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Existence and Stability Analysis of Fractional-Order SVEIR Epidemic Models with Generalized Incidence

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ABSTRACT: This paper develops a fractional-order epidemic model ($SVEIR$) incorporating a generalized incidence rate, analyzed within the framework of the Caputo fractional derivative. The foundational properties of the model, including existence, uniqueness, non-negativity, and boundedness of solutions, are rigorously established to ensure its well-posedness. The basic reproduction number R_0 is computed using the next-generation matrix method, providing threshold criteria for disease elimination and persistence. The model exhibits two equilibria: the disease-free equilibrium and the endemic equilibrium. Through stability analysis, we prove the global stability of these equilibria by constructing suitable Lyapunov functions and applying LaSalle's invariance principle. To validate the theoretical results and examine the effects of various epidemiological parameters, numerical simulations are performed using MATLAB. These simulations provide deeper insights into the dynamic behavior of the proposed fractional model, highlighting its potential applicability in understanding and controlling disease spread.

Key Words: Fractional derivative, $SVEIR$ epidemic model, global asymptotic stability.

Contents

Introduction

Infectious diseases have long been a significant threat to public health, often leading to widespread morbidity, mortality, and profound socio-economic consequences. Their global impact, in terms of both human life and economic burden, underscores the critical need to understand and control their spread. Epidemic modeling has emerged as an essential tool for predicting transmission patterns and guiding effective public health interventions [1,2]. Classical models such as the Susceptible-Infected-Recovered (SIR) model [4] and its extensions—including the Susceptible-Exposed-Infected-Recovered (SEIR) [20], Susceptible-Vaccinated-Infected-Recovered (SVIR) [21], and Susceptible-Infected-Quarantined-Recovered (SIQR) [22] models—have played a pivotal role in understanding disease dynamics. These compartmental models categorize individuals into distinct health states and describe transitions between these states based on predefined assumptions about disease transmission. Despite their success, classical epidemic models often rely on simplifying assumptions such as constant transmission rates, homogeneous populations, and straightforward dynamics. These assumptions may limit their predictive accuracy, especially in real-world scenarios influenced by time-dependent factors such as prior outbreaks, vaccination campaigns, and behavioral changes. In reality, disease dynamics frequently involve complex, non-local interactions, where past states and historical events exert significant influence on current and future trends. To address

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these limitations, there has been increasing interest in leveraging fractional calculus to enhance epidemiological modeling. Fractional calculus extends the concept of differentiation and integration to non-integer orders, offering a natural framework for incorporating memory-dependent behaviors and long-range interactions in complex systems. Originating from the works of mathematicians like Leibniz and Riemann, fractional calculus gained prominence in the 20th century as a powerful tool for modeling processes with memory effects. It has since found applications across various disciplines, including physics, engineering, finance, and biology [23,24,25,26]. Fractional derivatives are particularly well-suited for systems where the future behavior depends not only on the current state but also on the system's history. This characteristic is especially relevant for modeling infectious diseases, where factors such as past exposures, immunity, and delayed public health responses play critical roles in disease transmission. In the context of epidemic modeling, fractional-order differential equations provide a robust framework for capturing memory effects and non-local interactions in disease dynamics. These equations allow for a nuanced representation of how historical states—such as previous infections or vaccination campaigns—affect current transmission patterns. Fractional-order models have proven particularly valuable in studying complex scenarios like waning immunity, delayed interventions, and the long-term impacts of vaccination, as evidenced during the COVID-19 pandemic [6]. By incorporating fractional derivatives, these models deliver a more realistic portrayal of disease spread compared to traditional integer-order approaches. An integral aspect of epidemic modeling is the incidence rate, which reflects the mechanism by which susceptible individuals become infected. Classical models often use simplified functions for the incidence rate, but more complex, nonlinear formulations better capture real-world phenomena such as saturation effects, behavioral adaptations, and environmental influences [27,28]. When combined with fractional calculus, these nonlinear dynamics create a comprehensive framework for modeling epidemic outbreaks.

In this paper, we propose a fractional-order SVEIR (Susceptible-Vaccinated-Exposed-Infectious-Recovered) model, incorporating a general incidence function $g(S, I)$ and fractional derivatives using the Caputo formulation [7]. This approach allows us to account for memory effects while preserving mathematical tractability. Our model aims to provide a deeper understanding of the influence of historical dynamics—such as prior infections or vaccination efforts—on disease transmission.

We focus on analyzing the stability of disease equilibria, including the disease-free equilibrium (DFE) and endemic equilibrium. By calculating the basic reproduction number R_0, we establish thresholds for disease persistence or eradication. Furthermore, we employ Lyapunov functions and LaSalle's invariance principle to study the long-term behavior of the system [8]. Through these analyses, we aim to demonstrate how fractional-order dynamics can enhance the predictive power of epidemic models and offer insights into effective disease control strategies.

This paper is organized as follows:

- **Section 2** introduces the fractional-order SVEIR model and the mathematical formulation of the general incidence function $g(S, I)$.
- **Section 3** examines the well-posedness of the model, ensuring the existence and uniqueness of solutions.
- **Section 4** calculates the basic reproduction number R_0 and investigates the conditions for disease spread or eradication.
- **Section 5** establishes the stability of the disease-free and endemic equilibria.
- **Section 6** presents numerical simulations to illustrate the impact of the fractional-order parameter and other model parameters on disease dynamics.
- **Section 7** concludes the paper and discusses potential future research directions.

