Qualitative Analysis of some Ecological and Eco-epidemiological Systems

Thesis

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Doctor of Philosophy

By

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CERTIFICATE FROM THE SUPERVISOR(S)

This is to certify that, the thesis entitled " Qualitative Analysis of some Ecological and Eco-epidemiological Systems" submitted by Sri Chandan Maji who got his name registered on 10.07.2013 (Index No.- 145/13/Maths./22) for the award of Ph.D. (Science) degree of Jadavpur University, is absolutely based upon his own work under the supervision of Professor (Dr.) Dipak Kesh and Dr. Debasis Mukherjee and that neither this thesis nor any part of it has been submitted for either any degree/diploma or any other academic award anywhere before.

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To
my daughter Mehuli,
parents and grand parents

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1 Introduction

1.1 History of ecology and eco-epidemiology

Mathematical biology is a branch of biology that uses mathematical models to investigate the principles that govern the structure, development and behaviour of the systems. This is a very fast growing subject in applied mathematics and it has a wide range of applications in the study of various biological systems. Ecology and epidemiology are two most interesting field of studies of this part. Although these two fields are related to different branches but they have some similarities and the combinations of these two fields are popularly known as eco-epidemiology which shows the disease as a result of environmental interaction of host and parasite. Disease have the capacity to not only influence the dynamics of their hosts, but also interacting species like predators, prey and their competitors. Likewise, interacting species can also influence disease dynamics by altering the hosts dynamics. In 1920, Lotka and Volterra [114] first proposed a model which is based on a pair of differential equation that describes the prey-predator interaction and is known as Lotka-Volterra model. At the same time the classic

Kermack and McKendrick [93] SIR epidemic model for transmission of infectious disease has drawn much more attention among the epidemiologists. Later on, Anderson and May [6] was the pioneer who merged these two interesting fields and developed a predator-prey model with an infected prey population. Mathematical modelling in ecology, epidemiology is a interesting field in recent times and it is a systematic methodology which is powerful as well as successful in discovery and better understanding for the underline process. Usually mathematical models describe a relation between a set of equations and variables in a system.

In 1989, Hadeler and Freedman [61] introduced the eco-epidemic model. In their paper they considered a Rosenzweig-MacArthur predator-prey system with Holling type two functional response and where the disease infects both the predators and the prey population. After that, Chattopadhyay and Arino [25] used the term eco-epidemiology and formulated a predator-prey system where prey population is infected by a parasite. Subsequently, many researchers have conducted research work on this topic [172, 173, 134, 183, 76]. As modelling is one of the finest tool for prediction of the status of the ecosystem, thus in this thesis, some ecological and epidemiological models has been formulated and discussed. These models include predator-density dependent transmission rate, disease in prey population (CWD), refuge and Leslie-Gower prey-predator interaction in presence of competitor for prey. Now we will discuss the following issues which are related to our work.

1.2 Prey refuge

The study of prey refuge in the dynamics of predator–prey interaction has become a popular subject in applied mathematics and ecology [65, 64, 122, 67, 124]. According to our knowledge, one of the founders of population ecology, Gause et al. [57] first introduced the prey-predator model with the refuge region. Later on Krivan [98] reconsidered the Gause's model and described the model which was ill posed. Many researchers [138, 98, 160, 72, 97, 130, 60, 77, 33, 137, 139] have discussed the influence of prey refuge and they concluded that the prey refuge has a stabilizing effect on the predator-prey interaction and also prey species can be protected from extinction by using such type of policy. Ruxton [155] proposed a continuous-time predator-prey model by assuming that the prey

refuge is proportional to the predator density and he concluded that the hiding behaviour of prey has a stabilizing effect. Mainly there are two types of refuges, one is constant proportion of refuge and the other one is constant number of prey refuge. Due to fixed proportion of refuge the death rate does not increase with population density and so the presence of refuge does not cause any negative feedback effect, a necessary conditions for stabilization [16]. But it can enhance the persistence of the populations. On the other hand, in the case of constant number of prey refuge, the prey mortality rate increases with population density once the capacity of the refuge and this will cause negative feedback stabilizing effect. In fact, the effect of prey refuges on the population dynamics are very complex in nature, but for modelling purposes, it can be considered as constituted by two components [60]: the first effect, 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 trade-offs and by-products of the hiding behaviour of prey which could be advantageous or detrimental for all the interacting populations [72]. A classic secondary effect is the reduction in the birth rate of prey population, because refuges are safe but rarely offer feeding or mating opportunities.

1.3 Chronic wasting disease

Chronic wasting disease (CWD) is a fatal infectious prion disease among the deer family [181]. It was first identified as a clinical disease in captive mule deer in 1967 in a wildlife research facility in northern Colorado, USA and Southeastern Wyoming [159]. In 1980, the disease was determined to be the family of transmissible spongiform encephalopathies (TSEs) along with other well-known TSEs like transmissible mink encephalopathy (TME) in Mink, bovine spongiform encephalopathy (BSE) or "mad-cow disease" and both Creutzfeldt-Jakob disease (CJD) and variant Creutzfeldt-Jakob disease (vCJD) in humans. CWD is observed in many species of cervids, like white-tailed deer (*Odocoileus virginianus*), black-tailed deer (*Odocoileus columbianus*), mule deer (*Odocoileus hemionus*), Rocky Mountain elk (*Cervus elaphus nelsoni*) and Shira's moose (*Alces alces shirasi*) [159, 59]. In 1996 Chronic wasting disease was found for the first time outside Colorado and Wyoming, CWD endemic zone in a captive elk farm in

Saskatchewan [181]. CWD was also found in free-ranging deer in the Southwestern corner of Nebraska in May 2001. Infectious agents of CWD are neither bacteria nor viruses but are hypothesised by prions. CWD is transmitted through direct, animal to animal contact, vertically, from mother to offspring and indirectly, via environmental contamination [119, 125]. The disease has a long incubation period (12-34 months [94]) and it causes a spongy degeneration in the brain of infected animals, resulting in emaciation, abnormal behavior, loss of bodily functions, and ultimately death [59]. Clinical signs (e.g. staggering, lowered head/ears, lack of fear, drooling) only appear in the late stages of this disease. As the disease persist for a long time and cannot be prevented by vaccines or any other ways so the disease bears this term chronic. Furthermore, in this disease there is a gradual loss of weight, increased thirst and urination, excessive salivation, trouble walking and hence the disease is wasting. These two facts justify the particular name CWD of this disease.

1.4 Fractional-order derivative

In applied mathematics, a fractional-derivative is a derivative of any arbitrary order which may be real or complex. In 1695, it was first appeared in a letter written by Leibniz to Hospital [103]. The idea of fractional-order differentiation and integration was first introduced in one of Niels Henrik Abel's papers [2]. In 1832, this subject was independently developed by Lioville in his paper [112]. The k-th derivative of a function $f(x)$ at any point x is its local property when k is an integer but for non-integer case the derivative of $f(x)$ at $x = k$ depends on all values of f, even those far away from k. The Riemann-Liouville fractional integral operator [147] of order α of any function $f \in L_1[0, a],\ t \in [0, a]$ is presented as

$$J^{\alpha} f(t) = \frac{1}{\Gamma(\alpha)} \int_0^a (t - s)^{\alpha - 1} f(s) ds,$$

where $\Gamma(.)$ is the Gamma function. Due to progress of fractional calculus many researchers in different fields such as biology, physics, engineering, finance , medicine considered fractional calculus to develop their problems [32, 48, 152, 38, 47, 115, 116, 7, 51]. Qualitative analysis of fractional order system is much complicated rather than classical integer order system as fractional order derivatives are non

local and have weakly singular kernels but the main advantage of considering such system is that they admit greater degree of freedom in the model. Moreover, it is more realistic than integer order in biological modelling due to memory effects. Several studies are carried out numerically in fractional order system but few authors obtained some interesting results. Stability of fractional order nonlinear system is investigated in Li et al. [107]. The theory of Lyapunov direct method is further developed by Delavari et al. [39] with the help of Caputo type fractional order nonlinear system. Javidi and Nyamaradi [84] studied the dynamical behaviour of the fractional order predator-prey model and described the local stability of the system. Rihan et al. [153] developed a fractional order predator-prey system with Holling type II fractional response and time delay and they discussed local stability as well as global stability of steady states and Hopf bifurcation with respect to the delay parameter. Recently global stability analysis is discussed elaborately in Vargas-De-Leon [170]. Xu et al. analyzes the chaos synchronization between two different fractional order chaotic system by using active control [185].

1.5 Persistence

Persistence and permanence are two important properties of dynamical systems that describe the long time survival of all population in the future without depending on the initial populations but depends on solution behaviour near the boundaries. In ecological modelling, a system of ordinary differential equation has been used in the study of dynamical behaviour of entities and there are variety of analytical techniques for the investigation of the dynamical characteristics of linear and non-linear system of autonomous equations used in ecological and epidemiological models. For any dynamical system on $\mathbb{R}^n_+$, a persistence criteria is given by there should be a compact absorbing set M in the interior of $\mathbb{R}^n_+$ for all semi-orbits with initial values in int $\mathbb{R}^n_+$. Various forms of persistence and permanence are studied in [55, 56, 138].

1.6 Environmental fluctuation

It is well known that, population dynamics are often affected by human-nature interaction as well as environmental fluctuation, which is an important component in ecosystem. Ecologist and epidemiologists are now showing their interest to study how the noise affects the population dynamics. Hence stochastic differential equation models play a major role in the field of biology and ecology. In deterministic models, parameters are all deterministic irrespective of environmental fluctuation and hence they are very difficult to predict the future dynamics of the system correctly [12]. Therefore many authors introduced randomness in deterministic models to reveal the effect of environmental variability [12, 35, 149, 85, 24].

1.7 Research objective

In this thesis, we have mainly focused on ecological and eco-epidemic model through continuous and fractional order system. The motivation for the analysis to be presented in this thesis come from important problem in population biology namely to derive the conditions for which a system of interacting species survive in long term, coexist and oscillate.

1.8 Orientation of the thesis

This thesis consists of five main chapters and all chapters are separate but complementary, on ecological and eco-epidemiology aspects. Each chapter of this thesis has its own introduction and discussion, where the context of each chapter is discussed.

In **Chapter 2**, a predator-prey-pathogen model is analyzed where predator influences the transmission rate of the infection in its prey. The main results address the existence of interior equilibrium point and its stability. Bifurcation and persistence of the system are derived. A condition for non-existence of closed orbits is established.

In **Chapter 3**, a Holling type II predator-prey-pathogen model is studied, where predator is specialist in nature and infected prey can undergo refugia of

constant size to avoid predator attack. Model analysis shows that all the population remains in coexistence when predator consumes the infected prey rather then the susceptible one. Global stability of the coexistence equilibrium point is developed by using Li and Muldowney's high dimensional Bendixson's criterion [109].

In **Chapter 4**, a simple eco-epidemic model is analyzed where the host population is infected by Chronic wasting disease (CWD). The structure of equilibria and their linearized stability are investigated. By considering a suitable Lyapunov function, global stability of the endemic equilibrium point is discussed. Different type of local bifurcation including Hopf bifurcation are derived. Stochastic stability of the system is discussed by introducing a white type of noise into the system. This suggests that the deterministic model is robust with respect to stochastic perturbation.

In **Chapter 5**, a fractional order eco-epidemic model is studied where prey population is infected by Chronic wasting disease (CWD). The basic results on existence, uniqueness, non-negativity and boundedness of the solutions are investigated.The criterion for local as well as global stability of the equilibrium points is derived.

In **Chapter 6**, a Leslie-Gower predator-prey model is described in which one predator feeds on one of two competiting species. Existence condition for equilibrium point is discussed. By using differential inequality argument persistence criterion is developed. Sufficient condition for global stability of the unique positive equilibrium point is derived. The role of refuges have been shown on equilibrium density of prey, competitor for prey and predator respectively.

2

The Effect of Predator Density Dependent Transmission Rate in an Eco-Epidemic Model

2.1 Introduction

Control of disease through predation and harvesting is one of the oldest strategy in the world. Various studies on this phenomenon may be found in [31, 66, 80]. Predators and the parasites can have a large effect on prey population when they compete together and assert continuously pressure on prey species. Both the enemies are benefited in such type of interaction which transmits the disease separately in prey or predator species or in both [25, 172, 76, 183, 100]. Holt and

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Roy [75] and Packer et al. [143] showed that predators can increase and decrease the prevalence of infectious disease.

In the last few decades many works have been done in prey-predator model with parasite infection [25, 172, 76, 183, 133, 54, 134]. Most of the previous work deal with the spread of the disease assuming the considered species are not related with the other species for space or food, or is predated by the others. From theoretical studies it can be concluded that predators select prey species based on their vulnerability. It is a common fact that old, sick, injured and infected individuals from prey population are less active and can be caught more easily than the susceptible individuals. Several experimental studies have shown that parasite induce mortality increases vulnerability to predation. For example, anthelmintic treatment reduced the vulnerability of snowshoe hares [141, 140] and red grouse [78] to predators. So predation on infectious individuals from the prey population can prevent from spreading disease. Role of predator density on disease transmission rate have not focused so far in the previous studies [25, 172, 76, 183, 100, 156, 136]. It is observed that predator can modify the behaviour of prey population in presence of disease. Thus prey population becomes vulnerable to a disease in the presence of predator. Ultimately this signifies that disease transmission rate is a function of predator density. Recently Morozov [132] studied predator dependent disease transmission rate in linear form and concluded that this type of transmission can promote epidemics of highly virulent infectious disease. Sen et al. [156] analyzed an epidemic model where the predator is a generalist and the alternative food supply is a dynamic variable. They showed that the predator dependent transmission in the presence of a second prey influences the dynamical behaviour of the system. The following examples illustrate the predator density dependent transmission in eco-epidemic model. For example, in nature in the presence of predators, a fresh water snail spends a long time hiding insides its shell which makes it vulnerable to parasites because the organism cannot expel the blood which is very important for proper immune system functioning [151]. Another situation can be observed when tadpoles avoid predation by reducing their activity and taking refuge among rocks and plants. But in this mechanism it reduced their growth rates and foraging ability [111, 178]. More biological situation can be found in [166, 186, 11].

The form of transmission rate considered by Morozov [132] indicates unboundedness in the transmission rate when predator population increases. So to avoid such unboundedness, we consider saturation effect on it. The main thrust in this chapter is to modify the transmission term from linear to nonlinear one and also mass action term in proportional mixing form to get new dynamics of the system.

2.2 Model formulation

In general, disease transmission is described by interaction among individuals. The incidence function is modelled as $\beta(S, I)$ for which one of them are the mass action $\beta(S, I) = \lambda SI$ and the so called the standard incidence function $\frac{\lambda SI}{S+I}$. In both cases populations interacts randomly. Now the question arises which one is appropriate? Some literature shows that simple mass action is not an adequate model in many situation [123]. Furthermore, most of the studies [25, 172, 183, 100, 134, 26, 150] considered that λ is independent of predator density. In [132], Morozov considered the incidence function in the form $\lambda(S, I) = (\lambda_0 + aP)SI$. In our work, we modify the above incidence function as $\lambda(S, I) = (\lambda_0 + \frac{aP}{1+bP})\frac{SI}{S+I}$ which tends to a saturation level when P gets large. This incidence rate is more reasonable than the linear incidence rate because it includes the behavioural change and crowding effect of the predator and prevents unboundedness of the disease transmission rate. We also take the transmission rate as proportional mixing form. Our modified model is:

$$\begin{aligned}
\frac{dS}{dt} &= S\left[r\left(1-\frac{S+I}{K}\right)-\left(\lambda_0+\frac{aP}{1+bP}\right)\frac{I}{S+I}\right] \\
\frac{dI}{dt} &= \left(\lambda_0+\frac{aP}{1+bP}\right)\frac{SI}{S+I}-dI-\frac{\alpha_1 IP}{1+\beta I} \\
\frac{dP}{dt} &= P\left(-\delta+\frac{\alpha_2 I}{1+\beta I}\right)
\end{aligned} \tag{2.1}$$

with initial conditions $S(0) > 0, I(0) > 0, P(0) > 0$.

Here, $S(t), I(t)$ and $P(t)$ denote the density of the healthy prey, infected prey and predator population respectively. $S(t) + I(t)$ is the total prey density. We assume that the disease is not genetically inherited and the infected populations

Table 2.1: Parameters of system (2.1) and their units

Parameter	Definition
r	maximum per capita growth rate of healthy prey
K	carrying capacity of the environment
λ_0	transmission rate in the absence of predator
a	predator density mediated additional disease transmission rate
b	inhibitory effect
d	death rate of infected prey population
α_1	per capita predator consumption rate
β	the encounter rate between the predator and infected prey
α_2	the conversion efficiency of the predator
δ	the death rate of predator

do not recover or become immune. Here predator functional response is Holling type-II which describes how the consumption rate of the predator depends on prey density. All other model parameters are defined in Table 2.1.

2.3 Boundedness of the system

Lemma 2.3.1. *All solutions of system (2.1) that initiate in* $\mathbb{R}^3_+$ *are bounded and enter the region* D *defined by*
$D = \{(S, I, P) \in \mathbb{R}^3_+ : 0 < W(t) \leq \frac{M(r+1)}{v} + \epsilon\}$ *as* $t \to \infty$, *where* $v = min\{1, d, \delta\}$.

Proof. Assume $(S(t), I(t), P(t))$ be any solution of system (2.1).

Since $\frac{dS}{dt} \leq rS(1 - \frac{S}{K})$, by a standard comparison theorem we have $\limsup S(t) \leq M$, where $M = \max\{S(0), K\}$.

Define the function $W = S + I + \frac{\alpha_1}{\alpha_2}P$.

The time derivative along a solution of (2.1) is

$$\begin{aligned}\frac{dW}{dt} &= \frac{dS}{dt} + \frac{dI}{dt} + \frac{\alpha_1}{\alpha_2}\frac{dP}{dt} \\ &= rS(1 - \tfrac{S+I}{K}) - dI - \tfrac{\alpha_1}{\alpha_2}\delta P\end{aligned}$$

$$\leq rS - dI - \frac{\alpha_1}{\alpha_2}\delta P \leq (r+1)M - vW. \quad \text{where, } v{=}\min\{1, \delta, d\}$$

Thus, $\frac{dW}{dt} + vW \leq (r+1)M$.

Then by usual comparison theorem [18], we obtain,

$$0 \leq W(S(t), I(t), P(t)) \leq \frac{(r+1)M}{v} + \frac{W(S(0),I(0),P(0))}{e^{vt}}$$

and for $t \to \infty$, $0 \leq W(S(t), I(t), P(t)) \leq \frac{(r+1)M}{v}$. So, all solutions of system (2.1) enter the region $D = \{(S, I, P) \in \mathbb{R}^3_+ : W \leq \frac{M(r+1)}{v}\}$.

2.4 Equilibrium and their stability

In this section, we analyze the stability condition of the boundary equilibrium points and the interior equilibrium point of the system (2.1). The trivial equilibrium point $E_0(0, 0, 0)$ does not exist in the system as there is a singularity at the origin. The system (2.1) has one axial equilibrium point $E_1(K, 0, 0)$ and one predator free equilibrium point $E_2(\bar{S}, \bar{I}, 0)$, where $\bar{S} = \frac{d\bar{I}}{\lambda_0 - d}$ and $\bar{I} = \frac{rK(\lambda_0-d)-K(\lambda_0-d)^2}{r\lambda_0}$. Clearly E_1 is always exist and E_2 is feasible if $d < \lambda_0 < r + d$.

Next we are interested about the existence of interior equilibrium point $E^*(S^*, I^*, P^*)$ of system (2.1). To locate the positive equilibrium point of system (2.1), we consider

$$\begin{aligned} S\left[r\left(1 - \frac{S+I}{K}\right) - \left(\lambda_0 + \frac{aP}{1+bP}\right)\frac{I}{S+I}\right] &= 0, \\ \left(\lambda_0 + \frac{aP}{1+bP}\right)\frac{SI}{S+I} - dI - \frac{\alpha_1 IP}{1+\beta I} &= 0, \\ P\left(-\delta + \frac{\alpha_2 I}{1+\beta I}\right) &= 0. \end{aligned} \tag{2.2}$$

From the third subsection of equation (2.2), we obtain $I^* = \frac{\delta}{\alpha_2 - \delta\beta} = R(\text{say})$, it is clear that $I^* > 0$ if $\alpha_2 > \delta\beta$.

Now substitute the value of I^* in the subsection two of equation (2.2) and we get,

$$f(S,P) = \left(\lambda_0 + \frac{aP}{1+bP}\right)\frac{S}{S+R} - d - \frac{\alpha_1 P}{1+\beta R} = 0. \tag{2.3}$$

Again from subsection one of equation (2.2) we get,

$$g(S,P) = \left[r\left(1 - \frac{S+R}{K}\right) - \left(\lambda_0 + \frac{aP}{1+bP}\right)\frac{R}{S+R}\right] = 0. \tag{2.4}$$

From equation (2.3) we note the following: when $P \to 0$, then $S \to S_1$, where

$$S_1 = \frac{dR}{\lambda_0 - d}, \tag{2.5}$$

which requires $\lambda_0 > d$ to ensure the positivity of S_1.

We also have $\frac{dS}{dP} = -\frac{\partial f}{\partial P}/\frac{\partial f}{\partial S} = \frac{M_1}{N_1}$, where

$$M_1 = (S+R)\left[\frac{\alpha_1(S+R)}{1+\beta R} - \frac{aS}{(1+bP)^2}\right]$$

and

$$N_1 = R\left(\lambda_0 + \frac{aP}{1+bP}\right).$$

It is clear that $N_1 > 0$ so $\frac{dS}{dP} > 0$ if $M_1 > 0$ that is $\frac{\alpha_1(S+R)}{1+\beta R} > \frac{aS}{(1+bP)^2}$ hold.

From equation (2.4) we get the following: when $P \to 0$, then $S \to S_2$, where

$S_2 = \frac{-a_2 \pm \sqrt{a_1^2 - 4a_1a_3}}{2a_1}$
$a_1 = \frac{r}{K}$
$a_2 = \frac{r(2R-K)}{K}$
$a_3 = \frac{r}{K}R^2 + R(\lambda_0 - r)$.

Clearly $a_3 < 0$ if $R < \frac{K(r-\lambda_0)}{r}$ and $r > \lambda_0$.

We also get,

$\frac{dS}{dP} = -\frac{\partial g}{\partial P}/\frac{\partial g}{\partial S} = -\frac{M_2}{N_2}$,where

$$M_2 = \frac{aR}{(S+R)(1+bP)^2}$$

and

$$N_2 = \frac{r}{K} - \left(\lambda_0 + \frac{aP}{1+bP}\right)\frac{R}{(S+R)^2}.$$

Clearly we note that $\frac{dS}{dP} < 0$ if $\frac{r}{K} > \left(\lambda_0 + \frac{aP}{1+bP}\right)\frac{R}{(S+R)^2}$.

From the above analysis we conclude that the two isoclines (2.3) and (2.4) intersect at a unique point (S^*, P^*) depending on the conditions $\alpha_2 > \delta\beta$, $\frac{1}{(1+bP)^2} > \frac{\alpha_1}{1+\beta R}$, and

$$S_1 < S_2. \tag{2.6}$$

Knowing the values of S^*, I^* and P^*, one can easily conclude the existence of the interior equilibrium point E^*.

Now we compute the variational matrix of system (2.1). The signs of the real parts of the eigenvalues of the matrix evaluated at a given equilibrium determine its stability. The variational matrix of system (2.1) at any arbitrary point (S, I, P) is $V = (m_{ij}) \in \mathbb{R}^{3\times 3}$ with

$$V = (m_{ij}) = \begin{pmatrix} m_{11} & m_{12} & m_{13} \\ m_{21} & m_{22} & m_{23} \\ m_{31} & m_{32} & m_{33} \end{pmatrix}$$

$$\text{where}\begin{cases} m_{11} = r(1-\frac{S+I}{K}) - (\lambda_0 + \frac{aP}{1+bP})\frac{I}{S+I} - \frac{rS}{K} + \frac{SI}{(S+I)^2}(\lambda_0 + \frac{aP}{1+bP}) \\ m_{12} = -\frac{rS}{K} - (\lambda_0 + \frac{aP}{1+bP})\frac{S^2}{(S+I)^2} \\ m_{13} = -\frac{SI}{S+I}\frac{a}{(1+bP)^2} \\ m_{21} = (\lambda_0 + \frac{aP}{1+bP})\frac{I^2}{(S+I)^2} \\ m_{22} = (\lambda_0 + \frac{aP}{1+bP})\frac{S}{S+I} - d - \frac{\alpha_1 P}{1+\beta I} - (\lambda_0 + \frac{aP}{1+bP})\frac{SI}{(S+I)^2} + \frac{\alpha_1 IP\beta}{(1+\beta I)^2} \\ m_{23} = -\frac{\alpha_1 I}{1+\beta I} + \frac{aSI}{(S+I)(1+bP)^2} \\ m_{31} = 0 \\ m_{32} = \frac{\alpha_2 P}{(1+\beta I)^2} \\ m_{33} = -\delta + \frac{\alpha_2 I}{1+\beta I} \end{cases}$$

Lemma 2.4.1. *(i) E_1 is locally asymptotically stable if $\lambda_0 < d$ and unstable otherwise.*
(ii) E_2 is locally asymptotically stable if $\delta > \frac{\alpha_2 \bar{I}}{1+\beta \bar{I}}$ and unstable otherwise.

Proof. (i) By linearizing the system around the equilibrium point $E_1(K, 0, 0)$ we obtain three eigenvalues: $-r$, $\lambda_0 - d$, $-\delta$, for which two of the eigenvalues are negative and other one is negative if $\lambda_0 < d$. So, E_1 is locally asymptotically stable if $\lambda_0 < d$ and unstable if $\lambda_0 > d$.

(ii) Variational matrix around the planer equilibrium point $E_2(\bar{S}, \bar{I}, 0)$ is given by:

$$V(E_2) = \begin{pmatrix} -\frac{r\bar{S}}{K} + \frac{\lambda_0 \bar{S}\bar{I}}{(\bar{S}+\bar{I})^2} & -\frac{r\bar{S}}{K} - \frac{\lambda_0 \bar{S}^2}{(\bar{S}+\bar{I})^2} & -\frac{a\bar{S}\bar{I}}{\bar{S}+\bar{I}} \\ \frac{\lambda_0 \bar{I}^2}{(\bar{S}+\bar{I})^2} & -\frac{\lambda_0 \bar{S}\bar{I}}{(\bar{S}+\bar{I})^2} & -\frac{\alpha_1 \bar{I}}{1+\beta \bar{I}} + \frac{a\bar{S}\bar{I}}{\bar{S}+\bar{I}} \\ 0 & 0 & -\delta + \frac{\alpha_2 \bar{I}}{1+\beta \bar{I}} \end{pmatrix}.$$

The eigenvalues of $V(E_2)$ are $-\delta + \frac{\alpha_2 \bar{I}}{1+\beta \bar{I}}$ and $\lambda_{\pm} = \frac{1}{2}\big[-\frac{r\bar{S}}{K} \pm \sqrt{(\frac{r\bar{S}}{K})^2 - 4\frac{r\lambda_0 \bar{S}\bar{I}}{K(\bar{S}+\bar{I})}}\big]$. The signs of the real parts of λ_+ and λ_- are always negative. This implies that E_2 is locally asymptotically stable in the $S-I$ plane and asymptotically stable or unstable in the P direction according to whether $-\delta + \frac{\alpha_2 \bar{I}}{1+\beta \bar{I}}$ is negative or positive respectively (Table 2.1).
Now we observe that when E_2 is feasible, E_1 is unstable. To show E_2 is globally asymptotically stable in the $S - I$ plane, we use Bendixson-Dulac criterion.

To prove global stability of E_2, we define $H(S, I) = \frac{1}{SI}$. Clearly $H > 0$ when

Table 2.2: Existence conditions and properties of the boundary equilibrium points of system (2.1).

Equilibrium	Properties	Conditions
$E_1(K,0,0)$	Stable	$\lambda_0 < d$
$E_1(K,0,0)$	Unstable	$\lambda_0 > d$
$E_2(\bar{S},\bar{I},0)$	Stable	$\delta > \frac{\alpha_2\bar{I}}{1+\beta\bar{I}}$
$E_2(\bar{S},\bar{I},0)$	Unstable	$\delta < \frac{\alpha_2\bar{I}}{1+\beta\bar{I}}$

$S>0$ and $I>0$. Let $g_1(S,I) = S\left[r\left(1-\frac{S+I}{K}\right)-\frac{\lambda_0 I}{S+I}\right]$ and $g_2(S,I) = I\left[\frac{\lambda_0 S}{S+I}-d\right]$. Then $\Delta(S,I) = \frac{\partial}{\partial S}(g_1 H)+\frac{\partial}{\partial I}(g_2 H) = -\frac{r}{KI} < 0$. As E_2 is locally asymptotically stable so by Bendixson-Dulac criterion it is globally asymptotically stable.

Now we analyze the stability of E^*. The characteristic equation of the variational matrix V about the interior equilibrium point E^* is given by

$$\lambda^3 + p_1\lambda^2 + p_2\lambda + p_3 = 0 \tag{2.7}$$

where
$$\begin{cases} p_1 = \frac{rS^*}{K} - \frac{\alpha_1\beta I^* P^*}{(1+\beta I^*)^2} \\ p_2 = (\lambda_0 + \frac{aP^*}{1+bP^*})\frac{S^*I^*}{S^*+I^*}\left[\frac{r}{K} + \frac{rI^{*2}}{K(S^*+I^*)} + \frac{\alpha_1\beta I^*P^*}{(S^*+I^*)(1+\beta I^*)^2}\right] + \frac{\alpha_1\alpha_2 I^*P^*}{(1+\beta I^*)^3} \\ \qquad - \frac{S^*I^*P^*}{(1+\beta I^*)^2}\left[\frac{\alpha_1 r\beta}{K} + \frac{\alpha_2}{(S^*+I^*)(1+bP^*)^2}\right] \\ p_3 = \left[-\frac{rS^*}{K} + \frac{S^*I^*}{(S^*+I^*)^2}(\lambda_0 + \frac{aP^*}{1+bP^*})\right]\left[-\frac{\alpha_1 I^*}{1+\beta I^*} + \frac{aS^*I^*}{(S^*+I^*)(1+bP^*)^2}\right] \\ \qquad + \frac{aS^*I^*}{(S^*+I^*)(1+bP^*)^2}\left[\frac{I^{*2}}{(S^*+I^*)^2(\lambda_0+\frac{aP^*}{1+bP^*})}\right] \end{cases}$$

Now by the Routh-Hurwitz criterion, it follows that all the eigenvalues of characteristic equation have negative real part if and only if $p_1>0, p_3>0$ and $\triangle = p_1p_2 - p_3 > 0$.

Remark 2.4.1. *The interior equilibrium point $E^*(S^*,I^*,P^*)$ if exists, then it is locally asymptotically stable if the following conditions holds: $p_1>0, p_3>0$ and $\triangle>0$.*

2.5 Persistence

Ecologically point of view persistence means long time survival of all population in the future time and none of them will become extinct . It does not depend on the initial population but depends on solution behaviour near the boundaries. Mathematically it means that strictly positive solutions do not have omega limit set on the boundary of the non-negative cone. Persistence and permanence are studied in [55, 56, 138].

Theorem 2.5.1. *The system (2.1) is uniformly persistent if* $r + d > \lambda_0 > d$ *and* $\delta < \frac{\alpha_2 \bar{I}}{1+\beta \bar{I}}$.

