SENSITIVITY ANALYSIS OF HIV/AIDS MODEL WITH VERTICAL TRANSMISSION, TREATMENT AND PROGRESSION RATE

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ABSTRACT: In this study a nonlinear mathematical model of HIV/AIDS with treatment, vertical transmission and progression rate was considered. The next generation matrix was used to evaluate the basic reproduction number (R0) and the comparison approach was used to examine the global stability. The disease – free and the endemic equilibrium of the model were determined. The sensitivity analysis was carried out to determine the parameter that has high impact on the spread of the disease and the maple software was used for numerical simulation of the model. The disease free and endemic equilibrium were obtained and there stabilities studied . The model showed that the disease free equilibrium is locally asymptotically stable by using Routh Hurwitz criteria but globally of the disease free equilibrium is not stable by comparism approach. The numerical simulation shows that by using treatment measures and by controlling the rate of vertical transmission with time, the spread of the disease can reduced significantly and that providing treatment at the pre-AIDs stage reduces the infection much faster than starting treatment after progression into AIDs.

Keywords: - HIV/AIDs, Treatment, Vertical Transmission, Progression Rate, Sensitivity analysis

1. Introduction

The origin of Human Immunodeficiency Virus (HIV) and the mode through which it was introduced to humans is largely accepted to have occurred through humans' interaction with chimpanzees, who suffered from an older form of the disease [10]. The Human Immunodeficiency Virus (HIV) infection which leads to Acquired Immunodeficiency Syndrome (AIDS) has become a deadly infectious disease in both developed and developing nations. It is deadly because it usually breaks down the body immune system [23], leaving the victim vulnerable to a lot of other diseases. It has caused demise of millions of people and has increased money spent on health care and disease control [38].

HIV can be transfer by Transfusion with blood products, HIV-infected mother can transmit HIV to her infant during pregnancy, delivery or while breastfeeding which is called **Vertical transmission**, People can also become infected with HIV when using injection through sharing of needles and other equipment, Sexual intercourse with HIV infected individual and lots more [22].

Treatment is the process of offering the HIV positive individual with a life prolonging drug/medicine known as antiretroviral (ART) or Highly Active Antiretroviral **(HAART) t**herapy [11]. It is not a cure but it can prolong the life of a person for many years. It consists of drugs that have to be taken every day for the rest of the person's life. It keeps the amount of HIV in the body at low level [7,26,35].

According to the past works on epidemics, particularly HIV/AIDs , the researcher has come up with different mathematical modeling of HIV/AIDs dynamics with treatment and vertical transmission. Of interest in the researcher is to analyze the progression rate of individual affected with HIV from one compartment [1] to the other and the impact of each parameter on the model. The aim of the study is to investigate HIV/ AIDS dynamics with treatment, vertical transmission and progression rate.

The model of HIV/AIDS dynamics with treatment, vertical transmission and progression rate of was formulated, qualitative analysis of the model was done in order to determine the possibility of existence and stability of endemic and disease free equilibriums. The Comparison theorem [***1,7,19]*** was used to determine the global stability of the model. The basic reproduction number ***(Ro)*** which is cardinal parameter governing the spread of disease was computed using the next generation operation approach. The model will be validated by secondary data from other literature and the computer software: Maple was used in Numerical simulation.

2. Model Formulation

A non linear mathematical model is proposed and analyzed to study of HIV/AIDS with treatment and vertical transmission and progression rate. In modeling the population of size N(t) at time (t) with constant inflow of susceptible with rate πN where π is the rate of recruitment into susceptible population is divided into five groups: Susceptible S(t) , infective I(t) (also assumed to be infectious), pre-AIDS patients P(t) , treated class T(t) and AIDS patients A(t) with natural mortality rate μ in all classes [1,9,23].

The interaction between the classes will be assumed as follows: the susceptible become HIV infected via sexual contacts with infective which may also lead to the birth of infected children. A fraction of new born children are infected during birth and hence are directly recruited into the infective class with a rate $(1-\varepsilon)\theta$ and others die effectively at birth $(0 \leq 1 \leq \varepsilon)$ where ε is the fraction of newborns infected with HIV who dies immediately after birth and θ is the rate of newborns infected with HIV. We do not consider direct recruitment of the infected persons but by vertical transmission only.

It is also assumed that some of the infective join the pre-AIDS class, depending on the viral counts, with a rate $\sigma_1 \delta$ where δ is the rate of movement from infectious class and σ_1 is the fraction of δ joining the pre-AIDS class [1, 9, 23]. They then proceed with a rate γ to develop full blown AIDS. Some of the infective proceed to join the treated class with a rate $\sigma_2 \delta$ where σ_2 is the fraction of δ joining treated class and then proceed with a rate k to develop full blown AIDS while others with serious infection directly join the AIDS class with a rate $(1-\sigma_1 - \sigma_2)\delta$ (8,28).

The infective through vertical transmission at any time t is given by $\gamma \varepsilon I(t-\tau)$ because those infected at time $(t-\tau)$ becomes infectious at time τ later, if they do not develop AIDS by that time. The fraction of infective which develops AIDS during the period of getting sexual maturity, if they survive the maturity period joins the AIDS class [6, 23].

Thus, in our model the term $\gamma \varepsilon I(t-\tau)\ell^{-d\tau}$ represents the introduction of infective persons who survive the maturity period τ in which the time taken to become infectious is τ [5, 6, 29]. Here $\ell^{-d\tau}$ represents the probability that an individual survives the maturity period $[t-\tau, t]$ such that $0 < \ell^{-d\tau} \leq 1$. It is also assumed that all newborns are infected at birth ($\tau = 0$). As our purpose is to study the rate of movement from infective Population to AIDs Population and to determine what can be done to reduce it [2, 4, 41].

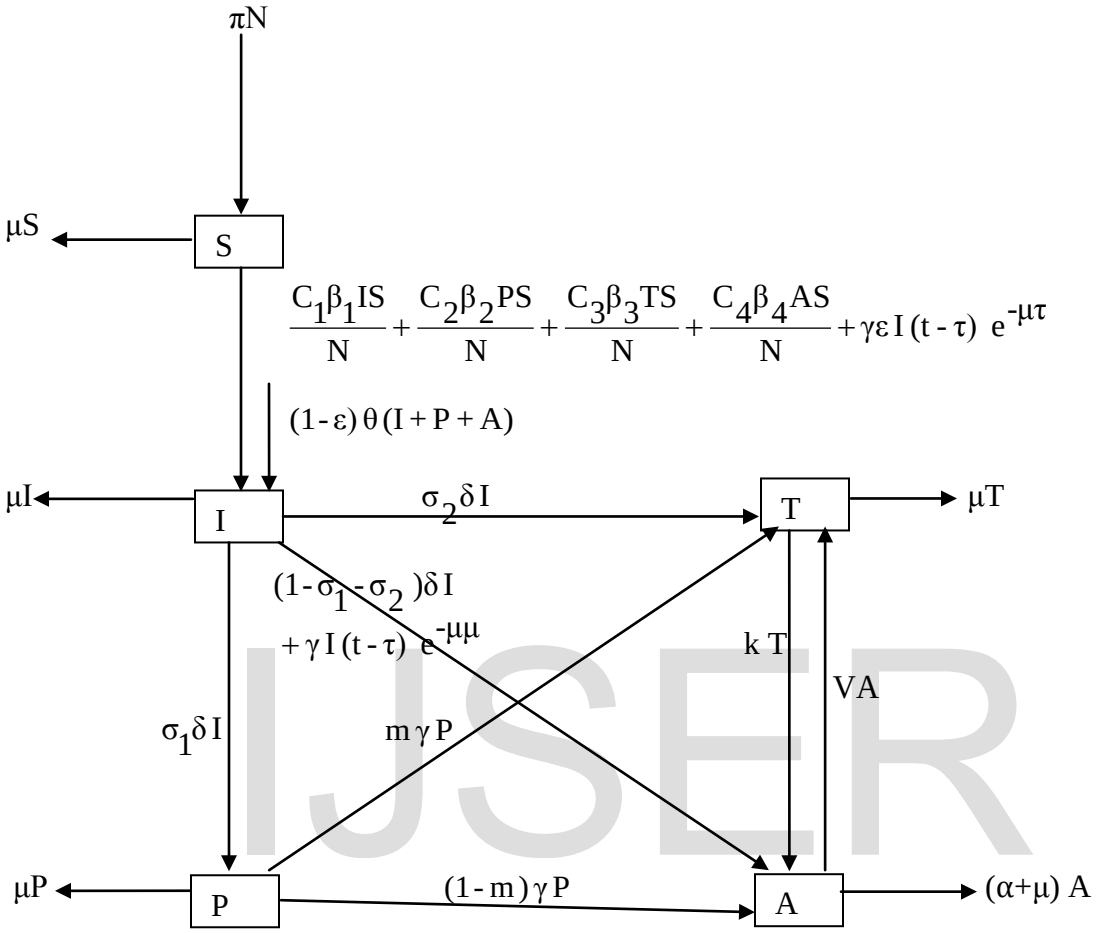

Figure 1. The flow Chart of the model.

