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Analysis of the HIV/AIDS transmission dynamics model using Caputo fractional-order derivative

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Abstract. Although national and international institutions, such as the World Health Organization (WHO) and UNAIDS, are making significant efforts to eradicate HIV by 2030, it remains a major threat to global public health. Despite its low prevalence, HIV continues to claim lives and remains a major public health issue, especially in developing countries. Thanks to the accessibility of antiretroviral drugs, the prevalence of this scourge has been gradually declining worldwide in recent years. Thus, this article investigates the effectiveness of antiretroviral therapy in controlling viral transmission through a fractional-order extension of a deterministic model. We study the boundedness of the model's solution by applying the Laplace transform to solve the fractional Gronwall inequality. To ensure the existence and uniqueness of the model's solution, we rely on the Picard-Lindelöf theorem. We also study the stability of the disease-free equilibrium point to qualitatively analyze the behavior of the model. Next, we perform a sensitivity analysis of the basic reproduction number $\mathcal{R}_0$ to evaluate its robustness concerning the model parameters. Finally, we simulate the approximate solutions of the fractional-order model in MATLAB for different values of the fractional order and present the results of the sensitivity analysis and numerical simulation. Our results demonstrate that the fractional model provides real added value in modeling, thanks to its ability to incorporate memory effects and finely tune transmission dynamics according to the fractional order, thereby allowing for a more realistic representation of epidemiological processes.

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1. Introduction

In the 1980s, acquired immunodeficiency syndrome (AIDS) first emerged in the United States [41]. It was in 1983 that the human immunodeficiency virus (HIV), the retrovirus responsible for this infection and the cause of AIDS, was identified at the Institut Pasteur in Paris [41]. This retrovirus weakens the immune system by affecting most cells, with a particularly destructive impact on $CD4^+$ T cells [8]. The human body becomes vulnerable to any infection when the number of $CD4^+$ T cells falls below a certain threshold, causing the cell-mediated immune system to collapse and the overall immune system to weaken [43]. AIDS is now considered a pandemic that has claimed approximately 42.3 million lives (35.7–51.1 million) between 1981 and 2023 [40]. To date, there is no cure or vaccine for AIDS, but antiretroviral therapy (ART) improves health, prolongs life, and significantly reduces the risk of HIV transmission [46]. Advances in treatment and prevention have led some to question whether the "end of AIDS" is now within reach [23]. Studies have shown, on the one hand, that the risk of HIV transmission among serodifferent heterosexual couples is reduced by 96% when the HIV-positive partner is undergoing treatment [17], and on the other hand, that individuals living with HIV who are on antiretroviral therapy have a very low probability (estimated at less than 0.04) of transmitting the virus [18]. For more information on the biology and epidemiology of HIV/AIDS, readers are directed to references [2, 7, 41], and the associated literature cited within.

Several mathematical models have proven effective in describing and analyzing the dynamics of HIV infection; see, for example, [3, 8, 9, 10, 11, 13, 25, 26, 33, 38, 45, 46, 47, 54] and related studies.

Fractional-order integral-differential operators generalize integration and differentiation to non-integer orders [21]. In recent decades, fractional-order models have consistently demonstrated superior accuracy compared to classical integer-order models in describing certain aspects of natural phenomena [21]. Regarding the transmission dynamics of infectious diseases using fractional-order mathematical modeling, several valuable studies have been conducted. Among these, we can mention [6, 8, 19, 22, 29, 30, 36, 42, 52], and the references cited therein. The authors of [8] extend the HIV-1 infection model of $CD4^+$ T cells to the concept of the Caputo-Fabrizio fractional derivative and present numerical results for different values of the fractional order to verify the efficiency and accuracy of the new fractional model. In [6], the authors construct their model by integrating the population of mildly infected individuals into the compartmental SIR model, transforming it into an SMIR model in the form of a system of fractional-order differential equations in the sense of Caputo. They performed numerical simulations and found that population dynamics varied with different values of the fractional order. They also observed that an increase in the infection rate of mild cases leads to a corresponding increase in the total population of infected individuals. Fractional-order derivative and integral operators depend not only on the current state of the system but also on all its past states due to the non-local nature of these operators. This property makes them particularly useful and powerful for evaluating the future evolution of the system. As a result, they prove to be more effective than classical deterministic operators, as they incorporate

memory effects, providing more accurate and realistic modeling of complex dynamic phenomena [30].
We here consider the deterministic SIAHR model describing the transmission dynamics of HIV/AIDS, including a compartment for individuals in remission, as studied in [12], to which we will generalize to a fractional-order model. Our objective is to extend the deterministic model by incorporating a fractional-order time derivative in the sense of Caputo. This allows us to demonstrate the power and effectiveness of fractional-order differentiation and integration operators in evaluating the future state of the model, compared to other classical deterministic operators.

The rest of the work is organized as follows: The deterministic model is provided in Section 2. In Section 3, we formulate and prove the existence and uniqueness of the solutions to the fractional-order model in the sense of Caputo relative to the deterministic model. In Section 4, we analyze the fractional-order model in the sense of Caputo. In Section 5, we address the sensitivity analysis of the basic reproduction number $\mathcal{R}_0$. Section 6 is dedicated to the study of numerical results and discussions of the fractional-order model. Finally, the conclusions are presented in Section 7.

2. The deterministic model

We consider the SIAHR model describing the transmission dynamics of HIV/AIDS, including a compartment for individuals in remission, as studied in [12]. The model segments the human population into five distinct and mutually exclusive compartments: the population of susceptible individuals S, the population of infected individuals showing no clinical symptoms of the disease I, the group of individuals with the AIDS disease A, the population of individuals undergoing antiretroviral treatment H, and the group of individuals in remission R.
The distribution of individuals across the different compartments occurs as follows: in S, a susceptible person contracts the infection after coming into contact with an infected individual. An individual in compartment I can start antiretroviral treatment and join class H, or they may ignore the treatment and naturally progress to compartment A. In class A, an individual undergoing treatment can also move to class H. An individual in class H can move to compartment R if they adhere to the treatment properly, or transition to compartment A if they fail to adhere to it.
As in [12], susceptible individuals acquire infection through contact with infected individuals at a rate K defined as

$$K = \frac{\alpha(I + \eta A)}{N}, \tag{2.1}$$

where α is the effective contact rate for HIV transmission, η is a modification factor ($\eta \geq 1$) accounting for the difference in infectiousness between individuals with AIDS symptoms and asymptomatic HIV-infected individuals. This model incorporates the fact that individuals with AIDS symptoms are more infectious than asymptomatic carriers due to their higher viral load. There are other studies in the scientific literature [46, 51] that have indeed demonstrated a positive correlation between viral load and HIV transmission potential.

