

A Mathematical Model Analysis on the Dynamics of HIV/AIDS with Age Structure and Inflow Immigrants in Ethiopia

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Abstract: In this work we considered a nonlinear deterministic dynamical system to study the dynamics of HIV/AIDS with age structure and different mode of transmissions in Ethiopia. We found that the diseases free equilibrium point and endemic equilibrium points exist and we perform their local stability and global stability analysis using nonlinear stability methods. We found that the basic reproduction number of the considered dynamical system depends on the considered parameters and using real data collected from different health sectors in Ethiopia we found the numerical value of the reproduction number is $R_0=1.05>1$. This shows that the considered disease spreads in the community. From the sensitivity index of the dynamical system we found that the most sensitive parameter is the transmission rate of unaware infective humans to aware infective θ . We also showed that the effect of all parameters on the basic reproduction number using numerical simulation.

Keywords: Age Structure, HIV/AIDS Dynamics, Stability Analysis, Sensitivity Analysis, Numerical Simulation

1. Introduction

HIV/AIDS remains a major global health problem affecting approximately 70 million people worldwide causing significant morbidity and mortality [1].

In Ethiopia in 2018, 690 000 people were living with HIV. HIV incidence per 1000 uninfected the number of new HIV infections among the uninfected population over one year among all people of all ages was 0.24. 23 000 people were newly infected with HIV and 11 000 people died from an AIDS-related illness. There has been progress in the number of AIDS-related deaths since 2010, with a 45% decrease, from 20 000 deaths to 11 000 deaths. The number of new HIV infections has also decreased, from 29 000 to 23 000 in the same period. The 90–90–90 targets envision that, by 2020, 90% of people living with HIV will know their HIV status, 90% of people who know their HIV-positive status will be accessing treatment and 90% of people on treatment will have suppressed viral loads. In terms of all people living with HIV, reaching the 90–90–90 targets means that 81% of all people living with HIV are on treatment and 73% of all

people living with HIV are virally suppressed. In 2018 in Ethiopia 79% of people living with HIV knew their status and 65% of people living with HIV were on treatment [2].

The Human Immunodeficiency Virus (HIV) infects cells of immune system such as helper T cells (specifically CD4+ T cells), macrophages, and dendritic cells. HIV compromises the human immune system and reduces the ability of the body to fight back infections and diseases. The most advanced stages of HIV infection is usually called Acquired Immunodeficiency Syndrome (AIDS). AIDS is one of the leading causes of death worldwide that is affecting virtually every nation. Even if HIV/AIDS is not permanently curable, main methods used to fight against it are preventive mechanisms (which include: abstinence, faithfulness and protection) which mainly rely on the level of behavioral change of the population, and providing Antiretroviral Therapy (ART) for those infected [3].

Mathematical models have played a major role in increasing our understanding of the dynamics of infectious diseases. Several models have been proposed to study the effects of some factors on the transmission dynamics of these

infectious diseases including HIV/AIDS and to provide guidelines as to how the spread can be controlled. Among these models include those of Anderson et al. [4] who presented a preliminary study of the transmission dynamics of HIV by proposing a model to study the effects of various factors on the transmission of the disease, Stilianakis et al. [5]. who proposed and gave a detailed analysis of a dynamical model that describes the pathogenesis of HIV, and Tripathi et al. [6] who proposed a model to study the effects of screening of unaware infective on the transmission dynamics of HIV/AIDS. Several other models proposed to study dynamics of HIV/AIDS can be found in ([7- 15], and the references therein).

This paper presents a deterministic model for age structure, the combined effect of unaware infective immigrants, different mode of transmissions and aware infective immigrants, for predicting the epidemiological trends of HIV that exploits HIV surveillance data to model the disease evolution in Ethiopia. The results are presented graphically and discussed qualitatively in the following sections.

2. The Mathematical Model

Our initial model [16] is represented by five ordinary differential equations. Our extended model is represented by seven ordinary differential equations by adding two more compartments based on the following basic assumptions. For this dynamical system we considered susceptible classes as sexually mature $S_1(t)$ and sexually immature $S_2(t)$, Unaware infective class $I_1(t)$, Aware infective class $I_2(t)$, Pre-AIDS class $P(t)$, AIDS class $A(t)$ and Seropositive/treatment class $S_p(t)$. Individuals will join the susceptible mature compartment $S_1(t)$ by immigrants. Some of these people will leave this compartment due to natural deaths and some others

will go to $I_1(t)$ compartment after getting infected. The remaining people will stay in the $S_1(t)$ compartment itself. People of $S_1(t)$ compartment are likely to get infected by the people of $I_1(t)$, $I_2(t)$ and $P(t)$ compartments only. Individuals will join the susceptible immature compartment $S_2(t)$ by birth. Some of these people will leave this compartment due to natural deaths and some others will go to $I_1(t)$ compartment after getting infected. The remaining people will stay in the $S_2(t)$ compartment itself. People of $S_2(t)$ compartment are likely to get infected by the people of $I_1(t)$, $I_2(t)$ and $P(t)$ compartments only. But the people of AIDS compartment $A(t)$ being physically too weak to participate in sexual activities, cannot transfer infection to susceptible people. In this study we considered that, the transfer of HIV from infected people to susceptible people is by sexual intercourse, vertical transmission and transferring HIV by any other means like sharing needles; blood transfusion.

The population under this study is heterogeneous and varying with time, the whole human population is divided in to seven classes, the HIV can be transmitted by the sexual intercourse with infective peoples, vertical transmission and blood borne transmission. The full blown AIDS class is sexually inactive, the seropositive class could not transmit the disease, all the new infected people are assumed to be initially unaware of the infection and the probability of transferring the disease to susceptible population by Pre-AIDS class person is more than unaware infected and aware infected person i. e. $\beta_3 > \beta_1, \beta_2$. The Pre-AIDS class people grow to AIDS much faster than unaware and aware infected people i. e. $\delta_3 > \delta_1, \delta_2$.

Based on these assumptions we construct the following flow chart which shows the movement of individuals from compartment to compartment.

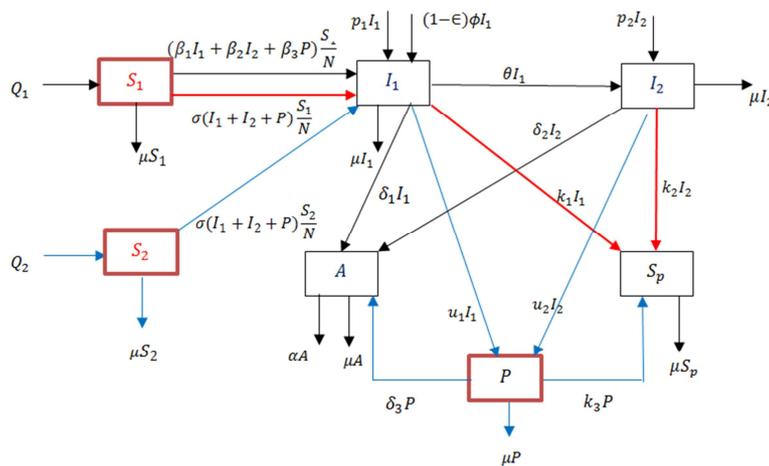


Figure 1. The flow chart of the model.

Based on the above basic assumptions and flow chart we do have the following corresponding dynamical system represented by seven non-linear ordinary differential equations.

$$\frac{dS_1}{dt} = Q_1 - [\beta_1 I_1 + \beta_2 I_2 + \beta_3 P] \frac{S_1}{N} - \sigma[I_1 + I_2 + P] \frac{S_1}{N} - \mu S_1 \quad (1)$$

$$\frac{dS_2}{dt} = Q_2 - \sigma[I_1 + I_2 + P] \frac{S_2}{N} - \mu S_2 \quad (2)$$

$$\frac{dI_1}{dt} = [\beta_1 I_1 + \beta_2 I_2 + \beta_3 P] \frac{S_1}{N} + \sigma[I_1 + I_2 + P] \frac{S_1}{N} + \sigma[I_1 + I_2 + P] \frac{S_2}{N} + p_1 I_1 + (1 - \epsilon)\phi I_1 - (k_1 + \theta + \delta_1 + \mu + u_1) I_1 \quad (3)$$

$$\frac{dI_2}{dt} = p_2 I_2 + \theta I_1 - (k_2 + \delta_2 + \mu + u_2) I_2 \quad (4)$$

$$\frac{dP}{dt} = u_1 I_1 + u_2 I_2 - (\delta_3 + k_3 + \mu)P \quad (5)$$

$$\frac{dS_p}{dt} = k_1 I_1 + k_2 I_2 + k_3 P - \mu S_p \quad (6)$$

$$\frac{dA}{dt} = \delta_1 I_1 + \delta_2 I_2 + \delta_3 P - (\alpha + \mu)A \quad (7)$$

With initial conditions

$$S_1(0) = S_{10}, S_2(0) = S_{20}, I_1(0) = I_{10}, I_2(0) = I_{20}, P(0) = P_0, S_p(0) = S_{p0} \text{ and } A(0) = A_0 \quad (8)$$

Theorem-1: /positivity/

The solutions of the dynamical system (1) - (7) with initial conditions satisfy $S_1(t) > 0, S_2(t) > 0, I_1(t) > 0, I_2(t) > 0, S_p(t) > 0, P(t) > 0, A(t) > 0$ for all $t > 0$. The region $\Omega \subset \mathbb{R}_+^7$ is positively invariant and attracting with respect to system (1) - (7).

