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Stability Analysis of the Transmission Dynamics of HIV/AIDS in the Presence of Treatment and Condom Usage

S. Abdulrahman[*], M. Bawa [**], Y. B. Aliyu[**], A. I. Ajike[***]
[*]Department of Mathematics and Statistics, Federal University of Technology, Minna
[**]Department of Mathematics, Ibrahim Badamasi Babangida University, Lapai
[***]Department of Mathematics, Abia State Polytechnic, Aba

ABSTRACT

We developed and analysed a new a mathematical model for the dynamics of Human Immunodeficiency Virus infection/Acquired Immunodeficiency Syndrome (HIV/AIDS) in a population with vital dynamics, incorporating vertical transmission, sexual maturity and the effect of public enlightenment campaign with respect to sexual behaviour change and treatment as control measures. We obtained the effective basic reproduction number, R_c which can be used to control the transmission of the disease and hence, established the conditions for local and global stability of the disease free equilibrium. Bifurcation analysis was carried out using centre manifold theory which reveals a subcritical (backward) bifurcation for the model. Numerical simulations validated the analytical results and further reveal that treatment with Antiretroviral Drugs Therapy (ART) by even 25% of the sexually active HIV^+ individuals will lead to a reduction of the disease morbidity, while ART treatment by 75% of HIV^+ children and/or AIDS infected individuals as encourage by World Health Organisation (WHO) does not have any positive impact on disease morbidity. Furthermore, condom usage (as the only control measure) must be as high as 75% before any positive result is achieved.

Keywords: *HIV/AIDS, Effective basic reproduction number, Stability, Bifurcation analysis*

1. INTRODUCTION

Human Immunodificiency Virus infection/Acquired Immunodeficiency Syndrome (HIV/AIDS) is a disease of the human immune system caused by the human immunodeficiency virus (Sepkowitz, 2001). When the virus enters the human body, it infects and damages the white blood cells known as lymphocytes or $CD4^+$ T cells. These cells are essential for the coordination of the body's immune system, which protect the body against infections. Due to the role of the cells in immune regulations, their depletion has widespread deleterious effects on the functioning of the immune system, making the body much more likely to get infections, including opportunistic infections caused by bacteria, viruses, fungi and parasites that are normally controlled by the immune system (Holmes et. al., 2003).

Since its discovery in the 1980s, HIV remains a major global menace. In addition to accounting for nearly 30 million deaths as of 2009 (UNAIDS, 2010), an estimated 34 million people live with the disease globally as of 2010 (UNAIDS, 2011). Population infectivity estimates range as high as 8.5% for sub-Saharan Africa and as low as 0.1% for East Asia (UNAIDS, 2011).

Transmission of HIV results from exposure to infectious blood or body fluids containing blood. Possible forms of transmissions include (but are not limited to) unprotected sexual contact, blood transfusions, re-use of contaminated needles and syringes, and vertical transmission from mother to child during pregnancy, delivery or breastfeeding. There is no risk of acquiring HIV if exposed to faeces, nasal secretions, saliva, sputum, sweat, tears, urine, or vomit unless these are contaminated with blood (Kripke, 2007).

The study of HIV/AIDS transmission dynamics has been of great interest to both applied Mathematicians and Biologist due to its universal threat to humanity (Naresh and Sharma, 2011). Several authors (Linda et. al., 1991; Bortolotti et.al., 1992; Blower and McLean, 1994; Velasco-Hernandez et. al., 1996; Fylkesnes et. al., 2001; Gumel et. al., 2006; Rapatski et. al., 2006; Lungu et. al., 2006; Nayeem et. al., 2009; Mukandavire et. al., 2010; Garba and Gumel, 2010; Naresh and Sharman, 2011) have over the last three decades used mathematical models to evaluate the effect of public health programs and provided long-term predictions regarding HIV/AIDS prevalence and control in various regions. Considering the work of all the authors mentioned above, we developed and analysed a new mathematical model to complements and extend their works by incorporating the following factors which are very important in the transmission and control of HIV/AIDS especially in sub-Saharan African countries where the disease is endemic.

i) Vital dynamics (number of birth not equal to number of death);
ii) Vertical transmission;
iii) Public enlightenment;
iv) Condom usage;
v) Standard incidence function;
vi) Disease induced death due AIDS;
vii) Treatment and
viii) Sexual maturity.

This paper is organised as follows: In section 2 we formulate the model. In section 3 we analysed the model by obtaining the effective basic reproduction number, R_c and establishing the conditions for local and global stability of the disease free equilibrium. Bifurcation analysis was carried out using centre manifold theory. We discuss the results in section 4.

2. MODEL FORMULATION

We formulate a model for the spread and control of HIV/AIDS in the human population with the total population size at time, t given by $N(t)$ with the following assumptions:

a) There is homogeneous mixing of the population, where all people are equally likely to be infected by the infectious individuals in case of contact;

b) The probability of AIDS Individuals to be sexually active or have susceptible sexual partner is not worth mentioning and thus, assumed does not contribute to the transmission of the disease;

c) Condom usage and treatment with ART are with full compliance.

The total population is compartmentalized into 8 epidemiological classes shown in figure 3.1.

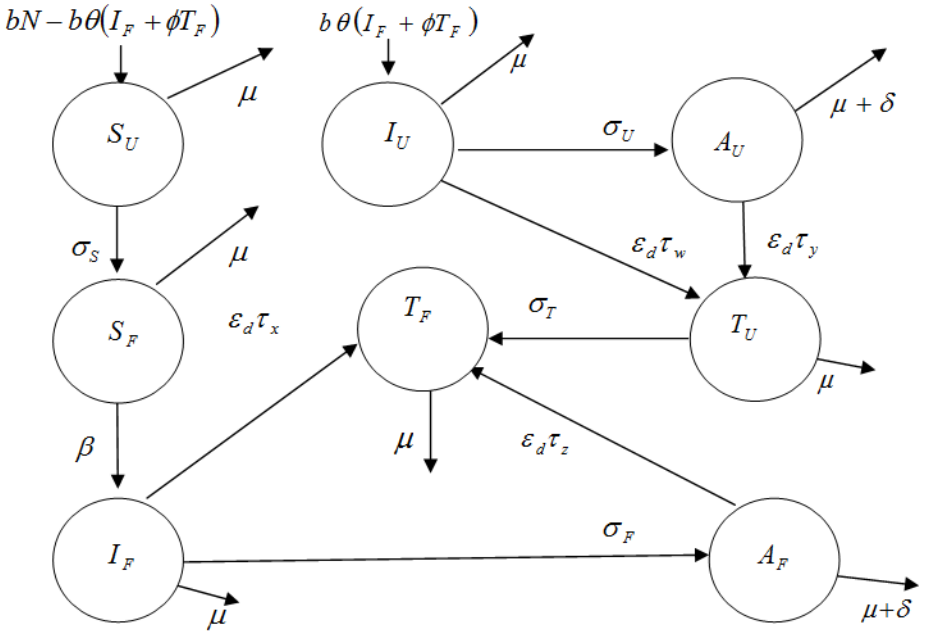

Figure 3.1 Schematic diagram of HIV transmission and control model

were the variables and parameters of the model are defined as follows:

