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ANALYSIS AND OPTIMAL CONTROL OF A FRACTIONAL-ORDER SEAIR EPIDEMIC MODEL WITH TWO-STRAINS

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Abstract. This study focuses on the analysis and optimal control of a fractional-order SEAIR epidemic model, which consists of two strains. The proposed model's well-posedness is evaluated by examining its existence, uniqueness, non-negativity, and boundedness. Furthermore, two basic regeneration numbers are computed, and the model's two equilibrium points are the endemic and disease-free equilibriums. Using suitable Lyapunov functions and LaSalle's invariance principle, we conduct a stability analysis to examine the global stability of these steady states. Ultimately, using Pontryagin's Maximum Principle, we created a time-dependent optimal control problem. We evaluated the impact of model parameters on the dynamics of disease transmission and determined the efficacy of control measures using numerical simulations.

1. Introduction

Epidemic models are crucial for grasping how infectious diseases spread and can be controlled. Through mathematical frameworks these models allow researchers to replicate the intricate patterns of disease transmission and forecast possible outbreak developments [32, 24, 12, 37]. In 1766, Daniel Bernoulli developed a model that examined the effect of smallpox variation on population survival rates, marking the start of the application of mathematics to disease transmission [9]. The foundation for further developments was established by this early work. In 1927, for example, Kermack and McKendrick defined epidemic dynamics using a system of differential equations, establishing a framework that is still widely used today [13]. Since then, mathematical modeling has become central to public health planning, with a vast array of models developed to capture various aspects of infectious disease dynamics. The majority of conventional models rely on integer-order differential equations, which have been extensively employed to analyze the stability and behavior of various epidemic scenarios, including SEIR, SEIAHR models with standard contact rates and immunity considerations, as

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well as delayed SIRS models that incorporate temporary immunity and saturation incidence [34, 8, 20, 36].

Recently, fractional-order differential equation models have become valuable tools in epidemiology. One of the key benefits of using fractional derivatives in mathematical models is that they permit integrals and derivatives of any real or complex order, indicating that a system's previous and current states have an impact on its future state. As a result, fractional derivatives can represent the memory and heredity features essential to many biological processes, making this property particularly valuable for modeling dynamic systems [10, 14, 17, 35, 6]. The foundations of fractional calculus trace back to 1695, when Leibniz was asked an intriguing question by L'Hospital about the meaning of a half-order derivative. Thanks to the efforts of mathematicians like Euler in the 1730s and Lagrange in the mid-18th century, this curiosity set the stage for further investigation. Laplace formulated a fractional derivative in 1812 through an integral approach, and by 1819, Lacroix had introduced the concept of derivatives of arbitrary order in his work. Over time, notable figures such as Riemann and Liouville developed what became known as the Riemann-Liouville fractional derivative, while Hadamard expanded on these concepts. In the 20th century, Caputo introduced a derivative that gained popularity for modeling physical processes, while recent developments by Atangana and Baleanu have provided new approaches that continue to drive innovation in the field of fractional calculus [30, 7].

Infectious diseases can be divided into two general categories: single-strain and multi-strain infections, according to the source of the pathogens. In single-strain infectious diseases, only one pathogen variation is involved, making their control methods and transmission dynamics easier to simplify. Examples include measles, hepatitis B, and rubella [4, 22, 11]. Conversely, several pathogen strains that coexist and can develop independently or in reaction to external circumstances are the source of multi-strain infectious diseases, including influenza, dengue, and COVID-19 with its various variants [15, 33, 16]. These multi-strain infections present significant challenges for public health due to the complex interactions between strains, which can lead to phenomena such as strain dominance, cocirculation, or competitive exclusion [1, 2]. While each strain may differ in its virulence, transmissibility, and sensitivity to control strategies, understanding the dynamics of multi-strain infections is crucial for creating successful treatments. Therefore, models that incorporate multiple strains offer a more complete framework for investigating the dynamics of infectious disease transmission and control in complex epidemiological environments.

In the management of infectious diseases, optimal control theory is essential since it provides a systematic framework for designing intervention strategies that are not only effective in reducing transmission but also resource-efficient. This strategy supports health authorities to prioritize actions that will have the most effect at the lowest possible cost by optimizing the allocation of limited public health resources, such as vaccines, antiviral drugs, and isolation facilities. The mathematical structure of optimal control provides a way to balance competing demands, such as the urgency to curb infection rates versus the need to minimize economic and social disruptions caused by interventions. It enables

the precise timing and scaling of measures, ensuring that interventions are deployed at the appropriate time and intensity to achieve the desired health results without exhausting available resources. The mathematical framework known as Pontryagin's Maximum Principle, which describes the ideal circumstances for an intervention strategy, is one of the most widely used tools in this domain. This strategy can be particularly useful in complicated epidemiological scenarios, as sustainable and efficient disease control needs to find a balance between several goals, such as reducing infection rates while conserving resources. In this context, Khan et al. apply Pontryagin's maximum principle to determine the optimal treatment control approach of an SEIR epidemic model incorporating treatment function and incidence saturation.

This paper is organized as follows: Section 2 Provides some fundamental properties of the Caputo fractional derivative operator. In Section 3, the $SEAIR$ model with two-strains and its fractional formulation under the Caputo derivative is presented. In Section 4, we investigate the existence, uniqueness, nonnegativity, and boundedness of the solutions to ensure that the model is mathematically feasible. Additionally, establish the basic reproduction number and the two equilibrium points of the model: the endemic equilibrium and the disease-free equilibrium. The global stability of the equilibrium points by a stability analysis using Lyapunov functions and LaSalle's invariance principle is confirmed in Section 5. Section 6 discusses the optimal control theory and provides the optimal control solution. In Section 7, we describe numerical solutions and get observations. Finally, the paper concludes with a conclusion of the main results .

2. Preliminaries

This section presents the basic concepts of fractional differential calculus, with an emphasis on derivatives defined in the Caputo sense and several essential lemmas for the subsequent analysis.

Definition 2.1. Considering an integrable function $f(t)$, the fractional integral of order $\alpha > 0$ is given as

$$\mathbb{I}_t^{\alpha} f(t) = \frac{1}{\Gamma(\alpha)} \int_0^t (t-s)^{\alpha-1} f(s)\, \mathrm{d}s, \quad t \geq 0.$$

$\Gamma(\cdot)$ is the gamma function, $\Gamma(\alpha) = \int_0^{\infty} r^{\alpha-1} e^{-r}\, \mathrm{d}r$.

Definition 2.2. ([28].)

The fractional derivative in the sens of Caputo of order α for the function $f(t) \in \mathcal{C}^n([0,\infty), \mathbb{R})$ is provided by

$${}_0^c\mathbb{D}_t^{\alpha} f(t) = \frac{1}{\Gamma(n-\alpha)} \int_0^t \frac{f^{(n)}(s)}{(t-s)^{\alpha-n+1}}\, \mathrm{d}s,$$

where $t \geq 0$, while n is a positive integer in a way that $n-1 < \alpha < n$. Furthermore, when $0 < \alpha < 1$

$${}_0^c\mathbb{D}_t^{\alpha} f(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t \frac{f'(s)}{(t-s)^{\alpha}}\, \mathrm{d}s.$$

Definition 2.3. ([5].)
The Mittag-Leffler function for two parameters is provided by

$$E_{\alpha,\beta}(z) = \sum_{n=0}^{\infty} \frac{z^n}{\Gamma(\alpha\ n + \beta)}, \qquad \alpha,\ \beta > 0,\ z \in \mathbb{C}.$$

It is significant to notice that the Mittag-Leffler function $E_{1,1}$ reduces to the exponential function $exp(z)$ when $\alpha = \beta = 1$.

Lemma 2.4. *([28].)*
For $r > 0$ and $\alpha, \beta > 0$, the Laplace transform of the Mittag-Leffler function is given by

$$\mathcal{L}(t^{\alpha-1}E_{\alpha,\beta}(rt)) = \frac{s^{\alpha-\beta}}{s^\alpha - a}.$$

And,

$$\mathcal{L}({}^{C}D_t^\alpha h(t)) = s^{\alpha-1}\hat{h}(s) - \sum_{i=0}^{n-1} h^i(0)s^{\alpha-i-1}.$$

Where $\hat{h}(s) = \mathcal{L}(h(t))$.

Lemma 2.5. *([26].)*
Suppose that $\Phi : [t_0, \infty) \to \mathbb{R}$ is a continuous function, and satisfy

$$\begin{cases} {}_0^c\mathbb{D}_t^\alpha \Phi(t) + \lambda\Phi(t) \leq \mu, \\ \Phi(t_0) = \Phi_0. \end{cases} \tag{2.1}$$

Where $\alpha \in (0, 1], \lambda, \mu \in \mathbb{R}$, and the initial time is $t_0 \geq 0$. Subsequently, there is the inequality that follows:

$$\Phi(t) \leq (\Phi_0 - \frac{\mu}{\lambda})E_\alpha(-\lambda(t - t_0)^\alpha) + \frac{\mu}{\lambda} \tag{2.2}$$

Lemma 2.6. *([21].)*
For $0 < \alpha < 0$, let $\xi \in [0, T]$ be a function with positive values. Thus, for all $t \in [0, T)$

$${}_0^c\mathbb{D}_t^\alpha \left(\xi(t) - \xi^* - \xi^* ln\frac{\xi(t)}{\xi^*}\right) \leq \left(1 - \frac{\xi^*}{\xi(t)}\right) {}^c\mathbb{D}_t^\alpha \xi(t),\ \textit{for all } \xi^* \in \mathbb{R}_+.$$

Lemma 2.7. *([23].)*
Consider the following fractional-order system using the Caputo sense:

$$\begin{cases} {}_0^c\mathbb{D}_t^\alpha w(t) = \Phi(t, w(t)),\ \textit{for all } t \geq 0, \\ w(0) = w_0 \in \mathbb{R}^n. \end{cases} \tag{2.3}$$

Assume that the function $\Phi : \mathbb{R}_+ \times \mathbb{R}^n \to \mathbb{R}^n$ for $n \geq 1$, satisfies

1. *Φ is a continuous function with regard to t for any $w(t) \in \mathbb{R}^n$,*

2. Φ *and* $\frac{\partial \Phi}{\partial w}$ *are continuous functions with respect to* $w(t) \in \mathbb{R}^n$,
3. $\|\Phi\| \leq a_1 + a_2\|w\|$ *for all* $w \in \mathbb{R}^n$, *and all* $a_1, a_2 > 0$.

Thus, the system (2.3) has a unique solution on $[0, +\infty)$.

3. Model Derivation

We suggest a SEAIR type model with two-strain. This structure provides a comprehensive framework to illustrate how a disease spreads over a population by describing how individuals progress through several phases of disease. The total population, $N(t) = S(t) + E_1(t) + E_2(t) + A_1(t) + A_2(t) + I_1(t) + I_2(t) + R(t)$, includes Susceptible $S(t)$, Exposed $(E_1(t), E_2(t))$, Asymptomatic $(A_1(t), A_2(t))$, Infectious $(I_1(t), I_2(t))$ and Recovered $R(t)$ compartments, where strains 1 and 2 are indicated by the subscripts, respectively. All individuals in each compartment die naturally throughout the time at a rate of μ. The susceptible population (S) is replenished through recruitment at a rate of A_N, while the assumption of constant recruitment simplifies the model, it offers a first-order approximation of population replenishment. Prospective extensions may incorporate time-dependent recruitment rates to better capture real-world demographic variations. The exposed compartments, E_1 and E_2, represent the time between beginning exposure to each strain and the moment at which people are able to spread the disease, this exposure is governed by the transmission terms $\beta_1 S(t) I_1(t)$, $\beta_2 S(t) A_1(t)$, $\beta_3 S(t) I_2(t)$ and $\beta_4 S(t) A_2(t)$, which represent the infection rates due to contact with infectious and asymptomatic individuals for each strain. Exposed individuals leave these compartments at rates w_1 and w_2 to become either asymptomatic or symptomatic. The asymptomatic class A_1 is reached by a proportion p of exposed individuals E_1 for strain 1, while the symptomatic infectious class I_1 is reached by the remaining $(1-p)$. A similar process occurs for E_2 with transition rates qw_2 and $(1-q)w_2$ to the asymptomatic A_2 and infectious I_2 compartments, respectively. While asymptomatic people in A_1 and A_2 can transmit the infection, they may also recover at γ_1 and γ_2 rates. Conversely, infectious persons I_1 and I_2 either recover at rates δ_1 and δ_2 or die of disease-specific causes at rates η_1 and η_2. Furthermore, after recovering, individuals move to recovered compartment R, where they are assumed to develop immunity and leave the cycle of disease transmission. For simplicity, the model assumes a homogeneous population, which enables a clearer analysis of the key dynamics governing the interactions between the two viral strains and the impact of control measures. Future studies could incorporate heterogeneity, such as age or spatial structure, to enhance the model's applicability to real-world scenarios.
Figure 1 illustrates the SEAIR prevalence flowchart.

