Results in Physics 22 (2021) 103851

Contents lists available at ScienceDirect

Results in Physics

journal homepage: www.elsevier.com/locate/rinp

# Numerical and bifurcation analysis of spatio-temporal delay epidemic model

Muhammad Jawaz [a], Muhammad Aziz ur Rehman [a], Nauman Ahmed [b,*], Dumitru Baleanu [c,d,e,*], Muhammad Rafiq [f]

[a] Department of Mathematics, University of Management and Technology, Lahore, Pakistan
[b] Department of Mathematics and Statistics, The University of Lahore, Lahore, Pakistan
[c] Department of Mathematics, Çankaya University, 06530, Balgat, Ankara, Turkey
[d] Institute of Space Sciences, Magurele-Bucharest, Romania
[e] Department of Medical Research, China Medical University Hospital, China Medical University, Taichung, Taiwan
[f] Department of Mathematics, Faculty of Sciences, University of Central Punjab, Lahore, Pakistan

ARTICLE INFO

*Keywords:*
HIV infection
Reaction-diffusion
Time delay
Structure preserving

ABSTRACT

HIV/AIDS is a distressing and incurable disease of the human beings. In this article, we have proposed a numerical structure for the HIV/AIDS compartmental model with diffusion and delay process. The proposed scheme has the proficiency to preserve the positivity of the state variables. Also, the proposed scheme leads to the consistency and stability. Two equilibrium states of the model have been described. Moreover, the stability of the scheme is examined at these two states. The contribution of the basic reproductive number $R_0$, in stability analysis is also investigated. The bifurcation value of the infection parameter $\gamma$, for different situations of $\tau$ is also investigated. Graphical solutions with the aid of computer simulations are presented to clarify the paramount features of the proposed numerical design.

## Introduction

Prevalence of Human Immunodeficiency Virus (HIV) infection is a tremendous challenge to the public health authorities, both in the developed and developing countries [1–3]. According to a report by UNAIDS (Joint Program of United Nations for AIDS), till the end of 2018, globally, there were 37.9 million people living with HIV and each year about 1 million people die due to HIV related illnesses. UNAIDS has set the goal to eliminate the disease from the world by 2030. HIV is a retrovirus that destroys the CD4 cells, the cells of human immune system (i.e. T helper cells, monocytes, macrophages, and dendritic cells) [4,5],if left untreated, HIV continues killing more CD4 cells over time and the HIV infection may attain its most serious stage in its host, known as Acquired Immunodeficiency Syndrome (AIDS). It takes 2 to 10 years to reach at the final stage [6]. At this point, the immune system of the body becomes too week to fight against the infectious diseases including, tuberculosis [7,8], cryptococcal meningitis (i.e. a fungal infection in the brain) [9,10], cryptosporidiosis [11] (i.e. an intestinal infection caused by a parasite) and some cancers. Generally, the HIV spreads through sexual transmission, blood diffusion and perinatal diffusion (e.g. mother feed). The symptoms of HIV infection may include fever, sore throat, chills, muscle aches, red rashes on the body, night sweats, swollen lymph nodes, sores of mouth, broadened organs or weight loss. Mostly, the HIV victims do not reflect the clear symptoms of the attack. As, the symptoms of the infection may be similar to other illnesses, so the only way to confirm the HIV status is the lab testing. Globally, 8.1 million HIV infected persons do not know their HIV status. The factors like social stigma, discrimination and expensive health services prevent people from HIV testing [12]. Two distinctive types of HIV are characterized as HIV-1 and HIV-2. However, within these main types of HIV, many genetically distinct subgroups also exist. Moreover, HIV-1 is found to be more virulent than HIV-2, which is mostly restricted to West Africa. Currently, no vaccine is available to cure the HIV due to the ability of its mutation and change within the infected persons. However, Anti-Retroviral Therapy (ART) can suppress the HIV load in the patient and onward spread of the disease. Life expectancy of HIV patients varies, depending upon the subtype and stage of the infection. Average survival time of an HIV infected person without treatment is 9 to 11 years, however proper treatment with ART can improve the life expectancy of the patient more than 10 years after the initiation of the AIDS [6].

* Corresponding authors at: Department of Mathematics, Çankaya University, 06530, Balgat, Ankara, Turkey (D. Baleanu).
*E-mail addresses:* nauman.ahmed@math.uol.edu.pk (N. Ahmed), dumitru@cankaya.edu.tr (D. Baleanu).

https://doi.org/10.1016/j.rinp.2021.103851
Received 17 December 2020; Received in revised form 11 January 2021; Accepted 12 January 2021
Available online 26 January 2021

According to the UNAID, 2030 will be the final year for the elimination of the AIDS from the planet and by the end of year 2020, 90% persons infected with HIV will know their HIV status. In Pakistan, the first case of HIV was reported in 1987 and by the end of 2018 the number of HIV infected persons have been reported as 0.165 million [13]. Pakistan is situated in between the HIV high risk countries like India and China [14]. Although, HIV adult prevalence rate among general public (15–45 yr) is very low in Pakistan i.e. less then 0.1% [13], but the recent publications are the major cause of concern. For example, [15] has reported an increase of 54% in pediatric HIV cases over the past 13 years and, according to UNAIDS report of (2018), there has been 369% increase in deaths due to AIDS related illnesses in Pakistan. It has also been reported by UNAIDS that 86% of HIV infected persons in Pakistan do not know about their status, and only 10% of those are aware with their HIV status. The factors conducive to the spread of HIV infection in Pakistan includes sharing of needles among injection drug users (IDUs), low condom use rate, unscreened blood transfusion, lack of facilities to screen the HIV reported persons. The multisectoral and coordinated actions are required by the government officials to eradicate the HIV infection from the country, which may include the free access to HIV testing facilities, availability of ART for the HIV diagnosed persons and strict compliance on screening before blood transfusion and use of sterilized instruments in hospitals and at barber shops. It is the need of the hour to raise the awareness level in general public.

Many infectious diseases spread by free interaction of the individuals in the population. So, isolation or quarantine is advised by the medical staff, for the infected person. This important factor is not envisaged in many infectious disease models. Due to this point the simple mathematical models were not fit for the prediction of the disease dynamics. Keeping in view this missing factor of the simple HIV/AIDS epidemic models, we have modified the model by including the diffusion process as well as the delay factor in the continuous system. Now, the modified model with be able to forecast the infection, more accurately.

## Mathematical model

HIV/AIDS is a virus which is still incurable. Although, a number of researchers are working to find the cure of such a dreadful disease. But, still there is nothing perfect, which can play a role to cure this virus properly. In such conditions mathematical models are very helpful to describe the nature of the disease and the spread of virus. Here, we are using SIR three compartmental model to analyze the HIV/AIDS system. Li and Ma investigated the key features of HIV-1 disease model by considering the delay factor [16]. The considered model was developed by two mathematicians Abdullahi and Nweze [17]. According to this model the following state variables and parameters are used. $S$ is used for the fraction of susceptible populace. $I$ is the fraction of infected populace and the value $R$ represents the fraction of recovered or removed populace. The parameter $\Lambda$ represents the population recruiting flow, $\delta$ denotes the casuality rate, $\gamma$ is the rate of becoming infected from the susceptible compartment, $\kappa$ is the rate of becoming recovered after getting infection, $\delta_0$ represents the casuality rate of the contaminated individuals and $\delta_1$ shows the causality rate of the cured individuals. The values $N$ is the complete population and $N(t) = S(t) + I(t) + R(t)$.