By incorporating fractional derivatives and nonlinear incidence functions, this study extends the classical approaches to epidemic modeling and offers a more comprehensive framework for understanding disease dynamics. The results have important implications for designing more effective public health strategies, particularly in cases where past disease dynamics and interventions play a significant role in shaping future outbreaks. This approach provides a more accurate and robust tool for predicting and

controlling the spread of infectious diseases, helping to inform better policy decisions in the fight against pandemics.

1. Preliminaries

This section introduces the fundamental concepts of fractional differential calculus, focusing on the Caputo fractional derivative, and presents essential lemmas for subsequent analysis.

Definition 1.1 *[23] Let $\Theta(t)$ be an integrable function. The fractional integral of order $\kappa > 0$ is defined as:*

$$\mathbb{I}_t^{\kappa}\Theta(t) = \frac{1}{\Gamma(\kappa)}\int_0^t (t-r)^{\kappa-1}\Theta(r)\,\mathrm{d}r, \quad t \geq 0,$$

where $\Gamma(\cdot)$ denotes the gamma function:

$$\Gamma(\kappa) = \int_0^{\infty} s^{\kappa-1}e^{-s}\,\mathrm{d}s.$$

Definition 1.2 *[23] The Caputo fractional derivative of order κ for a function $\Theta(t) \in \mathcal{C}^n([0,\infty),\mathbb{R})$ is defined as:*

$${}^{c}\mathbb{D}_t^{\kappa}\Theta(t) = \frac{1}{\Gamma(n-\kappa)}\int_0^t \frac{\Theta^{(n)}(r)}{(t-r)^{\kappa-n+1}}\,\mathrm{d}r,$$

where $t \geq 0$, n is a positive integer such that $n-1 < \kappa < n$. For $0 < \kappa < 1$, it reduces to:

$${}^{c}\mathbb{D}_t^{\kappa}\Theta(t) = \frac{1}{\Gamma(1-\kappa)}\int_0^t \frac{\Theta'(r)}{(t-r)^{\kappa}}\,\mathrm{d}r.$$

Definition 1.3 *[23] The Mittag-Leffler function with two parameters is defined as:*

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

Notably, the Mittag-Leffler function reduces to the exponential function $\exp(z)$ when $\kappa = \beta = 1$.

Lemma 1.1 *[23] For $r > 0$ and $\kappa, \beta > 0$, the Laplace transform of the Mittag-Leffler function is given by:*

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

Additionally, the Laplace transform of the Caputo fractional derivative satisfies:

$$\mathcal{L}\big({}^{c}\mathbb{D}_t^{\kappa}h(t)\big) = s^{\kappa}\hat{h}(s) - \sum_{i=0}^{n-1}h^{(i)}(0)s^{\kappa-i-1},$$

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

Lemma 1.2 *[23] For $0 < \kappa < 1$, let $\xi(t)$ be a positive function defined on $[0,T]$. Then, for all $t \in [0,T)$:*

$${}^{c}\mathbb{D}_t^{\kappa}\Big(\xi(t) - \xi^* - \xi^*\ln\frac{\xi(t)}{\xi^*}\Big) \leq \Big(1-\frac{\xi^*}{\xi(t)}\Big){}^{c}\mathbb{D}_t^{\kappa}\xi(t),$$

for any $\xi^* \in \mathbb{R}_+$.

Lemma 1.3 *[23] Consider the fractional-order system defined in the Caputo sense:*

$${}^{c}\mathbb{D}_t^{\alpha}w(t) = \Psi(t,w(t)), \quad t \geq 0, \quad w(0) = w_0 \in \mathbb{R}^n, \tag{1.1}$$

where $\Psi : \mathbb{R}_+ \times \mathbb{R}^n \to \mathbb{R}^n$ is a continuous function. Assume the following conditions are satisfied:

1. *$\Psi(t,w)$ is continuous with respect to t for all $w \in \mathbb{R}^n$.*
2. *$\Psi(t,w)$ and $\frac{\partial\Psi}{\partial w}$ are continuous with respect to $w \in \mathbb{R}^n$.*
3. *There exist constants $c_1, c_2 > 0$ such that $\|\Psi(t,w)\| \leq c_1 + c_2\|w\|$ for all $w \in \mathbb{R}^n$.*

Then, the system (1.1) has a unique solution on $[0,\infty)$.

2. Model Formulation

The Classical SVEIR Model

This section introduces the classical SVEIR model, which divides the total population $N(t)$ at time $t \geq 0$ into five compartments: susceptible $S(t)$, vaccinated $V(t)$, exposed $E(t)$, infected $I(t)$, and recovered $R(t)$, such that $N(t) = S(t)+V(t)+E(t)+I(t)+R(t)$. The exposed compartment represents individuals who have been exposed to the infection but are not yet infectious.

Each compartment is subject to natural mortality at the rate μ, while the population increases at a constant rate A_N, with new members entering the susceptible class. A fraction of the susceptible population receives vaccination at the rate γ, moving to the vaccinated class. Over time, some vaccinated individuals lose immunity and return to the susceptible class at the rate δ. The general incidence function $g(S, I)$ models the infection transmission. Infectious individuals recover at a rate ϵ, entering the recovered class, where they are assumed to be immune to reinfection.

