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Research Article

Mathematical analysis of a fractional-order influenza model with resistant and nonresistant strains

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Abstract

There are numerous acute viral infections worldwide. Influenza is a respiratory illness caused by the influenza virus. In the twenty-first century, fractional-order derivative modeling of

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infectious diseases has gained significant popularity. In the literature, several fractional-order derivative models have been developed. In this research, we propose a fractional two-strain influenza epidemic model that incorporates both resistant and nonresistant strains. We prove the existence and uniqueness of a global positive solution. The equilibrium points and the basic reproduction number are derived. We study the global stability of the equilibrium points using Lyapunov functions and LaSalle's invariance principle. Finally, a series of numerical simulations is presented to illustrate the behavior of the model and to verify our theoretical results. The results show that although the order of the fractional derivatives does not affect the stability of the equilibrium states, it plays an important role in the speed at which the system converges to these equilibria.

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1 Introduction

Influenza is a highly contagious viral infection that remains a significant public health concern worldwide. Caused primarily by influenza A and B viruses, it leads to seasonal epidemics and occasional pandemics that can have severe health and economic consequences [26]. According to the World Health Organization (WHO), annual influenza epidemics are responsible for approximately 3 to 5 million cases of severe illness and 290,000 to 650,000 respiratory-related deaths globally each year [31]. Influenza A viruses are further subdivided into subtypes based on the hemagglutinin (H) and neuraminidase (N) surface proteins, while antigenic drift and shift contribute to the virus's genetic variability and epidemiological complexity [30]. A critical challenge in influenza control is the emergence of multiple co-circulating strains, including those that develop resistance to antiviral medications such as neuraminidase inhibitors [13]. These resistant strains can compromise treatment efficacy and alter transmission dynamics, necessitating mathematical tools capable of capturing such complexities. Modeling the interactions between drug-resistant and drug-sensitive strains is essential for assessing intervention strategies, guiding vaccine formulation, and understanding long-term evolutionary pressures on the virus [7].

Classical compartmental models, such as the SIR (Susceptible–Infectious–Recovered) and SEIR (Susceptible–Exposed–Infectious–Recovered) frameworks, have been instrumental in studying infectious diseases, including influenza [17, 19, 6, 14, 20, 4, 5, 16, 35]. These models describe population dynamics through systems of ordinary differential equations (ODEs), assuming that

the rate of change in each compartment depends only on the current state and not on past states. However, real-world epidemiological processes often exhibit memory and hereditary properties. For example, immunity to influenza wanes gradually, contact rates may vary based on past exposure or social behavior, and virus–host interactions can show time-dependent adaptation [34]. To address these limitations, researchers have increasingly turned to fractional-order differential equations (FDEs), a generalization of classical calculus that allows for derivatives of noninteger order [25, 10]. The Caputo fractional derivative, in particular, enables the incorporation of initial conditions in a way that is consistent with physical and biological systems [18, 9, 32, 24]. Applications of fractional calculus in infectious disease modeling have demonstrated improved fitting to epidemic data compared to integer-order models. For example, sub-exponential growth patterns observed during the early phases of outbreaks of diseases such as COVID-19 and dengue fever have been effectively captured using fractional models [1, 28, 2, 15, 21, 3, 22]. In the context of influenza, incorporating fractional dynamics can help model the gradual loss of immunity, delays in antiviral response effectiveness, and heterogeneous contact structures, especially in the presence of multiple strains with varying transmission and resistance characteristics [23, 27, 12, 36]. In their study [8], Baba et al. developed and analyzed a deterministic two-strain influenza model with resistant and nonresistant strains. These strains are differentiated by their incidence rates, which are bilinear and saturated for the nonresistant strain, and saturated for the resistant strain. This work extends the previous model of Baba et al. by proposing and analyzing a new fractional-derivative model that incorporates Caputo derivatives to reflect the influence of past infection and intervention history on current disease progression.

This paper is organized into eight sections. In Section 2, we formulate a fractional two-strain influenza model incorporating two distinct incidence rates. In Section 3, we introduce some mathematical preliminaries that assist in the analysis of our model. Section 4 deals with the positivity, boundedness, and uniqueness of the solutions. Section 5 is devoted to the determination of the basic reproduction number, the identification of the equilibrium points, and their global stability analysis. Numerical simulations are presented in Section 6. Finally, Section 7 concludes the study.

2 Model formulation

The model represents the transmission dynamics of a disease involving two viral strains: a nonresistant strain, denoted by $I_N(t)$, and a resistant strain, denoted by $I_R(t)$. The system is formulated using Caputo fractional derivatives of order θ, allowing the incorporation of memory effects into the disease progression and treatment processes. The population is divided into

four compartments: susceptible individuals $S(t)$, individuals infected by the nonresistant strain $I_N(t)$, individuals infected by the resistant strain $I_R(t)$, and recovered individuals $R(t)$. Susceptible individuals are recruited at a constant rate b and may become infected through contact with either $I_N(t)$ or $I_R(t)$. Transmission by the nonresistant strain follows a standard bilinear form $aS(t)I_N(t)$, whereas transmission by the resistant strain is modeled by a saturated incidence function $\dfrac{\gamma S(t)I_R(t)}{1+kI_R(t)}$, where the parameter k represents a saturation effect that limits the effective transmission of the resistant strain as the number of infected individuals increases. This saturation may arise from behavioral changes, partial immunity, limited contact opportunities, or treatment-related constraints. Infectious individuals in $I_N(t)$ may recover at rate θ, die naturally, or move to the recovered class. In contrast, individuals in $I_R(t)$ represent infections caused by a strain that resists treatment; hence, the recovery rate γ may be lower. The saturated transmission term for $I_R(t)$ also indicates that as the number of resistant infections increases, the effective transmission rate declines, which can simulate immunological or medical saturation effects. Recovered individuals gain immunity through recovery from either strain and exit the system through natural death. The fractional-order derivatives account for nonlocal, history-dependent effects, which are biologically relevant for diseases in which immune response, treatment impact, or behavioral patterns are influenced by past states. This model is particularly suited to studying the coexistence and competition between resistant and nonresistant strains, and how memory effects influence the speed and stability of convergence toward the disease-free or endemic equilibrium. Based on [8], the mathematical representation of these interactions is given by the following system of fractional differential equations

$$\begin{cases} D^{\theta}S(t) = b - d\,S(t) - a\,S(t)\,I_N(t) - \dfrac{\gamma\,S(t)\,I_R(t)}{1+k\,I_R(t)}, \\ D^{\theta}I_N(t) = a\,S(t)\,I_N(t) - (d+\theta)\,I_N(t), \\ D^{\theta}I_R(t) = \dfrac{\gamma\,S(t)\,I_R(t)}{1+k\,I_R(t)} - (d+\gamma)\,I_R(t), \\ D^{\theta}R(t) = \theta\,I_N(t) + \gamma\,I_R(t) - d\,R(t). \end{cases} \tag{1}$$

