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

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Abstract:
This article consists of a three-species food web model that has been constructed by considering the relationships between susceptible prey, infected prey and predator species. In the absence of predators, it predicted that susceptible prey species grow logistically. It is assumed that predators consume both susceptible , infected prey and infected prey consumes susceptible prey. We consider the effect of fear on susceptible prey due to predator species.. Also, the predator consumes its prey in the form of Holling-type relationship. The positive invariance, positivity, and boundedness of the system are discussed. The criteria of all biologically feasible point of equilibria have been examined. The local stability of the systems around these point of equilibrium is investigated and global stability is analysed by suitable Lyapunov functions around these point of equilibrium. Furthermore, the occurrence of Hopf-bifurcation concerning fear (*f*) of the system has been investigated. Finally, we demonstrate some numerical simulation results to illustrate our main analytical findings.
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1 Introduction

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. Since KermackMckendrick's pioneering work on SIRS [13], epidemiological models have drawn a lot of interest from researchers. Many investigators have studied the population ecology of prey or 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 [15] and Volterra [18] 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], [16], [19]. 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 equilibria, 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 populations 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. Modelling in biological systems There are numerous of functional responses namely the type I-III of the response of Holling, type of Varley-Hassell response, type of Beddington-DeAngelis responses, type of Crowley-Martin responses; Arditi and Ginzburg's [2] 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 [5], [9], [3], [10], [6], [11]. 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 type of Crowley-Martin functional response to make the model system, more realistic and controllable in the eco-system. Several investigators 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 of 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 [17], Maynard Smith discovered that the presence of a static proportional size of refuge any size neutrally altered the static nature of equilibrium is stable the stochastic stability of a Lotka-Volterra unbiased stable model. A neutrally stable Lotka-Volterra model's dynamic stability was unaffected by the presence of a constant proportionate refuge. Tapan Kumar Kar [12] 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 examine for ecologists, bioeconomists, and resource managers

of nature. The impact of harvest is extensively used in forestry, management of wildlife and fisheries. This research uncovered a wide range of fascinating dynamics, such as point of equilibrias, analysis of bifurcation, and limit cycles. In eo-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 predatorprey interactions involve intensive searching, ratio-dependent predator-prey models are more suitable than other types [14]. Recently, [8], [1], [4] 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 [10]). 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, after the seminal work of 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 examine the relationship between the population density of some community are studied. Epidemiology systems are used to investigate the spread of illnesses between wildlife and humankind. 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, of research and 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. We investigate a Michaelis-Menten-type functional reaction coupled with a Lotka- Volterra-type predator-prey model. There has been an extensive amount of research done on the non-linear nature of an eco-epidemiological systems in the form of ratio-dependent. To address this problem, we study the impact of fear in an eco-epidemiological model with infected prey in this paper. To the great of available information, none of the scholars have explored the three-species food web model of prey-predator relationship that combines species relationship, such as Holling type II function and disease in prey populations with the influence of fear. We explore the diseased prey-predator model utilising Holling type II interaction, as well as the influence of fear on sensitive prey populations due to predators with Hopf-bifurcation, motivated by this fact. The rest of the paper is structured as follows: In Section 2, we present the mathematical analysis that has been investigated. In Section 3, some preliminary aspects of the model have been studied. Section 4 deals with the point of equilibria in boundary and their stability. In Sections 5 and 6, we determine the existence of the interior point of equilibria $E^*(u^*,v^*,w^*)$ 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,

$$\left.\begin{aligned} \frac{d\mathcal{S}}{dT} &= \frac{r_1\mathcal{S}}{1+\mathcal{F}\mathcal{P}}\left(1-\frac{\mathcal{S}+\mathcal{I}}{K}\right)-\lambda\mathcal{I}\mathcal{S}-\frac{\alpha_1\mathcal{S}\mathcal{P}}{a_1+\mathcal{S}}, \\ \frac{d\mathcal{I}}{dT} &= -d_1\mathcal{I}-\frac{b_1\mathcal{I}\mathcal{P}}{a_1+\mathcal{I}}+\lambda\mathcal{I}\mathcal{S}, \\ \frac{d\mathcal{P}}{dT} &= \frac{cb_1\mathcal{P}\mathcal{I}}{a_1+\mathcal{I}}+\frac{c\alpha_1\mathcal{P}\mathcal{S}}{a_1+\mathcal{S}}-d_2\mathcal{P}. \end{aligned}\right\} \quad (2.1)$$

