Modeling the Spread of HIV/AIDS with Infective Immigrants and Time Delay

Agraj Tripathi1 *, Ram Naresh2, Jean M.Tchuenche3, Dileep Sharma2
1 Department of Mathematics, Bhabha Institute of Technology, Kanpur-209204, India
2 Department of Mathematics, H.B. Technological Institute, Kanpur-208002, India
3 Department of Mathematics, University of Dar es Salaam, P.O. Box. 35062, Dar es Salaam, Tanzania

(Received 16 September 2012, accepted 13 September 2013)

Abstract: In this paper, we propose and analyze a nonlinear mathematical model to study the spread of HIV/AIDS in a variable population incorporating the roles of immigration, treatment and the effect of time delay. The model exhibits a unique endemic steady state which is globally asymptotically stable if a certain threshold quantity, the treatment-induced basic reproduction number $\mathcal{R}(\lambda)$, which depends solely on the parameters of the model, is greater than unity. Under certain conditions, it has a locally asymptotically stable disease-free equilibrium. The constant immigration of infective as well as susceptible individuals, however, makes the disease more endemic. The public health implication of this is that the disease-free equilibrium is not feasible when recruitment of new asymptomatic infectives into the population is allowed and consequently, making control of the epidemic more difficult.

Keywords: HIV/AIDS; time delay; global stability; Lyapunov’s function

1 Introduction

The impact of HIV/AIDS on humans is so devastating, since its inception in 1981, that HIV epidemic is widely acknowledged to be the most severe health crisis of the modern times. HIV continues to spread at alarming rates through many parts of the world, and there have been few victories in the efforts to contain it. This is true despite remarkable advances in our understanding of the epidemiology of the disease, the molecular biology of the virus and its effects on the body-advances that have led to major therapeutic discoveries in the second decade of the epidemic. For those who are able to obtain treatment with antiretroviral drugs, HIV infection has been transformed from a fatal illness into a chronic condition [17]. This has led to dramatic reductions in mortality and morbidity from the illness (which at present will eventually kill). However, despite these advances on the biomedical front, the epidemic continues to spread and treatment remains unavailable to the overwhelming majority of those who require it. The Human Immuno-deficiency Virus (HIV) infection which causes the Acquired Immuno-deficiency Syndrome popularly known as AIDS, has shown a very high degree of prevalence in both the developed and developing world.

Mathematical models (deterministic, stochastic, delay) play an important role to study the transmission dynamics of infectious diseases, and in some sense, delay models give better compatibility with reality, as they capture the dynamics for the time of infection to the infectiousness [13, 29]. 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. It is recognized that time delays are natural components of the dynamic process of biology, ecology, physiology, economics, epidemiology and mechanics [10]. A brief comment on recent works with delay provides the context of this paper.

In recent years, many studies have been made to model the transmission of infectious diseases and different issues have been addressed which affect the spread of the disease [2-3, 6-8, 10, 14, 17-18, 28, 30-31]. In particular, Hethcote and van den Driessche [8] developed an SIS epidemic model with delay corresponding to the infectious period and introduced the disease related death. Khan and Krishnan [10] examined a SIR model by introducing time delay in the recruitment of infected persons, and show that the introduction of a time delay into the transmission term can destabilize the system and periodic solutions can arise by Hopf bifurcation. Greenhalgh et al. [6] presented an SIRS epidemic model with
vaccination, while Kyrychko and Blyuss [14] studied a time delayed SIR model with a general incidence term, the time delay representing the temporal immunity period. Rao and Rao [7] developed two models of ratio-dependent prey-predator dynamical systems and analyzed their stability. Tchuenche et al. [25] presented a delayed SIR epidemic model, with the short delay time introduced in the form of survival during that time lag, and study the stability behavior of the model. Naresh et al. [18] studied the stability of a SIR model with time delay using nonlinear incidence.

A very little attention has been paid to study the role of time delay in HIV/AIDS models. Kovacs [11] considered an HIV/AIDS model with delay and used the delay as a bifurcation parameter to study the possibility of periodic solution. Mukandavire et al. [15] 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. Naresh and Sharma [20] proposed an HIV/AIDS model with vertical transmission and the period of sexual maturity of infected newborns incorporated as time delay. Naresh et al. [21] studied the spread of HIV/AIDS with screening of unaware of infectives and introduced time delay in the recruitment of infected persons in an adult population of variable size structure. Our model differs from previous ones in many aspects: We assume a constant birth rate, a constant flow of infective immigrants. Some infectious individuals move directly to the AIDS group after some time , with the assumption that their time of infection is not known as they arrive into the latter class. Therefore, there are two time delays in the system. This works greatly extends the one proposed in [15].