Where D^{θ} denotes the Caputo fractional derivative of order θ, with $0 < \theta \leq 1$. All parameters are described in Table 1. The two-strain diagram is shown in Figure 1. The initial conditions $S(0)$, $I_N(0)$, $I_R(0)$, and $R(0)$ of system (1) are assumed to be positive. In our model, we assume that individuals become susceptible only through birth or immigration, co-infection does not occur, and immunity is present.

Table 1: An explanation of the parameters of the model (1).

Parameter	Description
b	Recruitment into the susceptible class
d	Death rate
γ	Rate of infection by the resistant strain
a	Rate of infection by the nonresistant strain
γ	Rate at which individuals with the resistant strain are removed from the population
θ	Rate at which individuals with the nonresistant strain are removed from the population
k	Saturation parameter in the resistant-strain incidence rate

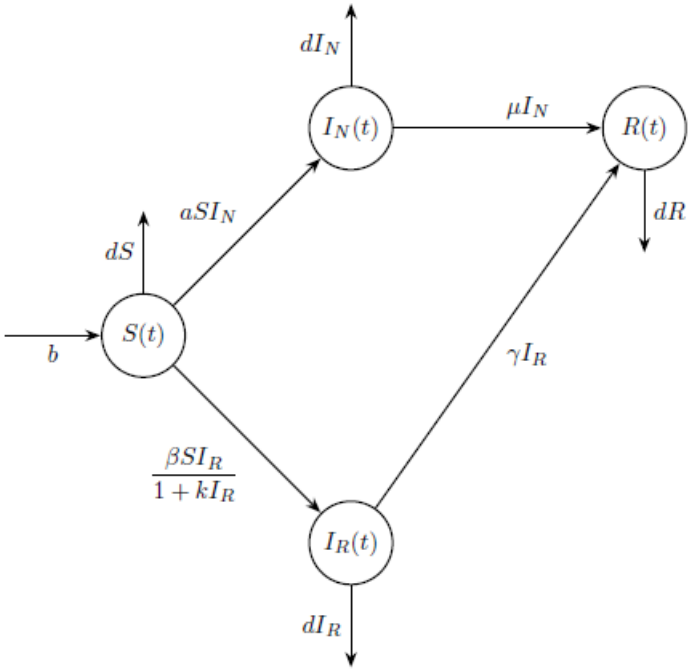

Figure 1: Compartmental diagram of the fractional-order model representing the transmission of nonresistant and resistant strains.

3 Preliminaries

We present in this section some theoretical results that will help us examine system (1) later in this study.

Definition 1. Consider the fractional integral of order $\theta > 0$ of a function $H : \mathbb{R}_+ \to \mathbb{R}$. It is defined as

$$I^{\theta} H(t) = \frac{1}{\Gamma(\theta)} \int_0^t (t-x)^{\theta-1} H(x)\, dx, \tag{2}$$

where the Gamma function $\Gamma(\theta)$ is defined by

$$\Gamma(\theta) = \int_0^{\infty} e^{-t} t^{\theta-1}\, dt.$$

Definition 2. For a function $H : \mathbb{R}_+ \to \mathbb{R}$, the Caputo fractional derivative of order $\theta > 0$ is defined by

$$D^{\theta} H(t) \equiv I^{\,n-\theta} D^n H(t), \tag{3}$$

where $D = \dfrac{d}{dt}$, n is the smallest integer greater than θ (i.e., $n-1 < \theta \leq n$), and $n \in \mathbb{N}$. In particular, when $0 < \theta \leq 1$, the fractional derivative can be expressed as

$$D^{\theta} H(t) = \frac{1}{\Gamma(1-\theta)} \int_0^t \frac{H'(x)}{(t-x)^{\theta}}\, dx. \tag{4}$$

Definition 3. The Mittag–Leffler function with parameter $\theta > 0$, denoted by E_θ, is defined as

$$E_{\theta}(t) = \sum_{i=0}^{\infty} \frac{t^i}{\Gamma(\theta i + 1)}. \tag{5}$$

Let $U : \mathbb{R}_+^n \to \mathbb{R}_+^n$, with $n \geq 1$. We consider the fractional-order system described by

$$D^{\theta} U(t) = G(U(t)), \tag{6}$$

with the initial condition $U(0) = U_0$, where $U_0 \in \mathbb{R}_+^n$ and $0 < \theta \leq 1$.

Lemma 1 ([8])**.** Let D be a compact subset of $\mathbb{R}_+^n$. Every solution of system (6) that starts in D remains in D for all $t \geq 0$, provided that there exists a continuously differentiable function $V(U) \in C^1(D)$ satisfying

$$D^{\theta} V(U) \leq 0. \tag{7}$$

Define E as the set of points in D where $D^{\theta} V = 0$, and let Ψ denote the largest invariant set contained in E. Then, every solution $U(t)$ starting in D asymptotically approaches Ψ as $t \to \infty$.

Lemma 2 ([8])**.** Suppose that $h(t) \in C[a,b]$ and $D^{\theta} h(t) \in C[a,b]$ for $0 < \theta \leq 1$. Then we have

$$h(t) = h(a) + \frac{1}{\Gamma(\theta)} D^{\theta} h(\nu)(t-a)^{\theta}, \quad a < \nu < t, \quad \forall t \in (a,b]. \tag{8}$$

Lemma 3 ([8]). Suppose that $h(t) \in C[a,b]$ and $D^\theta h(t) \in C[a,b]$ for $0 < \theta \leq 1$. If $D^\theta h(t) \geq 0$ for all $t \in [a,b]$, then $h(t)$ is nondecreasing on $[a,b]$. If $D^\theta h(t) \leq 0$ for all $t \in [a,b]$, then $h(t)$ is nonincreasing on $[a,b]$.