Proof

By considering the seven ordinary differential equations and after taking some steps on finding their solutions we do have

i. the first differential equation $\frac{dS_1}{dt} = Q_1 - [\beta_1 I_1 + \beta_2 I_2 + \beta_3 P] \frac{S_1}{N} - \sigma[I_1 + I_2 + P] \frac{S_1}{N} - \mu S_1$

ii. Whose solution is

$S_1(t) = S_1(0)e^{-Q(t)+Q(0)-\mu t} + \int_0^t Q_1 e^{(Q(s)-Q(t))+\mu(s-t)} ds$, since $S_1(0) > 0, Q_1 > 0$ and the exponential function always positive.

iii. the second differential equation

$$\frac{dS_2}{dt} = Q_2 - \sigma[I_1 + I_2 + P] \frac{S_2}{N} - \mu S_2$$

whose solution is $S_2(t) = S_2(0)e^{-Q(t)+Q(0)-\mu t} + \int_0^t Q_2 e^{(Q(s)-Q(t))+\mu(s-t)} ds$, $S_2(0) > 0, Q_2 > 0$ and the exponential function always positive.

iv. the third differential equation $\frac{dI_1}{dt} = [\beta_1 I_1 + \beta_2 I_2 + \beta_3 P] \frac{S_1}{N} + \sigma[I_1 + I_2 + P] \frac{S_1}{N} + \sigma[I_1 + I_2 + P] \frac{S_2}{N} + p_1 I_1 + (1 - \epsilon)\phi I_1 - (k_1 + \theta + \delta_1 + \mu + u_1)I_1$

Whose solution is

$I_1(t) = I_1(0)e^{-Kt+(\beta_1+\sigma)Q(t)-(\beta_1+\sigma)Q(0)+\sigma M(t)-M(0)} + \int_0^t [(\beta_2 + \sigma) \frac{I_2 S_1}{N} + (\beta_3 + \sigma) \frac{P S_1}{N} + \sigma(I_2 + P) \frac{S_2}{N}] e^{Ks-Kt+(\beta_1+\sigma)Q(t)-(\beta_1+\sigma)Q(s)+\sigma M(t)-\sigma M(s)} ds$, since $I_1(0) \geq 0$ and the exponential function always positive.

v. the fourth differential equation $\frac{dI_2}{dt} = p_2 I_2 + \theta I_1 - (k_2 + \delta_2 + \mu + u_2)I_2$ whose solution is $I_2(t) = I_2(0)e^{-ht} + e^{-ht} \int_0^t e^{hs} \theta I_1 ds$ since $I_2(0) \geq 0$, and the exponential function always positive.

vi. the fifth differential equation $\frac{dP}{dt} = u_1 I_1 + u_2 I_2 - (\delta_3 + k_3 + \mu)P$ whose solution is $P(t) = P(0)e^{-(\delta_3+k_3+\mu)t} + e^{-(\delta_3+k_3+\mu)t} \int_0^t e^{(\delta_3+k_3+\mu)s} (u_1 I_1 + u_2 I_2) ds$, since $P(0) \geq 0$ and the exponential function always positive.

vii. the fifth differential equation $\frac{dS_p}{dt} = k_1 I_1 + k_2 I_2 +$

$k_3 P - \mu S_p$ whose solution is $S_p(t) = S_p(0)e^{-\mu t} + e^{-\mu t} \int_0^t e^{\mu s} (k_1 I_1 + k_2 I_2 + k_3 P) ds$, since $S_p(0) \geq 0$ and the exponential function always positive.

viii. the fifth differential equation $\frac{dA}{dt} = \delta_1 I_1 + \delta_2 I_2 + \delta_3 P - (\alpha + \mu)A$ whose solution is $A(t) = A(0)e^{-(\alpha+\mu)t} + e^{-(\alpha+\mu)t} \int_0^t e^{(\alpha+\mu)s} (\delta_1 I_1 + \delta_2 I_2 + \delta_3 P) ds$, since $A(0) \geq 0$ and the exponential function always positive.

Theorem-2: /Boundedness/

The feasible region Ω of the dynamical system (1) - (7) is defined as:

$$\Omega = \left\{ (S_1(t), S_2(t), I_1(t), I_2(t), P(t), S_p(t), A(t)) \in \mathbb{R}_+^7 : 0 < N(t) \leq \frac{Q_1+Q_2}{\mu} \right\}$$
 is bounded.

Proof

Assume that all state variables and parameters are positive. Here we have $N = S_1 + S_2 + I_1 + I_2 + S_p + P + A$ then $\frac{dN}{dt} = \frac{dS_1}{dt} + \frac{dS_2}{dt} + \frac{dI_1}{dt} + \frac{dI_2}{dt} + \frac{dP}{dt} + \frac{dS_p}{dt} + \frac{dA}{dt}$ and thus we have $p_1 I_1 + (1 - \epsilon)\phi I_1 + p_2 I_2 \leq \alpha A$ we obtain $\frac{dN}{dt} \leq Q_1 + Q_2 - \mu N$. Which implies $\frac{dN}{Q_1+Q_2-\mu N} \leq dt$. After some simplification in the integration process we get $N(t) \leq \frac{Q_1+Q_2}{\mu} + N(0)e^{-\mu t}$. And hence as $t \rightarrow \infty$ we have $0 < N(t) \leq \frac{Q_1+Q_2}{\mu}$ which shows that the total population is bounded.

3. Equilibrium Points of the Dynamical System

3.1. Disease Free Equilibrium Point /DFE/

The disease free equilibrium point is obtained by setting the right hand side of the dynamical system (1) - (7) equal to zero with assumptions there are neither infective people nor AIDS patients, that is $I_1 = I_2 = A = P = 0$. And thus we obtain the disease free equilibrium point of the dynamical system is $E_0 = (\frac{Q_1}{\mu}, \frac{Q_2}{\mu}, 0, 0, 0, 0, 0)$.

3.1.1. Basic Reproduction Number R_0

The basic reproduction number is defined as the average number of secondary infections that occur when one infective is introduced into a completely susceptible host population [17].

We can calculate the basic reproduction number, R_0 , using the next generation approach proposed by van den Driessche and Watmough [18]. According to this approach, in order to compute the basic reproduction number, it is important to distinguish new infections from all other class transitions in the population. The infected classes are $I_1, I_2,$ and P . We can write system (1)-(7) as: $\dot{x} = \mathcal{F}(x) - \mathcal{V}(x)$, $\mathcal{V} = \mathcal{V}^- - \mathcal{V}^+$, where $x = (S_1, S_2, I_1, I_2, P, S_p, A)$. \mathcal{F} is the rate of appearance of new infection in each class, \mathcal{V} is the rate of transfer into each class by all other means, and \mathcal{V}^+ is the rate of transfer of the infectious individuals out of each class.

The associated matrices, $\mathcal{F}(x)$ for the new infection terms,

and $\mathcal{V}(x)$ for the remaining transition terms are respectively given by,

$$\mathcal{F}(x) = \begin{pmatrix} [\beta_1 I_1 + \beta_2 I_2 + \beta_3 P] \frac{S_1}{N} + \sigma [I_1 + I_2 + P] \frac{S_1}{N} + \sigma [I_1 + I_2 + P] \frac{S_2}{N} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \tag{9}$$

$$\mathcal{V}(x) = \begin{pmatrix} (k_1 + \theta + \delta_1 + \mu + u_1)I_1 - p_1 I_1 - (1 - \epsilon)\phi I_1 \\ (k_2 + \delta_2 + \mu + u_2)I_2 - p_2 I_2 - \theta I_1 \\ (\delta_3 + k_3 + \mu)P - u_1 I_1 - u_2 I_2 \\ (\alpha + \mu)A - \delta_1 I_1 - \delta_2 I_2 - \delta_3 P \\ \mu S_p - k_1 I_1 - k_2 I_2 - k_3 P \\ \mu S_1 - Q_1 \\ \mu S_2 - Q_2 \end{pmatrix} \tag{10}$$

Evaluating the partial derivatives of (9) and bearing in mind that system (1) - (7) has three infected classes, namely I_1, I_2 and P , we obtain

$$F = \begin{pmatrix} (\beta_1 + \sigma) \frac{S_1}{N} + \sigma \frac{S_2}{N} & (\beta_2 + \sigma) \frac{S_1}{N} + \sigma \frac{S_2}{N} & (\beta_3 + \sigma) \frac{S_1}{N} + \sigma \frac{S_2}{N} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} (k_1 + \theta + \delta_1 + \mu + u_1 - p_1 - (1 - \epsilon)\phi) & 0 & 0 \\ -\theta & (k_2 + \delta_2 + \mu + u_2 - p_2) & 0 \\ -u_1 & -u_2 & (\delta_3 + k_3 + \mu) \end{pmatrix}$$

