Global Stability and Optimal Control of an HIV/AIDS Epidemic Model with Behavioral Change and Treatment

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***Abstract*—In this paper, we consider a deterministic HIV/AIDS model to study the effect of information campaigns and treatment on the spread of HIV/AIDS. We demonstrate that the disease-free equilibrium is globally asymptotically stable when the basic reproduction numbers are less than one. However, if the basic reproduction number is greater than one, then a unique endemic equilibrium exists and it is globally asymptotically stable for a special case. The sensitivity analysis reveals that the effective contact rates of susceptible individuals with asymptomatic infected (pre-AIDS) individuals among other parameters contributed most significantly to the transmission and spread of HIV/AIDS. For the time-dependent controls, we formulated an appropriate optimal control problem. The Pontryagin's Maximum Principle was applied to find the necessary conditions for the existence of optimal control. The optimal system was solved using the fourth-order Runge-Kutta forward-backwards sweep method. The numerical results showed that the control strategies have a significant effect in reducing the numbers of infected individuals. The cost-effectiveness analysis reveals that the control measure implementing treatment is the most cost-effective among the strategies considered.**

***Index Terms*—HIV/AIDS model, education campaigns, basic reproduction number, optimal control**

I. INTRODUCTION

THE Human Immunodeficiency Virus (HIV) has been the subject of scientific research and debate since the virus was identified in the 1980s. The virus causes acquired immune deficiency syndrome (AIDS), which first spread along the historic trade routes of Congo valley in the 1920s [1]. Viruses destroy human immunity as a result of a decreased immune system. The spread of HIV/AIDS continues to increase worldwide and is a major threat to society. According to an estimate of the global situation and trends from the start of the epidemic (1980) until the end of 2018 from WHO [2], there were 37.9 million people living with HIV, and about 62% (23.5 million people) of people with HIV receiving antiretroviral treatment in 2018 [2]. Furthermore, there were 1.7 million people newly infected with HIV and 770,000 people who died from HIV-related illness in 2018. The initial global response to HIV focuses on prevention through behavioral changes and research on vaccines. With no primary vaccine to stop HIV transmission and expensive medical treatment, education campaigns are the less costly public response to reduce new infection. Over the years, the increasing epidemic in sub-Saharan Africa has been responded to using the ABC approach (Abstinence, Faithful, Condom). More studies showed that the ABC message initiated by the Uganda government in 1992 has changed behavior and there has been a dramatic decrease in HIV prevalence in Uganda [3], [4]. However, this approach is not enough to stop the epidemic. According to [5], to accelerate HIV/AIDS prevention efforts, it is essential to combine prevention efforts between behavioral strategy and biomedical strategy.

Mathematical models are useful to analyze the transmission dynamics of HIV and have received significant attention from researchers around the world (see, e.g. [6], [7], [8]]). So far, several studies have been developed to mathematically analyze the impact of public health education campaigns, screening of unaware infectives, and treatment (therapy) on the spread of HIV/AIDS. Joshi et al. [9] developed a modified SIR model dividing susceptible individuals into two classes based on AB and C behavior and the resulting different infectivity rates to investigate the effect of information campaigns on the HIV epidemic in Uganda. Moreover, research in [10], [11], [12], [13] showed that public health education campaigns were essential for HIV/AIDS transmission dynamics. This study showed that an important public health education campaigns that focused on the change of risky behavior with a reasonable coverage level could help prevent the spread of HIV/AIDS. Furthermore, treatment (antiretroviral therapy) or screening have been used to reduce the spread of HIV/AIDS (see [14], [15], [16] and the references therein). Global stability of the equilibrium point for the HIV/AIDS epidemic model has been of concern to some researchers, for example [18], [19], [20], [21], [22]. These studies, the global stability of equilibrium points is obtained using Lyapunov's direct method combined with LaSalle's invariance principle.

Optimal control theory is a powerful mathematical tool [23], [24] to solve epidemiological model problems in their

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efforts to find primary control strategies to minimize the number of people infected. Joshi *et al*. [25] studied an extended SIR model by changing behavior through information campaigns (education) as a control for managing disease epidemics if available treatment was not available or was too expensive. Hota *et al*. [26] extended the model in [25] by adding treatment interventions as a second control in addition to the education campaigns. Further studies on application of optimal control for HIV/AIDS can be found in [27], [28], [29], [30] and references therein.

In this paper, we considered an HIV/AIDS epidemic with behavioral change and treatment. The model developed considered the combination of aspects of behavioral interventions through information campaigns (ABC approach) in susceptible individuals and biomedical interventions through the treatment of individuals with pre-AIDS. This is in line with the effective prevention of HIV epidemic which requires a combination of behavioral interventions, biomedical interventions, and structural interventions [31].

This paper is organized as follows. In Section 2, an HIV model with information campaigns and treatment is formulated. The global asymptotic stability of the disease-free and endemic equilibrium points of the model is investigated in Section 3. In Section 4, we present the optimal control analysis followed by numerical simulations of the model in Section 5. Finally, some conclusions are described in Section 6.

II. MODEL FORMULATION

We considered a sexually active population and the total population N divided into seven subpopulations depending on individuals' HIV/AIDS status. The classes consisted of susceptibles individuals (S), susceptible individuals who choose to be abstinent and be faithful due to information campaigns or AB behavior group (S_1), susceptible individuals who used condom due to information campaigns or C behavior group (S_2), asymptomatic infected individuals (I) who did not receive treatment, pre-AIDS individuals (P) who did not receive treatment, treated individuals (T), and individuals with full-blown AIDS (A) not receive treatment, so that, $N = S + S_1 + S_2 + I + P + T + A$.

Due to the interaction of individuals in class S with the control of information campaigns E, susceptible individuals are divided into three subclasses (S, S_1, S_2). The proportion of the susceptibles S moves to S_1 and S_2 at the rate α_1 and α_2 is $\alpha_i ES_i, i = 1, 2$ [26], [27]. The susceptible individuals (S, S_1, S_2) are individuals who are not yet infected but can be infected through sexual contacts with two types of infective (*I*, *P*). These two types of infective are considered active in spreading the infection. Treated class are individuals who use the treatment after having pre-AIDS which can become full-blown AIDS. It is assumed that the incidence in human interaction is standard incidence. Asymptomatic infected individuals and pre-AIDS individuals can infect susceptible at different rates β_1 and β_2, respectively where $\beta_1 < \beta_2$. Thus, the force of infection is given by

$$\beta = \frac{\beta_1 I + \beta_2 P}{S + S_1 + S_2 + I + P + T + A}.$$

It is also assumed that the sexually mature susceptible individuals are recruited into the population at a constant rate Π. Susceptible individuals is reduced by infection, following primary contact with asymptomatic infected individuals at the rate β_1 and pre-AIDS individuals at the rate β_2 for the model with standard incidence rate. It is facilitated by the interaction of individual in susceptible individuals who have been impacted by the control of information campaigns (E) and move to susceptible individuals who are abstinent and faithful (AB behavior) at the rate α_1 and move to susceptible individuals who use a condom (C behavior) at the rate α_2. It is reduced further by a natural death at the rate μ. Therefore, the rate of change of the susceptible individuals is given by

$$\frac{dS}{dt} = \Pi - (\beta + E\alpha_1 + E\alpha_2)S - \mu S.$$

The population of susceptible individuals who AB behavior due to education campaigns is increased by the proportion of the susceptible individuals who leave the general susceptible at the rate α_1. It is diminished by the infection of susceptible individuals whose AB behavior due to education campaigns at the rate $(1-\psi_1)\beta$, where ψ_1 measured the efficacy of information campaigns into AB behavior group. It is reduced further by a natural death at the rate μ. Thus, the rate of change of susceptible individuals who have AB behavior due to information campaigns is given by

$$\frac{dS_1}{dt} = E\alpha_1 S - (1-\psi_1)\beta S_1 - \mu S_1.$$

The population of susceptible individuals who have C behavior due to information campaigns has increased by the proportion of the susceptible individuals who leave the general susceptible at the rate α_2. It is reduced by infection of susceptible individuals who have C behavior due to information campaigns at the rate $(1-\psi_2)\beta$, where for C behavior group. It is reduced further by natural death at the rate μ. Thus, the rate of change of susceptible individuals who have C behavior due to information campaigns is given by

$$\frac{dS_2}{dt} = E\alpha_2 S - (1-\psi_2)\beta S_2 - \mu S_2.$$

The population of asymptomatic infected individuals is increased by infection of susceptible individuals at the rate β, susceptible individuals who are abstinent and faithful, or have AB behavior, due to information campaigns at the rate $(1-\psi_1)\beta$, and susceptible individuals who use condom,

or have C behavior, due to information campaigns at the rate $(1-\psi_2)\beta$. It is reduced by treatment of progression to pre-AIDS individuals at the rate σ_1, and a natural death at the rate μ. Hence, mathematically, it is:

$$\frac{dI}{dt} = \beta S + (1-\psi_1)S_1 + (1-\psi_2)S_2 - (\sigma_1 + \mu)I.$$

The population of pre-AIDS individuals is generated by following the development of asymptomatic infected individuals at the rate σ_1. It is diminished by treatment of pre-AIDS individuals at the rate δ, progression to full-blown AIDS at the rate σ_2, and natural death at the rate μ. The corresponding differential equation is given by

$$\frac{dP}{dt} = \sigma_1 I - (\delta + \sigma_2 + \mu)P.$$

Finally, the population of individuals with full-blown AIDS is increased by progression to full-blown AIDS (at the rate σ_2 for pre-AIDS individuals and σ_3 for treated individuals). It is decreased by natural death at the rate μ and by disease-induced mortality at the rate γ. Thus,

$$\frac{dA}{dt} = \sigma_2 P + \sigma_3 T - (\gamma + \mu)A.$$

Thus, the model for HIV/AIDS prevention through information campaigns and treatment of pre-AIDS individuals is given by the following nonlinear differential equations (the detailed biological descriptions of all the parameters are given Table I):

$$\begin{aligned}
\frac{dS}{dt} &= \Pi - (\beta + E\alpha_1 + E\alpha_2)S - \mu S,\\
\frac{dS_1}{dt} &= E\alpha_1 S - (1-\psi_1)\beta S_1 - \mu S_1,\\
\frac{dS_2}{dt} &= E\alpha_2 S - (1-\psi_2)\beta S_2 - \mu S_2,\\
\frac{dI}{dt} &= \beta S + (1-\psi_1)\beta S_1 + (1-\psi_2)\beta S_2 - (\sigma_1 + \mu)I,\\
\frac{dP}{dt} &= \sigma_1 I - (\delta + \sigma_2 + \mu)P,\\
\frac{dT}{dt} &= \delta P - (\sigma_3 + \mu)T,\\
\frac{dA}{dt} &= \sigma_2 P + \sigma_3 T - (\gamma + \mu)A.
\end{aligned} \tag{1}$$

The initial conditions for system (1) are given as follows

$$\begin{aligned}
&S(0) = S^0,\ S_1(0) = S_1^0,\ S_2(0) = S_2^0,\ I(0) = I^0,\\
&P(0) = P^0,\ T(0) = T^0,\ A(0) = A^0.
\end{aligned} \tag{2}$$

A. Positivity of Solutions

Since (1) is the model that monitors changes in the human population, it is assumed that all parameters to be positive for all $t > 0$. Further, the solution of the system (1) is nonnegative, we stated and proved the following lemma.

Lemma 2.1. *Let* $S(0), S_1(0), S_2(0), I(0), P(0), T(0), A(0)$ *are positive, then the solution of the system (1) is nonnegative for all* $t > 0$.

