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OPTIMAL PREVENTION OF HIV-AIDS WITH EMPHASIS ON UNPROTECTED AND UNNATURAL CANAL ACTIVITIES: A DETERMINISTIC MODELLING PERSPECTIVE

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Abstract. HIV accounts for more than thirty three million death and approximately thirty eight million infected cases since it's inception. The disease unfolds in three stages; Chronic, Acute and fully blown AIDS. Adhering to preventive protocols such as use of condoms, preventing oneself from unprotected sex and limiting one's sexual partners could help minimize the disease spread. In this study, a deterministic model for HIV-AIDS is formulated. The equilibrium points, local and global stability of the equilibrium points, and HIV reproductive rate were determined and interpreted. The model was extended to optimal control by simulating the optimality system. This was done by incorporating the use of condoms and education of susceptible population as intervention strategies. It was established that the best and most effective control strategy was optimal education and sensitisation of susceptible population.

Keywords: HIV-model; HIV reproductive rate; equilibrium points; stability; unnatural canal activities.

2020 AMS Subject Classification: 92C60.

1. INTRODUCTION

The viral infection that thrive in the system of another living organism, especially humans, was detected in 1981 in the blood-streams of mostly gay men. The disease is identified with

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strains: HIV-1 and HIV-2. The HIV-1 is recognized as the lethal strain, causing pandemic in humans. The disease is transmissible when a contact of susceptible human is made with the infected person through unprotected and unnatural canal activity.

HIV accounts for more than 33 million death and 37.7 million infected cases since the disease's inception [1]. The disease unfolds in three stages; Chronic , Acute and full blown AIDS. The acute stage is characterized by rash, headache and fever. The acute stage is noted as the first two weeks to one month where the transmission of virus is very high, and one could easily get infected when in contact with the infected. The chronic stage is characterize by a decrease in the virus replication as the infected enters into clinical latency stage, but one can still get infected with the disease since it could be transferred by the infected. The last stage of the disease: AIDS happens when the person's immune system is substantially weakened and can no longer defend the body against foreign attacking pathogens.

Adhering to the preventive protocols such as use of condoms, preventing oneself from unprotected sex and limiting one's sexual partners could help minimize the number of the infected. However, the use of pharmaceutical drugs such as antiretroviral therapy (ART) may help reduce the multiplication of the virus and the swift progressing of the disease [2].

[3] proposed a model for the transition period of HIV/AIDS incorporated of weak uninfected $CD4^+$ cells as T(t). For a weak uninfected $CD4^+$ T cell, the authors estimate a very short transition period. As a result, when weak $CD4^+$ T-cells engage with HIV, some of these weak CD4+ T-cells shift directly into the viral class, which is a key factor in the fast spread of HIV. Another important finding was that the natural recovery of $CD4^+$ T-cells cannot be overlooked because a large proportion of T cells have recovered. The results of the study through numerical technique using confirmed the analytical results of the model as several unmeasured parameter values were assumed and used.

[4] used seven-dimensional nonlinear ordinary differential equation to establish a mathematical model to analyze the spread of HIV epidemic within an antiretroviral therapy (ART) treatment as an alternative intervention. In absence of antiretroviral therapy (ART) treatment in the model showed transition rate among infected compartment reduced. However, model analysis showed sensitivity of the antiretroviral therapy treatment to the basic reproduction number along the

numerical simulation. The findings of the study showed stationary in the number of susceptible humans, leading to a reduction in the number of infected individual who progressed to AIDS as a result of antiretroviral therapy treatment.

[5] considered new deterministic mathematical model for the transmission dynamics of HIV/AIDS virus on the role of female sex workers in India. The study employed homotopy perturbation method to derive an analytical solution to each nonlinear deterministic system containing initial condition for those individual sub-groups. The analytical solution was compared with numerical solution obtained by MATLAB function; fourth order Runge-Kutta method. The analytical results obtained can run sensitivity analysis of the estimated parameters to better understand the spread mechanism of HIV/AIDS and suggest possible prevention strategies.

[6] developed new HIV/AIDS deterministic model with compartments categorised into aware and unaware susceptible individuals, diagnosed and undiagnosed HIV infections. The model however considered the rate of recruitment of individuals in aware and unaware susceptible population as a function of engagements through latest media campaign like twitter, tiktok, telegram, Facebook, etc. while keeping other variables constant.

Epidemiological models generally explain the transmission dynamics of diseases and can determine the status of infections with time. Models that are incorporated with some control can determine the best optimal control strategy in combating infections [7, 8, 9, 10].

1.1. HIV-AIDS DATA.

TABLE 1. **Number of people tested for viral suppression among those on treatment**

Dis-aggregation	**2019**	**2020**	**2021**	**2022**
Children (0-14 yrs)	4,651	4,734	4,827	4,842
Males (15+ yrs)	21,393	21,778	22,163	22,555
Females (15+ yrs)	66,969	68,175	69,381	70,597
Total	93,013	94,687	96,371	97,994

Source: Ghana Health Service

TABLE 2. **Number of people living with HIV with suppressed viral load**

Dis-aggregation	**2019**	**2020**	**2021**	**2022**
Children (0-14 yrs)	3,176	3,455	3,734	4,023
Males (15+ yrs)	14,611	15,891	17,181	18,471
Females (15+ yrs)	45,739	49,745	53,551	57,367
Total	63,526	69,091	74,466	79,861

Source: Ghana Health Service

TABLE 3. **HIV population, new infections and total deaths**

Year	**Populations**	**New infections**	**AIDS Death**
2019	342,054	21,206	15,922
2020	346,120	18,928	12,758
2021	349,362	15,323	9,886
2022	352,498	12,383	6,974

Source: Ghana Health Service

2. MODEL FORMULATION

Model divides the total population under study into five compartment of Susceptible, S, Exposed, E_H, Fully blown HIV, A_H, Infected HIV, I_H and Treatment, T_H. Recruitment into the susceptible population is denoted by the rate Λ. λ is the rate at which the exposed individuals leaves the exposed compartment to the infected compartment. Further, the model assumes that individuals die as a result of the disease at a rate δ. β is the transmission rate as a results of contact between susceptible, infected and fully blown HIV individuals. The model assumes that infected individuals seek medical attention at a rate γ, while a fraction of the infected individuals progressed to fully blown HIV status. Fully blown HIV individuals also seek treatment at a rate σ. μ is the natural death rate. Table 4 and Table 5 shows the parameters, variables, and their descriptions used in the model formulation.

TABLE 4. **HIV Model and Parameter description**

Parameter	Description
Λ	The rate at which individuals enter the susceptible population
λ	The transmission rate
β	Recruitment into the infected component.
α	Recruitment into the treated compartment
$(1-\alpha)$	Rate at which people enter fully blown HIV compartment
μ	natural death rate
δ	HIV death rate.

