Int. J. Anal. Appl. (2024), 22:234

International Journal of Analysis and Applications

Mathematical Modeling and Optimal Control of an HIV/AIDS Transmission Model

Ouedraogo Boukary[1,*], **Zorom Malicki**[2], **Gouba Elisée**[1]

[1]*Université Joseph KI ZERBO, Laboratoire d'Analyse Numérique, d'Informatique et de Biomathématique (LANIBIO), 03 BP 7021 Ouagadougou 03, Burkina Faso*

[2]*Laboratoire Eaux Hydro-Systèmes et Agriculture (LEHSA), Institut International d'Ingénierie de l'Eau et de l'Environnement (2iE), BP 594 Ouagadougou 01, Rue de la Science, Burkina Faso*

[*]*Corresponding author:* aboubakrrambo1930@gmail.com

Abstract. In this article, we present a mathematical model for the transmission of HIV with the compartment of individuals in remission and vertical transmission describing the dynamics of the spread of the HIV/AIDS epidemic in a community. In the mathematical analysis of the model, we compute the basic reproduction number $\mathcal{R}_0$ and study the existence and stability of the disease-free equilibrium point. We also formulate an appropriate optimal control problem and study the conditions necessary for disease control to determine the role of preventive measures and treatment in reducing the spread of HIV/AIDS. Indeed, we study the impact of these control variables taken separately and combined. So we find that treatment is more cost-effective in reducing the spread of HIV than preventive measures. Finally, the numerical results conform to the theoretical analysis.

1. Introduction

AIDS (Acquired Immune Deficiency Syndrome) first appeared in the 1980s in the United States [34]. In 1983, HIV (Human Immunodeficiency Virus), the retrovirus that infects humans and causes AIDS was identified at the Pastor Institute of Paris [34]. This retrovirus weakens the immune system, making it vulnerable to multiple opportunistic infections. AIDS is now considered a pandemic that took away the lives of an estimated **40.4 million** [32.9 million - 51.3 million] people between 1981 and 2022 [1]. For the biology and epidemiology of HIV/AIDS, we refer the reader to [35], [34], [36] and references cited therein.

Numerous mathematical models have proven their usefulness in describing and understanding the dynamics of HIV infection: see, for example, [2], [3], [4], [5], [6], [7], [8], [9], [10], [11], and the

Received: Oct. 09, 2024.

2020 *Mathematics Subject Classification.* 34C60, 92D30, 34D23, 49K15.

Key words and phrases. HIV/AIDS epidemiology; mathematical model; local and global stability; numerical results; Optimal control.

https://doi.org/10.28924/2291-8639-22-2024-234
ISSN: 2291-8639

references cited therein. In [21], the authors presented the impact of optimal control on HIV/AIDS treatment and screening of unaware infected individuals on the dynamics of disease transmission. Their study focuses on a homogeneous population with constant immigration of susceptible individuals, incorporating the use of condoms, screening of unaware infected individuals, and treatment of those infected. The authors of [13] examined the mathematical modeling of HIV/AIDS transmission dynamics with drug resistance compartments. They studied the role of passive immunity and pharmacotherapy in reducing viral replication and disease transmission.
Karrakchou et al. [14] studied the fundamental role of chemotherapy treatment in controlling viral reproduction in an HIV patient. Additionally, Adams et al. [15] derived therapeutic strategies for HIV by formulating and analyzing an optimal control problem using two types of dynamic treatments representing reverse transcriptase (RT) inhibitors and protease inhibitors (PIs). Finally, Gul et al. [16] examined the stability of a SIR epidemic model and optimal vaccination. For optimal control applied to other epidemic models, we refer the reader to [17], [18], [19], [20], as well as the references cited therein. The authors of [21] also studied the impact of combined strategies in controlling HIV/AIDS and found that the most cost-effective approach combines all control strategies. In [22], the authors demonstrated that PrEP significantly reduces HIV transmission.
We based our work on the paper by [22], whose model also considers antiretroviral treatment for HIV-infected individuals with or without AIDS symptoms, as well as on the article by [23], whose authors included vertical transmission in their model, to which we added the compartment for individuals in remission. The addition of the remission compartment also takes into account current developments in the fight against HIV/AIDS. Indeed, according to the UNAIDS 95-95-95 strategy [1], [24], individuals who normally adhere to antiretroviral treatment until their viral load suppression do not transmit HIV to their sexual partners. The aim is to show that a mathematical model including vertical transmission and individuals in remission accurately reflects reality, both from a biological perspective and in terms of progress in the fight against HIV/AIDS. This could reduce disease spread if treatment is closely monitored and vertical transmission is controlled. The current availability of antiretroviral medications, combined with the possibility of preventing HIV infection through preventive measures, motivates our choice of two control variables: preventive measures (such as condom use, screening, awareness, abstinence, sexual partner fidelity, etc.) and antiretroviral treatment.
The paper is organized as follows: The model is formulated in Section 2. In Section 3, we address the mathematical analysis of the model. Section 4 presents a numerical simulation to demonstrate the consistency between the theoretical results of the analysis and the numerical results. Section 5 presents the study of optimal control analysis and its numerical solutions. Finally, the conclusion is given in Section 6.

2. Model formulation

In this section, we propose a compartmental SIAHR model to describe the transmission dynamics of HIV/AIDS. This model includes the individual remission compartment due to the technical

advancements in the fight against HIV. Vertical transmission, a significant transmission mode, especially in developing countries, is also considered in our model. *Lambda* denotes recruitment into the population. The introduction of vertical transmission reduces the recruitment in the population by an amount ΛpI because newborns from this transmission are already infected and this appears in the compartment of infected individuals without clinical signs of the disease. The model divides the human population into five mutually exclusive compartments: the susceptible compartment S, the compartment of infected individuals without clinical signs I, the compartment of individuals with AIDS A, the compartment of individuals under antiretroviral treatment H, and the compartment of individuals with suppressed viral load, i.e., the remission compartment R.
The movement between the different compartments occurs as follows: in S, a susceptible individual becomes infected after adequate contact with an infected individual. An individual in the I class can undergo antiretroviral treatment and move to the H class, or they may ignore the treatment and naturally progress to the A compartment. In the A class, an individual undergoing treatment can also move to the H class. Individuals in the H class can enter the R compartment if they adhere properly to the treatment or shift to the A compartment if they neglect it. The variables and parameters of the model are summarized in the following tables:

Table 1: Variables used in the model

Variables	Descriptions
S	Individuals susceptible to disease
I	HIV-infected individuals without clinical signs
A	HIV-infected individuals with clinical signs
H	HIV-infected individuals on treatment
R	Individuals in remission, i.e those who are on treatment and whose viral load is suppressed
N	Total population $N = S + I + A + H + R$

Table 2: Parameters used in the model

Parameters	Meaning
Λ	Recruitment of the population
η	Infectivity of AIDS patients compared to infected individuals without clinical symptoms of the disease
α	HIV/AIDS transmission rate
β_1	Rate of transfer from the compartment of infected individuals without clinical signs to the AIDS compartment
β_2	rate of transfer from the AIDS class to the compartment of individuals on treatment
λ	rate of transfer of individuals on treatment to the AIDS class
p	probability of HIV-positive newborns through vertical transmission
ϵ	probability of infected individuals without clinical signs progressing to the AIDS phase
ρ	Probability of individuals who have failed their treatment and move up to the AIDS class
μ_0	Natural mortality rate
μ_1	HIV-induced mortality rate

Susceptible individuals contract HIV infection following effective contact with infected persons, at a rate K defined by:

$$K = \frac{\alpha(I + \eta A)}{N}$$

where α is the effective contact rate for HIV transmission, and η is the modification parameter $\eta \geq 1$ that accounts for the relative infectivity of individuals with AIDS symptoms compared to those infected without symptoms of the disease.

