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Stability and bifurcation in a toxicity-affected prey-predator model with prey refuge and Holling type-II response

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Abstract

This study explores the stability and bifurcation dynamics of a toxicity-affected prey-predator model incorporating prey refuge and a Holling Type-II functional response. The model consists of two nonlinear differential equations in which prey populations increase logistically, and the predator functional response is modified by the presence of prey refuge, making the predators' effective predation rate lower. Both species are affected by toxicity, which changes the dynamics of the system by reducing prey reproduction and increasing predator mortality. This model incorporates the combined effects of prey refuge and toxicity within predator-prey interactions, thereby providing a more realistic ecological framework for studying species dynamics under environmental stress. The study establishes the positivity and boundedness of solutions, thereby affirming ecological viability. The local and global stability of the equilibria are analyzed using Lyapunov functions and standard stability criteria. Furthermore, conditions for Hopf bifurcation are established to ascertain the emergence of periodic oscillations. Hopf bifurcation analysis is performed numerically using MATLAB to investigate the impact of toxicity levels and prey refuge on system stability and species persistence. The numerical simulations confirm the analytical findings and provide insights into the ecological effects of environmental toxicity and the protective role of prey refuge. This work provides a theoretical framework for future research on ecosystem sustainability under environmental perturbations and enhances our understanding of toxin-mediated predator-prey interactions.

Keywords. Prey-predator model, Stability, Toxicity, Refuge, Hopf-bifurcation.

2010 Mathematics Subject Classification. 93E15, 60H40, 34D20.

1. Introduction

Interactions between predators and their prey are essential for sustaining ecological balance and promoting biodiversity. Mathematical models are crucial for comprehending these intricate interactions and forecasting population dynamics in response to different ecological factors. A considerable number of ecologists and mathematicians [5, 6, 8, 24] have significantly contributed to the analysis of prey–predator interactions throughout the years. The analysis of persistence and disappearance scenarios continues to be a significant area of focus, even amidst a variety of conceptual interests. Investigating these models within a dynamic context amplifies the importance and fascination of these results.

In recent years, the impact of toxicants on ecosystems has become a significant environmental issue. The research conducted in reference [7, 9, 10, 14, 23] focuses on the incorporation of toxic substances within mathematical models. Aquatic habitats are under-represented in most models, which instead focus on more generic populations of one or two species. When one species releases a toxin into its surroundings, it affects not only that species but also its potential prey and other species' development. Industries are increasingly releasing a significant amount of toxic substances into the environment due to the rising demands of society. The presence of these toxicants primarily impacts the species residing in that environment. The influence of hazardous chemicals [25] introduced within the two-species

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Lotka-Volterra competitive structure is analysed, taking into account that every species releases a harmful material in the presence of the remaining species. The durability features of the system were investigated by the author [3], but there is a restriction to the study because a substantial delay factor is not considered. A species requires a specific duration to reach a maturity level necessary for the production of the toxicant.The author [18] modified the model of [3] by incorporating the delay factor into the analysis.

According to the basic principle of functional response in mathematical ecology, the amount of prey available to a predator determines their feeding rate. This study sheds light on how predators adjust their feeding habits in reaction to changes in the abundance of their prey. Accurately predicting population fluctuations in ecological structures and gathering data on prey-predator interactions require a thorough understanding of functional response. The Michaelis-Menten, Lotka-Volterra, and Monod functional responses are among the most popular [11, 13, 15, 17]. The interaction between wolves and moose in the boreal forests of North America serves as a prime illustration of the Holling type II functional response. When the population of moose rises, wolves experience enhanced access to their prey, leading to a heightened rate of predation. This relationship ultimately reaches a threshold where the amount of predation stabilises, signifying the fact wolves are constrained by factors beyond simple prey abundance, such as social dynamics and the accessibility of predation areas [16]. In a Holling type II functional response, an increase in prey population results in a rise in the predation rate, albeit at a diminishing pace. Initially, as the population of prey increases, predators can consume a greater quantity of prey in a shorter duration. However, there reaches a point when every predator has consumed sufficient resources, and their consumption rates cease to rise. This is a common behaviour exhibited by predators when they require additional time to manage and consume each prey item [12]. In a similar vein, it can be posited that the demise of any prey population exposed to a toxic substance is inevitable. It is conceivable that predators could experience illness as a consequence of preying upon and consuming infected organisms. Consequently, there may be a reduction in the size of predators. Such outcomes motivate investigators to develop a prey-predator framework that includes harmful substances in the prey population [20, 22]. In the natural environment, groups of prey strive to seek safety in regions protected from their predators. Refuges can play a crucial role in reducing a threat of prey disappearance due to predation and in lessening the fluctuations between prey and predator populations [1, 4, 21, 26].

Inspired by the earlier discussions, we examine a model that corresponds with the following three elements: A portion of the prey population seeks refuge, which is introduced to safeguard them from predation, thereby altering the traditional Holling Type-II response. The predator gains advantages from consuming the prey; however, the presence of toxicity results in detrimental effects, contributing to higher mortality rates. The functional response is influenced by a saturation effect, illustrating a situation where predation efficiency decreases at elevated prey densities because of handling time constraints, leading to more complex dynamic behaviours. In order to better understand how these factors affect species persistence and stability, this work investigates a toxicity-affected prey-predator model that incorporates prey refuge and a Holling Type-II functional response.

This paper is organized as follows. Section 2 presents the mathematical model along with the description of its variables and parameters. Section 3 establishes the positivity and boundedness of solutions, ensuring the ecological feasibility of the system. Section 4 investigates the existence of equilibrium points and analyzes their local and global stability properties, including the global stability of the coexistence equilibrium point. Section 5 is devoted to the Hopf bifurcation analysis to determine the onset of population oscillations. Numerical simulations supporting the analytical results and illustrating the effects of toxicity and prey refuge on predator-prey interactions are presented in section 6. Finally, section 7 provides the discussion and concluding remarks.

2. Mathematical framework

This model enhances classical predator-prey theories by incorporating toxicity, prey refuge, and a Type-II functional response into a unified framework, providing important insights for ecological conservation and species management in disturbed environments. As an essential framework for the relationship between predators and prey, we use the following nonlinear framework:

$$\frac{du}{dt} = ru\left(1 - \frac{u}{k}\right) - \frac{a\,(1-m)\,uv}{1 + b\,(1-m)\,u} - \alpha_1 u^3, \tag{2.1}$$

TABLE 1. Description of model (2.1)-(2.2) parameters.