TABLE 5. **HIV Model variable description**

Variables	Description
S	Susceptible HIV population.
E_H	Exposed HIV individuals.
I_H	Infected HIV individuals.
T_H	Treated HIV individuals
R_H	Recovered HIV individuals

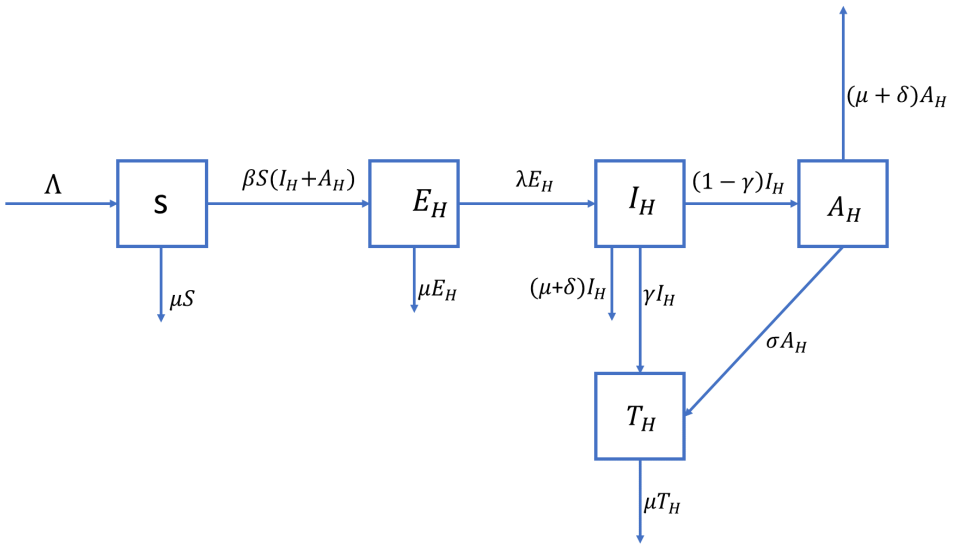

FIGURE 1. Schematic diagram of the HIV Model

Hence, the system differential equations describing the HIV model in Figure 1 is given by

$$
(1)\qquad \begin{cases} \frac{d}{dt}S = \Lambda - \beta S(I_H + A_H) - \mu S \\ \frac{d}{dt}E_H = \beta S(I_H + A_H) - (\mu + \lambda)E_H \\ \frac{d}{dt}I_H = \lambda E_H - (1-\gamma)I_H - (\mu + \delta + \gamma)I_H \\ \frac{d}{dt}T_H = \gamma I_H - \mu T_H + \sigma A_H \\ \frac{d}{dt}A_H = (1-\gamma)I_H - (\mu + \delta + \sigma)A_H \end{cases}
$$

3. Model analysis

3.1. Positivity. The positivity of variables in the model was proven based on the following theorem;

Theorem 3.1. *Let the initial values be $S(0)$, $E_c(0)$, $I_c(0)$, $T_c(0)$, $R_C(0)$ and $R_c(0)$ be non-negative, then the solution set of $\{S(t), E_c(t), I_c(t), T_c(t)$ and$R_c(t)\}$ of* **??** *is positive and bounded for all $t > 0$, whenever they exist.*

Proof.

$$
\begin{aligned}
\frac{ds}{dt} &= \Lambda - \beta S(I_h + A_h) - \mu S \\
\frac{ds}{dt} &\geq -(\beta I_H + \beta A_H + \mu)S \\
\int \frac{ds}{dt} &\geq -\int (\beta I_H + \beta A_H + \mu)dt \\
lnS &\geq -(\beta I_H + \beta A_H + \mu)t + c \\
At \quad t = 0, S = S_0 & \\
lnS_0 &\geq c \\
lnS &\geq -(\beta I_H + \beta A_H + \mu)t + lnS_0 \\
ln\left(\frac{S}{S_0}\right) &\geq (\beta I_H + \beta A_H + \mu)t \\
\frac{S}{S_0} &\geq e^{-(\beta I_H + \beta A_H + \mu)t}
\end{aligned}
$$

$$\begin{aligned} As \quad t \to \infty & \\ \frac{S}{S_0} &\geq 0 \\ S &\geq 0 \end{aligned}$$

Doing same for the entire compartments gives

$$\begin{aligned} E_H &\geq 0 \\ I_H &\geq 0 \\ T_H &\geq 0 \\ A_H &\geq 0. \end{aligned}$$

□

3.2. Region of feasibility.

Theorem 3.2. *The positive solution is a positively invariant set of the model and is given by* $\varphi = \{S, E_H, I_H, A_H, T_H, \in R_+^5 : N \leq \frac{\Lambda}{\mu}, \mu \neq 0\}$

$$N = S + E_H + I_H + A_H + T_H \tag{2}$$

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE_H}{dt} + \frac{dI_H}{dt} + \frac{dA_H}{dt} + \frac{dT_H}{dt} \tag{3}$$

$$\frac{dN}{dt} = \Lambda - \mu N - \delta I_H - \theta A_H \tag{4}$$

$$\begin{aligned} \frac{dN}{dt} &\leq \Lambda - \mu N \\ \frac{dN}{\Lambda - \mu N} &\leq dt \\ \frac{dN}{-\mu\left(N - \frac{\Lambda}{\mu}\right)} &\leq dt \\ \frac{dN}{\left(N - \frac{\Lambda}{\mu}\right)} &\leq -\mu dt \\ \int \frac{dN}{\left(N - \frac{\Lambda}{\mu}\right)} &\leq \int -\mu dt \\ ln\left(N - \frac{\Lambda}{\mu}\right) &\leq -\mu dt + c \end{aligned}$$

$$
\begin{aligned}
&At \quad t=0, N=N_0\\
&ln\left(N_0-\frac{\Lambda}{\mu}\right) quad \leq \quad c\\
&ln\left(N-\frac{\Lambda}{\mu}\right) \quad \leq \quad -\mu t+ln\left(N_0-\frac{\Lambda}{\mu}\right)\\
&ln\left(\frac{N-\frac{\Lambda}{\mu}}{N_0-\frac{\Lambda}{\mu}}\right) \quad \leq \quad -\mu t\\
&\left(\frac{N-\frac{\Lambda}{\mu}}{N_0-\frac{\Lambda}{\mu}}\right) \quad \leq \quad e^{-\mu t}\\
&As \quad t\to\infty\\
&\left(\frac{N-\frac{\Lambda}{\mu}}{N_0-\frac{\Lambda}{\mu}}\right) \quad \leq \quad 0\\
&N-\frac{\Lambda}{\mu} \quad \leq \quad 0\\
&N \quad \leq \quad \frac{\Lambda}{\mu} \qquad (5)
\end{aligned}
$$

Therefore, the positive solution set is an invariant set of the model and is given by

$$\varphi=\left(S,E_H,I_H,A_H,T_H\ \varepsilon\ R_+^s:\ N\leq\frac{\Lambda}{\mu}\right). \qquad (6)$$

3.3. Disease-free equilibrium. The disease free equilibrium points of the HIV model is given by $f_0=(S_0,E_{H_0},I_{H_0},T_{H_0},A_{H_0})=(\frac{\Lambda}{\mu},0,0,0,0)$.

3.4. HIV reproductive rate, R_0. Using the approach in [11, 12, 13, 14, 15, 16], the infection compartments are as follows

$$
\begin{aligned}
\frac{d}{dt}E_H &= \beta S(I_H+A_H)-(\mu+\lambda)E_H\\
\frac{d}{dt}I_H &= \lambda E_H-(1-\gamma)I_H-(\mu+\delta+\gamma)I_H\\
\frac{d}{dt}T_H &= \gamma I_H-\mu T_H+\sigma A_H \qquad (7)\\
\frac{d}{dt}A_H &= (1-\gamma)I_H-(\mu+\delta+\sigma)A_H
\end{aligned}
$$

The matrices F and V are generated from the infection compartments 7 as

$$\mathscr{F} = \begin{pmatrix} \beta S(I_H + A_H) \\ 0 \\ 0 \\ 0 \end{pmatrix}, \quad \mathscr{V} = \begin{pmatrix} (\mu+\lambda)E_H \\ (\mu+\delta+\gamma)I_H + (1-\gamma)I_H - \lambda E_H \\ \mu T_H - \gamma I_H - \sigma A_H \\ (\mu+\delta+\sigma)A_H - (1-\gamma)I_H \end{pmatrix} \tag{8}$$