Proof. Assume, α be any point in the positive octant and $o(\alpha)$ is the orbit through the point α and $\Omega(\alpha)$ is the bounded omega limit set of the orbit through α. Suppose E_1 not in $\Omega(\alpha)$. If $E_1 \in \Omega(\alpha)$ then by Butler-McGehee lemma [55], there exist a point p in $\Omega(\alpha) \cap W^s(E_1)$, where $W^s(E_1)$ be the strong manifold of E_1. Since $o(p) \in \Omega(\alpha)$ and $W^s(E_1)$ in the $S - P$ plane, we conclude that $o(p)$ is unbounded, which is a contradiction. Similarly we can show that $E_2 \notin \Omega(\alpha)$, by the condition $\delta < \frac{\alpha_2 \bar{I}}{1+\beta \bar{I}}$ implies that E_2 is a saddle point. $W^s(E_2)$ is the $S - I$ plane, hence $o(p)$ is unbounded in $\Omega(\alpha)$, another contradiction. Therefore there does not exist any equilibrium point in $S-P$ plane and there is no closed orbit in the $S - I$ plane. Thus $\Omega(\alpha)$ does not intersect any of the coordinates plane and system (2.1) is persistent. As system (2.1) is bounded, by Butler main theorem [21], the system is uniformly persistent.

Remark 2.5.1. *In 1986, Butler et al. [21] proved that in a Euclidean space, uniform persistence implies the existence of an interior equilibrium point. Hence* E^* *exists follows from Theorem 2.5.1.*

Corollary 2.5.1.1. *If* $\frac{\alpha_2 K}{1+\beta K} < \delta$ *be hold, then system (2.1) is impermanent.*

Proof. The condition $\frac{\alpha_2 K}{1+\beta K} < \delta$ implies that $\frac{\dot{P}}{P} < 0$ at E_2. So E_2 is strictly a saturated equilibrium point on the boundary. Hence, there exists at least one orbit in the interior that converges to the boundary [73]. Consequently the system (2.1) is impermanent [83].

2.6 Hopf-bifurcation

In this section we discuss Hopf bifurcation of system (2.1). We ensure whether Hopf bifurcation occur or not for system (2.1). Here we consider a as a bifurcation parameter. In the following theorem we will show that Hopf bifurcation occur for the system (2.1) at a critical value $a = a^*$.

Theorem 2.6.1. *The system possesses a Hopf bifurcation around its positive equilibrium point E^* when the predator density dependent disease transmission rate a passes through a critical value a^* provided the following conditions hold: (i) $p_1(a^*) > 0, (ii)$ $p_1(a^*)p_2(a^*) - p_3(a^*) = 0, and (iii)$ $[p_1(a)p_2(a)]' < p_3'(a)$ at $a = a^*$.*

Proof. We assume that the interior equilibrium point E^* of system (2.1) is locally asymptotically stable. Here we choose a is the bifurcation parameter, so we would like to know that whether the system loses its stability or not when value of the parameter a changes.

The characteristic equation is given in equation (2.7). Hopf bifurcation will occur iff there exist $a = a^*$ such that (i) $p_1(a^*) > 0$, (ii) $p_1(a^*)p_2(a^*) - p_3(a^*) = 0$ and $(iii)\left(\frac{dRe(\lambda(a))}{da}\right)_{a=a^*} \neq 0$.

Now when $a = a^*$ the characteristic equation (2.7) is of the form :

$$\lambda^3(a^*) + p_1(a^*)\lambda^2(a^*) + p_2(a^*)\lambda(a^*) + p_1(a^*)p_2(a^*) = 0$$

i.e

$$(\lambda(a^*) + p_1(a^*))(\lambda^2(a^*) + p_2(a^*)) = 0 \tag{2.8}$$

which has three roots $\lambda_j(a^*) = \pm i\sqrt{p_2(a^*)}, j = 1, 2$ and $\lambda_3(a^*) = -p_1(a^*)$, so that there is one strictly negative real eigenvalue and a pair of purely imaginary eigenvalues.

For all a, the roots are in general form:

$$\begin{cases} \lambda_1(a) = u(a) + iv(a), \\ \lambda_2(a) = u(a) - iv(a), \\ \lambda_3(a) = -p_1(a). \end{cases}$$

Now, we will verify the transversality condition:

$$\left(\frac{dRe(\lambda_j(a))}{da}\right)_{a=a^*} \neq 0, j = 1, 2.$$

Substitute the values of $\lambda_j(a), i = 1, 2$ into (2.8) and calculating the derivative, we get

$$R(a)u'(a) + S(a)v'(a) + A(a) = 0$$

$$R(a)u'(a) + S(a)v'(a) - B(a) = 0$$

$$\text{where} \begin{cases} R(a) = 3u^2(a) + p_2(a) - 3v^2(a) + 2p_1(a)u(a) \\ S(a) = 6u(a)v(a) + 2p_1(a)v(a) \\ A(a) = u^2(a)p_1'(a) + p_2'(a)u(a) + p_3'(a) - p_1'(a)v^2(a) \\ B(a) = 2u(a)v(a)p_1'(a) + p_2'(a)v(a) \end{cases}$$

Now,

$$\begin{aligned} \left(\frac{dRe(\lambda_j(a))}{da}\right)_{a=a^*} &= -\frac{S(a^*)B(a^*)+R(a^*)A(a^*)}{R^2(a^*)+S^2(a^*)} \\ &= \frac{p_3'(a^*)-p_1'(a^*)p_2(a^*)-p_1(a^*)p_2'(a^*)}{p_1^2(a^*)+p_2(a^*)} > 0 \end{aligned}$$

if $[p_1(a^*)p_2(a^*)]' < p_3'(a^*)$ and $\lambda_3(a^*) = -p_1(a^*) < 0$.

This completes the proof.

2.7 Non-existence periodic solution around interior equilibrium point

Here, we want to show that under some suitable conditions, the system (2.1) has no periodic solution around the positive equilibrium point E^*. To prove this we can apply the following criterion developed in [109].

Consider a general autonomous ordinary differential equation

$$\frac{dX}{dt} = F(X) \tag{2.9}$$

where F is a C^1 function in some open subset of $\mathbb{R}^N$. The Jacobian matrix of system (2.9) is denoted by $J = \frac{dF}{dX}$ and $J^{[2]}$ be the $\binom{N}{2} \times \binom{N}{2}$ matrix which is the second additive compound matrix associated the Jacobian matrix J. The definition of second additive compound matrix can be found in Li and Muldowney [109]. Let $J = (a_{ij})$ be an $n \times n$ matrix. Then the matrix can be defined as follows:

For any integer $i = 1, 2, 3, ...\binom{N}{2}$, let $(i) = (i_1, i_2)$ be the ith member in the lexicographic ordering of integer pairs (i_1, i_2), such that, $1 \leq i_1 \leq i_2 \leq n$. Then the element in the ith row and jth column of $J^{[2]}$ is

$$\begin{cases} a_{i_1 i_1} + a_{i_2 i_2}, & \text{if } (i) = (j), \\ (-1)^{r+s} a_{i_r j_s}, & \text{if exactly one entry } i_r \text{ of } (i) \text{ does not occur in } (j) \text{ and } j_s \text{ does not occurs in} \\ 0, & \text{if neither entry from } (i) \text{ occurs in } (j). \end{cases}$$

For a general 3×3 matrix

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

its second additive compound matrix $J^{[2]}$ is

$$J^{[2]} = \begin{pmatrix} a_{11}+a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11}+a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22}+a_{33} \end{pmatrix}$$

In this case $(1) = (1,2), (2) = (1,3), (3) = (2,3)$.

Theorem 2.7.1. *(Bendixson's criterion in $\mathbb{R}^n$). A simple closed rectifiable curve which is invariant with respect to the system (2.9) cannot exist if any one of the following conditions is satisfied on $\mathbb{R}^n$:*

$$(i) \quad sup\left\{\frac{\partial F_r}{\partial x_r}+\frac{\partial F_s}{\partial x_s}+\sum_{q\neq r,s}\left(\left|\frac{\partial F_q}{\partial x_r}\right|+\left|\frac{\partial F_q}{\partial x_s}\right|\right) : 1\leq r<s\leq n\right\}<0,$$

$$(ii) \quad sup\left\{\frac{\partial F_r}{\partial x_r}+\frac{\partial F_s}{\partial x_s}+\sum_{q\neq r,s}\left(\left|\frac{\partial F_r}{\partial x_q}\right|+\left|\frac{\partial F_s}{\partial x_q}\right|\right) : 1\leq r<s\leq n\right\}<0,$$

$(iii) \lambda_1+\lambda_2<0,$

$$(iv) \quad inf\left\{\frac{\partial F_r}{\partial x_r}+\frac{\partial F_s}{\partial x_s}-\sum_{q\neq r,s}\left(\left|\frac{\partial F_q}{\partial x_r}\right|+\left|\frac{\partial F_q}{\partial x_s}\right|\right) : 1\leq r<s\leq n\right\}>0,$$

$$(v) \quad inf\left\{\frac{\partial F_r}{\partial x_r}+\frac{\partial F_s}{\partial x_s}-\sum_{q\neq r,s}\left(\left|\frac{\partial F_r}{\partial x_q}\right|+\left|\frac{\partial F_s}{\partial x_q}\right|\right) : 1\leq r<s\leq n\right\}>0,$$

$(vi) \lambda_{n-1}+\lambda_n>0$

where $\lambda_1 \geq \lambda_2 \geq \lambda_3 \geq ... \geq \lambda_n$ are the eigenvalues of $(\frac{1}{2})((\frac{\partial F}{\partial x})^* + (\frac{\partial F}{\partial x}))$ and $\frac{\partial F}{\partial x}$ is the Jacobian matrix of F where asterisk denotes the transposition.

Let, $X \in \mathbb{R}^N$ then the corresponding logarithmic norm of $J^{[2]}$, denoted by $\mu_\infty(J^{[2]})$, endowed by the vector norm $|X| = sup_i |X_i|$ is

$$\mu_\infty(J^{[2]}) = sup\left\{\frac{\partial F_r}{\partial x_r}+\frac{\partial F_s}{\partial x_s}+\sum_{q\neq r,s}\left(\left|\frac{\partial F_q}{\partial x_r}\right|+\left|\frac{\partial F_q}{\partial x_s}\right|\right) : 1\leq r<s\leq n\right\},$$

where $\mu_\infty(J^{[2]}) < 0$ implies the diagonal dominance by row matrix $J^{[2]}$. Then, the following result holds.

Theorem 2.7.2. *A simple closed rectifiable curve that is invariant under system (2.1) cannot exist if* $\mu_\infty(J^{[2]}) < 0$.

Here we apply Li-Muldowney's criterion for the non-existence of periodic solutions of system (2.1). The logarithm norm μ_∞, endowed by the norm $|X|_\infty$ of the second additive compound matrix $J^{[2]}$, associated with the Jacobian J, is negative if the supremum of the following functions satisfy:

$$-d-\delta+2\left(\lambda_0+\frac{aP}{1+bP}\right)\frac{S^2}{(S+I)^2}-\frac{\alpha_1 P}{(1+\beta I)^2} +\frac{\alpha_2 I}{1+\beta I}+\frac{aSI}{(S+I)(1+bP)^2}+\frac{rS}{K}<0, \tag{2.10}$$

$$r-d-\frac{2rS}{K}-\frac{rI}{K}+\left(\lambda_0+\frac{aP}{1+bP}\right)\frac{S-I}{S+I}+\frac{(\alpha_2-\alpha_1)P}{(1+\beta I)^2}<0, \tag{2.11}$$

$$r-\delta-\frac{2rS}{K}-\frac{rI}{K}+\frac{(\alpha_1+\alpha_2)I}{1+\beta I}+\frac{aSI}{(S+I)(1+bP)^2}<0. \tag{2.12}$$

Now the left hand side of inequality (2.10)
$-d-\delta+2\left(\lambda_0+\frac{aP}{1+bP}\right)\frac{S^2}{(S+I)^2}-\frac{\alpha_1 P}{(1+\beta I)^2}+\frac{\alpha_2 I}{1+\beta I}+\frac{aSI}{(S+I)(1+bP)^2}+\frac{rS}{K}$
$\leq -d-\delta+2(\lambda_0+\frac{a}{b})+r+aK+\frac{\alpha_2}{\beta}$

since $\frac{aP}{1+bP}<\frac{a}{b}, \frac{S^2}{(S+I)^2}<1, \frac{I}{1+\beta I}<\frac{1}{\beta}, S<K, \frac{I}{S+I}<1, \frac{1}{(1+bP)^2}<1.$

Thus inequality (2.10) will follow if

$$d+\delta>2(\lambda_0+\frac{a}{b})+r+aK+\frac{\alpha_2}{\beta}. \tag{2.13}$$

So one can easily show the inequalities (2.10), (2.11), (2.12) if (i), (ii), (iii) hold respectively, where

(i) $d+\delta>2(\lambda_0+\frac{a}{b})+r+aK+\frac{\alpha_2}{\beta}$, (ii) $d>r+\lambda_0+\frac{a}{b}$, (iii) $\delta>r+aK$ and $\frac{r}{K}>\alpha_1+\alpha_2$.

Remark 2.7.1. *In our model it is quite difficult to construct a suitable Lyapunov function to prove global stability of the system. If the condition of Theorem 2.6.1 is not satisfied then there is a possibility that system (2.1) may be globally stable but our last result (i)-(iii) one can get some insight about the global convergence of the solutions.*

2.8 Numerical simulations of the model

In this following section, we will present some examples to verify our results obtained earlier based on computer simulation using MATLAB. We make some numerical observation. The axial equilibrium point E_1 is locally asymptotically stable if $\lambda_0 < d$, unstable otherwise. Now we choose the parameters of system (2.1) such as $r = 3, K = 5, \lambda_0 = 1.5, a = 1, b = 1, d = 0.5, \alpha_1 = 1, \alpha_2 = 1, \beta = 1, \delta = 0.5$ and $(S(0), I(0), P(0)) = (1.88, 1.35, 1.99)$ which satisfy the conditions $p_1 > 0, p_3 > 0$ and $p_1p_2 - p_3 > 0$ and consequently the interior equilibrium point $E^*(S^*, I^*, P^*) = (3.107, 1, 2.328)$ is locally asymptotically stable. The phase diagram is shown in Fig.(2.1)

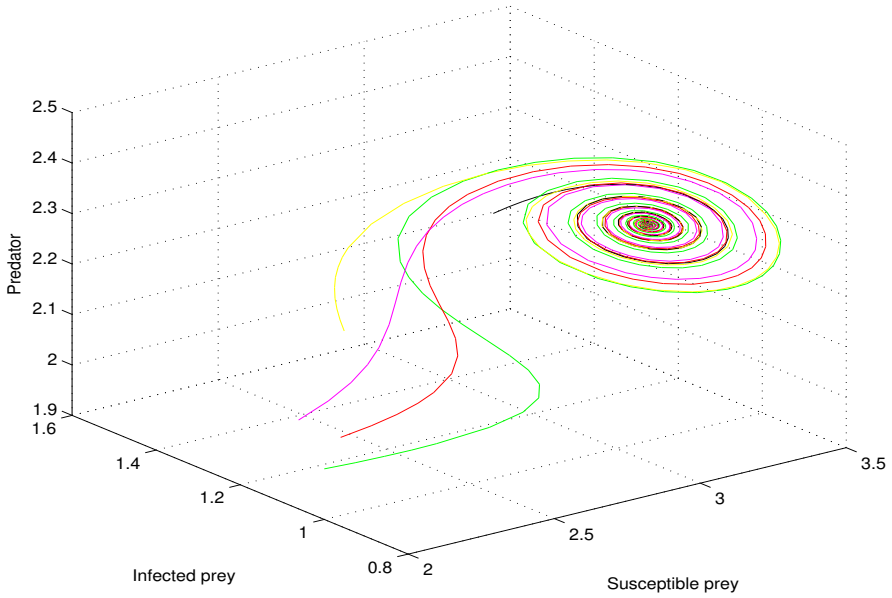

Figure 2.1: The interior equilibrium point is locally asymptotically stable

Now we increase the value of the parameter a from 1 to 2.8 and keeping all other parameters fixed. We observed unstable behaviour of the system (see Fig. (3.3)).

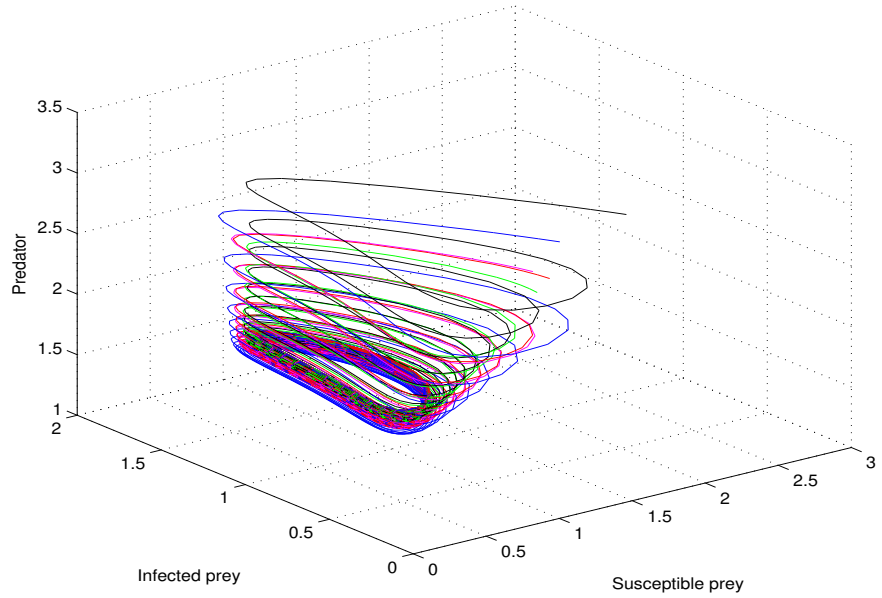

Figure 2.2: System (2.1) is unstable

We also studied the Hopf bifurcation of the system taking a as a bifurcation parameter. The transversality condition is satisfied when $a = a^* = 2.783$ and we consider all other parameters of the system are keeping same. It is clear that the system (2.1) has an equilibrium point (4.94,1.22,4.83). Then it follows from Theorem 2.6.1, Hopf bifurcation occurs at $a = 2.783$ (see Fig.(2.3)).

Now we consider another bifurcation parameter α_2. Suppose $\alpha_2 = 1.2$ and all other parameters remains unaltered. Then system (2.1) undergoes a Hopf bifurcation (See Fig.(2.4)).

Now we choose β as a bifurcation parameter. We will see that when β crosses a critical value then a Hopf bifurcation of periodic solution occurs at $\beta = \beta^* = 0.19$ and all other parameters remain unchanged (See Fig.(2.5)).

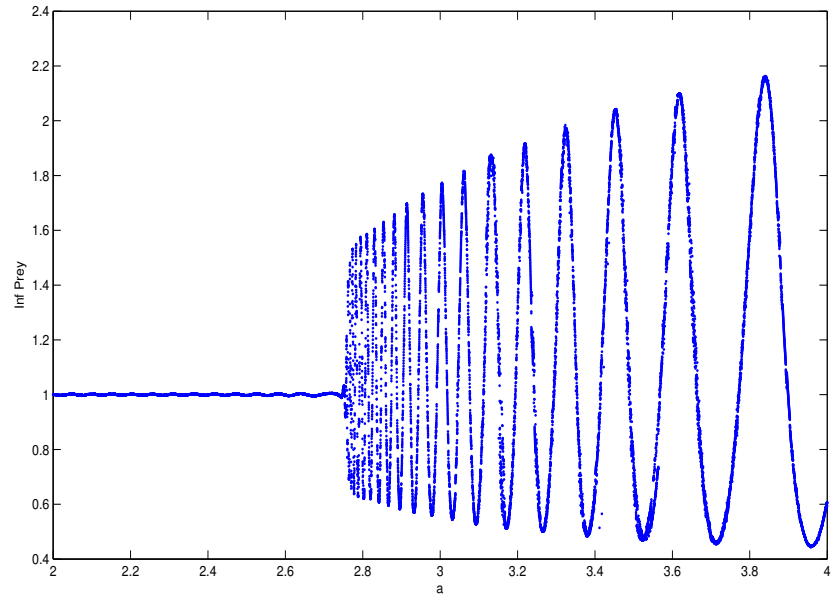

Figure 2.3: The interior equilibrium point E^* loses its stability and Hopf-bifurcation occurs

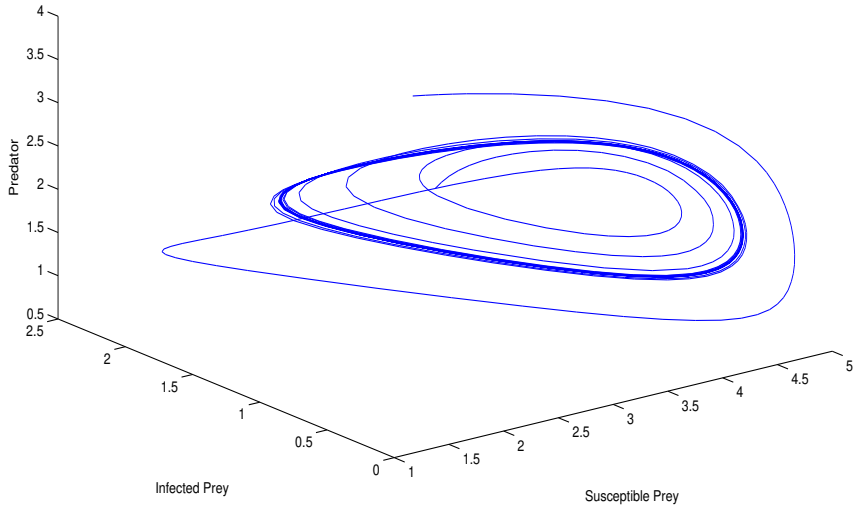

Figure 2.4: Hopf-bifurcation occurs at $\alpha_2 = \alpha_2^* = 1.2$

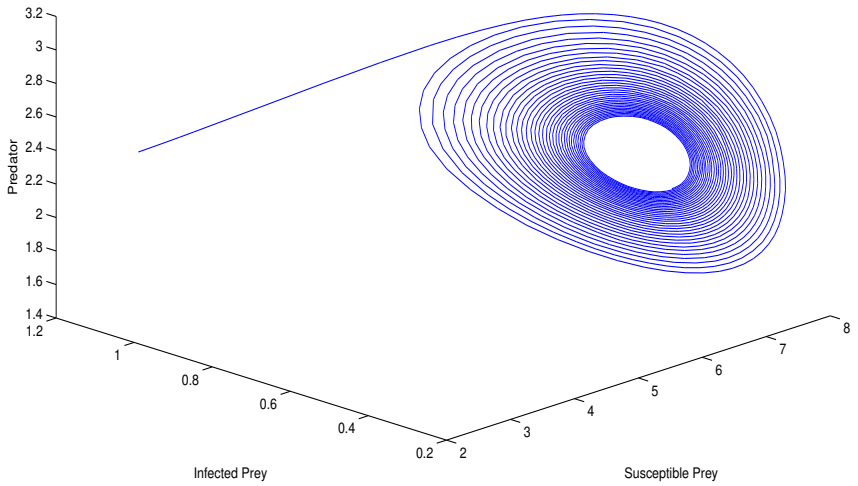

Figure 2.5: Hopf-bifurcation occurs at $\beta = \beta^* = 0.19$

2.9 Discussion

Morozov [132], considered a $S - I$ model with Rosenzweig-MacArther prey-predator model where, disease transmission rate in βSI form, β is predator density dependent and is taken in linear form $[\beta(P) = \lambda_0 + \alpha P]$. This chapter generalizes the above infection term in the form $(\lambda_0 + \frac{aP}{1+bP})\frac{SI}{S+I}$. This type of infection term indicates saturation of transmission for large predator densities and the transmission rate is ratio dependent. In [132], it is established that predator-dependent disease transmission can result in bi-stability and destabilization even for a Holling type-I predator functional response. In our work, we have shown coexistence results and non-existence of closed orbits. Here we got three equilibrium points. The first equilibrium point is $E_1(K, 0, 0)$ where only prey population can survive and this equilibrium point is unstable if $\lambda_0 > d$, i.e in the absence of predator as long as the disease transmission rate exceeds the death rate of infected prey population. The second equilibrium point is predator free equilibrium point and if this equilibrium exists then the first equilibrium point is unstable. The second equilibrium point is stable if the death rate of the

predator exceeds a certain threshold value ($\delta > \frac{\alpha_2 \bar{I}}{1+\beta \bar{I}}$), otherwise unstable. The third equilibrium point is interior equilibrium point where all the population are present. In section 4, we developed a result concerning the existence of positive equilibrium point. We have investigated the case when the system is persistent and we find out the condition that the system is persistent if $r + d > \lambda_0 > d$ and $\delta < \frac{\alpha_2 \bar{I}}{1+\beta \bar{I}}$. We have noticed that the predator density dependent transmission rate causes oscillation in our system. We identify the parameter 'a' (predator density mediated additional disease transmission rate) which controls the dynamics. We have also addressed bifurcation phenomena for the other parameters namely α_2 and β through numerical simulation. The system is impermanent if $\frac{\alpha_2 K}{1+\beta K} < \delta$. Also we have derived the condition for the non existence of closed orbits. All the results are verified by numerical simulation using Matlab.

3

Bifurcation and global stability in an eco-epidemic model with refuge

3.1 Introduction

In a real ecosystem, the interaction between the predator and their prey is a complex process. This complexity has attracted the attention of both theoretical and mathematical ecologists and hence prey-predator models have been investigated extensively. It is well known that most of the ecological populations suffer from various infectious diseases and these diseases have a significant role in regulating population sizes [45]. Thus, it is worthwhile to study the combined effect of epidemiological and demographic features on real ecological populations. Mathematical studies of such eco-epidemiological models have explored various

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unknown aspects of ecological populations. The classic Kermack and McKendrick [93] SIR model has drawn much more attention among the epidemiologists. Most of epidemiologists studies deal with the spread of the disease assuming the considered species are not related with the other species for space or food or predated by the others. It is observed that many species become extinct due to natural or man made reasons. Several studies mainly focus parasite infection in prey population only [171, 63, 184, 189, 68, 165] although there are few work where disease has been considered into the model on both species [76, 15]. From modelling point of view, infection on prey species is an important issue in the dynamics of a prey-predator system. It is observed that predators preferentially consume infected prey as they are vulnerable and less active than healthy prey [79]. Theoretically it can be shown that predation on infected prey populations can both increase and decrease the infection prevalence of the disease [75, 143]. However, there is a risk factor for predator to become infected by consuming infected prey. Relevant work in this regard may be found in [52]. Hadeler and Freedman [61] analyzed an eco-epidemic model where disease spreads not only in prey population but also in predator population. Lafferty [101] showed that predators consume infected prey if the cost of potential infection for the predator is low and catchability of prey is high. So predation on infected prey can decrease the disease virulence. Thus, infectious disease is an important factor to regulate human and animal population size.

The study of the consequences of hiding behavior of prey on the dynamics of predator-prey interactions can be recognized as a major issue in applied mathematics and theoretical ecology [65, 64, 122, 67, 124]. According to our knowledge, one of the founders of population ecology, Gause et al. [57] first introduced the prey- predator model with the refuge region. Later on Krivan [98] reconsidered the Gause's model and described the model which was ill-posed. Many researchers [138, 98, 160, 72, 97, 130, 60, 77, 33, 137, 139] have discussed the influence of prey refuge and they concluded that the prey refuge has a stabilizing effect on the predator-prey interaction and also prey species can be protected from extinction by using such type of policy.In this chapter, we propose a prey-predator model where prey is infected by a micro parasite. Refuge strategy is adopted by the population which are attacked by the predator. We have studied the effect of constant number of prey using refuges. The main question of the chapter is: Do

refuges promote the community stability of predator-prey dynamics?

3.2 Model formulation

We consider a habitat where prey and predator populations are living together and prey population is infected by a micro parasite. Naturally prey whether infected or not may avoid being killed by their predators. So they defend themselves by making refuges in different ways. Prey refuges are expected to affect population dynamics, but direct experimental tests of this hypothesis are scarce. Larvae of western flower thrips *Frankliniella occidentallis* use the web produced by spider mites as a refuge from predation by the predatory mite *Neoseiulus cucumeris*. Generally prey refuge enhance the growth rate of prey and decrease the growth rate of predator. A lot of studies shows that predators consume a disproportionate number of prey infected by parasites [168]. Many examples can be found in [74] where the parasite changes the behavior of the prey, so that infected prey are more vulnerable to predation. On the other hand infected prey are more weak than susceptible prey and less active, so that they can be caught more easily [43, 131]. This type of scenario observed in many laboratory and field observations [88, 87, 110, 44]. Here we shall consider the particular case when predator is a specialist i.e the prey population constitutes its only food source. It is to be noted here that such a modelling approach can be found in the literature. Our general model consists of three differential equations for the density of the prey S, infected prey I and predator P. The model can be formulated as :

$$\begin{aligned} \frac{dS}{dt} &= r - \beta SI - \alpha S \\ \frac{dI}{dt} &= \beta SI - c_1 f(I)P - \mu I \\ \frac{dP}{dt} &= P\Big(- d + c_2 f(I)\Big) \end{aligned} \tag{3.1}$$

$$\text{where, } f(I) = \begin{cases} \frac{I-m}{a+I-m} & \text{when } I > m \\ 0 & \text{when } I \leq m \end{cases}$$

with initial conditions $S(0) > 0, I(0) \geq 0, P(0) \geq 0$.

The first equation describes that in the absence of disease, the growth rate of the prey population is given by the solution

$$\frac{dS}{dt} = r - \alpha S, \tag{3.2}$$

where r is the recruitment rate of the prey population (including newborn and migratory) [50] and α denotes the natural death rate of prey population. In this work we have consider recruitment rate is prey density independent which means it limits the prey population growth rate. However, it may be prey density dependent but dynamics remains unchanged. We assumed that the disease is not genetically inherited. The infected population do not recover or become immune. The incidence assumed to be bilinear incidence βSI, with β as the transmission rate.

The second equation of model equation (3.1) represents the evolution of infected populations. The only positive contribution comes from susceptible class which are infected. The infected population is removed by a natural death rate μ or by predation before having the possibility of reproducing. The infected prey population is more vulnerable than the susceptible prey as they are very weak and they can be caught more easily than the healthy prey. Due to the high predation pressure infected prey population hide themselves at a constant rate m. We assume that predator population consumes infected prey with Holling type II functional response which is

$$f(I) = \frac{I - m}{a + I - m}, \tag{3.3}$$

where a is half saturation constant (means the concentration supporting half the maximum uptake rate). When $I \leq m$ predator population dies exponentially and in this case $f(I) = 0$. In the last equation when there is no prey, predator population suffers from a natural death rate d. In second term of the last equation c_2 be the conversation efficiency of the predator.

Table 3.1: Parameters of system (3.4) and their units

Parameter	Definition	Units
r	recruitment rate of the prey population including immigrants and the new Born's that are assumed to be susceptible	time^{-1}
m	constant quantities of prey using refuges	number
α	natural death rate of susceptible prey	time^{-1}
μ	natural death rate of infected prey	time^{-1}
d	natural death rate of predator	time^{-1}
c_1	predation coefficient	$\text{mass}^{-1}\text{time}^{-1}$
c_2	conversion efficiency of the predator	time^{-1}
a	half saturation constant	number
β	transmission coefficient	$\text{mass}^{-1}\text{time}^{-1}$

Thus when $I > m$, model (3.1) becomes

$$\begin{aligned} \frac{dS}{dt} &= r - \beta SI - \alpha S \\ \frac{dI}{dt} &= \beta SI - \frac{c_1(I-m)P}{a+I-m} - \mu I \\ \frac{dP}{dt} &= P\left(-d + \frac{c_2(I-m)}{a+I-m}\right) \end{aligned} \tag{3.4}$$

All the model parameters and their units are interpreted in Table 3.1.

