

RESEARCH ARTICLE

Analysis and Optimal Control of a Fractional Order SEIR Epidemic Model With General Incidence and Vaccination

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ABSTRACT In this article, we present an analysis and optimal control investigation of a fractional order SEIR epidemic model with General Incidence and Vaccination. We utilize fractional calculus to account for memory effects and non-local interactions in the disease transmission process, enhancing the model's ability to capture real-world complexities. The utilization of fractional derivatives plays a crucial role in accounting for long-term memory in the system, allowing us to better understand the disease dynamics. Our analysis focuses on investigating the existence, uniqueness, and stability of equilibrium points while considering the impact of vaccination on the disease dynamics. Additionally, we develop an optimal control strategy to minimize the number of infected individuals over a given time horizon by optimizing the vaccination rate. Numerical simulations are performed to validate the theoretical results and demonstrate the effectiveness of the proposed optimal control strategy in mitigating the spread of the epidemic. The findings of this study contribute to a better understanding of the dynamics of fractional order SEIR epidemic models and provide insights into the design of efficient control measures for infectious diseases. The ability to accurately capture memory effects and non-local interactions through fractional derivatives opens up new possibilities for developing more robust and effective intervention strategies in public health settings.

INDEX TERMS Fractional optimal control, fractional SEIR epidemic model, global dynamics, general incidence function, Lyapunov functional, numerical simulation.


I. INTRODUCTION

Mathematical modeling and analysis of epidemic dynamics are pivotal in comprehending and managing the spread of infectious diseases. See [1] and [2]. Over the years, the application of fractional calculus to epidemiological models has garnered significant interest, enabling the study of complex systems with long-range dependence and memory effects observed in real-world epidemic processes. This approach provides a nuanced understanding of the interplay between various factors in disease transmission, and researchers have found it useful in numerous physical scenarios and successful applications of the Caputo derivative.

Traditionally, epidemic modeling employed commonly used bilinear and standard incidence rates, βSI (with β as the transmission rate) [2] and $\beta SI/N$ (where N is the total

population size) [3], respectively, to describe the rate at which new cases of a disease occur in a population. However, it was recognized that these linear incidence rates might not accurately portray the progression of infectious diseases in human populations. As a remedy, researchers explored nonlinear incidence rates, such as the saturated incidence rate, $\beta SI/(1 + \alpha I)$, introduced by Capasso and Serio [4], which accounted for inhibitory effects like crowding or behavioral changes in susceptible individuals. This development and subsequent work by other authors incorporating various types of nonlinear incidence rates, paved the way for a more accurate representation of epidemic dynamics [5], [6], [7], [8], [9].

In the context of fractional calculus in epidemic modeling, fractional differential operators, including Riemann–Liouville, Hadamard, Caputo, Caputo–Fabrizio, Katugampola, and Atangana–Baleanu, have been developed and applied to study epidemic scenarios across a range of infectious diseases [10], [11], [12]. Among these operators, the

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Caputo operator stands out due to its effectiveness in capturing the characteristics of disease spread.

In light of the various advancements in epidemic modeling, our research aims to analyze the equilibrium points of the fractional order SEIR model and explore their existence, uniqueness, and stability properties. Additionally, we investigate the impact of vaccination on disease dynamics, considering key epidemiological parameters such as the transmission rate, recovery rate, and vaccination rate. By examining the relationships between these factors, we can gain valuable insights into the effectiveness of control strategies in curbing the spread of infectious diseases.

Furthermore, our research includes the development of an optimal control strategy to minimize the number of infected individuals over a given time horizon by optimizing the vaccination rate. This approach allows us to determine the most efficient allocation of resources for vaccination while considering the constraints and objectives of disease control.

It is worth noting that the applications of the Caputo derivative in epidemic modeling have expanded significantly, and numerous physical scenarios have been successfully addressed using this mathematical framework [13], [14], [15]. Additionally, dynamic and nonstandard computational examinations of epidemic models, such as the heroin epidemic model conducted by Raza et al., have contributed further insights into epidemic dynamics [16]. These developments emphasize the ever-growing importance of mathematical tools, especially fractional calculus, in enhancing our understanding and management of infectious diseases.

In our research, our primary objective is to analyze the equilibrium points of the fractional order SEIR model and explore their existence, uniqueness, and stability properties. Additionally, we aim to investigate the impact of vaccination on disease dynamics, considering epidemiological parameters such as the transmission rate, recovery rate, and vaccination rate. By examining the relationships between these factors, we can gain insights into the effectiveness of control strategies in curbing the spread of infectious diseases. We also develop an optimal control strategy to minimize the number of infected individuals over a given time horizon by optimizing the vaccination rate. The optimal control approach allows us to determine the optimal allocation of resources for vaccination, taking into account the constraints and objectives of disease control.

Motivated by the aforementioned studies, we consider a fractional order SEIR epidemic model with general incidence and vaccination. Hence, our study will be carried out on the following epidemic model:

$$\begin{cases} D_c^\alpha S(t) &= b - g(S(t), I(t)) - (\mu + \nu)S(t), \\ D_c^\alpha E(t) &= g(S(t), I(t)) - (\epsilon + \sigma + \mu + \delta)E(t), \\ D_c^\alpha I(t) &= \sigma E(t) - (\mu + \gamma + d)I(t), \\ D_c^\alpha R(t) &= \nu S(t) + \gamma I(t) + \epsilon E(t) - \mu R(t), \end{cases} \quad (I.1)$$

where

$-S(t)$ stand for the number of susceptible people at time t .

$-E(t)$ stand for the number of exposed people at time t .

$-I(t)$ stand for the number of infected people at time t .

$-R(t)$ stand for the number of recovered people at time t .

$-b$ denotes the recruitment rate, μ represents the death rate and ν is the rate of vaccination, σ indicate the transmission rate from the exposed people to infected people, δ is the mortality rate of exposed people due to virus, ϵ is the immunity rate of exposed people, γ the rate of treatment and d denotes the death rate of infected people due to virus.

