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# Optimal Strategy for HIV Model

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## Abstract
The human immune-deficiency virus (HIV) with its associated acquired immune-deficiency syndrome (AIDS) is one of the pandemics that have worst-afflicted the humanity in history. A deterministic mathematical model for HIV and an optimal control model for HIV transmission dynamics that can select the best strategy was developed. The basic model being studied by exploring the basic reproduction number (which is the largest eigenvalue of the next generation matrix) as an epidemic threshold. The local and global stability of the disease-free equilibrium (DFE) shows that the disease-free equilibrium state is locally and globally asymptotically stable whenever the basic reproduction number is less than unity. Furthermore, an optimal control model for HIV incorporating three time-dependent control functions such as measures of condom use, HIV compatibility test and treatment were formulated. The corresponding optimality system was characterized and derived using Pontryagin's maximum principle. The optimality system was numerically solved using the forward-backward Runge-Kutta method of order four. The simulated results for all possible control strategies using estimated and published parameter values showed that the model is able to select the optimal strategy. Findings from the study suggest that the combination of the three control measures can effectively reduce the spread of HIV in a community.

**Key words:** Optimal control, Pontryagin's maximum principle, HIV/AIDS

## 1. Introduction

Acquired immunodeficiency syndrome (AIDS) first reported in 1981 with its causative agent, human immunodeficiency virus (HIV) discovered in 1983, has wrecked a monumental havoc on the human species. This is evidenced by the morbidity, mortality and financial burdens borne by governments, donor agencies and households of the affected countries. Estimates of 74.9 to 76.1 million people have been infected with HIV since the start of the epidemic up to the end of 2018 (UNAIDS, 2017; UNAIDS, 2019) and 32 to 35 million people have died as a result of AIDS within the same period.

As at 2018, an estimated 37.9 million people were living with HIV, incidence accounting for about 1.7 million; and an estimated 770,000 people died from HIV-related illnesses worldwide (UNAIDS, 2019). An estimated 36.7 million people were living with HIV, with incidence of about 1.8 million; and an estimated 1 million deaths occurred from AIDS in 2016 worldwide (UNAIDS, 2017).

About 36.9 million people were living with HIV, with 2 million newly infected in 2014; and an estimated 1.2 million people died within the same year (UNAIDS, 2015).

Although the global burden is very high, the highest burden is borne by the worst-afflicted African region. Of the 37.9 million people living with HIV globally in 2018, 25.7 million of them were living in Africa, which accounts for about two-third of the global burden. At the country level, Nigeria is the third worst-hit, after South Africa and India.

As part of preventive and control measures, counseling and antiretroviral drugs, condoms and abstinence are available, but the data taken from the global HIV and AIDS statistics facts sheet of those people living with HIV and assessing antiretroviral therapy is increasing steadily. At the end of June 2019, about 24.5 million people living with HIV were accessing antiretroviral therapy, which is an increase over the approximately 23.3 million people living with HIV that were accessing antiretroviral therapy in 2018. This figure is high compared to the 7.7 million people living with HIV and assessing antiretroviral therapy in 2010 (UNAIDS, 2019). It is evident from the foregoing that HIV/AIDS has caused and is causing serious havoc to humanity.

Mathematical models are veritable tools for studying the dynamics of HIV and AIDS prevalence. For instance, Cai et al. (2009) investigated an HIV/AIDS epidemic model with treatment, studying the stability of its equilibrium points. Naresh et al.(2009) used a mathematical model to analyze the spread of the HIV/AIDS epidemic with recruitment of infectives. Nyabadza et al. (2010) used a model incorporating condom use, sexual partner acquisition, behavior change and treatment to study the epidemic trends of HIV/AIDS in South Africa. Basak et al. (2015) presented a deterministic HIV/AIDS epidemic model and established that the disease burden can be controlled by controlling the infective contact rate of the infected population.

