

Predator-prey model with SI disease dynamics in predators, increased hunger risk for infected predators, and optimal control strategies

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Abstract. This study presents a novel predator-prey model that incorporates Susceptible-Infected (SI) disease dynamics within the predator population. In this model, infected predators face a higher risk of death due to hunger compared to disease-induced death. We introduce two control variables aimed at reducing disease transmission and mitigating the impact of hunger on infected predators.

The optimal control problem is formulated using a smoothed prey penalty cost function to ensure the prey population remains above a critical threshold, thereby reducing predator mortality. Existence results for the optimal control are established, and numerical simulations illustrate the effectiveness of the proposed strategies.

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1. Introduction

Predator-prey interactions have been a cornerstone of ecological modeling, with foundational models like the Lotka-Volterra equations [5] providing deep insights into the cyclical dynamics of these systems. These models have been instrumental in understanding the oscillatory nature of

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predator and prey populations in the absence of external factors. However, natural ecosystems are far more complex, often influenced by additional biological and environmental factors, such as disease, resource availability, and interspecies competition [4, 7, 10]. These factors can dramatically alter the outcomes predicted by classical models.

In nature, numerous examples highlight the interplay between predation, disease, and resource availability. For instance, the Isle Royale wolf-moose dynamic is a well-documented predator-prey system where disease has periodically reduced wolf populations, altering the predator-prey balance [9]. Another example is the Serengeti ecosystem, where the introduction of rinderpest in the 19th century led to massive declines in herbivore populations, which in turn affected predator dynamics [9].

Similarly, the rabbit population in Australia has been heavily influenced by the introduction of myxomatosis, a disease that decimated populations and led to a complex interplay with predators such as foxes and feral cats [3].

This study builds on these concepts by introducing a predator-prey model that incorporates Susceptible-Infected (SI) disease dynamics within the predator population [6]. In this model, infected predators are not only subject to disease-induced mortality but also face an increased risk of death due to hunger, as their ability to hunt and consume prey is diminished. This dual-threat to infected predators is a realistic representation of many natural ecosystems where disease and resource scarcity jointly influence predator survival.

The addition of optimal control strategies in this model is essential for managing disease transmission and reducing hunger-induced mortality. Control variables are introduced to represent intervention strategies that

could, for example, involve disease vaccination or supplemental feeding programs aimed at reducing hunger-induced mortality [2]. By optimizing these controls, the model provides insights into how best to manage predator-prey dynamics in ecosystems where disease plays a significant role.

This paper is structured as follows: Section 2 presents the mathematical formulation of the model. Section 3 defines the optimal control problem and provides existence results for the optimal control. Section 4 discusses numerical simulations to demonstrate the model's effectiveness. Section 5 concludes the paper by highlighting the implications of the findings for ecosystem management and conservation strategies.

2. Model formulation

We consider a predator-prey system with SI disease dynamics in the predator population. The infected predators are at a higher risk of death due to hunger compared to disease-induced death. Let

- $S(t)$ denote the number of susceptible predators at time t ,
- $I(t)$ represent the number of infected predators at time t ,
- $P(t)$ indicate the number of prey at time t .

The dynamics of the system are governed by the following differential equations:

2.1. Prey dynamics

$$\frac{dP(t)}{dt} = rP(t) \left(1 - \frac{P(t)}{K} \right) - \alpha P(t)N(t), \quad (1)$$

where $N(t) = S(t) + I(t)$ is the total predator population, r is the intrinsic growth rate of the prey, K is the carrying capacity of the environment for the prey and α represents the predation rate, which determines the

rate at which predators consume prey, thereby influencing both the prey population's decline and the energy intake for predators.

2.2. Susceptible predator dynamics

$$\frac{dS(t)}{dt} = c\alpha P(t)S(t) - \beta S(t)I(t) - \delta S(t) + u_1(t)S(t), \quad (2)$$

where c is the conversion efficiency of prey into predator offspring, β is the transmission rate of the disease, δ is the natural death rate of susceptible predators, and $u_1(t)$ is a control variable representing efforts to reduce disease transmission.

