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Investigating the impact of stochasticity on HIV infection dynamics in CD4+ T cells using a reaction-diffusion model

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The disease dynamics affect the human life. When one person is affected with a disease and if it is not treated well, it can weaken the immune system of the body. Human Immunodeficiency Virus (HIV) is a virus that attacks the immune system, of the body which is the defense line against diseases. If it is not treated well then HIV progresses to its advanced stages and it is known as Acquired Immunodeficiency Syndrome (AIDS). HIV is typically a disease that can transferred from one person to another in several ways such as through blood, breastfeeding, sharing needles or syringes, and many others. So, the need of the hour is to consider such important disease dynamics and that will help mankind to save them from such severe disease. For the said purpose the reaction-diffusion HIV $CD4^+$ T cell model with drug therapy under the stochastic environment is considered. The underlying model is numerically investigated with two time-efficient schemes and the effects of various parameters used in the model are analyzed and explained in a real-life scenario. Additionally, the obtained results will help the decision-makers to avoid such diseases. The random version of the HIV model is numerically investigated under the influence of time noise in Itô sense. The proposed stochastic backward Euler (SBE) scheme and proposed stochastic Implicit finite difference (SIFD) scheme are developed for the computational study of the underlying model. The consistency of the schemes is proven in the mean square sense and the given system of equations is compatible with both schemes. The stability analysis proves that both schemes and schemes are unconditionally stable. The given system of equations has two equilibria, one is disease-free equilibrium (DFE) and the other is endemic equilibrium. The simulations are drawn for the different values of the parameters. The proposed SBE scheme showed the convergent behavior towards the equilibria for the given values of the parameters but also showed negative behavior that is not biological. The proposed SIFD scheme showed better results as compared with the stochastic SBE scheme. This scheme has convergent and positive behavior towards the equilibria points for the given values of the parameters. The effect of various parameters is also analyzed. Simulations are drawn to evaluate the efficacy of the schemes.

Keywords Stochastic HIV model, Proposed SBE scheme, Proposed SIFD scheme, Analysis of schemes, Simulations

Any phenomena in physical sciences can be modeled by partial differential equations (PDEs). For example, in physics, the flow of temperature in a body and transmission of waves are expressed by PDEs. In the field of

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biology, most models related to people are expressed by partial differential equations. It is very easy to define that most of the phenomena governing population dynamics, quantum physics, disease dynamics, fluid dynamics, and some other models lie inside the domain of PDEs. In Epidemiology, disease dynamics are considered and analyzed. Infectious diseases are causing serious threats to population dynamics. Several diseases affect human life severely and one of them is acquired immunodeficiency syndrome (AIDS) which spreads due to the virus known as human immunodeficiency virus (HIV). Since the initial days of HIV, numerous works have been devoted to the understanding complexity of this virus. The HIV models are divided into two categories: one is a within-host model and the other is a population-level model[1–5]. The immune system of the body fights with all types of germs and tries to save it from disease and if the immune system gets weak then the disease affects the body according to the severity of the virus. Similarly, HIV enters the body and starts to damage the $CD4^+$ T cells and if the immune system is strong then the $CD4^+$ T cells d remain to save and if it is weak then the number of the $CD4^+$ T cells starts to reduce. If the patient has a weak immune system and is checked timely and then by using a different medication facility, he can be cured. There are various mathematical models which describe the different dynamics of the HIV models. Zafar et al. worked on the approximate solution of a non-integer HIV epidemic model. They used the Grunwald Letnikov non-standard finite difference scheme for numerical study and this scheme preserves the positivity and boundedness. They analyzed the different dynamics of the given model and verified the theoretical results with simulations[6]. Raza et al. considered a stochastic HIV/AIDS model in a two-sex population with antiretroviral therapy and counseling. They compared the numerical results of the deterministic and stochastic HIV model and concluded that the stochastic model is more realistic than the deterministic[7]. Huo et al. incorporated the treatment T compartment in the HIV model and analyzed it. They analyzed the stability of disease-free and endemic equilibria for $R_0 < 1$ and $R_0 > 1$ respectively and proved that equilibria are globally asymptotically stable[8].

For human medication, it is necessary to test the drugs and other therapies on the animals. Hatziioannou and Evans worked on the animal models for HIV/AIDS[9]. Silva and Torres proposed an HIV/AIDS model of non-integer order. They analyzed the global and local stability of the given model and explained the theoretical results through the numerical simulations[10]. Khan et al. studied the fractional order HIV/AIDS having Atangana–Baleanu–Caputo and Liouville–Caputo derivatives. From analysis, they observed that some species moved from the symptomatic to the asymptomatic phase. They used various techniques for numerical study and discussed convergence as well. They used the simulation for the result illustration[11]. Cai et al. investigated the HIV/AIDS epidemic model and analyzed the global and local stability of the given model. They introduced the discrete time delay in the model and investigated the effect of the delay factor on the stability of endemic equilibrium[12]. El-Metwaly et al. investigated the dynamics of the HIV models with stochastic perturbation. They derived the global exponential stability of DFE with a reproductive number less than unity[13].

Many researchers are working on solving stochastic partial differential equations (SPDEs) numerically. Roth developed the finite difference method (FDM) for the solutions of SPDEs. He developed the analysis of the stochastic scheme[14]. Roth also used the FDM along with the Wong–Zakai technique for the numerical study of SPDEs[15]. Kamrani and Hosseini work on the solution of a general class of SPDEs. They discussed the stability and consistency of the schemes and analyzed the role of coefficients on the general SPDEs[16]. The authors showed the existence of a solution for the SPDEs and their solution by various techniques[17–19]. Yoo worked on the approximation of the SPDEs by using finite difference methods[20]. Allen et al. worked on the numerical solution of elliptic and parabolic SPDEs[21].

Wu and Zhao proposed a new age-space structured model that incorporates both two spatial diffusion, infection age, and highly active antiretroviral therapy to investigate the global dynamics of the HIV epidemic model and its transmission in humans. The authors established the well-posedness and positivity of the solution. They conclude that the disease-free equilibrium is globally asymptotically stable when the basic reproduction number is less than one[22]. Wu et al. worked on the global dynamics of the three-age-structured, spacial diffusion, viral load-dependent infection, and conversion rates of HIV/AIDS. The authors also established the global stability of steady states and the uniform persistence of the disease. From the graphical behavior, they concluded that safety measures are helpful at the individual and population levels in controlling the transmission of the disease[23]. Wu et al. proposed a model to analyze the impact of infection age, spatial diffusion, and treatment adherence on HIV/AIDS transmission among humans[24]. Wang et al. proposed a susceptible-infected-susceptible reaction-diffusion epidemic model with cognition and analyzed the impact of movement strategies on disease outbreak[25]. The authors[26] analyzed a dynamical model of an evolving epidemic in a spatially inhomogeneous environment. They established various results for the epidemic model. Li and Wei considered stochastic diffusive COVID-19 model, to analyzeits various dynamics[27].

The physical systems are the basic part of nature. Researchers used different techniques to understand these phenomena and demonstrated physical models by using a set of equations. They used analytical and approximation techniques to find the solution of the models. These solutions helped them to understand the complicated phenomena and make decisions for the betterment of human life. The life of man has links with living organisms, nonliving things, and disease dynamics. When disease dynamics are simply observed over time, labeled as temporal dynamics of disease and when observed over both time and space, they are referred to as spatiotemporal dynamics of the disease. When we see the disease dynamics at the micro-scale, they have stochastic behavior for their spread. It becomes difficult for scientists when disease dynamics are considered in time, space coordinates with diffusion process and stochastic behavior. We have tried to solve the underlying model with two computational schemes. The stochastic backward Euler scheme is applied and negative behavior for given values on the other hand stochastic implicit scheme preserves the stable, convergent, and positive behavior for given values of the parameters. The obtained solutions are explained in real-life applications. Both schemes are time efficient.

The novelty of this work is given below:

- The classical epidemic models cannot predict the true behavior of the infectious disease. So, stochastic epidemic models are more accurate and preferred. We are considering the stochastic epidemic model for HIV/AIDS disease.
- Two schemes are used for the numerical approximation of the underlying model.
- Both schemes are consistent with the system of equations and have stable behavior in the mean square sense.
- The given model has two equilibrium points: both are successfully gained.
- The graphical behavior of the state variables is explained from the biological point of view.
- The MATLAB 2015a is used for the graphical behavior of the test problem.

Problem statement

Nauman et al.[28] worked on below model

$$T_\tau = d_T T_{xx} + s - \mu T - k_0 LT + \eta\epsilon I + bI, \qquad I_\tau = d_I I_{xx} - (\mu_1 + b + \epsilon)I + k_0 LT,$$
$$V_\tau = d_V V_{xx} + (1-\eta)\epsilon I - \delta V, \qquad L_\tau = d_L L_{xx} + N\delta V - cL,$$

with suitable initial and boundary conditions. HIV is an infection that damages the $CD4^+$ T cells in the body and the white blood cells[29,30]. Where T represents the density of susceptible $CD4^+$ T cells, while I is a reverse transcription (RT) class, and L is shown as the infected $CD4^+$ T cells and L virus. The $d_T > 0, d_I > 0, d_V > 0$, and $d_L > 0$ are the diffusion coefficients, s represents the influx rate in $CD4^+$ T cells, the exchange contamination rate of $CD4^+$ T cells is k_0, while μ is the death rate, the death rate of contaminated cells by μ_1, the transition rate from pre-RT contaminated cells to post-RT contaminated cells is represented by ϵ, b is the conversion rate of contaminated cells to uncontaminated cells, δ show the death rate of the actively contaminated cell, c represents the virus's clearance rate and the viral particles N which are produced by the contaminated cells.

