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A Study of Fractional Order SIS Model with Fear Effect and Beddington-De Angelis Incidence Rate

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ABSTRACT: Many studies have demonstrated that during epidemics, fear can significantly influence human behaviour, often leading to a decline in birth rates. In this work, we propose a fractional-order SIS compartmental model that incorporates the effects of fear and employs a Beddington-De Angelis type incidence rate. This incidence function captures the impact of preventive measures taken by both susceptible and infected individuals, thereby reflecting more realistic disease transmission dynamics. Following the formulation of the model, we establish fundamental properties such as positivity and boundedness of solutions. We then compute the basic reproduction number, $\mathcal{R}_0$, and demonstrate the existence of an endemic equilibrium when $\mathcal{R}_0 > 1$. Furthermore, we analyze the local stability of both the disease-free and endemic equilibria using the linearized system. To support our analytical results, we conduct numerical simulations using the Adams-Bashforth-Moulton Predictor-Corrector method.

Key Words: Mathematical model, fear effect, Beddington-De Angelis incidence rate, stability analysis.

Contents

1. Introduction

Infectious diseases continue to pose a significant threat to public health and human lifestyles. Illnesses such as chickenpox, measles, cholera, tuberculosis, and influenza have far-reaching societal impacts due to their ability to spread rapidly through direct contact or intermediary carriers. Because of this high transmissibility, outbreaks can escalate into regional or even global epidemics in a short period. Consequently, researchers from diverse disciplines are increasingly involved in understanding the transmission dynamics of infectious diseases and developing strategies for their control.

Mathematical modeling has emerged as a powerful tool in the study and regulation of infectious diseases. Among various mathematical techniques, models based on differential equations are particularly valuable for analyzing disease spread and evaluating control measures. The foundational work in this area is attributed to Kermack and McKendrick [16], who introduced one of the earliest models of epidemic dynamics. Since then, numerous studies have expanded on their approach, applying mathematical models to explore strategies for disease mitigation (see [13,15,12,10,8], and references therein).

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More recently, researchers have started incorporating memory effects into these models using fractional differential equations. These equations are better suited to capture the temporal complexity of real-world epidemiological processes (see [3,5,18,14,7,2,11]). While traditional models assume that changes in disease states happen instantaneously, fractional-order models allow the inclusion of past interactions and delays, which is particularly relevant in contexts where prior exposure and immunity significantly affect disease dynamics. By integrating this historical dependency, fractional models offer a more accurate and nuanced representation, enhancing the predictive power of epidemic simulations. Motivated by these advantages, we adopt a fractional-order approach in modeling disease transmission dynamics in this study.

Public awareness and behavioural responses to infectious disease outbreaks have also intensified in recent times. The fear of infection often drives individuals to limit social interactions, thereby influencing the spread of the disease. Such fear-induced behaviour can result in self-isolation, reduced fertility rates, and changes in survival outcomes. Furthermore, studies in pathology suggest that psychological stress, including fear, can impair immune function, particularly the body's capacity to produce antibodies. Media coverage plays a pivotal role in amplifying this fear, as evidenced during the SARS outbreak (November 2002 to June 2003), which coincided with a noticeable drop in Hong Kong's birth rate- from 8,742 births in 2002 to 8,436 in 2003 [4,9,17].

Considering these observations, we propose a fractional-order SIS (Susceptible-Infected-Susceptible) epidemic model in which individuals who recover from the infection can become susceptible again. The model integrates fear-driven behavioural changes that influence both susceptible and infected individuals. To better represent the contact dynamics under such behavioural responses, we employ a Beddington-De Angelis type incidence function, which accounts for mutual interference and saturation effects during disease transmission.

The paper is organized as follows: Section 2 presents key definitions and results related to the Caputo fractional derivative. Section 3 outlines the assumptions and the mathematical formulation of the model. In Sections 4 and 5, we analyze the non-negativity and boundedness of solutions, derive the basic reproduction number, and investigate the existence of equilibria. Section 6 explores the local stability of the equilibrium points through linearization. In Section 7, numerical simulations are provided to validate the theoretical findings. Finally, the paper concludes with a summary and potential directions for future work in Section 8.

2. Preliminaries

Definition 2.1 *[13] For a function $f(t) \in \mathbb{C}^n$, the fractional derivative of order $\alpha > 0$ in sense of Caputo is given by*

$$D_t^\alpha f(t) = I^{(n-\alpha)} D_t^n f(t) = \frac{1}{\Gamma(n-\alpha)} \int_0^t \frac{f^n(x)}{(t-x)^{(\alpha+1-n)}} dx,$$

where $\alpha \in (n-1, n)$.