Here the conditions are S(0) ≥ 0,I(0) ≥ 0 and P(0) ≥ 0 .The table displays specific biological meanings of the parameters.
The condition for the fear effect is

$$\mathcal{F}_1(\beta, p) = \frac{1}{1+\beta p} \quad (2.2)$$

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

$$\beta \ (0,p) = \mathcal{F}_1(\beta, 0) = 1$$
$$lim\beta \to \infty \mathcal{F}_1(\beta, p) = 0 = limp \to \infty \mathcal{F}_1(\beta, p)$$
$$\frac{\partial \mathcal{F}_1(\beta, p)}{\partial} < 0,$$

Table 1: Biological representation of the model

Parameters	Units	Biological representation
S	Number of components per unit area (tons)	Population density of susceptible Prey
I	Number of components per unit area (tons)	Population density of prey with infection
P	Number of components per unit area (tons)	Population density of Predator
$r1$	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's amount of consumption
$b1$	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
F	Number of components per unit area (tons)	Impact of fear

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

In this work we incorporate prey and the fear effect β. Then the system change into the non-dimensional.
Here,
$s = K\underline{S}$,$i = K\underline{I}$,$p = K\underline{P}$.
Now the (2.1) becomes,

$$\left.\begin{aligned} \frac{ds}{dt} &= \frac{rs}{1+\beta p}(1-s-i) - is - \frac{s\alpha p}{a+s} \\ \frac{di}{dt} &= is - di - \frac{\theta ip}{a+i} \\ \frac{dp}{dt} &= -\delta p + \frac{c\theta ip}{a+i} + \frac{c\alpha sp}{a+s}. \end{aligned}\right\} \quad (2.3)$$

here the conditions are,

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

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

,

3.2Existence of the solutions

For $t < 0$,

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.(2.3)

3.1Positivity of solutions

THEOREM 3.1 *In the* $\mathbb{R}^3_+$ *all the (2.3) systems solutions are non-negative . Proof.* Since $\{s(0),i(0),p(0)\} \geq 0$. hence the system (2.3) written as,

let,Z = ($s(t)$ + $i(t)$ + $p(t)$), and E(Z) = $(O_1Z,O_2Z,O_3Z)^T$,

where, $\mathcal{O}_1\mathcal{Z} = \frac{rs}{1+\beta p}(1-s-i) - si - \frac{\alpha sp}{a+s},$

$$\mathcal{O}_2\mathcal{Z} = is - di - \frac{\theta ip}{a+i},$$
$$\mathcal{O}_3\mathcal{Z} = -\delta p + \frac{c\theta ip}{a+i} + \frac{c\alpha sp}{a+s}.$$

Then (2.3) 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, $E_) \in C^\infty(R)$ for*i* = 1,2,3.

As a result, the mathematical operator O is both locally Lipschitzian and completely continuous on $\mathbb{R}^3_+$. Therefore the solution of (2.3) are exists and unique. Hence the region$\mathbb{R}^3_+$ is an invariant domain of the system (2.3) solutions are positive. □ THEOREM 3.2 *If*

$$c < 1, Max\ \tfrac{rs}{1+\beta p}(1-s) = \tfrac{r}{8}, \textbf{\textit{and}}\ \ = min(h_1, d+h_2, \delta)$$

in the $\mathbb{R}^3_+$ *all the system*

(2.3) solutions are bounded.

*Proof. s,i,*and *p* denote the model (2.3) solutions with positive criteria, hence

$\frac{ds}{dt} \le sr(1-s)$

we know that

limsup$t \to \infty$ $s \le 1$, let, Z = *s* + *i* + *p*

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

we have,

$$\frac{d\mathcal{Z}}{dt} + \beta\ z \le \frac{r}{8}.$$

Using the differential inequality theorem, we obtain

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

For $t \to \infty$, we have $0 < \mathcal{Z} < \frac{r}{\beta}$ in the $\mathbb{R}^3_+$ all the systems (2.3) solutions are uniformly bounded, for $\epsilon > 0$ are in the region,

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

4 The existence of point of equilibrias

This section examines the potential points of equilibria (2.3). The system (refequ1) has three points of equilibria in observation and points of equilibria endemic.

$$\begin{aligned}\tfrac{rs}{1+\beta p}(1-i-s) - is - \tfrac{\alpha sp}{a+s} &= 0\\ si - id - \tfrac{\theta ip}{a+i} &= 0 \quad ,\\ -\delta p + \tfrac{c\theta ip}{a+i} + \tfrac{c\alpha sp}{a+s} &= 0 \quad ;\end{aligned}$$

