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On the existence, uniqueness, stability, and numerical aspects for a novel mathematical model of HIV/AIDS transmission by a fractal fractional order derivative

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Abstract

In this study, we explore a mathematical model of the transmission of HIV/AIDS. The model incorporates a fractal fractional order derivative with a power-law type kernel. We prove the existence and uniqueness of a solution for the model and establish the stability conditions by employing Banach's contraction principle and a generalized α-ψ-Geraghty type contraction. We perform stability analysis based on the Ulam–Hyers concept. To calculate the approximate solution, we utilize Gegenbauer polynomials via the spectral collocation method. The presented model includes two fractal and fractional order derivatives. The influence of the fractional and fractal derivatives on the outbreak of HIV is investigated by utilizing real data from the Cape Verde Islands in 1987–2014.

Keywords: Mathematical model of HIV/AIDS transmission; Fractal fractional derivative; Stability; α-ψ-Geraghty type contraction; Gegenbauer polynomials

1 Introduction

HIV, which stands for Human Immunodeficiency Virus, is a highly contagious virus that attacks the immune system of the human body. In individuals with acquired immunodeficiency syndrome (AIDS), the immune system is severely compromised. When HIV attacks the immune system, it impairs its ability to combat infections that a healthy body would typically be able to handle. Over time, the immune cells of those infected with this virus are gradually destroyed or impaired, resulting in immunodeficiency. Immune function can often be measured by examining the levels of $CD4^+$ T-cells. If left untreated, an HIV-infected person typically develops AIDS after approximately 10 to 15 years. Unfortunately, individuals afflicted with AIDS are at a high risk of developing severe clinical manifestations. First identified in the 1980s, HIV has become a global health problem, affecting millions of people worldwide. It is primarily transmitted through contact with certain bodily fluids, such as blood, semen, vaginal fluids, and breast milk.

Once a person becomes infected with HIV, the virus gradually weakens their immune system by targeting and destroying $CD4^+$ cells, which are crucial for fighting off infections and diseases. As the immune system weakens, the infected individual becomes increasingly susceptible to various infections, cancers, and opportunistic diseases.

There is no cure for HIV, but with the advances in medical science, an effective treatment strategy called antiretroviral therapy (ART) has been developed. ART helps control the virus, maintains a healthy immune system, and prevents the progression of HIV to AIDS. Additionally, various preventive measures are available, such as safe sexual practices, regular testing, and access to clean needles, which can significantly reduce the transmission of the virus.

Mathematical modeling is an essential tool used to study and understand complex phenomena, including the spread and impact of infectious diseases such as HIV/AIDS. This method involves applying mathematical equations and techniques to describe, analyze, and predict the transmission of these diseases in populations. The utilization of mathematical modeling in the context of HIV/AIDS has greatly contributed to our understanding, to decision-making, and to the implementation of effective prevention and control strategies. By allowing researchers to explore different scenarios and assess the potential impact of interventions, mathematical modeling has played a crucial role in shaping public health policies related to HIV/AIDS. The application of mathematical modeling techniques to study HIV/AIDS dates back to the early years of the epidemic. In the 1980s, as the virus started to spread rapidly worldwide, researchers began to recognize the need for a quantitative approach to understand its dynamics. This gave rise to the first mathematical models that aimed to capture the transmission, progression, and impact of HIV/AIDS within populations. Initially, these early models relied on simple mathematical frameworks, often using concepts from epidemiology and population dynamics.

Over time, the mathematical modeling of HIV/AIDS has evolved, becoming more complex and realistic. Researchers started integrating social and behavioral factors, such as sexual behavior, drug use, and individual-level heterogeneity, in their models. This shift allowed for a more comprehensive understanding of the epidemic, enabling the evaluation of various interventions, including behavioral interventions, HIV testing strategies, ART scale-up, and vaccination campaigns.

Advances in computer technology and increased availability of data have further enhanced the sophistication and accuracy of HIV/AIDS models. Researchers can now develop more complex, agent-based models that simulate individual behaviors and interactions within populations. These models can capture the dynamics of HIV transmission at different scales, from local communities to entire regions or countries.

The field of HIV/AIDS mathematical modeling has also played a critical role in informing policy decisions. Researchers have collaborated with policymakers, health organizations, and stakeholders to provide evidence on the potential impact and cost-effectiveness of different interventions. This collaboration has led to more targeted prevention strategies, increased access to treatment and care, and an improved global response to the HIV/AIDS epidemic.

Numerous authors across the globe have contributed articles focusing on HIV/AIDS to increase social awareness. There has been recent progress in the research field of HIV/AIDS transmission dynamics through the development of various mathematical models.

In recent years, fractional order models have consistently demonstrated superior accuracy compared with models with integer orders when describing some aspects of natural phenomena [1]. As a result, the fractional differential equations are widely employed in mathematical modeling of various diseases and engineering disciplines, such as analysis of the transmission of Middle East Respiratory Syndrome Coronavirus [2], Hopf bifurcation control of a fractional order delayed turbidostat model [3], mathematical modeling of the human liver with the Caputo–Fabrizio fractional derivative [4], numerical approximation and analysis of an epidemic model of drinking with constant proportional Caputo operator [5], solving fractional diffusion wave equations by using fractional wavelets [6], analysis of age-wise fractional order problems for COVID-19 with a non-singular Mittag-Leffler kernel [7], and stability analysis of fractional order uncertain BAM neural networks with mixed time delays [8].

In regard to the dynamics of HIV/AIDS transmission using fractional modeling, there are some valuable studies, for instance a mathematical model of HIV/AIDS transmission in a homogeneous mixing population, examining the impact of population size [9], utilizing the Caputo fractional derivative operator for analysis of the dynamics of HIV in East Asia, incorporating real statistics [10], and analysis of an HIV/AIDS model with Mittag-Leffler kernel [11].

One notable contribution was made by Atangana [12], who introduced the revolutionary concept of fractal fractional (FF) derivatives. A combination of fractal derivatives, alongside the generalized Mittag-Leffler law, exponential law, and power-law, constitutes the basis for constructing these derivatives. These operators have demonstrated a remarkable level of accuracy in numerically simulating solutions for various FF systems, leading to the development of several FF models by other mathematicians. For instance, the authors of [13] explored a model for malaria by using FF derivatives. The effect of FF operators in mathematical modeling of corruption was studied in [14]. Shah et al. investigated the outbreak of SARS-CoV-2 in Pakistan utilizing FF derivatives [15], and Farman et al. analyzed a fractional order COVID-19 model with Mittag-Leffler kernel [16]. In [17], the FF mathematical problem of an Atangana–Baleanu in the sense of Caputo fractional operator by a non-singular Mittag-Leffler kernel was studied. A TB and HIV co-infection model with Mittag-Leffler FF derivative was analyzed in [18]. The authors of [19] studied an FF waterborne disease model. A mathematical analysis of an FF model of the AH1N1/09 virus and its generalized Caputo type version is presented in [20]. FF derivative operators have been generalized in engineering branches, such as analysis of the FF nonlinear coupled Burgers equation by a coupled fixed point (FP) and hybrid generalized integral transform approach [21], investigation of the FF nonlinear Korteweg–de-Vries–Schrödinger system with power-law kernel [22], and dynamical properties of a meminductor chaotic system with FF power-law operator [23].

Etemad et al. introduced an advanced mathematical model of the transmission cycle of viruses causing Crimean-Congo hemorrhagic fever in an FF system with power-law type kernels [24]. Motivated by their study, in the present research, we present a novel FF model to describe HIV/AIDS transmission and employ a generalized α-ψ-Geraghty type contraction to establish the existence of a unique solution.

The structure of this article is as follows. The definitions of FF operators based on the Riemann–Liouville kernel of power-law type and some important theorems, which are needed in the sequel, are given in Sect. 2. The Gegenbauer polynomials (GPs) are also

briefly defined in Sect. 2. Section 3 is devoted to introducing the mathematical model of HIV/AIDS transmission by both integer and FF order derivatives. The existence and uniqueness of the model solutions are discussed in Sect. 4, with the stability theorems of the mentioned model. Simulations are reported in Sect. 5 based on real data where the impact of the fractional and fractal orders are assessed in detail. The conclusion of this research is presented in Sect. 6.

2 Preliminary definitions

In this section, first, we state the definitions of FF operators based on the Riemann–Liouville kernel of power-law type and some concepts which are required in the next sections. Also, the definitions of GPs and shifted GPs (SGPs) are given, which are the basis functions of the proposed numerical scheme.

Definition 2.1 Let $g \in C(t_0, t_f)$ be a fractal differentiable function of order μ. The FF derivative operator of order (ν, μ) $(n-1 < \nu,\ \mu \leq n \in \mathbb{N})$ is defined as [12]

$$^{\mathrm{FFP}}\mathbf{D}_{t_0,\mathrm{t}}^{\nu,\mu}\{g(\mathrm{t})\} = \frac{1}{\Gamma(n-\nu)}\frac{\mathrm{d}}{\mathrm{d}\mathrm{t}^{\mu}}\int_{t_0}^{\mathrm{t}}(\mathrm{t}-\eta)^{n-\nu-1}g(\eta)\,\mathrm{d}\eta,$$

where $\frac{\mathrm{d}g(\eta)}{\mathrm{d}\eta^{\mu}} = \lim_{\mathrm{t}\to\eta}\frac{g(\mathrm{t})-g(\eta)}{\mathrm{t}^{\mu}-\eta^{\mu}}$ indicates the fractal derivative of g.

The conventional fractional Riemann–Liouville derivative $^{\mathbf{RL}}\mathbf{D}_{t_0,\mathrm{t}}^{\nu}$ is the result of simplifying the FF derivative $^{\mathrm{FFP}}\mathbf{D}_{t_0,\mathrm{t}}^{\nu,\mu}$ regarding $\mu = 1$.

Definition 2.2 The FF integral of order (ν, μ) for the mentioned function g in Definition 2.1 is defined as [12]

$$^{\mathrm{FFP}}\mathfrak{I}_{t_0,\mathrm{t}}^{\rho,\mu}\{g(\mathrm{t})\} = \frac{\mu}{\Gamma(\rho)}\int_{t_0}^{\mathrm{t}}\eta^{\mu-1}(\mathrm{t}-\eta)^{\nu-1}g(\eta)\,\mathrm{d}\eta. \tag{1}$$

For a metric space (Y, d), the following definitions hold. In the following we denote $[0, \infty)$ by $\mathcal{B}$.

