IOSR Journal of Mathematics (IOSR-JM)
e-ISSN: 2278-5728, p-ISSN: 2319-765X. Volume 15, Issue 4 Ser. II (Jul – Aug 2019), PP 71-82
www.iosrjournals.org

Treatment and Inflow Infective Immigrants on the Dynamics of HIV/AIDS: A Mathematical Model Analysis

[a]TibebuTulu Guya, [b]Temesgen Tibebu Mekonnen
[a]*Department of Mathematics, Debre Berhan University, Debre Berhan, Ethiopia*
[b]*Department of Mathematics, Debre Berhan University, Debre Berhan Post Code 445, Ethiopia*

Abstract: *In this work we considered a nonlinear deterministic dynamical system to study dynamics of HIV/AIDS with different mode of transmissions. We found that the diseases free equilibrium point and endemic equilibrium point exist and we perform their local stability and global stability analysis using nonlinear stability methods. We found the reproduction number* $R_0 = \frac{(k_2+\delta_2+\mu-p_2)(\beta_1+\sigma)+\theta(\beta_2+\sigma)}{(k_1+\theta+\delta_1+\mu-p_1-(1-\epsilon)\phi)(k_2+\delta_2+\mu-p_2)}$ *which depends on twelve parameters? Using the collected standard data we found the numerical value of the reproduction number is* $R_0 = \mathbf{2.9895} > 1$**.** *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 rate of transmission of the disease to susceptible individuals by unaware infective* β_1*. We also show the effect of all parameters on the basic reproduction number using numerical simulation.*

Keywords: *Nonlinear dynamical system, HIV/AIDS dynamics, Stability analysis, Numerical simulation, Sensitivity analysis.*

Date of Submission: 17-07-2019 Date of acceptance: 05-08-2019

I. Introduction

Diseases can be transmitted in many ways some of which can be classified as either horizontal or vertical. In the case of HIV/AIDS, horizontal transmission can result from direct physical contact between an infected individual and a susceptible individual. Vertical transmission, on the other hand, can result from direct transfer of a disease from an infected mother to an unborn or newborn offspring[19].Researchers found that about 20% of the children infected with HIV develop AIDS in the first year of their lives, and most of them die by the age of four years. The others up to 80% of infected children develop symptoms of HIV/AIDS at school entry age or even during adolescence[2, 26].

The first simple HIV Mathematical epidemic model developed and analyzed by Anderson in 1986 and he suggested that behavioral change was recognized as the major way of combating the spread of HIV/AIDS epidemic as there was no treatment or vaccine to the virus.After the discovery of Anti-retroviral treatment, modeling of HIV/AIDS was directed towards incorporating behavioral change and effects of treatment. Treatment reduces the infectiousness of an infected individual reducing the probability of transmission from an infective individual to a susceptible individual. On the contrary anti- retroviral therapies increases the lifespan of the HIV infectives and as such they can infect more people if the treatment does not reduce infectiousness with no change in social behavior[21].

Mathematical modeling has proved to be an important tool in analyzing the spread and control of different kinds of infectious diseases [3, 18]. The results of modeling and analysis help to improve understanding of the major contributing factors to the pandemic. Mathematical models have been studied and important inferences have been drawn in case of epidemics[1, 5, 6, 7, 9, 15]. The Mathematical model analysis by Valesco - Hernandez and Hsieh found that only significant reductions in the transmission probability can contain the spread of the epidemic. Such reductions could be through adoption of safer sexual practices or through reductions in viral load due to treatmentA model by Ying -Yen and Cooke on Behavioral change and treatment of core groups and its effects on the spread of HIV/AIDS showed that behavioral change and treatment can eradicate the disease however if the treatment and behavioral change levels do not reach critical values, detrimental effects could be realized resulting from slower progression to AIDS without sufficiently lower transmission rates resulting in increased spread of HIV infection[11,12].

Several researchers have developed HIV/AIDS models so as to understand and explain the dynamics and the spread of the disease and succeeded to a large extent. Modeling and Analysis of the spread of AIDS epidemic with immigration of HIV infectives is studied in [10, 22]. A theoretical framework describing the transmission of HIV/AIDS with screening of unaware infective persons is presented in [23, 24]. The joint effect of both medical screening and variable inflow of aware and unaware infective immigrants on the disease

transmission has been studied by [13]. Modeling the Combined Effect of Vertical Transmission and Variable Inflow of Infective Immigrants on the Dynamics of HIV/AIDS has been studied by [25]. The spread of the disease due to vertical transmission has also been studied by [4].

In this work, we proposed an extension of the model studied entitled by Modeling the Combined Effect of Vertical Transmission and Variable Inflow of Infective Immigrants on the Dynamics of HIV/AIDS[25]. Here we have investigated the combined effect of unaware infective immigrants, different mode of transmissions and aware infective immigrants, on the dynamics of HIV/AIDS.

II. The Mathematical Model

Our initial model [25] is represented by four ordinary differential equations. Our extended model is represented by five ordinary differential equations by adding one more compartment based on the following basic assumptions. For this dynamical system we considered susceptible class$S(t)$, Unaware infective class$I_1(t)$, Aware infective class$I_2(t)$, AIDS class$A(t)$ and Seropositive class$S_p(t)$.Individuals will join the susceptible compartment $S(t)$by natural 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 $S(t)$compartment itself. People of $S(t)$compartment are likely to get infected by the people of $I_1(t)$and $I_2(t)$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 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 five classes, the HIV can be transmitted by the sexual intercourse with infective peoples 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 unaware infected person is more than by aware infected person i. e.$\beta_1 > \beta_2$, the unaware infected people grow to AIDS much faster than the aware infected people i. e. $\delta_1 > \delta_2$.