We are considering the HIV model influenced by stochastic perturbation as

$$T_t = d_T T_{xx} + s - k_0 LT - \mu T + (\eta\epsilon + b)I + \nu_1 T\dot{B}_1(t), \tag{1}$$

$$I_t = d_I I_{xx} + k_0 LT - (\mu_1 + \epsilon + b)I + \nu_2 I\dot{B}_2(t), \tag{2}$$

$$V_t = d_V V_{xx} + (1-\eta)\epsilon I - \delta V + \nu_3 V\dot{B}_3(t), \tag{3}$$

$$L_t = d_L L_{xx} + N\delta V - cL + \nu_4 L\dot{B}_4(t), \tag{4}$$

here ν_i, $i = 1,2,3,4$ are the noise strength of the stochastic process and $\dot{B}_i(t)$, $i = 1,2,3,4$ are the standard Wiener process. Let $\mathbb{T} > 0$ and $(\Omega, \mathfrak{F}, \mathfrak{P})$ is the probability space having normal filtration $(\mathfrak{F}_t)_{t\geq 0}$, and $\{B_i(t), i = 1,2,3,4.t \geq 0\}$ is the Brownian motion defined over the filtered probability space and it has the following properties

- B(s)-B(t) for $s > t$ does not depend on the past.
- B(s)-B(t) for $s > t$ has normal distribution with mean zero and variance $s - t$
- B(t), $t \geq 0$ are continuous function of t.

Numerical methods

For the numerical study of the given system of equations, first the discretization of the whole domain of space x and temporal t variables. The grid points (x_d, t_e) are explained as

$$x_d = dh, d = 0,1,2,3,\ldots,M.$$
$$\tau_e = ek, e = 0,1,2,3,\ldots,N_1.$$

Here, M and N_1 are the integers and $\Delta x = h$, $\Delta t = k$ are stepsizes of space and temporal respectively.

The proposed SBE scheme for the given system of equations is given below

$$-r_1 T_{d+1}^{e+1} + (1+2r_1)T_d^{e+1} - r_1 T_{d-1}^{e+1} = sk + (1 - k_0 kL_d^e - \mu k)T_d^e + k(\eta\epsilon + b)I_d^e + \nu_1 T_d^e \Delta B_1, \tag{5}$$

$$-r_2 I_{d+1}^{e+1} + (1+2r_2)I_d^{e+1} - r_2 I_{d-1}^{e+1} = k_0 kL_d^e T_d^e + (1 - k(\mu_1 + \epsilon + b))I_d^e + \nu_2 I_d^e \Delta B_2, \tag{6}$$

$$-r_3 V_{d+1}^{e+1} + (1+2r_3)V_d^{e+1} - r_3 V_{d-1}^{e+1} = k(1-\eta)\epsilon I_d^e - (k\delta - 1)V_d^e + \nu_3 V_d^e \Delta B_3, \tag{7}$$

$$-r_4 L_{d+1}^{e+1} + (1+2r_4)L_d^{e+1} - r_4 L_{d-1}^{e+1} = kN\delta V_d^e - (kc - 1)L_d^e + \nu_4 L_d^e \Delta B_4. \tag{8}$$

The Eqs. (5–8) are the proposed SBE scheme for the given system of equations.

The proposed SIFD scheme for the underlying model is given below

$$-r_1 T_{d+1}^{e+1} + (1 + 2r_1 + k_0 kL_d^e + \mu\Delta\tau)T_d^{e+1} - r_1 T_{d-1}^{e+1} = sk + T_d^e + k(\eta\epsilon + b)I_d^e + \nu_1 T_d^e \Delta B_1 \tag{9}$$

$$-r_2 I_{d+1}^{e+1} + (1 + 2r_2 + k(\mu_1 + \epsilon + b))I_d^{e+1} - r_2 I_{d-1}^{e+1} = kk_0 L_d^e T_d^e + I_d^e + \nu_1 I_d^e \Delta B_2 \tag{10}$$

$$-r_3 V_{d+1}^{e+1} + (1 + 2r_3 + k\delta)V_d^{e+1} - r_3 V_{d-1}^{e+1} = k(1-\eta)\epsilon I_d^e + V_d^e + \nu_3 V_d^e \Delta B_3, \tag{11}$$

$$-r_4 L_{d+1}^{e+1} + (1 + 2r_4 + kc)L_d^{e+1} - r_4 L_{d-1}^{e+1} = kN\delta V_d^e + L_d^e + \nu_4 L_d^e \Delta B_4, \tag{12}$$

here, $r_1 = \frac{d_T k}{h^2}, r_2 = \frac{d_I k}{h^2}, r_3 = \frac{d_V k}{h^2}, r_4 = \frac{d_L k}{h^2}$ and $\Delta B_i = (B_i^{(e+1)k} - B_i^{ek}), i = 1, 2, 3, 4.$

Stability

To Von-Neumann's method of stability,

$$Z_m^n = \frac{1}{\sqrt{2\pi}} \int_{\frac{-x}{\Delta x}}^{\frac{x}{\Delta x}} \exp^{im\Delta x\xi} \hat{Z}_m^n(\xi) d(\xi), \tag{13}$$

where $\hat{Z}^n$ is the Fourier Transformation of Z^n.

$$\hat{Z}_m^n = \frac{1}{\sqrt{2\pi}} \sum_{-\infty}^{\infty} \exp^{-im\Delta x\xi} Z_m^n \Delta x, \tag{14}$$

The stability technique for this method[31].

$$E\left|g(\xi\Delta x, \Delta t, \Delta x)\right|^2 \leq 1 + \eta\Delta t, \tag{15}$$

here η is the constant value.

Theorem 1 *This theorem is state that "in mean square sense, the scheme for T, I, V, L by Eqs. (5, 6, 7, 8) is unconditionally stable".*

Proof The stability analysis of numerical schemes is carried out by various criteria but Von-Neumann is used due to its linear analysis. So, Eq. (5) is linearized as follow

$$-r_1 T_{d+1}^{e+1} + (1 + 2r_1)T_d^{e+1} - r_1 T_{d-1}^{e+1} = (1 - \mu\Delta t)T_d^e + \nu_1 T_d^e \Delta B_1, \tag{16}$$

By using the Eq. (13), Eq. (16) becomes

$$\frac{1}{\sqrt{2\pi}} \int_{\frac{-\pi}{\Delta x}}^{\frac{\pi}{\Delta x}} e^{id\Delta x\xi} \left(-r_1 e^{i\Delta x\xi} + 1 + 2r_1 - r_1 e^{-i\Delta x\xi}\right) \hat{T}_d^{(e+1)}(\xi) d(\xi) =$$
$$\frac{1}{\sqrt{2\pi}} \int_{\frac{-\pi}{\Delta x}}^{\frac{\pi}{\Delta x}} e^{id\Delta x\xi} (1 - \mu\Delta t + \nu_1 \Delta B_1) \hat{T}^e(\xi) d(\xi),$$

The amplification factor, we get

$$g_1(\xi\Delta x, \Delta t, \Delta x) = \frac{1 - \mu\Delta t}{\left(1 + 4r_1 \sin^2(\frac{\Delta x\xi}{2})\right)} + \frac{\nu_1 \Delta B_1}{\left(1 + 4r_1 \sin^2(\frac{\Delta x\xi}{2})\right)},$$

by using the independence of the Wiener process and amplification factor becomes

$$E\left|g_1(\xi\Delta x, \Delta t, \Delta x)\right|^2 \leq \left|\frac{(1 - \mu\Delta t)}{\left(1 + 4r_1 \sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 + \left|\frac{\nu_1}{\left(1 + 4r_1 \sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 \Delta t,$$

$$\left|\frac{(1-\mu\Delta t)}{\left(1+4r_1 \sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 \leq 1,$$

$$E\left|g_1(\xi\Delta x, \Delta x, \Delta t)\right|^2 \leq 1 + \lambda_1 \Delta t,$$

where $\lambda_1 = \frac{\nu_1}{\left(1+4r_1 \sin^2(\frac{\Delta x\xi}{2})\right)}$. Thus, it is stable.

By using the same technique for the Eqs. (6, 7, 8), the amplification factor of these equations in means square sense with the independence of the Wiener process,

$$E\left|g_2(\xi\Delta x, \Delta t, \Delta x)\right|^2 \leq \left|\frac{1 - \Delta t(\mu_1 + \epsilon + b)}{\left(1 + 4r_2 \sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 + \left|\frac{\nu_2}{\left(1 + 4r_2 \sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 \Delta t,$$

$$\left|\frac{1-\Delta t(\mu_1+\epsilon+b)}{\left(1+4r_2\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 \leq 1,$$

$$E\left|g_2(\xi\Delta x,\Delta x,\Delta t)\right|^2 \leq 1+\lambda_2\Delta t,$$

where $\lambda_2 = \left|2\frac{\nu_2}{\left(1+4r_2\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2$ Thus, it is stable.

$$E\left|g_3(\xi\Delta x,\Delta t,\Delta x)\right|^2 \leq \left|\frac{(1-\Delta t\delta)}{\left(1+4r_3\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 + \left|\frac{\nu_3\Delta B_{3d}}{\left(1+4r_3\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2,$$

$$\left|\frac{(1-\Delta t\delta)}{\left(1+4r_3\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 \leq 1,$$

$$E\left|g_3(\xi\Delta x,\Delta x,\Delta t)\right|^2 \leq 1+\lambda_3\Delta t,$$

$\lambda_3 = \left|\frac{\nu_3}{\left(1+4r_3\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2$. Thus, it is stable.

$$E\left|g_4(\xi\Delta x,\Delta t,\Delta x)\right|^2 \leq \left|\frac{1-\Delta tc}{\left(1+4r_4\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 + \left|\frac{\nu_4\Delta B_{4d}}{\left(1+4r_4\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2,$$

$$\left|\frac{1-\Delta tc}{\left(1+4r_4\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 \leq 1,$$

$$E\left|g_4(\xi\Delta x,\Delta x,\Delta t)\right|^2 \leq 1+\lambda_4\Delta t$$

where $\lambda_4 = \left|\frac{\nu_4}{\left(1+4r_4\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2$. Thus, it is stable.

