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An HIV/AIDS Model with Vertical Transmission, Treatment and Progression Rate

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Authors' contributions

This work was carried out in collaboration between both authors. Both authors read and approved the final manuscript.

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Abstract

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 usually breaks down the body immune system, leaving the victim vulnerable to a lot of other diseases. Therefore, in this study a nonlinear mathematical model of HIV/AIDS with treatment, vertical transmission and progression rate were considered.

The basic reproduction number (R_0) was evaluate by next generation matrix and the global stability was examine by the comparison approach. The disease – free and the endemic equilibrium of the model were determined by setting all compartments to be zero. The sensitivity analysis was carried out to determine the parameter that has high impact on the spread of the disease using partial derivatives and the Maple software 14 was used for numerical simulation of the model.

The disease free and endemic equilibrium were obtained and their stabilities studied. The model showed that the disease free equilibrium is locally asymptotically stable by using Routh-Hurwitz criteria and globally the disease free equilibrium is stable by comparism approach. The numerical simulation showed that by using treatment measures and controlling the rate of vertical transmission with time, the spread of the disease can be reduced significantly and by providing treatment at the pre-AIDs stage reduces the infection much faster than starting treatment after progression into AIDs.

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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 [1]. 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 [2], 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 [3].

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** [4], 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 [5].

Treatment is the process of offering the HIV positive individual with a life prolonging drug/medicine known as antiretroviral therapy (ART) or Highly Active Antiretroviral **(HAART) t**herapy [6]. 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 [7,8,9].

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 [10] 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 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 [10,7,11] 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 qualitative simulation.

2 Model Formulations

A nonlinear mathematical model is proposed and analyzed to study of HIV/AIDS with treatment and vertical transmission and progression rate. 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 class S(t) , infective class I(t) (also assumed to be infectious), pre-AIDS class P(t) , treated class T(t) and AIDs class A(t) with natural mortality rate μ in all classes [10,12,2].

The classes' interacts between themselves with the following assumptions: the susceptible become HIV infected through sexual contacts with HIV infective which may also lead to the birth of infected children (vertical transmission). Some portion of new born children are infected during birth and hence are directly recruited into the infective class with a rate $(1-\varepsilon)\theta$, $(0 \leq \varepsilon \leq 1)$ and others die effectively at birth, where ε is the portion of newborns infected with HIV who dies immediately after birth and θ is the rate of newborns infected with HIV. Direct recruitment of the infected persons were not consider, it was only vertical transmission that was put into consideration.

It was 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 [10,12,2], proceed with a rate γ to develop full blown AIDS. Part 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 $(1-\sigma_1-\sigma_2)\delta$ [13,14].

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 portion of infective which develops AIDS during the period of getting sexual maturity, if they survive the maturity period joins the AIDS class [15,2].

Thus, in this 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 [16,15,17]. 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 children are infected at birth ($\tau = 0$). And the purpose of this study is to examine the rate of movement from infective class to AIDs class and to determine what can be done to reduce it with time. [18,19,20].

The work of Waziri et al. [10] was modified, and stated below is the work of Waziri et al. [10]:

$$\begin{aligned}
\frac{dS}{dt} &= \pi N - \frac{C_1\beta_1 IS}{N} - \frac{C_2\beta_2 PS}{N} - \frac{C_3\beta_3 TS}{N} - \frac{C_4\beta_4 AS}{N} - \mu S \\
\frac{dI}{dt} &= \frac{C_1\beta_1 IS}{N} + \frac{C_2\beta_2 PS}{N} + \frac{C_3\beta_3 TS}{N} + \frac{C_4\beta_4 AS}{N} - (\delta+\mu)I + (1-\varepsilon)\theta(I+P+A) \\
\frac{dP}{dt} &= \sigma_1\delta I - (\gamma+\mu)P \qquad (1^*) \\
\frac{dT}{dt} &= \sigma_2\delta I + m\gamma P + VA - (K+\mu)T \\
\frac{dA}{dt} &= (1-\sigma_1-\sigma_2)\delta I + (1-m)\gamma P + KT - (V+\alpha+\mu)A
\end{aligned}$$

