IOSR Journal of Mathematics (IOSR-JM)
e-ISSN: 2278-5728, p-ISSN: 2319-765X. Volume 11, Issue 4 Ver. II (Jul - Aug. 2015), PP 72-80
www.iosrjournals.org

A Transmission Model for HIV/AIDS In The Presence Of Treatment

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Abstract: *A model for HIV transmission with treatment is formulated for a heterosexual population of varying size. The dynamics of the spread of HIV is completely determined by the Basic Reproduction Number (BRR)* R_0 *.The model exhibits two equilibria, viz. a disease-free equilibrium and the endemic equilibrium. The disease-free equilibrium is globally stable if* $R_0 \leq 1$ *and the endemic equilibrium is locally stable when* $R_0 > 1$*. Numerical analysis of the model is presented to determine the role of some key epidemiological parameters of the model.*

Keywords*: HIV/AIDS, Epidemic model, Treatment, Basic Reproduction Number, Stability*

I. Introduction

The epidemic of AIDS has been steadily spreading for the past two decades and now affects every country in the world. The number of deaths from AIDS and the number of HIV positive people continue to rise every year, despite national and international HIV prevention policies and dedicated public health care strategies. According to the World Health Organization (WHO), there were approximately 35.0 [33.2–37.2] million people living with HIV at the end of 2013 as against 33.3 million people in 2009 [1]. There were an estimated 1.5 million [1.4 – 1.7 million] deaths in 2013, according to WHO [1].

AIDS is a disease of the human immune system caused by the Human Immunodeficiency Virus. AIDS was first recognized by the US Centers for Disease Control and Prevention in 1981. In 1993, the CDC referred to AIDS as, all HIV positive people with a CD4+T cell count below 200 per millimeter of blood on 14% of all lymphocytes. HIV is transmitted from an infected to a susceptible individual through- (a) contaminated blood products or syringes, (b) sexual intercourse and (c) mother-to-child transmission during birth or through breastfeeding. Once inside the body, the HIV particles infect the white blood cells by attaching to the CD4 protein embedded in the cell membranes of the helper T-cells, macrophages and dendritic cells. In a healthy person, the normal range of T4 lymphocyte cells (helper T-cells) is usually between 600 and 1200 cells per cubic millimeter. Eventually without treatment, the CD4+ helper T-cell count declines dramatically from about 1000 cells per cubic millimeter of blood to about 200 cells, signaling the onset of AIDS.

Early surveys of mathematical and statistical methods developed for HIV/AIDS transmission are found in May and Anderson [2], Anderson and May [3], Isham [4], Dietz and Hadeler [5], Fusaro et al. [6]. May and Anderson [7] formulated a model that represented the progression from HIV positive status to AIDS, with the population being divided into various infectious stages. Hyman and Stanley [8] considered both continuous and discrete HIV/AIDS models with heterogeneity and different mixing structures that analyze the spread from high to low risk groups. Dietz and Hadeler [5] discussed a model which takes into account the phenomenon of pair formation by introducing explicitly a pairing rate and a separation rate. Lin et al. [9] analyzed a model where individuals infected with HIV progress through various phases towards AIDS. Llyod and May [10] developed a generalized model for understanding the spread of HIV through multiple populations. Doyle et al. [11] examined the dynamics of a two-population model while distinguishing between sexes. Musekwa and Nybadza [12] proposed a model for the heterogeneous transmission of HIV/AIDS in the presence of disease carriers. The model assessed the role of screening, as an intervention program in slowing the epidemic. It was found in the study, that if 80% or more of the carrier population is screened, the epidemic could be controlled.

