A Fractional Two-Strain Epidemic Model with Treatment

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Abstract. In this study, we develop and examine a fractional-order epidemic model involving two strains, while taking into account treatment strategies and generalized incidence rates. The transmission dynamics of the two strains are described by two nonlinear incidence functions $f(S,I_1)I_1$ and $h(S,I_2)I_2$ allowing the model to capture more realistic infection mechanisms beyond the classical bilinear form. The proposed model is formulated as a system of four Caputo fractional differential equations describing the interactions among susceptible, infected, and recovered individuals. Initially, it is established that solutions exist, are unique, positive and bounded. Next, the basic reproduction numbers corresponding to each strain are computed using the next-generation matrix approach. The existence of both the disease-free and endemic equilibrium states is analyzed, and sufficient conditions for their global stability are established through appropriate Lyapunov functionals. Finally, numerical simulations are performed to support the analytical results and to demonstrate the impact of the fractional-order derivative on the system's convergence behavior. The role of treatment strategies in limiting the spread of the infection is also examined.

Keywords: Caputo fractional-order derivative, two-strain SIR epidemic model, general incidence function, local and global stability, treatment.

1 Introduction

Multi-strain epidemic models are vital mathematical tools that enable the analysis and understanding of the mechanisms governing the spread and evolution of infectious diseases caused by various mutations or variants. Many pathogens, including tuberculosis [1], human immunodeficiency virus HIV [2-6], COVID-19 [7-10], hepatitis B infection (HBV) [11-13], and hepatitis C infection (HCV) [14-16], exhibit multiple coexisting or competing strains that can significantly alter disease dynamics and control strategies [17-19]. Consequently, two or multi-strain compartmental models have been developed to better capture the complexity of such infections and to evaluate the interplay between viral competition, reinfection, and treatment efficiency [20-23].

Among the various compartmental frameworks, the SIR model divides the population into susceptible (S), infectious (T) and recovered ($\mathcal{R}$) classes-remain particularly useful for diseases with direct transmission, especially when the latent period can be neglected or approximated. For models involving multiple strains, the infected class is partitioned into several compartments, each associated with a specific strain, resulting in the $SI_1I_2\mathcal{R}$ structure used in this work. The SIR model, originally developed by Kermack and McKendrick in 1927 [24], constitutes a cornerstone framework for the assessment of key epidemiological indicators, notably the basic reproduction number R_0, the peak prevalence of infection, and herd immunity thresholds.

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Since then, numerous extensions have been proposed to incorporate multiple strains, age structures, vaccination strategies, and optimal control interventions [25-29]. Multi strain epidemic models have received considerable attention due to their ability to describe the coexistence and competition between different pathogen variants.

A key component of epidemic modeling is the incidence function, which governs how susceptible individuals become infected. The classical bilinear incidence βSI is widely used in epidemiological models [30]; however, it may fail to capture nonlinear transmission mechanisms, saturation effects, or behavioral changes occurring during disease outbreaks. To overcome these limitations, several authors have introduced more general nonlinear incidence functions that better describe realistic transmission processes. In this work, we consider a general incidence formulation for both strains, represented by the functions $f(S, I_1)I_1$ and $h(S, I_2)I_2$. This general framework allows the model to include a wide range of transmission mechanisms, such as saturated, Holling-type, or Beddington-DeAngelis incidence functions. Most existing epidemic models are formulated using classical integer-order differential equations. Although these models successfully describe the average dynamics of infectious diseases, they may not adequately capture memory effects, hereditary properties, or delayed responses that are often present in biological systems. To address these limitations, fractional order derivatives have been increasingly used in epidemiological modeling. In particular, the Caputo fractional derivative allows the incorporation of memory effects into the system dynamics, providing a more realistic description of disease transmission processes [31-36].
Motivated by these considerations, the present work proposes and analyzes a fractional order $SI_1I_2\mathcal{R}$ epidemic model with treatment and general incidence functions for both strains. The model incorporates memory effects through Caputo fractional derivatives and allows for nonlinear transmission dynamics. We derive conditions for the existence, positivity, and boundedness of solutions. Furthermore, the basic reproduction numbers associated with each strain are computed using the next-generation matrix method. The existence of the disease-free and endemic equilibria is established, and their global stability is investigated by constructing appropriate Lyapunov functionals. Numerical simulations are performed to support the analytical results. Furthermore, they assess how the fractional-order derivative affects the convergence of solutions to equilibrium. In addition, the effect of treatment strategies on reducing the number of infected individuals is examined. To the best of our knowledge, this is one of the first studies that simultaneously incorporates fractional-order dynamics, general nonlinear incidence functions, and treatment effects within a two-strain epidemic modeling framework. More recently, several works have addressed fractional modeling of viral infections and biological systems using advanced analytical and numerical approaches [37-40].

Accordingly, the following system of fractional differential equations formalizes the dynamics of the proposed model:

$$\begin{cases} {}^{C}D_t^{\alpha}\mathcal{S}(t) = \lambda - (1-u_1)f(\mathcal{S}, I_1)I_1 - (1-u_2)h(\mathcal{S}, I_2)I_2 - \mu\mathcal{S}, \\ {}^{C}D_t^{\alpha}I_1(t) = (1-u_1)f(\mathcal{S}, I_1)I_1 - (\sigma_1 + \mu)I_1, \\ {}^{C}D_t^{\alpha}I_2(t) = (1-u_2)h(\mathcal{S}, I_2)I_2 - (\sigma_2 + \mu)I_2, \\ {}^{C}D_t^{\alpha}\mathcal{R}(t) = \sigma_1 I_1 + \sigma_2 I_2 - \mu\mathcal{R}. \end{cases} \quad (1)$$

with initial conditions

$$S_0 \geq 0, I_{1,0} \geq 0, I_{2,0} \geq 0, \mathcal{R}_0 \geq 0.$$

Here, $\mathcal{S}(t)$ denotes the susceptible population, $T_1(t)$ and $T_2(t)$ the infectious classes, and $\mathcal{R}(t)$ the removed class. The parameter λ represents the recruitment rate, μ the natural mortality, σ_1 and σ_2 the recovery rates.

The general incidence functions $f(S, I_1)$ and $h(S, I_2)$ describe the transmission dynamics of each strain and satisfy standard biological conditions:
$(\mathrm{H}_1) f, h \in C^1(\mathrm{R}_+^2, \mathrm{R}_+)$, with $f(0, I_1) = h(0, I_2) = 0, \forall I_1, I_2 \geq 0$;
$(\mathrm{H}_2) \frac{\partial f}{\partial S}(S, I_1) \geq 0, \frac{\partial h}{\partial S}(S, I_2) \geq 0, \forall S > 0, \forall I_1, I_2 \geq 0$
$(\mathrm{H}_3) \frac{\partial f}{\partial I_1}(S, I_1) \leq 0, \frac{\partial h}{\partial I_2}(S, I_2) \leq 0, \forall S \geq 0, I_1, I_2 \geq 0.$

All the model (1) parameters are given in Fig. 1

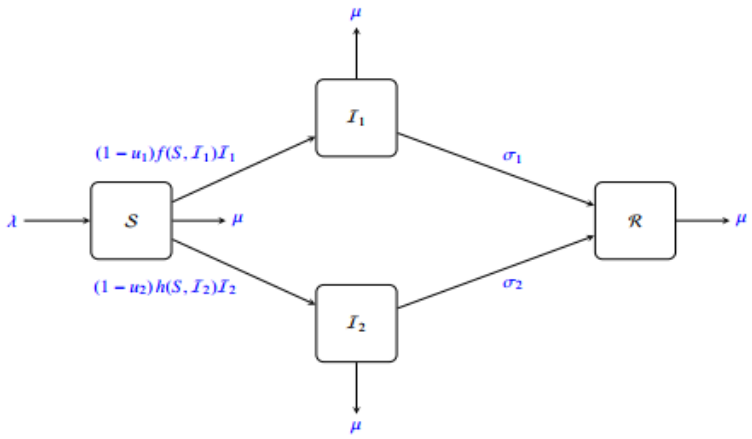

Fig. 1. Flowchart of the fractional model with general incidence functions.

