



Research article

Study of HIV model via recent improved fractional differential and integral operators

Abd-Allah Hyder^{1,2}, Mohamed A. Barakat^{3,4}, Doaa Rizk⁵, Rasool Shah⁶ and Kamsing Nonlaopon^{7,*}

¹ Department of Mathematics, College of Science, King Khalid University, P. O. Box 9004, Abha 61413, Saudi Arabia

² Department of Engineering Mathematics and Physics, Faculty of Engineering, Al-Azhar University, Cairo 11371, Egypt

³ Department of Computer Science, College of Al Wajh, University of Tabuk, Tabuk 71491, Saudi Arabia

⁴ Department of Mathematics, Faculty of Sciences, Al-Azhar University, Assiut 71524, Egypt

⁵ Department of Mathematics, College of Science and Arts, Qassim University, Al-Asyah, Saudi Arabia

⁶ Department of Mathematics, Abdul Wali khan university, Mardan 23200, Pakistan

⁷ Department of Mathematics, Faculty of Science, Khon Kaen University, Khon Kaen 40002, Thailand

* **Correspondence:** Email: nkamsi@kku.ac.th.

Abstract: In this article, a new fractional mathematical model is presented to investigate the contagion of the human immunodeficiency virus (HIV). This model is constructed via recent improved fractional differential and integral operators. Other operators like Caputo, Riemann-Liouville, Katugampola, Jarad and Hadamard are being extended and generalized by these improved fractional differential and integral operators. Banach's and Leray-Schauder nonlinear alternative fixed point theorems are utilized to examine the existence and uniqueness results of the proposed fractional HIV model. Moreover, different kinds of Ulam stability for the fractional HIV model are established. It is simple to recognize that the extracted results can be reduced to some results acquired in multiple works of literature.

Keywords: human immunodeficiency virus; fractional mathematical modeling; fractional operators

Mathematics Subject Classification: 26A33, 68Q07, 34E18

1. Introduction

Viruses and bacteria are the primary causes of a wide range of deadly infectious illnesses. Infectious diseases have a significant influence on society. These diseases accounting for one-fourth of all fatalities worldwide. In terms of viral infection, the Human Immunodeficiency Virus (HIV), which is a retrovirus that causes acquired immunodeficiency syndrome (AIDS), continues to be a global health issue, having claimed the lives of 36.3 million people so far [1, 2]. HIV targets the human body's immune system, especially damaging CD4⁺ T-cells (CD4-PTC), which are the immune system's key component. As the virus weakens the body's immunity, the infected individual becomes more susceptible to other diseases. When HIV enters a human, the virus attacks and multiplies CD4-PTC cells [3–5].

There are several phases to HIV life cycle [6]. Firstly, HIV binds to receptors on CD4-PTC, the envelope of the virus begins to fuse with the membrane of the CD4-PTC. This stage is called binding and fusion and in which the virus can enter the cell. Secondly, HIV releases and employs the reverse transcriptase enzyme within the CD4-PTC to transform its genetic material from RNA into DNA. This stage is called reverse transcription which allows HIV to reach the nucleus of the CD4-PTC. Thirdly, HIV releases an additional enzyme called integrase into the nucleus of the CD4-PTC. The virus uses this enzyme to connect its DNA to the DNA of the CD4-PTC. At this stage, the virus is deemed dormant, and even advanced lab tests have trouble in finding it. This stage can be called integration and transcription. Fourthly, because HIV has been integrated into the DNA of the CD4-PTC, it may now be able to utilize its machinery to create viral proteins. During this moment, HIV can also produce further of its genetic (RNA). It is conceivable for it to produce more viral particles because of these two factors. This stage is called replication. Fifthly, the new HIV proteins and RNA are delivered to the border of the CD4-PTC, where they develop into immature HIV. These viruses are not infectious in their present condition and this stage can be named the assembly. Finally, immature viruses take their way out of the CD4-PTC. Then, they release their protease enzyme, which alters the virus's proteins and transforms them into an infectious form. This stage can be mentioned as budding. Antiretroviral treatment (ART) is recognized as the employing of HIV medicines to cure HIV infection and save the immune system by prohibiting the virus from reproducing at diverse stages of its life cycle, like protease inhibitors, integrase inhibitors, reverse transcription inhibitors, and fusion inhibitors [7–9].

Mathematical models with simulations were utilized as an important tool to foresee the potential and severity of infections and to learn about the infection's dynamic behavior. These models are helpful tools that play an important role in explaining the dynamics of the immune response to HIV infection. We suggest readers to certain works, see [10–12]. According to some previous works on HIV model, scholars commonly employed systems of differential equations to demonstrate the relationship between HIV and uninfected CD4-PTC, as well as the effect of drug therapy on infected cells. To examine various dynamic behaviors of HIV infection of CD4-PTC, a simple model for HIV infection was proposed in [13]. Tuckwell et al. [14] proposed a two-component mathematical model for the early dynamics for HIV of first type. Despite the fact that their model can be numerically solved, there are general theoretical inferences available due to nonlinear effects. In [15], Rong et al. have employed a mathematical model to investigate the early restrictions that may form a viral resistance to antiretroviral drugs. Srivastava et al. [16] have suggested and investigated a primary HIV infection model throughout treatment. Only reverse transcription inhibitors were included in their model, and

the medication therapy model has been constructed appropriately.

Fractional calculus has played an important role in science and engineering, and many mathematicians and scientists have recently been working in this area. Fractional calculus has been applied in physics, biology, engineering, and other fields in recent decades [17]. The most significant conceptions of fractional derivatives with numerous scopes in literature are those of Riemann, Hilfer, Caputo, and Antagana Baleanu [18–21]. The literature [22–32] contains further information on fractional calculus and its applications. Employing fractional calculus to investigate HIV model has resulted in a variety of research findings. Lichae et al. [33] have investigated an HIV-1 infection model of CD4-PTC and described the effect of antiviral medication therapy using Caputo fractional derivative. Using the Laplace transform and the Adomian decomposition approach, an approximate solution was obtained. Using Caputo fractional derivative, Ferrari et al. [34] have developed an HIV model in which the presence of a reverse transcriptase inhibitor is indicated based on [16]. They demonstrated the model's existence and uniqueness, as well as its positive invariance. The model's equilibrium points and stability are also explored. Nowadays, the qualitative theory for fractional mathematical models has caught the interest of researchers. In 2020, Nazir et al. [35] have examined an HIV model with the fractional Caputo-Fabrizio derivative. They determined some existence conditions for solutions via the fixed point technique. Khan et al. [36] have investigated and examined some existence and stability findings for a fractional HIV-TB model based on Mittag-Leffler function. Numerical solutions are also achieved. In [37] Kongson et al. have used a generalized Caputo fractional derivative to present and analyze a mathematical model of HIV infections with antiretroviral medication and so on [38–40].

