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A Design of Predictive Eco-Epidemiological Fractional Model (3 Species) Induced by Disease in Prey

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Abstract: This study develops a fractional-order eco-epidemiological model to describe the dynamics of a three-species system comprising susceptible prey $U_S(t)$, infected prey $U_I(t)$, and predators $V(t)$. The disease is assumed to spread non-vertically among the prey, governed by a transmission rate γ, and only affects the susceptible prey. The proposed model uses Caputo fractional derivatives to incorporate memory effects, offering a more realistic framework for ecological systems. The existence and uniqueness of the solution are established using the Lipschitz condition. We identify and analyze biologically meaningful equilibrium points, including the infection-free equilibrium and coexistence equilibrium, and investigate their stability within the fractional-order setting. A fractional-order numerical scheme based on the Adams-Bashforth method is implemented to validate the analytical results. Numerical simulations are conducted using biologically relevant parameters: intrinsic growth rate $a = 0.8$, transmission rate $\gamma = 0.6$, natural death rate of infected prey $m = 0.1$, natural death rate of predators $n = 0.1$, and predation-related death rates $\alpha_1 = 0.3$, $\alpha_2 = 0.2$. The predator self-competition rate is set as $\beta = 0.4$. Simulation results reveal that reducing the transmission rate γ from 0.6 to 0.4 leads to a 42% reduction in the peak population of infected prey $U_I(t)$, while increasing the fractional order from 0.75 to 0.95 decreases predator fluctuations $V(t)$ by 36%, indicating stronger damping due to memory effects. Additionally, a 28% improvement in the survival of susceptible prey $U_S(t)$ is observed when the predation rate on infected prey α_2 is increased, highlighting potential ecological trade-offs. The results confirm the theoretical stability conditions and illustrate how variations in key parameters influence species persistence, disease burden, and predator-prey balance. The proposed model provides a predictive and flexible framework for analyzing eco-epidemiological systems with disease pressure in prey populations and can aid in formulating strategies for ecological disease management.

Keywords: Eco-epidemiological model; predator-prey dynamics, disease in prey population; Caputo derivative; stability analysis; time series simulation; existence and uniqueness.

1 Introduction, Motivation and Preliminaries

Foreign bodies, such as germs, viruses, fungi, or parasites, are the source of infectious diseases in the human body. These contaminants are transferred from one person to another through infection. Human beings, animals, birds, contaminated food, or other environmental factors could act as carriers for these contaminants. Infection arising from these foreign agents could indicate high body temperature, pain, and sore throat. These symptoms may vary depending on the site of infection, its type, and sensitivity. However, mild attacks of foreign bodies produce fewer infections that can be cured without any treatment. Alternatively, the infection could lead to death if not diagnosed or treated properly in a severe case, and it could alter the population balance of many species. The development of more virulent diseases in a sensitive population may even lead to the extinction of highly affected species. In such cases, mathematical modelling for predicting the evolution of species has become frequently utilized in the past few decades. Starting from Lotka-Volterra models [1, 2], these models prevented the loss of many species whose extinction was proven. Today, scientists in contemporary society are converging their efforts to utilize the Lotka-Volterra models as a significant tool for species conservation and to encourage governments to adopt suitable strategies to reduce species extinction. Several mathematical and ecological studies have been established on the Lotka-Volterra model, dealing with the predator-prey