With the above considerations and assumptions, the spread of the disease is assumed to be governed by the following system of nonlinear ordinary differential equations:

$$\frac{dS}{dt} = \pi N - \frac{C_1\beta_1 IS}{N} - \frac{C_2\beta_2 PS}{N} - \frac{C_3\beta_3 TS}{N} - \frac{C_4\beta_4 AS}{N} - \gamma\varepsilon I(t-\tau)\ e^{-\mu\tau} - \mu S$$

$$\frac{dI}{dt} = \frac{C_1\beta_1 IS}{N} + \frac{C_2\beta_2 PS}{N} + \frac{C_3\beta_3 TS}{N} + \frac{C_4\beta_4 AS}{N} - (\delta+\mu)\ I - \gamma(1-\varepsilon)I(t-\tau)\,e^{-\mu\tau} + (1-\varepsilon)\theta(I+P+A)$$

$$\frac{dP}{dt} = \sigma_1\delta I - (\gamma+\mu)P \qquad (1)$$

$$\frac{dT}{dt} = \sigma_2\delta I + m\gamma P + VA - (K+\mu)T$$

$$\frac{dA}{dt} = (1-\sigma_1-\sigma_2)\delta I + \gamma I(t-\tau)\ e^{-\mu\tau} + (1-m)\gamma P + K T - (V+\alpha+\mu)A$$

Where

C= Average number of sexual partners per unit time

β =Sexual contract rates

δ = Rate of movement from infectious class

V =Rate of which Aids Patients get treatment

K =Rate at which treated population become full blown Aids

α =Disease induced through vertical transmission at any time

The initial conditions are taken as: $S(0) = S_0$, $I(0) = I_0$, $P(0) = P_0$, $A(0) = A_0$, $T(0) = T_0$,

To simplify the model (1), we assume that the AIDS patients and those in pre-Aids class are isolated and sexually inactive and hence they are not capable of producing children; at $\tau = 0$ and $(1-\varepsilon)\theta P = (1-\varepsilon)\theta A = 0$ and also they do not contribute to vital transmission horizontally, that is β_2 and β_4 are negligible.

In view of the above assumptions, the system (1) reduces to:-

$$\frac{dS}{dt} = \pi N - \frac{C_1\beta_1 IS}{N} - \frac{C_3\beta_3 TS}{N} - \gamma\varepsilon I - \mu S$$

$$\frac{dI}{dt} = \frac{C_1\beta_1 IS}{N} + \frac{C_3\beta_3 TS}{N} - (\delta+\mu)I - \gamma(1-\varepsilon)I + (1-\varepsilon)\theta I \qquad (2)$$

$$\frac{dP}{dt} = \sigma_1\delta I - (\gamma+\mu)P$$

$$\frac{dT}{dt} = \sigma_2\delta I + m\gamma P + VA - (K+\mu)T$$

$$\frac{dA}{dt} = (1-\sigma_1-\sigma_2)\delta I + \gamma I + (1-m)\gamma P + KT - (V+\alpha+\mu)A$$

Total population N at anytime (t) is given by

$N(t) = S(t) + I(t) + P(t) + T(t) + A(t)$

This gives:

$$\frac{dN}{dt} = (\pi-\mu)N + (1-\varepsilon)\theta I - \alpha A \qquad [3]$$

From equation (3), if the disease, pre – AIDS and infective is removed, the total population size N is stationary for μ, and declining for $\pi < \mu$ and grows exponentially for $\pi > \mu$. Mortality rate (μ) is assumed to be a function of state variable [6]. Since the model is homogenous of degree one, the variable can be normalized by setting.

$$s = \frac{S}{N}, i = \frac{I}{N}, p = \frac{P}{N}, h = \frac{T}{N}, a = \frac{A}{N} \qquad (4)$$

That leads to the normalized system

$$\frac{ds}{dt} = \pi - C_1\beta_1 is - C_3\beta_3 hs - \gamma\varepsilon i - [\pi + (1-\varepsilon)\theta i - \alpha a]s$$

$$\frac{di}{dt} = C_1\beta_1 is + C_3\beta_3 hs - \gamma(1-\varepsilon)i + (1-\varepsilon)\theta i - [\pi+\delta+(1-\varepsilon)\theta i - \alpha a]i$$

$$\frac{dp}{dt} = \sigma_1\delta i - [\pi+\gamma+(1-\varepsilon)\theta i - \alpha a]p \qquad (5)$$

$$\frac{dh}{dt} = \sigma_2\delta i + m\gamma p + Va - [\pi + k + (1-\varepsilon)\theta i - \alpha a]h$$

$$\frac{da}{dt} = (1-\sigma_1-\sigma_2)\delta i + \gamma i + (1-m)\gamma p + k h - [\pi + V + \alpha + (1-\varepsilon)\theta i - \alpha a]a$$

where

$s + i + p + h + a = 1$ and $s(t) > 0; i(t) \geq 0; p(t) \geq 0; h(t) \geq 0; a(t) \geq 0; \forall t \geq 0$

Continuity of right-hand side of the system (3) and its derivative imply that the model is well posed for N > 0.

3. MODEL ANALYSIS

The qualitative analysis of a nonlinear system (5) was carried out to find the conditions for existence and stability of disease free equilibrium points [13]. Analysis of the model allows us to determine the impact of treatment and vertical transmission on the transmission of HIV/AIDS infection in a population. Also on finding the reproductive number R_0, one can determine if the disease become endemic in a population or not.

3.1 Existence and uniqueness of solution

The existence and uniqueness of solution of system (5) was carried out by using Derrick and Grossman 1976 and it was proved that equation (5) has a unique solution in D.

3.2 Positivity of Solution

For the model (5) to be epidemiological meaningful and well posed. We need to prove that all state variables are non-negative, $\forall t \geq 0$.

Theorem 1

Let $\Omega = [(s, i, p, h, a) \in \Re^5 : s + i + p + h + a = 1]$, then the solution $\{s(t), i(t), p(t), h(t), a(t)\}$ of the system [5] are positive $\forall t \geq 0$.

To prove the theorem, the differential equation of the system [5] will be used.

Using the first equation of system (5), and $s(0) > 0, i(0) \geq 0, p(0) \geq 0, h(0) \geq 0, a(0) \geq 0$

Then,

$$\frac{ds}{dt} \leq \pi - \pi s \qquad (6)$$

$$s(t) \leq 1 + A e^{-\pi t}$$

Applying initial condition, when t = 0, s (t) = s (0)

$$s(0) \leq 1 + A$$

$$At, \ t \to \infty, \ s(t) \leq 1,$$

$$\text{Therefore, } 0 \leq s(t) \leq 1.$$

Using similar approach on the equation in (5), it gives us:

$$i(t) \geq 0, p(t) \geq 0, h(t) \geq 0, a(t) \geq 0$$

Hence, all state variables are non-negatives, then it is epidemiological meaningful and well posed.

