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MATHEMATICAL MODELLING OF HIV INFECTION

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ABSTRACT

We formulate a deterministic mathematical model for the HIV/AIDS.

***Keywords**: HIV/AIDS, TB, vertical transmission, local stability, global stability.*

MODEL FORMULATION

In modeling the total human population at any time t, denoted by N(t). The total population is subdivided into sub-population namely, Susceptible S(t), who are not yet infected but can be infected by HIV individuals through sexual contacts, HIV infected individuals not yet displaying symptoms of AIDS $I_H(t)$, individuals treated for HIV showing symptoms of AIDS, T(t) and individuals with full blown AIDS still, A(t).

Thus we have $N(t) = S(t) + I_H(t) + T(t) + A(t)$ (1.1)

Where β_1 is per capita contact rate susceptible human with HIV infected individuals. λ is recruitment rate/birth rate of humans. δ_1 is rate at which HIV infected individuals progress for treatment. ε_2 is the progression rate at which HIV will go back to HIV infected class after treatment. δ is treatment rate and π is progression rate from treatment.

The resulting system of equations is shown below:

$$\begin{aligned}
\frac{dS}{dt} &= \lambda N - \frac{\beta_1 S}{N} - \mu_1 S, \\
\frac{dI}{dt} &= \frac{\beta_1 S}{N} + \varepsilon_2 \delta T - (\sigma_1 + \mu_1) I, \\
\frac{dT}{dt} &= (1-\pi)\sigma_1 I - (\delta + \mu_1) T, \\
\frac{dA}{dt} &= \pi\sigma_1 I + (1-\varepsilon_2)\delta T - (\alpha + \mu_1) A
\end{aligned} \qquad (1.2)$$

We normalize the system (3.2) by introducing the following $s = \frac{S}{N}, i = \frac{I}{N}, t = \frac{T}{N}, w = \frac{A}{N}$

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Thus the system becomes

$$\begin{aligned}
\frac{ds}{dt} &= \lambda(1-s) - \beta_1 s + w\alpha s, \\
\frac{di}{dt} &= \beta_1 s + w\alpha i - (\lambda + \sigma_1) i + \delta t \varepsilon_2, \\
\frac{dt_h}{dt} &= (1-\pi)\sigma_1 i - (\delta + \lambda) t + w\alpha t, \\
\frac{dw}{dt} &= \pi\sigma_1 i + (1-\varepsilon_2)\delta t - (\alpha + \lambda) w + w(w\alpha)
\end{aligned} \tag{1.3}$$

Positivity and boundedness of solutions

Theorem 3.1: If S(0), I(0) and A(0) are non-negative, then so are s(t), i(t), t_h(t) and w(t) for all t>0. Moreover

$$\lim_{x\to\infty} N(t) \le \frac{\lambda}{\mu_1} \tag{1.4}$$

Furthermore if $N(0) \le \frac{\lambda}{\mu_1}$ then $N(t) \le \frac{\lambda}{\mu_1}$. In particular the region $\Omega = \Omega_1 \subset R_+^4$ with

$\Omega = \left\{ (S, I, T, A) \in R_+^4 : S + I + T + A \le \frac{\lambda}{\mu_1} \right\}$ is positively invariant.

From this theorem we conclude that it is sufficient to consider the dynamics of 3.2 in Ω. In this region, the model can be considered as being epidemiologically well posed (Hethcote, 2000).

Stability of the disease-free equilibrium (DFE)

At disease free equilibrium, it is assumed that there is no infection. Then we set $i = t = w = 0$ (which can be interpreted as a quarantine program). But at disease free equilibrium, the susceptible population is equal to total population, that is to say $s = 1$.

Therefore the Disease Free Equilibrium (DFE) denoted by E_0 of the model system (1.3) is given by $E_0 = (s,0,0,0) = (1,0,0,0)$.