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relationship. Assuming integer-order derivatives, conventional predator-prey models typically imply memoryless interactions. However, many ecological and biological systems exhibit memory and genetic effects that classical models cannot adequately capture. To address this limitation, fractional-order differential equations have gained prominence in recent years. These models contain memory effects by allowing the order of the derivative to be non-integer, making them well-suited for ecological systems where the present state depends not just on current conditions but also on the historical states of the system. Ecological species are susceptible to infection, which can affect their evolution. By considering the predator-prey relationship, one can clearly understand that the infection can reduce some predators' ability to hunt, placing them at risk of extinction as they lose hunting efficiency. The literature review of [3, 4, 5, and 6] more clearly investigated the predation-pray interaction under infection. Conversely, extinction due to infection can be reduced by adopting strategies such as predator-hunting cooperation, in which several animals gather and collectively put in their efforts to achieve a successful hunt. The rate of hunting failure can be reduced by this method. Wild beasts like lions, hyenas, and wild dogs hunt this way. [7] Pioneered the modelling of predator hunting cooperation and simplified the most complex animal behaviour details. Since then, many publications, such as [8, 9, 10-14, 15, 16], have explored the behaviour of animals in predator-prey relationships that depict cooperation. The primary subject of this research paper is the predator-prey relationship under the influence of infection. We constructed a three-species model and examined how the infection spreads within the prey population. The term "prey population" refers to two distinct gene pools. i- Susceptible Prey and ii- infected prey. Thus, dependent on the predator's opportunism, the predator has two types of prey available to him. Considering the results of [17], it is easy to see how the time-fractional derivative is used, as it has numerous applications in real-world situations. The memory effect is well acquainted with the dynamic system's derivative; memory rate is used to denote the order of the derivative, and memory function is used to denote the kernel of the fractional derivative. [18-20] have utilized time-fractional derivatives for real-world modeling issues. A review of several publications revealed that a detailed study had been conducted on the common problems associated with the food chain. Species interaction boosts one population while dwindling the other, revealing the predator-prey relationship. The interaction of species brings alteration in species dynamics. One species feeds (predator) while the other gets hunted (prey) in such relations. The snowy owl vs. lemming interaction is the most common example of a food chain-based predator-prey relationship. The snowy owl feeds on arctic rodents called lemming, whereas lemming feeds on tundra plants for survival. The first-ever explanatory model, which provides a detailed view of the predator-prey relationship, was presented by the Lotka-Volterra model. This model explains the fundamentals of various oscillatory levels of Adriatic fish during the First World War [35, 36]. Nowadays, this scientific study has evolved into its best version in explaining the two-species competitive model systems [37]. Zhanyuan [38] explored the persistence behaviour of a Lotka–Volterra system with delays and varying intrinsic growth rates. Yonghui and Moan [39] have given novelty to the conditions and existence of the Lotka-Volterra system. Details can be seen in [40, 41-44]. Functional response [45, 46, 47], competition [48, 49], and cooperation [50, 51] are some of the factors which affect the dynamics of the predator-prey model. Fundamentals of behaviour and stability of the Lotka-Volterra model are investigated in [52-54]. While fractional derivatives have numerous definitions, the Riemann-Liouville and Caputo fractional derivatives are the most significant in practice. An explicit linear expression for these derivatives is the Riesz fractional derivative, which is the left- and right-hand Riemann-Liouville fractional derivatives. The fractional derivative calculated by Caputo is almost indistinguishable from the one calculated by Riemann-Liouville. Under the regularity assumptions of specific functions, the Caputo fractional derivative can be transformed from the Riemann-Liouville fractional derivative. The time-fractional derivatives are usually defined using the Caputo fractional derivatives for solving fractional partial differential equations (PDEs). The main reason for this is that the Riemann-Liouville method's initial conditions need to include the origin-time limit values of the fractional Riemann-Liouville derivative. The Caputo derivative uses the initial conditions of the fractional derivative, which is the main advantage of Caputo. The form is identical to that of differential equations of integer order, and at the bottom, you can see the limit of the integer derivative of the unknown function [55-57]. In [58], the authors provided the solution of the nonlinear delay fractional order model. In [59], the authors formulated a model for guava fruit. The stability and existence of the model are provided in detail. Owolabi et al. designed a fractional-order predator-prey model, with stability and spatial patterns being key discussions [60]. In [61], the authors investigated a Caputo fractional order predator-prey model. Variations of predator and prey populations are depicted graphically. In this study, we propose a novel fractional-order eco-epidemiological model that considers three interacting species: healthy prey, infected prey, and predator populations. The disease is assumed to affect only the prey species and to be transmitted non-vertically, that is, through contact between healthy and infected prey. This assumption reflects a range of real-world scenarios in which diseases spread horizontally within animal populations due to interactions or proximity. The incorporation of infection dynamics within the prey class adds a realistic layer to predator-prey systems, capturing phenomena such as predation on infected individuals and changes in predator preference due to prey health status. To analyze the proposed model, we first establish the existence and uniqueness of solutions using the Lipschitz continuity condition, which ensures that the model is mathematically well-posed. We then identify and classify the equilibrium points, focusing particularly on biologically significant cases: the infection-free equilibrium and the coexistence equilibrium, where all three classes persist. The stability of these points is studied under the framework of fractional-order systems, employing techniques such as linearization and eigenvalue analysis adapted for fractional dynamics. To support the theoretical analysis, we design a numerical scheme based on fractional calculus, which allows us to approximate the system's dynamics over time. The simulation results validate the theoretical findings and reveal the influence of key parameters on the system's evolution. Through a series of time-series plots and parameter variation analyses, we highlight the roles of disease transmission rate, predation rate, and fractional order in shaping the population dynamics. The main contributions of this work are summarized as follows: 1. We developed a three-species fractional-order eco-epidemiological model in which disease affects the prey species. 2. We established the model's well-posedness through existence and uniqueness results. 3. We classified the stability analysis of equilibrium points in the context of fractional calculus. 4. We designed and implemented a numerical scheme suitable for fractional systems. 5. We performed extensive simulations to explore the impact of biological parameters and to illustrate model dynamics. The detail of the present work is as described, and section (2) is devoted to some fundamental results of fractional calculus, which help discuss the next section. In section (3), we present the proposed model and the parameter description. A flow diagram is provided to help understand the model formulation. In section (4), the existence and uniqueness of the model are provided. Section (5) presents all of the model's equilibrium points and discusses their existence. Both the disease-free and coexisting critical equilibrium points are addressed in Section (6). The stability of two fixed points is discussed in Section (6). The numerical findings demonstrating the stability of the coexistence of the fixed point are presented in section 7. In addition, graphs display the model's time series solution. .

2 Preliminaries

Here, we discuss some related concepts of fractional calculus and differential equations that are helpful for further study.

2.1 Definition (Caputo Fractional Derivative)

Let $\alpha \in (n-1,n)$ where $n \in \mathbb{N}$, and let $f(t)$ be a sufficiently smooth function. The Caputo fractional derivative of order α is defined as:

$$D^{\alpha} f(t) = \frac{1}{\Gamma(n-\alpha)} \int_0^t (t-\varphi)^{n-\alpha-1} f^{(n)}(\varphi)\, d\varphi,$$

where $\Gamma(\cdot)$ denotes the Gamma function, and $f^{(n)}(\varphi)$ is the n^{th} derivative of f with respect to φ.

