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Stabilization Control for a Class of Fractional-Order HIV-1 Infection Model with Time Delays

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Abstract: In this study, we investigated a novel asymptotic stabilization control method for a fractional-order HIV-1 infection model. First, we constructed a mathematical model of the fractional-order HIV-1 infection using the state-space equations of Caputo fractional calculus. Subsequently, a new control strategy was designed for the fractional-order HIV-1 infection model, and the corresponding asymptotic stabilization criterion was proposed by combining a novel vector Lyapunov function with the M-matrix method. Additionally, we incorporated a time delay, which was generated by the interaction between different variables in the actual system, into the fractional-order HIV-1 infection model, forming a system with a time delay. Based on the vector Lyapunov function associated with the M-matrix measure and Razumikhin interpretation, a control strategy was developed for the fractional-order HIV-1 infection model with a time delay. Finally, we show the results of two numerical simulations of the fractional-order HIV-1 infection model, with and without time delay, to illustrate the accuracy, usefulness, and universality of the proposed measure in our paper.

Keywords: HIV-1; fractional-order infection model; virus dynamics; stabilization control

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1. Introduction

AIDS is known as "Super Cancer" and the "Plague of the Century" [1–3]; according to the new AIDS data released by the WHO Global Health Watch, there have been 78 million HIV patients and there are currently 39 million HIV patients around the world. The culprit of this tragedy is HIV-1 [4–6]. When the virus develops to a certain extent, it destroys the body's immune system. The initial infection period can develop into AIDS after about 10 to 15 years. HIV-1 can be mixed with blood in a variety of ways, such as a blood transfusion, the sharing of contaminated needles, unprotected sexual intercourse, childbirth, and breastfeeding. After HIV-1 infection, CD4+ T lymphocytes in the body become the primary molecule of infection. Long-term infection of HIV-1 will cause the failure of the CD4+ T cell bank, then affect the immune response of the body, and finally form acquired immunodeficiency syndrome [7,8]. The establishment of a dynamic model of HIV-1 infection based on HIV-1 can effectively inhibit the spread of infectious diseases and lay a foundation for an in-depth understanding of the virus content in the human body and its evolution over time [9,10]. The study of this dynamic characteristic is of great significance for the further understanding of AIDS and the development of AIDS prevention and a control scheme [11–13].

The modeling of physical systems is a hot topic in current research. For example, some studies investigated the identification of parameters of an anomalous diffusion model based on measurements or modeling of heat distribution in porous aluminum using fractional differential equations [14,15]. Up to now, this has been a very effective way to use mathematical models to describe the dynamic process of virus infection [16,17]. A reasonable mathematical model combined with the HIV-1 infection mechanism can provide a theoretical basis for studying the dynamic changes of HIV-1 in vivo, which can

help people to understand the pathogenesis and transmission pattern of HIV-1, and thus, theoretically develop drugs and antiviral treatment programs to better prevent and control the transmission of HIV-1 [18,19]. Mathematical modeling and analysis of viral kinetics with humoral immunity can help to design therapeutic strategies and provide insights into the evaluation of antiviral drug therapies, and it is believed that only a deeper understanding of the immune responses can lead to the development of a safe and effective HIV-1 vaccine. In [20], the researchers pointed out that humoral immunity plays an important role in overall human immunity and studied the kinetics of the viral model with cellular and humoral immune responses. Furthermore, [21–24] referred to the immune mechanism used against the disease as cellular immunity, and the immune mechanism of antibody cells that attack the virus was called humoral immunity. Many researchers believe that antibody cells play a vital role in the immune response against the virus. However, in most viral infections, the immune response is primarily mediated by non-specific and rapidly acting cytotoxic T cells (CTLs). These cells, along with specific immune components, such as cytotoxic T lymphocytes and antibody cells, target the virus and trigger an immune response in the body. This immune response plays a crucial role in eliminating the virus and inhibiting the progression of the disease. At present, the research on HIV-1 is mainly focused on its pathogenic mechanism, and the analysis of its pathogenic mechanism is of great significance for an in-depth understanding of its pathogenic mechanism and the development of effective anti-HIV-1 drugs.

Fractional derivatives are a hot topic that has been developed in recent years, and it is widely used in many disciplines [25–30], such as in mathematics [31,32], computer engineering [33], financial systems [34], and especially in the biological field [35]. At present, many mathematical workers and applied scientists are trying to use this model to simulate biological phenomena in complex network systems [36–40]. Researchers have found that biological cell membranes have fractional electron conductivity, and thus, it can be classified as a fractional model. Currently, there were also numerous studies that focused on studying the fractional-order HIV infection process [41–44], which has become a research hotspot. In addition, studies showed that a biological model based on fractional derivatives has better performance than a traditional integer model [45,46]. Therefore, we focused on a fractional-order HIV-1 infection model. Among the research on general fractional-order HIV-1 infection models, stabilization control is the most critical issue. However, since the stability analysis measure for the integer-order system is not able to be directly put into use in a fractional-order system, stability estimates and stabilization control for the fractional form pose difficulties during the study of such issues. Moreover, there have been few significant studies on the puzzle of gradual near-stability estimates and asymptotic stabilization. Additionally, in an actual HIV-1 infection model, the time delay is a significant factor. However, the introduction of a time delay greatly increases the system complexity, and thus, it has rarely been considered in previous research.

In this study, motivated by the challenges mentioned above, we surveyed asymptotic stabilization control of a fractional-order HIV-1 infection model by applying a novel measure, that is, a vector Lyapunov function associated with an M-matrix measure. There are two foremost innovation points regarding this study. On the one hand, we considered the time delay between different variables of a fractional-order HIV-1 infection model to construct a model that incorporated a time delay. On the other hand, we applied the novel measure to design an asymptotically stabilized control strategy for an open-loop fractional-order HIV-1 infection model. This technique can solve fractional-order cases, either with or without time delay.

2. Preliminaries

Definition 1 ([47]). *The Caputo fractional-order derivative is defined as follows*:

$$^{C}D^{\alpha}f(t) = \frac{1}{\Gamma(n-\alpha)}\int_{0}^{t}(t-\tau)^{n-\alpha-1}f^{(n)}(\tau)d\tau, \tag{1}$$

where $n \in N$, $\alpha \in (n-1, n)$, *and* $f(t)$ *is any integrable function; if* $n = 1$, *one has*

$$^{C}D^{\alpha}f(t) = \frac{1}{\Gamma(1-\alpha)}\int_{0}^{t}(t-\tau)^{-\alpha}f^{(1)}(\tau)d\tau. \tag{2}$$

Then, we considered the following fractional-order system:

$$^{C}D^{\alpha}x(t) = f(x(t)), \tag{3}$$

where $\alpha \in (0,1)$, $x(t) \in \mathbb{R}^n$ is the state of Equation (3), and $f : \mathbb{R}^n \to \mathbb{R}^n$ satisfies the condition $f(0) = 0$ and the local Lipschitz continuity condition.

Definition 2 ([48]). *The solution of Equation* (3) *is called stable if for any* $\varepsilon > 0$, *there exists* $\delta = \delta(\varepsilon) > 0$ *such that for every* $\|x_0\| < \delta$, $x_0 = x(t_0)$, *and* t_0 *representing the initial time, we have*

$$\|x(t)\| < \varepsilon, \text{ for any } t. \tag{4}$$

The solution of Equation (3) is called asymptotically stable if it is stable and there exists $\hat{\delta} > 0$ such that $\lim_{t\to\infty} x(t, x_0) = 0$ whenever $\|x_0\| < \hat{\delta}$.

Lemma 1 ([47]). *Let* $x(\cdot) \in \mathbb{R}^n$ *be a differentiable vector function. Then, for any time instant* $t \geq t_0$, *the following inequality holds:*

$$\frac{1}{2}{}^{C}D^{\alpha}[x^{T}(t)x(t)] \leq x^{T}(t)\,{}^{C}D^{\alpha}x(t), \forall \alpha \in (0,1). \tag{5}$$

Lemma 2 ([49]). *Let* $x = 0$ *be an equilibrium point for the fractional-order system* (*Equation* (3)) *if there is a Lyapunov function* $V(x(t))$ *and* class-κ *functions* $\beta_i (i = 1, 2, 3)$ *satisfying*

$$\beta_1(\|x\|) \leq V(x(t)) \leq \beta_2(\|x\|), \tag{6}$$

$$^{C}D^{\alpha}V(x(t)) \leq -\beta_3(\|x\|), \tag{7}$$

where the fractional-order operator $\alpha \in (0,1)$, *then the equilibrium point of a fractional-order system is asymptotically stable.*

Definition 3 ([50]). *A real*$_{n\times n}$ *matrix* $W = [w_{ij}]$ *is an* M-matrix *if the element* $w_{ij} \leq 0$, *for* $i \neq j$ *and if all of its principal minor determinants are positive.*

Lemma 3 ([50]). *If* $W = [w_{ij}]$ *is an* M-matrix, *there exists a diagonal matrix* $P = diag\{p_1, p_2, \ldots p_N\}$ *with elements* $p_i > 0$ *such that the matrix*

$$C = W^{T}P + PW, \tag{8}$$

is positive definite.

