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To cite this article: Marsudi *et al* 2019 *IOP Conf. Ser.: Mater. Sci. Eng.* **546** 052043

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Optimal Control of an HIV Model with Changing Behavior through an Education Campaign, Screening and Treatment

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Abstract. Optimal control theory was used on the system of differential equations to achieve the goal of minimizing the infected population and slow down the epidemic outbreak. Necessary conditions of optimal control problem were rigorously analysed using Pontryagin's maximum principle. Three control strategies were incorporated such as human education campaign, screening and treatment of infected human and its impact were graphically observed. Runge-Kutta forward-backward sweep numerical approximation method is used to solve the optimal control system. Numerical results with education campaign levels, screening and treatment rates as controls are illustrated.

1. Introduction

Mathematical modeling for epidemiological problems has become very important in the management and control of an epidemic of infectious diseases such as HIV/AIDS. Mathematical models based on the mechanism of the spread of HIV/AIDS can help medical or scientists understand and anticipate the spread of the epidemic and evaluate the potential effectiveness of different approaches to keep the epidemic under control. Reference [15] have examined the effect of screening on unaware infective in the spread of HIV infection and [13] examined the effects of screening and treatment on transmission of HIV/AIDS infection in a population. This model is more complex when compared with the model in [10] because there were additional treatment interventions in aware infectives and full blown AIDS. These three infected populations are moving towards AIDS at a constant rate. Reference [5] present a mathematical model that refers to [13] with the assumption that only screened infectives were treated to investigate the effects of screening and treatment of HIV on the HIV/AIDS infection dynamics in a population. This model does not consider (neglect) the progression of full blown AIDS to treated infections because it is assumed that transmission of HIV disease only through sexual contact (sexually transmitted diseases (STD)) and individuals in the full blown AIDS group undergo treatment.

Today, many HIV epidemiological problems are examined using control theory (see [2], [3], [4], [8], [9], [10], [12], and [14]). Qualitatively, optimal control analysis of the model is carried out and the necessary conditions for optimal disease control are determined using Pontryagin's Maximum Principle to determine the optimal strategy for controlling the spread of disease. Reference [3] reviewed the SIR type model expanded to include the use of education or information provided to the community as a control for managing disease outbreaks if effective treatments or vaccines were not available or too expensive and [2] reviewed the SIR epidemic model with an education and therapy campaign which are



two important interventions in disease management. In this paper, the model in [6] is extended by developing susceptible individuals into two groups which reverses to [2, 3], namely groups that get an education and behavior AB (Abstinence, Be faithful) or behavior C (Condom) with different levels of infectivity.

The paper is organized as follows: In Section 2, we derive a model consisting of ordinary differential equations that describes the interactions and the dynamics of the HIV disease with the underlying assumptions. In Section 3, the effective reproduction number R_e will be found by using the method of next generation matrix. In Section 4, we use Pontryagin's Maximum Principle to investigate optimal strategies and to find the necessary conditions for the optimal control of the disease. In Section 5, we show the simulation results and our conclusions are discussed in Section 6.

2. Model Formulation

The total human population ($N(t)$) was divided into seven subpopulations, namely: HIV negative individuals who did not have HIV infection or susceptible individuals ($S(t)$), susceptible who received an education campaign and the group following abstinence and faithful or AB behavior group ($S_1(t)$), susceptible who received an education campaign and the group using condom or C behavior group ($S_2(t)$), unaware infectives ($I_1(t)$), aware (screened) infectives ($I_2(t)$), full-blown AIDS class ($A(t)$) and screened infectives receiving therapy or treated class ($T(t)$).