2.2 Definition (Lipschitz Continuity)

A function $f : \mathbb{R} \to \mathbb{R}$ is said to satisfy the Lipschitz condition if there exists a positive constant k such that for all $y_1, y_2 \in \mathbb{R}$, the following inequality holds:

$$|f(y_1) - f(y_2)| \leq k|y_1 - y_2|.$$

2.3 Theorem (Stability of Fractional-Order Systems)

Consider the fractional-order differential equation:

$$D^{\alpha} x = f(x), \quad x(0) = x_0,$$

where $x \in \mathbb{R}^n$ and $0 < \alpha < 1$. Let x_* be an equilibrium point of the system. Then x_* is said to be *locally asymptotically stable* if all the eigenvalues λ_i of the Jacobian matrix $J\big|_{x=x_*}$ satisfy the condition:

$$|\arg(\lambda_i)| > \frac{\alpha\pi}{2}.$$

3 Mathematical Model Formulation

Fractional calculus has numerous applications in biology, physics, robotics, optics, electrical and mechanical engineering, and other sciences. Several applications are also found in electrochemistry, electromagnetism, signal processing, and population dynamics. Derivatives of fractional-order differential equations offer a suitable mathematical model for addressing practical issues. Due to the accuracy of mathematical modelling, fractional-order models have attracted numerous researchers. It is widely acknowledged that real-life problems are more accurately modelled via fractional-order derivatives than classical integer-order ones.

Motivated by the successful application of fractional calculus in various fields, we present a fractional-order predator–prey disease model incorporating a transmissible disease in the prey population. Many diseases exhibit characteristics of non-locality and memory. Therefore, we utilize the Caputo fractional-order derivative, as it is a non-local operator that appropriately reflects epidemic behavior. The Caputo derivative is particularly suitable for real-world problems due to its handling of standard initial conditions.

In this model, we assume that the infection spreads non-vertically and consider three population classes: predators, susceptible prey, and infected prey. Let $U_S(t)$ denote the density of susceptible prey, $U_I(t)$ the density of infected prey, and $V(t)$ the density of predators. The descriptions of the parameters involved in the model are summarized in Table 1.

Table 1: Model parameters and their biological meanings

Parameter	Physical Meaning
$U_S(t)$	Density of susceptible prey
$U_I(t)$	Density of infected prey
$V(t)$	Density of predator
a	Intrinsic growth rate of prey
α_1	Death rate of susceptible prey due to predation
α_2	Death rate of infected prey due to predation
β	Death rate due to self-competition among predators
γ	Transmission rate of disease
m	Natural death rate of infected prey
n	Natural death rate of predators

The proposed fractional-order system is governed by the following set of equations:

$$D^{\alpha}U_S(t) = U_S(t)\left[a\left(1-U_S(t)\right) - \gamma U_I(t) - \alpha_1 V(t)\right], \tag{1}$$

$$D^{\alpha}U_I(t) = U_I(t)\left[\gamma U_S(t) - \alpha_2 V(t) - m\right], \tag{2}$$

$$D^{\alpha}V(t) = V(t)\left[\alpha_1 U_S(t) + \alpha_2 U_I(t) - \beta V(t) - n\right], \tag{3}$$

where D^{α} denotes the Caputo fractional derivative with respect to time t.

The Caputo fractional derivative of a function $U(t)$ of order $\alpha \in (n-1,n)$, where $n \in \mathbb{N}$, is defined as:

$$D^{\alpha}U(t) = \frac{1}{\Gamma(n-\alpha)}\int_0^t (t-\phi)^{n-\alpha-1}U^{(n)}(\phi)\,d\phi, \tag{4}$$

where $\Gamma(\cdot)$ is the Gamma function and $U^{(n)}(\phi)$ denotes the n^{th} derivative of U with respect to ϕ.

4 Analysis of the model

This section deals with the mathematical analysis of the proposed model. Here, we prove the existence and uniqueness of the solution using the Lipschitz condition.

4.1 Existence and Uniqueness

We define the region $\mathscr{X} \times (0,t]$, where

$$\mathscr{X} = \left\{(U_S, U_I, V) \in \mathbb{R}^3 : \max(U_S, U_I, V) \le \mu\right\}.$$

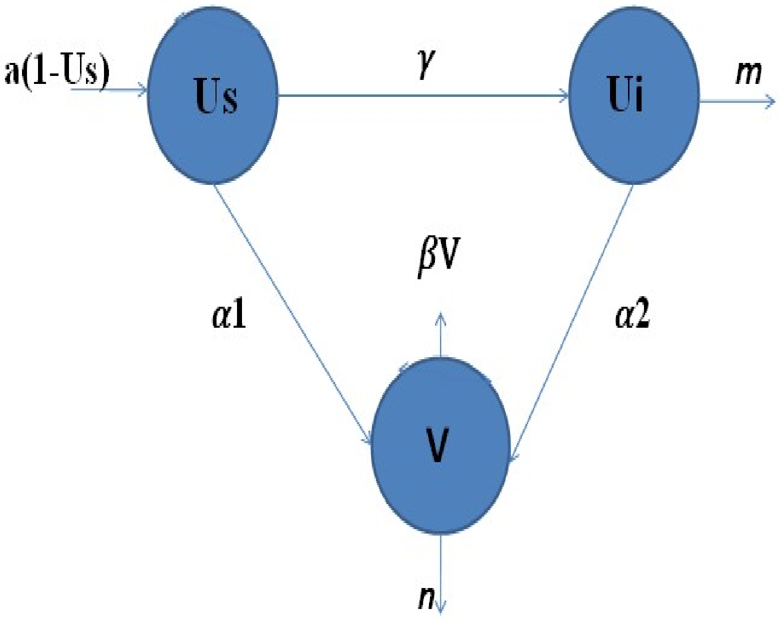

Fig. 1: Flow diagram for the proposed model

Theorem 1. *There exists a unique solution* $\boldsymbol{\tau}(t) = (U_S(t), U_I(t), V(t))$ *to the system with initial condition* $\boldsymbol{\tau}_0 = (U_{S0}, U_{I0}, V_0)$ *for all* $t \geq 0$.