It is well known that immigrants play a critical role in disease dynamics [19, 22-23]. Epidemics ignited or enhanced by immigration of infectious cases include HIV, SARS, avian influenza and measles [12]. Sexually transmitted diseases, such as gonorrhea do not confer immunity, while HIV/AIDS is not yet fully curable. In the proposed model, protective/control measures such as use of condoms, education, complacency, gradual behavioral change are not accounted for. The reason is basically that in rural communities (in the developing world such as Sub-Saharan Africa), individuals are either too poor to afford condoms (partially effective vaccine), and sex education is still a taboo [15]. The health and socioeconomic risks posed by disease such as HIV/AIDS are compelling scientists to design and implement more effective control and preparedness for emerging and re-emerging infectious diseases. Studies of epidemics that incorporate exponential natural death, disease-related death as well as constant recruitment of the population by migration and birth have become one important area in mathematical theory of epidemiology [5].

In view of the above, we propose a nonlinear mathematical model to study the spread of HIV/AIDS with immigration and treatment of infectives incorporating time delay within a population of varying size including demographic and epidemiological features. We have also considered that since there is no known cure of HIV infection, therefore, treated infectives are also bound to develop AIDS. In order to investigate the influence of some key parameters on the spread of the disease, graphical representations of the proposed model are illustrated.

## 2 Mathematical model

We consider a population \(N(t)\) at time \(t\), consisting of five compartments of susceptibles \(S(t)\), exposed individuals \(E(t)\), HIV infectives \(I(t)\), treated infectives \(T(t)\) and that of AIDS patients \(A(t)\). In the model, we assume that susceptibles become HIV infected via sexual contacts with HIV infectives. We consider that the susceptible population is generated from two sources i.e. by birth at a rate \(\Lambda\) and by immigration. There is a natural constant death rate \(\mu\), and excess constant death rate for infectives as well as AIDS individuals \(d\) and \(\alpha\), respectively. Individuals in the infective as well as AIDS groups have a greater risk of dying due to their illness, become sexually inactive if they are too ill which leads them out of the system [11]. A constant flow of \(Q_0\) new members arrives into the population in any given time unit, with the fraction \(\varepsilon\) arriving infected \((0 \leq \varepsilon \leq 1)\). The force of infection is assumed to be of frequency-dependent incidence or standard form, namely proportional to \(\frac{I(t)}{N(t)}\), where \(N(t)\) is the total (variable) population size and \(I(t)\) is the size of the infective population. We note that to be biologically relevant, \(I(t)\) and \(E(t)\) satisfy a certain integral equation [2, 8], therefore, they can not be negative. The latent periods are \(\tau\) and \(\omega\), with \(\tau = \max(\omega, \tau) = \omega\), since for HIV \(\omega > \tau\). The probability that an individual viral load remains very low \(t\) units of time after becoming exposed is given by the step function with value 1 for \(t \leq \tau\), and 0 for \(t > \tau\). Here it is remarked that the individuals in the exposed class are well aware of their infection and those in AIDS class are too ill and isolated that they may be not take part in sexual interaction. The treated infectives are also assumed not to take part in unsafe sexual interactions. However, in some recent studies these considerations have been taken into account [16]. The model in the present study can be generalized further by taking into account these aspects.

With the above assumptions and considerations, the spread of the disease is assumed to be governed by the following
system of nonlinear ordinary differential equations with two discrete delays in a variable population,

\[
\frac{dS(t)}{dt} = \Lambda + (1 - \varepsilon)Q_0 - \beta \frac{S(t)I(t)}{N(t)} - \mu S(t) \tag{1}
\]

\[
\frac{dE(t)}{dt} = \beta \frac{S(t)I(t)}{N(t)} - \beta e^{-\mu_1 \tau} \frac{S(t-\tau)I(t-\tau)}{N(t-\tau)} - \mu E(t) \tag{2}
\]

\[
\frac{dI(t)}{dt} = \varepsilon Q_0 + \beta e^{-\mu_1 \tau} \frac{S(t-\tau)I(t-\tau)}{N(t-\tau)} - (\lambda + \delta)I(t - \omega) - (\mu + d)I(t) \tag{3}
\]

\[
\frac{dT(t)}{dt} = \lambda I(t - \omega) - (\theta + \mu)T(t) \tag{4}
\]

\[
\frac{dA(t)}{dt} = \delta I(t - \omega) + \theta T(t) - (\alpha + \mu)A(t) \tag{5}
\]

