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Optimal Control of HIV/AIDS Epidemics: Integrating Media Awareness and Antiviral Treatment

Regina Wahyudyah Sonata Ayu[1*], Robiatul Witari Wilda[2], Sailah Ar Rizka[3], Mega Yumia[4], and Febrianto Afli[5]

[1,2,4,5]Department of Mathematics, Universitas Palangka Raya, Indonesia
[1]reginawsayu@mipa.upr.ac.id, [2]robiatulwitariwilda@mipa.upr.ac.id,
[4]megayumia@mipa.upr.ac.id, [5]febrianto.afli@mipa.upr.ac.id
[3]Department of Mathematics, Universitas Jember, Indonesia, sailaharrizka@gmail.com.
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Abstract. This paper investigates an optimal control strategy to mitigate the spread of HIV/AIDS by integrating media awareness campaigns and antiviral treatment efforts. A modified SI-type model is developed, dividing the population into five subgroups: unaware susceptible individuals, aware susceptible individuals, unaware infected individuals, aware infected individuals, and individuals undergoing treatment. Additionally, a separate compartment representing the level of media awareness is included to model the dynamics of awareness campaigns over time. Three control variables are introduced: the success of media awareness programs aimed at reducing contact between susceptible and infected individuals and encouraging infected individuals to seek and receive treatment; the effort to provide antiretroviral treatment; and the effort to strengthen the intensity of media awareness programs. The objective is to minimize the number of unaware susceptible and infected individuals while maximizing the number of individuals receiving treatment, and to reduce implementation costs. The model employs optimal control theory to identify the best combination of strategies by minimizing a cost functional. Numerical simulations explore seven control strategy combinations, ranging from single to multiple controls. The results indicate that combining all control variables yields the most significant reduction in unaware and infected individuals, a substantial increase in the number of individuals receiving treatment, and effective minimization of costs.

Key words and Phrases: antiviral treatment, HIV/AIDS, media awareness, optimal control

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*Corresponding author
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1. INTRODUCTION

The global HIV/AIDS epidemic remains a major challenge to public health. As of 2023, approximately 39.9 million people were reported to be living with HIV, 65% of whom reside in the WHO African Region. Despite advancements in prevention and treatment, 1.3 million people acquired HIV in 2023, and 630,000 died due to HIV-related illnesses. While there is no definitive cure, effective prevention strategies, timely diagnosis, and access to antiretroviral therapy (ART) have transformed HIV into a manageable chronic condition, enabling long and healthy lives for those affected. Global strategies from organizations like WHO, UNAIDS, and the Global Fund align with the Sustainable Development Goal (SDG) target 3.3, aiming to end the HIV epidemic by 2030. However, achieving this goal requires enhanced efforts, as data from 2023 show that only 72% of people living with HIV had achieved suppressed viral loads, falling short of the ambitious 95-95-95 targets set for 2025. Integrating media-driven awareness campaigns with antiviral treatments offers a promising avenue to bridge these gaps and accelerate progress toward epidemic control [1].

Over the past decades, numerous studies have explored the impact of various interventions on HIV/AIDS transmission. Research has consistently shown that the roll-out of antiretroviral therapy (ART) has significantly reduced HIV-related mortality in sub-Saharan Africa. During the first decade of ART expansion, overall mortality rates in the population decreased by approximately 30%, with HIV-attributable deaths falling by 30-50% [2]. In Ethiopia, studies reveal that the "test and treat" approach significantly reduces mortality rates among individuals living with HIV compared to those without treatment exposure. Early diagnosis, adherence to ART, and achieving optimal viral suppression are crucial for maximizing the benefits of this approach [3]. Despite these efforts, mortality rates among patients on ART remain influenced by baseline health conditions, such as CD4 counts, comorbidities, and disease stage at initiation. Close monitoring and tailored interventions for high-risk individuals are critical to improving outcomes [4]. Nevertheless, the ongoing high rate of new infections points to the need for complementary strategies, particularly behavioral interventions aimed at reducing high-risk behaviors. Recent stochastic modeling studies in other infectious diseases also suggest that unpredictability and noise in transmission dynamics can influence intervention success, reinforcing the value of adaptive control strategies in public health contexts [5, 6].

Incorporating media awareness programs is crucial for HIV prevention efforts, particularly in raising awareness and promoting behavior change. However, their effectiveness often depends on the campaign design and the target audience. For instance, exploratory reviews suggest that while mass media interventions are effective in reaching large populations, their impact on behavior change, such as HIV testing or disclosure, is limited unless combined with interpersonal skills development and more in-depth interventions [7]. These campaigns are most effective when focused on raising awareness and setting the context for reducing stigma and changing societal norms. In Zambia, studies examining health communication strategies among sex workers revealed that negatively framed campaigns that evoked fear about HIV/AIDS were more effective in encouraging behavior change, such as quitting sex work, compared to campaigns aimed at fighting stigma with positive messages [8]. Similarly, stochastic models in hepatitis B dynamics have shown that media awareness alone may not lead to disease eradication unless coupled with strong treatment or vaccination interventions, especially when accounting for random environmental fluctuations [9].

Previous studies have extensively employed mathematical modeling to explore various aspects of HIV/AIDS prevention, treatment, and control strategies. For instance, modeling approaches have been applied to understand the immune response to HIV, highlighting gaps in knowledge regarding innate immunity and the role of adaptive responses in controlling infection [10]. A general equilibrium model examined the Malawian epidemic, focusing on behavioral changes in sexual practices in response to public policy interventions, such as antiretroviral therapy (ART) and condom use, demonstrating the interplay between individual choices and epidemic dynamics [11]. The pivotal influence of social factors in health outcomes, including stigma and access to services, has also been modeled, emphasizing the need for holistic interventions that integrate both epidemiological and socioeconomic factors [12]. Additionally, optimal control strategies have been investigated to identify cost-effective treatment allocation schemes, revealing that early treatment can minimize infections, while late treatment is more cost-efficient for reducing deaths [13]. Public education campaigns and voluntary testing have been shown to significantly influence epidemic control, with optimal interventions tailored to specific population groups or resource constraints [14, 15]. Moreover, the effectiveness of combining media campaigns with screening and treatment strategies has been explored, underscoring their synergistic impact on controlling disease spread [16, 17]. In related settings, stochastic optimal control models for other infectious diseases have demonstrated how effective policies can be designed under uncertainty [18], and similar stochastic modeling frameworks have been extended even into cybersecurity contexts, such as worm propagation in sensor networks [19]. These works collectively underline the potential of integrating media awareness programs with antiviral treatments, providing a strong foundation for developing optimal control strategies to mitigate the HIV/AIDS epidemic effectively.