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

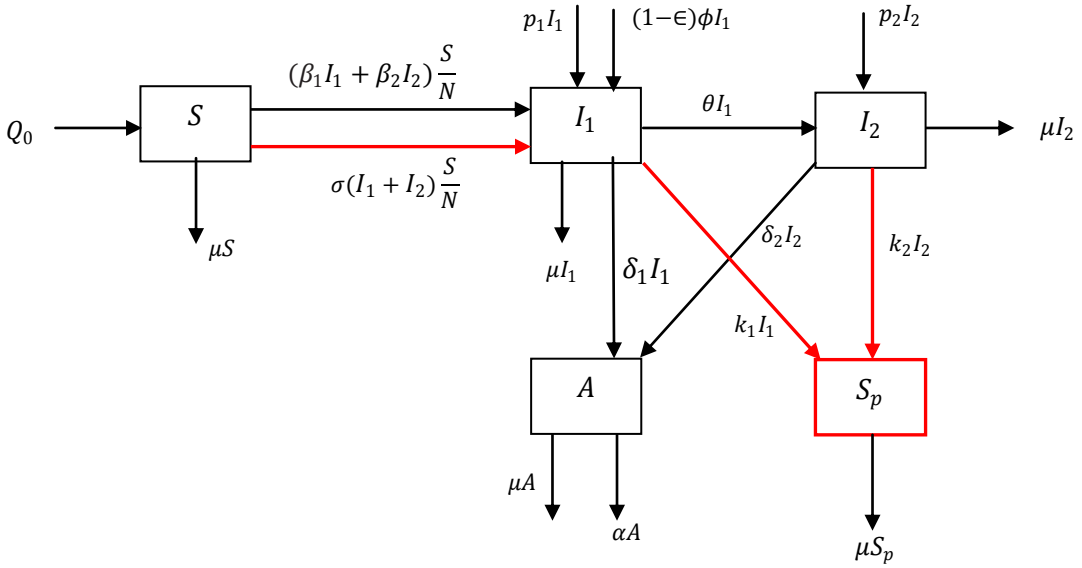

Figure 1: The flow chart of the model

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

$$\frac{dS}{dt} = Q_0 - [\beta_1 I_1 + \beta_2 I_2]\frac{S}{N} - \sigma[I_1 + I_2]\frac{S}{N} - \mu S \qquad (1)$$

$$\frac{dI_1}{dt} = [\beta_1 I_1 + \beta_2 I_2]\frac{S}{N} + \sigma[I_1 + I_2]\frac{S}{N} + p_1 I_1 + (1-\epsilon)\phi I_1 - (k_1 + \theta + \delta_1 + \mu)I_1 \qquad (2)$$

$$\frac{dI_2}{dt} = p_2 I_2 + \theta I_1 - k_2 I_2 - (\delta_2 + \mu)I_2 \qquad (3)$$

$$\frac{dS_p}{dt} = k_1 I_1 + k_2 I_2 - \mu S_p \qquad (4)$$

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

With initial conditions $S(0) = S_0,\ I_1(0) = I_{10},\ I_2(0) = I_{20},\ S_p(0) = S_{p_0}$ and $A(0) = A_0$.

Theorem-1: /positivity/

The solutions of the dynamical system (1) − (5) with initial conditions satisfy $S(t) > 0, I_1(t) > 0, I_2(t) > 0, S_p(t) > 0, A(t) > 0\ for\ all\ t > 0$. The region $\Omega \subset \mathbb{R}^5_+$is positively invariant and attracting with respect to system (1)− (5).

Proof

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

i. Consider the first differential equation $\frac{dS}{dt} = Q_0 - [\beta_1 I_1 + \beta_2 I_2]\frac{S}{N} - \sigma[I_1 + I_2]\frac{S}{N} - \mu S$

Whose solution is $S(t) = S(0)e^{-Q(t)+Q(0)-\mu t} + \frac{1}{e^{Q(t)+\mu t}}\int_0^t Q_0 e^{(Q(s)-Q(t))+\mu(s-t)}ds > 0$,since $S(0) > 0,\ Q_0 > 0$ and the exponential function always positive.

ii. Consider the second differential equation

$$\frac{dI_1}{dt} = [\beta_1 I_1 + \beta_2 I_2]\frac{S}{N} + \sigma[I_1 + I_2]\frac{S}{N} + p_1 I_1 + (1-\epsilon)\phi I_1 - (k_1 + \theta + \delta_1 + \mu)I_1$$

whose solution is $I_1(t) = I_1(0)e^{-Kt+(\beta_1+\sigma)Q(t)-(\beta_1+\sigma)Q(0)} + e^{-Kt+(\beta_1+\sigma)Q(t)}\int_0^t(\beta_1+\sigma)\frac{I_2 S}{N}e^{Ks-(\beta_1+\sigma)Q(s)}ds > 0$, since $I_1(0) \geq 0$ and the exponential function always positive.

iii. Consider the third differential equation$\frac{dI_2}{dt} = p_2 I_2 + \theta I_1 - k_2 I_2 - (\delta_2 + \mu)I_2$

Whose solution is$I_2(t) = I_2(0)e^{-ht} + e^{-ht}\int_0^t e^{hs}\theta I_1 ds > 0$, since $I_2(0) \geq 0$ and the exponential function always positive.

iv. Consider the fourth differential equation $\frac{dS_p}{dt} = k_1 I_1 + k_2 I_2 - \mu S_p$ whose solution is $S_p(t) = S_p(0)e^{-\mu t} + e^{-\mu t}\int_0^t e^{\mu s}(k_1 I_1 + k_2 I_2)ds > 0 S_p(0) \geq 0$ and the exponential function always positive.

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

Theorem-2: /Boundedness/

The feasible region Ω of the dynamical system (1) - (5) is defined as:

$\Omega = \left\{\left(S(t), I_1(t), I_2(t), S_p(t), A(t)\right) \epsilon \Re^5_+ : 0 < N(t) \leq \frac{Q_0}{\mu}\right\}$is bounded.

Proof

Assume that all state variables and parameters are positive. Here we have $N = S + I_1 + I_2 + S_p + A$ then $\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI_1}{dt} + \frac{dI_2}{dt} + \frac{dS_p}{dt} + \frac{dA}{dt}$and thus we have $p_1 I_1 + (1-\epsilon)\phi I_1 + p_2 I_2 \leq \alpha A$ that is $\frac{dN}{dt} \leq Q_0 - \mu N$. Which implies $\frac{dN}{Q_0 - \mu N} \leq dt$. After some simplification in the integration process we get $N(t) \leq \frac{Q_0}{\mu} + N(0)e^{-\mu t}$. And hence as $t \to \infty$ we have $0 < N(t) \leq \frac{Q_0}{\mu}$ which shows that the total population is bounded.

III. 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) - (5)equal to zero with assumptions there are neither infective people nor AIDS patients, that is $I_1 = I_2 = A = 0$. And thus we obtain the disease free equilibrium point of the dynamical system is $E_0 = (\frac{Q_0}{\mu}, 0, 0, 0, 0)$.