$S_U(t)$ Population of susceptible individuals less than 15 years of age at time t
$S_F(t)$ Population of susceptible individuals at or above 15 years of age at time t
$I_U(t)$ Population of HIV$^+$ infected individuals less than 15 years of age at time t
$I_F(t)$ Population of HIV$^+$ infected individuals at or above 15 years of age at time t
$T_U(t)$ Population of infected individuals receiving ART treatment that is less than 15 years of age at time t
$T_F(t)$ Population of infected individuals receiving ART treatment that is at or above 15 years of age at time t
$A_U(t)$ Population of AIDS infected individuals less than 15 years of age at time t
$A_F(t)$ Population of AIDS infected individuals at or above 15 years of age at time t

b Per capital birth rate of humans
μ Per capital natural death rate of humans
δ AIDS-induced death rate
c Average total sexual contacts
p HIV-Sexual transmission risk rate and therefore $\beta = pc$ is the effective sexual contact rate.
η Modification parameter associated with reduced sexual transmission rate by individuals receiving ARV treatment
ε_c Condom efficacy
τ_c Condom usage rate and therefore $\varepsilon_c \tau_c$ is the effective condom usage rate
ε_d Anti-retroviral drug's efficacy
τ_w Treatment rate for individuals in I_U and thus $\varepsilon_d \tau_w$ is the effective treatment rate for individuals in I_U
τ_x Treatment rate for individuals in I_F and thus $\varepsilon_d \tau_x$ is the effective treatment rate for individuals in I_F
τ_y Treatment rate for individuals in A_U and thus $\varepsilon_d \tau_y$ is the effective treatment rate for individuals in A_U
τ_z Treatment rate for individuals in A_F and thus $\varepsilon_d \tau_z$ is the effective treatment rate for individuals in A_F
θ Proportion of HIV$^+$ birth
ϕ Modification parameter associated with reduced proportion of HIV$^+$ birth by individuals receiving treatment
σ_S Average rate of moving from S_U to S_F
σ_I Average rate of moving from I_U to I_F
σ_U Average rate of disease progression from I_U to A_U
σ_F Average rate of disease progression from I_F to A_F

The corresponding mathematical equations of the schematic diagram can be described by a system of ordinary differential equations (3.1a – 3.1h)

$$\frac{dS_U}{dt} = bN - b\theta\left(I_F + \phi T_F\right) - \left(\sigma_S + \mu\right)S_U \qquad (3.1a)$$

$$\frac{dS_F}{dt} = \sigma_S S_U - \frac{pc\left(I_F + \eta T_F\right)\left(1-\varepsilon_c\tau_c\right)}{N}S_F - \mu S_F \qquad (3.1b)$$

$$\frac{dI_U}{dt} = b\theta\left(I_F + \phi T_F\right) - \left(\varepsilon_d\tau_w + \sigma_U + \mu\right)I_U \qquad (3.1c)$$

$$\frac{dI_F}{dt} = \frac{pc\left(I_F + \eta T_F\right)\left(1-\varepsilon_c\tau_c\right)}{N}S_F - \left(\varepsilon_d\tau_x + \sigma_F + \mu\right)I_F \qquad (3.1d)$$

$$\frac{dT_U}{dt} = \left(\varepsilon_d\tau_w\right)I_U + \left(\varepsilon_d\tau_y\right)A_U - \left(\sigma_T + \mu\right)T_U \qquad (3.1e)$$

$$\frac{dT_F}{dt} = \sigma_T T_U + \left(\varepsilon_d\tau_x\right)I_F + \left(\varepsilon_d\tau_z\right)A_F - \mu T_F \qquad (3.1f)$$

$$\frac{dA_U}{dt} = \sigma_U I_U - \left(\varepsilon_d\tau_y + \mu + \delta\right)A_U \qquad (3.1g)$$

$$\frac{dA_F}{dt} = \sigma_F I_F - \left(\varepsilon_d\tau_z + \mu + \delta\right)A_F \qquad (3.1h)$$

where,

$$N(t) = S_U(t) + S_F(t) + I_U(t) + I_F(t) + T_U(t) + T_F(t) + A_U(t) + A_F(t) \qquad (3.2)$$

So that

$$\frac{dN}{dt} = \left(b - \mu\right)N - \delta\left(A_U + A_F\right) \qquad (3.3)$$

Consider equations (3.1a – 3.1h) for the normalised quantities. Since, it is better and easier (convenient) to analyze our model in terms of proportions of quantities instead of actual populations as described in Busenberg (1990), Akinwande (1996), Li et al. (1999), Hethcote (2000), Tumwiine et al. (2007), Capasso (2008) and Benyah (2008). This can be done by scaling the population of each class by the total populations N. We

let denote the fractions of the classes $S_U, S_F, I_U, I_F, T_U, T_F, A_U$ and A_F in the population respectively. This is done by differentiating the fractions

$$s_U = \frac{S_U}{N}, s_F = \frac{S_F}{N}, i_U = \frac{I_U}{N}, i_F = \frac{I_F}{N}, t_U = \frac{T_U}{N}. t_F = \frac{T_F}{N}, a_U = \frac{A_U}{N} \text{ and } a_F = \frac{A_F}{N}$$

(using quotient rule) with respect to time, *t*. Then simplifying, we have from (3.1a – 3.1h) and (3.3)

$$\frac{ds_U}{dt} = b - (b_I i_F + b_T t_F) - K_1 s_U + \delta(a_U + a_F) s_U \quad (3.4a)$$

$$\frac{ds_F}{dt} = \sigma_S s_U - (\alpha_I i_F + \alpha_T t_F) s_F - b s_F + \delta(a_U + a_F) s_F \quad (3.4b)$$

$$\frac{di_U}{dt} = (b_I i_F + b_T t_F) - K_2 i_U + \delta(a_U + a_F) i_U \quad (3.4c)$$

$$\frac{di_F}{dt} = (\alpha_I i_F + \alpha_T t_F) s_F - K_3 i_F + \delta(a_U + a_F) i_F \quad (3.4d)$$

$$\frac{dt_U}{dt} = (\varepsilon_d \tau_w) i_U + (\varepsilon_d \tau_y) a_U - K_4 t_U + \delta(a_U + a_F) t_U \quad (3.4e)$$

$$\frac{dt_F}{dt} = \sigma_T t_U + (\varepsilon_d \tau_x) i_F + (\varepsilon_d \tau_z) a_F - K_5 t_F + \delta(a_U + a_F) t_F \quad (3.4f)$$

$$\frac{da_U}{dt} = \sigma_U i_U - K_6 a_U + \delta(a_U + a_F) a_U \quad (3.4g)$$

$$\frac{da_F}{dt} = \sigma_F i_F - K_7 a_F + \delta(a_U + a_F) a_F \quad (3.4h)$$

in the biological - feasible region:

$$\Omega = \left\{ \begin{array}{c} (s_U, s_F, i_U, i_F, t_U, t_F, a_U, a_F,) \in \Re_+^8 : \\ s_U + s_F + i_U + i_F + t_U + t_F \\ + a_U + a_F = 1 \end{array} \right\} \quad (3.5)$$

where

$$\begin{aligned} &b_I = b\theta,\ b_T = b\theta\phi,\ \alpha_I = pc(1-\varepsilon_c\tau_c) \\ &\alpha_T = pc\eta(1-\varepsilon_c\tau_c), K_1 = (\sigma_S + b) \\ &K_2 = (\varepsilon_d\tau_w + \sigma_U + b),\ K_3 = (\varepsilon_d\tau_x + \sigma_F + b) \\ &K_4 = (\sigma_T + b),\ K_5 = b,\ K_6 = (\varepsilon_d\tau_y + b + \delta) \\ &K_7 = (\varepsilon_d\tau_z + b + \delta) \end{aligned} \quad (3.6)$$

this can be shown to be positively invariant with respect to the system (3.4a – 3.4h). We note that the total population size $N(t)$ does not appear in (3.4a – 3.4h); this is as a direct result of the homogeneity of the equations in (3.1a – 3.1h).

3. MODEL ANALYSIS

We now determine the existence of equilibria points; computing the effective basic reproduction number; and establishing the conditions for stability of the equilibria points.

3.1 Existence of Disease Free Equilibrium State, E_f

At the disease free equilibrium state we have absence of infection. Thus, all the infected classes will be zero and the entire population will comprise of susceptible.

