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Sliding mode control-based analysis of a time-delay HIV dynamic model

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Abstract. In this paper, a sliding mode control strategy is developed for a biologically realistic fourth-order HIV infection model with explicit time delay. The model captures the effects of intracellular delay and virus production rate on the dynamics of healthy CD4+ T cells, infected cells, and free viral particles. A sliding mode controller is designed for all system states, and a rigorous finite-time convergence analysis is provided using Lyapunov theory. It is analytically shown that the proposed control law guarantees convergence of the system trajectories to the sliding surface in finite time, despite the presence of time delays and parameter uncertainties. Numerical simulations conducted under various delay values and virus production rates demonstrate that increasing delay and viral replication adversely affect system stability and may lead to divergence beyond a critical delay threshold. In contrast, the proposed controller effectively suppresses oscillations and enforces rapid convergence of the system states. These results confirm the robustness and effectiveness of the proposed sliding mode control framework for delayed HIV dynamics.

Keywords: Stability, time delay, sliding mode control, HIV infection.
AMS Subject Classification 2010: 93D15, 93B35,34K37,92C60.

1 Introduction

The Human Immunodeficiency Virus (HIV) remains a major global health challenge, motivating continuous research into its dynamic behavior and effective treatment strategies. The HIV primarily targets CD4+ T-cells, progressively weakening the immune system and potentially leading to Acquired Immunodeficiency Syndrome (AIDS) [15]. Understanding the interaction between viral dynamics, immune response, and therapeutic interventions is essential for designing effective control-oriented treatment strategies.

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Mathematical modeling has long played a central role in the study of HIV infection dynamics [5, 12, 20, 26]. Classical models based on ordinary differential equations (ODEs) have provided valuable insights into viral replication, immune system interaction, and antiretroviral therapy effects [7, 21]. However, such models often neglect important biological features, particularly intracellular delays associated with viral replication and immune response activation. As a result, delay-free models may fail to accurately capture transient dynamics, oscillatory behavior, and instability phenomena observed in real HIV progression [14, 17, 25].

To address these limitations, delay differential equation (DDE) models have been introduced to explicitly account for time delays arising from intracellular processes and immune response mechanisms. Previous studies have shown that incorporating delays can significantly alter system stability and may induce Hopf bifurcations, oscillations, or loss of stability beyond critical delay values [2, 24]. These findings highlight the destabilizing role of excessive intracellular delay and underscore the necessity of robust analytical and control tools for delayed HIV dynamics.

In parallel, control theory has emerged as a powerful framework for regulating HIV infection dynamics. Among various approaches, sliding mode control (SMC) has attracted particular attention due to its robustness against model uncertainties, external disturbances, and parameter variations [10,16,19,22,23]. Several studies have successfully applied SMC and related nonlinear control techniques to HIV models, demonstrating improved regulation of viral load and CD4+ T-cell populations [[3, 13]. Nevertheless, most existing control-based studies focus on low-order or delay-free HIV models, while the combined effects of high-order nonlinear dynamics and explicit time delays remain largely unexplored within the sliding mode control framework.

Despite recent advances in delayed HIV modeling and control design, there is a notable gap in the literature regarding the application of sliding mode control to biologically realistic high-order HIV models with explicit time delays. In particular, finite-time convergence properties, which are crucial for rapid stabilization and robustness in biomedical applications, have not been rigorously established for delayed high-order HIV systems [1, 18].

Motivated by this gap, the present study investigates the control of HIV infection dynamics using a fourth-order delayed model and a tailored sliding mode control strategy. The biological model structure is adopted from validated delayed HIV frameworks [4, 11, 20], while the primary contribution of this work lies in the systematic design of a state-feedback sliding mode controller and the rigorous Lyapunov-based proof of finite-time convergence in the presence of time delays and parametric uncertainties. Furthermore, the analysis reveals the existence of a critical delay threshold beyond which the uncontrolled system loses stability, providing insight into the destabilizing impact of intracellular delays.

The remainder of this paper is organized as follows. Section 2 presents the mathematical formulation of the delayed HIV model. Section 3 describes the sliding mode control design and finite-time stability analysis. Section 4 provides numerical simulations to validate the theoretical results under various delay and virus

2 The ODE model

Many efforts have been made to mathematically depict the behavior of the HIV virus, leading to the development of diverse models. The standard differential equation model of the HIV virus consists of three main components: uninfected cells, infected cells, and free viral particles.

These models generally describe the interactions between these three components and examine how the population of each component changes over time. In these models, uninfected cells (healthy CD4+ T cells) are infected by the virus and transformed into infected cells. The infected cells then produce new viral particles, which can infect more healthy cells. This cycle repeats, leading to an increase in viral load and a decrease in the number of healthy CD4+ T cells. This modeling without considering delay is as follows [4]:

$$\begin{cases} \frac{dT}{dt} = s - kV(t)T(t) - d_T T(t), \\ \frac{dI}{dt} = kV(t)T(t) - \rho I(t), \\ \frac{dV}{dt} = N\rho I(t) - d_V V(t). \end{cases} \tag{1}$$

Algorithm 1. Computational Framework for Simulating HIV-1 Infection Kinetics

1. **Input:** Parameter set $\Theta = \{s, k, d_1, \rho, N\}$.
2. **Output:** State trajectory $\mathbf{X}(t) = [T(t), I(t), V(t)]^T$.
3. Initialize $\mathbf{X} = \mathbf{X}_0$ and $t = 0$. Set trajectory list empty.
4. **While** $t \leq t_{\text{end}}$ **do**:
   (a) Compute the vector field
   $$\mathscr{F} = \begin{bmatrix} s - kVT - d_1 T \\ kVT - \rho I \\ N\rho I - d_1 V \end{bmatrix}.$$
   (b) Update the state
   $$\mathbf{X} \leftarrow \mathbf{X} + \Delta t\, \mathscr{F}.$$
   (c) Append $\mathbf{X}$ to the trajectory list.
   (d) Update $t \leftarrow t + \Delta t$.
5. **Return:** the full trajectory list.