In the present work, motivated by the studies [37, 41] we utilize recent improved fractional differential and integral operators to construct a new fractional mathematical model for HIV contagion. This model contains four fractional nonlinear differential equations under the improved fractional derivatives. By using Banach's and Leray-Schauder nonlinear alternative fixed point theorems, we examine the existence and uniqueness results of the proposed fractional HIV model for the suggested fractional HIV model. Also, we investigate different kinds of Ulam stability for this fractional HIV model. Moreover, we show that the acquired results can be diminished to some results obtained in many past research works.

2. Fundamental instruments

2.1. The improved fractional operators

This part contains notations, definitions, and essential findings for the improved fractional integral and derivative operators, which will be relevant throughout this study.

Definition 2.1 ([41]). Let $\eta \in \mathbb{C}$, $\text{Re}(\eta) > 0$ and $\theta \in (0, 1]$. The improved fractional integral operator of the function y is defined by:

$$\left({}^{\eta}\mathbb{I}_{\xi}^{\theta}y\right)(t) = \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \frac{y(u)}{\xi(u, \theta)} du, \quad t \geq 0, \quad (2.1)$$

where Γ denotes the Gamma function, $\Psi(t, u, \theta) = \int_u^t \frac{dv}{\xi(v, \theta)}$ and ξ is a continuous map from $\mathbb{R}_+ \times (0, 1]$ into \mathbb{R} with the properties: $\xi(t, 1) = 1$ for all $t \in \mathbb{R}_+$, $\xi(t, \theta) \neq 0$ for all $(t, \theta) \in \mathbb{R}_+ \times (0, 1]$ and $\xi(\cdot, \theta_1) \neq \xi(\cdot, \theta_2)$ whenever $\theta_1 \neq \theta_2$.

Definition 2.2 ([41]). The improved fractional derivative operator of the function y , in Riemann-Liouville manner, is defined by:

$$\begin{aligned}({}^{\eta}\mathbb{D}_{\xi}^{\theta}y)(t) &= O^n ({}^{n-\eta}\mathbb{I}^{\theta,\xi}y)(t) \\ &= \frac{O^n}{\Gamma(n-\eta)} \int_0^t \Psi^{n-\eta-1}(t,u,\theta) \frac{y(u)}{\xi(u,\theta)} du, \quad t \geq 0,\end{aligned}\tag{2.2}$$

where $O = \xi(t,\theta) \frac{d}{dt}$.

Definition 2.3 ([41]). The improved fractional derivative operator of the function y , in Caputo sense, is defined by:

$$({}^{\eta}\mathbb{D}_{\xi}^{\theta}y)(t) = \left({}^{\eta}\mathbb{D}_{\xi}^{\theta} \left(y(s) - \sum_{k=0}^{n-1} \frac{O^k y(0)}{k!} \Psi^k(s,0,\theta) \right) \right) (t),\tag{2.3}$$

where $n = [\text{Re}(\eta)] + 1$. If $\eta \in (0, 1)$, we have

$$({}^{\eta}\mathbb{D}_{\xi}^{\theta}y)(t) = ({}^{\eta}\mathbb{D}_{\xi}^{\theta}(y(s) - y(0)))(t).\tag{2.4}$$

An alternative form for the improved Caputo fractional derivative operator is given by the following theorems.

Theorem 2.1 ([41]). Let $\theta > 0$, $\text{Re}(\eta) > 0$, $a > 0$ and $n = [\text{Re}(\eta)] + 1$. If $y \in W_{\xi}^{n,\theta}([0, a])$, then the improved fractional derivative operator of the function y , in Caputo sense, is alternatively given by:

$$({}^{\eta}\mathbb{D}_{\xi}^{\theta}y)(t) = \frac{1}{\Gamma(n-\eta)} \int_0^t \Psi^{n-\eta-1}(t,u,\theta) \frac{(O^n y)(u)}{\xi(u,\theta)} du.\tag{2.5}$$

Theorem 2.2 ([41]). Let $\theta > 0$, $\text{Re}(\eta) > 0$, $a > 0$ and $n = [\text{Re}(\eta)] + 1$. If $y \in W_{\xi}^{n,\theta}([0, a])$, then the following equality holds:

$$({}^{\eta}\mathbb{I}_{\xi}^{\theta} {}^{\eta}\mathbb{D}_{\xi}^{\theta}y)(t) = y(t) - \sum_{k=0}^{n-1} \frac{O^k y(0)}{k!} \Psi^k(t,0,\theta).\tag{2.6}$$

Epecially, If $\eta \in (0, 1)$, we have

$$({}^{\eta}\mathbb{I}_{\xi}^{\theta} {}^{\eta}\mathbb{D}_{\xi}^{\theta}y)(t) = y(t) - y(0).\tag{2.7}$$

2.2. Model description

The current work relates to HIV models presented in [16, 34, 37], which are deemed the antiretroviral remedy of reverse transcriptase inhibitor. The following are the unknown variables and the deemed positive parameters that might be included in the model:

$X(t)$:	The number of susceptible CD4-PTC.
$Y(t)$:	The number of infected CD4-PTC prior to reverse transcription (pre-RT category).
$Z(t)$:	The number of infected CD4-PTC that have completed reverse transcription (post-RT category) and they are able to produce virus.
$W(t)$:	The density of the virus.
γ :	The influx rate of CD4-PTC.
l :	The rate of interaction-infection for CD4-PTC.
π_1 :	The normal death rate of CD4-PTC.
μ :	The effectiveness of RT inhibitor ($0 < \mu < 1$).
ν :	The transmission rate of pre-RT category infected CD4-PTC to pose-RT category.
p :	The rate at which infected cells revert to the uninfected state due to incomplete reverse transcription.
π_2 :	The rate of death for infected CD4-PTC.
ϕ :	The rate of death for actively infected CD4-PTC.
M :	The overall number of viral particles generated by the infected CD4-PTC.
q :	The riddance rate of the virus.

In light of all the above parameters and functions, we will discuss the next HIV model infection with CD4-PTC

$$\begin{cases} \left({}^{\eta}\mathcal{D}_{\xi}^{\theta}X\right)(t) = \gamma - lW(t)X(t) - \pi_1X(t) + (\mu\nu + p)Y(t), \\ \left({}^{\eta}\mathcal{D}_{\xi}^{\theta}Y\right)(t) = lW(t)X(t) - (\pi_2 + \nu + p)Y(t), \\ \left({}^{\eta}\mathcal{D}_{\xi}^{\theta}Z\right)(t) = (1 - \mu)\nu Y(t) - \phi Z(t), \\ \left({}^{\eta}\mathcal{D}_{\xi}^{\theta}W\right)(t) = M\phi Z(t) - qW(t), \end{cases} \quad (2.8)$$

where ${}^{\eta}\mathcal{D}_{\xi}^{\theta}$ is the recent improved fractional derivatives in Caputo sense. For easiness, we reset model (2.8) as the next form:

$$\begin{cases} \left({}^{\eta}\mathcal{D}_{\xi}^{\theta}X\right)(t) = \Lambda_1(t, X, Y, Z, W), \\ \left({}^{\eta}\mathcal{D}_{\xi}^{\theta}Y\right)(t) = \Lambda_2(t, X, Y, Z, W), \\ \left({}^{\eta}\mathcal{D}_{\xi}^{\theta}Z\right)(t) = \Lambda_3(t, X, Y, Z, W), \\ \left({}^{\eta}\mathcal{D}_{\xi}^{\theta}W\right)(t) = \Lambda_4(t, X, Y, Z, W), \end{cases} \quad (2.9)$$