Theorem 2 *In mean square sense, the scheme for T, I, V, L by Eqs. (9, 10, 11, 12) is unconditionally stable.*

Proof Von-Neumann is used for the stability analysis. So, Eq. (9) is linearized as

$$-r_1T_{d+1}^{e+1} + (1+2r_1+\mu\Delta t))T_d^{e+1} - r_1T_{d-1}^{e+1} = T_d^e + \nu_1T_d^e\Delta B_1, \tag{17}$$

Then, the above equation can be written as,

$$\frac{1}{\sqrt{2\pi}}\int_{\frac{-\pi}{\Delta x}}^{\frac{\pi}{\Delta x}} \exp^{id\Delta x\xi}\left(-r_1(e^{i\Delta x\xi}) + (1+2r_1+\mu\Delta t) - r_1(e^{-i\Delta x\xi})\right)\hat{T}^{(e+1)}(\xi)d(\xi)$$
$$= \frac{1}{\sqrt{2\pi}}\int_{\frac{-\pi}{\Delta x}}^{\frac{\pi}{\Delta x}} e^{id\Delta x\xi}(1+\nu_1\Delta B_1))\hat{T}^e(\xi)d(\xi),$$

So, by an amplification factor,

$$f_1(\xi\Delta x,\Delta t,\Delta x) = \frac{1+\nu_1\Delta B_1}{\left(1+\mu\Delta t+4r_1\sin^2(\frac{\Delta x\xi}{2})\right)},$$

using the independence of the Wiener process, then the amplification factor becomes

$$E\left|f_1(\xi\Delta x,\Delta t,\Delta x)\right|^2 \leq \left|\frac{1}{1+\mu\Delta t+4r_1\sin^2(\frac{\Delta x\xi}{2})}\right|^2 + \left|\frac{\nu_1}{\left(1+\mu\Delta t+4r_1\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2\Delta t,$$

$\left|\frac{1}{\left(1+\mu\Delta t+4r_1\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 \leq 1$, then and $\left|\frac{\nu_1}{\left(\mu\Delta t+1+4r_1\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 = \lambda_5$

$$E\left|f_1(\xi\Delta x,\Delta t,\Delta x)\right|^2 \leq 1+\lambda_5\Delta t,$$

Thus, it is stable.

By using the same technique for the Eqs. (10, 11, 12), the amplification factor of these equations in means square sense with the independence of the Wiener process,

$$E\left|f_2(\xi\Delta x,\Delta t,\Delta x)\right|^2 \le \left|\frac{1}{\left(1+\Delta t(\mu_1+\epsilon+b)+4r_2\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 + \left|\frac{\nu_2}{\left(1+\Delta t(\mu_1+\epsilon+b)+4r_2\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 \Delta t,$$

$$\left|\frac{1}{\left(1+\Delta t(\mu_1+\epsilon+b)+4r_2\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 \le 1,$$

$$E\left|f_2(\xi\Delta x,\Delta t,\Delta x)\right|^2 \le 1+\lambda_6\Delta t,$$

where $\lambda_6 = \left|2\frac{\nu_2}{\left(1+\Delta t(\mu_1+\epsilon+b)+4r_2\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2$ Thus, it is stable.

$$E\left|f_3(\xi\Delta x,\Delta t,\Delta x)\right|^2 \le \left|\frac{(1)}{\left(1+\Delta t\delta+4r_3\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 + \left|\frac{\nu_3\Delta B_{3d}}{\left(1+\Delta t\delta+4r_3\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2,$$

$$\left|\frac{1}{\left(1+\Delta t\delta+4r_3\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 \le 1,$$

$$E\left|f_3(\xi\Delta x,\Delta t,\Delta x)\right|^2 \le 1+\lambda_7\Delta t,$$

$\lambda_7 = \left|\frac{\nu_3}{\left(1+\Delta t\delta+4r_3\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2$. Thus, it is stable.

$$E\left|f_4(\xi\Delta x,\Delta t,\Delta x)\right|^2 \le \left|\frac{1}{\left(1+\Delta tc+4r_4\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2 + \left|\frac{\nu_4\Delta B_{4d}}{\left(1+\Delta tc+4r_4\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2,$$

$$\left|\frac{1}{1+\Delta tc+4r_4\sin^2(\frac{\Delta x\xi}{2})}\right|^2 \le 1,$$

$$E\left|f_4(\xi\Delta x,\Delta t,\Delta x)\right|^2 \le 1+\lambda_8\Delta t$$

where $\lambda_4 = \left|\frac{\nu_4}{\left(1+\Delta tc+4r_4\sin^2(\frac{\Delta x\xi}{2})\right)}\right|^2$. Thus, it is stable.

Consistency of schemes

The consistency showed the compatibility of a scheme with given equations.

Theorem 3 *In a mean square sense, the proposed SBE scheme given by Eqs. (5–8) for state variables T, I, V, L is consistent with Eqs. (1–4)*[31,32].

Proof Let us suppose that T is a smooth function and applying the integral $H(g)=\int_{ek}^{(e+1)k} g d\nu$ on (1) then we get

$$\begin{aligned} H(T)_d^e =& T(dh,(e+1)k)-T(dh,ek)-d_T\int_{ek}^{(e+1)k}T_{xx}(dh,\nu)d\nu-\int_{ek}^{(e+1)k}sd\nu \\ &+k_0\int_{ek}^{(e+1)k}(T(dh,\nu)L(dh,\nu))d\nu+\mu\int_{ek}^{(e+1)k}T(dh,\nu)d\nu \\ &-(\eta\epsilon+b)\int_{ek}^{(e+1)k}T(dh,\nu)d\nu-\nu_1\int_{ek}^{(e+1)k}T(dh,\nu)dB_1(\nu), \end{aligned} \tag{18}$$

Eq. (1) can be written as,

$$\begin{aligned} H_d^e(T) =& T(dh,(e+1)k)-T(dh,eK)-sk \\ &-d_Tk\left(\frac{T((d+1)\Delta x,(e+1)k)-2T(dh,(e+1)k)+T((d-1)\Delta x,(e+1)k)}{\Delta x^2}\right) \\ &+kk_0(T(dh,ek)L(dh,ek))+\mu kT(dh,ek)-(\eta\epsilon+b)T(dh,ek)-\nu_1kT(dh,ek)dB_{ek}, \end{aligned}$$

In the mean square sense, the equations take the form and by using the Itô integral's square property

$$E|H(T)_d^e - H_d^e(T)|^2 \leq +5k_0^2E\left|\int_{ek}^{(e+1)k}\Big(T(dh,t)L(dh,t) - T(dh,ek)L(dh,ek)\Big)dv\right|^2$$
$$+ 5d_T^2E\left|\int_{ek}^{(e+1)k}\left(-T_{xx}(dh,v) + \frac{T((d+1)\Delta x,(e+1)k) - 2T(dh,(e+1)k) + T((d-1)\Delta x,(e+1)k)}{\Delta x^2}\right)dv\right|^2$$
$$+ 4\mu^2E\left|\int_{ek}^{(e+1)k}\Big(-T(dh,v) + T(dh,ek)\Big)dv\right|^2 + 5(\eta\epsilon+b)^2E\left|\int_{ek}^{(e+1)k}\Big(-I(dh,v) + I(dh,ek)\Big)dv\right|^2$$
$$+ 5(\nu_1)^2\int_{ek}^{(e+1)k}E\left|\Big(-T(dh,v) + T(dh,ek)\Big)\right|^2dv,$$

$E|H(T)_d^e - H_d^e(T)|^2 \to 0$ as $(d,e) \to \infty$, so it is consistent. Similarly, the consistency of the scheme for the Eqs. (6–8) can be proved.

Theorem 4 *In a mean square sense, the proposed SIFD scheme given by Eqs. (9–12) for state variables T, I, V, L is consistent with Eqs. (1–4).*

Proof Let us suppose that T is a smooth function and applying the integral operator as $H(g) = \int_{ek}^{(e+1)k} gdv$ by applying the operator on (1) then we get

$$\begin{aligned} H(T)_d^e =& T(dh,(e+1)k) - T(dh,ek) - d_T\int_{ek}^{(e+1)k}T_{xx}(dh,v)dv - \int_{ek}^{(e+1)k}sdv \\ &+ k_0\int_{ek}^{(e+1)k}(T(dh,v)L(dh,t))dv + \mu\int_{ek}^{(e+1)k}T(dh,v)dv \\ &- (\eta\epsilon+b)\int_{ek}^{(e+1)k}T(dh,v)dt - \nu_1\int_{ek}^{(e+1)k}T(dh,v)dB_1v, \end{aligned} \tag{19}$$

Eq. (1) can be written as,

$$\begin{aligned} H_d^e(T) =& T(dh,(e+1)k) - T(dh,ek) - sk \\ &- d_Tk\left(\frac{T((d+1)\Delta x,(e+1)k) - 2T(dh,(e+1)k) + T((d-1)\Delta x,(e+1)k)}{\Delta x^2}\right) \\ &+ kk_0(T(dh,(e+1)k)L(dh,ek)) + \mu kT(dh,\Delta(e+1)k) - (\eta\epsilon+b)T(dh,k) - \nu_1kT(dh,ek)dB_{ek}, \end{aligned}$$

In the mean square sense, the equations take the form and by using the Itô integral's square property

$$E|H(T)_d^e - H_d^e(T)|^2 \leq +5k_0^2E\left|\int_{ek}^{(e+1)k}\Big(T(dh,v)L(dh,v) - T(dh,k)L(dh,(e+1)k)\Big)dv\right|^2$$
$$+ 5d_T^2E\left|\int_{ek}^{(e+1)k}\left(T_{xx}(dh,v) + \frac{T((d+1)\Delta x,(e+1)k) - 2T(dh,(e+1)k) + T((d-1)\Delta x,(e+1)k)}{\Delta x^2}\right)dv\right|^2$$
$$+ 4\mu^2E\left|\int_{ek}^{(e+1)k}\Big(-T(dh,v) + T(dh,(e+1)k)\Big)dv\right|^2 + 5(\eta\epsilon+b)^2E\left|\int_{ek}^{(e+1)k}\Big(-I(dh,v) + I(dh,K)\Big)dv\right|^2$$
$$+ 5(\nu_1)^2\int_{ek}^{(e+1)k}E\left|\Big(-T(dh,v) + T(dh,ek)\Big)\right|^2dv,$$

$E|H(T)_d^e - H_d^e(T)|^2 \to 0$ as $(d,e) \to \infty$, so this scheme is consistent. Similarly, the consistency of the scheme for the Eqs. (10–12) can be proved.

