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STRATEGIES OF OPTIMAL CONTROL FOR HIV SPREADS PREVENTION WITH HEALTH CAMPAIGN

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Abstract. In the present paper, we discuss an HIV (Human Immunodeficiency Virus) transmission model with health information campaign about HIV control policies. The reason behind the conception of this model is the idea to divide the human population that gains awareness of HIV, due to this campaign. We assume that HIV will not infect people who are aware about the dangers of HIV. We analyze the existence and local stability of the equilibrium points. We found that the disease-free equilibrium point will be locally asymptotically stable (LAS) if the basic reproduction number ($\mathscr{R}_0$) is less than one, and unstable otherwise. A forward bifurcation of the system is shown numerically, depending on the intervention parameters. Some numerical simulations for the autonomous system are given to see the evolution of the system, with respect to some scenarios that might appear in the field. To accommodate the limitation of budget issue for implementation in the field, the model is reconstructed as an optimal control problem with two control variables. Numerical simulations for optimal control problems are presented for five different scenarios. Numerical simulation results suggest that controlling strategies by providing health campaigns is better, if they precede an endemic prevention strategy than endemic reduction, since the cost needed for endemic reduction is five times higher, compared with the endemic prevention. Optimal intervention should also note the value of $\mathscr{R}_0$. Larger control levels are needed when $\mathscr{R}_0 > 1$ if compared with when $\mathscr{R}_0 < 1$. The results of numerical simulation also show that the lower the cost of the health campaign, the more health campaigns can be provided. Based on the numerical simulation calculations, the optimal intervention of health campaign can raise public awareness about HIV, in order to reduce the number of HIV-infected individuals.

Keywords: Human Immunodeficiency Virus; health campaign; equilibrium point; local stability; basic reproduction number; optimal control.

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1. INTRODUCTION

Human Immunodeficiency Virus (HIV) is a virus that attacks the human immune system. As long as the virus impairs immune cell functions, especially CD4 cells, the body becomes immunodeficient. The HIV infection further develops into Acquired Immunodeficiency Syndrome (AIDS). Individuals with AIDS experience severe immune system damage and suffer from opportunistic infections. HIV can be transmitted through the exchange of body fluids, such as blood, breast milk, semen, and vaginal secretions. HIV is spread mainly through unprotected sexual contact with HIV-infected individuals and sharing HIV-infected syringes. HIV can also be inherited vertically from an HIV-infected mother to her child. This transmission can occur during pregnancy, the birth process or breastfeeding [1].

In Indonesia, some government efforts to control HIV/AIDS include expanding access to CD4 testing and viral load, increasing coverage of antiretroviral treatment and improving the quality of healthcare facilities [2]. The government also conducts health campaigns to provide education, information and communication to adolescents and adults. The campaigns aim to enhance their knowledge concerning HIV and adopt positive behavior to prevent HIV. One of the programs is ”Aku Bangga, Aku Tahu” (I’m Proud, I Know)–an HIV prevention campaign aimed at young people aged 15-24 years [3]. There are many methods to understand how HIV spreads; one among them is through mathematical models.

Some mathematical models have already been introduced to understand how HIV spreads in the human population, such as by considering ART as prevention and treatment, as proposed by Huo et al. [4], Aldila D. and Maimunah [5], and Silva and Tores [6]. On the other hand, Naresh, Tripathi and Sharma [7] modeled the spread of HIV in the population with immigration from HIV-infected individuals. Furthermore, Giamberardino et al. [8] discussed the HIV prevalence model with three controls, such as information campaign, test campaign, and an HIV/AIDS therapy action. Dubey Preeti et al. [9] introduced a model of HIV dynamics in the body.

In this manuscript, a mathematical model of HIV involving informative campaign about the danger of HIV will be constructed. The model is based on the deterministic model in [8] by adding a specific compartment for susceptible and infected humans, who may be aware or unaware of HIV. Let the human population be divided into six sub-populations; let them be called

as unaware of HIV susceptible individuals (S_1), aware of HIV susceptible individuals (S_2), unaware of HIV-infected individuals in the acute stage (I_1), aware of HIV-infected individuals in the acute stage (I_2), infected individuals in the chronic stage (P) and individuals with AIDS (A). The total population at time t, denoted by $N(t)$, is given by

$$N(t) = S_1(t) + S_2(t) + I_1(t) + I_2(t) + P(t) + A(t).$$

Newborns will be entered into the unaware susceptible group (S_1). There are health campaigns (u_1 and u_2) conducted by the government to control the spread and increase public awareness of HIV/AIDS. Let us define that u_1 is the individuals transition rate from unaware to aware, while u_2 is the opposite. It is assumed that there is a consideration of individual consciousness due to health campaigns, for example, with electronic media campaigns. If the content of the campaign is focused on providing basic knowledge about HIV and its prevention, then the number of individuals aware of the dangers of HIV will increase. In our model, this kind of intervention denoted by the increase of the transition rate from the unaware to the aware compartment (u_1). Conversely, if the content of the campaign is focused on regular appeals to maintain healthy lifestyle habits as a preventive measure of HIV transmission, people are expected to remain aware of HIV. So it is expected that individuals transition rate from aware to unaware does not increase. Examples of implementations in the field include direct campaign in AIDS rehabilitation groups/organization, free distribution of condoms [11], etc. Increasing u_1 and reducing u_2 is the best way to control the HIV spread. Unfortunately, this means of intervention comes at a high cost. Therefore, in this article, both parameters will be treated as time-dependent variables, $u_1(t)$ and $u_2(t)$. Further explanation about the optimal control problem of these parameters will be discussed in section 3.

In this article, although there is a possibility of vertical transmission of HIV to newborn [12], it is assumed that HIV is only transmitted through sexual contact between unaware susceptible individuals and unaware infected individuals in the acute stage. The unaware susceptible individuals will be infected through sexual contact with unaware infected individuals in the acute stage at a rate of transmission β. Infected individuals in the chronic stage and individuals with AIDS are assumed to not transmit HIV because they are already aware of their status of the disease or because of their health conditions that do not permit to make sexual contact.

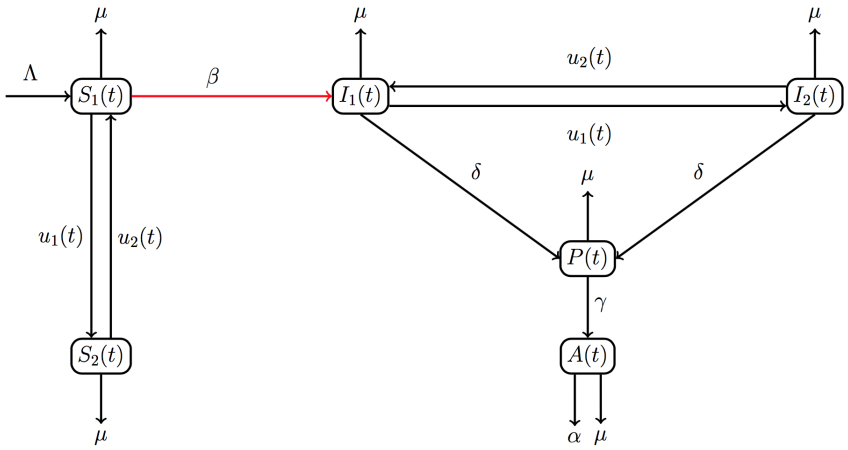


FIGURE 1. Epidemiological scheme of the model

Based on the development of HIV/AIDS in humans [10], it is assumed in this article that infected individuals in the acute stage will move to infected individuals in the chronic stage at a rate δ. In the other hand, infected individuals in the chronic stage will suffer AIDS at a rate γ and individuals with AIDS will die caused by AIDS at a rate of α. With these assumptions, the model is given as follows, together with the parameters described in Table 1 and the epidemiological scheme of the system (1) in Figure 1.

$$\frac{dS_1(t)}{dt} = \Lambda - u_1(t)S_1(t) + u_2(t)S_2(t) - \frac{\beta S_1(t)I_1(t)}{N_c(t)} - \mu S_1(t) \tag{1a}$$

$$\frac{dS_2(t)}{dt} = u_1(t)S_1(t) - u_2(t)S_2(t) - \mu S_2(t) \tag{1b}$$

$$\frac{dI_1(t)}{dt} = \frac{\beta S_1(t)I_1(t)}{N_c(t)} - u_1(t)I_1(t) + u_2(t)I_2(t) - \delta I_1(t) - \mu I_1(t) \tag{1c}$$

$$\frac{dI_2(t)}{dt} = u_1(t)I_1(t) - u_2(t)I_2(t) - \delta I_2(t) - \mu I_2(t) \tag{1d}$$

$$\frac{dP(t)}{dt} = \delta\left(I_1(t) + I_2(t)\right) - (\gamma+\mu)P(t) \tag{1e}$$

$$\frac{dA(t)}{dt} = \gamma P(t) - (\mu+\alpha)A(t), \tag{1f}$$

with $N_c(t) = S_1(t) + S_2(t) + I_1(t) + I_2(t)$, and the initial conditions given as follows

$$S_1(0) = (S_1)_0,\ S_2(0) = (S_2)_0,\ I_1(0) = (I_1)_0,\ I_2(0) = (I_2)_0,\ P(0) = (P)_0,\ A(0) = (A)_0.$$

Parameters	Description	Unit
$u_1(t)$	Transition rate from the unaware to the aware compartment caused by medical campaign	day^{-1}
$u_2(t)$	Transition rate from the aware to the unaware compartment caused by decreased awareness of the dangers of AIDS	day^{-1}
Λ	Recruitment rate from newborn	$\frac{individuals}{day}$
β	Infection rate	day^{-1}
γ	Transition rate from infected in chronic stage to AIDS	day^{-1}
μ	Natural death rate	day^{-1}
α	death rate caused by AIDS	day^{-1}

TABLE 1. Description of parameters in Model (1)

The paper is organized as follows: the state of the art of the model and model construction are carefully detailed in this section and followed by mathematical analysis about the existence and local stability of equilibrium points in section 2. The construction of basic reproduction number is also given in section 2. Mathematical results from the analysis of the basic reproduction number in section 2 are then used in section 3 to identify the optimal parameters as an optimal control problem. Numerical experiments on the autonomous model when control parameters are constant or varying with time are given in section 4. Finally the discussion and conclusion are presented in section 5.