The Basic Reproduction Number, R_0

The basic reproduction number of the model (3.3) is calculated using the next generation matrix (Driessche and Watmough, 2002). Using this approach, we have,

$$F_1 = \begin{pmatrix} F_1 \\ F_2 \\ F_3 \end{pmatrix} = \begin{pmatrix} \beta_1 i s \\ 0 \\ 0 \end{pmatrix} \text{ and } V_i = \begin{pmatrix} V_1 \\ V_2 \\ V_3 \end{pmatrix} = \begin{pmatrix} -W\alpha I + (\lambda + \sigma_1) i - \delta t \varepsilon_2 \\ -(1-\pi)\sigma_1 i + (\delta + \lambda) t - w\alpha t \\ -\pi\sigma_1 i - (1-\varepsilon_2)\delta t + (\alpha + \lambda) w - w(w\alpha) \end{pmatrix} \tag{1.5}$$

After taking partial derivatives we have

$$F = \begin{pmatrix} \beta_1 s & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} \lambda + \sigma_1 & -\delta\varepsilon_2 & 0 \\ \pi\sigma_1 - \sigma_1 & \delta + \lambda & 0 \\ -\pi\sigma_1 & \delta - \delta\varepsilon_2 & \alpha + \lambda \end{pmatrix}$$

And then we have

$$FV^{-1} = \begin{pmatrix} \dfrac{\beta_1(\delta+\lambda)}{\lambda(\delta+\lambda)+(\delta+(-1+\pi)\delta\varepsilon_2+\lambda)\sigma_1} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

The reproduction number is the dominant eigenvalues of FV^{-1}. Thus

$$R_0 = \frac{\beta_1(\delta+\lambda)}{\lambda(\delta+\lambda)+(\delta+(-1+\pi)\delta\varepsilon_2+\lambda)\sigma_1} \tag{1.6}$$

The threshold quantity R_0 is the basic reproduction number of the normalized model system (1.3) for HIV/AIDS infection in a population with treatment. It measures the average number of new infections generated by a single infected individual in a completely susceptible population (Anderson et al 1995)

Local Stability of Disease Free Equilibrium (DFE) – HIV/AIDS only

Theorem 1: The Disease-free Equilibrium of the system (2.3) is locally asymptotically stable if R0 <1, and unstable if R0 >1. . Thus the theorem implies the disease can be eliminated from the community. Now to determine the local stability of E0, the following variation matrix is computed corresponding to equilibrium point E0.

$$M_0 = \begin{pmatrix} -\lambda & -\beta_1 & 0 & \alpha \\ 0 & \beta_1-(\lambda+\sigma_1) & \delta\varepsilon_2 & 0 \\ 0 & (1-\pi)\sigma_1 & -(\delta+\lambda) & 0 \\ 0 & \pi\sigma_1 & (1-\varepsilon_2) & -(\alpha+\lambda) \end{pmatrix} \tag{1.7}$$

From (2.7) clearly $\eta_1 = \lambda$, $\eta_2 = -(\alpha+\lambda)$, η_3 and η_4 are obtained from

$$\begin{vmatrix} \beta_1-(\lambda+\sigma_1)-\eta & \delta\varepsilon_2 \\ (1-\pi)\sigma_1 & -(\delta+\lambda)-\eta \end{vmatrix} = 0,$$

Thus the characteristic equation corresponding to M_0 is given by

$$(\eta+\lambda)(\eta+\alpha+\mu)\{\eta^2+\eta(\delta-\beta_1+2\lambda+\sigma_1)-\beta(\delta+\lambda)+\delta\lambda+\lambda^2+\delta\sigma_1-\delta\varepsilon_2\sigma_1+\delta\pi\varepsilon_2\sigma_1+\lambda\sigma_1\} = 0,$$

We let $a_1 = (\delta-\beta_1+2\lambda+\sigma_1)$, and $a_2 = -\beta_1(\delta+\lambda)+\delta\lambda+\lambda^2+\delta\sigma_1-\delta\varepsilon_2\sigma_1+\delta\pi\varepsilon_2\sigma_1+\lambda\sigma_1$

