

Impact of Heterosexuality and Homosexuality on the transmission and dynamics of HIV/AIDS

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Abstract: In this paper more general deterministic sex – structured HIV/AIDS model is proposed. The effects of Homosexuality (MSM) and Heterosexuality on the dynamics of the HIV/AIDS are studied. The epidemic threshold is known as a reproduction number. In this study it is classified as basic and partial reproduction numbers. The basic reproduction number R_0 is constructed considering both the heterosexuality and homosexuality. The partial reproduction number is constructed by considering only one of the heterosexuality and homosexuality. The basic reproduction number is an algebraic composition of the partial reproduction numbers. It is found that both heterosexuality and homosexuality have their own influences on the dynamics of the epidemic. The partial reproduction number with larger values influences more the overall dynamics of the epidemic. If at least one of the partial reproduction numbers is greater than unity then the disease will exist in the population. The disease free and endemic equilibriums are determined and their stabilities are investigated. The disease free equilibrium point is locally and globally asymptotically stable when $R_0 < 1$. The positive endemic equilibrium point is locally asymptotically stable when $R_0 > 1$. The endemic equilibrium can be made globally stable for a selected set of values. Numerical simulation of the model is carried out to assess the impact of homosexuality and heterosexuality on the dynamics of HIV/AIDS disease. The result showed that as the rate of infection and the probability of disease transmission of homosexuality and heterosexuality increases then the male infective class and the female infective class also increase.

Keywords: HIV/AIDS; Homosexual (MSM); Heterosexual; Disease free equilibrium; local stability; reproduction number; partial reproduction number; sex structured model

I. Introduction

Acquired Immunodeficiency Syndrome or AIDS is a disease caused by the Human Immunodeficiency Virus or HIV. It is a sexually transmitted disease and initially started spreading worldwide during early 1980s. As of today, there has not been any cure for the disease. As a result, prevention and public awareness are the only ways for controlling HIV infection. HIV/AIDS is one of the most destructive diseases humankind has ever faced. The deadly disease leads to profound social, economic and public health consequences. It has become a full – blown pandemic and been affecting all parts of the world since more than three decades.

The number of HIV affected people in the world in 2010 was estimated to be 34 million; including 2.7 million new infections occurred during the same year, i.e., 7400 cases per day. The number of AIDS deaths occurred during that year was about 1.8 million [1], [14].

The spread of much infectious disease occurs in a diverse population so that it is desirable to consider heterogeneity. We can distinguish human population into different kinds of groups, viz., male and female, young and old, high sexual activity and low sexual activity, heterosexual and homosexual, and other subdivisions. These groupings can be used in the formulation of epidemiological modeling in order to improve its predictive and explanatory power and its applicability. If any of the epidemiological characteristics is gender dependent then groups of male and female is necessary.

In general, the epidemiological characteristics of sexually transmitted disease are different from male to female. The probability of transmission of Gonorrhoea from male to female is more than from female to male [2], [13]. Females are at risk of HIV infection more than males per contact [3], [11]. Initially, men were more exposed to the infection than women as a result of homosexual intercourse. But, the difference in the numbers of infected men and women has gradually narrowed as heterosexual transmission has become more common during early 1990s [4], [9].

The study of HIV/AIDS transmission and dynamics has been of great interest to both applied mathematicians and biologists due to its universal threat to humanity. Mathematical models play an important role in the study of the transmission dynamics of HIV/AIDS, and in some sense, sex structured models give better compatibility with reality. Many models available in the literature represent dynamics of disease by

system of nonlinear differential equations without considering the sex structure. However, inclusion of sex structure in such models makes them more realistic

Here we develop a general model and study the role of homosexuality and heterosexuality on the dynamics of the disease by considering sex structured model. Heterosexual contact is the primary mode of HIV transmission and it accounts for the largest number of infections [5], [15]. Lot of research has been done in this area. The common practice of the researchers is to divide humans in to heterosexuals and homosexuals groups. But, in this model the whole population is considered and grouped in to males and females. The aim of this investigation is to verify the influence of intra and inter sexual intercourse of these groups on the dynamics of the disease. The dynamics of these models are best studied in terms of their equilibria and stability.

In this model, individuals are grouped based on the gender. The population is split in to two groups, viz., males and females.

Here we developed a general model by combining the two modes of transmission i.e., Homosexual and Heterosexual. Further, we found the disease free and endemic equilibrium points and examined them for stability. We also compared the two modes of transmission and determined which of them is more responsible for high level of transmission of the disease. Also we identified the influence of partial reproduction numbers on the basic reproduction number. The results are presented lucidly and discussed clearly.

II. Homosexual or MSM Transmission Model

Here we consider male sex with male, MSM, and see its affect on the dynamics of the disease by considering its partial reproduction number. The flow chart of the disease transmission model due to homosexual is given as

Let s_f , I_f and A_f represent the number of susceptible, infective and AIDS cases of female population respectively at time t . Similarly, s_m , I_m and A_m represent the number of susceptible, infective and AIDS cases of male population respectively at time t

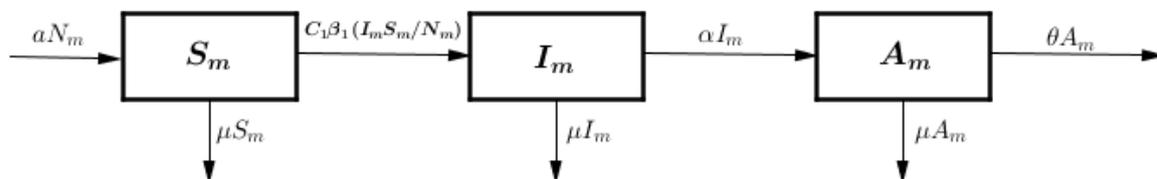


Figure -1: Flow chart of the MSM Homosexual model

The model for transmission between MSM Homosexuals can be described by the following system of differential equations

$$ds_m/dt = aN_m - C_1 \beta_1 (I_m S_m/N_m) - \mu S_m \quad (1)$$

$$dI_m/dt = C_1 \beta_1 (I_m S_m/N_m) - \alpha I_m - \mu I_m \quad (2)$$

$$dA_m/dt = \alpha I_m - \theta A_m - \mu A_m \quad (3)$$

Here in above, C_1 represents the rate at which *infected males* infect *susceptible males*; β_1 represents the probability with which the disease transmits per one contact from *infectious male* to *susceptible male*. The infected male will go to A_m compartment after confirmation of full AIDS disease at a rate of α . The A_m compartment will die with AIDS disease at a rate of θ and μ represents the natural mortality rate for all compartments. The total male population N_m is the sum of susceptible S_m , infected I_m and AIDS patients A_m that is, $N_m = S_m + I_m + A_m$.