The Jacobian of matrix F is given by

$$\mathscr{F} = \begin{pmatrix} 0 & \beta S & \beta S & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{9}$$

Evaluating matrix F at the disease-free equilibrium $f_0 = (S_0, E_{H_0}, I_{H_0}, T_{H_0}, A_{H_0}) = (\frac{\Lambda}{\mu}, 0, 0, 0, 0)$ gives the corresponding matrix

$$\mathscr{F} = \begin{pmatrix} 0 & \beta\frac{\Lambda}{\mu} & \beta\frac{\Lambda}{\mu} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{10}$$

Similarly, finding the Jacobian of matrix v gives

$$\mathscr{V} = \begin{pmatrix} (\mu+\lambda) & 0 & 0 & 0 \\ -\lambda & (\mu+\delta+\gamma)+(1-\gamma) & 0 & 0 \\ 0 & -\gamma & -\sigma & \mu \\ 0 & -(1-\gamma) & 0 & (\mu+\delta+\sigma) \end{pmatrix} \tag{11}$$

When $\mathscr{V}$ is evaluated at the disease-free equilibrium $f_0 = (S_0, E_{H_0}, I_{H_0}, T_{H_0}, A_{H_0}) = (\frac{\Lambda}{\mu}, 0, 0, 0, 0)$, we get

$$\mathscr{V} = \begin{pmatrix} (\mu+\lambda) & 0 & 0 & 0 \\ -\lambda & (\mu+\delta+\gamma)+(1-\gamma) & 0 & 0 \\ 0 & -\gamma & -\sigma & \mu \\ 0 & -(1-\gamma) & 0 & (\mu+\delta+\sigma) \end{pmatrix} \tag{12}$$

The inverse of v matrix, $v^{-\prime}$

$$v^{-\prime} = \begin{pmatrix} \frac{-1}{a} & 0 & 0 & 0 \\ -\frac{b}{ac} & \frac{1}{c} & 0 & 0 \\ \frac{(bdh-bfg)}{aceh} & -\frac{(dh-fg)}{ceh} & \frac{1}{e} & -\frac{f}{eh} \\ \frac{bd}{ach} & -\frac{g}{ch} & 0 & \frac{1}{h} \end{pmatrix} \tag{13}$$

with

$a=(\mu+\lambda), b=\lambda, c=(\mu+\delta+\gamma)+(1-\gamma), d=\gamma, e=\sigma, f=\mu, g=(1-\gamma), h=(\mu+\delta+\sigma)$

therefore

$$FV^{-\prime} = \begin{pmatrix} 0 & \beta\frac{\Lambda}{\mu} & \beta\frac{\Lambda}{\mu} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{-1}{a} & 0 & 0 & 0 \\ -\frac{b}{ac} & \frac{1}{c} & 0 & 0 \\ \frac{(bdh-bfg)}{aceh} & -\frac{(dh-fg)}{ceh} & \frac{1}{e} & -\frac{f}{eh} \\ \frac{bd}{ach} & -\frac{g}{ch} & 0 & \frac{1}{h} \end{pmatrix} \tag{14}$$

(15)

$$R_0 = \frac{\beta\Lambda\lambda}{\mu(\mu+\lambda)\big((\mu+\delta+\gamma)+(1-\gamma)\big)} + \frac{\beta\Lambda(\lambda\gamma(\mu+\delta+\sigma)-(\lambda\mu(1-\gamma))}{\mu(\lambda+\mu)((\mu+\delta+\gamma)+(1-\gamma))\sigma(\mu+\delta+\sigma)}$$

3.5. Endemic Equilibrium. Considering the model equation 1, an endemic equilibrium exist when S, E_H, I_H, T_H andA_H are not equal to zero. The endemic equilibrium $h_1 = (S^*, E_H^*, I_H^*, T_H^*, A_H^*)$ is therefore given as

$$S^* = \frac{\Lambda}{\big(\beta(I_H^*+A_H^*)+\mu\big)}$$

$$E_H^* = \frac{\beta S^*(I_H^*+A_H^*)}{(\mu+\lambda)}$$

$$I_H^* = \frac{\lambda E_H^*}{(1-\gamma)+(\mu+\delta+\gamma)}$$

$$T_H^* = \frac{\gamma I_H^* + \sigma A_H^*}{\mu}$$

$$A_H^* = \frac{(1-\gamma)I_H^*}{\mu+\delta+\sigma}$$

Local stability of the disease free equilibrium. The section presents the stability analysis of model equation 1 at the disease-free equilibrium. The linearization method is adopted in studying the asymptomatic stability of model equation 1 at the disease-free equilibrium [17, 18].

The Jacobian matrix at disease free equilibrium becomes

$$J_B = \begin{pmatrix} -\beta(I_H+A_H)-\mu & 0 & -\beta S & 0 & -\beta S \\ 0 & -(\mu+\lambda) & \beta S & 0 & \beta S \\ 0 & \lambda & -(1-\gamma)-(\mu+\delta+\gamma) & 0 & 0 \\ 0 & 0 & \gamma & -\mu & \sigma \\ 0 & 0 & (1-\gamma) & 0 & -(\mu+\delta+\sigma) \end{pmatrix} \tag{16}$$

When J_B is evaluated at the disease-free equilibrium point $f_0 = (S_0, E_{H_0}, I_{H_0}, T_{H_0}, A_{H_0}) = (\frac{\Lambda}{\mu}, 0, 0, 0, 0)$, we get

$$(J_{B_1}) = \begin{pmatrix} -\mu & 0 & -\beta\frac{\Lambda}{\mu} & 0 & -\beta\frac{\Lambda}{\mu} \\ 0 & -(\mu+\lambda) & \beta\frac{\Lambda}{\mu} & 0 & \beta\frac{\Lambda}{\mu} \\ 0 & \lambda & -(1-\gamma)-(\mu+\delta+\gamma) & 0 & 0 \\ 0 & 0 & \gamma & -\mu & \sigma \\ 0 & 0 & (1-\gamma) & 0 & -(\mu+\delta+\sigma) \end{pmatrix} \tag{17}$$

Hence

(18)
$$(J_{B_1}-l)=\begin{pmatrix} -\mu-l & 0 & -\beta\dfrac{\Lambda}{\mu} & 0 & -\beta\dfrac{\Lambda}{\mu} \\ 0 & -(\mu+\lambda)-l & \beta\dfrac{\Lambda}{\mu} & 0 & \beta\dfrac{\Lambda}{\mu} \\ 0 & \lambda & -\big((1-\gamma)+(\mu+\delta+\gamma)\big)-l & 0 & 0 \\ 0 & 0 & \gamma & -\mu-l & \sigma \\ 0 & 0 & (1-\gamma) & 0 & -(\mu+\delta+\sigma)-l \end{pmatrix}$$

Clearly, $l_1=-\mu$, $l_2=-\mu$.

Matrix A is the remaining matrix of $(J_{B_1}-l)$, given by

(19)
$$\begin{pmatrix} -(\mu+\lambda)-l & \beta\dfrac{\Lambda}{\mu} & \beta\dfrac{\Lambda}{\mu} \\ \lambda & -\big((1-\gamma)+(\mu+\delta+\gamma)\big)-l & 0 \\ 0 & (1-\gamma) & -(\mu+\delta+\sigma)-l \end{pmatrix}$$

Referring to 19, the model system 1 is locally unstable since according to Gershgorin circle, matrix 1 should be diagonally dominant matrix. But $|-(\mu+\lambda)-l| \not> \beta\dfrac{\Lambda}{\mu}$.

