

# Mathematical Modeling Applied to Understand the Dynamical Behavior of HIV Infection

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## Abstract

The study of viral dynamics of HIV/AIDS has resulted in a deep understanding of host-pathogenesis of HIV infection from which numerous mathematical modeling have been derived. Most of these models are based on nonlinear ordinary differential equations. In Bangladesh, the rate of increase of HIV infection comparing with the other countries of the world is not so high. Bangladesh is still considered to be a low prevalent country in the region with prevalence  $< 1\%$  among MARP (Most at risk populations). In this paper, we have presented the current situation of HIV infection in Bangladesh and also have discussed the mathematical representation of a three-compartmental HIV model with their stability analysis. We have determined the basic reproduction number  $R_0$  and shown the local and global stability at disease free and chronic infected equilibrium points. Also we have shown that if the basic reproduction number  $R_0 \leq 1$ , then HIV infection is cleared from T cell population and it converges to disease free equilibrium point. Whereas if  $R_0 > 1$ , then HIV infection persists.

## Keywords

CD<sup>4+</sup> T Cells, Dynamical Systems, Basic Reproduction Number, Equilibrium Points, Stability Analysis

## 1. Introduction

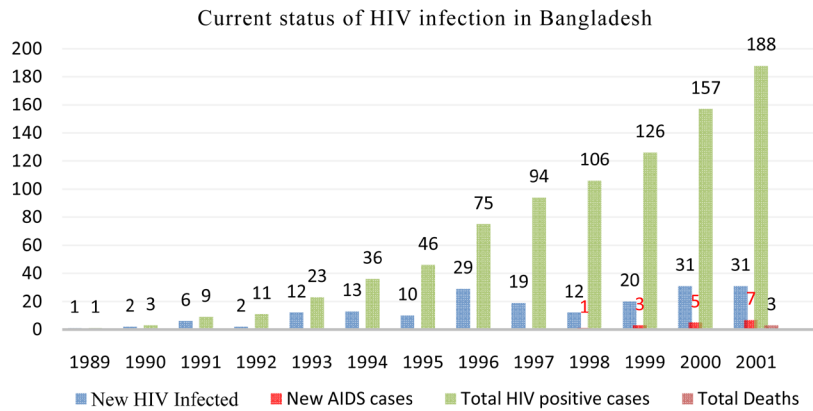
HIV stands for human immunodeficiency virus. The virus attacks the immune system, and weakens our ability to fight infections and disease. HIV/AIDS progresses in body slowly and its symptoms are shown after 6 - 8 years sometimes even later. At present, the most burning issue at the same time, the most dangerous phenomena is Human Immunodeficiency Virus (HIV) [1]. Since the beginning of the epidemic, more than 70 million people have been infected with

the HIV virus and about 35 million people have died of HIV. Globally, 36.7 million [34.0 - 39.8 million] people were living with HIV at the end of 2015 [2]. An estimated 0.8% [0.7% - 0.9%] of adults aged 15 - 49 years worldwide are living with HIV, although the burden of the epidemic continues to vary considerably between countries and regions. Sub-Saharan Africa remains the most severely affected, with nearly 1 in every 25 adults (4.4%) living with HIV and accounting for nearly 70% of the people living with HIV worldwide [2]. Acquired Immuno-deficiency Syndrome (AIDS) was first discovered in 1981, since then it has been considered as the most leading cause of mortality [3]. A detailed background and survey on HIV/AIDS is described in [4] [5] [6] [7]. HIV mainly targets CD<sup>4+</sup> T cells. The continuous attack HIV causes the depletion of CD<sup>4+</sup> T cells and this leads people to gradually become a victim of Acquired Immunodeficiency Syndrome (AIDS). For this reason, the count of CD<sup>4+</sup> T cells is considered as the primary indicator of progression of HIV. In recent times, mathematical modeling has become the most powerful tool to incorporate the dynamic behaviors of infectious diseases. Mathematical modeling is basically referred to as a method of simulating real-life situations with mathematical equations to forecast their future behavior [8]. Numerous mathematical models have been developed to identify the characteristics of human immunodeficiency virus [9] [10] [11]. HIV dynamic model, a set of ordinary differential equations (ODE) that describe the interaction between HIV virus and human body cells, has been proven useful for understanding the pathogenesis of HIV infection and developing treatment strategies [12]. In this paper, we have shown the present scenario of HIV/AIDS in Bangladesh. Also we have studied a three-compartmental HIV model and investigated their stability at disease free and endemic equilibrium points.