The interaction between the classes will be assumed as follows: transmission rate (λ) proportional to the susceptible, and the ratio between the number of infected population (I_1 , I_2 and T) and the total population; unaware infectives can be screened infectives at rate θ ; only screened infectives can be therapy infectives at rate δ ; unaware infectives, screened infectives and treated infectives move to full-blown AIDS at different rates σ_1 , σ_2 and σ_3 respectively ($\sigma_3 < \sigma_2 < \sigma_1$); unaware infective, screened infectives and treated infectives can infect susceptible at different rates β_1 , β_2 and β_3 respectively ($\beta_3 < \beta_2 < \beta_1$); the AIDS-related dead rate γ , the natural mortality rate μ and recruitment into susceptible at a rate Λ . Because of the interactions of individuals in class S with the control, education E , a proportion of the susceptibles leave S and move to S_1 and S_2 at the rate α_1 and α_2 is $\alpha_i E S_i$, $i = 1, 2$. The education campaign at S_1 and S_2 has the effect of reducing the infection rate $(1 - \psi_1)\lambda$ and $(1 - \psi_2)\lambda$ respectively. Here ψ_i measures the efficacy of education campaign such that $0 \leq \psi_i \leq 1$, $i = 1, 2$. Our model consists of following seven differential equations

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \lambda S - E\alpha_1 S - E\alpha_2 S - \mu S \\ \frac{dS_1}{dt} &= E\alpha_1 S - \lambda_1 S_1 - \mu S_1 \\ \frac{dS_2}{dt} &= E\alpha_2 S - \lambda_2 S_2 - \mu S_2 \\ \frac{dI_1}{dt} &= \lambda S + \lambda_1 S_1 + \lambda_2 S_2 - (\theta + \sigma_1 + \mu)I_1 \\ \frac{dI_2}{dt} &= \theta I_1 - (\delta + \sigma_2 + \mu)I_2 \\ \frac{dT}{dt} &= \delta I_2 - (\sigma_3 + \mu)T \\ \frac{dA}{dt} &= \sigma_1 I_1 + \sigma_2 I_2 + \sigma_3 T - (\gamma + \mu)A \end{aligned} \quad (1)$$

where $\lambda = \frac{\beta_1 c_1 I_1 + \beta_2 c_2 I_2 + \beta_3 c_3 T}{N}$, $\lambda_1 = (1 - \psi_1)\lambda$, $\lambda_2 = (1 - \psi_2)\lambda$, $N = S + S_1 + S_2 + I_1 + I_2 + T + A$ with initial conditions

$$S(0) = S_0, S_1(0) = S_{10}, S_2(0) = S_{20}, I_1(0) = I_{10}, I_2(0) = I_{20}, T(0) = T_0, A(0) = A_0. \quad (2)$$

3. Model Analysis

Since the model system of equation (1) monitors changes in the human population, the variables and the parameters of model are assumed to be positive for all $t \geq 0$. The model will be analyzed in a suitable feasible region where all state variables are positive. Hence, all feasible solution of the system (1) enters the region

$$\Gamma = \left\{ \Omega = (S, S_1, S_2, I_1, I_2, T, A) \in R_7^+ \mid S > 0, S_1 > 0, S_2 > 0, I_1 \geq 0, I_2 \geq 0, T \geq 0, A \geq 0, N \leq \frac{\Lambda}{\mu} \right\}. \quad (3)$$

3.1. The disease-free equilibrium and the effective reproductive number

In order to obtain the disease-free equilibrium point (DFE) of the model system (1) the right-hand sides of the model equations is set to zero. Thus, the disease-free equilibrium point of the basic model (1) is given

$$E_0 = (S^*, S_1^*, S_2^*, I_1^*, I_2^*, T^*, A^*) = \left(\frac{\Lambda}{E\alpha_1 + E\alpha_2 + \mu}, \frac{E\alpha_1\Lambda}{\mu(E\alpha_1 + E\alpha_2 + \mu)}, \frac{E\alpha_2\Lambda}{\mu(E\alpha_1 + E\alpha_2 + \mu)}, 0, 0, 0, 0 \right) \quad (4)$$

The second of equilibrium point of the model system (1) is the endemic equilibrium point, $E_1 = (S^{**}, S_1^{**}, S_2^{**}, I_1^{**}, I_2^{**}, T^{**}, A^{**})$ which occurs if the disease is in the population ($I_1^* \neq 0, I_2^* \neq 0, T^* \neq 0$ and $A^* \neq 0$).