3.3 Disease Free Equilibrium

When there is mo disease in the population is called disease free equilibrium (DFE), and then obtained by setting, system (5) to be zero.

$$\frac{ds}{dt}=\frac{di}{dt}=\frac{dp}{dt}=\frac{dh}{dt}=\frac{da}{dt}=0 \qquad (7)$$

For the DFE point $i=p=h=a=0$, when substitute into equation (5),

We have

$\pi-\pi s=0$

$s=\frac{\pi}{\pi}=1$

Therefore, the DFE $E_0 \, is\,(1,0,0,0,0)$ (8)

3.4 Computation of the Basic Reproduction Number (R_o)

The basic reproduction number R_0 is defined as the effective number of secondary infectious caused by typical infected individual during his interred periods of infectiousness [1, 7,38]. To computation of the basic reproduction number the next generation method was applied on system (5), R_0 is essential. This definition is given for the models that represent the spreading of infection in a population [1]. It was obtained by taking the largest (dominant) eigenvalue (spectral radius) of :

$$\left[\frac{\partial f_i(E_0)}{\partial x_j}\right]\left[\frac{\partial v_i(E_0)}{\partial x_j}\right]^{-1} \qquad (9)$$

Where

f_i = rate of appearance of new infection in compartment i.

$v^{+}{}_i$ = the transfer of individual out of the compartment i

E_0 = the disease free equilibrium.

By linearization approach, the associated matrix at disease free equilibrium is obtained as

$$F=\begin{bmatrix} C_1\beta_1 & 0 & C_3\beta_3 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

and

$$V=\begin{bmatrix} \pi+\delta-\gamma(1-\varepsilon)-(1-\varepsilon)\theta & 0 & 0 & 0 \\ -\sigma_1\delta & \pi+\gamma & 0 & 0 \\ -\sigma_2\delta & -m\gamma & \pi+k & -v \\ -\gamma-(1-\sigma_1\sigma_2)\delta & -(1-m)\gamma & -k & \pi+v+\alpha \end{bmatrix}$$

It can be shown that the Eigen values of FV^{-1} are (0, 0, 0, Z).

Where

$$Z=\frac{C_1\beta_1}{\pi+\delta-\gamma(1-\varepsilon)-(1-\varepsilon)\theta}+\frac{\begin{array}{l}C_3\beta_3(\gamma\sigma_1\delta m\pi+\gamma\sigma_1\delta m\alpha+\sigma_2\delta\pi^2+v\gamma\pi+v\delta\pi+v\gamma\delta+\delta\sigma_2\pi\alpha \\ +\delta\sigma_2\pi\gamma+\delta\sigma_2\gamma\alpha+v\gamma^2-v\sigma_1\delta\pi)\end{array}}{(\pi+\delta-\gamma(1-\varepsilon)-(1-\varepsilon)\theta)(\pi+\gamma)(\pi^2+\pi v+\pi\alpha+\pi k+\alpha k)} \qquad (10)$$

It follows that the basic reproduction number R_o for the normalized model system (5) with treatment and vertical transmission is given by

$$R_o = \frac{C_1\beta_1}{\pi+\delta-\gamma(1-\varepsilon)-(1-\varepsilon)\theta} + \frac{C_3\beta_3(\gamma\sigma_1\delta m\pi+\gamma\sigma_1\delta m\alpha+\sigma_2\delta\pi^2+v\gamma\pi+v\delta\pi+v\gamma\delta+\delta\sigma_2\pi\alpha+\delta\sigma_2\pi\gamma+\delta\sigma_2\gamma\alpha+v\gamma^2-v\sigma_1\delta\pi)}{(\pi+\delta-\gamma(1-\varepsilon)-(1-\varepsilon)\theta)(\pi+\gamma)(\pi^2+\pi v+\pi\alpha+\pi k+\alpha k)} \quad (11)$$

3.5 Local Stability of DFE

Theorem:

The disease free equilibrium of the system is locally asymptotically stable if $R_0<1$ and unstable if $R_0>1$. Now to determine the local stability of E_0, variation matrix is computed corresponding to equilibrium point E_0.

$$J_0=\begin{bmatrix} -\pi & -C_1\beta_1-(1-\varepsilon)\theta-\gamma\varepsilon & 0 & -C_3\beta_3 & \alpha \\ 0 & C_1\beta_1-\gamma(1-\varepsilon)+(1-\varepsilon)\theta-(\pi+\delta) & 0 & C_3\beta_3 & 0 \\ 0 & \sigma_1\delta & -(\pi+\gamma) & 0 & 0 \\ 0 & \sigma_2\delta & m\gamma & -(\pi+\gamma) & v \\ 0 & (1-\sigma_1-\sigma_2)\delta+\gamma & (1-m)\gamma & k & -(v+\alpha+\pi) \end{bmatrix}$$

The Characteristic equation correspondent to J_0 is given by

$$f(x)=(\pi+\lambda)(\lambda^4+a_1\lambda^3+a_2\lambda^2+a_2\lambda^1+a_4)=0 \quad (12)$$

Where

$a_1=\alpha+\delta+\theta\varepsilon+v+4\pi+k+\gamma-\gamma\varepsilon-C_1\beta_1-\theta$

$a_2=\delta[\alpha+v+k-\sigma_2 C_3\beta_3+\gamma]+\theta\varepsilon[3\pi-\gamma+k+v+\alpha]+\theta[\gamma-k-v-\alpha]+C_1\beta_1[\gamma-\pi-k-v-\alpha]$
$+3\pi[2\pi-\theta+\alpha+\delta-\gamma+k+v]-\gamma[k+v-\alpha]-\gamma\varepsilon[\gamma-3\pi-k-v-\alpha]+k\alpha$

$a_3=[\delta+\theta+\theta\varepsilon-\gamma\varepsilon][\alpha k-v\gamma-\alpha\gamma-k\gamma+2v\pi-2\pi\gamma+2\alpha\pi+2k\pi+3\pi^2]-C_1\beta_1[\alpha k-v\gamma-\alpha\gamma-k\gamma$
$+2k\pi-2\pi v+2\pi v+2\pi\alpha+3\pi^2]-C_3\beta_3\delta[2\sigma_2\pi+v-\sigma_1 v-\sigma_2\gamma+\sigma_2\alpha+m\sigma_1\gamma]-C_3\beta_3\gamma v[1-\varepsilon]$
$-\gamma[2\pi k+2\pi v+2\pi\alpha+\alpha k+3\pi^2]+2k\alpha\pi+3\pi^2 v+4\pi^3+3\pi^2\alpha+3\pi^2 k$

$a_4=(\pi+\delta-\gamma(1-\varepsilon)-(1-\varepsilon)\theta)(\pi+\gamma)(\pi^2+\pi v+\pi\alpha+\pi k+\alpha k)-C_1\beta_1(\pi+\gamma)(\pi^2+\pi v+\pi\alpha$
$+\pi k+\alpha k)-C_3\beta_3\gamma v[\gamma\varepsilon+\pi-\gamma-\pi\varepsilon]+C_3\beta_3(\gamma\sigma_1\delta m\pi+\gamma\sigma_1\delta m\alpha+\sigma_2\delta\pi^2+v\gamma\pi$
$+v\delta\pi+v\gamma\delta+\delta\sigma_2\pi\alpha+\delta\sigma_2\pi\gamma+\delta\sigma_2\gamma\alpha+v\gamma^2-v\sigma_1\delta\pi)$

Thus by Routh – Hurwitz criteria, E_o is locally asymptoticly stable as it can be seen for

$$a_1 > 0, a_2 > 0, a_3 > 0, a_4 > 0, a_1 a_3 - a_3 > 0 \quad and \quad a_1 a_2 a_3 - a_3^2 - a_1^2 a_4 > 0 \tag{13}$$

