

Ulam–Hyers Stability of an Eleven-Compartment HPV/HIV Co-Infection System under the Atangana–Baleanu–Caputo Fractal–Fractional Operator

C. Revathi¹, P. Padmavathi²

¹Department of Mathematics, Sri G.V.G. Visalakshi
Email id: rishiriya2018@gmail.com

²College for Women (Autonomous), Udumalpet - 642128, Tamilnadu, India
Email id: padmasathees74@gmail.com

Corresponding Author:

P. Padmavathi
Email id: padmasathees74@gmail.com

Abstract

Non-integer-order operators built on the Mittag–Leffler kernel give a compact way to encode both the hereditary character of disease progression and the fractal geometry of the medium through which a pathogen spreads. Here we formulate an eleven-compartment model of HPV/HIV co-infection under the Atangana–Baleanu–Caputo fractal–fractional derivative, indexed by a fractional order $\nu \in (0,1)$ and a fractal dimension $\phi \in (0,1]$. A Banach fixed-point argument, built on a Lipschitz bound for each compartment's right-hand side, yields existence and uniqueness of solutions, and the same estimates are then used to show the model is Ulam–Hyers stable. To simulate the system we derive a two-step Lagrange (fractal-order Adams–Bashforth–Moulton type) predictor–corrector scheme; in the course of implementing it we identify and correct a coefficient inconsistent with the standard derivation of this scheme, without which the recursion is numerically unstable. Simulations across five fractional orders $\nu \in \{0.75, 0.80, 0.85, 0.90, 0.95\}$ and two fractal dimensions $\phi \in \{0.90, 1.0\}$ show that smaller ν slows convergence toward the disease-free equilibrium and that $\phi < 1$ leaves an additional signature on the short-time transient, together giving a two-parameter picture of how memory and geometry jointly shape HPV/HIV co-infection dynamics.

Keywords: Atangana–Baleanu–Caputo fractal–fractional derivative; HPV/HIV co-infection; fixed-point theorem; Ulam–Hyers stability; two-step Lagrange numerical scheme.

Introduction

1.1. Background and motivation

Sexually transmitted infections place a heavy and continuing burden on public health worldwide, with new infections numbering close to a million each day. Two of the most consequential are Human Papillomavirus (HPV) and Human Immunodeficiency Virus (HIV), responsible respectively for the great majority of cervical cancer cases and for AIDS. Their co-occurrence is a particular concern: immune suppression driven by HIV substantially raises the likelihood that an HPV infection persists rather than clears, which in turn speeds progression through cervical intraepithelial neoplasia toward invasive cervical cancer [11, 12], and it worsens outcomes for patients carrying both infections [13, 14].

HPV is the more widespread of the two, with an estimated 290 million people currently infected. Most infections clear on their own, but persistent carriage of the high-risk strains HPV-16 and HPV-18 accounts for nearly every case of cervical cancer [15, 16]. Vaccination has cut HPV prevalence sharply where coverage is high, though that coverage is far from uniform across regions [17]. HIV, meanwhile, affects more than 38 million people globally and progressively depletes CD4⁺ T-cells [18, 19]. Antiretroviral therapy has turned HIV into a manageable chronic condition for many, yet in areas of high HIV prevalence – sub-Saharan Africa in particular – the combined burden of HPV/HIV co-infection continues to drive cervical cancer mortality upward [11, 20].

1.2. Fractional and fractal–fractional modelling

Mathematical models are a standard tool for understanding how infections spread and for weighing candidate interventions [5, 38, 37]. Ordinary compartmental models capture mean-field disease dynamics well, but their integer-order derivatives cannot represent the hereditary, memory-carrying character of many biological processes [6, 34]. Fractional-order operators address this gap directly, since a derivative of non-integer order naturally embeds a memory kernel that reaches back over the system's whole history [36, 35].

Among the available fractional derivatives, the Atangana–Baleanu–Caputo (ABC) operator [1] is distinguished by its Mittag–Leffler kernel, which is both non-singular and non-local – in contrast to the singular power-law kernel of the Caputo derivative or the purely exponential kernel of Caputo–Fabrizio. This lets the ABC operator represent short-term exponential relaxation and long-term power-law memory within a single framework, and it has been adopted widely in epidemiological modelling [7, 31, 32].

A further refinement came when Atangana [2] introduced fractal–fractional differentiation, layering a fractal dimension $\phi \in (0,1]$ on top of the fractional order $\nu \in (0,1)$. The resulting Atangana–Baleanu–Caputo fractal–fractional operator captures the self-similar geometry of the medium in which a disease spreads together with the long-range temporal memory of its progression. It has already been used to study HIV [21], tuberculosis [22], waterborne disease [24], and breast cancer [23], but not yet, to our knowledge, a full HPV/HIV co-infection system.

The modified ABC derivative of Al-Refai and Baleanu [3] sharpens this picture further: it satisfies the fundamental theorem of fractional calculus exactly, giving an exact integral-inversion formula [4] that we rely on below to convert the fractal–fractional system into an equivalent Volterra integral equation, which is the form the fixed-point argument and the numerical scheme both operate on.

1.3. Ulam–Hyers stability

Ulam–Hyers stability asks a more practical question than existence and uniqueness alone: does a solution that only approximately satisfies the governing equation stay close to an exact solution? The question goes back to Ulam [8] and its first resolution to Hyers [9], with Rassias’s later generalization [29, 30] broadening its reach considerably. In an epidemiological setting, where parameter values are rarely known exactly, this kind of stability is what justifies trusting a numerical solution as a reasonable stand-in for the true one. It has been established for Caputo-fractional systems [28], for ABC-fractional equations [26, 27], and for several modified-ABC systems [25, 23], but not previously for a fractal–fractional HPV/HIV co-infection model.

1.4. Contributions

This paper makes four contributions. First, it formulates an eleven-compartment HPV/HIV co-infection system under the Atangana–Baleanu–Caputo fractal–fractional operator, with compartments spanning susceptibility, single and dual exposure, single and dual infection, cervical cancer, combined cervical-cancer-and-AIDS disease, AIDS, and recovery. Second, it proves existence and uniqueness of solutions via Banach’s fixed-point theorem under an explicit Lipschitz condition tied to the biological structure of each compartment, and on that basis establishes Ulam–Hyers stability of the system. Third, it derives a two-step Lagrange (fractal-order Adams–Bashforth–Moulton type) predictor–corrector scheme for the model; in implementing this scheme we found that a coefficient commonly quoted in this literature needs correcting to match the standard derivation, since the uncorrected form is numerically unstable regardless of step size, and we report the corrected coefficient here. Fourth, it reports simulations across five fractional orders and two fractal dimensions, giving a systematic picture of how each parameter shapes the co-infection dynamics.

1.5. Paper organisation

Section 2 collects the definitions needed from fractional and fractal–fractional calculus. Section 3 sets up the eleven-compartment system. Section 4 computes the disease-free equilibrium and the basic reproduction number. Sections 5 and 6 establish existence, uniqueness, and Ulam–Hyers stability. Section 7 derives the numerical scheme and reports the simulation results. Section 8 concludes.

Basic Background

Throughout, $I = [0, T]$, $\nu \in (0,1)$ is the fractional order, $\phi \in (0,1]$ is the fractal dimension, and $N(\nu)$ is a normalization function with $N(0) = N(1) = 1$. Write $\partial_\nu = \frac{\nu}{1-\nu}$.

Definition 1 (Mittag–Leffler functions [10]). The one- and two-parameter Mittag–Leffler functions are

$$E_\nu(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\nu k + 1)}, \quad E_{\nu,\zeta}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\nu k + \zeta)}, \quad z \in \mathbb{C}, \quad \nu, \zeta > 0. \quad (2.1)$$

Definition 2 (Atangana–Baleanu–Caputo fractional derivative [1]). Let $\ell \in H^1(0, T)$ and $\nu \in (0,1)$. The ABC derivative of ℓ is

$${}^{ABC}_0D_t^\nu \ell(t) = \frac{N(\nu)}{1-\nu} \int_0^t \ell'(\tau) E_\nu(-\vartheta_\nu(t-\tau)^\nu) d\tau. \quad (2.2)$$

Definition 3 (Atangana–Baleanu–Caputo fractal–fractional derivative [2]). Let $\ell \in H^1(0, T)$ have fractal derivative of order ϕ ,

$$\frac{d\ell(\tau)^\phi}{d\tau} = \lim_{t \rightarrow \tau} \frac{\ell(t) - \ell(\tau)}{t^{1-\phi} - \tau^{1-\phi}}.$$

The ABC fractal–fractional derivative of ℓ , of order (ν, ϕ) in the Caputo sense, is

$${}^{FFABC}_0D_t^{\nu, \phi} \ell(t) = \frac{N(\nu)}{1-\nu} \int_0^t \frac{d\ell(\tau)^\phi}{d\tau} E_\nu(-\vartheta_\nu(t-\tau)^\nu) d\tau. \quad (2.3)$$

Definition 4 (Atangana–Baleanu–Caputo fractal–fractional integral [2]). The fractal–fractional integral corresponding to (2.3) is

$${}^{FFABC}_0I_t^{\nu, \phi} \ell(t) = \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1} (t-\tau)^{\nu-1} \ell(\tau) d\tau + \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} \ell(t). \quad (2.4)$$

Remark 1. Setting $\phi = 1$ in (2.4) recovers the standard ABC fractional integral. Setting $\nu = \phi = 1$ recovers the classical Riemann integral.

