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DYNAMICS OF THE PREDATOR–PREY MODEL WITH PREY GROUP DEFENSE, ALTERNATIVE FOOD, AND PREDATOR ECOLOGICAL COSTS: STABILITY ANALYSIS AND NUMERICAL SIMULATION

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ABSTRACT

This study examines the dynamics of a predator-prey model that integrates three main biological mechanisms: the availability of alternative food for predators, group defense among prey, and the ecological cost in the form of additional predator mortality. Alternative food is modeled as an energy subsidy that affects the saturation level of interactions and biomass conversion efficiency, while group defense among prey represents the collective ability of prey to reduce predation success. The ecological cost is introduced as a consequence of increased predator dependence on additional resources. Analytically, it is proven that the system has solutions that exist, are unique, and remain within the relevant biological domain. Equilibrium point analysis shows the presence of both predator extinction and a stable interior of both populations, with stability determined by key model parameters. The results show more specifically that the interaction among the three factors produces different long-term outcomes. When prey group defense becomes sufficiently strong, predation efficiency decreases substantially, so the predator population cannot be maintained and the system tends toward predator extinction. When prey defense is weaker, alternative food at moderate levels can compensate for reduced prey capture and support stable coexistence between predator and prey. However, when the availability of alternative food becomes excessive, the associated ecological cost increases predator mortality to the point that the energetic benefit of alternative food is no longer sufficient to sustain predator persistence, and the system shifts back to the predator-free state. Numerical simulations verify the analytical results and confirm that ecosystem persistence is governed by the nonlinear balance between prey protection, external food subsidy, and predator mortality cost. These results indicate that the interaction between external energy subsidies and prey defense mechanisms plays a crucial role in determining the long-term structural dynamics of ecosystems.

***Keywords**: Alternative food, Ecological costs, Population dynamics, Prey group defense, Predator–prey.*

1. Introduction

Mathematical modeling in population dynamics is rooted in classical ideas about population growth and interspecies interactions. The concept of exponential growth introduced by Malthus [1] laid the foundation for modern population dynamics theory. The predator–prey interaction model was first formally formulated by Lotka [2] and Volterra [3], demonstrating that the interaction between two species can produce periodic oscillations due to feedback mechanisms

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between the prey and predator populations. Further development was carried out through stochastic formulations by Leslie and Gower [4] and graphical representations and stability analyses by Rosenzweig and MacArthur [5], emphasizing the importance of predator functional response and environmental carrying capacity in determining system dynamics. Since then, the predator-prey model has become a fundamental framework in the study of mathematical ecology.

As the study of biomathematics has advanced, predator-prey models have been expanded from various perspectives to enhance biological realism and the complexity of dynamics. Many studies have included factors related to ecology and disease, such as how infections affect prey and predators, and different methods of harvesting them. Other research has looked at changes in time patterns using models that account for memory effects, age differences, and competition within the same species. Furthermore, diverse supplementary biological mechanisms, such as the administration of toxins to predators, anti-predator behavior, prey protection, and fear effects, have been examined to investigate bifurcation, limit cycles, and alterations in the system's stability structure [12, 13, 14, 15]. Overall, these findings show that predator-prey interactions are very affected by changes in biological mechanisms, so adding new factors to the model is important for better understanding stability changes and how populations survive.

In recent years, special attention has been given to group defense mechanisms in prey as an adaptive strategy that influences the success of predation. Ye and Zhao [16] showed that the combination of the Allee effect and group defense can alter system stability and give rise to complex bifurcations. Rahman et al. [17] noted that providing safe spaces for prey, along with a Holling type IV functional response, increases the area where populations can survive. Pal et al. [18] combined the Allee effect, fear, and prey protection, leading to multistability. Further studies by Resmawan et al. [19, 20] highlighted that the strength of group defense can influence whether predators die out or live alongside their prey. This literature indicates that group defense in prey is a significant structural mechanism in determining ecosystem sustainability.

Conversely, the significance of alternative food sources for predators has emerged as a focal point of current research. Debnath et al. [21] showed that the presence of supplemental food can alter the bifurcation structure of the system, leading to complex dynamics. Gökçe et al. [22] reported that providing alternative food can expand the domain of stable coexistence but also has the potential to induce instability. Sil et al. [23] and Yang and Fan [24] showed that when the Allee effect and extra food interact, the resulting changes can be easily affected by adjustments in certain factors. A recent study by Resmawan et al. [25] confirmed that alternative food does not always strengthen predator persistence but can instead lead to ambivalent dynamics, depending on its interactions with mortality and functional responses.

Although many predator–prey models have incorporated mechanisms such as disease, harvesting, Allee effects, memory, age structure, prey refuge, and supplementary food for predators, the ecological importance of studying prey group defense and alternative food within a unified framework has not been sufficiently emphasized. In many ecosystems, these two mechanisms arise simultaneously rather than independently. For instance, prey such as small fish, ungulates, or colonial birds often reduce predation risk through aggregation, coordinated movement, or collective vigilance, whereas predators facing reduced hunting success may increasingly rely on alternative food sources such as carrion, discarded fish, anthropogenic food waste, or other non-prey resources. This ecological situation is important because prey group defense directly weakens predation efficiency, while alternative food may temporarily buffer predator energy limitation and thereby alter the long-term balance between extinction and coexistence.

A concrete ecological example can be found in marine and coastal systems, where schooling prey reduce capture success through coordinated group behavior, while predators may shift part of their feeding activity toward fishery discards or carrion. Although such alternative food provides an additional energy subsidy, it may also increase predator mortality. Predators attracted to these food sources often spend more time in risky foraging areas, experience greater exposure to fishing gear, vessel traffic, contaminants, pathogens, or intensified competition, and may also incur behavioral and physiological costs associated with diet switching. As a result, the availability of alternative food does not necessarily strengthen predator persistence; instead, it may generate a trade-off between energetic gain and ecological cost. Despite this clear ecological relevance, most existing mathematical studies still treat prey group defense and alternative food separately, so the combined effect of prey protection, energy subsidy, and predator mortality cost remains poorly understood. This gap forms the main motivation for the present study.

This article introduces a new predator–prey model that combines the prey's group defense based on their numbers, the availability of alternative food for the predator, and ecological costs modeled as additional predator mortality resulting from the use of such alternative food. This research stands out because it integrates all three mechanisms into a single mathematical framework, making it possible to examine how the protective effect of prey aggregation interacts with external energy subsidies and the mortality burden borne by predators. The aim of this study is to construct a differential equation model representing these interacting mechanisms, analyze its fundamental properties and equilibrium structure, examine the local stability of the predator-free and interior equilibrium points, and verify the analytical findings through numerical simulations in order to clarify the ecological consequences of variation in key parameters.