Optimal control techniques have been used to determine best control strategies for infectious diseases such as malaria, Ebola, Influenza, tuberculosis, hepatitis B, tungiasis, to mention a few, ( see, Khamis et al. (2018); Otieno et al. (2016); Mwanga et al. (2014); Ebenezer et al. (2016); Kahuru et al. (2017); Hattaf et al. (2009); Silva and Torees (2014); Athithan and Gosh (2016); Tchuenche et al. (2011)). Optimal control theory has been applied to a quite a few HIV models. Karrakchou et al. (2006) investigates the fundamental role of chemotherapy treatment in controlling the viral production using optimal control theory. Sule and Abdullahi (2014) explored the optimal control representing public health education and antiretroviral therapy. Mastahun and Abdurahman (2017) formulated an optimal control problem that considers behavioural change and screening as a major control strategies. Optimal control theory is applied by Ngina et al. (2018) to investigate the key roles played by the various HIV treatment strategies. Semegni et al. (2019) studied the optimal control of rolling out a public health education campaign. These optimal control studies tend to emphasize public health education campaign and treatment, but studies on optimal control of preventive measures such as condom use, and pre-sex testing is not available. Thus it is instructive to carry out optimal control modeling studies that focus on condom use, pre – sex testing and treatment of infectious individuals. Basak et al. (2015) presented a deterministic model of HIV/AIDS epidemic model. Their results result revealed that the disease burden can be controlled by controlling the infective contact rate of the infected population. The study by Basak et al. (2015) did not incorporate any intervention strategy. The current study modifies and extends Basak et al. (2015) study by incorporating the compartment of infectious individuals receiving treatment and investigating for an optimal control strategy for condom use, pre-sex testing and treatment to minimize the spread of HIV/AIDS.

The plan of this paper is as follows. The basic HIV model is presented in section 2. Section 3 is devoted to analysis of the model. In Section 4, the extended and optimal control model was formulated and analyzed. Numerical simulation and results are presented in section 5. Discussion of results in section 6 and conclusive remarks are passed in section 7.

## 2. Model Formulation

The model by Basak et al. (2015), the heterosexual population at time $t$, denoted by $N(t)$ is compartmentalized into four classes namely: susceptible people, $S(t)$, the infected individuals who do not know they infected, $I_1(t)$, the infected individuals who know that they are infected, $I_2(t)$ and those who have developed AIDS, $A(t)$, so that:

$$N(t) = S(t) + I_1(t) + I_2(t) + A(t) \quad (1)$$

The governing equations by Basak et al. (2015) are given as:

$$\frac{dS}{dt} = \Pi - \lambda S - \mu S \quad (2)$$

$$\frac{dI_1}{dt} = \lambda S - (\omega + \delta_1 + \mu) I_1 \quad (3)$$

$$\frac{dI_2}{dt} = \omega I_1 - (\delta_2 + \mu) I_2 \quad (4)$$

$$\frac{dA}{dt} = \delta_1 I_1 + \delta_2 I_2 - (\mu + d) A \quad (5)$$

where $\lambda = \frac{\beta_1 I_1 + \beta_2 I_2}{N}$

Basak et al. (2015) put forward that individuals who do know that they are infected, $I_2$ are more infectious than those who did not know that they are infected, $I_1$. This is a flaw since infectiousness cannot be determined by the knowledge of one status but by not assessing antiretroviral therapy. Much more, the model did not capture any preventive or control measure, thus splitting the infected individuals will not have any effect on the result. On these premises, the current study modified the model by Basak et al. (2015) by merging the infected classes to one. Hence the total population at time $t$, can be written as:

$$N(t) = S(t) + I(t) + A(t) \quad (6)$$

The variables and parameters used in the model are defined in Table 1.

**Table 1:** Variables and Parameters used in the model and their description

| Variable/Parameter | Description |
|---|---|
| $S(t)$ | The number of susceptible to HIV infection at time t |
| $I(t)$ | The number of infectious individuals at time t |
| $A(t)$ | The number of those who have developed AIDS at time t |
| $\Delta$ | Recruitment rate |
| $\beta_1, \beta_2, \beta_3$ | Effective contact rate for HIV infection transmission |
| $\kappa$ | Progression rate of infectious individuals to AIDS |
| $\mu_h$ | Natural mortality rate |
| $\delta_2$ | Disease induced mortality rate |

From the above definition of variables and parameters, the interactions and flow in the different compartments are as depicted in the schematic flow diagram (Figure 1) below.