Lemma 2.1 *[13] Assume that $\alpha \in (0,1]$ and that both the function $f(t)$ and its fractional derivative ${}_{t_0}D_t^\alpha f(t)$ belong to the metric space $C[a,b]$. If ${}_{t_0}D_t^\alpha f(t) \geq 0$ for all $t \in [a,b]$, then $f(t)$ is monotonically increasing. Conversely, if ${}_{t_0}D_t^\alpha f(t) \leq 0$ for all $t \in [a,b]$, then $f(t)$ is monotonically decreasing.*

Lemma 2.2 *[13] Assume the following fractional-order system of the order $\alpha \in (0,1]$*

$${}_{t_0}D_t^\alpha x(t) = Mx, \qquad x(t_0) = x_0 > 0,$$

where $x \in \mathbb{R}^2$ and $M \in \mathbb{R}^{n \times n}$ (set of all $n \times n$ real matrices). The system is said to be stable asymptotically iff $|\arg(\lambda)| > \frac{\alpha\pi}{2}$ satisfy for each of the eigenvalues λ of the matrix M and the system is said to be stable only iff $|\arg(\lambda)| \geq \frac{\alpha\pi}{2}$ for each of the eigenvalues of matrix M with the eigenvalues satisfying the critical condition $|\arg(\lambda)| = \frac{\alpha\pi}{2}$ must have geometric multiplicity one.

3. Formulation of Fractional Order Epidemic Model

In this section, we develop a fractional-order SIS (Susceptible-Infected-Susceptible) epidemic model based on the Caputo derivative framework, incorporating both treatment interventions and behavioral changes driven by fear of the disease. The total population is divided into two distinct and time-dependent compartments: the susceptible class, $S(t)$, consisting of individuals vulnerable to infection, and the infected class, $I(t)$, which includes individuals currently carrying and capable of transmitting the disease. The dynamics of the epidemic are described using the following system of nonlinear fractional differential equations:

$$\begin{aligned} {}_0D_t^\alpha S(t) &= \frac{\Lambda}{1+\delta I} - \frac{\beta SI}{1+\rho S+\gamma I} + (\psi+u)\,I - \mu S, \\ {}_0D_t^\alpha I(t) &= \frac{\beta SI}{1+\rho S+\gamma I} - (\psi+u)\,I - (\mu+d)\,I, \end{aligned} \tag{3.1}$$

Subject to the conditions

$$S(0)=S_0 \geq 0, I(0)=I_0 \geq 0, \tag{3.2}$$

and the different model parameters are defined in Table 1.

Table 1: **Parameters of the model.**

Parameter	Description
Λ	Birth rate of Susceptible population
β	Disease transmission rate
δ	Level of fear
ρ	preventive measures taken by susceptibles
γ	preventive measures taken by infectives
μ	Natural Death rate
ψ	Natural Recovery rate
u	Recovery rate due to treatment
d	Disease Induced Death rate

4. Non-Negativity and Boundedness

To ensure biological relevance, it is essential that the solutions of system (3.1) remain non-negative and bounded over time. In this context, we define the feasible region as $\Omega^+ = \{(S,I) \in \Omega : S, I \in \mathbb{R}^+\}$, where $\mathbb{R}^+$ denotes the set of all non-negative real numbers. This guarantees that population variables remain meaningful within a biological framework.

Theorem 4.1 *Every solution of the system (3.1) remain non-negative and uniformly bounded starting in Ω^+.*

Proof:

We start by considering the system's initial solution $\Gamma_{t_0} = (S_{t_0}, I_{t_0}) \in \Omega^+$, then from system(3.1) we get,

$$\begin{aligned} D^\alpha S|_{S_{t_0}=0} &= \frac{\Lambda}{1+\delta I} + (\psi+u)I > 0, \\ D^\alpha I|_{I_{t_0}=0} &= 0. \end{aligned}$$

Using Lemma 2.1, we have $S(t), I(t) \geq 0$ for any $t \geq t_0$. Therefore, the solution of the system (3.1) will remain in Ω^+.

Again, consider the function $N(t) = S(t) + I(t)$, then

$$\begin{aligned} D^\alpha N &= D^\alpha S + D^\alpha I \\ &\leq \Lambda - \mu N - dI \\ i.e.\ \ D^\alpha N + \mu N &\leq \Lambda \quad as \ \ I > 0. \end{aligned}$$

We get $N(t) \leq \left(N(t_0) - \frac{\Lambda}{\mu}\right) E_\alpha[-\mu(t-t_0)] + \frac{\Lambda}{\mu} \to \frac{\Lambda}{\mu}$ as $t \to \infty$.

