Advances in Applied Mathematical Biosciences.
ISSN 2248-9983 Volume 7, Number 1 (2016), pp. 41-50

http://www.irphouse.com

# A Model for HIV Transmission among Intravenous Drug Users

**V. Vijayalakshmi**
*Assistant Professor of Mathematics,*
*Bharathiyar College of Engineering and Technology,*
*Karaikal, India.*

**Abstract**

The qualitative analysis of a model for HIV transmission among intravenous drug users is studied. Let us show global stability of the infection free equilibrium when$R_0 \leq 1$. For the case $R_0 > 1$, the stability of the endemic equilibrium is discuss*ed.*

***Keywords*:** Epidemiology, susceptible, homosexual population, HIVinfections, Endemic.

## 1. INTRODUCTION

In recent years, modeling the HIV transmission dynamics has become a major research topic in epidemiology. A compartmental model is presented for the spread of HIV in a homosexual population divided into subgroups by degree of sexual activity. The model includes constant recruitment rates for the susceptibles in the subgroups. It incorporates the long infectious period of HIV – infected individuals and allows one to vary infectiousness over the infectious period.

## 2. MODEL OF SPREAD IN A HOMOSEXUAL POPULATION

Let us present a model of HIV spread in a homosexual population that incorporates what we believe are three major factors in HIV spread. One factor is the effect of the

pattern of contacts between groups that differ in sexual activity. A second factor is the long period of infectiousness prior to the onset of AIDS, which we model by a series of compartments representing stages an individual must pass through from the time of infection to the onset of clinical disease. The use of such a compartmental model permits one to examine a third factor, the effect of variation in transmission probability over the stages of infectiousness. Let us examine the effects of these three factors on the dynamic and steady-state behaviour of the model.

***Assumptions and Notation***

Let us assume a population divided into n groups based on sexual activity, the number of contacts per unit time. In this first model there is no migration between groups, but they interact by way of sexual contacts. Figure 1 shows a compartmental model of the i[th] contact-rate group. For the $i^{th}$ group, $X$, is the number of susceptibles, $x$ is the total number of infecteds, and $Z$, is the number with AIDS. From first infection to the development of AIDS, the infected pass through a series of $m$ stages,

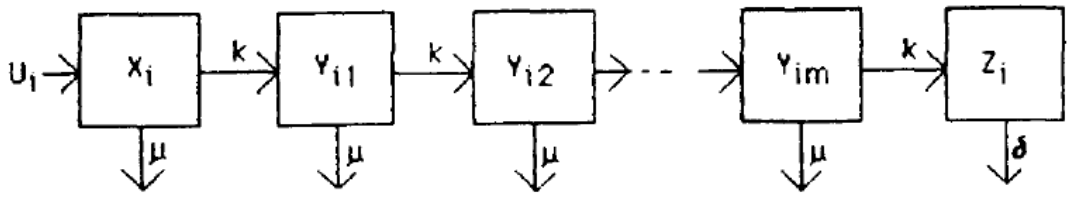


**Fig. 1.** Compartmental model for group $i$

$Y_{i1}, \ldots\ldots\ldots\ldots, Y_{im}$ with $Y_i = \sum_r Y_{ir}$. Those in $Z_i$ are presumed to be so ill with AIDS that they no longer take part in transmission. Thus the model consists of a set of $n$ $SI$ models that interact by way of contacts between the susceptibles and infecteds of the submodels. Let us assume that the recruitment rate $U_i$, for the $i$ [th] group, the number of new susceptibles entering the group per unit of time, is constant.

Let us define the following parameters:

$c_i$, the number of persons contacted (sex acts) per person in group $i$ per unit time. This has dimensions $t^{-1}$. For very small populations the number of contacts per person per unit time might well be a function of the size of the population. We assume that we are not in that range and take a constant value for $c_i$.

$\rho_{ij}$, the proportion of the contacts of a person in group $i$ made with persons in group $j$; it is dimensionless, and $\sum_j \rho_{ij} = 1$. The matrix $P = \left[\left(\rho_{ij}\right)\right]$ is the mixing matrix or contact fraction matrix.