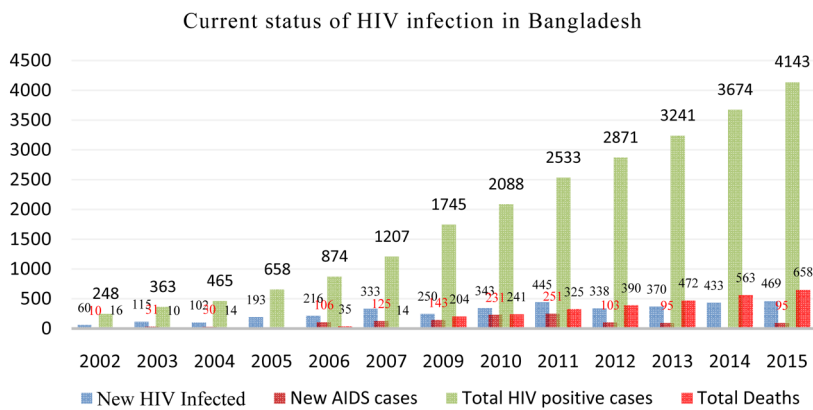
## 2. Current Status of HIV Infection in Bangladesh

HIV is a worldwide curse. There is no such country where this pandemic disease does not exist. Although Bangladesh is still considered to be a low responded HIV infected country in world, the present situation indicate that the influence of this pandemic disease is gradually increasing. The main reason for this low prevalence could be the early and sustained HIV prevention programs targeting high risk groups backed by a state-of-the-art surveillance system. Another contributing protective factor could be the high rates of male circumcision. There is, however, a concentrated HIV epidemic among injecting drug users (IDU), primarily due to sharing of unclean syringes and needles. As a result, the rate of new infections is still on the rise and Bangladesh is the only country in the South Asia Region where new infections are rising [13].

In Bangladesh, the first case of HIV was detected in 1989 [3]. Since then, it has been enhanced considerably. In 2015 (December 2014 to November 2015), the number of newly HIV infected people is 469 and the number of HIV/AIDS related death is 95. Till December 2015, there were 4143 reported cases of HIV and among them 658 died [6]. Here we show a graphical representation of HIV surveillance of Bangladesh (see **Figure 1**) from 1989 to 2015 (except 2008) [14].



(a)

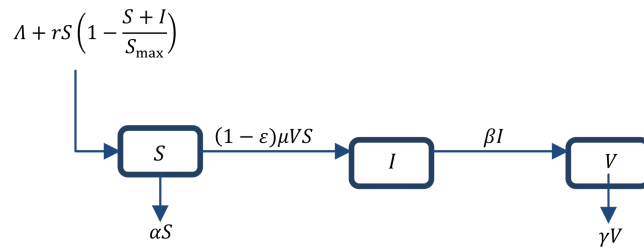


(b)

**Figure 1.** (a) Number of HIV and AIDS cases from 1989 to 2001; (b) Number of HIV and AIDS cases from 2002 to 2015 (except 2008).

### 3. Three-Compartmental HIV Model

To generate a realistic model of T cell infection by HIV, we first need to consider the population dynamics of T cells in the absence of HIV. Our interest is to present a mathematical model of HIV infection and analyze the model. In this paper, we present a three compartmental model of HIV which has been taken from [15]. We have modified this model and added a drug efficacy parameter  $\varepsilon$  whose value is in the range between 0 and 1 [16]. The total population size  $N$  is divided into three stages of HIV/AIDS progression; the susceptible population  $S$ , HIV infected individuals  $I$  and HIV virus  $V$ . The total population is given by  $N(t) = S(t) + I(t) + V(t)$ . The population  $CD^{4+}$  T cells starts with a source or production rate  $\Lambda$  and dead cells with rate  $\alpha$  are reduced from the susceptible class. It has a logistic growth with  $rS\left(1 - \frac{S+I}{S_{max}}\right)$  where  $r$  is the proliferation rate. Parameters  $\alpha$ ,  $\beta$  and  $\gamma$  are natural turnover rate of uninfected  $CD^{4+}$  T cells, infected  $CD^{4+}$  T cells and virus. Whereas  $S_{max}$  is the maximum level of  $CD^{4+}$  T cell concentration in the body [17]. Infected  $CD^{4+}$  T cells it has an infection rate which is concentrated as  $\mu$ . The transfer diagram of the model is shown in **Figure 2**.