We make the following assumptions:

(H_1): The total recruitment into the population is $\Lambda - \Lambda pI$;

(H_2): Vertical transmission of HIV can occur when the mother is infected and not undergoing treatment, with a proportion ΛpI;

(H_3): The probabilities p, ϵ, and ρ; the parameters Λ, α, β_1, β_2, λ, ν, μ_0, and μ_1 are all non-negative;

(H_4): Individuals infected with HIV only die from the disease when it progresses to the AIDS phase, at a rate of $(\mu_0 + \mu_1)A$;

(H_5): We base our model on the achievement of the 95-95-95 targets for testing, treatment, and viral load suppression, which the United Nations committed in June 2021 to reaching by 2030. [24];

(H_6): People living with HIV who undergo antiretroviral treatment until viral load suppression do not transmit the virus to their sexual partners and are categorized in the remission compartment;

(H_7): We assume that at least 95% of infected individuals are aware of their serological status;

(H_8): Those who do not properly follow their treatment transition to the AIDS phase, as the appearance of symptoms is an indicative sign;

(H_9): All newborns infected with HIV through vertical transmission are in the compartment of HIV-infected individuals without clinical signs;

(H_{10}): We assume that $\Lambda p < \beta_1 + \mu_0$, the rate of vertical transmission is less than the sum of the rate of progression from the phase of infection without clinical signs to the AIDS phase and the natural mortality rate. Thus, we have the following diagram:

The system of differential equations describing the HIV model with vertical transmission, while accounting for the remission compartment, is presented as follows:

$$\begin{cases} \dfrac{dS(t)}{dt} = \Lambda - \Lambda pI(t) - (K + \mu_0)S(t); \\ \dfrac{dI(t)}{dt} = KS(t) + \Lambda pI(t) - \epsilon\beta_1 I(t) - (1-\epsilon)\beta_1 I(t) - \mu_0 I(t); \\ \dfrac{dA(t)}{dt} = \epsilon\beta_1 I(t) - \beta_2 A(t) - (\mu_0 + \mu_1)A(t) + \rho\lambda H(t); \\ \dfrac{dH(t)}{dt} = (1-\epsilon)\beta_1 I(t) + \beta_2 A(t) - \rho\lambda H(t) - (1-\rho)\lambda H(t) - \mu_0 H(t); \\ \dfrac{dR(t)}{dt} = (1-\rho)\lambda H(t) - \mu_0 R(t) \end{cases} \tag{2.1}$$

With the following initial conditions:

$S(0) \geq 0, \quad I(0) \geq 0, \quad A(0) \geq 0, \quad H(0) \geq 0 \quad et \quad R(0) \geq 0.$

After reducing the model (2.1) and setting $k_1 = -\Lambda p + \beta_1 + \mu_0$, $k_2 = \beta_2 + \mu_0 + \mu_1$ and $k_3 = \lambda + \mu_0$,

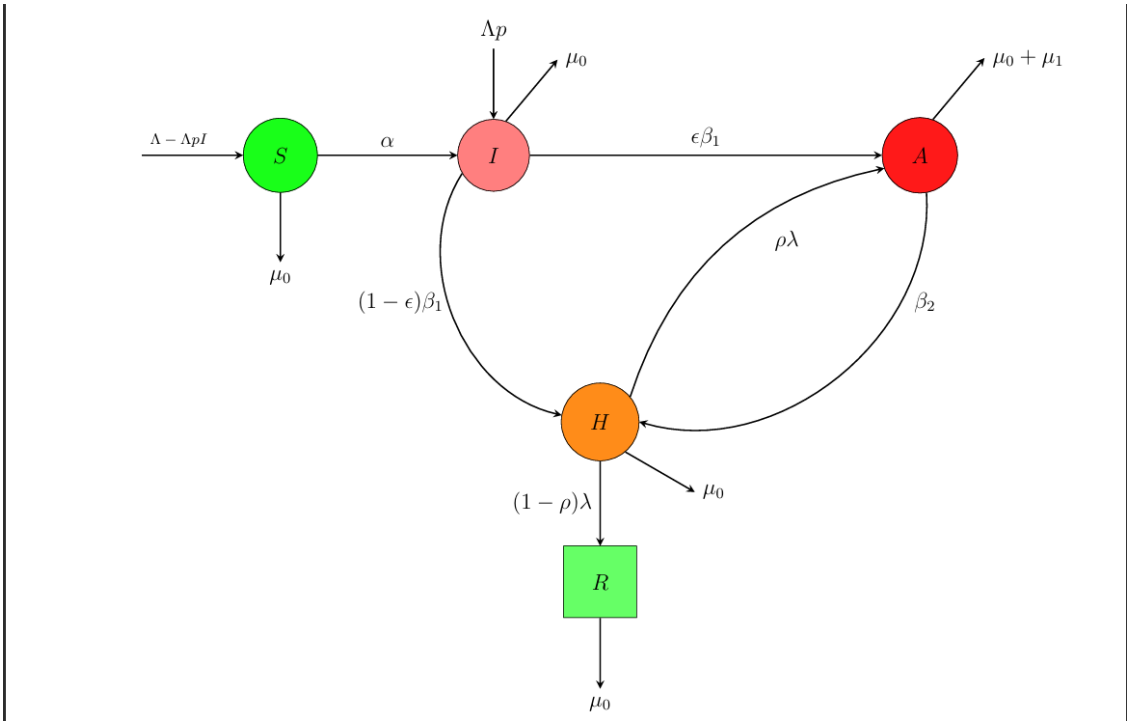

FIGURE 1. Diagram accounting for vertical and horizontal transmission of HIV

we obtain:

$$\begin{cases} \dfrac{dS(t)}{dt} = \Lambda - \Lambda pI(t) - (K(t) + \mu_0)S(t); \\ \dfrac{dI(t)}{dt} = K(t)S(t) - k_1 I(t); \\ \dfrac{dA(t)}{dt} = \epsilon\beta_1 I(t) - k_2 A(t) + \rho\lambda H(t); \\ \dfrac{dH(t)}{dt} = (1-\epsilon)\beta_1 I(t) + \beta_2 A(t) - k_3 H(t); \\ \dfrac{dR(t)}{dt} = (1-\rho)\lambda H(t) - \mu_0 R(t) \end{cases} \tag{2.2}$$

With the following initial conditions:

$S(0) \geq 0, \quad I(0) \geq 0, \quad A(0) \geq 0, \quad H(0) \geq 0 \quad et \quad R(0) \geq 0.$

3. MATHEMATICAL ANALYSIS OF THE MODEL

In this section, we show that the model is well-posed. We calculate the disease-free equilibrium point and the basic reproduction number $\mathcal{R}_0$. Finally, we study the stability of the disease-free equilibrium point of the model 2.2.

3.1. **Invariant region.**

Lemma 3.1. *the biologically feasible region of the HIV/AIDS model 2.2 given by:*

$$\Omega = \left\{ (S(t), I(t), A(t)H(t), R(t)) \in \mathbb{R}_+^5 : S + I + A + H + R \leq \frac{\Lambda}{\mu_0} \right\},$$

is positively invariant and attractive.

Proof: The rate of change of the total population is: $\frac{dN(t)}{dt} = \Lambda - \mu_0 N(t) - \mu_1 A(t)$
Thus:

$$\frac{dN(t)}{dt} + \mu_0 N(t) \leq \Lambda \tag{3.1}$$

because $\mu_1 > 0$ and $A(t) \geq 0$.
Let $\frac{dN(t)}{dt} + \mu_0 N(t) = 0$ the homogeneous equation associated with the differential inequality 3.1
The solution to our homogeneous equation is:$N(t) = ke^{-\mu_0 t}$, $k \in \mathbb{R}$. And for $t = 0$, we have $N(0) = k$
Using the method of variation of constants, we have:
$k'(t)e^{-\mu_0 t} - \mu_0 k(t)e^{-\mu_0 t} + \mu_0 k(t)e^{-\mu_0 t} = \Lambda \implies k'(t)e^{-\mu_0 t} = \Lambda$
$k'(t) = \Lambda e^{\mu_0 t}$, and after integration, we have: $k(t) = \frac{\Lambda}{\mu_0} e^{\mu_0 t} + C$ where C is an integration constant determined by the initial conditions.
Since $N(0) = k(0) = \frac{\Lambda}{\mu_0}$ then $C = N(0) - \frac{\Lambda}{\mu_0}$.
And finally $N(t) \leq \left(\frac{\Lambda}{\mu_0} e^{\mu_0 t} + N(0) - \frac{\Lambda}{\mu_0}\right) e^{-\mu_0 t}$
that is to say $N(t) \leq N(0)e^{-\mu_0 t} + \frac{\Lambda}{\mu_0}\left(1 - e^{-\mu_0 t}\right)$
And by letting t approach $+\infty$, we have: $N(t) \leq \frac{\Lambda}{\mu_0} < \infty$.
Consequently, Ω is positively invariant as long as $N(t) \leq \frac{\Lambda}{\mu_0}$.

Theorem 3.1. *the solutions $S(t)$, $I(t)$, $A(t)$, $H(t)$ and $R(t)$ of the model* (2.2) *of HIV/AIDS, with non-negative initial conditions in Ω remain non-negative in Ω for all $t \geq 0$.*

Before presenting the proof of the theorem 3.1, we will state a technical result that we will use subsequently.