3.1.1Basic Reproduction numberR_0

The reproduction number is defined as the average number of secondary cases produced by a typical infected individual during his or her entire life as infectious or infectious period when introduced or allowed to live in a population of susceptible. We shall now compute the basic reproduction number of the present model using the next generation method. The basic reproduction number is a threshold quantity used to study the spread of an infection disease in epidemiological modeling and it is the spectral radius of the next generation matrix **[8]**.In the dynamical system (1)-(5) the rate of appearance of new infections $\mathcal{F}$ and the transfer rate of individuals $\mathcal{V}$ at the disease free steady state $E_0 = (\frac{Q_0}{\mu}, 0, 0, 0, 0)$with $S{\sim}N$is $\mathcal{F} = \begin{bmatrix}\beta_1 + \sigma & \beta_2 + \sigma \\ 0 & 0\end{bmatrix}$, $\mathcal{V} =$

$$\begin{bmatrix}(k_1+\theta+\delta_1+\mu)-(p_1+(1-\epsilon)\phi) & 0\\ -\theta & (k_2+\delta_2+\mu)-p_2\end{bmatrix}$$

and

$$\mathcal{V}^{-1}=\begin{bmatrix}\frac{1}{k_1+\theta+\delta_1+\mu-p_1-(1-\epsilon)\phi} & 0\\ \frac{\theta}{[k_1+\theta+\delta_1+\mu-p_1-(1-\epsilon)\phi][k_2+\delta_2+\mu-p_2]} & \frac{1}{k_2+\delta_2+\mu-p_2}\end{bmatrix}$$

The spectral radius or Eigen value of $\mathcal{FV}^{-}$is the required basic reproduction number obtained by $R_0=\frac{(k_2+\delta_2+\mu-p_2)(\beta_1+\sigma)+\theta(\beta_2+\sigma)}{(k_1+\theta+\delta_1+\mu-p_1-(1-\epsilon)\phi)(k_2+\delta_2+\mu-p_2)}$.

3.1.2Local stability of the disease free equilibrium point E_0

Theorem-3:

The disease free equilibrium point E_0 of the dynamical system (1) - (5) is locally asymptotically stable if $R_0<1$ and unstable if$R_0>1$.

Proof

TheJacobean matrix of the dynamical system (1) - (5) at the DFE point $E_0=(\frac{Q_0}{\mu},0,0,0,0)$ is:

$$J(E_0)=\begin{bmatrix}-\mu & -(\beta_1+\sigma) & -(\beta_2+\sigma) & 0\\ 0 & \beta_1+\sigma-\Delta_1 & \beta_2+\sigma & 0\\ 0 & \theta & -\Delta_2 & 0\\ 0 & k_1 & k_2 & -\mu\end{bmatrix}$$

Where $\Delta_1=k_1+\theta+\delta_1+\mu-p_1-(1-\epsilon)\phi$ and $\Delta_2=k_2+\delta_2+\mu-p_2$

The corresponding characteristic equation for the eigenvalue λ is with

$$\begin{vmatrix}-\mu-\lambda & -(\beta_1+\sigma) & -(\beta_2+\sigma) & 0\\ 0 & \beta_1+\sigma-\Delta_1-\lambda & \beta_2+\sigma & 0\\ 0 & \theta & -\Delta_2-\lambda & 0\\ 0 & k_1 & k_2 & -\mu-\lambda\end{vmatrix}=0$$

Or $(\mu+\lambda)^2[(\beta_1+\sigma-\Delta_1-\lambda)(-\Delta_2-\lambda)-\theta(\beta_2+\sigma)]=0$ or $(\mu+\lambda)^2[\lambda^2+B\lambda+C]=0$ where $C=\Delta_1\Delta_2-(\beta_1+\sigma)\Delta_2-\theta(\beta_2+\sigma)$and $B=(\Delta_2-\beta_1-\sigma+\Delta_1)$.

If $R_0<1$ implies $\frac{\Delta_2(\beta_1+\sigma)+\theta(\beta_2+\sigma)}{\Delta_1\Delta_2}<1$which implies that $\Delta_2(\beta_1+\sigma)+\theta(\beta_2+\sigma)<\Delta_1\Delta_2$

And $\Delta_1\Delta_2-\Delta_2(\beta_1+\sigma)-\theta(\beta_2+\sigma)>0$ which implies that $C>0$. Since $\Delta_1\Delta_2-\Delta_2(\beta_1+\sigma)-\theta(\beta_2+\sigma)>0$ we have $(\Delta_1-(\beta_1+\sigma))\Delta_2-\theta(\beta_2+\sigma)>0$ which implies that $\Delta_1-(\beta_1+\sigma)>0$that is $B>0$. Therefore the quadratic equation $\lambda^2+B\lambda+C=0$ has two negative real roots.In general we have all eigenvalues of the Jacobian matrix are negative. Hence the disease free equilibrium point is locally asymptotically stable and if $R_0>1$ then the characteristic equation will have positive eigenvaluesthereforeE_0 is unstable.

3.1.3Global 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 $V=\alpha_1I_1+\alpha_2I_2+\alpha_3S_p+\alpha_4A$and thus we get V is continuous function and has first order partial derivatives and V has minimum at $E_0=(\frac{Q_0}{\mu},0,0,0,0)$ which is $V(\frac{Q_0}{\mu},0,0,0,0)=0$. Finally we calculate the time derivative of Valong the solution path yields

$\frac{dV}{dt}=\alpha_1\frac{dI_1}{dt}+\alpha_2\frac{dI_2}{dt}+\alpha_3\frac{dS_p}{dt}+\alpha_4\frac{dA}{dt}=\left[\alpha_1[(\frac{\beta_1+\sigma}{N})S-\Delta_1]+\alpha_2\theta+\alpha_3k_1+\alpha_4\delta_1\right]I_1+\left[\alpha_1(\frac{\beta_2+\sigma}{N})S-\alpha_2\Delta_2+\alpha3k2+\alpha4\delta2I2-\alpha3\mu Sp-\alpha4(\alpha+\mu)A\leq\alpha1[\beta1+\sigma-\Delta1]+\alpha2\theta+\alpha3k1+\alpha4\delta1I1+\alpha1\beta2+\sigma-\alpha2\Delta2+\alpha3k2+\alpha4\delta2I2-\alpha3\mu Sp-\alpha4(\alpha+\mu)A$. Take the coefficients of *I2, Sp* and *A* are equal to zero. Then we get$\alpha3=0$, $\alpha4=0$ and $\alpha_2=\frac{\alpha_1(\beta_2+\sigma)}{\Delta_2}$.