Thus according to Routh Hurwitz Criteria for a 2 x 2 system, $E0$ is locally asymptotically stable when

$a_1 > 0$ and $a_2 > 0$,

Then from $a_2 > 0$ we will have

$$-\beta_1(\delta+\lambda)+\delta\lambda+\lambda^2+\delta\sigma_1-\delta\varepsilon_2\sigma_1+\delta\pi\varepsilon_2\sigma_1+\lambda\sigma_1 > 0,$$

This will lead to

$$-\beta_1(\delta+\lambda) > -(\delta\lambda+\lambda^2+\delta\sigma_1-\delta\varepsilon_2\sigma_1+\delta\pi\varepsilon_2\sigma_1+\lambda\sigma_1), \tag{1.8}$$

Hence

$$\frac{\beta_1(\delta+\lambda)}{\delta\lambda+\lambda^2+\delta\sigma_1-\delta\varepsilon_2\sigma_1+\delta\pi\varepsilon_2\sigma_1+\lambda\sigma_1} < 1,$$

This means

$$R_0 < 1$$

The condition (3.8) is sufficient to satisfy all the equations

$a_1 > 0$ and $a_2 > 0$,

It is clear that for $R_0 < 1$ the disease free equilibrium E_0 is locally asymptotically stable such that the infection does not persist in the population and under this condition the endemic equilibrium does not exist. It is unstable for $R_0 > 1$ and then endemic equilibrium exists and the infection is maintained in the population.

Global stability of the disease free equilibrium

The disease free equilibrium of the system (3.3) is globally asymptotically stable whenever $R_0 < 1$ and unstable if $R_0 > 1.$

This is based on the comparison theorem (Lakshmkantham *et al*, 1989) to prove the global stability. The rate of change of the variables representing the infected components of the system can be written as follows.

$$\begin{pmatrix} \frac{di}{dt} \\ \frac{dt_h}{dt} \\ \frac{dw}{dt} \end{pmatrix} = (F - V)\begin{pmatrix} i \\ t_h \\ w \end{pmatrix} - F_i\begin{pmatrix} i \\ t_h \\ w \end{pmatrix} \tag{1.9}$$

Where

$$\begin{pmatrix} \frac{di}{dt} \\ \frac{dt_h}{dt} \\ \frac{dw}{dt} \end{pmatrix} = (F - V)\begin{pmatrix} \beta_1 - (\lambda + \sigma_1) + \delta\varepsilon_1 \\ -\pi\sigma_1 + \sigma_1 - (\delta + \lambda) \\ \pi\sigma_1 - (\alpha + \lambda) \end{pmatrix} - F_i\begin{pmatrix} i \\ t_h \\ w \end{pmatrix}, \tag{1.10}$$

Thus

$$\begin{pmatrix} \frac{di}{dt} \\ \frac{dt_h}{dt} \\ \frac{dw}{dt} \end{pmatrix} \leq (F - V)\begin{pmatrix} i \\ t_h \\ w \end{pmatrix} \tag{1.11}$$

According to (Castillo-Chavez et al 2002) and (Driessche and Watmough 2002), all eigenvalues of the matrix F -V have negative real parts. It follows that the linearized differential inequality above is stable whenever $R_0 < 1.$ Consequently $i = t_h = w = 0 \to (0,0,0,0)$ as $t \to \infty..$

Substituting $i = t_h = w = 0$ in (3.3) gives $s(t) \to s(0)$ as $t \to \infty.$ Thus we have established that the disease free equilibrium is globally asymptotically stable whenever $R_0 < 1$ and unstable if $R_0 > 1.$

Local Asymptotic Stability (LAS) of Endemic Equilibrium

We shall now analyze the local asymptotic stability (LAS) of endemic equilibrium, by using the Centre Manifold theory (Gumel et al 2009). The application of this theorem (Centre Manifold).