Definition 2.3 [25] Let $\alpha : Y \times Y \to \mathcal{B}$ be a function. The mapping $T : Y \to Y$ is said to be α-admissible if

$$\forall x, y \in Y; \quad \alpha(x, y) \geq 1 \quad \Longrightarrow \quad \alpha(Tx, Ty) \geq 1.$$

Now, we define

$$\Psi = \left\{\psi : \mathcal{B} \to \mathcal{B} | \psi^{-1}\big(\{0\}\big) = \{0\}, \psi(t) \leq t\right\},$$

where ψ is a continuously increasing function and

$$\mathcal{F} = \left\{\beta : \mathcal{B} \to [0, 1) \Big| \lim_{n\to\infty}\beta(t_n) = 1 \text{ implies } \lim_{n\to\infty} t_n = 0\right\}.$$

Definition 2.4 Let $T : Y \to Y$ be a generalized α-ψ-Geraghty contractive mapping, where there exists $\alpha : Y \times Y \to \mathcal{B}$ with the following condition:

$$\alpha(x,y)\psi\big(d(Tx,Ty)\big) \le \beta\big(\psi\big(d(x,y)\big)\big)\psi\big(d(x,y)\big), \tag{2}$$

for $x, y \in Y$, with $\beta \in \mathcal{F}$ and $\psi \in \Psi$.

2.1 Gegenbauer polynomials

Now, we briefly review the definition and properties of GPs (or ultraspherical polynomials) and SGPs on $[0, T]$.

The GP of degree m, $G_{m,\beta}$, in the interval $[-1, 1]$ is the particular solution of the following Gegenbauer equation:

$$\big(1-t^2\big)u'' - (2\beta+1)tu' + m(m+2\beta)u = 0, \quad \beta > -\frac{1}{2}.$$

By putting $\alpha = \frac{1}{2}$ and 1, GPs are reduced to the well-known Legendre and Chebyshev polynomials, respectively. The analytic form of these polynomials is [26]

$$G_{m,\beta}(t) = \sum_{j=0}^{\lfloor \frac{m}{2} \rfloor} (-1)^j \frac{2^{m-2j}\Gamma(m-j+\beta)}{\Gamma(\beta)j!(m-2j)!} t^{m-2j}, \quad m \ge 1. \tag{3}$$

GPs satisfy the following recurrence relation:

$$G_{m+1,\beta}(t) = \frac{1}{m+1}\big(2t(m+\beta)G_{m,\beta}(t) - (m+2\beta-1)G_{m-1,\beta}(t)\big), \quad m \ge 1. \tag{4}$$

These polynomials are orthogonal in the interval $[-1, 1]$ with respect to the weight function $\omega_\beta(t) = (1-t^2)^{\beta-\frac{1}{2}}$:

$$\big\langle G_{i,\beta}(t), G_{j,\beta}(t)\big\rangle_{\omega_\beta} = \int_{-1}^{1} G_{i,\beta}(t)G_{j,\beta}(t)\omega_\beta(t)\,dt = \varrho_{m,\beta}\delta_{ij}, \tag{5}$$

where $\varrho_{m,\beta} = \frac{\pi\Gamma(m+2\beta)}{2^{2\beta-1}m!(m+\beta)(\Gamma(\beta))^2}$ and δ_{ij} is the Kronecker delta function.

2.1.1 Shifted Gegenbauer polynomials

In order to employ GPs in an arbitrary interval $[0, T]$, we should carry them into the mentioned interval, with $T \in \mathbb{R}^+$. So, we introduce the SGPs (denoted as $\mathcal{G}_{m,\beta}$) by using an appropriate variable change:

$$\mathcal{G}_{m,\beta}(t) = G_{m,\beta}(2t-1). \tag{6}$$

It is clear that the following recurrence and orthogonality relations are satisfied for SGPs:

$$\mathcal{G}_{m+1,\beta}(t) = \frac{1}{m+1}\big((4t-2)(m+\beta)\mathcal{G}_{m,\beta}(t) - (m+2\beta-1)\mathcal{G}_{m-1,\beta}(t)\big), \tag{7}$$

$$\int_0^T \mathcal{G}_{i,\beta}(t)\mathcal{G}_{j,\beta}(t)\omega_\beta(t)\,dt = \varrho_{m,\beta}\delta_{ij}, \tag{8}$$

where $\omega_\beta(t) = 2^{2\beta-1}(t-t^2)^{\beta-\frac{1}{2}}$.

2.1.2 Function approximation by shifted Gegenbauer polynomials

Suppose $S_N^\beta = span\{\mathcal{G}_{1,\beta}(t), \dots, \mathcal{G}_{N,\beta}(t)\}$ and $u(t) \in L^2[0,T]$. We can express $u(t)$ in S_N^β by u_N as

$$u(t) \simeq u_N(t) = \sum_{m=1}^{N} c_m \mathcal{G}_{m,\beta}(t), \tag{9}$$

where the coefficients c_m are determined by

$$c_m = \frac{1}{\varrho_{m,\beta}} \langle u(t), \mathcal{G}_{m,\beta}(t) \rangle_{\omega_\beta}.$$

We can rewrite (9) in vector form as

$$u_N(t) = C_N^T \mathbf{G}_{N,\beta}(t), \tag{10}$$

where

$$C_N = (c_1, \dots, c_N)^T, \qquad \mathbf{G}_{N,\beta}(t) = \big(\mathcal{G}_{1,\beta}(t), \dots, \mathcal{G}_{N,\beta}(t)\big)^T. \tag{11}$$

3 Description of the model by an integer order model

Here we study a novel model of HIV/AIDS transmission by an FF order derivative with power-law type kernel. The existence of solutions will be investigated in terms of their non-negativity. Also, we examine the uniqueness of solutions in the following. We propose the model 1 from reference [27].

3.1 Mathematical model of HIV/AIDS transmission with integer order

We consider the following model of HIV/AIDS transmission [27]:

$$\begin{cases} \dot{\mathcal{P}}_s(t) = \Lambda - \Psi\Gamma(t)\mathcal{P}_s(t) - \zeta\mathcal{P}_s(t), \\ \dot{\mathcal{P}}_i(t) = -(\zeta + \Psi_l + \delta_l)\mathcal{P}_i(t) + \delta_c\mathcal{P}_c(t) + \tau_{AIDS}\mathcal{P}_a(t) + \Psi\Gamma(t)\mathcal{P}_s(t), \\ \dot{\mathcal{P}}_c(t) = -(\zeta + \delta_c)\mathcal{P}_c(t) + \Psi_l\mathcal{P}_i(t), \\ \dot{\mathcal{P}}_a(t) = -(\zeta + \zeta_{AIDS} + \tau_{AIDS})\mathcal{P}_a(t) + \delta_l\mathcal{P}_i(t). \end{cases} \tag{12}$$

The human population in this model is divided into four categories. Susceptible people, $\mathcal{P}_s(t)$, are in the first category. We denote HIV-positive individuals with no signs of AIDS by $\mathcal{P}_i(t)$. People in the chronic stage are denoted by $\mathcal{P}_c(t)$, including people who tested positive for HIV and use ART medication. Finally, $\mathcal{P}_a(t)$ denotes individuals who test positive for HIV and have AIDS clinical manifestations. Moreover, $N(t) = \mathcal{P}_s(t) + \mathcal{P}_i(t) + \mathcal{P}_c(t) + \mathcal{P}_a(t)$ is the total population at time t. Figure 1 denotes the diagram of HIV/AIDS transmission. We use the following notations:

- $\Gamma(t) = \frac{\mathcal{P}_i(t)}{N(t)}$,
- Ψ denotes the rate of affective contact leading to HIV/AIDS transmission,
- Λ is the rate of recruitment,
- ζ is the natural death rate,
- Ψ_l is the HIV treatment rate for individuals in $\mathcal{P}_i$,

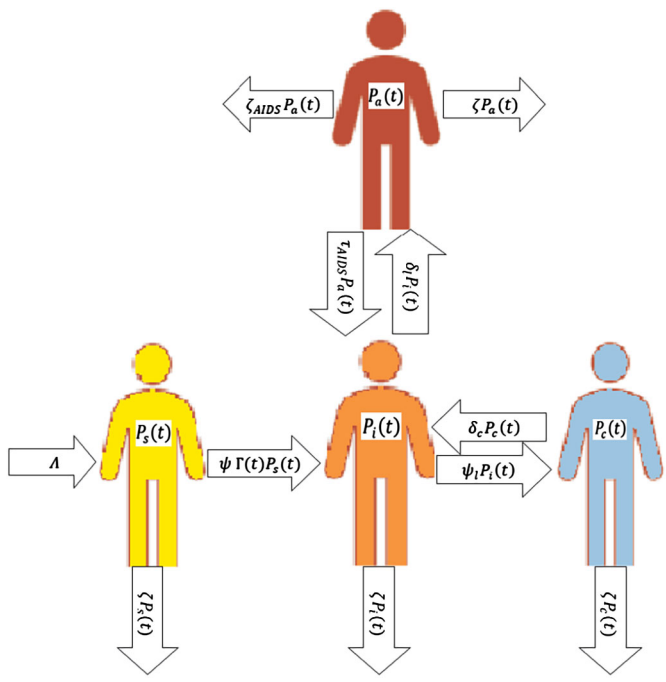

Figure 1 Diagram of HIV/AIDS transmission in a human population

- τ_{AIDS} is the spread rate of AIDS,
- δ_l is the default treatment rate of $\mathcal{P}_i$,
- ζ_{AIDS} is the death rate by AIDS,
- δ_c is the default treatment rate of $\mathcal{P}_c$.

