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EXISTENCE OF ECOLOGICAL PREY PREDATOR MATHEMATICAL MODEL IN THE CONTAMINATED DOMAIN

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Abstract. We examine a predator–prey model that incorporates toxicant dynamics and a distributed delay representing gradual toxicant uptake. We prove positivity and boundedness of solutions and determine the system's equilibria. Local stability of equilibria is examined via the Jacobian matrix and Routh–Hurwitz conditions, while global asymptotic stability of the coexistence equilibrium is established using a Lyapunov function and Sylvester's criterion. Numerical simulations support the analytical results and compare the full model with a reduced model that omits toxicants and delay; these comparisons show that toxicants and delay affect transient dynamics but have limited impact on long-term stability. Our results highlight the importance of including toxicant dynamics and time delays when modeling population interactions in contaminated habitats.

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1. Introduction

Industrial activities release contaminants that accumulate in natural habitats and threaten ecological communities. Toxic elements such as cadmium, zinc, and mercury enter air and water, impairing organism health and altering trophic interactions. Understanding how toxicants modify predator–prey dynamics is therefore important for both theoretical ecology and applied environmental management. In this paper we analyze a two-species predator–prey model that couples population dynamics with organismal and environmental toxicant concentrations and includes a distributed delay for toxicant uptake [1].

Every component of a natural ecosystem involves interactions between prey and predators, providing valuable opportunities to explore different aspects of their relationships. To understand and predict the behavior and interactions of biological populations, various functional models have been developed. Over the past several decades, researchers, mathematicians, and biologists have extensively studied predator–prey dynamics to gain deeper insights into these complex ecological processes [2–9]. Analyzing how predators consume their prey is a key aspect of understanding predator–prey interactions. Even small changes in the behavioral patterns of prey or predators can significantly alter the overall dynamics of the biological system.

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Over time, numerous mathematical models have been developed and studied to examine chemical-related issues at various levels of the biological food chain. These include models addressing population growth, ecosystem dynamics, landscape interactions, and toxicity extrapolation [10, 11]. The uncontrolled release of hazardous substances into the environment has led to the decline of many species. These pollutants often accumulate in the bodies of grazing animals and are transferred through the food chain, ultimately affecting consumers at higher trophic levels, including marine predators [12]. In general, toxicants disrupt ecological balance and hinder the growth of biological species. Many researchers have used mathematical models to study in detail how these toxic substances influence predator–prey communities and their long-term dynamics [13, 14]. In a predator–prey food chain, interference among predators naturally occurs when a top predator affects the behavior or population of an intermediate predator. The impact of this interaction on the dynamics of biological populations has been the focus of many research studies [15–20].

In the experimental investigation of [21, 22], researchers have found that consuming wastewater from textile dye industries, which often contains copper and synthetic coloring agents, is linked to reproductive disorders and other health problems. The textile sector is one of the major human-driven sources of such pollution. When predators feed on rats or other organisms exposed to these contaminants, their populations are also negatively affected.

To account for time delays of variable duration, many researchers have incorporated distributed delay differential equations into their mathematical models. Including distributed delays is important because the behavior of such models can differ significantly from models with discrete or random delays. Using numerical bifurcation analysis, researchers have shown that predator–prey models with distributed delays can exhibit complex dynamics, including period-doubling bifurcations [23]. The study found that the fear of predation can either stabilize or destabilize the dynamics of the system, depending on its intensity [24]. After incorporating distributed delays into their mathematical model, researchers observed periodic solutions and the occurrence of Hopf bifurcations [25–27].

Earlier studies have examined the effects of toxicants and delays on ecological systems. Misra et al. (2016) [26] investigated a three-species food chain model that incorporated a delay in toxicant uptake by prey and demonstrated that time delay could induce oscillations in species densities. Khare et al. (2011) [27] analyzed a planktonic ecosystem with toxic-producing phytoplankton and found that delay in toxin release significantly altered system stability. While these models provided important insights into aquatic or planktonic environments, the present study extends this approach to a two-species predator–prey system under toxicant stress with a distributed delay structure. This formulation captures both biological interactions and pollutant effects within a unified framework, offering a broader understanding of how toxicants and time-dependent processes jointly regulate population persistence and ecological balance.

From an ecological perspective, the model represents the interactions between prey and predator populations in an environment affected by chemical contamination. The prey species depend on available resources for growth, while predators rely on prey as their primary food source. However, toxicants released into the ecosystem influence both populations by altering their survival and reproductive capacities. Toxic substances, often originating from industrial discharge, accumulate in the environment and enter organisms through food or direct exposure. These pollutants interfere with physiological processes such as feeding, metabolism, and reproduction, thereby disturbing the natural predator–prey balance. Modeling such processes helps predict how environmental contamination affects long-term species persistence and ecosystem stability.

2. Mathematical Model

In order to investigate how toxicants affect a predator-prey system in a contaminated environment, we create a nonlinear mathematical model in this work. Prey density $X(T)$, predator

density $Y(T)$, organism toxicant concentration $P(T)$, and environmental toxicant levels $Q(T)$ are the four main state variables that make up the model.Prey grows logistically with intrinsic rate A and carrying capacity K. The predation rate B and a half-saturation constant D, which takes into account the predator's effectiveness in capturing prey, determine how much prey predators consume. Energy conversion efficiency G affects predator growth, whereas intraspecific competition F and natural mortality C control its survival.

The dynamics of the system are greatly impacted by toxicants. While the habitat is constantly exposed to external toxicant input q_0, the predator suffers from increased mortality as a result of toxicant accumulation at a rate E. Natural washout mechanisms q_1 and removal processes P_1 are how the toxicant dissipates. In addition to absorbing poisons from their environment, organisms have the ability to gradually get rid of them; prey excrete toxins at a rate of P_2, while predators do so at a rate of P_3.