3.6. Global Stability of the Disease-free equilibrium. We investigate the global asymptotic stability of the model system 1 by using the Castillo-Chavez's method. This is presented as follows;

Consider

(20)
$$\begin{aligned} \frac{dp_1}{dt} &= B(p_1,p_2), \\ \frac{dp_2}{dt} &= C(p_1,p_2), \end{aligned}$$

with p_1 and p_2 denote number of uninfected and infected individuals respectively. Thus, we denote $p_1=\big(S\big)\in R^2$ and $p_2=\big(E_H,I_H,T_H,A_H\big)\in R^4$. The disease-free equilibrium f_0 for the

model system (1) is given by $f_0 = (p_1^0, 0)$. Thus, the global stability at D_0 exists based on these conditions

- Given $\frac{dp_1}{dt} = Y(p_1, 0), z_1^0$ is globally asymptotically stable.
- $X(p_1, p_2) = Dw_2 - \hat{\mathrm{C}}(p_1, p_2)$, where $\hat{\mathrm{C}}(p_1, p_2) \geq 0$ for $(p_1, p_2) \in \in$,

where $D = Wy_2C(z_1^0, 0)$ is an M-matrix, with a positive off-diagonal entries and τ is the feasible biological region of model (1). When the above conditions are satisfied by model system (1), then the underlying theorem holds.

Theorem 3.3. *When $R_0 < 1$ and the above two conditions are satisfied by model (1), then, the equilibrium point $f_0 = (p_1^0, 0)$ is globally asymptotically stable.*

Proof. From model (1), we can deduce

$$\begin{cases} \frac{dp_1}{dt} = Y(p_1, p_2) \\ \frac{dp_1}{dt} = \Big(\Lambda - \beta S(I_H + A_H) - \mu S\Big), \end{cases} \tag{21}$$

Hence $Y(p_1, 0)$ becomes,

$$H(y_1, 0) = \Big(\Lambda - \mu S_0\Big),$$

$$X(p_1, p_2) = \begin{Bmatrix} \beta S(I_H + A_H) - (\mu + \lambda)E_H \\ \lambda E_H - (1-\gamma)I_H - (\mu + \delta + \gamma)I_H \\ \gamma I_H - \mu T_H + \sigma A_H \\ (1-\gamma)I_H - (\mu + \delta + \sigma)A_H \end{Bmatrix}$$

The Jacobian of $X(p_1, p_2)$ is given by

$$J_{df} = \begin{Bmatrix} -(\mu + \lambda) & \beta S & 0 & \beta S \\ \lambda & -((1-\gamma) + (\mu + \delta + \gamma)) & 0 & 0 \\ 0 & \gamma_2 & -\mu & \sigma \\ 0 & (1-\gamma) & 0 & -(\mu + \delta + \sigma) \end{Bmatrix} \tag{22}$$

Hence using the expression

$$X(p_1, p_2) = Dw_2 - \tilde{W}(p_1, p_2) \tag{23}$$

we deduce the following

$$X(p_1,p_2)=\begin{Bmatrix} -(\mu+\lambda) & \beta S_0 & 0 & \beta S_0 \\ \lambda & -((1-\gamma)+(\mu+\delta+\gamma) & 0 & 0 \\ 0 & \gamma_2 & -\mu & \sigma \\ 0 & (1-\gamma) & 0 & -(\mu+\delta+\sigma) \end{Bmatrix} \begin{Bmatrix} E_H \\ I_H \\ T_H \\ A_H \end{Bmatrix} - \begin{Bmatrix} D_1\tilde{w}(p_1,p_2) \\ D_2\tilde{w}(p_1,p_2) \\ D_3\tilde{w}(p_1,p_2) \\ D_4\tilde{w}(p_1,p_2) \end{Bmatrix}$$

Applying the equation 23, and solving for the expression $\tilde{w}(p_1,p_2)$ gives

$$\begin{Bmatrix} \beta S(I_H+A_H)-(\mu+\lambda)E_H \\ \lambda E_H-(1-\gamma)I_H-(\mu+\delta+\gamma)I_H \\ \gamma I_H-\mu T_H+\sigma A_H \\ (1-\gamma)I_H-(\mu+\delta+\sigma)A_H \end{Bmatrix} - \begin{Bmatrix} \beta S_0(I_H+A_H)-(\mu+\lambda)E_H \\ \lambda E_H-(1-\gamma)I_H-(\mu+\delta+\gamma)I_H \\ \gamma I_H-\mu T_H+\sigma A_H \\ (1-\gamma)I_H-(\mu+\delta+\sigma)A_H= \end{Bmatrix} = \begin{Bmatrix} D_1\tilde{w}(p_1,p_2) \\ D_2\tilde{w}(p_1,p_2) \\ D_3\tilde{w}(p_1,p_2) \\ D_4\tilde{w}(p_1,p_2) \end{Bmatrix}$$

$$\tilde{w}(p_1,p_2)=\begin{Bmatrix} \beta I_H(S_0-S) & \beta A_H(S_0-S) \\ 0 & 0 \\ 0 & 0 \\ 0 & 0 \end{Bmatrix}$$

It can be seen that, the total population of model (1) is bounded by S_0. It follows that $S,E_H,I_H,T_H,A_H \leq S_0$, and $\beta I_H S \leq \beta I_H S_0$, $\beta A_H S \leq \beta A_H S_0$ which implies $\hat{\mathrm{W}}(p_1,p_2)$ is positive definite. Additionally, matrix $J_d f$ is undoubtedly an M-matrix, with the off-diagonal entries positive. Hence, the requirement of the two conditions are met, which is a proof of the globally asymptotically stability of f_0. □

3.7. Local stability of the endemic equilibrium. Considering the state model 1, the subsection examines stability at endemic equilibrium by linearising the model equation 1 and evaluating the resulting matrix at the endemic equilibrium [19, 20].

Hence

$$\begin{pmatrix} -\beta(I_H+A_H)-\mu & 0 & -\beta S & 0 & -\beta S \\ 0 & -(\mu+\lambda) & \beta S & 0 & \beta S \\ 0 & \lambda & -(1-\gamma)-(\mu+\delta+\gamma) & 0 & 0 \\ 0 & 0 & \gamma & -\mu & \sigma \\ 0 & 0 & (1-\gamma) & 0 & -(\mu+\delta+\sigma) \end{pmatrix} \tag{24}$$

When 24 is evaluated at the endemic equilibrium $h_1 = (S^*, E_H^*, I_H^*, T_H^*, A_H^*)$, we get

$$J_y = \begin{pmatrix} -\beta(I_H^* + A_H^*) - \mu & 0 & -\beta S^* & 0 & -\beta S^* \\ 0 & -(\mu+\lambda) & \beta S^* & 0 & \beta S^* \\ 0 & \lambda & -(1-\gamma) - (\mu+\delta+\gamma) & 0 & 0 \\ 0 & 0 & \gamma & -\mu & \sigma \\ 0 & 0 & (1-\gamma) & 0 & -(\mu+\delta+\sigma) \end{pmatrix} \tag{25}$$

Now, $|J_y - n|$ gives

$$J_y = \begin{pmatrix} l_{11} & 0 & -\beta S^* & 0 & -\beta S^* \\ 0 & -(\mu+\lambda) - n & \beta S^* & 0 & \beta S^* \\ 0 & \lambda & l_{11} & 0 & 0 \\ 0 & 0 & \gamma & -\mu - n & \sigma \\ 0 & 0 & (1-\gamma) & 0 & -(\mu+\delta+\sigma) - n \end{pmatrix} \tag{26}$$

where

$l_{11} = -\big(\beta(I_H^* + A_H^*) + \mu\big) - n$

$l_{12} = -\big((1-\gamma) + (\mu+\delta+\gamma)\big) - n$

Hence, $n = -\mu$

The remaining matrix of J_y becomes

$$J_p = \begin{pmatrix} l_{11} & 0 & -\beta S^* & -\beta S^* \\ 0 & -(\mu+\lambda) - n & \beta S^* & \beta S^* \\ 0 & \lambda & l_{12} & 0 \\ 0 & 0 & (1-\gamma) & -(\mu+\delta+\sigma) - n \end{pmatrix} \tag{27}$$

By observation, the matrix J_p is not strictly diagonally dominant, since $|-(\mu+\lambda) - n| \not> \beta S^*$. Hence, the HIV model system 1 is not lacally stable.

