MSW MANAGEMENT -Multidisciplinary, Scientific Work and Management Journal
ISSN: 1053-7899
Vol. 36 Issue 1, Jan-June 2026, Pages: 239-250

Fractional Dynamics on Transmission of Oropouche Fever

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Abstract:
This study presents an enhanced fractional-order SEIR (Susceptible-Exposed- Infected-Recovered) model to analyse the transmission dynamics of Oropouche fever (Sloth Fever) an emerging arboviral disease. By incorporating memory effects through fractional calculus, the model better captures real-world disease progression. Traditional and enhanced fractional SEIR models are implemented using python with real epidemiological data. A comparative analysis highlights the fractional order and its advantage in modeling long-term persistence. Addi- tionally, a stability framework evaluates intervention strategies. Finally, the results indicate the enhanced fractional SEIR model offers superior predic- tive accuracy and flexibility, making it valuable for public health planning and outbreak mitigation.

Keywords: Oropouche fever(Sloth Fever), Fractional Calculus, Enhanced Fractional Order-SEIR model, Epidemic Modelling, Stability, Existence and Uniqueness, Adomian method.

1. INTRODUCTION

Fractional-order calculus has become an essential framework for constructing math- ematical models. complex biological systems exhibiting memory effects and anomalous diffusion dynamics. Since Caputo's foundational work in 1967 introducing fractional derivatives that preserve conventional initial conditions, this mathematical framework has proven particularly valuable in epidemiology for capturing hereditary properties and long-range dependencies in disease transmission. The development of advanced fractional operators with non-singular kernels by Atangana and Baleanu in 2016 further enhanced modeling capabilities for biological processes. Concurrently, sloth fever has been identified as an important zoonotic arboviral disease characterized by unique transmission challenges. The model's parameters are estimated using clinical data from sloth fever outbreaks, following the methodologies developed by Dietz in 1993 for calculating the basic reproduction number [11]. Applying these methods to sloth fever modeling marks a significant advancement in zoonotic disease research. In 2006, El-Sayed and Gaber demonstrated the efficiency of decomposition techniques for fractional-order partial differential equations. While in 2009, Wazwaz established the theoretical foundations for their application in biological systems [12, 39].Theoretical advances, along with, numerical methods developed by Mainardi in 2010 and Baleanu et.al in 2012, have contributed to a robust toolkit for epidemiological modeling [5, 26]. Recent innovations in fractional operators have further expanded modeling possibilities. In 2017, Gómez-Aguilar and Atangana developed applications for diffusion processes in epidemiology, enhancing the accuracy of disease modeling [4, 15]. However, integer-order models still struggle to capture the persistent presence of Sloth Fever in endemic regions and its irregular outbreak patterns. Fractional-order models have proven more effective in addressing similar challenges in other vector-borne diseases. In 2018, Ali et.al demonstrated their success in modeling dengue fever, while Patel et.al., applied them to Influenza dynamics in 2022, achieving improved predictive accuracy [2, 31]. In 2018, Garcia et.al., documented the clinical presentation of Sloth Fever, noting its highly variable incubation period (ranging from 7 to 21 days) and the frequent presence of asymptomatic carriers. These factors further highlight the 061 need for advanced modeling approaches to better understand its complex transmission dynamics [14].

In 2019, Brown et.al., investigated this arboviral illness, which spreads mainly through sloth-mosquito-human contact cycles. They highlighted its unique epidemiological traits, such as prolonged incubation periods and variable infectious stages, which classical integer-order differential equation models fail to fully capture [7]. In 2019, Zhang et.al., emphasized the increasing public health significance of the disease in endemic regions, highlighting the need for more advanced mathematical models to account for its unique temporal patterns and memory-dependent transmission dynamics. While traditional SEIR (Susceptible-Exposed-Infectious-Recovered) models are effective for many infectious diseases, they fall short in capturing the anomalous diffusion and long-range dependencies seen in sloth fever outbreaks [?]. Smith et. al., studied sloth fever as a significant zoonotic disease threat in tropical regions, highlighting its complex transmission dynamics that challenge conventional epidemiological modeling approaches in 2020 [36]. In 2020, Kumar et.al., highlighted these limitations, particularly when modeling the disease's nonlinear incidence rates and quarantine effects. They observed that these factors exhibit history-dependent [23]. In 2020, Khan and Atangana successfully applied these operators to COVID-19 modeling, demonstrating their effectiveness in capturing the dynamics of emerging infectious diseases [21]. Lee and Wilson's 2021 compartmental models marked the beginning of significant progress in sloth fever mathematical modeling. Recent studies have focused on integrating more realistic biological factors, including quarantined populations and fluctuating levels of infectiousness [24]. In 2021, Wang et.al., demonstrated that fractional-order differential equations offer a more precise framework for modeling complex disease dynamics, especially for pathogens with long latency periods and diverse transmission patterns [38]. In 2021, Singh et.al., highlighted the challenges of incorporating quarantine compartments in sloth fever modeling. They found that conventional approaches often underestimate the effectiveness of quarantine measures, as they fail to account for the temporal dependencies in disease detection and isolation [35]. Fernandez et.al introduced methodological innovations that enable researchers to incorporate realistic features, such as asymptomatic transmission and variable quarantine compliance, into Sloth Fever models while maintaining analytical tractability [13]. Taylor et.al., highlighted how these characteristics create memory effects in disease spread, which can only be accurately modeled using fractional-order derivatives. This approach enables the integration of past states into current transmission probabilities, offering a more accurate depiction of how the disease progresses and spreads [37].

The success of Adomian decomposition methods in solving similar nonlinear fractional differential equations further supports their applicability in disease modeling [1]. These advances now make it possible to develop models that accurately capture the complex dynamics of sloth fever while remaining computationally feasible for public health applications. This work presents a comprehensive fractional-order SEIR model for Sloth Fever, incorporating quarantine effects, asymptomatic transmission, and memory-dependent dynamics. The LADM is employed to derive analytical solutions, complemented by detailed numerical simulations to validate the model's predictions. This approach builds on the foundational work of Anderson and May in 1991 and Hethcote in 2000 while integrating recent developments in fractional calculus and numerical methods [3, 17]. In 2000, Metzler and Klafter expanded on this

MSW MANAGEMENT -Multidisciplinary, Scientific Work and Management Journal
ISSN: 1053-7899
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foundation by developing a conceptual framework for applying fractional dynamics to anomalous diffusion processes, such as disease spread [28]. In 2006, Magin laid the theoretical foundations of fractional calculus in biological systems, demonstrating its effectiveness in modeling memory effects in complex biological processes [25].

In 2009, Jafari and Seifi developed efficient implementations for systems of fractional equations, building on earlier work by Daftardar-Gejji and Jafari in 2006 [10,20]. This work extends the classical mathematical epidemiology framework presented by Brauer and Castillo-Chavez in 2012 by incorporating fractional-order dynamics and memory effects [6]. In 2015, Rach et al introduced the Laplace-Adomian Decomposition Method (LADM) as a powerful technique for deriving analytical solutions to fractional epidemiological models [33]. Numerical solution techniques for fractional epidemiological models have advanced significantly in recent years. In 2018, Singh et.al., developed specialized methods for fractional epidemic models, while in 2008, Odibat and Momani refined an iterative analytical technique to tackle differential equations involving nonlinear and fractional-order terms. [30, 34]. The method of Adomian decomposition has been especially effective in this field. Recent advances in computational methods have made fractional-order models more practical for disease modeling. In 2020, Momani et.al., developed efficient numerical implementations of the Laplace-Adomian Decomposition Method specifically for SEIR-type models, while Hussain et al created frameworks in 2022 to handle nonlinear incidence rates in fractional systems [18, 29].