where $\Lambda_i (i = 1, 2, 3, 4)$ are nonlinear functions given by:

$$\begin{cases} \Lambda_1(t, X, Y, Z, W) = \gamma - lW(t)X(t) - \pi_1X(t) + (\mu\nu + p)Y(t), \\ \Lambda_2(t, X, Y, Z, W) = lW(t)X(t) - (\pi_2 + \nu + p)Y(t), \\ \Lambda_3(t, X, Y, Z, W) = (1 - \mu)\nu Y(t) - \phi Z(t), \\ \Lambda_4(t, X, Y, Z, W) = M\phi Z(t) - qW(t), \end{cases} \quad (2.10)$$

with the conditions $(X(0), Y(0), Z(0), W(0))^{\mathcal{T}} = (X_0, Y_0, Z_0, W_0)^{\mathcal{T}}$ and the superscript \mathcal{T} denotes the vector transpose.

3. Existence and uniqueness results

Using some fixed point theorems, this section explores the existence and uniqueness of solution to the given model (2.8).

Assume $q \in (0, \infty)$, $\beta = (X, Y, Z, W)^T$ and $\Omega(t, \beta(t)) = (\lambda_i(t, X, Y, Z, W))$, $i = 1, 2, 3, 4$. Consider the Banach space $\mathbb{B} = C([0, q], \mathbb{R})$ of all continuous functions $\beta : [0, q] \rightarrow \mathbb{R}$ with the norm

$$\|\beta\| = \sup_{t \in [0, q]} |\beta(t)|, \quad (3.1)$$

where $|\beta(t)| = |X(t)| + |Y(t)| + |Z(t)| + |W(t)|$ and $X, Y, Z, W \in \mathbb{B}$. Therefore, we can write the model (2.8) as the next initial value problem:

$$\begin{cases} ({}^{\eta} \mathbb{D}_{\xi}^{\theta} \beta)(t) = \Omega(t, \beta(t)), \\ \beta(0) = \beta_0, \end{cases} \quad (3.2)$$

where $\beta_0 = (X_0, Y_0, Z_0, W_0)^T$.

According to Theorem 2.2, the problem (3.2) can be replaced equivalently by the following integral equation:

$$\beta(t) = \beta_0 + \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \frac{\Omega(u, \beta(u))}{\xi(u, \theta)} du. \quad (3.3)$$

Define an operator $\mathbb{T} : \mathbb{B} \rightarrow \mathbb{B}$ by

$$(\mathbb{T}\beta)(t) := \beta_0 + \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \frac{\Omega(u, \beta(u))}{\xi(u, \theta)} du. \quad (3.4)$$

Therefore, if \mathbb{T} has a fixed point, the equivalent problem (3.2) has a unique solution.

3.1. Existence result

Using Leray-Schauder nonlinear alternative, this subsection demonstrates the existence of solutions for model (2.8).

Theorem 3.1. *Suppose that:*

(S₁) \exists a function $\zeta \in C([0, T], \mathbb{R}^+)$ and a nondecreasing continuous function $\mathcal{Y} : [0, \infty) \rightarrow [0, \infty)$, such that

$$\begin{cases} \mathcal{Y}(q\beta) \leq q\mathcal{Y}(\beta), & \text{for all } q \geq 1 \text{ and } \beta \in \mathbb{B}, \\ \|\Omega(t, \beta(t))\| \leq \zeta(t)\mathcal{Y}(\|\beta(t)\|) & \text{foreach } (t, \beta) \in [0, T] \times \mathbb{R}^4. \end{cases} \quad (3.5)$$

(S₂) \exists a constant $G_1 > 0$ such that

$$\|\beta_0\| + \frac{\zeta_0 \mathcal{Y}(G_1) T^{\eta\theta}}{\theta^{\eta} \Gamma(\eta + 1)} < M_2, \quad (3.6)$$

where $\zeta_0 = \sup_{t \in [0, T]} \{\zeta(t)\}$. Then, the problem (3.2), as well as the model (2.8), has one solution on $[0, T]$ at least.

Proof. Let $\mathbb{S}_{\omega_1} = \{\beta \in \mathbb{B} : \|\beta\| \leq \omega_1\}$ be a bounded ball in \mathbb{B} with $\omega_1 > 0$. From the stipulation (S₁), for $t \in [0, T]$, we get

$$|(\mathbb{T}\beta)(t)| \leq \|\beta_0\| + \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \|\Omega(u, \beta(u))\| \frac{du}{\xi(u, \theta)}$$

$$\leq \|\beta_0\| + \frac{\zeta_0 \mathcal{Y}(\|\beta\|) T^{\eta\theta}}{\theta^\eta \Gamma(\eta + 1)}, \quad (3.7)$$

which implies that

$$\|\mathbb{T}\beta\| \leq \|\beta_0\| + \frac{\zeta_0 \mathcal{Y}(\omega_1) T^{\eta\theta}}{\theta^\eta \Gamma(\eta + 1)}. \quad (3.8)$$

Therefore, the operator \mathbb{T} transfers any bounded ball in \mathbb{B} into a bounded ball. Now, if $\sigma_1, \sigma_2 \in [0, T]$ with $\sigma_1 < \sigma_2$ and $\beta \in \mathbb{S}_{\omega_1}$. Then, we have

$$\begin{aligned} & |(\mathbb{T}\beta)(\sigma_2) - (\mathbb{T}\beta)(\sigma_1)| \\ &= \left| \frac{1}{\Gamma(\eta)} \int_0^{\sigma_2} \Psi^{\eta-1}(\sigma_2, u, \theta) \Omega(u, \beta(u)) \frac{du}{\xi(u, \theta)} - \frac{1}{\Gamma(\eta)} \int_0^{\sigma_1} \Psi^{\eta-1}(\sigma_1, u, \theta) \Omega(u, \beta(u)) \frac{du}{\xi(u, \theta)} \right| \\ &\leq \frac{\zeta_0 \mathcal{Y}(\|\beta\|)}{\theta^\eta \Gamma(\eta + 1)} \left(|\sigma_2^{\eta\theta} - \sigma_1^{\eta\theta}| + 2 |\sigma_2^\theta - \sigma_1^\theta|^\eta \right). \end{aligned} \quad (3.9)$$

Obviously, the right-hand side of (3.9) approaches zero if $\sigma_2 \rightarrow \sigma_1$. Hence, the operator \mathbb{T} transfers any bounded set into an equicontinuous set in \mathbb{B} . Also, by Arzelá-Ascoli theorem, the operator $\mathbb{T} : \mathbb{B} \rightarrow \mathbb{B}$ is completely continuous. Now, let $\beta \in \mathbb{B}$ be a solution to $\beta = p\mathbb{T}\beta$ for $0 < p < 1$. Then, for $t \in [0, T]$, we have

$$|\beta(t)| = |p(\mathbb{T}\beta)(t)| \leq \|\beta_0\| + \frac{\zeta_0 \mathcal{Y}(\|\beta\|) T^{\eta\theta}}{\theta^\eta \Gamma(\eta + 1)}. \quad (3.10)$$

