ISSN 1 746-7233, England, UK
World Journal of Modelling and Simulation
Vol. 7 (2011) No. 3, pp. 230-240

An HIV/AIDS model with vertical transmission and time delay*

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(*Received November 14 2010, Accepted May 17 2011*)

Abstract. We propose an HIV/AIDS model with vertical transmission and the period of sexual maturity of infected newborns which is incorporated in the model as a time delay. We have divided the sexually mature population into three subclasses: the susceptibles, the infectives and the AIDS population. Susceptibles are assumed to become HIV infected via sexual contacts with infectives. Model is analyzed using stability theory of differential equations. Both the infection-free and the endemic equilibria are found and their stability is investigated. Using Lyapunov functional approach, the sufficient conditions for global stability of the endemic equilibrium are obtained. It is shown that as the maturity period of infected newborns decreases, number of infectious individuals increases which in turn increases the AIDS population. Numerical simulations are also carried out to investigate the influence of key parameters on the spread of the disease, to support the analytical conclusion and illustrate possible behavioral scenario of the model.

Keywords: HIV/AIDS, vertical transmission, maturity period, time delay, local stability, global stability

1 Introduction

The study of HIV/AIDS transmission 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, delay models give better compatibility with reality, as they capture the dynamics from the time of infection to the infectiousness. Many models available in the literature represent dynamics of disease by system of nonlinear differential equations without delay. However, inclusion of delays in such models make them more realistic. Mathematical models incorporating time delay have been thoroughly investigated in early eighties of the twentieth century[11]. Recently delay has also been applied to biological models[15, 17, 19]. Most biological systems have time delays inherent in them, yet few scientists formulate models with time lags due to the complexity they introduce and also for mathematical convenience and tractability[7]. A brief comment on the related works provides the context for this paper. It is well known that the dynamical behaviors (including stability, persistence, etc.) of population models with time delay have become a subject of intense research[1, 12, 13]. Many kinds of SIR and SIRS models have embodied these properties[2, 4, 5, 8]. In particular, Wang and Ma [13] developed a delayed epidemiological model with standard mass action type interaction and studied the global stability of the model. Khan and Krishnan [9] examined a SIR model by introducing time delay in the recruitment of infected persons, and showed that the introduction of a time delay into the transmission term can destabilize the system and periodic solution can arise by Hopf bifurcation. Kyrychko and Blyuss [16] studied a time delayed SIR model with general incidence term and the time delay representing the temporal immunity period. Yoshida and Hara [8] presented a delayed SIR model with density dependent birth and death rate and studied the stability behavior of the model. Nevertheless, we note that both the local and global stability of the endemic equilibrium is

* One of the authors (DS) is thankful to the University Grants Commission, New Delhi for financial support in the form of JRF received through grant F. No.10-2(5)2003(I)-EU.II.

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shown in very few studies. Very little attention has been paid to incorporate time delay in HIV/AIDS models. Kovacs [6] considered an HIV/AIDS model with delay and used the delay as a bifurcation parameter to study the possibility of a periodic solution. Mukandavire et al. [21] presented an HIV/AIDS model with explicit incubation period as a system of discrete time delay and observed the impact of epidemic using the demographical and epidemiological parameters for Zimbabwe. The model presented here differs from previous ones in many aspects: we assume a constant flow of susceptible immigrants, vertical transmission of infectives and time delay as a length of maturity period. Our objective, however, is to study the role of time delay on the sexual maturity of infected newborns through vertical transmission on the spread of HIV/AIDS. In view of the above, in this paper, we have proposed and analyzed a simple nonlinear mathematical model to study the spread of HIV/AIDS with vertical transmission incorporating delay as a period of sexual maturity of infected newborns in an adult population of variable size structure. The numerical analysis of the proposed model is also carried out to investigate the influence of some important parameters on the spread of the disease.

The organization of the paper is as follows. In the next section we present the model with delay. Section 3 presents the invariant region and positivity of solutions of the model system. In Section 4, we analyze the equilibrium points of model, their local and global stability analysis. Section 5 presents the numerical simulations of the model system followed by conclusion in Section 6.

2 Mathematical model

We propose a simple HIV/AIDS model which incorporates time delay during which a newly born infected child attains sexual maturity and becomes infectious. In this model, the sexually mature population is divided into three subclasses: the susceptibles, the infectives (also assumed to be infectious) and the AIDS population whose numbers are denoted by S, I and A. The number of total population is denoted by $N(t)$, at any time t. In the model, we assume that the susceptibles become HIV infected via sexual contacts with infectives. It is also assumed that all newborns are infected at birth ($\tau = 0, \varepsilon = 1$)[10]. We do not consider direct inflow of other infected persons except through vertical transmission as our purpose is to study the role of delay, modeled as a period of sexual maturity of infected newborns. In the model, we have assumed that a fraction of infected newborns, who sustain treatment, joins the infective class while the others, who do not sustain treatment, joins AIDS class after getting sexual maturity. The infectives through vertical transmission at any time t is given by $\gamma\varepsilon I(t-\tau)$, because those infected at time $(t-\tau)$ becomes infectious at time τ later, if they do not develop AIDS by that time. The fraction of infectives which develop AIDS during the period of getting sexual maturity, if they survive the maturity period joins the AIDS class. However, for the model to be biologically reasonable, it may be more realistic to assume that not all those infected will survive after time τ units, and this claim supports the introduction of the survival term $e^{-d\tau}$[3, 14]. Thus, in our model the term $\gamma\varepsilon I(t-\tau)e^{-d\tau}$ represents the introduction of infective persons who survive the maturity period τ in which the time taken to become infectious is τ. Here $e^{-d\tau}$ represents the probability that an individual survives the maturity period $[t-\tau, t]$ such that $0 < e^{-d\tau} \leq 1$. It is also assumed that some of the infectives move to AIDS class with a rate coefficient δ to develop full blown AIDS.