Lemma 4 ([8]). Let $0 < \theta \leq 1$ and let $x(t)$ be a positive and sufficiently smooth function. For any constant $x^* > 0$, define

$$\psi(x) = x - x^* - x^* \ln\Big(\frac{x}{x^*}\Big).$$

Then, along any positive solution $x(t) > 0$, the fractional derivative satisfies

$$D^\theta \psi(x(t)) \leq \Big(1 - \frac{x^*}{x(t)}\Big) D^\theta x(t).$$

Consequently, for $X(t) = (x_1(t), \ldots, x_n(t))$ and

$$V(X) = \sum_{i=1}^{n} \Big(x_i - x_i^* - x_i^* \ln\big(\tfrac{x_i}{x_i^*}\big)\Big),$$

we have

$$D^\theta V(X(t)) \leq \sum_{i=1}^{n} \Big(1 - \frac{x_i^*}{x_i(t)}\Big) D^\theta x_i(t).$$

Here, D^θ denotes the Caputo fractional derivative.

4 Positivity, boundedness, and uniqueness of the solution

In this section, we show that, for any given initial data, system (1) admits a unique, positive, and bounded solution.

Theorem 1. For any positive initial conditions $S(0)$, $I_N(0)$, $I_R(0)$, and $R(0)$, system (1) admits a unique, positive, and bounded solution defined in $\mathbb{R}^4_+$ for all $t \geq 0$.

Proof. First, we demonstrate that the solutions of system (1) remain positive. Assuming that all model parameters are positive, we have

$$\begin{cases} D^\theta S\big|_{S=0} = b \geq 0, \\ D^\theta I_N\big|_{I_N=0} = 0 \geq 0, \\ D^\theta I_R\big|_{I_R=0} = 0 \geq 0, \\ D^\theta R\big|_{R=0} = \theta I_N + \gamma I_R \geq 0. \end{cases} \tag{9}$$

From Lemmas 2 and 3, it follows that the solutions of system (1) remain positive. We now show that the solutions of system (1) remain bounded by considering the total population given by

$$N(t) = S(t) + I_N(t) + I_R(t) + R(t). \tag{10}$$

Then,

$$D^\theta N(t) \leq b - dN(t), \tag{11}$$

Consequently, by applying the Laplace transform, we conclude that

$$N(t) \leq (-\frac{b}{d} + N(0))E_\theta(-dt^\theta) + \frac{b}{d}. \tag{12}$$

where E_θ is the Mittag–Leffler function with parameter θ. Consequently, we conclude that the set

$$\mathcal{T} := \left\{ (S, I_N, I_R, R) \in \mathbb{R}^4_+ \;\middle|\; S + I_N + I_R + R \leq \frac{b}{d} \right\}$$

is positively invariant for system (1). Hence, every solution of system (1) remains positive and bounded. □

Theorem 2. For every initial condition $U(0) = U_0 \in \mathcal{T}$, system (1) admits a unique solution $U(t)$ defined for all $t \geq 0$.

Proof. Let $U(t) = \big(S(t), I_N(t), I_R(t), R(t)\big)^\top$ and rewrite system (1) as

$${}^C D^\theta U(t) = G\big(U(t)\big), \quad 0 < \theta \leq 1, \tag{13}$$

where

$$G(U) = \begin{pmatrix} b - dS - aSI_N - \dfrac{\gamma SI_R}{1 + kI_R} \\ aSI_N - (d + \theta)I_N \\ \dfrac{\gamma SI_R}{1 + kI_R} - (d + \gamma)I_R \\ \theta I_N + \gamma I_R - dR \end{pmatrix}. \tag{14}$$

The mapping $G : \mathbb{R}^4_+ \to \mathbb{R}^4$ is continuously differentiable with respect to U on $\mathbb{R}^4_+$. Hence, G is locally Lipschitz on $\mathbb{R}^4_+$. Therefore, for any $U_0 \in \mathbb{R}^4_+$, there exists a unique local solution on $[0, T_{\max})$ [10]. Moreover, by Theorem 1, the set $\mathcal{T}$ is positively invariant, and all solutions starting in $\mathcal{T}$ remain bounded in $\mathcal{T}$ for as long as they exist. Assume, by contradiction, that the maximal existence time satisfies $T_{\max} < +\infty$. By the standard continuation criterion for Caputo fractional differential equations, a maximal solution can cease to exist in finite time only if it

becomes unbounded as $t \to T_{\max}^{-}$. This contradicts the boundedness of the solution in $\mathcal{T}$. Hence, $T_{\max} = +\infty$, and the solution exists globally and is unique. □

5 Global stability of equilibria

In this section, we show that system (1) admits a disease-free equilibrium, a nonresistant equilibrium, a resistant equilibrium, and an endemic equilibrium. Moreover, we employ appropriate Lyapunov functions and LaSalle's invariance principle to investigate the global stability of these equilibrium points.

5.1 Basic reproduction number

Following the epidemiological literature [29], the basic reproduction number R_0 is defined as the expected number of secondary infections generated by a single infected individual introduced into a wholly susceptible population. We compute R_0 for system (1) using the next-generation matrix approach [33]. At the disease-free equilibrium,

$$E_0 = \left(\frac{b}{d}, 0, 0, 0\right),$$

the matrices of new infection and transition terms are given by

$$F = \begin{pmatrix} \frac{ab}{d} & 0 \\ 0 & \frac{\gamma b}{d} \end{pmatrix}, \qquad V = \begin{pmatrix} d+\theta & 0 \\ 0 & d+\gamma \end{pmatrix}.$$

The inverse of V is

$$V^{-1} = \begin{pmatrix} \frac{1}{d+\theta} & 0 \\ 0 & \frac{1}{d+\gamma} \end{pmatrix},$$

hence,

$$FV^{-1} = \begin{pmatrix} \frac{ab}{d(d+\theta)} & 0 \\ 0 & \frac{\gamma b}{d(d+\gamma)} \end{pmatrix}.$$

