Stability Analysis of a delayed HIV/AIDS Epidemic Model with Saturated Incidence

Debashis Biswas [†*] , Samares Pal [‡0]

[†] *Calcutta Girls' College, Kolkata-13, India.*

[‡] *Department of Mathematics, University of Kalyani, Kalyani- 741235, India.*

Abstract

In this paper, we investigate the effect of time delay on an HIV/AIDS epidemic model with saturated incidence rate. We accept that the individuals are being recruited into sexually matured age group at a constant rate and incorporates time delay for one to become infected and the other become fullyblown. We assume that the disease spread only by sexually transmission.The model consists two equilibria, namely, a disease-free equilibrium and an endemic equilibrium. We calculate the basic reproduction number by using the next generation matrix. Mathematical analyses consecrated that the global dynamics of the spread of the HIV/AIDS infectious disease are totally determined by the basic reproduction number R_0. *For the basic reproduction number* $R_0 < 1$, *and* $\tau_1 \geq 0$ *the disease-free equilibrium is locally asymptotically stable. Whether* $R_0 > 1$, *and* $\tau_1 \geq 0$ *the endemic equilibrium point is locally asymptotically stable. Finally; we find the numerical solution of the model which justify the analytical results.*

Key words: *HIV/AIDS Epidemic model, Time Delay, Basic reproduction number, Stability, Saturated Incidence.*

1.Introduction

The first case of Acquired Immunodeficiency Syndrome (AIDS) was reported by Centers for Disease Control (CDC) of America in 1981 [1]. Human Immunodeficiency Virus (HIV) that causes AIDS, is on of the World most serious disease. There approximately 36.7 million people World wide living with HIV/AIDS at the end of 2015. Human Immunodeficiency Virus infection and acquired immunodeficiency syndrome (HIV/AIDS) is a spectrum of conditions caused by infection with the human immunodeficiency virus (HIV) [3-5]. Different observation have been conducted to study the dynamics of HIV/AIDS without time delay [6, 8, 9, 12]. However a few modeling studies have been conducted to describe the effect of time delays [13-15]. Mainly HIV transmission in human body has two basic modes, horizontal transmission and vertical transmission. Here we assume that the disease only spread by horizontal transmission means sexual contact. In 1986, the first known case of HIV in India was diagnosed amongst female sex workers in Chennai. In the very next the disease spread rapidly all over the India. Now India has the third highest number of people living with HIV in the world. The disease spread maximum by heterosexual transmission. A sex-structured mathematical model for heterosexual transmission of HIV/AIDS with explicit incubation period was studied by Mukandavire [16]. Here also we assume that when the susceptible class are contact with the infected class, then the disease spread. Hence it requires some time delay, say $\tau_1 > 0$, for it to be detectable; that is, for an infected individuals to become infective/infectious. Here is a time lag $\tau_2 > 0$, that is infective to become fully blown with AIDS symptoms. Here we proposed the mathematical model and find out the basic reproduction number by using next generation matrix and study their stability. The model is solved numerically by using an iterative numerical technique, which justify thetheoretical results.

2.*The mathematical model*

Since the disease spread only by sexually transmission and the individuals are being recruited sexually matured age group at a constant rate. Here we can divided the heterosexual population into three compartments namely, $S(t)$ the sexually mature susceptible at a time $t > 0$, $I(t)$ the number of individuals at a time t who are already infected with HIV, $A(t)$ the number of individuals who have developed full blown AIDS symptoms at a time t. The parameters, β is the probability of getting infected from a randomly chosen partner, μ is the natural death rate, α is a positive constant, θ the rate at which HIV infected individuals progress to AIDS, d AIDS related death rate, τ_1 is the time that taken for an individuals to become infectious after being in contact

with an infected τ_2 the time that taken for infected individuals to become fully blown with AIDS after becoming infectious, B recruitment rate of susceptible class into a sexually active population. We chosen the sexually matured individuals that is susceptible haphazardly and uniformly from the population. If the chosen individuals is infected, then the susceptible individuals is assumed to get the virus. When we tested the individuals after a time τ_1 then it will be considered as infectious. Without any awareness and drug intervention, the disease will taken progress to fully blown into the infected individuals after a time $\tau_2 > 0$. Here we also assumed that the following assumptions: (a) The recruitment rate of the population(sexually matured adults) is mainly by birth. (b) An individuals once at a time being infected then it become and remains infective until death. (c) In this study the population is homogeneous; that is, there is identical mixing. (d) The force of infection depends on the number of infective in the population by the product $\frac{\beta I}{1+\alpha I}$. (e) The full blown AIDS individuals are easily acknowledged in the population; that is, they do not engage in the transmission dynamics.