Then$\frac{dV}{dt} \leq [\alpha_1[(\beta_1+\sigma)-\Delta_1]+\alpha_2\theta]I_1 = \left[\alpha_1[(\beta_1+\sigma)-\Delta_1]+\frac{\alpha_1(\beta_2+\sigma)\theta}{\Delta_2}\right]I_1 = \left[\alpha_1(\beta_1+\sigma)-\alpha_1\Delta_1+\right.$
$\alpha 1\beta 2+\sigma\theta\Delta 2 I1=\alpha 1\Delta 2\beta 1+\sigma+\beta 2+\sigma\theta\Delta 2-\alpha 1\Delta 1 I1\leq\alpha 1 R0\Delta 1\Delta 2\Delta 2-\alpha 1\Delta 1 I1$

This implies that $\frac{dV}{dt} \leq [\alpha_1 R_0\Delta_1 - \alpha_1\Delta_1]I_1 = \alpha_1\Delta_1[R_0-1]I_1$

We observe that $\frac{dV}{dt} \leq 0$if $R_0 < 1$. Furthermore, $\frac{dV}{dt} = 0$ if and only if $I_1 = I_2 = S_p = A = 0$. Therefore, the largest compact invariant set in $\left(S,\ I_1,\ I_2,\ S_p,\ A\right) \in \Omega : \frac{dV}{dt} = 0$. Thus the endemic equilibrium point E_0 is globally asymptotically stable.

3.2Endemic equilibrium point

The endemic equilibrium point is obtained by setting the right hand side of the dynamical system (1)-(5) equal to zero. Thus we get the endemic equilibrium point is$E^* = (S^*, I_1^*, I_2^*, S_p^*,\ A^*)$where

$S^* = \frac{N}{R_0}$, $I_1{}^* = \frac{Q_0}{\Delta_1}\left(1-\frac{1}{R_0}\right)$, $I_2{}^* = \frac{Q_0\theta\left(1-\frac{1}{R_0}\right)}{\Delta_1\Delta_2}$, $S_p^* = \frac{k_1\Delta_2 Q_0\left(1-\frac{1}{R_0}\right)+k_2 Q_0\theta\left(1-\frac{1}{R_0}\right)}{\mu\Delta_1\Delta_2}$ and

$$A^* = \frac{\delta_1\Delta_2 Q_0\left(1-\frac{1}{R_0}\right)+\delta_2 Q_0\theta\left(1-\frac{1}{R_0}\right)}{(\alpha+\mu)\Delta_1\Delta_2}$$

3.2.1 Local stability of endemic equilibrium point

Theorem-5:

The positive endemic equilibrium point E^* of the system of equations (1) - (5) is locally asymptotically stable if $R_0 > 1$.

Proof

The Jacobian matrix of the system of equations (1) - (5) at the endemic equilibrium point is

$$J(E^*) = \begin{pmatrix} -a-\mu & -\left(\frac{\beta_1+\sigma}{N}\right)S^* & -\left(\frac{\beta_2+\sigma}{N}\right)S^* & 0 & 0 \\ a & \left(\frac{\beta_1+\sigma}{N}\right)S^* - \Delta_1 & \left(\frac{\beta_2+\sigma}{N}\right)S^* & 0 & 0 \\ 0 & \theta & -\Delta_2 & 0 & 0 \\ 0 & k_1 & k_2 & -\mu & 0 \\ 0 & \delta_1 & \delta_2 & 0 & -(\alpha+\mu) \end{pmatrix}$$

The corresponding characteristic equation is

$$\begin{vmatrix} -a-\mu-\lambda & -\left(\frac{\beta_1+\sigma}{R_0}\right) & -\left(\frac{\beta_2+\sigma}{R_0}\right) & 0 & 0 \\ a & \left(\frac{\beta_1+\sigma}{R_0}\right)-\Delta_1-\lambda & \left(\frac{\beta_2+\sigma}{R_0}\right) & 0 & 0 \\ 0 & \theta & -\Delta_2-\lambda & 0 & 0 \\ 0 & k_1 & k_2 & -\mu-\lambda & 0 \\ 0 & \delta_1 & \delta_2 & 0 & -(\alpha+\mu)-\lambda \end{vmatrix} = 0$$

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.

3.2.2Global stability of endemic equilibrium point

Theorem-6:

The endemic equilibrium point E^* is globally asymptotically stable if $\left(\frac{\beta_2+\sigma}{N}\right)S^*\frac{I_1^*I_2}{I_1} + \left(\frac{\beta_2+\sigma}{N}\right)I_2^*S <$
$-\left[\mu S^*\left(2-\frac{S}{S^*}-\frac{S^*}{S}\right)+\left(\frac{\beta_1+\sigma}{N}\right)I_1^*S^*\left(2-\frac{S^*}{S}-\frac{S}{S^*}\right)+\left(\frac{\beta_2+\sigma}{N}\right)I_2^*S^*\left(2-\frac{S^*}{S}-\frac{S}{S^*}\right)\right]+\left(\frac{\beta_2+\sigma}{N}\right)\frac{I_1^*}{I_1}I_2S -$
$\left(\frac{\beta_2+\sigma}{N}\right)S^*I_2^*\left(1-\frac{I_2^*I_1}{I_2I_1^*}-\frac{I_1^*I_2}{I_1I_2^*}\right)$.

Proof

We defined a Liapunov function by

$V = (S-S^*lnS)+(I_1-I_1^*lnI_1)+\gamma_1(I_2-I_2^*lnI_2)+\gamma_2\left(S_p-S_p^*lnS_p\right)+\gamma_3(A-A^*lnA)$and thus we get V is continuous function and has first order partial derivatives and V has minimum at E^* which is E^*0. Finally we calculate the time derivative of Valong the solution path yields$\frac{dV}{dt} = \left(1-\frac{S^*}{S}\right)\frac{dS}{dt}+\left(1-\frac{I_1^*}{I_1}\right)\frac{dI_1}{dt}+\gamma_1\left(1-\right.$
$I2*I2dI2dt+\gamma 21-Sp*SpdSpdt+\gamma 31-A*AdAdt$

$$\frac{dV}{dt} = \left(1 - \frac{S^*}{S}\right)\left[Q_0 - \left[\left(\frac{\beta_1 + \sigma}{N}\right)I_1 + \left(\frac{\beta_2 + \sigma}{N}\right)I_2\right]S - \mu S\right] + \left(1 - \frac{I_1^*}{I_1}\right)\left[\left[\left(\frac{\beta_1 + \sigma}{N}\right)I_1 + \left(\frac{\beta_2 + \sigma}{N}\right)I_2\right]S - \Delta_1 I_1\right]$$
$$+ \gamma_1\left(1 - \frac{I_2^*}{I_2}\right)[\theta I_1 - \Delta_2 I_2] + \gamma_2\left(1 - \frac{S_p^*}{S_p}\right)[k_1 I_1 + k_2 I_2 - \mu S_p] + \gamma_3\left(1 - \frac{A^*}{A}\right)[\delta_1 I_1 + \delta_2 I_2$$
$$- (\alpha + \mu)A]$$

$\frac{dV}{dt} = Z - Y$ where $Z = \left(\frac{\beta_2+\sigma}{N}\right)S^*\frac{I_1^* I_2}{I_1} + \left(\frac{\beta_2+\sigma}{N}\right)I_2^* S$ and $Y = -\left[\mu S^*\left(2 - \frac{S}{S^*} - \frac{S^*}{S}\right) + \left(\frac{\beta_1+\sigma}{N}\right)I_1^* S^*\left(2 - \frac{S^*}{S} - \frac{S}{S^*}\right) + \beta 2 + \sigma NI2 * S * 2 - S * S - SS * + \beta 2 + \sigma NI1 * I1I2S - \beta 2 + \sigma NS * I2 * 1 - I2 * I1I2I1 * - I1 * I2I1I2 *$. 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 $S = S^*, I_1 = I_1^*, I_2 = I_2^*, S_p = S_p^* \text{ and } A = A^*$. Therefore the endemic equilibrium point $E^*$is globally asymptotically stable in Ωif $Z < Y$.

