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Global Stability of a Fractional Virus Infection Model in the Presence of Humoral Immunity and Two Classes of Infected Cell

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ABSTRACT: It is well known that the benefit of fractional differentiation makes strong utility to model natural realities with vast range memory, hereditary properties, and viral infections such as SARS, COVID, HIV, and Dengue fever. According to biological evidence, complicated systems are more inclined to stability in comparison to simple systems, so in this article, we focused on a fractional derivative order system. Adequate qualifications for the global steady state of stationary points of a Caputo fractional derivative order system with Beddington-DeAngelis functional response will be obtained by using Lyapunov's method and LaSalle's invariance principle. We prove the global stability of the equilibria of the system by the values of the primary reproductive number (B_r) and the reproductive number for humoral immune response (R_H) as a natural reaction of antibodies. We support the analytical results through numerical simulations.

Key Words: Virus infection, humoral immunity, Caputo fractional derivative.

Contents

1. Introduction

Viral infections occur due to infection with a virus like the human immunodeficiency virus (HIV) that attacks $CD4$ cells and causes of the drastic illness known as acquired immunodeficiency syndrome (AIDS), the hepatitis B virus (HBV) and hepatitis C virus (HCV) that are responsible for most cases of chronic liver disease which can make progress to cirrhosis or liver cancer, as well as the severe acute respiratory syndrome coronavirus 2 (SARS-COV-2) that causes coronavirus disease 2019 (COVID-19). These viral infections represent a major health problem by causing many deaths. According to estimates from the World Health Organization (WHO) in 2023, approximately 630 000 people died from HIV-related causes and 1.3 million people acquired HIV [1]. Also, WHO estimated that 254 million people worldwide are living with chronic HBV in 2022 and 50 million people are living with HCV, and 1.3 million people died from HCV and HBV [2]. Furthermore, COVID-19 continue to emerge in the world attaining over 704 million people, and over 7 million deaths [3].

In the literature, numerous mathematical organic models have been proposed in order to understand and describe the dynamics viral infection. One of the basic of these models was introduced in 1996 by Nowak and Bangham [4] to study HIV infection, and later adapted to HBV [5,6] and HCV [7] infections. A general version of such viral infective model was offered by Hattaf et al. [8]. A category of HIV infection models with cure of infected cells in eclipse stage was investigated in [9]. In 2015, Elaiw [10] considered an ordinary differential equation (ODE) system including latently and actively infected

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cells with Beddington-DeAngelis functional response and in [11], Hattaf et al. considered a completely different ODE system including latently and actively infected cells with general incidence function.

On the other hand, the above-mentioned models used the classical integer derivative which is a local operator unlike the fractional derivative operators. Furthermore, fractional order models are more consistent with real phenomena than integer order models because fractional derivatives allow to describe the memory and hereditary traits inherent in various materials and processes [12,13,14,15,16]. Nowadays, fractional approximations are used in many applied and medical sciences widely.

The focal aim of the present study is to investigate the effect of memory on the dynamics of viral infection model presented in [10] by using the Caputo fractional derivative [17]. To do this, the next section presents some preliminaries and our model formulation with Caputo fractional derivative. Section 3 determines the threshold parameters and equilibria of our fractional model with humoral immunity. Section 4 discusses the uniqueness, positivity and boundedness of solutions. Section 5 establishes the global stability of the three equilibria. Section 6 presents numerical simulations to demonstrate the analytical outcomes. Finally, Section 7 draws some deductions.

2. Preliminaries and Model Formulation

In this part, we bring forward the fractional model below:

$$\begin{aligned} D^{\eta}X_1(t) &= \xi - \mu_1 X_1 - \frac{\rho X_1 X_4}{1 + a_1 X_1 + a_2 X_4}, \\ D^{\eta}X_2(t) &= \frac{(1-h)\rho X_1 X_4}{1 + a_1 X_1 + a_2 X_4} - (\mu_2 + r)X_2, \\ D^{\eta}X_3(t) &= \frac{h\rho X_1 X_4}{1 + a_1 X_1 + a_2 X_4} + rX_2 - aX_3, \\ D^{\eta}X_4(t) &= kX_3 - uX_4 - pX_4 X_5, \\ D^{\eta}X_5(t) &= cX_4 X_5 - bX_5, \end{aligned} \tag{2.1}$$

where $X_1(t)$, $X_2(t)$, $X_3(t)$, $X_4(t)$ and $X_5(t)$ are the condensations of the uninfected $CD4$ cells, latently infected cells which are part time unable to produce new infectious virus, actively infected cells which have that potential to produce new infectious virus, free virus particles and antibodies produced by B cells at time t, arranged orderly.

