DYNAMICS OF A CROWLEY-MARTIN ECO-EPIDEMIOLOGICAL MODEL WITH PREY REFUGE AND PREY HARVESTING

A. Divya[1]*, M. Sivabalan[1], A. Ashwin[1], M. Siva Pradeep[1]

Abstract
In this paper, constructing a three-species food web model involved using the interactions between diseased predator-prey models. The logistically growing prey population is split into two categories: susceptible and infected prey. Presumably, the prey population expands logistically in the absence of predators. In Crowley-Martin-type interactions, it is assumed that interdependence between predators occurs regardless of whether an individual predator is searching for prey or handling prey at the time. Also, the prey refuge and prey harvesting of susceptible prey and infected prey has been considered. The positive invariance, positivity, and boundedness of the model are investigated. The conditions for the existence of all the biologically feasible equilibrium points are established. The criterion for the local and global stability of equilibrium points in the non-delay system is examined. Further, we investigate the Hopf-bifurcation analysis for the corresponding proposed model in the presence of the fear effect. Finally, we demonstrate some numerical simulation results to illustrate our main analytical findings.

Keywords: Eco-epidemiological model, Crowley-Martin functional response, Prey refuge and harvesting, Stability, Hopf-bifurcation

[1*]Department of Mathematics, Sri Ramakrishna Mission Vidyalaya College of Arts and Science, Coimbatore, Tamilnadu, India. divyachandrakishore@rmv.ac.in

***Corresponding Author:** A. Divya
*Department of Mathematics, Sri Ramakrishna Mission Vidyalaya College of Arts and Science, Coimbatore, Tamilnadu, India. divyachandrakishore@rmv.ac.in

DOI: 10.48047/ecb/2023.12.si10.00474

1 Introduction

Eco-epidemiological models are applied to study the relationship between predator and prey, infection in a population or susceptible and diseased prey populations. Mathematical models have become an important tool for analysing disease transmission and control. Since Kermack Mckendrick's pioneering work on SIRS [12], epidemiological models have attracted much interest from researchers. Many investigators have studied the population ecology of prey, predators, or both. Ecology and epidemiology are two distinct and significant scientific fields. Predator-prey models developed by Lotka [13] and Volterra [21], are considered the first advances in modern mathematical ecology in coupled systems of non-linear differential equations. Environmental epidemiology is the combined study of ecology and epidemiology. Eco-epidemiology has a significant environmental impact. It is the study of disease transmission between interacting organisms. Various mathematical and statistical tools are available for analysing eco-epidemiological models. Many ecosystems around forests have predator-prey interactions between species, such as the deer-lion relationship. Prey and predator species abundance alters population growth and forest decline. Animal conservationists and mathematicians have long been interested in this investigation of changes in standard form. The mathematical dynamics of population growth models involving disease transmission are usually complex and non-linear. The main concern of these models is to investigate equilibrium points, their stability analysis, periodic solutions, bifurcations, chaotic behaviour, and so on.

Alfred J. Lotka was the first to investigate the relationships between populations of predators and their prey. One of the most important components of predator-prey population modelling is the mathematical model of predator-prey interactions known as "functional response". There are several types of functional responses, including Holling type I-III [7], [8]; Hassell-Varley type [6]; Beddington-DeAngelis type [1], [4]; Crowley-Martin type [3]. Crowley-Martin functional responses take both prey and predator into account. The Crowley-Martin functional response considered in this paper has the following form:

$$f(S,P) = \frac{\beta S}{(1+\eta S)(1+\mu P)},$$

where β,η,μ are non-negative numbers that represent the effect of seeking rate, processing time, and the magnitude of impact among predators, respectively. In recent decades, more information on predator-prey systems with Crowley-Martin functional responses has become available. In the recent era, some eminent authors [19], [17], [16], [5], [2] have studied to understand the importance and interactions of prey. They used some functional responses, such as Crowley Martin-type functional responses, to make the model system more realistic and controllable in the ecosystem. Many researchers have begun to study the predator-prey model with infection in either prey or predator or both populations [9]. Kadhim and Azhar [10] two forms of disease in a predator population model, with a linear functional response involving a type II Holling function. In [20], studied a non-linear analysis of a predator-prey model with discrete impacts. Global and local stability studies, including a bifurcation analysis for a ratio-dependent itraguild predation model, are discussed in [14]. Magudeeswaran et al. examined a prey-predator food web model with a type II Holling function [15]. Recently, several investigators have found a stable percentage of prey protected from predators by refuge. According to several studies and mathematical models, interactions between prey and predators can be stabilised by refugia. In [18], Maynard Smith discovered that the existence of a stable proportional refuge moderates the static behavior of the static equilibrium but not the dynamic stability of the neutrally stable Lotka-Volterra model. Tapan Kumar Kar [11] Holing type II response function is considered a predator model with integration and prey refuge. Commercial exploitation of biological resources to meet society's increasing needs is a concern for ecologists, bioeconomists, and natural resource managers. Harvesting is extensively used in fisheries, forestry, and wildlife management. These investigations revealed various and intriguing dynamics, such as equilibrium points, Hopf-bifurcation analysis, limit cycles, homoclinic loops, Bogdanov-Takens bifurcation, and even catastrophe. In eco-epidemiology, we explore predator-prey models that incorporate disease dynamics. We seek to explore the dynamics of the predator-prey model using this functional response.

In eco-epidemiology, we study predator-prey models along with disease dynamics. Several investigations have been conducted on the dynamical behaviour of Crowley-Martin ecoepidemiological models. To our knowledge, few scholars have investigated three species of prey-predator models that include species interaction, such as Crowley-Martin disease in prey populations. This work examines the

dynamics of a Crowley-Martin eco-epidemiological model involving prey refuge and prey harvesting. The rest of the paper is structured as follows: In Section 2, we present the mathematical analysis of the study. In Section 3, some preliminary aspects of the model are examined. Section 4 deals with boundary equilibrium points and their stability. In Sections 5 and 6, we determine the existence of the interior equilibrium point $E^*(u^*,v^*,w^*)$ and investigate its local and global stability. The occurrence of Hopf-bifurcation is shown in Section 7. Numerical simulations are studied for the proposed model in Section 8. The conclusion of the paper and the biological implications of our mathematical results are found in Section 8, which concludes the paper.

2 Mathematical Model Formation

The model explains the relationship between the structure of the infected prey and the following equations. The proposed framework was used to explore a non-linear prey and predator mathematical model,

$$\left.\begin{array}{rcl} \frac{dS}{dT} &=& RS(1-\frac{S+I}{K})-\frac{\alpha_1(1-\theta)SI}{a_1+(1-\theta)S}-\frac{\beta_1 SP}{(1+\eta_1 S)(1+\mu_1 P)}-H_1E_1S, \\ \frac{dI}{dT} &=& \frac{\alpha_1(1-\theta)SI}{a_1+(1-\theta)S}-D_1I-\frac{b_1IP}{(1+\eta_1 I)(1+\mu_1 P)}-H_2E_2I, \\ \frac{dP}{dT} &=& -D_2P+\frac{cb_1IP}{(1+\eta_1 I)(1+\mu_1 P)}+\frac{c\beta_1 SP}{(1+\eta_1 S)(1+\mu_1 P)}, \end{array}\right\} \quad (2.1)$$

and the positive conditions are described as $S_0 \geq 0, I_0 \geq 0$ and $P_0 \geq 0$.

