Modelling the spread of AIDS epidemic with vertical transmission

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Abstract

This paper considers a non-linear mathematical model for HIV epidemic that spreads in a variable size population through both horizontal and vertical transmission. The equilibrium points of the model system are found and their stability is investigated. The model exhibits two equilibria namely, the disease-free and the endemic equilibrium. It is found that if the basic reproduction number $R_0 < 1$, the disease-free equilibrium is always locally asymptotically stable and in such a case the endemic equilibrium does not exist. If $R_0 > 1$, a unique endemic equilibrium exists which is locally asymptotically stable and becomes globally asymptotically stable under certain conditions showing that the disease becomes endemic due to constant immigration of population into the community. Using stability theory and computer simulation, it is shown that by controlling the rate of vertical transmission, the spread of the disease can be reduced significantly and consequently the equilibrium values of infective and AIDS population can be maintained at desired levels. A numerical study of the model is also used to investigate the influence of certain other key parameters on the spread of the disease.

Keywords: AIDS epidemic; Vertical transmission; Immigration; Stability; Simulation

1. Introduction

The human immuno-deficiency virus (HIV) infection which can lead to acquired immuno-deficiency syndrome (AIDS), has become an important infectious disease in both the developed and developing nations. It is a fatal disease, which breaks down the body’s immune system, leaving the victim vulnerable to a host of life threatening opportunistic infections, neurological disorders or unusual malignancies. It causes mortality of millions of people and expenditure of enormous amount of money in health care and disease control. The AIDS epidemic is now spreading rapidly in Asia, where new infections are increasing faster than anywhere else in the world. Globally, India has the highest estimated number of HIV infected people in any single country, next only to South Africa. In India 86.27% cases of infection are due to sexual transmission which indicates...
that the heterosexual contact is the predominant mode of transmission of HIV [20]. Mathematical models have been used extensively in research into the epidemiology of HIV/AIDS, to help improve our understanding of the major contributing factors in a given epidemic. From the initial models of May and Anderson [2,3,19] various refinements have been added into modeling frameworks, and specific issues have been addressed by researchers (see for instance [4,8–16,21]). HIV infection spreads rapidly in populations through unsafe sexual interaction with an accompanying risk of vertical transmission. This is a key factor in many infectious diseases, including AIDS, chagas’s disease and hepatitis B. Therefore, this aspect should also be considered in the modeling of AIDS especially in Asian countries like India where the infection may be transmitted vertically at a very high rate.

Vertical transmission can be accomplished through transplacental transfer of disease agents. In recent years, a few studies of vertical transmission have been conducted to describe the effects of various epidemiological and demographical factors [1,5–7,17,18]. In particular, Busenberg and Cooke [7] discussed a variety of diseases that transmit both horizontally and vertically, and gave a comprehensive survey of the formulation and the mathematical analysis of compartmental models that also incorporate vertical transmission. Brauer [5] considered models for disease with vertical transmission with non-linear population dynamics and finite carrying capacity and analyzed the stability of equilibria in the special case in which the overall birth rate does not depend on infective population size. Li et al. [18] proposed a model for an infectious disease that spreads in the host population through both horizontal and vertical transmission. A little attention has been paid to study the role of vertical transmission in HIV/AIDS models. Agarwala [1] developed a density dependent HIV transmission model for Canadian population by taking into account the vertical transmission and by using simple mass action type interaction.

In this paper we have, therefore, developed a model for transmission of HIV into a population of varying size with vertical transmission and other demographic and epidemiological factors. Our purpose is to formulate a model for AIDS epidemic that may be transmitted either directly or vertically in populations and to study its behavior qualitatively and numerically.

2. The model

We consider a population of size \( N(t) \) at time \( t \) with constant inflow of susceptibles with a rate \( Q_0 \). The population size \( N(t) \) is divided into four subclasses of susceptibles \( S(t) \), infectives \( I(t) \) (also assumed to be infectious), pre-AIDS patients \( P(t) \) and AIDS patients \( A(t) \) with natural mortality rate \( d \) in all the classes. In the model, we assume that the susceptibles become HIV infected via sexual contacts with infectives which may also lead to the birth of infected children. It is assumed that a fraction of new born children are infected at birth and hence are directly recruited into the infective class with a rate \( (1 - \varepsilon)\theta \) and others die effectively at birth \( (0 < \varepsilon < 1) \). We do not consider direct recruitment of other infected persons but by vertical transmission only. The interaction between susceptibles and infectives is assumed to be of standard mass action type. It is also assumed that some of the infectives move to join pre-AIDS class, depending on the viral counts, with a rate \( \sigma \delta \) and then proceed with a rate \( \mu \) to develop full blown AIDS while others with serious infection directly join the AIDS class with a rate \( (1 - \sigma)\delta \) where \( 0 \leq \sigma \leq 1 \).