The theorem will be used to determine if the normalised model system (2.3) exhibit a backward or forward bifurcation at R_0 =1. This will be done by re-naming the variables as follows;

Let

$$s = x_1,\ i = x_2,\ t = x_3,\ w = x_4 \tag{1.12}$$

With

$$x_1 + x_2 + x_3 + x_4 = 1.$$

Furthermore, by introducing the vector notation $X = (x_1, x_2, x_3, x_4)^T$ the model can be rewritten in the form $\frac{dX}{dt} = F(x)$, whereby $F = (f_1, f_2, f_3, f_4)^T$ as follows;

$$\begin{aligned}
\frac{dx_1}{dt} &= f_1 = \lambda(1 - x_1) - \beta_1 x_2 x_1 + \alpha x_4 x_1, \\
\frac{dx_2}{dt} &= f_2 = \beta_1 x_2 x_1 + \alpha x_4 x_1 - (\lambda + \sigma_1) x_2 + \delta\varepsilon_2 x_3, \\
\frac{dx_3}{dt} &= f_3 = (1 - \pi)\sigma_1 x_2 - (\delta + \lambda) x_3 + \alpha x_4 x_3, \\
\frac{dx_4}{dt} &= f_4 = \pi\sigma_1 x_2 + (1 - \varepsilon_2)\delta x_3 - (\alpha + \lambda) x_4 + \alpha x_4^2
\end{aligned} \tag{1.13}$$

The Jacobian of the normalised model system at disease free is given by

$$M_{E0} = \begin{pmatrix} -\lambda & -\beta_1 & 0 & \alpha \\ 0 & \beta_1 - (\lambda + \sigma_1) & \delta\varepsilon_2 & 0 \\ 0 & (1 - \pi)\sigma_1 & -(\delta + \lambda) & 0 \\ 0 & \pi\sigma_1 & (1 - \varepsilon_2) & -(\alpha + \lambda) \end{pmatrix} \tag{3.14}$$

Let $\beta_1 = \beta^*$ be a bifurcation parameter and if we consider the case $R0$ =1 and solving for $\beta_1 = \beta^*$ from the equation

$$R_0 = \frac{\beta_1(\delta + \lambda)}{\lambda(\delta + \lambda) + (\delta + (-1 + \pi)\delta\varepsilon_2 + \lambda)\sigma_1} = 1,$$

We get

$$\beta_1 = \beta^* = \frac{\delta\lambda + \lambda^2 + \delta\sigma_1 - \delta\varepsilon_2\sigma_1 + \delta\pi\varepsilon_2\sigma_1}{\delta + \lambda} \tag{1.15}$$

The linearization system of equation (2.14) is transformed with $\beta_1 = \beta^*$ which has a simple zero eigenvalues. Hence the centre manifold theory can be used to analyse the dynamics of (2.14) near

$$\beta_1 = \beta^*$$

It can be shown that the Jacobian of (2.14) at $\beta_1 = \beta^*$ has a right eigenvector associated with the zero eigenvalues given by

$$\omega = (\omega_1, \omega_2, \omega_3, \omega_4)^T,$$

Then

$$\begin{pmatrix} -\lambda & -\beta_1 & 0 & \alpha \\ 0 & \beta_1-(\lambda+\sigma_1) & \delta\varepsilon_2 & 0 \\ 0 & (1-\pi)\sigma_1 & -(\delta+\lambda) & 0 \\ 0 & \pi\sigma_1 & (1-\varepsilon_2) & -(\alpha+\lambda) \end{pmatrix}\begin{pmatrix} \omega_1 \\ \omega_2 \\ \omega_3 \\ \omega_4 \end{pmatrix}=\begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \tag{1.16}$$

This gives

$\omega_3=\dfrac{(1-\pi)\sigma_1}{\delta+\lambda}\omega_2,\ \ \omega_4=\dfrac{\lambda(m_1+m_2)}{m_3}\omega_2,\ \omega_1=\dfrac{\alpha(m_1+m_2)-m_4}{m_3}\omega_2,$ for which $\omega_2>0$ is a free right eigenvector.

