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Dynamical Behavior of an Eco-epidemiological Model Incorporating Prey Refuge and Prey Harvesting

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Abstract

In this paper an eco-epidemiological model incorporating a prey refuge and prey harvesting with disease in the prey-population is considered. Predators are assumed to consume both the susceptible and infected prey at different rates. The positivity and boundedness of the solution of the system are discussed. The existence and stability of the biologically feasible equilibrium points are investigated. Numerical simulations are performed to support our analytical findings.

Keywords: Eco-epidemiological model; Disease in prey; Prey harvesting; Prey refuge; Endemic equilibrium point; Disease free equilibrium point; Local stability

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1. Introduction

The dynamical relationship between a predator and a prey is one of the dominant themes in both ecology and mathematical ecology due to its universal existence and importance (Berryman (1992)). Alfred James Lotka and Vito Volterra have done fundamental work on formulating and analyzing the first predator prey model.

Nature can provide some degree of protection to a given number of prey populations by providing refuges. Such refugia can help in prolonging prey predator interactions by reducing the chance of extinction due to predation (Huang et al. (2006); Kar (2005)) and damp prey predator oscillations (Collings (1995)). The effects of prey refuges on the population dynamics are very complex in nature, but for modeling purposes, it can be considered as constituted by two components: the first effects, which affect positively the growth of prey and negatively that of predators, comprise the reduction of prey mortality due to decrease in predation success. The second one may be the trade-offs and by-products of the hiding behavior of prey which could be advantageous or detrimental for all the interacting populations (Gonzalez-Olivares et al. (2003)).

In the literature's studies show that refuges have both stabilizing (Hassell (1974)) and destabilizing effect (McNair (1986)). The traditional ways in which the effect of refuge used by the preys has been incorporated in predator prey models is to consider either a constant number or a constant proportion of the prey population being protected from predation (Smith (1978)). Hassell (Hassell (1974)) notes that in reality refugia fall between these two extremes. It is pointed out that those protecting a proportion of the prey population appearing to be more common (Collings (1995)). However, as pointed out by the authors (Gonzalez-Olivares et al. (2003); Krivan (1998); Ma et al. (2009)), the refuges, which protect a constant number of preys, have a stronger stabilizing effect on population dynamics than the refuges, which protect a constant proportion of prey. For more biological backgrounds and results on the effects of a prey refuge, one could refer to (Collings (1995); Kar (2006); Ko and Ryu (2006); Krivan (1998); McNair (1986); McNair (1987); Sih (1987)) and the references therein.

In addition, many of the species in the natural ecosystem are being mined and harvested. The exploitation of biological resources and harvest of population species are commonly practiced in fishery, forestry and wildlife management. Concerning the conservation for the long-term benefits of humanity, there is a wide-range of interest in the use of bio-economic modelling to gain insight in the scientific management of renewable resources like fisheries and forestries (Tao et al. (2016)).

The mathematical modeling of epidemics has become a very important subject of research after the seminal model of Kermack-McKendric (Kermack and McKendrick (1927)) on SIRS systems, in which the evolution of a disease which gets transmitted upon contact is described. A lot of research work has been done in this area (see (Bailey (1975); Anderson and May (1981); Ma et al. (2009); Juneja and Agnihotri (2018); Kant and Kumar (2017a); Kant and Kumar (2017b)) and the references therein)

Ecology and epidemiology are major fields of study in their own right. But there are some common features between these systems. Due to these common features, mathematical biologists have been working on merging these two major areas of interest for a long time (Bhattacharya et al. (2014)). It is very important both from the ecological and mathematical points of view to study ecological systems under the influence of epidemiological factors. Anderson and May (Anderson and May (1981)) who were the pioneers for formulating an eco-epidemiological predator prey model and investigated the invasion, persistence, and spread of diseases. After the work of Anderson and May (Anderson and May (1981)), many eco-epidemiological researchers study ecological systems with disease either in prey (De Rossi et al. (2015); Saifuddin et al. (2017); Sarwardi et al. (2011); Zhou et al. (2010); Rahman and Chakravarty (2013); Sharma and Samanta (2014); Kant and Kumar (2017a); Nandi et al. (2015); Oliveira and Hilker (2010); Kooi and Venturino (2016); Sahoo (2016)) or in predator (Biswas et al. (2018); Das (2011); Das (2015); Das (2016); Haque (2010); Haque and Venturino (2007); Juneja and Agnihotri (2018); Sahoo (2016); Sahoo and Poria (2016); Venturino (2004); Jana et al. (2013); Wang et al. (2016); Mbava et al. (2017)) or in both populations (Gao et al. (2013); Bera et al. (2015); Agnihotri and Gakkhar (2012); Kant and Kumar (2017b)).

Some researchers have analyzed an eco-epidemiological predator-prey model incorporating a prey refuge in the system (Wang et al. (2018); Wang et al. (2012); Maji et al. (2019); Kant and Kumar (2015)). On the other hand, the authors (Chakraborty et al. (2010); Bhattacharyya and Mukhopadhyay (2010); Bhattacharya et al. (2014)) have considered an eco-epidemiological predator prey system with disease in prey species only and harvesting of both susceptible and infected prey. Agnihotri and Gakkhar (Agnihotri and Gakkhar (2012)) have studied a prey predator system with disease on both the species and aharvesting of the prey species only. Abdulghafour and Naji (Abdulghafour et al. (2018)) investigated an eco-epidemiological prey-predator model involving a prey refuge and harvesting from the predator. As far as our knowledge concerns, no one has considered the effect of prey refuge, in the infected prey only, and prey harvesting in an eco-epidemiological predator prey system.

The main aim of this paper is to study the effect of infection in prey, prey refuge and prey harvesting in a predator prey system. Here, we have studied the boundedness, positivity of the solution, local and global stabilities of the equilibrium points of this system.

The organization of this paper is as follows: Section 2 deals with the model formulation. Some preliminary results of the proposed model are discussed in Section 3. The exclusion criteria and Existence and stability of the biologically feasible equilibrium points are discussed in Sections 4 and 5, respectively. Numerical studies are carried out in Section 6 and conclusion is given in Section 7.