2. Model

The developed model is a two-species predator–prey system that takes into account three main mechanisms: logistic growth of the prey, the predator's functional response influenced by alternative food and group defense by the prey, as well as the ecological costs of consuming alternative food on predator mortality. Let $N(t)$ denote the prey density and $P(t)$ denote the predator density at time t. The prey population is assumed to grow logistically with an intrinsic growth rate r and environmental carrying capacity K. Predatory interactions are modeled through a nonlinear predation rate that depends on the densities of both populations and an inhibiting factor in the denominator.

The functional response denominator consists of three main components. The parameter β represents the basic saturation constant in the predation process. The component κmA describes the influence of alternative food on handling time or the predator's consumption capacity, with A indicating the availability of alternative food (assumed to be constant), m representing the predator's effectiveness in locating alternative food, and κ representing the weighting factor for the influence of alternatives on predation saturation. Furthermore, μN^2 represents the effect of prey group defense, which increases nonlinearly with prey density, causing the effective predation rate to decrease more sharply at high densities.

The growth of predators depends on the conversion of biomass from prey and alternative food through the conversion coefficient ζ. Unlike conventional models, in this study it is assumed that consumption of alternative food also incurs an ecological cost that increases the effective mortality of predators. This cost is modeled through the parameter θ, so that the predator's mortality rate becomes $\delta + \theta mA$, with δ representing the natural mortality rate of predators.

Based on these assumptions, the system model is expressed as follows:

$$\begin{aligned}\frac{dN}{dt} &= rN\left(1-\frac{N}{K}\right) - \frac{\alpha NP}{\beta+\kappa mA+\mu N^2},\\ \frac{dP}{dt} &= \frac{\zeta(N+mA)P}{\beta+\kappa mA+\mu N^2} - (\delta+\theta mA)P.\end{aligned} \tag{1}$$

with $r, K, \alpha, \beta, \kappa, m, A, \mu, \zeta, \delta, \theta > 0$. The parameter values and their biological interpretations used in the model are summarized in Table 1.

Table 1. Model parameters, descriptions, and units

Parameter	Description	Unit
r	Intrinsic growth rate of prey	time^{-1}
K	Environmental carrying capacity of prey	population density
α	Predation rate coefficient	time^{-1}
β	Predation saturation constant	population density
κ	Handling-time ratio between alternative food and prey	dimensionless
m	Predator efficiency in locating alternative food	dimensionless
A	Quantity of alternative food available to predators	population density
μ	Effectiveness of prey group defense	population density^{-1}
ζ	Biomass conversion efficiency from prey/alternative food to predator reproduction	dimensionless
δ	Natural mortality rate of predators	time^{-1}
θ	Ecological cost rate associated with the utilization of alternative food by predators	time^{-1}

3. Results and Discussions

3.1. Fundamental Properties of the Model

This section discusses the fundamental properties of system (1) that ensure the model is mathematically well-defined and biologically consistent. In particular, it is proven that for every nonnegative initial condition, the system admits a locally existing and unique solution. Furthermore, it is shown that the nonnegative quadrant is a positively invariant set of the system, implying that solutions starting from nonnegative initial conditions remain nonnegative for all time within the interval of existence. These results guarantee that the constructed population model has valid biological meaning prior to further analysis of equilibrium points and their stability.

Theorem 3.1 (Local Existence and Uniqueness)**.** *For every initial condition* $(N(0), P(0)) = (N_0, P_0) \in \mathbb{R}^2_{\geq 0}$, *system* (1) *admits a unique solution* $(N(t), P(t))$ *that exists locally on a time interval* $[0, T)$, *for some* $T > 0$.

Proof. Write system (1) in vector form

$$\dot{X} = F(X), \quad X = (N, P),$$

with

$$F(N,P) = \begin{pmatrix} rN\left(1-\frac{N}{K}\right) - \dfrac{\alpha NP}{\beta+\kappa mA+\mu N^2} \\ \dfrac{\zeta(N+mA)P}{\beta+\kappa mA+\mu N^2} - (\delta+\theta mA)P \end{pmatrix}.$$

Since all parameters are positive and

$$\beta + \kappa m A + \mu N^2 \geq \beta > 0, \quad \forall N \geq 0,$$

each component of $F(N, P)$ is a combination of polynomial and rational functions that are well-defined on the entire $\mathbb{R}^2$.

Moreover, the first-order partial derivatives of F with respect to N and P are continuous on every bounded subset of $\mathbb{R}^2$. Hence, $F \in C^1$ on every compact set and satisfies the local Lipschitz condition with respect to (N, P). Therefore, by the Picard–Lindelöf existence and uniqueness theorem [26], for every initial condition (N_0, P_0) there exists a unique solution that exists locally on a time interval $[0, T)$ for some $T > 0$. ■

Theorem 3.2 (Positivity)**.** *If the initial conditions satisfy* $N_0 \geq 0$ *and* $P_0 \geq 0$*, then the solution of system (1) satisfies*

$$N(t) \geq 0, \quad P(t) \geq 0,$$

for every $t \geq 0$ *as long as the solution is defined.*

Proof. Note that at the boundary $N = 0$, with $P \geq 0$, from the first equation of system (1), we obtain

$$\left.\frac{dN}{dt}\right|_{N=0} = 0,$$

since each term in the first equation contains the factor N. Thus, the trajectory cannot penetrate from $N = 0$ to $N < 0$.

Furthermore, at the boundary $P = 0$ with $N \geq 0$, from the second equation of system (1), we obtain

$$\left.\frac{dP}{dt}\right|_{P=0} = 0,$$

since each term in the second equation contains the factor P. Thus, the trajectory cannot penetrate from $P = 0$ to $P < 0$.

Since the system is autonomous and the vector field does not point outside the boundaries of the nonnegative quadrant, it follows that $\mathbb{R}^2_{\geq 0}$ is a positively invariant set. ■

Biologically, this result ensures that if the initial densities of prey and predator are nonnegative, then the populations will never become negative over time, making the model consistent with the interpretation of population as a density quantity that cannot be negative.

3.2. Equilibrium Point and Its Existence

The equilibrium points of system (1) are obtained by solving the following equations simultaneously:

$$rN\left(1 - \frac{N}{K}\right) - \frac{\alpha N P}{\beta + \kappa m A + \mu N^2} = 0, \tag{2}$$

$$\frac{\zeta(N + mA)P}{\beta + \kappa m A + \mu N^2} - (\delta + \theta m A)P = 0. \tag{3}$$

From equations (2)–(3), three types of equilibrium points are obtained, namely the trivial equilibrium point, the predator-free equilibrium point, and the interior equilibrium point. The existence of these equilibrium points is presented in Theorem 3.3.

