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A Numerical Scheme for a Predator-Prey Fractional Model with Disease in the Prey Species

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Abstract

In this paper, an existing predator–prey model with disease in the prey population is extended using the Caputo–Fabrizio fractional derivative. The fractional formulation incorporates memory effects through a non-singular kernel, allowing the model to capture delayed responses in eco-epidemiological dynamics. The well-posedness of the resulting system is established via existence and uniqueness of solutions.
A numerical scheme based on a three-step Adams–Bashforth method is employed to obtain approximate solutions.

Keywords: Caputo-Fabrizio derivative, Predator-prey model, Three-step Adams-Bashforth scheme, Pelican-tilapia system, Numerical simulations.
2010 Mathematics Subject Classification: 26A33, 81T80

1. Introduction

Riemann-Liouville fractional operators defined on finite intervals with a fixed lower limit, which generalize differentiation to non-integer orders and capture memory effects through dependence on the function's history [1, 2]. However, these operators presented challenges in applications since differentiating a constant does not give zero, and initial conditions do not align with those of classical differential equations.
To address these limitations, Caputo introduced in 1967 what is now known as the Caputo derivative by placing the integer-order derivative inside the fractional integral, ensuring that the derivative of a constant is zero and allowing classical initial conditions. Building on this, Caputo and Fabrizio [3], proposed a fractional derivative with a non-singular exponential kernel, now referred to as the Caputo-Fabrizio (CF) derivative, which avoids kernel singularities while retaining desirable properties and providing a flexible framework for modelling memory effects [2, 3, 4].

An important contribution to the application of fractional integrals came from Abel (1802-1829), who in 1823

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reformulated the tautochrone problem as an integral equation, demonstrating the applicability of fractional calculus to physical phenomena [1, 2]. Since then, fractional differential equations (FDEs) have been widely applied in viscoelasticity, anomalous diffusion, epidemiology, and eco-epidemiology. In particular, predator–prey models with disease in prey populations capture how memory effects influence equilibria, stability, and coexistence [3, 4, 5].These models have been extended to incorporate infection dynamics and seasonal effects via non-local operators and almost periodic functions, as well as piecewise differential equations that describe stage-based ecological processes and enable transitions from classical predator–prey systems to infected-prey models [6, 7]. Complementing these developments are non-singular kernel derivatives, such as the Atangana–Baleanu operator, which provide additional flexibility for modelling non-local interactions and memory effects in predator–prey–disease dynamics [8].

Motivated by this history and recent advances, we study a fractional-order predator–prey model with disease in the prey species under the Caputo–Fabrizio framework. The model incorporates logistic growth of prey, disease transmission, predator feeding responses and predator growth influenced by both susceptible and infected prey. We establish the mathematical well-posedness of the system by proving existence and uniqueness of solutions and analyse its dynamics using a fractional Adams–Bashforth numerical scheme. The scheme provides a step-by-step approximation that incorporates the influence of past states, thereby capturing memory effects in the system, while an associated error estimate ensures that the approximation remains controlled under suitable conditions. These findings show that memory effects influence the rate of disease spread, the persistence of predator and prey populations, and the time required for the system to approach equilibrium thus providing a more realistic description of predator–prey–disease interactions than classical models.

The remainder of this paper is structured as follows. In Section 2, we present the parameters and definitions used throughout the paper. Section 3 describes the model to be solved, along with the existence and uniqueness of its solution. The numerical method used to solve the model is presented in Section 4. Section 5 covers simulations, test problems, and their discussion. Finally, Section 6 concludes the paper.

2. Preliminaries

This section consist of definitions, variables and parameters, which will be used further.

Table 1: Biological meanings of parameters.

Parameter	Interpretation
r_1	Per capita growth rate of prey in the susceptible subpopulation.
r_2	Predator natural growth rate.
κ_1	Maximum sustainable size of the prey species.
κ_2	Maximum sustainable size of the killer species.
λ	Encounter coefficient between infected and susceptible prey.
γ	Infected prey dying rate caused by the disease.
p_1	Predator attack rates on susceptible prey.
p_2	Predator attack rates on infected prey.
ν	Predators' relative preference for infected versus susceptible prey.
n	Value at which consumption of infected prey reduces predator carrying capacity.
δ_1, δ_2	Rates of predator benefit and damage resulting from consumption of susceptible and infected prey, respectively.