Parameters	Description
u	Prey biomass at time t
v	Predator biomass at time t
r	Per capita growth rate of prey
k	Carrying capacity of prey
a	the capture rate of prey
c	the conversion rate of prey
m	prey refuge rate
b	the half saturation constant
e	Intrinsic death rate of the predator
α_1	Measure of toxicity of prey populations
α_2	Measure of toxicity of predator populations

$$\frac{dv}{dt} = \frac{ca\left(1-m\right)uv}{1+b\left(1-m\right)u} - ev - \alpha_2 v^2, \tag{2.2}$$

where $u\left(t\right) > 0$ and $v\left(t\right) > 0$. Table 1 lists the model (2.1)-(2.2)'s parameters, accompanied by their respective descriptions.

3. Non-Negativity and Boundedness

Here, following the procedures stated in [2], we see that the framework (2.1)-(2.2) has positive and bounded solutions. Under positive initial conditions, the system guarantees the local existence and positivity of solutions, allowing fluctuations of both prey and predator populations within a specified range while maintaining boundedness.

Theorem 3.1. *Any solution to system (2.1)-(2.2) that has initial conditions $u\left(0\right) > 0$, $v\left(0\right) > 0$ and is uniformly bounded stays positive for any time $t > 0$.*

Proof. Now, starting with Equation (2.1), $\frac{du}{dt} = ug_1\left(u,v\right)$, where $g_1\left(u,v\right) = r\left(1-\frac{u}{k}\right) - \frac{a\left(1-m\right)v}{1+b\left(1-m\right)u} - \alpha_1 u^2$, $u\left(t\right) = u\left(0\right)\exp\left(\int_0^t g_1\left(u,v\right)ds\right) > 0$ for $u\left(0\right) > 0$.

Let us move on to Equation (2.2), $\frac{dv}{dt} = vg_2\left(u,v\right)$, where

$$g_2\left(u,v\right) = \frac{ca\left(1-m\right)u}{1+b\left(1-m\right)u} - e - \alpha_2 v, \quad v\left(t\right) = v\left(0\right)\exp\left(\int_0^t g_1\left(u,v\right)ds\right) > 0, \quad \text{for} \quad v\left(0\right) > 0.$$

Thus, $u\left(t\right) > 0$ and $v\left(t\right) > 0$ whenever $u\left(0\right) > 0$, $v\left(0\right) > 0$. We shall now demonstrate the boundedness of system (2.1)-(2.2). Choose the function

$$\chi = u\left(t\right) + \frac{1}{c}v\left(t\right), \tag{3.1}$$

Upon differentiating (3.1)) with respect to t, we obtain the following,

$$\begin{aligned}\frac{d\chi}{dt} &= \frac{du}{dt} + \frac{1}{c}\frac{dv}{dt},\\ &= ru\left(1-\frac{u}{k}\right) - \alpha_1 u^3 - \frac{e}{c}v - \frac{\alpha_2}{c}v^2,\end{aligned}$$

$$\frac{d\chi}{dt} + (e+\alpha_2 v)\chi = ru\left(1-\frac{u}{k}\right) - \alpha_1 u^3 - (e+\alpha_2 v)u.$$

Since $\alpha_1 u^3$ dominates for large u and $(e+\alpha_2 v)\,u$ prevents unbounded growth of v, we estimate:

$$\frac{d\chi}{dt} + (e+\alpha_2 v)\,\chi \leq rk. \tag{3.2}$$

Making use of the differential inequality: which reduces to

$$0 < \chi(t) \leq \frac{rk}{e+\alpha_2}\left(1 - e^{-(e+\alpha_2)t}\right) + \chi(0)\,e^{-(e+\alpha_2)t}.$$

As $t \to \infty$, we see that the right-hand side is bounded by $\dfrac{rk}{e+\alpha_2}$, so $\chi(t)$ is bounded. Thus, all solutions of the system (2.1)-(2.2) remain a bounded subset of $\Re^2_+$. $\square$

4. Existence and stability of equilibria

4.1. **Existence of equilibria.** The equilibria of the system (2.1)-(2.2) are defined as the intersection points of the prey isocline, where $\dot{u} = 0$ and the predator isocline, where $\dot{v} = 0$. The analysis reveals that the system (2.1)-(2.2) contains a maximum of four steady states: one trivial steady state (E_0), two axial steady states $(E_{10},\, E_{01})$ and one interior equilibrium point (E^*), where $E_0 = (0,\,0)$, $E_{10} = (u^*,\,0)$, using the equation (2.1), for the nontrivial equilibrium $u \neq 0$, gives $u^* = \dfrac{-r+\sqrt{r^2+4\alpha_1 r k^2}}{2\alpha_1 k}$, $E_{01} = \left(0,\, -\dfrac{e}{\alpha_2}\right)$, however, this equilibrium is biologically infeasible since the predator population becomes negative, and $E^* = (u^*,\, v^*)$ in which $v^* = \dfrac{1}{\alpha_2}\left[\dfrac{ca(1-m)\,u^\star}{1+b(1-m)\,u^\star} - e\right]$ is positive for $u^* > \dfrac{e}{(ca-eb)(1-m)}$, provided $ca - eb > 0$ and u^* satisfies the quartic equation

$$A_0 u^4 + A_1 u^3 + A_2 u^2 + A_3 u + A_4 = 0, \tag{4.1}$$

where

$$\begin{aligned}
A_0 &= b^2 k \alpha_1 \alpha_2 (1-m)^2,\\
A_1 &= b^2 r \alpha_2 (1-m)^2 + 2kb\alpha_1\alpha_2\,(1-m)\,,\\
A_2 &= b^2 k r \alpha_2 (1-m)^2 - 2rb\alpha_2\,(1-m) - k\alpha_1\alpha_2,\\
A_3 &= a^2 ck(1-m)^2 - abek(1-m)^2 - \alpha_2 r + 2rbk\alpha_2\,(1-m)\,,\\
A_4 &= kr\alpha_2 + aek\,(1-m)\,.
\end{aligned}$$

According to Descartes' Rule of Signs, the number of positive real roots of Equation (4.1) is determined by the number of sign variations in the coefficient sequence A_0, A_1, A_2, A_3, A_4. Since $A_0 > 0, A_1 > 0$, and $A_4 > 0$, the existence of positive roots depends on the signs of A_2 andA_3. In particular:

(i) If $A_2 > 0$ and $A_3 > 0$ then Equation (4.1) admits no positive real roots.
(ii) If exactly one of A_2 or A_3 is negative, then Equation (4.1) admits exactly one positive real root.
(iii) If $A_2 < 0$ and $A_3 < 0$, only if Equation (4.1) admits either two or zero positive real roots.