We considered nonlinear incidence function Beddington-DeAngelis which describes HIV infection with infectious rate ρ. Fractions h and $(1-h)$ with $0 \leq h \leq 1$ are the probabilities of uninfected cells that will become either latently infected or actively infected.

System (2.1) has to be studied with this initial conditions:

$$X_1(0) > 0, \quad X_2(0) > 0, \quad X_3(0) > 0, \quad X_4(0) > 0, \quad X_5(0) > 0.$$

Definition 2.1 The Caputo fractional derivative of order $\alpha > 0$ for an arbitrary function g is given as follows:

$$D^{\alpha}g(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t (t-x)^{-\alpha} g'(x)dx$$

where $\Gamma(.)$ is the Gamma function and $0 < \alpha \leq 1$.

Definition 2.2 The Mittag-Leffler function of parameter $\eta > 0$ specified by

$$E_{\eta}(x) = \sum_{i=0}^{\infty} \frac{x^i}{\Gamma(\eta i + 1)}.$$

3. Threshold Parameters and Equilibria

Clearly, system (2.1) has every time an infection-free equilibeium (IFE) $Q_0\left(X_{10} = \frac{\xi}{\mu_1}, 0, 0, 0, 0\right)$. Then the primary reproductive number of (2.1) is given by

$$B_r = \frac{k\rho X_{10}(\mu_2 h + r)}{au(\mu_2 + r)(1 + a_1 X_{10})}. \tag{3.1}$$

This first threshold parameter measures the mean number of the infections which right come after one productive infected cell starts producing the viruses throughout the duration of infection when all cells are still healthy. For $B_r > 1$, we discuss two cases:

- In absence of humoral immunity, system (2.1) has a only IE without humoral immunity $Q_1(X_{11}, X_{21}, X_{31}, X_{41}, 0)$, where

$$X_{11} = \frac{X_{10}\Big(au(\mu_2+r)+\xi k a_2(\mu_2 h+r)\Big)}{au(\mu_2+r)\Big(a_1X_{10}(B_r-1)+B_r\Big)+\mu_1 k a_2 X_{10}(\mu_2 h+r)},$$

$$X_{21} = \frac{a(1-h)X_{31}}{(\mu_2 h+r)},$$

$$X_{31} = \frac{(\xi-\mu_1 X_{11})(\mu_2 h+r)}{a(\mu_2+r)},$$

$$X_{41} = \frac{kX_{31}}{u}.$$

- In presence of humoral immunity, system (2.1) has a only IE with humoral immunity $Q_2(X_{12}, X_{22}, X_{32}, X_{42}, X_{52})$, where

$$X_{12} = \frac{\xi a_1-(\rho X_{42}+\mu_1 a_2 X_{42}+\mu_1)+\sqrt{(\rho X_{42}+\mu_1 a_2 X_{42}+\mu_1-\xi a_1)^2+4a_1\mu_1\xi(1+a_2X_{42})}}{2a_1\mu_1},$$

$$X_{22} = \frac{(1-h)\rho X_{12}X_{42}}{(\mu_2+r)(1+a_1X_{12}+a_2X_{42})},$$

$$X_{32} = \frac{(\mu_2 h+r)\rho X_{12}X_{42}}{a(\mu_2+r)(1+a_1X_{12}+a_2X_{42})},$$

$$X_{42} = \frac{b}{c},$$

$$X_{52} = \frac{u}{p}\Big(\frac{kX_{32}}{uX_{42}}-1\Big).$$

By a simple computation, we have $X_{52} = \frac{u}{p}(R_H - 1)$, where

$$R_H = \frac{k\rho X_{12}(\mu_2 h + r)}{au(\mu_2 + r)(1 + a_1X_{12} + a_2X_{42})}. \tag{3.2}$$

This second threshold parameter named the reproductive number for humoral immunity. Hence, Q_2 exists only when $R_H > 1$.

We sum up the above discussions in the following results.