2. ANALYSIS OF THE AUTONOMOUS SYSTEM

Existence of the equilibrium points. To analyze the behaviour of the model in system (1), let us consider the control variables are constant $(u_1(t) = u_1, u_2(t) = u_2)$, such that model (1) now reads as:

$$\frac{dS_1(t)}{dt} = \Lambda - u_1 S_1(t) + u_2 S_2(t) - \frac{\beta S_1(t) I_1(t)}{N_c(t)} - \mu S_1(t) \tag{2a}$$

$$\frac{dS_2(t)}{dt} = u_1 S_1(t) - u_2 S_2(t) - \mu S_2(t) \tag{2b}$$

$$\frac{dI_1(t)}{dt} = \frac{\beta S_1(t) I_1(t)}{N_c(t)} - u_1 I_1(t) + u_2 I_2(t) - \delta I_1(t) - \mu I_1(t) \tag{2c}$$

$$\frac{dI_2(t)}{dt} = u_1 I_1(t) - u_2 I_2(t) - \delta I_2(t) - \mu I_2(t) \tag{2d}$$

$$\frac{dP(t)}{dt} = \delta \left(I_1(t) + I_2(t)\right) - (\gamma + \mu) P(t) \tag{2e}$$

$$\frac{dA(t)}{dt} = \gamma P(t) - (\mu + \alpha) A(t), \tag{2f}$$

Taking the right hand side of model (2), system (2) has two equilibrium points. First equilibrium is the HIV-free equilibrium point, which is given by:

$$\Omega_1 = (S_1, S_2, I_1, I_2, P, A) = \left(\frac{(\mu + u_2)\Lambda}{\mu(\mu + u_1 + u_2)}, \frac{u_1 \Lambda}{\mu(\mu + u_1 + u_2)}, 0, 0, 0, 0 \right). \tag{3}$$

With the HIV-free equilibrium in hand, we are ready to calculate the basic reproduction number of model (2).

Basic reproduction number. The Basic reproduction number ($\mathscr{R}_0$) is defined as the expected number of secondary infections caused by one primary infected individual during a period of infection in a population of all susceptible individuals [13, 14]. Some methods can be used to construct $\mathscr{R}_0$, such as with the Next-Generation matrix approach [14] or with graph theory [15]. In this article, the Next-Generation Matrix is the approach we choose to find the $\mathscr{R}_0$ of system (2). First, let's construct the infective sub-system of (2), which only involves the $I_1(t), I_2(t), P(t)$ and $A(t)$, i.e.,

$$\frac{dI_1(t)}{dt} = \frac{\beta S_1(t) I_1(t)}{N_c(t)} - u_1 I_1(t) + u_2 I_2(t) - \delta I_1(t) - \mu I_1(t) \tag{4a}$$

$$\frac{dI_2(t)}{dt} = u_1 I_1(t) - u_2 I_2(t) - \delta I_2(t) - \mu I_2(t) \tag{4b}$$

$$\frac{dP(t)}{dt} = \delta \left(I_1(t) + I_2(t)\right) - (\gamma + \mu) P(t) \tag{4c}$$

$$\frac{dA(t)}{dt} = \gamma P(t) - (\mu + \alpha) A(t), \tag{4d}$$

Next, using the small domain of next-generation matrix, then the next-generation matrix of system (2) is given by :

$$NGM = \left[\frac{(\mu + u_2)\beta \ (\delta + \mu + u_2)}{(\mu + u_2 + u_1)(\delta + \mu)(u_2 + \delta + u_1 + \mu)} \right].$$

Therefore, the basic reproduction number $\mathscr{R}_0$ of system (2) as the spectral radius of *NGM* is given by:

$$\mathscr{R}_0 = \frac{\beta(\mu+u_2)(\mu+\delta+u_2)}{(\mu+u_1+u_2)(\mu+\delta)(\mu+\delta+u_1+u_2)}. \tag{5}$$

Further discussion about $\mathscr{R}_0$ will be given in the later part of this section.

The next equilibrium is the endemic HIV-equilibrium point, where all susceptible and infected populations coexist. This equilibrium is given by:

$$\Omega_2 = (S_1, S_2, I_1, I_2, P, A) = (S_1^*, S_2^*, I_1^*, I_2^*, P^*, A^*), \tag{6}$$

where

$$\begin{aligned}
S_1^* &= \frac{(\delta+\mu+u_1+u_2)\Lambda(\mu+u_2)}{K_3(\frac{\mu}{\delta}\mathscr{R}_0+\mathscr{R}_0-1)},\\
S_2^* &= \frac{(\delta+\mu+u_1+u_2)\Lambda u_1}{K_3(\frac{\mu}{\delta}\mathscr{R}_0+\mathscr{R}_0-1)},\\
I_1^* &= \frac{\Lambda(\delta+\mu+u_2)K_2(\mathscr{R}_0-1)}{(\delta+\mu)(\delta+\mu+u_1+u_2)K_3(\frac{\mu}{\delta}\mathscr{R}_0+\mathscr{R}_0-1)},\\
I_2^* &= \frac{(\Lambda u_1)K_2(\mathscr{R}_0-1)}{(\delta+\mu)(\delta+\mu+u_1+u_2)K_3(\frac{\mu}{\delta}\mathscr{R}_0+\mathscr{R}_0-1)},\\
P^* &= \frac{(\lambda\delta)K_2(\mathscr{R}_0-1)}{(\gamma+\mu)(\delta+\mu)K_3(\frac{\mu}{\delta}\mathscr{R}_0+\mathscr{R}_0-1)},\\
A^* &= \frac{(\gamma\lambda\delta)K_2(\mathscr{R}_0-1)}{(\gamma+\mu)(\mu+\alpha)(\delta+\mu)K_3(\frac{\mu}{\delta}\mathscr{R}_0+\mathscr{R}_0-1)},
\end{aligned}$$

where

$$\begin{aligned}
K_1 &= \beta(\mu+u_2)(\mu+\delta+u_2),\\
K_2 &= (\mu+u_1+u_2)(\mu+\delta)(\mu+\delta+u_1+u_2),\\
K_3 &= \delta(\mu+u_1+u_2)(\mu+\delta+u_1+u_2).
\end{aligned}$$

From the expression above, it can be seen that all compartments in Ω_2 will be positive if $\mathscr{R}_0 > 1$. These results are stated in the following theorem.

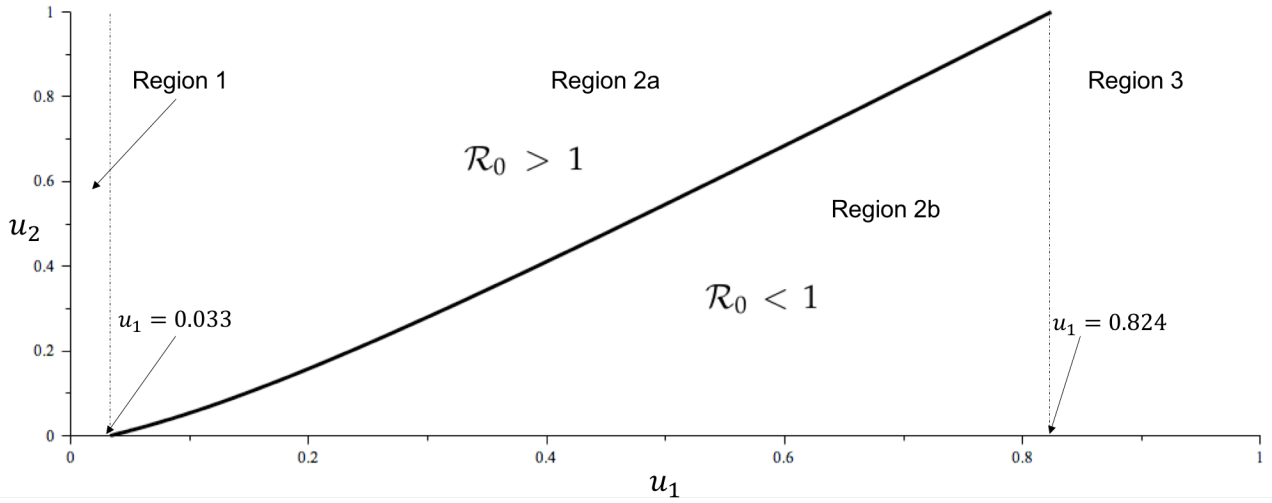


FIGURE 2. Sensitivity analysis of $\mathscr{R}_0$ respect to u_1 and u_2, where the black curve is when $\mathscr{R}_0 = 1$.

Theorem 2.1. *Model* (2) *has two equilibrium points, i.e., the HIV-free equilibrium* (3)*, which exists without any constraint and the endemic-HIV-equilibrium point* (6)*, which will exist if* $\mathscr{R}_0 > 1$*, where* $\mathscr{R}_0$ *is the basic reproduction number of model* (2) *given in equation* (5)*.*

Next, we will analyze how $\mathscr{R}_0$ changes with respect to the change in other parameters. Since

$$\frac{\partial \mathscr{R}_0}{\partial \beta} = \frac{(\delta+\mu+u_2)(\mu+u_2)}{(\delta+\mu)(\delta+\mu+u_1+u_2)(\mu+u_1+u_2)} > 0,$$

we know that $\mathscr{R}_0$ will increase when β increases linearly since $\frac{\partial \mathscr{R}_0}{\partial \beta}$ does not depend on β. On the other hand, since

$$\frac{\partial \mathscr{R}_0}{\partial u_1} = -\frac{(\delta+\mu+u_2)(\mu+u_2)}{(\delta+\mu)(\delta+\mu+u_1+u_2)^2(\mu+u_1+u_2)}\left(\frac{1}{\delta+\mu+u_1+u_2}\frac{1}{\mu+u_1+u_2}\right) < 0,$$

we know that $\mathscr{R}_0$ will be suppressed non linearly when u_1 increases. Please note that when u_1 is large enough, $u_1 >> 0$, the effect of u_1 on suppressing $\mathscr{R}_0$ is no longer significant. Next, since

$$\begin{aligned}\frac{\partial \mathscr{R}_0}{\partial u_2} &= \frac{\beta(\mu+u_2)}{(\delta+\mu)(\delta+\mu+u_1+u_2)(\mu+u_1+u_2)}\left(1-\frac{\delta+\mu+u_2}{\delta+\mu+u_1+u_2}\right)\\ &+ \frac{(\delta+\mu+u_2)\beta}{(\delta+\mu)(\delta+\mu+u_1+u_2)(\mu+u_1+u_2)}\left(1-\frac{\mu+u_2}{\mu+u_1+u_2}\right) > 0,\end{aligned}$$

then the increasing number of people who return to the unaware sub-population will increase the basic reproduction number.

