designed their model to explore the implications of a basic constraint in any sexual mixing process heterosexual population. Kaur et al. [14] investigated the impact of screening awareness and counseling on HIV transmission in the endemic region using a mathematical model in nonlinear form. They studied the impact of suitable choice of parameters to be consistent with screening awareness and counselling the disease can be removed in the population in a reasonable period of time. Naresh et ai. In [15] and Waziri et al. in [16] proposed nonlinear mathematical models to for examine the vertical transmission of HIV dynamics. In order to minimize the risk of transmitting the infection to future generations, providing treatment to individuals with HIV before they develop AIDS can significantly decrease the probability of transmission.

The obtained results in their study showed that in 1997 in the U.S fewer than 300 infants acquired HIV through vertical transmission. During the last few years, in studying the transmission and development of HIV models within the population and to the reflection of dynamics HIV epidemic, many scholars adopted the idea of compartment modelling, which is the division of the total population and corresponding categories. These models can be used in prediction of HIV incidence in a shorter long-term, they also become an important tool in analyzing the transmission and control of HIV [17]. Sensitivity analysis and optimal control of human immunodeficiency virus have studied by many researchers to show the effect of parameters on the dynamics of the transmission of HIV in a population. Various strategies such as treatment, prevention, and screening are used to control the disease [18–23].

Mathematical model is a very important scientific tool in representing the biological problems to the form of mathematical equations. In this instance, we examine and evaluate an HIV model where people are categorized as male or female. We discuss the role of the basic reproduction number in the study of stability analysis. Moreover, the elasticity of the parameters in which increase or decrease the value of the reproduction number is analyzed. Additionally, different control strategies are used in order to reduce or stop of HIV transmission in human population.

The article is arranged as follows: In Section 1, the parameters and variables are introduced and the mathematical model is formulated. In Section 2, the formulated model is analyzed mathematically to find the invariant region and provided that the solutions of the model are positive. In Section 3, the equilibrium points and the basic reproduction number R_0 are computed. In Section 4, the local stability of the system of equations at the equilibrium points are studied. In Section 5, local sensitivity analysis is discussed by using three different techniques. Section 6 describes the expansion of the model with the incorporation of optimal control and analyzed. In Section 7, the numerical simulation for different combinations of strategies is illustrated. In Section 8, conclusion is provided.

2. Model Equation

In this section, the proposed model equation is formulated. The total number of individuals $N(t)$ at time t is split into seven sub-populations for females and seven sub-populations for males which are fourteen sub-populations in total: susceptible populations $S_f(t)$ and $S_m(t)$, exposed populations $E_f(t)$ and $E_m(t)$ which have got the sickness, but are not yet infectious, infected populations $I_f(t)$ and $I_m(t)$, undetected populations $U_f(t)$ and $U_m(t)$, detected populations $D_f(t)$ and $D_m(t)$, treating populations $T_f(t)$ and $T_m(t)$, and AIDS populations $A_f(t)$ and $A_m(t)$. Therefore,

$$\begin{aligned} N(t) = S_f(t)+&S_m(t) + E_f(t) + E_m(t) + I_f(t) + I_m(t) + U_f(t) + U_m(t) + D_f(t) + D_m(t) \\ &+ T_f(t) + T_m(t) + A_f(t) + A_m(t) \end{aligned}$$

At a constant rate Λ, the population enters the susceptible compartment. Infection occurs only through successful contact between susceptible females S_f and infected males I_m and

susceptible males S_m and infected females I_f, with probabilities β_f or β_m respectively. Abstinence avoiding all forms of vaginal, anal, and oral sex are the most efficient methods of preventing the sexual transmission of HIV. For the suggested model, we account for the use of male or female condoms, which can prevent HIV transmission. The parameters ϵ and κ represent the effectiveness of condoms and the percentage of people who use condoms respectively. This leads to a reduction in transmission rates β_f and β_m by a factor of $1-\kappa \in$ where $0 < \epsilon < 1$ and $0 < \kappa < 1$. Higher values of κ and ϵ indicate a better condom a situation in which more individuals use condoms when having sex respectively. In this model, the transition rates from S_f to E_f and S_m to E_m are $(1-\kappa\epsilon)\beta_f$ and $(1-\kappa\epsilon)\beta_m$ respectively. Besides, the transitions from E_f and E_m to I_f and I_m are denoted by α_f and α_m respectively. Additionally, we have transitions from I_f to both D_f and U_f which are symbolized by γ_f and θ_f respectively, also from I_m to both D_m and U_m which are symbolized by γ_m and θ_m respectively. Furthermore, there are a transition from D_f and D_m to T_f and T_m as a result of an identification of HIV through medical test and denoted by ρ_f and ρ_m respectively. Moreover, the transitions from both U_f and T_f to A_f are given by λ_f and ω_f respectively and from both U_m and T_m to A_m are denoted by λ_m and ω_m respectively. The natural death rate of each compartment is symbolized by η and there is an additional death rate denoted by μ due to the disease for all compartments except S_f, E_f, S_m, and E_m. The transmission of the disease from an infected female to a healthy male or from an infected male to a healthy female is only through sexual contact. In Figure 1, the male and female subpopulations are shown where coupling represented by the dashed arrows.

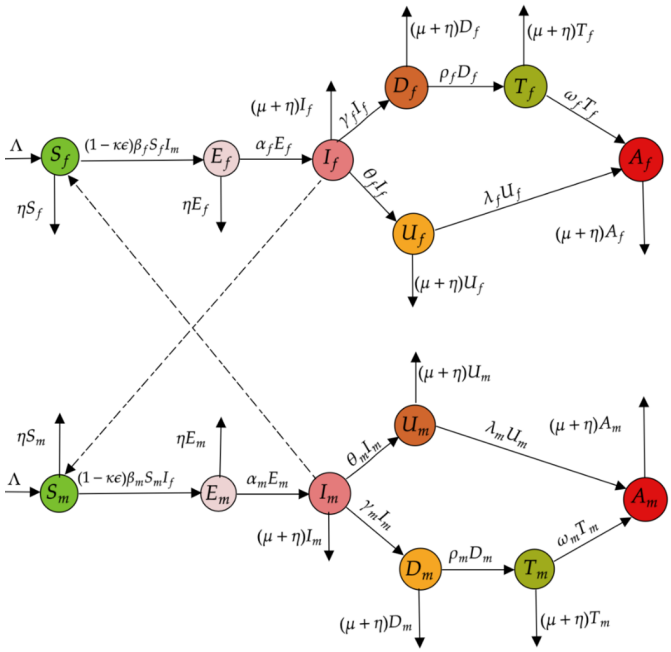

Figure 1: Flow diagram of the model

Based on the above diagram (Figure 1) and the considered assumptions, the following system of differential equations represents the mathematical model

$$\begin{aligned}
\frac{dS_f}{dt} &= \Lambda - (1-\kappa\epsilon)\beta_f I_m S_f - \eta S_f & \frac{dS_m}{dt} &= \Lambda - (1-\kappa\epsilon)\beta_m I_f S_m - \eta S_m \\
\frac{dE_f}{dt} &= (1-\kappa\epsilon)\beta_f I_m S_f - (\alpha_f + \eta)\, E_f & \frac{dE_m}{dt} &= (1-\kappa\epsilon)\beta_m I_f S_m - (\alpha_m + \eta)\, E_m \\
\frac{dI_f}{dt} &= \alpha_f E_f - (\mu + \eta + \gamma_f + \theta_f)\, I_f & \frac{dI_m}{dt} &= \alpha_m E_m - (\mu + \eta + \gamma_m + \theta_m)\, I_m \\
\frac{dD_f}{dt} &= \gamma_f I_f - (\mu + \eta + \rho_f)\, D_f & \frac{dD_m}{dt} &= \gamma_m I_m - (\mu + \eta + \rho_m)\, D_m \\
\frac{dT_f}{dt} &= \rho_f D_f - (\mu + \eta + \omega_f)\, T_f & \frac{dT_m}{dt} &= \rho_m D_m - (\mu + \eta + \omega_m)\, T_m \\
\frac{dU_f}{dt} &= \theta_f I_f - (\mu + \eta + \lambda_f)\, U_f & \frac{dU_m}{dt} &= \theta_m I_m - (\mu + \eta + \lambda_m)\, U_m \\
\frac{dA_f}{dt} &= \lambda_f U_f + \omega_f T_f - (\mu + \eta) A_f & \frac{dA_m}{dt} &= \lambda_m U_m + \omega_m T_m - (\mu + \eta) A_m
\end{aligned} \tag{1}$$

with the initial conditions $S_f(0), S_m(0) > 0$, and

$$E_f(0), E_m(0), I_f(0), I_m(0), D_f(0), D_m(0), T_f(0), T_m(0), U_f(0), U_m(0), A_f(0), A_m(0) \geq 0$$

In Table 1, the parameter values and initial variables of the model 1 are provided.

Table 1: Initial estimated values of model states and parameters

Symbols	Biological Meaning	Estimated	Values
$S_m(0)$	Initial susceptible males	19000000	Assumed
$S_f(0)$	Initial susceptible females	20000000	Assumed
$E_m(0)$	Initial exposed males	100000	Assumed
$E_f(0)$	Initial exposed females	120000	Assumed
$I_m(0)$	Initial infectious males	60000	Assumed
$I_f(0)$	Initial infectious females	65000	Assumed
$D_m(0)$	Initial detected males	25000	Assumed
$D_f(0)$	Initial detected females	28000	Assumed
$U_m(0)$	Initial undetected males	52000	Assumed
$U_f(0)$	Initial undetected males	60000	Assumed
$T_m(0)$	Initial treated males	9500	Assumed
$T_f(0)$	Initial treated females	11000	Assumed
$A_m(0)$	Initial symptomatic AIDS males	2500	Assumed
$A_f(0)$	Initial symptomatic AIDS females	2600	Assumed
Λ	Net flow rate into the susceptible	100,000	[22]

Symbols	Biological Meaning	Estimated	Values
η	Natural mortality rate	0.0222	[24]
μ	Disease-induced death rate	0.333	[25]
ϵ	Efficiency rate of condom	[0,1]	[23]
κ	Rate of people who use condoms	[0,1]	[23]
β_m	Male effective contact rate	0.025	[22]
β_f	Female effective contact rate	0.015	[22]
α_m	Rate at which expose males are infected	0.75	Assumed
α_f	Rate at which exposed females are infected	0.8	Assumed
γ_m	Rate at which infected males are detected	0.512	[22]
γ_f	Rate at which infected female are detected	0.675	[22]
ρ_m	Treatment rate of detected male	[0,1]	Assumed
ρ_f	Treatment rate of detected female	[0,1]	Assumed
ω_m	Progression rate of males under treatment to AIDS	0.004	[22]
ω_f	Progression rate of females under treatment to AIDS	0.004	[22]
θ_m	Transmission rate of infected males to undetected	0.12	Assumed
θ_f	Transmission rate of infected females to undetected	0.12	Assumed
λ_m	Progression rate of undetected males to AIDS	0.34	Assumed
λ_f	Progression rate of undetected females to AIDS	0.34	Assumed

3. Model Analysis

This section looks at the model solution's positivity and locates the invariant area. First, we discuss the invariant region in the following theorem.

Theorem 1. *With a non-negative initial condition in R_+^{14}, all solutions of the HIV/AIDS model in system (1) stays positive for all time $t > 0$.*

Proof. From the first equation of model (1) we have that

$$\frac{dS_f}{dt} = \Lambda - (1-\kappa\epsilon)\beta_f I_m S_f - \eta S_f > -\left[(1-\kappa\epsilon)\beta_f I_m - \eta\right] S_f,$$

so that,

$$\frac{dS_f}{dt} > -\left[(1-\kappa\epsilon)\beta_f I_m + \eta\right] S_f$$

Solving the above inequality, we obtain

$$S_f(t) > S_f(0) e^{-\int_0^t \left[(1-\kappa\epsilon)\beta_f I_m(x) + \eta\right] dx}.$$

It is given that $S_f(0) > 0$ and clearly $e^{-\int_0^t \left[(1-\kappa\epsilon)\beta_f I_m(x) + \eta\right] dx}$ is nonnegative. Therefore, $S_f(t)$ is nonnegative for all time $t > 0$. By the same manner, we can show that the solutions for the rest of variables of system (1) are nonnegative. Hence, we obtained nonnegative solutions for the model.

Now, in the following theorem we find the positive invariant area for the model (1).

Theorem 2. *The initial conditions are bounded within a bounded region $\Sigma \subset R_+^5$, ensuring that all solutions of the model remain bounded within this specified region. So that,*

$$\Sigma = \{S_f(t), S_m(t), E_f(t), E_m(t), I_f(t), I_m(t), U_f(t), U_m(t), D_f(t), D_m(t), T_f(t), T_m(t), A_f(t), A_m(t) \in \mathcal{R}_+^{14} : N(t) \leq \frac{2\Lambda}{\eta}\Big\}.$$

Proof. $N(t)$ denotes the total population number at time t which is the sum of all populations and given by

$$\begin{aligned} N(t) = S_f(t)+S_m(t) + E_f(t) + E_m(t) + I_f(t) + I_m(t) + U_f(t) + U_m(t) + D_f(t) + D_m(t) \\ + T_f(t) + T_m(t) + A_f(t) + A_m(t) \end{aligned}$$

Differentiating both sides of the above equation and simplifying, we acquire the following result

$$\begin{aligned} \frac{dN}{dt} &= 2\Lambda - \eta N - \mu N + \mu\left(S_f + S_m\right) \\ \frac{dN}{dt} &\leq 2\Lambda - \eta N \end{aligned}$$

Applying the comparison theorem to the above inequality we get that,

$$\begin{aligned} N(t) &\leq N(0)e^{-\eta t} + \frac{2\Lambda}{\eta}\left(1 - e^{-\eta t}\right) \\ N(t) &\leq \frac{2\Lambda}{\eta} + \left(N(0) - \frac{2\Lambda}{\eta}\right)e^{-\eta t} \end{aligned}$$

It follows that $N(t) \to \frac{2\Lambda}{\eta}$ as $t \to \infty$. Hence, $N(t) \leq \frac{2\Lambda}{\eta}$. We conclude that the invariant region Σ represents the feasible solution region of the model.