Theorem 3.3. *Let* $\Delta = \zeta\left(\zeta + 4\mu mA(\delta + \theta mA)\right) - 4\mu(\delta + \theta mA)^2(\beta + \kappa mA)$, *and*

$$N_{2,3} = \frac{\zeta \pm \sqrt{\Delta}}{2(\delta + \theta mA)\mu}.$$

Furthermore, define

$$P_i = \frac{r(\beta + \kappa mA + \mu N_i^2)(K - N_i)}{K\alpha}, i = 2, 3.$$

System (1) *has three types of equilibrium points describing the existence of populations in the ecosystem, namely:*

1. ***Trivial equilibrium point***

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

which indicates the absence of populations in the ecosystem. This point always exists in $\mathbb{R}^2_{\geq 0}$.

2. ***Predator-free equilibrium point***

$$E_1 = (K, 0),$$

which represents the condition where predators are absent from the ecosystem. This point always exists in $\mathbb{R}^2_{\geq 0}$.

3. ***Interior equilibrium points***

$$E_i = (N_i, P_i), \quad i = 2, 3,$$

which indicate the coexistence of prey and predator populations. The interior equilibrium exists if and only if

$$\Delta > 0 \quad \textit{and} \quad K > N_i.$$

Equivalently,

$$\mu < \frac{\zeta\left(\zeta + 4\mu mA(\delta + \theta mA)\right)}{4(\delta + \theta mA)^2(\beta + \kappa mA)}.$$

If $\Delta = 0$, *then there exists a unique interior equilibrium (double root), namely*

$$E_4 = (N_4, P_4),$$

with

$$N_4 = \frac{\zeta}{2\mu(\delta + \theta mA)}.$$

Proof. From equation (3), we obtain

$$P = 0 \quad \text{or} \quad \frac{\zeta(N + mA)}{\beta + \kappa mA + \mu N^2} - (\delta + \theta mA) = 0.$$

Case 1: $P = 0$**.** From equation (2), we obtain $N = 0$ or $N = K$, producing the equilibrium points

$$E_0 = (0, 0) \quad \text{and} \quad E_1 = (K, 0),$$

which always exist in $\mathbb{R}^2_{\geq 0}$.

Case 2: $P > 0$. In this case,

$$\frac{\zeta(N_i + mA)}{\beta + \kappa mA + \mu N_i^2} = \delta + \theta mA,$$

which leads to the quadratic equation

$$(\delta + \theta mA)\mu N_i^2 - \zeta N_i + (\beta + \kappa mA)(\delta + \theta mA) - \zeta mA = 0. \tag{4}$$

From equation (4), we obtain

$$N_{2,3} = \frac{\zeta \pm \sqrt{\Delta}}{2(\delta + \theta mA)\mu},$$

where

$$\Delta = \zeta\left(\zeta + 4\mu mA(\delta + \theta mA)\right) - 4\mu(\delta + \theta mA)^2(\beta + \kappa mA).$$

If $\Delta > 0$, equivalently

$$\mu < \frac{\zeta\left(\zeta + 4\mu mA(\delta + \theta mA)\right)}{4(\delta + \theta mA)^2(\beta + \kappa mA)},$$

then there exist two distinct real roots, namely N_2 and N_3. If $\Delta = 0$, equivalently

$$\mu = \frac{\zeta\left(\zeta + 4\mu mA(\delta + \theta mA)\right)}{4(\delta + \theta mA)^2(\beta + \kappa mA)},$$

then there exists a double real root, namely

$$N_4 = \frac{\zeta}{2(\delta + \theta mA)\mu}.$$

Furthermore, for $N > 0$, from equation (2) we obtain

$$r\left(1 - \frac{N_i}{K}\right) - \frac{\alpha P_i}{\beta + \kappa mA + \mu N_i^2} = 0,$$

which gives

$$P_i = \frac{r(\beta + \kappa mA + \mu N_i^2)(K - N_i)}{K\alpha}, \quad i = 2, 3.$$

It follows that $P_i > 0$ if and only if $K > N_i$. Therefore, the interior equilibrium points are

$$E_i = (N_i, P_i), \quad i = 2, 3,$$

or E_4 in the case of a double root. ■

3.3. Local Stability

From system (1), define

$$f(N, P) = rN\left(1 - \frac{N}{K}\right) - \frac{\alpha NP}{\beta + \kappa mA + \mu N^2},$$

$$g(N, P) = \frac{\zeta(N + mA)P}{\beta + \kappa mA + \mu N^2} - (\delta + \theta mA)P.$$

The Jacobian matrix of system (1) is given by

$$J(N,P)=\begin{pmatrix}\dfrac{\partial f}{\partial N} & \dfrac{\partial f}{\partial P}\\ \dfrac{\partial g}{\partial N} & \dfrac{\partial g}{\partial P}\end{pmatrix}=\begin{pmatrix}J_{11} & J_{12}\\ J_{21} & J_{22}\end{pmatrix}.$$

The partial derivatives are computed as follows.

$$\begin{aligned}
J_{11}&=\frac{\partial}{\partial N}\left[rN-\frac{rN^2}{K}-\frac{\alpha NP}{\beta+\kappa mA+\mu N^2}\right]\\
&=r-\frac{2r}{K}N-\frac{\alpha P(\beta+\kappa mA+\mu N^2)-2\alpha\mu N^2P}{(\beta+\kappa mA+\mu N^2)^2}\\
&=r-\frac{2r}{K}N-\frac{(\beta+\kappa mA-\mu N^2)\alpha P}{(\beta+\kappa mA+\mu N^2)^2}.\\
J_{12}&=\frac{\partial}{\partial P}\left[rN\left(1-\frac{N}{K}\right)-\frac{\alpha NP}{\beta+\kappa mA+\mu N^2}\right]\\
&=-\frac{\alpha N}{\beta+\kappa mA+\mu N^2}.\\
J_{21}&=\frac{\partial}{\partial N}\left[\frac{\zeta(N+mA)P}{\beta+\kappa mA+\mu N^2}-(\delta+\theta mA)P\right]\\
&=\frac{\zeta P(\beta+\kappa mA+\mu N^2)-2\mu\zeta(N+mA)NP}{(\beta+\kappa mA+\mu N^2)^2}\\
&=\frac{(\beta+\kappa mA-\mu N^2-2\mu mAN)\zeta P}{(\beta+\kappa mA+\mu N^2)^2}.\\
J_{22}&=\frac{\partial}{\partial P}\left[\frac{\zeta(N+mA)P}{\beta+\kappa mA+\mu N^2}-(\delta+\theta mA)P\right]\\
&=\frac{\zeta(N+mA)}{\beta+\kappa mA+\mu N^2}-(\delta+\theta mA).
\end{aligned}$$