Mathematical models of HIV are not only used to better understand the dynamics of the virus and its interaction with the immune system but are also employed to evaluate the effectiveness of various treatments, such as antiretroviral therapy (ART). These models help researchers design optimal treatment strategies that can reduce viral load and maintain CD4+ T cell counts within a healthy range.

However, standard differential equation models are sometimes unable to fully capture the complexities of HIV dynamics. For this reason, researchers have turned to developing more advanced models that include time delays, fractional-order derivatives, and advanced control techniques. These more advanced models contribute to a better and more precise understanding of the virus's behavior and the immune system's response, paving the way for the development of more effective treatments.In the following, we will delve into the nonlinear and delayed model put forth by Seryoastav and his colleagues.

Subsequently, we will derive the suggested approach for the interaction between healthy cells and virus-free particles, based on this model. The model employs delayed differential equations and is outlined as follows [4, 11, 20]:

$$\dot{\mathbf{X}}(t) = \mathscr{F}\big(\mathbf{X}(t), \mathbf{X}(t-\tau), u(t); \Theta\big),\ \mathbf{X}(t) = \begin{bmatrix} T(t) \\ I_1(t) \\ I_2(t) \\ V(t) \end{bmatrix},\ \Theta = \{s, k, d_T, r, T_{\max}, \alpha, \beta, \gamma, \rho, N, d_V\},$$

$$\mathscr{F}\big(\mathbf{X}, \mathbf{X}_\tau, u; \Theta\big) = \begin{bmatrix} s - kVT - d_T T + rT\left(1 - \dfrac{T}{T_{\max}}\right) + (u_1\alpha + \beta) I_1 \\ kV_\tau T_\tau - (\gamma + \alpha + \beta) I_1 \\ (1-u_1)\,\alpha I_1 - \rho I_2 \\ N\rho I_2 - d_V V \end{bmatrix},\ \mathbf{X}_\tau = \begin{bmatrix} T_\tau \\ I_{1,\tau} \\ I_{2,\tau} \\ V_\tau \end{bmatrix} = \begin{bmatrix} T(t-\tau) \\ I_1(t-\tau) \\ I_2(t-\tau) \\ V(t-\tau) \end{bmatrix}. \tag{2}$$

The equations presented in the Eq. (2) depict various stages of HIV infection dynamics within the body. Each equation models a critical aspect of disease progression, providing insights into the interactions between healthy and infected cells, as well as the viral load. The first equation, $T(t)$, represents the rate of change of healthy CD4+ T-cells. These cells increase through a production rate s and decrease due to infection by the virus at rate $kV(t)T(t)$. The natural death rate of CD4+ T-cells is accounted for by the coefficient d_T. Additionally, the repair or proliferation of healthy cells is modeled at rate $rT(t)(1-\frac{T(t)}{T_{\max}})$, while the effect of antiviral drugs is incorporated through the term $(u_1\alpha+\beta)I_1(t)$, which influences the transition of cells to the infected phase.

The second equation, $I_1(t)$, describes the dynamics of initially infected cells. Healthy CD4+ T-cells are infected after a time delay τ at rate $kV(t-\tau)T(t-\tau)$. These initially infected cells either die or transition to the next disease phase at rate $(\gamma+\alpha+\beta)I_1(t)$. This equation captures the early stages of infection, where the virus begins to replicate within the host cells.

The third equation, $I_2(t)$, represents a more advanced stage of infection. Here, some of the initially infected cells I_1 transition to the next disease phase with probability $(1-u_1)\alpha$ and become I_2. These advanced infected cells die at rate $\rho I_2(t)$. This stage reflects the progression of the disease as the virus continues to replicate and spread within the body.

The fourth equation, $V(t)$, models the dynamics of free viral particles in the blood. The virus is produced from infected cells at rate $N\rho I_2(t)$ and is cleared from the system at rate $d_V V(t)$. This equation provides a measure of the viral load, which is a key indicator of disease progression and the effectiveness of treatment.