At equilibrium state the rate of change of each variable is equal to zero. i.e.

$$\frac{dN}{dt} = \frac{ds_U}{dt} = \frac{ds_F}{dt} = \frac{di_U}{dt} = \frac{di_F}{dt} = \frac{dt_U}{dt} = \frac{dt_F}{dt} = \frac{da_U}{dt} = \frac{da_F}{dt} = 0$$

Let

$$(s_U, s_F, i_U, i_F, t_U, t_F, a_U, a_F) = (s_U^*, s_F^*, i_U^*, i_F^*, t_U^*, t_F^*, a_U^*, a_F^*)$$

at equilibrium state. Thus, substituting into (3.24) with $i_U^* = i_F^* = t_U^* = t_F^* = a_U^* = a_F^* = 0$, we obtained the disease – free equilibrium state given by:

$$\left(s_U^*, s_F^*, i_U^*, i_F^*, t_U^*, t_F^*, a_U^*, a_F^*\right) = \left(\frac{b}{K_1}, \frac{\sigma_S}{K_1}, 0,0,0,0,0,0\right) \tag{3.7}$$

3.2 Effective Basic Reproduction Number, R_c

Consideration of stability of a disease-free equilibrium gives certain conditions under which disease will die out or stay in the population called the Basic reproduction number, R_0. Using the approach of Diekmann and Heesterbeek (2000) we obtained the effective basic reproduction number, R_c of the system (3.1) which is the spectral radius (ρ) of the next generation matrix, K.

i.e.

$$R_C = \rho K, \text{ where } K = FV^{-1}$$

Now,

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha_I s_F^* & 0 & \alpha_T s_F^* & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \text{ and}$$

$$V = \begin{pmatrix} K_2 & -b_I & 0 & -b_T & 0 & 0 \\ 0 & K_3 & 0 & 0 & 0 & 0 \\ -(\varepsilon_d \tau_w) & 0 & K_4 & 0 & -(\varepsilon_d \tau_y) & 0 \\ 0 & -(\varepsilon_d \tau_x) & -\sigma_T & K_5 & 0 & -(\varepsilon_d \tau_z) \\ -\sigma_U & 0 & 0 & 0 & K_6 & 0 \\ 0 & -\sigma_F & 0 & 0 & 0 & K_7 \end{pmatrix}$$

Thus,

$$R_C = \frac{\alpha_I s_F^*}{K_3}\left(1 + \frac{\eta\left\{\left\{(\varepsilon_d \tau_x)K_7 + \sigma_F(\varepsilon_d \tau_z)\right\}K_2 K_4 K_6 + b_I\left((\varepsilon_d \tau_w)K_6 + (\varepsilon_d \tau_y)\sigma_U\right)\sigma_T K_7\right\}}{K_7\left\{K_2 K_4 K_5 K_6 - b_T \sigma_T\left((\varepsilon_d \tau_w)K_6 + (\varepsilon_d \tau_y)\sigma_U\right)\right\}}\right) \tag{3.8}$$

and

$$R_0 = \frac{\sigma_S}{(\sigma_S + b)} \times \frac{pc}{(\sigma_F + b)} \tag{3.9}$$

3.3 Local Stability of Disease Free Equilibrium, E_f

We used the Jacobian stability approach to prove the stability of the disease free equilibrium state. Using the relation

$$s_U = 1 - s_F - i_U - i_F - t_U - t_F - a_U - a_F \tag{3.10}$$

allows us as explained in Hethcote (2000), Benyah (2008) to attack (3.4) by studying the subsystem:

$$\frac{ds_F}{dt} = \sigma_S\left(1 - \left(s_F + i_U + i_F + t_U + t_F + a_U + a_F\right)\right) - (\alpha_I i_F + \alpha_T t_F)s_F - b s_F + \delta(a_U + a_F)s_F \tag{3.11a}$$

$$\frac{di_U}{dt} = (b_I i_F + b_T t_F) - K_2 i_U + \delta(a_U + a_F)i_U \tag{3.11b}$$

$$\frac{di_F}{dt} = (\alpha_I i_F + \alpha_T t_F)s_F - K_3 i_F + \delta(a_U + a_F)i_F \tag{3.11c}$$

$$\frac{dt_U}{dt} = (\varepsilon_d \tau_w)i_U + (\varepsilon_d \tau_y)a_U - K_4 t_U + \delta(a_U + a_F)t_U \tag{3.11d}$$

$$\frac{dt_F}{dt} = \sigma_T t_U + (\varepsilon_d \tau_x)i_F + (\varepsilon_d \tau_z)a_F - K_5 t_F + \delta(a_U + a_F)t_F \tag{3.11e}$$

$$\frac{da_U}{dt} = \sigma_U i_U - K_6 a_U + \delta(a_U + a_F)a_U \tag{3.11f}$$

$$\frac{da_F}{dt} = \sigma_F i_F - K_7 a_F + \delta(a_U + a_F)a_F \tag{3.11g}$$

Linearization of the equations in (3.11a – 3.11g) at E_f gives the Jacobian matrix

$$J(E_f)=\begin{pmatrix} -(\sigma_S+b) & -\sigma_S & -(\sigma_S+\alpha_I {s_F}^*) & -\sigma_S & -(\sigma_S+\alpha_T {s_F}^*) & -(\sigma_S-\delta {s_F}^*) & -(\sigma_S-\delta {s_F}^*) \\ 0 & -K_2 & b_I & 0 & b_T & 0 & 0 \\ 0 & 0 & -K_{3n} & 0 & \alpha_T {s_F}^* & 0 & 0 \\ 0 & (\varepsilon_d \tau_w) & 0 & -K_4 & 0 & (\varepsilon_d \tau_y) & 0 \\ 0 & 0 & (\varepsilon_d \tau_x) & \sigma_T & -K_5 & 0 & (\varepsilon_d \tau_z) \\ 0 & \sigma_U & 0 & 0 & 0 & -K_6 & 0 \\ 0 & 0 & \sigma_F & 0 & 0 & 0 & -K_7 \end{pmatrix} \tag{3.12}$$

Where

$$K_{3n}=(K_3-\alpha_I {s_F}^*)>0 \tag{3.13}$$

Using elementary row-transformation, we have

$$J(E_f)=\begin{pmatrix} -(\sigma_S+b) & -\sigma_S & -(\sigma_S+\alpha_I {s_F}^*) & -\sigma_S & -(\sigma_S+\alpha_T {s_F}^*) & -(\sigma_S-\delta {s_F}^*) & -(\sigma_S-\delta {s_F}^*) \\ 0 & -K_2 & b_I & 0 & b_T & 0 & 0 \\ 0 & 0 & -K_{3n} & 0 & \alpha_T {s_F}^* & 0 & 0 \\ 0 & 0 & 0 & -K_4 & m1 & (\varepsilon_d \tau_y) & 0 \\ 0 & 0 & 0 & 0 & m2 & \dfrac{\sigma_T(\varepsilon_d \tau_y)}{K_4} & (\varepsilon_d \tau_z) \\ 0 & 0 & 0 & 0 & 0 & m3 & m4 \\ 0 & 0 & 0 & 0 & 0 & 0 & m5 \end{pmatrix} \tag{3.14}$$

where

$$m1=\frac{(\varepsilon_d \tau_w)(b_I \alpha_T {s_F}^* + b_T K_{3n})}{K_2 K_{3n}}, m2=\frac{\sigma_T(\varepsilon_d \tau_w)(b_I \alpha_T {s_F}^* + b_T K_{3n})+(\varepsilon_d \tau_x)\alpha_T {s_F}^* K_2 K_4 - K_2 K_{3n} K_4 K_5}{K_2 K_{3n} K_4}$$

$$m3=\frac{\sigma_U(b_I \alpha_T {s_F}^* + b_T K_{3n})\sigma_T(\varepsilon_d \tau_y) - K_2 K_{3n} m1 K_4 K_6}{K_2 K_{3n} m1 K_4}, m4=\frac{\sigma_U(b_I \alpha_T {s_F}^* + b_T K_{3n})(\varepsilon_d \tau_z)}{K_2 K_{3n} m1}$$

$$m5=\frac{\sigma_F \alpha_T {s_F}^* \sigma_T(\varepsilon_d \tau_y) m3 + (\sigma_F \alpha_T {s_F}^*(\varepsilon_d \tau_z) - K_{3n} K_7) K_4 m2}{K_{3n} K_4 m2}$$