The local stability of E_0 was established by using the next generation matrix method on the system (1). The effective reproduction number R_e will be found by using the method of next generation matrix found in [19]. Hence, the effective reproduction number of the system (1) is given by

$$R_e = \frac{c_1\beta_1[(1-\psi_1)E\alpha_1 + (1-\psi_2)E\alpha_2 + \mu]}{(E\alpha_1 + E\alpha_2 + \mu)(\theta + \sigma_1 + \mu)} + \frac{c_2\beta_2\theta[(1-\psi_1)E\alpha_1 + (1-\psi_2)E\alpha_2 + \mu]}{(E\alpha_1 + E\alpha_2 + \mu)(\theta + \sigma_1 + \mu)(\delta + \sigma_2 + \mu)} + \frac{c_3\beta_3\theta\delta[(1-\psi_1)E\alpha_1 + (1-\psi_2)E\alpha_2 + \mu]}{(E\alpha_1 + E\alpha_2 + \mu)(\theta + \sigma_1 + \mu)(\delta + \sigma_2 + \mu)(\sigma_3 + \mu)}. \quad (5)$$

The effective reproduction number R_e measures the average number of new infections caused by a single HIV infected individual in a population where education campaign, screening, and therapy are used to control strategies are in place. Further, using Theorem 2 of [17], the following result is established.

Theorem 1. The DFE point of the model system (1), given by E_0 , is locally asymptotically stable (LAS) if $R_e < 1$ and unstable if $R_e > 1$.

3.2. Sensitivity analysis of model parameters

Initial disease transmission is directly related to the effective reproduction number. In These indices tell us how crucial each parameter is to disease transmission and discover parameters that have a high impact on R_e that should be targeted by intervention strategies. The sensitivity index of the effective reproduction number R_e to the parameters in the model was calculated using the approach of [1].

Definition 1 The normalized forward sensitivity index of R_e , that depends differentiably on the index on a parameter p , is defined as:

$$I_p^{R_e} = \frac{\partial R_e}{\partial p} \times \frac{p}{R_e}. \quad (6)$$

We derive an analytical expression for the sensitivity of R_e to each of the fourteenth different parameters involved in R_e described in Table 1 third column. Table 1 consists of parameter values for the sensitivity analysis that are arranged from most sensitive to the least (in order of magnitude). The specific interpretation of each parameter from Table 1 shows that, the most sensitive parameter is the contact rate of susceptible to unaware HIV infective β_1 and the least sensitive parameter is the progression rate of treated class to full-blown AIDS class.

Table 1. Sensitivity index of R_e

Parameter	Values (yr) ⁻¹	Sensitivity Index
$\beta_1 (c_1)$	0.86 (3)	+0.7870
θ	0.6	-0.4537
σ_1	0.2	-0.2222
μ	0.1	-0.1948
$\beta_3 (c_3)$	0.1 (1)	+0.1631
E	0.4	-0.0971
ψ_1	0.35	-0.0811
ψ_2	0.45	-0.0626
α_1	0.075	-0.0520
$\beta_2 (c_2)$	0.15 (2)	+0.0499
α_2	0.045	-0.0451
δ	0.99	-0.0286
σ_2	0.01	-0.0019
σ_3	0.001	-0.0016

The sensitivity index of R_e with respect to the contact rate of susceptible to unaware HIV infective (β_1) is +0.7870 that means, increasing (or decreasing) the parameter value β_1 by 10% , increases (or decreases) R_e by 7.87%. The sensitivity index of R_e with respect to the screening rate (θ) is -0.4537 that means, increasing (or decreasing) the parameter value the screening rate θ by 10% keeping other parameters constant, decreases (or increases) R_e by 4.537%.