With the above considerations and assumptions, the spread of the disease is assumed to be governed by the following system of nonlinear ordinary differential equations:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = Q_0 - \frac{\beta SI}{N} - dS, \tag{1}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \gamma\varepsilon I(t-\tau)e^{-d\tau} + \frac{\beta SI}{N} - (d+\delta)I, \tag{2}$$

$$\frac{\mathrm{d}A}{\mathrm{d}t} = \delta I + \gamma(1-\varepsilon)I(t-\tau)e^{-d\tau} - (d+\alpha)A, \tag{3}$$

$$S(0) = S_0 > 0, \quad I(0) = I_0 > 0, \quad A(0) = A_0 \geq 0,$$

where Q_0 is the constant rate of immigration of susceptibles, β is the contact rate of susceptibles with infectives, d is the natural death rate coefficient in all the classes, α is the disease related death rate constant of AIDS patients, γ is the birth rate coefficient of infected newborns, ε is the fraction of infected newborns

joining the infective class after getting sexual maturity to become infectious and the remaining part $(1-\varepsilon)$ of the infected newborns joins the AIDS class after getting sexual maturity $(0 \leq \varepsilon \leq 1)$. It is assumed that all the dependent variables and parameters of the model are non-negative. It is remarked here that in the modeling process, we have considered the constant immigration of sexually mature susceptibles only, ignoring the recruitment of newly born susceptibles either in susceptible class itself or from the infective class to avoid mathematical complexity[10]. It may further be noted that AIDS patients are generally exposed, isolated and may be sexually inactive and hence they do not take part in sexual interaction for fear of transmitting the disease to the offspring and therefore, we have not considered their flow from AIDS class to the infective class through vertical transmission. However, in some recent studies this aspect has been taken into account[18, 20]. The model can be further generalized by taking the above aspects into account.

Since we assume that the total population is given by $N(t) = S(t) + I(t) + A(t)$, the above equations can now be written as,

$$\frac{\mathrm{d}N}{\mathrm{d}t} = Q_0 + \gamma I(t-\tau)e^{-d\tau} - dN - \alpha A, \tag{4}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \gamma\varepsilon I(t-\tau)e^{-d\tau} + \frac{\beta I(N-I-A)}{N} - (d+\delta)I, \tag{5}$$

$$\frac{\mathrm{d}A}{\mathrm{d}t} = \delta I + \gamma(1-\varepsilon)I(t-\tau)e^{-d\tau} - (d+\alpha)A, \tag{6}$$

$$N(0) = N_0 > 0, \quad I(0) = I_0 > 0, \quad A(0) = A_0 > 0.$$

3 Invariant region

Lemma 1. *All solutions of the model system Eqs. (4) ∼ (6) starting in R^3_+ are bounded and eventually enter the attracting set Ω.*

$$\Omega = \{(N, I, A) \in R^3_+ : \frac{Q_0}{\alpha + d} < N \leqslant \overline{N}, 0 \leqslant I \leqslant \overline{I}, 0 \leqslant A \leqslant \overline{A}\}, \tag{7}$$

where

$$\overline{N} = \frac{Q_0 + \gamma\overline{I}}{d}, \quad \overline{I} = \frac{Q_0(\beta + \gamma\varepsilon - (d+\delta))}{d\beta}, \quad \overline{A} = \frac{\delta\overline{I} + \gamma(1-\varepsilon)\overline{I}}{\alpha + d}.$$

Proof. Continuity of right hand side of system Eqs. (4) ∼ (6) and its derivative imply that the model is well posed for $N(t) > 0$. The invariant region where solutions exist (and are biologically relevant) is obtained as follows,

$$\frac{Q_0}{\alpha + d} \leqslant \liminf N(t) \leqslant \limsup N(t) \leqslant \overline{N} = \frac{Q_0 + \gamma\bar{I}}{d}(ast \to \infty).$$

The bounds for N, I and A can be easily obtained from model Eqs. (4) ∼ (6). Since $N(t) > 0$ on $[-\tau, 0]$ by assumption, $N(t) > 0$ for all $t \geq 0$. Therefore, from Eq. (4) above, $N(t)$ cannot blow up to infinity in finite time. The model system is dissipative (solutions are bounded) and consequently, the solution exists globally for all $t > 0$ in the invariant and compact set Ω.

As N tends to zero, $S(t)$, $I(t)$, and $A(t)$ also tend to zero. Hence, each of these terms tends to zero as $N(t)$ does. It is, therefore, natural to interpret these terms as zero at $N(t) = 0$.

3.1 Positivity of solutions

The presented model describes a human population and therefore, it is important to prove that all quantities (susceptibles, infectives and AIDS patients) will be positive for all times. In other words, we want to prove that all solutions of the system Eqs. (4) ∼ (6) with positive initial data will remain positive for all times $t > 0$[16].

Lemma 2. *Let the initial data be* $N(0) = S_0 > 0, I(0) = I_0(u) \geq 0, A(0) = A_0(u) \geq 0$ *for all* $u \in [-\tau, 0]$, *with* $I_0(0) > 0$. *Then, the solution* $(S(t), I(t), A(t))$ *of the model remain positive for all time* $t > 0$.

Proof. From Eq. (5), we have

$$\frac{\mathrm{d}I(t)}{\mathrm{d}t} = \gamma\varepsilon I(t-\tau)e^{-d\tau} + \frac{\beta I(t)(N(t) - I(t) - A(t))}{N(t)} - (d+\delta)I(t),$$
$$\frac{\mathrm{d}I(t)}{\mathrm{d}t} \geq -(\delta + d)I(t).$$

From which we get,

$$I(t) \geq k_1 e^{-(\delta+d)t} > 0,$$

where k_1 is a constant of integration. A similar reasoning on the remaining equations shows that they are always positive in Ω for $t > 0$.