- The E_0(0,0,0) is the point of equilibria, which is trivial,
- E_1(1,0,0) be the free of infection and free of predator point of equilibria,
- The absence of predator point of equilibria is E_2(*s*,ˆ ˆ*i*,0), where,$\hat{s} = d, \hat{i} = \frac{r(1-d)}{r+1}$.
- endemic equilibrium is E^*(*s**,*i**,*p**), where,

$$i^* = \frac{a(a\delta + (\delta - c\alpha)s^*)}{(c\alpha s^* + (c\theta - \delta)(a+s^*))},$$

$$p^* = \frac{ac(s^* - d)(a+s^*)}{(c\alpha s^* + (c\theta - \delta)(a+s^*))},$$

and the *s*∗is the quadratic equation's unique positive root ,

$AS^2 + BS + C = 0$, where,

A =$r(\alpha c + \theta c - \delta)$,

B =$(\theta c - \delta)(ar - r) + \alpha c((1 + \beta p) - r) + a(\delta(1 + \beta p) + (\delta - c\alpha)r)$, C = $- a(r(1 + \beta p))(c\theta - \delta) + (c\alpha(1 + \beta p)(d) - a\delta((1 + \beta p) + r)))$.

. If endemic equilibrium exist for $\delta > \alpha c, r > h_1, s^* - d > h_2 > \frac{(1+r)a\delta}{a\alpha}$, and $a\delta + s^*(\delta - \alpha c)$

5 local stability analysis

In order to investigate the local stability property of the system(2.3).We first find the matrix in the form

$$\text{of Jacobian of the system } J(E) = \begin{bmatrix} n_{11} & n_{12} & n_{13} \\ n_{21} & n_{22} & n_{23} \\ n_{31} & n_{32} & n_{33} \end{bmatrix}.$$

Where,

$$n_{11} = \frac{r}{1+\beta p}(1-2s) - i\left(\frac{r}{1+\beta p}+1\right) - \frac{\alpha a p}{(a+s)^2}, n_{12} = -s(\frac{r}{1+\beta p}+1),$$
$$n_{13} = \frac{prs}{(1+\beta p)^2}(1-s-i) - \frac{\alpha s}{a+s}, n_{21} = i, n_{22} = s - d - \frac{a\theta p}{(a+i)^2}$$
$$n_{23} = -\frac{\theta i}{(a+i)}, n_{31} = \frac{ac\alpha p}{(a+s)^2}, n_{32} = \frac{ac\theta p}{(a+i)^2},$$
$$n_{33} = -\delta + \frac{c\theta i}{a+i} + \frac{\alpha cs}{a+s}.$$

THEOREM 5.1 *The E_0(0,0,0) is the point of equilibria, which is trivial, is unstable.*

Proof. The characteristic equation of the point of equilibria E_0 is,

$(\lambda_{01} - (r - h_1))(\lambda_{02} - (-d - h_2))(\lambda_{03} + \delta) = 0$, $\lambda_{01} = r, \lambda_{02} = -d, \lambda_{03} = -\delta$,

here,$\lambda_{01} > 0$ then E_0(0,0,0) is the point of equilibria, which is trivial, is unstable. □ THEOREM 5.2 E_1(1,0,0) *be the free of infection and free of the predator point of equilibria, is unstable due to the table value of the numerical simulation . Proof.* The characteristic equation of the point of equilibria E_1 is,

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

here,E_1(1,0,0) being free of infection and free of the predator point of equilibria, is unstable because $1 - d$ is never negative due to the table value of numerical simulation. □

THEOREM 5.3 *The equilibrium E_2(s,ˆ ˆi,0) which absence of predator is asymptotically stable if* $\delta > c(\theta + \alpha)$

$$\text{*Proof.* Matrix in the form of Jacobian at} E_3 \text{ is } J(E_3) = \begin{bmatrix} q_{11} & q_{12} & q_{13} \\ q_{21} & q_{22} & q_{23} \\ q_{31} & q_{32} & q_{33} \end{bmatrix},$$

where,

$$q_{11} = r(1-2\hat{s}) + i(r+1), q_{12} = (-1-r)\hat{s}, q_{13} = -\frac{\alpha\hat{s}}{a+s}$$
$$q_{21} = \hat{i}, q_{22} = 0, q_{23} = -\frac{(1-m)\theta\hat{i}}{a+\hat{i}(1-m)},$$
$$q_{31} = 0, q_{32} = 0, q_{33} = \frac{c\alpha\hat{s}}{a+\hat{s}} - \delta + \frac{(1-m)c\theta\hat{i}}{a+(1-m)\hat{i}}.$$

Here, the characteristic equation of the above matrix in the form of Jacobian is, $\lambda^3+A\lambda^2+B\lambda+C = 0$. Here,
L $= -q11 - q33$,
M $= -q21q12 + q33q11$, N $= q12q21q33$.