2.1 Disease free equilibrium point

The disease free equilibrium of the model, (1) to (3), is obtained by setting $(ds_m/dt) = (dI_m/dt) = (dA_m/dt) = 0$. Further at the disease free equilibrium point there are neither infective people nor AIDS patients. That is, $I_m = A_m = 0$. Up on substituting these, (1) implies that $aN_m - \mu S = 0$ or equivalently $S = aN_m/\mu$. Thus, the disease free equilibrium of the model is given by $E_0 = (aN_m/\mu, 0, 0)$.

2.2 Calculating the partial reproduction number

The reproduction number is defined as the average number of secondary cases produced by a typical infected male during his infectious period [6], [12]. Let R_{mm} denotes the partial reproduction number of Homosexual model. It is simple to compute that $R_{mm} = [C_1\beta_1/(\alpha + \mu)]$.

III. Heterosexual Transmission Model

The spread of the virus is through sexual contact between opposite sexes. Usually one of the male or female is infectious. The compartmental structure and flow directions of male and female in the model are given as follows:

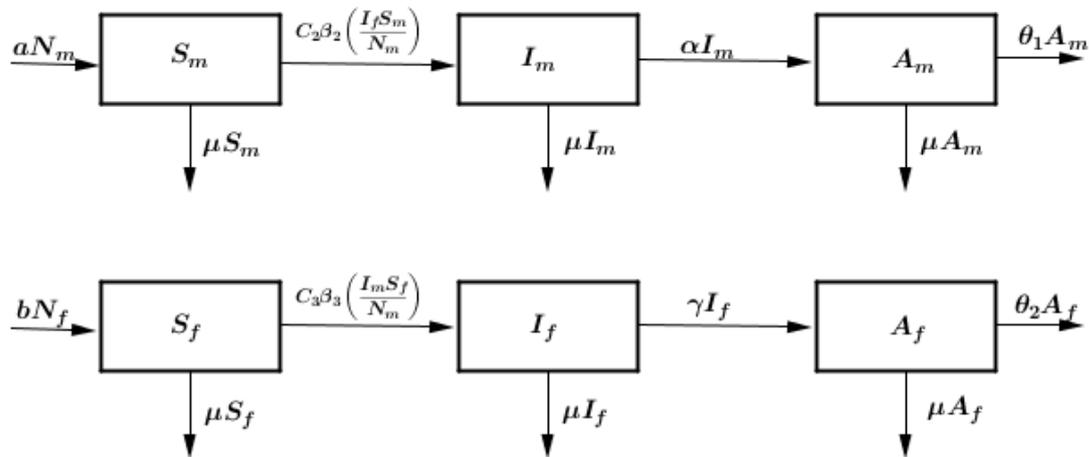


Figure -2 Flow chart of the Heterosexual model

In this model we focus exclusively on heterosexual activity. Therefore, we consider the sexual activity between the two opposite genders. That is, contact of susceptible male with infectious female and contact of susceptible female with infectious male are considered. The heterosexual transmission model can be described by the following system of differential equations

$$ds_m/dt = aN_m - C_2\beta_2 (I_f S_m/N_m) - \mu s_m \quad (4)$$

$$dI_m/dt = C_2\beta_2 (I_f S_m/N_m) - \alpha I_m - \mu I_m \quad (5)$$

$$dA_m/dt = \alpha I_m - \theta_1 A_m - \mu A_m \quad (6)$$

$$ds_f/dt = bN_f - C_3\beta_3 (I_m S_f/N_f) - \mu s_f \quad (7)$$

$$dI_f/dt = C_3\beta_3 (I_m S_f/N_f) - \gamma I_f - \mu I_f \quad (8)$$

$$dA_f/dt = \gamma I_f - \theta_2 A_f - \mu A_f \quad (9)$$

Here in (4) to (9), C_2 represents the rate at which infected females infect susceptible males; β_2 represents the probability with which the disease transmits per one contact from infectious female to susceptible male; C_3 represents the rate at which infected males infect susceptible females; β_3 represents the probability with which the disease transmits per one contact from infectious male to susceptible female. The infected male will go to A_m compartment after confirmation of full AIDS disease at a rate of α . The infected female will go to A_m compartment after confirmation of full AIDS disease at a rate of γ .

Population of the compartments A_m and A_f will die with disease at a rate of θ_1 and θ_2 respectively. Natural mortality rate in all the compartments is represented by μ . The total male population N_m is the sum of susceptible S_m , infected I_m and AIDS patients A_m . That is, $N_m = S_m + I_m + A_m$. Similarly, $N_f = S_f + I_f + A_f$.

3.1 Calculation of reproduction number for Heterosexually Transmission Model

The reproduction number for the whole population is the average number of secondary infection caused by a single infection in typical infectee introduced in to a susceptible population. Let R'_0 denotes the partial reproduction number of Heterosexual model. R'_0 can be calculated by using *simultaneous equation approach*, that is the typical infectee is some theoretical average of a male and female individual. If x represents the probability that the typical infectee is a male then $1 - x$ will represent the probability that the typical infectee is a female, R_0 is the maximum value and satisfies the following matrix equation [1]

$$\begin{pmatrix} R_{mm} & R_{mf} \\ R_{fm} & R_{ff} \end{pmatrix} \begin{pmatrix} x \\ 1-x \end{pmatrix} = R'_0 \begin{pmatrix} x \\ 1-x \end{pmatrix} \quad (10)$$

In (10), R_{mm} is the number of secondary infections in male group members generated by male group members, R_{mf} is the number of secondary infections in male group members generated by female group members, R_{fm} is the number of secondary infections in female group members generated by male group members and R_{ff} is the number of secondary infections in female group members generated by female group members.

Since the present model considers only Heterosexual transmission, there is no possibility for direct transmission from male to male or from female to female. Hence, $R_{mm} = R_{ff} = 0$. Further, R_{mf} and R_{fm} may differ because of variation in sexual behavior, transmission probability and duration of infection between male and females. That is the numbers R_{mf} and R_{fm} need not be equal. The matrix equation (10) becomes

$$\begin{pmatrix} 0 & R_{mf} \\ R_{fm} & 0 \end{pmatrix} \begin{pmatrix} x \\ 1-x \end{pmatrix} = R_0' \begin{pmatrix} x \\ 1-x \end{pmatrix} \quad (11)$$

The matrix equation (11) can be solved for R_0 by eliminating the unknown term x from the two implicit equations $R_{mf}(1-x) = R_0'x$ and $R_{fm}x = R_0'(1-x)$. After simple algebraic computation and rearrangement the equation for R_0 of two – gender hetero-sexual mixing model of HIV/AIDS is obtained as follows:

$$R_0' = \sqrt{R_{fm} R_{mf}} \quad (12)$$

The basic reproduction number is equal to the geometric mean of secondary infections of each group. Recall that here we have only two groups i.e., males and females.

IV. Homosexuals and Heterosexual Transmission Model or General Model

Here we include heterosexual and homosexual activities and develop general mathematical model. To achieve this purpose the sexual activities of (i) susceptible male with infected female (ii) susceptible female with infected male (iii) susceptible male with infected male are considered.

