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Full Length Research Paper

A deterministic model of HIV/AIDS with vertical transmission in the presence of infected immigrants

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A non-linear mathematical model for HIV epidemic that spreads in a variable size population through both horizontal and vertical transmission in the presence of HIV infected immigrants is studied. The equilibrium points of the model are found and the stability is investigated. The model exhibits two equilibria, namely, the disease-free and the endemic equilibrium. It is shown 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. This shows that the disease becomes endemic due to constant immigration of both HIV infected and non infected individuals 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 infected 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 and how to control their influence.

Key words: AIDS epidemic, vertical transmission, immigration, stability, simulation.

INTRODUCTION

The human immuno-deficiency virus (HIV) infection which can lead to acquired immuno-deficiency syndrome (AIDS). It is pointed out that CD4+ T cells are important constituent of human immune system and are primarily attacked by HIV (Srivastava et al., 2009). AIDS has become an important infectious disease in both the developed and developing nations. It is a fatal disease, which destroys 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 has spread rapidly in Africa than any other continent in the world, Sub-Saharan countries being the worst hit. In 2007, it was estimated that two thirds of the global total of 32.9 million people with HIV live in this region, and three quarters of all AIDS deaths in 2007 occurred in the sub-Saharan region. It is estimated that by 2020 the nine most severely hit Sub-Saharan countries may lose 13 to 26% of their agricultural labour force to AIDS. Those dying are more than agricultural workers. They are household heads, mothers and fathers of young children and adolescents, caregivers for the old

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and sick, transmitters of agricultural and livelihood knowledge and skills and custodians of social safety nets. The gross domestic product in countries with HIV prevalence rates of 10% or higher could drop by 18% by 2020 (Amoako at al., 2008).

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 Anderson et al. (1986) and May and Anderson (1987), various refinements have been added into modelling frameworks, and specific issues have been addressed by researchers (Corbett et al., 2003; Diekmann et al., 1990; Perelson and Nelson, 1999; Lewis and Greenhalgh, 2001). HIV infection spreads rapidly in populations through unsafe sexual interaction with an accompanying risk of vertical transmission.

Vertical transmission can occur 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 (Li et al., 2001). In particular, Busenberg and Cooke (1993) 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 (1995) considered models for disease with vertical transmission with non-linear population dynamics and finite carrying capacity, and analyzed the stability of equilibriums in the special case in which the overall birth rate does not depend on infective population size. Li et al. (2001) 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 (2002) 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. Also Naresh et al. (2006) examined a similar model to Agarwala with vertical transmission but did not consider infected immigrants coming into the community.

In this paper therefore, a deterministic model of HIV/AIDS with vertical transmission in the presence of infected immigrants is to be developed. In addition, the demographic and other epidemiological factors are to be taken into consideration. On the other hand the purpose is to formulate a model for AIDS epidemic that may be transmitted either directly or vertically in populations and to study its behaviour qualitatively and numerically.

THE MODEL

A population of size (N) at time t with constant inflow of susceptible at a rate Q is studied. The population size is divided into four subclasses of susceptible (S), infectives (I) also assumed to be infectious, both pre-AIDS patients (P) and AIDS patients (A) are assumed to be sexually inactive, and therefore non-infectious. The natural mortality rate is υ in all classes and the disease induced death rate is α in the AIDS patients class. In addition, β is a sexual contact rate, c is the number of partners per individual and μ is the rate of movement of pre-AIDS class individuals into AIDS class. It is also assumed 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 $(0 \le \varepsilon \le 1)$. Not only vertical transmission is considered for direct recruitment of infected persons within the population, but also infected immigrants are recruited directly into both infectives and pre-AIDS patients classes. Consequently, $m(1-\pi)I$ is the recruitment rate of infective immigrants into the population and $m\pi I$ is the recruitment rate of pre-AIDS immigrants into the population. 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 others with serious infection directly join the AIDS class with a rate $(1-\sigma)\delta$, where $(0 \le \sigma \le 1)$. The interaction between susceptibles and infectives is assumed to be of standard mass action type. Therefore, a system (Equation 1) is the model and Figure 1 is a flow chart of the model.

$$\left.\begin{aligned}
\frac{dS}{dt} &= Q - \beta cS\frac{I}{N} - \upsilon S \\
\frac{dI}{dt} &= m(1-\pi)I + \beta cS\frac{I}{N} - (\delta+\upsilon)I + (1-\varepsilon)\theta I \\
\frac{dP}{dt} &= (m\pi+\sigma\delta)I - (\mu+\upsilon)P \\
\frac{dA}{dt} &= (1-\sigma)\delta I + \mu P - (\alpha+\upsilon)A
\end{aligned}\right\} \quad (1)$$

with

$$S(0) = S_0, \quad I(0) = I_0, \quad P(0) = P_0, \quad A(0) = A_0$$

The model is rearranged

When all equations in the model (Equation 1) are added and $N = S + I + P + A$ is used, then

$$\frac{dN}{dt} = Q - \upsilon N - \alpha A + [m + (1-\varepsilon)\theta]I \ .$$

Generally the model can be rearranged as:

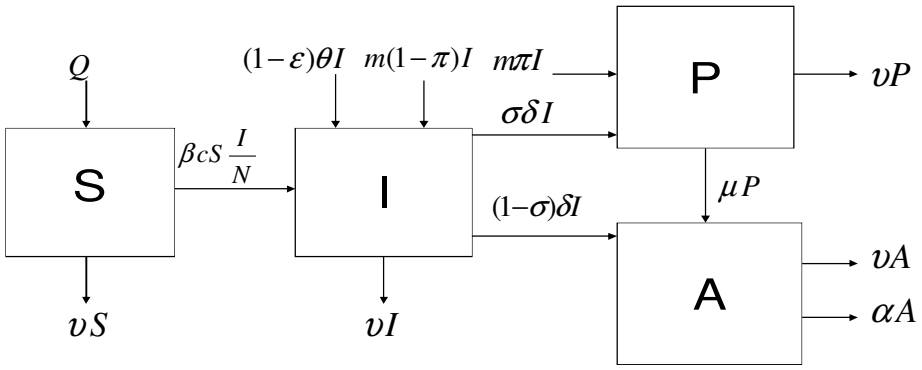

Figure 1. Flow chart.

$$\left.\begin{aligned}
\frac{dN}{dt} &= Q-\upsilon N-\alpha A+[m+(1-\varepsilon)\theta]I \\
\frac{dI}{dt} &= m(1-\pi)I+\beta c(N-I-P-A)\frac{I}{N}-(\delta+\upsilon)I+(1-\varepsilon)\theta I \\
\frac{dP}{dt} &= (m\pi+\sigma\delta)I-(\mu+\upsilon)P \\
\frac{dA}{dt} &= (1-\sigma)\delta I+\mu P-(\alpha+\upsilon)A
\end{aligned}\right\} \quad (2)$$

Continuity of the right-hand side of the system (Equation 2) and its derivative implies 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. It is also assumed that HIV infected classes do not die of the disease except the AIDS-patients class. However, other HIV infected class may die of the disease. This is left for future research.