Lemma 3.2. *Let $x(t)$, $a(t)$, and $y(t)$ be three functions of time, if*

$$\frac{dx(t)}{dt} - a(t)x(t) = y(t) \tag{3.2}$$

with $y(t) \geq 0$, $\forall t > 0$ and $x(0) \geq 0$ then any solution $x(t)$ of (3.2) *is positive for all $t > 0$;*

Proof: To demonstrate the positivity of the solutions of (3.2), let us multiply it by:$e^{-(\int_0^t a(s)ds)}$ We then have:

$$e^{-(\int_0^t a(s)ds)}\left[\frac{dx(t)}{dt} - a(t)x(t)\right] = e^{-(\int_0^t a(s)ds)} y(t) \tag{3.3}$$

That is to say $\frac{dx(t)}{dt} e^{-(\int_0^t a(s)ds)} - a(t)e^{-(\int_0^t a(s)ds)} x(t) = e^{-(\int_0^t a(s)ds)} y(t)$

Next, we have: $\frac{dx(t)}{dt}e^{-(\int_0^t a(s)ds)} + \frac{d}{dt}\Big(-\int_0^t a(s)ds\Big)e^{-\int_0^t a(s)ds}x(t) = e^{-\int_0^t a(s)ds}y(t)$
Furthermore, we have: $\frac{dx(t)}{dt}e^{-(\int_0^t a(s)ds)} + \frac{d}{dt}\Big(e^{-\int_0^t a(s)ds}\Big)x(t) = e^{-\int_0^t a(s)ds}y(t)$
Thus, we obtain the following:

$$\frac{d}{dt}\Big[e^{-\int_0^t a(s)ds}x(t)\Big] = e^{-\int_0^t a(s)ds}y(t) \tag{3.4}$$

Integrating equation (3.4) from 0 to t, we have:
$\int_0^t \frac{d}{dt}\Big[e^{-\int_0^t a(s)ds}x(t)\Big] = \int_0^t e^{-\int_0^t a(u)du}y(s)ds$
i.e.

$$e^{-\int_0^t a(s)ds}x(t) - x(0) = \int_0^t e^{-\int_0^t a(u)du}y(s)ds \tag{3.5}$$

By multiplying (3.5) by: $e^{\int_0^t a(s)ds}$, it follows that:

$$x(t) = e^{\int_0^t a(s)ds}x(0) + \int_0^t e^{-\int_s^t a(u)du}y(s)ds \tag{3.6}$$

Let $A(t) = \int_0^t a(s)ds$
then equation (3.6) becomes $x(t) = e^{A(t)}x(0) + \int_0^t e^{A(t-s)}y(s)ds$
Or alternatively $x(t) = e^{A(t)}\Big[x(0) + \int_0^t e^{-A(s)}y(s)ds\Big]$

We have thus constructed a positive solution for equation (3.2).
Proof: From the first equation of (2.2), we have:
$\frac{dS(t)}{dt} + (K(t) + \mu_0)S(t) = \Lambda - \Lambda pI(t)$ and according to lemma 3.2, we obtain:
$S(t) = e^{\int_0^t (K(s)+\mu_0)ds}\Big[S(0) + \int_0^t e^{-\int_0^t (K(s)+\mu_0)ds}(\Lambda - \Lambda pI(s))ds\Big] \geq 0.$
Similarly, we can demonstrate that the other state variables of the model (2.2) are positive for all $t \geq 0$, that is $I(t) \geq 0$, $A(t) \geq 0$, $H(t) \geq 0$ and $R(t) \geq 0$. This concludes the proof.
Based on the above results, it is sufficient to examine the dynamics of HIV/AIDS transmission represented by the model (2.2) in the biologically feasible region Ω, where the model is considered mathematically and epidemiologically well-posed.

3.2. **Disease-free equilibrium point.** Let E_0 be the disease-free equilibrium point commonly referred to as *DFE*. The following theorem provides the existence and uniqueness of this equilibrium point.

Theorem 3.2. *the system* (2.2) *has a disease-free equilibrium point given by:*

$$E_0 = \Big(\frac{\Lambda}{\mu_0}, 0, 0, 0, 0\Big) \tag{3.7}$$

Proof: In the absence of HIV, the infectious compartments (I, A, and H) are empty. This also means that no individuals will be in remission, i.e., R is also empty. We can then assume, taking

into account the initial conditions, that

$$I = A = H = R = 0 \tag{3.8}$$

By setting all the equations of the system (2.2) equal to zero, we obtain: $S = \dfrac{\Lambda}{\mu_0}$.

Hence, the disease-free equilibrium point is: $E_0 = \left(\dfrac{\Lambda}{\mu_0}, 0, 0, 0, 0\right)$.

3.3. **Basic reproduction number $\mathcal{R}_0$.** The basic reproduction number $\mathcal{R}_0$ is defined as the average number of secondary cases generated by an infectious individual introduced into a completely susceptible population during their infectious period. If $\mathcal{R}_0 < 1$, then HIV will die out in the population, whereas if $\mathcal{R}_0 > 1$, HIV will spread in the population and become endemic. $\mathcal{R}_0$ thus plays a crucial role in the management and control of an epidemic. To calculate $\mathcal{R}_0$, we use the method of Van den Driessche and Watmough [25] through the Next Generation Matrix algorithm they described. We know that the compartments containing the infected/infectious individuals are I, A, and H, while those not containing them are S and R. We can rewrite the system (2.2) in the form:

$$\frac{dx}{dt} = \mathcal{F}(x) - \mathcal{V}(x) \tag{3.9}$$

where $\mathcal{F}$ is the matrix of new infections and $\mathcal{V}$ is the transition matrix between the compartments of the system. Considering the system (2.2), it follows that:

$$\mathcal{F} = \begin{pmatrix} \dfrac{\alpha(I+\eta A)S}{N} \\ 0 \\ 0 \end{pmatrix} \quad \text{and} \quad \mathcal{V} = \begin{pmatrix} k_1 I \\ -\epsilon\beta_1 I + k_2 A - \rho\lambda H \\ -(1-\epsilon)\beta_1 I - \beta_2 A + k_3 H \end{pmatrix}$$

The Jacobian matrices of these two matrices at the disease-free equilibrium point E_0 are respectively:

$$F = \begin{pmatrix} \alpha & \alpha\eta & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad \textit{and} \quad V = \begin{pmatrix} k_1 & 0 & 0 \\ -\epsilon\beta_1 & k_2 & -\rho\lambda \\ -(1-\epsilon)\beta_1 & -\beta_2 & k_3 \end{pmatrix} \tag{3.10}$$

The matrix V is invertible because its determinant $detV = k_1(k_2k_3 - \beta_2\rho\lambda)$ is non-zero.

$$V^{-1} = \begin{pmatrix} \frac{1}{k_1} & 0 & 0 \\ \frac{\epsilon\beta_1 k_3 + \rho\lambda(1-\epsilon)\beta_1}{k_1(k_2k_3 - \beta_2\rho\lambda)} & \frac{k_3}{k_2k_3 - \beta_2\rho\lambda} & \frac{\rho\lambda}{k_2k_3 - \beta_2\rho\lambda} \\ \frac{\epsilon\beta_1\beta_2 + (1-\epsilon)\beta_1 k_2}{k_1(k_2k_3 - \beta_2\rho\lambda)} & \frac{-\beta_2}{k_2k_3 - \beta_2\rho\lambda} & \frac{k_2}{k_2k_3 - \beta_2\rho\lambda} \end{pmatrix} \tag{3.11}$$

The next-generation matrix FV^{-1} is given by

$$FV^{-1} = \begin{pmatrix} \frac{\alpha}{k_1} + \frac{\alpha\eta(\epsilon\beta_1 k_3 + \rho\lambda(1-\epsilon)\beta_1)}{k_1(k_2k_3 - \beta_2\rho\lambda)} & \frac{\alpha\eta k_3}{k_2k_3 - \beta_2\rho\lambda} & \frac{\alpha\eta\rho\lambda}{k_2k_3 - \beta_2\rho\lambda} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

The basic reproduction number $\mathcal{R}_0$ is given by the spectral radius of the next-generation matrix $\rho(FV^{-1})$.

$$\mathcal{R}_0 = \frac{\alpha}{k_1} + \frac{\alpha(\eta\epsilon\beta_1 k_3 + \eta\rho\lambda(1-\epsilon)\beta_1)}{k_1(k_2k_3 - \beta_2\rho\lambda)}$$

That is to say:

$$\mathcal{R}_0 = \frac{\alpha[k_2k_3 - \beta_2\rho\lambda + \eta\epsilon\beta_1 k_3 + \eta\rho\lambda(1-\epsilon)\beta_1]}{k_1(k_2k_3 - \beta_2\rho\lambda)} \tag{3.12}$$

3.4. **Local stability of the disease-free equilibrium point.** To conclude the stability of the disease-free equilibrium point, it is necessary to establish a relationship between the model parameters in (2.2), which leads to determining a threshold under the condition that the eigenvalues of the Jacobian matrix have negative real parts. For this, we use the characteristic polynomial and the Routh-Hurwitz stability conditions. The stability of the disease-free equilibrium is governed by the basic reproduction number $\mathcal{R}_0$.