From theorem 1 and theorem 2 , it is sufficient to take into account the dynamics of the system 1 in the region Σ where the positiveness, existence, and uniqueness of the solution hold.

4. Equilibrium Points and Basic Reproduction Number R_0

In this section, we compute two key points for system 1: the disease-free equilibrium (DFE) in the absence of the disease, and the endemic equilibrium (EE) during an outbreak. In addition, we utilize the next-generation matrix in calculating the basic reproduction number, R_0.
In the model 1, we set

$$\begin{array}{llll}
\Gamma_f = \alpha_f + \eta, & \Gamma_m = \alpha_m + \eta, & \Delta_f = \mu + \eta + \gamma_f + \theta_f, & \Delta_m = \mu + \eta + \gamma_m + \theta_m \\
\Omega_f = \mu + \eta + \rho_f, & \Omega_m = \mu + \eta + \rho_m, & \Psi_f = \mu + \eta + \omega_f, & \Psi_m = \mu + \eta + \omega_m \\
\Phi_f = \mu + \eta + \lambda_f, & \Phi_m = \mu + \eta + \lambda_m, & \zeta = \mu + \eta &
\end{array}$$

The system 1 becomes,

$$\begin{array}{ll}
\frac{dS_f}{dt} = \Lambda - (1-\kappa\epsilon)\beta_f I_m S_f - \eta S_f & \frac{dS_m}{dt} = \Lambda - (1-\kappa\epsilon)\beta_m I_f S_m - \eta S_m \\
\frac{dE_f}{dt} = (1-\kappa\epsilon)\beta_f I_m S_f - \Gamma_f E_f & \frac{dE_m}{dt} = (1-\kappa\epsilon)\beta_m I_f S_m - \Gamma_m E_m \\
\frac{dI_f}{dt} = \alpha_f E_f - \Delta_f I_f & \frac{dI_m}{dt} = \alpha_m E_m - \Delta_m I_m \\
\frac{dD_f}{dt} = \gamma_f I_f - \Omega_f D_f & \frac{dD_m}{dt} = \gamma_m I_m - \Omega_m D_m \\
\frac{dT_f}{dt} = \rho_f D_f - \Psi_f T_f & \frac{dT_m}{dt} = \rho_m D_m - \Psi_m T_m \\
\frac{dU_f}{dt} = \theta_f I_f - \Phi_f U_f & \frac{dU_m}{dt} = \theta_m I_m - \Phi_m U_m \\
\frac{dA_f}{dt} = \lambda_f U_f + \omega_f T_f - \zeta A_f & \frac{dA_m}{dt} = \lambda_m U_m + \omega_m T_m - \zeta A_m
\end{array} \tag{2}$$

4.1. Disease-Free Equilibrium Point (E_0)

The equilibrium point without the disease for the system 2 represents a stable state in which the population is uninfected by HIV/AIDS. We set all the variables to be zero except S_f and S_m. Hence, $E_f = E_m = I_f = I_m = U_f = U_m = D_f = D_m = T_f = T_m = A_f = A_m = 0$

Then we equal the right-hand side of all equations in system (2) to zero, we obtain that $S_f = S_m = \frac{\Lambda}{\eta}$, and $E_f = E_m = I_f = I_m = U_f = U_m = D_f = D_m = T_f = T_m = A_f = A_m = 0$.

Thus, the disease-free equilibrium is

$E_0 = (S_f, E_f, I_f, D_f, T_f, U_f, A_f, S_m, E_m, I_m, D_m, T_m, U_m, A_m) = \left(\frac{\Lambda}{\eta}, 0, 0, 0, 0, 0, 0, \frac{\Lambda}{\eta}, 0, 0, 0, 0, 0, 0\right)$.

4.2. Basic Reproduction Number (R_0)

We compute R_0 by utilizing the next generation matrix. The matrix serves the purpose of calculating the reproductive number, denoted as R_0. This technique defines R_0 as the greatest absolute eigenvalue within the next-generation matrix. In creating this matrix, we categorize all terms of the disease classes of the model into two groups, v represents the matrix for transition cases, while f represents the matrix for newly infected cases.

The disease classes are

$$\begin{array}{ll|ll}
\frac{dE_f}{dt} & = (1-\kappa\epsilon)\beta_f I_m S_f - \Gamma_f E_f & \frac{dE_m}{dt} & = (1-\kappa\epsilon)\beta_m I_f S_m - \Gamma_m E_m \\
\frac{dI_f}{dt} & = \alpha_f E_f - \Delta_f I_f & \frac{dI_m}{dt} & = \alpha_m E_m - \Delta_m I_m \\
\frac{dD_f}{dt} & = \gamma_f I_f - \Omega_f D_f & \frac{dD_m}{dt} & = \gamma_m I_m - \Omega_m D_m \\
\frac{dT_f}{dt} & = \rho_f D_f - \Psi_f T_f & \frac{dT_m}{dt} & = \rho_m D_m - \Psi_m T_m \\
\frac{dU_f}{dt} & = \theta_f I_f - \Phi_f U_f & \frac{dU_m}{dt} & = \theta_m I_m - \Phi_m U_m \\
\frac{dA_f}{dt} & = \lambda_f U_f + \omega_f T_f - \zeta A_f & \frac{dA_m}{dt} & = \lambda_m U_m + \omega_m T_m - \zeta A_m
\end{array} \tag{3}$$

In the system 3, the newly infected cases appear in the equations $\frac{dE_f}{dt}$ and $\frac{dE_m}{dt}$ which are $f_1 = (1-\kappa\epsilon)\beta_f I_m S_f$ and $f_7 = (1-\kappa\epsilon)\beta_m I_f S_m$ respectively and the last twelve equations are zero $f_2 = f_3 = f_4 = f_5 = f_6 = f_8 = f_9 = f_{10} = f_{11} = f_{12} = 0$. The rest of terms in each equation are $v_1, v_2, \ldots, v_{12}$. Hence f and v are as follows

$$f = \begin{bmatrix} f_1 \\ f_2 \\ f_3 \\ f_4 \\ f_5 \\ f_6 \\ f_7 \\ f_8 \\ f_9 \\ f_{10} \\ f_{11} \\ f_{12} \end{bmatrix} = \begin{bmatrix} (1-\kappa\epsilon)\beta_f I_m S_f \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ (1-\kappa\epsilon)\beta_m I_f S_m \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \text{ and } v = \begin{bmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \\ v_5 \\ v_6 \\ v_7 \\ v_8 \\ v_9 \\ v_{10} \\ v_{11} \\ v_{12} \end{bmatrix} = \begin{bmatrix} E_f \\ -\alpha_f E_f + \Delta_f I_f \\ -\gamma_f I_f + \Omega_f D_f \\ -\rho_f D_f + \Psi_f T_f \\ -\theta_f I_f + \Phi_f U_f \\ -\lambda_f U_f - \omega_f T_f + \zeta A_f \\ \Gamma_m E_m \\ -\alpha_m E_m + \Delta_m I_m \\ -\gamma_m I_m + \Omega_m D_m \\ -\rho_m D_m + \Psi_m T_m \\ -\theta_m I_m + \Phi_m U_m \\ -\lambda_m U_m - \omega_m T_m + \zeta A_m \end{bmatrix}$$

The Jacobian of f and v at E_0 are symbolized by F and V respectively. The next-generation matrix could be computed from FV^{-1} which is a 12×12 matrix and its absolute eigenvalues are provided below

$$\lambda_1^* = 0, \quad \lambda_2^* = 0, \quad \lambda_3^* = 0, \quad \lambda_4^* = 0, \quad \lambda_5^* = 0, \quad \lambda_6^* = 0, \quad \lambda_7^* = 0, \quad \lambda_8^* = 0, \quad \lambda_9^* = 0,$$
$$\lambda_{10}^* = 0, \quad |\lambda_{11}^*| = |\lambda_{12}^*| = \frac{\Lambda(1-\epsilon\kappa)\sqrt{\Delta_f\Delta_m\Gamma_f\Gamma_m\alpha_f\alpha_m\beta_f\beta_m}}{\Delta_f\Delta_m\Gamma_f\Gamma_m\eta}.$$

R_0 is the greatest absolute eigenvalue of the next-generation matrix which is $|\lambda_{11}^*|$ or $|\lambda_{12}^*|$. Therefore, R_0 is expressed as

$$R_0 = \frac{\Lambda(1-\epsilon\kappa)}{\eta}\sqrt{\frac{\alpha_f\alpha_m\beta_f\beta_m}{\Delta_f\Delta_m\Gamma_f\Gamma_m}}.$$

or

$$R_0 = \frac{\Lambda(1-\epsilon\kappa)}{\eta}\sqrt{\frac{\alpha_f\alpha_m\beta_f\beta_m}{(\mu+\eta+\gamma_f+\theta_f)(\mu+\eta+\gamma_m+\theta_m)(\alpha_f+\eta)(\alpha_m+\eta)}}$$

The basic reproduction number, R_0, in this HIV model is strongly influenced by key epidemiological factors. It increases with higher transmission rates between partners (β_f and β_m), as well as with a greater rate of new individuals entering the susceptible population (Λ). On the other hand, R_0 decreases when the natural death rate (η), disease-related death rate (μ), and the rates of treatment and behavior change due to interventions ($\gamma_f, \gamma_m, \theta_f$ and θ_m) are higher. Additionally, reducing the effectiveness of intervention parameters ($\epsilon\kappa$) leads to a rise in R_0.

To bring R_0 below 1 an important threshold for controlling the spread of HIV, public health strategies should focus on three main areas: raising γ_f and γ_m, and lowering β_f, and β_m and enhancing prevention efforts (maximizing the impact of $\epsilon\kappa$). This approach is in line with established HIV control strategies, which emphasize that combining early treatment with strong prevention measures is the most effective way to reduce the spread of the virus.

4.3. Endemic Equilibrium Point (E_1)

The endemic equilibrium refers to a state of stability when the illness continues to exit among the population. To determine this equilibrium, we solve system 2 for variables in terms of the parameters by equating the expressions on the right side of the system. Therefore, the obtained endemic equilibrium point is
$E_1 = \left(S_f^*, E_f^*, I_f^*, D_f^*, T_f^*, U_f^*, A_f^*, S_m^*, E_m^*, I_m^*, D_m^*, T_m^*, U_m^*, A_m^*\right)$ where ,

$$\begin{aligned}
S_f^* &= \frac{\Delta_m\Gamma_m(\Lambda\alpha_f\beta_m(\epsilon\kappa-1)-\Delta_f\Gamma_f\eta)}{\alpha_f\beta_m(\epsilon\kappa-1)(\Delta_m\Gamma_m\eta+\Lambda\alpha_m\beta_f(1-\epsilon\kappa))},\\
I_f^* &= \frac{\Lambda^2\alpha_f\alpha_m\beta_f\beta_m(\epsilon\kappa-1)^2-\Delta_f\Delta_m\Gamma_f\Gamma_m\eta^2}{\Delta_f\Gamma_f\beta_m(\epsilon\kappa-1)(\Lambda\alpha_m\beta_f(\epsilon\kappa-1)-\Delta_m\Gamma_m\eta)},\\
E_f^* &= \frac{\Delta_f}{\alpha_f}I_f^*, \quad D_f^* = \frac{\gamma_f}{\Omega_f}I_f^*, \quad T_f^* = \frac{\gamma_f\rho_f}{\Omega_f\Psi_f}I_f^*, \quad U_f^* = \frac{\theta_f}{\Phi_f}I_f^*,\\
A_f^* &= \frac{\Omega_f\Psi_f\lambda_f\theta_f+\Phi_f\gamma_f\omega_f\rho_f}{\Omega_f\Phi_f\Psi_f\zeta},\\
S_m^* &= \frac{\Delta_f\Gamma_f(\Lambda\alpha_m\beta_f(\epsilon\kappa-1)-\Delta_m\Gamma_m\eta)}{\alpha_m\beta_f(\epsilon\kappa-1)(\Delta_f\Gamma_f\eta+\Lambda\alpha_f\beta_m(1-\epsilon\kappa))},\\
I_m^* &= \frac{-\Delta_f\Delta_m\Gamma_f\Gamma_m\eta^2+\Lambda^2\alpha_f\alpha_m\beta_f\beta_m(\epsilon\kappa-1)^2}{\Delta_m\Gamma_m\beta_f(\epsilon\kappa-1)(\Lambda\alpha_f\beta_m(\epsilon\kappa-1)-\Delta_f\Gamma_f\eta)'},\\
E_m^* &= \frac{\Delta_m}{\alpha_m}I_m^*, \quad D_m^* = \frac{\gamma_m}{\Omega_m}I_m^*, \quad T_m^* = \frac{\gamma_m\rho_m}{\Omega_m\Psi_m}I_m^*, \quad U_m^* = \frac{\theta_m}{\Phi_m}I_m^*,\\
A_m^* &= \frac{\Omega_m\Psi_m\lambda_m\theta_m+\Phi_m\gamma_m\omega_m\rho_m}{\Omega_m\Phi_m\Psi_m\zeta}I_m^*.
\end{aligned}$$

Since $\epsilon, \kappa \in (0,1)$, hence $(\epsilon\kappa - 1)$ is always negative and clearly $S_f^*, S_m^* > 0$. Furthermore, $I_f^*, I_m^* > 0$ if and only if $R_0 > 1$. Therefore, all components of E_1 are positive if and only if $R_0 > 1$.

