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DYNAMICAL BEHAVIOR OF A DISEASED PREDATOR-PREY MODEL WITH FEAR EFFECT AND PREY HARVESTING

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Abstract. This article consists of a three-species food web model that has been developed by considering the interaction between susceptible prey, infected prey and predator species. It is assumed that susceptible prey species grow logistically in the absence of predators. It is assumed that predators consume susceptible and infected prey . We consider the effect of fear on susceptible prey due to predator species. Again, the harvesting of susceptible and infected prey has been considered. Furthermore, the predator consumes its prey in the form of Holling-type interactions. The positive invariance, positivity, and boundedness of the system are discussed. The conditions of all biologically feasible equilibrium points have been examined. The local stability of the systems around these equilibrium points is investigated. Furthermore, the occurrence of Hopf-bifurcation concerning the harvesting (h) of the system has been investigated. Finally, we demonstrate some numerical simulation results to illustrate our main analytical findings.

1. Introduction

The term ecology (*oecologie*) was coined in 1876 by the German evolutionary biologist Ernst Haeckel [1]. He combined two Greek words, "oikos," meaning "house" or "dwelling place," and "logos," meaning "science" or "study," to form the word. Ecology is the study of plants and animals activities. Plants and animals are the

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scientific study of the relationship of organisms to each other and to their physical environment. Epidemiology is the study of the prevalence and determinants of health-related conditions or events in specific populations and the application of this study to control health problems. Epidemiology began with Adam and Eve, both of whom sought to examine the quality of the "forbidden fruits." Epidemiology is the study of the distribution and determination of health-related conditions or events in specific populations and the application of this study. Mathematical ecology and mathematical epidemiology are major fields of study in their own right. But there are some commonalities between them. A branch of ecology that considers the effects of transmissible diseases is called eco-epidemiology. Eco-epidemiology is a new branch of mathematical biology that considers both ecological and epidemiological problems simultaneously. Eco-epidemiological research deals with diseases that spread in an interactive population in which epidemiologic and demographic features are incorporated into a model [2, 3].

Eco-epidemiological systems are used to investigate the dynamic connection between predator and prey in one population or a population of susceptible and infected animals. Mathematical models have become significant instruments in examining the flow and manipulation of prevention. Kermack-Mckendrick's [4] pioneering work on SIRS epidemiological models has drawn a lot of interest from researchers. Many investigators have studied the population ecology of prey, predators, or both. The non-linear relationship between populations of predators and their prey has been and will remain one of the subjects that are most frequently addressed in both mathematical ecology and epidemiology due to its worldwide existence and significance. Although these issues appear straightforward mathematically at first glance, they are challenging and complicated. Ecology and epidemiology are two distinct, essential, and significant areas of research. Lotka [5] and Volterra [6] models, the first advance in current mathematical ecology, can be examined using the system of dynamical equations. Environmental epidemiology is the complete study of epidemiology and ecology. Eco-epidemiology exerts a significant ecological impact. It is referred to as the study of infection spread between interacting organisms. A variety of mathematical and statistical methodologies are available for analysing eco-epidemiological data. Many ecosystems around the world have predator-prey interactions between species, as well as the lion-deer association. In the environment, predator and prey species display oscillations in population increase and decline or abundance. Animal conservationists and mathematicians have long been intrigued by the study of this volatility in seemingly stable patterns. As a result, many others have extensively studied the dynamics of prey-predator interactions over the last three decades. [7], [8], [9]. Population growth models with the spread of diseases frequently exhibit complicated, non-linear mathematical dynamics. The fundamental goal of these models is to investigate points of equilibrium, their analyses of stability, solutions in the type of periodic, bifurcations, system behaviour of chaotic nature, and so on.

Alfred J. Lotka was the first to investigate the relationships between populations of predators and their prey. A biological representation in terms of mathematical modelling of communications among the population density of predators and population density of prey, called "functional response," is the major part of biological modelling in the population density of predators and population density of prey. There are numerous functional responses, namely the types I–III of the Holling response, the Varley-Hassell response, the Beddington-DeAngelis response, and the Crowley-Martin response. Arditi and Ginzburg's [10] relatively popular type of ratio-dependent response. Much more information on predator-prey systems with Crowley-Martin functional responses has become available in recent decades. In the recent era, some renowned authors [11, 12], [13], [14], [15], [16], [17]. studied functional responses to comprehend the importance of the relationship between the prey and predator in the ecosystem. They used some functional responses, such as the Crowley-Martin functional response, to make the model system more realistic and controllable in the ecosystem. Several investigators [18–21] started exploring a non-linear analysis of the predator-prey scenario involving infection in either the prey or predator population or both populations or the two forms of infection in the predator population system with a functional non-linear response that includes the function of type II Holling. The global and local stability investigations explored the prey-predator food web model with the function of type II Holling, which included the bifurcation analysis for the ratio-dependent intraguild predation model. Recently, several investigators have discovered that there is frequently a constant percentage of prey that is shielded from predators by the refuge. The interactions between prey and predators may be stabilised by refugia, according to several studies and mathematical models. In [22], Maynard Smith discovered that the presence of a static proportional size of refuge of any size neutrally altered the static nature of equilibrium, according to the stochastic stability of a Lotka-Volterra unbiased model. A neutrally stable Lotka-Volterra model's dynamic stability was unaffected by the presence of a constant proportionate refuge. Tapan Kumar Kar [23] considered a Holing type II response function integration and predator model with prey refuge. Commercial exploitation of biological resources to meet society's increasing demands has long been a cause of concern for ecologists, bioeconomists, and resource managers of nature. The impact of harvests is extensively used in forestry, wildlife management, and fisheries. This research uncovered a wide range of fascinating dynamics, such as points of equilibrium, analyses of bifurcation, and limit cycles. In eco-epidemiology, we explore predator-prey models that include infection dynamics. We seek to investigate the dynamics of the predator-prey model using this functional response. A form of predator-dependent functional response is a ratio-dependent functional response. The predation rate of the prey is supposed to be the number of prey consumed by a predator per unit of time. When predator-prey interactions involve intensive searching, ratio-dependent predator-prey models are more suitable than other types. Recently, [24], [25], [26] many