Therefore, we get

$$\|\beta\| \leq \|\beta_0\| + \frac{\zeta_0 \mathcal{Y}(\|\beta\|) T^{\eta\theta}}{\theta^\eta \Gamma(\eta + 1)}. \quad (3.11)$$

According to the stipulation (S_2) , we have a constant $G_1 > 0$ such that $\|\beta\| \neq G_1$. Assume $\mathbb{V} := \{\beta \in \mathbb{B} : \|\beta\| < G_1\}$. It is evident that the operator $\mathbb{T} : \mathbb{V} \rightarrow \mathbb{C}$ is completely continuous. From the shape of \mathbb{V} , there is no $\beta \in \partial\mathbb{V}$ such that $\beta = p\mathbb{T}\beta$ for some $0 < p < 1$. Therefore, by Leray-Schauder nonlinear alternative, we conclude that the operator \mathbb{T} has a fixed point $\beta \in \mathbb{V}$ and the model (2.8) has at least one solution on $[0, T]$. This finishes the proof. \square

3.2. Uniqueness result

This subsection exhibits the uniqueness of the solution of model (2.8) by utilizing Banach fixed point theorem.

Theorem 3.2. *Let $\Omega : [0, T] \times \mathbb{B} \rightarrow \mathbb{R}^4$ be continuous function achieving the next stipulation.*

$(S_3) \exists$ a constant Υ_Ω such that

$$\|\Omega(t, \beta_1(t)) - \Omega(t, \beta_2(t))\| \leq \Upsilon_\Omega \|\beta_1(t) - \beta_2(t)\|, \quad (3.12)$$

for all $\beta_1, \beta_2 \in \mathbb{B}$ and $t \in [0, T]$. If

$$\Upsilon_\Omega T^{\eta\theta} < \theta^\eta \Gamma(\eta + 1), \quad (3.13)$$

then a unique solution for problem (3.2) exists on $[0, T]$. Accordingly, model (2.8) has a unique solution defined on $[0, T]$.

Proof. According to Eqs (3.3) and (3.4), the problem (3.2) can be seen as a fixed point problem. So, the proof will be completed if we show that operator \mathbb{T} has a unique fixed point.

Let $\sup_{t \in [0, T]} \|\Omega(t, 0)\| = G_2 < \infty$. Placing $\mathbb{S}_{\omega_2} = \{\beta \in \mathbb{B} : \|\beta\| \leq \omega_2\}$, where

$$\|\beta_0\| + \frac{G_2 T^{\eta\theta}}{\theta^\eta \Gamma(\eta + 1)} \leq \omega_2 \left(1 - \frac{G_2 T^{\eta\theta}}{\theta^\eta \Gamma(\eta + 1)} \right). \quad (3.14)$$

Hence, \mathbb{S}_{ω_2} is closed, bounded, and convex set in \mathbb{B} . For any $\beta \in \mathbb{S}_{\omega_2}$, we have

$$\begin{aligned} |(\mathbb{T}\beta)(t)| &\leq \|\beta_0\| + \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \frac{\|\Omega(u, \beta(u))\|}{\xi(u, \theta)} du \\ &\leq \|\beta_0\| + \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) (\|\Omega(u, \beta(u)) - \Omega(u, 0)\| + \|\Omega(u, 0)\|) \frac{du}{\xi(u, \theta)} \\ &\leq \|\beta_0\| + \frac{\omega \Upsilon_\Omega + G}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \frac{du}{\xi(u, \theta)} \\ &\leq \|\beta_0\| + \frac{T^{\eta\theta} (\omega \Upsilon_\Omega + G)}{\theta^\eta \Gamma(\eta + 1)} \leq \omega. \end{aligned} \quad (3.15)$$

Therefore, $\mathbb{T}\mathbb{S}_{\omega_2} \subset \mathbb{S}_{\omega_2}$. For any $\beta_1, \beta_2 \in \mathbb{B}$ and all $t \in [0, T]$, we have

$$\begin{aligned} |(\mathbb{T}\beta_1)(t) - (\mathbb{T}\beta_2)(t)| &\leq \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \|\Omega(u, \beta_1(u)) - \Omega(u, \beta_2(u))\| \frac{du}{\xi(u, \theta)} \\ &\leq \frac{\Upsilon_\Omega}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \|\beta_1(t) - \beta_2(t)\| \frac{du}{\xi(u, \theta)} \\ &\leq \frac{\Upsilon_\Omega T^{\eta\theta}}{\theta^\eta \Gamma(\eta + 1)} \|\beta_1 - \beta_2\|. \end{aligned} \quad (3.16)$$

As $\frac{\Upsilon_\Omega T^{\eta\theta}}{\theta^\eta \Gamma(\eta + 1)} < 1$, the operator \mathbb{T} is contraction. Therefore, by the contraction precept of Banach, a fixed point exists for the operator \mathbb{T} . Hence, a unique solution exists on $[0, T]$ for problem (3.3). Accordingly, the model (2.8) has a unique solution on $[0, T]$. This ends the proof. \square

Remark 3.1. (i) If $\xi(u, \theta) = u^{1-\theta}$ then $\Psi(t, u, \theta) = \frac{t^\theta - u^\theta}{\theta}$ and Definitions 2.2 and 2.3 are consistent with the Definitions 2.1 and 2.3 of Jarad et al. [43].

(ii) If $\xi(u, \theta) = u^{1-\theta}$, $\theta \rightarrow 0$ then $\Psi(t, u, \theta) \rightarrow \ln t - \ln u$ and Definitions 2.2 and 2.3 concur with fractional integrals and fractional derivatives of Hadamard type [48].

(iii) If $\xi(u, \theta) = u^{1-\theta}$, $\theta = 1$ then $\Psi(t, u, \theta) = t - u$ and Definitions 2.2 and 2.3 concur with fractional integrals and fractional derivatives of Riemann-Liouville defined in [48].

Corollary 3.1. Using $\xi(u, \theta) = u^{1-\theta}$, $\theta \rightarrow 0$ then $\Psi(t, u, \theta) \rightarrow \ln t - \ln u$ in Eqs (2.1), (2.2) and using all assumption of Theorem 3.1. Then, the problem (3.2), as well as the model (2.8) due to Hadamard fractional integral and derivative operators, has one solution on $[0, T]$ at least.

Corollary 3.2. Using $\xi(u, \theta) = u^{1-\theta}$, $\theta \rightarrow 0$ then $\Psi(t, u, \theta) \rightarrow \ln t - \ln u$ in Eqs (2.1), (2.2) and using all assumption of Theorem 3.2. Then a unique solution for problem (3.2) exists on $[0, T]$. Accordingly, model (2.8) due to Hadamard fractional integral and derivative operators has a unique solution defined on $[0, T]$.