4. Optimal Control Problem

We apply optimal control method using Pontryagin's Maximum Principle to determine the necessary conditions for the optimal control of the impact of changing behavior through an education campaign, screening and treatment on HIV transmission. We incorporate time-dependent controls into model (1) to determine the optimal strategy for controlling the disease. The basic model of HIV is generalized by incorporating three time-dependent controls education campaign ($u_1(t)$), screening of unaware infectives ($u_2(t)$), and treatment of aware infectives ($u_3(t)$). Hence, we have the following state equation,

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \lambda S - u_1 \alpha_1 S - u_2 \alpha_2 S - \mu S \\
\frac{dS_1}{dt} &= u_1 \alpha_1 S - \lambda_1 S_1 - \mu S_1 \\
\frac{dS_2}{dt} &= u_2 \alpha_2 S - \lambda_2 S_2 - \mu S_2 \\
\frac{dI_1}{dt} &= \lambda S + \lambda_1 S_1 + \lambda_2 S_2 - (u_2 + \sigma_1 + \mu) I_1 \\
\frac{dI_2}{dt} &= u_2 I_1 - (u_3 + \sigma_2 + \mu) I_2 \\
\frac{dT}{dt} &= u_3 I_2 + \delta_2 A - (\sigma_3 + \mu) T \\
\frac{dA}{dt} &= \sigma_1 I_1 + \sigma_2 I_2 + \sigma_3 T - (\gamma + \delta_2 + \mu) A
\end{aligned} \tag{7}$$

We want to find the optimal control values (u_1^*, u_2^*, u_3^*) that minimizes the objective functional $J(u_1, u_2, u_3)$ where

$$J(u_1, u_2, u_3) = \int_0^{t_f} [b_1 I_1 + \frac{1}{2} (w_1 u_1^2 + w_2 u_2^2 + w_3 u_3^2)] dt \tag{8}$$

where t_f stands for the final time to control HIV/AIDS. The constants $w_i \geq 0$ ($i=1, 2, 3$), are weights of the relative costs of the controls associated control u_1, u_2 , and u_3 , respectively, and the constant b_1 are a balancing cost factor for the unaware infected class I . The goal is to find the optimal control u_1^*, u_2^* , and u_3^* , such that

$$J(u_1^*, u_2^*, u_3^*) = \min \{J(u_1, u_2, u_3) \mid u_1, u_2, u_3 \in U\} \tag{9}$$

where $U = \{(u_1, u_2, u_3) \mid 0 \leq u_i \leq 1, i=1, 2, 3, \forall t \in [0, t_f]\}$ is the control set.

The necessary conditions for the existence of an optimal control triple (u_1, u_2, u_3) must satisfy come from the Pontryagin's Maximum Principle [12]. This principle convert equations (7) and (8) into a problem of minimizing pointwise a Hamiltonian H with respect to the controls (u_1, u_2, u_3) . We denote the right-hand side of the state system (7) by the vector $f = (f_1, f_2, f_3, f_4, f_5, f_6, f_7)$. Then the Hamiltonian function H associated with the problem (7)-(9) is defined as follows:

$$H = b_1 I_1 + \frac{1}{2} (w_1 u_1^2 + w_2 u_2^2 + w_3 u_3^2) + \sum_{i=1}^7 \lambda_i f_i \tag{10}$$

with $i = S, S_1, S_2, I_1, I_2, T, A$ and $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6$, and λ_7 are the adjoint variables or co-state variables related to state variables $(S, S_1, S_2, I_1, I_2, T, A)$. The system of adjoint equation is found by taking the appropriate partial derivatives of the Hamiltonian equation (10) with respect to the associated state and control variables.

Theorem 1. Given an optimal control triple (u_1^*, u_2^*, u_3^*) and solutions $(S^*, S_1^*, S_2^*, I_1^*, I_2^*, T, A)$ of the corresponding state system (7) that minimizes $J(u_1^*, u_2^*, u_3^*)$ over U . Then there are exists adjoint variables $\lambda_S, \lambda_{S_1}, \lambda_{S_2}, \lambda_{I_1}, \lambda_{I_2}, \lambda_T, \lambda_A$ satisfying

$$-\frac{d\lambda_i}{dt} = \frac{\partial H}{\partial i} \tag{11}$$

with transversality conditions

$$\lambda_i(T_f) = 0, \text{ where } i = S, S_1, S_2, I_1, I_2, T, A \tag{12}$$

and the controls u_1^* , u_2^* , and u_3^* satisfy the optimality conditions:

$$\begin{aligned} u_1^* &= \max \left\{ 0, \min \left(1, \frac{(\lambda_1 - \lambda_2)\alpha_1 S^* + (\lambda_1 - \lambda_3)\alpha_2 S^*}{w_1} \right) \right\}, \\ u_2^* &= \max \left\{ 0, \min \left(1, \frac{(\lambda_4 - \lambda_5)I_1^*}{w_2} \right) \right\}, \\ u_3^* &= \max \left\{ 0, \min \left(1, \frac{(\lambda_5 - \lambda_6)I_2^*}{w_3} \right) \right\}. \end{aligned} \quad (13)$$