This paper suggests an optimal control model to integrate media awareness programs and antiviral treatment efforts in controlling the spread of HIV/AIDS. We develop a modified SI-type model that divides the population into five subgroups: unaware susceptible individuals, aware susceptible individuals, unaware infected individuals, aware infected individuals, and individuals receiving treatment. The model extends the awareness-based HIV/AIDS model proposed by Roy et al. [20] by incorporating a treatment compartment and redefining the control strategy, to better represent behavioral responses and treatment dynamics. Three control variables are introduced: the success of media awareness programs, the effort to provide antiretroviral treatment, and the effort to strengthen the media awareness programs. The objective is to decrease the number of unaware susceptible and infected individuals, maximize the number of individuals receiving treatment, and minimize the associated costs of implementing these interventions.

2. MATHEMATICAL MODEL FORMULATION

This HIV/AIDS spread model incorporates media awareness. It assumes that when the media disseminates information about HIV/AIDS, some susceptible individuals reduce contact with infected persons or avoid sexual activities or other transmission risk factors. As a result, individuals are categorized into aware and unaware groups. In this model, it is assumed that aware susceptible individuals are effectively protected from infection due to behavioral changes (e.g., reduced contact, consistent condom use), such that their infection risk is negligible. In addition, aware infected individuals are more likely to seek treatment. Therefore, the total population is divided into five subpopulations: unaware susceptible individuals ($S_u(t)$), aware susceptible individuals ($S_a(t)$), unaware infected individuals ($I_u(t)$), aware infected individuals ($I_a(t)$), and those receiving treatment ($T(t)$). The model also introduces a dynamic variable $M(t)$ to represent the level of media awareness over time, which influences transitions between unaware and aware classes. The dynamics of HIV/AIDS spread with media awareness are shown in Figure 1.

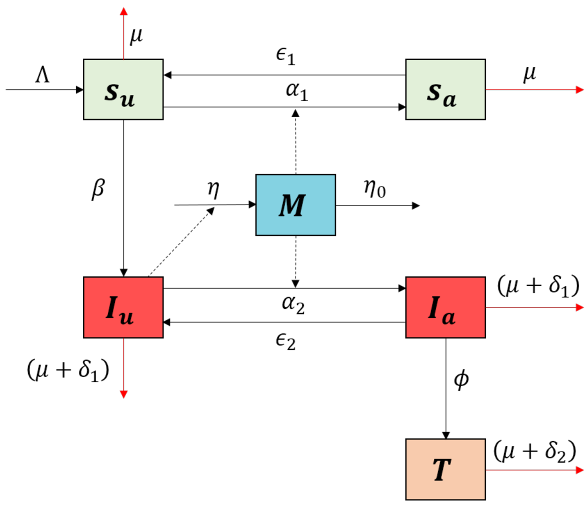

FIGURE 1. Transmission diagram of HIV/AIDS spread dynamics

The constant recruitment rate (Λ) adds new individuals to the susceptible unaware compartment (S_u). The susceptible individuals who are unaware can become infected by contact with unaware infected individuals at an infection rate of β or become aware through media campaigns at a rate of α_1. Additionally, due to media influence, unaware infected individuals become aware infected individuals at a rate of α_2. However, due to memory limitations, both aware susceptible and infected individuals may revert to unaware at rates ϵ_1 and ϵ_2, respectively. Some aware infected individuals can receive antiretroviral treatment at a rate ϕ. The model assumes natural death occurs at rate μ for all subpopulations, while infected individuals and those receiving treatment may die from HIV/AIDS at rates δ_1 and δ_2, respectively. Furthermore, individuals receiving treatment have a lower HIV/AIDS-related death rate than those who are not treated ($\delta_1 > \delta_2$). The media awareness program is implemented proportionally with changes in the number of unaware infected individuals at a rate of η, but decreases with a rate of η_0 due to its inefficiency.

Based on these assumptions and the transmission diagram, the HIV/AIDS spread model is expressed as a set of nonlinear differential equations.

$$\begin{aligned}
\frac{dS_u}{dt} &= \Lambda - \beta S_u I_u - \alpha_1 S_u M + \epsilon_1 S_a - \mu S_u;\\
\frac{dS_a}{dt} &= \alpha_1 S_u M - \epsilon_1 S_a - \mu S_a;\\
\frac{dI_u}{dt} &= \beta S_u I_u - \alpha_2 I_u M + \epsilon_2 I_a - (\mu + \delta_1) I_u;\\
\frac{dI_a}{dt} &= \alpha_2 I_u M - \phi I_a - \epsilon_2 I_a - (\mu + \delta_1) I_a;\\
\frac{dT}{dt} &= \phi I_a - (\mu + \delta_2) T;\\
\frac{dM}{dt} &= \eta I_u - \eta_0 M.
\end{aligned} \tag{1}$$

3. ANALYSIS OF THE MODEL

3.1. **Positivity and Boundedness of the Model.**

In this section, it will be shown that the state variables remain positive and bounded, which implies that the solution to system (1), with non-negative initial values, will stay non-negative for all times $t > 0$ and will be bounded by a non-negative value. To demonstrate positivity, first consider

$$\frac{dM}{dt} = \eta I_u - \eta_0 M \geq -\eta_0 M. \tag{2}$$

Solving equation (2) yields

$$M(t) \geq C_1 e^{-\eta_0 t}$$

where C_1 is a positive constant. Consequently, $\forall t \geq 0, M(t) \geq 0$. Then consider the equation

$$\frac{dT}{dt} = \phi I_a - (\mu + \delta_2) T \geq -(\mu + \delta_2) T. \tag{3}$$