The spectral radius or the largest eigenvalue of the next generation matrix FV^{-1} is the required basic reproduction number and is computed as

$$R_0 = A_1 \frac{1}{\nabla_1} + A_2 \frac{\theta}{\nabla_1 \nabla_2} + A_3 \frac{\theta u_2 + u_1 \nabla_2}{\nabla_1 \nabla_2 \nabla_3}$$

Where $A_1 = \beta_1 \frac{Q_1}{Q_1 + Q_2} + \sigma, A_2 = \beta_2 \frac{Q_1}{Q_1 + Q_2} + \sigma, A_3 = \beta_3 \frac{Q_1}{Q_1 + Q_2} + \sigma, \nabla_1 = (k_1 + \theta + \delta_1 + \mu + u_1 - p_1 - (1 - \epsilon)\phi), \nabla_2 = (k_2 + \delta_2 + \mu + u_2 - p_2)$ and $\nabla_3 = (\delta_3 + k_3 + \mu)$

$$J(E_0) = \begin{bmatrix} -\mu & 0 & -(\beta_1 + \sigma) \frac{Q_1}{Q_1 + Q_2} & -(\beta_2 + \sigma) \frac{Q_1}{Q_1 + Q_2} & -(\beta_3 + \sigma) \frac{Q_1}{Q_1 + Q_2} & 0 \\ 0 & -\mu & -\sigma \frac{Q_2}{Q_1 + Q_2} & -\sigma \frac{Q_2}{Q_1 + Q_2} & -\sigma \frac{Q_2}{Q_1 + Q_2} & 0 \\ 0 & 0 & A_1 - \nabla_1 & A_2 & A_3 & 0 \\ 0 & 0 & \theta & -\nabla_2 & 0 & 0 \\ 0 & 0 & u_1 & u_2 & -\nabla_3 & 0 \\ 0 & 0 & k_1 & k_2 & k_3 & -\mu \end{bmatrix}$$

The corresponding characteristic equation for the eigenvalue λ is with

$$\begin{vmatrix} -\mu - \lambda & 0 & -(\beta_1 + \sigma) \frac{Q_1}{Q_1 + Q_2} & -(\beta_2 + \sigma) \frac{Q_1}{Q_1 + Q_2} & -(\beta_3 + \sigma) \frac{Q_1}{Q_1 + Q_2} & 0 \\ 0 & -\mu - \lambda & -\sigma \frac{Q_2}{Q_1 + Q_2} & -\sigma \frac{Q_2}{Q_1 + Q_2} & -\sigma \frac{Q_2}{Q_1 + Q_2} & 0 \\ 0 & 0 & A_1 - \nabla_1 - \lambda & A_2 & A_3 & 0 \\ 0 & 0 & \theta & -\nabla_2 - \lambda & 0 & 0 \\ 0 & 0 & u_1 & u_2 & -\nabla_3 - \lambda & 0 \\ 0 & 0 & k_1 & k_2 & k_3 & -\mu - \lambda \end{vmatrix} = 0$$

At disease free equilibrium point we have $S_1 + S_2 \approx N$. Thus

$$F = \begin{pmatrix} \beta_1 \frac{S_1}{S_1 + S_2} + \sigma & \beta_2 \frac{S_1}{S_1 + S_2} + \sigma & \beta_3 \frac{S_1}{S_1 + S_2} + \sigma \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

$$\Rightarrow F = \begin{pmatrix} \beta_1 \frac{Q_1}{Q_1 + Q_2} + \sigma & \beta_2 \frac{Q_1}{Q_1 + Q_2} + \sigma & \beta_3 \frac{Q_1}{Q_1 + Q_2} + \sigma \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

Similarly, the partial derivatives of (10) with respect to I_1, I_2 and P at E_0 gives

3.1.2. Local Stability of the Disease Free Equilibrium Point E_0

Theorem-3:

The disease free equilibrium point E_0 of the dynamical system (1) - (7) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof

The Jacobean matrix of the dynamical system (1) - (7) at the DFE point $E_0 = (\frac{Q_1}{\mu}, \frac{Q_2}{\mu}, 0, 0, 0, 0, 0)$ is:

After some calculations using Routh Hurwitz stability criterion we found that all roots of the characteristic equation have negative real part. Hence the disease free equilibrium point is locally asymptotically stable.

3.1.3. Global Stability of Disease-free Equilibrium Point

Theorem-4:

The disease free equilibrium point E_0 is globally asymptotically stable if $R_0 < 1$.

Proof

We construct a Liapunov function by $V = \alpha_1 I_1 + \alpha_2 I_2 + \alpha_3 P + \alpha_4 S_p + \alpha_5 A$ and thus we get V is continuous function and has first order partial derivatives and V has minimum at $E_0 = (\frac{Q_1}{\mu}, \frac{Q_2}{\mu}, 0, 0, 0, 0, 0)$ which is $V((\frac{Q_1}{\mu}, \frac{Q_2}{\mu}, 0, 0, 0, 0, 0)) = 0$. Finally we calculate the time derivative of V along the solution path yields

$$\begin{aligned} \frac{dV}{dt} &= \alpha_1 \frac{dI_1}{dt} + \alpha_2 \frac{dI_2}{dt} + \alpha_3 \frac{dP}{dt} + \alpha_4 \frac{dS_p}{dt} + \alpha_5 \frac{dA}{dt} \\ &= \alpha_1 \left([\beta_1 I_1 + \beta_2 I_2 + \beta_3 P] \frac{S_1}{N} + \sigma [I_1 + I_2 + P] \frac{S_1}{N} \right. \\ &\quad \left. + \sigma [I_1 + I_2 + P] \frac{S_2}{N} - \nabla_1 I_1 \right) \\ &\quad + \alpha_2 (p_2 I_2 + \theta I_1 - (k_2 + \delta_2 + \mu + u_2) I_2) \\ &\quad + \alpha_3 (u_1 I_1 + u_2 I_2 - (\delta_3 + k_3 + \mu) P) \\ &\quad + \alpha_4 (k_1 I_1 + k_2 I_2 + k_3 P - \mu S_p) + \alpha_5 (\delta_1 I_1 \\ &\quad + \delta_2 I_2 + \delta_3 P - (\alpha + \mu) A) \end{aligned}$$

Take the coefficients of I_2, P, S_p and A are equal to zero. Then we get

$$\begin{aligned} -\alpha_5 (\alpha + \mu) A &= 0 \Rightarrow \alpha_5 = 0 \\ -\alpha_4 \mu S_p &= 0 \Rightarrow \alpha_4 = 0 \\ \alpha_1 A_3 - \alpha_3 (\delta_3 + k_3 + \mu) + \alpha_5 \delta_3 &= 0 \\ \Rightarrow \alpha_1 A_3 - \alpha_3 (\delta_3 + k_3 + \mu) &= 0 \\ \Rightarrow \alpha_1 A_3 &= \alpha_3 (\delta_3 + k_3 + \mu) \\ \Rightarrow \alpha_3 &= \frac{\alpha_1 A_3}{(\delta_3 + k_3 + \mu)} = \frac{\alpha_1 A_3}{\nabla_3} \end{aligned}$$

$$I_1^* = \frac{-(Q_1 + Q_2)(\Phi_1 \nabla_1 + \Phi_2 \nabla_1 - \Phi_1 \Phi_2) \pm \sqrt{((Q_1 + Q_2)(\Phi_1 \nabla_1 + \Phi_2 \nabla_1 - \Phi_1 \Phi_2))^2 - 4\Phi_1 \Phi_2 \nabla_1 \nabla_1 (Q_1 + Q_2)^2 (1 - R_0)}}{2\Phi_1 \Phi_2 \nabla_1}$$

$$S_1^* = \frac{Q_1}{(\frac{\Phi_1 I_1^*}{N^*} + \mu)}, S_2^* = \frac{Q_2}{(\frac{\Phi_2 I_1^*}{N^*} + \mu)}, I_2^* = \frac{\theta}{\nabla_2} I_1^*, P^* = \left(\frac{u_1 \nabla_2 + u_2 \theta}{\nabla_2 \nabla_3} \right) I_1^*,$$

$$S_p^* = \left(\frac{k_1 + k_2 \frac{\theta}{\nabla_2} + k_3 \left(\frac{u_1 \nabla_2 + u_2 \theta}{\nabla_2 \nabla_3} \right)}{\mu} \right) I_1^* \text{ and } A^* = \left(\frac{\delta_1 + \delta_2 \frac{\theta}{\nabla_2} + \delta_3 \left(\frac{u_1 \nabla_2 + u_2 \theta}{\nabla_2 \nabla_3} \right)}{(\alpha + \mu)} \right) I_1^*$$

$$\Phi_1 = (\beta_1 + \sigma) + (\beta_2 + \sigma) \frac{\theta}{\nabla_2} + (\beta_3 + \sigma) \left(\frac{u_1 \nabla_2 + u_2 \theta}{\nabla_2 \nabla_3} \right), \Phi_2 = \sigma \left(1 + \frac{\theta}{\nabla_2} + \left(\frac{u_1 \nabla_2 + u_2 \theta}{\nabla_2 \nabla_3} \right) \right)$$

3.2.1. Local Stability of Endemic Equilibrium Point

Theorem-5:

The positive endemic equilibrium point E^* of the system of equations (1) - (7) is locally asymptotically stable if $R_0 > 1$.