Lemma 3.1 *Let $X(t) = (\mathcal{P}_s(t), \mathcal{P}_i(t), \mathcal{P}_c(t), \mathcal{P}_a(t))^T$ be a state vector of the model (12). Also, $X(0) = (\mathcal{P}_s(0), \mathcal{P}_i(0), \mathcal{P}_c(0), \mathcal{P}_a(0))^T \geq 0$ is a non-negative vector. Then the model (12) is non-negative whenever $t > 0$. Also $\lim_{t\to\infty} N(t) \leq \frac{\Lambda}{\zeta}$.*

3.2 Description of HIV/AIDS transmission by the fractal fractional order derivative

Motivated by the above integer order model of HIV/AIDS transmission and a new definition of fractional derivative in the FF sense given in [27], we extend it to an FF model:

$$\begin{cases} {}^{\mathrm{FFP}}\mathbf{D}_{0,t}^{\xi,\sigma}\mathcal{P}_s(t) = \Lambda - \Psi\Gamma(t)\mathcal{P}_s(t) - \zeta\mathcal{P}_s(t), \\ {}^{\mathrm{FFP}}\mathbf{D}_{0,t}^{\xi,\sigma}\mathcal{P}_i(t) = -(\zeta + \Psi_l + \delta_l)\mathcal{P}_i(t) + \delta_c\mathcal{P}_c(t) + \tau_{AIDS}\mathcal{P}_a(t) + \Psi\Gamma(t)\mathcal{P}_s(t), \\ {}^{\mathrm{FFP}}\mathbf{D}_{0,t}^{\xi,\sigma}\mathcal{P}_c(t) = -(\zeta + \delta_c)\mathcal{P}_c(t) + \Psi_l\mathcal{P}_i(t), \\ {}^{\mathrm{FFP}}\mathbf{D}_{0,t}^{\xi,\sigma}\mathcal{P}_a(t) = -(\zeta + \zeta_{AIDS} + \tau_{AIDS})\mathcal{P}_a(t) + \delta_l\mathcal{P}_i(t), \end{cases} \tag{13}$$

subject to initial conditions

$$\mathcal{P}_s(0) = \mathcal{S}_0 > 0, \qquad \mathcal{P}_i(0) = \mathcal{I}_0 \geq 0, \qquad \mathcal{P}_c(0) = \mathcal{C}_0 \geq 0, \qquad \mathcal{P}_a(0) = \mathcal{A}_0 \geq 0,$$

where ${}^{\mathrm{FFP}}\mathbf{D}_{0,t}^{\xi,\sigma}$ is the FF derivative operator, mentioned in Definition 2.1 for $(\xi,\sigma) \in (0,1] \times (0,1]$.

It should be noticed that the values of the model parameters in system (13) depend on the populations under study and that they will be different for various case studies. The values of these parameters are estimated by using the statistics of cumulative cases in the classes $\mathcal{P}_s$, $\mathcal{P}_i$, $\mathcal{P}_c$, and $\mathcal{P}_a$ for the area of study in a certain period.

4 Existence of solutions

In all biological models, knowing the existence of solutions is very important. In this section, some new FP theorems are employed for this purpose. It should be noted that the FP theorems that we will use in this section are more general and comprehensive than other results in the literature.

For $S = C(\mathbb{J}, \mathbb{R})$, let $\Pi = S \times S \times S \times S$ be the Banach space equipped with the norm

$$\|X(\mathrm{t})\|_{\Pi} = \max\left\{\sum_{k=s,i,a,c} \left|\mathcal{P}_k(\mathrm{t})\right| : \mathrm{t} \in \mathbb{J}\right\},$$

where $X(\mathrm{t}) = (\mathcal{P}_s(\mathrm{t}), \mathcal{P}_i(\mathrm{t}), \mathcal{P}_c(\mathrm{t}), \mathcal{P}_a(\mathrm{t}))$. Also, for the sake of recapitulation of system (13), we put

$$\begin{cases} \mathcal{F}_s(\mathrm{t}, X(\mathrm{t})) = \Lambda - \Psi\Gamma(\mathrm{t})\mathcal{P}_s(\mathrm{t}) - \zeta\mathcal{P}_s(\mathrm{t}), \\ \mathcal{F}_i(\mathrm{t}, X(\mathrm{t})) = -(\zeta + \Psi_I + \delta_I)\mathcal{P}_i(\mathrm{t}) + \delta_c\mathcal{P}_c(\mathrm{t}) + \tau_{AIDS}\mathcal{P}_a(\mathrm{t}) + \Psi\Gamma(\mathrm{t})\mathcal{P}_s(\mathrm{t}), \\ \mathcal{F}_c(\mathrm{t}, X(\mathrm{t})) = -(\zeta + \delta_c)\mathcal{P}_c(\mathrm{t}) + \Psi_I\mathcal{P}_i(\mathrm{t}), \\ \mathcal{F}_a(\mathrm{t}, X(\mathrm{t})) = -(\zeta + \zeta_{AIDS} + \tau_{AIDS})\mathcal{P}_a(\mathrm{t}) + \delta_I\mathcal{P}_i(\mathrm{t}). \end{cases} \tag{14}$$

Due to the differentiability of the integral, the system (13) can be reformulated as

$$\begin{cases} {}^{\mathbf{RL}}\mathbf{D}^{\xi}_{0,\mathrm{t}}\{\mathcal{P}_s(\mathrm{t})\} = \sigma\,\mathrm{t}^{\sigma-1}\mathcal{F}_s(\mathrm{t}, X(\mathrm{t})), \\ {}^{\mathbf{RL}}\mathbf{D}^{\xi}_{0,\mathrm{t}}\{\mathcal{P}_i(\mathrm{t})\} = \sigma\,\mathrm{t}^{\sigma-1}\mathcal{F}_i(\mathrm{t}, X(\mathrm{t})), \\ {}^{\mathbf{RL}}\mathbf{D}^{\xi}_{0,\mathrm{t}}\{\mathcal{P}_c(\mathrm{t})\} = \sigma\,\mathrm{t}^{\sigma-1}\mathcal{F}_c(\mathrm{t}, X(\mathrm{t})), \\ {}^{\mathbf{RL}}\mathbf{D}^{\xi}_{0,\mathrm{t}}\{\mathcal{P}_a(\mathrm{t})\} = \sigma\,\mathrm{t}^{\sigma-1}\mathcal{F}_a(\mathrm{t}, X(\mathrm{t})), \end{cases} \tag{15}$$

where the symbol RL denotes the Riemann–Liouville fractional order derivative operator. According to system (15), the extended model may be written as

$$\begin{cases} {}^{\mathbf{RL}}\mathbf{D}^{\xi}_{0,\mathrm{t}}\{X(\mathrm{t})\} = \sigma\,\mathrm{t}^{\sigma-1}\mathbf{F}(\mathrm{t}, X(\mathrm{t})), \quad 0 < \xi,\ \sigma \leq 1, \\ X(0) = X_0, \end{cases} \tag{16}$$

and $\mathbf{F}(\mathrm{t}, X(\mathrm{t}))$ is the right hand side column vector, defined as

$$\mathbf{F}\big(\mathrm{t}, \Upsilon(\mathrm{t})\big) = \big[\mathcal{F}_k\big(\mathrm{t}, \mathcal{P}_s(\mathrm{t}), \mathcal{P}_i(\mathrm{t}), \mathcal{P}_c(\mathrm{t}), \mathcal{P}_a(\mathrm{t})\big)\big]_{k=s,i,c,a}. \tag{17}$$

By operating ${}^{\mathrm{FFP}}\mathfrak{I}^{\xi,\sigma}_{0,\mathrm{t}}$ (as Definition 2.2) on both sides of system (16),

$$X(\mathrm{t}) = X(0) + \frac{\sigma}{\Gamma(\xi)}\int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\mathbf{F}\big(\eta, X(\eta)\big)\,\mathrm{d}\eta. \tag{18}$$

In more detail, we have

$$\begin{cases} \mathcal{P}_s(\mathrm{t}) = \mathcal{S}_0 + \frac{\sigma}{\Gamma(\xi)} \int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\mathcal{F}_s(\eta, X(\eta))\,\mathrm{d}\eta, \\ \mathcal{P}_i(\mathrm{t}) = \mathcal{I}_0 + \frac{\sigma}{\Gamma(\xi)} \int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\mathcal{F}_i(\eta, X(\eta))\,\mathrm{d}\eta, \\ \mathcal{P}_c(\mathrm{t}) = \mathcal{C}_0 + \frac{\sigma}{\Gamma(\xi)} \int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\mathcal{F}_c(\eta, X(\eta))\,\mathrm{d}\eta, \\ \mathcal{P}_a(\mathrm{t}) = \mathcal{I}_0 + \frac{\sigma}{\Gamma(\xi)} \int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\mathcal{F}_a(\eta, X(\eta))\,\mathrm{d}\eta. \end{cases} \tag{19}$$

Theorem 4.1 [28] *For a complete metric space (Y,d), if $T : Y \to Y$ is a generalized α-ψ-Geraghty contractive mapping with the following conditions*:

(*i*) *T is α-admissible*,

(*ii*) *there exists $y_0 \in Y$ with $\alpha(y_0, Ty_0) \geq 1$, and*

(*iii*) *Y is α-regular*,

then T has an FP.

Theorem 4.2 *Let $\mathcal{E} : \mathbb{R} \times \mathbb{R} \to \mathbb{R}$ and $\mathbf{F} : \mathbb{J} \times \Pi \to \Pi$ be continuous and non-decreasing functions, respectively, and let $\psi :\in \Psi$ and $\beta \in \mathcal{F}$. If conditions $(\mathfrak{C}_1)$–$(\mathfrak{C}_3)$ hold, then the FF system* (16) *has a solution and consequently, the FF model* (13) *has a solution.*

$(\mathfrak{C}_1)$ *For $X_1, X_2 \in \Pi$ and $\mathrm{t} \in \mathbb{J}$, we have*

$$\left|\mathbf{F}\big(\mathrm{t}, X_1(\mathrm{t})\big) - \mathbf{F}\big(\mathrm{t}, X_2(\mathrm{t})\big)\right| \leq \delta\psi\big(\left|X_1(\mathrm{t}) - X_2(\mathrm{t})\right|\big),$$

where $\delta = \frac{\beta(\psi(|X_1(\mathrm{t})-X_2(\mathrm{t}))|)\Gamma(\sigma+\xi)}{\sigma T^{\sigma+\xi-1}\Gamma(\sigma)}$, and $\mathcal{E}(X_1(\mathrm{t}), X_2(\mathrm{t})) \geq 0$.