4. Stability results

In this part, we establish some adequate conditions for model (2.8) to meet the presumptions of several varieties of stability. Such as Ulam-Hyers stability, extended Ulam-Hyers stability, Ulam-Hyers-Rassias stability, and extended Ulam-Hyers-Rassias stability. The following definitions are needed before we display the stability theorems.

Let $\varepsilon \in \mathbb{R}_+$ and $\Sigma_\Omega : [0, T] \rightarrow \mathbb{R}_+$ be a continuous functions. The stability definitions will be based on the following inequalities.

$$\|{}_C^{\eta} \mathbb{D}_\xi^\theta g(t) - \Omega(t, g(t))\| \leq \varepsilon, \quad \forall t \in [0, T], \quad (4.1)$$

$$\|{}_C^{\eta} \mathbb{D}_\xi^\theta g(t) - \Omega(t, g(t))\| \leq \varepsilon \Sigma_\Omega(t), \quad \forall t \in [0, T], \quad (4.2)$$

$$\|{}_C^{\eta} \mathbb{D}_\xi^\theta g(t) - \Omega(t, g(t))\| \leq \Sigma_\Omega(t), \quad \forall t \in [0, T], \quad (4.3)$$

4.1. Ulam-Hyers stability and extended Ulam-Hyers stability

Here, we provide some sufficient conditions for model (2.8) to realize the assumptions of Ulam-Hyers stability and extended Ulam-Hyers stability. We begin with defining these kinds of stability.

Definition 4.1 ([42]). The problem (3.2) is said to be stable under Ulam-Hyers condition if for each $g \in \mathbb{B}$ satisfying the inequality (4.1) and for all $\varepsilon > 0$ there exists a solution $\beta \in \mathbb{B}$ for the problem (3.2) such that

$$\|g(t) - \beta(t)\| \leq C_\Omega \varepsilon, \quad t \in [0, T], \quad (4.4)$$

where $C_\Omega = \max(C_{\Omega_k})^{\mathcal{J}}$, $k = 1, 2, 3, 4$.

Definition 4.2 ([42]). The problem (3.2) is said to be stable under the extended Ulam-Hyers condition if there exists $\Sigma_\Omega \in C(\mathbb{R}_+, \mathbb{R}_+)$, with $\Sigma_\Omega(0) = 0$, such that for each $g \in \mathbb{B}$ satisfying the inequality (4.2) there exists a solution $\beta \in \mathbb{B}$ for the problem (3.2) such that

$$\|g(t) - \beta(t)\| \leq \Sigma_\Omega(t), \quad t \in [0, T], \quad (4.5)$$

where $\Sigma_\Omega = \max(\Sigma_{\Omega_k})^{\mathcal{J}}$, $k = 1, 2, 3, 4$.

Now, we display an essential property that can be employed to realize Ulam-Hyers and extended Ulam-Hyers stability.

Lemma 4.1. Assume $\theta \in (0, 1]$ and $\eta > 0$. If $g \in \mathbb{B}$ is a solution of the inequality (4.1), then g satisfies the next inequality

$$\left\| g(t) - g_0 - \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \Omega(u, g(u)) \frac{du}{\xi(u, \theta)} \right\| \leq \frac{\varepsilon T^{\theta\eta}}{\theta^\eta \Gamma(\eta + 1)}. \quad (4.6)$$

Proof. If g is a solution of the inequality (4.1), then there exists a function $h \in \mathbb{B}$ (which not rely on g) such that

$$\|h(t)\| \leq \varepsilon, \quad h = \max(h_k)^{\mathcal{J}}, \quad \forall t \in [0, T]. \quad (4.7)$$

Hence, we have the problem

$$\begin{cases} {}^{\eta}\mathbb{D}_{\xi}^{\theta}g(t) = \Omega(t, g(t)) + h(t), & t \in [0, T], \\ g(0) = g_0 \geq 0. \end{cases} \quad (4.8)$$

According to Theorem 2.2, the solution of the problem (4.8) can be given as

$$g(t) = g_0 + \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \Omega(u, g(u)) \frac{du}{\xi(u, \theta)} + \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) h(u) \frac{du}{\xi(u, \theta)}. \quad (4.9)$$

Therefore, by (4.7), we obtain

$$\begin{aligned} \left\| g(t) - g_0 - \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \Omega(u, g(u)) \frac{du}{\xi(u, \theta)} \right\| &\leq \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \|h(u)\| \frac{du}{\xi(u, \theta)} \\ &\leq \frac{\varepsilon T^{\theta\eta}}{\theta^{\eta}\Gamma(\eta+1)}. \end{aligned} \quad (4.10)$$

Hence, inequality (4.6) is proved. \square

Now, we are ready to prove the Ulam-Hyers stability and extended Ulam-Hyers stability.

Theorem 4.1. *Assume $\Omega(t, \beta(t))$ is continuous for $\beta \in \mathbb{B}$. If (S_3) and (3.13) are fulfilled, then problem (3.2), as well as model (2.8), are stable under Ulam-Hyers and extended Ulam-Hyers conditions.*

Proof. Let $g \in \mathbb{B}$ be a solution of the inequality (4.1). Assume $\varepsilon > 0$ and $\beta \in \mathbb{B}$ is the solution of problem (3.2). According to Eq (3.3) and Lemma 4.1, we have

$$\begin{aligned} \|g(t) - \beta(t)\| &\leq \left\| g(t) - \beta_0 - \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \Omega(u, g(u)) \frac{du}{\xi(u, \theta)} \right\| \\ &\leq \left\| g(t) - g_0 - \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \Omega(u, g(u)) \frac{du}{\xi(u, \theta)} \right\| \\ &\quad + \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \|\Omega(u, g(u)) - \Omega(u, \beta(u))\| \frac{du}{\xi(u, \theta)} \\ &\leq \left\| g(t) - g_0 - \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \Omega(u, g(u)) \frac{du}{\xi(u, \theta)} \right\| \\ &\quad + \frac{\Upsilon_{\Omega}}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \|g(u) - \beta(u)\| \frac{du}{\xi(u, \theta)} \\ &\leq \frac{\varepsilon T^{\theta\eta}}{\theta^{\eta}\Gamma(\eta+1)} + \frac{\Upsilon_{\Omega} T^{\theta\eta}}{\theta^{\eta}\Gamma(\eta+1)} \|g(t) - \eta(t)\|. \end{aligned} \quad (4.11)$$

Hence, $\|g(t) - \beta(t)\| \leq C_{\Omega}\varepsilon$, where

$$C_{\Omega} = \frac{T^{\theta\eta}}{\theta^{\eta}\Gamma(\eta+1)} \frac{1}{1 - \frac{\Upsilon_{\Omega} T^{\theta\eta}}{\theta^{\eta}\Gamma(\eta+1)}}. \quad (4.12)$$

Therefore, model (2.8) is Ulam-Hyers stable. Moreover, by placing $\Sigma_{\Omega}(\varepsilon) = C_{\Omega}\varepsilon$ such that $\Sigma_{\Omega}(0) = 0$ implies that model (2.8) is extended Ulam-Hyers stable. This completes the proof. \square

4.2. Ulam-Hyers-Rassias stability and extended Ulam-Hyers-Rassias stability

We present some sufficient conditions for model (2.8) to realize the Ulam-Hyers-Rassias and extended Ulam-Hyers-Rassias stability assumptions. These types of stability can be recognized as follows.