Assuming all parameters of model (I.1) to be positive constants. The Caputo fractional derivative D_c^α where α belongs to the interval $(0, 1]$, is applied to a function $h \in C^1(\mathbb{R}^+, \mathbb{R})$ and is defined as follows

$$D_c^\alpha h(t) = \frac{1}{\Gamma(1 - \alpha)} \int_{t_0}^t \frac{h'(\tau)}{(t - \tau)^\alpha} d\tau.$$

In this, work suppose that $g(S, I)$ is a continuously differentiable function on \mathbb{R}_+^2 with $g(S, 0) = g(0, I) = 0$ for $S \geq 0, I \geq 0$ and

$$(\mathcal{H}_1) : \frac{\partial g(S, I)}{\partial S} > 0, \text{ and } \frac{\partial g(S, I)}{\partial I} > 0 \text{ for all } S, I \geq 0,$$

$$(\mathcal{H}_2) : \frac{g(S, I)}{I} \text{ is a bounded and monotonically decreasing function of } I > 0 \text{ for any fixed } S \geq 0 \text{ and } \lim_{I \rightarrow 0^+} \frac{g(S, I)}{I} \text{ is a continuous and monotonically increasing function on } S \geq 0.$$

The rest of this research is organized as follows. In Section II, we provide a mathematical analysis of the model under consideration. Specifically, we discuss the positivity, boundedness, and global existence of solutions. Additionally, we determine the basic reproduction number R_0 and examine the equilibria. The global stability of system (I.1) is demonstrated in Section III. In Section IV, one control variable $\nu(t)$ is introduced in our formulated SEIR epidemic model, and its sufficient and necessary conditions are determined. In Section V, we present an example with numerical simulation to illustrate our theoretical results. And the final section gives the conclusions.

II. BASIC PROPERTIES AND EQUILIBRIA

A. POSITIVITY AND BOUNDEDNESS OF SOLUTIONS

Proposition 2.1: The closed region $\Psi = \{(S, E, I, R) \in \mathbb{R}^4 : 0 < N \leq \frac{b}{\mu}\}$ is positive invariant for the system (I.1).

Proof.

$$D_c^\alpha (S + E + I + R)(t) = b - \mu(S + E + I + R)(t) - \delta E - dI \leq b - \mu N(t),$$

where $N(t) = (S + E + I + R)(t)$.

According to Lemma 3 in [17], it can be concluded that

$$N(t) \leq \left(N(0) - \frac{b}{\mu}\right) E_\alpha(-\mu t^\alpha) + \frac{b}{\mu},$$

where $E_\alpha(y) = \sum_{j=0}^{\infty} \frac{y^j}{\Gamma(\alpha j + 1)}$ is the Mittag-Leffler function of parameter α [18].

Therefore,

$$\limsup_{t \rightarrow 0} N(t) \leq \frac{b}{\mu}$$

Finally, $S(t)$, $E(t)$, $I(t)$ and $R(t)$ are bounded.

B. THE BASIC REPRODUCTION NUMBER AND EQUILIBRIA

Note that R does not appear in the first three equations of system (I.1), this allows us to study the system

$$\begin{cases} D_c^\alpha S(t) = b - g(S(t), I(t)) - (\mu + \nu)S(t), \\ D_c^\alpha E(t) = g(S(t), I(t)) - (\sigma + \mu + \delta + \epsilon)E(t), \\ D_c^\alpha I(t) = \sigma E(t) - (\mu + \gamma + d)I(t), \end{cases} \quad (\text{II.1})$$

We can clearly see that the model (II.1) always has a disease-free equilibrium $E_0 = (S_0, 0, 0)$, where $S_0 = \frac{b}{\mu + \nu}$. Using the next-generation matrix techniques developed by van den Driessche and Watmough [19], the basic reproduction number for the model is given by

$$R_0 = \frac{\sigma \frac{\partial g(E_0)}{\partial I}}{(\sigma + \mu + \delta + \epsilon)(\mu + \gamma + d)}$$

Theorem 2.2: System (II.1) has a unique endemic equilibrium $E_{**} = (S_{**}, E_{**}, I_{**})$ if and only if $R_0 > 1$.

Proof. Let $R_0 > 1$, and (S, E, I) is any positive equilibrium of system (II.1), then

$$\begin{cases} b - g(S_{**}, I_{**}) - (\mu + \nu)S_{**} = 0, \\ g(S_{**}, I_{**}) - (\sigma + \mu + \delta + \epsilon)E_{**} = 0, \\ \sigma E_{**} - (\mu + \gamma + d)I_{**} = 0. \end{cases} \quad (\text{II.2})$$

From the last equation of the above system (II.2), we get

$$E_{**} = \frac{\mu + \gamma + d}{\sigma} I_{**},$$

and by adding the first two equations of model (II.2), we get

$$S_{**} = \frac{b}{\mu + \nu} - \frac{(\sigma + \mu + \delta + \epsilon)(\mu + \gamma + d)}{\sigma(\mu + \nu)} I_{**}.$$

We define the function K from \mathbb{R}^+ to \mathbb{R} as follow

$$K(I) = \frac{g\left(\frac{b}{\mu + \nu} - \frac{(\sigma + \mu + \delta + \epsilon)(\mu + \gamma + d)}{\sigma(\mu + \nu)} I, I\right)}{I} - \frac{(\sigma + \mu + \delta + \epsilon)(\mu + \gamma + d)}{\sigma}$$

By the hypotheses $(\mathcal{H}_1) - (\mathcal{H}_2)$, the function K is strictly monotone decreasing on \mathbb{R}^+ with

$$\begin{aligned} \lim_{I \rightarrow 0^+} K(I) &= \frac{\partial g(E_0)}{\partial I} - \frac{(\sigma + \mu + \delta + \epsilon)(\mu + \gamma + d)}{\sigma} \\ &= \frac{(\sigma + \mu + \delta + \epsilon)(\mu + \gamma + d)}{\sigma} (R_0 - 1) > 0, \end{aligned}$$

and $K\left(\frac{b\sigma}{(\sigma + \mu + \delta + \epsilon)(\mu + \gamma + d)}\right) = -\frac{(\sigma + \mu + \delta + \epsilon)(\mu + \gamma + d)}{\sigma} < 0$. Hence, there is a unique $I_{**} \in (0, \frac{b\sigma}{(\sigma + \mu + \delta + \epsilon)(\mu + \gamma + d)})$ such that $K(I_{**}) = 0$ if $R_0 > 1$. Hence the system (II.1) has a unique positive endemic equilibrium.