The model studied in [12] is presented by the following system of ordinary differential equations:

$$\begin{cases} \dfrac{dS(t)}{dt} = \Lambda - \Lambda p I(t) - (K(t) + \mu_0)S(t) \\ \dfrac{dI(t)}{dt} = K(t)S(t) - k_1 I(t) \\ \dfrac{dA(t)}{dt} = \epsilon\beta_1 I(t) - k_2 A(t) + \rho\lambda H(t) \\ \dfrac{dH(t)}{dt} = (1-\epsilon)\beta_1 I(t) + \beta_2 A(t) - k_3 H(t) \\ \dfrac{dR(t)}{dt} = (1-\rho)\lambda H(t) - \mu_0 R(t), \end{cases} \tag{2.2}$$

where $k_1 = -\Lambda p + \beta_1 + \mu_0$, $k_2 = \beta_2 + \mu_0 + \mu_1$ and $k_3 = \lambda + \mu_0$,
with the following initial conditions:
$S(0) \geq 0, \quad I(0) \geq 0, \quad A(0) \geq 0, \quad H(0) \geq 0 \quad$ and $\quad R(0) \geq 0.$
In [12], it was also shown that the model (2.2) is mathematically and epidemiologically well-posed, meaning that the proofs of existence and uniqueness of the solutions to the model (2.2) are studied there.

3. Formulation of the fractional-order model

Drawing inspiration from the results of the deterministic HIV/AIDS transmission model presented in reference [12], and motivated by the importance of the new concepts of fractional-order derivatives, we extend the model (2.2) to a fractional-order version. Applied problems require a definition of fractional-order derivatives that allows for the use of physically interpretable initial conditions, and it is the definition of the fractional-order derivative in the sense of Caputo that satisfies this requirement [48]. Thus, we will generalize or extend the model (2.2) to a fractional-order model in the sense of Caputo, accounting for memory effects and capturing long-term dynamics such as relapses in infections or delayed responses to treatment in epidemiology. In particular, interruptions or irregularities in the administration of antiretrovirals have a cumulative impact on viral load and immune responses.

3.1. Essential preliminaries

This section reviews the principal tools of generalized fractional operators, building on earlier advances. These fundamental mathematical notions underpin the analysis developed in this study [16, 20, 22, 24, 31, 32, 34, 42, 48].

Definition 3.1. *Let $x \in \mathbb{R}_*^+$, the Gamma function defined by:*

$$\Gamma(x) = \int_0^{+\infty} t^{x-1} e^{-t} dt. \tag{3.1}$$

(This integral is convergent for all $x > 0$).

Proposition 3.2. *For all $x > 0$ and for all $n \in \mathbb{N}$, we have:*
$\Gamma(x+1) = x\Gamma(x); \qquad \Gamma(n) = (n-1)!, \qquad \Gamma(n+1) = n!$
In particular $\Gamma(1) = \int_0^{+\infty} e^{-t} dt = 1; \qquad \Gamma\left(\dfrac{1}{2}\right) = \int_0^{+\infty} t^{-\frac{1}{2}} e^{-t} dt = \sqrt{\pi}.$

Definition 3.3. *Let $\sigma \in \mathbb{R}_*^+$, $a \in \mathbb{R}$,$n \in \mathbb{N}$ such that $n-1 < \sigma < n$ and let f be a locally integrable function defined on $[a; +\infty[$. The Caputo fractional derivative of order σ of $f(x)$ with lower limit a is defined by:*

$$ {}_a^c D_t^\sigma f(x) = \frac{1}{\Gamma(n-\sigma)} \int_a^x (x-t)^{n-\sigma-1} f^{(n)}(t) dt, \tag{3.2}$$

where $f^{(n)}$ denotes the $n-th$ order derivative of f.

Definition 3.4. *The fractional integral operator of order $\sigma > 0$ of the function $f(t)$, $t > 0$ is defined by:*

$$ {}_0 I_t^\sigma f(t) = \frac{1}{\Gamma(\sigma)} \int_0^t (t-s)^{\alpha-1} f(s) ds, \qquad t \geq 0 \tag{3.3}$$

Definition 3.5. *The function $F(s)$ of the complex variable s is defined by:*

$$ F(s) = \mathcal{L}\{f(s)\} = \int_0^\infty e^{-st} f(t) dt $$

called the Laplace transform of the function f.

Lemma 3.6. *Note that the Laplace transform of a derivative operator of order n is obtained as follows:*

$$ \mathcal{L}\{f^{(n)}(s)\} = S^n \mathcal{L}\{f(t)\} - \sum_{k=0}^{n-1} S^{n-k-1} f^{(k)}(t_0). \tag{3.4}$$

Similarly, for $\sigma \in [n-1; n]$, we obtain the Laplace transform of the Caputo fractional operator as:

$$ \mathcal{L}\{{}_a^c D_t^\sigma f(t)\} = S^\sigma \mathcal{L}\{f(t)\} - \sum_{k=0}^{n-1} S^{\sigma-k-1} f^{(k)}(t_0). \tag{3.5}$$

Definition 3.7. *The exponential function, e^z, plays a compelling role in the theory of integer-order differential equations. The generalization of the exponential function to a single parameter was introduced by G.M. Mittag-Leffler and is referred to as the function*

$$ E_\sigma(z) = \sum_{k=0}^{+\infty} \frac{z^k}{\Gamma(\sigma k + 1)}. \tag{3.6}$$

The Mittag-Leffler function with two parameters also plays a crucial role in the theory of fractional calculus. This two-parameter Mittag-Leffler function was introduced by Agarwal and is defined by a series expansion as follows:

$$ E_{\sigma,\beta}(z) = \sum_{k=0}^{+\infty} \frac{z^k}{\Gamma(\sigma k + \beta)}, \sigma, \beta > 0. \tag{3.7}$$

Remark 3.8. For $\beta = 1$, we have $E_{\sigma,1}(z) = E_\sigma(z)$ and for $\sigma = \beta = 1$, we have

$$ E_{1,1}(z) = \sum_{k=0}^{+\infty} \frac{z^k}{\Gamma(k+1)} = \sum_{k=0}^{+\infty} \frac{z^k}{k!} = e^z. \tag{3.8}$$

Theorem 3.9. *(Banach Contraction Theorem) Let X be a Banach space and $F : X \longrightarrow X$, a contraction, that is, there exists $0 < k < 1$ such that for all x, $y \in X$, we have*

$$||F(x) - F(y)||_X \leq k||x - y||_X.$$

Then F has a unique fixed point $x^* \in X$, i.e., $F(x^*) = x^*$.

Definition 3.10. *(Banach's Fixed Point Theorem) Let S be a closed subset of a Banach space X and let T be a mapping that assigns S to S. Suppose that:*

$$||T(x) - T(y)||_S \leq p||x - y||_S, \text{for all } x, y \in S, 0 \leq p < 1,$$

then:
a) There exists a unique vector $x^* \in S$ satisfying $x^* = T(x^*)$;
b) x^* can be obtained by the method of successive approximations, starting from any arbitrary initial vector in S.

Theorem 3.11. [6] *The equilibrium solutions x^* of the system*

$$\begin{cases} {}_a^c D_t^\sigma x(t) = f(t, x) \\ x(t_0) = x_0 \end{cases} \tag{3.9}$$

are locally and asymptotically stable if all the eigenvalues λ_i of the Jacobian $\dfrac{\partial f}{\partial x_i}$ evaluated at the equilibrium point satisfy

$$|arg(\lambda_i)| > \frac{\sigma\pi}{2},$$

for all $\sigma \in (0, 1)$.

