



Stability Analysis of a Predator-Prey Model with Disease in Prey, Predator Harvesting, and Internal Migration of Susceptible Prey in a Closed Ecosystem

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Abstract

This study develops and analyzes a mathematical model of a predator-prey system incorporating three simultaneous ecological factors: disease in the prey population, harvesting of the predator, and internal migration of healthy prey within a closed ecosystem. The prey population is divided into two compartments healthy prey and infected prey where disease transmission follows the law of mass action. The predator population is subject to a constant harvesting rate representing external pressure such as hunting or capture. Internal migration of healthy prey is modeled as a density-dependent flux governed by a migration parameter m , which drives the redistribution of healthy prey across spatial patches without allowing permanent emigration from the system. The model is formulated as a system of three autonomous ordinary differential equations. Six equilibrium points are identified, representing a range of ecological scenarios from total extinction to full coexistence of all populations. The local stability of each equilibrium point is analyzed by linearization via the Jacobian matrix, and stability conditions are derived in terms of the model parameters, including the application of the Routh–Hurwitz criterion for the coexistence equilibrium. A key finding is that the migration parameter m does not alter the location of equilibrium points but directly influences the eigenvalues of the Jacobian, thereby affecting the rate at which the system recovers from perturbations. Numerical simulations are conducted to verify and illustrate the analytical results. This work extends the model of Mansur et al. [1] by introducing an internal spatial dimension, offering a more ecologically realistic framework for understanding population dynamics in ecosystems where disease, harvesting, and migration co-occur.

Keywords: predator-prey model; eco-epidemiology; disease in prey; predator harvesting; internal migration; equilibrium stability; Routh–Hurwitz criterion; mathematical ecology

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1. Introduction

Mathematical modeling is one of the branches of applied mathematics that bridges real-world phenomena with abstract representations in the form of mathematical equations [2][3]. Through this approach, complex problems encountered in nature can be systematically simplified and analyzed, enabling predictions of a system's behavior over time. One field that extensively employs mathematical modeling is ecology, particularly in studying the interactions among organism populations within an ecosystem [4][5][6].

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In ecology, the food chain describes the flow of energy from one organism to another. The most fundamental component of this chain is the predator-prey relationship, in which the survival of a predator population is critically dependent on the availability of its prey. The predator-prey model was first introduced independently by Lotka [2] and Volterra [3], whose classical framework assumed that both populations are entirely healthy, so that neither the predator's ability to hunt nor the prey's capacity to escape is hindered by disease. In this model, the densities of prey and predator populations mutually influence each other in a dynamic oscillatory manner, constrained by environmental carrying capacity.

In reality, however, the assumption that the entire prey population is uniformly healthy does not always hold. In natural ecosystems, prey populations can be divided into two sub-groups: healthy individuals and those infected by disease. These two groups differ significantly in their ecological characteristics, as healthy prey possess superior escape ability and physical resilience compared to diseased prey, resulting in different predation rates for each group [7][8][9][10]. Mansur et al. [1] addressed this biological reality by developing a predator-prey model that incorporates disease within the prey population together with harvesting of the predator, yielding five equilibrium points that describe various possible ecosystem conditions ranging from total extinction to full coexistence of all populations. Further studies have extended eco-epidemiological frameworks by incorporating additional complexity such as prey refuge [9][11], cooperative hunting [6][8][12], fear effects [7][12][13], and harvesting strategies [14][15][16].

Beyond disease and harvesting, another ecologically significant factor often neglected in many models is population migration. Kant and Kumar [17] affirmed that the movement of populations across spatial regions is a universal demographic event occurring in nearly all types of organisms, including mammals, fish, birds, insects, and crustaceans. Dai et al. [18] further demonstrated that seasonal migration of prey between breeding and non-breeding regions can significantly affect population survival, even determining whether a species persists or goes extinct in a given ecosystem. Roy and Roy [15] likewise showed that incorporating migration and harvesting in a drainage-system predator-prey model produces richer dynamical behavior and more ecologically meaningful equilibria. These findings collectively reveal a persistent gap in the literature: most existing predator-prey models that combine disease and harvesting do not simultaneously account for internal prey migration within a closed ecosystem.

The present study addresses this gap by constructing a mathematical model of a predator-prey system that jointly incorporates disease in the prey, harvesting of the predator, and internal migration of healthy prey within a closed ecosystem. Specifically, this research aims to:

1. formulate the mathematical model with these three interacting components;
2. determine the equilibrium points of the model;
3. analyze the local stability of each equilibrium point;
4. verify the analytical results through numerical simulation.

The primary novelty of this work lies in the explicit integration of internal prey migration represented by a density-dependent migration term governed by parameter m into a predator-prey framework that already contains disease-structured prey and predator harvesting [1][17][18]. Unlike external migration models [15][16][19], the closed-ecosystem assumption ensures that the total population within the system is conserved, so that the migration term redistributes healthy prey across spatial patches without introducing permanent emigration. This spatial dimension directly influences the rate at which the system recovers from perturbations toward each equilibrium, an effect made analytically transparent through eigenvalue analysis and corroborated numerically. The model thus extends the work of Mansur et al. [1] by adding a spatial component that more faithfully reflects the ecological realities of many terrestrial and aquatic ecosystems [15][17][18].

The remainder of this paper is organized as follows. Section 2 presents the mathematical formulation of the model, including variable definitions, parameter descriptions, underlying

biological assumptions, and the complete analytical framework comprising equilibrium analysis and local stability conditions. Section 3 presents numerical simulations to verify the analytical results and discusses their ecological implications. Finally, Section 4 summarizes the main findings and outlines directions for future work.

2. Methods

This section presents the complete mathematical framework of the proposed model. The analysis follows a standard sequence that begins with model construction and ends with stability criteria, mirroring the approach adopted in related eco-epidemiological studies [1][17]. First, the model equations are derived from biological assumptions and the state variables and parameters are defined. Second, the positivity and boundedness of solutions are established to confirm biological validity. Third, the equilibrium points are determined by solving the algebraic system obtained by setting all time derivatives to zero. Fourth, the local stability of each equilibrium is examined through linearization, whereby the Jacobian matrix is evaluated at each equilibrium and either the eigenvalues are computed directly or the Routh–Hurwitz criterion is applied.

2.1. Model Formulation

The model developed in this study represents a closed predator-prey ecosystem structured into three population compartments whose densities evolve continuously over time. The three state variables are $x(t)$, the density of healthy (susceptible) prey at time t ; $y(t)$, the density of infected prey at time t ; and $p(t)$, the density of the predator population at time t . Healthy prey are individuals that have not been exposed to disease, are capable of natural growth, and interact fully with their environment including moving between spatial patches. Infected prey are individuals that have contracted disease through contact with other infected prey. This compartment has no recovery pathway, so the only way out is death, whether from disease or predation. The predator shares the same habitat as both prey groups, derives energy from both, but its population is subject to external harvesting pressure.

The model is constructed as an extension of the work of Mansur et al. [1], which formulated a predator-prey system with disease in the prey and constant harvesting of the predator. The present model retains that framework and introduces an additional term representing the internal migration of healthy prey within the closed ecosystem, motivated by the spatial dynamics studied by Kant and Kumar [17] and Dai et al. [18]. The disease transmission mechanism follows the law of mass action as adopted in [1][17], predation on healthy prey follows a linear (Holling Type I) functional response, and predation on infected prey likewise follows a linear rate, consistent with the formulation in [1].

All parameters in the model are assumed to be positive constants. Their biological interpretations are summarized in Table 1.

Table 1: Definition of model parameters

Symbol	Biological meaning
r	Intrinsic growth rate of healthy prey
K	Environmental carrying capacity of healthy prey
β	Disease transmission rate (contact rate between healthy and infected prey)
α	Predation rate of predator on healthy prey
ε	Predation rate of predator on infected prey
μ_1	Natural mortality rate of healthy prey
μ_2	Natural mortality rate of infected prey
μ_3	Natural mortality rate of predator
δ	Additional disease-induced mortality rate of infected prey
h	Harvesting rate of predator by external factors
a	Conversion coefficient from healthy prey to predator biomass
b	Conversion coefficient from infected prey to predator biomass
m	Internal migration rate of healthy prey across spatial patches
x_e	Reference density of healthy prey used as the migration equilibrium target

The model is built on the following biological assumptions, established to maintain ecological realism while preserving mathematical tractability. In the absence of disease and predator, healthy prey grow logistically with intrinsic growth rate r and carrying capacity K , reflecting the finite resource availability of the environment [1][17]. Disease transmission occurs exclusively within the prey population through direct contact between healthy and infected individuals, with intensity proportional to the product of their densities (law of mass action); the predator is not susceptible to the disease even when consuming infected prey [1][5]. Infected prey receive no treatment and cannot recover, so y can only decrease through natural death, disease-induced mortality, or predation [1]. The predator's consumption of both prey types follows a linear functional response, meaning the predation rate scales directly with prey density without saturation, an assumption that keeps the system analytically tractable and is consistent with the baseline formulation in [1]. Harvesting of the predator occurs at a constant proportional rate h , representing external pressure such as human hunting or trapping, following the approach of [1][15]. The ecosystem is strictly closed: no individual from any compartment permanently emigrates from or immigrates into the system. The only spatial dynamic is the internal redistribution of healthy prey between patches within the same ecosystem, modeled by a density-dependent flux term $m(x_e - x)$. When local density x falls below the reference level x_e an inflow occurs, and when x exceeds x_e an outflow occurs, so that the term continuously drives the local healthy prey density toward the reference distribution without changing the total number of individuals in the system. At equilibrium, it is assumed that $x = x_e$, so the net migration flux is zero. This means the migration term does not alter the location of any equilibrium point, but it does appear in the Jacobian matrix and therefore directly affects the eigenvalues governing the rate of convergence toward equilibrium [17][18].

Based on these variables, parameters, and assumptions, the dynamics of the three population compartments are described by the following system of nonlinear autonomous ordinary differential equations:

$$\frac{dx}{dt} = rx \left(1 - \frac{x}{K}\right) - \beta xy - \alpha xp - \mu_1 x + m(x_e - x), \quad (1)$$

$$\frac{dy}{dt} = \beta xy - \varepsilon yp - \mu_2 y - \delta y, \quad (2)$$

$$\frac{dp}{dt} = \alpha xp + b yp - \mu_3 p - hp. \quad (3)$$

The system in Eqs. (1)–(3) is subject to initial conditions $x(0) = x_0 > 0$, $y(0) = y_0 \geq 0$, and $p(0) = p_0 \geq 0$. Since all parameters are positive and all initial conditions are non-negative, the

solutions of the system remain non-negative for all $t \geq 0$.