3.8. Global Stability of the endemic equilibrium. We examine the global stability of the endemic equilibrium of the HIV model (1) by constructing a suitable Lyapunov function for model system model (1). The analysis is given as follows

Theorem 3.4. *Given that $S = S*$, $E_H = E_H^*$, $I_H = I_H^*$, $T_H = T_H^*$, and $A_H = A_H^*$, then, the endemic equilibrium E_n^* of the HIV model (1)is globally asymptotically stable in $R+^5$ whenever $R_0 > 1$*

Proof. We define a lyapunov function $L : \{(S, E_H, I_H, T_H, A_H,) \in \Phi | S, E_H, I_H, T_H, A_H > 0\} \to R$ given by

$$L(S, E_H, I_H, T_H, A_H) = \big(S - S^* - S^* ln\frac{S}{S^*}\big) + \big(E_H - E_H^* - E_H^* ln\frac{E_H}{E_H^*}\big) + \big(I_H - I_H^* - I_H^* ln\frac{I_H}{I_H^*}\big)$$
$$+ \big(T_H - T_H^* - T_H^* ln\frac{T_H}{T_H^*}\big) + \big(A_H - A_H^* - A_H^* ln\frac{A_H}{A_H^*}\big)$$

The time derivative of L becomes

$$\frac{dL}{dt} = \big(\frac{S-S^*}{S}\big)\frac{dS}{dt} + \big(\frac{E_H - E_H^*}{E_C}\big)\frac{dE_H}{dt} + \big(\frac{I_H - I_H^*}{I_H}\big)\frac{dI_H}{dt} + \big(\frac{T_H - T_H^*}{T_H}\big)\frac{dT_H}{dt} + \big(\frac{A_H - A_H^*}{A_H}\big)\frac{dA_H}{dt}$$

$$\frac{dL}{dt} = \big(\frac{S-S^*}{S}\big)\big(\Lambda - \beta S(I_H + A_H) - \mu S\big) + \big(\frac{E_H - E_H^*}{E_C}\big)\big(\beta S(I_H + A_H) - (\mu + \lambda)E_H\big)$$
$$+ \big(\frac{I_H - I_H^*}{I_H}\big)\big(\lambda E_H - (1-\gamma)I_H - (\mu + \delta + \gamma)I_H\big) + \big(\frac{T_H - T_H^*}{T_H}\big)\big(\gamma I_H - \mu T_H + \sigma A_H\big)$$
$$+ \big(\frac{A_H - A_H^*}{A_H}\big)\big((1-\gamma)I_H - (\mu + \delta + \sigma)A_H\big)$$

$$\frac{dL}{dt} = \big(\frac{S-S^*}{S}\big)\big(\Lambda - \beta(S-S^*)((I_H - I_H^*) + (A_H - A_H^*)) - \mu(S-S^*)\big)$$
$$+ \big(\frac{E_H - E_H^*}{E_C}\big)\big(\beta(S-S^*)((I_H - I_H^*) + (A_H - A_H^*)) - (\mu + \lambda)(E_H - E_H^*)\big)$$
$$+ \big(\frac{I_H - I_H^*}{I_H}\big)\big(\lambda(E_H - E_H^*) - (1-\gamma)(I_H - I_H^*) - (\mu + \delta + \gamma)(I_H - I_H^*)\big)$$
$$+ \big(\frac{T_H - T_H^*}{T_H}\big)\big(\gamma(I_H - I_H^*) - \mu(T_H - T_H^*) + \sigma(A_H - A_H^*)\big)$$
$$+ \big(\frac{A_H - A_H^*}{A_H}\big)\big((1-\gamma)(I_H - I_H^*) - (\mu + \delta + \sigma)(A_H - A_H^*)\big)$$

$$\frac{dL}{dt} = \big(\Lambda\big(\frac{S-S^*}{S}\big) - \beta\big(\frac{(S-S^*)^2}{S}\big)((I_H - I_H^*) + (A_H - A_H^*)) - \mu\big(\frac{(S-S^*)^2}{S}\big)\big)$$
$$+ \big(\beta(S-S^*)\big(\frac{E_H - E_H^*}{E_C}\big)((I_H - I_H^*) + (A_H - A_H^*)) - (\mu + \lambda)\big(\frac{(E_H - E_H^*)^2}{E_C}\big)\big)$$
$$+ \big(\lambda(E_H - E_H^*)\big(\frac{I_H - I_H^*}{I_H}\big) - (1-\gamma)\big(\frac{(I_H - I_H^*)^2}{I_H}\big) - (\mu + \delta + \gamma)\big(\frac{(I_H - I_H^*)^2}{I_H}\big)\big)$$
$$+ \big(\gamma(I_H - I_H^*)\big(\frac{T_H - T_H^*}{T_H}\big) - \mu\big(\frac{(T_H - T_H^*)^2}{T_H}\big) + \sigma(A_H - A_H^*)\big(\frac{T_H - T_H^*}{T_H}\big)\big)$$
$$+ \big((1-\gamma)(I_H - I_H^*)\big(\frac{A_H - A_H^*}{A_H}\big) - (\mu + \delta + \sigma)\big(\frac{(A_H - A_H^*)^2}{A_H}\big)\big)$$

$$\frac{dL}{dt} = \big(\Lambda - \Lambda\big(\frac{S^*}{S}\big) - \beta\big(\frac{(S-S^*)^2}{S}\big)\big((I_H - I_H^*) + (A_H - A_H^*)\big) - \mu\big(\frac{(S-S^*)^2}{S}\big)\big)$$
$$+ \big(\beta(S-S^*)\big(\frac{E_H - E_H^*}{E_C}\big)\big((I_H - I_H^*) + (A_H - A_H^*)\big) - (\mu+\lambda)\big(\frac{(E_H - E_H^*)^2}{E_C}\big)\big)$$
$$+ \big(\lambda(E_H - E_H^*)\big(\frac{I_H - I_H^*}{I_H}\big) - (1-\gamma)\big(\frac{(I_H - I_H^*)^2}{I_H}\big) - (\mu+\delta+\gamma)\big(\frac{(I_H - I_H^*)^2}{I_H}\big)\big)$$
$$+ \big(\gamma(I_H - I_H^*)\big(\frac{T_H - T_H^*}{T_H}\big) - \mu\big(\frac{(T_H - T_H^*)^2}{T_H}\big) + \sigma(A_H - A_H^*)\big(\frac{T_H - T_H^*}{T_H}\big)\big)$$
$$+ \big((1-\gamma)(I_H - I_H^*)\big(\frac{A_H - A_H^*}{A_H}\big) - (\mu+\delta+\sigma)\big(\frac{(A_H - A_H^*)^2}{A_H}\big)\big)$$