III. GLOBAL STABILITY

The objective of this part is to construct an appropriate Lyapunov functional in order to demonstrate the global stability of the system (II.1). First, we show the global asymptotic stability of E_0 .

Theorem 3.1: If $R_0 \leq 1$, then the disease free equilibrium E_0 point of the model (II.1) is globally asymptotically stable.

Proof. To prove our result, we consider the following Lyapunov functional:

$$L(t) = \int_{S_0}^{S(t)} \left(1 - \frac{h(S_0)}{h(\sigma)}\right) d\sigma + E(t) + \frac{\sigma + \mu + \delta + \epsilon}{\sigma} I(t),$$

where $h(S) = \lim_{I \rightarrow 0^+} \frac{g(S, I)}{I}$.

Then the time fractional derivative of $L(t)$ is

$$\begin{aligned} D_c^\alpha L(t) &\leq \left(1 - \frac{h(S_0)}{h(S(t))}\right) \\ &\quad \times (b - g(S(t), I(t)) - (\mu + \nu)S(t)) \\ &\quad + g(S(t), I(t)) - (\sigma + \mu + \delta + \epsilon)E(t) \\ &\quad + \frac{\sigma + \mu + \delta + \epsilon}{\sigma} \{\sigma E(t) - (\mu + \gamma + d)I(t)\} \\ &= -(\mu + \nu) \left(1 - \frac{h(S_0)}{h(S(t))}\right) (S(t) - S_0) \\ &\quad - \left(1 - \frac{h(S_0)}{h(S(t))}\right) (g(S(t), I(t))) \\ &\quad + g(S(t), I(t)) - \frac{\mu + \sigma + \delta + \epsilon}{\sigma} (\mu + \gamma + d)I(t) \\ &= -(\mu + \nu) \left(1 - \frac{h(S_0)}{h(S(t))}\right) (S(t) - S_0) \\ &\quad - \frac{\mu + \sigma + \delta + \epsilon}{\sigma} (\mu + \gamma + d)I(t) \\ &\quad \times \left(1 - \frac{\sigma}{(\mu + \sigma + \delta + \epsilon)(\mu + \gamma + d)} \frac{h(S_0)}{h(S(t))} \frac{g(S(t), I(t))}{I(t)}\right). \end{aligned}$$

Then

$$\begin{aligned} D_c^\alpha L(t) &= -(\mu + \nu) \left(1 - \frac{h(S_0)}{h(S(t))}\right) (S(t) - S_0) \\ &\quad - \frac{\mu + \sigma + \delta + \epsilon}{\sigma} (\mu + \gamma + d)I(t) \\ &\quad \times \left(1 - \frac{\sigma}{(\mu + \sigma + \delta + \epsilon)(\mu + \gamma + d)} \frac{h(S_0)}{h(S(t))} \frac{g(S(t), I(t))}{I(t)}\right). \end{aligned}$$

By applying the hypothesis (\mathcal{H}_2) we get that,

$$\begin{aligned} &\frac{\sigma}{(\mu + \sigma + \delta + \epsilon)(\mu + \gamma + d)} \frac{h(S_0)}{h(S(t))} \frac{g(S(t), I(t))}{I(t)} \\ &\leq \frac{\sigma h(S(t))}{(\mu + \sigma + \delta + \epsilon)(\mu + \gamma + d)} \frac{h(S_0)}{h(S(t))} = R_0. \end{aligned}$$

Then we conclude that

$$D_c^\alpha L(t) \leq -(\mu + \nu) \left(1 - \frac{h(S_0)}{h(S(t))} \right) (S(t) - S_0) - \frac{\mu + \sigma + \delta + \epsilon}{\sigma} (\mu + \gamma + d) I(t) (1 - R_0).$$

From the fact that $h(S)$ is a monotonically increasing function on S , we can deduce that $\frac{\partial h(S)}{\partial S} \geq 0$, and according to (\mathcal{H}_1) , we have

$$-(\mu + \nu) \left(1 - \frac{h(S_0)}{h(S(t))} \right) (S(t) - S_0) \leq 0.$$

Hence $D_c^\alpha L(t) \leq 0$. Clearly, $\{E_0\}$ is the largest compact invariant set in $\{(S, E, I) \in \mathbb{R}_+^3 : D_c^\alpha L(t) = 0\}$. By applying LaSalle's invariance principle [20], we get that the equilibrium point E_0 of system (II.1) is globally asymptotically stable, when $R_0 \leq 1$.

Now, we study the global stability of the endemic equilibrium E_{**} . It requires the following lemma.

Lemma 3.2 (Lemma 3.1 [21]): Let v be a positive function defined by $v(z) = z - \ln(z) - 1$, $z > 0$ and $z(t) \in R_+^*$ is a continuous differentiable function for all $\alpha \in [0, 1]$ and $t > t_0$,

$$D_c^\alpha \left[z_{**} v \left(\frac{z(t)}{z_{**}} \right) \right] \leq \left(1 - \frac{z_{**}}{z(t)} \right) D_c^\alpha z(t).$$

Theorem 3.3: The endemic equilibrium point E_{**} of the model (II.1) is globally asymptotically stable.