4.2. **Local Stability Analysis.** The Jacobian matrix for the system (2.1)-(2.2) as follows:

$$J = \begin{pmatrix} r - \dfrac{2ru}{k} - \dfrac{a(1-m)\,v}{(1+b(1-m)\,u)^2} - 3\alpha_1 u^2 & -\dfrac{a(1-m)\,u}{1+b(1-m)\,u} \\ \dfrac{ca(1-m)\,v}{(1+b(1-m)\,u)^2} & \dfrac{ca(1-m)\,u}{1+b(1-m)\,u} - e - 2\alpha_2 v \end{pmatrix}. \tag{4.2}$$

Theorem 4.1. *The trivial steady state $E_0 = (0,\,0)$ is always a saddle point.*

Proof. At $E_0 = (0, 0)$, the Jacobian matrix $J(0, 0) = \begin{pmatrix} r & 0 \\ 0 & -e \end{pmatrix}$ with characteristic roots are given by $\lambda_1 = r$ and $\lambda_2 = -e$ which are opposite sign and hence trivial steady state is always a saddle point. □

Theorem 4.2. *The predator-free equilibrium point* $E_{10} = (u^\star, 0)$ *is asymptotically if* $kr < 2ru^\star + 3k\alpha_1 u^{\star 2}$ *and* $u^\star < \dfrac{e}{(ca + eb)(1 - m)}$.

Proof. At E_{10}, the Jacobian matrix can be obtained as

$J(u^\star, 0) = \begin{pmatrix} r - \dfrac{2ru^\star}{k} - 3\alpha_1 u^{\star 2} & -\dfrac{a(1-m)u^\star}{1 + b(1-m)u^\star} \\ 0 & \dfrac{ca(1-m)u^\star}{1 + b(1-m)u^\star} - e \end{pmatrix}$, with the eigenvalues are given by $\lambda_1 = r - \dfrac{2ru^\star}{k} - 3\alpha_1 u^{\star 2}$, this is negative if $kr < 2ru^\star + 3k\alpha_1 u^{\star 2}$ and $\lambda_2 = \dfrac{ca(1-m)u^\star}{1 + b(1-m)u^\star} - e < 0$ if $u < \dfrac{e}{(ca + eb)(1 - m)}$. Hence $E_{10} = (u^\star, 0)$, is locally asymptotically stable if $kr < 2ru^\star + 3k\alpha_1 u^{\star 2}$ and $u^\star < \dfrac{e}{(ca + eb)(1 - m)}$. □

Theorem 4.3. *By applying the following criteria, we may identify the interior equilibrium point* E^* *where both species can coexist, is*

(i) *Stable node if* $\gamma < 0,\ \varphi > 0,\ \gamma^2 - 4\varphi \geq 0$,
(ii) *Unstable node if* $\gamma > 0,\ \varphi > 0,\ \gamma^2 - 4\varphi \geq 0$,
(iii) *Stable spiral if* $\gamma < 0,\ \gamma^2 - 4\varphi < 0$,
(iv) *Unstable spiral if* $\gamma > 0,\ \gamma^2 - 4\varphi < 0$,
(v) *Unstable saddle if* $\varphi < 0,\ \gamma^2 - 4\varphi \geq 0$,
(vi) *Stable centre if* $\gamma = 0,\ \varphi > 0$.

Proof. At $E^* = (u^*, v^*)$, the Jacobian matrix for the system (2.1)-(2.2) is given by

$$J(E^*) = \begin{pmatrix} -\dfrac{ru^\star}{k} + \dfrac{ab(1-m)^2 u^\star v^\star}{(1 + b(1-m)u^\star)^2} - 2\alpha_1 u^{\star 2} & -\dfrac{a(1-m)u^\star}{1 + b(1-m)u^\star} \\ \dfrac{ca(1-m)v^\star}{(1 + b(1-m)u^\star)^2} & -\alpha_2 v^\star \end{pmatrix}.$$

The characteristic equation for the matrix $J(E^*)$ is given by

$$\lambda^2 - \gamma\lambda + \varphi = 0, \tag{4.3}$$

where $\lambda_1 = \dfrac{\gamma + \sqrt{\gamma^2 - 4\varphi}}{2}$, $\lambda_2 = \dfrac{\gamma - \sqrt{\gamma^2 - 4\varphi}}{2}$.

Here

$$\gamma = Tr(J(E^*)) = -\frac{ru^\star}{k} + \frac{ab(1-m)^2 u^\star v^\star}{(1 + b(1-m)u^\star)^2} - 2\alpha_1 u^{\star 2} - \alpha_2 v^\star,$$

$$\varphi = \det(J(E^*)) = -\alpha_2 v \left[-\frac{ru^\star}{k} + \frac{ab(1-m)^2 u^\star v^\star}{(1 + b(1-m)u^\star)^2} - 2\alpha_1 u^{\star 2} \right] + \frac{ca^2(1-m)^2 u^\star v^\star}{(1 + b(1-m)u^\star)^3}.$$

Case(i): If $\gamma < 0,\ \varphi > 0,\ \gamma^2 - 4\varphi \geq 0$, both eigenvalues λ_1 and λ_2 are negative. So $E^* = (u^*, v^*)$ is a stable node steady state.
Case (ii): If $\gamma > 0,\ \varphi > 0,\ \gamma^2 - 4\varphi \geq 0$, both eigenvalues λ_1 and λ_2 are not negative. Hence $E^* = (u^*, v^*)$ is the unstable node steady state.
Case (iii): If $\gamma < 0,\ \gamma^2 - 4\varphi < 0$, both eigenvalues λ_1 and λ_2 are imaginary with negative real part. So $E^* = (u^*, v^*)$ is a stable spiral equilibrium point.
Case (iv): If $\gamma > 0,\ \gamma^2 - 4\varphi < 0$, both eigenvalues λ_1 and λ_2 are imaginary having non-negative real portion. So $E^* = (u^*, v^*)$ is an unstable spiral steady state.

