

STABILITY OF A FRACTIONAL MULTI-STRAIN EPIDEMIC MODEL

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ABSTRACT. We investigate a fractional-order epidemic model with three competing strains, incorporating memory effects through the Caputo derivative of order α between zero and one. Strain one follows a saturated Holling type-II incidence, while strains two and three spread through bilinear mass-action contact. We establish well-posedness by proving existence, uniqueness, positivity, and boundedness of solutions within a compact invariant region. The strain-specific basic reproduction numbers are computed via the next-generation matrix method. Applying Matignon's criterion, we prove local asymptotic stability of the disease-free and monomorphic endemic equilibria. Suitable Lyapunov functions then establish their global asymptotic stability.

Keywords. Fractional-order system; multi-strain model; saturated incidence; basic reproduction number; local stability; global stability.

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1. INTRODUCTION

Multi-strain epidemic models constitute a valuable analytical framework for uncovering the mechanisms that drive the spread and evolution of infectious diseases across their distinct mutational forms and circulating variants. A number of pathogens do not propagate as a single homogeneous entity: tuberculosis [1], the human immunodeficiency virus (HIV) [2], COVID-19 [3], hepatitis B (HBV) [4], and hepatitis C (HCV) [5] are all known to sustain several co-circulating strains that either persist together or compete for the same host population. When more than one variant is present, the resulting interactions can reshape the qualitative behavior of the epidemic in substantial ways. It is precisely to capture these coupled dynamics that multi-strain compartmental formulations have been developed [6, 7, 8]. Among the many available compartmental structures, the SIR model—partitioning the population into susceptible (S), infected (I), and recovered (R) individuals—remains particularly well suited to directly transmitted

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infections for which the latent phase is either negligible or absorbed into other parameters. When several strains are taken into account, the infected class is split according to variant type, so that the classical SIR structure naturally extends into an $SI_1I_2\cdots I_nR$ form. First introduced by Kermack and McKendrick in 1927 [9], the SIR paradigm continues to underpin the estimation of key epidemiological quantities such as the basic reproduction number R_0 , the epidemic peak, and the herd-immunity threshold. Building on this foundation, a broad body of subsequent work has enriched the original formulation with multiple strains, age structure, vaccination programs, and optimal-control strategies [10, 11, 12, 13]. Vaccination, in particular, has repeatedly been shown to act as a decisive control lever: for compartmental descriptions that separate a vaccinated class, disease elimination becomes achievable once the immunization coverage exceeds a critical threshold, below which the infection persists in the population [14]. Interest in multi-strain models has grown considerably, largely because they make it possible to describe how competing pathogen variants coexist or exclude one another.

A central ingredient of any such model is the incidence function, which specifies the rate at which susceptible hosts acquire infection. Although the standard bilinear form βSI is ubiquitous in the literature [15], it often proves inadequate when transmission is nonlinear, when saturation arises at high infection levels, or when host behavior shifts during an outbreak. Motivated by these shortcomings, several authors have proposed more flexible nonlinear incidence terms that reflect realistic contagion processes more faithfully. In the present study, we adopt a mixed transmission structure for a three-strain system: strain 1 follows a saturated Holling type-II response that accounts for crowding and psychological effects among infectives, whereas strains 2 and 3 spread through classical bilinear mass-action contact. This choice lets the model span a meaningful range of transmission behaviors within a single tractable framework. Most epidemic models are cast in terms of ordinary integer-order differential equations. While such formulations reproduce the average course of an outbreak reasonably well, they tend to overlook the memory, hereditary characteristics, and delayed responses that are commonly observed in biological systems. Employing the Caputo fractional derivative addresses this gap by embedding memory directly into the governing equations, thereby yielding a more faithful representation of transmission dynamics [16, 17, 18, 19].

Guided by these considerations, this paper studies a fractional-order $SI_1I_2I_3R$ model in which each of the three strains propagates through its own incidence mechanism, with memory effects encoded via Caputo derivatives. We first establish the well-posedness of the system by proving the existence, uniqueness, positivity, and boundedness of its solutions. The strain-specific basic reproduction numbers are then computed through the next-generation matrix method, and the existence of both the disease-free and the endemic equilibria is analyzed. Their global asymptotic stability is subsequently investigated by means of suitably constructed Lyapunov functionals. Finally, numerical experiments illustrate how the fractional order shapes the speed at which trajectories approach their equilibria. To the best of our knowledge, comparatively little attention has been devoted to the combined role of fractional order and heterogeneous incidence rates

within three-strain epidemic settings. In line with the recent surge of interest in fractional-order descriptions of infectious-disease dynamics through a variety of analytical techniques [14, 20, 21, 22], the fractional differential system introduced below formalizes the dynamics of the proposed model.

The remainder of this paper is organized as follows. Section 2 recalls the basic notions of fractional calculus and the auxiliary results used throughout the analysis. Section 3 introduces the fractional-order multi-strain model and establishes its well-posedness, namely the existence, uniqueness, positivity, and boundedness of solutions. Section 4 determines the equilibria of the system and derives the strain-specific basic reproduction numbers through the next-generation matrix method. Section 5 is devoted to the local asymptotic stability of the disease-free and endemic equilibria, whereas Section 6 addresses their global asymptotic stability by means of suitable Lyapunov functions. Section 7 presents numerical simulations that illustrate the theoretical findings and assess the influence of the fractional order on the dynamics. Finally, Section 8 summarizes the main conclusions and outlines some directions for future work.

2. MATHEMATICAL PRELIMINARIES

Throughout the analysis we use the Caputo fractional derivative ${}^C D_t^\alpha$ of order $\alpha \in (0, 1]$, together with the associated one- and two-parameter Mittag-Leffler functions; we refer to [23] for their definitions and basic properties. Existence and uniqueness of solutions will be obtained through the Banach fixed-point theorem [24]. Our global stability arguments rely on the following Volterra-type fractional comparison property: for any continuous and differentiable function $x(t) \in \mathbb{R}^+$ and any $x^* \in \mathbb{R}^+$,

$${}^C D_t^\alpha \left[x(t) - x^* - x^* \ln \left(\frac{x(t)}{x^*} \right) \right] \leq \left(1 - \frac{x^*}{x(t)} \right) {}^C D_t^\alpha x(t). \quad (2.1)$$

3. MODEL FORMULATION AND WELL-POSEDNESS

Based on the transmission structure, we define the following non-linear fractional system:

$$\begin{cases} {}^C D_t^\alpha S(t) = \Lambda - \frac{\beta_1 S(t) I_1(t)}{1 + a I_1(t)} - \beta_2 S(t) I_2(t) - \beta_3 S(t) I_3(t) - \mu S(t) \\ {}^C D_t^\alpha I_1(t) = \frac{\beta_1 S(t) I_1(t)}{1 + a I_1(t)} - (\sigma_1 + \mu) I_1(t) \\ {}^C D_t^\alpha I_2(t) = \beta_2 S(t) I_2(t) - (\sigma_2 + \mu) I_2(t) \\ {}^C D_t^\alpha I_3(t) = \beta_3 S(t) I_3(t) - (\sigma_3 + \mu) I_3(t) \\ {}^C D_t^\alpha R(t) = \sigma_1 I_1(t) + \sigma_2 I_2(t) + \sigma_3 I_3(t) - \mu R(t) \end{cases} \quad (3.1)$$