Thus, the Jacobian matrix becomes

$$J(N,P)=\begin{pmatrix} r-\dfrac{2r}{K}N-\dfrac{(\beta+\kappa mA-\mu N^2)\alpha P}{(\beta+\kappa mA+\mu N^2)^2} & -\dfrac{\alpha N}{\beta+\kappa mA+\mu N^2}\\ \dfrac{(\beta+\kappa mA-\mu N^2-2\mu mAN)\zeta P}{(\beta+\kappa mA+\mu N^2)^2} & \dfrac{\zeta(N+mA)}{\beta+\kappa mA+\mu N^2}-(\delta+\theta mA)\end{pmatrix}.$$

3.3.1. Stability Analysis of the Trivial Equilibrium Point

We begin the local stability analysis by examining the behavior of the system in the neighborhood of the trivial equilibrium point. The result of this analysis is stated in Theorem 3.4.

Theorem 3.4. *The trivial equilibrium point,* $E_0=(0,0)$, *is always unstable, indicating that total extinction of the population is impossible.*

Proof. The Jacobian matrix evaluated at $E_0=(0,0)$ is

$$J(E_0)=\begin{pmatrix} r & 0\\ 0 & \dfrac{\zeta mA}{\beta+\kappa mA}-(\delta+\theta mA)\end{pmatrix}.$$

This matrix produces the eigenvalue $\lambda_1=r>0$, hence E_0 is always unstable. ■

3.4. Stability Analysis of the Predator-Free Equilibrium Point

Next, we analyze the local stability of the predator-free equilibrium point to determine the conditions under which the predator population can persist or eventually become extinct. The result is presented in Theorem 3.5.

Theorem 3.5. *The predator-free equilibrium point* $E_1 = (K, 0)$ *is locally asymptotically stable if*

$$\mu > \frac{\zeta(K + mA) - (\delta + \theta mA)(\beta + \kappa mA)}{(\delta + \theta mA)K^2},$$

unstable (saddle point) if

$$\mu < \frac{\zeta(K + mA) - (\delta + \theta mA)(\beta + \kappa mA)}{(\delta + \theta mA)K^2},$$

and nonhyperbolic if equality holds.

Proof. The Jacobian matrix evaluated at $E_1 = (K, 0)$ is

$$J(E_1) = \begin{pmatrix} -r & -\dfrac{\alpha K}{\beta + \kappa mA + \mu K^2} \\ 0 & \dfrac{\zeta(K + mA)}{\beta + \kappa mA + \mu K^2} - (\delta + \theta mA) \end{pmatrix}.$$

This matrix yields two eigenvalues, namely

$$\lambda_1 = -r < 0 \quad \text{and} \quad \lambda_2 = \frac{\zeta(K + mA)}{\beta + \kappa mA + \mu K^2} - (\delta + \theta mA).$$

Therefore, the local stability of E_1 is determined by the sign of λ_2. If

$$\mu > \frac{\zeta(K + mA) - (\delta + \theta mA)(\beta + \kappa mA)}{(\delta + \theta mA)K^2},$$

then $\lambda_2 < 0$. Since $\lambda_1 < 0$ also holds, both eigenvalues are negative, and hence E_1 is locally asymptotically stable.

On the other hand, if

$$\mu < \frac{\zeta(K + mA) - (\delta + \theta mA)(\beta + \kappa mA)}{(\delta + \theta mA)K^2},$$

then $\lambda_2 > 0$. Since $\lambda_1 = -r < 0$, the Jacobian matrix at E_1 has one negative eigenvalue and one positive eigenvalue. Therefore, E_1 is an unstable saddle point.

Finally, if

$$\mu = \frac{\zeta(K + mA) - (\delta + \theta mA)(\beta + \kappa mA)}{(\delta + \theta mA)K^2},$$

then $\lambda_2 = 0$, so E_1 is nonhyperbolic. ■

3.5. Stability Analysis of Interior Equilibrium Point

We now proceed to investigate the local stability of the interior equilibrium point, which represents the coexistence state of both predator and prey populations. The corresponding analytical result is stated in Theorem 3.6.

Theorem 3.6. *Let* $E_i = (N_i, P_i)$, $i = 2,3,4$, $\Delta = \zeta(\zeta + 4\mu mA(\delta + \theta mA)) - 4\mu(\delta + \theta mA)^2(\beta + \kappa mA)$, $N_{2,3} = \dfrac{\zeta \pm \sqrt{\Delta}}{2(\delta + \theta mA)\mu}$, $N_4 = \dfrac{\zeta}{2(\delta + \theta mA)\mu}$, *and* $P_i = \dfrac{r(\beta + \kappa mA + \mu N_i^2)(K - N_i)}{K\alpha}$. *Next, assume that* $K > N_i$.

1. *If* $4\beta\delta^2\mu + 4\delta^2\mu\kappa mA < 4\delta\mu\zeta mA + \zeta^2$, *then* E_2 *is unstable (saddle point).*
2. *If* $\mu < \dfrac{\beta + \kappa mA}{(2mA + N_3)N_3}$ *and* $\alpha < \dfrac{r(\beta + \kappa mA + \mu N_3^2)^2}{2K\mu N_3 P_3}$, *then* E_3 *is locally asymptotically stable.*
3. E_4 *is nonhyperbolic.*

PROOF. Based on equations (2)–(3), if $N_i \neq 0$ and $P_i \neq 0$, then

$$r = \frac{rN_i}{K} + \frac{\alpha P_i}{\beta + \kappa mA + \mu N_i^2}, \qquad \delta + \theta mA = \frac{\zeta(N_i + mA)}{\beta + \kappa mA + \mu N_i^2}.$$