The model captures the initial stage of infection, known as the acute phase, where the virus begins infecting healthy CD4+ T-cells. As the infection progresses to the chronic phase, a dynamic balance is established between the immune system's efforts to control the infection and the virus's ability to replicate. Over time, the disease may advance to the AIDS stage, characterized by a severe decrease in CD4+ T-cells and an increase in viral load, making the body susceptible to opportunistic infections. By incorporating time delays in viral replication and the effects of drug treatment, this model offers a more accurate representation of the real behavior of HIV infection. It provides a valuable framework for understanding the complex dynamics of the disease and can be used to design more effective treatment strategies. The inclusion of parameters such as α, β, and ρ allows for a detailed analysis of the transitions

Table 1: System parameters and their corresponding values used in the model (2)

Parameter	Description	Value
s	Growth rate of $CD4^+T$ cells in bone marrow	5 $day^{-1}(mm^{-3})$
r	Proliferation rate of $CD4^+T$ cells	0.8 day^{-1}
d_T	Death rate of healthy $CD4^+T$ cells	0.01 day^{-1}
k	Infection rate of $CD4^+T$ cells by the virus	0.00002 $day^{-1}(mm^{-3})$
T_{max}	Maximum $CD4^+T$ cell count	1300 mm^{-3}
α	Transition rate from Pre-RT to Post-RT	0.4 day^{-1}
β	Return rate of infected cells to non-infected	0.05 day^{-1}
γ	Death rate of infected cells	0.015 day^{-1}
ρ	Death rate of activated infected cells	0.24 day^{-1}
d_V	Virus clearance rate	2.4 day^{-1}
N	Virus production rate per infected $CD4^+T$ cell	Variable
u_1	Killing efficiency of immune response	0.1

between different stages of infection, offering insights into potential therapeutic targets and interventions [4, 20]. The model parameters and corresponding values used in system (2) are given in Table 1. Based on biological considerations and using the theory of delay differential systems, specific initial conditions have been assumed for the model. These conditions ensure the existence of a unique solution [8, 20]. In the following, the implementation of the sliding mode control method on system (2) is considered.

3 Sliding mode control for time delay modeling in AIDS

The utilization of sliding mode control for modeling the delayed AIDS disease has gained significant popularity among researchers in recent times. This approach is preferred due to its reduced sensitivity to disturbances and parameter variations. Unlike other controllers, it does not necessitate an accurate system model. Sliding mode control is highly regarded in various industries due to its simplicity in design and its capability to mitigate the impact of external disturbances and uncertainties in the model. It is a robust nonlinear controller that effectively handles both structural and non-structural uncertainties within the system.

The fundamental principle of sliding mode control involves defining a stable sliding surface toward which the system states converge, typically to the equilibrium point, often the origin [6, 7]. In two-dimensional systems, the sliding surface is represented as a one-dimensional line, whereas in three-dimensional systems, it is depicted as a plane. Designing this controller requires careful consideration of two crucial aspects. Firstly, the sliding surface is designed based on the desired performance specifications of the system. Secondly, the control law is derived to ensure that the system states, regardless of being above or below the sliding surface, are attracted toward it. The equation of the sliding surface for a second-order system can be written as a function of error derivatives and error coefficients. Given that the system under study in this paper is of fourth order, the sliding surface equation will be of third order

and can be written as follows:

$$S = \mathbf{w}^{\mathrm{T}}\mathbf{x}, \tag{3}$$

where

$$\mathbf{w} = \begin{bmatrix} h^3 \\ h^2 \\ h \\ 1 \end{bmatrix}, \quad \mathbf{x} = \begin{bmatrix} e \\ \dot{e} \\ \ddot{e} \\ \dddot{e} \end{bmatrix}. \tag{4}$$

In Eq. (3), h is a parameter representing error coefficients. This error is essentially the one we aim to reduce to zero. Therefore

$$e = T - T_d. \tag{5}$$

Our desired state is equal to zero, that is, we assume $T_d = 0$:

$$\begin{aligned} S = & -k\dot{V}\dot{T} - kV\ddot{T} - kN\rho I_2\dot{T} - kTN\rho\dot{I}_2 + kd_V V\dot{T} + kd_V T\dot{V} - \ddot{T}d_T + \ddot{T}r \\ & - \frac{2r[\ddot{T}T + \dot{T}^2]}{T_{max}} + u_1\alpha\ddot{I}_1 + \beta\ddot{I}_1 + h[-kV\dot{T} - kT(N\rho I_2 - d_V V) \\ & - \dot{T}d_T + \dot{T}r - \frac{2r\dot{T}T}{T_{max}} + u_1\alpha\dot{I}_1 + \beta\dot{I}_1] + h^2\dot{T} + h^3 T. \end{aligned} \tag{6}$$

In order to derive the control law, we take the derivative of the sliding surface and set it to zero. Hence, by considering Eq. (3) we can express the relationship for system (2) as follows:

$$\begin{aligned} \dot{S} = & -k\left(\ddot{V}\dot{T} + \dot{V}\ddot{T} + \dot{V}\ddot{T} + V\dddot{T}\right) \\ & - kN\rho\left(\dot{I}_2\dot{T} + I_2\ddot{T} + \dot{I}_2\dot{T} + T\ddot{I}_2\right) \\ & + kd_V\left(\ddot{V}\dot{T} + V\ddot{T} + \dot{T}\dot{V} + T\ddot{V}\right) \\ & + \dddot{T}(r - d_T) + \frac{2r}{T_{\max}}\left(\dddot{T}T + 3\dot{T}\ddot{T}\right) \\ & + u_1\alpha\dddot{I}_1(t-\tau) + \beta\dddot{I}_1(t-\tau) \\ & - h\Big[-k\left(\ddot{V}\dot{T} + V\ddot{T}\right) - kN\rho\left(\dot{T}I_2 + T\dot{I}_2\right) \\ & + kd_V\left(\dot{T}V + T\dot{V}\right) - \ddot{T}(d_T - r) \\ & - \frac{2r}{T_{\max}}\left(\ddot{T}T + \dot{T}^2\right) + u_1\alpha\ddot{I}_1(t-\tau) + \beta\ddot{I}_1(t-\tau)\Big] \\ & + h^2\ddot{T} + h^3\dot{T}, \end{aligned} \tag{7}$$