Where

$m_1=\pi\sigma_1(\delta+\lambda),\ m_2=(1-\varepsilon_2)(1-\pi)\sigma_1,\ m_3=\lambda(\alpha+\lambda)(\delta+\lambda),\ m_4=\beta_1(\alpha+\lambda)(\delta+\lambda).$

Furthermore, the Jacobian at E_0 has left eigenvector associated with the zero Eigenvalues at $\beta_1=\beta^*$ given by $v=(v_1,v_2,v_3,v_4)^T$, implying that

$$\begin{pmatrix} -\lambda & 0 & 0 & 0 \\ -\beta_1 & \beta_1-(\lambda+\sigma_1) & (1-\pi)\sigma_1 & \pi\sigma_1 \\ 0 & \delta\varepsilon_2 & -(\delta+\lambda) & (1-\varepsilon_2) \\ \alpha & 0 & 0 & -(\alpha+\lambda) \end{pmatrix}\begin{pmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \end{pmatrix}=\begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \tag{1.17}$$

Where

$v_1=0,$

$v_2=-\dfrac{\sigma_1[(1-\pi)(1-\varepsilon_2)+\pi(\delta+\lambda)]}{(\delta+\lambda)[\beta_1-(\lambda+\sigma_1)+(1-\pi)\sigma_1\delta\varepsilon_2]}v_4,$

$v_3=\dfrac{(\delta\varepsilon_2)[\sigma_1[(1-\pi)(1-\varepsilon_2)+\pi(\delta+\lambda)]]+(1-\varepsilon_2)(\delta+\lambda)[\beta_1-(\lambda+\sigma_1)+(1-\pi)\sigma_1\delta\varepsilon_2]}{(\delta+\lambda)^2[\beta_1-(\lambda+\sigma_1)+(1-\pi)\sigma_1\delta\varepsilon_2]}v_4$

For which $v_4>0$ is a free left eigenvector.

The Computations of a and b

From the normalised model system (2.1) the associated non-zero partial derivatives of F at disease free equilibrium are given by

$$\frac{\partial^2 f_1}{\partial x_1\partial x_2}=-\beta_1,\ \frac{\partial^2 f_1}{\partial x_1\partial x_4}=\alpha,\ \frac{\partial^2 f_2}{\partial x_2\partial x_1}=\beta_1,\ \frac{\partial^2 f_2}{\partial x_2\partial x_4}=\alpha,\ \frac{\partial^2 f_3}{\partial x_3\partial x_4}=\alpha,\ \frac{\partial^2 f_4}{\partial x_4^2}=2\alpha, \tag{1.18}$$

It follows from the above expression that

$$a=v_1\sum_{j=2}^{4}\omega_1\omega_j\frac{\partial^2 f_1}{\partial x_1\partial x_2}+v_2\sum_{j=2}^{4}\omega_2\omega_j\frac{\partial^2 f_2}{\partial x_1\partial x_j}+v_3\omega_3\omega_4\frac{\partial^2 f_3}{\partial x_3\partial x_4}+v_4\omega_4^2\frac{\partial^2 f_4}{\partial x_4^2},$$

Since $v_1=0,\ a$ becomes

$$a=v_2\left(\omega_1\omega_2\frac{\partial^2 f_2}{\partial x_1\partial x_2}+\omega_2\omega_4\frac{\partial^2 f_2}{\partial x_2\partial x_4}\right)+v_3\omega_3\omega_4\omega_4\frac{\partial^2 f_3}{\partial x_3\partial x_4}+v_4\omega_4^2\frac{\partial^2 f_4}{\partial x_4^2}, \tag{1.19}$$

And subsequently

$$a = v_2\left(\beta_1\omega_1\omega_2 + \alpha\omega_2\omega_4\right) + v_3\alpha\omega_3\omega_4 + 2\alpha v_4\omega_4^2,$$