The ecological interpretation of each term in the system is as follows. In Eq. (1), the term $rx(1 - x/K)$ represents logistic growth of healthy prey; βxy is the reduction in healthy prey due to disease transmission through contact with infected prey; αxp is predation of healthy prey by the predator; $\mu_1 x$ is natural mortality of healthy prey; and $m(x_e - x)$ is the internal migration flux that redistributes healthy prey across patches. In Eq. (2), βxy is the inflow of newly infected individuals; εyp is predation of infected prey; and $(\mu_2 + \delta)y$ is the total mortality of infected prey from natural death and disease-induced mortality combined. In Eq. (3), $axp + byp$ represents the growth of the predator population through consumption of healthy and infected prey respectively, with conversion coefficients a and b ; and $(\mu_3 + h)p$ is the total reduction in the predator population from natural mortality and harvesting.

2.2. Positivity and Boundedness of Solutions

The biological validity of any population model requires that its solutions remain non-negative and bounded for all time $t \geq 0$. Non-negativity ensures that no population density takes a negative value, which has no ecological meaning, while boundedness ensures that populations do not grow without limit, consistent with the finite resource constraints encoded in the logistic growth term and the carrying capacity K .

Since all parameters in system Eqs. (1)–(3) are positive and all initial conditions satisfy $x(0) > 0$, $y(0) \geq 0$, $p(0) \geq 0$, the right-hand sides of the equations are locally Lipschitz continuous, guaranteeing the existence and uniqueness of solutions on some maximal interval. On the boundary of the non-negative orthant, each equation satisfies the condition that the rate of change is non-negative whenever the corresponding state variable is zero (with the others non-negative), so solutions that start in the non-negative orthant cannot leave it. Therefore, all solutions of the system are non-negative for all $t \geq 0$.

Boundedness follows from the logistic structure of healthy prey growth. Because healthy prey are capped by carrying capacity K , the total prey population $x + y$ is bounded above. The predator population is in turn bounded because its growth depends entirely on consumption of bounded prey populations while being reduced by natural mortality and harvesting. A formal upper bound for the full system can be constructed by considering the auxiliary function $V = x + y + p$ and showing that $\dot{V} \leq C - \sigma V$ for positive constants C and σ , which implies that the solution ultimately enters and remains within a compact invariant region in $\mathbb{R}_+^3$ [1][17].

2.3. Equilibrium Points

The equilibrium points of system Eqs. (1)–(3) are obtained by setting $dx/dt = 0$, $dy/dt = 0$, and $dp/dt = 0$ simultaneously. Since at equilibrium $x = x_e$ by assumption, the migration term vanishes and does not affect the equilibrium coordinates. Solving the resulting algebraic system yields six equilibrium points, each representing a distinct ecological scenario ranging from total extinction to full coexistence of all populations.

Equilibrium E_1 (Total extinction). Setting $x = y = p = 0$ trivially satisfies the equilibrium conditions, giving

$$E_1 = (0, 0, 0). \quad (4)$$

This equilibrium represents a scenario in which all three populations have gone extinct.

Equilibrium E_2 (Healthy prey only, no infected prey, no predator). Setting $y = 0$ and $p = 0$ in the equilibrium equations gives $x(r(1 - x/K) - \mu_1) = 0$. The nontrivial solution $x \neq 0$ yields

$$E_2 = \left(K \left(1 - \frac{\mu_1}{r} \right), 0, 0 \right). \quad (5)$$

This equilibrium exists if and only if $r > \mu_1$.

Equilibrium E_3 (Healthy and infected prey, no predator). Setting $p = 0$ and $y \neq 0$, from $dy/dt = 0$ one obtains $x^* = (\mu_2 + \delta)/\beta$. Substituting into $dx/dt = 0$ then gives

$$y^* = \frac{r}{\beta} \left(1 - \frac{\mu_2 + \delta}{\beta K} \right) - \frac{\mu_1}{\beta}. \quad (6)$$

Hence

$$E_3 = \left(\frac{\mu_2 + \delta}{\beta}, y^*, 0 \right), \quad (7)$$

which exists if and only if $\beta K(r - \mu_1) > r(\mu_2 + \delta)$.

Equilibrium E_4 (Healthy prey and predator, no infected prey). Setting $y = 0$, from $dp/dt = 0$ one obtains $x^* = (\mu_3 + h)/a$. Substituting into $dx/dt = 0$ gives

$$p^* = \frac{1}{\alpha} \left[r \left(1 - \frac{\mu_3 + h}{aK} \right) - \mu_1 \right]. \quad (8)$$

Hence

$$E_4 = \left(\frac{\mu_3 + h}{a}, 0, p^* \right), \quad (9)$$

which exists if and only if $ar(aK - (\mu_3 + h)) > aK\mu_1(\mu_3 + h)/r$.

Equilibrium E_5 . Setting $x = 0$ in the system forces $y = 0$ (since $dy/dt = 0$ with $x = 0$ and $y > 0$ would require $p < 0$), so no biologically distinct equilibrium with $x = 0$ and $y > 0$ exists; this case collapses back to E_1 .

Equilibrium E_6 (Full coexistence). The interior equilibrium $E_6 = (x^*, y^*, p^*)$ with all components positive is obtained by solving the full algebraic system simultaneously. From $dp/dt = 0$: $ax^* + by^* = \mu_3 + h$. From $dy/dt = 0$: $p^* = (\beta x^* - \mu_2 - \delta)/\varepsilon$. Substituting into $dx/dt = 0$ yields a polynomial equation in x^* whose positive root (when it exists) determines the coexistence state:

$$E_6 = (x^*, y^*, p^*), \quad x^*, y^*, p^* > 0. \quad (10)$$

The existence of E_6 requires $(\mu_2 + \delta)/\beta < x^* < (\mu_3 + h)/a$. The numerical values of x^* , y^* , and p^* under the simulation parameters are given in Section 3.

2.4. Local Stability Analysis

The local stability of each equilibrium point is assessed by linearizing system Eqs. (1)–(3) about that point and analyzing the eigenvalues of the Jacobian matrix evaluated there. An equilibrium is locally asymptotically stable if and only if every eigenvalue of the Jacobian has a strictly negative real part [1][17]. The general Jacobian of system Eqs. (1)–(3) is

$$J(x, y, p) = \begin{bmatrix} r \left(1 - \frac{x}{K} \right) - \frac{rx}{K} - \beta y - \alpha p - \mu_1 - m & -\beta x & -\alpha x \\ \beta y & \beta x - \varepsilon p - \mu_2 - \delta & -\varepsilon y \\ ap & bp & ax + by - \mu_3 - h \end{bmatrix}. \quad (11)$$

The migration parameter m appears exclusively in the (1, 1) entry of J , shifting that diagonal element without changing any equilibrium coordinate. For boundary equilibria the eigenvalues are read directly from the triangular or block-triangular structure of J ; for the full coexistence equilibrium the Routh–Hurwitz criterion is applied to the resulting cubic characteristic polynomial.

Theorem 1. *The trivial equilibrium $E_1 = (0, 0, 0)$ is locally asymptotically stable if and only if $r < \mu_1 + m$.*

Proof. Evaluating (11) at $E_1 = (0, 0, 0)$ gives the diagonal matrix

$$J(E_1) = \begin{bmatrix} r - \mu_1 - m & 0 & 0 \\ 0 & -(\mu_2 + \delta) & 0 \\ 0 & 0 & -(\mu_3 + h) \end{bmatrix}.$$

The eigenvalues are read directly from the diagonal:

$$\lambda_1 = r - \mu_1 - m, \quad \lambda_2 = -(\mu_2 + \delta) < 0, \quad \lambda_3 = -(\mu_3 + h) < 0.$$

Since all parameters are positive, $\lambda_2 < 0$ and $\lambda_3 < 0$ unconditionally. Thus E_1 is locally asymptotically stable if and only if $\lambda_1 < 0$, i.e., $r < \mu_1 + m$. $\square$

Theorem 2. *The disease-free predator-free equilibrium $E_2 = (K(1 - \mu_1/r), 0, 0)$ exists whenever $r > \mu_1$ and is locally asymptotically stable if and only if*

$$\beta K \left(1 - \frac{\mu_1}{r}\right) < \mu_2 + \delta \quad \text{and} \quad aK \left(1 - \frac{\mu_1}{r}\right) < \mu_3 + h.$$

Proof. Denote $x_2^* = K(1 - \mu_1/r) > 0$. Evaluating (11) at $E_2 = (x_2^*, 0, 0)$ yields the upper-triangular matrix

$$J(E_2) = \begin{bmatrix} -(r - \mu_1) - m & -\beta x_2^* & -\alpha x_2^* \\ 0 & \beta x_2^* - (\mu_2 + \delta) & 0 \\ 0 & 0 & ax_2^* - (\mu_3 + h) \end{bmatrix}.$$

Since the matrix is upper triangular, its eigenvalues are the diagonal entries:

$$\lambda_1 = -(r - \mu_1) - m < 0, \quad \lambda_2 = \beta x_2^* - (\mu_2 + \delta), \quad \lambda_3 = ax_2^* - (\mu_3 + h).$$

The existence condition $r > \mu_1$ ensures $\lambda_1 < 0$; the migration parameter m makes λ_1 more negative without affecting λ_2 or λ_3 . The condition $\lambda_2 < 0$ requires $\beta x_2^* < \mu_2 + \delta$ (disease cannot invade), and $\lambda_3 < 0$ requires $ax_2^* < \mu_3 + h$ (predator cannot invade). Hence E_2 is locally asymptotically stable if and only if both stated conditions hold. $\square$

Theorem 3. *The predator-free endemic equilibrium $E_3 = ((\mu_2 + \delta)/\beta, y_3^*, 0)$, where*

$$y_3^* = \frac{r}{\beta} \left(1 - \frac{\mu_2 + \delta}{\beta K}\right) - \frac{\mu_1}{\beta} > 0,$$

exists whenever $y_3^ > 0$ and is locally asymptotically stable if*

$$ax_3^* + by_3^* < \mu_3 + h$$

and the Routh–Hurwitz conditions for the 2×2 prey subsystem $J_{xy}(E_3)$ are satisfied.