5. Stability Analysis of The Model at Equilibrium Points

When there is no disease, the model possesses a unique disease-free steady-state denoted as E_0. Conversely, if there is the disease, the model features an endemic equilibrium which is unique and denoted by E_1. In the following theorems, we study local stability at both disease-free and endemic equilibrium points.

Theorem 3. *The disease-free equilibrium point is locally asymptotically stable if and only if $R_0 < 0$.*

Proof. The Jacobian matrix of the system at the disease-free equilibrium point E_0 is a 14×14 which is

$J(E_0) =$

$$\begin{bmatrix}
-\eta & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{(\epsilon\kappa-1)\beta_f\Lambda}{\eta} & 0 & 0 & 0 & 0 \\
0 & -\Gamma_f & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{(-\epsilon\kappa+1)\beta_f\Lambda}{\eta} & 0 & 0 & 0 & 0 \\
0 & \alpha_f & -\Delta_f & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \gamma_f & -\Omega_f & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \rho_f & -\Psi_f & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \theta_f & 0 & 0 & -\Phi_f & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \omega_f & \lambda_f & -\zeta & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \frac{(\epsilon\kappa-1)\beta_m\Lambda}{\eta} & 0 & 0 & 0 & 0 & -\eta & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \frac{(-\epsilon\kappa+1)\beta_m\Lambda}{\eta} & 0 & 0 & 0 & 0 & 0 & -\Gamma_m & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \alpha_m & -\Delta_m & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_m & -\Omega_m & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \rho_m & -\Psi_m & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \theta_m & 0 & 0 & -\Phi_m & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \omega_m & \lambda_m & -\zeta
\end{bmatrix}$$

The eigenvalues of the Jacobian matrix are provided below

$$\gamma_{1,2} = -\eta, \gamma_{3,4} = -\zeta, \gamma_5 = -\Omega_f, \gamma_6 = -\Omega_m, \gamma_7 = -\Psi_f, \gamma_8 = -\Psi_m, \gamma_9 = -\Phi_f, \gamma_{10} = -\Phi_m$$

and the last four eigenvalues $\gamma_{11}, \gamma_{12}, \gamma_{13}, \gamma_{14}$ are the roots of the following degree four polynomial

$f(\gamma) = \gamma^4 + \widetilde{D_1}\gamma^3 + \widetilde{D_2}\gamma^2 + \widetilde{D_3}\gamma + \widetilde{D_4}$, where

$$\begin{aligned}
\widetilde{D_1} &= \Gamma_f + \Gamma_m + \Delta_f + \Delta_m \\
\widetilde{D_2} &= \Gamma_f\Gamma_m + \Delta_f\Delta_m + \Delta_m\Gamma_m + \Delta_m\Gamma_f + \Delta_f\Gamma_m + \Delta_f\Gamma_f \\
\widetilde{D_3} &= \Delta_m\Gamma_f\Gamma_m + \Delta_f\Gamma_f\Gamma_m + \Delta_f\Delta_m\Gamma_m + \Delta_f\Delta_m\Gamma_f \\
\widetilde{D_4} &= \Delta_f\Delta_m\Gamma_f\Gamma_m - \frac{\alpha_f\alpha_m\beta_f\beta_m\Lambda^2}{\eta^2}(1-\epsilon\kappa)^2
\end{aligned}$$

Clearly $\widetilde{D_1} > 0, \widetilde{D_2} > 0$, and $\widetilde{D_3} > 0$, and also $\widetilde{D_4} > 0$ if and only if $R_0 < 1$

$$\begin{aligned}
&\left(\widetilde{D_1}\widetilde{D_2} - \widetilde{D_3}\right)\widetilde{D_3} - \widetilde{D_1}\widetilde{D_4} = ((\Gamma_f + \Gamma_m + \Delta_f + \Delta_m)(\Gamma_f\Gamma_m + \Delta_f\Delta_m + \Delta_m\Gamma_m + \Delta_m\Gamma_f \\
&\Delta_f\Gamma_m + \Delta_f\Gamma_f) - (\Delta_m\Gamma_f\Gamma_m + \Delta_f\Gamma_f\Gamma_m + \Delta_f\Delta_m\Gamma_m + \Delta_f\Delta_m\Gamma_f))(\Delta_m\Gamma_f\Gamma_m + \Delta_f\Gamma_f\Gamma_m \\
&+ \Delta_f\Delta_m\Gamma_m + \Delta_f\Delta_m\Gamma_f) - (\Gamma_f + \Gamma_m + \Delta_f + \Delta_m)^2(\Delta_f\Delta_m\Gamma_f\Gamma_m - \frac{\alpha_f\alpha_m\beta_f\beta_m\Lambda^2}{\eta^2}(\epsilon\kappa - 1)^2)
\end{aligned}$$

By expanding the above expression, we obtain that $\left(\widetilde{D_1}\widetilde{D_2} - \widetilde{D_3}\right)\widetilde{D_3} - \widetilde{D_1}^2\widetilde{D_4} > 0$. Thus, Routh-Hurwitz criterion yields that all roots of the polynomial $f(\gamma)$ have negative real parts. This follows that all eigenvalues $\gamma_{11}, \gamma_{12}, \gamma_{13}, \gamma_{14}$ have negative real parts. Hence, all eigenvalues of the Jacobian matrix $J(E_0)$ have negative real parts. It is concluded that, the disease-free equilibrium point E_0 is locally asymptotically stable if and only if $R_0 < 1$.

Theorem 4. *The endemic equilibrium point E_1 is locally asymptotically stable if and only if $R_0 > 1$.*

Proof. At the endemic equilibrium point E_1, the Jacobian matrix $J(E_1)$ has 14 eigenvalues which are given below $\gamma_1^* = -\Omega_f, \quad \gamma_2^* = -\Psi_f, \quad \gamma_3^* = -\Phi_f, \quad \gamma_4^* = -\Omega_m, \quad \gamma_5^* = -\Psi_m, \quad \gamma_6^* = -\Phi_m, \quad \gamma_{7,8}^* = \zeta$ and the roots of the following degree six polynomial are the last six eigenvalues

$$f(\gamma^*) = \gamma^{*6} + \widetilde{C_1}\gamma^{*5} + \widetilde{C_2}\gamma^{*4} + \widetilde{C_3}\gamma^{*3} + \widetilde{C_4}\gamma^{*2} + \widetilde{C_5}\gamma^* + \widetilde{C_6},$$

where

$$\widetilde{C_1} = \widetilde{W_1}I_m^* + \widetilde{W_2}I_f^* + \Delta_f + \Delta_m + \Gamma_f + \Gamma_m + 2\eta$$

$$\begin{aligned}
\widetilde{C_2} &= \widetilde{W_1}\widetilde{W_2}I_f^*I_m^* + \Delta_f\widetilde{W_1}I_m^* + \Delta_f\widetilde{W_2}I_f^* + \Delta_m\widetilde{W_1}I_m^* + \Delta_m\widetilde{W_2}I_f^* + \Gamma_f\widetilde{W_1}I_m^* + \Gamma_f\widetilde{W_2}I_f^* \\
&+ \Gamma_m\widetilde{W_1}I_m^* + \Gamma_m\widetilde{W_2}I_f^* + \widetilde{W_1}\eta I_m^* + \widetilde{W_2}\eta I_f^* + \Delta_f\Delta_m + \Delta_f\Gamma_f + \Delta_f\Gamma_m + 2\eta\Delta_f + \Delta_m\Gamma_f \\
&+ \Delta_m\Gamma_m + 2\eta\Delta_m + \Gamma_f\Gamma_m + 2\eta\Gamma_f + 2\eta\Gamma_m + \eta^2,
\end{aligned}$$

$$\widetilde{C_3} = \widetilde{W_1}\widetilde{W_2}\Delta_f I_f^* I_m^* + \widetilde{W_1}\widetilde{W_2}\Delta_m I_f^* I_m^* + \widetilde{W_1}\widetilde{W_2}\Gamma_f I_f^* I_m^* + \widetilde{W_1}\widetilde{W_2}\Gamma_m I_f^* I_m^* + \widetilde{W_1}\Delta_f\Delta_m I_m^*$$
$$+ \widetilde{W_2}\Delta_f\Delta_m I_f^* + \widetilde{W_1}\Delta_f\Gamma_f I_m^* + \widetilde{W_2}\Delta_f\Gamma_f I_f^* + \widetilde{W_1}\Delta_f\Gamma_m I_m^* + \widetilde{W_2}\Delta_f\Gamma_m I_f^* + \widetilde{W_1}\Delta_f\eta I_m^*$$
$$+ \widetilde{W_2}\Delta_f\eta I_f^* + \widetilde{W_1}\Delta_m\Gamma_f I_m^* + \widetilde{W_2}\Delta_m\Gamma_f I_f^* + \widetilde{W_1}\Delta_m\Gamma_m I_m^* + \widetilde{W_2}\Delta_m\Gamma_m I_f^* + \widetilde{W_1}\Delta_m\eta I_m^*$$
$$+ \widetilde{W_2}\Delta_m\eta I_f^* + \widetilde{W_1}\Gamma_f\Gamma_m I_m^* + \widetilde{W_2}\Gamma_f\Gamma_m I_f^* + \widetilde{W_1}\Gamma_f\eta I_m^* + \widetilde{W_2}\Gamma_f\eta I_f^* + \widetilde{W_1}\Gamma_m\eta I_m^*$$
$$+ \widetilde{W_2}\Gamma_m\eta I_f^* + \Delta_f\Delta_m\Gamma_f + \Delta_f\Delta_m\Gamma_m + 2\Delta_f\Delta_m\eta + \Delta_f\Gamma_f\Gamma_m + 2\Delta_f\Gamma_f\eta + 2\Delta_f\Gamma_m\eta$$
$$+ \Delta_f\eta^2 + \Delta_m\Gamma_f\Gamma_m + 2\Delta_m\Gamma_f\eta + 2\Delta_m\Gamma_m\eta + \Delta_m\eta^2 + 2\Gamma_f\Gamma_m\eta + \Gamma_f\eta^2 + \Gamma_m\eta^2,$$

$$\widetilde{C_4} = \Delta_f\Delta_m\widetilde{W_1}\widetilde{W_2}I_f^* I_m^* + \Delta_f\Gamma_f\widetilde{W_1}\widetilde{W_2}I_f^* I_m^* + \Delta_f\Gamma_m\widetilde{W_1}\widetilde{W_2}I_f^* I_m^* + \Delta_m\Gamma_f\widetilde{W_1}\widetilde{W_2}I_f^* I_m^*$$
$$+ \Delta_m\Gamma_m\widetilde{W_1}\widetilde{W_2}I_f^* I_m^* + \Gamma_f\Gamma_m\widetilde{W_1}\widetilde{W_2}I_f^* I_m^* - \widetilde{W_1}\widetilde{W_2}\alpha_f\alpha_m S_f^* S_m^* + \Delta_f\Delta_m\Gamma_f\widetilde{W_1}I_m^*$$
$$+ \Delta_f\Delta_m\Gamma_f\widetilde{W_2}I_f^* + \Delta_f\Delta_m\Gamma_m\widetilde{W_1}I_m^* + \Delta_f\Delta_m\Gamma_m\widetilde{W_2}I_f^* + \Delta_f\Delta_m\widetilde{W_1}\eta I_m^* + \Delta_f\Delta_m\widetilde{W_2}\eta I_f^*$$
$$+ \Delta_f\Gamma_f\Gamma_m\widetilde{W_1}I_m^* + \Delta_f\Gamma_f\Gamma_m\widetilde{W_2}I_f^* + \Delta_f\Gamma_f\widetilde{W_1}\eta I_m^* + \Delta_f\Gamma_f\widetilde{W_2}\eta I_f^* + \Delta_f\Gamma_m\widetilde{W_1}\eta I_m^*$$
$$+ \Delta_f\Gamma_m\widetilde{W_2}\eta I_f^* + \Delta_m\Gamma_f\Gamma_m\widetilde{W_1}I_m^* + \Delta_m\Gamma_f\Gamma_m\widetilde{W_2}I_f^* + \Delta_m\Gamma_f\widetilde{W_1}\eta I_m^* + \Delta_m\Gamma_f\widetilde{W_2}\eta I_f^*$$
$$+ \Delta_m\Gamma_m\widetilde{W_1}\eta I_m^* + \Delta_m\Gamma_m\widetilde{W_2}\eta I_f^* + \Gamma_f\Gamma_m\widetilde{W_1}\eta I_m^* + \Gamma_f\Gamma_m\widetilde{W_2}\eta I_f^* + \Delta_f\Delta_m\Gamma_f\Gamma_m$$
$$+ 2\Delta_f\Delta_m\Gamma_f\eta + 2\Delta_f\Delta_m\Gamma_m\eta + \Delta_f\Delta_m\eta^2 + 2\Delta_f\Gamma_f\Gamma_m\eta + \Delta_f\Gamma_f\eta^2 + \Delta_f\Gamma_m\eta^2$$
$$+ 2\Delta_m\Gamma_f\Gamma_m\eta + \Delta_m\Gamma_f\eta^2 + \Delta_m\Gamma_m\eta^2 + \Gamma_f\Gamma_m\eta^2,$$