Proof. Let $t_1 = \sup\{t > 0 : S > 0, S_1 > 0, S_2 > 0, I \geq 0, P \geq 0, P \geq 0, T \geq 0, A \geq 0 \text{ in } [0, t]\}$. From the first equation of system (1) we have

$$\frac{dS}{dt} = \Pi - \beta(t)S - (E\alpha_1 + E\alpha_2 + \mu)S, \tag{3}$$

where $\beta(t) = \frac{\beta_1 I + \beta_2 P}{N}$. The equation (3) is equivalent to

$$\frac{dS}{dt} + (E\alpha_1 + E\alpha_2 + \mu + \beta(t))S = \Pi,$$

and this implies that

$$\begin{aligned}
&\frac{dS}{dt}\left[S(t)\exp\left\{(E\alpha_1 + E\alpha_2 + \mu)u + \int_0^t \beta(u)du\right\}\right]\\
&= \Pi\exp\left\{(E\alpha_1 + E\alpha_2 + \mu)u + \int_0^t \beta(u)du\right\}.
\end{aligned}$$

Integrating both sides from $t = 0$ to $t = t_1$ we get

$$\begin{aligned}
&S(t_1)\left[\exp\left\{(E\alpha_1 + E\alpha_2 + \mu)t_1 + \int_0^{t_1} \beta(u)du\right\}\right] - S(0)\\
&= \int_0^{t_1} \Pi\exp\left\{(E\alpha_1 + E\alpha_2 + \mu)x + \int_0^x \beta(v)dv\right\}dx.
\end{aligned}$$

Hence,

$$\begin{aligned}
S(t_1) &= S(0)\exp\left\{-\left((E\alpha_1 + E\alpha_2 + \mu)t_1 + \int_0^{t_1}\beta(u)du\right)\right\}\\
&\quad + \exp\left\{-\left((E\alpha_1 + E\alpha_2 + \mu)t_1 + \int_0^{t_1}\beta(u)du\right)\right\}\\
&\quad \times \int_0^{t_1} \Pi\exp\left\{(E\alpha_1 + E\alpha_2 + \mu)x + \int_0^x \beta(u)du\right\}dx\\
&> 0,
\end{aligned}$$

where $S(0)$ represents the initial conditions of the susceptible. Thus, $S(t)$ equal to the sum of positive terms, it is also positive for $t > 0$.

In the same way, it can be shown that $S_1 > 0, S_2 > 0, I \geq 0, P \geq 0, T \geq 0, A \geq 0$ for all time $t > 0$. This completes the proof. □

B. Positively Invariant

Lemma 2.2. *Let* $(S, S_1, S_2, I, P, T, A)$ *be the solution of the system (1) with initial conditions* $S(0), S_1(0), S_2(0), I(0), P(0), T(0), A(0)$ *nonnegative and the biological feasible region*

Ω *with*

$$\Omega = \left\{ \left(S, S_1, S_2, I, P, T, A\right) \in R_+^7 \mid N \le \tfrac{\Pi}{\mu} \right\}. \tag{4}$$

Then, Ω *is positively invariant.*

Proof. The total population $N(t) = S(t) + S_1(t) + S_2(t) + I(t) + P(t) + T(t) + A(t)$. Differentiation of $N(t)$ with respect to time and by adding all the equations of system (1), we have

$$\frac{dN}{dt} = \Pi - \mu N(t) - \gamma A \le \Pi - \mu N(t). \tag{5}$$

Solving this differential equation and by using standard comparison theorem in [31], we obtain

$$N(t) \le N(0)e^{-\mu t} + \frac{\Pi}{\mu}\left(1 - e^{-\mu t}\right), \tag{6}$$

where $N(0)$ represents the initial values of the total population. Moreover, if $N(0) \le \Pi/\mu$, then $N(t) \le \Pi/\mu$. From equation (6), it is clear that $\limsup_{t\to\infty} N(t) \le \Pi/\mu$ we have $N(t) \le \Pi/\mu$. On the other hand, if $N(0) > \Pi/\mu$, then the solution N will decrease to Π/μ as $t \to \infty$. This means that if $N(0) > \Pi/\mu$, then the solution $(S, S_1, S_2, I, P, T, A)$ enters the closed set Ω or asymptotically approaches Ω. Thus, the region Ω is positively invariant.

III. MODEL ANALYSIS

A. Existence and Stability of Equilibria

Disease-Free Equilibrium and Basic Reproduction

The system (1) has a disease-free equilibrium E_0, obtained by setting the right-hand sides of the equations in (1) to zero, given by

$$E_0 = \left(\frac{\Pi}{E\alpha_1 + E\alpha_2 + \mu}, \frac{E\alpha_1\Pi}{\mu(E\alpha_1 + E\alpha_2 + \mu)}, \frac{E\alpha_2\Pi}{\mu(E\alpha_1 + E\alpha_2 + \mu)}, 0, 0, 0, 0 \right). \tag{7}$$

Using the next-generation matrix method described by [33], [34], we can calculate the basic reproduction number of the model system (1). By using the notation as in [33], $\mathcal{F}_i$ is the rate at which new infections appear in compartment i and $\mathcal{V}_i$ is the rate of transfer of individuals into and out of compartment i. Let $x = (I, P, T, A)^T$. The right-hand side of system (1) is written as $\mathcal{F}_i(x) - \mathcal{V}_i(x)$,

TABLE I
THE DESCRIPTIONS OF PARAMETERS OF MODEL

Parameter	Description of Parameters
Π	Recruitment rate
β_1, β_2	Effective contact rates of susceptible individuals with asymptomatic infected individuals and pre-AIDS individuals
σ_1, σ_2	Progression rate from asymptomatic infected into pre-AIDS and progression rate from pre-AIDS into full-blown AIDS individuals
σ_3	Rate at which treated individuals develop full-blown AIDS
ψ_1, ψ_2	The efficiency of information campaigns into S_1 and S_2
α_1, α_2	Rate of educating adults into S_1 and S_2
E	Rate of information campaigns
δ	Rate of treatment of screened infective
μ	Natural death rate
γ	AIDS-induced death rate

$$\mathcal{F}_i(x) = \begin{pmatrix} \beta S + k_1\beta S_1 + k_2\beta S_2 \\ 0 \\ 0 \\ 0 \end{pmatrix}, \quad \mathcal{V}_i(x) = \begin{pmatrix} (\sigma_1 + \mu)I \\ -\sigma_1 I + (\delta + \sigma_2 + \mu)P \\ -\delta P + (\sigma_3 + \mu)T \\ -\sigma_2 P - \sigma_3 T + (\gamma + \mu)A \end{pmatrix}. \tag{8}$$

The matrices F is the Jacobian matrices of $\mathcal{F}_i$ at E_0 and the matrices V is the Jacobian matrices of $\mathcal{V}_i$ at E_0 are given by

$$F = \begin{pmatrix} \frac{\beta_1 B}{Q} & \frac{\beta_2 B}{Q} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, V = \begin{pmatrix} K & 0 & 0 & 0 \\ -\sigma_1 & L & 0 & 0 \\ 0 & -\delta & M & 0 \\ 0 & -\sigma_2 & -\sigma_3 & W \end{pmatrix}, \tag{9}$$

where

$$B = k_1 E\alpha_1 + k_2 E\alpha_2 + \mu,\ Q = E\alpha_1 + E\alpha_2 + \mu,$$
$$K = \sigma_1 + \mu,\ L = \delta + \sigma_2 + \mu,\ M = \sigma_3 + \mu,\ W = \gamma + \mu,$$
$$k_1 = 1 - \psi_1,\ k_2 = 1 - \psi_2.$$

The eigenvalues of the next generation matrix FV^{-1} are

$$\left\{ 0, 0, 0, \frac{\beta_1 B}{QK} + \frac{\beta_2 \sigma_1 B}{QKL} \right\}. \tag{10}$$

The basic reproduction number of the system will be the spectral radius of matrix FV^{-1} denoted by R_0 and thus

$$R_0 = \rho(FV^{-1}) = \frac{\beta_1 B}{QK} + \frac{\beta_2 \sigma_1 B}{QKL}. \tag{11}$$

The basic reproduction number shows the average number of new infections caused by a single HIV-infected individual in a population where education campaigns and treatment are used to control strategies.

The local stability of the disease-free equilibrium E_0 holds the following Theorem 2 of [33].

Theorem 3.1. *The disease-free equilibrium of the system (1), E_0, is locally asymptotically stable if $R_0 < 0$ and unstable if $R_0 > 0$.*

Proof. The Jacobian matrix of the system (1) at the disease-free equilibrium point E_0 is

$$J(E_0) = \begin{pmatrix} -Q & 0 & 0 & -\frac{\beta_1\mu}{Q} & -\frac{\beta_2\mu}{Q} & 0 & 0 \\ E\alpha_1 & -\mu & 0 & -\frac{k_1\beta_1 E\alpha_1}{Q} & -\frac{k_1\beta_2 E\alpha_1}{Q} & 0 & 0 \\ E\alpha_2 & 0 & -\mu & -\frac{k_2\beta_1 E\alpha_2}{Q} & -\frac{k_2\beta_2 E\alpha_2}{Q} & 0 & 0 \\ 0 & 0 & 0 & \frac{\beta_1 B}{Q} - K & \frac{\beta_2 B}{Q} & 0 & 0 \\ 0 & 0 & 0 & \sigma_1 & -L & 0 & 0 \\ 0 & 0 & 0 & 0 & \delta & -M & 0 \\ 0 & 0 & 0 & 0 & \sigma_2 & \sigma_3 & -W \end{pmatrix}. \tag{12}$$

There are seven eigenvalues of the matrix $J(E_0)$, three of the eigenvalues are $\lambda_1 = -W, \lambda_2 = \lambda_3 = -\mu, \lambda_4 = -M, \lambda_5 = -Q$ and the remaining eigenvalues are given by reducing matrix $J(E_0)$ into 2×2 matrix as shown below,

$$J_1 = \begin{pmatrix} \frac{\beta_1 B}{Q} - K & \frac{\beta_2 B}{Q} \\ \sigma_1 & -L \end{pmatrix}. \tag{13}$$

Using an elementary row operation to reduce the matrix J_1 to echelon form, we obtain the following matrix

$$J_2 = \begin{pmatrix} -d_{11} & \frac{\beta_2 B}{Q} \\ 0 & -d_{22} \end{pmatrix}, \tag{14}$$

where

$$d_{11} = \frac{KQ - \beta_1 B}{Q} = K\left(1 - \frac{\beta_1 B}{QK}\right),$$

$$d_{22} = \frac{KLQ - \beta_1 BL - \beta_2\sigma_1 B}{KQ - \beta_1 B} = \frac{KLQ\left(1 - \frac{\beta_1 B}{QK} - \frac{\beta_2\sigma_1 B}{QKL}\right)}{KQ\left(1 - \frac{\beta_1 B}{QK}\right)} = \frac{L\left(1 - \frac{\beta_1 B}{QK} - \frac{\beta_2\sigma_1 B}{QKL}\right)}{1 - \frac{\beta_1 B}{QK}}.$$

From $R_0 < 1$ caused

$$1 > \frac{\beta_1 B}{QK} + \frac{\beta_2\sigma_1 B}{QKL} > \frac{\beta_1 B}{QK} \quad \text{and} \quad 1 > \frac{\beta_1 B}{QK} + \frac{\beta_2\sigma_1 B}{QKL}.$$

Hence,

$$1 - \frac{\beta_1 B}{QK} > 0 \quad \text{and} \quad 1 - \frac{\beta_1 B}{QK} - \frac{\beta_2\sigma_1 B}{QKL} > 0.$$