Therefore, solutions of the system (3.1) starting in the region Ω^+ are always lying in the region $\left\{(S,I) \in \Omega : 0 \leq S + I \leq \frac{\Lambda}{\mu}\right\}$. □

5. Basic Reproduction Number and Equilibria

It is straightforward to observe that the system (3.1) admits a disease-free equilibrium (DFE) given by $E_0 = E_0\left(\frac{\Lambda}{\mu}, 0\right)$, where the entire population is susceptible and no infection is present. Our next objective is to determine the basic reproduction number, denoted by $\mathcal{R}_0$, which quantifies the expected number of secondary infections generated by a single infectious individual introduced into a fully susceptible population. The value of $\mathcal{R}_0$ is a crucial threshold parameter for assessing the potential for disease spread or elimination. We compute $\mathcal{R}_0$ using the next-generation matrix approach as outlined in [1]. To proceed, we assume that

$$D_t^\rho X = \mathcal{F}(X) - \mathcal{V}(X),$$

where $X = (I)^T$ and $\mathcal{F}(X)$ be the matrix of new infection term, $\mathcal{V}(X)$ be the matrix of outgoing terms. The Jacobian matrices F and V of $\mathcal{F}(X)$ and $\mathcal{V}(X)$, respectively, are given as:

$$F = \left[\frac{\beta S(1+\rho S)}{(1+\rho S+\gamma I)^2}\right],$$

$$V = [\psi + u + \mu + d].$$

The next generation matrix, at infection-free equilibrium E_0, is

$$FV^{-1} = \left[\frac{\beta\Lambda}{(\mu+\rho\Lambda)(\psi+u+\mu+d)}\right].$$

Thus,

$$\mathcal{R}_0 = \frac{\beta\Lambda}{(\mu+\rho\Lambda)(\psi+u+\mu+d)}.$$

In addition, we will show that the model (3.1) has an endemic steady state when $\mathcal{R}_0 > 1$. Let $E_1 = E_1(S_*, I_*)$ be an endemic equilibrium such that $S_* > 0,\ I_* > 0$ and

$$\begin{cases} \frac{\Lambda}{1+\delta I_*} - \frac{\beta S_* I_*}{1+\rho S_*+\gamma I_*} + (\psi+u)\, I_* - \mu S_* = 0, \\ \frac{\beta S_* I_*}{1+\rho S_*+\gamma I_*} - (\psi+u)\, I_* - (\mu+d)\, I_* = 0, \end{cases} \tag{5.1}$$

It follows that,

$$S_* = \frac{(\psi+u+\mu+d)(1+\gamma I_*)}{\beta - (\psi+u+\mu+d)\rho}$$

and I_* can be obtained by solving the following equation:

$$A_2 {I_*}^2 + A_1 I_* + A_0 = 0, \tag{5.2}$$

where,

$$\begin{aligned} A_2 &= \delta\left(d^2\rho + d(-\beta - \gamma\mu + \rho(2\mu+u+\psi)) - \mu(\beta + (\gamma-\rho)(\mu+u+\psi))\right), \\ A_1 &= d^2\rho - d(\beta+\gamma\mu+\delta\mu-2\mu\rho-\rho\psi-\rho u) - \mu(\beta+(\gamma+\delta-\rho)(\mu+u+\psi)), \\ A_0 &= (\mathcal{R}_0 - 1)\,(\Lambda\rho+\mu)(d+\mu+u+\psi). \end{aligned} \tag{5.3}$$

Thus, it can be observed that, A_2 and A_1 can be positive or negative but $A_0 > 0$ for $\mathcal{R}_0 > 1$. So, according to Descarte's rule of sign, the polynomial (5.2) will have at least one positive root I_* for $\mathcal{R}_0 > 1$. In the current study, we have focused on the case of existence of unique positive equilibrium point. Consider $\mathcal{R}_0 > 1$, then the combinations of signs of coefficients A_1 and A_2 that allow the existence of a unique positive root for the polynomial (5.2) are as follows:

(i) $A_2 < 0$ and $A_1 < 0$,

(ii) $A_2 < 0$ and $A_1 > 0$,

When we get the value of I_*, we can obtain the unique positive endemic equilibrium point, $E_1 = E_1(S_*, I_*)$.

6. Stability Analysis

This section presents the stability analysis of the equilibrium points of the system. Local stability determines whether small perturbations around an equilibrium point will return to the equilibrium (stable) or move away from it (unstable). Let us assume the subsequent coordinate transform $S(t) = S_*(t) + s(t)$; $I(t) = I_*(t) + i(t)$, where $(S_*(t), I_*(t))$ denotes the equilibrium point of the model. The linearised system at any steady state is given by

$$\begin{cases} {}_0D_t^\alpha S(t) = -\left(\frac{\beta I_*(1+\gamma I_*)}{(1+\rho S_*+\gamma I_*)^2} + \mu\right)S - \left(\frac{\delta\Lambda}{(1+\delta I_*)^2} + \frac{\beta S_*(1+\rho S_*)}{(1+\rho S_*+\gamma I_*)^2} - (\psi+u)\right)I, \\ {}_0D_t^\alpha I(t) = \left(\frac{\beta I_*(1+\gamma I_*)}{(1+\rho S_*+\gamma I_*)^2}\right)S + \left(\frac{\beta S_*(1+\rho S_*)}{(1+\rho S_*+\gamma I_*)^2} - (\psi+u+\mu+d)\right)I. \end{cases} \tag{6.1}$$