$$\widetilde{C_5} = \Delta_f\Delta_m\Gamma_f\widetilde{W_1}\widetilde{W_2}I_f^* I_m^* + \Delta_f\Delta_m\Gamma_m\widetilde{W_1}\widetilde{W_2}I_f^* I_m^* + \Delta_f\Gamma_f\Gamma_m\widetilde{W_1}\widetilde{W_2}I_f^* I_m^* + \Delta_m\Gamma_f\Gamma_m\widetilde{W_1}\widetilde{W_2}I_f^* I_m^*$$
$$- 2\widetilde{W_1}\widetilde{W_2}\alpha_f\alpha_m\eta S_f^* S_m^* + \Delta_f\Delta_m\Gamma_f\Gamma_m\widetilde{W_1}I_m^* + \Delta_f\Delta_m\Gamma_f\Gamma_m\widetilde{W_2}I_f^* + \Delta_f\Delta_m\Gamma_f\widetilde{W_1}\eta I_m^*$$
$$+ \Delta_f\Delta_m\Gamma_f\widetilde{W_2}\eta I_f^* + \Delta_f\Delta_m\Gamma_m\widetilde{W_1}\eta I_m^* + \Delta_f\Delta_m\Gamma_m\widetilde{W_2}\eta I_f^* + \Delta_f\Gamma_f\Gamma_m\widetilde{W_1}\eta I_m^* + \Delta_f\Gamma_f\Gamma_m\widetilde{W_2}\eta I_f^*$$
$$+ \Delta_m\Gamma_f\Gamma_m\widetilde{W_1}\eta I_m^* + \Delta_m\Gamma_f\Gamma_m\widetilde{W_2}\eta I_f^* + 2\Delta_f\Delta_m\Gamma_f\Gamma_m\eta + \Delta_f\Delta_m\Gamma_f\eta^2 + \Delta_f\Delta_m\Gamma_m\eta^2$$
$$+ \Delta_f\Gamma_f\Gamma_m\eta^2 + \Delta_m\Gamma_f\Gamma_m\eta^2,$$

$$\widetilde{C_6} = \Delta_f\Delta_m\Gamma_f\Gamma_m\widetilde{W_1}\widetilde{W_2}I_f^* I_m^* - \widetilde{W_1}\widetilde{W_2}\alpha_f\alpha_m\eta^2 S_f^* S_m^* + \Delta_f\Delta_m\Gamma_f\Gamma_m\widetilde{W_1}\eta I_m^* + \Delta_f\Delta_m\Gamma_f\Gamma_m\widetilde{W_2}\eta I_f^*$$
$$+ \Delta_f\Delta_m\Gamma_f\Gamma_m\eta^2,$$

here,

$$\widetilde{W_1} = (1-\epsilon\kappa)\beta_f > 0, \widetilde{W_2} = (1-\epsilon\kappa)\beta_m > 0$$

If $R_0 > 1$, it is easy to show that $\widetilde{C_1}, \widetilde{C_2}, \widetilde{C_3}, \widetilde{C_4}, \widetilde{C_5}, \widetilde{C_6}$ are all positive as follows: Clearly $\widetilde{C_1} > 0, \widetilde{C_2} > 0$, and $\widetilde{C_3} > 0$. The only negative term in $\widetilde{C_4}$ is $\widetilde{W_1}\widetilde{W_2}\alpha_f\alpha_m S_f^* S_m^*$, and $\Delta_f\Delta_m\Gamma_f\Gamma_m - \widetilde{W_1}\widetilde{W_2}\alpha_f\alpha_m S_f^* S_m^* = 0$. Hence $\widetilde{C_4} > 0$. In $\widetilde{C_5}$ there is only one negative term which are $2\widetilde{W_1}\widetilde{W_2}\alpha_f\alpha_m\eta S_f^* S_m^*$, and $2\Delta_f\Delta_m\Gamma_f\Gamma_m\eta - 2\widetilde{W_1}\widetilde{W_2}\alpha_f\alpha_m\eta S_f^* S_m^* = 0$. Thus,

$\widetilde{C_5} > 0$. For $\widetilde{C_6}$, since $\Delta_f \Delta_m \Gamma_f \Gamma_m \eta^2 - \widetilde{W_1}\widetilde{W_2}\alpha_f \alpha_m \eta^2 S_f^* S_m^* = 0$, it follows that $\widetilde{C_6} > 0$. The necessary condition for stability of Routh-Hurwitz Criterion holds as $\widetilde{C_l} > 0$, for $i = 1, 2, 3, 4, 5, 6$ if and only if $R_0 > 1$. The Routh-Hurwitz Criterion array for $f(\gamma^*)$ is given by

$$\begin{array}{lccccc} \lambda^{*6}: & a_0 & a_2 & a_4 & a_6 & 0 \\ \lambda^{*5}: & a_1 & a_3 & a_5 & 0 & 0 \\ \lambda^{*4}: & b_1 & b_2 & b_3 & b_4 & 0 \\ \lambda^{*3}: & c_1 & c_2 & c_3 & c_4 & 0 \\ \lambda^{*2}: & d_1 & d_2 & d_3 & d_4 & 0 \\ \lambda^{*1}: & e_1 & e_2 & e_3 & e_4 & 0 \\ \lambda^{*0}: & f_1 & f_2 & f_3 & f_4 & 0 \end{array}$$

where

$$a_0 = 1, a_1 = \widetilde{c_1}, \mathrm{a}_2 = \widetilde{C_2}, a_3 = \widetilde{C_3}, a_4 = \widetilde{C_4}, a_5 = \widetilde{C_5}, a_6 = \widetilde{c_6}, a_7 = 0, b_1 = \frac{a_1 a_2 - a_0 a_3}{a_1},$$
$$b_2 = \frac{a_1 a_4 - a_0 a_5}{a_1}, b_3 = \frac{a_1 a_6 - a_0 a_7}{a_1}, b_4 = 0, c_1 = \frac{b_1 a_3 - a_1 b_2}{b_1}, c_2 = \frac{b_1 a_5 - a_1 b_3}{b_1},$$
$$c_3 = \frac{b_1 a_7 - a_1 b_4}{b_1}, c_4 = 0, d_1 = \frac{c_1 b_2 - b_1 c_2}{c_1}, d_2 = \frac{c_1 b_3 - b_1 c_3}{c_1}, d_3 = \frac{c_1 b_4 - b_1 c_4}{c_1}, d_4 = 0,$$
$$e_1 = \frac{d_1 c_2 - c_1 d_2}{d_1}, e_2 = \frac{d_1 c_3 - c_1 d_3}{d_1}, e_3 = \frac{d_1 c_4 - c_1 d_4}{d_1}, e_4 = 0, f_1 = \frac{e_1 d_2 - d_1 e_2}{e_1},$$
$$f_2 = \frac{e_1 d_3 - d_1 e_3}{e_1}, f_3 = \frac{e_1 d_4 - d_1 e_4}{e_1}, f_4 = 0.$$

For $R_0 > 1$ all entries of the first column of Routh-Hurwitz Criterion array are positive. Therefore, all roots of $f(\gamma^*)$ have negative real parts. Thus, it is concluded that all eigenvalues γ_i^* for $i = 1, 2, \ldots 14$ have negative real parts. Therefore, the endemic equilibrium point E_1 is locally asymptotically stable if and only if $R_0 > 1$.

6. Sensitivity Analysis of The Model and Basic Reproduction Number R_0

Sensitivity analysis is used to determine how variables react to change in the parameters. The normalized forward sensitivity index of R_0 with respect to a parameter p is as follows:

$$\varepsilon_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}$$

We already shown that the expression of R_0 is given by

$$R_0 = \frac{\Lambda(1 - \epsilon\kappa)}{\eta} \sqrt{\frac{\alpha_f \alpha_m \beta_f \beta_m}{(\mu + \eta + \gamma_f + \theta_f)(\mu + \eta + \gamma_m + \theta_m)(\alpha_f + \eta)(\alpha_m + \eta)}}.$$

R_0 's normalized forward sensitivity indices for its parameters are as follows:

$$\varepsilon_{\Lambda}^{R_0} = \frac{\partial R_0}{\partial \Lambda} \times \frac{\Lambda}{R_0} = 1, \qquad \varepsilon_{\alpha_f}^{R_0} = \frac{\partial R_0}{\partial \alpha_f} \times \frac{\alpha_f}{R_0} = \frac{\eta}{2(\alpha_f + \eta)},$$

$$\varepsilon_{\alpha_m}^{R_0} = \frac{\partial R_0}{\partial \alpha_m} \times \frac{\alpha_m}{R_0} = \frac{\eta}{2(\alpha_f + \eta)}, \qquad \varepsilon_{\beta_f}^{R_0} = \frac{\partial R_0}{\partial \beta_f} \times \frac{\beta_f}{R_0} = \frac{1}{2},$$

$$\varepsilon_{\beta_m}^{R_0} = \frac{\partial R_0}{\partial \beta_m} \times \frac{\beta_m}{R_0} = \frac{1}{2}, \qquad \varepsilon_{\kappa}^{R_0} = \frac{\partial R_0}{\partial \kappa} \times \frac{\kappa}{R_0} = \frac{\epsilon\kappa}{\epsilon\kappa - 1},$$

$$\varepsilon_{\epsilon}^{R_0} = \frac{\partial R_0}{\partial \epsilon} \times \frac{\epsilon}{R_0} = \frac{\epsilon\kappa}{\epsilon\kappa - 1},$$

$$\varepsilon_{\mu}^{R_0} = \frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0} = -\frac{(2\mu + 2\eta + \gamma_f + \theta_f + \gamma_m + \theta_m)\,\mu}{2(\mu + \eta + \gamma_f + \theta_f)(\mu + \eta + \gamma_m + \theta_m)},$$

$$\varepsilon_{\gamma_f}^{R_0} = \frac{\partial R_0}{\partial \gamma_f} \times \frac{\gamma_f}{R_0} = -\frac{\gamma_f}{2(\mu + \eta + \gamma_f + \theta_f)},$$

$$\varepsilon_{\gamma_m}^{R_0} = \frac{\partial R_0}{\partial \gamma_m} \times \frac{\gamma_m}{R_0} = -\frac{\gamma_m}{2(\mu + \eta + \gamma_m + \theta_m)},$$

$$\varepsilon_{\theta_f}^{R_0} = \frac{\partial R_0}{\partial \theta_f} \times \frac{\theta_f}{R_0} = -\frac{\theta_f}{2(\mu + \eta + \gamma_f + \theta_f)},$$

$$\frac{\partial R_0}{\partial \theta_m} \times \frac{\theta_m}{R_0} = -\frac{\theta_m}{2(\mu + \eta + \gamma_m + \theta_m)},$$

$$\varepsilon_{\eta}^{R_0} = \frac{\partial R_0}{\partial \eta} \times \frac{\eta}{R_0} = -\frac{6\eta^4 + 5\eta^3 h_1 + 4\eta^2 \mu h_2 + 4\eta^2 h_3 + 3\eta\mu h_4 + 3\eta\alpha_f\alpha_m h_5 + 3\eta h_6 + 2\alpha_f\alpha_m h_7}{2(\alpha_f + \eta)(\alpha_m + \eta)(\mu + \eta + \gamma_f + \theta_f)(\mu + \eta + \gamma_m + \theta_m)}$$

where,

$$\begin{aligned}
h_1 &= 2\mu + \alpha_f\alpha_m + \gamma_f + \gamma_m + \theta_f + \theta_m \\
h_2 &= \mu + 2\alpha_f + 2\alpha_m + \gamma_f + \gamma_m + \theta_f + \theta_m \\
h_3 &= \alpha_f\alpha_m + (\alpha_f + \alpha_m)(\gamma_f + \gamma_m + \theta_f + \theta_m) + \gamma_f\gamma_m + \gamma_f\theta_m + \gamma_m\theta_f + \theta_f\theta_m \\
h_4 &= (\alpha_f + \alpha_m)(\mu + \gamma_f + \gamma_m + \theta_f + \theta_m) + 2\alpha_f\alpha_m \\
h_5 &= \gamma_f + \gamma_m + \theta_f + \theta_m \\
h_6 &= (\alpha_f + \alpha_m)(\gamma_f\gamma_m + \gamma_f\theta_m + \gamma_m\theta_f + \theta_f\theta_m) \\
h_7 &= \mu^2 + \mu\gamma_f + \mu\gamma_m + \mu\theta_f + \mu\theta_m + \gamma_f\gamma_m + \gamma_f\theta_m + \gamma_m\theta_f + \theta_f\theta_m
\end{aligned}$$

In the below table, the sensitivity index for the estimated values of parameters are given

From the information in Table 2, we can observe positive sensitivity indices for the parameters $\Lambda, \alpha_f, \alpha_m, \beta_f$, and β_m while the other parameters $\kappa, \epsilon, \mu, \eta, \gamma_f, \gamma_m, \theta_f$, and θ_m exhibit negative sensitivity indices. Additionally, these parameters play a crucial role in determining the value of the reproduction number R_0, which aids in analyzing the persistence or extinction of the disease within the population. It's noteworthy that an

Table 2: Sensitivity Index of R_0

Parameters	Estimated Values	Sensitivity Index	Sign	Sources
Λ	100000	1	Positive	[22]
α_f	0.8	0.01350036488	Positive	Assumed
α_m	0.75	0.01437451438	Positive	Assumed
β_f	0.015	0.5	Positive	[22]
β_m	0.025	0.5	Positive	[22]
κ	$[0, 1]$	-0.2500000000	Negative	[23]
ϵ	$[0, 1]$	-0.25000000000	Negative	[23]
μ	0.333	-0.3134162666	Negative	[25]
η	0.0222	-1.048769296	Negative	[24]
γ_f	0.675	-0.2934272300	Negative	[22]
γ_m	0.512	-0.2593192868	Negative	[22]
θ_f	0.12	-0.05216484090	Negative	Assumed
θ_m	0.12	-0.06077795785	Negative	Assumed

increase in positive parameter values leads to an elevation in the value of R_0, indicating a substantial rise in disease transmission. In contrast, increasing negative parameter values reduces the reproduction number to some amount.