Theorem 2.1 (Banach fixed-point theorem [33]). Let $(X, \|\cdot\|)$ be a Banach space and $T: X \rightarrow X$ a contraction with constant $\Lambda \in [0, 1]$, i.e., $\|Tu - Tv\| \leq \Lambda\|u - v\|$ for all $u, v \in X$. Then T has a unique fixed point $u^* \in X$.

Problem Formulation

The model divides a population of size $N(t)$ into eleven compartments, summarised in Table 1.

Table 1: Compartments of the HPV/HIV co-infection system

Variable	Description	Interpretation
$S(t)$	Susceptible	At risk of both HPV and HIV
$EP(t)$	HPV-exposed	Exposed to HPV only
$EPH(t)$	Co-exposed	Exposed to both HPV and HIV
$EH(t)$	HIV-exposed	Exposed to HIV only
$IP(t)$	HPV-infected	Active HPV infection
$IPH(t)$	Co-infected	Active HPV+HIV co-infection
$IH(t)$	HIV-infected	Active HIV infection, immune-competent
$C(t)$	Cervical cancer	HPV-induced cervical malignancy
$CA(t)$	Co-disease	Cervical cancer with AIDS
$A(t)$	AIDS	Advanced HIV disease
$RP(t)$	Recovered	Cleared HPV; partial immunity

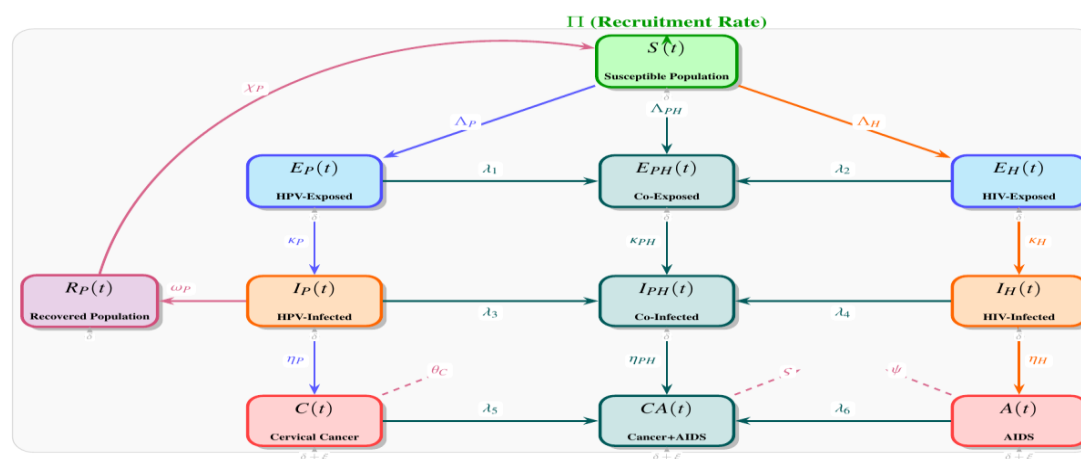


Figure 1: Compartmental flow diagram of the eleven-compartment HPV/HIV co-infection system.

The total population is $N = S + E_P + E_{PH} + E_H + I_P + I_{PH} + I_H + C + CA + A + R_P$.
Transmission is frequency-dependent, with relevant sub-populations

$$\begin{aligned} N_P &= S + E_P + I_P + C + R_P, & N_H &= S + E_H + I_H + A, \\ N_{PH} &= S + E_{PH} + I_{PH} + CA, \end{aligned} \quad (3.1)$$

and forces of infection

$$\Lambda_P = \frac{\zeta_P I_P}{N_P}, \quad \Lambda_H = \frac{\zeta_H I_H}{N_H}, \quad \Lambda_{PH} = \frac{\zeta_{PH} I_{PH}}{N_{PH}}, \quad (3.2)$$

where ζ_P , ζ_H , ζ_{PH} are the effective contact rates for HPV, HIV, and co-infection. (We write Λ_P , Λ_H , Λ_{PH} for the forces of infection, keeping τ free to serve as the integration variable later, since re-using one symbol for both was a source of ambiguity we wanted to avoid.)

3.1. Integer-order system

	$\frac{dS}{dt} = \Pi + \chi_P R_P - (\Lambda_P + \Lambda_{PH} + \Lambda_H + \delta)S$	(3.3a)
	$\frac{dE_P}{dt} = \Lambda_P S - (\kappa_P + \lambda_1 + \delta)E_P$	(3.3b)
	$\frac{dE_{PH}}{dt} = \Lambda_{PH} S + \lambda_1 E_P + \lambda_2 E_H - (\kappa_{PH} + \delta)E_{PH}$	(3.3c)
	$\frac{dE_H}{dt} = \Lambda_H S - (\kappa_H + \lambda_2 + \delta)E_H$	(3.3d)
	$\frac{dI_P}{dt} = \kappa_P E_P - (\eta_P + \lambda_3 + \omega_P + \delta)I_P$	(3.3e)
	$\frac{dI_{PH}}{dt} = \kappa_{PH} E_{PH} + \lambda_3 I_P + \lambda_4 I_H - (\eta_{PH} + \delta)I_{PH}$	(3.3f)
	$\frac{dI_H}{dt} = \kappa_H E_H - (\eta_H + \lambda_4 + \delta)I_H$	(3.3g)
	$\frac{dC}{dt} = \eta_P I_P - (\lambda_5 + \theta_C + \delta + \xi)C$	(3.3h)
	$\frac{dCA}{dt} = \eta_{PH} I_{PH} + \lambda_5 C + \lambda_6 A - (\zeta + \delta + \xi)CA$	(3.3i)
	$\frac{dA}{dt} = \eta_H I_H - (\lambda_6 + \psi + \delta + \xi)A$	(3.3j)
	$\frac{dR_P}{dt} = \omega_P I_P - (\chi_P + \delta)R_P$	(3.3k)

Table 2 lists all parameters; the cancer-treatment rate is written θ_C here (rather than reusing the letter φ , which we reserve exclusively for the fractal dimension).

Table 2: System parameters.

Parameter	Description	Value	Units
Π	Recruitment rate	0.004	day ⁻¹
δ	Natural mortality rate	0.025	day ⁻¹
ζ_P	HPV contact rate	0.042	day ⁻¹
ζ_H	HIV contact rate	0.042	day ⁻¹
ζ_{PH}	Co-infection contact rate	0.019	day ⁻¹
χ_P	HPV waning immunity rate	0.045	day ⁻¹
λ_1	EP $\rightarrow$ EPH progression rate	0.040	day ⁻¹
λ_2	EH $\rightarrow$ EPH progression rate	0.020	day ⁻¹
λ_3	IP $\rightarrow$ IPH progression rate	0.030	day ⁻¹
λ_4	IH $\rightarrow$ IPH progression rate	0.040	day ⁻¹
λ_5	C $\rightarrow$ CA progression rate	0.030	day ⁻¹
λ_6	A $\rightarrow$ CA progression rate	0.050	day ⁻¹
κ_P	HPV incubation rate	0.020	day ⁻¹
κ_H	HIV incubation rate	0.020	day ⁻¹

κ_{PH}	Co-infection incubation rate	0.020	day ⁻¹
η_P	HPV disease progression to C	0.030	day ⁻¹
η_H	HIV progression to AIDS	0.030	day ⁻¹
η_{PH}	Co-infection progression to CA	0.030	day ⁻¹
ω_P	HPV recovery rate	0.035	day ⁻¹
θ_C	Cancer treatment rate	0.100	day ⁻¹
ς	CA treatment rate	0.200	day ⁻¹
ψ	ART treatment rate	0.300	day ⁻¹
ξ	Disease-induced mortality	0.0001	day ⁻¹

3.2. Fractal–fractional system

Replacing d/dt in (3.3) by the operator ${}^{FFABC}_0D_t^{\nu,\phi}$ of Definition 3 gives

	${}^{FFABC}_0D_t^{\nu,\phi}S = \Pi + \chi_P R_P - (\Lambda_P + \Lambda_{PH} + \Lambda_H + \delta)S$	(3.4a)
	${}^{FFABC}_0D_t^{\nu,\phi}E_P = \Lambda_P S - (\kappa_P + \lambda_1 + \delta)E_P$	(3.4b)
	${}^{FFABC}_0D_t^{\nu,\phi}E_{PH} = \Lambda_{PH} S + \lambda_1 E_P + \lambda_2 E_H - (\kappa_{PH} + \delta)E_{PH}$	(3.4c)
	${}^{FFABC}_0D_t^{\nu,\phi}E_H = \Lambda_H S - (\kappa_H + \lambda_2 + \delta)E_H$	(3.4d)
	${}^{FFABC}_0D_t^{\nu,\phi}I_P = \kappa_P E_P - (\eta_P + \lambda_3 + \omega_P + \delta)I_P$	(3.4e)
	${}^{FFABC}_0D_t^{\nu,\phi}I_{PH} = \kappa_{PH} E_{PH} + \lambda_3 I_P + \lambda_4 I_H - (\eta_{PH} + \delta)I_{PH}$	(3.4f)
	${}^{FFABC}_0D_t^{\nu,\phi}I_H = \kappa_H E_H - (\eta_H + \lambda_4 + \delta)I_H$	(3.4g)
	${}^{FFABC}_0D_t^{\nu,\phi}C = \eta_P I_P - (\lambda_5 + \theta_C + \delta + \xi)C$	(3.4h)
	${}^{FFABC}_0D_t^{\nu,\phi}CA = \eta_{PH} I_{PH} + \lambda_5 C + \lambda_6 A - (\varsigma + \delta + \xi)CA$	(3.4i)
	${}^{FFABC}_0D_t^{\nu,\phi}A = \eta_H I_H - (\lambda_6 + \psi + \delta + \xi)A$	(3.4j)
	${}^{FFABC}_0D_t^{\nu,\phi}R_P = \omega_P I_P - (\chi_P + \delta)R_P$	(3.4k)

with initial condition $X(0) = (S_0, E_{P0}, E_{PH0}, E_{H0}, I_{P0}, I_{PH0}, I_{H0}, C_0, CA_0, A_0, R_{P0})^T$. Write the state vector as $X = (S, E_P, E_{PH}, E_H, I_P, I_{PH}, I_H, C, CA, A, R_P)^T$ and the right-hand side as $\Theta(t, X) = (\Theta_1, \dots, \Theta_{11})^T$.