Mathematical models have shown that Anti Retroviral Therapy (ART) can have a dramatic impact on HIV transmission, with substantial cost savings. Over the past few years, it has become clear that the benefits of HIV treatment extend far beyond saving the lives of individuals with HIV. ART plays a key role in decreasing HIV transmission. ART has been successful in reducing HIV replication in patients taking it, thereby reducing infectiousness and opportunistic infections, resulting in significantly reduced need for treatment of opportunistic infections [13]. Montaner et al. [14] developed a model in which all people would be treated after one year of infection and would not transmit HIV while receiving treatment. Bartlett [15] in his paper pointed out that ART administration could have a perverse outcome, if, it is not accompanied by an effective education program. The partially cured individuals may revert to risky activities, thereby creating new infections [16, 17]. Kimbir and Oduwole [18] proposed a mathematical model of HIV/AIDS transmission dynamics considering counselling

and ART as major means of control of infection. Cai et al. [19] investigated an HIV/AIDS model in which treatment was rendered to the infected individual in the symptomatic phase and on treatment, they moved from the symptomatic phase to the asymptomatic phase.

In this paper, an ODE model has been studied with two 'infective stages'- 'the asymptomatic and the symptomatic stages' and a 'treated AIDS stage' - and a - 'treatment failure stage'. The purpose is to investigate the effect of treatment on the long term dynamics of the disease.

II. Model Formulation

A population of size $N(t)$ is divided into six epidemiological classes. These includes- a Susceptible class $S(t)$, Infectious and Asymptomatic HIV infective class $I_1(t)$, Symptomatic individuals in the chronic HIV stage class $I_2(t)$, Full blown AIDS class $A(t)$, Treated AIDS class $A_T(t)$ and the Treatment-failure class $A_F(t)$. The total population is thus given by $N(t) = S(t) + I_1(t) + I_2(t) + A(t) + A_T(t) + A_F(t)$.

In designing the model, the following assumptions are made:

(i) A susceptible, upon infection by HIV, first enters the asymptomatic class $I_1(t)$. This stage lasts for about 10 years, but can be longer or shorter depending on the individual [20]. From the asymptomatic stage, he progresses to the symptomatic stage $I_2(t)$, which lasts for about 5-10 years and then from the symptomatic stage, he progresses to AIDS [12].

(ii) We consider only the heterosexual mode of transmission.

(iii) AIDS significantly reduces the stamina, health and physical capacities of those infected. Hence we assume that people at the AIDS stage do not contribute in the transmission process.

(iv) We assume that individuals who receive treatment are educated about risky sexual practices at the health centres and hence practice protected sex. Bunnel et al. [21] found that people who knew their HIV status were 3 times more likely to engage in protected sex than those who had not been tested.

(v) For the same reason, we assume that the treatment-failure class also does not transmit HIV infection.

(vi) Hyman and Li [22] assumed that different infected persons have differential infectivity. We assume that β_1 and β_2 are the probabilities of infection by the asymptomatic and symptomatic infectives respectively. Also we assume that $\beta_2 > \beta_1$ [12].

(vii) We assume that a fraction 'p' of individuals from A class undergo treatment and join the treated AIDS ($A_T(t)$) class at the rate pk_3 and the rest '1-p' fraction for whom treatment has failed join the treatment failure ($A_F(t)$) class at the rate $(1-p)k_3$.

Putting the above formulations and assumptions together, the AIDS model is given by the following system of ordinary differential equations:

$$\begin{aligned}
\dot{S} &= \mu N - \frac{c}{N}(\beta_1 I_1 + \beta_2 I_2)S - \mu S \\
\dot{I}_1 &= \frac{c}{N}(\beta_1 I_1 + \beta_2 I_2)S - (\mu + k_1)I_1 \\
\dot{I}_2 &= k_1 I_1 - (\mu + k_2)I_2 \\
\dot{A} &= k_2 I_2 - (\mu + k_3 + d_1)A \\
\dot{A}_T &= pk_3 A - (\mu + d_2)A_T \\
\dot{A}_F &= (1-p)k_3 A - (\mu + d_3)A_F \qquad \ldots \qquad (1)
\end{aligned}$$

where

μ is the natural birth and death rate constant,

c is the average number of sexual partners of an individual per unit time,

$c\beta_1$, $c\beta_2$ are the net disease transmission rates for the asymptomatic and symptomatic infectives respectively,

k_1 is the transfer rate constant from the asymptomatic class I_1 to the symptomatic class I_2,

k_2 is the transfer rate constant from the symptomatic class I_2 to the AIDS class *A*,

k_3 is the treatment rate,

d_1 is the disease related death rate for the individuals in the *A* class,

d_2 is the disease related death rate for the individuals in the A_T class and

d_3 is the disease related death rate for the individuals in the A_F class.