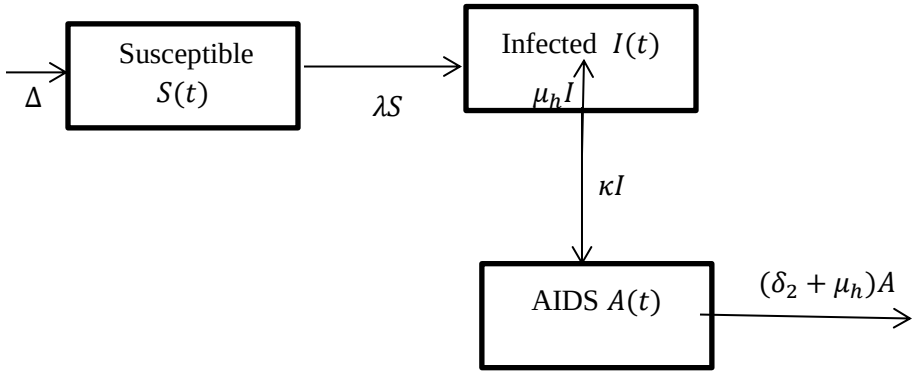


**Figure 1:** Schematic description of the model

The susceptible population $S(t)$, is generated by recruitment of individuals into the population at the rate Δ. It is reduced by infection, following effective contact with people infected with HIV at the rate λ and by natural death at the rate $\mu_h$.The rate $\lambda$ is given by

$\lambda = \beta_1 I + \beta_3 A$

where $\beta_1$, $\beta_3$ are the effective contact rate for HIV transmission. Thus

$$\frac{dS}{dt} = \Delta - \lambda S - \mu_h S$$

The population of infectious individuals $I(t)$ is generated following infection of susceptible population at the rate $\lambda$. The population of infectious individuals are decreased as a result of progression of infected individuals with HIV to AIDS at the rate $\kappa$, and natural death at the rate $\mu_h$, so that

$$\frac{dI}{dt} = \lambda S - (\kappa + \mu_h)I$$

The population of infectious individuals who have developed AIDS, $A(t)$ are generated as a result of progression of infected individuals with HIV into the AIDS class at the rate $\kappa$. The population of infectious individuals who have developed AIDS are decreased by diseased-induced death at the rate $\delta_2$ and natural death at the rate $\mu_h$. Hence,

$$\frac{dA}{dt} = \kappa I - (\delta_2 + \mu_h)A$$

## 2.1. The Modified Model Equations

The above assumptions and derivations leads to the following system of ordinary differential equations:

$$\frac{dS}{dt} = \Delta - \lambda S - \mu_h S \quad (7)$$

$$\frac{dI}{dt} = \lambda S - (\kappa + \mu_h)I \quad (8)$$

$$\frac{dA}{dt} = \kappa I - (\delta_2 + \mu_h)A \quad (9)$$

where $\lambda = \beta_1 I + \beta_3 A$

$S(t) \geq 0, I(t) \geq 0, A(t) \geq 0$

## 2.2 Invariant Region

We obtain the invariant region in which the model solution is bounded. All the associated parameters and state variables are non-negatives for $t \geq 0$. Consider the biological feasible region

$$\Omega = \left\{(S, I, A) \in \mathbb{R}^3 : N \leq \frac{\Delta}{\mu_h}\right\}$$

**Lemma 1**: The closed set Ω is positively and attracting with respect to the system of equations (7) – (9).

**Proof:**

Adding equations (7) – (9) gives the rate of change of the total population:

$$\frac{dN}{dt} = \Delta - \mu_h N - \delta_2 A$$

It is clear from equation (4) that

$$\frac{dN}{dt} \leq \Delta - \mu_h N \text{ it follows that}$$

$$\frac{dN}{dt} \leq 0 \text{ if } N(t) \geq \frac{\Delta}{\mu_h}$$

Thus, by a standard comparison theorem (Lakshmikantham et al. 1989) can be used to show that

$$N(t) \leq N(0)e^{-\mu_h t} + \frac{\Delta}{\mu_h}(1 - e^{-\mu_h t})$$

In particular

$N(t) \leq \frac{\Delta}{\mu_h}$ if $\mathrm{N}(0) \leq \frac{\Delta}{\mu_h}$. Thus the region Ω ispositively-invariant. However if $\mathrm{N(t)} \leq \frac{\Delta}{\mu_h}$, then either the solution enters Ω in finite time, or N(t) approaches $\frac{\Delta}{\mu_h}$ asymptotically. Hence the region Ω attracts all solutions in $\mathbb{R}^3$.

Therefore, it is sufficient to consider the dynamics of the flow generated by equations (7) – (9) in Ω, where the usual existence, uniqueness, continuation results hold for the system (7) – (9), that is the system is mathematically and epidemiological well-posed in Ω.

## 3. Analysis of the Modified Model

### 3.1 Local Stability of the Disease Free Equilibrium (DFE) State

The model (7) – (9) has a disease-free equilibrium (DFE) given by

$$\mathcal{E}_0 = (S, I, A) = (\frac{\Delta}{\mu_h}, 0, 0) \qquad (11)$$

We will study the local stability of the disease free equilibrium state, $\mathcal{E}_0$, exploring the basic reproduction number $R_0$. The basic reproduction, $R_0$, measures the average number of new infection generated by a single infected individual in a completely susceptible population.