Infected individuals may die due to natural death or progress of fully blown individuals compartment. After progression of fully blown compartment, individuals are removed from this compartment due to natural deaths or disease related deaths. Under this assumptions our model equation are given by.

$$\begin{aligned}
\frac{dS}{dt} &= B - \frac{\beta SI}{1+\alpha I} - \mu S, \\
\frac{dI}{dt} &= \frac{\beta SI}{1+\alpha I} - (\mu+\theta)I, \\
\frac{dA}{dt} &= \theta I - (\mu+d)A.
\end{aligned} \tag{1}$$

Suppose that an individuals being in contact with infected individuals, after some time τ_1, to be clinically infective. Further let an infective individuals become fully blown after a some time τ_2. Under this consideration our model (1) reduces to

$$\begin{aligned}
\frac{dS}{dt} &= B - \frac{\beta SI}{1+\alpha I} - \mu S, \\
\frac{dI}{dt} &= \frac{\beta S(t-\tau_1)I(t-\tau_1)}{1+\alpha I(t-\tau_1)} - (\mu+\theta)I, \\
\frac{dA}{dt} &= \theta I(t-\tau_2) - (\mu+d)A.
\end{aligned} \tag{2}$$

Let $\tau = max[\tau_1, \tau_2], \Psi(\theta) := [S(\theta), I(\theta), A(\theta)], \theta \in [-\tau, 0]$, with the norm of Ψ defined as $\|\Psi\| = \sup_{-\tau \le \theta \le 0} |\Psi(\theta)|$ where $\|.\|$ is the norm in $\in \mathbf{R}^3$. Therefore the initial condition for equation (2) is $\Psi(\theta) := [S(\theta), I(\theta), A(\theta)]$, where $S(\theta) \ge 0$, $I(\theta) \ge 0$, and $A(\theta) \ge 0$, for all $\theta \in [-\tau, 0]$. Equation (2) subject to the above assumption has a unique solution [7].

3.Basic Properties of the Model

3.1.Positivity of the Solutions

Since the model suggest human population we need to show that all the state variables remain non-negative for all times.

Lemma 3.1. 1. *Let* $\Gamma = [(S, I, A) \in \mathbf{R}^3 : S(0) > 0,\ I(0) > 0\ ,\ A(0) > 0\ ]$ *then the solutions of* $S(t)$, $I(t)$, $A(t)$ *of the system (2) are positive for all* $t \ge 0$.

Proof:Taking the first equation of equation (2), we have $\frac{dS}{dt} = B - \frac{\beta SI}{1+\alpha_1 I} - \mu S > -(\frac{\beta I}{1+\alpha_1 I} + \mu)S$,

$S(t) > S(0)exp[-\int(\frac{\beta I}{1+\alpha_1 I} + \mu)d\xi] \geq 0$, here integration limit is 0 to t, similarly we can prove that $I(t) \geq \frac{I(0)}{e^{(\mu+\theta)t}} \geq 0$, and $A(t) \geq \frac{A(0)}{e^{(\mu+d)t}} \geq 0$. Therefore all solutions of the system (2) are positive for all $t \geq 0$ in the region Γ.

3.2.Invariant Region

Lemma 3. 2.1.*The system (2) has solutions which are contained, in the feasible region* Γ.

Proof: Let $\Gamma = (S, I, A) \in \mathbf{R}^3$ be any solution of the systems with non negative initial conditions then adding the equation of the system(2), we have $[S(t)+I(t)+A(t)]' \leq B - \mu[S(t)+I(t)+A(t)]$, $Hence, \limsup_{t\to\infty}(S+I+A) \leq \frac{B}{\mu}$. This implies that all solutions of model $(2) \in \mathbf{R}^3$ are bounded and eventually enter the attracting set Γ.

4.Equilibrium points and Basic reproduction number

The system (2) convey the following equilibria: (a) the disease free equilibrium $E_0 = (\frac{B}{\mu}, 0, 0)$, (b) the endemic equilibrium $E^* = (S^*, I^*, A^*)$ where

$$S^* = \frac{B\beta + B\alpha\mu R_0}{\mu R_0(\beta+\alpha\mu)}, I^* = \frac{\mu(R_0-1)}{\beta+\alpha\mu}, A^* = \frac{\mu\theta(R_0-1)}{(\mu+d)(\beta+\alpha\mu)}$$

It is clear that if $R_0 > 1$ then there exists a unique equilibria exists and if $R_0 < 1$ then there is no positive equilibria. The local stability of E_0 is governed by the basic reproduction number R_0 which can be found from the next generation matrix. The non-negative matrix, f of the infection terms and the non singular matrix, v of the transition terms are

$$f = \begin{pmatrix} \frac{B\beta}{\mu} & 0 \\ \theta & 0 \end{pmatrix}, v = \begin{pmatrix} \mu+\theta & 0 \\ 0 & \mu+d \end{pmatrix} and\ T = FV^{-1} = \begin{pmatrix} \frac{B\beta}{\mu(\mu+\theta)} & 0 \\ \frac{\theta}{\mu+\theta} & 0 \end{pmatrix}$$

where $F = Jacobian\ of\ f\ at\ disease\ free\ equilibrium$ and $V = Jacobian\ of\ v\ at\ disease\ free\ equilibrium$

$R_0 = \rho FV^{-1}$. Spectral of the matrix $R_0 = \frac{B\beta}{\mu(\mu+\theta)}$