3.3 Boundedness

In this section, we first study positivity and boundedness of solutions of system (3.4). For biological validity of system, it is essential to prove that all solutions of system (3.4) with positive initial data will remain positive for all time $t > 0$.

Lemma 3.3.1. *All solutions $(S(t), I(t), P(t))$ of system (3.4) which start in $\mathbb{R}^3_+$, remain positive for all $t > 0$.*

Proof. From first subequation of system (3.4) we obtain,

$$\frac{dS(t)}{dt} > -(\beta I + \alpha)S(t).$$

Thus,

$$S(t) > S(0)\exp\{\int_0^t [-(\beta I(s) + \alpha)]ds\} > 0. \tag{3.5}$$

Similarly,

$$I(t) > I(0)\exp\left\{\int_0^t \left[\beta P(s) - \frac{c_1 P(s)}{a + I(s) - m} - \mu\right]ds\right\} > 0, \tag{3.6}$$

and

$$P(t) = P(0)\exp\left\{\int_0^t \left[-d + \frac{c_2(I(s) - m)}{a + I(s) - m}\right]ds\right\} > 0 \tag{3.7}$$

with $S(0), I(0), P(0) > 0$.

Lemma 3.3.2. *All solutions of system (3.4) that initiate in $\mathbb{R}^3_+$, enter the region $B = \{(S, I, P) \in \mathbb{R}^3_+, S > 0, I > m, P > 0 : W(t) = S + I + \frac{c_1}{c_2}P \leq \frac{r}{v}\}$ as $t \to \infty$, where v=min$\{\alpha, \mu, d\}$.*

Proof.

Assume, $\{S(t), I(t), P(t)\}$ be any solution of system (3.4). Define $W(t) = S + I + \frac{c_1}{c_2}P$. Differentiating W along the solutions of system (3.4), we get

$$\frac{dW}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{c_1}{c_2}\frac{dP}{dt}. \tag{3.8}$$

With the help of system (3.4) we get,

$$\frac{dW}{dt} = r - \left(\alpha S + \mu I + dP\frac{c_1}{c_2}\right) \leq r - vW, \text{ where, } v = \min\{\alpha, \mu, d\}.$$

By using differential inequality [18], we get,

$$W(t) \leq \frac{r}{v} + ce^{-vt} \tag{3.9}$$

c be any arbitrary positive constant. Therefore we have $W(t) \leq \frac{r}{v}$ as $t \to \infty$.

Therefore, all the solutions of (3.4) that initiate in $\mathbb{R}^3_+$ will ultimately remain in the region $B = \{(S, I, P) \in \mathbb{R}^3_+ : W(t) \leq \frac{r}{v}\}$. Hence, the lemma is proved.

3.4 Equilibria

In this section, we discuss the existence and stability behaviour of system (3.4) at various equilibrium points. The system (3.4) has following equilibria.

1. The axial equilibrium point : $E_1(\frac{r}{\alpha}, 0, 0)$ which is not in B.
2. One predator free equilibrium : $E_2(\overline{S}, \overline{I}, 0)$.

Here $\overline{S}$ and $\overline{I}$ are the positive solutions of the following algebraic equations:

$$\begin{aligned} r - \beta SI - \alpha S &= 0, \\ (\beta S - \mu)I &= 0. \end{aligned} \tag{3.10}$$

Solving (3.10), we get $\overline{S} = \frac{\mu}{\beta}, \overline{I} = \frac{r\beta - \alpha\mu}{\mu\beta}$.

E_2 is feasible, provided $r > \frac{\alpha\mu}{\beta}$, that means the recruitment rate of the susceptible prey exceeds some threshold value which depends on the death rate of the infected prey.

3. One coexistence equilibrium point : $E^*(S^*, I^*, P^*)$

Here S^*, I^* and P^* is the positive solution of the system of algebraic equations

given below:

$$\begin{aligned} r - \beta SI - \alpha S &= 0, \\ \beta SI - \frac{c_1(I-m)P}{a+I-m} - \mu I &= 0, \\ -d + \frac{c_2(I-m)}{a+I-m} &= 0. \end{aligned} \tag{3.11}$$

Solving (3.11), we get

$$S^* = \frac{r(c_2-d)}{(\beta m+\alpha)(c_2-d)+ad\beta},$$

$$I^* = m + \frac{ad}{c_2-d},$$

and

$$P^* = \frac{c_2}{c_1 d}\left(\frac{\beta r(c_2-d)}{(\beta m+\alpha)(c_2-d)+ad\beta} - \mu\right)\left(\frac{m(c_2-d)+ad}{c_2-d}\right).$$

Clearly, the interior equilibrium point E^* is feasible provided $c_2 > d$ and $r > \frac{ad\beta\mu+(\beta m+\alpha)(c_2-d)\mu}{\beta(c_2-d)} = \hat{r}(say)$.

3.4.1 Dynamical behaviour

Here, we investigate the local behaviour of system (3.4) around the steady states defined above. The Jacobian matrix of system (3.4) at any arbitrary point (S, I, P) is given by

$$J(S,I,P) = \begin{pmatrix} -\beta I - \alpha & -\beta S & 0 \\ \beta I & \beta S - \mu - \frac{ac_1P}{(a+I-m)^2} & -\frac{c_1(I-m)}{a+I-m} \\ 0 & \frac{ac_2P}{(a+I-m)^2} & -d + \frac{c_2(I-m)}{a+I-m} \end{pmatrix}.$$

At the predator free equilibrium $E_2(\overline{S}, \overline{I}, 0)$, the Jacobian matrix is

$$J(E_2) = \begin{pmatrix} -\frac{\beta r}{\mu} & -\alpha & 0 \\ \frac{r\beta-\alpha\mu}{\mu} & 0 & -\frac{c_1(r\beta-\alpha\mu-m\mu\beta)}{(a-m)\mu\beta+r\beta-\alpha\mu} \\ 0 & 0 & -d - \frac{c_2(r\beta-\alpha\mu-m\mu\beta)}{(a-m)\mu\beta+r\beta-\alpha\mu} \end{pmatrix}$$

for which two of the eigenvalues are negative real part and one is positive whenever $d < \frac{c_2(\bar{I}-m)}{a+\bar{I}-m}$. So E_2 is attractive along S and I direction and repulsive in the P direction when $d < \frac{c_2(\bar{I}-m)}{a+\bar{I}-m}$.

Thus predator free equilibrium point E_2 is locally stable when $d > \frac{c_2(\bar{I}-m)}{a+\bar{I}-m}$. As stability of the boundary point implies extinction of the predator populations, so the infected prey refuge size plays a major role for existence of the predator.

To show E_2 is globally asymptotically stable in the $S-I$ plane, we use Bendixson-Dulac criterion.

We define $H(S,I) = \frac{1}{I}$. Clearly $H > 0$ when $I > 0$. Let $g_1(S,I) = r - \beta SI - \alpha S$ and $g_2(S,I) = \beta SI - \mu I$. Then $\Delta(S,I) = \frac{\partial}{\partial S}(g_1 H) + \frac{\partial}{\partial I}(g_2 H) = -\beta - \frac{\alpha}{I} < 0$. As E_2 is locally asymptotically stable so by Bendixson-Dulac criterion it is globally asymptotically stable.

Now we state the following theorem to study the stability characteristics around the coexistence equilibrium E^*.

Theorem 3.4.1. *Suppose $m > \frac{ad^2}{(c_2-d)^2}$. Then interior equilibrium point E^* is locally asymptotically stable.*

Proof. The Jacobian matrix at the endemic equilibrium E^* is given by

$$J(E^*) = \begin{pmatrix} u_{11} & u_{12} & 0 \\ u_{21} & u_{22} & u_{23} \\ 0 & u_{32} & 0 \end{pmatrix}$$

where, $u_{11} = -\beta I^* - \alpha$, $u_{12} = -\beta S^*$, $u_{21} = \beta I^*$, $u_{22} = \beta S^* - \mu - \frac{ac_1 P^*}{(a+I^*-m)^2}$, $u_{23} = -\frac{c_1(I^*-m)}{a+I^*-m}$, $u_{32} = \frac{ac_2 P^*}{a+I^*-m}$.

The characteristic equation of the Jacobian matrix around E^* is given by

$$\lambda^3 + p_1\lambda^2 + p_2\lambda + p_3 = 0 \tag{3.12}$$

Table 3.2: Existence conditions and properties of the equilibrium points of system (3.4)

Equilibrium	Properties	Conditions
E_1	is not in Ω	-
E_2	locally asymptotically stable	$d > \frac{c_2(\bar{I}-m)}{a+\bar{I}-m}$
E_2	unstable	$d < \frac{c_2(\bar{I}-m)}{a+\bar{I}-m}$
E^*	locally asymptotically stable	$p_1p_2 - p_3 > 0$

$$\text{where} \begin{cases} p_1 = -(u_{11} + u_{22}) \\ p_2 = u_{11}u_{22} - u_{23}u_{32} - u_{12}u_{21} \\ p_3 = u_{11}u_{23}u_{32} > 0. \end{cases}$$

Assumption of the theorem implies that $u_{22} > 0$ and hence $p_1 > 0$. Positivity of p_2 and p_3 is obvious. Again

$$p_1p_2 - p_3 = (u_{12}u_{21} - u_{11}u_{22})(u_{11} + u_{22}) + u_{22}u_{23}u_{32} > 0.$$

Thus the result follows by the application of Routh-Hurwitz criterion (Table 3.2).

Next, we will study the Hopf-bifurcation of the above system. Bifurcation analysis deals with structurally unstable system. This subject is a branch of mathematics that ensures a certain qualitative changes of the dynamical systems with respect to some parameters. A small change in parameter creates a topological transform.

Our main aim is to check whether Hopf-bifurcation occur or not. Hopf bifurcation ensures the stability change that means either disease may be endemic or fluctuating or controlled. We identify the parameter m which has a main role in changing the dynamics. The next theorem ensures appearance of limit cycles through Hopf bifurcation.

Theorem 3.4.2. *Suppose $c_2 > d$, $r > \widehat{r}$. Then system (3.4) undergoes a Hopf bifurcation when the number of prey refuge m crosses a critical value $m = m^*$.*

Proof: The characteristic equation of the Jacobian matrix around E^* is given by

$$\lambda^3 + p_1\lambda^2 + p_2\lambda + p_3 = 0.$$

Now we choose bifurcation parameter $m = m^*$ such that $p_1p_2 = p_3$. From (3.12), we have the characteristic equation must be of the form

$(\lambda^2 + p_2)(\lambda + p_1) = 0$ whose roots are $\lambda_1 = i\sqrt{p_2}, \lambda_2 = -i\sqrt{p_2}, \lambda_3 = -p_1$

For all m, the roots of the characteristic equation are of the form $\lambda_1(m) = u(m) + iv(m), \lambda_2(m) = u(m) - iv(m)$, and $\lambda_3(m) = -u(m)$

Now we will verify the transversality condition.

$$\left[\frac{d(Re\lambda_j(m))}{dm}\right]_{m=m^*} \neq 0, \qquad j = 1, 2.$$

Substituting, $\lambda(m) = u(m) + iv(m)$ in equation (3.12), we get

$$(u + iv)^3 + p_1(u + iv)^2 + p_2(u + iv) + p_3 = 0.$$

Separating real and imaginary parts, we get,

$$u^3 - 3uv^2 + p_1(u^2 - v^2) + p_2u + p_3 = 0. \tag{3.13}$$

$$3u^2v - v^3 + 2p_1uv + p_2v = 0. \tag{3.14}$$

From (3.14), as $v \neq 0$, let

$$f(u) = v^2 = 3u^2 + 2p_1u + p_2. \tag{3.15}$$

Now put the value of v^2 in equation (3.13), we get,

$$u^3 - 3uf(u) + p_1(u^2 - f(u)) + p_2u + p_3 = 0. \tag{3.16}$$

Now differentiating (3.16) with respect to m we get,

$$3u^2\frac{du}{dm}-3uf'(u)\frac{du}{dm}-3f(u)\frac{du}{dm}+\frac{dp_1}{dm}(u^2-f(u))+p_1(2u-f'(u))\frac{du}{dm}+u\frac{dp_2}{dm}+p_2\frac{du}{dm}+\frac{dp_3}{dm}=0$$

Now $u(m^*)=0$ so, from equation (3.16) we get, $f(0)=p_2, p'(0)=2p_1$.

We have, $\left.\frac{du}{dm}\right|_{m=m^*}=\frac{1}{2(p_1^2+p_2)}\left[\frac{dp_3}{dm}-p_2\frac{dp_1}{dm}\right]<0.$

Hence interior equilibrium point E^* is unstable, when $m<m^*$ and stable when $m>m^*$. Thus Hopf bifurcation occurred at $m=m^*$.

3.4.2 Existence of transcritical bifurcation

To investigate local bifurcation around the equilibrium points of system (3.4), we mainly use Sotomayor's theorem [161]. Applicability of Sotomayor's theorem requires one of the eigenvalues of the variational matrix at the bifurcating equilibrium point must be zero.

$\frac{dX}{dt}=F(X)$ where $X=(S,I,P)^t$ and $F=(F_1,F_2,F_3)$ where

$$F_1=r-\beta SI-\alpha S,\ F_2=\beta SI-\frac{c_1(I-m)P}{a+I-m}-\mu I,\ F_3=P\left(-d+\frac{c_2(I-m)}{a+I-m}\right)$$

Then according to variational matrix of system (3.4), we obtain the following for non-zero vector $V=(v_1,v_2,v_3)^t$:

$$D^2F(S,I,P)(V,V)=\frac{\partial^2F}{\partial S^2}v_1^2+\frac{\partial^2F}{\partial S\partial I}v_1v_2+\frac{\partial^2F}{\partial I\partial S}v_2v_1+\frac{\partial^2F}{\partial I^2}v_2^2+\frac{\partial^2F}{\partial S\partial P}v_1v_3+\frac{\partial^2F}{\partial P\partial S}v_3v_1+\frac{\partial^2F}{\partial P^2}v_3^2+\frac{\partial^2F}{\partial I\partial P}v_2v_3+\frac{\partial^2F}{\partial P\partial I}v_3v_2.$$

Now we get after a simple calculations,

$$D^2F(S,I,P)(V,V)=\begin{pmatrix}-2\beta v_1v_2\\ 2\beta v_1v_2-\frac{2ac_1}{(a+I-m)^2}v_2v_3\\ \frac{2ac_2}{(a+I-m)^2}v_2v_3\end{pmatrix}$$

and

$$D^3F(S,I,P)(V,V,V) = \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix}.$$

Thus according to Sotomayor's theorem system (3.4) has no pitchfork bifurcation. In the following theorem, we discuss about local bifurcation near the equilibrium points.

Theorem 3.4.3. *System (3.4) at the axial equilibrium point E_2 undergoes a transcritical bifurcation but no saddle-node bifurcation when the bifurcation parameter d passes through the critical value $d^* = \frac{c_2(\bar{I}-m)}{a+\bar{I}-m}$.*

Proof. If we take $d = d^* = \frac{c_2(\bar{I}-m)}{a+\bar{I}-m}$, then one of eigenvalues of the variational matrix $V(E_2)$ will be zero. Now, $V(E_2)$ with zero eigenvalue is given by

$$V(E_2) = \begin{pmatrix} -\frac{r\beta}{\mu} & -\alpha & 0 \\ \frac{r\beta-\alpha\mu}{\mu} & 0 & \frac{c_1(r\beta-\alpha\mu-m\mu\beta)}{(a-m)\mu\beta+r\beta-\mu\alpha} \\ 0 & 0 & 0 \end{pmatrix}.$$

Let, $V = (v_1, v_2, v_3)^t$ be a eigenvector corresponding to the eigenvalue $\lambda = 0$. Thus $V = (v_1, -\frac{r\beta}{\alpha\mu}v_1, \frac{r\beta-\alpha\mu}{A\mu}v_1)^t$ where $A = \frac{c_1(r\beta-\alpha\mu-m\mu\beta)}{(a-m)\mu\beta+r\beta-\mu\alpha}$. Also, assume $W = (w_1, w_2, w_3)^t$ represents the corresponding eigenvector of $V(E_2)^t$ to the eigenvalues of $\lambda = 0$. Hence from $V(E_2)^tW = 0$ we get, $W = (0, 0, w_3)^t$. Now $F_d(E_2, d^*) = (0, 0, 0)^t$, here $F_d(E_2, d^*)$ is the derivative of $F = (F_1, F_2, F_3)^t$ with respect to d. Then we obtain $W^t[F_d(E_2, d^*)] = 0$.

Thus by the application of Sotomayor's theorem, system (3.4) has no saddle-node bifurcation near $d = d^*$.

Again

$$DF_d(E_2, d^*) = \begin{pmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & -1 \end{pmatrix}$$

Then, $W^t[DF_d(E_2, d^*)V] = -v_3w_3 \neq 0$.

Now

$$D^2F(E_2,d^*)(V,V)=\begin{pmatrix} -2\beta v_1v_2 \\ 2\beta v_1v_2-\frac{ac_1}{(a+I-m)^4}v_2^2 \\ -\frac{ac_2P}{(a+I-m)^4}v_2^2+2\frac{ac_1}{(a+I-m)^2}v_2v_3 \end{pmatrix}.$$

Therefore, $W^t[D^2F(E_2,d^*)(V,V)]=\frac{2ac_1}{(a+I-m)^2}v_2v_3w_3>0$.

Therefore, system (3.4) has a transcritical bifurcation near E_2 when the bifurcation parameter d passes the critical value d^*. Furthermore, as the characteristic equation of $V(E_2)$ does not contain any purely imaginary eigenvalues, so no Hopf bifurcation can occur there.

These bifurcations are ecologically important and can lead to potentially dramatic shifts to the system dynamics. Transcritical bifurcation transforms the predator free equilibrium point into a unstable equilibrium point .

Remark 3.4.1. *As in equation (3.12), $P_3>0$, so there does not exist any local bifurcation near the equilibrium point E^*.*

3.5 Uniform persistence

To prove persistence of a system one has to verify the invasion condition at the boundary states which consists only of equilibria. Clearly our system admits only one equilibrium point namely E_2. As there is no limit cycles around this equilibrium point thus we have to check the invasion condition at E_2. Species coexistence of system (3.4) requires that missing species here in particular predator must invade the equilibrial community of the remaining two species. Here we observe that if death rate of the predator d is less than a certain threshold value $\frac{c_2(\bar{I}-m)}{a+\bar{I}-m}$, than the predator can invade into the system. Thus all the three species coexist if the above condition hold.

Theorem 3.5.1. *Let the assumption of Theorem 3.4.1 be hold. Then system (3.4) is uniformly persistent provided $d<\frac{c_2(\bar{I}-m)}{a+\bar{I}-m}$.*

Proof. To prove the theorem we use the 'average Lyapunov function' and apply the theorem by Hutson [82]. Choose an average Lyapunov function of the form

$Q(x) = S^{r_1} I^{r_2} P^{r_3}$, where each $r_i, i = 1, 2, 3$ is assumed positive. In the interior of $\mathbb{R}^3_+$ we can get,

$$\begin{aligned}\frac{1}{Q(X)}\frac{dQ(X)}{dt} &= \phi(x)\\ &= \frac{r_1}{S}\frac{dS}{dt} + \frac{r_2}{I}\frac{dI}{dt} + \frac{r_3}{P}\frac{dP}{dt}\\ &= r_1\{\frac{r}{S} - \beta I - \alpha\} + r_2\{\beta S - \frac{c_1(I-m)P}{I(a+I-m)}\} + r_3\{-d + \frac{c_2(I-m)}{a+I-m}\}.\end{aligned}$$

We have to show $\phi(x) > 0$ for all $x \in bd\mathbb{R}^3_+$, for a suitable choice of $r_1, r_2, r_3 > 0$ to prove uniform persistence of system (3.4). So one has to satisfy the following conditions corresponding to the boundary equilibria E_2 only:

$$E_2 : -d + \frac{c_2(\bar{I} - m)}{a + \bar{I} - m} > 0. \tag{3.17}$$

Positivity of (3.17) follows from the assumption of the theorem. This completes the proof of the theorem.

Remark 3.5.1. *If the above condition is violated then predator population goes to extinction.*

Again since system (3.4) is uniformly persistent therefore there exists a time T such that $S(t), I(t), P(t) > \tilde{a}(0 < \tilde{a} < 1)$ for $t > T$.

3.6 The influence of prey refuge

In this following section, we shall discuss the influence of infected prey refuge on each population when the interior equilibrium point exists and stable. It is easy to see that $S^*, I^*.P^*$ are all continuous differential functions of parameter m and

$$\frac{dS^*}{dm} = -\frac{r\beta(c_2 - d)^2}{ad\beta + (\alpha + \beta m)(c_2 - d)} < 0,$$

$$\frac{dI^*}{dm} = 1 > 0,$$

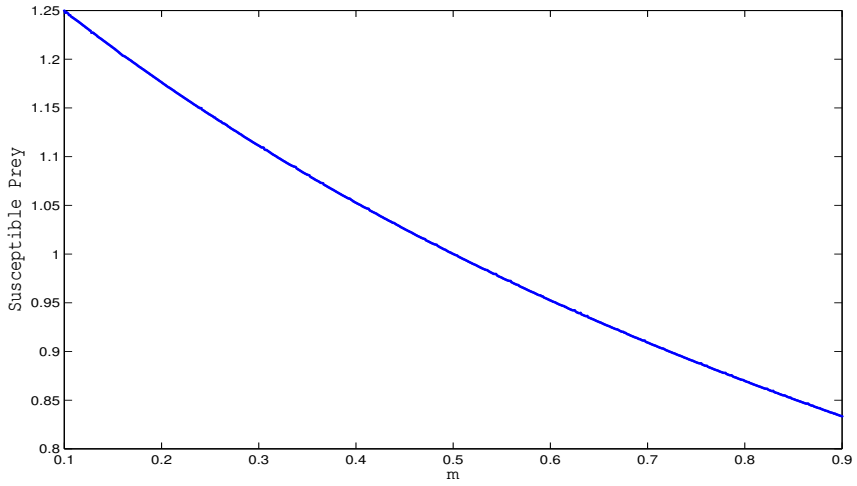
and

$$\frac{dP^*}{dm} = \frac{c_2(c_2 - d)}{c_1 d}\left[\frac{r\alpha\beta(c_2 - d)}{\{(\alpha + \beta m)(c_2 - d) + ad\beta\}^2} - \frac{\mu}{(c_2 - d)}\right].$$

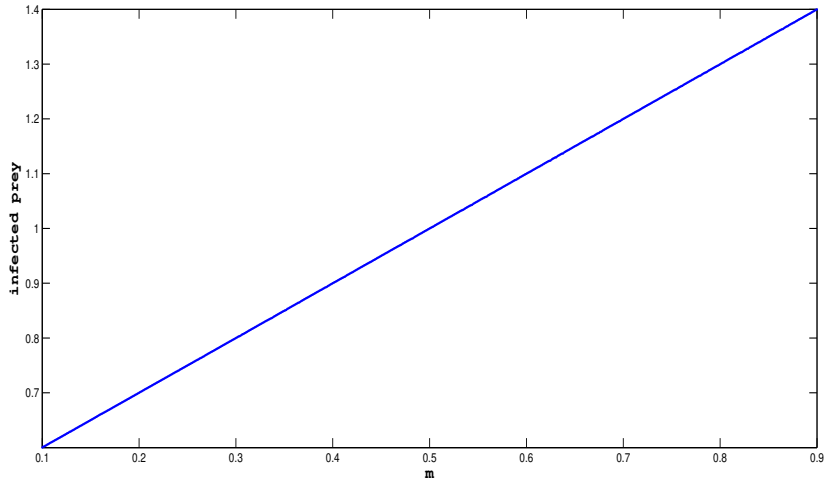
In the above analysis, we clearly see that S^* is strictly decreasing function of parameter m and increasing the amount of infected prey refuge leads to the decreasing the density of the susceptible prey population. I^* is strictly increasing function of parameter m and increasing the amount of infected prey refuge leads to the increasing the density of the infected prey species. The presence of negative term in the third equation indicates that increasing the amount of infected prey refuge may decrease the predator density when the interior equilibrium point is exist and stable.

3.6.1 Numerical example for influence of prey refuge

Here we choose a set of parameter as follows $r = 2, \alpha = 1, \beta = 1, c_1 = 2, c_2 = 1.5, \mu = 0.5, d = 0.5, a = 1$, when interior equilibrium point E^* is locally asymptotically stable (see Fig.(3.1) and Fig.(3.2)).



(a)



(b)

Figure 3.1: Influence of prey refuge on (a) susceptible prey population and (b) infected prey population

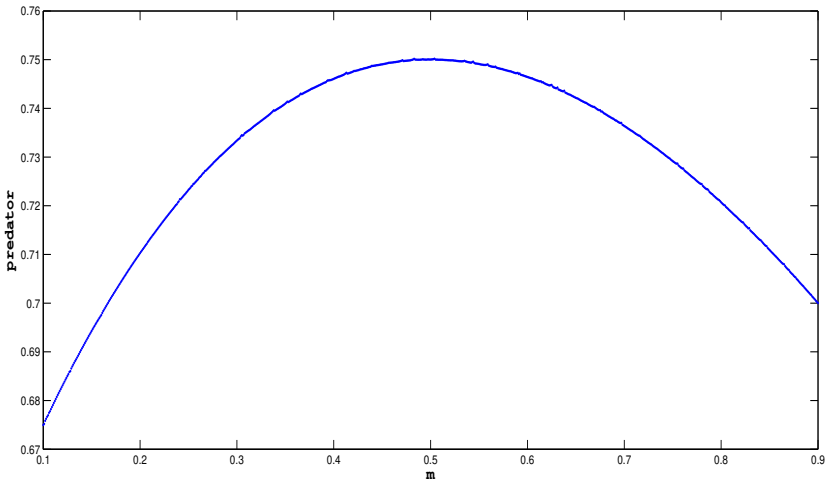


Figure 3.2: Influence of prey refuge on predator population

3.7 Global stability analysis

In this section, we analyse the global stability of system (3.4) when it is locally asymptotically stable. By constructing a suitable Lyapunov function to prove the global stability of a system is a more general and common method. In our system (3.4) it is quite difficult to construct such type of Lyapunov functions to prove the global stability and it is a fact that there is no general approach to construct them. Therefore, to analyse the global stability of the positive equilibrium of system (3.4), we use the high-dimensional Bendixson criterion as developed in Li and Muldowney [109], which we discuss in next.

Consider the open set $G \subset \mathbb{R}^n$. Let the differential equation be

$$\frac{dx}{dt} = f(x), \tag{3.18}$$

where the function $f : x \to f(x) \in \mathbb{R}^n, x \in G$ is continuous in G.

Denote J be an $n \times n$ matrix and $J^{[2]}$ be the second additive compound matrix of J. If $J = (a_{ij})_{3\times 3}$, then

$$J^{[2]} = \begin{pmatrix} a_{11} + a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11} + a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22} + a_{33} \end{pmatrix}.$$

To obtain a high-dimensional Bendixson criterion, one has to show that the second compound equation

$$\frac{dZ}{dt} = \frac{\partial f}{\partial x}(x(t, x_0))Z(t) \tag{3.19}$$

with respect to a solution $x(t, x_0) \in G$ of system (3.18) is equi-uniformly asymptotically stable, namely, for each $x_0 \in G$, system (3.19) is uniformly asymptotically stable, and the exponential decay rate is uniform for x_0 in each compact subset of G, where $G \subset \mathbb{R}^n$ is an open connected set. Thus it does not admit

any invariant simple closed rectifiable curve, including periodic orbits, homoclinic orbits, hetroclinic cycles etc.

Now we require a Lemma to prove the global stability of the positive equilibrium E^* which is already is used in [162].

Lemma 3.7.1. *[109]. $G \subset \mathbb{R}^n$ be a simply connected region. Assume that the family of linear systems (3.19) is equi-uniformly asymptotically stable. Then*

(i) G contains no simple closed invariant curves, including periodic orbits, homoclinic orbits, hetroclinic cycles;
(ii) each semi-orbit in G converges to a single equilibrium.

In particular, if G is positively invariant and contains a unique equilibrium $\bar{E}$, then $\bar{E}$ is globally asymptotically stable in G.

Theorem 3.7.1. *Assume that $\mu + d + c_1\tilde{a} > c_2 + \frac{r\beta}{v}$. If there exist positive numbers ω and θ such that $\max\{c_{11} + c_{12}\omega, \frac{c_{21}}{\omega} + c_{22} + \frac{c_{23}}{\theta}, c_{32} + c_{33}\theta\} < 0$, where $c_{11} = -\alpha - \mu + \frac{r\beta}{v} - \beta\tilde{a} - ac_1\tilde{a}$, $c_{12} = -c_1$, $c_{22} = -\alpha - d - \beta\tilde{a} + c_2$, $c_{32} = \frac{\beta r}{v}$, $c_{33} = -\mu - d + \frac{r\beta}{v} - c_1\tilde{a} + c_2$, then the positive equilibrium point E^* of system (3.4) is globally asymptotically stable.*

Proof. From system (3.4), the second compound matrix can be written as

$$\frac{\partial F^{[2]}}{\partial X} = \begin{pmatrix} b_{11} & b_{12} & b_{13} \\ b_{21} & b_{22} & b_{23} \\ b_{31} & b_{32} & b_{33} \end{pmatrix}$$

where

$$\begin{aligned} b_{11} &= \beta S - \beta I - \alpha - \mu - \frac{ac_1P}{(a+I-m)^2},\ b_{12} = -\frac{c_1(I-m)}{a+I-m},\ b_{13} = 0, \\ b_{21} &= \frac{ac_2P}{(a+I-m)^2},\ b_{22} = -\beta I - \alpha - d + \frac{c_2(I-m)}{a+I-m},\ b_{23} = -\beta S, \\ b_{31} &= 0,\ b_{32} = \beta I,\ b_{33} = \beta S - \mu - d - \frac{c_1P}{(a+I-m)^2} + \frac{c_2(I-m)}{a+I-m}. \end{aligned} \tag{3.20}$$

The second compound system $\frac{dZ}{dt} = \frac{\partial F^{[2]}}{\partial X} Z(t)$ then becomes

$$\begin{aligned}
\dot{z_1} &= [\beta S - \beta I - \alpha - \mu - \frac{ac_1P}{(a+I-m)^2}]z_1 - \frac{c_1(I-m)}{a+I-m}z_2,\\
\dot{z_2} &= \frac{ac_2P}{(a+I-m)^2}z_1 + [-\beta I - \alpha - d + \frac{c_2(I-m)}{a+I-m}]z_2 - \beta S z_3,\\
\dot{z_3} &= \beta I z_2 + [\beta S - \mu - d - \frac{c_1P}{(a+I-m)^2} + \frac{c_2(I-m)}{a+I-m}]z_3.
\end{aligned} \tag{3.21}$$

Now, set $W(Z) = \max\{\omega|z_1|, |z_2|, \theta|z_3|\}$.