Thus, the eigenvalues are:

$$\lambda_1=-(\sigma_S+b)<0, \lambda_2=-(\varepsilon_d \tau_w+\sigma_U+b)<0, \lambda_3=-(K_3-\alpha_I {s_F}^*)<0, \lambda_4=-(\sigma_T+b)<0$$

and

$$\lambda_5 = m2 = \frac{\sigma_T(\varepsilon_d\tau_w)\left(b_I\alpha_T s_F^{\ *} + b_T K_{3n}\right) + (\varepsilon_d\tau_x)\alpha_T s_F^{\ *} K_2K_4 - K_2K_{3n}K_4K_5}{K_2K_{3n}K_4}$$

now, for λ_5 to be negative, we must have

$$\frac{\sigma_T(\varepsilon_d\tau_w)\left(b_I\alpha_T s_F^{\ *} + b_T K_{3n}\right) + (\varepsilon_d\tau_x)\alpha_T s_F^{\ *} K_2K_4 - K_2K_{3n}K_4K_5}{K_2K_{3n}K_4} < 0$$

Simplifying, we have

$$K_2K_{3n}K_4K_5 > b_I\alpha_T s_F(\varepsilon_d\tau_w)\sigma_T + b_T K_{3n}(\varepsilon_d\tau_w)\sigma_T + \alpha_T s_F K_2K_4(\varepsilon_d\tau_x)$$

Thus,

$$\lambda_5 = m2 = \frac{\sigma_T(\varepsilon_d\tau_w)\left(b_I\alpha_T s_F^{\ *} + b_T K_{3n}\right) + (\varepsilon_d\tau_x)\alpha_T s_F^{\ *} K_2K_4 - K_2K_{3n}K_4K_5}{K_2K_{3n}K_4} < 0$$

Similarly,

$$\lambda_6 = m3 = \frac{\sigma_U\left(b_I\alpha_T s_F^{\ *} + b_T K_{3n}\right)\sigma_T(\varepsilon_d\tau_y) - K_2K_{3n}m1K_4K_6}{K_2K_{3n}m1K_4} < 0$$

This is clear since *m2* is negative. Next, for

$$\lambda_7 = m5 = \frac{\sigma_F\alpha_T s_F^{\ *}\sigma_T(\varepsilon_d\tau_y)m3 + \left(\sigma_F\alpha_T s_F^{\ *}(\varepsilon_d\tau_z) - K_{3n}K_7\right)K_4m2}{K_{3n}K_4m2}$$

to be negative, we should have,

$$\sigma_F\alpha_T s_F^{\ *}\sigma_T(\varepsilon_d\tau_y)m3 + \left(\sigma_F\alpha_T s_F^{\ *}(\varepsilon_d\tau_z) - K_{3n}K_7\right)K_4m2 < 0$$

Simplifying, gives

$$\frac{\alpha_I s_F^{\ *}}{K_3}\left(1 + \frac{\phi\left\{\left\{(\varepsilon_d\tau_x)K_7 + \sigma_F(\varepsilon_d\tau_z)\right\}K_2K_4K_6 + b_I\left((\varepsilon_d\tau_w)K_6 + (\varepsilon_d\tau_y)\sigma_U\right)\sigma_T K_7\right\}}{K_7\left\{K_2K_4K_5K_6 - b_T\sigma_T\left((\varepsilon_d\tau_w)K_6 + (\varepsilon_d\tau_y)\sigma_U\right)\right\}}\right) < 1$$

Thus, if $R_C < 1$, λ_7 is negative, implying all the eigenvalues have negative real parts, we thus, established the following result.

Theorem 1: The disease- free equilibrium E_f of (3.1) is locally asymptotically stable (LAS) if $R_C < 1$.

3.4 Global Stability of Disease Free Equilibrium, E_f

The epidemiological implication of the theorem is that HIV can be eliminated (control) from the population when $R_C < 1$, if the initial size of the sub-populations of the model are in the basin of attraction of the DFE. In order to ensure that HIV is independent of the initial size of the sub-populations of the model (3.1), it is necessary to show that the DFE is globally- asymptotically stable

(GAS). One common approach in studying the global asymptotic stability of the DFE is to construct an appropriate Lyapunov function (Li et al., 1999, Fall et al., 2007, Huo et al., 2010, Garba and Gumel, 2010). However, we applied the result introduced by Castillo-Chavez et al. (2002).

Theorem 2: The disease- free equilibrium E_f of (3.1) is globally asymptotically stable (GAS) in Ω if $R_C < 1$.

Proof: To establish the global stability of the disease free equilibrium, the two conditions (H1) and (H2) as in Castillo-Chavez et al. (2002) must be satisfied for $R_C < 1$. We rewrite the model (3.11) in the form:

$$\frac{dX_1}{dt} = F(X_1, X_2), \quad \frac{dX_2}{dt} = G(X_1, X_2); \; G(X_1, 0) = 0 \quad (3.15)$$

where

$$X_1 = \left(s_U^*, s_F^*\right) \text{ and } X_2 = \left(i_U^*, i_F^*, t_U^*, t_F^*, a_U^*, a_F^*\right),$$

with the components of $X_1 \in \Re^2$ denoting the uninfected population and the components of $X_2 \in \Re^6$ denoting the infected population.

The disease-free equilibrium is now denoted as

$$E_f = \left(X_1^*, 0\right), \; X_1^* = \left(\frac{b}{K_1}, \frac{\sigma_S}{K_1}\right)$$

Now, for the first condition, that is globally asymptotically stability of X_1^*, we have

$$\frac{dX_1}{dt} = F\left(X_1, 0\right) = \begin{bmatrix} b - \left(\sigma_S + b\right)s_U^* \\ \sigma_S - b s_F^* \end{bmatrix}$$

a linear differential equations.

Solving, we have

$$s_U^*(t) = \frac{b}{\left(\sigma_S + b\right)} - \frac{b}{\left(\sigma_S + b\right)} e^{-(\sigma_S + b)t} + s_U^*(0) e^{-(\sigma_S + b)t}$$

$$s_F^*(t) = \frac{\sigma_S}{b} - \frac{\sigma_S}{b} e^{-bt} + s_F^*(0) e^{-bt}$$

Now, clearly we have $s_U^*(t) + s_F^*(t) \to 1$ as $t \to \infty$, regardless of the value of $s_F^*(0)$.

Thus $X_1^* = \left(\frac{b}{K_1}, \frac{\sigma_S}{K_1}\right)$ is globally asymptotically stable.

Next, for the second condition, that is

$$\hat{G}(X_1, X_2) = AX_2 - G(X_1, X_2),$$

we have

$$A = \begin{bmatrix} -K_2 & b_I & 0 & b_T & 0 & 0 \\ 0 & -K_{3n} & 0 & \alpha_T s_F^* & 0 & 0 \\ (\varepsilon_d \tau_w) & 0 & -K_4 & 0 & (\varepsilon_d \tau_y) & 0 \\ 0 & (\varepsilon_d \tau_x) & \sigma_T & -K_5 & 0 & (\varepsilon_d \tau_z) \\ \sigma_U & 0 & 0 & 0 & -K_6 & 0 \\ 0 & \sigma_F & 0 & 0 & 0 & -K_7 \end{bmatrix}$$

This is clearly an M-matrix (the off-diagonal elements of A are non-negative).