2 Preliminary results

Definition 1. Let $\psi: [0, \infty) \to \mathbb{R}$ be a differentiable function and $0 < \alpha \leq 1$. The Caputo derivative of fractional order α is defined as

$$ {}^{c}D_t^{\alpha}\psi(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t \frac{\psi'(s)}{(t-s)^{\alpha}} ds, t > 0 $$

Definition 2 (Mittag-Leffler function). For $\alpha > 0$, the Mittag-Leffler function of parameter α is defined by

$$ E_{\alpha}(s) = \sum_{k=0}^{\infty} \frac{s^k}{\Gamma(\alpha k + 1)}. $$

Let $f: \mathbb{R}^n \longrightarrow \mathbb{R}^n$ where $n > 0$. We consider the following fractional-order system:

$$ D^{\alpha}X(t) = f(X(t)), 0 < \alpha \leq 1. $$

with $X(0) = X_0, X_0 \in \mathbb{R}^n$ and $0 < \alpha < 1$. To demonstrate the global stability of solutions of system (2) we will need the following lemma:

Lemma 1. If the function f satisfies the two following conditions:

1. $f(X)$ and $\frac{\partial f}{\partial X}(X)$ are continuous on $\mathbb{R}^n$.
2. $\|f(X)\| \leq q_1 + q_2\|X\|$ for all $X \in \mathbb{R}^n$, with q_1 and q_2 are two positive constants.

Then, system (2) admits a unique solution defined in R_+^n.

3 Existence, positivity and boundedness

In this section, we prove that the solutions of the proposed fractional SI_1I_2R model remain positive and bounded for all $t > 0$. For biological relevance, we consider non-negative initial conditions: $S(0) = S_0, I_1(0) = I_{1,0}, I_2(0) = I_{2,0}, R(0) = \mathcal{R}_0$, with all components in R_4. The results are summarized in the following proposition.

Proposition 1. For any non-negative initial conditions, the fractional system: ${}^cD_t^a X(t) = F\big(X(t)\big), 0 < a \leq 1$, admits a unique global solution which remains positive and bounded on $[0, \infty)$.

Proof. We rewrite system (1) in compact vector form:

$$X(t) = \begin{pmatrix} S \\ I_1 \\ I_2 \\ R \end{pmatrix}, F(X) = \begin{pmatrix} \lambda - (1-u_1)f(S,I_1)I_1 - (1-u_2)h(S,I_2)I_2 - \mu S \\ (1-u_1)f(S,I_1)I_1 - (\sigma_1+\mu)I_1 \\ (1-u_2)h(S,I_2)I_2 - (\sigma_2+\mu)I_2 \\ \sigma_1 I_1 + \sigma_2 I_2 - \mu R \end{pmatrix}.$$

Since $f, h \in C^1(\mathbb{R}_+^2)$, the function $\mathcal{F}$ is locally Lipschitz on $\mathbb{R}_+^4$. By standard results on Caputo fractional differential equations, problem

$${}^cD_t^a X(t) = \mathcal{F}\big(X(t)\big), \qquad X(0) = X_0$$

admits a unique local solution.
We now prove the positivity of the model solutions. First, we assume that all model parameters are positive; hence, we have

$$\begin{cases} {}^cD_t^a S|_{S=0} = \lambda > 0, \\ {}^cD_t^a I_1|_{I_1=0} = (1-u_1)f(S,I_1)I_1 - (\sigma_1+\mu)0 = 0, \\ {}^cD_t^a I_2|_{I_2=0} = (1-u_2)\mathrm{h}(S,I_2)I_2 - (\sigma\ \ {}_2+\mu)0 = 0, \\ {}^cD_t^a R|_{R=0} = \sigma_1 I_1 + \sigma_2 I_2 \geq 0. \end{cases}$$

Thus, trajectories starting in R_+^4 remain in R_+^4.
Finally, we prove that the solutions are bounded. Let the total population

$$N(t) = S(t) + I_1(t) + I_2(t) + R(t).$$

Summing the four equations yields

$$D_t^a \mathcal{N}(t) = \lambda - \mu \mathcal{N}(t).$$

This linear Caputo equation has solution $N(t) = \frac{\lambda}{\mu} + \left(N(0) - \frac{\lambda}{\mu}\right) E_a(-\mu t^2)$, where $E_a(\cdot)$ denotes the Mittag-Leffler function. Since $0 < E_a(-\mu t^a) < 1$ for all $t > 0$, we obtain

$$0 \leq N(t) \leq \max\left\{N(0), \frac{\lambda}{\mu}\right\}.$$

Thus, the biologically feasible region

$$\mathcal{H} = \left\{(S, I_1, I_2, R) \in \mathbb{R}_+^4 : S + I_1 + I_2 + \mathcal{R} \leq \frac{\lambda}{\mu}\right\}$$

is positively invariant. Therefore, $X(t)$ remains bounded for all $t > 0$.
The Lipschitz continuity of $\mathcal{F}$ together with invariance of $\mathcal{H}$ ensures global uniqueness of the solution.

4 The steady states

This subsection establishes the existence of the disease-free equilibrium and the endemic equilibria of the fractional model. Since the equation for R depends only on I_1, I_2 and R and does not influence the first three equations, the steady states can be determined from the subsystem satisfied by (S, I_1, I_2). Thus, system (1) reduces to

$$\begin{cases} {}^{c}D_t^{a} I_1(t) = (1-u_1) f(S, I_1) I_1 - (\sigma_1 + \mu) I_1, \\ {}^{c}D_t^{a} S(t) = \lambda - (1-u_1) f(S, I_1) I_1 - (1-u_2) h(S, I_2) - \mu S, \\ {}^{c}D_t^{a} I_2(t) = (1-u_2) h(S, I_2) - (\sigma_2 + \mu) I_2, \end{cases} \tag{3}$$

with the recovered population given by: $\mathcal{R}(t) = N(t) - S(t) - I_1(t) - I_2(t)$, where $\mathcal{N}(t)$ satisfies the scalar fractional equation ${}^{c}D_t^{a}\mathcal{N}(t) = \lambda - \mu\mathcal{N}(t)$.