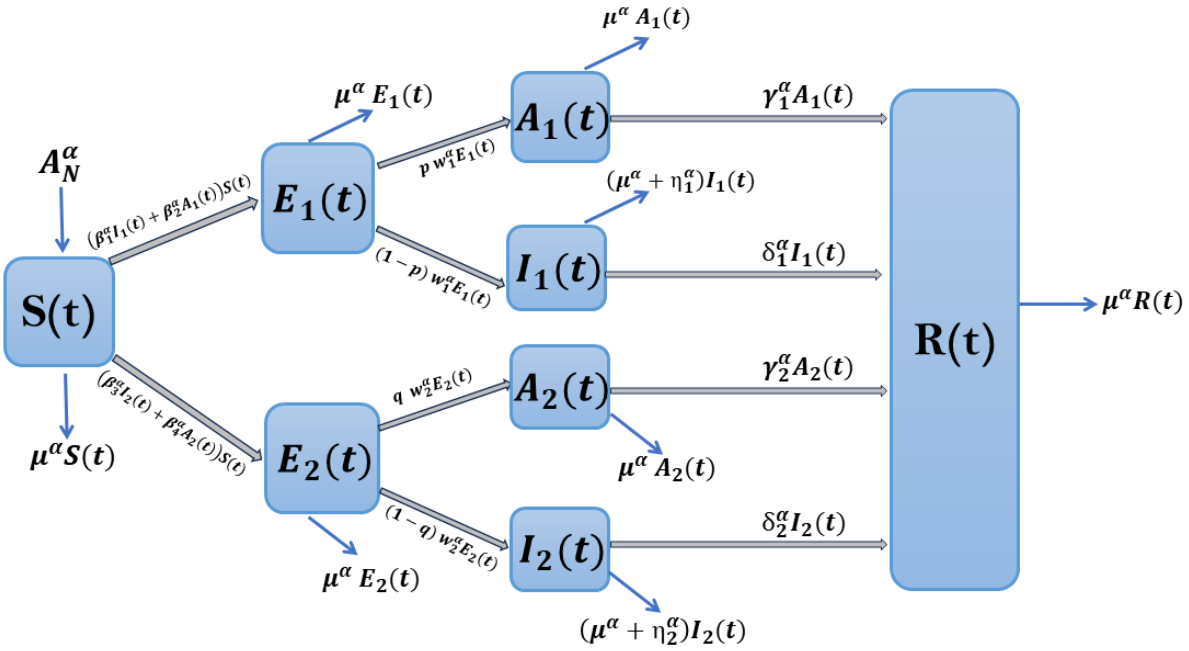


FIGURE 1. Flowchart of $SEAIR$ Epidemic model.

The following is an expression for the epidemic model SEAIR

$$\begin{cases} \frac{dS}{dt} = A_N - (\beta_1 I_1(t) + \beta_2 A_1(t) + \beta_3 I_2(t) + \beta_4 A_2(t))S(t) - \mu S(t), \\ \frac{dE_1}{dt} = (\beta_1 I_1(t) + \beta_2 A_1(t))S(t) - (w_1 + \mu)E_1(t), \\ \frac{dE_2}{dt} = (\beta_3 I_2(t) + \beta_4 A_2(t))S(t) - (w_2 + \mu)E_2(t), \\ \frac{dA_1}{dt} = pw_1 E_1(t) - (\gamma_1 + \mu)A_1(t), \\ \frac{dA_2}{dt} = qw_2 E_2(t) - (\gamma_2 + \mu)A_2(t), \\ \frac{dI_1}{dt} = (1-p)w_1 E_1(t) - (\delta_1 + \eta_1 + \mu)I_1(t), \\ \frac{dI_2}{dt} = (1-q)w_2 E_2(t) - (\delta_2 + \eta_2 + \mu)I_2(t), \\ \frac{dR}{dt} = \gamma_1 A_1(t) + \gamma_2 A_2(t) + \delta_1 I_1(t) + \delta_2 I_2(t) - \mu R(t). \end{cases} \tag{3.1}$$

with initial conditions

$$S(0) \geq 0,\ E_1(0) \geq 0,\ E_2(0) \geq 0,\ A_1(0) \geq 0,\ A_2(0) \geq 0,\ I_1(0) \geq 0,\ I_2(0) \geq 0,\ R(0) \geq 0. \tag{3.2}$$

The parameters of system (3.1) are positive and described as follows

TABLE 1. Description of model parameters in terms of biology.

Parameter	Description
A_N	The recruitment rate, which represents the new members joining the susceptible population.
β_i	the infection rate from S to E.
w_1, w_2	Progression rates from exposed to asymptomatic or symptomatic.
p, q	Proportion of exposed individuals who become asymptomatic (for strain 1 and 2)
δ_1, δ_2	Rates of recovery among individuals with symptoms (for strains 1 and 2).
γ_1, γ_2	Recovery rates of asymptomatic individuals (for strain 1 and 2).
η_1, η_2	Mortality rates from disease in patients with symptoms (for strains 1 and 2).

As previously mentioned, fractional-order differential equations, due to their nonlocal properties, offer a more effective approach than integer-order models. They excel in capturing dynamic systems characterized by path and time dependence, memory effects, and genetic traits. These unique features make fractional-order models particularly well-suited for accurately describing complex phenomena. Therefore, motivated by the classical SEAIR epidemic model (3.1) can be expanded further to the fractional-order system that follows, where ${}_0^c\mathbb{D}_t^\alpha$ represents the fractional Caputo derivative with order $0 < \alpha \leq 1$.

$$\begin{cases} {}_0^c\mathbb{D}_t^\alpha S(t) = A_N^\alpha - (\beta_1^\alpha I_1(t) + \beta_2^\alpha A_1(t) + \beta_3^\alpha I_2(t) + \beta_4^\alpha A_2(t))S(t) - \mu^\alpha S(t), \\ {}_0^c\mathbb{D}_t^\alpha E_1(t) = (\beta_1^\alpha I_1(t) + \beta_2^\alpha A_1(t))S(t) - (w_1^\alpha + \mu^\alpha)E_1(t), \\ {}_0^c\mathbb{D}_t^\alpha E_2(t) = (\beta_3^\alpha I_2(t) + \beta_4^\alpha A_2(t))S(t) - (w_2^\alpha + \mu^\alpha)E_2(t), \\ {}_0^c\mathbb{D}_t^\alpha A_1(t) = pw_1^\alpha E_1(t) - (\gamma_1^\alpha + \mu^\alpha)A_1(t), \\ {}_0^c\mathbb{D}_t^\alpha A_2(t) = qw_2^\alpha E_2(t) - (\gamma_2^\alpha + \mu^\alpha)A_2(t), \\ {}_0^c\mathbb{D}_t^\alpha I_1(t) = (1-p)w_1^\alpha E_1(t) - (\delta_1^\alpha + \eta_1^\alpha + \mu^\alpha)I_1(t), \\ {}_0^c\mathbb{D}_t^\alpha I_2(t) = (1-q)w_2^\alpha E_2(t) - (\delta_2^\alpha + \eta_2^\alpha + \mu^\alpha)I_2(t), \\ {}_0^c\mathbb{D}_t^\alpha R(t) = \gamma_1^\alpha A_1(t) + \gamma_2^\alpha A_2(t) + \delta_1^\alpha I_1(t) + \delta_2^\alpha I_2(t) - \mu^\alpha R(t). \end{cases} \tag{3.3}$$

with the primary conditions (3.2).

4. Fractional Order Model Analysis

4.1. **Existence and Uniqueness of Solutions.** The existence, non-negativity, uniqueness, and boundedness of solutions for the fractional-order model (3.3) are presented in this subsection.

To prove the existence and uniqueness, we will utilize the Banach contraction principle to derive the main results [19]. Let the fractional-order model (3.3) in the Caputo sense be expressed in the following form:

$$ {}_0^c\mathbb{D}_t^{\alpha} w(t) = \Phi(t, w(t)), \ \ For\ t \geq 0, \ and\ w(0) = w_0 \in \mathbb{R}_+^8. $$

Where

$$ w(t) = (S(t), E_1(t), E_2(t), A_1(t), A_2(t), I_1(t), I_2(t), R(t))^T. $$

And

$$ \Phi(t, w(t)) = \begin{pmatrix} A_N^{\alpha} - (\beta_1^{\alpha} I_1(t) + \beta_2^{\alpha} A_1(t) + \beta_3^{\alpha} I_2(t) + \beta_4^{\alpha} A_2(t))S(t) - \mu^{\alpha} S(t) \\ (\beta_1^{\alpha} I_1(t) + \beta_2^{\alpha} A_1(t))S(t) - (w_1^{\alpha} + \mu^{\alpha})E_1(t) \\ (\beta_3^{\alpha} I_2(t) + \beta_4^{\alpha} A_2(t))S(t) - (w_2^{\alpha} + \mu^{\alpha})E_2(t) \\ p w_1^{\alpha} E_1(t) - (\gamma_1^{\alpha} + \mu^{\alpha})A_1(t) \\ q w_2^{\alpha} E_2(t) - (\gamma_2^{\alpha} + \mu^{\alpha})A_2(t) \\ (1-p) w_1^{\alpha} E_1(t) - (\delta_1^{\alpha} + \eta_1^{\alpha} + \mu^{\alpha})I_1(t) \\ (1-q) w_2^{\alpha} E_2(t) - (\delta_2^{\alpha} + \eta_2^{\alpha} + \mu^{\alpha})I_2(t) \\ \gamma_1^{\alpha} A_1(t) + \gamma_2^{\alpha} A_2(t) + \delta_1^{\alpha} I_1(t) + \delta_2^{\alpha} I_2(t) - \mu^{\alpha} R(t) \end{pmatrix} $$

We set

$$ L = \begin{pmatrix} A_N^{\alpha} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \ ; \ Q_1 = \begin{pmatrix} -\mu^{\alpha} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -C_0^{\alpha} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -C_1^{\alpha} & 0 & 0 & 0 & 0 & 0 \\ 0 & p w_1^{\alpha} & 0 & -C_2^{\alpha} & 0 & 0 & 0 & 0 \\ 0 & 0 & q w_2^{\alpha} & 0 & -C_3^{\alpha} & 0 & 0 & 0 \\ 0 & (1-p) w_1^{\alpha} & 0 & 0 & 0 & -C_4^{\alpha} & 0 & 0 \\ 0 & 0 & (1-q) w_2^{\alpha} & 0 & 0 & 0 & -C_5^{\alpha} & 0 \\ 0 & 0 & 0 & \gamma_1^{\alpha} & \gamma_2^{\alpha} & \delta_1^{\alpha} & \delta_2^{\alpha} & -\mu^{\alpha} \end{pmatrix} $$

and

$$ Q_2 = \begin{pmatrix} 0 & 0 & 0 & -\beta_2^{\alpha} & -\beta_4^{\alpha} & -\beta_1^{\alpha} & -\beta_3^{\alpha} & 0 \\ 0 & 0 & 0 & \beta_2^{\alpha} & 0 & \beta_1^{\alpha} & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_4^{\alpha} & 0 & \beta_3^{\alpha} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} $$

Then the function Φ can be expressed as follow

$$ \Phi(t, w(t) = L + Q_1 w(t) + Q_2 S(t) w(t). \tag{4.1} $$

We have the vector function $w(t)$ satisfies the first and second conditions of Lemma 2.7 .

Furthermore, the third condition is verified. Indeed,

$$\|\Phi(t, w(t))\| \leq \|L\| + [\|Q_1\| + \|Q_2\| \ \|S\|]\|w\|. \tag{4.2}$$

Hence, by using lemma 2.7, the system (3.3) has a unique solution.

4.2. **Positivity and Boundedness of the Solution.** This section evaluates the boundedness and positivity of the solution to our model. Verifying these properties ensures that the system's behavior is consistent with epidemiological assumptions and offers a robust foundation for further analysis of the model's dynamics.

Theorem 4.1. *The set*

$$\Theta = \{(S, E_1, E_2, A_1, A_2, I_1, I_2, R) \in \mathbb{R}_+^8 \text{ Such that } :0 < S(t)+E_1(t)+E_2(t)+A_1(t)+A_2(t)+I_1(t)+I_2(t)+R(t) \leq \frac{A^\alpha}{\mu^\alpha}\}.$$

is a positively invariant and attraction region for system (3.3).

Remark 4.2. Assume that $f(t) \in C[a, b]$ and ${}_0^c\mathbb{D}_t^\alpha f(t) \in C[a, b]$, for $0 < \alpha \leq 1$.