Which simplifies to

$$a = v_2\frac{\left[\alpha\beta_1\left(m_1+m_2\right)+\left(m_1+m_3\right)-m_4\right]}{m_3}\omega_2^2 + \alpha v_3\sigma_1\lambda\frac{\left[\left(m_1+m_2\right)-\pi\left(m_1+m_3\right)\right]}{m_3\left(\delta+\lambda\right)}\omega_2^2 + 2\alpha v_4\lambda^2\frac{\left(m_1+m_2\right)^2}{m_3^2}\omega_2^2 \quad (1.20)$$

Where

m_1, m_2, m_3, and m_4 have maintained the same meaning as in equation (3.16)

Therefore $\alpha > 0$, if all the following inequalities are satisfied:

$$\alpha\beta_1\left(m_1+m_2\right)+\left(m_1+m_2\right) > m_4 \text{ and } \left(m_1+m_2\right) > \pi\left(m_1+m_3\right), \quad (1.21)$$

For the sign of b it can be shown that the associated non-zero partial derivatives of F at disease free equilibrium are

$$\frac{\partial^2 f_1}{\partial\beta_1\partial x_2} = -1 \text{ and } \frac{\partial^2 f_2}{\partial\beta_1\partial x_2} = 1$$

Implying that

$$b = v_1\omega_2\frac{\partial^2 f_1}{\partial\beta_1\partial x_2} + v_2\omega_2\frac{\partial^2 f_2}{\partial\beta_1\partial x_2}, \quad (1.22)$$

Which gives

$b = v_1\omega_2 + v_2\omega_2.$, But $v_1 = 0$ so that $b = v_2\omega_2$,

Where

$$v_2 = \left\{\frac{\sigma_1\left[\left(1-\pi\right)\left(1-\varepsilon_2\right)+\pi\left(\delta+\lambda\right)\right]}{\left(\lambda+\sigma_1\right)\left(\delta+\lambda\right)-\left(\delta+\lambda\right)\left[\beta_1+\left(1-\pi\right)\sigma_1\delta\varepsilon_2\right]}\right\}v_4,$$

Hence

$$b = \left\{\frac{\sigma_1\left[\left(1-\pi\right)\left(1-\varepsilon_2\right)+\pi\left(\delta+\lambda\right)\right]}{\left(\lambda+\sigma_1\right)\left(\delta+\lambda\right)-\left(\delta+\lambda\right)\left[\beta_1+\left(1-\pi\right)\sigma_1\delta\varepsilon_2\right]}\right\}\omega_2 v_4, \quad (1.23)$$

Implying that

$$b = \left\{\frac{\sigma_1\left[\left(1-\pi\right)\left(1-\varepsilon_2\right)+\pi\left(\delta+\lambda\right)\right]}{\left(\lambda+\sigma_1\right)\left(\delta+\lambda\right)-\left(\delta+\lambda\right)\left[\beta_1+\left(1-\pi\right)\sigma_1\delta\varepsilon_2\right]}\right\}\omega_2 v_4 > 0,$$

Therefore

$b > 0$ if $1 > \pi$, $1 > \varepsilon_2$ and $\left(\lambda+\sigma_1\right)\left(\delta+\lambda\right) > \left(\delta+\lambda\right)\left[\beta_1+\left(1-\pi\right)\sigma_1\delta\varepsilon_2\right]$.

Since $a > 0$ and $b > 0$ then the system (2.3) will exhibit a backward bifurcation otherwise it will exhibit a forward bifurcation and endemic equilibrium is locally asymptotically stable.

Numerical Sensitivity analysis

Numerical sensitivity analysis was done by computing sensitivity indices of basic reproduction number R_0 which measures initial disease transmission using the approach by (Issa ***et al.,*** 2010).