For the above biological and virological descriptions, it is very appropriate to use a fractional-order model to construct the kinetic process of HIV-1 infection, and the specific fractional-order HIV-1 infection model is shown below [51]:

$$\begin{aligned} D^{\alpha}x(t) &= a - bx(t) - cx(t)v(t) - dx(t)y(t), \\ D^{\alpha}y(t) &= cx(t)v(t) + dx(t)y(t) - ey(t) - hy(t)z(t), \\ D^{\alpha}v(t) &= ly(t) - mv(t), \\ D^{\alpha}z(t) &= \frac{ny(t)z(t)}{p+z(t)} - qz(t), \end{aligned} \tag{9}$$

where $x(t)$, $y(t)$, and $v(t)$ are the densities of uninfected target cells (DUTCs), infected target cells (ITCs), and the free virus (FV), respectively, at time t. c is the infection rate of uninfected cells by the virus. a is the constant rate that the uninfected cells are restored, and the uninfected cells are infected at a rate of $cx(t)v(t) + dx(t)y(t)$ and die at a rate of $bx(t)$. l is the constant rate of each producing HIV-1 particle, and m is the per capita rate. The density $z(t)$ represents the concentration of CTL cells, where CTL kills infected cells at a rate of $hy(t)z(t)$ and perish at a rate of $ey(t)$. CTL cells multiply at a rate of $\frac{ny(t)z(t)}{p+z(t)}$, and die at a rate of $qz(t)$.

Lemma 4 ([51]). *For the fractional-order HIV-1 infection model* (*Equation* (9)), *the state variable is non-negatively invariant, where the non-negative invariant compact set is given as*

$$\begin{aligned} \Theta = \Big\{ &(x, y, v, z) \in \mathbb{R}^4_{\geq 0} : 0 \leq x, y \leq \tfrac{a}{\min\{b,\, e/2,\, m,\, q\}}, \\ &0 \leq v \leq \tfrac{2la}{e\min\{b,\, e/2,\, m,\, q\}}, 0 \leq z \leq \tfrac{na}{hp\min\{b,\, e/2,\, m,\, q\}} \Big\}. \end{aligned} \tag{10}$$

3. Main Results

First of all, from the fractional-order HIV-1 infection model (Equation (9)), we assumed that x^*, y^*, v^*, and z^* were the equilibrium points of the system. Then, on the basis of [51], we obtained the following equilibrium points:

$$\begin{cases} E_1 = (x^*, y^*, v^*, z^*) = (\frac{a}{b}, 0, 0, 0), \\ E_2 = (x^*, y^*, v^*, z^*) = (\frac{em}{cl+dm}, \frac{bm}{cl+dm}[\frac{(cl+dm)a}{bem} - 1], \frac{bl}{cl+dm}[\frac{(cl+dm)a}{bem} - 1], 0), \\ E_3 = (x^*, y^*, v^*, z^*) = (\frac{amn}{bmn+q(cl+dm)(p+A)}, \frac{q}{n}(p+A), \frac{lq}{nm}(p+A), A), \end{cases} \tag{11}$$

where

$$A = \frac{-(cl+dm)(eq+hpq) - mnbh + mn\sqrt{\left(\frac{(cl+dm)(eq+hpq)}{mn} + hb\right)^2 - 4(\frac{cl+dm}{m}(\frac{a^2}{b} - \frac{eq}{n}) - 1)}}{2eq(cl+dm)}. \tag{12}$$

Then, to make the analysis clearer, we moved the equilibrium point to the origin. We set $w_1(t) = x(t) - x^*, w_2(t) = y(t) - y^*, w_3(t) = v(t) - v^*, w_4(t) = z(t) - z^*$, and thus, the following equations were obtained:

$$\begin{aligned} D^{\alpha}w_1(t) &= -bw_1(t) - cw_1(t)w_3(t) - dw_1(t)w_2(t) - c(w_1(t)v^* + w_3(t)x^*) - d(w_1(t)y^* + w_2(t)x^*) \\ &= (-b - cv^* - dy^*)w_1(t) - dx^*w_2(t) - cx^*w_3(t) - cw_1(t)w_3(t) - dw_1(t)w_2(t), \\ D^{\alpha}w_2(t) &= -cw_1(t)w_3(t) - dw_1(t)w_2(t) - c(w_1(t)v^* + w_3(t)x^*) - d(w_1(t)y^* + w_2(t)x^*) - ew_2(t) \\ &\quad -hw_2(t)w_4(t) - h(w_2(t)z^* + w_4(t)y^*) \\ &= (-cv^* - dy^*)w_1(t) - (e + dx^* + hz^*)w_2(t) - cx^*w_3(t) - hy^*w_4(t) - cw_1(t)w_3(t) \\ &\quad -dw_1(t)w_2(t) - hw_2(t)w_4(t), \\ D^{\alpha}w_3(t) &= lw_2(t) - mw_3(t), \\ D^{\alpha}w_4(t) &= \frac{n(w_2(t)w_4(t) + w_2(t)z^* + w_4(t)y^* + y^*z^*)}{p + w_4(t) + z^*} - q(w_4(t) + z^*) \\ &= -qw_4(t) + \frac{nz^*w_2(t) + (ny^* - qz^*)w_4(t) + nw_2(t)w_4(t)}{w_4(t) + p + z^*}, \end{aligned} \tag{13}$$

3.1. Stabilization Control of the Fractional-Order HIV-1 Infection Model

Then, we converted the fractional-order HIV-1 infection model (Equation (13)) into a matrix form as follows:

$$D^{\alpha} w_i(t) = f_i(w(t)) + g_i(w(t)), \qquad i = 1, 2, 3, 4. \tag{14}$$

where $w_i(t)$ is the i-th state of the system and is a differentiable vector, the function $f_i(x(t))$ represents the linear part:

$$\begin{array}{l} f_1(w(t)) = (-b - cv^* - dy^*)w_1(t) - dx^* w_2(t) - cx^* w_3(t), \\ f_2(w(t)) = -(e + dx^* + hz^*)w_2(t) + (-cv^* - dy^*)w_1(t) - cx^* w_3(t) - hy^* w_4(t), \\ f_3(w(t)) = lw_2(t) - mw_3(t), \\ f_4(w(t)) = -qw_4(t), \end{array} \tag{15}$$

and $g_i^*(x(t))$ describes the nonlinear part:

$$\begin{array}{l} g_1(w(t)) = -cw_1(t)w_3(t) - dw_1(t)w_2(t), \\ g_2(w(t)) = g_1(w(t)) - hw_2(t)w_4(t), \\ g_3(w(t)) = 0, \\ g_4(w(t)) = \frac{nz^* w_2(t) + (ny^* - qz^*)w_4(t) + nw_2(t)w_4(t)}{w_4(t) + p + z^*}, \end{array} \tag{16}$$

A series of controllers were added to the system as follows:

$$D^{\alpha} x_i(t) = f_i^*(x_i(t)) + g_i^*(x(t)) + u_i(t), \qquad i = 1, 2, 3, 4. \tag{17}$$

where

$$\begin{array}{l} u_1(t) = -k_1 w_1(t) + cw_1(t)w_3(t) + dw_1(t)w_2(t), \\ u_2(t) = -k_2 w_2(t) + u_1(t) + hw_2(t)w_4(t), \\ u_3(t) = -k_3 w_3(t), \\ u_4(t) = -k_4 w_4(t) \\ \qquad + \frac{(ny^* - qz^*)w_4^2(t) + (p + z^* - 1)(nz^* w_2(t) + ny^* w_4(t) - qz^* w_4(t)) + (nz^* - n)w_2(t)w_4(t)}{w_4(t) + p + z^*}, \end{array} \tag{18}$$

and k_1, k_2, k_3, and k_4 represent the control gain, then the system can be rewritten to give

$$\begin{array}{l} D^{\alpha} w_1(t) = (-b - cv^* - dy^* - k_1)w_1(t) - dx^* w_2(t) - cx^* w_3(t), \\ D^{\alpha} w_2(t) = (-cv^* - dy^*)w_1(t) - (e + dx^* + hz^* + k_2)w_2(t) - cx^* w_3(t) - hy^* w_4(t), \\ D^{\alpha} w_3(t) = lw_2(t) - (m + k_3)w_3(t), \\ D^{\alpha} w_4(t) = -(q + ny^* - qz^* + k_4)w_4(t) + nz^* w_2(t), \end{array} \tag{19}$$

Then, we have

$$D^{\alpha} w_i(t) = f_i^*(w(t)) + g_i^*(w(t)), \qquad i = 1, 2, 3, 4. \tag{20}$$

where $f_i^*(w(t)) = \beta_i w_i(t)$ and $g_i^*(w(t)) = \sum_{j=1, j \neq i}^{4} \eta_{ij} w_j(t)$.