researchers have investigated the apparent biological and physiological evidence of growth under different conditions. The prey population density is low in a ratio-dependent model, and as the number of prey grows, the reaction to every predator activity becomes more constant (i.e., a type II reaction under Holling). [15]). Recently, several investigators have discovered that there is frequently a constant percentage of prey that is shielded from predators by the refuge. Predator-prey interactions have been included in the Lotka-Volterra model for a very long time. In a similar vein, the seminal work on the interaction of the susceptible, infected, and recovered has been an interesting topic of study. The original predator-prey model was developed, in large part, by Vito Volterra and Alfred James Lotka. Ecology models and epidemiology models are the two basic categories into which mathematical models are often divided. In the ecological framework, the relationship between the population density of some communities is studied. Epidemiology systems are used to investigate the spread of illnesses between wildlife and humans. It is increasingly crucial to do research on the dynamics of illness within ecological systems. On the one hand, several studies of prey-predator dynamics have been conducted in recent decades, taking into account the impact of a range of biological characteristics. Many mathematical models have been created and investigated in the field of epidemiology, taking into consideration various incidence rates and illnesses. Experts were particularly interested in their recommended ecological models since it is well-accepted that species harvesting is necessary for species coexistence. Ecology models and epidemiology models are the two basic categories into which mathematical models are often divided. There are three different forms of harvesting: constant, proportional to density, nonlinear, and others. All of these have been proposed and investigated. There have been several suggestions for harvesting methods based on research, including harvesting continuously and depending on density in proportional harvesting. We research predator-prey models as well as disease dynamics in eco-epidemiology. Using this physiological response, we hope to investigate the dynamics of the predator-prey paradigm. To address this problem, we study the impact of fear in an eco-epidemiological model with infected prey in this paper. To the best of the available information, none of the scholars have explored the three-species food web model of prey-predator relationships that combines species relationships, such as Holling type II function and disease in prey populations, with the influence of fear in prey harvesting. We explore the diseased prey-predator model utilising Holling type II interaction as well as the influence of fear on susceptible prey populations due to predators and prey harvesting, motivated by this fact. The rest of the paper is structured as follows: The mathematical analysis is investigated in Section 2. In Section 3, some preliminary aspects of the model have been studied. Section 4 deals with the point of equilibrium at the boundary and its stability. In Sections 5 and 6, we determine the existence of the interior point of equilibrium $(E^*(s^*, i^*, p^*))$ and investigate its local and global stability. The occurrence of Hopf-bifurcation is shown in Section 7. Numerical

simulations are examined for the proposed model in Section 8. The conclusion of the paper and the biological consequences of our mathematical results are found in Section 8, which concludes the paper.

2. Model Formation

The framework demonstrates the relationship between the population density of prey with infection. Which leads to the following structure of non-linear differential equations. The suggested framework was applied to examine the non-linear population density of susceptible, infected prey and predator biological model

$$\left.\begin{array}{rcl} \frac{d\mathcal{S}}{dT} = & \frac{r_1\mathcal{S}}{1+\mathcal{F}\mathcal{P}}(1-\frac{\mathcal{S}+\mathcal{I}}{K}) - \lambda\mathcal{I}\mathcal{S} - \frac{\alpha_1\mathcal{S}\mathcal{P}}{a_1+\mathcal{S}} - H_1E_1\mathcal{S}, \\ \frac{d\mathcal{I}}{dT} = & \lambda\mathcal{I}\mathcal{S} - d_1\mathcal{I} - \frac{b_1\mathcal{I}\mathcal{P}}{a_1+\mathcal{I}} - H_2E_2\mathcal{I}, \\ \frac{d\mathcal{P}}{dT} = & -d_2\mathcal{P} + \frac{cb_1\mathcal{I}\mathcal{P}}{a_1+\mathcal{I}} + \frac{c\alpha_1\mathcal{S}\mathcal{P}}{a_1+\mathcal{S}}. \end{array}\right\} \tag{1}$$

Here the conditions are $\mathcal{S}(0) \geq 0, \mathcal{I}(0) \geq 0$ and $\mathcal{P}(0) \geq 0$ the table displays specific biological meanings of the parameters.

Table 1. Biological representation of the model

Parameters	Units	Biological representation
$\mathcal{S}$	Number of components per unit area (tons)	Population density of susceptible Prey
$\mathcal{I}$	Number of components per unit area (tons)	Population density of prey with infection
$\mathcal{P}$	Number of components per unit area (tons)	Population density of Predator
r_1	Per day (T^{-1})	Prey population densities growth rate
K	Number of components per unit area (tons)	The carrying ability of nature
λ	Per day (T^{-1})	Infection rate
a_1	Per day (V)	Constant of Half-saturation
α_1	Per day (T^{-1})	Susceptible prey to predator consumption
b_1	Per day (T^{-1})	Capture rate by predator
c	Per day	Conversion rate of prey to predator
d_1	Per day (T^{-1})	density of diseased prey mortality rate
d_2	Per day (T^{-1})	Density of predator population mortality rate
$\mathcal{F}$	Number of components per unit area (tons)	Impact of fear
E_1, E_2	Number of components per unit area (tons)	Harvesting Effect
H_1, H_2	Number of components per unit area (tons)	Prey's catchability coefficient

The condition for the fear effect is

$$\mathcal{F}_1(\beta, p) = \frac{1}{1+\beta p} \tag{2}$$

This describes the level of fear in susceptible prey as a consequence of the predator. Here, β represents the quantity of fear. Given the epidemiological meaning of β, the following condition is strongly acceptable:

$$\mathcal{F}_1(0, p) = \mathcal{F}_1(\beta, 0) = 1$$
$$lim_{\beta\to\infty}\mathcal{F}_1(\beta, p) = 0 = lim_{p\to\infty}\mathcal{F}_1(\beta, p)$$

$$\frac{\partial \mathcal{F}_1(\beta,p)}{\partial \beta} < 0,$$
$$\frac{\partial \mathcal{F}_1(\beta,p)}{\partial p} < 0.$$

