Backward bifurcation, oscillations and chaos in an eco-epidemiological model with fear effect

Amar Sha, Sudip Samanta, Maia Martcheva and Joydev Chattopadhyaya

ABSTRACT
This paper considers an eco-epidemiological model with disease in the prey population. The disease in the prey divides the total prey population into two subclasses, susceptible prey and infected prey. The model also incorporates fear of predator that reduces the growth rate of the prey population. Furthermore, fear of predator lowers the activity of the prey population, which reduces the disease transmission. The model is well-posed with bounded solutions. It has an extinction equilibrium, susceptible prey equilibrium, susceptible prey-predator equilibrium, and coexistence equilibria. Conditions for local stability of equilibria are established. The model exhibits fear-induced backward bifurcation and bistability. Extensive numerical simulations show the presence of oscillations and occurrence of chaos due to fear induced lower disease transmission in the prey population.

1. Introduction
Predator–prey interactions are a central topic of discussion in studying ecological communities. These interactions are often altered in nature due to the presence of infectious disease that affects the prey, the predator, or both. Understanding the predator–prey–pathogen dynamics requires the development and analysis of population models where one or more of the main populations are subjected to an infection. Models that incorporate disease in ecological communities are called eco-epidemiological models, and represent a natural extension of more classical population interaction models [35]. The first eco-epidemiological model with disease in the prey was introduced by Anderson and May [1]. This early model was followed by the work of a number of researchers in eco-epidemiology [2,4,9,15,16]. Eco-epidemiology now is a branch of mathematical biology connecting ecology with epidemiology. Although the literature of eco-epidemiology is rich enough, the impact of fear of predators on eco-epidemiological systems is not properly studied yet. Recent experimental results revealed that fear of predators can reduce
the growth rate of prey population and change the foraging behaviour [32,39]. In general, basal prey lower their predatory activity in the presence of their predators. According to optimal foraging theory, prey increase their survival probability by avoiding high-risk grazing zone and remain starving or grazing on the lower intake zone [6]. Such lower foraging activity reduces the chance of contacts between infected and susceptible individuals. Therefore, fear of predator can have a great impact on the dynamics of eco-epidemiological systems.

In classical predator–prey theory, the impact of predators on the prey population has been described through only direct killing of prey by predators, because the effect of direct killing on prey numbers can be directly observed. However, an emerging view is that the indirect effect of predator on prey numbers may be far greater than direct predation. Due to predation risk, all prey show a variety of anti-predator responses such as changes in foraging behaviours, changes in habitat usage, etc. [11]. Zanette et al. [39] have performed experimental studies and showed that predator–prey population dynamics has been affected enormously by the cost of fear. They eliminated direct predation by protecting every nest in the experiment with both electric fencing and seine netting and began predator play back several weeks before the first egg of the season was laid and continued predator call and sound broadcasts throughout the 130-days breeding season. They observed the reduction in numbers of eggs, hatchlings, and fledglings in the successive generations. They observed that the number of offspring was reduced by 40%. They demonstrated that the prey’s perception of predation risk alone is powerful enough to affect the population growth rate [39]. Other evidence suggest that fear can affect populations like snowshoe hares [28] and dugongs [37]. Recently, another field experiment performed by Suraci et al. [32] demonstrated that the fear of large carnivores can provide significant service in conserving the ecosystem function. In their experiment, they manipulated fear using month-long playbacks of large carnivore vocalizations. The experiment was conducted on wild, free-living mesocarnivores on several small coastal Gulf Islands. As a result, they showed that fear of large carnivore reduced mesocarnivore foraging and increased vigilance. They also observed mesocarnivore’s prey (interdial crabs, interdial fish, polychaete worms, subtidal red rocks crabs) were benefited due to fear of large carnivores. The authors concluded that the lower trophic level (prey) is benefited from the fear among mesocarnivores, and it could be useful in ecosystem conservation.

Recently, Wang et al. [36] modified the Rosenzweig-MacArthur predator–prey model [23] by considering the cost of fear in prey. Incorporating the cost of fear, the authors obtained both supercritical and subcritical Hopf bifurcation which is in contrast with classical predator–prey models where Hopf bifurcation only be supercritical. The results also showed that high levels of fear stabilize the predator–prey system by excluding the existence of periodic solutions.

On the other hand, during epidemic outbreak fear of infection can shape the force of infection by inducing behavioural changes and ultimately reduce disease prevalence. Capasso and Serio [7] studied an SIR model, where they considered the saturation phenomena for large numbers of infectives to capture the psychological effects. Recently, Epstein et al. [14] studied an SIR model with fear. Individuals with fear are assumed to respond with only two actions, namely self-isolation and spatial flight [14]. The authors studied spatial flight as a behavioural response and concluded that small levels of fear-inspired flight can
dramatically reduce the epidemic size. Although the impact of fear in the epidemic outbreak has been studied extensively, however, there is lack of understanding how the fear of predators can change the dynamics of eco-epidemiological systems.

In the present paper, we have considered an eco-epidemiological model with disease in the prey population. We assume that fear of predator reduces the reproduction rate [36] of the prey population. We also assume that fear of predator lowers the foraging activity and increases the vigilance in the prey population [32], which consequently reduces the probability of getting infected. Lower activity in prey implies lower chance of contact between susceptible and infected prey populations. In the present work, we study an eco-epidemiological model where prey population is subjected to disease infection and fear can reduce prey growth rate, lower foraging activity, which consequently lowers the force of infection.

The rest of the paper is organized as follows: In Section 2, we formulate the model in which the prey population responds behaviourally with fear to perceived predation risks. In Section 3, we check the positivity and boundedness of solutions of our model. In Section 4, we determine the equilibria and analyze our model with fear. In Section 5, we establish the presence of backward bifurcation for the predator-free equilibrium and determine rigorously the direction of the bifurcation. In Section 6, we perform some numerical simulations which reveal that the fear effect plays a crucial role in eco-epidemiology. At the end of this paper, we discuss the biological significance of our mathematical results and conclusions.

2. Mathematical formulation

Here we consider a predator–prey system, where the prey population is subjected to infection. Let \( u(t) \) be the prey population density and \( v(t) \) be the predator population density at time \( t \). We consider that birth rate of prey is \( r_0 \), \( d \) is the natural death rate of prey and \( a \) represents the death rate due to intra-species competition. To incorporate the fear phenomena, we multiply the reproduction term i.e. birth rate \( (r_0) \) of susceptible individuals with a decreasing function of the predator population size, \( f(k_1, v) = 1/(1 + k_1v) \), suggested by Wang et al. [36]. Here \( k_1 \) be the level of fear that reduces the growth rate of susceptible prey. From the biological point of view, \( f(k_1, v) \) is appropriate since

\[
\begin{align*}
f(0, v) &= 1, & f(k_1, 0) &= 1, & \lim_{k_1 \to \infty} f(k_1, v) &= 0, & \lim_{v \to \infty} f(k_1, v) &= 0, \\
\frac{\partial f(k_1, v)}{\partial k_1} &< 0, & \frac{\partial f(k_1, v)}{\partial v} &< 0.
\end{align*}
\]

Next we divide prey population into two subclasses, susceptible prey \( (u_1) \) and infected prey \( (u_2) \). We assume that only susceptible prey can reproduce and the disease is not genetically inherited. We also assume that infected prey do not compete for the resource for being weak due to disease infection. The susceptible prey becomes infected only through a contact with the infectious prey at a rate \( \beta \). We model the incidence through mass action law \( \beta u_1 u_2 \). We consider that fear of predator reduces the foraging activity among prey [32], which in turn reduces the disease transmission rate. We assume that scared prey spreads the disease at a rate \( \beta/(1 + k_2v) \), where \( k_2 \) be the cost of fear that lowers disease transmission.

In predator–prey theory, choice of a predator functional response is very crucial for modelling predator–prey dynamics when prey is divided into susceptible and infected
A. SHA ET AL.

compartments. Researchers [21,24,26] considered Holling type II functional responses for multiple prey populations, where all prey populations contribute to the saturation. Therefore, the functional responses are given by \( f_1(u_1) = \frac{p_1 u_1}{1 + q_1 u_1 + q_2 u_2} \) and 
\( f_2(u_2) = \frac{p_2 u_2}{1 + q_1 u_1 + q_2 u_2} \), where both the susceptible and infected prey contribute to saturation. Here \( p_1 \) and \( p_2 \) are the predator’s attack rate on susceptible and infected prey and 1/\( q_1 \) and 1/\( q_2 \) are the half-saturation constants, respectively.