Proof. Let define the Lyapunov function V as

$$V(t) = S(t) - S_{**} - \int_{S_{**}}^{S(t)} \frac{g(S_{**}, I_{**})}{g(\sigma, I_{**})} d\sigma + E_{**} v \left(\frac{E(t)}{E_{**}} \right) + \frac{g(S_{**}, I_{**})}{\sigma E_{**}} I_{**} v \left(\frac{I(t)}{I_{**}} \right),$$

According to Lemma 3.2, we can establish the following relationship:

$$\begin{aligned} D_c^\alpha V(t) &\leq \left[\frac{g(S(t), I_{**}) - g(S_{**}, I_{**})}{g(S(t), I_{**})} \right] D_c^\alpha S(t) \\ &\quad + \left[\frac{E(t) - E_{**}}{E(t)} \right] D_c^\alpha E(t) \\ &\quad + \frac{g(S_{**}, I_{**})}{\sigma E_{**}} \left[\frac{I(t) - I_{**}}{I(t)} \right] D_c^\alpha I(t) \\ &= \left[\frac{g(S(t), I_{**}) - g(S_{**}, I_{**})}{g(S(t), I_{**})} \right] \\ &\quad \times (b - (\mu + \nu)S(t) - g(S(t), I(t))) \\ &\quad + \left[1 - \frac{E_{**}}{E(t)} \right] (g(S(t), I(t)) - (\sigma + \mu + \delta + \epsilon)E(t)) \\ &\quad + \frac{g(S_{**}, I_{**})}{\sigma E_{**}} \left[1 - \frac{I_{**}}{I(t)} \right] (\sigma E(t) - (\mu + \gamma + d)I(t)). \end{aligned}$$

And we have

$$\begin{cases} b = g(S_{**}, I_{**}) + (\mu + \nu)S_{**}, \\ \sigma + \mu + \delta + \epsilon = \frac{g(S_{**}, I_{**})}{E_{**}}, \\ \mu + \gamma + d = \frac{\sigma E_{**}}{I_{**}}. \end{cases}$$

Hence we obtain

$$\begin{aligned} D_c^\alpha V(t) &\leq \left(1 - \frac{g(S_{**}, I_{**})}{g(S(t), I_{**})} \right) ((\mu + \nu)S_{**} - g(S(t), I(t))) \\ &\quad + g(S_{**}, I_{**}) - (\mu + \nu)S(t) \\ &\quad + \left(1 - \frac{E_{**}}{E(t)} \right) \left(g(S(t), I(t)) - \frac{g(S_{**}, I_{**})}{E_{**}} E(t) \right) \\ &\quad + \frac{g(S_{**}, I_{**})}{\sigma E_{**}} \left(1 - \frac{I_{**}}{I(t)} \right) \left(\sigma E(t) - \frac{\sigma E_{**}}{I_{**}} I(t) \right) \\ &= (\mu + \nu) \left(\frac{g(S(t), I_{**}) - g(S_{**}, I_{**})}{g(S(t), I_{**})} \right) (S_{**} - S(t)) \\ &\quad + \left(\frac{g(S(t), I_{**}) - g(S_{**}, I_{**})}{g(S(t), I_{**})} \right) \\ &\quad \times (g(S_{**}, I_{**}) - g(S(t), I(t))) \\ &\quad + \left(1 - \frac{E_{**}}{E(t)} \right) \left(\left(\frac{g(S(t), I(t))}{g(S_{**}, I_{**})} - \frac{E(t)}{E_{**}} \right) g(S_{**}, I_{**}) \right) \\ &\quad + \frac{g(S_{**}, I_{**})}{\sigma E_{**}} \left(1 - \frac{I_{**}}{I(t)} \right) \left(E_{**} \left(\frac{\sigma E(t)}{E_{**}} - \frac{\sigma I(t)}{I_{**}} \right) \right) \\ &= (\mu + \nu) \left(\frac{g(S(t), I_{**}) - g(S_{**}, I_{**})}{g(S(t), I_{**})} \right) (S_{**} - S(t)) \\ &\quad + \left(\frac{g(S(t), I_{**}) - g(S_{**}, I_{**})}{g(S(t), I_{**})} \right) \\ &\quad \times (g(S_{**}, I_{**}) - g(S(t), I(t))) \\ &\quad + \left[1 - \frac{E_{**}g(S(t), I(t))}{Ag(S_{**}, I_{**})} + \frac{g(S(t), I(t))}{g(S_{**}, I_{**})} - \frac{E(t)}{E_{**}} \right] \\ &\quad \times g(S_{**}, I_{**}) \\ &\quad + \frac{g(S_{**}, I_{**})}{\sigma E_{**}} \left(1 - \frac{I_{**}}{I(t)} \right) \left(\sigma E_{**} \left(\frac{E(t)}{E_{**}} - \frac{I(t)}{I_{**}} \right) \right), \end{aligned}$$

it follows that

$$\begin{aligned} D_c^\alpha V(t) &\leq -(\mu + \nu) \left(\frac{g(S(t), I_{**}) - g(S_{**}, I_{**})}{g(S(t), I_{**})} \right) (S(t) - S_{**}) \\ &\quad + \left[3 + \frac{g(S(t), I(t))}{g(S(t), I_{**})} - \frac{g(S_{**}, I_{**})}{g(S(t), I_{**})} \right. \\ &\quad \left. - \frac{E_{**}g(S(t), I(t))}{E(t)g(S_{**}, I_{**})} - \frac{I(t)}{I_{**}} - \frac{E(t)I_{**}}{E_{**}I(t)} \right] g(S_{**}, I_{**}). \end{aligned}$$