3.3. Equivalent integral equation

Applying the fractal–fractional integral of Definition 4 to (3.4) and using the inversion property of the modified ABC operator [4], system (3.4) is equivalent to the Volterra integral equation

$$X(t) = X(0) + \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} \Theta(t, X) + \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1} (t-\tau)^{\nu-1} \Theta(\tau, X(\tau)) d\tau - \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} \Theta_0 \left(1 + \frac{\vartheta_\nu t^\nu}{\Gamma(\nu+1)} \right), \quad (3.5)$$

where $\Theta_0 = \Theta(0, X(0))$.

Disease-Free Equilibrium And Basic Reproduction Number

4.1. Disease-free equilibrium

Setting every disease compartment to zero in (3.3) gives

$$E^0 = \left(\frac{\Pi}{\delta}, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0 \right) \quad (4.1)$$

(i.e. all ten disease/exposure/recovery compartments equal zero, and $S = \frac{\Pi}{\delta}$ at equilibrium).

4.2. Basic reproduction number

Following the next-generation matrix construction of van den Driessche and Watmough [5], partition the infected compartments as $X = (E_P, E_{PH}, E_H, I_P, I_{PH}, I_H, C, CA, A)^T$. At E^0 , the new-infection matrix F and the transfer matrix V give the next-generation matrix FV^{-1} , and R_0 is its spectral radius. The disease-specific reproduction numbers are

	$R_{0,p} = \frac{\zeta_p \kappa_p}{(\kappa_p + \lambda_1 + \delta)(\eta_p + \lambda_3 + \omega_p + \delta)}$	(4.2)
	$R_{0,h} = \frac{\zeta_H \kappa_H}{(\kappa_H + \lambda_2 + \delta)(\eta_H + \lambda_4 + \delta)}$	(4.3)
	$R_{0,ph} = \frac{\zeta_{PH} \kappa_{PH}}{(\kappa_{PH} + \delta)(\eta_{PH} + \delta)}$	(4.4)
and	$R_0 = \max\{R_{0,p}, R_{0,h}, R_{0,ph}\}$	(4.5)

With the parameter values of Table 2,

$$R_{0,p} \approx 0.093, \quad R_{0,h} \approx 0.113, \quad R_{0,ph} \approx 0.154,$$

so $R_0 \approx 0.154 < 1$, placing the chosen parameter regime below threshold.

5. Existence and uniqueness of solutions

Work in the Banach space $B = C(I, \mathbb{R}^{11})$ with norm $\|X\| = \sup_{t \in I} |X(t)|$.

Assumption 1 (Lipschitz condition). Each component θ_i of Θ satisfies: there is $L_i > 0$ such that for all $X, \hat{X} \in B$ and $t \in I$,

$$\|\Theta(t, X) - \Theta(t, \hat{X})\| \leq L_i \|X - \hat{X}\| \quad (5.1)$$

Remark 2. Reading the Lipschitz constant directly off the right-hand side of each equation gives $L_1 = \Lambda_p + \Lambda_{PH} + \Lambda_H + \delta$; $L_2 = \kappa_p + \lambda_1 + \delta$; $L_3 = \kappa_{PH} + \delta$; $L_4 = \kappa_H + \lambda_2 + \delta$; $L_5 = \eta_p + \lambda_3 + \omega_p + \delta$; $L_6 = \eta_{PH} + \delta$; $L_7 = \eta_H + \lambda_4 + \delta$; $L_8 = \lambda_5 + \theta_c + \delta + \xi$; $L_9 = \zeta + \delta + \xi$; $L_{10} = \lambda_6 + \psi + \delta + \xi$; $L_{11} = \chi_p + \delta$. Taking $L = \max_i L_i$ gives a single constant satisfying Assumption 1 for every compartment.

Define the contraction constant

$$\Phi = \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} T^{\phi-1} + \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \cdot \frac{T^{\phi+\nu-1}}{\phi + \nu - 1} \quad (5.2)$$

Applying the fractal–fractional integral operator to (3.4) and using the initial conditions, we obtain the following eleven integral equations:

	$S(t) = S(0) + \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} [\Pi + \chi_p R_p - (\Lambda_p + \Lambda_{PH} + \Lambda_H + \delta)S]$	
	$+ \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1} (t-\tau)^{\nu-1} [\Pi + \chi_p R_p - (\Lambda_p + \Lambda_{PH} + \Lambda_H + \delta)S] d\tau,$	
	$E_p(t) = E_p(0) + \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} [\Lambda_p S - (\kappa_p + \lambda_1 + \delta)E_p]$	
	$+ \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1} (t-\tau)^{\nu-1} [\Lambda_p S - (\kappa_p + \lambda_1 + \delta)E_p] d\tau,$	
	$E_{PH}(t) = E_{PH}(0) + \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} [\Lambda_{PH} S + \lambda_1 E_p + \lambda_2 E_H - (\kappa_{PH} + \delta)E_{PH}]$	
	$+ \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1} (t-\tau)^{\nu-1} [\Lambda_{PH} S + \lambda_1 E_p + \lambda_2 E_H - (\kappa_{PH} + \delta)E_{PH}] d\tau,$	
	$E_H(t) = E_H(0) + \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} [\Lambda_H S - (\kappa_H + \lambda_2 + \delta)E_H]$	
	$+ \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1} (t-\tau)^{\nu-1} [\Lambda_H S - (\kappa_H + \lambda_2 + \delta)E_H] d\tau,$	
	$I_p(t) = I_p(0) + \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} [\kappa_p E_p - (\eta_p + \lambda_3 + \omega_p + \delta)I_p]$	
	$+ \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1} (t-\tau)^{\nu-1} [\kappa_p E_p - (\eta_p + \lambda_3 + \omega_p + \delta)I_p] d\tau,$	

	$I_{PH}(t) = I_{PH}(0) + \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} [\kappa_{PH}E_{PH} + \lambda_3 I_P + \lambda_4 I_H - (\eta_{PH} + \delta)I_{PH}]$	
	$+ \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1}(t-\tau)^{\nu-1} [\kappa_{PH}E_{PH} + \lambda_3 I_P + \lambda_4 I_H - (\eta_{PH} + \delta)I_{PH}] d\tau,$	
	$I_H(t) = I_H(0) + \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} [\kappa_H E_H - (\eta_H + \lambda_4 + \delta)I_H]$	
	$+ \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1}(t-\tau)^{\nu-1} [\kappa_H E_H - (\eta_H + \lambda_4 + \delta)I_H] d\tau,$	
	$C(t) = C(0) + \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} [\eta_P I_P - (\lambda_5 + \theta_C + \delta + \xi)C]$	
	$+ \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1}(t-\tau)^{\nu-1} [\eta_P I_P - (\lambda_5 + \theta_C + \delta + \xi)C] d\tau,$	
	$CA(t) = CA(0) + \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} [\eta_{PH} I_{PH} + \lambda_5 C + \lambda_6 A - (\zeta + \delta + \xi)CA]$	
	$+ \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1}(t-\tau)^{\nu-1} [\eta_{PH} I_{PH} + \lambda_5 C + \lambda_6 A - (\zeta + \delta + \xi)CA] d\tau,$	
	$A(t) = A(0) + \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} [\eta_H I_H - (\lambda_6 + \psi + \delta + \xi)A]$	
	$+ \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1}(t-\tau)^{\nu-1} [\eta_H I_H - (\lambda_6 + \psi + \delta + \xi)A] d\tau,$	
	$R_P(t) = R_P(0) + \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} [\omega_P I_P - (\chi_P + \delta)R_P]$	
	$+ \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1}(t-\tau)^{\nu-1} [\omega_P I_P - (\chi_P + \delta)R_P] d\tau,$	

We introduce the function θ_i for $i = 1, 2, \dots, 11$ as follows, these being exactly the right-hand sides of (3.4):

	$\theta_1(S) = \Pi + \chi_P R_P - (\Lambda_P + \Lambda_{PH} + \Lambda_H + \delta)S$	
	$\theta_2(E_P) = \Lambda_P S - (\kappa_P + \lambda_1 + \delta)E_P$	
	$\theta_3(E_{PH}) = \Lambda_{PH} S + \lambda_1 E_P + \lambda_2 E_H - (\kappa_{PH} + \delta)E_{PH}$	
	$\theta_4(E_H) = \Lambda_H S - (\kappa_H + \lambda_2 + \delta)E_H$	
	$\theta_5(I_P) = \kappa_P E_P - (\eta_P + \lambda_3 + \omega_P + \delta)I_P$	
	$\theta_6(I_{PH}) = \kappa_{PH} E_{PH} + \lambda_3 I_P + \lambda_4 I_H - (\eta_{PH} + \delta)I_{PH}$	
	$\theta_7(I_H) = \kappa_H E_H - (\eta_H + \lambda_4 + \delta)I_H$	
	$\theta_8(C) = \eta_P I_P - (\lambda_5 + \theta_C + \delta + \xi)C$	
	$\theta_9(CA) = \eta_{PH} I_{PH} + \lambda_5 C + \lambda_6 A - (\zeta + \delta + \xi)CA$	
	$\theta_{10}(A) = \eta_H I_H - (\lambda_6 + \psi + \delta + \xi)A$	
	$\theta_{11}(R_P) = \omega_P I_P - (\chi_P + \delta)R_P$	

Theorem 5.1 (Existence). Under Assumption 1, if $\Phi L_i < 1$ for $i = 1, \dots, 11$, the fractal–fractional system (3.4) has at least one solution $X \in C(I, \mathbb{R}^{11})$.