where

$$\ddot{I}_1 = k\dot{V}(t-\tau)T(t-\tau) + KV(t-\tau)\dot{T}(t-\tau) - \gamma\dot{I}_1 - \alpha\dot{I}_1 - \beta\dot{I}_1, \tag{8}$$

$$\dddot{I}_1 = k\ddot{V}(t-\tau)T(t-\tau) + 2k\dot{V}(t-\tau)\dot{T}(t-\tau) + kV(t-\tau)\ddot{T}(t-\tau) - \gamma\ddot{I}_1 - \alpha\ddot{I}_1 - \beta\ddot{I}_1. \tag{9}$$

Based on the control law of system states, at any point in an n-dimensional space, the system tends to converge towards the sliding surface. Once the system states reach the sliding surface, they must remain

on it for the system to maintain stability. Hence, from Eq. (7), it can be inferred that the value of s should be zero ($\dot{S}=0$) for the system to exponentially converge towards the equilibrium point. In simpler terms, the control law needs to satisfy the condition of $S=0$. According to Eq. (7), the first part of the control law is expressed as follows:

$$u_{eq} = -\dot{S}. \tag{10}$$

Moreover, to ensure the stability of the sliding surface, we define the switching control u as follows:

$$u_{sw} = -hsign(S), \tag{11}$$

$$u = u_{eq} + u_{sw}, \tag{12}$$

where $h = max|E(t)| + \eta$, and η is a positive number and the sign function is defined as $\text{sign}[.] : \mathbb{R}^m \to \mathbb{R}^m$:

$$\text{sign}(S) = \begin{cases} 1, & S > 0, \\ 0, & S = 0, \\ -1, & S < 0. \end{cases}$$

Remark 1. *The control gain h must be chosen sufficiently large to dominate model uncertainties and satisfy the sliding condition. In simulations, h is selected to satisfy this condition while avoiding excessive chattering.*

The sliding mode control of system (2) has been designed in the MATLAB/Simulink environment. A MATLAB Function block is used to compute state derivatives through nonlinear differential equations. Initial conditions and system parameters (as listed in Table 1) are defined using constant and integrator blocks. All components are functionally connected within the control structure of system (2). By achieving control, the CD4+ T-cells population in the treatment model (2) is predetermined, and the number of HIV viruses, referred to as V, is also regulated. The control of system (2) is carried out using a sliding mode approach in the Simulink environment.

Theorem 1. *Assuming the sliding mode control law that is formulated as Eq. (12), the tracking error will converge to zero within a finite time.*

Proof. Consider the following positive definite function:

$$V = 0.5S^2. \tag{13}$$

By taking the time derivative of both sides of Eq. (13) and substituting $\dot{S}$, we have

$$\begin{aligned} \dot{V} = S\dot{S} = S[& -k\left(\ddot{V}\dot{T} + \dot{V}\ddot{T} + \dot{V}\dddot{T} + V\ddot{T}\right) \\ & - kN\rho\left(\dot{I}_2\dot{T} + I_2\ddot{T} + \dot{I}_2\dot{T} + T\dot{I}_2\right) \\ & + kd_V\left(\ddot{V}\dot{T} + V\ddot{T} + \dot{T}\dot{V} + T\ddot{V}\right) \\ & + \dddot{T}(r - d_T) + \frac{2r}{T_{\max}}\left(\dddot{T}T + 3\dot{T}\ddot{T}\right) \\ & + u_1\alpha\dddot{I}_1(t-\tau) + \beta\dddot{I}_1(t-\tau) \\ & - h\Big[-k\left(\dot{V}\dot{T} + V\ddot{T}\right) - kN\rho\left(\dot{T}I_2 + T\dot{I}_2\right) \end{aligned} \tag{14}$$

$$+kd_V\left(\dot{T}V+T\dot{V}\right)-\ddot{T}(d_T-r)$$
$$-\frac{2r}{T_{\max}}\left(\ddot{T}T+\dot{T}^2\right)+u_1\alpha\ddot{I}_1(t-\tau)+\beta\ddot{I}_1(t-\tau)\Bigg]$$
$$+h^2\ddot{T}+h^3\dot{T}].$$

Now let us assume that there is no change in the concentration of healthy cells. In other words, $\dot{T}=\ddot{T}=\dddot{T}=0$. By substituting u by T in Eq. (14), we obtain

$$\dot{V}=S\Bigg[\underbrace{T\left(-kN\rho\ddot{I}_2+kd_V\ddot{V}-hkN\rho\dot{I}_2+hkd_V\dot{V}\right)+u_1\alpha\dddot{I}_1(t-\tau)+\beta\dddot{I}_1(t-\tau)+hu_1\alpha\ddot{I}(t-\tau)+h\beta\ddot{I}_1(t-\tau)\right)}_{\Delta>0}\Bigg].$$

According to Eq. (11), we have

$$\dot{V}\leq S[u_{eq}-h\,\mathrm{sign}(S)]\Delta, \tag{15}$$

therefor

$$\dot{V}\leq -h|S|\Delta+|S|\Delta\leq-\eta|S|\Delta. \tag{16}$$

The parameters Δ and η are strictly positive, which guarantees asymptotic convergence of the tracking error to zero. Consequently, the controlled system satisfies all sliding mode conditions, thereby completing the proof.

