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Commun. Math. Biol. Neurosci. 2020, 2020:74

<https://doi.org/10.28919/cmbn/4986>

ISSN: 2052-2541

OPTIMAL CONTROLS ANALYSIS OF HIV/AIDS TRANSMISSION MODEL WITH AN AWARE POPULATION

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Abstract: In this paper, a mathematical model that describes the transmission dynamics of HIV/AIDS with an awareness effect is studied. We analyze the existence and stability of the model equilibriums of HIV/AIDS based on the basic reproduction number. Parameter sensitivity analysis was also conducted to determine the most influential parameters on the spread of this disease. Furthermore, we apply the optimal controls on the HIV model in the form of prevention, campaign, and treatment of Antiretroviral Therapy (ART). The optimal control problems are solved using Pontryagin's Maximum Principle. Numerical simulation results show that the combination of prevention and campaign effort can effectively minimize the number of human populations infected with HIV/AIDS.

Keywords: HIV/AIDS; mathematical model; sensitivity analysis; optimal control; awareness effect.

2010 AMS Subject Classification: 37N25, 93C15, 93D20.

1. INTRODUCTION

Human Immunodeficiency Virus (HIV) is a virus that attacks/infects cells in the immune system that causes a decrease in body immunity. HIV infection continues to be a grave health problem for

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Received September 1, 2020

the worldwide community, which has infected more than 37.9 million folks [1]. HIV will transmit through blood, semen, cervical or vaginal secretions, and breast milk that may transmit HIV from the mother to her baby. Acquired Immunodeficiency Syndrome (AIDS) is the late stage of HIV infection that happens once the virus is badly damaged the immune system of the patient's body.

Infectious diseases have an enormous impact on society as a result of a quarter of all deaths within the world every year are caused by transmittable diseases [2]. Several factors similar to coverage and mass media, vaccinations, population migration, and behavior changes have a significant influence on the transmission of the disease and its prevention policies [3]. Currently, there's no drug or vaccine which will cure HIV. However, there are antiretroviral therapy (ART) treatments that will improve the quality of life, prolong life, and scale back the chance of HIV transmission. Life expectancy in HIV patients who receive optimum treatment is also near to the life expectancy of uninfected populations [4]. In such things, the media also can be used as a way to convey health messages that have the potential to influence behavior and encourage folks to require preventative actions about epidemics [5]. If people already perceive the disease and aware of that, then it's expected that the spread of the disease can decrease. In developing and underdeveloped countries, mass media campaigns will play a crucial role in ever-changing these behaviors. For HIV control, two primary items that will stop HIV are implementing healthy sexual activities for vulnerable people and consciously not participating in sexual activities for HIV patients [6]. Each of those actions relies on the speedy dissemination of knowledge to facilitate accurate initial identification of HIV symptoms.

From 2000 to 2018, new HIV infections fell by 37% also HIV-related deaths fell by 45%, with 13.6 million lives saved owing to ART. By the end of 2018, an estimated 23.3 million folks accessed ART treatment. If a lot of and more countries adopt new tips from WHO to treat all individuals diagnosed with HIV directly, this public health advantage will be work out for people and the wider community [7].

The right mathematical model and compartment model have a crucial role in understanding the dynamics of an outbreak, including the spread of HIV/AIDS. Various studies have been conducted

to examine the spread of HIV/ AIDS with a mathematical model approach. Silva and Torres [8] have analyzed the global stability model concerning the spread of HIV/AIDS that refers to the case in Cape Verde 2015. Ghosh et al. [9] investigate the interaction between the escalate of awareness through the media and also the effects of psychological fear because of self-induced behavior changes within the spread of HIV/AIDS. The authors in [10] have formulated a fractional model of HIV transmission with awareness effect and used the data of infection case of HIV in Indonesia from 2006 until 2018. Besides, many mathematical models of HIV/AIDS transmission dynamics are developed by researchers with incorporate optimal control [11,12,13].

In this present paper, we extended the dynamics of the HIV/AIDS outbreak with an aware population using an ordinary differential system based on the model in [10]. We conjointly explore the impact of the optimal control strategy in reducing infectious HIV/AIDS populations. These controls are preventive measures using a contraceptive method, effective media campaign, and treatment of ART for the infected population on the spread of the disease. The work is put in order as follows. In section 2, we discuss model formulation and analysis of the model. In sections 3, the sensitivity analysis of the model parameters is discussed. Sections 4 explains the application of the optimal control problem. Sections 5 contains the numerical result to illustrate the results of the dynamics and the conclusion is summarized in sections 6.

2. MATHEMATICAL MODEL

This section will discuss the mathematical model of the spread of HIV/AIDS in the presence of an aware population. The basic model used refers to [10]. The model is divisible into five populations (compartments), including S_u is the number of unaware susceptible individuals. Compartment S_a is the number of aware susceptible individuals. Compartment I is the number of individuals infected with HIV that infectious. Compartment C is the number of individuals infected with HIV/AIDS (in chronic stage) and is undergoing treatment (ART). Compartment A is the number of individuals infected with HIV and has clinical symptoms of AIDS. The assumptions used in this mathematical model are as follows:

1. The population S_u can move to S_a populations, but not vice versa.
2. The population C and A are assumed not to spread HIV because of their condition.
3. The population of HIV and AIDS sufferers is considered to be able to access ART Treatment.
4. The population A who starts ART treatment will enter the HIV-infected class (I) and will get into population C if they continue to follow the therapy routinely.
5. The death rate due to HIV/AIDS is only experienced in populations affected by AIDS.

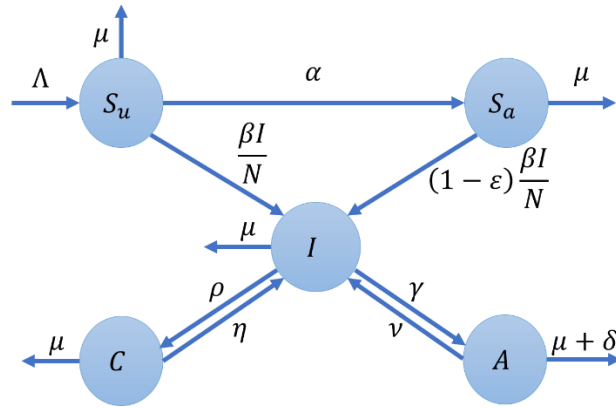


FIGURE 1. Transmission diagram of HIV/AIDS model with an aware population

Based on the Figure 1, the mathematical model of HIV/AIDS transmission with an aware population is as follows.

$$(1) \quad \begin{cases} \frac{dS_u}{dt} = \Lambda - \left(\alpha + \frac{\beta I}{N} + \mu \right) S_u, \\ \frac{dS_a}{dt} = \alpha S_u - \left((1 - \varepsilon) \frac{\beta I}{N} + \mu \right) S_a, \\ \frac{dI}{dt} = \frac{\beta I}{N} (S_u + (1 - \varepsilon) S_a) + \eta C + \nu A - (\rho + \gamma + \mu) I, \\ \frac{dC}{dt} = \rho I - (\eta + \mu) C, \\ \frac{dA}{dt} = \gamma I - (\nu + \delta + \mu) A. \end{cases}$$

Defining of the variables and parameters used in model (1) is shown in Table 1 and 2 below.