3.2. Formulation of the Caputo fractional-order model

A fractional-order mathematical model of HIV/AIDS transmission is developed in this section. We thus modify the system (2.2) to obtain a fractional-order system in the sense of Caputo. By generalizing the deterministic model to the fractional-order one, we use the fractional-order time derivative and integral operators in the sense of Caputo, as defined in (3.2). The fractional-order mathematical model in the sense of Caputo, obtained from the deterministic model given in (2.2), is described as follows:

$$\begin{cases} {}_0^c D_t^\sigma S(t) = \Lambda - \Lambda p I(t) - (K + \mu_0) S(t) \\ {}_0^c D_t^\sigma I(t) = K S(t) - k_1 I(t) \\ {}_0^c D_t^\sigma A(t) = \epsilon\beta_1 I(t) - k_2 A(t) + \rho\lambda H(t) \\ {}_0^c D_t^\sigma H(t) = (1 - \epsilon)\beta_1 I(t) + \beta_2 A(t) - k_3 H(t) \\ {}_0^c D_t^\sigma R(t) = (1 - \rho)\lambda H(t) - \mu_0 R(t), \end{cases} \tag{3.10}$$

with the following initial conditions:

$$\begin{aligned} S(0) = S_0 \geq 0; I(0) = I_0 \geq 0; A(0) = A_0 \geq 0; \\ H(0) = H_0 \geq 0 \quad \text{and} \quad R(0) = R_0 \geq 0, \end{aligned} \tag{3.11}$$

where ${}_0^cD_t^\sigma$ is the Caputo fractional derivative operator of order σ.
Then, by applying the fractional integral operator of order σ as given in (3.3) to the system (3.10) with the initial conditions (3.11), we obtain:

$$\begin{cases} S(t) = S_0 + \dfrac{1}{\Gamma(\sigma)} \int_0^t (t-\tau)^{\sigma-1}(\Lambda - \Lambda p I(\tau) - (K+\mu_0)S(\tau))d\tau \\ I(t) = I_0 + \dfrac{1}{\Gamma(\sigma)} \int_0^t (t-\tau)^{\sigma-1}(KS(\tau) - k_1 I(\tau))d\tau \\ A(t) = A_0 + \dfrac{1}{\Gamma(\sigma)} \int_0^t (t-\tau)^{\sigma-1}(\epsilon\beta_1 I(\tau) - k_2 A(\tau) + \rho\lambda H(\tau))d\tau \\ H(t) = H_0 + \dfrac{1}{\Gamma(\sigma)} \int_0^t (t-\tau)^{\sigma-1}(1 - (\epsilon)\beta_1 I(\tau) + \beta_2 A(\tau) - k_3 H(\tau))d\tau \\ R(t) = R_0 + \dfrac{1}{\Gamma(\sigma)} \int_0^t (t-\tau)^{\sigma-1}((1-\rho)\lambda H(\tau) - \mu_0 R(\tau))d\tau, \end{cases} \tag{3.12}$$

where $\sigma \in (0,1)$ and $t \geq 0$. We note that as $\sigma \to 1$, the fractional-order model (3.10) converges to the classical deterministic model (2.2).

3.3. Boundedness of solutions

The size of each population class in the model (3.10) varies over time, and the total population size $N(t)$ is given by:

$$N(t) = S(t) + I(t) + A(t) + H(t) + R(t).$$

Summing the equations of the model (3.10), we obtain the equation for the total population by linearizing the Caputo operator:

$${}_0^cD_t^\sigma N(t) = {}_0^c D_t^\sigma S(t) + {}_0^c D_t^\sigma I(t) + {}_0^c D_t^\sigma A(t) + {}_0^c D_t^\sigma H(t) + {}_0^c D_t^\sigma R(t),$$

which gives us, after simplification:

$${}_0^cD_t^\sigma N(t) = \Lambda - \mu_0 N(t) - \mu_1 A(t).$$

Applying an appropriate upper bound, we obtain the inequality:

$${}_0^cD_t^\sigma N(t) + \mu_0 N(t) \leq \Lambda. \tag{3.13}$$

Applying the Laplace transform method, we solve Grönwall's inequality (3.13) subject to the initial condition $N(0) \geq 0$:

$$\mathcal{L}\{{}_0^cD_t^\sigma N(t)\} + \mu_0 \mathcal{L}\{N\} \leq \mathcal{L}\{\Lambda\}.$$

It follows that:

$$\mathcal{L}\{N(t)\} \leq \frac{\Lambda}{S(S^\sigma + \mu_0)} + \sum_{k=0}^{n-1} \frac{S^{\sigma-k-1}N^{(k)}(0)}{S^\sigma + \mu_0}. \tag{3.14}$$

Splitting (3.14) to partial fractions gives:

$$\mathcal{L}\{N(t)\} \leq \frac{\Lambda}{\mu_0}\left(\frac{1}{S} - \frac{1}{S\left(1 + \dfrac{\mu_0}{S^\sigma}\right)}\right) + \sum_{k=0}^{n-1} \frac{N^{(k)}(0)}{S^{k+1}(1 + \dfrac{\mu_0}{S^\sigma})}. \tag{3.15}$$

Using the Taylor series expansion, we obtain:

$$\frac{1}{1+\frac{\mu_0}{S^\sigma}} = \sum_{n=0}^{\infty}\Big(\frac{-\mu_0}{S^\sigma}\Big)^n,$$

further:

$$\mathcal{L}\{N(t)\} \leq \frac{\Lambda}{\mu_0}\Big(\frac{1}{S} - \sum_{n=0}^{\infty}\Big(\frac{(-\mu_0)^n}{S^{n\sigma+1}}\Big)\Big) + \sum_{k=0}^{n-1}\sum_{n=0}^{\infty}\Big(\frac{(-\mu_0)^n N^{(k)}(0)}{S^{n\sigma+k+1}}\Big). \tag{3.16}$$

Applying the inverse Laplace transform to (3.16), we obtain:

$$\begin{aligned} N(t) \leq \frac{\Lambda}{\mu_0}\mathcal{L}^{-1}\Big(\frac{1}{S}\Big) - \frac{\Lambda}{\mu_0}\sum_{n=0}^{\infty}(-\mu_0)^n\mathcal{L}^{-1}\Big(\frac{1}{S^{n\sigma+1}}\Big) + \\ \sum_{k=0}^{n-1}\sum_{n=0}^{\infty}(-\mu_0)^n N^{(k)}(0)\mathcal{L}^{-1}\Big\{\frac{1}{S^{n\sigma+k+1}}\Big\}. \end{aligned} \tag{3.17}$$

Let us recall that:

$$\mathcal{L}\{t^n\} = \frac{n!}{S^{n+1}} = \frac{\Gamma(n+1)}{S^{n+1}}$$

and

$$\mathcal{L}^{-1}\Big\{\frac{1}{S^{n+1}}\Big\} = \frac{t^n}{\Gamma(n+1)}.$$

Thus, we have:

$$N(t) \leq \frac{\Lambda}{\mu_0} - \frac{\Lambda}{\mu_0}\sum_{n=0}^{\infty}\frac{(-\mu_0 t^\sigma)^n}{\Gamma(n\sigma+1)} + \sum_{k=0}^{n-1}\sum_{n=0}^{\infty}\frac{(-\mu_0)t^{n\sigma+k}N^{(k)}(0)}{\Gamma(\sigma n+k+1)}.$$