Proof. Define the mapping $\mathscr{F}(\boldsymbol{\tau}) = (F_1(\boldsymbol{\tau}), F_2(\boldsymbol{\tau}), F_3(\boldsymbol{\tau}))$ for $\boldsymbol{\tau}, \bar{\boldsymbol{\tau}} \in \mathscr{X}$ as:

$$F_1(\boldsymbol{\tau}) = U_S\left[a(1-U_S) - \gamma U_I - \alpha_1 V\right], \tag{5}$$
$$F_2(\boldsymbol{\tau}) = U_I\left[\gamma U_S - \alpha_2 V - m\right], \tag{6}$$
$$F_3(\boldsymbol{\tau}) = V\left[\alpha_1 U_S + \alpha_2 U_I - \beta V - n\right]. \tag{7}$$

To prove uniqueness, we apply the Lipschitz condition:

$$\|\mathscr{F}(\boldsymbol{\tau}) - \mathscr{F}(\bar{\boldsymbol{\tau}})\| = |F_1(\boldsymbol{\tau}) - F_1(\bar{\boldsymbol{\tau}})| + |F_2(\boldsymbol{\tau}) - F_2(\bar{\boldsymbol{\tau}})| + |F_3(\boldsymbol{\tau}) - F_3(\bar{\boldsymbol{\tau}})|.$$

Using boundedness assumptions and algebraic manipulations:

$$\begin{aligned}\|\mathscr{F}(\boldsymbol{\tau}) - \mathscr{F}(\bar{\boldsymbol{\tau}})\| \leq{}& (a + 2a\mu + 2\gamma\mu + 2\alpha_1\mu)|U_S - \bar{U}_S| \\ &+ (2\gamma\mu + 2\alpha_2\mu)|U_I - \bar{U}_I| \\ &+ (2\alpha_1\mu + 2\alpha_2\mu + 2\beta\mu + n)|V - \bar{V}|.\end{aligned}$$

Therefore,

$$\|\mathscr{F}(\boldsymbol{\tau}) - \mathscr{F}(\bar{\boldsymbol{\tau}})\| \leq M\|\boldsymbol{\tau} - \bar{\boldsymbol{\tau}}\|,$$

where

$$M = \max\left\{a + 2a\mu + 2\gamma\mu + 2\alpha_1\mu,\ 2\gamma\mu + 2\alpha_2\mu,\ 2\alpha_1\mu + 2\alpha_2\mu + 2\beta\mu + n\right\}.$$

Hence, $\mathscr{F}$ is Lipschitz continuous and the theorem is proved.

4.2 Non-Negativity of the Model

We now demonstrate that the proposed model preserves non-negativity of all population densities:

$$D^{\alpha}U_S(t)\big|_{U_S=0} = 0, \quad (8)$$
$$D^{\alpha}U_I(t)\big|_{U_I=0} = 0, \quad (9)$$
$$D^{\alpha}V(t)\big|_{V=0} = 0. \quad (10)$$

Thus, all the state variables $U_S(t)$, $U_I(t)$, and $V(t)$ remain non-negative for all $t \geq 0$.

5 Equilibrium Points of Model

These equations allow us to determine the equilibrium points of the model. The steady states occur when all time derivatives vanish, i.e.,

$$0 = U_S(t)\left[a(1-U_S(t)) - \gamma U_I(t) - \alpha_1 V(t)\right], \quad (11)$$
$$0 = U_I(t)\left[\gamma U_S(t) - \alpha_2 V(t) - m\right], \quad (12)$$
$$0 = V(t)\left[\alpha_1 U_S(t) + \alpha_2 U_I(t) - \beta V(t) - n\right]. \quad (13)$$

From the above system, the **trivial equilibrium point** (also called the ecological equilibrium) is:

$$E_0 = (0,0,0).$$

Computations yield the following biologically significant equilibrium points:

1. Predator-Free Fixed Point:

$$E_1 = \left(\frac{m}{\gamma}, \frac{-a(m-\gamma)}{\gamma^2}, 0\right). \quad (14)$$

This point exists when the predator species is eradicated. It is stable under the condition $m < \gamma$.

2. Infection-Free Predator-Free Fixed Point:

$$E_2 = (1,0,0). \quad (15)$$

This equilibrium corresponds to an environment where only susceptible prey survive. It exists unconditionally.

3. Infection-Free Fixed Point:

$$E_3 = \left(\frac{a\beta + n\alpha_1}{a\beta + \alpha_1^2}, 0, \frac{-(an - a\alpha_1)}{a\beta + \alpha_1^2}\right). \quad (16)$$

This point guarantees the extinction of infected prey and exists under the condition $\alpha_1 > a$.

4. Susceptible-Free Prey Fixed Point:

$$E_4 = \left(0, \frac{-(m\beta - n\alpha_2)}{\alpha_2^2}, \frac{-m}{\alpha_2}\right). \quad (17)$$

This point implies extinction of susceptible prey. However, this equilibrium is biologically invalid since $m > 0$.