$(\mathfrak{C}_2)$ *For all $\mathrm{t} \in \mathbb{J}$, there exists an X_0 belonging to Π such that*

$$\mathcal{E}\big(X_0(\mathrm{t}), G\big(X_0(\mathrm{t})\big)\big) \geq 0,$$

and if $\mathcal{E}(X_1(\mathrm{t}), X_2(\mathrm{t})) \geq 0$, then

$$\mathcal{E}\big(G\big(X_1(\mathrm{t})\big), G\big(X_2(\mathrm{t})\big)\big) \geq 0.$$

$(\mathfrak{C}_3)$ *Let $\{X_n\}_{n\geq 1}$ be a convergent sequence in Π ($\lim_{n\to\infty} X_n = X$). Then for*

$$\mathcal{E}\big(X_n(\mathrm{t}), X_{n+1}(\mathrm{t})\big) \geq 0, \quad \mathrm{t} \in \mathbb{J},$$

we get

$$\mathcal{E}\big(X_n(\mathrm{t}), X(\mathrm{t})\big) \geq 0.$$

Proof Consider that for $X_k \in \Pi\,(k = 1, 2)$, we have $\mathcal{E}(X_1(\mathrm{t}), X_2(\mathrm{t})) \geq 0$. By the definition of the Euler beta function, we deduce

$$\begin{aligned} \left|G\big(X_1(\mathrm{t})\big) - G\big(X_2(\mathrm{t})\big)\right| &\leq \frac{\sigma}{\Gamma(\xi)} \int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\left|\mathbf{F}\big(\eta, X_1(\eta)\big) - \mathbf{F}\big(\eta, X_2(\eta)\big)\right| \mathrm{d}\eta \\ &\leq \frac{\sigma\delta}{\Gamma(\xi)} \int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\psi\big(\left|X_1(\eta) - X_2(\eta)\right|\big)\,\mathrm{d}\eta \end{aligned}$$

$$\begin{aligned}
&\leq \frac{\sigma\delta\psi(\|X_1 - X_2\|_\Pi)}{\Gamma(\xi)} \int_0^{\mathfrak{t}} \eta^{\sigma-1}(\mathfrak{t}-\eta)^{\xi-1}\,\mathrm{d}\eta \\
&\leq \frac{\sigma\delta T^{\sigma+\xi-1}\mathcal{B}(\sigma,\xi)}{\Gamma(\xi)} \psi\big(\|X_1 - X_2\|_\Pi\big) \\
&\leq \beta(\psi\big(\|X_1 - X_2\|_\Pi\big)\psi\big(\|X_1 - X_2\|_\Pi\big). \qquad (20)
\end{aligned}$$

Since $\psi(\mathfrak{t}) \leq \mathfrak{t}$, we get

$$\psi\big(\big\|G(X_1) - G(X_2)\big\|_\Pi\big) \leq \beta(\psi\big(\|X_1 - X_2\|_\Pi\big)\psi\big(\|X_1 - X_2\|_\Pi\big).$$

Define $\phi : \Pi \times \Pi \to [0, +\infty)$ as

$$\phi(X_1, X_2) = \begin{cases} 1 & \mathcal{E}(X_1(\mathfrak{t}), X_2(\mathfrak{t})) \geq 0, \\ 0 & \text{otherwise.} \end{cases} \qquad (21)$$

Then for $X_1, X_2 \in \Pi$, we have

$$\phi(X_1, X_2)\psi\big(\mathbf{d}\big(G(X_1), G(X_2)\big)\big) \leq \beta\big(\psi\big(\mathbf{d}(X_1, X_2)\big)\big)\psi\big(\mathbf{d}(X_1, X_2)\big).$$

Therefore, G is a generalized ϕ-ψ-Geraghty contraction. Now, we prove that G is triangular α-admissible. Let $X_1, X_2, X_3 \in \Pi$ with $\phi(X_1, X_2) \geq 1$. By the property of ϕ, we get

$$\mathcal{E}\big(X_1(\mathfrak{t}), X_2(\mathfrak{t})\big) \geq 0.$$

From hypothesis $(\mathfrak{C}_2)$, we have

$$\mathcal{E}\big(G\big(X_1(\mathfrak{t})\big), G\big(X_2(\mathfrak{t})\big)\big) \geq 0.$$

On the other hand, from (21) we get

$$\phi\big(G(X_1), G(X_2)\big) \geq 1.$$

Thus, G is ϕ-admissible.

From condition $(\mathfrak{C}_2)$ there exists $X_0 \in \Pi$ such that $\mathcal{E}(X_0(\mathfrak{t}), G(X_0(\mathfrak{t}))) \geq 0$ for each $\mathfrak{t} \in \mathbb{J}$. Clearly, we get $\phi(X_0, G(X_0)) \geq 1$.

Suppose $\{X_n\}_{n\geq 1}$ is a sequence defined in Π, $X_n \to X$ and $\phi(X_n, X_{n+1}) \geq 1$ for $n \geq 1$. By the definition of ϕ,

$$\mathcal{E}\big(X_n(\mathfrak{t}), X_{n+1}(\mathfrak{t})\big) \geq 0,$$

and from $(\mathfrak{C}_3)$, we obtain

$$\mathcal{E}\big(X_n(\mathfrak{t}), X(\mathfrak{t})\big) \geq 0.$$

Thus, $\phi(X_n, X) \geq 1$ $(n \geq 1)$, that is, condition (3) of Theorem 4.1 is established. So, all the assumptions of Theorem 4.1 are valid and consequently, G has an FP $X^* \in \Pi$. In other words, $X^* = (\mathcal{P}_s^*, \mathcal{P}_i^*, \mathcal{P}_c^*, \mathcal{P}_a^*)^T$ is a solution of the FF model of HIV/AIDS transmission (13). □

5 Uniqueness result

To study this concept, we examine the Lipschitz condition of the mappings $\mathcal{F}_k$, $k = s, i, c, a$, defined in (14).

Lemma 5.1 *Let $\mathcal{P}_s, \mathcal{P}_i, \mathcal{P}_c, \mathcal{P}_a, \mathcal{P}_s^*, \mathcal{P}_i^*, \mathcal{P}_c^*, \mathcal{P}_a^* \in S = C(\mathbb{J}, \mathbb{R})$ and suppose $(\mathcal{H}_1)$ $\|\mathcal{P}_s\| \leq \gamma_1$, $\|\mathcal{P}_i\| \leq \gamma_2$, $\|\mathcal{P}_c\| \leq \gamma_3$, and $\|\mathcal{P}_a\| \leq \gamma_4$ for $\gamma_1, \gamma_2, \gamma_3, \gamma_4 > 0$.*

Then the functions $\mathcal{F}_s$, $\mathcal{F}_i$, $\mathcal{F}_c$, $\mathcal{F}_a$ defined in (14) satisfy the Lipschitz condition under the following parameters:

$$\begin{aligned} &\lambda_1 = \zeta - \Psi \frac{\gamma_2}{\gamma_1 + \gamma_2 + \gamma_3 + \gamma_4}, \qquad \lambda_2 = \zeta + \Psi_l + \delta_l, \\ &\lambda_3 = \zeta + \delta_c, \qquad \lambda_4 = \zeta + \zeta_{AIDS} + \tau_{AIDS}\gamma_4. \end{aligned} \tag{22}$$

Proof For each $\mathcal{P}_s, \mathcal{P}_s^* \in S = C(\mathbb{J}, \mathbb{R})$, we have

$$\begin{aligned} &\left\| \mathcal{F}_s\big(\mathrm{t}, \mathcal{P}_s(\mathrm{t}), \mathcal{P}_i(\mathrm{t}), \mathcal{P}_c(\mathrm{t}), \mathcal{P}_a(\mathrm{t})\big) - \mathcal{F}_s\big(\mathrm{t}, \mathcal{P}_s^*(\mathrm{t}), \mathcal{P}_i(\mathrm{t}), \mathcal{P}_c(\mathrm{t}), \mathcal{P}_a(\mathrm{t})\big) \right\| \\ &\quad = \left\| -\Psi\Gamma(t)\mathcal{P}_s(\mathrm{t}) - \zeta\mathcal{P}_s(\mathrm{t}) + \Psi\Gamma(t)\mathcal{P}_s^*(\mathrm{t}) + \zeta\mathcal{P}_s^*(\mathrm{t})) \right\| \\ &\quad \leq \left\| \zeta + \Psi\Gamma(t) \right\| \cdot \left\| \mathcal{P}_s^*(\mathrm{t}) - \mathcal{P}_s(\mathrm{t}) \right\| \\ &\quad \leq \left(\zeta + \Psi \frac{\gamma_1}{\gamma_1 + \gamma_2 + \gamma_3 + \gamma_4} \right) \left\| \mathcal{P}_s^*(\mathrm{t}) - \mathcal{P}_s(\mathrm{t}) \right\| \\ &\quad = \lambda_1 \left\| \mathcal{P}_s^*(\mathrm{t}) - \mathcal{P}_s(\mathrm{t}) \right\|. \end{aligned}$$

So $\mathcal{F}_s$ is Lipschitz with respect to $\mathcal{P}_s$ with the Lipschitz constant λ_1. For each $\mathcal{P}_i, \mathcal{P}_i^* \in S = C(\mathbb{J}, \mathbb{R})$, we have

$$\begin{aligned} &\left\| \mathcal{F}_i\big(\mathrm{t}, \mathcal{P}_s(\mathrm{t}), \mathcal{P}_i(\mathrm{t}), \mathcal{P}_c(\mathrm{t}), \mathcal{P}_a(\mathrm{t})\big) - \mathcal{F}_i\big(\mathrm{t}, \mathcal{P}_s(\mathrm{t}), \mathcal{P}_i^*(\mathrm{t}), \mathcal{P}_c(\mathrm{t}), \mathcal{P}_a(\mathrm{t})\big) \right\| \\ &\quad = \left\| -(\zeta + \Psi_l + \delta_l)\mathcal{P}_i(\mathrm{t}) + (\zeta + \Psi_l + \delta_l)\mathcal{P}_i^*(\mathrm{t}) \right\| \\ &\quad \leq (\zeta + \Psi_l + \delta_l) \left\| \mathcal{P}_i^*(\mathrm{t}) - \mathcal{P}_i(\mathrm{t}) \right\| \\ &\quad = \lambda_2 \left\| \mathcal{P}_i(\mathrm{t}) - \mathcal{P}_i^*(\mathrm{t}) \right\|. \end{aligned}$$