If and only if L,N and LM − N are positive, then the negative real parts are the roots of the above characteristic equation. According to the Routh-Hurwitz criterion.
now, LM−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_3 is locally asymptotically stable provided the above condition in theorem satisfied. □

THEOREM 5.4 The endemic or positive point of equilibria E^* is asymptotically stable.

Proof. Matrix in the form of Jacobian E^* is $J(E^*) = \begin{bmatrix} r11 & r12 & r13 \\ r21 & r22 & r23 \\ r31 & r32 & r33 \end{bmatrix}$, where,

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

Here, the characteristic equation of the Matrix in the form of Jacobian E^* is
$\lambda^3 + F\lambda^2 + G\lambda + H = 0$, (5.1)
here,
F $= -r11 - r33$,G $= -r21r12 + r22r11 - r13r31 + r23r32$, H $= r13(-r22r31 + r21r32) + r23(r12r31 - r11r32)$.
If F > 0,H > 0,FG −H > 0. The negative real parts are the roots of the above characteristic equation if and only if F,H and FG − H are non-negative, according to the Routh-Hurwitz criterion.. The E^* is locally asymptotically stable. □

6 Globel stability Analysis

THEOREM 6.1 The equilibrium point E_1 is globally asymptotically stable when $d < 1$ and $\frac{\alpha pc}{\delta(a+s)} < 1$. *Proof.* A Lyapunov function is defined as

$$V(s,i,p) = (1+\beta p)[s-1-lns] + i + \left(\tfrac{1}{c}\right)p.$$

Applying the derivative, we obtain

$$\begin{aligned}\frac{dV}{dt} &= \left(\frac{s-1}{s}\right)\frac{ds}{dt} + \frac{di}{dt} + \frac{1}{c}\frac{dp}{dt} \\ &= (1+\beta p)(s-1)\left[r(1-s-i) - i - \frac{\alpha sp}{a+s}\right] + \left[si - di - \frac{\theta ip}{a+i}\right] \\ &+ \frac{1}{c}\left[-\delta p + \frac{c\theta ip}{a+i} + \frac{\alpha sp}{a+s}\right] \\ &= -r[s^2 - 2s + 1] - rsi(1-s) - i(d-1) - p\left[\frac{\delta}{c} - \frac{\alpha sp}{a+s}\right] \\ &\leq -r[s-1]^2 - ri(1-s) + i(d-1) - p\left[\frac{\delta}{c} - \frac{\alpha p}{a+s}\right]\end{aligned}$$

Thus, $d < 1$.
Therefore, E_1 is globally asymptotically stable. □
THEOREM 6.2 The Predator-free equilibrium point E_2 is globally asymptotically stable if

Proof. A Lyapunov function is defined as

$$\mathcal{V}(s,i,p) = (1+\beta p)\left[s - \hat{s} - \hat{s}ln\tfrac{s}{\hat{s}}\right] + \left[i - \hat{i} - \hat{i}ln\tfrac{i}{\hat{i}}\right] + \tfrac{1}{c}p$$

$$\frac{dV}{dt} = (1+\beta\ \ p)\left(\frac{s-\hat{s}}{s}\right)\frac{ds}{dt} + \frac{i-\hat{i}}{i}\frac{di}{dt} + \frac{1}{c}\frac{dp}{dt}$$
$$= (s-\hat{s})\left[r(1-s-i) - i - \frac{\alpha p}{(a+s^*)}\right] + (i-\hat{i})\left[si - di - \frac{\theta ip}{a+i}\right]$$
$$+ \frac{1}{c}\left[-\delta p + \frac{c\theta ip}{a+i} + \frac{c\alpha sp}{(a+s^*)}\right]$$
$$= -rs^2 + rs - ris - r\hat{s} + rs\hat{s} + ri\hat{s} + \hat{i}s + \frac{\alpha p\hat{s}}{(a+s^*)} - di - s\hat{i} + d\hat{i}$$

$$- \frac{\theta\hat{i}p}{a+i} - \frac{\delta p}{c} + \frac{\delta\hat{p}}{c} - \frac{\alpha l\hat{p}}{(a+s^*)p)}$$

$$\leq r(s-\hat{s}) - rs(s-\hat{s}) - ri(s-\hat{s}) - i(d-\hat{s}) - \frac{\delta}{c}(p-\hat{p}) + \frac{\theta\bar{i}}{a+i} - \frac{\alpha\bar{s}}{(a+s^*)}$$