5.Global stability of endemic equilibrium

Theorem 5. 0.1.*If* $R_0 > 1$, *then the system (2) at endemic equilibrium* E^* *is globally asymptotically stable, and unstable otherwise,*

Proof: To proof this result, we construct the second additive compound matrix $J^{[2]}$ for the system () at E^* is

given below $J^{[2]} = \begin{pmatrix} A_{11} & 0 & 0 \\ \frac{\theta I}{A} & A_{22} & -\frac{\beta S}{(1+\alpha I)^2} \\ 0 & \frac{\beta I}{1+\alpha I} & A_{33} \end{pmatrix}$

where

$$A_{11} = -\frac{\beta I}{1+\alpha I} - \mu - (\mu+\theta) + \frac{\beta S}{(1+\alpha I)^2}, A_{22} = -\frac{\beta I}{1+\alpha I} - \mu - (\mu+d) \text{and } A_{33} = \frac{\beta S}{(1+\alpha I)^2} - (\mu+\theta) + (\mu+d)$$

and

$$P = P(S,I,A) = diag(1, \frac{I}{A}, \frac{I}{A}) \text{ with } P^{-1} = (1, \frac{A}{I}, \frac{A}{I}) \text{ and } P_f = (0, \frac{\dot{I}}{A} - \frac{I\dot{A}}{A^2}, \frac{\dot{I}}{A} - \frac{I\dot{A}}{A^2}), \text{so } P_f P^{-1} = (0, \frac{\dot{I}}{I} - \frac{\dot{A}}{A}, \frac{\dot{I}}{I} - \frac{\dot{A}}{A})$$

and

$$\overline{G} = P_f P^{-1} + PJ^{[2]}P^{-1} = \begin{pmatrix} 0 & 0 & 0 \\ 0 & \frac{\dot{I}}{I} - \frac{\dot{A}}{A} & 0 \\ 0 & 0 & \frac{\dot{I}}{I} - \frac{\dot{A}}{A} \end{pmatrix} + \begin{pmatrix} A_{11} & 0 & 0 \\ \frac{\theta I}{A} & A_{22} & -\frac{\beta S}{(1+\alpha I)^2} \\ 0 & \frac{\beta I}{1+\alpha I} & A_{33} \end{pmatrix}$$

Let

$$\hat{G} = \begin{pmatrix} \hat{G}_{11} & \hat{G}_{12} \\ \hat{G}_{21} & \hat{G}_{22} \end{pmatrix}, where \hat{G}_{11} = A_{11}, \hat{G}_{12} = \begin{pmatrix} 0 & 0 \end{pmatrix}, \hat{G}_{21} = \begin{pmatrix} \frac{\theta I}{A} \\ 0 \end{pmatrix} and \ \hat{G}_{22} = \begin{pmatrix} A_{22} + \frac{\dot{I}}{I} - \frac{\dot{A}}{A} & -\frac{\beta S}{(1+\alpha I)^2} \\ \frac{\beta I}{1+\alpha I} & A_{33} + \frac{\dot{I}}{I} - \frac{\dot{A}}{A} \end{pmatrix}$$

Let $(\hat{v}_1, \hat{v}_2, \hat{v}_3)$ represent the vector in R^3

and the associated norms is $\|.\|$, defined as $\|(\hat{v}_1, \hat{v}_2, \hat{v}_3)\| = max(|\hat{v}_1|, |\hat{v}_2|, |\hat{v}_3|)$. Let $\mu\hat{G}$ represents Lozinski measure with the help of the above defined norm, so as described in [17], we assume $\mu(\hat{G}) \le sup(g_1, g_2)$, where $g_1 = \mu(\hat{G}_{11}) + |\hat{G}_{12}|$, $g_2 = \mu(\hat{G}_{22}) + |\hat{G}_{21}|$, $|\hat{G}_{12}|$ and $|\hat{G}_{21}|$ are the matrix norm with respect to the vector λ and μ_1 represent the Lozinski measure with to this l norm, then

$$\mu(\hat{G}_{11}) = -\frac{\beta I}{1+\alpha I} - \mu - (\mu+\theta) + \frac{\beta S}{(1+\alpha I)^2}, \mu(\hat{G}_{22}) = max[A_{22} + \frac{\dot{I}}{I} - \frac{\dot{A}}{A}, A_{33}, \frac{\beta I}{1+\alpha I}, -\frac{\beta S}{(1+\alpha I)^2}]$$