In analysis we have previously conducted, $\mathscr{R}_0$ determines the existence and local stability criteria of the equilibrium points Ω_1 and Ω_2. Therefore, next we will show how the health campaign interventions u_1 and u_2 determine the magnitude of $\mathscr{R}_0$, as shown in Figure 2. The analysis of Figure 2 is given as follows. In region 1, $u_1 \in [0, 0.033)$, the $\mathscr{R}_0$ will always be greater than one, regardless of the magnitude of health campaign intensity. Therefore, the intervention of the health campaign cannot effectively reduce the spread of HIV to the HIV-free equilibrium point in region 1. On the other hand, in region 3, when $u_1 \in [0.824, 1]$, the $\mathscr{R}_0 < 1$ will always be smaller than one for all magnitude of u_1 and u_2. This means that if $u_1 > 0.0824$, then the disease will always be extinct in the population. Interesting discussions appear in regions 2a and 2b, where the combination of u_1 and u_2 will determine the $\mathscr{R}_0$, i.e., whether it is greater or less than one. To achieve the persistence of HIV in the field ($\mathscr{R}_0 < 1$), for specific values of u_1, u_2 should fulfill the condition

$$\begin{aligned} u_2 &> \frac{1}{2}\frac{-\beta(\delta+2\mu)+(\delta+\mu)(\delta+2(\mu+u_1))}{\beta-\delta-\mu} \\ &+ \frac{1}{2}\frac{\sqrt{\beta^2\delta^2-2\beta\,\delta^3-2\beta\,\delta^2\mu+4\beta\,\delta\,{u_1}^2+4\beta\,\mu\,{u_1}^2+\delta^4+2\,\delta^3\mu+\delta^2\mu^2}}{\beta-\delta-\mu}, \end{aligned}$$

or in the numerical example using the same parameters for Figure 2 except u_1 and u_2, previous expression can be written as $u_2 > -0.22+0.538u_1+0.641\sqrt{2.016u_1^2+0.097}$. Therefore, if the government can conduct a health campaign u_1 in the region of $(0.033, 0.824)$, then they have to really consider the rate of u_2 to achieve the persistence of HIV.

Local stability of equilibrium points. Next, the local stability of HIV-free equilibrium point is investigated analytically, while the local stability for endemic-HIV-equilibrium is investigated numerically using specific values of $\mathscr{R}_0$. The local stability of the equilibrium point is determined by the eigenvalues of the Jacobian matrix at the corresponding equilibrium point of the system (2). The general form of the Jacobian matrix of model (2) can be written by:

$$\mathscr{J} = \left[\begin{array}{c|c} \mathscr{J}_{11} & \mathscr{J}_{12} \\ \hline \mathscr{J}_{21} & \mathscr{J}_{22}, \end{array}\right] \tag{9}$$

where $N_c = S_1 + S_2 + I_1 + I_2$ and

$$\mathscr{J}_{11} = \begin{bmatrix} -u_1 - \frac{\beta I_1}{N_c} + \frac{S_1 \beta I_1}{{N_c}^2} - \mu & u_2 + \frac{S_1 \beta I_1}{{N_c}^2} & -\frac{S_1 \beta}{N_c} + \frac{S_1 \beta I_1}{{N_c}^2} \\ u_1 & -\mu - u_2 & 0 \\ \frac{\beta I_1}{N_c} - \frac{S_1 \beta I_1}{{N_c}^2} & -\frac{S_1 \beta I_1}{{N_c}^2} & \frac{S_1 \beta}{N_c} - \frac{S_1 \beta I_1}{{N_c}^2} - u_1 - \delta - \mu \end{bmatrix},$$

$$\mathscr{J}_{12} = \begin{bmatrix} \frac{S_1 \beta I_1}{{N_c}^2} & 0 & 0 \\ 0 & 0 & 0 \\ -\frac{S_1 \beta I_1}{{N_c}^2} + u_2 & 0 & 0 \end{bmatrix}, \quad \mathscr{J}_{21} = \begin{bmatrix} 0 & 0 & u_1 \\ 0 & 0 & \delta \\ 0 & 0 & 0 \end{bmatrix},$$

$$\mathscr{J}_{22} = \begin{bmatrix} -\delta - \mu - u_2 & 0 & 0 \\ \delta & -\gamma - \mu & 0 \\ 0 & \gamma & -\alpha - \mu \end{bmatrix}.$$

To analyze the local stability of the HIV-free equilibrium point, we evaluate $\mathscr{J}$ in HIV-free equilibrium point which yield:

$$\mathscr{J}(\Omega_1) = \begin{bmatrix} -u_1 - \mu & u_2 & -\frac{\beta(\mu + u_2)}{\mu + u_2 + u_1} & 0 & 0 & 0 \\ u_1 & -\mu - u_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta \mu + \beta u_2}{\mu + u_2 + u_1} - \delta - \mu - u_1 & u_2 & 0 & 0 \\ 0 & 0 & u_1 & -\delta - \mu - u_2 & 0 & 0 \\ 0 & 0 & \delta & \delta & -\gamma - \mu & 0 \\ 0 & 0 & 0 & 0 & \gamma & -\alpha - \mu \end{bmatrix}.$$

The HIV-free equilibrium point will be stable if all the resulting eigenvalues from the above matrix are negative. We have four explicit eigenvalues, all of which are negative, i.e., $-\mu, -(\mu + u_1 + u_2), -(\alpha + \mu)$, and $-(\mu + \gamma)$. The other two eigenvalues come from the solution of the second order characteristic polynomial given by :

$$a_0 + a_1 \lambda + a_2 \lambda^2 = 0, \tag{10}$$

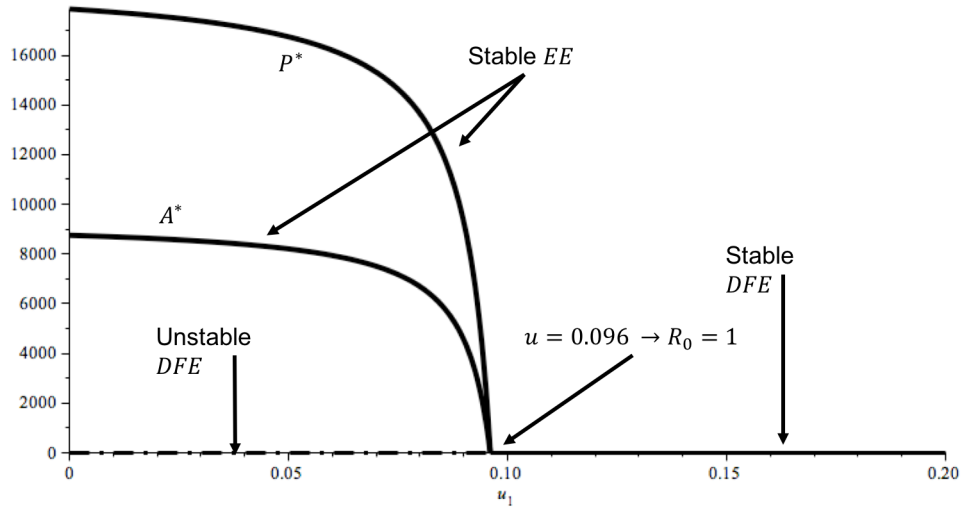
where

$$\begin{aligned} a_0 &= (1-\mathscr{R}_0)(\mu+u_1+u_2)(\mu+\delta)(\mu+\delta+u_1+u_2), \\ a_1 &= \left(1-\mathscr{R}_0\left(\frac{\mu+\delta}{\mu+\delta+u_2}\right)\left(\frac{\mu+\delta+u_1+u_2}{2\mu+2\delta+u_1+u_2}\right)\right)(\mu+u_1+u_2)(2\mu+2\delta+_1+u_2) \\ a_2 &= \mu+u_1+u_2. \end{aligned}$$

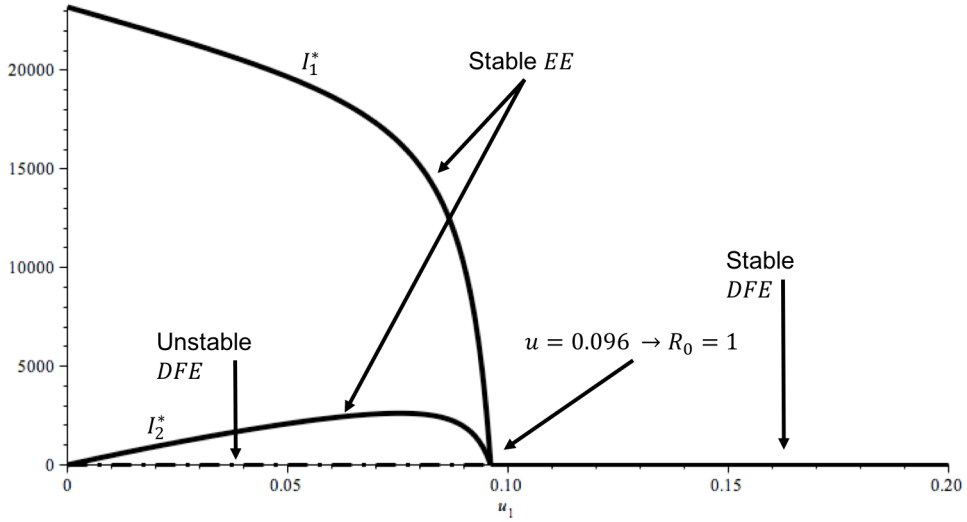
It can be seen that the polynomial in (10) will only have negative eigenvalues, if $\lambda_1\lambda_2 = \frac{a_0}{a_2} > 0 \iff a_0 > 0$ (if $\mathscr{R}_0 < 1$) and $\lambda_1+\lambda_2 = -\frac{a_1}{a_2} < 0 \iff a_1 > 0$ (always fulfilled when $a_0 > 0$). This result is given in the following theorem.