Proof. The differential equations governing the adjoint variables are obtained by the differentiation of the Hamiltonian function (10) and evaluated at the optimal controls. Then the adjoint system can be written as,

$$\begin{aligned} \frac{d\lambda_S}{dt} &= -\frac{\partial H}{\partial S}, \quad \lambda_S(t_f) = 0, & \frac{d\lambda_{I_2}}{dt} &= -\frac{\partial H}{\partial I_2}, \quad \lambda_{I_2}(t_f) = 0, \\ \frac{d\lambda_{S_1}}{dt} &= -\frac{\partial H}{\partial S_1}, \quad \lambda_{S_1}(t_f) = 0, & \frac{d\lambda_T}{dt} &= -\frac{\partial H}{\partial T}, \quad \lambda_T(t_f) = 0, \\ \frac{d\lambda_{S_2}}{dt} &= -\frac{\partial H}{\partial S_2}, \quad \lambda_{S_2}(t_f) = 0, & \frac{d\lambda_A}{dt} &= -\frac{\partial H}{\partial A}, \quad \lambda_A(t_f) = 0, \\ \frac{d\lambda_{I_1}}{dt} &= -\frac{\partial H}{\partial I_1}, \quad \lambda_{I_1}(t_f) = 0, \end{aligned} \quad (14)$$

evaluated at the optimal controls and corresponding state variables, results in the stated adjoint system (11) and (12). Furthermore, differentiating the Hamiltonian gives function with respect to the control variables in the interior of control set U , we have $\frac{\partial H}{\partial u_i} = 0 \quad i = 1, 2, 3$. That is,

$$\begin{aligned} \frac{\partial H}{\partial u_1} &= w_1 u_1 - (\lambda_1 - \lambda_2)\alpha_1 S - (\lambda_1 - \lambda_3)\alpha_2 S = 0 \\ \frac{\partial H}{\partial u_2} &= w_2 u_2 - (\lambda_4 - \lambda_5)I_1 = 0 \\ \frac{\partial H}{\partial u_3} &= w_3 u_3 - (\lambda_5 - \lambda_6)I_2 = 0 \end{aligned} \quad (15)$$

Hence solving for u_1^* , u_2^* , and u_3^* , we get

$$u_1^* = \frac{(\lambda_1 - \lambda_2)\alpha_1 S^* + (\lambda_1 - \lambda_3)\alpha_2 S^*}{w_1}, \quad u_2^* = \frac{(\lambda_4 - \lambda_5)I_1^*}{w_2}, \quad u_3^* = \frac{(\lambda_5 - \lambda_6)I_2^*}{w_3}. \quad (16)$$

Use boundary conditions for $0 \leq u_i \leq 1$, $i = 1, 2, 3$ in the control, we obtained

$$\begin{aligned} u_1^* &= \max \left\{ 0, \min \left(1, \frac{(\lambda_1 - \lambda_2)\alpha_1 S^* + (\lambda_1 - \lambda_3)\alpha_2 S^*}{w_1} \right) \right\}, \\ u_2^* &= \max \left\{ 0, \min \left(1, \frac{(\lambda_4 - \lambda_5)I_1^*}{w_2} \right) \right\}, \\ u_3^* &= \max \left\{ 0, \min \left(1, \frac{(\lambda_5 - \lambda_6)I_2^*}{w_3} \right) \right\}. \end{aligned} \quad (17)$$