\( S(t) \geq 0 \text{ on } [-\tau, 0] \rightarrow R_+, E(t) \geq 0 \text{ on } [-\tau, 0], T(t) \geq 0, \ A(t) \geq 0, N(t) \geq 0 \text{ on } [-\tau, 0] \rightarrow R_+ \)

\( N(0) = S_0 > 0, I(0) = I_0(\omega) \geq 0 \text{ for all } u \in [-\tau, 0], T(0) = T_0 \geq 0, A(0) = A_0 \geq 0 \)

where \( e^{-\mu_1 \tau} \) is the survival probability in the short time interval \([-\tau, 0], \) \( \lambda \) is the rate at which infectives seek treatment, \( \beta \) is the effective contact rate that leads to infection, \( \theta \) is the rate at which treated infectives move to the AIDS class (most likely after a long prophylactic treatment) and \( \delta \) is the rate at which some infectives move to the AIDS class (since some infective immigrants might have already spent some time in the I-class elsewhere) or those who know their status and decide to go for treatment), we assume a small proportion will spend a short time \( \omega \) before moving to the fatal class). Presumably \( \mu_1 \) would be at least as large as \( \mu \) \( (\mu_1 \leq \mu) \). Also treatment reduces the progression to AIDS at a rate \( \theta(0 \leq \theta < \delta) \).

The model system (1)-(5) can be rewritten as, using \( N(t) = S(t) + E(t) + I(t) + T(t) + A(t) \),

\[
\frac{dN(t)}{dt} = \Lambda + Q_0 - \mu N(t) - dI(t) - \alpha A(t) \tag{6}
\]

\[
\frac{dE(t)}{dt} = \beta \frac{(N(t) - E(t) - I(t) - T(t) - A(t))I(t)}{N(t)} - \mu E(t) - \beta e^{-\mu_1 \tau} \frac{(N(t-\tau) - E(t-\tau) - I(t-\tau) - T(t-\tau) - A(t-\tau))I(t-\tau)}{N(t-\tau)} \tag{7}
\]

\[
\frac{dI(t)}{dt} = \varepsilon Q_0 - (\lambda + \delta)I(t - \omega) - (\mu + d)I(t) + \beta e^{-\mu_1 \tau} \frac{(N(t-\tau) - E(t-\tau) - I(t-\tau) - T(t-\tau) - A(t-\tau))I(t-\tau)}{N(t-\tau)} \tag{8}
\]

\[
\frac{dT(t)}{dt} = \lambda I(t - \omega) - (\theta + \mu)T(t) \tag{9}
\]

\[
\frac{dA(t)}{dt} = \delta I(t - \omega) + \theta T(t) - (\alpha + \mu)A(t) \tag{10}
\]

\[2.1 \text{ Invariant region} \]

**Lemma 1** All solutions of the model system (6)-(10) starting in \( R_+^5 \) are bounded and eventually enter the attracting set

\[
\phi = [(S, E, I, T, A) \in R_+^5 : \frac{\Lambda + Q_0}{\mu + \alpha + d} + \zeta \leq N(t) \leq \frac{\Lambda + Q_0}{\mu} + \zeta] \tag{11}
\]

for any arbitrary small \( \zeta > 0 \).

\[2.2 \text{ Positivity of solutions} \]

**Lemma 2** Let the initial data be \( N(0) = S_0 > 0, I(0) = I_0(\omega) \) for all \( u \in [-\tau, 0] \rightarrow R_+, T(0) = T_0 \geq 0, A(0) = A_0 \geq 0 \).Then, the solution \((S(t), E(t), I(t), T(A), A(t))\) of the model remain positive for all time \( t > 0 \).
3 Existence of equilibria of the model

3.1 Existence of trivial and disease-free equilibrium

The important qualitative implication of having the recruitment terms \( \Lambda \) and \( Q_0 \) non-zero is that the population will not go extinct. Consequently, the trivial equilibrium is not feasible for this model (and it is possible that small changes in parameters can produce large changes in equilibrium behavior). The model system (6)-(10) has a disease-free equilibrium (DFE) if \( \varepsilon = 0 \) (i.e. all the immigrants are susceptible). The disease-free equilibrium is \( E_0 = (\frac{\Lambda + Q_0}{\mu}, 0, 0, 0, 0) \), where \( \frac{\Lambda + Q_0}{\mu} = K \), the asymptotic carrying capacity of the population.