Therefore, the basic reproduction number is given by

$$R_0 = \rho(FV^{-1}) = \max\{R_N, R_R\},$$

where

$$R_N = \frac{ab}{d(d+\theta)}, \qquad R_R = \frac{\gamma b}{d(d+\gamma)}.$$

5.2 Equilibrium points

In this subsection, we characterize the equilibrium structure of the proposed fractional-order epidemic model given in system (1). The system admits four steady states: the disease-free equilibrium E_0, the nonresistant equilibrium E_1, the resistant equilibrium E_2, and the endemic (coexistence) equilibrium E_3. We obtain the following equations by setting the right-hand side of system (1) equal to zero

$$\begin{cases} b - dS - aSI_N - \dfrac{\gamma SI_R}{1+kI_R} = 0, \\ aSI_N - (d+\theta)I_N = 0, \\ \dfrac{\gamma SI_R}{1+kI_R} - (d+\gamma)I_R = 0, \\ \theta I_N + \gamma I_R - dR = 0. \end{cases} \tag{15}$$

1. Disease-free equilibrium point

   The disease-free equilibrium point denotes the state in which the population carries no infection and is given by

   $$E_0 = \left(\frac{b}{d}, 0, 0, 0\right). \tag{16}$$

   For any set of positive parameters, this equilibrium point exists.

2. Nonresistant equilibrium point

   The nonresistant equilibrium point represents a steady state where $I_N^* > 0$ and $I_R^* = 0$. It is given by

   $$E_1 = \left(\frac{d+\theta}{a}, \frac{ab - d(d+\theta)}{a(d+\theta)}, 0, \frac{\theta}{d}.I_N^*\right). \tag{17}$$

   The nonresistant equilibrium point E_1 exists if and only if $R_N = \dfrac{ab}{d(d+\theta)} > 1$.

3. Resistant equilibrium point
   The resistant equilibrium point represents a steady state where $I_R^* > 0$, $I_N^* = 0$. It is represented by

$$E_2 = \left(\frac{d+\gamma}{\gamma} \left(1 + k\, I_R^*\right), 0, \frac{b\gamma - d(d+\gamma)}{(d+\gamma)(\gamma + dk)}, \frac{\gamma}{d} I_R^* \right). \tag{18}$$

   The resistant equilibrium point E_2 exists if only if $R_R = \dfrac{b\gamma}{d(d+\gamma)} > 1$.

4. Endemic equilibrium point
   The endemic equilibrium point E_3 corresponds to the case where $I_N^* > 0$ and $I_R^* > 0$, and it is given by

$$E_3 = \left(\frac{d+\theta}{a}, \frac{b + \dfrac{d+\gamma}{k} - \dfrac{d+\theta}{a}\left(d + \dfrac{\gamma}{k}\right)}{d+\theta}, \frac{1}{k}\left(\frac{\gamma(d+\theta)}{a(d+\gamma)} - 1 \right) \right.$$
$$\left. , \frac{\theta I_N^* + \gamma I_R^*}{d} \right). \tag{19}$$

The endemic equilibrium point E_3 exists if and only if

$$\frac{\gamma(d+\theta)}{a(d+\gamma)} > 1 \quad \text{and} \quad b + \frac{d+\gamma}{k} > \frac{d+\theta}{a}\left(d + \frac{\gamma}{k}\right). \tag{20}$$

These conditions are necessary and sufficient to ensure $S^* > 0$, $I_N^* > 0$ and $I_R^* > 0$. In particular, these inequalities imply that $R_N > 1$ and $R_R > 1$. Hence, the condition $R_R \geq R_N > 1$ should be interpreted as a sufficient but not equivalent condition for the existence of the coexistence equilibrium point.

5.3 Stability analysis

This subsection presents theorems on the global stability of the equilibrium points, derived via the Lyapunov method and LaSalle's invariance principle.

Theorem 3. E_0 is globally asymptotically stable if $\max\{R_N, R_R\} \leq 1$.

Proof. We introduce the following Lyapunov function

$$V_0 = (S - S_0 ln S) + I_R + I_N. \tag{21}$$

Using Lemma 4, the θ derivative of V_0 satisfies

$$D^\theta V_0 \leq (1-\frac{S_0}{S})D^\theta S + D^\theta I_R + D^\theta I_N \tag{22}$$

$$\begin{aligned} D^\theta V_0 \leq (1-\frac{S_0}{S})\left(b - dS - aSI_N - \frac{\gamma SI_R}{1+kI_R}\right) \\ + aSI_N - (d+\theta)I_N + \frac{\gamma SI_R}{1+kI_R} - (d+\gamma)I_R \end{aligned} \tag{23}$$

$$\begin{aligned} D^\theta V_0 \leq dS_0 - dS - \frac{dS_0^2}{S} + (d+\theta)I_N\left(\frac{aS_0}{d+\theta} - 1\right) \\ + (d+\gamma)I_R\left(\frac{\gamma S_0}{(d+\gamma)(1+kI_R)} - 1\right) \end{aligned} \tag{24}$$

$$\begin{aligned} D^\theta V_0 \leq dS_0\left(2 - \frac{S}{S_0} - \frac{S_0}{S}\right) + (d+\theta)I_N(R_N - 1) \\ + (d+\gamma)I_R(R_R - 1). \end{aligned} \tag{25}$$

The arithmetic mean is greater than or equal to the geometric mean, we Have

$$2 - \frac{S}{S_0} - \frac{S_0}{S} \leq 0. \tag{26}$$

Then, $D^\theta V_0 \leq 0$ if :$R_R \leq 1$ and $R_N \leq 1$. We denote A_0 The largest invariant set in $(S, I_N, I_R); D^\theta V_0 = 0$. Thus, $D^\theta V_0 = 0$ if and only if $S = S_0 = \frac{b}{d}$ and $I_N = I_R = 0$. Hence, the disease-free equilibrium is

$$A_0 = E_0 = \left(\frac{b}{d}, 0, 0, 0\right).$$

Since LaSalle's invariance principle that the E_0 is globally asymptotically stable if $R_R \leq 1$ and $R_N \leq 1$. □

Theorem 4. Assume $R_N > 1$ and $R_R \leq 1$. Then the equilibrium E_1 of (1) is globally asymptotically stable.

