Journal of Nonlinear Modeling and Analysis
Volume 8, Number 1, February 2026, 198–216
https://global-sci.com/jnma
DOI:10.12150/jnma.2026.198

Analysis of a Fractional Order Eco-Epidemiological Model with Prey Infection and Predator Cannibalism

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Received 26 May 2025; Accepted 23 September 2025

Abstract This study proposes a fractional-order prey-predator model that integrates disease dynamics within prey populations, fear induced behavioral responses, and predator cannibalism. The prey population is divided into susceptible and infected classes to represent disease dynamics effectively, while predator cannibalism is modeled as a survival strategy during prey scarcity. Essential properties such as non-negativity, boundedness, and the existence and uniqueness of solutions are thoroughly examined. Stability of equilibrium points is established, and global stability is proved using Lyapunov functions. Numerical simulations validate the theoretical results, highlighting how variations in cannibalism rates and fractional-order parameters influence population dynamics and system behavior.

Keywords Predator-prey system, Caputo fractional-order derivative, eco-epidemiological model, cannibalism, stability

MSC(2010) 92D40, 92D30, 92D25, 34D20, 34D23, 34D10

1. Introduction

The interaction between prey and predator is far more diversified and intricate than it might initially appear. Ecological dynamics encompass a wide range of phenomena such as parasitoidism [23], mutualism [21], prey-switching [8], trophic cascades [6], prey defences [45], prey's counter-attacks [20], and interspecies competition [44]. A particularly compelling aspect of predator-prey relationships is cannibalism, or intraspecific predation the act of killing and consuming individuals of the same species. Cannibalism is remarkably widespread in nature, documented in over 1300 species [36]. For example, male lions may consume unrelated cubs after

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taking control of a pride to maximize the reproductive success of their lineage [47]; octopuses are known to feed on their juveniles [9,17]; and certain females consume non-viable or sick eggs to prevent infection [18]. This behavior is not limited to a narrow group: it occurs across a wide spectrum of species including fishes [16], frogs [32], insects [41], primates [15], and birds [19]. Drivers of cannibalism can include food scarcity, developmental stage, sex-based behaviors, famine, humidity, and other environmental or physiological pressures [36,48]. These factors position cannibalism as an adaptive, though grim, strategy for survival and reproduction within the natural world.

In recent years, the profound influence of cannibalism on ecological systems has prompted researchers to incorporate it into mathematical models of population dynamics. Cannibalistic behavior has been modeled in predator species [31,49], prey species [33,37], and even within both populations simultaneously [4]. These models often extend to include additional ecological complexities such as time delays, infectious diseases, prey refuge mechanisms, and other modifying factors [27,46,50]. Analyses of such models reveal that cannibalism can exert either a stabilizing effect [46] or a destabilizing one [29] on population dynamics.

Cannibalism is sometimes referred to as the “lifeboat mechanism” because it can prevent the extinction of predator populations by providing an alternative energy source. Cannibalism and fear effects significantly influence predator-prey interactions. It allows predators to regulate their own populations, thereby reducing oscillations and promoting ecological stability. Fear effects, triggered by predators, alter prey behavior and limit their availability, which further impacts population dynamics and system balance. The combined impact of these factors creates a stabilizing force within ecosystems, as demonstrated in recent studies [7,25,31,33,50].

Species behavior is profoundly shaped by past experiences. For example, salamanders reduce foraging in the presence of garter snakes [28], while guppies modulate breeding based on predator-induced chemical cues [14]. Numerous adaptive responses such as altered foraging strategies, habitat preferences, and life-history traits emerge from these experiences and directly influence population dynamics [5]. In mathematical modeling, environmental parameters like reproduction and mortality rates vary over time, driven by psychological imprints retained by individuals. Recent experiences typically exert stronger influence due to the natural fading of older memories [10]. Capturing these evolving behavioral nuances is best achieved through fractional-order derivatives, which account for memory-dependent dynamics within interacting populations [40].

Fractional calculus has emerged as a powerful mathematical tool in recent years, finding widespread applications across diverse fields such as engineering, physics, economics, and applied sciences. Its significance lies in its ability to enhance the modeling and analysis of complex systems, especially in solving inverse problems that require fitting data with greater accuracy [3,11,22,24,38,39,42,43]. Unlike classical calculus, fractional calculus introduces an additional parameter – the order of the derivative within mathematical models, providing a flexible and refined approach for data adjustment and prediction. By incorporating fractional derivatives, researchers can achieve improved alignment with real-world data, resulting in more reliable predictions for the evolution of dynamic systems. This approach has proven particularly valuable in applications such as disease spread modeling, where the incorporation of fractional-order enhances the predictive capabilities and accuracy of epidemiological models [11,22,38,39,42]. As fractional calculus continues to demonstrate its potential, it has become an indispensable tool in advancing research across scientific disciplines.

This eco-epidemiological model explores the interplay between prey and predator populations. Within the prey population, there are two distinct groups: the susceptible prey, represented by $(S(t))$, and the infected prey, represented by $(I(t))$. At any given time (t), the overall prey population is expressed as $(N(t) = S(t) + I(t))$. The predator population, denoted by $(P(t))$, faces an increased mortality rate as a result of consuming infected prey. This model has been developed based on the following assumptions:

A1) It is assumed that the disease spreads through interactions between susceptible prey and infected prey in accordance with the fundamental concepts of the mass action law.

A2) The model does not consider the recovery of infected prey due to their rapid death from the disease. This quick mortality prevents them from reproducing or recovering.

A3) The susceptible population grows logistically when predators and infected prey are absent ($I(t) = 0$ and $P(t) = 0$).