5. Prey-Free Fixed Point:

$$E_5 = \left(0, 0, \frac{-n}{\beta}\right). \quad (18)$$

This prey-free equilibrium is not feasible under the assumption that all parameters are positive.

6. Coexistence Fixed Point:

$$E_6 = \left(\frac{-(-m\beta\gamma + n\gamma\alpha_2 - m\alpha_1\alpha_2 - a\alpha_2^2)}{\beta\gamma^2 + a\alpha_2^2}, \frac{-(am\beta - a\beta\gamma - n\gamma\alpha_1 + m\alpha_1^2 - an\alpha_2 + a\alpha_1\alpha_2)}{\beta\gamma^2 + a\alpha_2^2}, \frac{-(n\gamma^2 - m\gamma\alpha_1 + am\alpha_2 - a\gamma\alpha_2)}{\beta\gamma^2 + a\alpha_2^2} \right). \quad (19)$$

This equilibrium ensures the coexistence of all three species under the following conditions:

$$\frac{m\beta}{n\alpha_2} + \frac{1}{n\gamma}(m\alpha_1 + a\alpha_2) > 1, \quad (20)$$

$$a(\beta\gamma + n\alpha_2) + n\gamma\alpha_1 > a(m\beta + \alpha_1\alpha_2) + m\alpha_1^2, \quad (21)$$

$$(m\alpha_1 + a\alpha_2) > n\gamma + \frac{am\alpha_2}{\gamma}. \quad (22)$$

6 Stability of Equilibrium Points

This section discusses the stability of two significant fixed points: the infection-free fixed point and the coexistence fixed point. The stability is analyzed using the Routh-Hurwitz criterion adapted to fractional-order systems.

The Jacobian matrix of the system (11)–(13) is:

$$J = \begin{bmatrix} -aU_S + a(1 - U_S) - \gamma U_I - \alpha_1 V & -\gamma U_S & -\alpha_1 U_S \\ \gamma U_I & -m + \gamma U_S - \alpha_2 V & -\alpha_2 U_I \\ \alpha_1 V & \alpha_2 V & -n - 2\beta V + \alpha_1 U_S + \alpha_2 U_I \end{bmatrix}$$

6.1 Stability of the Infection-Free Fixed Point

Lemma 1. *The infection-free fixed point $E_{df} = \left(U_S^{df}, 0, V^{df}\right)$ is locally asymptotically stable if $B_2 > 0$, where*

$$B_2 = -a_{11} - a_{22} - a_{33}.$$

The Jacobian evaluated at the infection-free equilibrium yields:

$$U_S^{\text{df}} = \frac{a\beta + n\alpha_1}{a\beta + \alpha_1^2}, \quad V^{\text{df}} = \frac{-(an - a\alpha_1)}{a\beta + \alpha_1^2}$$

$$J(E_{\text{df}}) = \begin{bmatrix} a_{11} & a_{12} & a_{13} \\ 0 & a_{22} & 0 \\ a_{31} & a_{32} & a_{33} \end{bmatrix}$$

where

$$a_{11} = -\frac{a(a\beta + n\alpha_1)}{a\beta + \alpha_1^2}, \quad a_{12} = -\frac{\gamma(a\beta + n\alpha_1)}{a\beta + \alpha_1^2}, \quad a_{13} = -\frac{\alpha_1(a\beta + n\alpha_1)}{a\beta + \alpha_1^2},$$

$$a_{22} = \frac{a\beta(\gamma - m) + \alpha_1(n\gamma - m\alpha_1) + a(n - \alpha_1)\alpha_2}{a\beta + \alpha_1^2},$$

$$a_{31} = \frac{a\alpha_1(\alpha_1 - n)}{a\beta + \alpha_1^2}, \quad a_{32} = \frac{a\alpha_2(\alpha_1 - n)}{a\beta + \alpha_1^2},$$

$$a_{33} = \frac{a\beta(n - \alpha_1)}{a\beta + \alpha_1^2}.$$

The characteristic polynomial becomes:

$$P(\lambda) = \lambda^3 + B_2\lambda^2 + B_1\lambda + B_0,$$

where

$$B_0 = a_{13}a_{22}a_{31} - a_{11}a_{22}a_{33},$$
$$B_1 = a_{11}a_{22} - a_{13}a_{31} + a_{11}a_{33} + a_{22}a_{33},$$
$$B_2 = -a_{11} - a_{22} - a_{33}.$$

If $B_0 = B_1B_2$, the eigenvalues are:

$$\lambda_1 = -i\sqrt{B_1}, \quad \lambda_2 = i\sqrt{B_1}, \quad \lambda_3 = -B_2.$$

The arguments of the eigenvalues satisfy:

$$|\arg(\lambda_1)| = \frac{\pi}{2} > \frac{\alpha\pi}{2}, \quad |\arg(\lambda_2)| = \frac{\pi}{2} > \frac{\alpha\pi}{2}, \quad |\arg(\lambda_3)| = \pi > \frac{\alpha\pi}{2},$$

which confirms the local asymptotic stability when $B_2 > 0$.