TABLE 1. Defining of the variable in HIV/AIDS model (1)

Variable	Description
$S_u(t)$	The number of unaware susceptible populations at the time t .
$S_a(t)$	The number of aware susceptible populations at the time t .
$I(t)$	The number of HIV-infected population without clinical symptoms of AIDS at the time t .
$C(t)$	The number of populations infected with HIV/AIDS (at a chronic stage) and currently on ART treatment with a low amount of virus in the blood at the time t .
$A(t)$	The number of HIV-infected population with clinical symptoms of AIDS at the time t .

TABLE 2. Defining of the parameters in HIV/AIDS model (1)

Notation	Description	Unit
Λ	Natural birth rate	people/time
α	The rate of change from being unaware to aware	1/time
β	The rate of HIV transmission	1/time
μ	Natural death rate	1/time
γ	The rate of change from HIV to AIDS	1/time
ν	HIV treatment rates for A population	1/time
η	HIV treatment failure rates for C population	1/time
ρ	HIV treatment rates for I population	1/time
δ	Death rate due to AIDS	1/time
ε	Relative infection rate of S_a population to S_u population	

Furthermore, for reasons of simplicity, $S_u(t), S_a(t), I(t), C(t), A(t)$ written into S_u, S_a, I, C, A , with $S_u, S_a, I, C, A \geq 0$. Then defined $N(t)$ as total population at t , with $N = S_u + S_a + I + C + A \geq 0$. Afterwards, all parameters that have been defined are positive, with $\Lambda > 0$ and $0 < \alpha, \beta, \mu, \gamma, \delta, \eta, \nu, \rho, \varepsilon < 1$.

The disease-free equilibrium of HIV/AIDS is a condition where no spread of HIV/AIDS. This equilibrium attainable when no human is infected with HIV/AIDS, both of which have no clinical symptoms of AIDS or existing ($I = A = 0$). In addition, due to the absence of humans infected with HIV/AIDS, there is also no human being receiving treatment ($C = 0$). In model (1) a disease-free equilibrium is obtained by

$$(2) \quad E^0 = (S_u^0, S_a^0, I^0, C^0, A^0) = \left(\frac{\Lambda}{\mu + \alpha}, \frac{\alpha \Lambda}{\mu(\mu + \alpha)}, 0, 0, 0 \right).$$

Then the basic reproduction number (R_0) will be determined, which represents the expectation of the average number of new HIV infections due to contact between HIV patients and vulnerable individuals. In this study, we used the Next Generation Matrix (NGM) method to get R_0 which has been developed by [14] to obtain

$$(3) \quad R_0 = \frac{\beta(\mu + (1 - \varepsilon)\alpha)(\mu + \delta + \nu)(\mu + \eta)}{(\mu + \alpha)(\mu[(\mu + \eta)(\gamma + \mu + \delta + \nu) + \rho(\mu + \delta + \nu) + \gamma\delta] + \eta\gamma\delta)}.$$

The disease-free equilibrium E^0 will be locally asymptotically stable if $R_0 < 1$ and will be unstable when $R_0 > 1$.

The endemic equilibrium is the condition that there is an HIV/AIDS patient, as well as the spread of the disease, occurs. Endemic equilibrium $E^* = (S_u^*, S_a^*, I^*, C^*, A^*)$ is obtained when at least one of I^*, C^* or A^* is not equal to zero. Equation system (1) can be solved using the condition of the force of infection at steady-state (κ^*), with

$$(4) \quad \kappa^* = \frac{\beta I^*}{N^*}.$$

Setting the right-hand sides of the model (1) to zero and noting that $\kappa = \kappa^*$ at equilibrium gives

$$(5) \quad \begin{cases} S_u^* = \frac{\Lambda}{(\kappa^* + \mu + \alpha)}, \\ S_a^* = \frac{\alpha \Lambda}{((1-\varepsilon)\kappa^* + \mu)(\kappa^* + \mu + \alpha)}, \\ I^* = \frac{(\kappa^* \Lambda ((1-\varepsilon)\kappa^* + \mu + (1-\varepsilon)\alpha)) \xi_1 \xi_2}{((1-\varepsilon)\kappa^* + \mu)(\kappa^* + \mu + \alpha)(\mu[\xi_2(\gamma + \xi_1) + \rho\xi_1 + \gamma\delta] + \eta\gamma\delta)}, \\ C^* = \frac{\rho(\kappa^* \Lambda ((1-\varepsilon)\kappa^* + \mu + (1-\varepsilon)\alpha)) \xi_1}{((1-\varepsilon)\kappa^* + \mu)(\kappa^* + \mu + \alpha)(\mu[\xi_2(\gamma + \xi_1) + \rho\xi_1 + \gamma\delta] + \eta\gamma\delta)}, \\ A^* = \frac{\gamma(\kappa^* \Lambda ((1-\varepsilon)\kappa^* + \mu + (1-\varepsilon)\alpha)) \xi_2}{((1-\varepsilon)\kappa^* + \mu)(\kappa^* + \mu + \alpha)(\mu[\xi_2(\gamma + \xi_1) + \rho\xi_1 + \gamma\delta] + \eta\gamma\delta)}. \end{cases}$$

Using (5) in the expression for κ^* in (4) shows that the endemic equilibria of the model satisfies

$$(6) \quad a\kappa^{*2} + b\kappa^* + c = 0,$$

with $a = (1 - \varepsilon)$, $b = (1 - \varepsilon)(\mu + \alpha) + \mu - \left(\frac{(\mu + \alpha)(1 - \varepsilon)R_0\mu}{\mu + (1 - \varepsilon)\alpha}\right)$, and $c = \mu(\mu + \alpha)(1 - R_0)$.

It should be noted that the coefficient a in equation (6) is always positive, while the value of the coefficient c is positive or negative depending on the value of R_0 . If $R_0 < 1$ then c is positive, and if $R_0 > 1$ then c is negative. Endemic equilibrium point will exist when the solution of the equation (6) is positive ($\kappa > 0$). From this it is found that model (1) has:

1. Precisely one unique endemic equilibrium if $c < 0$ or $R_0 > 1$,
2. Precisely one unique endemic equilibrium if $b < 0$, and either $c = 0$ or $b^2 - 4ac = 0$,
3. Precisely two unique endemic equilibrium if $b < 0, c > 0$, and $b^2 - 4ac > 0$,
4. No endemic equilibrium otherwise.