Thus, using $a_4 > 0$

$$\frac{C_1 \beta_1}{\pi + \delta - \gamma(1-\varepsilon) - (1-\varepsilon)\theta} + \frac{\begin{matrix} C_3 \beta_3 (\gamma \sigma_1 \delta m \pi + \gamma \sigma_1 \delta m \alpha + \sigma_2 \delta \pi^2 + v \gamma \pi + v \delta \pi + v \gamma \delta + \delta \sigma_2 \pi \alpha \\ + \delta \sigma_2 \pi \gamma + \delta \sigma_2 \gamma \alpha + v \gamma^2 - v \sigma_1 \delta \pi) \end{matrix}}{(\pi + \delta - \gamma(1-\varepsilon) - (1-\varepsilon)\theta)(\pi + \gamma)(\pi^2 + \pi v + \pi \alpha + \pi k + \alpha k)} < 1 \tag{14}$$

Therefore, $R_o < 1$

The proofs of the theorem above, that is, the disease free equilibrium of the system is locally asymptotically stable if $R_o < 1$

3.6 Global Stability of the Disease Free Equilibrium

To compute global stability of the disease free equilibrium comparism theorem was employed that is: the rate of change of the infected, pre – AIDs, treated and AIDs classes of the model system (5) could be written as:

$$\begin{bmatrix} \frac{di}{dt} \\ \frac{dp}{dt} \\ \frac{dh}{dt} \\ \frac{da}{dt} \end{bmatrix} = (F - V) \begin{bmatrix} i \\ p \\ h \\ a \end{bmatrix} - F_i \begin{bmatrix} i \\ p \\ h \\ a \end{bmatrix} \tag{15}$$

At the disease free, this is $(s, i, p, h, a) \rightarrow (1, 0, 0, 0, 0)$

The characteristic equation of this matrix was carried out and gives us:

$g(\lambda) = \lambda^4 + F_1 \lambda^3 + F_2 \lambda^2 + F_3 \lambda + F_4 = 0$

where

$$F_1 = 4\pi + v + \alpha + k + \gamma - C_1\beta_1 + \delta - \gamma\varepsilon - \theta - \theta\varepsilon$$

$$\begin{aligned}F_2 = {} & \gamma\theta\varepsilon - \sigma_2\delta C_3\beta_3 - \gamma\theta + k\delta - k\theta + 3\pi\gamma + 3\pi\delta - 3\pi\theta + 3k\pi + k\gamma + \gamma\delta - \gamma^2\varepsilon + 6\pi^2 - kC_1\beta_1 \\ & + k\theta\varepsilon - 3\pi C_1\beta_1 - 3\pi\gamma\varepsilon + 3\pi\theta\varepsilon - k\gamma\varepsilon - \gamma C_1\beta_1 - vC_1\beta_1 + v\theta\varepsilon - \alpha C_1\beta_1 + \alpha\theta\varepsilon - v\gamma\varepsilon \\ & - \alpha\gamma\varepsilon + v\delta - v\theta + \alpha k + \alpha\delta - \alpha\theta + 3v\pi + v\gamma + 3\alpha\pi + \alpha\gamma\end{aligned}$$

$$\begin{aligned}F_3 = {} & C_3\beta_3 v\gamma\theta\varepsilon - m\gamma\sigma_1\delta C_3\beta_3 - 2\sigma_2\delta C_3\beta_3\pi - 2\pi\gamma C_1\beta_1 + 2\pi\gamma\theta\varepsilon - 2k\pi C_1\beta_1 - 2k\pi\gamma\varepsilon \\ & + 2k\pi\theta\varepsilon - k\gamma C_1\beta_1 + k\gamma\theta\varepsilon - C_3\beta_3 v\delta - \alpha k C_1\beta_1 + \alpha k\theta\varepsilon + v\gamma\theta\varepsilon - 2v\pi C_1\beta_1 - 2v\pi\gamma\varepsilon \\ & + 2v\pi\theta\varepsilon - v\gamma C_1\beta_1 + \alpha\gamma\theta\varepsilon - 2\alpha\pi C_1\beta_1 - 2\alpha\pi\gamma\varepsilon + 2\alpha\pi\theta\varepsilon - \alpha k\gamma\varepsilon - \alpha k C_1\beta_1 + 3\pi^2\delta \\ & - 3\pi^2\theta + 3\pi^2\gamma + 3\pi^2 k + 3\pi^2 v + 4\pi^3 - \sigma_2\delta C_3\beta_3\gamma + C_3\beta_3 v\sigma_1\delta - C_3\beta_3 v\gamma\theta - \alpha\sigma_2\delta C_3\beta_3 \\ & - 3\pi^2 C_1\beta_1 - 3\pi^2\gamma\varepsilon + 3\pi^2\theta\varepsilon + 2\pi\gamma\delta - 2\pi\gamma^2\varepsilon - 2\pi\gamma\theta + 2k\pi\delta - 2k\pi\theta + 2k\gamma\pi + k\gamma\delta \\ & - k\gamma^2\varepsilon - k\gamma\theta + \alpha k\delta - \alpha k\theta - v\gamma\theta + 2v\pi\gamma + 2v\pi\delta - 2v\pi\theta + v\gamma\delta - v\gamma^2\varepsilon - \alpha\gamma\theta + 2\alpha\pi\gamma \\ & + 2\alpha\pi\delta - 2\alpha\pi\theta + 2\alpha k\pi + \alpha k\gamma + \alpha\gamma\delta - \alpha\gamma^2\varepsilon + 3\alpha\pi^2\end{aligned}$$

$$\begin{aligned}F_4 = {} & \gamma^2\theta C_3\beta_3 v\varepsilon - \gamma\theta C_3\beta_3 v\pi + \sigma_1\delta C_3\beta_3 v\pi - \sigma_2\delta C_3\beta_3\gamma\pi - \sigma_2\delta C_3\beta_3\alpha\pi - \sigma_2\delta C_3\beta_3\alpha\gamma \\ & + \gamma\theta\varepsilon C_3\beta_3 v\pi - \sigma_1\delta C_3\beta_3 m\gamma\pi - \sigma_1\delta C_3\beta_3 m\gamma\alpha + \alpha k\gamma\pi + \alpha k\gamma\delta - \pi^2\gamma C_1\beta_1 + \pi^2\gamma\theta\varepsilon \\ & - \pi^2 k C_1\beta_1 - k\pi^2\gamma\varepsilon + k\pi^2\theta\varepsilon + k\pi\gamma\delta - k\pi\gamma^2\varepsilon - k\pi\gamma\theta - \pi^2 vC_1\beta_1 - v\pi^2\gamma\varepsilon + v\pi^2\theta\varepsilon \\ & + v\pi\gamma\delta - v\pi\gamma^2\varepsilon - v\pi\gamma\theta - \alpha\pi^2 C_1\beta_1 - \alpha\pi^2\gamma\varepsilon + \alpha\pi^2\theta\varepsilon + \alpha\pi\gamma\delta - \alpha\pi\gamma^2\varepsilon - \alpha\pi\gamma\theta \\ & + \alpha\pi k\delta - \alpha\pi k\theta - \delta C_3\beta_3 v\pi - C_3\beta_3 v\gamma^2\theta - \delta C_3\beta_3 v\gamma - \delta C_3\beta_3\sigma_2\pi^2 - \pi k\gamma C_1\beta_1 \\ & + \pi k\gamma\theta\varepsilon - v\pi\gamma C_1\beta_1 + v\pi\gamma\theta\varepsilon - \alpha\pi\gamma C_1\beta_1 + \alpha\pi\gamma\theta\varepsilon - \alpha k\pi C_1\beta_1 - \alpha k\pi\gamma\varepsilon + \alpha k\pi\theta\varepsilon \\ & - \alpha k\gamma C_1\beta_1 + \alpha k\gamma\theta\varepsilon - \pi^3 C_1\beta_1 - \pi^3\gamma\varepsilon + \pi^3\theta\varepsilon + \pi^2\gamma\delta - \pi^2\gamma^2\varepsilon - \pi^2\gamma\theta + \pi^3\delta - \pi^3\theta \\ & + \pi^3\gamma + \pi^3 k + \pi^3 v + \pi^3\alpha + \pi^4 - \alpha k\gamma^2\varepsilon - \alpha k\gamma\theta + k\pi^2\delta - k\pi^2\theta + k\pi^2\gamma + v\pi^2\delta - v\pi^2\theta \\ & + v\pi^2\gamma + \alpha\pi^2\delta - \alpha\pi^2\theta + \alpha\pi^2\gamma + \alpha k\pi^2\end{aligned}$$

$$F_1 > 0,\ F_2 > 0,\ F_3 > 0 \ \text{and}\ F_4 > 0$$

Hence all eigen- values are negatives which implies that the endemic equilibrium point is globally asymptotically stable.