Theorem 2.2. *The HIV-free equilibrium* (Ω_1) *is locally asymptotically stable when* $\mathscr{R}_0 < 1$ *and unstable otherwise.*

Due to the complexity of the model and the form of endemic-HIV-equilibrium (Ω_2) of system (2), the local stability criteria of this equilibrium will be investigated by choosing a specific set of parameters, such that $\mathscr{R}_0 > 1$, i.e., $\Lambda = 200, \mu = 0.02, \beta = 0.6, \delta = 0.1, \gamma = 0.5, \alpha = 0.1, u_1 = 0.1$ and $u_2 = 0.05$.With this data set, we have that $\mathscr{R}_0 > 1$ which makes the HIV-free equilibrium point unstable (Theorem (2.1)), and the endemic equilibrium point exist (Theorem (2.2)). Using this set of parameters, we have the endemic equilibrium point given by $\Omega_2 = (1482, 2118, 672, 395, 205, 101)$, and when we evaluate it in the Jacobian matrix $\mathscr{J}$, the result gives us six eigenvalues, all of which are negative. Therefore, we conclude that there is a set of parameters, such that $\mathscr{R}_0 > 1$ that makes the endemic-HIV-equilibrium point Ω_2 locally stable. A forward bifurcation diagram of the equilibrium points depending on u_1 is given in Figure 3. It can be seen that the larger the magnitude of u_1 in the set of $u_1 \in [0, 0.096]$, the endemic equilibrium point Ω_2, is stable and the population size of I_1, P and A is decreasing and finally reaches 0 when u_1 tends to 0.096. On the other hand, I_2 in the endemic equilibrium is initially increasing, but later decreases to 0, when $u_1 \to 0.096$ is caused by the high intensity of government campaigns to educate people about HIV. When $u > 0.096$, the $\mathscr{R}_0$ is decreasing to less than one. According to the Theorem (2.1) and Theorem (2.2), the HIV-endemic equilibrium no longer exist, while the HIV-free equilibrium becomes stable.



(a)



(b)

FIGURE 3. Forward bifurcation of equilibrium points in system (1) depend on u_1.

3. OPTIMAL CONTROL CHARACTERIZATION

To investigate the optimal application of the health campaign needed to eliminate the spread of HIV, we would like to minimize the number of infected population using as low as possible control interventions. Therefore, let us consider the following objective functional

$$\mathbf{J}(\mathbf{U_j}, \mathbf{X_i}) = \int_0^T (\omega_1 I_1(t) + \omega_2 I_2(t) + \omega_3 P(t) + \omega_4 A(t) + \varphi_1 u_1^2(t) + \varphi_2 u_2^2(t))\, dt, \tag{11}$$

subject to model in system (1) where T is the final time of the simulation. Let ω_i for $i = 1,2,3,4$ represent the weight parameters for the infected population, while φ_1 and φ_2 represent the wight parameters for the control variables. Please note that $\omega_1 I_1(t), \omega_2 I_2(t), \omega_3 P(t)$ and $\omega_4 A(t)$, represent the related cost as a consequence of the high number of infected populations at time t, for example for hospitalization cost. On the other hand, $\varphi_1 u_1(t)$ and $\varphi_2 u_2(t)$ represent the cost related to the effort of the government to implement the health campaign in order to educate people to become aware about the spread of HIV. To guarantee the balance of the cost function, the condition of $\omega_i \mathbf{X_i} \approx \varphi_j \mathbf{U_j}$ should be reached, where $\mathbf{X_i}$ is the infected compartment, while $\mathbf{U_j}$ is the control variable. In this article, the use of quadratic form in the cost function $\mathbf{J}$ is chosen, since there is no linear correlation between the cost and effect of intervention. Please see [19, 21, 22] for further example of optimal control in the epidemiological model, using a quadratic form on their cost function.

We aim to minimize $\mathbf{J}$ with the constraint given by

$$\frac{dS_1(t)}{dt} = \Lambda - u_1(t)S_1(t) + u_2(t)S_2(t) - \frac{\beta S_1(t)I_1(t)}{N_c(t)} - \mu S_1(t) \tag{12a}$$

$$\frac{dS_2(t)}{dt} = u_1(t)S_1(t) - u_2(t)S_2(t) - \mu S_2(t) \tag{12b}$$

$$\frac{dI_1(t)}{dt} = \frac{\beta S_1(t)I_1(t)}{N_c(t)} - u_1(t)I_1(t) + u_2(t)I_2(t) - \delta I_1(t) - \mu I_1(t) \tag{12c}$$

$$\frac{dI_2(t)}{dt} = u_1(t)I_1(t) - u_2(t)I_2(t) - \delta I_2(t) - \mu I_2(t) \tag{12d}$$

$$\frac{dP(t)}{dt} = \delta\left(I_1(t) + I_2(t)\right) - (\gamma + \mu)P(t) \tag{12e}$$

$$\frac{dA(t)}{dt} = \gamma P(t) - (\mu + \alpha)A(t), \tag{12f}$$

with $N(t) = S_1(t) + S_2(t) + I_1(t) + I_2(t) + P(t) + A(t)$, and the initial conditions given as follows

$$S_1(0) = (S_1)_0,\ S_2(0) = (S_2)_0,\ I_1(0) = (I_1)_0,\ I_2(0) = (I_2)_0,\ P(0) = (P)_0,\ A(0) = (A)_0.$$

while also minimizing the control variables $u_1(t)$ and $u_2(t)$. We seek an optimal control u_1^*, u_2^*, such that

$$\mathbf{J}(u_1^*, u_2^*) = \min\left\{\mathbf{J}(u_1^*, u_2^*) | (u_1^*, u_2^*) \in \mathscr{A}\right\}$$

where $\mathscr{A}$ is the admissible control depending on the lower (u_i^{min}) and upper (u_i^{max}) bounds for each control variable. Please note that $u_i^{min} \geq 0$ and $u_i^{max} < \infty$. To guarantee the existence of the optimal control, it is directly followed by standard results of optimal control theory [23]. To derive the solution of our optimal control problem, we use the Pontryangin's Maximum Principle and also to derive the necessary conditions [24]. The Hamiltonian $\boldsymbol{H}$ is defined as

$$
\begin{aligned}
\boldsymbol{H} &= \varphi_1 u_1(t)^2 + \varphi_2 u_2(t)^2 + \omega_4 A(t) + \omega_3 P(t) + \omega_1 I_1(t) + \omega_2 I_2(t) \\
&+ \lambda_1(t)\left(\Lambda - S_1(t)u_1(t) + S_2(t)u_2(t) - \frac{S_1(t)\beta I_1(t)}{S_1(t)+S_2(t)+I_1(t)+I_2(t)} - \mu S_1(t)\right) \\
&+ \lambda_2(t)\left(-\mu S_2(t) + S_1(t)u_1(t) - S_2(t)u_2(t)\right) \\
&+ \lambda_3(t)\left(\frac{S_1(t)\beta I_1(t)}{S_1(t)+S_2(t)+I_1(t)+I_2(t)} - I_1(t)u_1(t) + I_2(t)u_2(t) - \delta I_1(t) - \mu I_1(t)\right) \\
&+ \lambda_4(t)\left(-\delta I_2(t) - \mu I_2(t) + I_1(t)u_1(t) - I_2(t)u_2(t)\right) \\
&+ \lambda_5(t)\left(-P(t)\gamma - P(t)\mu + \delta I_1(t) + \delta I_2(t)\right) \\
&+ \lambda_6(t)\left(-A(t)\alpha - A(t)\mu + P(t)\gamma\right).
\end{aligned} \tag{13}
$$

where $\boldsymbol{\lambda}_k(t)$ for $k = 1,2,...,6$ is the adjoint variables .

Theorem 3.1. *There exists an optimal control solution (u_1^*, u_2^*) that minimizes* **J** *over $\mathscr{A}$, given by*

(14)

$$
\begin{aligned}
\hat{u_1}(t) &= min\left(u_{\max(1)}, max\left(u_{\min(1)}, \frac{I_1(t)\lambda_3(t) - I_1(t)\lambda_4(t) + S_1(t)\lambda_1(t) - S_1(t)\lambda_2(t)}{2\varphi_1}\right)\right), \\
\hat{u_2}(t) &= min\left(u_{\max(2)}, max\left(u_{\min(2)}, \frac{-I_2(t)\lambda_3(t) + I_2(t)\lambda_4(t) - S_2(t)\lambda_1(t) + S_2(t)\lambda_2(t)}{2\varphi_2}\right)\right),
\end{aligned}
$$

where $\boldsymbol{\lambda}_k(t)$ for $k = 1,2,...,6$ is the adjoint variable for S_1, S_2, I_1, I_2, P and A, respectively, which satisfy

(15)

$$
\frac{d\lambda_1}{dt} = \left(u_1(t) + \frac{\beta I_1(t)}{S_1(t)+S_2(t)+I_1(t)+I_2(t)}\right)\lambda_1(t) - \left(\frac{S_1(t)\beta I_1(t)}{(S_1(t)+S_2(t)+I_1(t)+I_2(t))^2} - \mu\right)\lambda_1(t)
$$

$$
\begin{aligned}
&- u_1(t)\lambda_2(t) - \frac{\lambda_3(t)\beta I_1(t)\left(S_2(t)+I_1(t)+I_2(t)\right)}{\left(S_1(t)+S_2(t)+I_1(t)+I_2(t)\right)^2}\\
\frac{d\lambda_2}{dt} =& -\left(u_2(t) + \frac{S_1(t)\beta I_1(t)}{\left(S_1(t)+S_2(t)+I_1(t)+I_2(t)\right)^2}\right)\lambda_1(t) + \left(\mu + u_2(t)\right)\lambda_2(t)\\
&+ \frac{\lambda_3(t)S_1(t)\beta I_1(t)}{\left(S_1(t)+S_2(t)+I_1(t)+I_2(t)\right)^2}\\
\frac{d\lambda_3}{dt} =& -\omega_1 + \frac{S_1(t)\beta\left(S_1(t)+S_2(t)+I_2(t)\right)\lambda_1(t)}{\left(S_1(t)+S_2(t)+I_1(t)+I_2(t)\right)^2}\\
&- \lambda_3(t)\left(\frac{S_1(t)\beta}{S_1(t)+S_2(t)+I_1(t)+I_2(t)} - \frac{S_1(t)\beta I_1(t)}{\left(S_1(t)+S_2(t)+I_1(t)+I_2(t)\right)^2}\right)\\
&- \lambda_3\left(-u_1(t)-\delta-\mu\right) - u_1(t)\lambda_4(t) - \lambda_5(t)\delta\\
\frac{d\lambda_4}{dt} =& -\omega_2 - \frac{\lambda_1(t)S_1(t)\beta I_1(t)}{\left(S_1(t)+S_2(t)+I_1(t)+I_2(t)\right)^2} - \lambda_3(t)\left(-\frac{S_1(t)\beta I_1(t)}{\left(S_1(t)+S_2(t)+I_1(t)+I_2(t)\right)^2} + u_2(t)\right)\\
&+ \lambda_4(t)\left(\delta+\mu+u_2(t)\right) - \lambda_5(t)\delta\\
\frac{d\lambda_5}{dt} =& \left(\mu+\gamma\right)\lambda_5(t) - \lambda_6(t)\gamma - \omega_3\\
\frac{d\lambda_6}{dt} =& \lambda_6(t)\left(\alpha+\mu\right) - \omega_4
\end{aligned}
$$

and the transversality condition $\boldsymbol{\lambda}_i(T) = 0$.