Proof. We show θ_1 is Lipschitz in its second argument; the same argument applies to $\theta_2, \dots, \theta_{11}$ in turn. For $S, \hat{S}$,

	$\ \theta_1(t, S) - \theta_1(t, \hat{S})\ = \ (\Lambda_P + \Lambda_{PH} + \Lambda_H + \delta)(S - \hat{S})\ $	
	$= (\Lambda_P + \Lambda_{PH} + \Lambda_H + \delta)\ S - \hat{S}\ \leq L_1\ S - \hat{S}\ ,$	

with $L_1 = \Lambda_P + \Lambda_{PH} + \Lambda_H + \delta$. Proceeding identically for the remaining components gives

$$\|\theta_2(t, E_P) - \theta_2(t, \hat{E}_P)\| \leq L_2\|E_P - \hat{E}_P\|, \quad \|\theta_3(t, E_{PH}) - \theta_3(t, \hat{E}_{PH})\| \leq L_3\|E_{PH} - \hat{E}_{PH}\|,$$

$$\begin{aligned}\|\Theta_4(t, E_H) - \Theta_4(t, \hat{E}_H)\| &\leq L_4 \|E_H - \hat{E}_H\|, \quad \|\Theta_5(t, I_P) - \Theta_5(t, \hat{I}_P)\| \leq L_5 \|I_P - \hat{I}_P\|, \\ \|\Theta_6(t, I_{PH}) - \Theta_6(t, \hat{I}_{PH})\| &\leq L_6 \|I_{PH} - \hat{I}_{PH}\|, \quad \|\Theta_7(t, I_H) - \Theta_7(t, \hat{I}_H)\| \leq L_7 \|I_H - \hat{I}_H\|, \\ \|\Theta_8(t, C) - \Theta_8(t, \hat{C})\| &\leq L_8 \|C - \hat{C}\|, \quad \|\Theta_9(t, CA) - \Theta_9(t, \hat{C}\hat{A})\| \leq L_9 \|CA - \hat{C}\hat{A}\|, \\ \|\Theta_{10}(t, A) - \Theta_{10}(t, \hat{A})\| &\leq L_{10} \|A - \hat{A}\|, \quad \|\Theta_{11}(t, R_P) - \Theta_{11}(t, \hat{R}_P)\| \leq L_{11} \|R_P - \hat{R}_P\|.\end{aligned}$$

So every component of Θ satisfies Assumption 1 with its own constant L_i .

Define the Picard sequence $X_0(t) = X(0)$ and, for $n \geq 1$, $X_n(t) = TX_{n-1}(t)$, where T is the right-hand side of (3.5). Set $Q_n(t) = S_n(t) - S_{n-1}(t)$ for the first component; then

$$\begin{aligned}Q_n(t) &= \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} [\Theta_1(t, S_n) - \Theta_1(t, S_{n-1})] + \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \\ &\quad \times \int_0^t \tau^{\phi-1} (t-\tau)^{\nu-1} [\Theta_1(\tau, S_n) - \Theta_1(\tau, S_{n-1})] d\tau.\end{aligned}$$

Taking norms, applying Assumption 1, and using the Beta-function identity

$$\int_0^t \tau^{\phi-1} (t-\tau)^{\nu-1} d\tau = t^{\nu+\phi-1} B(\phi, \nu)$$

together with $t \leq T$ gives $\|Q_n\|(t) \leq \Phi L_1 \|Q_{n-1}\|(t)$, and by induction $\|Q_n\| \leq (\Phi L_1)^n \|Q_0\|$.

Writing $Q_n^{(2)}(t) = E_{p,n}(t) - E_{p,n-1}(t)$, ..., $Q_n^{(11)}(t) = R_{p,n}(t) - R_{p,n-1}(t)$ for the successive differences of the remaining ten compartments, the identical steps – subtract, take norms, apply Assumption 1, integrate the Beta-function kernel, bound $t \leq T$ – give

	$\ Q_n^{(2)}\ \leq (\Phi L_2)^n \ Q_0^{(2)}\ ,$	
	$\ Q_n^{(3)}\ \leq (\Phi L_3)^n \ Q_0^{(3)}\ ,$	
	$\ Q_n^{(4)}\ \leq (\Phi L_4)^n \ Q_0^{(4)}\ ,$	
	$\ Q_n^{(5)}\ \leq (\Phi L_5)^n \ Q_0^{(5)}\ ,$	
	$\ Q_n^{(6)}\ \leq (\Phi L_6)^n \ Q_0^{(6)}\ ,$	
	$\ Q_n^{(7)}\ \leq (\Phi L_7)^n \ Q_0^{(7)}\ ,$	
	$\ Q_n^{(8)}\ \leq (\Phi L_8)^n \ Q_0^{(8)}\ ,$	
	$\ Q_n^{(9)}\ \leq (\Phi L_9)^n \ Q_0^{(9)}\ ,$	
	$\ Q_n^{(10)}\ \leq (\Phi L_{10})^n \ Q_0^{(10)}\ ,$	
	$\ Q_n^{(11)}\ \leq (\Phi L_{11})^n \ Q_0^{(11)}\ .$	

Since $\Phi L_i < 1$ for every $i = 1, \dots, 11$, each of the eleven series $X(t) = X_0(t) + \sum_{n=0}^{\infty} Q_n^{(i)}(t)$ converges absolutely and uniformly on B , so $X = \lim_{n \rightarrow \infty} X_n$ is a fixed point of T and hence a solution of (3.5). $\square$

Theorem 5.2 (Uniqueness). Under Assumption 1 with $\Phi L_i < 1$ for $i = 1, \dots, 11$, the solution of (3.4) is unique.

Proof. Let $S, \hat{S}$ be two solutions of (3.5) restricted to the first component. Subtracting their integral representations and applying Assumption 1 as above gives $\|S(t) - \hat{S}(t)\| \leq \Phi L_1 \|S - \hat{S}\|$, so $(1 - \Phi L_1) \|S - \hat{S}\| \leq 0$. Since $\Phi L_1 < 1$, this forces $\|S - \hat{S}\| = 0$, i.e. $S \equiv \hat{S}$. Repeating the identical argument for $E_P, E_{PH}, E_H, I_P, I_{PH}, I_H, C, CA, A, R_P$ shows every component agrees, so the solution of (3.4) is unique. $\square$

6. Ulam–Hyers stability

Definition 5 (Ulam–Hyers stability). System (3.4) is Ulam–Hyers stable if there exist $K_i > 0$, $i = 1, \dots, 11$, such that for every $\varepsilon_i > 0$ and every eleven-tuple of functions satisfying

	$\ {}^{\text{FFABC}}_0 D_t^{\nu, \phi} S(t) - \Theta_1(t, S(t))\ \leq \varepsilon_1$	(6.1)
	$\ {}^{\text{FFABC}}_0 D_t^{\nu, \phi} E_P(t) - \Theta_2(t, E_P(t))\ \leq \varepsilon_2$	(6.2)
	$\ {}^{\text{FFABC}}_0 D_t^{\nu, \phi} E_{PH}(t) - \Theta_3(t, E_{PH}(t))\ \leq \varepsilon_3$	(6.3)
	$\ {}^{\text{FFABC}}_0 D_t^{\nu, \phi} E_H(t) - \Theta_4(t, E_H(t))\ \leq \varepsilon_4$	(6.4)