Thus, the Jacobian matrix for $E_i = (N_i, P_i)$ is

$$J(E_i) = \begin{pmatrix} J_{11} & J_{12} \\ J_{21} & 0 \end{pmatrix} \tag{5}$$

where

$$\begin{aligned} J_{11} &= r - \frac{2r}{K}N_i - \frac{(\beta + \kappa mA - \mu N_i^2)\alpha P_i}{(\beta + \kappa mA + \mu N_i^2)^2}, \\ J_{12} &= -\frac{\alpha N_i}{\beta + \kappa mA + \mu N_i^2}, \\ J_{21} &= \frac{(\beta + \kappa mA - \mu N_i^2 - 2\mu mAN_i)\zeta P_i}{(\beta + \kappa mA + \mu N_i^2)^2}. \end{aligned}$$

Furthermore,

$$\begin{aligned} N_i &= \frac{\zeta \pm \sqrt{\Delta}}{2(\delta + \theta mA)\mu}, \quad i = 2,3, \\ N_4 &= \frac{\zeta}{2(\delta + \theta mA)\mu}, \\ P_i &= \frac{r(\beta + \kappa mA + \mu N_i^2)(K - N_i)}{K\alpha}, \quad i = 2,3,4. \end{aligned}$$

From equation (5), the determinant and trace of the Jacobian matrix are obtained as

$$\det(J(E_i)) = \frac{\alpha\zeta(\beta + \kappa mA - 2\mu mAN_i - \mu N_i^2)N_i P_i}{(\beta + \kappa mA + \mu N_i^2)^3}, \tag{6}$$

$$\operatorname{tr}(J(E_i)) = \frac{2K\alpha\mu N_i^2 P_i - rN_i(\beta + \kappa mA + \mu N_i^2)^2}{K(\beta + \kappa mA + \mu N_i^2)^2}. \tag{7}$$

The equilibrium E_i is locally asymptotically stable if

$$\det(J(E_i)) > 0 \quad \text{and} \quad \operatorname{tr}(J(E_i)) < 0.$$

Suppose $\Gamma = \beta + \kappa m_A - 2\mu m_A N_i - \mu N_i^2$, then the value of $\det(J_{E_i})$ depends on Γ. From equation (6), it can be shown that

$$\begin{aligned}\Gamma &= \beta + \kappa m_A - 2\mu m_A N_i - \mu N_i^2\\ &= \beta + \kappa m_A - 2\mu m_A \left(\frac{\zeta \pm \sqrt{\Delta}}{2(\delta + \theta m_A)\mu}\right) - \mu \left(\frac{\zeta \pm \sqrt{\Delta}}{2(\delta + \theta m_A)\mu}\right)^2\\ &= \beta + \kappa m_A - \frac{m_A(\zeta \pm \sqrt{\Delta})}{\delta + \theta m_A} - \frac{1}{4\mu}\left(\frac{\zeta \pm \sqrt{\Delta}}{\delta + \theta m_A}\right)^2.\end{aligned} \tag{8}$$

- For $E_2 = (N_2, P_2)$.
  From equation (8), it follows that

$$\begin{aligned}\Gamma = \beta &+ \kappa m_A\\ &- \frac{m_A\left(\zeta + \sqrt{\zeta(\zeta + 4\mu m_A(\delta + \theta m_A)) - 4\mu(\delta + \theta m_A)^2(\beta + \kappa m_A)}\right)}{\delta + \theta m_A}\\ &- \frac{1}{4\mu}\left(\frac{\zeta + \sqrt{\zeta(\zeta + 4\mu m_A(\delta + \theta m_A)) - 4\mu(\delta + \theta m_A)^2(\beta + \kappa m_A)}}{\delta + \theta m_A}\right)^2.\end{aligned}$$

  Based on the existence conditions in Theorem 3.3, we have $4\mu(\delta + \theta m_A)^2(\beta + \kappa m_A) < \zeta\big(\zeta + 4\mu m_A(\delta + \theta m_A)\big)$, then

$$\begin{aligned}\Gamma &< \beta + \kappa m_A - \frac{m_A\left(\zeta + \sqrt{\zeta(\zeta + 4\mu m_A(\delta + \theta m_A)) - \zeta(\zeta + 4\mu m_A(\delta + \theta m_A))}\right)}{\delta + \theta m_A}\\ &\quad - \frac{1}{4\mu}\left(\frac{\zeta + \sqrt{\zeta(\zeta + 4\mu m_A(\delta + \theta m_A)) - \zeta(\zeta + 4\mu m_A(\delta + \theta m_A))}}{\delta + \theta m_A}\right)^2\\ &= \beta + \kappa m_A - \frac{\zeta m_A}{\delta + \theta m_A} - \frac{\zeta^2}{4\mu(\delta + \theta m_A)^2}\\ &= \frac{4\mu(\beta + \kappa m_A)(\delta + \theta m_A)^2 - 4\mu\zeta m_A(\delta + \theta m_A) - \zeta^2}{4\mu(\delta + \theta m_A)^2}\\ &= \frac{4\mu(\beta + \kappa m_A)(\delta + \theta m_A)^2 - \zeta\big(\zeta + 4\mu m_A(\delta + \theta m_A)\big)}{4\mu(\delta + \theta m_A)^2} < 0.\end{aligned}$$

  Since $\Gamma < 0$, we obtain $\det(J_{E_2}) < 0$. Hence, the equilibrium point E_2 is unstable.

- For $E_3 = (N_3, P_3)$.
  From equation (6), it can be shown that $\det(J_{E_3}) > 0$ if $\mu < \dfrac{\beta + \kappa mA}{(2mA + N_3)N_3}$. Furthermore, from equation (7), $\mathrm{Tr}(J_{E_3}) < 0$ if the following condition holds:

$$2K\alpha\mu N_3^2 P_3 < rN_3(\beta + \kappa mA + \mu N_3^2)^2 \iff \alpha < \frac{r(\beta + \kappa mA + \mu N_3^2)^2}{2K\mu N_3 P_3}.$$

  Therefore, the equilibrium point E_3 is locally asymptotically stable if

  (i) $\mu < \dfrac{\beta + \kappa mA}{(2mA + N_3)N_3}$,

(ii) $\alpha < \dfrac{r(\beta + \kappa mA + \mu N_3^2)^2}{2K\mu N_3 P_3}$.

- For $E_4 = (N_4, P_4)$ with $\Delta = 0$.
  From equation (8), we obtain

$$\begin{aligned}\Gamma &= \beta + \kappa mA - \frac{\zeta mA}{\delta + \theta mA} - \frac{\zeta^2}{4\mu(\delta + \theta mA)^2} \\ &= \frac{4\mu(\beta + \kappa mA)(\delta + \theta mA)^2 - \zeta(\zeta + 4\mu mA(\delta + \theta mA))}{4\mu(\delta + \theta mA)^2}.\end{aligned}$$

  The equilibrium point E_4 exists when

$$4\mu(\kappa mA + \beta)(\delta + \theta mA)^2 = \zeta(\zeta + 4\mu mA(\delta + \theta mA)).$$

  This condition implies that $\Gamma = 0$, and therefore from equation (6) we obtain $\det(J(E_4)) = 0$. Since the determinant equals the product of the eigenvalues of $J(E_4)$, it follows that at least one eigenvalue is zero. An equilibrium point is said to be nonhyperbolic when its Jacobian matrix has at least one eigenvalue with zero real part. Therefore, E_4 is nonhyperbolic.