**Figure 2.** Transmission diagram of three compartmental HIV model.

Our modified model is governed by the following ordinary differential equations:

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \alpha S + rS \left(1 - \frac{S+I}{S_{\max}}\right) - (1 - \epsilon)\mu VS, \\ \frac{dI}{dt} &= (1 - \epsilon)\mu VS - \beta I, \\ \frac{dV}{dt} &= \beta I - \gamma V. \end{aligned} \tag{1}$$

The model is positively invariant and bounded in the region

$$\Omega = \left\{ S, I, V \in \mathbb{R}_+^3 : N(t) \leq \frac{\Lambda}{\mu} \right\}.$$

We have determined the basic reproduction number  $R_0$  which was first introduced by Ross (1909), which is defined in epidemiological modeling as the average number of infected individuals produced by one infected immigrant in a population which is completely susceptible [18]. Finding the basic reproduction number  $R_0$ , we can determine the endemic result of disease in populations. If  $R_0 < 1$ , the disease vanishes and if  $R_0 > 1$ , the disease spreads and goes to the endemic level.

### Parameter Specification

If one wishes to use a mathematical model to make predictions about a particular individual or population, estimation of model parameters from data is crucial. All the parameters and their values used for model (1) are taken from [15] [16] and presented in **Table 1**.

**Table 1.** Parameters used for model (1).

Description	Symbols	Values
CD <sup>4+</sup> T cell source rate	$\Lambda$	0.1 mm <sup>-3</sup> .day <sup>-1</sup>
Natural turnover rate of uninfected CD <sup>4+</sup> T cell	$\alpha$	0.02 day <sup>-1</sup>
Natural turnover rate of infected CD <sup>4+</sup> T cell	$\beta$	0.3 day <sup>-1</sup>
Natural turnover rate of virus	$\gamma$	2.4 day <sup>-1</sup>
Drug efficacy	$\epsilon$	0.5
CD <sup>4+</sup> T cell infection rate	$\mu$	0.0027 mm <sup>-3</sup> .day <sup>-1</sup>

## 4. Mathematical Analysis of Model

Here we investigate the positivity of the model, find out different equilibrium points, formulate the basic reproduction number and check the stability at disease free and endemic equilibrium points.

### 4.1. Positivity of the Solution

Here we check the positivity of each compartments such as susceptible  $S$  cells, infected  $I$  and HIV virus  $V$ . We must have the positive values of these biological compartments. To test the positivity of these biological compartments, we need the following Lemma 1.

**Lemma 1.** Let  $S(0) > 0, I(0) \geq 0, S(0) > 0, I(0) \geq 0, V(0) \geq 0 \in \Omega$  then the solutions  $S(t), I(t), V(t)$  of the model system of equations (1) are positives.

**Proof:** To prove the Lemma 1, we have used the system of equations of the model (1).

$$\frac{dS}{dt} = \Lambda - \alpha S + rS \left( 1 - \frac{S+I}{S_{\max}} \right) - (1-\varepsilon)\mu VS,$$

in order to find the positivity we have,

$$\begin{aligned} \frac{dS}{dt} &\geq \Lambda - \alpha S \Rightarrow \frac{dS}{dt} + \alpha S \geq \Lambda \\ \therefore IF = e^{\int \alpha dt} &\geq e^{\alpha t}. \end{aligned} \quad (2)$$

Multiplying both sides of (2) by  $e^{\alpha t}$  we have,

$$\begin{aligned} e^{\alpha t} \frac{dS}{dt} + \alpha e^{\alpha t} S &\geq \Lambda e^{\alpha t} \Rightarrow \frac{d}{dt} \left( S e^{\alpha t} \right) \geq e^{\alpha t} \Lambda \\ &\Rightarrow d \left( S e^{\alpha t} \right) \geq e^{\alpha t} \Lambda dt. \end{aligned} \quad (3)$$

Now Integrating (3)

$$S e^{\alpha t} \geq e^{\alpha t} \frac{\Lambda}{\alpha} + c_1, \quad (4)$$

where  $c_1$  is a constant. Applying the initial condition at  $t=0, S(t) \geq S(0)$ . Hence from (4),

$$S \geq \frac{\Lambda}{\alpha} + c_1 \Rightarrow c_1 \geq \left( \frac{\Lambda}{\alpha} - S \right).$$

Putting the value of  $c_1$  into (4), we get

$$S e^{\alpha t} \geq e^{\alpha t} \frac{\Lambda}{\alpha} + \left( S - \frac{\Lambda}{\alpha} \right) \Rightarrow S \geq \frac{\Lambda}{\alpha} + \left( S - \frac{\Lambda}{\alpha} \right) e^{-\alpha t}.$$

Hence  $S > 0$  at  $t=0$  and  $t \rightarrow \infty$ . Similarly we can find the positivity of  $I$  and  $V$  under the initial conditions.

Therefore, it is true that,  $(S(t) > 0, I(t) \geq 0, V(t) \geq 0, \forall t \geq 0)$ .