$$\frac{dS}{dt} = \Lambda N - \delta S - \gamma SI, \tag{2.1}$$

$$\frac{dI}{dt} = \gamma S(t-\tau)I(t-\tau)\mathrm{e}^{-\delta\tau} - (\kappa+\delta+\delta_0)I, \tag{2.2}$$

$$\frac{dR}{dt} = \kappa I - (\delta+\delta_1)R. \tag{2.3}$$

In this model, $\tau$is positive and finite [22,23], which represents the incubation period in which an infected individual will become infectious. The incidence rate $\gamma S(t-\tau)I(t-\tau)\mathrm{e}^{-\delta\tau}$ which is in the Eq. (2.2) of the model symbolizes the flow rate of susceptible people by which they are emitting the compartment at time $t-\tau$and entering into the contagious compartment at time $t$. So, the term $\mathrm{e}^{-\delta\tau}$sticks to the hypothesis that the casuality of all the persons in the model follow a linear relation described by the factor $\mathrm{e}^{-\delta\tau}$. This three compartment (SIR) model map out an HIV/AIDS, which consists of three non linear ordinary differential equations with delay factor. The assumptions are made by taking into account the interaction of the individuals in the population. As, the infectious disease can be spread with time as well as space, so we can take into account the diffusion factor. Thus, we can transform the above ODE model into:

$$\frac{\partial S}{\partial t} = d_1\frac{\partial^2 S}{\partial x^2} + \Lambda N - \delta S - \gamma SI, \tag{2.4}$$

$$\frac{\partial I}{\partial t} = d_2\frac{\partial^2 I}{\partial x^2} + \gamma S(x,t-\tau)I(x,t-\tau)e^{-\delta\tau} - (\kappa+\delta+\delta_0)I, \tag{2.5}$$

$$\frac{\partial R}{\partial t} = d_3\frac{\partial^2 R}{\partial x^2} + \kappa I - (\delta+\delta_1)R. \tag{2.6}$$

In the above system of equations the 2nd order partial derivatives with respect to $x$depict the spread in space. The very important factor $\gamma S(t-\tau)I(t-\tau)\mathrm{e}^{-\delta\tau}$ defines the rate of disease occurrence at the moment $(t-\tau)$. It is calculated by applying the principle of mass action. As the first two equations of the proposed model are independent of $R$. So the rest two equations can be considered for the purpose.

$$\frac{\partial S}{\partial t} = d_1\frac{\partial^2 S}{\partial x^2} + \Lambda N - \delta S - \gamma SI, \tag{2.7}$$

$$\frac{\partial I}{\partial t} = d_2\frac{\partial^2 I}{\partial x^2} + \gamma S(x,t-\tau)I(x,t-\tau)e^{-\delta\tau} - (\kappa+\delta+\delta_0)I. \tag{2.8}$$

By considering the initial data as,

$S(x,0) = f_1(x)$ and $I(x,0) = f_2(x)$, along with homogenous Neumann boundary constraints. Here, the homogenous Neumann boundary conditions describe the no flux boundary conditions.

The delay factor is one of the most significant feature of the infectious disease models. Many epidemics compartmental models take into account the delay feature. To study the role of $\tau$(delay factor) in disease dynamics, the following articles are recommended for studying [22–28].

### *Equilibria of the system*

This section is devoted to find the equilibrium points and reproductive number $R_0$. For equilibrium points put $\frac{dS}{dt} = \frac{dI}{dt} = 0$ in the Eqs. (2.1) and (2.2).

$$0 = \Lambda N - \delta S - \gamma SI, \tag{2.9}$$

$$0 = \gamma S(t-\tau)I(t-\tau)\mathrm{e}^{-\delta\tau} - (\kappa+\delta+\delta_0)I. \tag{2.10}$$

Note that we are not considering the Eq. (2.3), as $R$ is not the part of Eqs. (2.1) and (2.2) and $N = S + I + R$. In epidemic models there are two types of steady states namely disease free equilibrium (DFE) and endemic equilibrium (EE). The DFE point of this HIV/AIDS model is obtained by substituting $I = 0$ in equations (2.9) and (2.10), which is

$$\epsilon^0(S^0, I^0) = \epsilon^0\left(\frac{\Lambda N}{\delta}, 0\right).$$

For the calculation of $R_0$, we have $\frac{dI}{dt} > 0$, this implies that $\gamma SI\mathrm{e}^{-\delta\tau} - (\kappa + \delta + \delta_0)I > 0$, since $I > 0$, therefore $\gamma S\mathrm{e}^{-\delta\tau} > (\kappa + \delta + \delta_0)$,

$\Rightarrow \quad \frac{\gamma S\mathrm{e}^{-\delta\tau}}{(\kappa+\delta+\delta_0)} > 1.$

So the value of reproductive number $R_0 = \frac{\gamma\Lambda N\mathrm{e}^{-\delta\tau}}{\delta(\kappa+\delta+\delta_0)}$ when $d_1 = d_2 = d_3 = 0$. For the endemic equilibrium point, we have from Eqs. (2.1) and (2.2)

$\gamma SIe^{-\delta\tau} = (\kappa+\delta+\delta_0)I.$

This implies that

$$S^* = \frac{(\kappa+\delta+\delta_0)}{\gamma e^{-\delta\tau}} = \frac{\Lambda N}{\delta R_0}.$$

Now From (2.9), we have

$$\Lambda N - \delta S = \gamma SI, \quad \Rightarrow \quad I^* = \frac{\Lambda N - \delta S^*}{\gamma S^*}.$$

By substituting the value of $S^*$ in above and after some simplification we have,

$$I^* = \frac{\delta}{\gamma}(R_0 - 1).$$

Note that the value $R_0$is a reproductive number which decides whether the disease will go to an end or will persist in the society. If $R_0 < 1$the disease will die out and if $R_0 > 1$ then the disease persists in the population.

## Numerical analysis of the model

In mathematical modeling, when we study a physical phenomenon then we obtain a system of differential equations. The dynamical system with diffusion and delay becomes complicated and its analytical solution is a demanding task. In some cases the exact solution can not be found. In that case, a numerical design is required to find the numerical solutions. So, to study the behavior of the model we prefer to use the numerical techniques. Although, these techniques do not give us the exact or analytical solution of the model, but such schemes help us to study the true behavior of the model. There are many well known numerical schemes used to find the numerical solution of the model. But, in epidemiological models, structure preserving numerical technique is required that must possess some meaningful properties like positivity, consistency and boundedness of the population. In this regard, we develop the non standard finite difference schemes which will surely help us to study the physical behavior of the epidemiological models.

### *Proposed implicit scheme*

There are many numerical schemes used to find the numerical solutions of the systems of differential equations, but here we are using a non standard finite difference scheme which is very helpful to study the true behavior of the model. The nonstandard finite difference scheme (NSFD) was initially established by R.E Micken in 1989. This scheme provides the positivity and boundedness of the model that are the essential properties of the state variables. The finite difference methods are easy for approximating the solutions of the systems of linear and nonlinear systems of partial differential equations [18–20]. In these techniques, we convert the continuous model into a discrete formulation by the number of function values at selected finite number of points in the domain which is easy to handle. The Taylor's series is the best way to obtain these approximations. Now, let $M$ and $N$ be any two finite positive integers and $\tau$ be any other positive real number. The spatial interval $[a, b]$ over the time period $[0, \tau]$ are discretized according to the partitions $a = x_0 < x_1 < x_2 < \cdots < x_M = b$ and $0 = t_0 < t_1 < t_2 < \cdots < t_N = T$ respectively, with the norm $h = \frac{b-a}{M}$ and $k = \frac{T}{N}$. Divide $[a, b] \times [0, T]$ into $M \times N$ grid points with space and time step sizes $h$ and $k$ respectively. The points of the partitions now become as $x_j = jh$ and $t_m = mk$, where $i \in 0, 1, 2, \cdots, M$ and $m = 0, 1, 2, \cdots, N$. Suppose that, $S_j^m, I_j^m and R_j^m$ denotes the approximations of $S(x,t), I(x,t), and R(x,t)$ respectively at the grid point $(jh, mk)$. In this article, we use a non-standard finite difference implicit scheme [31] carrying some important physical properties for the discrete model, developed in [21]. Discrete model equations form a matrix or iterative process that are used to find the best approximation of the solution to the system (2.7)-(2.8). This system can be converted in discrete form by using the following approximations.

