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Deterministic Study of an Eco-epidemiological Model with Prey Refuge and Predator Harvesting

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ABSTRACT: This paper focuses on analyzing the dynamical behavior of an eco-epidemiological model in which the predator population is impacted by disease. The total population is categorized into three classes: prey, susceptible predators, and infected predators. The model incorporates two linear prey refuges against both susceptible and infected predators, along with a Holling type I functional response. Infectious diseases that spread between species can severely threaten ecosystem stability by reducing biodiversity and driving species toward extinction. To curb disease transmission, the model includes a strategy of selectively harvesting infected predators. The study explores the non-negativity and boundedness of solutions, along with the existence and stability of equilibrium points. Conditions for the global stability of these equilibrium points are also examined. Furthermore, the occurrence of transcritical and Hopf bifurcations is investigated theoretically. Finally, numerical simulations, supported by experimental data, are used to compare and validate the analytical findings.

Keywords: Prey refuge, predator harvesting, eco-epidemiology, bifurcation analysis, numerical simulation.

Contents

1. Introduction

In recent years, mathematical biology has emerged as one of the most significant subjects. The British economist Malthus [1] developed the first population biology model about 1800, and Verhulst [2] subsequently adjusted it for a more practical scenario. The Lotka-Volterra model, which was independently put forth by Alfred J. Lotka [3] and Vito Volterra [4] in the 1920s, is among the early studies in this field. In our world, diseases are fairly common phenomena and we know from our history that they can have a significant impact on the size of the size of the affected species population. Kermack and McKendric [5] were among the first to study disease transmission via mathemathical modelling. After that various epidemic models have been established and investigated extensively. For many years, epidemiology and ecology were two distinct disciplines. These two disciplines blended together in the late 1980s and early 1990s, giving rise to a brand-new hybrid field of study known as eco-epidemiology. Eco-epidemiological

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modeling were first introduced by Anderson and May [6]. They considered disease in the prey population and found that disease can destabilize the system. Chattopadhayay and Arino [7] considered an eco-epidemiological system consisting of three species, namely, the sound prey (which is susceptible), the infected prey (which becomes infective by some viruses) and the predator population. In [8] Chattopadhayay et al. analysed an eco-epidemiological system with disease in prey population and applied it to the fish-pelican system in the Salton Sea. In the same context, Bairagi et al. [9] investigated the role of infection in different types of functional responses. Kashyap et al. [10] investigated the effect of fear in the interaction between Tilapia and Pelican birds in the presence of a pathogen, utilizing the Caputo fractional derivative framework. Recently much work has been done incorporating disease in the prey population (See for example [11,12,13,14,15,16]).

While prey are often the primary victims of disease, nature sometimes flips the script—predators, too, can fall prey to contagious infections [17]. Venturino [18] proposed an eco-epidemiological model considering disease in predator species only. Haque et al. [19] analyzed a model incorporating disease in predator populations. In [20] Pal et al. proposed a prey-predator system with an infectious disease that spreads on the predator population and analysed the stability and direction of Hopf bifurcation. Biswas et al. [21] explored the population dynamics of a time-delayed interaction model, incorporating disease within the predator compartment. In addition, Sarwardi et al. [22], Kashyap et al. [23], Al-Jubouri & Naji [24], Fakhry & Naji [25] discussed the dynamics of predator-prey systems with disease in predator population. Das et al. [26], Arora et al. [27], Bezabih et al. [28], Kashyap et al. [29] etc. analyzed the dynamical behavior of predator-prey system with diseases affecting both species.

Biomathematical investigations frequently focus on predator–prey systems that incorporate prey refuge, recognizing its significance in shaping ecological outcomes. Prey refuge refers to a protected zone or condition that allows prey species to evade predators by reducing their detectability or accessibility. Prey species that utilize refuge are often less visible to predators and tend to occupy locations that are difficult or impossible for predators to reach. For instance, *Daphnia* avoid fish predation in Mediterranean shallow lakes by hiding within the sediments [30], while juvenile sea urchins find protection from predatory crabs by sheltering within articulated coralline algae [31]. Kar [32] examined a predator–prey model that includes prey refuge and demonstrated that an unstable positive equilibrium gives rise to a unique limit cycle. The study further concluded that if the positive equilibrium is locally asymptotically stable, it is also globally asymptotically stable. Mukherjee [33] analyzed a resource-dependent system with prey refuge using Holling type I and II responses, concluding that all three populations can persist if predator mortality is below a critical threshold. Chen et al. [34] examined the stability characteristics of a predator–prey model with a fixed number of prey refuges. In a separate study, the authors of [35] concluded that prey refuge does not affect the system's stability. Meanwhile, a model discussed in [36], which incorporates both prey refuge and alternative food source for predators, revealed that higher levels of refuge can support long-term coexistence of species. However, excessive refuge availability may lead to predator extinction. Interestingly, refuge can have both stabilizing [38] and destabilizing [39] effects on the system dynamics. For more biological backgrounds and results on the effects of a prey refuge, one could refer to [16,40,41,42,43,44,45].

Harvesting a population involves the removal of individuals from a group, usually through activities such as hunting, fishing, culling, or other methods of capture and extraction. This approach is often applied in wildlife management, fisheries, agriculture, and conservation to achieve specific goals. Researchers have extensively analyzed predator-prey systems with harvesting, accounting for infections in prey, predators, or both. In our model, a diseased predator population is considered with a harvesting infected population. Harvesting infected predators can help reduce the spread of diseases within predator populations, and eventually it will decrease the disease transmission rate to prey species. There are many real scenarios where we can see only infected population are harvested. In aquaculture, fish and other aquatic organisms face various challenges including disease outbreaks. These diseases can devastate fish populations, impacting both the livelihood of fish farmers and the wider ecosystem. For instance, sea lice are a common parasitic copepod that affects Atlantic Salmon farms. They feed on the fish's skin and mucus, weakening them and increasing disease susceptibility. In 2011, the Norwegian government acknowledged that sea lice from fish farms could be a serious threat to wild salmon. According to Scottish farmed salmon [46], a severe sea lice infection is one of the leading causes of high mortality rates of salmon.

To reduce the spread of sea lice to healthy fish, the industry often chooses to harvest diseased fish over treating them by selectively targeting and harvesting fish within farms or specific cages most affected by diseases or conditions [47]. *Koi herpesvirus*(KHV) is a severe viral disease that poses significant challenges to koi and carp populations worldwide. There is currently no specific treatment for KHV once fish are infected. Infected fish are often removed from ponds and aquaculture facilities to prevent the spread of the virus to other populations. Culling infected populations in an ecosystem (mainly in aquaculture) is also considered as a valuable tool for controlling disease spread and maintaining ecological balance. This practice involves selectively removing the species that are infected with a particular disease. *African swine fever* (ASF) is a contagious viral disease that highly affects wild boar. It is caused by the *African swine fever virus* (ASFV), which belongs to the *Asfarviridae* family. A proper surveillance and monitoring scheme is a requisite for disease control in wildlife. The authors of [48] state that wild boar culling is one of the essential tools for controlling ASF. *Saprolegnia disease*, also known as "*saprolegniasis*", is a common fungal infection in fish and fish eggs. It affects freshwater fish and is a significant problem in aquaculture, fisheries, and natural water bodies. In case of severe infection, to prevent further spread and reduce overall mortality, the infected fish should be harvested as soon as *saprolegniasis* is observed [49,52]. *Neon tetra disease* (NTD) is a parasitic infection that primarily affects neon tetras. The disease is caused by the microsporidian parasite *Pleistophora hyphessobryconis.* It is highly contagious and often fatal. There is no known cure for this disease. Culling or destroying the infected fish is the only option to prevent the disease [49]. *Lymphocystis* disease is a chronic viral infection affecting a wide range of freshwater and marine fish species. The disease is characterized by the formation of distinctive, wart-like nodules on the skin, fins, and occasionally the internal organs of infected fish. There is currently no specific cure for lymphocystis disease. To prevent the spread of the virus to healthy fish, the infected fish need to be harvested and removed [49]. *Mycobacteriosis* in fish is a chronic bacterial infection caused by various species of the genus Mycobacterium. This disease can affect a wide range of freshwater and marine fish species and is known for its persistence and difficulty to treat. It is also a zoonotic disease, meaning it can be transmitted to humans, typically causing skin infections. There is currently no completely effective cure for *mycobacteriosis* in fish. Severely affected fish need to be harvested to reduce the risk of transmission to other fish and humans [50]. According to [51], a Salmon farmer from Nothern Norway had to cull 900,000 fish due to bacterial wound infections. *Abalone withering syndrome*(AWS) is a serious disease that threatens both wild and cultured abalone populations, particularly along the western coast of North America. The authors of [52] studied red abalone fishery so impacted by AWS and reported that culling infected populations is feasible for red abalone because modern diagnostic tools can detect infections without harming the abalone that is caught. Additionally, the cost of diagnostics is minimal compared to the value of the catch.

Although the literature of harvesting a population is rich enough in eco-epidemiology, however, harvesting only infected predator from a population is not investigated well. In the present work, we have studied the impact of harvesting only infected predator on an eco-epidemiological system considering linear type of prey refuge against susceptible and infected predator with Holling type I functional response . This is the novelty of our study. Moreover, infectious diseases that are transmitted between species can pose a significant threat to the entire ecosystem. They can impact biodiversity, and even push species to the brink of extinction. Thus, it is crucial and logical to develop a mathematical model including harvesting only infected predator to describe how it affects on an ecosystem.

The remainder of the paper is organized as follows: Section 2 presents the formulation of the mathematical model based on fundamental assumptions. In Section 3, we establish essential preliminary results, including the positivity and boundedness of the system. The existence of equilibria are derived in Section 4. Section 5 deals with the stability analysis of the system. In Section 6, different types of bifurcation at the points of equilibria are discussed. We illustrate our theoretical findings numerically in Section 7. Finally, we summarize our results with a brief conclusion in Section 8.

2. Model Formulation

This section introduces a mathematical framework for constructing a deterministic model of a prey–predator ecosystem that incorporates prey refuge and harvesting of infected predators.

The total population is categorized into three distinct classes: prey ($x(t)$), susceptible predators ($y(t)$),

and infected predators ($z(t)$), at time t. Without the presence of predators, the prey population increases following a logistic growth pattern, governed by a natural growth rate r and a carrying capacity k. The disease is assumed to be non-heritable in predators, with transmission occurring exclusively through direct contact between susceptible and infected classes. The spread of infection is assumed to follow a standard mass-action (bilinear) incidence mechanism and recovery of infected predators is not taken into account. Both healthy (susceptible) and infected predators feed on prey following a Holling type I functional response; however, the feeding efficiency of infected predators is considered lower than that of susceptible ones due to the impact of illness. The prey population employs a refuge strategy, reducing its exposure to predation. A fraction δ_1 of prey escapes from susceptible predators and δ_2 from infected ones, where $\delta_1, \delta_2 \in [0, 1)$. This implies that only $(1-\delta_1)x$ prey are vulnerable to susceptible predators and $(1-\delta_2)x$ to infected predators. Moreover, infected predators are removed from the system through a linear harvesting mechanism.
Based on the assumptions outlined above, the corresponding mathematical model is formulated as follows:

$$\begin{aligned}
\frac{dx}{dt} &= rx\left(1-\frac{x}{k}\right) - m(1-\delta_1)xy - n(1-\delta_2)xz \\
\frac{dy}{dt} &= e_1 m(1-\delta_1)xy - \beta yz - d_1 y \\
\frac{dz}{dt} &= e_2 n(1-\delta_2)xz + \beta yz - d_2 q_1 z
\end{aligned} \tag{2.1}$$

with initial conditions $x(0) \geq 0$, $y(0) \geq 0$, $z(0) \geq 0$. All the parameters $r, k, m, n, \delta_1, \delta_2, e_1, e_2, \beta, d_1, d_2, q_1$ are assumed to be positive constants. Their respective meanings are provided in Table 1.