Theorem 3.3. *The disease-free equilibrium point E_0 is locally and asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

Proof: The Jacobian matrix J for the model (2.2) is expressed as:

$$J = \begin{pmatrix} -\frac{\alpha(I(t)+\eta A(t))(N-S)}{N^2} - \mu_0 & -\Lambda P - \frac{\alpha S(N-(I(t)+\eta A(t)))}{N^2} & -\frac{\alpha\eta S(N-(I(t)+\eta A(t)))}{N^2} & 0 & 0 \\ \frac{\alpha(I(t)+\eta A(t))(N-S)}{N^2} & \frac{\alpha S(N-(I(t)+\eta A(t)))}{N^2} - k_1 & \frac{\alpha\eta S(N-(I(t)+\eta A(t)))}{N^2} & 0 & 0 \\ 0 & \epsilon\beta_1 & -k_2 & \rho\lambda & 0 \\ 0 & (1-\epsilon)\beta_1 & \beta_2 & -k_3 & 0 \\ 0 & 0 & 0 & (1-\rho)\lambda & -\mu_0 \end{pmatrix}$$

The Jacobian matrix $J(E_0)$ of the model (2.2) at the disease-free equilibrium is given by:

$$J(E_0) = \begin{pmatrix} -\mu_0 & -\Lambda p - \alpha & -\alpha\eta & 0 & 0 \\ 0 & \alpha - k_1 & \alpha\eta & 0 & 0 \\ 0 & \epsilon\beta_1 & -k_2 & \rho\lambda & 0 \\ 0 & (1-\epsilon)\beta_1 & \beta_2 & -k_3 & 0 \\ 0 & 0 & 0 & (1-\rho)\lambda & -\mu_0 \end{pmatrix}$$

Let $P(\Psi) = \det(J(E_0) - \Psi I_5)$ be the characteristic polynomial associated with the Jacobian matrix $J(E_0)$, where Ψ is the set of eigenvalues of $J(E_0)$ and I_5 is the identity matrix.

$$P(\Psi) = \begin{vmatrix} -\mu_0 - \Psi & -\Lambda p - \alpha & -\alpha\eta & 0 & 0 \\ 0 & \alpha - k_1 - \Psi & \alpha\eta & 0 & 0 \\ 0 & \epsilon\beta_1 & -k_2 - \Psi & \rho\lambda & 0 \\ 0 & (1-\epsilon)\beta_1 & \beta_2 & -k_3 - \Psi & 0 \\ 0 & 0 & 0 & (1-\rho)\lambda & -\mu_0 - \Psi \end{vmatrix}$$

Two eigenvalues, $\Psi_1 = \Psi_2 = -\mu_0$, are negative. For the remaining eigenvalues, we consider the following square matrix of order 3, $Q(\Psi)$:

$$Q(\Psi) = \begin{vmatrix} \alpha - k_1 - \Psi & \alpha\eta & 0 \\ \epsilon\beta_1 & -k_2 - \Psi & \rho\lambda \\ (1-\epsilon)\beta_1 & \beta_2 & -k_3 - \Psi \end{vmatrix}$$

After some algebraic manipulations, we have:

$$Q(\Psi) = \Psi^3 + \Psi^2\Big(k_1 + k_2 + k_3 - \alpha\Big) + \Psi\Big(k_1k_2 + k_1k_3 + k_2k_3 - \alpha(k_2 + k_3) - \beta_2\rho\lambda - \alpha\eta\Big) + \Big(k_1k_2k_3 + \alpha\beta_2\rho\lambda - k_1\beta_2\rho\lambda - \alpha k_2k_3 - \alpha\eta\epsilon\beta_1k_3 - \alpha\eta\rho\lambda(1-\epsilon)\beta_1\Big).$$

By setting $a_1 = k_1 + k_2 + k_3 - \alpha$;

$a_2 = k_1k_2 + k_1k_3 + k_2k_3 - \alpha(k_2 + k_3) - \beta_2\rho\lambda - \alpha\eta$

and $a_3 = k_1k_2k_3 + \alpha\beta_2\rho\lambda - k_1\beta_2\rho\lambda - \alpha k_2k_3 - \alpha\eta\epsilon\beta_1k_3 - \alpha\eta\rho\lambda(1-\epsilon)\beta_1$,

The characteristic polynomial $Q(\Psi)$ becomes:

$$Q(\Psi) = \Psi^3 + a_1\Psi^2 + a_2\Psi + a_3. \tag{3.13}$$

To prove the local stability of the disease-free equilibrium point E_0 of the model (2.2) by applying the Routh-Hurwitz criteria, we must show that: $a_1 > 0$, $a_1a_2 - a_3 > 0$ and $a_3 > 0$.

We have $a_1 > 0$ because $k_1 + k_2 + k_3 > \alpha$;

$a_3 = k_1k_2k_3 + \alpha\beta_2\rho\lambda - k_1\beta_2\rho\lambda - \alpha k_2k_3 - \alpha\eta\epsilon\beta_1k_3 - \alpha\eta\rho\lambda(1-\epsilon)\beta_1 = k_1(k_2k_3 - \beta_2\rho\lambda)(1-\mathcal{R}_0)$
$a_3 > 0$, if $\mathcal{R}_0 < 1$;

$a_1a_2 - a_3 = \Big(k_1 + k_2 + k_3 - \alpha\Big)\Big(k_1k_2 + k_1k_3 + k_2k_3 - \alpha(k_2 + k_3) - \beta_2\rho\lambda - \alpha\eta\Big) - k_1(k_2k_3 - \beta_2\rho\lambda)(1-\mathcal{R}_0)$,
that is to say:

$a_1a_2 - a_3 = k_1k_2k_3(2 + \mathcal{R}_0) + \beta_2\rho\lambda k_1(1-\mathcal{R}_0) + k_1k_2\Big(k_1 + k_2 - 2\alpha\Big) + k_1k_3\Big(k_1 + k_3 - 2\alpha\Big) + k_2k_3\Big(k_2 + k_3 - 3\alpha\Big) + (k_2 + k_3)(\alpha^2 - \alpha\eta\epsilon\beta_1) + \alpha\eta\epsilon\beta_1(\alpha - k_1) - \alpha\eta\epsilon\beta_1k_2^2$

Thus, we have $a_1a_2 - a_3 > 0$.
Since the Routh-Hurwitz criteria are satisfied, we conclude that the disease-free equilibrium point is locally and asymptotically stable.

3.5. **Global stability of the disease-free equilibrium point.** To study the global stability of the disease-free equilibrium point E_0, we will construct a Lyapunov function using the matrix method developed by Zhisheng Shuai and P. Van den Drissche. [26].

Theorem 3.4. *The disease-free equilibrium point E_0 is globally and asymptotically stable in Ω if $\mathcal{R}_0 \leq 1$. If $\mathcal{R}_0 > 1$, E_0 is unstable and the disease persists uniformly in the population.*

Proof: The general disease transmission model can then be written as:

$$x' = \mathcal{F}(x, y) - \mathcal{V}(x, y) \tag{3.14}$$

with $x = (I, A, H)^T$ and $y = (S, R)^T$.
A systematic method is presented to guide the construction of the Lyapunov function.

$$f(x, y) = (F - V)x - \mathcal{F}(x, y) + \mathcal{V}(x, y) \tag{3.15}$$

Considering the compartments of infected/infectious individuals, then equation (3.14) can be written as:

$$x' = (F - V)x - f(x, y) \tag{3.16}$$

We have: $f(x,y) = \left[\begin{pmatrix} \alpha & \alpha\eta & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} - \begin{pmatrix} k_1 & 0 & 0 \\ -\epsilon\beta_1 & k_2 & -\rho\lambda \\ -(1-\epsilon)\beta_1 & -\beta_2 & k_3 \end{pmatrix}\right]\begin{pmatrix} I \\ A \\ H \end{pmatrix}$

$$+\begin{pmatrix} k_1 I & 0 & 0 \\ -\epsilon\beta_1 I + k_2 A - \rho\lambda H & & \\ -(1-\epsilon)\beta_1 I - \beta_2 A + k_3 H & & \end{pmatrix} - \begin{pmatrix} \frac{\alpha IS}{N} + \frac{\alpha\eta AS}{N} \\ 0 \\ 0 \end{pmatrix}$$

After some algebraic calculations, we have:

$$f(x,y) = \alpha(I + \eta A)\begin{pmatrix} 1 - \frac{S}{N} \\ 0 \\ 0 \end{pmatrix}$$

Since $S \leq N$, then $f(x,y) \geq 0$ and according to equation (3.10) and equation (3.11), $F \geq 0$ and $V^{-1} \geq 0$

We also have $f\left(x, \left(\frac{\Lambda}{\mu_0}, 0\right)\right) = \alpha(I + \eta A)\begin{pmatrix} 1 - 1 \\ 0 \\ 0 \end{pmatrix} = 0$ in Ω.

$$V^{-1}F = \begin{pmatrix} \frac{\alpha}{k_1} & \frac{\alpha\eta}{k_1} & 0 \\ \frac{\alpha[\epsilon\beta_1 k_3 + \rho\lambda(1-\epsilon)\beta_1]}{k_1(k_2k_3 - \beta_2\rho\lambda)} & \frac{\alpha\eta[\epsilon\beta_1 k_3 + \rho\lambda(1-\epsilon)\beta_1]}{k_1(k_2k_3 - \beta_2\rho\lambda)} & 0 \\ \frac{\alpha[\epsilon\beta_1\beta_2 + \rho\lambda(1-\epsilon)\beta_1 k_2]}{k_1(k_2k_3 - \beta_2\rho\lambda)} & \frac{\alpha\eta[\epsilon\beta_1\beta_2 + \rho\lambda(1-\epsilon)\beta_1 k_2]}{k_1(k_2k_3 - \beta_2\rho\lambda)} & 0 \end{pmatrix}$$

