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The effect of refugia on prey-predator model with parasite infection

Fitri Nur Azizah*
Indonesia Defense University, INDONESIA

Annisa Risda Alivia
Indonesia Defense University, INDONESIA

Habban Riawan Syahrul Haz
Indonesia Defense University, INDONESIA

Ubaidullah Yashkun
Sukkur IBA University, PAKISTAN

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Abstract

This study examines the impact of prey-hiding behavior on predator-prey dynamics within theoretical and mathematical ecology. We conduct both analytical and numerical analyses to explore the effects of protection on these dynamics. Our model divides the prey population into two subclasses: susceptible prey (S(t)) and infected prey (I(t)), changing over time. Utilizing a Holling's model type-1, we find that the trivial equilibrium of the model is unstable, while both the disease-free and disease equilibrium points are stable. These simulations provide insights into the characteristics of animals in natural environments.

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INTRODUCTION

Ecosystems are structures and functional units where living things interact in a balanced way (Mukhopadhyay & Bhattacharyya, 2012). Prey and predator activities are a form of interaction in an ecosystem. Humans, as part of the ecosystem, have a very high need for the formation of stability in the ecosystem (Haque et al, 2014). A good ecosystem will provide great benefits to life on Earth. Parasitic infections in populations in natural ecosystems often occur (Cortés García, 2023; Xiang, Huang, & Wang, 2023). Parasitic infections in ecosystem chains can destroy the food supply chain. Parasitic infections in preyed populations make the population decline in addition to the natural decline due to predators (Han, Guin, & Dai, 2021). This can directly impact the deterioration of predator populations due to a lack of food supply. Ecosystem imbalance can negatively impact human life, including public health, by reducing quality of life and increasing disease risk through the spread of zoonotic diseases that can be transmitted to humans.

In the mathematical modeling of predator-prey interactions, susceptible prey (S) and infected prey (I) are often used to represent the different states of the prey population. The susceptible prey are those that are healthy and not infected, while the infected prey are those that have contracted the parasite and may exhibit reduced fitness or increased vulnerability to predation. This compartmentalization allows researchers to study how the spread of infection affects the dynamics

***Corresponding Author:**
Fitri Nur Azizah, Indonesia Defense University, Indonesia, Email: nurzfitri182@gmail.com

of the prey population and its interactions with predators. For instance, studies have shown that infected prey may be more susceptible to predation due to their weakened condition, leading to a decline in the infected prey population and potentially affecting the predator population as well (Mekonen et al., 2024).

Predation can drastically reduce prey populations if not balanced by other factors, such as natural births and prey protection. Prey protection can determine population stability in the natural environment (Pal, Bhattacharyya, & Mondal, 2022). Several studies have examined the behavior and interactions resulting from parasitic infection of a population in ecosystems. Parasitic infections show that prey becomes very vulnerable to death and predation that occurs naturally (Cortés García, 2023; Lu, Li, & Liu, 2016). The case of water snails and fish shows that predators only prey on prey that are not infected, while infected prey will experience natural death and cannot be cured (Antwi-Fordjour, Parshad, & Beauregard, 2020). In an ecosystem, the growth of prey and predators is not always constant (Han, Guin, & Dai, 2021; Pal, Bhattacharyya, & Mondal, 2022). Catastrophic and environmental factors such as parasite infection are taken into consideration. In actual natural conditions, uninfected and infected prey will seek protection against predators (Molla et al., 2022). Research conducted by (Diz-Pita & Otero-Espinar, 2021) shows how several factors such as Allee effects, fear effects, cannibalism and immigration affect the predator-predator model, taking into account local equilibrium and global stability as well as the existence of limit cycles.

This study combines two conditions based on previous research: infection and the protection effect. Prey protection focuses on conditions intrinsic to the habitat, such as environmental factors. This paper presents an analytical and numerical study of the effects of protection on prey-predator dynamics. In real-world situations, the number of prey shelters depends on some factors; for example, the ability of prey to avoid predator attacks, predator efficiency in searching for prey, and the degree of prey disability caused by infection are some of them. Therefore, we have extended the basic model to include randomness in the protection measure. This article is divided into four sections. The second section will discuss the research methods, including research limitations and the model used in the research. The next section will discuss the research results, ranging from equilibrium to stability and model behavior. The last section will present the conclusion of this research.