(i): If ${}_0^c\mathbb{D}_t^\alpha f(t) \geq 0$, $\forall t \in [a, b]$, then $f(t)$ is non-decreasing for each $t \in [a, b]$.

(ii): If ${}_0^c\mathbb{D}_t^\alpha f(t) \leq 0$, $\forall t \in (a, b)$, then $f(t)$ is non-increasing for each $t \in [a, b]$.

Proof. We start by presenting the positivity of the solution with

$${}_0^c\mathbb{D}_t^\alpha S(t)|_{S=0} = A_N^\alpha \geq 0,$$

$${}_0^c\mathbb{D}_t^\alpha E_1(t)|_{E_1=0} = (\beta_1^\alpha I_1(t) + \beta_2^\alpha A_1(t))S(t) \geq 0,$$

$${}_0^c\mathbb{D}_t^\alpha E_2(t)|_{E_2=0} = (\beta_3^\alpha I_2(t) + \beta_4^\alpha A_2(t))S(t) \geq 0,$$

$${}_0^c\mathbb{D}_t^\alpha A_1(t)|_{A_1=0} = pw_1^\alpha E_1(t) \geq 0,$$

$${}_0^c\mathbb{D}_t^\alpha A_2(t)|_{A_2=0} = qw_2^\alpha E_2(t) \geq 0,$$

$${}_0^c\mathbb{D}_t^\alpha I_1(t)|_{I_1=0} = (1-p)w_1^\alpha E_1(t) \geq 0,$$

$${}_0^c\mathbb{D}_t^\alpha I_2(t)|_{I_2=0} = (1-q)w_2^\alpha E_2(t) \geq 0,$$

$${}_0^c\mathbb{D}_t^\alpha R(t)|_{R=0} = \gamma_1^\alpha A_1(t) + \gamma_2^\alpha A_2(t) + \delta_1^\alpha I_1(t) + \delta_2^\alpha I_2(t) \geq 0.$$

Subsequently, the boundedness of the solution is established by the sum of individual equations of the system (3.3).

Hence,

$$ {}_0^c\mathbb{D}_t^\alpha N(t) = A_N^\alpha - \mu^\alpha N(t) - \eta_1^\alpha I_1(t) - \eta_2^\alpha I_2(t), \tag{4.3} $$

$$ {}_0^c\mathbb{D}_t^\alpha N(t) \leq A_N^\alpha - \mu^\alpha N(t), \tag{4.4} $$

$$ {}_0^c\mathbb{D}_t^\alpha N(t) + \mu^\alpha N(t) \leq A_N^\alpha. \tag{4.5} $$

As a result, Lemma (2.5) indicates that

$$ N(t) \leq (N(t_0) - \frac{A_N^\alpha}{\mu^\alpha}) E_\alpha(-\mu^\alpha (t-t_0)^\alpha) + \frac{A_N^\alpha}{\mu^\alpha}. \tag{4.6} $$

Additionally, when $t \longrightarrow +\infty$

$$ N(t) \leq \frac{A_N^\alpha}{\mu^\alpha}. \tag{4.7} $$

In summary, this completes the proof of non-negativity and boundedness.

□

4.3. **Equilibrium Points and Basic Reproduction Number.** This subsection addresses the recognition of equilibrium points in the model, which represent steady states in the absence or presence of the infection. Additionally, we derive the basic reproduction number R_0, a critical threshold that establishes the potential for disease persistence within the population.

In the scenario where the disease is completely absent from the population, all infection-related compartments are assumed to have zero population (i.e., $E_{1,2}^* = A_{1,2}^* = I_{1,2}^* = 0$). During these circumstances, the system stabilizes at the Disease-Free Equilibrium (DFE), which can be formally defined as:

$$ P_0 = (\frac{A_N^\alpha}{\mu^\alpha}, 0, 0, 0, 0, 0, 0, 0). \tag{4.8} $$

Additionally, as a key threshold parameter in compartmental models of disease transmission represented by systems of ordinary differential equations, the basic reproduction number R_0, must be determined. Furthermore, if the effective reproduction number (R_0) is less than 1, the infection will eventually be eradicated, as each infected individual transmits the disease to fewer than one other person on average. Conversely, when R_0 exceeds 1, an infected individual spreads the disease to multiple people, allowing the infection to propagate through the community.

Hence, by using the next-generation matrix method outlined in [31], we derive the basic reproduction number, R_0. This computation involves determining the dominant eigenvalue, commonly known as the spectral radius, of the associated matrix

$$ FV^{-1} = \left[\frac{\partial \mathcal{F}_i(P_0)}{\partial x_j}\right] \left[\frac{\partial \mathcal{V}_i(P_0)}{\partial x_j}\right]^{-1}. $$

Where

$$\mathcal{F} = \begin{pmatrix} 0 \\ (\beta_1^\alpha I_1(t) + \beta_2^\alpha A_1(t))S(t) \\ (\beta_3^\alpha I_2(t) + \beta_4^\alpha A_2(t))S(t) \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

$$\mathcal{V} = \begin{pmatrix} -A_N^\alpha + (\beta_1^\alpha I_1(t) + \beta_2^\alpha A_1(t) + \beta_3^\alpha I_2(t) + \beta_4^\alpha A_2(t))S(t) + \mu^\alpha S(t) \\ (w_1^\alpha + \mu^\alpha)E_1(t) \\ (w_2^\alpha + \mu^\alpha)E_2(t) \\ -pw_1^\alpha E_1(t) + (\gamma_1^\alpha + \mu^\alpha)A_1(t) \\ -qw_2^\alpha E_2(t) + (\gamma_2^\alpha + \mu^\alpha)A_2(t) \\ -(1-p)w_1^\alpha E_1(t) + (\delta_1^\alpha + \eta_1^\alpha + \mu^\alpha)I_1(t) \\ -(1-q)w_2^\alpha E_2(t) + (\delta_2^\alpha + \eta_2^\alpha + \mu^\alpha)I_2(t) \\ -\gamma_1^\alpha A_1(t) - \gamma_2^\alpha A_2(t) - \delta_1^\alpha I_1(t) - \delta_2^\alpha I_2(t) + \mu^\alpha R(t) \end{pmatrix}$$

Therefore, we obtain

$$FV^{-1} = \begin{pmatrix} \frac{\beta_1^\alpha A_N^\alpha (1-p)w_1^\alpha}{\mu^\alpha(w_1^\alpha+\mu^\alpha)(\delta_1^\alpha+\eta_1^\alpha+\mu^\alpha)} + \frac{\beta_2^\alpha A_N^\alpha p w_1^\alpha}{\mu^\alpha(w_1^\alpha+\mu^\alpha)(\gamma_1^\alpha+\mu^\alpha)} & 0 & 0\ 0\ 0\ 0\ 0\ 0 \\ 0 & \frac{\beta_3^\alpha A_N^\alpha (1-q)w_2^\alpha}{\mu^\alpha(w_2^\alpha+\mu^\alpha)(\delta_2^\alpha+\eta_2^\alpha+\mu^\alpha)} + \frac{\beta_4^\alpha A_N^\alpha q w_2^\alpha}{\mu^\alpha(w_2^\alpha+\mu^\alpha)(\gamma_2^\alpha+\mu^\alpha)} & 0\ 0\ 0\ 0\ 0\ 0 \\ 0 & 0 & 0\ 0\ 0\ 0\ 0\ 0 \\ 0 & 0 & 0\ 0\ 0\ 0\ 0\ 0 \\ 0 & 0 & 0\ 0\ 0\ 0\ 0\ 0 \\ 0 & 0 & 0\ 0\ 0\ 0\ 0\ 0 \\ 0 & 0 & 0\ 0\ 0\ 0\ 0\ 0 \\ 0 & 0 & 0\ 0\ 0\ 0\ 0\ 0 \end{pmatrix}$$

Hence

$$R_0 = \rho(FV^{-1}) = max\{R_0^1; R_0^2\} \tag{4.9}$$

With

$$R_0^1 = \frac{\beta_1^\alpha A_N^\alpha (1-p)w_1^\alpha}{\mu^\alpha(w_1^\alpha+\mu^\alpha)(\delta_1^\alpha+\eta_1^\alpha+\mu^\alpha)} + \frac{\beta_2^\alpha A_N^\alpha p w_1^\alpha}{\mu^\alpha(w_1^\alpha+\mu^\alpha)(\gamma_1^\alpha+\mu^\alpha)}, \tag{4.10}$$

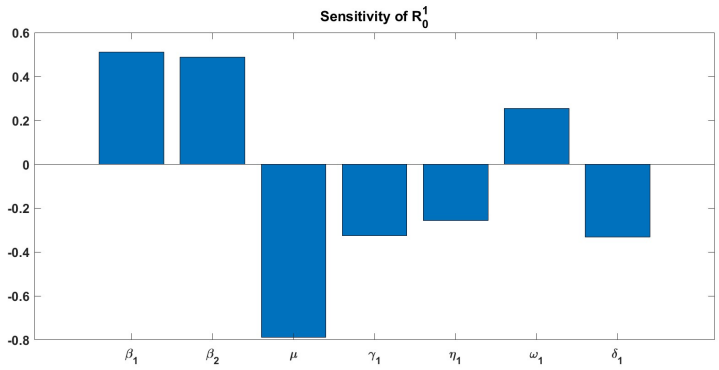
$$R_0^2 = \frac{\beta_3^\alpha A_N^\alpha (1-q)w_2^\alpha}{\mu^\alpha(w_2^\alpha+\mu^\alpha)(\delta_2^\alpha+\eta_2^\alpha+\mu^\alpha)} + \frac{\beta_4^\alpha A_N^\alpha q w_2^\alpha}{\mu^\alpha(w_2^\alpha+\mu^\alpha)(\gamma_2^\alpha+\mu^\alpha)} \tag{4.11}$$

.

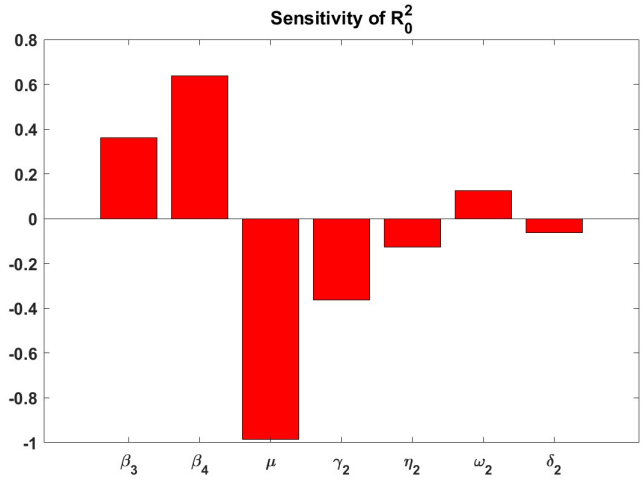
The expressions of R_0^1 and R_0^2 derived earlier provide valuable insights into the roles that different model parameters play in shaping the transmission dynamics of the disease. Through sensitivity analysis, we can determine which model parameters have the biggest effect on the basic reproduction number. Parameters with higher sensitivity values are more important in controlling how the disease spreads in the population. By adjusting these key parameters, we can help reduce or control the spread of the infection. Using the normalized forward sensitivity index method [3], we calculate the sensitivity of each parameter. The formula is shown below:

$$C_\rho^{R_0} = \frac{\partial R_0}{\partial \rho} \times \frac{\rho}{R_0} \tag{4.12}$$

A positive sensitivity index for a model parameter means that the basic reproduction number, R_0, increases as the parameter increases. Conversely, a negative sensitivity index means that R_0 decreases as the parameter increases.



(A)



(B)

FIGURE 2. (A) Impact of key parameters on R_0^1; (B) Impact of key parameters on R_0^2.

Following the analysis, it has been observed that the parameters β_1, β_2 and ω_1 exhibit a significant and positive correlation with R_0^1, while γ_1, η_1, δ_1 and μ exhibit a significant and negative correlation with R_0^1. Meanwhile, β_3, β_4 and ω_2 exhibit a significant and positive correlation with R_0^2, while the other parameters exhibit a significant and negative correlation.

In figure 2 , These parameters have different levels of impact on the disease. For strain-1, the spread of the disease can be reduced and controlled by decreasing β_1, β_2 and ω_1. For strain-2, the spread of the disease can also be reduced and controlled by decreasing β_3, β_4 and ω_2.

Next, we establish the existence of an endemic equilibrium according to the basic reproduction number (4.9).

Theorem 4.3. • *The strain 1 endemic equilibrium* $P_1^* = (S_{s1}^*, E_{1,s1}^*, 0, A_{1,s1}^*, 0, I_{1,s1}^*, 0, R_{s1}^*)$ *exists when* $R_0^1 > 1$ *and* $R_0^2 < 1$.