4 Equilibrium and stability analysis

In this section, we analyze the equilibrium points of the system Eqs. (4) $\sim$ (6) and their stability. There are only two types of physically as well as biologically relevant equilibria namely
(1) $E_0(Q_0/d, 0, 0)$, the infection-free equilibrium
(2) $E^*(N^*, I^*, A^*)$, the endemic equilibrium where the values of N^*, I^* and A^* are given in Section 4.2.

4.1 Infection-free equilibrium

The existence of infection-free equilibrium E_0 is obvious and it exists without any condition. The Jacobian matrix corresponding to system Eqs. (4) $\sim$ (6) about E_0 is obtained as follows:

$$M_0 = \begin{bmatrix} -d & -\gamma e^{-d\tau} & -\alpha \\ 0 & \gamma\varepsilon e^{-d\tau} + \beta - (d+\delta) & 0 \\ 0 & \delta + \gamma(1-\varepsilon)e^{-d\tau} & -(\alpha + d) \end{bmatrix}$$

Since all the parameters of the model are assumed to be non-negative, therefore, for a infection-free equilibrium E_0 to be locally asymptotically stable, the following condition must be satisfied,

$$\gamma\varepsilon e^{-d\tau} + \beta < (\delta + d). \tag{8}$$

The effect of sexual maturity period of infected newborns via vertical transmission can be seen through basic reproduction number R_0 with delay[21] defined as,

$$R_0 = \frac{\gamma\varepsilon e^{-d\tau} + \beta}{\delta + d}. \tag{9}$$

This threshold quantity R_0 for disease control defines the average number of secondary infections generated by a typical infected individual in a wholly uninfected population in a steady demographic state. Thus, using R_0 we get the following result indicating the stability of E_0.

Lemma 3. *The infection-free equilibrium is locally asymptotically stable if* $R_0 < 1$ *and unstable if* $R_0 > 1$.

4.2 Existence of the endemic equilibrium

In the section 4.1, we have shown that the system Eqs. (4) $\sim$ (6) has an infection-free steady state which is locally asymptotically stable under the condition Eq. (8) i.e.$R_0 < 1$. When $R_0 > 1$, the infection-free equilibrium is unstable and system Eqs. (4) $\sim$ (6) has a non-trivial endemic equilibrium $E^*(N^*, I^*, A^*)$. This equilibrium can be obtained by solving the following set of algebraic equations;

$$Q_0 + \gamma I(t-\tau)e^{-d\tau} - dN - \alpha A = 0, \tag{10}$$

$$\gamma\varepsilon I(t-\tau)e^{-d\tau} + \frac{\beta I(N-I-A)}{N} - (d+\delta)I = 0, \tag{11}$$

$$\delta I + \gamma(1-\varepsilon)I(t-\tau)e^{-d\tau} - (d+\alpha)A = 0. \tag{12}$$

From Eqs. (10) $\sim$ (12), we get

$$N^* = \frac{\beta(1+p)Q_0}{\beta(1+p)d + (\gamma\varepsilon e^{-d\tau} + \beta - (\delta+d))q},$$
$$I^* = \frac{Q_0(\gamma\varepsilon e^{-d\tau} + \beta - (\delta+d))}{\beta(1+p)d + (\gamma\varepsilon e^{-d\tau} + \beta - (\delta+d))q},$$
$$A^* = pI^*,$$

where

$$p = \frac{\delta + \gamma(1-\varepsilon)e^{-d\tau}}{\alpha+d}, \quad q = \frac{\alpha\delta - \gamma(d+\varepsilon)e^{-d\tau}}{\alpha+d}.$$

It may be noted that the endemic equilibrium will be positive if

$$\alpha\delta > \gamma(d+\varepsilon)e^{-d\tau} \tag{13}$$

4.3 Local stability of the endemic equilibrium

In this section, we study the local stability of the nontrivial endemic equilibrium E^*. For this, we obtain the Jacobian matrix corresponding to system Eqs. (4) $\sim$ (6) about E^* as follows:

$$M^* = \begin{bmatrix} -d & -\gamma e^{-(\lambda+d)\tau} & -\alpha \\ a_1 & \gamma\varepsilon e^{-(\lambda+d)\tau} - a_2 & -a_3 \\ 0 & \delta + \gamma(1-\varepsilon)e^{-(\lambda+d)\tau} & -(\alpha+d) \end{bmatrix}$$

where

$$a_1 = \frac{\beta I^*(I^*+A^*)}{N^{*2}}, \quad a_2 = \gamma\varepsilon e^{-d\tau} + \frac{\beta I^*}{N^*}, \quad a_3 = \frac{\beta I^*}{N^*}.$$

The characteristic equation corresponding to the Jacobian matrix M^* is given by $\det(\lambda I - M^*) = 0$ where I is the unit matrix. Thus, we get

$$\lambda^3 + m_2\lambda^2 + m_1\lambda + m_0 + (n_2\lambda^2 + n_1\lambda + n_0)e^{-\lambda\tau} = 0, \tag{14}$$

where

$$m_2 = 2d + \alpha + a_2, \quad m_1 = a_2(2d+\alpha) + \delta a_3 + d(\alpha+d), \quad m_0 = a_2 d(d+\alpha) + \delta d a_3 + \alpha a_1\delta,$$
$$n_2 = -\gamma\varepsilon e^{-d\tau}, \quad n_1 = \{-\gamma\varepsilon(\alpha+2d) + a_3\gamma(1-\varepsilon) - a_1\gamma\}e^{-d\tau},$$
$$n_0 = \{-\gamma\varepsilon(\alpha+d)d + a_3 d\gamma(1-\varepsilon) - a_1\gamma(\alpha+d) + \alpha a_1\gamma(1-\varepsilon)\}e^{-d\tau}.$$

We note that $m_2 > 0$, $m_1 > 0$, $m_0 > 0$ and $n_2 < 0$ whereas n_1 and n_0 may be positive or negative. For $\tau = 0$ we may state the following results that follow directly from Eq. (14) and the Routh-Hurwitz criteria.