Setting $s = S/N$, $i_1 = I_1/N$, $i_2 = I_2/N$, $a = A/N$, $a_T = A_T/N$ and $a_F = A_F/N$ and noting that $s(t)+i_1(t)+i_2(t)+a(t)+a_T(t)+a_F(t)=1$, (1) can be written as:

$$\begin{aligned}
\dot{s} &= \mu - c(\beta_1 i_1 + \beta_2 i_2)s - \mu s \\
\dot{i}_1 &= c(\beta_1 i_1 + \beta_2 i_2)s - (\mu + k_1)i_1 \\
\dot{i}_2 &= k_1 i_1 - (\mu + k_2)i_2 \\
\dot{a} &= k_2 i_2 - (\mu + k_3 + d_1)a \\
\dot{a}_T &= pk_3 a - (\mu + d_2)a_T \\
\dot{a}_F &= (1-p)k_3 a - (\mu + d_3)a_F \qquad \ldots \qquad (2)
\end{aligned}$$

The region of attraction of the above system is:

$$T = \left\{(s, i_1, i_2, a, a_T, a_F) \in \Re^6 : 0 \le s + i_1 + i_2 + a + a_T + a_F \le 1\right\}$$

2.1 Basic Reproduction Number (BRR)

We use the next generation operator method (as described by van den Driessche and Watmough [23]) to find the BRR, R_0. BRR is defined as the number of secondary cases produced by a typical infectious individual during its period of infectiousness in a completely susceptible population. The associated BRR for the AIDS model denoted by R_0 is given by:

$$R_0 = \frac{c\beta_1(\mu + k_2) + c\beta_2 k_1}{(\mu + k_1)(\mu + k_2)}$$

2.2 Equilibrium States

In this section we obtain the equilibrium states by considering the R.H.S. of each of the six differential equations equal to zero in system (2), obtaining the equations:

$$\begin{aligned}
&\mu - c(\beta_1 i_1 + \beta_2 i_2)s - \mu s = 0 \\
&c(\beta_1 i_1 + \beta_2 i_2)s - (\mu + k_1)i_1 = 0 \\
&k_1 i_1 - (\mu + k_2)i_2 = 0 \\
&k_2 i_2 - (\mu + k_3 + d_1)a = 0 \\
&pk_3 a - (\mu + d_2)a_T = 0 \\
&(1-p)k_3 a - (\mu + d_3)a_F = 0 \qquad \ldots \qquad (3)
\end{aligned}$$

which after simplification gives

$$\begin{aligned}
s^* &= \frac{1}{R_0} \\
i_1^* &= \frac{\mu}{(\mu + k_1)}\left(1 - \frac{1}{R_0}\right) \\
i_2^* &= \frac{k_1}{(\mu + k_2)} i_1^* \\
a^* &= \frac{k_1 k_2}{(\mu + k_2)(\mu + k_3 + d_1)} i_1^*
\end{aligned}$$

$$a_T^* = \frac{pk_1k_2k_3}{(\mu+k_2)(\mu+d_2)(\mu+k_3+d_1)} i_1^*$$

$$a_F^* = \frac{(1-p)k_1k_2k_3}{(\mu+k_2)(\mu+d_3)(\mu+k_3+d_1)} i_1^* \quad \ldots \quad (4)$$

There exist the following two equilibria corresponding to the above system, viz.

(i) A disease-free equilibrium state $E_0(1,0,0,0,0,0)$

(ii) An endemic equilibrium state $E_1(s^*, i_1^*, i_2^*, a^*, a_T^*, a_F^*)$

III. Stability Analysis

3.1 Local stability of the equilibrium points

Theorem 1: The equilibrium E_0 is stable if $R_0 \leq 1$, otherwise if $R_0 > 1$, it is unstable and the equilibrium E_1 exists and is stable.