Therefore, the predator-free equilibrium point E_2 is globally asymptotically stable. □

THEOREM 6.3 The interior equilibrium point E^* is globally asymptotically stable if *Proof.* A Positive Lyapunov function is defined as

$$\mathcal{V}(s,i,p) = \left[s - s^* - s^* ln\frac{s}{s^*}\right] + d_1\left[i - i^* - i^* ln\frac{i}{i^*}\right] + d_2\left[p - p^* - p^* ln\frac{p}{p^*}\right]$$

$$\frac{dV}{dt} = \left(\frac{s-s^*}{s}\right)\frac{ds}{dt} + d_1\left(\frac{i-i^*}{i}\right)\frac{di}{dt} + d_2\left(\frac{p-p^*}{p}\right)\frac{dp}{dt}$$
$$= (s-s^*)\left[r(1-s-i) - i - \frac{\alpha p}{(a+s^*)}\right] + d_1(i-i^*)\left[s - d - \frac{\theta p}{a+i}\right]$$
$$+ d_2(p-p^*)\left[-\delta + \frac{c\theta i}{a+i} + \frac{c\alpha s}{(a+s^*)}\right]$$
$$\leq -(s-s^*)\left[r\{si - (s^*+i^*)\} - (i-i^*) - \alpha\left\{\frac{p}{(a+s^*)} - \frac{p^*}{(a+s^*)}\right\}\right]$$
$$- d_1(i-i^*)\left\{\left[\frac{p}{a+i} - \frac{p^*}{(a+i^*)}\right] - (s-s^*)\right\}$$
$$- d_2(p-p^*)\left\{\frac{c\theta a(i-i^*)}{(a+i^*)}\right\}$$

we conclude that the interior equilibrium point E^* is globally asymptotically stable. □

7 Hopf-Bifurcation Analysis

In this part, we use the fear *beta* effect to analyse the model's bifurcation. Using the bifurcating factor *beta*, the following theorem shows the presence of Hope-bifurcation.

THEOREM 7.1 *The model (refequ1) confronts Hope-bifurcation if the bifurcation parameter beta surpasses a critical point. The following hope-bifurcation conditions arise at beta = beta∗:*

1.$A_1(\beta^*)A(\beta^*) - A_3(\beta^*) = 0$.

2.$_{d\beta}^{d}(Re(\lambda(\beta)))|_{\beta=\beta^*} \neq 0$ *Here lambda is the zero of the parametric solution correlated with the equilibria's interior point.*

Proof. For $\beta = \beta^*$, let the equation of characteristic (5.1) is in the form

$$(\lambda^2(\beta^*) + A_2(\beta^*))(\lambda(^*) + A_1(\beta^*)) = 0. \quad (7.1)$$

This indicates that the roots of the preceding equation are $\pm i^p A_2(\beta^*)$ and $-A_1(\beta^*)$.To achieve the Hopf-bifurcation at $\beta = \beta^*$ the following transversality criterion must be fulfilled.

$_{d\beta}^{d}* (Re(\lambda(\beta^*)))| \neq 0$.

For β , the above equation (7.1) has general roots $\lambda_1 = r(\beta) + is(\beta)$, $\lambda_2 = r(\beta) - is(\beta)$, $\lambda_3 = -A_1(\beta)$.

Weather check the criteria $_{d\beta}^{d}{}_* (Re(\lambda(\beta^*)))| \neq 0$.

Let $\lambda_1 = r(\beta) + is(\beta)$ in the (7.1), we get

$$C(\beta) + iD(\beta) = 0.$$

Where,

$C(\beta) = r^3(\beta) + r^2(\beta)A_1(\beta) - 3r(\beta)s^2(\beta) - s^2(\beta)A_1(\beta) + A_2(\beta)r(\beta) + A_1(\beta)A_2(\beta)$, $D(\beta) = A_2(\beta)s(\beta) + 2r(\beta)s(\beta)A_1(\beta) + 3r^2(\beta)s(\beta) + s^3(\beta)$.