Theorem 1. *When $\tau = 0, \varepsilon = 1$, the characteristic Eq. (14) yields*

$$\lambda^3 + b_2\lambda^2 + b_1\lambda + b_0 = 0, \tag{15}$$

where

$$\begin{aligned}
b_2 &= 2d + \alpha + \frac{\beta I^*}{N^*} > 0,\\
b_1 &= \frac{\beta I^*}{N^*}(2d+\alpha) + \delta\frac{\beta I^*}{N^*} + d(\alpha+d) - \frac{\beta I^*(I^*+A^*)}{N^{*2}}\gamma,\\
b_1 &> \frac{\beta I^*}{N^*}(2d+\alpha) + \delta\frac{\beta I^*}{N^*} + d(\alpha+d) - \frac{\beta I^*}{N^*}\gamma,\\
b_1 &> d(\alpha+d) + \frac{\beta I^*}{N^*}(2d+\alpha+\delta-\gamma) > 0,\\
b_0 &= d(d+\alpha)\frac{\beta I^*}{N^*} + \delta d\frac{\beta I^*}{N^*} + \alpha\frac{\beta I^*\,(I^*+A^*)}{N^{*2}}\delta - \gamma(\alpha+d)\frac{\beta I^*\,(I^*+A^*)}{N^{*2}},\\
b_0 &> d(d+\alpha)\frac{\beta I^*}{N^*} + \delta d\frac{\beta I^*}{N^*} + \alpha\frac{\beta I^*\,(I^*+A^*)}{N^{*2}}\delta - \gamma(\alpha+d)\frac{\beta I^*}{N^*},\\
b_0 &> \left(d\,(d+\alpha) + \delta d - \gamma\,(d+\alpha)\right)\frac{\beta I^*}{N^*} + \alpha\delta\frac{\beta I^*\,(I^*+A^*)}{N^{*2}}.
\end{aligned}$$

The equilibrium E^* is locally asymptotically stable if and only if the following inequalities holds:

$$b_0 > 0, \quad b_1 b_2 > b_0. \tag{16}$$

Theorem 2. *When $\tau = 0$, the equilibrium E^* is unstable if*

$$b_1 < 0 \quad \textit{or} \quad b_0 < 0.$$

The main purpose of this article is to study the stability behavior of E^* in the case $\tau \neq 0$. For this purpose, we determine the sign of real parts of the zeros of Eq. (14) that characterizes the stability behavior of E^*. Obviously, $i\eta$ ($\eta > 0$) is the root of Eq. (14) if and only if η satisfies

$$-i\eta^3 - m_2\eta^2 + m_1 i\eta + m_0 = -(-n_2\eta^2 + n_1 i\eta + n_0)(\cos(\eta\tau) - i\sin(\eta\tau)).$$

Separating the real and imaginary parts, we have

$$-m_2\eta^2 + m_0 = -(-n_2\eta^2 + n_0)(\cos(\eta\tau) - i\eta\sin(\eta\tau)), \tag{17}$$

$$-\eta^3 + m_1\eta = -n_1\eta\cos(\eta\tau) + (-n_2\eta^2 + n_0)\sin(\eta\tau). \tag{18}$$

Eliminating τ by squaring and adding Eqs. (17) and (18), we get the equation determining for η as,

$$\eta^6 + d_2\eta^4 + d_1\eta^2 + d_0 = 0, \tag{19}$$

where

$$d_2 = \left(m_2^2 - 2m_1 - n_2^2\right), \quad d_1 = \left(m_1^2 - 2m_0m_2 + 2n_0n_2 - n_1^2\right), \quad d_0 = m_0^2 - n_0^2.$$

Substituting $\eta^2 = z$ in Eq. (19), we define a polynomial

$$P(z) = z^3 + d_2z^2 + d_1z + d_0 = 0. \tag{20}$$

In terms of the coefficients in $P(z)$, define Δ by $\Delta = d_2^2 - 3d_1$. It is easy to know from the character of cubic algebraic equation that $P(z)$ is strictly monotonically increasing function if $\Delta \leq 0$. If $\Delta > 0$ and $z^* = \frac{-d_2+\sqrt{\Delta}}{3} < 0$ or $\Delta > 0$ and $z^* = \frac{-d_2+\sqrt{\Delta}}{3} > 0$ but $P(z^*) > 0$, then $P(z)$ has always no positive root. Therefore, under these conditions, Eq. (14) has no purely imaginary roots for any $\tau > 0$ and this also implies that the positive equilibrium $E^*(N^*, I^*, A^*)$ of system Eqs. (4) $\sim$ (6) is absolutely stable. Thus, we can obtain easily the following result on the stability of positive equilibrium $E^*(N^*, I^*, A^*)$ of system Eqs. (4) $\sim$ (6).