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:

$$\begin{aligned}
\frac{dS}{dt} &= \pi N - \frac{C_1\beta_1 IS}{N} - \frac{C_2\beta_2 PS}{N} - \frac{C_3\beta_3 TS}{N} - \frac{C_4\beta_4 AS}{N} - \gamma\varepsilon I(t-\tau)\, e^{-\mu\tau} - \mu S \\
\frac{dI}{dt} &= \frac{C_1\beta_1 IS}{N} + \frac{C_2\beta_2 PS}{N} + \frac{C_3\beta_3 TS}{N} + \frac{C_4\beta_4 AS}{N} - (\delta+\mu)I - \gamma(1-\varepsilon)I(t-\tau)e^{-\mu\tau} + (1-\varepsilon)\theta(I+P+A) \\
\frac{dP}{dt} &= \sigma_1\delta I - (\gamma+\mu)P \qquad (1) \\
\frac{dT}{dt} &= \sigma_2\delta I + m\gamma P + VA - (K+\mu)T \\
\frac{dA}{dt} &= (1-\sigma_1-\sigma_2)\delta I + \gamma I(t-\tau)\, e^{-\mu\tau} + (1-m)\gamma P + KT - (V+\alpha+\mu)A
\end{aligned}$$

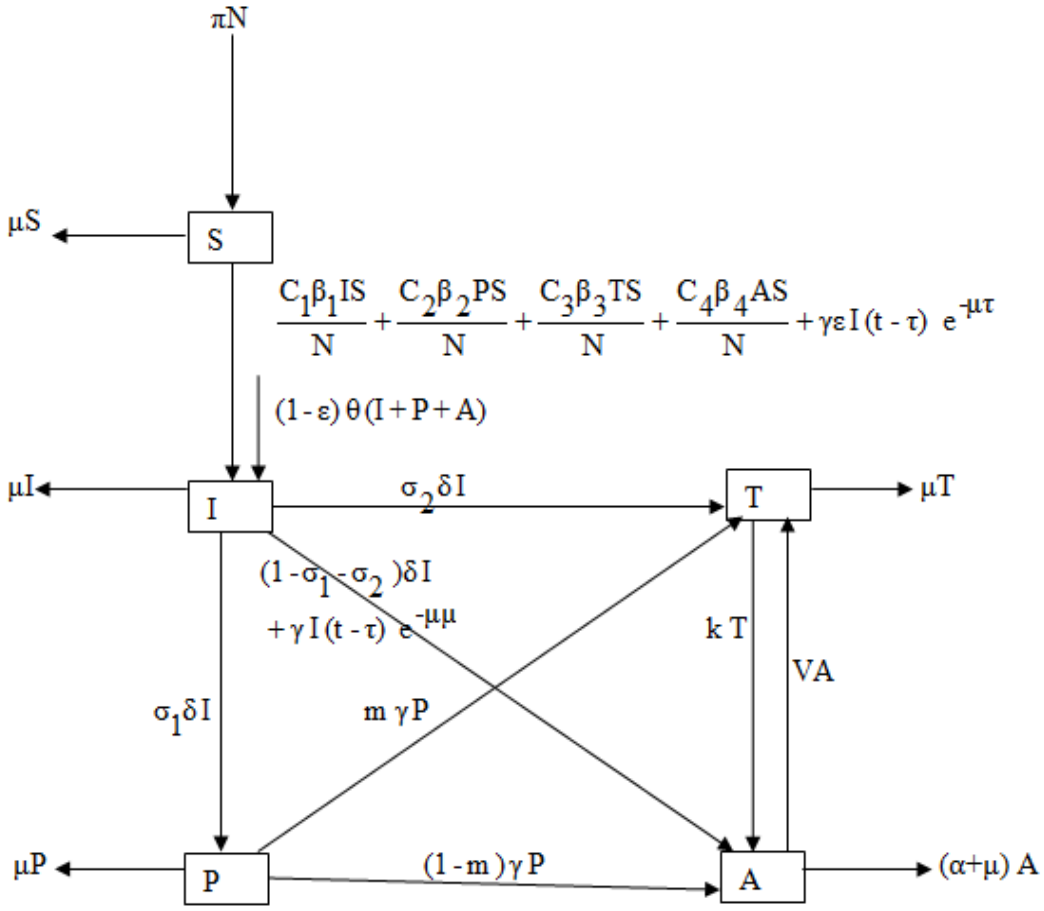

Fig. 1. The flow chart of the new model

where

C_i = Average number of sexual partners per unit time, where i =1, 2, 3, 4.