Value of the basic reproduction number, R_0

In order to achieve disease free equilibrium (DFE), it is expected that the whole population is to be occupied by susceptibles and other classes go to extinction after some time. It is assumed that $S = N$ and $I = P = A$ is estimated to zero when there are relatively few individuals infected in the population. This is substituted into the differential equation of the infective class and the following is obtained.

$$\begin{aligned}
\frac{dI}{dt} &= (1-\pi)I+\beta cI-(\delta+\upsilon)I+(1-\varepsilon)\theta I \\
&= [m(1-\pi)+\beta c-(\delta+\upsilon)+(1-\varepsilon)\theta]I \\
\Rightarrow \frac{1}{I}\frac{dI}{dt} &= m(1-\pi)+\beta c-(\delta+\upsilon)+(1-\varepsilon)\theta.
\end{aligned}$$

Solving for *I*:

$$\int \frac{1}{I}dI = \int [m(1-\pi)+\beta c-(\delta+\upsilon)+(1-\varepsilon)\theta]dt,$$

Then $I = I_0 \exp\left([m(1-\pi)+\beta c-(\delta+\upsilon)+(1-\varepsilon)\theta]t\right).$

If $m(1-\pi)+\beta c-(\delta+\upsilon)+(1-\varepsilon)\theta < 0$, then the number of infectives die exponentially with time. Therefore, there is no infection at infinity (that is, $t \to \infty$), if

$$m(1-\pi)+\beta c-(\delta+\upsilon)+(1-\varepsilon)\theta<0 \Leftrightarrow \frac{m(1-\pi)+\beta c+(1-\varepsilon)\theta}{\delta+\upsilon}<1$$

$$\Rightarrow R_0 = \frac{m(1-\pi)+\beta c+(1-\varepsilon)\theta}{\delta+\upsilon}.$$

We can rewrite I in terms of R_0: $I = I_0 \exp\left(\frac{R_0-1}{T}\right)$.

Since

$$\begin{aligned}
m(1-\pi)+\beta c-(\delta+\upsilon)+(1-\varepsilon)\theta &= \left(\frac{m(1-\pi)+\beta c+(1-\varepsilon)\theta}{\delta+\upsilon}-1\right)(\delta+\upsilon) \\
&= \left(\frac{m(1-\pi)+\beta c+(1-\varepsilon)\theta}{\delta+\upsilon}-1\right)\left(\frac{1}{\delta+\upsilon}\right)^{-1} \\
&= (R_0-1)T^{-1} \\
&= \frac{R_0-1}{T}.
\end{aligned}$$

where $T = \frac{1}{\delta+\upsilon}$ is the time during which people remain infective and

$$R_0 = \frac{m(1-\pi)+\beta c+(1-\varepsilon)\theta}{\delta+\upsilon}.$$

Doubling time, t_d of the epidemic

Consider when $t = t_d, I = 2I_0$ these values can be substituted in the expression $I = e^{(\frac{R_0-1}{T})t}$ so as to solve for doubling time as follows:

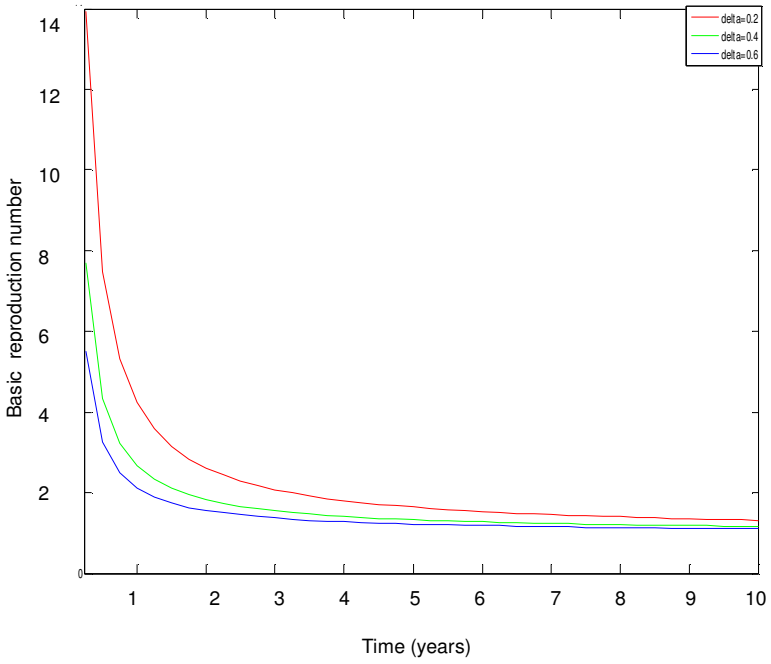

Figure 2. Variation of basic reproductive rate R_0 against doubling time, t_d for different values of delta, δ.

$$I = I_0 e^{(\frac{R_0-1}{T})t} \Rightarrow 2I_0 = I_0 e^{(\frac{R_0-1}{T})t_d}$$

$$\Leftrightarrow t_d = \frac{T \ln 2}{R_0 - 1}.$$

Therefore, doubling time, $t_d = \frac{T \ln 2}{R_0 - 1}$.

Thus, if $R_0 > 1$, the infection triggers an epidemic otherwise its prevalence is zero, that is, 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, increase in the number of sexual partners, results to an increase in the number of infectives 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 Figure 2, 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 of the disease. In addition, for this case the doubling time gets longer. A small increase in the basic reproduction rate results in the reduction of the doubling time which indicates the slow growth of the infection.

STABILITY ANALYSIS

Here, we present the results of stability analysis of the equilibrium points.

Equilibria of the model

The model (Equation 2) has two non-negative equilibrium points namely:

1. $E_0(\frac{Q}{\upsilon}, 0, 0, 0)$, the disease-free equilibrium,
2. $E^*(N^*, I^*, P^*, A^*)$, the endemic equilibrium.

Where $N^* = \beta c \lambda I^*$.

$$I^* = \frac{Q}{\beta c \upsilon \lambda + \alpha \frac{\delta[\mu + \upsilon(1-\sigma)] + \mu m \pi}{(\alpha + \upsilon)(\mu + \upsilon)} - [m + \theta(1-\varepsilon)]}.$$

$$P^* = \frac{(m\pi + \sigma\delta)I^*}{\mu + \upsilon}.$$

$$A^* = \frac{(\delta[\mu + \upsilon(1-\sigma)] + \mu m\pi)I^*}{(\alpha+\upsilon)(\mu+\upsilon)}.$$

where $\lambda = \dfrac{1 + \dfrac{m\pi + \sigma\delta}{\mu+\upsilon} + \dfrac{(\delta[\mu+\upsilon(1-\sigma)]+\mu m\pi)}{(\alpha+\upsilon)(\mu+\upsilon)}}{\beta c + (1-\varepsilon)\theta - (\delta+\upsilon) + m(1-\pi)}.$

λ is positive only when

$$\beta c + (1-\varepsilon)\theta + m(1-\pi) > \delta + \upsilon.$$

It is noted that E^* is positive if

$$\beta c \upsilon\lambda + \alpha\frac{\delta[\mu+\upsilon(1-\sigma)]+\mu m\pi}{(\alpha+\upsilon)(\mu+\upsilon)} > m + \theta(1-\varepsilon).$$

It is shown that equilibrium level of infectives I^* increases as Q or m increases; c or π decreases leading to increase in P^* and A^*. Also if θ increases then, the equilibrium values of I^*, P^* and A^* increase. Thus, an increase in the rate of vertical transmission increases 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 α increases. It is also noted that when the disease remain endemic, the disease induced deaths reduce the equilibrium population size from $\frac{Q}{\upsilon}$ to N^*.