Proof. Denote $x_3^* = (\mu_2 + \delta)/\beta$. Evaluating (11) at $E_3 = (x_3^*, y_3^*, 0)$ gives

$$J(E_3) = \begin{bmatrix} a_{11} & a_{12} & -\alpha x_3^* \\ \beta y_3^* & 0 & -\varepsilon y_3^* \\ 0 & 0 & ax_3^* + by_3^* - (\mu_3 + h) \end{bmatrix},$$

where $a_{11} = r(1 - 2x_3^*/K) - \beta y_3^* - \mu_1 - m$ and $a_{12} = -\beta x_3^* < 0$. The block-triangular structure isolates one eigenvalue $\lambda_3 = ax_3^* + by_3^* - (\mu_3 + h)$; the condition $\lambda_3 < 0$ ensures the predator cannot invade the endemic state. The remaining two eigenvalues are eigenvalues of the 2×2 prey subsystem $J_{xy}(E_3)$ with characteristic polynomial $\lambda^2 + d_1\lambda + d_2 = 0$, where $d_1 = -a_{11}$ and $d_2 = \beta^2 x_3^* y_3^* > 0$. By the Routh–Hurwitz criterion both eigenvalues have negative real parts if and only if $d_1 > 0$, i.e., $a_{11} < 0$. $\square$

Theorem 4. *The disease-free prey–predator equilibrium $E_4 = ((\mu_3 + h)/a, 0, p_4^*)$, where*

$$p_4^* = \frac{1}{\alpha} \left[r \left(1 - \frac{\mu_3 + h}{aK} \right) - \mu_1 \right] > 0,$$

exists whenever $p_4^ > 0$ and is locally asymptotically stable if*

$$\beta x_4^* - \varepsilon p_4^* < \mu_2 + \delta$$

and the Routh–Hurwitz conditions for the 2×2 prey–predator subsystem $J_{xp}(E_4)$ are satisfied.

Proof. Denote $x_4^* = (\mu_3 + h)/a$. Evaluating (11) at $E_4 = (x_4^*, 0, p_4^*)$ gives

$$J(E_4) = \begin{bmatrix} a_{11} & -\beta x_4^* & -\alpha x_4^* \\ 0 & \beta x_4^* - \varepsilon p_4^* - (\mu_2 + \delta) & 0 \\ \alpha p_4^* & 0 & 0 \end{bmatrix},$$

where $a_{11} = r(1 - 2x_4^*/K) - \alpha p_4^* - \mu_1 - m$. The $(2, 2)$ entry is an isolated eigenvalue $\lambda_2 = \beta x_4^* - \varepsilon p_4^* - (\mu_2 + \delta)$; the condition $\lambda_2 < 0$ states that disease cannot invade the healthy prey–predator system. The remaining eigenvalues come from the 2×2 subsystem $J_{xp}(E_4)$ with characteristic polynomial $\lambda^2 + e_1\lambda + e_2 = 0$, where $e_1 = -a_{11}$ and $e_2 = \alpha x_4^* p_4^* > 0$. Both eigenvalues have negative real parts if and only if $e_1 > 0$, i.e., $a_{11} < 0$. $\square$

Theorem 5. *The full coexistence equilibrium $E_6 = (x^*, y^*, p^*)$ with $x^*, y^*, p^* > 0$ is locally asymptotically stable if and only if the Routh–Hurwitz conditions*

$$P_1 > 0, \quad P_3 > 0, \quad P_1 P_2 - P_3 > 0 \quad (12)$$

are satisfied, where $P_1 = -\text{tr}(J(E_6))$, $P_3 = -\det(J(E_6))$, and P_2 is the sum of the three principal 2×2 minors of $J(E_6)$.

Proof. Evaluating (11) at $E_6 = (x^*, y^*, p^*)$ gives

$$J(E_6) = \begin{bmatrix} J_{11} & -\beta x^* & -\alpha x^* \\ \beta y^* & J_{22} & -\varepsilon y^* \\ \alpha p^* & b p^* & 0 \end{bmatrix}, \quad (13)$$

where

$$J_{11} = r \left(1 - \frac{2x^*}{K} \right) - \beta y^* - \alpha p^* - \mu_1 - m, \quad (14)$$

$$J_{22} = \beta x^* - \varepsilon p^* - \mu_2 - \delta. \quad (15)$$

Note that $J_{33} = ax^* + by^* - (\mu_3 + h) = 0$ because the equilibrium condition $dp/dt = 0$ gives $ax^* + by^* = \mu_3 + h$. The characteristic polynomial of $J(E_6)$ is cubic, $\lambda^3 + P_1\lambda^2 + P_2\lambda + P_3 = 0$,

with

$$\begin{aligned} P_1 &= -(J_{11} + J_{22}), \\ P_2 &= J_{11}J_{22} + \beta^2 x^* y^* + a\alpha x^* p^* + b\varepsilon y^* p^*, \\ P_3 &= -\det(J(E_6)). \end{aligned}$$

By the Routh–Hurwitz criterion for a cubic polynomial, all roots have strictly negative real parts if and only if conditions (12) hold. The role of the migration parameter is transparent from (14): increasing m decreases J_{11} , which increases $P_1 = -(J_{11} + J_{22})$ and directly aids the first Routh–Hurwitz condition. Numerical verification of (12) under the parameter values of Table 2 is provided in Section 3. $\square$

3. Results and Discussion

This section presents the analytical results of the stability analysis together with their ecological interpretations, and verifies them through numerical simulation. We first interpret each equilibrium point ecologically, then state the stability conditions numerically under the parameter set of Table 2, and finally present numerical time-series and phase portraits that corroborate the analytical conclusions. The presentation structure follows related eco-epidemiological studies [1][9][17].

3.1. Parameter Values and Equilibrium Points

Numerical analysis is carried out using the parameter values listed in Table 2, which are adapted from the eco-epidemiological literature [1]. The migration parameter m does not appear in the numerical parameter file; the stability and simulation analyses in this section are therefore conducted with $m = 0$, consistent with the Maple computations. The analytical role of m is discussed separately in Subsection 3.3.

Table 2: Numerical parameter values used in the stability analysis and simulations

Parameter	Biological meaning	Value	References
r	Intrinsic growth rate of healthy prey	20.4	[1]
K	Environmental carrying capacity	10.2	[1]
β	Disease transmission rate	2.2	Assumed
α	Predation rate on healthy prey	1.2	Assumed
ε	Predation rate on infected prey	0.7	Assumed
μ_1	Natural mortality of healthy prey	0.001	Assumed
μ_2	Natural mortality of infected prey	0.003	Assumed
μ_3	Natural mortality of predator	0.002	Assumed
δ	Disease-induced mortality of infected prey	0.02	Assumed
h	Predator harvesting rate	1.0	Assumed
a	Predator conversion rate from healthy prey	0.15	Assumed
b	Predator conversion rate from infected prey	0.5	Assumed

Under these parameter values, the model admits the following biologically relevant equilibrium points, computed symbolically in `Symbolik.mw` and evaluated numerically in `Nilai_Parameter.mw`:

1. $E_1 = (0, 0, 0)$: trivial total extinction equilibrium.
2. $E_2 = (10.1995, 0, 0)$: disease-free, predator-free equilibrium; exists since $r = 20.4 > \mu_1 = 0.001$.
3. $E_3 = (0.4773, 0, 9.2628)$: disease-free prey–predator equilibrium (healthy prey and predator without infection); exists since $p_3^* > 0$.

4. $E_4 = (0.0105, 9.2628, 0)$: predator-free endemic equilibrium (healthy and infected prey without predator); exists since $y_4^* > 0$.
5. $E_5 = (10.1995, 0, 0)$: this equilibrium collapses numerically to E_2 under the given parameter values and does not represent a biologically distinct state.
6. $E_6 = (3.1360, 1.0632, 9.8233)$: full coexistence equilibrium with $x^*, y^*, p^* > 0$.

3.2. Stability Analysis of Each Equilibrium

The local stability of each equilibrium is determined by substituting the parameter values of Table 2 into the Jacobian (11) and evaluating the eigenvalues.

Equilibrium E_1 . The Jacobian at $E_1 = (0, 0, 0)$ is diagonal with eigenvalues $\lambda_1 = r - \mu_1 = 20.399 > 0$, $\lambda_2 = -(\mu_2 + \delta) = -0.023 < 0$, and $\lambda_3 = -(\mu_3 + h) = -1.002 < 0$. Since $\lambda_1 > 0$, E_1 is **unstable**: the ecosystem is viable and total extinction does not occur under these parameters.

Equilibrium E_2 . Evaluating the Jacobian at $E_2 = (10.1995, 0, 0)$ yields eigenvalues 116.030, $20.152i$, and $-0.152i$. The presence of a large positive eigenvalue ($116.030 > 0$) confirms that E_2 is **unstable**: both disease and the predator can invade the disease-free predator-free state under these parameter values.

Equilibrium E_3 . The Jacobian at E_3 has eigenvalues -12.809 , -0.551 , and 110.567 . The positive eigenvalue ($110.567 > 0$) indicates that E_3 is **unstable**: disease can invade the healthy prey-predator system.

Equilibrium E_4 . The eigenvalues of the Jacobian at E_4 are $-0.011 \pm 0.685i$ and 13.631 . The positive real eigenvalue ($13.631 > 0$) confirms that E_4 is **unstable**: the predator can invade the disease-endemic, predator-free state.

Equilibrium E_5 . The Jacobian at E_5 has eigenvalues -20.399 , 122.416 , and 0.528 . Both $122.416 > 0$ and $0.528 > 0$ confirm that E_5 is **unstable**. As noted above, E_5 collapses to the same coordinate as E_2 under these parameters and represents no distinct ecological state.

Equilibrium E_6 . The Jacobian evaluated at $E_6 = (3.1360, 1.0632, 9.8233)$ is

$$J(E_6) = \begin{bmatrix} -6.2721 & -6.8993 & -3.7632 \\ 2.3390 & -0.0001 & -0.7442 \\ 1.4735 & 4.9117 & 0 \end{bmatrix}, \quad (16)$$

with $J_{33} = ax^* + by^* - (\mu_3 + h) \approx 0$ as expected. The characteristic polynomial $\lambda^3 + P_1\lambda^2 + P_2\lambda + P_3 = 0$ has coefficients

$$P_1 \approx 6.272, \quad P_2 \approx 25.338, \quad P_3 \approx 58.595, \quad (17)$$

and the Routh-Hurwitz discriminant satisfies

$$P_1P_2 - P_3 \approx 6.272 \times 25.338 - 58.595 \approx 100.33 > 0. \quad (18)$$

All three Routh-Hurwitz conditions $P_1 > 0$, $P_3 > 0$, $P_1P_2 - P_3 > 0$ are satisfied, confirming that E_6 is **locally asymptotically stable**. The three eigenvalues are

$$\lambda_1 \approx -3.703, \quad \lambda_{2,3} \approx -1.285 \pm 3.765i, \quad (19)$$

confirming stability: λ_1 is real and negative, while $\lambda_{2,3}$ are complex conjugates with negative real part -1.285 . The complex pair indicates that convergence to E_6 is oscillatory in the (y, p) -subsystem with angular frequency ≈ 3.765 , while the x -component converges monotonically.