In view of the above, spread of the disease is assumed to be governed by the following system of differential equations:

\[
\begin{align*}
\frac{dS}{dt} &= Q_0 - \frac{\beta_1 cSI}{N} - \frac{\beta_2 cSP}{N} - \frac{\beta_3 cSA}{N} - dS, \\
\frac{dI}{dt} &= \frac{\beta_1 cSI}{N} + \frac{\beta_2 cSP}{N} + \frac{\beta_3 cSA}{N} - (\delta + d)I + (1 - \varepsilon)\theta(I + P + A), \\
\frac{dP}{dt} &= \sigma \delta I - (\mu + d)P, \\
\frac{dA}{dt} &= (1 - \sigma)\delta I + \mu P - (\sigma + d)A, \\
S(0) &= S_0, \quad I(0) = I_0, \quad P(0) = P_0 \quad \text{and} \quad A(0) = A_0,
\end{align*}
\]
where \( c \) is the average number of sexual partners per unit time, \( \delta \) is the rate of movement from infectious class, so that \( 1/\delta \) denotes the average incubation period, \( \beta_i \) (\( i = 1, 2, 3 \)) are the contact rates of susceptibles with infectives, pre-AIDS and AIDS patients respectively and \( \alpha \) is the disease induced death rate due to AIDS. It is assumed that all the dependent variables and parameters of the model are non-negative.

To simplify the model, it is reasonable to assume that the AIDS patients and those in pre-AIDS class are exposed and sexually inactive as they are isolated and hence are not capable of producing children i.e. \((1 - \varepsilon)\theta P = (1 - \varepsilon)\theta A = 0\) and they also do not contribute to viral transmission horizontally i.e. \( \beta_2 \) and \( \beta_3 \) are taken negligible. It is remarked here that these assumptions are valid in developed countries following stringent screening measures but may not be true in under developed nations due to poor medical facilities or the social stigma attached with the disease.

In view of the above assumptions and using \( N = S + I + P + A \), the system (2.1) can now be written as follows:

\[
\begin{align*}
\frac{dN}{dt} &= Q_0 - dN - \alpha A + (1 - \varepsilon)\theta I, \\
\frac{dI}{dt} &= \beta_1 c (N - I - P - A) - (\delta + d)I + (1 - \varepsilon)\theta I, \\
\frac{dP}{dt} &= \sigma \delta I - (\mu + d)P, \\
\frac{dA}{dt} &= (1 - \sigma)\delta I + \mu P - (\alpha + d)A, \\
N(0) &= N_0, \quad I(0) = I_0, \quad P(0) = P_0 \quad \text{and} \quad A(0) = A_0.
\end{align*}
\]

Continuity of the right-hand side of system (2.2) and its derivative imply that the model is well posed. It is pointed out here that not all infected individuals take part in spreading the disease, as in the case of infected children, but they will all develop their own AIDS. However, the model can be modified to include a delay to make the infected children become adult to further spread the infection either vertically or horizontally. This is left for future research.

### 2.1. Early stage epidemic

From the model (2.2), it is noted that in the absence of infection, population size approaches the steady state value \( Q_0/d \). During the early stage of epidemic, if it is assumed that \( S \approx N \approx \frac{Q_0}{d} \) then the growth of infectious people \( I(t) \) can be approximately governed by the following equation:

\[
\frac{dI}{dt} = [\beta_1 c + (1 - \varepsilon)\theta - (\delta + d)]I,
\]

which gives

\[
I(t) = I_0 \exp \left[ \frac{R_0 - 1}{T} \right] t,
\]

where \( R_0 = \frac{\beta_1 c + (1 - \varepsilon)\theta}{(\delta + d)} \), the basic reproduction rate, and \( T = \frac{1}{(\delta + d)} \) the time during which people remain infective. The doubling time \( t_d \) of the epidemic can be obtained as

\[
t_d = \frac{\ln 2}{R_0 - 1}.
\]

Thus if \( R_0 > 1 \), the infection triggers an epidemic otherwise its prevalence is zero i.e. for \( R_0 < 1 \). From the solution \( I(t) \), it is noted that with an increase in \( R_0 \), which can be viewed as a function of \( c \), the number of sexual partners, the number of infectives increases which in turn increases the AIDS patients population. Thus in order to keep the spread of the disease at minimum, the number of sexual partners should be restricted.