We have

$$\begin{aligned} 3 + \frac{g(S(t), I(t))}{g(S(t), I_{**})} - \frac{g(S_{**}, I_{**})}{g(S(t), I_{**})} - \frac{E_{**}g(S(t), I(t))}{E(t)g(S_{**}, I_{**})} - \frac{I(t)}{I_{**}} \\ - \frac{E(t)I_{**}}{E_{**}I(t)} &= -v \left(\frac{g(S_{**}, I_{**})}{g(S(t), I_{**})} \right) + v \left(\frac{g(S(t), I)}{g(S(t), I_{**})} \right) \\ &\quad - v \left(\frac{E(t)I_{**}}{E_{**}I(t)} \right) - v \left(\frac{E_{**}g(S(t), I(t))}{E(t)g(S_{**}, I_{**})} \right) - v \left(\frac{I(t)}{I_{**}} \right) \\ &= -v \left(\frac{g(S_{**}, I_{**})}{g(S(t), I_{**})} \right) - v \left(\frac{E(t)I_{**}}{E_{**}I(t)} \right) - v \left(\frac{E_{**}g(S(t), I(t))}{E(t)g(S_{**}, I_{**})} \right) \\ &\quad + \frac{g(S(t), I(t))}{g(S(t), I_{**})} - \frac{I(t)}{I_{**}} + \ln \left(\frac{I}{I_{**}} \frac{g(S(t), I_{**})}{g(S(t), I(t))} \right). \end{aligned}$$

By using hypotheses (\mathcal{H}_1) and (\mathcal{H}_2) , we get that

$$\frac{g(S(t), I(t))}{g(S(t), I_{**})} - \frac{I(t)}{I_{**}} + \ln \left(\frac{I}{I_{**}} \frac{g(S(t), I_{**})}{g(S(t), I(t))} \right) \leq 0.$$

Then

$$\begin{aligned} 3 + \frac{g(S(t), I(t))}{g(S(t), I_{**})} - \frac{g(S_{**}, I_{**})}{g(S(t), I_{**})} - \frac{E_{**}g(S(t), I(t))}{E(t)g(S_{**}, I_{**})} - \frac{I(t)}{I_{**}} \\ - \frac{E(t)I_{**}}{E_{**}I(t)} \leq -v \left(\frac{g(S_{**}, I_{**})}{g(S(t), I_{**})} \right) - v \left(\frac{E(t)I_{**}}{E_{**}I(t)} \right) \\ - v \left(\frac{E_{**}g(S(t), I(t))}{E(t)g(S_{**}, I_{**})} \right) \leq 0. \end{aligned}$$

That is $D_c^\alpha V(t) \leq 0$. It is clearly seen that $\{E_{**}\}$ is the largest compact invariant set in $\{(S, E, I) \in \mathbb{R}_+^3 : D_c^\alpha V(t) = 0\}$. By applying LaSalle's invariance principle [20], we get that the equilibrium point E_{**} of system (II.1) is globally asymptotically stable, which completes the proof.

IV. THE SEIR MODEL WITH OPTIMAL CONTROL

Vaccination plays a crucial role in combating infectious diseases. Researchers such as Ding et al. [22] and Agarwal et al. [23] have made significant contributions to the field of optimum control theory in fractional calculus. The concept of optimal control in fractional calculus is closely linked to Pontryagin's maximum principle [24]. In our study, we aim to incorporate the impact of vaccination by introducing a control measure denoted $v(t)$. The control strategy is applied within the interval $[0, t_f]$ and normalized from 0 to 1 to correspond the admissible set

$$V_{ad} = \{v : v(t) \text{ measurable, } 0 \leq v(t) \leq v_{max}\}.$$

When the control $v(t)$ is added, the model (I.1) takes the following form

$$\begin{cases} D_c^\alpha S(t) &= b - g(S(t), I(t)) - \mu S(t) - v(t)S(t), \\ D_c^\alpha E(t) &= g(S(t), I(t)) - (\epsilon + \sigma + \mu + \delta)E(t), \\ D_c^\alpha I(t) &= \sigma E(t) - (\mu + \gamma + d)I(t), \\ D_c^\alpha R(t) &= v(t)S(t) + \gamma I(t) + \epsilon E(t) - \mu R(t), \end{cases} \quad (IV.1)$$

where $v(t) \in [0, 1]$ represents the percentage of susceptible individuals being vaccinated per unit of time.

We try to find the control function v^* that minimizes the functional Θ .

The objective function is

$$\Theta(v) = \int_0^{t_f} \left[E + I + \frac{1}{2} \tau v^2 \right] dt, \quad (IV.2)$$

where τ is a positive weight parameter which is associated with the control $v(t)$.

A. EXISTENCE OF AN OPTIMAL CONTROL

Theorem 4.1: There is an optimal control $v^* \in V_{ad}$ for the control system (IV.1) such that

$$\Theta(v^*) = \min_{v \in V_{ad}} \Theta(v).$$

Proof: To demonstrate the existence of an optimal control, it is easy to verify that

- The set of solution to (IV.1) together with the initial condition and the corresponding control function in V_{ad} is non empty,
- V_{ad} is convex and closed,
- the right hand side of the formulated model (IV.1) is bounded by a linear function in the state and control variables,
- the Lagrangian $L(E, I, v)$ of the model (IV.1) on V_{ad} ,
- there exist constants $\alpha_1 > 0$ and $\alpha_2 > 0$, and $\rho > 1$ such that the integrand $L(E, I, v)$ of the objective functional satisfies $\rho > 1$ and positive numbers α_1 and α_2 such that $L(E, I, v) \geq \alpha_2 + \alpha_1 (|v|)^\rho$.

The result is a direct consequence of [25].

B. CHARACTERIZATION OF THE OPTIMAL CONTROL

In this subsection, we derive a necessary condition for the optimal control by means of the Pontryagin's maximum principle [26].