2. The Mathematical Model Formulation

2.1. Model Assumptions

A mathematical model of a predator-prey system with infection in prey, prey refuge and harvesting is considered here. Let $N(t)$ and $P(t)$ represent the total prey population density and the predator population density, respectively. The following assumptions are taken for the formulation of our eco-epidemiological model.

(1) In the absence of disease, the prey population grows logistically with carrying capacity K and intrinsic birth rate r.
(2) In the presence of disease, the prey population is divided into two groups, namely susceptible prey denoted by $S(t)$ and infected prey denoted by $I(t)$. Therefore, at time t, the total population is $N(t) = S(t) + I(t)$.
(3) The susceptible prey is capable of reproducing only and the infected prey is removed by death at a natural rate d_1.
(4) The disease spreads among the prey population only by contact, and can not be transmitted vertically. The infected prey species do not recover or become immune. We assume that the disease transmission follows the simple law of mass action $\lambda S(t)I(t)$ with λ as the transmission rate.
(5) The infected prey species are assumed to take a refuge. That is $(1 - m)I$, m is prey refuge constant, of the infected prey is available for predation.
(6) Predators predate both susceptible and infected prey following a Holling type-II functional response with predation coefficients α_1 and b_1, respectively. The consumed prey is converted into predator with efficiency c.
(7) The predators suffer loss due to natural death at a constant rate d_2.
(8) The susceptible and infected preys are harvested by an external force according to linear type of functional harvesting.

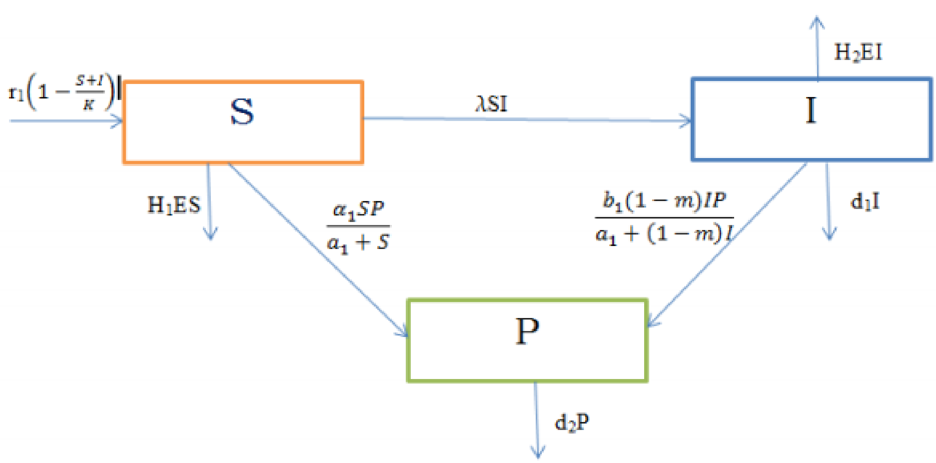

Figure 1. The Flow Diagram of the Model

2.2. Model Equation

Based on the above assumptions, parameters and flow diagram, the eco-epidemiological model is given by the following set of nonlinear differential equations.

$$\begin{aligned}\frac{dS}{dT} &= r_1 S\left(1-\frac{S+I}{K}\right) - \lambda IS - \frac{\alpha_1 SP}{a_1+S} - H_1E_1S,\\ \frac{dI}{dT} &= \lambda IS - d_1 I - \frac{b_1(1-m)IP}{a_1+(1-m)I} - H_2E_2I,\\ \frac{dP}{dT} &= -d_2P + \frac{cb_1(1-m)IP}{a_1+(1-m)I} + \frac{c\alpha_1 SP}{a_1+S},\end{aligned} \tag{1}$$

and the initial conditions are given as

$$S(0) = S_0 > 0,\ \ I(0) = I_0 > 0,\ \ P(0) = P_0 > 0.$$

We assume that all parameters of system (1) are positive. The detailed biological meanings of parameters are given in Table 1.

Table 1. Biological meaning of parameters

Parameters	**Biological meaning**
r	The intrinsic growth rate of prey,
K	The carrying capacity of the environment,
a_1	The half-saturation constant,
α_1	Predation rate of susceptible prey
b_1	Predation rate of infected prey
c	Conversion coefficient from the prey to predator
d_1	The death rate of infected prey.
d_2	The death rate of predator population,
λ	The infection rate,
m	The pey refuge constant
H_1	The catchability coefficient of the susceptible prey,
H_2	The catchability coefficient of the infected prey,
E	Harvesting effort

In order to reduce the number of parameters of the system (2.2), it is convenient to scale the variables as $s = \frac{S}{K}$, $i = \frac{I}{K}$, $p = \frac{P}{K}$, and to consider the dimensionless time $t = \lambda KT$. This transformation leads to the dimensionless equations

$$
\begin{aligned}
\frac{ds}{dt} &= rs(1-s-i) - si - \frac{\alpha sp}{a+s} - h_1 s,\\
\frac{di}{dt} &= is - di - \frac{\theta(1-m)ip}{a+(1-m)i} - h_2 i,\\
\frac{dp}{dt} &= -\delta p + \frac{c\theta(1-m)ip}{a+(1-m)i} + \frac{c\alpha sp}{a+s},
\end{aligned}
\tag{2}
$$

where

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

The initial conditions for the system (2) are given as

$$
s(0) = s_0 \geq 0,\ \ i(0) = i_0 \geq 0,\ \ p(0) = p_0 \geq 0.
$$

3. Preliminary Results

3.1. Existence and Positive Invariance

For $t > 0$, let $Y \equiv (s(t), i(t), p(t))^T$ and $F(Y) = (F_1(Y), F_2(Y), F_3(Y))^T$, where

$$
F_1(Y) = rs(1-s-i) - si - \frac{\alpha sp}{a+s} - h_1 s,\ \ F_2(Y) = is - di - \frac{\theta(1-m)ip}{a+(1-m)i} - h_2 i,
$$
$$
F_3(Y) = -\delta p + \frac{c\theta(1-m)ip}{a+(1-m)i} + \frac{c\alpha sp}{a+s}.
$$

Then, system (2) can be written as $\frac{dY}{dt} = F(Y)$ where $F : C_+ \to (\mathcal{R})^3_+$ with $Y(0) = Y_0 \in \mathcal{R}^3_+$. Here $F_i \in C^\infty(\mathcal{R})$ for $i = 1, 2, 3$. Thus, the function F is locally Lipschitzian and completely continuous on $\mathcal{R}^3_+$. Therefore, the solution of the system (2) with non-negative initial condition exists and is unique. Moreover, It can be shown that these solutions exist for all $t > 0$ and stay non-negative. Hence, the region $\mathcal{R}^3_+$ is an invariant domain of the system (2).

