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Stability Analysis of Fractional-Order SIR Model with General Incidence Rate and Treatment

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Abstract

In this work, we study a fractional two-strain epidemic SI_1I_2R model with different incidence rates: A general incidence rate for the first strain and a bilinear incidence rate for the second strain, incorporating treatment of each strain. The proposed model consists of four ordinary differential equations describing interactions among the susceptible, Infected and recovered individuals. Our first mathematical study focuses of proving the existence and uniqueness of positive solutions. To show the global stability of the model equilibria, suitable Lyapunov functions are used. Numerical simulations are performed to support the analytical results and to show the effect of the fractional derivative on convergence toward equilibria. Moreover, the role of treatment efficiency in minimizing the density of infected individuals is studied.

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

2010 Mathematics Subject Classification: 05C10, 05C35, 05C57

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 [46], human immunodeficiency virus HIV[7, 23, 39, 26, 5], COVID-19[19, 44, 27, 29], hepatitis B infection (HBV)[51, 18, 22] and hepatitis C infection (HCV)[42, 40, 34], exhibit multiple both or competing strains that can significantly alter disease dynamics and control strategies [6, 48, 50]. Consequently, 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

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treatment efficiency [15, 44, 17, 53]. Among the various compartmental frameworks, SIR models dividing the population into susceptible ($\mathcal{S}$), infectious ($\mathcal{I}$), and recovered ($\mathcal{R}$) classes remain particularly useful for directly transmitted diseases, especially when the latent period can be neglected or suitably approximated. In multi-strain extensions, the infectious population is subdivided into two or more compartments, each corresponding to a specific strain, leading to the SI_1I_2R framework considered in this work. The classical SIR model, introduced by Kermack and McKendrick in 1927 [28], has served as a fundamental tool for determining key epidemiological indicators such as the basic reproduction number R_0, peak infection prevalence, and herd immunity thresholds. Building on this seminal work, numerous extensions have been proposed to account for multiple strains, age structure, vaccination strategies, and optimal control interventions [9, 32, 24, 38, 14]. In this context, our work focuses on the interaction of two competing strains under general transmission functions.

A central component of epidemic modeling is the incidence function, which governs how susceptible individuals become infected. While the classical bilinear form $\beta\mathcal{S}\mathcal{I}$ is commonly used [52], it often fails to capture saturation effects or nonlinear behaviors arising from contact limitations or behavioral changes. Throughout this work, we formulate the model using a mixed incidence function: A general function $f(\mathcal{S},\mathcal{I}_1)\mathcal{I}_1$ for the first strain, which allows for nonlinear and saturating effects, and the classical bilinear form $\beta S\mathcal{I}_2$ for the second strain, which is appropriate for direct transmission under mass-action assumptions. This formulation enables a more flexible yet tractable model structure. In particular, the function $f(\mathcal{S},\mathcal{I}_1)$ may exhibit non-monotonic behavior with respect to $\mathcal{I}_1$, allowing the model to capture saturation effects and behavioral responses, especially when the number of infected individuals becomes large.

Most existing studies employ integer-order differential equations, which describe the average disease dynamics but may not capture memory effects, delays, or hereditary properties inherent in biological processes, previous multi-strain SEIR models with general incidence and treatment [4] provide important insights but assume instantaneous transitions, limiting their ability to model long-term memory or delayed immune responses. To overcome these limitations, we adopt fractional-order derivatives in the sense of Caputo, which naturally incorporate memory and hereditary effects, providing a more realistic framework for epidemics with delayed or history-dependent dynamics [2, 37, 36, 8, 41, 21]. Recent studies have demonstrated the effectiveness of fractional-order and fractal-fractional models in capturing environmental effects, social distancing, seasonality and memory-dependent dynamics in epidemic and biological systems [11, 12, 13]. More recently, several works have addressed fractional modeling of viral infections and biological systems using advanced analytical and numerical approaches [35, 1, 30, 45, 33, 16, 31].

The primary contribution of this work is the formulation and analysis of a fractional-order SI_1I_2R model with a mixed incidence function, combining a general function for one strain and a bilinear form for the other, while incorporating memory effects through fractional derivatives and explicitly including two control variables, u_1 and u_2, representing intervention strategies. We derive conditions for existence, positivity and global stability of the disease-free and endemic equilibria, emphasizing the role of the basic reproduction numbers $R_{0,1}$ and $R_{0,2}$. Moreover, we provide a Lyapunov-based stability analysis under biologically reasonable assumptions on the transmission function $f(\mathcal{S},\mathcal{I}_1)$, extending previous stability results established for fractional-order SIR models with bilinear incidence rate [43] to the fractional-order SI_1I_2R context.

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

$$\begin{cases} {}^{C}D_t^{\alpha}\mathcal{S}(t) = \lambda - (1-u_1)\, f(\mathcal{S},\mathcal{I}_1)\,\mathcal{I}_1 - (1-u_2)\,\beta\mathcal{S}\mathcal{I}_2 - \mu\mathcal{S}, \\ {}^{C}D_t^{\alpha}\mathcal{I}_1(t) = (1-u_1)\, f(\mathcal{S},\mathcal{I}_1)\,\mathcal{I}_1 - (\sigma_1+\mu)\,\mathcal{I}_1, \\ {}^{C}D_t^{\alpha}\mathcal{I}_2(t) = (1-u_2)\,\beta\mathcal{S}\mathcal{I}_2 - (\sigma_2+\mu)\,\mathcal{I}_2, \\ {}^{C}D_t^{\alpha}\mathcal{R}(t) = \sigma_1\mathcal{I}_1 + \sigma_2\mathcal{I}_2 - \mu\mathcal{R}. \end{cases} \tag{1.1}$$

with initial conditions

$$\mathcal{S}_0 \geq 0, \quad \mathcal{I}_{1,0} \geq 0, \quad \mathcal{I}_{2,0} \geq 0, \quad \mathcal{R}_0 \geq 0.$$

Here, $\mathcal{S}(t)$ denotes the susceptible population, $\mathcal{I}_1(t)$ and $\mathcal{I}_2(t)$ the infectious classes and $\mathcal{R}(t)$ the recovered class. The parameter λ represents the recruitment rate, μ the natural mortality, σ_1 and σ_2 the recovery rates. The general incidence functions $f(\mathcal{S}, \mathcal{I}_1)$ describe the transmission dynamics of each strain and satisfy standard biological conditions:

(H1) $f \in C^1(\mathbb{R}^2_+, \mathbb{R}_+)$, with $f(0, \mathcal{I}_1) = 0, \quad \forall \mathcal{I}_1, \mathcal{I}_2 \geq 0;$

(H2) $\dfrac{\partial f}{\partial \mathcal{S}}(\mathcal{S}, \mathcal{I}_1) > 0, \quad \forall \mathcal{S} > 0, \forall \mathcal{I}_1, \mathcal{I}_2 \geq 0;$

(H3) $\dfrac{\partial f}{\partial \mathcal{I}_1}(\mathcal{S}, \mathcal{I}_1) \leq 0, \quad \forall \mathcal{S} \geq 0, \mathcal{I}_1, \mathcal{I}_2 \geq 0.$

All the model (1.1) parameters are given in Fig.(1)