Local stability of the equilibria

Now to determine the local stability of E_0 and E^*, the following variation matrices M_0 and M^* are computed corresponding to equilibrium points E_0 and E^* respectively.

$$M_0 = \begin{bmatrix} -d & (1-\varepsilon)\theta + m & 0 & -\alpha \\ 0 & \beta c + (1-\varepsilon)\theta - (\delta+\upsilon) + m(1-\pi) & 0 & 0 \\ 0 & m\pi + \sigma\delta & -(\mu+\upsilon) & 0 \\ 0 & (1-\sigma)\delta & \mu & -(\alpha+\upsilon) \end{bmatrix}$$

$$M^* = \begin{bmatrix} -\upsilon & (1-\varepsilon)\theta + m & 0 & -\alpha \\ \left(\beta c + (1-\varepsilon)\theta - (\delta+\upsilon) + m(1-\pi)\right)\dfrac{I^*}{N^*} & -\dfrac{\beta c I^*}{N^*} & -\beta c\dfrac{I^*}{N^*} & -\beta c\dfrac{I^*}{N^*} \\ 0 & m\pi + \sigma\delta & -(\mu+\upsilon) & 0 \\ 0 & (1-\sigma)\delta & \mu & -(\alpha+\upsilon) \end{bmatrix}$$

From M_0 it is clear that E_0 is locally asymptotically stable provided $\beta c + (1-\varepsilon)\theta + m(1-\pi) < \delta + \upsilon$, that is, for $R_0 < 1$, the disease dies out and under this condition the endemic equilibrium 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:

$$f(\lambda) = \lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0. \quad (3)$$

where

$$a_1 = \alpha + \mu + 3\upsilon + \frac{\beta c I^*}{N^*},$$

$$a_2 = (\alpha+\upsilon)(\mu+\upsilon) + \upsilon(\alpha+\mu+2\upsilon) + \left(\beta c(\alpha+\mu+\delta+3\upsilon+m\pi+\delta) - [(1-\varepsilon)\theta+m][\beta c + (1-\varepsilon)\theta - (\delta+\upsilon) + m(1-\pi)]\right)\frac{I^*}{N^*},$$

$$a_3 = \upsilon(\alpha+\upsilon)(\mu+\upsilon) + [(1-\sigma)\delta(\mu+2\upsilon)+(m\pi+\sigma\delta)(\alpha+2\upsilon+\mu)+\alpha\mu+\upsilon(2\alpha+2\mu+3\upsilon)]\frac{\beta cI^*}{N^*} + \left[(1-\sigma)\alpha\delta-[(1-\varepsilon)\theta+m](\alpha+\mu+2\upsilon)\right]\left[\beta c+(1-\varepsilon)\theta-(\delta+\upsilon)+m(1-\pi)\right]\frac{I^*}{N^*},$$

$$a_4 = \upsilon\left[(\alpha+\upsilon)(\mu+\upsilon+m\pi+\sigma\delta)+\mu m\pi+\delta(\mu+\delta(1-\sigma))\right]\frac{\beta cI^*}{N^*} + \left[\beta c+(1-\varepsilon)\theta-(\delta+\upsilon)+m(1-\pi)\right] x\left[\alpha(\delta[\mu+(1-\sigma)\upsilon]+\mu m\pi)-[(1-\varepsilon)\theta+m](\mu+\upsilon)(\alpha+\upsilon)\right]\frac{I^*}{N^*}.$$

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 $a_1a_2a_3-a_3^2-a_1^2a_4>0.$

Global stability of the endemic equilibrium

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

Lemma

The region $\Omega=\{(N,I,P,A):\ 0<N\le\overline{N},\ 0<I\le\overline{I},\ 0<P\le\overline{P},\ 0<A\le\overline{A}\}$ is a region of attraction for $\beta c+\theta(1-\varepsilon)+m(1-\pi)>\delta+\upsilon$.

where $\overline{N}=\frac{Q+[(1-\varepsilon)\theta+m]\overline{I}}{\upsilon}$,

$\overline{I}=\frac{Q}{\upsilon}\left(1+\frac{\theta(1-\varepsilon)+m(1-\pi)-(\delta+\upsilon)}{\beta c}\right)$,

$\overline{P}=\frac{(m\pi+\sigma\delta)\overline{I}}{\mu+\upsilon}$ and $\overline{A}=\frac{(\delta[\mu+\upsilon(1-\sigma)]+\mu m\pi)\overline{I}}{(\alpha+\upsilon)(\mu+\upsilon)}$.

Theorem

If the endemic equilibrium E^* exists, then it is globally asymptotically stable provided the following conditions are satisfied in Ω :

$$\frac{\mu^2}{(1-\sigma)\delta}<\frac{2(\alpha+\upsilon)(\mu+\upsilon)}{3(m\pi+\sigma\delta)},\quad k>\max\{q_{11},q_{12}\},$$

where $q_{11}=\frac{3\alpha^2(1-\sigma)\delta N^*}{2\upsilon\beta c(\alpha+\upsilon)}$, $q_{12}=\frac{3N^*l^2}{2\upsilon\beta c}$ and $l=\frac{\beta ck(\overline{I}+\overline{P}+\overline{A})}{N^*\overline{N}}+(1-\varepsilon)\theta+m$.

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

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

where the constants k_1, k_2 and k_3 can be chosen suitably. The derivative of V along the solution of the system (Equation 2) can be written as:

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

After some algebraic calculations, the following is obtained:

$$\frac{dV}{dt}=-\upsilon(N-N^*)^2+\left(\frac{\beta ck(\overline{I}+\overline{P}+\overline{A})}{N^*\overline{N}}+(1-\varepsilon)\theta+m\right)(N-N^*)(I-I^*)-\alpha(N-N^*)(A-A^*)$$

$$-\frac{\beta ck}{N^*}(I-I^*)^2+\left(k_2(m\pi+\sigma\delta)-\frac{\beta ck}{N^*}\right)(I-I^*)(P-P^*)$$

$$-k_2(\mu+\upsilon)(P-P^*)^2+k_3\mu(P-P^*)(A-A^*)$$

$$-k_3(\alpha+\upsilon)(A-A^*)^2-\left(k_3\delta(1-\sigma)-\frac{\beta ck}{N^*}\right)(I-I^*)(A-A^*).$$