Theorem 3. *Assume that Eq. (13) holds if $\Delta \leq 0$ or $\Delta > 0$ and $z^* = \frac{-d_2+\sqrt{\Delta}}{3} < 0$ or $\Delta > 0$ and $z^* = \frac{-d_2+\sqrt{\Delta}}{3} > 0$ and $P(z^*) > 0$. Then the positive equilibrium $E^*\,(N^*,\ I^*,\ A^*)$ of system Eqs. (4) $\sim$ (6) is absolutely stable, namely, $E^*\,(N^*,\ I^*,\ A^*)$ is asymptotically stable for any delay $\tau \geq 0$.*

4.4 Global stability of the endemic equilibrium

The objective of this section is to establish sufficient conditions under which the endemic equilibrium is globally stable. We linearize system Eqs. (4) $\sim$ (6) about the endemic equilibrium point $E^*\,(N^*, I^*, A^*)$. Let us define new variables as

$$u_1 = N - N^*, \quad u_2 = I - I^* \quad u_3 = A - A^*.$$

After substituting these variables, system Eqs. (4) $\sim$ (6) can be written in the following form:

$$\frac{\mathrm{d}u_1}{\mathrm{d}t} = \gamma u_2(t-\tau)e^{-d\tau} - du_1 - \alpha u_3, \tag{21}$$

$$\frac{\mathrm{d}u_2}{\mathrm{d}t} = \gamma\varepsilon u_2(t-\tau)e^{-d\tau} + \frac{\beta\,(N-I-A)}{N}u_2 - \frac{\beta I^*}{N}(u_2+u_3) + \frac{\beta\,(I^*+A^*)}{N^*N}u_1 - (d+\delta)\,u_2, \tag{22}$$

$$\frac{\mathrm{d}u_3}{\mathrm{d}t} = \delta u_2 + \gamma(1+\varepsilon)u_2(t-\tau)e^{-d\tau} - (d+\alpha)u_3. \tag{23}$$

Now, let us introduce the following functional:

$$V(u) = \frac{1}{2}u_1^2 + \frac{w}{2}u_2^2 + \frac{1}{2}u_3^2, \tag{24}$$

where $w > 0$ is an arbitrary real constant. The derivative of V is

$$\begin{aligned}
\dot{V}(u) =& u_1\dot{u}_1 + wu_2\dot{u}_2 + u_3\dot{u}_3,\\
\dot{V}(u) =& \gamma u_1u_2(t-\tau)e^{-d\tau} - du_1^2 - \alpha u_1u_3 + w\gamma\varepsilon u_2(t-\tau)e^{-d\tau}u_2 + \frac{w\beta\,(N-I-A)}{N}u_2^2\\
&- w(d+\delta)u_2^2 + \frac{w\beta I^*\,(I^*+A^*)}{N^*N}u_1u_2 - \frac{w\beta I^*}{N}\left(u_2^2+u_2u_3\right) + \delta u_2u_3\\
&+ \gamma\,(1-\varepsilon)\,u_2u_3(t-\tau)e^{-d\tau} - (d+\alpha)u_3^2,
\end{aligned} \tag{25}$$

or, equivalently,

$$\begin{aligned}
\dot{V}(u) \le& \gamma u_1u_2(t-\tau)e^{-d\tau} - du_1^2 - \alpha u_1u_3 + w\gamma\varepsilon u_2(t-\tau)e^{-d\tau}u_2 + w\beta u_2^2\\
&- w(d+\delta)u_2^2 - \frac{w\beta I^*}{\bar{N}}u_2^2 + \frac{w\beta I^*\,(I^*+A^*)\,(\alpha+d)}{N^*Q_0}u_1u_2 + \left[\delta - \frac{w\beta I^*}{\bar{N}}\right]u_2u_3\\
&+ \gamma(1-\varepsilon)u_2u_3(t-\tau)e^{-d\tau} - (\alpha+d)u_3^2,
\end{aligned} \tag{26}$$

choosing w as follows,

$$\delta = \frac{w\beta I^*}{\bar{N}}, \quad w = \frac{\delta\bar{N}}{\beta' I^*},$$

and applying Cauchy-Schwartz inequality to all u_iu_j-type terms, we obtain the following expression,

$$\begin{aligned}
\dot{V} \le& \frac{\gamma}{2}e^{-d\tau}u_1^2 + \frac{\gamma}{2}u_2^2(t-\tau)e^{-d\tau} - du_1^2 + \frac{\alpha}{2}u_1^2 + \frac{\alpha}{2}u_3^2 + \frac{w\gamma\varepsilon}{2}e^{-d\tau}u_2^2 + \frac{w\gamma\varepsilon}{2}e^{-d\tau}u_2^2(t-\tau)\\
&+ w\beta u_2^2 \ + \frac{w\beta I^*\,(I^*+A^*)\,(\alpha+d)}{2N^*Q_0}u_1^2 + \frac{w\beta I^*\,(I^*+A^*)\,(\alpha+d)}{2N^*Q_0}u_2^2 - w(d+\delta)u_2^2\\
&- \frac{w\beta I^*}{\bar{N}}u_2^2 + \frac{\gamma(1-\varepsilon)}{2}u_3^2e^{-d\tau} + \frac{\gamma(1-\varepsilon)}{2}u_2^2(t-\tau)e^{-d\tau} - (\alpha+d)u_3^2.
\end{aligned} \tag{27}$$

Arranging similar terms in the last inequality gives

$$\begin{aligned}
\dot{V} \le& -\left[d + \frac{\alpha}{2} - \frac{\gamma}{2}e^{-d\tau} - \frac{w\beta I^*\,(I^*+A^*)\,(\alpha+d)}{2N^*Q_0}\right]u_1^2 - \left[w\,(d+\delta) + \frac{w\beta I^*}{\bar{N}} - \frac{w\gamma\varepsilon}{2}e^{-d\tau} - w\beta\right.\\
&\left.-\frac{w\beta I^*\,(I^*+A^*)\,(\alpha+d)}{N^*Q_0}\right]u_2^2 - w\left[(\alpha+d) + \frac{\alpha}{2} - \frac{\gamma(1-\varepsilon)}{2}e^{-d\tau}\right]u_3^2\\
&+ \left[\frac{\gamma(1-\varepsilon)}{2} + \frac{\gamma}{2} + \frac{w\gamma\varepsilon}{2}\right]e^{-d\tau}u_2^2(1-\varepsilon).
\end{aligned} \tag{28}$$