3. PARAMETER SENSITIVITY ANALYSIS

Parameter sensitivity analysis aims to determine the parameters that have a major influence on model. This can be known through the sensitivity index (e_p) of each parameter. Parameter sensitivity index (e_p) from R_0 is $e_p = \left(\frac{\partial R_0}{\partial p}\right) \frac{p}{R_0}$ with p is the parameter to be analyzed [15].

On R_0 (3), there are nine parameters that will be searched for sensitivity index, i.e. $\alpha, \beta, \mu, \gamma, \rho, \varepsilon, \nu, \eta$, and δ . Then the parameter values used to calculate the sensitivity index refer to the following Table 3.

TABLE 3. Parameters in HIV/AIDS Model (1)

Parameter	Value	Source
Λ	229,800,000/67.39	[16]
α	0.2351	[10]
β	0.3465	[10]
μ	1/67.39	[16]
γ	0.1882	[10]
ν	0.7661	[10]
η	0.2059	[10]
ρ	3.6523e-04	[10]
δ	0.7012	[10]
ε	0.3243	[10]

The following is an example of how to calculate the sensitivity index for parameter β based on the parameter values presented in Table 3.

$$e_{\beta} = \left(\frac{\partial R_0}{\partial \beta} \right) \frac{\beta}{R_0}$$

$$= \frac{(\mu + (1 - \varepsilon)\alpha)\xi_1\xi_2}{(\mu + \alpha)(\mu[\xi_2(\gamma + \xi_1) + \rho\xi_1 + \gamma\delta] + \eta\gamma\delta)} \frac{\beta(\mu + \alpha)(\mu[\xi_2(\gamma + \xi_1) + \rho\xi_1 + \gamma\delta] + \eta\gamma\delta)}{\beta(\mu + (1 - \varepsilon)\alpha)\xi_1\xi_2} = 1.$$

The results of the parameter sensitivity index analysis of R_0 against parameters in the mathematical model of the spread of HIV/AIDS in the presence of an aware population are given in Table 4 below.

TABLE 4. Parameter sensitivity index

Parameter (p)	Sensitivity Index	$R_0 \approx 2.2763$			
		$p - 10\%$	$p - 5\%$	$p + 5\%$	$p + 10\%$
α	-0.026	2.2822	2.2793	2.2733	2.2704
β	1	2.0487	2.1625	2.3901	2.5039
μ	-0.1236	2.3044	2.2904	2.2622	2.2482
γ	-0.8595	2.4719	2.3741	2.1785	2.0807
ν	0.4443	2.1752	2.2257	2.3269	2.3774
η	2.1649e-04	2.2763	2.2763	2.2763	2.2763
ρ	-2.3209e-04	2.2764	2.2763	2.2763	2.2762
δ	-0.435	2.3753	2.3258	2.2268	2.1773
ε	-0.4389	2.3762	2.3263	2.2263	2.1764

Based on the parameter value from Table 3, we have $R_0 \approx 2.2763$. The last four columns in Table 4 indicate the R_0 value after the parameter value is increased or decreased. For example, the sensitivity index of β is 1 which means that if the transmission rate of HIV spread increases by 10%, it will cause the value of R_0 to increase by 10% (R_0 value became 2.5039) and vice versa if β decreases by 10% then the value of R_0 will also decrease by 10% (R_0 value became 2.0487). But for the rate at which HIV leads to AIDS (γ) and the relative rate of infection of the S_a population to the S_u population (ε) increases by 10%, the value of R_0 will decrease by 8.595% (R_0 value became 2.0807) and 4.389% (R_0 value became 2.1764) respectively and vice versa. The analysis also applies to the parameters $\alpha, \mu, \nu, \eta, \rho$, and δ .

Based on the explanation above, it can be concluded that the β and γ parameters have the most influence on the mathematical model of the spread of HIV/AIDS in the presence of an aware population because the absolute value of the sensitivity index β and γ is the biggest among the other parameters. For clarity, the following simulation graph is given.

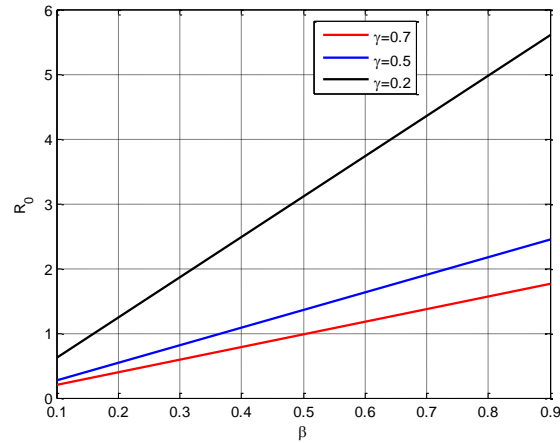


FIGURE 2. The sensitivity β to R_0 values with three different γ values

Based on Figure 2, it can be concluded that if the transmission rate of HIV transmission (β) is getting bigger then the value of R_0 will also be greater which means that the spread of HIV will be more widespread. This happens because the value of the sensitivity parameter β parameter is positive, so that when the value is greater than the value of R_0 will also be even greater. Then with a smaller value of γ resulting in a greater value of R_0 , this is because the parameter sensitivity index γ is negative.

Apart from the sensitivity index table and the graph above, to check whether the parameters β and γ really affect the simulation, the parameter value changes to I population. The initial population values are given $S_u(0) = 129,789,089$, $S_a(0) = 1 \times 10^8$, $I(0) = 7,195$, $C(0) = 0$, and $A(0) = 3,716$, and obtained Figure 3(a) and 3(b) below.

OPTIMAL CONTROLS OF HIV/AIDS MODEL WITH AWARENESS EFFECT

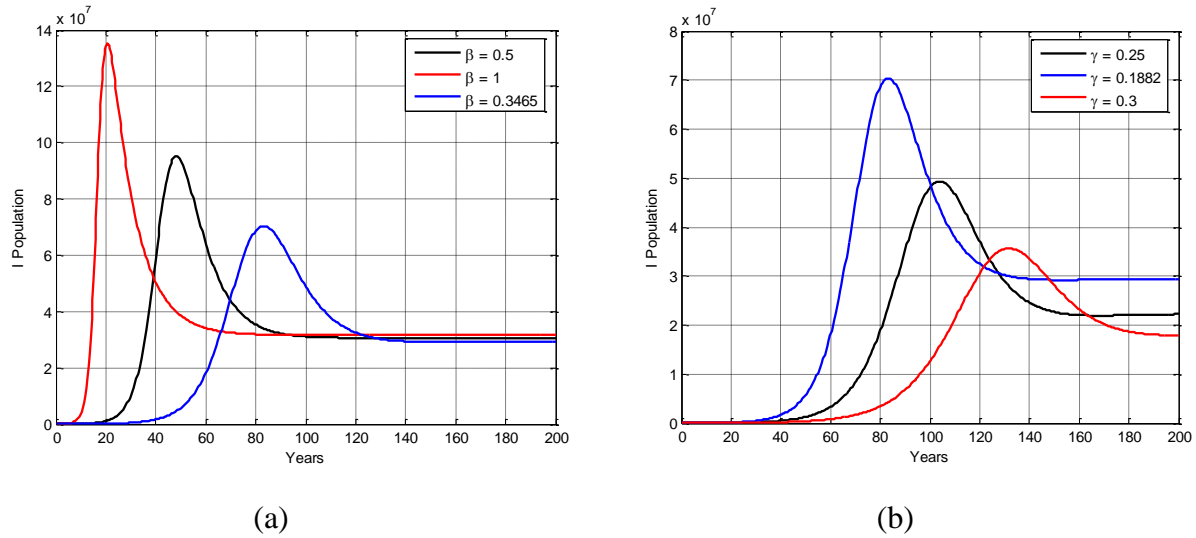


FIGURE 3. *I* Population graph with three different parameter value (a) parameter β and (b) parameter γ