Case(v): If $\varphi < 0$, $\gamma^2 - 4\varphi \geq 0$, among the eigenvalues λ_1 and λ_2, one is positive and another is negative. So $E^* = (u^*, v^*)$ is an unstable saddle steady state.
Case(vi): If $\gamma = 0$, $\varphi > 0$, both eigenvalues λ_1 and λ_2 are entirely imaginary. Hence $E^* = (u^*, v^*)$ is a stable centre steady state. □

4.3. **Global stability analysis.** In order to establish the global asymptotic stability of the positive equilibrium point, consider the following Lyapunov function:

$$\Lambda (u, v) = (u - u^*) - u^* \log \left(\frac{u}{u^*} \right) + (v - v^*) - v^* \log \left(\frac{v}{v^*} \right). \tag{4.4}$$

The time derivative of Λ along the trajectories of the system is expressed as:

$$\frac{d\Lambda}{dt} = \frac{u - u^*}{u} \frac{du}{dt} + \frac{v - v^*}{v} \frac{dv}{dt},$$

Using the equilibrium conditions
$$r - \frac{ru^*}{k} - \frac{a(1-m)v^*}{1 + b(1-m)u^*} - \alpha_1 (u^*)^2 = 0, \text{ and } \frac{ca(1-m)u^*}{1 + b(1-m)u^*} - e - \alpha_2 v^* = 0.$$
The derivative reduces to

$$\begin{aligned} \frac{d\Lambda}{dt} &= -\frac{r}{k}(u - u^*)^2 - \alpha_1 (u - u^*)^2 (u + u^*) - \alpha_2 (v - v^*)^2 \\ &\quad - a(1-m)(u - u^*) \left[\frac{v}{1 + b(1-m)u} - \frac{v^*}{1 + b(1-m)u^*} \right] \\ &\quad + ca(1-m)(v - v^*) \left[\frac{u}{1 + b(1-m)u} - \frac{u^*}{1 + b(1-m)u^*} \right]. \end{aligned}$$

Now,

$$\frac{u}{1 + b(1-m)u} - \frac{u^*}{1 + b(1-m)u^*} = \frac{u - u^*}{(1 + b(1-m)u)(1 + b(1-m)u^*)}.$$

Hence,

$$ca(1-m)(v - v^*) \left[\frac{u}{1 + b(1-m)u} - \frac{u^*}{1 + b(1-m)u^*} \right] = \frac{ca(1-m)}{(1 + b(1-m)u)(1 + b(1-m)u^*)} (u - u^*)(v - v^*).$$

Define $M = \dfrac{ca(1-m)}{(1 + b(1-m)u)(1 + b(1-m)u^*)}$.
Therefore,

$$\frac{d\Lambda}{dt} \leq -\frac{r}{k}(u - u^*)^2 - \alpha_1 (u - u^*)^2 (u + u^*) - \alpha_2 (v - v^*)^2 + M(u - u^*)(v - v^*).$$

Applying, $xy \leq \dfrac{x^2 + y^2}{2}$, we obtain

$$M(u - u^*)(v - v^*) \leq \frac{M}{2}(u - u^*)^2 + \frac{M}{2}(v - v^*)^2.$$

Thus, $\dfrac{d\Lambda}{dt} \leq -\left[\dfrac{r}{k} + \alpha_1 (u + u^*) - \frac{M}{2} \right] (u - u^*)^2 - \left[\alpha_2 - \frac{M}{2} \right] (v - v^*)^2$.
Hence, $\dfrac{d\Lambda}{dt} \leq 0$, provided $\dfrac{r}{k} + \alpha_1 (u + u^*) > \dfrac{M}{2}$, $\alpha_2 > \dfrac{M}{2}$.
Therefore, by LaSalle's invariance principle, the positive equilibrium point $E^* = (u^*, v^*)$ is globally asymptotically stable.

5. Hopf Bifurcation

Assuming α_1 as a variable parameter, the characteristic equation of the system (2.1)-(2.2) for the Jacobian matrix $J(E^*)$ can be expressed as

$$\lambda^2 - T(\alpha_1)\lambda + D(\alpha_1) = 0, \tag{5.1}$$

where $T(\alpha_1)$ and $D(\alpha_1)$ represent the sum and product of the elements of the Jacobian matrix associated with the non-trivial steady state E^*. In this case, the resilience of the non-trivial steady state E^* goes from stable to unstable as the sign of the real portion λ changes from negative to positive. This transformation takes place as a consequence of a Hopf bifurcation once the characteristic equation (5.1) has two entirely imaginary roots. Assume that $\alpha_1 = \alpha_1^{HB}$, is the location of those entirely imaginary roots. In that case,$T\left(\alpha_1^{HB}\right) = 0$ and $D\left(\alpha_1^{HB}\right) > 0$. We prove that the system has a Hopf bifurcation at $\alpha_1 = \alpha_1^{HB}$ in the following theorem.

Theorem 5.1. *In relation to the bifurcation parameter α_1, the system (2.1)-(2.2) undergoes a Hopf bifurcation at the non-trivial steady state E^* when $\alpha_1 = \alpha_1^{HB}$, given that $T\left(\alpha_1^{HB}\right) = 0$, $D\left(\alpha_1^{HB}\right) > 0$ and $\left[\frac{dT}{d\alpha_1}\right]_{\alpha_1=\alpha_1^{HB}} \neq 0$.*

Proof. At $\alpha_1 = \alpha_1^{HB}, T\left(\alpha_1^{HB}\right) = 0$ and $\left[\frac{dT}{d\alpha_1}\right]_{\alpha_1=\alpha_1^{HB}} \neq 0$. The characteristic equation (5.1) involves entirely imaginary roots, expressed in $\lambda_1 = i\sqrt{D\left(\alpha_1^{HB}\right)}$ and $\lambda_2 = -i\sqrt{D\left(\alpha_1^{HB}\right)}$. As a result, in every open neighbourhood of α_1^{HB}, the roots of the characteristic equation (5.1) take the forms $\lambda_1 = \omega(\alpha_1) + i\zeta(\alpha_1)$ and $\lambda_2 = \omega(\alpha_1) - i\zeta(\alpha_1)$, in which $\omega(\alpha_1)$ and $\zeta(\alpha_1)$ are α_1's real-valued functions. The Hopf-Bifurcation Theorem [19] states that a system experiences a Hopf bifurcation when the transversality criterion $\left[\frac{d}{d\alpha_1}\left(\mathrm{Re}\left(\lambda_i\left(\alpha_1\right)\right)\right)\right]_{\alpha_1=\alpha_1^{HB}} = \left[\frac{d\omega(\alpha_1)}{d\alpha_1}\right]_{\alpha_1=\alpha_1^{HB}} \neq 0$ is met.
From (5.1), one can obtain by using $\lambda = \omega(\alpha_1) + i\zeta(\alpha_1)$

$$(\omega(\alpha_1) + i\zeta(\alpha_1))^2 - T(\alpha_1)(\omega(\alpha_1) + i\zeta(\alpha_1)) + D(\alpha_1) = 0.$$