### 4.2. Disease Free Equilibrium Points

The disease free equilibrium of the above HIV model (1) can be obtained by set-

ting

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dV}{dt} = 0$$

thus we have,

$$\begin{aligned} \Lambda - \alpha S + rS \left( 1 - \frac{S+I}{S_{\max}} \right) - (1-\varepsilon)\mu VS &= 0, \\ (1-\varepsilon)\mu VS - \beta I &= 0 \text{ and } \beta I - \gamma V = 0. \end{aligned}$$

Since we have considered the disease free equilibrium, hence  $I = V = 0$ . Thus the above system reduces to,

$$\begin{aligned} \Lambda - \alpha S_0 + rS_0 - \frac{rS_0^2}{S_{\max}} &= 0 \Rightarrow rS_0 - \frac{rS_0^2}{S_{\max}} = \alpha S_0 - \Lambda \\ \Rightarrow rS_0 S_{\max} - rS_0^2 &= \alpha S_0 S_{\max} - \Lambda S_{\max} \\ \Rightarrow rS_0^2 - rS_0 S_{\max} + \alpha S_0 S_{\max} - \Lambda S_{\max} &= 0 \\ \therefore S_0 &= \frac{S_{\max}}{2r} \left[ (r-\alpha) + \sqrt{(r-\alpha)^2 + \frac{4\Lambda r}{S_{\max}}} \right]. \end{aligned}$$

Thus, the disease free equilibrium is  $W_0 = (S_0, 0, 0)$ .

Again for the endemic equilibrium point  $W^*$ , we find  $W^* = (S^*, I^*, V^*)$ , where

$$S^* = \frac{\gamma}{(1-\varepsilon)\mu}, \quad I^* = \frac{\gamma}{(1-\varepsilon)\beta} V^*, \quad V^* = \frac{\Lambda\mu^2 + (r-\alpha)\gamma\mu - \frac{r}{S_{\max}}\gamma^2}{\mu\gamma \left( \mu + \frac{r\gamma}{\beta S_{\max}} \right) (1-\varepsilon)}.$$

Now we calculate the basic reproduction number  $R_0$  at  $W_0$ .

### 4.3. Basic Reproduction Number $R_0$

Basic reproduction number represents the average number of secondary infection caused by a single infected T cell in an entirely susceptible T cell population, throughout its period. In order to find the basic reproduction number of the model (1), we need to identify the classes which are relevant to each other. From the model (1), we observe that the classes  $I$  and  $V$  are relevant. We find the gain and losses of  $I$  and  $V$  respectively.

Gains to  $I$  is  $(1-\varepsilon)\mu VS$ , gains to  $V$  is  $\beta I$  losses to  $I$  is  $\beta I$  and losses to  $V$  is  $\gamma V$ . Now, Matrix for the gain terms:

$$\begin{aligned} F &= \begin{bmatrix} \frac{\partial}{\partial I} (1-\varepsilon)\mu VS & \frac{\partial}{\partial I} \beta I \\ \frac{\partial}{\partial V} (1-\varepsilon)\mu VS & \frac{\partial}{\partial V} \beta I \end{bmatrix} \\ \therefore F &= \begin{bmatrix} 0 & \beta \\ (1-\varepsilon)\mu S & 0 \end{bmatrix}. \end{aligned}$$

Since basic reproduction number is to be calculated at disease free equilibrium point  $W_0$ , hence

$$F = \begin{bmatrix} 0 & \beta \\ (1-\varepsilon)\mu S_0 & 0 \end{bmatrix}.$$

Matrix for the loss terms;

$$L = \begin{bmatrix} \frac{\partial}{\partial I}(\beta I) & \frac{\partial}{\partial I}(\gamma V) \\ \frac{\partial}{\partial V}(\beta I) & \frac{\partial}{\partial V}(\gamma V) \end{bmatrix},$$

$$\therefore L = \begin{bmatrix} \beta & 0 \\ 0 & \gamma \end{bmatrix}.$$

Inverse of  $L$  is  $\therefore L^{-1} = \begin{bmatrix} \frac{1}{\beta} & 0 \\ 0 & \frac{1}{\gamma} \end{bmatrix}.$

Now we have to evaluate a matrix  $G$  such that

$$G = FL^{-1}$$

$$\Rightarrow G = \begin{bmatrix} 0 & \beta \\ (1-\varepsilon)\mu S_0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\beta} & 0 \\ 0 & \frac{1}{\gamma} \end{bmatrix}, \therefore G = \begin{bmatrix} 0 & \frac{\beta}{\gamma} \\ \frac{(1-\varepsilon)\mu S_0}{\beta} & 0 \end{bmatrix}.$$

Hence the largest eigen value of the matrix  $G$  is  $\frac{(1-\varepsilon)\mu S_0}{\gamma}$ . Thus, the basic reproduction number of the model (1) is  $R_0 = \frac{(1-\varepsilon)\mu S_0}{\gamma}$ . Now we check the local stability of the model (1) at disease free equilibrium point  $W_0$  and chronic infection equilibrium point  $W^*$ .