We choose Lyapunov functional to be of the form,

$$U(u_t) = V(u) + \left[\frac{\gamma(1-\varepsilon)}{2} + \frac{\gamma}{2} + \frac{w\gamma\varepsilon}{2}\right] e^{-d\tau} \int_{t-\tau}^{t} u_2^2(\theta)d\theta, \tag{29}$$

and hence

$$\dot{U}(u_t) = \dot{V}(u) + \left[\frac{\gamma(1-\varepsilon)}{2} + \frac{\gamma}{2} + \frac{w\gamma\varepsilon}{2}\right] e^{-d\tau} \left[u_2^2(t) - u_2^2(1-\varepsilon)\right].$$

Therefore,

$$\dot{U}(u_t) \leq -\left[d + \frac{\alpha}{2} - \frac{\gamma}{2}e^{-d\tau} - \frac{w\beta I^*(I^*+A^*)(\alpha+d)}{2N^*Q_0}\right] u_1^2 - \left[w(d+\delta) + \frac{w\beta I^*}{\bar{N}} - w\gamma\varepsilon e^{-d\tau} - w\beta - \frac{\gamma(2-\varepsilon)}{2}e^{-d\tau} - \frac{w\beta I^*(I^*+A^*)(\alpha+d)}{N^*Q_0}\right] u_2^2 - w\left[(\alpha+d) + \frac{\alpha}{2} - \frac{\gamma(1-\varepsilon)}{2}e^{-d\tau}\right] u_3^2. \tag{30}$$

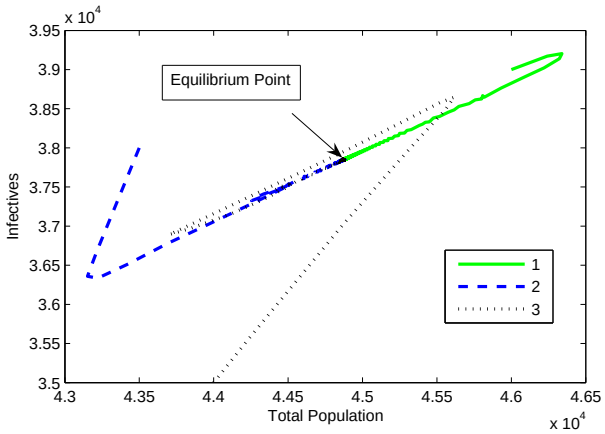

Fig. 1. Variation of total population against infective population

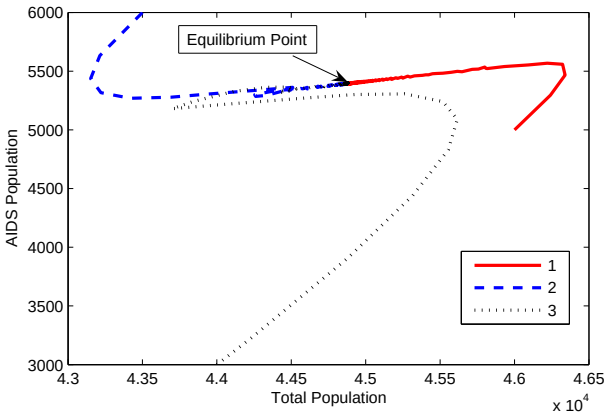

Fig. 2. Variation of total population against AIDS population

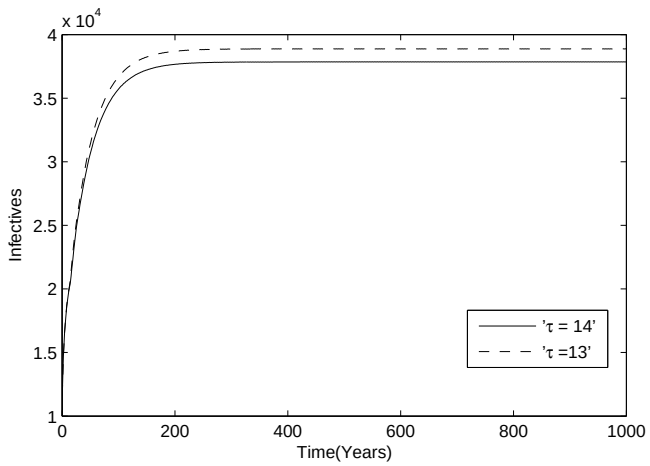

Fig. 3. Variation of infective population with time for different values of τ

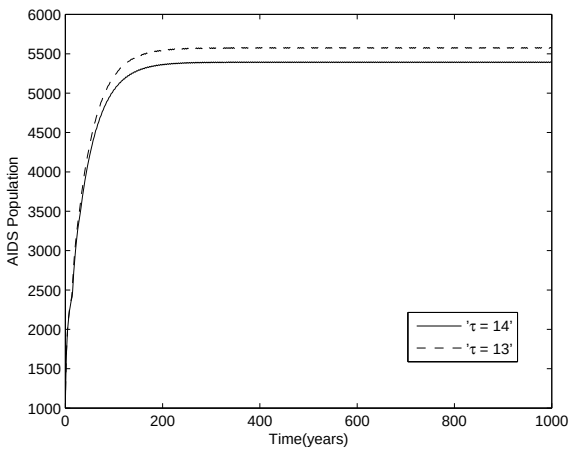

Fig. 4. Variation of AIDS population with time for different values of τ

The right hand expression of the above inequality is always negative provided,

$$\tau > \max\left\{\frac{1}{d}\ln\left[\frac{\gamma\left(\varepsilon+(2-\varepsilon)/2w\right)}{(d+\delta)N^*Q_0 + \left(Q_0\beta I^*N^*/\bar{N}\right) - \beta N^*Q_0 - \beta I^*(I^*+A^*)(\alpha+d)}\right],\right.$$
$$\left.\frac{1}{d}\ln\left[\frac{\gamma(1-\varepsilon)}{(3\alpha+2d)}\right], \quad \frac{1}{d}\ln\left[\frac{\gamma N^*Q_0}{(\alpha+2d)N^*Q_0 - w\beta I^*(I^*+A^*)(\alpha+d)}\right]\right\}. \tag{31}$$