From the above assumptions we obtain the following system of nonlinear differential equations:

\[
\begin{align*}
\frac{du_1}{dt} &= \frac{r_0 u_1}{1 + k_1 v} - du_1 - au_1^2 - \frac{p_1 u_1 v}{1 + q_1 u_1 + q_2 u_2} - \frac{\beta u_1 u_2}{1 + k_2 v} \\
\frac{du_2}{dt} &= \beta u_1 u_2 - \mu u_2 - \frac{p_2 u_2 v}{1 + q_1 u_1 + q_2 u_2} \\
\frac{dv}{dt} &= \frac{c_1 p_1 u_1 + c_2 p_2 u_2}{1 + q_1 u_1 + q_2 u_2} - mv,
\end{align*}
\]

where, \( c_1 \) and \( c_2 \) are the conversion efficiencies of captured susceptible and infected prey into predator biomass. \( \mu \) and \( m \) are the death rates of infected prey and predator populations, respectively. The system has to be analysed with the initial conditions \( u_1(0) \geq 0, u_2(0) \geq 0, v(0) \geq 0 \) and all parameters are assumed non-negative. We show below that the model is mathematically well posed in the positively invariant region \( X = \{ (u_1, u_2, v) \mid u_i \geq 0, i = 1, 2, v \geq 0 \} \) and solutions in \( X \) exist for all positive time.

3. Positivity and boundedness of solutions

We define \( X = (u_1, u_2, v) \in \mathbb{R}_+^3 = \{ (u_1, u_2, v) \mid u_i \geq 0, i = 1, 2, v \geq 0 \} \), according to ecological significance. The right-hand side of system (1) is continuously differentiable and locally Lipschitz in the first quadrant \( X = \{ (u_1, u_2, v) : u_1, u_2, v \geq 0 \} \). Therefore, Theorem A.4 in [33] implies that the solutions of the initial value problems with nonnegative initial conditions exist on the interval \( [0, b)[0, \infty) \)

**Lemma 3.1:** If \( c_1 < 1 \) and \( c_2 < 1 \), then the solutions of system (1) that start from initial conditions in \( \mathbb{R}_+^3 \) eventually enter the region \( S \) defined by the set \( S = \{ (u_1, u_2, v) \in \mathbb{R}_+^3 | F = \delta + H/N \) for some \( \delta > 0 \).

**Proof:** We define a function \( F \) as

\[
F(t) = u_1(t) + u_2(t) + v(t), \quad t \geq 0.
\]

The derivative of (2), with respect to time is

\[
\dot{F} = \frac{r_0 u_1}{1 + k_1 v} - du_1 - au_1^2 - \frac{p_1 u_1 v}{1 + q_1 u_1 + q_2 u_2} (1 - c_1) - \mu u_2 - \frac{p_2 u_2 v}{1 + q_1 u_1 + q_2 u_2} (1 - c_2) - mv.
\]

\[
\dot{F} = \frac{r_0 u_1}{1 + k_1 v} - du_1 - au_1^2 - \frac{p_1 u_1 v}{1 + q_1 u_1 + q_2 u_2} (1 - c_1) - \mu u_2 - \frac{p_2 u_2 v}{1 + q_1 u_1 + q_2 u_2} (1 - c_2) - mv.
\]
where the dot represents the derivative with respect to time. Assume \( c_1 < 1 \) and \( c_2 < 1 \). Then we have

\[
\dot{F} \leq r_0 u_1 - du_1 - au_1^2 - \mu u_2 - mv. \quad \blacksquare
\]

Now we choose an arbitrary positive real number \( N \) for which \( \dot{F} + NF \leq u_1(-au_1 + r_0 - d + N) - u_2(\mu - N) - v(m - N) \) holds. For simplicity we take \( 0 < N \leq \min\{\mu, m\} \). Therefore, \( \dot{F} + NF \leq u_1(-au_1 + r_0 - d + N) \). Here the maximum value of \( u_1(-au_1 + r_0 - d + N) \) is \((r_0 - d + N)^2/4a\), which is a positive constant say, \( H \). So \( \dot{F} + NF \leq H \). By a theorem of differential inequalities, we have

\[
0 < F(u_1, u_2, v) \leq \frac{H(1 - \exp(-Nt))}{N} + F(u_1(0), u_2(0), v(0)) \exp(-Nt).
\]

Thus, for large values of \( t \) we have \( 0 < F \leq H/N \). Consequently, solutions of the system that are initiating in \( R_+^3 \) eventually lie in the region \( S \) defined by

\[
S = \left\{ (u_1, u_2, v) \in R_+^3 \mid F = \frac{H}{N} \text{ for some } \delta > 0 \right\}.
\]

4. General stability analysis

4.1. Equilibrium analysis

System (1) admits the following five non-negative equilibria.

(i) The trivial equilibrium \( E_0 = (0, 0, 0) \) which is always exists.

(ii) The disease-free and predator–free prey equilibrium \( E_1 = (\tilde{u}_1, 0, 0) \), where \( \tilde{u}_1 = (r_0 - d)/a \). \( E_1 \) is feasible if \( r_0 - d > 0 \), i.e. if the reproduction rate of susceptible prey is larger than the death rate of susceptible prey. Under the condition \( r_0 > d \), \( E_1 \) always exists.

(iii) The predator–free equilibrium \( E_2 = (\tilde{u}_1, \tilde{u}_2, 0) \), where \( \tilde{u}_1 = \mu/\beta \), \( \tilde{u}_2 = (r_0 - d - a\mu/\beta)/\beta \). The predator–free equilibrium \( E_2 \) is feasible if \( r_0 - d > a\mu/\beta \), i.e. if the intrinsic growth rate of susceptible prey is larger than a threshold value determined by the ratio of death rate of infected prey and the disease transmission rate.

(iv) The disease-free predator–prey equilibrium \( E_3 = (\tilde{u}_1, 0, \tilde{v}) \), where \( \tilde{u}_1 = m/(c_1p_1 - mq_1) \) and \( \tilde{v} \) is the root of the given equation,

\[
\frac{r_0}{1 + k_1 v} - d - a\tilde{u}_1 = \frac{mv}{c_1 \tilde{u}_1}.
\]

This quadratic equation has a unique solution \( \tilde{v} > 0 \) if \( r_0 - d - a\tilde{u}_1 > 0 \). The disease-free predator–prey equilibrium \( E_3 \) is feasible if \( r_0 > d + am/(c_1p_1 - mq_1) \) and \( c_1p_1 > mq_1 \), i.e. if the intrinsic growth rate of susceptible prey is larger than the sum of the death rate of susceptible prey and density dependent death rate \( a \) times the susceptible prey at equilibrium \( \tilde{u}_1 \).
(v) The coexistence equilibrium \( E^* = (u_1^*, u_2^*, v^*) \), where \((u_1^*, u_2^*, v^*)\) is a positive solution of the system of equations

\[
\begin{align*}
\frac{r_0}{1 + k_1 v} - d - au_1 &- \frac{p_1 v}{1 + q_1 u_1 + q_2 u_2} - \frac{\beta u_2}{1 + k_2 v} = 0 \\
\beta u_1 &- \mu - \frac{p_2 v}{1 + q_1 u_1 + q_2 u_2} = 0 \\
c_1 p_1 u_1 + c_2 p_2 u_2 &- m = 0.
\end{align*}
\tag{3}
\]

To prove its existence we define the following reproduction numbers: First, we define predator invasion number of the infectious equilibrium of the prey as

\[
R_p = \frac{1}{m} \left[ \frac{c_1 p_1 \tilde{u}_1 + c_2 p_2 \tilde{u}_2}{1 + q_1 \tilde{u}_1 + q_2 \tilde{u}_2} \right].
\]

Second, we define disease invasion number of the predator–prey disease-free equilibrium

\[
R_0 = \left[ \frac{\beta \tilde{u}_1}{1 + k_2 \tilde{u}} + \frac{1}{\mu + \frac{p_2 \tilde{v}}{1 + q_1 u_1}} \right].
\]

**Theorem 4.1:** Assume that \( E_2 \) and \( E_3 \) exist and \( \bar{u}_1 > \tilde{u}_1 \). Assume also \( R_p > 1 \) and \( R_0 > 1 \). Then there exists at least one coexistence equilibrium \( E^* \).