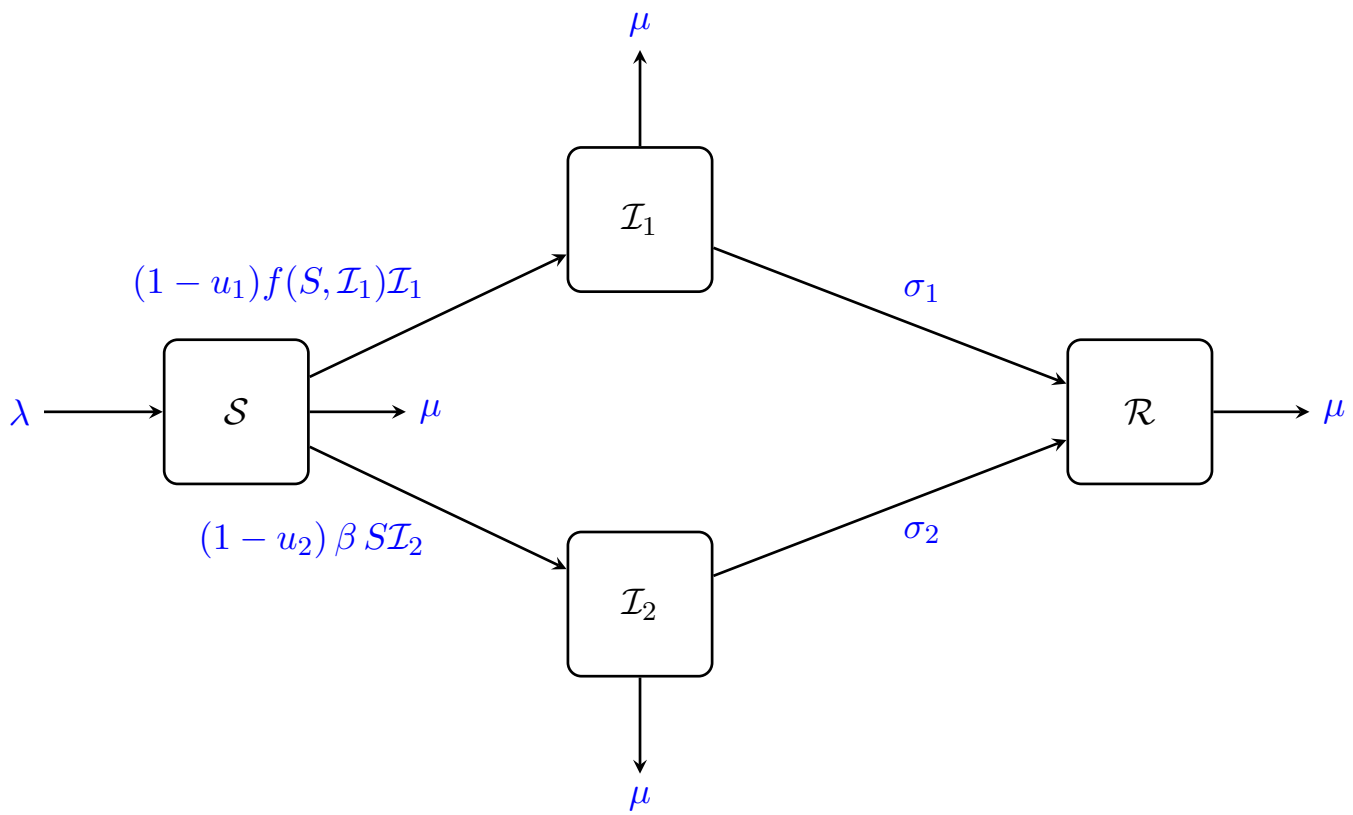

Figure 1: The flowchart of the two strain SI_1I_2R model.

This present work is divided into five sections: Section 2 provides the foundational definitions of the fractional derivative. Then, Section 3 presented the existence, uniqueness, positivity and boundedness of the fractional model. In Section 4, we prove the global stability of the model (1.1) equilibria. Numerical simulations and some discussions are given in section 5. The last section concludes this work.

2. Preliminary results

Definition 2.1. [10] 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

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

Definition 2.2 (Mittag–Leffler function)**.** [25] 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:

$$D^\alpha X(t) = f(X(t)), \quad 0 < \alpha \leq 1. \tag{2.1}$$

With $X(0) = X_0$, $X_0 \in \mathbb{R}^n$ and $0 < \alpha < 1$. To demonstrate the global stability of solutions of system (2.1) we will need the following lemma:

Lemma 2.3. *If the function f satisfies the two following conditions:*

1. *$f(X)$ and $\dfrac{\partial f}{\partial X}(X)$ 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, the system (2.1) has a only solution defined on $\mathbb{R}^n_+$.

Lemma 2.4. *Let $X(t)$ be a solution of the Caputo fractional differential system ${}^C D_t^\alpha X(t) = \mathcal{F}(X(t)), \quad 0 < \alpha \leq 1$, with initial condition $X(0) \in \mathbb{R}^n_+$.*

If the vector field $\mathcal{F}$ satisfies $\mathcal{F}_i(X) \geq 0 \quad$ whenever $X_i = 0, \quad i = 1, \ldots, n$, then the nonnegative orthant $\mathbb{R}^n_+$ is positively invariant. That is, the solution satisfies $X(t) \in \mathbb{R}^n_+$ for all $t > 0$.

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: $\mathcal{S}(0) = \mathcal{S}_0$, $\mathcal{I}_1(0) = \mathcal{I}_{1,0}$, $\mathcal{I}_2(0) = \mathcal{I}_{2,0}$, $\mathcal{R}(0) = \mathcal{R}_0$, with all components in $\mathbb{R}_+$. The results are summarized in the following proposition.

Proposition 3.1. *For any non-negative initial conditions $\mathcal{S}_0$, $\mathcal{I}_{1,0}$, $\mathcal{I}_{2,0}$ and $\mathcal{R}_0$, the fractional system*

$${}^C D_t^\alpha X(t) = \mathcal{F}(X(t)), \qquad 0 < \alpha \leq 1,$$

admits a unique global solution which remains positive and bounded on $[0, \infty[$.

Proof. We rewrite system (1.1) in compact vector form:

$$X(t) = \begin{pmatrix} \mathcal{S} \\ \mathcal{I}_1 \\ \mathcal{I}_2 \\ \mathcal{R} \end{pmatrix}, \qquad \mathcal{F}(X) = \begin{pmatrix} \lambda - (1-u_1) f(\mathcal{S}, \mathcal{I}_1)\mathcal{I}_1 - (1-u_2)\beta \mathcal{S}\mathcal{I}_2 - \mu \mathcal{S} \\ (1-u_1) f(\mathcal{S}, \mathcal{I}_1)\mathcal{I}_1 - (\sigma_1 + \mu)\mathcal{I}_1 \\ (1-u_2)\beta \mathcal{S}\mathcal{I}_2 - (\sigma_2 + \mu)\mathcal{I}_2 \\ \sigma_1 \mathcal{I}_1 + \sigma_2 \mathcal{I}_2 - \mu \mathcal{R} \end{pmatrix}.$$

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

$${}^C D_t^\alpha X(t) = \mathcal{F}(X(t)), \quad X(0) = X_0$$

admits a unique local solution.

Now we will prove the positivity of model solutions. First, we will assume that all model parameters are positive, we have

$$\begin{cases} {}^C D_t^\alpha \mathcal{S}\big|_{\mathcal{S}=0} = \lambda > 0, \\ {}^C D_t^\alpha \mathcal{I}_1\big|_{\mathcal{I}_1=0} = (1-u_1) f(S, \mathcal{I}_1)\mathcal{I}_1 - (\sigma_1 + \mu)0 = 0, \\ {}^C D_t^\alpha \mathcal{I}_2\big|_{\mathcal{I}_2=0} = (1-u_2)\beta S \mathcal{I}_2 - (\sigma_2 + \mu)0 = 0, \\ {}^C D_t^\alpha \mathcal{R}\big|_{\mathcal{R}=0} = \sigma_1 \mathcal{I}_1 + \sigma_2 \mathcal{I}_2 \geq 0. \end{cases}$$

Thus, the nonnegative orthant $\mathbb{R}^4_+$ is positively invariant. More rigorously, using standard invariance results for Caputo fractional-order systems Lemma (2.4), the nonnegative orthant $\mathbb{R}^4_+$ is positively invariant. Since the vector field $\mathcal{F}$ is nonnegative on the boundary of $\mathbb{R}^4_+$, any solution starting with nonnegative initial conditions remains nonnegative for all $t > 0$.

Finally, we will show the boundedness of the solutions. Let the total population

$$\mathcal{N}(t) = \mathcal{S}(t) + \mathcal{I}_1(t) + \mathcal{I}_2(t) + \mathcal{R}(t).$$

Summing the four equations yields: ${}^{C}D_t^{\alpha}\mathcal{N}(t) = \lambda - \mu\mathcal{N}(t)$. This linear Caputo equation has solution

$$\mathcal{N}(t) = \frac{\lambda}{\mu} + \Big(\mathcal{N}(0) - \frac{\lambda}{\mu}\Big)E_{\alpha}(-\mu t^{\alpha}),$$

where $E_{\alpha}(\cdot)$ denotes the Mittag–Leffler function. Since $0 < E_{\alpha}(-\mu t^{\alpha}) < 1$ for all $t > 0$, we obtain

$$0 \le \mathcal{N}(t) \le \max\Big\{\mathcal{N}(0), \frac{\lambda}{\mu}\Big\}.$$

Thus, the biologically feasible region

$$\mathcal{H} = \Big\{(\mathcal{S}, \mathcal{I}_1, \mathcal{I}_2, R) \in \mathbb{R}^4_+ : \mathcal{S} + \mathcal{I}_1 + \mathcal{I}_2 + \mathcal{R} \le \frac{\lambda}{\mu}\Big\}$$

is positively invariant. Therefore, $X(t)$ remains bounded for all $t > 0$.

The Lipschitz continuity of $\mathcal{F}$ together with invariance of $\mathcal{H}$ provide uniqueness of the solution. □

4. The steasy states

This subsection establishes the existence of the disease-free equilibrium and the endemic equilibria of the fractional SI_1I_2R model. Since the equation for $\mathcal{R}$ depends only on $\mathcal{I}_1$, $\mathcal{I}_2$ and $\mathcal{R}$, the equilibria can be determined from the subsystem satisfied by $\mathcal{S}$, $\mathcal{I}_1$, $\mathcal{I}_2$. Thus, the system (1.1) reduces to

$$\begin{cases} {}^{C}D_t^{\alpha}\mathcal{S}(t) = \lambda - (1-u_1)f(\mathcal{S},\mathcal{I}_1)I_1 - (1-u_2)\beta\mathcal{S}\mathcal{I}_2 - \mu S, \\ {}^{C}D_t^{\alpha}I_1(t) = (1-u_1)f(S,I_1)I_1 - (\sigma_1+\mu)I_1, \\ {}^{C}D_t^{\alpha}\mathcal{I}_2(t) = (1-u_2)\beta\mathcal{S}\mathcal{I}_2 - (\sigma_2+\mu)\mathcal{I}_2, \end{cases} \tag{4.1}$$

with the recovered population given by

$$\mathcal{R}(t) = \mathcal{N}(t) - \mathcal{S}(t) - \mathcal{I}_1(t) - \mathcal{I}_2(t),$$

where $\mathcal{N}(t)$ satisfies the scalar fractional equation: ${}^{C}D_t^{\alpha}\mathcal{N}(t) = \lambda - \mu\mathcal{N}(t)$.

