NUMERICAL SOLUTION WITH ANALYSIS OF HIV/AIDS DYNAMICS MODEL WITH EFFECT OF FUSION AND CURE RATE

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Abstract. The main objective of this manuscript is to study the dynamical behaviour and numerical solution of a HIV/AIDS dynamics model with fusion effect and cure rate. Local and global asymptotic stability of the model is established by Routh-Hurwitz criterion and Lyapunov functional method for infection-free equilibrium point. The numerical solutions of the model has also examined for support of analysis, through Mathematica software.

1. Introduction. As we know, AIDS is one of the unsafe transmittable diseases which repress the immune-system of any body, and till now it is not yet fully curable. In the current scenario, HIV/AIDS has turn into a major problem for human community. Literature of the infectious disease in biology indicates that HIV (human immunodeficiency virus) virus has attacked to uninfected CD4+T cells. First phase of HIV disease has classified by a sturdy viral response, and it is quickly followed by a prevailing immune repository system. And the subsequent stage of HIV disease has continual viral replications (exhibits no symptoms about infected individual), which ultimately responsible for development of AIDS [1, 5, 8].

Mojaver and Kheiri have proposed one cell and free virus model; another one, model for cell-to-cell transmission a class of HIV infection. They show that these models in [10] possess non-negative solutions; and also describe the asymptotic stability of equilibrium points with the adequate conditions. In 2016, Huo and friends have developed a new epidemic model [6]. They have introduced a new section of the population, that is, treatment compartment, and discuss the local and global stability of proposed model. Recently, Liu and Chen said that nanotechnology play an important role in HIV/AIDS vaccination [7]. With this motivation, HIV model
can play the important role for control the disease, which destroys the body’s ability to fight against infections.

The work of this manuscript is presented as follows. In the next section, the authors proposed a mathematical model for HIV infection and described the basic properties of the model. After that, Section 3 is committed to discuss the stability analysis, i.e., local and global stability for infection-free equilibrium state. The section 4. Numerical simulations has verified the analytical results and confirms the dynamical behaviour of proposed model. Finally, last section of this manuscript includes the Concluding observations.

2. The model. In this manuscript, we have considered three populations: uninfected population, infected population and the virus population as $T(t), I(t)$ and $V(t)$, respectively. Here, we modified the previous model of [12] with the help of [11] and [4]. Here, we incorporated two assumptions; the first one is a few portion of infected CD$4^+$ T cells be converted into to the uninfected class due to natural recovery of CD$4^+$ T cell. Another one is that a few portion of interaction of the uninfected CD$4^+$ T cells and virus cells will loss both healthy cells and virus cells due to fusion effect during infection. After that, the following model is proposed

$$\begin{align*}
\frac{dT}{dt} &= r - \rho_1 VT - \rho_2 VT - \sigma_1 T + \rho_3 I, \\
\frac{dI}{dt} &= \rho_2 VT - \rho_3 I, \\
\frac{dV}{dt} &= A\sigma_2 I - \rho_1 VT - \sigma_3 V,
\end{align*}$$

with initial conditions,

$$T(0) = T_0 > 0, \quad I(0) = I_0 \geq 0, \quad V(0) = V_0 \geq 0. \quad (2)$$

2.1. Positive invariance and boundedness. (see [3])

Theorem 2.1. The analytical solutions of a model (1), i.e., $T(t), I(t)$ and $V(t)$ including initial population $T(0)$ is positive, $I(0)$ and $V(0)$ are non-negative; are always non-negative for all $t > 0$.

Theorem 2.2. The possible domain $\phi$ classify by:

$$\phi = \{(T(t), I(t), V(t)) \in \mathbb{R}_+^3 : 0 \leq T + I \leq \frac{r}{\sigma_1}, V \leq \Lambda\} \quad (3)$$

for a number of $\Lambda \geq 0$, and it is absolutely invariant with respect to model (1).