Proof. First, to get the adjoint system (15), we differentiate the Hamiltonian $\boldsymbol{H}$ (13) with respect to each state variable

$$
\dot{\lambda}_1(t) = -\frac{\partial \mathscr{H}}{\partial S_1(t)}, \quad \dot{\lambda}_2(t) = -\frac{\partial \mathscr{H}}{\partial S_2(t)}, \quad \dot{\lambda}_3(t) = -\frac{\partial \mathscr{H}}{\partial I_1(t)},
$$

$$
\dot{\lambda}_4(t) = -\frac{\partial \mathscr{H}}{\partial I_2(t)}, \quad \dot{\lambda}_5(t) = -\frac{\partial \mathscr{H}}{\partial P(t)}, \quad \dot{\lambda}_6(t) = -\frac{\partial \mathscr{H}}{\partial A(t)},
$$

with the terminal condition $\boldsymbol{\lambda}_i(T) = 0$ for $i = 1,2,3,4,5,6$. To obtain the optimality condition (14), we will also differentiate the Hamiltonian in equation (13) with respect to control variables u_1 and u_2, which gives

$$
\frac{\partial \mathscr{H}}{\partial u_1(t)} = -I_1(t)\lambda_3(t) + I_1(t)\lambda_4(t) - S_1(t)\lambda_1(t) + S_1(t)\lambda_2(t) + 2u_1(t)\varphi_1 = 0,
$$

$$
\frac{\partial \mathscr{H}}{\partial u_2(t)} = I_2(t)\lambda_3(t) - I_2(t)\lambda_4(t) + S_2(t)\lambda_1(t) - S_2(t)\lambda_2(t) + 2u_2(t)\varphi_2 = 0,
$$

and set these equations equal to zero. Solving them with respect to each control variable, we obtain

$$u_1^*(t) = \frac{I_1(t)\lambda_3(t) - I_1(t)\lambda_4(t) + S_1(t)\lambda_1(t) - S_1(t)\lambda_2(t)}{2\varphi_1},$$

$$u_2^*(t) = \frac{-I_2(t)\lambda_3(t) + I_2(t)\lambda_4(t) - S_2(t)\lambda_1(t) + S_2(t)\lambda_2(t)}{2\varphi_2}.$$

To determine an acceptable control variable value based on the needs and ability in field applications (lower and upper bounds), the optimal control variables now are

$$\hat{u_1}(t) = \min\left(u_{\max(1)}, \max\left(u_{\min(1)}, \frac{I_1(t)\lambda_3(t) - I_1(t)\lambda_4(t) + S_1(t)\lambda_1(t) - S_1(t)\lambda_2(t)}{2\varphi_1}\right)\right),$$
$$\hat{u_2}(t) = \min\left(u_{\max(2)}, \max\left(u_{\min(2)}, \frac{-I_2(t)\lambda_3(t) + I_2(t)\lambda_4(t) - S_2(t)\lambda_1(t) + S_2(t)\lambda_2(t)}{2\varphi_2}\right)\right).$$

with $u_{\max(i)}$ and $u_{\min(i)}$ for $i = 1,2$ being the upper and lower bounds for each control variable, respectively. □

In the next section, numerical simulation for the autonomous model in system (2) and the optimal control problem explained in Theorem (3.1) will be conducted.

4. Numerical Results

4.1. Simulation of the autonomous model. We examined how the dynamics of HIV spread among the population, according to the changed values of some parameters in this section. To do this, all numerical simulations were carried out using the set of parameter values given in the following Table 2, except it is stated differently.

Parameter	Value	Parameter	Value
Λ	200	γ	0.5
μ	0.02	α	1
β	1.2	u_1	$(0,\infty)$
δ	0.4	u_2	$(0,\infty)$

TABLE 2. Numerical values used for simulation of the autonomous model in Fig. 4 and Fig. 5.

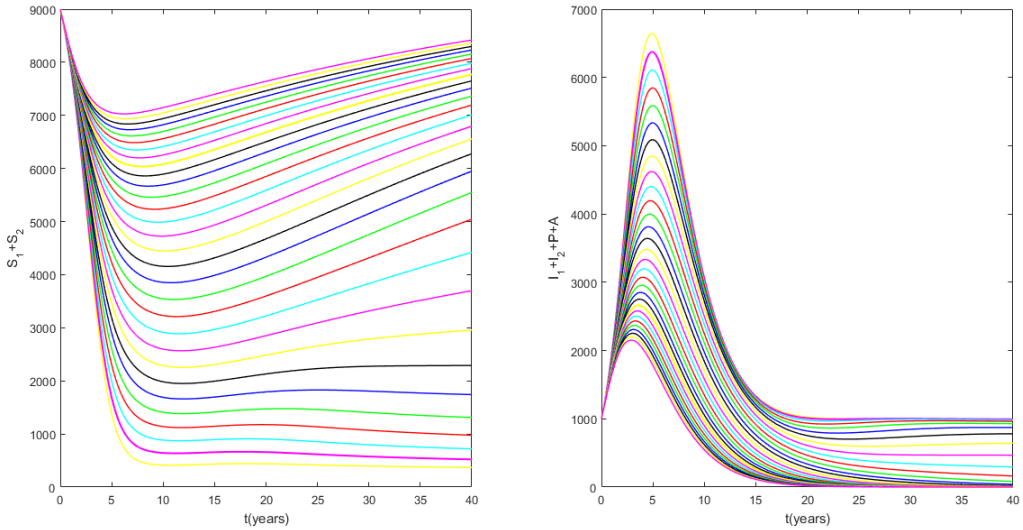


FIGURE 4. Dynamics of system (2) with respect to different values of $u_1 = 0.01k$, $k = 1,2,...,30$

Some parameters in Table 2 are given in intervals. This approach raises the question of how sensitive those parameters are in determining the results of the system (2). This is important to give a better understanding of the results of the optimal control problem in the next subsection. Thus, we have a sensitivity contour of parameters with respect to $\mathscr{R}_0$, before we gave the simulation of the autonomous model. In Figure 4 we can see how different values of $u_1 = 0.01\ k$, $k = 1,2,...,30$ affect the change of the dynamic of model (2). On the other hand, we can see how different values of $u_2 = 0.01\ k$, $k = 1,2,...,30$ affect the change of the dynamic of model (2) in Figure 5. Increasing the value of u_1 for u_2 as a constant, will increase total susceptible population $(S_1 + S_2)$ and suppress the total infected population $(I_1 + I_2 + P + A)$. On the other hand, for u_1 as a constant and u_2 increasing, it will increase the total infected population and decreasing the total susceptible population. The reason behind this is that larger values of u_2 make more people unaware of the risk of HIV, making them more vulnerable to infection by I_1, I_2, P or A individuals.

4.2. Simulation of the optimal control problem. In this section, we examine the optimal control problem of system (1) with the cost function given in (11). The optimal control set is obtained by solving the optimality system with a forward-backward scheme [16]. Please

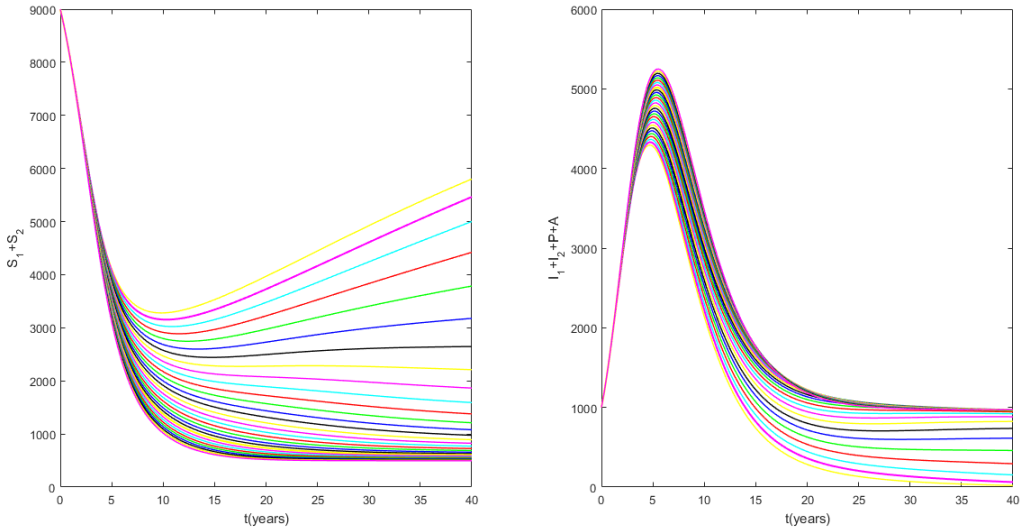


FIGURE 5. Dynamics of system (2) with respect to different values of $u_2 = 0.01k$, $k = 1, 2, ..., 30$