Convergence

The convergence of the scheme is discussed in the mean square sense.

Theorem 5 *The stochastic implicit finite difference scheme given by Eqs. (9–12) is convergent in the mean square sense.*

$$E\left|T_d^e - T\right|^2 = E\left|(L_d^e)^{-1}(L_d^eT_d^e - L_d^eT)\right|^2,$$

Proof

as the scheme is consistent in the mean square sense i.e., $L_d^eT_d^e \to L_d^eT$ as $\Delta x \to 0, \Delta t \to 0$ and $(d\Delta x, e\Delta t,) \to (x,t)$,

$$E\left|(L_d^e)^{-1}(L_d^e T_d^e - L_d^e T)\right|^2 \to 0,$$

also, scheme is stable, then $(L_d^e)^{-1}$ is bounded. So, $E\left|T_d^e - T\right|^2 \to 0$. Hence proposed scheme for T is convergent in the mean square sense. A similar process can be used to show the convergence of the scheme.

Discussion

We are using a test problem to check the efficacy of our proposed schemes. The given system of equations has two equilibrium points $DFE = (s/\mu, 0, 0, 0)$ and other is $EE = (T_*, I_*, V_*, L_*)$ where $T_* = \frac{bc+c\mu_1+c\epsilon}{kN\epsilon(1-\eta)}$, $I_* = \frac{k_0Ns\epsilon(1-\eta)-bc\mu+c\mu\mu_1+c\mu\epsilon}{k_0N\epsilon(1-\eta)(\mu_1+(1-\eta)\epsilon)}$, $V_* = \frac{k_0Ns\epsilon(1-\eta)-bc\mu+c\mu\mu_1+c\mu\epsilon}{\delta k_0N(\mu_1+(1-\eta)\epsilon)}$, $L_* = \frac{k_0Ns\epsilon(\eta-1)+bc\mu+c\mu\mu_1+c\mu\epsilon}{ck_0((\eta-1)\epsilon-\mu_1)}$. The EE exists if $\frac{s}{\mu} > \frac{bc+c\mu_1+c\epsilon}{k_0N\epsilon(1-\eta)}$.

Consider the Eqs. (1–4) with following equations

$$T(x,0) = \begin{cases} 300x & \text{if } 0 \le x \le \frac{1}{2} \\ 300 - 300x & \text{if } \frac{1}{2} \le x \le 1. \end{cases} \tag{20}$$

$$I(x,0) = \begin{cases} 10x & \text{if } 0 \le x \le \frac{1}{2} \\ 10 - 10x & \text{if } \frac{1}{2} \le x \le 1. \end{cases} \tag{21}$$

$$V(x,0) = \begin{cases} 10x & \text{if } 0 \le x \le \frac{1}{2} \\ 10 - 10x & \text{if } \frac{1}{2} \le x \le 1. \end{cases} \tag{22}$$

$$Ł(x,0) = \begin{cases} 10x & \text{if } 0 \le x \le \frac{1}{2} \\ 10 - 10x & \text{if } \frac{1}{2} \le x \le 1. \end{cases} \tag{23}$$

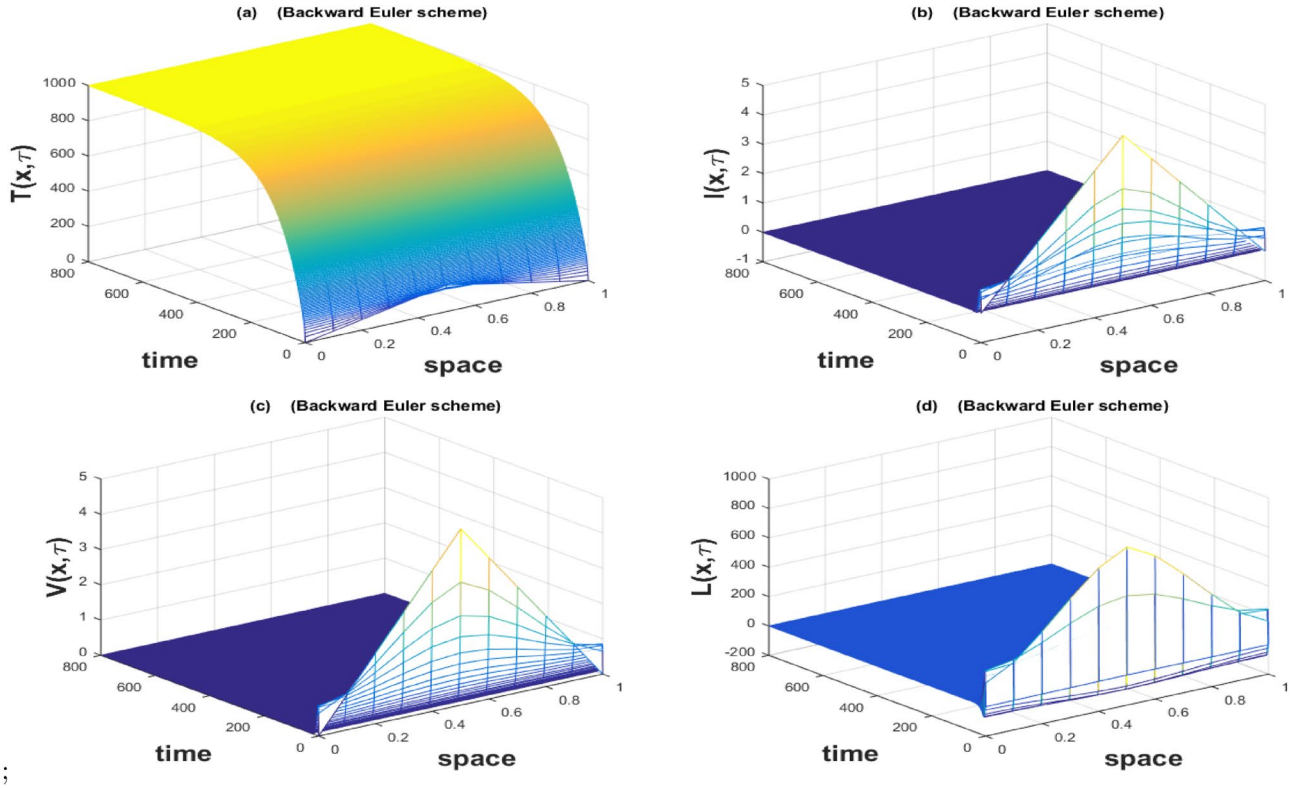
The simulations are drawn for the values in Table 1. The Fig. 1 is drawn by using the backward Euler scheme for the noise strength zero. It is seen from the graphical behavior that the density of contaminated $CD4^+$ T cells before pre-RT class and the virus density have gained negative values and it is not the behavior for the disease dynamics. Figure 2 is plotted by the proposed BE scheme for all noise strengths 0.02. The randomness behavior is observed for the state variable $T(x, t)$ and all other state converges to zero but have negative behavior as well. It is a disease-free equilibrium gained for zero and nonzero noise strength. The Fig. 3 is plotted by the proposed SIFD scheme with noise strength zero and state variable $T(x, t)$ is non-zero and all other state variables are zero. The DFE is successfully gained. The Fig. 4 is drawn for noise strength 0.02. The effect of the noise can be seen in the disease-free equilibrium and all the state variables possess positive behavior. The proposed stochastic IFD scheme is suitable for the solution of the HIV/AIDS model with disease-free equilibrium.

The given system of equations (1–4) has a coexistence equilibrium and it is given as $EE = (T_*, I_*, V_*, L_*)$. The EE is only exists when $\frac{s}{\mu} > \frac{bc+c\mu_1+c\epsilon}{kN\epsilon(1-\eta)}$ is satisfied. To attain this condition, we have chosen the value of $s = 100$. The physical behavior of endemic equilibrium is gained for the values of the parameters given above table. The Fig. 5 is constructed by the proposed SBE scheme with noise strength zero. The densities of susceptible $CD4^+$ T cells, pre-RT infected $CD4^+$ T cells, infected $CD4^+$ T cells, and virus density have positive behavior. The Fig. 6 is plotted for noise strength 0.02 and all densities have positive behavior except virus density. It has negative values for some points of the domain and it does not preserve the biological property. The Figs. 7 and 8 are drawn for zero and non-zero noise strength for all state variables. The endemic equilibrium has been successfully gained by the proposed stochastic IFD scheme. The T, V, I, and L have true behavior. The proposed SIFD scheme is consistent with the given system of equations. The Figs. 9 and 10 are drawn by the proposed IFD scheme with noise strength 0.025 by escalating the value of the b from 0 to $\frac{1}{10}$ and it is noticed that the number of $CD4^+$ T cells increases and the number of virus decreases. The Figs. 11 and 12 are plotted for the proposed stochastic IFD scheme with noise strength 0.025. The effect of the parameter η is observed by increasing its value from 0.7 to 0.8 and noticed that the densities of T, I increase and V, L decrease respectively.

The HIV models are disease dynamics and necessarily the solutions must be positive. We have employed two techniques for the numerical solutions of the underlying mode. One technique fails to preserve the positive behavior while the other preserves the positivity and converges towards the steady states. One of the most compelling reasons to consider this model with a numerical scheme is to construct and apply the scheme in a way that yields positive solutions. As the underlying model is disease dynamics disease can never be negative. So

k_0	c	b	η	μ	ϵ	δ	μ_1
0.000024	2.4	0.1	0.8	0.01	0.4	0.26	0.015

Table 1. Values of parameters.



;

Figure 1. 3D simulations of T, I, V, L, with $r_i = 0.8, \sigma_i = 0, i = 1, 2, 3, 4, s = 10$.