3.2 Existence of the endemic equilibrium

The model (6)-(10) has a unique non-negative endemic equilibrium given by \( E_1 = (N^*, E^*, I^*, T^*, A^*) \), where \( N^*, E^*, I^*, T^* \) and \( A^* \) are positive solutions of the following system of algebraic equations,

\[
\begin{align*}
\Lambda + Q_0 - \mu N - d I - \alpha A &= 0 \tag{12} \\
\beta \frac{(N - E - I - T - A)I}{N} - \mu E - \beta e^{-\mu_T} \frac{(N - E - I - T - A)I}{N} &= 0 \tag{13} \\
\varepsilon Q_0 - (\lambda + \delta)I - (\mu + d)I + \beta e^{-\mu_T} \frac{(N - E - I - T - A)I}{N} &= 0 \tag{14} \\
\lambda I - (\theta + \mu)T &= 0 \tag{15} \\
\delta I + \theta T - (\alpha + \mu)A &= 0 \tag{16}
\end{align*}
\]

On solving simultaneously the algebraic system of equations (12)-(16), we obtain

\[
\begin{align*}
T &= pf(N) \tag{17} \\
A &= qf(N) \tag{18} \\
I &= \frac{[\Lambda + Q_0 - \mu N]}{(\alpha q + d)} = f(N), \tag{19}
\end{align*}
\]

where \( p = \frac{\lambda}{\theta + \mu} \) and \( q = \frac{\delta (\theta + \mu) + \lambda \theta}{(\theta + \mu) (\alpha + \mu)} \). From equation (19), \( f(N) < 0 \) if \( \Lambda + Q_0 > \mu N \) otherwise \( I \to 0 \), that is the endemic equilibrium does not exist.

\[
E = \frac{\beta (1 - e^{-\mu_T}) [N - f(N)(1 + p + q)] f(N)}{\beta (1 - e^{-\mu_T}) f(N) + \mu N} = g(N) \tag{20}
\]

To show the existence of \( E_1 \) we write,

\[
F(N) = \varepsilon Q_0 - (\lambda + \delta + \mu + d) f(N) + \frac{\beta e^{-\mu_T} [N - g(N)(1 + p + q)] f(N)}{N}
\]

which can be further simplified to,

\[
F(N) = \left[ \varepsilon Q_0 - (\lambda + \delta + \mu + d) f(N) \right] [\beta (1 - e^{-\mu_T}) f(N) + \mu N] + \beta e^{-\mu_T} \mu f(N)^2 [N - f(N)(1 + p + q)] \tag{21}
\]

It would be sufficient if we show that \( F(N) = 0 \) has one and only one root.

To prove this, we have,

\[
F(\frac{\Lambda + Q_0}{\mu + \alpha + d}) = \left[ \varepsilon Q_0 - (\lambda + \delta + \mu + d) \right] \left[ \frac{\alpha \mu (\Lambda + Q_0)}{(\alpha q + d)(\mu + \alpha + d)} \right]^2 \\
\left[ \beta (1 - e^{-\mu_T}) + \frac{\mu (\alpha q + \mu)}{(\alpha + \mu)} - \beta e^{-\mu_T} \frac{\mu (\alpha + \mu)}{(\alpha q + d)(\mu + \alpha + d)} \right]^2 \\
\left[ d (\Lambda + Q_0) \alpha (1 + p) + \mu (p + q) \right] \\
\left( \frac{(\alpha q + d)(\mu + \alpha + d)}{(\alpha q + d)(\mu + \alpha + d)} \right)
\]

Here

\[
F\left( \frac{\Lambda + Q_0}{\mu + \alpha + d} \right) < 0, \quad i f \varepsilon Q_0 < (\lambda + \delta + \mu + d) \tag{23}
\]

IUNS email for contribution: editor@nonlinearscience.org.uk
\[ F(\frac{\Lambda + Q_0}{\mu}) = \varepsilon Q_0 (\Lambda + Q_0) > 0 \] (24)

Also
\[ F'(N) = \mu [1 - \frac{\beta(1 - e^{-\mu_1^2})}{(aq + d)}] [\varepsilon Q_0 - (\lambda + \delta + \mu + d) f(N)] + \frac{\mu(\lambda + \delta + \mu + d)}{(aq + d)} [\beta(1 - e^{-\mu_1^2}) f(N) + \mu N] \]
\[ + \beta e^{-\mu_1^2} f(N)^2 \mu [1 + \frac{\mu(1 + p + q)}{(aq + d)}] + 2\beta e^{-\mu_1^2} f(N) f(N)(1 + p + q) - N] \frac{d^2}{(aq + d)} \] (25)

It is noted that if \( F'(N) > 0 \) then, \( F(N) \) = 0 has exactly one root (say \( N^* \)) between \( \frac{\Lambda + Q_0}{\mu + \alpha + \beta} \) and \( \frac{\Lambda + Q_0}{\mu} \). Using \( N^* \), the values of \( I^* \), \( E^* \), \( T^* \) and \( A^* \) can be found easily.