The SVEIR epidemic model can be described by the following system of equations:

$$\begin{cases} \dfrac{dS}{dt} = A_N - g(S,I) - (\mu+\gamma)S + \delta V, \\ \dfrac{dV}{dt} = \gamma S - (\mu+\delta)V, \\ \dfrac{dE}{dt} = g(S,I) - (\mu+\sigma)E, \\ \dfrac{dI}{dt} = \sigma E - (\mu+\epsilon)I, \\ \dfrac{dR}{dt} = \epsilon I - \mu R. \end{cases} \tag{2.1}$$

The parameters of system (2.1) are positive constants, defined as follows:

Table 1: Description of model parameters.

Parameter	Description
A_N	Recruitment rate of new susceptible individuals
μ	Natural death rate
δ	Rate of immunity loss among vaccinated individuals
γ	Vaccination rate
σ	Rate of transition from exposed to infected class
ϵ	Recovery rate from infected class

The function $g(S, I)$ represents the general incidence rate, which models the rate at which susceptible individuals become infected upon contact with infectious individuals. This general form allows for more flexibility compared to the bilinear incidence βSI [9]. Commonly used forms include:

- Saturated incidence $g(S, I) = \frac{\beta SI}{1+kI}$, [10,11,12,13];
- Beddington-DeAngelis incidence $g(S, I) = \frac{\beta SI}{1+\alpha S+\gamma I}$, [14];
- Crowley-Martin incidence $g(S, I) = \frac{\beta SI}{1+\alpha S+\gamma I+\delta SI}$, [15].

To ensure biological relevance, the incidence function $g(S, I)$ satisfies the following assumptions:

$$(H_1) \quad g(S,0) = g(0,I) = 0, \quad \forall S, I \geq 0; \tag{2.2}$$

$$(H_2) \quad \frac{\partial g(S,I)}{\partial S} > 0, \quad \frac{\partial g(S,I)}{\partial I} \geq 0, \quad \forall S, I \geq 0; \tag{2.3}$$

$$(H_3) \quad \frac{g(S,I)}{I} \text{ is bounded and monotonically decreasing, } \forall S, I \geq 0. \tag{2.4}$$

The Fractional SVEIR Model

Motivated by system (2.1), we propose a fractional-order extension to account for memory effects in the dynamics:

$$\begin{cases} {}^c\mathbb{D}_t^\alpha S = A_N - g(S,I) - (\mu+\gamma)S + \delta V, \\ {}^c\mathbb{D}_t^\alpha V = \gamma S - (\mu+\delta)V, \\ {}^c\mathbb{D}_t^\alpha E = g(S,I) - (\mu+\sigma)E, \\ {}^c\mathbb{D}_t^\alpha I = \sigma E - (\mu+\epsilon)I, \\ {}^c\mathbb{D}_t^\alpha R = \epsilon I - \mu R. \end{cases} \tag{2.5}$$

Here, ${}^c\mathbb{D}_t^\alpha$ denotes the Caputo fractional derivative of order $0 < \alpha \leq 1$, capturing the memory effects in the system [18]. The initial conditions are:

$$S(0) = S_0 \geq 0, \quad V(0) = V_0 \geq 0, \quad E(0) = E_0 \geq 0, \quad I(0) = I_0 \geq 0, \quad R(0) = R_0 \geq 0. \tag{2.6}$$

3. Existence and Uniqueness of Positive Solutions

Proposition 3.1 *For any non-negative initial values $(S_0, V_0, E_0, I_0, R_0)$, the system (2.5) has a unique non-negative solution on $[0, +\infty)$.*

Proof: We rewrite system (2.5) as a Caputo fractional derivative system of order $0 < \alpha \leq 1$, as follows:

$${}^c\mathbb{D}_t^\alpha w(t) = \Psi(t, w(t)), \quad t \geq 0, \quad w(0) = w_0 \in \mathbb{R}_+^5,$$

where

$$w(t) = \begin{pmatrix} S(t) \\ V(t) \\ E(t) \\ I(t) \\ R(t) \end{pmatrix} \quad \text{and} \quad w_0 = \begin{pmatrix} S_0 \\ V_0 \\ E_0 \\ I_0 \\ R_0 \end{pmatrix}.$$

We define

$$\Psi(t, w(t)) = \begin{pmatrix} A_N^\alpha - g(S,I) - (\mu^\alpha + \gamma^\alpha)S(t) + \delta^\alpha V(t) \\ \gamma^\alpha S(t) - (\mu^\alpha + \delta^\alpha)V(t) \\ g(S,I) - (\mu^\alpha + \sigma^\alpha)E(t) \\ \sigma^\alpha E(t) - (\mu^\alpha + \epsilon^\alpha)I(t) \\ \epsilon^\alpha I(t) - \mu^\alpha R(t) \end{pmatrix}.$$

Let

$$L = \begin{pmatrix} A_N^\alpha \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, A_1 = \begin{pmatrix} -(\mu^\alpha + \gamma^\alpha) & \delta^\alpha & 0 & 0 & 0 \\ \gamma^\alpha & -(\mu^\alpha + \delta^\alpha) & 0 & 0 & 0 \\ 0 & 0 & -(\mu^\alpha + \sigma^\alpha) & 0 & 0 \\ 0 & 0 & \sigma^\alpha & -(\mu^\alpha + \epsilon^\alpha) & 0 \\ 0 & 0 & 0 & \epsilon^\alpha & -\mu^\alpha \end{pmatrix},$$

and

$$A_2 = \begin{pmatrix} 0 & 0 & 0 & -1 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}.$$

Additionally, $w(t)$ satisfies the first and second conditions of Lemma 1.3. Therefore, we have

$$\Psi(t, w(t)) = L + A_1 w(t) - \frac{g(S,I)}{I} A_2 w(t).$$

Now, taking the norm of $\Psi(t, w(t))$,

$$\|\Psi(t, w(t))\| \leq \|L\| + \left(\|A_1\| + \left\| \frac{g(S, I)}{I} \right\| \|A_2\| \right) \|w(t)\|.$$

By applying Lemma 1.3, we conclude that system (2.5) has a unique solution.