Accordingly, we investigate the system under strictly positive initial conditions, representing the realistic introduction of multiple competing pathogen strains

into a entirely susceptible population:

$$S(0) = S_0 > 0, \quad I_1(0) = I_{10} > 0, \quad I_2(0) = I_{20} > 0, \quad I_3(0) = I_{30} > 0, \quad R(0) = R_0 > 0. \quad (3.2)$$

3.1. Existence and uniqueness. To guarantee that the proposed fractional-order multi-strain epidemic model is mathematically well-behaved, we must establish the existence and uniqueness of its global solution. We utilize the fixed-point theorem approach combined with the properties of fractional calculus in a suitable Banach space, following the framework established by Li et El-Shahed.

Let us rewrite the main fractional system in a more compact operator form. For $t \in [0, T]$, where $T > 0$ denotes a finite time horizon, we define the state vector $X(t) = (S(t), I_1(t), I_2(t), I_3(t), R(t))^T \in \mathbb{R}^5$. The system can be expressed as:

$${}^C D_t^\alpha X(t) = G(X(t)), \quad X(0) = X_0 = (S_0, I_{10}, I_{20}, I_{30}, R_0)^T \quad (3.3)$$

where the vector-valued function $G(X) = (G_1(X), G_2(X), G_3(X), G_4(X), G_5(X))^T$ is defined by the components:

$$\begin{cases} G_1(X) = \Lambda - \frac{\beta_1 S I_1}{1 + a I_1} - \beta_2 S I_2 - \beta_3 S I_3 - \mu S \\ G_2(X) = \frac{\beta_1 S I_1}{1 + a I_1} - (\sigma_1 + \mu) I_1 \\ G_3(X) = \beta_2 S I_2 - (\sigma_2 + \mu) I_2 \\ G_4(X) = \beta_3 S I_3 - (\sigma_3 + \mu) I_3 \\ G_5(X) = \sigma_1 I_1 + \sigma_2 I_2 + \sigma_3 I_3 - \mu R \end{cases} \quad (3.4)$$

By applying the Riemann-Liouville fractional integral operator I^α to both sides of equation (3.3) and incorporating the initial state, we transform the fractional differential system into an equivalent Volterra-type integral equation:

$$X(t) = X(0) + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha-1} G(X(\tau)) d\tau \quad (3.5)$$

Let us consider the space of continuous functions $C([0, T], \mathbb{R}^5)$ equipped with the uniform norm:

$$\|X\| = \sup_{t \in [0, T]} (|S(t)| + |I_1(t)| + |I_2(t)| + |I_3(t)| + |R(t)|) \quad (3.6)$$

It is well established that $(C([0, T], \mathbb{R}^5), \|\cdot\|)$ constitutes a complete Banach space.

Lemma 3.1. *The vector function $G(X)$ defined in (3.4) satisfies the Lipschitz condition with respect to X within the bounded region*

$$\Omega = \left\{ (S, I_1, I_2, I_3, R) \in \mathbb{R}_+^5 : S(t) + I_1(t) + I_2(t) + I_3(t) + R(t) \leq \frac{\Lambda}{\mu} + \epsilon, \quad \forall \epsilon > 0 \right\}. \quad (3.7)$$

Proof. Let $X, \bar{X} \in \Omega$. Since Ω is compact, every state variable is bounded by $M = \frac{\Lambda}{\mu} + \epsilon$. Using the triangle inequality, the boundedness of the states, and the estimate

$$\left| \frac{SI_1}{1 + aI_1} - \frac{\bar{S}\bar{I}_1}{1 + a\bar{I}_1} \right| \leq M|S - \bar{S}| + M|I_1 - \bar{I}_1| + aM^2|S - \bar{S}|$$

for the saturated term, each component G_i satisfies a Lipschitz inequality. Summing these estimates, there exists a constant $L > 0$ such that

$$\|G(X) - G(\bar{X})\| \leq L\|X - \bar{X}\|, \quad (3.8)$$

so G is globally Lipschitz continuous on Ω . $\square$

Theorem 3.2. *The fractional-order multi-strain model (3.1) possesses a unique solution in $C([0, T], \mathbb{R}^5)$ for any finite time $T > 0$, provided that:*

$$\frac{LT^\alpha}{\Gamma(\alpha + 1)} < 1 \quad (3.9)$$

where L is the Lipschitz constant derived in Lemma 3.1.

Proof. Define $\Psi : C([0, T], \mathbb{R}^5) \rightarrow C([0, T], \mathbb{R}^5)$ from the integral form (3.5) by

$$\Psi[X(t)] = X(0) + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha-1} G(X(\tau)) d\tau. \quad (3.10)$$

Using the Lipschitz property (3.8) and $\int_0^t (t - \tau)^{\alpha-1} d\tau = \frac{t^\alpha}{\alpha}$, we obtain

$$\|\Psi[X] - \Psi[\bar{X}]\| \leq \frac{LT^\alpha}{\Gamma(\alpha + 1)} \|X - \bar{X}\|. \quad (3.11)$$

Under condition (3.9), Ψ is a contraction, and the Banach fixed-point theorem [23] yields a unique global solution on $[0, T]$. $\square$

3.2. Positivity and boundedness. Before conducting the qualitative analysis of the multi-strain dynamics, we recall some fundamental definitions and establish essential lemmas regarding the positivity and comparison principles within the framework of Caputo fractional calculus.

Lemma 3.3. *Let $\alpha \in (0, 1]$. Suppose that $x(t) \in C([a, b])$ and its Caputo fractional derivative satisfies ${}^C D_t^\alpha x(t) \in C([a, b])$. If $x(a) \geq 0$ and there exists a continuous function $g(x)$ such that the inequality ${}^C D_t^\alpha x(t) \geq g(x(t))x(t)$ holds for all $t \in [a, b]$, then $x(t) \geq 0$ for all $t \in [a, b]$.*

Lemma 3.4. *Let $\alpha \in (0, 1]$ and consider the linear fractional differential inequality given by ${}^C D_t^\alpha x(t) \leq \Lambda - \mu x(t)$ subject to the initial condition $x(0) = x_0$. Then, the solution satisfies:*

$$x(t) \leq x(0)E_{\alpha,1}(-\mu t^\alpha) + \Lambda t^\alpha E_{\alpha,\alpha+1}(-\mu t^\alpha) \quad (3.12)$$

Furthermore, if the initial state satisfies $x(0) \leq \frac{\Lambda}{\mu}$, then $x(t) \leq \frac{\Lambda}{\mu}$ holds for all $t \geq 0$.