In this work we incorporate prey and the fear effect β. Then the system change into the non-dimensional .
Here,
$s = \frac{\mathcal{S}}{K}, \quad i = \frac{\mathcal{I}}{K}, \quad p = \frac{\mathcal{P}}{K}$.
Now (1) becomes,

$$\left.\begin{array}{rcl} \frac{ds}{dt} = & \frac{rs}{1+\beta p}(1-s-i) - is - \frac{s\alpha p}{s+a} - h_1 s \\ \frac{di}{dt} = & is - di - \frac{\theta ip}{a+i} - h_2 i \\ \frac{dp}{dt} = & -\delta p + \frac{c\theta ip}{a+i} + \frac{c\alpha sp}{s+a}. \end{array}\right\} \tag{3}$$

here the conditions are,

$$r = \frac{r_1}{\lambda K}, \quad \alpha = \frac{\alpha_1}{\lambda K}, \quad h_1 = \frac{H_1 E_1}{\lambda K},$$
$$d = \frac{d_1}{\lambda K}, \quad h_2 = \frac{H_2 E_2}{\lambda K}, \quad \theta = \frac{b_1}{\lambda K},$$
$$a = \frac{a_1}{K}, \quad \delta = \frac{d_2}{\lambda K}, \quad \beta = \frac{\mathcal{F}}{K}.$$

According to the preliminary criteria $\{s(0), i(0), p(0)\} \geq 0$. The operations described over are in $\mathbb{R}^3_+$.

3. Positivity, Existence and Boundedness of solutions

In this section we discusses the positivity and boundedness solution of the system.(3)

3.1. **Positivity of solutions.**

Theorem 1. *In the $\mathbb{R}^3_+$ all the (3) systems solutions are non-negative .*

Proof. Since $\{s(0), i(0), p(0)\} \geq 0$, hence the system (3) written as,

$$s(t) = s(0)exp\left(\int_0^1 \left[\frac{r}{1+\beta p}(1-i-s) - i - \frac{p\alpha}{s+a} - h_1\right] ds\right) \geq 0,$$
$$i(t) = i(0)exp\left(\int_0^1 \left[-d + s - \frac{\theta p}{a+i} - h_2\right] ds\right) \geq 0,$$
$$p(t) = p(0)exp\left(\int_0^1 \left[\frac{c\theta i}{a+i} + \frac{c\alpha s}{s+a} - \delta\right] ds\right) \geq 0.$$

Existence of the solutions:
For $t < 0$, let $\mathcal{Z} = (s(t) + i(t) + p(t))$, and $\mathcal{E}(\mathcal{Z}) = (\mathcal{O}_1\mathcal{Z}, \mathcal{O}_2\mathcal{Z}, \mathcal{O}_3\mathcal{Z})^T$, where
$\mathcal{O}_1\mathcal{Z} = \frac{rs}{1+\beta p}(1 - s - i) - is - \frac{\alpha sp}{s+a} - h_1 s$,
$\mathcal{O}_2\mathcal{Z} = is - id - \frac{\theta ip}{a+i} - h_2$,
$\mathcal{O}_3\mathcal{Z} = -\delta p + \frac{c\theta ip}{a+i} + \frac{c\alpha sp}{s+a}$.
Then, (3) is then able to be formed as $\frac{d\mathcal{Z}}{dt} = \mathcal{E}(\mathcal{Z})$, where, $\mathcal{O} : \mathcal{C}_+ \to \mathbb{R}^3_+$ with, $\mathcal{Z}_(0) = \mathcal{Z}_0 \in \mathbb{R}^3_+$.
Here, $\mathcal{E}_\rangle \in \mathcal{C}^\infty(\mathbb{R})$ for $i = 1, 2, 3$. As a result, the mathematical operator $\mathcal{O}$ is both locally Lipschitzian and completely continuous on $\mathbb{R}^3_+$. Therefore, the solution of (3) exists and unique. Hence the region $\mathbb{R}^3_+$ is an invariant domain of the system (3) solutions are positive. □

Theorem 2. *If* $c < 1$, $Max\ \frac{rs}{1+\beta p}(1 - s) = \frac{r}{8}$, *and* $\beta = min(h_1, d + h_2, \delta)$ *in* $\mathbb{R}^3_+$ *all the system (3) solutions are bounded.*

Proof. s, i and p denote the model (3) solutions with positive criteria, hence $\frac{ds}{dt} \leq sr(1 - s)$.
We know that $\limsup_{t\to\infty} s \leq 1$,. Let, $\mathcal{Z} = s + i + p$.

$$\begin{aligned}
\frac{d\mathcal{Z}}{dt} =& \frac{ds}{dt} + \frac{di}{dt} + \frac{dp}{dt} \\
=& \frac{rs}{1+\beta p}(1 - s - i) - si - h_1 s - \frac{(1-c)s\alpha p}{s+a} \\
&+ si - id - \frac{(1-c)\theta ip}{a+i} - p\delta - h_2 i \\
&\leq \frac{rs}{1+\beta p} - p\delta - id - h_1 s - h_2 i \ \ (\text{where}, c < 1) \\
&\leq \frac{r}{8} - p\delta - id - h_1 s - h_2 i \ \left(\text{since}, (Max(\frac{rs}{1+\beta p}(1 - s) = \frac{r}{8})\right) \\
&\leq \frac{r}{8} - \beta\mathcal{Z}, \ \text{where } \beta = min(h_1, d + h_2, \delta),
\end{aligned}$$

we have, $\frac{d\mathcal{Z}}{dt} + \beta z \leq \frac{r}{8}$.
Using the differential inequality theorem, we obtain

$$0 < \mathcal{Z} \leq \frac{r}{4\beta}(1 - \exp^{-\beta t}) + \mathcal{Z}(s_0, i_0, p_0) \exp^{-\beta t}.$$

For $t \to \infty$, we have $0 < \mathcal{Z} < \frac{r}{4\beta}$ in the $\mathbb{R}^3_+$ all the systems (3) solutions are uniformly bounded, for $\epsilon > 0$ are in the region,

$$\Omega = \left\{(s, i, p) \in \mathbb{R}^3_+; s + i + p \leq \frac{r}{4\beta} + \epsilon\right\}.$$