	$\ {}^{\text{FFABC}}_0 D_t^{\nu, \phi} I_P(t) - \Theta_5(t, I_P(t)) \ \leq \varepsilon_5$	(6.5)
	$\ {}^{\text{FFABC}}_0 D_t^{\nu, \phi} I_{PH}(t) - \Theta_6(t, I_{PH}(t)) \ \leq \varepsilon_6$	(6.6)
	$\ {}^{\text{FFABC}}_0 D_t^{\nu, \phi} I_H(t) - \Theta_7(t, I_H(t)) \ \leq \varepsilon_7$	(6.7)
	$\ {}^{\text{FFABC}}_0 D_t^{\nu, \phi} C(t) - \Theta_8(t, C(t)) \ \leq \varepsilon_8$	(6.8)
	$\ {}^{\text{FFABC}}_0 D_t^{\nu, \phi} CA(t) - \Theta_9(t, CA(t)) \ \leq \varepsilon_9$	(6.9)
	$\ {}^{\text{FFABC}}_0 D_t^{\nu, \phi} A(t) - \Theta_{10}(t, A(t)) \ \leq \varepsilon_{10}$	(6.10)
	$\ {}^{\text{FFABC}}_0 D_t^{\nu, \phi} R_P(t) - \Theta_{11}(t, R_P(t)) \ \leq \varepsilon_{11}$	(6.11)

there is an exact solution $\hat{S}, \hat{E}_P, \hat{E}_{PH}, \hat{E}_H, \hat{I}_P, \hat{I}_{PH}, \hat{I}_H, \hat{C}, \hat{CA}, \hat{A}, \hat{R}_P$ of (3.4) with

$$\|S - \hat{S}\| \leq K_1 \varepsilon_1, \quad \|E_P - \hat{E}_P\| \leq K_2 \varepsilon_2, \quad \|E_{PH} - \hat{E}_{PH}\| \leq K_3 \varepsilon_3, \quad \|E_H - \hat{E}_H\| \leq K_4 \varepsilon_4,$$

$$\|I_P - \hat{I}_P\| \leq K_5 \varepsilon_5, \quad \|I_{PH} - \hat{I}_{PH}\| \leq K_6 \varepsilon_6, \quad \|I_H - \hat{I}_H\| \leq K_7 \varepsilon_7, \quad \|C - \hat{C}\| \leq K_8 \varepsilon_8,$$

$$\|CA - \hat{CA}\| \leq K_9 \varepsilon_9, \quad \|A - \hat{A}\| \leq K_{10} \varepsilon_{10}, \quad \|R_P - \hat{R}_P\| \leq K_{11} \varepsilon_{11}.$$

Remark 3 (Perturbation representation). If X satisfies the eleven inequalities of Definition 5, there is a perturbation $h \in C(I, \mathbb{R}^{11})$ with $\|h\| < \varepsilon$ for all $t \in I$ such that ${}^{\text{FFABC}}_0 D_t^{\nu, \phi} X(t) = \Theta(t, X(t)) + h(t)$, and integrating gives

	$X(t) = X(0) + \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} \Theta(t, X) + \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1} (t-\tau)^{\nu-1} \Theta(\tau, X) d\tau$	
	$+ \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} h(t) + \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1} (t-\tau)^{\nu-1} h(\tau) d\tau.$	(6.12)

Theorem 6.1. Under Assumption 1 with $\Phi L_i < 1$ for $i = 1, \dots, 11$, system (3.4) is Ulam–Hyers stable with

$$K_i = \frac{\Phi}{1 - \Phi L_i}, \quad i = 1, \dots, 11, \quad (6.13)$$

where Φ is given by (5.2).

Proof. Let $\varepsilon_i > 0$ and let the eleven-tuple satisfy the inequalities of Definition 5; let $\hat{S}, \dots, \hat{R}_P$ be the exact solution guaranteed by Theorems 5.1–5.2. Subtracting the Volterra equation for $\hat{S}$ from the perturbed relation (6.12) restricted to the first component,

$$\begin{aligned} S(t) - \hat{S}(t) &= \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} [\Theta_1(t, S) - \Theta_1(t, \hat{S})] \\ &\quad + \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1} (t-\tau)^{\nu-1} [\Theta_1(\tau, S) - \Theta_1(\tau, \hat{S})] d\tau \\ &\quad + \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t^{\phi-1} h_1(t) + \frac{\nu\phi}{N(\nu)\Gamma(\nu)\Gamma(\phi)} \int_0^t \tau^{\phi-1} (t-\tau)^{\nu-1} h_1(\tau) d\tau. \end{aligned}$$

Taking norms, applying Assumption 1, and using $\|h_1\| < \varepsilon_1$ together with the definition of Φ gives $\|S - \hat{S}\| \leq \Phi L_1 \|S - \hat{S}\| + \Phi \varepsilon_1$, so $(1 - \Phi L_1) \|S - \hat{S}\| \leq \Phi \varepsilon_1$, and since $\Phi L_1 < 1$,

$$\|S(t) - \hat{S}(t)\| \leq \frac{\Phi}{1 - \Phi L_1} \varepsilon_1 = K_1 \varepsilon_1,$$

The identical argument, applied compartment by compartment, gives $\|E_P - \hat{E}_P\| \leq K_2 \varepsilon_2, \dots, \|R_P - \hat{R}_P\| \leq K_{11} \varepsilon_{11}$, which completes the proof. $\square$

Numerical Scheme

7.1. Derivation

Discretise $I = [0, T]$ with step $h = T/N$ and nodes $t_n = nh$; write $X_n \approx X(t_n)$ and $\Theta_n = \Theta(t_n, X_n)$. From (3.5) at $t = t_{n+1}$, approximating $\Theta(\tau, X(\tau))$ on each subinterval $[t_\sigma, t_{\sigma+1}]$ by the two-point Lagrange polynomial through $(t_{\sigma-1}, \Theta_{\sigma-1})$ and $(t_\sigma, \Theta_\sigma)$ and integrating gives the predictor

	$\hat{X}_{n+1} = X_0 + \frac{(1-\nu)\phi}{N(\nu)\Gamma(\phi)} t_{n+1}^{\phi-1} \Theta_n$	
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	$+ \frac{h^v}{\Gamma(v+2)} \sum_{\sigma=1}^n t_{\sigma}^{\phi-1} [P_{\sigma,n} \theta_{\sigma} - Q_{\sigma,n} \theta_{\sigma-1}] - R_{n+1}.$	(7.1)
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with weights

	$P_{\sigma,n} = (n+1-\sigma)^v (n+2+v-\sigma) - (n-\sigma)^v (n+2+2v-\sigma),$	(7.2)
	$Q_{\sigma,n} = (n+1-\sigma)^{v+1} - (n-\sigma)^v (n-\sigma+1+v),$	(7.3)
	$R_{n+1} = \frac{(1-v)\phi}{N(v)\Gamma(\phi)} t_{n+1}^{\phi-1} \theta_0 \left(1 + \frac{\theta_v t_{n+1}^v}{\Gamma(v+1)} \right).$	(7.4)

The sum in (7.1)–(7.4) starts at $\sigma = 1$ rather than $\sigma = 0$: for $\phi < 1$ the weight $t_{\sigma}^{\phi-1}$ diverges at $t_0 = 0$, and the standard resolution is to exclude this single, measure-zero node from the discretised sum, its contribution being already accounted for through the boundary term R_{n+1} .

Remark 4. The coefficient $Q_{\sigma,n}$ in (7.3) is the form obtained directly from integrating the two-point Lagrange interpolant against the fractional kernel, following the derivation of Toufik and Atangana [32]. A form with $(n+1-\sigma)^v$ in place of $(n+1-\sigma)^{v+1}$ appears in some secondary sources; that variant does not satisfy the recursion obtained from direct integration, and using it makes the scheme (7.1)–(7.5) numerically unstable irrespective of step size, since the resulting weights fail to decay appropriately as σ ranges over the history. We use (7.3) throughout, which we have verified numerically is stable and convergent for the parameter ranges considered below.

The corrector replaces the implicit θ_{n+1} by $\theta(t_{n+1}, \hat{X}_{n+1})$:

$$X_{n+1} = X_0 + \frac{(1-v)\phi}{N(v)\Gamma(\phi)} t_{n+1}^{\phi-1} \theta(t_{n+1}, \hat{X}_{n+1}) + \frac{h^v}{\Gamma(v+2)} \sum_{\sigma=1}^n t_{\sigma}^{\phi-1} [P_{\sigma,n} \theta_{\sigma} - Q_{\sigma,n} \theta_{\sigma-1}] - R_{n+1}. \quad (7.5)$$

Applying (7.5) to each of the eleven compartments in turn, and iterating over $n = 0, 1, \dots, N-1$, gives the scheme used in the simulations below, with $N(v) \equiv 1$ (consistent with $N(0) = N(1) = 1$):

	$S_{n+1} = S_0 + \frac{(1-v)\phi}{\Gamma(\phi)} t_{n+1}^{\phi-1} \theta_1(t_{n+1}, \hat{S}_{n+1})$	
	$+ \frac{h^v}{\Gamma(v+2)} \sum_{\sigma=1}^n t_{\sigma}^{\phi-1} [P_{\sigma,n} \theta_{1,\sigma} - Q_{\sigma,n} \theta_{1,\sigma-1}] - R_{n+1}.$	(7.6)
	$E_{p,n+1} = E_{p,0} + \frac{(1-v)\phi}{\Gamma(\phi)} t_{n+1}^{\phi-1} \theta_2(t_{n+1}, \hat{E}_{p,n+1})$	
	$+ \frac{h^v}{\Gamma(v+2)} \sum_{\sigma=1}^n t_{\sigma}^{\phi-1} [P_{\sigma,n} \theta_{2,\sigma} - Q_{\sigma,n} \theta_{2,\sigma-1}] - R_{n+1}.$	(7.7)
	$E_{pH,n+1} = E_{pH,0} + \frac{(1-v)\phi}{\Gamma(\phi)} t_{n+1}^{\phi-1} \theta_3(t_{n+1}, \hat{E}_{pH,n+1})$	
	$+ \frac{h^v}{\Gamma(v+2)} \sum_{\sigma=1}^n t_{\sigma}^{\phi-1} [P_{\sigma,n} \theta_{3,\sigma} - Q_{\sigma,n} \theta_{3,\sigma-1}] - R_{n+1}.$	(7.8)
	$E_{H,n+1} = E_{H,0} + \frac{(1-v)\phi}{\Gamma(\phi)} t_{n+1}^{\phi-1} \theta_4(t_{n+1}, \hat{E}_{H,n+1})$	
	$+ \frac{h^v}{\Gamma(v+2)} \sum_{\sigma=1}^n t_{\sigma}^{\phi-1} [P_{\sigma,n} \theta_{4,\sigma} - Q_{\sigma,n} \theta_{4,\sigma-1}] - R_{n+1}.$	(7.9)
	$I_{p,n+1} = I_{p,0} + \frac{(1-v)\phi}{\Gamma(\phi)} t_{n+1}^{\phi-1} \theta_5(t_{n+1}, \hat{I}_{p,n+1})$	