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. Let F denote the matrix of new infection terms and V the matrix describing transition terms among infected compartments for the reduced system (4.1). Evaluated at the disease-free equilibrium, the matrices F and V for the infected variables $\mathcal{I}_1$, $\mathcal{I}_2$ are

$$F = \begin{pmatrix} (1-u_1)\, f(\frac{\lambda}{\mu}, 0) & 0 \\ 0 & (1-u_2)\,\beta\,\frac{\lambda}{\mu} \end{pmatrix}, \qquad V = \begin{pmatrix} \sigma_1+\mu & 0 \\ 0 & \sigma_2+\mu \end{pmatrix}.$$

Thus

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

The basic reproduction number is the spectral radius of the matrix FV^{-1}, hence

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

where

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

$R_{0,1}$ is the base reproduction number associated with the first strain and $R_{0,2}$ is associated with the other strain.

The basic reproduction number $R_0 = \max\{R_{0,1}, R_{0,2}\}$ represents the threshold parameter governing disease invasion in the population. If $R_0 < 1$, the disease-free equilibrium is stable and both strains die out. If $R_0 > 1$, at least one strain can invade the population and persist.

Moreover, $R_{0,1}$ and $R_{0,2}$ describe the transmission potential of each strain. If $R_{0,1} > R_{0,2}$, the first strain tends to dominate, whereas if $R_{0,2} > R_{0,1}$, the second strain becomes dominant. If both exceed unity, coexistence of the two strains may occur.

Theorem 4.1. *The system* (4.1) *admits the disease-free equilibrium $\mathcal{E}_f$ and three endemic equilibria: the strain 1 endemic equilibrium $\mathcal{E}_{S_1}$, the strain 2 endemic equilibrium $\mathcal{E}_{S_2}$, and the both equilibrium $\mathcal{E}_{S_t}$. Moreover:*

- *If $R_{0,1} > 1$, then the strain 1 endemic equilibrium $\mathcal{E}_{S_1}$ exists.*
- *If $R_{0,2} > 1$, then the strain 2 endemic equilibrium $\mathcal{E}_{S_2}$ exists.*
- *If $\min\{R_0^1, R_0^2\} > 1$, then the both strains endemic equilibrium $\mathcal{E}_{S_t}$ exists.*

Proof. In order to find the equilibria of the system (4.1), we solve the following

$$\begin{cases} 0 = \lambda - (1-u_1)f(\mathcal{S},\mathcal{I}_1)\mathcal{I}_1 - (1-u_2)\beta\mathcal{S}\mathcal{I}_2 - \mu\mathcal{S}, \\ 0 = (1-u_1)f(\mathcal{S},\mathcal{I}_1)\mathcal{I}_1 - (\sigma_1+\mu)\mathcal{I}_1, \\ 0 = (1-u_2)\beta\mathcal{S}\mathcal{I}_2 - (\sigma_2+\mu)\mathcal{I}_2, \\ 0 = \sigma_1\mathcal{I}_1 + \sigma_2\mathcal{I}_2 - \mu\mathcal{R}. \end{cases} \tag{4.2}$$

1. Disease-free equilibrium. When $\mathcal{I}_1 = \mathcal{I}_2 = 0$ the system admits the disease-free equilibrium: $\mathcal{E}_f = \left(\frac{\lambda}{\mu}, 0, 0, 0\right)$.

2. Strain 1 endemic equilibrium. When $\mathcal{I}_1 > 0$ and $\mathcal{I}_2 = 0$. From the second equation of (4.2) we get

$$(1-u_1)f(\mathcal{S},\mathcal{I}_1) = \sigma_1+\mu.$$

From the first equation we have: $\lambda - \mu\mathcal{S} = (\sigma_1+\mu)\mathcal{I}_1$, hence $\mathcal{I}_1 = \frac{\lambda-\mu\mathcal{S}}{\sigma_1+\mu}$.
Define the function $\Psi : [0,\infty) \to \mathbb{R}$ by

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

Then,

$$\frac{\partial \Psi(\mathcal{S})}{\partial \mathcal{S}} = (1-u_1)\left[\frac{\partial f}{\partial \mathcal{S}}(\mathcal{S}, I_1(\mathcal{S})) + \frac{\partial f}{\partial I_1}(\mathcal{S}, (\mathcal{I}_1)(\mathcal{S})) \cdot \frac{\partial \Psi(\mathcal{I}_1)}{\partial \mathcal{S}}\right],$$

$$with, \qquad \mathcal{I}_1(\mathcal{S}) := \frac{\lambda - \mu\mathcal{S}}{\sigma_1 + \mu}, \qquad \frac{\partial \mathcal{I}_1(\mathcal{S})}{\partial \mathcal{S}} = -\frac{\mu}{\sigma_1 + \mu}.$$

Hence,

$$\frac{\partial \Psi(\mathcal{S})}{\partial \mathcal{S}} = (1-u_1)\left[\frac{\partial f}{\partial \mathcal{S}}(\mathcal{S}, \mathcal{I}_1(\mathcal{S})) - \frac{\mu}{\sigma_1 + \mu}\frac{\partial f}{\partial I_1}(\mathcal{S}, \mathcal{I}_1(\mathcal{S}))\right].$$

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

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

Moreover,

$$\Psi(0) = (1-u_1)f\left(0, \frac{\lambda}{\sigma_1 + \mu}\right) - (\sigma_1 + \mu) = -(\sigma_1 + \mu) < 0,$$

and

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

Therefore, if $R_{0,1} > 1$ then $\Psi(\frac{\lambda}{\mu}) > 0$. Since Ψ is continuous and nondecreasing, there exists a unique $\mathcal{S}_1^* \in \left[0, \frac{\lambda}{\mu}\right]$ such that $\Psi(\mathcal{S}_1^*) = 0$. Hence, a unique strain 1 endemic equilibrium exists:

$$\mathcal{E}_{S_1} = \left(\mathcal{S}_1^*, \frac{\lambda - \mu\mathcal{S}_1^*}{\sigma_1 + \mu}, 0, \frac{\sigma_1(\lambda - \mu\mathcal{S}_1^*)}{\mu(\sigma_1 + \mu)}\right),$$

with $\mathcal{S}_1^* \in [0, \lambda/\mu]$ and $\mathcal{I}_1^* > 0$.

3. Strain 2 endemic equilibrium. When $\mathcal{I}_2 > 0$ and $\mathcal{I}_1 = 0$. From the third equation of (4.2) we have: $(1-u_2)\beta\mathcal{S} = \sigma_2 + \mu$, then, $\mathcal{S}_2^* = \frac{\sigma_2+\mu}{(1-u_2)\beta}$,then, $\mathcal{I}_2 = \frac{\lambda-\mu\mathcal{S}}{\sigma_2+\mu}$.
Define now

$$\Phi(\mathcal{S}) = (1-u_2)\beta\mathcal{S} - (\sigma_2 + \mu),$$

ten,

$$\frac{\partial \Phi(\mathcal{S})}{\partial \mathcal{S}} = (1-u_2)\beta > 0.$$

We have: $\Phi(0) = -(\sigma_2 + \mu) < 0$ and, $\Phi\left(\frac{\lambda}{\mu}\right) = (\sigma_2 + \mu)(R_{0,2} - 1)$.
Hence if $R_{0,2} > 1$ there exists a unique $\mathcal{S}_2^* \in \left[0, \frac{\lambda}{\mu}\right]$, solving $\Phi(\mathcal{S}_2^*) = 0$, yielding the strain 2 endemic equilibrium

$$\mathcal{E}_{S_2} = \left(\mathcal{S}_2^*, 0, \frac{\lambda - \mu\mathcal{S}_2^*}{\sigma_2 + \mu}, \frac{\sigma_2(\lambda - \mu\mathcal{S}_2^*)}{\mu(\sigma_2 + \mu)}\right).$$

4. Both endemic equilibrium: When $\mathcal{I}_1 > 0$ and $\mathcal{I}_2 > 0$. From the second and third equations we obtain

$$(1-u_1)f(\mathcal{S}, \mathcal{I}_1) = \sigma_1 + \mu, \qquad (1-u_2)\beta\mathcal{S} = \sigma_2 + \mu.$$

Then, $\mathcal{S}_t^* = \frac{\sigma_2+\mu}{(1-u_2)\beta}$. Substituting into the first equation provides the linear relation

$$(\sigma_1 + \mu)\mathcal{I}_1 + (\sigma_2 + \mu)\mathcal{I}_2 = \lambda - \mu\mathcal{S}_t^*.$$

A both equilibrium with positive components exists if and only if both $R_{0,1} > 1$ and $R_{0,2} > 1$, which ensures the right-hand side is positive and the pair $(\mathcal{I}_1, \mathcal{I}_2)$ can be chosen positive satisfying the above relation. The both equilibrium is then

$$\mathcal{E}_{S_t} = \left(\mathcal{S}_t^*,\, \mathcal{I}_{1,t}^*,\, \mathcal{I}_{2,t}^*,\, \frac{\sigma_1 \mathcal{I}_{1,t}^* + \sigma_2 \mathcal{I}_{2,t}^*}{\mu}\right),$$

with $\mathcal{I}_{1,t}^*, \mathcal{I}_{2,t}^* > 0$.