3.3. Positivity. In this subsection, we demonstrate the mathematical well-posedness of the proposed fractional-order multi-strain epidemic model. To establish biological plausibility, the state variables must remain non-negative and globally bounded at all times, thereby preserving the physical meaning of the populations.

Theorem 3.5. *If the initial conditions satisfy $(S_0, I_{10}, I_{20}, I_{30}, R_0) \in \mathbb{R}_+^5$, then all solution trajectories $(S(t), I_1(t), I_2(t), I_3(t), R(t))$ of the fractional-order system remain confined within the non-negative orthant $\mathbb{R}_+^5$ for all $t \geq 0$.*

Proof. To establish the invariance of the non-negative domain, we evaluate the behavior of the Caputo fractional derivative for each state variable along the boundaries of the positive hyper-octant $\mathbb{R}_+^5$:

$$\left\{ \begin{array}{l} {}^C D_t^\alpha S(t) \Big|_{S=0} = \Lambda > 0 \\ {}^C D_t^\alpha I_1(t) \Big|_{I_1=0} = 0 \geq 0 \\ {}^C D_t^\alpha I_2(t) \Big|_{I_2=0} = 0 \geq 0 \\ {}^C D_t^\alpha I_3(t) \Big|_{I_3=0} = 0 \geq 0 \\ {}^C D_t^\alpha R(t) \Big|_{R=0} = \sigma_1 I_1(t) + \sigma_2 I_2(t) + \sigma_3 I_3(t) \geq 0 \text{ since } I_1(t), I_2(t), I_3(t) \geq 0 \end{array} \right.$$

Furthermore, the vector field defined by the right-hand side of our system is locally Lipschitz continuous on $\mathbb{R}_+^5$. By invoking Lemma 3.3 the fractional positivity principle established by Li and Zhang, no solution trajectory originating inside the interior of $\mathbb{R}_+^5$ can cross any of the bounding coordinate hyperplanes. Hence, the non-negative domain $\mathbb{R}_+^5$ is completely invariant for all $t \geq 0$. $\square$

3.4. Boundedness.

Theorem 3.6. *All solution trajectories of the multi-strain system initiating in the positive region $\mathbb{R}_+^5$ are uniformly bounded within a compact invariant space.*

Proof. Let us define a continuous function representing the total host population over time:

$$\phi(t) = S(t) + I_1(t) + I_2(t) + I_3(t) + R(t) \quad (3.13)$$

Applying the linear Caputo fractional differential operator ${}^C D_t^\alpha$ to $\phi(t)$ yields the sum of the system equations:

$${}^C D_t^\alpha \phi(t) = {}^C D_t^\alpha S(t) + {}^C D_t^\alpha I_1(t) + {}^C D_t^\alpha I_2(t) + {}^C D_t^\alpha I_3(t) + {}^C D_t^\alpha R(t) \quad (3.14)$$

Substituting the model definitions, all non-linear disease transmission terms (incidence rates) and recovery pathways ($\sigma_i I_i$) cancel out exactly by algebraic balance:

$${}^C D_t^\alpha \phi(t) = \Lambda - \mu S(t) - \mu I_1(t) - \mu I_2(t) - \mu I_3(t) - \mu R(t) \quad (3.15)$$

Factoring out the natural mortality rate μ , we obtain the following fractional differential equation:

$${}^C D_t^\alpha \phi(t) + \mu \phi(t) = \Lambda \quad (3.16)$$

According to Lemma 3.4, the explicit analytic solution of equation (3.16) can be evaluated using the one-parameter and two-parameter Mittag-Leffler functions:

$$\phi(t) = \phi(0)E_{\alpha,1}(-\mu t^\alpha) + \Lambda t^\alpha E_{\alpha,\alpha+1}(-\mu t^\alpha) \quad (3.17)$$

where $\phi(0) = S_0 + I_{10} + I_{20} + I_{30} + R_0$ represents the total initial population size.

By applying the asymptotic expansion properties of Mittag-Leffler functions as $t \rightarrow \infty$, we know that $E_{\alpha,1}(-\mu t^\alpha) \rightarrow 0$ and $\Lambda t^\alpha E_{\alpha,\alpha+1}(-\mu t^\alpha) \rightarrow \frac{\Lambda}{\mu}$. Thus, we obtain the ultimate upper bound:

$$\lim_{t \rightarrow \infty} \phi(t) \leq \frac{\Lambda}{\mu} \quad (3.18)$$

Consequently, all solutions entering the positive domain $\mathbb{R}_+^5$ are bounded inside the biologically feasible and mathematically invariant region Ω which is defined in (3.7). This concludes the proof that the region Ω is a positively invariant set for the system. $\square$

4. EQUILIBRIUM AND BASIC REPRODUCTION NUMBERS

4.1. Disease-free equilibrium. In the total absence of infections ($I_1 = I_2 = I_3 = 0$), the system collapses to the Disease-Free Equilibrium (DFE), denoted by:

$$E_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0 \right) \quad (4.1)$$

4.2. Evaluation of R_0 . Using the next-generation matrix framework, we isolate the transmission matrix F and the transition matrix V computed at E_0 for the compartments $(I_1, I_2, I_3)^T$:

$$F = \begin{pmatrix} \beta_1 S_0 & 0 & 0 \\ 0 & \beta_2 S_0 & 0 \\ 0 & 0 & \beta_3 S_0 \end{pmatrix}, \quad V = \begin{pmatrix} \sigma_1 + \mu & 0 & 0 \\ 0 & \sigma_2 + \mu & 0 \\ 0 & 0 & \sigma_3 + \mu \end{pmatrix} \quad (4.2)$$

where $S_0 = \frac{\Lambda}{\mu}$. The next-generation matrix is FV^{-1} :

$$FV^{-1} = \begin{pmatrix} \frac{\beta_1 \Lambda}{\mu(\sigma_1 + \mu)} & 0 & 0 \\ 0 & \frac{\beta_2 \Lambda}{\mu(\sigma_2 + \mu)} & 0 \\ 0 & 0 & \frac{\beta_3 \Lambda}{\mu(\sigma_3 + \mu)} \end{pmatrix} \quad (4.3)$$

The spectral radius yields the strain-specific basic reproduction numbers:

$$R_{0,1} = \frac{\beta_1 \Lambda}{\mu(\sigma_1 + \mu)}, \quad R_{0,2} = \frac{\beta_2 \Lambda}{\mu(\sigma_2 + \mu)}, \quad R_{0,3} = \frac{\beta_3 \Lambda}{\mu(\sigma_3 + \mu)} \quad (4.4)$$

The overall basic reproduction number is $R_0 = \max\{R_{0,1}, R_{0,2}, R_{0,3}\}$.