However, the sexual activity between females (FSF), lesbian sex, is not included since its contribution to HIV transmission is neglected. The females may be of any type viz., susceptible, infected or AIDS patients. Transmission of the disease from a female to female through lesbian sex is very low [3], [8].

4.1 Flow of people and Description of parameters

Let S_f , I_f and A_f respectively denote the sizes of female – susceptible, female – Infective, female – AIDS cases. Since the total female population N_f is divided into susceptible, infective, AIDS compartments, we must have $s_f + I_f + A_f = N_f$. Similarly, the total male population N_m is divided into male – susceptible S_m , male – Infective I_m , male – AIDS A_m compartments and thus $s_m + I_m + A_m = N_m$.

In the present study the susceptible compartment is split into male susceptible S_m and female susceptible S_f compartments. People will join these two compartments with rates of aN_m and bN_f respectively. The people of male – susceptible S_m class are likely to become infected through sexual contact with the people of I_m and I_f classes. Thus, people from S_m will flow to I_m with a rate of λ_1 . Similarly, people of female – susceptible class S_f are likely to become infected through sexual contact with only the people of I_m class. Thus, people from S_f will flow to I_f with a rate of λ_2 . After confirmation of full AIDS disease people from I_m and I_f compartments will flow to A_m and A_f compartments respectively with the rates of α and γ . People of each compartment are assumed to die with natural reasons and leave at a rate of μ . People of A_m and A_f compartments are assumed to die with AIDS disease at the rates of θ_1 and θ_2 respectively.

The parameter β_1 is the probability of disease transmission from infectious male to susceptible male per one contact. The parameter β_2 is the probability of disease transmission from infectious female to susceptible male per one contact.

The parameter β_3 is the probability of disease transmission from infectious male to susceptible female per one contact. Infectious male infects susceptible male with a rate of C_1 . Infectious female infects susceptible male with a rate of C_2 . Infectious male infects susceptible female with a rate of C_3 . Male-to-female HIV transmission during vaginal intercourse is significantly more likely than female-to-male transmission [16], [17]. The general model with the inclusion of transmission among homosexuals and heterosexuals is described in the form of a flow chart as follows:

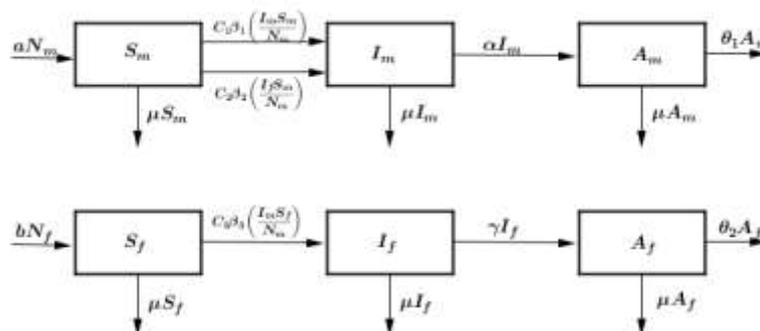


Figure-3 Flow chart of the Heterosexual and Homosexual model

The model looks more complicated since it considers both transmission of the disease among heterosexuals and homosexuals. The assumption is made based on the fact that the infective of one sex group spreads the infection to the susceptible of the same or other sex group.

4.2 Model Assumptions

The present general model is developed based on the following assumptions:

1. There is no direct transmission of the disease from female to female. Since the risk of transmission through female-to-female sex is very low.
2. The HIV can only be transmitted by the sexual intercourse with infective people.
3. The sexually Active human population is divided into six compartments.
4. The full-blown AIDS class is sexually inactive.
5. Recruitment rates of susceptible males and females are proportional to their respective population sizes i.e., aN_m and bN_f .
6. The population sizes of both genders are equal $N_m = N_f$.
7. Age structure is ignored.
8. The mortality rates are the same for both sexes.
9. Population sizes of both the infected genders are the same $I_m = I_f$.
10. All the AIDS patients will die either naturally or due to the disease.
11. Transmission through hetero-sex and homo-sex (MSM) is alone considered. Other means of transmission are considered negligible and thus excluded.
12. The natural mortality rates are assumed to be the same for all the compartments.

4.3 Governing equations of the General Model

The system of ordinary differential equations that describe the dynamics of AIDS population due to homo-sex and hetero-sex is constructed and is given as follows:

$$ds_m/dt = aN_m - \lambda_1 S_m - \mu s_m \quad (13)$$

$$ds_f/dt = bN_f - \lambda_2 S_f - \mu s_f \quad (14)$$

$$dI_m/dt = \lambda_1 S_m - \alpha I_m - \mu I_m \quad (15)$$

$$dI_f/dt = \lambda_2 S_f - \gamma I_f - \mu I_f \quad (16)$$

$$dA_m/dt = \alpha I_m - \theta_1 A_m - \mu A_m \quad (17)$$

$$dA_f/dt = \gamma I_f - \theta_2 A_f - \mu A_f \quad (18)$$

In the system (13) to (18) we used notations: $\lambda_1 = [(C_1\beta_1 I_m/N_m) + (C_2\beta_2 I_f/N_f)]$ and $\lambda_2 = (C_3\beta_3 I_m/N_m)$.

4.4 Positivity of solutions

The general model equations (13) to (18) are to be epidemiologically meaningful and well posed; we need to prove that all the state variables are non-negative.

Theorem 1: If $S_m(0) > 0, S_f(0) > 0, I_m(0) \geq 0, I_f(0) \geq 0, A_m(0) \geq 0, A_f(0) \geq 0$ then the solutions $\{S_m(t), S_f(t), I_m(t), I_f(t), A_m(t), A_f(t)\}$ of the system of equations (13) to (18) are non-negative for all $t > 0$

Proof: To show the positivity of the solution of the dynamical system comprising the equations (13) to (18), we have to consider and verify each differential equation and show that their solution is positive.

First let us consider the differential equation (13) of the dynamical system

$$ds_m/dt = aN_m - \lambda_1 S_m - \mu s_m \quad (19)$$

Also, (19) can be written as $(dS_m/dt) + [q(t) + \mu]S_m = aN_m(t)$ where $q(t) = [(C_1\beta_1 I_m + C_2\beta_2 I_f)/N_m(t)]$. This is a first order linear ordinary differential equation and can be solved to obtain a particular solution as:

$$S_m(t) = S_m(0)e^{-Q(t)+Q(0)-\mu t} + \int_0^t aN_m(s)e^{(Q(s)-Q(t))+\mu(s-t)} ds \quad (20)$$

Since, $S_m(0) > 0$, and the exponential function always positive. It is clear from (20) that $S_m(t)$ is positive. Secondly let us consider the differential equation (14) and that can be rewritten as

$$(dS_f/dt) + [(C_3\beta_3 I_m)/N_f(t) + \mu]S_f = bN_f(t) \quad (21)$$

Which is equivalent to

$$(dS_f/dt) + [q(t) + \mu]S_f = bN_f(t) \quad (22)$$

Here in (22), $q(t) = [(C_3\beta_3 I_m)/N_f(t)]$. This is a first order linear ordinary differential equation and can be solved to obtain a particular solution as:

$$S_f(t) = S_f(0)e^{-Q(t)+Q(0)-\mu t} + \int_0^t bN_f(s)e^{(Q(s)-Q(t))+\mu(s-t)} ds \quad (23)$$

It is clear from the solution that $S_f(t)$ is positive since $S_f(0) > 0$, and the exponential function is always positive. Thirdly, let us consider the differential equation (15) and that can be expressed as $dI_m/dt + (\alpha + \mu)I_m = \lambda_1 S_m$. This is a first order linear ordinary differential equation and can be solved to obtain a particular solution as $I_m(t) = I_m(0)e^{-(\alpha+\mu)t} + \int_0^t \lambda_1 S_m(s)e^{-(\alpha+\mu)t} ds$. From this solution we see that $I_m(t)$ is also nonnegative.