In order to fulfill the (7.1) we must have $C(\beta) = 0$ and $D(\beta) = 0$, then calculating C and D with respect to β. We have

$$\frac{d\mathcal{A}}{d} = \varsigma_1(\ )r'(\ ) - \varsigma_2(\ )s'(\ ) + \varsigma_3(\ ) = 0 \quad , (7.2)$$

$$\frac{d\mathcal{B}}{d} = \varsigma_2(\ )r'(\ ) + \varsigma_1(\ )s'(\ ) + \varsigma_4(\ ) = 0 \quad , \quad (7.3)$$

where,
$\varsigma_1 = 3r^2(\beta) + 2r(\beta)A_1(\beta) - 3s^2(\beta) + A_2(\beta)$, $\varsigma_2 = 6r(\beta)s(\beta) + 2s(\beta)a_1(\beta)$, $\varsigma_3 = r^2(\beta)A'_1(\beta) + s^2(\beta)A_1'(\beta) + A'_2(\beta)r(\beta)$,
$\varsigma_4 = A_2(\beta)s(\beta) + 2r(\beta)s(\beta)A_1(\beta)$.
On multiplying (7.2) by $\varsigma_1(\beta)$ and (7.3) by $\varsigma_2(\beta)$ respectively

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

$$s(\ ) = \sqrt{\mathcal{A}_2(\ )} \text{ a} \quad * \text{ on } \varsigma_1(\ ), \varsigma_2(\ ), \varsigma_3(\ ), \text{ and } \varsigma_4(\ ) \quad (7.4)$$

Substituting $r(\beta) = 0$ and, we obtain

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

The equation (7.4), implies

$$r'(\ *) = \frac{\mathcal{A}_3'(\ *) - (\mathcal{A}_1(\ *\mathcal{A}_2(\ *)))}{2(\mathcal{A}_2(\ *) + \mathcal{A}_1^2(\ *))} \quad , (7.5)$$

if $\mathcal{A}_3'(\ *) - (\mathcal{A}_1(\ *)\mathcal{A}_2(\ *))' \neq 0$ which implies that
$\frac{d}{d^*}(Re(\lambda(\ *)))| \neq 0$, and $\lambda_3(\ *) = -\mathcal{A}_1(\ *) \neq 0$.

Therefore the condition $\mathcal{A}_3'(\ *) - (\mathcal{A}_1(\ *)\mathcal{A}_2(\ *))' \neq 0$ It has been guaranteed that the transversality criterion is satisfied, hence the model (2.3) has attained the Hopf-bifurcation at $\beta = f*$. □

8 Numerical Simulations

In this section, several numerical experiments on the system (2.3) are carried out to verify the mathematical findings. The rate of fear β and predation rate are the essential parameters in this study, and they will be used as control parameters. For the specified fixed parameter values, the numerical simulation is carried out using the MATLAB and MATHEMATICA software packages.

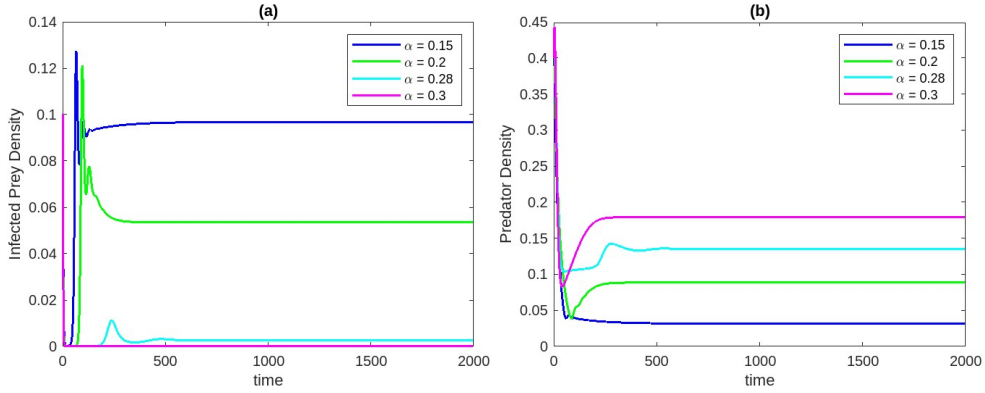

Figure 1: *Except for β = 0.3, the population of infected prey, and predators for the parametric values listed in the table is as follows. Where α = 0.15,0.2,0.28,0.3*

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

8.1Effect of varying the predation rate α

Let β = 0.3 For the parameters specified in Table 2. without infection point of equilibria E_2 and the endemic point of equilibria E^* exists for 0.1 < α < 035, respectively, for the given parametric values. The stability of for α = 0.3 and α = 0.28 is shown in Figure(2).