**Proof:** From \( R_p > 1 \) and \( R_0 > 1 \), we have

\[
\frac{c_1 p_1 \tilde{u}_1 + c_2 p_2 \tilde{u}_2}{1 + q_1 \tilde{u}_1 + q_2 \tilde{u}_2} > m. \tag{4}
\]

From third equation in system (3) we can express \( u_1 \) as a function of \( u_2 \). This follows from the implicit function theorem. Therefore,

\[
u_1 = f(u_2) = \frac{(mq_2 - c_2 p_2)u_2}{c_1 p_1 - mq_1} + \frac{m}{c_1 p_1 - mq_1},
\]

where \( f \) is a continuous and increasing function for all \( u_2 \geq 0 \) where \( \tilde{u}_1 = f(0) \) and exists for all \( u_2 \geq 0 \). Since \( c_1 p_1 - q_1 m > 0 \), we have assumed for simplicity

\[
\frac{c_1 p_1}{q_1} > m > \frac{c_2 p_2}{q_2}.
\]

Therefore, predator can persist on \( u_1 \) alone but not on \( u_2 \) alone. From the first equation of system (3), we express \( v \) as a function of \( u_2 \), say \( v = \tilde{v}(u_2) \). This equation may have more than one solution for \( v \) as this equation is not monotone in \( v \). We rewrite the first equation in the form

\[
\frac{r_0}{1 + k_1 v} - d - au_1 = \frac{\beta u_2}{1 + k_2 v},
\]

where both the left-hand side (LHS) and right-hand side (RHS) of this equation is a decreasing function of \( v \). We assume \( LHS(0) = r_0 - d - au_1 > 0 \), otherwise there does not exist any possible solution and \( RHS(0) = \beta u_2 \).
Case (i) LHS(0) > RHS(0) ⇒ there exists unique positive \( v \) that solves first equation of system (3).
Case (ii) LHS(0) < RHS(0) ⇒ there are no solution or there exists two solutions.
We neglect the second case and assume \( u_1, u_2 \) are such that

\[
r_0 - d - au_1 - \beta u_2 \geq 0
\]

which is a constraint equation for \( u_1, u_2 \). Let

\[
v = G_1(u_1, u_2) = G_1(f(u_2), u_2) = G(u_2).
\]

\( dG/du_2 \) is defined and positive for all \( u_2 \) such that

\[
r_0 - d - af(u_2) - \beta u_2 \geq 0,
\]

which implies \( u_2 \in (0, u^*_2) \) where \( r_0 - d - af(u^*_2) - \beta u^*_2 = 0 \). Hence, from the second equation of system (3), we get

\[
Q(u_2) = 0,
\]

where

\[
Q(u_2) = \frac{\beta f(u_2)}{1 + k_2 G(u_2)} - \mu - \frac{p_2 G(u_2)}{1 + q_1 f(u_2) + q_2 u_2}.
\]

Assume \( u_2 = 0 \), then

\[
Q(0) = \frac{\beta \bar{u}_1}{1 + k_2 G(0)} - \mu - \frac{p_2 G(0)}{1 + q_1 \bar{u}_1},
\]

where \( G(0) \) is the solution in \( v \) of the equation

\[
\frac{r_0}{1 + k_1 v} - d - a\bar{u}_1 - \frac{p_1 v}{1 + q_1 \bar{u}_1} = 0.
\]

The solution of the above equation is \( \bar{v} \). Hence, \( \bar{v} = G(0) \), we have

\[
Q(0) = \frac{\beta \bar{u}_1}{1 + k_2 \bar{v}} - \mu - \frac{p_2 \bar{v}}{1 + q_1 \bar{u}_1} > 0
\]
as we assume that \( R_0 > 1 \). Now let \( u_2 = u^*_2 \) and we get

\[
Q(u^*_2) = \frac{\beta f(u^*_2)}{1 + k_2 G(u^*_2)} - \mu - \frac{p_2 G(u^*_2)}{1 + q_1 f(u^*_2) + q_2 u^*_2}.
\]

Recall that \( v = G(u^*_2) \) is the solution of

\[
\frac{r_0}{1 + k_1 v} - d - af(u^*_2) - \frac{p_1 v}{1 + q_1 f(u^*_2) + q_2 u^*_2} - \frac{\beta u^*_2}{1 + k_2 v} = 0.
\]
Further, $v = 0$ is also a solution of this above equation, i.e.

$$G(u_2^*) = 0.$$ 

Therefore,

$$Q(u_2^*) = \beta f(u_2^*) - \mu.$$

We know that

$$r_0 - d - a\tilde{u}_1 - \beta\tilde{u}_2 = 0,$$

$$r_0 - d - af(u_2^*) - \beta u_2^* = 0.$$ 

i.e.

$$a(f(u_2^*) - \tilde{u}_1) = \beta(\tilde{u}_2 - u_2^*).$$ 

There are two possibilities occur, either

Case $- (A) f(u_2^*) < \tilde{u}_1, \tilde{u}_2 < u_2^*$ 

or

Case $- (B) f(u_2^*) > \tilde{u}_1, \tilde{u}_2 > u_2^*.$ 

Here, Case-(B) is impossible as from Equation (4) we have, $\tilde{u}_1 > f(\tilde{u}_2)$. Now using Case-(B), we have

$$f(u_2^*) > \tilde{u}_1 > f(\tilde{u}_2),$$

$$\Rightarrow f(u_2^*) > f(\tilde{u}_2),$$

$$\Rightarrow u_2^* > \tilde{u}_2$$

which is a contradiction. From Case-(A), we have

$$Q(u_2^*) = \beta f(u_2^*) - \mu < \beta \tilde{u}_1 - \mu < 0$$

i.e. there exists $\tilde{u}_2^* \in (0, u_2^*)$ such that $Q(\tilde{u}_2^*) = 0$. In this case, $\tilde{v}^* = G(\tilde{u}_2^*), \tilde{u}_1^* = f(\tilde{u}_2^*)$ which implies $E^* = (\tilde{u}_1^*, \tilde{u}_2^*, \tilde{v}^*) > 0$. This concludes the proof. 

4.2. Local stability analysis

To study the local stability properties of the equilibrium points, we calculate the Jacobian matrix around each equilibrium point of system (1). The Jacobian matrix of the model at
any arbitrary point \( E(u_1, u_2, v) \) is
\[
J(E) = \begin{pmatrix}
J_{11} & J_{12} & J_{13} \\
J_{21} & J_{22} & J_{23} \\
J_{31} & J_{32} & J_{33}
\end{pmatrix},
\]
where
\[
J_{11} = \frac{r_0}{1 + k_1 v} - d - 2au_1 - \frac{p_1 v(1 + q_2 u_2)}{(1 + q_1 u_1 + q_2 u_2)^2} - \frac{\beta u_2}{1 + k_2 v},
\]
\[
J_{12} = -\frac{p_1 q_2 u_1 v}{(1 + q_1 u_1 + q_2 u_2)^2} - \frac{\beta u_1}{1 + k_2 v},
\]
\[
J_{13} = -\frac{r_0 k_1 u_1}{(1 + k_1 v)^2} - \frac{p_1 u_1}{1 + q_1 u_1 + q_2 u_2} + \frac{\beta k_2 u_1 u_2}{(1 + k_2 v)^2},
\]
\[
J_{21} = \frac{\beta u_2}{1 + k_2 v} + \frac{p_2 q_1 u_2 v}{(1 + q_1 u_1 + q_2 u_2)^2},
\]
\[
J_{22} = \frac{\beta u_1}{1 + k_2 v} - \mu - \frac{p_2 v(1 + q_1 u_1)}{(1 + q_1 u_1 + q_2 u_2)^2},
\]
\[
J_{23} = -\frac{\beta k_2 u_1 u_2}{(1 + k_2 v)^2} - \frac{p_2 u_2}{1 + q_1 u_1 + q_2 u_2},
\]
\[
J_{31} = \frac{c_1 p_1 v + u_2 v(c_1 p_2 - c_2 p_1 q_1)}{(1 + q_1 u_1 + q_2 u_2)^2},
\]
\[
J_{32} = \frac{c_2 p_2 v + u_1 v(c_2 p_2 q_1 - c_1 p_1 q_2)}{(1 + q_1 u_1 + q_2 u_2)^2},
\]
\[
J_{33} = \frac{c_1 p_1 u_1 + c_2 p_2 u_2}{1 + q_1 u_1 + q_2 u_2} - m.
\]
The following lemmas show the local stability of equilibria.

**Lemma 4.1:** (i) The trivial equilibrium point \( E_0 \) is locally asymptotically stable if \( r_0 < d \), since the eigenvalues of the Jacobian evaluated at this equilibrium are \( r_0 - d, -\mu, -m \).

(ii) The disease-free and predator-free prey equilibrium \( E_1 \) is locally asymptotically stable if
\[
0 < r_0 - d < \min \left\{ \frac{a\mu}{\beta}, \frac{ma}{c_1 p_1 - m q_1} \right\}
\]
as the eigenvalues are \( -(r_0 - d), \beta(r_0 - d)/a - \mu, c_1 p_1(r_0 - d)/(a + q_1(r_0 - d)) - m \).

(iii) The predator-free equilibrium point \( E_2 \) is locally asymptotically stable if \( R_p < 1 \).

For the proof of this Lemma 4.1(iii) see Appendix 1.