$$\frac{\partial H}{\partial t} = \frac{H_j^{m+1} - H_j^m}{\Delta t},$$

$$\frac{\partial^2 H}{\partial x^2} = \frac{H_{j-1}^{m+1} - 2H_j^{m+1} + H_{j+1}^{m+1}}{(\Delta x)^2}.$$

Now, the discretization of the compartment $S$ in the model is:

$$\frac{S_j^{m+1} - S_j^m}{\Delta t} = d_1 \frac{S_{j-1}^{m+1} - 2S_j^{m+1} + S_{j+1}^{m+1}}{(\Delta x)^2} + \Lambda N - \delta S_j^{m+1} I_j^m - \gamma S_j^{m+1}.$$

$$S_j^{m+1} - S_j^m = \Delta t d_1 \frac{S_{j-1}^{m+1} - 2S_j^{m+1} + S_{j+1}^{m+1}}{(\Delta x)^2} + \Delta t \Lambda N - \Delta t \gamma S_j^{m+1} I_j^m - \Delta t \delta S_j^{m+1}.$$

$$-\lambda_1\left(S_{j-1}^{m+1} + S_{j+1}^{m+1}\right) + S_j^{m+1}(1 + 2\lambda_1 + \Delta t \gamma I_j^m + \Delta t \delta) = S_j^m + \Delta t \Lambda N. \tag{3.1}$$

The similar design is used for the compartment $I$,

$$\frac{I_j^{m+1} - I_j^m}{\Delta t} = d_2 \frac{I_{j-1}^{m+1} - 2I_j^{m+1} + I_{j+1}^{m+1}}{(\Delta x)^2} + \gamma S_j^{m-k} I_j^{m-k} e^{-\delta\tau} - (\kappa + \delta + \delta_0) I_j^{m+1}.$$

$$-\lambda_2\left(I_{j-1}^{m+1} + I_{j+1}^{m+1}\right) + I_j^{m+1}(1 + 2\lambda_2 + \Delta t(\kappa + \delta + \delta_0)) = I_j^m + \Delta t \gamma S_j^{m-k} I_j^{m-k} e^{-\delta\tau}. \tag{3.2}$$

and

$\lambda_1 = d_1 \frac{\Delta t}{\Delta x^2}, \lambda_1 = d_2 \frac{\Delta t}{\Delta x^2}.$

## Properties of the proposed numerical scheme

In this portion, some key properties of the system will be investigated.

### *Stability*

The main concern in the study of approximating the solutions to the system of differential equations is the growth of round off errors in the numerical solutions. Another main thing to observe is that a small change in the initial conditions may cause a large deviation in the solution to the underlying system. In this scenario, if the slight change in the initial data does not produce a huge variation in the approximate and exact solutions. We say the numerical scheme which gives such approximate solutions, is stable. To discuss the stability analysis of the proposed scheme, we use the method of Von-Neumann [29–33]. The Von-Neumaan criteria ensures the condition to check the stability of the numerical scheme. Here, the NSFD scheme is developed for the HIV/AIDS model. For this purpose, we decompose the numerical error occurred in the numerical solutions into the Fourier series. So, linearizing the Eqs. (3.1) and (3.2) and substitute.

$$\begin{aligned} S_j^m &= \xi_s^m e^{i\omega lh}, \\ S_j^{m+1} &= \xi_s^{m+1} e^{i\omega lh}, \\ S_{j-1}^{m+1} &= \xi_s^{m+1} e^{i\omega(j-1)h}, \\ S_{j+1}^{m+1} &= \xi_s^{m+1} e^{i\omega(l+1)h}. \end{aligned}$$

$$-\lambda_1\left(S_{j-1}^{m+1} + S_{j+1}^{m+1}\right) + S_j^{m+1}(1 + 2\lambda_1 + \Delta t \gamma I_j^m + \Delta t \delta) = S_j^m + \Delta \Lambda N. \tag{4.1}$$

$$-\lambda_1 \xi_s^{m+1}\left(e^{i\omega(l-1)h} + e^{i\omega(l+1)h}\right) + \xi_s^{m+1} e^{i\omega lh}(1 + 2\lambda_1 + \Delta t \delta) = \xi_s^m e^{i\omega lh}. \tag{4.2}$$

$$\xi_s\big(-\lambda_1(e^{-i\omega h}+e^{i\omega h})+1+2\lambda_1+\Delta t\delta\big)=1.$$

$$\xi_s(-2\lambda_1 cos(\omega h)+1+2\lambda_1+\Delta t\delta)=1.$$

$$\xi_s\left(-2\lambda_1+4\lambda_1 sin^2\frac{(\omega h)}{2}+1+2\lambda_1+\Delta t\delta\right)=1.$$

$$\xi_s\left(1+4\lambda_1 sin^2\frac{(\omega h)}{2}+\tau\delta\right)=1.$$

$$|\xi_s|=\left|\frac{1}{1+4sin^2\frac{(\omega h)}{2}+\Delta t\delta)}\right|<1. \tag{4.3}$$

By using similar process in Eq. (2.2), we have taken Eq. (2.2) and put

$$\begin{aligned} I_j^m &= \xi_I^m e^{i\omega lh},\\ I_j^{m+1} &= \xi_I^{m+1} e^{i\omega lh},\\ I_{j-1}^{m+1} &= \xi_I^{m+1} e^{i\omega(l-1)h},\\ I_{j+1}^{m+1} &= \xi_I^{m+1} e^{i\omega(l+1)h}. \end{aligned}$$

$$-\lambda_2\left(I_{j-1}^{m+1}+I_{j+1}^{m+1}\right)+I_j^{m+1}(1+2\lambda_2+\Delta t(\kappa+\delta+\delta\delta_0))=I_j^m+\Delta t\gamma S_j^{m-m}I_j^{m-k}\varrho^{-\Xi\delta\tau}.$$

$$-\lambda_2\xi_I^{m+1}\left(e^{i\omega(l-1)h}+e^{i\omega(l+1)h}\right)+\xi_I^{m+1}e^{i\omega lh}$$
$$(1+2\lambda_2+\Delta t(\kappa+\mu+\delta_0))=\xi_I^m e^{i\omega lh}+\Delta t\gamma\xi_I^{m-k-n}e^{j\omega lh}e^{-\delta\tau}.$$

$$\xi_I\big(-\lambda_2(e^{-i\omega h}+e^{i\omega h})+1+2\lambda_2+\Delta t(\kappa+\delta+\delta_0)\big)=1+\Delta t\xi_I^{-k}e^{-\delta\tau}.$$

$$\xi_I(-2\lambda_2 cos(\omega h)+1+2\lambda\lambda_2+\Delta t(\kappa+\delta+\delta_0))=1+\Delta t\gamma\xi_I^{-k}e^{-\delta\tau}.$$

$$\xi_I\left(-2\lambda_1+4\lambda_2 sin^2\frac{(\omega h)}{2}+1+2\lambda_2+\Delta t(\kappa+\delta+\alpha_0)\right)=1+\Delta t\gamma\xi_I^{-k}e^{-\delta\tau}.$$

$$\xi_I\left(1+4\lambda_2 sin^2\frac{(\omega h)}{2}+\Delta t(\kappa+\delta+\delta_0)\right)=1+\Delta t\gamma\xi_I^{-k}e^{-\delta\tau}.$$

$$|\xi_I|=\left|\frac{1+\Delta t\gamma\xi_I^{-k}e^{-\delta\tau}}{1+4\lambda_2 sin^{2\frac{(\omega h)}{2}}+\Delta t(\kappa+\delta+\delta_0)}\right|<1. \tag{4.4}$$

As $\xi_I^{-k}<1.$

*Positivity*

In this section, we will investigate the positivity property [33] of the numerical scheme with the help of *M*-matrix theory [20]. Positivity is a very important factor in epidemiological models to represent their true behavior. Because, in these models we divide the total population into different sub-populations according to the nature of the infection. So, Each subclass must have non-negative at every time *t*.