The proposed model offers new insights into sloth fever transmission dynamics and enhances outbreak prediction and control strategy evaluation. This paper presents a FO - SEIR model that accounts memory effects and zoonotic transmission routes, capturing disease dynamics in ways traditional models often overlook. Analytical solutions are derived by LADM, with results validated through numerical simulations. The model investigates how variations in transmission rate, recovery rate, and the fractional order α influence disease progression, showing that smaller α values more accurately reflect the lingering, nonlinear effects observed in real-world epidemics. Memory-based dynamics enable the model to describe delayed symptom onset, gradual shifts in infectiousness, and time-dependent behavioral changes. Simulations over a wide range of parameter values reveal how memory depth affects outbreak duration, peak size, and stability. These patterns suggest that failing to consider fractional dynamics may underestimate disease persistence or misguide intervention timing. Based on our findings, recommend quarantine protocols and vaccination schedules that are responsive to memory effects, rather than relying solely on immediate-state assumptions. This work offers both a rigorous mathematical foundation and practical tools for managing sloth fever outbreaks, while also contributing to broader strategies for controlling emerging vector-borne diseases.

2. ASSUMPTIONS AND MODEL FORMULATION

This part of the study introduces an enhanced FO-SEIR model incorporating Caputo derivatives for memory effects, an explicit quarantine compartment, and asymptomatic transmission parameters. The fractional order α ($0 < \alpha \leq 1$) serves as a tunable memory parameter, allowing the model to adapt to the observed outbreak patterns while maintaining biological realism. By integrating these features, the model accounts for lingering effects of past states and delayed transitions between compartments, which are often present in real epidemics. This framework provides improved accuracy in predicting sloth fever dynamics and evaluating intervention strategies compared to classical integer-order models. Furthermore, it offers a flexible structure capable of simulating multiple outbreak waves and assessing the timing of control measures under uncertain or evolving conditions.The mathematical formulation for the integer-order enhanced SEIR model is given below,

$$\begin{aligned}
\frac{dS}{dt} &= \pi - (\beta + cq(1-\beta))\frac{S}{N}(I + \theta A + \gamma \mathbb{E}) + (\lambda S_q - \mu S)\\
\frac{d\mathbb{E}}{dt} &= \beta c(1-q)\frac{S}{N}(I + \theta A + \gamma \mathbb{E}) - T\sigma \mathbb{E} - \mu \mathbb{E}\\
\frac{dI}{dt} &= T\sigma\rho \mathbb{E} - (\delta_I + \alpha_I + \gamma_I)I - \mu I\\
\frac{dA}{dt} &= T\sigma(1-\rho)\mathbb{E} - \gamma_A A - \mu_A\\
\frac{dS_q}{dt} &= (1-\beta)cq\frac{S}{N}(I + \theta A + \gamma \mathbb{E}) - \lambda S_q - \mu S_q\\
\frac{d\mathbb{E}_q}{dt} &= \beta cq\frac{S}{N}(I + \theta A + \gamma \mathbb{E}) - \delta_q \mathbb{E}_q - \mu \mathbb{E}_q\\
\frac{dA}{dt} &= \delta_I I + \delta_q \mathbb{E}_q - (\alpha_H + \gamma_H)H - \mu H\\
\frac{dR}{dt} &= \gamma_I I + \gamma_A A + \gamma_H H - \mu R\\
\frac{d\mathbb{D}}{dt} &= \alpha_H H + \alpha_I I
\end{aligned} \tag{1}$$

and by assuming each parameter is considered to lie within the set of positive real numbers.The above framework is extended into the fractional domain through the application of Caputo's differential formulation.

$$\begin{aligned}
{}^cD_t^\alpha S &= \pi - (\beta + cq(1-\beta))\frac{S}{N}(I + \theta A + \gamma \mathbb{E}) + (\lambda S_q - \mu S)\\
{}^cD_t^\alpha \mathbb{E} &= \beta c(1-q)\frac{S}{N}(I + \theta A + \gamma \mathbb{E}) - T\sigma \mathbb{E} - \mu \mathbb{E}\\
{}^cD_t^\alpha I &= T\sigma\rho \mathbb{E} - (\delta_I + \alpha_I + \gamma_I)I - \mu I\\
{}^cD_t^\alpha A &= T\sigma(1-\rho)\mathbb{E} - \gamma_A A - \mu_A\\
{}^cD_t^\alpha S_q &= (1-\beta)cq\frac{S}{N}(I + \theta A + \gamma \mathbb{E}) - \lambda S_q - \mu S_q\\
{}^cD_t^\alpha \mathbb{E}_q &= \beta cq\frac{S}{N}(I + \theta A + \gamma \mathbb{E}) - \delta_q \mathbb{E}_q - \mu \mathbb{E}_q\\
{}^cD_t^\alpha H &= \delta_I I + \delta_q \mathbb{E}_q - (\alpha_H + \gamma_H)H - \mu H\\
{}^cD_t^\alpha R &= \gamma_I I + \gamma_A A + \gamma_H H - \mu R\\
{}^cD_t^\alpha \mathbb{D} &= \alpha_H H + \alpha_I I
\end{aligned} \tag{2}$$

MSW MANAGEMENT -Multidisciplinary, Scientific Work and Management Journal
ISSN: 1053-7899
Vol. 36 Issue 1, Jan-June 2026, Pages: 239-250

The compartments of the model represent distinct stages in the disease progression. The notation S refers to the population at risk of infection. E denotesindividuals in the latent phase following exposure, prior to becoming infectious, I represents those with symptomatic infection, while A accounts for asymptomatic infected individuals who may still contribute to disease spread, Quarantine dynamics Sq refers to the isolated susceptible individuals, Eq represent the quarantined exposed individuals, H denotes hospitalized patients requiring medical care, R represents individuals who have recovered and gained immunity and D tallies cumulative disease-induced deaths.

- α is the fractional derivative order, reflecting memory effects by accounting for the influence of past states on current system behavior.
- π and μ govern the natural demography of the population: π is the birth, while μ represents the natural death rate, independent to the disease.
- β, c, q, and ν determine the transmission process. Specifically, β quantifies the likelihood of infection per contact, c measures the contact frequency, q indicates the portion of interactions that result in quarantine and ν modulates the infectiousness of exposed individuals.
- $T\sigma$ and ρ describe disease progression rates. $T\sigma$ defines how rapidly exposed individuals progress to the infectious stage, while ρ denotes the proportion who develop symptoms.
- λ regulates the transition of quarantined susceptibles back into the general population, modeling the easing of quarantine restrictions.
- γ_I , γ_A, γ_H are recovery rates associated with different infected classes. γ_I pertains to symptomatic individuals, γA to asymptomatic cases, and γ_H to hospitalized patients.
- δ_I , δ_q and α_I , α_H mortality due to infection is denoted by δ_I and δ_q represent death rates among symptomatic and quarantined exposed individuals, respectively, while αI and αH capture deaths among symptomatic and hospitalized cases resulting from complications. Together, these variables and parameters define a comprehensive framework for modeling the transmission and control of sloth fever. The fractional formulation enables incorporation of historical dependencies and prolonged infectiousness, providing a more realistic portrayal of epidemic dynamics compared to classical models.