Thus, the eigenvalues of the Jacobian matrix $J(E_0)$ are

$$\lambda_1 = -Q, \lambda_2 = \lambda_3 = -\mu, \lambda_4 = -M, \lambda_5 = -W,$$
$$\lambda_6 = -d_{11} = -K\left(1 - \frac{\beta_1 B}{QK}\right),$$
$$\lambda_7 = -d_{22} = -\left(\frac{L\left(1 - \frac{\beta_1 B}{QK} - \frac{\beta_2\sigma_1 B}{QKL}\right)}{1 - \frac{\beta_1 B}{QK}}\right).$$

Hence, all eigenvalues of the Jacobian matrix $J(E_0)$ have negative real parts. Using the Routh-Hurwitz criteria, the disease-free equilibrium point E_0 of system (1) is locally asymptotically stable in Ω when $R_0 < 1$. □

Global Stability of Disease-free Equilibrium

In the following, we will prove the global stability of the disease-free equilibrium E_0. We observed the global stability property of the disease-free equilibrium of the system (1) to ensure that the elimination of HIV did not depend on the number of initial sub-population of the model. For this purpose, we considered the feasible region

$$\Omega_1 = \left\{X = (S, S_1, S_2, I, P, T, A) \in \Omega | S \le S^0, S_1 \le S_1^0, S_2 \le S_2^0\right\}.$$

Lemma 3.1. *The region Ω_1 is positively invariant for the system (1).*

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

$$\frac{dS}{dt} = \Pi - \beta S - QS \le \Pi - QS.$$

Solving differential equation and by using standard comparison theorem [31], we obtain

$$S \le \tfrac{\Pi}{Q} - \left(\tfrac{\Pi}{Q} - S(0)\right)e^{-Qt} = S^0 - \left(S^0 - S(0)\right)e^{-Qt}$$

where $S^0 = \frac{\Pi}{Q}$. Thus, if $S(0) \le S^0$ for all $t \ge 0$, then $S(t) \le S^0$ for all $t \ge 0$.

Using similar approach, from the second and the third equations of the system (1), it is proved that for all $t \ge 0$, $S_1(t) \le S_1^0$ if $S_1(0) \le S_1^0$ where $S_1^0 = \frac{E\alpha_1\Pi}{\mu Q}$ and $S_2(t) \le S_2^0$ if $S_2(0) \le S_2^0$ where $S_2^0 = \frac{E\alpha_2\Pi}{\mu Q}$. Hence, by adding the above three we have the region Ω_1. □

In the next theorem we have studied the global asymptotic stability of the disease-free equilibrium E_0 of

the system (1). To investigate the global stability of E_0, we used the method presented by Castillo-Chavez *et al*. [35] and reading [32].

Theorem 3.2. *The disease-free equilibrium E_0 of the system (1) is globally asymptotically stable in Ω_1 if $R_0 \le 1$.*

Proof. Following Castillo-Chavez *et al*. [35], let $\mathbf{Y}=(S, S_1, S_2)$ represent the number of non-infectious individuals and $\mathbf{Z}=(I_1, I_2, T, A)$ represent the number of infected individuals. Then system (1) can be written as

$$\begin{aligned} \frac{d\mathbf{Y}}{dt} &= F(\mathbf{Y},\mathbf{Z}), \\ \frac{d\mathbf{Z}}{dt} &= G(\mathbf{Y},\mathbf{Z}),\ G(\mathbf{Y},\mathbf{0})=\mathbf{0}, \end{aligned} \tag{15}$$

where

$$\frac{d\mathbf{Y}}{dt} = F(\mathbf{Y},\mathbf{Z}) = \begin{pmatrix} \Pi - \beta S - QS \\ E\alpha_1 S - k_1\beta S_1 - \mu S_1 \\ E\alpha_2 S - k_2\beta S_2 - \mu S_2 \end{pmatrix} \text{ and}$$

$$G(\mathbf{Y},\mathbf{Z}) = \begin{pmatrix} \beta S + k_1\beta S_1 + k_2\beta S_2 - KI \\ \sigma_1 I - LP \\ \delta P - MT \\ \sigma_2 P + \sigma_3 T - WA \end{pmatrix}.$$

Consider the disease-free equilibrium

$$E_0 = (S^0, S_1^0, S_2^0, 0, 0, 0, 0) = (\mathbf{Y}^0, 0, 0, 0, 0),$$

where $\mathbf{Y}^0 = \left(\frac{\Pi}{Q}, \frac{E\alpha_1\Pi}{\mu Q}, \frac{E\alpha_2\Pi}{\mu Q}\right)$. We need show the two following conditions (H_1) and (H_2) are satisfied:

(H_1) For $\frac{d\mathbf{Y}}{dt} = F(\mathbf{Y},\mathbf{0})$, $\mathbf{Y}^0$ is globally asymptotically stable in Ω_1.

(H_2) $G(\mathbf{Y},\mathbf{0})=\mathbf{0}$ and $G(\mathbf{Y},\mathbf{Z}) = C\mathbf{Z} - \hat{G}(\mathbf{Y},\mathbf{Z})$, $\hat{G}(\mathbf{Y},\mathbf{Z}) \ge \mathbf{0}$ for $(\mathbf{Y},\mathbf{Z}) \in \Omega$ and $C = D_{\mathbf{Z}} G(\mathbf{Y}^0,\mathbf{0})$.

From the first equation of the system (15)

$$\frac{d\mathbf{Y}}{dt} = F(\mathbf{Y},\mathbf{0}) = \begin{pmatrix} \Pi - QS \\ E\alpha_1 S - \mu S_1 \\ E\alpha_2 S - \mu S_2 \end{pmatrix}. \tag{16}$$

The solution of (16) are

$$S = \frac{\Pi}{Q} + \left(S(0) - \tfrac{\Pi}{Q}\right)e^{-Qt},$$

$$S_1 = \frac{E\alpha_1\Pi}{\mu Q} - \left(\frac{E\alpha_1\left(\frac{\Pi}{\mu} - S(0)\right)}{Q-\mu} - S_1(0)\right)e^{-\mu t} - \left(\frac{E\alpha_1\left(S(0) - \frac{\Pi}{Q}\right)}{Q-\mu}\right)e^{-Qt},$$

$$S_2 = \frac{E\alpha_2\Pi}{\mu Q} - \left(\frac{E\alpha_2\left(\frac{\Pi}{\mu} - S(0)\right)}{Q-\mu} - S_2(0)\right)e^{-\mu t} - \left(\frac{E\alpha_2\left(S(0) - \frac{\Pi}{Q}\right)}{Q-\mu}\right)e^{-Qt}.$$

It can be shown $S \to \frac{\Pi}{Q}$, $S_1 \to \frac{E\alpha_1\Pi}{\mu Q}$, $S_2 \to \frac{E\alpha_2\Pi}{\mu Q}$ as $t \to \infty$, implying the global convergence of the solution of (16) in Ω_1. Hence $\mathbf{Y}^0$ is globally asymptotically stable. Thus, condition (H_1) is satisfied.

Next, we show that $G(\mathbf{Y},\mathbf{Z})$ satisfies the two conditions given in (H_2). It is clear that $G(\mathbf{Y},\mathbf{0}) = \mathbf{0}$. Let

$$C = D_Z G(\mathbf{Y}^0,\mathbf{0}) = \begin{pmatrix} \frac{\beta_1 S^0 + k_1\beta_1 S_1^0 + k_2\beta_1 S_2^0}{S^0 + S_1^0 + S_2^0} - K & \frac{\beta_2 S^0 + k_1\beta_2 S_1^0 + k_2\beta_2 S_2^0}{S^0 + S_1^0 + S_2^0} & 0 & 0 \\ \sigma_1 & -L & 0 & 0 \\ 0 & \delta & -M & 0 \\ 0 & \sigma_2 & \sigma_3 & -W \end{pmatrix}.$$

Now consider $G(\mathbf{Y},\mathbf{Z}) = C\mathbf{Z} - \hat{G}(\mathbf{Y},\mathbf{Z})$,

$$G(\mathbf{Y},\mathbf{Z}) = \begin{pmatrix} \beta S + k_1\beta S_1 + k_2\beta S_2 - KI \\ \sigma_1 I - LP \\ \delta P - MT \\ \sigma_2 P + \sigma_3 T - WA \end{pmatrix}.$$

Since all off-diagonal entries of matrix C are nonnegative, then C is an M-matrix. Then $G(\mathbf{Y},\mathbf{Z}) = C\mathbf{Z} - \hat{G}(\mathbf{Y},\mathbf{Z})$, where

$$C\mathbf{Z} = \begin{pmatrix} \left(\frac{\beta_1 S^0 + k_1\beta_1 S_1^0 + k_2\beta_1 S_2^0}{S^0 + S_1^0 + S_2^0} - K\right)I + \frac{\beta_2 S^0 + k_1\beta_2 S_1^0 + k_2\beta_2 S_2^0}{S^0 + S_1^0 + S_2^0} P \\ \sigma_1 I - LP \\ \delta P - MT \\ \sigma_2 P + \sigma_3 T - WA \end{pmatrix},$$

$$\hat{G}(\mathbf{Y},\mathbf{Z}) = \begin{pmatrix} \frac{(\beta_1 I + \beta_2 P)(S^0 + k_1 S_1^0 + k_2 S_2^0)}{S^0 + S_1^0 + S_2^0}\left(1 - \frac{S^0 + S_1^0 + S_2^0}{S^0 + k_1 S_1^0 + k_2 S_2^0}\frac{S + k_1 S_1 + k_2 S_2}{N}\right) \\ 0 \\ 0 \\ 0 \end{pmatrix}.$$

In the region Ω_1, $S \le S^0$, $S_1 \le S_1^0$, $S_2 \le S_2^0$ and hence we have $S + k_1 S_1 + k_2 S_2 \le S^0 + k_1 S_1^0 + k_2 S_2^0$ for $0 < k_i < 1$ $(i = 1, 2)$. Since total population is bounded by $N \le \frac{\Pi}{\mu}$ and $S^0 + S_1^0 + S_2^0 = \frac{\Pi}{\mu}$ we have $N \le S^0 + S_1^0 + S_2^0$. Thus, $1 - \frac{S^0 + S_1^0 + S_2^0}{S^0 + k_1 S_1^0 + k_2 S_2^0}\frac{S + k_1 S_1 + k_2 S_2}{N} > 0$ therefore $\hat{G}(\mathbf{Y},\mathbf{Z}) \ge 0$. Thus, $G(\mathbf{Y},\mathbf{Z})$ satisfies the two conditions which imply that condition (H_2) is satisfied. This completes the proof. □

Existence of Endemic Equilibrium

The endemic equilibrium $E_1=\left(S^*,S_1^*,S_2^*,I^*,P^*,T^*,A^*\right)$ of system (1) is obtained by solving the following equilibrium conditions of the system (1). With at least I^*,P^*,T^*,A^* being different from zero, we obtain

$$S^*=\frac{\Pi}{\beta^*+Q},\ S_1^*=\frac{E\alpha_1\Pi}{(k_1\beta^*+\mu)(\beta^*+Q)},$$
$$S_2^*=\frac{E\alpha_2\Pi}{(k_2\beta^*+\mu)(\beta^*+Q)},$$
$$I^*=\frac{\beta^*\Pi}{K(\beta^*+Q)}+\frac{k_1\beta^*E\alpha_1\Pi}{K(k_1\beta^*+\mu)(\beta^*+Q)}+\frac{k_2\beta^*E\alpha_2\Pi}{K(k_2\beta^*+\mu)(\beta^*+Q)},$$
$$P^*=w_1I^*,\ T^*=w_2I^*,\ P^*=w_3I^*, \tag{17}$$

where $w_1=\frac{\sigma_1}{L}$, $w_2=\frac{\delta\sigma_1}{LM}$, $w_3=\frac{\sigma_2w_1+\sigma_3w_2}{W}$,

$$\beta^*=\frac{\beta_1I^*+\beta_2P^*}{S^*+S_1^*+S_2^*+I^*+P^*+T^*}. \tag{18}$$

Substituting (17) into (18), we obtain

$$\beta^*\left(h_3\beta^{*3}+h_2\beta^{*2}+h_1\beta^*+h_0\right)=0, \tag{19}$$

where

$$\begin{aligned}
h_3&=k_1k_2(1+w_1+w_2+w_3),\\
h_2&=\left[k_1k_2E(\alpha_1+\alpha_2)+\mu(k_1+k_2)\right](1+w_1+w_2+w_3)\\
&\quad-k_1k_2(\beta_1+\beta_2w_1)+Kk_1k_2,\\
h_1&=-(\beta_1+\beta_2w_1)\left[k_1k_2E(\alpha_1+\alpha_2)+\mu(k_1+k_2)\right]\\
&\quad+\mu B(1+w_1+w_2+w_3)\\
&\quad+K\left[k_2E\alpha_1+k_1E\alpha_2+\mu(k_1+k_2)\right],\\
h_0&=KQ\mu\left(1-R_0\right).
\end{aligned}$$