By applying differentiation to either side pertaining to the α_1, we obtain

$$2(\omega(\alpha_1) + i\zeta(\alpha_1))\left(\dot\omega(\alpha_1) + i\dot\zeta(\alpha_1)\right) - T(\alpha_1)\left(\dot\omega(\alpha_1) + i\dot\zeta(\alpha_1)\right) - \dot T(\alpha_1)(\omega(\alpha_1) + i\zeta(\alpha_1)) + \dot D(\alpha_1) = 0.$$

When we compare the two sides' real and imaginary components, the following results are obtained:

$$(2\omega - T)\dot\omega - 2\dot\zeta\zeta - \dot T\omega + \dot D = 0, \tag{5.2}$$

$$(2\zeta)\dot\omega + (2\omega - T)\dot\zeta - \dot T\zeta = 0. \tag{5.3}$$

At the Hopf bifurcation point $\alpha_1 = \alpha_1^{HB}$, we have $\omega = 0$, $T\left(\alpha_1^{HB}\right) = 0$, $\zeta = \sqrt{D\left(\alpha_1^{HB}\right)}$.
Substituting these into the imaginary part equation, gives $2\zeta\dot\omega - \dot T\zeta = 0$.
Since $\zeta \neq 0$, it follows that $2\dot\omega - \dot T = 0$, which gives

$$[\dot\omega]_{\alpha_1=\alpha_1^{HB}} = \left[\frac{d\omega(\alpha_1)}{d\alpha_1}\right]_{\alpha_1=\alpha_1^{HB}} = \frac{1}{2}\left[\frac{dT(\alpha_1)}{d\alpha_1}\right]_{\alpha_1=\alpha_1^{HB}}.$$

Therefore, $\left[\frac{d\omega(\alpha_1)}{d\alpha_1}\right]_{\alpha_1=\alpha_1^{HB}} \neq 0$, whenever $\left[\frac{dT(\alpha_1)}{d\alpha_1}\right]_{\alpha_1=\alpha_1^{HB}} \neq 0$. Hence, the transversality condition is satisfied. Consequently, the system undergoes a Hopf bifurcation at the coexistence equilibrium point E^* when $\alpha_1 = \alpha_1^{HB}$. □

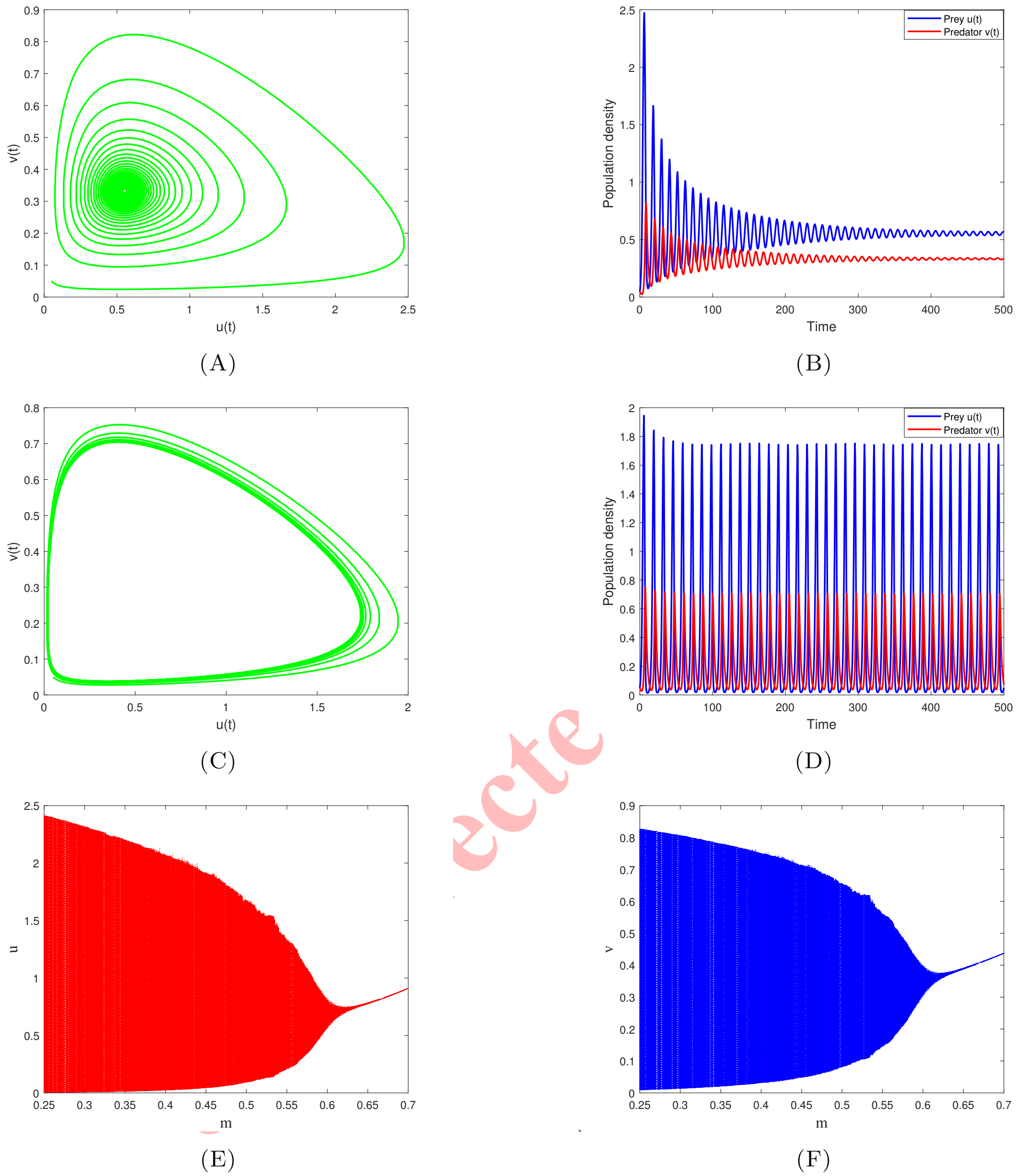

FIGURE 1. Phase portraits, Time series and Hopf bifurcation diagrams of the system (2.1)–(2.2) for the refuge parameter values $m = 0.576$ and $m = 0.376$.