$\beta_{ijr}$, the transmission fraction. The fraction of contacts between a susceptible person in group $i$ and an infected person in $Y_{jr}$, that transmits the virus; dimensionless. Let us look only at $\beta_{ijr}$ that vary with $r$, the stage of the infection, but do not depend on $i$ and $j$.

$k$, the fractional transfer rate from $Y_{ir}$to $Y_{i,r+1}$; dimension $t^{-1}$.

$\mu$, the fractional rate at which members transfer out of the groups for all reasons other than the development of AIDS. Let us call it the competing mortality rate, assumed constant for $X_i$ and $Y_i$ and assumed small relative to $k$.

$\delta$, the mortality rate for those with AIDS; dimension $t^{-1}$. This need not be used in the model ($\delta = 0$); if it is not used, $Z_i$ represents the total number who have developed AIDS, living and dead.

***The Equations***

With the parameters so defined, the rate at which susceptibles in group $i$ are infected by contact with members of $Y_j$ must be

$$c_i X_i \rho_{ij} \beta_{ijr} \frac{Y_{jr}}{X_j + Y_j}$$

In this expression, $c_i X_i$ is the total number of contacts of $X_i$ per unit time, $\rho_{ij}$ gives the fraction of these contacts that are with group $j, Y_{jr}/X_j + Y_j$ is the probability that the contact with group $j$ is with a person in the r th infectious subgroup, and $\beta_{ijr}$ is the fraction of those contacts that result in transmission. Thus the equation for the rate of change of $X_i$ is given by

$$\dot{X}_i = -c_i X_i \sum_{j=1}^{n} \rho_{ij} \sum_{r=1}^{m} \beta_{ijr} \frac{Y_{jr}}{X_j + Y_j} - \mu X_i + U_i$$

The equations for $Y_{i1}, \ldots\ldots\ldots\ldots, Y_{im}$ and $Z_i$ are then

$$\dot{Y}_{i1} = c_i X_i \sum_{j} \rho_{ij} \sum_{r} \beta_{ijr} \frac{Y_{jr}}{X_j + Y_j} - (k + \mu) Y_{i1}$$

$$\dot{Y}_{ir} = k Y_{i,r-1} - (k + \mu) Y_{ir}, \quad r = 2,3, \ldots\ldots, m$$

$$\dot{Z}_i = k Y_{im} - \delta Z_i$$

This model differs in important ways from models frequently used in epidemiological modeling:

1. With some exceptions, the models commonly used are for a system of constant population size in which births equal deaths. Let us look at a population with constant rates of recruitment into the different groups. In the

absence of AIDS and competing deaths, such populations would grow at constant rates. In the absence of HIV and with $\mu > 0$, each population comes to a constant equilibrium population. Let us start with such equilibrium populations, introduce HIV, and follow the dynamics, seeking the conditions that give endemic steady states.

2. The model explicitly includes a sequence of stages of infection that may differ in infectiousness,
3. Let us introduce a new type of mixing, preferred mixing, in which a fraction of each group's contacts can be reserved for intragroup contacts, a different fraction for each group.

An important consequence is that the rates of change of the susceptibles are not quadratic in susceptibles and infectives but are more complicated nonlinear, rational functions of the susceptibles and infectives.

## 3. MODEL FOR INTRAVENOUS DRUG USERS

A very interesting model, which takes the following form:

$$\frac{d\beta(t)}{dt} = \frac{\lambda}{m}\left[\left(1-\beta(t)\right)I(t) - \theta\beta(t)U(t)\right] \quad (1)$$

$$\frac{dI(t)}{dt} = \lambda\alpha\beta(t)U(t) - \mu_2 I(t) \quad (2)$$

$$\frac{dU(t)}{dt} = c[U(t)+I(t)]^{v} - \lambda\alpha\beta(t)U(t) - \mu_1 U(t) \quad (3)$$