Applying the expression $z = z_1 - z_2$ gives

$$z_1 = \Lambda + \big(\beta(S-S^*)\big(\frac{E_H - E_H^*}{E_C}\big)\big((I_H - I_H^*) + (A_H - A_H^*)\big) + \big(\lambda(E_H - E_H^*)\big(\frac{I_H - I_H^*}{I_H}\big)$$
$$+ \big(\gamma(I_H - I_H^*)\big(\frac{T_H - T_H^*}{T_H}\big) + \sigma(A_H - A_H^*)\big(\frac{T_H - T_H^*}{T_H}\big)\big)$$

$$z_2 = \Lambda\big(\frac{S^*}{S}\big) + \beta\big(\frac{(S-S^*)^2}{S}\big)\big((I_H - I_H^*) + (A_H - A_H^*)\big) + \mu\big(\frac{(S-S^*)^2}{S}\big)$$
$$+ (\mu+\lambda)\big(\frac{(E_H - E_H^*)^2}{E_C}\big) + (1-\gamma)\big(\frac{(I_H - I_H^*)^2}{I_H}\big) + (\mu+\delta+\gamma)\big(\frac{(I_H - I_H^*)^2}{I_H}\big)$$
$$+ \mu\big(\frac{(T_H - T_H^*)^2}{T_H}\big) + (\mu+\delta+\sigma)\big(\frac{(A_H - A_H^*)^2}{A_H}\big)$$

We notice that the inequality $z_1 < z_2$ holds, which means that $\frac{dL}{dt} \leq 0$ if $z_1 < z_2$. Hence, it follows that $\frac{dL}{dt} = 0$ when $S = S*$, $E_H = E_H^*$, $I_H = I_H^*$, $T_H = T_H^*$, and $A_H = A_H^*$. Therefore the largest compact invariant set $\{S, E_H, I_H, T_H, A_H \in \Phi : \frac{dL}{dt} = 0\}$ is the singleton E_n, where E_n is the endemic equilibrium. Hence from [21, 22, 23, 24], E_n is globally asymptotically stable in Φ. □

4. Optimal Control Model

The subsection 4 formulates an optimal control model for the HIV disease after analysing the non control model to identify control strategies that would help to minimize the disease. In view of achieving this purpose, we add control of condom use u_1 and education of the susceptible

individuals u_2 to the non-control model.

$$
\begin{cases}
\frac{d}{dt}S = \Lambda - (1-u_1)\beta S(I_H + A_H) - \mu S - u_2 S \\
\frac{d}{dt}E_H = (1-u_1)\beta S(I_H + A_H) + u_2 S - (\mu + \lambda)E_H \\
\frac{d}{dt}I_H = \lambda E_H - (1-\gamma)I_H - (\mu + \delta + \gamma)I_H \\
\frac{d}{dt}T_H = \gamma I_H - \mu T_H + \sigma A_H - u_2 I_H \\
\frac{d}{dt}A_H = (1-\gamma)I_H - (\mu + \delta + \sigma)A_H
\end{cases} \tag{28}
$$

We consider a quadratic function for the objective functional as in other literature [25]. Here, we seek to minimize the exposed and infected. The control of personal protection: condom use u_1 would be employed to achieve the above mentioned purpose of minimizing the exposed and infected population. Hence, the objective functional $\mathscr{J}$ is given by

$$
J = \int_0^{t_f} \left[G_1 E_H + G_2 I_H + \frac{1}{2} C_1 u_1^2 + \frac{1}{2} C_2 u_2^2 \right] .dt \tag{29}
$$

The quantities of objective functional (29) G_1 and G_2 are the weight coefficients of the exposed, infected, treatment and asymptomatic population. In addition, the expressions $\frac{C_1 u_1^2}{2}$ and $\frac{C_2 u_2^2}{2}$ are the cost that comes with minimizing the the exposed and infected population. Hence, we seek an optimal control u_1^* such that

$$
J(u_1^*) = min\{J(u_1, u_2) : (u_1, u_2) \in U\} \tag{30}
$$

where

$$
U = \{(u_1, u_2) |\ 0 \leq u_i \leq 1, i = 1 \text{ lebesgue measurable}\} \tag{31}
$$

Now, the analytic method of Pontryagin's maximum principle [**?**], would be employed to converts system 28 and 29 into a problem of minimizing the Hamiltonian H with respect to the controls u_1, u_2, where

$$
\begin{aligned}
H_f = & \left[G_1 E_H + G_2 I_H + \frac{1}{2} C_1 u_1^2 + \frac{1}{2} C_2 u_2^2 \right] \\
& + \lambda_1 \{\Lambda - (1-u_1)\beta S(I_H + A_H) - \mu S - u_2 S\} \\
& + \lambda_2 \{(1-u_1)\beta S(I_H + A_H) + u_2 S - (\mu + \lambda)E_H\}
\end{aligned}
$$

$$+\lambda_3\{\lambda E_H-(1-\gamma)I_H-(\mu+\delta+\gamma)I_H\}$$
$$+\lambda_4\{\gamma I_H-\mu T_H+\sigma A_H\}$$
$$+\lambda_5\{(1-\gamma)I_H-(\mu+\delta+\sigma)A_H\}. \tag{32}$$

Theorem 4.1. *There exists an optimal control $U^*=(u_1^*,u_2^*)\in U$ such that*

$$\mathscr{J}(u_1^*,u_2^*)=\min_U \mathscr{J}(u_1,u_2), \tag{33}$$

subject to the control system (28) with the initial conditions.

Proof. By the work of [26], the existence of optimal control is proved. We observe that the state and control variables are non-negative. We also observe that in minimizing the control problem, the necessary and convexity of the objective functional in u_1 are satisfied. The control space
$U=\{u|u_1,u_2$ are measurable, $0\leq u_1,u_2\leq u_max<\infty,\ t\in[0,t_f]\}$
is also convex and closed by definition. The optimal system is bounded which verifies the compactness needed for the existence of the optimal control. Also, the integrand in functional 29, $\left[G_1E_H+G_2I_H+\frac{1}{2}C_1u_1^2+\frac{1}{2}C_2u_2^2\right]$ is convex on the control u. Therefore, we see that there exist a constant $k>1$, positive numbers u_1,u_2 such that,
$J(u_1,u_2)\geq u_1\left(|u_1,u_2|^2\right)\frac{k}{2}-u^2$.
Hence, there exist an optimal control. In the quest to find the optimal solution, the Pontryagin's maximum principle [27, 21] is applied to the Hamiltonain 32 such that if (w,u) is an optimal solution of the optimal control problem, then there exist a non-trivial vector function $\lambda=(\lambda_1\ \ldots\ \lambda_6)$ satisfying the below equation

$$\begin{aligned}\frac{dz}{dt}&=\frac{\partial H(t,w,u,\lambda)}{\partial\lambda}\\ 0&=\frac{\partial H(t,w,u,\lambda)}{\partial u}\\ \frac{d\lambda}{dt}&=-\frac{\partial H(t,w,u,\lambda)}{\partial z}\end{aligned} \tag{34}$$

Hence, the necessary condition associated to the Hamitonian (32) is applied.