We observe that $h_3>0$ and $h_0<0$ when $R_0>0$. From polynomials (19), one solution is $\beta^*=0$ related to the disease-free equilibrium. Another solution is the roots of a cubic polynomial

$$h_3\beta^{*3}+h_2\beta^{*2}+h_1\beta^*+h_0=0, \tag{20}$$

which is related to situations where HIV disease exists. Using Cardan's formula as in [36], the cubic equation (20) has solutions one positive real root and two complex conjugates roots (see details in Appendix)

$$\begin{aligned}
\beta_1^*&=u+v-\frac{h_2}{3h_3},\\
\beta_2^*&=-\frac{u+v}{2}-\frac{h_2}{3h_3}+\frac{i\sqrt{3}}{2}(u-v),\\
\beta_3^*&=-\frac{u+v}{2}-\frac{h_2}{3h_3}-\frac{i\sqrt{3}}{2}(u-v),
\end{aligned} \tag{21}$$

where $u=\sqrt[3]{r+\sqrt{r^2+q^3}}$, $u=\sqrt[3]{r-\sqrt{r^2+q^3}}$, and $q=\frac{3h_3h_1-h_2^2}{9h_3^2}$, $r=\frac{9h_3h_2h_1-27h_3^2h_0-2h_2^2}{54h_3^2}$.

Components of the endemic equilibrium point E_1 can be obtained by substituting the value of $\beta^*=\beta_1^*$ into the steady-state expression for each state in (17).

This result is summarized in the following lemma.

Lemma 3.2. *The system (1) has a unique endemic equilibrium E_1 whenever $R_0>0$ with $\beta^*=\beta_1^*$ are the positive real roots of equation (20).*

Global Stability of Endemic Equilibrium

We will prove the global stability of the endemic equilibrium E_1. We investigated the global stability property of the endemic equilibrium of the HIV system (1) for the case when there is no disease-induced death ($\gamma=0$). By letting

$$\Omega_0=\left\{(S,S_1,S_2,I,P,T,A)\in\mathbb{R}_+^7\,|\,I=P=T=A=0\right\}$$

and $R_{01}=R_{0|\gamma=0}$. Then we claim the following theorem.

Theorem 3.3. *The endemic equilibrium E_1 of the model system (1) with $\gamma=0$ is globally asymptotically stable in $\Omega\setminus\Omega_0$ whenever $R_{01}>0$.*

Proof. Let $R_{01}>1$, then the endemic equilibrium of system (1) exists. To study the endemic equilibrium is globally asymptotically stable, we define the Lyapunov function *V*, using the ideas in [37]:

$$\begin{aligned}
V(t)=\frac{1}{2}\Big[&\left(S-S^*\right)+\left(S_1-S_1^*\right)+\left(S_2-S_2^*\right)+\left(I-I^*\right)\\
&+\left(P-P^*\right)+\left(T-T^*\right)+\left(A-A^*\right)\Big]^2.
\end{aligned} \tag{22}$$

Clearly, *V* is positive definite. We have

$$\begin{aligned}
\frac{dV}{dt}=\Big[&\left(S-S^*\right)+\left(S_1-S_1^*\right)+\left(S_2-S_2^*\right)+\left(I-I^*\right)\\
&+\left(P-P^*\right)+\left(T-T^*\right)+\left(A-A^*\right)\Big]\frac{dN}{dt}.
\end{aligned}$$

Since $S^*+S_1^*+S_2^*+I^*+P^*+T^*+A^*\le\frac{\Pi}{\mu}$ and $\frac{dN}{dt}=\Pi-\mu N(t)$, it follows that

$$\begin{aligned}
\frac{dV}{dt}&=\Big[(S,S_1,S_2,I,P,T,A)-\left(S^*,S_1^*,S_2^*,I^*,P^*,T^*,A^*\right)\Big]\frac{dN}{dt}\\
&\le\left(N(t)-\frac{\Pi}{\mu}\right)\left(\Pi-\mu N(t)\right)=\frac{1}{\mu}\left(\mu N(t)-\Pi\right)\left(\Pi-\mu N(t)\right)\\
&=-\frac{1}{\mu}\left(\Pi-\mu N(t)\right)^2.
\end{aligned}$$

It follows that, that $\frac{dV}{dt} \le 0$ for $R_{01} > 0$ and $\frac{dV}{dt} = 0$ if and only if $S = S^*, S_1 = S_1^*, S_2 = S_2^*, I = I^*, P = P^*, T = T^*$ and $A = A^*$. Hence, V is a Lyapunov function on $\Omega \setminus \Omega_0$. Thus, $S \to S^*, S_1 \to S_1^*, S_2 \to S_2^*, I \to I^*, P \to P^*, T \to T^*$ and $A \to A^*$ as $t \to \infty$. Hence, V is a Lyapunov function on $\Omega \setminus \Omega_0$ and the largest compact invariant set in $\left\{(S, S_1, S_2, I, P, T, A) \in \Omega \mid \frac{dV}{dt} = 0\right\}$ is the singleton set $\{E_1\}$. Thus, by LaSalle's invariance principle [38], [39], the endemic equilibrium E_1 is globally asymptotically stable (GAS) in $\Omega \setminus \Omega_0$ when $R_{01} > 1$. □

B. Sensitivity Analysis of the Basic Reproduction Number

The sensitivity analysis of the basic reproduction number is used to determine important parameters in the model that has a high transmission influence on HIV/AIDS. Initial disease transmission is directly related to the basic reproduction number R_0. This sensitivity analysis was done by calculating the sensitivity index of R_0 to the parameters in the model by using the approach of [40]. In performing the system analysis, we apply the method called normalized forward sensitivity index of a variable to a parameter, which is a ratio of the relative change in the variable to the relative change in the parameter. The sensitivity index of the basic reproduction number R_0, that depends differentially on a parameter, say θ, is defined as

$$\Upsilon_\theta^{R_0} = \frac{\partial R_0}{\partial \theta}\frac{\theta}{R_0}. \tag{23}$$

The basic reproduction number (R_0) of the system (1) depends on eleven parameters, namely, $\beta_1, \beta_2, \mu, \delta, \psi_1, \psi_2, E, \sigma_1, \sigma_2, \alpha_1,$ and α_2. For example, the sensitivity index of R_0 related to parameter $\beta_1, \delta,$ and E are

$$\begin{aligned}
\Upsilon_{\beta_1}^{R_0} &= \frac{\partial R_0}{\partial \beta_1}\frac{\beta_1}{R_0} = \frac{\beta_1 L}{\beta_1 L + \beta_2 \sigma_1},\\
\Upsilon_{\delta}^{R_0} &= \frac{\partial R_0}{\partial \delta}\frac{\delta}{R_0} = -\frac{\beta_2 \sigma_1 L}{L(\beta_1 L + \beta_2 \sigma_1)},\\
\Upsilon_{E}^{R_0} &= \frac{\partial R_0}{\partial E}\frac{E}{R_0} = -\left(\frac{B(\alpha_1+\alpha_2) - Q(k_1\alpha_1 + k_2\alpha_2)}{BQ}\right).
\end{aligned}$$

Other indices $\Upsilon_{\beta_2}^{R_0}, \Upsilon_{\mu}^{R_0}, \Upsilon_{\psi_1}^{R_0}, \Upsilon_{\psi_2}^{R_0}, \Upsilon_{\sigma_1}^{R_0}, \Upsilon_{\sigma_2}^{R_0}, \Upsilon_{\alpha_1}^{R_0}$, and $\Upsilon_{\alpha_2}^{R_0}$ are obtained following the same method.

C. Optimal Control

The purpose of this section is to develop a deterministic system (1) into an optimal control problem with the controls being continuous in time. The information campaigns control that varies in time is represented by u_1 and the treatment rate control that varies in time is represented by u_2. The control functions u_1 and u_2 were defined at the closed interval $[0, T_f]$, where $0 \le u_i(t) \le 1, t \in [0, T_f], i = 1, 2$ and T_f is the final time of the controls. The corresponding state system for the system (1) is given by

$$\begin{aligned}
\frac{dS}{dt} &= \Pi - (\beta + u_1\alpha_1 + u_1\alpha_2)S - \mu S,\\
\frac{dS_1}{dt} &= u_1\alpha_1 S - (1-\psi_1)\beta S_1 - \mu S_1,\\
\frac{dS_2}{dt} &= u_1\alpha_2 S - (1-\psi_2)\beta S_2 - \mu S_2,\\
\frac{dI}{dt} &= \beta S + (1-\psi_1)\beta S_1 + (1-\psi_2)\beta S_2 - (\sigma_1 + \mu)I,\\
\frac{dP}{dt} &= \sigma_1 I - (u_2 + \sigma_2 + \mu)P,\\
\frac{dT}{dt} &= u_2 P - (\sigma_3 + \mu)T,\\
\frac{dA}{dt} &= \sigma_2 P + \sigma_3 T - (\gamma + \mu)A,
\end{aligned} \tag{24}$$

where

$$\beta = \frac{\beta_1 I + \beta_2 P}{S + S_1 + S_2 + I + P + T + A}.$$

We will now find an optimal strategy that minimizes the number of infected individuals (I and P) during a specific period and the cost of information campaigns (u_1) on susceptible individuals and treatment on pre-AIDS individuals (u_2) during the intervention period. The objective functional J is given

$$J(u_1, u_2) = \int_0^{T_f}\left[b_1 I(t) + b_2 P(t) + \frac{1}{2}\left(w_1 u_1^2(t) + w_2 u_2^2(t)\right)\right]dt, \tag{25}$$

where b_1 and b_2 represent positive weight constant for the associated infected individuals (I) and pre-AIDS individuals (P). The constants $w_1, w_2 \ge 0$, are weights of the relative costs of the controls associated with u_1 and u_2.

We seek an optimal control pair (u_1^*, u_2^*) such that

$$J(u_1^*, u_2^*) = \min\left\{J(u_1, u_2) \mid u_1, u_2 \in U\right\} \tag{26}$$

where $U = \left\{u = (u_1, u_2) \mid 0 \le u_i \le 1, i = 1, 2, \forall t \in [0, T_f]\right\}$ is the control set.

Next, we prove the existence of optimal control for the model system (24).