It is observed that from system (6)-(10), if immigration of infectives is negligible, i.e. \( \varepsilon = 0 \), then the endemicity of the infection is reduced. This can simply be shown by comparing \( S_0 \) and \( S^* \) using a crude assumption.

\((H_1)\) Let only \( I(t) \) be zero at equilibrium in eq.(1), then \( \frac{S(t)}{S_0} = 1 - \frac{Q_0}{N^*}Q_0 < 1 \), this implies that the introduction of infective immigrants reduces \( S_0 \) and increases \( I(t) \).

### 4 Permanence of the system

The behavior of the local dynamics near the disease-free steady state \( E_0 \) implies that system (6)-(10) is uniformly persistent in \( \phi \). That is, there exists some positive constant \( c > 0 \), independent of the initial data, such that \( c \leq \liminf N(t), E(t), I(t), T(t), A(t) \).

Since the disease-free steady-state is isolated (it lies on the boundary \( \partial \phi \)), the only largest invariant subset of the interior \( \phi_0 \) of \( \phi \) is the endemic equilibrium \( E_1 \). Consequently, if the basic reproduction number \( R(\lambda) \), say, defined below is such that \( R(\lambda) > 1 \), then, \( E_0 \) will be unstable, and by a result of Hof-bauer and So [9] system (6)-(10) is uniformly persistent. In this case, all solutions starting in \( \phi \) and sufficiently close to \( E_0 \) move away from \( E_0 \) towards \( E_1 \). For any solution \((S(t), E(t), I(t), T(t), A(t))\) of system (6)-(10), \( c_1 = \frac{\Lambda + (1 - \varepsilon) Q_0}{\mu} \leq \liminf N(t) \) as \( t \to \infty \), as shown above. Hence, we have the following result.

**Lemma 3** System (6)-(10) is uniformly persistent for any time delays \( \tau \) and \( \omega \).

### 4.1 Computation of threshold ratio

To compute the disease threshold parameter which is closely related to disease transmission, we use the same approach as in [3, 24].

If we write \( I(t - \omega) = e^{-\mu_1^2} I(t) \) and \( I(t - \tau) = e^{-\mu_1^2} I(t) \), then
\[ R(\lambda) = \frac{\beta e^{-\mu_1^2}}{(\lambda + \delta)e^{-\mu_1^2} + (\mu + \delta)} \frac{\lambda e^{-\mu_1^2}}{\mu_1^2} S_0 = R_0 \frac{\mu_1^2}{(\lambda + \delta)} \frac{\lambda e^{-\mu_1^2}}{\mu_1^2} \] (26)

is the treatment-induced reproduction number, and \( R_0 = \frac{\beta e^{-\mu_1^2}}{(\lambda + \delta)} S_0 \) is the basic reproduction number. We observe that the introduction of infective immigrants may increase \( R(\lambda) \), the average number of new HIV infections generated by a single HIV infected individual in a population where a certain fraction of infected individuals are treated. That is, \( R(\lambda) > R_0 \).

For \( \lambda \geq 0 \), if \( \frac{\beta e^{-\mu_1^2}}{\mu_1^2} \leq 1 \), in this case, no amount of treatment can bring \( R(\lambda) \) below 1. Setting \( R(\lambda) = 1 \) and solving for \( \theta \), we obtain the critical threshold treatment induction rate
\[ \theta_c = \left[ \frac{-\beta \lambda(\Lambda + Q_0)e^{-\mu_1^2}}{\mu(\lambda + \delta + d)e^{-\mu_1^2}} - \mu \right] \] (27)

Using hypothesis \((H_1)\) above, we conclude that the introduction of infective immigrants will increase the critical treatment rate. A little algebraic manipulation shows that by comparing \( \theta_c \) and \( \theta_{cc} = \frac{\beta \lambda(\Lambda + Q_0)e^{-\mu_1^2}}{\mu(\lambda + \delta + d)e^{-\mu_1^2}} - \mu \), we have \( \theta_c > \theta_{cc} \).

The more infectives, the more is the demand for treatment. But, if the treatment rate is very effective and available to many infected individuals so that this critical threshold is exceeded, that is \( \frac{\beta e^{-\mu_1^2}}{\mu_1^2} < 1 \), then \( R_0 \) can be reduced below the critical value 1, and only in this case the disease will not persist.

Below, we use some heuristic values to illustrate the effect of treatment on the HIV infective individuals. For some model parameters, the exposed class grows unbounded, this is taken care of below to avoid explosion.