From Figure 3(a), it is clear that the value of the β parameter is very influential in changing of *I* population. When the β parameter is on the low value which means the transmission of the spread of HIV/AIDS is also low, then the population of people infected with the disease will tend to be lower compared to when the β parameter value is high. This happens because the sensitivity index of the β parameter is positive, so the greater the β value, the more the population is infected. Also, at Figure 3(b), the value of the parameter γ also influences changes in *I* population. When the parameter γ is low, which means the rate at which HIV leads to AIDS is also low, then the population of people infected with the disease will tend to be higher than when the parameter value γ high. This happens because the sensitivity index of the parameter γ is negative, so the greater the value of γ , the less the population is infected.

4. APPLICATION OF OPTIMAL CONTROL

In this study, an optimal control analysis will be conducted for a mathematical model of the spread of HIV/AIDS in the presence of an aware population. To determine the optimal control, the construction of the model is carried out with the addition of the control variables. The control

variables used in the form of prevention (u_1) of HIV/AIDS, campaigns (u_2) about the dangers and spread of HIV/AIDS, and also treatment (u_3) in the form of ART.

The description of the notations of each variable and parameters used in constructing the optimal control model is given in Table 5.

TABLE 5. Variable notation and description

Notation	Description
$u_1(t)$	Control input variables such as the prevention of HIV/AIDS at time t
$u_2(t)$	Control input variables such as the campaign of HIV/AIDS at time t
$u_3(t)$	Control input variables such as ART Treatment of HIV/AIDS at time t
c_1	Rate of campaign effectiveness
c_2	Rate of ART Treatment effectiveness to I population
c_3	Rate of ART Treatment effectiveness to A population
t_0	Initial time
t_f	End time
M_1, M_2, M_3, M_4	Weighting constant

The mathematical model with the following controls is,

$$(7) \left\{ \begin{array}{l} \frac{dS_u}{dt} = \Lambda - \left(\alpha + (1 - u_1) \frac{\beta I}{N} + \mu \right) S_u - u_2 c_1 S_u, \\ \frac{dS_a}{dt} = \alpha S_u - \left((1 - \varepsilon)(1 - u_1) \frac{\beta I}{N} + \mu \right) S_a + u_2 c_1 S_u, \\ \frac{dI}{dt} = (1 - u_1) \frac{\beta I}{N} (S_u + (1 - \varepsilon) S_a) + \eta C + \nu A - (\rho + \gamma + \mu) I - u_3 c_2 I + u_3 c_3 A, \\ \frac{dC}{dt} = \rho I - (\eta + \mu) C + u_3 c_2 I, \\ \frac{dA}{dt} = \gamma I - (\nu + \delta + \mu) A - u_3 c_3 A. \end{array} \right.$$

The application of this control variable aims to reduce the number of populations infected with HIV/AIDS that infectious by reducing the rate of spread of the disease and maximizing prevention

efforts, treatment, and campaigns with minimum costs. Pontryagin's Maximum Principle method is used to achieve this objective.

The performance index that can be formed based on the above explanation is as follows:

$$(8) \quad \min J(u_1, u_2, u_3) = \int_0^{t_f} \left[M_1 I + \frac{1}{2} (M_2 u_1^2 + M_3 u_2^2 + M_4 u_3^2) \right] dt$$

with $M_1, M_2, M_3,$ and M_4 respectively is a weighting constant in the form of costs that must be incurred for the infected population, the cost of using contraception, the cost of campaigns in the mass media, and the cost of treatment with ART. The controls are bounded on $0 \leq u_i(t) \leq 1$, with $i = 1, 2, 3$ and $0 \leq t \leq t_f$. The quadratic function of the control costs are adopted, as stated in [17, 18, 19].

Based on Pontryagin's Maximum Principle [20], the first step carried out in the analysis of the optimal control problem is to form a Hamiltonian (H) function, that is:

$$(9) \quad H = M_1 I + \frac{1}{2} (M_2 u_1^2 + M_3 u_2^2 + M_4 u_3^2) + \lambda_1 \left(\Lambda - \left(\alpha + (1 - u_1) \frac{\beta I}{N} + \mu \right) S_u - u_2 c_1 S_u \right) + \lambda_2 \left(\alpha S_u - \left((1 - \varepsilon)(1 - u_1) \frac{\beta I}{N} + \mu \right) S_a + u_2 c_1 S_u \right) + \lambda_3 \left((1 - u_1) \frac{\beta I}{N} (S_u + (1 - \varepsilon) S_a) + \eta C + v A - (\rho + \gamma + \mu) I - u_3 c_2 I + u_3 c_3 A \right) + \lambda_4 (\rho I - (\eta + \mu) C + u_3 c_2 I) + \lambda_5 (\gamma I - (v + \delta + \mu) A - u_3 c_3 A),$$

where $\lambda_1, \lambda_2, \lambda_3, \lambda_4,$ and λ_5 are adjoint variables or co-state variables.

Furthermore, in order to obtain optimal conditions, the Hamiltonian function above must meet stationary conditions, that:

$$(10) \quad \frac{\partial H}{\partial u} = 0 \Leftrightarrow \begin{pmatrix} \frac{\partial H}{\partial u_1} \\ \frac{\partial H}{\partial u_2} \\ \frac{\partial H}{\partial u_3} \end{pmatrix} = 0.$$

So that the optimal controller $u_1, u_2,$ and u_3 is obtained

$$(11) \quad \begin{cases} u_1^* = \min \left(1, \max \left(0, \frac{1}{M_2} \frac{\beta I}{N} (S_u (\lambda_3 - \lambda_1) + (1 - \varepsilon) S_a (\lambda_3 - \lambda_2)) \right) \right), \\ u_2^* = \min \left(1, \max \left(0, \frac{1}{M_3} c_1 S_u (\lambda_1 - \lambda_2) \right) \right), \\ u_3^* = \min \left(1, \max \left(0, \frac{1}{M_4} (c_2 I (\lambda_4 - \lambda_3) + c_3 A (\lambda_3 - \lambda_5)) \right) \right). \end{cases}$$

Because the controller form of u_1^* , u_2^* , and u_3^* , there are the state variables (S_u, S_a, I, C, A) and co-state variables $(\lambda_1, \lambda_2, \dots, \lambda_5)$, then the next step will be completed state and co-state equations to obtain these variables.