□

4. The Existence of Point of Equilibrium

This section examines the potential points of equilibrium (3). The system (3) has three points of equilibrium and one endemic point of equilibrium:

$$\begin{aligned}
&\frac{rs}{1+\beta p}(1-s-i) - si - \frac{\alpha sp}{s+a} - h_1 s = 0,\\
&is - di - \frac{\theta ip}{a+i} - h_2 i = 0,\\
&-\delta p + \frac{c\theta ip}{a+i} + \frac{c\alpha sp}{s+a} = 0.
\end{aligned}$$

- $E_0(0,0,0)$ is the point of equilibrium, which is trivial,
- $E_1(\frac{r-h_1}{r},0,0)$ is the free of infection and free of predator point of equilibrium that exists for $r > h_1$.
- The absence of predator point of equilibrium is $E_2(\hat{s},\hat{i},0)$, where, $\hat{s} = d + h_2, \hat{i} = \frac{r(1-d-h_2)-h_1}{r+1}$, it exists for $r(1-h_2-d) > h_1$
- Endemic or positive or interior equilibrium is $E^*(s^*,i^*,p^*)$, where $i^* = \frac{a(a\delta+(\delta-c\alpha)s^*)}{(c\alpha s^*+(c\theta-\delta)(s^*+a))}$, $p^* = \frac{ac(s^*-d)(s^*+a)}{(c\alpha s^*+(c\theta-\delta)(s^*+a))}$ and s^* is the unique positive root of the quadratic equation
  $\mathcal{A}S^2 + \mathcal{B}S + \mathcal{C} = 0$,
  where,
  $\mathcal{A} = r(\alpha c + \theta c - \delta)$,
  $\mathcal{B} = (\theta c - \delta)(ar - r) + \alpha c((1+\beta p) - r) + a(\delta(1+\beta p) + (\delta - c\alpha)r)$,
  $\mathcal{C} = -a(r(1+\beta p))(c\theta - \delta) + (c\alpha(1+\beta p)(d) - a\delta((1+\beta p) + r)))$.
  Endemic equilibrium exists for $\delta > \alpha c$.

5. Local Stability Analysis

In order to investigate the local stability property of the system(3), we first find the Jacobian matrix of the system in the form $J(E) = \begin{pmatrix} n_{11} & n_{12} & n_{13} \\ n_{21} & n_{22} & n_{23} \\ n_{31} & n_{32} & n_{33} \end{pmatrix}$.

Here,

$$\begin{aligned}
n_{11} =& \frac{r}{1+\beta p}(1-2s) - i\left(\frac{r}{1+\beta p}+1\right) - \frac{\alpha ap}{(s+a)^2} - h_1, n_{12} = -s(\frac{r}{1+\beta p}+1),\\
n_{13} =& \frac{prs}{(1+\beta p)^2}(1-s-i) - \frac{\alpha s}{s+a}, n_{21} = i, n_{22} = s - d - h_2 - \frac{a\theta p}{(a+i)^2},\\
n_{23} =& -\frac{\theta i}{(a+i)}, n_{31} = \frac{ac\alpha p}{(s+a)^2}, n_{32} = \frac{ac\theta p}{(a+i)^2},
\end{aligned}$$

$$n_{33} = -\delta + \frac{c\theta i}{a+i} + \frac{\alpha cs}{s+a}.$$

Theorem 3. *$E_0(0,0,0)$ is the point of equilibrium, which is trivial, is stable if $r < h_1$, otherwise unstable.*

Proof. The characteristic equation of the point of equilibrium E_0 is,
$(\lambda_{01} - (r - h_1))(\lambda_{02} - (-d - h_2))(\lambda_{03} + \delta) = 0,$
$\lambda_{01} = r - h_1, \lambda_{02} = -d - h_2, \lambda_{03} = -\delta,$
here,$\lambda_{02} < 0, \lambda_{03} < 0$. $E_0(0,0,0)$ is the point of equilibrium, which is trivial,is stable if $r < h_1$ otherwise it is unstable. □

Theorem 4. *$E_1(\frac{r-h_1}{r}, 0, 0)$, the free of infection and free of the predator point of equilibrium, is stable if $c\alpha < \delta$ and $h_1 > r(1 - d - h_2)$, otherwise unstable.*

Proof. The characteristic equation of the point of equilibrium E_1 is,

$$(\lambda_{11} - ((h_1 - r)))(\lambda_{12} - (1 - d - h_2 - \frac{h_1}{r}))(\lambda_{13} - (\frac{-\alpha(r - h_1)}{ra + (r - h_1)} - \delta)) = 0,$$

$$\lambda_{11} = h_1 - r, \lambda_{12} = 1 - d - h_2 - \frac{h_1}{r}, \lambda_{13} = \frac{-c\alpha(r - h_1)}{ra + (r - h_1)} - \delta,$$

here, $E_1(\frac{r-h_1}{r}, 0, 0)$ being free of infection and free of the predator point of equilibrium, is stable if $c\alpha < \delta$ and $h_1 > r(1 - d - h_2)$, otherwise unstable. □

Theorem 5. *The equilibrium $E_2(\hat{s}, \hat{i}, 0)$ which absence of predator is asymptotically stable if $\delta > c(\theta + \alpha)$.*

Proof. The matrix in the form of Jacobian at E_2 is $J(E_3) = \begin{pmatrix} q_{11} & q_{12} & q_{13} \\ q_{21} & q_{22} & q_{23} \\ q_{31} & q_{32} & q_{33} \end{pmatrix}$,
where,

$$q_{11} = r(1 - 2\hat{s}) + i(r + 1), \quad q_{12} = (-1 - r)\hat{s}, \quad q_{13} = -\frac{\alpha\hat{s}}{s + a},$$

$$q_{21} = \hat{i}, \quad q_{22} = 0, \quad q_{23} = -\frac{\theta\hat{i}}{a + \hat{i}},$$

$$q_{31} = 0, \quad q_{32} = 0, \quad q_{33} = \frac{c\alpha\hat{s}}{a + \hat{s}} - \delta + \frac{c\theta\hat{i}}{a + \hat{i}}.$$

Here, the characteristic equation of the above matrix in the form of Jacobian is, $\lambda^3 + \mathcal{L}\lambda^2 + \mathcal{M}\lambda + \mathcal{N} = 0$. Here,

$$\mathcal{L} = -q_{11} - q_{33},$$
$$\mathcal{M} = -q_{21}q_{12} + q_{33}q_{11},$$
$$\mathcal{N} = q_{12}q_{21}q_{33}.$$