Theorem 4. *Let the initial condition for system Eqs. (4) ∼ (6) be* $N(0) = N_0 > 0$, $I(s) = I_0(s) \geq 0$, *with* $I_0(0) > 0$ *and* $A(0) = A_0 > 0$. *Assume that the parameters of system Eqs. (4) ∼ (6) satisfy*

$$(d+\delta)N^*Q_0 + \frac{Q_0\beta I^*N^*}{\bar{N}} > \beta N^*Q_0 + \beta I^*(I^*+A^*)(\alpha+d), \tag{32}$$

$$(\alpha+2d)N^*Q_0 > \delta\bar{N}(I^*+A^*)(\alpha+d), \tag{33}$$

$$\alpha\delta > \gamma(d+\varepsilon)e^{-d\tau}, \tag{34}$$

$$\gamma\varepsilon e^{-d\tau} + \beta > \delta + d. \tag{35}$$

Then, for any maturity period τ *satisfying*

$$\tau > \max\left\{\frac{1}{d}\ln\left[\frac{\gamma(\varepsilon+(2-\varepsilon)/2w)}{(d+\delta)N^*Q_0 + (Q_0\beta I^*N^*/\bar{N}) - \beta N^*Q_0 - \beta I^*(I^*+A^*)(\alpha+d)}\right], \right.$$
$$\left.\frac{1}{d}\ln\left[\frac{\gamma(1-\varepsilon)}{3\alpha+2d}\right], \quad \frac{1}{d}\ln\left[\frac{\gamma N^*Q_0}{(\alpha+2d)N^*Q_0 - w\beta I^*(I^*+A^*)(\alpha+d)}\right]\right\}, \tag{36}$$

the endemic equilibrium E^* *is globally asymptotically stable.*

5 Numerical simulations

Since it is important to visualize the dynamical behavior of the model, the system Eqs. (4) ∼ (6) is integrated numerically with the help of MATLAB 7.1 using the following set of parameter values[12]: $Q_0 = 2000, d = 0.02, \beta = 1.43, \delta = 0.1, \alpha = 1, \tau = 14, \gamma = .15, \varepsilon = .6$ with initial values $N(0) = 15000, I(0) = 10000$, and $A(0) = 1000$. The equilibrium values are given as,

$$N^* = 44873, I^* = 37849, A^* = 5393$$

The computer simulation are performed for different initial starts in the following cases and displayed graphically in Figs. 1 and 2.
(1) $N(0) = 46000, I(0) = 39000$ and $A(0) = 5000$,
(2) $N(0) = 43500, I(0) = 38000$ and $A(0) = 6000$,
(3) $N(0) = 44000, I(0) = 35000$ and $A(0) = 3000$.

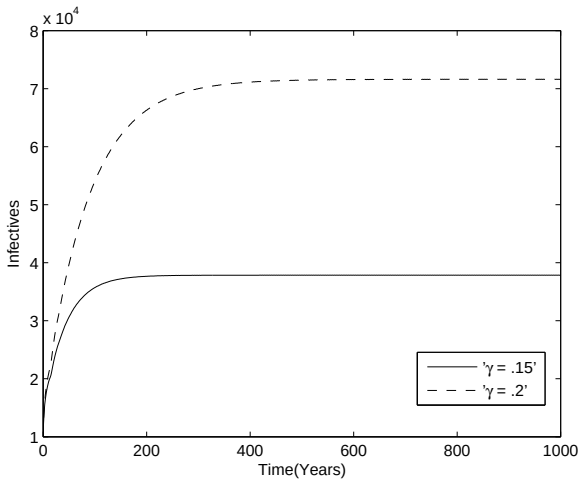

Fig. 5. Variation of infective population with time for different values of γ

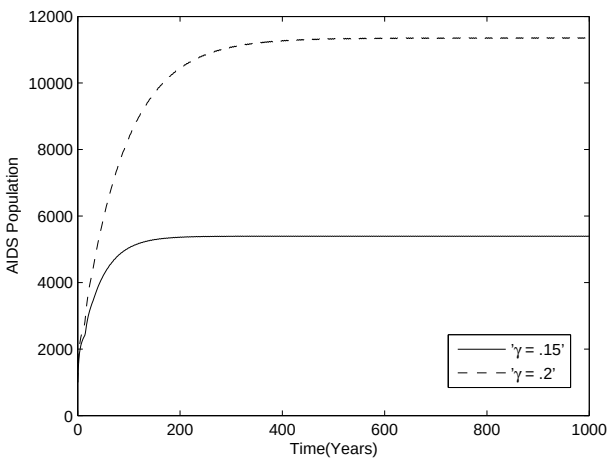

Fig. 6. Variation of AIDS population with time for different values of γ

In these figures, the infectives and the AIDS population are plotted against the total population. We see from these figures that for any initial start, the solution curves tend to endemic equilibrium E^*. Hence, we

infer that the system Eqs. (4) $\sim$ (6) may be globally stable about endemic equilibrium E^* for the above set of parameter values. The results of numerical simulation are displayed graphically in Figs. 3 $\sim$ 6. Figs. 3 $\sim$ 4 depict the variation of infective population and AIDS population respectively with time t for different values of time delay τ. It is seen that as time delay decreases, the number of infected individuals increases which in turn increases the AIDS population. It may be speculated that with decrease in sexual maturity period as time delay, more and more infected newborns attain sexual maturity quite early and become infectious. This decrease in sexual maturity period leads to increase the infectious population which ultimately increases the AIDS population. In Figs. 5 and 6, we have shown the effect of introduction of infected newborns via vertical transmission on the variation of infective population and AIDS population with time. We see from these figures that with increase in the rate of introduction of infected newborns, the infective population increases rapidly. This rapid increase in the infective population can be attributed to the cumulative effect of increased birth of infected newborns and decreased sexual maturity period τ. This increase in infective population consequently increases the AIDS population (see Fig. 6). Thus, if the spread of infection through vertical transmission is curbed either by promoting safety measures or by other effective treatments, the overall infective population can be controlled. Moreover, the delayed sexual maturity period will further restrict the infective population leading to reduced AIDS population. It is also observed that due to constant immigration into the population, the susceptible population increases continuously therefore infection becomes more endemic and always persists in the population.