We now analyze the local sensitivities. In the model 1, there are fourteen compartments and nineteen parameters, we can use three different methods to calculate the local sensitivities which are fullnormalization, half-normalization, and non-normalization.

First, the non-normalization's equation is given by

$$\varepsilon_{\pi_j}^{a_i} = \frac{\partial a_i}{\partial \pi_j},$$

where $\varepsilon_{\pi_j}^{a_i}$ denotes the sensitivity coefficient of each variable a_i with respect to each parameter π_j. The half-normalization sensitivities formula is therefore defined as follows:

$$\varepsilon_{\pi_j}^{a_i} = \frac{\partial a_i}{\partial \pi_j} \times \frac{1}{a_i}$$

Lastly, full-normalization sensitivities equation is defined by

$$\varepsilon_{\pi_j}^{a_i} = \frac{\partial a_i}{\partial \pi_j} \times \frac{\pi_j}{a_i}$$

We have employed MATLAB for computational simulations using the estimated parameters and initial variables provided in Table 1. The work's findings offer a significant advancement in our understanding of the dynamics of the model. This aids in the identification of crucial model parameters as well as the ways in which the parameters impact each model state.

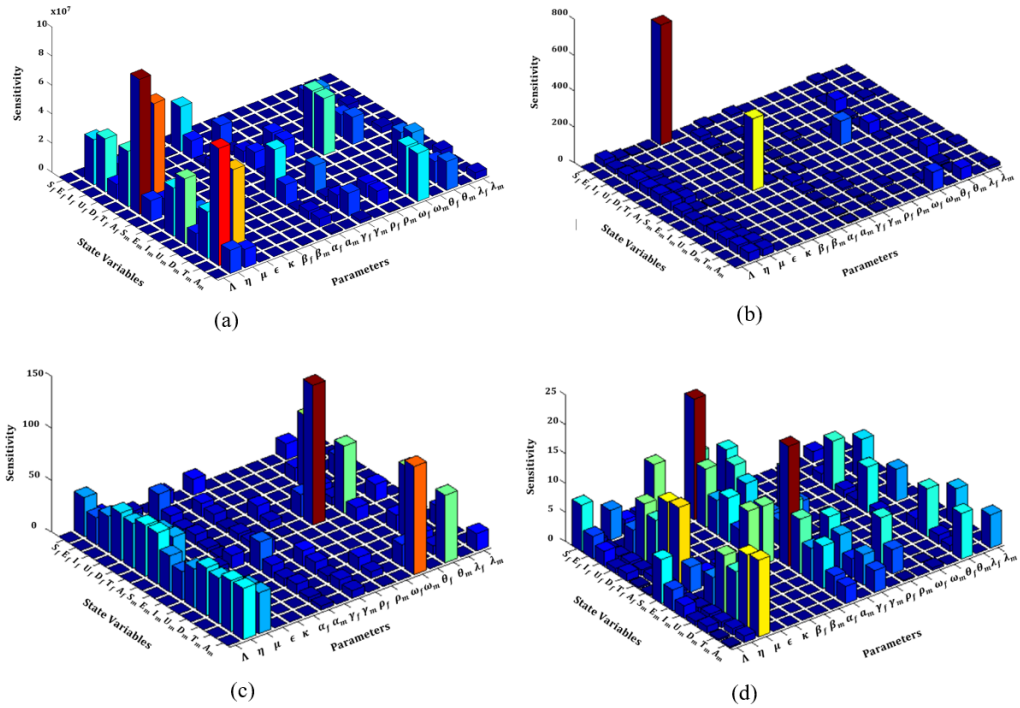

Figure 2: Local sensitivity analysis using techniques non-normalization (a), half-normalization (b,c), and full-normalization (d)

Figure 2 illustrates that the non-normalization technique significantly affects the sensitivity of almost all of the model's variables to the model parameters $\eta, \mu, \alpha_m, \gamma_f, \gamma_m, \theta_f$, and θ_m, while they are less sensitive with the rest parameters of the model; see Figure 2(a). In addition, the half-normalization method depicts that the model states S_f and S_m are highly sensitive to the parameters of the model β_f and β_m respectively, whereas the other system variables are less sensitive to the system parameters; see Figures 2(b)and 2(c). Finally, by utilizing the full-normalization technique demonstrates that almost all model variables are extremely sensitive to the parameters of the model $\Lambda, \eta, \mu, \alpha_f, \alpha_m, \gamma_f, \gamma_m, \theta_f, \theta_m, \lambda_f$, and λ_m, while they are less sensitive to the other model parameters, as shown in Figure 2(d).

7. Optimal Control

In this section, we incorporate three time-dependent control strategies to our model 1 with the goal of determining the best effective method for controlling and reducing the number of people with AIDS within a specific period of time. The optimum control problem introduces the use of the following time-dependent control measures.

- Preventive control measure: symbolized by u_1 include HIV/AIDS awareness and

education campaigns through health centers and social media to safeguard the individuals who are unaware of their susceptibility to HIV.

- Screening control measure: denoted by u_2 to assist undiagnosed infected persons in screening themselves.

- Treatment control measure, represented by u_3, is utilized by patients to reduce viral load in the body and prevent virus progression.

Integrating the control measures u_1, u_2 and u_3 into model 1 yields the optimal control model that results as follows:

$$\begin{aligned}
\frac{dS_f}{dt} &= \Lambda - (1-u_1)(1-\kappa\epsilon)\beta_f I_m S_f - \eta S_f \\
\frac{dS_m}{dt} &= \Lambda - (1-u_1)(1-\kappa\epsilon)\beta_m I_f S_m - \eta S_m \\
\frac{dE_f}{dt} &= (1-u_1)(1-\kappa\epsilon)\beta_f I_m S_f - (\alpha_f + \eta) E_f \\
\frac{dE_m}{dt} &= (1-u_1)(1-\kappa\epsilon)\beta_m I_f S_m - (\alpha_m + \eta) E_m \\
\frac{dI_f}{dt} &= \alpha_f E_f - (u_2 + \mu + \eta + \gamma_f + \theta_f) I_f \\
\frac{dl_m}{dt} &= \alpha_m E_m - (u_2 + \mu + \eta + \gamma_m + \theta_m) I_m \\
\frac{dD_f}{dt} &= (u_2 + \gamma_f) I_f - (u_3 + \mu + \eta + \rho_f) D_f \\
\frac{dD_m}{dt} &= (u_2 + \gamma_m) I_m - (u_3 + \mu + \eta + \rho_m) D_m \\
\frac{dT_f}{dt} &= u_3 A_f + (u_3 + \rho_f) D_f - (\mu + \eta + (1-u_3)\omega_f) T_f \\
\frac{dT_m}{dt} &= u_3 A_m + (u_3 + \rho_m) D_m - (\mu + \eta + (1-u_3)\omega_m) T_m \\
\frac{dU_f}{dt} &= \theta_f I_f - (\mu + \eta + \lambda_f) U_f \\
\frac{dU_m}{dt} &= \theta_m I_m - (\mu + \eta + \lambda_m) U_m \\
\frac{dt_f}{dt} &= \lambda_f U_f + (1-u_3)\omega_f T_f - (u_3 + \mu + \eta) A_f \\
\frac{dA_m}{dt} &= \lambda_m U_m + (1-u_3)\omega_m T_m - (u_3 + \mu + \eta) A_m
\end{aligned} \tag{4}$$

where
$E_f(0), E_m(0), I_f(0), I_m(0), D_f(0), D_m(0), T_f(0), T_m(0), U_f(0), U_m(0), A_f(0), A_m(0) \geq 0.$

To analyze the levels of optimal controls, it is necessary to establish the set U of Lebesgue measurable controls

$$U = \{(u_1(t), u_2(t), u_3(t)) : 0 \leq u_1 \leq 1, 0 \leq u_2 \leq 1, 0 \leq u_3 \leq 1, 0 \leq t \leq t_f\}.$$

The objective functional used in this paper extends the HIV control framework [1, 18]. by incorporating gender-stratified compartments ($S_f, E_f, I_f, D_f, T_f, U_f, A_f, S_m, E_m, I_m, D_m, T_m, U_m, A_m$) and three intervention controls. This work adopts the Pontryagin Maximum Principle discussed in [26] to derive necessary conditions for optimality.

The proposed control method applies the Pontryagin Maximum Principle to a gender-stratified HIV model, enabling the derivation of optimal intervention strategies across three control measures: prevention, screening, and treatment. Unlike traditional control approaches that often assume uniform populations or apply single interventions in isolation, this method accounts for gender-specific disease dynamics and interaction patterns. This stratification allows for more precise targeting of interventions, improving the effectiveness of resource allocation. Additionally, by formulating the control problem as an optimal control system, the method provides a rigorous mathematical framework for identifying time-dependent strategies that minimize infection levels.

Our objective is to determine the optimal controls u_1^*, u_2^* and u_3^* along with the optimal solutions S_f^*, E_f^*, I_f^*, D_f^*, T_f^*, U_f^*, A_f^*, S_m^*, E_m^*, I_m^*, D_m^*, T_m^*, U_m^* and A_m^* by setting the terminal time t_f in order to minimize the objective functional J defined by:

$$J(u_1, u_2, u_3) = \min_{u_1,u_2,u_3} \int_0^{t_f} \begin{pmatrix} b_1E_f + b_2I_f + b_3D_f + b_4T_f + b_5U_f + b_6A_f + b_7E_m + b_8I_m + \\ b_9D_m + b_{10}T_m + b_{11}U_m + b_{12}A_m + \frac{1}{2}\left(w_1u_1^2 + w_2u_2^2 + w_3u_3^2\right) \end{pmatrix} dt \tag{5}$$

Here, $b_i, i = 1, \ldots, 12$ and w_1, w_2, and w_3 are positive constants. The terms $0.5w_1u_1^2$, $0.5w_2u_2^2$ and $0.5w_3u_3^2$ represent costs associated with the controls. Our goal is to reduce the population sizes of infected
compartments by intervening with control measures $u_1(t), u_2(t)$ and $u_3(t)$ as well as the related costs. We have the following for the three optimal controllers u_1^*, u_2^* and u_3^* :

$$J(u_1^*, u_2^*, u_3^*) = \min\{J(u_1, u_2, u_3), u_1, u_2, u_3 \in U\}$$

Theorem 5. *(Existence of optimal control solution)*
There is the optimal control (u_1^*, u_2^*, u_3^*) along with their associated state variable solutions S_f^*, E_f^*, I_f^*, D_f^*, T_f^*, U_f^*, A_f^*, S_m^*, E_m^*, I_m^*, D_m^*, T_m^*, U_m^* and A_m^* for the state initial value problem defined in (4) and (5) that minimizes $J(u_1, u_2, u_3)$ over the set U.

Proof. We apply the method used in [27] theorem 4 to proof the existence of optimal control solution.
By applying the Pontryagin Maximum Principle (PMP) as outlined in [26], we derive the essential conditions that are met by the optimal pair. Hence, the Hamiltonian function

(H) can be obtained by this principle which is defined as

$$\begin{aligned}
&H\left(S_f, E_f, I_f, D_f, T_f, U_f, A_f, S_m, E_m, I_m, D_m, T_m, U_m, A_m, u_1, u_2, u_3\right) = b_1E_f + b_2I_f + b_3D_f \\
&+ b_4T_f + b_5U_f + b_6A_f + b_7E_m + b_8I_m + b_9D_m + b_{10}T_m + b_{11}U_m + b_{12}A_m \\
&+ \frac{1}{2}\left(w_1u_1^2 + w_2u_2^2 + w_3u_3^2\right) + \lambda_1\frac{dS_f}{dt} + \lambda_2\frac{dE_f}{dt} + \lambda_3\frac{dI_f}{dt} + \lambda_4\frac{dD_f}{dt} + \lambda_5\frac{dT_f}{dt} + \lambda_6\frac{dU_f}{dt} \\
&+ \lambda_7\frac{dA_f}{dt} + \lambda_8\frac{dS_m}{dt} + \lambda_9\frac{dE_m}{dt} + \lambda_{10}\frac{dI_m}{dt} + \lambda_{11}\frac{dD_m}{dt} + \lambda_{12}\frac{dT_m}{dt} + \lambda_{13}\frac{dU_m}{dt} + \lambda_{14}\frac{dA_m}{dt}
\end{aligned} \tag{6}$$

Here, λ_i, where $i = 1, 2, \ldots, 14$, denotes the adjoint variables associated with the state variables S_f,E_f, I_f, D_f, T_f, U_f, A_f, S_m, E_m, I_m, D_m, T_m, U_m and A_m, respectively, which must be ascertained utilising Pontryagin's maximum principle to establish the existence of optimal pairs.