Our model sheds light on how contaminants affect long-term ecosystem stability, change species survival, and disturb predator-prey dynamics by taking into account these ecological and toxicological aspects. This concept provides important insights for ecological conservation and pollution control, and it provides a basis for comprehending why species endure in contaminated habitats.

$$\begin{aligned}
\frac{dX}{dT} &= AX(1-\frac{X}{K}) - EPX - \frac{BXY}{X+D} \\
\frac{dY}{dT} &= \frac{GXY}{X+D} - CY - FY^2 \\
\frac{dP}{dT} &= P_1QX - P_2P - P_3XP \\
\frac{dQ}{dT} &= q_0 - q_1Q - P_1XQ + P_3XP
\end{aligned}$$

with $X(0) = X_0 \geq 0, Y(0) = Y_0 \geq 0, P(0) = P_0 \geq 0, Q(0) = Q_0 \geq 0$.
We have introduced distributed delay $R_1(t)$ in the term 'P_1XQ', that shows the uptake of environment toxicant by the prey population.

From (1) we obtain

$$\begin{aligned}
\frac{dP}{dT} &= P_1QX - P_2P - P_3XP \\
\frac{dP}{dT} &= P_1\int_{-\infty}^{t} \gamma_1 exp(-\gamma_1(t-s))Q(s)dsX - P_2P - P_3XP
\end{aligned}$$

From (1)

$$\begin{aligned}
\frac{dQ}{dT} &= q_0 - q_1Q - P_1XQ + P_3XP \\
\frac{dQ}{dT} &= q_0 - q_1Q - P_1X\int_{-\infty}^{t} \gamma_1 exp(-\gamma_1(t-s))Q(s)ds + P_3XP
\end{aligned}$$

By Putting

$$R_1(t) = \int_{-\infty}^{t} \gamma_1 exp(-\gamma_1(t-s))Q(s)ds,$$

the above system of delay differential equation can be written as:

Model A.

$$\begin{aligned}
\frac{dX}{dT} &= AX(1-\frac{X}{K}) - EPX - \frac{BXY}{X+D}\\
\frac{dY}{dT} &= \frac{GXY}{X+D} - CY - FY^2\\
\frac{dP}{dT} &= P_1R_1X - P_2P - P_3XP\\
\frac{dQ}{dT} &= q_0 - q_1Q - P_1XR_1 + P_3XP\\
\frac{dR_1}{dT} &= \gamma_1(Q - R_1)
\end{aligned}$$

with $X(0) = X_0 \geq 0$, $Y(0) = Y_0 \geq 0$, $P(0) = P_0 \geq 0$, $Q(0) = Q_0 \geq 0$, $R_1(0) = 0 \geq 0$.

Ecologically, the proposed model describes how toxicants influence the interaction between prey and predator populations in a contaminated habitat. The prey grows logistically but experiences stress from toxicant exposure and predation. Predators depend on the prey for energy, but their survival and reproduction are limited by both food availability and toxicant accumulation. The toxicant dynamics link the biological and environmental components of the system, toxicants enter the environment through external inputs, are absorbed by organisms, and are partially removed through natural decay and biological detoxification. The inclusion of distributed delay reflects that toxicant uptake and its physiological effects are gradual rather than immediate.

Overall, the model captures an ecosystem where pollution regulates population balance. Increased toxicant input suppresses both prey and predator densities, while efficient natural washout or detoxification enhances ecological stability. This formulation helps explain how contaminated environments alter species persistence, recovery rates, and the long-term coexistence of interacting populations.

To reduce parameters that are considered in the Model-A by using the following changes:

$$X = Kx,\ Y = Ay,\ P = \frac{Ap}{E},\ Q = \frac{Aq}{E}, R_1 = \frac{Ar}{E},\ T = \frac{t}{A},\ B = K,\ C = Ac,$$
$$D = dK,\ G = aA,\ F = b,\ P_1 = \frac{Aa_1}{K},\ P_2 = \frac{Aa_2}{K}, P_3 = \frac{Aa_3}{K},\ q_0 = \frac{A^2a_0}{E},\ q_1 = Aa_4.$$

After rescaling, The Mode A can be written as :

Model A'.

$$\frac{dx}{dt} = x(1-x) - px - \frac{xy}{x+d} \tag{1}$$
$$\frac{dy}{dt} = \frac{axy}{x+d} - cy - by^2 \tag{2}$$
$$\frac{dp}{dt} = a_1xr - a_2p - a_3xp \tag{3}$$
$$\frac{dq}{dt} = a_0 - a_4q - a_1xr + a_3xp \tag{4}$$
$$\frac{dr}{dt} = s(q-r) \tag{5}$$

with $x(0) \geq 0$, $y(0) \geq 0$, $p(0) \geq 0$, $q(0) \geq 0$, $r(0) \geq 0$.

3. Positivity of Model A'

In this section, we establish that the solutions of the proposed system remain non-negative for all $t \geq 0$. This property, known as positivity, ensures that state variables representing physical or

biological quantities do not take unrealistic negative values. By applying an invariance criterion, we prove that the non-negative domain R^5_{+0} is preserved under the system dynamics. The following lemma provides the theoretical foundation for this result.

Lemma 3.1. *Suppose n is a positive integer and $f_i(t, x_1, x_2, \ldots, x_n)$, $(i = 1, 2, \ldots, n)$ are smooth functions. If $f_i|_{x_i=0,\Omega\in R^n_{+0}} \geq 0$ (where $\Omega = (x_1, x_2, \ldots, x_n)^T \in R^n$), then R^n_{+0} is an invariant domain of the following equations:*

$$\frac{dx_i}{dt} = f_i(t, x_1, x_2, \ldots, x_n), (i = 1, 2, 3, \ldots, n).$$

If $f_i|_{x_i=0,\Omega\in R^n_{-0}} \leq 0$ (where $\Omega = (x_1, x_2, \ldots, x_n)^T \in R^n$), then R^n_{-0} is an invariant domain of the above equations [28].