Solving equation (3) yields

$$T(t) \geq C_2 e^{-(\mu+\delta_2)t}$$

where C_2 is a positive constant. Consequently, $\forall t \geq 0, T(t) \geq 0$. Using a similar approach, it can be verified that $\forall t \geq 0, S_u(t), S_a(t), I_u(t), I_a(t) \geq 0$. To demonstrate boundedness, consider that the total population of humans at time t is given by

$$N(t) = S_u(t) + S_a(t) + I_u(t) + I_a(t) + T(t).$$

Thus, based on the system (1), the time-dependent rate of change of the total human population is expressed as

$$\frac{dN}{dt} = \Lambda - \mu N - \delta_1(I_u + I_a) - \delta_2 T \leq \Lambda - \mu N.$$

Consequently, as $t \to \infty, N \to \frac{\Lambda}{\mu}$, which means, N, the total population is bounded above by max $\left(N(0), \frac{\Lambda}{\mu}\right)$. Since all variables are non-negative, the same bound on N also applies as the maximum value for S_u, S_a, I_u, I_a, and T.

3.2. **Equilibrium Analysis.**

The system (1) has an equilibrium point corresponding to a disease-free state, denoted as

$$E_0 = (S_{u0}, S_{a0}, I_{u0}, I_{a0}, T_0, M_0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0\right)$$

and the basic reproduction number R_0 is given by

$$R_0 = \frac{\Lambda\beta}{\mu(\mu+\delta_1)}.$$

Theorem 3.1. *Local asymptotic stability of the disease-free equilibrium point, E_0, holds if $R_0 < 1$.*

Proof. The Jacobian matrix corresponding to the system (1) is given by

$$J = \begin{pmatrix} a_{11} & \epsilon_1 & -\beta S_u & 0 & 0 & -\alpha_1 S_u \\ \alpha_1 M & a_{22} & 0 & 0 & 0 & \alpha_1 S_u \\ \beta I_u & 0 & a_{33} & \epsilon_2 & 0 & -\alpha_2 I_u \\ 0 & 0 & \alpha_2 M & a_{44} & 0 & \alpha_2 I_u \\ 0 & 0 & 0 & \phi & -(\mu+\delta_2) & 0 \\ 0 & 0 & \eta & 0 & 0 & -\eta_0 \end{pmatrix}$$

where $a_{11} = -(\beta I_u + \alpha_1 M + \mu)$, $a_{22} = -(\epsilon_1 + \mu)$, $a_{33} = \beta S_u - \alpha_2 M - (\mu+\delta_1)$, and $a_{44} = -\epsilon_2 - \phi - (\mu+\delta_1)$. The stability of E_0 can be determined by substituting the equilibrium point E_0 into the Jacobian matrix, resulting in

$$J = \begin{pmatrix} -\mu & \epsilon_1 & -\beta\frac{\Lambda}{\mu} & 0 & 0 & -\alpha_1\frac{\Lambda}{\mu} \\ 0 & -(\epsilon_1+\mu) & 0 & 0 & 0 & \alpha_1\frac{\Lambda}{\mu} \\ 0 & 0 & \beta\frac{\Lambda}{\mu}-(\mu+\delta_1) & \epsilon_2 & 0 & 0 \\ 0 & 0 & 0 & -\epsilon_2-\phi-(\mu+\delta_1) & 0 & 0 \\ 0 & 0 & 0 & \phi & -(\mu+\delta_2) & 0 \\ 0 & 0 & \eta & 0 & 0 & -\eta_0 \end{pmatrix}.$$

The eigenvalues of the Jacobian matrix $J(E_0)$ are

$$\lambda_1 = -\mu, \quad \lambda_2 = -(\epsilon_1+\mu), \quad \lambda_3 = -(\mu+\delta_2), \quad \lambda_4 = -\eta_0$$

and the remaining two eigenvalues are roots of the quadratic equation

$$\lambda^2 + \zeta_1\lambda + \zeta_2 = 0$$

where

$$\zeta_1 = -\frac{\Lambda\beta}{\mu} + 2(\mu+\delta_1) - \epsilon_2 + \phi;$$
$$\zeta_2 = \left(\beta\frac{\Lambda}{\mu} - \mu - \delta_1\right)(-\epsilon_2 - \phi - \mu - \delta_1).$$

The eigenvalues are

$$\lambda_{5,6} = \frac{-\zeta_1 \pm \sqrt{\zeta_1^2 - 4\zeta_2}}{2}.$$

The equilibrium E_0 will be locally asymptotically stable provided that $Re(\lambda_{5,6}) < 0$. The eigenvalues $\lambda_{5,6}$ .yield several possibilities for stability

(1) If $\zeta_1^2 - 4\zeta_2 < 0$, $\lambda_{5,6} \in \mathbb{C} Re(\lambda_{5,6}) < 0$;
(2) If $\zeta_1^2 - 4\zeta_2 = 0$, $\lambda_5 = \lambda_6 = -\frac{\zeta_1}{2} < 0$;
(3) If $\zeta_1^2 - 4\zeta_2 > 0$, $\lambda_{5,6} \in \mathbb{R}$ and

$$\lambda_{5,6} < 0 \quad \text{if} \quad \left(\beta\frac{\Lambda}{\mu} - \mu - \delta_1\right)(-\epsilon_2 - \phi - \mu - \delta_1) > 0$$

or

$$R_0 = \frac{\Lambda\beta}{\mu(\mu+\delta_1)} < 1.$$

□

4. OPTIMAL CONTROL PROBLEM

The application of optimal control is explored in this section for the HIV/AIDS spread dynamics model in system (1). The following control variables are introduced

(1) $u_1 \in [0,1]$ represents the success of the media awareness program for susceptible individuals to limit contact with infected individuals or vice versa, and for infected individuals who are aware to seek and receive treatment;
(2) $u_2 \in [0,1]$ represents efforts in antiretroviral treatment;

(3) $u_3 \in [0,1]$ represents efforts to strengthen the media awareness program.