Replacing the Mittag-Leffler function with its definition 3.7 yields:

$$N(t) \leq \frac{\Lambda}{\mu_0}(1 - E_{\sigma,1}(-\mu_0 t^\sigma)) + \sum_{k=0}^{n-1}E_{\sigma,k+1}(-\mu_0 t^\sigma)t^k N^{(k)}(0), \tag{3.18}$$

where $E_{\sigma,1}(-\mu_0 t^\sigma)$ and $E_{\sigma,k+1}(-\mu_0 t^\sigma)$ is the series of the Mittag-Leffler function that converges for every argument, from which we can say that the solutions of the model (3.10) are bounded. Thus, we have the biologically feasible region of the model (3.10) defined by:

$$\begin{aligned} \Omega = \Big\{(S(t), I(t), A(t), H(t), R(t)) \in \mathbb{R}_+^5 | N(t) \leq \frac{\Lambda}{\mu_0}(1 - E_{\sigma,1}(-\mu_0 t^\sigma)) + \\ \sum_{k=0}^{n-1}E_{\sigma,k+1}(-\mu_0 t^\sigma)t^k N^{(k)}(0)\Big\}. \end{aligned} \tag{3.19}$$

3.4. Uniqueness of the solution

Let us consider that the model (3.10) is written as follows:

$$\begin{cases} {}_0^cD_t^\sigma x(t) = F(t,x), \quad x(0) = x_0 \\ F(t,x) = Ax + g(x) + \eta, \end{cases} \tag{3.20}$$

where $F : \mathbb{R} \times \mathbb{R}^5 \longrightarrow \mathbb{R}^5$ is a vector field of dimension 5, the unknown $x = x(t) \in \mathbb{R}^5$ is the state vector defined as:

$$x(t) = (S(t), I(t), A(t), H(t), R(t))^T,$$

$$A = \begin{pmatrix} -\mu_0 & -\Lambda p & 0 & 0 & 0 \\ 0 & -k_1 & 0 & 0 & 0 \\ 0 & \epsilon\beta_1 & -k_2 & \rho\lambda & 0 \\ 0 & (1-\epsilon)\beta_1 & \beta_2 & -k_3 & 0 \\ 0 & 0 & 0 & (1-\rho)\lambda & -\mu_0 \end{pmatrix}, \qquad g(x) = \begin{pmatrix} -\dfrac{\alpha(I(t)+\eta A(t))}{N} \\ \dfrac{\alpha(I(t)+\eta A(t))}{N} \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

and

$$\eta = (\Lambda, 0, 0, 0, 0)^T.$$

The space $\mathbb{R}^5$, endowed with the standard Euclidean norm $||.||$, is a Banach space, i.e., a complete normed vector space.

Theorem 3.12. *The system* (3.20) *satisfies the Lipschitz continuity condition.*

Proof. As system (3.20) satisfies the Lipschitz continuity condition, we then obtain:

$$|F(t,x) - F(t,x^*)| = |A(x(t) - x^*(t)) + g(x(t) - g(x^*(t))|,$$

this implies

$$|F(t,x) - F(t,x^*)| \leq (||A|| + 1)||x(t) - x^*(t)||.$$

It follows that

$$||F(t,x) - F(t,x^*)|| \leq L||x(t) - x^*(t)||, \quad \text{where} \quad L = ||A|| + 1 < \infty. \tag{3.21}$$

Therefore, the function F is fully continuous and bounded. □

Concerning the Picard-Lindelöf [19, 31], we establish the following theorem:

Theorem 3.13. *Let* $0 < \sigma < 1$, $I = [0, h^*] \subseteq \mathbb{R}$ *and* $J = |x(t) - x(0)| \leq k$, *where* k *is a constant lying in the interval* $(0, 1)$.
Let $f : I \times J \longrightarrow \mathbb{R}$ *be a bounded and continuous function, that is,* $\exists! M > 0$ *such that* $|f(t,x)| \leq M$, *we know that* f *satisfies the Lipschitz conditions. If* $kL < M$, *then there exists a unique* $x \in C[0, h^*]$ *that satisfies the problem* (3.20), *where*

$$h^* = min\Big\{h, \Big(\frac{k\Gamma(\sigma+1)}{M}\Big)^{\frac{1}{\sigma}}\Big\}.$$

Proof. Let

$$T = \{x \in C[0, h^*] : ||x(t) - x(0)|| \leq k\}.$$

Given that $T \subseteq \mathbb{R}$ is a closed set, then T is a complete metric space. The continuous system (3.10) can be transformed into the following equivalent integral equations:

$${}_0^c D_t^{-\sigma} [{}_0^c D_t^{\sigma} x(t)] = {}_0^c D_t^{-\sigma} f(t, x),$$

this implies

$$x(t) - x(0) = {}_0^c I_t^{\sigma} f(t, x),$$

it follows that

$$x(t) - x(0) = \frac{1}{\Gamma(\sigma)} \int_0^t (t - \tau)^{\sigma - 1} f(\tau, x(\tau)) d\tau.$$

Thus, we obtain:

$$x(t) = x(0) + \frac{1}{\Gamma(\sigma)} \int_0^t (t - \tau)^{\sigma - 1} f(\tau, x(\tau)) d\tau. \tag{3.22}$$

The equation (3.22) is equivalent to the Volterra integral equation, which allows us to solve (3.20). Let us define an operator F^* in T

$$F^*[x] = x_0 + \frac{1}{\Gamma(\sigma)} \int_0^t (t - \tau)^{\sigma - 1} f(\tau, x(\tau)) d\tau. \tag{3.23}$$

We need to verify that the operator given in (3.23) satisfies the hypotheses of the Banach–Picard contraction theorem. We will first show that the operator F^* is invariant over T. To this aim, we compute:

$$\begin{aligned}
|F^*[x(t)] - x(0)| &= \left| \frac{1}{\Gamma(\sigma)} \int_0^t (t - \tau)^{\sigma - 1} f(\tau, x(\tau)) d\tau \right| \\
&\leq \frac{1}{\Gamma(\sigma)} \int_0^t (t - \tau)^{\sigma - 1} |f(\tau, x(\tau))| d\tau \\
&\leq \frac{1}{\Gamma(\sigma)} \int_0^t (t - \tau)^{\sigma - 1} M d\tau \\
&\leq \frac{M}{\Gamma(\sigma + 1)} t^{\sigma} \\
&\leq \frac{M}{\Gamma(\sigma + 1)} (h^*)^{\sigma} \\
&\leq \frac{M}{\Gamma(\sigma + 1)} \frac{k\Gamma(\sigma + 1)}{M} \\
|F^*[x(t)] - x(0)| &\leq k
\end{aligned}$$

Or equivalently,

$$x(0) - k \leq F^*[x(t)] \leq x(0) + k, \text{ for all } t \in [0, h^*].$$

Therefore, the operator F^* is a mapping from T into itself.
Next, we show that T is a contraction.

$$\begin{aligned}
|F^*[x](t) - F^*[x^*](t)| &= \left| \frac{1}{\Gamma(\sigma)} \int_0^t (t - \tau)^{\sigma - 1} [f(\tau, x(\tau)) - f(\tau, x^*(\tau))] d\tau \right| \\
&\leq \frac{1}{\Gamma(\sigma)} \int_0^t (t - \tau)^{\sigma - 1} |f(\tau, x(\tau)) - f(\tau, x^*(\tau))| d\tau.
\end{aligned}$$

$$\leq \frac{L}{\Gamma(\sigma+1)}||x-x^*||(h^*)^\sigma.$$
$$\leq \frac{L}{\Gamma(\sigma+1)}||x-x^*||\frac{k\Gamma(\sigma+1)}{M}.$$