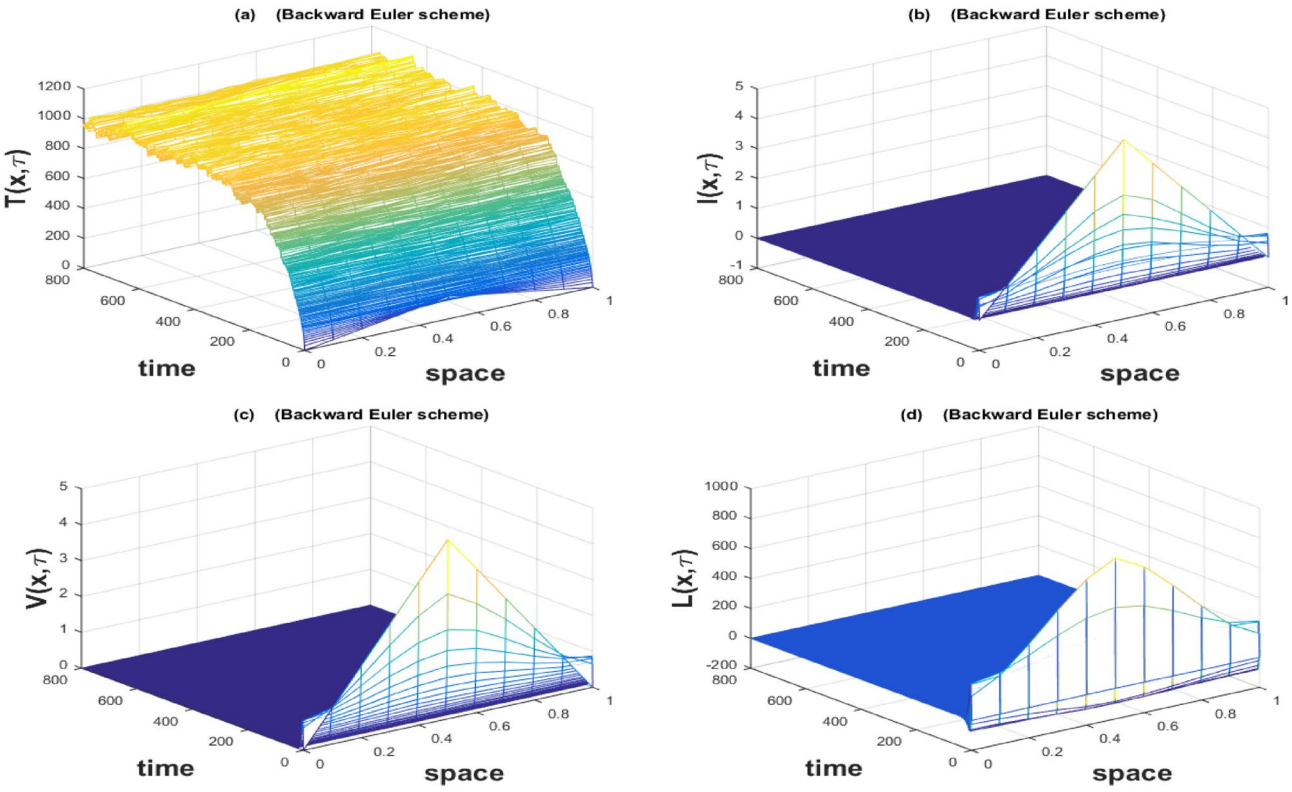


Figure 2. 3D simulations of T, I, V, L, with $r_i = 0.8, \sigma_i = 0.02, i = 1, 2, 3, 4, s = 10$.

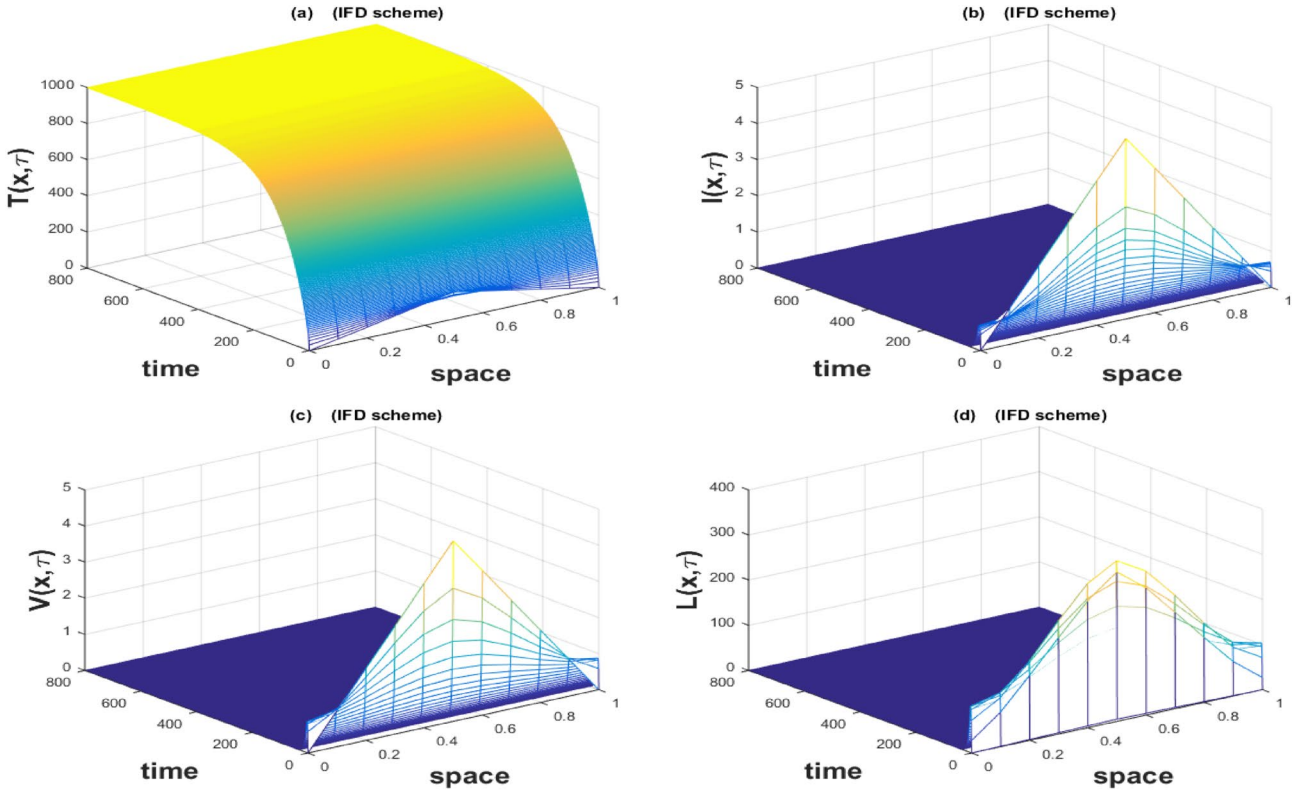


Figure 3. 3D simulations of T, I, V, L, with $r_i = 0.8, \sigma_i = 0., i = 1, 2, 3, 4, s = 10$.

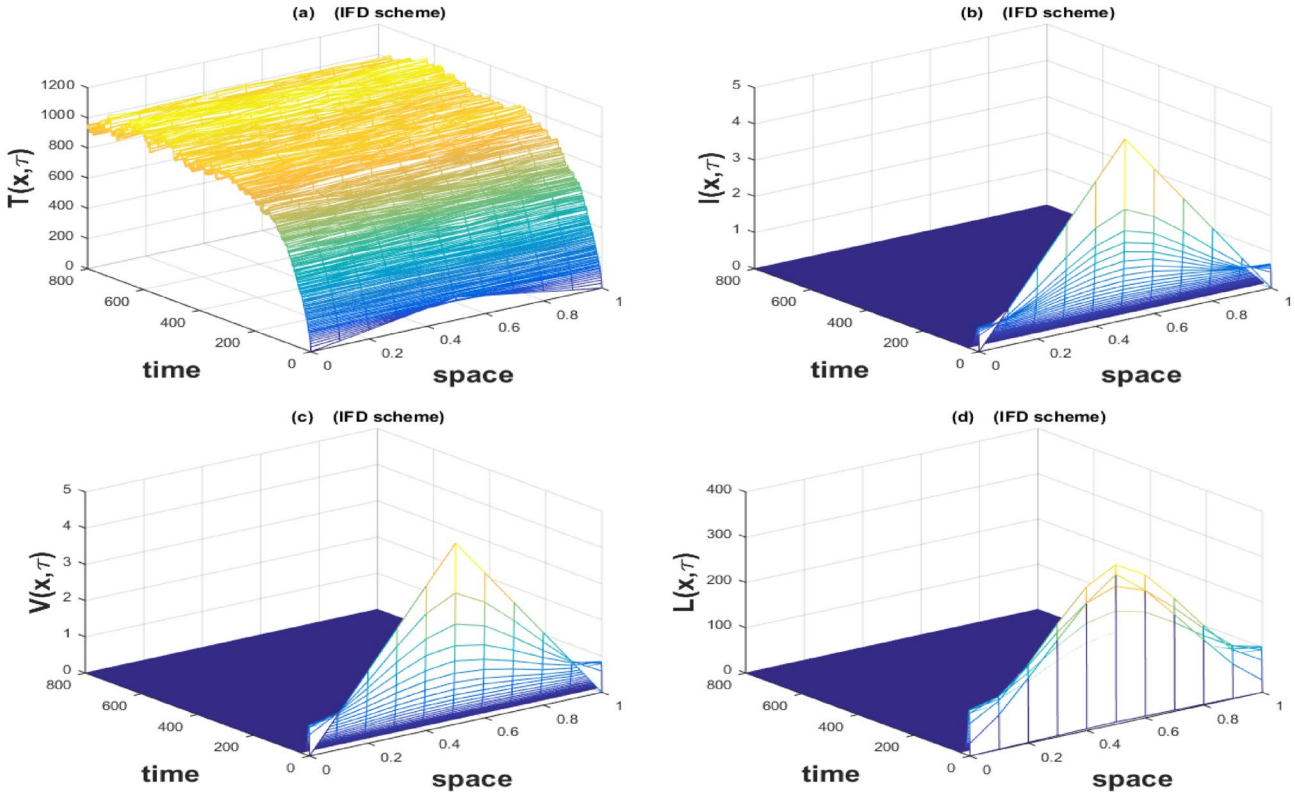


Figure 4. 3D simulations of T, I, V, L, with $r_i = 0.8, \sigma_i = 0.02, i = 1, 2, 3, 4, s = 10$.