The resulting controlled system is given by

$$\begin{aligned}
\frac{dS_u}{dt} &= \Lambda - \beta S_u I_u - \alpha_1(1+u_1)S_u M + \epsilon_1 S_a - \mu S_u;\\
\frac{dS_a}{dt} &= \alpha_1(1+u_1)S_u M - \epsilon_1 S_a - \mu S_a;\\
\frac{dI_u}{dt} &= \beta S_u I_u - \alpha_2(1+u_1)I_u M + \epsilon_2 I_a - (\mu+\delta_1)I_u;\\
\frac{dI_a}{dt} &= \alpha_2(1+u_1)I_u M - \phi(1+u_2)I_a - \epsilon_2 I_a - (\mu+\delta_1)I_a;\\
\frac{dT}{dt} &= \phi(1+u_2)I_a - (\mu+\delta_2)T;\\
\frac{dM}{dt} &= \eta(1+u_3)I_u - \eta_0 M.
\end{aligned} \tag{4}$$

The objective of this optimal control design is to minimize the number of susceptible and infected individuals who are unaware, maximize the number of individuals receiving treatment, and minimize all costs associated with implementing the controls u_1, u_2, and u_3. Therefore, the objective function for the system (4) is given by

$$\mathcal{J}(u_1,u_2,u_3) = \int_0^{t_f} \left[AS_u + BI_u - CT + \frac{1}{2}W_1u_1^2 + \frac{1}{2}W_2u_2^2 + \frac{1}{2}W_3u_3^2 \right] dt. \tag{5}$$

We seek optimal controls u_1, u_2 and u_3 in $\mathcal{U}$ such that

$$\min \mathcal{J}(u_1,u_2,u_3) \quad \text{subject to (4)} \tag{6}$$

for which

$$\mathcal{U}_{ad} = \{u_1,u_2,u_3 \mid u_1,\ u_2 \text{ and } u_3 \text{ are Lebesgue integrable}, 0 \le u_i \le 1, i=1,2,3\}$$

represents the control set.

4.1. **The existence of an optimal control.**

Theorem 4.1. *An optimal control (u_1^*, u_2^*, u_3^*) exists for problem* (6).

Proof. We apply the necessary criteria from [21] to prove this theorem, first by denoting the right-hand side of (4) by $g(t,\vec{x},\vec{u})$. We aim to prove that the following conditions hold

(a) g is a C^1 function and for some constant K, it holds that

$$|g(t,0,0)| \le K, \quad |g_{\vec{x}}(t,\vec{x},\vec{u})| \le K(1+|\vec{u}|), \quad |g_{\vec{u}}(t,\vec{x},\vec{u})| \le K;$$

(b) The set $\mathfrak{F}$, which includes all solutions to system (1) with controls within $\mathcal{U}_{ad}$ is nonempty;

(c) $g(t,\vec{x},\vec{u}) = a(t,\vec{x}) + b(t,\vec{x})\vec{u}$;

(d) The control set $\mathcal{U} = [0,1]\times[0,1]\times[0,1]$ is closed, convex and compact;

(e) The objective functional's integrand is convex with respect to $\mathcal{U}$.

To verify the conditions mentioned above, we write

$$g\left(t,\vec{x},\vec{u}\right)=\begin{pmatrix}\Lambda-\beta S_uI_u-\alpha_1(1+u_1)S_uM+\epsilon_1S_a-\mu S_u\\ \alpha_1(1+u_1)S_uM-\epsilon_1S_a-\mu S_a\\ \beta S_uI_u-\alpha_2(1+u_1)I_uM+\epsilon_2I_a-(\mu+\delta_1)I_u\\ \alpha_2(1+u_1)I_uM-\phi(1+u_2)I_a-\epsilon_2I_a-(\mu+\delta_1)I_a\\ \phi(1+u_2)I_a-(\mu+\delta_2)T\\ \eta(1+u_3)I_u-\eta_0M\end{pmatrix}.$$

It follows straightforwardly that $g\left(t,\vec{x},\vec{u}\right)$ is a C^1 function and $|g(t,0,0)|=\Lambda$. Moreover, we have

$$|g_{\vec{x}}\left(t,\vec{x},\vec{u}\right)|=\begin{vmatrix}g_{11}&\epsilon_1&-\beta S_u&0&0&-\alpha_1(1+u_1)S_u\\ \alpha_1(1+u_1)M&g_{22}&0&0&0&\alpha_1(1+u_1)S_u\\ \beta I_u&0&g_{33}&\epsilon_2&0&-\alpha_2(1+u_1)I_u\\ 0&0&\alpha_2(1+u_1)M&g_{44}&0&\alpha_2(1+u_1)I_u\\ 0&0&0&\phi(1+u_2)&g_{55}&0\\ 0&0&\eta(1+u_3)&0&0&-\eta_0\end{vmatrix}$$

where $g_{11}=-\beta I_u-\alpha_1(1+u_1)M-\mu$, $g_{22}=-\epsilon_1-\mu$, $g_{33}=\beta S_u-\alpha_2(1+u_1)M-(\mu+\delta_1)$, $g_{44}=-\phi(1+u_2)-\epsilon_2-(\mu+\delta_1)$, $g_{55}=-(\mu+\delta_2)$, and

$$|g_{\vec{u}}\left(t,\vec{x},\vec{u}\right)|=\begin{vmatrix}-\alpha_1S_uM&0&0\\ \alpha_1S_uM&0&0\\ -\alpha_2I_uM&0&0\\ \alpha_2I_uM&-\phi I_a&0\\ 0&\phi I_a&0\\ 0&0&\eta I_u\end{vmatrix}.$$

Since S_u, S_a, I_u, I_a, T, and M are bounded, there is a constant K for which

$$|g(t,0,0)|\le K,\quad |g_{\vec{x}}\left(t,\vec{x},\vec{u}\right)|\le K\left(1+|\vec{u}|\right),\quad |g_{\vec{u}}\left(t,\vec{x},\vec{u}\right)|\le K.$$

This implies that condition (a) is satisfied. As a consequence of condition (a), a unique solution to system (1) exists for a constant control, which in turn ensures that condition (b) is satisfied.