□

4.2. Global stability of equilibria

4.2.1. Global stability analysis of the disease-free equilibrium

Theorem 4.2. *If $R_{0,1} \leq 1$, then the disease-free equilibrium $\mathcal{E}_f$ with is globally asymptotically stable.*

Proof. In $\mathbb{R}_+$, we first consider the Lyapunov function as follows:

$$\mathcal{P}_0(\mathcal{S}, \mathcal{I}_1, \mathcal{I}_2) = \mathcal{S} - \mathcal{S}^0 - \int_{\mathcal{S}^0}^{\mathcal{S}} \frac{f(\mathcal{S}^0, 0)}{f(X, 0)}\, dX \;+\; \mathcal{I}_1 + \mathcal{I}_2,$$

where $S^0 = \dfrac{\lambda}{\mu}$ denotes the susceptible population at the disease-free equilibrium.

The integral is well defined by hypothesis (H1) and (H3) and $f(\cdot, 0) > 0$ for $\mathcal{S} > 0$.

Using properties of the Caputo derivative and differentiating along solutions of the SI_1I_2R system yields

$$\begin{aligned}
{}^C D_t^\alpha \mathcal{P}_0 &= {}^C D_t^\alpha \mathcal{S} \;-\; \frac{f(\mathcal{S}^0, 0)}{f(\mathcal{S}, 0)}\, {}^C D_t^\alpha \mathcal{S} \;+\; {}^C D_t^\alpha \mathcal{I}_1 \;+\; {}^C D_t^\alpha \mathcal{I}_2 \\
&= \Big(1 - \frac{f(\mathcal{S}^0, 0)}{f(\mathcal{S}, 0)}\Big)\big(\lambda - (1-u_1) f(\mathcal{S}, \mathcal{I}_1)\mathcal{I}_1 - (1-u_2)\beta \mathcal{S}\mathcal{I}_2 - \mu \mathcal{S}\big) \\
&\quad + (1-u_1) f(\mathcal{S}, \mathcal{I}_1)\mathcal{I}_1 - (\sigma_1 + \mu)\mathcal{I}_1 + (1-u_2)\beta \mathcal{S}\mathcal{I}_2 - (\sigma_2 + \mu)\mathcal{I}_2.
\end{aligned}$$

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

$$\begin{aligned}
{}^C D_t^\alpha \mathcal{P}_0 = \mu \mathcal{S}^0 \Big(1 - \frac{\mathcal{S}}{\mathcal{S}^0}\Big)\Big(1 - \frac{f(\mathcal{S}^0, 0)}{f(\mathcal{S}, 0)}\Big) + \mathcal{I}_1 \left((1-u_1)\frac{f(\mathcal{S}, \mathcal{I}_1)}{f(\mathcal{S}, 0)}\, f(\mathcal{S}^0, 0) - (\sigma_1 + \mu)\right) \\
+ \mathcal{I}_2 \left((1-u_2)\frac{\beta \mathcal{S}}{\beta \mathcal{S}^0}\, \beta \mathcal{S}^0 - (\sigma_2 + \mu)\right).
\end{aligned}$$

Using the definitions of the reproduction numbers at the DFE,

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

we can bound the last two terms to obtain

$${}^C D_t^\alpha \mathcal{P}_0 \leq \mu \mathcal{S}^0 \Big(1 - \frac{\mathcal{S}}{\mathcal{S}^0}\Big)\Big(1 - \frac{f(\mathcal{S}^0, 0)}{f(\mathcal{S}, 0)}\Big) + \mathcal{I}_1 \big(R_{0,1}\, \frac{f(\mathcal{S}, \mathcal{I}_1)}{f(\mathcal{S}, 0)} - 1\big)(\sigma_1 + \mu) + \mathcal{I}_2 \big(R_{0,2}\, \frac{\mathcal{S}}{\mathcal{S}^0} - 1\big)(\sigma_2 + \mu).$$

We now analyze two cases.

– If $\mathcal{S} \leq \mathcal{S}^0$. By (H2) and (H3) we have $\dfrac{f(\mathcal{S}, \mathcal{I}_1)}{f(\mathcal{S}, 0)} \leq 1$ and $\dfrac{\mathcal{S}}{\mathcal{S}^0} \leq 1$. Hence

$$\mathcal{I}_1 \big(R_{0,1}\, \frac{f(\mathcal{S}, \mathcal{I}_1)}{f(\mathcal{S}, 0)} - 1\big)(\sigma_1 + \mu) \leq \mathcal{I}_1 (R_{0,1} - 1)(\sigma_1 + \mu),$$

and

$$\mathcal{I}_2\big(R_{0,2}\,\frac{\mathcal{S}}{\mathcal{S}^0}-1\big)(\sigma_2+\mu)\le \mathcal{I}_2(R_{0,2}-1)(\sigma_2+\mu).$$

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

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

Therefore, if $R_{0,1}\le 1$ and $R_{0,2}\le 1$, each term is nonpositive and ${}^{C}D_t^{\alpha}\mathcal{P}_0\le 0$.

– If $\mathcal{S}>\mathcal{S}^0$. By (H2) we have $\dfrac{f(\mathcal{S},\mathcal{I}_1)}{f(\mathcal{S},0)}\ge 1$ and $\dfrac{\mathcal{S}}{\mathcal{S}^0}>1$, while $\dfrac{f(\mathcal{S}^0,0)}{f(\mathcal{S},0)}<1$, so the first term is again negative. Using $R_{0,1}\le 1$ and $R_{0,2}\le 1$ we obtain

$$\mathcal{I}_1\big(R_{0,1}\,\frac{f(\mathcal{S},\mathcal{I}_1)}{f(\mathcal{S},0)}-1\big)(\sigma_1+\mu)\le 0,\quad \mathcal{I}_2\big(R_{0,2}\,\frac{\mathcal{S}}{\mathcal{S}^0}-1\big)(\sigma_2+\mu)\le 0,$$

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

$${}^{C}D_t^{\alpha}\mathcal{P}_0(\mathcal{S},\mathcal{I}_1,\mathcal{I}_2)\le 0,$$

and equality holds only at the $\mathcal{E}_f$. By the direct Lyapunov method for Caputo fractional systems, this implies that $\mathcal{E}_f$ 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 $\mathcal{E}_{s_1}$:

$$\left(1-\frac{f(\mathcal{S},\mathcal{I}_1)}{f(\mathcal{S},\mathcal{I}^*_{1,s_1})}\right)\left(\frac{f(\mathcal{S},\mathcal{I}^*_{1,s_1})}{f(\mathcal{S},\mathcal{I}_1)}-\frac{\mathcal{I}_1}{\mathcal{I}^*_{1,s_1}}\right)\le 0,\qquad \forall\,\mathcal{S},\mathcal{I}_1>0. \tag{4.3}$$

Theorem 4.3. *If $R_{0,2}\le 1<R_{0,1}$, then the strain–1 endemic equilibrium $\mathcal{E}_{s_1}$ is globally asymptotically stable.*

Proof. We introduce the Lyapunov function

$$\mathcal{P}_1(\mathcal{S},\mathcal{I}_1,\mathcal{I}_2,\mathcal{R})=\mathcal{S}-\mathcal{S}^*_1-\int_{\mathcal{S}^*_1}^{\mathcal{S}}\frac{f(\mathcal{S}^*_1,\mathcal{I}^*_{1,s_1})}{f(X,\mathcal{I}^*_{1,s_1})}\,dX+\mathcal{I}^*_{1,s_1}\left(\frac{\mathcal{I}_1}{\mathcal{I}^*_{1,s_1}}-\ln\frac{\mathcal{I}_1}{\mathcal{I}^*_{1,s_1}}-1\right)+\mathcal{I}_2+\mathcal{R}.$$

Computing its Caputo derivative, we obtain

$$\begin{aligned}{}^{C}D_t^{\alpha}\mathcal{P}_1=\;&\mu(\mathcal{S}^*_1-\mathcal{S})\left(1-\frac{f(\mathcal{S}^*_1,\mathcal{I}^*_{1,s_1})}{f(\mathcal{S},\mathcal{I}^*_{1,s_1})}\right)+(\sigma_1+\mu)\,\mathcal{I}^*_{1,s_1}-(\sigma_1+\mu)\,\mathcal{I}_1\left(1-\frac{\mathcal{I}^*_{1,s_1}}{\mathcal{I}_1}\right)\\&-(1-u_1)f(\mathcal{S},\mathcal{I}_1)\mathcal{I}_1\left(\frac{\mathcal{I}^*_{1,s_1}}{\mathcal{I}_1}-\frac{f(\mathcal{S}^*_1,\mathcal{I}^*_{1,s_1})}{f(\mathcal{S},\mathcal{I}^*_{1,s_1})}\right)-\mu\,\mathcal{R}+\mathcal{I}_2\left((1-u_2)\beta\mathcal{S}-\mu\right)\end{aligned} \tag{4.4}$$