From Theorem 2 of [27], the following results follows:
Theorem 4 When \( \epsilon = 0 \), the disease-free equilibrium of the model given by (6)-(10), is locally as well as globally asymptotically stable in \( \phi \) if \( R(\lambda) < 1 \) provided \( (R_0 < 1) \) and unstable if \( R(\lambda) > 1 \).

5 Global stability of the endemic equilibrium

Now to analyze the global stability near the endemic equilibrium point \( E_1 = (S^*, E^*, I^*, T^*, A^*) \), we consider the equations for normalized quantities, since it is easier to analyze our model in terms of proportions. We make the following transformations: \( e_1 = \frac{E}{N}, i_1 = \frac{I}{N}, t_1 = \frac{T}{N}, a_1 = \frac{A}{N} \). Therefore, \( e_1 + i_1 + t_1 + a_1 \leq 1 \). Without any ambiguity, we shall revert to the usual notation later. Assume that the rate of new infections is approximately constant, then

\[
\frac{dI(t)}{dt} = i_1(t) \frac{e^{-\mu t}}{N} \quad \text{and} \quad \frac{dE}{dt} = \frac{1}{N} \left( \frac{dE}{dt} - e_i \frac{dN}{dt} \right)
\]

Therefore,

\[
\frac{de_i}{dt} = \beta [1 - e_i(t) - i_1(t) - t_1(t) - a_1(t)] i_1(t) - \mu e_i(t) + d_i(t) e_i(t) + a_1(t) e_i(t)
\]

\[
\frac{di_1}{dt} = -\beta e^{-\mu_t} i_1(t) [1 - e_i(t) - i_1(t) - t_1(t) - a_1(t) - \lambda (t - \tau)]
\]

\[
\frac{dt_1}{dt} = \lambda e^{-\mu_t} i_1(t) - \theta t_1(t) + d_i(t) t_1(t) + \alpha t_1(t) a_1(t)
\]

\[
\frac{da_1}{dt} = \delta e^{-\mu_t} i_1(t) + \theta t_1(t) - \alpha a_1(t) + d_i(t) a_1(t) + a_1(t) a_1(t)
\]

Now, we linearize eqs. (28)-(31) about the endemic equilibrium point \( E_1 = (N^*, E^*, I^*, T^*, A^*) \). Let us define

\[
U_1(t) = N(t) - N^*, U_2(t) = E(t) - E^*, U_3(t) = I(t) - I^*, U_4(t) = T(t) - T^*, U_5(t) = A(t) - A^*.
\]

Using the perturbations about the endemic equilibrium point we obtain:

\[
\frac{dU_2}{dt} = -U_2(t) - U_3(t) - U_4(t) - U_5(t) + d(U_3(t) E^* + U_2(t) I^*) + \alpha (U_3(t) E^* + U_2(t) A^*)
\]

\[
\frac{dU_3}{dt} = -[\lambda + \delta] e^{-\mu t} + \beta e^{-\mu t} I^* U_2(t - \tau) - \beta e^{-\mu t} U_3(t - \tau) - U_3(t - \tau) - U_3(t - \tau) - U_3(t - \tau) - U_3(t - \tau)
\]

\[
-\beta e^{-\mu t} U_3(t)(1 - E^* + I^* - T^* - A^*)
\]

\[
\frac{dU_3}{dt} = -[\lambda + \delta] e^{-\mu t} + \beta e^{-\mu t} I^* U_2(t - \tau) - \beta e^{-\mu t} U_3(t - \tau) - U_3(t - \tau) - U_3(t - \tau) - U_3(t - \tau) - U_3(t - \tau)
\]