For the recipe on computation of basic reproduction number using the next generation operator method, (see Van den Driessche and Watmough (2002), Castillo-Chavez et al. (2002), Diekmann et al. (1990) and Heffernan (2005)). Let the non-negative matrix, $F$, of new infection terms and the $M$-matrix, $V$, of transfer terms associated with the model (7) – (9) be given respectively, by

$$F = \begin{pmatrix} \beta_1 \frac{\Delta}{\mu_h} & \beta_3 \frac{\Delta}{\mu_h} \\ 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} \kappa + \mu_h & 0 \\ -\kappa & \delta_2 + \mu_h \end{pmatrix}$$

Now

$$V^{-1} = \begin{pmatrix} \frac{1}{\kappa+\mu_h} & 0 \\ \frac{\kappa}{(\kappa+\mu_h)(\delta_2+\mu_h)} & \frac{1}{\delta_2+\mu_h} \end{pmatrix}$$

so that

$$FV^{-1} = \begin{pmatrix} \frac{\beta_1\Delta}{\mu_h(\kappa+\mu_h)} + \frac{\beta_1\Delta\kappa}{\mu_h(\kappa+\mu_h)(\delta_2+\mu_h)} & \frac{\beta_3\Delta}{\mu_h(\kappa+\mu_h)} \\ 0 & 0 \end{pmatrix}.$$

The dominant eigenvalue of $FV^{-1} = \begin{pmatrix} \frac{\beta_1\Delta}{\mu_h(\kappa+\mu_h)} + \frac{\beta_1\Delta\kappa}{\mu_h(\kappa+\mu_h)(\delta_2+\mu_h)} & \frac{\beta_3\Delta}{\mu_h(\kappa+\mu_h)} \\ 0 & 0 \end{pmatrix}$ is given by

$$\lambda = \frac{\beta_1\Delta}{\mu_h(\kappa+\mu_h)} + \frac{\beta_1\Delta\kappa}{\mu_h(\kappa+\mu_h)(\delta_2+\mu_h)}$$

and therefore, the basic reproduction number is given by

$$R_0 = \frac{\beta_1\Delta}{\mu_h(\kappa+\mu_h)} + \frac{\beta_1\Delta\kappa}{\mu_h(\kappa+\mu_h)(\delta_2+\mu_h)}. \tag{12}$$

Using theorem 2 in Van den Driessche and Watmough (2002), we established the following result.

**Lemma 2**: The DFEs of the model (7) – (9), given by $\mathcal{E}_0$, locally asymptotically stable (LAS) if $R_0 < 1$ and $\mathcal{E}_0$ is unstable if $R_0 > 1$.

### 3.2. Global Asymptotical Stability (GAS) of the Disease Free Equilibrium (DFE) State

**Theorem 1:** The DFE of model (7) – (9), given by $\Omega_0$ is GAS whenever $R_0 \leq 1$.

**Proof**: Consider the Lyapunov function

$$L = I,$$

with Lyapunov derivative (where a prime represents differentiation with respect to $t$)

$$L' = (\beta_1 I + \beta_3 A)S - (\kappa + \mu_h)I$$

$$= \frac{\beta_1\Delta}{\mu_h} I + \frac{\beta_3\Delta\kappa}{\mu_h(\delta_2+\mu_h)} I - (\kappa + \mu_h)I$$

$$= \left(\frac{\beta_1\Delta}{\mu_h} + \frac{\beta_3\Delta\kappa}{\mu_h(\delta_2+\mu_h)} - (\kappa+\mu_h)\right) I$$

$$= (\kappa+\mu_h)I\left(\frac{\beta_1\Delta}{\mu_h(\kappa+\mu_h)} + \frac{\beta_3\Delta\kappa}{\mu_h(\delta_2+\mu_h)(\kappa+\mu_h)} - 1\right)$$

$$= (\kappa+\mu_h)I[R_0 - 1]$$

$$\leq 0 \text{ for } R_0 \leq 1$$

## 4. Optimal Control Model Formulation and Description

In this section, the model (7) – (9) is extended by incorporating the compartment of infectious individuals receiving treatment, $T(t)$ so that

$$N(t) = S(t) + I(t) + T(t) + A(t) \tag{13}$$

We also incorporate three control interventions. The first control efforts is preventive, $u_1$, represent condom use. The second control efforts $u_2$, represent pre-sex testing and the third efforts $u_3$, represents treatment of infectious individuals yet to develop symptoms and infectious individuals who have developed symptoms.