Theorem 1. *If the following inequality holds and the following matrix W is an M-*matrix*, then the fractional-order HIV-1 infection model (Equation* (13)*) is asymptotically stable*:

$$0 < \frac{(cl + dm)a}{emb} \leq 1, \tag{21}$$

$$\Xi = \begin{bmatrix} \varpi_{11} & \varpi_{12} & \cdots & \varpi_{1j} \\ \varpi_{21} & \varpi_{22} & \cdots & \varpi_{2j} \\ \vdots & \vdots & \ddots & \vdots \\ \varpi_{i1} & \varpi_{i2} & \cdots & \varpi_{ij} \end{bmatrix}_{i\times j}, \ \varpi_{ij} = \begin{cases} \mu_i^*, & \text{if } i = j, \\ -\eta_{ij}^*, & \text{otherwise,} \end{cases} \tag{22}$$

Proof of Theorem 1. According to (Equation (11)), we have that the equilibrium point of the fractional-order HIV-1 infection model is $E_1 = (x^*, y^*, v^*, z^*) = (\frac{a}{b}, 0, 0, 0)$.

Then, we choose the following vector Lyapunov function of the system:

$$V(x(t)) = \sum_{i=1}^{4} \delta_i V_i(x_i(t)), \tag{23}$$

where

$$\begin{aligned} x(t) &= [x_1(t), x_2(t), x_3(t), x_4(t)]^T, \\ V_i(x_i(t)) &= \tfrac{1}{2} x_i^2(t), \end{aligned} \tag{24}$$

Then, the following inequality can be derived:

$$\beta_{1i}(\|x_i(t)\|) \le V_i(x_i(t)) \le \beta_{2i}(\|x_i(t)\|), \tag{25}$$

where $\beta_{1i}(\|x_i(t)\|)$ and $\beta_{2i}(\|x_i(t)\|)$ can be selected as the following forms:

$$\beta_{1i}(\|x_i(t)\|) = \frac{1}{4}\|x_i(t)\|\|x_i(t)\|, \ \beta_{2i}(\|x_i(t)\|) = 2\|x_i(t)\|\|x_i(t)\|, \tag{26}$$

From (Equations (24)–(26)), we have

$$\beta_1(\|x(t)\|) \le V(x(t)) \le \beta_2(\|x(t)\|), \tag{27}$$

where

$$\beta_1(\|x(t)\|) = \sum_{i=1}^{4} \delta_i \beta_{1i}(\|x_i(t)\|), \beta_2(\|x(t)\|) = \sum_{i=1}^{4} \delta_i \beta_{2i}(\|x_i(t)\|), \tag{28}$$

Then, taking the fractional derivative of the sub-Lyapunov function and according to Lemma 4, we have

$$\begin{aligned} D^\alpha V(w(t)) &= D^\alpha \sum_{i=1}^{4} \delta_i V_i(w_i(t)) \\ &= D^\alpha \Big(\tfrac{1}{2}\delta_1 w_1^2(t) + \tfrac{1}{2}\delta_2 w_2^2(t) + \tfrac{1}{2}\delta_3 w_3^2(t) + \tfrac{1}{2}\delta_4 w_4^2(t) \Big) \\ &\le w_1(t)\delta_1 D^\alpha w_1(t) + w_2(t)\delta_2 D^\alpha w_2(t) + w_3(t)\delta_3 D^\alpha w_3(t) + w_4(t)\delta_4 D^\alpha w_4(t) \\ &\le w_1(t)\delta_1(-b - cv^* - dy^* - k_1)w_1(t) - w_2(t)\delta_2(e + dx^* + hz^* + k_2)w_2(t) \\ &-w_3(t)\delta_3(m + k_3)w_3(t) - w_4(t)\delta_4(q + ny^* - qz^* + k_4)w_4(t) + |w_1(t)||\delta_1 dx^*||w_2(t)| \\ &+|w_1(t)||\delta_1 cx^*||w_3(t)| + |w_2(t)||\delta_2(-cv^* - dy^*)||w_1(t)| + |w_2(t)||\delta_2 cx^*||w_3(t)| \\ &+|w_2(t)||\delta_2 hy^*||w_4(t)| + |w_3(t)||\delta_3 l||w_2(t)| + |w_4(t)||\delta_4 nz^*||w_2(t)| \\ &= -\sum_{i=1}^{4} \delta_i \mu_i^* \beta_{3i}(|w_i(t)|) + \sum_{i=1}^{4} \delta_i \beta_{3i}^{1/2}(|w_i(t)|) \sum_{j=1, j\neq i}^{N} \eta_{ij}^* \beta_{3j}^{1/2}(|w_j(t)|), \end{aligned} \tag{29}$$

Then, it can be converted into the following form:

$$\begin{aligned} D^\alpha V(w(t)) &\le -\sum_{i=1}^{4} \delta_i \mu_i^* \beta_{3i}(|w_i(t)|) + \sum_{i=1}^{4} \delta_i \beta_{3i}^{1/2}(|w_i(t)|) \sum_{j=1, j\neq i}^{N} \eta_{ij}^* \beta_{3j}^{1/2}(|w_j(t)|) \\ &= -\gamma_3^T(w(t))\Xi\gamma_3(w(t)) \\ &= -\beta_3(w(t)), \end{aligned} \tag{30}$$

where

$$\gamma(\|x(t)\|) = \left[\beta_{31}^{1/2}(\|x_1\|), \beta_{32}^{1/2}(\|x_2\|), \ldots, \beta_{3N}^{1/2}(\|x_N\|)\right]^T, \tag{31}$$

$$\beta_3(\|x(t)\|) = \gamma^T(\|x(t)\|)\Xi\gamma(\|x(t)\|). \tag{32}$$

According to Lemma 2, we can obtain that the controlled fractional-order HIV-1 infection model (Equation (14)) is asymptotically stable, which completes the proof. □

3.2. Stabilization Control of the Fractional-Order HIV-1 Infection Model with a Time Delay

The fractional-order HIV-1 infection model must inevitably be affected by a time delay in practical situations, and the generation of such a time delay is most likely to occur in the process of the interaction between different variables. To make the modeling more realistic, we added a time delay τ to the parts of the state-space equations where the different variables interact. The revised mathematical model is shown below:

$$D^{\alpha}w_i(t) = a_i w_i(t) + \sum_{j=1, j\neq i}^{4} b_{ij} w_j(t-\tau) + g_i(w(t-\tau)). \tag{33}$$

where

$$\begin{aligned} &g_1(w(t)) = -cw_1(t)w_3(t-\tau) - dw_1(t)w_2(t-\tau),\\ &g_2(w(t)) = -cw_1(t-\tau)w_3(t-\tau) - dw_1(t-\tau)w_2(t) - hw_2(t)w_4(t-\tau),\\ &g_3(w(t)) = 0,\\ &g_4(w(t)) = \frac{nz^*w_2(t-\tau)+(ny^*-qz^*)w_4(t)+nw_2(t-\tau)w_4(t)}{w_4(t)+p+z^*}, \end{aligned} \tag{34}$$

A controller was added to the system (Equation (33)) as follows:

$$D^{\alpha}x_i(t) = a_i w_i(t) + \sum_{j=1, j\neq i}^{4} b_{ij} w_j(t-\tau) + g_i(w(t-\tau)) + u_i(t), \qquad i = 1, 2, 3, 4. \tag{35}$$

where

$$\begin{aligned} &u_1(t) = -k_1 w_1(t) - \varphi_3 cw_1(t)w_3(t) - \varphi_2 dw_1(t)w_2(t),\\ &u_2(t) = -k_2 w_2(t) - \varphi_1\varphi_3 cw_1(t)w_3(t) - \varphi_1 dw_1(t)w_2(t) - \varphi_4 hw_2(t)w_4(t),\\ &u_3(t) = -k_3 w_3(t),\\ &u_4(t) = -k_4 w_4(t)\\ &\quad + \frac{(ny^*-qz^*)w_4^2(t)+(p+z^*-1)(nz^*\varphi_2 w_2(t)+ny^*w_4(t)-qz^*w_4(t))+(nz^*-n\varphi_2)w_2(t)w_4(t)}{w_4(t)+p+z^*}, \end{aligned} \tag{36}$$

Assumption 1. *According to the Razumikhin interpretation* [47], we considered that there is a continuous non-decreasing function $\zeta_j(u) > u, j = 1, 2, 3, 4$, *for* $u > 0$ *such that*

$$\left|x_j(t-\tau)\right| < \zeta_j(\left|x_j(t)\right|), \tag{37}$$

Theorem 2. *Assume that the controlled fractional-order HIV-1 infection model satisfies Assumption 1 and Equation* (21). *In addition, the following matrix W is an M-*matrix. *Then, the controlled fractional-order HIV-1 infection model with a time delay* (*Equation* (35)) *is asymptotically stable*.

$$\Xi = \begin{bmatrix} \varpi_{11} & \varpi_{12} & \cdots & \varpi_{1j} \\ \varpi_{21} & \varpi_{22} & \cdots & \varpi_{2j} \\ \vdots & \vdots & \ddots & \vdots \\ \varpi_{i1} & \varpi_{i2} & \cdots & \varpi_{ij} \end{bmatrix}_{i\times j}, \ \varpi_{ij} = \begin{cases} \mu_i, & \text{if } i = j, \\ -\eta_{ij}, & \text{otherwise}, \end{cases} \tag{38}$$