3.2. Boundedness

In theoretical eco-epidemiology model, boundedness of a system implies that the system is biologically well behaved. The following theorem ensures the boundedness of system (2).

Theorem 3.1.

All solutions of the system (2) starting in $\mathcal{R}^3_+$ are uniformly bounded.

Proof:

Let $s(t), i(t)$ and $p(t)$ be any solution of the system (2) with positive initial conditions. Since,

$$\frac{ds}{dt} \leq rs(1-s),$$

we have $\limsup_{t\to\infty} s(t) \leq 1$.

Let $w = s + i + p$. Taking the time derivative of w along the solutions of system (2) gives

$$\begin{aligned}\frac{dw}{dt} &= rs(1-s) - (1+r)si - \frac{(1-c)\alpha sp}{a+s} - h_1 s + is - di - \frac{(1-c)\theta(1-m)ip}{a+(1-m)i} - h_2 i - \delta p \\ &\leq rs(1-s) - h_1 s - (d+h_2)i - \delta p \ \ (\text{since } \ c<1) \\ &\leq \frac{r}{4} - h_1 s - (d+h_2)i - \delta p \left(\text{since } \ Max\{rs(1-s)\} = \frac{r}{4}\right) \\ &\leq \frac{r}{4} - \gamma w, \ \ \text{where } \ \gamma = min\{h_1, d+h_2, \delta\}.\end{aligned}$$

Hence, we have

$$\frac{dw}{dt} + \gamma w \leq \frac{r}{4}.$$

Applying the theory of differential inequality, we obtain

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

For $t \to \infty$, we have $0 < w < \frac{r}{4\gamma}$. Hence, all the solutions system (2) starting in $\mathcal{R}^3_+$ for any $\epsilon > 0$ are confined in the region

$$\Omega = \{(s,i,p) \in \mathcal{R}^3_+ : s + i + p \leq \frac{r}{4\gamma} + \epsilon\}.$$

Hence, the result. ■

4. Extinction Criteria

Theorem 4.1.

If $r < h_1$, then $\lim_{t\to\infty} s(t) = 0$. If $r < d + h_2$, then $\lim_{t\to\infty} i(t) = 0$. If $c(\theta+\beta) < \delta$, then $\lim_{t\to\infty} p(t) = 0$.

Proof:

For the susceptible prey, we have

$$\frac{ds}{dt} = rs(1-s-i) - si - \frac{\alpha sp}{a+s} - h_1 s \leq (r - h_1)s.$$

Therefore, $s(t) \leq s_0 \exp\{\int_0^t (r-h_1)dq\}$. Thus, if $r < h_1$, then $\lim_{t\to\infty} s(t) = 0$.

For the infected prey we have

$$\frac{di}{dt} = is - di - \frac{(1-m)ip}{a+(1-m)I} - h_2 i \leq (1-d-h_2)i, \ \ (since \ \ s(t) \leq 1).$$

Therefore, $i(t) \leq i_0 \exp\{\int_0^t (1-d-h_2)dq\}$. Thus, if $d+h_2>1$, then $\lim_{t\to\infty} i(t)=0$.

For the predator, we have

$$\begin{aligned}\frac{dp}{dt} &= -\delta p + \frac{c(1-m)ip}{a+(1-m)i} + \frac{\beta sp}{a+s}\\ &\leq (c(\theta+\beta)-\delta)p, \ \ (since \ \frac{s}{a+s}<1, \ \ \frac{(1-m)i}{a+(1-m)i}<1).\end{aligned}$$

Therefore, $p(t) \leq p_0 \exp\{\int_0^t (c(\theta+\beta)-\delta)dq\}$. Thus, if $c(\theta+\beta)<\delta$, then $\lim_{t\to\infty} p(t)=0$. ■

5. Equilibrium points and their stability

The system (2) has four boundary equilibrium points and the coexistence equilibrium point.

5.1. Boundary Equilibrium Points

(1) The trivial equilibrium point: $E_0(0,0,0)$.
(2) The Infected Prey and Predator-Free Equilibrium Point: $E_1(\frac{r-h_1}{r},0,0)$. E_1 exists for $h_1<r$.
(3) The Disease Free Equilibrium Point: $E_2(\tilde{s},0,\tilde{p})$ where $\tilde{s}=\frac{a\delta}{c\alpha-\delta}$ and $\tilde{p}=\frac{ac((c\alpha-\delta)(r-h_1)-ar\delta)}{(c\alpha-\delta)^2}$. E_2 exists for $\delta<c\alpha$ and $h_1<r(1-\frac{a\delta}{c\alpha-\delta})$.
(4) The Predator-Free Equilibrium Point: $E_3(\bar{s},\bar{i},0)$ where $\bar{s}=d+h_2$ and $\bar{i}=\frac{r(1-d-h_2)-h_1}{r+1},0)$. E_3 exists for $h_1<r(1-d-h_2)$.

5.2. The Positive Interior Equilibrium Point (The Endemic Equilibrium Point)

Theorem 5.1.

The positive interior equilibrium point of the system (2) $E_4(s^*,i^*,p^*)$ exists for

$$c\alpha<\delta, \ \ h_1<r, \ \ \frac{a\delta(1+r)}{c\alpha}-d<h_2<s^*-d, \quad (\delta-c\alpha)s^*+a\delta>0, \tag{3}$$

where

$$i^*=\frac{a(a\delta+(\delta-c\alpha)s^*)}{(1-m)(c\alpha s^*+(c\theta-\delta)(a+s^*))}, \quad p^*=\frac{ac(s^*-d-h_2)(a+s^*)}{(1-m)(c\alpha s^*+(c\theta-\delta)(a+s^*))}$$

and s^* is the unique positive root of the quadratic equation

$$As^2+Bs+C=0, \tag{4}$$

with

$$\begin{aligned}&A=r(1-m)(c\alpha+c\theta-\delta), C=-a((1-m)(r-h_1)(c\theta-\delta)+(c\alpha(d+h_2)-a\delta(1+r))),\\ &B=(1-m)((c\theta-\delta)(h_1-r+ar)+\alpha c(h_1-r))+a(\delta+(\delta-c\alpha)r).\end{aligned}$$

Proof:

The quadratic equation 4 will have a unique positive root provided the product of the coefficients A and C is negative. It is easy to see that under the condition 3, $A > 0$ and $C < 0$. Hence, the quadratic equation 4 has the unique positive root s^*. The positivity of s^* and the conditions given in 3 gives us that $i^* > 0$ and $p^* > 0$.