	$+ \frac{h^v}{\Gamma(v+2)} \sum_{\sigma=1}^n t_{\sigma}^{\phi-1} [P_{\sigma,n} \Theta_{5,\sigma} - Q_{\sigma,n} \Theta_{5,\sigma-1}] - R_{n+1}.$	(7.10)
	$I_{PH,n+1} = I_{PH,0} + \frac{(1-v)\phi}{\Gamma(\phi)} t_{n+1}^{\phi-1} \Theta_6(t_{n+1}, \hat{I}_{PH,n+1})$	
	$+ \frac{h^v}{\Gamma(v+2)} \sum_{\sigma=1}^n t_{\sigma}^{\phi-1} [P_{\sigma,n} \Theta_{6,\sigma} - Q_{\sigma,n} \Theta_{6,\sigma-1}] - R_{n+1}.$	(7.11)
	$I_{H,n+1} = I_{H,0} + \frac{(1-v)\phi}{\Gamma(\phi)} t_{n+1}^{\phi-1} \Theta_7(t_{n+1}, \hat{I}_{H,n+1})$	
	$+ \frac{h^v}{\Gamma(v+2)} \sum_{\sigma=1}^n t_{\sigma}^{\phi-1} [P_{\sigma,n} \Theta_{7,\sigma} - Q_{\sigma,n} \Theta_{7,\sigma-1}] - R_{n+1}.$	(7.12)
	$C_{n+1} = C_0 + \frac{(1-v)\phi}{\Gamma(\phi)} t_{n+1}^{\phi-1} \Theta_8(t_{n+1}, \hat{C}_{n+1})$	
	$+ \frac{h^v}{\Gamma(v+2)} \sum_{\sigma=1}^n t_{\sigma}^{\phi-1} [P_{\sigma,n} \Theta_{8,\sigma} - Q_{\sigma,n} \Theta_{8,\sigma-1}] - R_{n+1}.$	(7.13)
	$CA_{n+1} = CA_0 + \frac{(1-v)\phi}{\Gamma(\phi)} t_{n+1}^{\phi-1} \Theta_9(t_{n+1}, \hat{CA}_{n+1})$	
	$+ \frac{h^v}{\Gamma(v+2)} \sum_{\sigma=1}^n t_{\sigma}^{\phi-1} [P_{\sigma,n} \Theta_{9,\sigma} - Q_{\sigma,n} \Theta_{9,\sigma-1}] - R_{n+1}.$	(7.14)
	$A_{n+1} = A_0 + \frac{(1-v)\phi}{\Gamma(\phi)} t_{n+1}^{\phi-1} \Theta_{10}(t_{n+1}, \hat{A}_{n+1})$	
	$+ \frac{h^v}{\Gamma(v+2)} \sum_{\sigma=1}^n t_{\sigma}^{\phi-1} [P_{\sigma,n} \Theta_{10,\sigma} - Q_{\sigma,n} \Theta_{10,\sigma-1}] - R_{n+1}.$	(7.15)
	$R_{P,n+1} = R_{P,0} + \frac{(1-v)\phi}{\Gamma(\phi)} t_{n+1}^{\phi-1} \Theta_{11}(t_{n+1}, \hat{R}_{P,n+1})$	
	$+ \frac{h^v}{\Gamma(v+2)} \sum_{\sigma=1}^n t_{\sigma}^{\phi-1} [P_{\sigma,n} \Theta_{11,\sigma} - Q_{\sigma,n} \Theta_{11,\sigma-1}] - R_{n+1}.$	(7.16)

where $\Theta_{i,\sigma} = \Theta_i(t_{\sigma}, X_{\sigma})$ for brevity, and each R_{n+1} , $P_{\sigma,n}$, $Q_{\sigma,n}$ is as given in (7.2)–(7.4), evaluated with the Lipschitz constant appropriate to that compartment.

7.2. Results and discussion

Simulations use the parameters of Table 2, initial condition $S(0) = 600$, $E_P(0) = 170$, $E_{PH}(0) = 250$, $E_H(0) = 200$, $I_P(0) = 140$, $I_{PH}(0) = 140$, $I_H(0) = 160$, $C(0) = 60$, $CA(0) = 40$, $A(0) = 40$, $R_P(0) = 120$, time horizon $t \in [0, 100]$ days, step $h = 0.05$, fractional orders $v \in \{0.75, 0.80, 0.85, 0.90, 0.95\}$, and fractal dimensions $\phi \in \{0.90, 1.0\}$. Figures 2–12 present each of the eleven compartments in turn, with the five values of v overlaid in each panel and the two fractal dimensions shown as subgraphs (a) $\phi = 1.0$ and (b) $\phi = 0.90$ side by side for direct comparison.

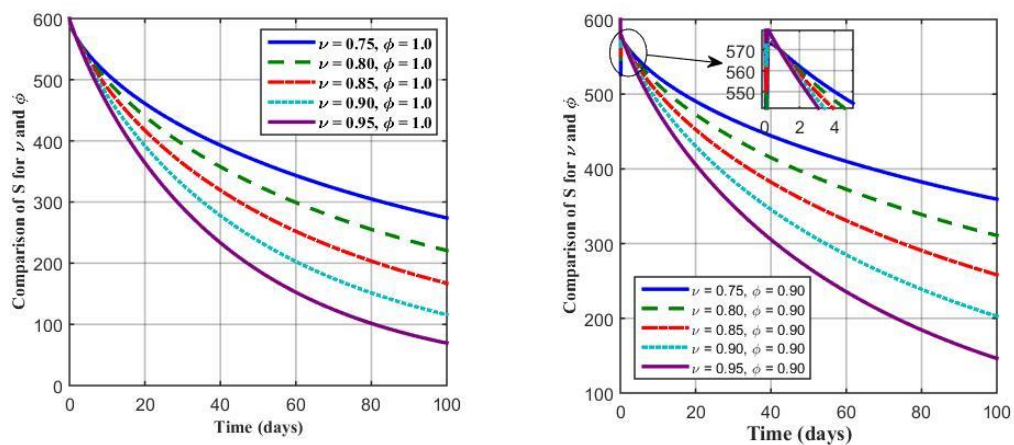


Figure 2: Time evolution of the susceptible population for $\nu \in \{0.75, 0.80, 0.85, 0.90, 0.95\}$, compared across fractal dimension $\phi = 1.0$ (left) and $\phi = 0.90$ (right).

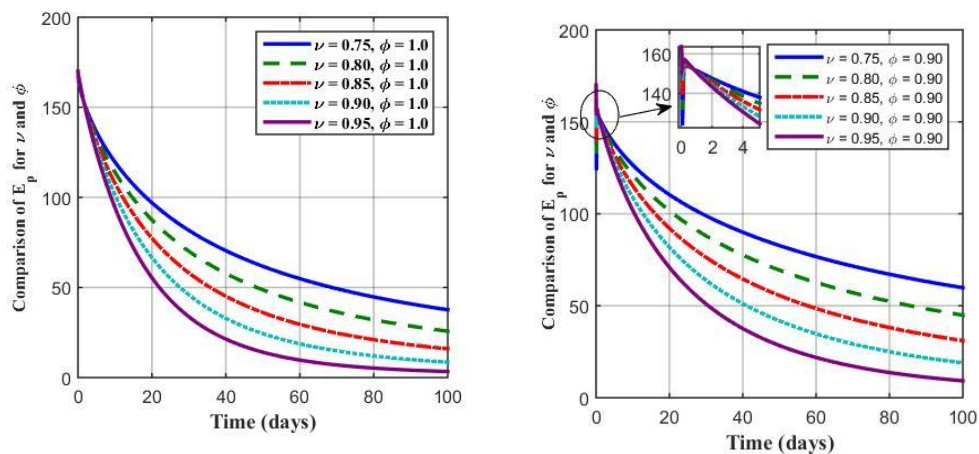


Figure 3: Time evolution of the HPV-exposed compartment for $\nu \in \{0.75, 0.80, 0.85, 0.90, 0.95\}$, compared across fractal dimension $\phi = 1.0$ (left) and $\phi = 0.90$ (right).