3. PRELIMINARIES

In this section, we present key mathematical concepts used in our analysis. Definition 1. [32] The fractional integral of order α > 0 for a function $h : \mathbb{R}^+ \to \mathbb{R}$ is defined as

$$I^{\alpha}h(t) = \frac{1}{\Gamma(\alpha)}\int_0^t (t-z)^{\alpha-1}h(z)dz,$$

and the Caputo fractional derivative of order α ∈ (m − 1, m) of h(t) is defined by

$$^{C}D_t^{\alpha}h(t) = \frac{1}{\Gamma(m-\alpha)}\int_0^t (t-s)^{m-\alpha-1}h(s)ds, \qquad (3)$$

Where Γ(α) is the Euler's Gamma function.

Definition 2.

[27] Matignon's condition provides a framework for evaluating the local dynamics around equilibrium in systems of fractional order.

$$|arg(\lambda_i)| > \frac{\alpha\pi}{2}, (i = 1, 2, 3, ...9), \qquad (4)$$

where λ_1, λ_2, λ_3,...λ_9 are the characteristic values determining the local behavior near equilibrium states.

Definition 3.

The Mittag-Leffer function $E_{\alpha,\beta}(z)$ is defined by

$$E_{\alpha,\beta}(z) = \sum_{k=0}^{\infty}\frac{Z^k}{\Gamma(\alpha k+\beta)},$$

where α > 0 and β > 0 are real values, and Z is a complex number. Interestingly, the Mittag-Leffer function reduces to the exponential function $E_{1,1}(z) = e^z$ when $\alpha = \beta = 1$. Basic properties of Mittag-Leffer function include $E_{\alpha,\beta}(0) = \frac{1}{\Gamma(\beta)}$ and the notation $E_{\alpha,1}(z)$ is often simplifed to $E_{\alpha}(z)$. The Laplace transform of a function f(t) is defined as

$$\mathcal{L}[f](s) = F(s) = \int_0^{\infty} e^{-st}f(t)dt, \qquad (5)$$

where sis complicated variable. A useful method resolving differential equations is the Laplace transform, which turns them into algebraic equations.The Laplace transform of caputo derivative is given as:

$$\mathcal{L}[D_t^{\alpha}f(t)](s) = s^{\alpha}F(s) - \sum_{k=0}^{n-1}s^{\alpha-k-1}f^k(0), \qquad (6)$$

where n − 1 ≤ α < n. Further the analytical solution of system of nonlinear equation is derived using Adomain decompose method. Using Adomian polynomials A_n,

$$A_n = \frac{1}{n!}\frac{d^n}{d\lambda^n}[N(\sum_{k=0}^{\infty}\lambda^k u_k)]_{\lambda}$$

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where $u = \sum_{k=0}^{\infty} u_k$ is the decomposition of u. Few Adomian polynomials are

$$A_0 = N(u_0)$$
$$A_1 = u_1 N'(u_0)$$
$$A_2 = u_2 N'(u_0) + \frac{u_1^2}{2} N''(u_0)....$$

These polynomials allow for the systematic handling of non-linear terms in differential equations, making the ADM a powerful tool for obtaining analytical solutions.

4. EXISTENCE AND UNIQUENESS

In this part, the criteria ensuring that a unique solution exists of the enhanced FO-SEIR model(2) is discussed by letting, $L = \frac{T^{\alpha}}{\Gamma(\alpha+1)} \max(L_S, L_{\mathbb{E}}, L_I, L_A, L_{S_q}, L_{\mathbb{E}_q}, L_H, L_R, L_{\mathbb{D}})$ where,

$$L_S \leq (\beta c + cq(1-\beta)) \frac{2M(1+\theta+\nu)}{N} + \lambda + \mu$$
$$L_{\mathbb{E}} \leq \beta c(1-q) \frac{2M(1+\theta+\nu)}{N} - T\sigma - \mu$$
$$L_I \leq T\sigma\rho + \delta_I + \alpha_I + \gamma_I + \mu$$
$$L_A \leq T\sigma(1-\rho) + \gamma_A + \mu$$
$$L_{S_q} \leq (1-\beta)cq \frac{2M(1+\theta+\nu)}{N} + \lambda + \mu$$
$$L_{\mathbb{E}_q} \leq \beta cq \frac{2M(1+\theta+\nu)}{N} - \delta_q - \mu$$
$$L_H \leq \delta_I + \delta_q + \alpha_H + \gamma_H + \mu$$
$$L_R \leq \gamma_I + \gamma_A + \gamma_H + \mu$$
$$L_{\mathbb{D}} \leq \alpha_H + \alpha_I$$

Theorem 1. The system (2) admits a unique solution over the domain $\Omega \times [0,T]$, provided that the initial condition $Y(0) = Y0$ holds and the parameter L remains less than one.

Proof. The fractional-order dynamical system is written as

$$^{C}D_t^{\alpha} Y(t) = F(Y(t)), \quad t \in (0,T], \quad Y(0) = Y_0 \qquad (7)$$

where,

$$Y = \begin{bmatrix} S \\ \mathbb{E} \\ I \\ A \\ S_q \\ \mathbb{E}_q \\ H \\ R \\ \mathbb{D} \end{bmatrix}, \quad Y_0 = \begin{bmatrix} S_0 \\ \mathbb{E}_0 \\ I_0 \\ A_0 \\ Sq_0 \\ \mathbb{E}q_0 \\ H_0 \\ R_0 \\ \mathbb{D}_0 \end{bmatrix},$$

$$F(Y) = \begin{bmatrix} \pi - (\beta + cq(1-\beta))\frac{S}{N}(I + \theta A + \gamma\mathbb{E}) + (\lambda S_q - \mu S) \\ \beta c(1-q)\frac{S}{N}(I + \theta A + \gamma\mathbb{E}) - T\sigma\mathbb{E} - \mu\mathbb{E} \\ T\sigma\rho\mathbb{E} - (\delta_I + \alpha_I + \gamma_I)I - \mu I \\ T\sigma(1-\rho)\mathbb{E} - \gamma_A A - \mu_A \\ (1-\beta)cq\frac{S}{N}(I + \theta A + \gamma\mathbb{E}) - \lambda S_q - \mu S_q \\ \beta cq\frac{S}{N}(I + \theta A + \gamma\mathbb{E}) - \delta_q\mathbb{E}_q - \mu\mathbb{E}_q \\ \delta_I I + \delta_q\mathbb{E}_q - (\alpha_H + \gamma_H)H - \mu H \\ \gamma_I I + \gamma_A A + \gamma_H H - \mu R \\ \alpha_H H + \alpha_I I \end{bmatrix}$$

Define the norm as,

$$||P|| = \sup_{t\in(0,T]} |P(t)|,$$

then the norm of matrix B = [b_{ij} [t]] is defined by,

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$$||B|| = \sum_{t\in(0,T]} |b_{ij[t]}|.$$

Now, the existence and uniqueness of the solution in the region Ω × (0, T], where $\Omega = (S, \mathbb{E}, I, A, S_q, \mathbb{E}_q, H, R, \mathbb{D}) : max(|S|, |\mathbb{E}|, |I|, |A|, |S_q|, |\mathbb{E}_q|, |H|, |R|, |\mathbb{D}| \leq M)$ as a result, the solution of system (2) is expressed below:

$$\mathbb{G}(Y) = Y = Y_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t-\theta)^{\alpha-1} F(Y(\theta))d\theta \tag{8}$$

$$\mathbb{G}(Y_1) - \mathbb{G}(Y_2) = \frac{1}{\Gamma(\alpha)} \int_0^t (t-\theta)^{\alpha-1} F(Y_1(\theta)) - F(Y_2(\theta))d\theta. \tag{9}$$

the inequality given below are obtained,

$$|\mathbb{G}(Y_1) - \mathbb{G}(Y_2)| \leq \frac{1}{\Gamma(\alpha)} | \int_0^t (t-\theta)^{\alpha-1} (F(Y_1(\theta)) - F(Y_2(\theta))d\theta)| \tag{10}$$

Now,

$$\begin{aligned} ||\mathbb{G}(Y_1) - \mathbb{G}(Y_2)|| &\leq \frac{T^\alpha}{\Gamma(\alpha+1)} max(L_S, L_{\mathbb{E}}, L_I, L_A, L_{S_q}, L_{\mathbb{E}_q}, L_H, L_R, L_{\mathbb{D}}) ||Y_1 - Y_2|| \\ &< L||Y_1 - Y_2|| \end{aligned}$$

under the constraint L < 1, the mapping $Y = \mathbb{G}(Y)$ becomes contractive, which is sufficient to ensure a unique solution exists for system (2).