3.7 Sensitivity Analysis

Computing numerical sensitivity indices was done in order to enable us to single out parameters that have a high impact on R_0 and which should be targeted by intervention strategies. The normalized forward sensitivity index of a variable to a parameter is a ratio of the relative change in the variable to the relative change in the parameter. When a variable is a differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivatives.

$$r^{Rm}{}_q = \frac{\partial R_m}{\partial q}\, x\, \frac{q}{R_m} \qquad (16)$$

Table 3.1 Table of Sensitivity analysis of the model

Number	Parameter and its meanings	value	Sensitivity value
1	C_1 Average number of sexual infective partners per unit time	**1**	**0.8048134033**
2	C_3 Average number of sexual treated partners per unit time	**3**	**0.1951865967**
3	β_1 Sexual contract rates of infective partners	**0.4**	**0.8048134033**
4	β_3 Sexual contract rates of treated partners	**0.05**	**0.1951865967**
5	π Rate of recruitment into susceptible population	**0.1**	**-0.4879649558**
6	δ Rate of movement from infectious class	**0.6**	**-1.979011836**
7	γ Rate of movement of pre – Aids population individual into Aids Population	**0.9**	**0.6826605824**
8	ε Fraction of newborns infected with HIV who dies immediately after birth	**0.2**	**0.4207361057**
9	θ Rate of newborns infected with HIV	**0.3**	**0.8884841479**
10	σ_1 Fraction of the rate of movement from Pre – AIDS Class joining the AIDs Class	**0.2**	**0.06720965772**
11	σ_2 Fraction of the rate of movement from treated class joining the AIDs Class	**0.01**	**0.009576505641**
12	α Disease induced through vertical transmission at any time	**1**	**-0.09752298139**
13	ν Rate of which Aids Patients get treatment	**0.1**	**0.1072752795**
14	**m** Fraction of γ who get treatment	**0.4**	**0.06895084060**
15	κ Rate at which treated population become full blown Aids	**0.08**	**-0.08257894476**

From the sensitivity analysis, it was discovered that the following parameters will have high impact on the transmission of the diseases: they are Sexual Contract rate of infective partners (β1) and vertical transmission (□).

4. Numerical Simulations of the Model

To study the dynamical behavior of the model (5) numerically, Runge-Kutta method of order four (4)applying and the following parameters values were used: θ= 0.3, ε= 0.2,ν = 0.1,β 1= 0.4,β 3 = 0.05,σ 1= 0.2, σ2= 0.01, k = 0.08, γ= 0.9, 1, m = 0.4, π = 0.4, δ= 0.6, α=1, C1 = 3, C3 = 1 [1]
With initial values $s(0)$=0.5, $i(0)$ = 0.3, $p(0)$ = 0.12, $h(0)$ = 0.07 , $a(0)$ = 0.01.

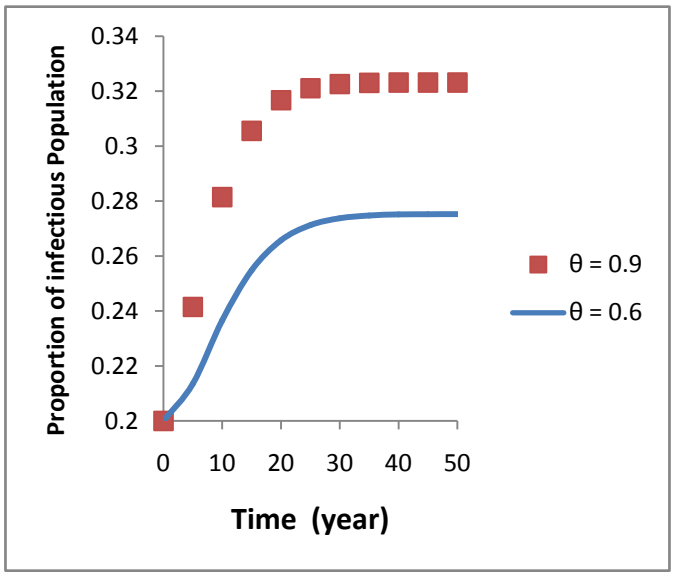

Figure 1a. Graph of Infectious class against time for different values of rate of newborns infected with HIV (θ)

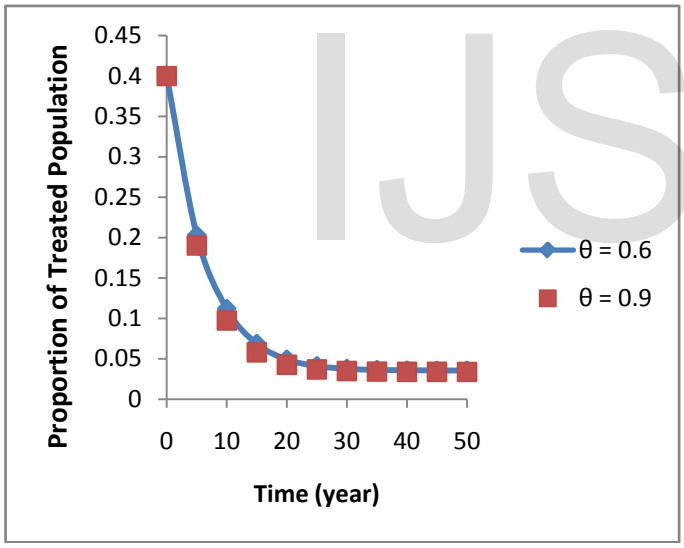

Figure 1b. Graph of Treated class against time for different values of rate of newborns infected with HIV (θ).

Figure 1a, It was discovered that as fraction of new born children increase the infectious population increase and the Pre – Aids population decrease with time in the presence of ARVs.

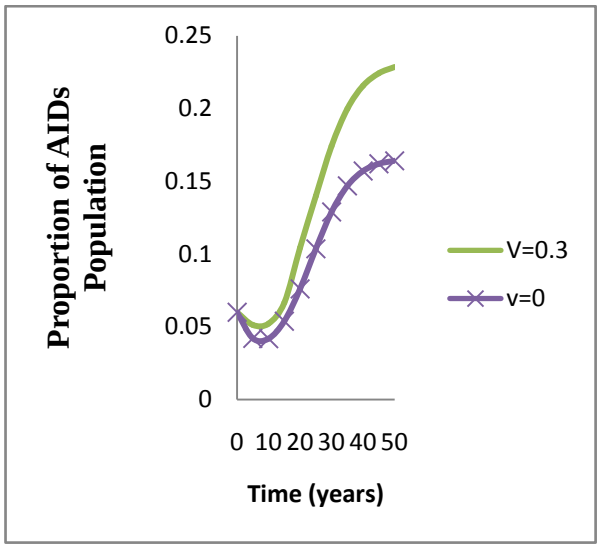

Figure 2a. Graph of AIDs class against time for different values of Rate of which Aids Patients get treatment (υ)

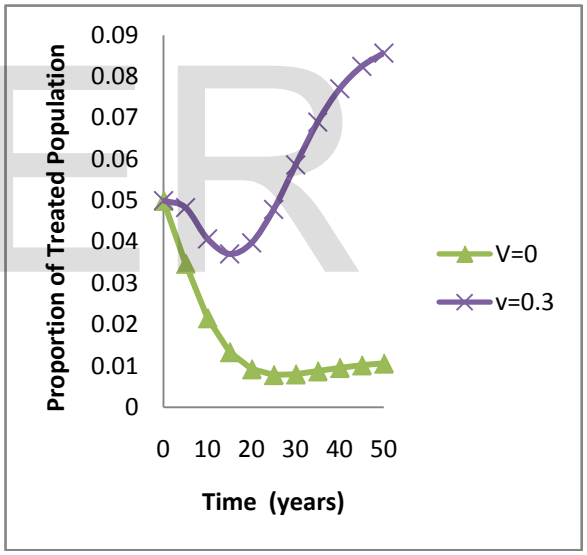

Figure 2b. Graph of Treatment class against time for different values of Rate of which Aids Patients get treatment (υ)

Figure 2a-b. Shows that if treatment rates is increasing the immune system increases with time and prolong life of AIDs and treatment population.