METHOD

This chapter outlines the limitations and models used in the research. Understanding these limitations is crucial to ensure that the developed model remains relevant and realistic in representing natural systems. Subsequently, the model subsection will explain the mathematical approach applied to simulate interactions between species and the role of refugia in population dynamics.

Limitation

The limitations that we build for the predator-prey model with prey infection and protection are as follows;

1. If there is no epidemic effect, the prey growth model is a logistic model with an increased rate of and a capacity limitation in the environment.
2. For the epidemiological assumption, the prey population is divided into subclasses, namely susceptible prey ($S(t)$) and infected prey ($I(t)$), which will change according to time.
3. Only unsuspecting prey can reproduce, assuming that births are always positive with a birth rate.
4. There is competition among S because of the capacity limitation in the environment.
5. Disease in prey can only be transmitted by I to S by direct contact at a constant rate and cannot be inherited.
6. I cannot be cured or have immune immunity.
7. S decreases due to the natural death rate d_1, while I decreases due to the natural death rate and infection mortality rate d_1+a.
8. S and I have a natural self-protection system from predators 1, 2.
9. Predators cannot be infected by infected prey; the increase in predators depends on prey availability.

10. Predation occurs between predators with S and I affected by S and I.
11. Decreased predators are affected by the natural death rate of predators d_2.
12. The model used is a Holling's model type-1.

Model

From the assumptions we will include the potential for epidemics by including susceptible prey (S) and Infected prey (I) compartments in the predator-prey model. Where is the interaction between prey and predators in epidemic conditions is formulated in the following model;

$$\left.\begin{aligned} \frac{dS}{dt} &= rS\left(1-\frac{S+I}{k}\right) - d_1 S - \beta SI - \alpha P(S-\gamma_1) \\ \frac{dI}{dt} &= \beta SI - \mu P(I-\gamma_2) - (d_1+a) \\ \frac{dP}{dt} &= \alpha_1 P(S-\gamma_1) + \mu_1 P(I-\gamma_2) - d_2 P \end{aligned}\right\} \quad (1)$$

From the first equation regarding the change in the number of S concerning time is affected by the increase in the number of S due to the birth rate of S, it is assumed that only S can reproduce. The birth rates of S are reduced by the birth rate of S interacting with I limited by birth control. The number of S will decrease due to the natural death rate of prey (d_1), I due to transmission from I with infection rate (β), and predation between predators (P) and unprotected S with predation rate (α).

Changes in the number of I with time are affected by the increase in I from S to I with the β, reduced number of unprotected I due to being preyed on by predators with a predation rate (μ), and a reduced number of I due to natural deaths and deaths due to infection with a mortality rate ($d_1 + a$).

Changes in the number of P with time are affected by the increase in the number of P as a result of preying on S and I prey that does not protect by the coefficient of predation conversion (α_1) and (μ_1) and reduced by natural death with the mortality rate (d_2). The parameters in this case study are based on assumptions and previous research (Mukhopadhyay & Bhattacharyya, 2012; Pal, Bhattacharyya, Mondal, 2022; Gray et al, 2011) as shown in Table 1.

Tabel 1. Table of Model's Parameters

Parameters	Simbols	Value	Units
Susceptible	S	Estimated	N
Infected	I	Estimated	N
Predator	P	Estimated	N
Susceptible Prey's Birth Rate	r	1.2	time^{-1}
Carrying Capacity of Prey	k	8	N
Prey's Infection Rate	β	1.2	time^{-1}
Natural Prey's Death rate	d_1	0.3	time^{-1}
Infection Death rate	a	0.2	time^{-1}
Predation Rate of Susceptible Prey	α	0.25	time^{-1}
Predation Rate of Infected Prey	μ	0.4	time^{-1}
Natural Predator's Death Rate	d_2	0.16	time^{-1}
Susceptible Prey with Refugia	γ_1	0.12; 0.4	N
Infected Prey with Refugia	γ_2	0.1; 0.42	N
Coefficient Convertion of Predation of susceptible prey	α_1	0.2	dimensionless
Coefficient Convertion of Predation of Infected prey	μ_1	0.2	dimentionless

RESULTS AND DISCUSSION

Equilibrium Points

Equilibrium occurs when $dS/dt=dI/dt=dP/dt= 0$, which results in the equilibrium as follows:

$$E = \left\{(0,0,0), \left(\frac{k(-d_1+r)}{r}0,0\right), \left(\frac{d_1+a}{\beta}, -\frac{-\beta kr+\beta kd_1+ar+rd_1}{\beta(\beta k+r)}, 0\right), E_4\right\}$$

$E_1(0,0,0)$ is a trivial equilibrium, $E_2\left(\frac{k(-d_1+r)}{r}0,0\right)$ is a disease-free equilibrium, $E_3 = \left(\frac{d_1+a}{\beta}, -\frac{-\beta kr+\beta kd_1+ar+rd_1}{\beta(\beta k+r)}, 0\right)$ is an epidemic equilibrium, and E_4 is a complex equilibrium epidemic so that the authors do not include it in the analysis, this is a limitation of this study.