4.3. Endemic equilibrium. To determine the steady states where the disease persists, we set all fractional derivatives in (3.1) to zero, yielding the following algebraic system:

$$\Lambda - \frac{\beta_1 S I_1}{1 + a I_1} - \beta_2 S I_2 - \beta_3 S I_3 - \mu S = 0 \quad (4.5)$$

$$I_1 \left(\frac{\beta_1 S}{1 + a I_1} - (\sigma_1 + \mu) \right) = 0 \quad (4.6)$$

$$I_2 (\beta_2 S - (\sigma_2 + \mu)) = 0 \quad (4.7)$$

$$I_3 (\beta_3 S - (\sigma_3 + \mu)) = 0 \quad (4.8)$$

$$\sigma_1 I_1 + \sigma_2 I_2 + \sigma_3 I_3 - \mu R = 0 \quad (4.9)$$

4.3.1. Monomorphic endemic equilibrium. We first consider the strain-1 monomorphic equilibrium E_1^* . Setting $I_2 = 0$ and $I_3 = 0$ with $I_1 > 0$, equation (4.6) gives $\frac{\beta_1 S^*}{1 + a I_1^*} = \sigma_1 + \mu$, so that $S^* = \frac{\sigma_1 + \mu}{\beta_1} (1 + a I_1^*)$. Substituting $I_2 = 0$, $I_3 = 0$ and $\frac{\beta_1 S^* I_1^*}{1 + a I_1^*} = (\sigma_1 + \mu) I_1^*$ into (4.5) yields $\Lambda - (\sigma_1 + \mu) I_1^* - \mu \frac{\sigma_1 + \mu}{\beta_1} (1 + a I_1^*) = 0$. Multiplying by β_1 leads to $\beta_1 \Lambda - \mu (\sigma_1 + \mu) = (\sigma_1 + \mu) (\beta_1 + a \mu) I_1^*$, and dividing by $\mu (\sigma_1 + \mu)$ gives $R_{0,1} - 1 = \frac{\beta_1 + a \mu}{\mu} I_1^*$. Hence

$$I_1^* = \frac{\mu (R_{0,1} - 1)}{\beta_1 + a \mu}, \quad S^* = \frac{\sigma_1 + \mu}{\beta_1} (1 + a I_1^*), \quad R^* = \frac{\sigma_1 I_1^*}{\mu}, \quad (4.10)$$

so that $E_1^* = \left(\frac{\sigma_1 + \mu}{\beta_1} (1 + a I_1^*), \frac{\mu (R_{0,1} - 1)}{\beta_1 + a \mu}, 0, 0, \frac{\sigma_1 I_1^*}{\mu} \right)$ exists uniquely if and only if $R_{0,1} > 1$.

For the strain-2 monomorphic equilibrium E_2^* , setting $I_1 = 0$ and $I_3 = 0$ with $I_2 > 0$, equation (4.7) gives $S^* = \frac{\sigma_2 + \mu}{\beta_2} = \frac{S_0}{R_{0,2}}$. Substituting into (4.5) and dividing by $(\sigma_2 + \mu)$ yields $I_2^* = \frac{\mu}{\beta_2} (R_{0,2} - 1)$. Thus $E_2^* = \left(\frac{S_0}{R_{0,2}}, 0, \frac{\mu}{\beta_2} (R_{0,2} - 1), 0, \frac{\sigma_2 I_2^*}{\mu} \right)$ exists if $R_{0,2} > 1$.

By a symmetric derivation with $I_1 = 0$, $I_2 = 0$ and $I_3 > 0$, the strain-3 monomorphic equilibrium is $E_3^* = \left(\frac{S_0}{R_{0,3}}, 0, 0, \frac{\mu}{\beta_3} (R_{0,3} - 1), \frac{\sigma_3 I_3^*}{\mu} \right)$, where $S^* = \frac{\sigma_3 + \mu}{\beta_3} = \frac{S_0}{R_{0,3}}$ and $I_3^* = \frac{\mu}{\beta_3} (R_{0,3} - 1)$; it exists uniquely if and only if $R_{0,3} > 1$.

4.3.2. Analysis of the coexistence equilibrium (E_{123}^*). For a total coexistence state, we assume $I_1 > 0$, $I_2 > 0$, and $I_3 > 0$ simultaneously. From equations (4.7) and (4.8), the conditions $S^* = \frac{\sigma_2 + \mu}{\beta_2}$ and $S^* = \frac{\sigma_3 + \mu}{\beta_3}$ must hold at the same time. This implies that total coexistence is mathematically impossible unless the biological parameters satisfy the strict identity

$$\frac{\sigma_2 + \mu}{\beta_2} = \frac{\sigma_3 + \mu}{\beta_3}, \quad \text{i.e.} \quad R_{0,2} = R_{0,3}. \quad (4.11)$$

In any real epidemiological scenario where $R_{0,2} \neq R_{0,3}$, competitive exclusion occurs and the strain with the lower fitness is driven to extinction; when $R_{0,2} = R_{0,3}$, a coexistence continuum can be derived as follows.

Since $I_1 > 0$, equation (4.6) must also hold. Substituting $S^* = \frac{\sigma_2 + \mu}{\beta_2}$ into (4.6) gives $1 + aI_1^* = \frac{\beta_1(\sigma_2 + \mu)}{\beta_2(\sigma_1 + \mu)} = \frac{R_{0,1}}{R_{0,2}}$, so that

$$I_1^* = \frac{1}{a} \left(\frac{R_{0,1}}{R_{0,2}} - 1 \right), \quad (4.12)$$

which is positive if and only if $R_{0,1} > R_{0,2}$.

Substituting S^* and I_1^* into the balance equation (4.5), and using $\beta_3 S^* = \sigma_3 + \mu$ from the coexistence conditions, we obtain after dividing by $(\sigma_2 + \mu)$ the coexistence line

$$I_2^* + \left(\frac{\sigma_3 + \mu}{\sigma_2 + \mu} \right) I_3^* = \frac{\mu}{\beta_2} (R_{0,2} - 1) - \frac{\sigma_1 + \mu}{a(\sigma_2 + \mu)} \left(\frac{R_{0,1}}{R_{0,2}} - 1 \right). \quad (4.13)$$

Hence, if $R_{0,2} = R_{0,3}$ and the right-hand side is strictly positive, there exists an infinite number of coexistence states lying on a line segment; otherwise, competitive exclusion prevails.