$$G(X_1, X_2) = \begin{bmatrix} \left(b_I i_F^* + b_T t_F^*\right) - K_2 i_U^* + \delta\left(a_U^* + a_F^*\right) i_U^* \\ \left(\alpha_I i_F^* + \alpha_T t_F^*\right) s_F^* - K_3 i_F^* + \delta\left(a_U^* + a_F^*\right) i_F^* \\ \left(\varepsilon_d \tau_w\right) i_U^* + \left(\varepsilon_d \tau_y\right) a_U^* - K_4 t_U^* + \delta\left(a_U^* + a_F^*\right) t_U^* \\ \sigma_T t_U^* + \left(\varepsilon_d \tau_x\right) i_F^* + \left(\varepsilon_d \tau_z\right) a_F^* - K_5 t_F^* + \delta\left(a_U^* + a_F^*\right) t_F^* \\ \sigma_U i_U^* - K_6 a_U^* + \delta\left(a_U^* + a_F^*\right) a_U^* \\ \sigma_F i_F^* - K_7 a_F^* + \delta\left(a_U^* + a_F^*\right) a_F^* \end{bmatrix}$$

then,

$$\hat{G}(X_1, X_2) = AX_2 - G(X_1, X_2) = \begin{bmatrix} \delta(a_U^* + a_F^*) i_U^* \\ \delta(a_U^* + a_F^*) i_F^* \\ \delta(a_U^* + a_F^*) t_U^* \\ \delta(a_U^* + a_F^*) t_F^* \\ \delta(a_U^* + a_F^*) a_U^* \\ \delta(a_U^* + a_F^*) a_F^* \end{bmatrix}$$

i.e.

$$\hat{G}(X_1, X_2) = \begin{bmatrix} \delta(a_U^* + a_F^*) i_U^*, \delta(a_U^* + a_F^*) i_F^*, \\ \delta(a_U^* + a_F^*) t_U^*, \delta(a_U^* + a_F^*) t_F^*, \\ \delta(a_U^* + a_F^*) a_U^*, \delta(a_U^* + a_F^*) a_F^* \end{bmatrix}^T$$

Since all parameters are assumed non-negative, we have

$$\delta(a_U^* + a_F^*) \geq 0$$

It is thus obvious that $\hat{G}(X_1, X_2) \geq 0$. Hence, the proof is complete.

3.5 Existence of Endemic Equilibrium State, E^*

The endemic equilibrium state is the state in which the disease is persistence. That is the coordinates should satisfy the conditions:

$$E^* = (s_U^*, s_F^*, i_U^*, i_F^*, t_U^*, t_F^*, a_U^*, a_F^*):$$
$$s_U^* > 0, s_F^* > 0, i_U^* > 0, i_F^* > 0, t_U^* > 0, t_F^*, a_U^* > 0, a_F^* > 0$$

As described in Akinwande (1996), Hethcote (2000), it is too cumbersome to explicitly find the coordinates points of the endemic state in models with vital dynamic (births not equal to deaths), standard incidence and disease induced death, but at least we can prove its existence.

Adding the equations of the system (3.11), at equilibrium state, gives

$$\begin{aligned} &\sigma_S(1 - s_F^* - i_U^* - i_F^* - t_U^* - t_F^* - a_U^* - a_F^*) \\ &-(\alpha_I i_F^* + \alpha_T t_F^*) s_F^* - b s_F^* + \delta(a_U^* + a_F^*) s_F^* \\ &+(b_I i_F^* + b_T t_F^*) - (\varepsilon_d \tau_w + \sigma_U + b) i_U^* + \delta(a_U^* + a_F^*) i_U^* \\ &+(\alpha_I i_F^* + \alpha_T t_F^*) s_F^* - (\varepsilon_d \tau_x + \sigma_F + b) i_F^* + \delta(a_U^* + a_F^*) i_F^* \\ &+(\varepsilon_d \tau_w) i_U^* + (\varepsilon_d \tau_y) a_U^* - (\sigma_T + b) t_U^* + \delta(a_U^* + a_F^*) t_U^* \\ &+\sigma_T t_U^* + (\varepsilon_d \tau_x) i_F^* + (\varepsilon_d \tau_z) a_F^* - b t_F^* + \delta(a_U^* + a_F^*) t_F^* \\ &+\sigma_U i_U^* - (\varepsilon_d \tau_y + b + \delta) a_U^* + \delta(a_U^* + a_F^*) a_U^* \\ &+\sigma_F i_F^* - (\varepsilon_d \tau_z + b + \delta) a_F^* + \delta(a_U^* + a_F^*) a_F^* = 0 \end{aligned}$$

Simplifying, we have

$$\begin{aligned} &(\sigma_S - \delta(a_U^* + a_F^*)) \begin{pmatrix} 1 - s_F^* - i_U^* - i_F^* - t_U^* \\ - t_F^* - a_U^* - a_F^* \end{pmatrix} \\ &= b \begin{pmatrix} s_F^* + i_U^* + (1-\theta) i_F^* + t_U^* \\ + (1 - \theta\phi) t_F^* + a_U^* + a_F^* \end{pmatrix} \end{aligned} \quad (3.16)$$

Since all parameters are non-negative, we have R.H.S > 0 and from (3.10)

$$(1 - s_F^* - i_U^* - i_F^* - t_U^* - t_F^* - a_U^* - a_F^*) > 0$$

then

$$(\delta a_U^* + \delta a_F^*) < \sigma_S \Rightarrow a_U^* < \frac{\sigma_S}{\delta}, a_F^* < \frac{\sigma_S}{\delta}$$

i.e.

$$a_U^* > 0, a_U^* < 1, a_U^* < \frac{\sigma_S}{\delta} \text{ and } a_F^* > 0, a_F^* < 1, a_F^* < \frac{\sigma_S}{\delta}$$

which gives the following ranges of :

$$0 < a_U^{\,*} < \min\left\{1, \frac{\sigma_S}{\delta}\right\} \text{ and } 0 < a_F^{\,*} < \min\left\{1, \frac{\sigma_S}{\delta}\right\}$$

Thus, an endemic equilibrium point exists, where $a_U^{\,*}$ and $a_F^{\,*}$ lies in the interval

$$\left(0, \min\left\{1, \frac{\sigma_S}{\delta}\right\}\right).$$

Remark

The relation $\sigma_S > \delta$ is of great importance and plays a great role when HIV persists. It shows that mortality rate due to HIV infection should be less than the rate at which the susceptible individuals under 15 years of age become sexually active. Thus we remark that $R_C > 1$ implies $\sigma_S > \delta$.

3.6 Bifurcation Analysis

We used the centre manifold theory as described in Castillo-Chavez and Song (2004) for bifurcation analysis. In order to apply the theorem, we make the following change of variables. Let

$$s_F = x_1, i_U = x_2, i_F = x_3, t_U = x_4, t_F = x_5, a_U = x_6, a_F = x_7$$

further by using the vector notation

$$X = (x_1, x_2, x_3, x_4, x_5, x_6, x_7)^T$$

the system (3.11 can be written in the form

$$\frac{dX}{dt} = F = (f_1, f_2, f_3, f_4, f_5, f_6, f_7)^T$$

such that

$$\frac{dx_1}{dt} = f_1 = \sigma_S(1 - x_1 - x_2 - x_3 - x_4 - x_5 - x_6 - x_7) - (\alpha_I x_3 + \alpha_T x_5)x_1 - bx_1 + \delta(x_6 + x_7)x_1 \quad (3.17a)$$

$$\frac{dx_2}{dt} = f_2 = (b_I x_3 + b_T x_5) - K_2 x_2 + \delta(x_6 + x_7)x_2 \quad (3.17b)$$

$$\frac{dx_3}{dt} = f_3 = (\alpha_I x_3 + \alpha_T x_5)x_1 - K_3 x_3 + \delta(x_6 + x_7)x_3 \quad (3.17c)$$

$$\frac{dx_4}{dt} = f_4 = (\varepsilon_d \tau_w)x_2 + (\varepsilon_d \tau_y)x_6 - K_4 x_4 + \delta(x_6 + x_7)x_4 \quad (3.17d)$$

$$\frac{dx_5}{dt} = f_5 = \sigma_T x_4 + (\varepsilon_d \tau_x)x_3 + (\varepsilon_d \tau_z)x_7 - K_5 x_5 + \delta(x_6 + x_7)x_5 \quad (3.17e)$$

$$\frac{dx_6}{dt} = f_6 = \sigma_U x_2 - K_6 x_6 + \delta(x_6 + x_7)x_6 \quad (3.17f)$$

$$\frac{dx_7}{dt} = f_7 = \sigma_F x_3 - K_7 x_7 + \delta(x_6 + x_7)x_7 \quad (3.17g)$$