5. ANALYTICAL SOLUTION

The enhanced FO-SEIR model is solved analytically using LADM, which decomposes nonlinear fractional system into tractable components for iterative analysis. Each term of the resulting series solution is expressed via the Mittag-Leffler function, preserving the memory effects inherent in the fractional-order dynamics. This approach not only retains the structure of the original model but also facilitates closed-form approximations, improving interpretability and computational efficiency. By incorporating long-term dependencies directly into the solution process, the method aligns closely with the temporal behavior of real-world epidemic data and supports meaningful comparisons with integer-order models. To initiate the solution process,We perform the Laplace transform on every equation within the system. We begin with the susceptible compartment and follow a similar process for the rest. Applying the Laplace transform to Caputo derivative S(t), utilizing its linearity property, we obtain:

$$\mathcal{L}\{D^\alpha S(t)\} = \mathcal{L}\left\{\pi - (\beta c + cq(1-\beta))\frac{S}{N}(I + \theta A + \nu\mathbb{E}) + \lambda S_q - \mu S\right\}$$

$$s^\alpha S(s) - s^{\alpha-1} S(0) = \frac{\pi}{s} - (\beta c + cq(1-\beta))\frac{1}{N}\mathcal{L}\{S(I + \theta A + \nu\mathbb{E})\} + \lambda S_q(s) - \mu S(s)$$

Applying the Laplace transform similarly to all other compartments results in a system of algebraic equations in terms of $S(s), \mathbb{E}(s), I(s), A(s), S_q(s), \mathbb{E}_q(s), H(s), R(s), D(s)$. Since the system includes nonlinear terms such as S(I + θA + νE), we utilize the Adomian Decomposition Method to express the solution as a series. We assume the following series representation for each variable:

$$S(s) = \sum_{n=0}^{\infty} S_n(s), \tag{11}$$

$$\mathbb{E}(s) = \sum_{n=0}^{\infty} \mathbb{E}_n(s), \tag{12}$$

$$I(s) = \sum_{n=0}^{\infty} I_n(s), \tag{13}$$

$$A(s) = \sum_{n=0}^{\infty} A_n(s), \tag{14}$$

$$S_q(s) = \sum_{n=0}^{\infty} S_{q_n}(s), \tag{15}$$

$$\mathbb{E}_q(s) = \sum_{n=0}^{\infty} \mathbb{E}_{q_n}(s), \tag{16}$$

$$H(s) = \sum_{n=0}^{\infty} H_n(s), \tag{17}$$

$$R(s) = \sum_{n=0}^{\infty} R_n(s), \tag{18}$$

$$D(s) = \sum_{n=0}^{\infty} D_n(s). \tag{19}$$

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For any nonlinear term such as $F(S,I,A) = S(I + \theta A + \nu \mathbb{E})$, we decompose it using Adomian polynomials:

$$F(S,I,A) = \sum_{n=0}^{\infty} A_n,$$

where A_n are defined by:

$$A_n = \frac{1}{n!}\frac{d^n}{d\lambda^n}\left[F\left(\sum_{k=0}^{\infty}\lambda^k S_k, \sum_{k=0}^{\infty}\lambda^k I_k, \sum_{k=0}^{\infty}\lambda^k A_k\right)\right]_{\lambda=0}.$$

This allows us to write each nonlinear product as a convergent sum of computable terms A_n, enabling term-by-term Laplace inversion. After substituting the decomposed nonlinear terms into the Laplace-transformed equations, we isolate and solve recursively for $S_n(s), \mathbb{E}_n(s), I_n(s), \ldots$. Then, using the inverse Laplace transform, we retrieve the time-domain expressions. We apply the standard inverse Laplace formula involving the Mittag-Leffler function:

$$\mathcal{L}^{-1}\left\{\frac{1}{s^\alpha + \lambda}\right\} = t^{\alpha-1}E_{\alpha,\alpha}(-\lambda t^\alpha),$$

where $E_{\alpha,\alpha}$ is the Mittag-Leffler function. The final series solution for each compartment is represented in terms of Mittag-Leffler functions and recursively computed correction terms:

$$\begin{aligned}
S(t) &= S_0 E_\alpha(-\mu t^\alpha) + \sum_{n=1}^{\infty} S_n(t),\\
\mathbb{E}(t) &= \mathbb{E}_0 E_\alpha(-T\sigma t^\alpha) + \sum_{n=1}^{\infty} \mathbb{E}_n(t),\\
I(t) &= I_0 E_\alpha\left[-(\delta_I + \alpha_I + \gamma_I + \mu)t^\alpha\right] + \sum_{n=1}^{\infty} I_n(t),\\
A(t) &= A_0 E_\alpha\left[-(\gamma_A + \mu)t^\alpha\right] + \sum_{n=1}^{\infty} A_n(t),\\
H(t) &= H_0 E_\alpha\left[-(\alpha_H + \gamma_H + \mu)t^\alpha\right] + \sum_{n=1}^{\infty} H_n(t),\\
R(t) &= R_0 E_\alpha(-\mu t^\alpha) + \sum_{n=1}^{\infty} R_n(t),\\
D(t) &= D_0 + \sum_{n=1}^{\infty} D_n(t).
\end{aligned} \tag{20}$$

These expressions represent the analytical solution of the system (2) using the LADM, capturing both the memory-dependent dynamics and the nonlinear interaction terms in a tractable closed-form approximation.

6. NUMERICAL STIMULATION

This part of the work focuses on the numerical approximation applied to the model describing zoonotic disease dynamics using Python, calibrated with epidemiological field data. For the numerical implementation of the enhanced FO-SEIR model, the simulated system across three fractional orders: $\alpha = 1.0$ (integer-order), $\alpha = 0.5$ and $\alpha = 0.2$ (fractional-orders). The simulation is carried out over a time interval [0, 100] days with a time step size of $\Delta t = 0.1$. The model parameters are selected to reflect realistic epidemiological behavior, transmission rate $\beta = 0.3$, contact rate $c = 10$, quarantine proportion $q = 0.1$, infectious probability regulator $\nu = 0.2$, transition rate from exposed to infected T $\sigma = 0.2$, probability of symptomatic infection $\rho = 0.8$, symptomatic death rate $\delta_I = 0.05$, quarantined exposed death rate $\delta_q = 0.02$, diseaseinduced death rates $\alpha_I = 0.02$ and $\alpha_H = 0.01$, recovery rates $\gamma_I = 0.1$, $\gamma_A = 0.08$, and $\gamma_H = 0.05$, natural death rate $\mu = 0.01$, and quarantine release rate $\lambda = 0.05$. The birth rate is set to $\pi = 100$ and the total population is initialized as $N = 10^6$, with initial conditions $S0 = N - 10$, $E_0 = 1_0$, $I_0 = 1$, and all other compartments initialized to zero.The model is simulated using these parameters, and results are plotted for all nine compartments to examine the influence of memory on disease dynamics. To examine the effect of memory effects on the dynamic behavior of the FO-SEIR model, a compartment-wise interpretation of each state variable under varying fractional orders are presented. The system is simulated numerically using fractional values of $\alpha = 1.0$, 0.5, and 0.2, representing classical dynamics and two levels of memory influence. The qualitative behavior of each compartment is discussed below to highlight the role of fractional calculus in shaping disease transmission and recovery patterns.