5. LOCAL STABILITY ANALYSIS

Theorem 5.1. *The disease-free equilibrium E_0 of the fractional-order system is locally asymptotically stable if $R_0 = \max\{R_{0,1}, R_{0,2}, R_{0,3}\} < 1$, and unstable if $R_0 > 1$.*

Proof. Evaluating the Jacobian matrix of the system at the disease-free equilibrium $E_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0 \right)$ yields the following upper block-triangular matrix:

$$J(E_0) = \begin{pmatrix} -\mu & -\frac{\beta_1 \Lambda}{\mu} & -\frac{\beta_2 \Lambda}{\mu} & -\frac{\beta_3 \Lambda}{\mu} & 0 \\ 0 & \frac{\beta_1 \Lambda}{\mu} - (\sigma_1 + \mu) & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_2 \Lambda}{\mu} - (\sigma_2 + \mu) & 0 & 0 \\ 0 & 0 & 0 & \frac{\beta_3 \Lambda}{\mu} - (\sigma_3 + \mu) & 0 \\ 0 & \sigma_1 & \sigma_2 & \sigma_3 & -\mu \end{pmatrix} \quad (5.1)$$

The eigenvalues of $J(E_0)$ are determined by its diagonal entries, which are given by:

$$\begin{aligned} \lambda_1 &= -\mu, \\ \lambda_2 &= -\mu, \\ \lambda_3 &= (\sigma_1 + \mu)(R_{0,1} - 1), \\ \lambda_4 &= (\sigma_2 + \mu)(R_{0,2} - 1), \\ \lambda_5 &= (\sigma_3 + \mu)(R_{0,3} - 1) \end{aligned}$$

If $R_0 = \max\{R_{0,1}, R_{0,2}, R_{0,3}\} < 1$, then all the eigenvalues λ_i ($i = 1, \dots, 5$) are strictly negative real numbers. Consequently, their arguments in the complex plane satisfy:

$$|\arg(\lambda_i)| = \pi > \frac{\alpha\pi}{2}, \quad \forall i \in \{1, 2, 3, 4, 5\} \quad (5.2)$$

According to Matignon's stability criterion for fractional-order systems [25], the DFE E_0 is locally asymptotically stable. Conversely, if $R_0 > 1$, there exists at least one eigenvalue (for instance, $\lambda_3 > 0$ if $R_{0,1} > 1$) which is a positive real number, meaning $|\arg(\lambda_i)| = 0 < \frac{\alpha\pi}{2}$. Thus, the DFE E_0 becomes unstable. $\square$

Theorem 5.2. *The Strain 1 monomorphic endemic equilibrium $E_1^* = (S^*, I_1^*, 0, 0, R^*)$ is locally asymptotically stable if $R_{0,1} > 1$, $R_{0,2} < \frac{S_0}{S^*}$, and $R_{0,3} < \frac{S_0}{S^*}$.*

Proof. Linearizing at E_1^* , the Jacobian is block-triangular. Two eigenvalues decouple directly,

$$\lambda_3 = (\sigma_2 + \mu) \left(\frac{S^*}{S_0} R_{0,2} - 1 \right), \quad \lambda_4 = (\sigma_3 + \mu) \left(\frac{S^*}{S_0} R_{0,3} - 1 \right), \quad (5.3)$$

which are strictly negative when $R_{0,2} < \frac{S_0}{S^*}$ and $R_{0,3} < \frac{S_0}{S^*}$, together with $\lambda_5 = -\mu < 0$. The remaining (S, I_1) block reads

$$J_1 = \begin{pmatrix} -A - \mu & -B \\ A & B - (\sigma_1 + \mu) \end{pmatrix}, \quad (5.4)$$

where $A = \frac{\beta_1 I_1^*}{1 + a I_1^*}$ and $B = \frac{\beta_1 S^*}{(1 + a I_1^*)^2}$. Since $\frac{\beta_1 S^*}{1 + a I_1^*} = \sigma_1 + \mu$, we have $B < \sigma_1 + \mu$, hence $\text{Tr}(J_1) = -A - \mu + B - (\sigma_1 + \mu) < 0$ and $\text{Det}(J_1) = A(\sigma_1 + \mu) + \mu(\sigma_1 + \mu - B) > 0$. By the Routh–Hurwitz criterion, both eigenvalues have negative real parts, so all arguments exceed $\frac{\alpha\pi}{2}$ and E_1^* is locally asymptotically stable. $\square$

Theorem 5.3. *The strain-2 endemic equilibrium E_2^* is locally asymptotically stable if $R_{0,2} > 1$, $R_{0,1} < R_{0,2}$, and $R_{0,3} < R_{0,2}$.*

Proof. Linearizing at E_2^* and using $\beta_2 S_2^* = \sigma_2 + \mu$, two eigenvalues decouple from the I_1 and I_3 directions,

$$\lambda_1 = (\sigma_1 + \mu) \left(\frac{R_{0,1}}{R_{0,2}} - 1 \right), \quad \lambda_2 = (\sigma_3 + \mu) \left(\frac{R_{0,3}}{R_{0,2}} - 1 \right), \quad (5.5)$$

which are strictly negative under $R_{0,1} < R_{0,2}$ and $R_{0,3} < R_{0,2}$, together with $\lambda_3 = -\mu < 0$. The remaining (S, I_2) block gives the characteristic equation $\lambda^2 + a_1 \lambda + a_2 = 0$ with

$$a_1 = \beta_2 I_2^* + \mu > 0, \quad a_2 = \beta_2^2 S_2^* I_2^* > 0. \quad (5.6)$$

By the Routh–Hurwitz criterion the two remaining roots have negative real parts, so all five eigenvalues satisfy $|\arg(\lambda_i)| > \frac{\alpha\pi}{2}$ and E_2^* is locally asymptotically stable. $\square$

And, by symmetry of the system dynamics, the strain-3 monomorphic equilibrium E_3^* is locally asymptotically stable if $R_{0,3} > 1$, $R_{0,1} < R_{0,3}$, and $R_{0,2} < R_{0,3}$.

The total coexistence state represents a polymorphic scenario E_{123}^* where all three pathogen strains persist simultaneously inside the host population. At this point, the state variables satisfy the simultaneous constraints:

$$\frac{\beta_1 S^*}{1 + a I_1^*} = \sigma_1 + \mu, \quad \beta_2 S^* = \sigma_2 + \mu, \quad \beta_3 S^* = \sigma_3 + \mu \quad (5.7)$$

Theorem 5.4. *Local stability of the coexistence equilibrium* Let $R_{0,1} > 1$, $R_{0,2} > 1$, and $R_{0,3} > 1$ hold, ensuring the existence and biological feasibility of the multi-strain coexistence equilibrium $E_{123}^* \in \text{int}(\Omega)$. Then, E_{123}^* is locally asymptotically stable if the coefficients of its characteristic polynomial satisfy the fractional Routh-Hurwitz conditions:

$$c_1 > 0, \quad c_4 > 0, \quad \text{and} \quad c_1 c_2 c_3 > c_3^2 + c_1^2 c_4 \quad (5.8)$$

Proof. Linearizing the full fractional system at the polymorphic coexistence equilibrium E_{123}^* yields the following 5×5 Jacobian matrix:

$$J(E_{123}^*) = \begin{pmatrix} -B^* - \mu & -\frac{\beta_1 S^*}{(1 + aI_1^*)^2} & -\beta_2 S^* & -\beta_3 S^* & 0 \\ \frac{\beta_1 I_1^*}{1 + aI_1^*} & -\frac{aI_1^*(\sigma_1 + \mu)}{1 + aI_1^*} & 0 & 0 & 0 \\ \beta_2 I_2^* & 0 & 0 & 0 & 0 \\ \beta_3 I_3^* & 0 & 0 & 0 & 0 \\ 0 & \sigma_1 & \sigma_2 & \sigma_3 & -\mu \end{pmatrix} \quad (5.9)$$

where $B^* = \frac{\beta_1 I_1^*}{1 + aI_1^*} + \beta_2 I_2^* + \beta_3 I_3^*$.