Table 1: Description of Parameters used in the Model

Parameter	Description
r	Intrinsic growth rate of prey
k	Carrying capacity of prey population
m	Predation rate of susceptible predators on prey
n	Predation rate of infected predators on prey
e_1	Conversion rate from prey to susceptible predators
e_2	Conversion rate from prey to infected predators
δ_1	Prey refuge coefficient against susceptible predators
δ_2	Prey refuge coefficient against infected predators
β	Disease transmission rate among predators
d_1	Natural death rate of susceptible predators
d_2	Harvesting effort applied to infected predators
q_1	Catchability coefficient of infected predators

3. Preliminary Results

To ensure the existence and uniqueness of system (2.1), we consider the solution in

$$\mathbb{R}^3_+ = \left\{(x(t), y(t), z(t)) \in \mathbb{R}^3_+ : x(t) \geq 0, y(t) \geq 0, z(t) \geq 0\right\}.$$

Positivity and Boundness of the System

Theorem 3.1 *Each solution of system (2.1) with initial conditions are positive.*

Proof: The initial conditions of the system (2.1) are all positive, therefore at any time t, we would acuire

$$x(t) = x(0)exp\left\{\int_0^t \left(r(1-\frac{x(s)}{k}) - m(1-\delta_1)y(s) - n(1-\delta_2)z(s)\right) ds\right\} \geq 0$$
$$y(t) = y(0)exp\left\{\int_0^t (e_1 m(1-\delta_1)x(s) - \beta z(s) - d_1)\, ds\right\} \geq 0$$
$$z(t) = z(0)exp\left\{\int_0^t (e_2 n(1-\delta_2)x(s) + \beta y(s) - d_2 q_1)\, ds\right\} \geq 0$$

Hence, for all $t \geq 0$, the trajectory $(x(t), y(t), z(t))$ of system (2.1) remains in the positive orthant $\mathbb{R}^3_+$, given positive initial conditions.

□

Theorem 3.2 *The prey population are always bounded above.*

Proof: From the first equation of the system (2.1), we obtain

$$\frac{dx}{dt} \leq rx\left(1 - \frac{x}{k}\right)$$

It follows that $\lim_{t\to\infty} sup\ x \leq k$. This completes the proof of the theorem. □

Theorem 3.3 *Each solution of the system (2.1) with initial value $(x(0), y(0), z(0)) \in \mathbb{R}^3_+$ is bounded.*

Proof: Let us define the function, $w = (e_1 + e_2)x + y + z$

$$\begin{aligned}\frac{dw}{dt} &= (e_1+e_2)\frac{dx}{dt} + \frac{dy}{dt} + \frac{dz}{dt}\\ &= (e_1+e_2)rx\left(1-\frac{x}{k}\right) - m(e_1+e_2)(1-\delta_1)xy - n(e_1+e_2)(1-\delta_2)xz\\ &+ e_1 m(1-\delta_1)xy + e_2 n(1-\delta_2)xz - d_1 y - d_2 q_1 z\end{aligned}$$

$$\frac{dw}{dt} \leq (e_1+e_2)rx\left(1-\frac{x}{k}\right) - d_1 y - d_2 q_1 z \qquad \text{as } 0 \leq e_1, e_2 \leq 1$$

Now for any $v > 0$,

$$\begin{aligned}\frac{dw}{dt} + vw &\leq (e_1+e_2)rx\left(1-\frac{x}{k}\right) - d_1 y - d_2 q_1 z + (e_1+e_2)vx + vy + vz\\ &\leq (e_1+e_2)\left[rx\left(1-\frac{x}{k}\right) + vx\right] + (v-d_1)y + (v-d_2 q_1)z\end{aligned}$$

Now if we choose v in such a way that $0 < v \leq min\{d_1, d_2 q_1\}$ then we obtain

$$\begin{aligned}\frac{dw}{dt} + vw &\leq (e_1+e_2)\left[rx\left(1-\frac{x}{k}\right) + vx\right]\\ &\leq \frac{k}{4r}(e_1+e_2)(r+v)^2 = \alpha(say)\end{aligned}$$

By employing the theory of differential inequalities, we derive the following result:

$$0 < w(x,y,z) \leq \frac{\alpha}{v}(1 - e^{-vt}) + w\left(x(0), y(0), z(0)\right) e^{-vt}.$$

For $t \to \infty$,

$$0 < w < \frac{\alpha}{v}.$$

It follows that all trajectories of system (2.1) starting in $\mathbb{R}^3_+$ are confined within the region B, defined as $B = \left\{(x,y,z) \in \mathbb{R}^3_+ : w = \frac{\alpha}{v} + \varepsilon, \varepsilon > 0\right\}$.

□

4. Equilibrium Points and Existence

We now study the existence of equilibria of system (2.1). Equilibrium points mean neither of the population level is changing i.e. the derivatives $\dot{x}$,$\dot{y}$,$\dot{z}$ becomes zero.
To begin with we list all possible equilibria :

(1) The trivial equilibrium point $E_0(0,0,0)$ always exist.
(2) The predator free equilibrium point $E_1(k,0,0)$ always exist.
(3) The prey free equilibrium point $E_2\left(0, \frac{d_2 q_1}{\beta}, \frac{-d_1}{\beta}\right)$ which is biologically not feasible.
(4) The susceptible predator free equilibrium point $E_3(x_1, 0, z_1)$ biologically not feasible.
(5) The infected predator free equilibrium point $E_4(x_2, y_2, 0)$ where,

$$x_2 = \frac{d_1}{me_1(1-\delta_1)} \text{ and } y_2 = \frac{r(k-x_2)}{km(1-\delta_1)}.$$

The equilibrium point E_4 exists if $k > x_2$ i.e. $k > \dfrac{d_1}{me_1(1-\delta_1)}$.

(6) The coexistence equilibrium point is given by $E_5(x^*, y^*, z^*)$ where

$$x^* = \frac{k\beta r + knd_1(1-\delta_2) - mkd_2q_1(1-\delta_1)}{kmn(1-\delta_1)(1-\delta_2)(e_1-e_2) + r\beta},$$

$$y^* = \frac{d_2q_1 - e_2n(1-\delta_2)x^*}{\beta} \text{ and}$$

$$z^* = \frac{e_1m(1-\delta 1)x^* - d_1}{\beta}.$$

The equilibrium point E_5 exists if

(a) $e_1 > e_2$.
(b) $k\beta r + knd_1(1-\delta_2) > mkd_2q_1(1-\delta_1)$.
(c) $d_2q_1 > e_2n(1-\delta_2)x^*$ i.e. $x^* < \dfrac{d_2q_1}{e_2n(1-\delta_2)}$.
(d) $e_1m(1-\delta 1)x^* > d_1$ i.e. $x^* > \dfrac{d_1}{e_1m(1-\delta 1)}$.

5. Stability Analysis

In this section we investigate the local stability around the equilibrium points of the system (2.1). The Jacobian matrix around any equilibrium point (x, y, z) is

$$J(x,y,z) = \begin{pmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{pmatrix}$$

where $a_{11} = r - \dfrac{2rx}{k} - m(1-\delta_1)y - n(1-\delta_2)z$, $a_{12} = -m(1-\delta_1)x$, $a_{13} = -n(1-\delta_2)x$, $a_{21} = e_1m(1-\delta_1)y$, $a_{22} = e_1m(1-\delta_1)x - \beta z - d_1$, $a_{23} = -\beta y$, $a_{31} = e_2n(1-\delta_2)z$, $a_{32} = \beta z$, $a_{33} = e_2n(1-\delta_2)x + \beta y - d_2q_1$.

Stability around $E_0(0,0,0)$

The Jacobian matrix at the equilibrium point E_0 is

$$J_0 = \begin{pmatrix} r & 0 & 0 \\ 0 & -d_1 & 0 \\ 0 & 0 & -d_2q_1 \end{pmatrix}$$

The eigenvalues of J_0 are r, $-d_1$, $-d_2q_1$. Hence the system (2.1) is unstable around E_0 as one eigenvalue of J_0 is always positive. This instability implies that the system cannot settle into a state of complete extinction. Biologically, it signifies that if a small prey population exists, it can regenerate, leading to the eventual revival of the predator populations as well. This reflects the inherent resilience of ecosystems and their ability to recover when essential components, like prey availability and reproductive potential, are maintained.

Stability around $E_1(k,0,0)$

The Jacobian matrix at the equilibrium point E_1 is

$$J_1 = \begin{pmatrix} -r & -m(1-\delta_1)k & -n(1-\delta_2)k \\ 0 & e_1mk(1-\delta_1)-d_1 & 0 \\ 0 & 0 & e_2nk(1-\delta_2)-d_2q_1 \end{pmatrix}$$

The eigenvalues of J_1 are $-r$, $e_1mk(1-\delta_1)-d_1$, $e_2nk(1-\delta_2)-d_2q_1$. So, E_1 is locally asymptotically stable if $k < min\left\{\dfrac{d_1}{e_1mk(1-\delta_1)}, \dfrac{d_2q_1}{e_2nk(1-\delta_2)}\right\}$, otherwise it is unstable.

Remark: Since The equilibrium points E_2 and E_3 are not biologically feasible so we will not discuss about their stability.

Stability around $E_4(x_2,y_2,0)$

The Jacobian matrix at the equilibrium point E_4 is

$$J_4 = \begin{pmatrix} b_{11} & b_{12} & b_{13} \\ b_{21} & 0 & b_{23} \\ 0 & 0 & b_{33} \end{pmatrix} \tag{5.1}$$

$$= \begin{pmatrix} \dfrac{-rx_2}{k} & -\dfrac{d_1}{e_1} & -n(1-\delta_2)x_2 \\ \dfrac{e_1r(k-x_2)}{k} & 0 & -\beta y_2 \\ 0 & 0 & e_2n(1-\delta_2)x_2+\beta y_2-d_2q_1 \end{pmatrix}$$

One eigenvalue of J_4 is $e_2n(1-\delta_2)x_2+\beta y_2-d_2q_1$. The other two eigenvalues are the roots of the characteristics equation $\lambda^2+b_1\lambda+b_2=0$, where $b_1=\dfrac{rx_2}{k}>0$ and $b_2=\dfrac{d_1r(k-x_2)}{k}$.
Therefore, the equilibrium point E_4 is locally asymptotically stable if

$$e_2n(1-\delta_2)x_2+\beta y_2 < d_2q_1 \text{i.e.} \frac{e_2n(1-\delta_2)x_2+\beta y_2}{d_2q_1} < 1 \quad \text{and} \quad k > x_2. \tag{5.2}$$

Stability around $E_5(x^*,y^*,z^*)$

The Jacobian matrix J_5 for the equilibrium point E_5 is given as follows:

$$J_5 = \begin{pmatrix} p_{11} & p_{12} & p_{13} \\ p_{21} & p_{22} & p_{23} \\ p_{31} & p_{32} & p_{33} \end{pmatrix}$$

where, $p_{11} = r - \dfrac{2rx^*}{k} - m(1-\delta_1)y^* - n(1-\delta_2)z^*$, $\quad p_{12} = -m(1-\delta_1)x^*$,
$p_{13} = -n(1-\delta_2)x^*$, $\quad p_{21} = e_1m(1-\delta_1)y^*$,
$p_{22} = e_1m(1-\delta_1)x^* - \beta z^* - d_1$, $\quad p_{23} = -\beta y^*$,
$p_{31} = e_2n(1-\delta_2)z^*$, $\quad p_{32} = \beta z^*$,
$p_{33} = e_2n(1-\delta_2)x^* + \beta y^* - d_2q_1$.
The characteristics equation of the Jacobian matrix J_5 around the equilibrium point E_5 is

$$\lambda^3 + c_0\lambda^2 + c_1\lambda + c_2 = 0 \tag{5.3}$$

where, $c_0 = -p_{11} - p_{22} - p_{33}$,
$c_1 = p_{11}p_{22} + p_{22}p_{33} + p_{11}p_{33} - p_{12}p_{21} - p_{13}p_{31} - p_{23}p_{32}$,
$c_2 = p_{11}p_{23}p_{32} + p_{12}p_{21}p_{33} + p_{13}p_{31}p_{22} - p_{11}p_{22}p_{33} - p_{12}p_{31}p_{23} - p_{13}p_{21}p_{32}$.
Thus by Routh-Hurwitz stability criterion the coexistence equilibrium point E_5 is locally asymptotically stable if $c_0 > 0$, $c_2 > 0$ and $c_0c_1 - c_2 > 0$.