Now

$$g_1 = \mu(\hat{G}_{11}) + |\hat{G}_{12}| = -\frac{\beta I}{1+\alpha I} - \mu - (\mu+\theta) + \frac{\beta S}{(1+\alpha I)^2} \le \frac{\dot{I}}{I} - \mu - \frac{\beta I}{1+\alpha I} - \frac{\beta S}{1+\alpha I} = \frac{\dot{I}}{I} - \mu$$

and

$$g_2 = \mu\hat{G}_{22} + |\hat{G}_{21}| = \frac{\theta I}{A} + \frac{\dot{I}}{I} - \frac{\dot{A}}{A} - \frac{\beta S}{(1+\alpha I)^2} + (\mu+\theta) + max(A_{22}, A_{33}) =$$

$$\frac{\theta I}{A} + \frac{\dot{I}}{I} - \frac{\dot{A}}{A} + (\mu+\theta) - \frac{\beta S}{(1+\alpha I)^2} - \frac{\beta I}{1+\alpha I} - \mu - (\mu+d) \le \frac{\dot{I}}{I} - \mu + (\mu+\theta) - \frac{\beta I}{1+\alpha I} = \frac{\dot{I}}{I} - \mu$$

Therefore, $\hat{\mu G} \le sup(g_1, g_2) \le \frac{\dot{I}}{I} - \mu$ then $p = \frac{1}{t}[\int \hat{\mu G}\, ds] \le \frac{1}{t}[\int (\frac{\dot{I}}{I} - \mu)]ds = \frac{1}{t}\lg \frac{I(t)}{I(0)} - \mu$, here integration limit is 0 to t implies that $p \le -\frac{\mu}{2} < 0$. Hence the result [18], conclude that the endemic equilibrium point E^* is globally asymptotically stable.

5.1. Global stability of disease free equilibrium E_0

Lemma 5. 1. 1.*If* $R_0 < 1$ *, then the disease free equilibrium point* E_0 *of the model(1) is globally asymptotically stable in* Γ *and unstable if* $R_0 > 1$.

Proof: Define Lyapunov function:

$L = \beta(\mu + d)I$

$L' = \beta(\mu + d)I'$

$L' = \beta(\mu + d)[\frac{\beta SI}{1+\alpha I} - (\mu + \theta)I$

$L' = \beta(\mu + d)[\frac{\beta S}{1+\alpha I} - (\mu + \theta)]I$

$L' \le \beta(\mu + d)[\frac{\beta B}{(1+\alpha I)\mu} - (\mu + \theta)]I$, $since\, S = \frac{B\beta}{\mu}$

$= \beta(\mu + d)(\mu + \theta)[\frac{R_0}{1+\alpha I} - 1]I$

If $I = 0$, $L' = 0$ but if $I \neq 0$ and $R_0 < 1$, $L' < 0$ therefore , the disease free equilibrium is globally asymptotically stable in Γ.

6.Stability Analysis of the delayed model

6.1. Local Stability

We find the local stability of the disease free and endemic states in current section. We present the local stability of disease free equilibrium $E_0 = (\frac{B}{\mu}, 0, 0)$ in the following theorem

Theorem 6. 1.1.*The disease free equilibrium* E_0 *is locally asymptotically stable if and only if* $\tau_1 \ge 0$ *whenever* $R_0 < 1$.

Proof. The variational matrix about the disease free equilibrium point E_0 is given by

$$J_0 = \begin{pmatrix} -\mu & -\frac{B\beta}{\mu} & 0 \\ 0 & \frac{B\beta}{\mu} e^{-\lambda\tau_1} - (\mu + \theta) & 0 \\ 0 & \theta e^{-\lambda\tau_2} & -(\mu + d) \end{pmatrix}$$

The eigenvalues associated to J_0 are, $\lambda_1 = -(\mu + d) < 0$ and the roots of the equation

$$P\lambda^2 + Q\lambda + R = 0 \qquad (3)$$

, where $P=1,\ Q=2\mu+\theta-\frac{B\beta}{\mu}e^{-\lambda\tau_1}$ and $R=\mu(\mu+\theta)-B\beta e^{-\lambda\tau_1}$

For $\tau_1=0$ the above equations (3) becomes

$$\lambda^2+\mu+(\mu+\theta)(1-R_0)\lambda+\mu(\mu+\theta)(1-R_0)=0 \tag{4}$$

If $R_0<1$ then the roots of the equation (4) have negative real parts. Therefore E_0 is locally asymptotically stable if and only if $\tau_1=0$ For $\tau_1\geq 0$, by corollary 2.4 in Ruan and Wei [10], it follows that if instability occurs for a particular value of the delay τ_1, a characteristic root of (3) must intersect the imaginary axis. Let $\lambda=\omega i$, $\omega>0$ is the roots of (3). Putting the value $\lambda=\omega i$ into the equation (3) we get

$$-\omega^2+i[\omega(2\mu+\theta)-\frac{\omega B\beta\cos\omega\tau_1}{\mu}+B\beta\sin\omega\tau_1]-\frac{\omega B\beta\sin\omega\tau_1}{\mu}-B\beta\cos\omega\tau_1+\mu(\mu+\theta)=0 \tag{5}$$

Separating the real and imaginary part, gives

$$\frac{\omega B\beta\sin\omega\tau_1}{\mu}+B\beta\cos\omega\tau_1=\mu(\mu+\theta)-\omega^2 \tag{6}$$

$$\frac{\omega B\beta\cos\omega\tau_1}{\mu}-B\beta\sin\omega\tau_1=\omega(2\mu+\theta) \tag{7}$$

Squaring and adding the equation (6) and (7), gives

$$\omega^4+\omega^2[(\mu+\theta)^2+\mu^2+\frac{\beta^2\mu^2}{\mu^2}]+\mu^2(\mu+\theta)^2(1-R_0^2)=0 \tag{8}$$

Therefore it is clear that if $R_0<1$, then the roots of (8) have negative real parts. Such that equation (3) cannot have purely imaginary solutions. Therefore we conclude that the disease free equilibrium E_0 is locally asymptotically stable if and only if $\tau_1\geq 0$ whenever $R_0<1$.