In Fig. 1, the variation of basic reproduction rate \( R_0 \) with doubling time \( t_d \) is shown. It is noted that if \( R_0 > 1 \) then the epidemic is said to be growing and otherwise for \( R_0 < 1 \) the epidemic is diminishing. If \( R_0 \) is just above 1, then there is a slow spread in the early stage as the doubling time is higher. A small increase

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in the basic reproduction rate results in the reduction of the doubling time which indicates the faster growth of the infection. This effect may have an important epidemiological consequence as in the absence of vaccine or other control mechanism, cultural change with respect to sexual practices becomes the only means of effective control.

3. Stability analysis

In this section, we present the results of stability analysis of the equilibrium points.

3.1. Equilibrium of the model

The model (2.2) has two non-negative equilibrium points namely:

(i) $E_0(Q_0/d,0,0,0)$, the disease-free equilibrium,
(ii) $E^*(N^*,I^*,P^*,A^*)$, the endemic equilibrium,

where

$$N^* = \frac{\beta_1 c r I^*}{\mu + d}, \quad I^* = \frac{Q_0}{\beta_1 c d r + \frac{\gamma (1 - \sigma) \delta}{(\mu + d)(\mu + d)}},$$

$$P^* = \frac{\sigma \delta I^*}{(\mu + d)}, \quad A^* = \frac{\delta [\mu + d (1 - \sigma)] I^*}{(\mu + d)(\mu + d)},$$

$$\gamma = \frac{1 + \frac{ax}{\mu + d} + \frac{\mu + d (1 - \sigma)}{(\mu + d)(\mu + d)}}{[\beta_1 c + (1 - \sigma) \theta - (\delta + d)].}$$

$\gamma$ is positive only when $[\beta_1 c + (1 - \sigma) \theta] > (\delta + d)$ i.e. $R_0 > 1$. It is noted that $E^*$ is positive if

$$\beta_1 c d r + \frac{\gamma (1 - \sigma) \delta}{(\mu + d)(\mu + d)} > [(1 - \sigma) \theta].$$

It is found that equilibrium level of infectives $I^*$ increases as $Q_0$ increases or $\gamma$ decreases leading to increase in $P^*$ and $A^*$. Also if $\theta$ increases then the equilibrium values of $I^*$, $P^*$ and $A^*$ increase. Thus an increase in the rate of vertical transmission is to increase the equilibrium level of infectives which in turn increases the equilibrium level of pre-AIDS and that of AIDS population. The equilibrium level of AIDS patients $A^*$ decreases as the disease induced death rate $\mu$ increases. It is also noted that when the disease remain endemic, the disease-induced deaths reduce the equilibrium population size from $Q_0/d$ to $N^*$.
3.2. Local stability of the equilibria

Now to determine the local stability of \( E_0 \) and \( E^* \), the following variational matrices are computed corresponding to equilibrium points \( E_0 \) and \( E^* \):

\[
M_0 = \begin{bmatrix}
-d & (1 - \varepsilon)\theta & 0 & -\alpha \\
0 & \beta_1c + (1 - \varepsilon)\theta - (\delta + d) & 0 & 0 \\
0 & \sigma\delta & -(\mu + d) & 0 \\
0 & (1 - \sigma)\delta & \mu & -(\alpha + d)
\end{bmatrix},
\]

\[
M^* = \begin{bmatrix}
-d & (1 - \varepsilon)\theta & 0 & -\alpha \\
0 & \beta_1c + (1 - \varepsilon)\theta - (\delta + d) & 0 & 0 \\
0 & \sigma\delta & -(\mu + d) & 0 \\
0 & (1 - \sigma)\delta & \mu & -(\alpha + d)
\end{bmatrix}.
\]

From \( M_0 \) it is clear that \( E_0 \) is locally asymptotically stable provided \([\beta_1c + (1 - \varepsilon)\theta] < (\delta + d)\) i.e. for \( R_0 < 1 \), the disease dies out and under this condition the equilibrium \( E^* \) does not exist. If \( R_0 > 1 \), then \( E^* \) exists and the infection is maintained in the population.