Furthermore,

$$g\left(t,\vec{x},\vec{u}\right)=\begin{pmatrix}\Lambda-\beta S_uI_u-\alpha_1S_uM+\epsilon_1S_a-\mu S_u\\ \alpha_1S_uM-\epsilon_1S_a-\mu S_a\\ \beta S_uI_u-\alpha_2I_uM+\epsilon_2I_a-(\mu+\delta_1)I_u\\ \alpha_2I_uM-\phi I_a-\epsilon_2I_a-(\mu+\delta_1)I_a\\ \phi I_a-(\mu+\delta_2)T\\ \eta I_u-\eta_0M\end{pmatrix}+\begin{pmatrix}-\alpha_1S_uM&0&0\\ \alpha_1S_uM&0&0\\ -\alpha_2I_uM&0&0\\ \alpha_2I_uM&-\phi I_a&0\\ 0&\phi I_a&0\\ 0&0&\eta I_u\end{pmatrix}\times\begin{pmatrix}u_1\\ u_2\\ u_3\end{pmatrix}.$$

Thus, condition (c) is satisfied. It is clear from the definition that condition (d) is satisfied, so we now proceed to prove condition (e). First, let $f\left(t,\vec{x},\vec{u}\right)$ be the function representing the integrand in the objective functional that is

$$f(t,\vec{x},\vec{u}) = AS_u + BI_u - CT + \frac{1}{2}W_1u_1^2 + \frac{1}{2}W_2u_2^2 + \frac{1}{2}W_3u_3^2$$

then it is necessary to prove that

$$(1-\tau)\, f(t,\vec{x},\vec{u}) + \tau f(t,\vec{x},\vec{v}) \geq f(t,\vec{x},(1-\tau)\,\vec{u}+\tau\vec{v})$$

where $\vec{u},\vec{v}$ are control vectors and $\tau \in [0,1]$. Consequently, we obtain that

$$\begin{aligned}(1-\tau)\, f(t,\vec{x},\vec{u}) + \tau f(t,\vec{x},\vec{v}) &= (1-\tau)\left[AS_u + BI_u - CT + \frac{1}{2}W_1u_1^2 + \frac{1}{2}W_2u_2^2 + \frac{1}{2}W_3u_3^2\right] \\ &\quad + \tau\left[AS_u + BI_u - CT + \frac{1}{2}W_1v_1^2 + \frac{1}{2}W_2v_2^2 + \frac{1}{2}W_3v_3^2\right] \\ &= AS_u + BI_u - CT + (1-\tau)\left[\frac{1}{2}W_1u_1^2 + \frac{1}{2}W_2u_2^2 + \frac{1}{2}W_3u_3^2\right] \\ &\quad + \tau\left[\frac{1}{2}W_1v_1^2 + \frac{1}{2}W_2v_2^2 + \frac{1}{2}W_3v_3^2\right]\end{aligned}$$

and

$$\begin{aligned}f(t,\vec{x},(1-\tau)\,\vec{u}+\tau\vec{v}) &= AS_u + BI_u - CT + \frac{1}{2}W_1\left[(1-\tau)\,u_1 + \tau v_1\right]^2 \\ &\quad + \frac{1}{2}W_2\left[(1-\tau)\,u_2 + \tau v_2\right]^2 + \frac{1}{2}W_3\left[(1-\tau)\,u_3 + \tau v_3\right]^2.\end{aligned}$$

Moreover, it follows that

$$\begin{aligned}&(1-\tau)\, f(t,\vec{x},\vec{u}) + \tau f(t,\vec{x},\vec{v}) - f(t,\vec{x},(1-\tau)\,\vec{u}+\tau\vec{v}) \\ &= \frac{1}{2}W_1\left[(1-\tau)\,u_1^2 + \tau v_1^2\right] + \frac{1}{2}W_2\left[(1-\tau)\,u_2^2 + \tau v_2^2\right] + \frac{1}{2}W_3\left[(1-\tau)\,u_3^2 + \tau v_3^2\right] \\ &\quad - \frac{1}{2}W_1\left[(1-\tau)\,u_1 + \tau v_1\right]^2 - \frac{1}{2}W_2\left[(1-\tau)\,u_2 + \tau v_2\right]^2 - \frac{1}{2}W_3\left[(1-\tau)\,u_3 + \tau v_3\right]^2 \\ &= \frac{1}{2}W_1\left\{(1-\tau)\,u_1^2 + \tau v_1^2 - \left[(1-\tau)\,u_1 + \tau v_1\right]^2\right\} + \\ &\quad \frac{1}{2}W_2\left\{(1-\tau)\,u_2^2 + \tau v_2^2 - \left[(1-\tau)\,u_2 + \tau v_2\right]^2\right\} + \\ &\quad \frac{1}{2}W_3\left\{(1-\tau)\,u_3^2 + \tau v_3^2 - \left[(1-\tau)\,u_3 + \tau v_3\right]^2\right\} \\ &= \frac{1}{2}W_1\left[\sqrt{\tau(1-\tau)}u_1 - \sqrt{\tau(1-\tau)}v_1\right]^2 + \frac{1}{2}W_2\left[\sqrt{\tau(1-\tau)}u_2 - \sqrt{\tau(1-\tau)}v_2\right]^2 \\ &\quad + \frac{1}{2}W_3\left[\sqrt{\tau(1-\tau)}u_3 - \sqrt{\tau(1-\tau)}v_3\right]^2 \geq 0\end{aligned}$$

which completes the proof. $\square$

4.2. **The optimality characterization.**

We proceed to establish the conditions that must be satisfied by a pair of optimal control and the corresponding state variables by applying Pontryagin's maximum principle [22]. First, we define the Hamiltonian function for the system as

$$\begin{aligned}\mathcal{H} = {} & AS_u + BI_u - CT + \frac{1}{2}W_1u_1^2 + \frac{1}{2}W_2u_2^2 + \frac{1}{2}W_3u_3^2 \\ & + \psi_1\left[\Lambda - \beta S_u I_u - \alpha_1(1+u_1)S_uM + \epsilon_1 S_a - \mu S_u\right] \\ & + \psi_2\left[\alpha_1(1+u_1)S_uM - \epsilon_1 S_a - \mu S_a\right] \\ & + \psi_3\left[\beta S_u I_u - \alpha_2(1+u_1)I_uM + \epsilon_2 I_a - (\mu+\delta_1)I_u\right] \\ & + \psi_4\left[\alpha_2(1+u_1)I_uM - \phi(1+u_2)I_a - \epsilon_2 I_a - (\mu+\delta_1)I_a\right] \\ & + \psi_5\left[\phi(1+u_2)I_a - (\mu+\delta_2)T\right] \\ & + \psi_6\left[\eta(1+u_3)I_u - \eta_0 M\right]\end{aligned} \tag{7}$$

where $\psi_i, i = 1, 2, \ldots, 6$ denote the adjoint variables. To determine the optimality system, the partial derivatives of the Hamiltonian (7) are taken with respect to the corresponding state variables.