After incorporating the controls into the basic HIV model, we get

$$\frac{dS}{dt} = \Delta - \lceil(1-u_1) + (1-u_2)\rceil \lambda S - \mu_h S \tag{14}$$

$$\frac{dI}{dt} = \lceil(1-u_1) + (1-u_2)\rceil \lambda S - (k + u_3 + u_h)I \tag{15}$$

$$\frac{dT}{dt} = u_3 I + u_3 A - (u_h + \delta_1)T \tag{16}$$

$$\frac{dA}{dt} = kI - (u_3 + u_h + \delta_2)A \tag{17}$$

where $\lambda = \beta_1 I + \beta_2 T + \beta_3 A$

The cost objective function is given by

$$J(u_1, u_2, u_3) = \int_0^t \left[A_1 u_1 S + A_2 u_2 S + A_3 I + A_4 u_3 (T+A) + \frac{1}{2}(B_1 u_1^2 + B_2 u_2^2 + B_3 u_3^2)\right] dt \tag{18}$$

where $U = (u_1, u_2, u_3)$ is a set of Lebesgue measurable functions. The variables/parameters are described in Table 2.

**Table 2:** Variables and Parameters used in the extended HIV model and their description

| Variable/Parameter | Description |
|---|---|
| $S(t)$ | The number of susceptible prove to HIV infection at time t |
| $I(t)$ | The number of infectious individuals yet to develop symptoms at time t |
| $T(t)$ | The number of infectious individuals receiving treatment at time t |
| $A(t)$ | The number of those who have developed AIDS at time t |
| $\Delta$ | Recruitment rate |
| $\beta_1, \beta_2, \beta_3$ | Effective contact rates for HIV infection transmission |

| | |
|---|---|
| $\kappa$ | Progression rate of infectious individuals to AIDS |
| $\mu_h$ | Natural mortality rate |
| $\mathrm{u}_1$ | Control effort of condom use |
| $u_2$ | Control effort of pre-sex HIV testing |
| $u_3$ | Control effort of treatment with antiretroviral drugs |
| $\delta_1$ | Diseased-induced mortality rate in the T-class |
| $\delta_2$ | Diseased-induced mortality rate in the A-class |
| $\mathrm{A}_1$ | Cost of condom per person per day |
| $\mathrm{A}_2$ | Cost of HIV test per person |
| $\mathrm{A}_3$ | Cost of treatment per person per day |
| $\mathrm{A}_4$ | Cost of HIV-related illness per person per day |
| $\mathrm{B}_1$ | Cost of implementation of condom per day |
| $\mathrm{B}_2$ | Cost of implementation of HIV testing per day |
| $\mathrm{B}_3$ | Cost of implementation of HIV treatment per day |

## 4.2. Optimal Control Analysis

In order to find the optimal solution, we define the Lagrangian and the Hamiltonian of our control problem (14) – (17). The Lagrangian of the optimal control problem is given by

$$L = \left[A_1u_1S + A_2u_2S + A_3I + A_4u_3(T + A) + \frac{1}{2}\left(B_1u_1^2 + B_2u_2^2 + B_3u_3^2\right)\right] \qquad (19)$$

We introduce the adjoint variables $\lambda_i, i = 1, \dots, 4$.

Therefore, the Hamiltonian $H$ is the sum of $L$ and the inner product of the adjoint variables $\lambda_i$ and the right hand sides of equations $(14) - (17)$

that is

$H = L + \sum_{i=1}^{6} \lambda_i f_i$ are the right-hand side of $(14) - (17)$

$$= \left[A_1u_1S + A_2u_2S + A_3I + A_4u_3(T + A) + \frac{1}{2}\left(B_1u_1^2 + B_2u_2^2 + B_3u_3^2\right)\right]$$
$$+\lambda_1[\Delta - \lceil(1 - u_1) + (1 - u_2)\rceil \lambda S - \mu_h S]$$
$$+\lambda_2\lceil(1 - u_1) + (1 - u_2)\rceil \lambda S - (k + u_3 + u_h)I] + \lambda_3[u_3I + u_3A - (u_h + \delta_1)T]$$
$$+\lambda_4 kI - (u_3 + u_h + \delta_2)A]$$