Proof: The Jacobian M corresponding to the system (2) is,

$$M = \begin{pmatrix} -(c\beta_1 i_1 + c\beta_2 i_2 + \mu) & -c\beta_1 s & -c\beta_2 s & 0 & 0 & 0 \\ c\beta_1 i_1 + c\beta_2 i_2 & c\beta_1 s - (\mu+k_1) & c\beta_2 s & 0 & 0 & 0 \\ 0 & k_1 & -(\mu+k_2) & 0 & 0 & 0 \\ 0 & 0 & k_2 & -(\mu+k_3+d_1) & 0 & 0 \\ 0 & 0 & 0 & pk_3 & -(\mu+d_2) & 0 \\ 0 & 0 & 0 & (1-p)k_3 & 0 & -(\mu+d_3) \end{pmatrix}$$

At the equilibrium point $E_0(1,0,0,0,0,0)$, the variational matrix M_0 is given by-

$$M_0 = \begin{pmatrix} A_{3\times3} & 0_{3\times3} \\ B_{3\times3} & C_{3\times3} \end{pmatrix}$$

where $0_{3\times3}$ is a zero matrix of order 3×3 and $A_{3\times3}$, $B_{3\times3}$, $C_{3\times3}$ are block matrices given by-

$$A_{3\times3} = \begin{pmatrix} -\mu & -c\beta_1 & -c\beta_2 \\ 0 & c\beta_1 - (\mu+k_1) & c\beta_2 \\ 0 & k_1 & -(\mu+k_2) \end{pmatrix}$$

$$B_{3\times3} = \begin{pmatrix} 0 & 0 & k_2 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad \text{and}$$

$$C_{3\times3} = \begin{pmatrix} -(\mu+k_3+d_1) & 0 & 0 \\ pk_3 & -(\mu+d_2) & 0 \\ (1-p)k_3 & 0 & -(\mu+d_3) \end{pmatrix}$$

Since the upper right block is a zero matrix, the eigen values of M_0 can be found by calculating the eigen values of the block matrices *A* and *C*. For stability of E_0, the eigen values of the matrices should be negative.

The characteristic equation to the matrix *A* is given by:

$$(\mu+\lambda)(\lambda^2 + a_1\lambda + a_2) = 0 \quad \ldots \quad (5)$$

where

$$a_1 = 2\mu + k_1 + k_2 - c\beta_1$$

$$a_2 = (\mu + k_1 - c\beta_1)(\mu + k_2) - ck_1\beta_2$$

One root of the equation (5) is $-\mu$ and the other two roots are given to be the roots of the equation $\lambda^2 + a_1\lambda + a_2 = 0$.

Using the Routh-Hurwitz criteria, we see that the equation $\lambda^2 + a_1\lambda + a_2 = 0$ has roots with negative real parts if a_1 and $a_2 > 0$, which is satisfied if $R_0 \le 1$.

The characteristic equation to the matrix C is given by:

$$(\mu + d_1 + k_3 + \lambda)(\mu + d_2 + \lambda)(\mu + d_3 + \lambda) = 0 \qquad \dots \qquad (6)$$

The above equation has roots which are all negative.

Thus $E_0(1,0,0,0,0,0)$ is locally asymptotically stable if $R_0 \le 1$. Further it is unstable if $R_0 > 1$ and in this case, the second equilibrium point E_1 exists.