In order to characterize the optimal control, we must first establish the Lagrangian for the optimal control problems (IV.1) and (IV.2) by:

$$L(E, I, v) = E + I + \frac{1}{2} \tau v^2,$$

and the corresponding Hamiltonian H by

$$\begin{aligned} H = L(E, I, v) + \psi_1(b - g(S(t), I(t)) - \mu S(t) - v(t)S(t)) \\ + \psi_2(g(S(t), I(t)) - (\epsilon + \sigma + \mu + \delta)E(t)) + \psi_3(\sigma E(t) \\ - (\mu + \gamma + d)I(t)) + \psi_4(v(t)S(t) \\ + \gamma I(t) + \epsilon E(t) - \mu R(t)), \end{aligned}$$

here ψ_1, ψ_2 , and ψ_3 are the adjoint functions that will be determined.

Theorem 4.2: If $v^*(t)$ is an optimal control and $S^*(t), E^*(t), I^*(t)$, and $R^*(t)$ are solutions of the corresponding state system (IV.1) and (IV.2), then there exists adjoint variables $\psi_1(t), \psi_2(t), \psi_3(t)$ and $\psi_4(t)$ that satisfy

$$\begin{aligned} D_c^\alpha \psi_1 &= \psi_1 \left(\frac{\partial g}{\partial S} + \mu + v \right) - \psi_2 \frac{\partial g}{\partial S} - \psi_4 v, \\ D_c^\alpha \psi_2 &= -1 + \psi_2(\epsilon + \sigma + \mu + \delta) - \sigma \psi_3 - \psi_4 \epsilon, \\ D_c^\alpha \psi_3 &= -1 + \psi_1 \frac{\partial g}{\partial I} - \psi_2 \frac{\partial g}{\partial I} + \psi_3(\mu + \gamma + d) - \psi_4 \gamma, \\ D_c^\alpha \psi_4 &= \psi_4 \mu, \end{aligned}$$

with transversality conditions

$$\psi_1(t_f) = \psi_2(t_f) = \psi_3(t_f) = \psi_4(t_f) = 0.$$

Furthermore, we give the optimal control $w^*(t)$ as follows

$$v^* = \max \left(0, \min \left(\frac{(\psi_1 - \psi_4)S}{\tau}, v_{max} \right) \right).$$

TABLE 1. Parameter values for model (V.1).

Parameter	Value
b	40
μ	0.003
c	4
σ	0.004
δ	0.001
ϵ	0.03
γ	0.07
β	0.01
τ	20
v_{max}	1

TABLE 2. Parameter values for model (V.1).

Parameter	Value
b	30
μ	0.003
c	4
σ	0.004
δ	0.001
ϵ	0.03
γ	0.07
β	0.9
τ	20
v_{max}	1

Proof: To obtain the adjoint equations and the transversality conditions, we differentiate the Hamiltonian H . Hence

$$\begin{aligned}
 D_c^\alpha \psi_1 &= -\frac{\partial H}{\partial S} \\
 &= \psi_1 \left(\frac{\partial g}{\partial S} + \mu + v \right) - \psi_2 \frac{\partial g}{\partial S} - \psi_4 v, \\
 D_c^\alpha \psi_2 &= -\frac{\partial H}{\partial E} \\
 &= -1 + \psi_2 (\epsilon + \sigma + \mu + \delta) - \sigma \psi_3 - \psi_4 \epsilon, \\
 D_c^\alpha \psi_3 &= -\frac{\partial H}{\partial I} \\
 &= -1 + \psi_1 \frac{\partial g}{\partial I} - \psi_2 \frac{\partial g}{\partial I} + \psi_3 (\mu + \gamma + d) - \psi_4 \gamma, \\
 D_c^\alpha \psi_4 &= -\frac{\partial H}{\partial R} \\
 &= \psi_4 \mu.
 \end{aligned}$$

By the optimal conditions, we get

$$\left. \frac{\partial H}{\partial v} \right|_{v=v^*} = \tau v^* - \psi_1 S + \psi_4 S = 0.$$

It follows that

$$v^* = \max \left(0, \min \left(\frac{(\psi_1 - \psi_4)S}{\tau}, v_{max} \right) \right).$$

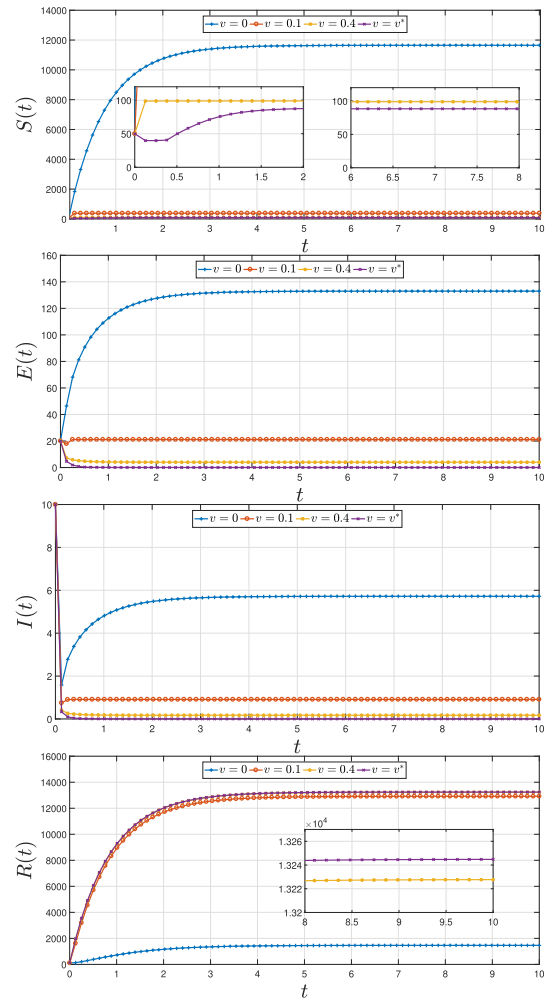


FIGURE 1. The dynamic behavior of compartments $S, E, I,$ and R in system (V.1) with the parameter values listed in Table 1 ($R_0 \leq 1$ for $v = 0.1, 0.4, v^*$ and $R_0 \geq 1$ for $v = 0$).