6.2 Stability of the Coexistence Fixed Point

Lemma 2. *The coexistence equilibrium point $E^* = (U_S^*, U_I^*, V^*)$ is locally asymptotically stable if $A_2 > 0$, where*

$$A_2 = -b_{11} - b_{33}.$$

Using:

$$U_S^* = \frac{-(-m\beta\gamma + n\gamma\alpha_2 - m\alpha_1\alpha_2 - a\alpha_2^2)}{\beta\gamma^2 + a\alpha_2^2},$$
$$U_I^* = \frac{-(am\beta - a\beta\gamma - n\gamma\alpha_1 + m\alpha_1^2 - an\alpha_2 + a\alpha_1\alpha_2)}{\beta\gamma^2 + a\alpha_2^2},$$
$$V^* = \frac{-(n\gamma^2 - m\gamma\alpha_1 + am\alpha_2 - a\gamma\alpha_2)}{\beta\gamma^2 + a\alpha_2^2}$$

$$J(E^*) = \begin{bmatrix} b_{11} & b_{12} & b_{13} \\ b_{21} & 0 & b_{23} \\ b_{31} & b_{32} & b_{33} \end{bmatrix}$$

where

$$b_{11} = -\frac{a(m\beta\gamma + \alpha_2(-n\gamma + m\alpha_1 + a\alpha_2))}{\beta\gamma^2 + a\alpha_2^2},$$
$$b_{12} = -\frac{\gamma(m\beta\gamma + \alpha_2(-n\gamma + m\alpha_1 + a\alpha_2))}{\beta\gamma^2 + a\alpha_2^2},$$
$$b_{13} = -\frac{\alpha_1(m\beta\gamma + \alpha_2(-n\gamma + m\alpha_1 + a\alpha_2))}{\beta\gamma^2 + a\alpha_2^2},$$
$$b_{21} = \frac{\gamma[a\beta(\gamma - m) + \alpha_1(n\gamma - m\alpha_1) + a(n - \alpha_1)\alpha_2]}{\beta\gamma^2 + a\alpha_2^2},$$
$$b_{23} = \frac{\alpha_2[a\beta(m - \gamma) + \alpha_1(-n\gamma + m\alpha_1) + a(-n + \alpha_1)\alpha_2]}{\beta\gamma^2 + a\alpha_2^2},$$
$$b_{31} = \frac{\alpha_1(-n\gamma^2 + m\gamma\alpha_1 + a(-m + \gamma)\alpha_2)}{\beta\gamma^2 + a\alpha_2^2},$$
$$b_{32} = \frac{\alpha_2(-n\gamma^2 + m\gamma\alpha_1 + a(-m + \gamma)\alpha_2)}{\beta\gamma^2 + a\alpha_2^2},$$
$$b_{33} = \frac{\beta(n\gamma^2 - m\gamma\alpha_1 + a(m - \gamma)\alpha_2)}{\beta\gamma^2 + a\alpha_2^2}.$$

The characteristic polynomial becomes:

$$P(\lambda) = \lambda^3 + A_2\lambda^2 + A_1\lambda + A_0$$

with

$$A_0 = b_{12}b_{21}b_{33} - b_{12}b_{23}b_{31} - b_{13}b_{21}b_{32} + b_{11}b_{23}b_{32},$$
$$A_1 = -b_{12}b_{21} - b_{13}b_{31} - b_{23}b_{32} + b_{11}b_{33},$$
$$A_2 = -b_{11} - b_{33}.$$

If $A_0 = A_1A_2$, then the eigenvalues are:

$$\lambda_1 = -A_2, \quad \lambda_2 = -i\sqrt{A_1}, \quad \lambda_3 = i\sqrt{A_1}$$

and satisfy:

$$|\arg(\lambda_1)| = \pi > \alpha\pi/2, \quad |\arg(\lambda_2)| = \frac{\pi}{2} > \alpha\pi/2, \quad |\arg(\lambda_3)| = \frac{\pi}{2} > \alpha\pi/2,$$

which confirms asymptotic stability for $A_2 > 0$..

7 Numerical Results

The findings of the simulations that back up the theoretical results are presented in this section. The stability of the coexistence fixed point and the time-series solutions of the proposed model are influenced by the fractional-order parameter α.

Effect of Fractional Order α

The phase portraits of the fractional-order model (11)–(13) for various values of the fractional-order parameter α are shown in Figure 2. It is evident from the plots that when $\alpha = 0.1$, the system is unstable. However, for higher values of α, the system exhibits stable behaviour.

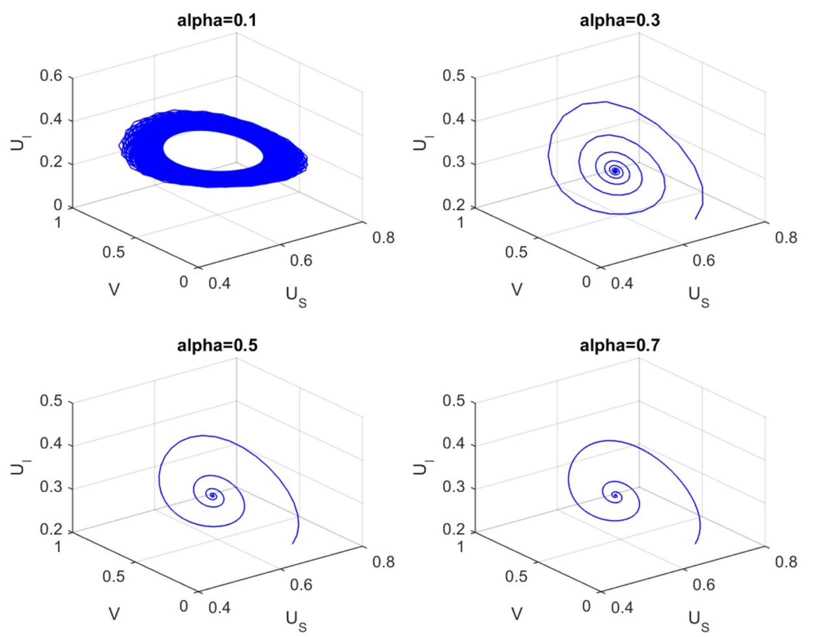

Fig. 2: Phase Portrait at $U_S(0) = 0.40$, $U_I(0) = 0.20$, $V(0) = 0.20$, $a = 0.9$, $\alpha_1 = 0.2$, $\beta = 0.2$, $m = 0.1$, $\alpha_2 = 0.9$, $\gamma = 0.91$, $n = 0.3$