Theorem 3.2. $R^5_{+0} = \{(x, y, p, q, r)^T | x \geq 0, y \geq 0, p \geq 0, q \geq 0, r \geq 0\}$ *is an invariant domain of equations* (1), (2), (3), (4), (5).

Proof. Denote that $\Omega = (x, y, p, q, r)^T$. For the Model-1, we notice that

$$\begin{aligned}
f_1|_{x=0,\Omega\in R^5_{+0}} &= (x(1-x) - px - \frac{xy}{x+d})|_{x=0,\Omega\in R^5_{+0}} = 0 \geq 0,\\
f_2|_{y=0,\Omega\in R^5_{+0}} &= (\frac{axy}{x+d} - cy - by^2)|_{y=0,\Omega\in R^5_{+0}} = 0 \geq 0,\\
f_3|_{p=0,\Omega\in R^5_{+0}} &= (a_1xr - a_2p - a_3xp)|_{p=0,\Omega\in R^5_{+0}} = a_1xr > 0,\\
f_4|_{q=0,\Omega\in R^5_{+0}} &= (a_0 - a_4q - a_1xr + a_3xp)|_{q=0,\Omega\in R^5_{+0}} = a_0 - a_1xr + a_3xp > 0,\\
f_5|_{r=0,\Omega\in R^5_{+0}} &= s(q-r)|_{r=0,\Omega\in R^5_{+0}} = sq > 0.
\end{aligned}$$

By the Lemma, it follows that R^5_{+0} is an invariant domain of equations (1), (2), (3), (4), (5). □

4. Boundedness of Model A'

Ensuring the boundedness of solutions is fundamental in analyzing dynamical systems. In this section, we establish a boundedness theorem for the proposed system, proving that all variables remain within finite limits over time. Using the comparison theorem, we derive upper bounds and confirm that the solution remains within a positively invariant set. The following theorem formalizes this result.

Theorem 4.1. *The set Ω represents a bounded region for the system, where the functions and parameters define the constraints on the values of x, y, p, q and r over time. Specifically,*

$$\Omega = \{(x, y, p, q, r) \in R^5_+ : \quad x \leq 1, \quad ax + y \leq G_1, \quad p + q \leq G_2, \quad q + r \leq G_3\},$$

where,

$$\begin{aligned}
G_1 &= \frac{ax}{\varphi_1} \quad with \quad \varphi_1 = \min\{p, c\},\\
G_2 &= \frac{a_0}{\varphi_2} \quad with \quad \varphi_2 = \min\{a_2, a_4\},\\
G_3 &= \frac{a_0 + a_3p}{\varphi_3} \quad with \quad \varphi_3 = \min\{a_4 - s, a_1 - s\}.
\end{aligned}$$

Proof. Equation (1) gives

$$dx/dt \leq x(1-x).$$

Applying the comparison theorem, we conclude that as $t \to \infty$, $x \leq 1$. Eq. (2) gives

$$dy/dt \geq -y(c + by).$$

Again, using the comparison theorem, we conclude that as $t \to \infty$, $y \geq 0$.

Define

$$\xi_1(t) = ax(t) + y(t).$$

Using Eqs. (1) and (2), we obtain:

$$d\xi_1/dt + \varphi_1\xi_1 \leq ax,$$

where $\varphi_1 = \min\{p, c\}$. Applying the comparison theorem, we conclude that as $t \to \infty$,

$$\xi_1 \leq \frac{ax}{\varphi_1},$$

which implies

$$ax(t) + y(t) \leq G_1,$$

where $G_1 = ax/\varphi_1$.

Define

$$\xi_2(t) = p(t) + q(t).$$

From Eqs. (3) and (4) we derive:

$$d\xi_2/dt + \varphi_2\xi_2 \leq a_0,$$

where $\varphi_2 = min\{a_2, a_4\}$. Using the comparison theorem, we obtain as $t \to \infty$,

$$\xi_2 \leq \frac{a_0}{\varphi_2},$$

which implies

$$p(t) + q(t) \leq G_2,$$

where $G_2 = a_0/\varphi_2$.

Define

$$\xi_3(t) = q(t) + r(t).$$

From Eqs. (4) and (5), we obtain:

$$d\xi_3/dt + \varphi_3\xi_3 \leq a_0 + a_3xp,$$

where $\varphi_3 = \min\{a_4 - s, a_1 - s\}$. Using the comparison theorem, we obtain as $t \to \infty$,

$$\xi_3 \leq \frac{a_0 + a_3p}{\varphi_3},$$

which implies,

$$q(t) + r(t) \leq G_3,$$

where $G_3 = (a_0 + a_3p)/\varphi_3$. □

By systematically bounding each variable in the system, we confirm that the solution remains within a defined limit. Thus, the model is bounded, completing the proof.

5. Equilibria of Model A'

In this section, we explore the equilibrium points of Model A' and derive the conditions necessary for their existence. The system admits three distinct equilibria:

(i) $E_0(0,0,0,q,r)$– A boundary equilibrium where certain variables vanish.
(ii) $\dot{E}_1(\dot{x},0,\dot{p},\dot{q},\dot{r})$– A semi-trivial equilibrium where partial coexistence occurs.
(iii) $\ddot{E}_2(\ddot{x},\ddot{y},\ddot{p},\ddot{q},\ddot{r})$ – A fully interior equilibrium representing coexistence.

By examining the governing equations, we identify the limitations required to ensure the viability and positivity of these equilibria. These findings provide a solid foundation for comprehending the system's stability and long-term dynamics.

Existence of $E_0(0,0,0,q,r)$. From (4)

$$q = \frac{a_0}{a_4} > 0.$$

The ratio of external toxicant input a_0 to washout rate a_4 determines the equilibrium concentration of environmental toxicants. This suggests that the equilibrium between the introduction and removal efficiency of toxicants determines their persistence. Toxins build up when intake is large and the washout rate is low, which may endanger the existence of the species. The importance of both artificial and natural detoxification processes is shown by the fact that a higher washout rate encourages a cleaner environment.