If and only if $\mathcal{L}, \mathcal{N}$ and $\mathcal{L}\mathcal{M} - \mathcal{N}$ are positive, then the negative real parts are the roots of the above characteristic equation. According to the Routh-Hurwitz

criterion. now, $\mathcal{LM} - \mathcal{N} = -q_{11}(-q_{12}q_{21} + q_{33}(q_{33} + q_{11}))$. Now, the sufficient conditions for q_{33} to be negative is $\delta > c(\alpha + \theta)$. The E_2 is locally asymptotically stable provided the above condition in theorem satisfied. □

Theorem 6. *The endemic or positive point of equilibrium E^* is asymptotically stable.*

Proof. The matrix in the form of Jacobian at E^* is $J(E^*) = \begin{pmatrix} r_{11} & r_{12} & r_{13} \\ r_{21} & r_{22} & r_{23} \\ r_{31} & r_{32} & r_{33} \end{pmatrix}$, where,

$$\begin{aligned} r_{11} =& -\frac{s^*(h_1 - r + ar + (1+r)i^* + 2rs^*)}{s^* + a}, \quad r_{12} = -s^*(\frac{r}{1+\beta p^*} + 1), \\ r_{13} =& \frac{p^* r s^*}{(1+\beta p^*)^2}(1 - s^* - i^*) - \frac{\alpha s^*}{s^* + a}, \\ r_{21} =& i^*, \quad r_{22} = \frac{a\theta p^* i^*}{(a+i^*)^2}, \quad r_{23} = \frac{\theta i^*}{(a+i^*)}, \\ r_{31} =& \frac{ac\alpha p^*}{(s^*+a)^2}, \quad r_{32} = \frac{ac\theta p^*}{(a+i^*)^2}, \quad r_{33} = 0. \end{aligned}$$

Here, the characteristic equation of the Matrix in the form of Jacobian E^* is

$$\lambda^3 + \mathcal{F}\lambda^2 + \mathcal{G}\lambda + \mathcal{H} = 0, \tag{4}$$

here,

$$\begin{aligned} \mathcal{F} = -r_{11} - r_{33}, \mathcal{G} = -r_{21}r_{12} + r_{22}r_{11} - r_{13}r_{31} + r_{23}r_{32}, \\ \mathcal{H} = r_{13}(-r_{22}r_{31} + r_{21}r_{32}) + r_{23}(r_{12}r_{31} - r_{11}r_{32}). \end{aligned}$$

If $\mathcal{F} > 0, \mathcal{H} > 0, \mathcal{FG} - \mathcal{H} > 0$. The negative real parts are the roots of the above characteristic equation if and only if $\mathcal{F}, \mathcal{H}$ and $\mathcal{FG} - \mathcal{H}$ are non-negative, according to the Routh-Hurwitz criterion. The E^* is locally asymptotically stable. □

6. Hopf-Bifurcation Analysis

In this part, we use the harvesting (h_1) effect to analyse the model's bifurcation. Using the bifurcating parameter h_1, the following theorem shows the presence of Hope-bifurcation.

Theorem 7. *The model (3) confronts Hopf-bifurcation if the bifurcation parameter h_1 surpasses a critical point. The following Hopf-bifurcation conditions arise at $h_1 = h_1^*$:*
1.$\mathcal{A}_1(h_1^*)A(h_1^*) - \mathcal{A}_3(h_1^*) = 0$.
2.$\frac{d}{df}(Re(\lambda(h_1)))|_{h_1 = h_1{}^*} \neq 0$ *Here lambda is the zero of the parametric solution correlated with the equilibrium's interior point.*

Proof. For $h_1 = h_1^*$, let the equation of characteristic (4) is in the form

$$(\lambda^2(h_1^*) + \mathcal{A}_2(h_1^*))(\lambda(h_1^*) + \mathcal{A}_1(h_1^*)) = 0. \tag{5}$$

This indicates that the roots of the preceding equation are $\pm i\sqrt{\mathcal{A}_2(h_1^*)}$ and $-\mathcal{A}_1(h_1^*)$. To achieve the Hopf-bifurcation at $h_1 = h_1^*$ the following transversality criterion must be fulfilled.

$$\tfrac{d}{dh_1^*}(Re(\lambda(h_1^*)))| \neq 0.$$

For h_1, the above equation (5) has general roots

$$\lambda_1 = r(h_1) + is(h_1),$$
$$\lambda_2 = r(h_1) - is(h_1),$$
$$\lambda_3 = -\mathcal{A}_1(h_1).$$

Weather check the criteria $\tfrac{d}{dh_1^*}(Re(\lambda(h_1^*)))| \neq 0$.
Let $\lambda_1 = r(h_1) + is(h_1)$ in the (5), we get

$$\mathcal{C}(h_1) + i\mathcal{D}(h_1) = 0.$$

Where,

$$\mathcal{C}(h_1) = r^3(h_1) + r^2(h_1)\mathcal{A}_1(h_1) - 3r(h_1)s^2(h_1) - s^2(h_1)\mathcal{A}_1(h_1) + \mathcal{A}_2(h_1)r(h_1) + \mathcal{A}_1(h_1)\mathcal{A}_2(h_1),$$
$$\mathcal{D}(h_1) = \mathcal{A}_2(h_1)s(h_1) + 2r(h_1)s(h_1)\mathcal{A}_1(h_1) + 3r^2(h_1)s(h_1) + s^3(h_1).$$