### 3.1 The Optimality System

Suppose $U = (u_1, u_2, \mathrm{u}_3)$ is a control vector, $x = (S, I, T, A)$ the state variables of the system $(14) - (17)$ and $H$ the Hamiltonian, then the optimality system is given by

$$\frac{dx_i}{dt} = \frac{\partial H}{\partial \lambda_i}, \quad -\frac{\mathrm{d}\lambda_i}{dt} = \frac{\partial H}{\partial x_i}, \mathrm{i} = 1, \dots, 4$$

with transversality conditions $\lambda_i(tf) = 0; \frac{\partial H}{\partial u_j} = 0, \mathrm{j} = 1, 2, 3.$

**Theorem 2.** Let $U = (u_1, u_2, u_3)$ be a control vector, $x = (S, I, T, A)$ be the state variables of the system $(7) - (10)$ and H the Hamiltonian. There exist an optimal control vector $U^*(t)$ and the corresponding state vector $x^*(t)$ that minimize $J(U)$, given by equation (18) over $\Omega$. Furthermore, there exist adjoint functions $\lambda_i$ satisfying the equations

$-\frac{d\lambda_i}{dt} = \frac{\partial H}{\partial x_i}$, i $= 1, \dots, 4$ with transversality conditions $\lambda_i(tf) = 0$.
In addition, the optimal controls are given by $u_j^* = \max\{0, \min(1, R_j)\}, j = 1, 2, 3$.

Proof: We use the recipe by Fleming and Rishel (1975). The existence of an optimal control vector follows from the convexity of the integrand $J$ with respect to $U$, a priori boundedness of the state solutions and the Lipscitz property of the state solutions with respect to the state variables. See [Fleming and Rishel (1975)] (corollary 4.1). The adjoint equations and transversality conditions can be obtained by using the Pontryagin's Maximum Principle (Mardanov and Sharifov, 2015) such that

$$-\frac{d\lambda_1}{dt} = \frac{\partial H}{\partial S} = -\lambda_1\{\lceil(1 - u_1) + (1 - u_2)\rceil\lambda + u_h\} + \lambda_2\lceil(1 - u_1) + (1 - u_2)\rceil\lambda + A_1u_1 + A_2u_2$$

$$-\frac{d\lambda_2}{dt} = \frac{\partial H}{\partial I} = A_3 - \lambda_1\lceil(1 - u_1) + (1 - u_2)\rceil\beta_1 S + \lambda_2\lceil(1 - u_1) + (1 - u_2)\rceil\beta_1 S - (k + u_3 + u_h) + \lambda_3 u_3 + \lambda_4 k$$

$$-\frac{d\lambda_3}{dt} = \frac{\partial H}{\partial T} = A_4u_3 - \lambda_1\lceil(1 - u_1) + (1 - u_2)\rceil\beta_2 S + \lambda_2\lceil(1 - u_1) + (1 - u_2)\rceil\beta_2 S - \lambda_3(u_2 + \delta_1)$$

$$-\frac{d\lambda_4}{dt} = \frac{\partial H}{\partial A} = A_4u_3 - \lambda_1\lceil(1 - u_1) + (1 - u_2)\rceil\beta_3 S + \lambda_2\lceil(1 - u_1) + (1 - u_2)\rceil\beta_3 S - \lambda_4(u_3 + u_h + \delta_2) + \lambda_3 u_3$$

The Optimality conditions are given by

$$u_1^* = \max\{0, \min(1, R_1)\}, R_1 = \frac{(\lambda_2 - \lambda_1)\lambda S - A_1 S}{B_1}$$

$$u_2^* = \max\{0, \min(1, R_2)\}, R_2 = \frac{(\lambda_2 - \lambda_1)\lambda S - A_2 S}{B_2}$$

$$u_3^* = \max\{0, \min(1, R_3)\}, R_3 = \frac{(\lambda_2 - \lambda_3)\mathrm{I} + (\lambda_4 - \lambda_3)A - A_4(T + A)}{B_3}$$

Therefore, the optimality system consists of the state equations, the adjoint equations, the optimality conditions and the transversality conditions.

## 5. Numerical Simulation and Results

For numerical simulations, we made use of the parameter values in Table 2 and obtained numerical results in Figures 1 through 7.