The table displays the specific biological meanings of the parameters.

It is appropriate to modify the variables as follows in order to decrease the number of system Table 1: Biological representation of the model

Parameters	**Units**	**Biological representation**
S	Number per unit area (tons)	Susceptible Prey
I	Number per unit area (tons)	Prey with infection
P	Number per unit area (tons)	Predator
R	Per day (T^{-1})	Prey growth rate
K	Number per unit area (tons)	Environmental carrying capacity
α_1	Per day (T^{-1})	Infection rate
a_1	Per day (V)	Half-saturation constant
θ	Per day (V^{-1})	Refuge constant of prey
β_1	Per day (T^{-1})	Susceptible prey to predator's rate of consumption
η_1	per day	Time for handling a predator
μ_1	Per day	Interaction between predators on a large scale
$b1$	Per day (T^{-1})	Capture rate by predator
H_1	Per day	The catchability coefficient of the susceptible prey
H_2	Per day	The catchability coefficient of the infected prey
E	Per day	Harvesting effort
c	Per day	Prey to predator conversion rate
D_1	Per day (T^{-1})	Mortality rate Diseased prey
D_2	Per day (T^{-1})	Mortality rate among predator

variables $s = \frac{S}{K}, i = \frac{I}{K}, p = \frac{P}{K}$, and to consider the dimension time $t = \lambda KT$. Now, we applying the following transformations

$$r = \frac{R}{\lambda K}, \alpha = \frac{\alpha_1}{\lambda K}, a = \frac{a_1}{K}, \beta = \frac{\beta_1}{\lambda}, \eta = \eta_1 K, \mu = \mu_1 K,$$
$$h_1 = \frac{H_1E_1}{\lambda K}, b = \frac{b_1}{\lambda}, h_2 = \frac{H_2E_2}{\lambda K}, d = \frac{D_1}{\lambda K}, \delta = \frac{D_2}{\lambda K}. \quad (2.1)$$

The equation (2.1) can be expressed as non-dimensional form using the above transformations.

$$\left.\begin{array}{rcl} \frac{ds}{dt} &=& rs(1-s-i)-\frac{\alpha(1-\theta)si}{a+(1-\theta)s}-\frac{\beta sp}{(1+\eta s)(1+\mu p)}-h_1 s, s(0)\geq 0, \\ \frac{di}{dt} &=& \frac{\alpha(1-\theta)si}{a+(1-\theta)s}-di-\frac{bip}{(1+\eta i)(1+\mu p)}-h_2 i, i(0)\geq 0, \\ \frac{dp}{dt} &=& -\delta p+\frac{cbip}{(1+\eta i)(1+\mu p)}+\frac{c\beta sp}{(1+\eta s)(1+\mu p)}, p(0)\geq 0, \end{array}\right\} \quad (2.2)$$

3 Preliminaries

Here, the following preliminary properties of solutions of the proposed system are discussed.

3.1 Positivity

Theorem 3.1 *All solutions of (2.2) are positive in* $\mathbb{R}^3_+$.

Proof. Since s_0, i_0, and p_0 are all greater than or equal to zero, the system (2.2) becomes,

$$\begin{aligned} s(t) &= s(0)exp\left(\int_0^t \left[r(1-s-i) - \frac{\alpha(1-\theta)i}{a+(1-\theta)s} - \frac{\beta p}{(1+\eta s)(1+\mu p)} - h_1\right] ds\right) \geq 0 \\ i(t) &= i(0)exp\left(\int_0^t \left[\frac{\alpha(1-\theta)s}{a+(1-\theta)s} - d - \frac{bp}{(1+\eta i)(1+\mu p)} - h_2\right] di\right) \geq 0, \\ p(t) &= p(0)exp\left(\int_0^t \left[-\delta + \frac{cbi}{(1+\eta i)(1+\mu p)} + \frac{c\beta s}{(1+\eta s)(1+\mu p)}\right] dp\right) \geq 0, \end{aligned}$$

then the solution of (2.2) are non-negative.

3.2 Positive Invariance

Let $\gamma \equiv (s(t), i(t), p(t))^T$ and $U(\gamma) = (U_1(\gamma), U_2(\gamma), U_3(\gamma))^T$, where

$$\mathcal{U}_1(\gamma) = rs(1-s-i) - \frac{\alpha(1-\theta)si}{a+(1-\theta)s} - \frac{\beta sp}{(1+\eta s)(1+\mu p)} - h_1 s,$$

$$\mathcal{U}_2(\gamma) = \frac{\alpha(1-\theta)si}{a+(1-\theta)s} - di - \frac{bip}{(1+\eta i)(1+\mu p)} - h_2 i,$$

$$\mathcal{U}_3(\gamma) = -\delta p + \frac{cbip}{(1+\eta i)(1+\mu p)} + \frac{c\beta sp}{(1+\eta s)(1+\mu p)}.$$

Then, the system (2.2) can be written as $\frac{d\gamma}{dt} = U(\gamma)$ where $\mathcal{U} : C_+ \to \mathbb{R}^3_+$ with $\gamma(0) = \gamma_0 \in \mathbb{R}^3_+$.

Here, $U_n \in C^\infty(R)$ for $n = 1,2,3$. Therefore, the function U is Lipschitzian and continuous on $\mathbb{R}^3_+$. The system (2.2) has positive initial conditions, so it can be demonstrated that these solutions exist. As a result, in the region $\mathbb{R}^3_+$, (2.2) is an invariant.

3.3 Boundedness of the solutions

Theorem 3.2 *The system (2.2) solutions starting at* $\mathbb{R}^3_+$ *are all positive and bounded.*

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

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

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

Let $\phi = s + i + p$.

$$\begin{aligned} \frac{d\phi}{dt} &= rs(1-s) - rsi - \frac{(1-c)\beta sp}{(1+\eta s)(1+\mu p)} - h_1 s - di - \frac{(1-c)bip}{(1+\eta i)(1+\mu p)} - h_2 i - \delta p, \\ &\leq rs(1-s) - h_1 s - (d+h_2)i - \delta p \ (since\ c < 1), \\ &\leq \frac{\xi}{4} - h_1 s - (d+h_2)i - \delta p \ (\text{since } Max\ \{rs(1-s)\} = \frac{\xi}{4}), \\ &\leq \frac{\xi}{4} - \phi\zeta, \text{ where } \zeta = min\ \{h_1, d+h_2, \delta\}. \end{aligned}$$

Hence, we have

$$\frac{d\phi}{dt} + \phi\zeta \leq \frac{\xi}{4}.$$

The differential inequality theorem is used to determine

$$0 < \phi \leq \frac{\xi}{4\zeta}(1 - exp^{-\zeta t}) + \phi(s_0, i_0, p_0)exp^{-\zeta t}.$$

For $t \to \infty$, we have $0 < \phi \leq \frac{\xi}{4\zeta}$. Hence, each and every one of the model (2.2) are confined to non-negative initial conditions around Ω, where $\Omega = \{(s,i,p) \in \mathcal{R}^3_+ : s + i + p \leq \frac{\xi}{4\zeta} + \in\}$.