2.2. Basic reproduction number and equilibria. By a simple calculation, the model (1) always holds one infection-free equilibrium state

$$E_0 = (T_0^*, I_0^*, V_0^*) = \left(\frac{r}{\sigma_1}, 0, 0\right) \quad (4)$$

Assume $X = (I, V, T)$, the model (1) can be rewrite as

$$\frac{dX}{dt} = P(X) - Q(X),$$

(5)
Table 1. List of parameters

| Parameters | Explanations |
|------------|--------------|
| \( r \)     | Natural production rate of uninfected CD4+ T cells |
| \( \rho_1 \) | Fusion rate of CD4+ T-cells and virus |
| \( \rho_2 \) | Rate of new infection into the infective compartment |
| \( \rho_3 \) | Recovery rate of infected cells |
| \( \sigma_1 \) | Normal death rate of uninfected CD4+ T cells |
| \( \sigma_2 \) | Lytic death rate of infected cells |
| \( \sigma_3 \) | Loss rate of virus |
| \( A \)     | Average number of viral particles produced by an infected CD4+ T-cell |

where, \( P(X) \) is the rate of new infections appear in system (5), and \( Q(X) \) is the shifting rate of populations in another compartment and

\[
P(X) = \begin{bmatrix} \rho_2 VT & 0 \\ 0 & 0 \end{bmatrix}, \quad Q(X) = \begin{bmatrix} \rho_3 I \\ \rho_1 VT + \sigma_3 V - A\sigma_2 I \\ \rho_1 VT + \rho_2 VT + \sigma_1 T - \rho_3 I - r \end{bmatrix}.
\]  

(6)

Therefore, Jacobian of \( P(X), Q(X) \) at \( E_0 \) are defined as,

\[
P(X) = \begin{bmatrix} p_{2 \times 2} & 0_{2 \times 1} \\ 0_{1 \times 2} & 0 \end{bmatrix}, \quad Q(X) = \begin{bmatrix} q_{2 \times 2} & 0_{2 \times 1} \\ \rho_3 (\rho_1 + \rho_2) \frac{r}{\sigma_1} & \sigma_1 \end{bmatrix},
\]  

(7)

where,

\[
p_{2 \times 2} = \begin{bmatrix} 0 & \rho_2 \frac{r}{\sigma_1} \\ 0 & 0 \end{bmatrix}, \quad q_{2 \times 2} = \begin{bmatrix} \rho_3 & 0 \\ -A\sigma_2 & \rho_1 \frac{r}{\sigma_1} + \sigma_3 \end{bmatrix}.
\]  

(8)

By the next generation matrix method [13], the maximal radius of square matrix \( pq^{-1} \) is defined as

\[
R_0 = \rho(pq^{-1}) = \frac{Ar\sigma_2 \rho_2}{r\rho_1 \rho_3 + \sigma_1 \sigma_3 \rho_3}.
\]

3. Analysis of the model. The classification of the local and global stability of the infection-free equilibrium has determined in the following subsections.

3.1. Local stability of infection-free steady state.

Theorem 3.1. The infection-free equilibrium point \( E_0 \) is locally asymptotically stable in the region \( \phi \), if \( R_0 < 1 \), and it is unstable for \( R_0 \geq 1 \).

Proof. The Jacobian matrix for the system (1) at non-infected equilibrium point \( E_0 \), is derived as

\[
J|_{E_0} = \begin{bmatrix} -\sigma_1 & \rho_3 & -\rho_1 \frac{r}{\sigma_1} - \rho_2 \frac{r}{\sigma_1} \\ 0 & -\rho_3 & \rho_2 \frac{r}{\sigma_1} \\ 0 & A\sigma_2 & -\rho_1 \frac{r}{\sigma_1} - \sigma_3 \end{bmatrix}
\]  

(9)

The eigenvalue equation for the matrix (9) is

\[
(\lambda + \sigma_1)(\lambda^2 + a_1 \lambda + a_2) = 0
\]  

(10)

From above equation (10), first value of \( \lambda \) is defined by \( -\sigma_1 \). And the remaining two values of \( \lambda \) are calculated by the following quadratic equation

\[
(\lambda^2 + a_1 \lambda + a_2) = 0,
\]
where
\[ a_1 = \rho_3 + \rho_1 \frac{r}{\sigma_1} + \sigma_3, \quad a_2 = \frac{1}{\sigma_1} (r\rho_1\rho_3 + \rho_3\sigma_1\sigma_2 - A\rho_2\sigma_2) \]  
(11)

Now, if \( R_0 < 1 \), then \( a_1 > 0 \) and \( a_2 > 0 \). According to Routh-Hurwitz criterion, we can say that if \( R_0 < 1 \) the non-infected equilibrium point \( E_0 \) is locally asymptotically stable. And, if \( R_0 > 1 \), the eigenvalue equation (10) has given at least one positive eigenvalues, therefore, \( E_0 \) is unstable. \( \square \)

3.2. **Global stability of infection-free steady state.** In this portion of the manuscript, we discuss the global stability of \( E_0 \) by Lyapunov function method (see [2, 9]).