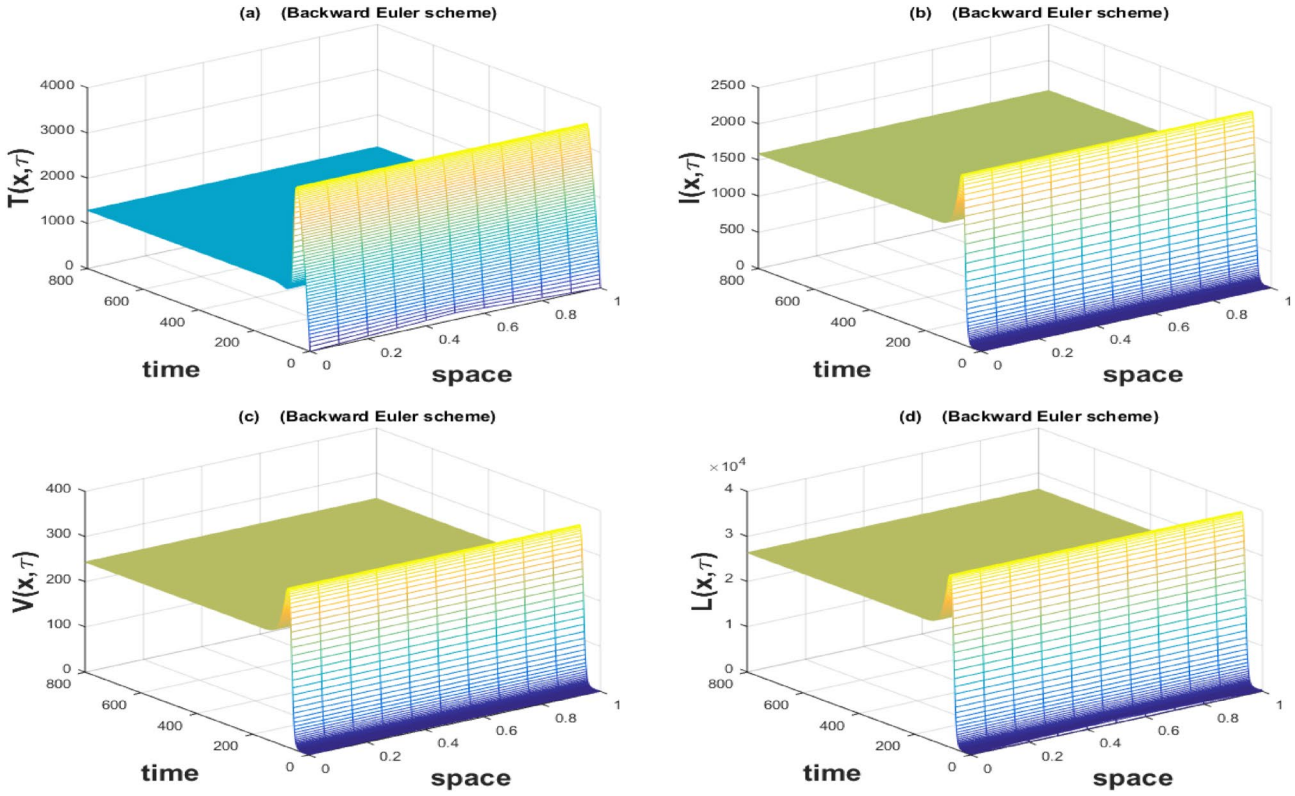


Figure 5. 3D simulations of T, I, V, L, with $r_i = 0.8, \sigma_i = 0, i = 1, 2, 3, 4, s = 100$.

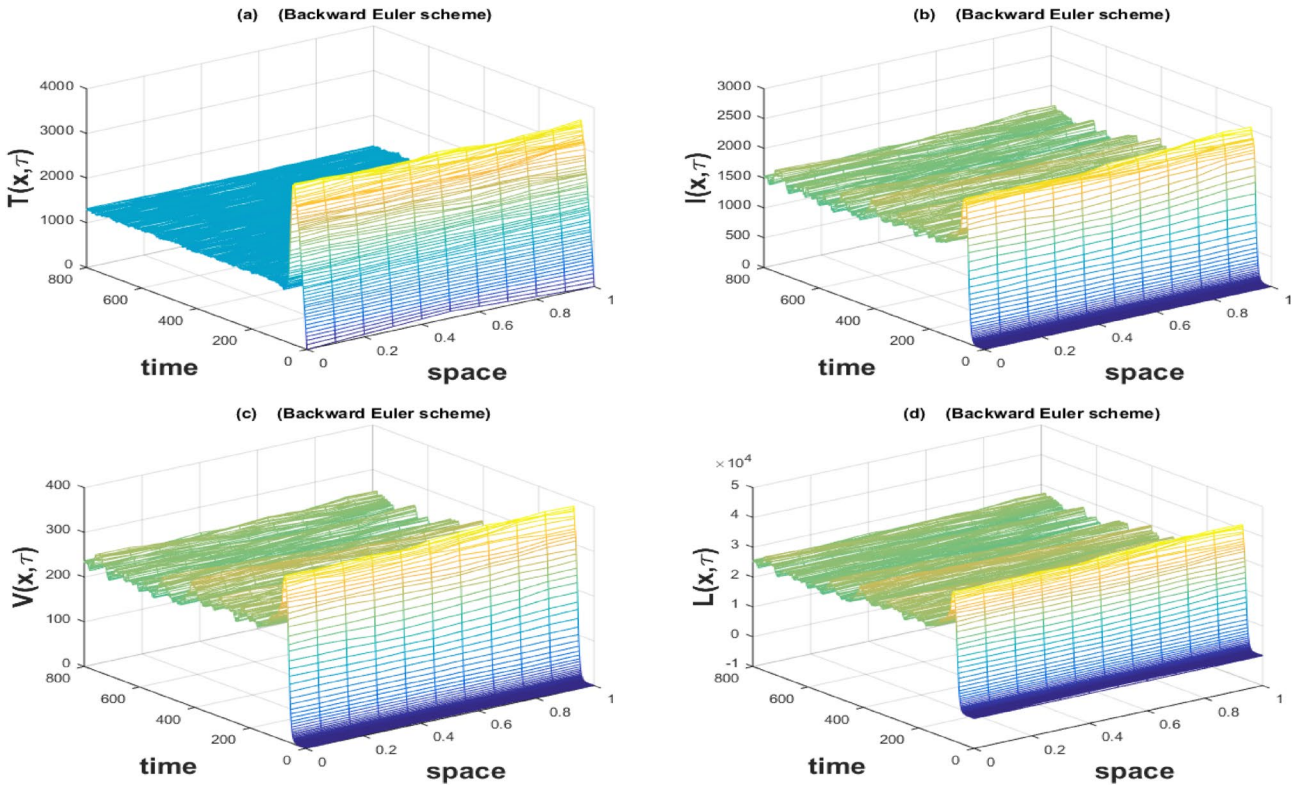


Figure 6. 3D simulations of T, I, V, L, with $r_i = 0.8, \sigma_i = 0.02, i = 1, 2, 3, 4, s = 100$.

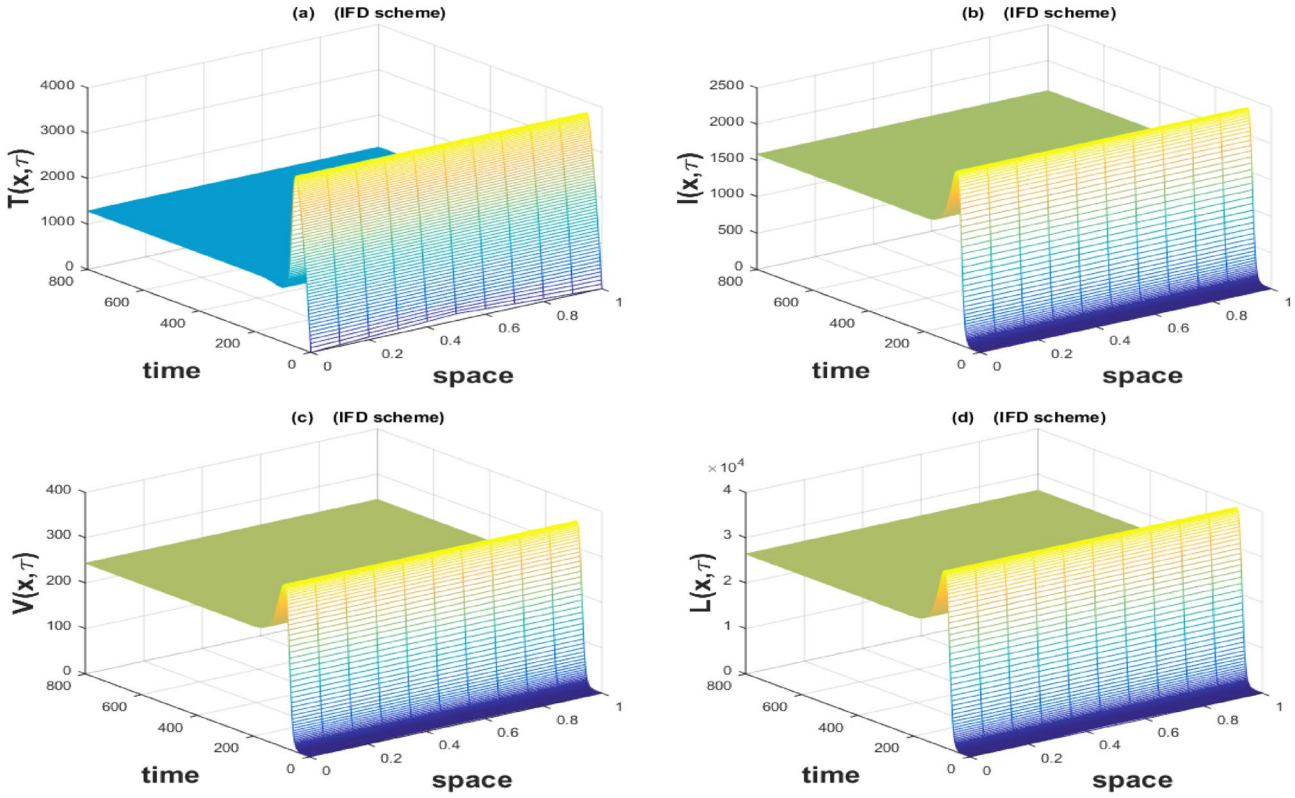


Figure 7. 3D simulations of T, I, V, L, with $r_i = 0.8, \sigma_i = 0, i = 1, 2, 3, 4, s = 100$.