Sensitive indices measures the relative change in state variable when the parameter changes. 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 the variable is a differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivatives. The normalized forward sensitivity index of a variable R_0 that depends differentiable on parameter q is defined as

$$r_q^{R_0} = \frac{\partial R_0}{\partial q} \times \frac{q}{R_0},$$

For example, the sensitivity index of parameter value with respect to β_1 is given by

$$X_q^{R_0} = \frac{\partial R_0}{\partial \beta_2} \times \frac{\beta_1}{R_0} = 1$$

and other indices

$$X_\delta^{R_0}, \quad X_{\sigma_1}^{R_0}, \quad X_{\lambda_h}^{R_0}, \quad X_\pi^{R_0} \quad and \quad X_{\varepsilon_2}^{R_0}$$

were obtained following the same method and tabulated as follows see Tables 3.3 and 3.4.

Interpretation of Sensitivity Indices

From Table 3.3 generally shows that when parameters β_1 and π increases whilst other parameters are kept constant, the value of R_0 increases. This implies that the endemicity of the disease increases since they have positive indices. While these parameters $\varepsilon_2, \lambda, \delta, \sigma_1$ and π when each one decreases, while keeping other parameters constant they decrease the value of 0 R implying that they decrease the endemicity of the disease as they have negative indices.

Analyzing the parameters singularly, we notice that the most sensitive parameter is the contact rate of HIV only infection β_1 with the susceptible. This is followed by the progression rate to treatment σ_1. The rest follow in this decreasing order of sensitivity, human recruitment λ, tuberculosis related death γ, HIV treatment rate δ, progression rate to HIV class for AIDS infectives' accessing ARVS ε_2 and the least sensitive parameter is the progression rate to AIDS class π.

Reducing the number of contacts between susceptible individuals and HIV infected individuals will have a significant effect in the reduction of disease transmission and increase treatment awareness on the use of ARVs.

Numerical simulations and discussions

In this section, we illustrate the analytical results of the study by carrying out numerical simulations of the normalised model system (2.3) using parameter values from literature.

Other parameter values are estimated to vary within realistic means, see Table 4.1.

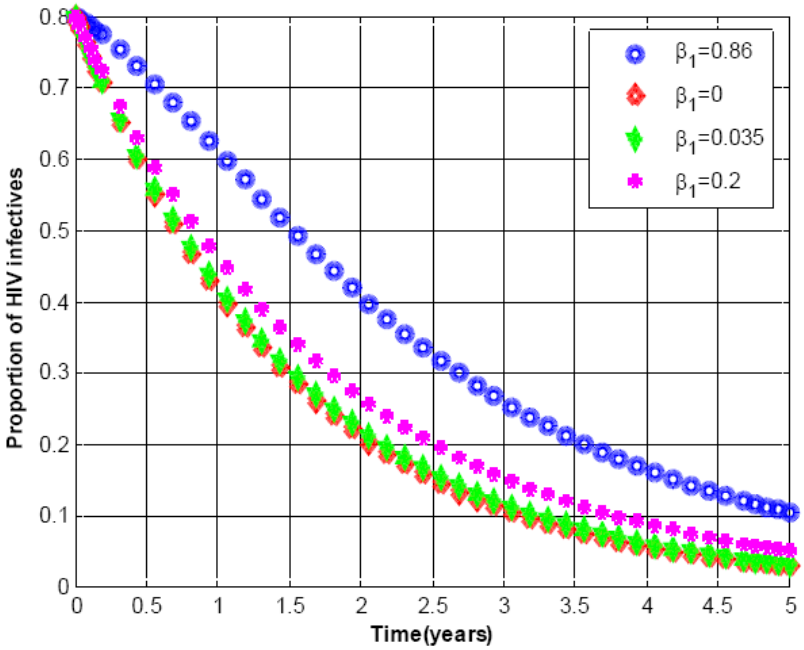

Fig 1.1: Variation of proportion of HIV infected population for different values of β_1

We observe that there is an increase in the population of HIV infectives as we vary the values of the per contact rate β_1 upwards .If no control measures are in place, a large population will be infected with HIV/AIDS.