Theorem 3.1

(i) *If $B_r \leq 1$, then system (2.1) has one IFE of the form $Q_0\Big(\frac{\xi}{\mu_1}, 0, 0, 0, 0\Big)$, where $X_{10} = \frac{\xi}{\mu_1}$.*

(ii) *If $B_r > 1$, then system (2.1) has an IE without humoral immunity of the form $Q_1 = (X_{11}, X_{21}, X_{31}, X_{41}, 0)$, where*

$$X_{11} = \frac{X_{10}\Big(au(\mu_2+r)+\xi k a_2(\mu_2 h+r)\Big)}{au(\mu_2+r)\Big(a_1X_{10}(B_r-1)+B_r\Big)+\mu_1 k a_2 X_{10}(\mu_2 h+r)},$$

$$X_{21} = \frac{a(1-h)X_{31}}{(\mu_2 h+r)},\ X_{31} = \frac{(\xi-\mu_1 X_{11})(\mu_2 h+r)}{a(\mu_2+r)},\ \text{and } X_{41} = \frac{kX_{31}}{u}.$$

(iii) *If $R_H > 1$, then system (2.1) has an IE with humoral immunity of the form $Q_2(X_{12}, X_{22}, X_{32}, X_{42}, X_{52})$, where*

$$X_{42} = \frac{b}{c},$$

$$X_{12} = \frac{\xi a_1-(\rho X_{42}+\mu_1 a_2 X_{42}+\mu_1)+\sqrt{(\rho X_{42}+\mu_1 a_2 X_{42}+\mu_1-\xi a_1)^2+4a_1\mu_1\xi(1+a_2X_{42})}}{2a_1\mu_1},$$

$$X_{22} = \frac{(1-h)\rho X_{12}X_{42}}{(\mu_2+r)(1+a_1X_{12}+a_2X_{42})},\ X_{32} = \frac{(\mu_2 h+r)\rho X_{12}X_{42}}{a(\mu_2+r)(1+a_1X_{12}+a_2X_{42})},\ \text{and } X_{52} = \frac{u}{p}(R_H-1).$$

4. Features of Solutions

In this part, for any positive initial conditions, the uniqueness, non-negativity, and boundedness of responses of system (2.1) will be set. Since model (2.1) represents the growth and evolution of the cell particles, hence, biologically it is acceptable that the number of cells must remain positive and bounded.

Lemma 4.1 *Consider the fractional order system*

$$D^{\eta}x(t) = g(x),$$

$$x(t_0) = x_0,$$

with $0 < \eta < 1, t_0 \in R$, *and* $x_0 \in R^n$. *Now assume that* g *satisfies the conditions below:*

a) g *and* ∂g *are continuous for all* $x \in R^n$ *and* g *is Lebesgue measurable with respect to* t.

b) $\| g(x) \| \leq \nu + \rho \| x \|$ *for all* $x \in R^n$, *where* ν *and* ρ *are two positive constants.*

Then, the above system has a unique solution on $[t_0, +\infty)$.

The proof follows sharply from [18].

Theorem 4.1 *All solutions of system* (2.1) *with non-negative initial conditions exist uniquely for all* $t > 0$ *and remain bounded and non-negative.*

Proof: Since (2.1), we have
$D^{\eta}X_1(t)\mid_{X_1=0} = \xi \geq 0$,
$D^{\eta}X_2(t)\mid_{X_2=0} = \frac{(1-h)\rho X_1 X_4}{1+a_1X_1+a_2X_4} \geq 0$, for all $X_1, X_4 \geq 0$,
$D^{\eta}X_3(t)\mid_{X_3=0} = \frac{h\rho X_1 X_4}{1+a_1X_1+a_2X_4} + rX_2 \geq 0$, for all $X_1, X_2, X_4 \geq 0$,
$D^{\eta}X_4(t)\mid_{X_4=0} = kX_3 \geq 0$, for all $X_3 \geq 0$,
$D^{\eta}X_5(t)\mid_{X_5=0} = 0$, for all $X_5 \geq 0$.
Hence, the nonnegativity of solutions is established.

Let $\Lambda(t) = \begin{bmatrix} X_1(t) \\ X_2(t) \\ X_3(t) \\ X_4(t) \\ X_5(t) \end{bmatrix}$ and $\chi = \begin{bmatrix} \xi \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$. Clearly, system (2.1) satisfies the first conditions of Lemma (4.1). For proving the second condition, consider cases as follows:

a) If $a_1 \neq 0$, then our system can be written as

$$D^{\eta}\Lambda(t) = \chi + H_1\Lambda + \frac{a_1X_1}{1+a_1X_1+a_2X_4}H_2\Lambda + X_5H_3\Lambda + X_4H_4\Lambda,$$

where

$$H_1 = \begin{bmatrix} -\mu_1 & 0 & 0 & 0 & 0 \\ 0 & -(\mu_2+r) & 0 & 0 & 0 \\ 0 & \delta & -a & 0 & 0 \\ 0 & 0 & k & -u & 0 \\ 0 & 0 & 0 & 0 & -b \end{bmatrix},$$