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.

The model (1) was simplify with this assumption that the AIDs class and those in pre-AIDs class are isolated and sexually inactive, which means they are not capable of producing children; at $\tau = 0$ and $(1-\varepsilon)\theta P = (1-\varepsilon)\theta A = 0$ and 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:-

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

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

Total population N at any time t is given by

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

This gives:

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

From equation (3), if the disease, AIDs and infective is removed, the total population size N is stationary for μ, and declining for $\pi < \mu$ and grows exponentially for $\pi > \mu$. It was assumed that mortality rate μ is a function of state variable [15]. Since the model is homogenous of degree one, the variable was be normalized by setting:

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

That leads to the normalized system:-

$$\frac{ds}{dt} = \pi - C_1\beta_1 is - C_3\beta_3 hs - \gamma\varepsilon i - [\pi + (1-\varepsilon)\theta i - \alpha a]s$$
$$\frac{di}{dt} = C_1\beta_1 is + C_3\beta_3 hs - \gamma(1-\varepsilon)i + (1-\varepsilon)\theta i - [\pi + \delta + (1-\varepsilon)\theta i - \alpha a]i$$
$$\frac{dp}{dt} = \sigma_1\delta i - [\pi + \gamma + (1-\varepsilon)\theta i - \alpha a]p \quad (5)$$
$$\frac{dh}{dt} = \sigma_2\delta i + m\gamma p + Va - [\pi + k + (1-\varepsilon)\theta i - \alpha a]h$$
$$\frac{da}{dt} = (1 - \sigma_1 - \sigma_2)\delta i + \gamma i + (1-m)\gamma p + kh - [\pi + V + \alpha + (1-\varepsilon)\theta i - \alpha a]a$$

where

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

Continuity of right-hand side of the system (3) and its derivative imply that the model is correctly set 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 [6]. The reproductive number R_0, was examine to determine if the disease become endemic in a population or not, other analysis of the model was carried out to determine the impact of treatment, vertical transmission and progression rate on the transmission of HIV/AIDS infection in a population.

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 region D.

3.2 Positivity of solution

For the model (5) to be epidemiological meaningful and correctly set. There is a 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,

$$\frac{ds}{dt} \leq \pi - \pi s \tag{6}$$

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

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 no disease in the population is called disease free equilibrium (DFE), and then obtained by setting, system (5) to be zero.

$$\frac{ds}{dt}=\frac{di}{dt}=\frac{dp}{dt}=\frac{dh}{dt}=\frac{da}{dt}=0 \tag{7}$$

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 [10,7,3]. To compute the basic reproduction number, the next generation method was applied on system (5). This definition is given for the models that represent the spreading of infection in a population [10]. It was obtained by taking the largest (dominant) eigenvalue (spectral radius) of:

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

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

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

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

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

3.5 Local stability of DFE

Theorem 2:

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

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

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 $a_4 > 0$

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

Therefore, $R_o < 1$

The proof 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 $(s, i, p, h, a) \to (1, 0, 0, 0, 0)$