Definition 4.3 ([42]). The problem (3.2) achieves Ulam-Hyers-Rassias stability with respect to $\Sigma_\Omega \in C([0, T]; \mathbb{R}_+)$ if $\exists L_{\Sigma_\Omega} > 0$ such that for each $g \in \mathbb{B}$ satisfying the inequality (4.2) and for all $\varepsilon > 0$ there exists a solution $\beta \in \mathbb{B}$ for the problem (3.2) with

$$\|g(t) - \beta(t)\| \leq L_{\Sigma_\Omega} \varepsilon \Sigma_\Omega(t), \quad t \in [0, T]. \quad (4.13)$$

Definition 4.4 ([42]). The problem (3.2) attains the extended Ulam-Hyers-Rassias stability with respect to $\Sigma_\Omega \in C([0, T]; \mathbb{R}_+)$ if $\exists L_{\Sigma_\Omega} > 0$ such that for any $g \in \mathbb{B}$ fulfilling the inequality (4.3) there exists a solution $\beta \in \mathbb{B}$ for the problem (3.2) with

$$\|g(t) - \beta(t)\| \leq L_{\Sigma_\Omega} \Sigma_\Omega(t), \quad t \in [0, T]. \quad (4.14)$$

Now, we show an important characteristic that can be utilized to recognize Ulam-Hyers-Rassias and extended Ulam-Hyers-Rassias stability.

Lemma 4.2. Assume the following stipulation:

(S₄) There exists a non-decreasing function $\Sigma_\Omega \in \mathbb{B}$ and there exists $\Theta_{\Sigma_\Omega} > 0$, such that the next inequality holds:

$${}^\eta \mathbb{I}_\xi^\theta \Sigma_\Omega(t) \leq \Theta_{\Sigma_\Omega} \Sigma_\Omega(t) \quad t \in [0, T]. \quad (4.15)$$

If $\theta \in (0, 1]$, $\eta > 0$, and $g \in \mathbb{B}$ is a solution of the inequality (4.2), then g satisfies the next inequality

$$\|g(t) - g_0 - {}^\eta \mathbb{I}_\xi^\theta \Omega(t, g(t))\| \leq \varepsilon \Theta_{\Sigma_\Omega} \Sigma_\Omega(t). \quad (4.16)$$

Proof. If g is a solution of the inequality (4.2), then there exists a function $d \in \mathbb{B}$ (which not rely on g) such that

$$\|d(t)\| \leq \varepsilon \Sigma_\Omega(t), \quad d = \max(d_k)^T, \quad \forall t \in [0, T]. \quad (4.17)$$

So, we obtain the problem

$$\begin{cases} {}^\eta \mathbb{D}_\xi^\theta g(t) = \Omega(t, g(t)) + d(t), & t \in [0, T], \\ g(0) = g_0 \geq 0. \end{cases} \quad (4.18)$$

Hence, the problem (4.18) has the solution

$$g(t) = g_0 + {}^\eta \mathbb{I}_\xi^\theta \Omega(t, g(t)) + {}^\eta \mathbb{I}_\xi^\theta d(t). \quad (4.19)$$

By applying (4.17), we have

$$\|g(t) - g_0 - {}^\eta \mathbb{I}_\xi^\theta \Omega(t, g(t))\| \leq {}^\eta \mathbb{I}_\xi^\theta \|d(t)\| \leq \varepsilon {}^\eta \mathbb{I}_\xi^\theta \Sigma_\Omega(t) \leq \varepsilon \Theta_{\Sigma_\Omega} \Sigma_\Omega(t). \quad (4.20)$$

Thus, inequality (4.16) is attained. \square

Now, for model (2.8), Ulam-Hyers-Rassias stability and extended Ulam-Hyers-Rassias stability can be proved as follows.

Theorem 4.2. *Assume $\Omega(t, \beta(t))$ is continuous for $\beta \in \mathbb{B}$. If (S_3) , (S_4) and (3.13) are satisfied, then problem (3.2), as well as model (2.8), are stable under Ulam-Hyers-Rassias and extended Ulam-Hyers-Rassias conditions.*

Proof. Let $g \in \mathbb{B}$ be a solution of the inequality (4.3). Assume $\varepsilon > 0$ and $\beta \in \mathbb{B}$ is the solution of problem (3.2). According to Eq (3.3) and Lemma 4.2, we have

$$\begin{aligned} \|g(t) - \beta(t)\| &\leq \|g(t) - \beta_0 - {}^{n-\eta} \mathbb{I}_{\xi}^{\theta} \Omega(t, \beta(t))\| \\ &\leq \|g(t) - g_0 - {}^{n-\eta} \mathbb{I}_{\xi}^{\theta} \Omega(t, g(t))\| + \frac{1}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \|\Omega(u, g(u)) - \Omega(u, \beta(u))\| \frac{du}{\xi(u, \theta)} \\ &\leq \|g(t) - g_0 - {}^{n-\eta} \mathbb{I}_{\xi}^{\theta} \Omega(t, g(t))\| + \frac{\Upsilon_{\Omega}}{\Gamma(\eta)} \int_0^t \Psi^{\eta-1}(t, u, \theta) \|g(s) - \beta(s)\| \frac{du}{\xi(u, \theta)} \\ &\leq \varepsilon \Theta_{\Sigma_{\Omega}} \Sigma_{\Omega}(t) + \frac{\Upsilon_{\Omega} T^{\theta \eta}}{\theta^{\eta} \Gamma(\eta + 1)} \|g(t) - \beta(t)\|, \end{aligned} \quad (4.21)$$

Therefore,

$$\|g(t) - \beta(t)\| \leq L_{\Sigma_{\Omega}} \varepsilon \Sigma_{\Omega}(t), \quad (4.22)$$

where

$$L_{\Sigma_{\Omega}} = \frac{\Theta_{\Sigma_{\Omega}}}{1 - \frac{\Upsilon_{\Omega} T^{\theta \eta}}{\theta^{\eta} \Gamma(\eta + 1)}}. \quad (4.23)$$

Hence, model (2.8) is stable under Ulam-Hyers-Rassias condition. Moreover, when $\varepsilon = 1$ in (4.22) with $\Sigma_{\Omega}(0) = 0$, then model (2.8) is stable under the extended Ulam-Hyers-Rassias condition. This completes the proof. \square

5. Conclusions

The present work discussed the possibility of constructing a new fractional mathematical model for HIV infection. This fractional model has been built via recent improved fractional differential and integral operators. For this fractional HIV model, the existence and uniqueness results have been examined through Banach's and Leray-Schauder nonlinear alternative fixed point theorems. Also, diverse types of Ulam stability for the proposed fractional HIV model are analyzed. By comparing the results extracted in the present work with the results obtained in the previous literature, one can see that if $\xi(t, \theta) = t^{1-\theta}$, then $\Psi(t, u, \theta) = \frac{t^{\theta}-u^{\theta}}{\theta}$ and Definitions 2.2 and 2.3 concur with the Definitions 2.1 and 2.3 of Jarad et al. [43], respectively. In this case, our results obtained in Theorems 3.1, 3.2, 4.1 and 4.2 coincide with the results acquired by Kongson et al. [37] for the HIV infection. Moreover, if $\theta \rightarrow 1$, the results in Theorems 3.1, 3.2 and 4.1 tend to the traditional results for the HIV infection that obtained by the classical Newton's derivative. Moreover, the existence, uniqueness, and stability results presented in this work can serve the study of the optimal control problem of the HIV model with a fractional objective functional [44–46].