**Lemma 4.2:** The disease-free equilibrium point \( E_3(u_1, 0, \bar{v}) \) be locally asymptotically stable if \( B_1 < 0 \), where \( B_1 \) is given in Appendix 2.

Proof of this lemma is given in Appendix 2.
Lemma 4.3: The coexistence equilibrium $E_*(u_1^*, u_2^*, v^*)$ is locally asymptotically stable if $\psi_1 > 0, \psi_3 > 0$ and $\psi_1 \psi_2 > \psi_3$.

Proof of this lemma and supportive calculations are given in Appendix 3.

4.3. Hopf bifurcation analysis

In this section, we have explored the possibility for occurrence of Hopf bifurcation of system (1). We observe that system (1) undergoes a Hopf bifurcation for gradual increase of the disease transmission rate. Hopf bifurcation around the interior equilibrium $E_*$ of system (1) with bifurcation parameter $\beta$ is given in the Theorem below.

Theorem 4.2: When the disease transmission rate ($\beta$) crosses a critical value, system (1) exhibits Hopf bifurcation around the positive coexistence equilibrium. The necessary and sufficient condition for Hopf bifurcation [22] to occur is that there exists $\beta = \beta_c$ such that,

(a) $F(\beta_c) \equiv \psi_1(\beta_c)\psi_2(\beta_c) - \psi_3(\beta_c) = 0$,
(b) $(d/d\beta)(\text{Re}(\lambda(\beta))) |_{\beta=\beta_c} \neq 0$,

where $\lambda$ is the root of the characteristic equation corresponding to the interior equilibrium point.

Proof: For $\beta = \beta_c$, we can write the characteristic equation $\lambda^3 + \psi_1\lambda^2 + \psi_2\lambda + \psi_3 = 0$ as $(\lambda^2 + \psi_2)(\lambda + \psi_1) = 0$. This equation has three roots $\lambda_1 = i\sqrt{\psi_2}$, $\lambda_2 = -i\sqrt{\psi_2}$ and $\lambda_3 = -\psi_1$.

For all $\beta$, the roots are in general of the form

$\lambda_1(\beta) = \eta_1(\beta) + i\eta_2(\beta),
\lambda_2(\beta) = \eta_1(\beta) - i\eta_2(\beta),
\lambda_3(\beta) = -\psi_1$.

Now, we shall verify the transversality condition

$\frac{d}{d\beta}(\text{Re}(\lambda(\beta))) |_{\beta=\beta_c} \neq 0, \ j = 1, 2.$

Substituting $\lambda_j(\beta) = \eta_1(\beta) + i\eta_2(\beta)$ into the characteristic equation and calculating the derivative, we have

$P(\beta)\eta_1'(\beta) - Q(\beta)\eta_2'(\beta) + U(\beta) = 0,$
$Q(\beta)\eta_1'(\beta) + P(\beta)\eta_2'(\beta) + V(\beta) = 0,$

where,

$P(\beta) = 3\eta_1^2(\beta) + 2\psi_1(\beta)\eta_1(\beta) + \psi_2(\beta) - 3\eta_2^2(\beta),$
$Q(\beta) = 6\eta_1(\beta)\eta_2(\beta) + 2\psi_1(\beta)\eta_2(\beta),$
$U(\beta) = \eta_1^2(\beta)\psi_1'(\beta) + \psi_2'(\beta)\eta_1(\beta) + \psi_3'(\beta) - \psi_1'(\beta)\eta_2^2(\beta),$
$V(\beta) = 2\eta_1(\beta)\eta_2(\beta)\psi_1'(\beta) + \psi_2'(\beta)\eta_2(\beta).$
Noticing that \( \eta_1(\beta_c) = 0, \eta_2(\beta_c) = \sqrt{\psi_2(\beta_c)} \), we have \( P(\beta_c) = -2\psi_2(\beta_c), \quad Q(\beta_c) = 2\psi_1(\beta_c)\sqrt{\psi_2(\beta_c)}, \quad U(\beta_c) = \psi_3'(\beta_c) - \psi_1'(\beta_c)\psi_2(\beta_c) \) and \( V(\beta_c) = \psi_2'(\beta_c)\sqrt{\psi_2(\beta_c)} \). Now,

\[
\frac{d}{d\beta} (\text{Re}(\lambda(\beta))) \mid_{\beta = \beta_c} = \frac{Q(\beta_c)V(\beta_c) + P(\beta_c)U(\beta_c)}{P(\beta_c)^2 + Q(\beta_c)^2} = \frac{2\psi_1(\beta_c)\sqrt{\psi_2(\beta_c)} \times \psi_3'(\beta_c)\sqrt{\psi_2(\beta_c)} + (-2\psi_2(\beta_c))(\psi_3'(\beta_c) - \psi_1'(\beta_c)\psi_2(\beta_c))}{(-2\psi_2(\beta_c))^2 + (2\psi_1(\beta_c)\sqrt{\psi_2(\beta_c)})^2} = \frac{\psi_1(\beta_c)\psi_3'(\beta_c) - \psi_3'(\beta_c) + \psi_1'(\beta_c)\psi_2(\beta_c)}{2(\psi_2(\beta_c) + (\psi_1(\beta_c))^2) \neq 0, \quad \text{if} \quad \psi_1(\beta_c)\psi_3'(\beta_c) - \psi_3'(\beta_c) + \psi_1'(\beta_c)\psi_2(\beta_c) \neq 0,}
\]

and \( \lambda_3(\beta_c) = -\psi_1(\beta_c) \neq 0 \).

Therefore, the transversality condition holds. This implies that a Hopf bifurcation occurs at \( \beta = \beta_c \). This concludes the proof of the Theorem. \( \square \)

5. Backward bifurcation analysis

Recently, Boldin [5] investigated the possible effects of an invasion when the parameters of a model are varied so that \( R_0 \) of the invading population passes the value 1. They performed uniform study of ecological, adaptive dynamics and disease transmission models and derived a simple formula for the direction of bifurcation from a steady state in which only the resident populations are present. In the present investigation, we also investigate the backward bifurcation and the direction of backward bifurcation for our model.

Theorem 4.1 shows that a coexistence equilibrium exists. Here we show that the coexistence equilibrium may not be unique. Co-existence equilibria in this system can occur in one of two ways: (1) if predator invades prey-disease equilibrium; (2) if disease invades predator–prey equilibrium.

Here we show that fear in the prey generates backward bifurcation and allows the predator to persist for \( R_p < 1 \). To eliminate the effect of the Holling II functional response terms, we take \( q_1, q_2 = 0 \). Thus system (1) for \( E_n \) becomes

\[
\frac{r_0}{1 + k_1v} - d - au_1 - p_1v - \frac{\beta u_2}{1 + k_2v} = 0,
\]

\[
\frac{\beta u_1}{1 + k_2v} = \mu + p_2v,
\]

\[
c_1p_1u_1 + c_2p_2u_2 = m.
\]

From the above system of equations we get,

\[
u_1 = \frac{(\mu + p_2v)(1 + k_2v)}{\beta} \quad \text{that is,} \quad u_1 = f_1(v),
\]

\[
u_2 = \frac{1 + k_2v}{\beta} \left[ \frac{r_0}{1 + k_1v} - d - af_1(v) - p_1v \right], \quad \text{that is,} \quad u_2 = f_2(v).
\]
Therefore, \( c_1 p_1 f_1(v) + c_2 p_2 f_2(v) = m \), that is, \( v \) is a solution of the following equation:

\[
c_1 p_1 (\mu + p_2 v) + c_2 p_2 \left[ \frac{r_0}{1 + k_1 v} - d - a \frac{(\mu + p_2 v)(1 + k_2 v)}{\beta} - p_1 v \right] = \frac{m\beta}{1 + k_2 v}.
\]

Plotting the solution of the above equation in the \((R_p, v)\) plane for gradual increase of \(c_1\). We see that the curve bifurcates backward from the critical value \(c_1^*\) such that \(R_p(c_1^*) = 1\) (see Figure 11). Plotting with respect to \(R_p\) (rather than \(c_1\)) in Figure 11, we see that even if \(R_p < 1\) the predator may still persist. Therefore, the invader (predator population densities) can meet with success even if \(R_p < 1\) [5]. Thus fear in prey allows the predator to persist for values of its invasion number below one.

### 5.1. Direction of backward bifurcation analysis

When \(R_p\) is less than unity, system (1) shows backward bifurcation i.e. a small positive unstable coexistence equilibrium appears while the predator-free equilibrium and a larger positive coexistence equilibrium are locally asymptotically stable. For the clarification, we outline a theory which determines the backward bifurcation of the predator-free equilibrium. This theory is based on the general centre manifold theory [8,12,18].