Applying the Laplace transform on both side of equation (6.1), the reduced system can be written in the following matrix form:

$$\bigtriangledown(s)\begin{pmatrix} \mathcal{L}\{S(t)\} \\ \mathcal{L}\{I(t)\} \end{pmatrix} = \begin{pmatrix} \nu_1(s) \\ \nu_2(s) \end{pmatrix},$$

where,

$$\nu_1(s) = s^{\alpha-1}S(0),\ \nu_2(s) = s^{\alpha-1}I(0),$$

and

$$\bigtriangledown(s) = \begin{pmatrix} s^\alpha + \frac{\beta I_*(1+\gamma I_*)}{(1+\rho S_*+\gamma I_*)^2} + \mu & \frac{\delta\Lambda}{(1+\delta I_*)^2} + \frac{\beta S_*(1+\rho S_*)}{(1+\rho S_*+\gamma I_*)^2} - (\psi+u) \\ -\frac{\beta I_*(1+\gamma I_*)}{(1+\rho S_*+\gamma I_*)^2} & s^\alpha - \left(\frac{\beta S_*(1+\rho S_*)}{(1+\rho S_*+\gamma I_*)^2} - (\psi+u+\mu+d)\right) \end{pmatrix}. \tag{6.2}$$

In this case, the characteristic polynomial of system (3.1) is $\det(\bigtriangledown(s))$, and the characteristic matrix is $\bigtriangledown(s)$. The distribution of eigenvalues of the characteristic polynomial $\det(\bigtriangledown(s))$ provides a means to analyze the local stability of the system (3.1).

6.1. Local stability of disease-free equilibrium

In this subsection, we will show the local stability of disease-free equilibrium (DFE) point, $E_0 = E_0\left(\frac{\Lambda}{\mu}, 0\right)$. for which the characteristic matrix at DFE is as follows:

$$\bigtriangledown(s) = \begin{pmatrix} s^\alpha + \mu & \delta\Lambda + \frac{\beta\Lambda}{\mu+\rho\Lambda} - (\psi+u) \\ 0 & s^\alpha - \left(\frac{\beta\Lambda}{\mu+\rho\Lambda} - (\psi+u+\mu+d)\right) \end{pmatrix}, \tag{6.3}$$

or

$$\bigtriangledown(s) = \begin{pmatrix} s^\alpha + \mu & \delta\Lambda + \frac{\beta\Lambda}{\mu+\rho\Lambda} - (\psi+u) \\ 0 & s^\alpha - (\psi+u+\mu+d)\big(\mathcal{R}_0 - 1\big) \end{pmatrix}. \tag{6.4}$$

Since stability is determined by the eigenvalues of the characteristic matrix (6.4), we obtain two eigenvalues: $\omega_1 = -\mu$ and $\omega_2 = (\psi+u+\mu+d)\big(\mathcal{R}_0 - 1\big)$. The disease-free equilibrium is locally asymptotically stable if all eigenvalues are negative. In particular, the second eigenvalue ω_2 is negative when $\mathcal{R}_0 < 1$, indicating local stability, and becomes positive when $\mathcal{R}_0 > 1$, leading to instability. Thus, we have the following theorem.

Theorem 6.1 *The disease-free equilibrium E_0 is locally asymptotically stable if and only if the threshold value $\mathcal{R}_0$ is less than one, otherwise unstable.*

6.1.1. Local stability of endemic equilibrium. In this subsection, we will discuss the local stability of endemic equilibrium, $E_1 = E_1(S_*, I_*)$, for which we find the characteristic matrix $\bigtriangledown(s)$ from equation (6.2) at E_1, which is given by:

$$\bigtriangledown(s) = \begin{pmatrix} s^\alpha + \frac{\beta I_*(1+\gamma I_*)}{(1+\rho S_* + \gamma I_*)^2} + \mu & \frac{\delta\Lambda}{(1+\delta I_*)^2} + \frac{\beta S_*(1+\rho S_*)}{(1+\rho S_* + \gamma I_*)^2} - (\psi + u) \\ -\frac{\beta I_*(1+\gamma I_*)}{(1+\rho S_* + \gamma I_*)^2} & s^\alpha - \left(\frac{\beta S_*(1+\rho S_*)}{(1+\rho S_* + \gamma I_*)^2} - (\psi + u + \mu + d)\right) \end{pmatrix}. \tag{6.5}$$