4 Existence of Equilibrium points

In this section, we explore the possible equilibrium points (2.2). The system (2.2) exhibits the following equilibrium points based on observation.

$$rs(1-s-i)-\frac{\alpha(1-\theta)si}{a+(1-\theta)s}-\frac{\beta sp}{(1+\eta s)(1+\mu p)}-h_1 s=0$$
$$\frac{\alpha(1-\theta)si}{a+(1-\theta)s}-di-\frac{bip}{(1+\eta i)(1+\mu p)}-h_2 i=0,$$
$$-\delta p+\frac{cbip}{(1+\eta i)(1+\mu p)}+\frac{c\beta sp}{(1+\eta s)(1+\mu p)}=0.$$

,

1. The trivial equilibrium point is $E_0(0,0,0)$.
2. The diseased prey free and predator-free equilibrium point $E_1(s,0,0)$ exists if $h_1 < r$, where $s=\frac{r-h_1}{r}$.
3. The predator-free equilibrium point $E_2(s,\hat{}\,\hat{}\,i,0)$, where

$\hat{s}=\frac{a(d+h_2)}{(1-\theta)(\alpha-(d+h_2))}$ and $\hat{\hat{i}}=\frac{a(r-h_1)[(1-\theta)(\alpha-(d+h_2))-a(d+h_2)]}{(1-\theta)(\alpha-(d+h_2))[ar+(1-\theta)(\alpha-(d+h_2))]}$.

E_2 exists for $d+h_2<\alpha, \theta<1, h_1<r$, $(d+h_2)((1-\theta)+a)<(1-\theta)\alpha$, and $(d+h_2)((1-\theta)<ar+(1-\theta)\alpha$.

4. The infection-free equilibrium point $E_3(\bar{s},0,\bar{p})$, where $\bar{s}=\frac{\delta(1+\mu p)}{c\beta-\delta\eta(1+\mu p)}$ and $\bar{p}=\frac{(1+\eta s)(r(1-s)-h_1)}{\beta-\mu(1+\eta s)(r(1-s)-h_1)}$.

Thus, the conditions must exist for the infection-free equilibrium point E_3 are $\frac{\delta\eta(1+\mu p)}{c}<\beta$ and $r(1-s)-h_1<\frac{\beta}{\mu(1+\eta s)}$ (assume $h_1<r(1-s)$ and $s<1$).

5. The endemic equilibrium point $E^*(s^*,i^*,p^*)$, where

$$s^*=\frac{\delta(1+\eta i^*)(1+\mu p^*)-bci^*}{bc\eta i^*+(1+\eta i^*)(c\beta-\delta\eta(1+\mu p^*))},$$
$$i^*=\frac{b(a+(1-\theta)s^*)p^*-(1+\mu p^*)[\alpha(1-\theta)s^*-(d+h_2)(a+(1-\theta)s^*)]}{\eta(1+\mu p^*)[\alpha(1-\theta)s^*-(d+h_2)(a+(1-\theta)s^*)]}$$
$$p^*=\frac{(1+\eta s^*)[r(1-s^*-i^*)-\frac{\alpha(1-\theta)i^*}{a+(1-\theta)s^*}-h_1]}{\beta-\mu(1+\eta s^*)[r(1-s^*-i^*)-\frac{\alpha(1-\theta)i^*}{a+(1-\theta)s^*}-h_1]}.$$

,

Thus, the conditions must exist for the endemic equilibrium point E^* are $\frac{\delta\eta(1+\mu p)}{c}<\beta$, $d+h_2<\frac{\alpha(1-\theta)s^*}{a+(1-\theta)s^*}$, $r(1-s^*-i^*)<\frac{\alpha(1-\theta)i^*}{a+(1-\theta)s^*}-h_1+\frac{\beta}{\mu(1+\eta s^*)}$.

5 Stability analysis

In order to determine local stability around various equilibrium points, we compute the Jacobian matrix. At each given point (s,i,p), the Jacobian matrix is given by

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

Where,

$$L_{11}=r(1-2s-i)-\frac{a\alpha(1-\theta)i}{(a+(1-\theta)s)^2}-\frac{\beta p}{(1+\eta s)^2(1+\mu p)}-h_1,$$
$$L_{12}=-s(r+\frac{\alpha(1-\theta)}{a+(1-\theta)s}), L_{13}=-\frac{\beta s}{(1+\eta s)(1+\mu p)^2},$$
$$L_{21}=\frac{\alpha a(1-\theta)i}{(a+(1-\theta)s)^2}, L_{22}=\frac{\alpha(1-\theta)s}{a+(1-\theta)s}-\frac{bp}{(1+\mu p)(1+\eta i)^2}-(d+h_2),$$
$$L_{23}=-\frac{bi}{(1+\eta i)(1+\mu p)^2}, L_{31}=\frac{\beta cp}{(1+\eta s)^2(1+\mu p)},$$
$$L_{32}=\frac{bcp}{(1+\eta i)^2(1+\mu p)}, L_{33}=-\delta+\frac{bci}{(1+\eta i)(1+\mu p)^2}+\frac{\beta cs}{(1+\eta s)(1+\mu p)^2}.$$

Theorem 5.1 *The trivial equilibrium point* $E_0(0,0,0)$ *is always unstable.*
Proof. Now, the Jacobian matrix of system (2.2) is given by

$$J(E_0) = \begin{bmatrix} r - h_1 & 0 & 0 \\ 0 & -d - h_2 & 0 \\ 0 & 0 & -\delta \end{bmatrix}$$

Therefore, eigenvalues of the characteristic equation of $J(E_0)$ are $r - h_1$, $-d - h_2$, $-\delta$. Hence, E_0 is locally asymptotically stable only if $r < h_1$ and unstable otherwise.