■

3.6. Numerical Simulation

In this section, numerical simulations are conducted to verify and illustrate the results of the local stability analysis obtained in the previous section. The simulations are carried out using the fourth–fifth order Runge–Kutta method via the `solve_ivp` function in Python, with sufficiently small numerical tolerances to ensure computational accuracy. Several positive initial conditions are employed in the phase portraits to clarify the trajectory directions and the dynamical structure of the system around the equilibrium points, allowing the stability characteristics to be visually observed. The basic parameter values used in the simulations are provided in Table 2.

Table 2. Parameter values in numerical simulation

Parameter	**Description**	**Value**
r	Intrinsic growth rate of prey	1.0
K	Environmental carrying capacity	10.0
α	Predation rate	1.0
β	Saturation constant	1.0
κ	Handling time ratio of alternative food	0.5
m	Predator efficiency in finding alternative food	1.0
ζ	Biomass conversion coefficient	0.5
δ	Natural mortality rate of predator	0.4
θ	The ecological cost rate associated with the utilization of alternative food by predators	0.1
μ	Prey group defense intensity	Varied
A	Quantity of alternative food	Varied

Parameter μ is varied to investigate the effect of prey group defense effectiveness on changes in the stability of equilibrium points. Meanwhile, parameter A is varied to evaluate the impact of alternative food availability on the dynamical structure of the system, particularly in determining whether the system evolves toward predator extinction or stable coexistence.

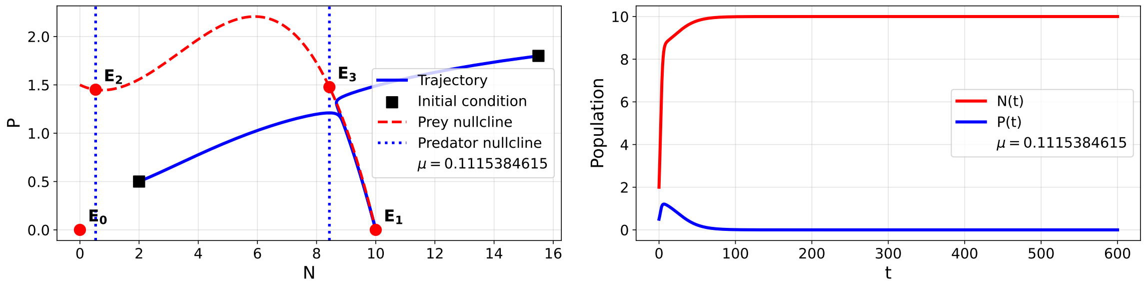
3.6.1. The Influence of Prey Group Defense on Ecosystem Sustainability

In this section, numerical simulations are performed to evaluate the role of prey group defense effectiveness on the system dynamics. In system (1), parameter μ represents the effectiveness level of collective defense mechanisms in the prey population, such as group formation, coordinated avoidance, or other defensive behaviors. Mathematically, this parameter appears in the denominator $\beta + \kappa mA + \mu N^2$, so increasing μ amplifies the saturation effect and reduces predation efficiency as prey density increases. Consequently, larger values of μ correspond to stronger prey capacity to mitigate predation pressure.

To examine this effect, the parameter values listed in Table 2 are employed, with $A = 0.1$. Two conditions are considered for parameter μ: one in which μ is relatively large so that $\lambda_2 < 0$ is obtained, and another in which μ is relatively small so that $\lambda_2 > 0$ is obtained, respectively, where

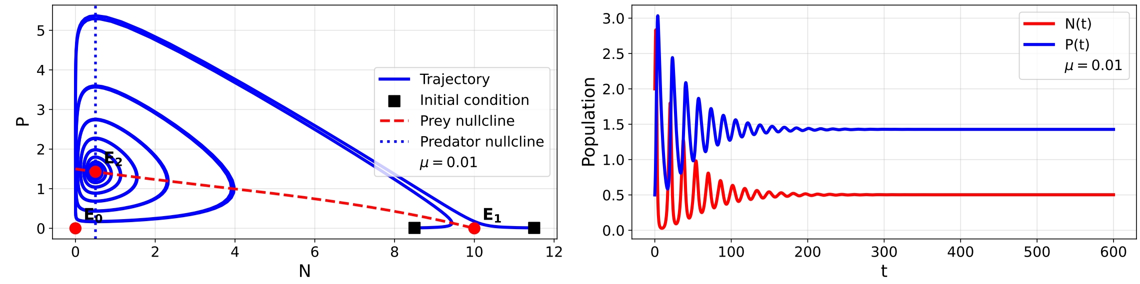
$$\lambda_2 = \frac{\zeta(K + mA)}{\beta + \kappa mA + \mu K^2} - (\delta + \theta mA).$$

The change in the sign of λ_2 determines the local stability of the predator-free equilibrium point E_1. The numerical simulation results for both conditions are presented in Figure 1.



(a) Phase portrait (E_1 stable)

(b) Time series (converging to E_1)



(c) Phase portrait (E_1 saddle)

(d) Time series (converging to interior equilibrium)

Figure 1. System dynamics for two values of the prey group defense parameter μ. Panels (a) and (b) correspond to $\mu = 0.1115384615$, where $E_1 = (K, 0)$ is locally asymptotically stable. Panels (c) and (d) correspond to $\mu = 0.01$, where E_1 is a saddle point and the trajectories converge to the interior equilibrium.

Figures 1(a) and 1(b) illustrate the system dynamics for $\mu = 0.1115384615$. The phase portrait in Figure 1(a) shows that the solution trajectories gradually approach the predator-free equilibrium point $E_1 = (K, 0)$. The corresponding time series in Figure 1(b) show that the predator population $P(t)$ declines toward zero, while the prey population $N(t)$ approaches the carrying capacity K. These results confirm that E_1 is locally asymptotically stable under this parameter condition. Ecologically, this indicates that when prey group defense is sufficiently strong, predation becomes ineffective, so the predator cannot maintain its population and eventually goes extinct, whereas the prey population survives and grows toward its environmental limit.