A4) The term $\frac{rS}{1+fP}$ can be expressed as follows:

$$\frac{rS}{1+fP} = S \cdot \frac{r}{1+fP},$$

where the first factor S, represents the size of the susceptible prey population, and the second factor $\frac{r}{1+fP}$, accounts for the growth rate of the susceptible prey, reduced by the predator population fP that induces fear, f represents the level of fear.

- When $f \to 0$, $\frac{r}{1+fP} \to r$, indicating that the predator population is absent, allowing prey to grow at their maximum natural rate.
- When $f \to \infty$, $\frac{r}{1+fP} \to 0$, suggesting that a very high predator population suppresses prey population growth entirely.
- Thus, $0 < \frac{r}{1+fP} < r$, where r represents the maximum growth rate of the susceptible prey in the absence of predators.

Based on the assumptions outlined above, we propose the following system of ordinary differential equations to represent the described phenomenon:

$$\begin{aligned} \frac{dS(t)}{dt} &= \frac{rS}{1+fP}\left(1 - \frac{S+I}{K}\right) - \lambda SI, \\ \frac{dI(t)}{dt} &= \lambda SI - \mu I - \frac{mIP}{a+I}, \\ \frac{dP(t)}{dt} &= \frac{\theta IP}{a+I} - dP - \frac{nP^2}{b+P}, \end{aligned} \tag{1.1}$$

with the given initial values $S(0) \geq 0,\ I(0) \geq 0,\ P(0) \geq 0$; the detailed biological meanings of the parameters used in the model are provided in Table 1. For $0 < \alpha \leq 1$, we propose the fractional-order eco-epidemiological model that aligns with model (1.1):

$${}^{c}D^{\alpha} S(t) = \frac{rS}{1+fP}\left(1 - \frac{S+I}{K}\right) - \lambda SI,$$

Parameter	Description
r	Growth rate of susceptible prey population
K	Environmental carrying capacity for prey
λ	Disease transmission rate
μ	Mortality rate of infected prey
m	Interaction rate between infected prey and predators
a	Half-saturation constant for infected prey
θ	Predation rate on infected prey
d	Death rate of predator population
n	Competition coefficient for predator cannibalism
b	Half-saturation constant for predator population

Table 1. Biological meanings of the parameters used in the model.

$$\begin{aligned} {}^cD^\alpha I(t) &= \lambda SI - \mu I - \frac{mIP}{a+I}, \\ {}^cD^\alpha P(t) &= \frac{\theta IP}{a+I} - dP - \frac{nP^2}{b+P}, \end{aligned} \tag{1.2}$$

with initial conditions $S(0) \geq 0,\ I(0) \geq 0,\ P(0) \geq 0$. For $0 < \alpha < 1$, where $({}^cD^\alpha)$ represents the standard Caputo differentiation, while the parameters of the fractional-order eco-epidemiological model (1.2) are considered positive. The parameters are summarized in Table 1, with the Caputo fractional derivative of order α defined as shown below:

$${}^cD^\alpha f(t) = \frac{1}{\Gamma(n-\alpha)} \int_0^t (t-s)^{n-\alpha-1} f^{(n)}(s)ds, \quad n-1 < \alpha < n, n \in \mathbb{N}.$$

This paper is structured as follows. The upcoming section delves into the analysis of the fractional-order eco-epidemiological model (1.2). Section 3 gives the local stability and we construct a specific Lyapunov function in Section 4 to establish a suitable condition for the global stability. Lastly, Section 5 presents numerical simulations to validate the theoretical findings.

2. Mathematical analysis

This section presents the fractional-order eco-epidemiological model (1.2) and its mathematical analysis.

2.1. Existence and uniqueness

In the domain $(\Lambda \times (0, T])$, the existence and uniqueness of solutions for the fractional-order system (1.2) are explored, where

$$\Lambda = \{(S, I, P) \in \mathbb{R}^3 : \max(|S|, |I|, |P|) \leq \Psi\}.$$

Theorem 2.1. *For every* ($X_0 = (S_0, I_0, P_0) \in \Lambda$)*, there exists a unique solution* ($X(t) \in \Lambda$) *to the fractional-order system (1.2), satisfying the initial condition* (X_0) *and satisfied for all* ($t \geq 0$)*.*

Proof. For $X, \bar{X} \in \Lambda$, one can consider a mapping

$$M(X) = (M_1(X), M_2(X), M_3(X))$$

where

$$\begin{aligned}
M_1(X) &= \frac{rS}{1+fP}\left(1 - \frac{S+I}{K}\right) - \lambda SI,\\
M_2(X) &= \lambda SI - \mu I - \frac{mIP}{a+I},\\
M_3(X) &= \frac{\theta IP}{a+I} - dP - \frac{nP^2}{b+P}.
\end{aligned}$$

$$\|M(X) - \overline{M}(\overline{X})\| = |M_1(X) - M_1(\overline{X})| + |M_2(X) - M_2(\overline{X})| + |M_3(X) - M_3(\overline{X})|,$$