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

$$\frac{dV}{dt}=-\frac{1}{2}a_{11}(N-N^*)^2+a_{12}(N-N^*)(I-I^*)-\frac{1}{2}a_{21}(I-I^*)^2$$
$$-\frac{1}{2}a_{11}(N-N^*)^2+a_{13}(N-N^*)(A-A^*)-\frac{1}{2}a_{33}(A-A^*)^2$$
$$-\frac{1}{2}a_{21}(I-I^*)^2+a_{22}(I-I^*)(P-P^*)-\frac{1}{2}a_{31}(P-P^*)^2$$
$$-\frac{1}{2}a_{21}(I-I^*)^2+a_{23}(I-I^*)(A-A^*)-\frac{1}{2}a_{33}(A-A^*)^2$$
$$-\frac{1}{2}a_{31}(P-P^*)^2+a_{32}(P-P^*)(A-A^*)-\frac{1}{2}a_{33}(A-A^*)^2$$

where

$$a_{11}=\upsilon, \qquad a_{12}=\frac{\beta ck(\bar{I}+\bar{P}+\bar{A})}{N^*\bar{N}}+(1-\varepsilon)\theta+m, \quad a_{13}=-\alpha,$$
$$a_{21}=\frac{2\beta ck}{3N^*}, \qquad a_{22}=k_2(m\pi+\sigma\delta)-\frac{\beta ck}{N^*}, \qquad a_{23}=k_3(1-\sigma)\delta-\frac{\beta ck}{N^*},$$
$$a_{31}=k_2(\mu+\upsilon), \quad a_{32}=k_3\mu, \qquad a_{33}=\frac{2}{3}k_3(\alpha+\upsilon).$$

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

$$\left.\begin{aligned} &a_{12}^2-a_{11}a_{21}<0, \quad a_{13}^2-a_{11}a_{33}<0, \quad a_{22}^2-a_{21}a_{31}<0,\\ &a_{23}^2-a_{21}a_{33}<0, \quad a_{32}^2-a_{31}a_{33}<0. \end{aligned}\right\} \tag{4}$$

Now choosing $k_2=\frac{\beta ck}{(m\pi+\sigma\delta)N^*}$ and $k_3=\frac{\beta ck}{(1-\sigma)\delta N^*}$ the condition (Equation 4) give;

$\frac{\mu^2}{(1-\sigma)\delta}<\frac{2(\alpha+\upsilon)(\mu+\upsilon)}{3(m\pi+\sigma\delta)}$ and $k>\max\{q_{11},q_{12}\}$.

where $q_{11}=\frac{3\alpha^2(1-\sigma)\delta N^*}{2\upsilon\beta c(\alpha+\upsilon)}$, $q_{12}=\frac{3N^*l^2}{2\upsilon\beta c}$ and $l=\frac{\beta ck(\bar{I}+\bar{P}+\bar{A})}{N^*\bar{N}}+(1-\varepsilon)\theta+m.$

Hence, is a Lyapunov function with respect to E^* whose domain contains Ω proving the theorem.

NUMERICAL ANALYSIS

We give numerical simulation of the equilibrium and stability conditions of the model (Equation 2). Using the set of parameter values:

$$Q=2000, \upsilon=\frac{1}{70}, \ c=25, \alpha=1, \ \beta=0.05,$$
$$\varepsilon=0.4, \theta=0.01, \mu=0.5, m=0.01, \pi=0.3$$

and the initials:

$$N_0=12700, \qquad S_0=10000,$$
$$I_0=2000, P_0=500, \textit{and } A_0=200$$

the endemic equilibrium values using MatLab are computed as $S^*=2045$, $I^*=9791$, $P^*=819$ and $A^*=1949$.

The results of numerical simulation are displayed graphically in Figures 3 to 10. In Figure 3, the distribution of population with time is shown in different classes without immigration into the population, that is, $Q=0.0, \theta=0.0$ and $m=0.0$ into the population. It is seen that in the absence of immigration into the community, the susceptible population decreases continuously as the population is closed which result in

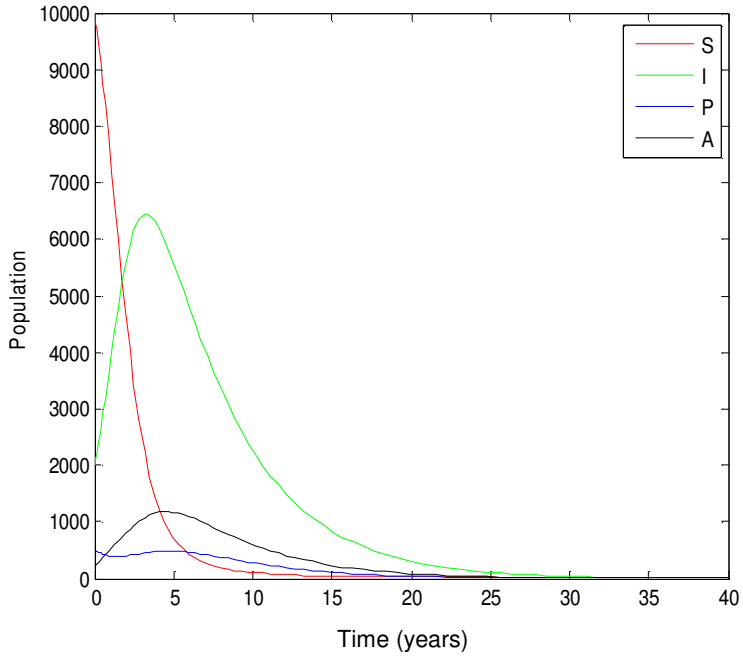

Figure 3. Variation of population in different classes for $Q = 0.0$ and $m = 0.0$.

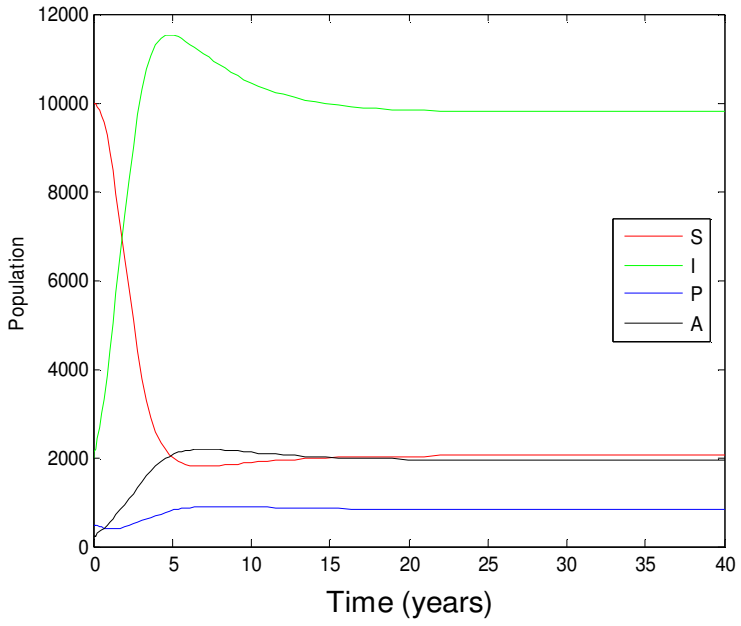

Figure 4. Variation of population in different classes for $Q = 2000$, $\theta = 0.01$ and $m = 0.01$

the increase of infective population first and then, it decreases as all infectives subsequently develop AIDS and then, die out by disease-induced deaths. Thus, the total population is eradicated after some time.