Hence, the result. ■

5.3. Local stability analysis

In order to investigate the local stability property of the system (2), we shall calculate the Jacobian matrix at each equilibrium. The Jacobian matrix of the system (2) at any arbitrary equilibrium $E(s, i, p)$ is given as

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

where

$$a_{11} = r(1-2s) - i(r+1) - \frac{\alpha a p}{(a+s)^2} - h_1, \;\; a_{12} = -s(r+1), \;\; a_{13} = -\frac{\alpha s}{a+s},$$
$$a_{22} = s - d - h_2 - \frac{a\theta(1-m)p}{(a+(1-m)i)^2}, \;\; a_{21} = i, \;\; a_{23} = -\frac{\theta(1-m)i}{(a+(1-m)i)},$$
$$a_{31} = \frac{ac\alpha p}{(a+s)^2}, \;\; a_{32} = \frac{ac\theta(1-m)p}{(a+(1-m)i)^2}, \;\; a_{33} = -\delta + \frac{c\theta(1-m)i}{a+(1-m)i} + \frac{\alpha cs}{a+s}.$$

Theorem 5.2.

The trivial equilibrium point $E_0(0,0,0)$ is locally asymptotically stable if $r < h_1$. Otherwise, it is unstable.

Proof:

The Eigenvalues at $E_0(0,0,0)$ are $-d-h_2, -h_1+r, -\delta$. Hence, $E_0(0,0,0)$ is locally asymptotically stable only if $r < h_1$ and unstable otherwise. ■

Theorem 5.3.

The infected prey and predator free equilibrium point $E_1(\frac{r-h_1}{r}, 0, 0)$ is locally asymptotically stable if $c\alpha < \delta$ and $h_1 > r(1-d-h_2)$.

Proof:

The Eigenvalues at $E_1(\frac{r-h_1}{r}, 0, 0)$ are $1 - d - h_2 - \frac{h_1}{r}, \;\; h_1 - r, \;\; c\alpha\left(\frac{r-h_1}{r-h_1+ar}\right) - \delta$. Hence,

$E_1(\frac{r-h_1}{r}, 0, 0)$ is locally asymptotically stable if $c\alpha < \delta$ and $h_1 > r(1-d-h_2)$. ■

Theorem 5.4.

The disease free equilibrium point $E_2\left(\frac{a\delta}{c\alpha-\delta}, 0, \frac{ac((c\alpha-\delta)(r-h_1)-ar\delta)}{(c\alpha-\delta)^2}\right)$ is locally asymptotically stable if

$$\min\left\{\frac{a\delta}{c\alpha-\delta} - d,\ r\left(1 - \frac{2a\delta}{c\alpha-\delta}\right)\right\} < h_1. \tag{5}$$

Proof:

The entries of the Jacobean matrix J evaluated at the equilibrium point E_2 are

$$a_{11} = -h_1 + r - \frac{2ar\delta)}{(c\alpha-\delta)} - \frac{(c\alpha-\delta)^2\tilde{p}}{(a\alpha c^2)}, a_{12} = -\frac{a(1+r)\delta}{c\alpha-\delta}, a_{13} = -\frac{\delta}{c}, a_{21} = 0, a_{23} = 0,$$
$$a_{22} = -d - h_2 + \frac{a\delta}{c\alpha-\delta} - \frac{(1-m)\theta\tilde{p}}{a}, a_{31} = \frac{(c\alpha-\delta)^2\tilde{p}}{ac\alpha}, a_{32} = \frac{c(1-m)\theta\tilde{p}}{a},\ a_{33} = 0.$$

The characteristic equation corresponding to $J(E_2)$ is

$$\lambda^3 + A\lambda^2 + B\lambda + C = 0, \tag{6}$$

where

$$A = -a_{11} - a_{22},\ \ B = -a_{31}a_{13} + a_{22}a_{11},\ \ C = a_{13}a_{22}a_{31}.$$

According to Routh-Hurwitz criteria, all the roots of the characteristics equation (6) have negative real parts if and only if A, C and $AB - C$ are positive.

From the sign of the entries of the Jacobean matrix $J(E_2)$, we can see that C becomes positive when a_{22} is negative. The negativeness of a_{22} and the signs of the entries of the matrix $J(E_2)$ guarantees us to take $a_{11} < 0$ as a sufficient condition for A and $AB - C$ to be positive as $AB - C = -a_{11}a_{22}(a_{11} + a_{22}) + a_{11}a_{13}a_{31}$. Now, the sufficient conditions for a_{11} and a_{22} to be negative are $\frac{a\delta}{c\alpha-\delta} - d < h_1$ and $r(1 - \frac{2a\delta}{c\alpha-\delta}) < h_1$, respectively.

Therefore, the disease free equilibrium point E_2 is locally asymptotically stable provided the condition 5 is satisfied. Hence, the result. ■

Theorem 5.5.