Recalling that $V=\frac{1}{2}S^2$, we rewrite $|S|=\sqrt{2V}$. Substituting into the inequality gives

$$\dot{V}\leq-\eta\Delta\sqrt{2V}.$$

This is a standard differential inequality of the form

$$\dot{V}\leq-c\sqrt{V},$$

where $c=\eta\Delta\sqrt{2}>0$. Integrating both sides yields

$$\int_{V(0)}^{V(t)}\frac{dV}{\sqrt{V}}\leq-c\int_0^t dt,$$

which implies

$$2\sqrt{V(t)}\leq2\sqrt{V(0)}-ct.$$

Therefore, the sliding variable reaches zero in finite time

$$t\leq\frac{2\sqrt{V(0)}}{c}=\frac{\sqrt{2}|S(0)|}{\eta\Delta}.$$

Thus, the system reaches the sliding surface $S=0$ in finite time. This completes the proof. □

Remark 2. *In the proof above, the derivatives of the healthy CD4$^+$ T-cell population $T(t)$ are assumed to be zero, i.e., $\dot{T}=\ddot{T}=\dddot{T}=0$. This assumption is justifiable from both mathematical and biological perspectives. Mathematically, it allows for a local stability analysis around an equilibrium or quasi-equilibrium state, where $T(t)$ varies slowly and higher-order derivatives become negligible. Biologically, such an assumption reflects either a stable therapeutic state—where antiviral treatments maintain*

a constant population of healthy T-cells—or a chronic infection phase, during which the immune system dynamics reach a steady or slowly varying behavior. This simplification enables a more tractable analysis of the sliding surface behavior without significantly compromising the accuracy of the model within a biologically meaningful range.

Algorithm 2. Computational Framework for the Delayed HIV Model with Sliding Mode Control

1. **Input:** Model parameters

$$\Theta = \{s, r, d_T, k, T_{\max}, \alpha, \beta, \gamma, \rho, d_V, N, u_1\},$$

SMC parameters $\{h, \eta\}$, initial conditions $T(0), I_1(0), I_2(0), V(0)$.

2. **Initialization:** Set $\mathbf{X} \leftarrow \mathbf{X}_0$, $t \leftarrow 0$, and initialize the trajectory list as empty.

3. **While** $t \leq t_{\text{end}}$ **do**:

   (a) Compute sliding surface

$$S = \mathbf{w}^T \mathbf{x} \quad \text{(Eq. (3))}.$$

   (b) Compute equivalent control

$$u_{\text{eq}} = \text{solution of } \dot{S} = 0 \quad \text{(Eq. (6))}.$$

   (c) Compute switching control

$$u_{\text{sw}} = -h\,\text{sign}(S) \quad \text{(Eq. (11))}.$$

   (d) Total control

$$u = u_{\text{eq}} + u_{\text{sw}} \quad \text{(Eq. (12))}.$$

   (e) Update the delayed dynamical system (Eq. (2)):

$$\begin{aligned}
\dot{\mathbf{X}}(t) &= \mathscr{F}\big(\mathbf{X}(t), \mathbf{X}(t-\tau), u(t); \Theta\big), \\
T'(t) &= f_T(T, V, I_1; u, \Theta), \\
I_1'(t) &= f_{I_1}(T(t-\tau), V(t-\tau), I_1; \Theta), \\
I_2'(t) &= f_{I_2}(I_1, I_2; u_1, \Theta), \\
V'(t) &= f_V(I_2, V; \Theta).
\end{aligned}$$

   (f) Numerically integrate the system (e.g., RK4 or Simulink solver).

   (g) Append current state $\mathbf{X}(t)$ to trajectory list.

   (h) Update the time variable

$$t \leftarrow t + \Delta t.$$

4. **Return:** The full trajectory list.