The state equations are as follows.

$$(12) \quad \left\{ \begin{array}{l} \dot{S}_u = \frac{\partial H}{\partial \lambda_1} = \Lambda - \left(\alpha + (1 - u_1) \frac{\beta I}{N} + \mu \right) S_u - u_2 c_1 S_u, \\ \dot{S}_a = \frac{\partial H}{\partial \lambda_2} = \alpha S_u - \left((1 - \varepsilon)(1 - u_1) \frac{\beta I}{N} + \mu \right) S_a + u_2 c_1 S_u, \\ \dot{I} = \frac{\partial H}{\partial \lambda_3} = (1 - u_1) \frac{\beta I}{N} (S_u + (1 - \varepsilon) S_a) + \eta C + \nu A - (\rho + \gamma + \mu) I - u_3 c_2 I + u_3 c_3 A, \\ \dot{C} = \frac{\partial H}{\partial \lambda_4} = \rho I - (\eta + \mu) C + u_3 c_2 I, \\ \dot{A} = \frac{\partial H}{\partial \lambda_5} = \gamma I - (\nu + \delta + \mu) A - u_3 c_3 A. \end{array} \right.$$

The co-state equations are as follows.

$$(13) \quad \left\{ \begin{array}{l} \dot{\lambda}_1 = -\frac{\partial H}{\partial S_u} = (1 - u_1) \left((\lambda_1 - \lambda_3) \left(\frac{\beta I}{N} - \frac{\beta I}{N^2} S_u \right) + (\lambda_3 - \lambda_2)(1 - \varepsilon) \frac{\beta I}{N^2} S_a \right) \\ \quad \quad \quad + (\lambda_1 - \lambda_2)(u_2 c_1 + \alpha) + \lambda_1 \mu, \\ \dot{\lambda}_2 = -\frac{\partial H}{\partial S_a} = (1 - u_1) \left((\lambda_2 - \lambda_3)(1 - \varepsilon) \left(\frac{\beta I}{N} - \frac{\beta I}{N^2} S_a \right) + (\lambda_3 - \lambda_1) \frac{\beta I}{N^2} S_u \right) \\ \quad \quad \quad + \lambda_2 \mu, \\ \dot{\lambda}_3 = -\frac{\partial H}{\partial I} = (1 - u_1) \left((\lambda_1 - \lambda_3) S_u + (\lambda_2 - \lambda_3)(1 - \varepsilon) S_a \right) \left(\frac{\beta}{N} - \frac{\beta I}{N^2} \right) \\ \quad \quad \quad + (\lambda_3 + \lambda_4) u_3 c_2 + \lambda_3 (\rho + \gamma + \mu) - \lambda_4 \rho - \lambda_5 \gamma - M_1 \\ \dot{\lambda}_4 = -\frac{\partial H}{\partial C} = (1 - u_1) \left((\lambda_3 - \lambda_1) \frac{\beta I}{N^2} S_u + (\lambda_3 - \lambda_2)(1 - \varepsilon) \frac{\beta I}{N^2} S_a \right) \\ \quad \quad \quad + (\lambda_4 - \lambda_3) \eta + \lambda_4 \mu, \\ \dot{\lambda}_5 = -\frac{\partial H}{\partial A} = -(1 - u_1) \left((\lambda_3 - \lambda_1) S_u + (\lambda_3 - \lambda_2)(1 - \varepsilon) S_a \right) \frac{\beta I}{N^2} \\ \quad \quad \quad + (\lambda_5 - \lambda_3)(u_3 c_3 + \nu) + \lambda_5 (\mu + \delta). \end{array} \right.$$

with $\lambda_i(t_f) = 0$, for $i = 1, 2, \dots, 5$.

Based on the description above, to get the value of S_u, S_a, I, C , and A from the optimal form u^* then it is necessary to solve the non-linear state and co-state equations. Because the non-linear equation system is difficult to solve analytically, it will be solved numerically.

5. NUMERICAL RESULTS

This numerical simulation is done by comparing the mathematical model of the spread of HIV/AIDS without the control variables and with the presence of the control variables. It aims to recognize the level of effectiveness of the control effort so that the purpose of the cost function provided will be achieved. We utilize the fourth order Runge-Kutta (RK4) scheme to solve the strategy of optimal control. To start with, we actualize the forward RK4 scheme to solve the state system. From that point onward, we use the backward RK4 scheme to unravel the co-state system. We update the controls until the current state, the adjoint, and the control value converge adequately [21].

The simulation is done using the initial value for each condition is $S_u(0) = 129,789,089$, $S_a(0) = 1 \times 10^8$, $I(0) = 7,195$, $C(0) = 0$, and $A(0) = 3,716$ [10] and performed at $t = 0$ and $t = 20$ years. The parameter values used refer to Table 3. Weighting constants for infected populations, the cost of using contraception, the cost of campaigns in the mass media, and the cost of treatment with ART respectively $M_1 = 1$, $M_2 = 20$, $M_3 = 100$, and $M_4 = 500$. Then the value of the rate of effectiveness of the campaign, the rate of effectiveness of ART for I population, and the rate of effectiveness of ART for A population are, respectively, $c_1 = 0,7$, $c_2 = 0,85$, and $c_3 = 0,75$. We investigate four control scenario which are given as follows.

1. Combination of HIV/AIDS prevention (u_1) and campaign of HIV/AIDS (u_2).
2. Combination of HIV/AIDS prevention (u_1) and HIV/AIDS treatment (u_3).
3. Combination of campaign of HIV/AIDS (u_2) and HIV/AIDS treatment (u_3).
4. Combination of HIV/AIDS prevention (u_1), campaign of HIV/AIDS (u_2), and HIV/AIDS treatment (u_3).

5.1. First Scenario

In the first scenario, combination of HIV/AIDS prevention (u_1) and campaign of HIV/AIDS (u_2) is used. Meanwhile, the HIV/AIDS treatment control is not used ($u_3 = 0$). The profile of optimal controls u_1 and u_2 is plotted in Figure 4. The HIV/AIDS prevention should be done

intensively for almost 20 years. Meanwhile, the campaign of HIV/AIDS is simply not done effectively.

Furthermore, the dynamics of infected HIV/AIDS populations that infectious are given in Figure 5 and the dynamics of the C and A populations are given in Figure 6. From Figure 5, it is shown that HIV/AIDS prevention and campaign of HIV/AIDS controls provide a significant reduction in HIV/AIDS patients that infectious (I) compared to having no controls. Similar conditions also hold for C and A populations, their populations are lower compared to running the model without controls as depicted in Figures 6(a)-6(b).