IV. Parameter Estimation For Numerical Simulation And Senstivity Analysis

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

Parameter	Description	Estimated value	Ref.
β_1	Probability of transmission of the disease to susceptible individuals by unaware infective	0.9	[25]
β_2	Probability of transmission of the disease to susceptible individuals by aware infectives	0.7	[25]
δ_1	Rate of development to AIDS from unaware infectives	0.3	[25]
δ_2	Rate of development to AIDS from aware infectives	0.02	[25]
σ	Rate of transmission through blood borne	0.003	[13]
μ	Natural mortality	0.02	[15]
θ	Rate of aware infectives from unaware infectives	0.3	[25]
k_1	Rate of treatment of unaware infectives	0.1	Estimated
k_2	Rate of treatment of aware infectives	0.4	[17]
p_1	Rate of unaware infectives immigrants	0.1	[25]
p_2	Rate of aware infectives immigrants	0.2	[25]
ϕ	Rate of vertical transmission	0.03	[25]
ϵ	Probability of death at birth	0.2	[25]
α	AIDS induced death rate	0.9	[25]

Table 1: Parameter estimation

4.1 Estimation of basic reproduction number R_0

$$R_0 = \frac{(k_2 + \delta_2 + \mu - p_2)(\beta_1 + \sigma) + \theta(\beta_2 + \sigma)}{(k_1 + \theta + \delta_1 + \mu - p_1 - (1 - \epsilon)\phi)(k_2 + \delta_2 + \mu - p_2)}$$

$$R_0 = \mathbf{2.9895}$$

1. **Numerical Simulations**

The numerical analysis is obtained from the graphs of basic reproduction number with respect to the parameters obtained and given in **Table1**.

Rate of transmission of the disease from unaware infective class β_1

Graphical representation of the basic reproduction number R_0versus rate of transmission of the disease from unaware infective class $\beta_1$and keeping other parameters constant

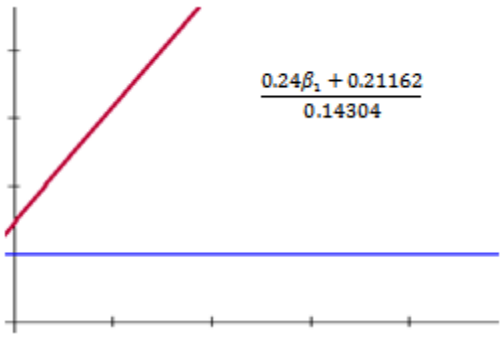

Figure 2: This figure showsan increase in the rate of transmission, β_1, makes an increase in the reproduction number, R_0. That is the disease always persists for any value of parameterβ_1.

Rate of transmission of the disease from aware infective class β_2

Graphical representation of the basic reproduction number R_0versus rate of transmission of the disease from aware infective class $\beta_2$and keeping other parameters constant

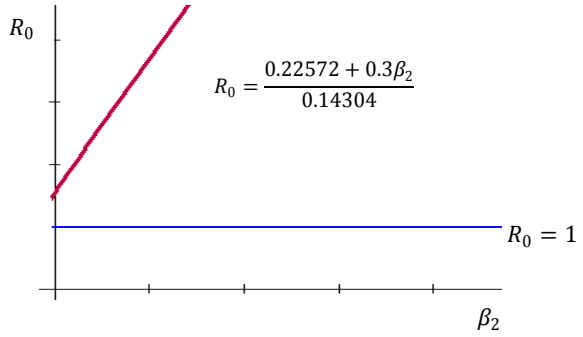

Figure 3: This figure showsan increase in the rate of transmission, β_2, makes an increase in the reproduction number, R_0 . That is the disease always persists for any value of parameter β_2

Rate of blood borne transmission of the disease σ

Graphical representation of the basic reproduction number R_0versus rate of blood borne transmission of the disease $\sigma$and keeping other parameters constant

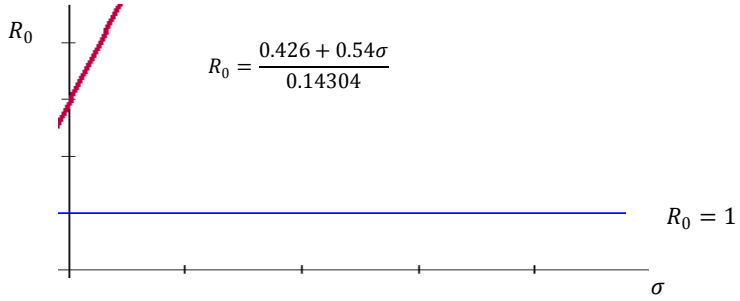

Figure 4: This figure showsan increase in the rate of blood borne transmission, σ, makes an increase in the reproduction number, R_0. That is the disease always persists for any value of parameterσ

Rate of progress of unaware infective to AIDS δ_1

Graphical representation of the basic reproduction number R_0versus rate of progress of unaware infective to AIDS $\delta_1$and keeping other parameters constant

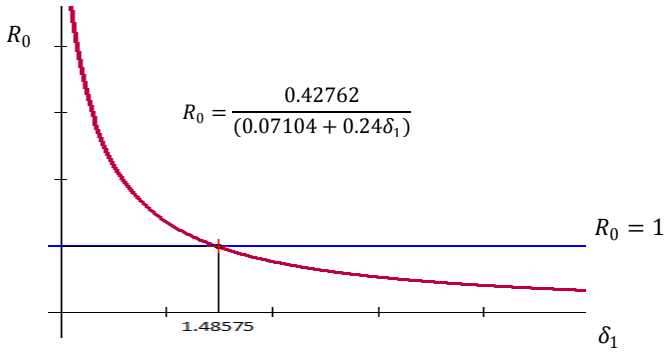

Figure 5: This figure showsan increase in the rate of progress of unaware infective to AIDS, δ_1, between the parametric values 0 and 1.48575 makes a decrease in the reproduction number, R_0, but the reproduction number is greater than one that indicates the disease persists. If the parameter value of δ_1 greater than 1.48575, then the reproduction number decreases and becomes less than one where the disease dies out.