Fourthly, let us consider the differential equation (16) and that can be expressed as $dI_f/dt + (\gamma + \mu)I_f = \lambda_2 S_f$. This is a first order linear ordinary differential equation and can be solved to obtain a particular solution as $I_f(t) = I_f(0)e^{-(\gamma+\mu)t} + \int_0^t \lambda_2 S_f(s)e^{-(\gamma+\mu)t} ds$. From this solution we see that $I_f(t)$ is also nonnegative. Fifthly, let us consider the differential equation (17) and that can be expressed as $dA_m/dt + (\theta_1 + \mu)A_m = \alpha I_m$. This is a first order linear ordinary differential equation and can be solved to obtain a particular solution as $A_m(t) = A_m(0)e^{-(\theta_1+\mu)t} + \int_0^t \alpha I_m(s)e^{-(\theta_1+\mu)t} ds$. From this solution we see that $A_m(t)$ is also nonnegative. Finally, let us consider the differential equation (18) and that can be expressed as $dA_f/dt + (\theta_2 + \mu)A_f = \alpha I_f$. This is a first order linear ordinary differential equation and can be solved to obtain a particular solution as $A_f(t) = A_f(0)e^{-(\theta_2+\mu)t} + \int_0^t \alpha I_f(s)e^{-(\theta_2+\mu)t} ds$. From this solution we see that $A_f(t)$ is also nonnegative.

Boundedness of the solution region

The total population size $N(t)$ given by $N(t) = S_m(t) + S_f(t) + I_m(t) + I_f(t) + A_m(t) + A_f(t)$ implies using the equations (13) to (18) that $dN/dt = (dS_m/dt) + (dS_f/dt) + (dI_m/dt) + (dI_f/dt) + (dA_m/dt) + (dA_f/dt) = aN_m + bN_f - \mu N - \theta_1 A_m - \theta_2 A_f$ (24)

From (24) we obtain $dN/dt \leq aN_m + bN_f - \mu N$. The latter differential inequality has a solution of the form $N(t) \leq [(aN_m + bN_f/\mu) - N_0 e^{-\mu t}]$ or equivalently it implies that $0 < N(t) \leq (aN_m + bN_f/\mu)$ as $t \rightarrow \infty$. Therefore the solutions of system are bounded.

4.5 Disease free equilibrium point

The disease free equilibrium of the model (1) is obtained by setting $(ds_m/dt) = (ds_f/dt) = (dI_m/dt) = (dI_f/dt) = (dA_m/dt) = (dA_f/dt) = 0$ and solving. Note that at the disease free equilibrium point there are neither infective people nor aids patients i.e., $I_m = I_f = A_m = A_f = 0$. Thus the disease free equilibrium DFE of the general model is given by $E_0 = (aN_m/\mu, bN_f/\mu, 0, 0, 0, 0)$. We now investigate the local stability of the disease free equilibrium point E_0

4.6 Computation of Reproduction number

Reproduction number for the general model (13) to (18) can be computed using two different approaches viz., (i) simultaneous equation approach and (ii) next generation matrix method.

4.6.1 Computation of Reproduction number using next generation matrix method

The reproduction number can also be computed using the next generation matrix method. It is defined as $R_0 = \rho(FV^{-1})$. Here $\rho(FV^{-1})$ represents the spectral radius of the matrix FV^{-1} . Also the matrix is given by $FV^{-1} = [(\partial/\partial x_j)F_i(x_0)][(\partial/\partial x_j)V_i(x_0)]^{-1}$. Here F_i is the rate of appearance of new infections in the compartment i ; V_i is the resultant number of individuals leaving from the compartment i i.e., number of transfers minus recruits; and E_0 is the disease free equilibrium point. Consequently,

$$\begin{bmatrix} F_{I1} \\ F_{I2} \end{bmatrix} = \begin{bmatrix} f_1 \\ f_2 \end{bmatrix} = \begin{bmatrix} [(c_1\beta_1 I_m + c_2\beta_2 I_f)/N_m]S_m \\ [(c_3\beta_3 I_m)/N_f]S_f \end{bmatrix} \tag{25}$$

By linearization approach, the associated matrix F at the disease free equilibrium point E_0 is given by

$$F = \begin{bmatrix} \frac{\partial f_1}{\partial I_m} & \frac{\partial f_1}{\partial I_f} \\ \frac{\partial f_2}{\partial I_m} & \frac{\partial f_2}{\partial I_f} \end{bmatrix} \text{ at } E_0 = \begin{bmatrix} c_1\beta_1 & c_2\beta_2 \\ c_3\beta_3 & 0 \end{bmatrix} \text{ and } \begin{bmatrix} v_{I1} \\ v_{I2} \end{bmatrix} = \begin{bmatrix} v_1 \\ v_2 \end{bmatrix} = \begin{bmatrix} \mu I_m + \alpha I_m \\ \mu I_f + \gamma I_f \end{bmatrix}$$

Again by linearization, we get

$$V = \begin{bmatrix} \frac{\partial v_1}{\partial I_m} & \frac{\partial v_1}{\partial I_f} \\ \frac{\partial v_2}{\partial I_m} & \frac{\partial v_2}{\partial I_f} \end{bmatrix}_{E_0} = \begin{bmatrix} \mu + \alpha & 0 \\ 0 & \mu + \gamma \end{bmatrix} \text{ and } V^{-1} = \begin{bmatrix} \frac{1}{\mu + \alpha} & 0 \\ 0 & \frac{1}{\mu + \gamma} \end{bmatrix}. \text{ Thus, } FV^{-1} = \begin{bmatrix} \frac{c_1\beta_1}{\mu + \alpha} & \frac{c_2\beta_2}{\mu + \gamma} \\ \frac{c_3\beta_3}{\mu + \alpha} & 0 \end{bmatrix}$$