Proof

The Jacobian matrix of the system of equations (1) - (7) at the endemic equilibrium point is

$$\begin{aligned} &\Rightarrow \alpha_1 A_2 + \alpha_2 (p_2 - (k_2 + \delta_2 + \mu + u_2)) + \alpha_3 u_2 = 0 \\ &\Rightarrow \alpha_1 A_2 + \alpha_2 (p_2 - (k_2 + \delta_2 + \mu + u_2)) + \frac{\alpha_1 A_3}{\nabla_3} u_2 = 0 \\ &\Rightarrow \frac{\alpha_1 A_2 \nabla_3 + \alpha_1 A_3 u_2}{\nabla_3} - \alpha_2 \nabla_2 = 0 \\ &\Rightarrow \alpha_2 = \frac{\alpha_1 [A_2 \nabla_3 + A_3 u_2]}{\nabla_2 \nabla_3} \end{aligned}$$

Then

$$\begin{aligned} \frac{dV}{dt} &\leq [\alpha_1 (A_1 - \nabla_1) + \alpha_2 \theta + \alpha_3 u_1 + \alpha_4 k_1 + \alpha_5 \delta_1] I_1 \\ &\Rightarrow \frac{dV}{dt} \leq [\alpha_1 (A_1 - \nabla_1) + \alpha_2 \theta + \alpha_3 u_1] I_1 \\ &\Rightarrow \frac{dV}{dt} \leq \left[\alpha_1 \nabla_1 \left(\frac{A_1 \nabla_2 \nabla_3 + A_2 \theta \nabla_3 + A_3 [u_1 \nabla_2 + \theta u_2]}{\nabla_1 \nabla_2 \nabla_3} - 1 \right) \right] I_1 \\ &\Rightarrow \frac{dV}{dt} \leq [\alpha_1 \nabla_1 (R_0 - 1)] I_1 \end{aligned}$$

We note that $\frac{dV}{dt} \leq 0$ if $R_0 < 1$. Furthermore, $\frac{dV}{dt} = 0$ if and only if $I_1 = I_2 = P = S_p = A = 0$. Therefore, the largest compact invariant set in $(S_1, S_2, I_1, I_2, P, S_p, A) \in \Omega: \frac{dV}{dt} = 0$, where $R_0 < 1$ is the singleton $\{E_0\}$. LaSalle's (1976) invariance principle then implies that E_0 is globally stable in Ω if $R_0 < 1$ otherwise it is unstable.

3.2. Endemic Equilibrium Point

The endemic equilibrium point is obtained by setting the right hand side of the dynamical system (1)-(7) equal to zero. Thus we get the endemic equilibrium point is $E^* = (S_1^*, S_2^*, I_1^*, I_2^*, P^*, S_p^*, A^*)$ where

$$J(E^*) = \begin{pmatrix} M_{11} & 0 & M_{13} & M_{14} & M_{15} & 0 & 0 \\ 0 & M_{22} & M_{23} & M_{24} & M_{25} & 0 & 0 \\ \Lambda_1^* & \Lambda_2^* & M_{33} - \nabla_1 & M_{34} & M_{35} & 0 & 0 \\ 0 & 0 & \theta & -\nabla_2 & 0 & 0 & 0 \\ 0 & 0 & u_1 & u_2 & -\nabla_3 & 0 & 0 \\ 0 & 0 & k_1 & k_2 & k_3 & -\mu & 0 \\ 0 & 0 & \delta_1 & \delta_2 & \delta_3 & 0 & -(\alpha + \mu) \end{pmatrix}$$

Where $M_{11} = -(\Lambda_1 + \mu)$, $M_{13} = -\left(\frac{\beta_1 + \sigma}{N}\right)S_1$, $M_{14} = -\left(\frac{\beta_2 + \sigma}{N}\right)S_1$, $M_{15} = -\left(\frac{\beta_3 + \sigma}{N}\right)S_1$

$$M_{22} = -(\Lambda_2 + \mu), M_{23} = -\sigma \frac{S_2}{N}, M_{24} = -\sigma \frac{S_2}{N}, M_{25} = -\sigma \frac{S_2}{N},$$

$$M_{33} = \left(\frac{\beta_1 + \sigma}{N}\right)S_1 + \sigma \frac{S_2}{N}, M_{34} = \left(\frac{\beta_2 + \sigma}{N}\right)S_1 + \sigma \frac{S_2}{N}, M_{35} = \left(\frac{\beta_3 + \sigma}{N}\right)S_1 + \sigma \frac{S_2}{N}$$

The corresponding characteristic equation is

$$\begin{vmatrix} M_{11} - \lambda & 0 & M_{13} & M_{14} & M_{15} & 0 & 0 \\ 0 & M_{22} - \lambda & M_{23} & M_{24} & M_{25} & 0 & 0 \\ \Lambda_1^* & \Lambda_2^* & M_{33} - \nabla_1 - \lambda & M_{34} & M_{35} & 0 & 0 \\ 0 & 0 & \theta & -\nabla_2 - \lambda & 0 & 0 & 0 \\ 0 & 0 & u_1 & u_2 & -\nabla_3 - \lambda & 0 & 0 \\ 0 & 0 & k_1 & k_2 & k_3 & -\mu - \lambda & 0 \\ 0 & 0 & \delta_1 & \delta_2 & \delta_3 & 0 & -(\alpha + \mu) - \lambda \end{vmatrix} = 0$$

After some calculations using Routh Hurwitz stability criterion we found that all roots of the characteristic equation have negative real part, therefore the endemic equilibrium point is locally asymptotically stable.

3.2.2. Global Stability of Endemic Equilibrium Point

Theorem-6:

The endemic equilibrium point E^* is globally asymptotically stable if $Z < Y$, where

$$Z = \gamma_1(\theta I_1 + \Delta_2 I_2^*) + \gamma_2(u_1 I_1 + u_2 I_2 + \nabla_3 P^*) + \gamma_3(k_1 I_1 + k_2 I_2 + k_3 P + \mu S_p^*) + \left(\frac{\beta_1 + \sigma}{N^*}\right)I_1^* S_1 + \left(\frac{\beta_2 + \sigma}{N^*}\right)I_2^* S_1 + \left(\frac{\beta_3 + \sigma}{N^*}\right)P^* S_1 + \left(\frac{\beta_1 + \sigma}{N}\right)I_1 S_1^* + \left(\frac{\beta_2 + \sigma}{N}\right)I_2 S_1^* + \left(\frac{\beta_3 + \sigma}{N}\right)P S_1^* + \frac{\sigma}{N^*} I_1^* S_1 + \frac{\sigma}{N^*} I_2^* S_1 + \frac{\sigma}{N^*} P^* S_2 + \frac{\sigma}{N} I_1 S_2^* + \frac{\sigma}{N} I_2 S_2^* + \frac{\sigma}{N} P S_2^*$$

And

$$Y = -[\mu S_1^* \left(2 - \frac{S_1}{S_1^*} - \frac{S_1^*}{S_1}\right) + \mu S_2^* \left(2 - \frac{S_2}{S_2^*} - \frac{S_2^*}{S_2}\right) + \left(2 - \frac{S_1^*}{S_1} - \frac{S_1}{S_1^*}\right) \left(\frac{\beta_1 + \sigma}{N^*}\right) I_1^* S_1^* + \left(2 - \frac{S_1^*}{S_1} - \frac{S_1}{S_1^*}\right) \left(\frac{\beta_2 + \sigma}{N^*}\right) I_2^* S_1^* + \left(2 - \frac{S_1^*}{S_1} - \frac{S_1}{S_1^*}\right) \left(\frac{\beta_3 + \sigma}{N^*}\right) P^* S_1^* + \left(2 - \frac{S_2^*}{S_2} - \frac{S_2}{S_2^*}\right) \frac{\sigma}{N^*} I_1^* S_2^* + \left(2 - \frac{S_2^*}{S_2} - \frac{S_2}{S_2^*}\right) \frac{\sigma}{N^*} I_2^* S_2^* + \left(2 - \frac{S_2^*}{S_2} - \frac{S_2}{S_2^*}\right) \frac{\sigma}{N^*} P^* S_2^*] - \left[\left(\frac{\beta_1 + \sigma}{N}\right) I_1^* S_1 + \left(\frac{\beta_2 + \sigma}{N}\right) \frac{I_1^*}{I_1} I_2 S_1 + \left(\frac{\beta_3 + \sigma}{N}\right) \frac{I_1^*}{I_1} P S_1 + \frac{\sigma}{N} I_1^* S_2 + \frac{\sigma}{N} \frac{I_1^*}{I_1} I_2 S_2 + \frac{\sigma}{N} \frac{I_1^*}{I_1} P S_2 + \Delta_1 I_1 + \gamma_1 \left(\theta \frac{I_2^*}{I_2} I_1 + \Delta_2 I_2\right) + \gamma_2 \left(u_1 \frac{P^*}{P} I_1 + u_2 \frac{P^*}{P} I_2 + \nabla_3 P\right) + \gamma_3 \left(k_1 \frac{S_p^*}{S_p} I_1 + k_2 \frac{S_p^*}{S_p} I_2 + k_3 \frac{S_p^*}{S_p} P + \mu S_p\right)\right]$$