6. Global Stability

Through this section, we discuss the global asymptotic stability (GAS) of the equilibrium points using the Lyapunov function whenever it exists as described in the following theorems.

Theorem 6.1 *If $E_1(k,0,0)$ is locally asymptotically stable then it is GAS whenever the condition $k < min\{x(1-e_1), x(1-e_2)\}$ hold.*

Proof: We took a positive definite real-valued function around E_1 as

$$L_1 = (x - k - ln\frac{x}{k}) + y + z$$

Then, we have

$$\begin{aligned}\frac{dL_1}{dt} &= \left(\frac{x-k}{x}\right)\frac{dx}{dt} + \frac{dy}{dt} + \frac{dz}{dt}\\ &= \left(\frac{x-k}{x}\right)\left\{rx\left(1-\frac{x}{k}\right) - m(1-\delta_1)xy - n(1-\delta_2)xz\right\} + e_1m(1-\delta_1)xy - \beta yz - d_1y\\ &\quad + e_2n(1-\delta_2)xz + \beta yz - d_2q_1z\\ &= -\frac{1}{k}r(x-k)^2 - my(1-\delta_1)\{(x-k) - e_1x\} - nz(1-\delta_2)\{(x-k) - e_2x\} - (d_1y + d_2q_1z)\end{aligned}$$

Thus $\frac{dL_1}{dt} < 0$ when $k < min\{x(1-e_1), x(1-e_2)\}$.

Hence, system (2.1) is globally asymptotically stable around E_1. □

Theorem 6.2 *Let $L_2 = \frac{(x-x^*)^2}{2} + \alpha_1\frac{(y-y^*)^2}{2} + \alpha_2\frac{(z-z^*)^2}{2}$ where $\alpha_1, \alpha_2 > 0$ are to be chosen such that $L_2'(E_5) = 0$ where $E_5(x^*, y^*, z^*)$. The time derivative of L_2 is $\frac{dL_2}{dt} \leq 0$, $\forall x, y, z \in \mathbb{R}^+$. It then follows that $\frac{dL_2}{dt} = 0$, $\forall x^*, y^*, z^* \in \mathbb{R}^+$ implies that E_5 of the system is Lyapunov stable and $\frac{dL_2}{dt} < 0$ $\forall x, y, z \in \mathbb{R}^+$ near E_5 implies that E_5 is globally stable.*

Proof: We have, $L_2 = \frac{(x-x^*)^2}{2} + \alpha_1\frac{(y-y^*)^2}{2} + \alpha_2\frac{(z-z^*)^2}{2}$

Taking time derivative of the above equation, we get

$$\frac{dL_2}{dt} = (x-x^*)\frac{dx}{dt} + \alpha_1(y-y^*)\frac{dy}{dt} + \alpha_2(z-z^*)\frac{dz}{dt}.$$

Now, substituting the value of $\frac{dx}{dt}, \frac{dy}{dt}$ and $\frac{dz}{dt}$ from the system (2.1) and putting these values in the above equation, we get

$$\begin{aligned}\frac{dL_2}{dt} &= (x-x^*)\left\{rx\left(1-\frac{x}{k}\right) - m(1-\delta_1)xy - n(1-\delta_2)xz\right\}\\ &\quad +\alpha_1(y-y^*)\{e_1m(1-\delta_1)xy - \beta yz - d_1y\}\\ &\quad +\alpha_2(z-z^*)\{e_2n(1-\delta_2)xz + \beta yz - d_2q_1z\}\\ &= (x-x^*)\left[\left\{r\left(1-\frac{x}{k}\right) - m(1-\delta_1)y - n(1-\delta_2)z\right\}(x-x^*)\right]\\ &\quad +\alpha_1(y-y^*)\left[\{e_1m(1-\delta_1)x - \beta z - d_1\}(y-y^*)\right]\\ &\quad +\alpha_2(z-z^*)\left[\{e_2n(1-\delta_2)x + \beta y - d_2q_1\}(z-z^*)\right]\end{aligned}$$

By rearranging, we obtain

$$\begin{aligned}&= -(x-x^*)^2\left\{r\left(\frac{x}{k}-1\right) + m(1-\delta_1)y + n(1-\delta_2)z\right\}\\ &\quad -\alpha_1(y-y^*)^2\{-e_1m(1-\delta_1)x + \beta z + d_1\}\\ &\quad -\alpha_2(z-z^*)^2\{-e_2n(1-\delta_2)x - \beta y + d_2q_1\}\end{aligned}$$

Thus it is possible to set α_1, α_2 such that $\frac{dL_2}{dt} \leq 0$ and the equilibrium point E_5 is globally asymptotically stable. □

7. Bifurcation Analysis

Bifurcation in a dynamical system signifies a qualitative change in system behavior triggered by the variation of a key parameter. Such transitions can lead to the emergence or disappearance of equilibria or oscillatory dynamics. In the context of ecological systems, bifurcations may reflect critical shifts in population stability, coexistence, or extinction scenarios. In this section, we examine the bifurcation phenomena that may occur in our model and highlight their biological relevance to the stability and persistence of interacting species.

Hopf Bifurcation

Theorem 7.1 *The system* (2.1) *undergoes a Hopf bifurcation around the coexistence equilibrium point when the rate of disease transmission* (β) *exceeds a critical value. The Hopf bifurcation occurs at* $\beta = \beta^*$ *if and only if the following conditions hold.*

1. $c_0(\beta^*)c_1(\beta^*) - c_2(\beta^*) = 0$
2. $\dfrac{d}{d\beta}\left(Re(\lambda(\beta))\right)|_{\beta=\beta^*} \neq 0$, *transversality condition.*

where λ *is the root of the characteristic equation* $\lambda^3 + c_0\lambda^2 + c_1\lambda + c_2 = 0$, *where* c_0, c_1, c_2 *are mentioned in the equation (5.3).*

Proof: Let $\beta = \beta^*$, then the characteristic equation $\lambda^3 + c_0\lambda^2 + c_1\lambda + c_2 = 0$ turns into

$$(\lambda^2 + c_1)(\lambda + c_0) = 0.$$

Clearly, the roots of the above equation are $\lambda_1 = i\sqrt{c_1}$, $\lambda_2 = -i\sqrt{c_1}$ and $\lambda_3 = -c_0$.
Therefore there exists a strictly negative eigenvalue and a pair of purely imaginary eigenvalues. We can rewrite the roots considering β as bifurcation parameter as follows

$$\begin{aligned}\lambda_1(\beta) &= b_1(\beta) + ib_2(\beta),\\ \lambda_2(\beta) &= b_1(\beta) - ib_2(\beta),\\ \lambda_3(\beta) &= -c_0(\beta)\end{aligned}$$

Substituting $\lambda_i(\beta) = b_1(\beta) + ib_2(\beta)$ into the above characteristics equation and then differentiating with respect to β and separating real and imaginary parts, we obtain

$$\begin{aligned}M(\beta){b_1}'(\beta) - N(\beta){b_2}'(\beta) + R(\beta) = 0,\\ N(\beta){b_1}'(\beta) + M(\beta){b_2}'(\beta) + S(\beta) = 0\end{aligned}$$

where,

$$\begin{aligned}M(\beta) &= 3b_1^2(\beta) + 2c_0(\beta)b_1(\beta) + c_1(\beta) - 3b_2^2(\beta),\\ N(\beta) &= 6b_1(\beta)b_2(\beta) + 2c_0(\beta)b_2(\beta),\\ R(\beta) &= {c_0}'(\beta)b_1^2(\beta) + {c_1}'(\beta)b_1(\beta) + {c_2}'(\beta) - {c_0}'(\beta)b_2^2(\beta),\\ S(\beta) &= 2{c_0}'(\beta)b_1(\beta)b_2(\beta) + {c_1}'(\beta)b_2(\beta)\end{aligned}$$

At $\beta = \beta^*$, we have $b_1(\beta^*) = 0$ and $b_2(\beta^*) = \sqrt{c_1(\beta^*)}$. Then,

$$\begin{aligned}M(\beta^*) &= -2c_1(\beta^*),\\ N(\beta^*) &= 2c_0(\beta^*)\sqrt{c_1(\beta^*)},\\ R(\beta^*) &= {c_2}'(\beta^*) - {c_0}'(\beta^*)c_1(\beta^*),\\ S(\beta^*) &= {c_1}'(\beta^*)\sqrt{c_1(\beta^*)}\end{aligned}$$

Now,

$$\begin{aligned}\frac{d}{d\beta}\left(Re(\lambda(\beta))\right)|_{\beta=\beta^*} &= -\frac{M(\beta^*)R(\beta^*)+N(\beta^*)S(\beta^*)}{M^2(\beta^*)+N^2(\beta^*)}\\ &= \frac{{c_2}'(\beta^*)-{c_0}'(\beta^*)c_1(\beta^*)-c_0(\beta^*){c_1}'(\beta^*)}{2\{c_1(\beta^*)+c_0^2(\beta^*)\}}\\ &\neq 0, if {c_2}'(\beta^*)-{c_0}'(\beta^*)c_1(\beta^*)-c_0(\beta^*){c_1}'(\beta^*)\neq 0\end{aligned}$$

and $\lambda_3(\beta^*) = -c_0(\beta^*) \neq 0$. So, the transversality condition holds and the system (2.1) exhibits Hopf bifurcation at $\beta = \beta^*$. Hence the theorem.

□

Theorem 7.2 *The system (2.1) undergoes a Hopf bifurcation around the coexistence equilibrium point when the prey refuge coefficient δ_1 against susceptible predator passese through a critical value. The Hopf bifurcation occurs at $\delta_1 = \delta_1^*$ if and only if the following conditions hold.*

1. $c_0(\delta_1^*)c_1(\delta_1^*) - c_2(\delta_1^*) = 0$
2. $\frac{d}{d\delta_1}\left(Re(\lambda(\delta_1))\right)|_{\delta_1=\delta_1^*} \neq 0$ *, transversality condition.*

where λ is the root of the characteristic equation given in (5.3).

Proof: The proof is very similar to the Theorem (7.1). Hence the proof is omitted. □

Theorem 7.3 *The system governed by the model (2.1) exihibits a Hopf bifurcation near the coexistence equilibrium point as the catchability co-efficient q_1 of the infected predator exceeds the critical value q_1^*. The Hopf bifurcation occurs at $q_1 = q_1^*$ if and only if the following conditions hold.*

1. $c_0(q_1^*)c_1(q_1^*) - c_2(q_1^*) = 0$
2. $\frac{d}{dq_1}\left(Re(\lambda(q_1))\right)|_{q_1=q_1^*} \neq 0$ *, transversality condition.*

where λ is the root of the characteristic equation given in (5.3).

Proof: The proof is very similar to the Theorem (7.1). Hence the proof is omitted. □

Transcritical Bifurcation

We have used Sotomayor's Theorem [53] to observe the transcritical bifurcation of the system (2.1). We rewrite the system (2.1) in the form :

$$\frac{dX}{dt} = F(X)$$

where $X = (x, y, z)^T$ and $F = (f_1, f_2, f_3)^T$ with $f_1 = rx\left(1-\frac{x}{k}\right) - m(1-\delta_1)xy - n(1-\delta_2)xz$, $f_2 = e_1m(1-\delta_1)xy - \beta yz - d_1y$, $f_3 = e_2n(1-\delta_2)xz + \beta yz - d_2q_1z$.