Theorem 5.2 *The diseased prey free and predator-free equilibrium point* $E_1(1,0,0)$ *is locally asymptotically stable if* $\alpha(1-\theta)(r-h_1) < (d+h_2)(ar+(1-\theta)(r-h_1))$ *and* $\beta c(r-h_1) < \delta(r+\eta(r-h_1))$.
Proof. The Jacobian matrix of system (2.2) at $E_1(\frac{r-h_1}{r}, 0, 0)$ is given by

$$J(E_1) = \begin{bmatrix} M_1 & M_2 & M_3 \\ 0 & M_4 & 0 \\ 0 & 0 & M_5 \end{bmatrix}$$

Where,

$$M_1 = -r + h_1, M_2 = -s[r + \frac{\alpha(1-\theta)}{a+(1-\theta)s}], M_3 = -\frac{\beta s}{1+\eta s},$$
$$M_4 = \frac{\alpha(1-\theta)(r-h_1)}{ar+(1-\theta)(r-h_1)} - d - h_2, M_5 = -\delta + c\beta[\frac{r-h_1}{r+\eta(r-h_1)}].$$

Therefore, eigenvalues of the characteristic equation of $J(E_1)$ are $h_1 - r$, $\frac{\alpha(1-\theta)(r-h_1)}{ar+(1-\theta)(r-h_1)} - (d+h_2)$, and $-\delta + c\beta[\frac{r-h_1}{r+\eta(r-h_1)}]$. If $\lambda_1 < 0$ i.e., $h_1 < r$, $\lambda_2 < 0$, i.e., $\alpha(1-\theta)(r-h_1) < (d+h_2)(ar+(1-\theta)(r-h_1))$ and $\lambda_3 < 0$, i.e., $\beta c(r-h_1) < \delta(r+\eta(r-h_1))$. Thus E_1 is locally asymptotically stable if $\alpha(1-\theta)(r-h_1) < (d+h_2)(ar+(1-\theta)(r-h_1))$ and $\beta c(r-h_1) < \delta(r+\eta(r-h_1))$. □

Theorem 5.3 *The predator-free equilibrium point* $E_2(s,\hat{i},0)$ *is locally asymptotically stable if* $X_{11} > 0$, $X_{12} > 0$ *and* $\delta > \frac{bc\hat{i}}{1+\eta\hat{i}} + \frac{\beta c\hat{s}}{1+\eta\hat{s}}$.
Proof. The Jacobian matrix of system (2.2) is given by

$$J(E_2) = \begin{bmatrix} N_1 & N_2 & N_3 \\ N_4 & N_5 & N_6 \\ 0 & 0 & N_7 \end{bmatrix}$$

Where,

$$N_1 = r(1-2\hat{s}-\hat{i}) - \frac{a\alpha(1-\theta)\hat{i}}{(a+(1-\theta)\hat{s})^2} - h_1, N_2 = -\hat{s}[r + \frac{\alpha(1-\theta)}{a+(1-\theta)\hat{s}}],$$
$$N_3 = -\frac{\beta\hat{s}}{1+\eta\hat{s}}, N_4 = \frac{\alpha a(1-\theta)\hat{i}}{(a+(1-\theta)\hat{s})^2}, N_5 = \frac{\alpha(1-\theta)\hat{s}}{a+(1-\theta)\hat{s}} - (d+h_2)$$
$$N_6 = -\frac{b\hat{i}}{1+\eta\hat{i}}, N_7 = -\delta + \frac{bc\hat{i}}{1+\eta\hat{i}} + \frac{\beta c\hat{s}}{1+\eta\hat{s}}.$$

Therefore, the characteristic equation of $J(E_2)$ is $(N_7-\lambda)(\lambda^2 + X_{11}\lambda + X_{12}) = 0$, where $X_{11} = -(N_1+N_5)$ and $X_{12} = N_1N_5 - N_2N_4$.

In the above characteristic equation, we get one of the eigenvalue is N_7, which is negative as $\delta > \frac{bc\hat{i}}{1+\eta\hat{i}} + \frac{\beta c\hat{s}}{1+\eta\hat{s}}$ and the other two eigenvalues should be negative if $X_{11} > 0$ and $X_{12} > 0$.

Hence, E_2 is locally asymptotically stable if $X_{11} > 0$, $X_{12} > 0$ and $\delta > \frac{bc\hat{i}}{1+\eta\hat{i}} + \frac{\beta c\hat{s}}{1+\eta\hat{s}}$.

Theorem 5.4 *The equilibrium point E_3 is locally asymptotically stable if $Y_{11} > 0$, $Y_{12} > 0$ and* $d + h_2 > \frac{\alpha(1-\theta)\bar{s}}{a+(1-\theta)\bar{s}} - \frac{b\bar{p}}{1+\mu\bar{p}}$.

Proof. The Jacobian matrix of system (2.2) at $E_3(\bar{s},0,\bar{p})$ is given by,

$$J(E_3) = \begin{bmatrix} P_1 & P_2 & P_3 \\ 0 & P_4 & 0 \\ P_5 & P_6 & P_7 \end{bmatrix}$$

Where,

$$P_1 = r(1-2\bar{s}) - \frac{\beta\bar{p}}{(1+\eta\bar{s})^2(1+\mu\bar{p})} - h_1, P_2 = -\bar{s}[r + \frac{\alpha(1-\theta)}{a+(1-\theta)\bar{s}}],$$
$$P_3 = -\frac{\beta\bar{s}}{(1+\eta\bar{s})(1+\mu\bar{p})^2}, P_4 = \frac{\alpha(1-\theta)\bar{s}}{a+(1-\theta)\bar{s}} - \frac{b\bar{p}}{1+\mu\bar{p}} - (d+h_2), P_5 = \frac{\beta c\bar{p}}{(1+\eta\bar{s})^2(1+\mu\bar{p})}$$
$$P_6 = -\frac{bc\bar{p}}{1+\mu\bar{p}}, P_7 = -\delta + \frac{\beta c\bar{s}}{(1+\eta\bar{s})(1+\mu\bar{p})^2}.$$

Now, the characteristic equation for $J(E_3)$ is $(P_4-\lambda)(\lambda^2+Y_{11}\lambda+Y_{12}) = 0$, where $Y_{11} = -(P_1+P_7)$ and $Y12 = P1P7 - P3P5$.

In the above characteristic equation, we get one of the eigenvalue is P_4, which is negative as $d+h_2 > \frac{\alpha(1-\theta)\bar{s}}{a+(1-\theta)\bar{s}} - \frac{b\bar{p}}{1+\mu\bar{p}}$ and the other two eigenvalues should be negative if $Y_{11} > 0$ and $Y_{12} > 0$.

So, the infection-free equilibrium point $E_3(\bar{s},0,\bar{p})$ is locally asymptotically stable if $d + h_2 > \frac{\alpha(1-\theta)\bar{s}}{a+(1-\theta)\bar{s}} - \frac{b\bar{p}}{1+\mu\bar{p}}$, $Y_{11} > 0$, $Y_{12} > 0$, otherwise the system (2.2) will be unstable.