The predator free equilibrium point $E_3(\bar{s}, \bar{i}, 0)$ is locally asymptotically stable if

$$\delta > c(\alpha + \theta). \tag{7}$$

Proof:

The entries of the Jacobean matrix J evaluated at the equilibrium point E_3 are

$$a_{11} = -(d+h_2)r,\ \ a_{12} = (-1-r)\bar{s},\ \ a_{13} = -\frac{\alpha\bar{s}}{a+\bar{s}},\ \ a_{21} = \bar{i},\ \ a_{22} = 0,$$
$$a_{23} = -\frac{(1-m)\theta\bar{i}}{a+(1-m)\bar{i}}, a_{31} = 0,\ \ a_{32} = 0,\ \ a_{33} = \frac{c\alpha\bar{s}}{a+\bar{s}} - \delta + \frac{c(1-m)\theta\bar{i}}{a+(1-m)\bar{i}}.$$

The characteristic equation corresponding to $J(E_3)$ is

$$\lambda^3 + A\lambda^2 + B\lambda + C = 0, \tag{8}$$

where

$$A = -a_{11} - a_{33}, \;\; B = -a_{21}a_{12} + a_{33}a_{11}, \;\; C = a_{12}a_{21}a_{33}.$$

According to Routh-Hurwitz criteria, all the roots of the characteristics equation (8) have negative real parts if and only if A, C and $AB - C$ are positive.

From the sign of the entries of the Jacobean matrix $J(E_3)$, we can see that C becomes positive when a_{33} is negative. Thus, if a_{33} is negative, then, we can see that $A > 0$ and $AB - C > 0$, where $AB - C = -a_{11}(-a_{12}a_{21} + a_{33}(a_{33} + a_{11}))$. Now, the sufficient condition for a_{33} to be negative is $\delta > c(\alpha + \theta)$. Therefore, the predator free equilibrium point E_3 is locally asymptotically stable provided the condition 7 is satisfied. Hence, the result. ■

Table 2. Summary of Existence and Stability Conditions for the Boundary Equilibrium Points

Equilibrium	Existence Conditions	Stability Conditions	Remark
$E_0(0,0,0)$	Unconditional	$r < h_1$	Theorem 5.2
$E_1(\frac{r-h_1}{r},0,0)$	$h_1 < r$	$h_1 > r(1-d-h_2)$, $\delta > c\alpha$	Theorem 5.3
$E_2(\tilde{s},0,\tilde{p})$	$h_1 < r\left(1 - \frac{a\delta}{c\alpha-\delta}\right)$, $\delta > c\alpha$	$\min\left\{\frac{a\delta}{c\alpha-\delta} - d, r\left(1 - \frac{2a\delta}{c\alpha-\delta}\right)\right\} < h_1$	Theorem 5.4
$E_3(\bar{s},\bar{i},0)$	$h_1 < r(1-d-h_2)$	$\delta > c(\alpha+\theta)$	Theorem 5.5

The existence and stability conditions for the Boundary Equilibrium Points are summarized in Table 2.

Remark 5.1.

From Table 2, one can easily see that

(1) If E_0 is stable, then all the rest equilibrium points. E_1, E_2 and E_3 do not exist.
(2) If E_1 is stable, then the equilibrium points E_2 and E_3 do not exist.
(3) If E_3 is stable, then E_2 does not exist.
(4) If E_3 exists, then E_1 becomes unstable.

Theorem 5.6.

The positive endemic equilibrium point of E^* of the system (2) is locally asymptotically stable if

$$A > 0, \;\; C > 0, \;\; AB - C > 0, \tag{9}$$

where

$$A = -a_{11} - a_{22}, \;\; B = -a_{21}a_{12} + a_{22}a_{11} - a_{13}a_{31} + a_{23}a_{32},$$
$$C = a_{13}(-a_{22}a_{31} + a_{21}a_{32}) + a_{23}(a_{12}a_{31} - a_{11}a_{32}),$$

and $a_{ij}(i,j=1,12,3)$ are the entries of the Jacobean matrix at E^* which are given as

$$a_{11}=-\frac{s^*(h_1-r+ar+(1+r)i^*+2rs^*)}{a+s^*},\ \ a_{12}=-(r+1)s^*,\ \ a_{13}=-\frac{\alpha s^*}{a+\bar{s}},$$
$$a_{21}=i^*,\ \ a_{22}=\frac{a\theta(1-m)^2i^*p^*}{(a+(1-m)i^*)^2},\ a_{23}=-\frac{(1-m)\theta i^*}{a+(1-m)i^*},$$
$$a_{31}=\frac{ac\alpha p^*}{(a+s^*)^2},\ a_{32}=\frac{ac(1-m)\theta p^*}{(a+(1-m)i^*)^2},\ a_{33}=0.$$

Proof:

The characteristic equation of the Jacobean matrix at the positive endemic equilibrium point E^* is

$$\lambda^3+A\lambda^2+B\lambda+C=0. \tag{10}$$

According to Routh-Hurwitz criteria, all the roots of the characteristics equation (10) have negative real parts if and only if A, C and $AB-C$ are positive. Therefore, the positive endemic equilibrium point E^* is locally asymptotically stable provided the condition 9 is satisfied. Hence, the result. ∎

6. Numerical Simulations

In this section, several numerical simulations on the system (2) are performed in order to verify the theoretical findings. In the present study, the rate of harvesting (h_1), predation rate (α) and refuge coefficient (m) are the key parameters, which will be taken as control parameters. The numerical simulation is carried out using MATLAB software package for the set of parameter values given in table 3 below.

Table 3. Parametric values of the system (2)

Parameter	Numeric Value
r	0.5
a	0.2
d	0.1
c	0.5
δ	0.1
θ	0.4
h_2	0.15
α	variable
h_1	variable
m	variable

6.1. Effect of varying the predation rate α

Let us fix the variables in Table 3 as $h_1 = 0.2$ and $m = 0.2$. For the given parametric values, the disease free equilibrium point E_2 and the endemic equilibrium point E_4 exists for $\alpha > 0.26667$ and $0.130909 < \alpha < 0.283305$, respectively. It is observed that system (2) approaches the disease free equilibrium point for $\alpha < 0.283305$, while it approaches the endemic equilibrium point when $0.130909 < \alpha < 0.283305$. Figure 2(a) shows the stability of E_2 for $\alpha = 0.3$ and Figure 2(b) shows the stability of E_4 for $\alpha = 0.28$.