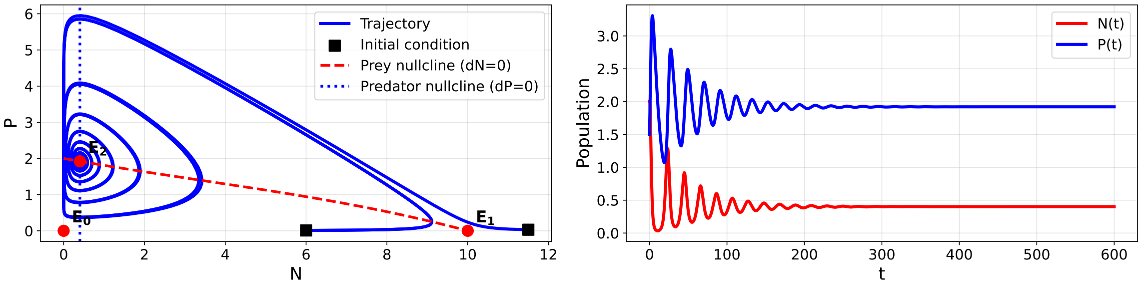
In contrast, Figures 1(c) and 1(d) show the system dynamics for $\mu = 0.01$. The phase portrait in Figure 1(c) indicates that E_1 is a saddle point, since the trajectories move away from it and approach an interior equilibrium point. The corresponding time series in Figure 1(d) show that neither population goes extinct; instead, both populations converge to positive steady-state values. Therefore, panel (d) represents the time series converging to the interior equilibrium, not to E_1. Ecologically, this means that when prey group defense is weaker, predators are still able to exploit the prey population effectively, allowing both species to persist and eventually reach a stable coexistence state.

Overall, these numerical results are consistent with the previously derived local stability analysis. Changes in the value of μ alter the stability of the predator-free equilibrium E_1 and determine whether the system evolves toward predator extinction or stable coexistence. Thus, the numerical simulations not only validate the analytical findings but also clarify the ecological role of prey group defense effectiveness in regulating predator persistence.

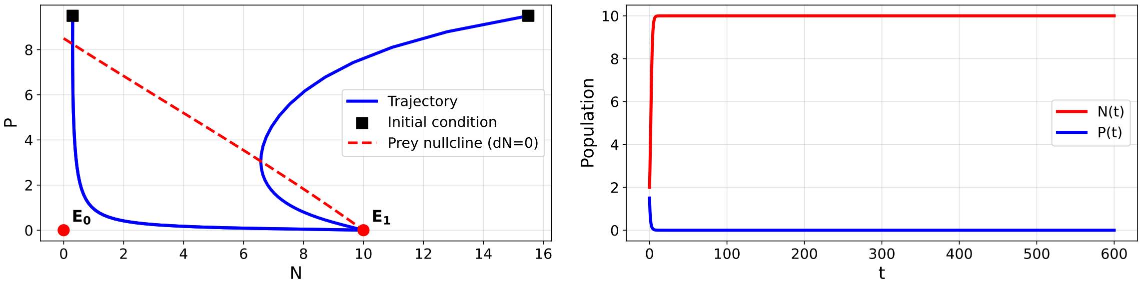
3.6.2. The Influence of Alternative Food on Ecosystem Sustainability

In this section, numerical simulations are performed to demonstrate how changes in the quantity of alternative food, A, affect the stability of the system's equilibrium points and induce transitions in the dynamics from one equilibrium state to another. Analytically, the stability of the predator-free equilibrium point, $E_1 = (K, 0)$, is determined by the sign of the eigenvalue

$$\lambda_2(E_1) = \frac{\zeta(K + mA)}{\beta + \kappa mA + \mu K^2} - (\delta + \theta mA).$$



(a) Phase portrait (A = 2) (b) Time series (A = 2)



(c) Phase portrait (A = 15) (d) Time series (A = 15)

Figure 2. System dynamics for two values of A. Panels (a) and (b) correspond to $A = 2.0$, where the trajectories converge to the interior equilibrium, while panels (c) and (d) correspond to $A = 15.0$, where the trajectories converge to the predator-free equilibrium $E_1 = (K, 0)$.

Changes in the value of A directly affect both components on the right-hand side, namely by increasing the contribution of alternative food to predator growth while simultaneously increasing additional mortality through the term θmA. Consequently, variation in A may alter the sign of λ_2 at E_1, ultimately determining whether the predator persists or goes extinct.

In this simulation, the parameter values listed in Table 2 are used with $\mu = 0.01$. Two levels of alternative food are considered to represent distinct cases: $A = 2.0$, corresponding to limited alternative food availability, and $A = 15.0$, representing abundant alternative food availability. These two cases are selected to illustrate how variations in A influence the stability of E_1. The numerical simulation results for both conditions are presented in Figure 2.

Figures 2(a) and 2(b) illustrate the system dynamics when the amount of alternative food is limited ($A = 2.0$). The phase portrait in Figure 2(a) shows that the solution trajectories move toward the interior equilibrium E_2. The time series in Figure 2(b) indicate that both populations converge to stable positive levels. This behavior confirms that predator and prey achieve stable coexistence when alternative food is available in moderate quantities. In contrast, Figures 2(c) and 2(d) present the dynamics when alternative food is abundant ($A = 15.0$). The phase portrait in Figure 2(c) shows that the trajectories move toward the predator-free equilibrium $E_1 = (K, 0)$. The time series in Figure 2(d) demonstrate that the predator population declines toward zero, while the prey population increases toward the carrying capacity K, indicating predator extinction.

Biologically, predator extinction under abundant alternative food occurs because increasing A not only enhances energy input through the mA component but also simultaneously increases the ecological cost rate associated with the utilization of alternative food by predators, represented by the additional mortality term θmA. Moreover, larger values of mA amplify the saturation component in the denominator, thereby limiting the efficiency of energy conversion. At sufficiently high levels of A, the combined effects of increased ecological cost and saturation dominate the energetic benefits, resulting in negative net predator growth. Consequently, the predator fails to sustain its population, and the system ultimately shifts toward the predator-free equilibrium E_1.

3.6.3. Numerical Illustration of the Nonhyperbolic Equilibrium Point

To numerically illustrate the existence of the nonhyperbolic equilibrium point E_4, we consider the same baseline parameter values as in Table 2 and fix $A = 1.0$. Under this parameter setting, the discriminant of the interior equilibrium equation is $\Delta = \zeta\,(\zeta + 4\mu mA(\delta + \theta mA)) - 4\mu(\delta + \theta mA)^2(\beta + \kappa mA)$.