Existence of an Optimal Control Pair

Pontryagin's maximum principle converts the state system (24) with the objective functional (25) and (26) into a problem of minimizing pointwise a Hamiltonian H with respect to u_1 and u_2. The Lagrangian of the optimal control problem is given by

$$L = b_1 I(t) + b_2 P(t) + \frac{1}{2}\left(w_1 u_1^2(t) + w_2 u_2^2(t)\right) \tag{27}$$

and the Hamiltonian H is defined as follows

$$\begin{aligned} H = {} & b_1 I + b_2 P + \frac{1}{2}\left(w_1 u_1^2 + w_2 u_2^2\right) \\ & + \lambda_1\left(\Pi - \beta S - (u_1\alpha_1 + u_1\alpha_2 + \mu)S\right) \\ & + \lambda_2\left(u_1\alpha_1 S - k_1\beta S_1 - \mu S_1\right) \\ & + \lambda_3\left(u_1\alpha_2 S - k_2\beta S_2 - \mu S_2\right) \\ & + \lambda_4\left(\beta S + k_1\beta S_1 + k_2\beta S_2 - (\sigma_1 + \mu)I\right) \\ & + \lambda_5\left(\sigma_1 I - (u_2 + \sigma_2 + \mu)P\right) \\ & + \lambda_6\left(u_2 P - (\sigma_3 + \mu)T\right) \\ & + \lambda_7\left(\sigma_2 P + \sigma_3 T - (\gamma + \mu)A\right). \end{aligned} \tag{28}$$

Next, we examine the sufficient conditions for the existence of a solution to the optimal control for the model system (25).

Theorem 3.4. *For the model system (24) with control measure and initial conditions at* $t = 0$, *there exists an optimal control pair* $(u_1^*, u_2^*) \in \Omega$ *with a corresponding solution* $(S^*, S_1^*, S_2^*, I^*, P^*, T^*, A^*)$, *that minimizes* $J(u_1, u_2)$ over *U*.

Proof. The existence of an optimal control pair (u_1^*, u_2^*), we need to show the following conditions are satisfied based on [41].

(i) The set of controls and corresponding state variable is nonempty.

(ii) The control set *U* is closed and convex.

(iii) The right-hand side of the state system (24) is bounded by a linear function in the state and control variables.

(iv) The integrand *L* in (27) is convex on U and additionally satisfies,

$$L \geq \xi_1 \left\|(u_1, u_2)\right\|^{\varepsilon} - \xi_2,$$

where $\xi_1 > 0, \xi_2 > 0, \text{and } \varepsilon > 1$.

All the model states $(S, S_1, S_2, I, P, T, A) \in \Omega$ are bounded below and above, it can be said that any solutions to the state equations are bounded. The state system of Lipschitz property concerning the state variables is satisfied since the state solutions are bounded. Thus, the condition (i) is satisfied. By the definition of the control set, *U* is closed and convex such that the condition (ii) is satisfied. From the state equation system (24), the state equations can be expressed as a linear function of the controls u_1 and u_2 with coefficients depending on state variables**.** Thus, condition (iii) is satisfied. The integrand *L* into *J* defined by (27) is convex on Ω since it is a quadratic function of (u_1, u_2) on *U*. Then, bounded on *L* is shown as follows:

$$\begin{aligned} L &= b_1 I + b_2 P + \tfrac{1}{2}(w_1 u_1^2 + w_2 u_2^2) \\ &\geq \tfrac{1}{2}(w_1 u_1^2 + w_2 u_2^2) \text{ since } b_i > 0,\ i = 1, 2 \\ &\geq \tfrac{1}{2}(w_1 u_1^2 + w_2 u_2^2) - w_1 \text{ since } w_1 u_1^2 - w_1 \leq 0 \\ &\geq \min(\tfrac{1}{2} w_1, \tfrac{1}{2} w_2)(u_1^2 + u_2^2) - w_1 \\ &\geq \omega \left\|(u_1, u_2)\right\|^2 - w_1, \end{aligned}$$

where $\xi_1 = \omega = \min(\frac{1}{2} w_1, \frac{1}{2} w_2)$, $\xi_2 = w_1$, and $\varepsilon = 2$. Thus, condition (iv) also holds. The proof is completed. □

Characterization of the Optimal Control Pair

We characterized the optimal control pair $u^* = (u_1^*, u_2^*)$ of the system and the corresponding states $x^* = (S^*, S_1^*, S_2^*, I^*, P^*, T^*, A^*)$ with its control functions u_1 and u_2 with the objective functional (25). Using the Pontryagin's Maximum Principle [23], [24], we obtained the necessary conditions for the optimal controls.

Theorem 3.5. Given *an optimal control* $u^* = (u_1^*, u_2^*)$ *and solution* $x^* = (S^*, S_1^*, S_2^*, I^*, P^*, T^*, A^*)$ *that are corresponding optimal state variables of the control problem (24)-(26), then there exists an adjoint variable* λ_j, $j = 1, 2, 3, 4, 5, 6, 7$ *are associated by* S, S_1, S_2, I, P, T, A *satisfying the following equations:*

$$\begin{aligned} \frac{d\lambda_1}{dt} &= (\lambda_1 - \lambda_4)\left(\beta - \frac{\beta S}{N}\right) + (\lambda_1 - \lambda_2)u_1\alpha_1 + (\lambda_1 - \lambda_3)u_1\alpha_2 \\ &\quad + (\lambda_4 - \lambda_2)\frac{k_1\beta S_1}{N} + (\lambda_4 - \lambda_3)\frac{k_1\beta S_2}{N} + \lambda_1\mu, \\ \frac{d\lambda_2}{dt} &= (\lambda_4 - \lambda_1)\frac{\beta S}{N} + (\lambda_2 - \lambda_4)\left(k_1\beta - \frac{k_1\beta S_1}{N}\right) \\ &\quad + (\lambda_4 - \lambda_3)\frac{k_2\beta S_2}{N} + \lambda_2\mu, \\ \frac{d\lambda_3}{dt} &= (\lambda_4 - \lambda_1)\frac{\beta S}{N} + (\lambda_4 - \lambda_2)\frac{k_1\beta S_1}{N} \\ &\quad + (\lambda_3 - \lambda_4)\left(k_2\beta - \frac{k_2\beta S_2}{N}\right) + \lambda_3\mu, \\ \frac{d\lambda_4}{dt} &= -b_1 + (\lambda_1 - \lambda_4)\frac{(\beta_1 - \beta)S}{N} + (\lambda_2 - \lambda_4)\frac{k_1(\beta_1 - \beta)S_1}{N} \\ &\quad + (\lambda_3 - \lambda_4)\frac{k_2(\beta_1 - \beta)S_2}{N} + \lambda_4(\sigma_1 + \mu) - \lambda_5\sigma_1, \\ \frac{d\lambda_5}{dt} &= -b_2 + (\lambda_1 - \lambda_4)\frac{(\beta_2 - \beta)S}{N} + (\lambda_2 - \lambda_4)\frac{k_1(\beta_2 - \beta)S_1}{N} \\ &\quad + (\lambda_3 - \lambda_4)\frac{k_2(\beta_2 - \beta)S_2}{N} + \lambda_5(u_2 + \sigma_2 + \mu) - \lambda_6 u_2 \\ &\quad - \lambda_7\sigma_2, \\ \frac{d\lambda_6}{dt} &= (\lambda_4 - \lambda_1)\frac{\beta S}{N} + (\lambda_4 - \lambda_2)\frac{k_1\beta S_1}{N} \\ &\quad + (\lambda_4 - \lambda_3)\frac{k_2\beta S_2}{N} + \lambda_6(\sigma_3 + \mu) - \lambda_7\sigma_3, \\ \frac{d\lambda_7}{dt} &= (\lambda_4 - \lambda_1)\frac{\beta S}{N} + (\lambda_4 - \lambda_2)\frac{k_1\beta S_1}{N} \\ &\quad + (\lambda_4 - \lambda_3)\frac{k_2\beta S_2}{N} + \lambda_7(\gamma + \mu), \end{aligned} \tag{29}$$

with transversality conditions

$$\lambda_i(T_f) = 0 \text{ for } i = 1, 2, 3, 4, 5, 6, 7. \tag{30}$$

Furthermore, use boundary conditions for $0 \leq u_i \leq 1$, $i = 1, 2$ *to obtain* $u^* = (u_1^*, u_2^*)$,

$$\begin{aligned} u_1^* &= \min\left\{\max\left(0, \frac{\left[(\lambda_1 - \lambda_2)\alpha_1 + (\lambda_1 - \lambda_3)\alpha_2\right]S^*}{w_1}\right), 1\right\}, \\ u_2^* &= \min\left\{\max\left(0, \frac{(\lambda_5 - \lambda_6)P^*}{w_2}\right), 1\right\}. \end{aligned} \tag{31}$$

Proof. The adjoint equations in (29) are obtained by differentiating the Hamiltonian function H in (28) with respect to each of the state variables:

$$\frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial S}, \quad \frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial S_1}, \quad \frac{d\lambda_3}{dt} = -\frac{\partial H}{\partial S_2},$$
$$\frac{d\lambda_4}{dt} = -\frac{\partial H}{\partial I}, \quad \frac{d\lambda_5}{dt} = -\frac{\partial H}{\partial P}, \quad \frac{d\lambda_6}{dt} = -\frac{\partial H}{\partial T},$$
$$\frac{d\lambda_7}{dt} = -\frac{\partial H}{\partial A},$$

with $\lambda_i(T_f) = 0$, for $i = 1,2,3,4,5,6,7$.

Furthermore, to obtain the expression for optimal control u_1^* and u_2^*, we differentiate (29) with respect to u_1 and u_2 on the set U, respectively to get the following optimality equations:

$$\frac{\partial H}{\partial u_1} = w_1 u_1 - \left[(\lambda_1 - \lambda_2)\alpha_1 + (\lambda_1 - \lambda_3)\alpha_2\right] S \text{ at } u_1 = u_1^*,$$
$$\frac{\partial H}{\partial u_2} = w_2 u_2 - (\lambda_5 - \lambda_6) P \text{ at } u_2 = u_2^*.$$

Hence, solving for u_1^* and u_2^* at the interior sets gives

$$u_1^* = \hat{u}_1 = \frac{\left[(\lambda_1 - \lambda_2)\alpha_1 - (\lambda_1 - \lambda_3)\alpha_2\right] S^*}{w_1},$$
$$u_2^* = \hat{u}_2 = \frac{(\lambda_5 - \lambda_6) P^*}{w_2}. \tag{32}$$

Let us consider the control bound for $0 \le u_i^* \le 1$, $i = 1,2$. By using the bounds on the control u_1^* and u_2^* we conclude that

$$u_1^* = \begin{cases} 0, & \text{if} \quad \hat{u}_1 \le 0, \\ \hat{u}_1, & \text{if} \quad 0 < \hat{u}_1 < 1, \\ 1, & \text{if} \quad \hat{u}_1 \ge 1, \end{cases} \quad u_2^* = \begin{cases} 0, & \text{if} \quad \hat{u}_2 \le 0, \\ \hat{u}_2, & \text{if} \quad 0 < \hat{u}_2 < 1, \\ 1, & \text{if} \quad \hat{u}_2 \ge 1. \end{cases}$$

Hence, optimal control u_1^* and u_2^* are formulated as follows:

$$u_1^* = \min\left\{\max\left(0, \frac{\left[(\lambda_1 - \lambda_2)\alpha_1 + (\lambda_1 - \lambda_3)\alpha_2\right] S^*}{w_1}\right), 1\right\},$$
$$u_2^* = \min\left\{\max\left(0, \frac{(\lambda_5 - \lambda_6) P^*}{w_1}\right), 1\right\}.$$

Consequently, the characterization of the optimal control as in equation (31) can be derived. □

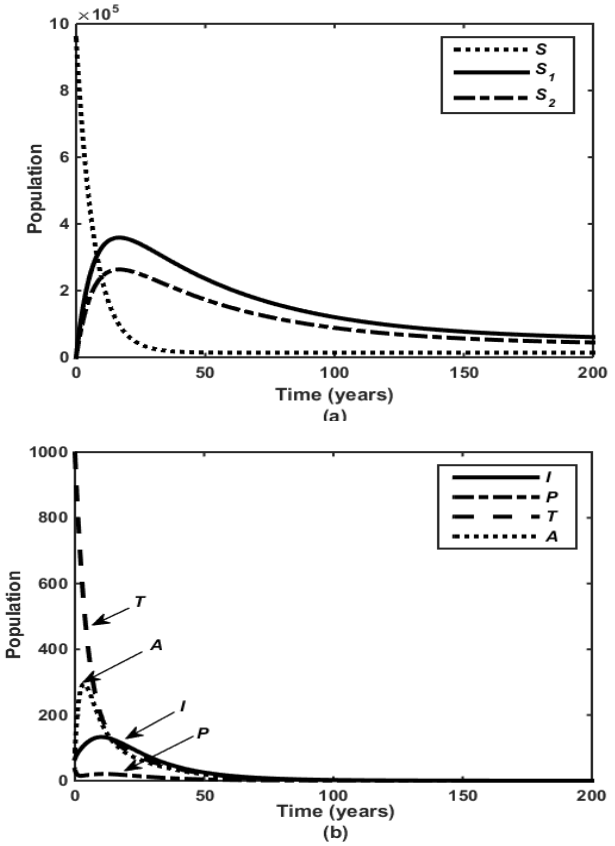

Fig. 1. Simulation of the system (1) showing the stable behavior of the model at E_0 (a) the trend of S, S_1, S_2; (b) the trend of I, P, T, A.