Here we consider a system of autonomous differential equations

\[
\frac{du}{dt} = f(u, \phi), \quad f : \mathbb{R}^n \times \mathbb{R} \to \mathbb{R}^n \quad \text{and} \quad f \in C^2(\mathbb{R}^n \times \mathbb{R}), \quad (5)
\]

where \(\phi\) is a bifurcation parameter and \(u = (u_1, u_2, u_3, \ldots, u_n) \in \mathbb{R}^n\). Without loss of generality, it is assumed that \(\bar{0} = (0,0,0,\ldots,0)\) n-times is an equilibrium for system (5) for all values of the parameter \(\phi\), that is

\[
f(\bar{0}, \phi) \equiv \bar{0} \quad \text{for all } \phi.
\]

Now we state a particular part of Theorem (4.1) of [8] and give a rigorous proof for the backward bifurcation of our system (1).

**Theorem 5.1:** Assume \(\hat{A} = D_u f(\bar{0}, 0)\) is the linearization matrix of system (5) around the equilibrium \(\bar{0}\) with \(\phi\) evaluated at 0. Zero is a simple eigenvalue of \(\hat{A}\) and all other eigenvalues of \(\hat{A}\) have negative real parts. Further, assume that matrix \(\hat{A}\) has a nonnegative right eigenvector \(\hat{w} = (w_1, w_2, w_3, \ldots, w_n)\) and a left eigenvector \(\hat{h} = (h_1, h_2, h_3, \ldots, h_n)\) corresponding to the zero eigenvalue.

Let \(f_k\) be the kth component of \(f = (f_1, f_2, f_3, \ldots, f_n)\) and

\[
\hat{a} = \sum_{k,i,j=1}^{n} h_k w_i w_j \frac{\partial^2 f_k}{\partial u_i \partial u_j}(\bar{0}, 0), \quad \hat{b} = \sum_{k,i=1}^{n} h_k w_i \frac{\partial^2 f_k}{\partial u_i \partial \phi}(\bar{0}, 0).
\]

Also let, \(\hat{a} > 0, \hat{b} > 0\). When \(\phi < 0\) with \(|\phi| \ll 1\), \(\bar{0}\) is locally asymptotically stable, and there exists a positive unstable equilibrium; when \(0 < \phi \ll 1\), \(\bar{0}\) is unstable and there exists a negative and locally asymptotically stable equilibrium and at \(\phi = 0\) a backward bifurcation takes place.
For system (1), let $\phi = c_1$ be the bifurcation parameter. When $R_p = 1$, we considered the bifurcation point as

$$
\phi = \phi^* = \frac{\beta + q_1 \mu + q_2 \left( r_0 - d - \frac{a \mu}{\beta} \right)}{p_1 \mu} \left( m - \frac{c_2 p_2 \left( r_0 - d - \frac{a \mu}{\beta} \right)}{\beta + q_1 \mu + q_2 \left( r_0 - d - \frac{a \mu}{\beta} \right)} \right),
$$

where we have used the predator-free equilibrium $[\tilde{u}_1 = \mu / \beta, \tilde{u}_2 = (r_0 - d - a \mu / \beta) / \beta, \tilde{v} = 0]$. The linearization matrix of system (1) around the predator-free equilibrium point when $\phi = \phi^*$ is

$$
D_u(f) = \begin{pmatrix}
-\frac{a \mu}{\beta} & -\frac{r_0 k_1 \mu}{\beta} & \frac{p_1 \mu}{\beta} + \frac{k_2 \mu \left( r_0 - d - \frac{a \mu}{\beta} \right)}{\beta + q_1 \mu + q_2 \left( r_0 - d - \frac{a \mu}{\beta} \right)} \\
\beta + q_1 \mu + q_2 \left( r_0 - d - \frac{a \mu}{\beta} \right) & -\frac{k_2 \mu}{\beta} \left( r_0 - d - \frac{a \mu}{\beta} \right) - \frac{p_2 \left( r_0 - d - \frac{a \mu}{\beta} \right)}{\beta + q_1 \mu + q_2 \left( r_0 - d - \frac{a \mu}{\beta} \right)} \\
0 & 0 & 0
\end{pmatrix}.
$$

It is easy to see that 0 is an eigenvalue of $D_u(f)$. A right eigenvector associated with the 0 eigenvalue is $\bar{w} = (w_1, w_2, w_3)^t$, where

$$
w_1 = \frac{k_2 \mu}{\beta} + \frac{p_2}{\beta + q_1 \mu + q_2 \left( r_0 - d - \frac{a \mu}{\beta} \right)},
$$

$$
w_2 = -\frac{a}{\beta} \left( \frac{k_2 \mu}{\beta} + \frac{p_2}{\beta + q_1 \mu + q_2 \left( r_0 - d - \frac{a \mu}{\beta} \right)} \right) - \frac{r_0 k_1 \mu}{\beta + q_1 \mu + q_2 \left( r_0 - d - \frac{a \mu}{\beta} \right)} + \frac{k_2 \mu \left( r_0 - d - \frac{a \mu}{\beta} \right)}{\beta + q_1 \mu + q_2 \left( r_0 - d - \frac{a \mu}{\beta} \right)},
$$

$$
w_3 = 1.
$$

A left eigenvector $\bar{h}$ associated with the 0 eigenvalue, satisfying $\bar{h} \cdot \bar{w} = 1$ is $\bar{h} = [0,0,1]$. Furthermore, at the predator-free equilibrium point we get,

$$
\frac{\partial^2 f_3}{\partial v \partial u_1} = \frac{c_1 p_1 + \tilde{u}_2 (c_1 p_1 q_2 - c_2 p_2 q_1)}{(1 + q_1 \tilde{u}_1 + q_2 \tilde{u}_2)^2},
$$

$$
\frac{\partial^2 f_3}{\partial v \partial u_2} = \frac{c_2 p_2 + \tilde{u}_1 (c_2 p_2 q_1 - c_1 p_1 q_2)}{(1 + q_1 \tilde{u}_1 + q_2 \tilde{u}_2)^2},
$$

$$
\frac{\partial^2 f_3}{\partial v \partial \phi} = \frac{p_1 \mu}{\beta + q_1 \mu + q_2 \left( r_0 - d - \frac{a \mu}{\beta} \right)}.
$$
Figure 1. Figure shows that system (1) is stable around the endemic coexistence equilibrium point for parameter values: $\beta = 0.381, r_0 = 0.03, k = 0.07, d = 0.01, p_1 = 0.5, q_1 = 0.1, m = 0.05, c_1 = 0.4, p_2 = 0.5, q_2 = 0.1, c_2 = 0.4, \mu = 0.08$, with the initial condition $[u_1(0), u_2(0), v(0)] = [0.25, 0.02, 0.035]$. The rest of the second derivatives appearing in the formula for $\hat{a}$ and $\hat{b}$ are all zero. Hence,

$$\hat{a} = \left( \frac{k_2 \mu}{\beta} + \frac{p_2}{\beta (1 + q_1 \tilde{u}_1 + q_2 \tilde{u}_2)} \right) \frac{c_1 p_1 + \tilde{u}_2 (c_1 p_1 q_2 - c_2 p_2 q_1)}{(1 + q_1 \tilde{u}_1 + q_2 \tilde{u}_2)^2}$$

$$+ \left( -\frac{a}{\beta} \left( \frac{k_2 \mu}{\beta} + \frac{p_2}{\beta (1 + q_1 \tilde{u}_1 + q_2 \tilde{u}_2)} \right) \frac{r_0 k_1}{\beta} - \frac{p_1}{\beta (1 + q_1 \tilde{u}_1 + q_2 \tilde{u}_2)} + k_2 \tilde{u}_2 \right)$$

$$\times \frac{c_2 p_2 + \tilde{u}_1 (c_2 p_2 q_1 - c_1 p_1 q_2)}{(1 + q_1 \tilde{u}_1 + q_2 \tilde{u}_2)^2}$$,

$$\hat{b} = \frac{p_1 \mu}{\beta + q_1 \mu + q_2 (r_0 - d - \frac{a \mu}{\beta})} > 0.$$

Therefore, if

$$k_2 > k_2' = \left( \frac{p_1 \beta + p_2 a}{\beta^2 (1 + q_1 \tilde{u}_1 + q_2 \tilde{u}_2)} + \frac{r_0 k_1}{\beta} \right) \frac{1}{\tilde{u}_2 - \frac{a \mu}{\beta}}$$

provided $\tilde{u}_2 \neq a \mu / \beta^2$ then $\hat{a} > 0$. So, the direction of the bifurcation of system (1) is backward for $k_2 > k_2'$. Figures 11(a,b) show that there is a parameter set for which these conditions may occur.
Figure 2. For $k = 0.5$, the infectious prey population goes to extinction while susceptible prey and predator populations coexist in a stable manner. The other parameter values are the same as in Figure 1.