Direct calculations lead to the following inequalities:

$$\begin{aligned}
\tfrac{d^+}{dt}\omega|z_1| &\le c_{11}\bar{\omega}|z_1| + c_{12}\bar{\omega}|z_2|,\\
\frac{d^+}{dt}|z_2| &\le \frac{c_{21}\omega}{\omega}|z_1| + c_{22}|z_2| + \frac{c_{23}}{\theta}\theta|z_3|,\\
\tfrac{d^+}{dt}\theta|z_3| &\le c_{32}|z_2| + c_{33}\theta|z_3|
\end{aligned} \tag{3.22}$$

in which $\frac{d^+}{dt}$ denotes the right hand derivative and

$$\begin{aligned}
c_{11} &= -\alpha - \mu + \tfrac{r\beta}{v} - \beta\tilde{a} - ac_1\tilde{a},\\
c_{12} &= -c_1, \quad c_{21} = \tfrac{ac_2r}{v}, \quad c_{22} = -\alpha - d - \beta\tilde{a} + c_2,\\
c_{23} &= -\beta\tilde{a}, \quad c_{32} = \frac{\beta r}{v}, \quad c_{33} = -\mu - d + \frac{r\beta}{v} - c_1\tilde{a} + c_2.
\end{aligned} \tag{3.23}$$

Therefore $\frac{d^+}{dt}W(Z(t)) \le \phi W Z(t)$,

with $\phi = \max\{c_{11} + c_{12}\omega, \frac{c_{21}}{\omega} + c_{22} + \frac{c_{23}}{\theta}, c_{32} + c_{33}\theta\}$.

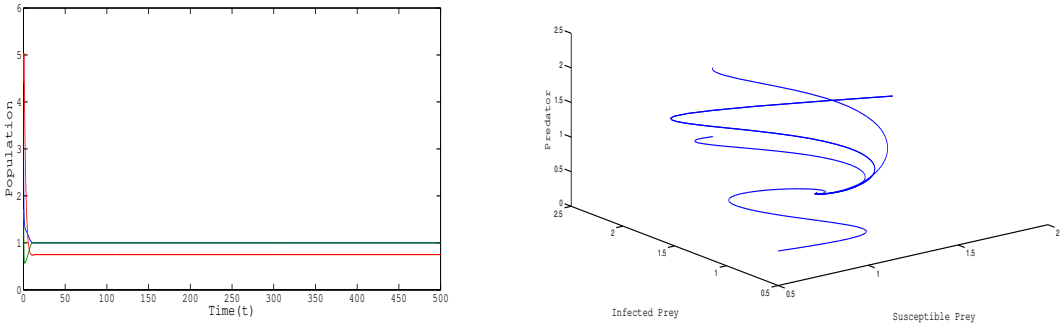
Thus, by the assumption of the theorem and by the boundedness of the solution of system (3.4), we find a positive constant ξ such that $\phi \le -\xi < 0$, and hence $W(Z(t)) \le W(Z(s))\exp(-\xi(t-s)), t \ge s > 0$.

This guarantees the equi-uniform asymptotic stability of the second compound system, and so global stability of the positive equilibrium E^* follows from the Lemma 3.7.1.

3.8 Numerical simulation

In this section, we numerically simulate the dynamics of the deterministic model system (3.4) around the positive interior steady state for a large range of parameter values. In most of the eco-epidemiological studies the impact of refuge plays a major role in describing the dynamics of the system. Here m is the main parameter controlling the system behavior. We varied the parameter m and observe different type of behavior of system (3.4). The parameter values of the system are r=2, $\alpha = 1$, $\beta = 1$, $c_1 = 2$, $c_2 = 1.5$, $\mu = .5$, $d = .5$, a=1, m=.5. Then system (3.4) has an equilibrium point (1,1,0.75) and conditions of Theorem (3.4.1) and Theorem (3.5.1) are satisfied. Hence system (3.4) is locally asymptotically stable and persistent. Phase diagram is shown in Fig.3.3.

Next, we take the parameter set of system (3.4) are r=11, $\alpha = 1$, $\beta = 1$,

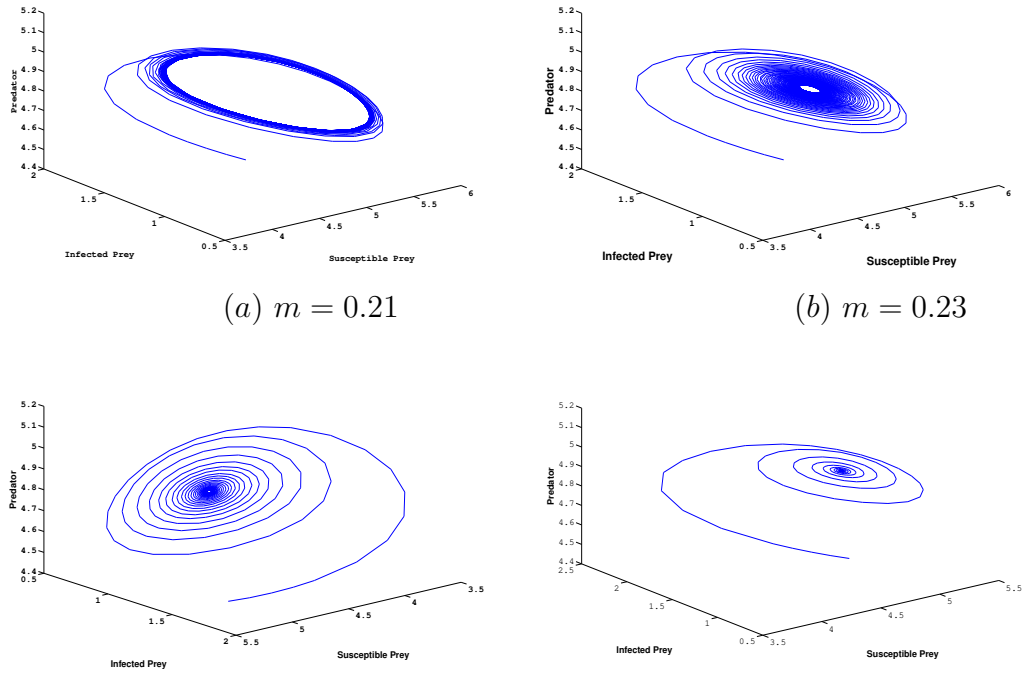


(a) (b)

FIGURE 3.3: (a) stable behavior of susceptible prey, infected prey and predator in time of model system (3.4), (b) stable phase portrait of system (3.4) around interior equilibrium point E^* and it is locally asymptotically stable.

$c_1 = 2$, $c_2 = 1$, $\mu = 1$, $d = .5$, a=1. Here m is a refuge parameter and it has a great impact in the dynamics of the system. We observed that, increasing the

amount of infected prey refuge can stabilizes the system (see Fig.(3.4)(a)-(d)). Low amount of refuge as well as predation process create instability of the system.



(a) $m = 0.21$ (b) $m = 0.23$

(c) $m = 0.25$ (d) $m = 0.30$

Figure 3.4: Phase portrait of system (3.4) for different values of m. Increasing the amount of infected prey refuge (m), the system changes its dynamical behavior from unstable to stable.

We also observed that the system undergoes a Hopf bifurcation taking m as a bifurcation parameter. If we choose $m = m^* = 0.225$ it is easy to see that system (3.4) has an equilibrium point (4.94,1.22,4.83). Then it follows from Theorem 3.4.2 that a Hopf bifurcation of periodic solution occurs at $m = m^* = 0.225$. When $m = 0.21$ the positive equilibrium point is unstable (see Fig 3.4(a)) and when $m = 0.25$ the positive equilibrium point is stable (see Fig 3.4(c)). To demonstrate the dynamical behaviors of system (3.4), we have plotted the bifurcation diagram of the system in (m, S, I) plane and its projections on (m, S) and (m, I) plane. Using Matlab we have obtained the following Figures (see Fig.3.5).

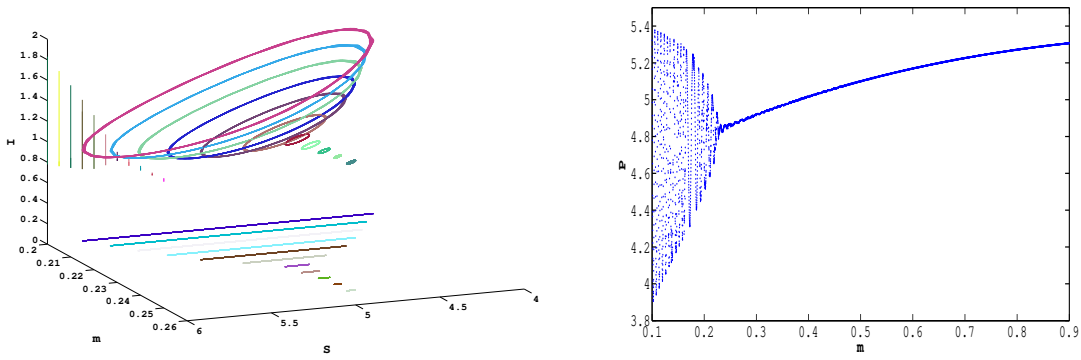


Figure 3.5: Left side represents bifurcation diagram of the system (3.4) with bifurcation parameter m in (m, S, I) space and its projections on (m, S) and (m, I) planes while the other side represents the same for P in (m, P) plane. These figures show that Hopf-bifurcation occurs at $m = m^* = 0.225$.

Again we choose r=2, $\alpha = .5$, $\beta = 1$, $c_1 = 2$, $c_2 = 1$, $\mu = .5$, $d = .5$, a=1, m=0.5, and $\tilde{a} = 3$. We see that conditions of Theorem 3.4.1 is satisfied, and hence system (3.4) is locally asymptotically stable. We now substitute these value along with $\omega = 1$ and $\theta = 2.5$ in equation (3.20) and we obtain $\phi = \max\{-8.000, -0.2000, -1.0000\} < 0$. Therefore, conditions of Theorem 3.7.1 is satisfied and hence the interior equilibrium point is globally asymptotically stable (see Fig.3.6).

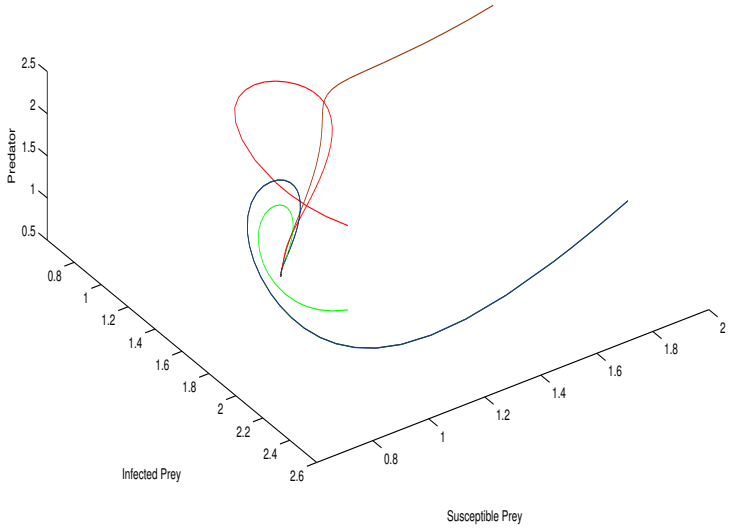


FIGURE 3.6: Interior equilibrium point E^* is globally asymptotically stable.

3.9 Discussion

In eco-epidemic model, it is well known fact that if the predator consumes the infected prey, it will allow the susceptible prey to survive but may drive the infected prey to extinction through over exploitation. Thus infected prey can decrease their predation risk by using refuge. A refuge can be useful for the biological control of the pest, however, it has a stabilizing effect on the system. The effect of constant proportion of refuge on eco-epidemic models has been studied in [157, 144, 91]. In their studies, presence of a constant proportion refuge acts on the system as an external decreasing of uptake rate and half saturation constant, that does not alter the dynamical behavior of the model. So inclusion of constant number of refuge is more appropriate rather than the constant proportion of refuge. This motivated us to incorporate constant number of prey refuge in our present system to make it more realistic from the ecological point of view.

The existence of refuges has an important role on the coexistence of prey and predator interaction. Several studies show that prey extinction can be prevented

by considering prey refuge [124, 160, 60]. It is shown that (in Lemma 3.3.1) system (3.4) is bounded which in turn implies that the system is biologically well behaved. The system can evolve only toward the predator free equilibrium E_2 and the coexistence equilibrium E^*. We investigated the stability of these equilibria by analytical approach. Local stability condition of the equilibrium E_2 indicates that disease cannot eradicate predator while refuge mechanism can do this. We note that coexistence is possible when disease affect the prey and refuge decreases the predation risk. The condition of Theorem 3.4.1 implies that if the prey refuge rate crosses a certain threshold value then the ecological system becomes more stable. From Theorem 3.7.1, we observed that if the disease transmission rate and conversion efficiency of the predator is small then positive equilibrium point is globally asymptotically stable that implies the disease is persist in the system. For disease eradication, such type of conditions should be avoided. We have observed transcritical bifurcation around the predator free equilibrium point E_2 when death rate of predator crosses a critical value d^*. Bifurcation analysis reveals that increasing amount of refuge stabilize the system.

The main novelty between our work and the other recent works in the inclusion of infected prey refuge in the system that allow some protection of the infected prey from extinction. This additional ecological component enriches the dynamics of the system and the model becomes realistic than the existing ones. The model analysis shows that refuge has a major impact on each population. Increasing the amount of infected prey refuge decrease the susceptible prey density whereas opposite hold for the infected prey density. Furthermore, predator density may decrease with the increase of infected prey refuge when the coexistence equilibrium point E^* is exists and stable.

However, our study also shows complex behavior of the proposed model. In particular, when the refuge capacity lies in a certain range, the periodic oscillation may appear. If this refuge rate exceeds the some threshold value, periodic solution disappears. Lastly, we infer that our model with infected prey refuge give rise to rich dynamics. Here we have assumed constant recruitment rate of the susceptible prey and infected prey population. These assumption can be modified in future work.

4

Deterministic and stochastic analysis of an eco-epidemiological model

4.1 Introduction

In the mid-1990s, CWD had been detected among free-ranging deer and elk in the region of northeastern Colorado and southeastern Wyoming [128]. According to the hunter-harvested animal surveillance, the overall prevalence of the disease in this area from 1996 through 1999 was estimated at approximately 5% in mule deer, 2% in white-tailed deer, and $< 1\%$ in elk [128]. This disease can be highly transmissible within captive deer and elk populations. A prevalence of $> 90\%$ was reported among mule deer in facilities where the disease has been endemic for > 2 years [181, 180]. The origin and transmission mechanism of the

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prions causing CWD is incompletely understood. Research on CWD epidemics in captive deer and elk has shown that CWD prions can remain infectious in the environment for at least two years [126] and thus it has the potential to disrupt the ecosystems where deer occur in abundance. Recent works on CWD can be found in [148, 142, 53]. Through numerical simulations, Hobbs [71] suggested that CWD can be removed from an infected elk population allowing selective predation by wolves. It was remarked in [13, 102] that increasing mortality rates in diseased population can delay disease transmission and decrease disease prevalence. This motivated Hobbs to modify the model of Miller et al. [125] by introducing density dependent effects on recruitment into the population, where the model is based on three approximations, the density of infected population, susceptible population and the mass of infectious material in the environment. Explicit form of predation term is not considered in [71]. It is well known fact that predation on a species subjected to an infectious can affect both the infection prevalence and population dynamics. As the disease is fatal and causes harms to deer populations, elk and many other cervids and at the present time, there is no known cure for the disease, and many aspects of the disease are still unclear so if we remove the infected animals from the population by predation then we can protect the animals from the disease. So appropriate eco-epidemiological model can give some insight of the disease dynamics. Research on eco-epidemic models [25, 172, 76, 183, 134, 135, 173] addressed on direct transmission but for indirect transmission on eco-epidemic model is not focused so much. Thus our main aim in this article to control CWD transmission through predation by developing eco-epidemic model with indirect transmission.

Another important fact that, population dynamics are often affected by environmental noise, which is an important component in ecosystem. There have been a growing interest to study how the noise affects the population dynamics. Hence stochastic differential equation models play a major role in the field of biology and ecology. In deterministic models, parameters are all deterministic irrespective of environmental fluctuation and hence they are very difficult to predict the future dynamics of the system correctly [12]. Therefore many authors introduced randomness in deterministic models to reveal the effect of environmental variability [12, 35, 149, 85, 24].

4.2 The eco-epidemic model

Chronic Wasting Disease (CWD) is a fatal disease of deer, elk and moose. Experimentally it can be shown that CWD can be transmitted to susceptible animals from residues of excreta left in the environment by infected animals and their carcasses which is more appropriate than traditional models of direct, animal-to-animal contact [143, 126]. Thorough observation of these diseases can be difficult because prolonged epizootic can result in low, usually undetected level of infection morbidity or mortality. To understand the incident and spatial dynamics of chronic wildlife disease requires long-term studies that may be difficult to carry out in natural population due to financial and logical constraints. Due to such complexity modeling disease dynamics may be the only practical way to measure the spatial and temporal patterns of chronic disease in wildlife. Its suggest alternative transmission mechanism and explore the spread of the disease. Lot of works have been done to modeling direct transmission, but despite the fact CWD prions can remain infectious in the environment for years, relatively little information exists about the potential effects of indirect transmission of CWD dynamics. So in the present study, we are consider a prey-predator system where the prey population is divided into two groups infected and non-infected and a third dynamic variable which represents the mass of infectious material in the environment. Our model is based on traditional susceptible-infected-recovered (SIR) models of disease transmission in humans [14, 46, 92]. The model is described by a system of four ordinary differential equations. The predator functional response is assumed to be Holling type-II. In view of above, we proposed the following model :

$$\begin{aligned}
\frac{dS}{dt} &= S\left[r\left(1-\frac{S+I}{k}\right)-\beta E\right] \\
\frac{dI}{dt} &= \beta SE-\mu I-\frac{aIP}{m+I} \\
\frac{dE}{dt} &= \varepsilon I-\tau E \\
\frac{dP}{dt} &= P\left(-d+\frac{bI}{m+I}\right)
\end{aligned} \tag{4.1}$$

with initial condition $S(0)>0, I(0)>0, E(0)>0, P(0)\geq 0$. Here, S denotes

the density of the susceptible animals and I be the infected ones. E be the mass of infectious material in the environment and P be the density of the predator.

The first equation describes a logistic growth of the prey, with per capita berth rate r and environmental carrying capacity k. Susceptible prey population become infected when it comes in contact with the infectious material in the environment. β denotes the indirect transmission coefficient for the disease.

The second equation represents the evolution of the infected populations. The only positive contribution comes from the susceptible class which are infected. Infected populations are affected by natural death at the rate μ. The infected prey is more vulnerable than susceptible prey as they are very weak and can be caught more easily than healthy prey. We assume that predator population consumes infected prey population with Holling type II functional response which is $\frac{aI}{m+I}$, where m is half saturation constant and a denotes the maximal predator per capita consumption rate i.e the maximum number of prey population can be eaten by a predator in each unit time.

The first term of third equation shows that deposition of infectious material through excretion by infected animals. The second term denote the loss of infectious material from the environment.

In the last equation when there is no prey, predator population suffers a natural death at rate d. In second term b represents the conversion efficiency of the predator. Our model is also applicable to propagation of other disease like tuberculosis in livestock [20], avian influenza [19], vibrio cholerae [89, 36], viral hepatitis A [5] etc. Biological sugnificance and units of the model parameters are given in Table 4.1.

Table 4.1: Parameters of model (4.1) and their units.

Parameter	Defination	Units
r	maximum per capita growth rate	time^{-1}
k	environmental carrying capacity of the prey	number
β	indirect transmission coefficient for the disease	$\text{mass}^{-1}\text{time}^{-1}$
μ	death rate of the infected animals through CWD	time^{-1}
a	predation coefficient	$\text{mass}^{-1}\text{time}^{-1}$
b	the conversion coefficient of the predator	time^{-1}
m	half saturation constant	number
ε	per capita rate of excretion of infectious material by infected animals	time^{-1}
τ	the mass specific rate of loss of infectious material from the environment	time^{-1}
d	death rate of predator	time^{-1}

4.3 Boundedness of the system

In theoretical eco-epidemiology, boundedness of a system implies that the system is biologically valid and well behaved. The following lemma ensures the boundedness of system (4.1).

Lemma 4.3.1. *All the solutions of system (4.1) which start in* $\mathbb{R}^4_+$ *are uniformly bounded within a region* Ω *defined by* $\Omega=\{(S,I,E,P)\in\mathbb{R}^4_+ : 0\leq W(t)\leq \frac{k(r+1)}{v}\}$ *where* $v=min\{1,\frac{\mu}{2},\tau,d\}$.

Proof. Let $(S(t),I(t),E(t),P(t))$ be one of the solutions of system (4.1). As $\frac{dS}{dt}\leq rS(1-\frac{S+I}{k})$, we have $\lim_{t\to\infty} S(t)\leq k$.

Let a time dependent function $W(t)=S(t)+I(t)+\frac{\mu}{2\epsilon}E(t)+\frac{a}{b}P(t)$, we get,

$$\frac{dW}{dt}=\frac{dS}{dt}+\frac{dI}{dt}+\frac{\mu}{2\epsilon}\frac{dE}{dt}+\frac{a}{b}\frac{dP}{dt}$$

By using system (4.1),

$$\begin{aligned}\tfrac{dW}{dt} &= rS(1-\tfrac{S+I}{k})-\tfrac{\mu}{2}I-\tfrac{\mu\tau}{2\epsilon}E-\tfrac{a}{b}dP\\ &\leq rS-\tfrac{\mu}{2}I-\tfrac{\mu\tau}{2\epsilon}E-\tfrac{a}{b}dP\\ &= (r+1)S-[S+\tfrac{\mu}{2}I+\tfrac{\mu\tau}{2\epsilon}E+\tfrac{a}{b}dP]\end{aligned}$$

$\leq (r+1)k - vW$, where $v = \min\{1, \frac{\mu}{2}, \tau, d\}$.

Hence we have

$$\frac{dW}{dt} + vW \leq (r+1)k.$$

Now applying the theory of differential inequality in [18], we get,

$$0 \leq W(t) \leq \frac{(r+1)k}{v} + \frac{W(S(0), I(0), E(0), P(0))}{e^{vt}}$$

$$0 \leq W(t) \leq \frac{(r+1)k}{v} \quad (t \to \infty).$$

Hence all the solution of the system will enter the region $\Omega = \{(S, I, E, P) : 0 \leq W(t) \leq \frac{k(r+1)}{v}\}$.

4.4 Equilibria and stability analysis

Here we discuss the stability of boundary equilibria and the interior equilibrium point of system (4.1). The system has four equilibrium points, one is population free equilibrium point $E_0(0, 0, 0, 0)$ which always exists. One is susceptible prey only equilibrium point $E_1(k, 0, 0, 0)$ and the other one is predator free equilibrium point $E_2(\overline{S}, \overline{I}, \overline{E}, 0)$.

where $\overline{S} = \frac{\mu\tau}{\beta\epsilon}$, $\overline{I} = \frac{\tau\overline{E}}{\epsilon}$, and $\overline{E} = \frac{r(k\beta\epsilon - \mu\tau)}{\beta(r\tau + k\epsilon\beta)}$).

E_2 is feasible provided $k > \frac{\mu\tau}{\epsilon\beta} = \bar{S}$. Thus the carrying capacity of the environment is high enough to support the predator free equilibrium.

The interior equilibrium point is given by $\hat{E}(S^*, I^*, E^*, P^*)$ where,

$$S^* = \frac{kr\tau(b-d) - \beta\epsilon dmk - r\tau dm}{r\tau(b-d)},\ I^* = \frac{dm}{b-d},\ E^* = \frac{\epsilon dm}{\tau(b-d)} \text{ and } P^* = \frac{bm(\beta\epsilon S^* - \mu\tau)}{a\tau(b-d)}.$$

Clearly the interior equilibrium point $\hat{E}$ is feasible if $b > d$ and $kr\tau(b-d) > \beta\epsilon dmk + r\tau dm$ i.e $r > \frac{\beta\epsilon dmk}{\tau[kb - d(k+m)]} = \hat{r}, S^* > \frac{\mu\tau}{\beta\epsilon}$.

4.4.1 Stability analysis

We will now study the dynamical behavior of the system about all these equilibrium points. The variational matrix for system (4.1) at an arbitrary point (S, I, E, P) is given by

$$V(S,I,E,P) = \begin{pmatrix} r - \frac{2rS}{k} - \frac{rI}{k} - \beta E & -\frac{rS}{k} & -\beta S & 0 \\ \beta E & -\mu - \frac{maP}{(m+I)^2} & \beta S & -\frac{aI}{m+I} \\ 0 & \epsilon & -\tau & 0 \\ 0 & \frac{mbP}{(m+I)^2} & 0 & -d + \frac{bI}{m+I} \end{pmatrix}.$$

Lemma 4.4.1. *(i) E_0 is always unstable, (ii) E_1 is locally asymptotically stable if $\tau\mu > \beta k\epsilon$ and unstable if the inequality is reversed.*

Proof. (i) System (4.1) has the following variational matrix at E_0

$$V(E_0) = \begin{pmatrix} r & 0 & 0 & 0 \\ 0 & -\mu & 0 & 0 \\ 0 & \epsilon & -\tau & 0 \\ 0 & 0 & 0 & -d \end{pmatrix}.$$

Then the characteristic equation of $V(E_0)$ is

$$(\lambda - r)(\lambda + \mu)(\lambda + \tau)(\lambda + d) = 0.$$

Clearly it has only one positive root and other three negative roots therefore the vanishing equilibrium point E_0 is always unstable.

(ii) The variational matrix of system (4.1) at E_1 is given by

$$V(E_1) = \begin{pmatrix} -r & -r & -\beta k & 0 \\ 0 & -\mu & \beta k & 0 \\ 0 & \epsilon & -\tau & 0 \\ 0 & 0 & 0 & -d \end{pmatrix}.$$

Thus the characteristic equation of $V(E_1)$ is given by

$$(\lambda + r)(\lambda + d)\{\lambda^2 + \lambda(\tau + \mu) + \tau\mu - \beta k\epsilon\} = 0.$$

Here all the eigenvalues of the above equation has negative real part if $\tau\mu > \beta k\epsilon$. So the equilibrium point E_1 is locally asymptotically stable if the above condition hold otherwise unstable.

Lemma 4.4.2. *E_2 is locally asymptotically stable if (i) $\tau < \frac{k\beta\epsilon}{\mu} < r$ and (ii) $d < \frac{b\bar{I}}{m+\bar{I}}$.*

Proof. The variational matrix of system (4.1) at E_2 is given by

$$V(E_2) = \begin{pmatrix} -\frac{r\bar{S}}{k} & -\frac{r\bar{S}}{k} & -\beta\bar{S} & 0 \\ \beta\bar{E} & -\mu & \beta\bar{S} & -\frac{a\bar{I}}{m+\bar{I}} \\ 0 & \epsilon & -\tau & 0 \\ 0 & 0 & 0 & -d + \frac{b\bar{I}}{m+\bar{I}} \end{pmatrix}.$$

Therefore the characteristic equation of $V(E_2)$ is

$$\big(\lambda + d - \frac{b\bar{I}}{m+\bar{I}}\big)(\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3) = 0. \tag{4.2}$$

$$\text{where} \begin{cases} a_1 = r\frac{\bar{S}}{k} + \mu + \tau > 0 \\ a_2 = r\frac{\bar{S}}{k}(\mu + \tau + \beta\bar{E}) > 0 \\ a_3 = \frac{r\beta\tau\bar{S}\bar{E}}{k} + \beta^2\epsilon\bar{S}\bar{E} > 0. \end{cases} \tag{4.3}$$

Clearly, one of the eigenvalue say $-d + \frac{b\bar{I}}{m+\bar{I}}$ which is negative follows from the assumption. Further we note that $a_1, a_2, a_3 > 0$ and also $a_1a_2 - a_3 > 0$ by the inequality (i) of Lemma 4.4.2 and hence all the eigenvalues of the characteristic equation (4.2) has negative real parts. Therefore the equilibrium point E_2 is locally asymptotically stable.

Finally, the variational matrix at the interior equilibrium point $\hat{E}$ can be written

as

$$V(\hat{E}) = \begin{pmatrix} -\frac{rS^*}{k} & -\frac{rS^*}{k} & -\beta S^* & 0 \\ \beta E^* & -\mu - \frac{maP^*}{(m+I)^2} & \beta S^* & -\frac{aI^*}{m+I^*} \\ 0 & \epsilon & -\tau & 0 \\ 0 & \frac{mbP^*}{(m+I^*)^2} & 0 & 0 \end{pmatrix}.$$

Then the characteristic equation of $V(\hat{E})$ becomes

$$\lambda^4 + p_1\lambda^3 + p_2\lambda^2 + p_3\lambda + p_4 = 0 \tag{4.4}$$

$$\text{where} \begin{cases} p_1 = -b_{11} - b_{22} - b_{33} > 0 \\ p_2 = b_{11}b_{22} + b_{11}b_{33} - b_{23}b_{32} - b_{24}b_{42} + b_{22}b_{33} - b_{12}b_{21} \\ p_3 = b_{11}b_{23}b_{32} + b_{11}b_{24}b_{42} - b_{11}b_{22}b_{33} + b_{24}b_{42}b_{33} + b_{12}b_{21}b_{33} - b_{13}b_{21}b_{32} \\ p_4 = -b_{11}b_{24}b_{42}b_{33} > 0 \end{cases} \tag{4.5}$$

$b_{11} = -\frac{rS^*}{k}$, $b_{12} = -\frac{rS^*}{k}$, $b_{13} = -\beta S^*$, $b_{21} = \beta E^*$, $b_{22} = -\mu - \frac{maP^*}{(m+I^*)^2}$, $b_{23} = \beta S^*$, $b_{24} = -\frac{aI^*}{m+I^*}$,$b_{32} = \epsilon$, $b_{33} = -\tau$, $b_{42} = \frac{mbP^*}{(m+I^*)^2}$, $b_{14} = b_{31} = b_{34} = b_{41} = b_{43} = b_{44} = 0$.

Clearly, $p_1 > 0, p_3 > 0, p_4 > 0$. The expressions for $p_1p_2 - p_3$ and $p_1p_2p_3 - p_3^2 - p_1^2p_4$ are quite complicated and hence we cannot say about the signs of the above expressions. So we will show local stability of the interior equilibrium point by giving numerical example.

We now state and prove the global stability of the interior equilibrium point with the help of Lyapunov function.

Theorem 4.4.1. *Suppose the interior equilibrium point $\hat{E}$ is locally asymptotically stable in $\mathbb{R}^4_+$ then it is globally asymptotically stable if the following conditions hold :*

(i) $\mu > \frac{P^*}{m+I^*}$

(ii) $4(\mu - \frac{P^*}{m+I^*})(\frac{r\tau}{k} + \frac{\beta}{2}) > \tau(\frac{r}{k} + \beta E^*)^2 + \frac{3\beta}{4}(\beta k + \epsilon)(\frac{r}{k} + \beta E^*) + \frac{r}{k}(\beta k + \epsilon)^2$

(iii) $det(M) > 0$.