Theorem 5.5 *The equilibrium point E^* is locally asymptotically stable if $Z_1 > 0$, $Z_2 > 0$, and $Z_1Z_2 - Z_3 > 0$.*

Proof. Now, the Jacobian matrix of system (2.2) at $E^*(s^*,i^*,P^*)$ is given by

$$J(E^*) = \begin{bmatrix} Q_{11} & Q_{12} & Q_{13} \\ Q_{21} & Q_{22} & Q_{23} \\ Q_{31} & Q_{32} & Q_{33} \end{bmatrix}$$

Where,

$$Q_{11} = -rs^* + \frac{\alpha(1-\theta)^2 s^* i^*}{(a+(1-\theta)s^*)^2} + \frac{\eta\beta s^* p^*}{(1+\eta s^*)^2(1+\mu p^*)},$$
$$Q_{12} = -s^*[r + \frac{\alpha(1-\theta)}{a+(1-\theta)\bar{s}}], Q_{13} = -\frac{\beta s^*}{(1+\eta s^*)(1+\mu p^*)^2},$$
$$Q_{21} = \frac{\alpha a(1-\theta)i^*}{(a+(1-\theta)s^*)^2}, Q_{22} = \frac{\eta b i^* p^*}{(1+\eta i^*)^2(1+\mu p^*)}, Q_{23} = -\frac{bi^*}{(1+\eta i^*)(1+\mu p^*)^2}$$
$$Q_{31} = \frac{\beta c p^*}{(1+\eta s^*)^2(1+\mu p^*)}, Q_{32} = \frac{bcp^*}{(1+\eta i^*)^2(1+\mu p^*)},$$
$$Q_{33} = -\frac{\mu bci^* p^*}{(1+\eta i^*)(1+\mu p^*)^2} - \frac{\mu\beta cs^* p^*}{(1+\eta s^*)(1+\mu p^*)^2}.$$

At the endemic equilibrium point E^*, the Jacobian matrix's characteristic equation is

$$\lambda^3 + Z_1\lambda^2 + Z_2\lambda + Z_3 = 0. \quad (5.1)$$

Where,

$Z1 = -(Q11 + Q22 + Q33)$,
$Z2 = -(Q12Q21 + Q13Q31 + Q23Q32 - Q11Q22 - Q11Q33 - Q22Q33)$,
$Z3 = -(Q11Q22Q33 + Q12Q23Q31 + Q13Q21Q32 - Q13Q31Q22 - Q12Q21Q33 - Q11Q23Q32)$.

According to Routh-Hurwitz criterion, Z_1, Z_2, and $Z_1Z_2 - Z_3$ must all be positive, the characteristic of all the roots be negative. Hence, E^* is locally asymptotically stable.

6 Global stability analysis

Here, we study the global stability of the model (2.2) around the endemic equilibrium $E^*(s^*,i^*,p^*)$. A function of Lyapunov form

$$L_1(s,i,p) = (s - s^* - s^* ln\frac{s}{s^*}) + L_2(i - i^* - i^* ln\frac{i}{i^*}) + L_3(p - p^* - p^* ln\frac{p}{p^*}),$$

where L_2,L_3 are positive constants.

Here, $L_1(s,i,p) \geq 0$ since $\psi - 1 \geq ln\ \psi$ for $\psi > 0$ and $L_1(s^*,i^*,p^*) = 0$. Differentiating L_1 with respect to t, we obtain

$$\begin{aligned}\frac{dL_1}{dt} =&(\frac{s-s^*}{s})\frac{ds}{dt} + L_2(\frac{i-i^*}{i})\frac{di}{dt} + L_3(\frac{p-p^*}{p})\frac{dp}{dt}\\ =&[r(1-s-i) - \frac{\alpha(1-\theta)i}{a+(1-\theta)s} - \frac{\beta p}{(1+\eta s)(1+\mu p)} - h_1](s-s^*)\\ &+ L_2[\frac{\alpha(1-\theta)s}{a+(1-\theta)s} - d - \frac{bp}{(1+\eta i)(1+\mu p)} - h_2](i-i^*)\\ &+ L_3[-\delta + \frac{cbi}{(1+\eta i)(1+\mu p)} + \frac{c\beta s}{(1+\eta s)(1+\mu p)}](p-p^*).\end{aligned}$$

After some simplifications we get,

$$\begin{aligned}\frac{dL_1}{dt} =&-(s-s^*)[r((s+i)-(s^*+i^*)) + \alpha(\frac{(1-\theta)i}{a+(1-\theta)s} - \frac{(1-\theta)i^*}{a+(1-\theta)s^*})\\ &+ \beta(\frac{p}{(1+\eta s)(1+\mu p)} - \frac{p^*}{(1+\eta s^*)(1+\mu p^*)})]\\ &- L_2(i-i^*)[b(\frac{p}{(1+\eta i)(1+\mu p)} - \frac{p^*}{(1+\eta i^*)(1+\mu p^*)})\\ &- \alpha(\frac{(1-\theta)s}{a+(1-\theta)s} - \frac{(1-\theta)s^*}{a+(1-\theta)s^*})]\\ &- L_3(p-p^*)c[b(\frac{\eta\mu(i^*p - i^*p^*) - \mu(ip^* - i^*p) - (i-i^*)}{(1+\eta i)(1+\mu p)(1+\eta i^*)(1+\mu p^*)})\\ &+ \beta(\frac{\eta\mu(s^*p - s^*p^*) - \mu(sp^* - s^*p) - (s-s^*)}{(1+\eta s)(1+\mu p)(1+\eta s^*)(1+\mu p^*)})].\end{aligned}$$

Now, we see that $\frac{dL_1}{dt}$ is negative definite in the region:

$G = \{(s,i,p) : s > s^*,i > i^*$ and $p > p^*)$ or $s < s^*,i < i^*$ and $p < p^*\}$ and Consequently, for all solutions in G, L is a Lyapunov function. Summarising our previous discussions, we arrive at the following conclusion:

Theorem 6.1 *If E^* is globally asymptotically stable then E^* is globally asymptotically stable in $G = \{(s,i,p) : s > s^*,i > i^*$ and $p > p^*)$ or $s < s^*,i < i^*$ and $p < p^*\}$.*

7 Hopf-bifurcation analysis

Theorem 7.1 *If the critical value for the bifurcation parameter h_1 is exceeded, the model (2.2) experiences the Hopf-bifurcation. The existence of the following*

Hopf-bifurcation criteria at $h_1 = h_1^*$,

1. $\mathcal{U}(h_1^*)\mathcal{V}(h_1^*) - \mathcal{W}(h_1^*) = 0$,
2. $\frac{d}{dh_1}(Re(\lambda(h_1)))|_{h_1=h_1^*} \neq 0$, *where λ is the zero of the characteristic equation corresponds to the non-negative equilibrium point.*

Proof. For $h_1 = h_1^*$, let the characteristic equation (5.1) implies that

$$(\lambda^2(h_1^*) + \mathcal{V}(h_1^*))(\lambda(h_1^*) + \mathcal{U}(h_1^*)) = 0. \tag{7.1}$$

$\implies \pm i\sqrt{\mathcal{V}(h_1^*)}$ and $-\mathcal{U}(h_1^*)$ be the zeros of the above equation (7.1). The following transversality requirement must be satisfied in order to achieve the Hopf-bifurcation at $h_1^* = h_1$.

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

For all h_1, the general roots of the form
$\lambda_1(h_1) = r(h_1) + is(h_1)$, $\lambda_2(h_1) = r(h_1) - is(h_1)$, and $\lambda_3(h_1) = -A_1(h_1)$. Now, we check the condition $\frac{d}{dh_1}(Re(\lambda_j(h_1)))|_{h_1=h_1^*} \neq 0, j = 1, 2$.