At the equilibrium point $E_1(s^*, i_1^*, i_2^*, a^*, a_T^*, a_F^*)$, the Jacobian M_1 is given by:

$$M_1 = \begin{pmatrix} P_{3\times3} & 0_{3\times3} \\ Q_{3\times3} & R_{3\times3} \end{pmatrix}$$

where $0_{3\times3}$ is a zero matrix of order 3×3 and $P_{3\times3}$, $Q_{3\times3}$, $R_{3\times3}$ are block matrices given by:

$$P_{3\times3} = \begin{pmatrix} -(c\beta_1 i_1^* + c\beta_2 i_2^* + \mu) & -c\beta_1 s^* & -c\beta_2 s^* \\ c\beta_1 i_1^* + c\beta_2 i_2^* & c\beta_1 s^* - (\mu + k_1) & c\beta_2 s^* \\ 0 & k_1 & -(\mu + k_2) \end{pmatrix};$$

$$Q_{3\times3} = \begin{pmatrix} 0 & 0 & k_2 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad \text{and}$$

$$R_{3\times3} = \begin{pmatrix} -(\mu + k_3 + d_1) & 0 & 0 \\ pk_3 & -(\mu + d_2) & 0 \\ (1-p)k_3 & 0 & -(\mu + d_3) \end{pmatrix}$$

Since the upper right block is a zero matrix, the eigen values of M_1 can be found by calculating the eigen values of the block matrices *P* and *R*. For stability of E_1, the eigen values of the matrices should be negative.

The characteristic equation to the matrix *P* is given by:

$$\lambda^3 + b_1\lambda + b_2\lambda + b_3 = 0 \qquad \dots \qquad (7)$$

where

$$b_1 = 3\mu + c\beta_1 i_1^* + c\beta_2 i_2^* + k_1 + k_2 + c\beta_1 s^*$$
$$b_2 = (2\mu + k_1 + k_2)(c\beta_1 i_1^* + c\beta_2 i_2^* + \mu) + c\beta_1 \mu s^*$$
$$b_3 = (\mu + k_1)(\mu + k_2)(c\beta_1 i_1^* + c\beta_2 i_2^*)$$

The equilibrium point E_1 will be stable if it satisfies the Routh-Hurwitz criteria, which for the present case includes b_1, b_2, $b_3 > 0$ and $b_1 b_2 - b_3 > 0$. We observe that b_1, b_2 and $b_3 > 0$. Also under the condition $R_0 > 1$, $b_1 b_2 - b_3 > 0$.

The characteristic equation corresponding to the matrix *R* is the same as matrix *C*, which has all negative roots. Thus $E_1(s^*, i_1^*, i_2^*, a^*, a_T^*, a_F^*)$ is locally asymptotically stable.

3.2 Global stability of the disease-free equilibrium point

Theorem 2: The equilibrium $E_0(1,0,0,0,0,0)$ is globally stable in *T* if $R_0 \le 1$, along with

$$\max\left\{\frac{c\beta_1}{\mu}, \frac{c\beta_2}{\mu}\right\} < \frac{1}{2},$$

and it is unstable if $R_0 > 1$.

Proof: To prove the global stability of E_0, we construct a Lyapunov function of the form:

$$L = \frac{1}{2}(s-1)^2 + (i_1 + i_2 + a + a_T + a_F)$$

Differentiating we have,

$$\dot{L} = (s-1)\dot{s} + (\dot{i}_1 + \dot{i}_2 + \dot{a} + \dot{a}_T + \dot{a}_F)$$

Putting values of $\dot{s}$, $\dot{i}_1$, $\dot{i}_2$, $\dot{a}$, $\dot{a}_T$ and $\dot{a}_F$ from (2) into the above equation and simplifying we obtain,

$$\dot{L} = -\mu(s-1)^2 - (c\beta_1 i_1 + c\beta_2 i_2)s^2 + \Theta$$

where

$$\Theta = 2(c\beta_1 i_1 + c\beta_2 i_2)s - \mu(i_1 + i_2 + a + a_T + a_F) - (d_1 a + d_2 a_T + d_3 a_F)$$

Now we are to prove that $\Theta < 0$. After simplification of the above equation, we get,

$$\Theta = \left(\frac{2c\beta_1}{\mu} - 1\right)\mu i_1 + \left(\frac{2c\beta_2}{\mu} - 1\right)\mu i_2 - \mu(a + a_T + a_F) - (d_1 a + d_2 a_T + d_3 a_F)$$

Hence $\Theta < 0$ if $\max\left\{\frac{c\beta_1}{\mu}, \frac{c\beta_2}{\mu}\right\} < \frac{1}{2}$.