Theorem 6. *Let $S_f, E_f, I_f, D_f, T_f, U_f, A_f, S_m, E_m, I_m, D_m, T_m, U_m$ and A_m denote optimum state solutions associated with the optimal control variables u_1, u_2 and u_3 within the optimal control model. Correspondingly, there exist co-state variables λ_i, where $i = 1, 2, \ldots, 14$ such that:*

$$\frac{d\lambda_i}{dt} = -\frac{\partial H}{\partial j}$$

Considering transversality or final time conditions, where $\lambda_i\left(t_f\right) = 0$ for $i = 1, 2, \ldots, 14$, $j = S_f$, E_f, I_f, D_f, T_f, U_f, A_f, S_m, E_m, I_m, D_m, T_m, U_m, A_m, and H is the Hamiltonian function as defined in equation 6. Additionally, the optimal controls u_1^*, u_2^* and u_3^* are

$$u_1^* = \max\left\{\min\left\{1, \frac{(1-k\epsilon)\left[\beta_f I_m S_f(\lambda_2-\lambda_1)+\beta_m I_f S_m(\lambda_9-\lambda_8)\right]}{w_1}\right\}, 0\right\},$$

$$u_2^* = \max\left\{\min\left\{1, \frac{I_f(\lambda_3-\lambda_4)+I_m(\lambda_{10}-\lambda_{11})}{w_2}\right\}, 0\right\},$$

$$u_3^* = \max\left\{\min\left\{1, \frac{(\lambda_4-\lambda_5)D_f+(\lambda_7-\lambda_5)\left(A_f+\omega_f T_f\right)+(\lambda_{11}-\lambda_{12})D_m+(\lambda_{14}-\lambda_{12})(A_m+\omega_m T_m)}{w_3}\right\}, 0\right\}.$$

Proof. The Hamiltonian H corresponding to the optimum model is expressed as

$$\begin{aligned}
H &= b_1E_f + b_2I_f + b_3D_f + b_4T_f + b_5U_f + b_6A_f + b_7E_m + b_8I_m + b_9D_m + b_{10}T_m \\
&+ b_{11}U_m + b_{12}A_m + \frac{1}{2}\left(w_1u_1^2 + w_2u_2^2 + w_3u_3^2\right) + \lambda_1\frac{dS_f}{dt} + \lambda_2\frac{dE_f}{dt} + \lambda_3\frac{dI_f}{dt} + \lambda_4\frac{dD_f}{dt} \\
&+ \lambda_5\frac{dT_f}{dt} + \lambda_6\frac{dU_f}{dt} + \lambda_7\frac{dA_f}{dt} + \lambda_8\frac{dS_m}{dt} + \lambda_9\frac{dE_m}{dt} + \lambda_{10}\frac{dI_m}{dt} + \lambda_{11}\frac{dD_m}{dt} + \lambda_{12}\frac{dT_m}{dt} \\
&+ \lambda_{13}\frac{dU_m}{dt} + \lambda_{14}\frac{dA_m}{dt}
\end{aligned}$$

So, it can be written as

$$
\begin{aligned}
H &= b_1E_f + b_2I_f + b_3D_f + b_4T_f + b_5U_f + b_6A_f + b_7E_m + b_8I_m + b_9D_m + b_{10}T_m + b_{11}U_m \\
&+ b_{12}A_m + \frac{1}{2}\left(w_1u_1^2 + w_2u_2^2 + w_3u_3^2\right) + \lambda_1\left(\Lambda - (1-u_1)(1-\kappa\epsilon)\beta_f I_m S_f - \eta S_f\right) \\
&+ \lambda_2\left((1-u_1)(1-\kappa\epsilon)\beta_f I_m S_f - (\alpha_f + \eta)E_f\right) + \lambda_3\left(\alpha_f E_f - (u_2 + \mu + \eta + \gamma_f + \theta_f)I_f\right) \\
&+ \lambda_4\left((u_2 + \gamma_f)I_f - (u_3 + \mu + \eta + \rho_f)D_f\right) + \lambda_5\left(u_3A_f + (u_3 + \rho_f)D_f - (\mu + \eta + (1-u_3)\omega_f)T_f\right) \\
&+ \lambda_6\left(\theta_f I_f - (\mu + \eta + \lambda_f)U_f\right) + \lambda_7\left(\lambda_f U_f + (1-u_3)\omega_f T_f - (u_3 + \mu + \eta)A_f\right) \\
&+ \lambda_8\left(\Lambda - (1-u_1)(1-\kappa\epsilon)\beta_m I_f S_m - \eta S_m\right) + \lambda_9\left((1-u_1)(1-\kappa\epsilon)\beta_m I_f S_m - (\alpha_m + \eta)E_m\right) \\
&+ \lambda_{10}\left(\alpha_m E_m - (u_2 + \mu + \eta + \gamma_m + \theta_m)I_m\right) + \lambda_{11}\left((u_2 + \gamma_m)I_m - (u_3 + \mu + \eta + \rho_m)D_m\right) \\
&+ \lambda_{12}\left(u_3A_m + (u_3 + \rho_m)D_m - (\mu + \eta + (1-u_3)\omega_m)T_m\right) + \lambda_{13}\left(\theta_m I_m - (\mu + \eta + \lambda_m)U_m\right) \\
&+ \lambda_{14}\left(\lambda_m U_m + (1-u_3)\omega_m T_m - (u_3 + \mu + \eta)A_m\right)
\end{aligned}
$$

Based on the PMP's second condition, adjoint variables λ_i, where $i = 1, 2, \ldots, 14$ exist such that

$$\frac{d\lambda_i}{dt} = -\frac{\partial H}{\partial j},$$

where i $= 1, 2..14,$ and $j = S_f, E_f, I_f, D_f, T_f, U_f, A_f, S_m, E_m, I_m, D_m, T_m, U_m, A_m$

Hence, the expressions for the adjoint equations can be formulated as:

$$
\begin{aligned}
\frac{d\lambda_1}{dt} &= -\frac{\partial H}{\partial S_f} = (1-u_1)(1-\kappa\epsilon)\beta_f I_m(\lambda_1 - \lambda_2) + \eta\lambda_1, \\
\frac{d\lambda_2}{dt} &= -\frac{\partial H}{\partial E_f} = -b_1 + \eta\lambda_2 + \alpha_f(\lambda_2 - \lambda_3), \\
\frac{d\lambda_3}{dt} &= -\frac{\partial H}{\partial I_f} = -b_2 + (\mu + \eta)\lambda_3 + (u_2 + \gamma_f)(\lambda_3 - \lambda_4) + \theta_f(\lambda_3 - \lambda_6) \\
&\qquad + (1-u_1)(1-\kappa\epsilon)\beta_m S_m(\lambda_8 - \lambda_9), \\
\frac{d\lambda_4}{dt} &= -\frac{\partial H}{\partial D_f} = -b_3 + (\mu + \eta)\lambda_4 + (u_3 + \rho_f)(\lambda_4 - \lambda_5), \\
\frac{d\lambda_5}{dt} &= -\frac{\partial H}{\partial T_f} = -b_4 + (\mu + \eta)\lambda_5 + (1-u_3)\omega_f(\lambda_5 - \lambda_7), \\
\frac{d\lambda_6}{dt} &= -\frac{\partial H}{\partial U_f} = -b_5 + (\mu + \eta)\lambda_6 + \lambda_f(\lambda_6 - \lambda_7) \\
\frac{d\lambda_7}{dt} &= -\frac{\partial H}{\partial A_f} = -b_6 + (u_3 + \mu + \eta)\lambda_7 - u_3\lambda_5 \\
\frac{d\lambda_8}{dt} &= -\frac{\partial H}{\partial S_m} = (1-u_1)(1-\kappa\epsilon)\beta_m I_f(\lambda_8 - \lambda_9) + \eta\lambda_8 \\
\frac{d\lambda_9}{dt} &= -\frac{\partial H}{\partial E_m} = -b_7 + \eta\lambda_9 + \alpha_m(\lambda_9 - \lambda_{10})
\end{aligned}
$$

$$\begin{aligned}
\frac{d\lambda_{10}}{dt} &= -\frac{\partial H}{\partial I_m} = -b_8 + (\mu+\eta)\lambda_{10} + (u_2+\gamma_m)(\lambda_{10}-\lambda_{11}) + \theta_m(\lambda_{10}-\lambda_{13}) \\
&\quad + (1-u_1)(1-\kappa\epsilon)\beta_f S_f(\lambda_1-\lambda_2), \\
\frac{d\lambda_{11}}{dt} &= -\frac{\partial H}{\partial D_m} = -b_9 + (\mu+\eta)\lambda_{11} + (u_3+\rho_m)(\lambda_{11}-\lambda_{12}), \\
\frac{d\lambda_{12}}{dt} &= -\frac{\partial H}{\partial T_m} = -b_{10} + (\mu+\eta)\lambda_{12} + (1-u_3)\omega_m(\lambda_{12}-\lambda_{14}) \\
\frac{d\lambda_{13}}{dt} &= -\frac{\partial H}{\partial U_m} = -b_{11} + (\mu+\eta)\lambda_{13} + \lambda_m(\lambda_{13}-\lambda_{14}) \\
\frac{d\lambda_{14}}{dt} &= -\frac{\partial H}{\partial A_m} = -b_{12} + (u_3+\mu+\eta)\lambda_{14} - u_3\lambda_{12}
\end{aligned}$$

Further, the optimality conditions give us

$$\left.\frac{\partial H}{\partial u_i}\right|_{u_i=u_i^*} = 0, \text{ where } i = 1,2,3$$

Then solving for u_1^*, u_2^* and u_3^*, we obtain

$u_1^* = \frac{(1-\kappa\epsilon)\left[\beta_f I_m S_f(\lambda_2-\lambda_1)+\beta_m I_f S_m(\lambda_9-\lambda_8)\right]}{w_1}$,

$u_2^* = \frac{I_f(\lambda_3-\lambda_4)+I_m(\lambda_{10}-\lambda_{11})}{w_2}$, and

$u_3^* = \frac{(\lambda_4-\lambda_5)D_f+(\lambda_7-\lambda_5)\left(A_f+\omega_f T_f\right)+(\lambda_{11}-\lambda_{12})D_m+(\lambda_{14}-\lambda_{12})(A_m+\omega_m T_m)}{w_3}$.

Hence, taking into account the control constraints $0 \le u_i \le 1$ where i $= 1, 2, 3$, the optimal control variables are expressed as:

$$u_1^* = \begin{cases} 0 & \text{if } \frac{(1-\kappa\epsilon)\left[\beta_f I_m S_f(\lambda_2-\lambda_1)+\beta_m I_f S_m(\lambda_9-\lambda_8)\right]}{w_1} < 0 \\ \frac{g_1}{w_1} & \text{if } 0 < \frac{(1-\kappa\epsilon)\left[\beta_f I_m S_f(\lambda_2-\lambda_1)+\beta_m I_f S_m(\lambda_9-\lambda_8)\right]}{w_1} < 1 \\ 1 & \text{if } 1 < \frac{(1-\kappa\epsilon)\left[\beta_f I_m S_f(\lambda_2-\lambda_1)+\beta_m I_f S_m(\lambda_9-\lambda_8)\right]}{w_1} \end{cases}$$

$$u_2^* = \begin{cases} 0 & \text{if } \frac{I_f(\lambda_3-\lambda_4)+I_m(\lambda_{10}-\lambda_{11})}{w_2} < 0 \\ \frac{g_2}{w_2} & \text{if } 0 < \frac{I_f(\lambda_3-\lambda_4)+I_m(\lambda_{10}-\lambda_{11})}{w_2} < 1 \\ 1 & \text{if } 1 < \frac{I_f(\lambda_3-\lambda_4)+I_m(\lambda_{10}-\lambda_{11})}{w_2} \end{cases}$$

$$u_3^* = \begin{cases} 0 & \text{if } \frac{(\lambda_4-\lambda_5)D_f+(\lambda_7-\lambda_5)\left(A_f+\omega_f T_f\right)+(\lambda_{11}-\lambda_{12})D_m+(\lambda_{14}-\lambda_{12})(A_m+\omega_m T_m)}{w_3} < 0 \\ \frac{g_3}{w_3} & \text{if } 0 < \frac{(\lambda_4-\lambda_5)D_f+(\lambda_7-\lambda_5)\left(A_f+\omega_f T_f\right)+(\lambda_{11}-\lambda_{12})D_m+(\lambda_{14}-\lambda_{12})(A_m+\omega_m T_m)}{w_3} < 1 \\ 1 & \text{if } 1 < \frac{(\lambda_4-\lambda_5)D_f+(\lambda_7-\lambda_5)\left(A_f+\omega_f T_f\right)+(\lambda_{11}-\lambda_{12})D_m+(\lambda_{14}-\lambda_{12})(A_m+\omega_m T_m)}{w_3} \end{cases}$$

where

$$g_1 = (1-\kappa\epsilon)\left[\beta_f I_m S_f\left(\lambda_2-\lambda_1\right)+\beta_m I_f S_m\left(\lambda_9-\lambda_8\right)\right],$$
$$g_2 = I_f\left(\lambda_3-\lambda_4\right)+I_m\left(\lambda_{10}-\lambda_{11}\right)$$
$$g_3 = \left(\lambda_4-\lambda_5\right)D_f+\left(\lambda_7-\lambda_5\right)\left(A_f+\omega_f T_f\right)+\left(\lambda_{11}-\lambda_{12}\right)D_m+\left(\lambda_{14}-\lambda_{12}\right)\left(A_m+\omega_m T_m\right)$$

Concisely, the optimal controls can be expressed in a compact form as:

$$u_1^* = \max\left\{\min\left\{1,k_1\right\},0\right\},$$
$$u_2^* = \max\left\{\min\left\{1,k_2\right\},0\right\},$$
$$u_3^* = \max\left\{\min\left\{1,k_3\right\},0\right\},$$

where.