Proof

We defined a Liapunov function by

$$V = (S_1 - S_1^* \ln S_1) + (S_2 - S_2^* \ln S_2) + (I_1 - I_1^* \ln I_1) + \gamma_1 (I_2 - I_2^* \ln I_2) + \gamma_2 (P - S_p^* \ln P) + \gamma_3 (S_p - S_p^* \ln S_p) + \gamma_4 (A - A^* \ln A)$$

and thus we get V is continuous function and has first order partial derivatives and V has minimum at E^* . Finally we calculate the time derivative of V along the solution path yields

$$\frac{dV}{dt} = \left(1 - \frac{S_1^*}{S_1}\right) \frac{dS_1}{dt} + \left(1 - \frac{S_2^*}{S_2}\right) \frac{dS_2}{dt} + \left(1 - \frac{I_1^*}{I_1}\right) \frac{dI_1}{dt} + \gamma_1 \left(1 - \frac{I_2^*}{I_2}\right) \frac{dI_2}{dt} + \gamma_2 \left(1 - \frac{P^*}{P}\right) \frac{dP}{dt} + \gamma_3 \left(1 - \frac{S_p^*}{S_p}\right) \frac{dS_p}{dt} + \gamma_4 \left(1 - \frac{A^*}{A}\right) \frac{dA}{dt}$$

Substituting the expressions for the derivatives in $\frac{dV}{dt}$, it follows that

$$\begin{aligned} \frac{dV}{dt} = & \left(1 - \frac{S_1^*}{S_1}\right) \left[Q_1 - \left[\left(\frac{\beta_1 + \sigma}{N}\right)I_1 + \left(\frac{\beta_2 + \sigma}{N}\right)I_2 + \left(\frac{\beta_3 + \sigma}{N}\right)P\right]S_1 - \mu S_1\right] \\ & + \left(1 - \frac{S_2^*}{S_2}\right) \left[Q_2 - \frac{\sigma}{N}[I_1 + I_2 + P]S_2 - \mu S_2\right] \\ & + \left(1 - \frac{I_1^*}{I_1}\right) \left[\left[\left(\frac{\beta_1 + \sigma}{N}\right)I_1 + \left(\frac{\beta_2 + \sigma}{N}\right)I_2 + \left(\frac{\beta_3 + \sigma}{N}\right)P\right]S_1 + \frac{\sigma}{N}[I_1 + I_2 + P]S_2 - \Delta_1 I_1\right] \\ & + \gamma_1 \left(1 - \frac{I_2^*}{I_2}\right) [\theta I_1 - \Delta_2 I_2] + \gamma_2 \left(1 - \frac{P^*}{P}\right) [u_1 I_1 + u_2 I_2 - \nabla_3 P] \\ & + \gamma_3 \left(1 - \frac{S_p^*}{S_p}\right) [k_1 I_1 + k_2 I_2 + k_3 P - \mu S_p] + \gamma_4 \left(1 - \frac{A^*}{A}\right) [\delta_1 I_1 + \delta_2 I_2 + \delta_3 P - (\alpha + \mu)A] \end{aligned}$$

$\Rightarrow \frac{dV}{dt} = Z - Y$. Hence, if $Z < Y$ then, $\frac{dV}{dt}$ will be negative definite, implying that $\frac{dV}{dt} < 0$. Also $\frac{dV}{dt} = 0$ if and only if $S_1 = S_1^*, S_2 = S_2^*, I_1 = I_1^*, I_2 = I_2^*, P = P^*, S_p = S_p^*$ and $A = A^*$.

Therefore the endemic equilibrium point E^* is globally asymptotically stable in Ω if $Z < Y$.

4. Parameter Values for Numerical Simulation and Sensitivity Analysis

To perform numerical simulation and sensitivity analysis we collected the following parameter values from different data sources.

Table 1. Parameter descriptions and values.

Parameter	Parameter Description	Value	Data Source
Q_1	Recruitment in to sexual mature population	230000	[19]
Q_2	Recruitment in to sexual immature population	330465	[20]
β_1	The horizontal transmission rate of unaware infective to susceptible individuals	0.83	[21]
β_2	The horizontal transmission rate of aware infective to susceptible individuals	0.7	[21]
β_3	The horizontal transmission rate of Pre-AIDS to susceptible individuals	0.9	[21]
σ	Rate of transmission through blood borne,	0.03	[22]
δ_1	Rate at which unaware infective develop full blown AIDS	0.06	[23]
δ_2	Rate at which aware infective develop full blown AIDS	0.06	[23]
δ_3	Progression rate of pre-AIDS individuals to full blown AIDS	0.4621	[24]
μ	Natural mortality	0.0065	[25]
θ	Rate of status awareness due to screening method	0.79	[2]
k_1	Rate of treatment of unaware infective	0.02	[2]
k_2	Rate of treatment of aware infective	0.65	[2]
k_3	Rate of treatment of Pre-AIDS	0.65	[2]
p_1	Rate of unaware infective immigrants	0.016	[19]
p_2	Rate of aware infective immigrants	0.013	[19]
u_1	Rate of progress to Pre-AIDS from unaware infective	0.36	[24]
u_2	Rate of progress to Pre-AIDS from aware infective	0.57	[24]
ϕ	Rate of vertical transmission	0.45	[26]
ϵ	Probability of death at birth	0.0281	[27]
α	AIDS induced death rate	0.0159	[20]

4.1. Estimation of Basic Reproduction Number R_0

$$R_0 = \left(\beta_1 \frac{Q_1}{Q_1 + Q_2} + \sigma\right) \frac{1}{\nabla_1} + \left(\beta_2 \frac{Q_1}{Q_1 + Q_2} + \sigma\right) \frac{\theta}{\nabla_1 \nabla_2} + \left(\beta_3 \frac{Q_1}{Q_1 + Q_2} + \sigma\right) \frac{\theta u_2 + u_1 \nabla_2}{\nabla_1 \nabla_2 \nabla_3} = 1.0498$$

4.2. Numerical Simulations

The numerical analysis is obtained from the graphs of basic reproduction number with respect to the parameters obtained and given in Table 1.

Rate of transmission of the disease from unaware & aware infective classes

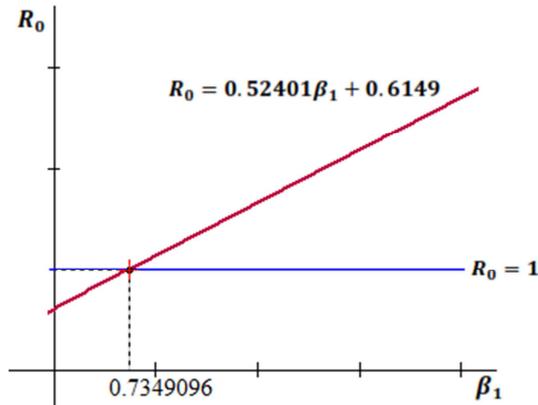


Figure 2. Reproduction number versus the horizontal transmission rate of unaware infective.

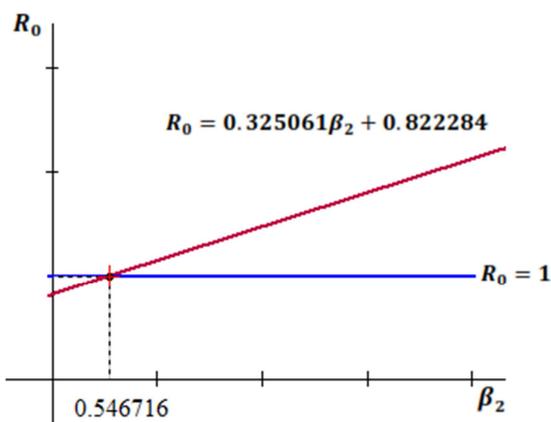


Figure 3. Reproduction number versus the horizontal transmission rate of aware infective.