**Table 2**

| Parameter | Value | Source |
|---|---|---|
| $\Delta$ | 1.56 | estimated |
| $\beta_1$ | 0.0000003175 | estimated |
| $\beta_2$ | 0.0000001 | estimated |
| $\beta_3$ | 0.0000004185 | estimated |
| $k$ | 0.0002283 | estimated |
| $u_h$ | 0.00005447 | estimated |
| $\delta_1$ | 0.0001 | estimated |
| $\delta_2$ | 0.0002 | estimated |
| $A_1$ | \$0.5 | estimated |
| $A_2$ | \$2 | estimated |
| $A_3$ | \$6 | estimated |
| $A_4$ | \$10 | estimated |
| $B_1$ | \$50 | estimated |
| $B_2$ | \$50 | estimated |
| $B_3$ | \$50 | estimated |
| $S(0)$ | 1500 | assumed |
| $I(0)$ | 20 | assumed |
| $T(0)$ | 0 | assumed |
| $A(0)$ | 1 | assumed |

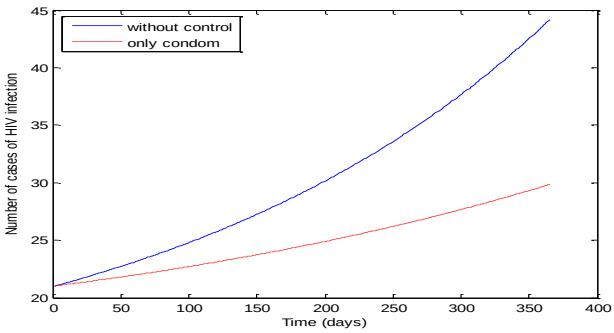


Figure 2: Plot of the population of HIV- infected persons using condom only and without control measure.

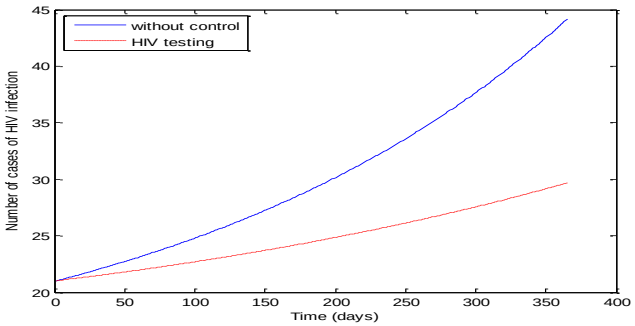


Figure 3:Plot of the population of HIV-infected persons with HIV testing

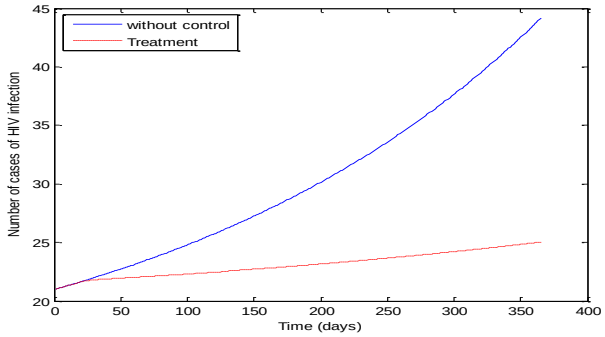


Figure 4: Plot of the population of HIV-infected persons in the presence of treatment

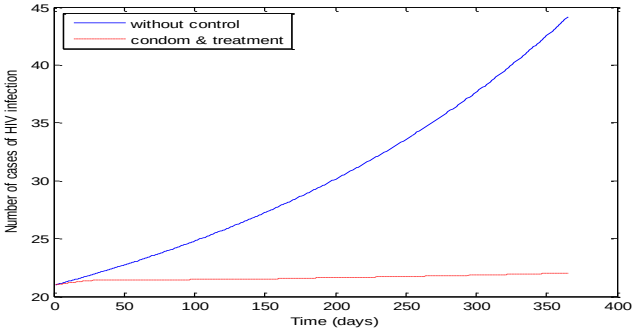


Figure 5:Plot of the population of HIV-infected persons in the presence of condom use and treatment.