In this model, it is assumed that a needle – sharing intravenous (IV) drug user population is divided into two disjoint classes: HIV – infected users and uninfected users. $I(t)$ and $U(t)$ represent the number of infected users and uninfected users, respectively at time $t$. $\beta(t)$ represents the fraction of needles which are HIV – infectious at time $t$. The parameters in the model are defined as follows:

$m$ = the number of shooting galleries, i.e., places where needles are rented to users;

$h$ = the rate at which users visit shooting galleries;

$\alpha$ = the probability an uninfected user who injects with an infectious needle will become infected;

$\theta$ = the probability an infectious needle ceases to be infected after use by an uninfected user;

$c$ = the recruitment rate into the population;

$\mu_1 =$ the exit rate for uninfected users;

$\mu_2 =$ the exit rate for infected users;

$v =$ the aggregation index, $0 \leq v < 1$.

The nonnegative $\beta -$ axis contains only equilibria when $v > 0$ and there is always an equilibrium

$$E_0 = \left(0,0,\left(\frac{c}{\mu_1}\right)^{1/1-v}\right)$$

on the positive $U$ – axis. The equilibrium $E_0$ is called the infection-free equilibrium. Furthermore, there is a threshold parameter $R_0 = \lambda\alpha/\theta\mu_2$ such that when $R_0 \leq 1$, there is no positive equilibrium and when $R_0 > 1$ there is a unique positive equilibrium, called the endemic equilibrium. The parameter $R_0$ is called the basic reproductive rate of infection since it is the average number of users who are infected by an infected user over the period of addiction.

The value of equilibria is often used to predict the future population size of a target population. However, such predictions are more reliable if a stability analysis is performed. The objective of this paper is to carry out a complete qualitative analysis of the model (1) – (3) and examine the epidemiological implications. Let us investigate the behavior of solutions of the model.

### *3.1 Stability Analysis*

Let us begin with describing some basic properties of the model. It is easy to see that the set

$$\mathcal{G} = \{(\beta, I, U); 0 \leq \beta \leq 1, 0 \leq I, 0 \leq U, \\ 1 + U \leq \left[\frac{c}{min(\mu_1, \mu_2)}\right]^{1/(1-v)}\}$$

is a positive invariant set which attracts all feasible solutions, i.e., solutions with $0 \leq \beta(t) \leq 1, I(t) \geq 0, U(t) \geq 0$. Thus, it suffices to study the behavior of solutions lying in $\mathcal{G}$. For $v > 0$, the set $\mathcal{B} = \{(\beta, I, U); I = U = 0, 0 \leq \beta \leq 1\}$ is an invariant set which contains only equilibria. That implies that if there are no users, the fraction of infectious needles remains as it is.

The set

$$\mathcal{U} = \{\beta, I, U); \beta = I = 0, U > 0\}$$

is a positive invariant set, called the infection – free set. There is a unique equilibrium $E_0 = \left(0,0,\left(\frac{c}{\mu_1}\right)^{1/(1-v)}\right)$ in $\mathcal{U}$ which is globally asymptotically stable with respect to $\mathcal{U}$, Hence, without HIV epidemic, the population size will approach $\left(\frac{c}{\mu_1}\right)^{1/(1-v)}$.

***Theorem 1***

Let $R_0 = \lambda\alpha/\theta\mu_2$. If $R_0 \leq 1$, there is no positive equilibrium. If $R_0 > 1$, there is a unique positive equilibrium $E_1 = (\beta_1, I_1, U_1)$

where

$$\beta_1 = 1 - \frac{\theta\mu_2}{\lambda\alpha}, \qquad I_1 = (\lambda\alpha - \theta\mu_2)\left[\frac{c(\mu_2 + \lambda\alpha - \theta\mu_2)^v}{\mu_2(\mu_1 + \lambda\alpha - \theta\mu_2)}\right]^{1/1-v}$$

$$U_1 = \mu_2\left[\frac{c(\mu_2 + \lambda\alpha - \theta\mu_2)^v}{\mu_2(\mu_1 + \lambda\alpha - \theta\mu_2)}\right]^{1/1-v}$$