Theorem 7.4 *Assume that condition (5.2) holds then the system (2.1) posseses a transcritical bifurcation around the infected predator free equilibrium point $E_4(x_2, y_2, 0)$ when the catchability co-efficient q_1 of the infected predator exceeds the critical value $q_1 = q_1^{[tc]} = \frac{e_2n(1-\delta_2)x_2+\beta y_2}{d_2}$.*

Proof: The jacobian matrix of the system at the equilibrium point $E_4(x_2, y_2, 0)$ is as given in (5.1). At $q_1 = q_1^{[tc]}$ the system has the following jacobian matrix, say $J_4(E_4, q_1^{[tc]}) = \tilde{J}$,which has a zero eigenvalue.

$$\tilde{J} = \begin{pmatrix} b_{11} & b_{12} & b_{13} \\ b_{21} & 0 & b_{23} \\ 0 & 0 & 0 \end{pmatrix} \text{ where } b_{ij}; \forall i,j = 1,2,3 \text{ are given in (5.1)}$$

Now, let $U = (u_1, u_2, u_3)^T = (m_1 u_2, u_2, m_2 u_2)^T$ where, $m_1 = \dfrac{b_{23}b_{12}}{b_{13}b_{21} - b_{11}b_{23}} < 0$, $m_2 = -\dfrac{b_{21}b_{12}}{b_{13}b_{21} - b_{11}b_{23}} > 0$ and u_2 is any non zero real number, be the eigenvector corresponding to the zero eigenvalue of the matrix $\tilde{J}$ and $V = (v_1, v_2, v_3)^T = (0, 0, v_3)^T$, where v_3 is any non zero real number, represents the eigenvector corresponding to the zero eigenvalue of the matrix $(\tilde{J})^T$. Now because we have

$$\frac{\partial F}{\partial q_1} = F_{q_1}(X, q_1) = (0, 0, -d_2 z)^T$$

Thus, $F_{q_1}(E_4, q_1^{[tc]}) = (0, 0, 0)^T$, which gives $V^T F_{q_1}(E_4, q_1^{[tc]}) = 0$. So according to Sotomayor's theorem for local bifurcation, system (2.1) has no saddle-node bifurcation at $q_1 = q_1^{[tc]}$. Furthermore because we have

$$DF_{q_1}(E_4, q_1^{[tc]}) = \begin{pmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & -d_2 \end{pmatrix}$$

We can show that

$$V^T(DF_{q_1}(E_4, q_1^{[tc]})U) = (0, 0, v_3)(0, 0, -m_2 u_2 d_2)^T = -m_2 u_2 d_2 v_3 \neq 0.$$

Again,

$$D^2F(E_4, q_1^{[tc]})(U, U) = \begin{pmatrix} -\frac{2r}{k} m_1^2 u_2^2 - 2m m_1 (1-\delta_1) u_2^2 - 2n m_1 m_2 (1-\delta_2) u_2^2 \\ 2e_1 m m_1 (1-\delta_1) u_2^2 - 2\beta m_2 u_2^2 \\ 2e_2 n m_1 m_2 (1-\delta_2) u_2^2 + 2\beta m_2 u_2^2 \end{pmatrix}$$

Hence, it is obtained that

$$V^T[D^2F(E_4, q_1^{[tc]})(U, U)] = 2[e_2 n(1-\delta_2) m_1 + \beta] m_2 u_2^2 v_3 \neq 0.$$

Therefore, by Sotomayor's theorem, system (2.1) at infected predator free equilibrium point has a transcritical bifurcation as the parameter q_1 passes through the bifurcation value $q_1^{[tc]}$, while Pitchfork bifurcation cannot occurs and hence the proof is complete.

□

Theorem 7.5 *Assume that condition (5.2) holds then the system (2.1) undergoes a transcritical bifurcation around the infected predator free equilibrium point $E_4(x_2, y_2, 0)$ when the rate of disease transmission β passes through a critical point $\beta = \beta^{[tc]} = \dfrac{d_2 q_1 - e_2 n(1-\delta_2) x_2}{y_2}$.*

Proof: The proof can be readily derived from the proof of Theorem (7.4) and is therefore omitted. □

Theorem 7.6 *Suppose condition (5.2) holds then the system (2.1) exihibits a transcritical bifurcation around the infected predator free equilibrium point $E_4(x_2, y_2, 0)$ when the prey refuge coefficient δ_2 against infected predator passes through a critical value $\delta_2 = \delta_2^{[tc]} = 1 - \dfrac{d_2 q_1 - \beta y_2}{e_2 n x_2}$.*

Proof: The proof can be readily derived from the proof of Theorem (7.4) and is therefore omitted. □

Theorem 7.7 *Assume that condition (5.2) holds then the system (2.1) shows a transcritical bifurcation around the infected predator free equilibrium point $E_4(x_2, y_2, 0)$ when the carrying capacity of prey exceeds the critical value $k = k^{[tc]} = \dfrac{r\beta x_2}{r\beta + m(1-\delta_1)[e_2 n(1-\delta_2) x_2 - d_2 q_1]}$.*

Proof: The proof can be readily derived from the proof of Theorem (7.4) and is therefore omitted. □

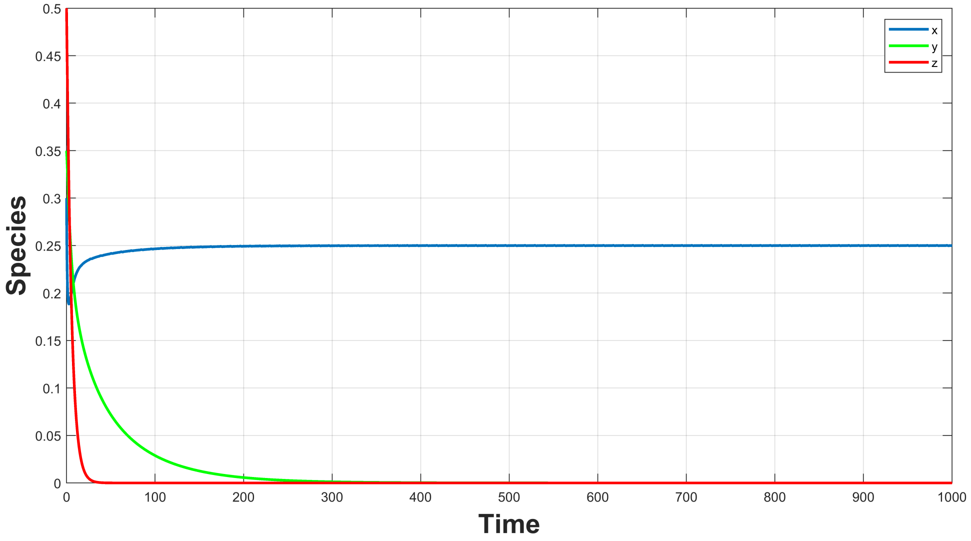


Figure 1: Stability behavior of the model system (1) around E_1 with k=0.25.

8. Numerical Simulation

To demonstrate our theoretical findings, numerical simulations are conducted in this section. For convenience, we choose the following biologically feasible parameter values of system (2.1):

Table 2: Parameter values for numerical simulation

Parameters	Description of parameters	Default value
r	Intrinsic growth rate of prey	1.2
k	Carrying capacity of prey population	15
m	Predation rate of susceptible predators on prey	0.57
n	Predation rate of infected predators on prey	0.45
e_1	Conversion rate from prey to susceptible predators	0.95
e_2	Conversion rate from prey to infected predators	0.258
δ_1	Prey refuge coefficient against susceptible predators	0.02
δ_2	Prey refuge coefficient against infected predators	0.03
β	Disease transmission rate among predators	0.095
d_1	Natural death rate of susceptible predators	0.148
d_2	Harvesting effort applied to infected predators	0.628
q_1	Catchability coefficient of infected predators	0.364

For these parameter values we see, $\dfrac{d_1}{e_1 m(1-\delta_1)} = 0.278893$ and $\dfrac{d_2 q_1}{e_2 n(1-\delta_2)} = 2.02982$. It is observed that for $k = 0.25 < min\,\{0.278893, 2.02982\}$ together with other parameters as mentioned above, the system 2.1 has a predator-free equilibrium point $E_1(0.25, 0, 0)$ which is locally asymptotically stable (Figure 1).

For the same set of parameters, the system (2.1) has a unique coexistence equilibrium point $E_5(0.288724, 2.06397, 0.054915)$. At this point, $mkd_2q_1(1-\delta_1) = 1.91537 < k\beta r + knd_1(1-\delta_2) = 2.67903$ and $\dfrac{d_1}{e_1 m(1-\delta_1)} < x^* < \dfrac{d_2 q_1}{e_2 n(1-\delta_2)}$, implying that all the conditions for the existence of equilibrium point E_5 are satisfied. Also, the conditions of Routh-Hurwitz criteria hold $c_0 = 0.0230979 > 0, c_1 = 0.178451, c_2 = 0.000548182 > 0$ and $c_0c_1 - c_2 = 0.00357366 > 0$ which confirms locally asymptotically stable behavior of the coexistence equilibrium point. Figure 2, represents the time series solution and the phase portrait of the system (2.1).

The impact of β on the stability of E_5

The disease transmission rate β has crucial role for the behaviour of the system around E_5. Initiating from the interior equilibrium point $E_5(0.288724, 2.06397, 0.054915)$ with free parameter β we computed the curve of interior equilibria. It is observed that, at $\beta = \beta^*(\approx 0.109746)$, the interior equilibrium shows oscillatory behavior via a Hopf bifurcation where the state space is $E_5(0.3864, 1.6862, 0.5203)$. We also varified the transversality condition at the bifurcation point β^* and observe that $\frac{d}{d\beta}\left(Re(\lambda(\beta))\right)|_{\beta=\beta^*} = -0.0131267 \neq 0$. The numerically computed first Lyapunov coefficient at $\beta = \beta^*$ $(l_1 = -1.629292 \times -03)$ confirms the supercritical natures of the periodic oscillations. In the ecological context, when the disease transmission rate β crosses a threshold value β^*, the population of the system starts oscillating with small and stable periodic solutions. Figure 4 shows all species coexist with oscillatory behaviour for $\beta = 0.115$. The corresponding bifurcation diagram is shown in Figure 3-(a). Small changes in disease transmission might push the system into oscillations, but these oscillations tend to settle into a stable cycle, rather than leading to the collapse or explosion of one population. A similar scenario is also observed for increasing the rate of disease transmission through a threshold value $\beta = \beta^* \approx 0.302538$ (Figure 3-(b)). In Figure 5 we plotted the time series and phase portrait diagram for $\beta = 0.295$, which represents the oscillatory nature of the system.