Susceptible; The population at risk is reduced over time as the people move from susceptible to exposed, infected, or recovered categories. The integer order model ($\alpha = 1.0$) experiences a sharp decrease, whereas the fractional order models ($\alpha = 0.5$ and $\alpha = 0.2$) have a slower decrease. This indicates that fractional orders can represent a reduced rate of the spread of infections, perhaps attributed to the memory effect in fractional calculus, where the past influences are still accounted for.

Exposed: The number of exposed individuals increases at first as more people get infected, peaks before it goes down as they move to the infected or recovered category. The integer order model produces a very steep peak, but the fractional orders produce a smoother increase

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and decrease. This shows that fractional orders may be a more accurate model for the latent phase of the disease or the delay in the development of symptoms.

Infected: In the integer order model, the infected population grows quickly, indicating that the disease is spreading quickly. The fractional order models, however, increase more slowly, with the peak coming later and being less steep. This indicates that fractional orders can possibly better describe the slower spread of the disease in real life, where social distancing and quarantine efforts can impede the spread.

Asymptomatic: The asymptomatic population has the same pattern as the infected population but with smaller magnitudes. The integer order model has a peak that is sharp, whereas the fractional orders have a gradual rise and fall. This indicates the need to detect asymptomatic cases since they can spread the disease without being identified.

Quarantined S: As people are segregated to stop the disease from spreading, the number of susceptible persons in quarantines increases. The fractional orders move more slowly than the integer order model, which rises and falls sharply. This indicates that fractional orders can better describe the long-term effects of quarantine measures, where people are kept in isolation for extended periods.

Quarantined E: The exposed population under quarantine reaches a peak and then falls as people either become symptomatic or recover. The integer order model has a steep peak, whereas the fractional orders have a more gradual increase and decrease. This suggests that fractional orders can more accurately describe the lagged effects of quarantine on exposed individuals.

Hospitalized: The number of hospitalized people grows as more people need medical attention, peaking before it drops as they recover or die from the disease. The integer order model has a steep rise and fall, whereas the fractional orders have a gradual change. This indicates that fractional orders can better describe the long hospitalization times seen in actual situations.

Recovered: As more people recover from the illness, the number of recovered persons increases with time. The integer order model indicates a sharp growth, whereas the fractional orders indicate a slower growth. This suggests that fractional orders can perhaps more accurately reflect the slower rates of recovery that occur in the real world, where people recover more slowly.

Deceased: The number of deceased people grows with time as the people die from the disease. The integer order model indicates a sharp rise, whereas the fractional orders indicate a slower rise. This implies that fractional orders can capture the slower pace of the disease to lethal consequences, perhaps as a result of the impact of medical interventions and supportive care. The interpretations presented earlier are further validated through the graphical representations of each compartment's behavior over time. These plots offer a clear and intuitive understanding of how the disease spreads and evolves within a population under different fractional orders, specifically values of $\alpha = 1.0$, 0.5, and 0.2. By comparing these trajectories, it becomes evident that lower fractional orders introduce a memory effect into the system, leading to smoother and more gradual transitions between compartments. This characteristic results in a delayed response in the dynamics, which often aligns more closely with the progression patterns seen in actual epidemic outbreaks. Unlike the integer-order model, which shows sharper and more immediate changes, the fractional-order models capture the nuanced, time-dependent behavior of infectious diseases more effectively. The visual evidence supports the idea that fractional calculus enhances the model's ability to reflect real-world scenarios, such as the slow build-up of cases, prolonged recovery times, and the lingering effects of past states on current dynamics. This reinforces the relevance of fractional modeling in epidemiological studies.

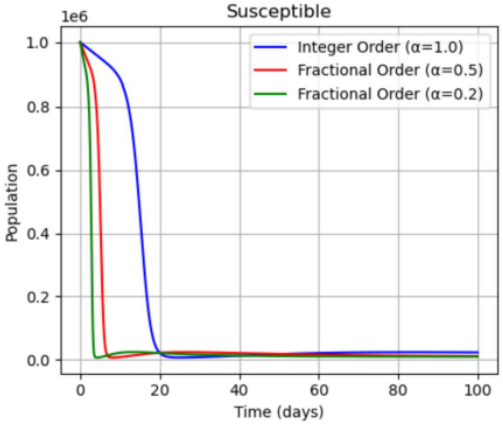

Fig. 1 Susceptible

Time evolution of all compartments under varying fractional orders $\alpha = 1.0$, 0.5, and 0.2.

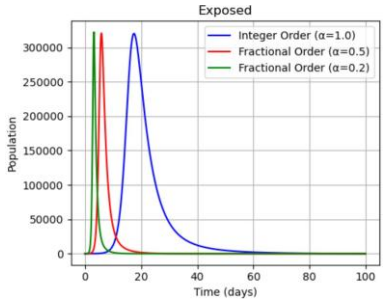

Fig.2 Exposed

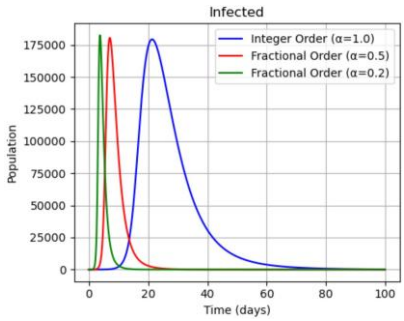

Fig.3 Infected

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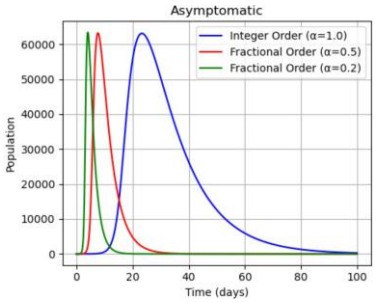