4.1 The basic reproduction number

The basic reproduction number represents the average number of secondary infections produced by a typical infected individual introduced into a fully susceptible population. To compute it, we use the next-generation matrix approach. Let F denote the matrix of new infection terms and V the matrix describing transition terms among infected compartments for the reduced system (3).

Evaluated at the disease-free equilibrium $\varepsilon_f = \left(\frac{\lambda}{\mu}, 0, 0\right)$, the matrices F and V for the infected variables (I_1, I_2) are

$$F = \begin{pmatrix} (1-w_1) f\left(\frac{\lambda}{\mu}, 0\right) & 0 \\ 0 & (1-w_2) h\left(\frac{\lambda}{\mu}, 0\right) \end{pmatrix}, V = \begin{pmatrix} \sigma_1 + \mu & 0 \\ 0 & \sigma_2 + \mu \end{pmatrix}.$$

Thus

$$FV^{-1} = \begin{pmatrix} \dfrac{(1-u_1) f\left(\frac{\lambda}{\mu}, 0\right)}{\sigma_1 + \mu} & 0 \\ 0 & \dfrac{(1-u_2) h\left(\frac{\lambda}{\mu}, 0\right)}{(\sigma_2 + \mu)} \end{pmatrix}.$$

The basic reproduction number is the spectral radius of the matrix $\mathcal{F}V^{-1}$, hence

$$R_0 = \max\{R_{0,1}, R_{0,2}\},$$

where

$$R_{0,1} = \frac{(1-u_1)f\left(\frac{\lambda}{\mu}, 0\right)}{\sigma_1 + \mu}, R_{0,2} = \frac{(1-u_2)h\left(\frac{\lambda}{\mu}, 0\right)}{\sigma_2 + \mu}.$$

$R_{0,1}$ and $R_{0,2}$ denote the basic reproduction numbers of the first and second strains, respectively.

Theorem 2. The system (3) has a unique disease-free equilibrium $\mathcal{E}_f$ and three endemic equilibria under the following conditions:

- The strain 1 endemic equilibrium ε_{s_1} exists if and only if $R_{0,1} > 1$.
- The strain 2 endemic equilibrium ε_{s_2} exists if and only if $R_{0,2} > 1$.
- The coexistence equilibrium ε_t, exists when both $R_{0,1} > 1$ and $R_{0,2} > 1$.

Proof. We determine stationary states by solving

$$\begin{cases} 0 = \lambda - (1-u_1)f(S, I_1)I_1 - (1-u_2)h(S, I_2)I_2 - \mu S, \\ 0 = (1-u_1)f(S, I_1)I_1 - (\sigma_1 + \mu)I_1, \\ 0 = (1-u_2)h(S, I_2)I_2 - (\sigma_2 + \mu)I_2, \\ 0 = \sigma_1 I_1 + \sigma_2 I_2 - \mu R. \end{cases}$$

1. Disease-free equilibrium. Setting $I_1 = I_2 = 0$ gives $\mathcal{E}_f = \left(\frac{\lambda}{\mu}, 0,0,0\right)$.
2. Strain-1 endemic equilibrium. Assume $I_1 > 0$ and $I_2 = 0$. From the second equation of (4) we get

$$(1-u_1)f(S, I_1) = \sigma_1 + \mu.$$

From the first equation we have

$$\lambda - \mu S = (\sigma_1 + \mu)I_1, \text{ hence } I_1 = \frac{\lambda - \mu S}{\sigma_1 + \mu}.$$

Define the function $\Psi: [0, \infty) \to \mathbb{R}$ by

$$\Psi(S) := (1-u_1)f\left(S, \frac{\lambda - \mu S}{\sigma_1 + \mu}\right) - (\sigma_1 + \mu).$$

We compute its derivative with respect to S :

$$\frac{\partial \Psi(S)}{\partial S} = (1 = \mu_1)\left[\frac{\partial f}{\partial S}(S, I_1(S)) + \frac{\partial f}{\partial I_1}(S, (I_1)(S)) \cdot \frac{\partial \Psi(I_1)}{\partial S}\right],$$
$$\text{with } I_1(S) := \frac{\lambda = \mu S}{\sigma_1 + \mu}, \frac{\partial I_1(S)}{\partial S} = -\frac{\mu}{\sigma_1 + \mu}.$$

Hence

$$\frac{\partial \Psi(S)}{\partial S} = (1 = n_1)\left[\frac{\partial f}{\partial S}(S, h_1(S)) = \frac{\mu}{\sigma_1 + \mu}\frac{\partial f}{\partial h_1}(S, I_1(S))\right].$$

By hypotheses ($H2$) and ($H3$) we have $\frac{\partial f}{\partial S} \geq 0$ and $\frac{\partial f}{\partial I_1} \leq 0$, hence each term in square brackets is nonnegative and therefore

$$\frac{\partial \Psi(S)}{\partial S} \geq 0 \text{ for all } S \geq 0$$

Moreover, $\Psi(0) = (1-\mu_1)\rho\left(0, \frac{\lambda}{\sigma_1+\mu}\right) = (\sigma_1+\mu) = -(\sigma_1+\mu) < 0$

and, $\Psi\left(\frac{d}{\mu}\right) = (1=n_1)f\left(\frac{1}{\mu}, 0\right) = (\sigma_1+\mu) = (\sigma_1+\mu)\left(R_{0,1}=1\right)$.

Therefore, if $R_{a,u} > 1$ then $\Psi\left(\frac{A}{\mu}\right) > 0$. Since Ψ is continuous and nondecreasing. there exists a unique $S_1^* \in \left[0, \frac{\lambda}{\mu}\right]$ such that $\Psi(S_1^*) = 0$. This gives the unique strain-1 endemic equilibrium

$$\varepsilon_{S_1} = \left(S_1', \frac{\lambda - \mu S_1^+}{\sigma_1+\mu}, 0, \frac{\sigma_1(\lambda-\mu S_1^+)}{\mu(\sigma_1+\mu)}\right)$$

with, $S_{\mathrm{i}} \in \left[0, \frac{\lambda}{\mu}\right]$ and $I_{\mathrm{i}} > 0$.

3. Strain-2 endemic equilibrium. If $I_2 > 0$ and $I_1 = 0$, then from the third equation

$$(1-u_2)h(S, I_2) = \sigma_2 + \mu$$

Moreover, $I_2 = \frac{\lambda - \mu S}{\sigma_2+\mu}$.

Define the function: $\Phi(S) = (1=\mu_2)g\left(S, \frac{\mu - s}{\sigma_2+\mu}\right) = (\sigma_2+\mu)$.

We have, $\Phi(0) = -(\sigma_2+\mu) < 0$, and $\Phi\left(\frac{\lambda}{\mu}\right) = (\sigma_2+\mu)(R_{\mathrm{A2}}-1)$.

Thus, if $R_2 > 1$, there exists a unique $S_2 \in (0, \lambda/\mu)$ such that $\Phi(S_2) = 0$.

Hence the strain-2 endemic equilibrium exists.