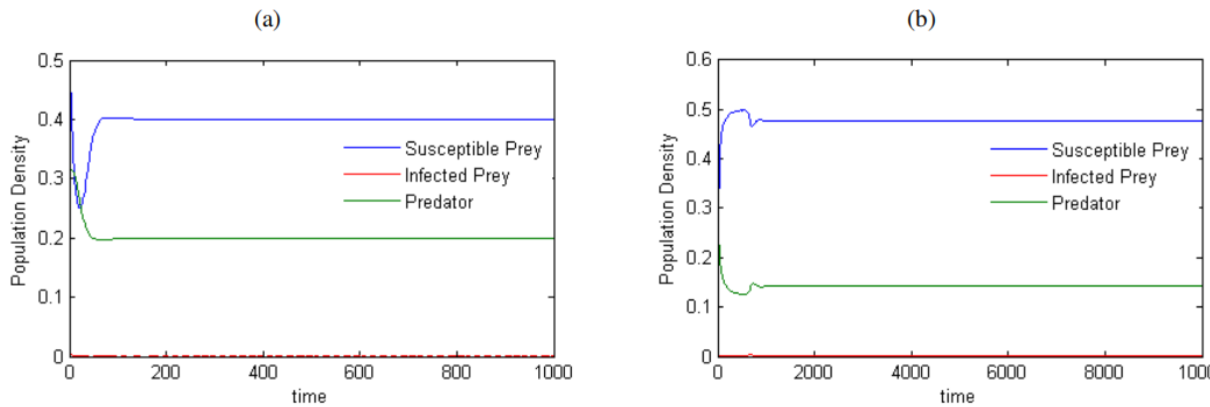

Figure 2. *The time series solution of the system (2) (a) around the equilibrium point E_2 with parametric values as in table 3 except ($h_1 = 0.2$, $m = 0.2$ and $\alpha = 0.3$)., (b) around the equilibrium point E_4 with parametric values as in table 3 except ($h_1 = 0.2$, $m = 0.2$ and $\alpha = 0.28$)*

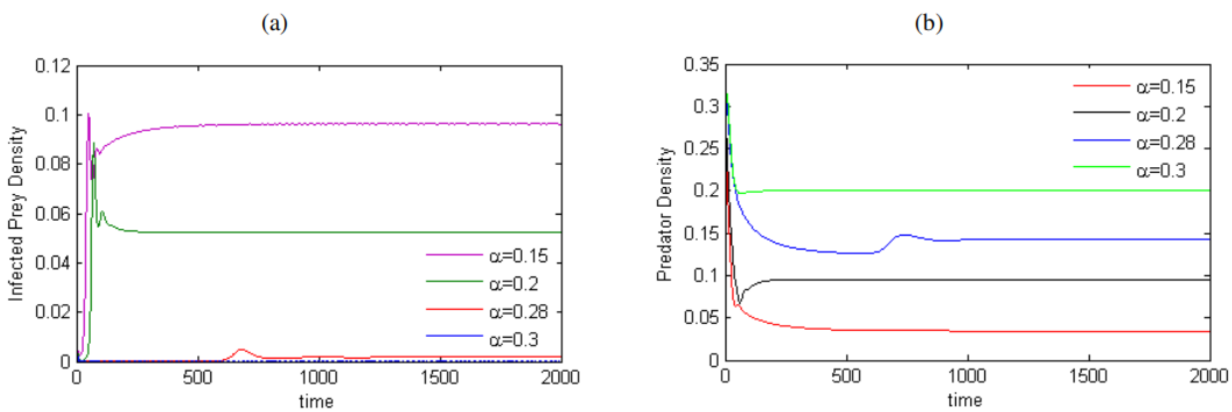

Figure 3. *(a) The density of infected prey population for the parametric values as in table 3 except ($h_1 = 0.2$, $m = 0.2$) and $\alpha = 0.15,\ 0.2,\ 0.28,\ 0.3$., (b) The density of predator population for the parametric values as in table 3 except ($h_1 = 0.2$, $m = 0.2$) and ($\alpha = 0.15,\ 0.2,\ 0.28,\ 0.3$)*

Figure 3(a) shows that an increase in the susceptible predation rate results in a decrease in infected prey population. Whereas, as the rate of susceptible prey predation rate increases the predator population density increases, see Figure 3(b).

6.2. Effect of varying the harvesting rate h_1

For the parametric values as in table 3 with $\alpha = 0.25$ and $m = 0.2$, the disease free equilibrium point E_2 and the endemic equilibrium point E_4 exists for $h_1 < 0.1$ and $0.0140625 < h_1 < 0.307377$, respectively. It is observed that system (2) approaches the disease free equilibrium point for $h_1 < 0.0148768$, while it approaches the endemic equilibrium point when $0.0140625 < h_1 < 0.307377$. Thus, Figure 4 shows the stability of E_2 for $h_1 = 0.01$ and Figure 5 shows the stability of E_4 for $h_1 = 0.08$.

From Figure 6 (a), Figure 6(b) and Figure 6(c), it can be observed that an increase in the harvesting rate of susceptible prey leads to a decrease in susceptible prey and predator population whereas an increase in infected prey population.

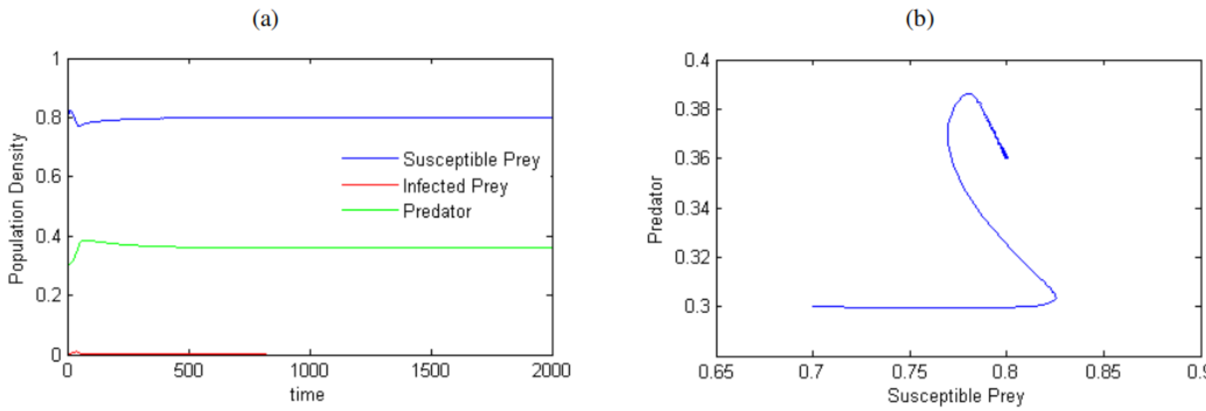

Figure 4. *(a) The time series solution of the system (2) about the equilibrium point E_2 with parametric values as in table 3 except ($\alpha = 0.25$, $m = 0.2$ and $h_1 = 0.01$)., (b) The Parametric Plot of the equilibrium point E_2 with parametric values as in table 3 except ($\alpha = 0.25$, $m = 0.2$ and $h_1 = 0.01$)*