with

$$\begin{aligned}
|M_1(X) - M_1(\overline{X})| &= \left| \frac{rS}{1+fP}\left(1 - \frac{S+I}{K}\right) - \lambda SI - \frac{r\overline{S}}{1+f\overline{P}}\left(1 - \frac{\overline{S}+\overline{I}}{K}\right) + \lambda\overline{SI} \right|\\
&= \left| \frac{rS(1+f\overline{P}) - r\overline{S}(1+fP)}{(1+fP)(1+f\overline{P})} - \frac{rS^2(1+f\overline{P}) - r\overline{S}^2(1+fP)K}{(1+fP)(1+f\overline{P})K} \right.\\
&\qquad \left. - \frac{rSI(1+f\overline{P})K - r\overline{SI}(1+fP)K}{(1+fP)(1+f\overline{P})K} - \lambda(IS - \overline{IS}) \right|\\
&= \left| \frac{r(S-\overline{S}) + rf(S\overline{P} - \overline{S}P)}{(1+fP)(1+f\overline{P})} - \frac{r(S^2 - \overline{S}^2) + rf(S^2\overline{P} - \overline{S}^2P)}{(1+fP)(1+f\overline{P})} \right.\\
&\qquad \left. - \frac{r(SI - \overline{SI}) + rf(SI\overline{P} - \overline{SI}P)}{(1+fP)(1+f\overline{P})} - \lambda\big[S(I-\overline{I}) + \overline{I}(S-\overline{S})\big] \right|\\
&\leq \Big[r(1-3\Psi) + rf\Psi(1-\Psi) - \lambda\Psi\Big]|S-\overline{S}| + \Big[r\Psi(f\Psi - 1) - \lambda\Psi\Big]\\
&\quad |I - \overline{I}| - rf\Psi|P - \overline{P}|.
\end{aligned}$$

Similarly, for $|M_2(X) - M_2(\overline{X})|$ and $|M_3(X) - M_3(\overline{X})|$, we have

$$\begin{aligned}
|M_2(X) - M_2(\overline{X})| &= \left| \lambda SI - \mu I - \frac{mIP}{a+I} - \lambda\overline{SI} + \mu\overline{I} + \frac{m\overline{IP}}{a+\overline{I}} \right|\\
&\leq \lambda\Psi|S - \overline{S}| + \Big[\lambda\Psi - \mu - ma\Psi\Big]|I - \overline{I}| + m\Psi(\Psi - a)|P - \overline{P}|,
\end{aligned}$$

and

$$|M_3(X) - M_3(\overline{X})| = \left| \frac{\theta IP}{a+I} - dP - \frac{nP^2}{b+P} + \frac{\theta\overline{IP}}{a+\overline{I}} - d\overline{P} + \frac{n\overline{P}^2}{b+\overline{P}} \right|$$

$$\leq \theta a\Psi|I-\overline{I}| + \Big[\theta\Psi(a+\Psi) - n\Psi(2b+\Psi)\Big]|P-\overline{P}|.$$

Therefore,

$$\begin{aligned}
&\|M(X)-\overline{M}(\overline{X})\| \\
=&|M_1(X)-M_1(\overline{X})| + |M_2(X)-M_2(\overline{X})| + |M_3(X)-M_3(\overline{X})| \\
\leq&\Big[r(1-3\Phi)+fr\Phi(1-\Phi)\Big]|S-\overline{S}| + \Big[r\Phi(f\Phi-1)-\mu+a\Phi(\theta-\Phi)\Big] \\
&|I-\overline{I}| + \Big[\theta\Phi(a+\Phi)-n\Phi(a+2b)-rf\Phi\Big]|P-\overline{P}| \\
\leq&\mathbb{L}||X-\overline{X}||,
\end{aligned}$$

with

$$\begin{aligned}
\mathbb{L} =&max\{r(1-3\Phi)+fr\Phi(1-\Phi), r\Phi(f\Phi-1)-\mu+a\Phi(\theta-\Phi), \theta\Phi(a+\Phi) \\
&-n\Phi(a+2b)-rf\Phi\}.
\end{aligned}$$

Therefore, $M(X)$ satisfies the Lipschitz condition, proving that solutions to the fractional-order eco-epidemiological model (1.2) exist and are unique. ☐

2.2. Non-negativity and boundedness

The following results verify the non-negative nature of the solutions for the Caputo fractional differential system (1.2).

Lemma 2.1. *[34] Let $f(t) \in C[a,b]$ and $D_a^\alpha f(t) \in C(a,b]$ for $0 < \alpha \leq 1$. Then*

$$f(t) = f(a) + \frac{1}{\Gamma(\alpha)}(D_a^\alpha f)(\xi)(t-a)^\alpha,$$

where $a \leq \xi \leq x$, $(\forall x \in (a,b]$.

Lemma 2.2. *[26] Suppose $u(t)$ is a continuous function on $[t_0,\infty)$ that satisfies*

$${}^cD^\alpha u(t) \leq -au(t)+b, \quad u(t_0) = u_{t_0},$$

where $0 < \alpha < 1$, $(a,b) \in \mathbb{R}^2$, $a \neq 0$ and $t_0 \geq 0$ is the initial time. Therefore, the solution assumes the form

$$\nu(t) \leq \left(u_{t_0} - \frac{b}{a}\right) E_\alpha[-a(t-t_0)^\alpha] + \frac{b}{a}.$$

Theorem 2.2. *The fractional-order system (1.2) guarantees that $\mathbb{R}_+^3$ is a positively invariant set.*

Proof. We assume that $S(t) \geq 0$ for all $t \geq 0$ and consider the case where this assumption is false. Specifically, there exists a time $t_1 > 0$ such that $S(t)$ is positive for $0 \leq t < t_1$, reaches zero at $t = t_1$, and becomes negative for $t > t_1$. This contradiction establishes the required non-negativity of $S(t)$ for all $t \geq 0$.

$${}^cD^\alpha S(t)|_{t=t_1} = 0.$$

Based on Lemma 2.1, we have $S(t_1^+) = 0$, which is in direct conflict with the condition $S(t_1^+) < 0$, implying that $S(t) < 0$ holds true for $t > t_1$. Hence, we establish that $S(t) \geq 0$ for all $t \geq 0$. Applying similar reasoning, we also prove that $I(t) \geq 0$ for all $t \geq 0$ and $P(t) \geq 0$ for all $t \geq 0$. Next, we demonstrate that every solution of system (1.2) originating in $\mathbb{R}^3_+$ remains uniformly bounded.