Stability Analysis

For the stability, we can analyze that by getting the eigenvalue from the Jacobian matrices (Ma & Wang, 2012; Yıldız, Bilazeroğlu, & Merdan, 2023; Rihan, Alsakaji, & Rajivganthi, 2020). Since the model in this study is shown in Equation 2, the Jacobian matrix in this article can provide information about the non-linear transformation of the model. Each term in the following Jacobian matrix represents the partial derivative of one compartment with respect to each compartment. The Jacobian matrices from the equation (1) is given by;

$$J = \begin{bmatrix} r\left(1-\frac{S+I}{k}\right)-\frac{rS}{k}-d_1-\beta I-\alpha P & -\frac{rS}{k}-S\beta & -\alpha(S-\gamma_1) \\ \beta I & -\mu P+S\beta-a-d_1 & -\mu(I-\gamma_2) \\ \alpha_1 P & \mu_1 P & \alpha_1(S-\gamma_1)+\mu_1(I-\gamma_2) \end{bmatrix} \quad (2)$$

Trivial equilibrium happen when, $E_1 = (0,0,0)$. The stability of the trivial equilibrium we get from substituting E_1 to J.

$$J_{E_1} = \begin{bmatrix} -d_1+r & 0 & \alpha\gamma_1 \\ 0 & -\alpha-d_1 & \mu\gamma_2 \\ 0 & 0 & -\alpha_1\gamma_1-\mu_1\gamma_2 \end{bmatrix} \quad (3)$$

Generate eigenvalues;

$$\lambda_1 = -\alpha_1\gamma_1-\mu_1\gamma_2$$
$$\lambda_2 = -d_1+r$$
$$\lambda_3 = -a-d_1$$

Since $\alpha_1, \gamma_1, \mu_1, \gamma_2 > 0$, then $\lambda_1 < 0$ because $a, d_1 > 0$; then $\lambda_3 < 0$. The model will be stable around E_1, if $\lambda_2 < 0$; $d_1 > r$; but if $d_1 < r$, then the model is unstable at the E_1 approximation.

The disease-free equilibrium is defined as the point which haven't disease in the present population (Saha & Samanta, 2021). The disease-free equilibrium (DFE) is said to be stable if, after a small disturbance (e.g., the introduction of a few infected individuals), the population returns to the disease-free state. This can be achieved if the eigenvalues of the Jacobian matrix are negative. In this model, it is happened when $E_2 = \left(\frac{k(-d_1+r)}{r}0,0\right)$. The stability of the Disease-free equilibrium we get from substituting E_2 to J.

$$J_{E_2} = \begin{bmatrix} r\left(1-\frac{-d+r}{r}\right)-r & d_1 - r - \frac{k(-d_1+r)\beta}{r} & -\alpha\left(\frac{k(-d_1+r)}{r}-\gamma_1\right) \\ 0 & \frac{k(-d_1+r)\beta}{r} - a - d_1 & \mu\gamma_2 \\ 0 & 0 & \alpha_1\left(\frac{k(-d_1+r)}{r}-\gamma_1\right)-\mu_1\gamma_2 \end{bmatrix} \quad (4)$$

Generate eigenvalues;

$$\lambda_1 = d_1 - r$$
$$\lambda_2 = \frac{\big((k-\gamma_1)\alpha_1 - \mu_1\gamma_2\big)r - k\alpha_1 d_1}{r}$$
$$\lambda_3 = \frac{(\beta k - a - d_1)r - \beta k d_1}{r}$$

Since $d_1 < r$, then $\lambda_1 < 0$, the model will be stable if $\lambda_2, \lambda_3 < 0$ with, $k\alpha_1(r-d_1) < \gamma_1\alpha_1 r + \gamma_2\mu_1 r$ and $\beta k(r-d_1) < ar + d_1 r$.