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

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

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

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

$$\begin{aligned} F_4 = {} & \gamma^2\theta C_3\beta_3\nu\varepsilon - \gamma\theta C_3\beta_3\nu\pi + \sigma_1\delta C_3\beta_3\nu\pi - \sigma_2\delta C_3\beta_3\gamma\pi - \sigma_2\delta C_3\beta_3\alpha\pi - \sigma_2\delta C_3\beta_3\alpha\gamma \\ & + \gamma\theta\varepsilon C_3\beta_3\nu\pi - \sigma_1\delta C_3\beta_3 m\gamma\pi - \sigma_1\delta C_3\beta_3 m\gamma\alpha + \alpha k\gamma\pi + \alpha k\gamma\delta - \pi^2\gamma C_1\beta_1 + \pi^2\gamma\theta\varepsilon \\ & - \pi^2 kC_1\beta_1 - k\pi^2\gamma\varepsilon + k\pi^2\theta\varepsilon + k\pi\gamma\delta - k\pi\gamma^2\varepsilon - k\pi\gamma\theta - \pi^2\nu C_1\beta_1 - \nu\pi^2\gamma\varepsilon + \nu\pi^2\theta\varepsilon \\ & + \nu\pi\gamma\delta - \nu\pi\gamma^2\varepsilon - \nu\pi\gamma\theta - \alpha\pi^2 C_1\beta_1 - \alpha\pi^2\gamma\varepsilon + \alpha\pi^2\theta\varepsilon + \alpha\pi\gamma\delta - \alpha\pi\gamma^2\varepsilon - \alpha\pi\gamma\theta \\ & + \alpha\pi k\delta - \alpha\pi k\theta - \delta C_3\beta_3\nu\pi - C_3\beta_3\nu\gamma^2\theta - \delta C_3\beta_3\nu\gamma - \delta C_3\beta_3\sigma_2\pi^2 - \pi k\gamma C_1\beta_1 \\ & + \pi k\gamma\theta\varepsilon - \nu\pi\gamma C_1\beta_1 + \nu\pi\gamma\theta\varepsilon - \alpha\pi\gamma C_1\beta_1 + \alpha\pi\gamma\theta\varepsilon - \alpha k\pi C_1\beta_1 - \alpha k\pi\gamma\varepsilon + \alpha k\pi\theta\varepsilon \\ & - \alpha k\gamma C_1\beta_1 + \alpha k\gamma\theta\varepsilon - \pi^3 C_1\beta_1 - \pi^3\gamma\varepsilon + \pi^3\theta\varepsilon + \pi^2\gamma\delta - \pi^2\gamma^2\varepsilon - \pi^2\gamma\theta + \pi^3\delta - \pi^3\theta \\ & + \pi^3\gamma + \pi^3 k + \pi^3\nu + \pi^3\alpha + \pi^4 - \alpha k\gamma^2\varepsilon - \alpha k\gamma\theta + k\pi^2\delta - k\pi^2\theta + k\pi^2\gamma + \nu\pi^2\delta - \nu\pi^2\theta \\ & + \nu\pi^2\gamma + \alpha\pi^2\delta - \alpha\pi^2\theta + \alpha\pi^2\gamma + \alpha k\pi^2; \end{aligned}$$

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

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

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, $\boldsymbol{\beta_1}$ = 0.4, $\boldsymbol{\beta_3}$ = 0.05, $\boldsymbol{\sigma_1}$ = 0.2, $\boldsymbol{\sigma_3}$ = 0.01, k = 0.08, γ= 0.9, m = 0.4, π = 0.4, δ= 0.6, α=1, $\boldsymbol{c_1}$ = 3, $\boldsymbol{c_3}$ = 1 [10]

With initial values $s(0)$=0.5, $i(0)$ = 0.3, $p(0)$ = 0.12, $h(0)$ = 0.07 , $a(0)$ = 0.01.
Figs. 1a-b, it was discovered that as fraction of new born children increase the infectious population increase and the Treatment population decrease with time in the presence of ARVs.

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

It is seen from Fig. 3a that as **δ** increase the infected population decreases while it is seen from Fig. 3b that as **δ** increase the AIDs population increases this depends on the viral counts of individual.

It is seen from Fig. 3c., that as **δ** increase the treatment population decreases while It is seen from Fig. 3d., that as **δ** increase the Pre-AIDs population increases this depends on the viral counts of individual.

Figs. 4a-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.

Figs. 4c-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.

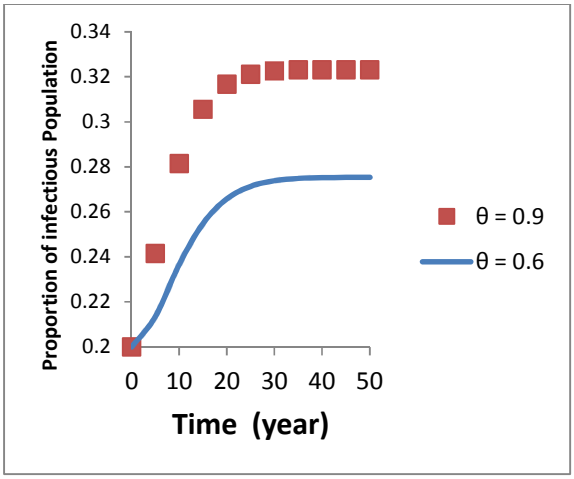

Fig. 1a. Graph of Infectious class against time for different values of rate of newborns infected with HIV (θ)