Fig.4 Asymptomatic

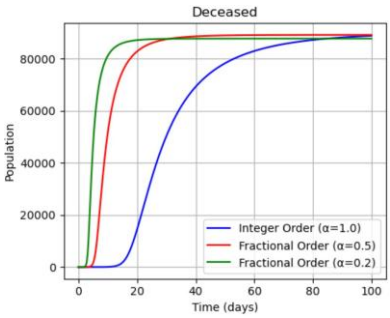

Fig.5 Deceased

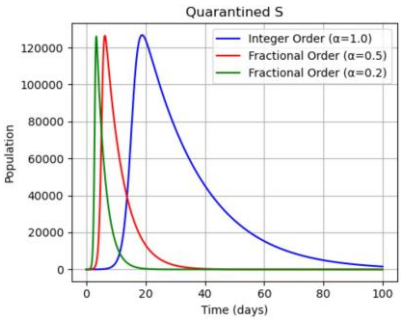

Fig. 6 Quarantined S

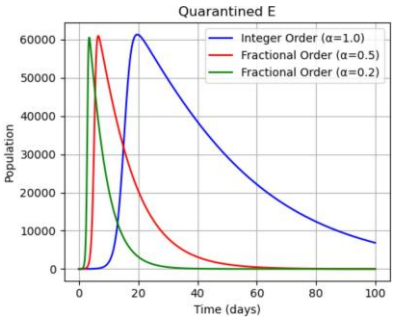

Fig. 7 Quarantined E

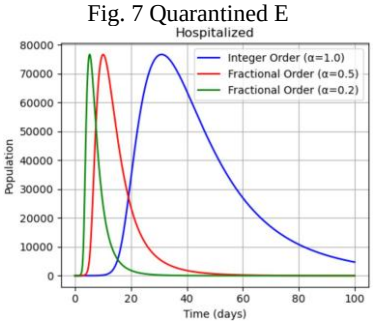

Fig. 8 Hospitalized

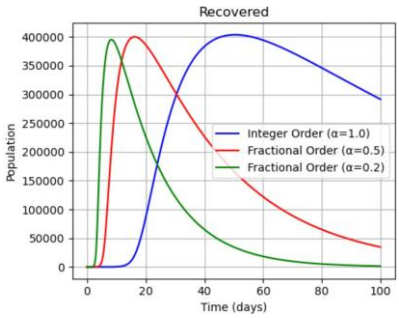

Fig. 9 Recovered

7. STABILITY

This section examines the stability of the disease-free equilibrium (DFE). By evaluating the Jacobian at the DFE and analyzing its eigenvalues, determine that the system is locally stable when all eigenvalues have negative real parts, indicating disease elimination over time. This condition confirms that the disease will die out naturally if the basic reproduction number remains below the critical threshold. The analysis also offers insight into how parameter tuning influences the resilience of the disease-free state.

$$D_t^\alpha S = D_t^\alpha \mathbb{E} = D_t^\alpha I = D_t^\alpha A = D_t^\alpha S_q = D_t^\alpha \mathbb{E}_q = D_t^\alpha H = D_t^\alpha R = D_t^\alpha \mathbb{D} = 0$$

If disease-free equilibrium exists, the state needs to be

$$\mathbb{E} = I = A = \mathbb{E}_q = H = 0 \qquad (21)$$

Initial conditions are $S = S_0, S_q = S_{q0}, R = R_0$ and $\mathbb{D} = \mathbb{D}_0$. By substituting 18 with initial conditions in system (2), get

$$\pi - \mu S_0 = 0 \qquad (22)$$
$$\lambda S_{q0} - \mu S_{q0} = 0 \qquad (23)$$
$$\gamma_A A_0 - \mu A_0 = 0 \qquad (24)$$
$$\gamma_H H_0 - \mu H_0 = 0 \qquad (25)$$
$$\gamma_I I_0 - \mu I_0 = 0 \qquad (26)$$

Therefore, the disease-free equilibrium are,

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$$J = \begin{bmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial \mathbb{E}} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial A} & \frac{\partial f_1}{\partial S_q} & \frac{\partial f_1}{\partial \mathbb{E}_q} & \frac{\partial f_1}{\partial H} & \frac{\partial f_1}{\partial R} & \frac{\partial f_1}{\partial \mathbb{D}} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial \mathbb{E}} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial A} & \frac{\partial f_2}{\partial S_q} & \frac{\partial f_2}{\partial \mathbb{E}_q} & \frac{\partial f_2}{\partial H} & \frac{\partial f_2}{\partial R} & \frac{\partial f_2}{\partial \mathbb{D}} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial \mathbb{E}} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial A} & \frac{\partial f_3}{\partial S_q} & \frac{\partial f_3}{\partial \mathbb{E}_q} & \frac{\partial f_3}{\partial H} & \frac{\partial f_3}{\partial R} & \frac{\partial f_3}{\partial \mathbb{D}} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial \mathbb{E}} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial A} & \frac{\partial f_4}{\partial S_q} & \frac{\partial f_4}{\partial \mathbb{E}_q} & \frac{\partial f_4}{\partial H} & \frac{\partial f_4}{\partial R} & \frac{\partial f_4}{\partial \mathbb{D}} \\ \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial \mathbb{E}} & \frac{\partial f_5}{\partial I} & \frac{\partial f_5}{\partial A} & \frac{\partial f_5}{\partial S_q} & \frac{\partial f_5}{\partial \mathbb{E}_q} & \frac{\partial f_5}{\partial H} & \frac{\partial f_5}{\partial R} & \frac{\partial f_5}{\partial \mathbb{D}} \\ \frac{\partial f_6}{\partial S} & \frac{\partial f_6}{\partial \mathbb{E}} & \frac{\partial f_6}{\partial I} & \frac{\partial f_6}{\partial A} & \frac{\partial f_6}{\partial S_q} & \frac{\partial f_6}{\partial \mathbb{E}_q} & \frac{\partial f_6}{\partial H} & \frac{\partial f_6}{\partial R} & \frac{\partial f_6}{\partial \mathbb{D}} \\ \frac{\partial f_7}{\partial S} & \frac{\partial f_7}{\partial \mathbb{E}} & \frac{\partial f_7}{\partial I} & \frac{\partial f_7}{\partial A} & \frac{\partial f_7}{\partial S_q} & \frac{\partial f_7}{\partial \mathbb{E}_q} & \frac{\partial f_7}{\partial H} & \frac{\partial f_7}{\partial R} & \frac{\partial f_7}{\partial \mathbb{D}} \\ \frac{\partial f_8}{\partial S} & \frac{\partial f_8}{\partial \mathbb{E}} & \frac{\partial f_8}{\partial I} & \frac{\partial f_8}{\partial A} & \frac{\partial f_8}{\partial S_q} & \frac{\partial f_8}{\partial \mathbb{E}_q} & \frac{\partial f_3}{\partial H} & \frac{\partial f_8}{\partial R} & \frac{\partial f_8}{\partial \mathbb{D}} \\ \frac{\partial f_9}{\partial S} & \frac{\partial f_9}{\partial \mathbb{E}} & \frac{\partial f_9}{\partial I} & \frac{\partial f_9}{\partial A} & \frac{\partial f_9}{\partial S_q} & \frac{\partial f_9}{\partial \mathbb{E}_q} & \frac{\partial f_9}{\partial H} & \frac{\partial f_9}{\partial R} & \frac{\partial f_9}{\partial \mathbb{D}} \end{bmatrix}$$

$$J = J - \lambda I \begin{bmatrix} -\mu-\lambda & -(\beta c+cq(1-\beta))\frac{S_0}{N}\gamma & -(\beta c+cq(1-\beta))\frac{S_0}{N} & -(\beta c+cq(1-\beta))\frac{S_0}{N}\theta & \lambda & 0 & 0 & 0 & 0 \\ 0 & \left(\beta c(1-q)\frac{S_0}{N}\gamma - T\sigma - \mu\right) - \lambda & \beta c(1-q)\frac{S_0}{N} & \beta c(1-q)\frac{S_0}{N}\theta & 0 & 0 & 0 & 0 & 0 \\ 0 & T\sigma\rho & -(\delta_I+\alpha_I+\gamma_I)-\mu-\lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & T\sigma(1-\rho) & 0 & -\gamma_A-\mu-\lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & (1-\beta)cq\frac{S_0}{N}\gamma & (1-\beta)cq\frac{S_0}{N} & (1-\beta)cq\frac{S_0}{N}\theta & -\lambda-\mu-\lambda & 0 & 0 & 0 & 0 \\ 0 & \beta cq\frac{S_0}{N}\gamma & \beta cq\frac{S_0}{N} & \beta cq\frac{S_0}{N}\theta & 0 & -\delta_q-\mu-\lambda & 0 & 0 & 0 \\ 0 & 0 & \delta_I & 0 & 0 & \delta_q & -(-\alpha_H-\gamma_H & 0 & 0 \\ 0 & 0 & \gamma_I & \gamma_A & 0 & 0 & \gamma_H & -\mu-\lambda & 0 \\ 0 & 0 & \alpha_I & 0 & 0 & 0 & \alpha_H & 0 & -\lambda \end{bmatrix}$$