Definition 2.1 (See [9])**.** Let $\mu \in (0,1]$, $v \in H^1(0,1)$ and $M(\mu)$ be a normalization function such that $M(0) = M(1) = 1$. Then, the CF-derivative is defined as

$$ {}^{CF}D_t^{\mu} v(t) = \frac{M(\mu)}{1-\mu} \int_0^t e^{\frac{\mu}{1-\mu}(t-\ell)} v'(\ell)\, d\ell. \tag{2.1}$$

This CF-derivative was later revisited and modified by authors in [10]

$$ {}^{CF}D_t^{\mu} v(t) = \frac{(2-\mu)M(\mu)}{2(1-\mu)} \int_0^t e^{\frac{\mu}{1-\mu}(t-\ell)} v'(\ell)\, d\ell, \quad 0 < \mu \le 1. \tag{2.2}$$

Definition 2.2. (See [10]). Let $\mu \in (0,1]$, the CF integral is defined as

$$ {}^{CF}I_t^{\mu} v(t) = \frac{2(1-\mu)}{(2-\mu)T(\mu)} v(t) + \frac{2\mu}{(2-\mu)T(\mu)} \int_0^t v(\ell) d\ell, \quad t \ge 0. \tag{2.3}$$

where

$$ T(\mu) = \frac{2}{2-\mu}, \quad 0 < \mu \le 1. \tag{2.4}$$

3. Model description

The model under consideration is adapted from [4], where both the classical (integer order) formulation and its fractional extension in the Caputo sense were introduced. For completeness, we briefly recall the model structure and its underlying assumptions, following by corresponding formulations.

The system describes a predator-prey interaction with disease dynamics in the prey population. The prey population is a combination of two distinct classes: susceptible ($S(t)$), and infected ($I(t)$). Consequently, total prey population ($N(t) = I(t) + S(t)$) , at time t. Predator population ($X(t)$), experiences increased mortality as a result of consuming stricken prey.

The model assumptions are biologically motivated: disease transmission follows the mass-action principle; infected prey do not recover due to high mortality; susceptible prey exhibit logistic growth in the absence of predation and infection; and the functional response ensures bounded predation rates (see [4, 11, 12, 13, 14]). Under these assumptions, the dynamics of the system can be described by the following ordinary differential equations:

$$\begin{aligned} \dot{S} &= r_1 S\big(1 - \frac{S+I}{\kappa_1}\big) - \lambda S I - \frac{p_1 S^2 X}{S + \nu I + 1}, \\ \dot{I} &= \lambda S I - \frac{p_2 I^2 X}{1 + S + \nu I} - \gamma I, \\ \dot{X} &= r_2 X \left[1 - \frac{X}{\kappa_2 + S + nI}\right] + \delta_1 \left[\frac{p_1 S^2 X}{1 + S + \nu I}\right] + \delta_2 \left[\frac{p_2 I^2 X}{1 + S + \nu I}\right], \end{aligned} \tag{3.1}$$

subject to non-negative initial conditions.

The corresponding fractional-order model in the Caputo sense, as presented in [4], is given by

$$\begin{aligned} {}^{C}D_t^{\mu} S &= r_1^{\mu} S\big(1 - \frac{S+I}{\kappa_1^{\mu}}\big) - \lambda^{\mu} S I - \frac{p_1^{\mu} S^2 X}{1 + S + \nu^{\mu} I}, \\ {}^{C}D_t^{\mu} I &= \lambda^{\mu} S I - \frac{p_2^{\mu} I^2 X}{1 + S + \nu^{\mu} I} - \gamma^{\mu} I, \\ {}^{C}D_t^{\mu} X &= r_2^{\mu} X \Big(1 - \frac{X}{\kappa_2^{\mu} + S + n^{\mu} I}\Big) + \delta_1^{\mu} \Big(\frac{p_1^{\mu} S^2 X}{1 + S + \nu^{\mu} I}\Big) + \delta_2^{\mu} \Big(\frac{p_2^{\mu} I^2 X}{1 + S + \nu^{\mu} I}\Big), \end{aligned} \tag{3.2}$$