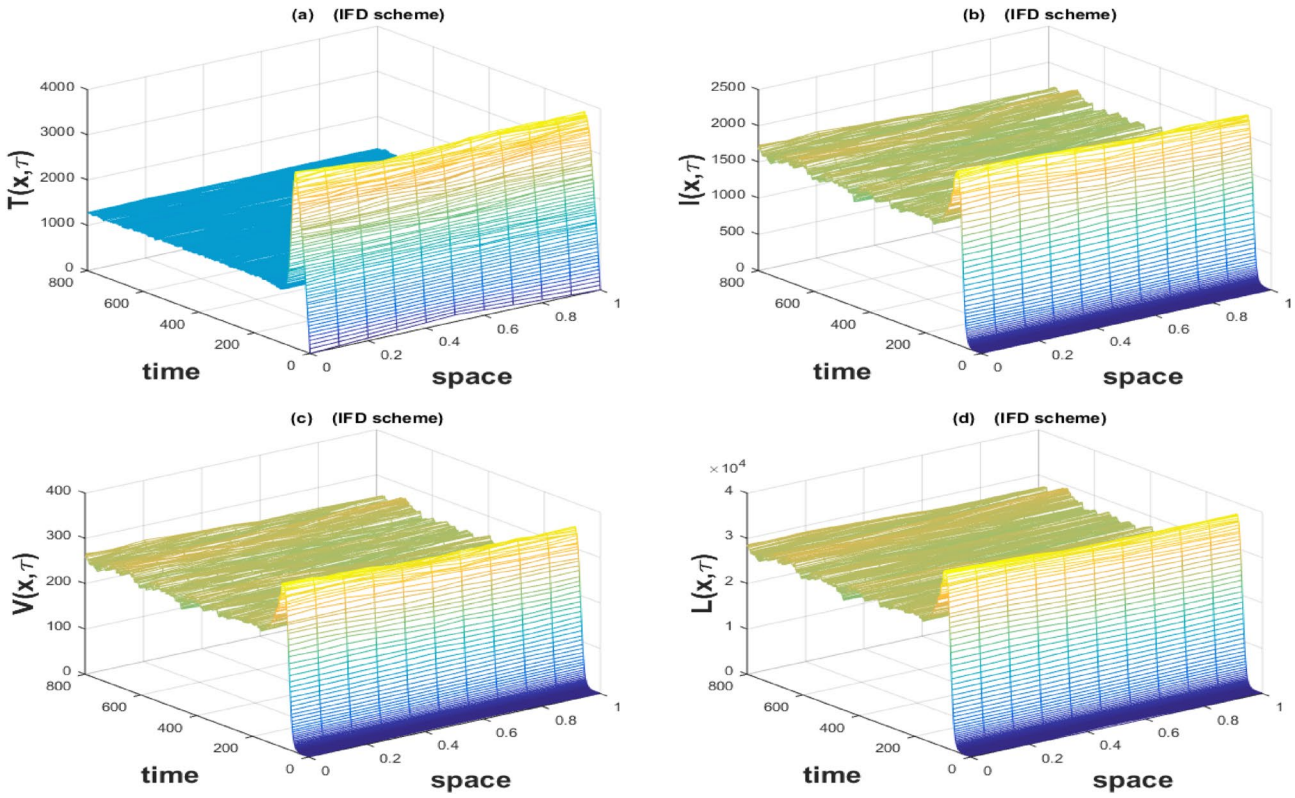


Figure 8. 3D simulations of T, I, V, L, with $r_i = 0.8, \sigma_i = 0.02, i = 1, 2, 3, 4, s = 100$.

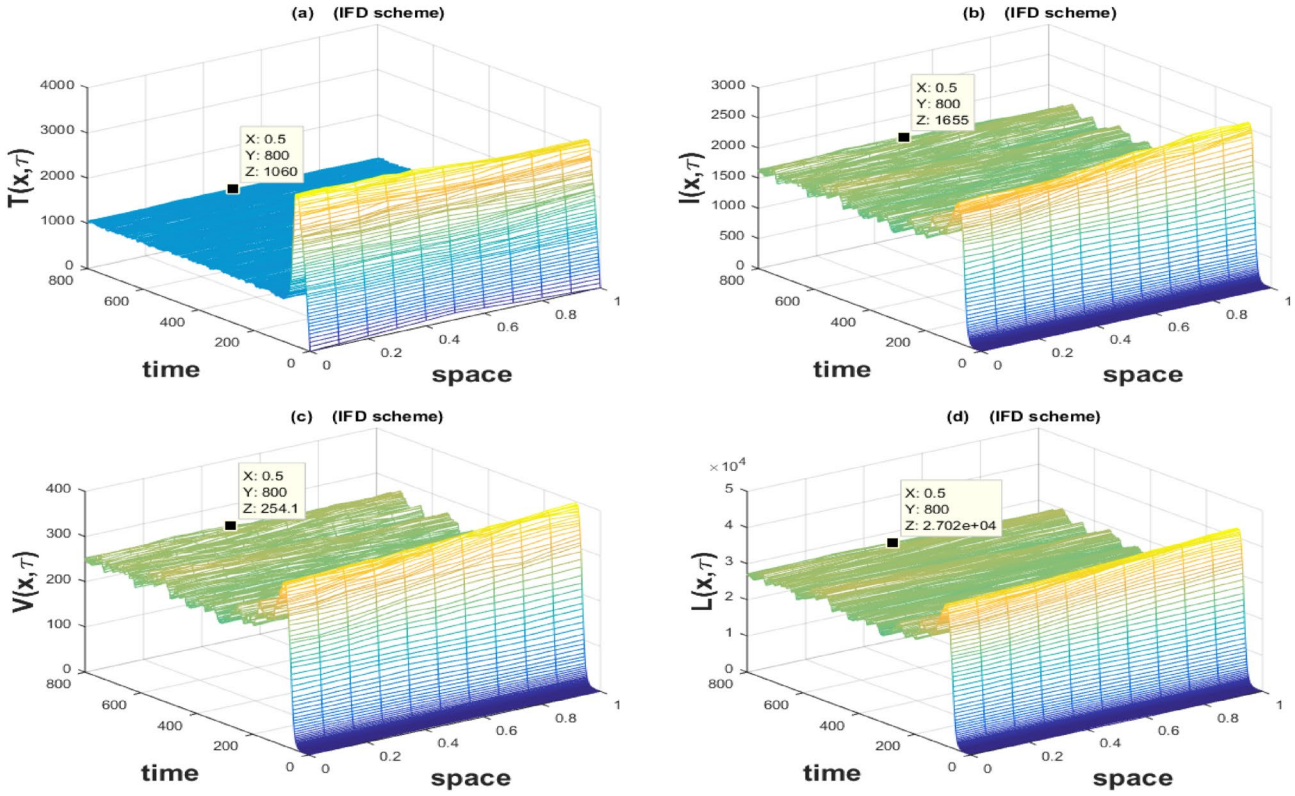


Figure 9. 3D simulations of T, I, V, L, with $r_i = 0.8, \sigma_i = 0.025, i = 1, 2, 3, 4, b = 0, s = 100$.

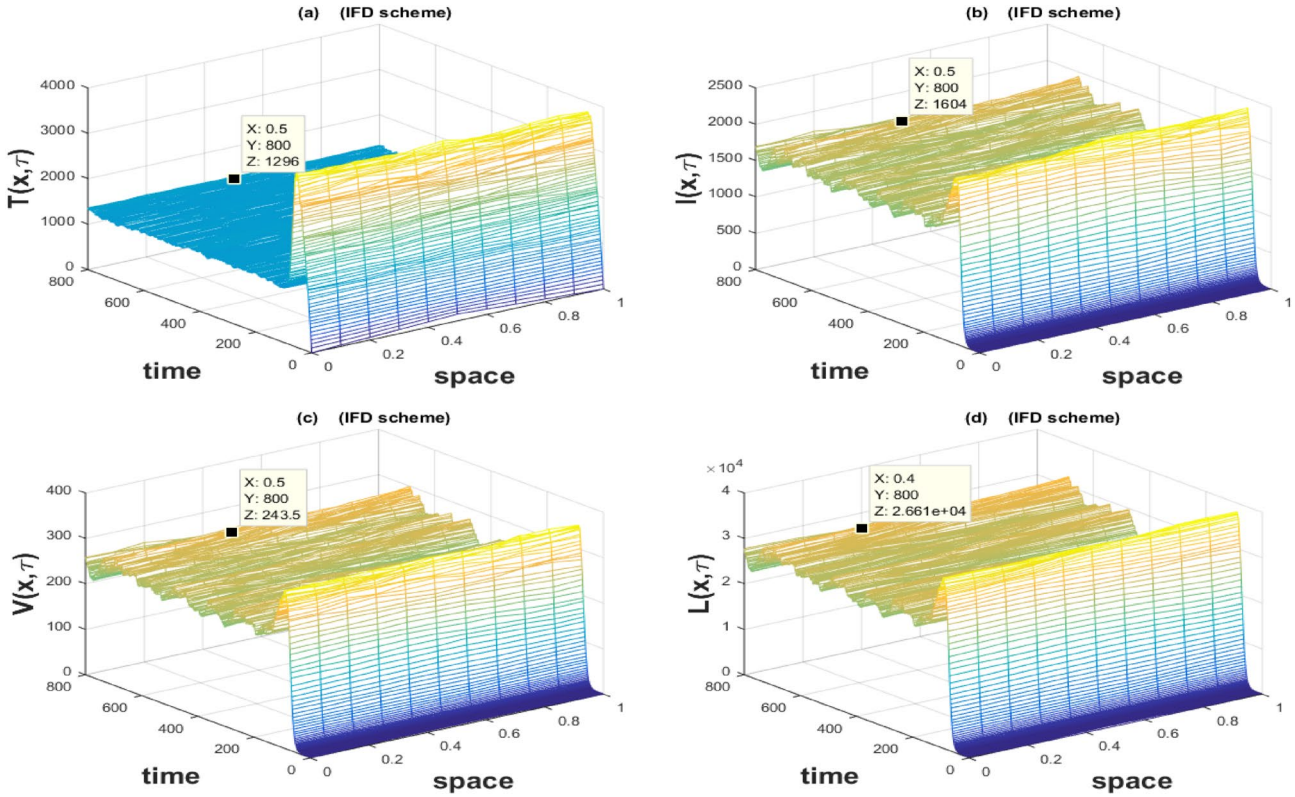


Figure 10. 3D simulations of T, I, V, L, with $r_i = 0.8, \sigma_i = 0.025, i = 1, 2, 3, 4, b = 0.1, s = 100$.