Proof. Define a positive definite real valued function

$$V(S,I,E,P) = S - S^* - S^* \ln \frac{S}{S^*} + \frac{1}{2}(I-I^*)^2 + \frac{1}{2}(E-E^*)^2 + \frac{1}{2}(P-P^*)^2 \quad (4.6)$$

V will be a Lyapunov function if $\dot{V} \leq 0$ when $(S,I,E,P) \neq (0,0,0,0)$. Now the time derivative of V along the solution of (4.1) is given by

$$\begin{aligned}
\tfrac{dV}{dt} &= (S-S^*)\tfrac{\dot{S}}{S^*} + (I-I^*)\dot{I} + (E-E^*)\dot{E} + (P-P^*)\dot{P} \\
&= (S-S^*)\{r(1-\tfrac{S+I}{k}) - \beta E\} + (I-I^*)\{\beta SE - \mu I - \tfrac{aIP}{m+I}\} \\
&\quad + (E-E^*)\{\epsilon I - \tau E\} + (P-P^*)\{P(-d + \tfrac{bI}{m+I})\} \\
&= -\tfrac{r}{k}(S-S^*)^2 - \tfrac{r}{k}(S-S^*)(I-I^*) - \beta(S-S^*)(E-E^*) + \beta S(I-I^*)(E-E^*) \\
&\quad + \beta E^*(S-S^*)(I-I^*) - \mu(I-I^*)^2 - \tfrac{I(P-P^*)(I-I^*)}{m+I} - \tfrac{mP^*(I-I^*)^2}{(m+I)(m+I^*)} + \epsilon(I-I^*)(E-E^*) \\
&\quad - \tau(E-E^*)^2 - d(P-P^*)^2 + \tfrac{bI(P-P^*)^2}{m+I} + \tfrac{P^*(I-I^*)(P-P^*)}{(m+I)(m+I^*)} \\
&\leq -\tfrac{r}{k}(S-S^*)^2 - \{\mu - \tfrac{P^*}{m+I^*}\}(I-I^*)^2 - \tau(E-E^*)^2 - \{d - \tfrac{bk}{m}\}(P-P^*)^2 \\
&\quad + (\tfrac{r}{k} + \beta E^*)|S-S^*||I-I^*| + \beta|S-S^*||E-E^*| + (\beta k + \epsilon)|I-I^*||E-E^*| \\
&\quad + (\tfrac{k}{m} + \tfrac{P^*}{m(m+I^*)})|E-E^*||P-P^*| \\
&= -X^T M X,
\end{aligned}$$

where $X^T = \{|S-S^*|, |I-I^*|, |E-E^*|, |P-P^*|\}$ and $M = [a_{ij}]_{4\times 4}$. Elements of the matrix M are given by

$$\begin{aligned}
&a_{11} = \tfrac{r}{k}, \quad a_{22} = \mu - \tfrac{P^*}{m+I^*}, \quad a_{33} = \tau, \quad a_{44} = d - \tfrac{bk}{m}, \quad a_{12} = a_{21} = -\tfrac{1}{2}(\tfrac{r}{k} + \beta E^*), \\
&a_{13} = a_{31} = -\tfrac{\beta}{2}, \quad a_{14} = a_{41} = 0, \quad a_{23} = a_{32} = -\tfrac{\beta k + \epsilon}{2}, \\
&a_{24} = a_{42} = -\tfrac{1}{2}(\tfrac{k}{m} + \tfrac{P^*}{m(m+I^*)}), \quad a_{34} = a_{43} = 0.
\end{aligned}$$

Hence, M is positive definite if

(i) $\mu > \frac{P^*}{m+I^*}$

(ii) $4(\mu - \frac{P^*}{m+I^*})(\frac{r\tau}{k} + \frac{\beta}{2}) > \tau(\frac{r}{k} + \beta E^*)^2 + \frac{3\beta}{4}(\beta k + \epsilon)(\frac{r}{k} + \beta E^*) + \frac{r}{k}(\beta k + \epsilon)^2$

and

(iii) $det(M) > 0$.

Conditions of the theorem imply that M is positive definite and hence $\dot{V} < 0$ and consequently, V is a Lyapunov function with respect to all solutions in the interior of the positive orthant. So $\hat{E}$ is globally asymptotically stable.

4.5 Local bifurcation analysis

In this section, we used the application of Sotomayor's theorem [161] to investigate the local bifurcation around the equilibrium points of system (4.1). As the existence of non-hyperbolic equilibrium point is a necessary but not sufficient condition for bifurcation to occur. Therefore we choose a parameter which gives zero eigenvalue to the variational matrix at the equilibria. Now rewrite system (4.1) in the form :

$\frac{dx}{dt} = F(X)$ where $X = (S, I, E, P)^t$ and $F = (F_1, F_2, F_3, F_4)$ where

$$F_1 = S\left[r\left(1 - \tfrac{S+I}{k}\right) - \beta E\right],\ F_2 = \beta SE - \mu I - \tfrac{aIP}{m+I},$$
$$F_3 = \varepsilon I - \tau E,\ F_4 = P\left(-d + \tfrac{bI}{m+I}\right)$$

Then according to variational matrix of system (4.1) we obtain the following for non-zero vector $V = (v_1, v_2, v_3, v_4)^t$:

$$\begin{aligned} D^2F(S,I,E,P)(V,V) = {} & \tfrac{\partial^2 F}{\partial S^2}v_1^2 + \tfrac{\partial^2 F}{\partial S\partial I}v_1v_2 + \tfrac{\partial^2 F}{\partial I\partial S}v_2v_1 + \tfrac{\partial^2 F}{\partial I^2}v_2^2 + \tfrac{\partial^2 F}{\partial S\partial E}v_1v_3 \\ & + \tfrac{\partial^2 F}{\partial E\partial S}v_3v_1 + \tfrac{\partial^2 F}{\partial S\partial P}v_1v_4 + \tfrac{\partial^2 F}{\partial P\partial S}v_4v_1 + \tfrac{\partial^2 F}{\partial E^2}v_3^2 \\ & + \tfrac{\partial^2 F}{\partial I\partial E}v_2v_3 + \tfrac{\partial^2 F}{\partial E\partial I}v_3v_2 + \tfrac{\partial^2 F}{\partial I\partial P}v_2v_4 + \tfrac{\partial^2 F}{\partial P\partial I}v_4v_2 \\ & + \tfrac{\partial^2 F}{\partial E\partial P}v_3v_4 + \tfrac{\partial^2 F}{\partial P\partial E}v_4v_3 + \tfrac{\partial^2 F}{\partial P^2}v_4^2. \end{aligned}$$

Now after simple calculations we obtain that,

$$D^2F(S,I,E,P)(V,V) = \begin{pmatrix} -\frac{2r}{k}v_1^2 - \frac{2r}{k}v_1v_2 - 2\beta v_1v_3 \\ 2\beta v_1v_3 \\ 0 \\ \frac{2bm}{(m+I)^2}v_2v_4 \end{pmatrix}$$

and

$$D^3F(S,I,E,P)(V,V,V) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}.$$

Thus, system (4.1) has no pitchfork bifurcation. The local bifurcation near the equilibrium points is investigated in the following theorems:

Theorem 4.5.1. *System (4.1) undergoes a transcritical bifurcation near the axial equilibrium point E_1 but no saddle node bifurcation when the parameter β crosses the critical value $\beta^* = \frac{\mu\tau}{k\epsilon}$.*

Proof. One of the eigenvalues of the variational matrix $V(E_1)$ will be zero if $\beta = \beta^* = \frac{\mu\tau}{k\epsilon}$. Now the variational matrix of system at(4.1) E_1 with zero eigenvalue is given by

$$V(E_1) = \begin{pmatrix} -r & -r & -\beta k & 0 \\ 0 & -\mu & \beta k & 0 \\ 0 & \epsilon & -\tau & 0 \\ 0 & 0 & 0 & -d \end{pmatrix}$$

Let $V = (v_1, v_2, v_3, v_4)^t$ be the eigenvector corresponding to eigenvalue $\lambda = 0$. Thus $V = (\frac{\tau(r+\mu)}{\epsilon r}, \frac{\tau}{\epsilon}, 1, 0)^t$. Also, let $W = (w_1, w_2, w_3, w_4)^t$ represents the corresponding eigenvector of $V(E_1)^t$ to the eigenvalues of $\lambda = 0$. Hence $V(E_1)^tW = 0$ gives that $W = (0, \frac{\epsilon}{\mu}, 1, 0)^t$. Now $F_\beta(E_1, \beta^*) = (0,0,0,0)^t$, $F_\beta(E_1, \beta)$ represents the derivative of $F = (F_1, F_2, F_3, F_4)^t$ with respect to β. Then we get $W^t[F_\beta(E_1, \beta^*)] = 0$.

Thus according to Sotomayor's theorem system (4.1) has no saddle-node bifurcation at $\beta = \beta^*$.

Again

$$DF_\beta(E_1, \beta^*) = \begin{pmatrix} 0 & 0 & -k & 0 \\ 0 & 0 & k & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

Then, $W^t[DF_\beta(E_1, \beta^*)V] = \frac{k\epsilon}{\mu} \neq 0$.

Now

$$D^2F(E_1, \beta^*)(V, V) = \begin{pmatrix} -\frac{2r}{k}v_1^2 - \frac{2r}{k}v_1v_2 - 2\beta v_1v_3 \\ 2\beta v_1v_3 \\ 0 \\ 0 \end{pmatrix}.$$

Therefore, $W^t[D^2F(E_1, \beta^*)(V, V)] = 2\beta v_1v_3w_3 \neq 0$.

Thus, according to Sotomayor's theorem system (4.1) has a transcritical bifurcation at E_1 when the parameter β crosses the critical value β^*. Furthermore, as the characteristic equation of $V(E_1)$ has no purely imaginary eigenvalues, so no Hopf bifurcation can occur.

Theorem 4.5.2. *System (4.1) admits a transcritical bifurcation but no saddle-node bifurcation around the equilibrium point E_2 when d crosses the critical value $\bar{d} = \frac{b\bar{I}}{m+\bar{I}}$.*

Proof. Proof is similar to the proof of Theorem 4.5.1.

4.6 Hopf-bifurcation around interior equilibrium point

Now in this section, we will study whether the Hopf-bifurcation occurs or not of system (4.1). In our system, β is a crucial parameter; we will show that Hopf

bifurcation occurs for the system (4.1) at a critical value $\beta = \beta_0$ using the result developed in [179, 95].

Theorem 4.6.1. *If the indirect transmission rate β of the disease crosses a critical value β_0, then the system undergoes a Hopf-bifurcation around its positive equilibrium point $\hat{E}$ if the following conditions hold:*

(i) $p_i(\beta_0) > 0, \forall\ i = 1, 2, 3, 4,$

(ii) $p_1(\beta_0)p_2(\beta_0) > p_3(\beta_0),$

(iii) $p_1(\beta_0)p_2(\beta_0)p_3(\beta_0) - p_3^2(\beta_0) + p_1^2(\beta_0)p_4(\beta_0) = 0,$

and (iv) $\frac{du_i}{d\beta} \neq 0$, *i=1,2,3,4 at* $\beta = \beta_0$.

Proof. Consider, the eigenvalues of the characteristic equation (4.4) is of the form $\lambda_i{=}u_i + iv_i$. Put these in equation (4.4) and separating real and imaginary parts we get

$$u^4 - 6u^2v^2 + v^4 + p_1(u^3 - 3uv^2) + p_2(u^2 - v^2) + p_3u + p_4 = 0, \tag{4.7}$$

$$-4uv^3 + 4u^3v + p_1(3u^2 - v^2)v + 2p_2uv + p_3v = 0. \tag{4.8}$$

From (4.8), we get $f(u) = v^2 = \frac{1}{4u+p_1}(4u^3 + 3p_1u^2 + 2p_2u + p_3)$.

Substituting the value of v^2 in equation (4.7), we get

$u^4(4u + p_1)^2 + (4u^3 + 3p_1u^2 + 2p_2u + p_3)^2 + (4u^3 + 3p_1u^2 + 2p_2u + p_3)(-6u^2 - 3p_1u - p_2)(4u + p_1) + (p_1u^3 + p_2u^2 + p_3u + p_4)(4u + p_1)^2 = 0.$

Now differentiating with respect to β at $\beta = \beta_0$ we get,

$$\left[\frac{du}{d\beta}\right]_{\beta=\beta_0} = \frac{(-\frac{d}{d\beta})(p_1p_2p_3-p_3^2-p_1^2p_4)}{4p_3(p_2-p_3)-2p_1(p_2^2-p_1p_3)+8p_1p_2} \neq 0$$

So the system has a Hopf-bifurcation at a critical value β_0 around the interior equilibrium point $\hat{E}$.

The conditions for obtaining Hopf-bifurcation are too unwidely and complicated to be biologically interpretable. Thus to analyze the model we numerically simulated population dynamics with β less than Fig. 4.4(a) and greater than Fig. 4.4(b) β_0.

4.7 Persistence

Persistence that means long-term survival of all populations in the future time. It does not depend on the initial populations. Mathematically it means strictly positive solutions do not have omega limit points on the boundary of the non-negative cone. We have shown the persistence by using Butler-McGehee lemma [55].

Theorem 4.7.1. *Suppose $d < \frac{b\bar{I}}{m+\bar{I}}$, then system (4.1) is uniformly persistent.*

Proof. Consider, α be any point in the positive octant and $o(\alpha)$ be any orbit through the point $\alpha = (S, I, E, P)$. $\Omega(\alpha)$ is the omega limit set of $o(\alpha)$, which is bounded. Suppose E_0 is not in $\Omega(\alpha)$. If it belongs to the region $\Omega(\alpha)$ then by Butler-McGehee lemma [55], there exist a point u in $\Omega(\alpha) \cap W^s(E_0)$ where $W^s(E_0)$ be the strong manifold of E_0. Since o(u) lies in $\Omega(\alpha)$ and $W^s(E_0)$ is the $I - E - P$ plane, we conclude that o(u) is unbounded, which is a contradiction.

Similarly, we can show that $E_1 \notin \Omega(\alpha)$. If $E_1 \in \Omega(\alpha)$ then by Butler-McGehee lemma [55], there exist a point u in $\Omega(\alpha) \cap W^s(E_1)$ where $W^s(E_1)$ be the strong manifold of E_1. Since o(u) lies in $\Omega(\alpha)$ and $W^s(E_1)$ is the $S - P$ direction, we conclude that o(u) is unbounded, which is a contradiction. Next we show that $E_2 \notin \Omega(\alpha)$, by the condition $d < \frac{b\bar{I}}{m+\bar{I}}$, E_2 is a saddle point. $W^s(E_2)$ is the P direction, hence o(u) is unbounded in $\Omega(\alpha)$, again a contradiction.
Thus, $\Omega(\alpha)$ lies in the positive octant and system (4.1) is persistent. Since system (4.1) is bounded, by the main theorem of Butler et al. [21], the system is

uniformly persistent.

The condition of Theorem 4.7.1 indicates that if the death rate of the predator remains a certain threshold value then none of the populations facing extinction.

Remark 4.7.1. *If the condition of Theorem 4.7.1 is reversed then system (4.1) becomes impermanent [83]. This indicates extinction of one population from the system.*

4.8 Stochastic analysis of the model

Stochastic perturbations have been introduced in some of the model parameters [134, 118, 37]. Here we allow stochastic perturbations of the variables S, I, E, P around their positive interior equilibrium point $\hat{E}$, when it is feasible and locally asymptotically stable. Locally stability of $\hat{E}$ is implied by the existence condition of $\hat{E}$. So in the model we assume that stochastic perturbation of the variables around their values at $\hat{E}$ are of white noise type which are proportional to the distances of S, I, E, P from values S^*, I^*, E^*, P^*. So system (4.1) can be written as

$$\begin{aligned} dS &= S\left[r\left(1-\frac{S+I}{K}\right)-\beta E\right]dt+\sigma_1(S-S^*)d\xi_t^1 \\ dI &= \left[\beta SE-\mu I-\frac{aIP}{m+I}\right]dt+\sigma_2(I-I^*)d\xi_t^2 \\ dE &= \left[\varepsilon I-\tau E\right]dt+\sigma_3(E-E^*)d\xi_t^3 \\ dP &= \left[P\left(-d+\frac{bI}{m+I}\right)\right]dt+\sigma_4(P-P^*)d\xi_t^4 \end{aligned} \tag{4.9}$$

where σ_i, i=1,2,3,4 are real constants, $\xi_t^i = \xi_i(t)$, i=1,2,3,4 are independent from each other standard Wiener processes [58]. We determine whether the dynamical nature of the continuous model (4.1) is robust with respect to such a kind of stochasticity by investigating the asymptotic stochastic stability behaviour of the equilibrium $\hat{E}$ for system (4.9) and by comparing the results with those obtained for system (4.1). We will consider (4.9) as the Ito stochastic differential system.

4.8.1 Stochastic stability of the positive equilibrium

The SDEs system of (4.9) can be centered at $\hat{E}$ by the change of the variables

$$u_1 = S - S^*, \qquad u_2 = I - I^*, \qquad u_3 = E - E^*, \qquad u_4 = P - P^*. \tag{4.10}$$

The linearized SDEs around the positive $\hat{E}$ take the form

$$du(t) = f(u(t))dt + g(u(t))d\xi(t) \tag{4.11}$$

where $u(t) = \text{col}(u_1(t), u_2(t), u_3(t), u_4(t))$ and

$$f(u(t)) = \begin{pmatrix} -\frac{rS^*}{K} & -\frac{rS^*}{K} & -\beta & 0 \\ \beta E^* & -\mu - \frac{maP^*}{(m+I^*)^2} & \beta S^* & -\frac{aI^*}{m+I^*} \\ 0 & \epsilon & -\tau & 0 \\ 0 & \frac{mbP^*}{(m+I^*)^2} & 0 & 0 \end{pmatrix} u(t),$$

$$g(u(t)) = \begin{pmatrix} \sigma_1 u_1 & 0 & 0 & 0 \\ 0 & \sigma_2 u_2 & 0 & 0 \\ 0 & 0 & \sigma_3 u_3 & 0 \\ 0 & 0 & 0 & \sigma_4 u_4 \end{pmatrix}.$$

The positive equilibrium in (4.11) corresponding to the trivial solution u(t)=0.

Let U=$(t \geq t_0) \times \mathbb{R}^n$, $t_0 \in \mathbb{R}^+$. Consider $V \in C_2^0(U)$ be a twice differential function with respect to u and a continuous function with respect to t.

Now we require the following theorem to prove the asymptotically mean square stability of trivial solution of (4.11).

Theorem 4.8.1. *[3] Assume that $V(t,u) \in C_2^0(U)$ satisfying the inequalities*

$$K_1|u|^p \leq V(t,u) \leq K_2|u|^p, \tag{4.12}$$

$$LV(t,u) \leq -K_3|u|^p, K_i > 0, p > 0. \tag{4.13}$$

Then the trivial solution of (4.11) is exponentially p-stable for $t \geq 0$.

Here if $p = 2$ then the trivial solution of (4.1) is globally asymptotically stable in probability.

Again with the reference of [3], we get

$$LV(t,u) = \frac{\partial V(t,u)}{\partial t} + f^T(u)\frac{\partial V(t,u)}{\partial u} + \frac{1}{2}Tr\left[g^T(u)\frac{\partial^2 V(t,u)}{\partial u^2}g(u)\right]$$

where

$$\frac{\partial V}{\partial u} = \text{Col}\left(\frac{\partial V}{\partial u_1}, \frac{\partial V}{\partial u_2}, \frac{\partial V}{\partial u_3}, \frac{\partial V}{\partial u_4}\right),$$

$$\frac{\partial^2 V(t,u)}{\partial u^2} = \frac{\partial^2 V}{\partial u_j \partial u_i}, i, j = 1, 2, 3, 4.$$

and T represents transposition.

Now we state asymptotic mean square stability of the zero solution of (4.1).

Theorem 4.8.2. *Assume that $\sigma_1^2 < \frac{2rS^*}{K}, \sigma_2^2 < 2\left(\mu + \frac{maP^*}{(m+I^*)^2}\right), \sigma_3^2 < 2\tau, \sigma_4^2 < d + 2d_1P^* - \frac{bI^*}{m+I^*}$ and $(\frac{rS^*}{K} - \frac{1}{2}\sigma_1^2)(\tau - \frac{1}{2}\sigma_3^2) > \frac{1}{4}\beta S^* w_1$. Then the zero solution of (4.1) asymptotically mean square stable.*

Proof. Consider a Lyapunov function

$$V(u) = \frac{1}{2}(w_1u_1^2 + w_2u_2^2 + w_3u_3^2 + w_4u_4^2)$$

where w_i all are real positive constants. It is easy to check that inequalities (4.12) hold true with $p = 2$.

Therefore

$$\begin{aligned}LV(u) &= w_1[-\tfrac{rS^*}{K}u_1 - \tfrac{r}{K}S^*u_2 - \beta S^*u_3]u_1 \\ &+ w_2[\beta E^*u_1 - (\mu + \tfrac{maP^*}{(m+I^*)^2})u_2 + \beta S^*u_3 - \tfrac{aI^*}{m+I^*}u_4]u_2 \\ &+ w_3[\epsilon\mu_2 - \tau\mu_3]u_3 \\ &+ w_4[\tfrac{bI^*}{(m+I^*)^2}u_2 - (d + 2d_1P^* - \tfrac{bI^*}{m+I^*}u_4)]u_4 \\ &+ \tfrac{1}{2}Tr[g^T(u)\tfrac{\partial^2 V}{\partial u^2}g(u)].\end{aligned}$$

Note that $\frac{\partial^2 V}{\partial u^2} = \begin{pmatrix} w_1 & 0 & 0 & 0 \\ 0 & w_2 & & 0 \\ 0 & 0 & w_3 & 0 \\ 0 & 0 & 0 & w_4 \end{pmatrix}$

and hence

$$g^T(u)\tfrac{\partial^2 V}{\partial u^2}g(u) = \begin{pmatrix} w_1\sigma_1u_1^2 & 0 & 0 & 0 \\ 0 & w_2\sigma_2u_2^2 & 0 & 0 \\ 0 & 0 & w_3\sigma_3u_3^2 & 0 \\ 0 & 0 & 0 & w_4\sigma_4u_4^2 \end{pmatrix}.$$

So

$$\frac{1}{2}Tr[g^T(u)\frac{\partial^2 V}{\partial u^2}g(u)] = \frac{1}{2}[w_1\sigma_1^2u_1^2 + w_2\sigma_2^2u_2^2 + w_3\sigma_3^2u_3^2 + w_4\sigma_4^2u_4^2]. \tag{4.14}$$

Now if we choose, $w_1\frac{r}{K}S^* = w_2\beta E^*$, $w_2\beta S^* = w_3\epsilon$, and $w_2\frac{a}{m+I^*} = w_4\frac{b}{(m+I^*)^2}$,

from (4.14) it is easy to check that,

$$\begin{aligned}LV(u) &= -(\tfrac{rS^*}{K} - \tfrac{1}{2}\sigma^2)w_1u_1^2 - [\mu + \tfrac{maP^*}{(m+I^*)^2} - \tfrac{1}{2}\sigma_2^2]w_2u_2^2 \\ &\quad - [\tau - \tfrac{1}{2}\sigma_3^2]w_3u_3^2 - [d + 2d_1P^* - \tfrac{bI^*}{m+I^*} - \tfrac{1}{2}\sigma_4^2]w_4u_4^2 - \beta S^*w_1u_1u_3 \\ &= a_{11}u_1^2 - a_{12}u_1u_3 - a_{22}u_3^2 - a_{31}u_2^2 - a_{32}u_4^2\end{aligned}$$

where, $a_{11} = -(\frac{rS^*}{K} - \frac{1}{2}\sigma^2)w_1$, $a_{31} = [\mu + \frac{maP^*}{(m+I^*)^2} - \frac{1}{2}\sigma_2^2]w_2$,
$a_{22} = [\tau - \frac{1}{2}\sigma_3^2]w_3$, $a_{12} = \beta S^*w_1$, $a_{32} = [d + 2d_1P^* - \frac{bI^*}{m+I^*} - \frac{1}{2}\sigma_4^2]w_4$.

It is always negative if $a_{11}a_{22} > \frac{1}{4}a_{12}^2$ that is if $(\frac{rS^*}{K} - \frac{1}{2}\sigma_1^2)(\tau - \frac{1}{2}\sigma_3^2) > \frac{1}{4}\beta S^* w_1$. Hence the result follows from Theorem 4.8.1.

In this next section, we have performed some numerical simulations to verify our results obtained earlier. Numerical simulations are performed by the help of Matlab R2014a software package for hypothetical set of data.

4.9 Numerical simulation

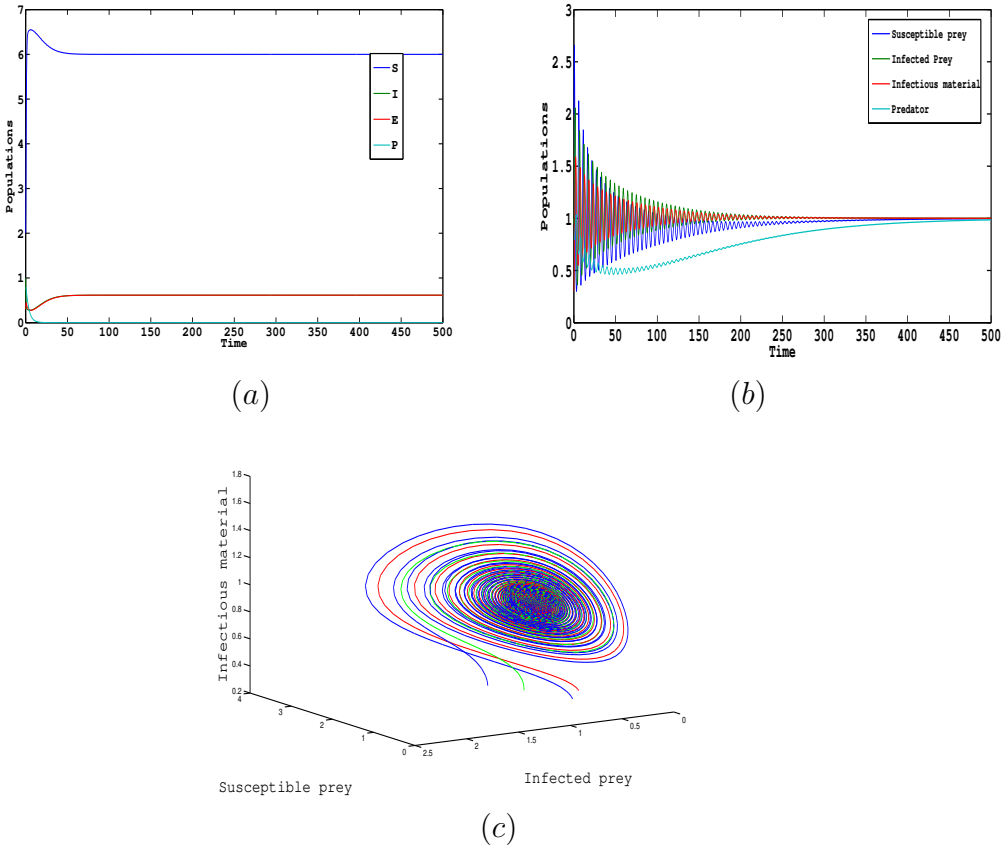
In most of eco-epidemiological studies the infection rate plays a crucial role in describing the dynamics of of the system. Keeping this in mind we varied the infection rate β to observe different type of behavior of system (4.1). Suppose $r = \frac{14}{5}, k = 7,\ \mu = 1.5,\ a = 1, m = 1,\ \varepsilon = 1,\ \tau = 1,\ d = 0.5, b = 1$, our results show that the predator population will become extinct for the parametric values of $\beta = 0.25$ (see Fig. 4.1(a)). Increasing the value of β we observe that the system has a positive equilibrium $(1, 1, 1, 1)$ at $\beta = 2$, which is locally asymptotically stable (see Fig. 4.1(b)). A three dimensional phase diagram represents the stability of system (4.1) (see Fig. 4.1(c)).

We observed that increasing amount of transmission rate $\beta = 2.5$ stabilizes the system and the interior equilibrium point does not exist, consequently system (4.1) is impermanent (see Fig. 4.2).

Again if we take $\beta = 2.07$ and all other parameter remains same, then system (4.1) has a positive equilibrium point $(\frac{1}{3}, 1, 1, \frac{1}{3})$ and we observe that all the species of system (4.1) enter an oscillatory state from a stable situation and in this case the system is uniformly persistent (permanent). A three dimensional phase diagram represents the limit cycle oscillations of system (4.1) (see Fig. 4.3).

Furthermore, the interior equilibrium point $\hat{E}$ undergoes a Hopf bifurcation and stability changes occur when the parameter β exceeds its critical value $\beta_0 = 2.049$. To demonstrate these dynamical behaviors of system (4.1), we have plotted the bifurcation diagram of the system in (β, S, I) plane and its projections on (β, S) and (β, I) plane in Fig. 4.4.

To show the interplay between the disease transmission rate β and the half saturation constant m in the stability of the system around the interior equilibrium $\hat{E}$, we plot the stability region of $\hat{E}$ in $\beta - m$ parametric space which shown



(a) (b)

(c)

FIGURE 4.1: Influence of indirect transmission rate β on predator population: (a) extinction of predator population for $\beta = 0.25$,(b) for $\beta = 2.0$ time series plot indicate coexists of all the species and (c) phase portrait shows local stability of endemic equilibrium point $\hat{E}$. Other model parameters are $r = \frac{14}{5}, k = 7$, $\mu = 1.5$, $a = 1, m = 1$, $\varepsilon = 1$, $\tau = 1$, $d = 0.5, b = 1$.

in Fig. 4.5. Red region indicates the stable coexistence of all populations.

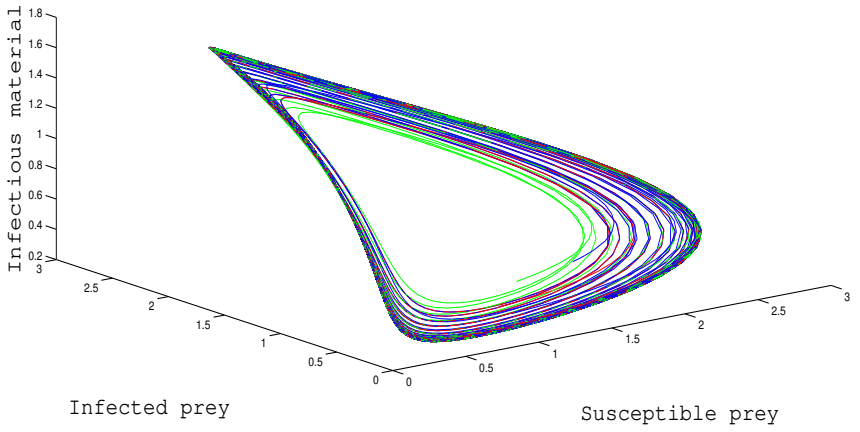


Figure 4.2: The interior equilibrium point $\hat{E}$ does not exist and system (4.1) is impermanent for parametric values $r = \frac{14}{5}, k = 7$, $\beta = 2.5$, $\mu = 1.5$, $a = 1, m = 1$, $\varepsilon = 1$, $\tau = 1$, $d = 0.5, b = 1$.

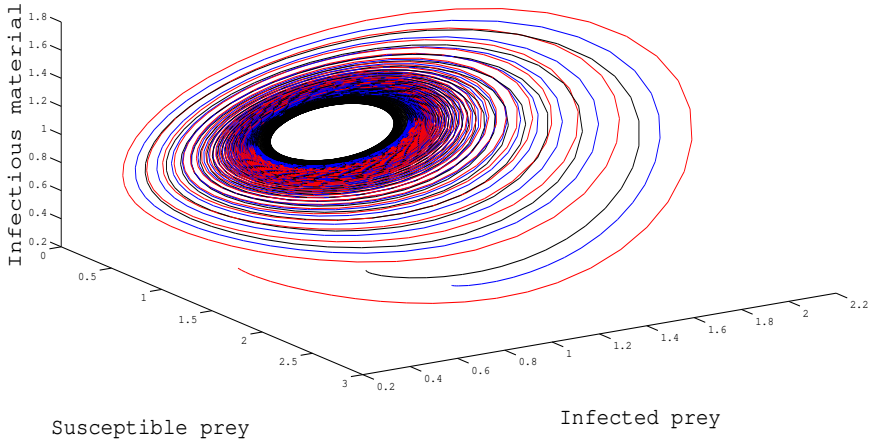


Figure 4.3: Limit cycle appears and the system is permanent for the parametric values $r = \frac{14}{5}, k = 7$, $\beta = 2.07$, $\mu = 1.5$, $a = 1, m = 1$, $\varepsilon = 1$, $\tau = 1$, $d = 0.5, b = 1$.