Let, $\lambda_1(h_1)= r(h_1) + is(h_1)$ in (7.1), we get
$\zeta_1(h_1) + i\zeta_2(h_1) = 0$,

where, $\zeta_1(h_1) = r^3(h_1) + r^2(h_1)U(h_1) - 3r(h_1)s^2(h_1) - s^2(h_1)U(h_1) + r(h_1)V(h_1) + U(h_1)V(h_1)$, $\zeta_2(h_1) = 3r^2(h_1)s(h_1) + 2r(h_1)s(h_1)U(h_1) - s^3(h_1) + s(h_1)V(h_1)$.

$$\frac{d\zeta_1}{dh_1} = \phi_1(h_1)r'(h_1) - \phi_2(h_1)s'(h_1) + \phi_3(h_1) = 0, \quad (7.2)$$

$$\frac{d\zeta_2}{dh_1} = \phi_2(h_1)r'(h_1) + \phi_1(h_1)s'(h_1) + \phi_4(h_1) = 0, \quad (7.3)$$

where,
$\phi_1(h_1) = 3r^2(h_1) + 2r(h_1)U(h_1) - 3s^2(h_1) + V(h_1)$, $\phi_2(h_1) = 6r(h_1)s(h_1) + 2s(h_1)U(h_1)$, $\phi_3(h_1) = r^2(h_1)U'(h_1) - s^2(h_1)U'(h_1) + W'(h_1) + V'(h_1)r(h_1)$,
$\phi_4(h_1) = 2r(h_1)s(h_1)U'(h_1) + s(h_1)V'(h_1)$.
On multiplying (7.2) and (7.3) by $\phi_1(h_1)$ and $\phi_2(h_1)$ respectively,

$$r'(h_1) = -\frac{\phi_1(h_1)\phi_3(h_1)+\phi_2(h_1)\phi_4(h_1)}{\phi_1^2(h_1)+\phi_2^2(h_1)}. \quad (7.4)$$

Substituting $r(h_1) = 0$ and $s(h_1) = \sqrt{\mathcal{V}(h_1)}$ at $h_1 = h_1^*$ on $\phi_1(h_1), \phi_2(h_1), \phi_3(h_1)$ and $\phi_4(h_1)$ we obtain

$$\phi_1(h_1^*) = -2\mathcal{V}(h_1^*), \phi_2(h_1^*) = 2\sqrt{\mathcal{V}(h_1^*)}\mathcal{U}(h_1^*),$$

$$\phi_3(h_1^*) = -\mathcal{V}(h_1^*)\mathcal{U}'(h_1^*) + \mathcal{W}'(h_1^*), \phi_4(h_1^*) = \sqrt{\mathcal{V}(h_1^*)}\mathcal{V}'(h_1^*).$$

The equation (7.4), implies

$$r'(h_1^*) = \frac{\mathcal{W}'(h_1^*)-(\mathcal{U}(h_1^*)\mathcal{V}'(h_1^*)+\mathcal{V}(h_1^*)\mathcal{U}'(h_1^*))}{2(\mathcal{V}^2(h_1^*)+\mathcal{U}^2(h_1^*))}, \quad (7.5)$$

If $\mathcal{W}'(h_1^*) - (\mathcal{U}(h_1^*)\mathcal{V}'(h_1^*) + \mathcal{V}(h_1^*)\mathcal{U}'h_1^*)) \neq 0$, which implies that
$\frac{d}{dh_1}(Re(\lambda_j(h_1)))|_{h_1=h_1^*} = r'(h_1^*) \neq 0. j = 1, 2$,
and
$\lambda_3(h_1^*) = -\mathcal{U}(h_1^*) \neq 0$.
If $\mathcal{W}'(h_1^*) - (\mathcal{U}(h_1^*)\mathcal{V}'(h_1^*) + \mathcal{V}(h_1^*)\mathcal{U}'(h_1^*)) \neq 0$, is ensured if the transversality criterion holds, and at this point, the model (2.2) enters the Hopf-bifurcation at $h_1 = h_1^*$. □

8 Numerical Analysis

We show some numerical simulations of the model (2.2) in this section. To accomplish this, we use Diethelm et al.'s predictor-corrector approach to solve the proposed model. The rate of harvesting h_1, and refuge coefficient (θ) are the main characteristics applied as control parameters in this study. Since no field data is available, the simulations are carried out with the following assumed parameter values:

Table 2: The system (2.2) parametric values

Parameters	Numerical Value	Parameters	Numerical Value
r	2	α	0.7
a	0.6	β	0.2
η	0.1	μ	0.1
d	0.1	b	0.55
h_2	0.1	δ	0.1
c	0.5		
θ	variable	h_1	variable

8.1 Effect of changing the harvesting rate h_1

Let us fix the parameter values in Table 2 with $\theta = 0.2$ and $h_1 = 0.2$. The positive equilibrium point $E^*(0.7331,0.119554,0.230971)$ exists for $0.01 < h_1 < 0.3$, respectively. Figure (1) illustrates the stability of E^* for $h_1 = 0.08$, while Figure (2) illustrates the stability of E^* for $h_1 = 0.2$.

Figure 3(a), Figure 3(b), and Figure 3(c) show that increasing the harvesting rate of susceptible prey leads to a decrease in the population of vulnerable prey and predators while increasing the population of diseased prey.

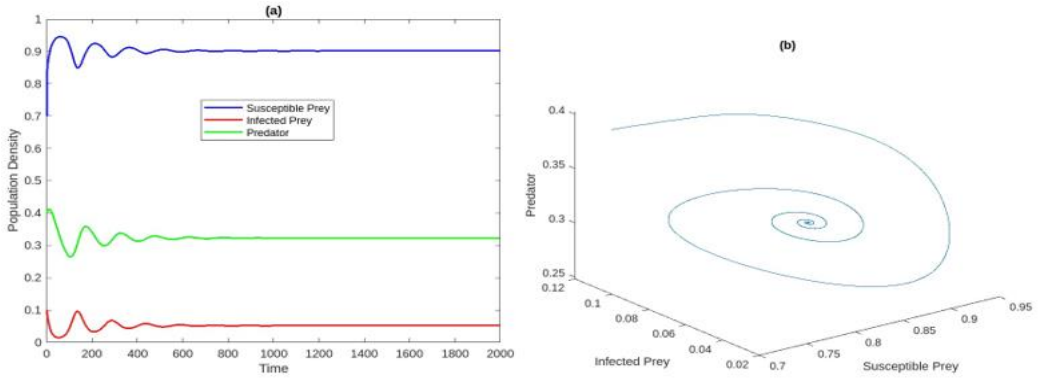

Figure 1: Time analysis for the system (2.2) of susceptible prey, infected prey, and phase portrait for ($\theta = 0.2$, and $h_1 = 0.08$.)

8.2 Effect of changing the refuge constant θ

Let us fix the parameter values in Table 2 with $h_1 = 0.2$. The positive equilibrium point $E^*(0.17331,0.119554,0.230971)$ exists for $0.1 < \theta < 0.5$, respectively.