$$F(t) = S(t) + I(t) + \frac{m}{\theta}P.$$

By applying the fractional time derivative, we arrive at the following expression:

$${}^cD^\alpha F(t) = r\left(1 - \frac{S+I}{K}\right)S - \frac{md}{\theta}P - \frac{m}{\theta}\frac{nP^2}{b+P} - \mu I.$$

For all $\tau > 0$, the following holds:

$$\begin{aligned} {}^cD^\alpha F(t) + \tau F(t) &= rS\left(1 - \frac{S+I}{K}\right) - \mu I - \frac{md}{\theta}P - \frac{m}{\theta}\frac{nP^2}{b+P} + \tau S + \tau I + \frac{\tau m}{\theta}P \\ &= -rS^2 + (r+\gamma)S - \frac{mn}{\theta}P^2 + (\gamma - \mu) + \frac{m}{\theta}(\gamma - d)P \\ &\leq \frac{(r+\gamma)^2}{4r} + \frac{(r-d)^2}{4mn}. \end{aligned}$$

Taking $\tau < \min(\mu, d)$, we ensure:

$${}^cD^\alpha F(t) + \tau F(t) \leq \frac{(r+\gamma)^2}{4r} + \frac{(r-d)^2}{4mn},$$

where $l = \frac{(r+\gamma)^2}{4r} + \frac{(r-d)^2}{4mn} > 0$. Applying Lemma 2.2, we get

$$\begin{aligned} F(t) &\leq F(0) - \frac{l}{\tau}E_\alpha[1 - \tau^{\alpha t}] + \frac{l}{\tau} \\ &\leq F(0)E_\alpha[1 - \tau^{\alpha t}] + \frac{l}{\tau}(1 - E_\alpha[1 - \tau^{\alpha t}]). \end{aligned}$$

Thus, $F(t) \to \frac{l}{\tau}$ as $t \to \infty$ and $0 < F(t) \leq \frac{l}{\tau}$. Thus, every solution of system (1.2) originating in $\mathbb{R}^3_+$ remains bounded within the specified region

$$\Theta = \{(S, I, P) \in \mathbb{R}^3_+ | F(t) \leq \frac{l}{\tau} + \epsilon, \text{ for any } \epsilon > 0\}.$$

□

3. Equilibrium points and stability analysis

The local stability of the Caputo fractional differential system (1.2) around its biologically feasible equilibrium points has been analyzed in this study. To achieve this, the fractional basic reproduction number $\mathcal{R}_0 = \frac{\lambda K}{\mu}$, was computed for the model by using the next generation method [13]. This calculation was performed to determine the conditions that govern both the existence and stability of the equilibrium points in the system.

3.1. Equilibrium points

We find equilibrium points by setting the derivatives to zero:

$$\begin{aligned} \frac{rS}{1+fP}\left(1-\frac{S+I}{K}\right)-\lambda SI&=0,\\ \lambda SI-\mu I-\frac{mIP}{a+I}&=0,\\ \frac{\theta IP}{a+I}-dP-\frac{nP^2}{b+P}&=0. \end{aligned} \tag{3.1}$$

1. The disease-free and predator extinction equilibrium point
$$E_1=(k,0,0).$$
2. The predator extinction equilibrium point.
$$E_2=\left(\frac{\mu}{\lambda},\frac{r\mu(\mathcal{R}_0-1)}{\lambda(r-\lambda k)},0\right).$$
3. The coexistence equilibrium point $E^*=(S^*,I^*,P^*)$ with
$$S^*=1+\frac{I}{K}\frac{(1+fP)I\lambda}{r},$$
$$I^*=-a+\frac{mP}{\mu(\mathcal{R}_0-1)},$$
and P^* is the unique positive root of the quadratic equation
$$AP^2+BP+C=0,$$
where,
$$A=\frac{m}{\mu(\mathcal{R}_0-1)}(n+(d-\theta)),B=(a\theta-\frac{bm}{\mu(\mathcal{R}_0-\mu)}(d-\theta)),C=ab\theta.$$

The equilibrium E_1 is always present. The planar equilibrium E_2 occurs when $r>\lambda K$ and $\mathcal{R}_0>1$, where $\mathcal{R}_0=\frac{\lambda K}{\mu}$. The condition $\mathcal{R}_0>1$ combined with $AC>0$ guarantees the existence of the interior equilibrium E^*.

3.2. Local stability analysis

Using the Jacobian matrix and the Routh-Hurwitz stability criteria [1, 2, 35], the local stability of the equilibrium points for the Caputo fractional differential system (1.2) is examined.

Theorem 3.1. *[30] Consider that the following fractional-order system is of the form*

$$\begin{aligned} {}^cD^{\alpha}x(t)&=f(t,x),\quad 0<q<1,\\ x(0)&=x_0, \end{aligned} \tag{3.2}$$

where $f(t,x):\mathbb{R}^+\times\mathbb{R}^n\to\mathbb{R}^n$. For the Caputo fractional differential system the equilibrium points are locally asymptotically stable if all eigenvalues μ_i of the Jacobian matrix $\frac{\partial f(t,x)}{\partial x}$ evaluated at the equilibrium points satisfy the following condition:

$$|\arg(\mu_i)|>\frac{q\pi}{2}. \tag{3.3}$$

The Jacobian matrix of system (1.2) is given by

$$J(S,I,P)$$
$$= \begin{bmatrix} \frac{r}{1+fP}\left(1-\frac{S+I}{K}\right)-\frac{rS}{K(1+fP)}-\lambda I & -\frac{rS}{K(1+fP)}-\lambda S & -\frac{rSf}{(1+fP)^2}\left(1-\frac{S+I}{K}\right) \\ \lambda I & \lambda S-\mu-\frac{mPa}{(a+I)^2} & -\frac{mI}{a+I} \\ 0 & \frac{\theta aP}{(a+I)^2} & \frac{\theta I}{a+I}-\frac{2nP(b+P)-nP^2}{(b+P)^2}-d \end{bmatrix}.$$

Theorem 3.2. *Assume that $\mathcal{R}_0 < 1$. Then the disease-free and predator extinction equilibrium point E_1 is locally asymptotically stable.*

Proof. The Jacobian matrix associated with E_1 is expressed as:

$$\begin{bmatrix} -r-\lambda K & 0 & 0 \\ 0 & \lambda K-\mu & 0 \\ 0 & 0 & -d \end{bmatrix}.$$

The eigenvalues of this matrix are $\theta_1 = -r(< 0)$, $\theta_2 = \lambda K - \mu$, and $\theta_3 = -d(< 0)$. To analyze the stability of E_1, two cases are considered based on the value of $\mathcal{R}_0$.