Figure (1) shows that as the predator population grows, so does the predation rate *alpha* and the number of infected prey.

8.2Effect of varying the level of fear β

For the parameters specified in Table 2, with α = 0 without predator point of equilibria E_2 and the endemic point of equilibria E^* exists for 0.1 < f < 1, respectively.

Figure (3) demonstrates an increase in the rate of fear β and a decrease in the population density of infected prey. As the population density of susceptible prey grows, so does the population of predators.

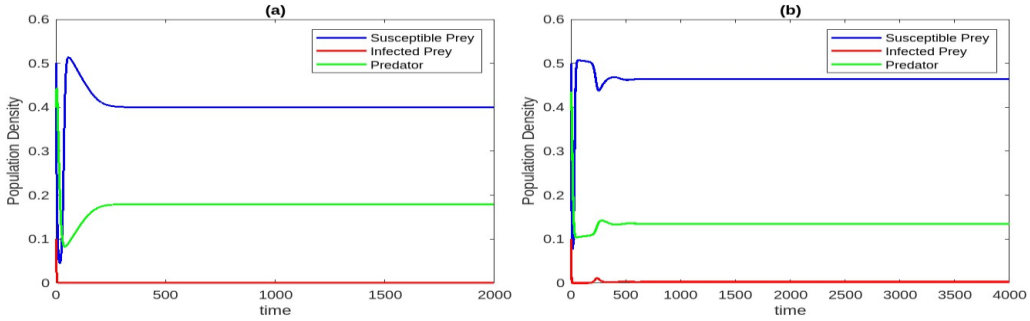

Figure 2: *Solutions of time series (2.3) around the point of equilibria E_2 and the point of equilibria E_4.*

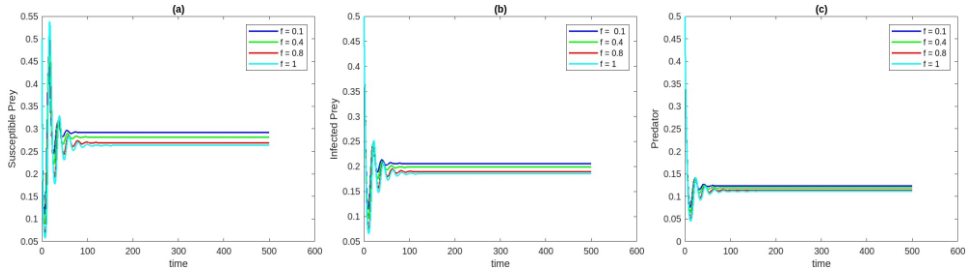

Figure 3: Population concentrations (2.3) where α = 0.2. Where β = 0.1,0.4,0.8,1.

8.3 Bifurcation of predation rate α

If β = 0.3, then the model (2.3) is asymptotically stable about the positive point of equilibria E^*(0.52861,0.0917829,0.204774) and other parameter values are the same, which is shown in Figure (4). Now, we increased the value of the bifurcation parameter, β = 0.6, and the model (2.3) lost its stability, arising a limit cycle at E^*(0.4899,0.0920924,0.220149), which is shown in figure(5). The model (refequ1) then meets the transversality criteria for $(Re(\lambda(\beta)))|_{\beta=\beta^*}$ = 0.002185 $\neq$ 0. The figure (6) shows the behavioural shifts of the system (2.3) at rate of predation, β = 0.6.