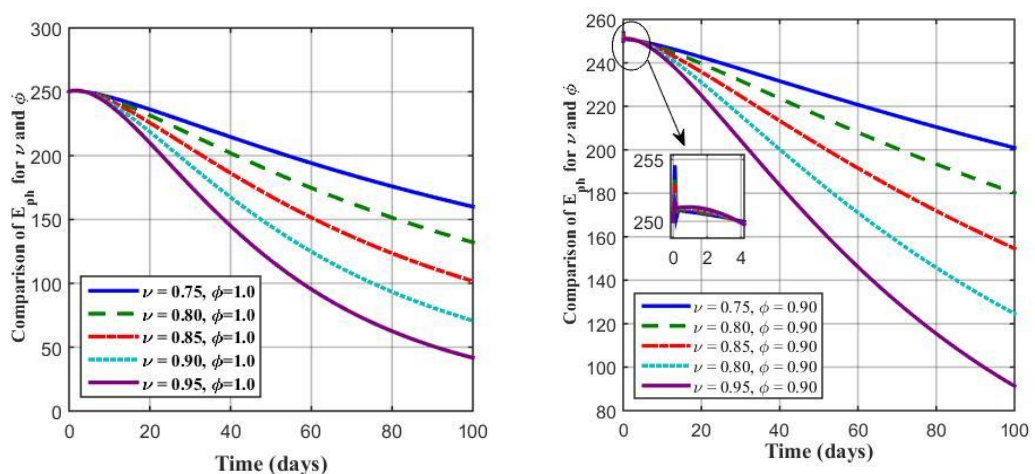


Figure 4: Time evolution of the co-exposed compartment for $\nu \in \{0.75, 0.80, 0.85, 0.90, 0.95\}$, compared across fractal dimension $\phi = 1.0$ (left) and $\phi = 0.90$ (right).

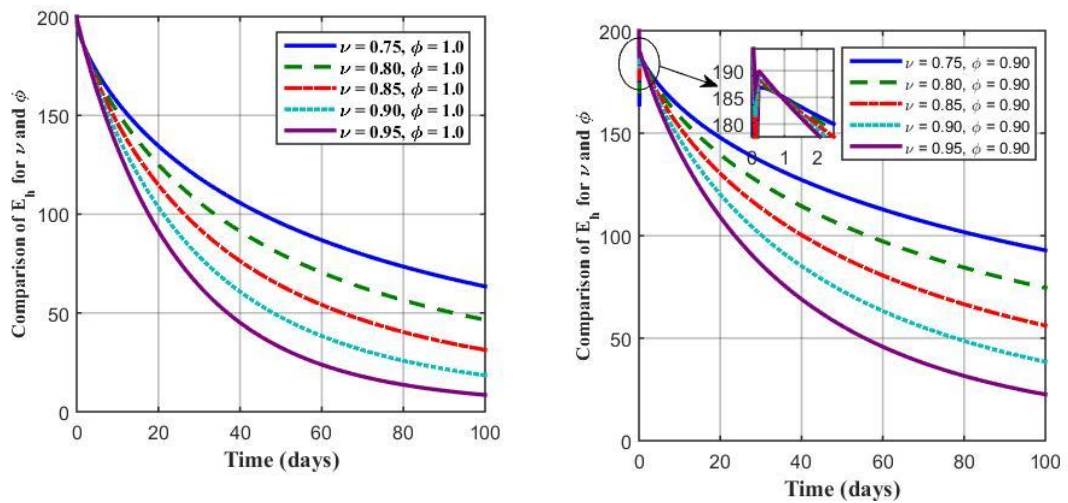


Figure 5: Time evolution of the HIV-exposed compartment for $\nu \in \{0.75, 0.80, 0.85, 0.90, 0.95\}$, compared across fractal dimension $\phi = 1.0$ (left) and $\phi = 0.90$ (right).

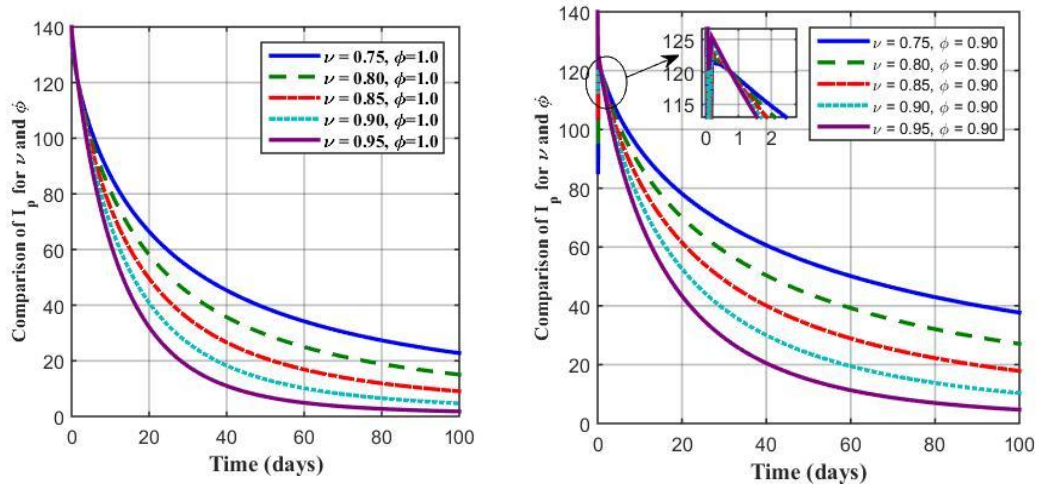


Figure 6: Time evolution of the HPV-infected population for $\nu \in \{0.75, 0.80, 0.85, 0.90, 0.95\}$, compared across fractal dimension $\phi = 1.0$ (left) and $\phi = 0.90$ (right).

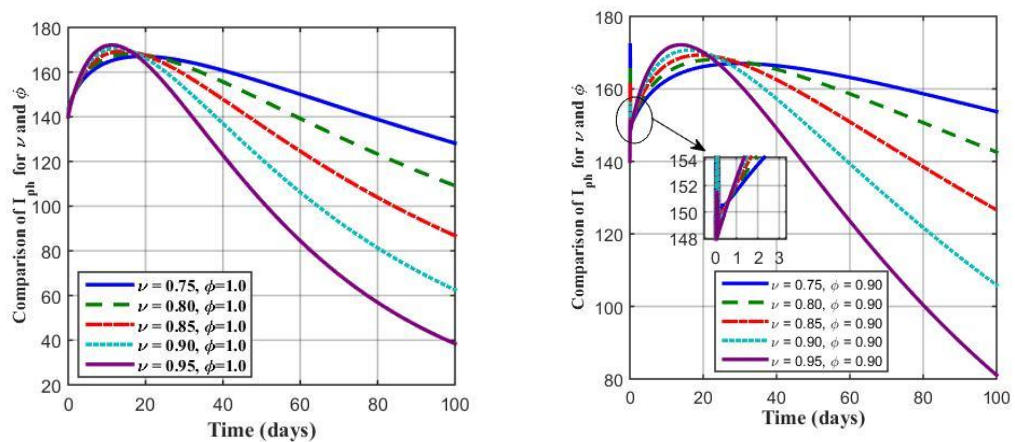


Figure 7: Time evolution of the co-infected compartment for $\nu \in \{0.75, 0.80, 0.85, 0.90, 0.95\}$, compared across fractal dimension $\phi = 1.0$ (left) and $\phi = 0.90$ (right).

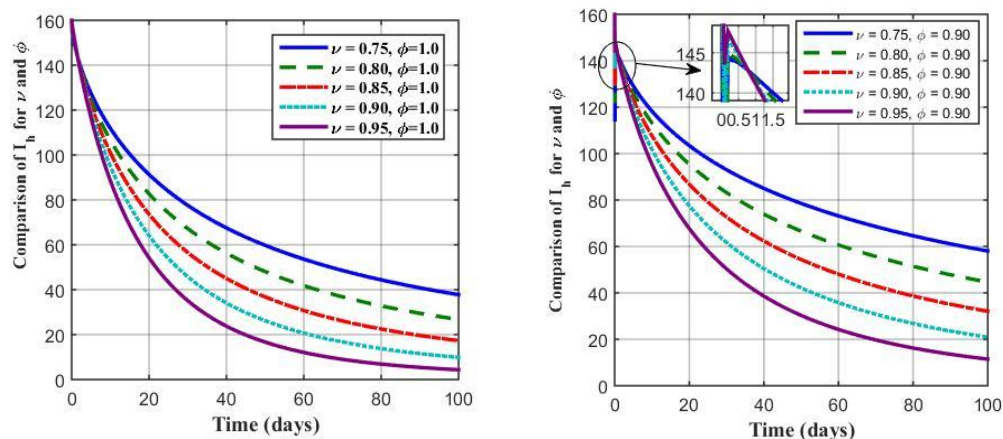


Figure 8: Time evolution of the HIV-infected class for $\nu \in \{0.75, 0.80, 0.85, 0.90, 0.95\}$, compared across fractal dimension $\phi = 1.0$ (left) and $\phi = 0.90$ (right).