Case 1: If $\mathcal{R}_0 < 1$, it follows that $|\arg(\theta_i)| = \pi > \frac{q\pi}{2}$ for all $q \in (0,1]$ and $i = 1,2,3$. Hence, the equilibrium E_1 is locally asymptotically stable.

Case 2: If $\mathcal{R}_0 > 1$, then $|\arg(\theta_2)| = 0$, indicating that E_1 is unstable. □

Theorem 3.3. *Assume that $a_1 > 0, a_2 > 0$ and $\mathcal{R}_0 > 1$. Then the disease-free and predator extinction equilibrium point E_2 is locally asymptotically stable.*

Proof. The Jacobian matrix of $J(E_2)$ is given by

$$\begin{bmatrix} r\left(1-\frac{\mu}{\lambda K}-\frac{r\mu(\mathcal{R}_0-1)}{\lambda K(r-\lambda K)}\right) & -\frac{r\mu}{\lambda K} & 0 \\ \lambda I_2 & 0 & \frac{-mI_2}{a+I_2} \\ 0 & 0 & \frac{\theta I_2}{a+I_2}-d \end{bmatrix}.$$

The characteristic equation corresponding to the Jacobian matrix $J(E_2)$ can be represented as:

$$\left(\theta - \frac{\theta I_2}{a+I_2} - d\right)(\theta^2 + a_1\theta + a_2) = 0, \tag{3.4}$$

where $a_1 = r\left(1-\frac{\mu}{\lambda K}+\frac{r\mu(\mathcal{R}_0-1)}{\lambda K(r-\lambda K)}\right) > 0$ and $a_2 = -\frac{r\mu I_2}{K} > 0$. Therefore, one eigenvalue is $\theta_1 = \frac{\theta I_2}{a+I_2} - d$ and the other two are given by $\theta_{2,3} = \frac{1}{2}(-a_1 \pm \sqrt{a_1^2 - 4a_2})$. By Theorem 3.1, $|\arg(\theta)| = q$ if $\frac{\theta I_2}{a+I_2} - d < 0$. On the other hand, note that the quadratic polynomial $\theta^2 + a_1\theta + a_2 = 0$ is Hurwitz if: $a_1 > 0$ and $a_2 > 0$. This ensures that the eigenvalues of the polynomial $\theta^2 + a_1\theta + a_2 = 0$ have a negative real part. Thus, by Theorem 3.1, $|\arg(\theta)| = \pi$ if and only if $\mathcal{R}_0 > 1$.

Therefore, the predator-free equilibrium point E_2 is locally asymptotically stable. □

For the interior equilibrium E^*, the Jacobian matrix is evaluated as:

$$J(S^*, I^*, P^*) = \begin{bmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{bmatrix},$$

where the entries are defined as follows:

$$\begin{aligned}
a_{11} &= \frac{r}{1+fP^*}\left(1 - \frac{S^*+I^*}{K}\right) - \frac{rS^*}{K(1+fP^*)} - \lambda I^*, \\
a_{12} &= -\frac{rS^*}{K(1+fP^*)} - \lambda S^*, \\
a_{13} &= -\frac{rS^* f}{(1+fP^*)^2}\left(1 - \frac{S^*+I^*}{K}\right), \\
a_{21} &= \lambda I^*, \\
a_{22} &= \lambda S^* - \mu - \frac{mP^* a}{(a+I^*)^2}, \\
a_{23} &= -\frac{mI^*}{a+I^*}, \\
a_{31} &= 0, \\
a_{32} &= \frac{\theta a P^*}{(a+I^*)^2}, \\
a_{33} &= \frac{\theta I^*}{a+I^*} - \frac{2nP^*(b+P^*) - n(P^*)^2}{(b+P^*)^2} - d.
\end{aligned}$$

The characteristic equation of the Jacobian matrix is given by:

$$\lambda^3 + N_1\lambda^2 + N_2\lambda + N_3 = 0, \tag{3.5}$$

where:

$$\begin{aligned}
N_1 &= -(a_{11} + a_{22} + a_{33}), \\
N_2 &= a_{11}a_{22} + a_{22}a_{33} + a_{11}a_{33} - a_{12}a_{21} - a_{23}a_{32}, \\
N_3 &= a_{11}a_{23}a_{32} + a_{12}a_{21}a_{33} - a_{11}a_{22}a_{33}.
\end{aligned}$$

To determine the stability of the interior equilibrium point, we evaluate the discriminant $D(p)$ of the polynomial (3.5) in the following form:

$$D(p) = 18N_1N_2N_3 + (N_1N_2)^2 - 4N_1^3N_3 - 4N_2^3 - 27N_3^2.$$

By applying the Routh-Hurwitz criteria for fractional calculus, the stability conditions of the positive equilibrium are established and summarized in the following theorem.