Next, we demonstrate the positivity of the solution:

$$\begin{aligned}
{}^{c}\mathbb{D}_t^{\alpha} S(t)\big|_{S=0} &= A_N^{\alpha} + \delta^{\alpha} V \geq 0, \\
{}^{c}\mathbb{D}_t^{\alpha} V(t)\big|_{V=0} &= \gamma^{\alpha} S \geq 0, \\
{}^{c}\mathbb{D}_t^{\alpha} E(t)\big|_{E=0} &= 0 \geq 0, \\
{}^{c}\mathbb{D}_t^{\alpha} I(t)\big|_{I=0} &= \sigma^{\alpha} E \geq 0, \\
{}^{c}\mathbb{D}_t^{\alpha} R(t)\big|_{R=0} &= \epsilon^{\alpha} I \geq 0.
\end{aligned}$$

□

Proposition 3.2 *The set*

$$\Theta = \{(S, V, E, I, R) \in \mathbb{R}_+^5 : 0 < S(t) + V(t) + E(t) + I(t) + R(t) \leq \frac{A^{\alpha}}{\mu^{\alpha}}\}$$

is a positively invariant and attracting region for system (2.5).

Proof: We have

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

Thus, by adding the equations of system (2.5), we obtain

$${}^{c}\mathbb{D}_t^{\alpha} N(t) = A_N^{\alpha} - \mu^{\alpha} N(t).$$

Applying the Laplace transform to this equation yields

$$p^{\alpha} \hat{N}(p) - p^{\alpha - 1} N(0) = \frac{A_N^{\alpha}}{p} - \mu^{\alpha} \hat{N}(p).$$

Therefore,

$$\hat{N}(p) = A_N^{\alpha} \frac{p^{\alpha - (1+\alpha)}}{p^{\alpha} + \mu^{\alpha}} + N(0) \frac{p^{\alpha - 1}}{p^{\alpha} + \mu^{\alpha}}.$$

Hence, we have

$$\begin{aligned}
N(t) &= A_N^{\alpha} t^{\alpha} E_{\alpha, \alpha+1}(-\mu^{\alpha} t^{\alpha}) + N(0) E_{\alpha,1}(-\mu^{\alpha} t^{\alpha}), \\
N(t) &= \frac{A_N^{\alpha}}{\mu^{\alpha}} - \frac{A_N^{\alpha}}{\mu^{\alpha}} E_{\alpha,1}(-\mu^{\alpha} t^{\alpha}) + N(0) E_{\alpha,1}(-\mu^{\alpha} t^{\alpha}).
\end{aligned}$$

Since $0 \leq E_{\alpha,1}(-\mu^{\alpha} t^{\alpha}) \leq 1$ and $N(0) \leq \frac{A_N^{\alpha}}{\mu^{\alpha}}$, we conclude that $N(t) \leq \frac{A_N^{\alpha}}{\mu^{\alpha}}$.

Thus, Θ is a positively invariant set, and all initial solutions that belong to Θ remain in Θ for all $t > 0$. This completes the proof. □

4. Equilibrium Points and Basic Reproduction Number

In this section, we first establish that the system (2.5) has a unique disease-free equilibrium. Next, we calculate the basic reproduction number R_0 using the next-generation matrix method.

Disease-Free Equilibrium (DFE)

To find the disease-free equilibrium, we solve for the values of the variables $P_0 = (\bar{S}, \bar{V}, \bar{E}, \bar{I}, \bar{R}) \in \mathbb{R}^5_+$ that satisfy the system of equations when no individuals are infected ($\bar{I} = 0$ and $\bar{E} = 0$). The system of equations becomes:

$$\begin{cases} A_N^\alpha - g(\bar{S}, \bar{I}) - (\mu^\alpha + \gamma^\alpha)\bar{S} + \delta^\alpha \bar{V} = 0, \\ \gamma^\alpha \bar{S} - (\mu^\alpha + \delta^\alpha)\bar{V} = 0, \\ g(\bar{S}, \bar{I}) - (\mu^\alpha + \sigma^\alpha)\bar{E} = 0, \\ \sigma^\alpha \bar{E} - (\mu^\alpha + \epsilon^\alpha)\bar{I} = 0, \\ \epsilon^\alpha \bar{I} - \mu^\alpha \bar{R} = 0. \end{cases} \tag{4.1}$$

Since the disease-free equilibrium corresponds to the absence of infected individuals ($\bar{I} = 0$) and exposed individuals ($\bar{E} = 0$), we solve the reduced system:

$$\begin{aligned} A_N^\alpha - (\mu^\alpha + \gamma^\alpha)\bar{S} + \delta^\alpha \bar{V} = 0, \\ \gamma^\alpha \bar{S} - (\mu^\alpha + \delta^\alpha)\bar{V} = 0. \end{aligned}$$

Solving these two equations for $\bar{S}$ and $\bar{V}$, we find:

$$\bar{S} = \frac{A_N^\alpha(\mu^\alpha + \delta^\alpha)}{(\mu^\alpha)^2 + (\gamma^\alpha + \delta^\alpha)\mu^\alpha},$$

$$\bar{V} = \frac{A_N^\alpha \gamma^\alpha}{(\mu^\alpha)^2 + (\gamma^\alpha + \delta^\alpha)\mu^\alpha}.$$

Thus, the disease-free equilibrium is given by:

$$P_0 = (\bar{S}, \bar{V}, 0, 0, 0).$$

The Basic Reproduction Number R_0

The basic reproduction number R_0 is a threshold parameter that determines whether an epidemic will occur. For a general compartmental disease transmission model, R_0 is defined as the spectral radius of the next-generation matrix.