Proof of Theorem 2. Choosing the same vector Lyapunov function of the system:

$$V(x(t)) = \sum_{i=1}^{4} \delta_i V_i(x_i(t)), \tag{39}$$

we can also obtain the following inequality:

$$\beta_1(\|x(t)\|) \leq V(x(t)) \leq \beta_2(\|x(t)\|), \tag{40}$$

According to the conditions in Assumption 1 and (Equation (37)), we selected the function $\zeta_j(\beta_{3j}^{1/2}(\|x_j(t)\|))$ to be

$$\zeta_i(\beta_{3j}^{1/2}(\|x_j(t)\|)) = \varphi_j\|x_j(t)\|, \tag{41}$$

where $\varphi_j, j = 1,2,3,4$, satisfies $\varphi_j > 1$. Then, taking the fractional derivative of the sub-Lyapunov function and according to Lemma 4, we have

$$\begin{aligned}
&D^\alpha V(x(t))D^\alpha V(w(t)) = D^\alpha \sum_{i=1}^{4} p_i V_i(w_i(t))\\
&= D^\alpha\left(\tfrac{1}{2}\delta_1 w_1^2(t) + \tfrac{1}{2}\delta_2 w_2^2(t) + \tfrac{1}{2}\delta_3 w_3^2(t) + \tfrac{1}{2}\delta_4 w_4^2(t)\right)\\
&\leq w_1(t)\delta_1 D^\alpha w_1(t) + w_2(t)\delta_2 D^\alpha w_2(t) + w_3(t)\delta_3 D^\alpha w_3(t) + w_4(t)\delta_4 D^\alpha w_4(t)\\
&= w_1(t)\delta_1(-b - cv^* - dy^* - k_1)w_1(t) - w_1(t)\delta_1 dx^* w_2(t-\tau) - w_1(t)\delta_1 cx^* w_3(t-\tau) +\\
&w_2(t)\delta_2(-cv^* - dy^*)w_1(t-\tau) - w_2(t)\delta_2(e + dx^* + hz^* + k_2)w_2(t) - w_2(t)\delta_2 cx^* w_3(t-\tau)\\
&-w_2(t)\delta_2 hy^* w_4(t-\tau) + w_3(t)\delta_3 l w_2(t-\tau) - w_3(t)\delta_3(m + k_3)w_3(t)\\
&-w_4(t)\delta_4(q + ny^* - qz^* + k_4)w_4(t) + w_4(t)\delta_4 nz^* w_2(t-\tau) - w_1(t)cw_1(t)w_3(t-\tau)\\
&-w_1(t)dw_1(t)w_2(t-\tau) - w_2(t)cw_1(t-\tau)w_3(t-\tau) - w_2(t)dw_1(t-\tau)w_2(t)\\
&-w_2(t)hw_2(t)w_4(t-\tau) + w_4(t)\tfrac{nz^* w_2(t-\tau) + (ny^* - qz^*)w_4(t) + nw_2(t-\tau)w_4(t)}{w_4(t) + p + z^*}\\
&-w_1(t)\varphi_3 w_1(t)w_3(t) - w_1(t)\varphi_2 w_1(t)w_2(t) - w_2(t)\varphi_1\varphi_3 w_1(t)w_3(t)\\
&-w_2(t)\varphi_1 w_1(t)w_2(t) - w_2(t)\varphi_4 w_2(t)w_4(t)\\
&+w_4(t)\tfrac{(ny^* - qz^*)w_4^2(t) + (p + z^* - 1)(nz^*\varphi_2 w_2(t) + ny^* w_4(t) - qz^* w_4(t)) + (nz^* - n\varphi_2)w_2(t)w_4(t)}{w_4(t) + p + z^*},
\end{aligned} \tag{42}$$

According to (Equations (40)–(42)), this can be converted into the following form:

$$\begin{aligned}
&D^\alpha V(x(t))\\
&\leq w_1(t)\delta_1(-b - cv^* - dy^* - k_1)w_1(t) + w_1(t)\delta_1 dx^*\varphi_2 w_2(t) + w_1(t)\delta_1 cx^*\varphi_3 w_3(t) +\\
&w_2(t)|\delta_2(-cv^* - dy^*)|\varphi_1 w_1(t) - w_2(t)\delta_2(e + dx^* + hz^* + k_2)w_2(t) + w_2(t)\delta_2 cx^*\varphi_3 w_3(t) + w_2(t)\delta_2 hy^*\varphi_4 w_4(t)\\
&+w_3(t)\delta_3 l\varphi_2 w_2(t) - w_3(t)\delta_3(m + k_3)w_3(t) - w_4(t)\delta_4(q + k_4)w_4(t)\\
&+w_1^2(t)c\varphi_3 w_3(t) + w_1^2(t)d\varphi_2 w_2(t) + w_2(t)c\varphi_1 w_1(t)\varphi_3 w_3(t) + w_2(t)d\varphi_1 w_1(t)w_2(t) + w_2(t)hw_2(t)\varphi_4 w_4(t)\\
&+w_4(t)\tfrac{nz^*\varphi_2 w_2(t) + (ny^* - qz^*)w_4(t) + n\varphi_2 w_2(t)w_4(t)}{w_4(t) + p + z^*} - w_1(t)\varphi_3 w_1(t)w_3(t) - w_1(t)\varphi_2 w_1(t)w_2(t)\\
&-w_2(t)\varphi_1\varphi_3 w_1(t)w_3(t) - w_2(t)d\varphi_1 w_1(t)w_2(t) - w_2(t)h\varphi_4 w_2(t)w_4(t)\\
&+w_4(t)\tfrac{(ny^* - qz^*)w_4^2(t) + (p + z^* - 1)(nz^*\varphi_2 w_2(t) + ny^* w_4(t) - qz^* w_4(t)) + (nz^* - n)\varphi_2 w_2(t)w_4(t)}{w_4(t) + p + z^*}\\
&= w_1(t)\delta_1(-b - cv^* - dy^* - k_1)w_1(t) + w_1(t)\delta_1 dx^*\varphi_2 w_2(t) + w_1(t)\delta_1 cx^*\varphi_3 w_3(t) +\\
&w_2(t)|\delta_2(-cv^* - dy^*)|\varphi_1 w_1(t) - w_2(t)\delta_2(e + dx^* + hz^* + k_2)w_2(t) + w_2(t)\delta_2 cx^*\varphi_3 w_3(t) + w_2(t)\delta_2 hy^*\varphi_4 w_4(t)\\
&+w_3(t)\delta_3 l\varphi_2 w_2(t) - w_3(t)\delta_3(m + k_3)w_3(t) - w_4(t)\delta_4(q + ny^* - qz^* + k_4)w_4(t) + w_4(t)\delta_4 nz^*\varphi_2 w_2(t)\\
&= -\sum_{i=1}^{4}\delta_i\mu_i\beta_{3i}(w_i(t)) + \sum_{i=1}^{4}\delta_i\beta_{3i}^{1/2}(w_i(t))\sum_{j=1,j\neq i}^{N}\eta_{ij}\beta_{3j}^{1/2}(w_j(t)),
\end{aligned} \tag{43}$$

Then, it can be converted into the following form:

$$\begin{aligned}
D^\alpha V(w(t)) &\leq -\sum_{i=1}^{4}\delta_i\mu_i\beta_{3i}(|w_i(t)|) + \sum_{i=1}^{4}\delta_i\beta_{3i}^{1/2}(|w_i(t)|)\sum_{j=1,j\neq i}^{N}\eta_{ij}\beta_{3j}^{1/2}(|w_j(t)|)\\
&= -\gamma_3^T(w(t))\Xi\gamma_3(w(t))\\
&= -\beta_3(w(t)),
\end{aligned} \tag{44}$$

where

$$\gamma(\|x(t)\|) = \left[\beta_{31}^{1/2}(\|x_1\|), \beta_{32}^{1/2}(\|x_2\|), \ldots, \beta_{3N}^{1/2}(\|x_N\|)\right]^T, \tag{45}$$

$$\beta_3(\|x(t)\|) = \gamma^T(\|x(t)\|)\Xi\gamma(\|x(t)\|). \tag{46}$$

According to Lemma 2, we can obtain that the controlled fractional-order HIV-1 infection model (Equation (35)) is asymptotically stable, which completes the proof. □

4. Numerical Simulation

In this section, we give the results of the numerical simulation of the fractional-order HIV-1 infection model, which verified the effectiveness and feasibility of the proposed method through the time responses. According to the setting of the structural parameters of the fractional-order HIV-1 infection model in the actual situation [51], the structural parameters of the fractional-order HIV-1 infection model were set to the values shown in Table 1.

Table 1. The structural parameters of the fractional-order HIV-1 infection model.