Theorem 3.4. *The interior equilibrium point E^* is locally asymptotically stable if any one of the following conditions holds:*

- $D(p) > 0, \quad N_1 > 0, \quad N_3 > 0, \quad N_1N_2 > N_3.$
- $D(p) < 0, \quad N_1 \geq 0, \quad N_2 \geq 0, \quad N_3 > 0, \quad \beta < \frac{2}{3}.$
- $D(p) < 0, \quad N_1 > 0, \quad N_2 > 0, \quad N_1N_2 = N_3, \quad \beta \in (0, 1).$

4. Global stability

Next, we establish the global stability of the interior equilibrium point for the system (1.2).

Lemma 4.1. *[26] Assume that $x(t) \in \mathbb{R}^+$ denote a continuous and differentiable function. Then, for all $t > t_0$, it holds that:*

$$ {}^{c}_{t_0}D^{\alpha}_{t}\left[x(t) - x^* - x^* \ln\left(\frac{x(t)}{x^*}\right)\right] \leq \left(1 - \frac{x^*}{x(t)}\right)\ {}^{c}_{t_0}D^{\alpha}_{t}x(t), $$

where $x^* \in \mathbb{R}^+$ and $\forall \alpha \in (0,1)$.

Theorem 4.1. *$E^* = (S^*, I^*, P^*)$ the interior equilibrium point is globally asymptotically stable if $\frac{-rf(1-(S^*+I^*))}{(1+fP)((1+fP^*)} > 0$ and $\frac{-mI^*+a(\theta-m)}{(a+I^*)(a+I^*)} > 0$.*

Proof. To demonstrate the global stability of E^*, we construct the Lyapunov function as follows:

$$ \vartheta(S,I,P) = \left(S - S^* - S^* \ln\frac{S}{S^*}\right) + \left(I - I^* - I^* \ln\frac{I}{I^*}\right) + \frac{m}{\theta}\left(P - P^* - P^* \ln\frac{P}{P^*}\right). $$

By applying the fractional-order derivative to both sides, we obtain:

$$
\begin{aligned}
&{}^{c}D^{\alpha}\vartheta(S,I,P)\\
&\leq \frac{(S-S^*)}{S}\ {}^{c}D^{q}S(t) + \frac{(I-I^*)}{I}\ {}^{c}D^{q}I(t) + \frac{m(P-P^*)}{\theta P}\ {}^{c}D^{q}P(t)\\
&= (S-S^*)\left[\frac{r}{1+fP}\left(1-\frac{S+I}{K}\right) - \lambda I\right] + (I-I^*)\left[\lambda S - \mu - \frac{mP}{a+I}\right]\\
&\quad + (P-P^*)\left[\frac{\theta I}{a+I} - d - \frac{nP}{b+P}\right]\\
&= (S-S^*)\left[\frac{r(1+fP^*) - r(1+fP)}{(1+fP)(1+fP^*)} - \frac{rS(1+fP^*) - rS^*(1+fP)}{(1+fP)(1+fP^*)K}\right]\\
&\quad \left[-\frac{rI^*(1+fP^*) - rI(1+fP)}{(1+fP)((1+fP^*)K)} - \lambda(I-I^*)\right] + (I-I^*)\lambda(S-S^*)\\
&\quad - \frac{mP(a+I^*) - mP^*(a+I)}{(a+I^*)(a+I^*)} - (P-P^*)\\
&\quad \left[\frac{\theta I(a+I^*) - \theta I^*(a+I)}{(a+I^*)(a+I)} - \frac{nP(b+P^*) - nP^*(b+P)}{(b+P^*)(b+P)}\right]\\
&= \frac{-rf(S-S^*)(P-P^*)}{(1+fP)((1+fP^*)} - \frac{r(S-S^*)^2}{(1+fP)((1+fP^*)K} - \frac{rfP^*(S-S^*)^2}{(1+fP)((1+fP^*)K}\\
&\quad + \frac{rfS^*(P-P^*)(S-S^*)}{(1+fP)((1+fP^*)K} - \frac{rfP^*(I-I^*)(S-S^*)}{(1+fP)((1+fP^*)K} + \frac{rfI^*(P-P^*)(S-S^*)}{(1+fP)((1+fP^*)K}\\
&\quad - \frac{ma(P-P^*)(I-I^*)}{(a+I^*)(a+I^*)} - \frac{mI^*(P-P^*)(I-I^*)}{(a+I^*)(a+I^*)} + \frac{mP^*(I-I^*)^2}{(a+I^*)(a+I^*)}\\
&\quad + \frac{\theta a(I-I^*)(P-P^*)}{(a+I^*)(a+I^*)} - \frac{nb(P-P^*)^2}{(b+P^*)(b+P)}\\
&\leq \left[\frac{-rf(1-(S^*+I^*))}{(1+fP)((1+fP^*)}\right](P-P^*)(S-S^*) - \left[\frac{r(fP^*+r)}{(1+fP)((1+fP^*)}\right](I-I^*)
\end{aligned}
$$