Thus $\dot{L} < 0$ and hence the DFE E_0 is globally asymptotically stable.

IV. Numerical Results And Discussion

In the last section, the stability of the equilibrium points was examined. In this section, the results are demonstrated numerically.

The system (2) is solved using the fourth order Runge- Kutta method. A time unit of months has been used to study the system. The parameter values that have been used are as follows:

The birth and natural death rate parameter (μ) is taken as 0.02 per year. The average number of sexual partners (*c*) is taken as 3. The disease transmission rates for the asymptomatic and symptomatic infectives, which are β_1 and β_2 are taken as 0.25 and 0.325 respectively ($\beta_1 < \beta_2$). The progression rate k_1 from I_1 to I_2 is 0.2 and the progression rate k_2 from I_2 to A is 0.4. Treatment rate k_3 is taken as 0.2. The disease related death rates for the *A*, A_T and A_F classes which are d_1, d_2 and d_3 are taken to be 1, 0.05 and 0.15 respectively. The fraction of people, who have responded positively to treatment, which is *p* is taken as 0.4, so that 1- *p* is 0.6.

The initial values used for the $S(t)$, $I_1(t)$, $I_2(t)$, $A(t)$, $A_T(t)$ and $A_F(t)$ classes at time *t* = 0 are $S(0)$ = 9000, $I_1(0)$ = 4000, $I_2(0)$ = 3000, $A(0)$ = 1000, $A_T(0)$ = 600, $A_F(0)$ = 400 and $N(0)$ = 18000. Hence $s(0)$ = 0.5, $i_1(0)$ = 0.222, $i_2(0)$ = 0.167, $a(0)$ = 0.056, $a_T(0)$ = 0.033, $a_F(0)$ = 0.022.

Mathematical models often include parameters, for which their actual values are not known precisely. Some of the present model parameters have been estimated and others have been taken from Anderson and May [20] and Musekwa and Nyabadza [12]. The parameter values are suited for Africa and some parts of the developing world, where the major mode of HIV transmission is the heterosexual mode.

V. Figures

Fig. 1 illustrates the behavior and relationship of the Basic Reproduction Number (BRR) R_0 as the number of sexual partners *c* increases. It is clear from the figure that with an increase in *c*, R_0 increases, leading to an endemic state of the disease.

In Fig. 2, the AIDS population *a* is plotted against time for various values of the number of sexual partners *c*. It is found that, as people increase their number of sexual partners, it also increases the number of AIDS cases *a*.

In Fig. 3, the Treated AIDS class a_T is plotted against time for various values of the treatment rate k_3. We observe that as k_3 increases there is a rise in a_T. And subsequently there is a decline in the AIDS population *a*. This is depicted in Fig. 4.

We explore various values of the fraction *p* in Fig. 5 and Fig. 6. We take *p* = 0.3, 0.5, 0.7, 0.9. It is observed that as *p* (the fraction of people, who have responded positively to treatment) increases, there is a rise in the Treated AIDS population a_T (Fig. 5) and there is a decline in the Treatment-failure population a_F (Fig. 6).

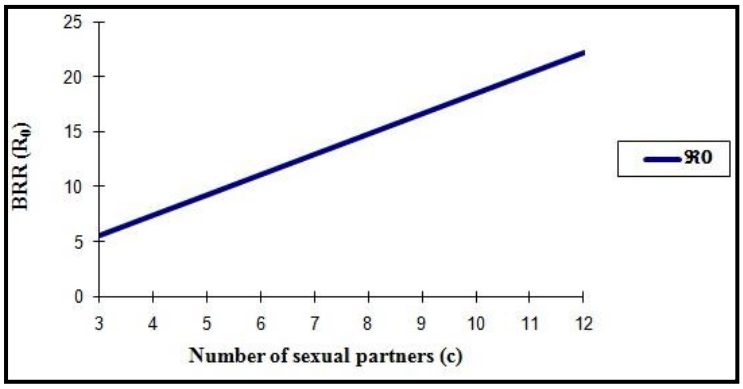

Fig. 1: Variation of the Basic Reproduction Number (BRR) R_0 for various values of the number of sexual partners *c*.