\[
-\beta e^{-\mu t} U_3(t)(1 - E^* + I^* - T^* - A^*)
\]
\[ \frac{dU}{dt} = \lambda e^{-\mu t} U_3(t) - \theta U_4(t) + d[U_3(t)T^*U_4(t)I^*] + \alpha[U_3(t)T^*U_4(t)A^*] \]  
(34)  
\[ \frac{dU}{dt} = \delta e^{-\mu t} U_3(t) + \theta U_4(t) - \alpha U_5(t) + d[U_3(t)A^*U_5(t)I^*] + 2\alpha U_5(t)A^* \]  
(35)
Without loss of reality, we write instead of and similarly for the other terms \((i_1', i_1', a_1')\). We shall employ Lyapunov functional technique. Let us first introduce the functional,  
\[ V(U) = \frac{1}{2}(U_2^2 + U_4^2 + U_5^2) + \frac{1}{2} \beta S^* e^{-\mu t}[U_3^2(t) - U_3^2(t - \tau)] + \frac{1}{2} [U_2^2(t) - U_2^2(t - \omega)] \]  
(36)
Differentiating \(V(U)\) with respect to \(t\), we get
\[ V(U) = U_2(t)U_2(t) + U_4(t)U_4(t) + U_5(t)U_5(t) + \frac{1}{2} \beta S^* e^{-\mu t}[U_3^2(t) - U_3^2(t - \tau)] + \frac{1}{2} [U_2^2(t) - U_2^2(t - \omega)] \]  
(37)
Using (32)-(35) and simplifying equation (37) we get
\[ V(U) = -(1 - dI^* - \alpha A^*)U_2^2(t) - (\theta - dI^* - \alpha A^*)U_2^2(t) - (\alpha - dI^* - 2\alpha A^*)U_2^2(t) \]  
\[ + (1 + \beta e^{-2\mu t}(1 - E^* - T^* - A^*) - dE^*)[U_2^2(t) - U_2^2(t - \tau)] \]  
\[ + \frac{1}{2} \beta S^* e^{-\mu t}[U_3^2(t) - U_3^2(t - \tau)] + \frac{1}{2} \beta S^* e^{-\mu t}[U_3^2(t) - U_3^2(t - \tau)] \]  
(38)
On applying the Cauchy-Schwarz-type terms, we arrive at the following equation:
\[ V(U) = -(1 - dI^* - \alpha A^*)U_2^2(t) - (\theta - dI^* - \alpha A^*)U_2^2(t) - (\alpha - dI^* - 2\alpha A^*)U_2^2(t) \]  
\[ + (1 + \beta e^{-2\mu t}(1 - E^* - T^* - A^*) - dE^*)[U_2^2(t) - U_2^2(t - \tau)] \]  
\[ + \frac{1}{2} \beta S^* e^{-\mu t}[U_3^2(t) - U_3^2(t - \tau)] \]  
(39)
Now, let us assume that
\[ (H_2) \frac{1}{2} + \frac{1}{2} \beta S^* e^{-\mu t} + \frac{1}{2} [1 + \beta e^{-2\mu t}(1 - N^*) - dE^*] + \frac{1}{2} e^{-\mu t}(\delta + \lambda) + \frac{1}{2} d(T^* + A^*) = 0 \]
In the simple case when \(\delta, \omega \to 0\), the last equation reads \(d(E^* - T^* - A^*) = 1\). This allows us to choose a Lyapunov function of the form
\[ W(U) = V(U) + \frac{1}{2} \beta e^{-2\mu t} I\int_{t-\tau}^{t} U_3^2(\phi) + U_2^2(\phi) + U_2^2(\phi) \]  
\[ \frac{dU}{dt} W(U) = V(U) + \frac{1}{2} \beta e^{-2\mu t} I\int_{t-\tau}^{t} U_3^2(\phi) + U_2^2(\phi) + U_2^2(\phi) \]  
Its derivative is given by
\[ \frac{dU}{dt} W(U) = \frac{dU}{dt} V(U) + \frac{1}{2} \beta e^{-2\mu t} I\int_{t-\tau}^{t} U_3^2(\phi) + U_2^2(\phi) + U_2^2(\phi) \]  
\[ \leq [-1(1 - dI^* - \alpha A^*) - \frac{1}{2} (1 - \alpha E^*) - 2\beta e^{-2\mu t} I - \frac{1}{2} (1 + \beta e^{-2\mu t} I(1 - N^*) - dE^*)][U_2^2(t)] \]  
(40)
\[ - (1 - dI^* - \alpha A^*) - \frac{1}{2} \beta S^* e^{-\mu t} + \frac{1}{2} \beta S^* e^{-\mu t} \int_{t-\tau}^{t} U_3^2(\phi) \]  
(41)
Thus, \(\frac{d}{dt} W(U)\) is negative semi-definite. Hence, we have proved the following result.

**Theorem 5** If the parameters of the system satisfy the cruise assumption \(H_2\) above, then the endemic steady state \(E_3\) is globally asymptotically stable in \(\phi_0\), the interior of \(\phi\), provided \(R(\lambda) > 1\).

The endemic steady state \(E_3\) is globally asymptotically stable (with the above cruise assumption, which may indirectly be interpreted as the absence of infective immigrants). If on the contrary the above assumption is not made, then the endemic equilibrium may be unstable and will then require a feedback control in order to stabilize it, an approach which we are not exploring in the present study.

Graphical representations are given below to see the effects of time delays on the dynamics of infectives, treated infectives and AIDS classes and their stability. For large time delays, oscillations occur at the early stages \([10]\), but settle down towards the equilibrium values as time becomes large.