Therefore, $\mathcal{F}_i$ is Lipschitz. For each $\mathcal{P}_c, \mathcal{P}_c^* \in S = C(\mathbb{J}, \mathbb{R})$, we have

$$\begin{aligned} &\left\| \mathcal{F}_c\big(\mathrm{t}, \mathcal{P}_s(\mathrm{t}), \mathcal{P}_i(\mathrm{t}), \mathcal{P}_c(\mathrm{t}), \mathcal{P}_a(\mathrm{t})\big) - \mathcal{F}_c\big(\mathrm{t}, \mathcal{P}_s(\mathrm{t}), \mathcal{P}_i(\mathrm{t}), \mathcal{P}_c^*(\mathrm{t}), \mathcal{P}_a(\mathrm{t})\big) \right\| \\ &\quad = \left\| -(\zeta + \delta_c)\mathcal{P}_c(\mathrm{t}) + (\zeta + \delta_c)\mathcal{P}_c^*(\mathrm{t}) \right\| \\ &\quad \leq (\zeta + \delta_c) \left\| \mathcal{P}_c^*(\mathrm{t}) - \mathcal{P}_c(\mathrm{t}) \right\| \\ &\quad = \lambda_3 \left\| \mathcal{P}_c^*(\mathrm{t}) - \mathcal{P}_c(\mathrm{t}) \right\|. \end{aligned}$$

That is, $\mathcal{F}_c$ is Lipschitz. For each $\mathcal{P}_a, \mathcal{P}_a^* \in \mathbb{K} = C(\mathbb{J}, \mathbb{R})$, we have

$$\begin{aligned} &\left\| \mathcal{F}_a\big(\mathrm{t}, \mathcal{P}_s(\mathrm{t}), \mathcal{P}_i(\mathrm{t}), \mathcal{P}_c(\mathrm{t}), \mathcal{P}_a(\mathrm{t})\big) - \mathcal{F}_a\big(\mathrm{t}, \mathcal{P}_s(\mathrm{t}), \mathcal{P}_i(\mathrm{t}), \mathcal{P}_c(\mathrm{t}), \mathcal{P}_a^*(\mathrm{t})\big) \right\| \\ &\quad = \left\| -(\zeta + \zeta_{AIDS} + \tau_{AIDS})\mathcal{P}_a(\mathrm{t}) + (\zeta + \zeta_{AIDS} + \tau_{AIDS})\mathcal{P}_a^*(\mathrm{t}) \right\| \\ &\quad \leq (\zeta + \zeta_{AIDS} + \tau_{AIDS}) \left\| \mathcal{P}_a^*(\mathrm{t}) - \mathcal{P}_a(\mathrm{t}) \right\| \\ &\quad = \lambda_4 \left\| \mathcal{P}_a^*(\mathrm{t}) - \mathcal{P}_a(\mathrm{t}) \right\|. \end{aligned}$$

Similarly, $\mathcal{F}_a$ is Lipschitz. Consequently, the functions $\mathcal{F}_k$, $k = s, i, c, a$, are Lipschitz with respect to the corresponding component with constants λ_k, $k = 1, 2, 3, 4$, respectively. □

Here, we study the uniqueness of solutions to the mentioned model (13).

Theorem 5.2 *Let* $(\mathcal{H}_1)$ *hold. Then the given FF HIV/AIDS transmission model* (13) *has a unique solution if*

$$\frac{\sigma T^{\sigma+\xi-1}\Gamma(\sigma)}{\Gamma(\sigma+\xi)}\lambda_i < 1, \quad i \in \{1, \dots, 4\}. \tag{23}$$

Proof We prove this theorem by contradiction. Suppose that there exist two distinct solutions $X(\mathrm{t})$ and $X^*(\mathrm{t})$ for (13). By (19), we have

$$\begin{cases} \mathcal{P}_s^*(\mathrm{t}) = \mathcal{S}_0 + \frac{\sigma}{\Gamma(\xi)}\int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\mathcal{F}_s(\eta, \mathcal{P}_s^*(\eta), \mathcal{P}_i^*(\eta), \mathcal{P}_c^*(\eta), \mathcal{P}_a^*(\eta))\,\mathrm{d}\eta, \\ \mathcal{P}_i^*(\mathrm{t}) = \mathcal{I}_0 + \frac{\sigma}{\Gamma(\xi)}\int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\mathcal{F}_i(\eta, \mathcal{P}_s^*(\eta), \mathcal{P}_i^*(\eta), \mathcal{P}_c^*(\eta), \mathcal{P}_a^*(\eta))\,\mathrm{d}\eta, \\ \mathcal{P}_c^*(\mathrm{t}) = \mathcal{C}_0 + \frac{\sigma}{\Gamma(\xi)}\int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\mathcal{F}_c(\eta, \mathcal{P}_s^*(\eta), \mathcal{P}_i^*(\eta), \mathcal{P}_c^*(\eta), \mathcal{P}_a^*(\eta))\,\mathrm{d}\eta, \\ \mathcal{P}_a^*(\mathrm{t}) = \mathcal{A}_0 + \frac{\sigma}{\Gamma(\xi)}\int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\mathcal{F}_a(\eta, \mathcal{P}_s^*(\eta), \mathcal{P}_i^*(\eta), \mathcal{P}_c^*(\eta), \mathcal{P}_a^*(\eta))\,\mathrm{d}\eta, \end{cases}$$

so we get

$$\begin{aligned} \left|\mathcal{P}_s(\mathrm{t}) - \mathcal{P}_s^*(\mathrm{t})\right| &\le \frac{\sigma}{\Gamma(\xi)}\int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\left|\mathcal{F}_s\big(\eta, \mathcal{P}_s(\eta), \mathcal{P}_i(\eta), \mathcal{P}_c(\eta), \mathcal{P}_a(\eta)\big)\right. \\ &\quad \left. - \mathcal{F}_s\big(\eta, \mathcal{P}_s^*(\eta), \mathcal{P}_i^*(\eta), \mathcal{P}_c^*(\eta), \mathcal{P}_a^*(\eta)\big)\right|\mathrm{d}\eta \\ &\le \frac{\sigma}{\Gamma(\xi)}\lambda_1\left\|\mathcal{P}_s - \mathcal{P}_s^*\right\|\int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\,\mathrm{d}\eta \\ &\le \frac{\sigma T^{\sigma+\xi-1}\Gamma(\sigma)}{\Gamma(\sigma+\xi)}\lambda_1\left\|\mathcal{P}_s - \mathcal{P}_s^*\right\|. \end{aligned}$$

Hence,

$$\left(1 - \frac{\sigma T^{\sigma+\xi-1}\Gamma(\sigma)}{\Gamma(\sigma+\xi)}\lambda_1\right)\left\|\mathcal{P}_s - \mathcal{P}_s^*\right\| \le 0.$$

By relation (23), we get $\mathcal{P}_s = \mathcal{P}_s^*$. Similarly, we can show that

$$\left(1 - \frac{\sigma T^{\sigma+\xi-1}\Gamma(\sigma)}{\Gamma(\sigma+\xi)}\lambda_2\right)\left\|\mathcal{P}_i - \mathcal{P}_i^*\right\| \le 0, \tag{24}$$

$$\left(1 - \frac{\sigma T^{\sigma+\xi-1}\Gamma(\sigma)}{\Gamma(\sigma+\xi)}\lambda_3\right)\left\|\mathcal{P}_c - \mathcal{P}_c^*\right\| \le 0, \tag{25}$$

$$\left(1 - \frac{\sigma T^{\sigma+\xi-1}\Gamma(\sigma)}{\Gamma(\sigma+\xi)}\lambda_4\right)\left\|\mathcal{P}_a - \mathcal{P}_a^*\right\| \le 0. \tag{26}$$

Therefore, $\mathcal{P}_i = \mathcal{P}_i^*$, $\mathcal{P}_c = \mathcal{P}_c^*$, and $\mathcal{P}_a = \mathcal{P}_a^*$. Consequently,

$$(\mathcal{P}_s, \mathcal{P}_i, \mathcal{P}_c, \mathcal{P}_a) = \big(\mathcal{P}_s^*, \mathcal{P}_i^*, \mathcal{P}_c^*, \mathcal{P}_a^*\big).$$

So the proof is completed. □

6 Stability analysis

In this section, we analyze the stability of the model (13) in the sense of Ulam–Hyers and its extended version for solutions of the model of HIV/AIDS transmission (13).

Definition 6.1 For $\varepsilon_i > 0$, $i = 1, 2, 3, 4$, and for $X^* \in \Pi$, where

$$\begin{cases} |{}^{\mathbf{FFP}}\mathbf{D}_{0,\mathrm{t}}^{\xi,\sigma}\mathcal{P}_s^*(\mathrm{t}) - \mathcal{F}_s(\mathrm{t}, X^*\mathrm{t})| < \varepsilon_1, \\ |{}^{\mathbf{FFP}}\mathbf{D}_{0,\mathrm{t}}^{\xi,\sigma}\mathcal{P}_i^*(\mathrm{t}) - \mathcal{F}_i(\mathrm{t}, X^*\mathrm{t})| < \varepsilon_2, \\ |{}^{\mathbf{FFP}}\mathbf{D}_{0,\mathrm{t}}^{\xi,\sigma}\mathcal{P}_c^*(\mathrm{t}) - \mathcal{F}_c(\mathrm{t}, X^*\mathrm{t})| < \varepsilon_3, \\ |{}^{\mathbf{FFP}}\mathbf{D}_{0,\mathrm{t}}^{\xi,\sigma}\mathcal{P}_a^*(\mathrm{t}) - \mathcal{F}_a(\mathrm{t}, X^*\mathrm{t})| < \varepsilon_4, \end{cases}$$

there exist constants $\kappa_j \in \mathbb{R}^+$, $j = s, i, c, a$, and a solution, $X(\mathrm{t}) \in \Pi$, for (13) such that for all $\mathrm{t} \in \mathbb{J}$

$$\begin{cases} |\mathcal{P}_s^*(\mathrm{t}) - \mathcal{P}_s(\mathrm{t})| \leq \kappa_s\varepsilon_1, \\ |\mathcal{P}_i^*(\mathrm{t}) - \mathcal{P}_i(\mathrm{t})| \leq \kappa_i\varepsilon_2, \\ |\mathcal{P}_c^*(\mathrm{t}) - \mathcal{P}_c(\mathrm{t})| \leq \kappa_c\varepsilon_3, \\ |\mathcal{P}_a^*(\mathrm{t}) - \mathcal{P}_a(\mathrm{t})| \leq \kappa_a\varepsilon_4. \end{cases} \tag{27}$$

Then the model (13) is Ulam–Hyers stable.