Rate of progress of aware infective to AIDS δ_2

Graphical representation of the basic reproduction number R_0versus rate of progress of aware infective to AIDS δ_2 and keeping other parameters constant

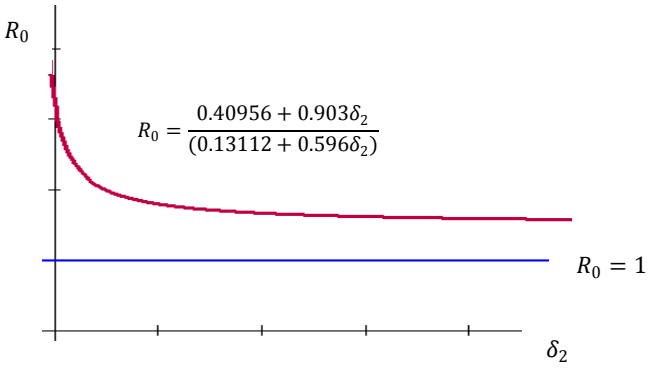

Figure 6: This figure shows an increase in the rate of progress of aware infective to AIDS, δ_2, makes a decrease in the reproduction number, R_0, but the reproduction number is greater than one that indicates the disease still persists.

Rate of unaware infective immigrants p_1

Graphical representation of the basic reproduction number R_0versus rate of unaware infective immigrants p_1 and keeping other parameters constant

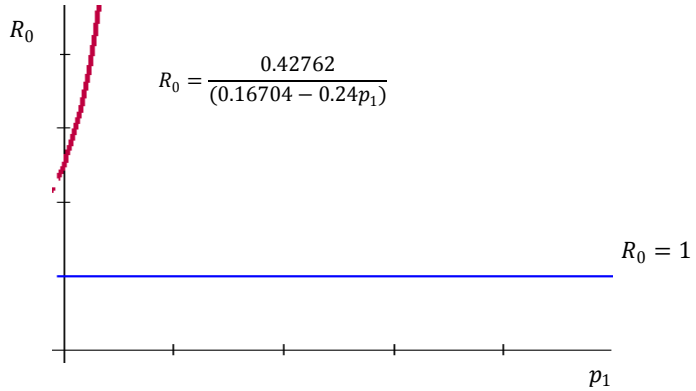

Figure 7: This figure shows an increase in the rate of unaware infective ımmıgrants, p_1, between 0 and 0.696, makes an increase in the reproduction number, $R_0 > 1$ and tell us the disease persists

Rate of aware infective immigrants p_2

Graphical representation of the basic reproduction number R_0versus rate of aware infective immigrants p_2 and keeping other parameters constant

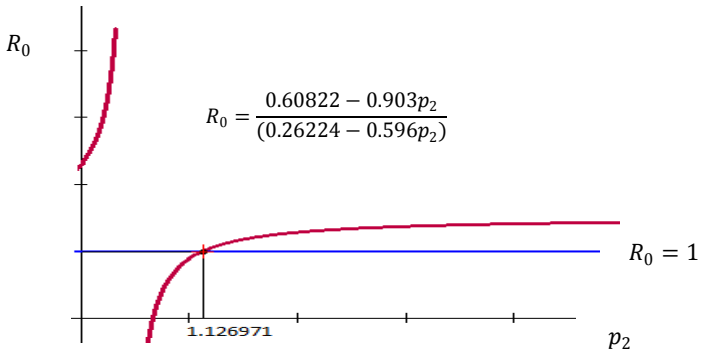

Figure 8: This figure shows an increase in the rate of aware infective immigrants, p_2, between 0 and 0.44, makes an increase in the reproduction number, $R_0 > 1$ and tell us the disease persists. If the rate of aware

infective immigrants between 0.44 and 1.126971 makes an increase in the reproduction number with, $R_0 < 1$ and tell us the disease not persists. Whereas, the rate of aware infective immigrants greater than 1.126971, makes an increase in the reproduction number, $R_0 > 1$, and tell us the disease persists.

Rate of transmission of unaware infective to seropositive class k_1

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

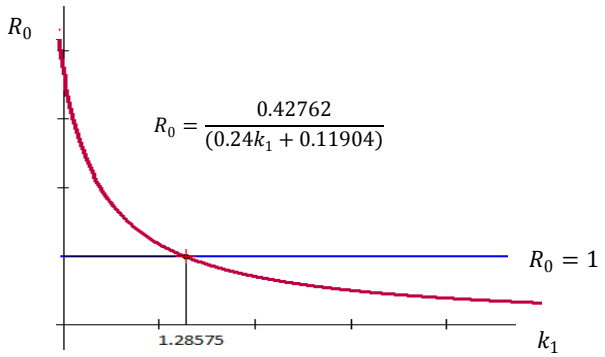

Figure 9: This figure shows an increase in the rate of transmission of unaware infective to seropositive class, k_1, between 0 and 1.28575, makes a decrease in the reproduction number, with $R_0 > 1$ and tell us the disease persists. If the rate of transmission of unaware infective to seropositive class greater than 1.28575 makes a decrease in the reproduction number with, $R_0 < 1$ and tell us the disease dies out.

Rate of transmission of aware infective to seropositive class k_2

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

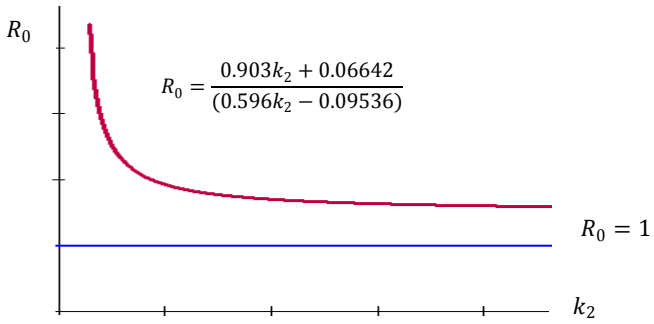

Figure 10: This figure shows an increase in the rate of transmission of aware infective to seropositive class, k_2, makes a decrease in the reproduction number, with $R_0 > 1$ and tell us the disease still persists.