The Eigen values for FV^{-1} are found by solving the characteristic equation $|FV^{-1} - \lambda I| = 0$ which is equivalent to $\lambda^2 - a\lambda - bc = 0$. Here $a = [c_1\beta_1/(\mu + \alpha)]$, $b = [c_3\beta_3/(\mu + \alpha)]$ and $c = [c_2\beta_2/(\mu + \gamma)]$. Thus, the two eigenvalues are $\lambda_1 = \{[a + \sqrt{a^2 + 4bc}]/2\}$ and $\lambda_2 = \{[a - \sqrt{a^2 + 4bc}]/2\}$. But, $R_0 = \rho(FV^{-1}) = \text{Max} \{ \lambda_1, \lambda_2 \}$ and hence we obtain

$$R_0 = (1/2) \left[\left(\frac{c_1\beta_1}{\mu+\alpha} \right) + \sqrt{\left(\frac{c_1\beta_1}{\mu+\alpha} \right)^2 + 4 \left(\frac{c_3\beta_3}{\mu+\alpha} \right) \left(\frac{c_2\beta_2}{\mu+\gamma} \right)} \right] \quad (26)$$

4.6.2 Computation of Reproduction number using Simultaneous equation approach

Following simultaneous equation approach we here calculate the reproduction number R_0 . Maximum value of R_0 satisfies the following matrix equation:

$$\begin{bmatrix} R_{mm} & R_{mf} \\ R_{fm} & R_{ff} \end{bmatrix} \begin{bmatrix} x \\ 1-x \end{bmatrix} = R_0 \begin{bmatrix} x \\ 1-x \end{bmatrix} \quad (27)$$

Here R_{mf} , R_{fm} and R_{mm} are referred to as *partial reproductive numbers*. Since the possibility for direct transmission of the disease from female to female is very low, hence $R_{ff} = 0$. Thus the matrix equation (24) takes the form as

$$\begin{bmatrix} R_{mm} & R_{mf} \\ R_{fm} & 0 \end{bmatrix} \begin{bmatrix} x \\ 1-x \end{bmatrix} = R_0 \begin{bmatrix} x \\ 1-x \end{bmatrix} \quad (28)$$

The two implicit algebraic equations of (20) can be solved for R_0 by eliminating the unknown variable x as

$$\begin{aligned} R_{mm} x + R_{mf} (1-x) &= R_0 x \\ R_{fm} x &= R_0 (1-x) \end{aligned} \quad (29)$$

After simple algebraic computation and rearrangement of (21) R_0 is obtained as

$$R_0 = (1/2) [R_{mm} + \sqrt{R_{mm}^2 + 4R_{fm}R_{mf}}] \quad (30)$$

On comparing equations (23) and (27), it can be observed that $R_{mm} = [c_1\beta_1/(\mu + \alpha)]$, $R_{mf} = [c_2\beta_2/(\mu + \gamma)]$ and $R_{fm} = [c_3\beta_3/(\mu + \alpha)]$. If $R_{MM} = 0$ then the reproduction number of (27) will reduce to that of the production number of hetero sexual mixing model (12). The expression (27) suggests that heterosexuality and homosexuality contributes for growth of the epidemic. Further, as long as at least one of the partial reproductive numbers R_{mf} , R_{fm} and R_{mm} is greater than unity, the disease remains in the population.

Let the numbers of secondary infected male R_m and female R_f are defined by $R_m = R_{mf} + R_{mm}$ and $R_f = R_{fm}$ respectively. The assumption $R_{mf} \geq R_{fm}$ leads to the fact that the number of secondary infected males R_m is more than that of secondary infected females R_f . It can be concluded that more males are infected with HIV than females. Hence the contribution of males influences the dynamics of HIV more than females.

4.7 Stability Analysis of the general Model

In this section, the equilibrium points for the general model are identified and their stability analysis is made. The system exhibits two types of equilibrium point viz., disease free equilibrium points and endemic equilibrium points.

4.7.1 Disease Free Equilibrium Point

Theorem – 2: The disease free equilibrium point E_0 of the system of ordinary differential equations (13)-(18) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: The AIDS patients are sexually inactive and hence they do not propagate the disease either to susceptible or to infected people. That is, s_m and I_m do not depend on A_m similarly s_f and I_f do not depend on A_f [7][10]. Therefore, without loss of generality this system of equation can be written as subsystem of four equations { (13) – (16) }.

Using the scaling parameters $s'_m = s_m/N_m$, $s'_f = s_f/N_f$, $I'_m = I_m/N_m$, $I'_f = I_f/N_f$ the subsystem of differential equations { (13) – (16) } will take the form

$$ds'_m/dt = a - (C_1\beta_1 I'_m + C_2\beta_2 I'_f) s'_m - \mu s'_m \quad (31)$$

$$ds'_f/dt = b - C_3\beta_3 I'_m s'_f - \mu s'_f \quad (32)$$

$$dI'_m/dt = (C_1\beta_1 I'_m + C_2\beta_2 I'_f) s'_m - (\alpha + \mu) I'_m \quad (33)$$

$$dI'_f/dt = C_3\beta_3 I'_m s'_f - (\gamma + \mu) I'_f \quad (34)$$

The Jacobian matrix associated with the subsystem of equations{ (28) – (31) } at the disease free equilibrium point E_0 is

$$J(E_0) = \begin{bmatrix} -\mu & 0 & -C_1\beta_1 & -C_2\beta_2 \\ 0 & -\mu & -C_3\beta_3 & 0 \\ 0 & 0 & C_1\beta_1 - (\alpha + \mu) & C_2\beta_2 \\ 0 & 0 & C_3\beta_3 & -(\gamma + \mu) \end{bmatrix} \quad (35)$$

It is enough to show that, if $R_0 < 1$ holds then two inequalities $\text{Tr} J(E_0) < 0$ and $\det [J(E_0)] > 0$ do also hold. However, setting of $R_0 < 1$ and after few manipulations it results two relations as $[c_1\beta_1/(\mu + \alpha)] < 1$ and $[c_3\beta_3/(\mu + \alpha)][c_2\beta_2/(\mu + \gamma)] < 1$. Of the couple, the former relation

implies $\text{Tr}[J(E_0)] < 0$. Also setting of $R_0 < 1$ leads to the inequality condition that $(\alpha + \varpi)(\gamma + \varpi) - (\gamma + \varpi)C_1\beta_1 - C_2\beta_2C_3\beta_3 > 0$ and is equivalent to put it as $\text{Det}[J(E_0)] > 0$. From these two observations namely $\text{Tr}[J(E_0)] < 0$ and $\text{Det}[J(E_0)] > 0$ it can be concluded that the disease free equilibrium point is stable as long as $R_0 < 1$, otherwise unstable.

Further, it is a stable node since the discriminate $D = [\text{Tr} J(E_0)]^2 - 4\text{Det}[J(E_0)]$ is a positive quantity.