6.2.Local Stability of Endemic Equilibrium :

Theorem 6. 2.1. *For $R_0>1$, the endemic equilibrium point E^* is locally asymptotically stable, if and only if $\tau_1\geq 0$.*

Proof.The Jacobian matrix J^* about the endemic equilibrium point E^* is given by,

$$J^*=\begin{pmatrix} -\frac{\beta I^*}{1+\alpha I^*}-\mu & -\frac{\beta S^*}{(1+\alpha I^*)^2} & 0 \\ \frac{\beta I^* e^{-\lambda\tau_1}}{1+\alpha I^*} & \frac{\beta S^* e^{-\lambda\tau_1}}{(1+\alpha I^*)^2}-(\mu+\theta) & 0 \\ 0 & \theta e^{-\lambda\tau_2} & -(\mu+d) \end{pmatrix}$$

The eigenvalues associated to J^* are, $\lambda_1=-(\mu+d)<0$ and the roots of the equation

$$\lambda^2+p_1\lambda+p_2-(q_2+q_3\lambda)e^{-\lambda\tau_1}=0 \tag{9}$$

$$p_1=\mu+\theta+\mu+\frac{\beta I^*}{1+\alpha I^*}=\mu+\theta+\mu+\frac{\beta\mu(R_0-1)}{\beta+\alpha\mu R_0},$$

$$p_2=\mu(\mu+\theta)+\frac{(\mu+\theta)\beta I^*}{1+\alpha I^*}=\mu(\mu+\theta)+\frac{\beta\mu(\mu+\theta)(R_0-1)}{\beta+\alpha\mu R_0},$$

$$q_2 = \frac{\beta\mu S^*}{(1+\alpha I^*)^2} = \frac{B\beta(\beta+\alpha\mu)}{R_0(\beta+\alpha\mu R_0)} \text{ and } q_3 = \frac{\beta S^*}{(1+\alpha I^*)^2} = \frac{B\beta(\beta+\alpha\mu)}{\mu R_0(\beta+\alpha\mu R_0)}.$$

For $\tau_1 = 0$, equation (9) becomes

$$\lambda^2 + a_1\lambda + a_2 = 0, \text{ where} \tag{10}$$

$$a_1 = p_1 - q_3 = \mu+\theta+\mu+\frac{\beta\mu(R_0-1)}{\beta+\alpha\mu R_0} - \frac{B\beta(\beta+\alpha\mu)}{\mu R_0(\beta+\alpha\mu R_0)} = \frac{\alpha\mu(\mu+\theta)(R_0-1)+\mu R_0(\beta+\alpha\mu)}{\beta+\alpha\mu R_0} \text{ and}$$

$$a_2 = p_2 - q_2 = \mu(\mu+\theta)+\frac{\beta\mu(\mu+\theta)(R_0-1)}{\beta+\alpha\mu R_0} - \frac{B\beta(\beta+\alpha\mu)}{R_0(\beta+\alpha\mu R_0)} = \frac{(R_0-1)(\alpha\mu^3+\alpha\theta\mu^2+\beta\mu^2+\beta\mu)}{\beta+\alpha\mu R_0}$$

.

It is clear that, for $R_0 > 1$, $(a_1, a_2) > 0$. Therefore, using the Routh-Hurwitz criterion [11], the roots of the equation (10) have negative real parts. Then we conclude that the endemic equilibrium E^* is locally asymptotically stable for $\tau_1 = 0$. For, after putting $\lambda = \omega i$, $\omega > 0$ into the equation (9) and separating the real and imaginary parts we get

$$q_2\cos\omega\tau_1 + q_3\omega\sin\omega\tau_1 = p_2 - \omega^2 \tag{11}$$

$$q_3\omega\cos\omega\tau_1 - q_2\sin\omega\tau_1 = p_1\omega \tag{12}$$

Squaring and adding equation no (11) and (12), we have

$$\omega^4 + \omega^2(p_1^2 - 2p_2 - q^2) + p_2^2 - q_2^2 = 0 \tag{13}$$

Let $u_1 = \omega^2$, then equation (13) can be rewritten as

$$u_1^2 + pu_1 + q = 0, \text{ where} \tag{14}$$

$$p = p_1^2 - 2p_2 - q_3^2$$

$$= [\mu+\theta+\mu+\frac{\beta\mu(R_0-1)}{\beta+\alpha\mu R_0}]^2 - 2[\mu(\mu+\theta)+\frac{\beta\mu(\mu+\theta)(R_0-1)}{\beta+\alpha\mu R_0}] - [\frac{B\beta(\beta+\alpha\mu)}{\mu R_0(\beta+\alpha\mu R_0)}]^2$$