2.3. Infected predator dynamics

$$\frac{dI(t)}{dt} = \beta S(t)I(t) - \delta_I I(t) - \mu_I \alpha P(t)I(t) + u_2(t)I(t), \quad (3)$$

where δ_I is the disease-induced death rate of infected predators, μ_I represents the increased mortality of infected predators due to hunger (or decreased predation efficiency), and $u_2(t)$ is a control variable aimed at reducing the hunger-induced mortality of infected predators.

3. Optimal control problem

We now formulate the optimal control problem with the goal of identifying the control strategies $u_1(t)$ and $u_2(t)$ that minimize the total cost associated with managing the disease and hunger-induced mortality in the predator population.

3.1. Cost function

The cost function $J(u_1, u_2)$ captures the trade-offs between control efforts and the ecological impacts of population reductions. We propose the following cost function:

$$J(u_1, u_2) = \int_0^T \left[A_1 S(t) + A_2 I(t) + \frac{A_3}{1 + P(t)} + B_1 u_1^2(t) + B_2 u_2^2(t) \right] dt, \quad (4)$$

where

- A_1, A_2, A_3 represent the weights associated with the costs of susceptible predators, infected predators, and a smoothed prey penalty term,
- B_1, B_2 are the costs associated with implementing the control variables $u_1(t)$ and $u_2(t)$.

3.2. Existence of optimal control

We establish the existence of an optimal control pair (u_1^*, u_2^*) using standard results from optimal control theory.

Theorem 1. *Consider the optimal control problem defined by the state equations (1)-(3) and cost functional (4), subject to the control constraints. If the state variables $S(t)$, $I(t)$, and $P(t)$ are bounded, and the coefficients A_1, A_2, A_3, B_1, B_2 are positive, then there exists an optimal control pair (u_1^*, u_2^*) that minimizes the cost functional $J(u_1, u_2)$.*

Proof. The admissible control set $\mathcal{U} = \{(u_1(t), u_2(t)) \mid 0 \leq u_1(t), u_2(t) \leq 1, \forall t \in [0, T]\}$ is non-empty, convex, and closed in L^2 space. The state equations, while nonlinear, are Lipschitz continuous, ensuring the existence and uniqueness of solutions $S(t)$, $I(t)$, and $P(t)$ under given initial conditions. The cost functional $J(u_1, u_2)$ is continuous in the control variables $u_1(t)$ and $u_2(t)$, with the logarithmic terms being well-defined for positive state variables and parameters.

Given that the control set $\mathcal{U}$ is compact in the weak topology, the direct method in the calculus of variations ensures that a minimum of the cost functional exists. The combined properties of the control set and the cost functional ensure that an optimal control pair (u_1^*, u_2^*) exists, satisfying the necessary conditions for minimization [1]. $\square$

3.3. Characterization of optimal control

We now characterize the optimal control (u_1^*, u_2^*) using Pontryagin's Maximum Principle. The Hamiltonian $\mathcal{H}$ associated with this optimal control problem is defined as:

$$\begin{aligned} \mathcal{H} = & A_1 S(t) + A_2 I(t) + \frac{A_3}{1 + P(t)} + B_1 u_1^2(t) + B_2 u_2^2(t) \\ & + \lambda_1(t) [c\alpha P(t)S(t) - \beta S(t)I(t) - \delta S(t) + u_1(t)S(t)] \\ & + \lambda_2(t) [\beta S(t)I(t) - \delta_I I(t) - \mu_I \alpha P(t)I(t) + u_2(t)I(t)] \\ & + \lambda_3(t) \left[rP(t) \left(1 - \frac{P(t)}{K} \right) - \alpha P(t)(S(t) + I(t)) \right], \end{aligned} \quad (5)$$

where $\lambda_1(t)$, $\lambda_2(t)$, and $\lambda_3(t)$ are the adjoint variables associated with the state dynamics of susceptible predators, infected predators, and prey, respectively.