5. Numerical Simulation

In this section, numerically studied the impact of optimal control strategies using parameter values ([13]) and assumed the recruitment rate $\Lambda = 4000$ and induced death rate due to disease $\gamma = 0.9$. Some

parameter values were obtained from assumption. We simulate the model system by using ODE solver coded in Matlab program language by using the following initial conditions:

$$\begin{aligned} S(0) = 2.000.000, S_1(0) = 100.000, S_2(0) = 50.000, I_1(0) = 200.000, I_2(0) = 100.000, \\ T(0) = 10.000, A(0) = 5000. \end{aligned} \quad (18)$$

The solution to the optimal control problem is obtained by completing the optimality system from the adjoint system through the forward-backward Sweep method [4]. We start with an initial guess for the control and solve the state system forward in time. Using the new state values, the adjoint system is solved backward in time. The control is updated using a convex combination of the old control values and the new control values from the characterization. The iterative method is repeated until convergence. The adjoint system (equations system (14)) is solved by the Runge-Kutta scheme using advanced solutions from the state equation. Furthermore, in describing the control strategy the parameter values are used in (9) and weights at the final time ($t_f=20$),

$$b_1 = 1, w_1 = 25, w_2 = 55, w_3 = 75. \quad (19)$$

By using parameter values shown in Table 1 and assumption is obtained the effective reproduction numbers of the model system (1) is $R_e = 3.1848$. Because $R_e > 1$, the HIV/AIDS infection still exists within the population.

5.1. Numerical simulation of the optimal control problem

This section focuses on demonstrating our numerical results of optimal control problem (7)-(9) through an iterative method that examines the influence of education campaign (u_1), screening in unaware infectives (u_2), and treatment in aware infectives (u_3). We will consider the following combination of time-dependent controls making up four control strategies.

Strategy A: Implementation education campaign (u_1), screening (u_2) and treatment (u_3)

Figure 1(a) show that the combination of education campaign, screening on unaware infectives, and treatment on aware infectives resulted in a significant reduction in the number of unaware infectives, compared to cases without controls. Figure 1(b) shows the control profile of education campaign (u_1) is at the upper limit from the beginning to the time $t = 18.86$ years before dropping gradually periodically to the lower limit at the end time. The control profile of treatment (u_3) is at the upper bound till the time $t = 10.44$ years before dropped slowly to the lower limit at the end time while the control profile of screening (u_2) is at the upper bound of the beginning to the time $t = 19.97$ years before dropped gradually to the lower limit at the end time ($t_f = 20$).

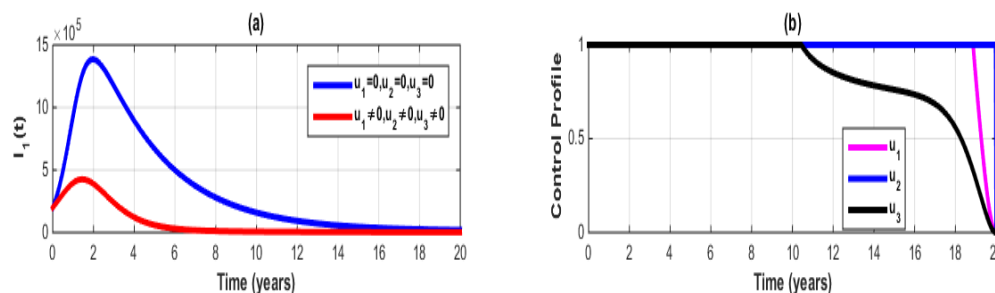


Figure 1. Simulation results of model with and without controls. (a) Fraction of unaware infectives; (b) The optimal control profiles of u_1 , u_2 and u_3

Strategy B: Implementation screening (u_2) and treatment (u_3)

Figure 2(a) show that the combination of screening on unaware infectives and treatment on aware infectives resulted in a significant reduction in the number of unaware infectives, compared to cases without controls. Figure 2(b) shows the control profile of screening on unaware infectives (u_2) is at the upper bound of the beginning to the time $t = 19.97$ years before dropped gradually to the lower

limit at the end time while the control profile of treatment (u_3) is at the upper bound till the time $t = 9.54$ years before dropped slowly to the lower bound to zero at the final time ($t_f = 20$).