Let $s^\alpha = \lambda$, then the characteristic equation corresponding to characteristic matrix (6.5) is:

$$\begin{aligned} &\frac{{I_*}^2\beta\gamma\delta\Lambda}{(I_*\delta+1)^2(I_*\gamma+\rho S_*+1)^2} + \frac{{I_*}^2\beta\gamma\lambda}{(I_*\gamma+\rho S_*+1)^2} + d\left(\frac{I_*\beta(I_*\gamma+1)}{(I_*\gamma+\rho S_*+1)^2} + \lambda + \mu\right) - \\ &\frac{\beta\lambda\rho {S_*}^2}{(I_*\gamma+\rho S_*+1)^2} - \frac{\beta\mu\rho {S_*}^2}{(I_*\gamma+\rho S_*+1)^2} + \frac{I_*\beta\delta\Lambda}{(I_*\delta+1)^2(I_*\gamma+\rho S_*+1)^2} + \frac{I_*\beta\lambda}{(I_*\gamma+\rho S_*+1)^2} \\ &- \frac{\beta\lambda S_*}{(I_*\gamma+\rho S_*+1)^2} - \frac{\beta\mu S_*}{(I_*\gamma+\rho S_*+1)^2} + u\left(\frac{I_*\beta(I_*\gamma+1)}{(I_*\gamma+\rho S_*+1)^2} + \lambda + \mu\right) \\ &+ \lambda^2 + \psi(\lambda+\mu) + 2\lambda\mu + \mu^2 = 0 \end{aligned}$$

which can be simplified into the following polynomial form:

$$\lambda^2 - 2A_1\lambda + A_0 = 0, \tag{6.6}$$

where

$$\begin{aligned} A_1 &= \frac{\beta(S_*(\rho S_*+1) - I_*(I_*\gamma+1))}{2(I_*\gamma+\rho S_*+1)^2} - \frac{(d+2\mu+u+\psi)}{2}, \\ A_0 &= \frac{\beta\left({I_*}^2\gamma(d+u) + I_*(d+u) - \mu S_*(\rho S_*+1)\right)}{(I_*\gamma+\rho S_*+1)^2} + \frac{I_*\beta\delta\Lambda(I_*\gamma+1)}{(I_*\delta+1)^2(I_*\gamma+\rho S_*+1)^2} \\ &\quad + \mu(d+\mu+u+\psi). \end{aligned}$$

The eigenvalues are

$$\lambda_{1,2} = A_1 \pm \sqrt{{A_1}^2 - A_0}.$$

The different values of λ_1 and λ_2 are depending on the coefficients A_1 and A_0. Thus, the possibilities arises for which the eigenvalues will be negative and endemic equilibrium will be locally stable, according to Lemma 2.2, are given in the following theorem:

Theorem 6.2 *Consider the endemic equilibrium point $E_1 = (S_*, I_*)$ of the system. The local asymptotic stability of E_1 depends on the characteristic equation coefficients A_0 and A_1, and it is determined by the following conditions:*

(i) If $A_1 < 0$ and $A_1^2 \geq A_0$, then the equilibrium E_1 is locally asymptotically stable.

(ii) If $A_1 \geq 0$ and $A_1^2 \geq A_0$, then the equilibrium E_1 is unstable.

(iii) If $A_1 > 0$ and $A_1^2 < A_0$, then the equilibrium E_1 is locally asymptotically stable.

(iv) If $A_1 < 0$ and $A_1^2 < A_0$, then the equilibrium E_1 is locally asymptotically stable.

(v) If $A_1 = 0$ and $A_1^2 < A_0$, then the equilibrium E_1 is locally asymptotically stable.

Table 2: **Parameter Values for simulation.**

Parameter	Values
Λ	3
β	0.004
δ	0.006
ρ	0.002
γ	0.001
μ	0.04
ψ	0.05
u	0.03
d	0.05

7. Numerical Simulation and Discussion

In this section, numerical simulations are carried out using MATLAB to validate the theoretical results, employing the set of parameter values listed in Table 2. The simulations utilize the fractional Adams-Bashforth-Moulton method, as described in [6], to solve the system of equations. For the initial conditions, the susceptible and infected populations are taken as $S(0) = 73$ and $I(0) = 1$, respectively. Based on the parameter values provided in Table 2, the coefficients of the polynomial equation (5.2) are computed as follows:

$$A_2 = -2.0172 \times 10^{-6}, \quad A_1 = -0.000377, \quad A_0 = 0.00418.$$

These values satisfy the necessary conditions for the existence of a unique, biologically feasible endemic equilibrium. Consequently, the system admits a single endemic equilibrium point given by $E^* = (S^*, I^*) = (46.9357, 10.4979)$, for which the basic reproduction number is calculated as $\mathcal{R}_0 = 1.5345$.

Using the initial population values, we generate Figures 1 and 2, which illustrate the effect of the fractional-order parameter α on the susceptible and infected sub-populations.