Acknowledgements

The authors extend their appreciation to the Deanship of Scientific Research at King Khalid University for funding this work through Research Groups Program under grant RGP.2/15/43.

Conflict of interest

The authors declare that they have no conflicts of interest.

References

1. W. H. Organization, HIV, 2022. Available from: <https://www.who.int/news-room/fact-sheets/detail/hiv-aids>.
2. R. A. Weiss, How does HIV cause AIDS, *Science*, **260** (1993), 1273–1279. <https://doi.org/10.1126/science.8493571>
3. X. Zhang, L. Liu, W. Chen, F. Wang, Y. Cheng, Y. Liu, et al., Gestational Leucylation suppresses embryonic T-box transcription factor 5 signal and causes congenital heart disease, *Adv. Sci.*, **9** (2022), 2201034. <https://doi.org/10.1002/advs.202201034>
4. X. Zhang, Y. Qu, L. Liu, Y. Qiao, H. Geng, Y. Lin, et al., Homocysteine inhibits pro-insulin receptor cleavage and causes insulin resistance via protein cysteine-homocysteinylation, *Cell Rep.*, **37** (2021), 109821. <https://doi.org/10.1016/j.celrep.2021.109821>
5. K. Cai, F. Wang, J. Lu, A. Shen, S. Zhao, W. Zang, et al., Nicotinamide mononucleotide alleviates cardiomyopathy phenotypes caused by short-chain enoyl-CoA hydratase 1 deficiency, *JACC: Basic Trans. Sci.*, **7** (2022), 348–362. <https://doi.org/10.1016/j.jacbts.2021.12.007>
6. F. Kirchhoff, *Encyclopedia of AIDS*, Springer, 2013. <https://doi.org/10.1007/978-1-4614-9610-6-60-1>
7. M. A. Nowak, S. Bonhoeffer, G. M. Shaw, R. M. May, Anti-viral drug treatment: Dynamics of resistance in free virus and infected cell populations, *J. Theor. Biol.*, **184** (1997), 203–217. <https://doi.org/10.1006/jtbi.1996.0307>
8. T. B. Kepler, A. S. Perelson, Drug concentration heterogeneity facilitates the evolution of drug resistance, *Proc. Natl. Acad. Sci. USA.*, **95** (1998), 11514–11519. <https://doi.org/10.1073/pnas.95.20.11514>
9. R. J. Smith, L. M. Wahl, Distinct effects of protease and reverse transcriptase inhibition in an immunological model of HIV-1 infection with impulsive drug effects, *Bull. Math. Biol.*, **66** (2004), 1259–1283. <https://doi.org/10.1016/j.bulm.2003.12.004>
10. Z. Cao, Y. Wang, W. Zheng, L. Yin, Y. Tang, W. Miao, et al., The algorithm of stereo vision and shape from shading based on endoscope imaging, *Biomed. Signal Process. Control*, **76** (2022), 103658. <https://doi.org/10.1016/j.bspc.2022.103658>
11. F. Brauer, C. Castillo-Chavez, *Mathematical models in population biology and epidemiology*, Springer, 2001. <https://doi.org/10.1007/978-1-4614-1686-9>

12. C. Duan, H. Deng, S. Xiao, J. Xie, H. Li, X. Zhao, et al., Accelerate gas diffusion-weighted MRI for lung morphometry with deep learning, *Eur. Radiol.*, **32** (2022), 702–713. <https://doi.org/10.1007/s00330-021-08126-y>
13. A. S. Perelson, D. E. Kirschner, R. De Boer, Dynamics of HIV infection of CD4+ T cells, *Math. Biosci.*, **114** (1993), 81–125. [https://doi.org/10.1016/0025-5564\(93\)90043-a](https://doi.org/10.1016/0025-5564(93)90043-a)
14. H. C. Tuckwell, F. Y. M. Wan, On the behavior of solutions in viral dynamical models, *Biosystems*, **73** (2004), 157–161. <https://doi.org/10.1016/j.biosystems.2003.11.004>
15. L. Rong, M. A. Gilchrist, Z. Feng, A. S. Perelson, Modeling within-host HIV-1 dynamics and the evolution of drug resistance: Trade-offs between viral enzyme function and drug susceptibility, *J. Theor. Biol.*, **247** (2007), 804–818. <https://doi.org/10.1016/j.jtbi.2007.04.014>
16. P. K. Srivastava, M. Banerjee, P. Chandra, Modeling the drug therapy for HIV infection, *J. Biol. Syst.*, **17** (2009), 213–223. <https://doi.org/10.1142/S0218339009002764>
17. L. Liu, J. Wang, L. Zhang, S. Zhang, Multi-AUV dynamic maneuver countermeasure algorithm based on interval information game and fractional-order DE, *Fractal Fract.*, **6** (2022), 235. <https://doi.org/10.3390/fractalfract6050235>
18. C. Dineshkumar, R. Udhayakumar, V. Vijayakumar, K. S. Nisar, A. Shukla, A note concerning to approximate controllability of Atangana-Baleanu fractional neutral stochastic systems with infinite delay, *Chaos Solitons Fractals*, **157** (2022), 111916. <https://doi.org/10.1016/j.chaos.2022.111916>
19. A. Shukla, V. Vijayakumar, K. S. Nisar, A new exploration on the existence and approximate controllability for fractional semilinear impulsive control systems of order $r \in (1, 2)$, *Chaos Solitons Fractals*, **154** (2022), 111615. <https://doi.org/10.1016/j.chaos.2021.111615>
20. S. Kumar, A. Kumar, B. Samet, H. Dutta, A study on fractional host–parasitoid population dynamical model to describe insect species, *Numer. Methods Part. Differ. Equ.*, **37** (2021), 1673–1692. <https://doi.org/10.1002/num.22603>
21. S. Kumar, R. P. Chauhan, S. Momani, S. Hadid, Numerical investigations on COVID-19 model through singular and non-singular fractional operators, *Numer. Methods Part. Differ. Equ.*, **37** (2020), 1–27. <https://doi.org/10.1002/num.2270>
22. A. Atangana, D. Baleanu, New fractional derivatives with non-local and non-singular kernel theory and application to heat transfer model, *Therm. Sci.*, **20** (2016), 763–769. <https://doi.org/10.2298/TSCI160111018A>
23. J. A. T. Machado, M. E. A. Mata, A fractional perspective to the bond graph modelling of world economies, *Nonlinear Dyn.*, **80** (2015), 1839–1852. <https://doi.org/10.1007/s11071-014-1334-0>
24. S. Zeng, J. Cen, A. Atangana, V. T. Nguyen, Qualitative analysis of solutions of obstacle elliptic inclusion problem with fractional Laplacian, *Z. Angew. Math. Phys.*, **72** (2021), 30. <https://doi.org/10.1007/s00033-020-01460-z>
25. Y. Penga, J. Zhaoa, K. Sepehrnoori, Z. Li, Fractional model for simulating the viscoelastic behavior of artificial fracture in shale gas, *Eng. Fract. Mech.*, **228** (2020), 106892. <https://doi.org/10.1016/j.engfracmech.2020.106892>