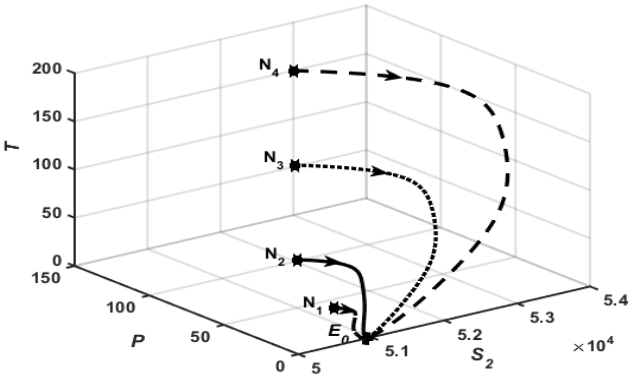

Fig. 2. 3-dimension diagram of the population showing global stability of E_0 with various initial values.

IV. NUMERICAL SIMULATIONS

In this section, we first present the numerical simulations of the autonomous system (1). Next, we investigate numerically the optimal control strategies that are designed and presented using one or both control strategies to find the optimal values of the objective functional one by one.

A. Stability of Equilibria

In this subsection, we illustrate the analytical results by carrying out numerical simulations of the model system (1) using the set of parameter values given in Table II. The parameters that were not available in the literature were assumed.

Next, we illustrate the invariance properties of the system (1). Precisely, for varying initial conditions the model solutions converge to either the disease-free equilibrium or the endemic equilibrium point.

Case 1: $R_0 < 1$

First, we choose $E = 0.8$ and $\delta = 0.8$. The numerical simulation of the system (1) shows that the disease-free equilibrium (DFE) point is globally stable for some other parameter values in Table II. The corresponding basic reproduction number is equal to $R_0 = 0.85988 < 1$. Fig. 1 illustrates stable behavior of the model at E_0 proved in Theorem 3.1. Theorem 3.2 is numerically illustrated in Fig. 2, which shows globally stable behavior of the model at E_0. This figure shows the dynamics of the population of educated susceptible that have C behavior (S_2), pre-AIDS individuals (P), and treated individuals (T) in 3-dimension phase portrait of the model system (1). In this figure, all solution trajectories converge to E_0 for four different initial

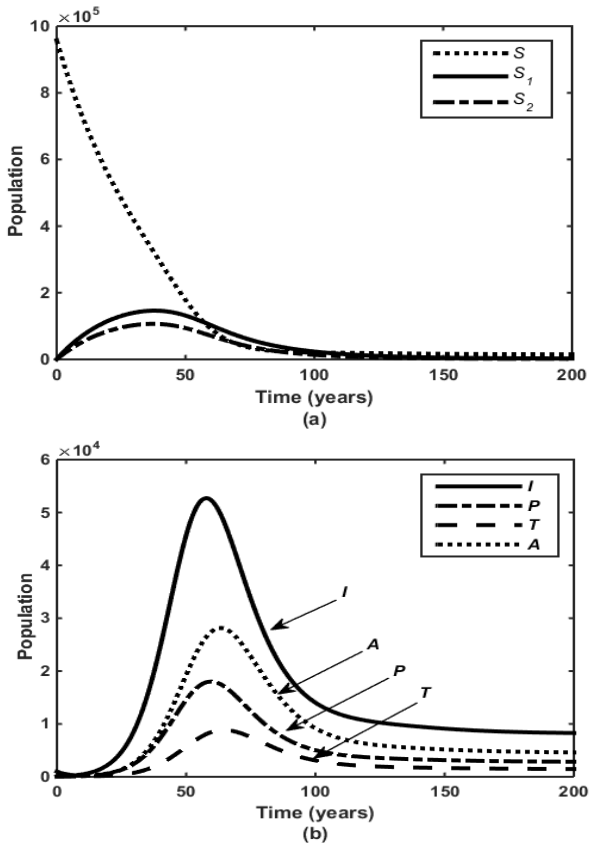

Fig. 3. Simulation of the system (1) showing the stable behavior of the model at E_1 (a) the trend of S, S_1, S_2; (b) the trend of I, P, T, A.

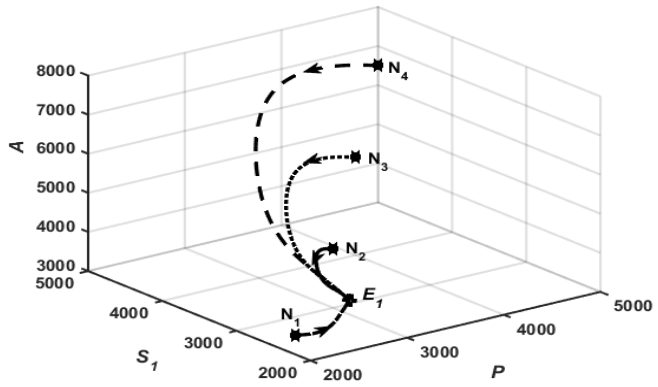

Fig. 4. 3-dimension diagram of the population showing global stability of E_1 with various initial values

TABLE II
THE DESCRIPTIONS OF PARAMETERS OF MODEL

Parameter	Value (year^{-1})	References
Π	2000	Assumed
β_1, β_2	0.3, 0.45	Assumed
$\sigma_1, \sigma_2, \sigma_3$	0.198, 0.4621, 0.18	[29], [29], [11]
ψ_1, ψ_2	0.6, 0.53	Assumed
α_1, α_2	0.091, 0.067	Assumed
E	0.1, 0.8	Assumed
δ	0.1, 0.8	Assumed
μ	0.0196	[15]
γ	0.33	[12]

populations:

$$N_1(1.3\times10^4,\ 5.9\times10^4,\ 3.7\times10^4,\ 20,\ 20,\ 20,\ 20),$$
$$N_2(1.4\times10^4,\ 5.1\times10^4,\ 3.8\times10^4,\ 50,\ 50,\ 50,\ 50),$$
$$N_3(1.5\times10^4,\ 5.2\times10^4,\ 3.9\times10^4,\ 100,\ 100,\ 100,\ 100),$$
$$N_4(1.6\times10^4,\ 5.3\times10^4,\ 4\times10^4,\ 150,\ 150,\ 150,\ 150).$$

Case 2: $R_0 > 1$

Second, we choose $E = 0.1$ and $\delta = 0.1$. The numerical simulation of the system (1) shows that the disease-free equilibrium (DFE) point is globally stable for some other parameter values in Table II. The corresponding basic reproduction number is equal to $R_0 = 1.553162 < 1$. We have the cubic equation (22) with $h_3 = 0.385143$, $h_2 = -0.003269$, $h_1 = -0.007896$, and $h_0 = -0.000083$. Using Cardan's formula, this cubic equation has the positive real roots $\beta^* = 0.101203$. Hence, the endemic equilibrium E_1 is given by $E_1 = (14640.9, 2217.5, 1460.5, 7541.1, 2566.9, 1286, 4054.9)$. The numerical result illustrated in Fig.3 confirms that system (1) has only one unique positive endemic equilibrium when $R_0 > 1$. This implies that HIV infection will persist in the population. Theorem 3.3 is numerically illustrated in Fig. 4, which shows the dynamics of the population of pre-AIDS (P), educated susceptible that have AB behavior (S_1), and AIDS individuals (A) in drawing of stability diagram in three-dimension. This Figure shows that all solution trajectories converge to the endemic equilibrium E_1 for four different initial populations:

$$N_1(1.4\times10^4, 2.2\times10^3, 1.4\times10^3, 7\times10^3, 2\times10^3, 1\times10^3, 3.5\times10^3),$$
$$N_2(1.5\times10^4, 3\times10^3, 2\times10^3, 8\times10^3, 3\times10^3, 2\times10^3, 4.5\times10^3),$$
$$N_3(1.6\times10^4,\ 4\times10^3, 3\times10^3, 9\times10^3, 4\times10^3, 3\times10^3, 5.5\times10^3),$$
$$N_4(1.7\times10^4,\ 5\times10^3, 4\times10^3, 1\times10^4, 5\times10^3, 4\times10^3, 6.5\times10^3).$$

B. Sensitivity Index

The basic reproduction number R_0 of system (1) depends on eleven parameters, namely, $\beta_1, \beta_2, \sigma_1, \sigma_2, \delta, \psi_1, \psi_2, \alpha_1, \alpha_2, E, \mu$. From an explicit formula for R_0 in (24), we derived an analytical expression for the sensitivity of R_0 to each of the eleven different the parameters involved in R_0. Thus, using parameter values in Table II, we obtained the results presented in Table III. Table III shows that the

sensitivity indices of β_1, β_2, and μ have a positive sign and the remaining $(\sigma_1, \sigma_2, \delta, \psi_1, \psi_2, \alpha_1, \alpha_2)$ have a negative sign. The parameters are arranged from the most sensitive parameter to the least. The most sensitive parameter is the effective contact rates (β_1). $\Upsilon_{\beta_1}^{R_0} = 0.6620$ implies that an increase (or a decrease) of the effective contact rates β_1 by 10% will be followed by an increase (or a decrease) in R_0 by 6.62%. Similarly, an increase (or a decrease) of the parameters β_2, μ by 10% will be followed by an increase (or a decrease) in R_0 by 3.379%, 0.876% respectively. On the other hand, $\Upsilon_{\sigma_1}^{R_0} = -0.57193$ indicates that an increase (or a decrease) of σ_1 by 10% will be followed by a decrease (or an increase) in R_0 by 5.7193%. In the same way, an increase (or a decrease) of the parameters $\sigma_2, \psi_1, E, \psi_2, \alpha_1, \alpha_2, \delta$ indicates a decrease (or an increase) in R_0 by 2.685%, 2.069%, 1.891%, 1.191%, 0.699%, 0.581%, respectively.

Thus, the sensitivity analysis of the basic reproductive rate model (1) provides excellent insights into the dynamics of disease transmission. In particular, it assists public health authorities in implementing appropriate intervention strategies to prevent and control the spread of HIV/AIDS such as information campaigns and treatment. In from Fig. 5, we show the relationship between $R_0, E,$ and δ. As the values of E and δ increase at the time, the basic reproduction number decreases sharply. This shows that increasing the level of information campaigns and the rate of treatment has a significant effect in reducing the numbers of I, P, A.