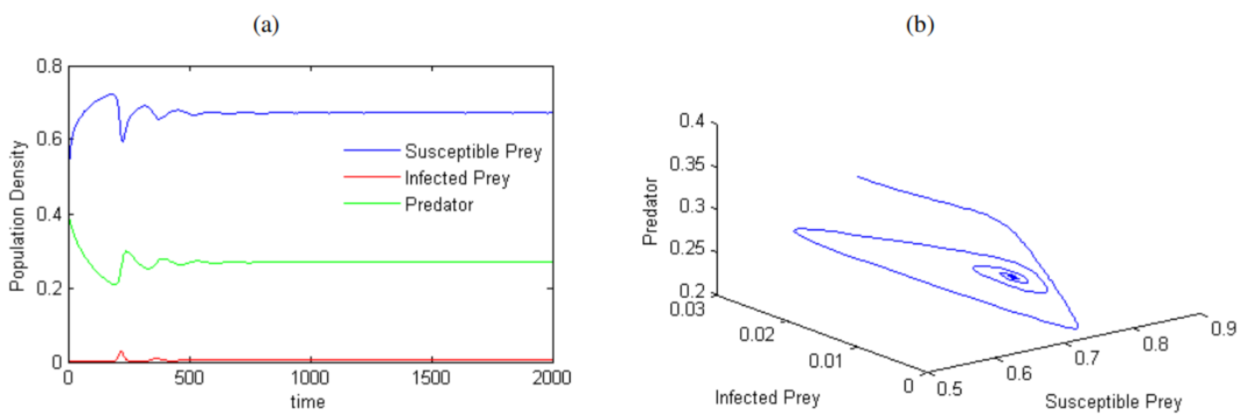

Figure 5. *(a) The time series solution of the system (2) about the equilibrium point E_4 with parametric values as in table 3 except ($\alpha = 0.25$, $m = 0.2$ and $h_1 = 0.08$)., (b) The Parametric Plot of the equilibrium point E_4 with parametric values as in table 3 except ($\alpha = 0.25$, $m = 0.2$ and $h_1 = 0.08$)*

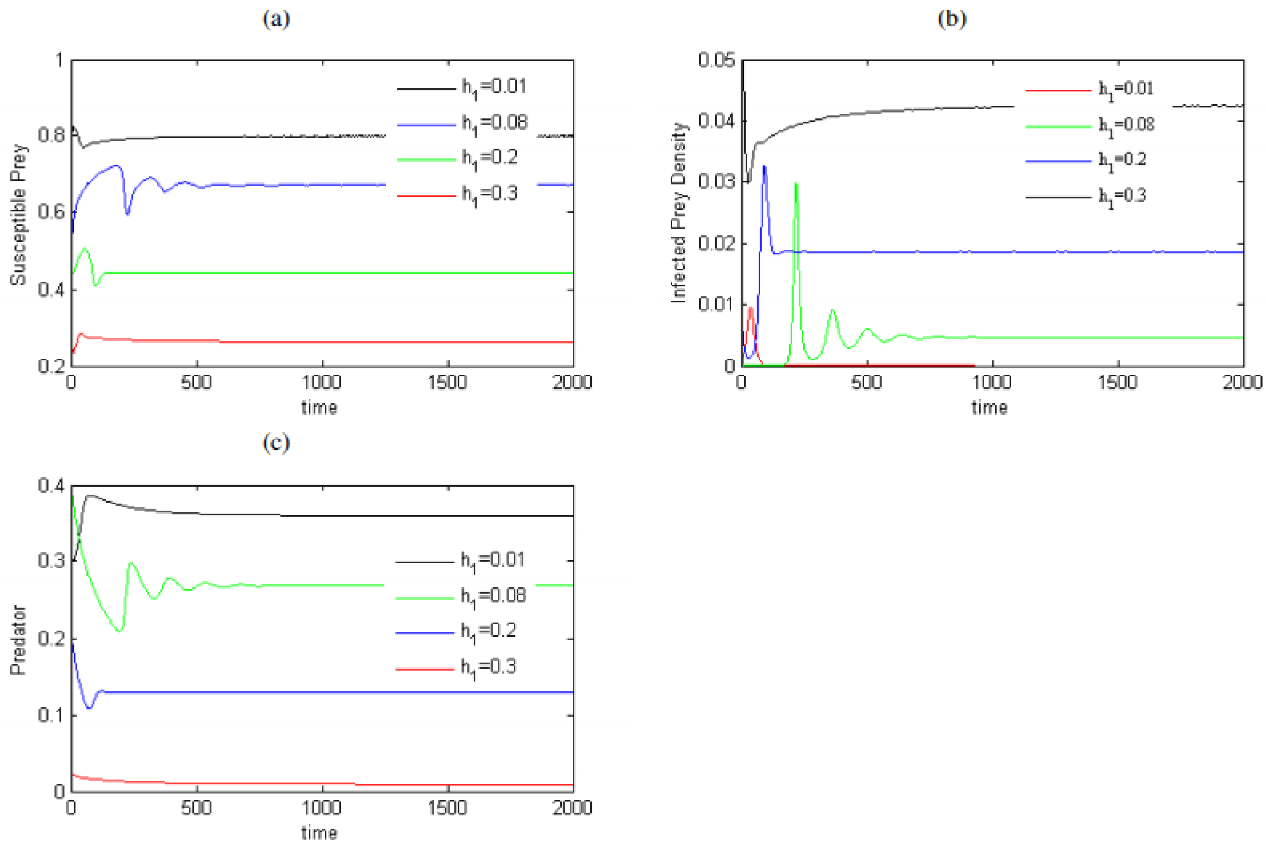

Figure 6. *The density of (a) Susceptible prey population, (b) infected prey population, (c) predator population for the parametric values as in table 3 except (*$\alpha = 0.25$*,* $m = 0.2$*) and* $h_1 = 0.01,\ \ 0.08,\ \ 0.2,\ \ 0.3$