Figure 3 illustrates the time-series solutions of the system for two different values of α. The blue, green, and red curves represent the populations of susceptible prey, infected prey, and predator, respectively. It can be seen that for $\alpha = 0.1$, the solutions diverge, indicating instability, whereas for $\alpha = 0.2$, the time-series converges, confirming the stability of the coexistence equilibrium. All parameters and initial conditions are as specified in Figure **??**.

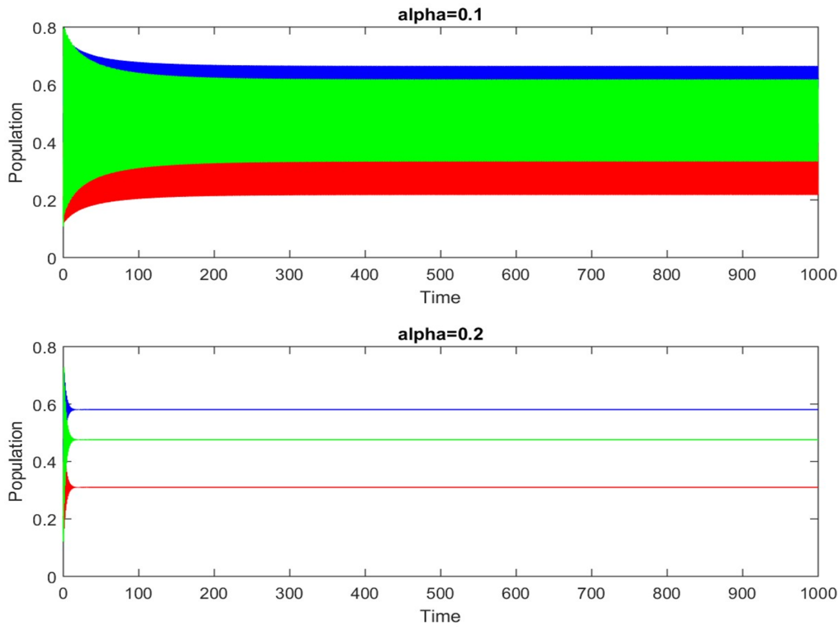

Fig. 3: Time-series solution at $a = 0.1$, where other parameters have the same values as in Fig. 2

Effect of Disease Transmission Parameter γ

Figures 4 shows the impact of varying the disease transmission parameter γ on the phase portraits and time-series solutions, respectively. The values of all other parameters are held constant as given in Figure 2.

In Figure 5, we observe that for smaller values such as $\gamma = 0.5$, the system stabilizes more rapidly. When $\gamma = 0.6$, the system remains stable but takes a longer duration to settle. Larger values of γ disturb the stability and delay convergence toward the coexistence fixed point.

Effect of Intraspecific Competition Parameter β

Figure 6 presents the time-series solutions for different values of the intraspecific competition parameter β. It is observed that the system reaches stability faster with higher β values. Lower values of β delay the convergence of the system toward equilibrium. The same color scheme is used as in Figure 5, where blue denotes susceptible prey, green represents infected prey, and red corresponds to the predator population.

8 Conclusion

We present a dynamically nonlinear eco-epidemiological population fractional model in the current research. The fractional-order derivative enables memory effects, which is a property of population models. In this study, the Caputo fractional-order derivative is employed, which retains the characteristics of epidemic models, including memory effects. We have considered two species, the predator and the prey. A disease in prey species is considered by taking two classes of prey species (susceptible and infected). We have assumed that infection in the prey species spreads non-vertically in

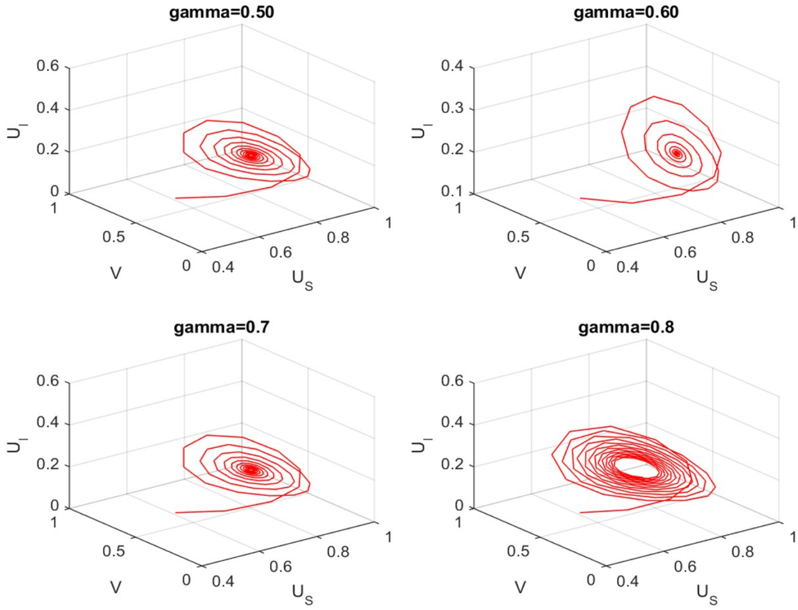

Fig. 4: Phase Portrait at $\alpha = 0.2$, for different values of disease transmission parameter γ