All state variables are assumed to be non-negative with initial conditions $I(0) \geq 0$, $S(0) \geq 0$, and $X(0) \geq 0$. The feasible region (Ω) and the parameter space (Υ) of the system are given as:

$$\begin{aligned}
\Omega &= \{(S, I, X) \in \mathbf{R}_+^3 : S \geq 0, I \geq 0, X \geq 0\} \quad \text{and} \\
\Upsilon &= \{(r_1, r_2, \kappa_1, \kappa_2, \lambda, \gamma, p_1, p_2, \nu, n, \delta_1, \delta_2) \in \mathbf{R}^{12}\colon r_1, r_2, \kappa_1, \kappa_2, \lambda, \gamma, p_1, p_2 > 0; \nu, n, \delta_1, \delta_2 \geq 0\}. \quad (3.3)
\end{aligned}$$

The biological interpretation of parameters is given in Table 1.
Motivated by the advantages of fractional operators with non-singular kernels, we extend this framework by reformulating the model using the Caputo-Fabrizio fractional derivative. This provides an alternative description of the system dynamics while preserving the biological consistency of the original model. The corresponding fractional-order model in the Caputo-Fabrizio sense, is as follows:

$$\begin{aligned}
{}^{CF}D_t^{\mu} S &= r_1^{\mu} S\big(1 - \frac{S+I}{\kappa_1^{\mu}}\big) - \lambda^{\mu} SI - \frac{p_1^{\mu} S^2 X}{1 + S + \nu^{\mu} I}, \\
{}^{CF}D_t^{\mu} I &= \lambda^{\mu} SI - \frac{p_2^{\mu} I^2 X}{1 + S + \nu^{\mu} I} - \gamma^{\mu} I, \qquad (3.4) \\
{}^{CF}D_t^{\mu} X &= r_2^{\mu} X\Big(1 - \frac{X}{\kappa_2^{\mu} + S + n^{\mu} I}\Big) + \delta_1^{\mu}\Big(\frac{p_1^{\mu} S^2 X}{1 + S + \nu^{\mu} I}\Big) + \delta_2^{\mu}\Big(\frac{p_2^{\mu} I^2 X}{1 + S + \nu^{\mu} I}\Big),
\end{aligned}$$

3.1. On the Existence and Uniqueness of Solutions for Model 3.4

The existence and uniqueness of solutions for the classical and Caputo formulations of the model were established in [4]. For the Caputo-Fabrizio formulation, the system can be rewritten as an equivalent integral equation with a non-singular kernel. Specifically, system (3.4) can be expressed as

$$\begin{aligned}
S(t) &= S(0) + {}_0^{CF}I_t^{\mu}\Big[\Psi_1(t, S, I, X)\Big], \\
I(t) &= I(0) + {}_0^{CF}I_t^{\mu}\Big[\Psi_2(t, S, I, X)\Big], \\
X(t) &= X(0) + {}_0^{CF}I_t^{\mu}\Big[\Psi_3(t, S, I, X)\Big],
\end{aligned}$$

where Ψ_1, Ψ_2, Ψ_3 denote the nonlinear right-hand side functions of the system.
Since these functions are continuous and satisfy a Lipschitz condition on a bounded domain, the associated integral operator defines a contraction mapping. Therefore, by the Banach fixed-point theorem (see, [15] and [16]), the existence and uniqueness of solutions follow. Consequently, the proposed model admits a unique solution for given non-negative initial conditions.

4. The method

The three-step Adams-Bashforth scheme is a well-known explicit multistep method with established convergence and error properties [9, 17]. It has been successfully applied in similar contexts, supporting its suitability for the present model.

To obtain approximate solutions of system (3.4), we employ a three-step Adams–Bashforth scheme adapted to the Caputo–Fabrizio fractional derivative. The method is based on the discretization of the equivalent integral formulation and follows the approach presented in [17]. Applying this scheme to the model, the numerical solutions are obtained through the following iterative relations:

$$\begin{aligned}
S_{n+1} &= S_n + \left(\frac{1-\mu}{M(\mu)} + \frac{23h\mu}{12M(\mu)}\right) f_1(t_n, S_n, I_n, X_n) \\
&- \left(\frac{1-\mu}{M(\mu)} + \frac{16h\mu}{12M(\mu)}\right) f_1(t_{n-1}, S_{n-1}, I_{n-1}, X_{n-1}) + \frac{5h\mu}{12M(\mu)} f_1(t_{n-2}, S_{n-2}, I_{n-2}, X_{n-2}),
\end{aligned}$$