Consequently, E_6 is the **unique locally asymptotically stable equilibrium** under the given parameter set. All other equilibria (E_1 – E_5) are unstable, and the long-term dynamics are governed entirely by convergence to the full coexistence state E_6 .

3.3. Effect of the Migration Parameter m

Although m is set to zero in the numerical parameter file, its analytical role is transparent from the Jacobian structure. The migration parameter enters exclusively through the (1,1) entry,

$$J_{11} = r \left(1 - \frac{2x^*}{K} \right) - \beta y^* - \alpha p^* - \mu_1 - m, \quad (20)$$

so that increasing m strictly decreases J_{11} without altering any equilibrium coordinate.

At E_1 , the eigenvalue $\lambda_1 = r - \mu_1 - m$ decreases with m ; once $m > r - \mu_1 = 20.399$, total extinction becomes locally stable. At E_2 , the eigenvalue $\lambda_1 = -(r - \mu_1) - m$ becomes more negative with larger m , accelerating return to the prey-only state following an x -perturbation, but the invasion thresholds λ_2 and λ_3 are unaffected by m . At E_6 , increasing m increases $P_1 = -(J_{11} + J_{22})$ and thereby enlarges the parameter region satisfying the first Routh–Hurwitz condition, strengthening stability and accelerating recovery after perturbation.

This finding is consistent with [17], who demonstrated that migration modifies eigenvalues without altering equilibrium locations, and with [15][18], who showed that spatial movement affects dynamical stability in predator-prey systems.

3.4. Numerical Simulations

System Eqs. (1)–(3) is solved numerically using the fourth-order Runge-Kutta method (`dsolve` with `numeric` in Maple) on $t \in [0, 10]$ for the four initial conditions taken from `SimulasiNumerik_E6.mw`:

- IC₁: $(x_0, y_0, p_0) = (1.0, 0.5, 2.0)$,
- IC₂: $(x_0, y_0, p_0) = (5.0, 2.0, 5.0)$,
- IC₃: $(x_0, y_0, p_0) = (8.0, 3.0, 15.0)$,
- IC₄: $(x_0, y_0, p_0) = (2.0, 0.2, 8.0)$.

These initial conditions span a range of distances from E_6 and include cases in which all three populations are initially below (IC₁), near (IC₂), above (IC₃), or mixed relative to (IC₄) their equilibrium values. At $t = 10$, all four trajectories have converged to within 10^{-3} of $E_6 \approx (3.1360, 1.0632, 9.8233)$:

- IC₁: (3.1361, 1.0632, 9.8232),
- IC₂: (3.1360, 1.0632, 9.8233),
- IC₃: (3.1361, 1.0632, 9.8232),
- IC₄: (3.1360, 1.0632, 9.8233).

Figure 1 shows the trajectory of the healthy prey population $x(t)$. All four initial conditions exhibit damped oscillations that converge to $x^* = 3.1360$ (dashed line). IC₃, starting at $x_0 = 8.0$, undergoes the largest transient, descending to a first trough near $x \approx 0.9$ before recovering and settling. The oscillatory profile is consistent with the complex eigenvalue pair $\lambda_{2,3} \approx -1.285 \pm 3.765i$ that governs the coupled (y, p) -subsystem.

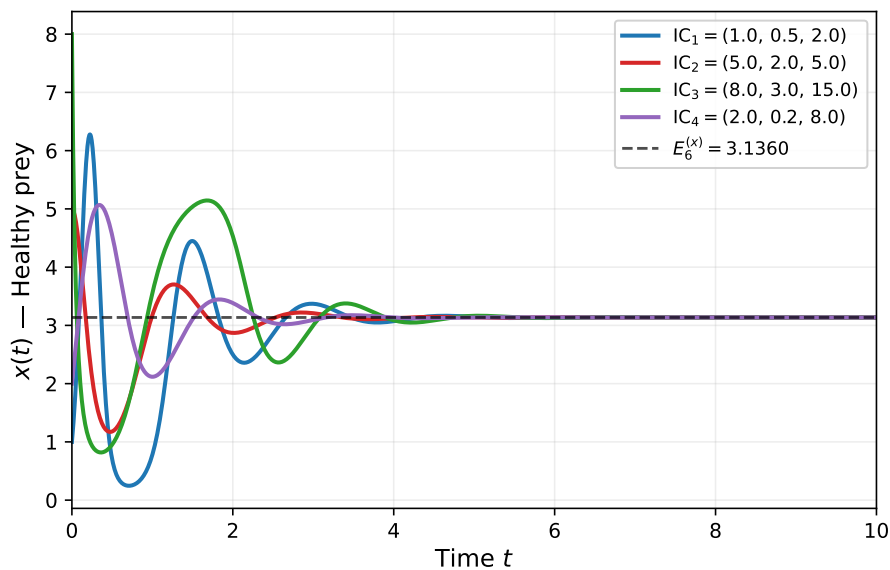


Fig. 1: Time-series of the healthy prey population $x(t)$ for four initial conditions converging to $x^* = 3.1360$ (dashed line) under the parameter values of Table 2. Colors: IC_1 (blue), IC_2 (red), IC_3 (green), IC_4 (purple).

Figure 2 shows the infected prey population $y(t)$. All trajectories exhibit a sharp initial peak caused by rapid disease transmission, followed by damped oscillations settling at $y^* = 1.0632$. The angular frequency of the oscillations (≈ 3.765) corresponds directly to the imaginary part of the complex eigenvalue pair $\lambda_{2,3}$.

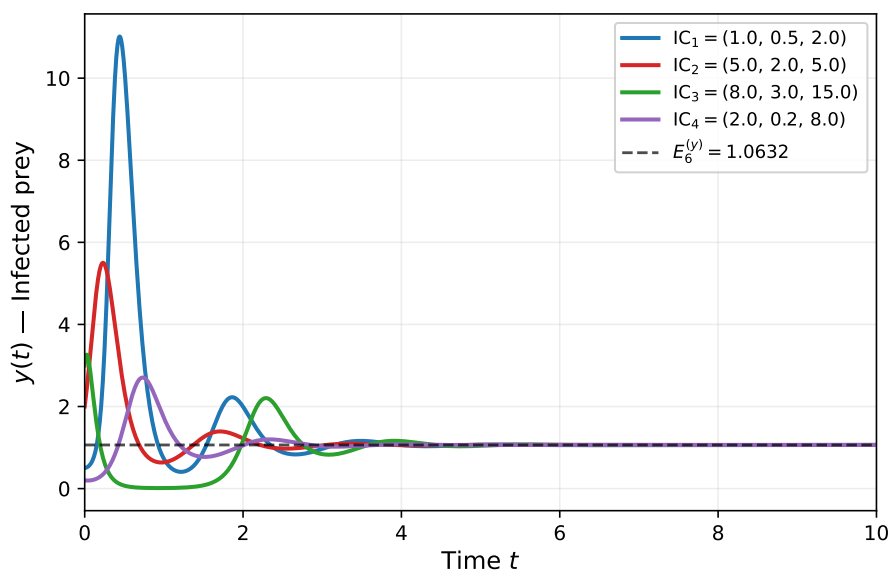


Fig. 2: Time-series of the infected prey population $y(t)$ for four initial conditions converging to $y^* = 1.0632$ (dashed line). Colors follow the same convention as Fig. 1.

Figure 3 shows the predator population $p(t)$. IC_3 (initialized at $p_0 = 15.0$, above p^*) undergoes an initial decline, while IC_1 (initialized at $p_0 = 2.0$, well below p^*) rises steeply before converging. All trajectories settle at $p^* = 9.8233$ by $t \approx 6$, confirming the robustness of the asymptotic stability of E_6 .

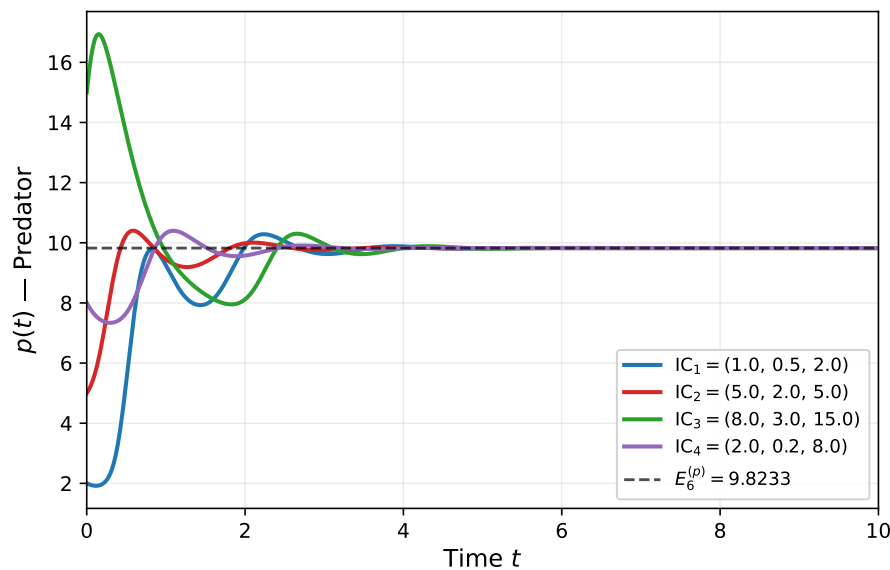


Fig. 3: Time-series of the predator population $p(t)$ for four initial conditions converging to $p^* = 9.8233$ (dashed line). Colors follow the same convention as Fig. 1.

Figure 4 presents a combined overlay of all three populations for all four initial conditions, illustrating the relative magnitudes and convergence rates simultaneously. The predator trajectory (green) dominates in magnitude; the healthy prey (blue) shows the widest oscillation amplitude; the infected prey (red) remains at a smaller density throughout. Dotted horizontal lines indicate the respective equilibrium values.