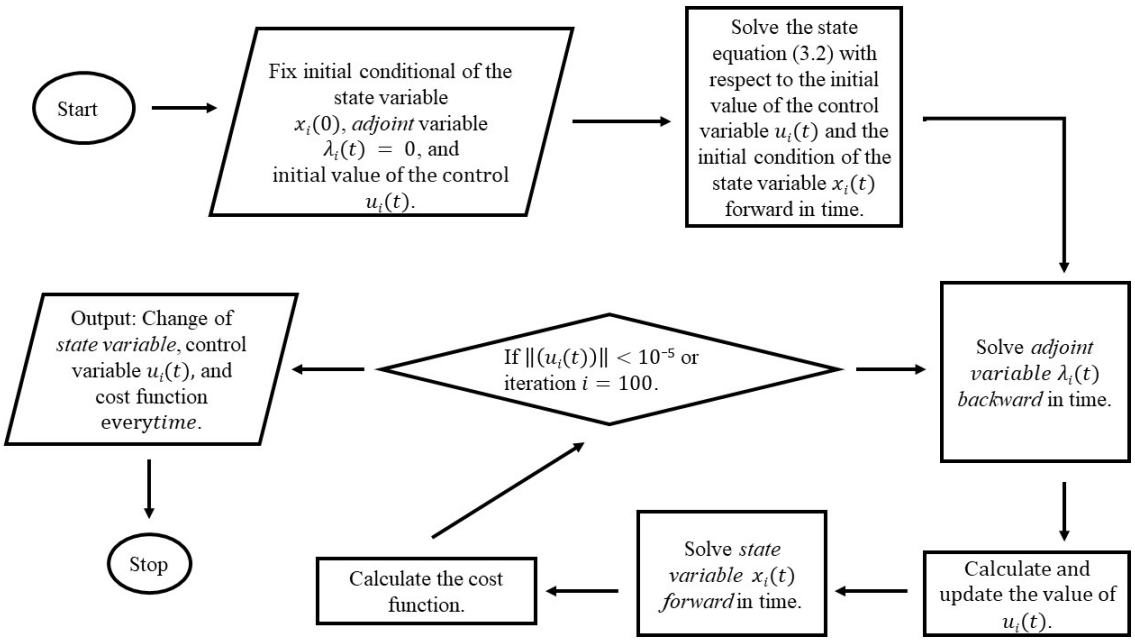


FIGURE 6. Flowchart diagram of a forward-backward method to solve the optimal control problem of system (1) with respect to cost function in (11).

see [17, 18, 19, 20] for further examples of implementation of this method in epidemiological models. The rough algorithm is given in flowchart in Figure 6.

To investigate the optimal control simulation, the numerical experiments will be conducted into four different scenario, i.e :

Scenario I: : Different initial $\mathscr{R}_0$.

Scenario II: : Different initial conditions of infected population.

Scenario III: : Different weight parameters of infected compartment ω_i.

Scenario IV: : Different weight parameters of control variables φ_i.

There are two different initial conditions used to conduct numerical simulations in this section. The first initial condition is when the number of infected individuals already relatively high, which named as the ”endemic reduction” scenario. The second initial condition is the ”endemic prevention” scenario, where the number of infected individuals is still relatively small in $t = 0$. Therefore, we have $(S_1, S_2, I_1, I_2, P, A) = (8000, 0, 2000, 0, 0, 0)$ for the endemic reduction scenario, and $(S_1, S_2, I_1, I_2, P, A) = (9800, 0, 200, 0, 0, 0)$ for the endemic prevention scenario.

Recapitulation of the numerical experiments related to the cost functions and equilibrium points are given in Table 3, before the control variables are implemented in Table 4, when the control variables are implemented into the system (1).

Scenario	Condition	**J**	Equilibrium Point ($u_1 = u_2 = 0$)							
			S_1	S_2	I_1	I_2	P	A	S_1+S_2	I_1+I_2+P+A
1	$\mathscr{R}_0 > 1$	286.3238	2009	2	380	0	293	143	2391	436
	$\mathscr{R}_0 < 1$	56.1561	9992	8	0	0	0	0	10000	0
2	Endemic Prevention	51.5710	2009	2	380	0	293	143	2391	436
	Endemic Reduction	137.9025	2009	2	380	0	293	143	2391	436
3	Small ω_i	65.2435	2009	2	380	0	293	143	2391	436
	Large ω_i	935.9736	2009	2	380	0	293	143	2391	436
4	Small φ_i	467.9888	2009	2	380	0	293	143	2391	436
	Large φ_i	130.4865	2009	2	380	0	293	143	2391	436

TABLE 3. Equilibrium points and cost functions for numerical results when no control variables are implemented into system (1).

Scenario	Condition	**J**	Equilibrium point ($u_1 \neq u_2 \neq 0$)							
			S_1	S_2	I_1	I_2	P	A	S_1+S_2	I_1+I_2+P+A
1	$\mathscr{R}_0 > 1$	286.3238	5613	2837	0	0	1	0	8450	1
	$\mathscr{R}_0 < 1$	56.1561	6342	1797	1	0	1	0	8139	2
2	Endemic Prevention	51.5710	5757	1565	134	23	115	59	7322	331
	Endemic Reduction	137.9025	3262	759	127	3	108	55	4021	293
3	Small ω_i	65.2435	3589	902	112	3	97	49	4491	261
	Large ω_i	935.9736	5486	3179	0	0	0	0	8665	0
4	Small φ_i	467.9888	5486	3179	0	0	0	0	8665	0
	Large φ_i	130.4865	3589	902	112	3	97	49	4491	261

TABLE 4. Equilibrium points and cost functions for numerical results when control variables are implemented into system (1).

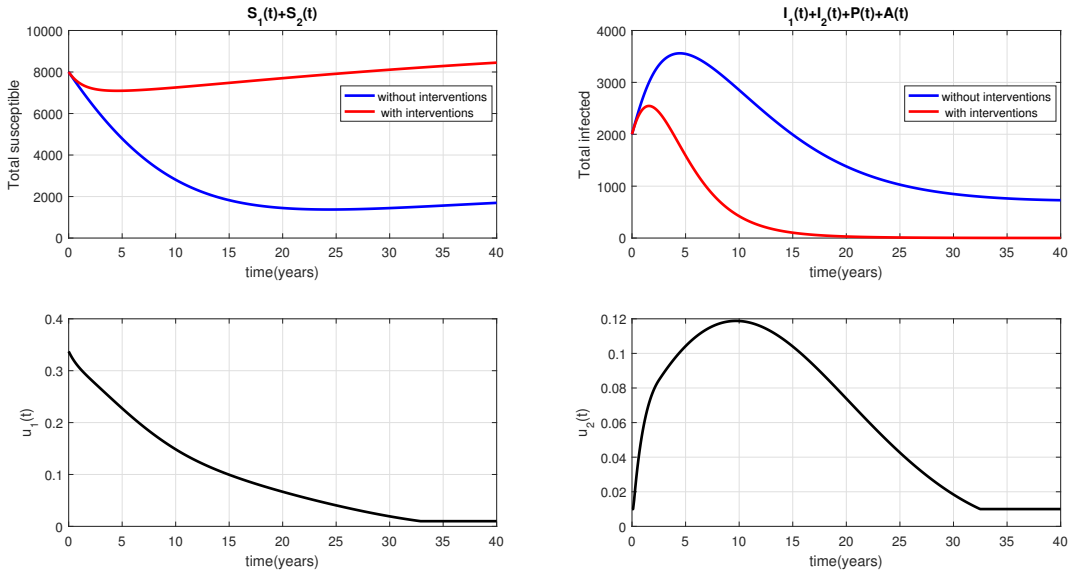
4.2.1. *Scenario I: Different initial $\mathscr{R}_0$.* The simulation in this chapter is provided to illustrate how environmental conditions (infectious rate, recovery rate and other factor that described in $\mathscr{R}_0$) affect the dynamics of control variables needed to suppress the spread of HIV. The simulation is given for two different $\mathscr{R}_0$ conditions at $t = 0$ when $u_1 = u_2 = 0$, but with the same initial value for each variable in system (1), i.e $\mathscr{R}_0 > 1$ and $\mathscr{R}_0 < 1$. According to the analytical results in Theorem (2.1) and (2.2), system (1) will tend to HIV-endemic equilibrium when $\mathscr{R}_0 > 1$ and goes to HIV-free equilibrium when $\mathscr{R}_0 < 1$. For this simulation, we use the set of parameters in Table 2 except $\beta = 0.5$ such that $\mathscr{R}_0 = 1.19 > 1$ and $\beta = 0.3$ such that $\mathscr{R}_0 = 0.71 < 1$. We use the "endemic reduction" scenario for the initial value in this section. We also bound the value of control variables, i.e $u_1 \in [0.01, 0.5]$ and $u_2 \in [0.01, 0.5]$. The result is given in Table 5, while the details for each compartment before and after implementation of controls can be seen in Table 3 and 4.

Scenario I	**J**	Total number of infected individual in $t = 40$ years	
		$u_1 = u_2 = 0$	$u_1 \neq u_2 \neq 0$
$\mathscr{R}_0 > 1$	286.3238	729	1
$\mathscr{R}_0 < 1$	56.1561	25	2

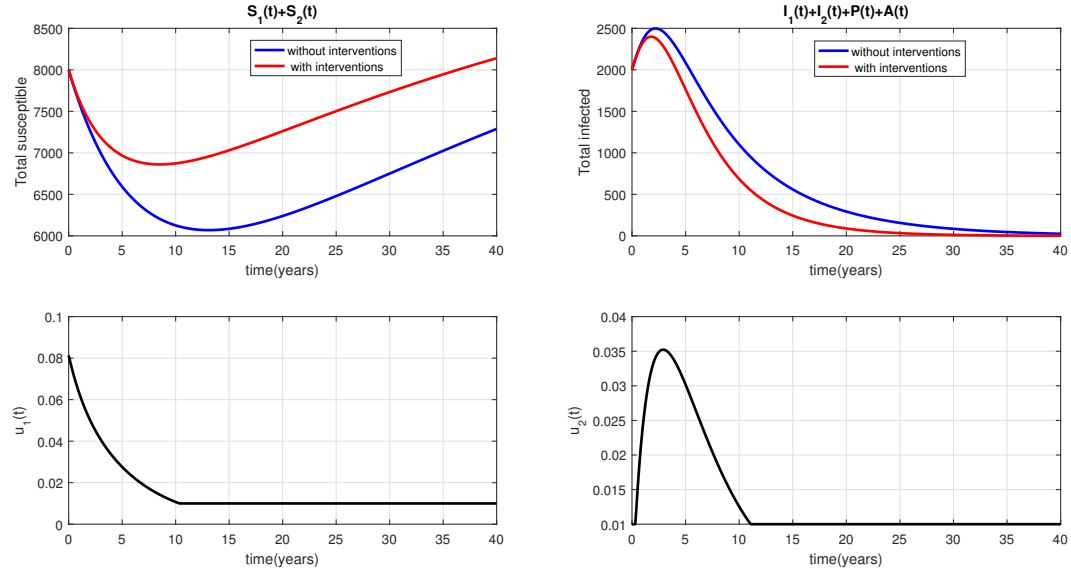
TABLE 5. Final condition of total susceptible and infected population in $t = 40$ for scenario I and the cost functions related to it.