Proof. Consider the Lyapunov function

$$V_1(S, I_N, I_R) = S - S_1 - S_1 \ln\left(\frac{S}{S_1}\right) + I_N - I_{N_1} - I_{N_1} \ln\left(\frac{I_N}{I_{N_1}}\right) + I_R. \tag{27}$$

Using Lemma 4, the θ derivative of V_1 satisfies

$$D^\theta V_1 \leq \left(1 - \frac{S_1}{S}\right)D_t^\theta S + \left(1 - \frac{I_{N_1}}{I_N}\right)D_t^\theta I_N + D_t^\theta I_R. \tag{28}$$

It is clear that,

$$aS_1 = d + \theta, \qquad b - dS_1 - aS_1 I_{N_1} = 0, \tag{29}$$

then

$$D^\theta V_1 \leq -d\,\frac{(S - S_1)^2}{S} \;-\; (d+\theta)\,\frac{(I_N - I_{N_1})^2}{I_N} \;+\; \Big(\frac{\gamma S_1}{1 + kI_R} - (d+\gamma)\Big)\, I_R. \tag{30}$$

Since $k > 0$ and $I_R \geq 0$, one has $1 + kI_R \geq 1$. Hence,

$$\frac{\gamma S_1}{1 + kI_R} - (d+\gamma) \;\leq\; \gamma S_1 - (d+\gamma) \;=\; (d+\gamma)\big(R_R - 1\big) \;\leq\; 0.$$

Therefore, the last term in (30) is nonpositive, and we conclude that

$$D^\theta V_1 \;\leq\; -d\,\frac{(S - S_1)^2}{S} \;-\; (d+\theta)\,\frac{(I_N - I_{N_1})^2}{I_N} \;+\; (d+\gamma)\big(R_R - 1\big) \;\leq\; 0, \tag{31}$$

with equality if and only if $S = S_1$, $I_N = I_{N_1}$, and $I_R = 0$.

By LaSalle's invariance principle, every solution with initial data in $\mathcal{T}$ converges to the largest invariant subset of $\{D^\theta V_1 = 0\}$, which is $\{(S_1, I_{N_1}, 0)\}$. Finally, since $N = S + I_N + I_R + R$ satisfies $D^\theta N = b - dN$, we have $N(t) \to S_1$, and thus

$$R(t) = N(t) - S(t) - I_N(t) - I_R(t) \to R_1.$$

Hence, the full state converges to E_1, proving global asymptotic stability.

□

Theorem 5. Assume $R_R > 1$ and $R_N \leq 1$. Then the equilibrium E_2 of (1) is globally asymptotically stable.

Proof. Consider the Lyapunov function

$$V_2(S, I_N, I_R) = S - S_2 - S_2 \ln\Big(\frac{S}{S_2}\Big) + I_R - I_{R_2} - I_{R_2} \ln\Big(\frac{I_R}{I_{R_2}}\Big) + I_N. \tag{32}$$

Using Lemma 4, the θ derivative of V_2 satisfies

$$D^\theta V_2 \;\leq\; \Big(1 - \frac{S_2}{S}\Big) D^\theta S + \; D^\theta I_N + \Big(1 - \frac{I_{R_2}}{I_R}\Big) D^\theta I_R. \tag{33}$$

We have

$$\frac{\gamma S_2}{1 + kI_{R_2}} = d + \gamma, \qquad b - dS_2 - \frac{\gamma S_2 I_{R_2}}{1 + kI_{R_2}} = 0, \tag{34}$$

So,

$$D^\theta V_2 \leq -d\,\frac{(S - S_2)^2}{S} \;-\; (d+\gamma)\,\frac{(I_R - I_{R_2})^2}{I_R} \;+\; \big(aS_2 - (d+\theta)\big)\, I_N. \tag{35}$$

We have $aS_2 - (d+\theta) \leq 0$, hence the last term is also nonpositive. Therefore $D^\theta V_2 \leq 0$, with equality if and only if $S = S_2$, $I_R = I_{R_2}$, and $I_N = 0$. By LaSalle's invariance principle, every solution starting in Ω converges to the largest invariant subset of $\{D^\theta V_2 = 0\}$, which reduces to $\{(S_2, 0, I_{R_2})\}$. Finally, since $N = S + I_N + I_R + R$ satisfies $D^\theta N = b - dN$, we have $N(t) \to S_2$, so $R(t) = N(t) - S(t) - I_N(t) - I_R(t) \to R_2$. Hence the full state converges to E_2, which proves global asymptotic stability. □

Theorem 6. Assume that the endemic equilibrium E_3 exists, i.e.,

$$\frac{\gamma(d+\theta)}{a(d+\gamma)} > 1 \quad \text{and} \quad b + \frac{d+\gamma}{k} > \frac{d+\theta}{a}\left(d + \frac{\gamma}{k}\right).$$

Then the endemic equilibrium E_3 is globally asymptotically stable.

Proof. Consider the Lyapunov function

$$\begin{aligned} V_3(S, I_R, I_N) =& \left(S - S_3 - S_3 \ln \frac{S}{S_3}\right) + \left(I_R - I_{R_3} - I_{R_3} \ln \frac{I_R}{I_{R_3}}\right) \\ &+ \left(I_N - I_{N_3} - I_{N_3} \ln \frac{I_N}{I_{N_3}}\right), \end{aligned} \tag{36}$$

Using Lemma 4, the θ derivative of V_3 satisfies

$$D^\theta V_3 \leq \left(1 - \frac{S_3}{S}\right) D^\theta S + \left(1 - \frac{I_{R_3}}{I_R}\right) D^\theta I_R + \left(1 - \frac{I_{N_3}}{I_N}\right) D^\theta I_N. \tag{37}$$

Substituting the system gives

$$\begin{aligned} D^\theta V_3 \leq& \left(1 - \frac{S_3}{S}\right)\left(b - aSI_N - \frac{\gamma S I_R}{1 + kI_R} - dS\right) \\ &+ \left(1 - \frac{I_{R_3}}{I_R}\right)\left(\frac{\gamma S I_R}{1 + kI_R} - (d+\gamma) I_R\right) \\ &+ \left(1 - \frac{I_{N_3}}{I_N}\right)(aSI_N - (d+\theta) I_N). \end{aligned} \tag{38}$$

At the endemic equilibrium E_3 we have

$$aS_3 = d + \theta, \qquad \frac{\gamma S_3}{1 + kI_{R_3}} = d + \gamma, \qquad b = aS_3 I_{N_3} + \frac{\gamma S_3 I_{R_3}}{1 + kI_{R_3}} + dS_3.$$