$$k_1 = \frac{(1-\kappa\epsilon)\left[\beta_f I_m S_f(\lambda_2-\lambda_1)+\beta_m I_f S_m(\lambda_9-\lambda_8)\right]}{w_1}$$
$$k_2 = \frac{I_f(\lambda_3-\lambda_4)+I_m(\lambda_{10}-\lambda_{11})}{w_2}$$
$$k_3 = \frac{(\lambda_4-\lambda_5)D_f+(\lambda_7-\lambda_5)\left(A_f+\omega_f T_f\right)+(\lambda_{11}-\lambda_{12})D_m+(\lambda_{14}-\lambda_{12})(A_m+\omega_m T_m)}{w_3}$$

Therefore, we formulate the optimality system by combining the set of equations for the state variables and the set of equations for the adjoint variables with the initial conditions and final time conditions respectively

$$\frac{dS_f}{dt} = \Lambda-(1-u_1)(1-\kappa\epsilon)\beta_f I_m S_f-\eta S_f$$
$$\frac{dE_f}{dt} = (1-u_1)(1-\kappa\epsilon)\beta_f I_m S_f-(\alpha_f+\eta)E_f$$
$$\frac{dI_f}{dt} = \alpha_f E_f-(u_2+\mu+\eta+\gamma_f+\theta_f)I_f$$
$$\frac{dD_f}{dt} = (u_2+\gamma_f)I_f-(u_3+\mu+\eta+\rho_f)D_f$$
$$\frac{dT_f}{dt} = u_3 A_f+(u_3+\rho_f)D_f-(\mu+\eta+(1-u_3)\omega_f)T_f$$
$$\frac{dU_f}{dt} = \theta_f I_f-(\mu+\eta+\lambda_f)U_f$$
$$\frac{dA_f}{dt} = \lambda_f U_f+(1-u_3)\omega_f T_f-(u_3+\mu+\eta)A_f$$
$$\frac{dS_m}{dt} = \Lambda-(1-u_1)(1-\kappa\epsilon)\beta_m I_f S_m-\eta S_m$$
$$\frac{dE_m}{dt} = (1-u_1)(1-\kappa\epsilon)\beta_m I_f S_m-(\alpha_m+\eta)E_m$$
$$\frac{dI_m}{dt} = \alpha_m E_m-(u_2+\mu+\eta+\gamma_m+\theta_m)I_m$$
$$\frac{dD_m}{dt} = (u_2+\gamma_m)I_m-(u_3+\mu+\eta+\rho_m)D_m$$

$$
\begin{aligned}
\frac{dT_m}{dt} &= u_3 A_m + (u_3 + \rho_m) D_m - (\mu + \eta + (1 - u_3)\omega_m) T_m \\
\frac{dU_m}{dt} &= \theta_m I_m - (\mu + \eta + \lambda_m) U_m \\
\frac{dA_m}{dt} &= \lambda_m U_m + (1 - u_3)\omega_m T_m - (u_3 + \mu + \eta) A_m \\
\frac{d\lambda_1}{dt} &= (1 - u_1)(1 - \kappa\epsilon)\beta_f I_m (\lambda_1 - \lambda_2) + \eta\lambda_1 \\
\frac{d\lambda_2}{dt} &= -b_1 + \eta\lambda_2 + \alpha_f (\lambda_2 - \lambda_3) \\
\frac{d\lambda_3}{dt} &= \begin{array}{l} -b_2 + (\mu + \eta)\lambda_3 + (u_2 + \gamma_f)(\lambda_3 - \lambda_4) + \theta_f (\lambda_3 - \lambda_6) \\ + (1 - u_1)(1 - \kappa\epsilon)\beta_m s_m (\lambda_8 - \lambda_9) \end{array} \\
\frac{d\lambda_4}{dt} &= -b_3 + (\mu + \eta)\lambda_4 + (u_3 + \rho_f)(\lambda_4 - \lambda_5) \\
\frac{d\lambda_5}{dt} &= -b_4 + (\mu + \eta)\lambda_5 + \omega_f (\lambda_5 - \lambda_7) \\
\frac{d\lambda_6}{dt} &= -b_5 + (\mu + \eta)\lambda_6 + \lambda_f (\lambda_6 - \lambda_7) \\
\frac{d\lambda_7}{dt} &= -b_6 + (\mu + \eta)\lambda_7 \\
\frac{d\lambda_8}{dt} &= (1 - u_1)(1 - \kappa\epsilon)\beta_m I_f (\lambda_8 - \lambda_9) + \eta\lambda_8 \\
\frac{d\lambda_9}{dt} &= -b_7 + \eta\lambda_9 + \alpha_m (\lambda_9 - \lambda_{10}) \\
\frac{d\lambda_{10}}{dt} &= \begin{array}{l} -b_8 + (\mu + \eta)\lambda_{10} + (u_2 + \gamma_m)(\lambda_{10} - \lambda_{11}) + \theta_m (\lambda_{10} - \lambda_{13}) \\ + (1 - u_1)(1 - \kappa\epsilon)\beta_f S_f (\lambda_1 - \lambda_2) \end{array} \\
\frac{d\lambda_{11}}{dt} &= -b_9 + (\mu + \eta)\lambda_{11} + (u_3 + \rho_m)(\lambda_{11} - \lambda_{12}) \\
\frac{d\lambda_{12}}{dt} &= -b_{10} + (\mu + \eta)\lambda_{12} + \omega_m (\lambda_{12} - \lambda_{14}) \\
\frac{d\lambda_{13}}{dt} &= -b_{11} + (\mu + \eta)\lambda_{13} + \lambda_m (\lambda_{13} - \lambda_{14}) \\
\frac{d\lambda_{14}}{dt} &= -b_{12} + (\mu + \eta)\lambda_{14}
\end{aligned}
$$

With conditions $\lambda_i(t_f) = 0$ where $i = 1, 2, \ldots, 14$, $S_f(0), S_m(0) > 0$, and

$$E_f(0), E_m(0), I_f(0), I_m(0), D_f(0), D_m(0), T_f(0), T_m(0), U_f(0), U_m(0), A_f(0), A_m(0) \geq 0$$

8. Numerical Simulations

This section qualitatively examines the influence of optimum control measures on the transmission of HIV/AIDS within a community. This work utilizes the fourth-order Runge-Kutta technique which enables precise modelling of HIV transmission dynamics and the effects of various interventions across time. This method allows the evaluation of how various combinations of strategies preventative, screening, and treatment might collectively impact the different compartments including Infected and AIDS-stage individuals. Numerical simulations are performed to demonstrate the analytical results from the aforementioned study by using the initial conditions and parameter values provided in Table 1 and setting the terminal time $t_f = 20$. Additionally, the values of parameters in the objective functional are assumed and given in Table 3.

Table 3: Values of parameters in the objective functional

Cost Parameters	Values	Cost Parameters	Values
b_1	15	b_2	20
b_3	20	b_4	25
b_5	10	b_6	18
b_7	15	b_8	20
b_9	20	b_{10}	25
b_{11}	10	b_{12}	18
w_1	10	w_2	15
w_3	20		

We used MATLAB software to provide graphical representations of the state system with and without optimum control, as well as the adjoint systems within different scenarios. The optimal control profiles for each strategy and state system for both male and female are displayed together in all the graphs.

Figure 3 highlights the effect of preventative efforts in the absence of screening and treatment strategies in controlling HIV/AIDS epidemic, showing significant reductions in both infections and AIDS cases over time for both genders. It is shown in Figure 3(a) that the infections peak sharply around year 2 which is about six million for both males and females. Contrastingly, the infected populations reduced dramatically with prevention and staying below 200,000 in the period. On the other hand, as demonstrated in Figure 3(b) the AIDS compartments peak around year 5 and reach about 450 thousand with slightly fewer female cases, whereas with prevention strategy is much lower about 25,000 cases. The AIDS cases with prevention approach zero by the year 20 while without prevention it remains significant. Prevention not only reduces case the number of males and females but also shortens and flattens the epidemic curves for both infections and AIDS. The graphs indicate that females are somewhat less impacted in both the infected and AIDS stages;

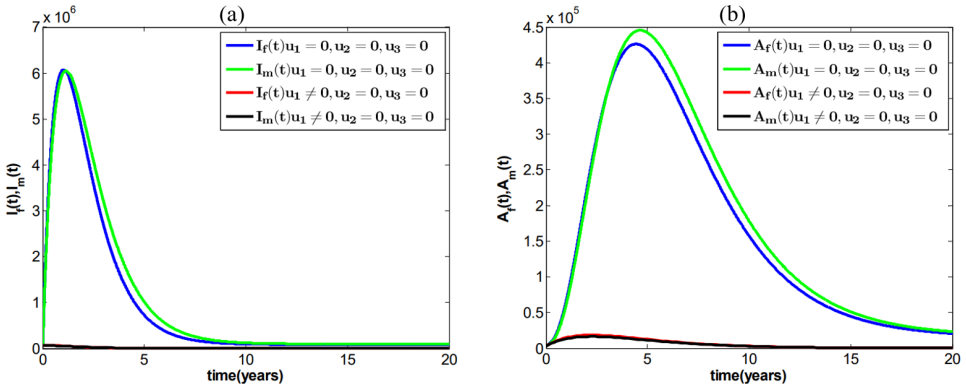

Figure 3: Simulation of optimal control of Infected and AIDS population for both male and female with prevention

however, preventative measures reduce this gender disparity.

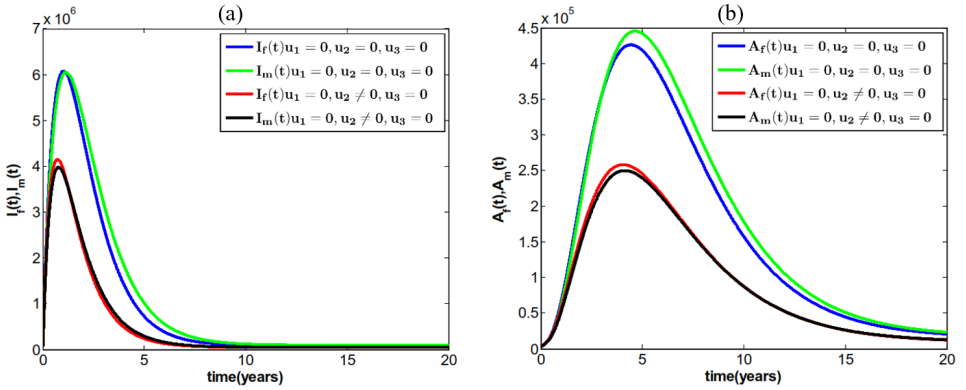

Figure 4: Simulation of optimal control of Infected and AIDS population for both male and female with screening

In Figure 4, the simulation of both infected and AIDS populations for males and females are depicted with and without screening control interventions and excluding prevention and treatment. The Figures 4(a) and 4(b) demonstrate the significant role of screening in decreasing the HIV/AIDS epidemic. While not as dramatically effective as prevention strategies, screening shows substantial benefits in reducing both infection rates and AIDS cases for males and females.

Figure 5 illustrates the dynamics of infected and AIDS population's progression over a 20-year period, comparing scenarios with and without treatment interventions in the absence of prevention and screening controls for both males and females. With treatment, there is minimal visible impact on the infection rates and the curves for treated and untreated scenarios are almost identical this is provided in Figure 5(a). It clearly can be

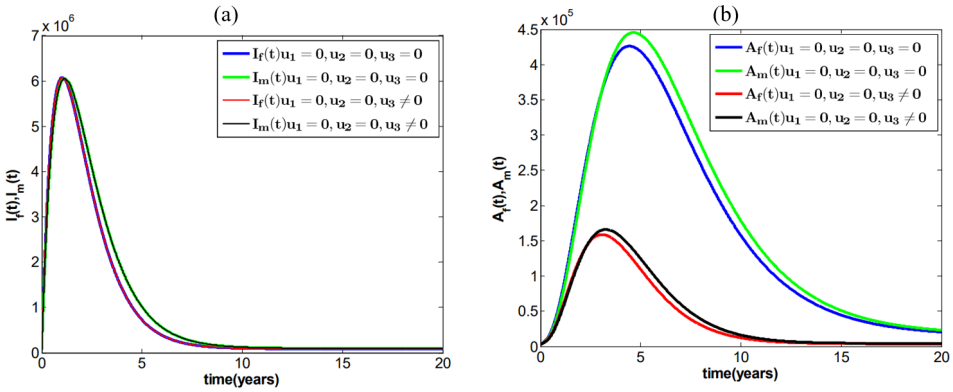

Figure 5: Simulation of optimal control of Infected and AIDS population for both male and female with treatment

seen in Figure 5(b) that the peak of AIDS population of both genders with treatment is substantially lowered to about 170,000 cases for males and 160,000 for females and approach zero by year 20. This underscores the importance of treatment in improving quality of life and survival for those who infected with HIV. The model suggests that comprehensive HIV/AIDS management should combine treatment with other strategies like prevention and screening to address both infection rates and disease progression.