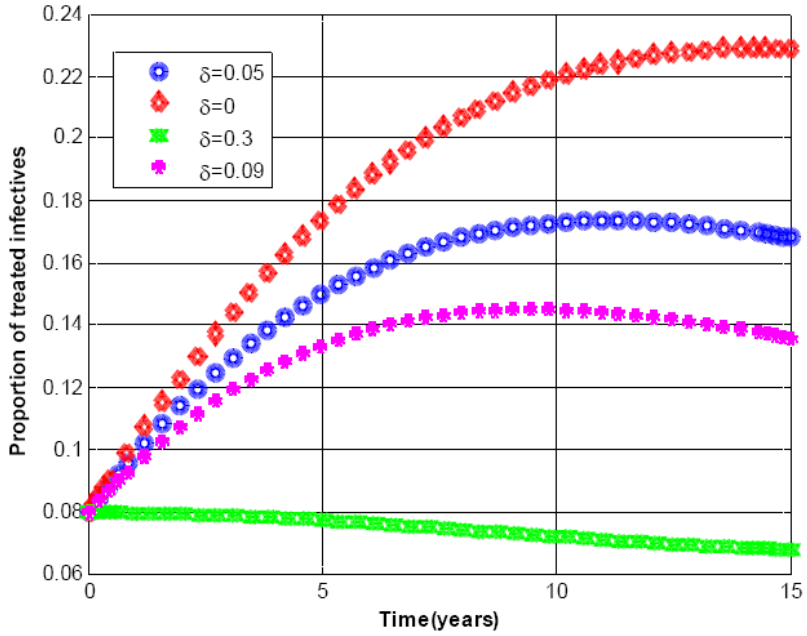

Fig 1.2: Variation of proportion of HIV treated population for different values of δ

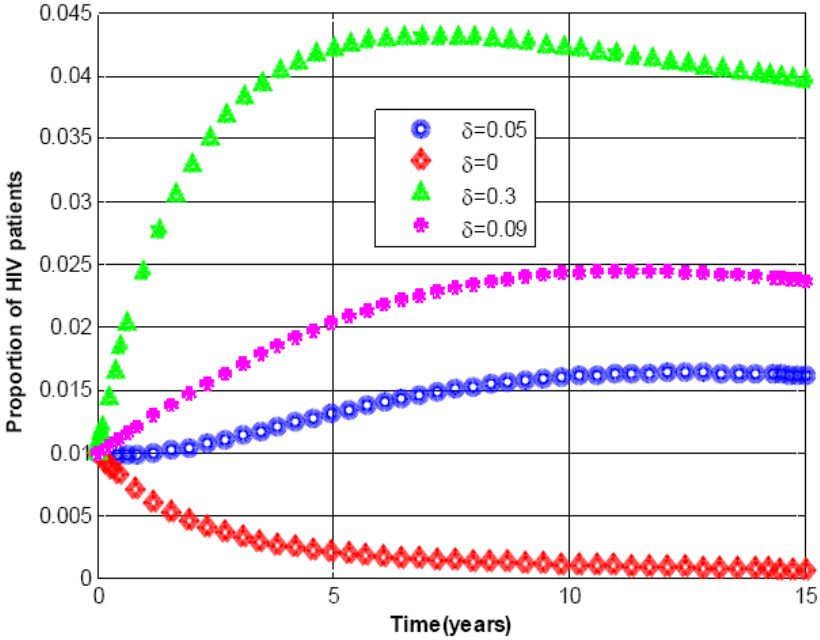

Fig 1.3: Variation of proportion of HIV patients' population for different values of δ

Fig 1.2 shows that as we increase treatment, the treatment proportion decreases due to treated individuals leave the class. In Fig 2.4 we observe that when there is no treatment, that is $\delta = 0$, the AIDS population reduces. This implies that death rate is high as there since there is no treatment However, when treatment is in progress a significant increase in the AIDS patients is observed. This also implies that patients access ARV's which prolongs their

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