C. Numerical Simulation of the Optimal

In this subsection, we discuss the numerical results of the system (24) to investigate the effect of the following itemized optimal control strategies on the spread of the disease in a population. Using the MATLAB software tools, this section focuses on demonstrating some numerical results of qualitative analysis and optimal control problem (24)-(26) through the forward-backwards Sweep method [24]. The state equations (24) are solved forward in time with the initial guess for the controls over the time interval

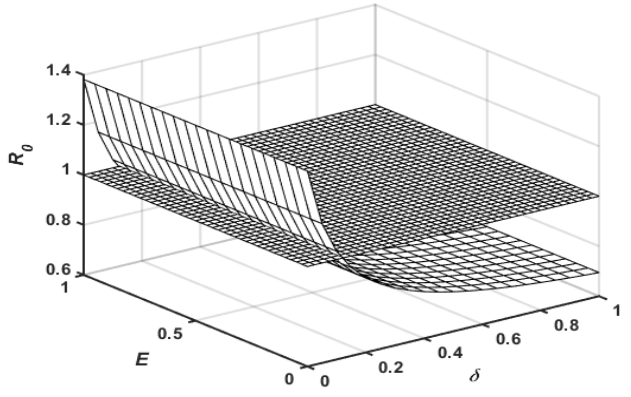

Fig. 5. The relationship among $R_0, E,$ and δ.

TABLE III
SENSITIVITY INDEX OF R_0

Parameters	Sensitivity Index
β_1	+0.6620
σ_1	-0.5719
β_2	+0.3379
σ_2	-0.2685
ψ_1	-0.2069
E	-0.1891
ψ_2	-0.1346
α_1	-0.1191
μ	+0.0876
α_2	-0.0699
δ	-0.0581

$[0, T_f]$ using the fourth order Runge-Kutta scheme. Using the transversality conditions (30), the adjoint equations (29) is solved by a backward fourth-order Runge-Kutta scheme using the current iteration solutions of the state equations. Then the controls are updated using a convex combination of the previous control values and the new control values (31). The process continues and iterations are stopped if the values of the solution of the state equations at the present iteration is very close to the previous iteration values [24]. Furthermore, in describing the control strategy the parameter values are used in [29] and weights at the end of the period ($T_f = 20$).

$$b_1 = 10, b_2 = 10, w_1 = 1, w_2 = 1000,$$

and the initial conditions as in [30],

$$S(0) = 957263, S_1(0) = 500, S_2(0) = 459,$$
$$I(0) = 67, P(0) = 34, T(0) = 996, A(0) = 89.$$

The parameter values given in Table II used in the simulations (Case 2 with $R_0 = 1.553162 > 1$). Thus, to investigate the effect of the different optimal control strategies on the spread of HIV/AIDS in population, we will investigate and compare numerical results from the simulation using the following scenario:

Strategy 1: Using Information Campaigns and Treatment

In this strategy, we used controls of information campaigns (u_1) and treatment (u_2) to optimize the objective function (25). Following Figs. 6(a)-6(c), this strategy results in a significant decrease in the numbers of asymptomatic infected individuals, pre-AIDS individuals, and AIDS individuals when compared to cases without controls. The numbers of asymptomatic infected individuals, pre-AIDS individuals, and AIDS individuals with controls and without controls at the end of the period are $I^* = 78.87, P^* = 23.36,$ $A^* = 72.84$ and $I = 4986, P = 1432, A = 1206$ respectively. Fig. 6(d) represents the control profile for the implementation of Strategy 1. The control u_1 (black solid line in Fig. 6(d)) is at the upper bound for almost the whole period (19.97 years) before dropping to the lower bound and the control u_2 (black dotted line in Fig. 6(d)) is at the upper

bound for 15.55 years before dropping to the lower bound at the end of the control period.

Strategy 2: Using Treatment Only

In Strategy 2, we used the treatment control (u_2) to optimize the objective functional *J*, whereas we set the control u_1 to zero. There is a significant reduction in the numbers of asymptomatic infected individuals, pre-AIDS individuals, and AIDS individuals compared to cases without control (Figs. 7(a)-7(c)). The numbers of asymptomatic infected individuals, pre-AIDS individuals, and AIDS individuals with control at the end of the period are $I^* = 1147, P^* = 184, A^* = 310.6$ and these numbers become lower when compared with the optimal control strategy in Figs. 6(a)-6(c). The control profile u_2 (black dotted line in Fig. 7(d)) is at the upper bound for 18.8 years before dropping to the lower bound at the end of the period.

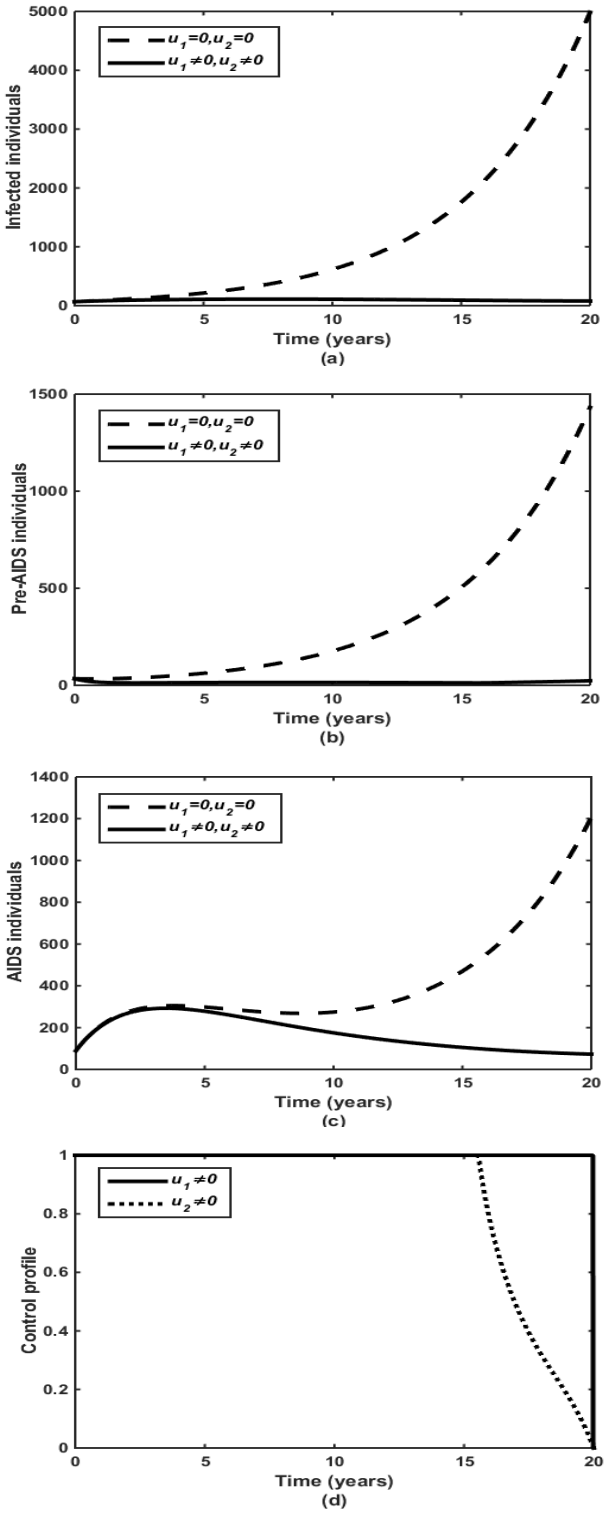

Fig. 6. Simulation results of the system (25) showing the effect of control of information campaigns: (a) infected individuals, (b) pre-AIDS individuals, (c) AIDS individuals, and (d) control profile of u_1 and u_2.

Strategy 3: Using Information Campaigns Only

The control of information campaigns (u_1) is used to optimize the objective function (25), whereas we set the control u_2 to zero. Figs. 8(a)-8(c) shows a significant reduction in the numbers of asymptomatic infected individuals, pre-AIDS individuals, and AIDS individuals when compared to cases without controls. The numbers of asymptomatic infected individuals, pre-AIDS individuals, and AIDS individuals with controls at the end of the period are $I^* = 246.5, P^* = 99.52, A^* = 146.1$. The reduction in the numbers of asymptomatic infected individuals, pre-AIDS individuals, and AIDS individuals was greater compared to the optimal control strategies in Figs. 7(a)-7(c), but this number is lower when compared to the optimal control strategy in Figs. 6(a)-6(c). The control profile of the control u_1 (black dotted line in Fig. 8(d)) is the same when compared to the strategy in Figure 6(d).

Based on the explanation above, it appears that the control strategies implemented either of the strategies considered have a significant effect in reducing the number of infected individuals.

D. Cost-effectiveness Analysis

It is important to determine the most cost-effective strategy of all the three strategies (1, 2, and 3) in Subsection C. To compare the differences between the costs and health outcomes of these three strategies competing for the same limited sources, we used the incremental cost-effectiveness ratio (ICER) [28], [30], [42]. The ICER is used to compare any two competing intervention strategies incrementally, one intervention should be compared with the next less-effective alternative. The ICER formula is as follows:

$$\text{ICER} = \frac{\text{Difference in total costs of strategies i and j}}{\text{Difference in total infection averted of strategies i and j}}.$$

The total cost for the implemented strategies in the given period is $\int_0^{T_f} [w_1 u_1^* S^* + w_2 u_2^* P^*] dt$, whereas the total number of infections averted is calculated from the difference between the total number of infected individuals without and with control.

Table IV summarizes the strategies ranked in increasing order of the total infection averted. Using the ICER formula, the ICER for Strategies 1, 2, and 3 controls measures shown in Table IV are calculated as follows.

$$\text{ICER}(2) = \frac{918,570}{25,134} = 36.5469,$$

$$\text{ICER}(3) = \frac{5,396,200 - 918,570}{29,089 - 25,134} = 1132.144,$$

$$\text{ICER}(1) = \frac{5,659,600 - 5,396,200}{32,774 - 29,089} = 71.479.$$

The comparison between Strategy 2 and Strategy 3 shows that ICER(2) is less than ICER(3). This means that Strategy 3 is strongly dominant over Strategy 2. In other words, Strategy 3 is more costly and less effective than Strategy 2. Therefore, Strategy 3 is excluded from the set of alternatives and ICER recomputed for Strategies 2 and 1 using a similar procedure, see Table V.