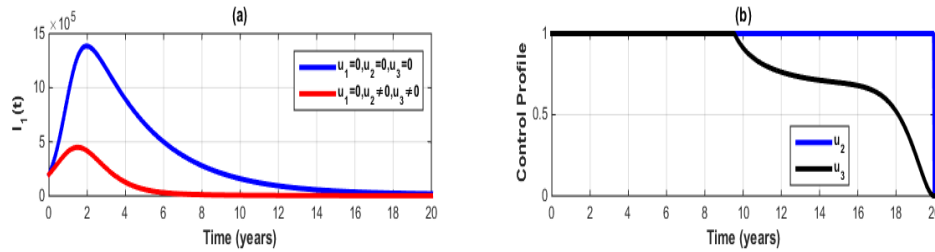


Figure 2. Simulation results of model with and without controls. (a) Fraction of unaware infectives; (b) The optimal control profiles of u_2 and u_3

Strategy C: Implementation education campaign (u_1) and screening (u_2)

In this strategy, the combination of education campaign (u_1) and screening on unaware infectives (u_2) resulted in a significant reduction in the number of unaware infectives, compared to cases without controls (Figure 3(a)). Figure 3(b) shows the control profile of screening on unaware infectives (u_2) is the same as in previous strategies while the control profile of education campaign (u_1) is at the upper bound till the time $t = 19.11$ years before dropped gradually to the lower bound to zero at the final time.

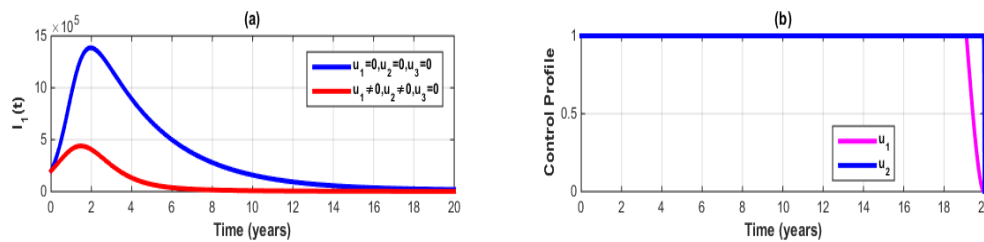


Figure 3. Simulation results of model with and without controls. (a) Fraction of unaware infectives; (b) The optimal control profiles of u_1 and u_2

Strategy D: Implementation education campaign (u_1) and treatment (u_3)

In this strategy, the combination of education campaign (u_1) and treatment (u_3) is shown in Figure 4(a) who did not get significant results between cases with control or without control. Figure 4(b) shows the control profile of treatment (u_3) is at the upper bound till the time $t = 1.75$ years before dropped slowly to the lower bound to zero at the final time while the control profile of screening on unaware infectives (u_2) is at the upper bound until $t = 5$ years then goes down at $u_1 = 9.46$ and increase at the time $t = 7.28$ years and at the upper bound until $t = 19.24$ years before dropped gradually to the lower bound to zero at the final time.

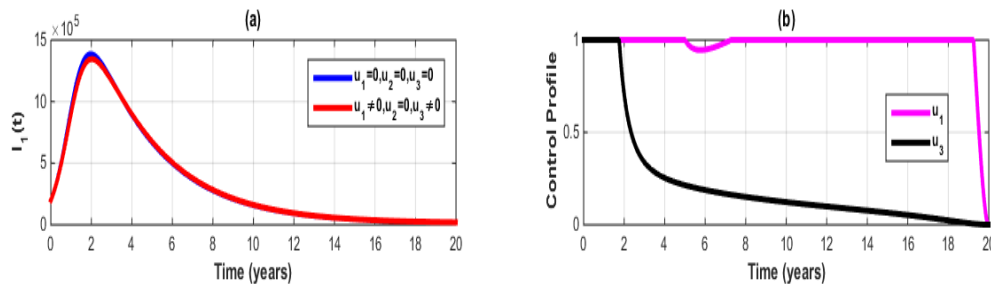


Figure 4. Simulation results of model with and without controls. (a) Fraction of unaware infectives; (b) The optimal control profiles of u_1 and u_3