Theorem 4.2. *Given that S, E_H, I_H, T_H and A_H are optimal state solutions with associated control variables (u_1^*, u_2^*) for the optimal control problem 28 and* **??***, then there exist adjoint variables λ_i for $i = 1, \ldots, 6$, satisfying*

$$\begin{aligned}
\lambda_1' &= -\frac{\partial H}{\partial S} = (\lambda_1 - \lambda_2)(1-u_1)\beta(I_H + A_H) + (\lambda_1 - \lambda_2)u_2 + \mu\lambda_1 \\
\lambda_2' &= -\frac{\partial H}{\partial E_H} = -G_1 + (\lambda_2 - \lambda_3) + \mu\lambda \\
\lambda_3' &= -\frac{\partial H}{\partial I_H} = -G_2 + (\lambda_1 - \lambda_2)(1-u_1)\beta S + (\lambda_3 - \lambda_4)\gamma + (\lambda_3 - \lambda_5)(1-\gamma) + (\delta + \mu)\lambda_3 \\
\lambda_4' &= -\frac{\partial H}{\partial T_H} = \mu\lambda_4 \\
\lambda_5' &= -\frac{\partial H}{\partial A_H} = (\lambda_1 - \lambda_2)(1-u_1)\beta S + (\lambda_5 - \lambda_4)\sigma + (\mu + \delta)\lambda_5 \qquad (35)
\end{aligned}$$

with boundary condition

$$\lambda_i(t_f) = 0, \ i = 1, 2, \ldots, 5 \qquad (36)$$

The optimal control u_1^* are given by

$$u_1' = \min\left\{1, \max\left\{0, \left((\lambda_2 - \lambda_1)\frac{\beta S(I_H + A_H)}{C_1}\right)\right\}\right\}$$

$$u_2' = min\left\{1, \max\left\{0, \left(\frac{(\lambda_1 - \lambda_2)}{C_2}\right)\right\}\right\} \qquad \square$$

Proof. The adjoint and boundary conditions are derived by applying the Hamiltonian 32. Thus we equate $S = S^*$, $E_H = E_H^*$, $I_H = I_H^*$, $T_H = T_H^*$ and $A_H = A_H^*$ and differentiating the Hamiltonain with respect to S, E_H, I_H, T_H and A_H to obtain (35). Further, the equations $\dfrac{\partial H}{\partial u_1} = 0$ are determined on the interior of the control set and using the optimal conditions and the property of the control space u_1 and u_2, and we derive 28. From (28), The control is characterize by solving the optimal system. Thus, the transversality and the charcterisation of the optimal control (u_1) are use in solving the optimal system [28, 29, 30]. $\square$

The controls u_1^* and u_2 when substituted into the control system (28) gives

$$
\begin{cases}
\frac{d}{dt}S & = \Lambda - \left(1 - min\left\{1, max\left\{0, \left((\lambda_2 - \lambda_1)\frac{\beta S(I_H + A_H)}{C_1}\right)\right\}\right\}\right)\beta S(I_H + A_H) \\
 & \quad -(min\left\{1, \max\left\{0, \left(\frac{(\lambda_1 - \lambda_2)}{C_2}\right)\right\}\right\})s - \mu S \\
\frac{d}{dt}E_H & = \left(1 - min\left\{1, max\left\{0, \left((\lambda_2 - \lambda_1)\frac{\beta S(I_H + A_H)}{C_1}\right)\right\}\right\}\right)\beta S(I_H + A_H) - (\mu + \lambda)E_H \\
\frac{d}{dt}I_H & = \lambda E_H - (1 - \gamma)I_H - (\mu + \delta + \gamma)I_H \\
\frac{d}{dt}T_H & = \gamma I_H - \mu T_H + \sigma A_H \\
\frac{d}{dt}A_H & = (1 - \gamma)I_H - (\mu + \delta + \sigma)A_H
\end{cases} \tag{37}
$$

5. NUMERICAL SIMULATIONS

In determining the best control strategy that would help combat the spread of infection, an iterative scheme that uses a fourth-order Runge-Kutta method to run the optimal system is designed. This approach runs state equation forward and the adjoint system backwards in time. Iteration runs until a stopping criterion is met, and it stops. Effectiveness of the considered controls on the model are assessed, these controls are paired, and a numerical simulation carried out. Output plots generated for each considered strategy are carefully assessed for consideration. Table 6 shows some of the parameter values used in the numerical simulations that generated these outputs. Following are the observations from various plots as indicated in Figures 2, 3, 4, 5, 6, 7, 8, and 9.

TABLE 6. **HIV Model and Parameters**

Parameter	Baseline	Source
Λ	50	[31]
β	1.2450	Estimated
λ	$0.025 - 0.075$	[31]
α	0.8205	[31]
μ	0.00025	[31]
δ	0.204	Estimated
γ	0.0345	Estimated
σ	0.225	Estimated

5.1. Strategy 1: Optimal control with use of condoms by both susceptible and infected populations only. We simulated the optimality system by incorporating the use of condoms as the only intervention. It can be observed that there have been an exponential decrease in the number of susceptible and infected populations as shown in Figure 2. This is an indication of the effectiveness this intervention on control of HIV spread. However, there have a been small change in population exposed to the HIV infection in the system as shown in Figure 3. Moreover, there have been a small reduction in population under treatment (anti-antiretroviral therapy) as indicated in Figure 4 and 5.