4 Simulation results

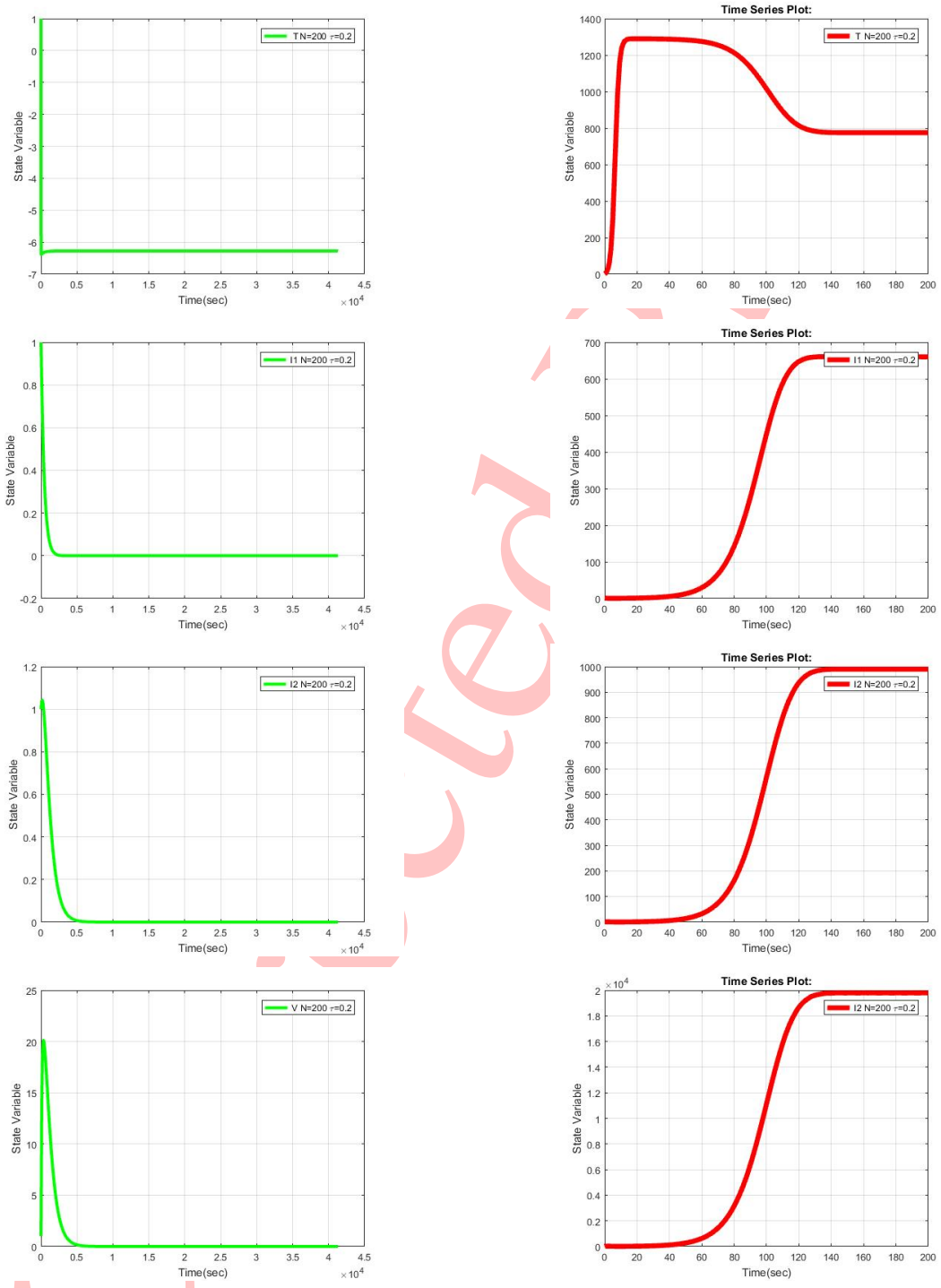
In this section, the performance of the proposed controller on system (2) is evaluated. When the count of host cells drops below 200 cells per milliliter of blood and drug treatment is not administered, the patient will typically succumb to various cancers and opportunistic infections within less than one year [9]. The simulations were carried out using *MATLAB Simulink*, which is implemented according to the Algorithm 2.

In order to better analyze the stability of the nonlinear system, two key parameters the time delay and the number of viruses N, produced by infected $CD4^+T$ cells were considered for different values of time τ. The selected delay values are within biologically reported ranges for intracellular viral replication delays. To enable a more accurate comparison of the results when implementing sliding mode control on system (2), the control gain parameter was fixed at $h = 10$. The value $h = 10$ satisfies the theoretical lower bound and was fixed across all simulations to enable consistent comparison between different delay and virus production scenarios.

Figure 1 (a) and (b) respectively illustrate the system behavior assuming a time delay of 0.2 and the production of 200 viruses by infected $CD4^+T$ cells, without and with the application of sliding mode control. To better compare the results, while keeping the number of viruses constant at $N = 200$, we increased the time delay in the system. For $\tau \geq 4$, the system's behavior rapidly diverged, and it was unable to provide a valid solution. Therefore, Figure 2 (a) and (b) respectively show the results for a time delay of 3 and 200 viruses produced by infected $CD4^+T$ cells, again both without and with the application of sliding mode control. Subsequently, the validation of the proposed controller was carried out assuming a fixed delay of 0.2 and 1200 viruses produced by the infected $CD4^+T$ cells. For this purpose, Figure 3 (a) and (b) have been presented. The analysis of the results obtained from solving the nonlinear time-delay system (2) and applying sliding mode control will be discussed after presenting the graphs.

Based on the results obtained from Figures 1, 2, and 3 (a) and (b), the following conclusions can be drawn. The system, by employing the sliding mode control method, demonstrates faster convergence to the sliding surface and suppression of oscillatory behavior and higher convergence in comparison to the system without the controller. As observed, with an increase in time delay within the system, convergence occurs later. Moreover, for values of $\tau \geq 4$, the system's runtime increases and it becomes unable to provide a solution. Assuming a constant time delay and increasing the number of viruses produced by infected $CD4^+T$ cells from 200 to 1200, the system without the controller exhibits oscillatory behavior. In each of the aforementioned variations, the use of the sliding mode controller directs all parameters toward the sliding surface and subsequently toward the origin, demonstrating finite-time convergence and robustness against delay-induced instability to enforce convergence. This ability highlights the effectiveness of the proposed control law in this control strategy.