Therefore,

$$||F^*[x](t)-F^*[x^*](t)|| \leq \frac{kL}{M}||x-x^*||.$$

Since $\dfrac{kL}{M} < 1$, the operator T is a contraction and consequently, by theorem 3.13, it has a unique fixed point; thus, the problem (3.10) has a unique solution. □

4. Mathematical analysis of the fractional-order model

In this section, we study the local and global stability of the disease-free equilibrium point. Before that, we remind that the disease-free equilibrium point E_0 and the basic reproduction number $\mathcal{R}_0$ are determined in the model of the reference [12], which are the same as for the model (3.10). The disease-free equilibrium point E_0 is given by:

$$E_0 = \left(\frac{\Lambda}{\mu_0}, 0, 0, 0, 0\right) \tag{4.1}$$

and the basic reproduction number $\mathcal{R}_0$ is given by:

$$\mathcal{R}_0 = \frac{\alpha[k_2k_3 - \beta_2\rho\lambda + \eta\epsilon\beta_1k_3 + \eta\rho\lambda(1-\epsilon)\beta_1]}{k_1(k_2k_3-\beta_2\rho\lambda)}. \tag{4.2}$$

4.1. Local stability of the disease-free equilibrium point

The following theorem confirms that the disease is locally and asymptotically stable at the disease-free equilibrium point:

Theorem 4.1. *The disease-free equilibrium point* $E_0 = \left(\dfrac{\Lambda}{\mu_0}, 0, 0, 0, 0\right)$ *is locally and asymptotically stable in* Ω *if* $\mathcal{R}_0 < 1$ *and unstable if* $\mathcal{R}_0 > 1$.

Proof. Let us determine the Jacobian matrix $J(E_0)$ of the model (3.10) at the disease-free equilibrium:

$$J(E_0) = \begin{pmatrix} -\mu_0 & -\Lambda P - \alpha & -\alpha\eta & 0 & 0 \\ \alpha & \alpha - k_1 & \alpha\eta & 0 & 0 \\ 0 & \epsilon\beta_1 & -k_2 & \rho\lambda & 0 \\ 0 & (1-\epsilon)\beta_1 & \beta_2 & -k_3 & 0 \\ 0 & 0 & 0 & (1-\rho)\lambda & -\mu_0 \end{pmatrix}. \tag{4.3}$$

Let $P(\Psi) = \det(J(E_0) - \Psi I_5)$ denote the characteristic polynomial of the Jacobian matrix $J(E_0)$, where Ψ represents the set of eigenvalues of $J(E_0)$ and I_5 is the identity

matrix of size 5×5

$$P(\Psi) = \begin{vmatrix} -\mu_0 - \Psi & -\Lambda p - \alpha & -\alpha\eta & 0 & 0 \\ 0 & \alpha - k_1 - \Psi & \alpha\eta & 0 & 0 \\ 0 & \epsilon\beta_1 & -k_2 - \Psi & \rho\lambda & 0 \\ 0 & (1-\epsilon)\beta_1 & \beta_2 & -k_3 - \Psi & 0 \\ 0 & 0 & 0 & (1-\rho)\lambda & -\mu_0 - \Psi \end{vmatrix}. \tag{4.4}$$

The disease-free equilibrium point is locally and asymptotically stable if all the eigenvalues of the Jacobian matrix $J(E_0)$ have negative real parts, or if $|Arg(\Psi_i)| > \dfrac{\pi\sigma}{2}$ with $i \in \{1, 2, 3, 4, 5\}$ and $0 < \sigma < 1$.
Solving $det(J(E_0) - \Psi I_5) = 0$ and after some algebraic manipulations, we obtain:

$$(\Psi + \mu_0)(\Psi + \mu_0) \begin{vmatrix} \alpha - k_1 - \Psi & \alpha\eta & 0 \\ \epsilon\beta_1 & -k_2 - \Psi & \rho\lambda \\ (1-\epsilon)\beta_1 & \beta_2 & -k_3 - \Psi \end{vmatrix} = 0. \tag{4.5}$$

We have a double eigenvalue, which is negative $(-\mu_0)$, in other words, $|Arg(-\mu_0)| = \pi > \dfrac{\pi\sigma}{2}$. Thus, the stability of E_0 depends on the remaining eigenvalues.
After algebraic computations and defining $a_1 = k_1 + k_2 + k_3 - \alpha$;
$a_2 = k_1k_2 + k_1k_3 + k_2k_3 - \alpha(k_2 + k_3) - \beta_2\rho\lambda - \alpha\eta$,
and $a_3 = k_1k_2k_3 + \alpha\beta_2\rho\lambda - k_1\beta_2\rho\lambda - \alpha k_2k_3 - \alpha\eta\epsilon\beta_1k_3 - \alpha\eta\rho\lambda(1-\epsilon)\beta_1$, we obtain the following characteristic polynomial $Q(\Psi)$ corresponding to the remaining undetermined eigenvalues:

$$Q(\Psi) = \Psi^3 + a_1\Psi^2 + a_2\Psi + a_3. \tag{4.6}$$

To demonstrate the local stability of the disease-free equilibrium point E_0 of the model (3.10) using the Routh-Hurwitz criteria, it is necessary to show that: $a_1 > 0$, $a_1a_2 - a_3 > 0$, and $a_3 > 0$.
$a_1 > 0$ because $k_1 + k_2 + k_3 > \alpha$.
$a_3 = k_1(k_2k_3 - \beta_2\rho\lambda)(1 - \mathcal{R}_0)$; $a_3 > 0$, if $\mathcal{R}_0 < 1$.
$a_1a_2 - a_3 = k_1k_2k_3(2 + \mathcal{R}_0) + \beta_2\rho\lambda k_1(1 - \mathcal{R}_0) + k_1k_2\Big(k_1 + k_2 - 2\alpha\Big) + k_1k_3\Big(k_1 + k_3 - 2\alpha\Big) + k_2k_3\Big(k_2 + k_3 - 3\alpha\Big) + (k_2 + k_3)(\alpha^2 - \alpha\eta\epsilon\beta_1) + \alpha\eta\epsilon\beta_1(\alpha - k_1) - \alpha\eta\epsilon\beta_1k_2^2 > 0$.
As the Routh-Hurwitz conditions are satisfied, we conclude that the disease-free equilibrium point is locally and asymptotically stable. □

4.2. Global stability of the disease-free equilibrium point

The following theorem confirms that the disease is globally and asymptotically stable at the disease-free equilibrium point:

Theorem 4.2. *The disease-free equilibrium point E_0 is globally and asymptotically stable in Ω when $\mathcal{R}_0 \leq 1$. If $\mathcal{R}_0 > 1$, E_0 becomes unstable, and the disease continues to spread uniformly in the population.*

Proof. The global stability analysis of the disease-free equilibrium point is based on the construction of a Lyapunov function using the matrix method proposed by