Theorem 4.2. *Let the optimal control (u_1^*, u_2^*, u_3^*) and corresponding state solutions S_u, S_a, I_u, I_a, T, M of the system (4), there exist adjoint variables, ψ_i, for $i = 1, 2, \ldots, 6$ that satisfy*

$$\begin{aligned}\frac{d\psi_1}{dt} &= -A + (\beta I_u + \alpha_1(1+u_1)M + \mu)\,\psi_1 - \alpha_1(1+u_1)M\psi_2 - \beta I_u\psi_3; \\ \frac{d\psi_2}{dt} &= -\epsilon_1\psi_1 + (\epsilon_1+\mu)\,\psi_2; \\ \frac{d\psi_3}{dt} &= -B + \beta S_u\psi_1 - \left[\beta S_u - \alpha_2(1+u_1)M - (\mu+\delta_1)\right]\psi_3 - \\ &\quad\ \alpha_2(1+u_1)M\psi_4 - \eta\,(1+u_3)\,\psi_6; \\ \frac{d\psi_4}{dt} &= -\epsilon_2\psi_3 + \left[\phi\,(1+u_2) + \epsilon_2 + (\mu+\delta_1)\right]\psi_4 - \phi\,(1+u_2)\,\psi_5; \\ \frac{d\psi_5}{dt} &= C + (\mu+\delta_2)\,\psi_5; \\ \frac{d\psi_6}{dt} &= \alpha_1(1+u_1)S_u\,(\psi_1-\psi_2) + \alpha_2(1+u_1)I_u\,(\psi_3-\psi_4) + \eta_0\psi_6;\end{aligned} \tag{8}$$

with terminal conditions

$$\psi_i(t_f) = 0 \quad \textit{for} \quad i = 1, 2, \ldots, 6.$$

In addition, the optimal controls u_1^*, u_2^*, u_3^* are defined by

$$\begin{aligned}u_1^* &= \max\left\{0, \min\left\{\frac{1}{W_1}\left[\alpha_1 S_u M(\psi_1-\psi_2) + \alpha_2 I_u M(\psi_3-\psi_4)\right], 1\right\}\right\}; \\ u_2^* &= \max\left\{0, \min\left\{\frac{\phi}{W_2}I_a(\psi_4-\psi_5), 1\right\}\right\}; \\ u_3^* &= \max\left\{0, \min\left\{-\frac{\eta}{W_3}I_u\psi_6, 1\right\}\right\}.\end{aligned} \tag{9}$$

Proof. We get the adjoint system (8) as a result of Pontryagin's Principle

$$\frac{d\psi_1}{dt} = -\frac{\partial \mathcal{H}}{\partial S_u}, \quad \frac{d\psi_2}{dt} = -\frac{\partial \mathcal{H}}{\partial S_a}, \quad \ldots, \quad \frac{d\psi_6}{dt} = -\frac{\partial \mathcal{H}}{\partial M}.$$

subject to zero conditions at the final time (transversality).

To determine the optimal control as characterized in (9), we solve the equations within the interior of the control set

$$\frac{\partial \mathcal{H}}{\partial u_1} = 0, \qquad \frac{\partial \mathcal{H}}{\partial u_2} = 0, \qquad \frac{\partial \mathcal{H}}{\partial u_3} = 0.$$

By applying the control bounds, the desired characterization is obtained. □

5. NUMERICAL SIMULATION

The state and adjoint equations obtained are nonlinear and difficult to solve analytically. Therefore, these equations will be solved numerically using the Forward Backward Sweep Method. Subsequently, all combinations of the three controls are numerically tested to observe their effects on minimizing the number of unaware individuals, maximizing the number of individuals receiving treatment, and minimizing the cost function. In total, there are seven (7) control scenarios

(1) **Strategy 1**: Success of the media awareness program for susceptible individuals to limit contact with infected individuals or vice versa, and for infected individuals who are aware to seek and receive treatment (u_1), where $u_2 = u_3 = 0$.
(2) **Strategy 2**: Antiretroviral treatment efforts (u_2), where $u_1 = u_3 = 0$.
(3) **Strategy 3**: Efforts to strengthen the media awareness program (u_3) where $u_1 = u_2 = 0$.
(4) **Strategy 4**: Success of the media awareness program for susceptible individuals to limit contact with infected individuals or vice versa, and for infected individuals who are aware to seek and receive treatment (u_1) and antiretroviral treatment efforts (u_2), where $u_3 = 0$.
(5) **Strategy 5**: Success of the media awareness program for susceptible individuals to limit contact with infected individuals or vice versa, and for infected individuals who are aware to seek and receive treatment (u_1) and efforts to strengthen the media awareness program (u_3), where $u_2 = 0$.
(6) **Strategy 6**: Antiretroviral treatment efforts (u_2) and efforts to strengthen the media awareness program (u_3), where $u_1 = 0$.
(7) **Strategy 7**: Using all controls, i.e., the success of the media awareness program for susceptible individuals to limit contact with infected individuals or vice versa, and for infected individuals who are aware to seek and receive treatment (u_1), antiretroviral treatment efforts (u_2), and efforts to strengthen the media awareness program (u_3).