Let us now look at the Jacobian $J_0$ of the system (1) – (3) at $E_0$

$$J_0 = \begin{bmatrix} -\frac{\lambda\theta}{m}\left(\frac{c}{\mu_1}\right)^{1/1-v} & \frac{\lambda}{m} & 0 \\ \lambda\alpha\left(\frac{c}{\mu_1}\right)^{r/1-v} & -\mu_2 & 0 \\ -\lambda\alpha\left(\frac{c}{\mu_1}\right)^{1/1-v} & v\mu_1 & -(1-v)\mu_1 \end{bmatrix}$$

Thus, $J_0$ is a stable matrix if $R_0 < 1$ and unstable if $R_0 > 1$.

***Theorem 2***

The equilibrium $E_0$ is globally asymptotically stable with respect to $\mathcal{G}\backslash\mathcal{B}$ if and only if $R_0 \leq 1$.

***Proof***

If $E_0$ is globally asymptotically stable and $R_0 > 1$, then $J_0$ is unstable, which implies $E_0$ is unstable, contradiction. Thus, $R_0 \leq 1$.

Now suppose $R_0 \leq 1$. Let $V = m\mu_2\beta + \lambda I$. Then the derivative of $V$ along solutions of (1) – (3) is

$$\frac{dV}{dt} = \lambda\mu_2[I - \beta I - \theta\beta U] + \lambda^2\alpha\beta U - \lambda\mu_2 I$$

$$= -\lambda\mu_2\beta I - (\theta\mu_2 - \lambda\alpha)\lambda\beta U \leq 0$$

since $\theta\mu_2 - \theta\alpha \geq 0$. And $\frac{dV}{dt}$ implies $\beta = 0$ or $I = 0$ and $U = 0$.

Therefore, the set

$$\mathcal{V} = \left\{(\beta, I, U); \beta = 0, 0 \leq I \leq \left[\frac{c}{min(\mu_1, \mu_2)}\right]^{1/1-v} or \right.$$
$$\left. 0 \leq \beta \leq 1, I = 0 \; and \; U = 0\right\}$$

is a global attractor to $\mathcal{G}\backslash\mathcal{B}$.

Adding (2) and (3) , we have

$$\frac{d}{dt}(U + I) = c(U + I)^v - \mu_2 I - \mu_1 U$$
$$\geq c(U + I)^v - (\mu_1 + \mu_2)(U + I)$$

Let $\varepsilon = (c/\mu_1 + \mu_2)^{1/1-v}$. Then if $U + I < \varepsilon$,

$$\frac{d(U + I)}{dt} \geq (U + I)^v[c - (\mu_1 + \mu_2)(U + I)^{1-v}] > 0$$

Hence, the set $\mathcal{B}$ is repellent. Therefore, the global attractor is the maximum invariant set contained in $\mathcal{V}\backslash\mathcal{B}$. Since $\{E_0\}$ is the only invariant set, so that $\{E_0\}$ is the global attractor. In other words, $E_0$ is globally asymptotically stable with respect to $\mathcal{G}\backslash\mathcal{B}$. The proof of the theorem is complete.

***Theorem 3***

The endemic equilibrium $E_1$ is locally asymptotically stable if it exists.

***Proof***

The Jacobian $J_1$ at $E_1$ is

$$J_1 = \begin{bmatrix} -\frac{\lambda^2\alpha K}{m} & \frac{\theta\mu_2}{m\alpha} & -\frac{\lambda\theta}{m}\left(1 - \frac{\theta\mu_2}{\lambda\alpha}\right) \\ \lambda\alpha\mu_2 K & -\mu_2 & \lambda\alpha - \theta\mu_2 \\ -\lambda\alpha\mu_2 K & \frac{v\mu_2(\mu_1 + \lambda\alpha - \theta\mu_2)}{\mu_2 + \lambda\alpha - \theta\mu_2} & \frac{-(\mu_1 + \lambda\alpha - \theta\mu_2)\left((1 - v)\mu_2 + \lambda\alpha - \theta\mu_2\right)}{\mu_2 + \lambda\alpha - \theta\mu_2} \end{bmatrix}$$

where $K = [c(\mu_2 + \lambda\alpha - \theta\mu_2)^v/\mu_2(\mu_1 + \lambda\alpha - \theta\mu_2)]^{1/1-v}$.