We now reorganize the nonlinear terms in $\mathcal{I}_1$. Write

$$(\sigma_1+\mu)\mathcal{I}^*_{1,s_1}=(1-u_1)f(\mathcal{S}^*_1,\mathcal{I}^*_{1,s_1})\,\mathcal{I}^*_{1,s_1}.$$

Using this identity and collecting all contributions involving $(1-u_1)f(\mathcal{S},\mathcal{I}_1)\mathcal{I}_1$, we obtain the compact factorization

$$(1-u_1)\mathcal{I}^*_{1,s_1}\left[\xi(\mathcal{S},\mathcal{I}_1)+\Theta(\mathcal{S},\mathcal{I}_1)\right],$$

where the first bracket

$$\xi(\mathcal{S},\mathcal{I}_1)=\frac{f(\mathcal{S}_1^*,\mathcal{I}_{1,s_1}^*)}{f(\mathcal{S},\mathcal{I}_{1,s_1}^*)}-\frac{\mathcal{I}_1}{\mathcal{I}_{1,s_1}^*},$$

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

$$\Theta(\mathcal{S},\mathcal{I}_1)=\frac{f(\mathcal{S},\mathcal{I}_{1,s_1}^*)}{f(\mathcal{S},\mathcal{I}_1)}+\frac{f(\mathcal{S},\mathcal{I}_1)}{f(\mathcal{S},\mathcal{I}_{1,s_1}^*)}\frac{\mathcal{I}_1}{\mathcal{I}_{1,s_1}^*}-\frac{\mathcal{I}_1}{\mathcal{I}_{1,s_1}^*}-1.$$

By the arithmetic–geometric inequality,

$$\Theta(\mathcal{S},\mathcal{I}_1)\le 0.$$

Hence all nonlinear terms involving $\mathcal{I}_1$ are nonpositive.

For the $\mathcal{I}_2$–component, using again MA–MG and the condition $R_{0,2}\le 1$, one obtains

$$\mathcal{I}_2\left((1-u_2)\beta\mathcal{S}-\mu\right)-(1-u_2)\beta\mathcal{S}\mathcal{I}_2\left(1-\frac{f(\mathcal{S}_1^*,\mathcal{I}_{1,s_1}^*)}{f(\mathcal{S},\mathcal{I}_{1,s_1}^*)}\right)\le 0.$$

Finally the term $-\mu\,\mathcal{R}$ is nonpositive.

Collecting all contributions in (4.4), we conclude

$${}^{C}D_t^{\alpha}\mathcal{P}_1(\mathcal{S},\mathcal{I}_1,\mathcal{I}_2,\mathcal{R})\le 0.$$

Equality holds only when $\mathcal{I}_2=0$, $\mathcal{R}=\frac{\sigma_1}{\mu}\mathcal{I}_1$ and the brackets $\xi(\mathcal{S},\mathcal{I}_1)=0$, $\Theta(\mathcal{S},\mathcal{I}_1)=0$, which implies $\mathcal{S}=\mathcal{S}_1^*$ and $\mathcal{I}_1=\mathcal{I}_{1,s_1}^*$. 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 $R_{0,2}\le 1<R_{0,1}$. □

4.2.3. Global stability analysis of the strain 2 endemic equilibrium

Theorem 4.4. *If $R_{0,1}\le 1<R_{0,2}$, then the strain-2 endemic equilibrium $\mathcal{E}_{s_2}$ is globally asymptotically stable.*

Proof. We introduce the Lyapunov function

$$\mathcal{P}_2(\mathcal{S},\mathcal{I}_1,\mathcal{I}_2,\mathcal{R})=\mathcal{S}-\mathcal{S}_2^*-\int_{S_2^*}^{\mathcal{S}}\frac{\beta\mathcal{S}_2^*\mathcal{I}_2^*}{\beta X\mathcal{I}_2^*}\,dX+\mathcal{I}_2^*\left(\frac{\mathcal{I}_2}{\mathcal{I}_2^*}-\ln\frac{\mathcal{I}_2}{\mathcal{I}_2^*}-1\right)+\mathcal{I}_1+\mathcal{R}$$

The function $\mathcal{P}_2$ is nonnegative and vanishes only at $\mathcal{E}_{s_2}$.

The Caputo derivative along the solutions is

$$\begin{aligned}{}^{C}D_t^{\alpha}\mathcal{P}_2=&\left[\lambda-f(\mathcal{S},\mathcal{I}_1)\,\mathcal{I}_1-\beta\mathcal{S}\mathcal{I}_2-\mu\mathcal{S}\right]\left(1-\frac{\mathcal{S}_2^*}{\mathcal{S}}\right)+\left[\beta\mathcal{S}\mathcal{I}_2-(\sigma_2+\mu)\mathcal{I}_2\right]\left(1-\frac{\mathcal{I}_2^*}{\mathcal{I}_2}\right)\\&+f(\mathcal{S},\mathcal{I}_1)\mathcal{I}_1-(\sigma_1+\mu)\mathcal{I}_1+\sigma_1\mathcal{I}_1+\sigma_2\mathcal{I}_2-\mu\mathcal{R}.\end{aligned}$$

Using the equilibrium relations

$$\lambda=\mu\mathcal{S}_2^*+\beta\mathcal{S}_2^*\mathcal{I}_2^*,\qquad(\sigma_2+\mu)\mathcal{I}_2^*=\beta\mathcal{S}_2^*\mathcal{I}_2^*,$$

we obtain after rearrangement:

$${}^{C}D_t^{\alpha}\mathcal{P}_2=\mu(\mathcal{S}_2^*-\mathcal{S})\left(1-\frac{\mathcal{S}_2^*}{\mathcal{S}}\right)+\left(1-\frac{\mathcal{I}_2^*}{\mathcal{I}_2}\right)(\beta\mathcal{S}\mathcal{I}_2-\beta\mathcal{S}_2^*\mathcal{I}_2^*)+f(\mathcal{S},\mathcal{I}_1)\mathcal{I}_1-(\sigma_1+\mu)\mathcal{I}_1+\sigma_1\mathcal{I}_1-\mu\mathcal{R}.$$

We now show how the conditions $R_{0,1}\le 1$ and $R_{0,2}>1$ guarantee the nonpositivity of each block.

– For $R_{0,1} \leq 1$, since $R_{0,1} = \frac{f(\mathcal{S}_2^*,0)}{\sigma_1+\mu} \leq 1$, the infection pressure of strain 1 is insufficient to sustain $\mathcal{I}_1 > 0$. In particular, the linearized coefficient satisfies

$$f(S_2^*, 0) \leq \sigma_1 + \mu.$$

Thus the $\mathcal{I}_1$-dependent block reduces to

$$f(\mathcal{S}, \mathcal{I}_1)\mathcal{I}_1 - (\sigma_1 + \mu)\mathcal{I}_1 + \sigma_1\mathcal{I}_1 = -\mu\mathcal{I}_1 \leq 0.$$

– For $R_{0,2} > 1$, Since $R_{0,2} = \frac{\beta\mathcal{S}_0}{\sigma_2+\mu} > 1$, strain 2 admits a positive endemic equilibrium $\mathcal{I}_2^* > 0$. This yields the identity

$$(\sigma_2 + \mu)\mathcal{I}_2^* = \beta\mathcal{S}_2^*\mathcal{I}_2^*,$$

which ensures that the block

$$\left(1 - \frac{\mathcal{I}_2^*}{\mathcal{I}_2}\right)(\beta\mathcal{S}\mathcal{I}_2 - \beta\mathcal{S}_2^*\mathcal{I}_2^*)$$

has the classical form

$$(1 - x^*/x)(F(x) - F(x^*)),$$

hence it is always nonpositive.

– The $\mathcal{S}$-term satisfies

$$\mu(\mathcal{S}_2^* - \mathcal{S})\left(1 - \frac{\mathcal{S}_2^*}{\mathcal{S}}\right) \leq 0,$$

and the $\mathcal{R}$-term is simply

$$-\mu\mathcal{R} \leq 0.$$

Collecting all estimates, we obtain

$${}^{C}D_t^{\alpha}\mathcal{P}_2 \leq 0.$$

By LaSalle's invariance principle for fractional-order systems, the largest invariant set contained in $\{{}^{C}D_t^{\alpha}\mathcal{P}_2 = 0\}$ is exactly the endemic equilibrium $\mathcal{E}_{s_2}$.

Therefore, under the condition $R_{0,1} \leq 1 < R_{0,2}$, the strain-2 endemic equilibrium $\mathcal{E}_{s_2}$ is globally asymptotically stable.