Figure 1 highlights how variations in α influence the convergence behavior of system (3.1). An increase in α leads to faster stabilization of the susceptible population toward its steady state. In contrast, decreasing α strengthens the memory effect inherent in the fractional-order system, thereby slowing down the rate of convergence. In practical terms, a lower value of α results in a prolonged presence of the disease in the population.

In Figure 2, we can see that when $\alpha = 1$, the infected population quickly reach a steady state. However, as the value of α decreases, the time it takes for these populations to reach the steady state increases. This shows how the epidemic evolves over time. Moreover, from Figures 1 and 2, we can infer that as the disease progresses, the susceptible individuals decrease while the infected individuals increase, eventually reaching its steady state.

Figure 3 provides valuable insights into the long-term behaviour of disease transmission within a population by depicting a phase portrait of susceptible versus infected individuals. This graphical representation allows us to visualize how the populations of susceptible and infected individuals evolve over time and interact with one another. From the phase portrait, we observe that as time progresses, the number of susceptible individuals increases while the number of infected individuals decreases. This inverse relationship suggests that the disease is gradually being brought under control. The increase in the susceptible population may initially seem strange, however this reflects the effect of reduced transmission of infection, that is, fewer individuals are becoming infected, allowing more individuals to remain in the susceptible class.

The work done in this paper focuses on the role of fear and the Beddington-De Anglis incidence rate in shaping the dynamics of infectious disease transmission. Specifically, it explores how behavioural responses driven by fear and preventive actions influence the susceptible and infected populations. Figures 4, 5 and 6 illustrate the impact of these key parameters, namely the level of fear δ and the rates of preventive measures (ρ and γ), on the disease dynamics.

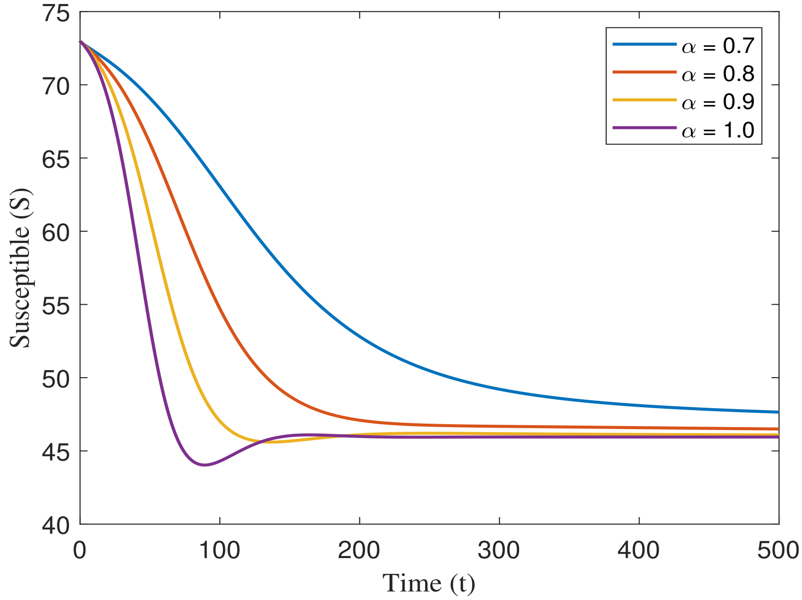

Figure 1: **Time series plot of susceptible population for different values of fractional order α**

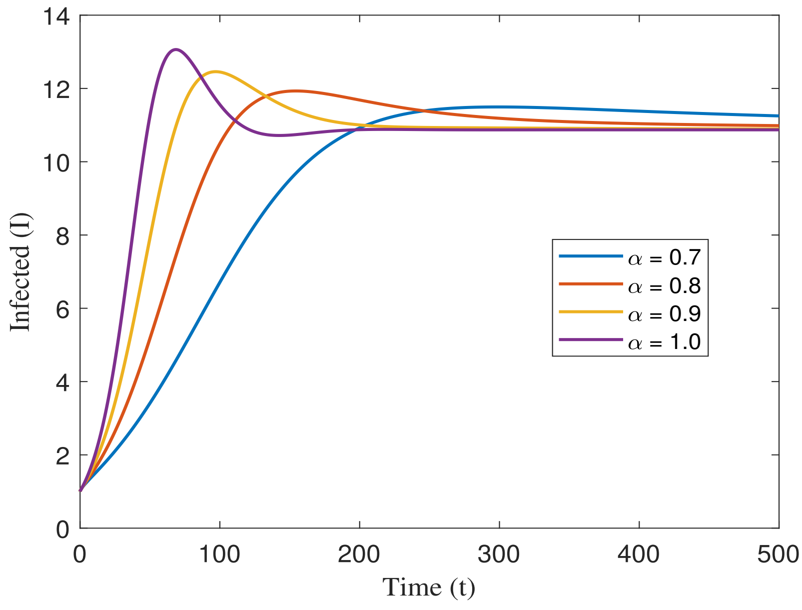

Figure 2: **Time series plot of infected population for different values of fractional order α**

From Figure 4, we observe that as the fear level within the population increases, there is a decline in the number of infected individuals. This outcome can be attributed to fear-induced behavioural changes such as social distancing, improved hygiene, and reduced contact rates, all of which contribute to lowering disease transmission.