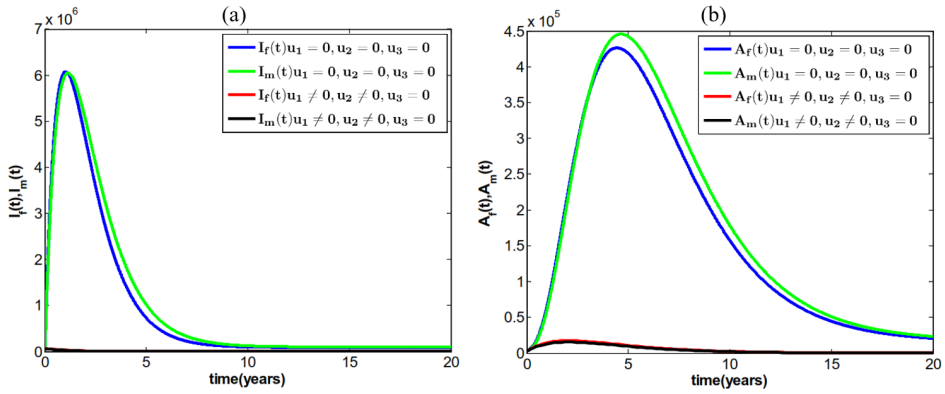

Figure 6: Simulation of optimal control of Infected and AIDS population for both male and female with prevention and screening

Figure 6 shows how the combination of preventive and screening strategies affects both infected and AIDS population's dynamics over the period. There is a remarkable drop in using both prevention and screening strategies together in infected and AIDS compartments for both male and female populations this can be seen in Figures 6(a) and 6(b). The intervention scenario considerably flattens the epidemic curves, eliminating new infections and bringing AIDS incidence to a minimum by year 20. The significant decrease in both compartments signifies that this integrated strategy address both new infections by prevention and the progression to AIDS through screening and probable early treatment

commencement.

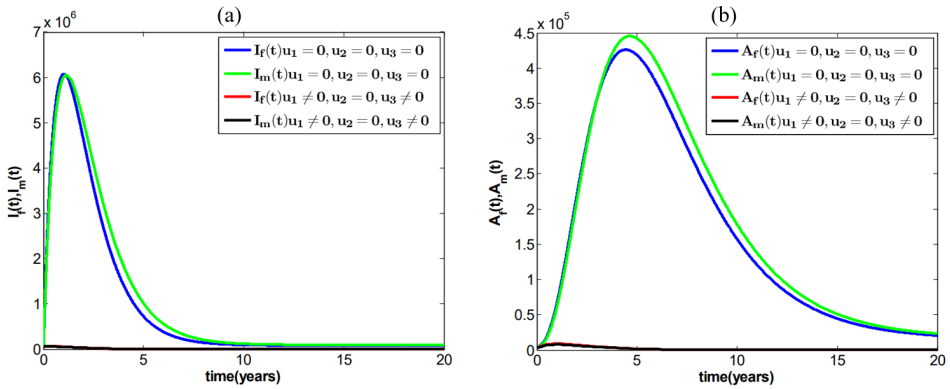

Figure 7: Simulation of optimal control of Infected and AIDS population for both male and female with prevention and treatment

Figure 7 depicts the infection and AIDS populations with and without prevention and treatment strategies and how the interventions affect the reduction of both compartments for males and females. Combining prevention and treatment controls significantly decreases infection rates and AIDS cases for both sexes, maintaining them at minimal levels over the time period. Integrating preventative and treatment techniques can successfully manage the transmission of infection and progression to AIDS over the period.

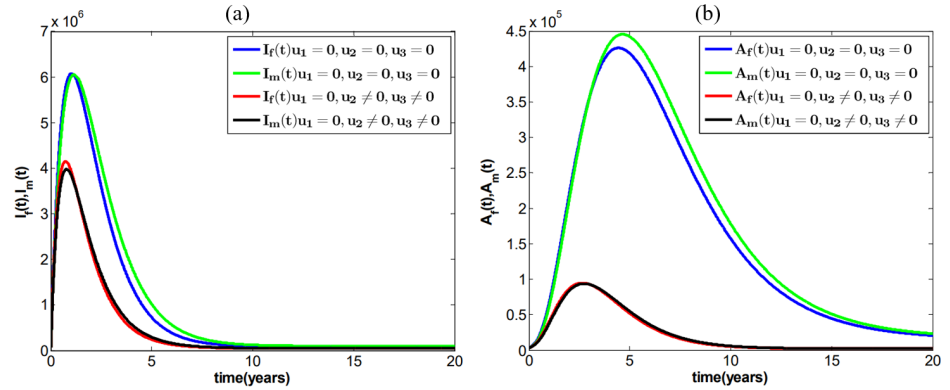

Figure 8: Simulation of optimal control of Infected and AIDS population for both male and female with screening and treatment

Figure 8 demonstrates the substantial influence of screening and treatment strategies on the management of both infection and AIDS populations for men and women over 20 years, even without preventive efforts. Through screening and treatment, as shown in Figure 8(a) the number of infections for both genders reach a lower high of around 4 million whereas AIDS cases for both sexes peak significantly lower, at around 100,000 which

can be seen in Figure 8(b). Screening and treatment interventions substantially decrease infective and AIDS populations for both sexes, but not as markedly as when prevention was incorporated in the prior scenarios.

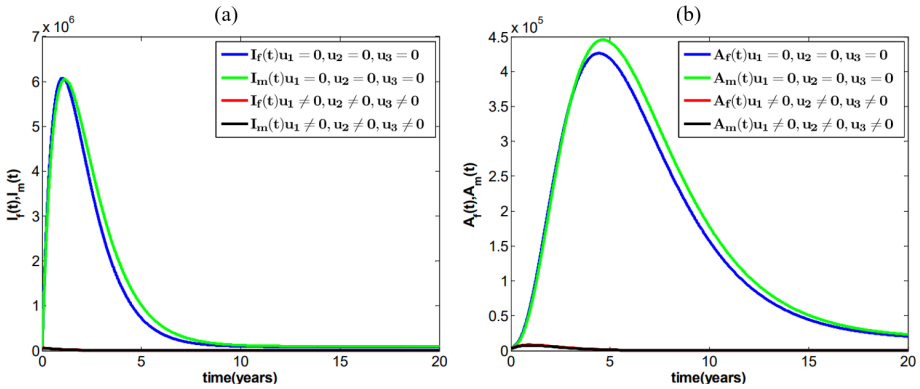

Figure 9: Simulation of optimal control of Infected and AIDS population for both male and female with prevention, screening and treatment

Figure 9 presents how the infected and AIDS populations change with and without combining prevention, screening and treatment strategies for both genders over time. In Figure 9(a), it is shown that the number of infected individuals is dramatically reduced with the interventions. This indicates that the combination of prevention, screening and treatment is highly effective in spreading the disease. On the other hand, the interventions have a considerable impact on minimizing the number of AIDS cases, similar to the infection graph which is provided in Figure 9(b). This signifies that the therapies are also effective in stopping the progression to AIDS. The treatments appear to exert an immediate and sustained impact, preventing the sharp increase in infections and progression to AIDS cases, even two decades later, a significant disparity persists between intervention and non-intervention situations

In Figure 10, the impact of different combinations of prevention, screening, and treatment interventions on infected and AIDS populations for both genders over 20 years period are illustrated. In Figure 10(a), the green line is the female infected population which peaks at around 6 million about year 2. In addition, there is some reduction of women infected individuals in the use of a single intervention, while the combination of two interventions presents a greater reduction and all three strategies together is the highly effective in minimizing infections which is drawn by black color. The graphs of female AIDS cases with various scenarios are provided in Figure 10(b). Women AIDS population peaks approximately at 4.3 million about year 5. Moreover, in the case of including a single and double interventions exhibit different level of reduction whereas all three strategies together yield the fewest female AIDS cases. Further, in Figure 10(c) it is shown that the male infected population has analogous patterns to the infected female population with all different

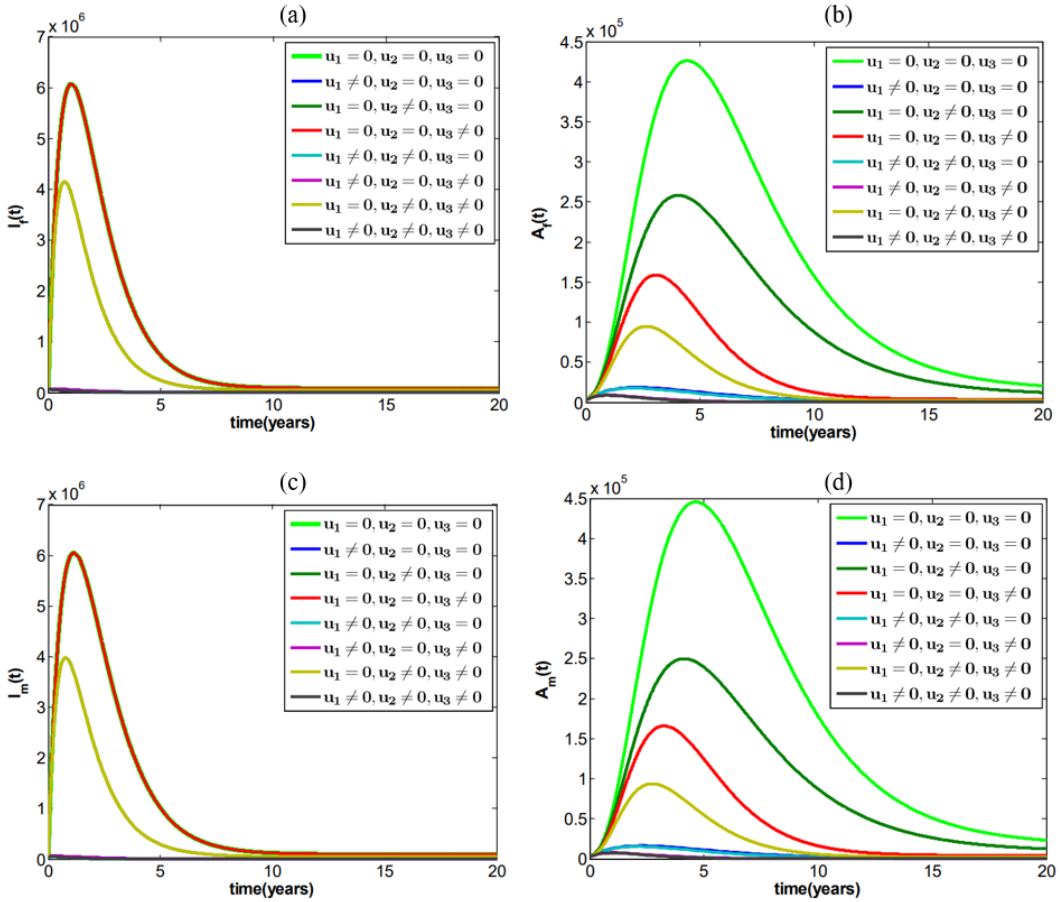

Figure 10: Simulation of optimal control of Infected and AIDS population for both males and females with all scenarios

combinations of the interventions, although male infections appear to peak slightly higher than female infections in the absence of interventions. Additionally, the illustration of male AIDS cases with different combinations of interventions are given in Figure 10(d) which closely have a similar pattern as female AIDS cases. However, male AIDS individuals peak at around 4.5 million at year 5 without interventions which is higher than the peak of female AIDS cases.

The results show that the simultaneous use of all three interventions (prevention, screening, and treatment) is the most successful method for lowering infections and AIDS cases in both genders. Furthermore, the patterns for males and females are quite comparable, suggesting that interventions are equally successful for both genders. Although all interventions are beneficial, treatment alone appears to be less successful than preventive or screening alone, particularly in decreasing initial infection rates. To sum up, the presented model strongly supports the implementation of comprehensive HIV/AIDS control measures that include prevention, screening, and treatment. It illustrates that although

single interventions are
advantageous, a comprehensive strategy is significantly more successful in managing the pandemic for both genders over time.

9. Limitations and Future Directions

While this study provides valuable insights into gender-specific HIV transmission dynamics, several limitations should be considered. The deterministic modeling approach cannot account for random transmission events that occur in real populations. Although gender stratification offers important granularity, additional factors like age differences and risk-group variations would further improve the model's accuracy. Our analysis also assumes perfect adherence to control measures, while actual implementation faces behavioral challenges like risk compensation. Nevertheless, despite these simplifications, the current model establishes a useful foundation for assessing gender-targeted HIV interventions and can be progressively refined to better reflect real-world complexity.

This framework can be extended by incorporating age-structured transmission, stochastic dynamics, and behavioral feedback mechanisms to more accurately represent real-world HIV transmission. Future research should include cost-effectiveness analyses to inform optimal allocation of intervention resources and validate the model using country-specific data. Incorporating machine learning techniques may enhance predictive accuracy, while network-based extensions could improve targeting of high-risk populations. Furthermore, the model is adaptable to other infectious diseases, providing policymakers with a flexible tool for evaluating integrated prevention strategies under resource constraints

10. Conclusion

This paper presents a framework for understanding HIV/AIDS transmission dynamics between male and female populations. We analyzed the model's equilibrium points and determined the basic reproduction number, R_0. The stability analysis showed that the disease-free equilibrium is stable when $R_0 < 1$, while the endemic equilibrium is stable when $R_0 > 1$.

To identify the most influential factors, we conducted local sensitivity analysis using fullnormalization, half-normalization, and non-normalization methods. We also examined how key parameters affect R_0.

Optimal control strategies prevention, screening, and treatment were then introduced. The control problem was formulated and solved numerically, revealing the most efficient use of resources and timing for interventions. Results showed that combining all three strategies significantly reduces the number of infections and AIDS cases in both sexes. Numerical simulations supported the effectiveness of these interventions in reducing HIV/AIDS prevalence over time.

These findings highlight the significance of focused, evidence-driven strategies in public health policy. This study not only improves our understanding of HIV/AIDS dynamics in

different populations but also provides the path for effective intervention strategies that can have a dramatic impact on public health outcomes.

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