$$= [\frac{(\mu+\theta)}{(\beta+\alpha\mu R_0)}]^2[\alpha\mu(R_0-1)(2\beta+\alpha\mu+\alpha\mu R_0)] + \mu^2 + \frac{2\beta\mu^2(R_0-1)}{\beta+\alpha\mu R_0} + [\frac{\beta\mu(R_0-1)}{\beta+\alpha\mu R_0}]^2 \text{ and}$$

$$q = p_2^2 - q_2^2$$

$$= [\mu(\mu+\theta)+\frac{\beta\mu(\mu+\theta)(R_0-1)}{\beta+\alpha\mu R_0}]^2 - [\frac{B\beta(\beta+\alpha\mu)}{R_0(\beta+\alpha\mu R_0)}]^2$$

$$= [\mu(\mu+\theta)+\frac{\beta\mu(\mu+\theta)(R_0-1)}{\beta+\alpha\mu R_0} + \frac{\mu(\mu+\theta)(\beta+\alpha\mu)}{(\beta+\alpha\mu R_0)}][\frac{(R_0-1)(\alpha\mu^3+\alpha\mu^2\theta+\beta\mu^2+\beta\mu\theta)}{\beta+\alpha\mu R_0}].$$

Therefore it is clear that if $R_0 > 1$, then $(p, q) > 0$, which satisfy the Lemma 3.3.1 in [2]. Then, we conclude that if $R_0 > 1$, the endemic equilibrium E^* is locally asymptotically stable for $\tau_1 \geq 0$.

7.Numerical Simulations

We now present numerical simulations for the stability analysis of a delayed HIV/AIDS epidemic model with saturated incidence to illustrate our results. Now for $B = 1.1$, $\mu = 0.01$, $\theta = 1.1$, $d = 0.002$, $\beta = 0.01$, $\alpha = 0.3$, $\tau_1 = 1$, and $\tau_2 = 0$ Fig. 1 shows the disease free equilibrium is locally asymptotically stable when $R_0 = 0.990991 < 1$. We choose $B = 3$, $\mu = 0.01$, $\theta = 1$, $d = 0.3$, $\beta = 0.07$, $\alpha = 0.01$, $\tau_1 = 1$

and $\tau_2 = 0$ while $R_0 = 20.792079 > 1$ then Fig. 2 shows the endemic equilibrium is locally asymptotically stable.

8.Conclusion

In this paper, we have proposed and analyzed a non-linear mathematical model to study the effect of time delay in the recruitment of infected persons on the transmission dynamics of HIV/AIDS. By analyzing the model, we have found a basic reproduction number R_0. It is noted that when $R_0 < 1$ then disease dies out and when $R_0 > 1$ then disease become endemic. The model has two non-negative equilibria namely, $E_0 = (\frac{B}{\mu},0,0)$, the disease free equilibrium and $E^* = (S^*, I^*, A^*)$, the endemic equilibrium . It is found that the equilibrium state E_0 corresponding to disappearance of disease is locally asymptotically stable if $R_0 < 1$ and $\tau_1 \geq 0$, unstable if $R_0 > 1$. The endemic equilibrium E^*, which exists only when $R_0 > 1$ is always asymptotically stable by using Jacobian matrix and globally asymptotically stable (by using second additive compound matrix) while $\tau_1 \geq 0$. There is no effect of double delay. We know there is no cure for HIV or AIDS and there are medicines and treatment that fight HIV and help people with HIV and AIDS live longer.