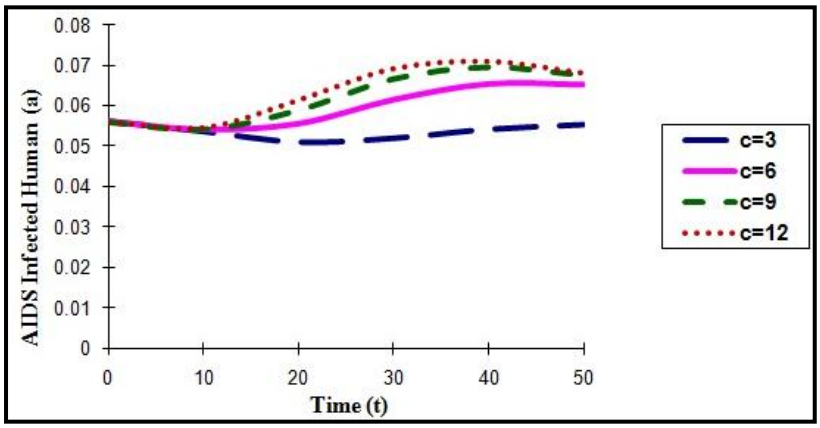

Fig. 2: Variation in the AIDS class *a* with time for various values of the number of sexual partners *c*.

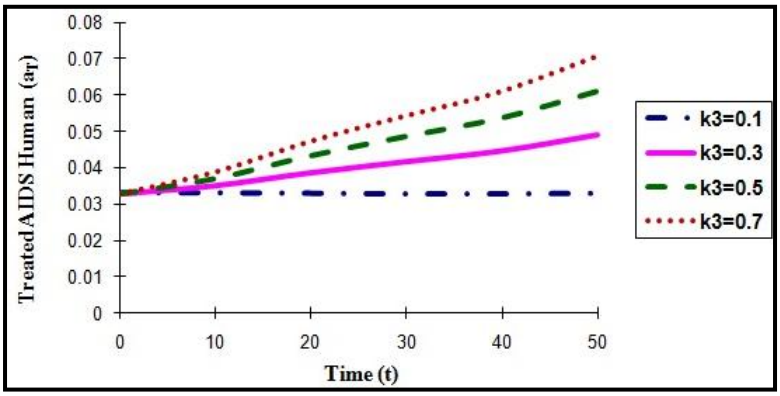

Fig. 3: Variation in the Treated AIDS class a_T with time for various values of the treatment rate k_3.

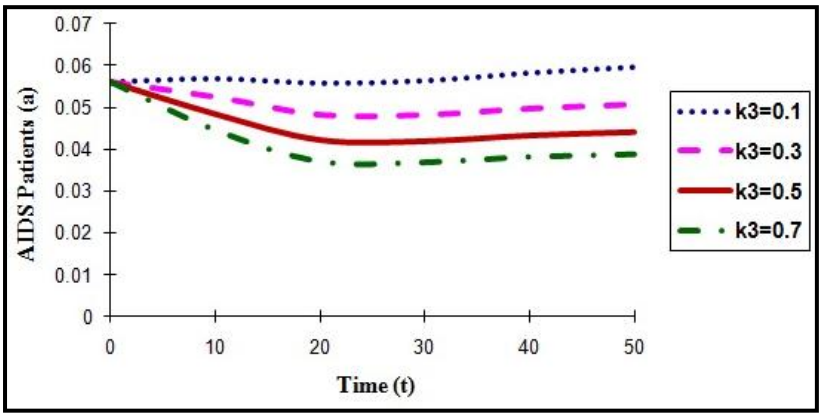

Fig. 4: Variation in the AIDS class *a* with time for various values of the treatment rate k_3.