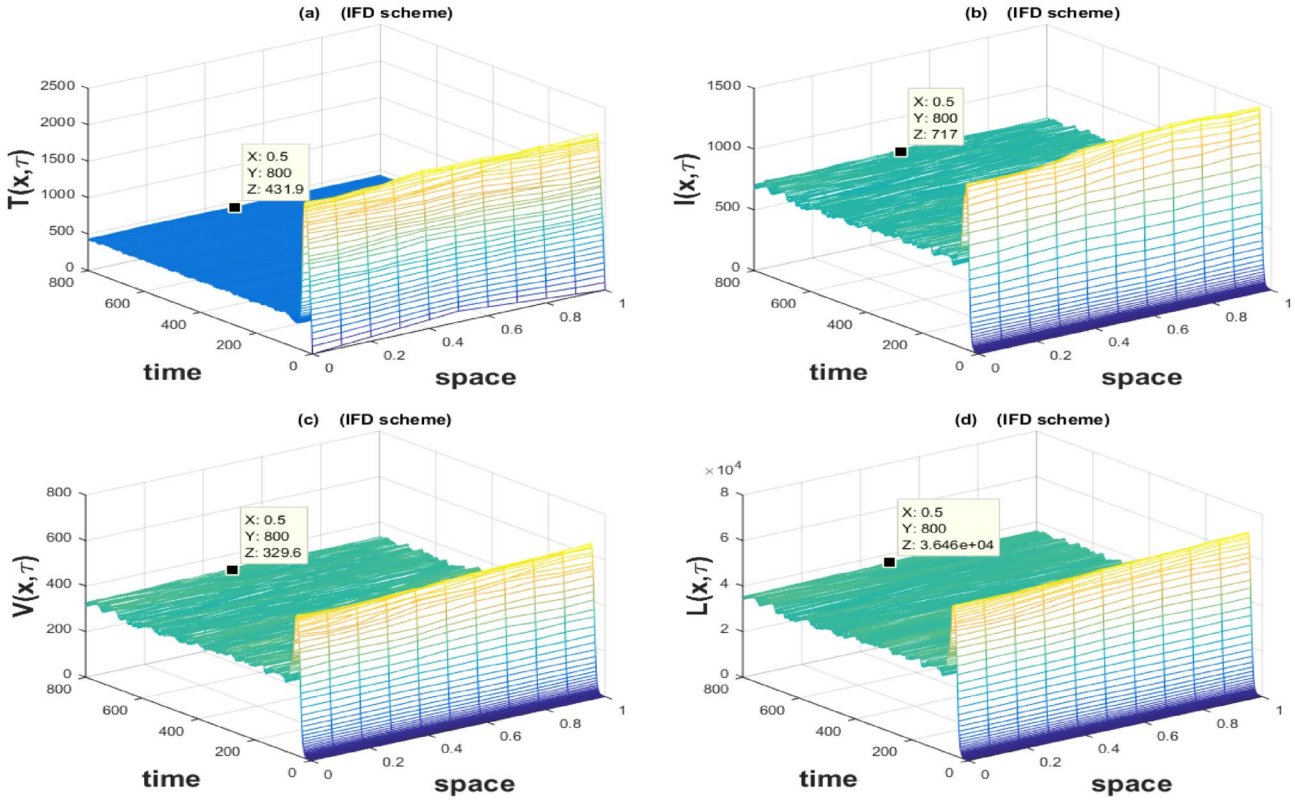


Figure 11. 3D simulations of T, I, V, L, with $r_i = 0.8, \eta = 0.7, \sigma_i = 0.025, i = 1, 2, 3, 4, s = 100$.

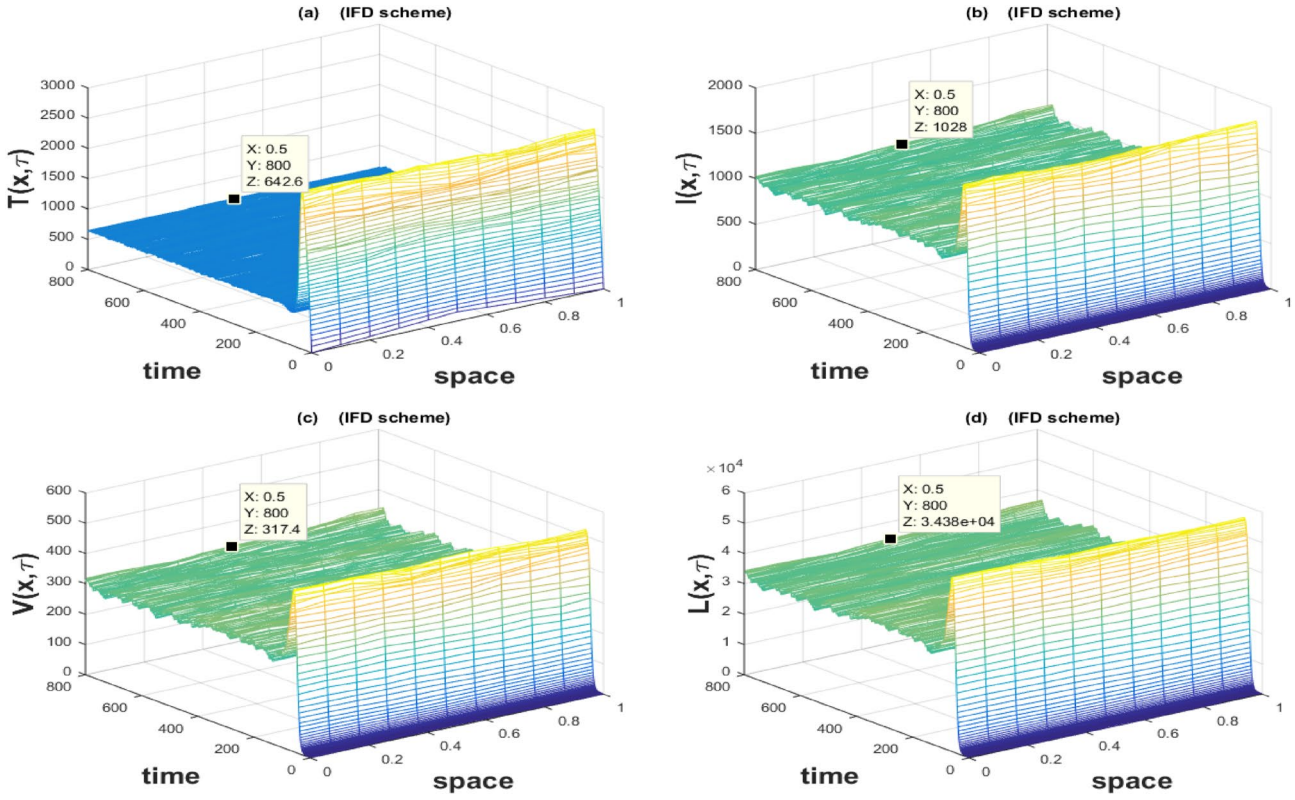


Figure 12. 3D simulations of T, I, V, L, with $r_i = 0.8, \sigma_i = 0.025, i = 1, 2, 3, 4, \eta = 0.8, s = 100$.

its solution must preserve the positivity. Such solutions are preferred which preserve the positivity and bounded behavior for the whole domain. The results of the stochastic implicit finite difference scheme are aligned with the actual steady states which are the positive steady states. The disease dynamics have random behavior. So, it is quite better to consider the continuous model with a random effect. Such random behavior is observed in every physical phenomenon at a certain level. So we incorporate diffusion as well as random behavior in the underlying model.

Conclusion

In this article, the stochastic HIV/AIDS model with drug therapy is analyzed by two proposed numerical schemes. HIV/AIDS breaks up the immunity of susceptible patients and produces different disorders in the body. The classical models fail to predict the true behavior of the disease dynamics. It is more suitable to consider the classical model under the influence of some random process. So, we considered the stochastic version of the HIV/AIDS model for this study. The linear stability and consistency analysis of schemes is carried out. The underlying model has two equilibria, one is disease and the other is endemic equilibrium. The proposed SBE scheme is used to gain the numerical solution of the given model. The simulations are drawn for the zero and non-zero noise strength. The graphical behavior of the solutions by the proposed stochastic SBEs scheme showed negative behavior and it is meaningless in biological nature. The graphical behavior of the proposed SIFD scheme showed convergent and positive behavior for the specified parameter values. The simulations by the proposed SIFD scheme are drawn for the various values of the noise strength. The effect of various values of parameters on the solutions is also discussed. These findings will aid researchers in their consideration of the random effect on continuous systems. In the future, such analysis and numerical solution can be extended to higher dimensional stochastic PDEs and fractional stochastic PDEs related to the disease and population dynamics.

Data availability

Data will be provided by corresponding author on reasonable request.

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Author contributions

N.A. and M.W.Y. and S.M.A. and A.A. and A.R. wrote the main manuscript text and M.R. and M.A. and M.K.H. prepared Figs. 1, 2, 3, 4, 5, 6, 7 and 8. All authors reviewed the manuscript.

Competing interests

The authors declare no competing interests.

Additional information

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