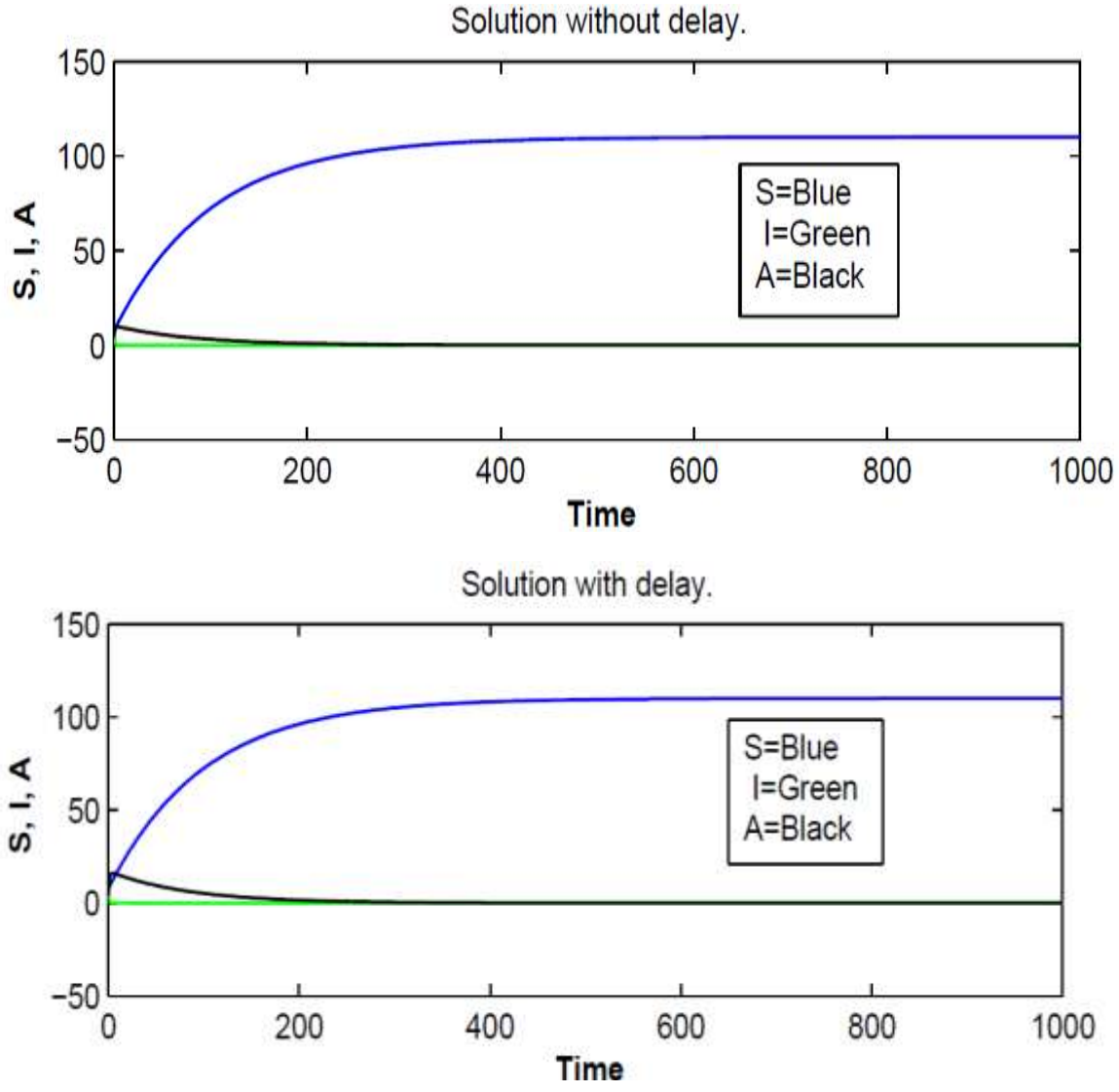

Figure 1: The equilibrium point E_0 is asymptotically stable while $R_0 < 1$.