Rate of the uninfected cells	a	260
Rate parameter of uninfected cell death	b	0.2
Infection rate of uninfected cells by the virus	c	0.001
Infected rate parameter of uninfected cells	d	0.0008
Rate parameter of CTL perishing	e	2.5
Rate parameter of CTL killing infected cells	h	0.04
Rate of each reproducing HIV-1 particle	l	1.5
Per capita rate	m	3.2
Rate numerator parameter of CTL cells multiplying	n	0.03
Rate denominator parameter of CTL cells multiplying	p	0.8
Rate parameter of CTL cell death	q	2.7

Then, according to the control strategy in Theorem 1, the control gain could be set to

$$k_1 = 10, k_2 = 12, k_3 = 3, k_4 = 6, \tag{47}$$

Then, according to (Equation (29)), we had

$$W = \begin{bmatrix} \mu_1^* & -\eta_{12}^* & -\eta_{13}^* & -\eta_{14}^* \\ -\eta_{21}^* & \mu_2^* & -\eta_{23}^* & -\eta_{24}^* \\ -\eta_{31}^* & -\eta_{32}^* & \mu_3^* & \eta_{34}^* \\ -\eta_{41}^* & -\eta_{42}^* & -\eta_{43}^* & \mu_4^* \end{bmatrix} = \begin{bmatrix} 10.2 & -1.04 & -1.3 & 0 \\ 0 & 14.5 & -1.3 & 0 \\ 0 & -1.5 & 6.4 & 0 \\ 0 & 0 & 0 & 8.7 \end{bmatrix}. \tag{48}$$

Obviously, it can be determined via a calculation that W is an M-matrix. In order to verify the effectiveness of the proposed control strategy, the time responses of different fractional orders are shown in the figures below (Figures 1–10).

In light of the time responses, we were able to draw the conclusion that the controlled fractional-order HIV-1 infection model could quickly converge and had almost no overshoot. This showed that our control strategy was effective and had a fast control rate and high control accuracy. Then, we conducted control strategy verification experiments on the controlled fractional-order HIV-1 infection model with a time delay. The structural parameter values are shown in Table 2.

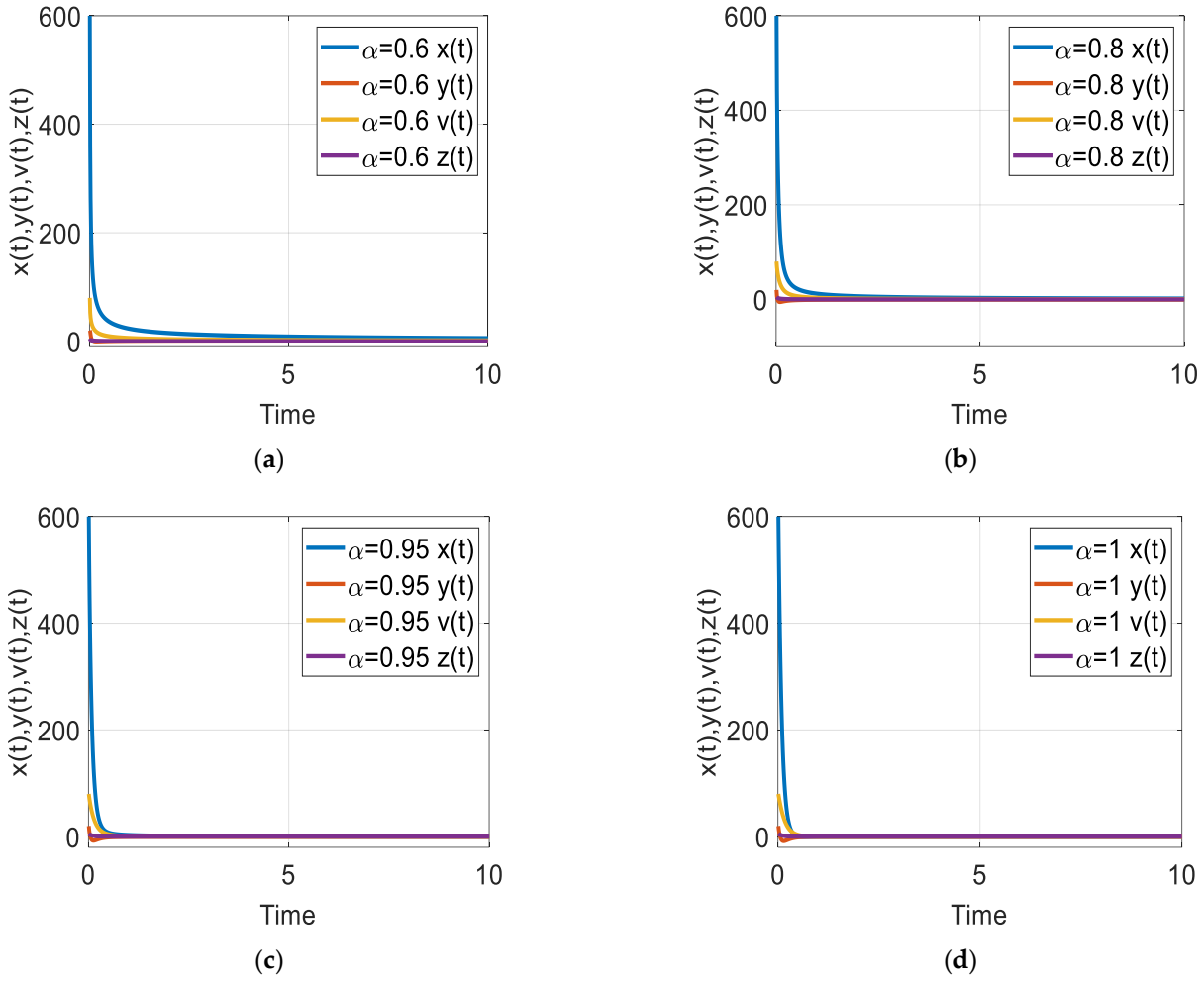


Figure 1. Time response of the fractional-order HIV-1 infection model with control when x(0) = 600, $y(0) = 20$, $v(0) = 80$, and $z(0) = 5$. Each subfigures (**a**–**d**) represent the time response of the system in a different fractional order.

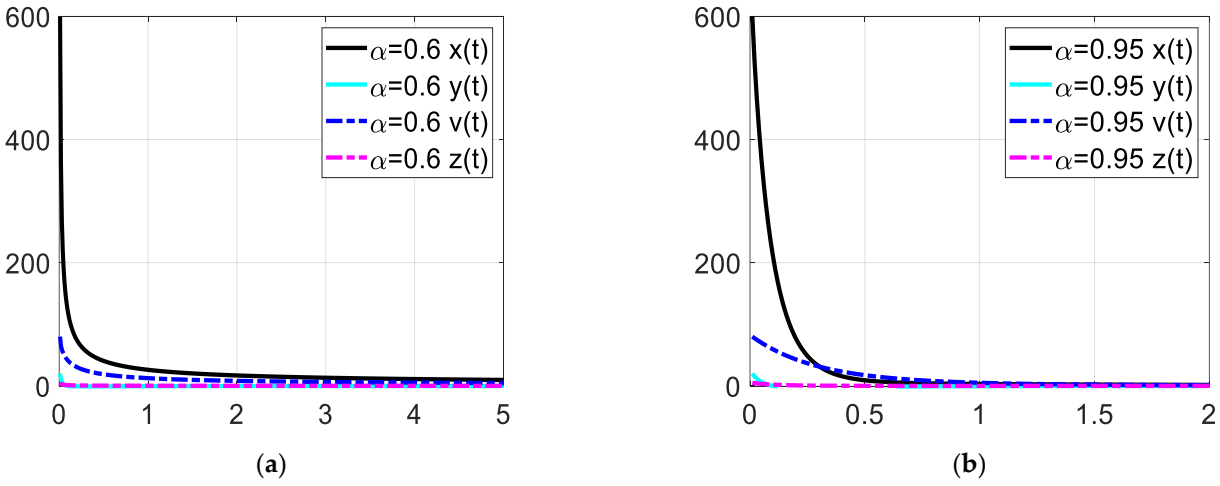


Figure 2. Enlarged view of the time response at low time values. Each subfigures (**a**,**b**) represent the time response of the system in a different fractional order.