• *The strain 2 endemic equilibrium* $P_2^* = (S_{s2}^*, 0, E_{2,s2}^*, 0, A_{2,s2}^*, 0, I_{2,s2}^*, R_{s2}^*)$ *exists when* $R_0^2 > 1$ *and* $R_0^1 < 1$.

Proof. The following equations can be used to determine the endemic equilibrium points:

$$A_N^\alpha - (\beta_1^\alpha I_1^* + \beta_2^\alpha A_1^* + \beta_3^\alpha I_2^* + \beta_4^\alpha A_2^*)S^* - \mu^\alpha S^* = 0$$

$$(\beta_1^\alpha I_1^* + \beta_2^\alpha A_1^*)S^* - (w_1^\alpha + \mu^\alpha)E_1^* = 0$$

$$(\beta_3^\alpha I_2^* + \beta_4^\alpha A_2^*)S^* - (w_2^\alpha + \mu^\alpha)E_2^* = 0$$

$$pw_1^\alpha E_1^* - (\gamma_1^\alpha + \mu^\alpha)A_1^* = 0$$

$$qw_2^\alpha E_2^* - (\gamma_2^\alpha + \mu^\alpha)A_2^* = 0$$

$$(1-p)w_1^\alpha E_1^* - (\delta_1^\alpha + \eta_1^\alpha + \mu^\alpha)I_1^* = 0$$

$$(1-q)w_2^\alpha E_2^* - (\delta_2^\alpha + \eta_2^\alpha + \mu^\alpha)I_2^* = 0$$

$$\gamma_1^\alpha A_1^* + \gamma_2^\alpha A_2^* + \delta_1^\alpha I_1^* + \delta_2^\alpha I_2^* - \mu^\alpha R^* = 0$$

Case 1: For strain-1 endemic equilibrium P_1^*. Let $E_2^* = A_2^* = I_2^* = 0$.
As a result, the endemic equilibrium of strain-1 is given by

$$P_1^* = (S_{s1}^*, E_{1,s1}^*, 0, A_{1,s1}^*, 0, I_{1,s1}^*, 0, R_{s1}^*). \tag{4.13}$$

Where

$$S_{s1}^* = \frac{A_N^\alpha - (w_1^\alpha + \mu^\alpha)E_{1,s1}^*}{\mu^\alpha} \; ; \; A_{1,s1}^* = \frac{pw_1^\alpha E_{1,s1}^*}{\gamma_1^\alpha + \mu^\alpha} \; ; \; I_{1,s1}^* = \frac{(1-p)w_1^\alpha E_{1,s1}^*}{\delta_1^\alpha + \eta_1^\alpha + \mu^\alpha} \; ;$$

$$R^*_{s1} = \frac{\gamma_1^\alpha A^*_{1,s1} + \delta_1^\alpha I^*_{1,s1}}{\mu^\alpha} \quad \text{and } E^*_{1,s1} = \frac{(w_1^\alpha + \mu^\alpha)(R_0^1 - 1)}{M_1},$$
$$\text{with } M_1 = \left[\frac{\beta_1^\alpha (1-p) w_1^\alpha (w_1^\alpha + \mu^\alpha)}{\mu^\alpha(\delta_1^\alpha + \eta_1^\alpha + \mu^\alpha)} + \frac{\beta_2^\alpha p w_1^\alpha (w_1^\alpha + \mu^\alpha)}{\mu^\alpha(\gamma_1^\alpha + \mu^\alpha)}\right].$$

Case 2: For strain-2 endemic equilibrium P_2^*. Let $E_1^* = A_1^* = I_1^* = 0$. The endemic equilibrium of strain-2 is therefore established by

$$P_2^* = (S^*_{s2}, 0, E^*_{2,s2}, 0, A^*_{2,s2}, 0, I^*_{2,s2}, R^*_{s2}). \tag{4.14}$$

Where

$$S^*_{s2} = \frac{A_N^\alpha - (w_2^\alpha + \mu^\alpha) E^*_{2,s2}}{\mu^\alpha} \; ; \; A^*_{2,s2} = \frac{q w_2^\alpha E^*_{2,s2}}{\gamma_2^\alpha + \mu^\alpha} \; ; \; I^*_{2,s2} = \frac{(1-q) w_2^\alpha E^*_{2,s2}}{\delta_2^\alpha + \eta_2^\alpha + \mu^\alpha} \; ;$$
$$R^*_{s2} = \frac{\gamma_2^\alpha A^*_{2,s2} + \delta_2^\alpha I^*_{2,s2}}{\mu^\alpha} \quad \text{and } E^*_{2,s2} = \frac{(w_2^\alpha + \mu^\alpha)(R_0^2 - 1)}{M_2},$$
$$\text{with } M_2 = \left[\frac{\beta_3^\alpha (1-q) w_2^\alpha (w_2^\alpha + \mu^\alpha)}{\mu^\alpha(\delta_2^\alpha + \eta_2^\alpha + \mu^\alpha)} + \frac{\beta_4^\alpha q w_2^\alpha (w_2^\alpha + \mu^\alpha)}{\mu^\alpha(\gamma_2^\alpha + \mu^\alpha)}\right].$$

□

5. Global Stability Analysis

In this section, we establish the global stability of the FDEs (3.3) in the Caputo sense of both the disease-free equilibrium and the endemic equilibrium by creating appropriate Lyapunov functions.

We propose a function $\varpi : \mathbb{R}_+ \to \mathbb{R}_+$ such that

$$\varpi(\xi(t)) = \xi(t) - \xi^* - \xi^* ln \frac{\xi(t)}{\xi^*}, \; for \; all \; t \geq 0.$$

It is important to note that $\varpi(\xi)$ is a non-negative function for any $\xi > 0$, reaching its global minimum at $\xi = 1$.

Theorem 5.1. *The disease-free equilibrium P_0 of system (3.3) is globally asymptotically stable, if $R_0 \leq 1$.*

Proof. We suggest the Lyapunov function in the following form:

$$V_1(t) = 2\varpi(S(t)) + 2E_1(t) + 2E_2(t) + \frac{\alpha_1}{p w_1^\alpha} A_1 + \frac{\alpha_2}{q w_2^\alpha} A_2 + \frac{\alpha_1}{(1-p) w_1^\alpha} I_1 + \frac{\alpha_2}{(1-q) w_2^\alpha} I_2 \tag{5.1}$$

Such that

$$\alpha_1 = w_1^\alpha + \mu^\alpha \; ; \; \alpha_2 = w_2^\alpha + \mu^\alpha \; ; \; \alpha_3 = \gamma_1^\alpha + \mu^\alpha \; ; \; \alpha_4 = \gamma_2^\alpha + \mu^\alpha \; ; \; \alpha_5 = \delta_1^\alpha + \eta_1^\alpha + \mu^\alpha$$
$$and \; \alpha_6 = \delta_2^\alpha + \eta_2^\alpha + \mu^\alpha \tag{5.2}$$

.

Using the Caputo fractional derivative, we obtain

$$
\begin{aligned}
{}_0^c\mathbb{D}_t^\alpha V_1(t) &\leq 2\left(1-\frac{S_0}{S}\right)(A_N^\alpha-(\beta_1^\alpha I_1+\beta_2^\alpha A_1)S-(\beta_3^\alpha I_2+\beta_4^\alpha A_2)S-\mu^\alpha S)\\
&+2(\beta_1^\alpha I_1+\beta_2^\alpha A_1)S-2(w_1^\alpha+\mu^\alpha)E_1+2(\beta_3^\alpha I_2+\beta_4^\alpha A_2)S-2(w_2^\alpha+\mu^\alpha)E_2\\
&+\alpha_1 E_1-\frac{\alpha_1\alpha_3}{pw_1^\alpha}A_1+\alpha_2 E_2-\frac{\alpha_2\alpha_4}{qw_2^\alpha}A_2+\alpha_1 E_1-\frac{\alpha_1\alpha_5}{(1-p)w_1^\alpha}I_1+\alpha_2 E_2+\frac{\alpha_2\alpha_6}{(1-q)w_2^\alpha}I_2,\\
&\leq \frac{-2\mu^\alpha(S-S_0)^2}{S}+2\beta_1^\alpha I_1S_0+2\beta_2^\alpha A_1S_0+2\beta_3^\alpha I_2S_0+2\beta_4^\alpha A_2S_0-\frac{\alpha_1\alpha_3}{pw_1^\alpha}A_1-\frac{\alpha_2\alpha_4}{qw_2^\alpha}A_2\\
&-\frac{\alpha_1\alpha_5}{(1-p)w_1^\alpha}I_1+\frac{\alpha_2\alpha_6}{(1-q)w_2^\alpha}I_2,\\
&\leq \frac{-2\mu^\alpha(S-S_0)^2}{S}+\frac{2\alpha_1\alpha_3}{pw_1^\alpha}A_1\left(\frac{\beta_2^\alpha S_0pw_1}{\alpha_1\alpha_3}-\frac{1}{2}\right)+\frac{2\alpha_2\alpha_4}{qw_2^\alpha}A_2\left(\frac{\beta_4^\alpha S_0qw_2}{\alpha_2\alpha_4}-\frac{1}{2}\right)\\
&+\frac{2\alpha_1\alpha_5}{(1-p)w_1^\alpha}I_1\left(\frac{\beta_1^\alpha S_0(1-p)w_1}{\alpha_1\alpha_5}-\frac{1}{2}\right)+\frac{2\alpha_2\alpha_6}{(1-q)w_2^\alpha}I_2\left(\frac{\beta_3^\alpha S_0(1-q)w_2}{\alpha_2\alpha_6}-\frac{1}{2}\right).
\end{aligned}
$$

As a result, the condition $R_0 \leq 1$ guarantees that ${}_0^c\mathbb{D}_t^\alpha V_1(t) \leq 0$ for all $t \geq 0$. Furthermore, it can be shown that ${}_0^c\mathbb{D}_t^\alpha V_1(t) = 0$ particularly at the disease-free equilibrium point P_0. According to the Lyapunov-LaSalle asymptotic stability theorem [18, 25], P_0 is globally asymptotically stable.

□

Theorem 5.2. *Concerning the endemic strain 2. If $R_0^2 > 1$, then the endemic equilibrium point P_2^* of model (3.3) is globally asymptotically stable in the region Ω.*

Proof. The candidate Lyapunov function is defined as

$$V_2(t) = \varpi(S(t)) + \varpi(E_2(t)) + \varpi(A_2(t)) + \varpi(I_2(t)) \tag{5.3}$$

The Caputo fractional derivative is applied to obtaining

$$
\begin{aligned}
{}_0^{c}\mathbb{D}_t^{\alpha} V_2(t) &\leq \left(1-\frac{S^*}{S}\right)\left(A_N^{\alpha}-(\beta_1^{\alpha} I_1+\beta_2^{\alpha} A_1)S-(\beta_3^{\alpha} I_2+\beta_4^{\alpha} A_2)S-\mu^{\alpha} S\right)\\
&+\left(1-\frac{E_2^*}{E_2}\right)\left[(\beta_3^{\alpha} I_2+\beta_4^{\alpha} A_2)S-(w_2^{\alpha}+\mu^{\alpha})E_2\right]+\left(1-\frac{A_2^*}{A_2}\right)\left[q w_2^{\alpha} E_2-(\gamma_2^{\alpha}+\mu^{\alpha})A_2\right]\\
&+\left(1-\frac{I_2^*}{I_2}\right)\left[(1-q) w_2^{\alpha} E_2-(\delta_2^{\alpha}+\eta_2^{\alpha}+\mu^{\alpha})I_2\right],\\
&\leq \left(1-\frac{S^*}{S}\right)\left(A_N^{\alpha}-(\beta_3^{\alpha} I_2+\beta_4^{\alpha} A_2)S-\mu^{\alpha} S\right)+\left(1-\frac{E_2^*}{E_2}\right)\left[(\beta_3^{\alpha} I_2+\beta_4^{\alpha} A_2)S-(w_2^{\alpha}+\mu^{\alpha})E_2\right]\\
&+\left(1-\frac{A_2^*}{A_2}\right)\left[q w_2^{\alpha} E_2-(\gamma_2^{\alpha}+\mu^{\alpha})A_2\right]+\left(1-\frac{I_2^*}{I_2}\right)\left[(1-q) w_2^{\alpha} E_2-(\delta_2^{\alpha}+\eta_2^{\alpha}+\mu^{\alpha})I_2\right],\\
&\leq \left(1-\frac{S^*}{S}\right)\left[(\beta_3^{\alpha} I_2^*+\beta_4^{\alpha} A_2^*)S^*+\mu^{\alpha} S^*-(\beta_3^{\alpha} I_2+\beta_4^{\alpha} A_2)S-\mu^{\alpha} S\right]+(\beta_3^{\alpha} I_2+\beta_4^{\alpha} A_2)S\\
&-(w_2^{\alpha}+\mu^{\alpha})E_2-\frac{E_2^*}{E_2}(\beta_3^{\alpha} I_2+\beta_4^{\alpha} A_2)S+(w_2^{\alpha}+\mu^{\alpha})E_2^*+q w_2^{\alpha} E_2-(\gamma_2^{\alpha}+\mu^{\alpha})A_2\\
&-\frac{A_2^*}{A_2} q w_2^{\alpha} E_2+(\gamma_2^{\alpha}+\mu^{\alpha})A_2^*+(1-q) w_2^{\alpha} E_2-(\delta_2^{\alpha}+\eta_2^{\alpha}+\mu^{\alpha})I_2-\frac{I_2^*}{I_2}(1-q) w_2^{\alpha} E_2\\
&+(\delta_2^{\alpha}+\eta_2^{\alpha}+\mu^{\alpha})I_2^*,
\end{aligned}
$$