The characteristic equation corresponding to \( M^* \) is given by

\[
f(\lambda) = \lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0,
\]

where

\[
a_1 = \alpha + \mu + 3d + \frac{\beta_1cI^*}{N^*},
\]

\[
a_2 = (\alpha + d)(\mu + d) + d(\alpha + \mu + 2d) + [\beta_1c(\alpha + \mu + 3d) - (1 - \varepsilon)\theta(\beta_1c + (1 - \varepsilon)\theta - (\delta + d))] \frac{I^*}{N^*},
\]

\[
a_3 = d(\alpha + d)(\mu + d) + [(\alpha + d)(\mu + d) + \delta(\mu + d + \alpha\sigma) + d(\alpha + \mu + \delta + 2d)] \frac{\beta_1cI^*}{N^*}
\]

\[
+ [(1 - \sigma)\alpha\delta - (1 - \varepsilon)\theta(\alpha + \mu + 2d)][\beta_1c + (1 - \varepsilon)\theta - (\delta + d)] \frac{I^*}{N^*},
\]

\[
a_4 = d[(\alpha + d)(\mu + d) + \delta(\mu + d + \alpha\sigma)] \frac{\beta_1cI^*}{N^*} + [\beta_1c + (1 - \varepsilon)\theta - (\delta + d)] \frac{I^*}{N^*}
\]

\[
\times [\delta(\mu + (1 - \sigma)d) - (1 - \varepsilon)\theta(\mu + d)(\alpha + d)]\alpha.
\]

Thus by Routh–Hurwitz criteria, \( E^* \) is locally asymptotically stable as it can be seen for \( a_1 > 0, a_2 > 0, a_3 > 0, a_4 > 0, a_1a_2 - a_3 > 0 \), and \( a_1a_2a_3 - a_2^2 - a_1a_4 > 0 \).

3.3. Global stability of the endemic equilibrium

Now to show the globally stability behavior of \( E^* \), we need the bounds of dependent variables involved. For this we find the region of attraction stated in the form of the following lemma.

**Lemma 1.** The region

\[
\Omega = \left\{ (N,I,P,A); 0 < N \leq \overline{N} ; 0 \leq I \leq \overline{I} ; 0 \leq P \leq \frac{\sigma\delta I}{\mu + d} ; 0 \leq A \leq \frac{\delta(\mu + d)(1 - \sigma)I}{(\alpha + d)(\mu + d)} \right\}
\]

is a region of attraction for \([\beta_1c + (1 - \varepsilon)\theta] > (\delta + d)\) where

\[
\overline{N} = \frac{Q_0 + (1 - \varepsilon)(\overline{I})}{d} \quad \text{and} \quad \overline{I} = \frac{Q_0}{d} \left\{ 1 + \frac{\theta(1 - \varepsilon) - (\delta + d)}{\beta_1c} \right\}.
\]

(3.2)
**Theorem 1.** If the endemic equilibrium $E^*$ exists, then it is globally asymptotically stable provided the following conditions are satisfied in $\Omega$:

\[
\frac{\sigma^2}{(1-\sigma)} < \frac{2(\alpha + d)(\mu + d)}{3\sigma},
\]

\( k_1 > \min\{q_{11}, q_{12}\}, \tag{3.3} \)

where $q_{11} = \frac{3\epsilon^2}{2\alpha^2} I^N + 3\alpha^2$, $q_{12} = \frac{\alpha^2\epsilon^2}{2\alpha^2}$ and $m = \left[ \frac{\beta_0 c_k}{N} \left( \frac{I - I^*}{N} \right) + (1-\epsilon)\theta \right]$.

**Proof.** Consider the following positive definite function about $E^*$:

\[
V = \frac{1}{2} (N - N^*)^2 + k_1 \left( I - I^* - I^* \ln \frac{I}{I^*} \right) + \frac{1}{2} k_2 (P - P^*)^2 + \frac{1}{2} k_3 (A - A^*)^2, \tag{3.5} \]

where the constants $k_1$, $k_2$ and $k_3$ can be chosen suitably.