4.7.2 Endemic Equilibrium Point

Just similar to the DFE, here also consider the subsystem of four equations $\{(13) - (16)\}$. At the endemic equilibrium point $E^* = (S_m^*, S_f^*, I_m^*, I_f^*)$ the disease persists or exists. The local stability of endemic equilibrium point is stated and proved in the form of

Local Stability of Endemic Equilibrium Point

Theorem – 3 The positive endemic equilibrium point E^* of the system of equations (13) – (16) is locally asymptotically stable, if $R_0 > 1$.

Proof: Using the scaled subsystem of differential equations $\{(28) - (31)\}$, Which is equivalent to:

$$\begin{aligned} \frac{ds_m}{dt} &= a - (k_1I_m' + k_2I_f')s_m' - \mu s_m' \\ \frac{ds_f'}{dt} &= b - k_3I_m's_f' - \mu s_f' \\ \frac{dI_m'}{dt} &= (k_1I_m' + k_2I_f')s_m' - (\alpha + \mu)I_m' \\ \frac{dI_f'}{dt} &= k_3I_m's_f' - (\gamma + \mu)I_f' \end{aligned}$$

Here we let $k_1 = C_1\beta_1, k_2 = C_2\beta_2$ and $k_3 = C_3\beta_3$. We set right hand side of each equation in the subsystem to zero and express each dependent variable in terms of parameters at the equilibrium point. Thus, we obtain

$$\begin{aligned} S_m^* &= [(\alpha + \mu)/(k_1 + k_2)], & I_m^* &= \{[a/(\alpha + \varpi)] - [\mu(\alpha + \mu)/(k_1 + k_2)]\} \\ S_f^* &= \frac{b(\alpha + \varpi)(k_1 + k_2)}{\varpi(\alpha + \varpi)(k_1 + k_2) + ak_3(k_1 + k_2) - k_3\mu(\alpha + \varpi)(\alpha + \varpi)} & I_f^* &= \frac{1}{\gamma + \varpi} \left[\frac{b\varpi(\alpha + \varpi)(k_1 + k_2) + ak_3(k_1 + k_2) + b\varpi k_3(\alpha + \varpi)}{\varpi(\alpha + \varpi)(k_1 + k_2) + ak_3(k_1 + k_2) - k_3\mu(\alpha + \varpi)} \right] \end{aligned}$$

Since $R_0 > 1$ the parameters I_m^*, S_f^* and I_f^* are all non negative terms.

The Jacobian matrix of the subsystem at the endemic equilibrium point takes the form as

$$J(E^*) = \begin{bmatrix} -(k_1I_m^* + k_2I_f^* + \mu) & 0 & -k_1S_m^* & -k_2S_m^* \\ 0 & -(k_3I_m^* + \mu) & -k_3S_f^* & 0 \\ k_1I_m^* + k_2I_f^* & 0 & [k_1S_m^* - (\alpha + \mu)] & k_2S_m^* \\ 0 & k_3I_m^* & k_3S_f^* & -(\gamma + \mu) \end{bmatrix}$$

We have to show that, if $R_0 > 1$ then $\text{Tr}[J(E^*)] < 0$ and $\text{Det}[J(E^*)] > 0$. It can be verified that $k_1S_m^* - (\alpha + \varpi)$ is equal to $\{k_1[(\alpha + \varpi)/(k_1 + k_2)] - (\alpha + \varpi)\}$ and is a negative quantity. Therefore, $\text{Tr}[J(E^*)]$ has also a negative value. Next let us show $\text{Det}[J(E^*)] > 0$ as follows: Consider, let $p = k_1I_m^* + k_2I_f^*$ and $q = k_3I_m^*$. For the Jacobian matrix

$$J(E^*) = \begin{bmatrix} -(p + \mu) & 0 & -k_1S_m^* & -k_2S_m^* \\ 0 & -(q + \mu) & -k_3S_f^* & 0 \\ p & 0 & [k_1S_m^* - (\alpha + \mu)] & k_2S_m^* \\ 0 & q & k_3S_f^* & -(\gamma + \mu) \end{bmatrix}$$

The determinant can be computed as

$$\text{Det}[J(E^*)] = [(p + \mu)][(q + \mu)]\{(\alpha + \varpi) - k_1S_m^*\}(\gamma + \varpi) - k_2k_3S_m^*S_f^* + (p + \mu)k_3S_f^*k_2k_3S_m^*I_m^* + p k_1S_m^*\{(\gamma + \mu)\} + p k_2S_m^*\{[(q + \mu)]k_3S_f^* - k_3S_f^*q\}.$$

Up on considering $[(q + \mu)]k_3S_f^* - k_3S_f^*q$ as a positive quantity or equivalently saying that $[(k_3I_m^* + \mu)] - k_3I_m^* > 0$ it can be observed that $\text{Det} J(E^*) > 0$, if $[(\gamma + \mu)((\alpha + \mu) - k_1S_m^*)] - [k_2k_3S_m^*S_f^*] > 0$. Hence, it can be concluded that $\text{Det} J(E^*) > 0$, if $R_0 > 1$.

Theorem – 4: The disease free equilibrium point E_0 of the system of ordinary differential equations (1) is globally stable if $R_0 < 1$.

Proof: The proof is based on using a comparison theorem. The equation of the infected components can be written in terms of

$$\begin{bmatrix} \frac{dI_1}{dt} \\ \frac{dI_2}{dt} \end{bmatrix} = (F - V) \begin{bmatrix} I_1 \\ I_2 \end{bmatrix} - \begin{bmatrix} (C_1\beta_1I_m + C_2\beta_2I_f) - \left(\frac{C_1\beta_1I_m}{N_m} + \frac{C_2\beta_2I_f}{N_f}\right)S_M \\ C_3\beta_3I_m - \left(\frac{C_3\beta_3I_m}{N_m}\right)S_f \end{bmatrix} \quad (36)$$

It follows that

$$\begin{bmatrix} \frac{dI_1}{dt} \\ \frac{dI_2}{dt} \end{bmatrix} \leq (F - V) \begin{bmatrix} I_1 \\ I_2 \end{bmatrix} \tag{37}$$

All the eigenvalues of the matrix $[F - V]$ have negative real parts. It follows that the system of linear differential inequalities (27) is stable whenever $R_0 < 1$. Also we will have $I_1 \rightarrow 0$ and $I_2 \rightarrow 0$ as $t \rightarrow \infty$. Therefore by comparison theorem, it follows that $(I_1, I_2) \rightarrow (0, 0)$ and the remaining equations of model (1) give us solutions as $E_0 = (aN_m/\mu, bN_f/\mu, 0, 0)$. Thus, $(S_m, S_f, I_m, I_f) \rightarrow E_0$ and also as $t \rightarrow \infty$ the condition $R_0 < 1$ implies that the disease free equilibrium point E_0 is globally and asymptotically stable [7].