Zhisheng Shuai and P. Van den Driessche [44] and later expanded in [12]. Thus, we obtain:

$$\begin{aligned} L = \Big[\Big(\frac{(\mathcal{R}_0 - \frac{\alpha}{k_1})}{\alpha} + \frac{1}{k_1}\Big)I + \frac{k_1 k_3(\mathcal{R}_0 - \frac{\alpha}{k_1})}{\alpha(\epsilon\beta_1 k_3 + \rho\lambda(1-\epsilon)\beta_1)}A \\ + \frac{k_1\rho\lambda(\mathcal{R}_0 - \frac{\alpha}{k_1})}{\alpha(\epsilon\beta_1 k_3 + \rho\lambda(1-\epsilon)\beta_1)}H\Big]\zeta_1, \end{aligned} \tag{4.7}$$

where ζ_1 is one of the components of the non-negative left eigenvector, normalized to unit length, associated with the spectral radius $\mathcal{R}_0$ of $V^{-1}F$.
We have: L is a Lyapunov function for the system (3.3), and $L \geq 0 \ \forall \ \mathcal{R}_0 \geq \frac{\alpha}{k_1}$
The fractional derivative of L to time t in the sense of Caputo gives

$$\begin{aligned} {}_0^c D_t^\sigma L = (\mathcal{R}_0 - 1)\left(\zeta_1 I + \frac{\mu_0 k_1\Big(\mathcal{R}_0 - \frac{\alpha}{k_1}\Big)(k_2k_3 - \beta_2\rho\lambda)}{\alpha(\epsilon\beta_1 k_3 + \rho\lambda(1-\epsilon)\beta_1}\zeta_1 A\right) \\ - \left(\frac{1}{k_1} + \frac{\Big(\mathcal{R}_0 - \frac{\alpha}{k_1}\Big)}{\alpha}\right)\alpha(I + \eta A)\left(1 - \frac{S}{N}\right)\zeta_1. \end{aligned} \tag{4.8}$$

Since $S \leq N$, if $\mathcal{R}_0 \leq 1$ then ${}_0^c D_t^\sigma L < 0$. Moreover ${}_0^c D_t^\sigma L = 0$ for $I = A = 0$ and $S = \frac{\Lambda}{\mu_0}$. Thus, the largest invariant set of the model (3.10), when ${}_0^c D_t^\sigma L = 0$, is the disease-free equilibrium point E_0.
We have formulated a suitable Lyapunov function for system (3.10) and shown that it strictly decreases along the system's trajectories, i.e., ${}_0^c D_t^\sigma L \leq 0$.
As a result, by LaSalle's invariance principle [31], E_0 is globally and asymptotically stable. □

5. Sensitivity analysis

In this section, we study the sensitivity analysis of the basic reproduction number $\mathcal{R}_0$. Sensitivity analysis is a crucial technique for complex systems, as it helps determine the influence of the model parameters in (3.10) on the transmission of HIV/AIDS. Sensitivity analysis enables us to understand how changes in the model parameters affect the disease's propagation dynamics [4, 5, 35, 53]. By performing local sensitivity analysis, we can evaluate the effect of a single parameter on the basic reproduction number $\mathcal{R}_0$ while keeping the other parameters constant.
According to the section 4,

$$\mathcal{R}_0 = \frac{\alpha[k_2k_3 - \beta_2\rho\lambda + \eta\epsilon\beta_1 k_3 + \eta\rho\lambda(1-\epsilon)\beta_1]}{k_1(k_2k_3 - \beta_2\rho\lambda)}.$$

By setting $\Gamma = k_2k_3 - \beta_2\rho\lambda + \eta\epsilon\beta_1k_3 + \eta\rho\lambda(1-\epsilon)\beta_1$ and $\Omega = k_1(k_2k_3 - \beta_2\rho\lambda)$ then:

$$\mathcal{R}_0 = \alpha\frac{\Gamma}{\Omega}.$$

Definition 5.1. [15] *The normalized sensitivity index of $\mathcal{R}_0$, which depends differentially on a parameter p, is given by:*

$$\gamma_p^{\mathcal{R}_0} = \frac{\partial\mathcal{R}_0}{\partial p}.\frac{p}{\mathcal{R}_0}. \tag{5.1}$$

For conducting the sensitivity analysis, other authors [4, 5, 39, 46, 47, 53] have also used this fundamental formula to calculate the sensitivity index of each parameter appearing in $\mathcal{R}_0$.

The expressions for the sensitivity indices of the 11 parameters of $\mathcal{R}_0$ are as follows:

$\gamma_\alpha^{\mathcal{R}_0} = +1$; $\gamma_p^{\mathcal{R}_0} = \dfrac{\Lambda p}{k_1}$; $\gamma_\Lambda^{\mathcal{R}_0} = \dfrac{\Lambda p}{k_1}$; $\gamma_{\beta_1}^{\mathcal{R}_0} = \dfrac{\beta_1\eta[\epsilon k_3 + \rho\lambda(1-\epsilon)]}{\Gamma} - \dfrac{\beta_1(k_2k_3 - \beta_2\rho\lambda)}{\Omega}$;

$\gamma_{\beta_2}^{\mathcal{R}_0} = \dfrac{\beta_2(k_3 - \rho\lambda)}{\Gamma} - \dfrac{\beta_2k_1(k_3 - \rho\lambda)}{\Omega}$

$\gamma_{\mu_0}^{\mathcal{R}_0} = \dfrac{\mu_0(k_2 + k_3 + \eta\epsilon\beta_1)}{\Gamma} - \dfrac{\mu_0[k_2k_3 - \beta_2\rho\lambda + k_1(k_2 + k_3)]}{\Omega}$; $\gamma_{\mu_1}^{\mathcal{R}_0} = \dfrac{\mu_1k_3}{\Gamma} - \dfrac{\mu_1k_1k_3}{\Omega}$;

$\gamma_\lambda^{\mathcal{R}_0} = \dfrac{\lambda[k_2 - \beta_2\rho + \eta\epsilon\beta_1 + \eta\rho(1-\epsilon)\beta_1]}{\Gamma} - \dfrac{\lambda k_1k_2}{\Omega}$

$\gamma_\rho^{\mathcal{R}_0} = \dfrac{\rho[-\beta_2\lambda + \eta\lambda(1-\epsilon)\beta_1]}{\Gamma} + \dfrac{\rho k_1\beta_2\lambda}{\Omega}$; $\gamma_\eta^{\mathcal{R}_0} = \dfrac{\eta\beta_1[\epsilon k_3 + \rho\lambda(1-\epsilon)]}{\Gamma}$;

$\gamma_\epsilon^{\mathcal{R}_0} = \dfrac{\epsilon\eta\beta_1(k_3 - \rho\lambda)}{\Gamma}$.

Table 1 gives a description and the values of the parameters, and Table 2 gives sensitivity indices of the parameters.

Parameters	Meaning	Values	Reference
N(0)	Total population	100000	assumed
Λ	Recruitment rate	380	assumed
α	HIV/AIDS transmission rate	0.00011	assumed
β_1	Rate of transfer from the compartment of infected individuals without clinical signs to the AIDS compartment	0.3	[50]
β_2	rate of transfer from the AIDS class to the compartment of individuals on treatment	0.33	[14]
λ	rate of transfer of individuals on treatment to the AIDS class	0.2	assumed
η	AIDS infectivity versus HIV infection without clinical signs of the disease	1.05	[45]
p	probability of HIV-positive newborns through vertical transmission	0.0001	Fitting
ϵ	probability of infected individuals without clinical signs progressing to the AIDS phase	0.1	assumed
ρ	Probability of individuals who have failed their treatment and move up to the AIDS class	0.09	[14]
μ_0	HIV natural mortality rate	1/57	assumed
μ_1	HIV-induced mortality rate	0.7	assumed

TABLE 1. Values of the parameters used in the model (3.3) of HIV/AIDS

Parameters	Sensitivity analysis
α	+1
Λ	+0.1359
p	+0.1359
β_1	-1.0221
β_2	-0.0152
λ	-0.0249
η	+0.0511
ϵ	+0.0269
ρ	+0.0232
μ_0	-0.0655
μ_1	-0.351

TABLE 2. Values of the sensitivity indices of the parameters of $\mathcal{R}_0$.