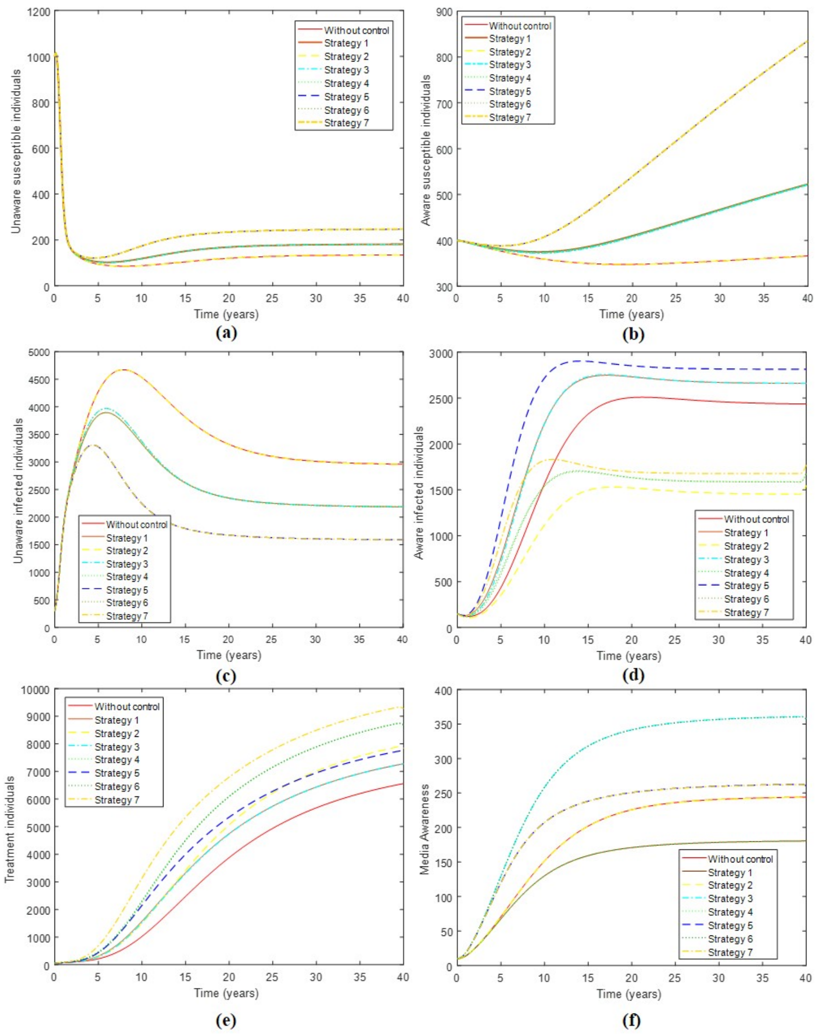

FIGURE 2. Simulation results for all states under seven control strategies

The simulation of the optimal control model is carried out using parameter values consistent with previous studies and assumptions. Specifically, the recruitment rate is assumed to be $\Lambda = 1000$ year^{-1}, and the infection rate between unaware susceptible and unaware infected individuals is set as $\beta = 0.0025$ year^{-1} [23]. The media influence rates are taken as $\alpha_1 = 0.0002$ year^{-1} [24] for susceptible individuals and $\alpha_2 = 0.001$ year^{-1} (assumed) for infected individuals.

The proportion of aware infected individuals who seek and receive antiretroviral treatment is assumed to be $\phi = 0.2$, while the reversion rates due to memory limitations are $\epsilon_1 = 0.02$ year^{-1} [24] for aware susceptible individuals and $\epsilon_2 = 0.0015$ year^{-1} (assumed) for aware infected individuals. The natural death rate is taken as $\mu = 1/71.3$ year^{-1} [25], while HIV/AIDS-induced death rates are $\delta_1 = 0.09$ year^{-1} for infected individuals [26] and $\delta_2 = 0.06$ year^{-1} (assumed) for those receiving treatment. Media-related parameters include the media production rate $\eta = 0.005$ year^{-1} and the media decay rate $\eta_0 = 0.06$ year^{-1} [24].

Numerical simulations for all strategies were conducted using MATLAB with chosen weight values for the objective function (5) as $A = 500, B = 100, C = 1, W_1 = 500, W_3 = 200$, with a control period of $t_f = 40$ years. The results of the numerical simulation of all strategies were then compared with the system's condition before control, i.e., when $u_1 = u_2 = u_3 = 0$.

Strategy 7 showed a significant decrease in the number of unaware infected individuals (Figure 2.c) and an increase in individuals undergoing treatment (Figure 2.e), as well as a significant increase in the number of aware susceptible individuals (Figure 2.b) compared to all other strategies. When three controls were applied, the number of infected individuals decreased significantly (Figures 2.c and 2.d), resulting in an increase in susceptible individuals (Figures 2.a and 2.b).

In addition, Figure 2.f illustrates the dynamics of media awareness. Strategy 3 and Strategy 6, which directly involve strengthening media campaigns (u_3), resulted in the most significant increase in media awareness over time. However, despite this increase, the number of unaware infected individuals (I_u) remains relatively high (Figures 2.c). In contrast, Strategy 7 shows both a high level of media awareness and the lowest level of I_u, highlighting that combining all control efforts is more effective than relying on media campaigns alone.

Table 1. The ICER from all control strategies

Strategy	Population size at t_f			Averted	Cost	ICER
	I_u	I_a	T			
No control	2957	2436	6559			
Strategy 3	2187	2660	7274	169	13,719,042	81335
Strategy 1	2189	2658	7276	171	13,673,322	-22422
Strategy 5	1594	2812	7767	221	12,104,100	-31488
Strategy 2	2954	1548	7838	388	16,200,741	24455
Strategy 6	2185	1682	8686	600	13,675,130	-11894
Strategy 4	2187	1680	8687	602	13,629,326	-23840
Strategy 7	1592	1772	9270	682	12,058,493	-19633

To evaluate the most cost-effective combination of the seven strategies considered in this paper, we carry out an analysis of cost-effectiveness using Incremental Cost-Effectiveness Ratio (ICER). The ICER enables us to evaluate the cost-effectiveness of combining two or more control strategies. Incremental comparisons are made by assessing each intervention against the following least effective alternative [27]. The results of the ICER analysis for the seven control strategies are presented in Table 1.