#### 4.4. Local Stability of Disease Free Equilibrium Point $W_0$

Firstly, we investigate the local stability at disease free equilibrium point  $W_0$  but before that we need the following theorem.

**Theorem 1:** If  $R_0 < 1$ , the disease free equilibrium point  $W_0$  of system (1) is locally asymptotically stable. If  $R_0 = 1$ ,  $W_0$  is locally stable and if  $R_0 > 1$ , then  $W_0$  is unstable.

**Proof:** To prove the above theorem, the following variation matrix is computed corresponding to equilibrium point  $W_0$ . From the model (1), let

$$x = \frac{dS}{dt}, \quad y = \frac{dI}{dt}, \quad z = \frac{dV}{dt}$$

then the system (1) reduces to,

$$x = \Lambda - \alpha S + rS \left( 1 - \frac{S+I}{S_{\max}} \right) - (1-\varepsilon)\mu VS,$$

$$y = (1-\varepsilon)\mu VS - \beta I \quad \text{and} \quad z = \beta I - \gamma V.$$

The Jacobian Matrix of the system (1) is

$$J = \begin{bmatrix} \frac{\partial x}{\partial S} & \frac{\partial x}{\partial I} & \frac{\partial x}{\partial V} \\ \frac{\partial y}{\partial S} & \frac{\partial y}{\partial I} & \frac{\partial y}{\partial V} \\ \frac{\partial z}{\partial S} & \frac{\partial z}{\partial I} & \frac{\partial z}{\partial V} \end{bmatrix}, \tag{5}$$

$$\Rightarrow J = \begin{bmatrix} -\alpha + r \left( 1 - \frac{S+I}{S_{\max}} \right) - \frac{rS}{S_{\max}} - (1-\varepsilon)\mu V & -\frac{rS}{S_{\max}} & -(1-\varepsilon)\mu S \\ & (1-\varepsilon)\mu V & -\beta & (1-\varepsilon)\mu S \\ & 0 & \beta & -\gamma \end{bmatrix},$$

at  $W_0$ , Equation (5) becomes

$$J(W_0) = \begin{bmatrix} -\alpha + r \left( 1 - \frac{S_0}{S_{\max}} \right) - \frac{rS_0}{S_{\max}} & -\frac{rS_0}{S_{\max}} & -(1-\varepsilon)\mu S_0 \\ 0 & -\beta & (1-\varepsilon)\mu S_0 \\ 0 & \beta & -\gamma \end{bmatrix}.$$

Now we have to find out the characteristic equation. To do that, first we have to calculate  $J(W_0) - \lambda I$  where,  $\lambda$  is a scalar and  $I$  is identity matrix. Let  $B = J(W_0) - \lambda I$ , then

$$B = \begin{bmatrix} -\alpha + r \left( 1 - \frac{S_0}{S_{\max}} \right) - \frac{rS_0}{S_{\max}} & -\frac{rS_0}{S_{\max}} & -(1-\varepsilon)\mu S_0 \\ 0 & -\beta & (1-\varepsilon)\mu S_0 \\ 0 & \beta & -\gamma \end{bmatrix} - \begin{bmatrix} \lambda & 0 & 0 \\ 0 & \lambda & 0 \\ 0 & 0 & \lambda \end{bmatrix},$$

$$\therefore B = \begin{bmatrix} -\alpha + r \left( 1 - \frac{S_0}{S_{\max}} \right) - \frac{rS_0}{S_{\max}} - \lambda & -\frac{rS_0}{S_{\max}} & -(1-\varepsilon)\mu S_0 \\ 0 & -\beta - \lambda & (1-\varepsilon)\mu S_0 \\ 0 & \beta & -\gamma - \lambda \end{bmatrix}.$$