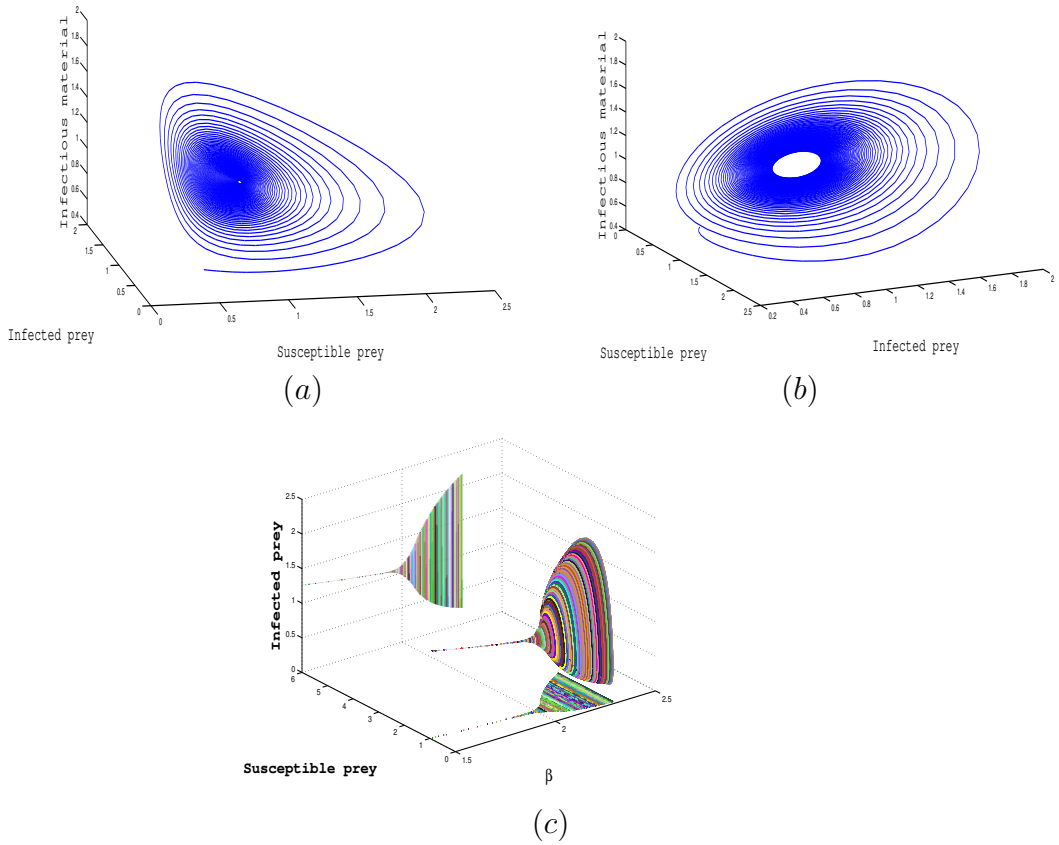


Figure 4.4: Phase portrait and bifurcation diagram: (a) system is stable when $\beta = 2.030 < \beta_0 = 2.049$, (b) system loses its stability when $\beta = 2.050 > \beta_0 = 2.049$, (c) bifurcation diagram of system (4.1) in (β, S, I) plane and its projection on (β, S) and (β, I) planes for $\beta \in (1.5, 2, 5)$ and other model parameters are $r = \frac{14}{5}, k = 7, \mu = 1.5, a = 1, m = 1, \varepsilon = 1, \tau = 1, d = 0.5, b = 1$.

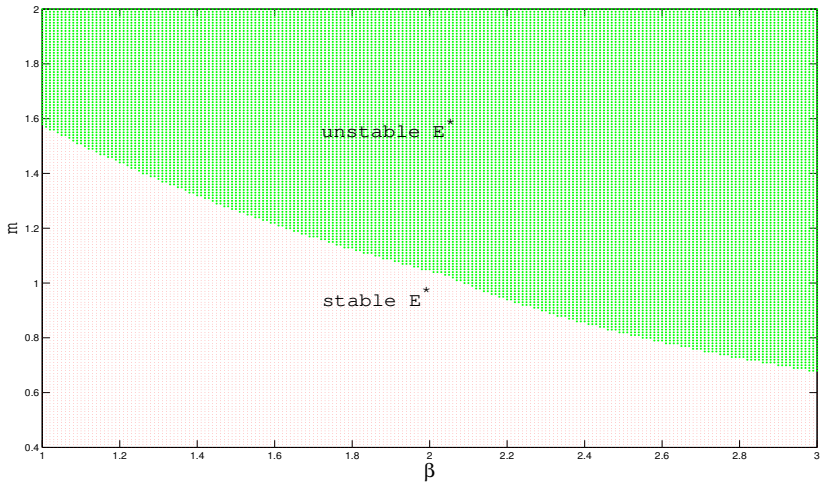


Figure 4.5: Stability region of system (4.1) in $\beta - m$ plane. Red region represents the stability of the coexistence equilibrium and green region represents the unstability of the coexistence equilibrium point.

4.10 Discussion

In this chapter, we have generalized an existing eco-epidemic model of the dynamics of chronic wasting disease affecting an animal population. Including the dynamics of the predator population we arrive at a 4-dimensional systems of coupled nonlinear differential equations. To eradicate diseases, vaccination and prevention policies are mainly used [69]. But for fatal diseases like CWD, there is no vaccination programme still now. However, it may be possible for disease affecting a population by allowing in the environment another population which is predator of the former. It is well known fact that infected prey individuals become easy to catch by the predator . This property has been considered in a various number of eco-epidemiological models [25, 172, 183, 135, 24, 70]. In our model we have considered that predator consumes infected prey only to control the disease [99, 145]. The predation process follows Holling type II response function. The proposed model is shown biologically well-behaved as the system is bounded. The local stability of the system in different steady states has been discussed. Further, the system cannot collapse for any values of parameters as the population free equilibrium (E_0) is never stable. It is observed that if carrying capacity of the environment remains below a certain threshold value ($k < \frac{\tau\mu}{\beta\epsilon}$), then the susceptible prey only equilibrium (E_1) is an attractor. If predators has large death rate ($d > \frac{b\bar{I}}{m+\bar{I}}$) then predator free equilibrium (E_2) cannot be stable. We have also derived the conditions under which the coexistence equilibrium is globally asymptotically stable i.e. the disease persists in the populations. For disease eradication, such type of conditions should be avoided. Sotomayor's theorem [161] is applied to ensure the existence of transcritical bifurcation. This bifurcation transforms a susceptible prey only equilibrium point into a unstable equilibrium point and at the predator free equilibrium to a unstable one. The local existence of limit cycle is obtained through Hopf-bifurcation when transmission rate β crosses a critical value. Miller et al. [127] address a model for CWD dynamics without considering the growth equations of predator populations for obtaining coexistence results. Furthermore, they have not investigated the effect of environmental noise on the system. These two aspects are analyzed in our model. We have chosen selective predation to eradicate disease in our system. Still non-selective predation be effective in controlling the disease.

To investigate the effect of environmental noise on the system we apply stochastic perturbation around the positive equilibrium. Constructing a suitable Lyapunov function and using Ito's formula, we note that deterministic model is robust with respect to stochastic perturbation. The criterion of stochastic stability depends on the intensities of noise $\sigma_i, i = 1, 2, 3, 4$. If the intensities of noise are small the stochastic stability can be achieved under certain condition. Further the stochastic stability of the system shows that the intensities $\sigma_i, i = 1, 2, 3, 4$ have the tolerance limits.

5

Study of a fractional-order model of chronic wasting disease

5.1 Introduction

There has been a growing interest in the study of chronic wasting disease (CWD) [159]. It is a prion disease of North American cervids and mainly found in mule deer. This disease belongs to the family of transmissible spongiform encephalopathies (TSEs). It was first identified in the year 1980. To understand the disease incidence and spatial dynamics, long term study is essential which is difficult to carry out in natural population due to economic and logical constraints. In most of the cases the disease spreads indirectly via environmental contamination, though some literature shows that the disease transmitted through direct transmission [119, 125]. There are several works on chronic wasting disease which may

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be found in [148, 142, 53, 117]. Other disease model studied in [174, 175, 176, 22] can be applicable to the chronic wasting disease for better understanding the system. In 2006, Miller et al. [125] developed a chronic wasting disease model by considering well-known susceptible-infected-recover (SIR) models in human beings. Later on Sharp and Pastor [158] modified the model of Miller et al. by changing the constant birth rate with a logistic growth term and described the model as :

$$\begin{aligned} \frac{dS(t)}{dt} &= rS\left(1-\frac{S}{k}\right)-\beta SE, \\ \frac{dI(t)}{dt} &= \beta SE - I(m+\mu), \\ \frac{dE(t)}{dt} &= \epsilon I - \tau E \end{aligned} \tag{5.1}$$

with initial conditions $S(0) > 0, I(0) > 0$ and $E(0) > 0$.

Here S and I represent the total density of the susceptible and infected animals respectively. The dynamic variable E denotes the mass of infectious material in the environment. r is the net per capita growth rate of the susceptible population and k is environmental carrying capacity. The indirect transmission coefficient for the disease is denoted by β. μ is the natural death rate of the infected populations. ϵ corresponds to the deposition of infectious material through excretion by infected animals and the mass specific rate of loss of infectious material from the environment is described by τ.

Due to the progress of fractional calculus, fractional order differential equations have been used in different fields including biological systems. It was first initiated by Leibniz and Hospital in 1965 [146]. Recently, fractional order system has attracted the attention of scientists, ecologists and engineers. Behind, the reason to choose fractional order system rather than integer order system as modelling of such systems by fractional order differential equations have more advantages than classical integer order mathematical modelling. The integer order derivatives demonstrates the local properties of a certain position for a physical phenomenon while fractional order derivative can take care of the whole process. In particular, fractional order differential equations are used to explain certain

phenomena [96]. Developing a integer order system into a fractional order becomes an important issue in dynamical system. Details study of this issue can be found in the review work [10, 8, 9]. There are many applications of fractional order differential equations in the field of system biology [32, 48, 152], physics [38, 47], chemistry and biochemistry [188], engineering [115, 116] and medicine [7, 51]. Fractional order biological systems are studied in [4, 49, 84, 154].

Qualitative analysis of fractional order system is much complicated rather than classical integer order system as fractional order derivatives are non local and have weakly singular kernels but the main advantage of considering such system is that they admit greater degree of freedom in the model. Moreover, it is more realistic than integer order in biological modelling due to memory effects. Several studies are carried out numerically in fractional order system but few authors obtained some interesting results. Stability of fractional order nonlinear system is investigated in [107]. The theory of Lyapunov direct method is further developed by Delavari et al. [39] with the help of Caputo type fractional order nonlinear system. Javidi and Nyamaradi [84] studied the dynamical behaviour of the fractional order predator-prey model and described the local stability of the system. Rihan et al. [153] developed a fractional order predator-prey system with Holling type II fractional response and time delay and they discussed local stability as well as global stability of steady states and Hopf bifurcation with respect to the delay parameter. Recently global stability analysis is discussed elaborately in [170]. Xu et al. analyzes the chaos synchronization between two different fractional order chaotic system by using active control [185].

In the present chapter, we now transform the integer order derivative by Caputo type derivative and the modified model becomes:

$$\begin{aligned} {}^cD^\alpha S(t) &= rS\left(1-\frac{S}{k}\right)-\beta SE, \\ {}^cD^\alpha I(t) &= \beta SE - I(m+\mu), \\ {}^cD^\alpha E(t) &= \epsilon I - \tau E \end{aligned} \tag{5.2}$$

with initial conditions $S(0) = S_0 \geq 0, I(0) \geq 0$ and $E(0) \geq 0$, where $\alpha \in (0,1)$ and ${}^cD^\alpha$ is the standard Caputo differentiation.

5.2 Basic definitions

In this work, we use a Caputo fractional-order derivative because the initial conditions of fractional differential equations with Caputo derivatives assume on the identical form as for integer-order ones, which can be used in modelling and analysis. In this section, we present some definitions for fractional derivatives.

Definition 5.2.1. *[147] The Riemann-Liouville fractional integral operator of order α of any function $f \in L_1[0,a]$, $t \in [0,a]$ is presented as*

$$J^{\alpha} f(t) = \frac{1}{\Gamma(\alpha)} \int_0^a (t-s)^{\alpha-1} f(s) ds,$$

where $\Gamma(.)$ is the Gamma function.

Definition 5.2.2. *[147] The Caputo fractional derivative of order α for a function $f \in \mathbb{C}^n([0,+\infty],\mathbb{R})$ is defined by*

$${}^{c}D^{\alpha} f(t) = \frac{1}{\Gamma(n-\alpha)} \int_0^a (t-s)^{n-\alpha-1} f^{(n)}(s) ds,$$

where $\Gamma(.)$ is the Gamma function , $t \geq 0$ and n is the positive integer such that $n-1 < \alpha < n, n \in \mathbb{N}$.

Particularly, when $0 < \alpha < 1$,

$${}^{c}D^{\alpha} f(t) = \frac{1}{\Gamma(n-\alpha)} \int_0^t \frac{f'(s)}{(t-s)^{\alpha}} ds.$$

The idea of fractional derivative was first introduced by Riemann-Liouville (R-L). In R-L fractional differential equation, initial value is usually taken in the form of fractional derivative, which is not appropriate in real sense whereas in Caputo fractional derivative, the derivative is not defined locally at time t, it depends on the total effects of the so called n-order integer derivative on the interval $[0,s]$. Thus it is reasonable to consider the variation of a system in which the instantaneous change rate depends on the past rate, which is known as "memory effect"[129].

5.3 Main results

The existence, uniqueness, non-negativity and boundedness of the solutions are presented in the following section. Moreover, we will also discuss the global asymptotic stability of the predator-extinction equilibrium point and coexistence equilibrium point by choosing suitable Lyapunov functions.

5.3.1 Existence and uniqueness

We now prove the existence and uniqueness of the solution for a fractional order system (5.2).

Lemma 5.3.1. *[108] Define the system*

$$^{c}D^{\alpha}x(t) = f(t,x), t > 0 \tag{5.3}$$

with initial condition x_0, where $\alpha \in (0,1], f : [0,\infty) \times \Omega \to \mathbb{R}^n, \Omega \in \mathbb{R}^n$, then there exists a unique solution of (5.2) whenever $f(t,x)$ follows locally Lipschitz condition with respect to x on $[0,\infty) \times \Omega$.

Theorem 5.3.1. *Fractional order system (5.2) admits unique solution for any non-negative initial conditions.*

Proof. Existence and uniqueness of system (5.2) will be shown in the region $B \times (0,T]$ where

$B = \{(S,I,E) \in \mathbb{R}^3 : \max(|S|,|I|,|P| \leq M)\}$. We follow the approach used in Hong et al. [106]. We denote $X = (S,I,E)$ and $\bar{X} = (\bar{S},\bar{I},\bar{E})$. Consider a mapping
$H(X) = (H_1(X), H_2(X), H_3(X))$ and

$$\begin{aligned} H_1(X) &= rS\left(1 - \frac{S}{k}\right) - \beta SE \\ H_2(X) &= \beta SE - I(m+\mu), \\ H_3(X) &= \epsilon I - \tau E. \end{aligned} \tag{5.4}$$

where all the parameters used in (5.4) are same as in system (5.2).

For $X, \bar{X} \in B$, it follows from (5.4) that

$\|H(X) - H(\bar{X})\|$

$= |H_1(X) - H_1(\bar{X})| + |H_2(X) - H_2(\bar{X})| + |H_3(X) - H_3(\bar{X})|$

$= |rS\left(1 - \frac{S}{k}\right) - \beta SE - r\bar{S}\left(1 - \frac{\bar{S}}{k}\right) + \beta\bar{S}\bar{E}| + |\beta SE - I(m+\mu) - \beta\bar{S}\bar{E} + \bar{I}(m+\mu)| + |\epsilon I - \tau E - \epsilon\bar{I} + \tau\bar{E}|$

$= |r(S - \bar{S}) - \frac{r}{k}(S^2 - \bar{S}^2) - \beta(SE - \bar{S}\bar{E})| + |\beta(SE - \bar{S}\bar{E}) - (m+\mu)(I - \bar{I})| + |\epsilon(I - \bar{I}) - \tau(E - \bar{E})|$

$\leq (r + \frac{2rM}{k} + 2M\beta)|S - \bar{S}| + (m + \mu + \epsilon)|I - \bar{I}| + (2M\beta + \tau)|E - \bar{E}|$

$\leq L||X - \bar{X}||,$

where $L = \max\{r + \frac{2rM}{k} + 2M\beta, m + \mu + \epsilon, 2M\beta + \tau\}$.

Hence, Lipschitz condition is obvious for $H(X)$. Thus existence of unique solution $X(t)$ of system (5.2) follows from Lemma 5.3.1. This completes the proof.

5.3.2 Non-negativity and boundedness

To justify the model from biological point of view, we are confined in solutions which are non-negative and bounded. The following result ensures the non-negativity and boundedness of the solutions of system (5.2).

Theorem 5.3.2. *Solutions of system (5.2) initiating in $\mathbb{R}^3_+$ are uniformly bounded and non-negative.*

Proof. Define a function $V(t) = S(t) + I(t) + \frac{m}{\epsilon}E$.

Hence for $\lambda = \min\{\mu, \tau\}$

${}^cD^\alpha V(t) + \lambda V(t) = rS(1 - \frac{S}{k}) - \beta SE + \beta SE - I(m+\mu) + mI - \frac{m}{\epsilon}\tau E + \lambda S + \lambda I + \lambda\frac{m}{\epsilon}E$

$$= -\tfrac{r}{k}S^2 + (r+\lambda)S + (\lambda-\mu)I + \tfrac{m}{\epsilon}(\lambda-\tau)E$$

$$= -\tfrac{r}{k}(S - \tfrac{k(r+\lambda)}{2r})^2 + \tfrac{k(r+\lambda)^2}{4r} + (\lambda-\mu)I + \tfrac{m}{\epsilon}(\lambda-\tau)E$$

$$\leq \tfrac{k(r+\lambda)^2}{4r}$$

By applying the standard comparison theorem for fractional order in Choi et.al. [30], we get

$$V(t) \leq V(0)G_\alpha(-\lambda(t)^\alpha) + (\frac{k(r+\lambda)^2}{4r})t^\alpha G_{\alpha,\alpha+1}(-\lambda(t)^\alpha),$$

where G_α is the Mittag-Leffler function. So application of Lemma 5 and Corollary 6 in [30] yields

$$V(t) \leq \frac{k(r+\lambda)^2}{4r\lambda}, t \to \infty.$$

Hence, all the solutions of system (5.2) initiating $\mathbb{R}^3_+$ remains in the region Ω where

$$\Omega = \{(S,I,E) \in \mathbb{R}^3_+ : V \leq \frac{k(r+\lambda)^2}{4\lambda r} + \epsilon, \epsilon > 0\}. \tag{5.5}$$

Now, we want to prove the solutions of system (5.2) are non-negative.

The first equation of system (5.2) gives

$${}^cD^\alpha S(t) = rS\left(1 - \frac{S}{k}\right) - \beta SE \tag{5.6}$$

From (5.5), it can be noted that

$$S + I + \frac{m}{\epsilon}E \leq \frac{k(r+\lambda)^2}{4\lambda r} = p \tag{5.7}$$

From (5.6) and (5.7), we have

$${}^cD^\alpha S(t) \geq rS\big(1 - \frac{p}{k}\big) - \frac{\beta q}{m}S = S\big(r - \frac{rp}{k} - \frac{\beta q}{m}\big) = qS$$

where $q = r - \frac{rp}{k} - \frac{\beta q}{m}$.

From the standard comparison theorem for fractional order in [30] and the positivity of Mitlang-Leffler function $G_{\alpha,1}(t) > 0$ for any $\alpha \in (0,1)$ [177], it follows that

$$S(t) \geq S_0 G_{\alpha,1}(qt^\alpha) \Rightarrow S(t) \geq 0.$$

From second sub-equation of system (5.2),

$$^cD^\alpha I(t) = \beta SE - I(m+\mu) \geq -I(m+\mu).$$

Therefore, $I(t) \geq I_0 G_{\alpha,1}(-(\mu+m)t^\alpha) \Rightarrow I \geq 0$.

Again from the third equation of (5.2),

$$^cD^\alpha E(t) = \epsilon I - \tau E \geq -\tau E.$$

So, $E(t) \geq E_0 G_{\alpha,1}(-\tau t^\alpha) \Rightarrow E \geq 0$.

Hence, all solution of system (5.2) are non-negative.

5.4 Equilibria and their stability

To find the equilibria of system (5.2) we compute

$$^cD^\alpha S(t) = 0, ^cD^\alpha I(t) = 0, ^cD^\alpha E(t) = 0.$$

Thus, the equilibrium points are $E_0(0,0,0)$, $E_1(k,0,0)$, and $\tilde{E}(S^*, I^*, E^*)$ where

$$S^* = \frac{\tau(m+\mu)}{\beta\epsilon}, I^* = \frac{r(k\beta\epsilon - \tau(m+\mu))}{k\beta\epsilon(m+\mu)}, E^* = \frac{r(k\beta\epsilon - \tau(m+\mu))}{k\beta\tau(m+\mu)}.$$

Clearly, $\tilde{E}$ is feasible if $k > \frac{\tau(m+\mu)}{\beta\epsilon}$.

The Jacobian matrix of system (5.2) at any point (S, I, E) is given by:

$$J(S,I,E) = \begin{pmatrix} r(1-\frac{2S}{k}-\beta E) & 0 & -\beta S \\ \beta E & -(m+\mu) & \beta S \\ 0 & \epsilon & -\tau \end{pmatrix}.$$

Theorem 5.4.1. *The equilibrium point E_0 of system (5.2) is always a saddle point.*

Proof. The Jacobian matrix at E_0 is given by

$$J(E_0) = \begin{pmatrix} r & 0 & 0 \\ 0 & -(m+\mu) & 0 \\ 0 & \epsilon & -\tau \end{pmatrix}.$$

Eigenvalues of matrix $J(E_0)$ are obtained by solving the characteristic equation

$$P(\lambda) = \det(J(E_0) - I\lambda) = (r-\lambda)(m+\mu+\lambda)(\tau+\lambda)=0$$

We now describe the eigenvalues of the Jacobian matrix evaluated at E_0

$\lambda_1 = r > 0, \lambda_2 = -(m+\mu) < 0$ and $\lambda_3 = -\tau < 0$. Thus $|\arg(\lambda_1)| = 0 < \frac{\alpha\pi}{2}, |\arg(\lambda_2)| = \pi > \frac{\alpha\pi}{2}, |\arg(\lambda_3)| = \pi > \frac{\alpha\pi}{2}$. It follows from convergence of Mitlag-Leffler function [120] that the equilibrium E_0 is always a saddle point.

Theorem 5.4.2. *The equilibrium E_1 of system (5.2) is locally asymptotically stable if $(m+\mu)\tau > \beta\epsilon k$*

Proof. The Jacobian matrix of system (5.2) around the equilibrium point E_1 is presented by

$$J(E_1) = \begin{pmatrix} -r & 0 & -\beta k \\ 0 & -(m+\mu) & \beta k \\ 0 & \epsilon & -\tau \end{pmatrix}.$$

Eigenvalues of matrix $J(E_1)$ are $\lambda_1 = -r$ and other two λ_2, λ_3 are obtained by solving the characteristic equation

$$P(\lambda) = \lambda^2 + (m+\mu+\tau)\lambda + (m+\mu)\tau - \beta\epsilon k = 0.$$

The eigenvalues corresponding to the equation $P(\lambda)$ are

$$\lambda_{2,3} = \frac{-(m+\mu+\tau) \pm \sqrt{(m+\mu+\tau)^2 - 4\{(m+\mu)\tau - \beta\epsilon k\}}}{2}.$$

Thus, E_1 is locally asymptotically stable if $|\arg(\lambda_i)| > \frac{\alpha\pi}{2}, i = 1, 2, 3.$

Now $|\arg(\lambda_1)| = \pi > \frac{\alpha\pi}{2}$. If $(m+\mu)\tau > \beta\epsilon k$ then $\lambda_2, \lambda_3 < 0$ such that $|\arg(\lambda_{2,3})| = \pi > |\frac{\alpha\pi}{2}$. This completes the proof.

To analyze the stability of equilibrium point $\tilde{E}$, we compute $J(\tilde{E})$, where

$$J(\tilde{E}) = \begin{pmatrix} -\frac{rS^*}{k} & 0 & -\beta S^* \\ \beta E^* & -(m+\mu) & \beta S^* \\ 0 & \epsilon & -\tau \end{pmatrix}.$$

Eigenvalues of matrix $J(\tilde{E})$ are obtained by solving the characteristic equation

$$P(\lambda) = \det(J(\tilde{E}) - I\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0,$$

where $a_1 = \frac{rS^*}{k} + m + \mu + \tau, a_2 = \frac{rS^*}{k}(m+\mu+\tau), a_3 = \beta^2 S^* E^* \epsilon.$

Let $D(P)$ is the discriminant of a polynomial $P(\lambda)$, it can be written as

$$\begin{pmatrix} 1 & a_1 & a_2 & a_3 & 0 \\ 0 & 1 & a_1 & a_2 & a_3 \\ 3 & 2a_1 & a_2 & 0 & 0 \\ 0 & 3 & 2a_1 & a_2 & 0 \\ 0 & 0 & 3 & 2a_1 & a_2 \end{pmatrix},$$

$$D(P) = 18a_1a_2a_3 + (a_1a_2)^2 - 4a_3a_1^2 - 4a_2^3 - 27a_3^2.$$

Proposition 5.4.1. *Suppose $k > \frac{\tau(m+\mu)}{\beta\epsilon}$. Then the equilibrium $\tilde{E}$ of system (5.2) is asymptotically stable if one of the following conditions [4] are satisfied.*

1. *$D(P) > 0, a_1 > 0, a_3 > 0$ and $a_1a_2 > a_3$.*

2. $D(P) < 0, a_1 \geq 0, a_2 \geq 0, a_3 > 0$ *and* $\alpha < \frac{2}{3}$.

3. $D(P) < 0, a_1 > 0, a_2 > 0, a_1 a_2 = a_3$ *and for all* $\alpha \in (0, 1)$.

5.5 Global stability

In this section, we present global stability of E_1 and $\tilde{E}$.

Theorem 5.5.1. *The susceptible individuals only equilibrium point* E_1 *of system (5.2) is globally asymptotically stable if* $k < \frac{(m+\mu)\tau}{\beta\epsilon}$.

Proof. Let us choose a positive definite Lyapunov function $V(S, I, E)$ as

$$V(S, I, E) = [S - k - k \ln \frac{S}{k}] + I + \frac{k\beta}{\tau} E$$

We compute the α order derivative of $V(S, I, E)$ along the solutions of system (5.2). Applying a result in [170], we get

$$\begin{aligned} {}^cD^\alpha V(S, I, E) &\leq (1 - \tfrac{S}{k})^c D^\alpha S(t) +^c D^\alpha I(t) + \tfrac{k\beta}{\tau}{}^c D^\alpha E(t). \\ &= (S - k)\{r(1 - \tfrac{S}{k}) - \beta E\} + [\beta SE - I(m + \mu)] + \tfrac{k\beta}{\tau}[\epsilon I - \tau E] \\ &= -\tfrac{r(S-k)^2}{k} + [\tfrac{k\beta\epsilon}{\tau} - (m + \mu)]I. \end{aligned}$$

Thus, ${}^cD^\alpha V(S, I, E) \leq 0$ when $\frac{k\beta\epsilon}{\tau} < (m + \mu)$. The result follows by the application of Lemma 4.6 in [81].

Now let us define $F(S) = \beta S$.

Theorem 5.5.2. *The positive equilibrium point* $\tilde{E}$ *of system (5.2) is globally asymptotically stable if* $\bar{S} + S^* \geq k$, *where* $\bar{S} = \inf S(t)$.

Proof. Consider the following positive definite function about $\tilde{E}$

$$V(S, I, E) = \int_{S^*}^{S} \frac{F(\theta) - F(S^*)}{F(\theta)} d\theta + \int_{I^*}^{I} \frac{\theta - I^*}{\theta} d\theta + \frac{F(S^*)E^*}{I^*\epsilon} \int_{E^*}^{E} \frac{\theta - E^*}{E^*} d\theta.$$

We compute the α order derivative of $V(S, I, E)$ around the solution of system (5.2). By applying a result in [170], we obtain

$${}^cD^\alpha V(S,I,E) \leq \frac{F(S)-F(S^*)}{F(S)}\{rS(1-\frac{S}{k}) - F(S)E\} + \frac{(I-I^*)}{I}(F(S)E - I(m+\mu)) + \frac{F(S^*)E^*}{I^*\epsilon}\frac{(E-E^*)}{E}(\epsilon I - \tau E)$$

$$= \frac{F(S)-F(S^*)}{F(S)}\{rS(1-\frac{S}{k}) - F(S)E - rS^*(1-\frac{S^*}{k}) + F(S^*)E^*\} + (1-\frac{I^*}{I})\{F(S)E - I(m+\mu)\} + \frac{F(S^*)E^*}{I^*\epsilon}(1-\frac{E^*}{E})(\epsilon I - \tau E)$$

$$= \frac{r(F(S)-F(S^*))(S-S^*)}{F(S)}\{1-\frac{(S+S^*)}{k}\} + (1-\frac{F(S^*)}{F(S)})\{F(S^*)E^* - F(S)E\} + F(S)E - I(m+\mu) - \frac{I^*F(S)E}{I} + I^*(m+\mu) + \frac{F(S^*)E^*I}{I^*} - F(S^*)E - \frac{F(S^*)E^*E^*I}{I^*E} + F(S^*)E^*$$

$$= \frac{r\beta(S-S^*)^2}{\beta S}\{1-\frac{(S+S^*)}{k}\} - F(S^*)E^*\{\frac{F(S^*)}{F(S)} + \frac{F(S)EI^*}{F(S^*)E^*I} + \frac{E^*I}{I^*E} - 2\} + I^*(m+\mu).$$

$$= \frac{r\beta(S-S^*)^2}{\beta S}\{1-\frac{(S+S^*)}{k}\} - F(S^*)E^*\{\frac{F(S^*)}{F(S)} + \frac{F(S)EI^*}{F(S^*)E^*I} + \frac{E^*I}{I^*E} - 3\} - F(S^*)E^* + I^*(m+\mu).$$

$$= \frac{r\beta(S-S^*)^2}{\beta S}\{1-\frac{(S+S^*)}{k}\} - F(S^*)E^*\{\frac{F(S^*)}{F(S)} + \frac{F(S)EI^*}{F(S^*)E^*I} + \frac{E^*I}{I^*E} - 3\}$$

as $F(S^*)E^* = I^*(m+\mu)$.

Now, using the arithmetic mean is greater than or equal to the geometric mean, it is clear that,

$$\frac{F(S^*)}{F(S)} + \frac{F(S)EI^*}{F(S^*)E^*I} + \frac{E^*I}{I^*E} \geq 3,$$

and the equality holds only for $S = S^*, I = I^*, E = E^*$. Therefore

$${}^cD^\alpha V(S,I,E) \leq \frac{r\beta(S-S^*)^2}{\beta S}\{1-\frac{(S+S^*)}{k}\}.$$

Further, if we consider $\inf S(t) = \bar{S}$, then $S(t) \geq \bar{S}$.

Thus, if $\bar{S} + S^* \geq k$, then ${}^cD^\alpha V(S,I,E)$ is negative definite. Hence V is a Lyapunov function with respect to $\tilde{E}$ whose domain of attraction is B, proving the theorem.