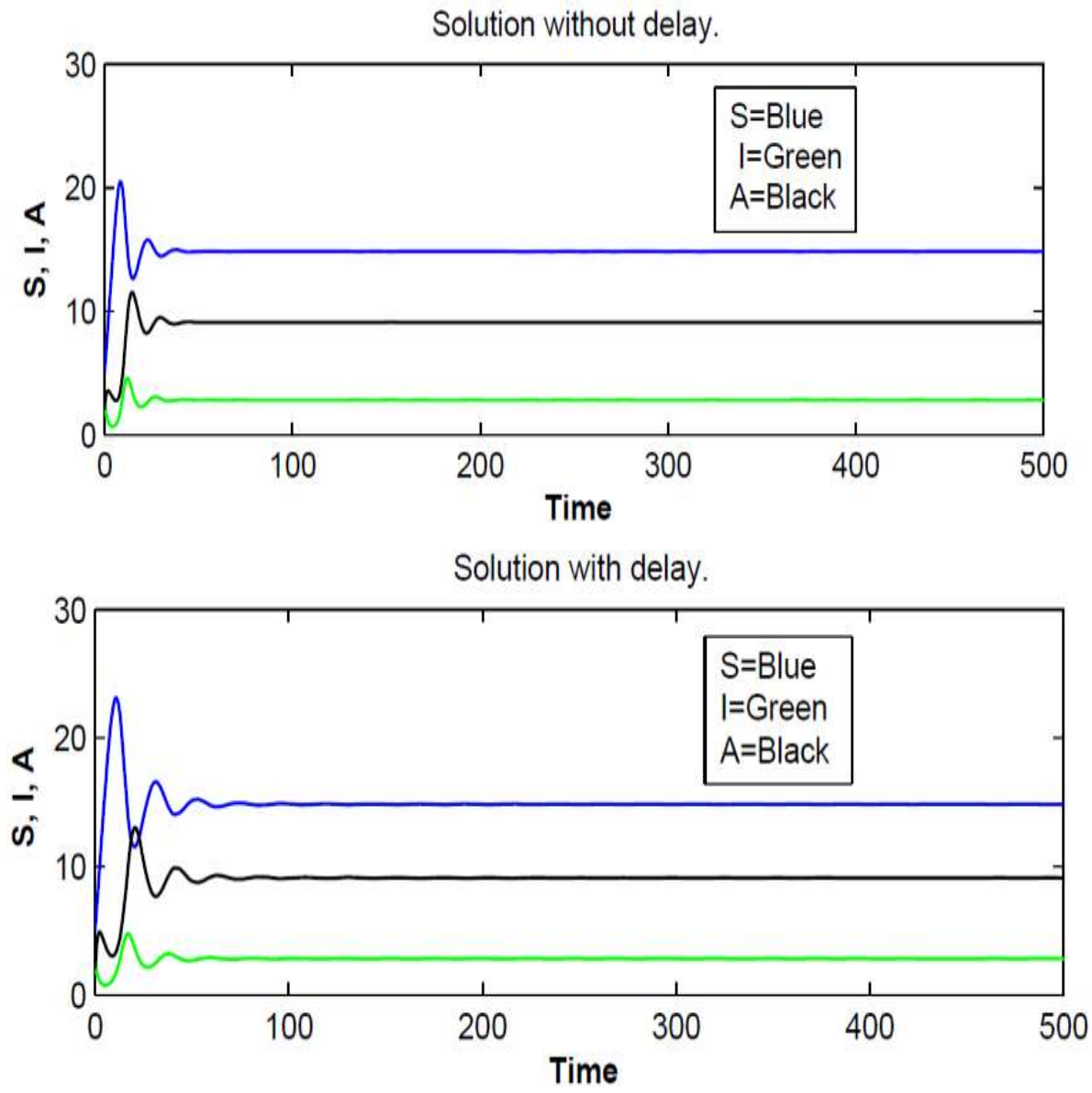

Figure 2: The equilibrium point E^* is locally asymptotically stable while $R_0 > 1$.

References

[1] Centers for Disease Control, (1981) Pneumocystis Pneumonia-Los Angeles Morbidity and Mortality Weekly Report. , **30**, 250-252.

[2] H. Wei, X. Li, and M. Martchev, " An epidemic model of a vector-borne disease with direct transmission and time delay, " J. Math. Anal. Appl., **342**, 895-908, (2008).

[3] Sepkowitz KA, " AIDS-the first 20 years " . N. Engl. J. Med. , **344(23)**, **2001**, 1764-72.

[4] Alexander Kramer, Mirjam Kretzschmar, Klaus Krickeberg, Modern infection disease epidemiology concepts, methods,mathematical models, and publichealth. New. York. Springer. , **2010**, p. 88.

[5] Wilhelm. Kirch. Encyclopedia of public health. New. York. Springer. , **2008**, pp. 676-677.

[6] Y. H. Hsieh, C. H. Chen, Modeling the social dynamics of a sex industry: its implications for spread of HIV/AIDS, Bull. Math. Biol. , **66**, **2004** , 143-166.

[7] Jack, H., Hale, K., Theory of Functional Differential Equations .2nd ed., Spinger Verlag, New York. , (1977).

[8] C. M. Kribs-Zaleta, M. Lee, C. Roman, S. Wiely, C. M. Hernandez-Suarez, The effect of the HIV/AIDS epidemic on Africa's truck drivers, Math. Bios. Eng.
2(4), **2005**, 771-788.

[9] A. S. R. Srinivasa Rao, Mathematical modeling of AIDS epidemic in India, Curr. Sci. , **84**, **2003**, 1192-1197.

10] S. Ruan and J. Wei, " On the zeros of transcendental functions with applications to stability of delay differential equations with two delays, " Dyn. Contin. Discrete. Implus. Syst., Ser. A. Math. Anal., **10**, 863-874, (2003).

[11] J. L. Willems, Stability Theory of Dynamical systems, New York: Nelson , (1988).

[12] A. Tripathi, R. Naresh, D. Sharma, Modelling the effect of screening of unaware infectives on the spread of HIV infection, Appl. Math. Comput. , **184**, **2007**, 1053-1068.

[13] Z. Mukandavire, W. Garia, C. Chiyaka, Asymptotic properties of an HIV/AIDS model with time delay, J. Math. Anal.Appl. , **330**, **2007**, 916-933.

[14] R. Naresh, A. Tripathi, J. M. Tchuenche, D. Sharma, Stability analysis of a time delay SIR epidemic model with nonlinear incidence rate, Comp. Math. Appl. **58**, **2009**, 348-359.

[15] S. Kovacs, Dynamics of HIV/AIDS model-The effect of time delay, Appl. Math. Comp. , **188(2)**, **2007**, 1597-1609.

[16] Mukandavire, Z., Chiyaka, C., Garira, W., and Musuka, G. Mathematical analysis of a sex-structured HIV/AIDS model with a discrete time delay. Nonlinear Analysis,, **71**, **2009**, 1082-1093.

17] R. Martin, Logarithmic norms and projections applied to linear differential systems, J. Math. Anal. Appl. , **45**, 432-454, (1974).

[18] M. Y. Li, J. S. Muldowney, A geometric approach to global-stability problems, SIAM J. Math. Anal. , **27**, 1070-1083, (1996).