Now, the Jacobian of the system (3.17) above at the disease free equilibrium (which is the same as the Jacobian of (3.12)) is given by

$$J(E_f)=\begin{pmatrix} -(\sigma_S+b) & -\sigma_S & -(\sigma_S+\alpha_I x_1^*) & -\sigma_S & -(\sigma_S+\alpha_T x_1^*) & -(\sigma_S-\delta x_1^*) & -(\sigma_S-\delta x_1^*) \\ 0 & -K_2 & b_I & 0 & b_T & 0 & 0 \\ 0 & 0 & -K_{3n} & 0 & \alpha_T x_1^* & 0 & 0 \\ 0 & (\varepsilon_d \tau_w) & 0 & -K_4 & 0 & (\varepsilon_d \tau_y) & 0 \\ 0 & 0 & (\varepsilon_d \tau_x) & \sigma_T & -K_5 & 0 & (\varepsilon_d \tau_z) \\ 0 & \sigma_U & 0 & 0 & 0 & -K_6 & 0 \\ 0 & 0 & \sigma_F & 0 & 0 & 0 & -K_7 \end{pmatrix} \tag{3.18}$$

from which it has been shown in (3.8) that the effective basic reproduction number, R_C is given by

$$R_C=\frac{\alpha_I x_1^*}{K_3}\left(1+\frac{\eta\left\{\left\{(\varepsilon_d\tau_x)K_7+\sigma_F(\varepsilon_d\tau_z)\right\}K_2K_4K_6+b_I\left((\varepsilon_d\tau_w)K_6+(\varepsilon_d\tau_y)\sigma_U\right)\sigma_T K_7\right\}}{K_7\left\{K_2K_4K_5K_6-b_T\sigma_T\left((\varepsilon_d\tau_w)K_6+(\varepsilon_d\tau_y)\sigma_U\right)\right\}}\right)$$

Consider the case when $R_C=1$. Suppose, further, that $\alpha_I=\alpha^*$ is chosen as a bifurcation parameter, since R_C is often inconvenient to use directly as bifurcation parameter. Solving for α_I gives $R_C=1$ when

$$\alpha_I=\alpha^*=\frac{K_3}{x_1^*\left(1+\frac{\eta\left\{\left\{(\varepsilon_d\tau_x)K_7+\sigma_F(\varepsilon_d\tau_z)\right\}K_2K_4K_6+b_I\left((\varepsilon_d\tau_w)K_6+(\varepsilon_d\tau_y)\sigma_U\right)\sigma_T K_7\right\}}{K_7\left\{K_2K_4K_5K_6-b_T\sigma_T\left((\varepsilon_d\tau_w)K_6+(\varepsilon_d\tau_y)\sigma_U\right)\right\}}\right)} \tag{3.19}$$

Let V and W be the corresponding left and right eigenvectors associated with the zero eigenvalues of the Jacobian of (3.18) at $\alpha_I=\alpha^*$ (denoted by J_{α^*}) chosen such that $VJ(E_0)=0$ and $J(E_0)W=0$ with $VW=1$ where

$V=[v_1,v_2,v_3,v_4,v_5,v_6]$, and $W=[w_1,w_2,w_3,w_4,w_5,w_6]^T$. Thus,

$$VJ(E_f)=[v_1,v_2,v_3,v_4,v_5,v_6,v_7]\times$$
$$\begin{pmatrix} -(\sigma_S+b) & -\sigma_S & -(\sigma_S+\alpha_I x_1^*) & -\sigma_S & -(\sigma_S+\alpha_T x_1^*) & -(\sigma_S-\delta x_1^*) & -(\sigma_S-\delta x_1^*) \\ 0 & -K_2 & b_I & 0 & b_T & 0 & 0 \\ 0 & 0 & -K_{3n} & 0 & \alpha_T x_1^* & 0 & 0 \\ 0 & (\varepsilon_d \tau_w) & 0 & -K_4 & 0 & (\varepsilon_d \tau_y) & 0 \\ 0 & 0 & (\varepsilon_d \tau_x) & \sigma_T & -K_5 & 0 & (\varepsilon_d \tau_z) \\ 0 & \sigma_U & 0 & 0 & 0 & -K_6 & 0 \\ 0 & 0 & \sigma_F & 0 & 0 & 0 & -K_7 \end{pmatrix}=\begin{pmatrix}0\\0\\0\\0\\0\\0\\0\end{pmatrix}$$

Solving, gives

$$v_1 = 0, v_2 = \frac{\{(\varepsilon_d \tau_w)\sigma_T K_6 + \sigma_U (\varepsilon_d \tau_y)\sigma_T\} v_5}{K_2 K_4 K_6}$$
$$v_3 = \frac{\{b_I \{(\varepsilon_d \tau_w)\sigma_T K_6 + \sigma_U (\varepsilon_d \tau_y)\sigma_T\} K_7 + (\varepsilon_d \tau_x) K_2 K_4 K_6 K_7 + \sigma_F (\varepsilon_d \tau_z) K_2 K_4 K_6\} v_5}{K_2 K_{3n} K_4 K_6 K_7} \quad (3.20)$$
$$v_4 = \frac{\sigma_T v_5}{K_4}, v_5 = v_5, v_6 = \frac{(\varepsilon_d \tau_y)\sigma_T v_5}{K_4 K_6}, v_7 = \frac{(\varepsilon_d \tau_z) v_5}{K_7}$$

Similarly,

$$J(E_f)W = \begin{pmatrix} -(\sigma_S + b) & -\sigma_S & -(\sigma_S + \alpha_I x_1^*) & -\sigma_S & -(\sigma_S + \alpha_T x_1^*) & -(\sigma_S - \delta x_1^*) & -(\sigma_S - \delta x_1^*) \\ 0 & -K_2 & b_I & 0 & b_T & 0 & 0 \\ 0 & 0 & -K_{3n} & 0 & \alpha_T x_1^* & 0 & 0 \\ 0 & (\varepsilon_d \tau_w) & 0 & -K_4 & 0 & (\varepsilon_d \tau_y) & 0 \\ 0 & 0 & (\varepsilon_d \tau_x) & \sigma_T & -K_5 & 0 & (\varepsilon_d \tau_z) \\ 0 & \sigma_U & 0 & 0 & 0 & -K_6 & 0 \\ 0 & 0 & \sigma_F & 0 & 0 & 0 & -K_7 \end{pmatrix} \begin{pmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \\ w_5 \\ w_6 \\ w_7 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

and solving, we have

$$w_1 = \frac{\left\{\begin{array}{c} \sigma_S w_2 + (\sigma_S + \alpha_I x_1^*) w_3 + \sigma_S w_4 + (\sigma_S + \alpha_T x_1^*) w_5 \\ + (\sigma_S - \delta x_1^*) w_6 + (\sigma_S - \delta x_1^*) w_7 \end{array}\right\}}{(\sigma_S + b)}$$

$$w_2 = \frac{\{b_I \alpha_T x_1^* + b_T K_{3n}\} w_5}{K_2 K_{3n}}, \quad w_3 = \frac{\alpha_T x_1^* w_5}{K_{3n}}$$

$$w_4 = \frac{(b_I \alpha_T x_1^* + b_T K_{3n})\{(\varepsilon_d \tau_w) K_6 + (\varepsilon_d \tau_y)\sigma_U\} w_5}{K_2 K_{3n} K_4 K_6}$$

$$w_5 = w_5, w_6 = \frac{\sigma_U \{b_I \alpha_T x_1^* + b_T K_{3n}\} w_5}{K_2 K_{3n} K_6}, w_7 = \frac{\sigma_F \alpha_T x_1^* w_5}{K_{3n} K_7}$$

Computation of a and b:

For a, we have

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)$$

Thus, computing the associated non-zero partial derivatives of F at the DFE for the sign of a we have:

$$a = 2(v_2 w_2 + v_3 w_3 + v_4 w_4 + v_5 w_5)(w_6 + w_7)\delta + 2v_3 w_1 (w_3 \alpha_I + w_5 \alpha_T) + v_6 w_6 (w_6 + 2w_7)\delta + v_7 w_7 (2w_6 + w_7)\delta > 0$$

Similarly, for b, we have

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \alpha^*}(0,0)$$

And thus, computing the associated non-zero partial derivatives of F at the DFE for the sign of b we have:

$$b = x_1^* + \eta x_1^* = x_1^*(1 + \eta) > 0$$

Thus we established the following result.