From (5)

$$r = q.$$

At every equilibrium point, the condition $r = q$ indicates that the amounts of toxicants in the system are constant. This indicates that the two variables are directly correlated, meaning that changes in one will immediately result in changes in the other. A steady-state toxicant distribution is ensured by this equilibrium, which stops excessive buildup or depletion over time.

Existence of $\dot{E}_1(\dot{x},0,\dot{p},\dot{q},\dot{r})$. From (1)

$$\dot{p} = 1 - \dot{x} \tag{6}$$

$\dot{p} > 0$ if $\dot{x} < 1$. At equilibrium $\dot{E}_1$, the prey population density $\dot{x}$ affects the organismal toxicant level $\dot{p}$. This condition means that as long as the prey population remains below a crucial threshold $\dot{x} < 1$, the organismal toxicant will remain positive. According to biology, this implies that organisms accumulate fewer toxicants when there is a greater density of prey. If prey density reaches or exceeds this threshold, the toxicant level within organisms becomes negligible, indicating a potential self-regulating mechanism where high prey abundance dilutes the toxicant burden per individual.

From (3) and (4)

$$\dot{q} = \frac{a_0 - a_2 + a_2\dot{x}}{a_4} \tag{7}$$

$\dot{q} > 0$ if $a_2(1-\dot{x}) < a_0$.

At equilibrium $\dot{E}_1$, the environmental toxicant level is determined by the balance between external toxicant input a_0, the removal rate of toxicant from organisms a_2, and the prey population. This expression suggests that an increase in prey density enhances the environmental toxicant level if the removal rate a_2 is significant. Biologically, this highlights the role of prey in toxicant dynamics, indicating that higher prey populations can contribute to toxicant accumulation in the environment despite natural removal processes.

From Eq. (5)

$$\dot{r} = \dot{q} \tag{8}$$

Using Eqs. (6), (7) and (8) in (3), it follows that

$$(a_1a_2 + a_3a_4)\dot{x}^2 + (a_0a_1 - a_1a_2 + a_2a_4 - a_3a_4)\dot{x} - a_2a_4 = 0. \tag{9}$$

Eq. (9), which always has a positive root.

Existence of $\ddot{E}_2(\ddot{x}, \ddot{y}, \ddot{p}, \ddot{q}, \ddot{r})$. From Eq. (2)

$$\ddot{y} = \frac{a\ddot{x}}{b(\ddot{x} + d)} - \frac{c}{b} \tag{10}$$

$\ddot{y} > 0$ if $c(\ddot{x} + d) < a\ddot{x}$.

From Eq. (5)

$$\ddot{r} = \ddot{q} \tag{11}$$

By using Eq. (10) in (1) we obtain

$$\ddot{p} = (1 + \frac{c}{b(\ddot{x} + d)}) - \ddot{x}(1 + \frac{a}{b(\ddot{x} + d)^2}) \tag{12}$$

$\ddot{p} > 0$ if $1 + \frac{c}{b(\ddot{x}+d)} > \ddot{x}(1 + \frac{a}{b(\ddot{x}+d)^2})$.

Using Eqs.(11) and (12) in Eqs. (3) and (4) it follows that

$$\ddot{q} = \frac{a_0}{a_4} - \frac{a_2}{a_4}[(1 - \ddot{x}) - (\frac{a\ddot{x}}{b(\ddot{x} + d)^2} - \frac{c}{b(\ddot{x} + d)})] \tag{13}$$

$\ddot{q} > 0$ if $b(\ddot{x} + d)^2[a_2(1 - \ddot{x}) - a_0]) < a_2(a_1\ddot{x} - c(\ddot{x} + d))$.

Using Eqs.(11), (12) and (13) in Eq. (3), hence

$$\ddot{x} = \frac{a_2\ddot{p}}{(a_1\ddot{q} - a_3\ddot{p})}$$

$\ddot{x} > 0$ if $a_3\ddot{p} < a_1\ddot{q}$.

6. Local stability of Model A'

In the previous section, we identified three positive equilibrium points of the mathematical model: $E_0(0, 0, 0, q, r)$, $\dot{E}_1(\dot{x}, 0, \dot{p}, \dot{q}, \dot{r})$ and $\ddot{E}_2(\ddot{x}, \ddot{y}, \ddot{p}, \ddot{q}, \ddot{r})$. In this section, we analyze the local stability of these equilibrium points by deriving the variational (Jacobian) matrix and examining its eigenvalues. The stability conditions are determined using the Routh-Hurwitz criterion, which provides necessary constraints on system parameters for equilibrium points to remain stable. This analysis helps in understanding how small perturbations affect system dynamics and offers insights into the long-term behavior of the model.

$$J_2 = \begin{bmatrix} 1 - 2x - p - \frac{dy}{(x+d)^2} & \frac{-x}{x+d} & -x & 0 & 0 \\ \frac{ady}{(x+d)^2} & \frac{ax}{x+d} - c - 2by & 0 & 0 & 0 \\ a_1r - a_3p & 0 & -a_2 - a_3x & 0 & a_1x \\ -a_1r + a_3p & 0 & a_3x & -a_4 & -a_1x \\ 0 & 0 & 0 & s & -s \end{bmatrix}.$$

From the above variational matrix, we have observed the following results for $E_0, \dot{E}_1$ & $\ddot{E}_2$:

(1) The equilibrium point E_0 is unstable, because one of the eigen value of variational matric is positive ($\lambda_1 = 1$).