In order to fulfill the (5) we must have $\mathcal{C}(h_1) = 0$ and $\mathcal{D}(h_1) = 0$, then calculating $\mathcal{C}$ and $\mathcal{D}$ with respect to h_1. We have

$$\frac{d\mathcal{C}}{dh_1} = \varsigma_1(h_1)r^{'}(h_1) - \varsigma_2(h_1)s^{'}(h_1) + \varsigma_3(h_1) = 0, \tag{6}$$

$$\frac{d\mathcal{D}}{dh_1} = \varsigma_2(h_1)r^{'}(h_1) + \varsigma_1(h_1)s^{'}(h_1) + \varsigma_4(h_1) = 0, \tag{7}$$

where,

$$\varsigma_1 = 3r^2(h_1) + 2r(h_1)\mathcal{A}_1(h_1) - 3s^2(h_1) + \mathcal{A}_2(h_1),$$
$$\varsigma_2 = 6r(h_1)s(h_1) + 2s(h_1)a_1(h_1),$$
$$\varsigma_3 = r^2(h_1)\mathcal{A}_1^{'}(h_1) + s^2(h_1)\mathcal{A}_1^{'}(h_1) + \mathcal{A}_2^{'}(h_1)r(h_1),$$
$$\varsigma_4 = \mathcal{A}_2^{'}(h_1)s(h_1) + 2r(h_1)s(h_1)\mathcal{A}_1^{'}(h_1).$$

On multiplying (6) by $\varsigma_1(h_1)$ and (7) by $\varsigma_2(h_1)$ respectively

$$r(h_1)^{'} = -\frac{\varsigma_1(h_1)\varsigma_3(h_1) + \varsigma_2(h_1)\varsigma_4(h_1)}{{\varsigma_1}^2(h_1) + {\varsigma_2}^2(h_1)}. \tag{8}$$

Substituting $r(h_1) = 0$ and $s(h_1) = \sqrt{\mathcal{A}_2(h_1)}$ at $h_1 = h_1^*$ on $\varsigma_1(h_1), \varsigma_2(h_1), \varsigma_3(h_1)$, and $\varsigma_4(h_1)$, we obtain

$$\begin{aligned}
\varsigma_1(h_1^*) =& -2\mathcal{A}_2(h_2^*),\\
\varsigma_2(h_1^*) =& 2\mathcal{A}_1(h_1^*)\sqrt{\mathcal{A}_2(h_1^*)}\\
\varsigma_3(h_1^*) =& \mathcal{A}_3^{'}(h_1^*) - \mathcal{A}_2(h_1^*)\mathcal{A}_1^{'}(h_1^*),\\
\varsigma_4(h_1^*) =& \mathcal{A}_2^{'}(h_1^*)\sqrt{\mathcal{A}_2 h_1^*}.
\end{aligned}$$

The equation (8), implies

$$r^{'}(h_1^*) = \frac{\mathcal{A}_3^{'}(h_1^*) - (\mathcal{A}_1(h_1^*\mathcal{A}_2(h_1^*)))}{2(\mathcal{A}_2(h_1^*) + \mathcal{A}_1^2(h_1^*))}, \tag{9}$$

if $\mathcal{A}_3^{'}(h_1^*) - (\mathcal{A}_1(h_1^*)\mathcal{A}_2(h_1^*))^{'} \neq 0$ which implies that $\frac{d}{dh_1^*}(Re(\lambda(h_1^*)))| \neq 0$, and $\lambda_3(h_1^*) = -\mathcal{A}_1(h_1^*) \neq 0$.
Therefore the condition $\mathcal{A}_3^{'}(h_1^*) - (\mathcal{A}_1(h_1^*)\mathcal{A}_2(h_1^*))^{'} \neq 0$ It has been guaranteed that the transversality criterion is satisfied, hence the model (3) has attained the Hopf-bifurcation at $h_1 = h_1^*$. □

7. Numerical Simulations

In this section, several numerical experiments on the system (3) are carried out to verify the mathematical findings. The rate of fear β , predation rate α and harvesting h_1 are the essential parameters in this study, and they will be used as control parameters. For the specified fixed parameter values given in Table 2, the numerical simulation is carried out using the MATLAB and MATHEMATICA software packages.

Table 2. Parameter values

Parameters	**Numeric value**
r	0.5
a	0.3
c	0.6
d	0.25
θ	0.4
δ	0.2
β	Variable
α	Variable

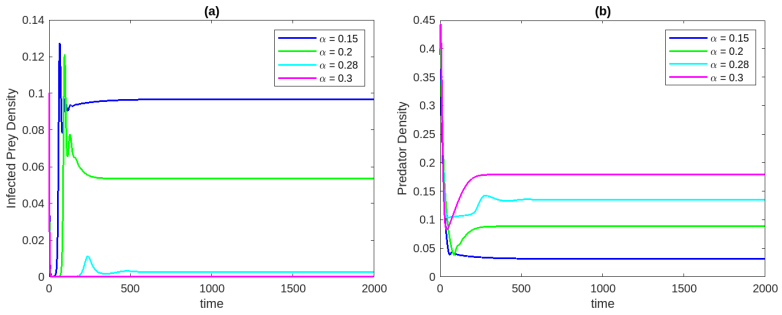

FIGURE 1. *The population of infected prey, and predators for* $\alpha = 0.15, 0.2, 0.28, 0.3$.

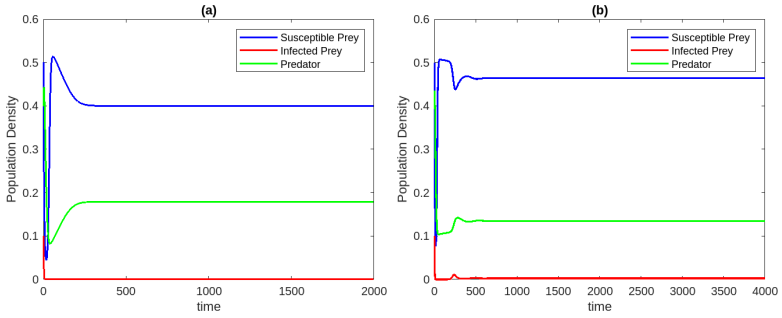

FIGURE 2. *Solutions of time series (3) around the point of equilibrium* E_2 *and the point of equilibrium* E_4.

7.1. **Effect of varying the predation rate** α**.** Let $\beta = 0.3, h_1 = 0.2$ For the parameters specified in Table 2. E_2 is predator free equilibrium and the endemic point of equilibrium E^* exists for $0.1 < \alpha < 035$, respectively, for the given parametric values. The stability of for $\alpha = 0.3$ and $\alpha = 0.28$ is shown in Figure(2). Figure (1) shows that as the predator population grows, so does the predation rate α and the number of infected prey.