□

4.2.4. Global stability of the both endemic equilibrium

Theorem 4.5. *If $R_{0,1} > 1$ and $R_{0,2} > 1$, then the both endemic equilibrium $\mathcal{E}_{s_t}$ is globally asymptotically stable.*

Proof. We define the Lyapunov function

$$\mathcal{P}_c(\mathcal{S}, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}) = \mathcal{S} - \mathcal{S}^* - \int_{\mathcal{S}^*}^{\mathcal{S}} \frac{f(\mathcal{S}^*, \mathcal{I}_1^*)\,\mathcal{I}_1^* + \beta\mathcal{S}^*\mathcal{I}_2^*}{f(X, \mathcal{I}_1^*)\,\mathcal{I}_1^* + \beta X\mathcal{I}_2^*}\,dX$$
$$+ \mathcal{I}_1^*\left(\frac{\mathcal{I}_1}{\mathcal{I}_1^*} - \ln\frac{\mathcal{I}_1}{\mathcal{I}_1^*} - 1\right) + \mathcal{I}_2^*\left(\frac{\mathcal{I}_2}{\mathcal{I}_2^*} - \ln\frac{\mathcal{I}_2}{\mathcal{I}_2^*} - 1\right) + \mathcal{R} - \mathcal{R}^* - \mathcal{R}^*\ln\frac{\mathcal{R}}{\mathcal{R}^*}.$$

Its Caputo derivative along the trajectories of the system is

$${}^{C}D_t^{\alpha}\mathcal{P}_c = \left(\lambda - f(\mathcal{S}, \mathcal{I}_1)\mathcal{I}_1 - \beta\mathcal{S}\mathcal{I}_2 - \delta\mathcal{S}\right)\left(1 - \frac{\mathcal{S}^*}{\mathcal{S}}\right) + \left(f(\mathcal{S}, \mathcal{I}_1)\mathcal{I}_1 - (\sigma_1 + \delta)\mathcal{I}_1\right)\left(1 - \frac{\mathcal{I}_1^*}{\mathcal{I}_1}\right)$$
$$+ \left(\beta\mathcal{S}\mathcal{I}_2 - (\sigma_2 + \delta)\mathcal{I}_2\right)\left(1 - \frac{\mathcal{I}_2^*}{\mathcal{I}_2}\right)\left(\sigma_1\mathcal{I}_1 + \sigma_2\mathcal{I}_2 - \delta\mathcal{R}\right)\left(1 - \frac{\mathcal{R}^*}{\mathcal{R}}\right).$$

Substituting the both endemic equilibrium conditions:

$$\begin{cases} \lambda = \delta\mathcal{S}^* + f(\mathcal{S}^*, \mathcal{I}_1^*)\mathcal{I}_1^* + \beta\mathcal{S}^*\mathcal{I}_2^*, \\ f(\mathcal{S}^*, \mathcal{I}_1^*)\mathcal{I}_1^* = (\sigma_1 + \delta)\mathcal{I}_1^*, \\ \beta\mathcal{S}^*\mathcal{I}_2^* = (\sigma_2 + \delta)\mathcal{I}_2^*, \\ \sigma_1\mathcal{I}_1^* + \sigma_2\mathcal{I}_2^* = \delta\mathcal{R}^*. \end{cases}$$

Now we expand each term.

$$\text{Term for } \mathcal{S}: \quad (\lambda - f(\mathcal{S}, \mathcal{I}_1)\mathcal{I}_1 - \beta\mathcal{S}\mathcal{I}_2 - \delta\mathcal{S})\left(1 - \frac{\mathcal{S}^*}{\mathcal{S}}\right)$$

$$= -\frac{\delta(\mathcal{S} - \mathcal{S}^*)^2}{\mathcal{S}} - \frac{\big(f(\mathcal{S}, \mathcal{I}_1)\mathcal{I}_1 - f(\mathcal{S}^*, \mathcal{I}_1^*)\mathcal{I}_1^*\big)(\mathcal{S} - \mathcal{S}^*)}{\mathcal{S}} - \frac{\beta(\mathcal{I}_2\mathcal{S} - \mathcal{I}_2^*\mathcal{S}^*)(\mathcal{S} - \mathcal{S}^*)}{\mathcal{S}} \leq 0.$$

$$\text{Term for } \mathcal{I}_1: \quad (f(\mathcal{S}, \mathcal{I}_1)\mathcal{I}_1 - (\sigma_1 + \delta)\mathcal{I}_1)\left(1 - \frac{\mathcal{I}_1^*}{\mathcal{I}_1}\right)$$

$$= -\frac{(\mathcal{I}_1 - \mathcal{I}_1^*)^2(\sigma_1 + \delta)}{\mathcal{I}_1} \leq 0.$$

$$\text{Term for } \mathcal{I}_2: \quad (\beta\mathcal{S}\mathcal{I}_2 - (\sigma_2 + \delta)\mathcal{I}_2)\left(1 - \frac{\mathcal{I}_2^*}{\mathcal{I}_2}\right)$$

$$= -\frac{(\mathcal{I}_2 - \mathcal{I}_2^*)^2(\sigma_2 + \delta)}{\mathcal{I}_2} \leq 0.$$

$$\text{Term for } \mathcal{R}: \quad (\sigma_1\mathcal{I}_1 + \sigma_2\mathcal{I}_2 - \delta\mathcal{R})\left(1 - \frac{\mathcal{R}^*}{\mathcal{R}}\right)$$

$$= -\delta\frac{(\mathcal{R} - \mathcal{R}^*)^2}{\mathcal{R}} \leq 0.$$

Hence, every term of ${}^{C}D_t^{\alpha}\mathcal{P}_c$ is non-positive, with equality only at

$$(\mathcal{S}, \mathcal{I}_1, \mathcal{I}_2, \mathcal{R}) = (\mathcal{S}^*, \mathcal{I}_1^*, \mathcal{I}_2^*, \mathcal{R}^*).$$

By LaSalle's invariance principle for fractional-order systems, all trajectories converge to the endemic equilibrium $\mathcal{E}_t$. Thus, the coexisting endemic equilibrium is globally asymptotically stable whenever

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

□

For clarity, we summarize the stability results of the equilibria in Table 1.

Table 1: Stability of equilibria

Condition	Equilibrium	Stability
$R_0 < 1$	Disease-free equilibrium	Globally asymptotically stable
$R_{0,1} > 1$	First strain equilibrium	Globally asymptotically stable
$R_{0,2} > 1$	Second strain equilibrium	Globally asymptotically stable
$R_{0,1} > 1$, $R_{0,2} > 1$	Coexistence equilibrium	Globally asymptotically stable

5. Numerical simulations

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 [3]. More precisely, we use a fractional Adams–Bashforth–Moulton predictor–corrector scheme [20] given by:

$$\begin{aligned} U(t_j) &= \frac{h^\alpha}{\Gamma(\alpha+2)}\Big((j-1)^{\alpha+1} - (j-\alpha-1)j^\alpha\Big)\mathcal{F}(U(t_0)) + U(t_0) \\ &+ \frac{h^\alpha}{\Gamma(\alpha+2)}\sum_{i=1}^{j-1}\Big((j-i+1)^{\alpha+1} - 2(j-i)^{\alpha+1} + (j-i-1)^{\alpha+1}\Big)\mathcal{F}(U(t_i)) \\ &+ \frac{h^\alpha}{\Gamma(\alpha+2)}\mathcal{F}(U(t_{j-1})) + \frac{h^\alpha}{\Gamma(\alpha+1)}\mathcal{F}(U(t_{j-1})). \end{aligned}$$

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

The stability behavior of the endemic equilibrium $\mathcal{E}_{s_t}$ is investigated under a non-monotonic incidence function $f(\mathcal{S}, \mathcal{I}_1) = \frac{\beta_1 \mathcal{S}}{1+m\mathcal{I}_1^2}$. The parameter values used in the numerical simulations are chosen based on typical ranges reported in the literature on multi-strain epidemic models [47, 49]. Although these values are not fitted to a specific dataset, they are selected to illustrate the qualitative behavior and stability properties of the model. The values of the parameters used in the simulations are summarized in Table (2).

Table 2: Description of the model parameters

Parameter	Description	Value
λ	Recruitment rate	1 $year^{-1}$
μ	Natural death rate	0.2 $year^{-1}$
β	Transmission rate	0.6
σ_1, σ_2	Recovery rates	0.15

For the numerical simulations, We illustrate the system dynamics in Fig. 2, Fig. 3, Fig. 4 and Fig. 5 for $\alpha = 1$, $\alpha = 0.9$, $\alpha = 0.7$ and $\alpha = 0.5$, respectively. We perform numerical simulations using the parameter set and different values of the treatment control parameters u_1 and u_2: $\lambda = 1, \quad \beta = 0.6, \quad \sigma_1 = \sigma_2 = 0.15, \quad \mu = 0.2, \quad m = 0.14$. Under these conditions, we observe convergence toward the steady state $(0.8982, 0.8433, 0.8309, 1.2557)$ and obtain $R_{0,1} = R_{0,2} = 6.1224$. In this case, both basic reproduction numbers satisfy $R_{0,1} > 1$ and $R_{0,2} > 1$, which confirms that the endemic equilibrium $\mathcal{E}_{s_t}$ 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 illustrate that the proposed generalized model captures a wide variety of incidence functions, providing a comprehensive framework for the study of stability and coexistence dynamics in multi-strain epidemic models.