According to [26], the Lyapunov function is defined by: $L = \omega^T V^{-1} x$ where $\omega^T = (\zeta_1, \zeta_2, \zeta_3) \geq 0$ is a left eigenvector of the matrix $V^{-1}F$ corresponding to the eigenvalue $\mathcal{R}_0$. Thus, we have the

following equality:$(\zeta_1,\zeta_2,\zeta_3)V^{-1}F=\mathcal{R}_0(\zeta_1,\zeta_2,\zeta_3)$ that is to say:

$$\begin{cases}\dfrac{\alpha}{k_1}\zeta_1+\dfrac{\alpha[\epsilon\beta_1k_3+\rho\lambda(1-\epsilon)\beta_1]}{k_1(k_2k_3-\beta_2\rho\lambda)}\zeta_2+\dfrac{\alpha[\epsilon\beta_1\beta_2+\rho\lambda(1-\epsilon)\beta_1k_2]}{k_1(k_2k_3-\beta_2\rho\lambda)}\zeta_3=\mathcal{R}_0\zeta_1\\ \dfrac{\alpha\eta}{k_1}\zeta_1+\dfrac{\alpha\eta[\epsilon\beta_1k_3+\rho\lambda(1-\epsilon)\beta_1]}{k_1(k_2k_3-\beta_2\rho\lambda)}\zeta_2+\dfrac{\alpha\eta[\epsilon\beta_1\beta_2+\rho\lambda(1-\epsilon)\beta_1k_2]}{k_1(k_2k_3-\beta_2\rho\lambda)}\zeta_3=\mathcal{R}_0\zeta_2\\ 0=\mathcal{R}_0\zeta_3\end{cases}\tag{3.17}$$

Resolving equation (3.17) gives $\omega^T=\Big(\zeta_1,\dfrac{k_1(\mathcal{R}_0-\frac{\alpha}{k_1})(k_2k_3-\beta_2\rho\lambda}{\alpha(\epsilon\beta_1k_3+\rho\lambda(1-\epsilon)\beta_1)}\zeta_1,0\Big);\zeta_1\in\mathbb{R}^+$

We have: $L=\omega^TV^{-1}x$, that is to say:

$$L=\Big[\Big(\frac{(\mathcal{R}_0-\frac{\alpha}{k_1})}{\alpha}+\frac{1}{k_1}\Big)I+\frac{k_1k_3(\mathcal{R}_0-\frac{\alpha}{k_1})}{\alpha(\epsilon\beta_1k_3+\rho\lambda(1-\epsilon)\beta_1)}A+\frac{k_1\rho\lambda(\mathcal{R}_0-\frac{\alpha}{k_1})}{\alpha(\epsilon\beta_1k_3+\rho\lambda(1-\epsilon)\beta_1)}H\Big]\zeta_1$$

We have: L is a Lyapunov function for the system (2.2) and $L\geq 0\ \forall\ \mathcal{R}_0\geq\dfrac{\alpha}{k_1}$
The differentiation with respect to time t of L gives:

$L'=\omega^TV^{-1}x'=\omega^TV^{-1}[(F-V)x-f(x,y)]$,
that is to say
$L'=\omega^T(\mathcal{R}_0-1)x-\omega^TV^{-1}f(x,y)$

Next, we have:

$$L'=\left(\zeta_1,\frac{k_1\Big(\mathcal{R}_0-\frac{\alpha}{k_1}\Big)(k_2k_3-\beta_2\rho\lambda}{\alpha(\epsilon\beta_1k_3+\rho\lambda(1-\epsilon)\beta_1)}\zeta_1,0\right)(\mathcal{R}_0-1)\begin{pmatrix}I\\A\\H\end{pmatrix}-$$

$$\left[\Big(\frac{(\mathcal{R}_0-\frac{\alpha}{k_1})}{\alpha}+\frac{1}{k_1}\Big)\zeta_1;\frac{k_1k_3(\mathcal{R}_0-\frac{\alpha}{k_1})}{\alpha(\epsilon\beta_1k_3+\rho\lambda(1-\epsilon)\beta_1)}\zeta_1;\frac{k_1\rho\lambda(\mathcal{R}_0-\frac{\alpha}{k_1})}{\alpha(\epsilon\beta_1k_3+\rho\lambda(1-\epsilon)\beta_1)}\zeta_1\right]\alpha(I+\eta A)\begin{pmatrix}1-\frac{S}{N}\\0\\0\end{pmatrix}$$

This finally gives us:

$$L' = (\mathcal{R}_0 - 1)\left(\zeta_1 I + \frac{k_1\left(\mathcal{R}_0 - \frac{\alpha}{k_1}\right)(k_2k_3 - \beta_2\rho\lambda)}{\alpha(\epsilon\beta_1k_3 + \rho\lambda(1-\epsilon)\beta_1}\zeta_1 A\right) - \left(\frac{1}{k_1} + \frac{\left(\mathcal{R}_0 - \frac{\alpha}{k_1}\right)}{\alpha}\right)\alpha(I + \eta A)\begin{pmatrix} 1 - \frac{S}{N} \\ 0 \\ 0 \end{pmatrix}\zeta_1$$

Since $S \leq N$, if $\mathcal{R}_0 \leq 1$, then $L' < 0$. Furthermore, $L' = 0$ for $I = A = 0$ and $S = \frac{\Lambda}{\mu_0}$.Therefore, the largest invariant set of the model (2.2) when $L' = 0$ is the disease-free equilibrium point E_0.
We have constructed an appropriate Lyapunov function for the system (2.2) and shown that this function is strictly decreasing along the trajectories of the system, that is, $L' \leq 0$.
Therefore, according to LaSalle's invariance principle [37], E_0 is globally and asymptotically stable.

4. Numerical simulation

A picture is worth a thousand words, as they say. In this section, we perform numerical simulations to illustrate the theoretical results obtained from the mathematical analysis. We use Matlab Runge-Kutta ode45 [27], [38]. The values of the various parameters used for the numerical simulation of the model (2.2) are based on the literature or assumed to be biologically plausible (see Table 3). The system (2.2) is considered with the initial conditions given by:

$$S(0) = 89040; I(0) = 4700; A(0) = 100; H(0) = 3580; R(0) = 2580, \tag{4.1}$$

the fixed parameter values from Table 3 and a final time value of $T = 20$ (years).
Table 3: Parameter values used for the HIV/AIDS model (2.2).

Parameters	Meaning	Values	Reference
$N(0)$	Total population	100000	assumed
Λ	Recruitment rate	380	assumed
α	HIV/AIDS transmission rate	0.00011	assumed
β_1	Rate of transfer from the compartment of infected individuals without clinical signs to the AIDS compartment	0.3	[28]
β_2	rate of transfer from the AIDS class to the compartment of individuals on treatment	0.33	[29]
λ	rate of transfer of individuals on treatment to the AIDS class	0.2	assumed
η	AIDS infectivity versus HIV infection without clinical signs of the disease	1.05	[30]
p	probability of HIV-positive newborns through vertical transmission	0.0001	Fitting
ϵ	probability of infected individuals without clinical signs progressing to the AIDS phase	0.1	assumed
ρ	Probability of individuals who have failed their treatment and move up to the AIDS class	0.09	[29]
μ_0	HIV natural mortality rate	1/57	assumed
μ_1	HIV-induced mortality rate	0.7	assumed

Figure 2 shows the solution of the system of equations (2.2) with the initial conditions (4.1). The numerical result in Figure 3 for the system (2.2) indicates that the disease-free equilibrium is locally, globally, and asymptotically stable. Indeed, in this case, the transmission rate α is very low, while the recovery rate λ is high relative to the transmission rate, making the basic reproduction number $\mathcal{R}_0$ less than one, $\mathcal{R}_0 = 0,0777 < 1$.

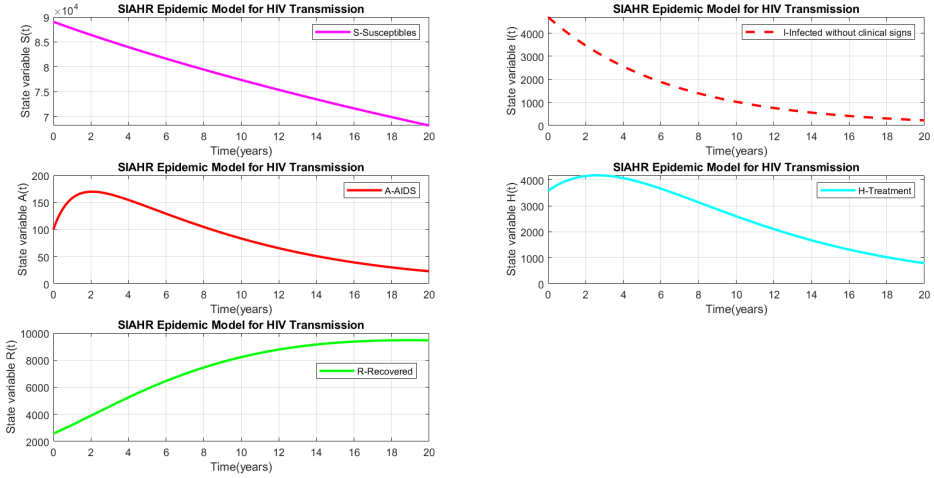

FIGURE 2. *The graph shows the behavior of the equations of the HIV/AIDS transmission model* (2.2).