**Theorem 4.1.** *For any $h>0$ and $\Delta t>0$, the system (2.1)-(2.2) is positive, i.e. $S^k>0, E^k>0$ and $I^k>0$ for all $k=0,1,2\ldots$*

**Proof.** The system (2.1)-(2.3) can be written as

$$AS^{k+1}=S^k. \tag{4.5}$$

$$BE^{k+1}=E^k. \tag{4.6}$$

Where A and B are square matrices as

$$A=\begin{pmatrix} a_3 & a_1 & 0 & \cdots & \cdots & \cdots & \cdots & 0\\ a_2 & a_3 & a_4 & \ddots & & & & \vdots\\ 0 & a_2 & a_3 & a_4 & \ddots & & & \vdots\\ \vdots & \ddots & \ddots & \ddots & \ddots & \ddots & & \vdots\\ \vdots & & \ddots & \ddots & \ddots & \ddots & \ddots & \vdots\\ \vdots & & & \ddots & a_2 & a_3 & a_4 & 0\\ \vdots & & & & \ddots & a_2 & a_3 & a_4\\ 0 & \cdots & \cdots & \cdots & \cdots & 0 & a_1 & a_3 \end{pmatrix}. \tag{4.7}$$

$$B=\begin{pmatrix} b_3 & b_1 & 0 & \cdots & \cdots & \cdots & \cdots & 0\\ b_2 & b_3 & b_4 & \ddots & & & & \vdots\\ 0 & b_2 & b_3 & b_4 & \ddots & & & \vdots\\ \vdots & \ddots & \ddots & \ddots & \ddots & \ddots & & \vdots\\ \vdots & & \ddots & \ddots & \ddots & \ddots & \ddots & \vdots\\ \vdots & & & \ddots & b_2 & b_3 & b_4 & 0\\ \vdots & & & & \ddots & b_2 & b_3 & b_4\\ 0 & \cdots & \cdots & \cdots & \cdots & 0 & b_1 & b_3 \end{pmatrix}. \tag{4.8}$$

The principle diagonal elements are $a_1=-(2\lambda_1), a_2=-\lambda_1, a_4=-\lambda_1$ and the other elements of the matrices *A*are $a_3=(1+2\lambda_1+\Delta t\gamma I_j^m+\Delta t\delta)$. The off-diagonal entries of B are $b_1=-2\lambda_2$, $b_2=-\lambda_2$, $b_4=-\lambda_2$and diagonal entries are $b_3=(1+2\lambda_2+\Delta t(\kappa+\delta+\delta_0)$. The entry of column matrix of $S^k$is $S_j^m+\Delta tb\Lambda N$, entry of column matrix $E^k$ is $I_j^m+\Delta t\gamma S_j^{m-k}I_j^{m-k}e^{-\delta\tau}$ of the equations (4.1)-(4.2). Thus A, B are M-matrices. So the equations become,

$$S^{k+1}=A^{-1}S^k. \tag{4.9}$$

$$E^{k+1}=B^{-1}E^k. \tag{4.10}$$

If we consider that $S^k>0, E^k>0$ and $I^k>0$, then from the property of M-matrix we get $S^{k+1}>0, E^{k+1}>0$ and $I^{k+1}>0$. So, by the principle of mathematical induction, the theorem is proved. □

*Consistency of the scheme*

Here, we investigate the consistency of the proposed scheme by using Taylor series [29–31,35], which is given as,

$$\begin{aligned} \pounds_S &= \frac{S_j^{m+1}-S_j^m}{\Delta t}-d_1\frac{S_{j-1}^{m+1}-2S_j^{m+1}+S_{j+1}^{m+1}}{(\Delta x)^2}-\Lambda N+\gamma I_j^m S_j^{m+1}+\delta S_j^{m+1}.\\ &= \left(\frac{\partial S}{\partial t}+\frac{\Delta t}{2!}\frac{\partial^2 S}{\partial t^2}+\frac{(\Delta t)^2}{3!}\frac{\partial^3 S}{\partial t^3}+\ldots\right)-\frac{d_1}{(\Delta x)^2}\\ &\left((\Delta x)^2\left(\frac{\partial^2 S}{\partial x^2}+2\frac{(\Delta x)^2}{4!}\frac{\partial^4 S}{\partial x^4}+\ldots.\right)-2\left(\Delta t\frac{\partial S}{\partial t}+\frac{\Delta t}{2!}\frac{\partial^2 S}{\partial t^2}+\frac{\Delta t^2}{3!}\frac{\partial^3 S}{\partial t^3}+\ldots\right)\right)\\ &+(\gamma I_j^m+\delta)\left(S_j^m+\Delta t\frac{\partial S}{\partial t}+\frac{(\Delta t)^2}{2!}\frac{\partial^2 S}{\partial t^2}+\frac{(\Delta t)^3}{3!}\frac{\partial^3 S}{\partial t^3}+\ldots\right)-\Lambda N.\\ &= -\frac{d_1(\Delta x)^2}{12}\left(\frac{\partial^4 S}{\partial x^4}\right)+\Delta t\left(\frac{\partial S}{\partial t}+\frac{\Delta t}{2!}\frac{\partial^2 S}{\partial t^2}+\frac{\tau^2}{3!}\frac{\partial^3 S}{\partial t^3}+\ldots\right)\\ &\left(-2\frac{d_1}{h^2}+kL_i^n+\mu+\ldots.\right). \end{aligned} \tag{4.11}$$

$\to 0$ as $h,\tau\to 0$.

Now take

$$\pounds_I=\frac{I_i^{n+1}-I_i^n}{\tau}-d_2\frac{I_{i-1}^{n+1}-2I_i^{n+1}+I_{i+1}^{n+1}}{h^2}-\beta S_i^{n-m}I_i^{n-m}e^{-\delta\tau}+(k+\delta+\alpha_0)I_i^{n+1}.$$

**Table 2**
Numerical stability of HIV model at $\epsilon^*$ for $\tau = 0$.

| Parameters | $\Xi_1$ | $\Xi_2$ |
|---|---|---|
| case-1 | 0.763633736291161 | 0.378233100392336 |
| case-2 | 1.477919450576875 | 0.895857393965710 |
| case-3 | 2.192205164862589 | 1.413481687539084 |
| case-4 | 2.906490879148303 | 1.931105981112458 |
| case-5 | 3.620776593434018 | 2.448730274685832 |