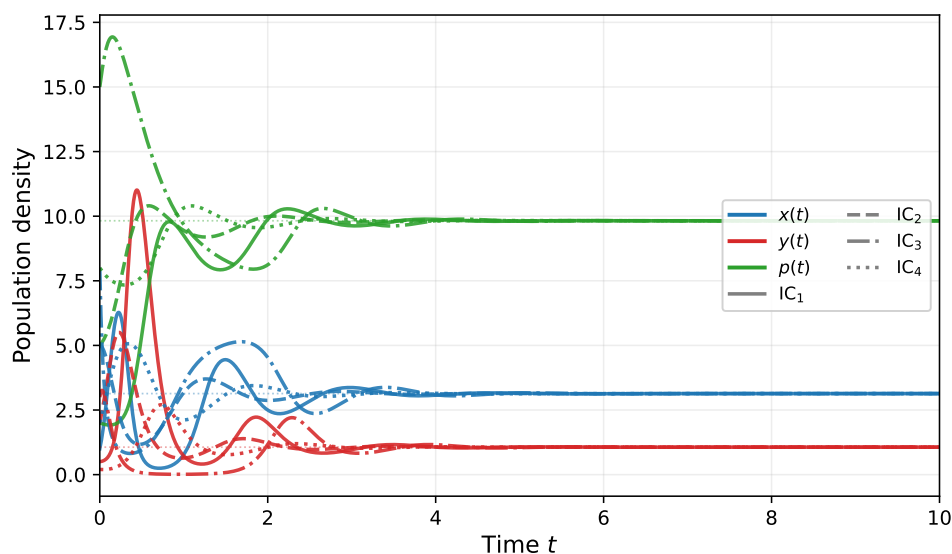


Fig. 4: Combined time series overlay of $x(t)$ (blue), $y(t)$ (red), and $p(t)$ (green) for all four initial conditions.

Line styles: IC_1 (solid), IC_2 (dashed), IC_3 (dash-dot), IC_4 (dotted). Horizontal dotted lines indicate equilibrium values.

The time-series dynamics are complemented by the phase portraits in Figures 5. shows the three-dimensional phase portrait in (x, y, p) -space. All four trajectories originate from their respective initial conditions (filled circles) and spiral inward toward E_6 (star marker), confirming the stable-focus behavior implied by the complex eigenvalue pair. The shadow projections onto the three coordinate planes are included for spatial reference.

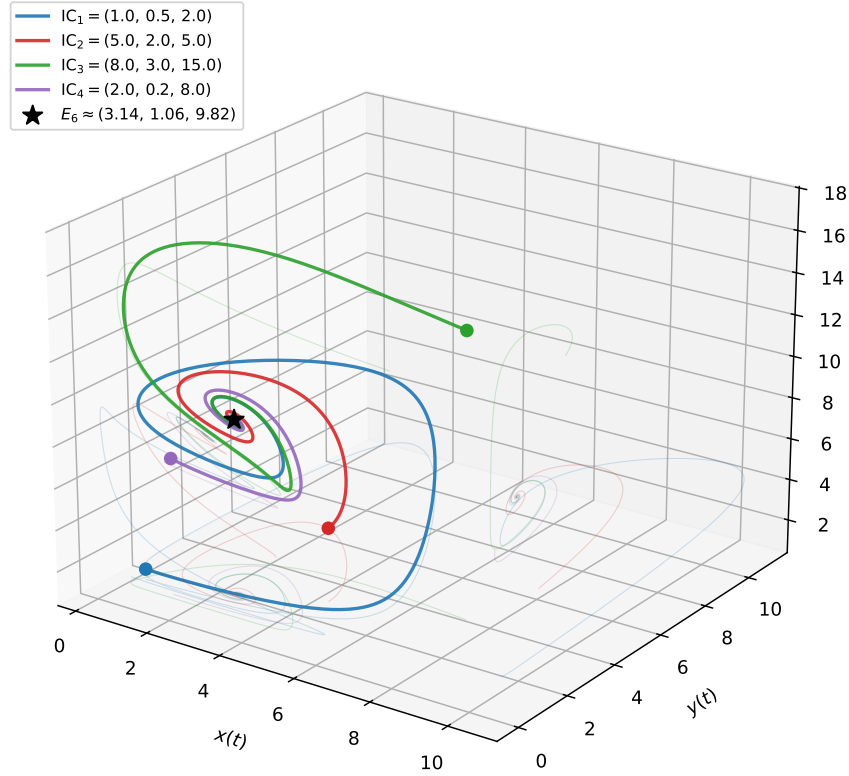


Fig. 5: Three-dimensional phase portrait in (x, y, p) -space.

All trajectories converge to $E_6 \approx (3.14, 1.06, 9.82)$ (star marker). Filled circles denote initial conditions; shadow projections on the coordinate planes are shown for spatial reference. Colors follow the same convention as Fig. 1.

3.5. Bifurcation Analysis

The stability analyses in the preceding subsections were conducted under the fixed baseline parameter set (Table 2), for which the coexistence equilibrium E_6 is the unique locally asymptotically stable attractor. This subsection examines how the qualitative long-run behaviour of the system changes as the predation rate on infected prey, ε , is varied continuously from zero to five.

The parameter ε is selected as the bifurcation parameter because it simultaneously governs the biological control of the disease (via the removal term εyp in the infected prey equation) and the predator growth rate (via the conversion term bpy), making it the principal parameter that determines whether the predator can suppress infection. This approach is consistent with the analysis of Pal et al. [20], who demonstrated in an eco-epidemiological model with harvesting that changes in key rate parameters particularly those coupling the infected prey and predator compartments induce Hopf bifurcation and govern the stable or unstable behaviour of the interior equilibrium. Similarly, Bezabih et al. [21] established in a five-compartment eco-epidemiological prey-predator model that the persistence or extinction of infection is entirely determined by whether the basic reproduction number crosses a threshold, which in the present context corresponds to whether ε exceeds the critical values identified below.

Numerical continuation of the equilibrium branches was performed in Maple (`Infeksi_preymw`), yielding two critical values:

- **Hopf bifurcation (H)** at $\varepsilon_H = 0.220995$, located at the coexistence equilibrium $E_6 = (1.212942, 1.640117, 11.970715)$;
- **Transcritical bifurcation (BP, branch point)** at $\varepsilon_{BP} = 2.501435$, located at the

disease-free equilibrium $E_3 = (6.68, 0, 5.865833)$.

Bifurcation Diagram

Figure 6 shows the bifurcation diagram of the system with the infected prey equilibrium density y^* plotted against ε .

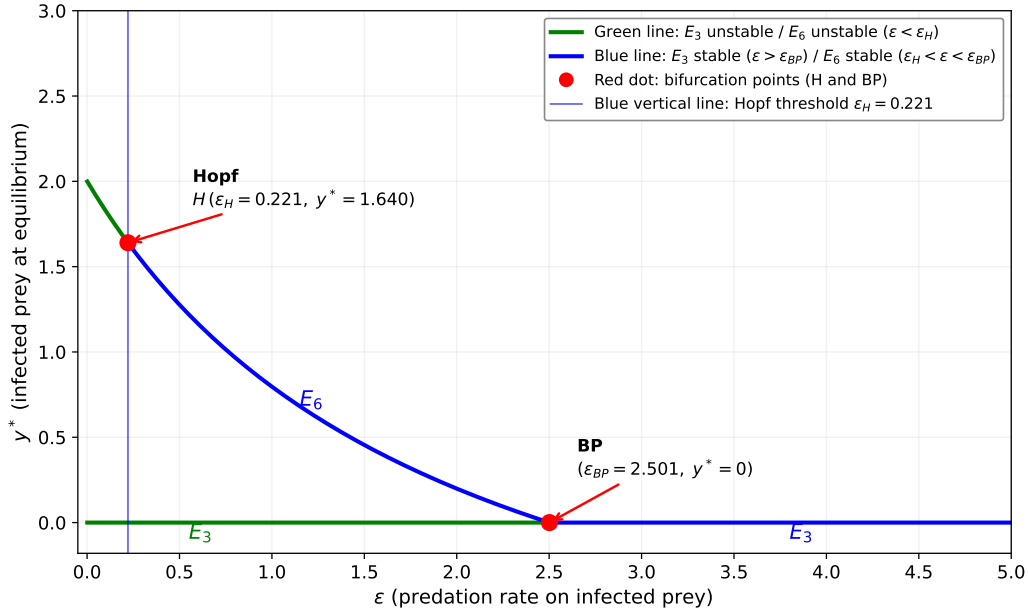


Fig. 6: Bifurcation diagram of the model with respect to the predation rate on infected prey ε (y^* = infected prey density at equilibrium).

Green line: unstable branches E_3 unstable and E_6 unstable ($\varepsilon < \varepsilon_H$); in this regime the coexistence equilibrium loses stability and a stable limit cycle exists. **Blue line:** stable branches E_6 stable ($\varepsilon_H < \varepsilon < \varepsilon_{BP}$) and E_3 stable ($\varepsilon > \varepsilon_{BP}$); higher predation on infected prey first stabilises coexistence, then eradicates the disease. **Red filled circles:** the two bifurcation points H (Hopf, $\varepsilon_H = 0.220995$, at E_6) and BP (transcritical, $\varepsilon_{BP} = 2.501435$, at E_3). **Blue vertical line:** Hopf threshold $\varepsilon_H = 0.221$ separating the oscillatory and stable-coexistence regimes. The diagram shows three qualitatively distinct dynamical regimes.

Regime I Oscillatory coexistence ($\varepsilon < \varepsilon_H = 0.220995$). In this regime the coexistence equilibrium E_6 is **unstable** (green curve above the axis). No stable fixed point exists; instead, all trajectories converge to a **stable limit cycle** the populations oscillate permanently about the unstable focus E_6 . When predation on infected prey is very weak, the disease-prey-predator feedback loop generates sustained oscillations that cannot be damped.

The condition for this regime can be understood through the Routh-Hurwitz framework: as ε decreases below ε_H , the product $P_1 P_2 - P_3$ (where P_1, P_2, P_3 are the coefficients of the characteristic polynomial of $J(E_6)$) crosses zero from positive to negative, violating stability. This is precisely the Hopf bifurcation criterion: a pair of complex eigenvalues of the Jacobian crosses the imaginary axis, giving rise to a stable limit cycle. Pal et al. [20] proved an analogous result for their eco-epidemiological model with harvesting, showing that the system undergoes a Hopf bifurcation around the coexistence equilibrium E_4 when the harvesting rate h_1 passes through the critical value h_1^* , with the same purely imaginary eigenvalue condition $\lambda_{1,2} = \pm i\sqrt{A_2}$ and $\lambda_3 = -A_1 < 0$ as the transversality condition [20, Theorem 5.7]. The present result is the parameter- ε analogue of that mechanism.