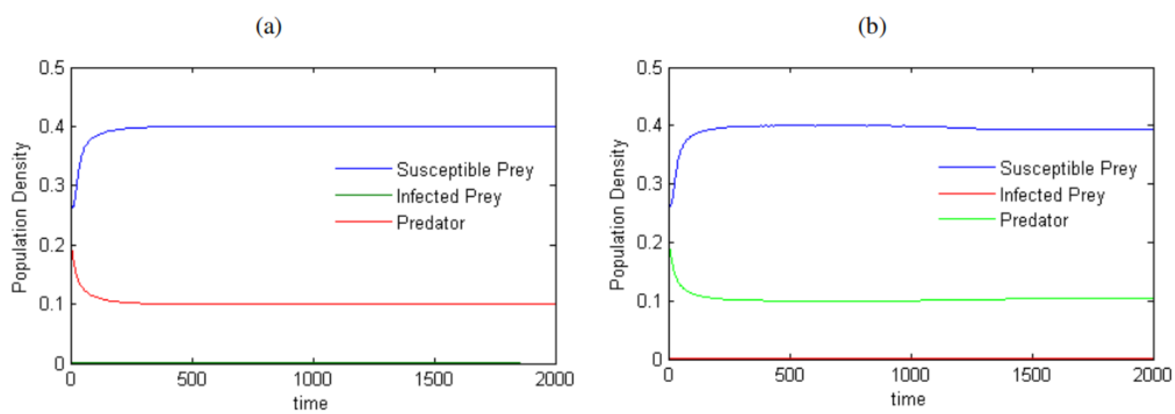

Figure 7. *The time series solution of the system (2) about the equilibrium point (a)* E_2 *with parametric values as in table 3 except (*$\alpha = 0.3$*,* $h_1 = 0.25$*) and* $m = 0.2$*., (b)* E_4 *with parametric values as in table 3 except (*$\alpha = 0.3$*,* $h_1 = 0.25$*) and* $m = 0.3$

6.3. Effect of varying the refuge constant m

It is observed that for the parametric values given in Table 3 with $\alpha = 0.3$ and $h_1 = 0.25$, the trajectories of the system (2) approaches asymptomatically disease free equilibrium point for $m < 0.25$ while it approaches the endemic equilibrium point for $0.25 < m < 0.781818$. Figure 7(a) shows the stability of E_2 for $m = 0.2$ and figure 7(b) show the stability of E_4 for $m = 0.3$.

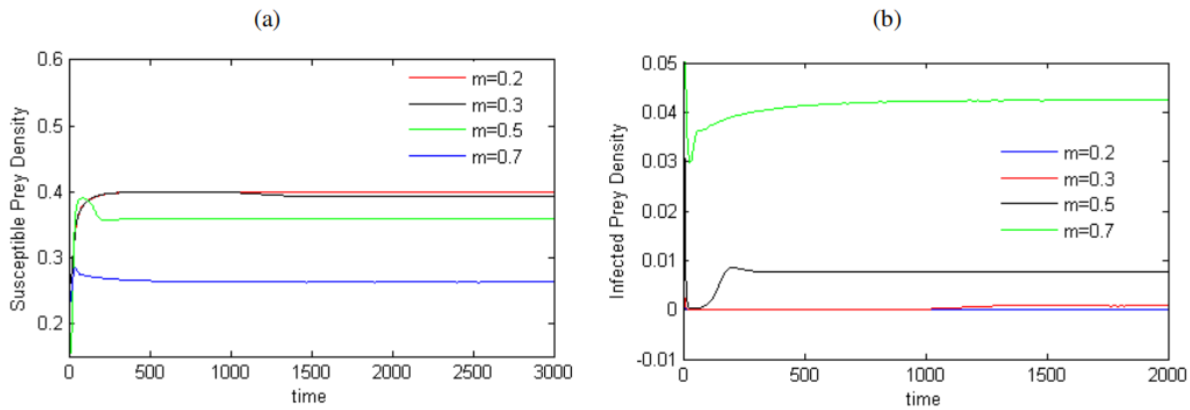

Figure 8. *The density of (a) Susceptible prey population, (b) infected prey population for the parametric values as in table 3 except (*$\alpha = 0.3$*,* $h_1 = 0.25$*) and* $m = 0.2,\ 0.3,\ 0.5,\ 0.7$

From Figure 8(a), we can observe that the density of the susceptible prey population decreases as the refuge constant increases. Figure 8(b) shows an increase in infected prey population as the refuge constant m increases from 0.2 to 0.7.

7. Conclusion

In this paper, we have studied an eco-epidemiological model incorporating a prey refuge and a prey harvesting with disease in the prey population, where the predator predates both the infected and susceptible prey. The boundedness and positivity results show that the developed system 2 is biologically well behaved. Theorem 1 shows that the population go to extinction when the intrinsic growth rate of the susceptible prey is less than the harvesting rate of the susceptible prey.

The local stability of each biologically feasible equilibrium points of the system (2) has been established. The rate of harvesting (h_1), predation rate (α) and refuge coefficient (m) are taken as control parameters. The disease free equilibrium point loses its stability whereas the endemic equilibrium point gains its stability for an increase in infected prey refuge and susceptible prey harvesting rate. However, the disease free equilibrium point becomes stable for an increase in predation rate.

The analytical and numerical results show that harvesting rate, refuge and predation rate have a major impact on each population. Increasing the amount of infected prey refuge decreases the susceptible prey density, whereas the opposite holds for the infected prey density. Increasing the amount of susceptible predation rate results in a decrease in infected prey population and an increase in predator population density. Furthermore, increasing the susceptible prey harvesting rate leads to a decrease in susceptible prey and predator population whereas an increase in infected prey population.

This study shows complex behavior of the proposed model. In particular, when the infected refuge, susceptible prey harvesting rate and susceptible prey predation rate lies in a certain range, the disease free and endemic equilibrium points exist and become stable. The model with infected prey refuge and harvesting of prey gives rise to rich dynamics.

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