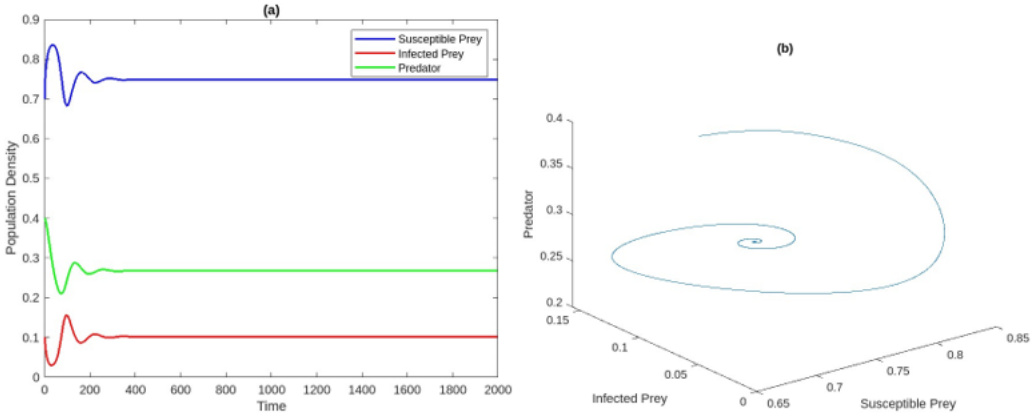

Figure 2: Time analysis of susceptible prey, infected prey and phase portrait for the system (2.2) when ($\theta = 0.2$, and $h_1 = 0.2$.)

From Figure 3, it shows that increasing the refuge rate of vulnerable prey leads to an increase in the population of susceptible prey while decreasing the population of diseased prey.

9 Conclusion

In this study, we investigated the refuge and harvesting rates in a Crowley-Martin eco-epidemiological model with infection in a population of prey where a predator attacked susceptible and infected prey. Local stability (2.2) is applied to each set of biologically possible equilibrium points of the system. It is used to modify the refuge rate (θ) and the harvesting rate (h_1) as control parameters. In addition, we investigated the local stability of the proposed model (2.2) and studied the Hopf-bifurcation phenomenon. As a result, we found that modifying the harvesting rate h_1 significantly affects the stability of the proposed model (2.2). The analytical and numerical findings demonstrate that refuge coefficient and harvesting rate have a significant effect on each population. The susceptible prey density increases as the refuge from infection decreases, whereas the infected prey density decreases. A decrease in the population of susceptible prey and an increase in infected prey population density are the effects of increasing the harvesting rate. This study shows the complex behavior of the proposed model.

References

1. John R Beddington. Mutual interference between parasites or predators and its effect on searching efficiency. *The Journal of Animal Ecology*, pages 331–340, 1975.

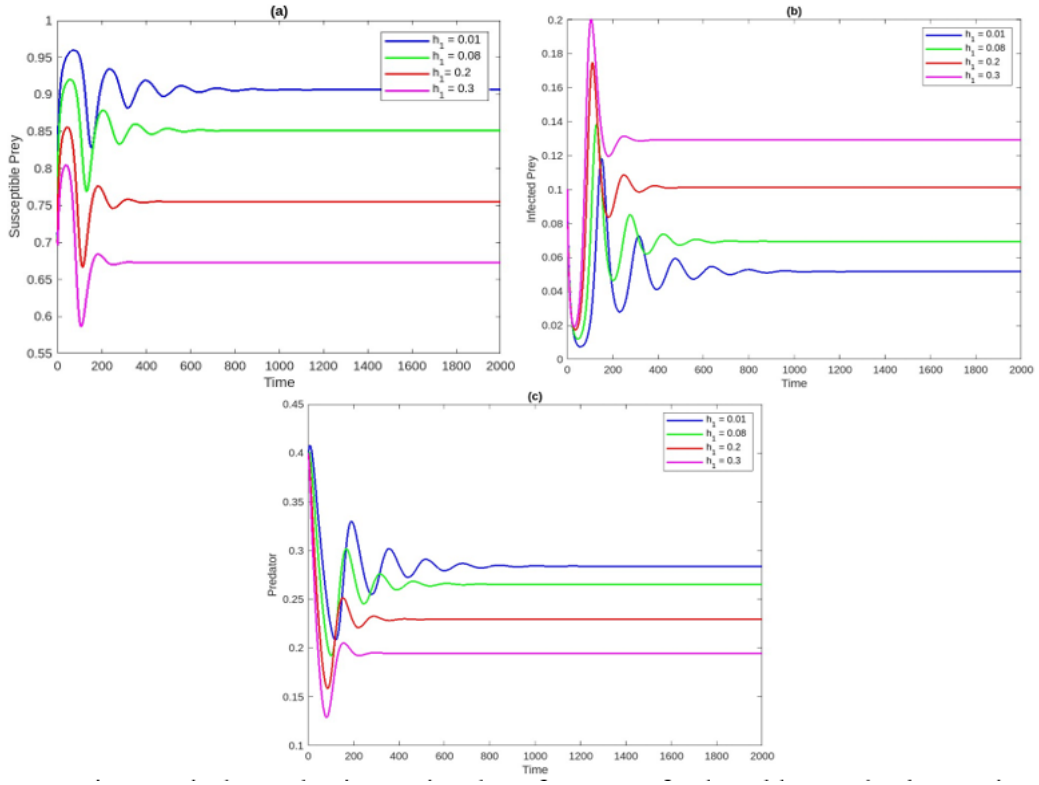

Figure 3: Except for ($\theta = 0.2$) in Table 2, the concentrations of susceptible prey, diseased prey, and predators are as follows for the parameter values shown in Table 2. Where h_1 =0.01,0.08,0.2,0.3.

2. Xun Cao and Weihua Jiang. Turing–hopf bifurcation and spatiotemporal patterns in a diffusive predator–prey system with crowley–martin functional response. *Nonlinear Analysis: Real World Applications*, 43:428–450, 2018.
3. Philip H Crowley and Elizabeth K Martin. Functional responses and interference within and between year classes of a dragonfly population. *Journal of the North American Benthological Society*, 8(3):211–221, 1989.
4. Donald Lee DeAngelis, RA Goldstein, and Robert V O'Neill. A model for tropic interaction. *Ecology*, 56(4):881–892, 1975.
5. Balram Dubey, Shikhar Agarwal, and Ankit Kumar. Optimal harvesting policy of a prey–predator model with crowley–martin-type functional response and stage structure in the predator. *Nonlinear Analysis: Modelling and Control*, 23(4):493–514, 2018.
6. MP Hassell and GC Varley. New inductive population model for insect parasites and its bearing on biological control. *Nature*, 223:1133–1137, 1969.