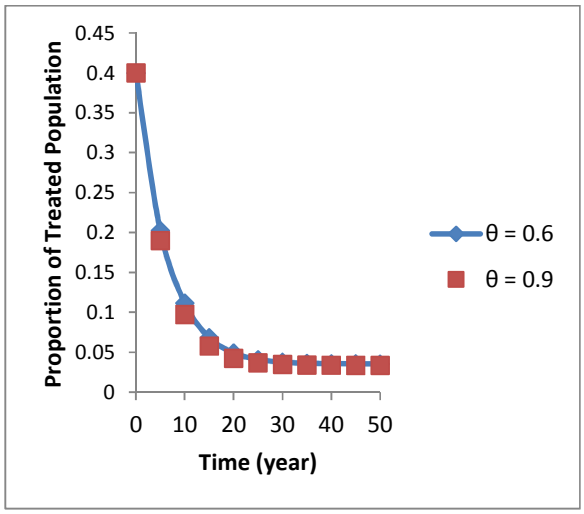

Fig. 1b. Graph of treated class against time for different values of rate of newborns infected with HIV(Θ)

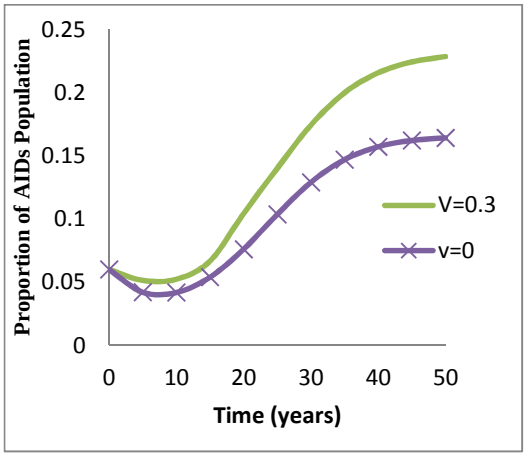

Fig. 2a. Graph of AIDs class against time for different values of rate of which Aids patients get treatment (υ)

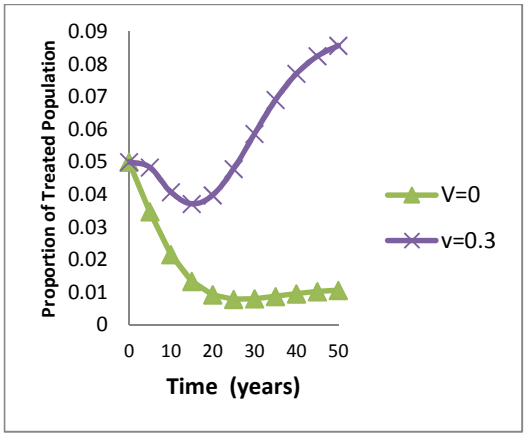

Fig. 2b. Graph of Treatment class against time for different values of rate of which Aids patients get treatment (υ)

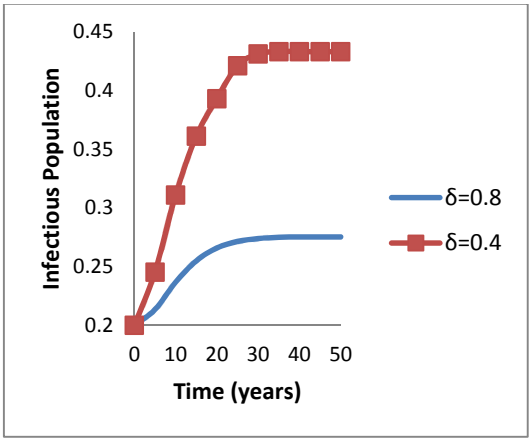

Fig. 3a. Graph of Infectious class against time for different values of rate of movement from infectious class (δ)