The epidemic equilibrium is defined as the point which have disease in the present population, in this model its happened when $E_3 = \left(\frac{d_1+a}{\beta}, -\frac{-\beta kr+\beta k d_1+ar+rd_1}{\beta(\beta k+r)}, 0\right)$. The stability of the Epidemic equilibrium we get from substituting E_3 to J.

$$J_{E_3} = \begin{bmatrix} A_{11} & A_{12} & A_{13} \\ A_{21} & A_{22} & A_{23} \\ A_{31} & A_{32} & A_{33} \end{bmatrix} \quad (5)$$

The elements of the matrix are the follows:

$$A_{11} = \frac{r\left(1-\frac{d_1+a}{\beta}-\frac{-rk\beta+\beta k d_1+ra+rd_1}{\beta(\beta k+r)}\right)}{k} - \frac{r(d_1+a)}{\beta k} - d_1 + \frac{-rk\beta+\beta k d_1+ra+rd_1}{\beta k+r}$$
$$A_{12} = -\frac{r(d_1+a)}{\beta k}$$
$$A_{13} = -\alpha\left(\frac{d_1+a}{\beta}-\gamma_1\right)$$
$$A_{21} = -\frac{-rk\beta+\beta k d_1+ra+rd_1}{\beta k+r}$$
$$A_{22} = 0$$
$$A_{23} = -\mu\left(-\frac{-rk\beta+\beta k d_1+ra+rd_1}{\beta(\beta k+r)}-\gamma_2\right)$$
$$A_{31} = 0$$
$$A_{32} = 0$$
$$A_{33} = \alpha_1\left(\frac{d_1+a}{\beta}-\gamma_1\right) + \mu_1\left(-\frac{-rk\beta+\beta k d_1+ra+rd_1}{\beta(\beta k+r)}-\gamma_2\right)$$

Generate eigenvalues;

$$\lambda_1 = \frac{\sqrt{4}\sqrt{(d_1+a)\left(\left(\frac{a}{4}+\frac{d_1}{4}\right)r^2+k\beta(-\beta k+a+d_1)r+k^2\beta^2 d_1\right)}+(-a-d_1)r}{2k\beta}$$
$$\lambda_2 = \frac{-\sqrt{4}\sqrt{(d_1+a)\left(\left(\frac{a}{4}+\frac{d_1}{4}\right)r^2+k\beta(-\beta k+a+d_1)r+k^2\beta^2 d_1\right)}+(-a-d_1)r}{2k\beta} \quad (6)$$
$$\lambda_3 = \frac{\left(-k(\gamma_1\alpha_1+\mu_1\gamma_2)\beta^2+\left((k\mu_1-\gamma_1\alpha_1-\mu_1\gamma_2)r+k\big((d_1+a)\alpha_1-d_1\mu_1\big)\right)\beta-r(\mu_1-\alpha_1)(d_1+a)\right)}{\beta(\beta k+r)}$$

where $\lambda_1 = \lambda_2 = \lambda_3 = 0$, a is obtained at $a = \frac{\beta\alpha_1\gamma_1+\beta\gamma_2\mu_1-\alpha_1 d_1}{\alpha_1}$; k at $k = \frac{r(\gamma_1\alpha_1+\mu_1\gamma_1)}{\alpha_1(r-d_1)}$; and r at $r = r$, The matrix characteristic equation J_{E_3} is obtaned;

$$\lambda^3 + N_1\lambda^2 + N_2\lambda + N_3 = 0$$

with;

$$N_1 = -(J_{11} + J_{33})$$
$$N_2 = J_{11}J_{33} + J_{21}J_{12}$$
$$N_3 = J_{21}J_{12}J_{33}$$

To ensure stability on E_3 using Routh-Hurwitz (Verma & Misra, 2018), with terms $N_1 > 0, N_2 > 0, N_3 > 0$, and $N_1N_2 > N_3$. Thus, the equilibrium point E_3 is said to be stable if it satisfies the equations below:

$$N_1 = -(J_{11} + J_{33}) > 0$$
$$N_2 = J_{11}J_{33} + J_{21}J_{12} > 0$$
$$N_3 = J_{21}J_{12}J_{33} > 0$$
$$N_1N_2 = -(J_{11} + J_{33})(J_{11}J_{33} + J_{21}J_{12}) > J_{21}J_{12}J_{33} = N_3$$