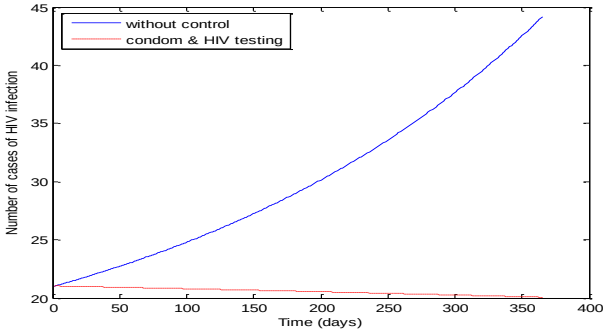


Figure 6: Plot of the population of HIV-infected persons in the presence of condom use and HIV testing

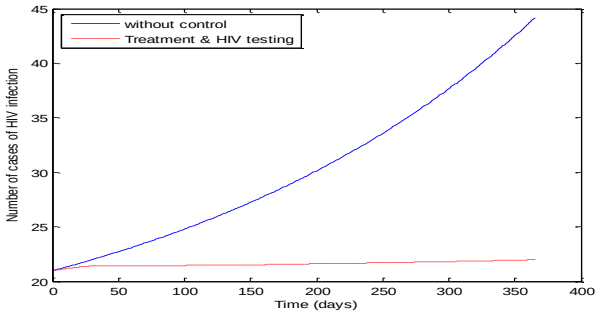


Figure 7: Plot of the population of HIV-infected persons in the presence of treatment and HIV testing.

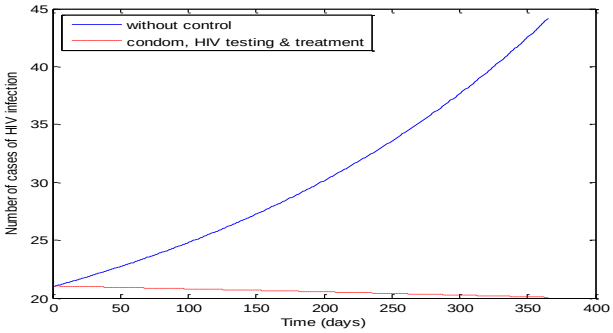


Figure 8: Plot of the population of HIV-infected persons in the presence of condom use, HIV testing and treatment.

## 6. Discussion

A deterministic model for the dynamics of HIV is proposed and this section discusses the analytical and numerical results of the study. The qualitative analysis of the model solution is bounded and the local as well as global stability of the disease free equilibrium (DFE) state was established. The HIV model has a globally asymptotically stable (GAS) disease-free (DFE) whenever the basic reproduction number, $R_0$, is less than unity. The basic reproduction number, given by $R_0 = \frac{\beta_1 \Delta}{\mu_h(\kappa+\mu_h)} + \frac{\beta_1 \Delta \kappa}{\mu_h(\kappa+\mu_h)(\delta_2+\mu_h)}$ determines whether the disease can spread or not. Also the analytical result of the optimal control model can be seen in theorem 2. Theorem 2 guarantees the existence of the optimality system for the optimal control model.

The results from the numerical experiments can be seen in Figures 2 through Figure 8. Figure 2 shows the impact of the use of condom as a control function on the dynamics of infectives over the time period. The result shows that condom usage has the capability of significantly reducing new infection. Since it is not a curative measure, the number of infectives slowly decreases. Figure 3 shows the impact of the HIV status testing as a control function on the dynamics of infectives over the time period. The result shows that HIV status testing has the capability of significantly reducing new infections in the same measure as condom usage. Figure 4 depicts the impact of treatment alone as a control measure on the dynamics of infective persons. The result shows that treatment, though a palliative measure can reduce the number of new infections because of its power in suppressing the viral load and inhibiting the growth of viral cells. The impacts of double interventions can be seen in Figures 5 through Figure 7. The results show that combination of condom and HIV status test before intercourse as a strategy is able to reduce HIV/AIDS

transmission. However, the combination of the three controls plays a key strategy in reducing HIV/AIDS burden in a community.

## 7. Conclusion

In this paper, we develop a deterministic model for the dynamics of HIV, where the heterosexual population is compartmentalized into four classes namely: susceptible people who are prone to HIV infection, the infectious individuals who are yet to develop symptoms, infectious individuals receiving treatment and infectious individuals who have developed AIDS. The qualitative analysis of the basic model solution is bounded and the local as well as global stability of the disease free equilibrium (DFE) state were established.

Using Pontryagin's maximum principle, the optimal control problem is formulated and the conditions for the optimal control of the disease are analyzed and the existence of the optimal control was established.

The extended model assesses the impact of time preventives (condom use, pre-sex HIV testing and treatment of infectious individuals yet to develop symptoms and infectious individuals who have developed symptoms). The numerical results showed that combination of condom and HIV status test before intercourse as a strategy is able to reduce HIV/AIDS transmission. However, the combination of the three controls plays a key strategy in reducing HIV/AIDS burden in a community.

Therefore, the findings of the study suggest that the combination of the three controls as a strategy is the optimal strategy and should be adopted.

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