Therefore, we have the following optimality system:

$$\begin{cases}
 D_c^\alpha \psi_1 &= \psi_1 \left(\frac{\partial g}{\partial S} + \mu + v \right) - \psi_2 \frac{\partial g}{\partial S} - \psi_4 v, \\
 D_c^\alpha \psi_2 &= -1 + \psi_2 (\epsilon + \sigma + \mu + \delta) - \sigma \psi_3 - \psi_4 \epsilon, \\
 D_c^\alpha \psi_3 &= -1 + \psi_1 \frac{\partial g}{\partial I} - \psi_2 \frac{\partial g}{\partial I} + \psi_3 (\mu + \gamma + d) - \psi_4 \gamma, \\
 D_c^\alpha \psi_4 &= \psi_4 \mu, \\
 v^* &= \max \left(0, \min \left(\frac{(\psi_1 - \psi_4)S}{\tau}, v_{max} \right) \right), \\
 \psi_j(t_f) &= 0, \quad j = 1, 2, 3, 4.
 \end{cases}$$

V. APPLICATION AND NUMERICAL SIMULATIONS

To see the applicability of the above result, we will study the following example:

Let

$$\begin{cases}
 D_c^\alpha S(t) &= b - (\mu + v)S(t) - \beta \frac{S(t)I(t)}{1+cI^2(t)}, \\
 D_c^\alpha E(t) &= \beta \frac{S(t)I(t)}{1+cI^2(t)} - (\sigma + \mu + \delta + \epsilon)E(t), \\
 D_c^\alpha I(t) &= \sigma E(t) - (\mu + \gamma + d)I(t), \\
 D_c^\alpha R(t) &= vS(t) + \gamma I(t) + \epsilon E(t) - \mu R(t).
 \end{cases} \quad (V.1)$$

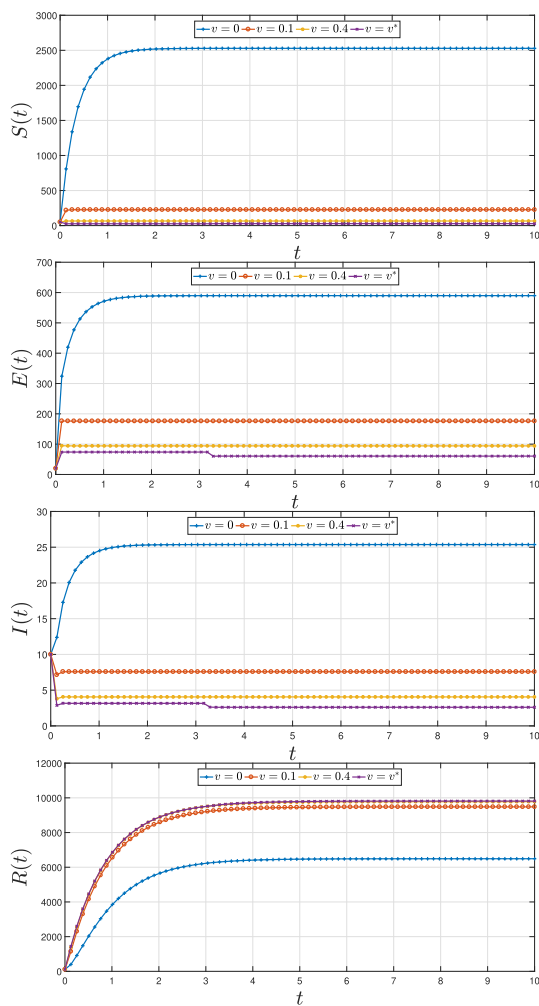


FIGURE 2. The dynamic behavior of compartments S , E , I , and R in system (V.1) with the parameter values listed in Table 2. In this case, $R_0 > 1$ for $v = 0, 0.1, 0.4, v^*$.

We choose the following parameter values:

The theoretical results and numerical simulation are in good agreement. FIGURE 1 illustrates the dynamic behavior of individuals in the system with a fractional order of $\alpha = 0.94$. Comparing the cases with a constant vaccination rate of $v = 0.1$ and $v = 0.4$, it is evident that the number of susceptible, infected, and exposed individuals decreases and the number of the recovered people decrease as the vaccination rate increases. Furthermore, when the optimal control is included $v = v^*$, the reduction in the number of $S(t)$, $E(t)$, and $I(t)$ is even greater compared to the previous case. Additionally, the number of individuals in the recovered compartment, $R(t)$, increases more significantly than before precisely converges to the free equilibrium E_0 . Then, the unique equilibrium E_0 is globally asymptotically stable. However, in case of $R_0 > 1$, it is just the opposite, due to obvious reasons as be seen in FIGURE 2 and in FIGURE 1 when $v = 0$. Here the solutions converges to the endemic equilibrium E_{**} . Therefore, the unique equilibrium E_{**} is globally asymptotically stable.

These findings suggest that increasing the vaccination rate, especially when considering optimal control strategies, has a positive impact on reducing the number of susceptible, exposed, and infected and increase the number of recovered people.

VI. CONCLUSION

In this work, we presents an analysis and optimal control study of a fractional order SEIR epidemic model with General Incidence and Vaccination. By incorporating fractional calculus, the model captures memory effects and non-local interactions in disease transmission. The analysis explores equilibrium points, their existence, uniqueness, and global stability for the equilibria, while considering the impact of vaccination on disease dynamics. Furthermore, an optimal control strategy is developed to minimize the number of infected individuals by optimizing the vaccination rate over a specific time period. Numerical simulations validate the theoretical results and demonstrate the effectiveness of the proposed control strategy in mitigating the spread of the epidemic. The findings contribute to a deeper understanding of fractional order SEIR epidemic models and offer insights for designing effective control measures against infectious diseases.