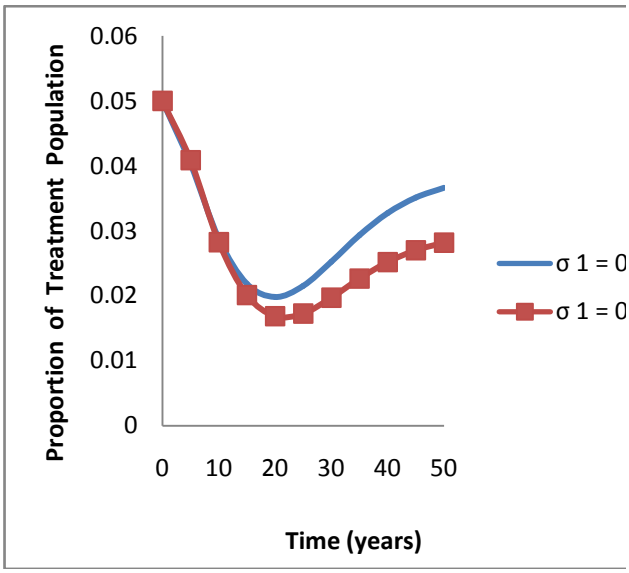

Figure 3a. Graph of Treatment class against time for different values of Fraction of the rate of movement from Pre – AIDS Class joining the AIDs Class (σ_1)

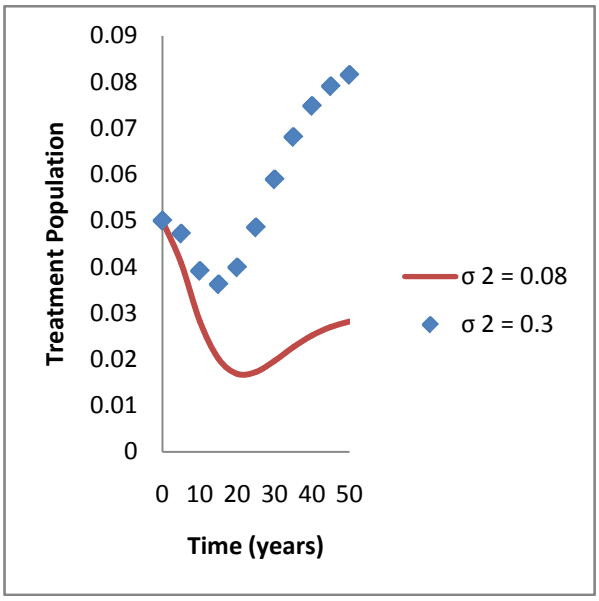

Figure 4a. Graph of Treatment class against time for different values of Fraction of the rate of movement from Treatment Class joining the AIDs Class (σ_2)

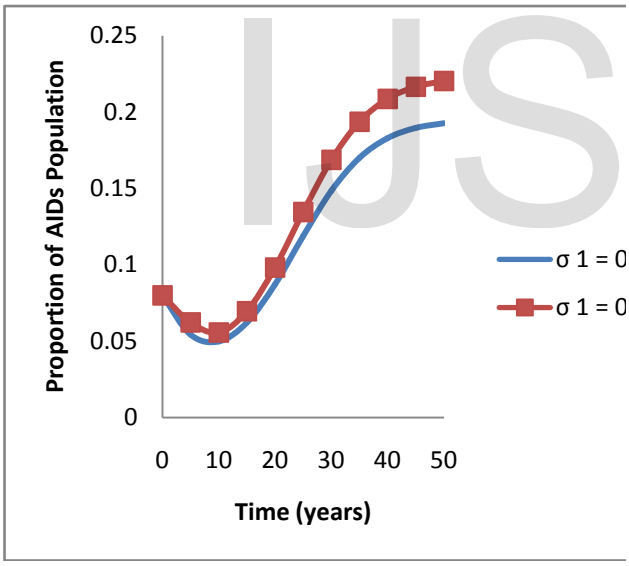

Figure 3b. Graph of Aids class against time for different values of Fraction of the rate of movement from Pre – AIDS Class joining the AIDs Class (σ_1)

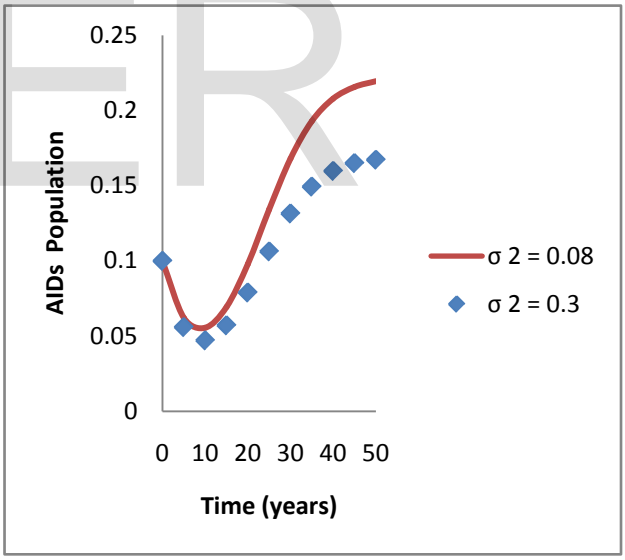

Figure 4b. Graph of Aids class against time for different values of Fraction of the rate of movement from Treatment Class joining the AIDs Class (σ_2)

It is seen that from the figures 3a-b. when $\boldsymbol{\sigma_1}$ increases, the Treatment population also decreases then start to increases in the presence of treatment and the AIDS population decreases with time then start to increase. This is caused by ARVs it prolonging the life span.

It is found from the Figure 4a, that as $\boldsymbol{\sigma_2}$ increases Treated population increases while it is found from the Figure 4b, that as $\boldsymbol{\sigma_2}$ increases AIDs population decreases.

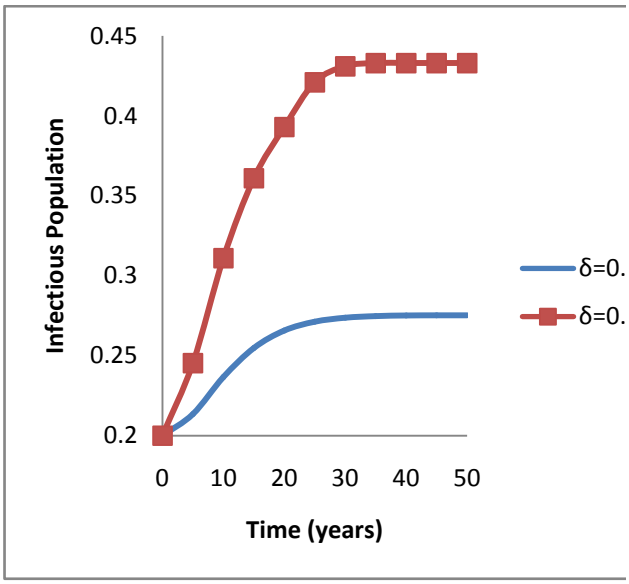

Figure 5a. Graph of Infectious class against time for different values of Rate of movement from infectious class (δ)