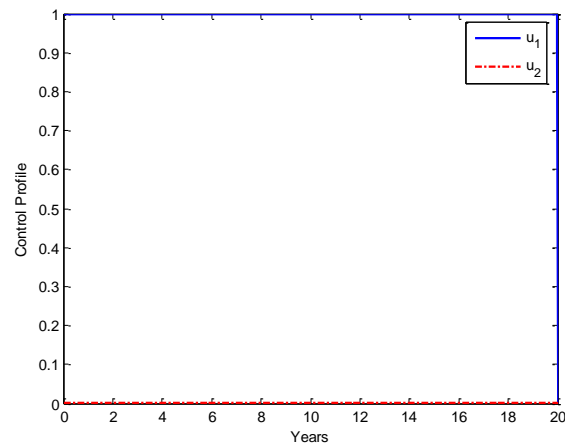


FIGURE 4. Control profiles of u_1 and u_2

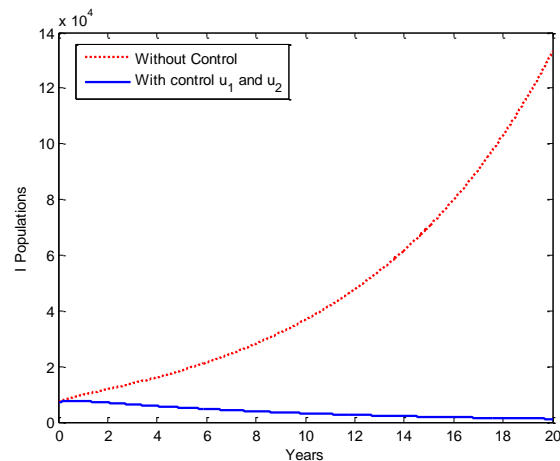


FIGURE 5. The dynamics of I populations using optimal controls u_1 and u_2

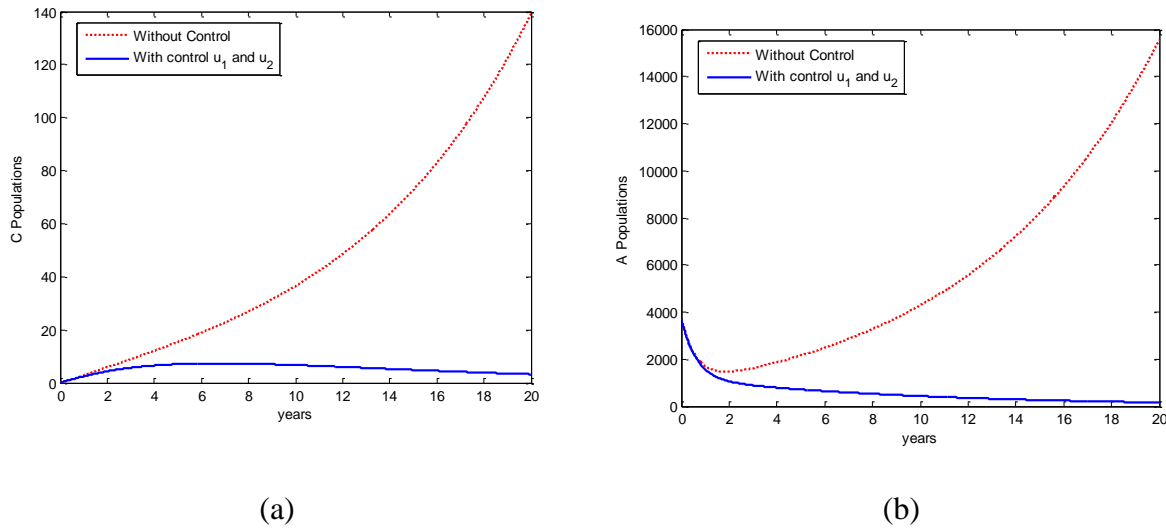


FIGURE 6. The dynamics of (a) C populations and (b) A populations using optimal controls u_1 and u_2

5.2. Second Scenario

In the second scenario, combination of HIV/AIDS prevention (u_1) and treatment of HIV/AIDS (u_3) is used. Meanwhile, the HIV/AIDS campaign control is not used ($u_2 = 0$). The profile of optimal controls u_1 and u_3 is plotted in Figure 7. The HIV/AIDS prevention should be done intensively for almost 20 years. Meanwhile, the treatment of HIV/AIDS should be done intensively just for the first year.

Furthermore, the dynamics of infected HIV/AIDS populations that infectious are given in Figure 8 and the dynamics of the C and A populations are given in Figure 9. Based on Figure 8, it is shown that HIV/AIDS prevention and treatment of HIV/AIDS controls provide a significant reduction in HIV/AIDS patients that infectious (I) compared to having no controls. Similar conditions also hold for C and A populations, their populations are lower compared to running the model without controls as depicted in Figures 9(a)-9(b).