In this case, we calculate R_0 using the next-generation matrix method as described in [4]. The basic reproduction number is given by:

$$R_0 = \rho\left(FV^{-1}\right),$$

where F and V are the matrices representing the new infections and the transitions between compartments, respectively. Specifically:

$$F = \begin{pmatrix} 0 & \frac{\partial g}{\partial I}(\bar{S}, 0) \\ 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} -(\mu^\alpha + \sigma^\alpha) & 0 \\ \sigma^\alpha & -(\mu^\alpha + \epsilon^\alpha) \end{pmatrix}.$$

The basic reproduction number R_0 is then:

$$R_0 = \frac{\sigma^\alpha}{C_2 C_3} \frac{\partial g}{\partial I}(\bar{S}, 0),$$

where $C_2 = (\mu^\alpha + \sigma^\alpha)$ and $C_3 = (\mu^\alpha + \epsilon^\alpha)$.

Endemic Equilibrium (EE)

To find the endemic equilibrium point $P^* = (S^*, V^*, E^*, I^*, R^*)$, we solve the system of equations corresponding to the steady-state of the system with infected individuals present. The system of equations for the endemic equilibrium is:

$$\begin{aligned} A_N^\alpha - g(S^*, I^*) - C_1 S^* + \delta^\alpha V^* &= 0, \\ g(S^*, I^*) - C_2 E^* &= 0, \\ \sigma^\alpha E^* - C_3 I^* &= 0, \\ \epsilon^\alpha I^* - \mu^\alpha R^* &= 0, \\ \gamma^\alpha S^* - C_4 V^* &= 0, \end{aligned}$$

where $C_1 = (\mu^\alpha + \gamma^\alpha)$, $C_2 = (\mu^\alpha + \sigma^\alpha)$, $C_3 = (\mu^\alpha + \epsilon^\alpha)$, and $C_4 = (\mu^\alpha + \delta^\alpha)$.
From these equations, we find the following relationships:

$$A_N^\alpha - C_1 S^* + \delta^\alpha V^* = C_2 E^* = C_2 \frac{C_3 I^*}{\sigma^\alpha},$$

and

$$V^* = \frac{\gamma^\alpha S^*}{C_4}.$$

By solving these equations, we obtain the following expressions for the endemic equilibrium:

$$S^* = \frac{A_N^\alpha - C_2 \frac{C_3 I^*}{\sigma^\alpha}}{C_1 - \frac{\delta^\alpha \gamma^\alpha}{C_4}},$$

$$S^* \geq 0 \quad \text{if} \quad I^* \leq \frac{A_N^\alpha \sigma^\alpha}{C_2 C_3}.$$

To determine the value of I^*, we define the function $F(I)$ on the interval $[0, \frac{A_N^\alpha \sigma^\alpha}{C_2 C_3}]$:

$$F(I) = \frac{g(S, I)}{I} - \frac{C_2 C_3}{\sigma^\alpha}.$$

Since $F(I)$ is a monotonically decreasing function of I, we have:

$$\lim_{I \to 0^+} F(I) = \frac{\partial g(\bar{S}, 0)}{\partial I} - \frac{C_2 C_3}{\sigma^\alpha},$$

and

$$\lim_{I \to 0^+} F(I) = \frac{C_2 C_3}{\sigma^\alpha}(R_0 - 1).$$

Thus, if $R_0 > 1$, we have $\lim_{I \to 0^+} F(I) > 0$, and $F\left(\frac{A_N^\alpha \sigma^\alpha}{C_2 C_3}\right) < 0$. Therefore, there exists a unique endemic equilibrium P^* when $R_0 > 1$, given by:

$$S^* = \frac{A_N^\alpha - C_2 \frac{C_3 I^*}{\sigma^\alpha}}{C_1 - \frac{\delta^\alpha \gamma^\alpha}{C_4}},$$

$$E^* = \frac{C_3 I^*}{\sigma^\alpha}, \quad V^* = \frac{\gamma^\alpha S^*}{C_4}, \quad R^* = \frac{\epsilon^\alpha I^*}{\mu^\alpha}.$$

5. Global Stability of Steady States

Global Stability of Disease-Free Equilibrium P_0

This section examines the global stability of the endemic equilibrium P^* and the disease-free equilibrium P_0 for system (2.5) by creating suitable Lyapunov functions.