Figure 2. it is graphical representation of the basic reproduction number R_0 versus rate of transmission of the disease from unaware infective class β_1 and keeping other parameters constant. This figure shows that an increase in the rate of horizontal transmission of unaware infective, β_1 , makes an increase in the reproduction number, R_0 . If $\beta_1 > 0.7349096$ the reproduction number $R_0 > 1$ that indicates the disease persists. When $\beta_1 < 0.7349096$ the reproduction number $R_0 < 1$ this indicates that the disease not persists.

Figure 3. it is graphical representation of the basic reproduction number R_0 versus rate of transmission of the disease from aware infective class β_2 and keeping other parameters constant. This figure shows that an increase in the rate of horizontal transmission of aware infective, β_2 , makes an increase in the reproduction number, R_0 . If $\beta_2 > 0.546716$ the reproduction number $R_0 > 1$ that indicates the disease persists. When $\beta_2 < 0.546716$ the reproduction number $R_0 < 1$ this indicates that the disease not persists.

Rate of transmission of the disease from Pre-AIDS class & blood born transmission

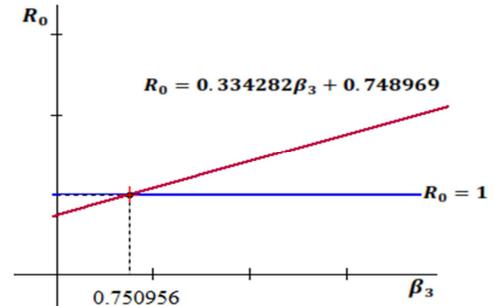


Figure 4. Reproduction number versus the horizontal transmission rate of Pre-AIDS.

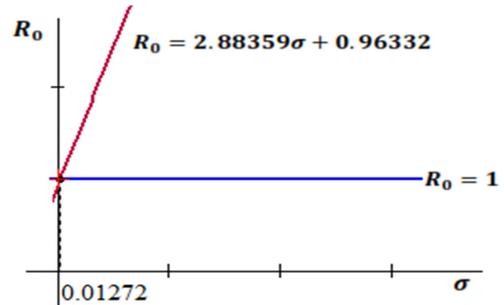


Figure 5. Reproduction number versus Blood born transmission.

Figure 4. it is graphical representation of the basic reproduction number R_0 versus rate of transmission of the disease from Pre-AIDS class β_3 and keeping other parameters constant. This figure shows that an increase in the rate of horizontal transmission of Pre-AIDS, makes an increase in the reproduction number. For $\beta_3 > 0.750956$ we can see the reproduction number $R_0 > 1$ that indicates the disease persists. When $\beta_3 < 0.750956$ the reproduction number $R_0 < 1$ and this indicates that the disease not persists.

Figure 5. it is graphical representation of the basic reproduction number R_0 versus rate of blood born transmission σ and keeping other parameters constant. This figure shows that an increase in the rate of blood born transmission between the parametric values 0 and 0.01272 makes an increase in the reproduction number with $R_0 < 1$ and this indicates that the disease not persists. For $\sigma > 0.01272$ we can see the reproduction number $R_0 > 1$ that indicates the disease persists.

Rate of progress of unaware infective to AIDS class & rate of transmission of Pre-AIDS individuals to seropositive class

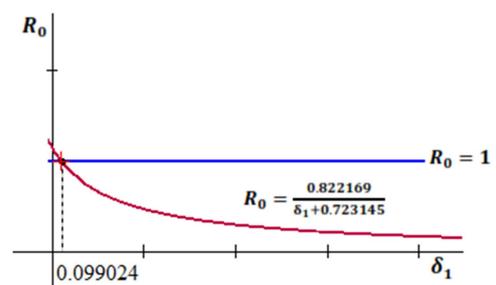


Figure 6. Reproduction number versus the rate of progress of unaware infective to AIDS.

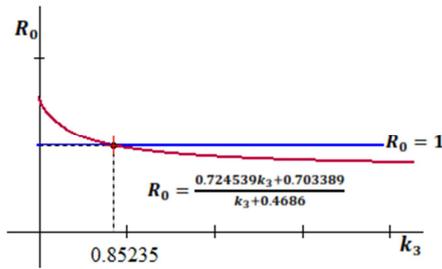


Figure 7. Reproduction number versus the rate of transmission of Pre-AIDS to seropositive class.

Figure 6. it is graphical representation of the basic reproduction number R_0 versus rate of progress of unaware infective to AIDS, δ_1 and keeping other parameters constant. This figure shows that an increase in the rate of progress of unaware infective to AIDS, between the parametric values 0 and 0.099024 makes a decrease in the reproduction number but the reproduction number is greater than one that indicates the disease persists. If the parameter value of δ_1 greater than 0.099024, then the reproduction number decreases and becomes less than one where the disease not persists.

Figure 7. it is graphical representation of the basic reproduction number R_0 versus rate of transmission of Pre-AIDS to seropositive class, k_3 and keeping other parameters constant. This figure shows that an increase in the rate of transmission of Pre-AIDS to seropositive class between the parametric values 0 and 0.85235 makes a decrease in the reproduction number but the reproduction number is greater than one that indicates the disease persists with a decreasing rate. If the parameter value of k_3 greater than 0.85235, then the reproduction number is less than one and we can say the disease not persists.

Rate of progress of Pre-AIDS to AIDS & rate of transmission of unaware infective to aware infective

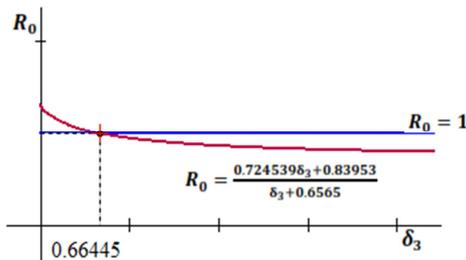


Figure 8. Reproduction number versus the rate of progress of Pre-AIDS to AIDS.

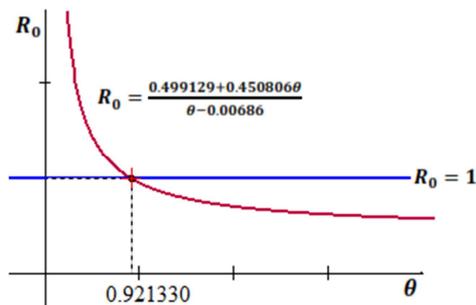


Figure 9. Reproduction number versus the rate of transmission of unaware infective to aware infective.

Figure 8. it is graphical representation of the basic reproduction number R_0 versus rate of progress of Pre-AIDS to AIDS, δ_3 and keeping other parameters constant. This figure shows that an increase in the rate of progress of Pre-AIDS to AIDS between the parametric values 0 and 0.66445 makes a decrease in the reproduction number but the reproduction number is greater than one that indicates the disease persists with a decreasing rate. If the parameter value of δ_3 greater than 0.66445, then the reproduction number decreases and becomes less than one where the not persists.

Figure 9. it is graphical representation of the basic reproduction number R_0 versus rate of transmission of unaware infective to aware infective, θ and keeping other parameters constant. This figure shows that the rate of transmission of unaware infective to aware infective between the parametric values 0 and 0.921330 makes a decrease in the reproduction number but the reproduction number is greater than one that indicates the disease persists with a decreasing rate. If the parameter value of θ greater than 0.921330, then the reproduction number decreases and becomes less than one where the not persists.

Rate of transmission of unaware & and aware infective to seropositive class

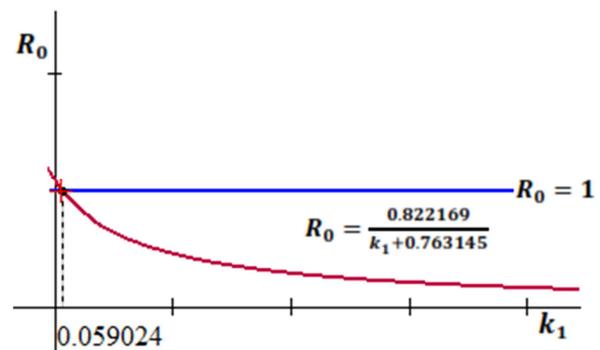


Figure 10. Reproduction number versus the rate of transmission of unaware infective to seropositive class.

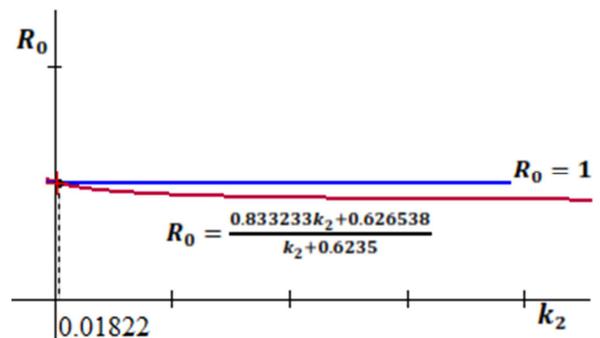


Figure 11. Reproduction number versus the rate of transmission of aware infective to seropositive class.