$$
\begin{aligned}
I_{n+1} &= I_n + \left(\frac{1-\mu}{M(\mu)} + \frac{23h\mu}{12M(\mu)}\right) f_2(t_n, S_n, I_n, X_n) \\
&- \left(\frac{1-\mu}{M(\mu)} + \frac{16h\mu}{12M(\mu)}\right) f_2(t_{n-1}, S_{n-1}, I_{n-1}, X_{n-1}) + \frac{5h\mu}{12M(\mu)} f_2(t_{n-2}, S_{n-2}, I_{n-2}, X_{n-2}). \\
X_{n+1} &= X_n + \left(\frac{1-\mu}{M(\mu)} + \frac{23h\mu}{12M(\mu)}\right) f_3(t_n, S_n, I_n, y_n) \\
&- \left(\frac{1-\mu}{M(\mu)} + \frac{16h\mu}{12M(\mu)}\right) f_3(t_{n-1}, S_{n-1}, I_{n-1}, X_{n-1}) + \frac{5h\mu}{12M(\mu)} f_3(t_{n-2}, S_{n-2}, I_{n-2}, X_{n-2}),
\end{aligned}
$$

where h denotes the time step and f_1, f_2, f_3 represent the right-hand side functions of system (3.4).
S_n, I_n, X_n are known from the initial condition. $S_{n-1}, I_{n-1}, X_{n-1}$ and $S_{n-2}, I_{n-2}, X_{n-2}$ are predicted with Matlab ode 45.
This scheme is used to generate the numerical simulations presented in the following section.

5. Simulation and test problems

For the numerical analysis, we adopt parameters from eco-epidemiological studies in the Salton Sea, where tilapia as a prey and pelicans as a predator [5, 11, 18, 19]. Initial population is given as $(S; I; X) = (10; 3.5; 1.5)$.

Example 5.1 ([4])**.** With the parameters and initial conditions given as follows: $S(0) = 10$, $I(0) = 3.5$, $X(0) = 1.5$, $r_1 = 1.8$, $r_2 = 0.0015$, $\kappa_1 = 50$, $\kappa_2 = 20$, $\lambda = 0.06$, $\gamma = 0.24$, $p_1 = 0.05$, $p_2 = 0.05$, $\nu = 6$, $n = 0.25$, $\delta_1 = 0.35$, $\delta_2 = 0.18$, and with a step size of $h = 0.1$. Using the 3-step AB scheme, we illustrate the solution families for the integer-order system (3.1) and the fractional-order system using the classical Caputo–Fabrizio derivative (3.4) (see Figures 1–4).

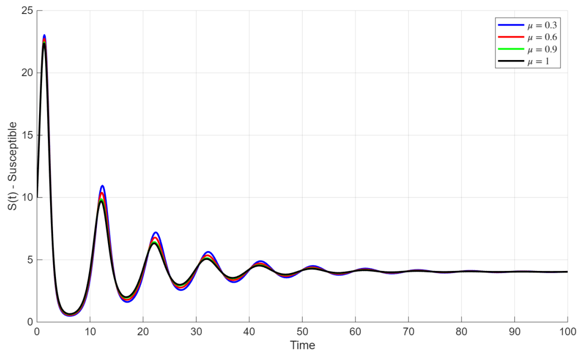

Figure 1: Numerical solution for Susceptible population for an integer and fractional model.

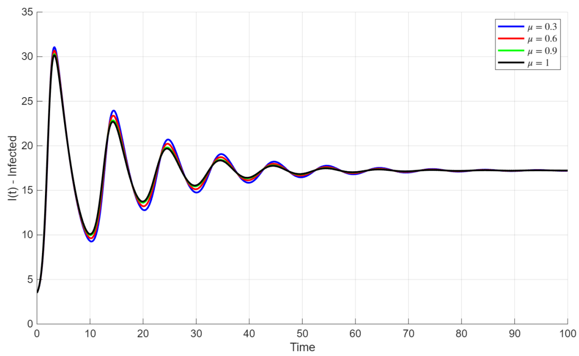

Figure 2: Numerical solution for infected population for an integer and a fractional model.

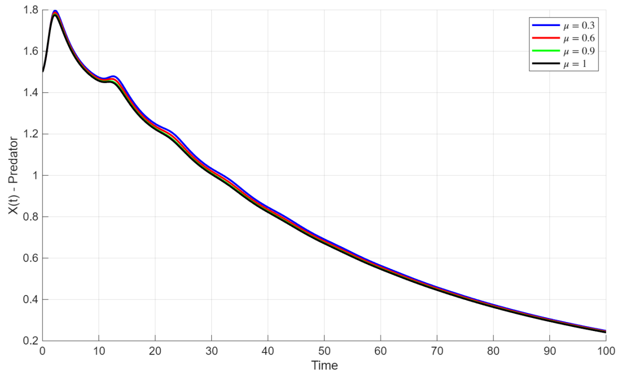

Figure 3: Numerical solution for predator population for an integer and a fractional model.