Regime II Stable coexistence ($\varepsilon_H < \varepsilon < \varepsilon_{BP} = 2.501435$). Once ε crosses the Hopf threshold, E_6 becomes **stable** (blue curve descending from $y^* = 1.640$ toward zero). The limit cycle disappears and the system settles at a fixed three-species equilibrium. As ε increases within this interval, the equilibrium infected prey density y^* decreases monotonically: stronger predation on infected prey progressively suppresses the disease while the system remains stable. The baseline simulation in Subsection 3.2 ($\varepsilon = 0.7$) belongs to this regime. The disease-free equilibrium E_3 is unstable throughout (green horizontal line at $y^* = 0$): complete disease eradication is not yet possible.

The behaviour in Regime II is ecologically consistent with the findings of Bezabih et al. [21], who showed that in an eco-epidemiological prey-predator model with treatment, the disease *persists* in the system whenever the basic reproduction number $\mathcal{R}_0 > 1$, while the endemic equilibrium remains locally asymptotically stable under the Routh-Hurwitz conditions. In the present context, $\varepsilon \in (\varepsilon_H, \varepsilon_{BP})$ corresponds precisely to this endemic-but-stable regime: the infection is present at equilibrium density $y^* > 0$, yet all perturbations decay and the system returns to E_6 [21].

Regime III Disease eradication ($\varepsilon > \varepsilon_{BP} = 2.501435$). At the transcritical bifurcation (BP), the coexistence equilibrium E_6 collides with the disease-free equilibrium E_3 (both converge to the point $y^* = 0$) and they exchange stability. For $\varepsilon > \varepsilon_{BP}$, the E_3 branch becomes **stable** (blue horizontal line at $y^* = 0$) while E_6 loses biological feasibility ($y^* < 0$, not shown). Sufficiently intense predation on infected prey drives the infection to extinction, leaving a disease-free prey-predator state at $E_3 = (6.68, 0, 5.866)$.

This outcome mirrors the disease-free equilibrium stability established by Bezabih et al. [21], where the axial equilibrium E_A (equivalent to E_3 here) becomes locally asymptotically stable whenever the infection-invasion conditions are reversed corresponding to $\mathcal{R}_0 < 1$. Similarly, Pal et al. [20] observed that harvesting of susceptible prey and predator populations can regulate the cyclic behaviour of the system and even eliminate prey infection altogether, which is precisely the eradication outcome observed here for $\varepsilon > \varepsilon_{BP}$.

Three-Dimensional Phase Portrait at the Hopf Point ($\varepsilon = 0.220995$, Regime I)

Figure 7 illustrates the dynamics of Regime I in full (x, y, p) -space. Three trajectories from different initial conditions are integrated up to $t = 150$. Each trajectory starts from a filled circle, spirals *outward* from the unstable focus E_6 (red star), and converges onto a common closed orbit the **stable limit cycle** born at the Hopf bifurcation. The trajectories originating both inside (blue, red) and outside (green) the limit cycle all converge to the same orbit, confirming that the Hopf bifurcation is **supercritical**: a globally attracting limit cycle exists for $\varepsilon \leq \varepsilon_H$ [5][6].

This three-dimensional portrait is consistent with the bifurcation diagrams reported by Pal et al. [20], who showed that when the harvesting parameter passes through its critical value $h_1^* = 0.0452$, the coexistence equilibrium transitions from stable to unstable and the solution trajectories spiral outward to a limit cycle an identical qualitative pattern to what is observed here.

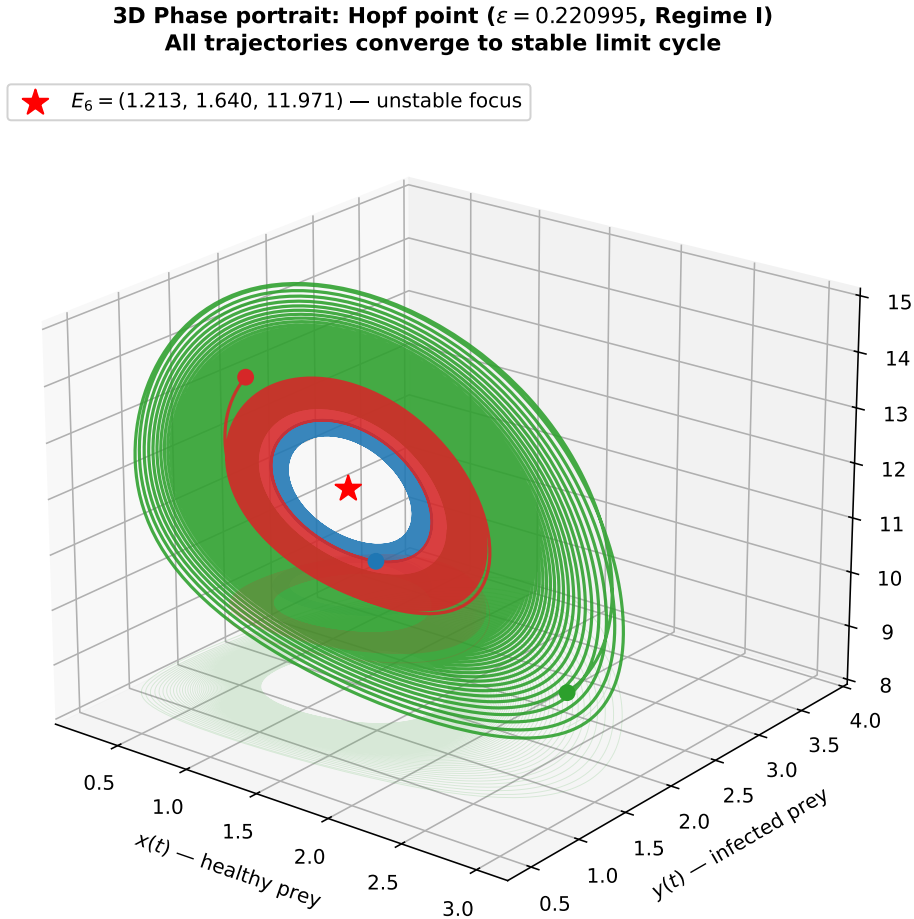


Fig. 7: Three-dimensional phase portrait at the Hopf bifurcation point $\varepsilon = \varepsilon_H = 0.220995$ (Regime I: oscillatory coexistence).

Filled circles: initial conditions. Red star: unstable focus $E_6 = (1.213, 1.640, 11.971)$. All three trajectories spiral outward from E_6 and converge to the stable limit cycle, confirming a supercritical Hopf bifurcation [20]. Faint curves on the floor are shadow projections onto the xy -plane.

Three-Dimensional Phase Portrait in the Disease-Free Regime ($\varepsilon = 3.0$, Regime III)

Figure 8 shows the phase portrait at $\varepsilon = 3.0 > \varepsilon_{BP}$, deep in the disease-eradication regime. Four trajectories starting from widely separated initial conditions all converge directly to the disease-free equilibrium $E_3 = (6.68, 0, 5.866)$ (red star). The infected prey component $y(t)$ decays monotonically to zero in every case: the predator is now efficient enough at removing infected prey to eradicate the disease entirely. No oscillations are present, and the coexistence equilibrium E_6 no longer exists as a biologically feasible point in this regime.

The local asymptotic stability of E_3 in this regime is verified by the Routh-Hurwitz criterion applied to the Jacobian $J(E_3)$ evaluated at $\varepsilon = 3.0$, which yields all eigenvalues with strictly negative real parts. This is analogous to the stability of the disease-free equilibrium established by Bezabih et al. [21] using the variation matrix and Routh-Hurwitz criterion for their five-compartment model: the disease-free equilibrium point E_A is locally asymptotically stable whenever the disease-invasion eigenvalue is negative, i.e., when predation pressure is sufficient to prevent re-invasion of the infected class [21].

3D Phase portrait: E_3 -stable regime ($\varepsilon = 3.0$, Regime III)
All trajectories converge to disease-free E_3

★ $E_3 = (6.68, 0, 5.866)$ — disease-free stable

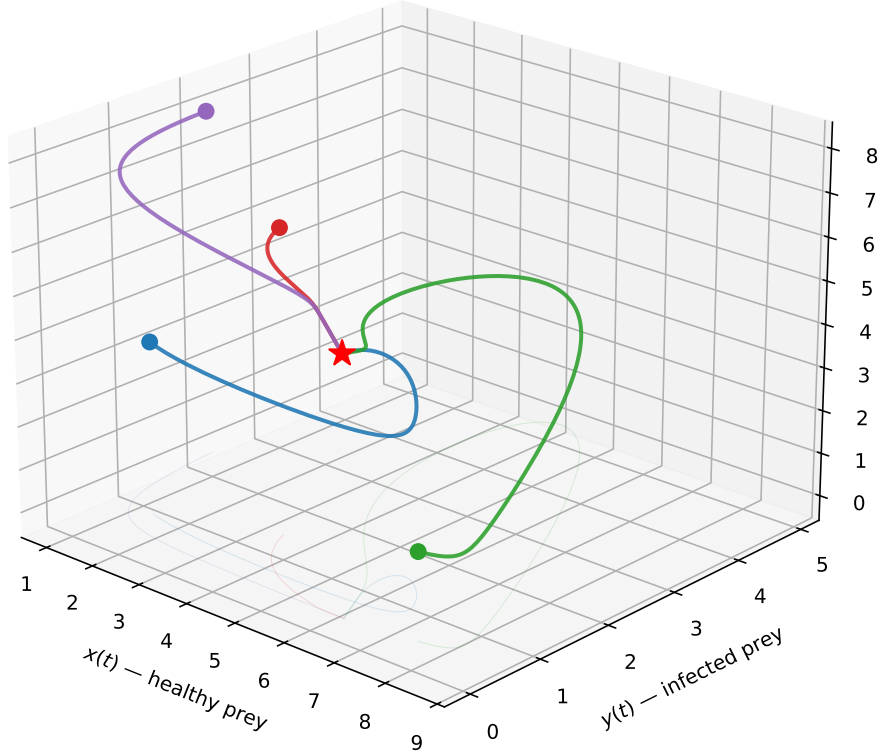


Fig. 8: Three-dimensional phase portrait at $\varepsilon = 3.0 > \varepsilon_{BP}$ (Regime III: disease eradication). Filled circles: initial conditions.

Red star: stable disease-free equilibrium $E_3 = (6.68, 0, 5.866)$. All four trajectories converge to E_3 ; the infected prey $y(t) \rightarrow 0$ in every case, confirming complete disease eradication [21].