After performing several rigorous and systematic mathematical arrangements, we arrive at

$$
\begin{aligned}
{}_0^c\mathbb{D}_t^\alpha V_2(t) &\leq \mu^\alpha S^* \left(2 - \frac{S}{S^*} - \frac{S^*}{S}\right) + (\beta_3^\alpha I_2^* + \beta_4^\alpha A_2^*) S^* \left(2 - \frac{S^*}{S} - \frac{E_2}{E_2^*}\right) \\
&+ (\beta_3^\alpha I_2 + \beta_4^\alpha A_2) S^* \left(1 - \frac{E_2^* S}{E_2 S^*}\right) + q w_2^\alpha E_2 - q w_2^\alpha A_2 \frac{E_2^*}{A_2^*} - q w_2^\alpha E_2 \frac{A_2^*}{A_2} + q w_2^\alpha E_2^* \\
&- \frac{(1-q) w_2^\alpha E_2^* I_2}{I_2^*} - (1-q) w_2^\alpha E_2 \frac{I_2^*}{I_2} + (1-q) w_2^\alpha E_2^* + (1-q) w_2^\alpha E_2, \\
&\leq \mu^\alpha S^* \left(2 - \frac{S}{S^*} - \frac{S^*}{S}\right) + (\beta_3^\alpha I_2^* + \beta_4^\alpha A_2^*) S^* \left(2 - \frac{S^*}{S} - \frac{E_2}{E_2^*}\right) \\
&+ (\beta_3^\alpha I_2 + \beta_4^\alpha A_2) S^* \left(1 - \frac{E_2^* S}{E_2 S^*}\right) + q w_2^\alpha E_2 \left(1 - \frac{A_2 E_2^*}{A_2^* E_2} - \frac{A_2^*}{A_2} + \frac{E_2^*}{E_2}\right) \\
&+ (1-q) w_2^\alpha E_2 \left(1 - \frac{I_2 E_2^*}{I_2^* E_2} - \frac{I_2^*}{I_2} + \frac{E_2^*}{E_2}\right)
\end{aligned}
$$

Considering that the arithmetic mean is always higher than or equal to the geometric mean. Consequently, ${}_0^c\mathbb{D}_t^\alpha V_2(t) \leq 0$. Whereas ${}^c\mathbb{D}_t^\alpha V_2(t) = 0$ if and only if $S = S^*$, $A_2 = A_2^*$, $E_2 = E_2^*$ and $I_2 = I_2^*$. Thus, the largest compact invariant set in Ω where ${}_0^c\mathbb{D}_t^\alpha V_2(t) = 0$ is the singleton set $\{P^*\}$. Hence, by the LaSalle's invariance principale [18, 25], P^* is globally asymptotically stable for $R_0 > 1$.

□

Theorem 5.3. *Concerning the endemic strain 1. If $R_0^1 > 1$, then the endemic equilibrium point P_1^* of model (3.3) is globally asymptotically stable in the region Ω.*

Proof. As the methodology is identical. The global stability of the first strain can be established using an approach analogous to the proof provided for the second strain. In which the expression of the candidate Lyapunov function is

$$\varpi(S(t)) + \varpi(E_1(t)) + \varpi(A_1(t)) + \varpi(I_1(t)). \tag{5.4}$$

□

6. Optimal Control Strategy

Optimal control theory provides a powerful framework for developing strategies that optimize system performance while adhering to its dynamic constraints. Through the context of epidemiological models, it provides a systematic approach to identifying interventions that minimize disease spread and related costs, such as healthcare expenses and distribution of resources. This work focuses on applying optimal control to achieve the two objectives of minimizing infection disease and decreasing treatment expenditures. Besides, providing a systematic approach to

managing disease dynamics effectively over time. System (3.3) is modified as follows after improving the previously mentioned:

$$\begin{cases} {}_0^c\mathbb{D}_t^\alpha S(t) = A_N^\alpha - (1-u_1(t))\beta_1^\alpha I_1(t)S(t) - (1-u_2(t))\beta_2^\alpha A_1(t)S(t) \\ \qquad\qquad -(1-u_3(t))\beta_3^\alpha I_2(t)S(t) - (1-u_4(t))\beta_4^\alpha A_2(t)S(t) - \mu^\alpha S(t), \\ {}_0^c\mathbb{D}_t^\alpha E_1(t) = ((1-u_1(t))\beta_1^\alpha I_1(t) + (1-u_2(t))\beta_2^\alpha A_1(t))S(t) - (w_1^\alpha + \mu^\alpha)E_1(t), \\ {}_0^c\mathbb{D}_t^\alpha E_2(t) = ((1-u_3(t))\beta_3^\alpha I_2(t) + (1-u_4(t))\beta_4^\alpha A_2(t))S(t) - (w_2^\alpha + \mu^\alpha)E_2(t), \\ {}_0^c\mathbb{D}_t^\alpha A_1(t) = pw_1^\alpha E_1(t) - (\gamma_1^\alpha + \mu^\alpha)A_1(t), \\ {}_0^c\mathbb{D}_t^\alpha A_2(t) = qw_2^\alpha E_2(t) - (\gamma_2^\alpha + \mu^\alpha)A_2(t), \\ {}_0^c\mathbb{D}_t^\alpha I_1(t) = (1-p)w_1^\alpha E_1(t) - (\delta_1^\alpha + \eta_1^\alpha + \mu^\alpha)I_1(t), \\ {}_0^c\mathbb{D}_t^\alpha I_2(t) = (1-q)w_2^\alpha E_2(t) - (\delta_2^\alpha + \eta_2^\alpha + \mu^\alpha)I_2(t), \\ {}_0^c\mathbb{D}_t^\alpha R(t) = \gamma_1^\alpha A_1(t) + \gamma_2^\alpha A_2(t) + \delta_1^\alpha I_1(t) + \delta_2^\alpha I_2(t) - \mu^\alpha R(t). \end{cases} \tag{6.1}$$

With the initial conditions (3.2).

The admissible controls set U is provided by

$$U = \{(u_1, u_2, u_3, u_4) \mid u_i \text{ is Lebesgue measurable on } [0,1],\ i = 1,2,3,4\}. \tag{6.2}$$

When T indicates the control period. Consequently, the objective function is defined as

$$J(u_1(t), u_2(t), u_3(t), u_4(t)) = \int_0^T [K_1 A_1^2(t) + K_2 I_1^2(t) + K_3 A_2^2(t) + K_4 I_2^2(t) + K_5 u_1^2(t) + K_6 u_2^2(t) + K_7 u_3^2(t) + K_8 u_4^2(t)]\, dt. \tag{6.3}$$

K_1, K_2, K_3 *and* K_4 indicate the weight cost of control population contact.

K_5, K_6, K_7 *and* K_8 indicate the weight cost of establishing treatment controls.

Therefore, the objective is to determine $u^*(t) = (u_1^*, u_2^*, u_3^*, u_4^*)$ such that

$$J(u_1^*, u_2^*, u_3^*, u_4^*) = min_{(u_1,u_2,u_3,u_4)\in U} J(u_1, u_2, u_3, u_4) \tag{6.4}$$

All variables within the model are continuous, ensuring that the solution to the control system remains bounded. Convexity of the objective function with respect to the control variable $u(t)$ is also verified. Therefore, the optimal control problem 6.1 has a solution, according to the Filippov-Cesari theorem (see [29]).

Theorem 6.1. *Concerning the control problems (6.1) and (6.4), we consider* $(S_o^*, E_{1o}^*, E_{2o}^*, A_{1o}^*, A_{2o}^*, I_{1o}^*, I_{2o}^*, R_o^*)$ *as an optimal solution associated with the optimal control variables* $u^*(t)$ *on* $[0, T]$*, so that the adjoints variables* $\lambda_i(.)$*,* $i = 1, ..., 8$ *are presented and verifying*

$$
\begin{aligned}
&{}_0^c\mathbb{D}_t^\alpha \lambda_1(t) = -\frac{\partial H}{\partial S} \ ; \ {}_0^c\mathbb{D}_t^\alpha \lambda_2(t) = -\frac{\partial H}{\partial E_1} \ ; \ {}_0^c\mathbb{D}_t^\alpha \lambda_3(t) = -\frac{\partial H}{\partial E_2} \ ; \ {}_0^c\mathbb{D}_t^\alpha \lambda_4(t) = -\frac{\partial H}{\partial A_1} \ ; \\
&{}_0^c\mathbb{D}_t^\alpha \lambda_5(t) = -\frac{\partial H}{\partial A_2} \ ; \ {}_0^c\mathbb{D}_t^\alpha \lambda_6(t) = -\frac{\partial H}{\partial I_1} \ ; \ {}_0^c\mathbb{D}_t^\alpha \lambda_7(t) = -\frac{\partial H}{\partial I_2} \ ; \ {}_0^c\mathbb{D}_t^\alpha \lambda_8(t) = -\frac{\partial H}{\partial R}.
\end{aligned} \tag{6.5}
$$

With the following transversality conditions $\lambda_i(T) = 0$ *for* $i = 1, ..., 8$.

Furthermore, the control functions are given by

$$
\begin{cases}
u_1^* = max\{0, min\{\dfrac{(\lambda_2 - \lambda_1)\beta_1^\alpha I_{1o}^* S_o^*}{2K_5}, 1\}\}, \\
u_2^* = max\{0, min\{\dfrac{(\lambda_2 - \lambda_1)\beta_2^\alpha A_{1o}^* S_o^*}{2K_6}, 1\}\}, \\
u_3^* = max\{0, min\{\dfrac{(\lambda_3 - \lambda_1)\beta_3^\alpha I_{2o}^* S_o^*}{2K_7}, 1\}\}, \\
u_4^* = max\{0, min\{\dfrac{(\lambda_3 - \lambda_1)\beta_4^\alpha A_{2o}^* S_o^*}{2K_8}, 1\}\}.
\end{cases}
$$

Proof. Following Pontryagin's maximum principle [27], a Hamiltonian function is formulated based on the system's state equations (6.1) and the objective function (6). This approach makes it possible to transform the optimal control problem into an equivalent Hamiltonian function minimization problem.
Hence, the Hamiltonian function H is described by

$$
\begin{aligned}
&H(S, E_1, E_2, A_1, A_2, I_1, I_2, R, \lambda, u(t))\\
&= K_1 A_1^2(t) + K_2 I_1^2(t) + K_3 A_2^2(t) + K_4 I_2^2(t) + K_5 u_1^2(t) + K_6 u_2^2(t) + K_7 u_3^2(t) + K_8 u_4^2(t)\\
&+ \lambda_1[A_N^\alpha - (1-u_1(t))\beta_1^\alpha I_1(t)S(t) - (1-u_2(t))\beta_2^\alpha A_1(t)S(t) - (1-u_3(t))\beta_3^\alpha I_2(t)S(t)\\
&- (1-u_4(t))\beta_4^\alpha A_2(t)S(t) - \mu^\alpha S(t)]\\
&+ \lambda_2[((1-u_1(t))\beta_1^\alpha I_1(t) + (1-u_2(t))\beta_2^\alpha A_1(t))S(t) - (w_1^\alpha + \mu^\alpha)E_1(t)]\\
&+ \lambda_3[((1-u_3(t))\beta_3^\alpha I_2(t) + (1-u_4(t))\beta_4^\alpha A_2(t))S(t) - (w_2^\alpha + \mu^\alpha)E_2(t)]\\
&+ \lambda_4[pw_1^\alpha E_1(t) - (\gamma_1^\alpha + \mu^\alpha)A_1(t)]\\
&+ \lambda_5[qw_2^\alpha E_2(t) - (\gamma_2^\alpha + \mu^\alpha)A_2(t)]\\
&+ \lambda_6[(1-p)w_1^\alpha E_1(t) - (\delta_1^\alpha + \eta_1^\alpha + \mu^\alpha)I_1(t)]\\
&+ \lambda_7[(1-q)w_2^\alpha E_2(t) - (\delta_2^\alpha + \eta_2^\alpha + \mu^\alpha)I_2(t)]\\
&+ \lambda_8[\gamma_1^\alpha A_1(t) + \gamma_2^\alpha A_2(t) + \delta_1^\alpha I_1(t) + \delta_2^\alpha I_2(t) - \mu^\alpha R(t)].
\end{aligned}
$$