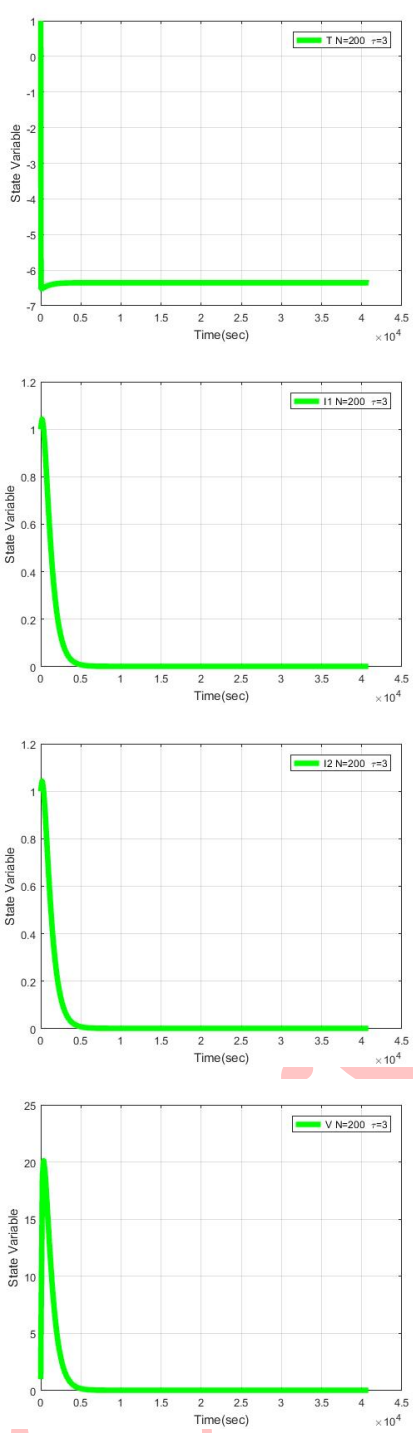
The novelty of this study lies not only in the incorporation of sliding mode control into a biologically realistic delayed HIV model, but also in the demonstration of finite-time convergence to the sliding surface, ensuring rapid and robust suppression of the viral load. This feature is especially valuable in real-world clinical scenarios where treatment efficacy must be ensured despite parameter uncertainties and time delays in biological processes. The proposed control method can be potentially applied to guide real-time antiretroviral drug administration, ensuring optimal therapeutic impact while minimizing side effects and resource usage.



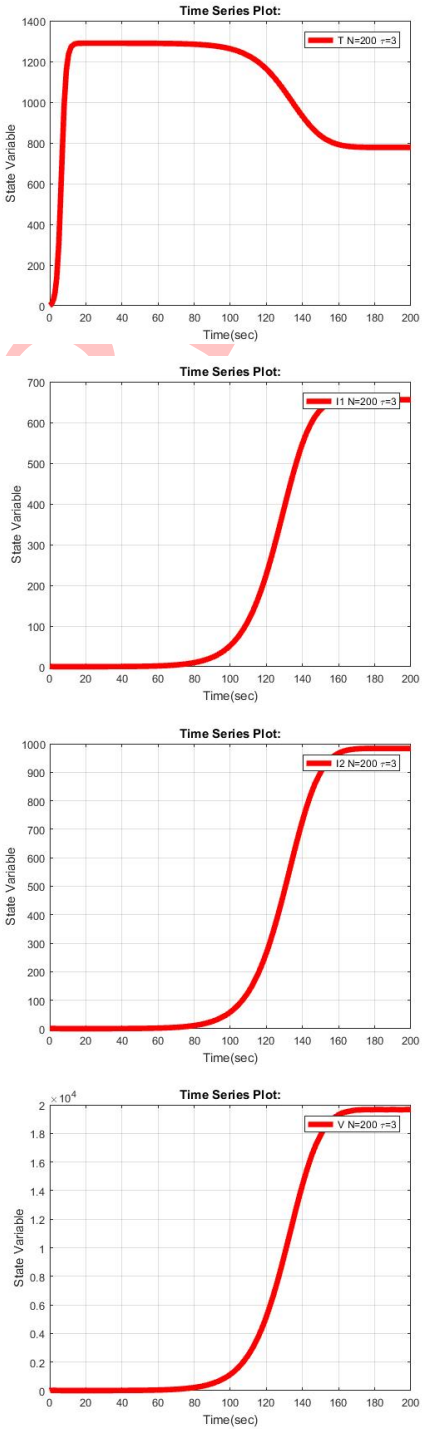
(a) For $\tau = 0.2$ and $N = 200$, and $h = 10$ using sliding mode control