Theorem 5: The model (3.1) exhibit a subcritical (backward) bifurcation at $\alpha^* = 0$.

By occurrence of backward bifurcation, it means that for $R_C < 1$, a stable disease free-equilibrium coexists with two endemic equilibria: a smaller equilibrium (i.e., with a smaller number of infective individuals) which is stable and a larger one (i.e., with a larger number of infective individuals) which is stable. These two endemic equilibria disappear by saddle node bifurcation when the effective basic reproduction number, R_C is decreased below the critical values $R_C^{sn} < 1$. And for $R_C > 1$, there are only two equilibria: the disease free-equilibrium, which is unstable, and the larger endemic equilibrium, which is stable (Buonomo and Lacitignola). Thus, it is highly remarkable that $R_C^{sn} < 1$ is a necessary and sufficient condition for disease control (elimination) and not $R_C \leq 1$.

4. NUMERICAL VERIFICATION

In this section, we presented some numerical simulation to monitor the dynamics of the full model (3.1) for various values of the associated effective basic reproduction number in order to confirm our analytical results on the global stability of the disease free-equilibrium, occurrence of a backward bifurcation as well as effect of different control strategies. Parameter values based on HIV epidemiology and published data studies shown in table 1 below as in Abdulrahman et. al., (2013) is used for the simulation. A total population of 10,000 is assumed with initial total infected population as 100 (except where stated otherwise).

Table 3.1 Baseline values for parameters for the model (3.1)

S/N	Parameter	Baseline Value	S/N	Parameter	Baseline Value
1	b	0.027	9	ε_d	0.85
2	μ	0.016	10	$\tau_w, \tau_x, \tau_y, \tau_z$	(0-1)
3	δ	0.923	11	θ	0.25
4	c	20	12	ϕ	0.733
5	p	0.01	13	σ_S	0.067
6	η	0.05	14	σ_T	0.067
7	ε_c	0.8	15	σ_U	1.091
8	τ_c	(0-1)	16	σ_F	0.05

4.1 Threshold Simulations

Figure 1 confirms the global asymptotic stability of the endemic equilibrium, with the persistence of the solution profiles for $R_C = 1.571$ and different initial infected population of 100 and 500. Figure 2 illustrate the backward bifurcation phenomenon of the model, with the persistence of the disease despite the two different effective basic reproduction numbers are less than one, i.e. $R_C = 0.935$ and $R_C = 0.947$. And with $R_C^{sn} = 0.320$, using same variables as in Figure 1, we clearly see in Figure 3 that the solution profiles converges to the disease free equilibrium in all cases which confirms our analytical result of global asymptotical stability of disease free equilibrium.

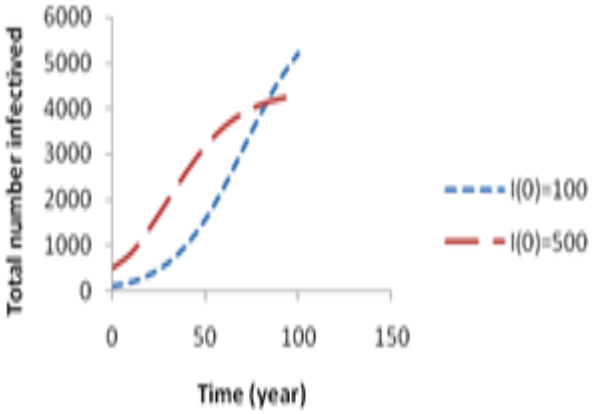

Figure 1: Total number of infected individuals with different initial infected population of $I(0) = 100$ and $I(0) = 500$. Control parameters values are, $\tau_c = \tau_w = \tau_y = 0, \tau_x = 0.01,$ and $\tau_z = 0.1$, which gives $R_C = 1.571$.

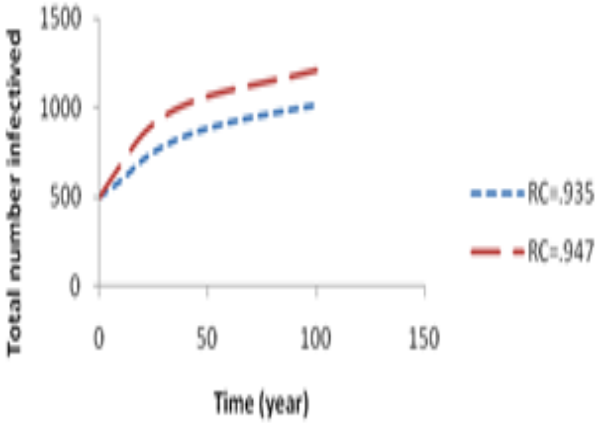

Figure 2: Total number of infected individuals with two different effective basic reproduction numbers which are

less than unity, i.e. $R_C = 0.935$ and $R_C = 0.947$. Control parameters values are $\tau_c = \tau_w = \tau_x = 0, \tau_y = \tau_z = 0.1$ and $\tau_c = \tau_w = \tau_x = \tau_y = \tau_z = 0.1$ respectively.

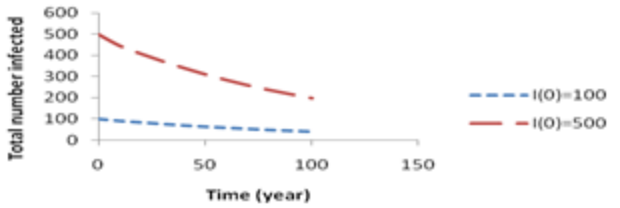

Figure 3: Total number of infected individuals with different initial infected population of $I(0) = 100$ and $I(0) = 500$. Control parameters values are $\tau_c = \tau_w = \tau_x = \tau_y = \tau_z = 0.5$, which gives $R_C = 0.320$.

4.1.1 Effect of Control Strategies

The full model is simulated to assess the effect of condom usage and ART ($\tau_c, \tau_w, \tau_x, , \tau_y$ and τ_z) as follows:

No Control

Figure 4 illustrate the situation in which there is no control. The infected individuals are increasing. Though, after some years law of diminishing returns set in, where there are fewer susceptible individuals to be infected.

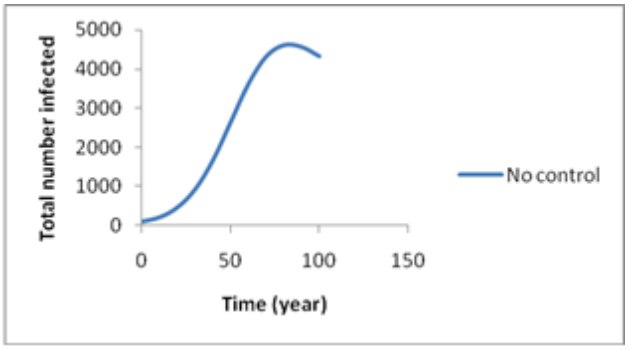

Figure 4: Total number of infected individuals without any control measure. Control parameters used are $\tau_c = \tau_w = \tau_x = \tau_y = \tau_z = 0$, which gives $R_C = 1.851$.