6. Numerical Simulations

The present approach is framed around the premise that each of the two rival species generates a toxin that affects the other, and there is a notable scarcity of quantitatively valid data regarding this form of interaction. Consequently, we shall present a set of hypothetical data solely to exemplify the findings expressed in the preceding section. The subsequent numerical illustrations represent various scenarios pertaining to distinct data sets.

The parameters under consideration are $r = 0.98$, $k = 6.67418$, $a = 7.3494$, $b = 0.736$, $e = 0.453, \alpha_1 = 0.052$, $\alpha_2 = 0.1$, $c = 0.331$. Figures 1(A)–1(D) illustrate that the top-left phase portrait exhibits a stable spiral, signifying that the system ultimately converges to a stable equilibrium characterised by damped oscillations. The phase portrait in the bottom-left Figure 1(C) indicates the presence of a limit cycle, which suggests that population sizes will exhibit sustained oscillations over time. The top-right Figure 1(A) plot illustrates an initial oscillatory behaviour that progressively stabilises, indicating a movement towards equilibrium. The plot Figure 1(D) in the bottom-right quadrant illustrates sustained oscillations, characterised by the continuous cycling of both prey and predator populations at relatively high amplitudes over time. It can be concluded that an increased refuge level (m) decreases predation pressure on the prey, which may result in sustained oscillations. A diminished refuge level enables predators to capture a greater number of prey, potentially resulting in stabilisation as prey populations decline. Furthermore, Figures 1(E) and 1(F) illustrates the Hopf bifurcation occurring for $m = 0.55$, which results in the development of periodic oscillations within both prey and predator populations. In conditions of diminished refuge, the pressure from predators escalates, resulting in enduring cycles between predator and prey populations. This illustrates the significant impact of refuge accessibility on the stability of populations and the development of oscillatory dynamics within predator-prey relationships.

We will now examine the effects of the prey toxicity element (α_1) utilising the same selection of settings employed in Figure 1. An increase in prey toxicity α_1 clearly results in a decline in the population amounts of both prey and predator species. An increase in the parameter α_1 stabilises the system by diminishing oscillations and guiding it towards equilibrium, as illustrated in Figures 2(A) and 2(B). A reduced level of α_1 facilitates sustained predator-prey cycles, as illustrated in Figure 2(b), suggesting that less toxic prey contribute to continuing population oscillations. A Hopf bifurcation takes place at the threshold value of α_1, resulting in a transition from periodic oscillations to a steady state, as illustrated in Figures 2(E) and 2(F). For low levels of $\alpha_1\,(< 0.02)$, the system demonstrates oscillatory behaviour; however, for larger amounts of $\alpha_1\,(> 0.05)$, the system settles down, suggesting that severe prey toxicity suppresses predator-prey cycles.

Additionally, we carry out an investigation involving different predator toxic amounts (α_2) using identical settings as illustrated in Figure 1. The rise in toxicity among predators results in a corresponding increase in prey populations, while simultaneously causing a decline in the numbers of predators. At elevated amounts of α_2, the populations tend to stabilise at reduced levels. The increased toxicity in predators diminishes their ability to effectively consume prey, leading to a rise in prey populations and a corresponding decline in predator populations, as illustrated in Figures 3(A)–3(D). When α_2 surpasses a critical threshold, the system shifts from periodic oscillations to a steady state, indicating the occurrence of a Hopf bifurcation and the disappearance of periodic solutions, as illustrated in Figures 3(E) and 3(F).

Next, we looked at how an attack by a predator affected its prey using the following parameters:$r = 0.98$, $k = 6.67418$, $b = 0.736$, $e = 0.453, m = 0.576, c = 0.331, \alpha_1 = 0.052$, $\alpha_2 = 0.0998$. Figures 4(A)–4(D) illustrate that an increase in the predation rate (a) enhances predator efficiency, resulting in stronger predator-prey interactions. However, once a critical threshold is reached, the system attains stability due to limited prey availability and other regulatory effects. A Hopf bifurcation occurs at a critical value of (a), causing the system to transition from a stable equilibrium to periodic oscillations, as illustrated in Figure 4(E) and 4(F).