Based on Table 5, it can be seen that the cost function, when $\mathscr{R}_0 > 1$, is almost five times higher than when $\mathscr{R}_0 < 1$. This result is not surprising, since the system will tend to endemic equilibrium when $\mathscr{R}_0 > 1$, i.e., $(S_1, S_2, I_2, I_2, P, A) = (2009, 2, 380, 0, 293, 143)$. Therefore, more intervention is needed to control the spread of HIV. After $t = 40$, the number of infected individuals could be suppressed to 1 when $\mathscr{R}_0 > 1$ and to 2 when $\mathscr{R}_0 < 1$. The dynamics of the total susceptible population, the total infected population, and control variables for each case of scenario I could be seen in Figure 7 .

In Figure 7, it can be seen that in both cases, the behavior of control variables is almost the same. $u_1(t)$ behaves monotonically decreasing for all $t \in [0, 40]$ while $u_2(t)$ is increasing,



(a)



(b)

FIGURE 7. The numerical result of scenario I for the dynamics of total susceptible, total infected and control variables. Figure (a) is the case when $\mathscr{R}_0 > 1$, while Figure (b) when $\mathscr{R}_0 < 1$.

in the beginning, to suppress the number of infected individuals and then decreasing when the dynamics of infected individuals show a decreasing trend. Even though the magnitude of controls when $\mathscr{R}_0 < 1$ is slightly higher than when $\mathscr{R}_0 > 1$, the cost function is higher when $\mathscr{R}_0 > 1$. This is because the number of infected individuals when $\mathscr{R}_0 > 1$ that should be reduced

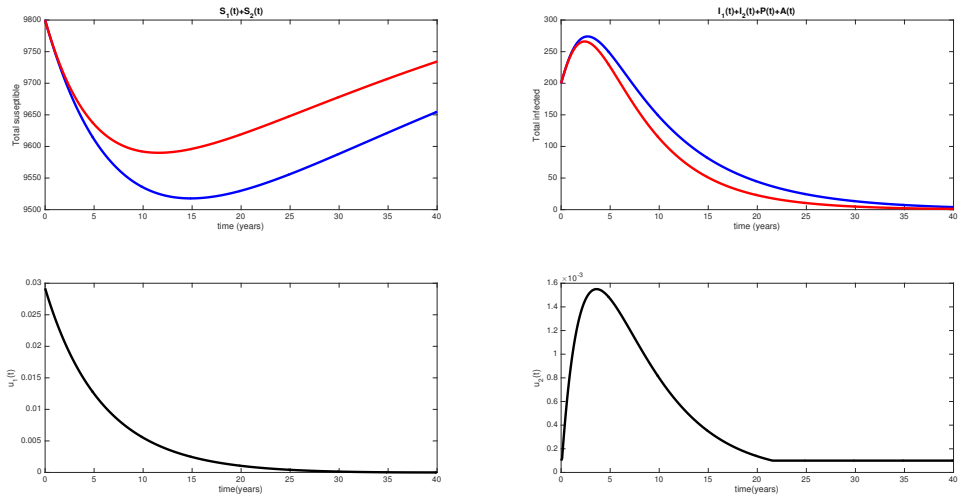
is much higher than when $\mathscr{R}_0 < 1$, since the number of infected individuals determines the cost function.

4.2.2. *Scenario II : Different initial conditions of infected population.* In this scenario, control variables u_1 and u_2 were used to optimize the cost function (11) for different initial conditions of each infected population as mentioned before in the early part of section (4.2) : endemic prevention and endemic reduction scenario. This simulation is important to illustrate the influence of alertness from the government in controlling the spread of HIV. As with the previous simulation in section 4.2.1 when $\mathscr{R}_0 > 1$, we use the set of parameters in Table 2, $u_1 \in [0.01, 0.5]$ and $u_2 \in [0.01, 0.5]$.

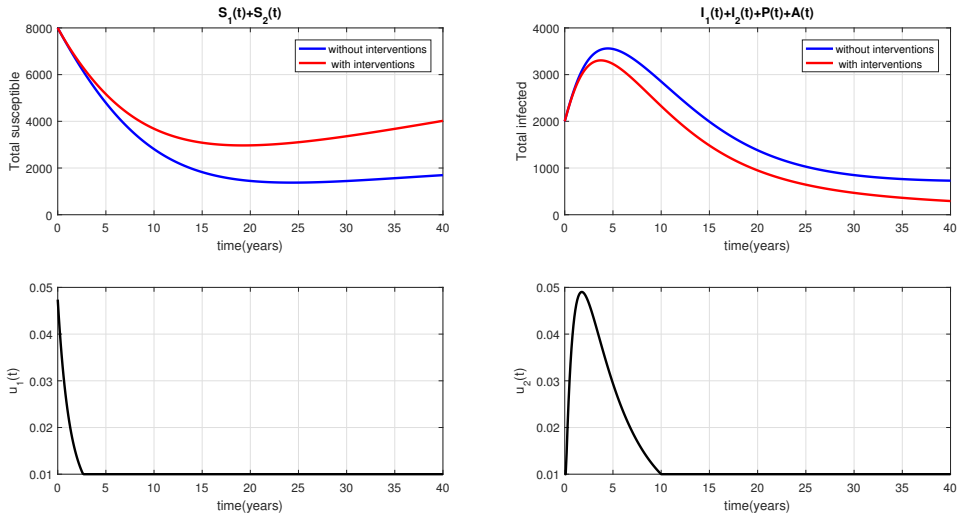
Scenario II	**J**	Total number of infected individual in $t = 40$ years	
		$u_1 = u_2 = 0$	$u_1 \neq u_2 \neq 0$
Endemic prevention	51.5710	1530	331
Endemic reduction	137.9025	729	293

TABLE 6. Final conditions of total susceptible and infection population in $t = 40$ for scenario II and the cost function related to it.

It can be seen from Table 6 that the cost function for endemic reduction is relatively large, compared to the endemic prevention case. This is simply because more intervention is needed to control the number of infected individuals in an endemic reduction case. The dynamics of the total susceptible population, total infected population, and control variables for scenario II are given in Figure 8. Although the dynamics of humans for both cases has no big difference, the extreme difference is shown in the dynamics of control variables. In the endemic reduction case, the dynamic of u_1 follows the dynamics of infected individuals. A huge amount of u_1 is needed when the number of infected individuals is increasing. Different from the endemic reduction case, the behavior of control variables in the endemic prevention case is almost always constant all the time. The value of $u_1 \in [0, 40]$ is the lower bound of u_1, which means that no major intervention is needed, which gave a small magnitude of the cost function.



(a)



(b)

FIGURE 8. The numerical results of scenario II for the dynamics of total susceptible, total infected and control variables. Figure (a) is the case for endemic prevention scenario, while Figure (b) is for the endemic reduction case.

4.2.3. *Scenario III: Different weight parameters of infected compartment* ω_i. We consider the different possibilities of the cost function as a consequence of the existence of infected individuals. As already mentioned before, $\int_0^T (\omega_1 I_1(t) + \omega_2 I_2(t) + \omega_3 P(t) + \omega_4 A(t))\, dt$ is related to a cost, which should be incurred as a consequence of the cost of hospitalization of infected humans, improvement of health service quality, etc. To do this simulation, we use different

value of ω_i i.e., $(\omega_1, \omega_2, \omega_3, \omega_4)$ i.e., (0.05,0.05,0.05,0.05) when the cost of hospitalization is low and (0.2,0.2,0.2,0.2) when the cost of hospitalization is high. Other parameters' values are the same with the previous simulation when $\mathscr{R}_0 > 1$.

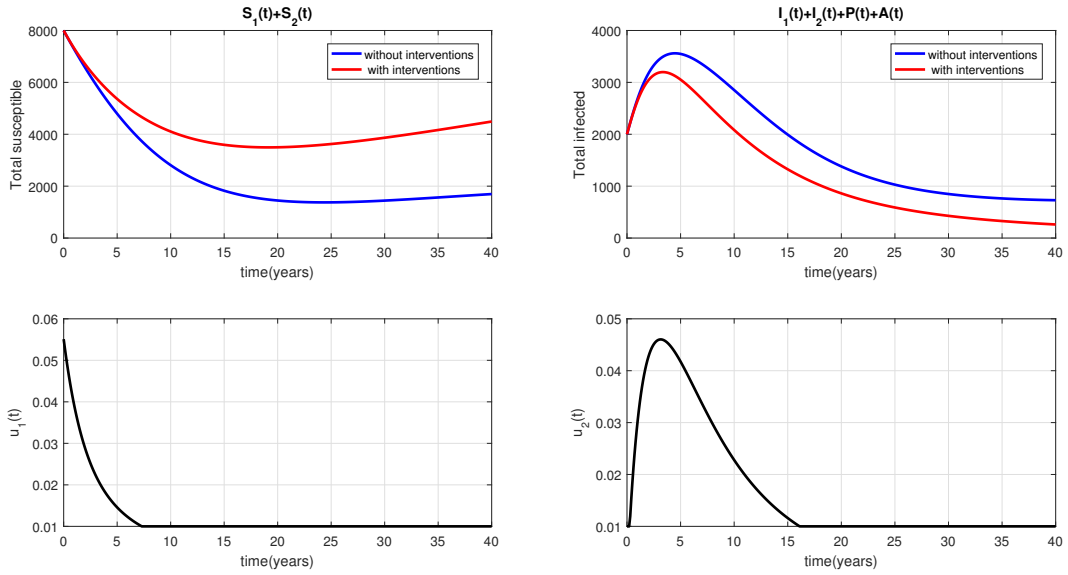
Scenario III	**J**	Total number of infected individuals in $t = 40$ years	
		$u_1 = u_2 = 0$	$u_1 \neq u_2 \neq 0$
small ω_i	65.2435	729	261
large ω_i	935.9736	729	0

TABLE 7. Final conditions of total susceptible and infected population in $t = 40$ for scenario III and the cost function related to it.