Model Solution Behavior

Runge-Kutta 4th order method is used for getting the result and simulation. For the first simulation, let $r = 1.2$; $k = 8$; $\beta = 1.2$; $d_1 = 0.3$; $\mu = 0.4$; $a = 0.2$; $\alpha = 0.25$; $\alpha_1 = 0.2$; $\mu_1 = 0.2$; $d_3 = 0.16$. Based on parameter values, the equilibrium point E_1, E_2 and E_3 are unstable asymptotically. Predators are more likely to prey on infected prey compared to susceptible, as seen in the value of μ being greater than the value of α. In this simulation, γ_1 and γ_2 are respectively 0.1 and 0.12, so that only a few prey are protected. Thus, there are many predation interactions between predators and prey. This causes a decrease in the number of prey, especially infected prey and an increase in the number of predators. In addition, the decrease in the number of infected prey is also influenced by the death factor caused by infection, where this factor does not occur in susceptible prey so that the decrease in the number of infected prey is more significant as shown in Figure 1.

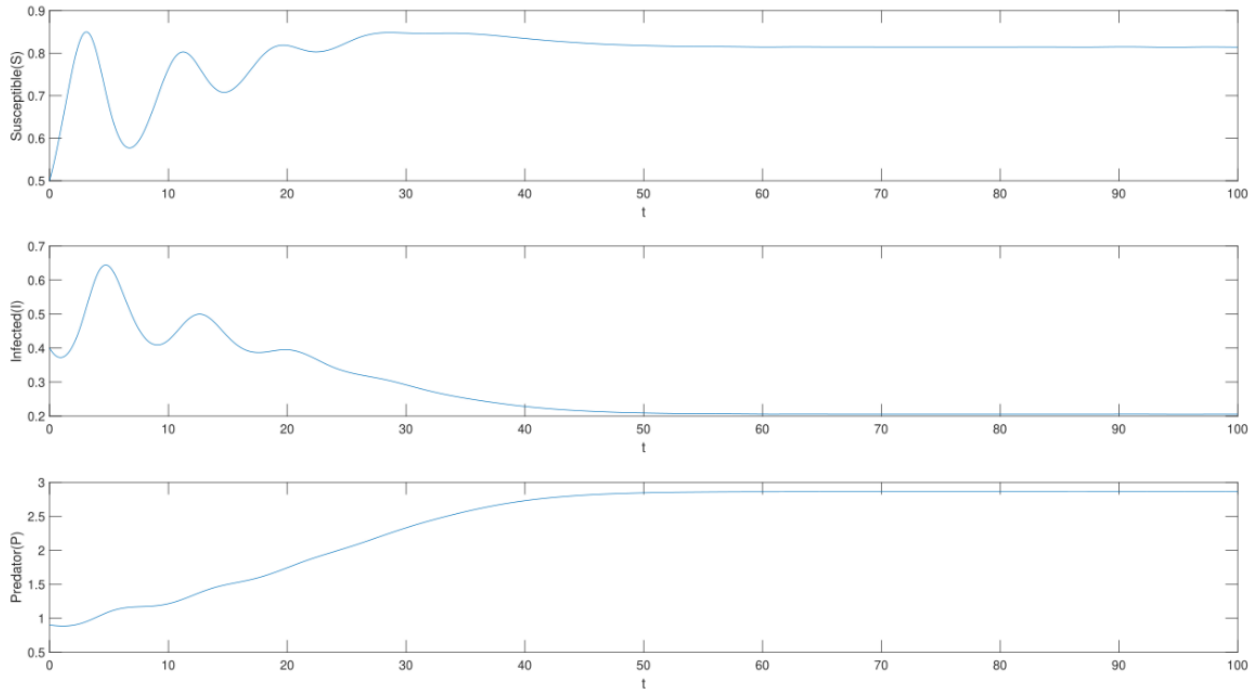

Figure 1. Figure of numerical result of first simulation.

The equilibrium point E_1 and E_2 are unstable and E_3 is asymptotically stable. In Figure 2, plotting result of some numerical solutions using several initial values. In this simulation, γ_1 and γ_2

are respectively 0.42 and 0.4, so that the protected prey is greater in number compared to the previous simulation. Thus, the predation interaction between predators and prey is also reduced. This causes a decrease in the number of predators due to the reduced number of prey that can be preyed on or not protected. In addition, it is also seen that susceptible prey and infected prey tend to be stable.