(2) At the Equilibrium point $\dot{E}_1$, one of the eigen value is given by

$$\lambda_1 = \frac{a\dot{x}}{\dot{x} + d} - c.$$

For stability, we require $\lambda_1 < 0$, which hold when

$$\frac{a\dot{x}}{\dot{x}+d} < c \quad or \quad \dot{x} < \frac{cd}{a-c}.$$

The remaining eigenvalues are determined by the characteristic equation:

$$\lambda^4 + M_1\lambda^3 + M_2\lambda^2 + M_3\lambda + M_4 = 0,$$

where the coefficients are given by:

$$\begin{aligned}
M_1 &= (a_2 + a_3\dot{x}) + (a_4 + s) + (\dot{p} + 2\dot{x} - 1),\\
M_2 &= (a_2 + a_3\dot{x})(a_4 + s) + a_4 s + (\dot{p} + 2\dot{x} - 1)(a_2 + a_3\dot{x})\\
&\quad + (\dot{p} + 2\dot{x} - 1)(a_4 + s) + \dot{x}(a_1\dot{r} - a_3\dot{p}),\\
M_3 &= (a_2 + a_3\dot{x})a_4 s + a_1 a_2 s\dot{x} + (\dot{p} + 2\dot{x} - 1)(a_2 + a_3\dot{x})(a_4 + s)\\
&\quad + (\dot{p} + 2\dot{x} - 1)a_4 s + \dot{x}(a_1\dot{r} - a_3\dot{p})(a_4 + s),\\
M_4 &= (\dot{p} + 2\dot{x} - 1)(a_2 + a_3\dot{x})a_4 s + (\dot{p} + 2\dot{x} - 1)a_1 a_2 s\dot{x} + x(a_1\dot{r} - a_3\dot{p})a_4 s.
\end{aligned}$$

By applying the Routh-Hurwitz stability criterion, $\dot{E}_1$ is locally stable if the following conditions hold:

$$M_1 > 0, \quad M_2 > 0, \quad M_3 > 0, \quad M_4 > 0, \quad M_1 M_2 M_3 > M_3^2 + M_1^2 M_4.$$

(3) For the equilibrium point $\ddot{E}_2$ the characteristic equation governing its stability is given by:

$$\lambda^5 + N_1\lambda^4 + N_2\lambda^3 + N_3\lambda^2 + N_4\lambda + N_5 = 0$$

where the coefficients are defined as:

$$\begin{aligned}
N_1 &= (a_4 + s) + (a_2 + a_3\ddot{x}) - \left(A_{11} + \frac{a\ddot{x}}{\ddot{x}+d} - c - 2b\ddot{y}\right),\\
N_2 &= a_1 s\ddot{x} + (a_4 + s)(a_2 + a_3\ddot{x}) - \left(A_{11} + \frac{a\ddot{x}}{\ddot{x}+d} - c - 2b\ddot{y}\right)\\
&\quad (a_4 + s + a_2 + a_3\ddot{x}) + \ddot{x}(a_1\ddot{r} - a_3 b) + A_{11}\left(\frac{a\ddot{x}}{\ddot{x}+d} - c - 2b\ddot{y}\right) + \frac{ad\ddot{x}\ddot{y}}{(\ddot{x}+d)^3},\\
N_3 &= -\left(A_{11} + \frac{a\ddot{x}}{\ddot{x}+d} - c - 2b\ddot{y}\right)(a_1 s\ddot{x} + a_4 s + (a_4 + s)(a_2 + a_3\ddot{x}))\\
&\quad + a_1 a_2 s\ddot{x} + (A_{11}(a_4 + s - \ddot{x}(a_1\dddot{r} - a_3\dddot{p}) + A_{11}(a_2 + a_3\ddot{x})))\\
&\quad \left(\frac{a\ddot{x}}{\ddot{x}+d} - c - 2b\ddot{y}\right) + \frac{ad\ddot{x}\ddot{y}}{(\ddot{x}+d)^3}(a_2 + a_3\ddot{x} + a_4 + s) + a_4 s(a_2 + a_3\ddot{x}) + (a_4 + s)(a_1\ddot{r} - a_3\dddot{p})\ddot{x},\\
N_4 &= -\left(A_{11} + \frac{a\ddot{x}}{\ddot{x}+d} - c - 2b\ddot{y}\right)(a_1 a_2 s\ddot{x} + a_4 s(a_2 + a_3\ddot{x}))\\
&\quad + \left(\frac{a\ddot{x}}{\ddot{x}+d} - c - 2b\ddot{y}\right)(A_{11}s(a_1\ddot{x} + a_4) + (a_4 + s)(A_{11}(a_2 + a_3\ddot{x}) -\\
&\quad (a_1\ddot{r} - a_3\dddot{p}))) + \frac{ad\ddot{x}\ddot{y}}{(\ddot{x}+d)^3}(a_1 s\ddot{x} + a_4 s + (a_2 + a_3\ddot{x})(a_4 + s)) + a_4 s\ddot{x}(a_1\ddot{r} - a_3\dddot{p}),\\
N_5 &= (A_{11}s(a_4(a_2 + a_3\ddot{x}) + a_1 a_2\ddot{x}) - a_4 s\ddot{x}(a_1\ddot{r} - a_3\dddot{p}))\\
&\quad \left(\frac{a\ddot{x}}{\ddot{x}+d} - c - 2b\ddot{y}\right) + (a_1 a_2\ddot{x} + a_4(a_2 + a_3\ddot{x}))\frac{ads\ddot{x}\ddot{y}}{(\ddot{x}+d)^3},
\end{aligned}$$

Additionally, the term A_{11} is defined as

$$A_{11} = 1 - 2\ddot{x} - \ddot{p} - \frac{d\ddot{y}}{(\ddot{x}+d)^2}.$$

According to the Routh-Hurwitz stability criterion, the equilibrium point $\ddot{E}_2$ is locally stable if the following conditions are met:

$$N_1 > 0, \quad N_2 > 0, \quad N_3 > 0, \quad N_4 > 0, \quad N_5 > 0, \quad N_1N_2 > N_3,$$
$$N_1N_2N_3 > (N_3^2 + N_1^2N_4), \quad (N_3N_4 - N_2N_5)(N_1N_2 - N_3) > (N_1N_4 - N_5)^2.$$

Based on the stability conditions of the equilibrium $\dot{E}_1$ ($\dot{x} < \frac{cd}{a-c}$), it is observed that the prey population density remains lower than a critical threshold. The product of the predator's natural mortality rate and half-saturation constant, as well as the difference between the predator's growth rate and natural death rate, determine this threshold. According to biology, the prey density needs to be high enough to guarantee predator survival and reproduction in order for the predator population to support itself.