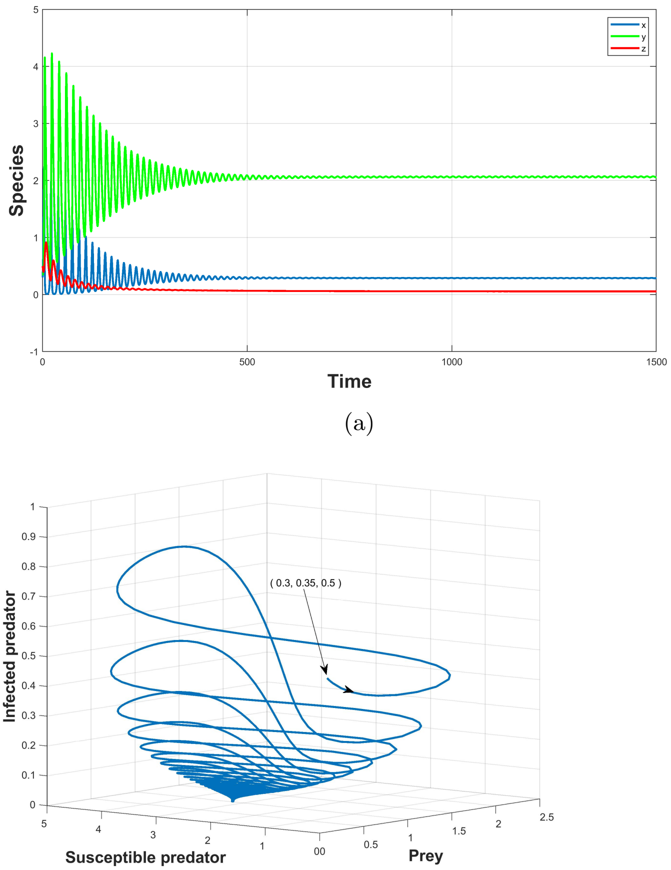
The impact of δ_1

Another important parameter is prey refuge coefficient δ_1 against the susceptible predator. Initiating from the interior equilibrium point E_5, we computed the curve of equilibria by varying δ_1. At $\delta_1 = \delta_1^* \approx 0.160768$, the interior equilibrium loses its stability producing stable limit cycles via a supercritical Hopf bifurcation. The numerically computed first Lyapunov coefficient at $\delta_1 = \delta_1^*$ is $l_1 = -1.402447 \times 10^{-03}$. Figure 6 represents time series and phase portrait diagram for $\delta_1 = 0.18$. On further increasing, at $\delta_1 = \delta_1^* \approx 0.482042$, the interior equilibrium again undergoes another supercritical Hopf bifurcation where the first Lyapunov coefficient at $\delta_1 = \delta_1^*$ is $l_1 = -1.103770 \times 10^{-03}$. For $\delta_1 = 0.475$ the system shows oscillatory behavior (Figure 7). The bifurcation diagram for δ_1 is shown in Figure 8.

The impact of q_1

Next, we analyse the effect of the catchability coefficient q_1 on the interior equilibrium state. As the parameter q_1 increases, the interior equilibrium experiences a supercritical Hopf bifurcation at the critical value $q_1 = q_1^* \approx 0.314036$. For $q_1 = 0.305$ we see the system oscillates around the coexistence equilibrium point from Figure 9. On further decreasing q_1, the interior equilibrium again undergoes a supercritical Hopf bifurcation at $q_1 = q_1^* \approx 0.167621$. The corresponding First Lyapunov coefficients at these points are respectively $l_1 = -1.420644 \times 10^{-03}$ and $l_1 = -5.449888 \times 10^{-03}$. Figure 10 shows the system's oscillation for $q_1 = 0.177$. The corresponding bifurcation diagram for q_1 is shown in the Figure 11.

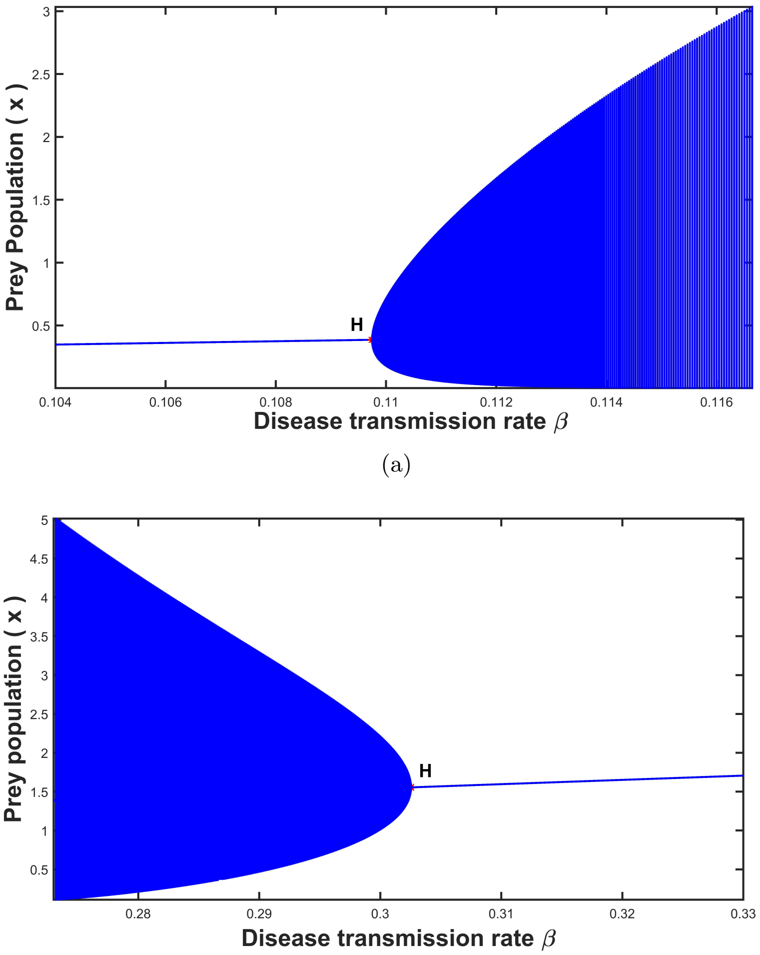
Next, we discuss an important behavior of the system, stability changes from a coexistence equilibrium point to an infected predator-free equilibrium point. At $q_1 = q_1^{BP} = 0.368941$, the coexistence equilibrium transitions to an infected predator-free equilibrium, $E_4(0.278893, 2.108286, 0)$. At this critical value, two distinct infected predator-free equilibria arise, each exhibiting opposite stability properties (Figure 12). Again, at $k = k^{BP} = 8.251581$, the coexistence equilibrium transitions to an infected predator-free equilibrium, $E_4(0.278893, 2.075620, 0)$. At this critical value, two distinct infected predator-free equilibria arise, each exhibiting opposite stability properties (Figure 13). The coexistence equilibrium point switches it's behavior to infected predator-free equilibrium, $E_4(0.278893, 2.108286, 0)$ at $\beta = \beta^{BP} = 0.093528$. Figure 14 depicts when β exceeds the critical value β^{BP} the infected predator free equilibrium point becomes unstable from it's stable behavior. Again from figure 15 we observe that at $\delta_2 = \delta_2^{BP} = 0.125840$ two distinct infected predator-free equilibia emerge, each showing opposite stability properties. At this critical point the coexistence equilibria shifts to the infected predator free equilibrium $E_4(0.278893, 2.108286, 0)$.



(a)

(b)

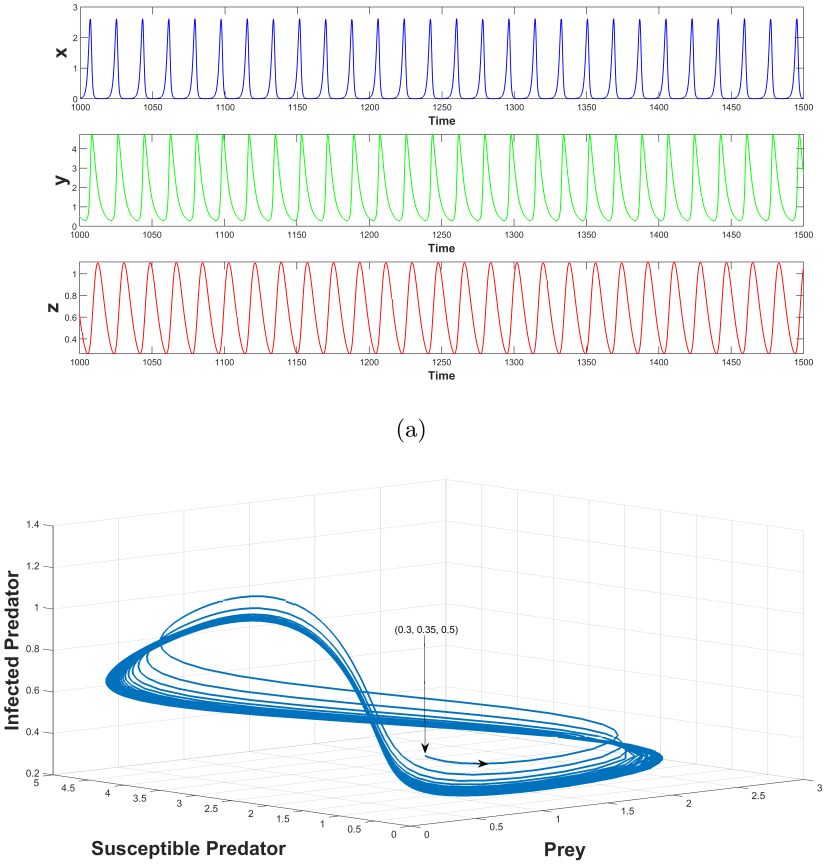
Figure 2: Stability of the system (1) near the co-existence equilibrium point E_5. (a) time series solution, (b) phase portrait diagram.



(a)

(b)

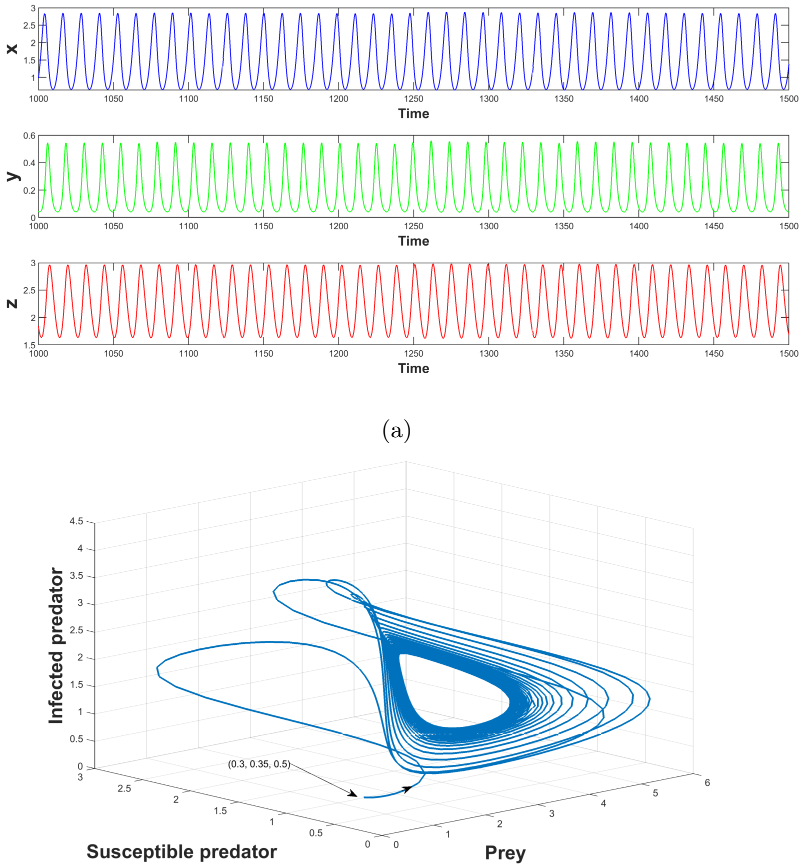
Figure 3: Emergence of stable periodic solutions of the system (2.1) at the interior equilibrium at (a) $\beta = \beta^{*}(\approx 0.109746)$, (b) $\beta = \beta^{*}(\approx 0.302538)$



(a)

(b)

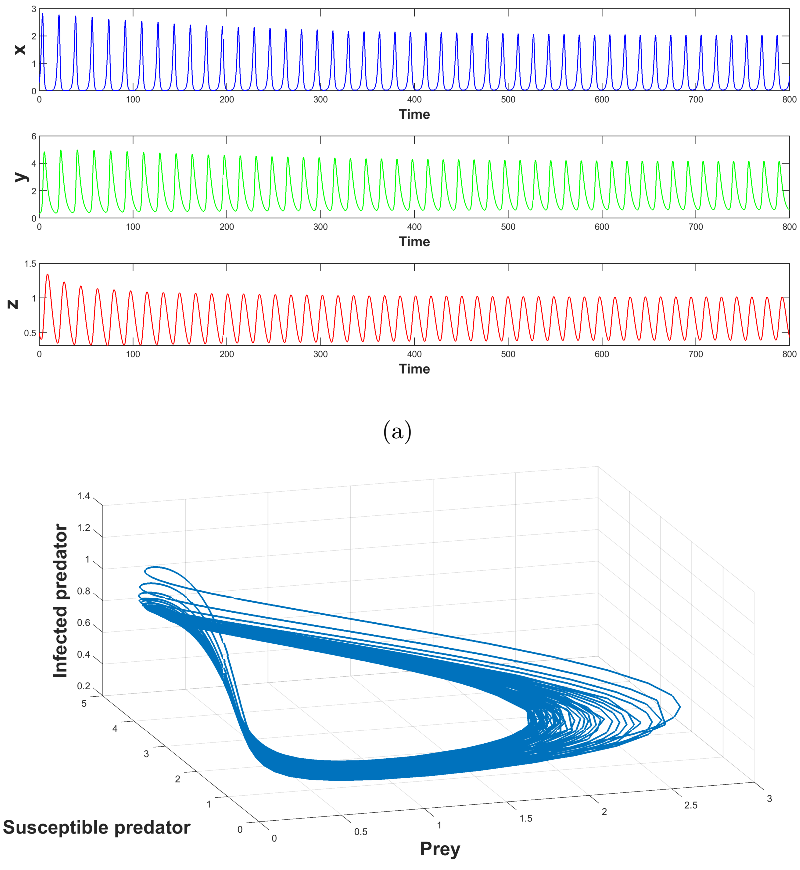
Figure 4: Figure (a) and (b) shows time series and phase portrait of Hopf bifurcation around the co-existence equilibrium point E_5 of the system (1) when $\beta = 0.115$.