According to Pontryagin's Maximum Principle, the necessary conditions for optimality are given by:

$$\frac{\partial \mathcal{H}}{\partial u_1} = 2B_1 u_1(t) + \lambda_1(t)S(t) = 0, \quad (6)$$

$$\frac{\partial \mathcal{H}}{\partial u_2} = 2B_2 u_2(t) + \lambda_2(t)I(t) = 0, \quad (7)$$

which lead to the characterization of the optimal controls $u_1^*(t)$ and $u_2^*(t)$:

$$u_1^*(t) = -\frac{\lambda_1(t)S(t)}{2B_1}, \quad (8)$$

$$u_2^*(t) = -\frac{\lambda_2(t)I(t)}{2B_2}. \quad (9)$$

These control expressions are subject to the constraints $0 \leq u_1(t) \leq 1$ and $0 \leq u_2(t) \leq 1$, meaning that $u_1^*(t)$ and $u_2^*(t)$ should be projected onto the interval $[0, 1]$.

The adjoint equations for $\lambda_1(t)$, $\lambda_2(t)$, and $\lambda_3(t)$ are derived from:

$$\frac{d\lambda_1(t)}{dt} = -\frac{\partial \mathcal{H}}{\partial S}, \quad (10)$$

$$\frac{d\lambda_2(t)}{dt} = -\frac{\partial \mathcal{H}}{\partial I}, \quad (11)$$

$$\frac{d\lambda_3(t)}{dt} = -\frac{\partial \mathcal{H}}{\partial P}, \quad (12)$$

with terminal conditions:

$$\lambda_1(T) = 0, \quad \lambda_2(T) = 0, \quad \lambda_3(T) = 0. \quad (13)$$

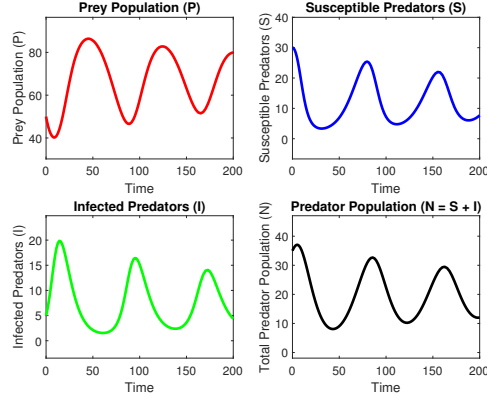
Thus, the optimal control strategy is determined by solving the state equations (1)-(3) simultaneously with the adjoint equations under the optimal control laws (10) and (11).

4. Numerical simulations

To illustrate the effectiveness of the proposed control strategies, numerical simulations are conducted using the predator-prey model with SI disease dynamics. These simulations demonstrate how the optimal control strategies influence the time evolution of the susceptible predator, infected predator, and prey populations.

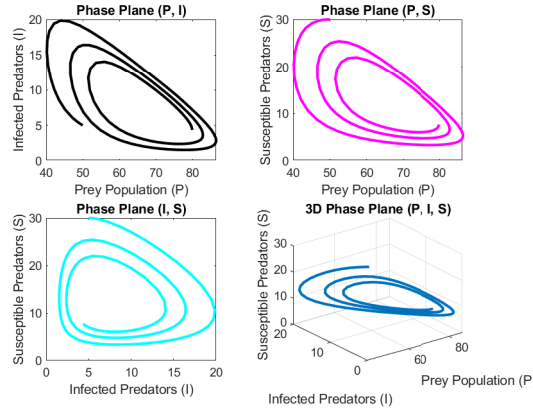
4.1. Time series analysis

Figure 1 shows the time series of the susceptible predator $S(t)$, infected predator $I(t)$, total predator $N(t) = S(t) + I(t)$ and prey $P(t)$ populations under optimal control. The introduction of optimal controls $u_1^*(t)$ and $u_2^*(t)$ significantly affects the system's dynamics.