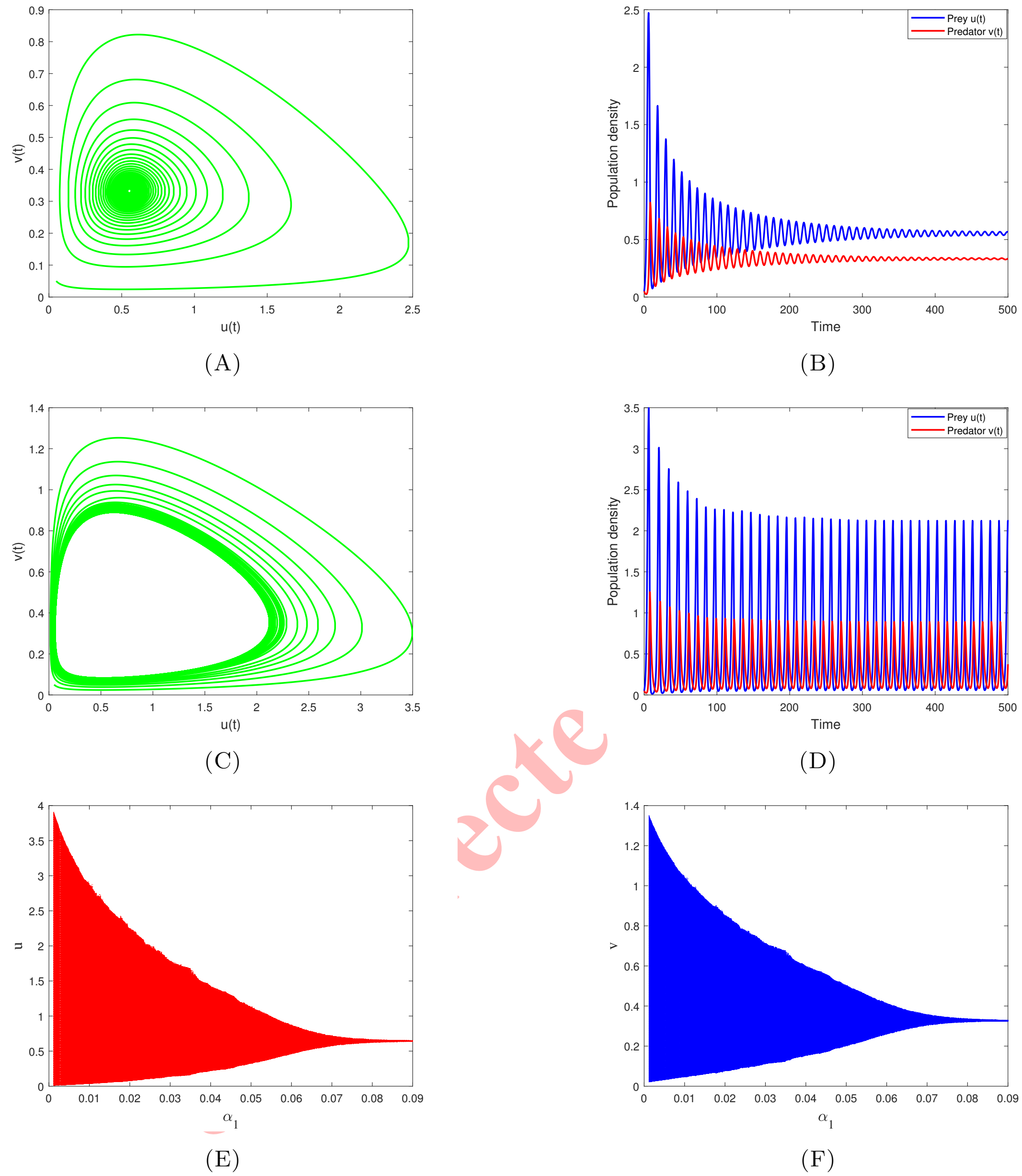

FIGURE 2. Phase portraits, Time series and Hopf bifurcation diagrams of the system (2.1)–(2.2) for the prey toxicity parameter values $\alpha_1 = 0.052$ and $\alpha_1 = 0.0012$.

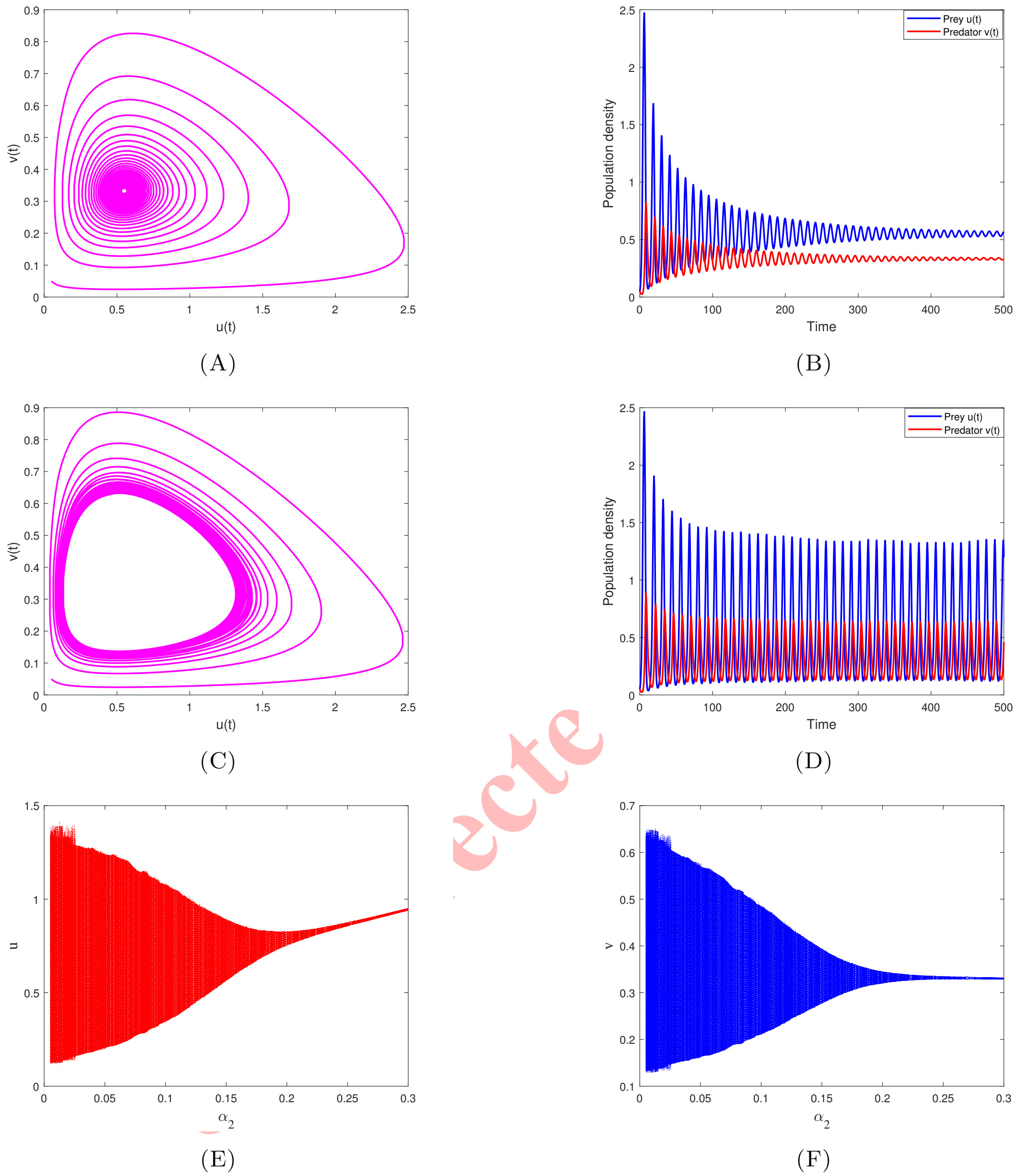

FIGURE 3. Phase portraits, Time series and Hopf bifurcation diagrams of the system (2.1)–(2.2) for the predator toxicity parameter values $\alpha_2 = 0.0935$ and $\alpha_2 = 0.0015$.