The critical condition $\Delta = 0$ yields

$$\mu_c = \frac{\zeta^2}{4(\delta + \theta mA)\,((\delta + \theta mA)(\beta + \kappa mA) - \zeta mA)} = 0.5.$$

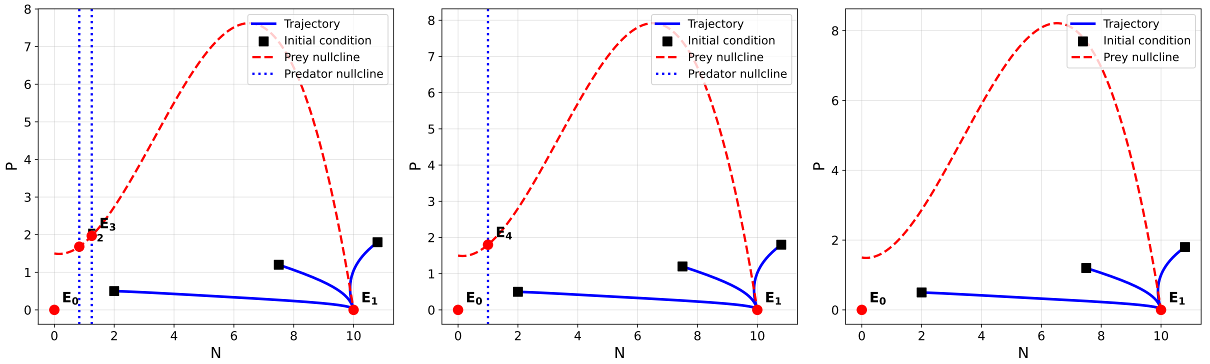
Therefore, when $\mu = \mu_c = 0.5$, the two interior equilibrium points merge into a single interior equilibrium point

$$E_4 = (N_4, P_4) = (1.0, 1.8).$$

To illustrate this transition numerically, three nearby values of μ are considered, namely $\mu = 0.48$, $\mu = 0.50$, and $\mu = 0.52$. For $\mu = 0.48$, one has $\Delta > 0$, so two interior equilibrium points exist. For $\mu = 0.50$, one obtains $\Delta = 0$, so the two interior equilibrium points coalesce into the single equilibrium point E_4. For $\mu = 0.52$, one has $\Delta < 0$, and therefore no interior equilibrium point exists. The corresponding phase portraits are shown in Figure 3.

Figure 3(a) shows that when $\mu = 0.48$, the system admits two interior equilibrium points, labeled E_2 and E_3, indicating that the coexistence structure is still present. In Figure 3(b), when $\mu = 0.50$, the two interior equilibrium points merge into a unique interior equilibrium point $E_4 = (1.0, 1.8)$, which corresponds to the critical case $\Delta = 0$. This numerically illustrates the existence of the nonhyperbolic equilibrium point obtained in Theorem 3.6. In Figure 3(c), when $\mu = 0.52$, no interior equilibrium point is observed, confirming that the coexistence equilibrium disappears once the parameter passes the critical threshold.

Ecologically, this result indicates the presence of a threshold in the effectiveness of prey group defense. When the defense intensity is below the critical level, predator and prey may still



(a) Two interior equilibria ($\mu = 0.48$, $\Delta = 0.0100 > 0$)

(b) Unique nonhyperbolic equilibrium ($\mu = 0.50$, $\Delta = 0.0000$)

(c) No interior equilibrium ($\mu = 0.52$, $\Delta = -0.0100 < 0$)

Figure 3. Phase portraits showing the existence of the nonhyperbolic equilibrium point E_4 for three nearby values of μ with $A = 1.0$: (a) $\mu = 0.48$ with two interior equilibria, (b) $\mu = 0.50$ with the unique nonhyperbolic equilibrium $E_4 = (1.0, 1.8)$, and (c) $\mu = 0.52$ with no interior equilibrium.

admit coexistence states. At the critical value, the coexistence structure collapses into a single threshold equilibrium, while beyond that value the interior coexistence equilibrium disappears. Thus, the numerical simulation supports the analytical result by showing that the nonhyperbolic equilibrium E_4 appears exactly at the transition between the existence and nonexistence of interior equilibrium points.

4. Conclusion

This study constructs and analyzes a predator-prey model that integrates three main biological mechanisms: the availability of alternative food for predators, prey group defense, and ecological cost represented as additional predator mortality. The resulting model is characterized by a nonlinear interaction structure in which prey group defense reduces predation efficiency through the term μN^2, alternative food contributes to predator growth through the term mA, and ecological cost increases predator mortality through the term $\theta m A$. Thus, the model captures not only the beneficial role of alternative food as an external energy subsidy, but also its adverse effect when accompanied by additional mortality pressure. Analytically, it is shown that the system possesses well-defined solutions that remain within the biologically relevant region and admits several types of equilibrium points, namely, the trivial equilibrium, the predator-free equilibrium, and the interior equilibrium points. The local stability analysis shows that whether the predator-free equilibrium point is stable depends on how the factors affecting predation efficiency, prey defense, and predator mortality interact. Changes in key parameter values may shift the system dynamics from predator extinction to a stable interior. Numerical simulations confirm these findings and demonstrate that the effectiveness of prey group defense acts as a primary controlling factor. When prey defense is sufficiently strong, the predator fails to persist; conversely, when prey defense weakens, the system tends toward a stable interior equilibrium.

The simulations also show that the effect of alternative food on predator persistence is nonlinear. At moderate levels, alternative food can sustain predator survival and promote coexistence. However, when alternative food is excessively abundant and accompanied by high ecological costs, the increase in predator mortality may outweigh the energetic benefits, leading to predator extinction. This result highlights that external energy subsidies in ecosystems are not always beneficial and may generate complex dynamical consequences. Overall, the study demonstrates that the interaction among prey group defense, alternative food availability, and predator ecological cost jointly determines the long-term dynamical structure of the system.

The novelty of this research lies in the integration of these three mechanisms into a single predator-prey framework, whereas previous studies have generally treated prey group defense and alternative food separately. In addition, this study introduces ecological cost as an explicit mortality consequence of alternative food use, thereby revealing that supplementary food does not necessarily strengthen predator persistence. Another novelty is the identification of a critical transition structure involving predator extinction, stable coexistence, and the emergence of a nonhyperbolic equilibrium point at the threshold between the existence and nonexistence of interior equilibria. These findings provide a broader ecological interpretation of how external food subsidy, prey protection, and mortality trade-offs jointly shape ecosystem sustainability.

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