(a)

(b)

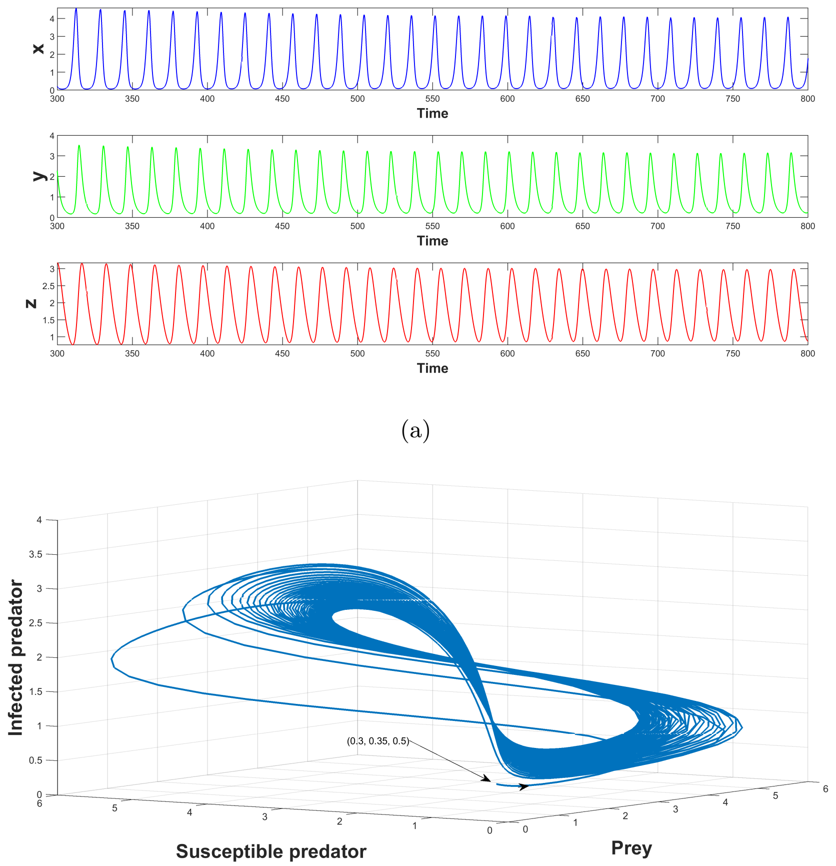
Figure 5: Hopf bifurcation around the co-existence equilibrium point E_5 of the system (1)when $\beta = 0.295$, (a) time series solution ,(b) phase portrait



(a)

(b)

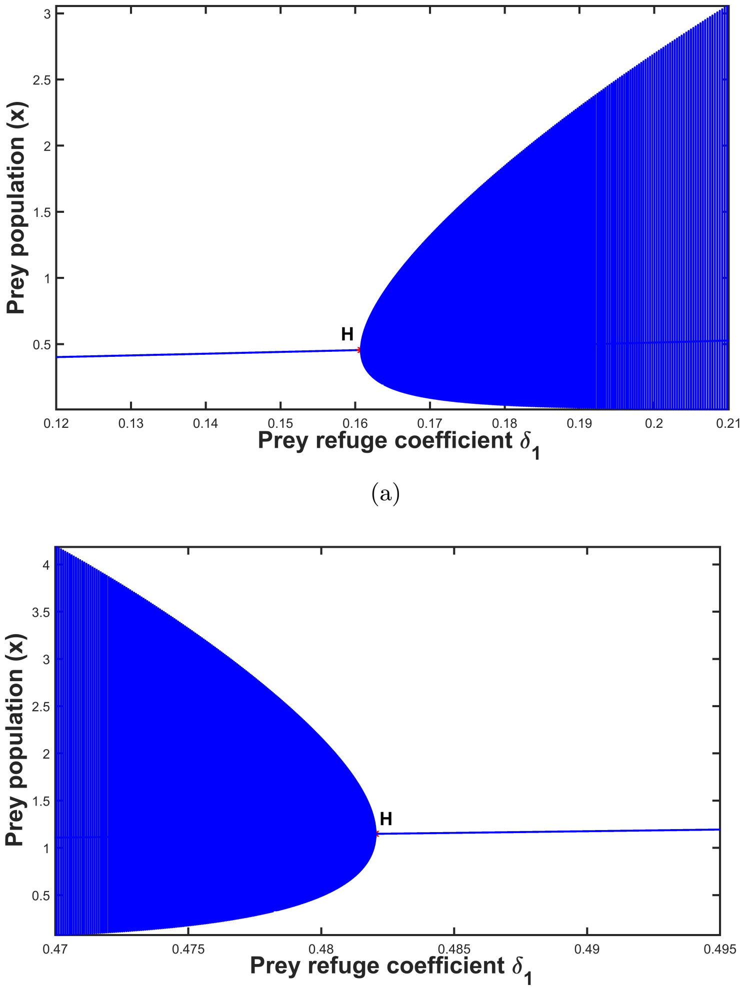
Figure 6: Figure shows limit cycle oscillation when $\delta_1 = 0.18$.(a)time series solution ,(b)phase portrait diagram.



(a)

(b)

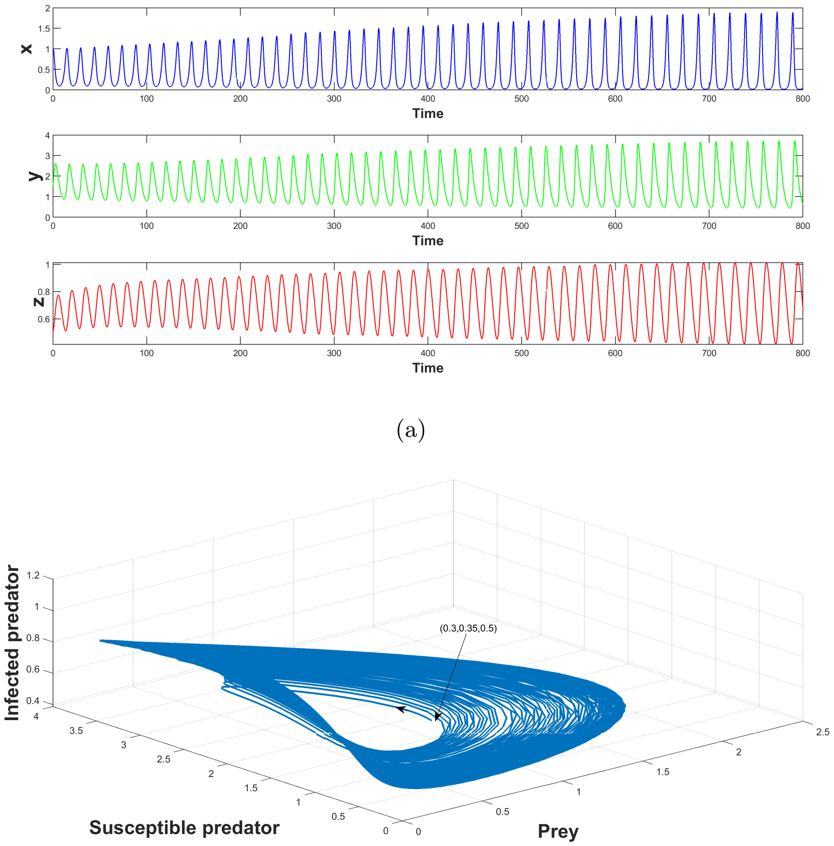
Figure 7: Bifurcation behavior around the co-existence equilibrium point E_5 of the system (1)when $\delta_1 = 0.475$.(a)time series solution ,(b)phase portrait



(a)

(b)

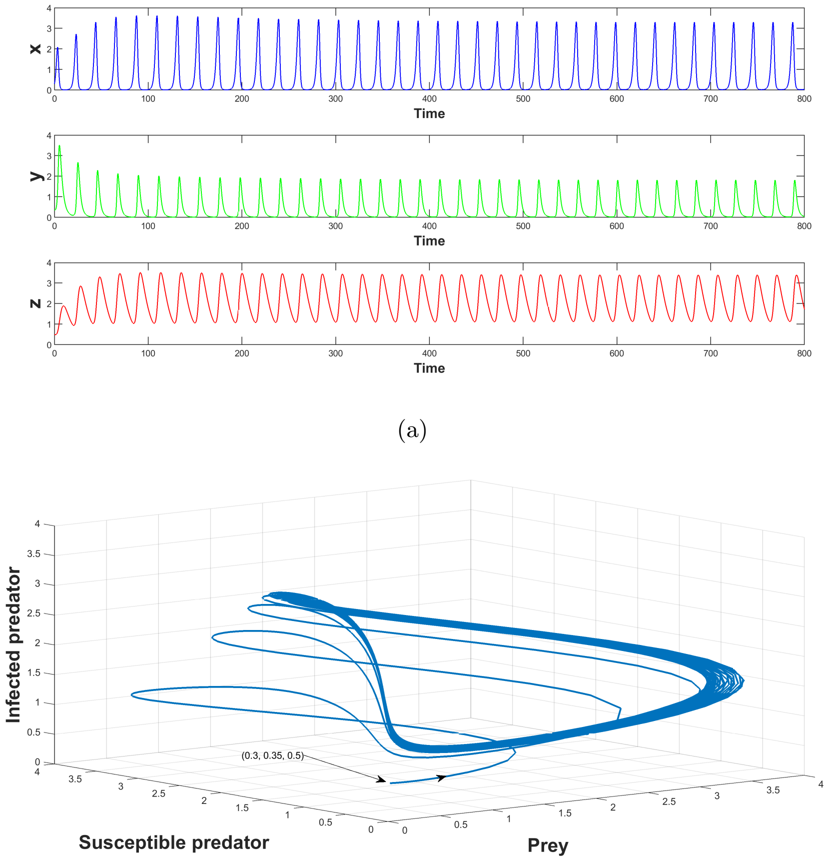
Figure 8: Emergence of stable periodic solutions of the system (2.1) at the interior equilibrium at (a) $\delta_1 = \delta_1^*(\approx 0.160768)$, (b) $\delta_1 = \delta_1^*(\approx 0.482042)$



(a)

(b)