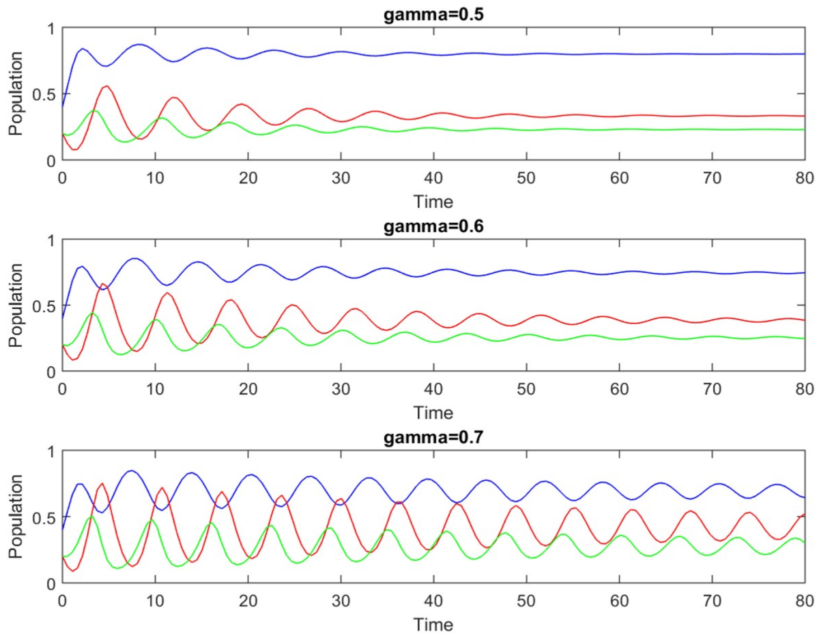

Fig. 5: Time series solution at different values of γ

the prey species. The fixed points of the fractional-order model are calculated. We proved that the model has a unique, non-negative solution. Providing existence requirements allows us to discuss the existence of fixed points.

The study focuses on the stability of infection-free and coexistence-specific points. This coexisting equilibrium point of the original system is shown to be conditionally stable. To validate the analytical findings, we employed a numerical scheme modelled to fractional systems, demonstrating the dynamic behaviour of species populations over time. The simulations revealed the significant roles of fractional order, disease transmission rate, and predation parameters in shaping the stability and long-term behaviour of the ecosystem. Notably, the fractional-order parameter was shown to modulate

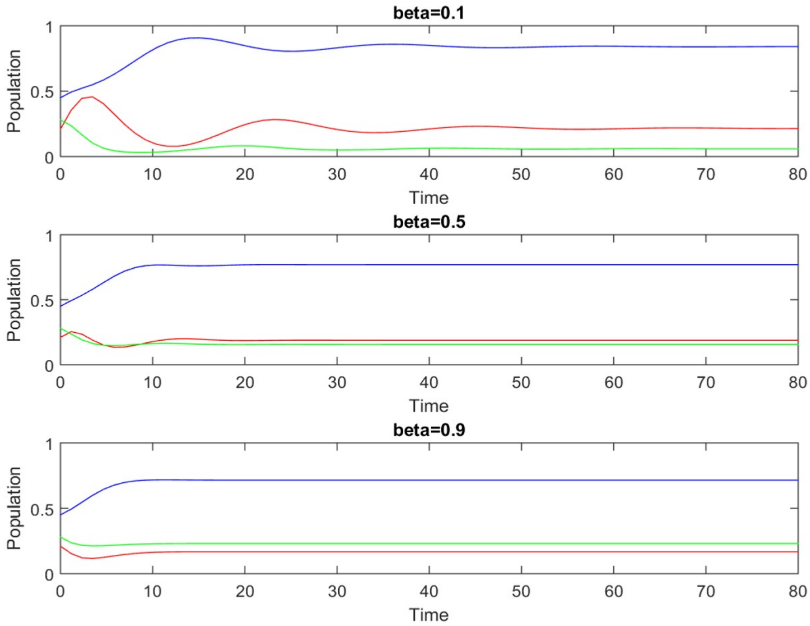

Fig. 6: Time series solution for different values of β

the speed of convergence and the oscillatory nature of solutions, emphasizing the importance of memory in ecological interactions. The study demonstrates the crucial role of fractional parameters in determining the population of species. The impact of fractional order derivatives on the stability of coexistence fixed point is studied. It is shown that for a small value of α, the system is unstable; this fact is evident from Fig. 2. For various values of the fractional-order parameter, the model's time series solution is provided. The effect of the disease transmission rate parameter on the model's solution is investigated for various values of this parameter. It is shown that smaller values of transmission rate parameters cause the system stability earlier.

This research highlights the predictive potential of fractional-order modelling in eco-epidemiological contexts. It provides a flexible framework for studying complex interactions where both disease dynamics and ecological processes are intertwined. The model can be extended in future work to incorporate additional ecological features, such as time delays, spatial diffusion, or control strategies for disease management..

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