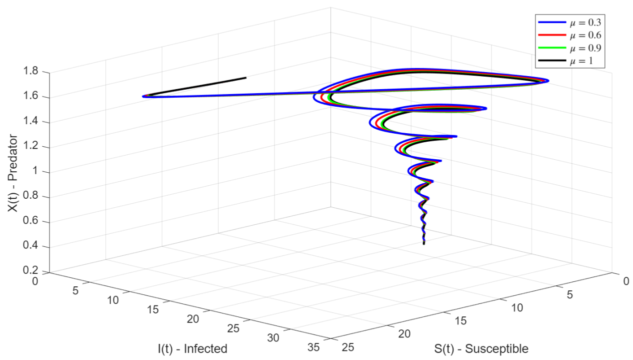

Figure 4: Phase portrait of the coexistence.

Example 5.2 ([4])**.** With the parameters and initial conditions given as follows: $S(0) = 10$, $I(0) = 3.5$, $X(0) = 1.5$, $r_1 = 1.8$, $r_2 = 0.15$, $\kappa_1 = 50$, $\kappa_2 = 20$, $\lambda = 0.06$, $\gamma = 0.24$, $p_1 = 0.05$, $p_2 = 0.05$, $\nu = 6$, $n = 0.25$, $\delta_1 = 0.35$, $\delta_2 = 0.18$, and with a step size of $h = 0,1$. Using the 3-step AB scheme, we illustrate the solution families for the integer-order system (3.1) and the fractional-order system using the classical Caputo–Fabrizio derivative (3.4) (see Figures 5,6,7, and 8).

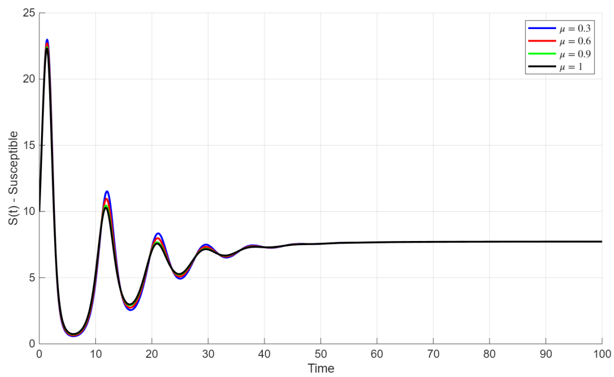

Figure 5: Numerical solution for Susceptible population for an integer and fractional model.

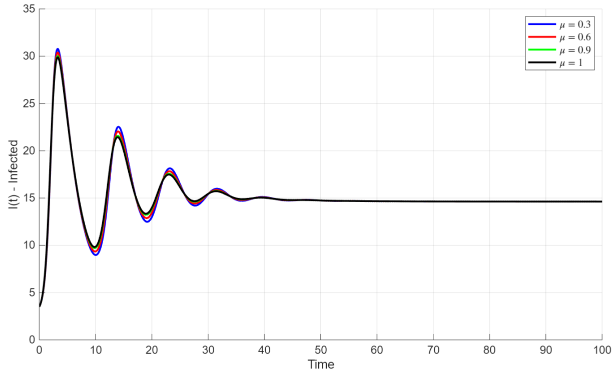

Figure 6: Numerical solution for infected population for an integer and a fractional model.

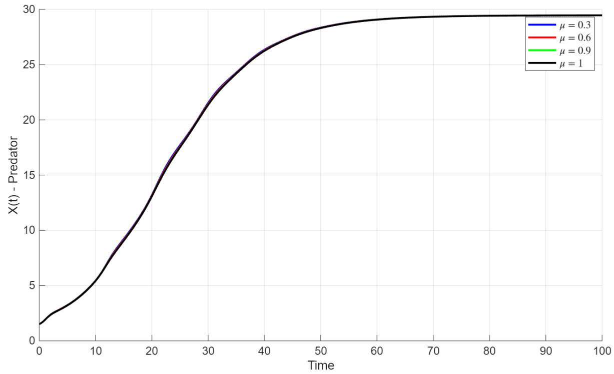

Figure 7: Numerical solution for predator population for an integer and a fractional model.