$$H_2 = \begin{bmatrix} 0 & 0 & 0 & \frac{-\rho}{a_1} & 0 \\ 0 & 0 & 0 & \frac{(1-h)\rho}{a_1} & 0 \\ 0 & 0 & 0 & \frac{h\rho}{a_1} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

$$H_3 = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -p \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

$$H_4 = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & c \end{bmatrix}.$$

So, we have

$$\| D^{\eta}\Lambda(t) \| \leq^{Triangle}_{Cauchy-Schwarz} \| \chi \| + \Big(\| H_1 \| + \| H_2 \| + \| X_5 \| \| H_3 \| + \| X_4 \| \| H_4 \| \Big) \| \Lambda \|.$$

b) If $a_2 \neq 0$, then

$$D^{\eta}\Lambda(t) = \chi + H_1\Lambda + \frac{a_2 X_1}{1 + a_1 X_1 + a_2 X_4} H_5\Lambda + X_5 H_3\Lambda + X_4 H_4\Lambda,$$

where

$$H_5 = \begin{bmatrix} 0 & 0 & 0 & \frac{-\rho}{a_2} & 0 \\ 0 & 0 & 0 & \frac{(1-h)\rho}{a_2} & 0 \\ 0 & 0 & 0 & \frac{h\rho}{a_2} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

Hence,

$$\| D^{\eta}\Lambda(t) \| \leq \| \chi \| + \Big(\| H_1 \| + \| H_5 \| + \| X_5 \| \| H_3 \| + \| X_4 \| \| H_4 \| \Big) \| \Lambda \|.$$

c) If $a_1 = a_2 = 0$, then

$$D^{\eta}\Lambda(t) = \chi + H_1\Lambda + X_4 H_6\Lambda + X_5 H_3\Lambda + X_4 H_4\Lambda,$$

where

$$H_6 = \begin{bmatrix} -\rho & 0 & 0 & 0 & 0 \\ (1-h)\rho & 0 & 0 & 0 & 0 \\ h\rho & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

Thus,

$$\| D^{\eta}\Lambda(t) \| \leq \| \chi \| + \Big(\| X_4 \| (\| H_6 \| + \| H_4 \|) + \| H_1 \| + \| X_5 \| \| H_3 \| \Big) \| \Lambda \|.$$

Therefore, our system has a unique solution on $[0, \infty)$.

Consider the function below:

$$N_1(t) = X_1(t) + X_2(t) + X_3(t).$$

We have

$$\begin{aligned} D^{\eta}N_1(t) &= \xi - \mu_1 X_1 - \mu_2 X_2 - a X_3 \\ &\leq \xi - p_1 N_1(t), \end{aligned}$$

where $p_1 = \min\{\mu_1, \mu_2, a\}$

Hence,

$$N_1(t) \leq N_1(0) E_{\eta}(-p_1 t^{\eta}) + \frac{\xi}{p_1}\Big(1 - E_{\eta}(-p_1 t^{\eta})\Big).$$

Since $0 \leq E_{\eta}(-p_1 t^{\eta}) \leq 1$, we get

$$N_1(t) \leq N_1(0) + \frac{\xi}{p_1}.$$

Therefore, $X_1(t)$, $X_2(t)$, $X_3(t)$ are bounded.

Now assume the following function:

$$N_2(t) = X_4(t) + \frac{p}{c}X_5(t).$$

We have

$$D^\eta N_2(t) = kX_3 - uX_4 - \frac{bp}{c}X_5 \leq kL_1 - p_2\left(X_4 + \frac{p}{c}X_5\right) = kL_1 - p_2N_2,$$

$$D^\eta N_2(t) \leq kL_1 - p_2N_2(t),$$

where $p_2 = \min\{u, b\}$. Hence,

$$N_2(t) \leq N_2(0)E_\eta(-p_2t^\eta) + \frac{kL_1}{p_2}(1 - E_\eta(-p_2t^\eta)).$$

Since $0 \leq E_\eta(-p_2t^\eta) \leq 1$, we get

$$N_2(t) \leq N_2(0) + \frac{kL_1}{p_2}.$$

So $X_4(t), X_5(t)$ are bounded and this ends the proof. □

5. Globally Stable Concepts

Clearly, the portraying ordinary differential equations (ODEs) of system (2.1) is given by

$$\dot{u} = f(u), \tag{5.1}$$

where $u = \begin{bmatrix} X_1 \\ X_2 \\ X_3 \\ X_4 \\ X_5 \end{bmatrix}$ and $f(u) = \begin{bmatrix} \xi - \mu_1X_1 - \frac{\rho X_1X_4}{1+a_1X_1+a_2X_4} \\ \frac{(1-h)\rho X_1X_4}{1+a_1X_1+a_2X_4} - (\mu_2+r)X_2 \\ \frac{h\rho X_1X_4}{1+a_1X_1+a_2X_4} + rX_2 - aX_3 \\ kX_3 - uX_4 - pX_4X_5 \\ cX_4X_5 - bX_5 \end{bmatrix}$.