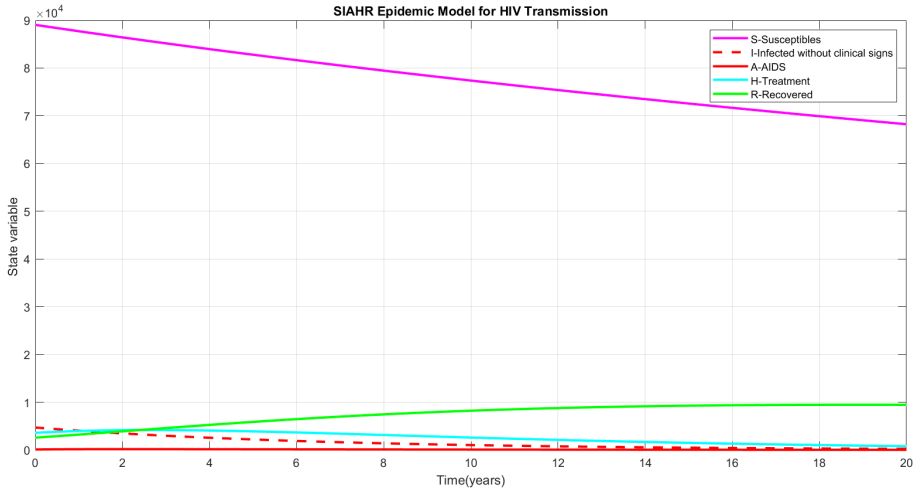

FIGURE 3. *The graph shows the solution curves at the disease-free equilibrium.*

By observing the graphs in these two figures 2, 3, we see that the dynamics of susceptible decreases, as individuals in this compartment migrate to the compartment of infected individuals without clinical signs of the disease. Next, the dynamics of the two infection classes decrease because individuals in these classes undergo treatment and eventually move to the compartment of individuals under treatment. Moreover, the dynamics of this last compartment decrease due to the suppression of viral load in individuals under treatment. These individuals then move to the remission compartment, which explains the growth of the dynamics for this compartment. These results are consistent with the analytical results seen in the previous sections.

5. Analysis of optimal control

In this section, we formulate and study an optimal control problem. The dynamics of an epidemic are observed using time-dependent controls over a finite time interval $[0, T]$. We then apply the Pontryagin maximum principle to determine the conditions for effective control over this interval [13]. The objective is to explore different epidemic control scenarios by incorporating the following control variables into the system model (2.2):

1. The control $u_1 \in [0, 1]$ represents preventive measures (condom use, behavior change, awareness, testing, etc.) taken by susceptible individuals to protect themselves against HIV over the time interval $[0, T]$.

2. The control $u_2 \in [0, 1]$ represents the treatment of infected individuals, with or without clinical symptoms, who have decided to undergo treatment over a certain time interval $[0, T]$.

The controls $u_1(t)$ and $u_2(t)$ aim to minimize both the number of infected individuals and the cost of treatment over the time interval $[0, T]$.

The model (2.2) then becomes:

$$\begin{cases} \dfrac{dS(t)}{dt} = \Lambda - \Lambda pI(t) - \mu_0 S(t) - (1 - u_1(t))\dfrac{\alpha S(t)(I(t) + \eta A(t))}{N}; \\ \dfrac{dI(t)}{dt} = (1 - u_1(t))\dfrac{\alpha S(t)(I(t) + \eta A(t))}{N} + \Lambda pI(t) - (u_2(t)\beta_1 + \mu_0)I(t); \\ \dfrac{dA(t)}{dt} = \epsilon\beta_1 I(t) + \rho\lambda H(t) - (u_2(t)\beta_2 + \mu_0 + \mu_1)A(t); \\ \dfrac{dH(t)}{dt} = (u_2(t) - \epsilon)\beta_1 I(t) + u_2(t)\beta_2 A(t) - (\lambda + \mu_0)H(t); \\ \dfrac{dR(t)}{dt} = (1 - \rho)\lambda H(t) - \mu_0 R(t). \end{cases} \tag{5.1}$$

The objective is to find the optimal values u_1^* and u_2^* of the controls u_1 and u_2 over time, such that the associated state trajectories S^*, I^*, A^*, H^*, and R^* are solutions of the system described in (5.1) over the time interval $[0, T]$ with the following given initial conditions:

$$S(0) \geq 0, I(0) \geq 0, A(0) \geq 0, H(0) \geq 0, R(0) \geq 0 \tag{5.2}$$

and (u_1^*, u_2^*) minimizes the objective functional given by:

$$J(u_1(.), u_2(.)) = \int_0^T (A_1 I + A_2 A + \frac{1}{2}(B_1 u_1^2 + B_2 u_2^2))dt \tag{5.3}$$

Where A_1, A_2, B_1, and B_2 are positive weights. The terms $B_1u_1^2$ and $B_2u_2^2$ represent the costs associated, respectively, with preventive measures (i.e., condom use, behavior change, awareness, testing, $\cdots$) and the treatment of infected individuals with or without clinical symptoms. We chose a quadratic cost for the controls in line with other publications on epidemic control [31], [32], [21]. Indeed, costs are rarely linear and are often presented as nonlinear functions of control. Moreover, this choice also allows for an analogy with the energy expended for these two measures.
The control system described in equation (5.1), consisting of ordinary differential equations in $\mathbb{R}^5$, is considered with the set of admissible control functions defined by:

$$\mathcal{U} = \{(u_1, u_2) \in L^\infty(0,T) | 0 \leq (u_1(t), u_2(t)) \leq u_{max}, \forall t \in [0,T]\} \tag{5.4}$$

We consider that the optimal control problem consists of determining $(S^*(.), I^*(.), A^*(.), H^*(.), R^*(.))$ associated with an admissible control $u_1^*(.) \in \mathcal{U}$ and $u_2^*(.) \in \mathcal{U}$ over the time interval $[0,T]$, satisfying equation (5.1) and the initial conditions from equation (5.2), while minimizing both the number of infected individuals and the cost function from equation (5.3):

$$J(u_1^*(.), u_2^*(.)) = \min_{\mathcal{U}} J(u(.)) \tag{5.5}$$

5.1. **The Pontryagin maximum principle.** he problem is solved using a well-established control theory, as outlined in the book [39], [40] by Lenhart and Workman. The necessary conditions that an optimal control problem must satisfy arise from Pontryagin's Maximum Principle [39]. Thus, the Hamiltonian $\mathcal{H}$ associated with the system (5.1) and the cost (5.3) is given by:

$$\mathcal{H} = A_1I + A_2A + \frac{1}{2}(B_1u_1^2 + B_2u_2^2) + \lambda_S[\Lambda - \Lambda pI(t) - \mu_0 S(t) - (1-u_1(t))\frac{\alpha S(t)(I(t)+\eta A(t))}{N}] +$$
$$\lambda_I[(1-u_1(t))\frac{\alpha S(t)(I(t)+\eta A(t))}{N} + \Lambda pI(t) - (u_2(t)\beta_1 + \mu_0)I(t)] + \lambda_A[\epsilon\beta_1 I(t) + \rho\lambda H(t) - (u_2(t)\beta_2 +$$
$$\mu_0 + \mu_1)A(t)] + \lambda_H[(u_2(t)-\epsilon)\beta_1 I(t) + u_2(t)\beta_2 A(t) - (\lambda+\mu_0)H(t)] + \lambda_R[(1-\rho)\lambda H(t) - \mu_0 R(t)]$$

Where λ_S, λ_I, λ_A, λ_H, and λ_R are adjoint variables, also known as Lagrange multipliers.
By applying Pontryagin's Maximum Principle [39], [33], as well as the existence results for an optimal control problem from [33], we obtain:

Theorem 5.1. *Let S^*, I^*, A^*, H^* and R^* be the optimal solutions with the associated optimal control variables (u_1^*, u_2^*) for the optimal control problems* (5.1) *and* (5.3)*, then there exist adjoint variables λ_S ; λ_I ; λ_A ; λ_H and λ_R satisfying:*

$$\dot{\lambda_S} = (1-u_1^*)\frac{\alpha(I^*+\eta A^*)(N-S^*(t))}{N^2}(\lambda_S - \lambda_I) + \lambda_S\mu_0;$$
$$\dot{\lambda_I} = -A_1 + [(1-u_1^*)\alpha S^*(t)\frac{N-(I^*(t)+\eta * A^*(t))}{N^2} + \Lambda p](\lambda_S - \lambda_I) + \lambda_I(u_2^*\beta_1 + \mu_0) - \lambda_A\epsilon\beta_1 - \lambda_H(u_2^* -$$
$$\epsilon)\beta_1;$$
$$\dot{\lambda_A} = -A_2 + (1-u_1^*)\alpha\eta S^*(t)\frac{N-(I^*(t)+\eta * A^*(t))}{N^2}(\lambda_S - \lambda_I) + \lambda_A(u_2^*\beta_2 + \mu_0 + \mu_1) - \lambda_H u_2^*\beta_2$$
$$\dot{\lambda_H} = -\lambda_A\rho\lambda + \lambda_H(\lambda+\mu_0) - \lambda_R(1-\rho)\lambda;$$
$$\dot{\lambda_R} = \lambda_R\mu_0.$$