We obtain

$$D^\theta V_3 \leq -d\,\frac{(S - S_3)^2}{S} - (d+\gamma)\,\frac{(I_R - I_{R_3})^2}{I_R} - (d+\theta)\,\frac{(I_N - I_{N_3})^2}{I_N} \leq 0. \tag{39}$$

So, $D^\theta V_3 = 0$ holds only at $S = S_3$, $I_R = I_{R_3}$ and $I_N = I_{N_3}$. Therefore, by LaSalle's invariance principle for fractional-order systems, E_3 is globally asymptotically stable whenever it exists, i.e. when $R_R \geq R_N > 1$. □

6 Numerical simulations and discussion

In this section, we give some numerical simulations to illustrate our analytical results. For this end, we apply the numerical algorithm presented by Erturk et al. [11] to numerically solve the fractional-order model (1). Let us consider the following problem

$$\begin{cases} D^\theta U(t) = G(U(t)), \\ U(0) = U_0, \quad U_0 \in \mathbb{R}^4_+, \end{cases} \tag{40}$$

where

$$U(t) = (S(t), I_N(t), I_R(t), R(t))^\top \quad \text{and} \quad U_0 = (S(0), I_N(0), I_R(0), R(0))^\top$$

is the initial condition. The problem above is solved using the following numerical scheme

$$\begin{aligned} U(t_j) =& \frac{h^\theta}{\Gamma(\theta+2)} \left((j-1)^{\theta+1} - (j-\theta-1)^{\theta+1}\right) G(U_0) \\ &+ \frac{h^\theta}{\Gamma(\theta+2)} \sum_{i=1}^{j-1} \left[(j-i+1)^{\theta+1} - 2(j-i)^{\theta+1} + (j-i-1)^{\theta+1}\right] G(U(t_i)) \\ &+ \frac{h^\theta}{\Gamma(\theta+2)} G(U(t_{j-1})) + \frac{h^\theta}{\Gamma(\theta+1)} G(U(t_{j-1})), \end{aligned} \tag{41}$$

with $t_{j+1} = t_j + h$, for $j = 0, 1, \ldots, N-1$, and $U_0 = (10,\ 10,\ 3,\ 0)$. The numerical scheme is implemented with a fixed time step $h = 0.01$ over the interval $t \in [0, T]$, where T varies between 80 and 9000. The parameter values used in each figure are reported in Table 2. The fractional derivative order is chosen from $\theta \in \{1,\ 0.9,\ 0.7,\ 0.5\}$.

6.1 The free-disease equilibrium point

Figure 2 illustrates the time evolution of the system around the disease-free equilibrium $E_0 = (0.4,\ 0,\ 0,\ 0)$, for different values of the fractional-order derivative θ. It is observed that the infected populations $I_N(t)$ and $I_R(t)$ decrease monotonically to zero for all considered values of θ. The recovered population $R(t)$ initially increases due to recovery from infection and then gradually declines to zero as the disease dies out. Meanwhile, the susceptible population $S(t)$

Table 2: The parameters values of model (1).

Parameter	Figure 2	Figure 3	Figure 4	Figure 5
γ	0.1	0.1	0.1	0.1
γ	0.01	0.01	0.6	0.6
a	0.05	0.6	0.05	0.9
b	0.04	0.2	0.2	0.2
d	0.1	0.1	0.1	0.1
θ	0.05	0.05	0.05	0.05
k	0.2	0.2	0.2	0.2
R_N	0.13333	8.0000	0.6666	5.9999
R_R	0.0200	0.1999	5.9999	9.3333
Theorem	Theorem 3	Theorem 4	Theorem 5	Theorem 6

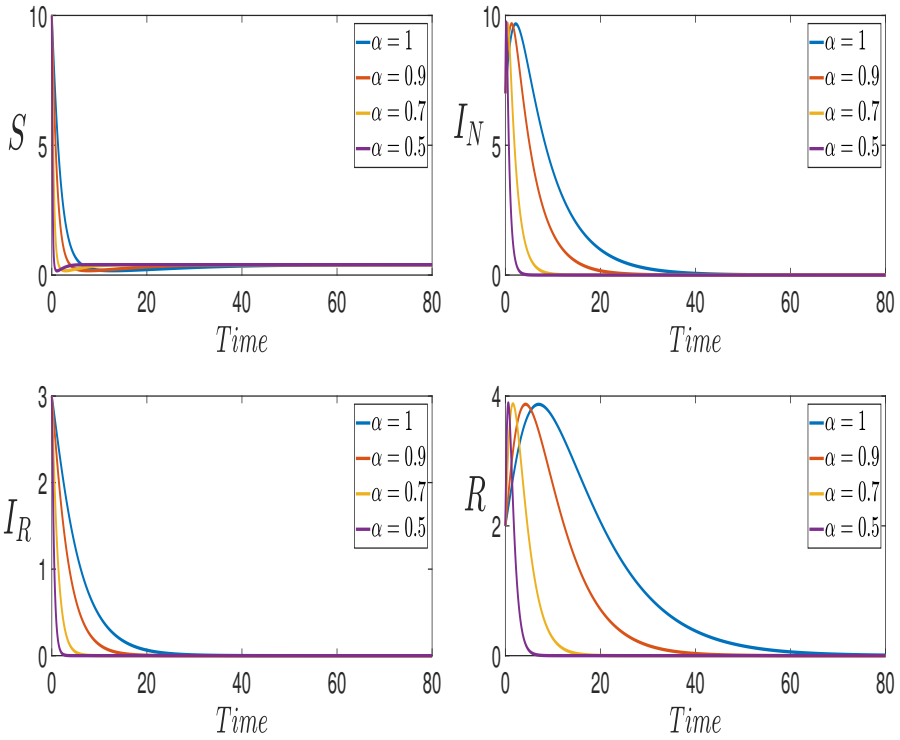

Figure 2: Stability of the disease-free equilibrium for different values of θ.

converges to the constant value $S_0 = \dfrac{b}{d} = 0.4$, in full agreement with the disease-free equilibrium. These numerical results are fully consistent with Theorem 3, which guarantees the global asymptotic stability of E_0 when $\max(R_N, R_R) \leq 1$. In this case, the basic reproduction numbers associated with the nonresistant and resistant strains are $R_N = 0.1333$ and $R_R = 0.0200$, respec-

tively, clearly satisfying the stability condition and confirming that the disease cannot persist in the population.