Rate of transmission of unaware infective to aware infective θ

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

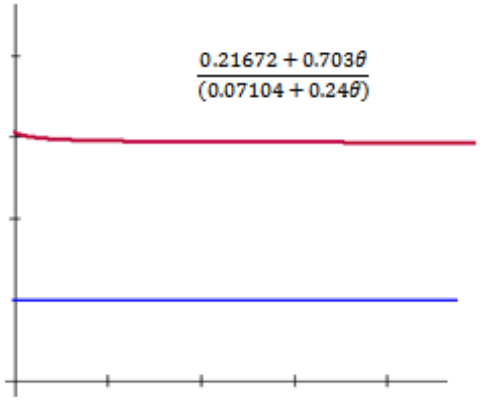

Figure 11: This figure shows an increase in the rate of transmission of unaware infective to aware infective, θ, then the reproduction number almost constant, with $R_0 > 1$ and tell us the disease still persists with constant reproduction number(i.e approximately 2.9292).

Rate of vertical transmission ϕ

Graphical representation of the basic reproduction number R_0versus rate of vertical transmission ϕ and keeping other parameters constant

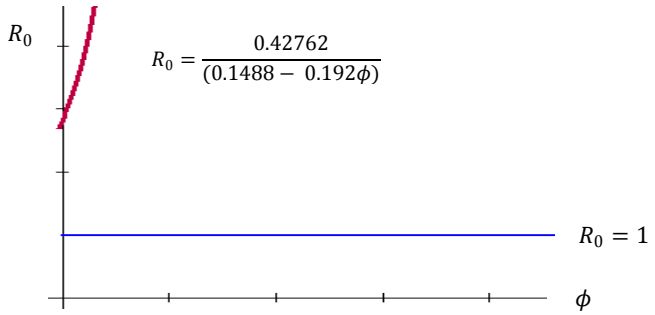

Figure 12: This figure shows an increase in the rate of vertical transmission, ϕ, between 0 and 0.775 then the reproduction number also increases, with $R_0 > 1$ and tell us the disease persists.

Natural death rate μ

Graphical representation of the basic reproduction number R_0versus natural death rate $\mu$and keeping other parameters constant

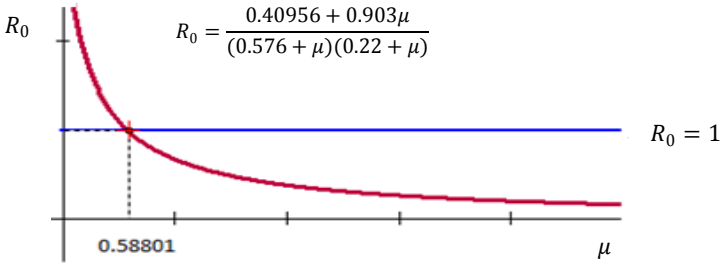

Figure 13: This figure shows an increase in the natural death rate, μ, between 0 and 0.58801 then the reproduction number decreases, with $R_0 > 1$ and tell us the disease still persists. If the natural death rate is greater than 0.58801, then the reproduction number is decreases, with $R_0 < 1$ and this tell us the disease dies out.

V. 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:

Parameter	Sensitivity Index
β_1	0.50512137
δ_1	-0.50335570
β_2	0.49109022
p_2	0.41099574
k_2	-0.280622983
p_1	0.16778523
k_1	-0.16778523
θ	0.09332127
μ	-0.07465662
δ_2	-0.04109957
ϕ	0.04026846
σ	0.00378841

Table 2: Sensitive indices

VI. Results and Discussion

Results from Numerical simulation show that as the probability of transmission of the disease from unaware infective and aware infective increases, the basic reproduction number increases. This will result in increasing on the transmission of HIV/AIDS.We can also observe that an increase in the rate of bloodborne transmission,σ, makes an increase in the reproduction number, R_0. That is the disease always persists for any value of parameterσ. Moreover an increase in the rate of progress of unaware infective to AIDS, δ_1, between the parametric values 0 and 1.48575 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 1.48575, then the reproduction number decreases and becomes less than one where the disease dies out.

An increase in the rate of progress of aware infective to AIDS, δ_2, makes a decrease in the reproduction number, R_0, but the reproduction number is greater than one that indicates the disease still persists. We also observed that an increase in the rate of unaware infective immigrants, p_1, between 0 and 0.696, makes an increase in the reproduction number, $R_0 > 1$ and tell us the disease persists. In addition to an increase in the rate of aware infective immigrants, p_2, between 0 and 0.44, makes an increase in the reproduction number, $R_0 > 1$ and tell us the disease persists. If the rate of aware infective immigrants between 0.44 and 1.126971 makes an increase in the reproduction number with, $R_0 < 1$ and tell us the disease dies out. Whereas, the rate of aware infective immigrants greater than 1.126971, makes an increase in the reproduction number, $R_0 > 1$, and tell us the disease persists.

We can also observed that an increase in the rate of transmission of unaware infective to seropositive class, k_1, between 0 and 1.28575, makes a decrease in the reproduction number, with $R_0 > 1$ and tell us the disease persists. If the rate of transmission of unaware infective to seropositive class greater than 1.28575 makes a decrease in the reproduction number with, $R_0 < 1$ and tell us the disease dies out.An increase in the rate of transmission of aware infective to seropositive class, k_2, makes a decrease in the reproduction number, with $R_0 > 1$ and tell us the disease still persists,we also observed that an increase in the rate of transmission of unaware infective to aware infective, θ, then the reproduction number almost constant, with $R_0 > 1$ and tell us the disease still persists with constant reproduction number(i.e approximately 2.9292). Whereas an increase in the rate of vertical transmission, ϕ, between 0 and 0.775 then the reproduction number also increases, with $R_0 > 1$ and tell us the disease persists. Moreover we can also observed that an increase in the natural death rate, μ, between 0 and 0.58801 then the reproduction number decreases, with $R_0 > 1$ and tell us the disease still persists. If the natural death rate is greater than 0.58801, then the reproduction number is decreases, with $R_0 < 1$ and this tell us the disease dies out.

From sensitive analysis we observed that the most sensitive parameter is the probability of the disease transmits to susceptible people by unaware infective humans, β_1 and the least sensitive parameter is the rate of transmission through bloodborne, σ. The indices having positive signs increase the value of R_0as one increase them and those having negative signs decrease the value of R_0, when they are increased.

VII. Conclusion

In this paper, we proposed an improvement of the model [25] that is to show the effect of unaware infective immigrants, aware infective immigrants, vertical and bloodborne transmission and treatment on the dynamics of HIV/AIDS. A non-linear differential equation was formulated to represent the model. The stability

analysis on the model shows that the disease frees equilibrium point E_0 is shown 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 $Z < Y$. Results from Numerical simulation show that as the probability of transmission of the disease to susceptible individuals by unaware and aware infective individuals increases, the basic reproduction number also increases. This will result in increasing on the transmission of HIV/AIDS. A sensitivity analysis of the basic reproduction number indicates that transmission probability, the rate of progress to AIDS and the rate of aware infective immigrants' are the most sensitive parameters that can be used to control the spread of the disease.