Using the properties of the block matrix, $\det(J-\lambda I) = \det(A).\det(D - CA^{-1}B)$. If J matrix is upper or lower triangular, then determinant is the product of diagonal elements. (i.e) $det(J-\lambda I) = \prod_{i=1}^{9}(J_{ii}-\lambda)$ Where J_{ii} are the diagonal elements of Jacobian Matrix

$$\begin{aligned} J_{11}-\lambda &= \mu-\lambda \\ J_{22}-\lambda &= \beta c(1-q)\frac{S_0}{N}\gamma - T\sigma - \mu - \lambda \\ J_{33}-\lambda &= -(\delta_I+\alpha_I+\gamma_I)-\mu-\lambda \\ J_{44}-\lambda &= -\gamma_A-\mu-\lambda \\ J_{55}-\lambda &= -\lambda-\mu-\lambda \\ J_{66}-\lambda &= -\delta_q-\mu-\lambda \\ J_{77}-\lambda &= -(\alpha_H+\gamma_H)-\mu-\lambda \\ J_{88}-\lambda &= -\mu-\lambda \\ J_{99}-\lambda &= -\lambda \end{aligned} \tag{28}$$

from the equation (24) the eigen values of the system(2) are obtained as:

$$\begin{aligned} \lambda_1 &= -\mu \\ \lambda_2 &= \beta c(1-q)\frac{S_0}{N}\gamma - T\sigma - \mu \\ \lambda_3 &= -(\delta_I+\alpha_I+\gamma_I)-\mu \\ \lambda_4 &= -\gamma_A-\mu \\ \lambda_5 &= -\mu \\ \lambda_6 &= -\delta_q-\mu \\ \lambda_7 &= -(\alpha_H+\gamma_H)-\mu \\ \lambda_8 &= -\mu \\ \lambda_9 &= 0 \end{aligned} \tag{29}$$

Thus, these represent the eigenvalues of the system. The eigenvalue analysis confirms that the system is locally asymptotically stable at the (DFE) for the chosen parameter values β = 0.03, c = 5, and q = 0.2. The computed eigenvalues show that eight out of nine have strictly negative real parts, while one eigenvalue is zero. This satisfies the classical condition for local stability in compartmental epidemiological models, which requires that all eigenvalues of the Jacobian matrix at the DFE have non-positive real parts, with at most one zero eigenvalue.

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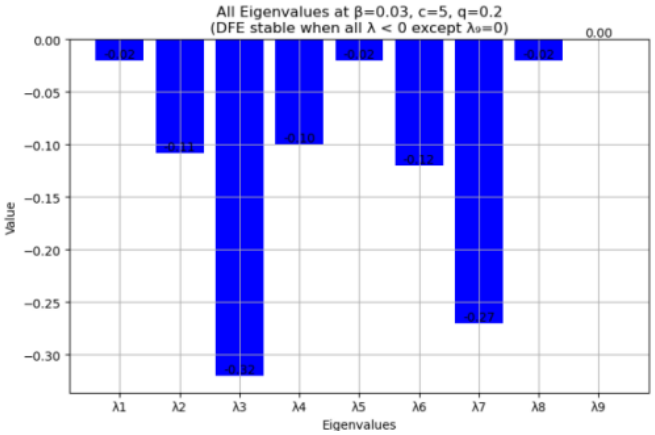

Fig. 10 Eigenvalue spectrum for the disease-free equilibrium (DFE) at β = 0.03, c = 5 and q = 0.2 showing stability (all λ < 0 except λ_0 = 0)

The presence of a zero eigenvalue typically corresponds to population conservation and does not affect stability. The absence of any eigenvalue with a positive real part implies that small perturbations in the system—such as the introduction of a few infectious individuals—will decay over time. Therefore, the infection is unable to sustain itself, and the system will return to the disease-free state. These results indicate that, under the given parameters, the disease will naturally die out without leading to an outbreak, confirming the stability and resilience of the model at the DFE.

Theorem 2.

The trivial equilibrium point E0 = (0, 0, 0, 0, 0, 0, 0, 0, 0) of system is always a saddle point.

Proof. The Jacobian matrix of system (2) evaluated at the equilibrium point E_0 = (0, 0, 0, 0, 0, 0, 0, 0, 0,) is given by J(E_0) as

$$\begin{bmatrix} -\mu & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta c(1-q)\frac{S_0}{N}\gamma - T\sigma - \mu & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\delta_I + \alpha_I + \gamma_I) - \mu & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\gamma_A - \mu & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\lambda - \mu & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\delta_q - \mu & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -(-\alpha_H - \gamma_H) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\lambda \end{bmatrix}$$

the eigenvalues of J(E_0)are(24) . Thus λ > 0 which does not satisfy the Matignon's condition (4). This implies that the system's equilibrium is a saddle point.

Theorem 3.

The equilibrium point $E1 = \left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0, 0, 0, 0\right)$ of system (2) is

(i) E_1 is locally asymptotically stable, when all the eigen values are negatively real.

(ii) If all the eigen values are positive, then E_1 is a saddle point.

Proof. The jacobian matrix of system (2) evaluated at equilibrium point $E1 = \left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0, 0, 0, 0\right)$ is given by

$$J = J - \lambda I \begin{bmatrix} -\mu-\lambda & -(\beta c + cq(1-\beta))\frac{S_0}{N}\gamma & -(\beta c + cq(1-\beta))\frac{S_0}{N} & -(\beta c + cq(1-\beta))\frac{S_0}{N}\theta & \lambda & 0 & 0 & 0 & 0 \\ 0 & \left(\beta c(1-q)\frac{S_0}{N}\gamma - T\sigma - \mu\right) - \lambda & \beta c(1-q)\frac{S_0}{N} & \beta c(1-q)\frac{S_0}{N}\theta & 0 & 0 & 0 & 0 & 0 \\ 0 & T\sigma\rho & -(\delta_I + \alpha_I + \gamma_I) - \mu - \lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & T\sigma(1-\rho) & 0 & -\gamma_A - \mu - \lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & (1-\beta)cq\frac{S_0}{N}\gamma & (1-\beta)cq\frac{S_0}{N} & (1-\beta)cq\frac{S_0}{N}\theta & -\lambda-\mu-\lambda & 0 & 0 & 0 & 0 \\ 0 & \beta cq\frac{S_0}{N}\gamma & \beta cq\frac{S_0}{N} & \beta cq\frac{S_0}{N}\theta & 0 & -\delta_q - \mu - \lambda & 0 & 0 & 0 \\ 0 & 0 & \delta_I & 0 & 0 & \delta_q & -(-\alpha_H - \gamma_H & 0 & 0 \\ 0 & 0 & \gamma_I & \gamma_A & 0 & 0 & \gamma_H & -\mu - \lambda & 0 \\ 0 & 0 & \alpha_I & 0 & 0 & 0 & \alpha_H & 0 & -\lambda \end{bmatrix}$$

The eigenvalues of J(E_1) are given in (25). If all of them lie in the negative real axis and fulfill the criterion in Matignon's condition (4), then the equilibrium E_1 is locally asymptotically stable. However, if any eigenvalue satisfies λ > 0, the condition is violated, and E_1 behaves as a saddle point.