Figures 5 and 6 further demonstrates that higher values of ρ and γ, which represent the rates at which susceptible individuals adopt preventive measures, lead to an increase in the susceptible population. This is because effective preventive behaviours reduce the likelihood of infection, thereby increasing the

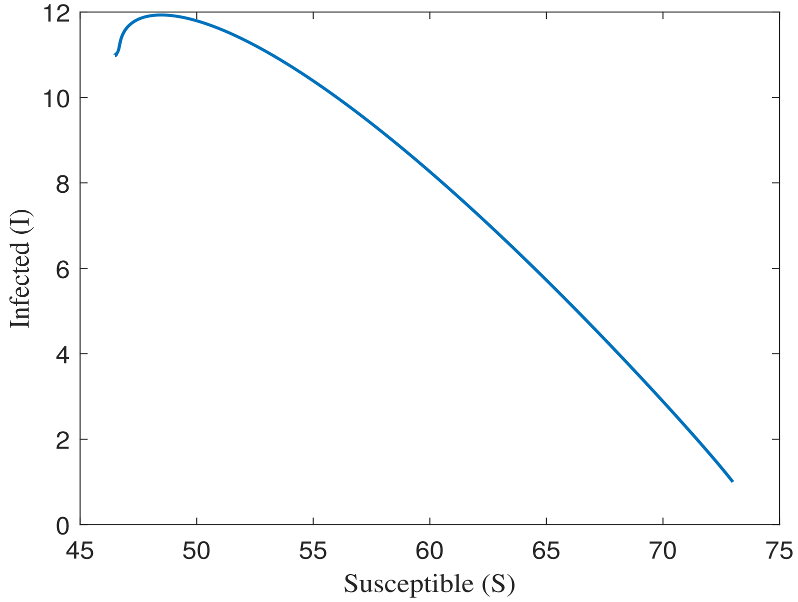

Figure 3: **Phase diagram for susceptible and infected population for fractional order** $\alpha = 0.8$

susceptible population over time.

Altogether, these findings highlight the critical role of psychological and behavioural factors, particularly fear and preventive actions, in modulating epidemic outcomes. Incorporating these elements into epidemiological models helps capture more realistic disease dynamics and can inform more effective public health interventions.

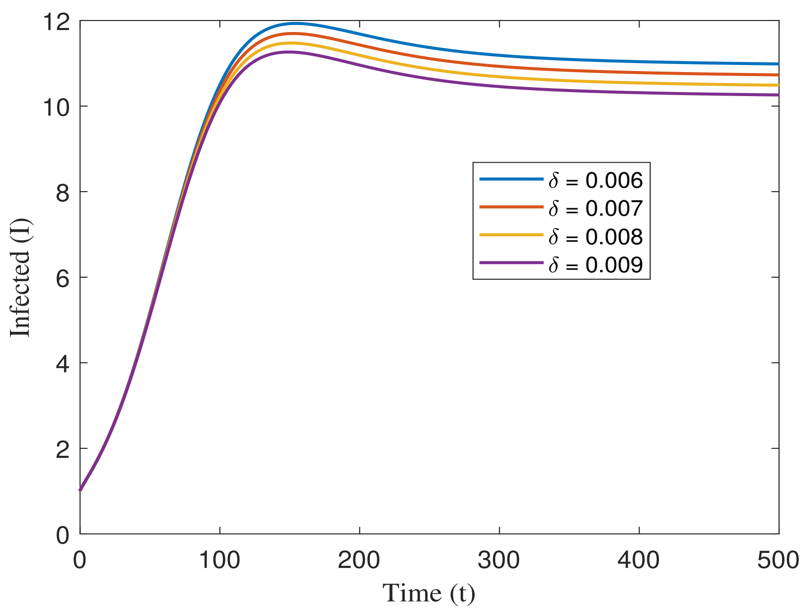

Figure 4: **Effect of fear level** δ **on I for fractional order** $\alpha = 0.8$