7. Global Stability of Model A'

This section uses Lyapunov's approach to show that the equilibrium point $\ddot{E}_2$ is globally stable. We prove conditions guaranteeing global asymptotic stability by constructing a suitable Lyapunov function and verifying that its derivative is negative definite. Sylvester's rule is used to validate the conditions stated in the theorem.

Theorem 7.1. *In the region Ω, the equilibrium point $\ddot{E}_2(\ddot{x}, \ddot{y}, \ddot{p}, \ddot{q}, \ddot{r})$ is globally asymptotically stable if the following conditions hold:*

$$\ddot{y} < \sigma_1, , \tag{14}$$
$$(a_1\overline{r} - a_3\ddot{p})^2, < (1 - \frac{\ddot{y}}{\sigma_1})a_4, \tag{15}$$
$$a_3^2\overline{x}^2 < \tilde{N_2}(a_2 + a_3\ddot{x})a_4, \tag{16}$$
$$\tilde{N_2}a_1^2\ddot{x}^2 < (a_2 + a_3\ddot{x})s, \tag{17}$$
$$(a_1\ddot{x} - s)^2 < a_4^2s^2, \tag{18}$$

where,

$$\sigma_1 = (\ddot{x}+d)(\overline{x}+d), \quad \tilde{N_1} = \frac{\ddot{x}+d}{ad} > 0 \quad and \quad \tilde{N_2} = \frac{1}{a_1\overline{r} - a_3\overline{p}} > 0.$$

Proof. For examining the stability of $\ddot{E}_2(\ddot{x}, \ddot{y}, \ddot{p}, \ddot{q}, \ddot{r})$, an appropriate positive definite function is defined as:

$$V_{11} = \left(\ddot{x} - \overline{x} - \overline{x}log\left(\frac{\ddot{x}}{\overline{x}}\right)\right) + \tilde{N_1}\left(\ddot{y} - \overline{y} - \overline{y}log\left(\frac{\ddot{y}}{\overline{y}}\right)\right) + \frac{\tilde{N_2}}{2}(\ddot{p} - \overline{p})^2 + \frac{\tilde{N_3}}{2}(\ddot{q} - \overline{q})^2 + \frac{\tilde{N_4}}{2}(\ddot{r} - \overline{r})^2.$$

When we take the derivative of V_{12} with respect to t, we obtain,

$$\frac{dV_{12}}{dt} = \left(\frac{\ddot{x} - \overline{x}}{\ddot{x}}\right)\frac{d\ddot{x}}{dt} + \tilde{N_1}\left(\frac{\ddot{y} - \overline{y}}{\ddot{y}}\right)\frac{d\ddot{y}}{dt} + \tilde{N_2}\left(\frac{\ddot{p} - \overline{p}}{\ddot{p}}\right)\frac{d\ddot{p}}{dt} + \tilde{N_3}\left(\frac{\ddot{q} - \overline{q}}{\ddot{q}}\right)\frac{d\ddot{q}}{dt} + \tilde{N_4}\left(\frac{\ddot{r} - \overline{r}}{\ddot{r}}\right)\frac{d\ddot{r}}{dt},$$

from (1), (2), (3), (4), (5)

$$\begin{aligned}\frac{dV_{12}}{dt} &= -(\ddot{x}-\overline{x})^2\left(1-\frac{\ddot{y}}{\sigma_1}\right) - (\ddot{y}-\overline{y})^2 b\tilde{N}_1 - (\ddot{p}-\overline{p})^2(\tilde{N}_2 a_2 + \tilde{N}_2 a_3 \ddot{x}) \\ &\quad - (\ddot{q}-\overline{q})^2\tilde{N}_3 a_4 - (\ddot{r}-\overline{r})^2\tilde{N}_4 s - (\ddot{x}-\overline{x})(\ddot{p}-\overline{p})(1-\tilde{N}_2(a_1\overline{r}+a_3\overline{p})) \\ &\quad - (\ddot{x}-\overline{x})(\ddot{q}-\overline{q})(\tilde{N}_3(a_1\overline{r}-a_3\ddot{p})) - (\ddot{p}-\overline{p})(\ddot{q}-\overline{q})(-\tilde{N}_3 a_3\overline{x}) \\ &\quad - (\ddot{x}-\overline{x})(\ddot{y}-\overline{y})\left(\frac{1}{\sigma_1}(\ddot{x}+d-\tilde{N}_1 ad)\right) - (\ddot{p}-\overline{p})(\ddot{r}-\overline{r})(-\tilde{N}_2 a_1\ddot{x}) \\ &\quad - (\ddot{q}-\overline{q})(\ddot{r}-\overline{r})(\tilde{N}_3 a_1\ddot{x} - \tilde{N}_4 s),\end{aligned}$$

where $\sigma_1 = (\ddot{x}+d)(\overline{x}+d)$ and choosing

$$\tilde{N}_1 = \frac{\ddot{x}+d}{ad} > 0, \quad \tilde{N}_2 = \frac{1}{a_1\overline{q}-a_3\overline{p}} > 0.$$