Where $\lambda_i(t)$ for $i = 1, ..., 8$, are the adjoint variables with a nontrivial absolutely continuous mapping $\lambda : [0, t^*] \longrightarrow \mathbb{R}$, such that

$$
\begin{cases}
{}_0^c\mathbb{D}_t^\alpha \lambda_1(t) = -\frac{\partial H}{\partial S} = (\lambda_1 - \lambda_2)[(1-u_1(t))\beta_1^\alpha I_1(t) + (1-u_2(t))\beta_2^\alpha A_1(t)]\\
\qquad\qquad +(\lambda_1 - \lambda_3)[(1-u_3(t))\beta_3^\alpha I_2(t) + (1-u_4(t))\beta_4^\alpha A_2(t)] + \lambda_1\mu^\alpha,\\
{}_0^c\mathbb{D}_t^\alpha \lambda_2(t) = -\frac{\partial H}{\partial E_1} = \lambda_2(w_1^\alpha + \mu^\alpha) - \lambda_4 pw_1^\alpha - \lambda_6(1-p)w_1^\alpha,\\
{}_0^c\mathbb{D}_t^\alpha \lambda_3(t) = -\frac{\partial H}{\partial E_2} = \lambda_3(w_2^\alpha + \mu^\alpha) - \lambda_5 qw_2^\alpha - \lambda_7(1-q)w_2^\alpha,\\
{}_0^c\mathbb{D}_t^\alpha \lambda_4(t) = -\frac{\partial H}{\partial A_1} = -2k_1A_1(t) + (\lambda_1 - \lambda_2)[(1-u_2(t))\beta_2^\alpha S(t)] + \lambda_4(\gamma_1^\alpha + \mu^\alpha) - \lambda_8\gamma_1^\alpha,\\
{}_0^c\mathbb{D}_t^\alpha \lambda_5(t) = -\frac{\partial H}{\partial A_2} = -2k_3A_2(t) + (\lambda_1 - \lambda_3)[(1-u_4(t))\beta_4^\alpha S(t)] + \lambda_5(\gamma_2^\alpha + \mu^\alpha) - \lambda_8\gamma_2^\alpha,\\
{}_0^c\mathbb{D}_t^\alpha \lambda_6(t) = -\frac{\partial H}{\partial I_1} = -2k_2I_1(t) + (\lambda_1 - \lambda_2)[(1-u_1(t))\beta_1^\alpha S(t)] + \lambda_6(\delta_1^\alpha + \eta_1^\alpha + \mu^\alpha) - \lambda_8\delta_1^\alpha,\\
{}_0^c\mathbb{D}_t^\alpha \lambda_7(t) = -\frac{\partial H}{\partial I_2} = -2k_4I_2(t) + (\lambda_1 - \lambda_3)[(1-u_3(t))\beta_3^\alpha S(t)] + \lambda_7(\delta_2^\alpha + \eta_2^\alpha + \mu^\alpha) - \lambda_8\delta_2^\alpha,\\
{}_0^c\mathbb{D}_t^\alpha \lambda_8(t) = -\frac{\partial H}{\partial R} = \lambda_8\mu^\alpha.
\end{cases}
$$

As for the control equations, its expressions are satisfying:

$$
\begin{aligned}
{}_0^c\mathbb{D}_t^\alpha S(t) = \frac{\partial H}{\partial \lambda_1}\ ;\ {}_0^c\mathbb{D}_t^\alpha E_1(t) = \frac{\partial H}{\partial \lambda_2}\ ;\ {}_0^c\mathbb{D}_t^\alpha E_2(t) = \frac{\partial H}{\partial \lambda_3}\ ;\ {}_0^c\mathbb{D}_t^\alpha A_1(t) = \frac{\partial H}{\partial \lambda_4}\ ;\ {}_0^c\mathbb{D}_t^\alpha A_2(t) = \frac{\partial H}{\partial \lambda_5}\\
;\ {}_0^c\mathbb{D}_t^\alpha I_1(t) = \frac{\partial H}{\partial \lambda_6}\ ;\ {}_0^c\mathbb{D}_t^\alpha I_2(t) = \frac{\partial H}{\partial \lambda_7}\ ;\ {}_0^c\mathbb{D}_t^\alpha R(t) = \frac{\partial H}{\partial \lambda_8} \qquad (6.6)
\end{aligned}
$$

Additionally, we take into consideration the optimality criteria for presenting the description of the optimal control u^* by solving the equations

$$\frac{\partial H}{\partial u_1}|_{u_1=u_1^*}=0\ ;\ \frac{\partial H}{\partial u_2}|_{u_2=u_2^*}=0\ ;\ \frac{\partial H}{\partial u_3}|_{u_3=u_3^*}=0\ ;\ \frac{\partial H}{\partial u_4}|_{u_4=u_4^*}=0 \tag{6.7}$$

Hence,

$$u_1^*=\frac{(\lambda_2-\lambda_1)\beta_1^\alpha I_{1o}^* S_o^*}{2K_5}\ ;\ u_2^*=\frac{(\lambda_2-\lambda_1)\beta_2^\alpha A_{1o}^* S_o^*}{2K_6}\ ;\ u_3^*=\frac{(\lambda_3-\lambda_1)\beta_3^\alpha I_{2o}^* S_o^*}{2K_7}\ ;$$
$$u_4^*=\frac{(\lambda_3-\lambda_1)\beta_4^\alpha A_{2o}^* S_o^*}{2K_8} \tag{6.8}$$

Ultimately, the optimal control solution u^*, can be written as

$$u_1^*=\begin{cases}0, & \frac{(\lambda_2-\lambda_1)\beta_1^\alpha I_{1o}^* S_o^*}{2K_5}\le 0,\\ \frac{(\lambda_2-\lambda_1)\beta_1^\alpha I_{1o}^* S_o^*}{2K_5}, & 0<\frac{(\lambda_2-\lambda_1)\beta_1^\alpha I_{1o}^* S_o^*}{2K_5}<1,\\ 1, & \frac{(\lambda_2-\lambda_1)\beta_1^\alpha I_{1o}^* S_o^*}{2K_5}\ge 1.\end{cases}$$

$$u_2^*=\begin{cases}0, & \frac{(\lambda_2-\lambda_1)\beta_2^\alpha A_{1o}^* S_o^*}{2K_6}\le 0,\\ \frac{(\lambda_2-\lambda_1)\beta_2^\alpha A_{1o}^* S_o^*}{2K_6}, & 0<\frac{(\lambda_2-\lambda_1)\beta_2^\alpha A_{1o}^* S_o^*}{2K_6}<1,\\ 1, & \frac{(\lambda_2-\lambda_1)\beta_2^\alpha A_{1o}^* S_o^*}{2K_6}\ge 1.\end{cases}$$

$$u_3^*=\begin{cases}0, & \frac{(\lambda_3-\lambda_1)\beta_3^\alpha I_{2o}^* S_o^*}{2K_7}\le 0,\\ \frac{(\lambda_3-\lambda_1)\beta_3^\alpha I_{2o}^* S_o^*}{2K_7}, & 0<\frac{(\lambda_3-\lambda_1)\beta_3^\alpha I_{2o}^* S_o^*}{2K_7}<1,\\ 1, & \frac{(\lambda_3-\lambda_1)\beta_3^\alpha I_{2o}^* S_o^*}{2K_7}\ge 1.\end{cases}$$

$$u_4^*=\begin{cases}0, & \frac{(\lambda_3-\lambda_1)\beta_4^\alpha A_{2o}^* S_o^*}{2K_8}\le 0,\\ \frac{(\lambda_3-\lambda_1)\beta_4^\alpha A_{2o}^* S_o^*}{2K_8}, & 0<\frac{(\lambda_3-\lambda_1)\beta_4^\alpha A_{2o}^* S_o^*}{2K_8}<1,\\ 1, & \frac{(\lambda_3-\lambda_1)\beta_4^\alpha A_{2o}^* S_o^*}{2K_8}\ge 1.\end{cases}$$

considering the bounded of the control variable set U. The demonstration is complete.

□

7. Numerical Simulations

7.1. **Memory Effects on Epidemic Dynamics.** This subsection aims to illustrate the impact of various fractional order α values by conducting a numerical simulation of model (3.3). By utilizing memory effects, these fractional derivatives allow the model to consider how previous states have influenced the dynamics at

present.

Now, we show the simulation results of the Disease-Free Equilibrium (DFE) along with the following parameter values: $A_N^\alpha = 10$; $\beta_1^\alpha = 0.055$; $\beta_2^\alpha = 0.037$; $\beta_3^\alpha = 0.075$; $\beta_4^\alpha = 0.04$; $\gamma_1^\alpha = 0.37$; $\gamma_2^\alpha = 0.47$; $\delta_1^\alpha = 0.65$; $\delta_2^\alpha = 0.75$; $\eta_1 = 0.025$; $\eta_2 = 0.035$; $\omega_1 = 0.3$; $\omega_2 = 0.4$; $p = 0.5$; $q = 0.3$; $\mu^\alpha = 0.2$. In addition, the presumably initial population given as $(10, 8, 6, 4, 3, 5, 7, 0)$.

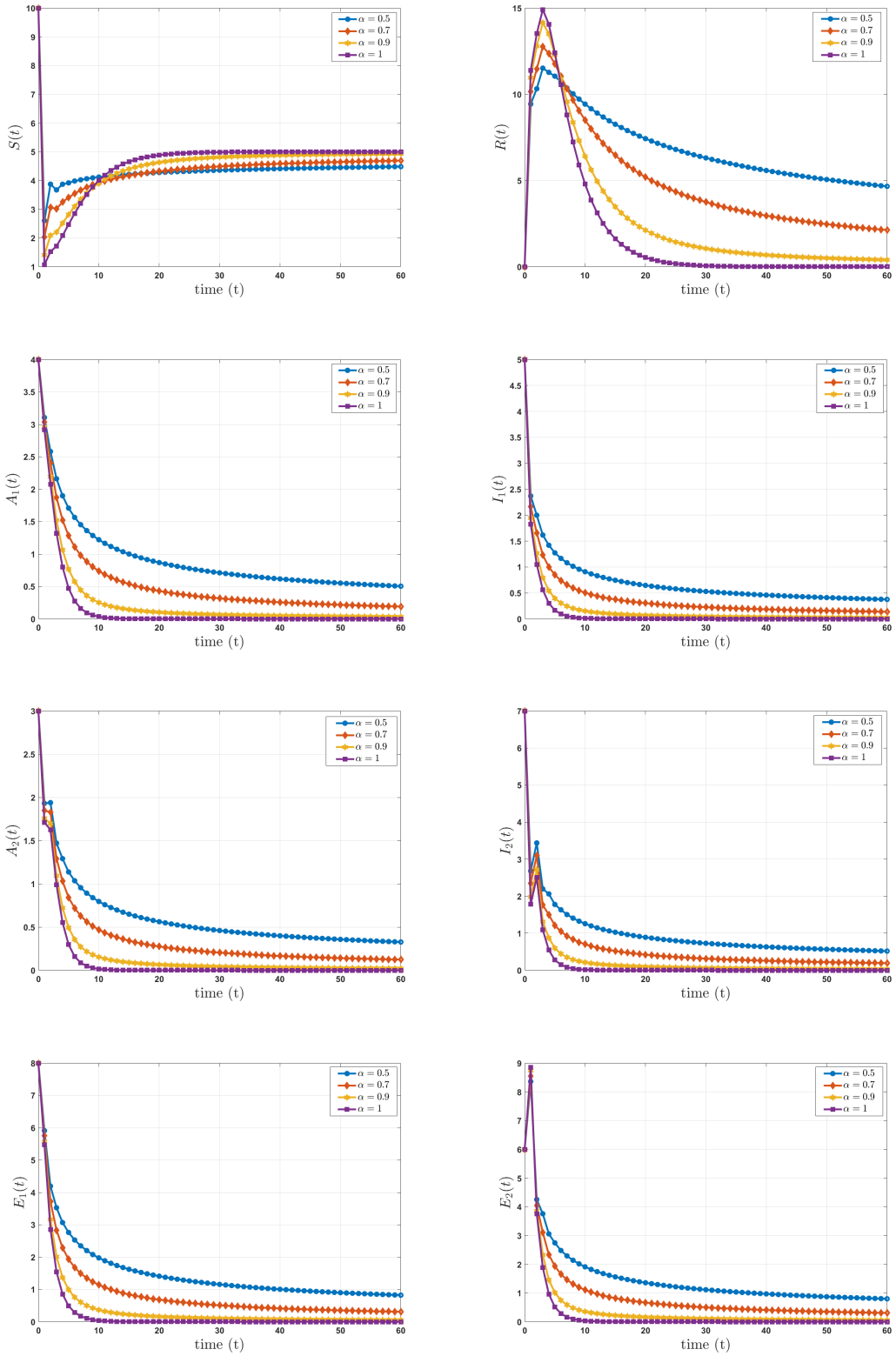


FIGURE 3. The dynamic behavior of system (3.3) presenting the stability at the disease-free equilibrium (DFE), under the condition $R_0 < 1$, along with different values of the fractional order α.