Hence, system (2.1) can be written as

$$D_t^\eta u = f(u).$$

where D_t^η is the fractional derivative in the Caputo sense of order $\eta \in (0, 1]$. For $\eta = 1$, we get the ODE model (5.1).

Theorem 5.1 *If $B_r \leq 1$, then Q_0 is globally asymptotically stable (GAS).*

Proof: We define Lyapunov functional as follows

$$L_0(u) = \frac{X_{10}}{1 + a_1X_{10}}\phi\left(\frac{X_1}{X_{10}}\right) + \frac{r}{\mu_2h + r}X_2 + \frac{(\mu_2 + r)}{\mu_2h + r}X_3 + \frac{a(\mu_2 + r)}{k(\mu_2h + r)}X_4 + \frac{ap(\mu_2 + r)}{kc(\mu_2h + r)}X_5.$$

where $\phi(\xi) = \xi - 1 - \ln(\xi) \geq 0$, $\xi > 0$. Based on the results of [10], we infer that L_0 is a Lyapunov functional at Q_0 when $B_r \leq 1$. Moreover, we have

$$D_t^\eta L_0(u) \leq \nabla L_0(u).f(u).$$

By using Theorem 1 (i) in [20], we conclude that L_0 is also a Lyapunov functional for FDE model (2.1) at Q_0 when $B_r \leq 1$. Therefore, Q_0 is GAS when B_r is less than unity or maximum one. □

Theorem 5.2 *If $R_H \leq 1 < B_r$, then Q_1 is GAS.*

Proof: We consider the following Lyapunov functional

$$L_1(u) = X_1 - X_{11} - \int_{X_{11}}^{X_1} \frac{I(X_{11},X_{41})}{I(\theta,X_{41})} d\theta + \frac{r}{\mu_2 h + r} X_{21} \phi\left(\frac{X_2}{X_{21}}\right) + \frac{\mu_2 + r}{\mu_2 h + r} X_{31} \phi\left(\frac{X_3}{X_{31}}\right) + \frac{a(\mu_2 + r)}{k(\mu_2 h + r)} X_{41} \phi\left(\frac{X_4}{X_{41}}\right) + \frac{ap(\mu_2 + r)}{kc(\mu_2 h + r)} X_5.$$

where $I(X_1, X_4) = \frac{\rho X_1}{1 + a_1 X_1 + a_2 X_4}$, $\phi(\xi) = \xi - 1 - \ln(\xi) \geq 0$, $\xi > 0$. By means of [10], we get that L_1 is a Lyapunov functional at Q_1 when $R_H \leq 1 < B_r$. Furthermore, we have

$$D_t^{\eta} L_1(u) \leq \nabla L_1(u).f(u).$$

By applying Theorem 1 (i) of [20], we infer that L_1 is also a Lyapunov functional for FDE model (2.1) at Q_1 when $R_H \leq 1 < B_r$. Thus, Q_1 is GAS when B_r is greater than unity, and R_H is less than unity or maximum one. □

Theorem 5.3 *If $R_H > 1$, then Q_2 is GAS.*

Proof: Assume the following Lyapunov functional

$$L_2(u) = X_1 - X_{12} - \int_{X_{12}}^{X_1} \frac{I(X_{12},X_{42})}{I(\theta,X_{42})} d\theta + \frac{r}{\mu_2 h + r} X_{22} \phi\left(\frac{X_2}{X_{22}}\right) + \frac{\mu_2 + r}{\mu_2 h + r} X_{32} \phi\left(\frac{X_3}{X_{32}}\right) + \frac{a(\mu_2 + r)}{k(\mu_2 h + r)} X_{42} \phi\left(\frac{X_4}{X_{42}}\right) + \frac{ap(\mu_2 + r)}{kc(\mu_2 h + r)} X_{52} \phi\left(\frac{X_5}{X_{52}}\right).$$

where $I(X_1, X_4) = \frac{\rho X_1}{1 + a_1 X_1 + a_2 X_4}$, $\phi(\xi) = \xi - 1 - \ln(\xi) \geq 0$, $\xi > 0$. By means of [10], we get that L_2 is a Lyapunov functional at Q_2 when $R_H > 1$. Furthermore, we have

$$D_t^{\eta} L_2(u) \leq \nabla L_2(u).f(u).$$

By applying Theorem 1 (i) of [20], we infer that L_2 is also a Lyapunov functional for FDE model (2.1) at Q_2 when $R_H > 1$. Thus, Q_2 is GAS when R_H is greater than unity. □

6. Embodied Numeric Comparisons

In this part, we give some numerical simulations to depict our scientific yields. We assume cases for $\eta = 0.5, 0.7, 0.8, 0.9, 1$. We select the values of parameters observable in the Table below.