6. Numerical results

In this section, we have performed some numerical simulations on system (1) to illustrate the analytical results observed in the previous sections. All the simulations are carried out in MATLAB. This study demonstrates stability, the presence of limit cycle, Hopf bifurcation, bistability, higher order periodic oscillations and chaos. Here disease transmission rate $\beta$ and cost of fear $k_1, k_2$ are important parameters under investigation. For simplicity, we consider $k_1 = k_2 = k$ throughout the simulation.

For illustration purposes, we choose the parameter values as: $r_0 = 0.03, k = 0.07, d = 0.01, a = 0.01, p_1 = 0.5, q_1 = 0.1, m = 0.05, c_1 = 0.4, p_2 = 0.5, q_2 = 0.1, c_2 = 0.4, \mu = 0.08, \beta = 0.381$, where most of the parameters are taken from [36]. For numerical simulation, we choose the initial values of population densities as $[u_1(0), u_2(0), v(0)] = [0.25, 0.02, 0.035]$.

For the above set of parameter values, the system shows that the endemic coexistence steady state is stable (see Figure 1). In Figure 1, we see that the trajectories starting inside the region of attraction approach the endemic coexistence equilibrium point ($E^*_e$).

To investigate the impact of fear, we fix $\beta = 0.381$ and gradually increase the strength of fear ($k$). For $k = 0.5$, we observe that the disease becomes extinct from the system and susceptible prey and predator populations coexist in a stable manner (Figure 2).

We choose another parameter set $r_0 = 0.05, k = 0.01, d = 0.01, a = 0.01, p_1 = 0.5, q_1 = 0.6, m = 0.05, c_1 = 0.4, p_2 = 0.5, q_2 = 0.6, c_2 = 0.4, \mu = 0.08, \beta = 0.399$ such that
Figure 3. Figure shows a periodic solution around the coexistence equilibrium point $E_*$ of the system (1) for $\beta = 0.399$. Other parameter values are $r_0 = 0.05, k = 0.01, d = 0.01, a = 0.01, p_1 = 0.5, q_1 = 0.6, m = 0.05, c_1 = 0.4, p_2 = 0.5, q_2 = 0.6, c_2 = 0.4, \mu = 0.08$.

system (1) shows a limit cycle around the interior equilibrium point $E_*$ (see Figure 3). Further increase of $\beta$, stabilizes the system around the endemic equilibrium point $E_*$.

To observe the long-term behaviour of system (1) for a range of values of $\beta$, we draw the bifurcation diagram considering $\beta$ as a bifurcation parameter. In Figure 4, we observe that the system shows limit cycle oscillations for $0.3987 < \beta \leq 0.3999$; Hopf bifurcation (HB) occurs at $\beta = 0.39999$ and system become stable focus for $0.3999 < \beta \leq 0.4012$, and for $\beta > 0.4012$, the predator population becomes extinct through transcritical bifurcation (TB) from system (1) while susceptible and infected prey populations co-exist in a stable mode.

To investigate the impact of fear when the system is unstable around the interior equilibrium, we fix the value of $\beta$ at 0.399 such that susceptible prey, infected prey and predator populations show periodic oscillation. We draw the bifurcation diagram with respect to $k$ (see Figure 5). In Figure 5, we observe that if we gradually increase $k$ then above a threshold value ($k = 0.5$) the system enters into chaotic regime from limit cycle oscillations. If we further increase $k$, then above a threshold value ($k = 6$) the infected population goes to extinction while the susceptible prey and predator populations coexist in an oscillatory manner. Then above a critical value $k = 12.75$, the system becomes stable around the disease-free predator–prey equilibrium $E_3$.

For $k = 4$, system (1) shows chaotic behaviour (see Figure 6), where other parameter values are as same as in Figure 3. In Figure 7, we draw the Poincare map of system (1) for $\beta = 0.399, k = 4$ and rest of the parameter values are as same as in Figure 3. Here we fix
the susceptible population density $u_1$ at 0.45. In this figure, the scattered distribution of the sampling points implies the chaotic behaviour of the system (1). In Figure 8, we also plot the Lyapunov exponents with time, where for $\beta = 0.399, k = 4$ and other parameter values are fixed as in Figure 3. We use Wolf algorithm [38] to compute the Lyapunov exponents. The initial values are fixed at $[u_1(0), u_2(0), v(0)] = [0.25, 0.2, 0.035]$. Here the maximum Lyapunov exponent is positive. Non-negativity of the maximum Lyapunov exponent indicates that system (1) is chaotic.

Now we explore the possibility of bistability in our system. For $\beta = 0.4$ and the others parameter values are as same as in Figure 3, by taking different initial values we observe that the solution trajectories of system (1) go to the different attractors. In Figure 9, we take the two different initial values as $[0.8, 0.12, 0.001]$ and $[0.25, 0.2, 0.035]$ for which the system converges to the different equilibrium points $E_2 = [0.2014, 0.0973, 0]$ and $E_* = [0.2142, 0.0859, 0.0124]$, which are shown in the figure by red and blue colours, respectively. So here we get a bistability. Bistability is a phenomenon where the system converges to two different equilibria for the same parameter values based on the variation of initial conditions. Here any trajectory starting from the interior $R_3^+$ either converges to $E_*$ or $E_2$. To find the basin of attraction for the bistability, we plot Figure 10, where the blue dotted region is the basin of attraction for the equilibrium point $E_*$ and the red dotted region is the basin of attraction for the equilibrium point $E_2$. In Figure 10(a), keeping the initial density of $v$ fixed at 0.0075 we get the basin of attraction of system (1) on the $u_1 - u_2$
Figure 5. Bifurcation diagram of system (1) with respect to the bifurcating parameter $k$. The other parameter values are same as in Figure 3. Here the maximum and minimum values of the oscillations are plotted in blue and red colours, respectively.

Figure 6. Figure shows chaotic behaviours of system (1) for $\beta = 0.399$ and $k = 4$ and the other parameter values are as fixed as in Figure 3.
Figure 7. Figure shows Poincare section in the \( u_2 - v \) plane for system (1) when \( u_1 \) is fixed at 0.45.

plane; in Figure 10(b), for initial density of \( u_2 \) at 0.02095 we get the basin of attraction of system (1) on the \( u_1 - v \) plane.

Further, to observe the backward bifurcation of system (1) with respect to the predator invasion number \( R_p \) between the predator-free equilibrium and the coexistence equilibrium, we plot the backward bifurcation diagram (see Figure 11) for the corresponding set of parameter values: \( \beta = 0.4, r_0 = 0.5, k_1 = 25, d = 0.01, a = 0.01, p_1 = 0.5, q_1 = 0, m = 0.35, p_2 = 0.5, q_2 = 0, k_2 = 250, c_2 = 0.501, \mu = 0.08 \). In Figure 11, we take \( R_p \) as a bifurcation parameter. We vary the parameter \( c_1 \) in the region \([0.33, 0.35]\) and plot \( R_p \) along x-axis and plot the equilibrium predator density along y-axis. Figure clearly shows that fear permits the predator to exist for values of its invasion number below one.

In Figure 11, the invasion curve marked by red and blue colour is a curve of backward transcritical bifurcation of equilibrium densities [19]. Here we draw blue curve and black line for stable branches of the endemic equilibrium and predator-free equilibrium, respectively. The red curve and green line indicate the unstable branches of the endemic equilibrium and the predator-free equilibrium, respectively. Here for predator invasion number \( R_p < 1 \), if we take any point from the area between the red curve and black line, then an unstable interior equilibrium appears and the predator-free equilibrium becomes stable. Again, for \( R_p < 1 \), if we take any point from the area between the blue and red curve, then a stable interior equilibrium also appears. Therefore, backward bifurcation creates a bistability between endemic equilibrium \( (E_\ast) \) and predator-free equilibrium \( (E_2) \). Further, we choose \( c_1 = 0.339 \) when other parameter values are fixed as in
Figure 8. Figure shows the Lyapunov exponents of system (1). Non-negativity of the maximum Lyapunov exponent indicates the chaotic behaviour of the system.

Figure 9. The figure shows bistability between predator-free equilibrium point ($E_2$) and endemic equilibrium ($E_*$) for $\beta = 0.4$ and the other parameter values same as in Figure 3. Here two solution trajectories (red and blue lines) initiating from (0.8, 0.12, 0.001) and (0.25, 0.2, 0.035) converge to $E_2$ and $E_*$, respectively.
Figure 10. Figure shows the basin of attraction of system (1) for \( E_2 \) and \( E_\ast \) (red and blue regions, respectively). Here Figure 7(a) shows basin of attraction in the \( u_1 - u_2 \) plane when \( v \) is fixed at 0.0075 and Figure 7(b) shows the basin of attraction in the \( u_1 - v \) plane when \( u_2 \) is fixed at 0.02095.