With the transversality conditions $\lambda_S(T) = \lambda_I(T) = \lambda_A(T) = \lambda_H(T) = \lambda_R(T) = 0$.
Moreover, the optimal control variables $u_1^*(t)$ *and* $u_2^*(t)$ *are given by:*

$$u_1^*(t) = min\Big\{u_{max}, max\Big\{0, \frac{\alpha S^*(t)(I^*(t) + \eta A^*(t))(\lambda_I - \lambda_S)}{B_1 N}\Big\}\Big\}.$$

$$u_2^*(t) = min\Big\{u_{max}, max\Big\{0, \frac{\lambda_I \beta_1 I^*(t) - \lambda_A \beta_2 A^*(t) - \lambda_H(\beta_1 I^*(t) + \beta_2 A^*(t))}{B_2}\Big\}\Big\}.$$

Proof: In particular, the Hamiltonian is convex with respect to $u_1^*(t)$ and $u_2^*(t)$, which implies the existence of a solution. The first-order condition for optimization is satisfied. Next, we differentiate the Hamiltonian with respect to all the state variables $S, I, A, H \quad and \quad R$, and we obtain the time derivatives $\dot{\lambda}_S(t)$, $\dot{\lambda}_I(t)$, $\dot{\lambda}_A(t)$, $\dot{\lambda}_H(t)$ and $\dot{\lambda}_R(t)$ of the adjoint variables, that is:

$$\frac{d\lambda_S}{dt} = -\frac{\partial \mathcal{H}}{\partial S} = (1 - u_1^*)\frac{\alpha(I^* + \eta A^*)(N - S^*(t))}{N^2}(\lambda_S - \lambda_I) + \lambda_S \mu_0;$$

$$\frac{d\lambda_I}{dt} = -\frac{\partial \mathcal{H}}{\partial I} = -A_1 + [(1 - u_1^*)\alpha S^*(t)\frac{N - (I^*(t) + \eta * A^*(t))}{N^2} + \Lambda p](\lambda_S - \lambda_I) + \lambda_I(u_2^*\beta_1 + \mu_0) - \lambda_A \epsilon \beta_1 - \lambda_H(u_2^* - \epsilon)\beta_1;$$

$$\frac{d\lambda_A}{dt} = -\frac{\partial \mathcal{H}}{\partial A} = -A_2 + (1 - u_1^*)\alpha\eta S^*(t)\frac{N - (I^*(t) + \eta * A^*(t))}{N^2}(\lambda_S - \lambda_I) + \lambda_A(u_2^*\beta_2 + \mu_0 + \mu_1) - \lambda_H u_2^* \beta_2$$

$$\frac{d\lambda_H}{dt} = -\frac{\partial \mathcal{H}}{\partial H} = -\lambda_A \rho \lambda + \lambda_H(\lambda + \mu_0) - \lambda_R(1 - \rho)\lambda$$

$$\frac{d\lambda_R}{dt} = -\frac{\partial \mathcal{H}}{\partial R} = \lambda_R \mu_0.$$

Thus, we obtain the equations stated in Theorem 5.1 with the transversality conditions.$\lambda_S(T) = \lambda_I(T) = \lambda_A(T) = \lambda_H(T) = \lambda_R(T) = 0$.
We then consider the optimal controls. $u_1^*(t)$ and $u_2^*(t)$:

$$\frac{\partial \mathcal{H}}{\partial u_1(t)} = 0 \implies u_1^*(t) = \frac{\alpha S^*(I^* + \eta A^*)(\lambda_I - \lambda_S)}{B_1 N};$$

$$\frac{\partial \mathcal{H}}{\partial u_1(t)} = 0 \implies u_2(t)^* = \frac{\lambda_I \beta_1 I^* - \lambda_A \beta_2 A^* - \lambda_H(\beta_1 I^* + \beta_2 A^*)}{B_2}$$

Next, using standard control arguments involving bounds on the controls, we conclude that for $u_1^*(t)$:

$$u_1(t)^* = \begin{cases} 0, & \frac{\alpha S^*(I^* + \eta A^*)(\lambda_I - \lambda_S)}{B_1 N} \leq 0 \\ \frac{\alpha S^*(I^* + \eta A^*)(\lambda_I - \lambda_S)}{B_1 N}, & 0 < \frac{\alpha S^*(I^* + \eta A^*)(\lambda_I - \lambda_S)}{B_1 N} < 1 \\ u_{max}, & \frac{\alpha S^*(I^* + \eta A^*)(\lambda_I - \lambda_S)}{B_1 N} \geq 1 \end{cases}$$

which can be expressed in compact form as:

$$u_1(t)^* = min\Big\{u_{max}, max\Big\{0, \frac{\alpha S^*(I^* + \eta A^*)(\lambda_I - \lambda_S)}{B_1 N}\Big\}\Big\}.$$

Similarly, for $u_2^*(t)$, we have:

$$u_2(t)^* = min\Big\{u_{max}, max\Big\{0, \frac{\lambda_I \beta_1 I^*(t) - \lambda_A \beta_2 A^*(t) - \lambda_H(\beta_1 I^*(t) + \beta_2 A^*(t))}{B_2}\Big\}\Big\}.$$

The proof is thus completed.

5.2. **Numerical solution of the optimal control problem for HIV.** The extremum given by Theorem 5.1 is now computed numerically by implementing a fourth-order backward Runge-Kutta

method (see, for example, Reference [21]). This iterative method involves solving the system of equation (5.1) with a hypothesis for the controls over the time interval $[0, T]$ using a fourth-order Runge-Kutta scheme and the transversality conditions. $\lambda_S(T) = \lambda_I(T) = \lambda_A(T) = \lambda_H(T) = \lambda_R(T) = 0$. Next, the adjoint system in Theorem 5.1 is solved backward in time using a fourth-order Runge-Kutta scheme, employing the current iteration solution of the system (5.1). The controls are updated using a convex combination of the previous controls and the values u_1^* and u_2^* from **Theorem 5**. The iteration is stopped when the values of the unknowns from the previous iteration are very close to those of the current iteration.

For the numerical simulations, we consider the initial conditions given by (4.1). Additionally, we assume that $u_{max} = 0.5$, represents a shortage of resources or poor utilization of preventive measures against HIV $u_1(.)$ or the treatment of individuals infected with HIV $u_2(.)$, meaning that the set of admissible controls is given by:

$$\mathcal{U} = \{(u_1, u_2) \in L^\infty(0, T) | 0 \leq (u_1(t), u_2(t)) \leq 0.5, \forall t \in [0, T]\} \tag{5.6}$$

with $T = 20$ (years), the weighting constants are assumed to be $A_1 = 0,75$, $A_2 = 0,25$, $B_1 = 2$ and $B_2 = 1$.

Figure 4 shows the numerical solution of the optimal control problem for equations (5.1)-(5.5) with the initial conditions from equation (4.1) and the admissible control set from equation (5.6) for u_1^* only

Figure 5 shows the numerical solution of the optimal control problem for equations (5.1)-(5.5) with the initial conditions from equation (4.1) and the admissible control set from equation (5.6) for u_2^* only.

Figure 6 shows the numerical solution of the optimal control problem for equations (5.1)-(5.5) with the initial conditions from equation (4.1) and the admissible control set from equation (5.6) for u_1^* and u_2^* simultaneously.

Figures 7 and 8 illustrate the behavior of the optimal controls u_1^* and u_2^*, respectively.

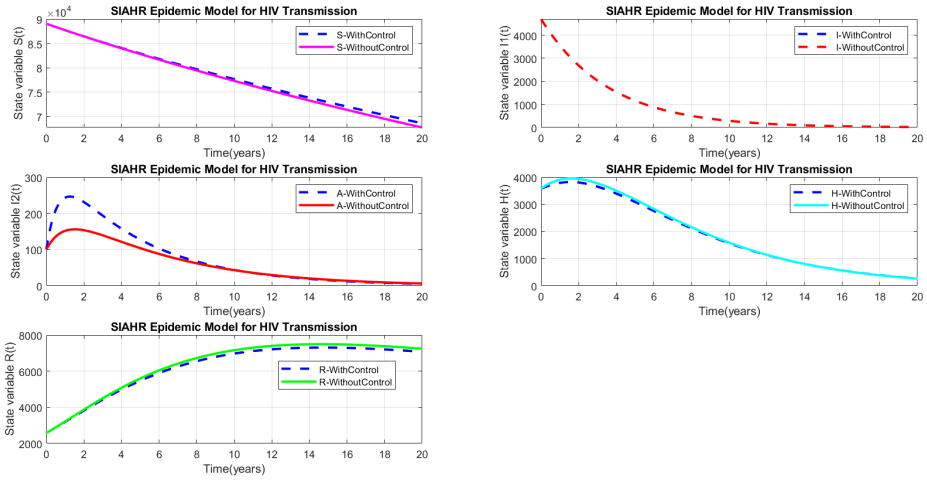

FIGURE 4. *Optimal state variables for the control problem of equations* (5.1)-(5.5) *subject to the initial conditions of equation* (4.1) *and the admissible control set of equation* (5.6) *concerning the dynamics of state variables S, I, A, H, and R using only the control* u_1^*.