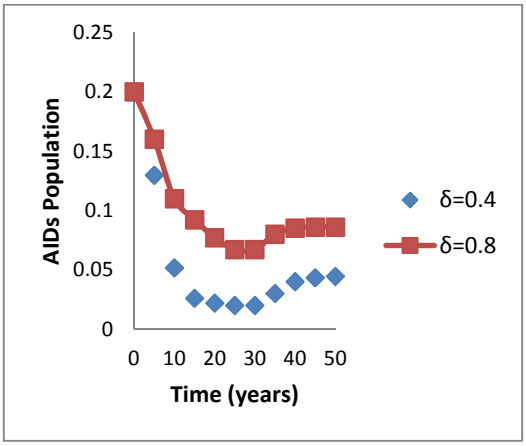

Fig. 3b. Graph of AIDs class against time for different values of rate of movement from infectious class (δ)

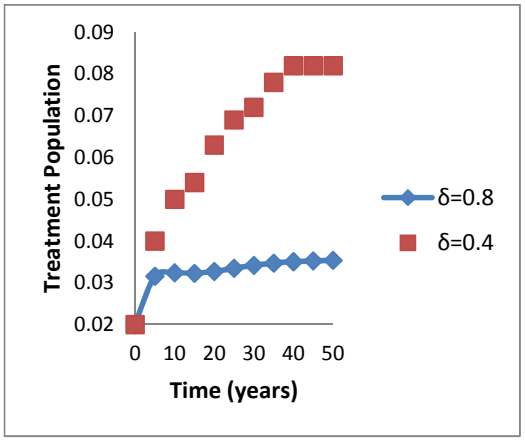

Fig. 3c. Graph of Treatment class against time for different values of rate of movement from infectious class (δ)

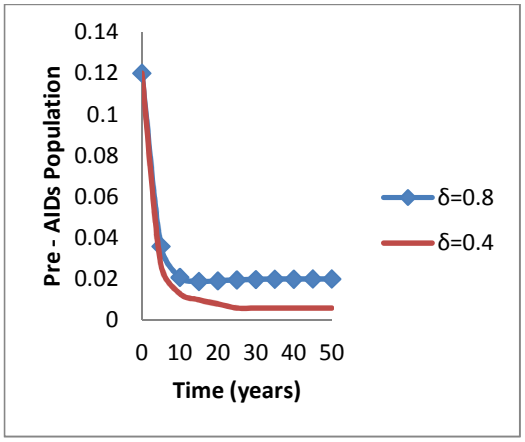

Fig. 3d. Graph of Pre-AIDs class against time for different values of rate of movement from infectious class (δ)

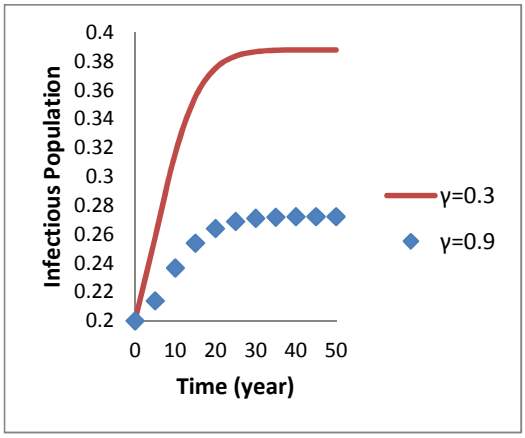

Fig. 4a. Graph of Infectious class against time for different values of rate of movement of infected individual into AIDs population (γ)

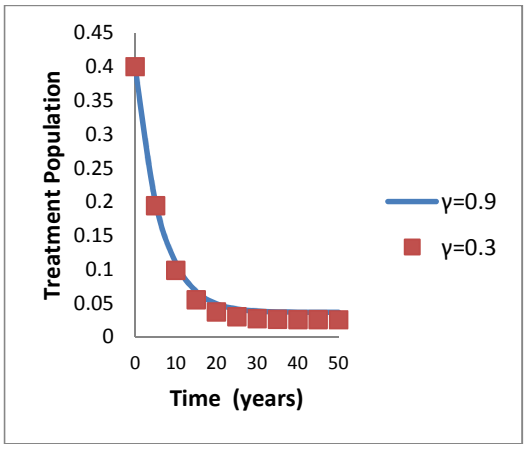

Fig. 4b. Graph of Treatment class against time for different values of rate of movement of infected individual into AIDs population (γ)