Figure 4 shows the variation of population in all classes with both immigration of susceptible (I) and infected individuals both infectives and pre-AIDS patients. It follows that susceptibles decrease continuously due to

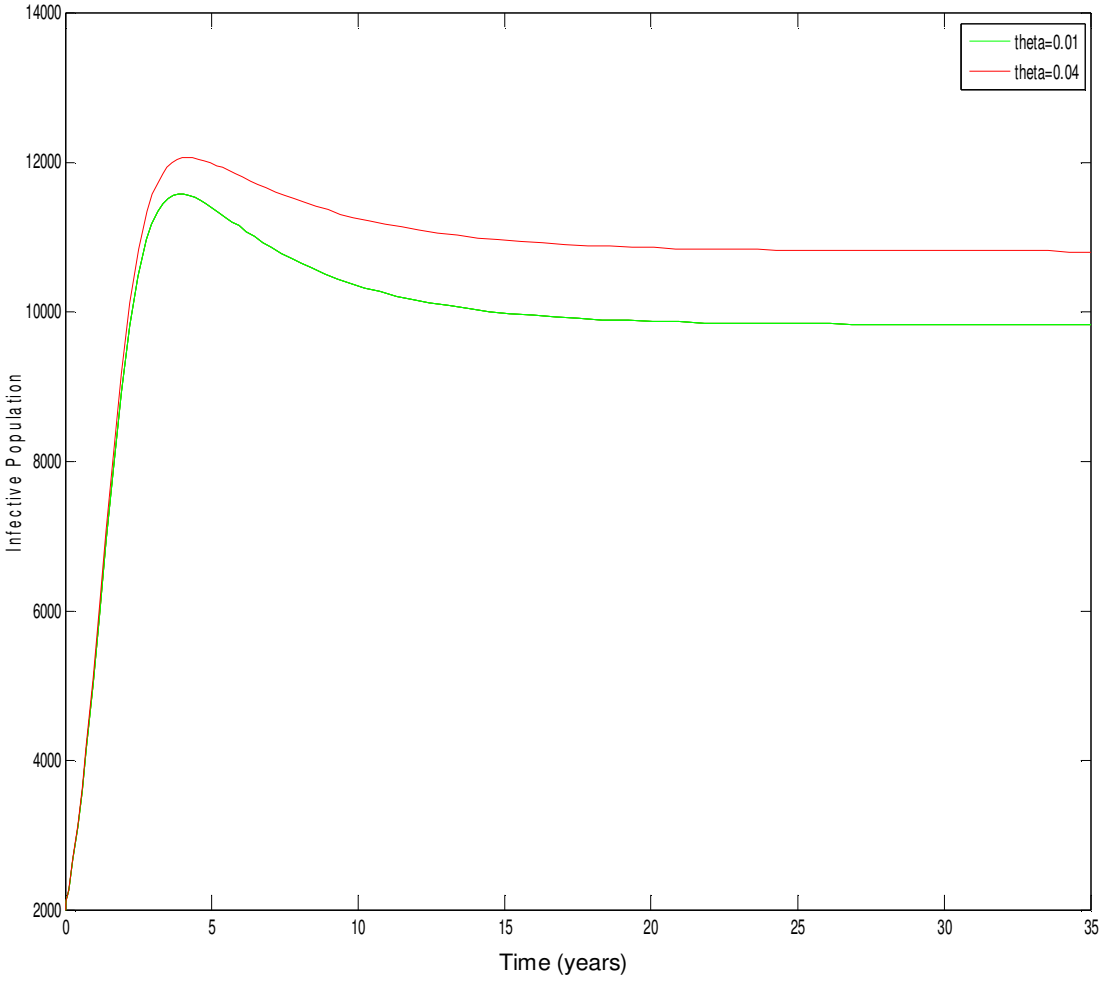

Figure 5. Variation of infective population (I) for different values of theta, θ.

potential infections from infectives; therefore, infection becomes more endemic and always persists in the population. On comparing Figures 3 and 4, it is noted that the increase in the rate of immigration into community increases the AIDS population. Thus, if the rate of immigration is restricted into susceptible community, the spread of the disease can be kept under control.

The role of vertical transmission in the model is revealed by the rate of recruitment of infected children directly into infective class, explicitly shown in Figures 5 and 6. It is seen that as the rate of infected children born increases, the infective population also increase and as much 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, but they do not take part in the horizontal transmission as they are sexually inactive. Thus, if the births of infected children are controlled by way of promoting condom use or other control mechanisms, the overall infective population will

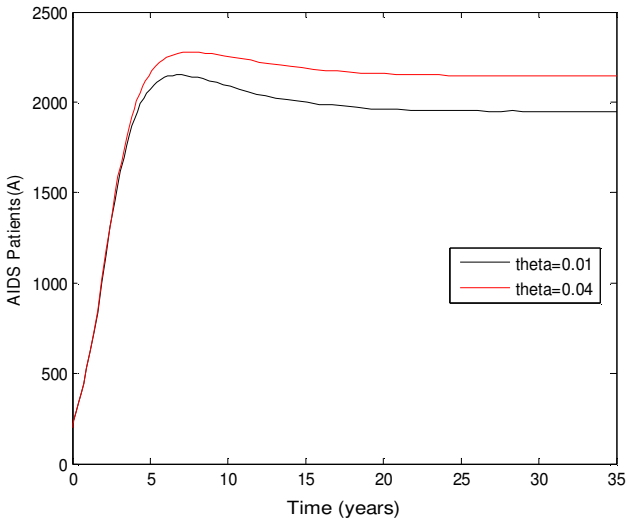

Figure 6. Variation of AIDS patient population for different values of theta, θ.

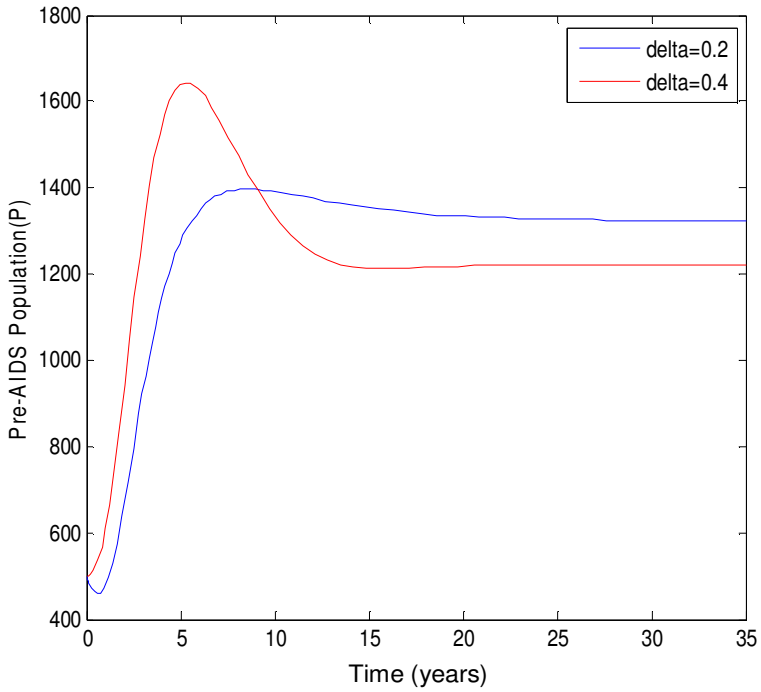

Figure 7. Variation of infected population for different values of delta,δ.