4. Coexistence equilibrium. If $I_1 > 0$ and $I_2 > 0$, then

$$(1-u_1)f(S, I_1) = \sigma_1 + \mu, (1=u_2)h(S, I_2) = \sigma_2 + \mu$$

Substituting into the first equation yields

$$(\sigma_1+\mu)I_1 + (\sigma_2+\mu)I_2 = \lambda = \mu S.$$

A positive coexistence equilibrium exists if and only if

$$R_{0,1} > 1 \text{ and } R_{0,2} > 1.$$

4.2 Global stability of equilibria

4.2.1 Global stability analysis of the disease-free equilibrium

Theorem 3. If $R_{0,1} < 1$, then the disease-free equilibrium $\mathcal{E}_f$ with is globally asymptotically stable.

Proof. We perform the proof by constructing a suitable Lyapunov function. Introduce the Lyapunov as

$$\mathcal{P}_0(\mathcal{S}, I_1, I_2) = \mathcal{S} - \mathcal{S}^0 - \int_{\mathcal{S}^0}^{\mathcal{S}} \frac{f(\mathcal{S}^0, 0)}{f(X, 0)} dX + I_1 + I_2$$

The integral is well defined by hypothesis (H1) − (H3) and $f(\cdot, 0) > 0$ for $S > 0$.
Using properties of the Caputo derivative and differentiating along solutions of the SI_1I_2R system yields

$$\begin{aligned} {}^cD_t^\alpha \mathcal{P}_0 &= {}^cD_t^\alpha \mathcal{S} - \frac{f(\mathcal{S}^0, 0)}{f(\mathcal{S}, 0)} {}^cD_t^\alpha \mathcal{S} + {}^cD_t^\alpha I_1 + {}^cD_t^\alpha I_2 \\ &= \left(1 - \frac{f(\mathcal{S}^0, 0)}{f(\mathcal{S}, 0)}\right)(\lambda - (1-u_1) f(\mathcal{S}, I_1) I_1 - (1-u_2)\beta S I_2 - \mu \mathcal{S}) \\ &\quad + (1-u_1) f(\mathcal{S}, I_1) I_1 - (\sigma_1 + \mu) I_1 + (1-u_2)\beta S I_2 - (\sigma_2 + \mu) I_2 \end{aligned}$$

After regrouping terms and using $S^0 = \frac{\lambda}{\mu}$ we obtain

$$\begin{aligned} {}^cD_t^\alpha \mathcal{P}_0 &= \mu S^0 \left(1 - \frac{S}{\mathcal{S}^0}\right)\left(1 - \frac{f(\mathcal{S}^0, 0)}{f(\mathcal{S}, 0)}\right) \\ &\quad + I_1 \left\{(1-u_1)\frac{f(\mathcal{S}, I_1)}{f(\mathcal{S}, 0)} f(\mathcal{S}^0, 0) - (\sigma_1 + \mu)\right\} \\ &\quad + I_2 \left\{(1-u_2)\frac{\beta \mathcal{S}}{\beta \mathcal{S}^0}\beta S^0 - (\sigma_2 + \mu)\right\} \end{aligned}$$

Using the definitions of the reproduction numbers at the DFE,

$$R_{0,1} = \frac{(1-u_1) f(S^0, 0)}{\sigma_1 + \mu}, R_{0,2} = \frac{(1-u_2)\beta S^0}{\sigma_2 + \mu}$$

we can bound the last two terms to obtain

$$\begin{aligned} {}_{cD_t^\alpha \mathcal{P}_0 \le} \mu \mathcal{S}^0 \left(1 - \frac{\mathcal{S}}{\mathcal{S}^0}\right)\left(1 - \frac{f(\mathcal{S}^0, 0)}{f(\mathcal{S}, 0)}\right) \\ + I_1 \left(R_{0,1}\frac{f(\mathcal{S}, I_1)}{f(\mathcal{S}, 0)} - 1\right)(\sigma_1 + \mu) \\ + I_2 \left(R_{0,2}\frac{\mathcal{S}}{\mathcal{S}^0} - 1\right)(\sigma_2 + \mu) \end{aligned}$$

We now analyze two cases.
Case 1: $S \le S^0$. By (H2) and (H3) we have $\frac{f(S, I_1)}{f(S, 0)} \le 1$ and $\frac{S}{S^0} \le 1$. Hence

$$I_1 \left(R_{0,1}\frac{f(\mathcal{S}, I_1)}{f(\mathcal{S}, 0)} - 1\right)(\sigma_1 + \mu) \le I_1 \big(R_{0,1} - 1\big)(\sigma_1 + \mu),$$

and

$$I_2 \left(R_{0,2}\frac{S}{S^0} - 1\right)(\sigma_2 + \mu) \le I_2 \big(R_{0,2} - 1\big)(\sigma_2 + \mu).$$

Moreover, since $S \le S^0$ one has $1 - \frac{S}{S^0} \ge 0$ and by monotonicity of f the factor $1 - \frac{f(S^0, 0)}{f(S, 0)} \le 0$, hence the product

$$\mu S^0\left(1-\frac{S}{S^0}\right)\left(1-\frac{f(S^0,0)}{f(S,0)}\right)\le 0.$$

Therefore, if $R_{0,1}\le 1$ and $R_{0,2}\le 1$, each term is nonpositive and

$${}^cD_t^\alpha \mathcal{P}_0 \le 0.$$

Case 2: $S > S^0$. By (H2) we have $\frac{f(S,I_1)}{f(S,0)} \ge 1$ and $\frac{S}{S^0} > 1$, while $\frac{f(S^0,0)}{f(S,0)} < 1$, so the first term is again ≤ 0.
Using $R_{0,1}\le 1$ and $R_{0,2}\le 1$ we obtain

$$I_1\left(R_{0,1}\frac{f(S,I_1)}{f(S,0)}-1\right)(\sigma_1+\mu)\le 0, I_2\left(R_{0,2}\frac{S}{S^0}-1\right)(\sigma_2+\mu)\le 0,$$

hence again ${}^cD_r^\alpha \mathcal{P}_0 \le 0$.
Thus, in both cases, provided $R_{0,1}\le 1$ and $R_{0,2}\le 1$, we have

$${}^cD_t^\alpha \mathcal{P}_0(\mathcal{S},I_1,I_2)\le 0,$$

and equality holds only at the $\mathcal{E}_f$. By the direct Lyapunov method for Caputo fractional systems, this implies that $\mathcal{E}_0$ is globally asymptotically stable.

4.2.2 Global stability analysis of the strain 1 endemic state

We assume that the incidence function f satisfies the structural condition used to establish the global stability of ε_{s_1} :

$$\left(1=\frac{f(S,I_1)}{f\left(S,T_{1,n}^+\right)}\right)\left(\frac{f\left(S,T_{1,\mathrm{a_a}}^+\right)}{f(S,I_1)}=\frac{I_1}{T_{1,n}^+}\right)\le 0, \forall S,I_1>0.$$

Theorem 4. If $R_{0,2}\le 1 < R_{0,1}$, then the strain-1 endemic equilibrium $\mathcal{E}_{s_1}$ is globally asymptotically stable.