Now, We can write $\frac{dV_{12}}{dt}$ in quadratic form:

$$\begin{aligned}\frac{dV_{12}}{dt} &\le -\frac{c_{11}}{3}(\ddot{x}-\overline{x})^2 - c_{14}(\ddot{x}-\overline{x})(\ddot{q}-\overline{q}) - \frac{c_{44}}{3}(\ddot{q}-\overline{q})^2 - \frac{c_{33}}{3}(\ddot{p}-\overline{p})^2 \\ &\quad - c_{34}(\ddot{p}-\overline{p})(\ddot{q}-\overline{q}) - \frac{c_{44}}{3}(\ddot{q}-\overline{q})^2 + \frac{c_{33}}{3}(\ddot{p}-\overline{p})^2 - c_{35}(\ddot{p}-\overline{p})(\ddot{r}-\overline{r}) \\ &\quad - \frac{c_{55}}{3}(\ddot{r}-\overline{r})^2 - \frac{c_{44}}{3}(\ddot{q}-\overline{q})^2 - c_{45}(\ddot{r}-\overline{r})(\ddot{q}-\overline{q}) - \frac{c_{55}}{3}(\ddot{r}-\overline{r})^2 - \frac{c_{22}}{3}(\ddot{y}-\overline{y})^2,\end{aligned}$$

where

$$\begin{aligned}c_{11} &= 1-\frac{\ddot{y}}{\sigma_1}, \\ c_{22} &= b\tilde{N}_1, \\ c_{33} &= \tilde{N}_2(a_2+a_3\ddot{x}), \\ c_{44} &= a_4, \\ c_{55} &= s, \\ c_{14} &= a_1\overline{r}-a_3\ddot{p}, \\ c_{34} &= -a_3\ddot{x}\overline{x}, \\ c_{35} &= -\tilde{N}_2 a_1\ddot{x}, \\ c_{45} &= a_1\ddot{x}-s.\end{aligned}$$

Applying Sylvester's criterion and setting $\tilde{N}_3 = 1$ and $\tilde{N}_4 = 1$, we establish that $\frac{dV_{12}}{dt}$ remains negative, provided the following inequalities hold:

$$c_{11} > 0, \tag{19}$$

$$c_{11}c_{44} > c_{14}^2, \tag{20}$$

$$c_{33}c_{44} > c_{34}^2, \tag{21}$$

$$c_{33}c_{55} > c_{35}^2, \tag{22}$$

$$c_{44}c_{55} > c_{45}^2. \tag{23}$$

Furthermore, we observe the direct implications: (14) $\implies$ (19), (15) $\implies$ (20), (16) $\implies$ (21), (17) $\implies$ (22) and (18) $\implies$ (23). Thus, V_{12} is a valid Lyapunov function for $\tilde{E}_2$ in Ω, completing the proof. □

8. Sensitivity Analysis of Model A'

Sensitivity analysis is carried out to examine how small changes in key parameters influence the system variables and their stability. It helps identify the most influential parameters governing toxicant accumulation and ecosystem recovery. Here, the sensitivity of the environmental toxicant level q and the delayed toxicant level r is analyzed with respect to the parameters a_0 external toxicant input rate and s delay rate, respectively.

The sensitivity index for the variable q with respect to parameters a_0 is:

$$\Delta_q^{a_0} = \frac{\partial q}{\partial a_0}\frac{a_0}{q} = \frac{a_0}{a_4 q} > 0.$$

Since the sensitivity index is positive, it indicates that the environmental toxicant concentration q increases with higher external toxicant input a_0.

The sensitivity index for the variable r with respect to parameters s is:

$$\Delta_r^{s} = \frac{\partial r}{\partial s}\frac{s}{r} = \frac{q}{r} - 1.$$

The sign of this sensitivity index depends on the ratio $\frac{q}{r}$:

(1) If $\frac{q}{r} > 1$, then $\Delta_r^s > 0$, meaning the delayed toxicant level r increases as s increases.
(2) If $\frac{q}{r} < 1$, then $\Delta_r^s < 0$, meaning the delayed toxicant level r decreases as s increases.

9. Numerical Simulation of Model A

In this section, we conduct numerical simulations to analyze the behavior of the proposed Model A at different equilibrium points. The stability of each equilibrium point is examined using time series plots, and the impact of delay and toxicant is assessed by comparing the full model with a reduced version where toxicant and delay are removed. The numerical solutions are obtained using MATLAB, and all simulations are performed over a sufficiently long time interval to ensure stability analysis.

Stability of Equilibrium Points.

Example 9.1. *For the given parameter values:*

$$D = 0.5, \quad E = 0.4, \quad B = 0.5, \quad A = 2, \quad K = 8, \quad G = 0.3, \quad C = 0.4,$$
$$F = 0.05, \quad P_1 = 1.9, \quad P_2 = 0.1, \quad P_3 = 0.2, \quad q_0 = 0.3, \quad q_1 = 0.2, \quad \gamma_1 = 0.5$$

the system attains an equilibrium point at:

$$\dot{E}_1(4.1167, \quad 0.0000, \quad 2.4270, \quad 0.2865, \quad 0.2865).$$

The corresponding stability graph is presented in Figure 1, which visually confirms the equilibrium's stability.

Example 9.2. *For the given parameter values:*

$$D = 0.5, \quad E = 0.03, \quad B = 0.5, \quad A = 2, \quad K = 10, \quad G = 0.6, \quad C = 0.4,$$
$$F = 0.05, \quad P_1 = 1.9, \quad P_2 = 0.1, \quad P_3 = 0.2, \quad q_0 = 0.3, \quad q_1 = 0.01, \quad \gamma_1 = 0.5$$

the system attains an equilibrium point at:

$$\ddot{E}_2(8.6405, \quad 3.3436, \quad 2.9672, \quad 0.3300, \quad 0.3304).$$

The corresponding stability graph is presented in Figure 2, which visually confirms the equilibrium's stability.

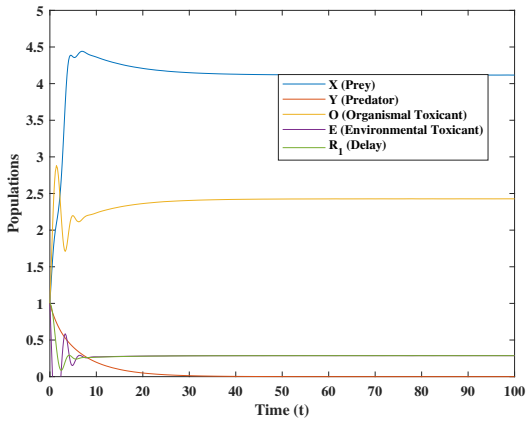

FIGURE 1. Stable dynamics of the system at equilibrium $\dot{E}_1$.