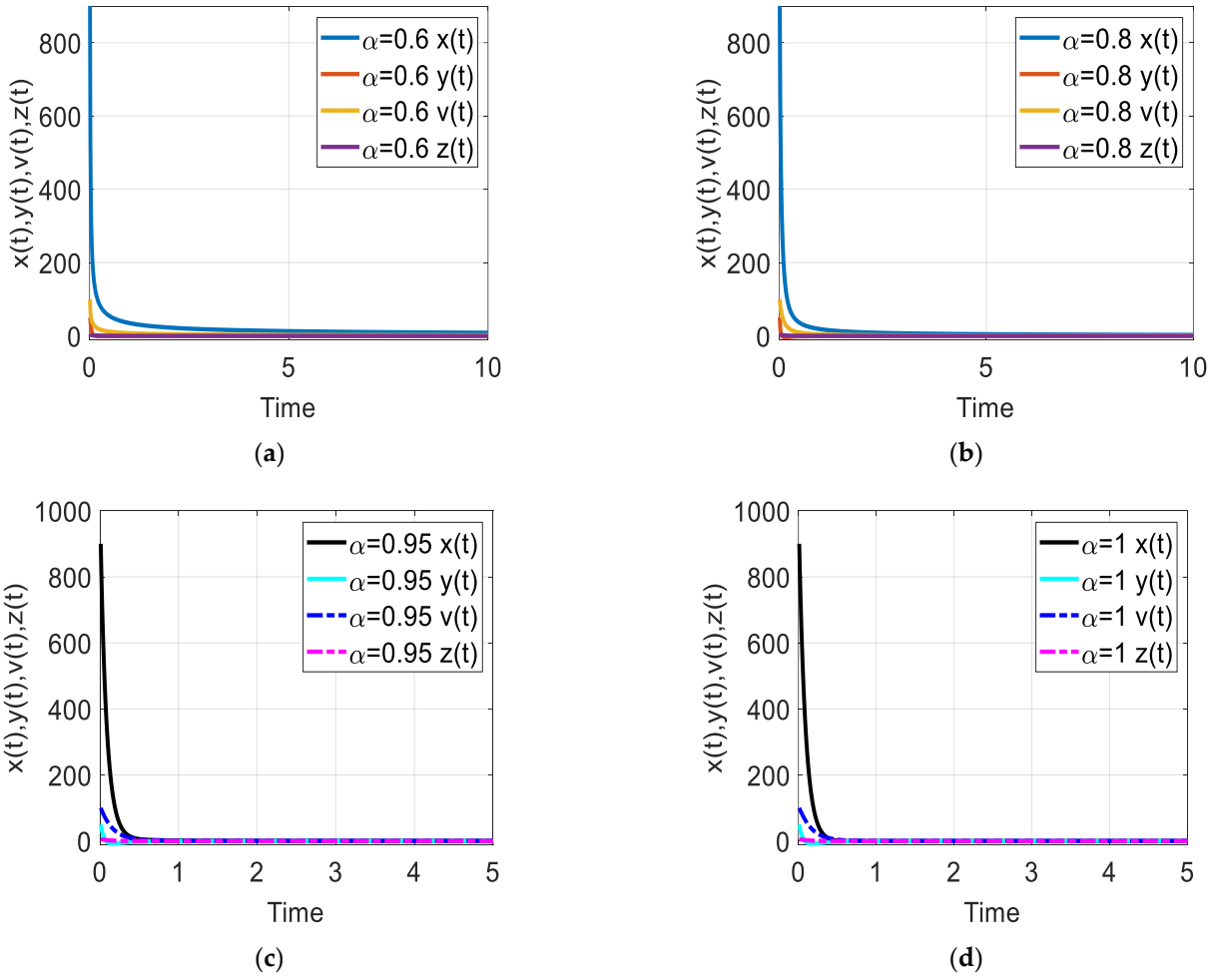


Figure 3. Time response of the fractional-order HIV-1 infection model with control when $x(0) = 900$, $y(0) = 50$, $v(0) = 100$, and $z(0) = 5.5$. Each subfigures (**a**–**d**) represent the time response of the system in a different fractional order.

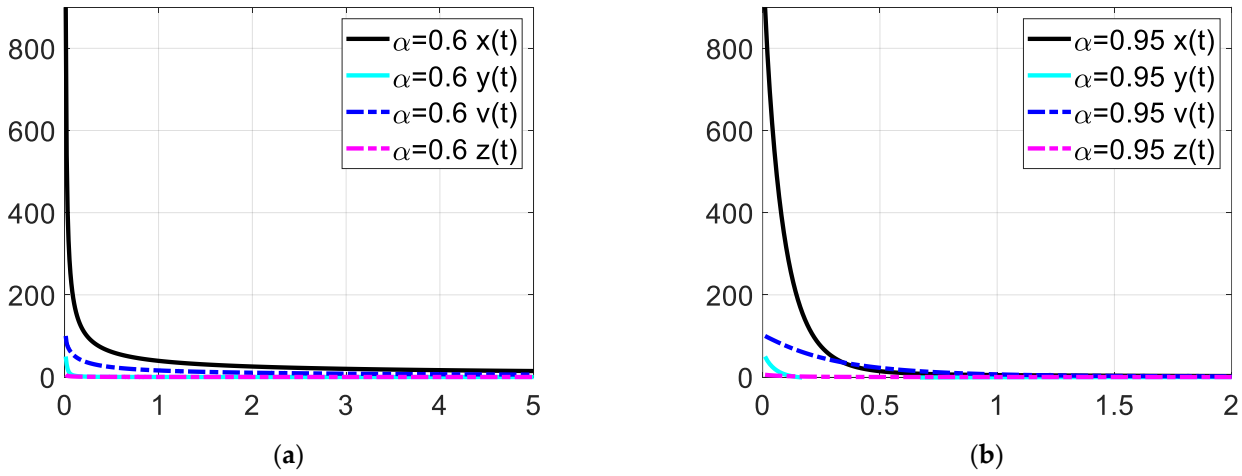


Figure 4. Enlarged view of the time response at low time values. Each subfigures (**a**,**b**) represent the time response of the system in a different fractional order.

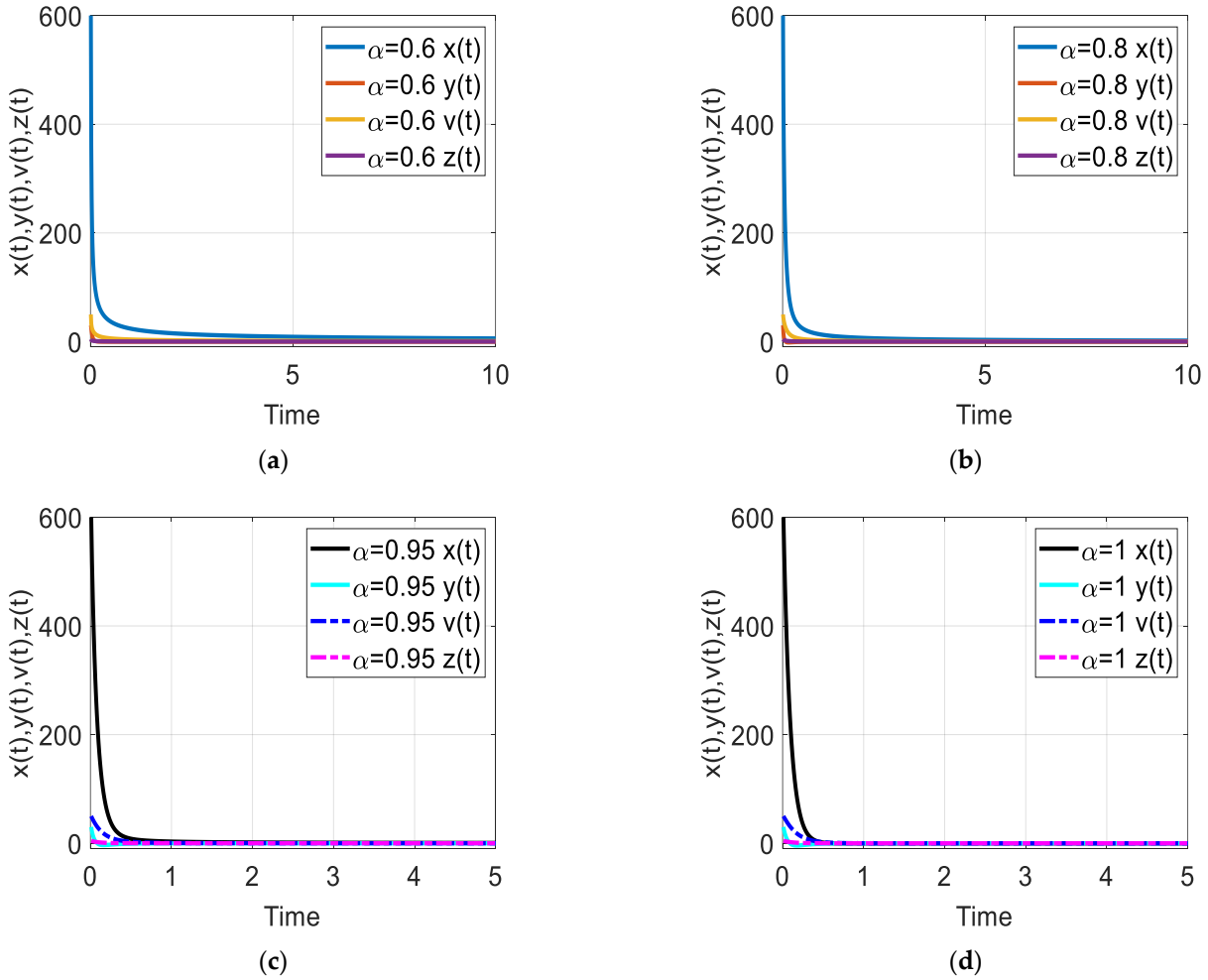


Figure 5. Time response of the fractional-order HIV-1 infection model with control when x(0) = 600, $y(0) = 30$, $v(0) = 50$, and $z(0) = 4.5$. Each subfigures (**a**–**d**) represent the time response of the system in a different fractional order.