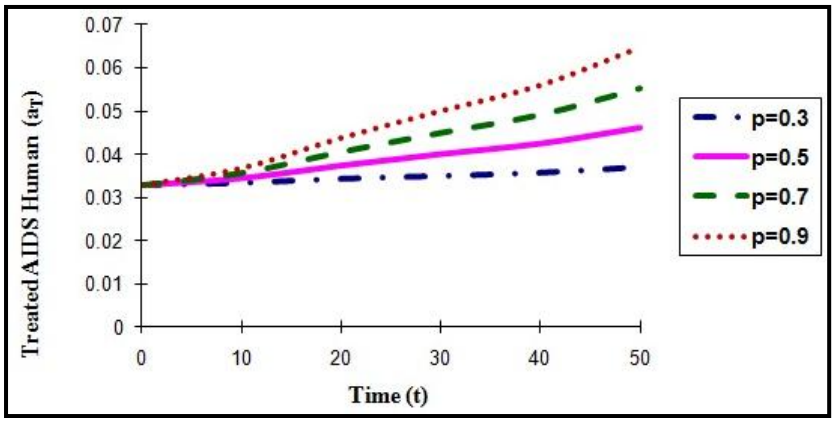

Fig. 5: Variation in the Treated AIDS class a_T with time for various values of *p*.

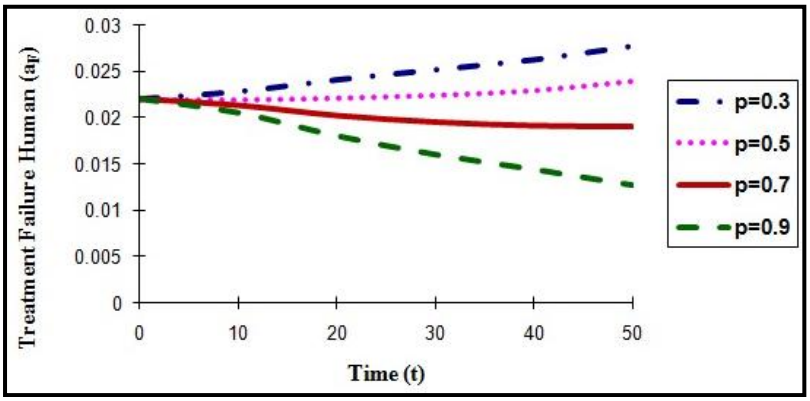

Fig. 6: Variation in the Treatment-failure class a_F with time for various values of *p*.

VI. Conclusion

In this paper, a model for heterosexual transmission of HIV/AIDS has been formulated and analyzed. The model has been analyzed both analytically and numerically. The results of the study provide some insights on the benefits of treatment which would prolong the patients' survival time and also at the same time bring some behavioral changes which reduce their transmission probability.

From an epidemiological perspective, the Basic Reproduction Number (BRR) R_0 is an important indicator of the initial course of HIV. When $R_0 > 1$, an epidemic prevails. R_0 is dependent on the parameter *c* (the number of sexual partners). It is found in the study that, as people indulge in risky sexual practices with newly acquired sexual partners, it gives an endemic state to the disease (Fig. 1). The model also studies the co-relation of the Treated AIDS class and the Treatment-failure class with *p* (the fraction of people, who have responded positively to treatment).

AIDS stigma exists around the world in a variety of ways, including ostracism, rejection, discrimination and avoidance of HIV infected people, compulsory HIV testing without prior consent or protection of confidentiality, violence against HIV infectives and quarantine of HIV infected individuals. Stigma-related violence prevents many people from seeking HIV testing or securing treatment, resulting in the spread of HIV. Hence the rate at which individuals join the class of those under treatment, is of significant importance. Higher values of the treatment are a sign of decreased stigma. Testing for HIV is valuable for those who are HIV infected, because at the health centers, where from, they receive treatment, they are also educated about risky sexual practices and hence brings about a behavioral change, so as to, not to spread the virus, thus lowering the incidence and prevalence of HIV.

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