### 6 Numerical simulation

To see the dynamical behavior of the model system, the system (6)-(10) is integrated numerically with the help of MATLAB-7.1 using the following parameter values with units in per year:

**LINS homepage:** [http://www.nonlinearscience.org.uk/](http://www.nonlinearscience.org.uk/)
$\Lambda = 500, Q_0 = 2000, d = 0.02, \mu = 0.08, \alpha = 1, \beta = 1.5, \mu_1 = 0.01, \epsilon = 0.3, \lambda = 0.2, \theta = 0.1, \delta = 0.3, \tau = 1, \omega = 0.5$ with initial values $N(0) = 1000, E(0) = 500, I(0) = 300, T(0) = 100, A(0) = 20.$

The equilibrium values are computed as, $N^* = 19206, E^* = 1083, I^* = 3947, T^* = 6577, A^* = 1806$

The computer simulations have been performed for different initial starts in the following four cases and displayed graphically in Figs.3-4.

- $N(0) = 1000, E(0) = 500, I(0) = 300, T(0) = 100$ and $A(0) = 20;
- N(0) = 20000, E(0) = 2000, I(0) = 1000, T(0) = 2500$ and $A(0) = 1200;
- N(0) = 15000, E(0) = 700, I(0) = 900, T(0) = 1500$ and $A(0) = 3000;
- N(0) = 30000, E(0) = 3000, I(0) = 1500, T(0) = 800$ and $A(0) = 400.$

Figure 3: Variation of susceptibles with infectives. Figure 4: Variation of susceptibles with AIDS patients.

In these figures, the infective population and AIDS population is plotted against the susceptible population. We see from these figures that for any initial start, the solution curves tend to the equilibrium $E_1.$ Hence, we infer that the system (6)-(10) is globally stable about this endemic equilibrium point $E_1$ for the above set of parameters.

The effect of time delays on the infective, treated infective and AIDS classes and their stability are shown in Figs. (5-10).

Figure 5: Effect of time delay $\tau$ taken alone on the dynamics of infective individuals.

Figure 6: Effect of time delay $\tau$ taken alone on the dynamics of AIDS individuals.

Figure 7: With large time delay $\omega,$ oscillations occur at the early stage of the infection.

From these figures, it is seen that large time delays, as expected, cause the model to have some complex behaviour (damped oscillations), especially at the beginning of the process. But, this phenomenon settles down towards the steady states at a later time. Introduction of time delay destabilizes the system (complex dynamics occur) and periodic solutions arise. It is also noted that the joint effects of both time delays $\tau$ and $\omega$ on the model show no difference in the shape of the graphs.

IJNS email for contribution: editor@nonlinearscience.org.uk
7 Conclusions

This paper is based on the analysis of a seemingly new model of HIV/AIDS transmission with infective immigrants with time delay and treatment. The model incorporates some essential parameters of the HIV/AIDS transmission and enables the assessment of the effect of recruitment of infected immigrants into the community. Indeed, the roles of immigration, treatment and the effect of time delay are studied. Conditions for the existence of the steady states are given and their stability is analyzed.

The model system has only a non-negative equilibrium due to the direct inflow of infectives, but in the absence of the latter, there is an addition to it, namely; the disease-free steady state. Some inferences have been drawn regarding the spread of the disease by way of establishing stability results. The model system is globally stable under a severe condition (see hypothesis $H_2$). It is found that if the direct inflow of the infectives is allowed in the community, the disease always persist in the population, as this continuously triggers the epidemic. This direct inflow of infectives makes the disease more difficult to control. Hence, the spread of infection can be slowed down if direct inflow of infectives is restricted into the population, and more infectives move into the treated class.

In the nutshell, we presented an HIV/AIDS model with some qualitative analysis, including positivity and boundedness of solutions, local and global stability of the equilibrium points as well as persistence. The proposed model can be extended in various ways, by including for instance a vaccinated class (pending the success of obtaining a perfect/imperfect vaccine). The model accounts for those who know about their infection and take preventive attitude or change their behavior and use some medication such as AZT, Zidovudine, etc. A possible refinement is to include compliance as some individuals may rather continue infecting others (negative behavioral change). It is also shown (Fig. 1) that early treatment is beneficial, but at later stage of the infection, there is only a very light difference in the incidence rate. Large time delays as expected, cause the model to exhibit complex behaviors (damped oscillations), especially at the beginning of the process. But, this phenomenon settles down towards the steady states (global stability under some strict conditions as shown in Lemma 2 above) at a later time. Introduction of time delay destabilizes the system (complex dynamics occur) and periodic solutions arise (see Figures 5-10).

References


IJNS homepage: http://www.nonlinearscience.org.uk/