Definition 6.2 The model (13) is extended Ulam–Hyers stable if there exist $\kappa_j \in C(\mathbb{R}^+, \mathbb{R}^+)$, $j = s, i, c, a$, with $\kappa_j(0) = 0$ and a solution, $X \in \Pi$, for model (13) such that for all $\varepsilon_i > 0$ and for all $X^* \in \Pi$ satisfying the relations (27), we have

$$\begin{cases} |\mathcal{P}_s^*(\mathrm{t}) - \mathcal{P}_s(\mathrm{t})| \leq \kappa_s(\varepsilon_1), \\ |\mathcal{P}_i^*(\mathrm{t}) - \mathcal{P}_i(\mathrm{t})| \leq \kappa_i(\varepsilon_2), \\ |\mathcal{P}_c^*(\mathrm{t}) - \mathcal{P}_c(\mathrm{t})| \leq \kappa_c(\varepsilon_3), \\ |\mathcal{P}_a^*(\mathrm{t}) - \mathcal{P}_a(\mathrm{t})| \leq \kappa_a(\varepsilon_4), \end{cases} \tag{28}$$

for all $\mathrm{t} \in \mathbb{J}$.

Remark 1 Note that $X^* \in \Pi$ is said to be a solution of the relations of Definition 6.1 if and only if there exist $\theta_s, \theta_i, \theta_c, \theta_a \in C([0, T], \mathbb{R})$ with the following properties:

(*i*) $|\theta_j(\mathrm{t})| < \varepsilon_i$, $j = s, i, c, a$, $i = 1, \ldots, 4$;

(*ii*) the following inequalities hold:

$$\begin{cases} {}^{\mathbf{FFP}}\mathbf{D}_{0,\mathrm{t}}^{\xi,\sigma}\mathcal{P}_s^*(\mathrm{t}) = \mathcal{F}_s(\mathrm{t}, X^*(\mathrm{t})) + \theta_s(\mathrm{t}), \\ {}^{\mathbf{FFP}}\mathbf{D}_{0,\mathrm{t}}^{\xi,\sigma}\mathcal{P}_s^*(\mathrm{t}) = \mathcal{F}_i(\mathrm{t}, X^*(\mathrm{t})) + \theta_i(\mathrm{t}), \\ {}^{\mathbf{FFP}}\mathbf{D}_{0,\mathrm{t}}^{\xi,\sigma}\mathcal{P}_s^*(\mathrm{t}) = \mathcal{F}_c(\mathrm{t}, X^*(\mathrm{t})) + \theta_c(\mathrm{t}), \\ {}^{\mathbf{FFP}}\mathbf{D}_{0,\mathrm{t}}^{\xi,\sigma}\mathcal{P}_s^*(\mathrm{t}) = \mathcal{F}_a(\mathrm{t}, X^*(\mathrm{t})) + \theta_a(\mathrm{t}). \end{cases} \tag{29}$$

Theorem 6.3 *Considering the condition* $(\mathcal{H}_1)$, *the given model of HIV/AIDS transmission* (13) *is Ulam–Hyers stable on* $\mathbb{J} = [0, T]$, *and it is also extended Ulam–Hyers stable*

if

$$\frac{\sigma T^{\sigma+\xi-1}\Gamma(\sigma)}{\Gamma(\sigma+\xi)}\lambda_k < 1, \quad k = 1,\dots,4,$$

where λ_k *are defined by* (22).

Proof Let $\varepsilon_1 > 0$ and $\mathcal{P}_s^* \in S$ be arbitrary so that

$$\left|{}^{\mathbf{FFP}}\mathbf{D}_{0,\mathrm{t}}^{\xi,\sigma}\mathcal{P}_s^*(\mathrm{t}) - \mathcal{F}_s\big(\mathrm{t},\mathcal{P}_s^*(\mathrm{t}),\mathcal{P}_i^*(\mathrm{t}),\mathcal{P}_c^*(\mathrm{t}),\mathcal{P}_a^*(\mathrm{t})\big)\right| < \varepsilon_1.$$

Then, in view of Remark 1, we can find a function $\theta_s(\mathrm{t})$ satisfying

$${}^{\mathbf{FFP}}\mathbf{D}_{0,(\mathrm{t})}^{\xi,\sigma}\mathcal{P}_s^*(\mathrm{t}) = \mathcal{F}_s\big(\mathrm{t},\mathcal{P}_s^*(\mathrm{t}),\mathcal{P}_i^*(\mathrm{t}),\mathcal{P}_c^*(\mathrm{t}),\mathcal{P}_a^*(\mathrm{t})\big) + \theta_s(\mathrm{t}),$$

with $|\theta_s(\mathrm{t})| \le \varepsilon_1$. It follows that

$$\begin{aligned}\mathcal{P}_s^*(\mathrm{t}) = {}& \mathcal{S}_0 + \frac{\sigma}{\Gamma(\xi)}\int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\mathcal{F}_s\big(\eta,\mathcal{P}_s^*(\eta),\mathcal{P}_i^*(\eta),\mathcal{P}_c^*(\eta),\mathcal{P}_a^*(\eta)\big)\,\mathrm{d}\eta \\ &+ \frac{\sigma}{\Gamma(\xi)}\int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\theta_s(\eta)\,\mathrm{d}\eta.\end{aligned}$$

By using Theorem 5.2, let $\mathcal{P}_s \in \mathbb{K}$ be the unique solution of the given FF model of HIV/AIDS transmission (13). Then $\mathcal{P}_s(\mathrm{t})$ is given by

$$\mathcal{P}_s(\mathrm{t}) = \mathcal{S}_0 + \frac{\sigma}{\Gamma(\xi)}\int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\mathcal{F}_s\big(\eta,\mathcal{P}_s(\eta),\mathcal{P}_i(\eta),\mathcal{P}_c(\eta),\mathcal{P}_a(\eta)\big)\,\mathrm{d}\eta.$$

Then

$$\begin{aligned}\left|\mathcal{P}_s^*(\mathrm{t}) - \mathcal{P}_s(\mathrm{t})\right| \le {}& \frac{\sigma}{\Gamma(\xi)}\int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\big|\mathcal{F}_s\big(\eta,\mathcal{P}_s^*(\eta),\mathcal{P}_i^*(\eta),\mathcal{P}_c^*(\eta),\mathcal{P}_a^*(\eta)\big) \\ &- \mathcal{F}_s\big(\eta,\mathcal{P}_s(\eta),\mathcal{P}_i(\eta),\mathcal{P}_c(\eta),\mathcal{P}_a(\eta)\big)\big|,\mathrm{d}\eta \\ &+ \frac{\sigma}{\Gamma(\xi)}\int_0^{\mathrm{t}} \eta^{\sigma-1}(\mathrm{t}-\eta)^{\xi-1}\left|\theta_s(\eta)\right|\mathrm{d}\eta \\ \le {}& \frac{\sigma T^{\sigma+\xi-1}\Gamma(\sigma)}{\Gamma(\sigma+\xi)}\lambda_1\left\|\mathcal{P}_s^* - \mathcal{P}_s\right\| + \frac{\sigma T^{\sigma+\xi-1}\Gamma(\sigma)}{\Gamma(\sigma+\xi)}\varepsilon_1.\end{aligned}$$

Hence, we get

$$\left\|\mathcal{P}_s^* - \mathcal{P}_s\right\| \le \frac{\sigma T^{\sigma+\xi-1}\Gamma(\sigma)\varepsilon_1}{\Gamma(\sigma+\xi) - \sigma T^{\sigma+\xi-1}\Gamma(\sigma)\lambda_1}.$$

By putting $\kappa_s = \frac{\sigma T^{\sigma+\xi-1}\Gamma(\sigma)}{\Gamma(\sigma+\xi)-\sigma T^{\sigma+\xi-1}\Gamma(\sigma)\lambda_1}$, we obtain $\|\mathcal{P}_s^* - \mathcal{P}_s\| \le \kappa_s\varepsilon_1$. In the above way, we get

$$\left\|\mathcal{P}_i^* - \mathcal{P}_i\right\| \le \kappa_i\varepsilon_2, \qquad \left\|\mathcal{P}_c^* - \mathcal{P}_c\right\| \le \kappa_c\varepsilon_3, \qquad \left\|\mathcal{P}_a^* - \mathcal{P}_a\right\| \le \kappa_a\varepsilon_4, \tag{30}$$

where

$$\kappa_j = \frac{\sigma T^{\sigma+\xi-1}\Gamma(\sigma)}{\Gamma(\sigma+\xi) - \sigma T^{\sigma+\xi-1}\Gamma(\sigma)\lambda_j}, \quad j = i, c, a.$$

Thus, the Ulam–Hyers stability of the HIV/AIDS transmission model (13) is established. Also, by assuming

$$\kappa_j(\varepsilon_i) = \frac{\sigma T^{\sigma+\xi-1}\Gamma(\sigma)\varepsilon_i}{\Gamma(\sigma+\xi) - \sigma T^{\sigma+\xi-1}\Gamma(\sigma)\lambda_j}, \quad j = i, c, a, i = 1, 2, 3, 4,$$

with $\kappa_j(0) = 0$, the extended Ulam–Hyers stability of (13) is established. □

7 Numerical simulation

Here, the approximated solutions of system (12) are investigated by using SGPs via the spectral collocation method. First, by applying the FF integral operator of order ξ, σ on both sides of system (13), we get

$$\begin{cases} \mathcal{P}_s(\mathrm{t}) = S(0) + \frac{\sigma}{\Gamma(\xi)} \int_0^t \eta^{\sigma-1}(t-\eta)^{\xi-1}\mathcal{F}_s(\eta, \Upsilon(\eta))\, d\eta, \\ \mathcal{P}_i(\mathrm{t}) = I(0) + \frac{\sigma}{\Gamma(\xi)} \int_0^t \eta^{\sigma-1}(t-\eta)^{\xi-1}\mathcal{F}_i(\eta, \Upsilon(\eta))\, d\eta, \\ \mathcal{P}_c(\mathrm{t}) = C(0) + \frac{\sigma}{\Gamma(\xi)} \int_0^t \eta^{\sigma-1}(t-\eta)^{\xi-1}\mathcal{F}_c(\eta, \Upsilon(\eta))\, d\eta, \\ \mathcal{P}_a(\mathrm{t}) = A(0) + \frac{\sigma}{\Gamma(\xi)} \int_0^t \eta^{\sigma-1}(t-\eta)^{\xi-1}\mathcal{F}_a(\eta, \Upsilon(\eta))\, d\eta. \end{cases} \tag{31}$$