We describe a function ϕ: $\mathbb{R}_+ \to \mathbb{R}_+$ given by

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

Note that $\phi(\xi)$ is a non-negative function for any $\xi > 0$ that attains a global minimum at $\xi = 1$. Additionally, we define

$$\Xi = \{(S, V, E, I) \in \mathbb{R}^4 : S > 0,\ V > 0,\ E > 0,\ I > 0\}.$$

Theorem 5.1 *The disease-free equilibrium P_0 of system (2.5) is globally asymptotically stable on Ξ, if $R_0 < 1$, and unstable when $R_0 > 1$.*

Proof: We create a Lyapunov function as follows: $V_1 : \Xi \longrightarrow \mathbb{R}$:

$$V_1(t) = \sigma^\alpha E(t) + (\sigma^\alpha + \mu^\alpha) I(t).$$

In relation to the disease-free steady state P_0, the function V_1 is non-negative and attains a global minimum. By using the Caputo fractional derivative on equations of system (2.5), we obtain

$$\begin{aligned}
{}^c\mathbb{D}_t^\alpha V_1(t) &= \sigma^\alpha\, {}^c\mathbb{D}_t^\alpha E(t) + (\sigma^\alpha + \mu^\alpha)\, {}^c\mathbb{D}_t^\alpha I(t) \\
&= \sigma^\alpha \left(g(S,I) - (\sigma^\alpha + \mu^\alpha)E(t)\right) + (\sigma^\alpha + \mu^\alpha)\left(\sigma^\alpha E(t) - (\epsilon^\alpha + \mu^\alpha)\right) I(t) \\
&= (\sigma^\alpha + \mu^\alpha)(\epsilon^\alpha + \mu^\alpha) I(t) \left(\frac{\sigma^\alpha}{(\sigma^\alpha + \mu^\alpha)(\epsilon^\alpha + \mu^\alpha)} \frac{g(S,I)}{I(t)} - 1 \right) \\
&= (\sigma^\alpha + \mu^\alpha)(\epsilon^\alpha + \mu^\alpha) I(t) \left(\frac{\sigma^\alpha}{(\sigma^\alpha + \mu^\alpha)(\epsilon^\alpha + \mu^\alpha)} \frac{g(S,I) - g(S,0)}{I(t)} - 1 \right) \\
&= (\sigma^\alpha + \mu^\alpha)(\epsilon^\alpha + \mu^\alpha) I(t)(R_0 - 1).
\end{aligned}$$

Consequently, $R_0 < 1$ ensures that for all $(S(t), V(t), E(t), I(t))$,

$${}^c\mathbb{D}_t^\alpha V_1(t) \leq 0 \quad \text{for all } t \geq 0.$$

Furthermore, it is easy to verify that ${}^c\mathbb{D}_t^\alpha V_1(t) = 0$ at the disease-free equilibrium point. By using LaSalle's invariance principle [19], we have $\{P_0\}$ as the largest invariant subset of the set

$$\{(S, V, E, I) \in \Xi \mid {}^c\mathbb{D}_t^\alpha V_1(t) = 0\},$$

which implies that P_0 is globally asymptotically stable. □

Global Stability of Endemic Equilibrium P^*

Theorem 5.2 *For all $t \geq 0$, assume that $V^* > V(t)$. If $R_0 > 1$, the endemic equilibrium P^* of system (2.5) is globally asymptotically stable on Ξ.*

Proof: We define a Lyapunov function given by $V_2 : \Xi \longrightarrow \mathbb{R}$:

$$V_2(t) = \phi(S(t)) + \frac{\delta^\alpha}{\mu^\alpha + \delta^\alpha}\phi(V(t)) + \phi(E(t)) + \frac{\mu^\alpha + \sigma^\alpha}{\sigma^\alpha}\phi(I(t)).$$

The function V_2 is non-negative and continuous for all $t \geq 0$. Applying the Caputo fractional derivative, we obtain

$$ {}^c\mathbb{D}_t^\alpha V_2(t) = {}^c\mathbb{D}_t^\alpha \phi(S(t)) + \frac{\delta^\alpha}{\mu^\alpha + \delta^\alpha} {}^c\mathbb{D}_t^\alpha \phi(V(t)) + {}^c\mathbb{D}_t^\alpha \phi(E(t)) + \frac{\mu^\alpha + \sigma^\alpha}{\sigma^\alpha} {}^c\mathbb{D}_t^\alpha \phi(I(t)), $$

$$ \leq \left(1 - \frac{S^*}{S}\right) {}^c\mathbb{D}_t^\alpha S(t) + \frac{\delta^\alpha}{\mu^\alpha + \delta^\alpha} \left(1 - \frac{V^*}{V}\right) {}^c\mathbb{D}_t^\alpha V(t) + \left(1 - \frac{E^*}{E}\right) {}^c\mathbb{D}_t^\alpha E(t) + \frac{\mu^\alpha + \sigma^\alpha}{\sigma^\alpha} \left(1 - \frac{I^*}{I}\right) {}^c\mathbb{D}_t^\alpha I(t), $$

$$ = \left(1 - \frac{S^*}{S}\right) (A_N^\alpha - g(S,I) - (\mu^\alpha + \gamma^\alpha) S(t) + \delta^\alpha V(t)) $$

$$ + \frac{\delta^\alpha}{\mu^\alpha + \delta^\alpha} \left(1 - \frac{V^*}{V}\right) (\gamma^\alpha S(t) - (\mu^\alpha + \delta^\alpha) V(t)) $$

$$ + \left(1 - \frac{E^*}{E}\right) (g(S,I) - (\mu^\alpha + \sigma^\alpha) E(t)) $$

$$ + \frac{\mu^\alpha + \sigma^\alpha}{\sigma^\alpha} \left(1 - \frac{I^*}{I}\right) (\sigma^\alpha E(t) - (\mu^\alpha + \epsilon^\alpha) I(t)) . $$