Currently, we will explore the simulation results for the Endemic Equilibrium (EE), focusing on the dynamics of each strain. This will illustrate how the interactions between the two strains influence the system's evolution and the convergence to equilibrium points under varying fractional orders derivative.

Concerning figure 4 the parameters given as ($A_N^\alpha = 7$; $\beta_1^\alpha = 0.65$; $\beta_2^\alpha = 0.25$; $\beta_3^\alpha = 0.035$; $\beta_4^\alpha = 0.02$; $\gamma_1^\alpha = 0.67$; $\gamma_2^\alpha = 0.47$; $\delta_1^\alpha = 0.65$; $\delta_2^\alpha = 0.75$; $\eta_1 = 0.025$; $\eta_2 = 0.035$; $\omega_1 = 0.06$; $\omega_2 = 0.04$; $p = 0.7$; $q = 0.5$; $\mu^\alpha = 0.2$). Such that, $R_0^1 = 1.62$ and $R_0^2 = 0.19$. Meanwhile, with regard to figure 5, the parameters given as ($A_N^\alpha = 7$; $\beta_1^\alpha = 0.07$; $\beta_2^\alpha = 0.035$; $\beta_3^\alpha = 0.025$; $\beta_4^\alpha = 0.2$; $\gamma_1^\alpha = 0.3$; $\gamma_2^\alpha = 0.57$; $\delta_1^\alpha = 0.165$; $\delta_2^\alpha = 0.175$; $\eta_1 = 0.25$; $\eta_2 = 0.35$; $\omega_1 = 0.06$; $\omega_2 = 0.14$; $p = 0.6$; $q = 0.7$; $\mu^\alpha = 0.2$). As a result, $R_0^1 = 0.70$ and $R_0^2 = 2.76$.

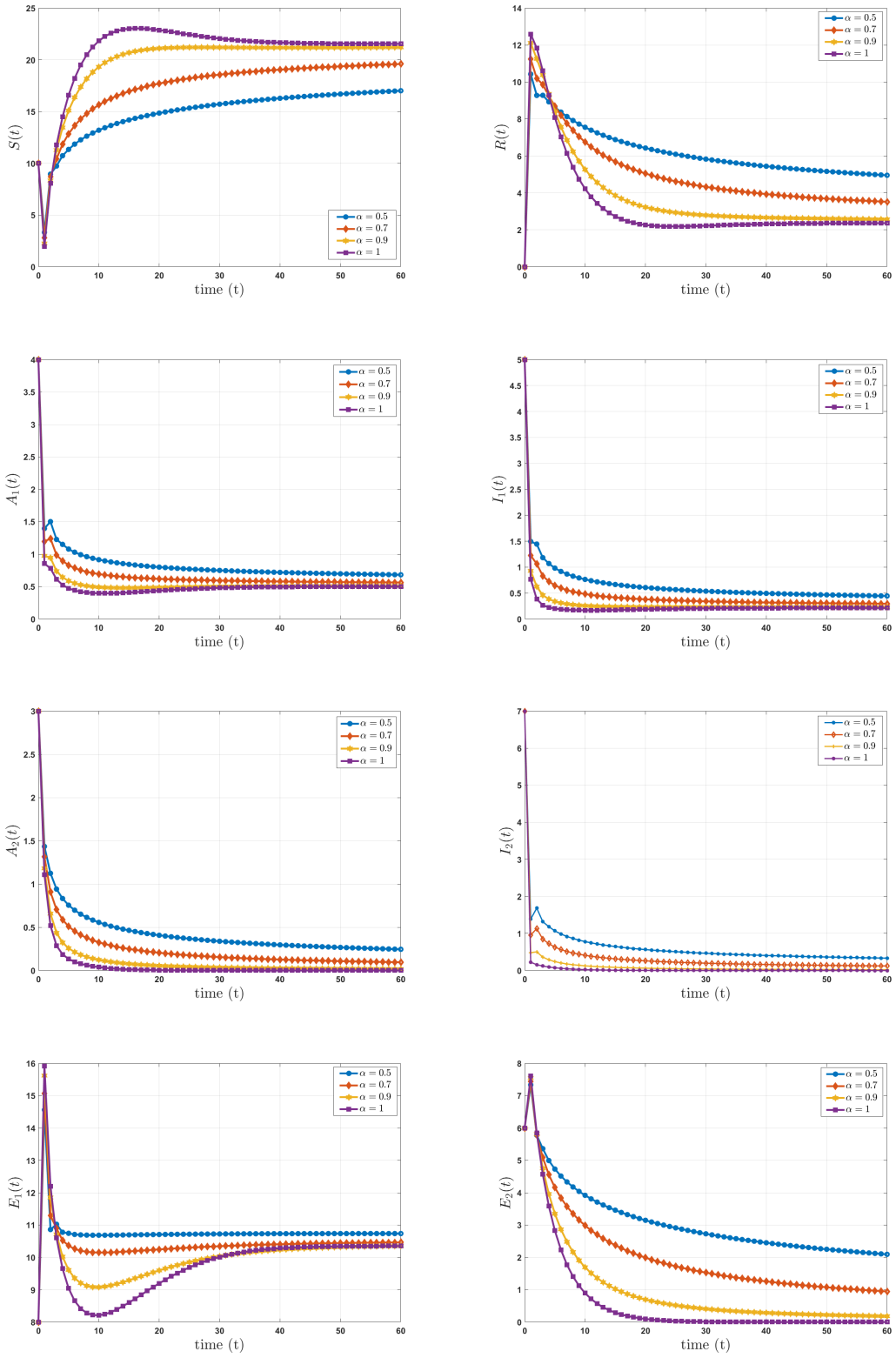


FIGURE 4. The dynamic behavior of the system (3.3) presenting the stability at the first endemic equilibrium (4.13), under the condition $R_0^1 > 1$, along with different values of the fractional order α.

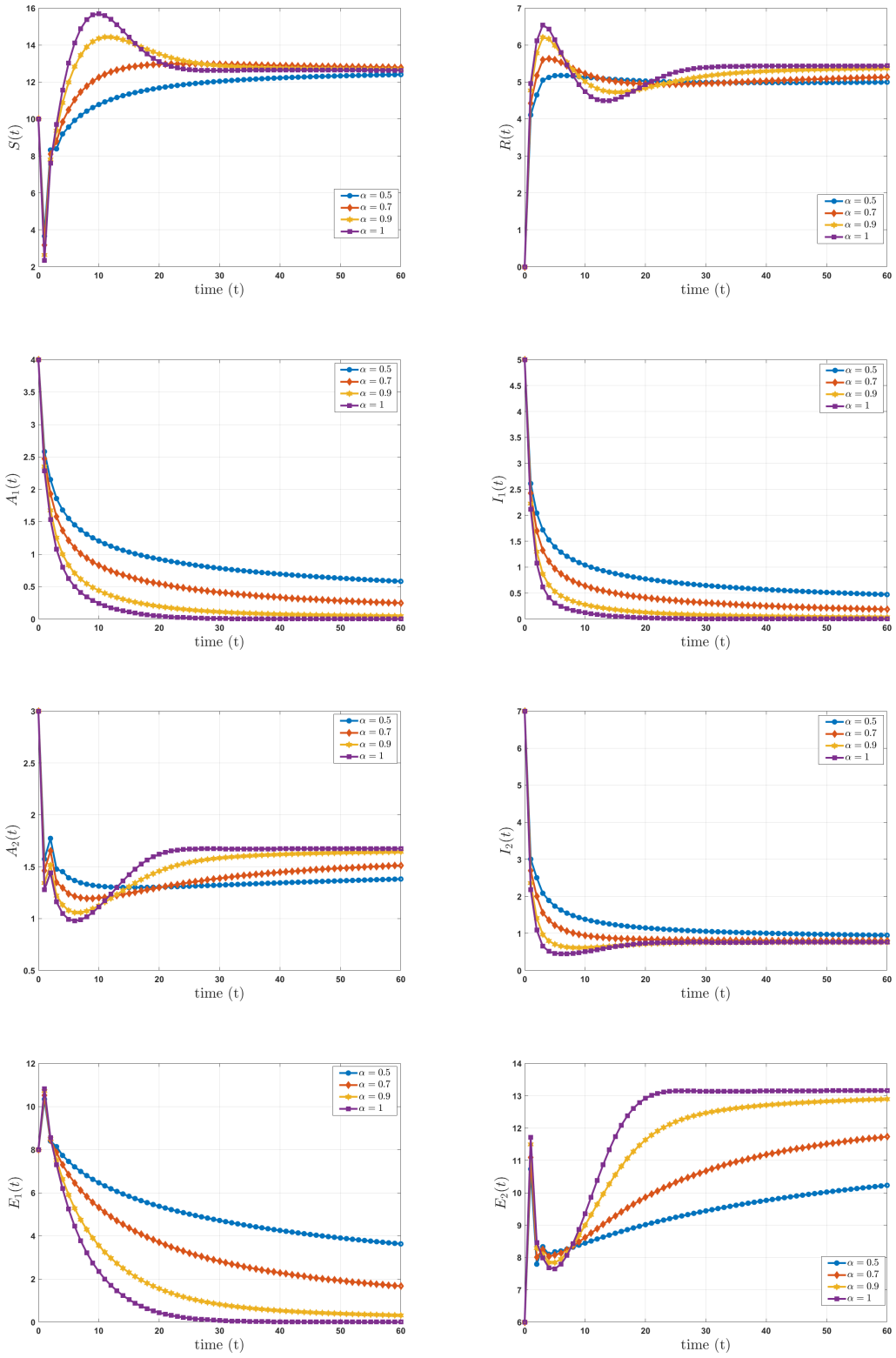


FIGURE 5. The dynamic behavior of system (3.3) presenting the stability at the second endemic equilibrium (4.14), under the condition $R_0^2 > 1$, along with different values of the fractional order α.

The information found validates the theoretical study by emphasizing how well it is able to represent the long-term persistence of the disease under specific conditions. Furthermore, the influence of different fractional order derivatives (α) is noticeable; slower system responses are caused by lower values of α, suggesting a more gradual stabilization towards equilibrium. In contrast, faster convergence is caused by higher values of α, indicating the crucial role that memory effects play in the model's behavior.

7.2. **Numerical Simulation Using Control Measures.** The numerical results of the model (6.1) will be presented following the implementation of control measures. The initial conditions and parameter settings are identical to those in Sect. 7.1 and are applied this time.

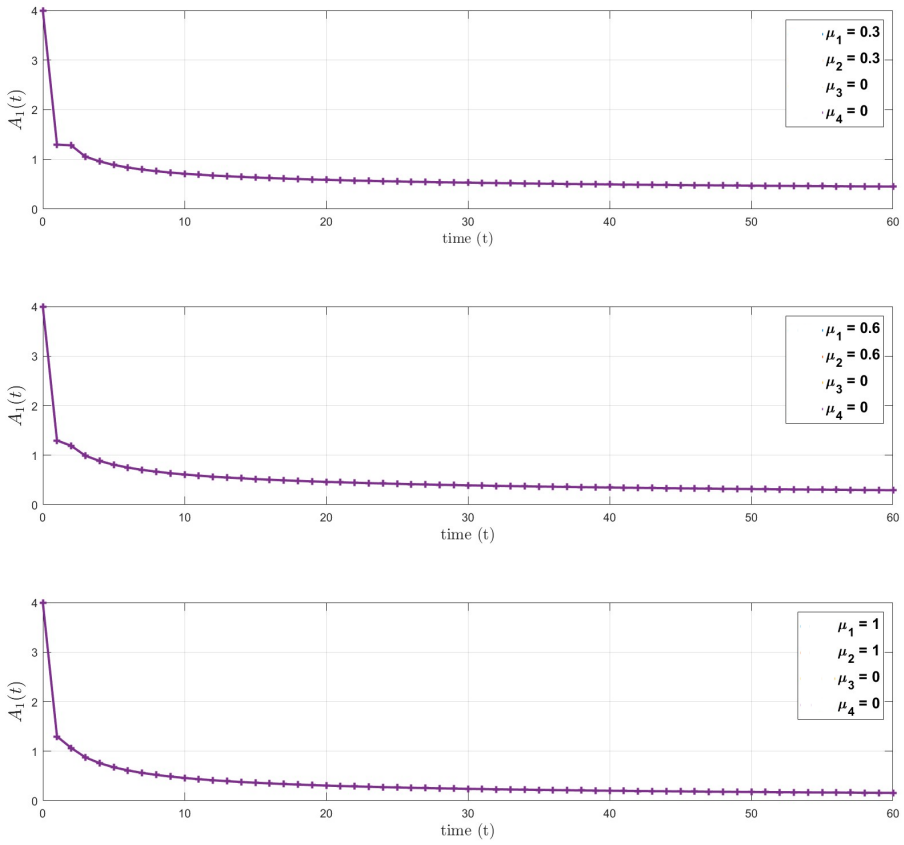


FIGURE 6. Number of Asymptomatic Cases (A_1) Under the Proposed Optimal Control Strategy for Different Values.