Table: Parameter Values used for simulations

Parameters	Meaning	Value	References
ξ	Continuous rate of output of CD4 target cells	10 $cells\ mm^{-3} day^{-1}$	[10]
μ_1	Decay rate of susceptible cells	0.01 day^{-1}	[10]
ρ	Viral infectivity rate	0.001,0.005 $mm^3\ virus^{-1}\ day^{-1}$	Varied
a_1	Positive parameter that describes capture rate's effects	0.1 $cells^{-1}\ mm^3$	[10]
a_2	Positive parameter that describes capture rate's effects	0.1 $virus^{-1}\ mm^3$	[10]
μ_2	Death rate of latent infectious cells	0.1 day^{-1}	[10]
r	Transmission rate of latent infectious cell to active infectious cell	0.2 day^{-1}	[10]
a	Death rate of active infectious cells	0.1 day^{-1}	[10]
k	Productive rate of virus from activated infected cells	10 $virus\ cells^{-1}\ day^{-1}$	[10]
u	Clearing rate of virus	3 day^{-1}	[10]
h	The probability of a healthy cell turns to latently infectious or actively infectious	0.5 day^{-1}	[10]
p	Clearing rate of virus killing by B cells	0.01 $cells^{-1}\ mm^3\ day^{-1}$	[10]
c	Activation rate of B cells	0.001, 0.005 $virus^{-1}\ mm^3\ day^{-1}$	Varied
b	Dying rate of B cells	0.2 day^{-1}	[10]

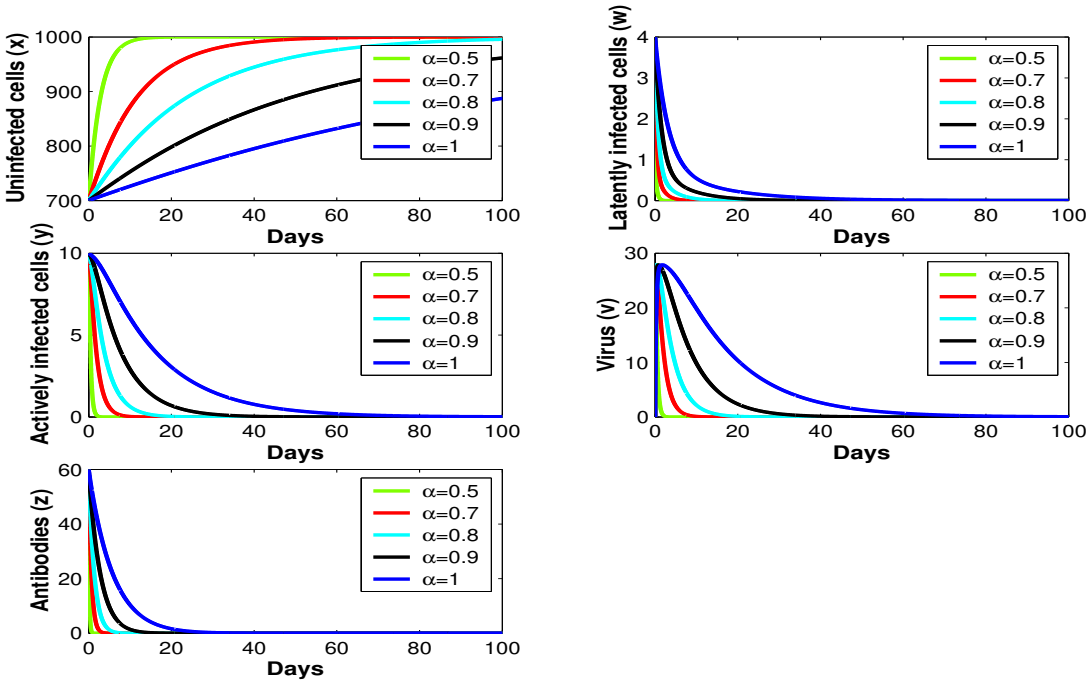

Figure 1: Stable inclination of plots towards the IFE Q_0 for distinct values of $\alpha = \eta$, and $x = X_1, w = X_2, y = X_3, v = X_4$, and $z = X_5$ related to system (2.1).