To find out the characteristic equation we need to perform  $\det(B) = 0$ , hence

$$\begin{aligned} & \left[ -\alpha + r \left( 1 - \frac{S_0}{S_{\max}} \right) - \frac{rS_0}{S_{\max}} - \lambda \right] \left[ (\beta + \lambda)(\gamma + \lambda) - (1-\varepsilon)\mu\beta S_0 \right] + \frac{rS_0}{S_{\max}} \times 0 - \mu S_0 \times 0 = 0, \\ \Rightarrow & \left[ -\alpha + r \left( 1 - \frac{S_0}{S_{\max}} \right) - \frac{rS_0}{S_{\max}} - \lambda \right] \left[ \beta\gamma + \lambda(\beta + \gamma) + \lambda^2 - (1-\varepsilon)\mu\beta S_0 \right] = 0, \\ \Rightarrow & \left[ -\alpha + r \left( 1 - \frac{S_0}{S_{\max}} \right) - \frac{rS_0}{S_{\max}} - \lambda \right] \left[ \lambda^2 + \lambda(\beta + \gamma) + \beta\gamma \left( 1 - \frac{(1-\varepsilon)\mu S_0}{\gamma} \right) \right] = 0, \\ \Rightarrow & \left[ -\alpha + r \left( 1 - \frac{S_0}{S_{\max}} \right) - \frac{rS_0}{S_{\max}} - \lambda \right] \left[ \lambda^2 + \lambda(\beta + \gamma) + \beta\gamma(1-R_0) \right] = 0, \\ \Rightarrow & \left[ -\alpha + r \left( 1 - \frac{S_0}{S_{\max}} \right) - \frac{rS_0}{S_{\max}} - \lambda \right] \left[ \lambda^2 + a_1\lambda + a_2 \right] = 0. \end{aligned}$$



Thus, the characteristic equation is

$$\left[ -\alpha + r \left( 1 - \frac{S_0}{S_{\max}} \right) - \frac{rS_0}{S_{\max}} - \lambda \right] [\lambda^2 + a_1\lambda + a_2] = 0,$$

where

$$\begin{aligned} a_1 &= \beta + \gamma, \\ a_2 &= \beta\gamma(1 - R_0). \end{aligned}$$

We observe that, first root of the characteristic equation is

$$\lambda_1 = -\alpha + r \left( 1 - \frac{S_0}{S_{\max}} \right) - \frac{rS_0}{S_{\max}} < 0.$$

If  $R_0 > 1$ , then  $a_2 > 0$ . Also,  $\gamma > (1 - \varepsilon)\mu S_0$ . Again  $a_1 > 0$ , hence by Routh-Hurwitz criteria [19],  $W_0$  locally asymptotically stable. If  $R_0 = 1$ , then  $R_0$  and  $W_0$  becomes locally stable. If  $R_0 < 1$ , then  $a_2 < 0$  and  $W_0$  becomes unstable. Now we investigate the local stability of endemic equilibrium point  $W^*$ .

#### 4.5. Local Stability of Chronic Infection Equilibrium Point $W^*$

Now we investigate the local stability of chronic infection equilibrium point  $W^*$ . We need the following Lemma 2.

**Lemma 2:** Let  $M$  be a  $3 \times 3$  real matrix. If  $\text{tr}(M)$ ,  $\det(M)$  and  $\det(M^{[2]})$  are all negative, then all of the eigen values of  $M$  have negative real part [20].

Before we apply the Lemma 2, we need the following definition of second additive compound matrix.

**Definition 1:** Let  $A = (a_{ij})$  be an  $n \times n$  real matrix. The second additive compound matrix of  $A$  is the matrix  $A^{[2]} = (b_{ij})$  defined as follows [21] [22]:

$$\begin{aligned} n = 2: A^{[2]} &= a_{11} + a_{22} \\ n = 3: A^{[2]} &= \begin{bmatrix} a_{11} + a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11} + a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22} + a_{33} \end{bmatrix}. \end{aligned}$$

**Theorem 2:** The chronic infection equilibrium point  $W^*$  of the system (1) is locally asymptotically stable if  $R_0 > 1$ .

**Proof:** From Equation (5), we have

$$\Rightarrow J = \begin{bmatrix} -\alpha + r \left( 1 - \frac{S+I}{S_{\max}} \right) - \frac{rS}{S_{\max}} - (1-\varepsilon)\mu V & -\frac{rS}{S_{\max}} & -(1-\varepsilon)\mu S \\ (1-\varepsilon)\mu V & -\beta & (1-\varepsilon)\mu S \\ 0 & \beta & -\gamma \end{bmatrix},$$

at chronic infection equilibrium point  $W^* = (S^*, I^*, V^*)$ ,

$$J(W^*) = \begin{bmatrix} -\alpha + r \left(1 - \frac{S^* + I^*}{S_{\max}}\right) - \frac{rS^*}{S_{\max}} - (1-\varepsilon)\mu V^* & -\frac{rS^*}{S_{\max}} & -(1-\varepsilon)\mu S^* \\ (1-\varepsilon)\mu V^* & -\beta & (1-\varepsilon)KS_0^* \\ 0 & \beta & -\gamma \end{bmatrix}$$

$$\Rightarrow J(W^*) = \begin{bmatrix} -\bar{a} & -\frac{rS^*}{S_{\max}} & -(1-\varepsilon)\mu S^* \\ (1-\varepsilon)\mu V^* & -\beta & (1-\varepsilon)\mu S_0^* \\ 0 & \beta & -\gamma \end{bmatrix}$$

where  $\bar{a} = \alpha - r \left(1 - \frac{S^* + I^*}{S_{\max}}\right) + \frac{rS^*}{S_{\max}} + (1-\varepsilon)\mu V^* > 0$ .