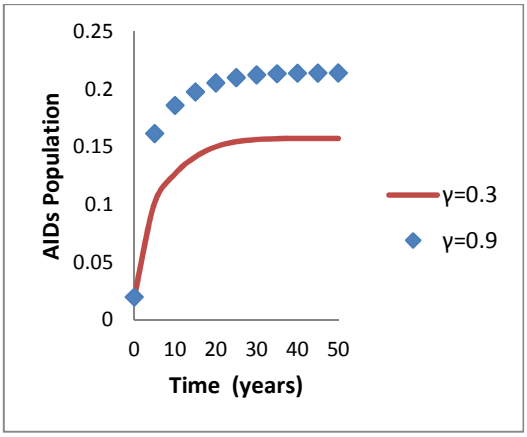

Fig. 4c. Graph of AIDs population class against time for different values of rate of movement of infected individual into AIDs population (γ)

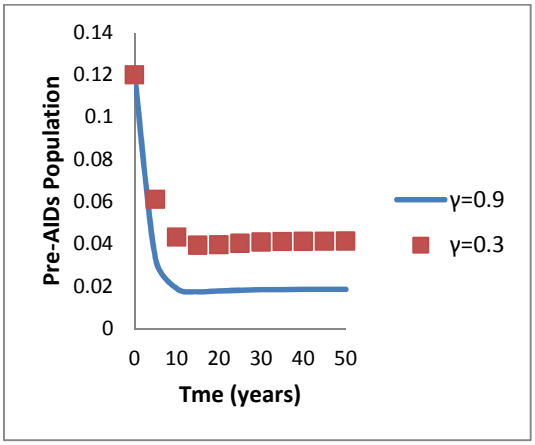

Fig. 4d. Graph of Pre-AIDs class against time for different values of rate of movement of infected individual into Aids Population (γ)

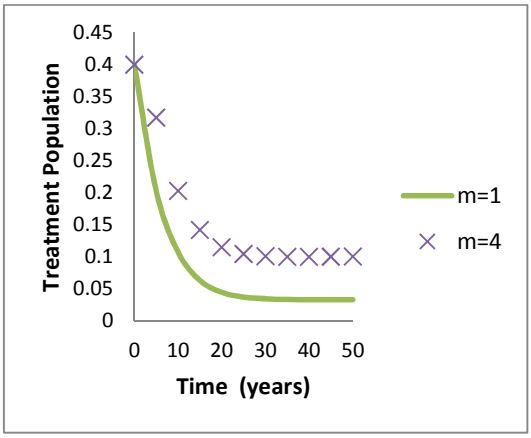

Fig. 5a. Graph of Treatment class against time for different values of fraction of γ who get treatment (m)

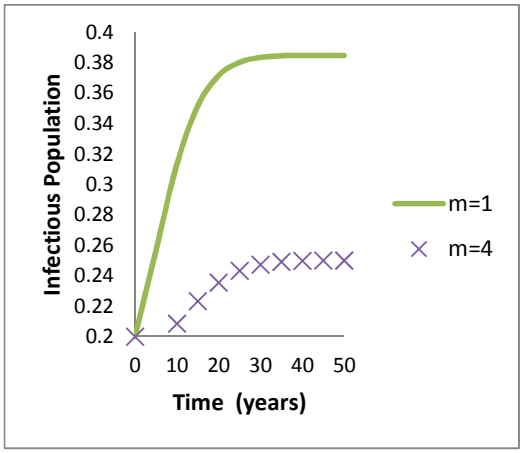

Fig. 5b. Graph of Infectious class against time for different values of fraction of γ who get treatment (m)

Figs. 5a-b shows that as the fraction of γ who get treatment increase the treatment population increases while the infectious population decreases. This is caused by ARVs it prolong the life span.

5 Discussion and Conclusions

In the study, a nonlinear 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 and globally the disease free equilibrium is stable by comparism approach.

Provision of treatment to HIV infected individual will prolongs the life span of such fellow and sexual contact rate and vertical transmission contribute majorly to 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.

Competing Interests

Authors have declared that no competing interests exist.

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