Based on Table 1, it is evident that Strategy 2, Strategy 3, and Strategy 6 are less effective, as indicated by their high ICER values. For example, when comparing Strategy 1 and Strategy 3, the lower ICER for Strategy 1 suggests that Strategy 3 is both more costly and less effective compared to Strategy 1. Therefore, Strategy 3 is excluded to avoid wasting limited resources. The same applies to Strategy 2 and Strategy 6. Subsequently, the ICER is recalculated for the rest of the strategies, as presented in Table 2.

TABLE 2. The ICER for Strategy 1, Strategy 4, Strategy 5, and Strategy 7

Strategy	Population size at t_f			Averted	Cost	ICER
	I_u	I_a	T			
No control	2957	2436	6559			
Strategy 1	2189	2658	7276	171	13,673,322	80095
Strategy 5	1594	2812	7767	221	12,104,100	-31488
Strategy 4	2187	1680	8687	602	13,629,326	3995
Strategy 7	1592	1772	9270	682	12,058,493	-19633

The analysis between Strategy 1 and Strategy 5 shows that Strategy 1 offers a cost saving of 80,095 compared to Strategy 5. However, the lower ICER for Strategy 5 suggests that Strategy 1 is more costly and less effective than Strategy 5. Thus, Strategy 1 is omitted to avoid depleting limited resources. The ICER calculations are repeated and presented in Table 3.

TABLE 3. The ICER for Strategy 4, Strategy 5, and Strategy 7

Strategy	Population size at t_f			Averted	Cost	ICER
	I_u	I_a	T			
No control	2957	2436	6559			
Strategy 5	1594	2812	7767	221	12,104,100	54882
Strategy 4	2187	1680	8687	602	13,629,326	3995
Strategy 7	1592	1772	9270	682	12,058,493	-19633

Strategy 5 offers a cost saving of 54,882 compared to Strategy 4. However, Strategy 5 is dominated due to its higher cost and lower effectiveness, as indicated by the ICER. Thus, Strategy 5 is excluded from the alternatives, and the ICER is recalculated, as shown in the Table 4.

Based on Table 4, Strategy 7 has the smallest ICER value, making it more effective than Strategy 4 and, consequently, the most effective strategy. This is consistent with Figure 3.a, which illustrates the effects of the seven strategies in

Table 4. The ICER for Strategy 4 and Strategy 7

Strategy	Population size at t_f			Averted	Cost	ICER
	I_u	I_a	T			
No control	2957	2436	6559			
Strategy 4	2187	1680	8687	602	13,629,326	22628
Strategy 7	1592	1772	9270	682	12,058,493	-19633

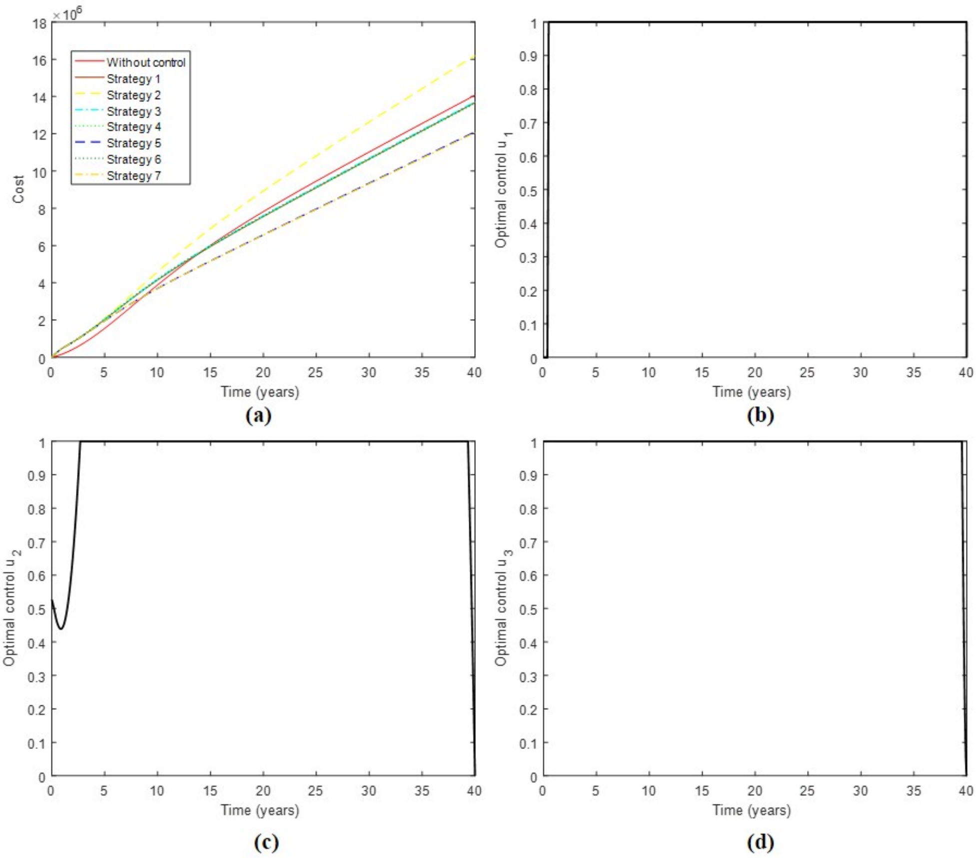

Figure 3. Simulation of the cost function and optimal control

minimizing the cost function $\mathcal{J}$. The optimal control that minimizes $\mathcal{J}$ is presented in Figures 3.b, 3.c, and 3.d.

6. CONCLUSIONS AND FUTURE WORK

A mathematical model for the transmission of HIV/AIDS is formulated, involving a media awareness program $M(t)$, where the total human population $N(t)$ is classified into five subpopulations: unaware susceptible individuals $S_u(t)$, aware susceptible individuals $S_a(t)$, unaware infected individuals $I_u(t)$, aware infected individuals $I_a(t)$, and individuals receiving treatment $T(t)$. It is shown through the analysis that the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$ and that all solutions are bounded and positive for $t > 0$. Additionally, numerical simulations along with the cost-effectiveness analysis indicate that the strategy involving all three controls (u_1, u_2, and u_3) is the most effective.

Future research may extend the current model by incorporating robust optimal control strategies to address parameter uncertainties. Such methods can improve the reliability of control outcomes in the presence of incomplete or variable epidemiological data, thereby enhancing the practical relevance of the model.

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