Figure 11. Figure shows backward bifurcation of system (1) when \( R_p < 1 \) for the parameter values \( \beta = 0.4, r_0 = 0.5, k_1 = 25, d = 0.01, a = 0.01, p_1 = 0.5, q_1 = 0, m = 0.35, p_2 = 0.5, q_2 = 0, k_2 = 250, c_2 = 0.501, \mu = 0.08. \)
Figure 11 such that the predator invasion number is less that unity ($R_p = 0.97$). For this set of parameter values, we compute the interior equilibrium points. For above parameter values, system (1) has two endemic equilibrium points $E_\ast(8.2889, 0.1376, 0.0986)$ and $E_\ast(1.6564, 4.4866, 0.0247)$, where first one is locally stable and the later is unstable.

7. Conclusion

In this paper, we have considered the scenario that fear of predator reduces the reproduction rate of prey population and it also suppresses the disease transmission among the prey population. Several studies find reduced reproduction in species due to fear of predator [3,27,31]. Fear effects on any population can measure the influence of long-term population dynamics and ecosystem function [13]. Not only fear changes the ecosystem function but also influences epidemic functions. So to make the model more biologically relevant to the population dynamics, incorporation of fear effect in the model is an important factor.

We have explored a large variety of complex dynamics, which is much broader than the previous studies in ecology and eco-epidemiology. In the present study, we explore the rich dynamics of the eco-epidemiological system for Holling type II functional response, when both susceptible and infected prey contribute for saturation.

To study the impact of fear in the eco-epidemic system, we consider two situations where the eco-epidemiological system shows stable dynamics and unstable (limit cycle oscillations) dynamics. Then we gradually increase the strength of the fear and explore the impact of fear in an eco-epidemic system. We observe that when the system is stable around the endemic equilibrium, if we increase the strength of fear, then the disease will be wiped out from the system. We also observe that when the system shows limit cycle oscillations around the endemic equilibrium, if we increase the strength of the fear, then above a critical value of fear, the disease will become extinct from the system and the system becomes purely ecological system. However, for intermediate value of fear, the system shows chaotic oscillation. Therefore, fear in the eco-epidemiological systems may produce chaos. Chaotic oscillations may potentially be explained by the incommensurate frequencies of population cycles. If the frequency of the oscillation of the susceptible-infected prey subsystem is incommensurate with the frequency of the oscillation in either susceptible prey-predator subsystem or infected prey-predator subsystem, then the system may produce chaos [17]. If we further increase the strength of the fear, then the system becomes stable, replacing the population oscillations by a stable disease-free predator–prey equilibrium. Therefore, if the level of fear increases, then the infected prey population goes to extinction from the endemic steady state or oscillations of coexistence. Fear in prey population lowers the growth rate and the foraging activity. Lower foraging activity reduces the chance of being infected. Therefore, in the presence of fear, the effectual rate of disease transmission reduces significantly, which may lead to eradication of disease from the system. We observe that fear can suppress infection in both situations, stable endemic state and coexisting oscillations.

We further explore some rich dynamics such as Hopf bifurcation and bistability in our model. We observe that if the disease transmission rate is increased gradually, then the predator–prey oscillations become stable via Hopf bifurcation. We observe that disease can stabilize the population oscillation by replacing limit cycle oscillation by stable coexistence equilibrium. As the disease transmission rate increases more prey individuals become
infected which also contribute for the saturation on predator consumption on susceptible prey. Such indirect saturation effect may lower the consumption of susceptible prey and enhance the stability of the eco-epidemic system. For a different set of parameter values, we observe a bistability between predator-free equilibrium \((E_2)\) and endemic equilibrium \((E_\ast)\). Depending on the initial condition the system converges to either predator-free or endemic steady state. In contrast, Siekmann et al. [29] found a bistability between disease-free and predator-free equilibrium in a predator–prey system with disease in prey. The bistability in our system is likely produced by a backward bifurcation of the coexistence equilibrium with respect to the predator invasion number. In bistability situation, predator population may become extinct from the system, where initial population density plays a crucial role in the persistence of the predator population. Fear is a necessary factor for the backward bifurcation and allows for the predator to persist even if its invasion number is smaller than one. Therefore, bistability is an important issue as it relates to the predator persistence and extinction.

Several studies in eco-epidemiology describe complex dynamics. To the best of our knowledge, the first eco-epidemiological paper to show chaos is Upadhyay et al. [34] who used an existing model of Chattopadhyay and Bairagi [10]. They showed the chaos via a cascade of period-doubling bifurcations. Stiefs et al. [30] described quasi-periodicity and chaos through a generalized predator–prey model with disease in the predator population. Siekman et al. [29] found bistability in a predator–prey system by incorporating a free-living virus stage in the model and a disease in the prey population. Kooi et al. [20] found more complex dynamics, including period-doubling cascade into chaos, bistability and transcritical bifurcations of limit cycles in an eco-epidemiological system. Recently, Saifuddin et al. [25] also demonstrated that disease can produce chaos in a predator–prey model. However, the present investigation first time reports that fear factor has the potential to produce backward bifurcation and chaos by suppressing prey growth and disease transmission.

**Disclosure statement**

No potential conflict of interest was reported by the authors.

**Funding**

M. Martcheva acknowledges partial support from Division of Mathematical Sciences (NSF) grant DMS-1515661. Amar Sha thankfully acknowledges the Council of Scientific and Industrial Research, New Delhi, India for the financial assistance in the form of Junior Research Fellowship.

**References**

[1] R.M. Anderson, R.M. May, *Infectious diseases and population cycles of forest insects*. Science 210(4470) (1980), pp. 658–661.
[2] N. Bairagi, P.K. Roy, and J. Chattopadhyay, *Role of infection on the stability of a predator–prey system with several response functions – a comparative study*, J. Theor. Biol. 248(1) (2007), pp. 10–25.
[3] M.F. Benard, *Predator-induced phenotypic plasticity in organisms with complex life histories*, Annu. Rev. Ecol. Evol. Syst. 35 (2004), pp. 651–673.
[4] E. Beretta and Y. Kuang, *Modeling and analysis of a marine bacteriophage infection*, Math. Biosci. 149(1) (1998), pp. 57–76.
[5] B. Boldin, Introducing a population into a steady community: The critical case, the center manifold, and the direction of bifurcation, SIAM J. Appl. Math. 66(4) (2006), pp. 1424–1453.
[6] J.S. Brown, J.W. Laundré, and M. Gurung, The ecology of fear: Optimal foraging, game theory, and trophic interactions, J. Mammal. 80(2) (1999), pp. 385–399.
[7] V. Capasso and G. Serio, A generalization of the Kermack–McKendrick deterministic epidemic model, Math. Biosci. 42(1–2) (1978), pp. 43–61.
[8] C. Castillo-Chavez and B. Song, Dynamical models of tuberculosis and their applications, Math. Biosci. Eng. 1(2) (2004), pp. 361–404.
[9] J. Chattopadhyay and O. Arino, A predator–prey model with disease in the prey, Nonlinear Anal. Ser. A Theory Methods Appl. 36(6) (1999), pp. 747–766.
[10] J. Chattopadhyay and N. Bairagi, Pelicans at risk in Salton sea-an eco-epidemiological model, Ecol. Model. 136(2) (2001), pp. 103–112.
[11] W. Cresswell, Predation in bird populations, J. Ornithol. 152(1) (2011), pp. 251–263.
[12] J. Dushoff, W. Huang, and C. Castillo-Chavez, Backwards bifurcations and catastrophe in simple models of fatal diseases, J. Math. Biol. 36(3) (1998), pp. 227–248.
[13] K.H. Elliott, D.R. Norris, G.S. Betini, and I. Dworkin, Scared fitless: Context-dependent response of fear to loss of predators over evolutionary time in Drosophila melanogaster, FACETS 2(1) (2017), pp. 342–354.
[14] J.M. Epstein, J. Parker, D. Cummings, and R.A. Hammond, Coupled contagion dynamics of fear and disease: Mathematical and computational explorations, PLoS One 3(12) (2008), pp. e3955.
[15] H.I Freedman, A model of predator–prey dynamics as modified by the action of a parasite, Math. Biosci. 99(2) (1990), pp. 143–155.
[16] K.P. Hadeler and H.I. Freedman, Predator–prey populations with parasitic infection, J. Math. Biol. 27(6) (1989), pp. 609–631.
[17] A. Hastings and T. Powell, Chaos in a three-species food chain, Ecology 72(3) (1991), pp. 896–903.
[18] W. Huang, K.L. Cooke, and C. Castillo-Chavez, Stability and bifurcation for a multiple-group model for the dynamics of HIV/AIDS transmission, SIAM J. Appl. Math. 52(3) (1992), pp. 835–854.
[19] E. Kisdi, Dispersal polymorphism in stable habitats, J. Theor. Biol. 392 (2016), pp. 69–82.
[20] B.W. Kooi, G.A.K. van Voorn, and K.P. Das, Stabilization and complex dynamics in a predator–prey model with predator suffering from an infectious disease, Ecol. Complex. 8(1) (2011), pp. 113–122.
[21] N.D. Lewis, M.N. Breckels, M. Steinke, and E.A. Codling, Role of infochemical mediated zooplankton grazing in a phytoplankton competition model, Ecol. Complex. 16 (2013), pp. 41–50.
[22] W.M Liu, Criterion of Hopf bifurcations without using eigenvalues, J. Math. Anal. Appl. 182(1) (1994), pp. 250–256.
[23] M.L. Rosenzweig and R.H. MacArthur, Graphical representation and stability conditions of predator–prey interactions, Am Nat 97 (1963), pp. 209–223.
[24] B. Sahoo and S. Poria, Diseased prey predator model with general Holling type interactions, Appl. Math. Comput. 226 (2014), pp. 83–100.
[25] M. Saifuddin, S. Biswas, S. Samanta, S. Sarkar, and J. Chattopadhyay, Complex dynamics of an eco-epidemiological model with different competition coefficients and weak Allee in the predator, Chaos Solitons Fractals 91 (2016), pp. 270–285.
[26] S. Sarwardi, M. Haque, and E. Venturino, A Leslie-Gower Holling-type II ecoepidemiological model, J. Appl. Math. Comput. 35(1–2) (2011), pp. 263–280.
[27] G.J. Scrimgeour and J.M. Culp, Foraging and evading predators: The effect of predator species on a behaviour trade-off by a lotic mayfly, Oikos 69(1) (1994), pp. 71–79.
[28] M.J. Sheriff, C.J. Krebs, and R. Boonstra, The sensitive hare: sublethal effects of predator stress on reproduction in snowshoe hares, J. Anim. Ecol. 78(6) (2009), pp. 1249–1258.
[29] I. Siekmann, H. Malchow, and E. Venturino, On competition of predators and prey infection, Ecol. Complex. 7(4) (2010), pp. 446–457.
Appendices