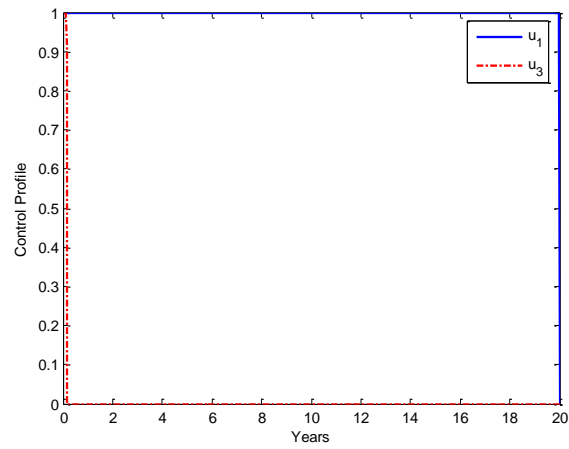


FIGURE 7. Control profiles of u_1 and u_3

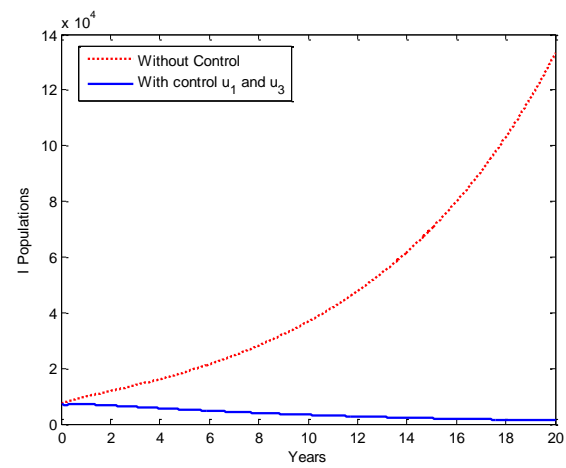
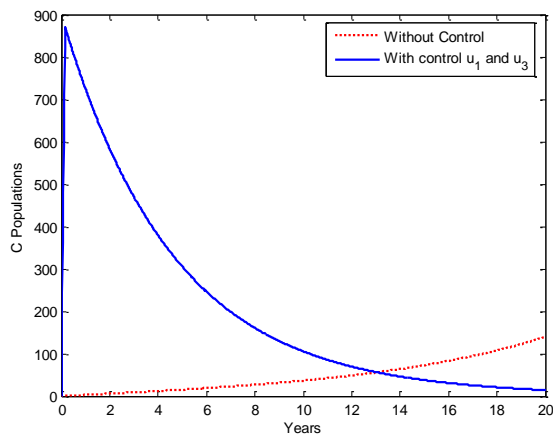
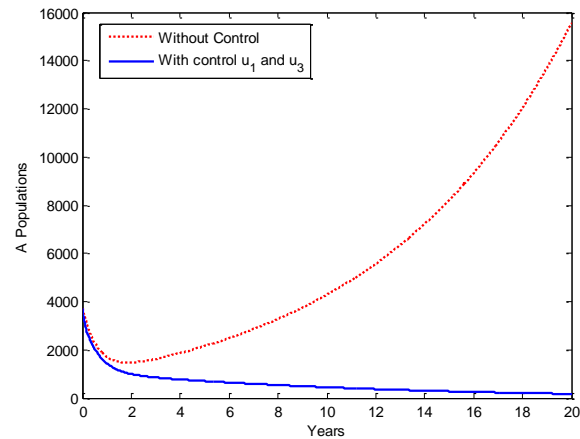


FIGURE 8. The dynamics of I populations using optimal controls u_1 and u_3



(a)



(b)

FIGURE 9. The dynamics of (a) C populations and (b) A populations using optimal controls u_1 and u_3

5.3. Third Scenario

In the third scenario, combination of HIV/AIDS campaign (u_2) and treatment of HIV/AIDS (u_3) is used. Meanwhile, the HIV/AIDS prevention control is not used ($u_1 = 0$). The profile of optimal controls u_2 and u_3 is plotted in Figure 10. The HIV/AIDS campaign should be done intensively just for the last year. Meanwhile, the treatment of HIV/AIDS is simply not done effectively.

Furthermore, the dynamics of infected HIV/AIDS populations that infectious are given in Figure 11 and the dynamics of the C and A populations are given in Figure 12. From Figure 11, it is depicted that HIV/AIDS prevention and treatment of HIV/AIDS controls provide insignificant reduction in HIV/AIDS patients that infectious (I) compared to having no controls. Because HIV/AIDS campaign control just be done intensively for the last year, so the reduction just happened in the last year. Similar conditions also hold for C and A populations, their populations are little bit lower compared to running the model without controls as depicted in Figures 12(a)-12(b).

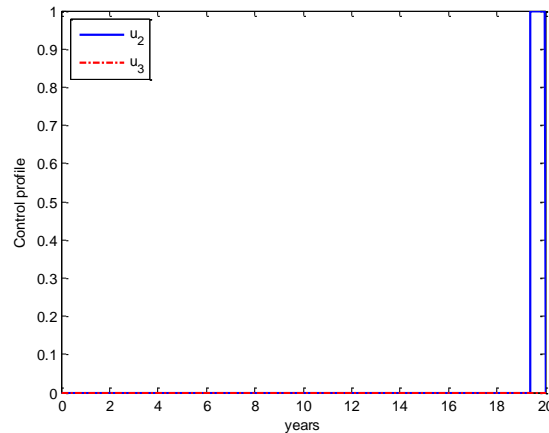


FIGURE 10. Control profiles of u_2 and u_3

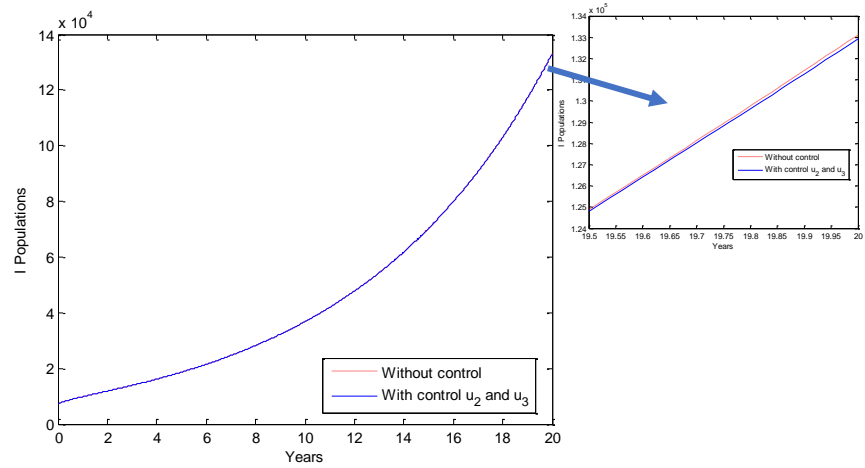


FIGURE 11. The dynamics of I populations using optimal controls u_2 and u_3

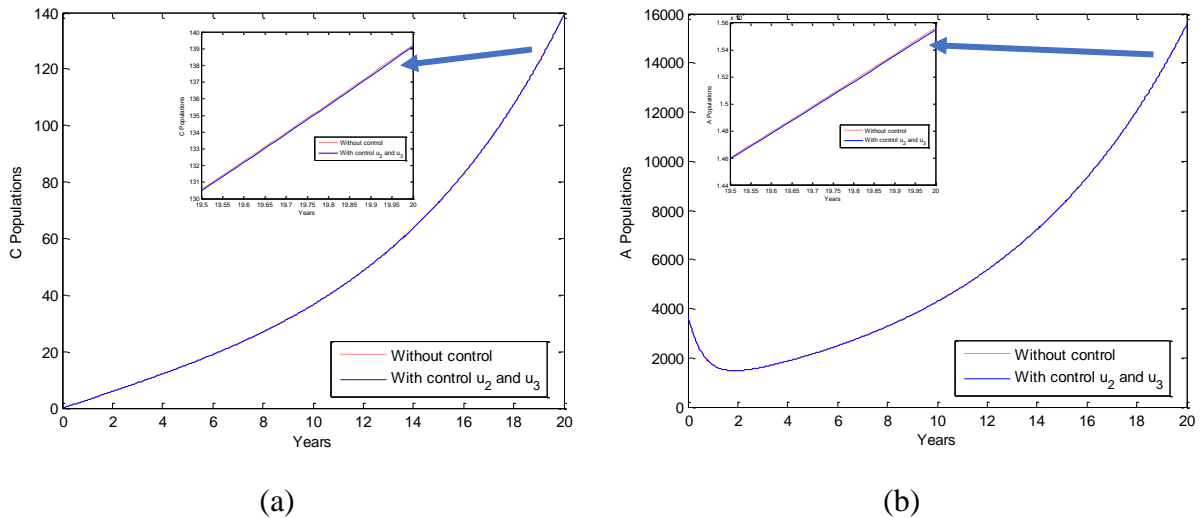


FIGURE 12. The dynamics of (a) C populations and (b) A populations using optimal controls

u_2 and u_3

5.4. Fourth Scenario

In the fourth scenario, all of the combinations of the controls (u_1 , u_2 , and u_3) are used. The profile of optimal controls u_1 , u_2 , and u_3 is plotted in Figure 13. As in the first and second scenarios, HIV/AIDS prevention control should be done intensively for almost 20 years. Meanwhile, the campaign of HIV/AIDS are simply not done effectively, and the treatment of HIV/AIDS should be done intensively just for the first year.