The derivative of $V$ along the solution of the system (2.2) can be written as

\[
\frac{dV}{dt} = (N - N^*) \frac{dN}{dt} + k_1 (I - I^*) \frac{dI}{dt} + k_2 (P - P^*) \frac{dP}{dt} + k_3 (A - A^*) \frac{dA}{dt}. \]

After some algebraic calculations we get

\[
\frac{dV}{dt} = -d(N - N^*)^2 + \left[ \frac{\beta_0 c_k}{N^*} \left( \frac{I + P + A}{N} \right) + (1-\epsilon)\theta \right] (N - N^*) (I - I^*) - \alpha (N - N^*) (A - A^*) \]

\[
- \frac{\beta_0 c_k}{N^*} (I - I^*)^2 + \left( k_3 \sigma \delta - \frac{\beta_0 c_k}{N^*} \right) (I - I^*) (P - P^*) - k_2 (\mu + d) (P - P^*)^2 \]

\[
+ \left[ k_3 (1-\sigma) - \frac{\beta_0 c_k}{N^*} \right] (I - I^*) (A - A^*) - k_3 (\alpha + d) (A - A^*)^2 + k_3 \sigma (P - P^*) (A - A^*). \]

It can now be written as the sum of the quadratics as

\[
\frac{dV}{dt} = \frac{1}{2} a_{11} (N - N^*)^2 + a_{12} (N - N^*) (I - I^*) - \frac{1}{2} a_{22} (I - I^*)^2 - \frac{1}{2} a_{33} (A - A^*)^2 \]

\[
+ a_{14} (N - N^*) (A - A^*) - \frac{1}{2} a_{44} (A - A^*)^2 - \frac{1}{2} a_{23} (I - I^*) (A - A^*) - \frac{1}{2} a_{34} (P - P^*) (A - A^*)^2 \]

\[
+ a_{34} (P - P^*) (A - A^*) - \frac{1}{2} a_{44} (A - A^*)^2, \tag{3.6} \]

where

\[
a_{11} = d, \quad a_{12} = \left[ \frac{\beta_0 c_k}{N^*} \left( \frac{I + P + A}{N} \right) + (1-\epsilon)\theta \right], \quad a_{14} = -\alpha, \]

\[
a_{22} = \frac{2\beta_0 c_k}{3N^*}, \quad a_{23} = \left( k_3 \sigma \delta - \frac{\beta_0 c_k}{N^*} \right), \quad a_{24} = \left( k_3 (1-\sigma) \delta - \frac{\beta_0 c_k}{N^*} \right), \]

\[
a_{33} = k_2 (\mu + d), \quad a_{34} = k_3 \sigma, \quad a_{44} = \frac{2}{3} k_3 (\alpha + d). \]

Thus, a sufficient condition for $\frac{dV}{dt}$ to be negative definite is that

\[
a_{11}^2 - a_{11} a_{22} < 0, \]

\[
a_{12}^2 - a_{11} a_{44} < 0, \]

\[
a_{23}^2 - a_{22} a_{33} < 0, \]

\[
a_{24}^2 - a_{22} a_{44} < 0, \]

\[
a_{34}^2 - a_{33} a_{44} < 0. \tag{3.7} \]
Now choosing \( k_2 = \frac{\beta_1 c k_1}{\alpha b N} \) and \( k_3 = \frac{\beta_1 c k_1}{(1 - \sigma) b N} \), the conditions (3.7) give

\[
\frac{\sigma^2}{(1 - \sigma)} < \frac{2(x + d)(\mu + d)}{3\sigma} \quad \text{and} \quad k_1 > \text{min}\{q_{11}, q_{12}\},
\]

where \( q_{11} = \frac{3x^2(1 - \sigma)bN^2}{2d\beta c(x + d)}, q_{12} = \frac{3N^*m^2}{2d\beta c} \) and \( m = \left[ \frac{\beta_1 c k_1}{N^*} \left( \frac{1 + \frac{1}{N^*} \theta}{1 + \frac{1}{N^*} \theta} \right) + (1 - \sigma)\theta \right] \). Hence \( V \) is a Lyapunov function with respect to \( E^* \) whose domain contains \( \Omega \), proving the theorem. □

4. Numerical analysis

We give here numerical simulation of the equilibrium and stability conditions of the model (2.2). We integrate the system (2.2) by fourth order Runge–Kutta method using the following set of parameter values:

- \( Q_0 = 2000 \),
- \( d = 1/70 \),
- \( x = 1 \),
- \( \beta = 0.05 \),
- \( c = 25 \),
- \( \varepsilon = 0.4 \),
- \( \theta = 0.01 \),
- \( \mu = 0.5 \),
- \( \sigma = 0.3 \),
- \( \delta = 0.2 \)

with initial values

- \( N(0) = 10,000 \),
- \( I(0) = 2000 \),
- \( P(0) = 500 \),
- \( A(0) = 200 \).