7.2. **Effect of varying the harvesting rate** h_1**.** Let $\alpha = 0.3, \beta = 0.15$ For the parameters specified in Table 2.E_2 is predator free equilibrium and the endemic point of equilibrium E^* exists for$0.0140625 < h_1 < 0.307377$, respectively, for the given parametric values. From Figure (3) shows that increasing the rate of harvesting in susceptible prey leads to a decrease in population of susceptible prey and population of predator while increasing the population of infected prey.

7.3. **Bifurcation of harvesting rate** h_1**. Case-I**:(Changing only the parameter value h_1 and $h_2 = 0$)

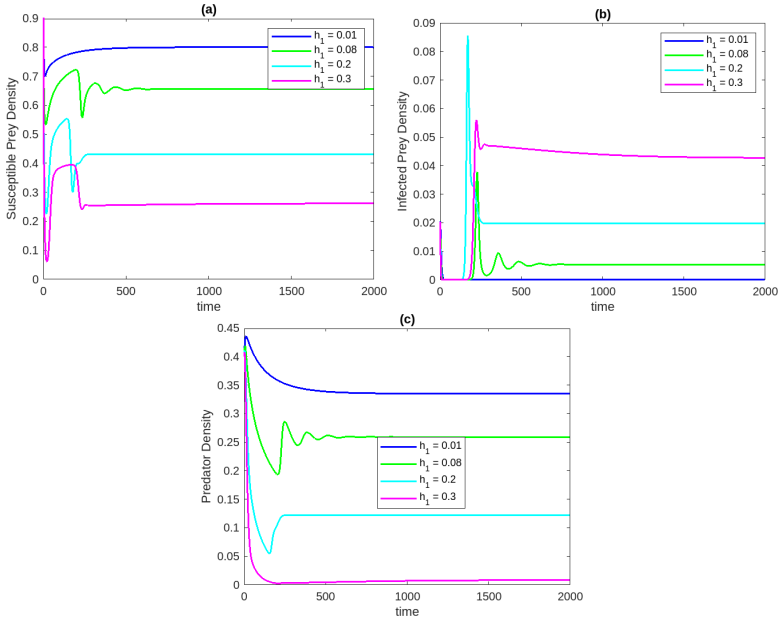

FIGURE 3. *For $\alpha = 0.25$, the population concentrations of susceptible prey, infected prey, and predators are as follows for the parametric values shown in the table. Where $h_1 = 0.01, 0.08, 0.2, 0.3$*

If $h_1 = 0.08$ then the model (3) is locally asymptotically stable about the positive equilibrium point $E^*(0.052861, 0.917829, 0.204774)$ and other parameter values are same, which is shown in Figure (4). Now, we increasing the value of bifurcation parameter $h_1 = 0.133$, then the model (3) lost its stability, arise limit cycle at $E^*(0.04899, 0.920924, 0.220149)$ which shown in figure(5) .

Case-II:(Changing the parameters values both h_1 and h_2)

Now, we choose $h_1 = 0.08$ and $h_2 = 0.15$ then the model (3) will behaves the locally asymptotically stable corresponding to the interior equilibrium point $E^*(0.150488, 0.839649, 0.496640)$, which is shown in Figure (6). We fix $h_2 = 0.15$ and increase the value $h_1 = 0.35$ then the model (3) lost its stability, arise limit cycle and undergoes the Hopf-bifurcation around the positive equilibrium point $E^*(0.151952, 0.838477, 0.465983)$, it is projected in Figure 7. Then the dynamical changes of the model (3) for $h_1 \in (0.01, 0.5)$, $h_2 = 0$ and $h_1 \in (0.2, 0.5)$, $h_2 = 0.15$, respectively displayed in Figure (8) and Figure (9).

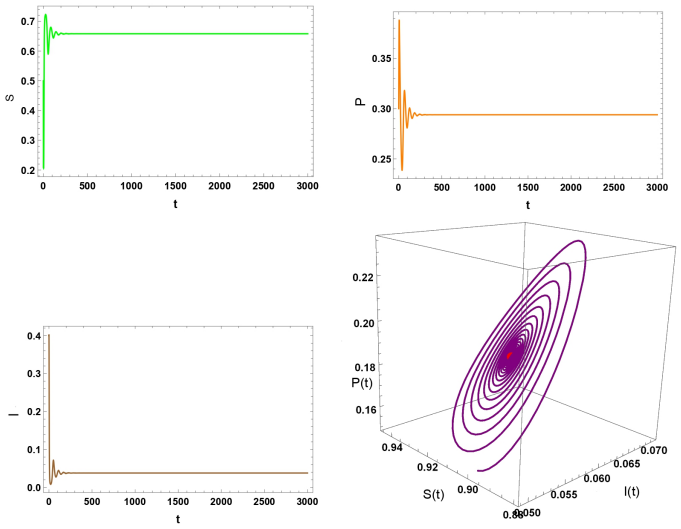

FIGURE 4. *The time analysis and phase portrait for the model(3) when* $h_1 = 0.08$ *and* $h_2 = 0$.

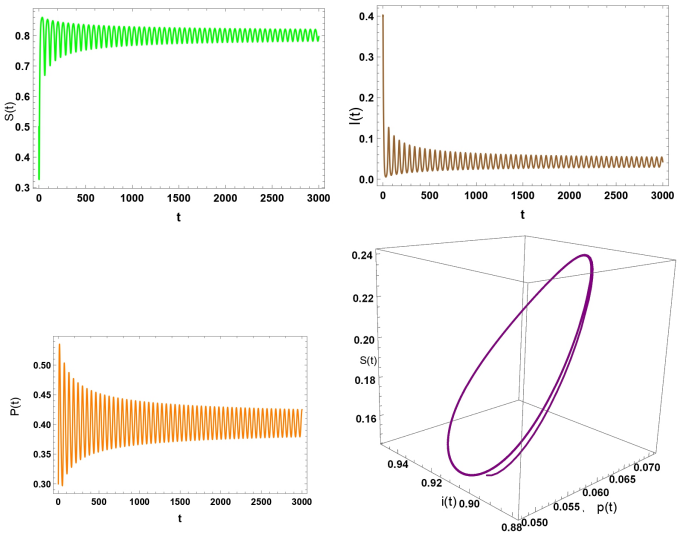

FIGURE 5. *The time analysis and phase portrait for the model (3) when* $h_1 = 0.35$ *and* $h_2 = 0$.