Proof. Consider the strain- 1 endemic equilibrium $\mathcal{E}_{s_1}=\left(\mathcal{S}_1^*, I_{1,s_1}^*, 0, \mathcal{R}_{s_1}^*\right)$, which satisfies the equilibrium relations

$$(1-u_1)f\left(S_1^*, I_{1,s_1}^*\right)=\sigma_1+\mu, \lambda=\mu S_1^*+(1-u_1)f\left(S_1^*, I_{1,s_1}^*\right)I_{1,s_1}^*.$$

We introduce the Lyapunov functional

$$\begin{aligned}\mathcal{P}_1(S,I_1,I_2,\mathcal{R}) =& S-S_1^*-\int_{S_1^*}^{S}\frac{f\left(S_1^*,I_{1,s_1}^*\right)}{f\left(X,I_{1,s_1}^*\right)}dX\\ &+I_{1,s_1}^*\left(\frac{I_1}{I_{1,s_1}^*}-\ln\frac{I_1}{I_{1,s_1}^*}-1\right)+I_2+\mathcal{R}.\end{aligned}$$

Computing its Caputo derivative and after inserting (6) and simplifying, we obtain

$$
\begin{aligned}
c_{D_t^{\alpha}\mathcal{P}_1=}&\mu(\mathcal{S}_1^* - \mathcal{S})\left(1 - \frac{f\left(\mathcal{S}_1^*, I_{1,s_1}^*\right)}{f\left(\mathcal{S}, I_{1,s_1}^*\right)}\right) \\
&+(\sigma_1 + \mu)I_{1,s_1}^* - (\sigma_1 + \mu)I_1\left(1 - \frac{I_{1,s_1}^*}{I_1}\right) \\
&-(1-u_1)f(\mathcal{S}, I_1)I_1\left(\frac{I_{1,s_1}^*}{I_1} - \frac{f\left(\mathcal{S}_1^*, I_{1,s_1}^*\right)}{f\left(\mathcal{S}, I_{1,s_1}^*\right)}\right) \\
&-\mu\mathcal{R} + I_2\big((1-u_2)\beta\mathcal{S} - \mu\big)
\end{aligned}
$$

We now reorganize the nonlinear terms in I_1. Write

$$(\sigma_1 + \mu)I_{1,s_1}^* = (1-u_1)f\left(S_1^*, I_{1,s_1}^*\right)I_{1,s_1}^*.$$

Using this identity and collecting all contributions involving $(1-u_1)f(S, I_1)I_1$, we obtain the compact factorization

$$(1-u_1)I_{1,s_1}^*[\xi(S, I_1) + \Theta(S, I_1)],$$

where the first bracket

$$\xi(S, I_1) = \frac{f\left(S_1^*, I_{1,s_1}^*\right)}{f\left(S, I_{1,s_1}^*\right)} - \frac{I_1}{I_{1,s_1}^*},$$

is nonpositive by the monotonicity assumptions (MA-MG) on f, and the key bracket

$$\Theta(\mathcal{S}, I_1) = \frac{f\left(\mathcal{S}, I_{1,s_1}^*\right)}{f(\mathcal{S}, I_1)} + \frac{f(\mathcal{S}, I_1)}{f\left(\mathcal{S}, I_{1,s_1}^*\right)}\frac{I_1}{I_{1,s_1}^*} - \frac{I_1}{I_{1,s_1}^*} - 1.$$

By the arithmetic-geometric inequality (AGM),

$$\Theta(\mathcal{S}, I_1) \leq 0.$$

Hence all nonlinear terms involving I_1 are nonpositive.
For the I_2-component, using again MA-MG and the condition $R_{0,2} \leq 1$, one obtains

$$I_2\big((1-u_2)\beta S - \mu\big) - (1-u_2)\beta S I_2\left(1 - \frac{f\left(S_1^*, I_{1,s_1}^*\right)}{f\left(S, I_{1,s_1}^*\right)}\right) \leq 0.$$

Finally, the term $-\mu\mathcal{R}$ is nonpositive.
Collecting all contributions in (7), we conclude

$${}^{c}D_t^{\alpha}\mathcal{P}_1(\mathcal{S}, I_1, I_2, \mathcal{R}) \leq 0$$

Equality holds only when $I_2 = 0, \mathcal{R} = \frac{\sigma_1}{\mu}I_1$ and the brackets $\xi(S, I_1) = 0, \Theta(S, I_1) = 0$, which implies $S = S_1^*$ and $I_1 = I_{1,s_1}^*$. Therefore, the largest invariant set is the singleton ($\mathcal{E}_{s_1}$).

Applying LaSalle's invariance principle for Caputo fractional systems, the equilibrium $\mathcal{E}_{s_1}$ is globally asymptotically stable whenever $R_{0,2} \leq 1 < R_{0,1}$.

4.2.3 Global stability analysis of the strain-2 endemic equilibrium

Theorem 5. If $R_{0,1} \leq 1 < R_{0,2}$, then the strain- 2 endemic equilibrium $\mathcal{E}_{s_2}$ is globally asymptotically stable.

Proof. We introduce the Lyapunov function

$$\mathcal{P}_2(S, I_1, I_2, \mathcal{R}) = S - S_2^* - \int_{S_2^*}^{S} \frac{h(S_2^*, I_2^*)}{h(X, I_2^*)} dX + I_2^* \left(\frac{I_2}{I_2^*} - \ln \frac{I_2}{I_2^*} - 1 \right) + I_1 + \mathcal{R}.$$

where $(S_2^*, 0, I_2^*, \mathcal{R}_2^*)$ denotes the endemic equilibrium associated with strain 2 . The function $\mathcal{P}_2$ is nonnegative and vanishes only at $\mathcal{E}_{s_2}$.

The Caputo derivative along the system trajectories gives

$$\begin{aligned} {}^{c}D_t^{\alpha}\mathcal{P}_2 =& [\lambda - f(\mathcal{S}, I_1)I_1 - h(\mathcal{S}, I_2)I_2 - \mu S] \left(1 - \frac{\mathcal{S}_2^*}{\mathcal{S}}\right) \\ &+ [h(\mathcal{S}, I_2)I_2 - (\sigma_2 + \mu)I_2] \left(1 - \frac{I_2^*}{I_2}\right) \\ &+ f(\mathcal{S}, I_1)I_1 - (\sigma_1 + \mu)I_1 + \sigma_1 I_1 + \sigma_2 I_2 - \mu R. \end{aligned}$$

Using the equilibrium relations

$$\lambda = \mu S_2^* + h(S_2^*, I_2^*)I_2^*, (\sigma_2 + \mu)I_2^* = h(S_2^*, I_2^*)I_2^*,$$

we obtain after rearrangement

$$\begin{aligned} {}^{c}D_t^{\alpha}\mathcal{P}_2 =& \mu(\mathcal{S}_2^* - \mathcal{S}) \left(1 - \frac{\mathcal{S}_2^*}{\mathcal{S}}\right) \\ &+ \left(1 - \frac{I_2^*}{I_2}\right) (h(\mathcal{S}, I_2)I_2 - h(\mathcal{S}_2^*, I_2^*)I_2^*) \\ &+ f(\mathcal{S}, I_1)I_1 - (\sigma_1 + \mu)I_1 + \sigma_1 I_1 - \mu\mathcal{R} \end{aligned}$$

We now analyze each block.
(i) Effect of $R_{0,1} \leq 1$.