Time-Series at the Hopf Bifurcation Point ($\varepsilon = 0.220995$, Regime I)

Figure 9 shows the time-series of all three populations at $\varepsilon = \varepsilon_H = 0.220995$, starting from $(x_0, y_0, p_0) = (1.5, 1.5, 11.0)$. After a short initial transient, all three populations enter a state of **persistent periodic oscillation** about the unstable equilibrium values (dashed lines), maintained without decay for the entire duration $t \in [0, 200]$.

The oscillation period is

$$T = \frac{2\pi}{\omega} \approx \frac{2\pi}{3.796} \approx 1.66 \text{ time units}, \quad (21)$$

where $\omega = 3.796$ is the imaginary part of the critical eigenvalue pair $\lambda_{1,2} = \pm 3.796i$ at ε_H . This is consistent with the Hopf bifurcation condition verified analytically by Pal et al. [20]: at the critical parameter value, the characteristic polynomial admits purely imaginary roots $\lambda_{1,2} = \pm i\sqrt{A_2}$ together with a strictly negative real root $\lambda_3 = -A_1$, and the transversality condition $\frac{d}{dh_1} \text{Re}(\lambda_i)|_{h_1=h_1^*} \neq 0$ is satisfied, guaranteeing the birth of a limit cycle [20, Theorem 5.7].

The three populations oscillate in a coordinated predator-disease-prey cycle: peaks in healthy prey $x(t)$ are followed by peaks in infected prey $y(t)$, which in turn trigger a lagged response in the predator $p(t)$, illustrating the classical trophic feedback that underlies eco-epidemiological oscillations [9][10].

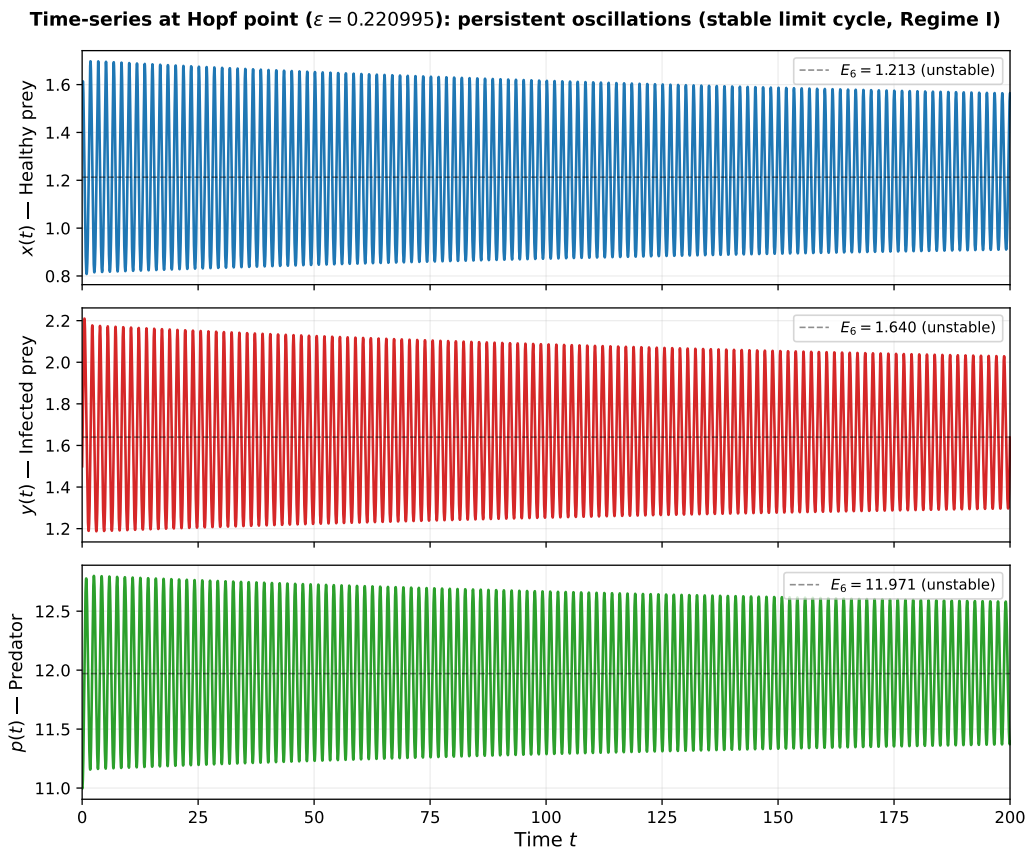


Fig. 9: Time-series of $x(t)$ (blue), $y(t)$ (red), and $p(t)$ (green) at the Hopf point $\varepsilon = \varepsilon_H = 0.220995$ (Regime I), starting from $(x_0, y_0, p_0) = (1.5, 1.5, 11.0)$.

Dashed horizontal lines mark the unstable equilibrium values of E_6 . All populations sustain permanent periodic oscillation; the amplitude remains constant for $t \leq 200$, confirming the stable limit cycle [20].

Summary of Dynamical Regimes

Table 3 summarises the three regimes identified by the bifurcation analysis.

Table 3: Summary of dynamical regimes as a function of the predation rate on infected prey ε .

Regime	ε range	Stable attractor	Ecological outcome
I	$\varepsilon < 0.221$	Stable limit cycle	Permanent oscillation; all species persist
II	$0.221 < \varepsilon < 2.501$	$E_6 = (x^*, y^*, p^*)$, all > 0	Stable three-species coexistence with disease
III	$\varepsilon > 2.501$	$E_3 = (6.68, 0, 5.866)$	Disease eradication; healthy prey + predator

Ecological Interpretation

The three regimes identified above carry clear ecological implications. In Regime I ($\varepsilon < 0.221$), the predator is too ineffective at removing infected prey to break the disease feedback loop, resulting in permanent population oscillations. Such oscillatory dynamics in eco-epidemiological systems have been linked to the risk of transient local extinction during extreme phases of the cycle [10][21].

In Regime II ($0.221 < \varepsilon < 2.501$), the three species coexist at a stable equilibrium and the infected prey density decreases as ε increases. This is precisely the *endemic-but-stable* regime described by Bezabih et al. [21]: the disease is present ($\mathcal{R}_0 > 1$) but the system does not oscillate, and the endemic equilibrium is globally stable under the Lyapunov conditions they established.

Regime II is also the regime studied by Pal et al. [20] for intermediate harvesting rates, where both prey and predator persist in the presence of infection.

In Regime III ($\varepsilon > 2.501$), the increased predation efficiency on infected prey is sufficient to drive $\mathcal{R}_0 < 1$, analogous to the disease-eradication condition obtained by Bezabih et al. [21]. Pal et al. [20] similarly showed that harvesting can *eliminate prey infection altogether* when the harvesting rate is large enough the same qualitative outcome obtained here through the parameter ε , confirming the generality of the disease-eradication mechanism across different eco-epidemiological model structures [5][6][20][21].

3.6. Effect of the Harvesting Rate h

To illustrate how the harvesting rate influences the coexistence equilibrium, we fix all parameters at the baseline values of Table 2 and vary $h \in \{0.5, 1.0, 1.5, 2.0\}$, using the common initial condition $(x_0, y_0, p_0) = (2.0, 0.5, 5.0)$. Under each value of h the system still converges to a locally asymptotically stable coexistence equilibrium; however, the location of that equilibrium shifts monotonically with h .

Figure 10 shows the effect of h on the healthy prey $x(t)$. Higher harvesting slightly raises the long-term density $x^*(h)$, since reduced predator pressure relaxes predation on x .

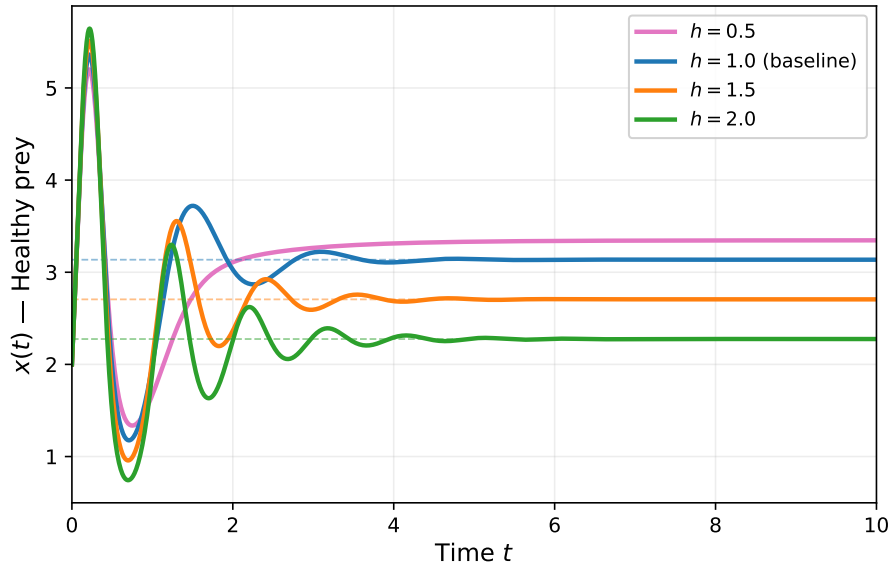


Fig. 10: Healthy prey population $x(t)$ for four values of the harvesting rate $h \in \{0.5, 1.0, 1.5, 2.0\}$.

Dashed lines indicate the corresponding equilibrium value $x^*(h)$. Baseline $h = 1.0$ is shown in blue.

Figure 11 shows the effect of h on the infected prey $y(t)$. As predator pressure is reduced by harvesting, the biological control of infected prey weakens, allowing the disease to persist at a higher equilibrium level. Notably, the lowest harvesting rate $h = 0.5$ (pink) leads to near-zero infected prey at equilibrium, suggesting a predator-protection threshold below which the predator is effective enough to suppress the disease entirely.