5.6 Existence of Hopf-bifurcation

In this section, we will study the Hopf bifurcation of system (5.2). Hopf bifurcation can be occured when a system has a pair of complex conjugate eigenvalues of the Jacobian matrix at an equilibrium point and when the bifurcation parameter crosses a critical value, the system changes its stability [40, 182, 23]. We identify the parameter α as key parameter which has an effect on the stability of model (5.2). Thus we can choose it as a bifurcation parameter. There are few studies which consider the existence of Hopf bifurcation as the existence of Hopf bifurcation in this system differs from integer order system [1]. Although in Tavazoei [164], the authors gave an example where the solutions of the system are not periodic, but converge to a periodic signals.

Now we consider a following three dimensional fractional order system:

$$^{c}D^{\alpha}x(t) = f(\beta, x), t > 0 \tag{5.8}$$

where $\alpha \in (0, 2),\ x \in \mathbb{R}^3$.

In integer order system when $\alpha = 1$, the stability of interior equilibrium point $\tilde{E}$ is depends on the sign of $\mathrm{Re}(\lambda_i),\ i = 1, 2, 3$ where λ_i are the eigenvalues of the Jacobian matrix $\frac{\partial f}{\partial x}$ about the equilibrium point $\tilde{E}$

If $\mathrm{Re}(\lambda_i) < 0, \text{for all } i = 1, 2, 3$ then $\tilde{E}$ is locally asymptotically stable otherwise unstable.

In system (5.8), when $\alpha = 1$ and β crosses a critical value β_0, then the system undergo a Hopf bifurcation around the interior equilibria if the following conditions hold:

(i) the Jacobian matrix has two complex conjugate eigenvalues $\lambda_{1,2} = \mu(\beta) \pm \eta(\beta)$, and one real $\lambda_3(\beta)$ which is denoted by $\mathrm{D}(P_E(\beta_0)) < 0$).

(ii) $\mu(\beta_0) = 0$ and $\lambda_3(\beta_0) \neq 0$.

(iii) $\frac{d\mu}{d\beta}|_{\beta=\beta_0} \neq 0$.

But in fractional order system, the stability of $\tilde{E}$ is determined by the sign of

$$f_i(\alpha, \beta) = \frac{\alpha\pi}{2} - |\arg(\lambda_i(\beta))|, \ i = 1, 2, 3.$$

Here also if $f_i(\alpha, \beta) < 0$ for all $i = 1, 2, 3$ then interior equilibrium point $\tilde{E}$ is locally asymptotically stable otherwise unstable. As $f_i(\alpha, \beta)$ has a similar effect as the real part of eigenvalues in integer order system so we can extend the Hopf bifurcation conditions to the fractional systems by replacing $\mathrm{Re}(\lambda_i)$ with $f_i(\alpha, \beta)$ as follows:

(i) $\mathrm{D}(P_{E^*}(\beta_0)) < 0$,

(ii) $f_{1,2}(\alpha, \beta_0) = 0$ and $\lambda_3(\beta_0) \neq 0$,

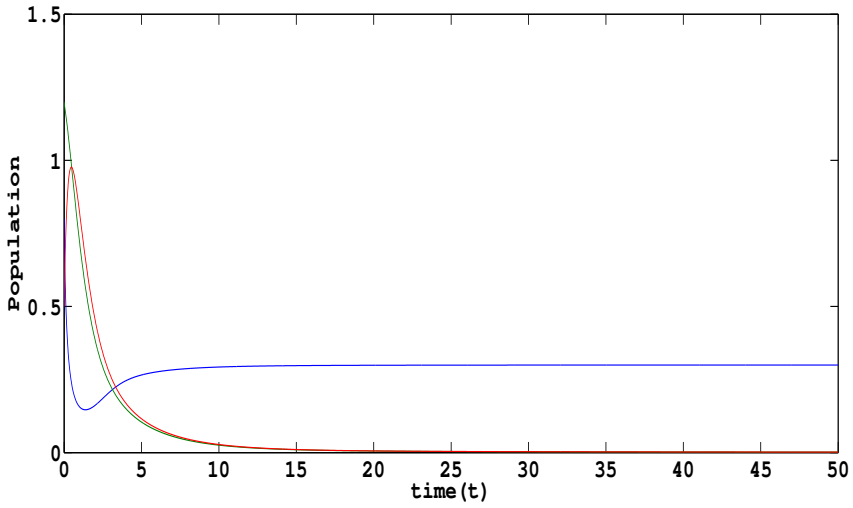
(iii) $\frac{\partial f_i}{\partial \beta}_{\beta=\beta_0} \neq 0$.

5.7 Numerical simulation

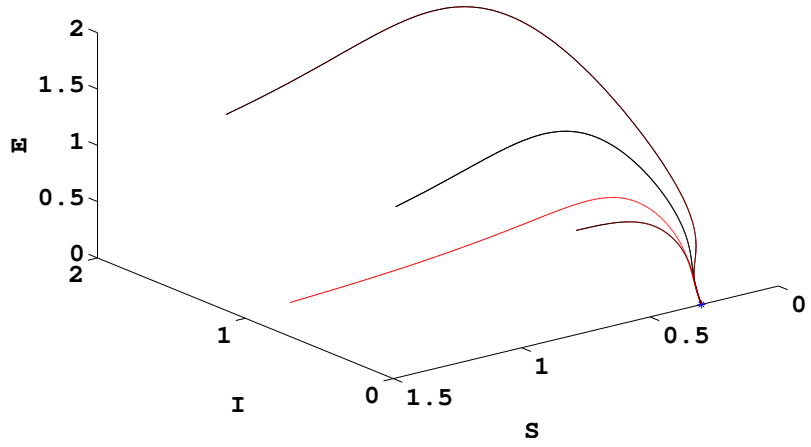
In this section, we perform some numerical simulations to verify our theoretical results. There are different numerical methods for solving nonlinear fractional differential equations [164, 41, 105, 17, 42]. Despite of all these, Adams method [105] is more appropriate and useful for solving the dynamical behaviour of the solutions of fractional differential equations. Thus in our study we have applied Adams type predictor corrector method to solve model equation (5.2) by Matlab software. The main aim for this numerical solutions is to study the effect of fractional order α and indirect transmission rate β on the dynamics behaviour of the system.

The parameter used in our first simulations are $r = 2.42; k = 0.3; \beta = 2; m = 0.5; \mu = 0.5; \tau = 4; \epsilon = 4$ and $\alpha = 0.98$. We obtain $\frac{\tau(m+\mu)}{\beta\epsilon} = 0.5 > 0.3 = k$. It follows from Theorem 5.5.1 that the susceptible individuals only equilibrium point E_1 of system (5.2) is globally asymptotically stable, which is shown in Fig.

5.1. The effect of fractional order on each population density of system (5.2) is shown in Fig. 5.2. with $\alpha = 0.6, 0.7, 0.8$ and 0.9.



(a)



(b)

Figure 5.1: (a) Time series plot of S, I, E and (b) phase portrait indicates that susceptible individuals only equilibrium point E_1 is globally asymptotically stable.

Secondly, we set the parameter values as follows: $r = 2; k = 7; \beta = 2; m = 0.1; \mu = 1.5; \tau = 1; \epsilon = 1$ and $\alpha = 0.8$. We obtain $\frac{\tau(m+\mu)}{\beta\epsilon} = 0.8 < 7 = k$, which satisfy the Theorem 6 and hence the coexistence equilibrium point is globally asymptotically stable (see Fig. 5.3) and in this case E_1 is unstable.

In fractional order system the fractional order α plays a crucial role in describing the dynamics of the system. Increasing the fractional order value α, a limit cycle will appear around the coexistence equilibrium point $\tilde{E}$ (see Fig. 5.4).

In Fig. 5.5(a) and 5.5(b), we observe that system (5.2) is locally asymptotically stable near $\tilde{E}$ by Proposition 5.4.1 which implies the trajectory converges to the equilibrium point $\tilde{E}$ and both values of α satisfies $\alpha < \alpha^*$. Now if we take the value $\alpha = 0.97$ and $\alpha = 0.99$ in which both values satisfy $\alpha > \alpha^*$. The simulations result in Fig. 5.5(c) and 5.5(d) indicates that the coexistence equilibrium point losses its stability and becomes unstable that means the trajectory diverges from the equilibrium point $\tilde{E}$. Thus we conclude that a Hopf bifurcation occurs near the equilibrium point $\tilde{E}$ when fractional order α crosses a critical value α^*. In Fig. 5.6, we draw bifurcation diagram of system (5.2) around the equilibrium point $\tilde{E}$ considering α as a bifurcation parameter.

Again, we assign a set of parameters where we fixed the derivative order $\alpha = 0.99$ and varying indirect transmission rate β to observe the effect on population dynamics. In Fig. 5.7(a) we set the parameters for system (5.2) as $r = 14/7; k = 7; \beta = 2; m = 1; \mu = 0.5; \tau = 1; \epsilon = 1$. With the choice of above parameters a limit cycles appeared and coexistence equilibrium point is $(0.75, 1.67, 1.67)$ unstable by Proposition 1, which means all trajectories diverge from this equilibrium point. But an interesting phenomena observed if we decrease the value of β, limit cycles disappears and system becomes stable. In Fig. 5.7(b), a three dimensional phase diagram represents the stability of system (5.2) when $\beta = 1.2$.

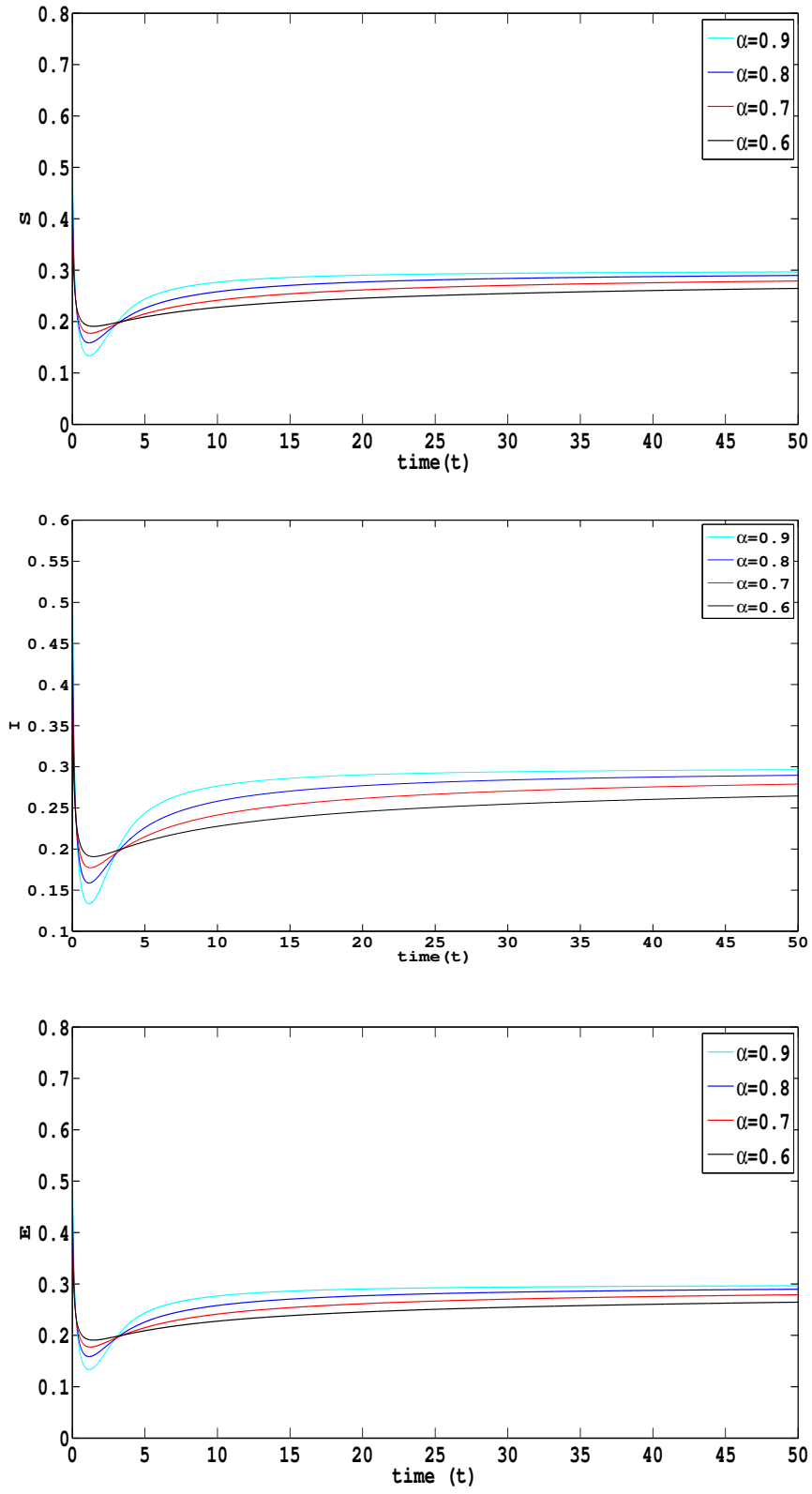


Figure 5.2: Asymptotically stable solution of S, I and E for equilibrium E_1 with different values of $\alpha \in (0, 1)$ with above given parameters.

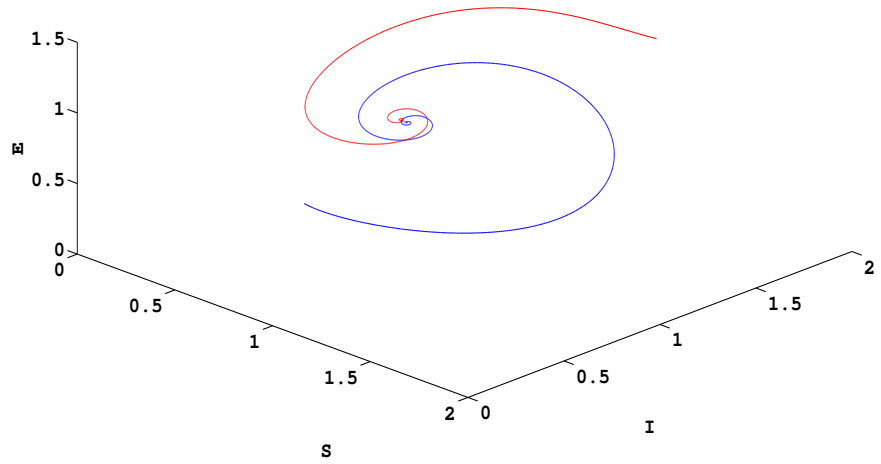
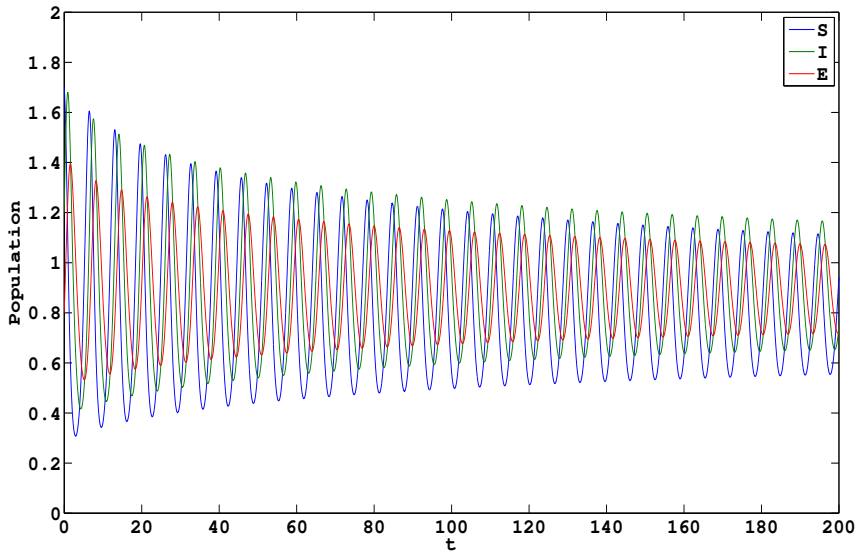
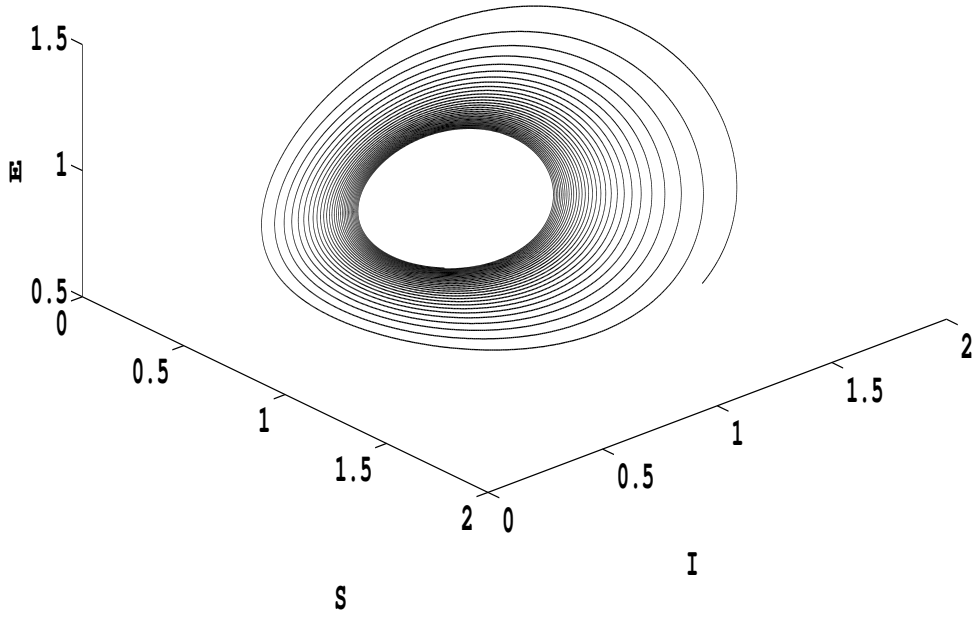


Figure 5.3: Phase portrait shows that coexistence equilibrium point is globally asymptotically stable.



(a)



(b)

Figure 5.4: (a) Numerical simulation around the equilibrium point $\tilde{E}$ and (b) phase portrait indicates increasing the value of $\alpha = 0.96$ a limit cycle appeared.

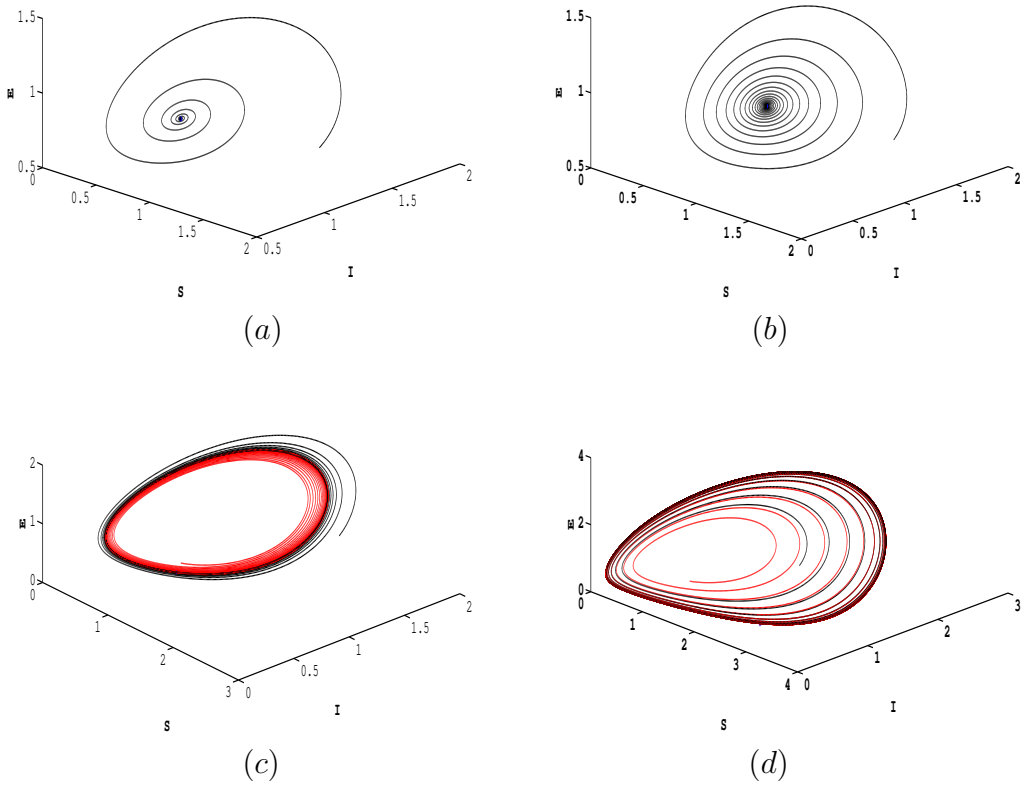


Figure 5.5: Effects of derivative order α on the dynamical behavior of system (2) with the following parameters : $r = 2; k = 7; \beta = 2; m = 0.1; \mu = 1.5; \tau = 1; \epsilon = 1$. (a) derivative order $\alpha = 0.90$, (b) derivative order $\alpha = 0.94$, (c) derivative order $\alpha = 0.97$, (d) derivative order $\alpha = 0.99$.

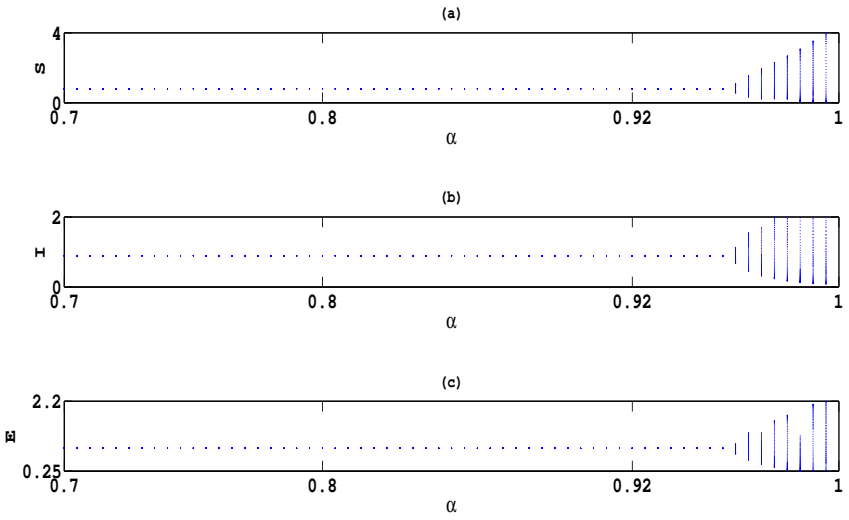
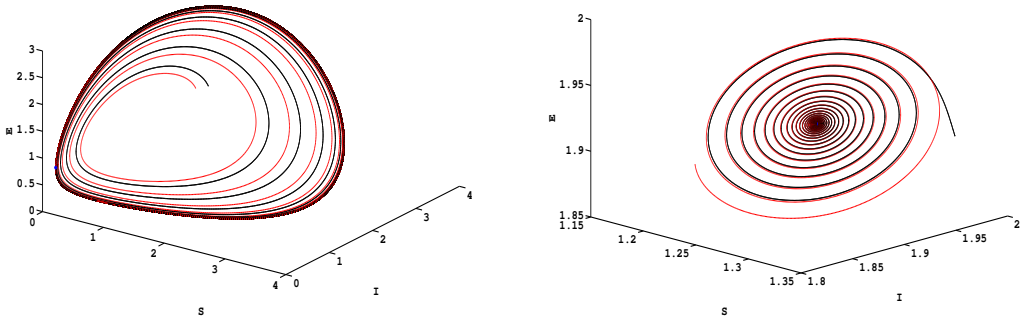


Figure 5.6: One parameter bifurcation diagram with respect to $\alpha \in (0, 1)$ and the parameters are: $r = 2; k = 7; \beta = 2; m = 0.1; \mu = 1.5; \tau = 1; \epsilon = 1$.



(a) (b)

Figure 5.7: (a) Limit cycles appears and $\tilde{E}$ unstable when $\beta = 2.0$, (b) a phase portrait indicates stability of system (5.2) when $\beta = 1.2$.

5.8 Discussion

Study of chronic wasting disease (CWD) is very important topic from theoretical as well as ecological point of view. There has been lot of work done on integer-order models describing CWD [125, 117, 158]. In integer-order model, Sharp and Pastor [158] studied the effect of carrying capacity on the system, and they observed stable limit cycle when carrying capacity crosses a critical value, but other parameter like disease transmission rate is not focused so much in their work. From literature survey, the dynamics of a fractional-order modelling CWD has not studied before. In this work , we analyzed the fractional counterpart of the integer-order model studied in [158]. We identify two important parameters α (fractional order) and β (disease transmission rate) that give rise complicated dynamics in our proposed model.

The classical time derivative is modelled with fractional derivative in the sense of Caputo. The dynamics of the introduced system has been analyzed from the perspective of existence, uniqueness, non-negativity, and boundedness, and conditions of local stability have been derived. Sufficient condition for global stability of the equilibrium point E_1 and coexistence equilibrium point $\tilde{E}$ have been derived by constructing two suitable Lyapunov function.

Through the theoretical and numerical analyses, it has been concluded that there is a possibility of Hopf bifurcation due to variation of the fractional-order α in $0 < \alpha \leq 1$. The dynamical studies also show that the impact of fractional-order derivative α on each population densities and this fractional order model is more stable than the integer model. Despite the effect of derivative order α on the system, indirect transmission rate has an important role in our model (5.2). If we fixed the derivative order $\alpha = 0.99$ and $\beta = 2$ then we observed limit cycle (see Fig. 5.7(a)) but if we decrease the value of β from 2 to 1.2 and α fixed, we see that the system is stable (see Fig. 5.7(b)).

Finally, it is noteworthy to mention that mathematical models considering fractional derivative are in broad, a more effective process to eco-epidemic models not only because we select the order α of the fractional derivative but also because of the memory properties of the fractional derivative.

6

Persistence and global stability of a Leslie-Gower predator-prey refuge system with a competitor for the prey

6.1 Introduction

There has been a growing interest in the study of refuges in predator-prey system. González-Oilvares and Ramos-Jiliberto [60] studied a predator-prey system with Holling type-II functional response and a prey refuge. They showed that there is a trend from limit cycles through non-zero stable points up to predator extinction and prey stabilizing at high densities. Kar [90] investigated a Lotka-Volterra type predator-prey system incorporating a constant proportion of prey refuges with Holling type-II response function. He remarked that it is possible to break

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the cyclic behaviour of the system if harvesting effects as controls. Chen et al. [29] analysed the uniqueness of limit cycles and global stability of the unique positive equilibrium of predator-prey system with Holling type-II functional response and a constant number of refuges. Chen et al. [27] , and Yue [187] studied Leslie-Gower predator-prey system incorporating a constant proportion prey refuge and showed the global stability at the interior equilibrium point. More results on the effects of a prey refuge can be found in [137, 139, 91, 34, 28, 86, 113, 163]. Previous studies on Leslie-Gower predator-prey system are mainly confined into constant proportion of refuge which acts on the system as an external decreasing of the uptake rate and half saturation constant, does not change the dynamical behaviour of the prey-predator model. Thus our main object in this work to modify the refuge term. Recently, Mukherjee [138] studied the effect of immigration and refuge on the dynamics of three species system. He discussed about the persistence of the system and global stability. Model considered by him is of Lotka-Volterra type. In another paper [139] Mukherjee investigated same type of situation without immigration and predation process follows Holling-type II response function. In [138, 139], the author did not addressed what will be dynamical consequence if Leslie-Gower form is taken. Furthermore, we are interested to know the dynamics consequence of the predator-prey system in presence of a competitor for the prey in a Leslie-Gower model.

6.2 Mathematical model

In Leslie-Gower prey-predator model, predator equation is taken logistic growth with carrying capacity proportional to the prey density. This type of situation are applicable in ecology [104, 121, 167] because the direct conversion of prey density into offspring is inappropriate for a small mammalian predator that uses most of its energy intake on generating heat and because model of Leslie's type assume interferences of predators which is justifiable for territorial predators [167]. In this chapter we introduce a predator-prey model with Leslie-Gower functional response incorporating a positive constant prey refuge with the presence of a competitor for the prey :

$$\begin{aligned}\frac{dx}{dt} &= x(r_1 - b_1x) - \alpha xy - a_1(x-m)z \\ \frac{dy}{dt} &= y(r_2 - b_2y) - \beta xy \\ \frac{dz}{dt} &= z\left(r_3 - \frac{a_2z}{k+x-m}\right)\end{aligned} \tag{6.1}$$

with initial conditions $x(0) > m, y(0) \geq 0, z(0) \geq 0$.

Here x, y, z denotes the density of the prey, competitor for the prey and predator respectively. r_1 is the intrinsic growth rate of the prey species and r_2 is the intrinsic growth rate of the competitor for the prey species. b_1 is the infraspecific competition coefficient of the prey. α denotes the interspecific competition coefficient of the competitor for the prey. b_2 represents the intraspecific competition coefficient of the competitor for the prey. β corresponds to the intraspecific competition coefficient of the competitor for the prey. r_3 describes the growth rate of predator. a_1 is the per capita predator consumption rate. a_2 is the efficiency with which predators convert consumed prey. m is the constant number of prey using refuge. k is the half saturation constant.

Specific example illustrates the above model: Consider two species of aphid *(Acyrthosiphon pisum and Megoura viciae)* competing for the same food plant and a species of a specialist parasitoid *(Aphidius ervi)* that attacks only one of the aphids *(A. pisum)*. From experimental studies van Veen et al. [169] showed that (i) when the two aphid species compete for resources in the absence of parasitoid. *A. pisum* excludes *M. viciae*. (ii) When the aphid species and the parasitoid are all present, all three species can coexist.

6.3 Preliminaries

6.3.1 Positivity

Lemma 6.3.1. *All solution of system (6.1) with positive initial conditions are positive i.e* $x(t) > 0, y(t) > 0, z(t) > 0$ *for all* $t \geq 0$ *in the interval* $[0, \infty)$.

Proof. Since the right hand side of system (6.1) is continuous and locally Lipschitzian on C, the solution $(x(t), y(t), z(t))$ of system (6.1) with initial conditions

exists and is unique on $[0, \phi)$, where $0 < \phi \leq \infty$ [62].

From system (6.1), we have

$$x(t) \geq x(0) \exp\left\{ \int_0^t (r_1 - b_1 x(\xi) - \alpha y(\xi) - a_1 z(\xi)) d\xi \right\} \geq 0,$$
$$y(t) = y(0) \exp\left\{ \int_0^t (r_2 - b_2 y(\xi) - \beta x(\xi)) d\xi \right\} \geq 0,$$
$$z(t) = z(0) \exp\left\{ \int_0^t (r_3 - \tfrac{a_2 z(\xi)}{k + x(\xi) - m}) d\xi \right\} \geq 0.$$

Thus any trajectory starting in $\mathbb{R}^3_+$ cannot cross the co-ordinate axes. This completes the proof.

6.3.2 Boundedness

Lemma 6.3.2. *The set* $B = \{(x, y, z) \in \mathbb{R}^3_+ : 0 < W(t) = x + y + z \leq \frac{M}{\zeta}\}$ *is a region of attraction for all solutions initiating in* $\mathbb{R}^3_+$ *with positive initial conditions, where* $M = \frac{(r_1+\lambda)^2}{4b_1} + \frac{(r_2+\lambda)^2}{4b_2} + \frac{\zeta(r_3+\lambda)^2}{4a_2}, \zeta = \frac{b_1}{(r_1 + b_1(k-m))}$ *provided* $k > m$.