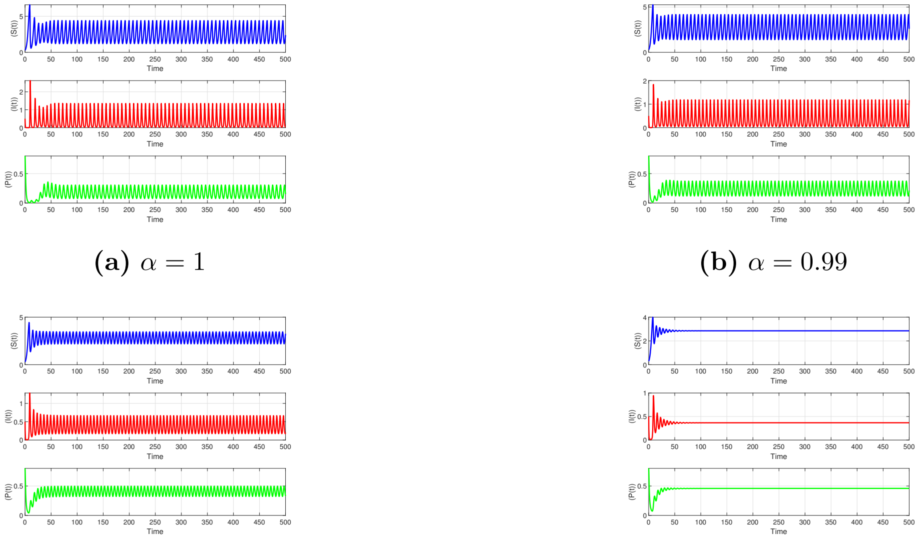
$$(S-S^*)-\left[\frac{-mI^*+a(\theta-m)}{(a+I^*)(a+I^*)}\right](P-P^*)(I-I^*)-\frac{r(1+fP^*)}{(1+fP)((1+fP^*)}$$
$$(S-S^*)^2-\frac{mP^*}{(a+I^*)(a+I^*)}(I-I^*)^2-\frac{nb}{(b+P^*)(b+P)}(P-P^*)^2.$$

□

Observe that $\frac{-rf(1-(S^*+I^*))}{(1+fP)((1+fP^*)}>0$, $\frac{-mI^*+a(\theta-m)}{(a+I^*)(a+I^*)}>0$. In this case ${}^cD^\alpha\vartheta(S,I,P)\le 0$, $\forall(S,I,P)\in\mathbb{R}^3_+$, and ${}^cD^\alpha\vartheta(S,I,P)=0$ at (S^*,I^*,P^*). By Lemma 4.1, whenever the interior equilibrium E^* exists, E^* is globally asymptotically stable.

5. Numerical computations

Numerical simulations for system (1.2) are extensively carried out across various fractional orders $0<\alpha\le 1$. The Adamas-type predictor-corrector method is employed for solving the fractional-order differential equations [12].



(a) $\alpha=1$ **(b)** $\alpha=0.99$

(c) $\alpha=0.95$ **(d)** $\alpha=0.90$

Figure 1. Time series of system (1.2) changes from oscillatory behavior to stable equilibrium by varying fractional-order ($\alpha=1$, $\alpha=0.99$, $\alpha=0.95$, and $\alpha=0.90$)

$$\begin{aligned}S(t)&=S(0)+D_t^{-\alpha}\left(\frac{rS}{1+fP}\left(1-\frac{S+I}{K}\right)-\lambda SI\right),\\ I(t)&=I(0)+D_t^{-\alpha}\left(\lambda SI-\mu I-\frac{mIP}{a+I}\right),\qquad(5.1)\\ P(t)&=P(0)+D_t^{-\alpha}\left(\frac{\theta IP}{a+I}-dP-\frac{nP^2}{b+P}\right).\end{aligned}$$

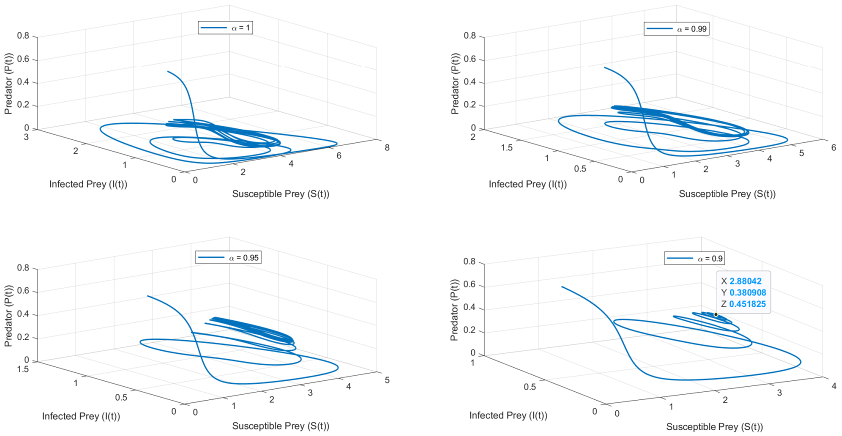


Figure 2. Occurrence of $E^*(2.88042, 0.38090, 0.451825)$ by varying α in the system (1.2).

The PECE (predict, evaluate, correct, evaluate) method is subsequently employed. The following five examples are presented to validate our analytical results.

Example 5.1. Using the given parameter values, the numerical simulation investigates the dynamics of the predator-prey system under the fractional-order α. The parameters are fixed as follows: $r = 0.5, f = 0.05, K = 10, \lambda = 0.9, m = 0.7, d = 0.5, a = 0.2, b = 1, \theta = 1.5$. By systematically reducing the fractional-order α from $\alpha = 1$ to $\alpha = 0.99$, $\alpha = 0.95$, and finally $\alpha = 0.90$, the numerical simulations provide insights into the evolving dynamics of the predator-prey system. With each reduction in α, the oscillatory behavior observed in the solution curves diminishes progressively. At $\alpha = 0.90$, the system transitions from a limit cycle to a stable equilibrium point, reflecting the system's adaptation toward stability as α decreases. In Figure 1, the trajectory planes clearly demonstrate the impact of fractional-order dynamics on the ecological stability of the system, effectively highlighting the system (1.2) progression toward equilibrium.

Example 5.2. The phase portraits of Figure 2 are generated using varying fractional-orders $\alpha = 1, 0.99, 0.95, 0.90$ illustrate the occurrence of the equilibrium point E^*. These visualizations, derived from the predator-prey model with parameters $r = 0.5$, $f = 0.05$, $K = 10$, $\lambda = 0.9$, $m = 0.7$, $\mu = 2$, $a = 0.2$, $n = 1.5$, $b = 1$, $d = 0.5$, and $\theta = 1.5$, reveal the influence of fractional-order dynamics on system stability. As α decreases, the trajectories converge toward equilibrium, emphasizing the role of fractional order in stabilizing ecological interactions.