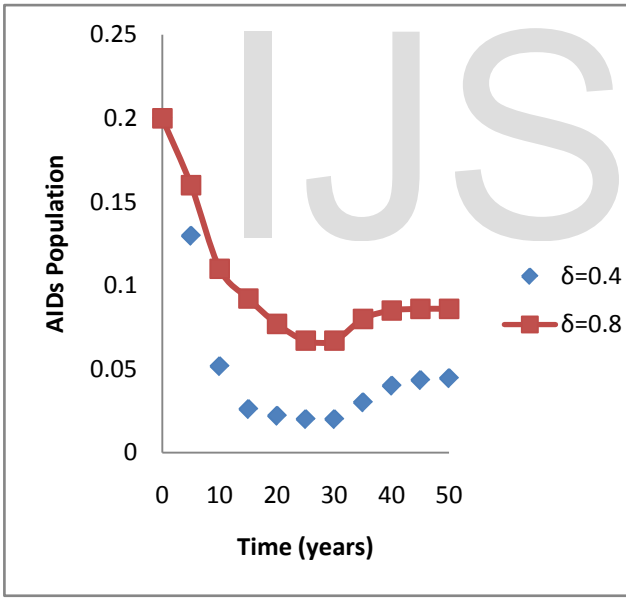

Figure 5b. Graph of AIDs class against time for different values of Rate of movement from infectious class (δ)

It is seen from figure 5a., that as **δ** increase the infected population decreases while It is seen from figure 5b., that as **δ** increase the AIDs population increases this depends on the viral counts of individual .

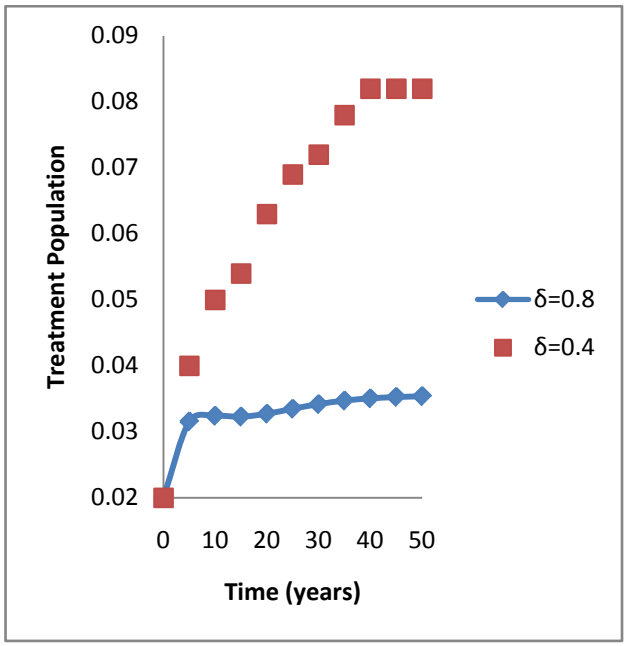

Figure 5c. Graph of Treatment class against time for different values of Rate of movement from infectious class (δ)

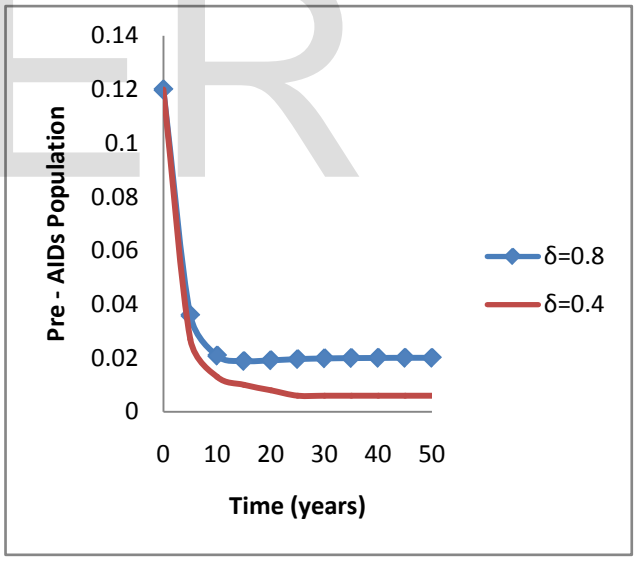

Figure 5d. Graph of Pre - AIDs class against time for different values of Rate of movement from infectious class (δ)

It is seen from figure 5c., that as **δ** increase the treatment population decreases while It is seen from figure 5d., that as **δ** increase the Pre-AIDs population increases this depends on the viral counts of individual .

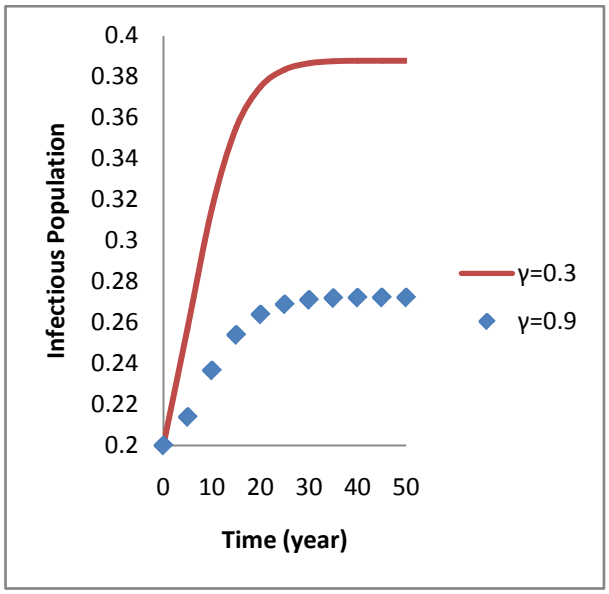

Figure 6a. Graph of Infectious class against time for different values of Rate of movement of infected individual into Aids Population (γ)

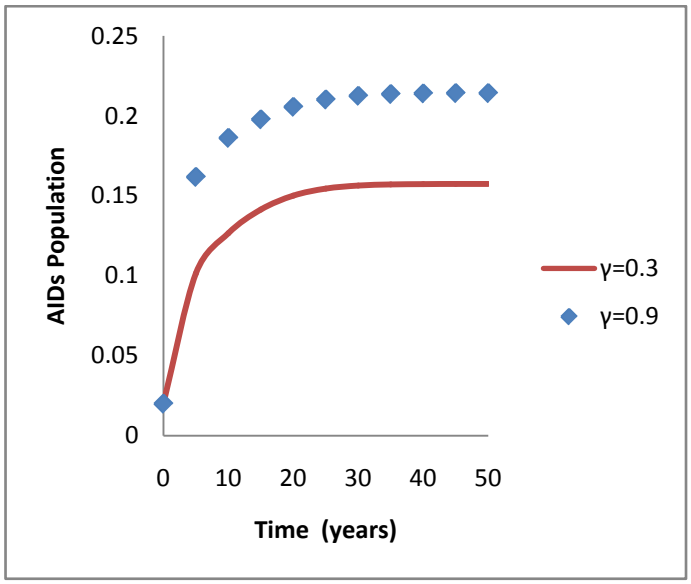

Figure 6c. Graph of AIDs Population class against time for different values of Rate of movement of Infected individual into Aids Population (γ)

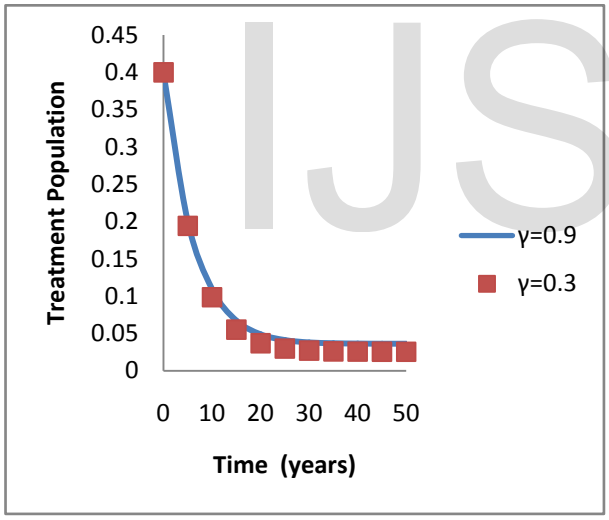

Figure 6b. Graph of Treatment class against time for different values of Rate of movement of Infected individual into Aids Population (γ)