4.8 Numerical Simulation

To illustrate the dynamical behavior of the model system (1) is solved using ODE 45. The estimated parametric values are chosen to be: $a = 0.02, b = 0.0001, \alpha = 0.03, \lambda_1 = 0.03, \lambda_2 = 0.03, \mu = 0.003, \gamma = 0.03, \theta_1 = 0.9, \theta_2 = 0.9, \beta_1 = 0.9, \beta_2 = 0.9, \beta_3 = 0.99, C_1 = 0.9, C_2 = 0.9$ and $C_3 = 0.99$ together with four different initial values given below:

- (i) $S_m(0) = 9000, I_m(0) = 11000, A_m(0) = 1500, S_f(0) = 9100, I_f(0) = 11000, A_f(0) = 800.$
- (ii) $S_m(0) = 8009, I_m(0) = 7800, A_m(0) = 800, S_f(0) = 8009, I_f(0) = 7800, A_f(0) = 870.$
- (iii) $S_m(0) = 8000, I_m(0) = 10000, A_m(0) = 1000, S_f(0) = 9000, I_f(0) = 1000, A_f(0) = 600.$
- (iv) $S_m(0) = 9200, I_m(0) = 10000, A_m(0) = 500, S_f(0) = 9200, I_f(0) = 10000, A_f(0) = 300.$

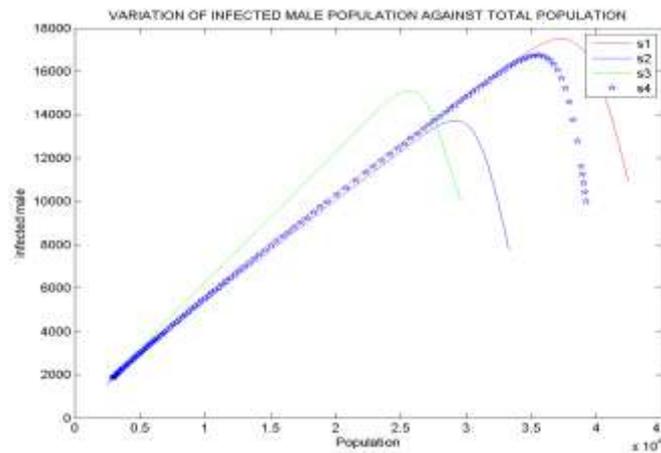


Figure 4: Variation of infected male population against total population

Figure 4 displays the plot of infected male populations against the total population. For that purpose the set of initial values given in four cases (i) to (iv) are used. This figure demonstrates that, for the given set of initial values, the solution curves tend to the Endemic equilibrium point E^* . Hence, the numerical simulation indicates that system (1) is globally asymptotically stable about E^* .

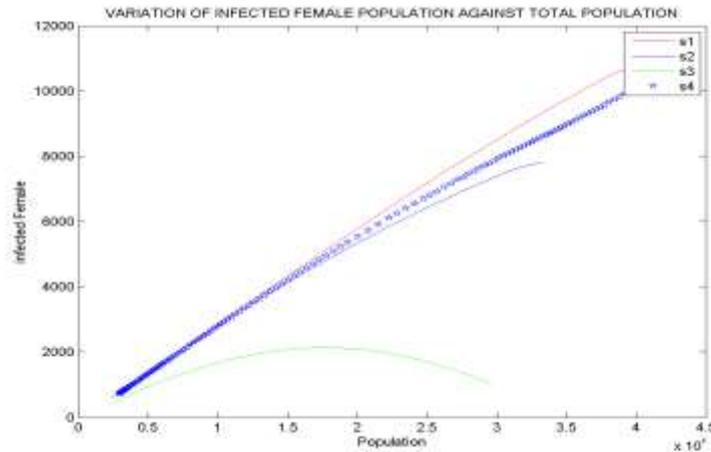


Figure 5: Variation of infected female population against total population

Figure 5 displays the plot of infected female populations against the total population. For that purpose the set of initial values given in four cases (i) to (iv) are used. This figure demonstrate that, for the given set of initial values, the solution curves tend to the Endemic equilibrium point E^* . Hence, the numerical simulation indicates that system (1) is globally asymptotically stable about E^*

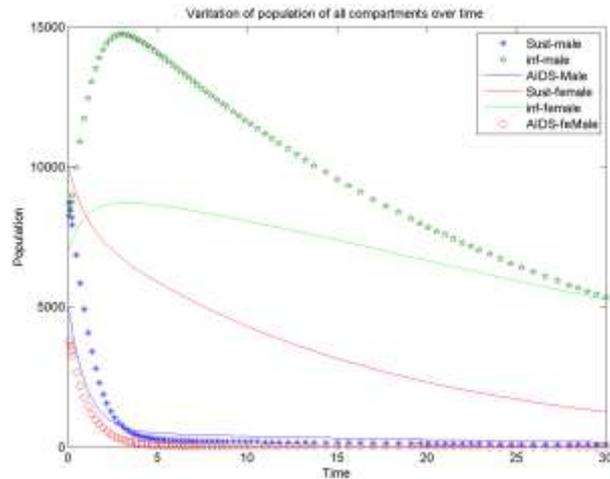


Figure 6: Variation of population of all compartments over time for the given parametric values

Figure-6 Shows the distribution of proportion of population with time with different classes. It is seen that for those estimated values the proportion of both male and female susceptible population decrease continuously resulting in the increase of the proportion of both infective male and infective female initially but in this case the infective male class increases faster than the infective female class this is due to the contribution of both homosexuality and heterosexuality but then both classes decreases as all infectives subsequently develop to full blown AIDS and die due to natural death and AIDS.

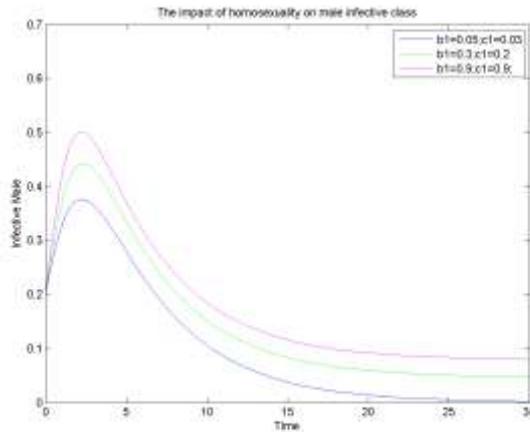


Figure 7: The impact of Homosexuality on Male infective class.

Figure-7 Shows the impact of homosexuality on male infective population. It is seen that for different values of the coefficient parameters of homosexuality C_1, β_1 . As the rate of infection and the probability of disease transmission of homosexuality increases the male infective population also increases.

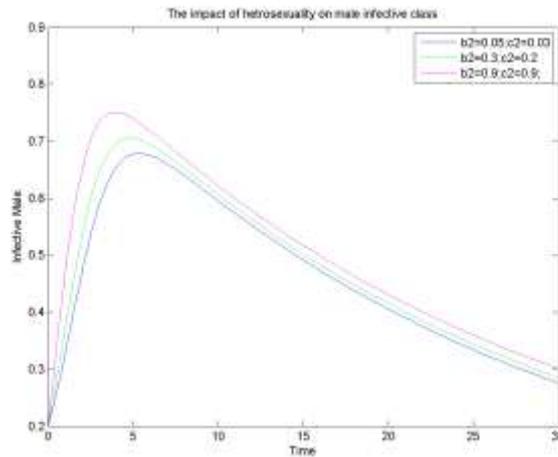


Figure 8: The impact of Heterosexuality over Male infective class

Figure-8 Shows the impact of Heterosexuality on male infective population. It is seen that for different values of the parameters C_2, β_2 . As the rate of infection and the probability of disease transmission of heterosexuality increases the male infective population also increases.