Since

$$R_{0,1} = \frac{f(\mathcal{S}_2^*, 0)}{\sigma_1 + \mu} \leq 1,$$

the infection pressure of strain 1 cannot sustain $I_1 > 0$. Hence

$$f(\mathcal{S}, I_1)I_1 - (\sigma_1 + \mu)I_1 + \sigma_1 I_1 = -\mu I_1 \leq 0$$

(ii) Effect of $R_{0,2} > 1$.

The basic reproduction number associated with strain 2 is $R_{0,2} = \frac{h(S_0,0)}{\sigma_2+\mu} > 1$,
which guarantees the existence of a positive equilibrium $I_2^* > 0$. The equilibrium condition

$$(\sigma_2 + \mu)I_2^* = h(S_2^*, I_2^*)I_2^*$$

ensures that the block

$$\left(1 - \frac{I_2^*}{I_2}\right)(h(S, I_2)I_2 - g(S_2^*, I_2^*)I_2^*)$$

has the classical Lyapunov form $\left(1 - \frac{x^*}{x}\right)\left(F(x) - F(x^*)\right)$, which is nonpositive under the standard monotonicity assumptions on g.
(iii) Remaining terms.

The $\mathcal{S}$-term satisfies $\mu(\mathcal{S}_2^* - \mathcal{S})\left(1 - \frac{\mathcal{S}_2^*}{\mathcal{S}}\right) \leq 0$, and $-\mu R \leq 0$.
Collecting all terms gives ${}^{C}D_t^{\alpha}\mathcal{P}_2 \leq 0$.
The largest invariant set contained in $\{ {}^{C}D_t^{\alpha}\mathcal{P}_2 = 0\}$ is the singleton $\mathcal{E}_{s_2}$.
Therefore, by LaSalle's invariance principle for fractional-order systems, the strain-2 endemic equilibrium $\mathcal{E}_{s_2}$ is globally asymptotically stable.

4.2.4 Global stability of the both endemic equilibrium

Theorem 6. If $R_{0,1} > 1$ and $R_{0,2} > 1$, then the coexisting endemic equilibrium $\mathcal{E}_{s_t}$ is globally asymptotically stable.

Proof. We define the Lyapunov function

$$\begin{aligned}\mathcal{P}_c(\mathcal{S}, I_1, I_2, \mathcal{R}) =& \mathcal{S} - \mathcal{S}^* - \int_{\mathcal{S}^*}^{\mathcal{S}} \frac{f(\mathcal{S}^*, I_1^*)T_1^* + h(\mathcal{S}^*, I_2^*)I_2^*}{f(X, T_1^*)T_1^* + h(X, T_2^*)T_2^*} dX \\ &+ I_1^*\left(\frac{I_1}{TI_1^*} - \ln\frac{I_1}{I_1^*} - 1\right) \\ &+ I_2^*\left(\frac{I_2}{I_2^*} - \ln\frac{I_2}{I_2^*} - 1\right) \\ &+ \mathcal{R} - \mathcal{R}^* - \mathcal{R}^*\ln\frac{\mathcal{R}}{\mathcal{R}^*}\end{aligned}$$

The Caputo derivative along the trajectories of the system is

$$
\begin{aligned}
{}^{c}D_t^{\alpha}\mathcal{P}_c =&(\lambda - f(\mathcal{S},I_1)I_1 - h(\mathcal{S},I_2)I_2 - \delta\mathcal{S})\left(1-\frac{\mathcal{S}^*}{\mathcal{S}}\right)\\
&+(f(\mathcal{S},I_1)I_1 - (\sigma_1+\delta)I_1)\left(1-\frac{T_1^*}{I_1}\right)\\
&+(h(\mathcal{S},I_2)I_2 - (\sigma_2+\delta)I_2)\left(1-\frac{T_2^*}{I_2}\right)\\
&+(\sigma_1 I_1 + \sigma_2 I_2 - \delta\mathcal{R})\left(1-\frac{\mathcal{R}^*}{\mathcal{R}}\right)
\end{aligned}
$$

Substituting the equilibrium relations

$$
\begin{cases}
\lambda = \delta S^* + f(S^*,T_1^*)I_1^* + h(S^*,T_2^*)I_2^*\\
f(S^*,T_1^*)T_1^* = (\sigma_1+\delta)T_1^*\\
h(S^*,T_2^*)T_2^* = (\sigma_2+\delta)I_2^*\\
\sigma_1 T_1^* + \sigma_2 I_2^* = \delta\mathcal{R}^*
\end{cases}
$$

we analyze each component.
For the S component we obtain

$$
(\lambda - f(S,I_1)I_1 - h(S,I_2)I_2 - \delta S)\left(1-\frac{S^*}{S}\right) \le 0.
$$

For the I_1 component

$$
(f(S,I_1)I_1 - (\sigma_1+\delta)I_1)\left(1-\frac{T_1^*}{I_1}\right) = -\frac{(\sigma_1+\delta)(I_1-T_1^*)^2}{I_1} \le 0.
$$

For the I_2 component

$$
(h(S,I_2)I_2 - (\sigma_2+\delta)I_2)\left(1-\frac{T_2^*}{I_2}\right) = -\frac{(\sigma_2+\delta)(I_2-I_2^*)^2}{I_2} \le 0.
$$

For the $\mathcal{R}$ component

$$
(\sigma_1 I_1 + \sigma_2 I_2 - \delta\mathcal{R})\left(1-\frac{\mathcal{R}^*}{\mathcal{R}}\right) = -\delta\frac{(\mathcal{R}-\mathcal{R}^*)^2}{\mathcal{R}} \le 0.
$$

Thus

$$
{}^{C}D_t^{\alpha}\mathcal{P}_c \le 0
$$

with equality only at

$$
(\mathcal{S},I_1,I_2,\mathcal{R}) = (\mathcal{S}^*,I_1^*,I_2^*,\mathcal{R}^*)
$$

Therefore, by LaSalle's invariance principle for fractional-order systems, all trajectories converge to the endemic equilibrium $\mathcal{E}_{s_t}$.

Hence the coexistence equilibrium is globally asymptotically stable whenever $R_{0,1} > 1$ and $R_{0,2} > 1$.

5 Numerical simulations and discussions

In this section, we will give some numerical simulations to show the effect of fractional derivative and the treatment parameters on infection. These numerical simulations are performed using MATLAB. The first numerical simulation was done using an algorithm presented [41]. More precisely, we use the following numerical scheme:

$$U(t_j) = \frac{h^\alpha}{\Gamma(\alpha+2)}((j-1)^{\alpha+1} - (j-\alpha-1)j^\alpha)\mathcal{F}(U(t_0)) + U(t_0)$$
$$+ \frac{h^\alpha}{\Gamma(\alpha+2)}\sum_{i=1}^{j=1}((j-i+1)^{\alpha+1} - 2(j-i)^{\alpha+1} + (j-i-1)^{\alpha+1})\mathcal{F}(U(t_i))$$
$$+ \frac{h^\alpha}{\Gamma(\alpha+2)}\mathcal{F}\left(U(t_{j-1})\right) + \frac{h^\alpha}{\Gamma(\alpha+1)}\mathcal{F}\left(U(t_{j-1})\right)$$

Where $t_j = t(j-1) + h$, for $j = 0,1,\ldots\ldots,N-1$.