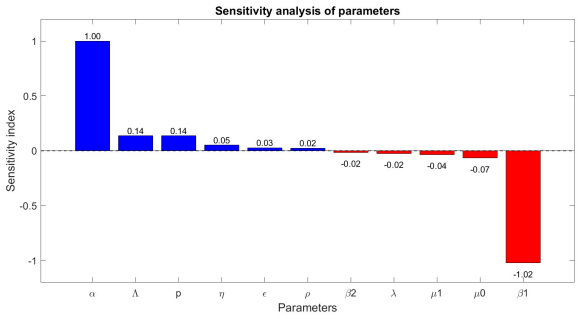

FIGURE 1. Diagram of the sensitivity analysis of the parameters of $\mathcal{R}_0$

The parameter with a sensitivity index of high absolute value is more influential than the one with a low sensitivity index in absolute value, and the signs of the sensitivity indices of the parameters show their positive or negative impacts in the model. From the sensitivity analysis of $\mathcal{R}_0$, we observe that α and β_1 are the most influential parameters of $\mathcal{R}0$. Indeed, $\gamma\alpha^{\mathcal{R}_0} = +1$, so a 1% increase in α leads to a 1% increase in $\mathcal{R}0$; $\gamma{\beta_1}^{\mathcal{R}_0} = -1.0221$ means that a 1% increase in β_1 leads to a 1.0221% decrease in $\mathcal{R}_0$. The same interpretation can be applied to the remaining parameters in the expression of $\mathcal{R}_0$, depending on the sign of the sensitivity index [1, 4, 5, 15, 35, 46, 47]. We can say that an increase in the parameters with positive sensitivity indices leads to a proportional increase in $\mathcal{R}_0$, while an increase in the parameters with negative sensitivity indices results in an inversely proportional change in $\mathcal{R}_0$. It is imperative to implement strategic initiatives aimed at reducing the transmission of HIV/AIDS and managing the progression of individuals from the asymptomatic infection stage to the stage of full-blown AIDS. In this regard, the promotion of preventive measures and the expanded access to antiretroviral therapy should be considered public health priorities.

6. Numerical results and discussions

This section focuses on the numerical simulation of the system (3.10). The goal is to investigate how the fractional order σ influences the transmission and control of HIV/AIDS, using the model represented by this system.

6.1. Discretization of the model (3.3)

In this section, we present a numerical simulation of the results of the system (3.10). The objective is to examine the impact of the fractional order σ on the transmission and control of HIV/AIDS, through the model (3.10). To achieve this, we use the iterative Euler method for fractional differential equations, where the fractional integral of each variable in system (3.3) is approximated by a discrete sum (See [27, 28, 37, 49] for more details on Euler methods for ordinary and fractional differential equations). This approach involves transforming the fractional differential equation into an integral equation and then solving it by applying a discrete method.
We introduce a uniform time discretization of the interval $[0, T]$, defined by the points $t_k = k\Delta t$ for $k = 0, ..., n_T$, where $\Delta t = \dfrac{T}{n_T}$ is the step size, and $n_T \in \mathbb{N}^*$ is the number of subdivisions. This discretization is used to apply the generalized Euler method to a fractional-order differential equation.
By considering system (3.12), and setting $t = t_{n+1}$, $n \in \mathbb{N}$, we obtain:

$$\begin{cases} S(t_{n+1}) = S_0 + \dfrac{1}{\Gamma(\sigma)} \int_0^{t_{n+1}} (t_{n+1} - \tau)^{\sigma-1} \\ \times \Big(\Lambda - \Lambda p I(\tau) - \mu_0 S(\tau) - \dfrac{\alpha S(\tau)(I(\tau) + \eta A(\tau))}{N}\Big) d\tau \\ I(t_{n+1}) = I_0 + \dfrac{1}{\Gamma(\sigma)} \int_0^{t_{n+1}} (t_{n+1} - \tau)^{\sigma-1} \Big(\dfrac{\alpha S(\tau)(I(\tau) + \eta A(\tau))}{N} - k_1 I(\tau)\Big) d\tau \\ A(t_{n+1}) = A_0 + \dfrac{1}{\Gamma(\sigma)} \int_0^{t_{n+1}} (t_{n+1} - \tau)^{\sigma-1} \Big(\epsilon\beta_1 I(\tau) - k_2 A(\tau) + \rho\lambda H(\tau)\Big) d\tau \\ H(t_{n+1}) = H_0 + \dfrac{1}{\Gamma(\sigma)} \int_0^{t_{n+1}} (t_{n+1} - \tau)^{\sigma-1} \Big((1-\epsilon)\beta_1 I(\tau) + \beta_2 A(\tau) - k_2 H(\tau)\Big) d\tau \\ R(t_{n+1}) = R_0 + \dfrac{1}{\Gamma(\sigma)} \int_0^{t_{n+1}} (t_{n+1} - \tau)^{\sigma-1} \Big((1-\rho)\lambda H(\tau) - \mu_0 R(\tau)\Big) d\tau. \end{cases} \tag{6.1}$$

After some algebraic manipulations and setting:

$$f(s, u(s))_{|_{[t_k, t_{k+1}]}} \simeq f(t_k, u(t_k)) \quad with \quad u(t) = (S(t), I(t), A(t), H(t), R(t)), \tag{6.2}$$

we have:

$$
\begin{cases}
S(t_{n+1}) = S_0 + \dfrac{\Delta t^\sigma}{\Gamma(\sigma+1)} \displaystyle\sum_{k=0}^{n} [(n-k+1)^\sigma - (n-k)^\sigma] \\
\qquad \times \left(\Lambda - \Lambda p I(t_k) - \mu_0 S(t_k) - \dfrac{\alpha S(t_k)(I(t_k) + \eta A(t_k))}{N} \right) \\
I(t_{n+1}) = I_0 + \dfrac{\Delta t^\sigma}{\Gamma(\sigma+1)} \displaystyle\sum_{k=0}^{n} [(n-k+1)^\sigma - (n-k)^\sigma] \\
\qquad \times \left(\dfrac{\alpha S(t_k)(I(t_k) + \eta A(t_k))}{N} + k_1 I(t_k) \right) \\
A(t_{n+1}) = A_0 + \dfrac{\Delta t^\sigma}{\Gamma(\sigma+1)} \displaystyle\sum_{k=0}^{n} [(n-k+1)^\sigma - (n-k)^\sigma] \\
\qquad \times (\epsilon \beta_1 I(t_k) - k_2 A(t_k) + \rho \lambda H(t_k)) \\
H(t_{n+1}) = H_0 + \dfrac{\Delta t^\sigma}{\Gamma(\sigma+1)} \displaystyle\sum_{k=0}^{n} [(n-k+1)^\sigma - (n-k)^\sigma] \\
\qquad \times ((1-\epsilon)\beta_1 I(t_k) + \beta_2 A(t_k) - k_3 H(t_k)) \\
R(t_{n+1}) = R_0 + \dfrac{\Delta t^\sigma}{\Gamma(\sigma+1)} \displaystyle\sum_{k=0}^{n} [(n-k+1)^\sigma - (n-k)^\sigma] \\
\qquad \times ((1-\rho)\lambda H(t_k) - \mu_0 R(t_k)) \,.
\end{cases}
\tag{6.3}
$$