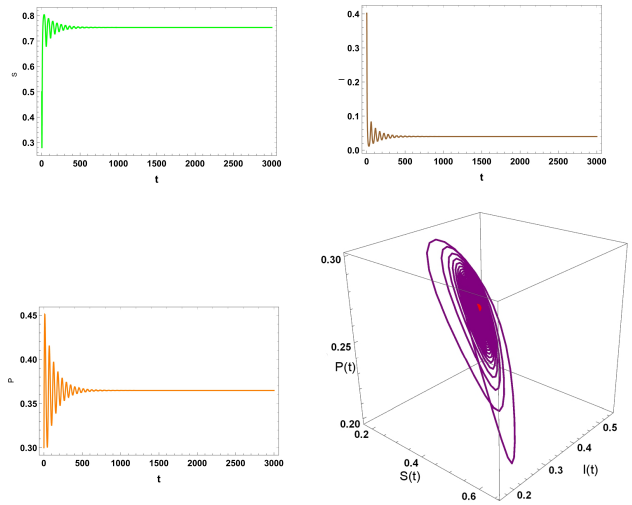

FIGURE 6. *The time analysis and phase portrait for the model (3) when* $h_1 = 0.08$ *and* $h_2 = 0.15$

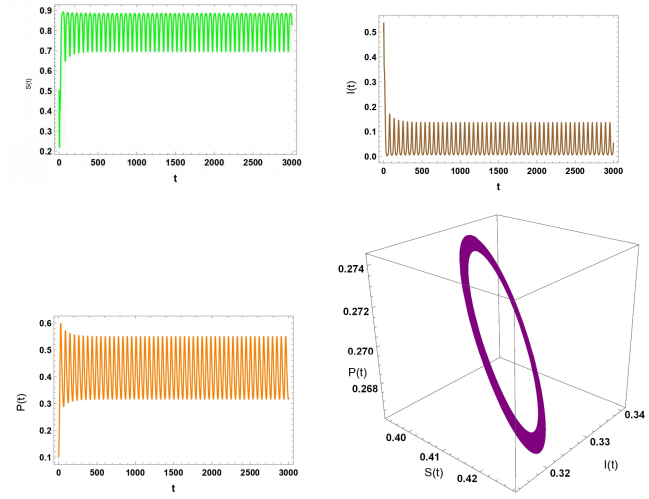

FIGURE 7. *The time analysis and phase portrait for the model (3) when* $h_1 = 0.35$ *and* $h_2 = 0.25$

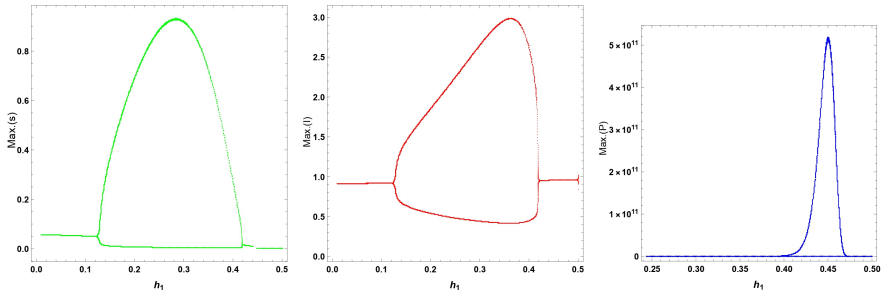

FIGURE 8. *The dynamical changes of the model(3) with* $h_1 \in (0.01, 0.5)$ *and* $h_2 = 0$

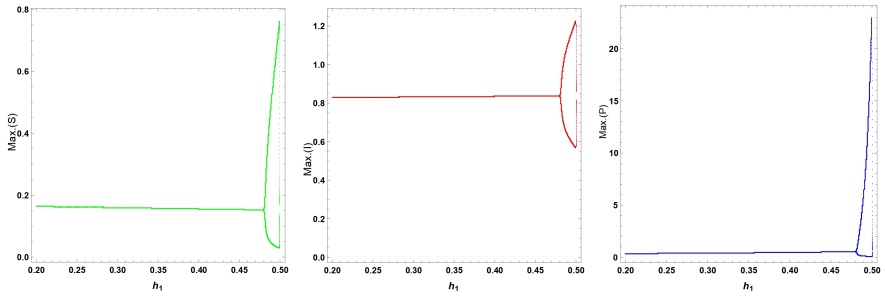

FIGURE 9. *The dynamical changes of the model (3) with* $h_1 \in (0.2, 0.5)$ *and* $h_2 = 0.15$.

8. Conclusion

We researched an eco-epidemiological system that included infection in the population density of prey and fear in the susceptible prey population density as a result of predator attacks on susceptible and diseased prey and harvesting in both prey populations. An eco-epidemic model deals with ecosystems of interacting populations among which a disease spreads. Different control measures and techniques are used to control the disease; harvesting is one of them. It is observed that harvesting plays a very crucial role in preventing the spread of infectious diseases. The positivity ensures that the population cannot be negative, while the boundedness of the solution could be understood as a natural limitation for growth due to limited resources. In addition, each biologically possible point of equilibrium can be represented (3). Furthermore, we investigated the suggested model's local stability (3) and observed the occurrence of Hopf-bifurcation, and we determined that modifying the cost of fear β and modifying the cost of harvesting h_1 has an instantaneous effect on the model's stability (3). As a result, Hopf-bifurcation constrained the developed analytical arguments around the E^* simulation findings. In

the proposed models, we deduce that the existence of fear has a higher impact on stability shifts via the Hopf bifurcation. Finally, for the non-delayed models, we examine the time series of the impact of fear and the effect of harvesting in phase portraits and bifurcation diagrams. However, the future direction of the research seems more attractive. Moving forward, we plan to conduct an in-depth analysis of the model and delve into the effect of delay on the dynamics of the model. These future studies will yield exciting results related to the effect of delay.

Author Contribution Statements The authors contributed equally to this work. All authors read and approved the final copy of this paper.

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