The stability behavior of the coexistence endemic equilibrium $\mathcal{E}_{s_r}$ is investigated under two non-monotonic incidence functions given by $f(\mathcal{S}, I_1) = \frac{\beta_1 \mathcal{S}}{1+mI_1^2}$ and $h(\mathcal{S}, I_2) = \beta_2 \mathcal{S} I_2$. For the numerical simulations, the system dynamics are illustrated in Figures [2-5] using the parameter set [42], and the different value of treatment parameters: $\lambda = 1, \beta_1 = \beta_2 = 0.6, \alpha_1 = \alpha_2 = 0.5, \sigma_1 = \sigma_2 = 0.15, \mu = 0.2, m_1 = 0.14$. Under these conditions, we see the convergence towards the steady state: $(0.8982, 0.5904, 0.5815, 0.8433, 0.8309, 1.2557)$ and we have $R_{0,1} = R_{0,2} = 6.1224$. In this case, both basic reproduction numbers satisfy: $R_{0,1} > 1$ and $R_{0,2} > 1$, which confirms that the coexistence endemic equilibrium E_I is locally asymptotically stable. This equilibrium is characterized by the persistence of both strains in the population, with non-zero latent and infectious compartments for each strain. These numerical results further illustrate that the proposed generalized model captures a wide variety of well-known incidence functions, providing a comprehensive framework for the study of stability and coexistence dynamics in multi-strain epidemic models.

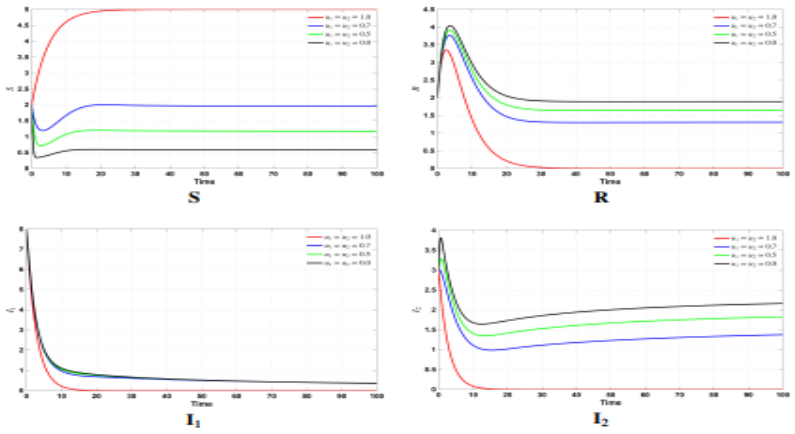

Fig. 2. Population dynamics at $\alpha = 1$ for various treatment parameters values.

Figure 2 displays the temporal dynamics of the four epidemiological compartments for the classical derivative case $\alpha = 1$, under different choices of the control functions u_1 and u_2. The simulations demonstrate that both infectious classes $I_1(t)$ and $I_2(t)$ reach non-zero steady states, indicating the coexistence of the two circulating strains, while the susceptible class $\mathcal{S}(t)$ converges to a positive equilibrium level. When infection recovery and the controls work together, the eliminated population $\mathcal{R}(t)$ also stabilizes. The local asymptotic stability of the coexistence equilibrium E_f, at which both strains continue to exist in the population, is confirmed by these numerical results. Furthermore, the impact of the controls is evident: changing the intensities of u_1 and u_2 alters the height and timing of the peaks in $I_1(t)$ and $I_2(t)$, emphasizing the function of the suggested intervention strategies in lowering each strain's infectious burden while preserving the system's overall stability.

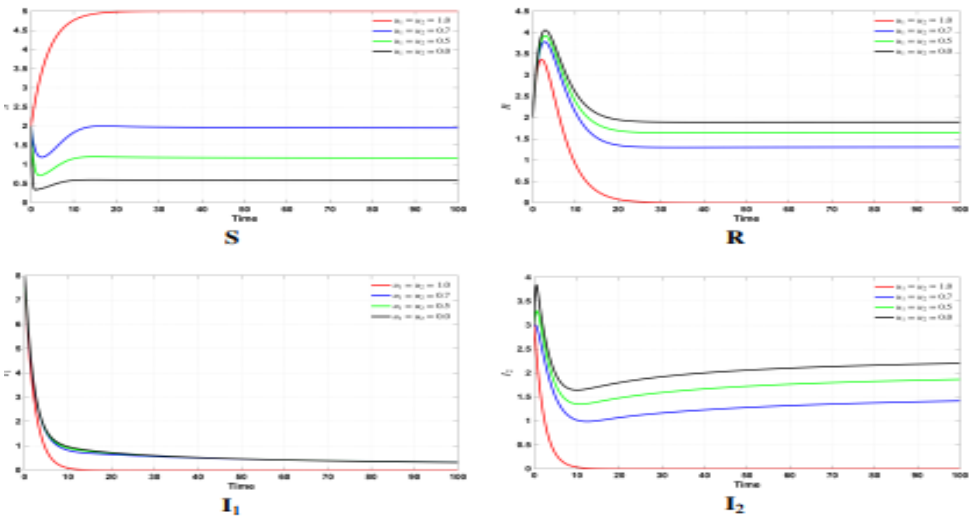

Fig. 3. Population dynamics at $\alpha = 0.9$ for various treatment parameters values.

Fig.3 presents the time evolution of the four compartments in the fractional-order case $\alpha = 0.9$, for various choices of the control parameters u_1 and u_2. In contrast to the classical case $\alpha = 1$, the use of a fractional derivative accounts for memory effects, which reduce the speed of epidemic dynamics and result in a smoother convergence toward equilibrium. The susceptible class $\mathcal{S}(t)$ and the removed class $\mathcal{R}(t)$ approach steady values comparable to those obtained in the integer-order case, while the infectious classes $I_1(t)$ and $I_2(t)$ exhibit slower decay or stabilization phases, reflecting the impact of memory effects on the ongoing transmission dynamics. The numerical results indicate that the coexistence endemic equilibrium E_t remains locally asymptotically stable for $\alpha = 0.9$, and highlight that the fractional-order formulation modifies the transient dynamics of the two-strain system without affecting the long-term persistence of both infections.

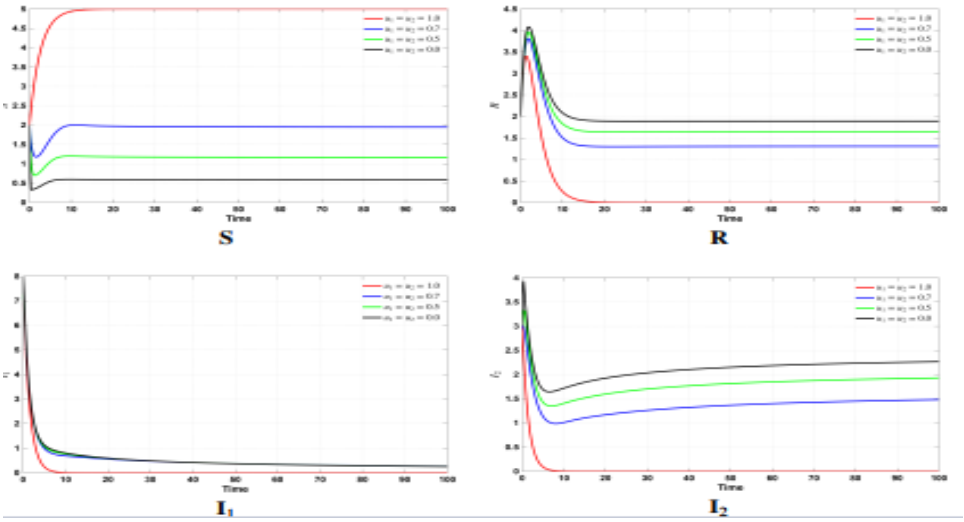

Fig. 4. Population dynamics at $\alpha = 0.7$ for various treatment parameters values.