Suppose that the characteristic polynomial of $J_1$ is

$$f(\tau) = \tau^3 + a_1\tau^2 + a_2\tau + a_3$$

Then by the Routh – Hurwitz criterion, $J_1$, is stable if and only if $a_1 > 0$, $a_3 > 0, a_1 a_2 > a_3$. Let us now verify these conditions.

$$a_1 = \frac{\lambda^2 \alpha K}{m} + \mu_2 + \frac{(\mu_2 + \lambda\alpha - \theta\mu_2)[(1-v)\mu_2 + \lambda\alpha - \theta\mu_2]}{\mu_2 + \lambda\alpha - \theta\mu_2}$$

$$> \frac{\lambda^2 \alpha K}{m} > 0$$

$$a_2 = \frac{\lambda\mu_2 K}{m}(\lambda\alpha - \theta\mu_2) + \frac{\lambda^2\alpha K}{m}\left\{\frac{(\mu_1 + \lambda\alpha - \theta\mu_2)[(1-v)\mu_2 + \lambda\alpha - \theta\mu_2]}{\mu_2 + \lambda\alpha - \theta\mu_2}\right.$$

$$\left. - \frac{\theta\mu_2}{\lambda\alpha}(\lambda\alpha - \theta\mu_2)\right\} + (1-v)\mu_2(\mu_1 + \lambda\alpha - \theta\mu_2)$$

$$= \frac{\lambda^2\alpha K(\mu_1 + \lambda\alpha - \theta\mu_2)[(1-v)\mu_2 + \lambda\alpha - \theta\mu_2]}{m(\mu_2 + \lambda\alpha - \theta\mu_2)}$$

$$+ \frac{\lambda\mu_2 K(1-\theta)(\lambda\alpha - \theta\mu_2)}{m} + (1-v)\mu_2(\mu_1 + \lambda\alpha - \theta\mu_2)$$

$$> (1-v)\mu_2(\mu_1 + \lambda\alpha - \theta\mu_2)$$

$$a_3 = -det(J_1)$$

$$= \frac{(1-v)\lambda^2\alpha\mu_2 K}{m}\left(1 - \frac{\theta\mu_2}{\lambda\alpha}\right)(\mu_1 + \lambda\alpha - \theta\mu_2) > 0$$

Hence,

$$a_1 a_2 > \frac{\lambda^2\alpha K}{m}(1-v)\mu_2(\mu_1 + \lambda\alpha - \theta\mu_2) > a_3$$

Therefore, $J_1$ is a stable matrix, which implies that $E_0$ is locally asymptotically stable.

***Theorem 4***

Suppose $R_0 > 1$. If $\mu_1 = \mu_2$ or $\theta = 0$, then the endemic equilibrium $E_1$ is globally asymptotically stable with respect to $Int\ \mathcal{G}$, the interior of $\mathcal{G}$.

***Proof***

(i). $\mu_1 = \mu_2 = \mu$. From

$$\frac{d(U+I)}{dt} = c(U+I)^v - \mu(U+I)$$

$$\lim_{t\to\infty}[U(t)+I(t)] = \left(\frac{c}{\mu}\right)^{1/1-v}$$

Since $E_1$ is locally asymptotically stable, the global stability of $E_1$ with respect to the interior of $\mathcal{G}_1$, where

$$\mathcal{G}_1 = \left\{(\beta, I, U) \in \mathcal{G}; U + I = \left(\frac{c}{\mu}\right)^{1/1-v}\right\}$$

implies the global stability of $E_1$ with respect to the interior of $\mathcal{G}$.