6 Conclusion

In this paper, a nonlinear HIV/AIDS model with vertical transmission and the period of sexual maturity of infected newborns, incorporated as a time delay, is proposed in a population of varying size with constant immigration. The model is analyzed using stability theory of differential equations and numerical simulation. It is found that when reproduction number with delay $R_0 < 1$, the infection-free equilibrium $E_0$is locally asymptotically stable, and no other equilibrium exits. When $R_0 > 1$, the infection-free equilibrium E_0 loses its stability and becomes unstable and a non-trivial endemic equilibrium E^* appears. Using Lyapunov functional technique, we show that under certain restrictions on the parameter values and time delay, this equilibrium is globally asymptotically stable.

To support the analytical results, we have performed numerical simulations for a set of parameter values and it is shown that for $R_0 > 1$ and a reasonable time delay τ, the system is found to be stable. It is found that as the period of sexual maturity of infected newborns decreases, the number of infected individuals increases which in turn increases the AIDS population. It is speculated that with decrease in sexual maturity period, modeled as time delay, more and more infected newborns attain sexual maturity quite early and become infectious. This decrease ultimately increases the AIDS population. The infective population and hence the AIDS population also increases with increase in the rate of introduction of infected newborns via vertical transmission. This increase in infective population seems to be due to cumulative effect of increased birth of infected newborns and decreased sexual maturity period. Thus, curbing the spread of infection through vertical transmission and delayed sexual maturity period may restrict the infective population as well as AIDS population.

References

[1] F. Zhang, Z. Li, F. Zhang. Global stability of an SIR epidemic model with constant infectios period. *Applied Mathematics and Computation*, 2008, **199**: 285–291.

[2] H. Hethcote, P. van den Driessche. Two SIS epidemiological models with delays. *Journal of Mathematical Biology*, 2000, **40**: 3–26.

[3] J. Tchuenche, A. Nwagwo, R. Levins. Global behaviour of an SIR epidemic model with time delay. *Mathematic Method in the Applied Science*, 2007, **30**: 733–749.

[4] J. Zhang, W. Li, X. Yan. Hopf bifurcation and stability of periodic solutions in a delayed eco-epidemiological system. *Applied Mathematics and Computation*, 2008, **198(2)**: 865–876.

[5] K. Cooke, P. van den Driessche. Analysis of an SEIR epidemic model with two delays. *Journal of Mathematical Biology*, 1996, **35**: 240–260.
[6] S. Kovacs. Dynamics of an HIV/AIDS model: The effect of time delay. *Applied Mathematics and Computation*, 2007, **188(2)**: 1597–1609.
[7] Y. Kuang. *Delay differential equations with applications in population dynamics*. Academic Press, Boston, 1993.
[8] N. Yoshida, T. Hara. Global stability of a delayed SIR epidemic model with density dependent birth and death rates. *Journal of Computational and Applied Mathematics*, 2007, **201**: 339–347.
[9] Q. Khan, E. Krishnan. An epidemic model with a time delay in transmission. *Applications of Mathematics*, 2003, **3**: 193–203.
[10] R. Naresh, A. Tripathi, S. Omar. Modelling the spread of AIDS epidemic with vertical transmission. *Applied Mathematics and Computation*, 2006, **178**: 262–272.
[11] R. Nisbet, W. Gurney. *Modelling Fluctuatiing Populations*. Blackburn Press, USA, 1982.
[12] S. Ma, Q. Lu, M. Song. Dynamics of a logistic population model with maturation delay and nonlinear birth rate. *Discrete and Continuous Dynamical Systems*, 2005, **5(3)**: 735–752.
[13] W. Wang, Z. Ma. Global dynamics of an epidemic model with time delay. *Nonlinear Analysis: Real World Applications*, 2002, **3**: 365–373.
[14] W. Ma, M. Song, Y. Takeuchi. Global stability of an SIR epidemic model with time delay. *Applied Mathematics Letters*, 2004, **17**: 1141–1145.
[15] X. Yan. Stability and hopf bifurcation for a delayed prey- predator system with diffusion effects. *Applied Mathematics and Computation*, 2007, **192**: 552–566.
[16] Y. Kyrychko, K. Blyuss. Global properties of a delayed sir model with temporary immunity and nonlinear incidence rate. *Nonlinear Analysis: Real World Applications*, 2005, **6**: 495–507.
[17] Y. Song, S. Yan Bifurcation analysis in a predator- prey system with time delay. *Nonlinear Analysis: Real World Applications*, 2006, **7(2)**: 265–284.
[18] Z. Mukandavire, P. Das, et al. Global analysis of an HIV/AIDS epidemic model. *World Journal of Modelling and Simulation*, 2010, **6(3)**: 231–240.
[19] Z. Ma, H. Huo, C. Liu. Stability and hopf bifurcation analysis on a prey- predator model with discrete and distributed delays. *Nonlinear Analysis: Real World Applications*, 2009, **10(2)**: 1160–1172.
[20] Z. Mukandavire, A. Gumel. Mathematical analysis of a model for HIV-malaria co-infection. *Mathematical Biosciences and Engineering*, 2009, **6(2)**: 333–363.
[21] Z. Mukandavire, W. Garira et al. Asymptotic properties of an HIV/AIDS model with a time delay. *Journal of Mathematical Analysis and Applications*, 2007, **330**: 916–933.