In order to solve the current system, the spectral collocation method is utilized. First, the functions $\mathcal{F}_j$, $j = s, i, c, a$, are expressed in SGP terms as

$$\mathcal{F}_j(\mathrm{t}) \simeq \mathcal{F}_j^N(\mathrm{t}) = \sum_{m=1}^{N} f_{j,m}\mathcal{G}_{m,\beta}(\mathrm{t}). \tag{32}$$

Considering (32), the integral terms of system (31) are obtained as

$$\int_0^t \eta^{\sigma-1}(t-\eta)^{\xi-1}\mathcal{F}_j\big(\eta, \mathcal{X}(\eta)\big)\, d\eta = \sum_{m=1}^{N} f_{j,m}\mathcal{Q}_{m,\beta}(\mathrm{t}), \tag{33}$$

where $\mathcal{Q}_{m,\beta}(\mathrm{t}) = \int_0^t \eta^{\sigma-1}(t-\eta)^{\xi-1}\mathcal{G}_{m,\beta}(\eta)\, d\eta$. Now, we approximate the unknowns of the system (31) by SGPs as

$$\begin{aligned} &\mathcal{P}_s(\mathrm{t}) = \mathcal{S}_N^T\mathbf{G}_{N,\beta}(\mathrm{t}), \qquad \mathcal{P}_i(\mathrm{t}) = \mathcal{I}_N^T\mathbf{G}_{N,\beta}(\mathrm{t}), \\ &\mathcal{P}_c(\mathrm{t}) = \mathcal{C}_N^T\mathbf{G}_{N,\beta}(\mathrm{t}), \qquad \mathcal{P}_a(\mathrm{t}) = \mathcal{A}_N^T\mathbf{G}_{N,\beta}(\mathrm{t}), \end{aligned} \tag{34}$$

and $\mathcal{S}_N = [s_j]_N$, $\mathcal{I}_N = [I_j]_N$, $\mathcal{C}_N = [c_j]_N$, and $\mathcal{A}_N = [a_j]_N$ are defined similar to (11). By substituting (33)–(34) in (31), we get

$$\begin{cases} \mathcal{S}_N^T\mathbf{G}_{N,\beta}(\mathrm{t}) = S(0) + \mathcal{F}_{s,N}^T\mathbf{Q}_{N,\beta}(\mathrm{t}), \\ \mathcal{I}_N^T\mathbf{G}_{N,\beta}(\mathrm{t}) = I(0) + \mathcal{F}_{i,N}^T\mathbf{Q}_{N,\beta}(\mathrm{t}), \\ \mathcal{C}_N^T\mathbf{G}_{N,\beta}(\mathrm{t}) = C(0) + \mathcal{F}_{c,N}^T\mathbf{Q}_{N,\beta}(\mathrm{t}), \\ \mathcal{A}_N^T\mathbf{G}_{N,\beta}(\mathrm{t}) = A(0) + \mathcal{F}_{a,N}^T\mathbf{Q}_{N,\beta}(\mathrm{t}), \end{cases} \tag{35}$$

where

$$\mathcal{F}_{j,N} = \frac{\sigma}{\Gamma(\xi)}(f_{j,1},\dots,f_{j,N})^T, \qquad \mathbf{Q}_{N,\beta}(\mathrm{t}) = \big(\mathcal{Q}_{1,\beta}(\mathrm{t}),\dots,\mathcal{Q}_{N,\beta}(\mathrm{t})\big)^T.$$

So, we have an algebraic system with $8 \times N$ unknowns. To solve this system, we define the residual functions for the equations in (35) as

$$\begin{cases} \mathcal{R}_{s,N}(t) = \mathcal{S}_N^T \mathbf{G}_{N,\beta}(\mathrm{t}) - \mathcal{F}_{s,N}^T \mathbf{Q}_{N,\beta}(\mathrm{t}) - S(0), \\ \mathcal{R}_{i,N}(t) = \mathcal{I}_N^T \mathbf{G}_{N,\beta}(\mathrm{t}) - \mathcal{F}_{i,N}^T \mathbf{Q}_{N,\beta}(\mathrm{t}) - I(0), \\ \mathcal{R}_{c,N}(t) = \mathcal{C}_N^T \mathbf{G}_{N,\beta}(\mathrm{t}) - \mathcal{F}_{c,N}^T \mathbf{Q}_{N,\beta}(\mathrm{t}) - C(0), \\ \mathcal{R}_{a,N}(t) = \mathcal{A}_N^T \mathbf{G}_{N,\beta}(\mathrm{t}) - \mathcal{F}_{a,N}^T \mathbf{Q}_{N,\beta}(\mathrm{t}) - A(0), \end{cases} \tag{36}$$

and we set $\mathcal{R}_{\cdot,N}(t_k) = 0$ in the collocation points $t_k = \frac{k}{N}T$, $k = 1,\dots,N$. On the other hand, by putting $\mathcal{F}_j(t_k) = \mathcal{F}_{j,N}^T \mathbf{G}_{N,\beta}(\mathrm{t}_k)$, we have

$$\begin{cases} \mathcal{F}_{s,N}^T \mathbf{G}_{N,\beta}(\mathrm{t}_k) = \Lambda - (\Psi \frac{\mathcal{I}_N^T \mathbf{G}_{N,\beta}(\mathrm{t}_k)}{\mathcal{N}_N^T \mathbf{G}_{N,\beta}(\mathrm{t}_k)} - \zeta)\mathcal{S}_N^T \mathbf{G}_{N,\beta}(\mathrm{t}_k), \\ \mathcal{F}_{i,N}^T \mathbf{G}_{N,\beta}(\mathrm{t}_k) = -((\zeta + \Psi_l + \delta_l)\mathcal{I}_N^T + \delta_c \mathcal{C}_N^T + \tau_{AIDS}\mathcal{A}_N^T \\ \qquad\qquad + \Psi \frac{\mathcal{I}_N^T \mathbf{G}_{N,\beta}(\mathrm{t}_k)}{\mathcal{N}_N^T \mathbf{G}_{N,\beta}(\mathrm{t}_k)} \mathcal{S}_N^T)\mathbf{G}_{N,\beta}(\mathrm{t}_k), \\ \mathcal{F}_{c,N}^T \mathbf{G}_{N,\beta}(\mathrm{t}_k) = (-(\zeta + \delta_c)\mathcal{C}_N^T + \Psi_l \mathcal{I}_N^T)\mathbf{G}_{N,\beta}(\mathrm{t}_k), \\ \mathcal{F}_{a,N}^T \mathbf{G}_{N,\beta}(\mathrm{t}_k) = (-(\zeta + \zeta_{AIDS} + \tau_{Aids})\mathcal{A}_N^T + \delta_l \mathcal{I}_N^T)\mathbf{G}_{N,\beta}(\mathrm{t}_k). \end{cases}$$

Therefore, by combining the obtained equations from the collocated residual function and the equations in (10), we get a nonlinear algebraic system of $8 \times N$ equations and $8 \times N$ unknowns. The iterative Newton method is applied to solve it, where the initial values for the unknowns are $s_1 = \mathcal{S}_0$, $l_1 = \mathcal{I}_0$, $c_1 = \mathcal{C}_0$, $a_1 = \mathcal{A}_0$, and

$$\begin{cases} a_{1,1} = \Lambda - \Psi \frac{l_1}{n_1} s_1 + \zeta s_1, \\ a_{2,1} = -(\zeta + \Psi_l + \delta_l)l_1 + \delta_c c_1 + \tau_{AIDS} a_1 + \Psi \frac{l_1}{n_1} s_1, \\ a_{3,1} = -(\zeta + \delta_c)c_1 + \Psi_l l_1, \\ a_{4,1} = -(\zeta + \zeta_{AIDS} + \tau_{AIDS})a_1 + \delta_l l_1, \end{cases}$$

where $n_1 = s_1 + l_1 + c_1 + a_1$ and for $k = 2,\dots,N$, $j = 1,\dots,4$ we set $s_k = l_k = c_k = a_k = a_{j,k} = 0$.

8 Simulations

In this section, we use the procedure introduced in the previous section to numerically solve the FF system (13). To achieve results with high accuracy, the real values of parameters involved in model (12) and also the initial conditions $S(0)$, $I(0)$, $C(0)$, and $A(0)$ are required. For this purpose, we utilize the reported data from the World Health Organization (WHO) and references [27, 29] for the Cape Verde Islands in 1987–2014, which are tabulated in Table 1. Based on these parameters and initial values, the dynamics of the model (13) and the behaviors of the state populations $\mathcal{P}_s$, $\mathcal{P}_i$, $\mathcal{P}_c$, and $\mathcal{P}_a$ are shown in the figures. To determine the effects of the fractal and fractional orders on the dynamics of the state functions, we used various values for ξ and σ. The approximated solutions

Table 1 Baseline values of parameters and initial values of model (13)

Parameter	Value	Initial condition	Value
Λ	10,724	$S(0)$	338,923
ψ	0.866		
ψ_I	1	$I(0)$	61
δ_I	0.1		
δ_c	0.09	$C(0)$	0
ξ	0.014		
ξ_H	1	$A(0)$	0
τ_H	0.33		

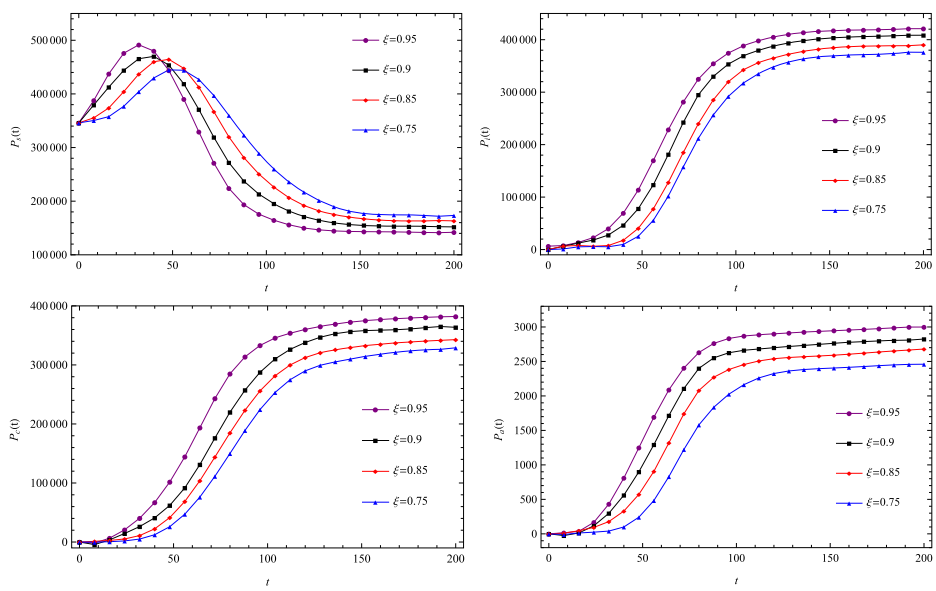

Figure 2 Dynamics of model (13) for $\sigma = 0.9$ and $\xi = 0.75$, 0.85, 0.9, and 0.95

were investigated in the space $S_5^{1.5}$ and the Newton approach was used for six iterations. All results were calculated using the same desktop, an Asus DESKTOP-M0F5LBS, with an Intel(R) Core(TM) i7-6700HQ CPU@2.60 GHz, with 16 GB memory.