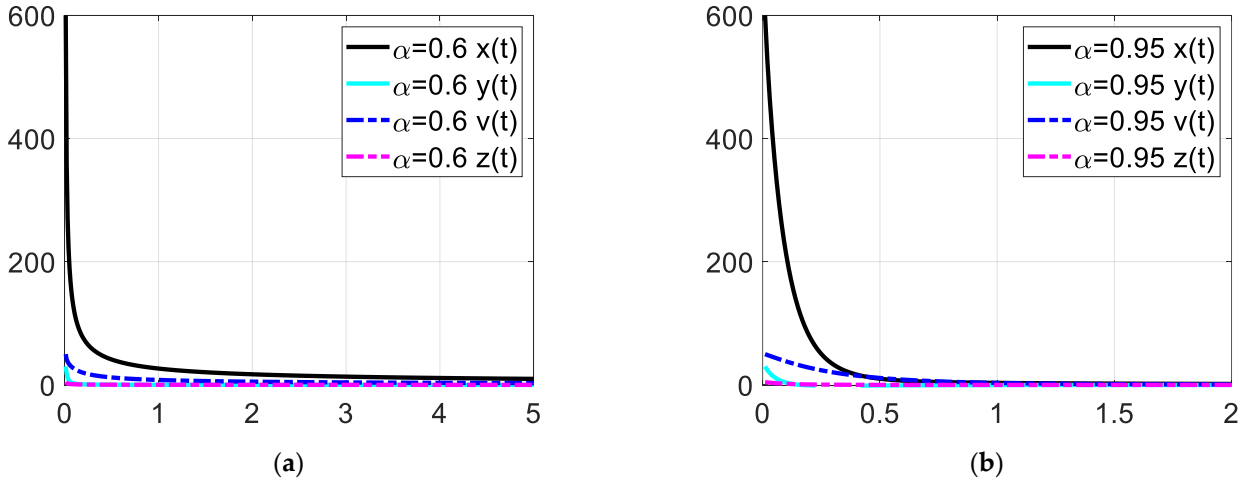


Figure 6. Enlarged view of the time response at low time values. Each subfigures (**a**,**b**) represent the time response of the system in a different fractional order.

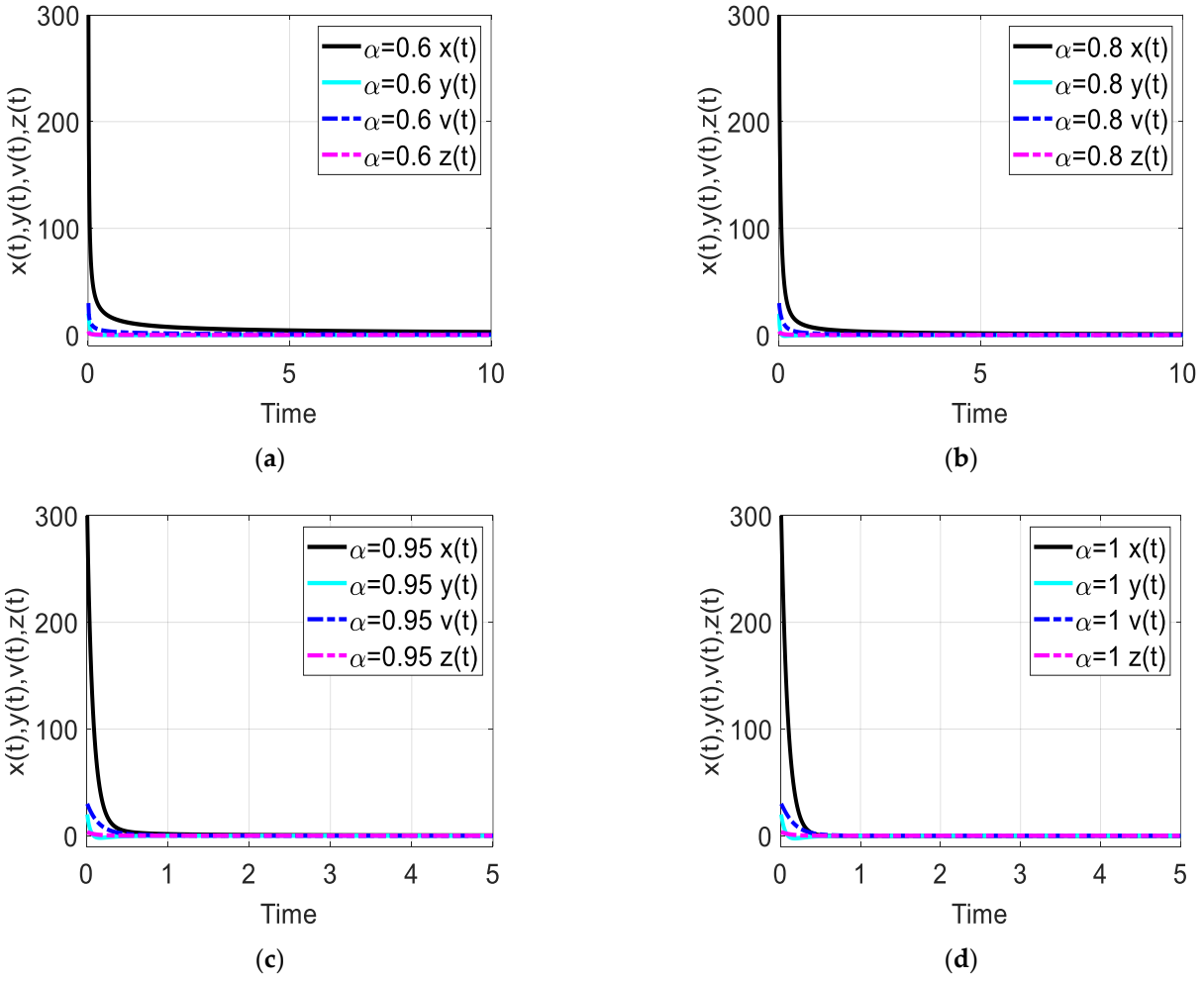


Figure 7. Time response of the fractional-order HIV-1 infection model with control when x(0) = 300, $y(0) = 20$, $v(0) = 30$, and $z(0) = 3.5$. Each subfigures (**a**–**d**) represent the time response of the system in a different fractional order.

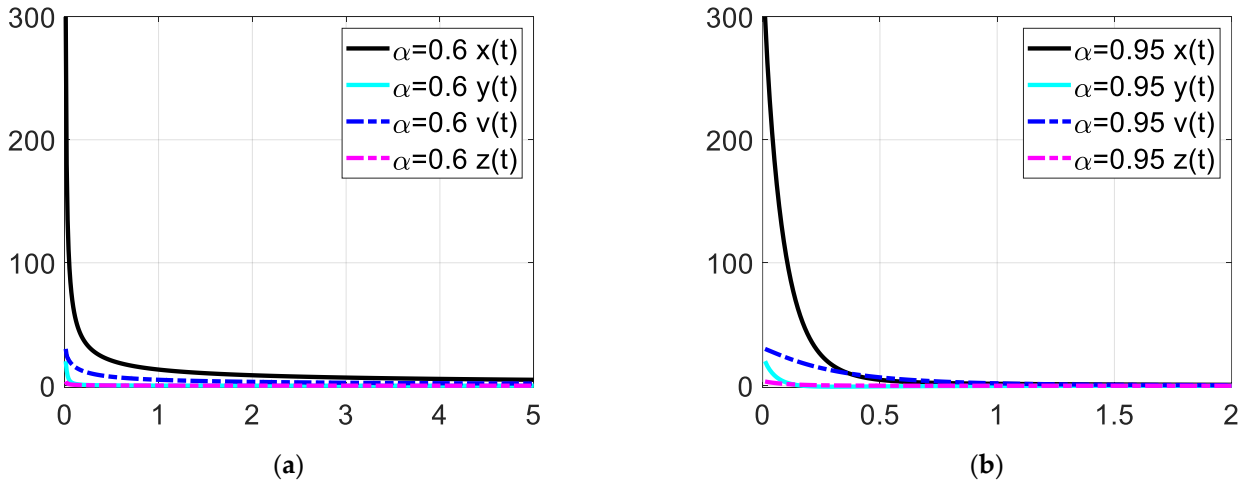
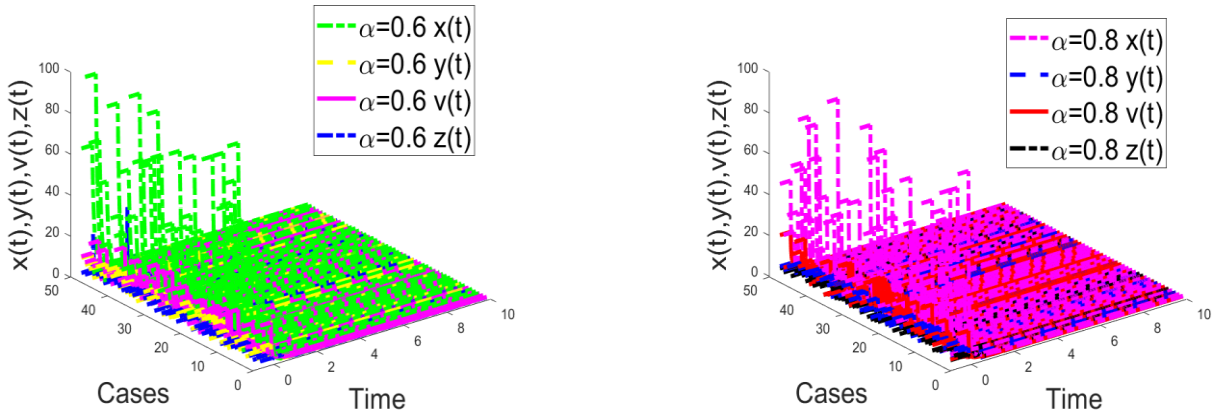
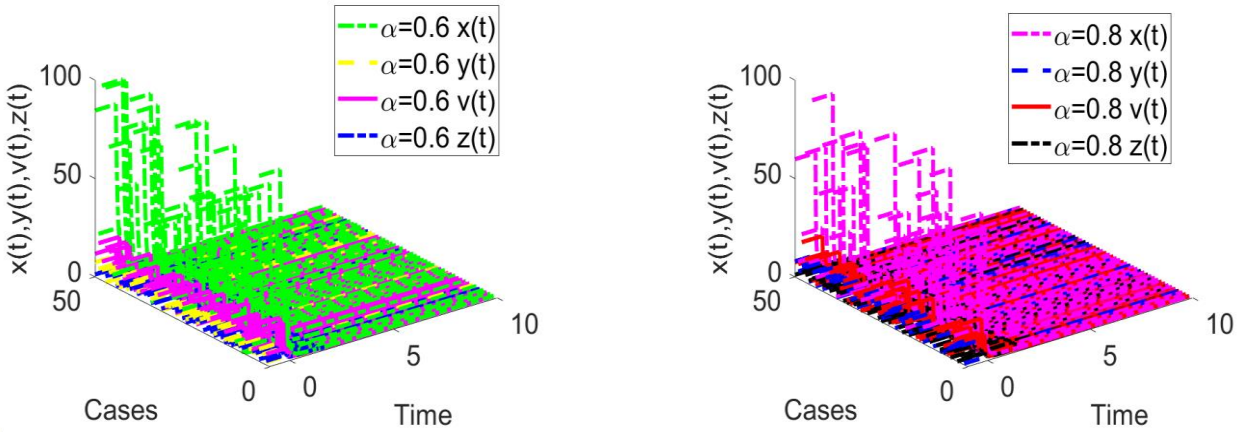