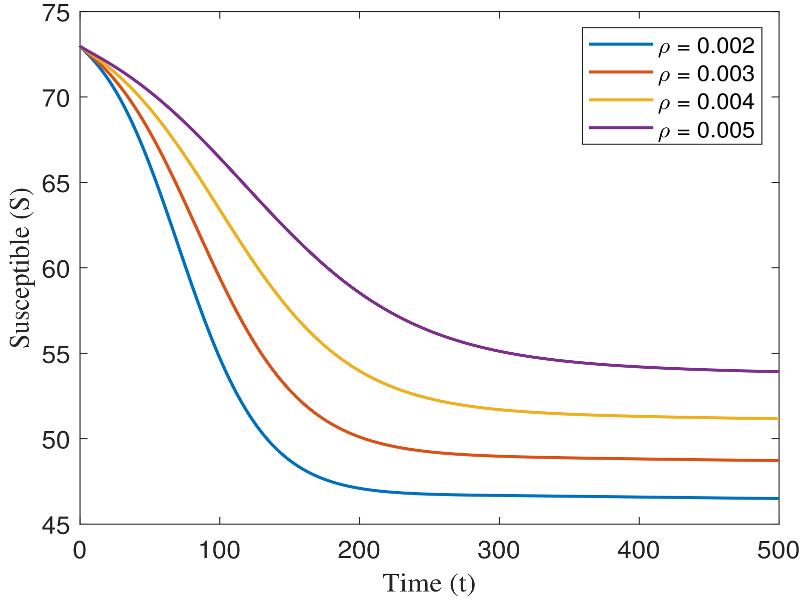

Figure 5: **Effect of preventive measures ρ on S for fractional order** $\alpha = 0.8$

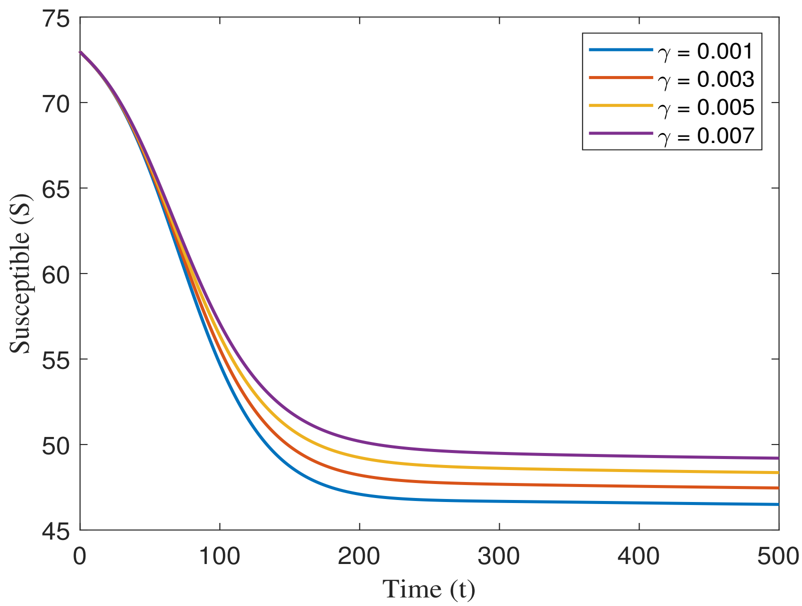

Figure 6: **Effect of preventive measures γ on S for fractional order** $\alpha = 0.8$

8. Conclusion

Mathematical modeling is a valuable tool for understanding the dynamics of epidemics and for planning and evaluating intervention strategies. In the present article, we propose and analyze a Caputo-type fractional-order Susceptible-Infected-Susceptible (SIS) model that incorporates the fear effect and preventive measures adopted by both susceptible and infected individuals. These behavioural effects are modeled through a Beddington-De Angelis type incidence rate. First, we establish the well-posedness of the model by proving the positivity and boundedness of the solutions, ensuring that they remain positive and uniformly bounded within a biologically relevant region. We show that the model admits two equilibria: a disease-free equilibrium point E_0, and an endemic equilibrium point E_1, which exists when the basic reproduction number $\mathcal{R}_0 > 1$. The reproduction number $\mathcal{R}_0$ is derived using the next-generation matrix approach. We then analyze the stability of the disease-free equilibrium (DFE) using $\mathcal{R}_0$, demonstrating that the disease dies out when $\mathcal{R}_0 < 1$ and persists when $\mathcal{R}_0 > 1$. The local stability behaviour of the endemic equilibrium point is also discussed. Furthermore, numerical simulations are conducted to validate our theoretical results and to study the effects of memory (fractional-order dynamics) using the Adams-Bashforth-Moulton Predictor-Corrector method in MATLAB. We observe the influence of the fear level on the infected population, as well as the impact of preventive measures ρ and γ on the susceptible population. The results indicate that these preventive measures are highly effective in controlling the spread of the disease, and the fear effect significantly reduces the disease burden in the population. Overall, the proposed model, incorporating both the fear effect and the Beddington-De Angelis incidence rate within a fractional-order framework, offers valuable insights for epidemiologists, policymakers, and public health officials.

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