**Table 3**
Numerical stability of HIV model at $\epsilon^*$ for $\tau = 4$.

| Parameters | $\Xi_1$ | $\Xi_2$ |
|---|---|---|
| case-1 | 0.370297282089176 | 0.093192394452195 |
| case-2 | 0.691246542172906 | 0.325775982085428 |
| case-3 | 1.012195802256636 | 0.558359569718660 |
| case-4 | 1.333145062340366 | 0.790943157351893 |
| case-5 | 1.654094322424095 | 1.023526744985126 |

$$= \left(\frac{\partial I}{\partial t} + \frac{\tau}{2!}\frac{\partial^2 I}{\partial t^2} + \frac{\tau^2}{3!}\frac{\partial^3 I}{\partial t^3} + \ldots\right)$$

$$-\frac{d_1}{h^2}\left(h^2\left(\frac{\partial^2 I}{\partial x^2} + 2\frac{h^2}{4!}\frac{\partial^4 I}{\partial x^4} + \ldots.\right) - 2\tau\left(\frac{\partial I}{\partial t} + \frac{\tau}{2!}\frac{\partial^2 I}{\partial t^2} + \frac{\tau^2}{3!}\frac{\partial^3 I}{\partial t^3} + \ldots\right)\right)$$

$$+(k+\mu+\alpha_0)\left(I_i^n + \tau\frac{\partial I}{\partial t} + \frac{\tau^2}{2!}\frac{\partial^2 I}{\partial t^2} + \frac{\tau^3}{3!}\frac{\partial^3 I}{\partial t^3} + \ldots\right) -$$

$$\beta S_i^{n-m} I_i^{n-m} e^{-\delta\tau}.$$

$$= -\frac{d_2 h^2}{12}\left(\frac{\partial^4 I}{\partial x^4}\right) + \tau\left(\frac{\partial I}{\partial t} + \frac{\tau}{2!}\frac{\partial^2 I}{\partial t^2} + \frac{\tau^2}{3!}\frac{\partial^3 I}{\partial t^3} + \ldots\right) \tag{4.12}$$

$$\to 0 \quad \text{as} \quad h, \tau \to 0.$$

*System Stability at* $\epsilon^*$

To investigate the stability of the model, we linearize it around the point $\epsilon^*$ [35,34] As a consequence, the variational matrix around the equilibrium point is given by

$$\mathfrak{V} = \begin{pmatrix} \alpha_{11} - d_1\kappa^2 & \alpha_{12} \\ \alpha_{21} & \alpha_{22} - d_2\kappa^2 \end{pmatrix}. \tag{4.13}$$

where,

$$\begin{aligned} \alpha_{11} &= -\delta - \gamma I, \\ \alpha_{12} &= -\gamma S, \\ \alpha_{21} &= \gamma I e^{-\delta\tau}, \\ \alpha_{22} &= \gamma S e^{\delta\tau} - (\kappa + \delta + \delta_0). \end{aligned}$$

The characteristic polynomial associated with $\mathfrak{V}$ is expressed as;

$$\Upsilon^2 + \Xi_1\Upsilon + \Xi_2 = 0.$$

With

$$\Xi_1 = -(\alpha_{11} + \alpha_{22} - d_1\kappa^2 - d_2\kappa^2).$$

and

$$\Xi_2 = \alpha_{11}\alpha_{22} - \alpha_{12}\alpha_{21} - \alpha 11 d_1\kappa^2 - \alpha d_2\kappa^2 + d_1 d_2\kappa^4.$$

The stability condition furnished by Routh-Hurwitz guarantees that $\Xi_1 > 0$ and $\Xi_2 > 0$ are strictly positive. The stability of the submitted HIV infectious model for $\tau = 0$ and $\tau = 4$ is studied in the subsequent tables i.e. Table 2 and Table 3.

**Table 1**
Different sets of parametric values.

| Parameters | $N$ | $\Lambda$ | $\gamma$ | $\delta$ | $\kappa$ | $\delta_0$ |
|---|---|---|---|---|---|---|
| case-1 | 1000 | 0.05 | 0.01 | 0.2 | 0.1 | 0.4 |
| case-2 | 1000 | 0.05 | 0.02 | 0.2 | 0.1 | 0.4 |
| case-3 | 1000 | 0.05 | 0.03 | 0.2 | 0.1 | 0.4 |
| case-4 | 1000 | 0.05 | 0.04 | 0.2 | 0.1 | 0.4 |
| case-5 | 1000 | 0.05 | 0.05 | 0.2 | 0.1 | 0.4 |

**Significance role of the infection parameter** $\gamma$

**Case-I**, When $\tau = 0$.

The determinative value of the disease parameter $\gamma$, for the first case of Table 1, are calculated by taking benefit of the famous criterion set by Routh-Hurvitz [35,34]. To investigate the decisive value of the disease parameter bifurcation value. The value of $S^*$ and $I^*$ are substituted in the expressions of $\alpha_{11}, \alpha_{12}, \alpha_{21}$ and $\alpha_{22}$ to get the following relations,

$$\begin{aligned} \alpha_{11} &= -71.42857142857142\gamma, \\ \alpha_{12} &= -0.7, \\ \alpha_{21} &= 71.42857142857142\gamma - 0.2, \\ \alpha_{22} &= 0. \end{aligned}$$

By applying the Routh-Hurwitz principle, for the stability of the system, we get,

$$\begin{aligned} \Xi_1 &= 71.42857142857143\gamma + 0.0493480220054 = J_1(\gamma), \\ \Xi_2 &= 51.7624293573374\gamma - 0.139391193181037 = J_2(\gamma). \end{aligned}$$

$J_2(\gamma) = 0$ provides the bifurcation value for the parameter $\beta$, which stables the equilibrium state, i.e, the point of equilibrium. $J_2(\gamma) = 0$ provides the value of $\gamma$ as $\gamma = 0.002692902844$, $\gamma \geqslant 0.002692902844$ are the values that ensure the stability of the system at equilibrium state, otherwise unstable i.e. system is unstable, when $\gamma < 0.002692902844$.

**Case-II**, When $\tau = 4$.

Now for the case-II, the bifurcation values of $\gamma$ in the table (1) are also calculated by considering Routh-Hurwitz condition. To examine the decisive value of the infection parameter $\gamma$, the value of $S^*$ and $I^*$ are substituted in $\alpha_{11}, \alpha_{12}, \alpha_{21}$ and $\alpha_{22}$, as

$$\begin{aligned} \alpha_{11} &= -32.09492600837\gamma, \\ \alpha_{12} &= -1.5578786499447, \\ \alpha_{21} &= 14.4211798567611\gamma - 0.089865792823444, \\ \alpha_{22} &= 0. \end{aligned}$$

The well known stability condition described by Routh-Hurwitz ensures the following two expressions

$$\begin{aligned} \Xi_1 &= 32.09492600837297\gamma + 0.04934802200545 = J_1(\gamma), \\ \Xi_2 &= 23.25835876332327\gamma - 0.13939119318103 = J_2(\gamma). \end{aligned}$$

$J_2(\gamma) = 0$ decides the bifurcation value for the parameter $\gamma$ that ensures the stability of the system at endemic equilibrium. $J_2(\gamma) = 0$ decides that $\gamma = 0.0059931654938$. The endemic equilibrium is stable when $\gamma \geqslant 0.0059931654938$ and unstable otherwise.

**Simulations with example**

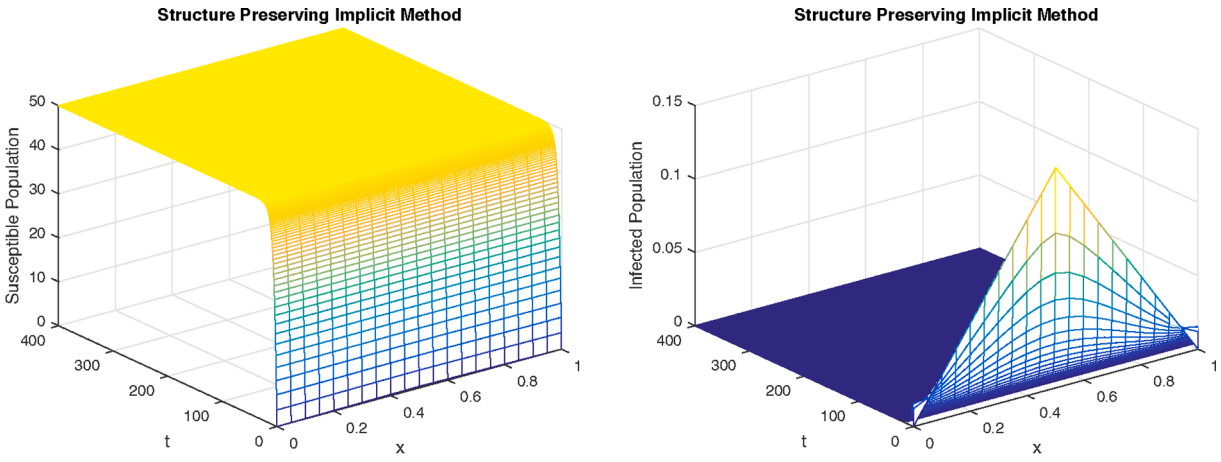
This part is devoted to present the numerical example with graphical behavior by using our suggested numerical approach. For disease free state, we consider the values of parameters as,

$N = 1000, \Lambda = 0.01, \gamma = 0.01, \delta = 0.2, \kappa = 0.1$ and $\delta_0$.