Now the second additive compound matrix  $J^{[2]}(W^*)$  is

$$J^{[2]}(W^*) = \begin{bmatrix} -(\bar{a} + \beta) & (1-\varepsilon)\mu S^* & (1-\varepsilon)\mu S^* \\ \beta & -(\bar{a} + \gamma) & \frac{rS^*}{S_{\max}} \\ 0 & (1-\varepsilon)\mu V^* & -(\beta + \gamma) \end{bmatrix}. \tag{6}$$

Now we compute  $\text{tr}(J(W^*)), \det(J(W^*)), \det(J^{[2]}(W^*))$  respectively. Hence

$$\text{tr}(J(W^*)) = -\bar{a} - \beta - \gamma < 0,$$

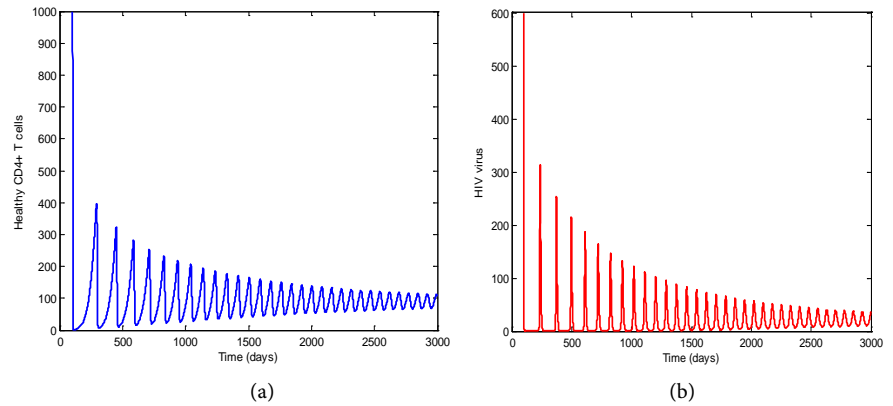
$$\begin{aligned} \det(J(W^*)) &= -\bar{a}[\beta\gamma - (1-\varepsilon)\beta\mu S^*] + \frac{rS^*}{S_{\max}}(1-\varepsilon)\mu V^*\gamma - (1-\varepsilon)\mu S^*(1-\varepsilon)\beta\mu V^* \\ &= -\bar{a}[\beta\gamma - (1-\varepsilon)\beta\mu S^*] + \frac{rS^*}{S_{\max}}(1-\varepsilon)\mu V^*\gamma - (1-\varepsilon)^2\mu^3 S^*\beta V^* < 0, \end{aligned}$$

$$\begin{aligned} \det(J^{[2]}(W^*)) &= -(\bar{a} + \beta) \left[ (\bar{a} + \gamma)(\beta + \gamma) - \frac{rS^*}{S_{\max}}(1-\varepsilon)\mu V^* \right] \\ &\quad - (1-\varepsilon)\mu S^*[-\beta(\beta + \gamma)] + (1-\varepsilon)\mu S^*[\beta(1-\varepsilon)\mu V^*] \\ &= -(\bar{a} + \beta)(\bar{a} + \gamma)(\beta + \gamma) + \frac{rS^*}{S_{\max}}(1-\varepsilon)(\bar{a} + \beta)\mu V^* \\ &\quad + (1-\varepsilon)\mu S^*\beta[(\beta + \gamma) + (1-\varepsilon)\mu V^*] < 0. \end{aligned}$$

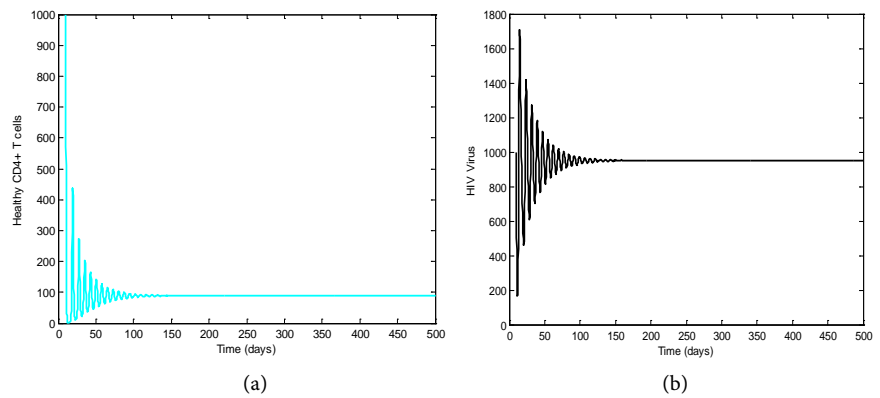
Hence by Lemma 2,  $W^*$  is locally asymptotically stable.