8.1 Effects of the fractional derivative order

To examine the effects of the fractional order on the approximated state functions, we put $\sigma = 0.9$ (fixed) and examined some values for ξ. The numerical results are reported in Fig. 2. As we can see, for the highest fractional order, $\xi = 0.95$, the susceptible population reached the maximum value of about 484,000 and the lowest value of about 141,000. Also, its graph has the highest slope. For the lowest fractional order $\xi = 0.75$, the maximum and minimum numbers of susceptible people are about 450,000 and 173,000, respectively. It is clear that the smoothness of plots has an inverse relation with the fractional order.

The plots of all HIV-positive groups with no signs of AIDS are increasing, and the rate of increase has a direct relation with the fractional order, that is, the highest increase for $\mathcal{P}_i$ is found for $\xi = 0.95$ and the lowest increase is found for $\xi = 0.75$. After 150 days, these plots show linear growth and the numbers of the populations of this group are close to each other. From the plots of $\mathcal{P}_c$ and $\mathcal{P}_a$ in Fig. 2, it is evident that the plots of these populations have similar behavior to $\mathcal{P}_i$. Indeed, considering the use of medical tests and ART, the

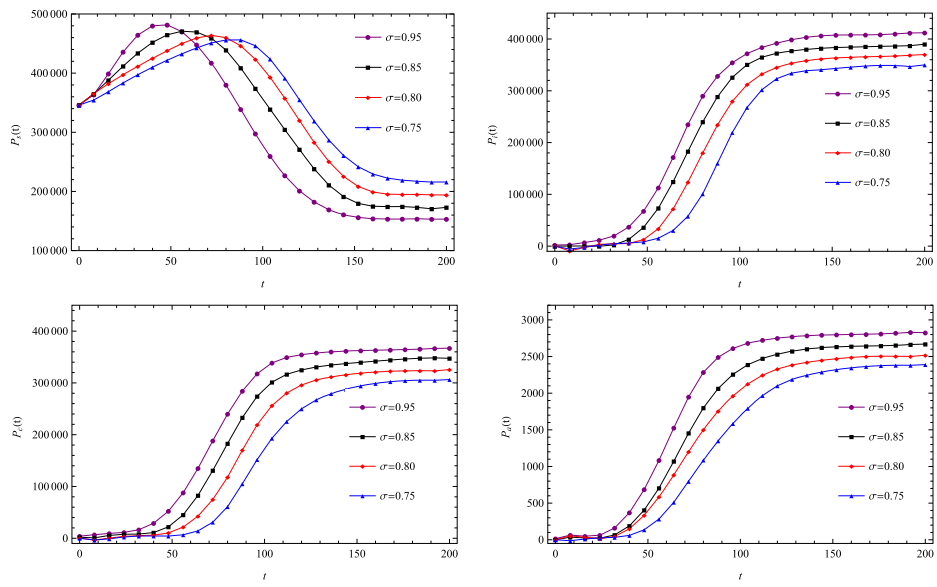

Figure 3 Dynamics of model (13) for $\xi = 0.9$ and $\sigma = 0.75$, 0.80, 0.85, and 0.95

decrease in the susceptible group and the increase in other groups were predictable. There is a direct link between the order of the fractional derivative and the slope of the reported plots. For the same fractal order σ, the diagrams of all four state functions have a high slope for the biggest fractional order ξ, and for $\xi = 0.75$ the plots are smoother.

8.2 Effects of the fractal derivative order

To analyze the effects of the fractal derivative order on the system solutions, we fixed the fractional order as $\xi = 0.9$ and changed the fractal orders in (13) and solved the obtained system by the proposed scheme. The numerical solutions are shown in Fig. 3. In the four parts of this figure, the maximum numbers of the populations are registered for $\sigma = 0.95$. Although the decrease of the slopes for decreasing order σ is obviously evident, the rate of decrease is low. For $\sigma = 0.95$, the number of susceptible people reaches its maximum value (480,000) after 35 days, and after 150 days it shows stable behavior, having decreased to (about) 150,000 in the interval $[150, 200]$. For the lowest fractal order, $\sigma = 0.75$, the maximum and minimum numbers are 454,000 and 220,000, respectively. It should be noted that the differences between the maximum and minimum values for $\sigma = 0.95$ and 0.75 are 330,000 and 234,000, respectively.

The second part of Fig. 3 shows the positive growth of $\mathcal{P}_i$ by increasing σ; however, all four branches of the plot show almost stable behavior after some t. In detail, the growth of the group of HIV-positive individuals with no signs of AIDS decays after about 100 days. Similar dynamics are evident for the diagrams of $\mathcal{P}_c$ and $\mathcal{P}_a$.

8.3 Simultaneous effects of the fractal dimension and fractional order

To study the effects of both fractional order and fractal dimension, we solved the system (13) by the introduced numerical scheme for different values of ξ and σ. The plots of approximated solutions are shown in Fig. 4. As we can see in the upper left figure, for $\xi = 0.9$ and $\sigma = 0.95$, the susceptible group reaches the highest number (about 473,000)

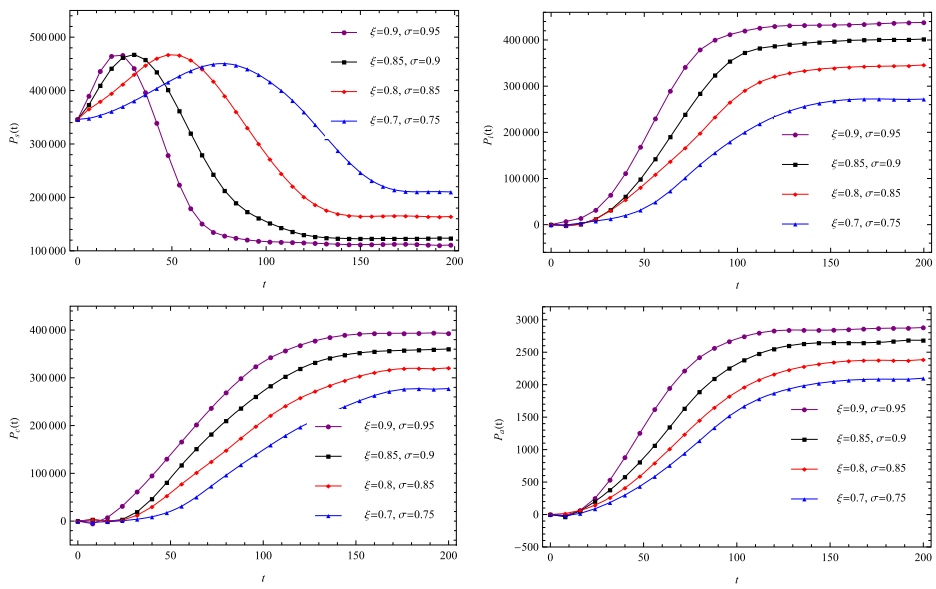

Figure 4 Dynamics of model (13) for $\xi = 0.9$ and $\sigma = 0.75$, 0.80, 0.85, and 0.95

after 24 days and the group exhibits negative growth until 83 days, exhibiting stable behavior after 83 days. For the same values of ξ and σ, the number of HIV-positive individuals with no signs of AIDS increases until 90 days and then stabilizes. It is evident from all parts of Fig. 4 that for the smallest values of ξ and σ, the $\mathcal{P}_i$, $\mathcal{P}_c$, and $\mathcal{P}_a$ groups have a low growth rate.

9 Conclusion

In the present study, we analyzed the transmission of HIV/AIDS in the context of a nonlinear system with four state functions. The system was equipped with FF derivatives and power-law type kernels. We employed Banach's contraction principle and a generalized α-ψ-Geraghty type contraction to establish the existence of a unique solution of the proposed model. Ulam–Hyers stability analysis was conducted. To define the initial values, data reported by the WHO and from the references [27, 29] for the Cape Verde Islands in 1987–2014 were applied. The GPs were utilized via the spectral collocation method to reduce the main FF model to some algebraic system and the Newton iterative scheme was used to find the numerical solutions. The approximated solutions were simulated for different values of fractal dimensions and fractional orders, and the results are shown in figures.

Regarding the numerical results, we examined the impact of the fractional derivative order on the solutions by choosing a fixed value for the fractal dimension and varying the fractional order (Fig. 2). Also, the effect of the fractal dimension was investigated by using different values and setting a fixed value for the fractional order (Fig. 3). As we can see in all diagrams, the susceptible population decreases while the population of HIV-positive individuals increases. For larger values of fractional orders, the rate of decay in susceptible populations is faster than for smaller values. On the contrary, the growth rate of other populations is faster for high fractional orders. Consequently, the obtained results are helpful in predicting HIV/AIDS transmission in human populations in various coun-

tries, of course using appropriate system parameters and initial values. It should be noted that by controlling the parameter Ψ, the growth rates of the HIV-positive population will decrease, so the AIDS-positive population goes to zero for a sufficiently small value of Ψ.

Ulam–Hyers stability guarantees the convergence of the numerical sequence of the proposed computational method for solving the main system. As we can see in Figs. 2–4, the solutions are convergent and exhibit similar behavior in the studied time intervals.

In our future research, we will try to model the dynamics of some types of cancer by using FF derivatives and solving the investigated systems by numerical approaches.

Data Availability
No datasets were generated or analysed during the current study.

Declarations

Competing interests
The authors declare no competing interests.

Author contributions
Yanru Wu: Formal analysis, Writing review, M. Nosrati: Conceptualization, Investigation, H. Afshari: Conceptualization, Investigation, Writing–original draft, M. Atapour: Writing–original draft, Editing, A. Mohammadzadeh: Writing –review, Editing.

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