For the numerical simulation of the stability behavior, by adopting standard epidemiological parameter values commonly used in fractional-order SEIR models. Specifically, the transmission rate β = 0.35, contact rate c = 10, quarantine effectiveness q = 0.6, and birth and natural death rates as π = 0.01 and μ = 0.01, respectively. The quarantine release rate is taken as λ = 0.2 and the progression rate from exposed to infected is σ = 0.2, modified by latency factor T = 1. The probability of symptomatic infection is ρ = 0.7, while hospitalization rates are δI = 0.03 for symptomatic individuals and δq = 0.01 for quarantined exposed individuals. Death rates are set as αI = 0.005 and α_H = 0.002,

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recovery rates are chosen as $\gamma_I = 0.08$, $\gamma_A = 0.05$, and $\gamma_H = 0.03$ for symptomatic, asymptomatic, and hospitalized individuals, respectively. The relative infectiousness of asymptomatic individuals is $\theta = 0.5$ and the transmission potential during latency is modulated by $\nu = 0.1$. These values are used consistently across simulations for all considered fractional orders $\alpha = 1.0, 0.985, 0.97, 0.95$, enabling a focused investigation of memory effects on system dynamics and stability.

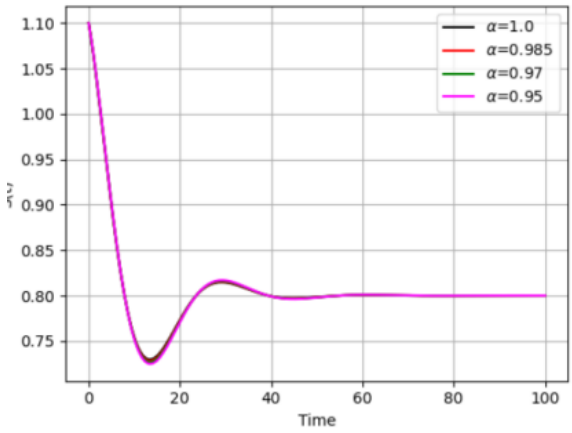
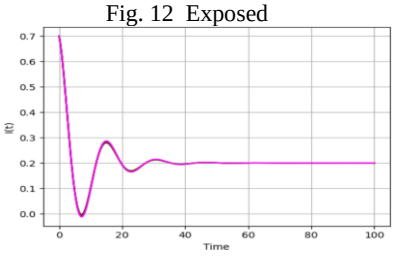


Fig. 11 Susceptible

Simulated trajectories of the state variables for different fractional orders.

Fig. 12 Exposed



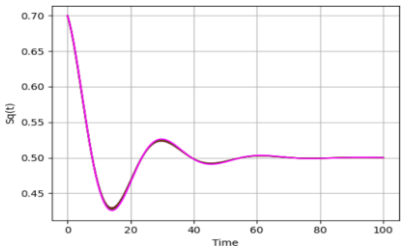


Fig. 16 Quarantined S

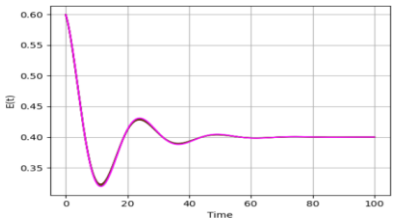


Fig. 13 Infected

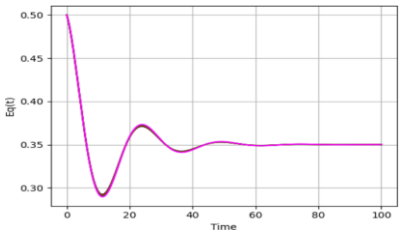


Fig. 17 Quarantined E

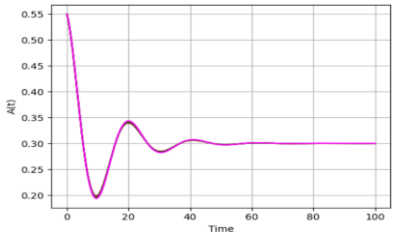


Fig. 14 Asymptomatic

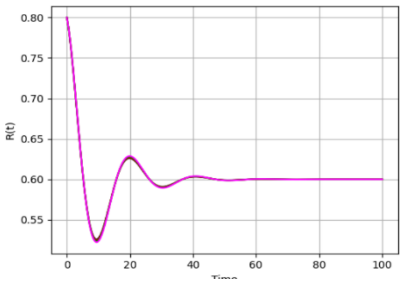


Fig. 18 Hospitalized

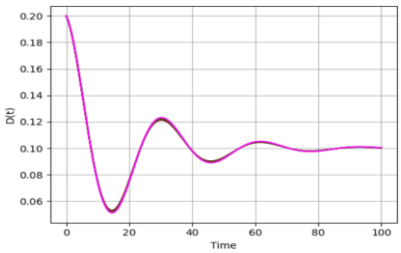


Fig. 15 Deceased

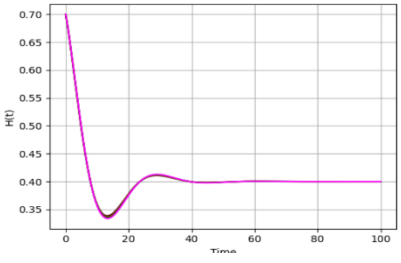


Fig. 19 Recovered

MSW MANAGEMENT -Multidisciplinary, Scientific Work and Management Journal
ISSN: 1053-7899
Vol. 36 Issue 1s, 2026, Pages: 239-250

Observation

Across all subplots, the trajectories of the state variables converge smoothly to equilibrium values, regardless of the fractional order α, confirming the asymptotic stability of the disease-free equilibrium. Lower values of α lead to slightly slower damping, indicating prolonged memory effects, but the overall system remains stable, with no oscillatory divergence or outbreak resurgence. This reinforces that the model behaves stably for all tested fractional orders. The graphical results demonstrate that all state variables exhibit convergence toward a stable equilibrium point across all tested values of the fractional order α. The susceptible population S(t), as well as the exposed and infected classes E(t) and I(t), decline rapidly and stabilize, suggesting effective containment of disease spread. Similarly, the asymptomatic A(t), quarantined Sq(t), Eq(t), and hospitalized H(t) compartments settle to steady-state levels without resurgence.Lower fractional orders (α=0.95, 0.97) exhibit a delayed but smoother transition, reflecting stronger memory effects and inertia in the system—typical of fractional-order dynamics. Notably, the recovery R(t) and death D(t) compartments also reach equilibrium without overshoot or instability. The consistent damping in all compartments, regardless of α, confirms the asymptotic local stability of the disease-free equilibrium. These results validate the model's robustness in preserving system stability under memory-aware dynamics and support the use of fractional-order formulations for more accurate epidemiological modeling.

8. CONCLUSION

The advanced Fractional SEIR model described in this work is a tremendous leap forward in infectious disease modeling.We have identified a set of criteria that guar- antee a unique and well-defined solution for the fractional-order model. Compared to traditional integer-order models, the fractional calculus model provides more accurate, customisable, and realistic disease dynamics. Its capacity for memory effect represen- tation, smooth transition, and delay response makes the model an important tool for controlling and understanding infectious diseases.The stability insight into how param- eter tuning influences the resilience of the disease-free state. This model is especially useful for public health professionals and policymakers, as it facilitates more effective planning and intervention implementation to reduce the effect of disease outbreaks. The model's success comes from the fact that it is able to link theoretical modeling and observed facts, hence constituting a good addition to epidemiology. The accuracy of the model in predicting disease dynamics, public health planning, and the avail- ability of a generalized framework for use in different situations highlight its role as a disease-fighting instrument.

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