**Theorem 3.2.** If \( R_0 \leq 1 \), then the infection-free equilibrium point \( E_0 \) is globally asymptotically stable on \( \phi \). If \( R_0 > 1 \), then \( E_0 \) is unstable.

**Proof.** Consider the Lyapunov candidate function for the model (1) is
\[ L = \frac{A\sigma_2}{\rho_3} I + V. \]  
(12)

Now, differentiate the equation (12) with respect to \( t \), we get
\[ \frac{dL}{dt} = \frac{A\sigma_2}{\rho_3} \frac{dI}{dt} + \frac{dV}{dt}. \]  
(13)

With the help of equation (1), the equation (13) is
\[ \frac{dL}{dt} = \frac{A\sigma_2}{\rho_3} (\rho_2 VT - \rho_3 I) + (A\sigma_2 I - \rho_1 VT - \sigma_3 V). \]

or
\[ \frac{dL}{dt} = (R_0 - 1)(\rho_1 T + \sigma_3) V. \]  
(14)

From equation (14), it is easy to observe that if \( R_0 \leq 1 \), then \( \frac{dL}{dt} \leq 0 \). Therefore, inside the region \( \phi \) all solution paths tends to the non-infected equilibrium point \( E_0 \).

We can straightforwardly apparent from matrix (9) for the model (1) at non-infected equilibrium point that at least a root of eigenvalue equation (10) is positive if reproduction number is greater than one. As a result, the non-infected equilibrium point \( E_0 \) is unstable. \( \square \)

4. **Numerical Simulations.** The values of all the parameters in vivo circumstances are difficult to find; so, all the parameter values in the proposed model (1) has not measured by the authors, and we are keeping these parameter values from ref. [12]. In this portion of the manuscript, we simulate the system of equations (1) using MATHEMATICA with the specified parameter values \( \rho_1 = 0.000002 \text{mm}^3\text{day}^{-1} \), \( \rho_2 = 0.000004 \text{mm}^3\text{day}^{-1} \), \( \rho_3 = 0.2 \text{day}^{-1} \), \( \sigma_1 = 0.01 \text{day}^{-1} \), \( \sigma_2 = 0.16 \text{day}^{-1} \), \( \sigma_3 = 3.4 \text{day}^{-1} \), \( A = 1000 \), \( r = 10 \text{mm}^{-3}\text{day}^{-1} \). The numerical results are demonstrated in Figs. 1-2 for initial values \( S = (1000, 0, 0.001) \).

For infection-free state, Fig. 1(a) demonstrates that the uninfected CD4\(^+\) T cell population will remain constant with increase of time. Fig. 1(b) verify that infected CD4\(^+\) T cell population is rapidly increases in the first few days, after that it decreases exponentially with increase of time and it tends to zero. Similarly, Fig. 1(c) exhibit that first few days the virus population is decreases drastically after that it tends to zero.
For infection state (endemic), Fig. 2(a) exhibits that first few days the uninfected CD4$^+$ T cell population remain constant after that it decreases drastically and tends to endemic equilibrium point with increase of time. Fig. 2(b) verify that initial days the infected T cell population remain constant after that it increases rapidly and then decay drastically and tends to endemic equilibrium point with increase of
Figure 2. Dynamical behaviour of the model (1) for $R_0 = 5.6140 > 1$.

Time. Similarly, Fig. 2(c) exhibits that first few days the virus population remain constant after that it increases rapidly and then decay drastically and tends to endemic equilibrium point with increase of time.
Conclusion. In this manuscript, we derive a complete mathematical analysis of a proposed model which illustrates the novel dynamical behaviour during HIV/AIDS infection. The essential criteria e.g., non-negativity and boundedness of the solutions has been carried out in Theorem 1 and Theorem 2, and define the constraint for obtained solutions. In the context of HIV infection, the basic reproduction is the average number of infected cells, which is produced by one virus. The $R_0$ is analytically calculated and we have proven that if it is less than one ($R_0 = 0.9406 < 1$), then the infection-free equilibrium point is globally stable and otherwise, if it is more than one, the infection-free equilibrium point becomes unstable and this case one more steady state is found, which is endemic equilibrium point and it is globally asymptotically stable if $R_0 = 5.6140 > 1$.

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