The endemic equilibrium values are computed as

\[
N^* = 14867.1865, \quad I^* = 9449.1165, \quad P^* = 1103.0097, \quad A^* = 1848.5021.
\]

The results of numerical simulation are displayed graphically in Figs. 2–9. In Fig. 2 the distribution of population with time is shown in different classes without migration e.g. \( Q_0 = 0 \). It is seen that in the absence of migration into the community, the susceptible population decreases continuously as the population is closed.
which results in an increase in infective population first and then it decreases as all infectives will develop AIDS and will die out by disease-induced deaths. Thus the total population, being constant in this case, will be eradicating after some time. Fig. 3 shows the variation of population in all classes with migration. It is found that susceptible population first increases with time and then reaches its equilibrium position. Since due to migration susceptible population increases continuously therefore infection becomes more endemic.
and always persists in the population. On comparing Figs. 2 and 3 it is noted that the increase in the rate of migration into community increases the AIDS population. Thus, if the rate of migration is restricted into susceptible community, the spread of the disease can be kept under control. The role of vertical transmission i.e. the rate of recruitment of infected children directly into infective class is explicitly shown in Figs. 4 and 5. It is seen that as the rate of infected children born increases, the infective population also increases and as such the AIDS population increases. It may be noted here that the birth of infected children make the infective
population increase. Such children will take their own time to develop full blown AIDS even if they do not take part in horizontal transmission as they are sexually inactive. Thus, if the birth of infected children is controlled by way of promoting the condom use or other control mechanisms, the overall infective population will remain under control. This will help in reducing the AIDS population. Figs. 6–8 depict the variation of infected, pre-AIDS and AIDS population respectively with time for different values of movement rate \( \delta \). It is seen that with increase in the value of movement rate \( \delta \), the infected population decreases which in turn increases the pre-AIDS and AIDS population. This is expected because of shorter incubation period. In Fig. 9 the effect of disease induced death rate \( \alpha \) is shown and it is found that as \( \alpha \) increases, the population of AIDS patients decreases. From the figures, it can also be seen that the respective populations are tending to the equilibrium level. This has also been observed for different initial values of the variables. Hence the equilibrium \( E^* \) is globally asymptotically stable for this set of parameters.

5. Conclusions

In this paper, a non-linear mathematical model is proposed and analyzed to study the transmission of HIV/AIDS in a population of varying size with constant recruitment into susceptible population and with vertical transmission under the assumption that due to sexual interaction of susceptibles with infectives, the infected babies are born to increase the growth of infective population directly. It is assumed that people in pre-AIDS and AIDS classes are exposed and incapable of producing children. By analyzing the model, we have found a threshold parameter \( R_0 \). It is noted that when \( R_0 < 1 \) then disease dies out and when \( R_0 > 1 \) the disease becomes endemic. The model has two non-negative equilibria namely \( E_0(Q_0/d, 0, 0, 0) \), the disease-free equilibrium and \( E^*(N^*, I^*, P^*, A^*) \) the endemic equilibrium. It is found that the equilibrium state \( E_0 \), corresponding to disappearance of disease is locally asymptotically stable if \( R_0 < 1 \) and for \( R_0 > 1 \) it is unstable and the infection is maintained in the population. The endemic equilibrium \( E^* \), which exists only when \( R_0 > 1 \), is always locally asymptotically stable. This equilibrium is also shown to be globally asymptotically stable under certain conditions. It is found that an increase in the rate of vertical transmission leads to increase the population of infectives which in turn increases the pre-AIDS and AIDS population. Thus, the vertical spread of the disease should be controlled by way of promoting the condom use or other effective treatment to keep the overall infective population under control. This will help in reducing the AIDS population as well. By simulation also it is shown that by controlling the rate of vertical transmission, the spread of the disease can be reduced significantly and consequently the equilibrium values of infective and AIDS population can be maintained at desired levels. It is also found that the increase in the number of sexual partners further reduces the total population by way of spreading the disease. Thus in order to reduce the spread of the disease, the number of sexual partners as well as unsafe sexual interaction with an infective is to be restricted. It is also found that the disease becomes more endemic due to immigration. If the rate of migration is restricted into susceptible community, the spread of the disease can also be kept under control. The effect of an increase in disease-induced death rate is, however, to decrease the AIDS patients population. From the analysis, it may also be speculated that if the HIV infection is suppressed at an early stage by effectively treating the infectives, the progression to the AIDS can be slowed down and the life span of HIV infectives can be increased.

References