Figure 10. it is graphical representation of the basic reproduction number R_0 versus rate of transmission of unaware infective to seropositive class, k_1 and keeping other parameters constant. This figure shows that an increase in the rate of transmission of unaware infective to seropositive class between the parametric values 0 and 0.059024 makes a

decrease in the reproduction number, but the reproduction number is greater than one that indicates the disease persists with a decreasing rate. If the parameter value of k_1 greater than 0.059024, then the reproduction number decreases and becomes less than one where the disease dies out.

Figure 11. it is graphical representation of the basic reproduction number R_0 versus rate of transmission of aware infective to seropositive class, k_2 and keeping other parameters constant. This figure shows that an increase in the rate of transmission of aware infective to seropositive class, k_2 , between the parametric values 0 and 0.01822 makes a decrease in the reproduction number, R_0 , but the reproduction number is greater than one that indicates the disease persists with a decreasing rate. If the parameter value of k_2 greater than 0.01822, then the reproduction number is less than one and almost constant in value.

Rate of aware infective immigrants & rate of progress of unaware infective to Pre-AIDS

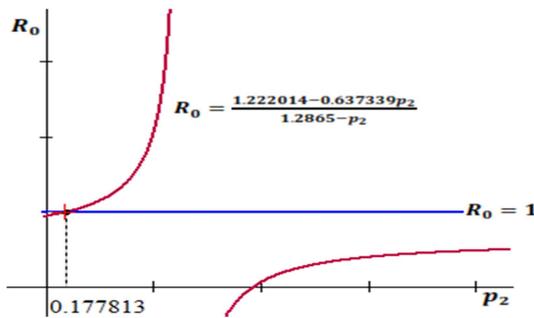


Figure 12. Reproduction number versus the rate of aware infective immigrants.

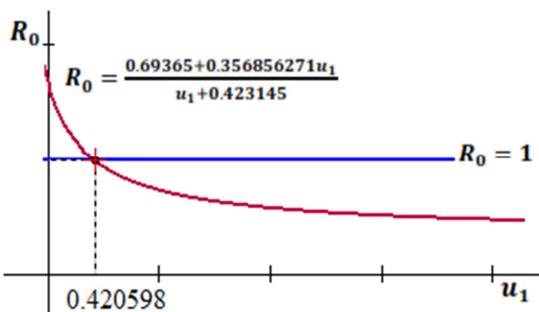


Figure 13. Reproduction number versus rate of progress of unaware infective to Pre-AIDS.

Figure 12. it is graphical representation of the basic reproduction number R_0 versus rate of transmission of aware infective immigrants, p_2 and keeping other parameters constant. This figure shows an increase in the rate of aware infective immigrants between the parametric values 0 and 0.177813 makes an increase in the reproduction number but the reproduction number is less than one that indicates the disease not persists. If the rate of aware infective immigrants between 0.177 and 1.2865 makes an increase in the reproduction number with, $R_0 > 1$ and tell us the disease persists. Whereas, the rate of aware infective immigrants greater than 1.2865, makes an increase in the reproduction number, $R_0 < 1$, and tell us the disease not persists.

Figure 13. it is graphical representation of the basic

reproduction number R_0 versus rate of progress of unaware infective to Pre-AIDS, u_1 and keeping other parameters constant. This figure shows an increase in the rate of progress of unaware infective to Pre-AIDS between the parametric values 0 and 0.420598 makes a decrease in the reproduction number but the reproduction number is greater than one that indicates the disease persists with a decreasing rate. If the parameter value of u_1 greater than 0.420598, then the reproduction number is less than one and we can say the disease not persists.

Rate of vertical transmission & natural mortality

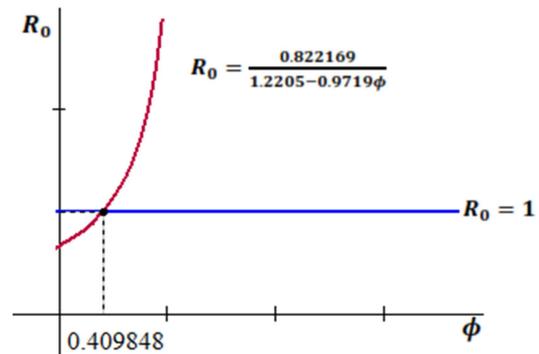


Figure 14. Reproduction number versus rate of vertical transmission.

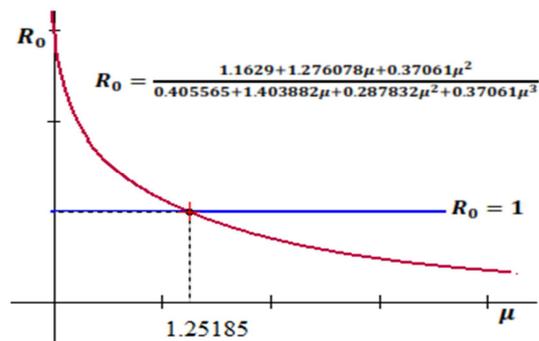


Figure 15. Reproduction number versus Natural mortality.

Figure 14. it is graphical representation of the basic reproduction number R_0 versus rate of vertical transmission, ϕ and keeping other parameters constant. This figure shows that an increase in the rate of vertical transmission between the parametric values 0 and 0.409848 makes an increase in the reproduction number but the reproduction number is less than one that indicates the disease not persists. Whereas, the rate of vertical transmission greater than 0.409848, makes an increase in the reproduction number with $R_0 > 1$, and tell us the disease persists.

Figure 15. it is graphical representation of the basic reproduction number R_0 versus natural mortality, μ and keeping other parameters constant. This figure shows an increase in natural mortality between the parametric values 0 and 1.25185 makes a decrease in the reproduction number but the reproduction number is greater than one that indicates the disease persists with a decreasing rate. If the parameter value of μ greater than 1.25185, then the reproduction number is less than one and we can say the disease not persists.

4.3. Sensitivity Analysis

The parameter values and assumptions of any model are subject to change and error. Sensitivity analysis is the investigation of these potential changes & errors and their impacts on conclusions to be drawn from the model. Here we use it to discover parameters that have a high impact on reproduction number R_0 . We calculate the normalized forward sensitivity index of a variable u that depends differentiable on a parameter p is defined by $SI(p) = \frac{\partial u}{\partial p} X \frac{p}{u}$.

After some simplifications and numerical calculation we get values of sensitivity index for the important parameters mentioned by the table below:

Table 2. Sensitivity indices.

Parameter	Sensitivity Index
θ	-0.61584077
ϕ	0.5584598
β_1	0.414282153
u_1	-0.30336838
β_3	0.286573535
β_2	0.216742618
k_2	-0.200544214
k_3	-0.180049296
δ_3	-0.1280012
σ	0.076614165
δ_1	-0.076614165
u_2	0.072372159
k_1	-0.025538055
p_1	0.020430444
δ_2	-0.018511774
ϵ	-0.016146435
μ	-0.0121058
p_2	0.004010884
Q_1	0.000002469
Q_2	-0.000001719

5. Results and Discussion

Results from Numerical simulation show that as the transmission rate of unaware infective humans to aware infective increases, the basic reproduction number decreases. This will result in decreasing on the transmission of HIV/AIDS. An increase in the rate of horizontal transmission of unaware infective, β_1 , makes an increase in the reproduction number, R_0 . If $\beta_1 > 0.7349096$ the reproduction number $R_0 > 1$ that indicates the disease persists. When $\beta_1 < 0.7349096$ the reproduction number $R_0 < 1$ this indicates that the disease not persists.

An increase in the rate of horizontal transmission of aware infective, β_2 , makes an increase in the reproduction number, R_0 . If $\beta_2 > 0.546716$ the reproduction number $R_0 > 1$ that indicates the disease persists. When $\beta_2 < 0.546716$ the reproduction number $R_0 < 1$ this indicates that the disease not persists. We can also observe that an increase in the rate of horizontal transmission of Pre-AIDS, β_3 , makes an increase in the reproduction number, R_0 . For $\beta_3 > 0.750956$ we can see the reproduction number $R_0 > 1$ that indicates the disease persists. When $\beta_3 < 0.750956$ the reproduction number $R_0 < 1$ and this indicates that the disease not persists. Similarly we can see that an increase in the rate of progress

of unaware infective to AIDS, δ_1 , between the parametric values 0 and 0.099024 makes a decrease in the reproduction number, R_0 , but the reproduction number is greater than one that indicates the disease persists. If the parameter value of δ_1 greater than 0.099024, then the reproduction number decreases and becomes less than one where the disease not persists.