(b) For $\tau = 0.2$, $N = 200$ without using sliding mode control

Figure 1: Comparison of two different system conditions

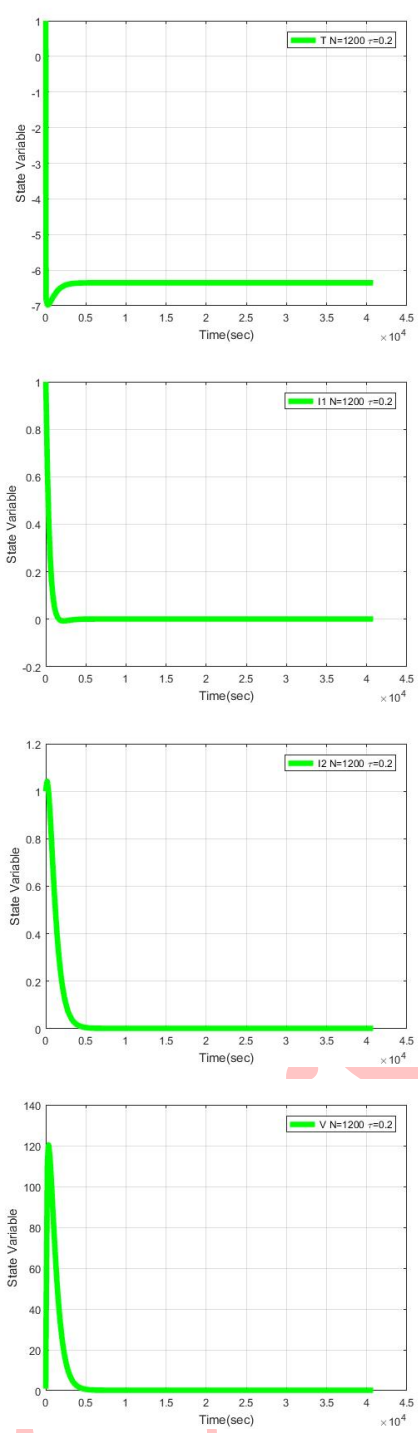


(a) For $\tau = 3$ and $N = 200$, and $h = 10$ using sliding mode control

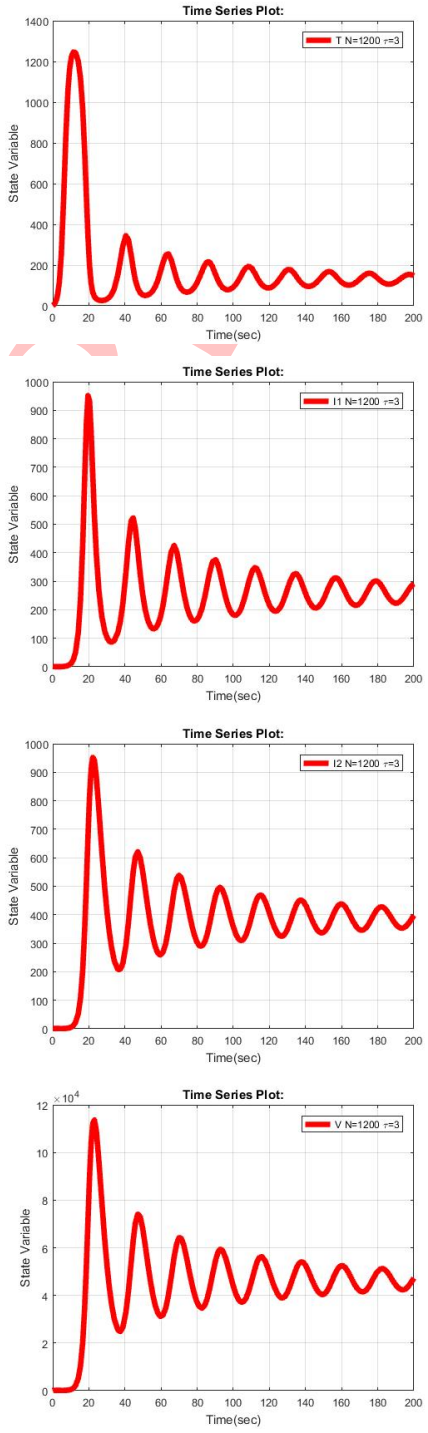


(b) For $\tau = 3$, $N = 200$ without using sliding mode control

Figure 2: Comparison of two different system conditions



(a) For $\tau = 0.2$ and $N = 1200$, and $h = 10$ using sliding mode control



(b) For $\tau = 0.2$, $N = 1200$ without using sliding mode control

Figure 3: Comparison of two different system conditions

5 Conclusion

In this paper, a sliding mode control strategy was developed for a biologically realistic fourth-order HIV infection model incorporating explicit intracellular time delays. The proposed approach addresses key limitations of classical HIV models by simultaneously accounting for nonlinear high-order dynamics, time-delay effects, and robustness requirements. A state-feedback sliding mode controller was systematically designed, and a Lyapunov-based analysis rigorously established finite-time convergence of the system trajectories to the sliding surface in the presence of time delays and parametric uncertainties. The analytical results demonstrate that increasing intracellular delay and virus production rate adversely affect system stability and may lead to loss of convergence beyond a critical delay threshold. Numerical simulations confirm the theoretical findings and reveal that excessive intracellular delay can destabilize the uncontrolled HIV dynamics, resulting in oscillatory behavior and divergence. In contrast, the proposed controller effectively suppresses oscillations, enforces finite-time convergence, and stabilizes all system states within a therapeutically desirable regime. Importantly, the control strategy does not promote convergence to a chronic infected equilibrium; rather, it mitigates delay-induced instability and regulates viral dynamics by stabilizing system trajectories.

Conflict of interest

The authors declare that they have no conflict of interest.

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