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Furthermore, the dynamics of infected HIV/AIDS populations that infectious are given in Figure 14 and the dynamics of the C and A populations are given in Figure 15. Figure 14 shows that HIV/AIDS prevention and treatment of HIV/AIDS controls provide a significant reduction in HIV/AIDS patients that infectious (I) compared to having no controls. Similar conditions also hold for C and A populations, their populations are lower compared to running the model without controls as depicted in Figures 15(a)-15(b).

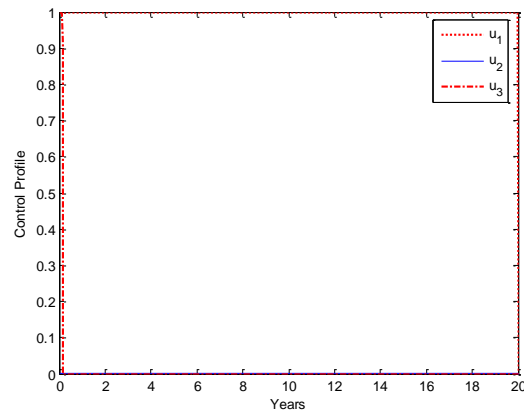


FIGURE 13. Control profiles of u_1 , u_2 , and u_3

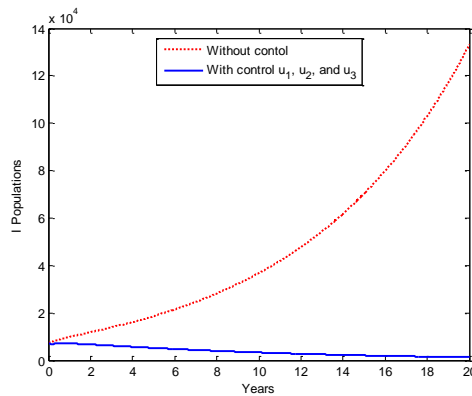


FIGURE 14. The dynamics of I populations using optimal controls u_1 , u_2 , and u_3

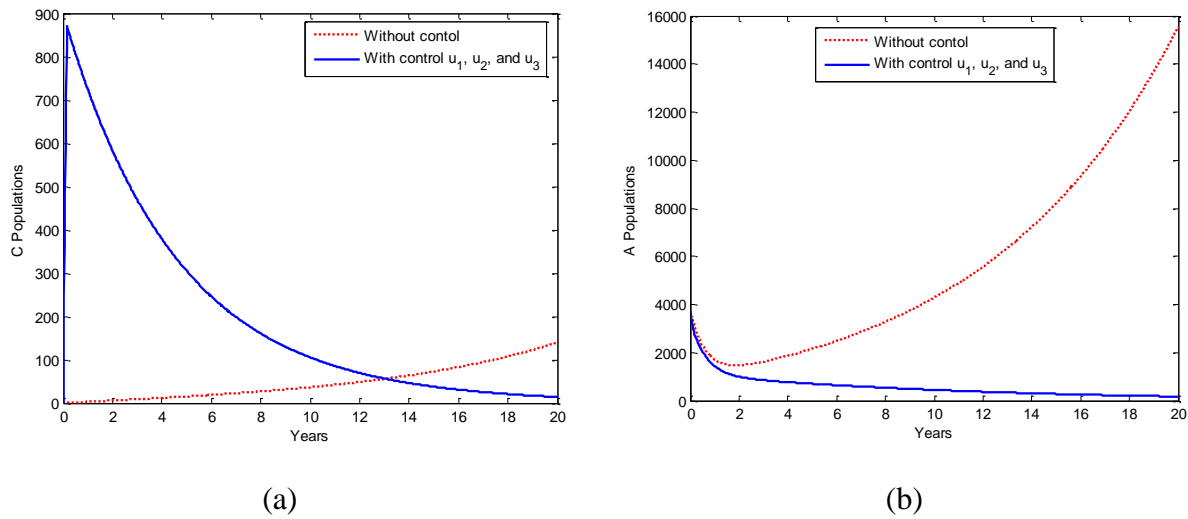


FIGURE 15. The dynamics of (a) C populations and (b) A populations using optimal controls $u_1, u_2, \text{ and } u_3$

The following table presents a comparison of the cost functions required for various scenarios of applying the controls used.

TABLE 6. Cost function of every scenario

Scenario	Cost Function
Controls u_1 and u_2	74,267
Controls u_1 and u_3	74,459
Controls u_2 and u_3	948,950
Controls $u_1, u_2, \text{ and } u_3$	74,459

From Table 6, it is shown that the value of the smallest cost function or performance index to minimize the number of human populations infected with HIV that infectious (I) are by applying the first scenario. It happens because the first scenario giving u_1 control is the most dominant. Because giving u_1 control, namely prevention using contraception, especially condoms, is very efficient in preventing the spread of HIV/AIDS through sexual activity. Not only efficient, but it

is also cheaper than the cost of treatment for people with HIV/AIDS or the campaigns in the mass media about HIV/AIDS. Meanwhile, for the second and third scenarios, the cost function is slightly greater than the first scenario because there are costs for the third control (u_3). The second control (u_2) is not effective at all. So, for all scenarios u_2 control is not activated.

From the results obtained, it can be concluded that within 20 years of observation, to optimize the number of human populations infected with HIV that infectious (I), including populations C or A is to apply the scenario that giving u_1 and u_3 control or only giving u_1 control. But if to minimize the cost function as well, then giving only u_1 control is very optimal.

6. CONCLUSION

In this paper, we have analyzed the model of the spread of HIV/AIDS with an aware population by incorporate the optimal control problem. The HIV model has two equilibria, namely the disease-free equilibrium and the endemic equilibrium. The disease-free equilibrium will be locally asymptotically stable when the basic reproduction number less than one. Next, parameter sensitivity analysis is examined to determine the most influential parameters on the spread of this disease. The optimal controls are then applied to the HIV model in the form of prevention, campaign, and treatment of Antiretroviral Therapy (ART). Based on the results of numerical simulations before and after being given control shows that the provision of control in the form of prevention and campaign is the most effective way to reduce the number of human populations infected with HIV that infectious (I), including populations C or A and also minimizing the cost function.

ACKNOWLEDGEMENTS

Part of this research is funded by Universitas Airlangga through Riset Mandat 2018.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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