Let us now show the global stability of $E_1$ with respect to the interior of $\mathcal{G}_1$. In $\mathcal{G}_1$, the system is equivalent to

$$\frac{d\beta}{dt} = \frac{\lambda}{m}\left\{[1-(1-\theta)\beta]I - \theta\left(\frac{c}{\mu}\right)^{1/1-v}\beta\right\} \quad (4)$$

$$\frac{dI}{dt} = \lambda\alpha\left(\frac{c}{\mu}\right)^{1/1-v}\beta - \lambda\alpha\beta I - \mu I \quad (5)$$

Denote the right – hand sides of (4) and (5) by $F_1$, and $F_2$ respectively.

Then

$$\frac{\partial F_1}{\partial \beta} = -(1-\theta)I - \theta\left(\frac{c}{\mu}\right)^{1/1-v} < 0$$

$$\frac{\partial F_2}{\partial I} = -\lambda\alpha\beta - \mu < 0$$

By the Dulac's criterion, there is no periodic solution for (4) – (5). Since $E_1$ is the only locally stable equilibrium of the system, it is globally asymptotically stable with respect to the interior of $\mathcal{G}_1$.

(ii).$\theta = 0$. In this case, (1) becomes

$$\frac{d\beta}{dt} = \frac{\lambda}{m}(1-\beta)I \geq 0$$

Hence, $\lim_{t\to\infty}\beta(t) = 1$. A similar argument to (i) shows that $E_1$ is globally asymptotically stable if the system

$$\frac{dI}{dt} = \lambda\alpha U - \mu_2 I$$

$$\frac{dU}{dt} = c(U+I)^v - \lambda\alpha U - \mu_1 U$$

has no periodic solution in the interior of $\mathcal{G}_2$, where $\mathcal{G}_2 = \{(\beta, I, U) \in \mathcal{G}; \beta = 1\}$.
Since

$$\frac{d(U+I)}{dt} = c(U+I)^{v} - \mu_1 U - \mu_2 I$$

$$\liminf_{t\to\infty} [U(t) + I(t)] \geq \left(\frac{c}{\mu_1 + \mu_2}\right)^{1/1-v}$$

for any solution $(I(t), U(t))$. Hence, it suffices to prove that there is no periodic solution in

$$\mathcal{G}_3 = \left\{(I,U); (\beta, I, U) \in \mathcal{G}_2, I + U \geq \left(\frac{c}{\mu_1 + \mu_2}\right)^{1/1-v}\right\}$$

Let $(G_1, G_2)$ be the right – hand sides of (4) – (5). Then

$$\frac{\partial G_1}{\partial I} + \frac{\partial G_2}{\partial U} = cv(U+I)^{v-1} - \mu_1 - \mu_2 - \lambda\alpha \leq -\lambda\alpha < 0$$

By the Dulac's criterion again, $\mathcal{G}_3$ does not contain any periodic solution. Therefore the theorem is proved.

## REFERENCES


[1] Anderson. R . M and May. R. M, Infectious Diseases of Humans. Oxford Science, Oxford, 1991.

[2] Castillo- Chavez. C, Cooke. K, Huang. W and S.A. Levin, On the role of long incubation periods in the dynamics of AIDS, Part 1. Single population models, J. Math. Bio.27: 373-398(1989).

[3] Hethcote. H.W, Van Ark. J.W and I.M. Longini, Jr, A Simulation Model of AIDS in San Francisco: I. Model formulation and Parameter Estimation, Math. Biosci. 106: 203-222(1991).

[4] Hethcote. H.W, Van Ark. J.W. and J.M.Karon, A Simulation Model of AIDS in San Francisco: II. Simulations, therapy and sensitivity analysis, Math. Biosci. 106: 223-247(1991).

[5] Hirsch. M. W, Systems of differential equations which are competitive or cooperative, I: Limit sets, SIAM J. Math. Anal. 13:167-179 (1982).

[6] Jacquez. J.A, Simon. C.P, koopman. J, Satterspiel. L and T. Perry, Modelling and analyzing HIV transmission: The effect of contact patterns, Math. Biosci. 92: 119-199(1988).

[7] Bailey. N.J.T, The Mathematical Theory of Infectious Diseases (2nd ed.), Macmillan, New York, 1975.