For disease free state, we consider the values of parameters as,

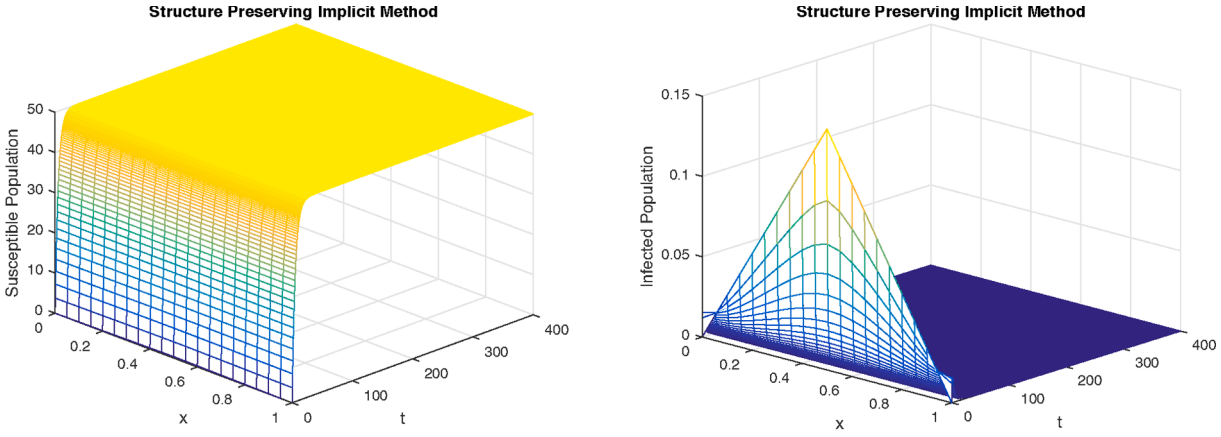
$N = 1000, \Lambda = 0.05, \gamma = 0.05, \delta = 0.2, \kappa = 0.1$ and $\delta_0$. The initial conditions are considered as,

$$S(x,0) = \begin{cases} 0.7x & 0 \leqslant x \leqslant 1/2, \\ 0.7(1-x) & 1/2 \leqslant x \leqslant 1. \end{cases}$$



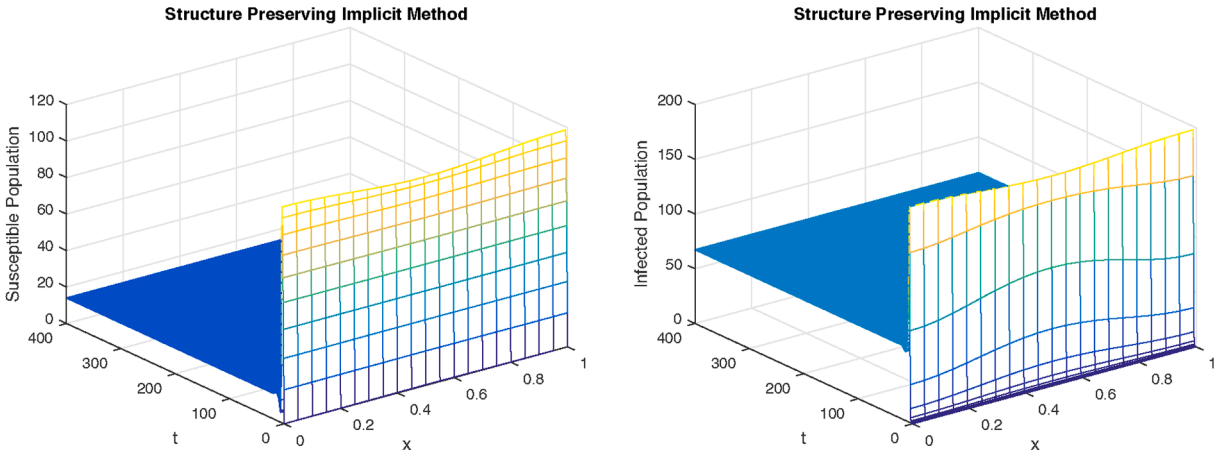

(a) Graphical solution of $S(x,t)$ (b) Graphical solution of $I(x,t)$

**Fig. 1.** Numerical simulations of $S(x,t)$ (susceptible population) using structure preserving implicit method for DFE point at $\Delta x = 0.05, \Delta t = 0.4, d_1 = d_2 = 0.01$ and $\tau = 0$.



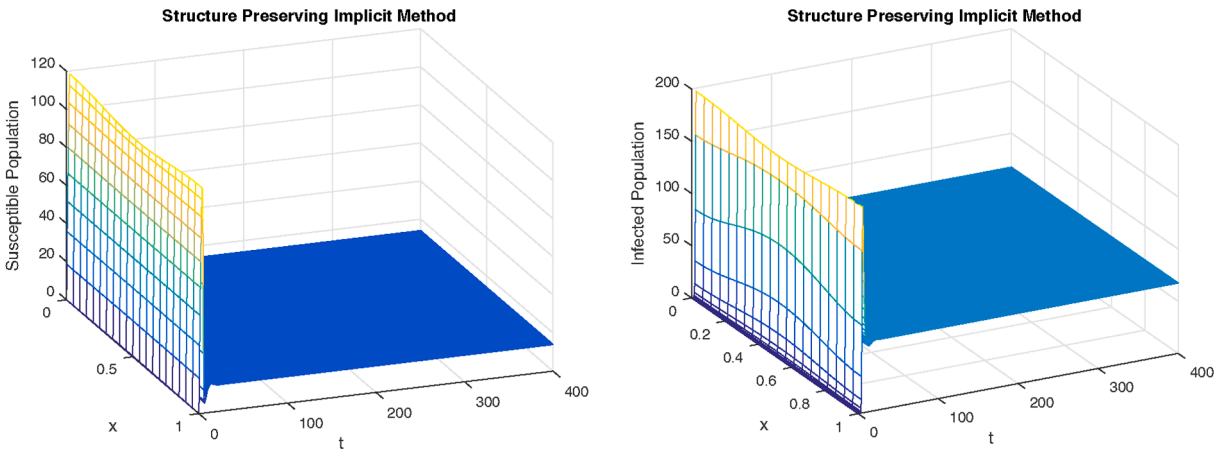

(a) Graphical solution of $S(x,t)$ (b) Graphical solution of $I(x,t)$

**Fig. 2.** Numerical simulations of $S(x,t)$ (susceptible population) using structure preserving implicit method for DFE point at $\Delta x = 0.05, \Delta t = 0.4, d_1 = d_2 = 0.01$ and $\tau = 0$ with different rotation.