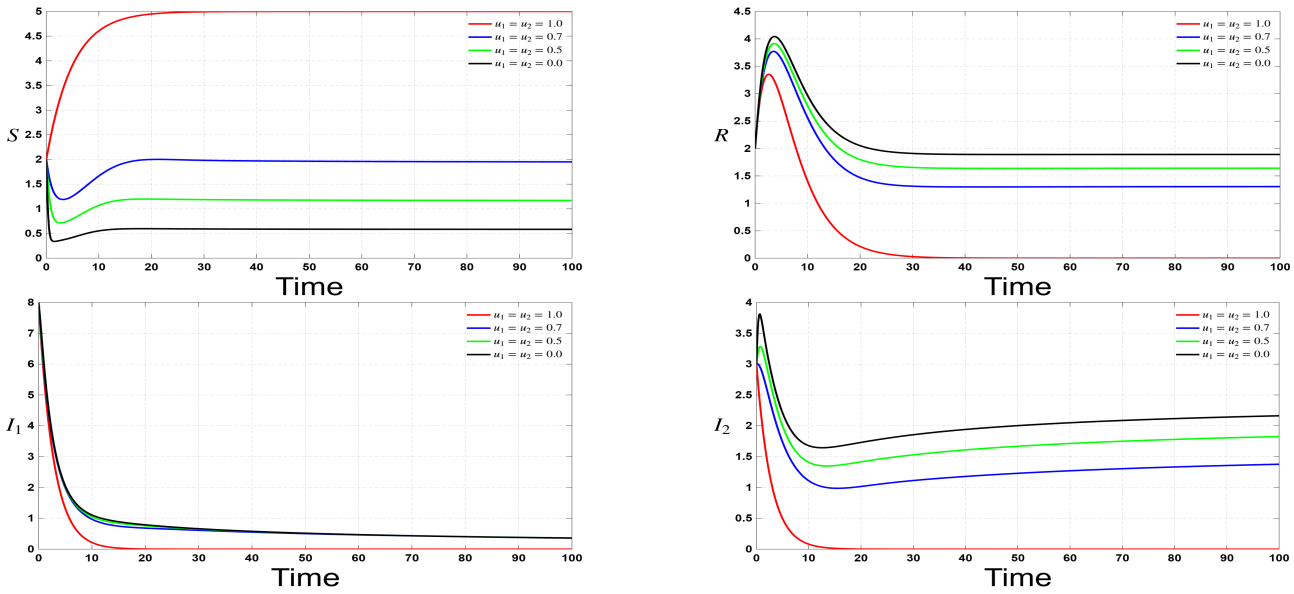

Figure 2: The dynamics of the population for $\alpha = 1$ and the different values of treatment parameters.

Fig. 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 show that the susceptible class $\mathcal{S}(t)$ converges to a positive equilibrium level, while both infectious classes $\mathcal{I}_1(t)$ and $\mathcal{I}_2(t)$ approach non-zero equilibria, reflecting the coexistence of the two circulating strains. The recovered population $\mathcal{R}(t)$ also stabilizes as a result of infection recovery combined with the action of the controls. These numerical observations confirm the local asymptotic stability of the both equilibrium $\mathcal{E}_{s_t}$, at which both strains persist in the population. In addition, the effect of the controls is clearly visible: varying the intensities of u_1 and u_2 modifies the height and timing of the peaks in $\mathcal{I}_1(t)$ and $\mathcal{I}_2(t)$, highlighting the role of the proposed intervention strategies in reducing the infectious burden of each strain while maintaining the overall stability of the system.

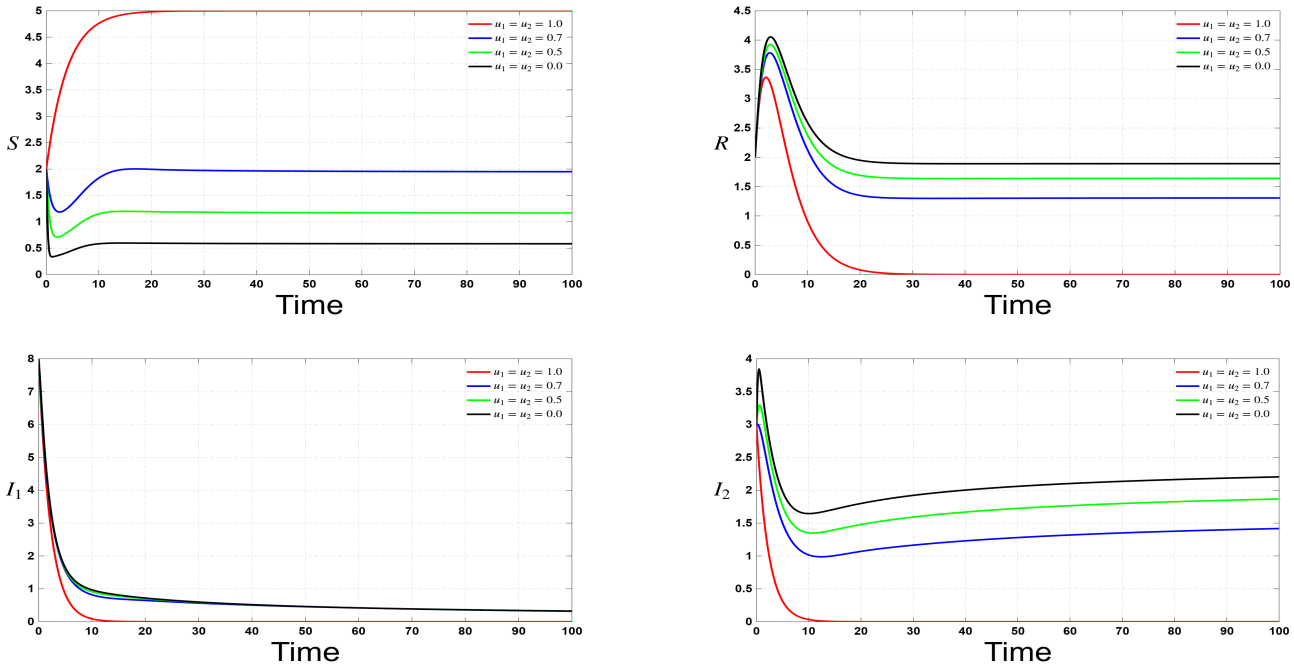

Figure 3: The dynamics of the population for $\alpha = 0.9$ and the different values of treatment parameters.

Fig.3 illustrates the time evolution of the four compartments for the fractional-order case $\alpha = 0.9$, under several choices of the control parameters u_1 and u_2. In comparison with the classical case $\alpha = 1$, the fractional derivative introduces memory effects that slow down the epidemic dynamics, producing a smoother and more gradual convergence toward equilibrium. The susceptible class $\mathcal{S}(t)$ and the recovered class $\mathcal{R}(t)$ stabilize at values close to those obtained in the integer-order framework, while the infectious populations $\mathcal{I}_1(t)$ and $\mathcal{I}_2(t)$ exhibit noticeably slower decay or stabilization phases, reflecting the influence of past states on the present transmission process. These numerical findings indicate that the both endemic equilibrium $\mathcal{E}_{s_t}$ remains locally asymptotically stable for $\alpha = 0.9$ and they demonstrate how the fractional-order formulation modifies the transient behavior of the two-strain system while preserving the long-term persistence of both infections.