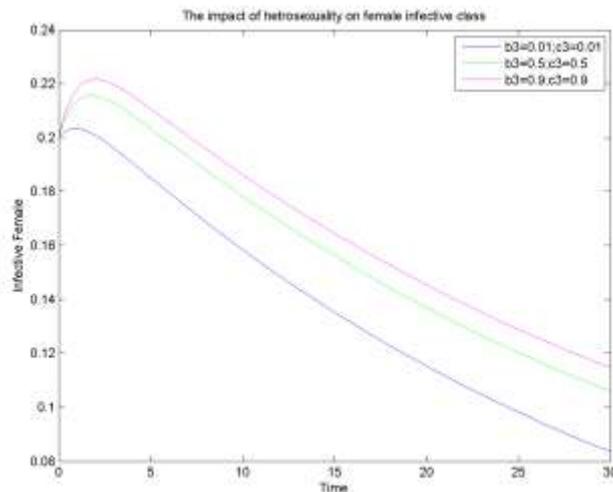


Figure 9: The impact of Heterosexuality on female infective class.

Figure-9 Shows the impact of heterosexuality on female infective population. It is seen that for different values of the parameters (C_3, β_3) . As the rate of infection and the probability of disease transmission of heterosexuality increases the female infective population also increases.

V. Conclusions

In this paper, we proposed a general model by including hetero-sex and homo-sex. Also, verified its effect on the dynamics of HIV/AIDS. A system of non – linear differential equations was formulated to represent the model.

The stability analysis shows that (i) the disease free equilibrium point E_0 is globally asymptotically stable if $R_0 < 1$ and (ii) the positive endemic equilibrium point E^* is locally asymptotically stable if $R_0 > 1$. It is pointed out that both hetero-sex and homo-sex influence the growth of HIV/AIDS epidemic. Further, it is shown that the partial reproductive numbers R_{mm} , R_{mf} and R_{fm} influence the overall dynamics of the epidemic. It is observed that as long as at least one of the partial reproduction numbers is greater than unity the disease will exist in the population.

Results from Numerical Simulation shows that, as the rate of infection and the probability of disease transmission of homosexuality and heterosexuality increases then both the male infective class and the female infective class also increase.

It is also shown that the male infective class at the beginning increases faster. This fact is evident and expected, due to the contribution of both heterosexuality and homosexuality. Furthermore the endemic equilibrium is stable for the given set of numerical values and the population sizes of all the compartments decrease to the equilibrium point over time.

References

- [1]. Emilia Vynnycky, Richard White, An introduction to infectious diseases modeling; Oxford university press, 2010 ISBN 978-0-19-856-576-5
- [2]. European Study Group on Heterosexual; Transmission of HIV Comparison of female to male and male to female transmission of HIV in 563 stable couples; BMJ Vol(304):1992
- [3]. Hellen Namawejje, Livingstone S. Luboobi, Dmitrykuznetsov and Eric Wobudeya. Bifurcation Analysis of model for the effect of vaccination on the transmission dynamics of Rotavirus Disease. Asian Journal of Mathematics and applications Vol(4):2015
- [4]. Ying-Hen Hsieh. A two-sex model for treatment of HIV/AIDS and behavior change in a population of varying size. IMA Journal of Mathematics Applied in medicine and Biology vol. (13):151-173, 1996
- [5]. Issa Shabani and S. Massawe, Modeling the effect of screening on the spread of HIV infection in a population with variable inflow of infective immigrants. Scientific research and essays, Vol. 20, 4397-4405, 2011
- [6]. Diekmann O., Heesterbeek J. A and Metz J. A., on the definition and computation of R_0 in the model for infectious disease in heterogeneous population. *Journal of mathematical Biology*, 28 (1990), 365-382.
- [7]. Sa'ndor Kovacs, Dynamic s of an HIV/AIDS model – The effect of time delay. Science Direct applied mathematics and Computation, 188(2007), 1597-1609.
- [8]. Guido David and Fenina Mae Gomez, Sexually –based compartmental model of spread of HIV in the Philippines, Institute of Mathematics university of Philippines Article Vol. 7, 362-369, 2014.
- [9]. Mary T. Dunne, B. A., H. Dip. Ed., A Modified Mathematical Model for HIV Transmission, AIDS and Intervention Strategies in Ireland. Msc Thesis 1995.
- [10]. Tadele Tesfa, Purnachandra Rao Koya and Temesgen Tibebu. Modeling the Combined Effect of Vertical Transmission and Variable Inflow of Infective Immigrants on the Dynamics of HIV/AIDS. American J. of App. Mathematics. vol.(4):11-19, 2016.
- [11]. James M. Hyman and E. Annstanely. A risk-based heterosexual model for the AIDS epidemic with biased sexual partner selection; Modeling the Aids epidemic(1994)
- [12]. Anderson R. M. and May R. M., (1991). Infectious Diseases of Human, *Dynamics and Control*. Oxford: Oxford university press.
- [13]. Onoja Matthew Akpa and B. A. Oyejola, Modeling the transmission dynamics of HIV/AIDS epidemics, an introduction and review, J. Infect Dev Ctries V(10), 597-608.
- [14]. Egbetade, S.A and Babalola, F.M .A Stability Analysis for a Two-Sex Mathematical Model of HIV. The international Journal of Engineering and Science(IJES) vol.(3):20-23, 2013
- [15]. Eti Dwi Wiraningsih, Folashade Agosto, Lina Aryati. A Stability Analysis of Rabies Model with Vaccination Effect and Culling in Dogs. Applied Mathematical Sc. Vol.(9):3805-3817, 2015
- [16]. Fekadu Tadege Kobe, Purnachandra Rao Koya. Controlling the Spread of Malaria Disease Using Intervention Strategies. Journal of Multidisciplinary Engineering Science and Technology (JMEST). Vol. 2, Issue 5, May 2015, pp 1068 – 74. ISSN: 3159 – 0040.
- [17]. <http://www.jmest.org/wp-content/uploads/JMESTN42350745.pdf>
- [18]. Dancho Desaleng, Purnachandra Rao Koya. The Role of Polluted Air and Population Density in the Spread of Mycobacterium Tuberculosis Disease. Journal of Multidisciplinary Engineering Science and Technology (JMEST). Vol. 2, Issue 5, May – 2015, Pp 1212 – 20. ISSN: 3159 – 0040. <http://www.jmest.org/wp-content/uploads/JMESTN42350782.pdf>