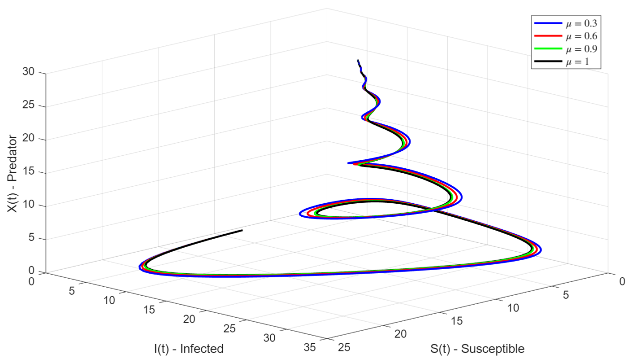

Figure 8: Phase portrait of the coexistence.

5.1. Discussion

The model under study consists of a tilapia population, divided into susceptible $S(t)$ and infected $I(t)$ classes, and a pelican predator population $X(t)$. This eco-epidemiological system was originally proposed to describe interactions in the Salton Sea ecosystem. In the present work, the model is reformulated using the Caputo-Fabrizio fractional derivative of order μ, which governs the strength of memory effects in the system.

The inclusion of the Caputo–Fabrizio operator implies that the current dynamics of the tilapia–pelican interactions depend not only on the present state but also on their recent history. Biologically, this reflects more realistic eco-epidemiological processes, such as delayed responses to infection and the cumulative effects of predation on infected prey.

As observed in the numerical simulations (see Figures 1–8), smaller values of μ lead to more pronounced oscillations and prolonged transient dynamics. This suggests that stronger memory effects are associated with sustained infection levels within the prey population. Consequently, decreasing μ may delay the stabilization of the system and promote persistence of the disease.

Furthermore, we investigated the sensitivity of the system to variations in the pelican intrinsic growth rate r_2 , which represents the natural reproduction capacity of the predator. Biologically, this parameter is associated with reproductive success and environmental suitability. Increasing r_2 results in a higher pelican population and a corresponding reduction in the tilapia population, particularly the infected class (see Figures 5–7).

In addition, the influence of r_2 is modulated by the fractional order μ. For smaller values of μ, the predator response is delayed due to memory effects, meaning that even with increased r_2, the adjustment of the pelican population occurs more gradually. This highlights the interplay between biological parameters and memory effects in shaping the overall system dynamics.

6. Conclusion

In this paper, we extended an existing predator-prey model with diseased prey by reformulating it within the Caputo-Fabrizio fractional framework. The use of this operator replaces the classical derivative with a non-singular fractional derivative, allowing the model to incorporate memory effects through an exponential kernel. As a result, the system is able to capture delayed responses and persistence in population dynamics.

The existence and uniqueness of solutions were established under standard conditions, ensuring that the proposed model is mathematically well-posed. Furthermore, a three-step Adams-Bashforth numerical scheme was employed to approximate the solutions of the fractional system.

Numerical simulations were conducted to compare the classical integer-order case ($\mu = 1$) with fractional-order cases ($0 < \mu < 1$). The results show that smaller values of μ lead to stronger oscillations and slower convergence, indicating prolonged transient dynamics. Although the fractional model exhibits more pronounced oscillatory behavior, it preserves the qualitative structure of the classical solution. As $\mu \to 1$, the fractional solutions converge to their classical counterparts.

A similar trend was observed when varying the predator growth rate, where smaller values of μ resulted in increased oscillations and delayed stabilization. These findings highlight the significant role of memory effects in shaping eco-epidemiological dynamics and demonstrate the relevance of the Caputo-Fabrizio approach in modeling such systems.

Despite these contributions, the present model is based on several simplifying assumptions. In particular, disease transmission is modeled under homogeneous mixing and mass action incidence, and stochastic effects arising from environmental fluctuations are not included. Moreover, the use of the Caputo-Fabrizio derivative introduces an exponential-type fading memory, which may not fully capture long-range memory effects observed in some ecological systems. Future work may focus on extending the model to include stochastic perturbations, spatial diffusion, or alternative fractional operators with different memory kernels in order to provide a more comprehensive description of eco-epidemiological dynamics.

Conflict of interest

The authors declare no conflict of interest.

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