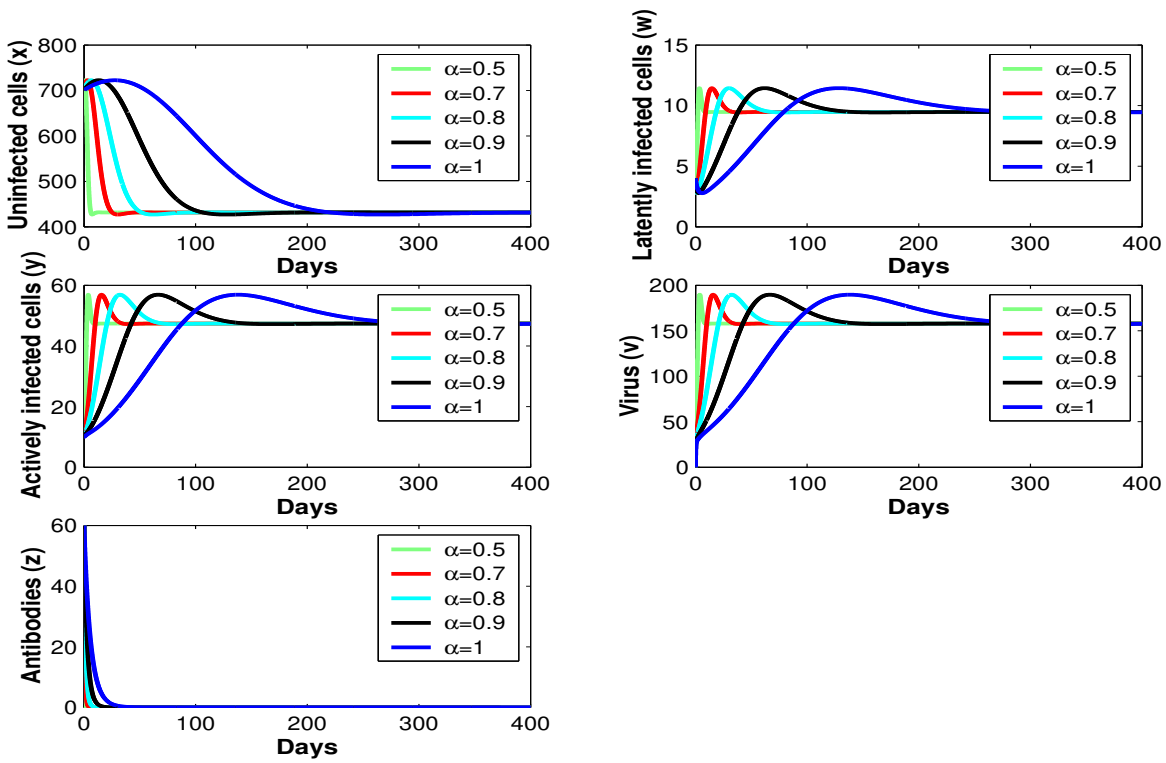

Figure 2: Global asymptotic stability of the IE without humoral immunity Q_1 for distinct values of $\alpha = \eta$, and $x = X_1, w = X_2, y = X_3, v = X_4$, and $z = X_5$ related to system (2.1).