Appendix 1

Hence $E_2$ (predator-free equilibrium point) is $(\tilde{u}_1, \tilde{u}_2, 0)$, where
\[ \tilde{u}_1 = \frac{\mu}{\beta}, \quad \tilde{u}_2 = \frac{r_0 - d - \frac{a\mu}{\beta}}{\beta}. \]

The Jacobian matrix for the equilibrium point $E_2$ is given by
\[
J(E_2) = \begin{pmatrix}
A_1 & A_2 & A_3 \\
A_4 & 0 & A_5 \\
0 & 0 & A_6
\end{pmatrix},
\]
where,
\[
A_1 = -\frac{a\mu}{\beta},
A_2 = -\mu,
A_3 = \frac{r_0 k_1 \mu}{\beta} - \frac{p_1 \mu}{\beta + q_1 \mu + q_2 \left( r_0 - d - \frac{a\mu}{\beta} \right)} + \frac{k_2 \mu \left( r_0 - d - \frac{a\mu}{\beta} \right)}{\beta},
A_4 = r_0 - d - \frac{a\mu}{\beta},
A_5 = -\frac{k_2 \mu \left( r_0 - d - \frac{a\mu}{\beta} \right)}{\beta} - \frac{p_2 \left( r_0 - d - \frac{a\mu}{\beta} \right)}{\beta + q_1 \mu + q_2 \left( r_0 - d - \frac{a\mu}{\beta} \right)},
A_6 = \frac{c_1 p_1 \mu + c_2 p_2 \left( r_0 - d - \frac{a\mu}{\beta} \right)}{\beta + q_1 \mu + q_2 \left( r_0 - d - \frac{a\mu}{\beta} \right)} - m.
\]
So the eigenvalues of \( J(E_2) \) are \( A_6 \) and the roots of the characteristic polynomial
\[
\lambda^2 + \frac{\lambda a \mu}{\beta} + \mu \left( r_0 - d - \frac{a \mu}{\beta} \right) = 0.
\]
Here the roots are \( \lambda_1 + \lambda_2 = -(a \mu/\beta) < 0 \) and \( \lambda_1 \lambda_2 = \mu(r_0 - d - a \mu/\beta) \). Therefore, all the eigenvalues of the Jacobian matrix \( J(E_2) \) are negative or having negative real part if \( A_6 < 0 \) and \( r_0 - d - a \mu/\beta > 0 \).

**Appendix 2**

Here, the Jacobian at \( E_3(\bar{u}_1, 0, \bar{v}) \) is given by
\[
J(E_3) = \begin{pmatrix}
B_1 & B_2 & B_3 \\
0 & B_4 & 0 \\
B_5 & B_6 & 0
\end{pmatrix},
\]
where
\[
B_1 = -a \bar{u}_1 + \frac{p_1 q_1 \bar{u}_1 \bar{v}}{(1 + q_1 \bar{u}_1)^2},
\]
\[
B_2 = -\frac{p_1 q_2 \bar{u}_1 \bar{v}}{(1 + q_1 \bar{u}_1)^2} - \frac{\beta \bar{u}_1}{1 + k_2 \bar{v}},
\]
\[
B_3 = -\frac{r_0 k_1 \bar{u}_1}{(1 + k_1 \bar{v})^2} - \frac{p_1 \bar{u}_1}{1 + q_1 \bar{u}_1},
\]
\[
B_4 = \frac{\beta \bar{u}_1}{1 + k_2 \bar{v}} - \mu - \frac{p_2 \bar{v}}{1 + q_1 \bar{u}_1},
\]
\[
B_5 = \frac{c_1 p_1 \bar{v}}{(1 + q_1 \bar{u}_1)^2},
\]
\[
B_6 = \frac{c_2 p_2 \bar{v} + \bar{u}_1 \bar{v}(c_2 p_2 q_1 - c_1 p_1 q_2)}{(1 + q_1 \bar{u}_1)^2}.
\]
The eigenvalues of this Jacobian are \( \lambda_1 = B_4 \) and the roots of the quadratic equation
\[
\lambda^2 - B_1 \lambda - B_3 B_5 = 0.
\]
Here \(-B_3 B_5\) is always positive. Therefore, if \( B_1 < 0 \) then \( E_3 \) is locally asymptotically stable. If \( B_1 > 0 \) then we get Hopf bifurcation.

**Appendix 3**

The Jacobian matrix around the equilibrium point \( E_*(u_1^*, u_2^*, v^*) \) is
\[
J(E_*) = \begin{pmatrix}
C_1 & C_2 & C_3 \\
C_4 & C_5 & C_6 \\
C_7 & C_8 & C_9
\end{pmatrix},
\]
where,
\[
C_1 = -a u_1^* + \frac{p_1 q_1 u_1^* v^*}{(1 + q_1 u_1^* + q_2 u_2^*)^2},
\]
\[
C_2 = -\frac{\beta u_1^*}{1 + k_2 v^*} - \frac{p_1 q_2 u_1^* v^*}{(1 + q_1 u_1^* + q_2 u_2^*)^2},
\]
\[
C_3 = -\frac{r_0 k_1 u_1^*}{(1 + k_1 v^*)^2} - \frac{p_1 u_1^*}{1 + q_1 u_1^* + q_2 u_2^*} + \frac{\beta k_2 u_1^* u_2^*}{(1 + k_2 v^*)^2},
\]
The characteristic equation is
\[ \lambda^3 - (C_1 + C_5)\lambda^2 + (C_1 C_5 - C_6 C_8 - C_2 C_4 - C_3 C_7)\lambda 
+ (C_1 C_6 C_8 + C_3 C_5 C_7 - C_2 C_6 C_7 - C_3 C_4 C_8) = 0, \]
i.e. \( \lambda^3 + \psi_1 \lambda^2 + \psi_2 \lambda + \psi_3 = 0. \)

Where \( \psi_1 = -(C_1 + C_5), \psi_2 = (C_1 C_5 - C_6 C_8 - C_2 C_4 - C_3 C_7), \psi_3 = (C_1 C_6 C_8 + C_3 C_5 C_7 - C_2 C_6 C_7 - C_3 C_4 C_8). \) \( E^* \) is locally asymptotically stable if all the eigenvalues are negative or have negative real part. If the coefficients of the characteristic equation satisfy the Routh–Hurwitz stability criterion, i.e. \( \psi_1 > 0, \psi_3 > 0 \) and \( \psi_1 \psi_2 > \psi_3, \) then the equilibrium point is stable.