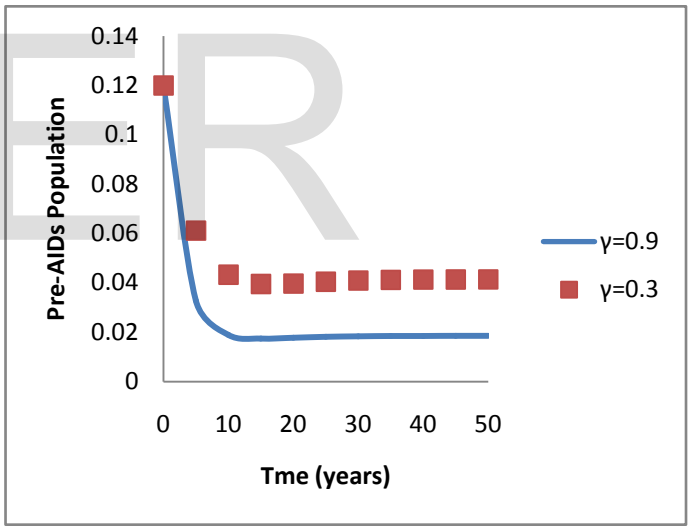

Figure 6d. Graph of Pre-AIDs class against time for different values of Rate of movement of Infected individual into Aids Population (γ)

Figure 6a-b., Shows that as rate of movement of infected individual increase the infectious population decreases while the treatment population increases slightly. This is caused by ARVs it prolonging the life span.

Figure 6c-d., Shows that as rate of movement of infected individual increase the AIDs population increase while the Pre-AIDs population decrease. This is caused by ARVs it prolonging the life span.

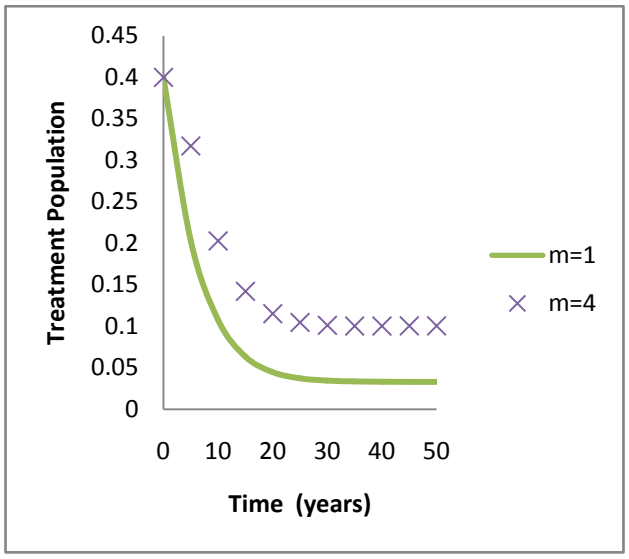

Figure 7a. Graph of Treatment class against time for different values of Fraction of γ who get treatment (m)

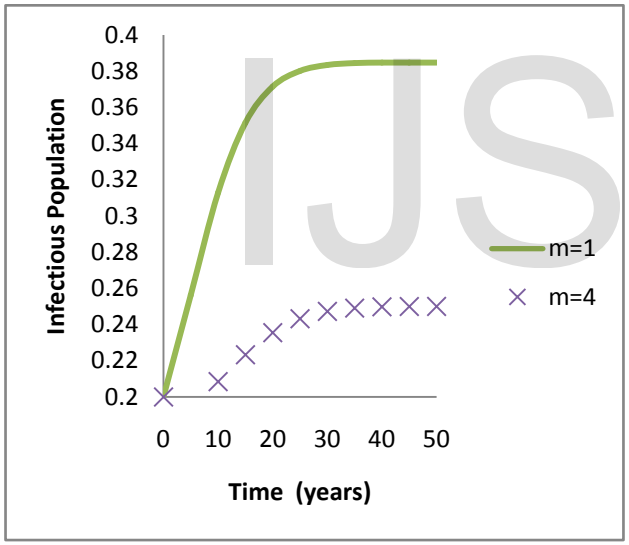

Figure 7b. Graph of Infectious class against time for different values of Fraction of γ who get treatment (m)

Figure 7a-b., Shows that as the fraction of γ who get treatment increase the treatment population increase while the Infectious population decrease. This is caused by ARVs it prolonging the life span.

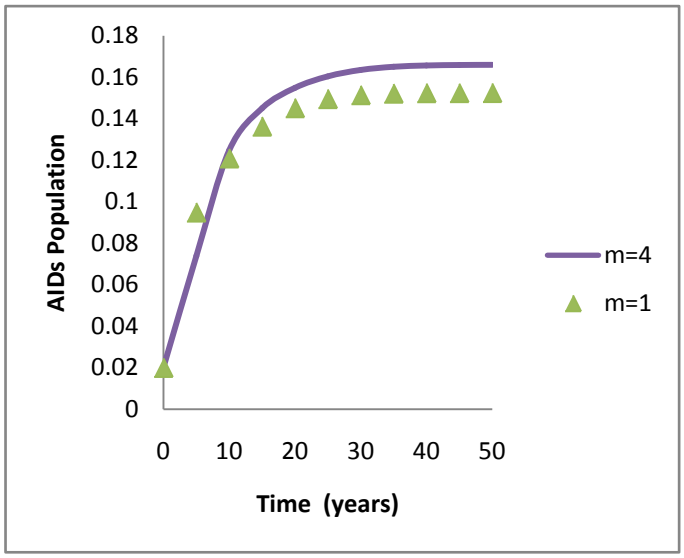

Figure 7c. Graph of AIDs class against time for different values of Fraction of γ who get treatment (m)

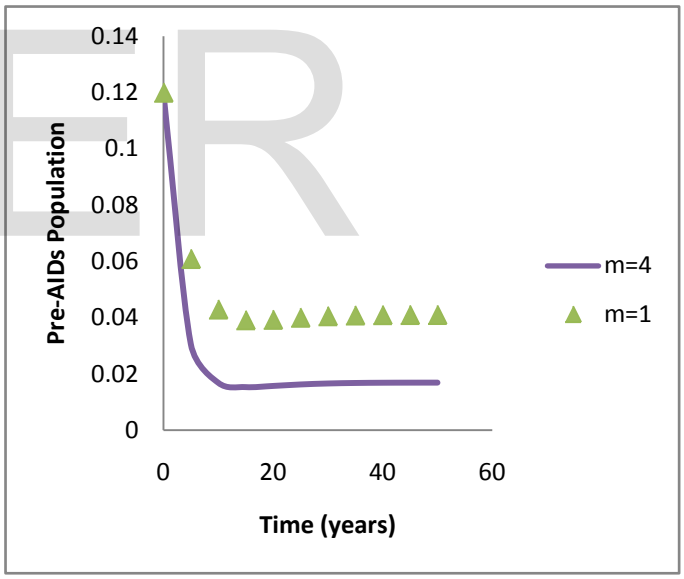

Figure 7d. Graph of Pre-AIDs class against time for different values of Fraction of γ who get treatment (m)

Figure 7c.-d, Shows that as the fraction of γ who get treatment increase the AIDs population increase and the Pre-AIDs population decrease. This is caused by ARVs it prolonging the life span.

5. Discussion and conclusions

In the study, a non linear mathematical model has been proposed and analysis to the study of Progression rate, treatment and vertical transmission of HIV/AIDs . The disease free and endemic equilibrium Were obtained and there stabilities investigated . The model showed that the disease free equilibrium is locally asymptotically stable by using Routh Hurwitz criteria but globally the disease free equilibrium is not Stable by comparism approach.

The presence of treatment prolong the life span of the individual infected with HIV and sexual contact rate and vertical transmission have high impact rate on the spread of the disease.

In conclusion the results show that increased change in sexual habits and providing ART treatment at the pre-AIDs stage reduce the infection much faster than starting treatment after progression into AIDs.

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