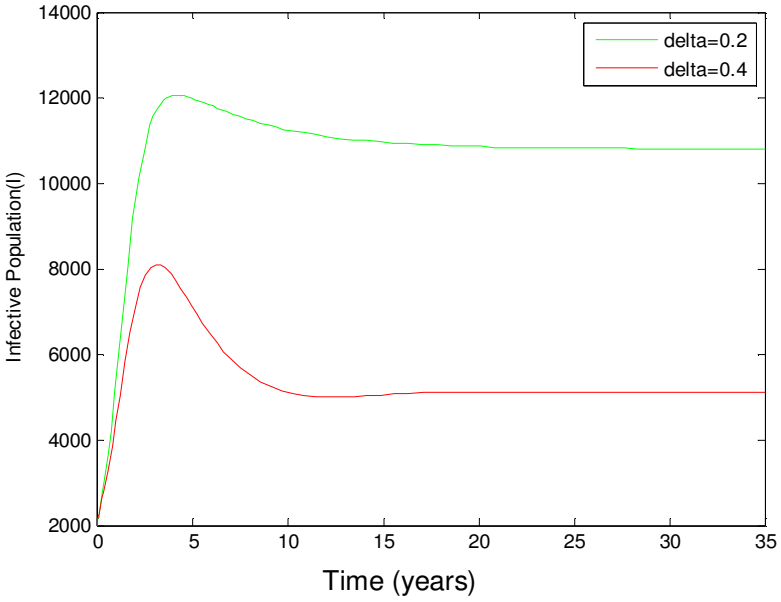

Figure 8. Variation of Pre-AIDS population for different values of delta,δ.

remain under control. This will help in reducing the AIDS population.

Figures 7 to 9 depict the variation of infectives, pre-AIDS and AIDS population, respectively with time for different values of movement rate delta, δ. It is seen that with increase in the value of movement rate, δ, the infective population decreases which in turn increases the pre-AIDS and AIDS population. This is expected, because of shorter incubation period.

In Figure 10, the effect of disease induced death rate α

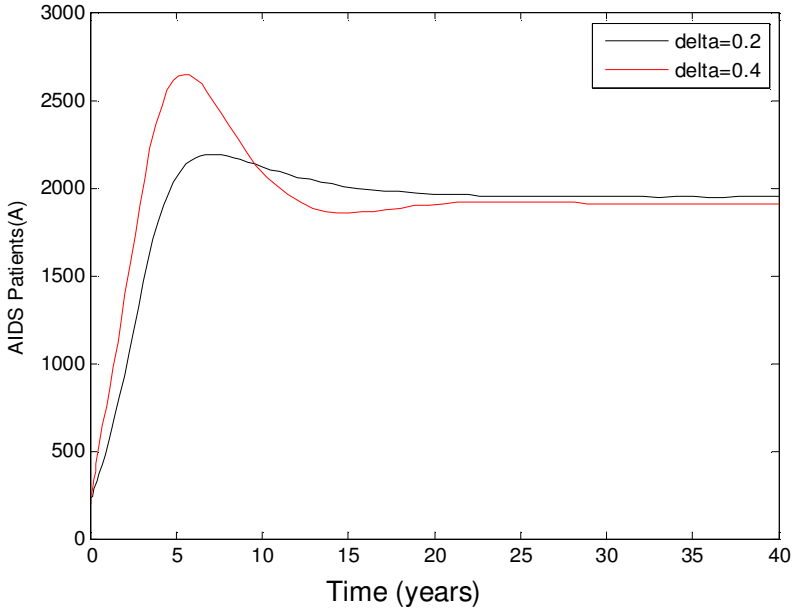

Figure 9. Variation of AIDS patients population for different values of delta, δ.

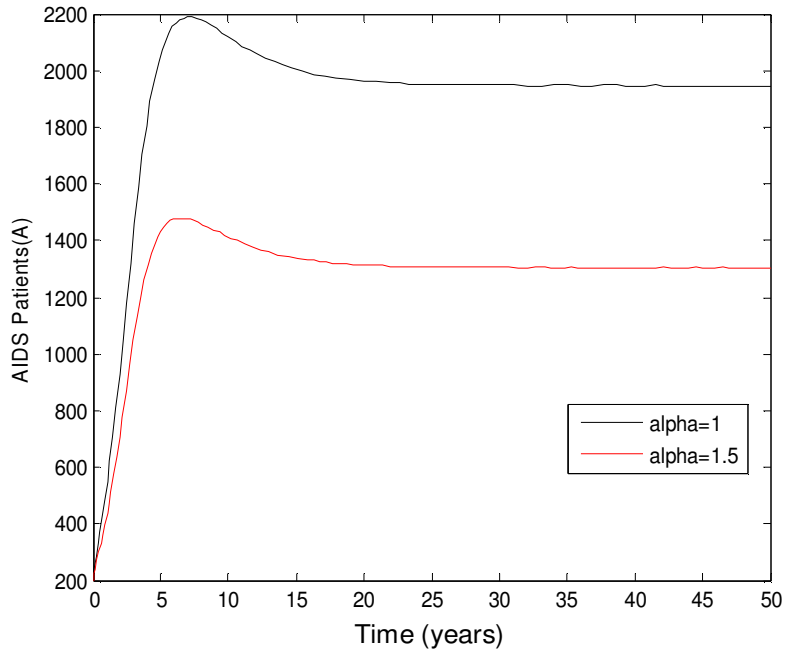

Figure 10. Variation of AIDS patients' population for different values of alpha, α.

is shown and it is found that as α increases, the population of AIDS patients' decreases. The number of susceptibles (S), is lowered as recruitment of infected immigrants is intensified in the population, this is because more susceptible individuals are infected by HIV due to increase in contact rate. This is observed in Figure 11, by varying the values of m and keeping $\pi > 0$ constant, therefore, susceptibles can be maintained at desired level by restricting the number of infected immigrants.