One eigenvalue is immediately decoupled via the recovery equation, yielding:

$$\lambda_1 = -\mu < 0 \quad (5.10)$$

The remaining four eigenvalues are determined by the roots of the characteristic equation $\det(\lambda I - \tilde{J}) = 0$ associated with the remaining 4×4 submatrix $\tilde{J}$ governing the (S, I_1, I_2, I_3) dynamics:

$$Q(\lambda) = \lambda^4 + c_1 \lambda^3 + c_2 \lambda^2 + c_3 \lambda + c_4 = 0 \quad (5.11)$$

By computing the algebraic trace and the sums of principal minors of $\tilde{J}$, the coefficients are explicitly derived as:

$$c_1 = B^* + \mu + \frac{aI_1^*(\sigma_1 + \mu)}{1 + aI_1^*} > 0 \quad (5.12)$$

$$c_2 = \frac{aI_1^*(\sigma_1 + \mu)(B^* + \mu)}{1 + aI_1^*} + \frac{\beta_1^2 S^* I_1^*}{(1 + aI_1^*)^3} + \beta_2^2 S^* I_2^* + \beta_3^2 S^* I_3^* > 0 \quad (5.13)$$

$$c_3 = \frac{aI_1^*(\sigma_1 + \mu)}{1 + aI_1^*} (\beta_2^2 S^* I_2^* + \beta_3^2 S^* I_3^*) > 0 \quad (5.14)$$

$$c_4 = \frac{\beta_1^2 \beta_2^2 (S^*)^2 I_1^* I_2^*}{(1 + aI_1^*)^3} + \frac{\beta_1^2 \beta_3^2 (S^*)^2 I_1^* I_3^*}{(1 + aI_1^*)^3} + \beta_2^2 \beta_3^2 (S^*)^2 I_2^* I_3^* > 0 \quad (5.15)$$

According to the fractional extension of the Routh-Hurwitz stability criterion [26], if the conditions (5.8) hold, then all roots of the polynomial (5.11) have strictly negative real parts, hence they satisfy $|\arg(\lambda_i)| = \pi > \frac{\alpha\pi}{2}$. Note that, for a fractional-order system with $\alpha \in (0, 1]$, these Routh-Hurwitz conditions are sufficient but not necessary for stability, since the fractional stability sector $|\arg(\lambda_i)| > \frac{\alpha\pi}{2}$ is strictly larger than the classical left half-plane. When these

parametric inequalities are satisfied, the multi-strain coexistence equilibrium E_{123}^* is locally asymptotically stable. $\square$

6. GLOBAL STABILITY ANALYSIS

Theorem 6.1. *If $R_0 \leq 1$, the Disease-Free Equilibrium E_0 is globally asymptotically stable in Ω .*

Proof. Construct the following Volterra-type fractional Lyapunov function:

$$L_0(t) = S - S_0 - S_0 \ln \left(\frac{S}{S_0} \right) + I_1 + I_2 + I_3 \quad (6.1)$$

Applying Lemma 2.3, the fractional derivative along the trajectories yields:

$${}^C D_t^\alpha L_0(t) \leq \left(1 - \frac{S_0}{S} \right) {}^C D_t^\alpha S + {}^C D_t^\alpha I_1 + {}^C D_t^\alpha I_2 + {}^C D_t^\alpha I_3 \quad (6.2)$$

Substituting the model equations:

$$\begin{aligned} {}^C D_t^\alpha L_0(t) \leq & \left(1 - \frac{S_0}{S} \right) (\Lambda - \mu S) + \left[\frac{\beta_1 S_0 I_1}{1 + a I_1} - (\sigma_1 + \mu) I_1 \right] \\ & + [\beta_2 S_0 I_2 - (\sigma_2 + \mu) I_2] + [\beta_3 S_0 I_3 - (\sigma_3 + \mu) I_3] \end{aligned} \quad (6.3)$$

Using $\Lambda = \mu S_0$, we obtain:

$${}^C D_t^\alpha L_0(t) \leq -\frac{\mu(S - S_0)^2}{S} + (\sigma_1 + \mu)(R_{0,1} - 1)I_1 + (\sigma_2 + \mu)(R_{0,2} - 1)I_2 + (\sigma_3 + \mu)(R_{0,3} - 1)I_3 \quad (6.4)$$

Since $R_{0,i} \leq 1$ for $i = 1, 2, 3$, we conclude that ${}^C D_t^\alpha L_0(t) \leq 0$. By the fractional LaSalle invariance principle, E_0 is globally asymptotically stable. $\square$

Theorem 6.2. *If $R_{0,1} > 1$, the Strain 1 monomorphic endemic equilibrium E_1^* is globally asymptotically stable under the competitive exclusion conditions $R_{0,2} \leq 1$ and $R_{0,3} \leq 1$.*

Proof. Define the following candidate Lyapunov function:

$$L_1(t) = S - S^* - S^* \ln \left(\frac{S}{S^*} \right) + I_1 - I_1^* - I_1^* \ln \left(\frac{I_1}{I_1^*} \right) + I_2 + I_3 \quad (6.5)$$

Computing the Caputo derivative and utilizing Lemma 2.3:

$${}^C D_t^\alpha L_1(t) \leq \left(1 - \frac{S^*}{S} \right) {}^C D_t^\alpha S + \left(1 - \frac{I_1^*}{I_1} \right) {}^C D_t^\alpha I_1 + {}^C D_t^\alpha I_2 + {}^C D_t^\alpha I_3 \quad (6.6)$$

Substituting system values and simplifying with the balance relationship $\Lambda = \mu S^* + \frac{\beta_1 S^* I_1^*}{1 + a I_1^*}$:

$$\begin{aligned} {}^C D_t^\alpha L_1(t) \leq & -\frac{\mu(S - S^*)^2}{S} + \frac{\beta_1 S^* I_1^*}{1 + a I_1^*} \left(3 - \frac{S^*}{S} - \frac{I_1}{I_1^*} \frac{1 + a I_1^*}{1 + a I_1} - \frac{S}{S^*} \frac{1 + a I_1}{1 + a I_1^*} \right) \\ & + (\sigma_2 + \mu)(R_{0,2} - 1)I_2 + (\sigma_3 + \mu)(R_{0,3} - 1)I_3 \end{aligned} \quad (6.7)$$

By applying the arithmetic-geometric mean inequality (AM-GM), the term in brackets remains negative or zero. Given $R_{0,2} \leq 1$ and $R_{0,3} \leq 1$, we obtain ${}^C D_t^\alpha L_1(t) \leq 0$. Thus, E_1^* is globally asymptotically stable. $\square$

7. NUMERICAL SIMULATIONS

In this section, we validate the theoretical results established previously through numerical simulations of the fractional-order multi-strain system (3.1). The Caputo system is solved using the predictor–corrector Adams–Bashforth–Moulton (ABM) scheme developed by Diethelm et al [27], which is well suited to non-local fractional operators. Our main objective is twofold: first, to illustrate the asymptotic behavior of each compartment toward the predicted equilibria, and second, to highlight the influence of the fractional order α on the memory-driven convergence speed of the trajectories.