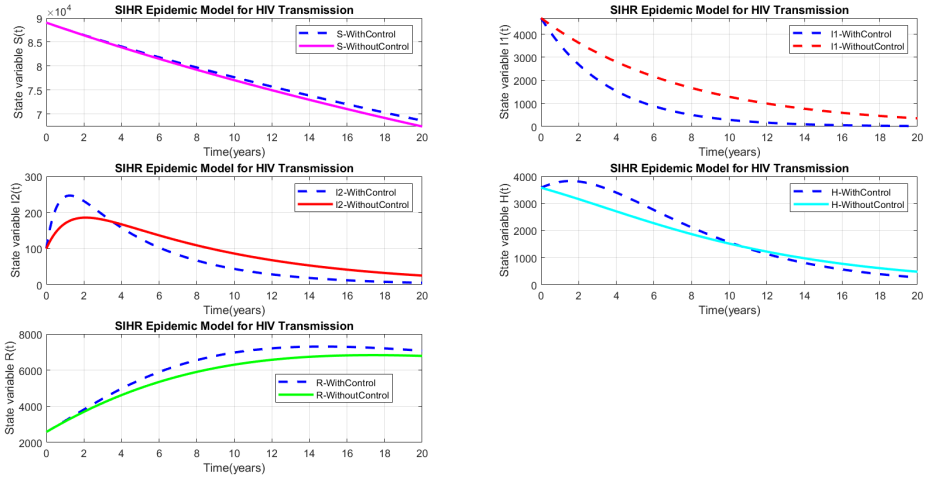

FIGURE 5. *Optimal state variables for the control problem of equations* (5.1)-(5.5) *subject to the initial conditions of equation* (4.1) *and the admissible control set of equation* (5.6) *concerning the dynamics of state variables S, I, A, H, and R using only the control* u_2^*.

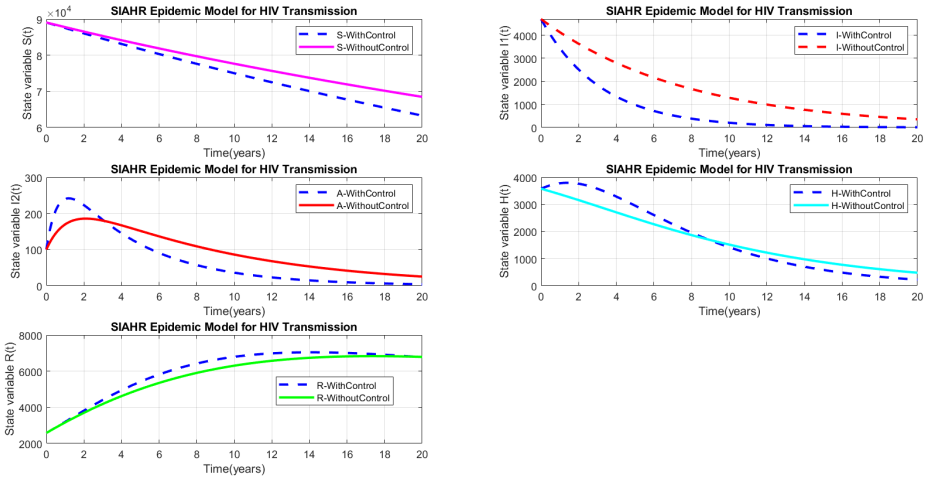

FIGURE 6. *Optimal state variables for the control problem of equations* (5.1)-(5.5) *subject to the initial conditions of equation* (4.1) *and the admissible control set of equation* (5.6)*, concerning the dynamics of state variables S, I, A, H, and R using the control measures* u_1^* *and* u_2^*.

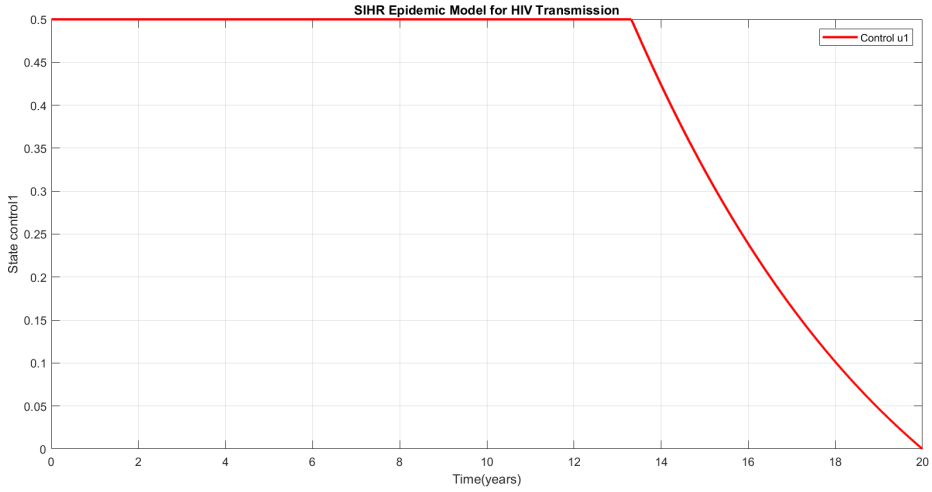

FIGURE 7. *Optimal control* u_1^* *for the HIV optimal control problem in equations* (5.1)-(5.5) *subject to the initial conditions of equation* (4.1) *and the admissible control set of equation* (5.6).

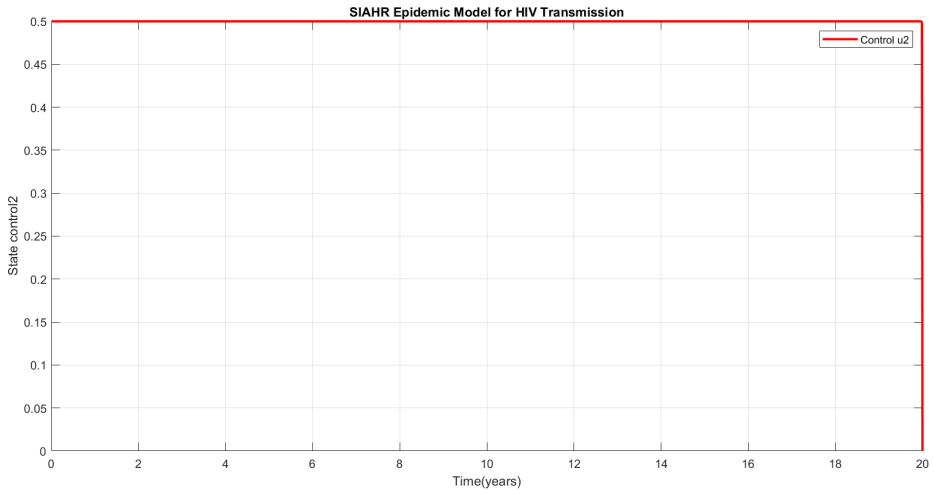

FIGURE 8. *Optimal control u_2^* for the HIV optimal control problem in equations* (5.1)-(5.5) *subject to the initial conditions of equation* (4.1) *and the admissible control set of equation* (5.6).

By performing numerical simulations using only the preventive measures u_1, then with treatment u_2 alone, and finally combining both controls u_1 and u_2, we observe that treatment is more effective than preventive measures in reducing the spread of HIV/AIDS. Indeed, in Figure 4, where only u_1 was used, the results differ from those in Figure 6, where both controls u_1 and u_2 were applied. On the other hand, in Figure 5, where only u_2 was used, we obtain trajectories that are approximately equal to those in Figure 6, where both controls u_1 and u_2 were applied. By comparing Figure 2, which presents the model without control variables, and Figure 6, where two control variables have been introduced the preventive measures u_1 and the treatment u_2 we observe that the combined interventions, as illustrated in Figure 6, are effective in reducing the number of individuals infected with HIV/AIDS. The numerical results clearly illustrate our main objective in applying the optimal control tool, which is to minimize the number of infected individuals and treatment costs while maximizing the number of individuals in remission.

6. CONCLUSION

In this article, we formulated a deterministic model to describe the spread and transmission of HIV/AIDS in a population. After formulating the model, we determined the basic reproduction number $\mathcal{R}_0$, which predicts that the disease will vanish in the population if $\mathcal{R}_0 < 1$ or will persist if $\mathcal{R}_0 > 1$, after studying the existence and stability of the disease-free equilibrium. Numerical simulations show that with antiretroviral treatments, HIV can disappear or at least be controlled in a given population. Medical research should aim at preventing HIV through vaccines or developing new antiretroviral molecules that will eliminate HIV from the human body so that patients can permanently stop lifelong treatments. This will allow us to have mathematical models of HIV with

compartments for recovered individuals. Additionally, we included two time-dependent control variables: preventive measures and the treatment of infected individuals. The results show that treatment is more cost-effective in reducing the spread of HIV than preventive measures. Finally, the numerical results from the optimal control section clearly show that combined interventions, as illustrated in Figure 6, are effective in reducing the number of individuals infected with HIV/AIDS.

Conflicts of Interest: The authors declare that there are no conflicts of interest regarding the publication of this paper.

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