5.2. Effects of the weight factors on the control profile

Figure 5 (a)-(c) shows a simulation that shows the impact of different weight factors in the control on the spread of disease. From the control profiles (Figure 5(a) and 5(c)), we observe that the weight factors values w_1 and w_3 in the control strategy have a significant impact in determining the amount of efforts and the time period required for intervention. That is, the smaller the weight values w_1 and w_3 the longer the intervention period (effort time) required to effectively control the disease. The weight factors values w_2 in the control strategy did not have a significant impact in determining the amount of efforts and the time period required for intervention (Figure 5(b)).

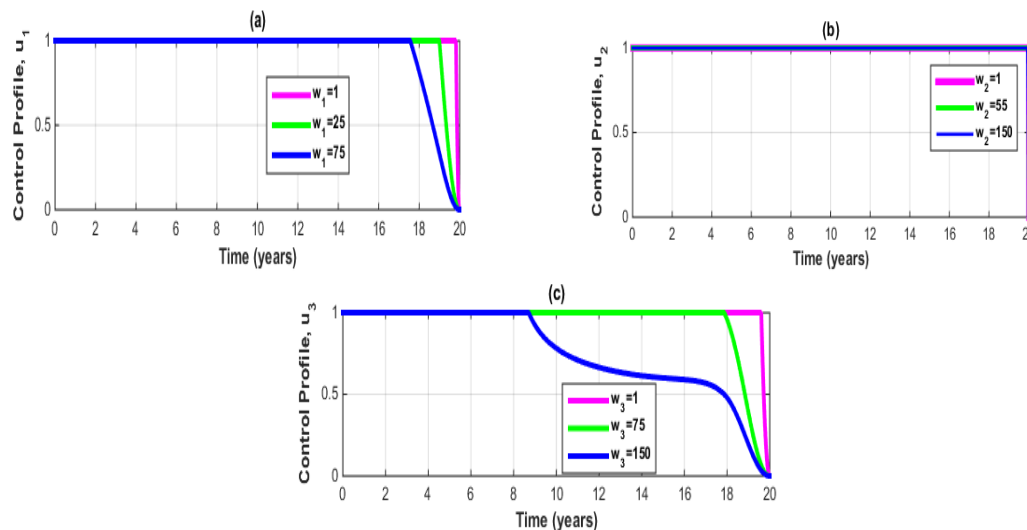


Figure 5. Simulations showing the impacts of different weight factors on the disease spread

6. Conclusion

We presented a deterministic model for assessing the effect of the education campaign on susceptible, screening on unaware infectives, and treatment on aware infectives in the spread of HIV in the population. The analysis shows that the education campaign of susceptible, the screening of unaware HIV infective and treatment of screened HIV infective have the effect of reducing the transmission of the disease. A sensitivity analysis shows that the contact rate of susceptible to unaware HIV infective is the most sensitive parameter on the effective reproduction number and the least sensitive parameter is the progression rate of treated class to full-blown AIDS. It is observed that the education campaign, the screened infective, and therapy infective participate in the transmission of the infection, the unaware infectives population is significantly reduced in comparison to the case where there is no education campaign, screening and treatment.

We introduced three time-dependent control variables into the model and investigated the effect of different optimal control strategies on the spread of disease in a population. Pontryagin's maximum principle is used to derive and analyze the necessary conditions for optimal control strategies: education campaigns (u_1), screening in unaware infectives (u_2), and treatment in aware infectives (u_3) to minimize the spread of HIV. The optimal control analysis shows that strategy B (optimal screening of unaware infectives and treatment of aware infectives) and strategy A (optimal education campaign of susceptible, optimal screening of unaware infectives, and optimal treatment of aware infectives) are control strategy get more significantly result in reducing the number of infected population (unaware infectives). We suggest that where resources are limited, strategy B, is preferred over strategy A, because the implementation of the A strategy will require additional costs due to the presence of education campaign of susceptibles.

Acknowledgments

This work was part of a dissertation study on the doctoral program in mathematics at the University of Brawijaya, Malang, Indonesia.

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