Figure 4 depicts the dynamics of the four epidemiological compartments in the fractional-order setting $\alpha = 0.7$, for various configurations of the control parameters u_1 and u_2. A lower fractional order enhances memory effects, which decelerate the epidemic spread and lengthen the transient phase prior to equilibrium. The susceptible population $\mathcal{S}(t)$ and the removed population $\mathcal{R}(t)$ stabilize at levels similar to those observed for higher values of α, whereas the infectious classes $I_1(t)$ and $I_2(t)$ exhibit a markedly slower approach to their steady states. The numerical findings suggest that the coexistence endemic equilibrium E_t retains its local asymptotic stability for $\alpha = 0.7$, and underline the role of the fractionalorder derivative in shaping the timing and amplitude of infection peaks, without altering the long-term persistence of the two strains in the population.

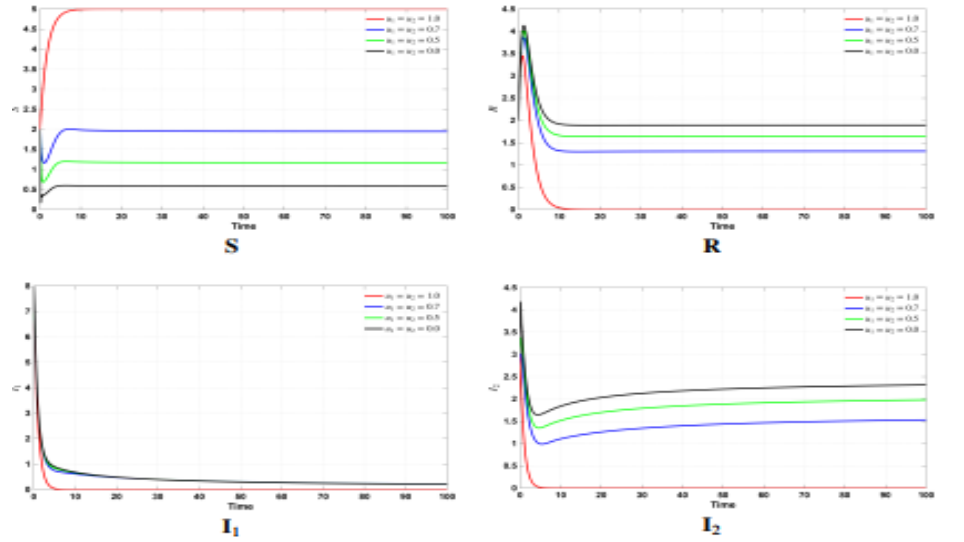

Fig. 5. Population dynamics at $\alpha = 0.5$ for various treatment parameters values

Fig. 5 presents the dynamics of the four epidemiological compartments for the fractional-order case $\alpha = 0.5$, considering different values of the control parameters u_1 and u_2. At this lower fractional order, the memory effects become even more pronounced, which further slows down the evolution of the system. The susceptible population $S(t)$ and the removed population $\mathcal{R}(t)$ stabilize at levels comparable to those obtained for higher values of α, while the infectious classes $T_1(t)$ and $T_2(t)$ require significantly more time

to approach their steady states. This prolonged transient behavior reflects the strong influence of past states on the current dynamics. The numerical simulations confirm that the coexistence endemic equilibrium E_t remains locally asymptotically stable when $\alpha = 0.5$ and they underline the impact of the fractional-order derivative on the timing and intensity of the epidemic peaks, while maintaining the persistence of both strains within the population. A comparative analysis of the system dynamics corresponding to the fractional orders $\alpha = 1.0, 0.9, 0.7$ and 0.5 highlights the significant role of memory effects in shaping the epidemic evolution. As α decreases, the convergence of the infectious classes $T_1(t)$ and $I_2(t)$ toward the coexistence endemic equilibrium E_t becomes slower, reflecting an increased influence of past states on the present dynamics. Meanwhile, the asymptotic levels of the susceptible class $S(t)$ and the removed class $\mathcal{R}(t)$ remain almost unaffected by variations in α. The most notable differences arise in the transient regime: decreasing the fractional order results in delayed and smoother infection peaks, as well as prolonged convergence times for both infectious compartments. These findings demonstrate that the fractional-order framework captures diverse memory-induced dynamics while maintaining the local asymptotic stability and persistence of the two strains. Overall, this comparison highlights the key role of fractional derivatives in representing memory-dependent epidemic processes and confirms that the control parameters u_1 and u_2 remain effective across various dynamical regimes.

6 Conclusion

In this work, we proposed and analyzed a fractional-order two-strain epidemic model of $SI_1I_2\mathcal{R}$ type, in which the transmission dynamics of both strains are governed by general incidence functions $f(S, I_1)$ and $h(S, I_2)$. The model also incorporates two constant treatment controls acting independently on each infectious class. By formulating the system using Caputo fractional derivatives of order $0 < \alpha \leq 1$, memory effects and hereditary characteristics of the epidemic process are considered, which cannot be captured by classical integer-order models.

We first established fundamental analytical properties of the system, including the existence, uniqueness, positivity, and boundedness of solutions. The mathematical analysis allowed us to determine the disease-free equilibrium, the strain-specific endemic equilibria, and the coexistence equilibrium. The basic reproduction numbers $R_{0,1}$ and $R_{0,2}$ were derived using the next-generation matrix approach and were shown to play a crucial role in determining the persistence or elimination of each strain. By constructing suitable Lyapunov functionals and under biologically reasonable assumptions on the general incidence functions, we established the global asymptotic stability of the equilibria under appropriate threshold conditions.

Furthermore, numerical simulations were carried out for several values of the fractional order. The results confirmed the analytical findings and highlighted the influence of memory effects on the epidemic dynamics. Smaller values of the fractional order α were observed to slow down the convergence of solutions toward their equilibrium states and to delay the peak of infection, while the long-term stability properties remained unchanged. The treatment parameters u_1 and u_2 are shown to effectively reduce the number of infected individuals for each strain. The proposed fractional-order model offers a flexible and realistic framework for analyzing the interaction and coexistence of multiple strains under nonlinear transmission mechanisms. By combining general incidence functions, strain-dependent treatment controls, and memory effects, the model enhances its descriptive capability and provides meaningful insights into the long-term behavior of multi-strain epidemics. Future work could extend this framework by including spatial diffusion, vaccination strategies, time varying control measures, or stochastic effects, thereby improving the understanding of complex epidemic dynamics. Although constant control parameters are considered in this study, time dependent control strategies may provide a more realistic description of intervention policies and will be investigated in future work.

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