**Figure 1: Ecosystem Dynamics**

4.2. Phase plane analysis

Phase plane plots (Figure 2) offer deeper insights into the interrelationships between the populations. They demonstrate that the system preserves the typical dynamic features of traditional predator-prey models without disease, suggesting effective management of both disease transmission and hunger-induced mortality.

**Figure 2: Phase Plots**

5. Discussion of optimal control strategies

The analysis of the optimal control strategies reveals their effectiveness in stabilizing predator and prey populations, while simultaneously managing disease spread and reducing hunger-induced mortality among infected predators. By ensuring that prey populations remain above a critical threshold, the model supports predator health and sustains ecosystem stability.

The control variables $u_1(t)$ and $u_2(t)$ enable adaptive interventions that respond to varying ecological conditions. For instance, when disease transmission intensifies, an increase in $u_1(t)$ can curb the spread among predators. Conversely, during periods of prey scarcity, adjusting $u_2(t)$ mitigates the heightened hunger risk for infected predators, preventing a sharp increase in mortality due to insufficient food.

A key feature of the model is the inclusion of a smoothed prey penalty within the cost function, which plays a vital role in balancing predator needs with prey availability. By discouraging scenarios where prey numbers fall too low, the model promotes sustainable predator-prey dynamics, crucial for preventing overexploitation and subsequent prey population collapse.

These optimal control strategies have practical applications in wildlife management, particularly in areas where disease and resource limitations pose significant challenges. In conservation efforts, especially where predator species face endangerment, these strategies could help maintain the delicate balance between predator survival and prey conservation, securing the long-term viability of both populations.

Furthermore, the flexibility of this framework allows it to be adapted to a variety of ecological contexts, including those involving different species and diseases.

Future research could expand on these strategies to address multi-species ecosystems, offering deeper insights into comprehensive ecosystem management.

6. Conclusion

This study developed an optimal control framework for a predator-prey system incorporating SI disease dynamics among predators. The model addresses increased mortality in infected predators due to hunger and introduces control variables to manage disease transmission and hunger-induced deaths.

The existence of an optimal control solution is established, and numerical simulations confirm the model's efficacy. The integration of disease dynamics into the predator-prey framework provides a more realistic representation of natural ecosystems, where disease and resource availability interact to shape population dynamics.

The proposed control strategies facilitate targeted interventions that effectively manage these dynamics, ensuring the stability and sustainability of predator and prey populations alike.

From a conservation perspective, these findings offer valuable insights. In regions where predator species are at risk and where disease and food scarcity are pressing concerns, the strategies proposed here could inform management policies that balance predator protection with prey conservation.

For instance, vaccination programs could reduce disease spread, while optimized supplemental feeding could mitigate hunger-induced mortality. The model's inclusion of a smoothed prey penalty highlights the importance of maintaining prey populations above critical levels, thereby preventing overexploitation and ensuring ecosystem sustainability.

The versatility of this framework also presents opportunities for application across various ecological scenarios. While this study focused on a single predator-prey system with SI disease dynamics, the model could be adapted to incorporate multiple species, different diseases, or varying environmental conditions. Such extensions could yield even greater insights into the intricate interactions that govern ecosystems. Beyond wildlife management, the model's relevance extends to other fields, such as agricultural pest control, where predator-prey-like dynamics exist between pests and crops. Adapting this framework could guide the development of strategies to manage pest populations effectively, minimizing their impact on crop yields.

Lastly, ethical considerations are paramount when applying these control strategies in real-world ecosystems. While the model offers a theoretical basis for optimizing predator-prey interactions, its practical implementation must consider broader ecological and ethical implications.

Collaboration between ecologists, wildlife managers, and ethicists will be crucial to ensuring that these strategies are scientifically sound and ethically responsible.

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