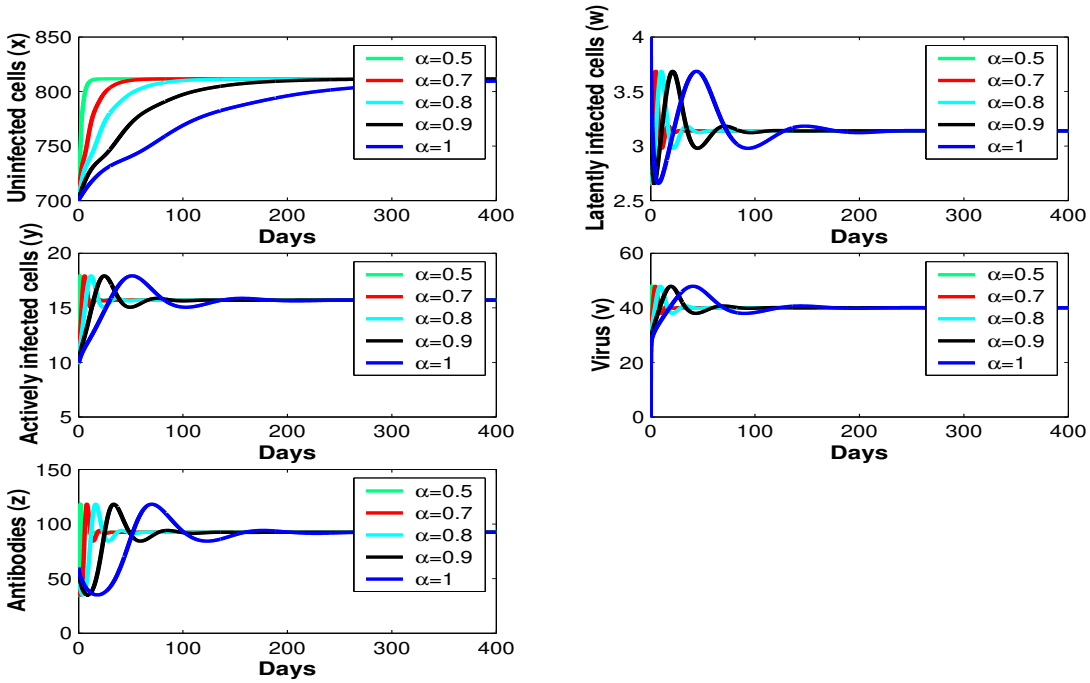

Figure 3: Global asymptotic stability of the IE with activated humoral immunity Q_2 for distinct values of $\alpha = \eta$, and $x = X_1, w = X_2, y = X_3, v = X_4$, and $z = X_5$ related to system (2.1).

If we select the values of parameters shown in Table 1, choose $\rho = 0.001$ and $c = 0.001$, we calculate $B_r = 0.2750 \leq 1$. Then system (2.1) has a IFE $Q_0(1000, 0, 0, 0, 0)$. By Theorem 5.1, the response of system (2.1) converges to Q_0 (see Fig.1). Based on Fig.1, the density of uninfected cells is increasing and tends to its normal value $\xi/\mu_1 = 1000\ cells\ mm^{-3}$, while the density of latent infectious cells, active infectious cells and free viruses are declining greatly and tend to zero. As a consequence, the virus is removed and the infection ends.

If we take $\rho = 0.005$, $c = 0.001$ and fix the other parameter values, we obtain $B_r = 1.3751 > 1$ and $R_H = 0.2220 < 1$. Therefore, the stationary point $Q_1(431.6667, 9.4722, 47.3611, 157.8704, 0)$ is GAS that means that the virus insists in the body generously and infection turns to chronic. By Theorem 5.2, the response of system (2.1) meets Q_1. Fig.2 depicts this resultant.

If we take $\rho = 0.005$, $c = 0.005$ and fix the other parameter values, we obtain $B_r = 1.3751 > 1$ and $R_H = 1.3082 > 1$. Therefore, the stationary point $Q_2(811.6062, 3.1399, 15.6995, 40.0000, 92.4870)$ is GAS that means that the virus insists in the body and infection turns to chronic. By Theorem 5.3, the response of system (2.1) joins to Q_2. Fig.3 shows this fact.

7. Drawn Deductions

In this paper, we have proposed a fractional HIV infected model with five main compartments that are target host cells, latent mood of infectious cells which is the state that the infected cells are unable to produce new infectious viruses, activated infected cells, matured viruses, and antibodies. We used specific incidence rate of type Beddington-DeAngelis as functional response. We derived two threshold parameters, the primary infection reproductive number B_r and the reproductive number for humoral immune response R_H. Under defined presumptions, it is shown that the proposed model has a bounded and nonnegative response as desired in any population dynamics. By using stability analysis of Caputo fractional derivative order system, we have proved that if the primary reproductive number $B_r \leq 1$, then the uninfected steady state is GAS for all $\eta \in (0, 1]$. consequently the viruses are unable to invade the target cells and be cleared hence, using antiviral drug treatment can control and prevent the infection. If $R_H \leq 1 < B_r$, then the IE without humoral immune response is GAS. This advanced stage includes a

time period so-called open-window period due to low levels of antibodies and viruses have strong ability to invade the host and weaken the immune system. To stop replicating the viruses in the body, HIV medicines and medical devices are essential approaches to fight HIV. If R_H is greater than one, then IE with humoral immune response is GAS. In this case, HIV can become AIDS and using antiretroviral therapy can affect on life expectancy. Based on the above theoretical analysis, we realize that the global dynamics of the model are completely determined by computations of the reproductive numbers B_r and R_H. Furthermore, we observe that the fractional η does not affect on our model related to global dynamics, but it can affect the time for reaching the steady states (see Fig.1, Fig.2, and Fig.3).

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This section should come before the References and should be unnumbered. Funding information may also be included here.

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