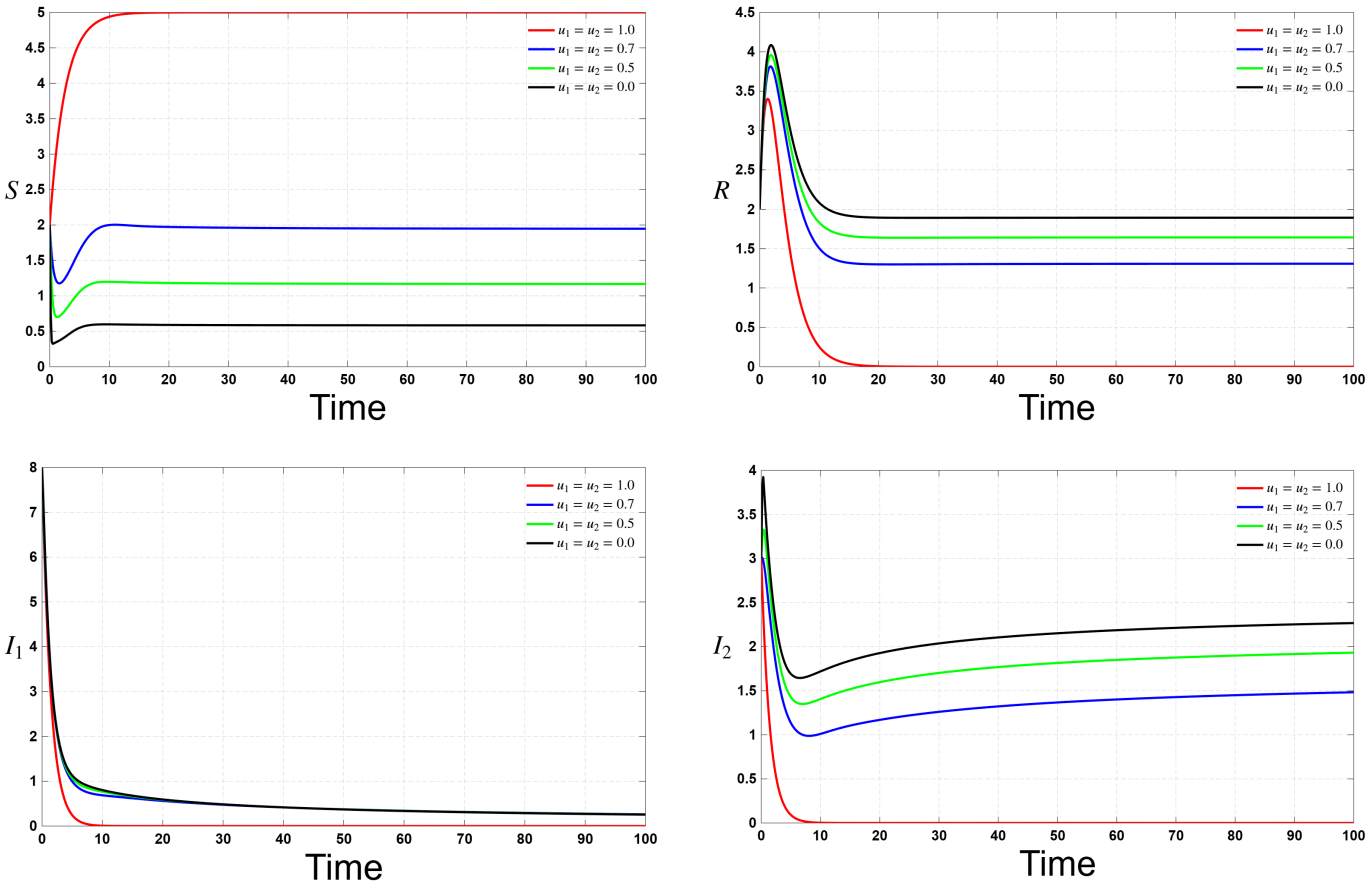

Figure 4: The dynamics of the population for $\alpha = 0.7$ and the different values of treatment parameters.

Fig.4 shows the evolution of the four epidemiological compartments for the fractional-order case $\alpha = 0.7$, under various choices of the control parameters u_1 and u_2. When the fractional order decreases, memory effects become more significant, which slows down the epidemic progression and prolongs the transient phase before reaching equilibrium. The susceptible population $\mathcal{S}(t)$ and the recovered population $\mathcal{R}(t)$ stabilize at levels similar to those observed for higher values of α, whereas the infectious classes $\mathcal{I}_1(t)$ and $\mathcal{I}_2(t)$ exhibit a markedly slower approach to their equilibria. These numerical results confirm that the both endemic equilibrium $\mathcal{E}_{s_t}$ remains locally asymptotically stable even for $\alpha = 0.7$ and they highlight the impact of the fractional-order derivative on the timing and magnitude of the infection peaks while preserving the long-term persistence of both strains in the population.

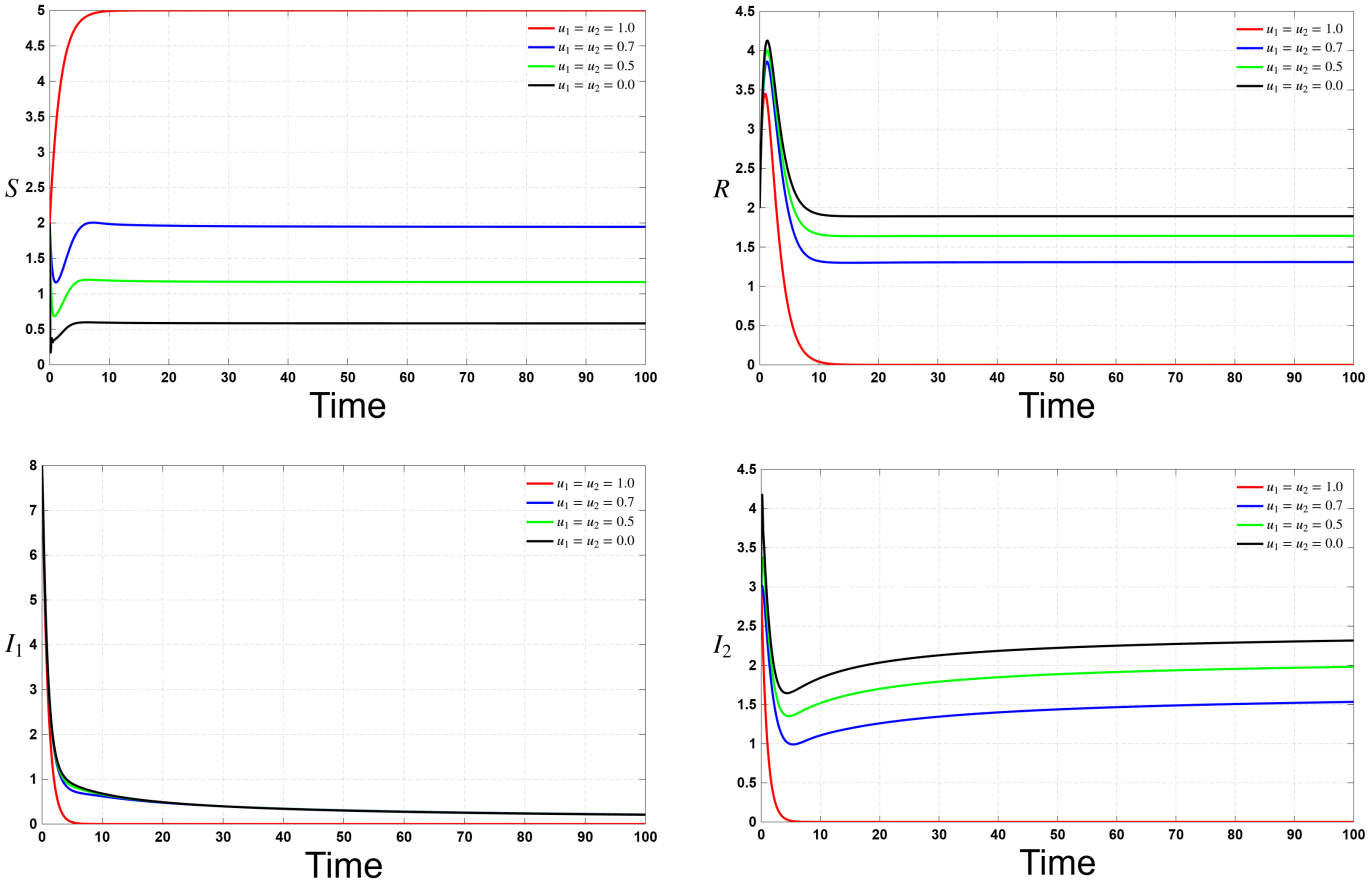

Figure 5: The dynamics of the population for $\alpha = 0.5$ and the different values of treatment parameters.

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 $\mathcal{S}(t)$ and the recovered population $\mathcal{R}(t)$ stabilize at levels comparable to those obtained for higher values of α, while the infectious classes $\mathcal{I}_1(t)$ and $\mathcal{I}_2(t)$ require significantly more time to approach their equilibria. This prolonged transient behavior reflects the strong influence of past states on the current dynamics. The numerical simulations confirm that the both endemic equilibrium $\mathcal{E}_{s_t}$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 for the four fractional orders $\alpha = 1,\ 0.9,\ 0.7$ and 0.5 highlights the significant effect of memory on the epidemic evolution. As α decreases, the trajectories of the infectious classes $\mathcal{I}_1(t)$ and $\mathcal{I}_2(t)$ converge more slowly toward the both endemic equilibrium $\mathcal{E}_{s_t}$, indicating that past states exert a stronger influence on the current dynamics. In contrast, the long-term levels of the susceptible population $\mathcal{S}(t)$ and the recovered class $\mathcal{R}(t)$ remain nearly unchanged across the different values of α.

The main differences appear in the transient behavior: lower fractional orders lead to delayed and smoother peaks of infection, as well as extended convergence times for both infectious compartments. These observations confirm that the fractional-order formulation captures a wide range of memory-driven dynamics while preserving the local asymptotic stability and persistence of the two strains. Overall, this comparison underlines the crucial role of fractional derivatives in modeling memory-dependent epidemic processes and illustrates how the control parameters u_1 and u_2 remain effective across different dynamical regimes.

6. Conclusion

In this work, we analyze a fractional two-strain epidemic SI_1I_2R model with different incidence rates; a general incidence rate for the first strain and a bilinear incidence rate for the second strain. The four epidemiological compartments that make up the suggested structure are: The susceptible population, two infectious classes and the recovered individuals. We demonstrate the existence of solutions, positivity and boundedness. The following formula is used to determine the disease-free equilibrium, the strain-1 endemic equilibrium, the strain-2 endemic equilibrium and the both endemic equilibrium involving both strains. We determine the global stability of the equilibria based on the value of the basic reproduction number R_0 by constructing an appropriate Lyapunov functional. A numerical simulation was provided to validate our theoretical results and to demonstrate the effect of the fractional derivative α on the stability. Additionally, constant control measures u_1 and u_2 were incorporated for each strain to evaluate their impact on the epidemic dynamics. We conclude from all the figures that the different values of the fractional derivative order do not affect the stability of the equilibria but only the convergence time of the solutions toward the equilibria. For high values of this parameter, corresponding to long memory, the solutions converge very slowly to the equilibrium points; for low values, corresponding to short memory, the solutions converge more quickly to the equilibria. Moreover, higher values of the controls u_1 and u_2 effectively reduce the exposed and infectious populations, confirming the efficiency of constant control in mitigating the spread of the disease.

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.

Conflict of Interest

The authors declare that they have no conflict of interest.

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