Also, an increase of infectives through immigration of

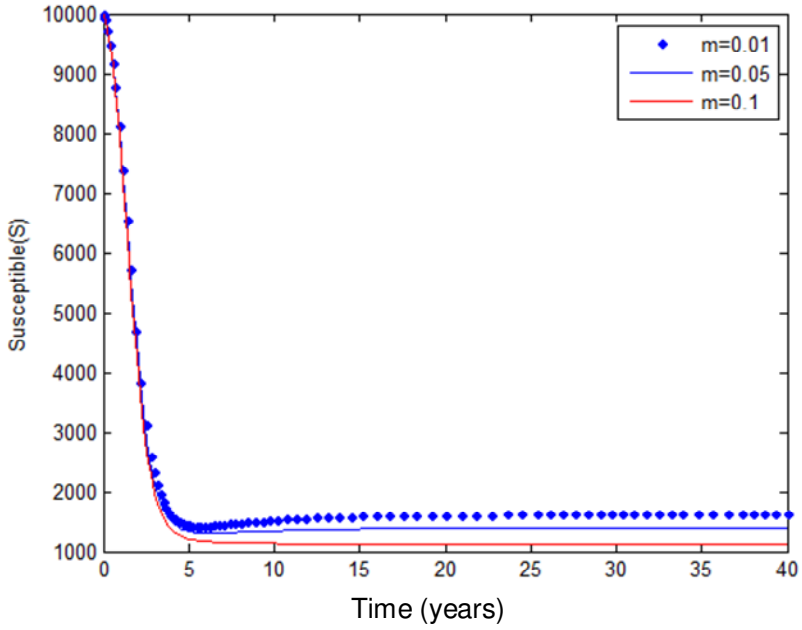

Figure 11. Variation of susceptible (S) population for different values of m.

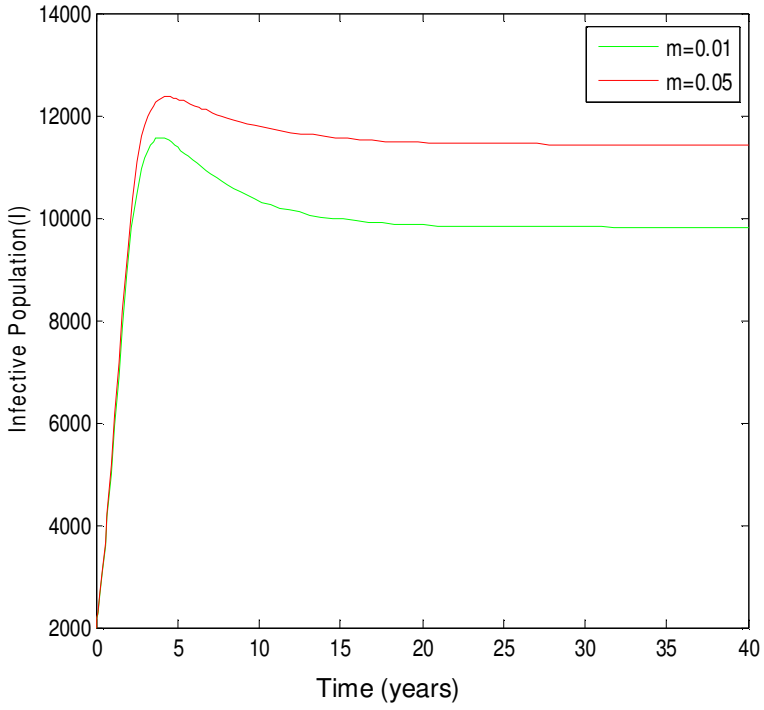

Figure 12. Variation of infected population for different values.

infected individuals accelerates the infectives class as simulated in Figure 12: similar character is observed to pre-AIDS and AIDS patients simply because the infectives eventually develop into pre-AIDS or AIDS class depending on viral counts, as seen in both Figures 13 and 14 population for different values of m.

A further increase in infected immigrants into the population, t hat is $m \to \infty$ leads to uncontrollable

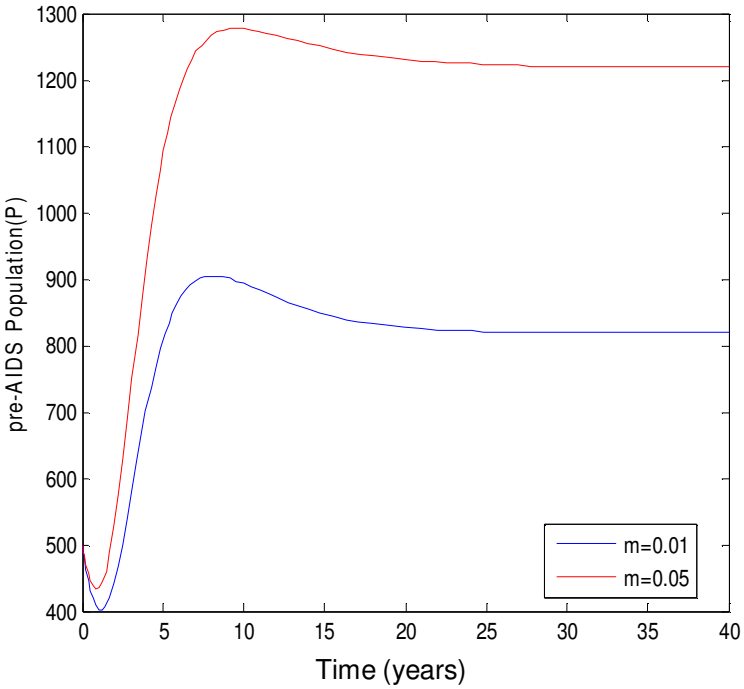

Figure 13. Variation of pre-AIDS population for different values of m.

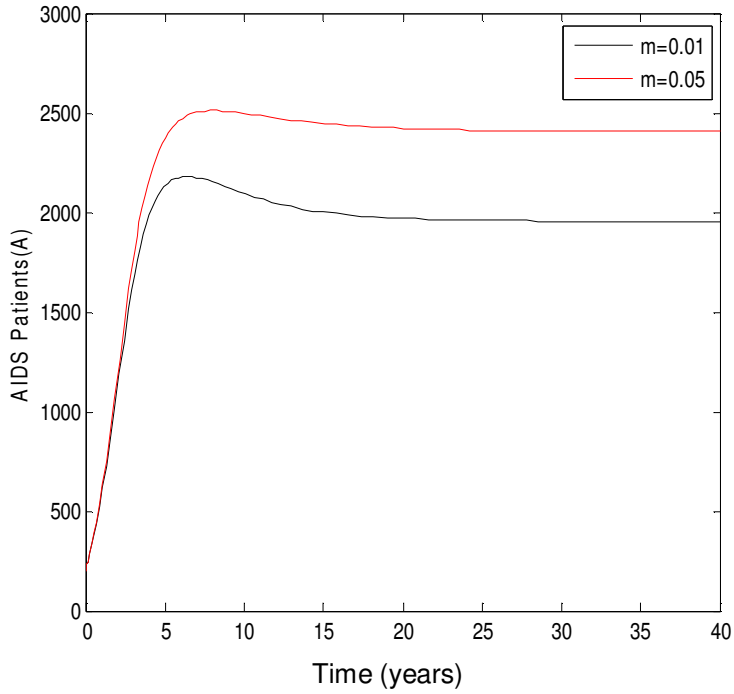

Figure 14. Variation of AIDS population for different values of m.