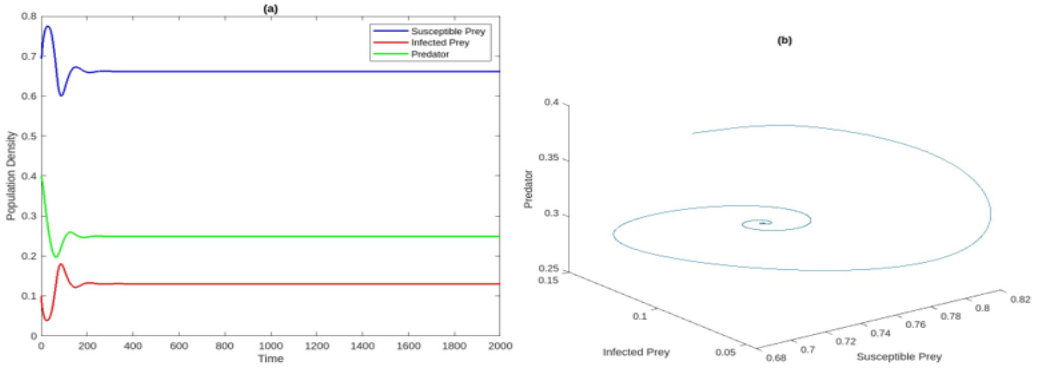

Figure 4: Time analysis for the system (2.2) of susceptible prey, infected prey and phase portrait for (h_1 = 0.2, and $\theta = 0.1$.)

7. Crawford S Holling. The components of predation as revealed by a study of small-mammal predation of the european pine sawfly1. *The Canadian Entomologist*, 91(5):293–320, 1959.
8. Crawford S Holling. Some characteristics of simple types of predation and parasitism1. *The canadian entomologist*, 91(7):385–398, 1959.
9. Wassan Hussein and Huda Abdul Satar. The dynamics of a prey-predator model with infectious disease in prey: Role of media coverage. *Iraqi Journal of Science*, 62(12):4930– 4952, 2021.
10. Atheer Jawad Kadhim and Azhar A Majeed. Epidemiological model involving two diseases in predator population with holling type-ii functional response. *International Journal of Nonlinear Analysis and Applications*, 12(2):2085–2107, 2021.
11. Tapan Kumar Kar. Stability analysis of a prey–predator model incorporating a prey refuge. *Communications in Nonlinear Science and Numerical Simulation*, 10(6):681–691, 2005.
12. William Ogilvy Kermack and Anderson G McKendrick. A contribution to the mathematical theory of epidemics. *Proceedings of the royal society of london. Series A, Containing papers of a mathematical and physical character*, 115(772):700–721, 1927.
13. Alfred James Lotka. *Elements of physical biology*. Williams & Wilkins, 1925.
14. S Magudeeswaran, K Sathiyanathan, R Sivasamy, S Vinoth, M Sivabalan, et al. Analysis on dynamics of delayed intraguild predation model with ratio-dependent functional response. *Discontinuity, Nonlinearity, and Complexity*, 10(03):381–396, 2021.

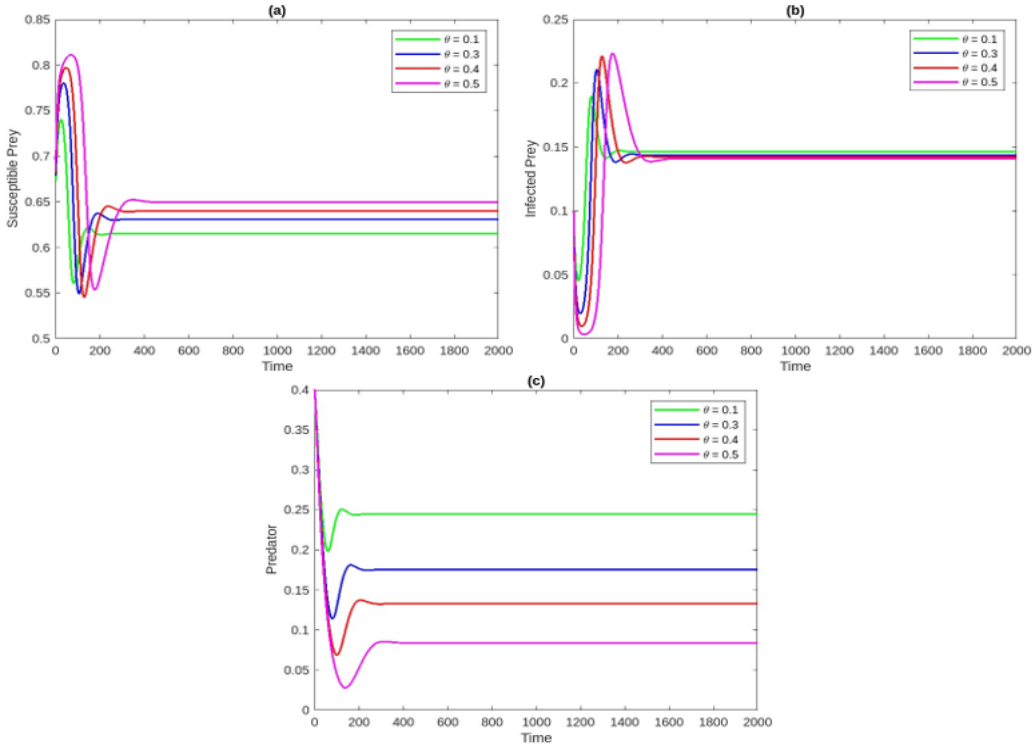

Figure 5: Except for ($h_1 = 0.2$) in Table 2, the concentrations of susceptible prey, diseased prey, and predators are as follows for the parameter values shown in Table 2, . Where $\theta = 0.1, 0.3, 0.4, 0.5$.

15. S Magudeeswaran, S Vinoth, K Sathiyanathan, and M Sivabalan. Impact of fear on delayed three species food-web model with holling type-ii functional response. *International Journal of Biomathematics*, 15(04):2250014, 2022.
16. Atasi Patra Maiti, Balram Dubey, and A Chakraborty. Global analysis of a delayed stage structure prey–predator model with crowley–martin type functional response. *Mathematics and Computers in Simulation*, 162:58–84, 2019.
17. Atasi Patra Maiti, Chandan Jana, and Dilip Kumar Maiti. A delayed eco-epidemiological model with nonlinear incidence rate and crowley–martin functional response for infected prey and predator. *Nonlinear Dynamics*, 98:1137–1167, 2019.
18. J Smith. Maynard: Models in ecology, 1974.
19. Vandana Tiwari, Jai Prakash Tripathi, Syed Abbas, Jin-Shan Wang, Gui-Quan Sun, and

Zhen Jin. Qualitative analysis of a diffusive crowley–martin predator–prey model: the role of nonlinear predator harvesting. *Nonlinear Dynamics*, 98:1169–1189, 2019.

20. S Vinoth, R Sivasamy, K Sathiyanathan, Grienggrai Rajchakit, P Hammachukiattikul, R Vadivel, and Nallappan Gunasekaran. Dynamical analysis of a delayed food chain model with additive allee effect. *Advances in Difference Equations*, 2021:1–20, 2021.
21. V Volterra. Variazioni e fluttuazioni del numero d'individui in specie animali conviventi mem. accad. lincei roma 2 31; fluctuations in the abundance of a species considered mathematically. *Nature (London)*, 118:558, 1926.