(a) Graphical solution of $S(x,t)$ (b) Graphical solution of $I(x,t)$

**Fig. 3.** Numerical simulations of $S(x,t)$ (susceptible population) using structure preserving implicit method for EE point at $\Delta x = 0.05, \Delta t = 0.4, d_1 = d_2 = 0.01$ and $\tau = 0$.

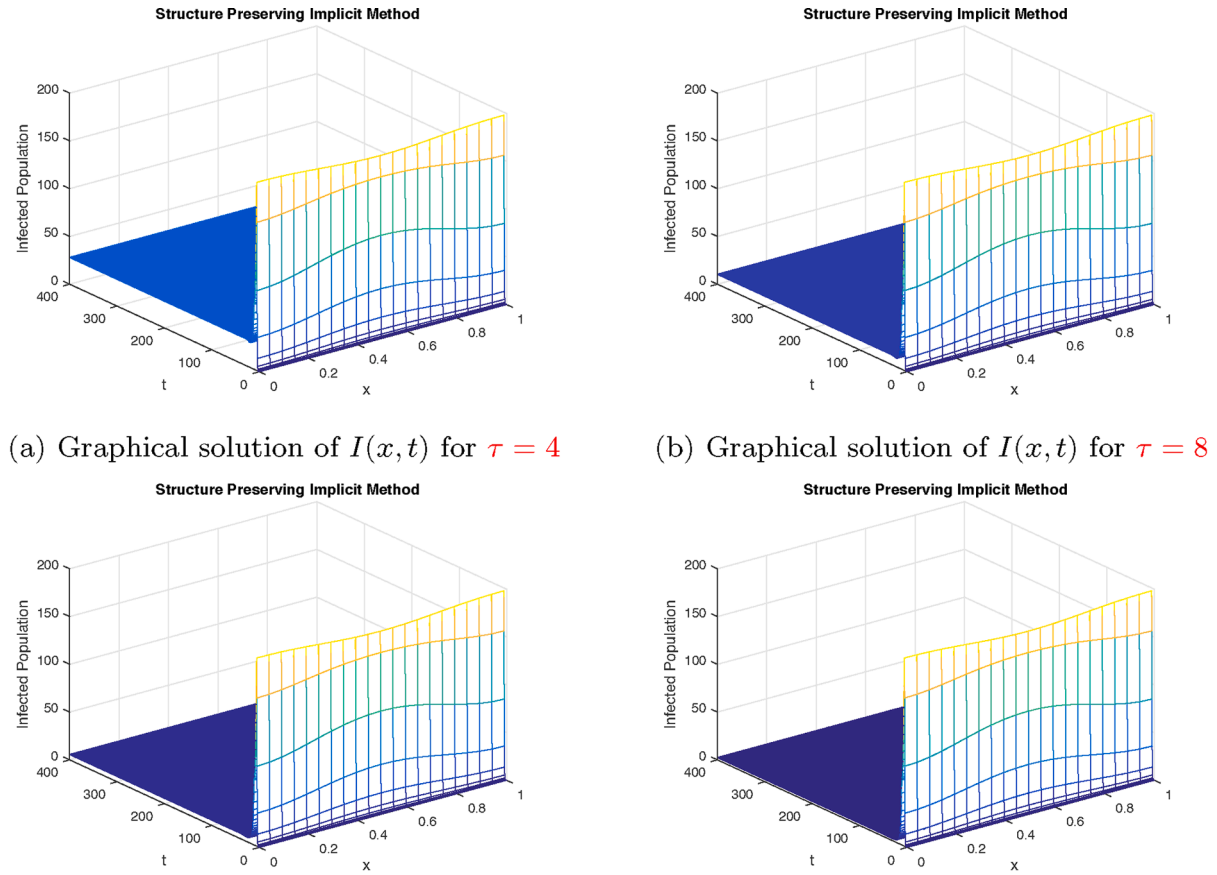



(a) Graphical solution of $S(x,t)$ (b) Graphical solution of $I(x,t)$

**Fig. 4.** Numerical simulations of $S(x,t)$ (susceptible population) using structure preserving implicit method for EE point at $\Delta x = 0.05, \Delta t = 0.4, d_1 = d_2 = 0.01$ and $\tau = 0$ with different rotation.

$$I(x,0) = \begin{cases} 0.3x & 0 \leqslant x \leqslant 1/2, \\ 0.3(1-x) & 1/2 \leqslant x \leqslant 1. \end{cases}$$

In Figs. 1 and 2, the numerical solution of HIV reaction-diffusion infection model is portrayed when $\tau = 0$ at the infection-free point with the aid of designed structure-preserving implicit technique. It can easily be determined from the graphical behavior that our numerical technique maintains the positivity specified by the underlying HIV infection model. Also, graphs with different view in Figs. 1 and 3 depict that this method sustains the stability at infection-free point. The proposed numerical scheme deals with the human population in a certain region. Also, the state variables are associated with the group of individuals having certain stage of infection. So, the values of the stat




(a) Graphical solution of $I(x,t)$ for $\tau = 4$ (b) Graphical solution of $I(x,t)$ for $\tau = 8$

(c) Graphical solution of $I(x,t)$ for $\tau = 10$ (d) Graphical solution of $I(x,t)$ for $\tau = 12$

**Fig. 5.** Numerical simulations of $I(x,t)$ (infected population) using structure preserving implicit method for EE point at $\Delta x = 0.05, \Delta t = 0.4, d_1 = d_2 = 0.01$.

variables can never be negative. The simulated graphs are in line with the fact mentioned above. The graphs in Figs. 1 and 2 provide a strong evidence that our scheme provides a non-negative solution at every moment of the time. Further, the numerical values of $S(x,t)$ and $I(x,t)$ converges to the exact values of $S(x,t)$ and $I(x,t)$ as investigated in Section "Mathematical model", i.e. the proposed scheme preserves the steady state (disease free) solution of continuous system.

Figs. 3 and 4 unveil the solution of HIV reaction-diffusion epidemic model graphically by using the suggested structure-preserving implicit technique at infection existence point. It is noted that these graphs are presented for $\tau = 0$. Again the under discussion scheme sustains the positivity of the state variable associated with the underlying HIV epidemic model. Also, this technique holds the stability of infection existence point as it is illustrated with different view in Figs. 3 and 4. Since, the projected system is a compartmental model, in which each compartment describes a definite populace of the community, according to the status of the infection. Additionally, the populace of any compartment is always positive at endemic state. The graphs $(a)$ and $b$ in both the Figs. 3 and 4 disclose that numerical solutions are always positive at every instance of time and ultimately the graphs converges towards the value $S^*$ and $I^*$ respectively. It shows that our proposed scheme retains the steady (endemic equilibrium) state solution of the continuous system.

In Fig. 5, the decisive role of the delay factor $\tau$ is illustrated. All the graphs in Fig. 5 show the infected individuals for the same values of parameters involved in the model for the existence of infection in the population, but the value of $\tau$ in each graph is changed. It is evident that the size of infected population decreases against the greater value of $\tau$. Equivalently, there is an inverse relation between the value of $\tau$ and the infected individuals. Furthermore, the infected population can be controlled up to a desired level against a certain value of $\tau$. The value of $\tau$ is considered as 4 in graph $(a)$, while $\tau = 8$ in sketch $(b)$, $\tau = 10$ in plot $(c)$ and the value of $\tau = 12$ in pattern $(d)$ of Fig. 5. I is apparent that the size of the infected group shrinks as the value of $\tau$ increased. So, it is obvious that infection can be controlled, significantly, by enhancing the delay factor level in the model.