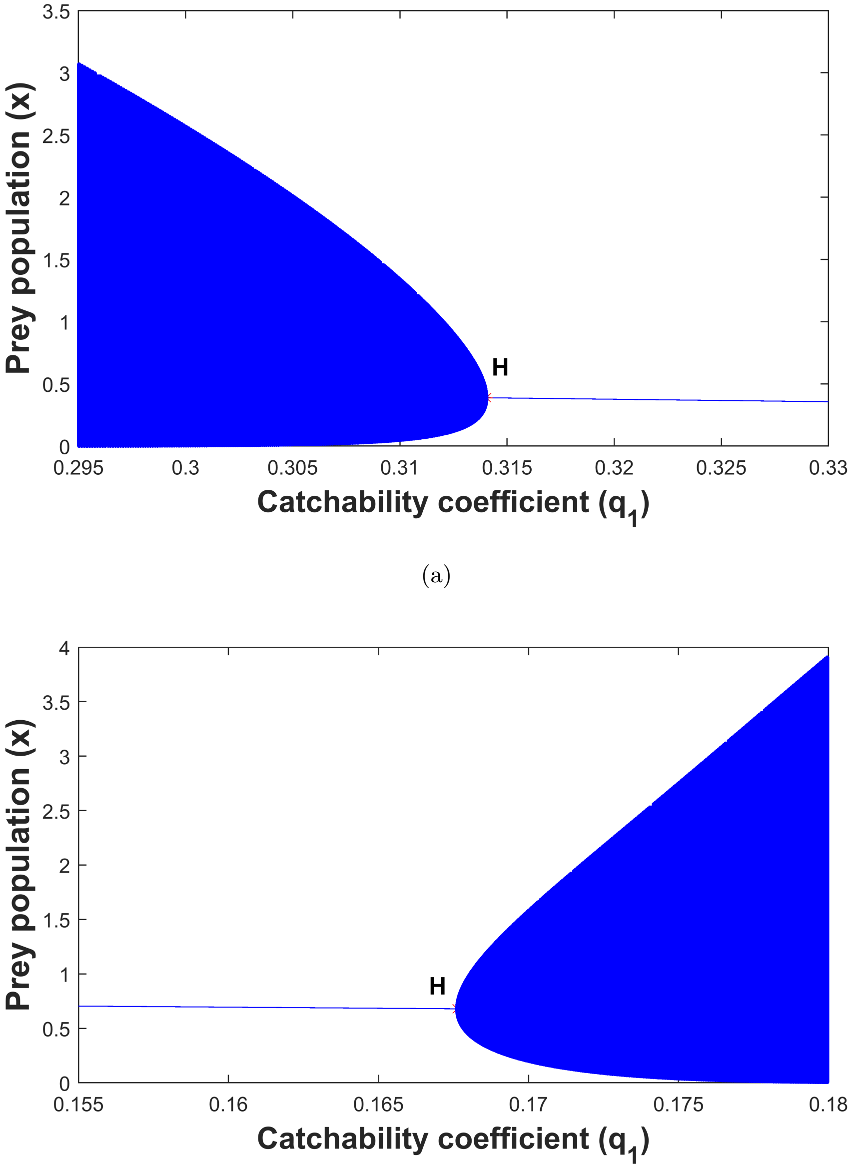
Figure 9: Hopf bifurcation behavior around the co-existence equilibrium point E_5 of the system (1) when $q_1 = 0.305$. (a)time series solution ,(b) phase portrait



(a)

(b)

Figure 10: Figure shows limit cycle oscillations when $q_1 = 0.177$.(a) time series solution,(b) phase portrait diagram.



(a)

(b)

Figure 11: Emergence of stable periodic solutions of the system (2.1) at the interior equilibrium at (a)$q_1 = q^*(\approx 0.314036)$, (b) $q_1 = q^*(\approx 0.167621)$

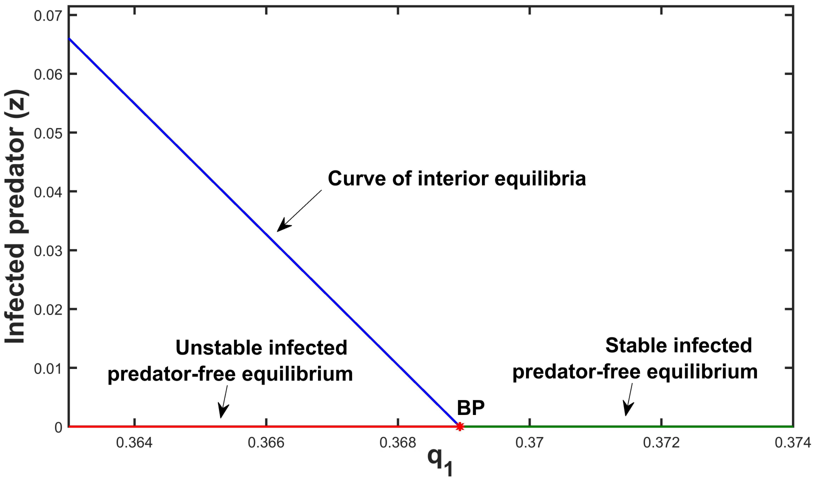


Figure 12: Transcritical bifurcation of infected predator-free equilibrium for infected predator population with respect to $q_1 = q_1^{BP} = 0.368941$.

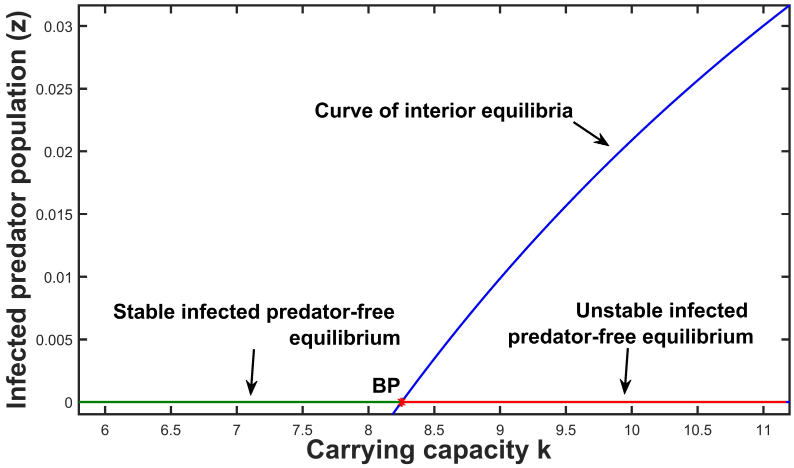


Figure 13: Transcritical bifurcation of E_4 for infected predator population at $k = k^{BP} = 8.251581$.

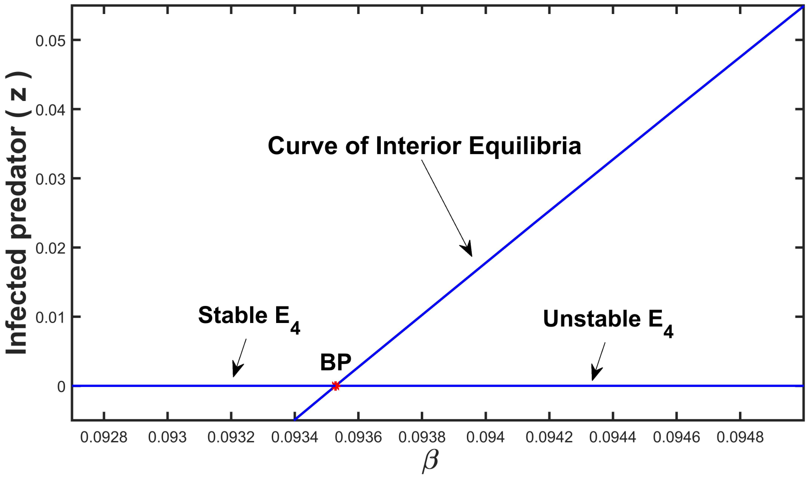


Figure 14: Transcritical bifurcation of infected predator-free equilibrium for infected predator population with respect to $\beta = \beta^{BP} = 0.093528$.

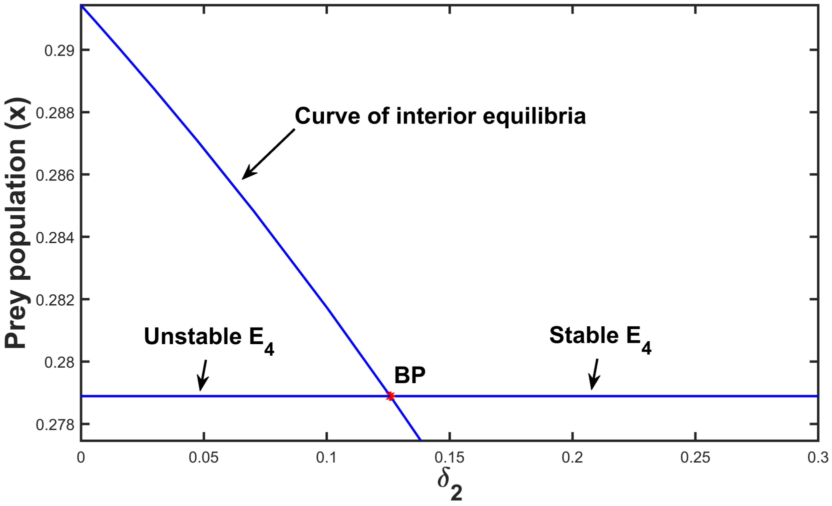


Figure 15: Transcritical bifurcation of E_4 for prey population at $\delta_2 = \delta_2^{BP} = 0.125840$.

9. Conclusion

In this study, we investigate an eco-epidemiological prey–predator model that accounts for the presence of an infectious disease within the predator population, resulting in its division into two classes: susceptible and infected predators. The model further assumes that a fraction of the prey population employs a linear refuge mechanism to evade predation by both predator groups. Predation is modeled using a Holling type I functional response. There are four biologically feasible equilibria, for which we discussed their existence and local stabilities. It is seen that the trivial equilibria is unstable unconditionally. The other three feasible equilibria are conditionally asymptotically stable. The theoretical and numerical results shows that the disease transmission rate (β) , the prey refuge rate (δ_1) against the susceptible predator and the catchability coefficient of the infected predator (q_1) have a major impact on the dynamics of the system (2.1). Increasing the value of β the prey population increases, whereas the susceptible predator diminishes and when β crosses 0.38 , the susceptible predator go to extinct. On the otherhand, for gradual increase of β, the density of infected predator population increases. Therefore, we can treat the disease transmission rate as one of the crucial parameter of the system. Increasing the amount of catchability coefficient (q_1) causes a diminishment in the infected predator population and therefore the susceptible predator population eventually increases. The hiding behaviour of prey plays an important role in the present paper. We observed that if the strength of refuge δ_1 increases in some range , the susceptible predator population decreases and further increament of δ_1 leads the susceptible predator go to extinct while the opposite holds for infected predator. Larger the value of δ_1 helps in the increasing of prey population density. We have seen the system undergoes transcritical bifurcation for catchability coefficient of infected predator, disease transmission rate, prey refuge coefficient δ_2 against infected predator and carrying capacity of prey population.

The existing body of research on eco-epidemiological prey–predator models has significantly advanced our understanding of disease dynamics within ecological systems. A considerable number of these studies have focused on infections in predator populations, yet relatively few have addressed the role of harvesting as a control strategy. For example, Zhang et al. [54] and A.A. Shaikh et al. [55] developed models incorporating disease transmission among predators, but neither incorporated harvesting mechanisms, which are often crucial for managing population levels and disease spread. Similarly, Murthy and Bahlool [57] examined the interaction of prey refuge with predator infection, but harvesting effects were not included in their analysis. In another stream of research, Melese et al. [45] and Thota [56] analyzed the impacts of infection and harvesting on predator–prey systems, yet omitted the influence of prey refuge. More recently, Islam and Sarwardi [37] proposed a comprehensive model that integrates nonlinear prey refuge and harvesting from both susceptible and infected predator populations. While their model presents a significant step forward, it does not isolate the effect of harvesting only the infected predators—a strategy that may be more realistic and effective in practical disease control efforts. Addressing this gap, our study introduces a novel eco-epidemiological model that incorporates linear prey refuge and implements harvesting exclusively on the infected predator population. This modification not only reflects a more targeted and biologically feasible approach but also contributes to improved understanding of how selective harvesting can be employed as a tool for disease regulation in predator populations.