Proof. Let us define $W(t) = x + y + z$ and $\lambda > 0$ be a constant. Then

$$\frac{dW}{dt} + \lambda W = x(r_1 - b_1 x) - \alpha xy - a_1(x - m)z + \lambda x + y(r_2 - b_2 y - \beta x + \lambda) + z(r_3 + \lambda - \tfrac{a_2 z}{k + x - m})$$

$$\leq x(r_1 - b_1 x + \lambda) + y(r_2 - b_2 y + \lambda) + z(r_3 + \lambda - \tfrac{a_2 z}{k + x - m})$$

$$\leq \tfrac{(r_1+\lambda)^2}{4b_1} + \tfrac{(r_2+\lambda)^2}{4b_2} + \tfrac{\xi(r_3+\lambda)^2}{4a_2} = M, \text{ where } \zeta = \tfrac{b_1}{(r_1 + b_1(k-m))}.$$

By using differential inequality [18] we obtain,

$$0 < W(x(t), y(t), z(t)) \leq \tfrac{M(1 - e^{-\zeta t})}{\zeta} + (x(0), y(0), z(0)) e^{-\zeta t}$$

Taking limit $t \to \infty$, we have $0 < W(t) \leq \frac{M}{\zeta}$.
This proves the Lemma.

6.4 Equilibria

Evidently, system (6.1) has at most five equilibrium points: the trivial equilibrium point $E_0 = (0, 0, 0)$ which does not belongs to B. The axial equilibrium point $E_1 = (\frac{r_1}{b_1}, 0, 0)$. Planner equilibrium point $E_{12} = (\bar{x}, \bar{y}, 0)$ where $\bar{x} = \frac{r_1 b_2 - r_2 \alpha}{b_1 b_2 - \alpha\beta}, \bar{y} = \frac{r_2 b_1 - r_1 \beta}{b_1 b_2 - \alpha\beta}$. E_{12} is feasible if $b_1 b_2 > \alpha\beta$ and $r_1 b_2 > r_2\alpha, r_2 b_1 > r_1\beta$ or $b_1 b_2 < \alpha\beta$ and $r_1 b_2 < r_2\alpha, r_2 b_1 < r_1\beta$. Another planner equilibrium point $E_{13} = (\widehat{x}, 0, \widehat{z})$ where $\widehat{x}$ is the positive root of the equation

$$(a_2 b_1 + a_1 r_3)x^2 + (a_1 r_3 k - 2m a_1 r_3 - a_2 r_1)x + a_1 r_3 m(m - k) = 0. \tag{6.2}$$

and $\widehat{z} = \frac{r_3(k + \widehat{x} - m)}{a_2}$. The interior equilibrium point is given by $E^* = (x^*, y^*, z^*)$ where $y^* = \frac{r_2 - \beta x^*}{b_2}, z^* = \frac{r_3(k + x^* - m)}{a_2}$ and x^* is the positive root of the equation

$$(b_1 a_2 b_2 - \alpha\beta a_2 + r_3 b_2 a_1)x^2 - \{r_1 a_2 b_2 - \alpha r_2 a_2 - r_3 b_2 a_1(k - 2m)\}x - r_3 b_2 a_1 m(k - m) = 0. \tag{6.3}$$

E^* is feasible if $r_2 > \beta x^*, k + x^* > m$.

Theorem 6.4.1. *(i) Equilibrium points E_1 and E_{12} are always unstable. (ii) E_{13} is locally asymptotically stable if $r_2 < \beta\widehat{x}$.*

Proof. Proof follows immediately by linearising around the equilibria.

Theorem 6.4.2. *The interior equilibrium point E^* of system (6.1) is locally asymptotically stable if $\left(\frac{a_1 m z^*}{x^*} + b_1 x^*\right) b_2 \geq \alpha\beta x^*$.*

Proof. The Jacobian matrix of system (6.1) at the equilibrium point E^* is given by

$$J(E^*) = \begin{pmatrix} -\frac{a_1 m z^*}{x^*} - b_1 x^* & -\alpha x^* & -a_1(x^* - m) \\ -\beta_2 y^* & -b_2 y^* & 0 \\ \frac{r_3^2}{a_2} & 0 & -r_3 \end{pmatrix}$$

The characteristic equation about E^* is given by:

$$\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0 \tag{6.4}$$

$$\text{where} \begin{cases} A_1 = \frac{a_1 m z^*}{x^*} + b_1 x^* + b_2 y^* + r_3, \\ A_2 = (\frac{a_1 m z^*}{x^*} + b_1 x^*)(b_2 y^* + r_3) + b_2 r_3 y^* - \alpha\beta x^* y^* + a_1(x^* - m)\frac{r_3^2}{a_2}, \\ A_3 = (\frac{a_1 m z^*}{x^*} + b_1 x^*) b_2 y^* r_3 - \alpha\beta x^* y^* r_3 + a_1(x^* - m)\frac{r_3^2}{a_2} b_2 y^* \end{cases}$$

Now $A_1 > 0, A_3 > 0$ follows from the assumption of the Theorem (6.4.2). Also $A_1 A_2 > A_3$. Therefore by Routh-Hurwitz criterion the result follows.

6.5 Local bifurcation analysis

In this section, we use the application of Sotomayor's theorem [161] to investigate the local bifurcation around the equilibrium points of system (6.1). As the existence of non-hyperbolic equilibrium point is a necessary but not sufficient condition for bifurcation to occur therefore we choose a parameter which gives zero eigenvalues to the Jacobian at the equilibria. Now rewrite system (6.1) in the form :

$\frac{dX}{dt} = F(X)$ where $X = (x, y, z)^t$ and $F = (F_1, F_2, F_3)$ where $F_1 = x(r_1 - b_1 x) - \alpha xy - a_1(x - m)z$, $F_2 = y(r_2 - b_2 y) - \beta xy$ and $F_3 = z\big(r_3 - \frac{a_2 z}{k+x-m}\big)$. The local bifurcation near the equilibrium points is investigated in the following theorems:

Theorem 6.5.1. *System (6.1) undergoes a transcritical bifurcation at the axial equilibrium point E_1 but no saddle node bifurcation can occur when the parameter β crosses the critical value $\beta^* = \frac{b_1 r_2}{r_1}$.*

Proof. One of the eigenvalues of the Jacobian matrix $J(E_1)$ will be zero if $\beta = \beta^* = \frac{b_1 r_2}{r_1}$. Now the Jacobian matrix of system (6.1) at E_1 with zero eigenvalue

is given by

$$J(E_1) = \begin{pmatrix} -r_1 & -\frac{\alpha r_1}{b_1} & -a_1(\frac{r_1}{b_1} - m) \\ 0 & 0 & 0 \\ 0 & 0 & r_3 \end{pmatrix}$$

Let $V = (v_1, v_2, v_3)^t$ be the eigenvector corresponding to eigenvalue $\lambda = 0$. Thus $V = (v_1, -\frac{v_1 b_1}{\alpha}, 0)^t$ where v_1 be any non zero real number. Also, let $W = (w_1, w_2, w_3)^t$ represents the corresponding eigenvector of $J(E_1)^t$ to the eigenvalues of $\lambda = 0$. Hence $J(E_1)^t W = 0$ gives that $W = (0, w_2, 0)^t$ where w_2 be any non zero real number. Now $F_\beta(E_1, \beta^*) = (0, 0, 0)^t$, here $F_\beta(E_1, \beta)$ represents the derivative of $F = (F_1, F_2, F_3)^t$ with respect to β. Then we get $W^t[F_\beta(E_1, \beta^*)] = 0$.

Thus according to Sotomayor's theorem system (6.1) has no saddle-node bifurcation at $\beta = \beta^*$.

Again

$$DF_\beta(E_1, \beta^*) = \begin{pmatrix} 0 & 0 & 0 \\ 0 & -\frac{r_1}{b_1} & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

Then, $W^t[DF_\beta(E_1, \beta^*)V] = -\frac{r_1 v_2 w_2}{b_1} \neq 0$.

Now

$$D^2F(E_1, \beta^*)(V, V) = \begin{pmatrix} (-2b_1 - \alpha - a_1)v_1^2 - \alpha v_1 v_2 - a_1 v_1 v_3 \\ -\beta v_1 v_2 - (\beta + 2b_2)v_2^2 \\ -\frac{2a_2}{k+x-m} v_3^2 \end{pmatrix}$$

Therefore, $W^t[D^2F(E_1, \beta^*)(V, V)] = \frac{b_1 k^2}{\alpha}[\beta - (\beta + 2b_2)b_1] \neq 0$.

Thus according to Sotomayor's theorem system (6.1) has a transcritical bifurcation at E_1 when the parameter β crosses the critical value β^*. Furthermore, as the Jacobian matrix of E_1 has three linear factors, so no Hopf bifurcation can occurs.

6.5.1 Numerical example for transcritical bifurcation

Choose $r_1 = 12, b_1 = 10, \alpha = 2, a_1 = 2, m = 0.5, r_2 = 6, \beta = 5, a_2 = 2, b_2 = 1, r_3 = 1, k = 1.5$ then system (6.1) admits a transcritical bifurcation at $E_1(1.2, 0, 0)$ with respect to β (see Fig. 6.1).

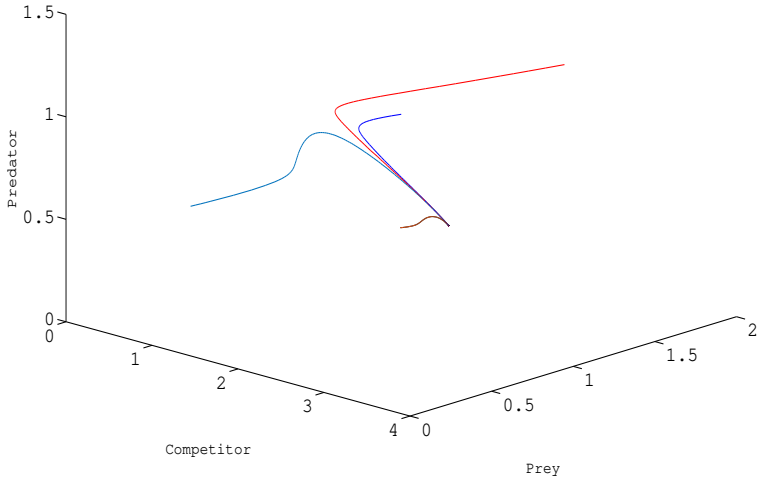


Figure 6.1: Transcritical bifurcation near E_1.

Remark 6.5.1. *System (6.1) does not admits any local bifurcation (saddle-node, transcritical or Hopf-bifurcation) at E_{12} as the Jacobian matrix $J(E_{12})$ has no zero eigenvalues due to the existence of E_{12}.*

Theorem 6.5.2. *System (6.1) admits a transcritical bifurcation but no saddle-node bifurcation around the equilibrium point E_{13} when r_2 crosses the critical value $r_2^* = \beta\widehat{x}$.*

Proof. Proof is similar to the proof of Theorem 6.5.1.

Remark 6.5.2. *System (6.1) does not undergoes any Hopf-bifurcation around the interior equilibrium point E^* as in equation (6.4), $A_1 > 0$ and $A_1A_2 - A_3$ cannot be equal to zero.*

Theorem 6.5.3. *Suppose that* $\frac{4b_1b_2a_2}{k+x^*-m} > \left\{\frac{(\alpha+\beta)^2a_2}{k+x^*-m} + b_2\left(\frac{(x^*-m)a_1}{x^*} + \frac{r_3^2}{k+x^*-m}\right)\right\}$ *and* $r_2 > \beta x^*$. *Then the interior equilibrium point* E^* *is globally asymptotically stable.*

Proof. First note that, E_{13} is unstable as $r_2 > \beta x^*$ and other boundary equilibrium points are always unstable whenever they exist.

Consider the following positive definite function about E^*

$$V(t) = (x - x^* - x^* \ln \frac{x}{x^*}) + (y - y^* - y^* \ln \frac{y}{y^*}) + (z - z^* - z^* \ln \frac{z}{z^*})$$

Differentiating V with respect to t along the solution of system (6.1), we get

$$\frac{dV}{dt} = (x-x^*)\{r_1-b_1x-\alpha y-\frac{a_1(x-m)}{x}\}+(y-y^*)\{r_2-b_2y-\beta x\}+(z-z^*)\{r_3-\frac{a_2z}{k+x-m}\}$$

$$= (x-x^*)\{-b_1(x-x^*)-\alpha(y-y^*)+\frac{a_1(x^*-m)}{x^*}-\frac{a_1(x-m)}{x}\}+(y-y^*)\{-b_2(y-y^*)-\beta(x-x^*)\}+(z-z^*)\{\frac{a_2z^*}{K+x^*-m}-\frac{a_2z}{k+x-m}\}$$

$$\leq -b_1(x-x^*)^2+(\alpha+\beta)|(x-x^*)||(y-y^*)|-b_2(y-y^*)^2-\frac{a_2(z-z^*)^2}{k+x^*-m}+\{\frac{(x^*-m)a_1}{x^*}+\frac{r_3}{k+x^*-m}\}|x-x^*||z-z^*|$$

We note that $\dot{V}$ is negative definite if

$$\frac{b_1b_2a_2}{k+x^*-m} > \frac{1}{4}\left[\frac{(\alpha+\beta)^2a_2}{k+x^*-m} + b_2\left\{\frac{(x^*-m)a_1}{x^*} + \frac{r_3^2}{k+x^*-m}\right\}\right]$$

Thus the condition of Theorem 6.5.3 implies that V is a Lyapunov function and hence the theorem follows.

6.6 Persistence

Biologically persistence means the long time survival of all population in a future time whatever may be the initial populations. By differential inequality argument

we state some result guaranteeing the persistence of all the populations of system (6.1).

Theorem 6.6.1. *(i) If* $x(t) > m$ *then* $\lim_{t\to\infty} \sup x(t) \leq \frac{r_1}{b_1}$ *(ii) If* $x(t) \leq m$ *and* $r_1 b_2 > \alpha r_2$ *then* $\lim_{t\to\infty} \inf x(t) \geq \frac{r_1 b_2 - \alpha r_2}{b_1 b_2}$.

Proof. (i) When $x(t) > m$, $\frac{dx}{dt} \leq (r_1 - b_1 x)x \implies \lim_{t\to\infty} \sup x(t) \leq \frac{r_1}{b_1}$

(ii) When $x(t) \leq m$, $\frac{dx}{dt} \geq (r_1 - b_1 x)x - \alpha x \frac{r_2}{b_2} = (r_1 - \frac{\alpha r_2}{b_2} - b_1 x)x \implies \lim_{t\to\infty} \inf x(t) \geq \frac{r_1 b_2 - \alpha r_2}{b_1 b_2}$.

Theorem 6.6.2. *(i) If* $x(t) > m$ *and* $r_2 > \frac{\beta r_1}{b_1}$ *then* $\lim_{t\to\infty} \inf y(t) \geq \frac{b_1 r_2 - \beta r_1}{b_1 b_2}$ *(ii) If* $x(t) \leq m$ *and* $r_2 > \beta m$ *then* $\lim_{t\to\infty} \inf y(t) \geq \frac{r_2 - \beta m}{b_2}$.

Proof. when $x(t) > m$, $\lim_{t\to\infty} \sup x(t) \leq \frac{r_1}{b_1}$ then from the second equation of (6.1) we have $\frac{dy}{dt} \geq (r_2 - \frac{\beta r_1}{b_1} - b_2 y)y \implies \lim_{t\to\infty} \inf y(t) \geq \frac{b_1 r_2 - \beta r_1}{b_1 b_2}$.

(ii) If $x(t) \leq m$, then $\frac{dy}{dt} \geq (r_2 - \beta m - b_2 y)y$.

As, $r_2 > \beta m$, this implies that $\lim_{t\to\infty} \inf y(t) \geq \frac{r_2 - \beta m}{b_2}$.

Theorem 6.6.3. *If* $k > m$ *then* $\lim_{t\to\infty} \inf z(t) \geq \frac{r_3(k-m)}{a_2}$.

Proof. Since $k > m$ then $k + x - m > k - m$ and hence $-\frac{1}{k+x-m} > -\frac{1}{k-m}$.

From third equation of (6.1), we have $\frac{dz}{dt} \geq z(r_3 - \frac{a_2 z}{k-m}) \implies \lim_{t\to\infty} \inf z(t) \geq \frac{r_3(k-m)}{a_2}$.

6.7 Influence of the prey refuge

In the following we shall discuss the influence of prey refuge on each population when the coexistence equilibrium point E^* is exists and is stable. It is easy to see that x^*, y^*, z^* are all continuous differential functions of parameter m.

Now let α be any positive root of equation (6.3).

Then $\alpha = \frac{-B \pm \sqrt{B^2 - 4AC}}{2A}$ where

$A = b_1a_2b_2 - \alpha\beta a_2 + r_3b_2a_1, B = -\{r_1a_2b_2 - \alpha r_2a_2 - r_3b_2a_1(k-2m)\}, C = -r_3b_2a_1m(k-m)$.

Now $\frac{d\alpha}{dm} = -\frac{dB}{dm} + \frac{1}{2}\frac{2B\frac{dB}{dm} - 4A\frac{dC}{dm}}{\sqrt{B^2-4AC}} > 0$ provided,

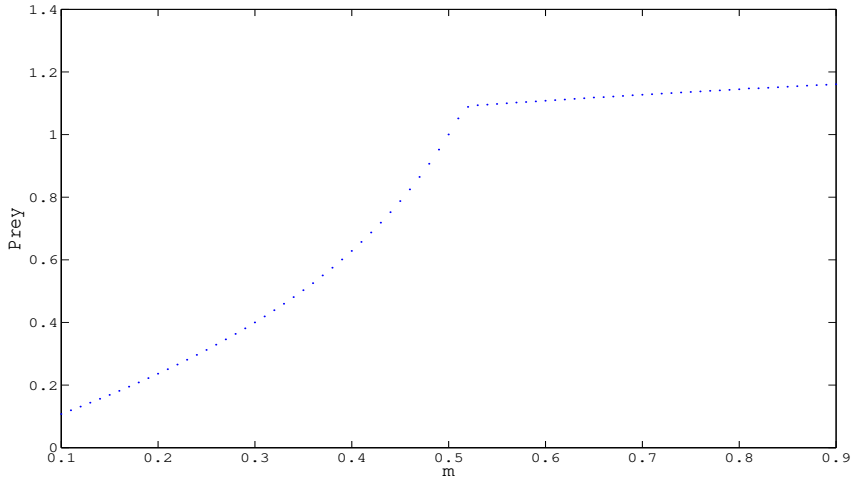
$$\frac{a_2}{a_1} > \frac{r_3}{b_1} \text{ and } \min\left\{\frac{\alpha\beta a_2}{a_1r_3}, \frac{r_2a_2}{r_1a_1}\right\} < b_2 < \frac{\alpha\beta}{b_1}. \tag{6.5}$$

Again $\frac{dy^*}{dm} = -\frac{\beta}{b_2}\frac{dx^*}{dm} < 0$ and $\frac{dz^*}{dm} = \frac{r_3}{a_2}(\frac{dx^*}{dm} - 1)$.

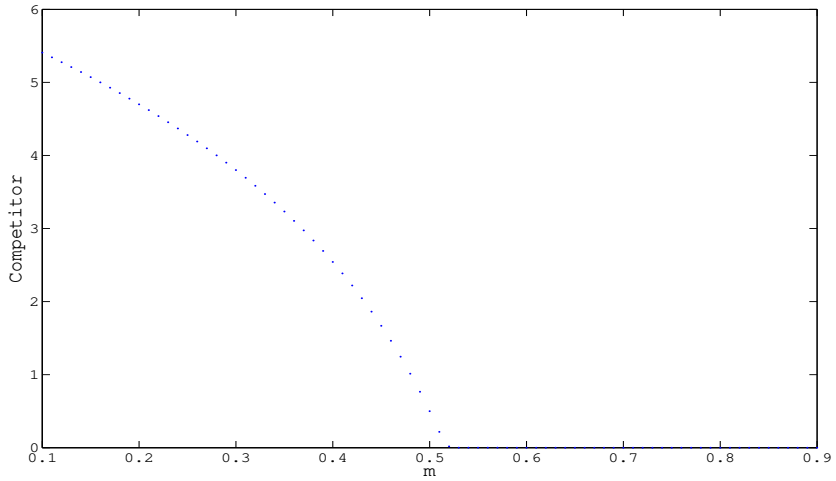
Clearly x^* is strictly increasing function of parameter m whenever (6.5) holds and increasing the amount of prey refuge leads to the increasing density of the prey species. y^* is strictly decreasing function of parameter m and increasing the amount of prey refuge leads to the decreasing density of the competitor prey species. The presence of negative term in the third equation indicates that increasing the amount of prey refuge may decrease the predator density as long as $\frac{dx^*}{dm} < 1$.

6.7.1 Numerical example for influence of the prey refuge

Here we choose a set of parameters $r_1 = 12, b_1 = 10, \alpha = 2, a_1 = 2, m = 0.5, r_2 = 6, \beta = 5.5, a_2 = 2, b_2 = 1, r_3 = 1, k = 1.5$ and in this case interior equilibrium point E^* is locally asymptotically stable. Influence of prey refuge on susceptible and infected prey population is given in Fig. 6.2. and Fig. 6.3.



(a)



(b)

Figure 6.2: Influence of prey refuge on (a) susceptible prey population and (b) infected prey population

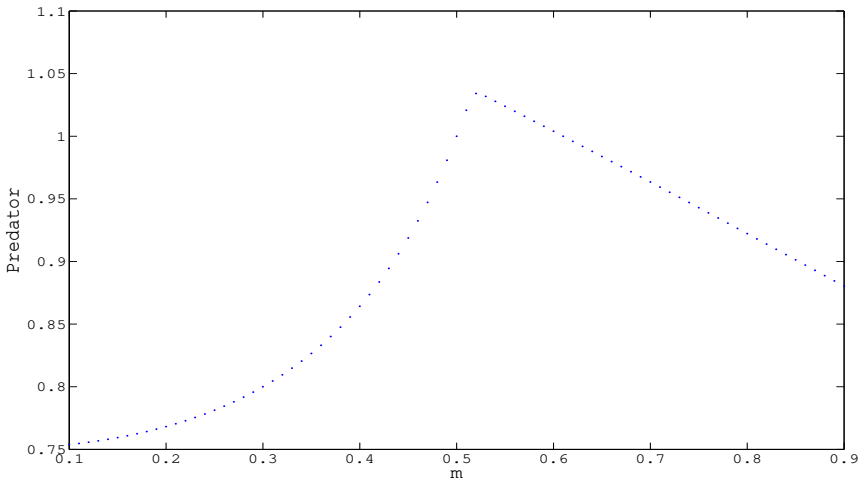


Figure 6.3: Influence of prey refuge in predator population

6.8 Numerical simulation

Dynamical behaviour of Leslie-Gower predator-prey model is not affected by refuge. If interspecific competition is allowed into the system, oscillation can emerge. We have taken the rate of interspecific competition low and high. We select $r_1 = 12, b_1 = 10, \alpha = 2, a_1 = 2, m = 0.5, r_2 = 6, \beta = 5.5, a_2 = 2, b_2 = 1, r_3 = 1, k = 1.5$. Our numerical result shows that system (6.1) converges to this point $E^*(1, 0.5, 1)$(see Fig. 6.4).

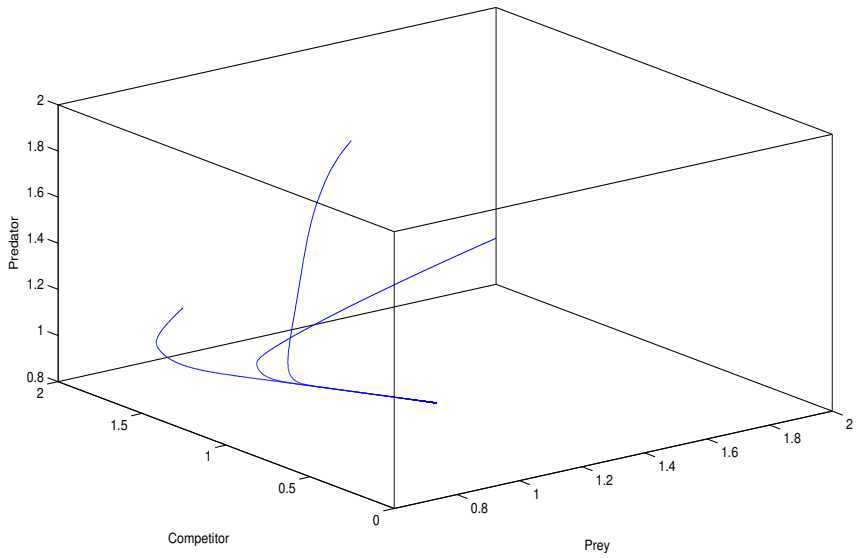


FIGURE 6.4: The figure shows that system (6.1) is locally stable

Further we considered $r_1 = 4, b_1 = 1, \alpha = 1.2, a_1 = 2, m = 0.5, r_2 = 6, \beta = 5, a_2 = 1.5, b_2 = 1, r_3 = 2, k = 0.25$, the system oscillates near this equilibrium point $E^*(\frac{5}{7}, \frac{17}{7}, \frac{13}{21})$. Also some chaotic type oscillation is observed (see Fig. 6.5).

In the first case $\alpha\beta = 11$ and $b_1 b_2 = 10$ that indicates interspecific competition is low. In the second case $\alpha\beta = 6$ and $b_1 b_2 = 1$ that implies interspecific competition is high i.e all the equilibrium points are unstable in nature. That causes chaotic type motion.

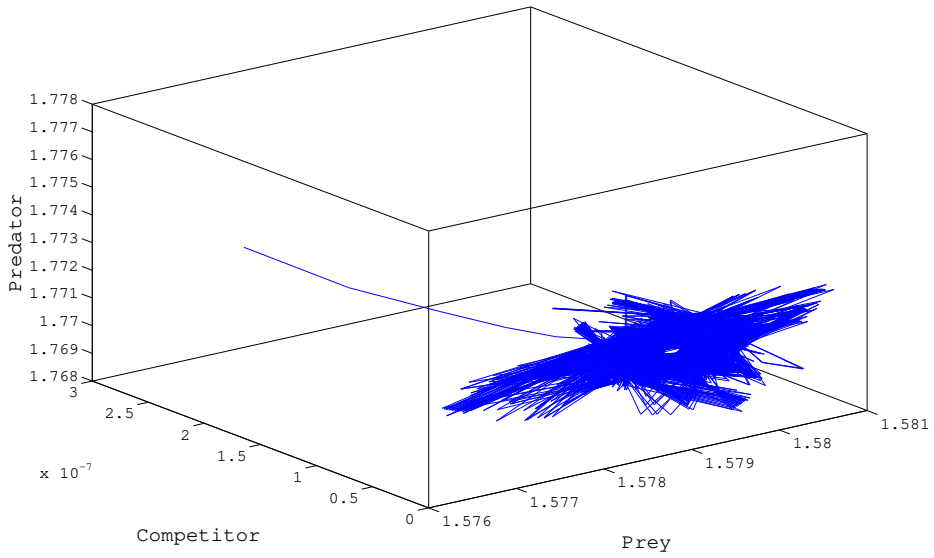


Figure 6.5: The system (6.1) is unstable and chaotic type oscillation observed

6.9 Discussion

In this chapter, we have considered a prey-predator system where prey has a competitor. Due to predation pressure prey population uses refuge mechanism. The model is formulated according to Leslie-Gower. This type of model usually exhibits stable behaviour with or without refuge. But the dynamics of the model may be changed if the interspecific competition is allowed. We have throughly investigated bifurcation analysis around the equilibria. We note that certain parameters of the system are very sensitive to give transcritical bifurcation. We further observe that Hopf-bifurcation cannot occur around the equilibria which is either interior or boundary. Thus our system is either stable or unstable around the coexistence equilibrium point. We have found five possible equilibria, namely trivial equilibrium point E_0, axial equilibrium point E_1, predator free equilibrium point E_{12}, competition free equilibrium point E_{13} and coexistence equilibrium point E^*. Here the boundary equilibrium points E_0, E_1, E_{12} are always unstable in nature where as E_{13} may be stable when the intrinsic growth rate of competitor remains below certain threshold value ($r_2 < \beta\widehat{x}$). Local stability at the coexistence equilibrium point can be checked from the condition of Theorem 6.4.2

We observed that from the numerical simulation that chaotic motion can arrise if the condition of Theorem 6.4.2 is violated. Further we have derived a sufficient condition for global stability condition of the coexistence equilibrium point. By using differential inequality argument we found persistence condition of the population. The novelty of our paper is the occurrence of transcritical bifurcation but no Hopf-bifurcation around the equilibria. Though Mukherjee [138, 139] showed Hopf-bifurcation in his system and did not carried out local bifurcation analysis.

7

Conclusion

Predator-prey interaction is a complex process in nature. We study this process in this thesis through mathematical models available in ecology and eco-epidemiology in integer-order as well as fractional order system. We have found some results in each chapter that are very interesting in the field of ecology and eco-epidemiology. Through out the thesis we have considered predator is specialist in nature.

A predator-prey-pathogen model has been considered in Chapter 2, where predator influences the transmission rate of infection in its prey. This chapter is developed from the modification of [132], where Morozov considered a predator dependent transmission rate in its linear form and concluded that the transmission rate can result in bi-stability and destabilization even for a Holling type-1 predator functional response. But the transmission rate in linear form shows a unboundedness in the system when predator population increases. Thus, we modified the incidence function by including a saturation level therefore the modified transmission rate is more reasonable than the linear one as it includes the

behavioural change and crowding effect of the predator and prevents unboundedness of the disease transmission rate. We have seen that the predator density dependent transmission rate can causes oscillation in our system. We also have noticed that the system is impermanent if the death rate of predator crosses a certain threshold value.

Generally most of ecological populations are suffer from different kind of infectious diseases and many researchers have focused on this issue through modelling [171, 63, 184, 189, 68]. Due to disease in prey population they are very weak and they can be easily vulnerable to the predator. However there may be a risk factor for predator to consume the infected prey [52]. Naturally the prey population can protect themselves in various ways to avoid being killed by their predators whether they are infected or not. Some literature survey [72, 97, 130, 60, 77] conclude that Prey refuge have a stabilizing effect on predator-prey interaction, and using such policies can save prey species from extinction. In Chapter 3 we have focused on these two issues and we have seen that prey refuge has a major impact on each population. Increasing the amount of infected prey refuge decreases the density of susceptible prey whereas in contrast to the density of infected prey but the density of predators may decreases. In particular, when the refuge capacity lies in a certain range, the periodic oscillation may appear. If this refuge rate exceeds the some threshold value, periodic solution disappears.

Chapter 4, is based on a simple eco-epidemic model where the prey population is infected by chronic wasting disease (CWD). This is a prion disease of mule deer, white-tailed deer, elk, and moose and this disease belongs to the family of transmissible spongiform encephalopathy (TSEs). As this is a fatal disease so to eradicate this disease no vaccination is still available thus we have introduced predator population who only consume the infected population to control the disease. A fractional-order eco-epidemic model of chronic wasting disease is investigated in Chapter 5. In integer order system Sharp and Pastor [158] observed a stable limit cycle when carrying capacity crosses a critical value but in our system we observed that fractional order derivative has an great impact on the system dynamics and it has the potential to change the stable limit cycle to unstable one. We also have seen that there is a possibility of Hopf bifurcation when the fractional order derivative crosses a certain value.

In Chapter 6, we have discussed a Leslie-Gower predator prey refuge system in presence of a competitor for prey population. Generally this Leslie-Gower type model exhibits stable behaviour with or without refuge but in presence of a competitor the system dynamics of the model may be changed. In this analysis we have thoroughly investigated local bifurcation analysis around the equilibria. The novelty of this chapter is the occurrence of transcritical bifurcation but no Hopf-bifurcation around the equilibria.

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