### 5. Numerical Simulations

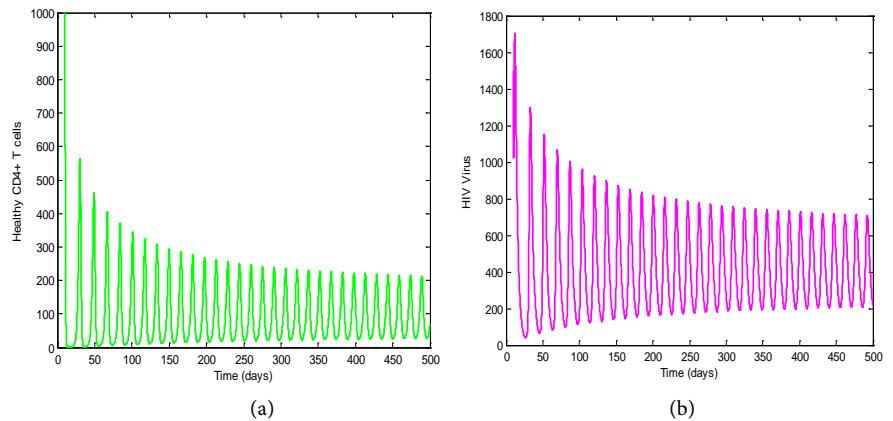
We have discussed the locally asymptotically stability of both infection free equilibrium  $W_0$  and chronic infection equilibrium  $W^*$  above. When  $R_0 > 1$ , the endemic equilibrium  $W^*$  may only be stable for  $r$  small or large. Our numerical solutions consistently show the existence of periodic solutions when  $W^*$  is unstable. For the numerical result we use the parametric values used in **Table 1** taken from [15] and [16] but with the variation of  $r$ . Considering  $S_{\max} = 1500 \text{ mm}^{-3}$ , we have shown local stability of both the healthy  $CD^{4+}$  T cells and HIV virus at  $r = 0.05$  and  $r = 3$  (see in **Figure 3** and **Figure 4**). Whereas at  $r = 0.8$ ,  $W^*$  is unstable and a periodic solution exists (see in **Figure 5**).



**Figure 3.** Using the parameter values of Table 1,  $W^*$  is stable, when  $r = 0.05$  and  $R_0 = 0.5081$ .



**Figure 4.**  $W^*$  is stable, when  $r = 3$  and  $R_0 = 0.48431$ .



**Figure 5.** When  $r = 0.8$  and  $R_0 = 0.8227$ , a periodic solution is observed.

We observe  $W^*$  is unstable within the range of  $r$  between 0.093453 and 1.9118. From Figure 4, we observe viral load  $600 \text{ mm}^{-3}$  persists when  $r = 3$  while it is below  $100 \text{ mm}^{-3}$  at  $r = 0.05$ . Again when  $r = 3$  the initial oscillation disappears after 145+ days whereas at  $r = 0.05$  the damped oscillation are clearly visible after 2000 days. We also note that, the values of  $R_0$  in these three cases are 0.5081, 0.8227 and 0.48431 respectively.

## 6. Conclusion

Bangladesh government and several NGO's have played a magnificent role in keeping the HIV prevalence low by enhancing awareness to people. But this low prevalence rate is increasing day by day and becoming a great threat to us. In this paper, we have shown a brief report of HIV/AIDS of Bangladesh from 1989 to 2014 (except 2008). Again we have discussed the mathematical presentation of HIV infection in a three-compartmental model. In the model, we added a probability term  $\varepsilon$  with the infected T cells. Then we have calculated the basic reproduction number  $R_0 = \frac{(1-\varepsilon)\mu S_0}{\gamma}$ , where  $S_0$  is considered as equilibrium of  $CD^{4+}$  T cells in the absence of HIV infection. At disease free equilibrium point, the model is assumed to be stable and later we conclude the stable and unstable condition for the chronic infected equilibrium points. With the proliferation term  $r$  and reproduction number, we find the solution of it. We find the numerical solution at different equilibrium points and have observed the curve in periodic and damped oscillation.

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