6.2 The nonresistant equilibrium point E_1

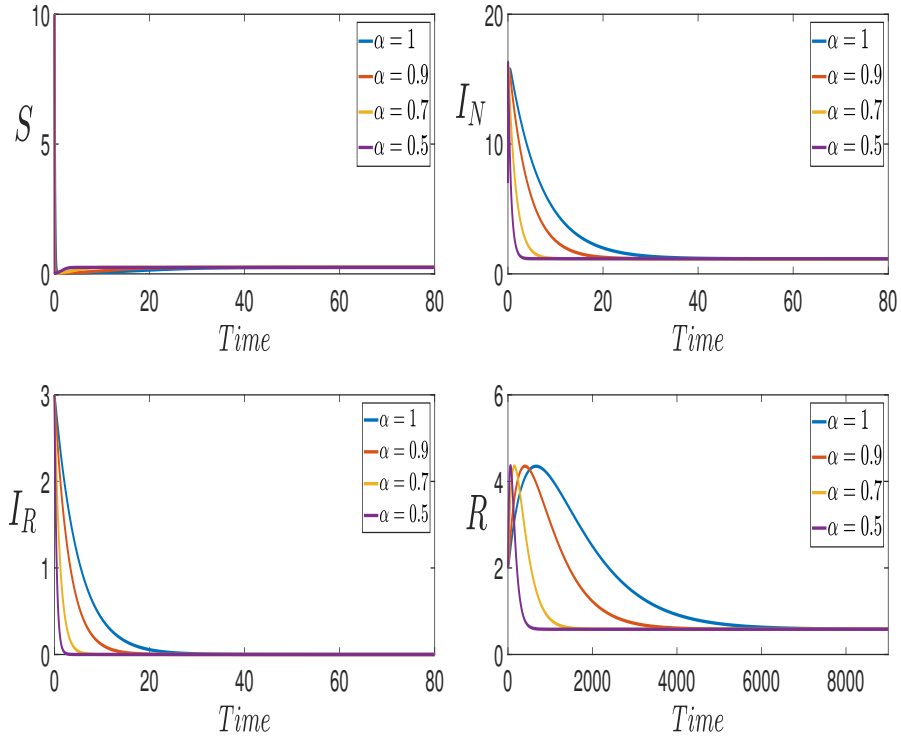

Figure 3: Stability of the nonresistant equilibrium point E_1 for different values of θ.

Figure 3 illustrates the behavior of the system toward the nonresistant equilibrium point $E_1 = (0.25, 1.1667, 0, 0.5833)$, under the conditions $R_N > 1$ and $R_N > R_R$. According to Theorem 4, these conditions ensure the global asymptotic stability of the equilibrium point E_1, which corresponds to a situation where the nonresistant strain persists while the resistant strain is eliminated. The numerical simulations are performed using parameter values satisfying $R_N > 1 \geq R_R$, with $R_N = 8.00$ and $R_R = 0.1999$, thereby confirming the theoretical stability conditions for E_1.

6.3 The resistant equilibrium E_2

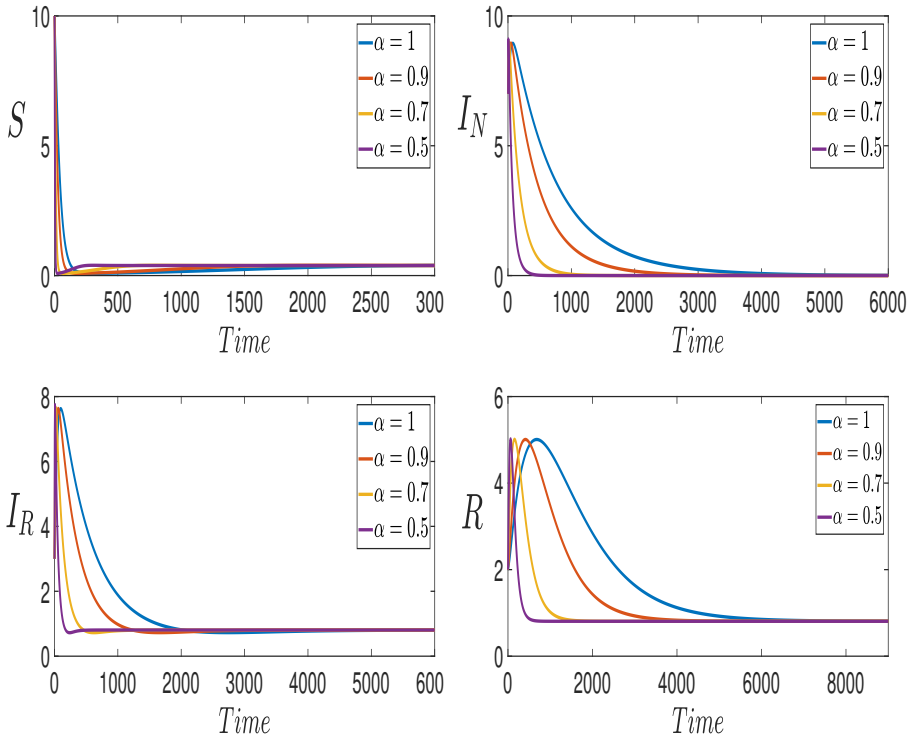

Figure 4: Stability of the resistant equilibrium point E_2 for different values of θ.

Figure 4 illustrates the dynamics of the system toward the resistant equilibrium point $E_2 = (0.3871, 0, 0.8065, 0.8065)$, for different values of the fractional-order parameter θ, where the resistant strain dominates the infection dynamics. According to Theorem 5, this equilibrium is globally asymptotically stable whenever the basic reproduction number of the resistant strain satisfies $R_R > 1$ and $R_R > R_N$. The numerical simulations are in full agreement with these theoretical results. In particular, the resistant infected population $I_R(t)$ converges to a positive steady-state value, indicating the persistent circulation of the resistant strain in the population. In contrast, the nonresistant infected class $I_N(t)$ decreases monotonically to zero, showing that the nonresistant strain cannot persist under these conditions. Moreover, the recovered population $R(t)$ initially increases due to recovery from infection and subsequently approaches a constant equilibrium level, while the susceptible population $S(t)$ converges to a low-level steady state after

a transient phase. For the chosen parameter values, the reproduction numbers are $R_R = 5.9999$ and $R_N = 0.6666$, which confirms the validity of the stability conditions stated in Theorem 5.