Figure 8. Enlarged view of the time response at low time values. Each subfigures (**a**,**b**) represent the time response of the system in a different fractional order.



(**a**) (**b**)

Figure 9. Time response of the fractional-order HIV-1 infection model with a time delay when $\tau = 0.5$. Each subfigures (**a**,**b**) represent the time response of the system in a different fractional order.



(**a**) (**b**)

Figure 10. Time response of the fractional-order HIV-1 infection model with a time delay when $\tau = 1.5$. Each subfigures (**a**,**b**) represent the time response of the system in a different fractional order.

Table 2. The structural parameters of the fractional-order HIV-1 infection model with a time delay.

Rate of the uninfected cells	a	260
Rate parameter of uninfected cell death	b	6.2
Infection rate of uninfected cells by the virus	c	0.001
Infected rate parameter of uninfected cells	d	0.0008
Rate parameter of CTL perishing	e	0.5
Rate parameter of CTL killing infected cells	h	0.04
Rate of each reproducing HIV-1 particle	l	1.5
Per capita rate	m	3.2
Rate numerator parameter of CTL cells multiplying	n	0.03
Rate denominator parameter of CTL cells multiplying	p	0.8
Rate parameter of CTL cell death	q	2.7
Time delay of the process of interaction of different variables	τ	0.5

According to (Equation (41)) and the control strategy in Theorem 2, φ_j could be selected as 1.5, and the control gain could be set to

$$k_1 = 15,\ k_2 = 8,\ k_3 = 13,\ k_4 = 6, \tag{49}$$

Then, according to (Equation (43)), we had

$$W = \begin{bmatrix} \mu_1 & -\eta_{12} & -\eta_{13} & -\eta_{14} \\ -\eta_{21} & \mu_2 & -\eta_{23} & -\eta_{24} \\ -\eta_{31} & -\eta_{32} & \mu_3 & \eta_{34} \\ -\eta_{41} & -\eta_{42} & -\eta_{43} & \mu_4 \end{bmatrix} = \begin{bmatrix} 21.2 & -0.05 & -0.06 & 0 \\ 0 & 8.53 & -0.06 & 0 \\ 0 & -2.25 & 16.2 & 0 \\ 0 & 0 & 0 & 8.7 \end{bmatrix}. \quad (50)$$

Obviously, it can be determined via a calculation that W is an M-matrix. Then, for the purpose of further expounding on the universality of our new control strategy, 50 groups of initial values were selected arbitrarily. The state curves of the controlled fractional-order HIV-1 infection model with a time delay are demonstrated below.

From the time responses, the same result could be obtained. Without loss of generality, another time delay $\tau = 1.5$ was selected randomly, and 50 groups of initial values were also chosen; the corresponding state curves of the controlled fractional-order HIV-1 infection model with a time delay are demonstrated below.

From the time responses, the same result could be obtained. To further verify the robustness of our control strategy, a larger time delay of $\tau = 10$ was set, and the corresponding state curves of the controlled fractional-order HIV-1 infection model with a time delay are demonstrated below.

The same conclusion could be clearly obtained, as shown from the time responses in Figure 11.

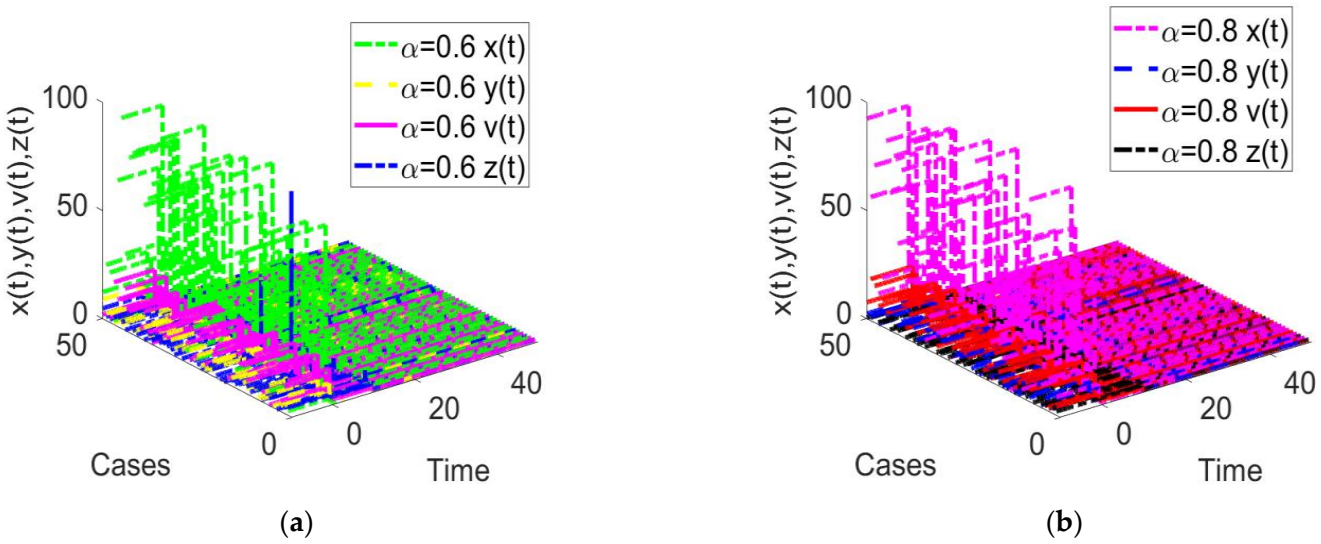


Figure 11. Time response of the fractional-order HIV-1 infection model with a time delay when $\tau = 10$. Each subfigures (**a**,**b**) represent the time response of the system in a different fractional order.

5. Conclusions

In summary, we first applied a combination of a vector Lyapunov function and the M-matrix measure to a fractional-order HIV-1 infection model. Also, we conducted asymptotic stabilization control to design two new control strategies for situations with or without a time delay. At the same time, we proposed a corresponding asymptotic stabilization criterion. The experimental consequences clearly revealed that our proposed measure had outstanding effectiveness and universality for fractional-order HIV-1 infection models. When different initial values and time delays were selected, the controlled system could always achieve asymptotic stability. This method is helpful for revealing the infection process of HIV-1 and provides a new theoretical basis for controlling the infection rate of the virus. However, in this study, we only analyzed the case of a constant delay and did not consider a fluctuating delay. Therefore, in future work, we will focus on the case with a variable time lag.

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