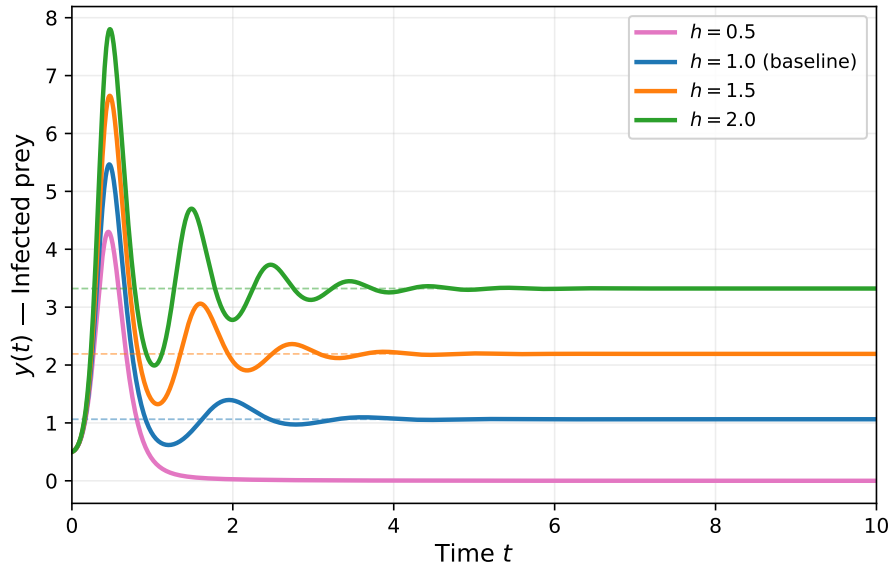


Fig. 11: Infected prey population $y(t)$ for four values of the harvesting rate $h \in \{0.5, 1.0, 1.5, 2.0\}$.

Dashed lines indicate the corresponding equilibrium value $y^*(h)$. At $h = 0.5$ (pink), disease prevalence is effectively eliminated.

Figure 12 shows the effect of h on the predator $p(t)$. Higher harvesting dramatically reduces the predator equilibrium $p^*(h)$, as expected: heavier harvesting directly removes predators from the system. This confirms the ecological intuition that excessive harvesting destabilizes biological pest control by depleting the predator guild.

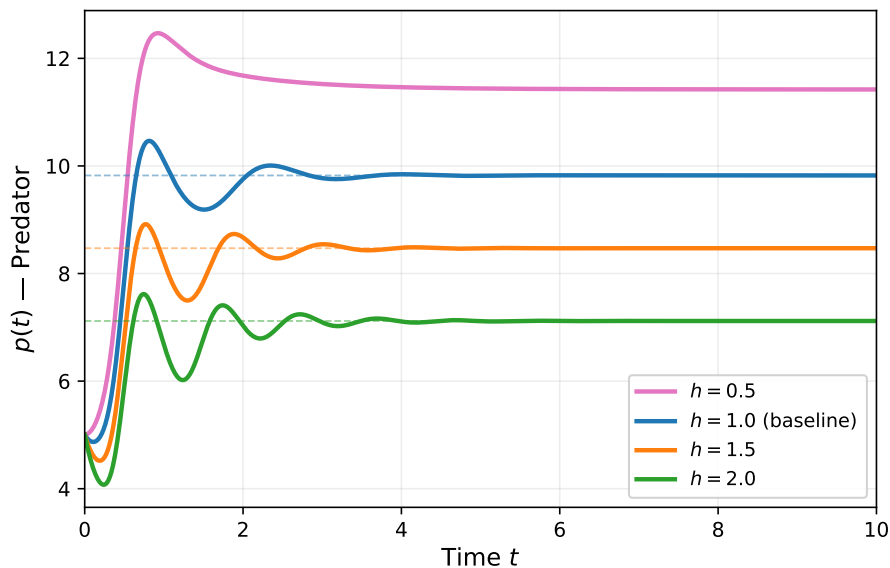


Fig. 12: Predator population $p(t)$ for four values of the harvesting rate $h \in \{0.5, 1.0, 1.5, 2.0\}$.

Dashed lines indicate the corresponding equilibrium value $p^*(h)$. The predator density decreases monotonically with increasing h .

Taken together, Figures 10–12 reveal a clear ecological trade-off: higher harvesting rates benefit healthy prey (lower predation pressure) but increase disease prevalence and deplete the predator guild. This finding has direct management implications: unrestricted harvesting of predators may inadvertently exacerbate disease outbreaks in prey populations, consistent with results reported in [1][17].

These results collectively confirm the analytical predictions of Subsection 3.2: E_6 is the

unique locally asymptotically stable equilibrium under the given parameter set, and its location shifts predictably with the harvesting parameter h .

Further studies have extended eco-epidemiological frameworks by incorporating additional complexity such as prey refuge [9][11], cooperative hunting [6][8][12], group defense effects [22][23], fear effects [7][12][13], and harvesting strategies [14][15][16].

4. Conclusion

This study has developed and analyzed a three-dimensional eco-epidemiological predator-prey model that simultaneously incorporates disease in the prey population, constant harvesting of the predator, and internal migration of healthy prey within a closed ecosystem. The model extends the foundational work of Mansur et al. [1] by introducing a density-dependent migration term $m(x_e - x)$ into the healthy prey equation, motivated by the spatial dynamics studied by Kant and Kumar [17] and Dai et al. [18].

The following principal results were established.

1. Equilibrium analysis. The system admits six equilibrium points: the trivial extinction equilibrium E_1 , the disease-free predator-free equilibrium E_2 , the disease-free prey-predator equilibrium E_3 , the predator-free endemic equilibrium E_4 , a degenerate state E_5 that collapses to E_2 under the given parameter values, and the full coexistence equilibrium $E_6 = (x^*, y^*, p^*)$ with all components strictly positive. The existence conditions for each equilibrium are expressed analytically in terms of the model parameters. A key structural feature is that the migration term does not alter any equilibrium coordinate, since the flux $m(x_e - x)$ vanishes identically at equilibrium ($x = x_e$), so the six equilibrium points are the same whether $m > 0$ or $m = 0$.

2. Local stability. The local stability of each equilibrium was determined by Jacobian linearization and, for the interior equilibrium E_6 , by the Routh-Hurwitz criterion applied to the resulting cubic characteristic polynomial. Under the numerical parameter set adapted from [1], equilibria E_1 through E_5 are all unstable, while the full coexistence equilibrium $E_6 = (3.1360, 1.0632, 9.8233)$ is the unique locally asymptotically stable attractor, with Routh-Hurwitz coefficients $P_1 \approx 6.272$, $P_2 \approx 25.338$, $P_3 \approx 58.595$, and discriminant $P_1P_2 - P_3 \approx 100.33 > 0$. The three eigenvalues $\lambda_1 \approx -3.703$ and $\lambda_{2,3} \approx -1.285 \pm 3.765i$ confirm asymptotic stability with oscillatory convergence in the infected prey-predator subsystem.

3. Role of internal migration. Although the migration parameter m does not affect the equilibrium locations, it appears in the $(1, 1)$ entry of the Jacobian and therefore influences the eigenvalues and Routh-Hurwitz conditions. Increasing m decreases J_{11} , increases $P_1 = -(J_{11} + J_{22})$, and thereby enlarges the parameter region satisfying the first Routh-Hurwitz condition, strengthening the local asymptotic stability of E_6 and accelerating the rate of return after perturbation. This result confirms and extends the findings of Kant and Kumar [17], who demonstrated that migration modifies eigenvalues without shifting equilibrium locations, and is consistent with the broader conclusion of Dai et al. [18] and Roy and Roy [15] that spatial movement of prey directly affects the stability properties of predator-prey systems.

4. Numerical simulation. The analytical stability predictions were verified numerically using the Runge-Kutta method on $t \in [0, 10]$ for four initial conditions spanning a wide range of distances from E_6 . All four trajectories converged to within 10^{-3} of E_6 by $t = 10$, corroborating the local asymptotic stability established analytically. The oscillatory transients in all three population components are consistent with the imaginary part $\omega \approx 3.765$ of the complex eigenvalue pair. The three-dimensional phase portrait and population time-series confirm that the long-run state of the ecosystem is stable coexistence of healthy prey, infected prey, and predator at the equilibrium densities $x^* = 3.1360$, $y^* = 1.0632$, and $p^* = 9.8233$.

5. Effect of harvesting. Varying the harvesting rate $h \in \{0.5, 1.0, 1.5, 2.0\}$ reveals a monotone trade-off: higher harvesting relaxes predation pressure on healthy prey (raising x^*) but simultaneously weakens biological control of infected prey (raising y^*) and reduces the predator equilibrium density p^* . This quantifies the risk that unrestricted predator harvesting inadvertently amplifies disease prevalence in prey populations, a finding with direct ecological management implications consistent with [1][20].

6. Bifurcation analysis. Continuation of equilibrium branches with respect to the predation rate on infected prey ε reveals two critical values: a supercritical Hopf bifurcation at $\varepsilon_H = 0.220995$, where E_6 loses stability and a stable limit cycle is born, and a transcritical bifurcation at $\varepsilon_{BP} = 2.501435$, where E_6 and E_3 exchange stability and the disease is eradicated. These two bifurcation points partition the parameter space into three dynamical regimes: (I) oscillatory coexistence for $\varepsilon < \varepsilon_H$, (II) stable three-species coexistence for $\varepsilon_H < \varepsilon < \varepsilon_{BP}$ (the regime of the baseline simulation), and (III) disease-free stable coexistence for $\varepsilon > \varepsilon_{BP}$. The bifurcation structure is consistent with eco-epidemiological results in [9][10][20][21].

Taken together, the results demonstrate that disease in prey, predator harvesting, and internal prey migration jointly govern the stability and long-run behavior of the ecosystem, with each factor contributing a distinct and analytically transparent mechanism. The coexistence equilibrium E_6 acts as a robust global attractor under the baseline parameter set, and its stability is enhanced by internal migration and by sufficiently strong predation on infected prey.

Several directions remain open for future investigation. First, an analysis of global stability for E_6 using an appropriate Lyapunov function a programme carried out for related models in [21] and [10] would strengthen the local results obtained here. Second, the present model assumes a linear (Holling Type I) functional response for both prey types; extending to Holling Type II or Type III responses, as studied in [7][8], is likely to produce richer bifurcation structures including additional Hopf points and limit-cycle branches. Third, the internal migration term $m(x_e - x)$ could be replaced by a fully spatial two-patch or reaction-diffusion formulation to capture more realistic heterogeneous habitats, following the approach of Kant and Kumar [17] and Dai et al. [18]. Finally, the analytical expression for the Hopf bifurcation threshold ε_H in terms of the model parameters, and verification of the transversality condition confirming the bifurcation is supercritical, constitute valuable extensions of the present bifurcation analysis [5][6][20].

CRedit Authorship Contribution Statement

Muhammad Thariq Firmansyah: Conceptualization, Methodology, Writing-Original Draft, Visualization. **Dian Savitri:** Data Curation, Formal Analysis, Project Administration, Validation

Declaration of Generative AI and AI-assisted technologies

No generative AI or AI-assisted technologies were used during the preparation of this manuscript.

Declaration of Competing Interest

The authors declare no competing interests.

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Data and Code Availability

The data and code supporting the findings of this study are available from the corresponding author upon reasonable request.

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