26. A. Hyder, M. A. Barakat, A. Fathallah, Enlarged integral inequalities through recent fractional generalized operators, *J. Inequal. Appl.*, **2022** (2022), 95. <https://doi.org/10.1186/s13660-022-02831-y>
27. S. Kumar, A. Kumar, M. Jleli, A numerical analysis for fractional model of the spread of pests in tea plants, *Numer. Methods Part. Differ. Equ.*, **38** (2022), 540–565. <https://doi.org/10.1002/num.22663>
28. S. Kumar, R. P. Chauhan, S. Momani, S. Hadid, A study of fractional TB model due to mycobacterium tuberculosis bacteria, *Chaos Solitons Fractals*, **153** (2021), 111452. <https://doi.org/10.1016/j.chaos.2021.111452>
29. M. A. Barakat, A. H. Soliman, A. Hyder, Langevin equations with generalized proportional Hadamard–Caputo fractional derivative, *Comput. Intell. Neurosci.*, **2021** (2021), 6316477. <https://doi.org/10.1155/2021/6316477>
30. M. A. Khan, S. Ullah, S. Kumar, A robust study on 2019-nCoV outbreaks through non-singular derivative, *Eur. Phys. J. Plus*, **136** (2021), 168. <https://doi.org/10.1140/epjp/s13360-021-01159-8>
31. A. Hyder, A. A. Almoneef, H. Budak, M. A. Barakat, On new fractional version of generalized Hermite-Hadamard inequalities, *Mathematics*, **10** (2022), 3337. <https://doi.org/10.3390/math10183337>
32. S. Kumar, A. Kumar, B. Samet, J. F. Gómez-Aguilar, M. S. Osman, A chaos study of tumor and effector cells in fractional tumor-immune model for cancer treatment, *Chaos Solitons Fractals*, **141** (2020), 110321. <https://doi.org/10.1016/j.chaos.2020.110321>
33. B. H. Lichae, J. Biazar, Z. Ayati, The fractional differential model of HIV-1 infection of CD4⁺ T-Cells with description of the effect of antiviral drug treatment, *Comput. Math. Methods Med.*, **2019** (2019), 4059549. <https://doi.org/10.1155/2019/4059549>
34. A. J. Ferrari, E. A. Santillan Marcus, Study of a fractional-order model for HIV infection of CD4⁺ T-Cells with treatment, *J. Fractional Calculus Appl.*, **11** (2020), 12–22.
35. G. Nazir, K. Shah, A. Debbouche, R. A. Khan, Study of HIV mathematical model under nonsingular kernel type derivative of fractional order, *Chaos Solitons Fractals*, **139** (2020), 110095. <https://doi.org/10.1016/j.chaos.2020.110095>
36. H. Khan, J. F. Gómez-Aguilar, A. Alkhazzan, A. Khan, A fractional order HIV-TB coinfection model with nonsingular Mittag-Leffler law, *Math. Methods Appl. Sci.*, **43** (2020), 3786–3806. <https://doi.org/10.1002/mma.6155>
37. J. Kongson, C. Thaiprayoon, W. Sudsutad, Analysis of a fractional model for HIV CD4⁺ T-cells with treatment under generalized Caputo fractional derivative, *AIMS Math.*, **6** (2021), 7285–7304. <https://doi.org/10.3934/math.2021427>
38. M. Areshi, A. Khan, R. Shah, K. Nonlaopon, Analytical investigation of fractional-order Newell-Whitehead-Segel equations via a novel transform, *AIMS Math.*, **7** (2022), 6936–6958. <https://doi.org/10.3934/math.2022385>
39. K. Nonlaopon, M. Naeem, A. M. Zidan, R. Shah, A. Alsanad, A. Gumaei, Numerical investigation of the time-fractional Whitham-Broer-Kaup equation involving without singular kernel operators, *Complexity*, **2021** (2021), 1–21. <https://doi.org/10.1155/2021/7979365>

40. N. A. Shah, H. A. Alyousef, S. El-Tantawy, R. Shah, J. D. Chung, Analytical investigation of fractional-order Korteweg-de-Vries-type equations under Atangana-Baleanu-Caputo operator: Modeling nonlinear waves in a plasma and fluid, *Symmetry*, **14** (2022), 739. <https://doi.org/10.3390/sym14040739>
41. A. Hyder, M. A. Barakat, Novel improved fractional operators and their scientific applications, *Adv. Differ. Equ.*, **2021** (2021), 389. <https://doi.org/10.1186/s13662-021-03547-x>
42. I. A. Rus, Ulam stabilities of ordinary differential equations in a Banach space, *Carpathian J. Math.*, **26** (2010), 103–107.
43. F. Jarad, E. Uğurlu, T. Abdeljawad, D. Baleanu, On a new class of fractional operators, *Adv. Differ. Equ.*, **2017** (2017), 247. <https://doi.org/10.1186/s13662-017-1306-z>
44. A. Shukla, N. Sukavanam, D. N. Pandey, Controllability of semilinear stochastic control system with finite delay, *IMA J. Math. Control Inf.*, **35** (2018), 427–449. <https://doi.org/10.1093/imamci/dnw059>
45. C. Dineshkumar, R. Udhayakumar, V. Vijayakumar, A. Shukla, K. S. Nisar, A note on approximate controllability for nonlocal fractional evolution stochastic integrodifferential inclusions of order $r \in (1, 2)$ with delay, *Chaos Solitons Fractals*, **153** (2021), 111565. <https://doi.org/10.1016/j.chaos.2021.111565>
46. A. Shukla, N. Sukavanam, D. N. Pandey, Approximate controllability of semilinear stochastic control system with nonlocal conditions, *Nonlinear Dyn. Syst. Theory*, **15** (2015), 321–333.
47. U. N. Katugampola, New approach to a generalized fractional integral, *Appl. Math. Comput.*, **218** (2011), 860–865. <https://doi.org/10.1016/j.amc.2011.03.062>
48. A. A. Kilbas, Hadamard type fractional calculus, *J. Korean Math. Soc.*, **38** (2001), 1191–1204.



AIMS Press

©2023 the Author(s), licensee AIMS Press. This is an open access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>)