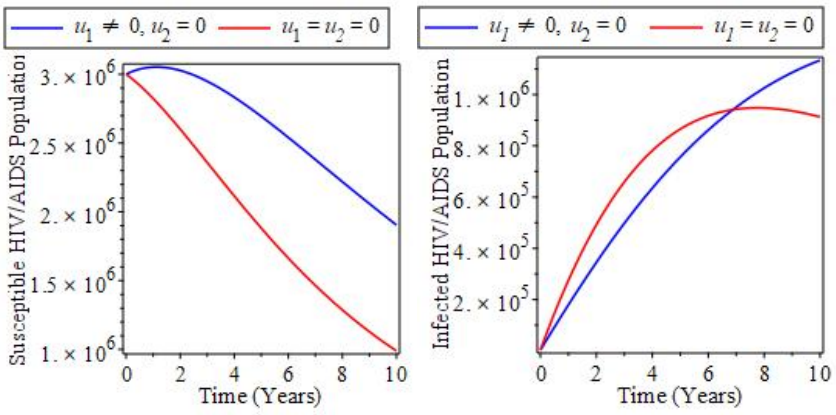

FIGURE 2. plot of phase portraits with u_1 and u_2

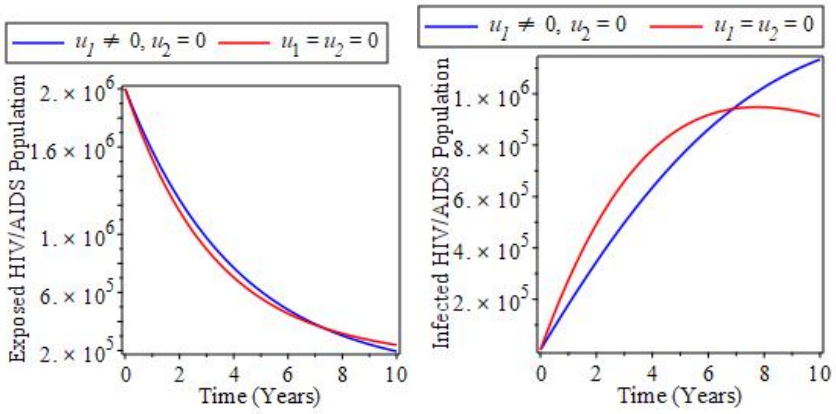

FIGURE 3. plot of phase portraits with u_1 and u_2

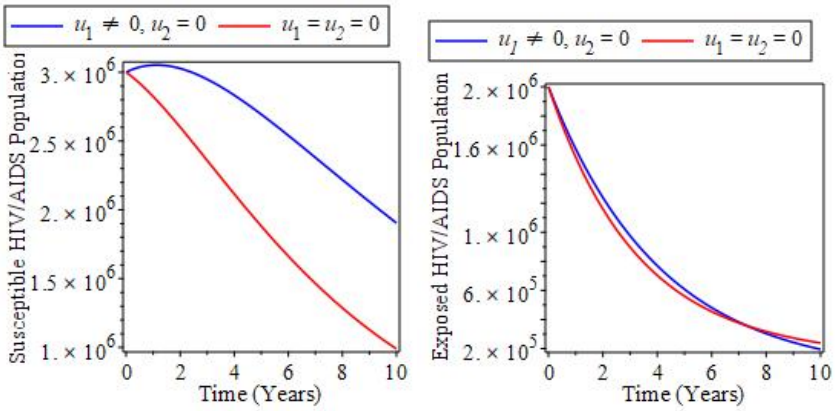

FIGURE 4. plot of phase portraits with u_1 and u_2

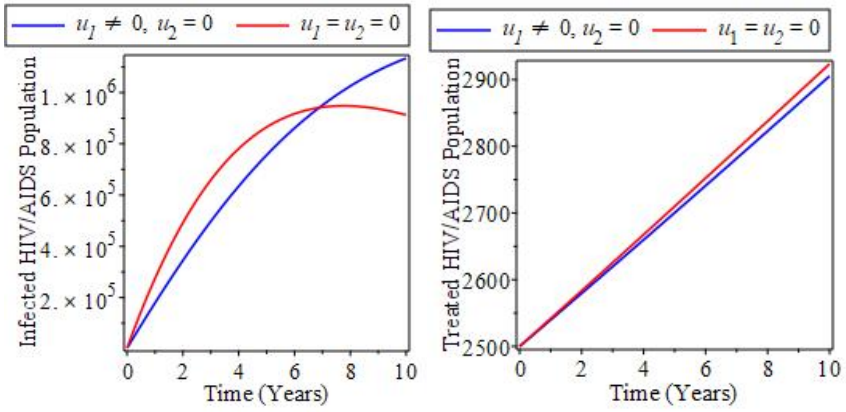

FIGURE 5. plot of phase portraits with u_1 and u_2

5.2. Strategy 2: Optimal control with education of susceptible on complete abstinence.

The optimality system was simulated by incorporating education as the only intervention. It was observed that there have substantial change in population of susceptible individuals. Moreover, there have been a reduction in the of individuals getting infected with infection. An indication of the possibility of this intervention. Figure 6, 7, 8 and 9 show the dynamics of intervention strategy.

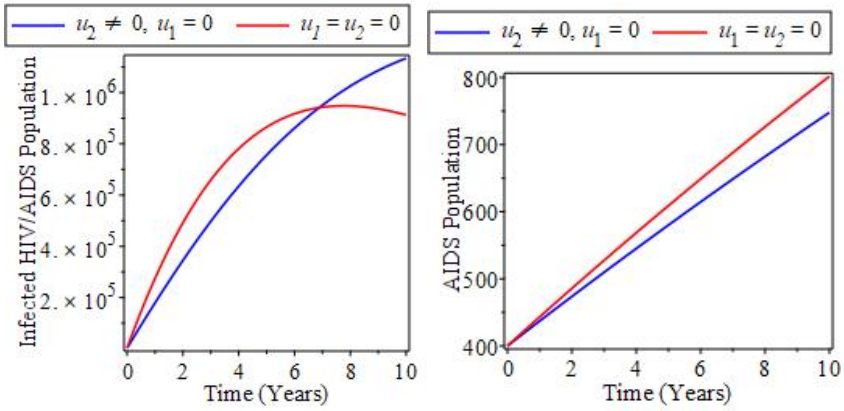

FIGURE 6. plot of phase portraits with u_1 and u_2

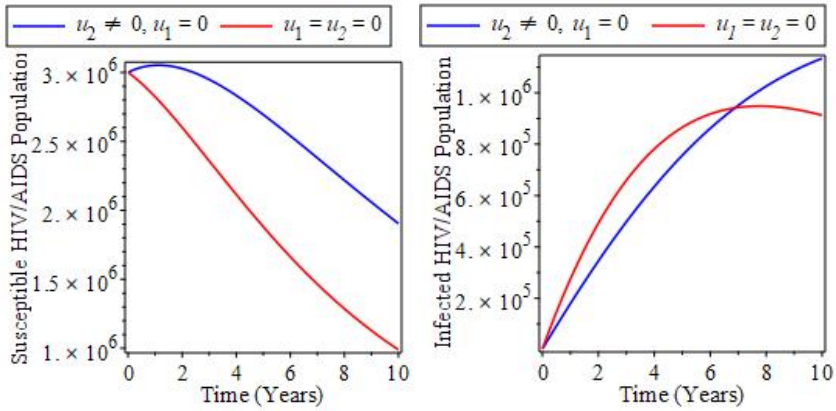

FIGURE 7. plot of phase portraits with u_1 and u_2

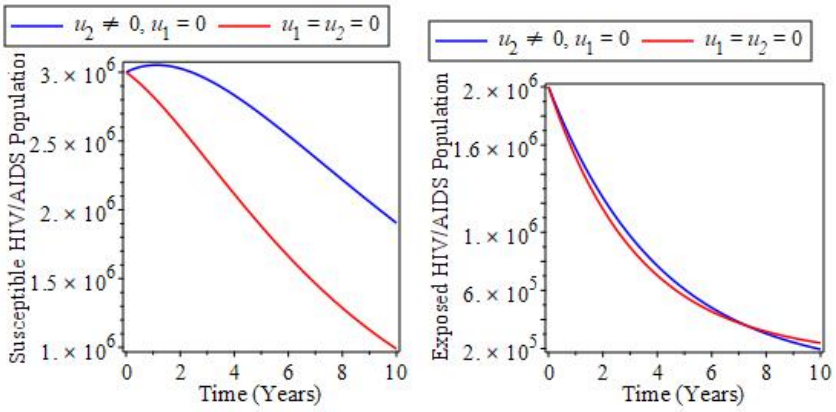

FIGURE 8. plot of phase portraits with u_1 and u_2

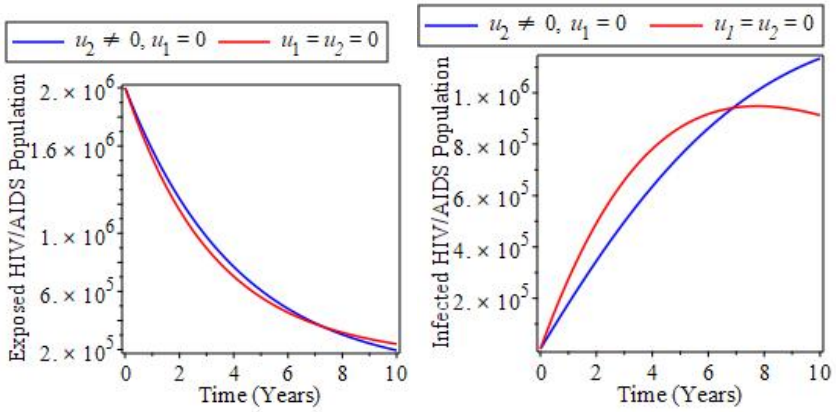

FIGURE 9. plot of phase portraits with u_1 and u_2

6. CONCLUSION

In this study, a deterministic model for HIV-AIDS is formulated. The equilibrium points, local and global stability of the equilibrium points, and HIV reproductive rate were determined and interpreted. The model was extended to optimal control and it was established that the best and most effective control strategy was optimal education and sensitisation of susceptible population.

We simulated the optimality system by incorporating the use of condoms as the only intervention. It can be observed that there have been an exponential decrease in the number of susceptible and infected populations. Then the optimality system was simulated by incorporating education as the only intervention. It was observed that there have substantial change in

population of susceptible individuals. Moreover, there have been a reduction in the of individuals getting infected with infection. An indication of the possibility of this intervention.
In combating the infection, more resources should placed on sensitisation and education of the susceptible population.

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CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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