6.4 The endemic equilibrium E_3

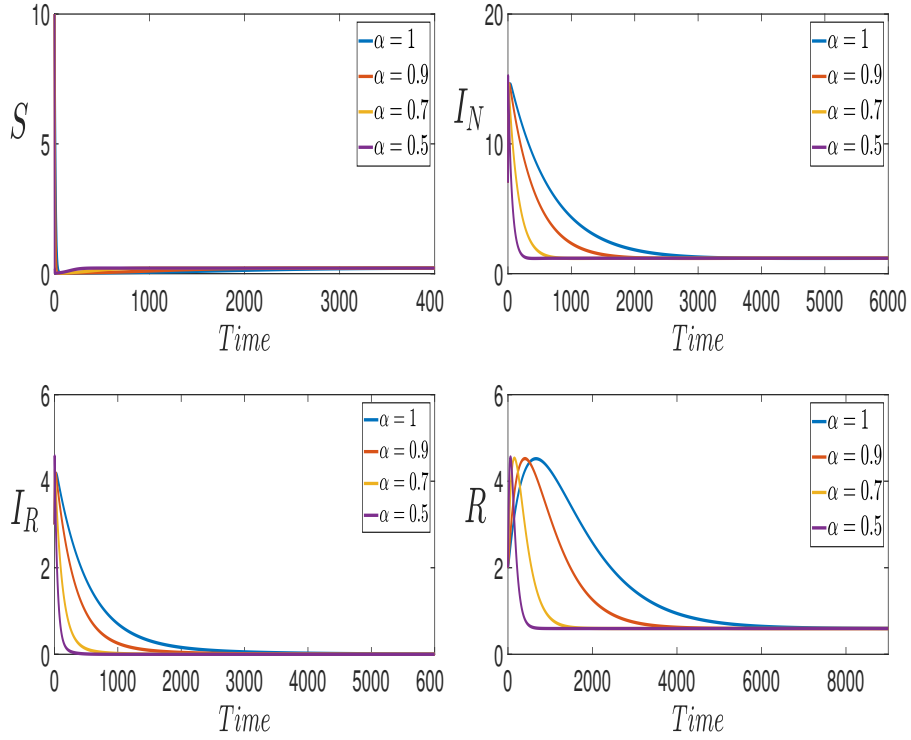

Figure 5: Stability of the endemic equilibrium point E_3 for different values of θ.

Figure 5 illustrates the temporal evolution of the system in all compartments. For different values of the fractional-order parameter θ, all trajectories converge toward the endemic equilibrium point $E_3 = (0.1667, 1.1111, 0.8333, 1.3889)$. According to Theorem 6, whenever the endemic equilibrium E_3 exists, it is globally asymptotically stable. The numerical simulations confirm that both infected classes $I_N(t)$ and $I_R(t)$ approach positive steady-state values, highlighting the long-term coexistence of the nonresistant and resistant strains. Similarly, the susceptible population $S(t)$ and the recovered population $R(t)$ converge to constant equilibrium levels, indicating the persistence of the infection in the population. For the selected parameter set, the

basic reproduction numbers satisfy $R_N > 1$ and $R_R > 1$, in full agreement with the theoretical results.

Now, regarding the effect of the fractional-order derivative on the behavior of the infection dynamics, the previous figures show that for smaller values of the fractional-order parameter θ, the model converges rapidly toward the corresponding stable equilibrium states. In contrast, larger values of θ result in a much slower convergence to the steady states, which can be interpreted as a stronger memory effect. Moreover, variations in the fractional-order parameter do not affect the stability of the equilibrium points themselves, but rather influence the time required for the system to reach these steady states. However, our model has some limitations and does not allow predicting the long-term dynamics for the other cases.

7 Conclusion

In this work, we proposed a fractional-order SIR-type epidemic model incorporating two viral strains resistant and nonresistant via Caputo derivatives to capture memory effects in the biological processes better. We rigorously established the existence, uniqueness, positivity, and boundedness of solutions, ensuring the model's consistency with epidemiological reality.

The analysis centered around the basic reproduction numbers R_N and R_R, derived for the nonresistant and resistant strains, respectively. Using Lyapunov functions and the fractional version of LaSalle's invariance principle, we proved the global asymptotic stability of four biologically meaningful equilibria, depending on the comparative values of R_N and R_R. Specifically, the disease-free equilibrium E_0 is globally asymptotically stable when $\max(R_N, R_R) \leq 1$; the nonresistant equilibrium E_1 becomes globally stable when $R_N > 1$ and $R_N > R_R$; the resistant equilibrium E_2 is stable when $R_R > 1$ and dominates R_N; and the endemic equilibrium E_3 is globally asymptotically stable when both reproduction numbers are equal and greater than one, that is, $R_N \geq R_R > 1$.

Numerical simulations were performed to validate the theoretical results. They demonstrated that, regardless of the value of the fractional order $\theta \in (0, 1]$, the system consistently converges to the appropriate equilibrium predicted by the reproduction numbers. Importantly, the value of θ influences the speed of convergence, but not the qualitative stability with smaller θ inducing faster dynamics due to memory effects.

This study illustrates the added realism provided by fractional calculus in epidemic modeling, especially when dealing with treatment response and mutation-induced resistance. As future work, one may consider extending this model to include stochastic perturbations or investigating bifurcation behavior to further characterize transitions between equilibria. This suggested model

has some limitations and does not allow predicting the long-term dynamics for the other reproduction numbers cases. An important extension of the present work would be to incorporate a treatment-induced transition from the nonresistant infected class I_N to the resistant class I_R, which may represent amplification due to incomplete or inappropriate treatment. Such a mechanism would modify the structure of the endemic equilibrium and the Lyapunov-based stability analysis. Although the coexistence of both strains is expected to persist for sufficiently small amplification rates, the global asymptotic stability of the coexistence equilibrium E_3 cannot be guaranteed without a separate analysis. This issue will be addressed in future work.

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Conflicts of Interest

The author declares no conflicts of interest.

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