In future studies, we aim to enhance the present model by incorporating a Holling type II functional response, which accounts for predator satiation and provides a more realistic representation of predator–prey interactions. Additionally, we plan to investigate optimal harvesting strategies, with particular attention to how selective or time-dependent harvesting can influence population sustainability and disease control. The inclusion of prey harvesting may also introduce richer and more intricate dynamical behavior, potentially leading to multiple stability regimes or bifurcation patterns not observed in the current model. Furthermore, we envision refining the model by integrating other ecologically relevant factors such as fear-induced behavioral changes in prey, immigration of predator or prey species, group defense mechanisms, and cooperative hunting among predators. These enhancements could offer a more comprehensive understanding of real-world ecological systems and support more effective wildlife management and conservation strategies.

References

1. Malthus, T.R., *An Essay on the Principle of Population*, J. Johnson in St. Paul's Churchyard, London, (1798).

2. Verhulst, P.F., *Notice sur la loi que la population suit dans son accroissement, in: J.G. Garnier, A. Quetelet (Eds.)*, Correspondance Math´ematique et Physique, 10, 113–121, (1838).
3. Lotka, A. J., *Elements of Physical Biology.* Williams and Wilkins, Baltimore, (1925).
4. Volterra, V., *Variazione e fluttuazioni del numero d'individui in specie animali conviventi*, Memorie della Reale Accademia Nazionale dei Lincei, 2 , 31–113, (1926).
5. Kermack, W.O., McKendrick, A.G, *Contributions to the mathematical theory of epidemics. II .—the problem of endemicity*, Proc. R. Soc. Lond. A , 138(834),55–83, (1932).
6. Anderson, R.M., May, R.M., *Regulation and stability of host-parasite population interactions: I. Regulatory processes*, J. Anim. Ecol, 47, 219–267, (1978).
7. Chattopadhyay, J., Arino, O., *A predator–prey model with disease in the prey*, Nonlin. Anal. Th. Meth. Appl. , 36, 747–766, (1999).
8. Chattopadhaya, J., Bairagi, N., *Pelicans at risk in salton sea-an eco epidemiological model*, Ecological Modelling , 136,103-112, (2001).
9. Bairagi, N., Roy, P.K., Chattopadhyay, J., *Role of infection on the stability of a predator-prey system with several response functions a comparative study*, Journal of Theoretical Biology , 248, 10-25, (2007).
10. Kashyap, A.J., Bhattacharjee, D., Sarmah, H.K., *A fractional model in exploring the role of fear in mass mortality of pelicans in the Salton Sea*, An International Journal of Optimization and Control: Theories & Applications, 11(3), 28-51, (2021) .
11. Jang, S.R.J., Wei, H.C., *Deterministic predator–prey models with disease in the prey population*, J. Biol. Syst., 28, 751-784, (2020).
12. Bhatia, S.K., Chauhan, S., *Role of refuge on dynamics of prey-predator model with infected prey*, Commun.Math.Biol.Neurosci. ,11, (2019).
13. Maiti, A.P., Jana, C., Maiti, D.K., *A delayed eco-epidemiological model with nonlinear incidence rate and Crowley–Martin functional response for infected prey and predator*, Nonlinear Dyn., 98, 1137-1167, (2019).
14. Gaber, T., Rahman,R., Widowati, *Dynamical analysis of an eco-epidemiological model experiencing the crowding effect of infected prey*, Commun. Math. Biol. Neurosci., 2024(3), (2024).
15. Triparthi, D., Singh, A., *An eco-epidemiological model with predator switching behavior*, Computational and Mathematical Biophysics, 2023(11), (2023).
16. Pal, A.K., Bhattacharyya, A., Pal, S., *Study of Delay Induced Eco-Epidemiological Model Incorporating a Prey Refuge*, Filomat, 36(2), 557–578, (2022) .
17. Xu, R., Zhang, S., *Modelling and analysis of a delayed predator–prey model with disease in the predator*, Appl. Math. Comput., 224, 372–386, (2013).
18. Venturino E., *Epidemics in predator–prey models: disease in the predators*, IMA J Math Appl Med Biol , 19, 285-205, (2002).
19. Haque M., Venturino E., *Modelling disease spreading in symbiotic communities, Wildlife Destruction, Conservation and Biodiversity*, Nova Science Publishers, (2009).
20. Pal, P., Haque, M., Mandal, P., *Dynamics of a predator–prey model with disease in the predator*, Math. Meth. Appl. Sci., 37, 2429–2450, (2014).
21. Biswas, S., Samanta, S., Chattopadhyay, J., *Cannibalistic predator–prey model with disease in predator — A delay model*, Int. J. Bifurcation and Chaos, 25, 1550130-1–31, (2015).
22. Sarwardi, S., Haque, M., Venturino, E., *Global stability and persistence in LG-Holling type II diseased predator ecosystems*, J. Biol. Phys., 37 , 91–106, (2011).
23. Kashyap,A.J., Bhattacharjee, D., Sarmah, H.K., Dehingia, K., *Dynamical Analysis Of A Predator-Prey Epidemiological Model With Density Dependent Disease Recovery*, Commun. Math. Biol. Neurosci. ,80, (2020).
24. Al-Jubouri, K.Q., Naji, R.K., *Delay in eco-epidemiological prey-predator model with predator fear and hunting cooperation*, Commun. Math. Biol. Neurosci., 89, (2023).
25. Fakhry, N.H., Naji, R.K., *Fear and Hunting Cooperation's Impact on the Eco-Epidemiological Model's Dynamics*, Int. J. Anal. Appl., 22(15), (2024).
26. Das, K., Kundu, K., Chattopadhyay, J., *A predator–prey mathematical model with both the populations affected by diseases*, Ecol. Compl., 8, 68–80, (2011).
27. Arora, C., Kumar, V., Kant, S., Bhowmick, S., *A Delayed Prey–Predator System with Migration and Disease Infection*, Int. J. Appl. Comput. Math., 6:132, (2020).
28. Bezabih, A.F., Edessa, G.K., Rao, K.P., *Eco-epidemiological Model and Analysis of Prey-Predator System*, J. Appl. Math., 2021(4), 1-17, (2021) .

29. Kashyap, A.J., Bhattacharjee, D., Sarmah, H.K., Paul R., *Dynamics in a ratio-dependent eco-epidemiological predator-prey model having cross species disease transmission*, Commun. Math. Biol. Neurosci., 2021(15), (2021).

30. Tavsanoglu, U.N., Cakiroglu, A.I., Erdogan, S., Meerhoff, M., Jeppesen, E., Beklioglu, M., *Sediments, not plants, offer the preferred refuge for Daphnia against fish predation in Mediterranean shallow lakes: An experimental demonstration*, Freshw. Biol., 57, 795–802, (2012).

31. Yiu, D.S., Feehan, C.J., *Articulated coralline algae provide a spatial refuge to juvenile sea urchins from predatory crabs*, Mar. Biol., 164, 76, (2017).

32. Kar, T.K., *Stability analysis of a prey–predator model incorporating a prey refuge*, Commun. Nonlinear Sci.Numer. Simul. , 10, 681–691, (2005) .

33. Mukherjee D., *The effect of prey refuges on a three species food chain model*, Differential Equations and Dynamical Systems, 22(4), 413–26, (2014).

34. Chen, F., Ma, Z., Zhang, H., *Global asymptotical stability of the positive equilibrium of the Lotka–Volterra prey-predator model incorporating a constant number of prey refuges*, Nonlinear Anal.: Real World Appl., 13(6), 2790–2793, (2012).

35. Chen, F., Chen, L., Xie, X., *On a Leslie–Gower predator–prey model incorporating a prey refuge*, Nonlinear Anal., Real World Appl., 10(5), 2905–2908, (2009).

36. Ghosh, J., Sahoo, B., Poria, S., *Prey-predator dynamics with prey refuge providing additional food to predator*, Chaos, Solitons & Fractals, 96, 110-119, (2017).

37. Islam, Md.S., Sarwardi, S., *Analysis of an Eco-Epidemic Predator-Prey Model with Nonlinear Prey Refuges and Predator Harvesting*, J. Appl. Nonlinear Dynam., 12(3) , 465-483, (2023).

38. Hassell, M.P., *The Dynamics of Arthopod Predator-Prey Systems*, (MPB-13), 13, Princeton University Press, (2020).

39. McNair, J.N., *The effects of refuges on predator-prey interactions: a reconsideration*, Theor. Popul. Biol., 29(1), 38-63, (1986).

40. Huang , Y., Zhu, Z., Li, Z., *Modeling the Allee effect and fear effect in predator–prey system incorporating a prey refuge*, Adv. Differ. Equ., 2020(321), (2020).

41. Khajanchi, S., Banerjee, S., *Role of constant prey refuge on stage structure predator–prey model with ratio dependent functional response*, Appl. Math. Comput. 314, 193–198, (2017).

42. Xiao, Z., Li, Z., Zhu, Z., et al., *Hopf bifurcation and stability in a Beddington–DeAngelis predator–prey model with stage structure for predator and time delay incorporating prey refuge*, Open Math., 17(1), 141–159, (2019).

43. Ma, Z., Li, W., Zhao, Y., Wang, W., Zhang, H., Li, Z., *Effects of prey refuges on a predator-prey model with a class of functional responses: The role of refuges*, Math. Biosci., 218(2), 73–79, (2009).

44. Ibrahim, H.A., Bahlool, D.K., Satar, H.A., Naji, R.K., *Stability and bifurcation of a prey-predator system incorporating fear and refuge*, Commun. Math. Biol. Neurosci., 2022(32), (2022).

45. Melese, D., Muhye, O., Sahu, S.K., *Dynamical Behavior of an Eco-epidemiological Model Incorporating Prey Refuge and Prey Harvesting*, Appl. Appl. Math., 15(2), 1193 – 1212, (2020).

46. https://wildfish.org/wp-content/uploads/2022/10/Scottish-salmon-farming-Harvesting-sea-lice-and-disease.pdf

47. Barrett, Luke, T., Oldham,T., Kristiansen, T.S., Oppedal, F., Stien, L.H., *Declining size-atharvest in Norwegian salmon aquaculture: Lice, disease, and the role of stunboats*, Aquaculture, 559, 738440, (2022).

48. Palencia, P., Blome, S., Brook, R.K., Ferroglio, E., Jo, Y., Linden, A., Montoro, V., Penrith, M.L., Plhal, R., Vicente, J., Viltrop, A., Gortázar, C., *Tools and opportunities for African swine fever control in wild boar and feral pigs: a review*, Eur. J. Wildl. Res., 69, (2023).

49. https://www.agrifarming.in/fish-diseases-symptoms-and-control-methods

50. https://thefishsite.com/articles/mycobacterial-infections-of-fish tags

51. https://www.intrafish.com/salmon/salmon-farmer-cermaq-to-cull-900-000-fish-due-to-infection/2-1-1616232

52. Ben-Horin, T., Lafferty, K.D., Bidegain, G., Lenihan, H.S., *Fishing diseased abalone to promote yield and conservation*, Phil. Trans. R. Soc., B 371: 20150211, (2016).

53. Perko, L., *Differential Equations and Dynamical Systems.* Third edition (New York: Springer-Verlag-Inc), (2001).

54. Zhang, C., Liu, S., Huang, J., Wang, W., *Stability and Hoph bifurcation in an eco-epidemiological system with the cost of anti-predators behaviors*, MBE ,20, 8146-8161, (2023).

55. Shaikh, A.A., Das, H., Ali, N., *Study of LG-Holling type III predator-prey model with disease in predator*, J.Appl.Math.Comput., (2017).

56. Thota, S., *A mathematical study on a diseased prey-predator model with predator harvesting*, Asian Journal of Fuzzy and Applied Mathematics, 8(2), (2020).

57. Murthy, M.V.R., Bahlool, D.K., *Dynamics of a prey-predator model involving a prey refuge and disease in the predator*, Mathematical Theory and Modeling , 5(11), (2015).

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