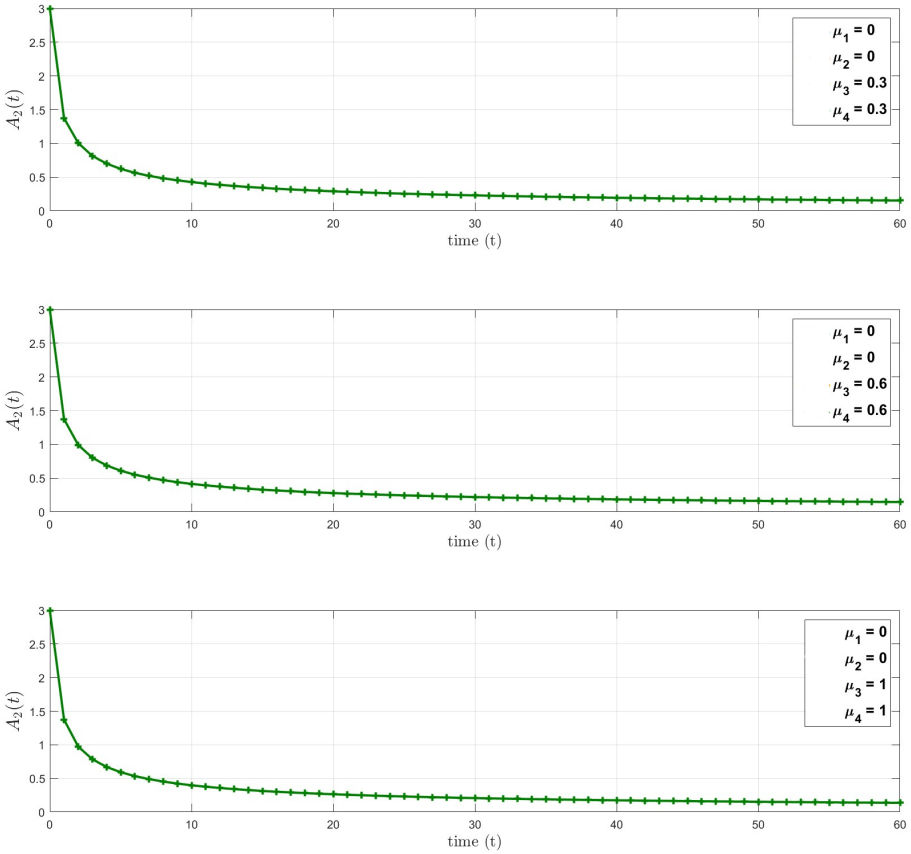


FIGURE 7. Number of Asymptomatic Cases (A_2) Under the Proposed Optimal Control Strategy for Different Values.

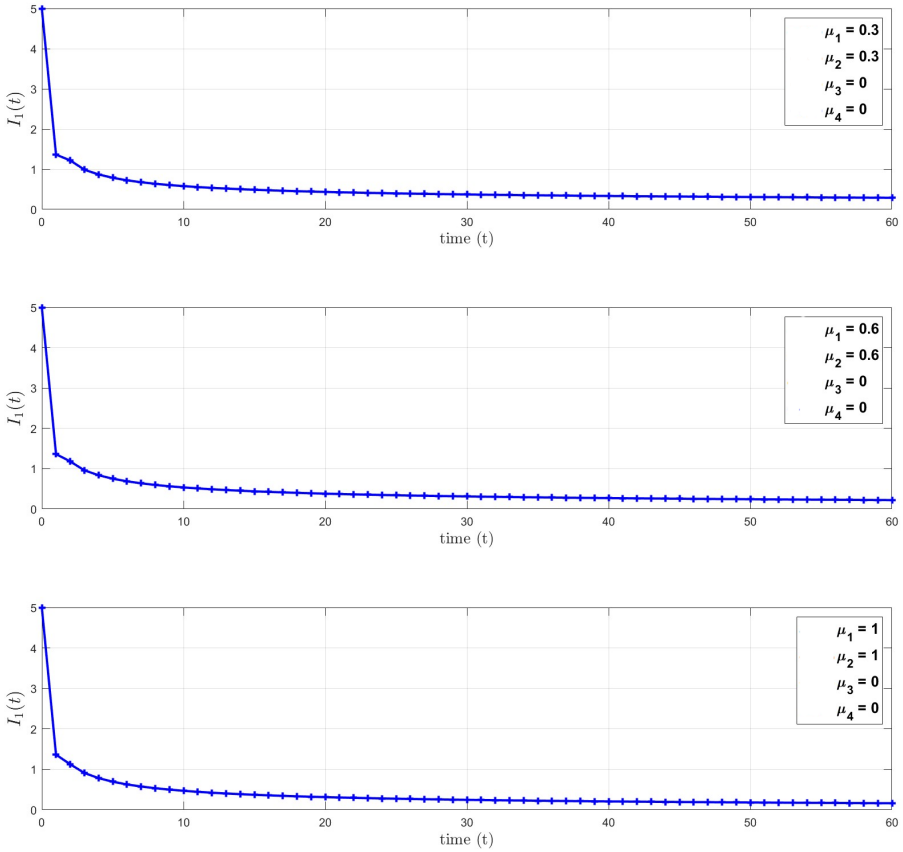


FIGURE 8. Number of Infected Cases (I_1) Under the Proposed Optimal Control Strategy for Different Values.

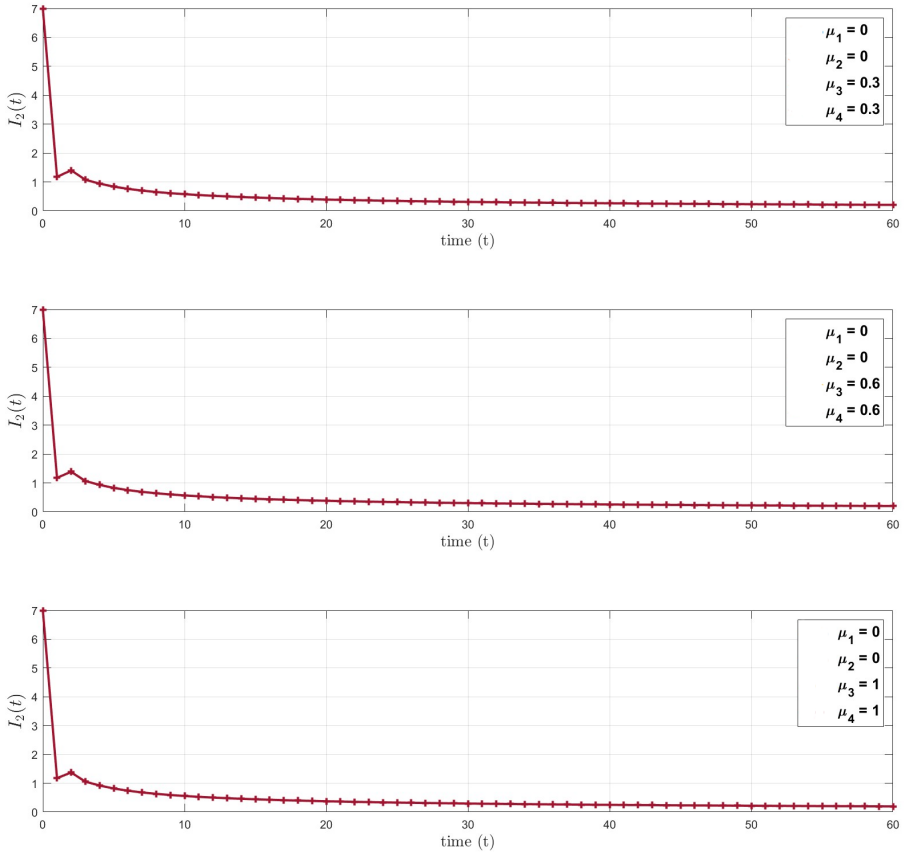


FIGURE 9. Number of Infected Cases (I_2) Under the Proposed Optimal Control Strategy for Different Values.

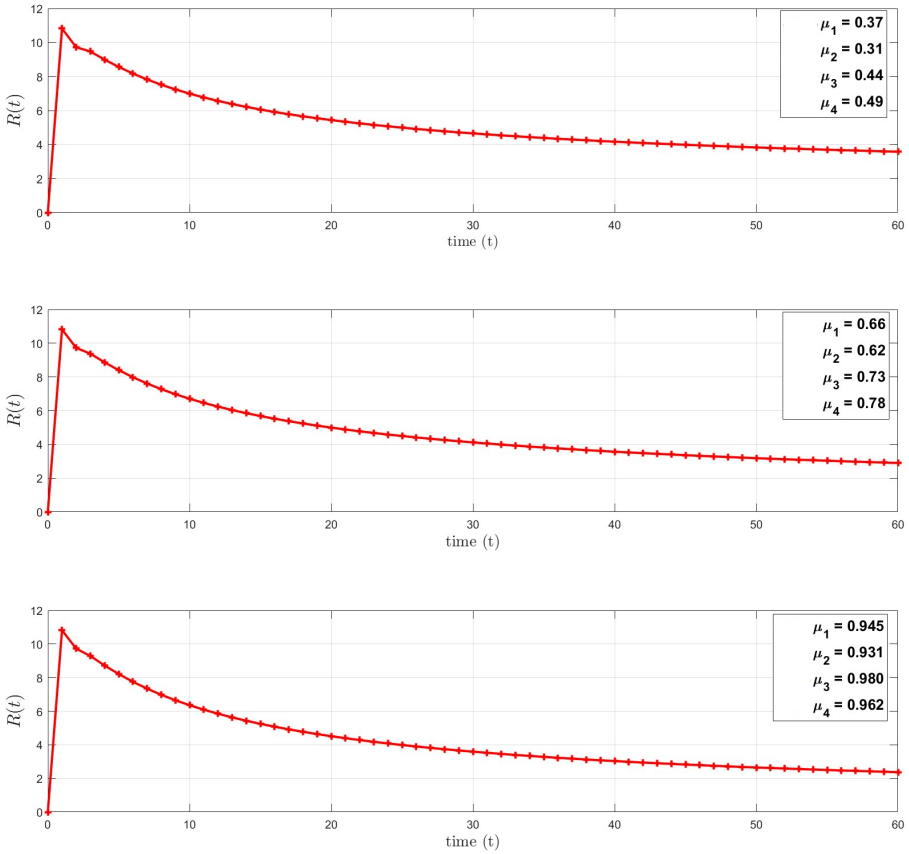


FIGURE 10. Number of Recovered Cases (R) Under the Proposed Optimal Control Strategy for Different Values.

Figures 6, 7, 8, 9 and 10 clearly illustrate the profound impact of implementing the suggested optimal control strategy on the dynamics of the epidemic. The results demonstrate a substantial reduction in the number of infections in all compartments as the treatment control parameters are intensified. Additionally, the control measures help the recovered population by reducing infections and accelerating recovery rates. This dual effect is essential in mitigating the overall burden of the epidemic, as fewer active cases result in reduced strain on healthcare resources and a faster return to normalcy for the population.
The findings confirm the effectiveness of the proposed optimal control strategy, emphasizing its practical value for cost-effective epidemic management, especially with limited resources or in cases requiring immediate interventions.

8. Conclusion

This study investigates the effective control approach and qualitative behavior of a fractional SEAIR model incorporating two viral strains. The outcomes of the simulation show the impact of fractional calculus on how the two virus strains respond, showing that variations in the fractional order affect both the rate of convergence and the stability of the system. We have also analyzed and identified

the optimal treatment strategy to minimize the number of infected individuals and the costs associated with the treatment of recovered individuals. These results emphasize the crucial role of treatment control as an effective and practical approach for mitigating the spread of epidemics and managing their long-term impact.

Conflicts Of Interest

The authors declare that they have no conflicts of interest.

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