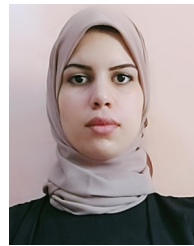
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REFERENCES

- [1] T. Khan, Z. Ullah, N. Ali, and G. Zaman, "Modeling and control of the hepatitis B virus spreading using an epidemic model," *Chaos, Solitons Fractals*, vol. 124, pp. 1–9, Jul. 2019.
- [2] R. M. Anderson and R. M. May, *Infectious Diseases of Humans: Dynamics and Control*. Oxford, U.K.: Oxford Univ. Press, 1991.
- [3] C. Vargas-De-León, "On the global stability of SIS, SIR and SIRS epidemic models with standard incidence," *Chaos Solitons Fractals*, vol. 44, Dec. 2011, Art. no. 110610.
- [4] V. Capasso and G. Serio, "A generalization of the Kermack–McKendrick deterministic epidemic model," *Math. Biosci.*, vol. 42, pp. 43–61, Nov. 1978.
- [5] Y. Enatsu, Y. Nakata, and Y. Muroya, "Lyapunov functional techniques for the global stability analysis of a delayed SIRS epidemic model," *Nonlinear Anal., Real World Appl.*, vol. 13, no. 5, pp. 2120–2133, Oct. 2012.
- [6] R. P. Sigdel and C. C. McCluskey, "Global stability for an SEI model of infectious disease with immigration," *Appl. Math. Comput.*, vol. 243, pp. 684–689, Sep. 2014.
- [7] T. Li, F. Zhang, H. Liu, and Y. Chen, "Threshold dynamics of an SIRS model with nonlinear incidence rate and transfer from infectious to susceptible," *Appl. Math. Lett.*, vol. 70, pp. 52–57, Aug. 2017.
- [8] Q. Tang, Z. Teng, and X. Abdurahman, "A new Lyapunov function for SIRS epidemic models," *Bull. Malaysian Math. Sci. Soc.*, vol. 40, no. 1, pp. 237–258, Jan. 2017.
- [9] E. J. Avila-Vales and A. G. Cervantes-Pérez, "Global stability for SIRS epidemic models with general incidence rate and transfer from infectious to susceptible," *Boletín de la Sociedad Matemática Mexicana*, vol. 25, pp. 637–658, Nov. 2019.
- [10] N. Özalp and E. Demirci, "A fractional order SEIR model with vertical transmission," *Math. Comput. Model.*, vol. 54, nos. 1–2, pp. 1–6, Jul. 2011.
- [11] M. Naim, F. Lahmidi, and A. Namir, "Stability analysis of a delayed fractional order SIRS epidemic model with nonlinear incidence rate," *Int. J. Applied Math.*, vol. 32, no. 5, Dec. 2019.

- [12] M. A. Khan and A. Atangana, "Modeling the dynamics of novel coronavirus (2019-nCov) with fractional derivative," *Alexandria Eng. J.*, vol. 59, no. 4, pp. 2379–2389, Aug. 2020.
- [13] V. S. Ertürk, G. Zaman, and S. Momani, "A numeric-analytic method for approximating a giving up smoking model containing fractional derivatives," *Comput. Math. with Appl.*, vol. 64, no. 10, pp. 3065–3074, Nov. 2012.
- [14] A. S. Shaikh and K. S. Nisar, "Transmission dynamics of fractional order typhoid fever model using Caputo–Fabrizio operator," *Chaos, Solitons Fractals*, vol. 128, pp. 355–365, Nov. 2019.
- [15] S. Ullah, M. A. Khan, M. Farooq, Z. Hammouch, and D. Baleanu, "A fractional model for the dynamics of tuberculosis infection using Caputo–Fabrizio derivative," Tech. Rep., 2020.
- [16] A. Raza, Y.-M. Chu, M. Y. Bajuri, A. Ahmadian, N. Ahmed, M. Rafiq, and S. Salahshour, "Dynamical and nonstandard computational analysis of heroin epidemic model," *Results Phys.*, vol. 34, Mar. 2022, Art. no. 105245.
- [17] H.-L. Li, L. Zhang, C. Hu, Y.-L. Jiang, and Z. Teng, "Dynamical analysis of a fractional-order predator-prey model incorporating a prey refuge," *J. Appl. Math. Comput.*, vol. 54, nos. 1–2, pp. 435–449, Jun. 2017.
- [18] D. Kai, *The Analysis of Fractional Differential Equations: An Application-Oriented Exposition Using Differential Operators of Caputo Type*. Berlin, Germany: Springer, 2010.
- [19] P. van den Driessche and J. Watmough, "Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission," *Math. Biosci.*, vol. 180, nos. 1–2, pp. 29–48, Nov. 2002.
- [20] J. P. La Salle, *The Stability of Dynamical Systems*. Philadelphia, PA, USA: Society for Industrial and Applied Mathematics, 1976.
- [21] C. Vargas-De-León, "Volterra-type Lyapunov functions for fractional-order epidemic systems," *Commun. Nonlinear Sci. Numer. Simul.*, vol. 24, nos. 1–3, pp. 75–85, Jul. 2015.
- [22] Y. Ding, Z. Wang, and H. Ye, "Optimal control of a fractional-order HIV-immune system with memory," *IEEE Trans. Control Syst. Technol.*, vol. 20, no. 3, pp. 763–769, May 2012.
- [23] O. P. Agrawal, "A general formulation and solution scheme for fractional optimal control problems," *Nonlinear Dyn.*, vol. 38, nos. 1–4, pp. 323–337, Dec. 2004.
- [24] R. Kamocki, "Pontryagin maximum principle for fractional ordinary optimal control problems," *Math. Methods Appl. Sci.*, vol. 37, no. 11, pp. 1668–1686, Jul. 2014.
- [25] G. Birkhoff and G. C. Rota, *Ordinary Differential Equations*, 4th ed. New York, NY, USA: Wiley, 1989.
- [26] W. H. Fleming and R. W. Rishel, *Deterministic and Stochastic Optimal Control*. New York, NY, USA: Springer, 1975.



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