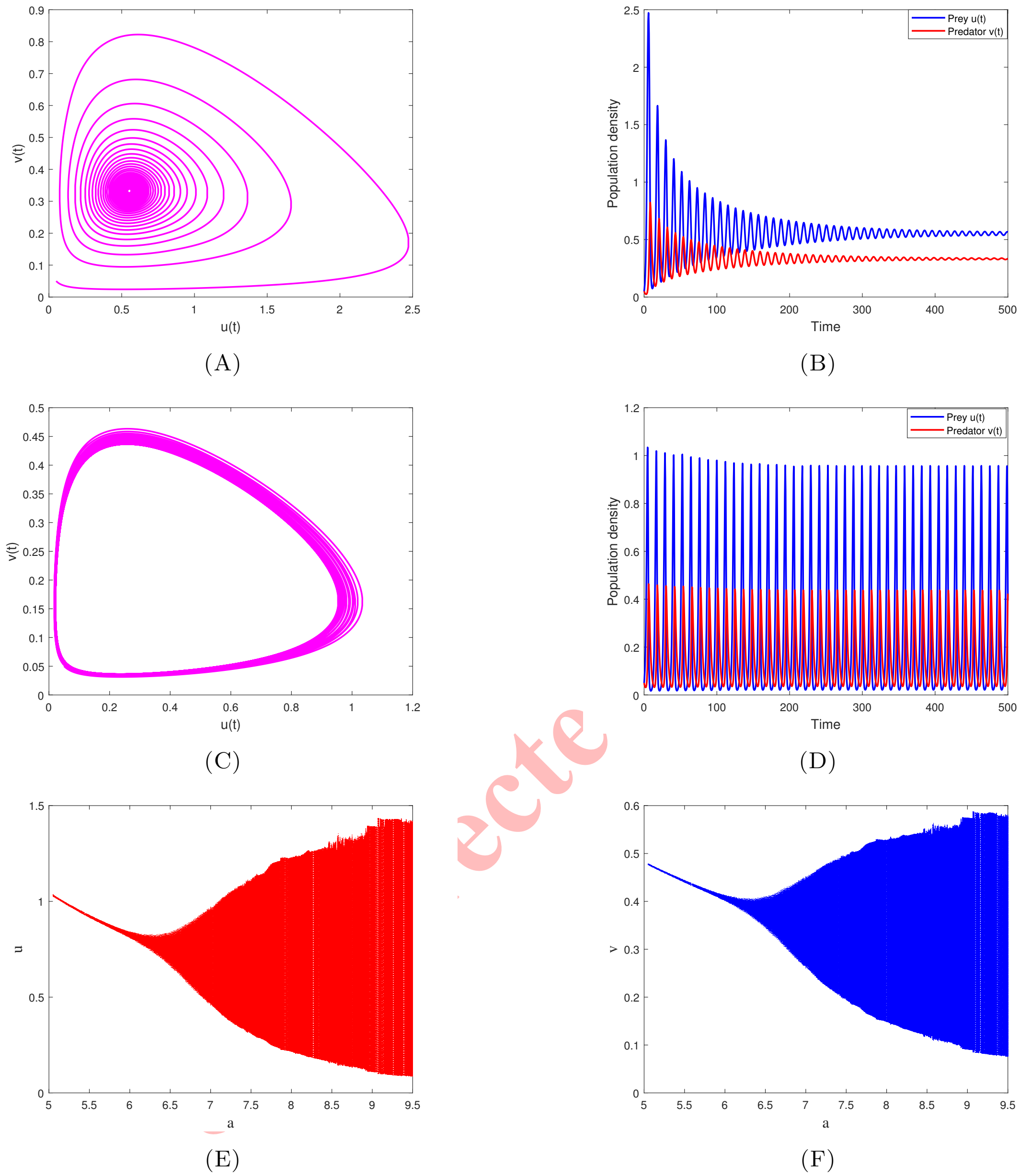

FIGURE 4. Phase portraits, Time series and Hopf bifurcation diagrams of the system (2.1)–(2.2) for the prey capture rate parameter values $a = 7.3494$ and $a = 11.642$.

7. Discussion and Conclusion

Evaluating the dynamical complexity of ecosystems and determining the ecological factors which impact such complexity are essential parts of ecological research. A number of mathematical frameworks were developed in the last several years to examine diverse ecological mechanisms under varied environmental restrictions. The predator-prey interaction model serves as a specific instance of an ecological model. A thorough examination of a predator-prey framework reveals the essential components that significantly influence the development of community structure, integrating various ecological or physiological characteristics while also considering the preservation of biodiversity. Toxicity serves as a significant physiological component for both prey and predator organisms. Toxicants present in prey species have the potential to influence behavioural changes in predator species. This investigation introduces a predator-prey framework that integrates the impacts of harmful effects over the reproductive as well as survival rates of both prey and predator organisms. The proposed model integrates prey refuge alongside a Holling type II functional response in order to increase the credibility of the behaviour.

This study confirms the positivity and boundedness of the system, thereby ensuring ecological feasibility. The analysis focusses on the local and global stability of steady states, along with the derivation of conditions for Hopf bifurcation to identify the initiation of population oscillations. Employing bifurcation analysis in MATLAB, this study investigates the impact of critical parameters on system stability, highlighting transitions among stable coexistence, periodic cycles, and species extinction. The inference indicates that an increased refuge (m) level diminishes predation pressure on prey, resulting in sustained oscillations. In contrast, a reduced refuge (m) level enables predators to increase their consumption of prey, leading to stabilisation as prey populations decline. This analysis illustrates the significant impact of refuge accessibility on population stability, determining the shift between equilibrium and oscillatory dynamics in predator-prey interactions.

The same holds true for predators: when prey is more toxic (α_1), fewer predators will be able to survive, which could lead to extinction. Additionally, we examine the influence of the predator toxicity parameter (α_2) on the dynamics between predator and prey. Initially, an increase in (α_2) results in a rise in the prey population and a decline in the predator population. At elevated values of (α_2) the populations reach a state of stability at reduced levels. The findings indicate that raised predator toxicity may influence predator-prey dynamics by reducing population oscillations and fostering a more stable coexistence. Higher prey capture rates (a) initially lead to increased oscillations in predator populations, but ultimately serve to regulate both populations, thereby preventing extreme fluctuations. The analysis indicates that prey capture efficiency is a critical factor influencing the stability and oscillatory dynamics of the predator-prey system.

Conflict of interest

The authors declare no conflict of interest.

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Author contributions

B. Ramesh: Conceptualization, mathematical modeling, analytical methods, bifurcation analysis, and manuscript drafting. K. Ramesh: Numerical simulations, visualization, interpretation of results, manuscript editing, and review. Both authors have read and approved the final version of the manuscript.

Use of AI tools

Not applicable.

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