Using the endemic conditions

$$ A_N^\alpha = g(S^*, I^*) + (\mu^\alpha - \gamma^\alpha) S^* + \delta^\alpha V^*, $$

we get

$$ {}^c\mathbb{D}_t^\alpha V_2(t) \leq -(\mu^\alpha + \gamma^\alpha) \frac{(S - S^*)^2}{S} + g(S^*, I^*) - \frac{g(S^*, I^*) S^*}{S} - \delta^\alpha V^* + \frac{\delta^\alpha V^* S^*}{S}. $$

After some arrangement and simplifications, we reach

$$ {}^c\mathbb{D}_t^\alpha V_2(t) \leq -(\mu^\alpha + \gamma^\alpha) \frac{(S - S^*)^2}{S} + g(S^*, I^*) \left(3 - \frac{S^*}{S} - \frac{\sigma^\alpha}{\mu^\alpha + \epsilon^\alpha} \frac{E^*}{I} - \frac{\mu^\alpha + \epsilon^\alpha}{\sigma^\alpha} \frac{I}{E^*}\right). $$

Since the arithmetic mean is greater than or equal to the geometric mean, we have

$$ \left(3 - \frac{S^*}{S} - \frac{\sigma^\alpha}{\mu^\alpha + \epsilon^\alpha} \frac{E^*}{I} - \frac{\mu^\alpha + \epsilon^\alpha}{\sigma^\alpha} \frac{I}{E^*}\right) \leq 0. $$

Consequently, ${}^c\mathbb{D}_t^\alpha V_2(t) \leq 0$. Meanwhile, ${}^c\mathbb{D}_t^\alpha V_2(t) = 0$ if and only if $S = S^*$, $I = I^*$, $V = V^*$, and $E = E^*$. Thus, the largest compact invariant set in

$$ \{(S, V, E, I) \in \Xi \mid {}^c\mathbb{D}_t^\alpha V_2(t) = 0\} $$

is the singleton set $\{P^*\}$. By LaSalle's invariance principle, P^* is globally asymptotically stable for $R_0 > 1$. □

6. Numerical Simulations

The objective of this section is to validate the theoretical results presented in previous sections by performing numerical simulations of the model, incorporating fractional derivatives of various orders. This allows us to examine the impact of different memory effects on the disease transmission dynamics. Additionally, we model the general incidence function as a saturated incidence rate, represented as $\frac{\beta SI}{1+kI}$, which highlights the dynamics of disease spread with two key factors: β, the transmission rate, and k, the saturation parameter.

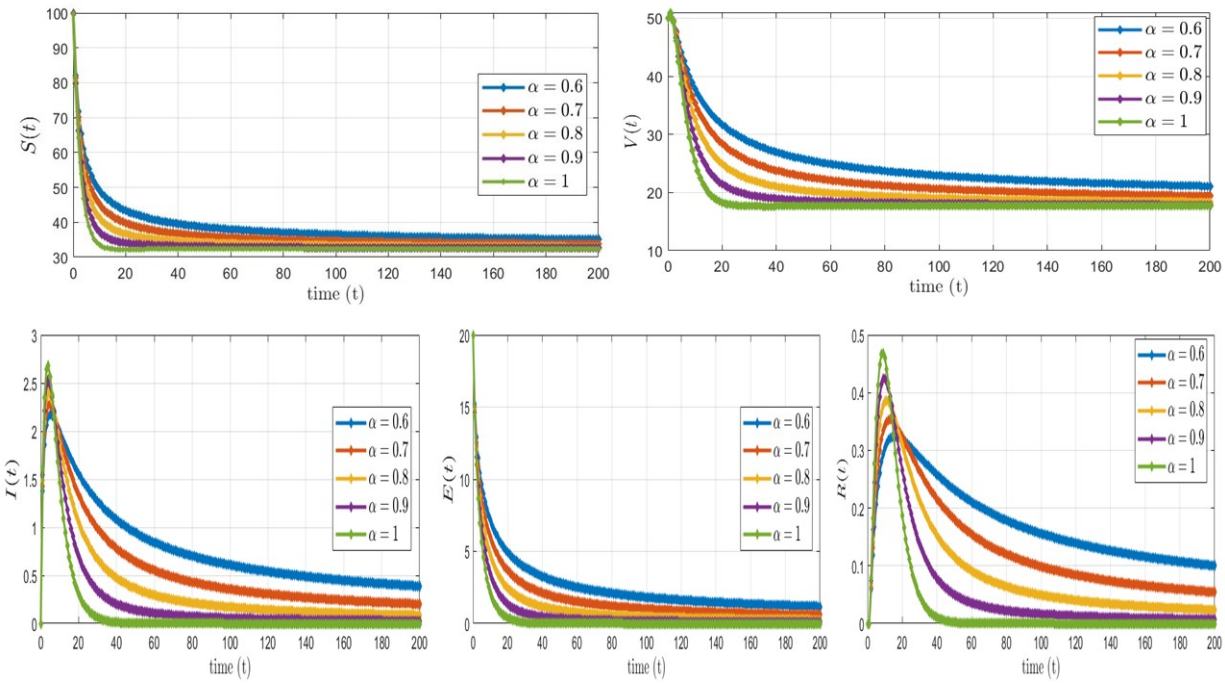

Figure 1: The dynamic behavior of compartments S, V, E, I, and R for $\alpha = 0.6$, $\alpha = 0.7$, $\alpha = 0.8$, $\alpha = 0.9$, and $\alpha = 1$.