Single Strategies

Here simulations are carried out to monitor the impact of 3 different rates level (low, moderate and high) of each of the 5 control strategies. Figure 5 reveals that condom usage, τ_c only cannot control the disease at low or moderate rates. Though, the disease can be control with high rate of condom usage. The control parameters τ_w, τ_y and τ_z at all the 3 rates level show disease persistence and thus, not enough for disease control as illustrated in Figure 6, 8 and 9 respectively. The τ_x control at any of the 3 rates level shows convergence of the solutions to disease free-equilibrium as depicted in figure 7.

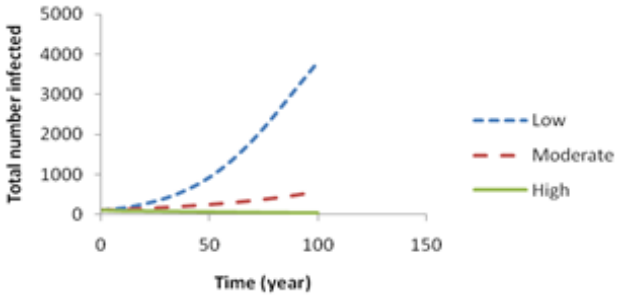

Figure 5: Total number of infected individuals with respect to the 3 different condom usage rates only. Control parameters used are $\tau_w = \tau_x = \tau_y = \tau_z = 0$ and $\tau_c = 0.25, 0.5, 0.75$ respectively for the 3 control levels which give $R_C = 1.481$ for low rate, $R_C = 1.111$ for moderate rate and $R_C = 0.741$ for high rate.

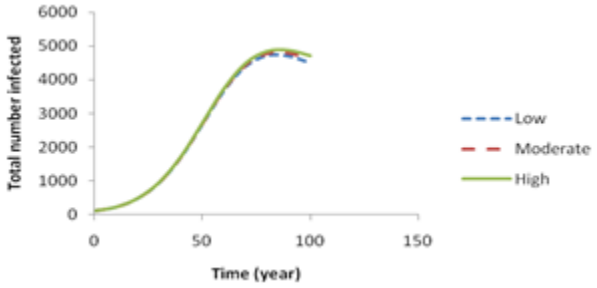

Figure 6: Total number of infected individuals with respect to the 3 different treatment rates for individuals in I_U class only. Control parameters used are $\tau_c = \tau_x = \tau_y = \tau_z = 0$ and $\tau_w = 0.25, 0.5, 0.75$ respectively for the 3 control levels which give $R_C = 1.854$ for low rate, $R_C = 1.856$ for moderate rate and $R_C = 1.858$ for high rate.

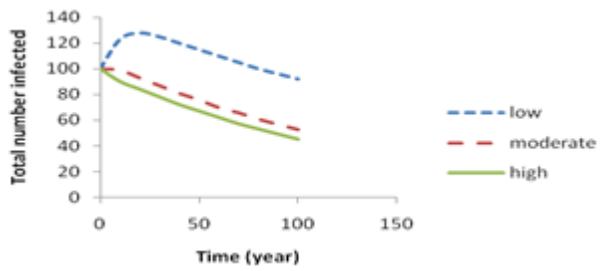

Figure 7: Total number of infected individuals with respect to the 3 different treatment rates for individuals in I_F class only. Control parameters used are $\tau_c = \tau_w = \tau_y = \tau_z = 0$ and $\tau_x = 0.25, 0.5, 0.75$ respectively for the 3 control levels which give $R_C = 0.686$ for low rate, $R_C = 0.507$ for moderate rate and $R_C = 0.435$ for high rate.

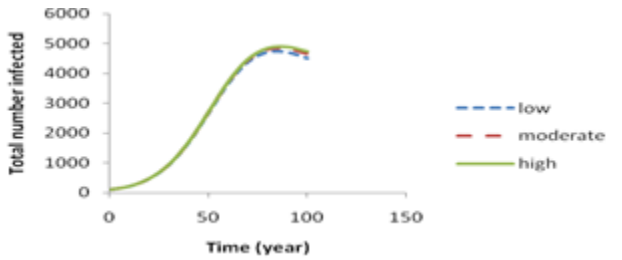

Figure 8: Total number of infected individuals with respect to the 3 different treatment rates for individuals in A_U class only. Control parameters used are $\tau_c = \tau_w = \tau_x = \tau_z = 0$ and $\tau_y = 0.25, 0.5, 0.75$ respectively for the 3 control levels which give $R_C = 1.854$ for low rate, $R_C = 1.857$ for moderate rate and $R_C = 1.858$ for high rate.

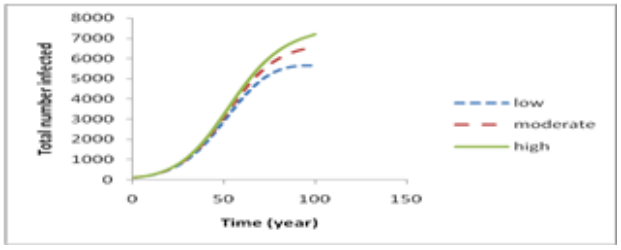

Figure 9: Total number of infected individuals with respect to the 3 different treatment rates for individuals in A_F class only. Control parameters used are $\tau_c = \tau_w = \tau_x = \tau_y = 0$ and $\tau_z = 0.25, 0.5, 0.75$ respectively for the 3 control levels which give $R_C = 1.883$ for low rate, $R_C = 1.904$ for moderate rate and $R_C = 1.920$ for high rate.

Combined Strategies

Here simulations are carried out to monitor the impact of the 3 different rate levels (low, moderate and high) of the 5 combined control strategies $\tau_c, \tau_w, \tau_x, \tau_y$ and τ_z. Figure 9 shows convergence of the solutions to disease free equilibrium for even a low level of the combine strategies.

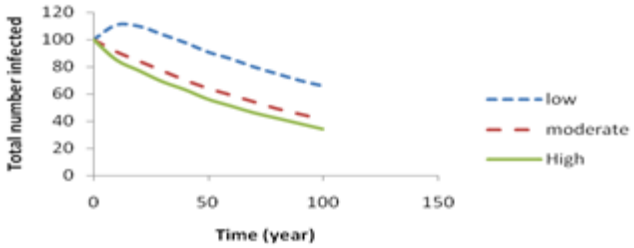

Figure 10: Total number of infected individuals with respect to the 3 different rates of the combined 5 controls. Values used for each control parameter are 0.25, 0.5, 0.75 which give $R_C = 0.564$ for low rate, $R_C = 0.320$ for moderate rate and $R_C = 0.186$ for high rate respectively.

5. CONCLUSION

In this paper, we have presented a mathematical model which incorporated some important factors that plays significant role in the transmission dynamics and control of HIV/AIDS. These factors are: vital dynamics, vertical transmission, public enlightenment, condom usage, standard incidence, disease induced death due AIDS infection, treatment with ART and sexual maturity. Our analysis reveals that the disease can be control if the effective basic reproduction number, R_C is less than one and also the existence of backward bifurcation which can be attributed to the effect of treatment of individuals with AIDS.

Furthermore, our analysis reveals that treatment of HIV^+ infected individuals under 15 years of age and/or AIDS infected individuals gives rise to HIV/AIDS morbidity. This is because treatment prolongs their lives, thereby increasing the disease morbidity. Thus, for optimum result, treatment with ART should target more seriously on HIV^+ sexually active individuals regardless of whether their CD4 counts is high or low. And not necessarily on those with CD4 counts less than 350/uL neither the children as recommended by WHO [a, b], 2010 respectively. This is so because HIV^+ sexually active individuals' treatment with ART will reduce both sexual and vertical transmission rate which are the most common ways of HIV transmission and hence highly reduce the risk of children to be born HIV^+ as well as progression to AIDS and death.

Finally, there is need to model the effects of condom usage and treatment on both morbidity and mortality of HIV/AIDS disease. This would enable us to determine the efficiency and cost-effectiveness of different intervention strategies on reducing both the morbidity and mortality of HIV/AIDS pandemic.

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