7.1. Parameter Setting. Unless stated otherwise, the baseline parameters used throughout the simulations are reported in Table 1. These values are chosen so that the disease-free and the strain-1 monomorphic endemic scenarios can both be exhibited by varying the transmission rates.

TABLE 1. Baseline parameter values used in the numerical simulations.

Parameter	Value	Parameter	Value
Λ	10	σ_1	0.20
μ	0.10	σ_2	0.25
β_1	0.0040	σ_3	0.30
β_2	0.0025	a	0.05
β_3	0.0020	—	—

With these values, $S_0 = \Lambda/\mu = 100$, and the strain-specific basic reproduction numbers are $R_{0,1} = \frac{\beta_1 \Lambda}{\mu(\sigma_1 + \mu)} = \frac{0.004 \times 100}{0.30} = 1.33$, $R_{0,2} = \frac{\beta_2 \Lambda}{\mu(\sigma_2 + \mu)} = 0.71$, $R_{0,3} = \frac{\beta_3 \Lambda}{\mu(\sigma_3 + \mu)} = 0.50$.

Since $R_{0,1} > 1$ while $R_{0,2}, R_{0,3} < 1$, Theorem 6.2 predicts convergence toward the strain-1 monomorphic endemic equilibrium E_1^* with competitive exclusion of strains 2 and 3. The initial condition is fixed at $(S(0), I_1(0), I_2(0), I_3(0), R(0)) = (90, 5, 5, 5, 0)$.

7.2. Effect of the Fractional Order α . Figures 1a–1e depict the time evolution of each compartment for several values of the fractional order, α . In every case, the trajectories converge to the same steady state E_1^* , in full agreement with the global stability result of Theorem 6.2. As expected from the memory nature of the Caputo derivative, smaller values of α induce a slower, more strongly damped transient response, whereas $\alpha \rightarrow 1$ recovers the classical integer-order dynamics with the fastest relaxation toward equilibrium. Notably, the infected compartments I_2 and I_3 decay to zero for all α , numerically confirming competitive exclusion, while I_1 stabilizes at its positive endemic level I_1^* . The simulations

corroborate the analytical findings: the equilibrium reached by the system is independent of α it is determined solely by the model parameters and the reproduction numbers, while α governs the transient memory and the rate of convergence. This confirms the robustness of the stability conclusions across the whole admissible range $\alpha \in (0, 1]$ and illustrates the additional modeling flexibility offered by the fractional-order formulation.

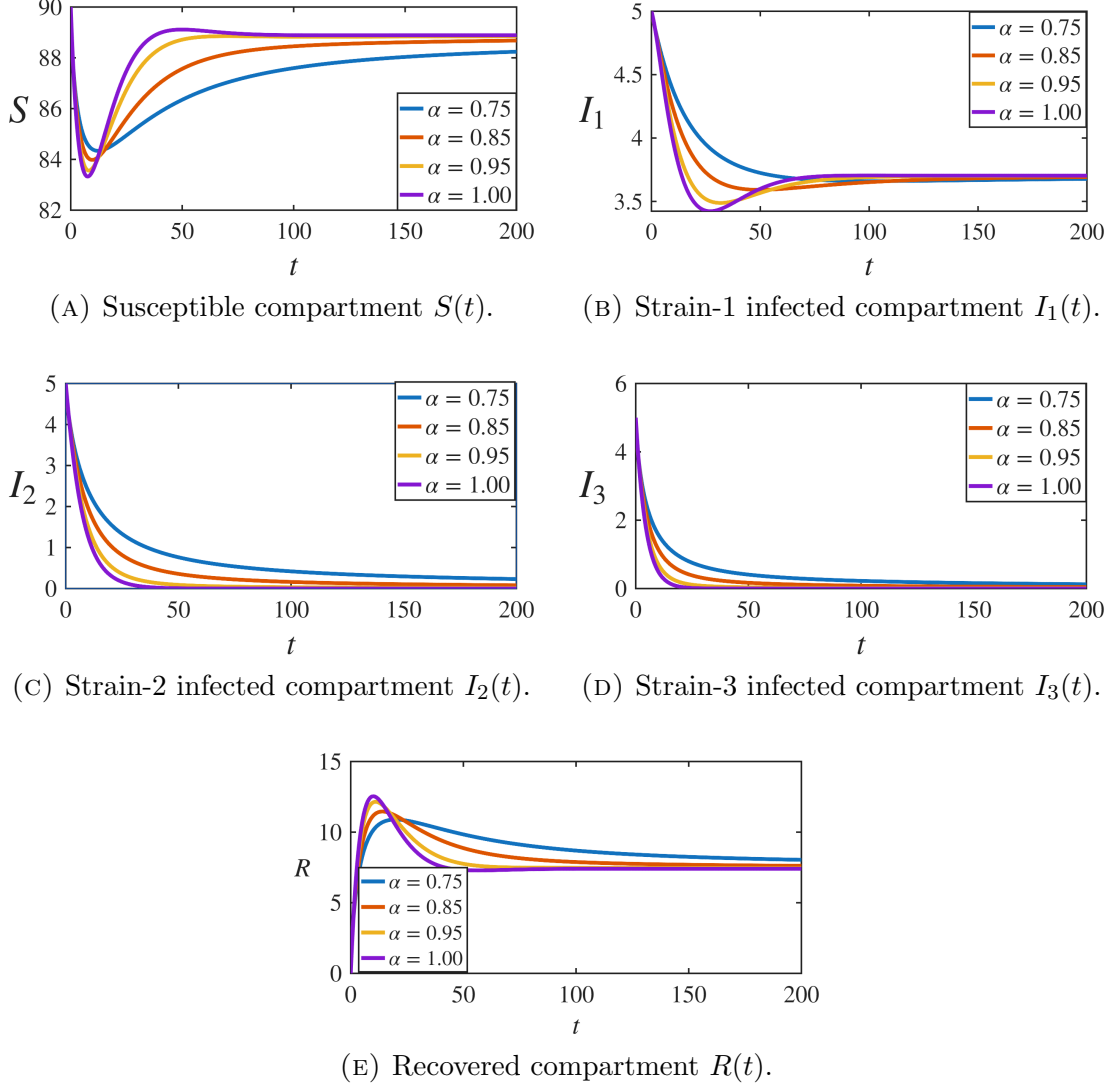


FIGURE 1. Convergence of each compartment for α : S , I_1 , I_2 and I_3 arranged two per row, with R centered on the last line.