6.2. Numerical results

To study the effects of the fractional order on the model (3.3), we implemented in MATLAB, inspired by the work of [27, 28, 37, 49], the approximate solution (6.3) based on the Euler method, for different values of σ. The parameter values used for the numerical simulation of the model (3.10) are derived from the literature or assumed to be biologically plausible (see Table 1). The system (3.10) is considered subject to the initial conditions given by:

$$
S(0) = 89040; I(0) = 4700; A(0) = 100; H(0) = 3580; R(0) = 2580, \tag{6.4}
$$

the values of the parameters from Table 1 and a final time value of $T = 10$ (years). The following figures present the graphs of the approximate solutions:

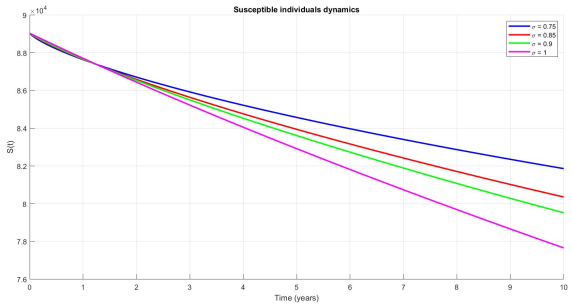

FIGURE 2. Dynamics of the susceptible individuals compartment of the model (3.10) when $\sigma = 0.75, 0.85, 0.90, 1$

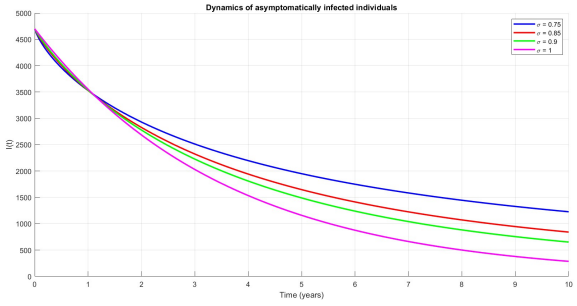

FIGURE 3. Dynamics of infected without clinical signs compartment of the model (3.10) when $\sigma = 0.75, 0.85, 0.90, 1$.

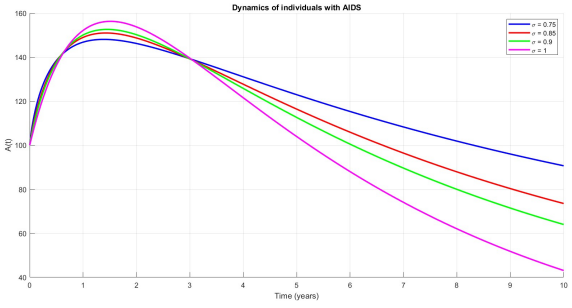

FIGURE 4. Dynamics of infected with clinical signs compartment of the model (3.10) when $\sigma = 0.75, 0.85, 0.90, 1$.

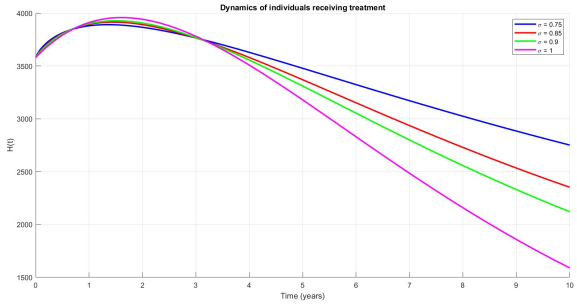

FIGURE 5. Dynamics of the treated individuals compartment of the model (3.10) when $\sigma = 0.75, 0.85, 0.90, 1$.

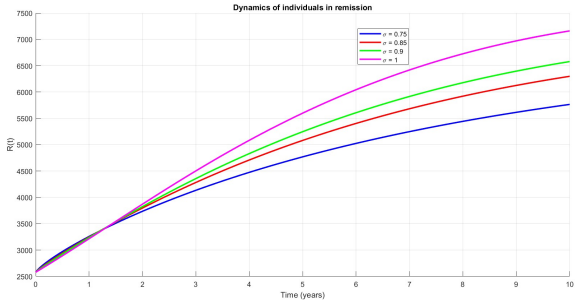

FIGURE 6. Dynamics of individuals in remission compartment of the model (3.10) when $\sigma = 0.75, 0.85, 0.90, 1$.

In figures 2 to 6, the graphs show the dynamics of susceptible individuals, individuals infected without clinical signs of the disease, individuals with AIDS, individuals infected with HIV under antiretroviral treatment, and individuals in remission for different values of the fractional order σ.

In the compartment of susceptible individuals, the decrease in the dynamics of individuals is less pronounced as the fractional order decreases. Specifically, the smallest decrease is observed for $\sigma = 0.75$, while the largest decrease is noted for $\sigma = 1$.

In the class of infected individuals, with or without clinical signs of AIDS, the dynamics of individuals' decline and the rate of decline are directly linked to the fractional order. Thus, the smallest decline is observed for $\sigma = 0.75$, and the largest decline is noted for $\sigma = 1$.

In the compartment of individuals under treatment, the dynamics of individuals decrease, with a reduction rate proportional to the fractional order, as in the previous cases.

In the class of individuals in remission, the dynamics of individuals increase, with a growth rate proportional to the fractional order. Specifically, the most significant

increase is observed for $\sigma = 1$, while the smallest increase is noted for $\sigma = 0.75$. In light of the foregoing, the results indicate that the fractional-order model, by its ability to incorporate long-term memory effects, offers a more relevant framework than the deterministic model for capturing the dynamics of HIV transmission. Specifically, variations in the fractional order produce distinct trajectories, thereby enabling a more refined representation of transmission dynamics and enhancing the effectiveness of strategies aimed at curbing the spread of the virus, as demonstrated in the figures presented above.

7. Conclusion

In this study, we first analyzed the transmission of HIV/AIDS using a fractional model in the sense of Caputo. We applied Banach's contraction principle to demonstrate the existence of a unique solution for the developed model and subsequently performed stability analyses of the disease-free equilibrium point. Next, we conducted a sensitivity analysis of the basic reproduction number $\mathcal{R}_0$ concerning the various parameters of the fractional model. We found that the transmission rate and the progression rate of individuals from the group of infected individuals who show no clinical signs to the AIDS compartment are the most sensitive parameters of $\mathcal{R}_0$. Finally, approximate solutions of the fractional-order model were simulated for various values of the fractional order, and the corresponding results were analyzed accordingly. The study revealed that the fractional model offers a superior fit compared to the deterministic model, primarily due to its ability to capture long-term dynamics such as infection relapses and delayed responses to antiretroviral therapy. Moreover, this modeling framework allows for the generation of distinct trajectories based on variations in the fractional order, thereby providing a more nuanced representation of viral transmission. Consequently, the outcomes derived from this approach are particularly relevant for predicting the spread of HIV/AIDS within a population while accounting for the variability introduced by the fractional order.

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