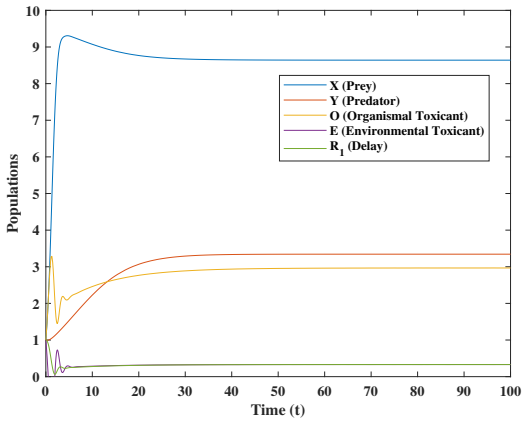

FIGURE 2. Stable dynamics of the system at equilibrium $\ddot{E}_2$.

Comparison of Full and Reduced Models at Equilibrium Points. To understand the influence of toxicants and delays on population dynamics, we compare the equilibrium values from the full and reduced models. The reduced model focuses solely on prey X and predator Y populations, ignoring the effects of toxicants and delays.

Equilibrium $\dot{E}_1$ (Predator-Free State).

(i) Full Model: $(X, Y) = (4.1167, 0)$
(ii) Reduced Model: $(X, Y) = (8.0053, 0)$

Figure 3 illustrates the equilibrium state $\dot{E}_1$. In both models, the predator population is absent $y = 0$, but the prey population is notably larger in the reduced model. This suggests that the additional ecological factors in the full model impose constraints on prey growth.

Equilibrium $\ddot{E}_2$ (Coexistence State).

(i) Full Model: $(X, Y) = (8.6405, 3.3436)$
(ii) Reduced Model: $(X, Y) = (9.1182, 3.3765)$

Figure 4 illustrates the equilibrium state $\ddot{E}_2$, where both species coexist. The equilibrium values are comparable, with the reduced model predicting slightly larger prey and predator populations. This suggests that toxicants and delays impact transient behavior but have little effect on long-term stability.

The significance of ecological complexity in controlling population dynamics and preserving species balance is highlighted by these findings.

10. Conclusion

In this work, the effects of toxicants and time delays were incorporated into a predator-prey model. The theoretical examination confirmed the model's positiveness and boundedness, guaranteeing its viability in biology. After identifying the equilibrium points, the local stability of each was examined using the Routh-Hurwitz criteria and the Jacobian matrix. The global stability was confirmed using Lyapunov's approach with conditions developed using Sylvester's criterion. The sensitivity analysis were performed on two important parameters toxicant input rate a_0 and delay rate s. A solid mathematical basis for comprehending the system's long-term behavior was established by these findings.

By illustrating the stability of equilibrium points and the effects of delays and toxicants, numerical simulations provided additional validation for the analytical findings. Time series plots were used

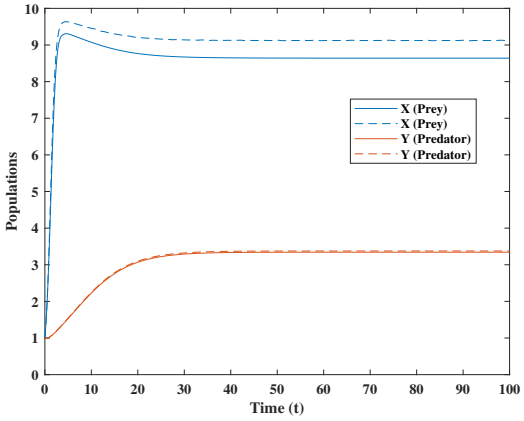

FIGURE 3. A state of equilibrium $\ddot{E}_1$ comparison, prey population is higher in the reduced model since there are no toxicant.

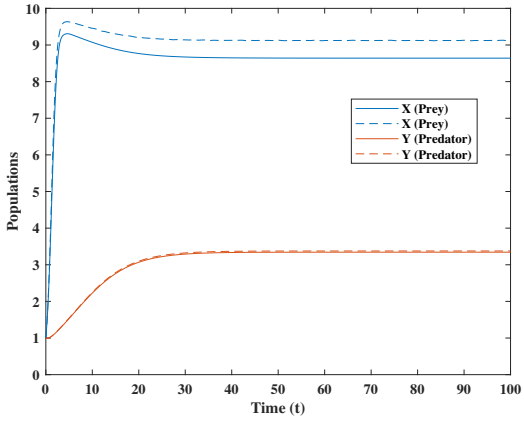

FIGURE 4. A state of equilibrium $\ddot{E}_2$ comparison, both models exhibit coexistence, with the reduced model exhibiting somewhat greater levels.

to confirm the stability of the predator-free equilibrium $\dot{E}_1$, as seen in Figure 1, where the system stabilized at a condition in which there were no predators. Predator and prey populations continue to exist under the specified circumstances, as demonstrated by Figure 2, which also showed the stability of the coexistence equilibrium $\ddot{E}_2$.

Their ecological consequences were further elucidated by comparing the entire model, which included delays and toxicants, with a scaled-down one that does not. As seen in Figure 3, the results showed that the reduced model had a larger prey population in the predator-free condition. This implies that delays and toxicants place extra restrictions on prey growth, halting unchecked growth. Conversely, Figure 4 showed that both models predicted population levels that were similar, with only minor variations, for the coexistence equilibrium. This suggests that whereas delays and toxicants affect short-term dynamics, they have little effect on the system's long-term stability.

Overall, this study highlights the crucial role of toxicants and delays in shaping predator-prey interactions. By integrating theoretical analysis with numerical simulations, we demonstrated that these factors serve as regulatory mechanisms that influence population dynamics. The findings emphasize the importance of considering ecological complexities in population modeling, particularly in environments affected by pollutants and time-dependent interactions.

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