VIII. Recommendation

From the above results and discussion we would like to recommend the following to control the spread of HIV/AIDS: keep the rate of progress of unaware infective to AIDS, δ_1, greater than 1.48575, where the reproduction number is less than one, keep the rate of aware infective immigrants between 0.44 and 1.126971, keep the rate of transmission of unaware infective to seropositive class greater than 1.28575,keep the natural death rate greater than 0.58801 and The most sensitive parameters like transmission probability, the rate of progress to AIDS and the rate of aware infective immigrants', these parameters are those that should be targeted most by policymakers in the fight against the disease,.

References

[1]. AbdulsamadEngidaSado, PurnachandraRaoKoya. Application of Brody Growth Function to Describe Dynamics of Breast Cancer Cells. American Journal of Applied Mathematics(AJAM).Vol.3,No.3,2015,pp. 138-145. Doi:10.11648/j.ajam.20150303.20.

[2]. AIDSMAP, "World Life Expectancy Chart", 2005.

[3]. Anderson R. M. and May R. M., (1991). Infectious Diseases of Human, *Dynamics and Control.* Oxford: Oxford university press.

[4]. Basavarajaiah D. B., K. Mahessahppa (2012). *Mathematical Models Approach to HIV/AIDS transmission from mother to child.* J. Science & tech.

[5]. DanchoDesaleng, PurnachandraRaoKoya. The Role of Polluted Air and Population Density in the Spread of Mycobacterium Tuberculosis Disease. Journal of Multidisciplinary Engineering Science and Technology (JMEST). Vol. 2, Issue 5, May 2015,pp1212-20.ISSN:3159-0040. http://www.jmest.org/wpcontent/uplods/JMESTN42350782.pdf.

[6]. DanchoDesaleng, PurnachandraRaoKoya. The Role of Polluted Air and Population Density in the Spread of Mycobacterium Tuberculosis Disease. Journal of Multidisciplinary Engineering Science and Technology (JMEST). Vol. 2, Issue 5, May-2015,Pp1212-20.ISSN:3159-0040. http://www.jmest.org/wpcontent/uploads/JMESTN42350782.pdf.

[7]. DejenKetemaMamo, PurnachandraRaoKoya. Mathematical Modeling and Simulation Study of *SEIR* disease and Data Fitting of Ebola Epidemic in West Africa. Journal of Multidisciplinary Engineering Science and Technology (JMEST). Vol. 2,Issue1,January2015,pp106-14.ISSN:3159-0040.http://www.jmest.org/wpcontent/uploads/JMESTN42350340.pdf.

[8]. 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.

[9]. FekaduTadege Kobe, PurnachandraRaoKoya. Controlling the Spread of Malaria Disease Using Intervention Strategies. Journal of Multidisciplinary Engineering Science and Technology (JMEST). Vol. 2, Issue 5, May 2015, pp 1068 –74. ISSN: 3159 – 0040. http://www.jmest.org/wpcontent/uploads/JMESTN42350745.pdf.

[10]. GeXiaoqing and Arcak M. A sufficient condition for additive D stability and application to reaction-diffusion models, Systems and Control Letters 58 (2009) Ñ736-74.

[11]. H. Velasco and Y. Hsieh, Modeling the effect of treatment and behavior change in HIV transmission dynamics, J. Math. Biol.,32. 1994.

[12]. H. Ying - Yen and K. Cooke, Behaviour change and treatment of core groups: it's effects on the spread of HIV/AIDS. IMA J. Math. App. Med.Biol.17, 2000.

[13]. https://www.who.int/bulletin/archives/77(10)801.pdf /April 2019.

[14]. IssaShabani and S. Massawe, Modeling the effect of screening on the spread of HIV infection in a population with variable inflow of infective immigrants. Scientific research and essays, Vol. 20, 4397-4405, 2011.

[15]. K.O. Okosun, O.D. Makinde, I. Takaidza , Impact of optimal control on the treatment of HIV/AIDS and screening of unaware infectives , Applied Mathematical Modelling 37 (2013) 3802–3820 .

[16]. NakulChitnis, James M. Hyman, Jim M. Cushing,Determining Important Parameters in the Spread of Malaria through the Sensitivity Analysis of a Mathematical Model.Bulletin of Mathematical Biology (2008) 70: 1272–1296.

[17]. NavjotKaur , Mini Ghosh , S. Bhatia , Modelling the role of awareness and screening of infectives in the transmission dynamics of HIV, ISSN 1 746-7233, England, UK World Journal of Modelling and Simulation Vol. 12 (2016) No. 2, pp. 97-111.

[18]. Onoja Matthew Akpa and B. A. Oyejola, Modeling the transmission dynamics of HIV/AIDS epidemics, an introduction and review, J. Infect DEvCtries V (10), 597-608.

[19]. Program for appropriate technology in health, "Preventing HIV/AIDS in low-resource settings", outlook, (19), 1, 2001.

[20]. R. Adelman, "Mother to child HIV transmission in Africa", Policy Fact, 2001.

[21]. R.M. Anderson and R.M. May, Infectious diseases of humans: Dynamics and control, Oxford University Press, Oxford U.K., 1991.

[22]. Ram Naresh, A. Tripathi, D. Sharama, Modelling and Analysis of the spread of AIDS epidemic with immigration of HIV infectives. Mathematical and computer Modelling V (49), 880-892, 2009.

[23]. S. Al-sheikh, F. Musali, M. Alsomali, Properties of Stability and Hope Bifurcation for an HIV/AIDS epidemic model with screening and time delay, Vol-11, 87-99, 2012.

[24]. S. Al-sheikh, F. Musali, M. Alsomali, Stability Analysis of an HIV/AIDS epidemic model with screening, Vol 6, 3252-3273,2011.

[25]. TadeleTesfaTegegne, PurnachandraRaoKoya, TemesgenTibebu Mekonnen. Modeling the Combined Effect of Vertical Transmission and Variable Inflow of Infective Immigrants on the Dynamics of HIV/AIDS. *American Journal of Applied Mathematics.* Vol. 4, No. 1, 2016, pp. 11-19. doi: 10.11648/j.ajam.20160401.12

[26]. UNAIDS/WHO, "Paediatric HIV infection and AIDS: Point of view", UNAIDS, Geneva, 2004.