Case of Disease-Free Equilibrium

We first investigate the case of the disease-free equilibrium, where $R_0 = 0.184 < 1$. The following parameter values are used:

$$\beta = 0.005, \quad \gamma = 0.12, \quad \delta = 0.02, \quad \mu = 0.2, \quad A = 10, \quad \epsilon = 0.05, \quad \sigma = 0.08, \quad k = 0.0095.$$

The initial conditions are given by $(S(0), V(0), E(0), I(0), R(0)) = (100, 50, 20, 0, 0)$.

Figure 1 confirms the theoretical results regarding the stability of the disease-free equilibrium as mentioned in Theorem 5.1. It shows that the disease eventually disappears as expected. Additionally, it is observed that increasing the fractional-order parameter α significantly reduces the spread of infections within the population.

Case of Endemic Equilibrium

Next, we consider the situation of the endemic equilibrium with $R_0 = 2.5 > 1$. The following parameter values are used:

$$\beta = 0.0434, \quad \gamma = 0.1152, \quad \delta = 0.0594, \quad \mu = 0.1906, \quad A = 10.0034, \quad \epsilon = 0.0474, \quad \sigma = 0.1176, \quad k = 0.075.$$

The initial conditions are $(S(0), V(0), E(0), I(0), R(0)) = (100, 50, 20, 0, 0)$.

Figure 2 validates the theoretical findings regarding the stability of the endemic equilibrium as established in Theorem 5.2. It shows that the disease persists within the population and reaches a stable endemic state. Furthermore, it is observed that the value of α influences the convergence rate of the infection spread. Specifically, a decrease in α results in a slower convergence, meaning that the infection spreads more slowly as the memory effect (represented by α) decreases.

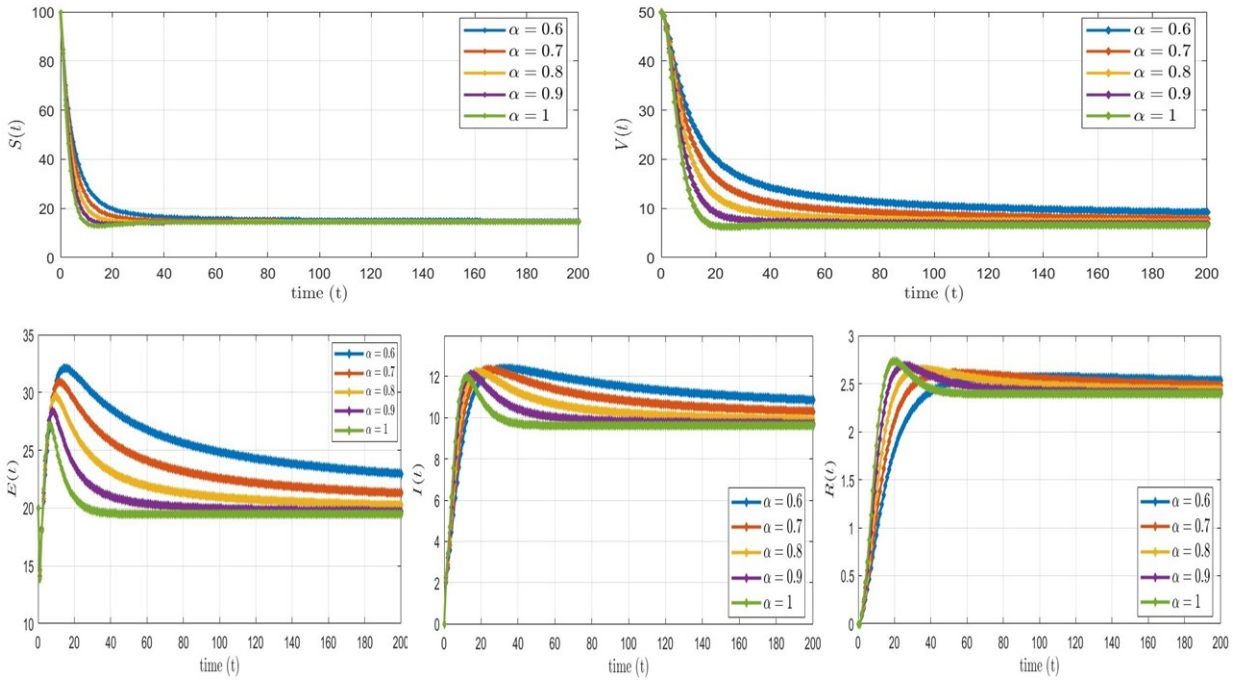

Figure 2: The dynamic behavior of compartments S, V, E, I, and R for $\alpha = 0.6$, $\alpha = 0.7$, $\alpha = 0.8$, $\alpha = 0.9$, and $\alpha = 1$.

7. Conclusion

In this study, we proposed a fractional-order epidemic model ($SVEIR$) with a generalized incidence rate, analyzed using the Caputo fractional derivative framework. The model's well-posedness was rigorously established by proving the existence, uniqueness, non-negativity, and boundedness of its solutions. Through the computation of the basic reproduction number R_0 using the next-generation matrix method, we derived critical threshold conditions for disease control. The stability analysis of the disease-free and endemic equilibria confirmed their global stability under specific conditions, utilizing Lyapunov functions and LaSalle's invariance principle. Numerical simulations conducted with MATLAB not only validated the theoretical results but also highlighted the influence of key parameters on the dynamics of the epidemic. These findings emphasize the utility of fractional-order models in capturing complex epidemiological behaviors and provide a robust framework for understanding disease transmission and control strategies.

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Conflict of interest

The authors declare that they have no conflict of interest.

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