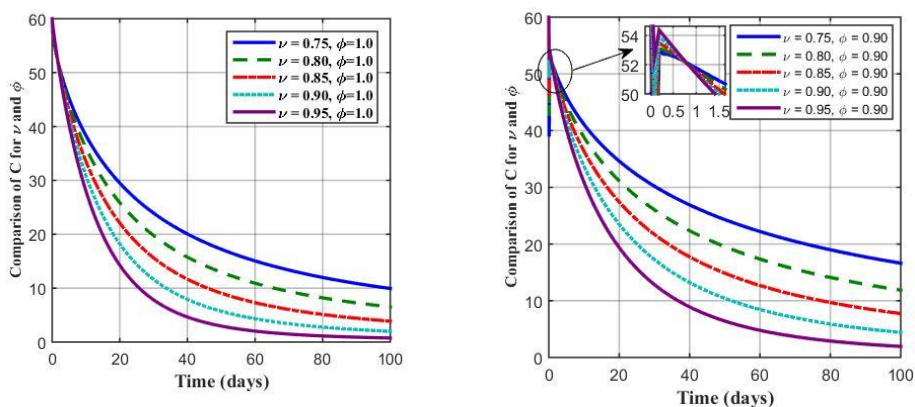


Figure 9: Time evolution of the cervical cancer compartment for $\nu \in \{0.75, 0.80, 0.85, 0.90, 0.95\}$, compared across fractal dimension $\phi = 1.0$ (left) and $\phi = 0.90$ (right).

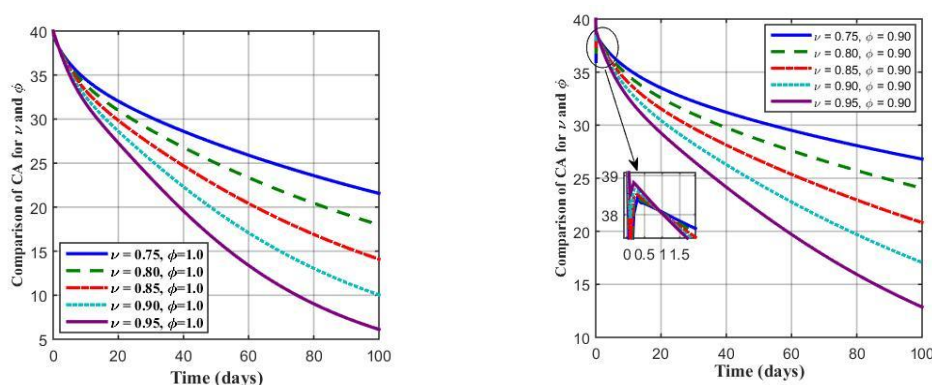


Figure 10: Time evolution of the co-disease compartment for $\nu \in \{0.75, 0.80, 0.85, 0.90, 0.95\}$, compared across fractal dimension $\phi = 1.0$ (left) and $\phi = 0.90$ (right).

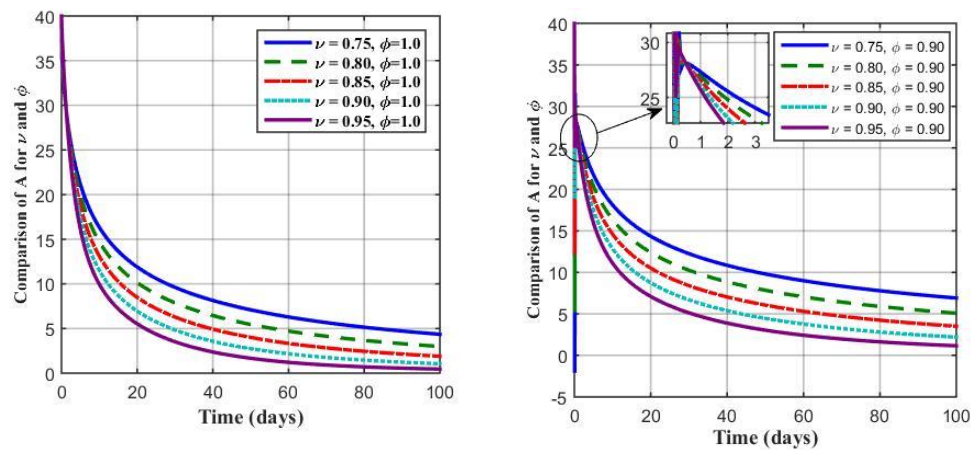


Figure 11: Time evolution of the AIDS compartment for $\nu \in \{0.75, 0.80, 0.85, 0.90, 0.95\}$, compared across fractal dimension $\phi = 1.0$ (left) and $\phi = 0.90$ (right).

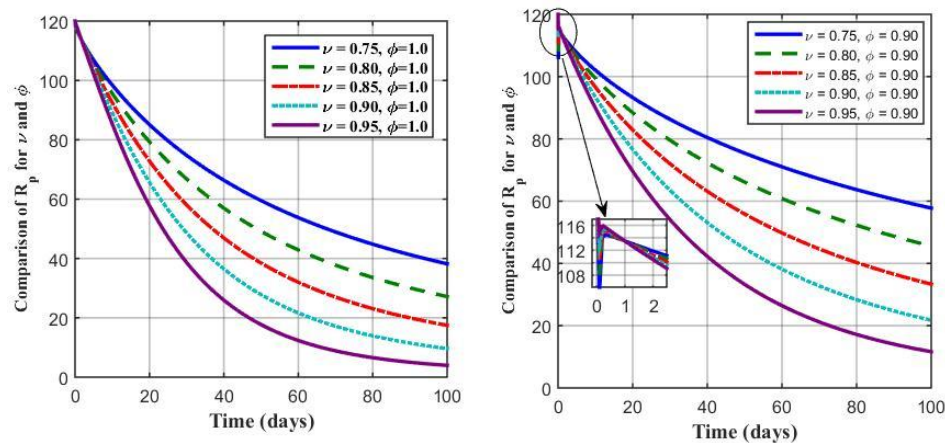


Figure 12: Time evolution of the recovered compartment for $\nu \in \{0.75, 0.80, 0.85, 0.90, 0.95\}$, compared across fractal dimension $\phi = 1.0$ (left) and $\phi = 0.90$ (right).

At $\phi = 1.0$ (subgraphs (a) of Figures 2–12), the susceptible population $S(t)$ declines from its initial value of 600, with smaller ν producing slower depletion – the stronger Mittag–Leffler memory at low ν retains individuals in the susceptible pool longer (Figure 2). The singly-exposed classes $E_p(t)$ and $E_H(t)$ decline without a pronounced transient (Figures 3, 5), while the co-exposed class $E_{pH}(t)$ holds closer to its initial level for longer before declining, consistent with dual exposure delaying progression out of that compartment (Figure 4). The HPV-infected class $I_p(t)$ decreases steadily under the sub-threshold reproduction number $R_0 \approx 0.154$ (Figure 6), whereas the co-infected class $I_{pH}(t)$ rises from its initial value of 140 to a peak around $t \approx 17$ days driven by inflow from I_p at rate λ_3 and from I_H at rate λ_4 – before declining; smaller ν both delays and raises this peak, giving a direct numerical read on how memory amplifies the HPV–HIV interaction (Figure 7). The HIV-infected class $I_H(t)$ mirrors $I_p(t)$ qualitatively (Figure 8). The cervical cancer and AIDS compartments $C(t)$ and $A(t)$ decline under their respective treatment rates $\theta_c = 0.100$ and $\psi = 0.300$, with lower ν again slowing convergence (Figures 9, 11), and the co-disease compartment $CA(t)$, requiring both conditions simultaneously, declines more slowly still (Figure 10). The recovered compartment $R_p(t)$ decreases monotonically as recovered individuals gradually deplete through natural mortality and waning immunity (Figure 12).

At $\phi = 0.90$ (subgraphs (b) of Figures 2–12), the same broad pattern holds in every compartment, but each curve shows a visibly different early-time profile: since the fractal weight $t^{\phi-1}$ is largest just after $t = 0$ and decays as t grows, its effect is concentrated in the first several days rather than distributed evenly across the simulation window, giving each curve a sharper initial departure from its starting value before settling into a trajectory that runs roughly parallel to the corresponding $\phi = 1.0$ curve for the remainder of $[0, 100]$. This is

clearest in the co-infected and co-disease compartments (Figures 7, 10), where the early-time separation between different values of ν is noticeably wider at $\phi = 0.90$ than at $\phi = 1.0$. This is the numerical signature of the fractal dimension acting as a genuinely separate control parameter from ν : ν governs the long-run rate of convergence, while ϕ reshapes the short-time transient. Taken together, the two-parameter (ν, ϕ) family gives a more detailed picture of HPV/HIV co-infection dynamics than either a classical integer-order model or a single-parameter fractional model would provide on its own

8. Conclusion

We formulated an eleven-compartment HPV/HIV co-infection system under the Atangana–Baleanu–Caputo fractal–fractional operator, proved existence and uniqueness of solutions via Banach’s fixed-point theorem, and established Ulam–Hyers stability with an explicit constant $K_i = \Phi/(1 - \Phi L_i)$ for each compartment. In deriving the two-step Lagrange predictor–corrector scheme used to simulate the system, we found and corrected a coefficient that, left as commonly quoted, renders the recursion numerically unstable; with the corrected coefficient the scheme is stable across the fractional orders and fractal dimensions considered. Simulations over $\nu \in \{0.75, 0.80, 0.85, 0.90, 0.95\}$ and $\phi \in \{0.90, 1.0\}$ show that ν chiefly controls the long-run rate of convergence toward the disease-free equilibrium while ϕ reshapes the early-time transient, giving a two-parameter account of how memory and fractal geometry jointly influence HPV/HIV co-infection dynamics. Extending this framework to optimal control of vaccination and antiretroviral therapy, to Lyapunov-based global stability, and to parameter estimation from field incidence data are natural next steps.

Declarations

Availability of data and materials

All data used in this study are given within the manuscript.

Conflict of interest

The authors declare that they have no competing interests.

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Authors’ contributions

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