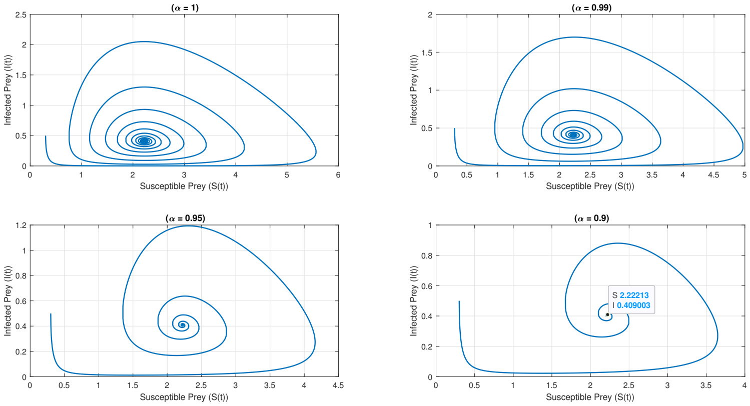


Figure 3. Occurrence of $E_2(2.22213, 0.409003, 0)$ by varying values of α in the system (1.2).

Example 5.3. We fix the values $r = 0.5$, $f = 0.05$, $K = 10$, $\lambda = 0.9$, $m = 0.7$, $\mu = 2$, $a = 0.2$, $n = 1.5$, $b = 1$, $d = 0.5$, and $\theta = 1.5$. Figure 3 demonstrates the occurrence of the equilibrium point E_2 as the fractional order α varies.

Example 5.4. Figure 4 shows the impact of varying the cannibalism parameter n on the predator-prey system's stability. By adjusting n through values 0.9, 1.0, 1.2, and 1.5, significant changes in predator population dynamics are observed. Higher values of n, representing increased cannibalistic behavior among predators, lead to a reduction in oscillatory fluctuations and contribute to the system's stabilization over time.

Example 5.5. For $\alpha = 0.90$, the fractional-order system is analyzed using fixed parameters and five initial conditions (0.3, 0.5, 0.8), (0.2, 0.6, 0.9), (0.4, 0.3, 0.7), (0.5, 0.4, 0.6), (0.5, 0.4, 0.6). From Figure 5, the numerical simulations show that all trajectories converge to the same stable equilibrium point $E^*(2.85084, 0.367857, 0.459236)$, irrespective of the starting conditions. This demonstrates global stabiity under the given settings, where the populations $S(t)$, $I(t)$, and $P(t)$ evolve to steady-state values E^* and satisfy the conditions of Theorem 4.1.

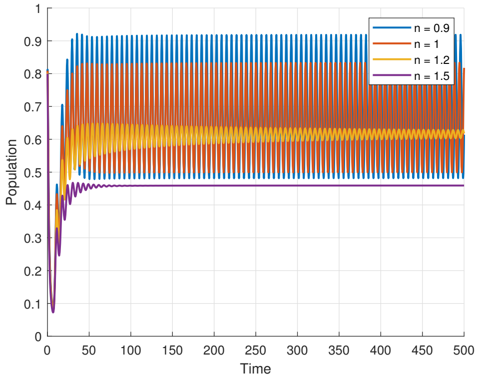


Figure 4. Effect of cannibalism parameter n on predator population dynamics of the system (1.2).

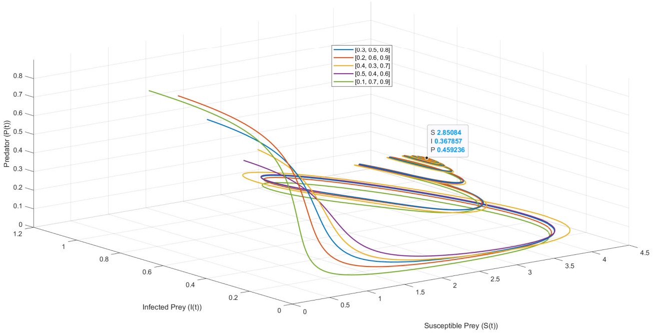


Figure 5. Global asymptotic stability of the co-existence equilibrium point $E^*(2.88042, 0.38090,$ $0.451825)$ with different initial values.

6. Conclusion

This paper proposes a fractional-order prey-predator model that incorporates disease transmission in prey, the fear effect induced by predators, and cannibalism within the predator population. First, the positivity and boundedness of the system are established. The existence of four potential non-negative equilibrium points is demonstrated. The local stability of these equilibrium points is analyzed, and stability conditions are determined. Global stability is further studied using Lyapunov-based techniques. Additionally, numerical simulations illustrate the role of various model parameters, such as the fear effect, cannibalism rates, and fractional-order parameters, in shaping population dynamics. These results provide a deeper understanding of the ecological system and its complex interactions.

Increasing the cannibalism parameter n in the interval $[0.8, 1.5]$ the system (1.2) directly reduces oscillations in predator populations when $n = 1.5$ and promoting system stability. Higher values of n reflect stronger self-regulation among predators through cannibalistic behavior, which dampens excessive growth and interaction fluctuations. This stabilization shifts the system toward equilibrium, highlighting cannibalism as a critical ecological factor. These findings emphasize n as a key determinant of stability in predator-prey relationships and ecosystem resilience. Additionally, the presence of disease in prey and fear effects play a crucial role in shaping the system's dynamics by influencing prey behavior and limiting their accessibility to predators. Together, these factors – cannibalism, disease, and fear play a pivotal role in maintaining resilience within predator-prey systems.

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