7.3. Effect of the parameter a . Figures 2b–2e show the effect of the saturation parameter a on the dynamics, with the fractional order fixed at $\alpha = 0.95$ and a taken in $\{0, 0.05, 0.20, 0.50, 1.00\}$. Since a does not enter the expression of $R_{0,1}$, the epidemic threshold is unchanged and strain 1 remains endemic in all cases, while I_2 and I_3 still tend to zero, so competitive exclusion is preserved. The parameter a mainly affects the endemic level of strain 1: as a increases, the

saturation term $1 + aI_1$ lowers the force of infection at high infection densities, and the infected compartment I_1 settles at a smaller equilibrium value I_1^* . This is consistent with the analytical expression $I_1^* = \mu(R_{0,1} - 1)/(\beta_1 + a\mu)$, which decreases with a . The susceptible compartment S stabilizes at a slightly higher level for larger a , since fewer individuals are infected by strain 1, and the recovered compartment R follows the same trend as I_1 . The case $a = 0$ corresponds to a purely bilinear incidence for strain 1 and gives the highest endemic level, which confirms the crowding effect modeled by the saturated term.

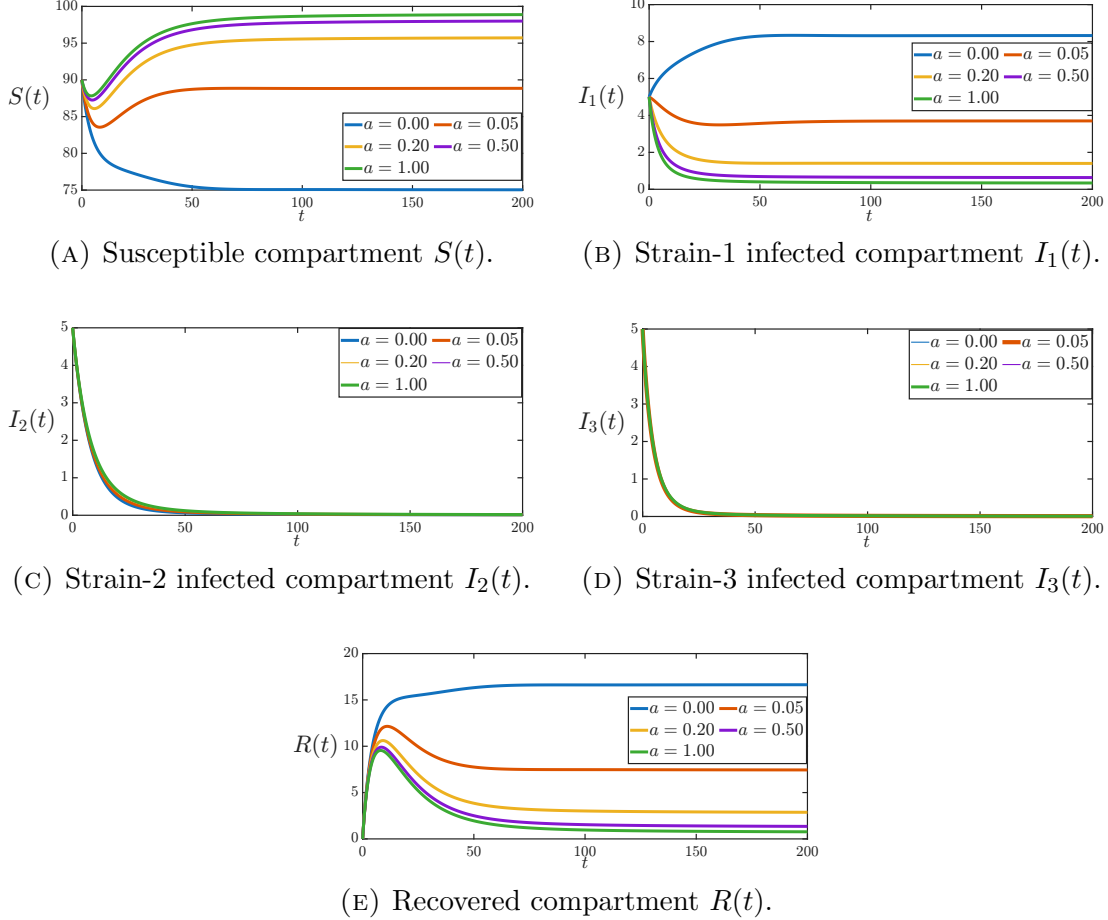


FIGURE 2. Effect of the saturation parameter a on the dynamics of the compartments S , I_1 , I_2 , I_3 , and R , with $\alpha = 0.95$ and a . The increase of a reduces the endemic level of strain 1, while I_2 and I_3 converge to zero in all cases, confirming the persistence of competitive exclusion.

8. CONCLUSION

In this paper, we proposed and analyzed a fractional-order $SI_1I_2I_3R$ epidemic model describing the competition of three co-circulating strains, in which strain 1 is transmitted through a saturated Holling type-II incidence while strains 2 and 3

follow a classical bilinear mass-action law. The memory and hereditary features inherent to the transmission process were incorporated by means of the Caputo fractional derivative of order $\alpha \in (0, 1]$. We first established the mathematical well-posedness of the model. Using the Banach fixed-point theorem, we proved the existence and uniqueness of a global solution, and we showed that all trajectories issued from non-negative initial data remain non-negative and uniformly bounded within a biologically feasible compact region. We then derived the strain-specific basic reproduction numbers $R_{0,1}$, $R_{0,2}$, and $R_{0,3}$ through the next-generation matrix method, and identified the disease-free equilibrium together with the strain-specific monomorphic endemic equilibria. The analysis of the coexistence state revealed that the simultaneous persistence of strains 2 and 3 requires the non-generic condition $R_{0,2} = R_{0,3}$; away from this equality, competitive exclusion drives the less fit strain to extinction. The stability of the equilibria was investigated at both the local and the global level. By examining the arguments of the eigenvalues of the associated Jacobian matrices and invoking Matignon's criterion, we obtained the local asymptotic stability conditions of the disease-free and monomorphic endemic equilibria. Suitable Volterra-type Lyapunov functions, adapted to the fractional-order setting, then allowed us to establish the global asymptotic stability of the disease-free equilibrium when $R_0 \leq 1$ and of the strain-1 endemic equilibrium when $R_{0,1} > 1$ under the competitive exclusion conditions $R_{0,2}, R_{0,3} \leq 1$. Finally, numerical simulations based on the predictor-corrector Adams-Bashforth-Moulton scheme confirmed the theoretical results. In particular, they showed that the fractional order α does not alter the location of the equilibrium reached by the system, which is governed solely by the model parameters and the reproduction numbers, but instead controls the memory of the transient regime: smaller values of α yield a slower and more strongly damped relaxation toward equilibrium, whereas $\alpha \rightarrow 1$ recovers the classical integer-order dynamics with the fastest convergence. Several directions naturally extend the present study. A more detailed investigation of the role of the saturation parameter a , which tempers the incidence of strain 1 at high infection levels, would clarify how crowding and behavioral effects reshape the endemic level and the speed of convergence. Beyond this, the framework could be enriched with a latent compartment, an explicit vaccination class, or optimal-control strategies, and the constant-order Caputo derivative could be replaced by a variable-order or a non-singular-kernel operator to capture more intricate memory effects.

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