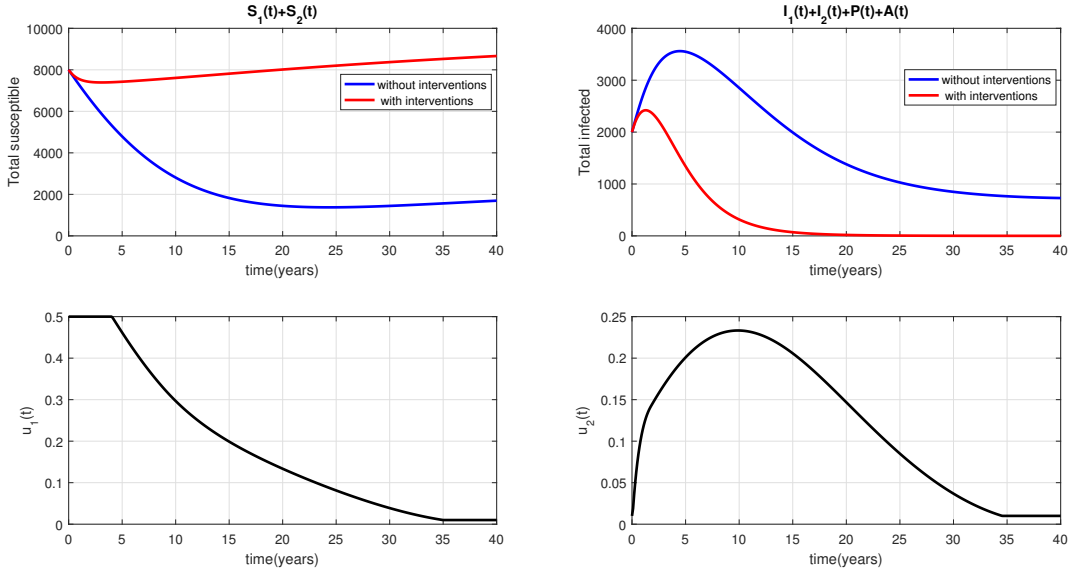
The dynamics of control and human population for this scenario can be seen in Figure 9, where the numerical result is given in Table 7. A large value of ω_i will increase the cost function. It can also be seen in Figure 9(b) that since the cost function for hospitalization is four times larger than in Figure 9(a), then the solution will be emphasized in the control function. Therefore, we can see that the dynamics of the control variable in Figure 9(b) is much larger than in Figure 9(a) to avoid the larger cost caused by hospitalization. Please note that the total number of infected humans in Figure 9(b) already tends to 0, when $t > 20$ years, while in Figure 9(a) it needs more than 40 years.

4.2.4. *Scenario IV : Different weight parameters of control variables* φ_i. In this scenario, the simulation is obtained to illustrate a condition when the unit cost of each control variable (φ_i) is different. We use $\varphi_1 = 7500, \varphi_2 = -1400$ to describe when the cost for health campaign is relatively low and $\varphi_1 = 30000, \varphi_2 = -560$ when the cost are relatively high. The other parameters are the same as with the simulation in section 4.2.1 when $\mathscr{R}_0 > 1$, and the initial condition is for the endemic reduction scenario.

The dynamics of controls and human population for the fourth scenario can be seen in Figure 10, where the numerical results regarding the cost function and total of infected population are given in Table 8. The huge value of weighted parameters for control variables will intervene with controls not in high numbers (Figure 10(b)) if compared with a situation when the weight



(a)



(b)

FIGURE 9. The numerical results of scenario III for the dynamics of total susceptible, total infected and control variables. Figure (a) is for the case when ω_i is relatively small while Figure (b) is when ω_i is relatively large.

parameters for control variables are smaller (Figure 10(a)). This situation will result in a large infected population. The cost function for the case when φ_i is relatively small is 467.9888,

Scenario IV	**J**	Total number of infected individual in $t = 40$ years	
		$u_1 = u_2 = 0$	$u_1 \neq u_2 \neq 0$
small φ_j	467.9888	729	0
large φ_j	130.4865	729	261

TABLE 8. Final conditions of total susceptible and infected population in $t = 40$ for scenario IV and the cost function related to it.

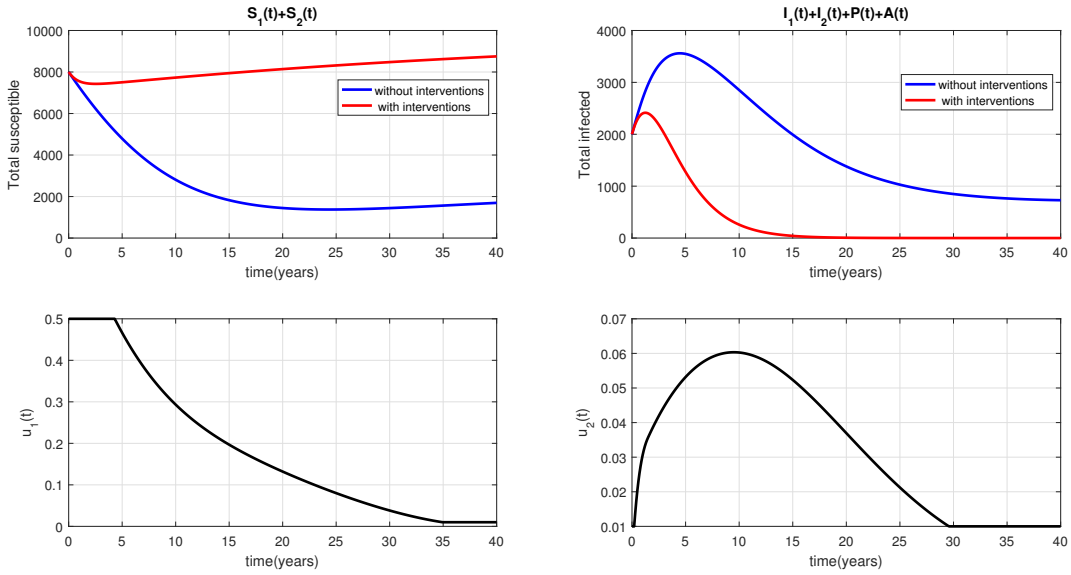
which is dominated by the cost for controls. On the other hand, the cost related to hospitalization is more dominant, when φ_i is relatively large, i.e., 130.4865. This is because the preferred solution is to spend all the resources on hospitalization, rather than on the expense of controls.

5. CONCLUSION

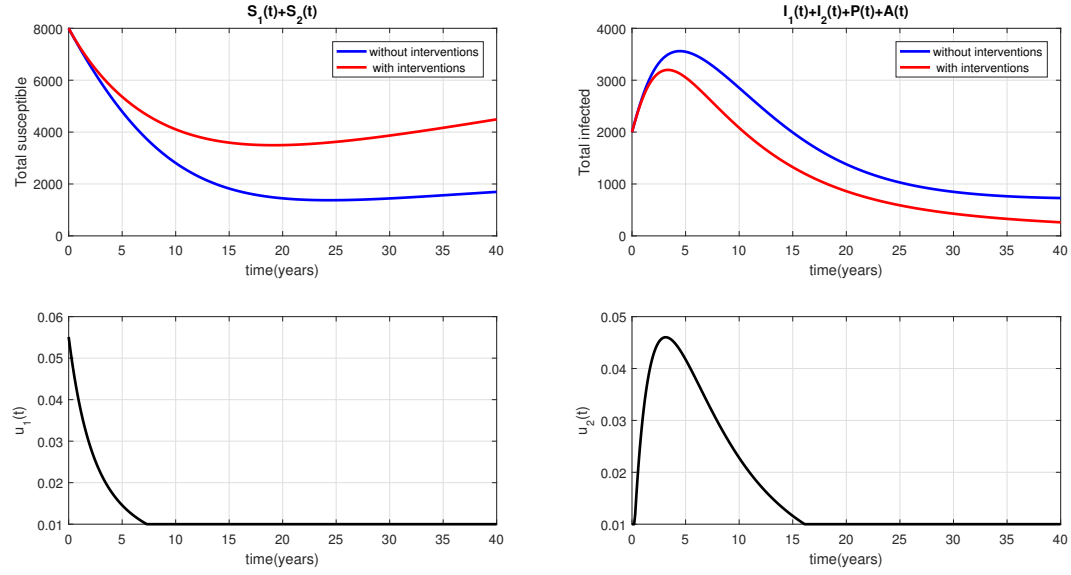
Mathematical modelling is an important tool to understand how a disease spreads and how many factors effect it. In this article, we have developed a mathematical model about the spread of HIV in the population, where medical campaigns are required to encourage the human population to become more aware about HIV. The model was constructed as a system of six dimensional ordinary differential equations with two control variables. This paper aims to find an optimal dynamics of control variables (medical campaign) to reduce the number of infected individuals to as small as possible. This task is designed as an optimal control problem.

Mathematical analyses concerning the equilibrium points and their local stability, along with the related basic reproduction numbers have been conducted analytically and numerically. We find that the HIV-free equilibrium point is locally stable when the basic reproduction number is smaller than one, and unstable otherwise. The instability of HIV-free equilibrium points then make the existence of the HIV-endemic equilibrium exist and locally stable. From analysis of the basic reproduction number with respect to the medical campaign parameters, we find that these controls are likely success to control the spread of HIV significantly.

The optimal control problem is solved with the Pontryagin Maximum/Minimum Principle (PMP). Four different numerical simulation scenarios are conducted to describe a possible situation that might occur in the field. We find that conducting the intervention in the early stage of



(a)



(b)

FIGURE 10. The numerical results of scenario IV for the dynamics of total susceptible, total infected and control variables. Figure (a) is when φ_j is relatively small, while Figure(b) is when φ_j is relatively large.

the endemic (endemic prevention scenario) can reduce the intervention cost significantly, rather than waiting for the endemic to already occur.

In this present study, we consider the medical campaign as the only intervention to control the spread of HIV. There are many more interventions that can be considered and modelled with mathematics, such as controlling the misuse of needles, mass campaign about digress sex behaviour, and many more. Also, many other factors should be considered to make the model become more realistic, such as the vertical transmission of HIV, impact of blood transfusion containing HIV, etc. Thus, we can consider more detailed models to accommodate these issues.

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CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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