9 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. In addition, each biologically possible point of equilibria can be represented (2.3). Furthermore, we investigated the suggested model's local stability (refequ1) and observed the occurrence of Hopf-bifurcation, and we determined that modifying the cost of fear β has an instantaneous effect on the model's stability (refequ1). As a result, Hopf-bifurcation constrained the developed analytical arguments

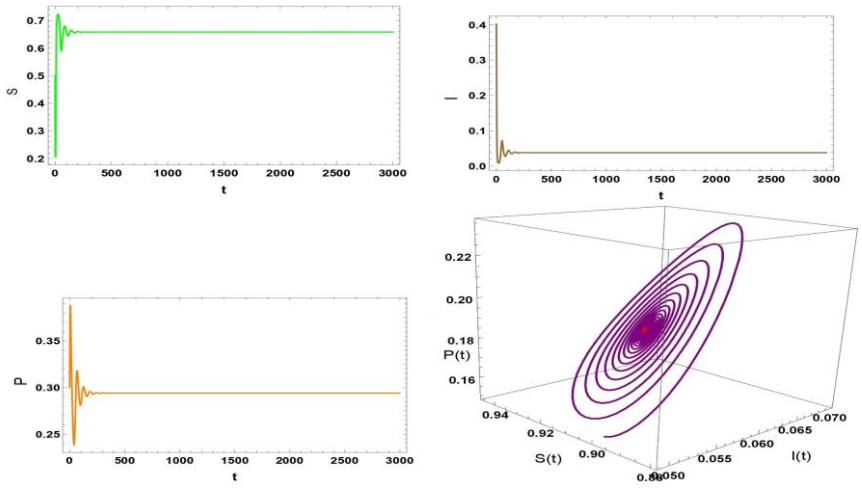

Figure 4: The time analysis of model(2.3) and phase portrait for the model (2.3) when $\beta = 0.3$.

around the $E*$ simulation findings. In the proposed models, we deduce that the existence of dread 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, phase portraits, and bifurcation diagrams.

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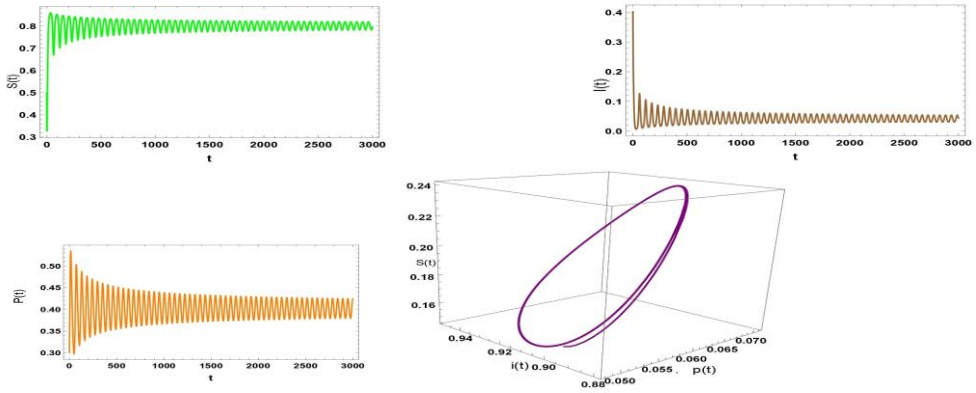

Figure 5: The time analysis of model(2.3) and phase portrait for the model (2.3) when $\beta = 0.6$.

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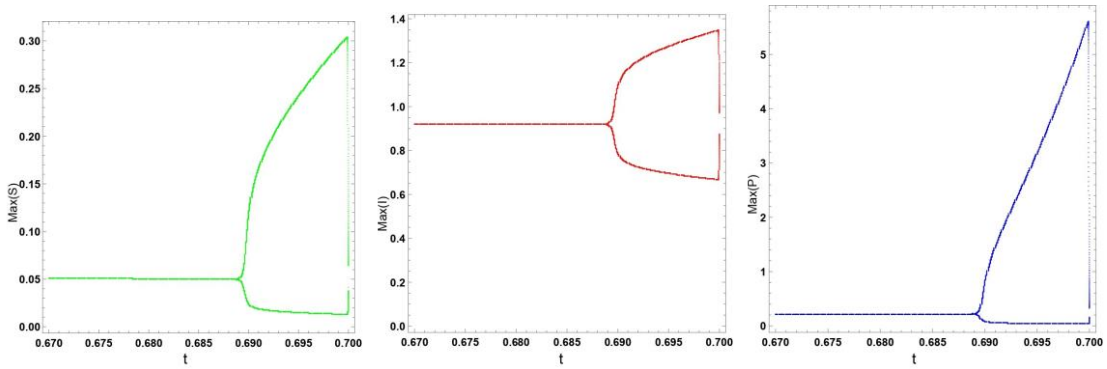

Figure 6: The dynamical change of the model (2.3) at $\beta = 0.6$

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