TABLE IV
TOTAL INFECTED AVERTED, TOTAL COST, AND ICER

Strategy	Total Infection Averted	Total Cost	ICER
Strategy 2	25,134	918,570	36.547
Strategy 3	29,089	5,396,200	1,132.144
Strategy 1	32,774	5,659,600	71.479

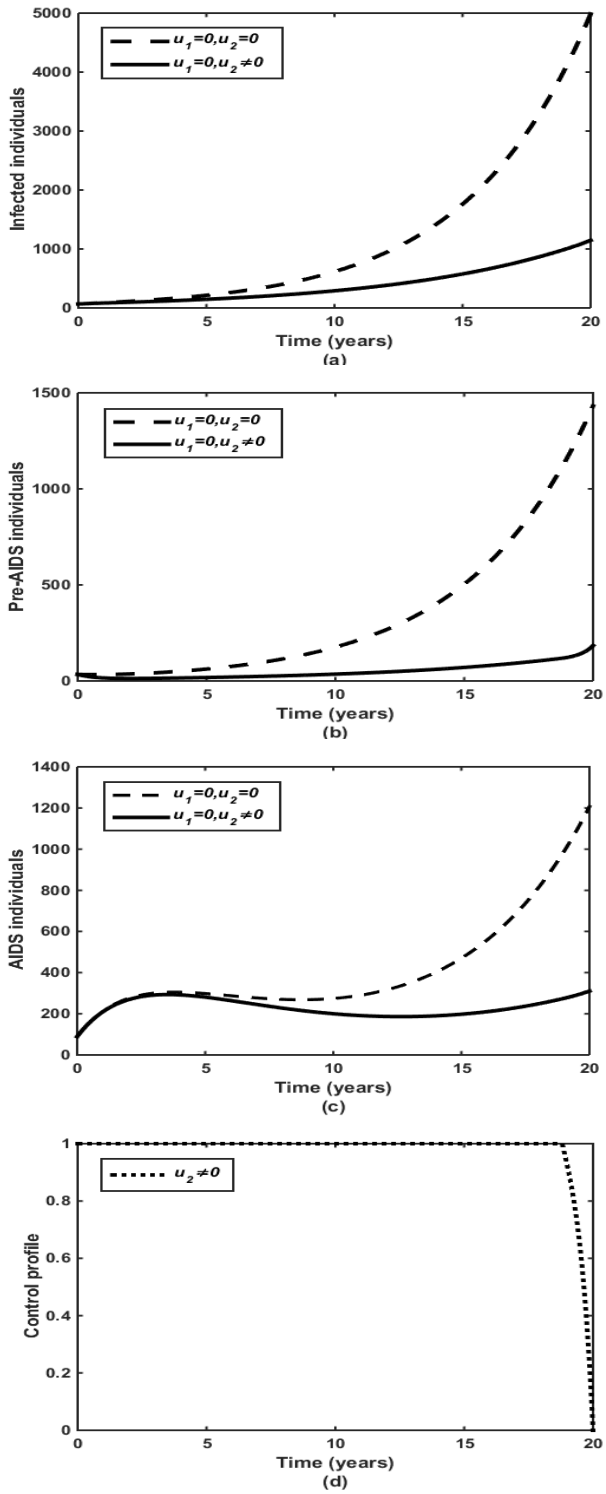

Fig. 7. Simulation results of the system (25) showing the effect of control of information campaigns: (a) infected individuals, (b) pre-AIDS individuals, (c) AIDS individuals, and (d) control profile of u_2.

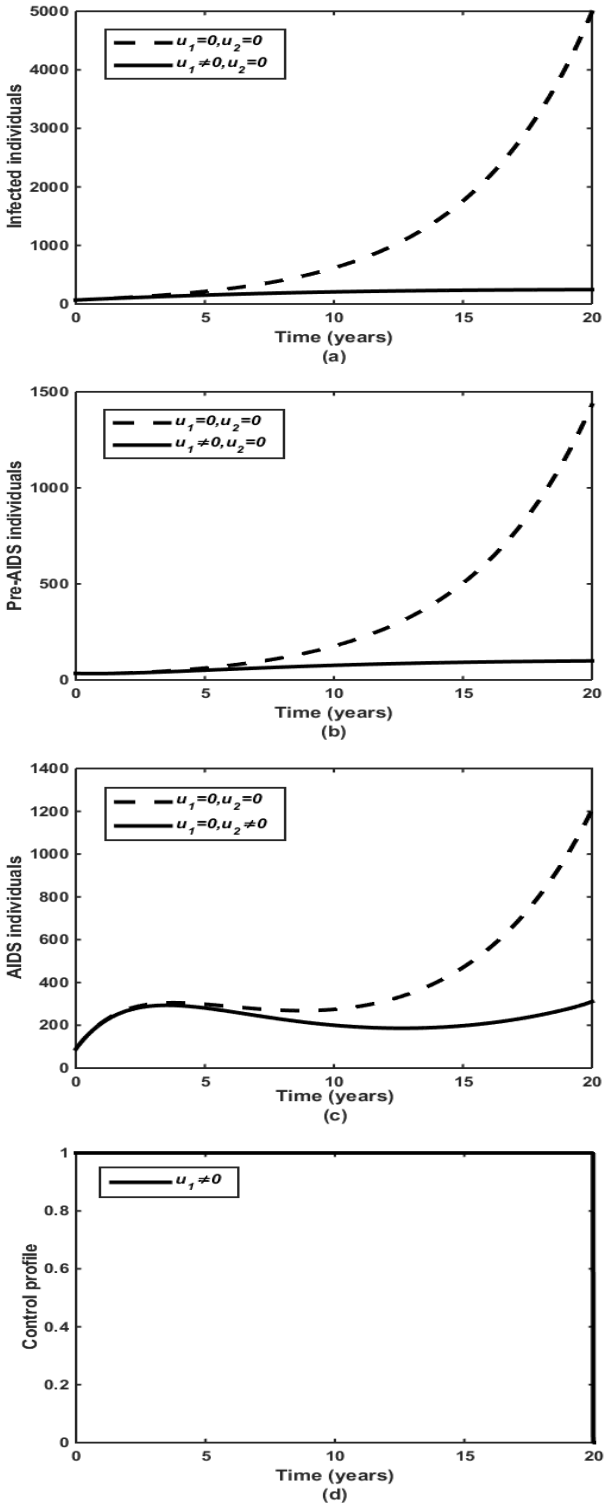

Fig. 8. Simulation results of the system (25) showing the effect of control of information campaigns: (a) infected individuals, (b) pre-AIDS individuals, (c) AIDS individuals, and (d) control profile of u_1.

The comparison between Strategy 2 and Strategy 1 shows that ICER(2) is less than ICER(1). This means that Strategy 1 is strongly dominant over Strategy 2. In other words, Strategy 1 is more costly and less effective than Strategy 2.

With these results, it can be concluded that Strategy 2 implementing treatment is the most cost-effective of all the strategies for the control of the HIV/AIDS consider in this work.

V. CONCLUSION

In this paper, we formulated and analyzed a mathematical model with the inclusion of two control strategies to study the effect of education campaigns of susceptible individuals and the treatment of pre-AIDS individuals on the transmission of HIV infection in a population. Using the theory of differential equations, the positivity and boundedness of solutions for the model is proved. By analyzing the model, we derive the basic reproduction number R_0 by using the next-generation matrix method. The existence of a disease-free equilibrium and an endemic equilibrium point is shown. The components of the endemic equilibrium can be obtained by substituting the positive real root of the cubic equations. The disease-free equilibrium point is globally asymptotically stable whenever the basic reproduction number is less than unity. Further, by using a Lyapunov function and LaSalle's invariant set theorem, the endemic equilibrium point is also globally asymptotically stable whenever the basic reproduction number greater than unity. The unique endemic equilibrium point of the model is shown to be globally asymptotically stable for a special case.

Numerical simulations have been performed to support the analysis results. The results of the sensitivity analysis of the basic reproduction number show that the most sensitive parameters of the model are the effective contact rates of susceptible individuals with asymptomatic infected individuals, followed by progression rate from asymptomatic infected into pre-AIDS and the effective contact rates of susceptible individuals with pre-AIDS infected individuals. Numerically, the optimal control analysis shows that control strategies have a significant effect in reducing the number of infected individuals in the population. By cost-effectiveness analysis, we conclude that Strategy 2 implementing treatment is the most cost-effective among the three strategies considered.

For future work. it will be interesting to include the effect of progression rate from asymptomatic infected into pre-AIDS as a control measure because it has a large sensitivity index.

APPENDIX

Theorem (*The Cardan's Formula*). *The cubic polynomial*

$$h_3\beta^{*3} + h_2\beta^{*2} + h_1\beta^{*} + h_0 = 0 \tag{A.1}$$

has solutions

TABLE V
TOTAL INFECTED AVERTED, TOTAL COST, AND ICER

Strategy	Total Infection Averted	Total Cost	ICER
Strategy 2	25,134	918,570	36.547
Strategy 1	32,774	5,659,600	620.554

$$\beta_1^* = u + v - \frac{h_2}{3h_3},$$
$$\beta_2^* = -\frac{u+v}{2} - \frac{h_2}{3h_3} + \frac{i\sqrt{3}}{2}(u-v),$$
$$\beta_3^* = -\frac{u+v}{2} - \frac{h_2}{3h_3} - \frac{i\sqrt{3}}{2}(u-v),$$

where

$$u = \sqrt[3]{r + \sqrt{r^2+q^3}},\ v = \sqrt[3]{r - \sqrt{r^2+q^3}},\ q = \frac{3h_3h_1 - h_2^2}{9h_3^2},$$
$$r = \frac{9h_3h_2h_1 - 27h_3^2h_0 - 2h_2^2}{54h_3^2}.$$

Proof. To depress the cubic polynomial (A.1), we substitute $\beta^* = y - \frac{h_2}{3h_3}$ and make it monic by dividing by h_3. We get

$$y^3 + \left(\frac{3h_3h_1 - h_2^2}{3h_3^2}\right)y + \left(\frac{2h_2^3 - 9h_3h_2h_1 + 27h_3^2h_0}{27h_3^3}\right) = 0.$$

Let $q = \frac{3h_3h_1 - h_2^2}{9h_3^2}$, $r = \frac{9h_3h_2h_1 - 27h_3^2h_0 - 2h_2^2}{54h_3^2}$, we obtain

$$y^3 + 3qy - 2r = 0. \tag{A.2}$$

Now, consider the identity $(u+v)^3 - 3uv(u+v) - (u^3+v^3) = 0.$ If we make it match with (A.2), we get the system of equations

$$y = u + v,\ uv = -q,\ u^3 + v^3 = 2r. \tag{A.3}$$

Then, we get

$$\begin{aligned} &(u+v)^3 + 3q(u+v) - 2r = 0 \\ \Leftrightarrow\ &u^3 + v^3 - 2r = 0 \\ \Leftrightarrow\ &u^3 - \frac{q^3}{u^3} - 2r = 0 \\ \Leftrightarrow\ &u^6 - 2ru^3 - q^3 = 0. \end{aligned}$$

The sixth order polynomial can be solved by using the quadratic formula, by taking $z = u^3$ and the equation reduces to

$$z^2 - 2rz - q^3 = 0. \tag{A.4}$$

Solving using the quadratic formula, we get the roots of (A.4)

$$z = \frac{2r \pm \sqrt{4r^2 + 4q^3}}{2} = r \pm \sqrt{r^2 + q^3}.$$

By substituting z back to u, we get $u^3 = r + \sqrt{r^2 + q^3}$.

Notice that the system of equation is symmetric in u and v, so the order we choose does not matter, and the value of y will the same. So

$$u = w^m \sqrt[3]{r + \sqrt{r^2 + q^3}}, \quad v = w^n \sqrt[3]{r - \sqrt{r^2 + q^3}},$$

where $0 \le m, n \le 2$ and w is any 3rd primitive root of unity ($w^3 = 1$ and $1 + w + w^2 = 0$). We see that then we have 9 combinations for the value $u + v$, but only 3 of them work. By looking at the second equation, we see that $m + n$ must be a multiple 0f 3, so $(m, n) = (0, 0), (1, 2), (2, 1)$ and our solutions are

$$y_1 = u + v,\ y_2 = uw + vw^2,\ y_3 = uw^2 + vw.$$

We choose $w = \frac{-1+i\sqrt{3}}{2}$ and $w^2 = \frac{-1-i\sqrt{3}}{2}$, so

$$y_2 = -\frac{u+v}{2} + \frac{i\sqrt{3}(u-v)}{2},\ y_3 = -\frac{u+v}{2} - \frac{i\sqrt{3}(u-v)}{2}.$$

Undo the change $\beta^* = y - \frac{h_2}{3h_3}$, we get our desired solutions. □

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