disease situation. As a consequence, despite direct increase of infectives through immigration, this leads to high rate of increase in infected individuals through vertical and horizontal transmission. As a result, this

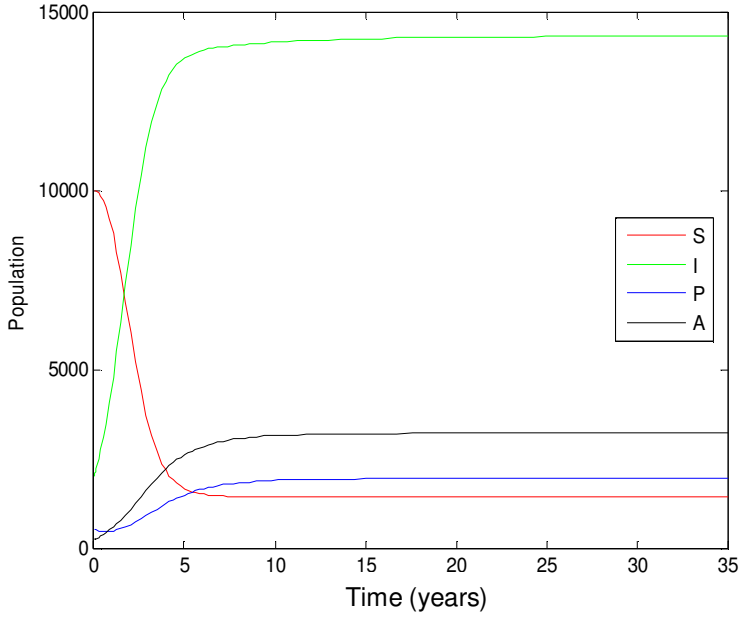

Figure 15. Variation of population in different classes for $Q = 2000$, $\theta = 0.01$ and $m = 0.1$.

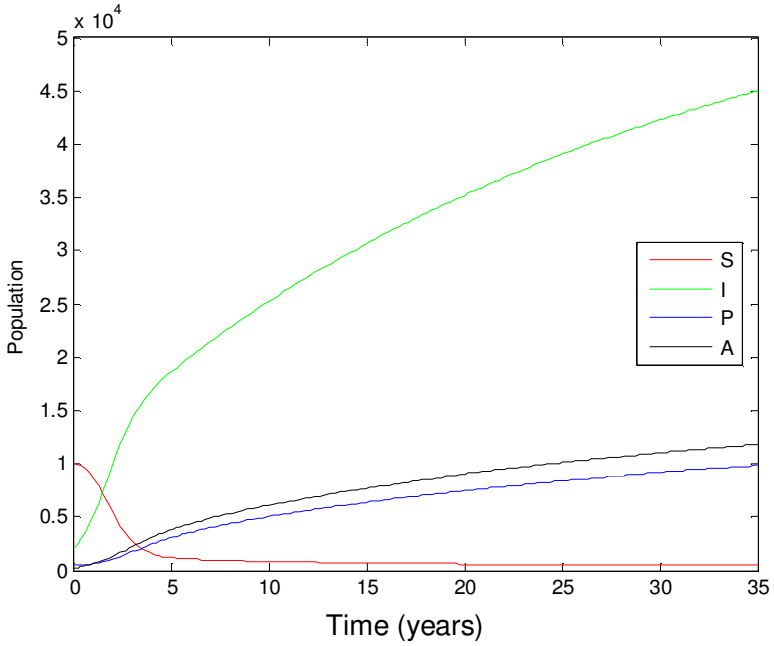

Figure 16. Variation of population in different classes for $Q = 2000$, $\theta = 0.01$ and $m = 0.25$.

decreases the susceptible population; when the value of m is high enough, the susceptible population size becomes negligible, as it is seen in Figures 15 to 17. On the other hand, the value of $R_0 = \frac{m(1-\pi) + \beta c + (1-\varepsilon)\theta}{\delta + \upsilon}$ for $\pi \neq 0$ increases indefinitely as m increases leading to $R_0 > 1$ in which the

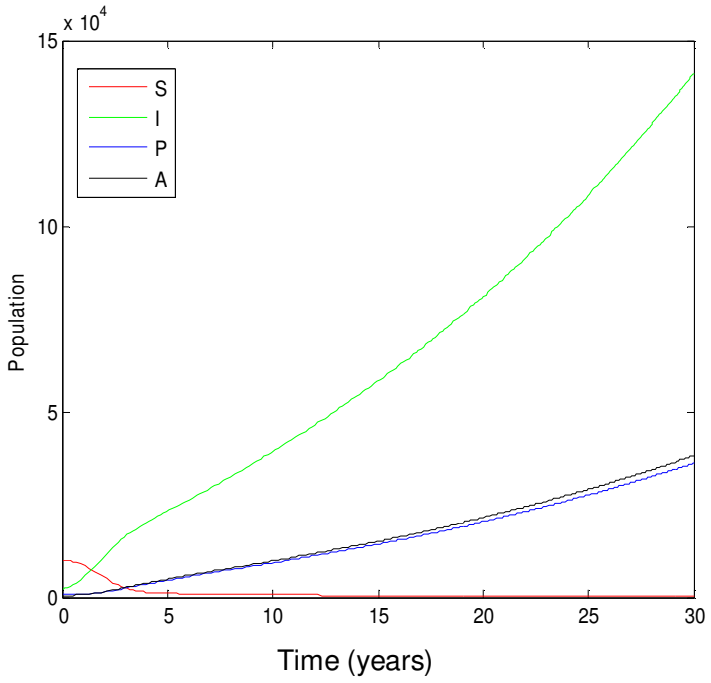

Figure 17. Variation of population in different classes for $Q = 2000$, $\theta = 0.01$ and $m = 0.35$.

disease becomes endemically uncontrollable, because it spreads without bound. Therefore, with no restriction on infected immigrants will lead to negligible size of susceptibles in the population relative to other classes of population, hence the whole population become HIV/AIDS victims.

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.

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 susceptibles (S), infectives (I) and pre-AIDS patients (P) of the population; with vertical transmission under the assumption of 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. However, as long as there are infective immigrant joining the population, the disease free equilibrium will always be unattainable, since the parameter $m > 0$ will always make $R_0 > 1$. When $R_0 > 1$, the disease becomes endemic. It is found that an increase in the rate of vertical transmission or infectives through immigration lead to increase in the population of infectives which in turn increases the pre-AIDS and AIDS population. As a result, infective immigrants contribute to the spread of HIV both horizontal and vertical.

Thus, we need to control the vertical transmission of HIV by suppressing horizontal transmission; including condom use and other safer sexual contact, the infective immigrants should be restricted in such a way that only susceptibles are allowed to immigrate into the community. By simulation also, it is shown that by controlling the rate of vertical transmission, the spread of the disease can be reduced significantly. 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 should be restricted as well, and consequently the equilibrium values of infective and AIDS population can be maintained at desired levels.

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