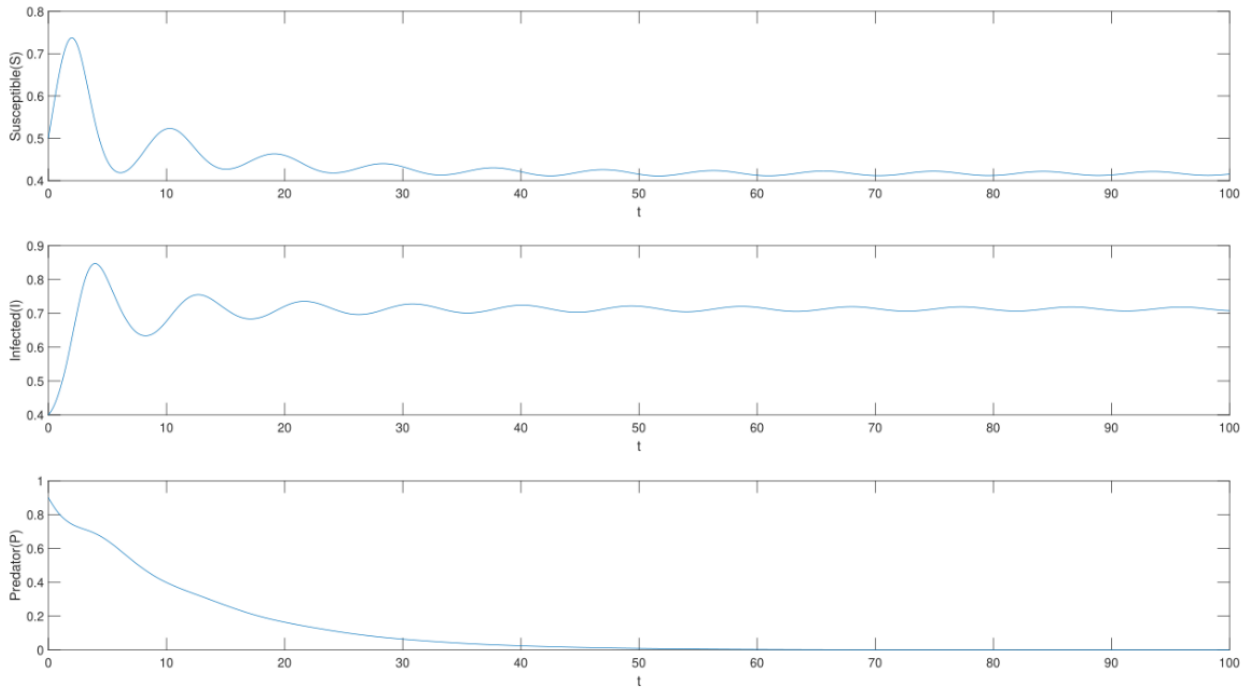

Figure 2. Figure of numerical result of second simulation.

CONCLUSION

From the results of numerical analysis and simulation, it can be concluded that the role of refugia in this predatory prey model is significant. Refugia can minimize the number of predators with two following cases:

1. If the total number of prey and the number of refugee prey are the same so the change in the number of preys only is caused by the birth rate of prey, the natural death of prey, and prey infected by infected prey for susceptible prey. Furthermore, changes in the number of infected preys over time will only be caused by the number of infected prey and the natural mortality rate of prey.
2. If the number of prey remaining is all protected, the predator will get a decrease in population along with the natural death rate of the predator.
3. The numerical analysis revealed that the presence of refugia can significantly delay or even prevent the extinction of prey populations. This is because refugia act as a buffer against predator pressure, allowing prey populations to recover and persist even under challenging conditions. This finding highlights the crucial role of refugia in maintaining ecological resilience and ensuring the long-term sustainability of ecosystems.

The next research is expected to conduct additional simulations that explore variations in parameters like population density, birth rates, and natural mortality rates. This will provide a more comprehensive understanding of how refugia impact different scenarios within the predator-prey model. This approach can offer insights into the intricate dynamics influenced by refugia and shed light on the broader implications for ecosystem stability and species interactions. By delving deeper into these variables, the research aims to uncover the nuanced effects of refugia on predator-prey relationships, ultimately contributing to a more holistic perspective on ecological systems.

AUTHOR CONTRIBUTIONS

Each author of this article played an important role in the process of method conceptualization, simulation, and article writing.

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CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest.

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