## Conclusion

In this research work, we have successfully constructed the proposed implicit numerical scheme for the non linear HIV/AIDS model with diffusion and delay phenomenon. The projected scheme adequately preserves the core attributes of the continuous system, for instance positivity of the solutions, stability of the system at the points of equilibria and consistency. The system stability is also examined by using the Routh-Hurwitz criterion for different situations. The numerical and bifurcation analysis against different situations is also the part of this study. Numerical graphs are also presented with the help of computer simulations by selecting the suitable set of parametric values. These graphs provide the strong evidence about the reliability and efficiency of the scheme i.e. the proposed scheme is reliable and efficient numerical design for the non linear dynamical delay diffusion system. In future, the current work may be applied to various types of delay diffusive systems with multi space dimensions.

## CRediT authorship contribution statement

**Muhammad Jawaz:** Conceptualization, Methodology, Writing - original draft. **Muhammad Aziz ur Rehman:** Supervision, Writing - review & editing. **Nauman Ahmed:** Software, Validation. **Dumitru Baleanu:** Supervision, Writing - review & editing, Visualization. **Muhammad Rafiq:** Investigation, Validation, Formal analysis.

## Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

## Acknowledgement

We are very thankful to anonymous referees for their valuable suggestions to improve this manuscript.

## References

[1] Campbell EM, Jia H, Shankar A, Hanson D, Luo W, Masciotra S, Blosser SJ. Detailed transmission network analysis of a large opiate-driven outbreak of HIV infection in the United States. J Infect Dis 2017;216(9):1053–62.
[2] Qiao YC, Xu Y, Jiang DX, Wang X, Wang F, Yang J, Wei YS. Epidemiological analyses of regional and age differences of HIV/AIDS prevalence in China, 2004–2016. Int J Infect Dis 2019;81:215–20.
[3] Paraskevis D, Nikolopoulos G, Tsiara C, Paraskeva D, Antoniadou A, Lazanas M, Hatzakis A. HIV-1 outbreak among injecting drug users in Greece, 2011: a preliminary report. Eurosurveillance 2011;16(36):19962.
[4] Doitsh G, Greene WC. Dissecting how CD4 T cells are lost during HIV infection. Cell Host Microbe 2016;19(3):280–91.
[5] Cummins NW, Badley AD. Making sense of how HIV kills infected CD4 T cells: implications for HIV cure. Mol Cell Ther 2014;2(1):20.
[6] Poorolajal J, Hooshmand E, Mahjub H, Esmailnasab N, Jenabi E. Survival rate of AIDS disease and mortality in HIV-infected patients: a meta-analysis. Public Health 2016;139:3–12.
[7] Alemu A, Yesuf A, Zerihun B, Getu M, Worku T, Bitew ZW. Incidence and determinants of tuberculosis among HIV positives in Addis Ababa, Ethiopia: a Retrospective Cohort Study. Int J Infect Dis 2020.
[8] WHO. Global Tuberculosis Report Geneva; 2018.
[9] Ramírez BC, Vega YC, Shepherd BE, Le C, Turner M, Frola C, McGowan CC. Outcomes of HIV-positive patients with cryptococcal meningitis in the Americas. Int J Infect Dis 2017;63:57–63.
[10] Rajasingham R, Smith RM, Park BJ, Jarvis JN, Govender NP, Chiller TM, Boulware DR. Global burden of disease of HIV-associated cryptococcal meningitis: an updated analysis. Lancet Infect Dis 2017;17(8):873–81.
[11] Mekonnen Y, Hadush T, Tafere A, Tilahun A. A review article on cryptosporidiosis; 2016.
[12] Sullivan MC, Rosen AO, Allen A, Benbella D, Camacho G, Cortopassi AC, Kalichman SC. Falling Short of the First 90: HIV Stigma and HIV Testing Research in the 90–90-90 Era; 2020.
[13] NACP.https://www.nacp.gov.pk/index.html; 2019 (accessed on March 02, 2020).
[14] Chen X, Yu B, Zhao L. The evaluation of global epidemic of HIV/AIDS with a novel approach using country-specific counts of HIV infections and three rates controlled for population and geographic area. Global Health J 2019;3(3):66–72.
[15] Mir F, Mahmood F, Siddiqui AR, Baqi S, Abidi SH, Kazi AM, Memon SA. HIV infection predominantly affecting children in Sindh, Pakistan, 2019: a cross-sectional study of an outbreak. Lancet Infect Dis 2019.
[16] Li D, Ma W. Asymptotic properties of a HIV-1 infection model with time delay. J Math Anal Appl 2007;335:683–91. https://doi.org/10.1016/j.jmaa.2007.02.006.
[17] Abdullahi YM, Nweze O. A simulation of an sir mathematical model of HIV transmission dynamics using the classical Euler's method. Shiraz Med J 2011;12:196–202.
[18] Ahmed N, Rafiq M, Rehman MA, Iqbal MS, Ali M. Numerical modeling of three dimensional Brusselator system. AIP Adv 2019;09(01):01–19.
[19] Charpentier BMC, Kojouharov HV. Unconditionally positive preserving scheme for advection-diffusion-reaction equations. Math Comput Model 2013;57(10):2177.1285.
[20] Fujimoto T, Ranade R. Two characterizations of inverse positive matrices: the Hawkins-Simon condition and the le chatelier-braun principle. Electron J Linear Algebra ELA 2004;11(1):01–18.
[21] Plemmons RJ. M-matrix characterizations nonsingular m-matrices. Linear Algebra Appl 1977;18(02):175–88.
[22] Jiang X, Chen X, Huang T, Yan H. Bifurcation and control for a predator-prey system with two delays. IEEE Trans Circuits Syst II: Express Briefs 2021;68(1):376–80.
[23] Jiang X, Chen X, Chi M, Chen J. On Hopf bifurcation and control for a delay systems. Appl Math Comput 2020;370(1):124906.
[24] Asif M, Ullah Jan S, Haider N, Mdallal QA, Jawad TA. Numerical modeling of NPZ and SIR models with and without diffusion. Result Phys 19; 2020.
[25] Asif M, Khan ZA, Haider N, Mdallal QA. Numerical simulation for solution of SEIR models by meshless and finite difference methods. Chaos Soliton Fractals 2020;141.
[26] Hajji MA, Mdallal QA. Numerical simulations of a delay model for immune system-tumor interaction. Sultan Qaboos Univ J Sci 2018;23(1):19–31.
[27] Rihan FA, Al-Mdallal QM, AlSakaji HJ, Hashish A. A fractional-order epidemic model with time-delay and nonlinear incidence rate. Chaos Solitons Fractals 2019;126:97–105.

[28] Ullah I, Ahmad S, Mdallal QA, Khan ZA, Khan H, Khan A. Stability analysis of a dynamical model of tuberculosis with incomplete treatment. Adv Differ Equ 2020; 499.
[29] Smith GD. Numerical solution of partial differential equations: finite difference methods. 3rd ed. Oxford, UK: Clarendon Press; 1985.
[30] Mitchell AR, Griffiths DF. Finite difference methods in partial differential equations. Hoboken, NJ, USA: T. Wiley; 1980.
[31] Chinviriyasit S, Chinviriyasit W. Numerical modelling of an SIR epidemic model with diffusion. Appl Math Comput 2010;216:395–409.
[32] Kustiawan C. A Numerical scheme for a reaction diffusion equation with time delay and impulses. Far East J Math Sci 2018;106(2):451–62.
[33] Ahmed N, Elsonbaty A, Adel W, Baleanu D, Rafiq M. Stability analysis and numerical simulations of spatiotemporal HIV CD4+ T cell model with drug therapy. Chaos 2020;30:083122.
[34] Chakrabrty A, Singh M, Lucy B, Ridland P. Predator-prey model with pry-taxis and diffusion. Math Comput Model 2007;46:482–98.
[35] Ahmed N, Tahira SS, Rafiq M, Rehman MA, Ali M, Ahmad MO. Positivity preserving operator splitting nonstandard finite difference methods for SEIR reaction diffusion model. Open Math 2019;17:313–30.