We can see that an increase in the rate of progress of Pre-AIDS to AIDS, δ_3 , between the parametric values 0 and 0.66445 makes a decrease in the reproduction number, R_0 , but the reproduction number is greater than one that indicates the disease persists with a decreasing rate. If the parameter value of δ_3 greater than 0.66445, then the reproduction number decreases and becomes less than one where the disease not persists. For an increase in the rate of transmission of unaware infective to aware infective, θ , between the parametric values 0 and 0.921330 makes a decrease in the reproduction number, R_0 , but the reproduction number is greater than one that indicates the disease persists with a decreasing rate. If the parameter value of θ greater than 0.921330, then the reproduction number decreases and becomes less than one where the not persists.

An increase in the rate of transmission of aware infective to seropositive class, k_2 , between the parametric values 0 and 0.01822 makes a decrease in the reproduction number, R_0 , but the reproduction number is greater than one that indicates the disease persists with a decreasing rate. If the parameter value of k_2 greater than 0.01822, then the reproduction number is less than one and almost constant in value this indicates the disease not persists. An increase in the rate of progress of unaware infective to Pre-AIDS, u_1 , between the parametric values 0 and 0.420598 makes a decrease in the reproduction number, R_0 , but the reproduction number is greater than one that indicates the disease persists with a decreasing rate. If the parameter value of u_1 greater than 0.420598, then the reproduction number is less than one and we can say the disease not persists. We can also observe that an increase in the rate of vertical transmission, ϕ , between the parametric values 0 and 0.409848 makes an increase in the reproduction number, R_0 , but the reproduction number is less than one that indicates the disease not persists. Whereas, the rate of vertical transmission greater than 0.409848, makes an increase in the reproduction number, $R_0 > 1$, and tell us the disease persists.

From sensitive analysis we observed that the most sensitive parameter is the transmission rate of unaware infective humans to aware infective, θ and the least sensitive parameter is the recruitment into sexually immature class, Q_2 . The indices having positive signs increase the value of R_0 as one increase them and those having negative signs decrease the value of R_0 , when they are increased.

6. Conclusion

In this study we have developed a deterministic mathematical model for Age structure and Inflow Infective Immigrants on the Dynamics of HIV/AIDS: dividing susceptible individuals in to sexually immature (i.e age below 15 years) and sexually mature (i.e age 15 years and

above), aware and unaware infective, infective immigrants, Pre-AIDS individuals and treatments of infectious individuals. The stability analysis on the model shows that the disease-free equilibrium point E_0 is to be locally asymptotically stable and globally asymptotically stable when $R_0 < 1$ and the positive endemic equilibrium point E^* is shown to be locally asymptotically stable and globally asymptotically stable for $Z < Y$. Results from Numerical simulation show that as the transmission rate of unaware infective humans to aware infective increases, the basic reproduction number decreases. This will result in decreasing on the transmission of HIV/AIDS. We evaluated the numerical value of the basic reproduction number. Consequently, $R_0 = 1.0498$ that shows the HIV/AIDS disease spread in the community. A sensitivity analysis of the basic reproduction number indicates that the transmission rate of unaware infective humans to aware infective, the rate of vertical transmission and horizontal transmission rate are the most sensitive parameters that can be used to control the spread of the disease.

7. Recommendation

From the above results and discussion we would like to recommend the following to control the spread of HIV/AIDS: the most sensitive parameters like transmission rate of unaware infective humans to aware infective, vertical transmission rate, and horizontal transmission rate of unaware infective and Pre-AIDS individuals are those that should be targeted most by policymakers in the fight against the disease.

References

- [1] WHO, HIV/AIDS- Global Health Observatory (GHO) data. Retrieved from. <http://www.who.int/gho/hiv/en/> (2018).
- [2] <https://www.unaids.org/en/regionscountries/countries/ethiopia>, (January 2020).
- [3] Temesgen Debas Awoke and Semu Mitiku Kassa, "Optimal Control Strategy for TB-HIV/AIDS Co-Infection Model in the Presence of Behaviour Modification" *Processes* 2018, 6, 48.
- [4] R. M. Anderson, G. F. Medley, R. M. May, and A. M. Johnson, "A preliminary study of the transmission dynamics of the human immunodeficiency virus (HIV), the causative agent of AIDS," *IMA Journal of Mathematics Applied in Medicine and Biology*, vol. 3, no. 4, pp. 229–263, 1986.
- [5] N. I. Stilianakis, K. Dietz, and D. Schenzle, "Analysis of a model for the pathogenesis of AIDS," *Mathematical Biosciences*, vol. 145, no. 1, pp. 27–46, 1997.
- [6] A. Tripathi, R. Naresh, and D. Sharma, "Modelling the effect of screening of unaware infectives on the spread of HIV infection," *Applied Mathematics and Computation*, vol. 184, no. 2, pp. 1053–1068, 2007.
- [7] A. B. Gumel, P. N. Shivakumar, and B. M. Sahai, "A mathematical model for the dynamics of HIV-1 during the typical course of infection," *Nonlinear Analysis: Theory, Methods and Applications*, vol. 47, no. 3, pp. 1773–1783, 2001.
- [8] B. D. Aggarwala, "On two ode models for HIV/AIDS development in Canada and a logistic seir model," *Far East Journal of Applied Mathematics*, vol. 6, no. 1, pp. 25–70, 2002.
- [9] B. Flugentius, J. Y. T. Mugisha, and L. S. Luboobi, "An HIV/AIDS model with variable force of infection and its application to the epidemic in Uganda," *The American Journal of Applied Sciences*, vol. 2, pp. 1274–1278, 2005.
- [10] D. Mohammed Ibrahim and B. Seidu, "Modelling the effect of irresponsible infective immigrants on the transmission dynamics of HIV/AIDS," *Advances in Applied Mathematical Biosciences*, vol. 3, pp. 31–40, 2012.
- [11] J. Chin, "Current and future dimensions of the HIV/AIDS pandemic in women and children," *The Lancet*, vol. 336, no. 8709, pp. 221–224, 1990.
- [12] J. T. Bertrand, K. O'Reilly, J. Denison, R. Anhang, and M. Sweat, "Systematic review of the effectiveness of mass communication programs to change HIV/AIDS-related behaviors in developing countries," *Health Education Research*, vol. 21, no. 4, pp. 567–597, 2006.
- [13] M. I. Daabo, O. D. Makinde, and B. Seidu, "Modelling the spread of HIV/AIDS epidemic in the presence of irresponsible infectives," *African Journal of Biotechnology*, vol. 11, no. 51, pp. 11287–11295, 2012.
- [14] M. Coffee, M. N. Lurie, and G. P. Garnett, "Modelling the impact of migration on the HIV epidemic in South Africa," *AIDS*, vol. 21, no. 3, pp. 343–350, 2007.
- [15] P. Essunger and A. S. Perelson, "Modeling HIV infection of CD4⁺ t-cell subpopulations," *Journal of Theoretical Biology*, vol. 170, no. 4, pp. 367–391, 1994.
- [16] Tibebe Tulu Guya and Temesgen Tibebe Mekonnen, "Treatment and Inflow Infective Immigrants on the Dynamics of HIV/AIDS" *IOSR Journal of Mathematics (IOSR-JM)*.
- [17] Diekmann O., Heesterbeek J. A and Metz J. A., on the definition and computation of R_0 in the model for infectious disease in heterogeneous population. *Journal of mathematical Biology*, 28 (1990), 365-382.
- [18] P. van den Driessche and J. Watmough, "Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission," *Mathematical Biosciences*, vol. 180, pp. 29–48, 2002.
- [19] The UN Refugee Agency, ETHIOPIA Refugees and Asylum-seekers as of 30 November 2016.
- [20] https://www.indexmundi.com/ethiopia/demographics_profile.html, (Dec. 13, 2019).
- [21] E. O. Omondi, R. W. Mbogo and L. S. Luboobi, Mathematical modeling of the impact of testing, treatment and control of HIV transmission in Kenya, Omondi et al., *Cogent Mathematics & Statistics* (2018), 5: 1475590 <https://doi.org/10.1080/25742558.2018.1475590>
- [22] Mulugeta, H., Dessie, G., Wagne, F. et al. Seroprevalence and trend of human immunodeficiency virus among blood donors in Ethiopia: a systematic review and meta-analysis. *BMC Infect Dis* 19, 383 (2019). <https://doi.org/10.1186/s12879-019-4012-5>

- [23] <https://www.avert.org/about-hiv-aids/symptoms-stages>, (January 29, 2020)
- [24] Tunde T. Yusuf & Francis Benyah (2012) Optimal strategy for controlling the spread of HIV/AIDS disease: a case study of South Africa, *Journal of Biological Dynamics*, 6:2, 475-494, DOI:10.1080/17513758.2011.628700.
- [25] <https://www.macrotrends.net/countries/ETH/ethiopia/death-rate>, (December 2019)
- [26] Endalamaw, A., Demsie, A., Eshetie, S. *et al.* A systematic review and meta-analysis of vertical transmission route of HIV in Ethiopia. *BMC Infect Dis* 18, 283 (2018). <https://doi.org/10.1186/s12879-018-3189-3>.
- [27] <https://knoema.com/atlas/Ethiopia/Neonatal-mortality-rate>, (February 01, 2020).